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Assessment of Risk Factors, Disease Burden, Clinical Phenotypes, Biomarkers and Treatment of Hidradenitis Suppurativa

DOCTORAL DISSERTATION

Medicine and Health Sciences
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Pūlingo hidradenito rizikos veiksnių, ligos naštos, klinikinių fenotipų, biologinių žymenų ir gydymo vertinimas

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ABBREVIATIONS

CV – cardiovascular

DLQI – Dermatology Life Quality Index

EHSF – European Hidradenitis Suppurativa Foundation

HiSCR – Hidradenitis Suppurativa Clinical Response

HS – Hidradenitis Suppurativa

IHS4 – International Hidradenitis Suppurativa Severity Score System

IL – Interleukin

JAKi – Janus Kinase inhibitor

PASH – Pyoderma gangrenosum, Acne, Suppurative Hidradenitis

PAPASH – Pyogenic Arthritis, Pyoderma gangrenosum, Acne, Suppurative Hidradenitis

QoL – Quality of Life

SCC – Squamous Cell Carcinoma

STAT – Signal Transducer and Activator of Transcription

STEEP – Skin-Tissue-sparing Excision with Electrosurgical Peeling

TNF-α – Tumour Necrosis Factor alpha

VUHSK – Vilnius University Hospital Santaros Klinikos

1. INTRODUCTION

1.1. Brief introduction

Hidradenitis suppurativa (HS), also known as acne inversa, is a lifelong inflammatory skin disease characterized by the presence of deep-seated purulent lesions and scars, which primarily manifest in areas where skin folds meet and where apocrine glands are abundant (1). The heterogeneous nature of HS has led to the identification of various stages and phenotypes within the disease spectrum (2).

HS, being a chronic inflammatory disease, exemplifies the connection between skin-related conditions and comorbid systemic diseases, as they share common inflammatory pathways. Patients with HS tend to have a higher burden of comorbidities compared to the general population, and HS itself is independently associated with several specific comorbid conditions (such as polycystic ovary syndrome, diabetes mellitus, metabolic syndrome, inflammatory bowel disease, psychiatric disorders, lymphoma, etc.) (3,4).

Individuals affected by HS encounter a substantial time lapse, estimated at 7.2 years globally, between the onset of the disease and its diagnosis. This delay is influenced by various factors, such as doctors' lack of recognition, the embarrassment and stigma attached to the condition, and socioeconomic obstacles (5–7). The limited availability of effective therapies and frequent exacerbations present a difficult obstacle in treatment, resulting in a detrimental effect on the quality of life (8,9). In addition, given the painful nature of the lesions, their occurrence in sensitive areas, the presence of drainage, unpleasant odour, and resulting scarring, HS can significantly impact a person's psychological and social well-being. As HS significantly compromises the quality of a patient’s life, it is crucial that diagnosis should be identified without delays and decrease the burden of the disease on patients and the healthcare system (1).

1.2. Aim of the study

The aim is to analyse the association of risk factors, clinical presentation, biomarkers and treatment efficacy of hidradenitis suppurativa in the Lithuanian context.

1.3. Objectives of the study

1. Evaluate the demographic data, risk factors and disease burden of HS.
2. Evaluate the clinical presentation and concomitant diseases of HS.
3. Evaluate the efficacy of HS treatment.

1.4. Practical value and novelty of the study

Hidradenitis suppurativa still remains largely underrecognized. This study is a first of its kind in Lithuania, emphasizing the complexity and severe burden of hidradenitis suppurativa. Even though, HS currently is one of the most researched areas in dermatology, in-depth studies on clinical data are still lacking. The patients typically present with late disease, facing a diagnostic delay of 5 years. Which is associated with severe burden and high costs to national healthcare systems. This study will try to highlight that timely diagnosis, and proper treatment can improve the quality of life of such patients and stop disease progression. However, currently available treatment options have limited efficacy.

This study also offers valuable insights into the associations between demographic factors, comorbidities, and disease severity, which can assist clinicians in more accurately identifying individuals at higher risk of HS. By exploring the connections between smoking, obesity, family history of inflammatory diseases, and the progression and severity of symptoms, this research enhances our understanding of HS risk factors. These findings could support the development HS diagnostic tools, enabling healthcare providers to prioritize early intervention for patients with HS and potentially reduce the impact of delayed diagnosis.

Furthermore, the study examines the real-world effectiveness of currently available treatments, such as adalimumab, providing data on patient outcomes that could guide clinical decision-making. By documenting the efficacy limitations of standard therapies and the mixed results seen with biologics, the research underscores the urgent need for early treatment, new treatment options and potentially combined therapeutic approaches, such as biologics paired with surgery. This aspect of the study adds a novel perspective on how HS can be managed more effectively, paving the way for future research into innovative treatments that could offer more robust and sustained benefits for patients.

1.5. Research hypotheses

1. HS develops in early adulthood, mostly in overweight patients who are smokers and has a severe negative impact on quality of life.
2. Patients face a diagnostic delay of multiple years, frequent misdiagnoses, delaying timely treatment.
3. HS has a varying clinical presentation with multiple clinical subtypes, typically presenting as inflammatory nodules, abscesses and draining fistulae in the apocrine sweat gland-bearing areas.
4. Adalimumab is an effective treatment for HS in about 50% of patients, highlighting the unmet need for more effective therapies.

2. LITERATURE REVIEW

2.1. Definition and epidemiology

Hidradenitis suppurativa (HS), also known as *acne inversa*, is a lifelong inflammatory skin disease characterized by the presence of deep-seated nodules, abscesses, draining tunnels, and fibrotic scars. These distressing lesions primarily manifest in areas where skin folds meet and where apocrine glands are abundant. The most commonly affected regions include the armpits, groin, perianal region, perineum, and inframammary area (1). The heterogeneous nature of HS has led to the identification of various stages and phenotypes within the disease spectrum (2). HS typically begins after puberty, with the most common age of onset being between 20 and 29 years (10). However, HS can affect individuals of all ages, including children and older adults (11).

The global prevalence of HS in Western countries varies significantly, ranging from 0.053% to 4.1% (12,13). Evidence on the prevalence of HS comes from numerous studies conducted in different populations, using varied diagnostic methods and over different time periods (14). The highest prevalence of 4.1% was found in Denmark, among patients visiting a Sexually Transmitted Disease Clinic in Copenhagen (13). The prevalence may be relatively overestimated due to a potential bias from the age of participants, primarily those in their 30s and 40s (15). A recent comprehensive meta-regression analysis, encompassing 16 quantitatively assessed studies from the United States, Western Europe, Scandinavia, and Australia, identified an overall HS prevalence of 0.4% (16). In the paediatric population, the estimated prevalence of HS in the US is 0.028% (17). Given the frequent under-reporting and misdiagnosis of HS, the epidemiological data likely underestimates the true prevalence of the condition (14). Epidemiological data from other continents are relatively limited. A population-based study conducted nationwide in South Korea indicated a 10-year prevalence rate of HS of 0.06%, based on the National Health Insurance database (18). A population survey in Brazil identified an overall prevalence of 0.41% (19). The estimated prevalence of HS in Australia is 0.67%, determined through a validated HS screening questionnaire in a cross-sectional study (20). In the United States, the prevalence of HS varies among different ethnic groups. African Americans tend to have the highest prevalence, followed by Caucasians, while Hispanics and Asians have lower prevalence rates (21). Within Asian populations, individuals with darker skin tones are disproportionately affected compared to those with lighter skin (22,23).

2.2. Genetics

Because of the complexity and genetic variability associated with HS, it can be divided into three distinct categories: sporadic, familial, and syndromic (24). The initial pedigree studies revealed that familial occurrence was reported by only 30 to 40% of individuals with HS, indicating a limited influence of genetics as a risk factor in the development of the disease (25). Nonetheless, recent twin studies have shown a significant heritability rate in HS, ranging from 77% to 80%, suggesting that genetic factors may have a more substantial impact on the condition than previously believed (26,27). In 2006, Min Gao *et al*. identified the region 1p21.1–1q25.3 as a potential HS locus, which represented the first causative gene linked to the condition and laid the groundwork for further understanding the molecular mechanisms involved in this disease (28). In 2011, Min Gao *et al*. provided further confirmation of the NCSTN gene's role in HS (29). Following this, numerous researchers have provided evidence supporting the notion of a monogenic origin for familial HS, primarily concentrating on loci that encode proteins in the γ-secretase complex (GSC) (30). The human GSC is a multi-subunit, intramembrane-cleaving protease that consists of four distinct subunit domains: Nicastrin (NCSTN), Presenilin Enhancer 2 (PEN2), Presenilin 1 (PSEN1) or PSEN2, and Anterior Pharynx Defective (APH) 1A or B (31). Nicastrin, the largest subunit of the GSC, is crucial for substrate recruitment, assembly of the transmembrane domain, and GSC stability and maturation (32). Research efforts have focused on sequencing the essential subunits of γ-secretase in HS patient cohorts, uncovering a total of 20 reported mutations in NCSTN (33). The literature shows that the prevalence of HS is three times greater in African Americans compared to Caucasians, and the γ-secretase mutation linked to autosomal dominant HS is more frequently found in African American patients, indicating a potential genetic cause (34,35). Individuals of African descent might have an anatomical predisposition to HS, characterized by a higher number of prominent and active apocrine glands than those of Caucasian descent (21). HS can also be inherited as a polygenic condition that encompasses changes in genes related to epidermal homeostasis or those involved in both innate and adaptive immune system functions (36).

2.3. Immune system dysregulation

The development of HS is influenced by a combination of factors, including genetic predispositions, environmental factors (such as smoking and obesity), hormonal factors, and immune dysfunction (37). There is a growing

understanding that the interaction between endogenous and exogenous factors contributes to the activation of the innate immune system, primarily leading to inflammation around the hair follicles (38). This inflammatory response causes hyperkeratosis and hyperplasia of the follicular epithelium, particularly in the infundibulum. Consequently, follicular occlusion occurs, resulting in the blockage of the follicle (39). Subsequent dilation and rupture of the hair follicle triggers a strong inflammatory immune response, characterised by recruitment of various immune cells including neutrophils, macrophages, B-cells, Th1 and Th17 cells into the affected skin areas. This immune cell activity leads to the formation of inflammatory nodules or abscesses (37). During this immune response, proinflammatory cytokines such as tumour necrosis factor-alpha (TNF-α), interleukin (IL)-1β, IL-6, CXC chemokine ligand (CXCL)/IL-8, IL-12p70, IL-23p40, IL-17A, and IL-36 are produced (37,40–43). These cytokines play a significant role in the immune dysregulation seen in both acute and chronic stages of HS (44). The presence of these proinflammatory cytokines, particularly those associated with the Th17 immune response, is considered to be a prominent feature of HS (37,40–43). Recent research has provided additional evidence supporting an autoinflammatory mechanism in HS. One notable finding is the increased formation of neutrophil extracellular traps (NETs) observed in HS skin. The immune reactions triggered by neutrophils and NET-related antigens have been linked to heightened immune dysregulation and inflammation in the disease (45). As the inflammatory process progresses, tissue scarring becomes more prominent (46–49). The development of scarring and sinus tracts has been associated with the presence of metalloproteinase-2, tumour growth factor (TGF)-β, and intercellular adhesion molecule (ICAM)-1. Additionally, it is speculated that specific components of the microbiome may enhance TGF-β and ICAM-1 signalling, contributing to these processes (46,47).

2.4. Risk factors

2.4.1. Smoking

One of the most important environmental factors in HS pathophysiology is smoking, as around 90% of individuals diagnosed with HS are either current or former smokers, and smoking is believed to contribute to the development and progression of the disease through various mechanisms. One such mechanism involves the pro-inflammatory role of polycyclic hydrocarbons, which are produced as a result of cigarette combustion (50).

Nicotine may also impact the propagation of bacteria and the formation of biofilms in various ways. For instance, it can promote the initial attachment and accumulation of these microorganisms, modify the anti-inflammatory lipid film on the skin, and facilitate the colonisation of pathogenic bacteria in the affected area (51–55). HS is considered to be more of a dysbiosis rather than an infection, as early lesions exhibit a reduction in regulatory T-cells and the absence of biofilms and bacteria is noticeable (56). In fact, bacterial colonisation and the formation of biofilms are observed as late-stage events in HS, whereas early lesions exhibit an absence of bacteria and lack a normal biofilm structure (57–61). Additionally, nicotine interacts with nicotinic acetylcholine receptors (nAChRs) in the pilosebaceous unit, leading to epidermal hyperplasia and subsequent infundibular occlusion (62). Nicotine also stimulates neutrophil chemotaxis, promotes the release of TNF-alpha from keratinocytes, and triggers IL-10 production by monocytes (63–65).

A population-based cohort study involving 6,230,189 participants revealed that quitting smoking and maintaining a smoke-free status were linked to a decreased risk of developing HS compared to those who continued smoking. Conversely, both resuming smoking and starting to smoke may have a similarly harmful impact on the development of HS as ongoing smoking (66). Nonetheless, *Saleem et al.* emphasized that the causal link between cigarette smoking and HS remains uncertain, as it would require 3300 smokers to result in one new case of HS attributed to tobacco use (67). Conversely, cigarette smoking may also be a result of the disease, as patients might smoke to alleviate anxiety and depression, which are often linked to HS (68,69).

2.4.2. Obesity

There is a positive association between increased body mass index (BMI) and the presence and severity of HS (70,71). Approximately 50% of individuals with HS are classified as obese, and around 40% of HS patients show signs of metabolic syndrome. A meta-analysis revealed that patients with HS had a 3.5 times greater probability of being obese than individuals in the control group (72). In a retrospective cohort study of patients with HS, it was found that BMI was notably elevated compared to matched controls, with an average BMI among patients with HS being 31.51 kg/m^2 (73). Obesity is thought to play a role in the pathogenesis of HS through mechanisms such as subclinical inflammation, metabolic alterations, and heightened friction in skin folds (74). The higher count of skin folds in individuals with excess weight, can foster follicular blockage and elevate mechanical friction that could initiate the rupture of enlarged follicles (75). Obesity induces a mild

inflammatory state within the body, substantiated by elevated levels of circulating proinflammatory cytokines observed in these individuals (76). The heightened quantity of adipose cells, which function as endocrine tissues capable of releasing inflammatory cytokines, is viewed as a contributing factor to the persistent inflammatory state observed in HS (77).

At the molecular level, HS and obesity both are marked by a chronic, low-grade proinflammatory state with a similar cytokine profile (78,79). Excess fat accumulation, especially in visceral depots, is linked to adipose tissue dysfunction, which is characterized by increased infiltration of proinflammatory immune cells and upregulation of various proatherogenic and diabetogenic adipocytokines, including IL-1β, IL-6, TNF-β, visfatin, and resistin (80). The increased systemic levels of these adipocytokines are positively correlated with visceral fat mass and serve as the molecular connection between adipose tissue, insulin resistance, and the cardiometabolic complications associated with obesity (81). HS shares a similar adipokine profile with metabolic syndrome, characterized by elevated levels of leptin, resistin, and visfatin, along with reduced adiponectin levels (78).

A higher BMI is an independent indicator of more severe, treatment-resistant HS and is associated with a lower quality of life in individuals with HS compared to those with a lower BMI (82–85). Moreover, obesity is linked to a poor response to treatment, with a higher BMI serving as a negative predictor of the effectiveness of adalimumab therapy (86,87). In contrast, being overweight or obese does not appear to adversely affect the treatment response to the combination of antibacterial agents such as clindamycin and rifampicin (88).

2.5. Diagnosis and clinical features

In the absence of a diagnostic biomarker, HS is diagnosed clinically using three main criteria. These criteria include the presence of inflammatory nodules, abscesses, and tunnels (fistulas) in the skin. Secondly, diagnosis is based on typical anatomical locations such as the axillae, inframammary area, inguinal region, perineum, and buttocks. Finally, the chronicity and recurrence of the lesions are also considered in the diagnostic process. HS diagnosis can be made if there are at least two recurrent episodes of purulent lesions in the typical anatomical locations within 6 months (89). The majority of patients present with more than one lesion during the time of diagnosis (90). These lesions typically bring about sensations of discomfort, itching, and pain (91,92). A significant number of patients undergo prodromal pain symptoms (93). Various factors such as heat, sweating, physical exertion, shaving, and

friction intensify symptoms (94,95). The standard pattern involves acute flare-ups followed by phases of dormancy (93). The lesions may be associated with foul-smelling discharge due to suppuration and can cause significant pain, leading to a marked decline in the patients' quality of life (96). Diagnosis of HS relies primarily on patient history and physical examination, which is considered the gold standard (97). However, recent advancements in ultrasound imaging have exposed the limitations of physical examination and manual palpation in accurately assessing HS, emphasizing the need for alternative diagnostic approaches (98–101). Ultrasound imaging often influences changes in clinical management and treatment strategies (102).

Individuals affected by HS encounter a substantial time lapse, estimated at 7.2 years globally, between the onset of the disease and its diagnosis. This delay is influenced by various factors, such as doctors' lack of recognition, the embarrassment and stigma attached to the condition, and socioeconomic obstacles (5–7). Of significant note, *Kokolakis et al*. revealed there was a positive correlation between the extent of diagnostic delay and the severity of HS at the point of diagnosis. As a result, patients who experienced delays in diagnosis exhibited a notably higher Hurley stage at the time of diagnosis, in contrast to those who were diagnosed earlier. Moreover, individuals who faced delayed HS diagnosis presented a greater number of comorbidities compared to those who received an earlier diagnosis (103).

In clinical settings, HS has typically been classified using the Hurley staging system. Hurley Stage I is defined by the development of abscesses without any associated tracts or scars. Hurley Stage II features recurring abscesses along with sinus tracts and scarring, presenting as single or multiple lesions that are widely dispersed. Hurley Stage III is marked by widespread involvement, with numerous interconnected tracts and abscesses throughout the entire affected region (89).

HS manifests through a variety of symptoms, including inflammatory nodules, purulent or serosanguineous abscesses, sinus tracts, comedones, and scarring (104). Painful abscesses, which can produce a malodorous discharge, often arise from nodules and typically persist for about 6.9 days. While these abscesses may sometimes resolve without discharging, the majority of patients (62%) report experiencing persistent, painful nodules or abscesses (105). Patients typically develop a median of two new nodules or abscesses per month (99). Sinus tracts often form due to recurrent, multiple nodules and contribute to the development of HS sinus networks in advanced stages. While they can be detected through manual palpation, they are frequently not visible through clinical assessment alone (99,106).

This circumstance has prompted initiatives to create efficient screening methods for HS. Inquiring whether a patient has experienced episodes of abscesses in the past six months, with a minimum of two occurrences in any of the following five areas: armpits, groin, genital, inframammary area, and unspecified locations such as perianal, neck, and abdomen, can result in a sensitivity of 90%, specificity of 97%, and a positive predictive value of 96% (107). Additionally, an alternative option is a visual questionnaire featuring illustrative images of the prevailing HS lesions (108). Ultrasound imaging has proven valuable in enhancing the understanding of the morphology and depth of lesional HS and shall be used more frequently in the future as a valuable diagnostic tool (109). The sonographic scoring system for HS (SOS-HS) integrates ultrasound findings with Hurley staging to enhance the diagnosis and staging of HS (98). Ultrasound shows greater sensitivity compared to clinical scoring in HS. For example, it has the capacity to illustrate subclinical accumulation of fluids, elevated dermal thickness, and follicular expansion during the initial phases of HS, as well as the progression of sinus tracts as the disease advances (100,110). Wortsman et al., during the development of the SOS-HS criteria, revealed that clinical scores alone consistently underestimated disease severity. The SOS-HS employs ultrasound to detect specific lesion features (*Figure 1*) and determines the stage of HS by quantifying these features (98). Furthermore, the accuracy of HS scoring indices is crucial for determining the appropriate therapeutic strategy for patients with HS, as treatment approaches vary based on the severity of the disease (98,111). Moreover, colour Doppler can assess the vascularization of typical HS lesions, with increased vascular flow correlating with higher levels of localized pain (112).

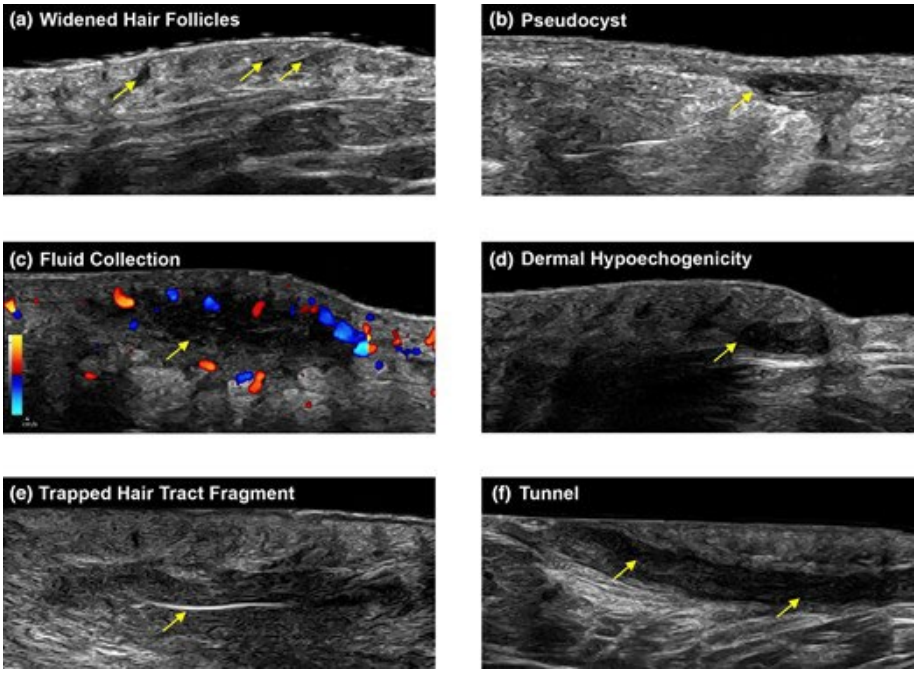


Figure 1. Lesional features according to the sonographic scoring for hidradenitis suppurativa (SOS-HS) criteria. Arrows highlight the feature(s) of interest. The widening of a hair follicle (a) can cause it to rupture, allowing fluid to enter the resulting hypoechoic cavity, leading to the formation of a pseudocyst (b). As more fluid accumulates in the cavity, an anechoic fluid collection (c) can form. Colour Doppler sonography detects a high concentration of vessels at the periphery, indicating inflammation. Dermal hypoechogenicity (d) occurs due to edema. Ectopic hair tract fragments (e) may provide a scaffold for the formation of tunnels (f), which are horizontal, permeable channels that connect multiple suppurative cavities or skin openings. *Br J Dermatol*, Volume 188, Issue 5, May 2023, Pages 591–600, https://doi.org/10.1093/bjd/ljad028

Ultrasound is also employed to assess the severity of tunnels, which appear as hypoechoic or anechoic band-like structures linked to the base of widened hair follicles. These tunnels are typically evaluated and classified according to their location and morphological pattern (Types A–D; *Figure 2*) (113). Since tunnels are linked to more advanced disease and may limit the effectiveness of medical treatments, their early detection through ultrasound

can provide a critical therapeutic window. This helps physicians select the appropriate intervention and predict the likely response to treatment (114).

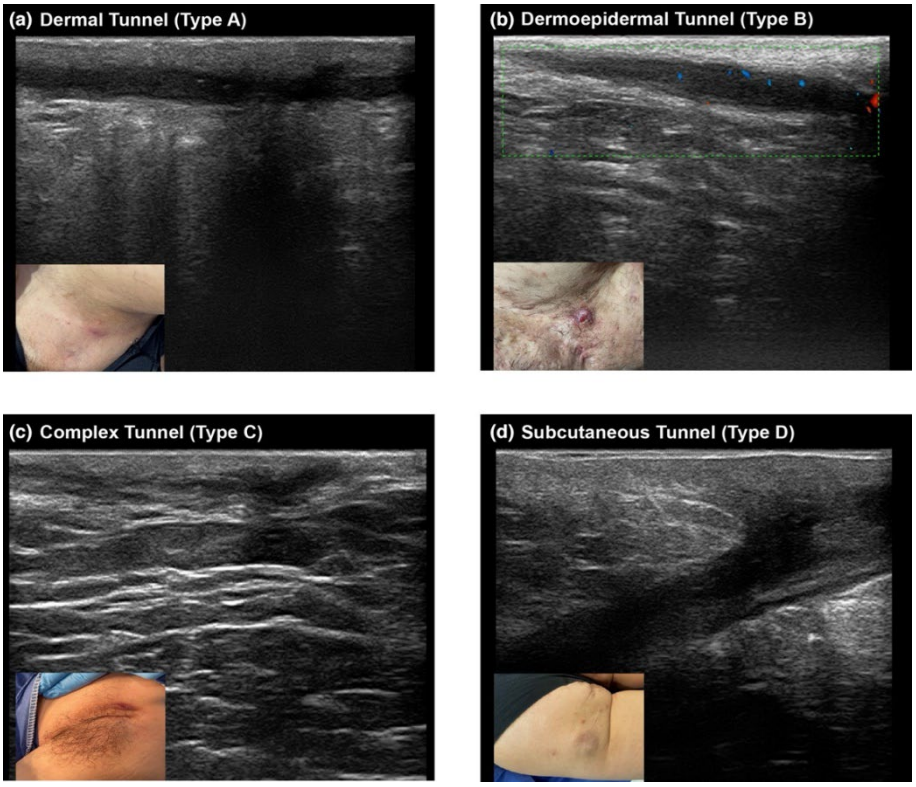


Figure 2. Tunnel patterns based on morphology and location. Ultrasound imaging used to assess tunnels severity as type A, B, C, or D. Dermal tunnels (a) are band-shaped structures that remain confined to the dermis, without extending into the epidermal or subcutaneous layers. Dermoepidermal tunnels establish a connection with the epidermis (b). Complex tunnels (c) comprise multiple interconnected band-like structures that spread over a broad area. Subcutaneous tunnels (d) extend deeply into the subcutaneous fat, reaching towards muscle. *Br J Dermatol*, Volume 188, Issue 5, May 2023, Pages 591–600, https://doi.org/10.1093/bjd/ljad028

Differences in clinical presentation between sexes have been noted. Women are more likely to have HS lesions on the front of the body, groin, and breasts, while men more commonly experience involvement in the gluteal area (3). Atypical sites, such as the retroauricular areas and chest, along with associated pilonidal cysts and acne, are more commonly reported in men. However, axillary lesions are similarly distributed between both sexes (70).

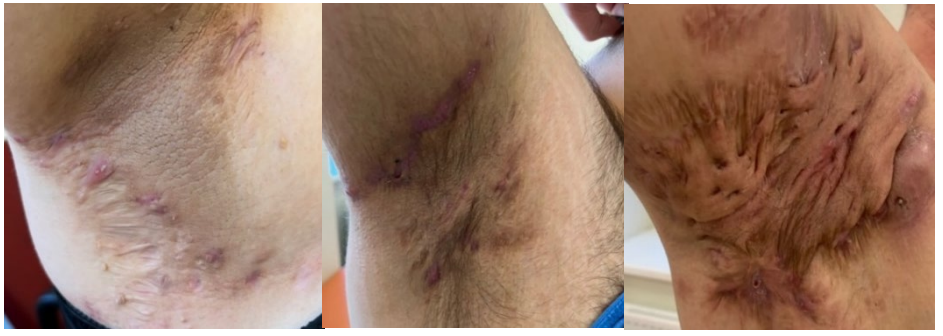


Figure 3. HS clinical presentation in the axillar region according to Hurley I with multiple inflammatory nodules, Hurley II with scarring and fistula formation, and Hurley III, affecting the whole axillary region with sinus tract and scar formation. VUHSK Centre of Dermatovenereology.

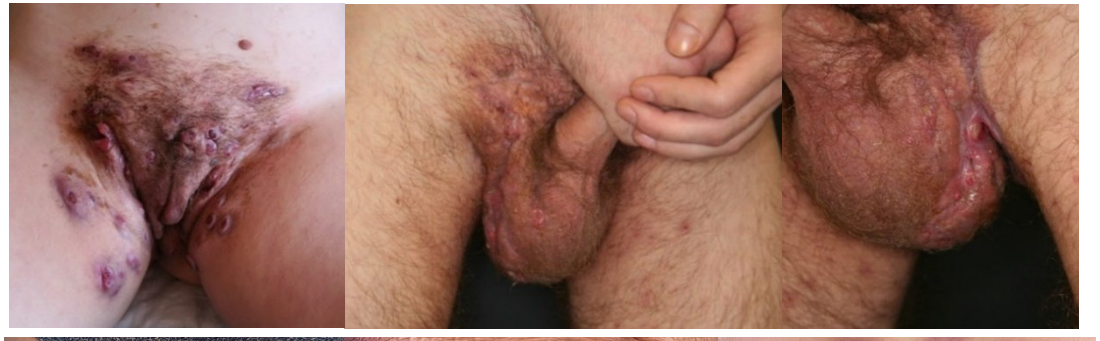


Figure 4. HS clinical presentation in the genital area, Hurley III. Multiple inflammatory nodules and abscesses interconnected with sinus tracts. VUHSK Centre of Dermatovenereology.

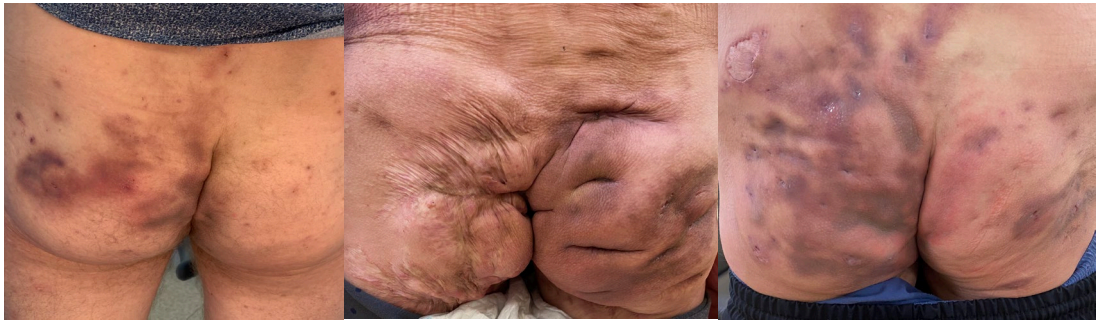


Figure 5. HS clinical presentation in the buttock and perineal area of Hurley III patients. Multiple inflammatory nodules and abscesses interconnected with sinus tracts. VUHSK Centre of Dermatovenereology.

2.6. Comorbid conditions

HS, being a chronic inflammatory disease, exemplifies the connection between skin-related conditions and comorbid systemic diseases, as they share common inflammatory pathways. Patients with HS tend to have a higher burden of comorbidities compared to the general population, and HS itself is independently associated with several specific comorbid conditions (such as polycystic ovary syndrome, diabetes mellitus, metabolic syndrome, inflammatory bowel disease, psychiatric disorders, lymphoma, etc.) (3,4).

Patients with HS often meet the criteria for metabolic syndrome, which includes obesity, hypertension dyslipidaemia, and diabetes mellitus (78). A cross-sectional study examining the relationship between HS and metabolic syndrome reported pooled odds ratios (ORs) of 3.89 (95% CI, 1.90-7.98) for hospitalized HS patients and 2.08 (95% CI, 1.61-2.69) for non-hospitalized patients compared to healthy controls. Among the conditions in metabolic syndrome, obesity showed the strongest association with HS, reflecting the highest pooled ORs (115). A systematic review and meta-analysis involving 6,174 HS patients and 24,993 controls found a significant link between HS and hypertriglyceridemia (OR 1.67, 95% CI 1.14-2.47) and low high-density lipoprotein (OR 2.48, 95% CI 1.49-4.16) (72). Additionally, a UK population-based study reported a strong association between HS and type 2 diabetes mellitus (OR 3.39, 95% CI 3.09-3.71) (116). Furthermore, insulin resistance was more prevalent in HS patients, occurring in 43.4% of cases compared to 16.4% in controls (117).

A Danish population-based cohort study examined cardiovascular (CV) risk in HS patients. The findings revealed a significantly higher risk of CV outcomes, including myocardial infarction (adjusted incidence rate ratio 1.57, 95% CI 1.14-2.17), ischemic stroke (1.33, 95% CI 1.01-1.76), CV-related death (1.95, 95% CI 1.42-2.67), major adverse CV events, and all-cause mortality. The study noted that patients with HS faced a greater risk of CVD-related death compared to those with severe psoriasis (118). Additionally, individuals with HS exhibited a higher prevalence of subclinical atherosclerosis, tachycardia, and risk factors for adverse CV outcomes (119,120). Elevated serum levels of C-reactive protein and TNF-α are associated with both atherosclerosis and HS (118).

Psychiatric comorbidities in HS patients may arise from factors such as malodour, pain, and other symptoms, leading to a considerable psychological burden. Research indicates that individuals with HS are at a higher risk of mental health disorders, including depression, anxiety, and schizophrenia, compared to those with conditions like psoriasis or melanocytic nevi (121). A

meta-analysis encompassing 28 studies on depression and 12 studies on anxiety in patients with HS found that the overall prevalence of depression (21%) and anxiety (12%) was higher in this population compared to the general population. Nine case-control studies identified a significant association between HS and depression (OR 1.99, 95% CI 1.63-2.43) as well as between HS and anxiety (OR 1.97, 95% CI 1.65-2.35) (122). Following an increase in psychological comorbidities, a retrospective cohort study utilizing data from the Danish national registry found that patients with HS have 2.42 times the odds of suicide (95% CI 1.07-5.45) compared to individuals without HS (123).

Squamous cell carcinoma (SCC) occurs in 4.6% of HS patients (124,125), and represents the most dreaded complication, with mortality rate reaching 40%, primarily due to metastatic disease and sepsis (126,127). There is a lack of extensive data in the literature regarding SCC formation within HS lesions as well as screening and management of these cases (126,127). Individuals with chronic inflammation are predisposed to developing proliferative diseases like SCC (124), with key risk factors being male gender, prolonged disease duration, perianal lesion localization, and Hurley stage III (125–127). Additionally, the role of the human papilloma virus (HPV) was noted in one-third of SCC cases in preexisting HS lesions (127). Aggressive tumour treatment, including wide excision, is recommended, with radiotherapy in specific cases (125).

2.7. Quality of Life

It is well established that HS has a considerable negative impact on a patient's quality of life. The limited availability of effective therapies and frequent exacerbations present a difficult obstacle in treatment, resulting in a detrimental effect on the quality of life (8,9). In addition, given the painful nature of the lesions, their occurrence in sensitive areas, the presence of drainage, unpleasant odour, and resulting scarring, HS can significantly impact a person's psychological and social well-being.

In a cross-sectional epidemiological study involving 145 Irish patients with HS, 91.7% of respondents reported feelings of embarrassment and self-consciousness regarding their skin. In addition, 82.8% of patients expressed difficulty participating in social activities, while 76.4% reported challenges engaging in sports. A significant number of respondents (21.3%) were unemployed, and 9.4% were unable to work due to temporary or permanent disability from illness (128). A Polish study involving 103 patients with HS found that 41.7% experienced pruritus, while 77.5% reported pain. The most

significant symptom of HS was pain, followed by exudation, pruritus, concerns about appearance, and malodour (91). A cross-sectional analysis conducted in the United States revealed that the prevalence of substance use disorder was higher among patients with HS (4%) than in those without the condition (2%). The most frequently used substances included alcohol, followed by opioids and cannabis (129). As HS significantly compromises the quality of a patient's life, it is crucial that diagnosis should be identified without delays and decrease the burden of the disease on patients and the healthcare system (1).

2.8. Treatment

The management of HS depends on the severity of the condition and may involve a combination of topical and systemic antibiotics, hormone therapy, immune-modulating medications, and surgical interventions (1). Surgically addressing locally recurring lesions is a suitable approach, while medical treatment, either as a standalone therapy or in conjunction with surgery, is more appropriate for wide spread lesions (89). Nevertheless, achieving consistent symptom control and complete resolution of lesions is challenging and requires patients to undergo multiple extended treatment regimens of systemic antibiotics, potentially resulting in escalated healthcare expenses (130,131).

2.8.1. Topical treatment

Chlorhexidine wash, pyrithione zinc shampoo, bleach baths, and benzoyl peroxide are commonly utilized in the management of HS as supplementary treatments, appreciated for their anti-inflammatory characteristics and their ability to reduce the development of antibacterial resistance (89,132,133). The selection of a particular agent is frequently based on empirical evidence and expert recommendations (134). Treatment should also encompass efforts to prevent skin trauma. This can be achieved by discontinuing the use of tight or synthetic clothing, refraining from harsh products or abrasive cleaning tools like loofahs, washcloths, or brushes, and avoiding adhesive dressings (135). The choice of wound dressings for patients with HS depends on various factors, including lesion location, disease extent, lesion morphology (such as ulcers, tunneling sinuses, or abscesses), exudate volume, dressing cost, and availability. For cavities and tunneling, dressings should adequately pack the area and absorb fluids, whereas superficial lesions may only require plain absorptive dressings. Depending on the exudate level,

options range from superabsorbent dressings to foams, hydrofibers, and calcium alginates. Dressings should remain securely in place to prevent friction and conform to curved areas like skin folds. Atraumatic adhesives, such as silicone and nonadherent wound contact layers, help reduce skin damage and minimize pain during dressing changes (136).

In cases of mild HS (Hurley stage I–II), topical antibiotics, particularly 1% clindamycin, are the first-line treatment (137). The suggested dosing regimen is twice daily for a duration of 3 months. If there is no clinical improvement after this period, alternative treatment options should be explored (138). When using topical clindamycin, superficial lesions (such as pustules and folliculitis) exhibited a better response compared to deeper lesions (like nodules and abscesses). Given these observations, it is advised to use 1% clindamycin gel for HS lesions classified as Hurley stage I or for cases with superficial lesions during periods of exacerbation (139,140). In addition, resorcinol 15% cream, known for its antiseptic properties and keratolytic effects, can be prescribed (137).

2.8.2. Pain management

Effective pain management is a crucial aspect of addressing HS, as the pain associated with this condition can stem from both inflammatory and non-inflammatory sources. The sources of pain may include scarring (resulting in tensile pain), keloids, open ulcerations, abscesses, sinus tracts, frictional discomfort, anal fissures, lymphedema, and arthritis. Depending on the severity of the disease and the nature of the pain, various approaches can be considered. These may involve topical agents such as lidocaine and anti-inflammatories, systemic nonsteroidal anti-inflammatories, acetaminophen, atypical anticonvulsants like gabapentin or pregabalin, and serotonin-norepinephrine reuptake inhibitors. Duloxetine can be particularly beneficial in cases where there is comorbid depression (141,142).

2.8.3. Systemic antibiotics

If there are numerous lesions and frequent flare-ups, systemic tetracyclines such as doxycycline may be considered for treatment (143). A single antibiotic from the same class can be utilized for up to 12 weeks (144). In patients with Hurley stage II/III and multiple active lesions, systemic clindamycin and rifampicin (at a dosage of 300 mg twice daily) for a typical duration of 10 weeks are recommended (145,146). An alternative approach may involve a triple therapy regimen of rifampicin (10 mg/kg once daily),

moxifloxacin (400 mg once daily), and metronidazole (500 mg three times daily) for a period of up to 12 weeks, with metronidazole discontinued after 6 weeks (144). Dapsone may also be effective in mild to moderate HS cases, particularly in the early stages of new lesion formation, where neutrophils play a key role (147,148).

2.8.4. Other systemic therapies

Metformin

Antidiabetic drugs like metformin can inhibit the proliferation of proinflammatory cytokines and improve insulin sensitivity, leading to reduced inflammation and potentially affecting HS-related gene expression due to its anti-androgenic properties. Furthermore, metformin offers additional benefits, such as modest weight loss and improvements in metabolic parameters, which may support its early use in patients with HS and obesity, a known aggravating factor for the condition (117,149,150).

Zinc

Zinc supplementation is widely recommended as a non-pharmacologic treatment for HS (151–154). Numerous studies indicate that patients with HS often have low zinc levels (155–157). Multiple retrospective studies have documented the effectiveness of oral zinc treatment in alleviating symptoms and reducing inflammatory skin lesions in HS (137,154). Research indicates a dose-response relationship with zinc treatment, as reducing the dosage from 90 mg to 60 mg or lower has been associated with relapsing lesions in HS patients who were previously in remission (158).

Retinoids

Several studies on oral retinoids, such as acitretin, isotretinoin, and alitretinoin, have reported notable clinical improvement. However, their effectiveness in HS is inconsistent, with some studies reporting lower response rates and higher rates of recurrence. These medications may be more helpful in mild cases and are generally considered second- or third-line treatments for HS (159).

Semaglutide

Individuals with HS who are overweight or obese may find benefits from using semaglutide, a glucagon-like peptide 1 (GLP-1) receptor agonist, as an adjunctive treatment. A nonrandomized, observational, retrospective

study analysed data from 30 patients between June 2020 and March 2023. The findings indicated that semaglutide-assisted weight loss was linked to improvements in dermatology-related quality of life, as measured by the Dermatology Life Quality Index (DLQI), as well as a reduction in the frequency of HS flare-ups. The average duration of semaglutide therapy was 8.2 months, with a mean dose of 0.8 mg administered once per week. Results showed a reduction in the mean body mass index (BMI), dropping from 43.1 to 41.5 kg/m², the average weight of patients decreased from 117.7 kg to 111.6 kg, with 33.33% of participants losing at least 10 kg over the course of the study. The frequency of patient-reported flares also declined, from once every 8.5 weeks before treatment to once every 12 weeks after starting semaglutide. The mean DLQI score improved, decreasing from 13 out of 30 to 9 out of 30. A reduction of 4 points or more in the DLQI, was seen in one-third of the patients. Haemoglobin A1c levels also significantly decreased, from 39.3 to 36.6 (160).

2.8.5. Biologics

Guidelines for HS suggest that biologic therapy should be considered as an alternative if conventional treatments prove ineffective (144).

TNF-α inhibitors

Specifically, the monoclonal antibody adalimumab, designed to target tumour necrosis factor (TNF)-α, demonstrated effectiveness compared to a placebo in two phase III trials, and for several years was the only biologic drug approved by the Food and Drug Administration (FDA) for the treatment of HS (161). The response rate for this treatment reaches about 60% of patients based on the Hidradenitis Suppurativa Clinical Response (HiSCR), defined as a 50% reduction in the count of abscesses and inflammatory nodules with no increase in abscesses or draining fistula count; however, extensive long-term data from routine clinical practice for HS are still lacking (161–163). An example of a successfully treated HS patient with adalimumab is shown in ***Figure 6.***

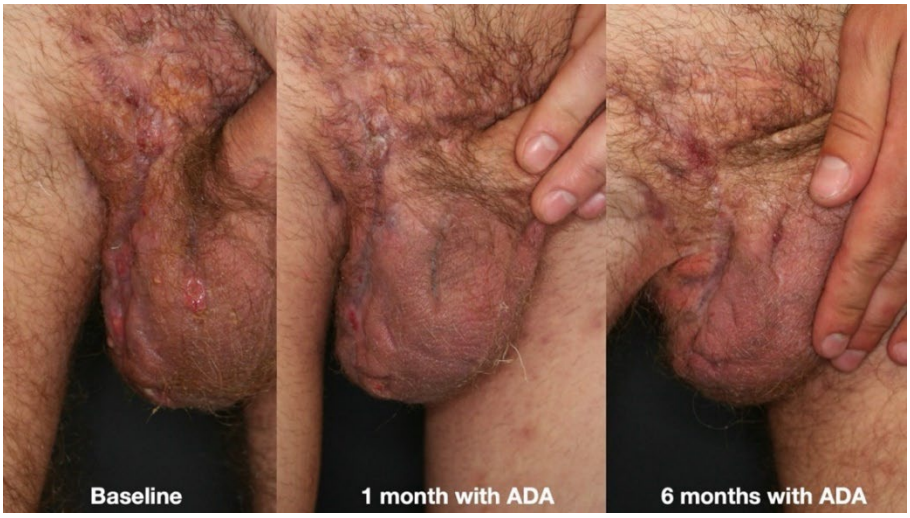


Figure 6. Adalimumab efficacy in HS management. Inflammatory nodule, draining fistula regression can be observed over 6 months of treatment. VUHSK Centre of Dermatovenereology archive.

Infliximab, a biologic TNF-α inhibitor administered intravenously, has also demonstrated efficacy and may be a viable option for individuals with moderate-to-severe HS (164). In a clinical trial, 26% of patients in the treatment group experienced a 50% or greater improvement, compared to only 5% in the placebo group; however, there is a lack of prospective studies regarding infliximab therapy (164,165). Although new treatment methods have emerged, adalimumab remains the preferred biological agent in treatment algorithms, making it essential to evaluate its effectiveness (144).

IL-17 inhibitors

Secukinumab, an anti-interleukin 17A medication, has recently been approved for adults with active moderate-to-severe HS who have not responded adequately to conventional HS therapy (166). The SUNSHINE and SUNRISE trials were identical, multicenter, randomized, placebo-controlled, double-blind phase 3 studies which evaluated the efficacy of secukinumab. Participants were randomly assigned (1:1:1) to receive either 300 mg of secukinumab subcutaneously every 2 or 4 weeks, or a placebo. The primary endpoint was the proportion of patients achieving a HiSCR by week 16. In SUNSHINE trial, significantly more patients receiving secukinumab every 2 weeks (45%) achieved a clinical response compared to the placebo group (34%). However, the every 4 weeks group showed no significant difference from the placebo. In the SUNRISE trial, both secukinumab groups (42% for

every 2 weeks and 46% for every 4 weeks) had significantly higher response rates than the placebo (31%). Responses were sustained through week 52. Headache was the most common adverse event, impacting 9-12% of patients in both trials, with no study-related deaths reported (167).

Brodalumab (BRO) is a monoclonal antibody that targets the IL-17RA subunit of the IL-17 receptor dimer, inhibiting the signalling of several IL-17 isoforms, primarily IL-17A, IL-17C, and IL-17F (159). In an open-label trial where patients received brodalumab at a dose of 210 mg every two weeks, all 10 participants achieved Hidradenitis Suppurativa Clinical Response (HiSCR), defined as a 50% reduction in abscesses and nodules without any increase in draining fistulae. Among these, five patients reached HiSCR75, while three achieved HiSCR100. The authors emphasized the swift reduction in tunnel drainage and ultrasonographic signs of inflammation, which are typically resistant to other treatments. However, during the maintenance phase, two patients with extensive gluteal tunnels experienced a recurrence of drainage one week after each dose administration (168). A subsequent open-label trial with 10 patients on a weekly dosing regimen showed enhanced efficacy, with 80% of patients achieving HiSCR75 and 50% reaching HiSCR100. Unlike the biweekly dosing, the weekly regimen did not result in cyclical patterns of disease suppression or recurrence of tunnel drainage (169).

Bimekizumab is a humanized monoclonal antibody that targets IL-17A and IL-17F (170). The efficacy and safety of bimekizumab were assessed in patients with moderate-to-severe HS in two randomized, double-blind, multicentre phase 3 trials BE HEARD I and BE HEARD II, over a 48-week period. The primary outcome at week 16 was met in the bimekizumab every 2 weeks group, showing higher responder rates compared to placebo in both trials. In BE HEARD I, 48% of patients responded versus 29% in the placebo group, while in BE HEARD II, 52% responded compared to 32% in placebo. Additionally, in BE HEARD II, HiSCR50 was achieved in the bimekizumab every 4 weeks group (54% vs. 32% with placebo). Responses were maintained or improved by week 48. Serious treatment-emergent adverse events occurred in 8% of patients in BE HEARD I and 5% in BE HEARD II receiving bimekizumab. Common adverse events included hidradenitis, with additional reports of coronavirus infection and diarrhoea in BE HEARD I, and oral candidiasis and headache in BE HEARD II. One death from congestive heart failure in BE HEARD I was deemed unrelated to bimekizumab by the investigator (171).

Sonelokimab is developed to specifically target inflammatory sites by blocking the IL-17A/A, IL-17A/F, and IL-17F/F dimers, allowing it to effectively access inflamed tissues. Encouraging 24-week results from a

global phase 2 MIRA trial were reported, which assessed the safety and efficacy of subcutaneously administered sonelokimab for treating adults with moderate to severe HS. Following maintenance treatment with sonelokimab 120 mg administered every 4 weeks, 56.9% of patients reached HiSCR75 by week 24. Complete resolution of inflammatory nodules and abscesses (AN 100) was observed in 31% of patients, while 49% achieved complete resolution of draining tunnels (DT 100). Furthermore, 24% of patients reached complete inflammatory remission (IHS4-100) by week 24 (172).

IL-36 inhibitor

Spesolimab is a monoclonal antibody (mAb) that targets the IL-36 receptor (IL-36R) and selectively blocks IL-36 signalling (173). A randomized, double-blind, placebo-controlled clinical trial investigated the effects of spesolimab in patients with moderate-to-severe HS. For the primary endpoint, the mean percentage change from baseline in total abscess and nodule (AN) count at week 12 was –38.8% for the spesolimab group compared to –34.7% for the placebo group, resulting in a difference of –4.1% favouring spesolimab. At week 12, the spesolimab group demonstrated a greater overall clinical improvement according to IHS4, with a difference of –13.9 compared to the placebo group. The most frequently reported adverse events included headache, nasopharyngitis, nausea, fatigue, injection site erythema, and injection site pain. No serious adverse events were reported among those receiving spesolimab (174).

IL-1 inhibitor

Lutikizumab is an innovative human dual-variable domain immunoglobulin that effectively inhibits both IL-1α and IL-1β simultaneously. The 16-week randomized, double-blind, multicentre, placebo-controlled study included 153 adult patients with moderate to severe HS who had previously not responded to anti-TNF therapy. Patients receiving lutikizumab at doses of 300 mg every other week or 300 mg weekly had higher response rates of 59.5% and 48.7%, respectively, compared to 35.0% in the placebo group for the primary endpoint of achieving HS Clinical Response (HiSCR 50) at week 16. The trial also demonstrated that patients receiving lutikizumab 300 mg weekly and 300 mg every other week experienced higher rates of reduced skin pain as measured by NRS30 and a higher level of HS clinical response HiSCR75, compared to the placebo group. However, lutikizumab 100 mg every other week did not show improved efficacy over placebo (175).

2.8.6. JAK inhibitors

Povorcitinib is an oral, small-molecule, that selectively targets Janus kinase (JAK)-1. Two proof-of-concept phase 2 studies indicated that povorcitinib was linked to better outcomes and was generally well tolerated in patients with moderate to severe HS (176). Placebo-controlled phase 2 study randomized patients with HS in a 1:1:1:1 ratio to receive povorcitinib at doses of 15, 45, or 75 mg, or a placebo. At week 16, povorcitinib significantly decreased the abscess and inflammatory nodule count from baseline (15 mg, -5.2; 45 mg, -6.9; 75 mg, -6.3) compared to placebo (-2.5). A greater proportion of patients treated with povorcitinib achieved HS Clinical Response at week 16 (15 mg, 48.1%; 45 mg, 44.2%; 75 mg, 45.3%,) compared to placebo (28.8%). Adverse events were reported in 60.0% of patients receiving povorcitinib and 65.4% of those on placebo (177).

Upadacitinib (UPA), an oral selective inhibitor of JAK-1, has the potential to reduce inflammation and enhance outcomes in patients with HS. In a phase 2, multicentre, randomized, placebo-controlled, double-blind study, adults with moderate-to-severe hidradenitis suppurativa were assigned in a 2:1 ratio to receive either UPA 30 mg (UPA30) once daily or a placebo. At week 12, a greater percentage of patients receiving UPA30 achieved at least a 50.0% reduction in abscess and nodule count without an increase in abscesses or draining fistulae (HiSCR), with results of 38.3% for UPA30 compared to 25.0% for placebo (178). The Phase 3 Step-Up HS study is currently ongoing, assessing the efficacy of upadacitinib in adults and adolescents with moderate to severe HS who have not responded to anti-TNF therapy or one approved non-anti-TNF inhibitor treatment for HS (179).

A 16-week randomized phase 2 trial found that the oral Bruton's tyrosine kinase (BTK) inhibitor, remibrutinib, was more effective than placebo in patients with moderate-to-severe HS. Participants were assigned to receive remibrutinib at doses of either 25 mg twice daily, 100 mg twice daily, or a placebo twice daily. At week 16, 72.7% of patients treated with remibrutinib 25 mg twice daily reached the HiSCR ≥50% endpoint, compared to 48.5% in the 100 mg group and 34.7% in the placebo group. HiSCR ≥75% was achieved by 42.4% of patients treated with remibrutinib 25 mg, 27.3% of those on the 100 mg dose, and 18.4% of patients in the placebo group. Remibrutinib also showed a greater impact on reducing inflammatory lesions, draining abscesses, and pain response. Most adverse events were mild to moderate, with only one serious case reported. The most common adverse events in the treatment groups were infections, primarily affecting the upper respiratory tract (180).

2.8.7. Surgery

Incision and drainage

Incision and drainage are often employed as a treatment option for acute cases involving tender, fluctuant abscesses with pus accumulation, providing quick pain relief. However, this approach does not address the underlying diseased tissue, resulting in only temporary relief and an almost 100% rate of recurrence (181–183). Once wide circumferential local anaesthesia is administered, an incision is made, and digital pressure is used to expel the purulent material. Any remaining contents can be flushed out with saline irrigation. Packing is generally unnecessary after draining HS lesions but may be considered if there is a possibility of an infectious abscess (184).

Deroofing

Deroofing is a cost-effective surgical technique suitable for treating Hurley stage II and III lesions. The procedure involves removing the "roof" of abscesses or sinus tracts to expose the base of the affected areas. Scissors or a metal probe is used to gently trace and identify all connecting tracts, which are then deroofed. The gelatinous proliferative sinus tissue is carefully extracted with a scalpel, curette, or moistened gauze (184–186). Preserving the base of the lesion allows epithelial cells from sweat glands and hair follicle remnants to promote rapid re-epithelialization, facilitating wound healing by secondary intention (187,188).

Deroofing is a minimally invasive procedure that can be performed using various methods, including blunt surgical scissors, carbon dioxide (CO2) laser, or an electrosurgery probe. In an open study by Vanderzee *et al*., 44 patients underwent deroofing on a total of 88 lesions, followed by healing by secondary intention. The majority of treated lesions were located in the groin (47%) and axillae (44%). The average healing time for defects, which had a mean length of 3 cm, was around 14 days. Recurrence was noted in 17% of the lesions after a median period of 4.6 months, while 83% showed no recurrence over a median follow-up of 34 months. Additionally, 90% of the patients expressed a willingness to recommend the deroofing procedure to others with HS (189).

Deroofing offers several benefits: it can be conducted under local anaesthesia, results in low morbidity, provides cosmetically acceptable outcomes, and helps prevent contractures (190). Deroofing is the main technique used to treat persistent nodules and sinus tracts in Hurley stage I or II. Potential complications of this procedure include bleeding after surgery, infection, and the formation of scars (191,192).

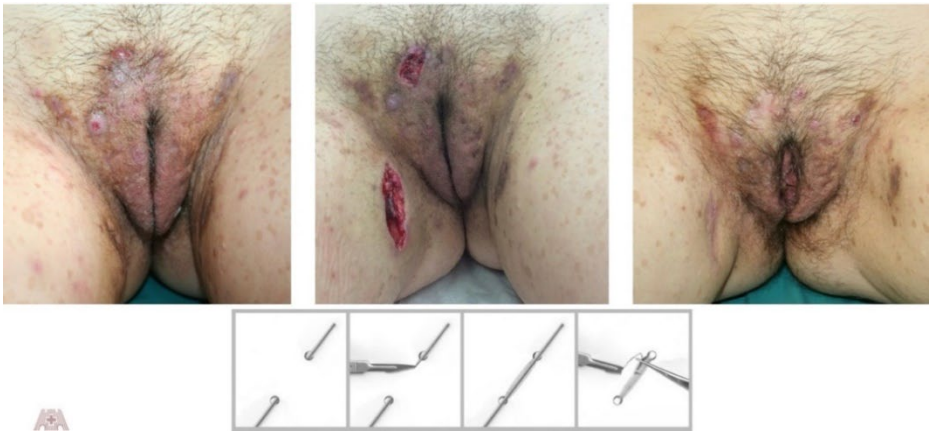


Figure 7. Deroofing procedure: before, immediately after and 4 months follow-up. VUHSK Centre of Dermatovenereology archive, photo courtesy of Milda Banienė.

Extensive surgery

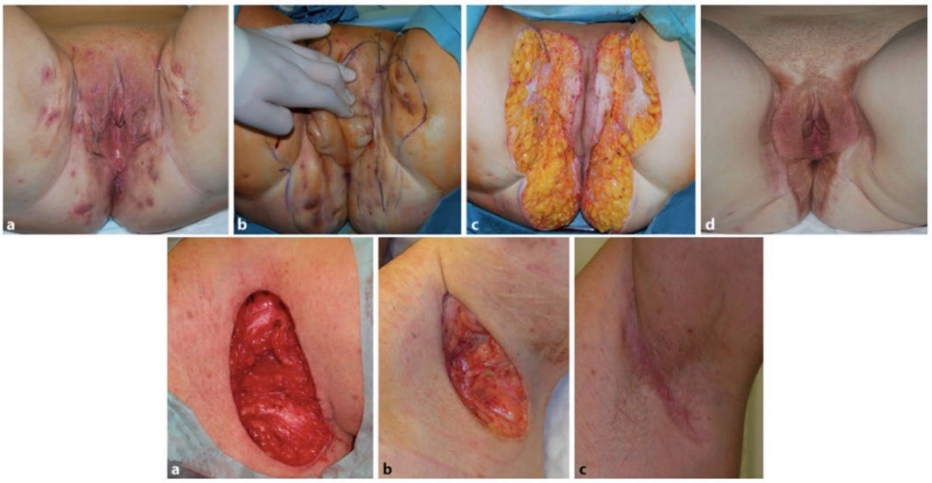
Excision is a more aggressive method focused on completely removing affected tissue. This procedure can vary in invasiveness: it may be limited, where individual diseased areas are removed along with a margin of healthy tissue, or wide, where a larger section containing all lesions is excised. In severe cases, a radical excision may be necessary to remove the entire area of a body region exhibiting disease activity (193).

Wide excision is recommended for severe cases that do not respond to medical treatment, especially when there is a risk of significant fibrosis or loss of tissue architecture. This procedure entails the surgical removal of lesions along with a margin of healthy tissue, which may include subcutaneous fat or a lateral margin of 1–2 cm of intertriginous skin (181). According to Alharbi et al., wide surgical excisions are characterized by the removal of diseased tissue along with a broad margin of up to 1 cm, extending down to the subcutaneous tissue until the fascia is reached (194).

The location and extent of sinus tracts, along with any fluid collections, can be visualized preoperatively using ultrasound or magnetic resonance imaging (98). Intraoperatively, dye mapping techniques such as methyl violet

and iodine starch can also be used to highlight the entire area of HS lesions, facilitating their identification and removal (195,196).

The research conducted by Shanmugam *et al*. showed that biologic therapy is associated with a more rapid decrease in disease activity, especially among patients who also underwent surgical intervention for HS (197). The study by Aarts *et al*. found that the combination of adalimumab and surgery led to significantly improved clinical outcomes and a better quality of life compared to adalimumab alone, along with higher levels of patient satisfaction. After 12 months of treatment, the surgery group showed a significantly greater reduction in IHS4 scores compared to the monotherapy group, with a mean decrease of -9.1 versus -7.8. Additionally, the surgery group experienced a more pronounced decrease in DLQI scores post-treatment compared to the monotherapy group, with a mean reduction of -8.2 compared to -4 (198).



Scholl L., Hessam S, Reitenbach S, Bechara F. G. Operative Behandlungsoptionen bei Hidradenitis suppurativa/ Acne inversa. Hautarzt. 2018; 69:149–161.

Figure 8. Extensive surgery of HS-affected anogenital area (top row) and left axilla (lower row)

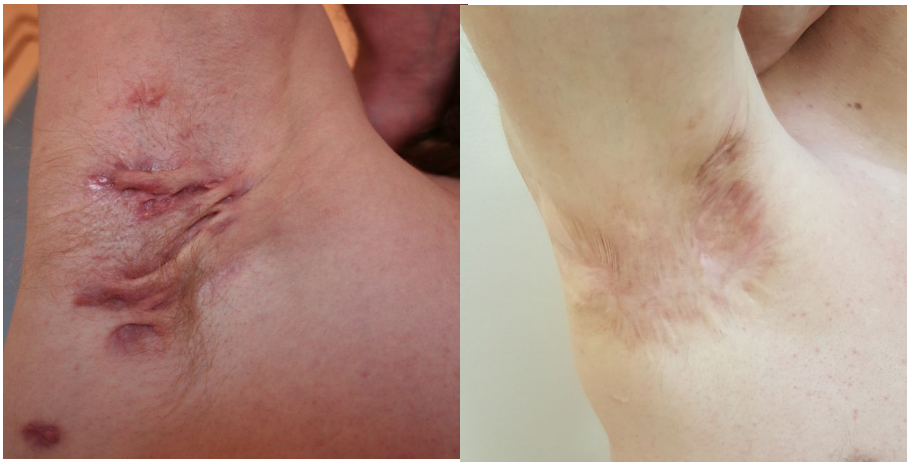


Figure 9. Extensive surgery results of the axillar region. Hypertrophic scarring, fistulae can be observed in the left. Atrophic scarring can be seen on the right after 5 years with no recurrence of HS. VUHSK Centre of Dermatovenereology.

2.8.8. Future treatment developments

IL-36 has been associated with the development of inflammatory bowel disease, psoriasis, acne, and HS. In these conditions, IL-36 has been linked with inflammation, the attraction of neutrophils, and the thickening of the epidermis. Spesolimab is presently employed to target IL-36. The approach of using spesolimab to target IL-36 for treating HS is well-suited to the pathogenic framework of the disease. At present, the supporting evidence is primarily derived from individual cases and case series, but there are several studies underway to further investigate its potential efficacy (199).

The involvement of the Janus kinase (JAK)/signal transducer and activator of transcription signalling (STAT) has been linked to the pathophysiological processes of HS. Based on observations from skin biopsies and blood samples of individuals with moderate-to-severe HS, an 8-week treatment with the JAK1 inhibitor, povorcitinib, was associated with the reversal of a previously identified HS transcriptomic pattern. Additionally, there were dose-dependent alterations in several circulating proteins that may play a role in the development of the disease. These observed changes in biomarkers largely align with the mechanism of action of povorcitinib, which primarily affects the pathways controlled by JAK/STAT signalling. Furthermore, some biomarker changes appear to mirror the clinical effectiveness of the treatment, underscoring the potential for JAK1 inhibition to influence the fundamental disease processes in HS (200).

3. MATERIALS AND METHODS

3.1. Study design and setting

All analyses were performed using data from a reference centre cross-sectional prospective study, which was conducted at Vilnius University Hospital Santaros Klinikos Centre of Dermatovenereology in Lithuania from March 2021 to June 2023. All HS patients presenting to the Centre were invited to participate in the study. Subjects were recruited by consecutive sampling based on their clinic appointment and included according to the following enrolment criteria:

Inclusion criteria

- Subject is capable to read, understand and sign the informed consent form before any investigative procedures
- Adult males and females, aged 18-70 y.o.
- Diagnosis of hidradenitis suppurativa
- Subject is able to attend planned visits

Exclusion criteria:

- Subject refusal to participate in the study
- Underage subjects
- Subject is unable to understand protocol procedures and agree with them
- Pregnant or breastfeeding females
- Subjects with active malignancy

3.2. Study organisation

HS patients presenting to the Centre of Dermatovenereology were offered to participate in the study. Patients were informed about the study and provided consent forms to sign. Next, they were assessed according to European Hidradenitis Suppurativa Foundation (EHSF) Registry questionnaire guidelines (**Appendix I**) at various stages of disease and treatment.

Data collection focused on demographic variables such as age, gender, body mass index (BMI), education level, skin phototype, hair structure, occupation, comorbidities, family history of inflammatory diseases, lesion location, and clinical subtypes of HS based on the *Canoui-Pouitrine* criteria (201). Smoking habits, their relationship to HS severity (according to Hurley

stage), age at diagnosis, disease duration, diagnostic delays, and common misdiagnoses were recorded. Pain intensity was measured using the visual analogue scale (VAS), and anxiety was scored on a 0–10 scale. Previous and current treatments were documented, with a specific focus on adalimumab treatment for patients with moderate-to-severe HS who had not responded to previous systemic antibiotic therapy from 2018 to 2023. Clinical metrics, such as the number of nodules, abscesses, fistulas, International Hidradenitis Suppurativa Severity Score System (IHS4), Dermatology Life Quality Index (DLQI), and VAS pain scores, were recorded both before and during treatment.

IHS4 score

The severity of HS can be evaluated using the International Hidradenitis Suppurativa Severity Scoring System (IHS4), a validated tool created by the European HS Foundation. The IHS4 score is determined by multiplying the number of inflammatory nodules by 1, the number of abscesses by 2, and the number of draining tunnels by 4. After calculating the total score, HS severity is categorized as mild (score ≤3), moderate (score 4 to 10), or severe (score ≥11) (202).

Dermatology Life Quality Index

Dermatology Life Quality Index (DLQI) is a self-administered, easy, and user-friendly questionnaire with an average completion time of 126 s. It consists of 10 questions concerning patients' perception of the impact of skin diseases on different aspects of their QoL over the last week. It has been validated for dermatology patients aged 16 years and above. The questions in the DLQI are classified into 6 heading items: symptoms and feelings (questions 1 and 2), daily activities (questions 3 and 4), leisure (questions 5 and 6), and personal relationships (questions 8 and 9), each item with a maximum score of 3, work and school (question 7) and treatment (question 10), each item with a maximum score of 3. An example of a questionnaire in the Lithuanian language is in **Appendix II**.

HiSCR

The treatment efficiency of patients receiving biological therapy was assessed using the HiSCR scale. This scale was designed to serve as a tool for evaluating clinical response to treatment. The proposed definition of treatment response (reaching HiSCR) involves achieving at least a 50% reduction in the total number of abscesses and inflammatory nodules, without any increase in

the number of abscesses or tunnels, regardless of inflammatory activity, compared to the baseline count (203).

Skin biopsy

Along with clinical data, subjects also consented to punch (3–5 mm) skin biopsies being taken from their HS lesions and healthy-looking skin 3–4 cm away from the lesion. The obtained biopsies were fixed in formalin 10% solution and processed in the National Centre of Pathology, where they were embedded in paraffin blocks and stored for future use.

3.3. Ethical statements

Ethical approval for the study was obtained from the Vilnius Regional Biomedical Research Ethics Committee (No. 2021/2-1310-793). The ethical approval was updated on 28 JUN 2023 (No. 2023-LP-55) (**Appendix III**) to incorporate changes related to data and skin biopsy sampling outside the EU. The study protocol corresponds to the 1975 Declaration of Helsinki revised in 2013. All subjects informed about the study and provided signed consent forms.

3.4. Collected data

Sociodemographic data:

- Age, gender, ethnic origin, skin phototype, hair structure
- Marital, professional status, education

Life history:

- Comorbidities: acne, psoriasis, IBD, pilonidal sinus/cyst, depression, arthritis,
- Metabolic diseases: dyslipidaemia, ischemic heart disease, diabetes, cerebrovascular disease, hypertension, metabolic syndrome,
- Family history
- Past and current medications
- Smoking history
- Alcohol and drug use
- Allergies

History of HS:

- Age when the first boil appeared, when the patient first went to the doctor, age at diagnosis of HS
- The time it took to see a dermatovenereologist after the family physician referral
- Specialist who diagnosed the disease, misdiagnoses
- Number of disease relapses over the past year, average duration of disease relapse
- Number of boils (new or flared) in the last 4 weeks
- Boil soreness from 0 to 10, number of painful days in the last 4 weeks, type of pain
- The progression of the disease since its onset
- Absence from work due to HS, impact of illness on professional career
- Exacerbating factors of the disease

Complications:

- Health problems caused by HS
- Hospitalization due to HS illness, times, number of days

Quality of life:

- DLQI
- Anxiety score (from 0 to 10)
- Impact of illness on sleep and behaviour from 0 to 10

Treatment:

- Local antiseptics, antibiotics
- Systemic antibiotics
- Other systemic treatment
- Biological therapy
- Laser therapy
- Surgery

Physical examination:

- The number of lesions, total number inflammatory lesions
- Associated skin lesions
- Blood pressure
- Waist circumference
- Hurley stage: I, II, III

- IHS4 score
- Subclinical variant
- Weight, height, BMI

3.5. Statistical analysis

Statistical analysis was performed using MS Excel 2021 and IBM SPSS 26.0, employing descriptive statistics for both qualitative and quantitative variables. The homogeneity of the groups was evaluated using the chi-square test. Normality of data distribution was assessed via the Shapiro-Wilk test. For normally distributed data, the paired-samples t-test was applied, while non-parametric data were analysed using the Wilcoxon test. The Mann-Whitney U and Kruskal-Wallis tests were used for comparisons between independent groups, and chi-square or Fisher's exact tests were employed for qualitative parameters. Differences were considered statistically significant when p-values were below 0.05 ($p<0.05$).

4. RESULTS

4.1. Demographics (Publication I)

The study included 49 patients, 57.14% (N=28) were male and 42.86% (N=21) were female. The average age of the subjects was 39.91 ±13.665 years, average BMI – 28.44 ±6.142, average waist circumference – 91.85 cm, 30.61% (N=15) were overweight and 36.73% (N=18) were obese (**Table 1**).

Table 1. Average BMI, waist circumference, weight, and obesity rate.

Variables	**Patients (n=49)**	***P* value**
Average BMI kg/m^2 Female BMI kg/m^2 Male BMI kg/m^2	28.44±6.142 28.12±5.819 28.68±6.142	0.372
Waist circumference, cm Females, cm Males, cm	91.85±16.806 85.90±19.944 96.32±12.619	0.077
Weight, kg Females, kg Males, kg	87.93±21.025 80.04±24.340 93.85±16.497	<0.001
Overweight, n, (%)	33 (67.35%)	

There was no statistical correlation between gender and BMI variables. There was a statistically significant correlation between obesity and smoking status ($r=0.673$, $p<0.01$). 72% of overweight individuals were smokers, compared to only 28% in the normal weight group. Similarly, 64% of overweight individuals were previous smokers, while only 36% of those with normal weight fall into this category. Nonsmokers showed a similar pattern, with 65% of overweight individuals being nonsmokers, compared to 35% in the normal weight group. (***Figure 10***).

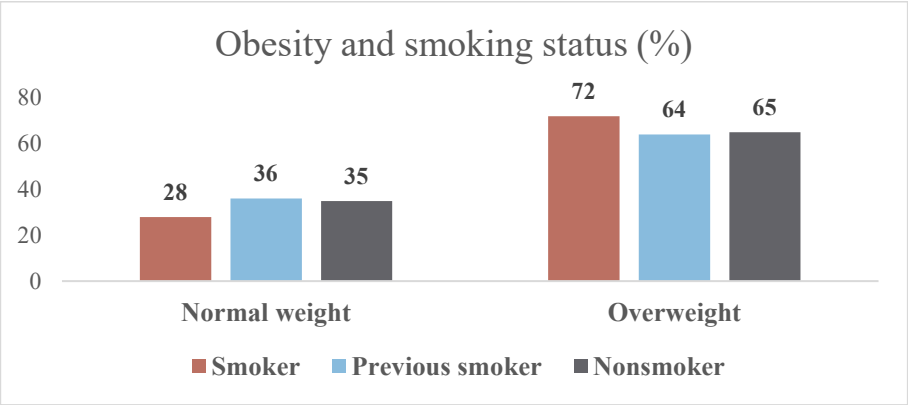


Figure 10. Smoking and obesity association among HS patients.

51.02% (N=25) of subjects had higher education. Disease severity did not depend on hair structure (p=0.467) or skin phototype (p=0.631). The data is distributed according to the normality curve, the groups are homogeneous (**Table 2**).

Table 2. HS patient characteristics: subject distribution according to age, sex, education level, skin phototype, hair structure and professional status

Variables	**Patients (n=49)**	***P* value**
Age, years		
Mean±SD	39.91±13.665	0.565
Median (range)	39.24 (18.73)	
Sex		
Female, n, (%)	21 (42.85%)	0.378
Male, n, (%)	28 (57.14%)	
Education level		
Primary education, n (%)	7 (14.28%)	
Secondary education, n (%)	17 (34.69%)	
Higher education, n (%)	25 (51.02%)	
Professional status, n, (%)		
Working/Student	41 (83.67%)	
Unemployed	2 (4.08%)	
Disability	5 (10.20%)	
Pensioner	1 (2.04%)	
Skin phototypes, n, (%)		
I	15 (30.61%)	
II	19 (38.77%)	
III	13 (26.53%)	
IV	2 (4.08%)	

Variables	Patients (n=49)	*P* value
Hair structure, n (%)		
Straight Wavy Curly	32 (65.30%) 13 (26.53%) 4 (8.16%)	

Average disease onset was at 25.71±13.743 years, patients sought medical care at 28 years, and the mean time to diagnosis was 5.2±7,607 years. Female patients were diagnosed significantly later than males, on average 6.5 years versus 4.2 years (p=0.01) (**Table 3**).

Table 3. The age of disease onset, initiation of medical care, and time to diagnosis.

Variables	Patients (n=49)	*P* value
Disease onset (years±SD):	25.71±13.743	0.425
- Females	26.61±13.93	0.865
- Males	25.03±13.743	0.243
Medical care started (years±SD)	28.22±14.000	0.391
- Females	27.57± 14.204	0.452
- Males	28.71±14.003	0.841
Time to diagnosis (years±SD)	5.2±7.607	0.201
- Females	6.5±7.748	<0.001
- Males	4.2±7.607	0.540

70.2% (N=33) were previously misdiagnosed; 51.51% (N=17) subjects were diagnosed with a furuncle (**Table 4**). 87.75% (N=43) of patients were diagnosed with HS by dermatovenereologist.

Table 4. The most common misdiagnosis.

Misdiagnosis, N, %	Patients (n=49)
Total	33 (70.2%)
Furuncle	17 (51.51%)
Ulcer	4 (12.12%)
Abscess	3 (9.09%)
Acne	2 (6.06%)
Allergy; Complication of Diabetes; Mycosis; Lymphadenitis; Pilonidal sinus; Folliculitis; Psoriasis	1 (3.03%)

4.2. Comorbidities (Publication II)

It was found that 34.69% (N=17) of subjects had severe acne and in 64.70% (N=11) of them, it was still ongoing; however, no correlation between acne and Hurley stage (r=0.088) or severity of HS (r=0.091) was found. We found that 61.22% (N=30) pf patients had comorbidities such as psoriasis, inflammatory bowel disease, joint, metabolic disease, dyslipidaemia, and hypertension (**Table 5**).

Table 5. HS patient demographic data and comorbidities.

Variables	**Patients (n = 49)**	**Hurley stages:**	
Caucasians	49 (100%)	**I**	25 (51.02%)
Metabolic comorbidities:		**II**	12 (24.48%)
Hypertension	10 (29.4%)	**III**	12 (24.48%)
Dyslipidaemia	6 (12.24%)	**Other comorbidities:**	
Diabetes	3 (6.12%)	Depression	8 (16.32%)
Metabolic disease	1 (2.04%)	Joint pain	6 (12.24%)
Inflammatory diseases:			
Acne	17 (34.69%)	Psoriasis	4 (8.16%)
Pilonidal cyst	10 (29.4%)	Inflammatory bowel disease	3 (6.12%)

Only 18.36% (N=9) had a family history of HS; however, a moderate positive correlation was found between a family history of inflammatory diseases (69.38% (N=34)), which include acne, psoriasis, inflammatory bowel disease, and joint disease, and the severity of HS according to Hurley stage ($r=0.71$, $p<0.05$). We found that 30.6% (N=15) of patients had comorbidities related to cardiovascular disease, and 60% (N=9) of them had a positive family history of inflammatory diseases. Comparing the severity of HS, a strong statistically significant correlation ($r=0.944$, $p=0.02$) was found between metabolic comorbidities and Hurley stage, and 55.0% (N=10) of them were Hurley stage III (***Figure 11***).

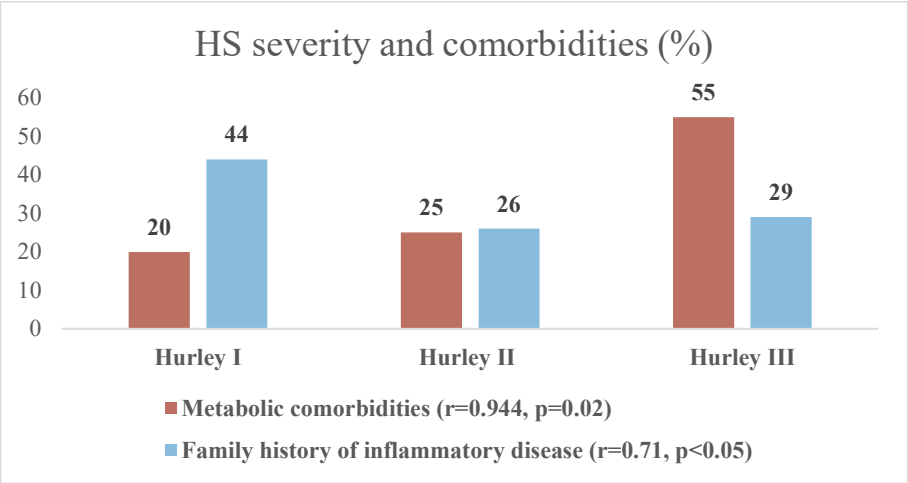


Figure 11. Association between Hurley stage and metabolic comorbidities as well as family history of inflammatory disease.

4.3. Smoking (Publication I)

36.73% (N=18) of subjects are active smokers and 22.44% (N=11) are previous smokers with an average of 26.56 pack-years. 40.81% (N=20) of participants are nonsmokers. Among 12 Hurley III patients, 91.66% (N=11) were smokers, statistically significant correlation was found ($r=0.659$, $p<0.01$) between these two variables. Distribution between smokers and nonsmokers with different Hurley stage was statistically significant as well ($p<0.01$). There are statistically more male smokers than female ($p<0.01$), with 33.33% (n=7) of women and 78.57% (n=22) of men being smokers.

4.4. Clinical presentation (Publication II)

75.51% (N=37) had lesions in the axillary region, 59.18% (N=29) in the groin area, 28.57% (N=14) in the pubic, and 26.53% (N=13) in other areas (**Table 6**). There were no significant differences between males and females. In 88% (N=16) of obese subjects, the groin area was affected.

Table 6. Distribution of HS lesions in males and females.

Location of lesions	All patients (N=49)	Males (N=28)	Females (N=21)	*p*-Value
Axillary	37 (75.51%)	20 (71.42%)	17 (80.95%)	0.295
Groin	29 (59.18%)	18 (64.28%)	11 (52.38%)	0.042
Pubic	14 (28.57%)	8 (28.57%)	6 (28.57%)	0.395
Other areas	13 (26.53%)	7 (25.00%)	6 (28.57%)	0.467

HS lesion types				
Axillary region:				
Abscesses	7 (18.91%)	4 (20%)	3 (17.64%)	0.271
Nodules	21 (56.75%)	9 (45%)	12 (70.58%)	0.249
Fistulas	9 (24.32%)	5 (25%)	4 (23.52%)	0.543
Groin region:				
Abscesses	7 (24.13%)	5 (27.77%)	3 (27.27%)	0.042
Nodules	15 (51.72%)	9 (50%)	7 (63.63%)	0.284
Fistulas	7 (24.13%)	4 (22.22%)	1 (9.09%)	0.031
Pubic region:				
Abscesses	4 (28.57%)	2 (25%)	1 (16.66%)	0.038
Nodules	8 (57.14%)	4 (50%)	4 (66.66%)	0.782
Fistulas	2 (14.28%)	2 (25%)	1 (16.66%)	0.081
Other regions (face, chin, abdomen, breasts):				
Abscesses	6 (46.15%)	4 (57.14%)	2 (33.33%)	0.246
Nodules	5 (38.46%)	3 (42.85%)	3 (50%)	0.291
Fistulas	2 (15.38%)	0 (0%)	1 (16.66%)	0.823

4.5. Histological findings in HS patients

The histological examination of early lesions in suppurative hidradenitis reveals follicular hyperkeratosis, hyperplasia of the follicular epithelium, and perifolliculitis (**Figure 10**). There is a mixed inflammatory cell infiltrate within the dermis, sometimes extending into the subcutaneous layer. Neutrophilic abscesses may be present, often connecting to cysts and sinus tracts lined by squamous epithelium, which open to the skin surface. These cysts and tracts typically contain laminated keratin and occasionally hair follicles. Approximately 25% of cases show granulation tissue with occasional foreign body giant cells. Inflammation may involve the apocrine glands in some cases. Additionally, there is dense fibrosis surrounding sites of follicular rupture. (***Figures 12–15***)

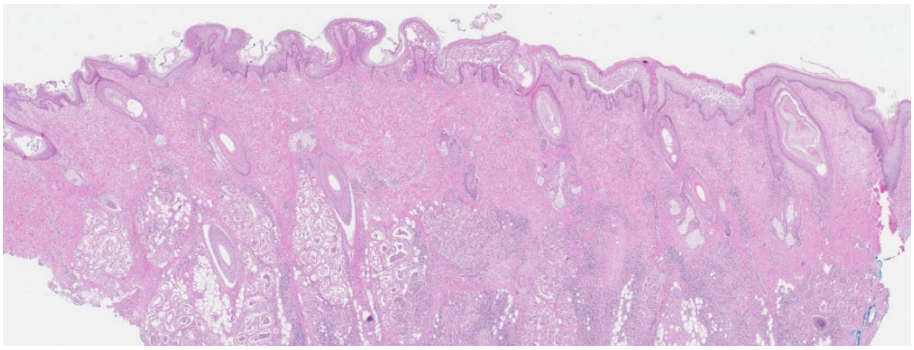


Figure 12. HS histopathology: pronounced follicular hyperkeratosis, follicular hyperplasia and perifolliculitis

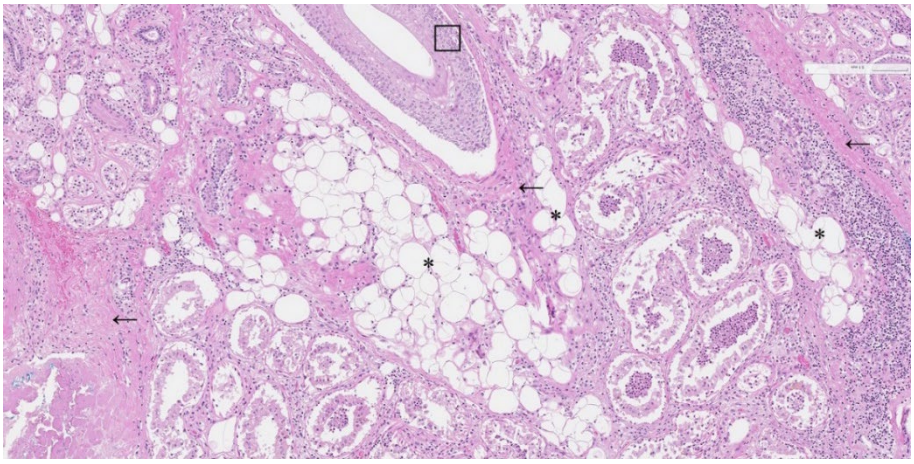


Figure 13. HS histopathology: Dilated and ruptured hair follicles with keratin and inflammatory infiltrate. There is a prominent mixed cell type inflammation, particularly around the hair follicles and within the the dermis, suggesting an acute and destructive inflammatory response.

Adipocytes (*). Inflammation extends into the subcutaneous tissue, which can lead to painful, deep lesions typical of hidradenitis suppurativa. Granulation tissue and fibrosis (→). This suggests attempts at healing and scar formation, common in chronic and recurrent cases of the condition. Epithelium of the hair follicle (□).

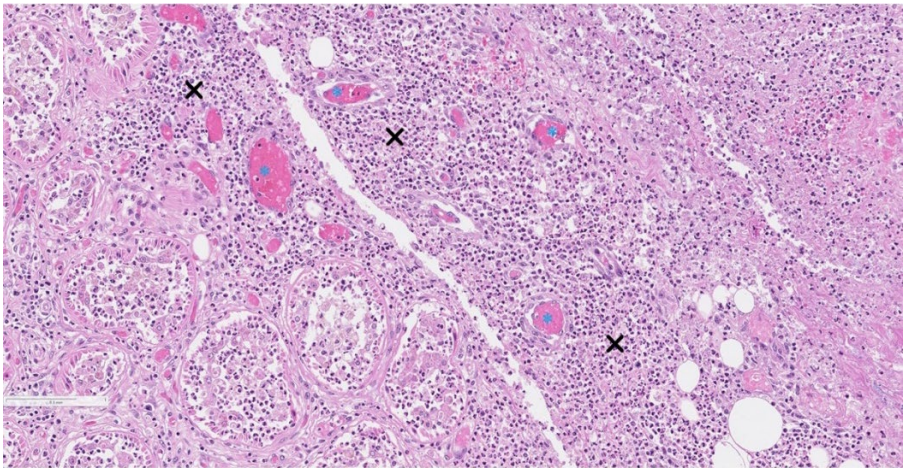


Figure 14. HS histopathology: intense inflammatory infiltrate throughout the dermis.

The inflammatory infiltrate (✕) includes neutrophils, lymphocytes, plasma cells, and histiocytes, indicative of both acute and chronic inflammation. This can occur due to tissue damage from the chronic inflammation and abscess formation. Some of apocrine glands appear to be dilated with infiltration of inflammatory cells, which is a characteristic finding. Congestive capillaries (*).

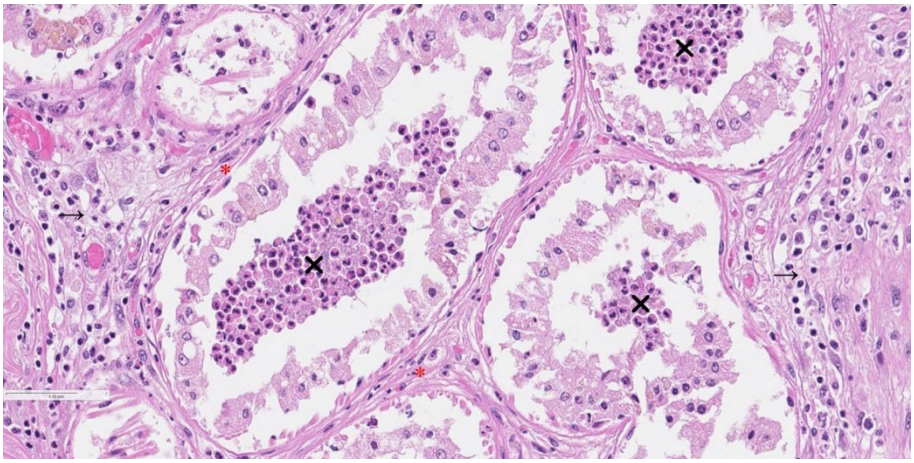


Figure 15. HS histopathology: dilated apocrine glands are filled with inflammatory cells

The inflammatory infiltrate contains predominantly neutrophils (✕). This indicates acute inflammation, which can lead to the formation of microabscesses within the glands. Neutrophilic infiltration is a hallmark of

active, suppurative inflammation. The epithelial lining of apocrine ducts appears to be edematous, raptured and desquamated (*). The stroma surrounding the glands appears fibrotic and swollen, with evidence of inflammatory infiltrate containing lymphocytes, histiocytes, neutrophils and plasma cells (→).

4.6. Clinical subtypes of HS (Publication II)

The HS phenotype of all patients was determined using the *Canoui-Pouitrine et al*. criteria (201). We found that 40.81% (N=20) had an axillary mammary phenotype, 34.69% (N=17) follicular, and 24.48% (N=12) gluteal phenotype. A follicular phenotype had a higher percentage of non-smokers compared to the axillary mammary phenotype and more Hurley III subjects than the gluteal and axillary mammary phenotypes (**Table 7**).

Table 7. Clinical subtypes of HS and distribution between variables.

	Axillary mammary phenotype (N=20)	**Follicular phenotype (N=17)**	**Gluteal phenotype (N=12)**	***p*-value**
Gender				
Female	9 (45%)	7 (41.17%)	5 (41.66%)	0.651
Male	11 (55%)	10 (58.82%)	7 (58.33%)	0.743
Smoking status				
Smoker	12 (60%)	7 (41.17%)	5 (41.66%)	<0.001
Non-smoker	8 (40%)	10 (58.82%)	7 (58.33%)	
Hurley Stage				
I	13 (65%)	6 (35.29%)	6 (50%)	0.231
II	5 (25%)	3 (17.64%)	4 (33.33%)	0.482
III	2 (10%)	8 (47.05%)	2 (16.66%)	<0.001

4.7. Pain (Publication I)

On average, subjects had 6.17 ±6.98 painful days over the last 4 weeks. The average pain intensity score according to the VAS scale was 5.60 ±3.36. Females indicated that their average pain intensity score was 5.42 points, males – 5.74 (**Table 8**). 30.61% (N=15) had persistent pain, and 69.38% (N=34) intermittent. Male patients statistically significantly complained more often with intermittent pain (p=0.003). 24.48% (N=12) of subjects claim that HS exacerbation is caused by pressure or mechanical friction.

Table 8. Pain caused by HS: number of painful days over the last 4 weeks and average VAS scores.

Variables	All patients (n=49)	Females (n=21)	Males (n=28)	p-value
Painful days over last 4 weeks (±SD)	6.17±6.98	6.61±7.06	5.92±6.85	0.005
Average VAS score (±SD)	5.60±3.36	5.42±3.34	5.74±3.36	0.003
Persistent pain N, %	15 (30.61%)	8 (38.09%)	7 (25%)	0.062
Intermittent pain N, %	34 (69.38%)	13 (61.90%)	21 (75%)	0.003

4.8. Disease burden and quality of life (Publication I)

36.73% (N=18) were hospitalized for HS with an average length of 19.5 days of hospitalization; of them, 66.66% (N=12) were males and 61.11% (N=11) had severe HS with Hurley stage III. 26.53% (N=13) of all subjects admitted that HS affected their professional career. 24.48% (N=12) missed work due to HS; on average, over 6 months, patients missed 20.5 working days. 76.92% (N=10) of them have higher education diploma.

Patients are worried about their illness, the average score of their anxiety level is 6.5±2.586 points out of 10. Male patients worry about the disease more often, they rated their anxiety level at 6.84 vs 6.04 for females ($p=0.014$). Age has a slight correlation with anxiety level – the younger the patient, the higher the anxiety score ($r=0.231$, $p=0.002$). Patients rated the disease impact on quality of sleep with an average of 2.68±3.060 points out of 10. 77.55% (N=38) marked their anxiety level >5, all these patients had worse sleep quality than subjects with an anxiety score of <5 ($p=0.02$).

Mean dermatology life quality index (DLQI) at baseline was 8.30±7.461 for all subjects (***Figure 16***). A total of 32.65% (N=16) patients had a DLQI of >10, 62.5% (N=10) of them were males. A total of 93.75% (N=15) had a BMI >25 ($p<0.01$) and their anxiety level score was 7.5 points.

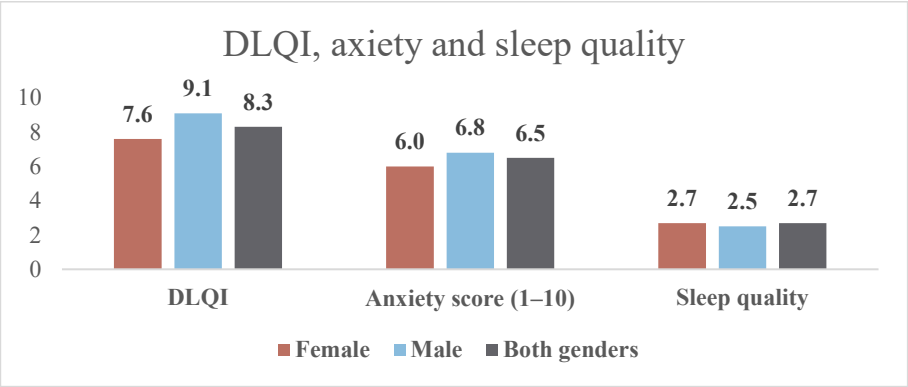


Figure 16. Dermatological quality of life, anxiety levels and sleep quality of HS patients.

4.9. Treatment (Publication II)

Before the assessment, patients used a variety of medications for HS which are presented in **Table 9**.

Table 9. Previous treatments for HS patients.

Medication/treatment	Patients (n=49)	Efficacy (N, %)
Topical treatment		
Antiseptics	29 (59.18%)	Partial (N=28, 96.55%) None (N=1, 3.44%)
Antibiotics	46 (92.03%)	Partial (N=44, 95.65%) None (N=1, 2.17%) Complete (N=1, 2.17%)
Intralesional corticosteroids	10 (20.40%)	Partial (N=7, 70%) None (N=2, 20%) Complete (N=1, 10%)
Systemic treatment		
NSAIDs	5 (10.20%)	Partial (N=4, 80%) None (N=1, 20%)
Doxycycline	31 (63.26%)	Partial (N=24, 77.41%) None (N=4, 12.90%) Complete (N=3, 9.6%)
Rifampicin + Clindamycin	17 (34.69%)	Partial (N=15, 88.23%) Complete (N=2, 11.76%)
Clindamycin	10 (20.40%)	Partial (N=9, 90%) Complete (N=1, 10%)
Penicillin + Clavulanic acid	5 (10.20%)	Partial (N=2, 40%) None (N=3, 60%)

Medication/treatment	Patients (n=49)	Efficacy (N, %)
Topical treatment		
Retinoids	7 (14.28%)	Partial (N=5, 71.42%) None (N=1, 14.28%) Complete (N=1, 14.28%)
Adalimumab	14 (28.57%)	Partial (N=12, 85.71%) Complete (N=2, 14.28%)
Interventions		
Previous incisions and drainage	24 (48.97%)	Partial (N=14, 58.33%) Complete (N=10, 41.66%)

After the initial assessment, the following medications were usually prescribed, which are presented in **Table 10**.

Table 10. Prescribed treatments for HS patients.

Medication/treatment	Patients (n=49)
Topical treatment	
Antiseptics	49 (100%)
Clindamycin	44 (89.79%)
Intralesional corticosteroids	1 (2.04%)
Systemic treatment	
Rifampicin + Clindamycin	8 (16.32%)
Doxycycline	17 (34.69%)
Clindamycin	7 (14.28%)
Retinoids	3 (6.12%)
Biologics	
Adalimumab	11 (22.44%)
Secukinumab	5 (10.20%)
Interventions	
Incisions and drainage	3 (6.12%)

We found out that 32.65% (N=16) were on biological therapy, 65.30% (N=32) were on long-term systemic antibiotics, 14.89% (N=7) on both biologics and systemic antibiotics, and 44.68% (N=21) were treated only with topicals. Patients on biologics initially had a mean DLQI of 7.4, systemic antibiotics of 10.5, and others of 7. Data from the follow-up was included after 6 months of the initial assessment and medication prescription. Patients on biologic treatment had a mean IHS4 of 7.38 at the beginning of treatment and 3.22 at follow-up ($p<0.05$). Meanwhile, for patients not on biologics, the initial IHS4 score was 6.21 and 5.42 at follow-up ($p>0.05$) (***Figure 17***). We

found that 29.78% (n=14) had recurrent inflammatory lesions in the last 4 weeks, with no differences between patients on biologics or systemic antibiotics.

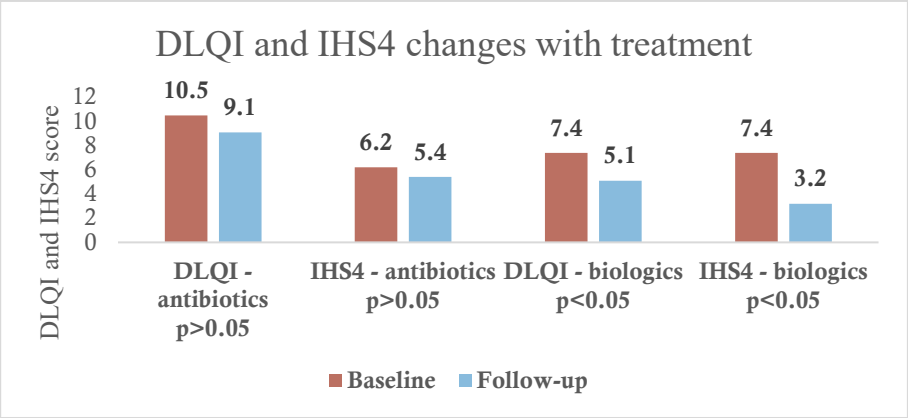


Figure 17. Systemic HS treatment impact on the DLQI and IHS4 scores.

4.9.1. Adalimumab sub-study (Publication III)

Demographics of patients receiving adalimumab

In total, 21 patients diagnosed with moderate-to-severe HS were included in this study, comprising 8 women (38.1%) and 13 men (61.9%) with a mean age of 42.9±14.1 years. The mean body mass index (BMI) of the patients was 30.33±7.13, with eight patients (38.1%) considered overweight and eight (38.1%) considered obese (**Table 11**).

Table 11. Demographics characteristics of patients receiving adalimumab.

Characteristic	**Patients (n = 21)**
Sex	
Female, n, (%)	8 (38.1%)
Male, n, (%)	13 (61.9%)
Mean age, years, (±SD)	42.9 (±14.1)
Mean BMI kg/m^2 (±SD)	30.33 (±7.13)
Normal (18.5–24.9), n, (%)	5 (23.8%)
Overweight (25.0–29.9), n, (%)	8 (38.1%)
Obese (≥30.0), n, (%)	8 (38.1%)

Clinical Characteristics of patients receiving adalimumab

The majority of patients, constituting 13 individuals (61.9%), were classified as Hurley stage III. The mean duration of the disease was 15.48±12.83 years. All 21 (100%) patients received prior systemic antibiotic treatment, 14 of whom (66.7%) had prior surgical treatment. The average baseline IHS4 score was 19±10.78, with most patients categorized as having severe HS. The initial DLQI score averaged 15.76±7.73, signifying a very large effect of the disease on an individual's quality of life. The mean pain intensity according to the VAS at baseline was 6.69±1.59, indicating a moderate level of pain experienced by patients (**Table 12**).

Table 12. Baseline clinical characteristics of patients receiving adalimumab.

Characteristic	**Patients (n = 21)**
Hurley stage, n, (%)	
II	8 (38.1%)
III	13 (61.9%)
Mean duration of HS, years, (±SD)	15.48 (±12.83)
Prior systemic antibiotic use, n, (%)	21 (100%)
Prior surgery for HS, n, (%)	14 (66.7%)
Lesion counts	
Mean no. of inflammatory nodules (±SD)	5.62 (±4.12)
Mean no. of abscesses (±SD)	1.76 (±2.63)
Mean no. of fistulas (±SD)	2.62 (±1.86)
Mean IHS4 score (±SD)	19 (±10.78)
Moderate (4–10), n, (%)	7 (33.3%)
Severe (≥11), n, (%)	14 (66.7%)
Mean DLQI score (±SD)	15.76 (±7.73)
Mean VAS score (±SD)	6.69 (±1.59)

Notably, 10 (47.62%) patients achieved HiSCR after one year of adalimumab treatment. A significant decrease in all HS lesions compared to the baseline counts was observed after one year of therapy ($p<0.05$). The average count of inflammatory nodules reduced from 5.62±4.12 to 3±3.46, the

mean count of abscesses decreased from 1.76±2.63 to 0.81±1.4, and the mean count of fistulas decreased from 2.62±1.86 to 2±1.9 (***Figure 18***).

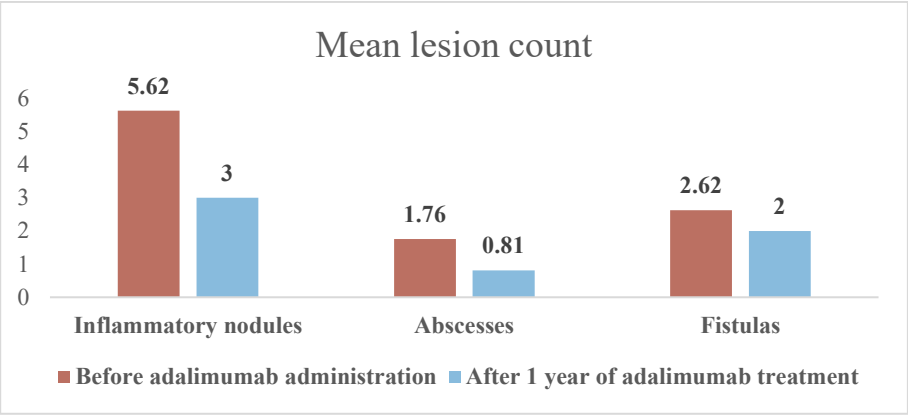


Figure 18. Mean lesion count of inflammatory nodules, abscesses, and fistulas before adalimumab administration and after 1 year of treatment.

Before adalimumab administration, 14 patients (66.7%) had a severe HS according to the IHS4 scores, which decreased to 11 patients (52.4%) after treatment. Notably, there were no patients with mild HS initially, but after one year of treatment, seven patients (33.3%) with a moderate-to-severe form transitioned to the mild HS category (***Figure 19***).

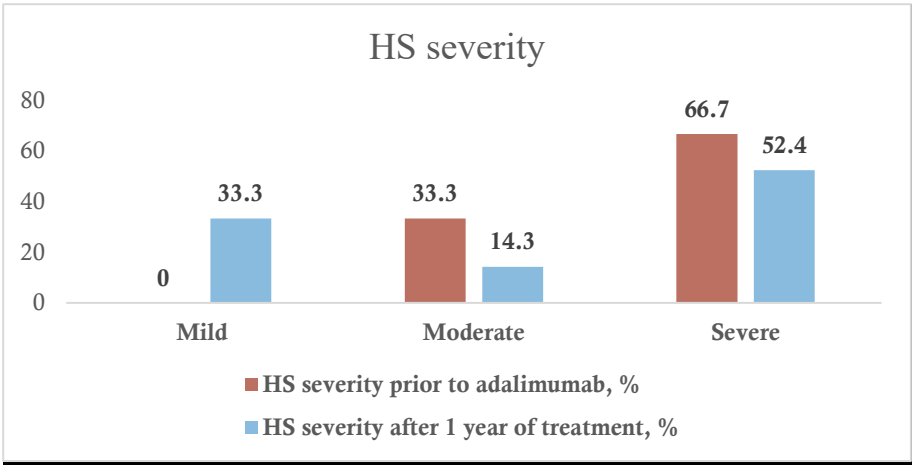


Figure 19. Distribution of HS severity categories before adalimumab treatment and after 1 year of treatment.

A reduction in the number of inflammatory lesions corresponded with the IHS4 scores. The reduction in the IHS4 score after 1 year of adalimumab treatment was statistically significant compared to the baseline (p=0.001). A

mean IHS4 score of 19±10.78 dropped to 12.62±11.13. There was also a statistically significant enhancement in patients' quality of life, as evidenced by the DLQI questionnaire (p<0.001). At the baseline, the mean DLQI score was 15.76±7.73, which decreased to 7.43±7.76 after one year of treatment, indicating that on average, the very substantial effect on patients' quality of life was reduced to moderate. Moreover, we assessed the VAS scores before initiating adalimumab treatment and after one year of therapy. The initial mean VAS score of 6.69±1.59 decreased statistically significantly to 3.64±2.65 (p<0.001) (***Figure 20***).

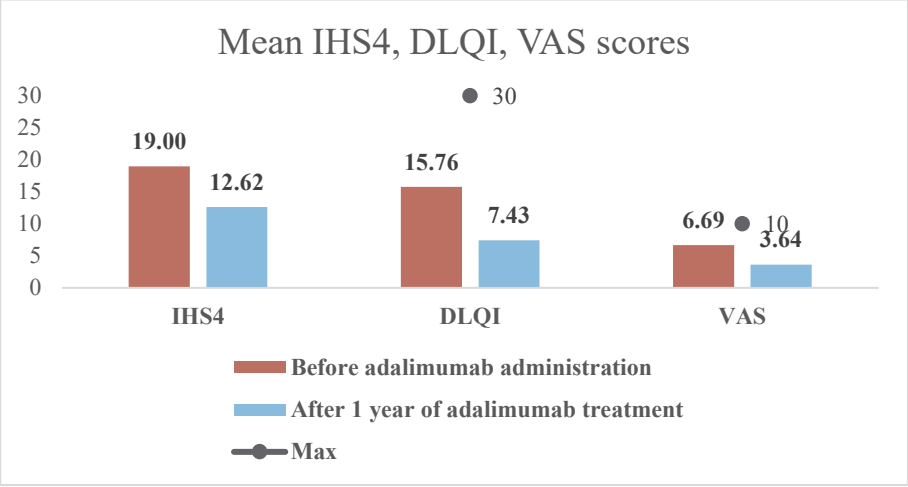


Figure 20. Mean IHS4, DLQI, and VAS scores before the administration of adalimumab and following one year of treatment.

Influence of BMI and Surgical Treatment

There was no significant association between BMI category (normal, overweight, and obese) and achievement of HiSCR (p=0.350) (**Table 13**). The mean BMI of those who achieved the HiSCR was 32.17±8.66, and those who did not achieve this clinical score had a mean BMI of 28.65±5.27.

Table 13. Association between BMI category and achievement of HiSCR.

	Achieved HiSCR (n = 10)	**Did not Achieve HiSCR (n = 11)**	**Total (n = 21)**	***p*-Value**
Normal BMI, Count (%)	3 (60%)	2 (40%)	5	0.350
Overweight, Count (%)	5 (62.5%)	3 (37.5%)	8	
Obese, Count (%)	2 (25%)	6 (75%)	8	

There were no statistically significant differences observed in the pre-treatment IHS4 estimate (p=0.928), the baseline DLQI score (p=0.232), or the baseline VAS score (p=0.316) between patients with a normal BMI and those who are overweight or obese. The highest mean IHS4 and VAS values were in the group of patients with normal BMI (20.4±13,01 and 7.4±1.08, respectively); however, the highest mean DLQI (19.25±7.23) was in the group of overweight patients (**Table 14**).

Table 14. Distribution of initial mean IHS4, DLQI, and VAS estimates between BMI groups.

	Normal BMI	**Overweight**	**Obese**	***p*-Value**
Mean IHS4 (±SD)	20.4 (±13.01)	19.75 (±13.18)	17.38 (±7.52)	0.928
Mean DLQI (±SD)	12.8 (±7.95)	19.25 (±7.23)	14.13 (±7.68)	0.232
Mean VAS (±SD)	7.4 (±1.08)	6.13 (±1.71)	6.81 (±1.69)	0.316

There was also no difference in the proportion of patients achieving a HiSCR between those who received prior surgical treatment and those who did not (p=0.659) (**Table 15**).

Table 15. Association between patients with or without prior surgical treatment and the attainment of HiSCR.

	Achieved HiSCR (n = 10)	**Did not Achieve HiSCR (n = 11)**	**Total (n = 21)**	***p*-Value**
Prior surgical treatment, N (%)	6 (42.86%)	8 (57.14%)	14	0.659
No prior surgery, N (%)	4 (57.14%)	3 (42.86%)	7	

There was a significant difference (p=0.001) in the IHS4 scores before adalimumab treatment, with a mean score of 23.86±9.4 for patients who had prior surgery, compared to the mean score of 9.29±5.53 for those who had not undergone prior surgery. There were no statistically significant differences in the initial DLQI (p=0.585) and VAS scores (p=0.4) between patient groups with and without prior surgery for HS, with the mean scores being higher in patients who did not have prior surgery (**Table 16**).

Table 16. Distribution of initial mean IHS4, DLQI, and VAS estimates between patients who had surgical treatment and those who did not.

	Prior Surgery	**No Prior Surgery**	***p*-Value**
Mean IHS4 (±SD)	23.86 (±9.4)	9.29 (±5.53)	0.001
Mean DLQI (±SD)	15.5 (±8.92)	16.29 (±5.12)	0.585
Mean VAS (±SD)	6.46 (±1.82)	7.14 (±0.9)	0.4

4.10. Challenges of rare HS complications – squamous cell carcinoma (Publication IV)

Long-lasting inflammation of the skin and mucous membranes can lead to neoplasm development. One of the study subjects had a 16-year history of severe hidradenitis suppurativa treated with adalimumab 40 mg weekly for the last 5 years. Despite a stable disease with anti-TNF treatment, the patient experienced a relapse in 2023 with new purulent eruptions, diffuse infiltration, and rapid ulceration in the sacral and perineal areas, consistent with Hurley stage III (***Fig 21A***). Due to dramatic change in patient's condition, adalimumab was eventually discontinued. Combined intravenous antibiotic therapy was administered due to the multidrug-resistant *Acinetobacter baumanii* and elevated inflammatory markers (C-reactive protein – 76.82 mg/l). A biopsy from the border of the ulcer confirmed a well-differentiated squamous cell carcinoma (SCC), which tested negative for high-risk human papillomavirus (HPV). A computed tomography (CT) scan revealed sacral lesions with infiltration, liquefaction zones, skin involvement, and inguinal lymphadenopathy. A suspicious lesion in the S6 segment of the right lung suggested possible metastasis. The patient was consulted by a multidisciplinary team. A course of radiation therapy was initiated to reduce the tumour size followed by a wide excision resulting in a 20x20 centimetre tissue defect (***Fig. 21B***) Reconstructive surgery was performed, accompanied by the application of Vacuum-Assisted Closure (VAC) system, staged debridement, wound irrigations, and dressings. Skin grafting was performed four times. A follow-up CT scan after 6 months did not show any disease progression. The patient was under a close follow-up schedule of every 3 months, skin grafts had almost completely covered the defect (***Fig. 21C***), however, the patient passed away 10 months after the surgery from causes that remain unknown to us.

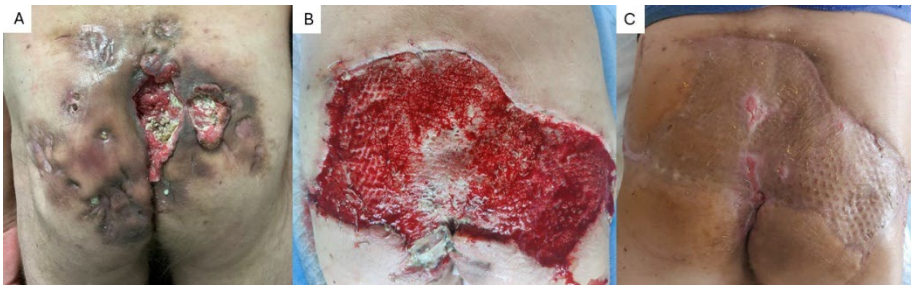


Figure 21. **A** – Purulent eruptions, diffuse infiltration, and rapid ulceration in the sacral and perianal area. **B** – Wide excision with a significant tissue defect, followed by a first grafting procedure. **C** – Four-month follow-up after last skin grafting procedure. VUHSK archive.

5. DISCUSSION

5.1. Demographics

Studies show that in North American and European populations, HS is more prevalent among females, with a female-to-male ratio of 3:1 (34,71,116). However, a different pattern emerges in South Korea, where the female-to-male ratio is approximately 1:2 (18,204). The highest prevalence of HS is observed in individuals in their fourth and fifth decades of life, however, this chronic skin condition commonly emerges during young adulthood, with a notable spike in new diagnoses occurring among those aged 18 to 29 years in the USA (34,71,116,205). In our study, which included 49 patients, 57.14% were male, the average age of the subjects was 39.91 ±13.665 years, and the average disease onset was at 25.71±13,743 years. This substantial gap indicates not only a delay in diagnosis and under-recognition of hidradenitis suppurativa among healthcare providers, but it may also reflect a tendency among patients to delay seeking medical treatment. The hypothesis claims that HS onset often coincides with or shortly follows puberty. An association has been noted between HS and a lower socioeconomic status, which could be partially linked to an increased prevalence of HS risk factors within this demographic group, or alternatively, it could arise as a result of HS itself (5). A retrospective Dutch study found that despite the fact that HS is more common in women, men tend to suffer from a more severe form of the disease (206). Our study shows similar results, with females facing a longer diagnostic delay, which indicates milder disease.

5.2. Comorbidities

Patients with HS experience an exceptionally elevated burden of comorbidities (207,208). Dyslipidaemia, hypertension, obesity, thyroid disorders, psychiatric disorders, arthropathies, and polycystic ovarian syndrome have each shown independent associations with HS (207). Although the precise relationship between HS and its comorbidities is not fully understood, numerous related conditions are characterized by inflammation as well (209). Regardless of age, gender, socioeconomic status, smoking, and medication use, patients with HS experience a notable rise in major adverse cardiovascular events such as ischemic stroke, myocardial infarction, and cardiovascular-related death, as well as overall mortality. Elevated levels of C-reactive protein and tumour necrosis factor–α, which have been linked to atherosclerosis, are also heightened in HS (118). The

connection between chronic inflammation and HS is observed in association with metabolic syndrome, a condition that comprises diabetes mellitus, dyslipidaemia, hypertension, and obesity (93,115,210,211). For both hospitalized and nonhospitalized individuals with HS, the odds ratios for being diagnosed with metabolic syndrome, as compared to healthy patients, are 3.89 and 2.08, respectively (115). In our study, a similar tendency was observed: 30.6% of the patients had comorbidities associated with cardiovascular disease, and among them, 60% had a positive family history of inflammatory diseases.

An association also exists between HS and psoriasis. In our study, psoriasis was present in 8.16% of HS patients. An analysis involving 68.836 psoriasis patients revealed a higher prevalence of HS in comparison to sex-, age-, and ethnicity-matched control subjects (0.3% versus 0.2%). Psoriasis patients who have concurrent HS tend to be younger and exhibit a greater prevalence of obesity and smoking (212).

Moreover, HS has been linked to various other conditions characterized by a proinflammatory state. Inflammatory bowel disease (IBD), notably Crohn's disease (CD), exhibits potential epidemiological and pathogenic correlations with HS (213,214). The analysis of four studies indicated that the prevalence of HS among patients with IBD and CD was found to be 12.8% and 17.3%, respectively. Moreover, HS populations exhibit a higher prevalence of inflammatory arthritis compared to the general population (208,215). In a prospective study, 3.7% exhibited concurrent spondylarthritis as a comorbidity, with HS preceding articular symptoms in over 90% of those individuals (215). Consistent with the findings in the literature, our study showed that comorbidities were present in 61.22% of the patients. 6.12% of patients had comorbid inflammatory bowel disease, 12.24% presented with joint disorders, 2.04% with metabolic disorders, 12.24% with dyslipidaemia, and 29.4% with hypertension. When assessing the severity of HS, a statistically significant correlation was identified between metabolic comorbidities and the Hurley stage, with 55.0% of those comorbid cases classified as Hurley stage III. This tendency was also outlined in the study by *Kimball et al*., which asserted that the higher the severity of the disease, the greater the burden of comorbidities, particularly those such as a sebaceous cyst, pilonidal cyst, kidney disease, hypertension, diabetes, deficiency anaemia and congestive heart failure (216).

Certain dermatological conditions display a shared cutaneous pathology with HS. Follicular occlusion, hyperplasia of the pilosebaceous apparatus, and bacterial invasion are contributing factors in acne conglobate, dissecting cellulitis, as well as HS. Collectively, these conditions form the

follicular occlusion triad (217). Inclusion of the pilonidal cyst completes the follicular occlusion tetrad (218). Acne and HS can also be elements of autoinflammatory syndromes. The presence of acne, pyoderma gangrenosum, and hidradenitis suppurativa (PASH) together defines an established syndrome. In cases where a patient has PASH along with the inclusion of pyogenic arthritis, the condition is labelled as PAPASH (219). Ultimately, persistent HS has the potential to progress into squamous cell carcinoma. A link to dermatological conditions has been observed in our study as well. Severe acne was evident in 34.69% of the participants, and among them, it remains ongoing in 64.70% of cases. These numbers in the general HS population could be even higher, considering that the prevalence of acne is higher among females than males (similar to the higher prevalence of HS in the Western population), yet our study had a higher proportion of males than females (female-to-male ratio 0.75:1) (220,221).

5.3. The association between squamous cell carcinoma and HS

Approximately 4.6% of HS cases are linked to the presence of squamous cell carcinoma (222). According to a systematic literature review by Gierek et al., the majority of patients who develop SCC in previously existing HS lesions are middle-aged Hurley III stage males with a longstanding disease history (126), which aligns closely with our patient's profile. In these cases, SCC is usually well differentiated and often localizes in the gluteal region. In a systematic literature review, only 5 patients out of the 74 cases described were on immunosuppressive treatment while developing SCC (126); therefore, we do not consider TNF treatment as the only risk factor contributing to the development of a malignant lesion in our presented case. However, further research is needed to determine the potential impact of biologics on the development of cancerous lesions within this patient population (125).

Given that SCC is a rare complication of longstanding HS, clinical decision-making is not standardized, and management typically relies on case reports and expert opinion (223). Due to the aggressive nature of SCC in HS cases, an aggressive approach is almost always warranted. The timely and routine screening of individuals with hidradenitis for this complication is of utmost importance. The delay in diagnosis is possible as SCC may be missed in chronic HS lesions. Therefore, a low threshold for repeated deep tissue biopsies in non-healing lesions is recommended (124,127,224). The most commonly described morphology of SCC lesions were tumorous growth and ulcerations (126).

In cases of histologically confirmed SCC, appropriate imaging techniques such as magnetic resonance imaging and positron emission tomography should

be employed. Additionally, sentinel lymph nodes should be evaluated. The treatment of first choice is a large and deep surgical excision with a minimum margin of 2 cm. However, performing such excisions can be challenging in patients with chronic wounds due to severe tissue defects, and the risk for metastatic disease and recurrence is high (125,127). Patients should be managed by a multidisciplinary team that includes oncologists for consideration of radiotherapy and reconstructive surgeons to discuss wound closure options. Chemotherapy has not shown efficacy in these cases (125). The benefits of using topical negative pressure to promote wound healing and granulation tissue production were described in the literature (225) and utilized in our case.

Chemoradiotherapy is the standard treatment for perianal SCC, allowing for anal sphincter preservation and avoiding surgical intervention (226). Nonetheless, there is a 20–30% failure rate, leading to disease recurrence in approximately 10–15% of cases (227,228). In these instances, abdominoperineal resection of the rectum is required to manage the disease and lower the risk of mortality (229). The literature presents a clinical case in which a 58-year-old man who developed a lesion in the perianal region close to the HS lesions. On physical examination, an ulcerated, painful, and friable lesion was found in the right perianal region, indicative of SCC originating from an HS scar. The patient received chemotherapy and radiotherapy; however, the lesions recurred, requiring abdominoperineal resection of the rectum (***Figure 23 and 24***) (230).

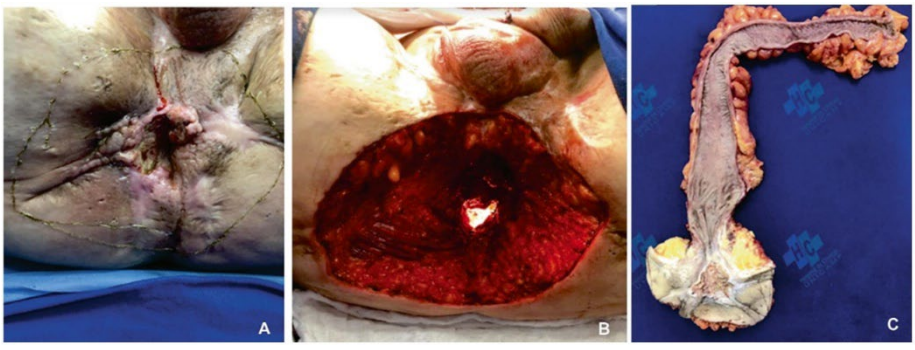


Figure 23. Well to moderately differentiated squamous cell carcinoma with extensive chorion fibrosis. (A) Surgical margin delineation. (B) Perineal area following neoplasia resection. (C) Resected specimen. Filho ASM at al. Squamous cell carcinoma arising in chronic hidradenitis suppurativa: A case report and comprehensive literature review. International Journal of Surgery Case Reports. 2024 Oct 1;123:110271.

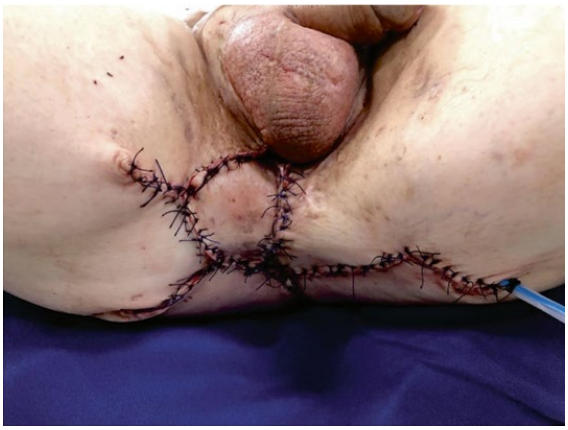


Figure 24. Immediate postoperative appearance showing total excision of the lesion and perineal reconstruction with a V-Y flap on the right side and a rotation flap on the left. Filho ASM at al. Squamous cell carcinoma arising in chronic hidradenitis suppurativa: A case report and comprehensive literature review. International Journal of Surgery Case Reports. 2024 Oct 1;123:110271.

In regards to SCC prevention, HPV vaccination should be considered, as it has been described as a potential risk factor (126). Optimal intervals for cancer screening have not been established; therefore, vigilant surveillance for potential transformation should be maintained at every patient visit, with thorough full-body examinations performed regularly (127).

5.4. Smoking and obesity

HS is highly influenced by two environmental factors: smoking and obesity. A thorough analysis revealed a significant correlation: the odds ratio (OR) for current smoking and HS stood at 4.34, while for obesity and HS, the OR was 3.45 (72). Increased smoking pack-years and elevated BMI have both been associated with more pronounced disease severity (206). While obesity and smoking are acquired disease factors, their role in disease development might be connected to the suppression of Notch signalling. Downregulation of the Notch pathway has been documented in individuals who smoke, and this pathway also plays a role in regulating metabolism. The disruption of this pathway results in an impaired innate immune response, which could provide insight into the immune dysregulation believed to be a key factor in both the development and clinical manifestation of HS (231).

According to *Konig et al*., the prevalence of active cigarette smokers among HS patients was close to 90% (232). Based on these results, *Breitkopf*

and his team discovered that the occurrence of smoking behaviours among male and female patients was 85% and 84% respectively (233). *Dessinotti et al*. revealed that a mere 16% of HS patients had not smoked at any point (63). The connection between tobacco smoking and disease severity among HS patients has also been evaluated. *Sartorius at al*. observed that individuals who smoked exhibited greater disease severity, as measured by the modified Hidradenitis Suppurativa Score (mHSS), when compared to non-smokers (234). The impact of cigarette smoking on disease management is noteworthy, as research revealed that not smoking was associated with higher chances of responding positively to initial treatment and a greater frequency of self-reported remission from HS (235,236). Our analysis yielded comparable results, with 59.18% of our participants having a history of active or past smoking. Moreover, a statistically significant association was discovered between being diagnosed with Hurley III and having a smoking history, as 91.66% of Hurley III patients were smokers. Taking these factors into account, active tobacco smoking could be regarded as a potential risk factor for HS (237). As previously indicated, cigarette smoking might also play a role in providing insight into the gender distribution of HS (238). Our study found a significantly higher proportion of male smokers compared to female smokers, with 78.57% of men and 33.33% of women identified as smokers. This disparity may help explain the predominance of male participants in the study. Conversely, cigarette smoking could potentially be a result of the disease, as patients might turn to smoking as a means to alleviate the anxiety and depression often connected with HS (68,69).

Between 50% and 75% of HS patients fall into the overweight or obese category (209). In our study, the average BMI was 27.84±7.362. Among the participants, 30.61% were categorized as overweight and 36.73% fell into the obese category. Multiple studies have shown a correlation between higher BMI and increased severity of HS. In two case-control studies, it was observed that for every unit increase in BMI, there was a 1.12-fold higher risk of developing HS (239). Other research examining the impact of weight loss on HS severity revealed notable findings: a substantial decrease in the proportion of patients experiencing HS symptoms (up to 35%) after weight loss and a decrease in the number of affected anatomical sites from 1.93 to 1.22 (75,76). Therefore, it can be reasonably deduced that bariatric surgery, an intervention for severe obesity, holds the potential to alleviate the impacts of inflammatory skin conditions like HS (240). In recent developments, semaglutide and liraglutide, both recognized as glucagon-like peptide-1 receptor analogues (GLP-1RA) and authorized for managing diabetes and obesity, have demonstrated efficacy in not only causing notable weight loss but also in

reducing several obesity-related comorbidities (241,242). Two case reports demonstrated a decrease in the clinical severity of HS following the administration of liraglutide, accompanied by significant weight loss (243,244). In a phase 3 trial involving individuals with obesity or overweight, the utilization of tirzepatide, a dual agonist targeting both GLP-1 receptors and another incretin, gastric inhibitor polypeptide (GIP), led to weight reduction of as much as 20.9% over an 18-month period (245). Furthermore, the antidepressant bupropion showed a dose-dependent decrease in weight among individuals categorized as obese or overweight (246,247). Therefore, these medications could be offered as an adjunctive treatment in overweight and obese HS patients.

5.5. Duration until a diagnosis is determined

The difficulty in diagnosing HS clinically arises from the extensive diversity in the disease's presentation and the inconsistent responses to suggested treatment plans (248). The delay in diagnosing HS can lead to prolonged patient distress, hinder the gathering of epidemiological information, and result in inadequate health results. As a result of the resemblance of early hidradenitis suppurativa stages to other conditions, on a global scale, the average diagnostic delay ranges from 7 to 10 years (5,249–251). This is similar to what we observed in our study, with the mean time to diagnosis being 5.2±7.607 years with most diagnoses being made by a dermatologist. As there are no definitive tests available, the diagnosis of HS relies on clinical observation and the patient's description of their condition (89). The research conducted in Germany demonstrated a positive association between the duration of diagnostic delay and the number of medical practitioners consulted by the patient prior to receiving an HS diagnosis. Consequently, individuals with the most prolonged HS diagnostic delay had, on average, consulted nearly 5 physicians. Among the patients, the primary medical contacts were general practitioners, followed by dermatologists, surgeons, and gynaecologists. Furthermore, individuals with delayed diagnosis encountered an average of nearly 5 instances of misdiagnosis. In general, there exists a positive correlation between the duration of diagnostic delay and the frequency of misdiagnoses. Common erroneous assessments included abscesses, ingrown hairs, and folliculitis. Other incorrect diagnoses comprised conditions like *acne conglobata* or even *acne vulgaris* (103). Our study drew analogous conclusions, as 70.2% of patients were misdiagnosed, and the diagnoses included furuncle, ulcer, abscess, acne, and allergy. The definitive HS diagnosis for each patient was confirmed following their visit to

the VUH SK Centre of Dermatovenereology. The multicenter, epidemiological, non-interventional cross-sectional study also showed that an accurate diagnosis of HS is achieved for the majority of cases by a dermatovenereologist (103). Misdiagnosis is correlated with factors such as non-white race, heightened disease severity, and an increased number of comorbidities (103,252). Due to the involvement of intimate body areas, individuals with severe disease may be hesitant to reveal their symptoms or seek medical attention, influenced by feelings of shame or fear (253).

5.6. Clinical presentation and subtypes of HS

According to the adapted *Dessau* definition, three diagnostic criteria need to be fulfilled: the existence of characteristic lesions, occurrence in typical areas, and chronicity (89). Characteristic lesions encompass deeply situated, painful nodules, suppurative sinus tracts or channels, abscesses, and connected scars, as well as double- and multi-ended comedones, often referred to as "tombstone comedones" (254). Abscesses and subcutaneous nodules have the potential to rupture, leading to bleeding and the release of a purulent discharge. This ongoing progression ultimately culminates in dermal contraction and fibrosis of the affected skin. The axillary, infra-, and inframammary; inguinal; perineal; and gluteal regions are frequently impacted anatomical areas (255). Less commonly mentioned sites include the lower abdomen, suprapubic area, retroauricular region, eyelids, nape, and scalp (77,256,257). A defining characteristic of HS is its chronic nature, marked by two recurrences within a 6-month period. These three criteria by themselves generally exhibit a high level of diagnostic sensitivity and specificity (258). Our research showed that in terms of lesion distribution, 75.51% were situated in the axillary region, 59.18% in the groin area, 28.57% in the pubic area, and 26.53% in other areas. No significant gender-based differences were observed. Among the obese participants, 88% exhibited involvement in the groin area.

Various phenotypes of HS exist. The research conducted by Canoui-Poitrine *et al*. identified the presence of three distinct phenotypes: “axillary mammary”, “follicular”, and “gluteal”, with the majority (48%) having the “axillary mammary” phenotype (201). In our research, 40.81% of participants displayed the “axillary mammary” phenotype, which exhibited a high prevalence of armpit and breast involvement, as well as hypertrophic scars. The remaining two phenotypes, labelled as “follicular” and “gluteal”, represent atypical variations of the condition (201,259). In our study, the “follicular” phenotype, characterized by the presence of follicular lesions,

including epidermal cysts, pilonidal sinus, and comedones, along with severe acne, was found in 34.69% of cases. The "follicular" phenotype showed a greater proportion of non-smokers in comparison to the "axillary mammary" phenotype, while in Canoui-Poitrine *et al.*'s study, the "follicular" phenotype was identified in 26% of patients, with a higher proportion of male participants and individuals who were current or former smokers (201). In our investigation, this phenotype also had a higher number of Hurley III subjects than the "gluteal" and "axillary mammary" phenotypes. The Canoui-Poitrine *et al*. study also confirmed that patients with the "follicular" phenotype experienced more severe symptoms, earlier onset of the condition, and a longer duration of illness compared to the typical "axillary-mammary" phenotype (201). In our research, 24.48% of patients showcased the gluteal phenotype, which is marked by engagement with the gluteal region, the presence of follicular papules, and the development of folliculitis. According to Canoui-Poitrine *et al*., this phenotype had a higher proportion of smokers, a lower average BMI, and, surprisingly, less severe symptoms, despite a longer duration of HS compared to the "axillary-mammary" phenotype (201). Our results show a lower proportion of smokers (42%) in this group and milder disease, with 50% of subjects being Hurley I and only 17% – Hurley III.

5.7. Patients' quality of life

Jørgensen *et al*. showed that there is a noticeable distinction in the average total DLQI score among the different Hurley severity groups. In their study, the mean Hurley score for people with HS was 11.9. Patients with more severe disease had a higher mean total DLQI score: 8.6 for Hurley I, 12.4 for Hurley II, and 16.1 for Hurley III. Additionally, they exhibited significantly elevated mean scores for each of the ten individual DLQI questions. Moreover, a higher overall DLQI score was linked to several factors, including younger age (below 60), unemployment, smoking, experiencing boils in the past month, having a higher boil-associated pain score, greater overall disease-related distress score, involvement of multiple anatomical regions, and specific locations such as the axillary, groin, and gluteal regions, as well as the presence of diabetes (260). In addition, the DLQI and physician-assessed International HS Severity Score System (IHS4) scores demonstrated an upward trend with increasing disease severity according to the refined (seven-stage) Hurley classification. There was a noteworthy positive correlation between both DLQI and IHS4 scores and the progression of severity as evaluated by the refined Hurley substages (261). In our study,

patients expressed concerns about their health, with an average anxiety level score of 6.5 out of 10. Those with elevated anxiety also experienced a noticeable impact on their sleep quality. The average DLQI score at the beginning of the study was 8.9 for all subjects. Of the patients, 32.65% had a DLQI score exceeding 10; 62.5% of these were male, 93.75% had a BMI above 25, and their average anxiety level score was 7.5. Furthermore, individuals experienced an average of 6.17±6.98 days of pain in the past 4 weeks. The average pain intensity, as assessed by the VAS scale, was 5.60±3.36. Among the participants, 30.61% reported consistent or persistent pain, while 69.38% described their pain as intermittent. Male patients exhibited a statistically significant tendency to experience intermittent pain more frequently.

5.8. Treatment

For individuals in Hurley stages I/II who have mild and localized HS marked by a small number of lesions, the use of topical clindamycin 1% is recommended when there are no deep-seated inflammatory lesions present (139). The majority of our subjects were prescribed topical clindamycin with benzoyl peroxide but with limited efficacy. The use of intralesional steroids can be beneficial for managing acute inflammatory nodules in conjunction with other therapies across all stages of Hurley classification (262), and more than 20% of our subjects were previously treated with intralesional triamcinolone at 10 mg/mL, which helps to promptly reduce inflammation in HS lesions. For patients classified under Hurley stages I/II with multiple lesions and frequent worsening of symptoms, systemic tetracyclines can be used for 10–12 weeks (263,264). For patients in Hurley stages II/III who have multiple ongoing lesions, the recommended treatment involves systemic clindamycin with rifampicin 300 mg twice daily for 10 weeks (265–268). In our research, 65.30% of participants were undergoing long-term systemic antibiotic treatment with doxycycline or clindamycin/rifampicin. Patients on systemic antibiotics showed a mean baseline DLQI of 10.5, decreasing to 9.2 at follow-up, with initial and follow-up IHS4 scores of 6.21 and 5.42, respectively. In cases of moderate to severe HS where conventional treatments have proven ineffective, adalimumab should be regarded as the first choice among biologic agents (269–271). The use of adalimumab for HS was evaluated in the PIONEER I and II trials. The clinical response rates at week 12 were notably greater in the groups receiving weekly adalimumab compared to the placebo. Specifically, in PIONEER I, it was 41.8% versus 26.0%, and in PIONEER II, it was 58.9% versus 27.6% (271). In our study, 22.44% of the

patients were treated with adalimumab. The patients on biologics had a mean DLQI of 7.4 at baseline and 5.1 at follow-up. Regarding the initial IHS4 score, the patients on biologic treatment had a mean score of 7.38, which decreased to 3.22 at follow-up. However, 29.78% of participants experienced lesion recurrence in the last 4 weeks, with no significant differences observed between those on biologics and those on systemic antibiotics.

Infliximab has demonstrated its effectiveness and could be taken into consideration as a secondary biologic option for individuals with moderate-to severe-HS; however, it still does not have an official indication for HS (272) and its efficacy is usually of limited duration due to the neutralizing antibody formation (273,274).

Translational research has revealed that IL-17A plays a pivotal role in HS (275). Initially, case reports and open-label studies published after 2018 demonstrated clinical enhancements in patients treated with secukinumab. Two identical (SUNSHINE and SUNRISE), multicentre, randomized, placebo-controlled, double-blinded phase 3 trials were performed. In both trials, secukinumab demonstrated a clinical response rate of 42-46%, with better efficacy observed with biweekly administration, compared to 31-34% in the placebo group (276). It was approved for HS treatment by the European Medicines Agency in June, 2023. In our study, five patients were administered secukinumab treatment, but the investigation into its efficacy was not pursued due to the limited number of subjects.

5.8.1. Adalimumab

Despite the availability of therapeutic choices, managing HS remains challenging due to the considerable number of cases that do not respond to treatment (277). The effectiveness of adalimumab in treating individuals with moderate-to-severe HS unresponsive to traditional treatments has been extensively established through two 12-week controlled clinical trials and two extension studies, suggesting its viability as a suitable option for medium-to-long-term management of HS (161,278,279). HiSCR, a validated measure of outcome, is endorsed by robust evidence and is recommended for assessing the effectiveness of treatments in controlling inflammatory lesions in patients with HS (203).

Since adalimumab is prescribed for moderate-to-severe HS, patients typically have a long history of the disease and have often tried multiple treatments. In a multicentre observational study conducted in Japan, 39.8% of patients had HS for ≥10 years, and 84.3% of patients had previously received pharmacological treatment for HS before initiating adalimumab therapy (280).

In our study, patients had HS for an average of 15.48 years. In Lithuania, adalimumab is typically prescribed only after systemic antibiotic treatment has proven ineffective, so all patients had previously used antibiotics before starting adalimumab. Our study shows the efficacy of adalimumab in clinical practice at the VUH SK Centre of Dermatovenereology in Lithuania. A clinical response to adalimumab, evaluated by HiSCR after one year of treatment, was observed in 47.62% of patients, aligning with the results from previously documented controlled clinical trials (161,278,279). These findings closely resemble those of a retrospective, real-life multicentre cohort study conducted in Italy, where 53.9% of patients attained HiSCR after one year (52 weeks) of treatment. Throughout adalimumab treatment, there was a significant decrease in the abscess, nodule, and fistula count. The outcomes align with the response observed in the study by Smetanova *et al*., where after 12 months of treatment, the reduction in mean values was as follows: inflammatory nodules decreased from 8.15 to 2.83, abscesses from 2.98 to 0.67, and draining fistulas from 4.96 to 1.43 (281). In our study, inflammatory nodules decreased from 5.62 to 3, abscesses from 1.76 to 0.81, and fistulas from 2.62 to 2.

The IHS4, often integrated as a secondary outcome measure, serves as a straightforward validation system adapted for routine clinical practice (282). We also employed this scoring system to evaluate therapeutic responses within our study group. A mean IHS4 score dropped from 19 at the baseline to 12.62 after one year of adalimumab treatment, and the proportion of severe HS decreased from 66.7% to 52.4%. A similar result was obtained by Chiricozzi *et al*., indicating that the mean IHS4 score after 52 weeks of adalimumab treatment decreased from 22.2 to 14.1, and the proportion of severe HS decreased from 85.4% to 50% (283).

In our study, there was a notable decrease in the baseline DLQI and VAS pain scores following one year of adalimumab treatment, with mean scores dropping from 15.76 to 7.43 and from 6.69 to 3.64, respectively. Smetanova *et al*. observed a similar trend, with the mean DLQI estimate decreasing from 17.61 to 6.08 (281), while Chiricozzi *et al*. reported a decrease in the mean VAS score from 5.5 to 2.7 (283).

In our study, the mean BMI of the patient receiving adalimumab was 30.33 kg/m^2, and only 23.8% of them had a normal BMI. Although our study did not find a statistical association between BMI and the achieved clinical response (HiSCR), disease severity (IHS4), DLQI, and VAS, other studies suggest that higher BMI is linked to a more severe disease course and a poorer response to adalimumab treatment (87,284). Weight loss and bariatric procedures are linked with a notable decrease in HS severity; however, it may

exacerbate symptoms if it leads to a substantial increase in skin folds, necessitating the excision of excess skin (285,286).

5.8.2. Surgery

A range of surgical interventions are at one's disposal, and there is not a single optimal treatment, necessitating a personalized strategy for every patient (287).

Incision and drainage should not be seen as a standalone treatment option, as recurrence is nearly certain (182–184,288). Surgical interventions, including limited excision, deroofing, and Skin-Tissue-sparing Excision with Electrosurgical Peeling (STEEP), can be employed for isolated lesions. In Hurley stage III, a comprehensive excision of the entire affected region, involving the removal of both inflamed and non-inflamed sinuses, nodules, and scar tissue, may be conducted as a preventative measure against recurrence (194,289,290). The use of biologic therapy before and after surgery could result in a decreased likelihood of recurrence and a longer period free from the disease (291–293). In our study, a surgical procedure was performed for most patients receiving adalimumab (66.7%), with those patients having a significantly higher mean IHS4 score than patients who did not have surgery, at 23.86 and 9.29, respectively. This suggests that surgical treatment was more frequently administered to patients with a more severe form of the disease. Due to frequent recurrences of the disease, there has been a growing interest in utilizing targeted biological therapy for managing HS (294,295). The study by *Shanmugam et al*. demonstrated that biologic therapy is linked to a quicker reduction in disease activity, particularly notable in patients who also underwent surgery for HS (197). *Aarts et al*. study found that the combined approach of adalimumab with surgery resulted in notably greater clinical effectiveness and enhanced quality of life compared to adalimumab monotherapy, accompanied by increased patient satisfaction. Following 12 months of treatment, the surgery group demonstrated a significantly greater decrease in IHS4 scores than the monotherapy group (mean reduction of −9.1 versus −7.8). Moreover, the surgery group experienced a more substantial decrease in DLQI scores after treatment compared to the monotherapy group (mean reduction of −8.2 versus −4) (198). In our study, no significant difference was observed in the achieved clinical response (HiSCR) between individuals who received surgical treatment before adalimumab administration and those who did not; however, we did not investigate the effect of surgical treatment during adalimumab treatment.

6. LIMITATIONS

This study has several limitations. It was conducted at a single site with a relatively small sample size, which may limit the generalizability of the findings. Due to low awareness of HS in Lithuania, enrolling subjects was challenging, resulting in a limited scope for data collection. Nearly all patients who visited the VUH SK Centre of Dermatovenereology during the studied years were included, yet the sample size remains modest. A larger cohort would likely provide more robust and statistically reliable data, offering a better understanding of HS trends and patterns. Future research should aim for multi-center studies with larger, more diverse populations to strengthen the reliability and applicability of the findings.

7. STRENGTHS AND PERSPECTIVES

Currently, it is the first study in Lithuania analysing the demographics, risk factors, clinical presentation, subtypes and treatment efficacy in such depth and vast number of data points of Lithuanian HS patients. The high impact HS has on quality of life, anxiety, sleep quality and pain associated with HS symptoms highlights the need of early and effective treatment. It can also be used as an example of disease under-recognition among local specialists and the severe burden all the comorbidities create on the national healthcare system. 31 of our subjects also consented to skin biopsies being taken from their HS lesions and healthy-looking skin, with a total of 58 samples collected. The collected skin tissue samples with such an extensive data set will be a solid starting point of future translational research in HS.

The EHSF (European Hidradenitis Suppurativa Foundation) is working together with Regeneron Pharmaceuticals, Inc., a US-based biotechnology company, to identify distinct molecular biological patterns by using in-depth genomic and molecular profiling of HS. The subject biopsies collected from our study will be included in a pilot sub-study by Regeneron (0000-HS-CES-2379), titled Analysis of Histopathology and In Situ Biomarker Localization in Hidradenitis Suppurativa (HS) (**Appendix IV**). The aim of this study is to explore biomarkers and molecular signatures in the localized tissue environment of HS. Histopathological and multiplex immunohistochemical (IHC) analysis will be performed on retrospectively acquired samples from patients with HS. As part of the study, technical methodologies will be validated to analyse samples from HS for future studies. Multiplex IHC uses multiple stains allowing simultaneous detection of multiple markers on a single tissue section to visualize distinct cells, extracellular components and their interaction.

The finalization of the contract and material transfer agreement took a lot more time than initially anticipated, the signature process by Vilnius University and Regeneron was completed on 13th SEP 2024. The samples were shipped and arrived to Regeneron in October and initial results are expected in 2025.

One example of how multiplex IHC technology can be used in HS research has highlighted the role of neutrophil extracellular traps (NETs) and panniculitis-associated dermatoses (PADs) in the disease's inflammatory process. In HS, the excessive formation of NETs—DNA and antimicrobial proteins released by neutrophils—contributes to immune dysregulation, amplifying inflammation and leading to tissue damage, abscesses, and fibrosis (296,297). Understanding how NETs drive disease progression could open

new therapeutic avenues, such as targeting NET formation with specific inhibitors or modulating neutrophil activity to reduce inflammation and improve patient outcomes. Additionally, PADs, characterized by inflammation of subcutaneous fat, have been implicated in HS pathogenesis, particularly in severe cases. The inflammation within the deeper layers of the skin and fat tissue may trigger systemic immune responses, sustaining chronic inflammation and worsening disease (298,299). Exploring the links between PADs and HS could lead to treatments aimed at modulating adipose tissue inflammation or immune cell infiltration. Overall, future research focusing on NETs, PADs, and immunomodulatory therapies holds promise for advancing HS treatment and improving patient quality of life.

8. CONCLUSIONS

a) HS often begins in the mid-20s, with a 5.2-year diagnostic delay, frequently due to misidentification as furuncles, especially in females. *This aligns with existing data, which also highlights the typical onset in early adulthood and diagnostic delays due to misdiagnosis.*
b) Many patients are overweight or obese, with smoking linked to higher disease severity, which coincides with previous studies.
c) One-third report a DLQI >10, highlighting significant quality-of-life impact, particularly in males. Younger patients show higher anxiety levels. *Our study adds the novel insight that the disease affects men more severely than women, with younger patients experiencing heightened anxiety.*
d) Family history of inflammatory diseases and metabolic comorbidities are linked to higher HS severity. Lesions most commonly affect the axillary and groin regions, regardless of gender. *This observation is novel, as no studies in Lithuania have previously examined the connection between family history and metabolic comorbidities with increased HS severity, offering new insights into potential genetic and metabolic risk factors.*
e) Adalimumab reduces inflammatory lesions and improves the quality of life. Prior surgery is associated with higher baseline disease severity. *These results are consistent with global data on adalimumab's effectiveness, while also emphasizing the need for more focused attention on developing more effective treatment options for HS*

9. PRACTICAL RECOMMENDATIONS AND FUTURE DIRECTIONS

- Early diagnosis of hidradenitis suppurativa is crucial for improving patient outcomes. It is essential to increase awareness of HS among other healthcare specialists in Lithuania, enabling more accurate and timely diagnosis. This can be achieved through professional education, training, and awareness campaigns to help dermatologists and other specialists recognize HS at an early stage and refer patients for appropriate treatment
- Correction of the two main risk factors – smoking and obesity – are crucial for better disease control. New developments in weight loss medications could aid HS management in close collaboration with endocrinologists, dietologists and bariatric surgeons, smoking cessation can be managed with the help of psychiatry and psychotherapy.
- The early use of biologic therapy can play a key role in halting disease progression in HS patients. Initiating biologic treatments at an early stage can help control inflammation and prevent the development of severe lesions and complications, ultimately improving the patient's quality of life and reducing the need for more intensive interventions later.
- A combined approach using both biologic therapy and surgical treatment may yield better results for managing HS, especially in patients with advanced disease.
- The development of a HS centre in Lithuania could shorten the diagnostic delay by raising disease awareness and offering a multidisciplinary approach utilizing modern treatment, surgery, pain management, weight loss and mental health.
- Continued research and the development of new medications targeting different aspects of the disease's pathophysiology are essential for addressing the needs of patients with hidradenitis suppurativa. Exploring novel therapies that could provide additional options for patients who do not respond adequately to existing treatments

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APPENDIX I. EHSF Registry questionnaire

Pūlingo hidradenito Europos registras. Pirmo apsilankymo apklausa (2020 02 17 versija)

A1. Paciento LIN: _____
A2. Pildymo data: _____
B. SOCIODEMOGRAFINIAI DUOMENYS
B1. Lytis : ☐ Moteris ☐ Vyras
B2. Gimimo data:_____ (MMMM-mm-dd)
B3. Svoris: _____kg
B4. Ūgis:____cm
B5. Etninė kilmė (galimi keli atsakymai):

☐Baltosios rasės	☐Juodosios rasės
☐Kaukaziečių	☐Azijos
☐Indėnų (vietinių amerikiečių)	☐Havajų
☐Viduržemio	☐Kita :_____
☐Šiaurės Afrikos	

B6. Odos fototipas:
☐I (balta, blyški oda, mėlynos/rusvos akys, šviesūs/raudoni plaukai, oda visada nudega saulėje, neįdega)
☐II (šviesi oda, mėlynos akys, oda lengvai nudega saulėje, įdega sunkiai)
☐III (tamsesnė šviesi oda, įdega po pradinio nudegimo)
☐IV (šviesiai ruda oda, nudega retai, įdega lengvai)
☐V (ruda oda, retai nudega, lengvai susidaro tamsus įdegis)
☐VI (tamsiai ruda ar juoda oda, niekada nenudega, visada susidaro tamsus įdegis)
B7. Plaukų struktūra:

☐tiesūs	☐garbanoti
☐banguoti	☐nežinoma

B8. Gyvenoji vieta: ☐Kaimas ☐Miestas
B9. Vedybinis statusas:
☐Nevedęs (netekėjusi)
☐Susituokęs(-usi)/turi sugyventinį(-ę)
☐Išsiskyręs(-usi)/gyvena atskirai
☐Našlys(-ė)
B10. Profesinis statusas:
☐Dirbantis(-i) pilnu darbo krūviu/Studentas(-ė)
☐Nedarbingumo atostogose
☐Neįgalumas ☐Reabilitacijoje
☐Pensininkas(-ė) ☐Bedarbis(-ė)
B11. Išsilavinimas:
☐Pagrindinis (7-10 klasių)
☐Profesinis ☐Vidurinis
☐Aukštasis (kolegijoje/universitete) (mažiau nei 4 m.)
☐Aukštasis (kolegijoje/universitete) (4 m. ir daugiau)
C. GYVENIMO ANAMNEZĖ
CA1. Ar esate sirgęs(-usi) sunkia aknės forma?
☐Taip ☐Ne
Jei taip, koks buvo izotretinoino efektyvumas?

☐Visiškas	☐Jokio efekto	☐Nebuvo naudojama
☐Dalinis	☐Sunkinantis būklę	

Jei taip, kokia buvo aknės simptomų chronologija:
Kiek jums buvo metų, kai prasidėjo bėrimas? ___ m.
Kiek jums buvo metų, kai aknei būdingas bėrimas baigėsi?
___ m. ☐Aknės bėrimas vis dar yra
CA2. **Ar sergate žvyneline (psoriaze)?**
☐Taip ☐Ne
CA3. **Ar sergate uždegimine žarnyno liga?**
☐Ne ☐Taip *Jei taip, kuri tai buvo liga?*
☐Krono liga ☐Opinis kolitas ☐Kita: _____
CA4. **Ar esate turėjęs(-usi) pilonidinį sinusą/cistą?**
☐Ne ☐Taip *Jei taip, kiek jums buvo metų?* ____ m.
CA5. **Ar esate sirgęs(-usi) depresija?**
☐Taip ☐Ne
Jei taip, ar depresija buvo susijusi su jūsų liga?
☐Taip ☐Ne
CA6. **Ar esate sirgęs(-usi) artritu/buvę skausmingi sąnariai?** ☐Ne ☐Taip **Jei taip, koks artritas tai buvo?**

☐Reumatoidinis artritas	☐Ankilozinis spondilitas
☐Osteoartritas	☐Podagra
☐Tendinitas	☐Lumbalgija/rachialgija

Jei taip : Ar būdingas rytinis sustingimas?
☐Taip ☐Ne ☐Nežinoma
Koks atsakas į nesteroidinius vaistus nuo **uždegimo?** ☐Geras ☐Blogas ☐Nežinoma

☐Daktilitas	☐Adamantiades-Behçet liga
☐Sarkoidozė	☐Kita: _____

Jei taip, kuris sąnarys buvo labiausiai paveiktas artrito?
☐Lokacija (Prašome patikslinti): _____
CA7. **Ar galite sau priskirti bent vieną iš paminėtų būklių? (galimi keli atsakymai)**

☐Dislipidemija	☐Išeminė širdies liga
☐Cukrinis diabetas	☐Cerebrovaskulinė liga
☐Hipertenzija	☐Metabolinis sindromas
☐Širdies ritmo ar laidumo sutrikimai	☐Nei vienas iš paminėtų

CA8. Ar vartojate vaistus, mažinančius cholesterolio koncentraciją kraujyje? ☐Taip ☐Ne
Jei taip, kokie tai vaistai?
☐Statinai
☐Kiti (veiklioji medžiagą):_____
CA9. Ar vartojate vaistus, mažinančius triglicerido koncentraciją kraujyje? ☐Taip ☐Ne
Jei taip, kokie tai vaistai?
☐Fibratai
☐Kiti (veiklioji medžiagą):_____
CA10. Ar esate sirgęs(-usi) lėtine dantų ar dantenų liga:
☐Taip ☐Ne **Jei taip:**
☐Dantenų uždegimas ☐Abscesas
☐Dantenų kraujavimas ☐Dantų netekimas
☐Kita (prašome patikslinti): _____
CA11. Ar galite sau priskirti bent vieną iš paminėtų būklių? (galimi keli atsakymai)
☐Limfoma ☐Gangreninę piodermija

☐Plokščių ląstelių karcinoma PH pažeidimo vietoje
☐Egzema (prašome patikslinti): _____
☐Lengva/vidutinio sunkumo aknė
☐Rožinė
☐Kitos odos ligos (prašome patikslinti): _____
☐Skrandžio opa
☐Stemplės išvarža arba gastroezofaginis refliuksas
☐Hepatitas, etiologija (patikslinti): _____
☐Fibromialgija ☐Migrena
☐Periferinė neuropatija
☐Psichiatrinė patologija (patikslinti): _____
☐Uveitas : _____
☐Kita akių patologija (prašome patikslinti): _____
☐Kitos ligos (prašome patikslinti): _____
☐Nei viena iš paminėtų

CA12. HLA-B27 statusas :-
☐Teigiamas ☐Neigiamas ☐Nežinomas

CB. GINEKOLOGIJA (vyrams – praleisti)

CB1. Jei jūs moteris, kiek jums buvo metų, kai prasidėjo pirmosios mėnesinės? _____ m.

CB2 *Ar jūsų mėnesinių ciklas reguliarus (kas 28 dienas vidutiniškai)?* ☐Taip ☐Ne

CB3. *Ar jums yra buvę, kad pasireikštų padidėjęs planuotumas ant kūno ir veido)?* ☐Taip ☐Ne

CB4. *Ar jūs sergate policistinių kiaušidžių sindromu?* ☐ Taip ☐Ne

CC. ŠEIMINĖ ANAMNEZĖ

CC1.Ar yra šeimoje sergančių pūlingu hidradenitu?
☐Taip ☐Ne ☐Nežinoma

Jei taip:

☐Mama	☐Dukra	☐Pusbrolis/ pusseserė
☐Tėvas	☐Sūnus	☐Seneliai iš motinos pusės
☐Sesuo	☐Teta	☐Seneliai iš tėvo pusės
☐Brolis	☐Dėdė	☐Kita_____

Jei taip: ar žinote šeimos nario Hurley klasifikacijos laipsnį?
☐Hurley I ☐Hurley II ar III ☒Nežinoma

CC2. Ar yra būdingas šeiminis polinkis žemiau paminėtoms būklėms?
☐Aknė ☐Psoriazė
☐Skalpo disekuojantis celiulitas
☐Pilonidinis sinusas (cista)
☐Krono liga ☐Opinis kolitas
☐Reumatoidinis artritas ☐Ankilozinis spondilitas
☐Adamantiades-Behçet liga ☐Sarkoidozė
☐Limfoma ☐Gangrenuojanti piodermija
☐Plokščių ląstelių karcinoma PH pažeidimo vietoje
☐Kita (prašome patikslinti): _____
☐Nei vienas iš paminėtų

CD. GYDYMAS

CD1. Ar vartojate kitus vaistus (įskaitant kontraceptikus) kartu su pūlingo hidradenito gydymu?
_____
_____

CE. RŪKYMAS CE1. Ar rūkote?
☐Nerūkau ☐Mečiau rūkyti ☐Šiuo metu rūkau

Jei šiuo metu rūkote ar metėte rūkyti :

CE2. ***Ar jūs jau tuo metu rūkėte****, kai prasidėjo pirmieji ligos simptomai?* ☐Taip ☐Ne

CE3. ***Kokią įtaką*** *rūkymas turi/turėjo jūsų ligai?*
☐Neigiamą ☐Teigiamą ☐Neturi/ neturėjo įtakos

CE4. *Kiek jums buvo metų, kai* ***pradėjote****?* _____ m.

CE5. ***Kiek cigarečių*** *surūkote/surūkėte per dieną?*
_____cigarečių per dieną.

CE6. *Kiek jums buvo metų, kai metėte?* _____ m.

CE7. *Kokią įtaką turėjo rūkymo atsisakymas jūsų ligos sunkumui?*
☐Ligos simptomai pasunkėjo
☐Nepastebėjau jokio skirtumo
☐ Ligos simptomai palengvėjo ☐ Liga pasitraukė

CF ALKOHOLIS. CF1. Kaip dažnai vartojate alkoholį?

☐Niekada	☐Kiekvieną savaitę
☐Retai	☐Kiekvieną dieną

Jie vartojate alkoholį kiekvieną dieną, kokios rūšies gėrimą(-us) renkatės? Kokio stiprumo? (galimi keli atsakymai)
☐Vynas (stiprumas: _____)
☐Alus (stiprumas: _____)
☐Stiprus likeris (stiprumas: _____)
☐Kita (patikslinti): _____ (stiprumas: _____)

CG NARKOTIKAI. CG1. Ar rūkote/vartojate marihuaną?
☐Taip ☐Ne
Jei taip, kiek ilgai? _____ m.
Jei taip, kiek suktinių? _____ suktinių(-es)
Nurodykite, per kokį laiko tarpą:

☐per dieną	☐per mėnesį
☐per savaitę	☐per metus

Jei taip, ar vartojate kaip skausmą mažinančią priemonę ar rekreaciniu tikslu?

☐skausmui mažinti	☐dėl abiejų priežasčių
☐rekreaciniu tikslu	☐Kita :_____

CG2. Ar vartojate kitos rūšies narkotikus (rekreacinius narkotikus)? ☐Taip ☐Ne
Jei taip, prašome patikslinti: _____
Jei taip, kiek ilgai? ____ *m.*
*Jei taip kokį kiekį vartojate?*___
Nurodykite, per kokį laiko tarpą :

☐per dieną	☐per mėnesį
☐per savaitę	☐per metus

CH ALERGIJOS CH1. Ar turite alergiją ir/ar atopiją?
☐Taip ☐Ne **Jei taip, kurią?**
☐Atopinė egzema
☐Kita egzema, nurodykite alergeną: _____
☐Astma ☐Alerginis rinitas
☐Lėtinis ar pasikartojantis sinusitas
☐Dilgėlinė, nurodykite alergeną: _____
☐Angioedema, nurodykite alergeną: _____
☐Alergija plėviasparnių nuodams

☐Alergija vaistams, prašome nurodyti reakcijos tipą:
☐Bėrimas ☐Angioedema
☐Kita (prašome patikslinti): _____

D PŪLINGO HIDRADENITO ANAMNEZĖ

DA1. Kiek jums buvo metų, kai atsirado pirmasis furunkulas*? _____ m.
(*uždegiminis mazgas ir/ar abcesas, dažnai skausmingas)
DA2. Kiek jums buvo metų, kai pirmą kartą kreipėtės į gydytoją dėl furunkulo? _____ m.
DA3. Kiek jums buvo metų, kai jums buvo diagnozuotas pūlingas hidradenitas? _____ m.
DA4. Kiek praėjo laiko nuo kreipimosi į šeimos gydytoją iki patekimo pas dermatovenerologą konsultacijai dėl furunkulo? _____ mėnesiais
DA5. Kas diagnozavo jums ligą?

☐Šeimos gydytojas	☐Ginekologas
☐Infekcinių ligų gydytojas	☐Jūs pats (pati)
☐Dermatovenerologas	☐Skubios medicinos gydytojas
☐Chirurgas	☐Kita:_____

DA6. Ar prieš pūlingo hidradenito diagnozę jums buvo neteisingai diagnozuota kita liga? ☐Taip ☐Ne
Jei taip, kuri kita liga buvo įtariama? _____
DA7. Kiek kartų susidūrėte su ligos atkryčiu (atsirado nauji arba paūmėjo seni furunkulai) per pastaruosius metus? _____
DA8. Vidutinė jūsų ligos atkryčio trukmė? _____d.
DA9. Kur dažniausiai atsiranda furunkulai atkryčio metu?

☐Dažniausiai įprastose vietose	☐Tiek įprastose, tiek
☐Dažniausiai naujose vietose	naujose vietose

DA10. Kiek turėjote furunkulų (naujų ar paūmėjusių senų) per paskutines 4 savaites? _____
DA11. Kokio skausmingumo buvo pats skausmingiausias furunkulas (prašome įvertinti skausmą nuo 0 iki 10) _____
DA11.5 30 dienų VAS skalės suma (0-300) _____
DA12. Kiek dėl ligos skausmingų dienų turėjote per paskutines 4 savaites? _____d.
DA13. Kokio tipo skausmas yra susijęs su jūsų liga?
☐Nutrūkstantis skausmas
☐Besitęsiantis skausmas :
☐_____ savaitę(-es)☐_____ mėnesį(-ius)☐_____ metus
DA14. Kaip stipriai buvo supūliavęs jus labiausiai neraminantis furunkulas per paskutines 4 savaites (prašome įvertinti supūliavimą nuo 0 iki 10)? _____
DA15. Kaip stipriai jautėte odos niežėjimą per paskutines 4 savaites? (vertinant nuo 0 iki 10) : _____
DA16. Kaip jūsų liga progresavo nuo jos atsiradimo pradžios? ☐Liko stabili ☐Būklė pablogėjo
☐Būklė pagerėjo (sumažėjo ligos stiprumas)
DA17. Ar kada nors jautėte karščiavimo epizodus per uždegiminius PH protrūkius? ☐Taip ☐Ne
DA18. Ar kada nors nenuėjote į darbą dėl PH?
☐Taip ☐Ne ☐Netaikoma
Jei taip, kiek darbo dienų turėjote praleisti dėl PH per visą savo darbingo gyvenimo laikotarpį (grubiai)? _____ d.
Jei taip, kiek darbo dienų turėjote praleisti dėl PH per paskutinius 6 mėnesius? _____d.
Jei taip, kiek darbo dienų turėjote praleisti dėl PH per paskutines 4 savaites? _____ d.
DA19. Ar liga paveikė jūsų profesinę karjerą?
☐Visiškai nepaveikė ☐Truputį ☐Labai ☐Netaikoma
DA20. Paūmėjimą keliantys ligą sunkinantys veiksniai:
☐Stresas ☐Alkoholis ☐Maistas ir gėrimai
☐Nesteroidiniai vaistai nuo uždegimo
☐Spaudimas ar mechaninė trintis (pavyzdžiui, trintis ar aptempti rūbai/apatiniai)
☐Kita (prašome patikslinti): _____
☐Nėra jokių ligą sunkinančių veiksnių
DA21. Ar turite tatuiruočių? ☐Taip ☐Ne
Jei taip, kiek ilgai? _____ m.
Jei taip, kokį plotą užima? ☐<1% ☐1-3% ☐> 3%
Jei taip, kaip manote, kokią įtaką turi tatuiruotė(-ės) jūsų pūlingam hidradenitui?
☐Neigiamą ☐Teigiamą ☐Neturi įtakos
Jei pastebėjote įtaką, kokio ji tipo?
☐Lokali (ligos išraiškos pakitimai tik ištatuiruotoje zonoje)
☐Generalizuota (ligos išraiškos pakitimai nepriklausomai nuo ištatuiruotos zonos)

MOTERIMS

DA22. Ar menstruacijos turi įtakos jūsų ligai?
☐Taip, ligos simptomai pasunkėja
☐Taip, ligos simptomai palengvėja
☐Nėra jokios įtakos
DA23. Ar kada nors buvote pastojusi? ☐Taip ☐Ne
Jei taip, kiek kartų? _____
Ar nėštumas turėjo įtakos pūlingo hidradenito sunkumui?

☐Taip, simptomai pasunkėjo	☐Nebuvo jokios įtakos
☐Taip, simptomai palengvėjo	☐Liga dar nebuvo prasidėjusi

DA24. Ar buvo sunkumų siekiant pastoti? ☐Taip ☐Ne

DB KOMPLIKACIJOS

DB1. Ar turėjote kokių nors kitų sveikatos problemų, kurias sukėlė pūlingas hidradenitas? ☐Taip ☐Ne
Jei taip, kokią(-as)? _____
DB2. Ar kada nors buvote hospitalizuotas(-a) dėl savo ligos? ☐Taip ☐Ne
Jei taip kiek kartų? _____ kartų(-us)
Jei taip, kiek dienų? _____dienų(-as)

DC. Gyvenimo kokybė

DC GYVENIMO KOKYBĖ

DC1. DLQI įvertis : _____
DC2. Kaip stipriai jūs nerimaujate dėl savo ligos? (prašome įvertinti nuo 0 iki 10, kur 0 reiškia, kad jūs nejaučiate nerimo dėl savo ligos, 10 – labai nerimaujate)
_____ **DC3. HidraDisk įvertis :** _____
DC4. Kurie žodžiai geriausiai apibūdina jūsų ligą?

☐Skausmas	☐Chroniškumas	☐Gėda
☐Drovėjimasis	☐Negalia	☐Kita :_____

DC5. Ar jaučiate ką nors susijusio su odos pažeidimais remisijos laikotarpiu? ☐Taip ☐Ne

Jei taip: ☐Niežėjimas ☐Kita, apibūdinkite: _____

DC6. Prašome įvertinti PH įtaką toliau paminėtiems aspektams (įvertinkite įtaką skalėje nuo 0 iki 10, kur 0 reiškia, kad nėra jokios įtakos, o 10 – blogiausia galima įtaka): miego rėžimas: _____ elgesys: _____

DC7. Kokie žodžiai geriausiai apibūdina idealų pūlingo hidradenito gydymą?

☐Greitas ☐Finansuojamas valstybės ☐Efektyvus

☐Retai pasireiškia nepageidaujamas poveikis

☐Kita (prašome patikslinti): _____

DC8. Ar kada nors esate sutikęs(-usi) kitą žmogų, sergantį pūlingu hidradenitu? ☐Taip ☐ Ne

Jei taip, apibūdinkite, kokiame kontekste susitikote:

_____

DD GYDYMAS

DD1. Kokius anksčiau gydymo būdus (medikamentus) naudojote PH gydyti? Patikslinkite, ar vis dar naudojate gydymą ar nutraukėte jo naudojimą.

VIETINIAI ANTISEPTIKAI, IŠ KURIŲ:

Chloramfenikolis : ☐Vis dar naudoju ☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Povidono-jodo muilas ☐ Vis dar naudoju ☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Povidono-jodo kremas☐Vis dar naudoju☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Kita (patikslinti): _____

☐Vis dar naudoju☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

VIETINIAI ANTIBIOTIKAI, IŠ KURIŲ:

Klindamicinas : ☐Vis dar naudoju ☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Kita (prašome patikslinti): _____

☐Vis dar naudoju ☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Kortikosteroidų injekcija į pažeidimo vietas

☐Vis dar naudoju ☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

SISTEMINIAI ANTIBIOTIKAI, IŠ KURIŲ:

Tetraciklinas -Preparatai su tetraciklino chlorchidratU

☐Vis dar naudoju☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Limeciklinas : ☐Vis dar naudoju ☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Doksiciklinas ☐Vis dar naudoju ☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Minociklinas ☐Vis dar naudoju ☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Klindamicinas☐Vis dar naudoju☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiška

Rifampicinas ☐Vis dar naudoju ☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Penicilinas + klavulano rūgštis

☐Vis dar naudoju☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Kita (prašome nurodyti): _____

☐Vis dar naudoju☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

KITAS SISTEMINIS GYDYMAS, IŠ KURIŲ:

Cinkas ☐Vis dar naudoju ☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Retinoidai ☐Vis dar naudoju ☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

NVNU ☐Vis dar naudoju ☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Kortikosteroidai ☐Vis dar naudoju ☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

BIOLOGINĖ TERAPIJA

Adalimumabas Efektas: ☐Nėra ☐Dalinis ☐Visiškas

☐Vis dar naudoju (pradėtas _____)

☐Nutraukiau (nutrauktas _____)

Infliksimabas Efektas: ☐Nėra ☐Dalinis ☐Visiškas

☐Vis dar naudoju (pradėtas _____)

☐Nutraukiau (nutrauktas _____)

Ustekinumabas Efektas: ☐Nėra ☐Dalinis ☐Visiškas

☐Vis dar naudoju (pradėtas _____)

☐Nutraukiau (nutrauktas _____)

Guselkumabas Efektas: ☐Nėra ☐Dalinis ☐Visiškas

☐Vis dar naudoju (pradėtas _____)

☐Nutraukiau (nutrauktas _____)

anti-IL17 Efektas: ☐Nėra ☐Dalinis ☐Visiškas

☐Vis dar naudoju (pradėtas _____)

☐Nutraukiau (nutrauktas _____)

Kita (prašome patikslinti): _____

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

☐Vis dar naudoju (pradėtas _____)

☐Nutraukiau (nutrauktas _____)

Kita (prašome patikslinti): _____

☐Vis dar naudoju ☐Nutraukiau

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

TERAPIJA LAZERIU, IŠ KURIŲ:

CO2 lazeris. Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Plaukų šalinimas aleksandrito lazeriu

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Plaukų šalinimas diodiniu lazeriu

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Plaukų šalinimas intensyvia pulsine šviesa

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Plaukų šalinimas neodynium YAG lazeriu

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

OPERACIJOS, IŠ KURIŲ:

Incizija ir drenažas: ☐Taip ☐Ne

Jei taip, operacijų skaičius: _____

Jei taip, efektas: ☐ Nėra ☐ Dalinis ☐ Visiškas

Ribotos ekscizijos: ☐Taip ☐Ne
Jei taip, operacijų skaičius: _____
Jei taip, efektas: ☐ Nėra ☐ Dalinis ☐ Visiškas

Plačios ekscizijos: ☐Taip ☐Ne
Jei taip, operacijų skaičius: _____
Jei taip, efektas: ☐ Nėra ☐ Dalinis ☐ Visiškas
Kita (prašome patikslinti): _____
Efektas: ☐Nėra ☐Dalinis ☐Visiškas

DE. FIZINIS IŠTYRIMAS

	DPažastis	KPažastis	Dkirkšnis	KKirkšnis	DLyt. orga	KLyt.organ	Analinė	Perianalin	DSėdmuo	KSėdmuo	DKrūtis	KKrūtis	Kaklas	Skalpas	Nugara	Gakta	Kita	Iš viso
Uždeg mazgeliai																		
Neuždeg mazgeliai																		
Abscesai																		
Drenuojančios fistulės																		
Nedrenuojančios fist.																		
Cords/HyperScars																		
Kribriforminiai randai																		
Papulės ir folikulitas																		
Komedonai																		
Cistos																		
Piogeninė granuloma																		
Limfedema																		
Gangreninė piodermija																		
Pilonidinė cista																		
Kita :																		
Kita :																		
Kita :																		
Didžiausias atstumas (per regioną)																		
BSA (per regioną)																		
"Uždegiminė spalva " (0-3)																		
"Induracija"(0-3)																		
"Opėjimas" (0-3)																		
"Drenažas" (0-3)																		
"Jautrumas" (0-3)																		
"Storumas" (0-3)																		

Asocijuoti pažeidimai
Sunki aknė (>10 uždeginių pažeidimų): ☐Taip ☐Ne
Jei taip: ☐acne vulgaris ☐acne conglobata ☐acne/PH
Skalpo folikulitas: ☐Taip, apibūdinkite: ☐Disekuojantis
☐Kita(patikslinti):_____☐Ne
DE2. Ar visi pažeidimai yra atskirti nuo normalios odos? ☐Taip ☐Ne
DE3. Body Surface Area (BSA) paveikta pagal Hurley 3
☐ < 1% ☐≥ 1%
DE4. Maksimalus atstumas tarp 2 pažeidimų toje pačioje vietoje: o neaktyvūs pažeidimai
☐< 5 cm ☐< 10 cm ☐> 10 cm
DE5. Sistolinis kraujo spaudimas _____ mmHg
Diastolinis kraujo spaudimas _____ mmHg
DE6. Liemens apimtis : _____ cm

DF. PAPILDOMA APŽIŪRA DG. GYDYMO PLANAS
Gydymas neskiriamas /priežiūra: ☐ Taip ☐ Ne

Vietinis: ☐Taip ☐Ne
☐Povidono-jodo muilas
☐Povidono-jodo kremas
☐Klindamicinas
Jei taip:
☐Kortikosteroidai
☐Rezorcinolis
☐Kita :_____

Nutrūkstantis sisteminis gydymas: ☐Taip ☐Ne
Jei taip, antibiotikų terapija 8-15 dienų paūmėjimui nutraukti: ☐Taip ☐Ne
☐Klindamicinas
☐Tetraciklinai :
☐Preparatai su tetraciklino chlorhidratu
☐Limeciklinas
☐Doksiciklinas
☐Minociklinas
☐Rifampicinas
☐Metronidazolis
☐Moksifloksacinas
☐Penicilinas + klavulano r.
☐Azitromicinas
☐Klaritromicinas
☐Kita: _____
Jei ne : kita (patikslinti) : _____

Ilgalaikis sisteminis gydymas: ☐Taip ☐Ne

*Antibiotika*i

☐Tetraciklinai:	☐Klindamicinas
☐Preparatai su tetraciklino chlorhidratu	☐Metronidazolis
	☐Penicilinas + klavulano r.
☐Limeciklinas	☐Moksifloksacinas
☐Doksiciklinas	☐Azitromicinas
☐Minociklinas	☐Klaritromicinas
☐Rifampicinas	☐Kita: _____

Ne antibiotikai – Biologinė terapija:

☐Adalimumabas	☐Guselkumabas
☐Infliksimabas	☐anti-IL17
☐Ustekinumabas	☐Kita : _____

☐Acitretinas ☐Cinkas ☐Sulfonai ☐Dapsonas

☐Kita: _____

Chirurginis gydymas: ☐Taip (Suplanuota _____) ☐Ne

Jei taip: ☐ Ribota ekscizija (1 pažeidimas) ☐Plati ekscizija

Gydymas lazeriu: ☐Taip ☐Ne Jei taip :

☐CO2 lazeris

Plaukų šalinimas :

☐ aleksandrito lazeriu ☐ diodiniu lazeriu

☐ intensyvia pulsine šviesa ☐neodynium YAG lazeriu

Kita (prašome patikslinti):_____

Kita (prašome patikslinti):_____

Kita (prašome patikslinti):_____

DH. Automatiškai suskaičiuojami įverčiai

Hurley laipsnis? (Nustatydami laipsnį, atsižvelkite į labiausiai pažeistą vietą) ☐I ☐II ☐III

Sartorius įvertis = _____

HS-PGA įvertis:

☐Švari oda	☐Vidutiniškas
☐Minimalus	☐Sunkus
☐Nestiprus	☐Labai sunkus

IHS4: Balai_____ ☐Lengvas ☐Vidutinis ☐Sunkus

SAHS:_____ **SASH :** _____**HASI :** _____

Bendras skaičius ILOF (uždegiminiai ar skausmingi pažeidimai, bet ne fistulė):_____

Subklinikinis variantas pagal Canoui-Poitrine and al.?

☐Ašinis – pieno liaukų	☐Sėdmenų
☐Folikulinis	☐Kita :_____

Subklinikinis variantas pagal van der Zee ir Jemec?

☐Įprastas tipas	☐Sindrominis tipas
☐Frikcinis furunkulo tipas	☐Ektopinis tipas
☐Randinis furunkulo tipas	☐Kita (prašome
☐Konglobatinis tipas	patikslinti): _____

KMI: _____

E. UŽRAŠAI :

Paciento LIN:_____
Data:_____

CA MEDICININĖ ANAMNEZĖ

CA1. Ar pasireiškė kitų sveikatos sutrikimų nuo paskutinio vizito? ☐Ne ☐Taip (patikslinti):
_____

CD GYDYMAS

CD1. Ar pradėjote naujus vaistus nuo praėjusio vizito?
_____
_____

CE RŪKYMAS

CE1. Ar jūsų rūkymo statusas pasikeitė? ☐Taip ☐Ne
Jei taip, apibūdinkite:
☐Anksčiau rūkiau, bet šiuo metu mečiau (patikslinti kada):
_____
☐Sumažinau surūkomų cigarečių skaičių
(nurodykite, kiek cigarečių surūkote per dieną:___)
☐Rūkau daugiau nei anksčiau
(nurodykite kiek cigarečių surūkote per dieną:___)
Jei metėte rūkyti, kaip neberūkymas paveikė jūsų ligos sunkumą?

☐ Būklė pasunkėjo	☐ Būklė pagerėjo
☐ Būklė nepasikeitė	☐ Liga pasitraukė

CF ALKOHOLIS

CF1. Ar jūsų alkoholio suvartojimas pasikeitė?
☐Taip ☐Ne
Jei taip, nurodykite, kaip dažnai vartojate alkoholį:
☐Niekada ☐ Retai ☐Kiekvieną savaitę ☐Kiekvieną dieną.
Jei vartojate alkoholį kiekvieną dieną, kokios rūšies gėrimą(-us) renkatės? Kokio stiprumo? (galimi keli atsakymai)
☐Vynas (stiprumas: _____)
☐Alus (stiprumas: _____)
☐Stiprus likeris (stiprumas: _____)
☐Kita (patikslinti): _____ (stiprumas: _____)

CG NARKOTIKAI

CG1. Ar pasikeitė jūsų marihuanos rūkymo ar jos vartojimo kitais būdais statusas? ☐Taip ☐Ne
Jei taip, apibūdinkite:
☐Anksčiau rūkiau marihuaną, bet šiuo metu mečiau (patikslinti kada): _____
☐Anksčiau rūkiau marihuaną, šiuo metu vis dar rūkau, bet sumažinau vartojimą:
(nurodykite, kiek suktinių surūkote per dieną:___)
☐Kita: _____
CG2. Ar marihuaną vartojate kaip skausmą mažinančią priemonę ar rekreaciniu tikslu?

☐skausmui mažinti	☐dėl abiejų priežasčių
☐rekreaciniu tikslu	☐Kita: _____

CG3. Ar jūsų kitų rekreacinių narkotikų vartojimas pasikeitė? ☐Ne ☐Taip, apibūdinkite:_____

D PŪLINGO HIDRADENITO ANAMNEZĖ

DA1. Kiek furunkulų* (naujų ar paūmėjusių senų furunkulų) turėjote per paskutines 4 savaites? _____
(*uždegiminis mazgas ir/ar abcesas, dažnai skausmingas)
DA2. Kur dažniausiai atsiranda furunkulai ligos atkryčio metu?
☐Dažniausiai įprastose vietose ☐Dažniausiai naujose vietose ☐Tiek įprastose, tiek naujose vietose
DA3. Kokio skausmingumo buvo pats skausmingiausias furunkulas per paskutines 4 savaites (įvertinti skausmą nuo 0 iki 10) _____
DA3.5 30 dienų VAS skalės suma (0-300) _____
DA4. Kiek dėl ligos skausmingų dėl ligos dienų turėjote per paskutines 4 savaites? _____d.
DA5. Kaip stipriai buvo supūliavęs jus labiausiai neraminantis furunkulas per paskutines 4 savaites (įvertinti supūliavimą nuo 0 iki 10)? _____
DA6. Kaip stipriai oda niežėjo per paskutines 4 savaites? (vertinant nuo 0 iki 10) : _____
DA7. Koks gydymas numalšino jūsų ligos paūmėjimą?
_____
DA8. Kokia dabartinė jūsų ligos gydymo taktika?
Vietiniai antiseptikai, iš kurių:
Chloramfenikolis : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Povidono-jodo muilas :
Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Povidono-jodo kremas :
Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Kita (patikslinti): _____
Efektas: ☐Nėra ☐Dalinis ☐Visiškas
VIETINIAI ANTIBIOTIKAI, IŠ KURIŲ:
Klindamicinas : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Kita (prašome patikslinti): _____
Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Kortikosteroidų injekcija į pažeidimo vietas :
Efektas: ☐Nėra ☐Dalinis ☐Visiškas
SISTEMINIAI ANTIBIOTIKAI, IŠ KURIŲ:
Preparatai su tetraciklino chlorchidratu
Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Limeciklinas : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Doksiciklinas : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Minociklinas : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Klindamicinas : Efektas: ☐Nėra ☐Dalinis ☐Visiška
Rifampicinas : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Penicilinas + klavulano rūgštis
Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Kita (prašome nurodyti): _____
Efektas: ☐Nėra ☐Dalinis ☐Visiškas
KITAS SISTEMINIS GYDYMAS, IŠ KURIŲ:
Cinkas : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Retinoidai : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
NVNU : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Kortikosteroidai : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
BIOLOGINĖ TERAPIJA **(pradėta _____)**
Adalimumabas : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Infliksimabas : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Ustekinumabas : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Guselkumabas : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
anti-IL17 : Efektas: ☐Nėra ☐Dalinis ☐Visiškas
Kita (prašome patikslinti): _____
Efektas: ☐Nėra ☐Dalinis ☐Visiškas

TERAPIJA LAZERIU, IŠ KURIŲ:

CO2 lazeris. Efektas: ☐Nėra ☐Dalinis ☐Visiškas

Plaukų šalinimas, efektas:

Aleksandrito lazeriu: ☐Nėra ☐Dalinis ☐Visiškas

Diodiniu lazeriu: ☐Nėra ☐Dalinis ☐Visiškas

Intensyvia pulsine šviesa: ☐Nėra ☐Dalinis ☐Visiškas

Neodynium YAG lazeriu: ☐Nėra ☐Dalinis ☐Visiškas

OPERACIJA, IŠ KURIŲ:

Incizija ir drenažas: ☐Ne

☐ Taip (data_____, lokalizacija_____)

Jei taip, operacijų skaičius: _____

Jei taip, efektas: ☐ Nėra ☐ Dalinis ☐ Visiškas

Ribotos ekscizijos: ☐Ne

☐ Taip (data_____, lokalizacija_____)

Jei taip, operacijų skaičius: _____

Jei taip, efektas: ☐ Nėra ☐ Dalinis ☐ Visiškas

Skausmo įvetinimas šiandien pagal VAS skausmo skalę operuotame regione (nuo 0 iki 10) : _____

Pasitenkinimas (nuo 0 iki 10) : _____

Ar rekomenduotumėte šią operaciją kitiems PH sergantiems pacientams? (nuo 0 iki 10) : _____

Plačios ekscizijos: ☐Ne

☐ Taip (data_____, lokalizacija_____)

Jei taip, operacijų skaičius: _____

Jei taip, efektas: ☐ Nėra ☐ Dalinis ☐ Visiškas

Skausmo įvertinimas šiandien pagal VAS skausmo skalę operuotame regione (nuo 0 iki 10) : _____

Pasitenkinimas (nuo 0 iki 10) : _____

Ar rekomenduotumėte šią operaciją kitiems PH sergantiems pacientams? (nuo 0 iki 10) : _____

Kita (prašome patikslinti): _____

Efektas: ☐Nėra ☐Dalinis ☐Visiškas

DA9. Nuo jūsų ligos pradžios, koks gydymas, jūsų nuomone, buvo pats efektyviausias? _____

DA10. Ar nuo paskutinio vizito turėjote praleisti darbo dienų dėl PH? ☐Taip ☐Ne ☐Nedirbu (netaikoma)

Jei taip, kiek darbo dienų turėjote praleisti dėl PH nuo paskutinio vizito? _____ d.

DB KOMPLIKACIJOS

DB1. Ar nuo paskutinio vizito pajautėte kokį nors kitą sveikatos sutrikimą, sukeltą pūlingo hidradenito?

☐Ne ☐Taip, _____

DB2. Ar nuo paskutinio vizito buvote hospitalizuotas dėl savo ligos? ☐Ne ☐Taip:

_____kartų(-us), _____dienų(-as)

DC GYVENIMO KOKYBĖ

DC1. DLQI įvertis _____

DC2. Kaip stipriai jūs nerimaujate dėl savo ligos? (prašome įvertinti nuo 0 iki 10) _____

DC3. HidraDisk įvertis : _____

DE. FIZINIS IŠTYRIMAS

	DPažastis	KPažastis	Dkirkšnis	KKirkšnis	DLyt.	KLyt.orga	Analinė	Perianali	DSėdmuo	KSėdmuo	DKrūtis	KKrūtis	Kaklas	Skalpas	Nugara	Gakta	Kita	Iš viso
Uždeg mazgeliai																		
Neuždeg mazgeliai																		
Abscesai																		
Drenuojančios fistulės																		
Nedrenuojančios fist.																		
Cords/HyperScars																		
Kribriforminiai randai																		
Papulės ir folikulitas																		
Komedonai																		
Cistos																		
Piogeninė granuloma																		
Limfedema																		
Gangreninė piodermija																		
Pilonidinė cista																		
Kita :																		
Kita :																		
Kita :																		
Did atstumas (per regioną)																		
BSA (per regioną)																		
"Uždegiminė spalva " (0-3)																		
"Induracija"(0-3)																		
"Opėjimas" (0-3)																		
"Drenažas" (0-3)																		
"Jautrumas" (0-3)																		
"Storumas" (0-3)																		

Asocijuoti pažeidimai

Sunki aknė (>10 uždeginių pažeidimų): ☐Taip ☐Ne

Jei taip: ☐acne vulgaris ☐acne conglobata ☐acne/PH

Skalpo folikulitas: ☐Taip, apibūdinkite: ☐Disekuojantis ☐Kita(patikslinti):_____ ☐Ne

DE2. Ar visi pažeidimai yra atskirti nuo normalios odos? ☐Taip ☐Ne

DE3. Body Surface Area (BSA) paveikta pagal Hurley 3

☐ < 1% ☐≥ 1%

DE4. Maksimalus atstumas tarp 2 pažeidimų toje pačioje vietoje: o neaktyvūs pažeidimai

☐< 5 cm ☐< 10 cm ☐> 10 cm

DE5. Sistolinis kraujo spaudimas _____ mmHg

Diastolinis kraujo spaudimas _____ mmHg

DE6. Liemens apimtis : _____ cm

DF PAPILDOMI TYRIMAI

Ar nuo paskutinio vizito pridavėte kraują ištyrimui?

☐Taip ☐Ne

Nurodykite datą: _____

Nurodykite CRB koncentraciją: _____ (mg/dl)

Nurodykite leukocitų skaičių: _____ (/mm^3)

Nurodyite gliukozės konc. nevalgius: _____ (mg/dl)

Nurodykite DTL koncentraciją: _____ (mg/dl)

Nurodykite trigliceridų koncentraciją: _____ (mg/dl)

DG GYDYMO PLANAS

Gydymas neskiriamas /priežiūra: ☐ Taip ☐ Ne

Vietinis: ☐Taip ☐Ne Jei taip:

☐Povidono-jodo muilas	☐Kortikosteroidai
☐Povidono-jodo kremas	☐Rezorcinolis
☐Klindamicinas	☐Kita :_____

Nutrūkstantis sisteminis gydymas: ☐Ne

☐Taip, nuo šiandien ☐Taip, tęsiamas

Jei taip, antibiotikų terapija 8-15 dienų paūmėjimui nutraukti: ☐Taip ☐Ne

☐Klindamicinas	☐Rifampicinas
☐Tetraciklinai :	☐Metronidazolis
☐Preparatai su tetraciklino chlorhidratu	☐Moksifloksacinas
	☐Penicilinas + klavulano r.
☐Limeciklinas	☐Azitromicinas
☐Doksiciklinas	☐Klaritromicinas
☐Minociklinas	☐Kita: _____

Jei ne : kita (patikslinti) : _____

Ilgalaikis sisteminis gydymas ☐Ne

☐Taip, nuo šiandien ☐Taip, tęsiamas

Antibiotikai

☐Tetraciklinai:	☐Klindamicinas
☐Preparatai su tetraciklino chlorhidratu	☐Metronidazolis
	☐Penicilinas + klavulano r.
☐Limeciklinas	☐Moksifloksacinas
☐Doksiciklinas	☐Azitromicinas
☐Minociklinas	☐Klaritromicinas
☐Rifampicinas	☐Kita: _____

Ne antibiotikai – Biologinė terapija:

☐Adalimumabas	☐Guselkumabas
☐Infliksimabas	☐anti-IL17
☐Ustekinumabas	☐Kita : _____

☐Acitretinas ☐Cinkas ☐Sulfonai ☐Dapsonas

☐Kita: _____

Chirurginis gydymas: ☐Taip (Suplanuota _____) ☐Ne

Jei taip: ☐ Ribota ekscizija (1 pažeidimas) ☐Plati ekscizija

Gydymas lazeriu, iš kurių: ☐Taip ☐Ne

Jei taip

☐CO2 lazeris

Plaukų šalinimas :

☐ aleksandrito lazeriu ☐ diodiniu lazeriu

☐ intensyvia pulsine šviesa ☐neodynium YAG lazeriu

Kita (prašome patikslinti):_____

Kita (prašome patikslinti):_____

Kita (prašome patikslinti):_____

DH. Automatiškai suskaičiuojami įverčiai

Hurley laipsnis? (Nustatydami laipsnį, atsižvelkite į labiausiai pažeistą vietą) ☐I ☐II ☐III

Sartorius įvertis = _____

HS-PGA įvertis:

☐Švari oda	☐Vidutiniškas
☐Minimalus	☐Sunkus
☐Nestiprus	☐Labai sunkus

IHS4: Balai_____ ☐Lengvas ☐Vidutinis ☐Sunkus

SAHS:_____ **SASH :** _____**HASI :** _____

Bendras skaičius ILOF (uždegiminiai ar skausmingi pažeidimai, bet ne fistulė):_____

Subklinikinis variantas pagal Canoui-Poitrine and al.?

☐Ašinis – pieno liaukų	☐Sėdmenų
☐Folikulinis	☐Kita :_____

Subklinikinis variantas pagal van der Zee ir Jemec?

☐Įprastas tipas	☐Sindrominis tipas
☐Frikcinis furunkulo tipas	☐Ektopinis tipas
☐Randinis furunkulo tipas	☐Kita (prašome patikslinti): _____
☐Konglobatinis tipas	

KMI: _____

E.UŽRAŠAI :

APPENDIX II Dermatology Life Quality Index (DLQI)

VšĮ Vilniaus universiteto ligoninė
Santaros klinikos
Dermatovenerologijos centras

PATVIRTINTA
VšĮ VUL Santaros klinikų generalinio
direktoriaus 2018-10-23 įsakymu Nr. V-892
GP 134 „Žvynelinės diagnostika ir gydymas“
3 priedas

DLQI (*Dermatology Life Quality Index*)
Dermatologinis gyvenimo kokybės indeksas **Data:** .....

Šio klausimyno tikslas – nustatyti, kaip odos problema paveikė jūsų gyvenimą PASTARĄJĄ SAVAITĘ.
Prašome prie kiekvieno klausimo kryželiu (×) pažymėti vieną atsakymą

1.	Ar pastarąją savaitę Jūsų oda buvo **opi**, **skausminga**, ją **niežėjo** arba **dilgėjo**?	Labai stipriai Stipriai Šiek tiek Visai ne	□ □ □ □	
2.	Ar pastarąją savaitę dėl savo odos **varžėtės** ar **drovėjoties**?	Labai stipriai Stipriai Šiek tiek Visai ne	□ □ □ □	
3.	Ar pastarąją savaitę dėl Jūsų odos būklės kilo sunkumų **apsiperkant**, **tvarkantis namuose** ar **dirbant sode**?	Labai daug Daug Nedaug Visai ne	□ □ □ □	Netaikoma □
4.	Ar pastarąją savaitę Jūsų odos būklė turėjo įtakos pasirenkant dėvimus **drabužius**?	Labai daug Daug Nedaug Visai ne	□ □ □ □	Netaikoma □
5.	Ar pastarąją savaitę Jūsų odos būklė turėjo įtakos **visuomeninei** veiklai ar **laisvalaikio** užsiėmimams?	Labai daug Daug Nedaug Visai ne	□ □ □ □	Netaikoma □
6.	Ar pastarąją savaitę patyrėte sunkumų dėl savo odos būklės **sportuodami**?	Labai daug Daug Nedaug Visai ne	□ □ □ □	Netaikoma □
7.	Ar pastarąją savaitę dėl savo odos būklės negalėjote **dirbti** ar **mokytis**?	Taip Ne	□ □	Netaikoma □
	Jei atsakėte „Ne“, ar pastarąją savaitę dėl Jūsų odos būklės jums kilo problemų **dirbant** ar **mokantis**?	Daug Nedaug Visai ne	□ □ □	
8.	Ar pastarąją savaitę dėl Jūsų odos būklės kilo problemų bendraujant su **partneriu** ar **artimais draugais** ar **giminaičiais**?	Labai daug Daug Nedaug Visai ne	□ □ □ □	Netaikoma □
9.	Ar pastarąją savaitę dėl Jūsų odos būklės kilo **lytinio gyvenimo** problemų?	Labai daug Daug Nedaug Visai ne	□ □ □ □	Netaikoma □
10.	Ar pastarąją savaitę Jūsų odos **gydymas** kėlė problemų, pvz., dėl jo kilo netvarka namuose arba užėmė daug laiko?	Labai daug Daug Nedaug Visai ne	□ □ □ □	Netaikoma □

Prašome pasitikrinti, ar atsakėte į VISUS klausimus. Dėkojame.

DLQI vertinimas:

Atsakymai	**Atsakymų vertinimas balais**
Labai stipriai, labai daug, taip	3
Stipriai, daug	2
Šiek tiek, nedaug	1
Visai ne, ne, netaikoma	0

LIN: .....

Bendra balų suma: .....

APPENDIX III Ethical approval and bioethics update

VILNIAUS REGIONINIS BIOMEDICININIŲ TYRIMŲ ETIKOS KOMITETAS
sui generis darinys prie VILNIAUS UNIVERSITETO

LEIDIMAS
ATLIKTI BIOMEDICININĮ TYRIMĄ

2021 02 23 Nr.2021/2-1310-793

Tyrimo pavadinimas:

Biologiniai supūliavusio hidradenito žymenys

Protokolo Nr.:	01
Versija:	1.2
Data:	2021 03 05
Informuoto asmens sutikimo forma:	1.2 2021 03 05
Pagrindinis tyrėjas:	**Jūratė Grigaitienė**
Įstaigos pavadinimas: Adresas:	VšĮ Vilniaus universiteto ligoninė Santaros klinikos Santariškių g. 2, Vilnius
Leidimas galioja iki:	**2023 10**

Leidimas išduotas Vilniaus regioninio biomedicininių tyrimų etikos komiteto posėdžio (protokolas Nr. 2021/2), vykusio 2021 m. vasario 23 d. sprendimu.

Pirmininkas doc. dr. Alfredas Laurinavičius

Viešoji įstaiga
Universiteto g. 3
01513 Vilnius

Duomenys kaupiami ir saugomi
Juridinių asmenų registre
Kodas 211950810

Komiteto duomenys:
M. K. Čiurlionio g. 21, LT- 03101 Vilnius
Tel. (8 5) 268 6998, el. p. rbtek@mf.vu.lt

VILNIAUS REGIONINIS BIOMEDICININIŲ TYRIMŲ ETIKOS KOMITETAS
***sui generis* darinys prie VILNIAUS UNIVERSITETO**

Biomedicininio tyrimo „Biologiniai supūliavusio hidradenito žymenys“ 2023-06-28 Nr. 2023-LP-55
pagrindinei tyrėjai Jūratė Grigaitienė

PRITARIMAS
BIOMEDICININIO TYRIMO DOKUMENTŲ PAKEITIMAMS

Leidimo Nr. 2021/2-1310-793 pakeitimas Nr. 1

Vilniaus regioninis biomedicininių tyrimų etikos komitetas išnagrinėjęs prašymą atlikti su vykdomu biomedicininiu tyrimu „*Biologiniai supūliavusio hidradenito žymenys*“ (leidimas Nr. 2021/2-1310-793, išduotas 2021 02 23 d.) susijusių dokumentų pakeitimus nusprendė, kad pakeitimai **atitinka** Lietuvos Respublikos biomedicininių tyrimų etikos įstatymo II skyriuje nustatytiems biomedicininių tyrimų etikos reikalavimams. Atsižvelgiant į tai **pritariama**, kad būtų:

- tyrimas pratęsiamas iki 2025 rugsėjo 30 d.
- vadovaujamasi protokolu (Nr. 01, versija Nr. 1.3, data 2023 04 23 d.);
- teikiama informuoto asmens sutikimo forma (versija Nr. 1.3, data 2023 04 11 d.).

Pirmininkas doc. dr. Alfredas Laurinavičius

Viešoji įstaiga
Universiteto g. 3
01513 Vilnius

Duomenys kaupiami ir saugomi
Juridinių asmenų registre
Kodas 211950810

Komiteto duomenys:
M. K. Čiurlionio g. 21, LT- 03101 Vilnius
Tel. (8 5) 268 6998, el. p. rbtek@mf.vu.lt

APPENDIX IV Material transfer agreement between Vilnius University and Regeneron Pharmaceuticals, Inc.

Vilnius University
3 Universiteto St.
Vilnius, Lithuania LT-01513
Email: tadas.raudonis@mf.vu.lt
Attention: Tadas Raudonis, M.D.

Regeneron Pharmaceuticals, Inc.
777 Old Saw Mill River Road
Tarrytown, NY 10591-6707
Email: LegalNotices@regeneron.com
Attention: General Counsel

IN WITNESS WHEREOF, the parties have executed this Agreement as of the Effective Date.

Regeneron Pharmaceuticals, Inc.

By: Matthew Sleeman (Sep 13, 2024 08:47 EDT)
Name: Matthew A. Sleeman, Ph.D.
Title: Vice President, Research Therapeutic Focus Areas
Date: Sep 13, 2024

Vilnius University

By: Kristina Babelytė-Labanauskė (Sep 13, 2024 16:31 GMT+3)
Name: Kristina Babelytė-Labanauskė
Title: Head of Innovation Office at Science and Innovatio Department (acting according to authorization dated December 13, 2022, No. RI-232)
Date: Sep 13, 2024

Approved as to legal form, per Regeneron Corporate Policy #950.

Regeneron: Clinical Experimental Sciences and Research-VILNIUS UNIVERSITY-2024_067148

Final Audit Report 2024-09-13

Created:	2024-09-12
By:	Monica Chawla (monica.chawla@regeneron.com)
Status:	Signed
Transaction ID:	CBJCHBCAABAA_oXjlqnXt8QWcF8mUEV0u_XzZQPSHDzc

"Regeneron: Clinical Experimental Sciences and Research-VILNIUS UNIVERSITY-2024_067148" History

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Signer kristina.babelyte-labanauske@cr.vu.lt entered name at signing as Kristina Babelytė-Labanauskė
2024-09-13 - 1:31:54 PM GMT- IP address: 71.162.198.114

Document e-signed by Kristina Babelytė-Labanauskė (kristina.babelyte-labanauske@cr.vu.lt)
Signature Date: 2024-09-13 - 1:31:56 PM GMT - Time Source: server- IP address: 71.162.198.114

Agreement completed.
2024-09-13 - 1:31:56 PM GMT

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SUMMARY (SANTRAUKA)

1. TRUMPAS ĮVADAS

Supūliavęs hidradenitas (SH), taip pat žinomas kaip *acne inversa*, yra lėtinė uždegiminė odos liga, pasireiškianti apokrininių liaukų gausiose kūno vietose, kuriai būdingi uždegiminiai mazgai, abscesai, fistulės ir randai (1). Dėl nevienalyčio SH pobūdžio yra nustatyti įvairūs ligos fenotipai ir sunkumo laipsniai (2). Pacientai, sergantys SH, turi didesnę gretutinių ligų naštą, tokių kaip policistinių kiaušidžių sindromas, cukrinis diabetas, metabolinis sindromas, uždegiminės žarnų ligos, psichikos sutrikimai, limfoma ir kt., palyginus su bendra populiacija (3,4).

Asmenys, sergantys SH, patiria vidutiniškai 7,2 metų laiko tarpą nuo ligos pradžios iki jos diagnozavimo. Vėlyvą diagnostiką įtakoja įvairūs veiksniai, pavyzdžiui, gydytojų ligos neatpažinimas, ligos stigmatizavimas visuomenėje bei socialinės ir ekonominės kliūtys (5–7). Ribotas veiksmingo gydymo prieinamumas ir dažni paūmėjimai neigiamai veikia pacientų gyvenimo kokybę (8, 9). Be to, atsižvelgiant į skausmingą pažeidimų pobūdį, jų atsiradimą jautriose vietose, nemalonų kvapą ir besiformuojančius randus, SH gali reikšmingai paveikti žmogaus psichologinę būklę. Todėl labai svarbu, kad diagnozė būtų nustatyta laiku, o savalaikis gydymas sumažintų ligos naštą pacientams ir sveikatos priežiūros sistemai (1).

1.1. Tikslas ir uždaviniai

Tikslas – išanalizuoti pūlingo hidradenito rizikos veiksnių ryšį, klinikinį vaizdą, biologinius žymenis ir gydymo efektyvumą.

Uždaviniai:

1. Įvertinti SH demografinius duomenis, rizikos veiksnius ir ligos naštą.
2. Įvertinti SH klinikinį vaizdą ir gretutines ligas.
3. Įvertinti SH gydymo efektyvumą.

2. METODAI

2.1. Tyrimo organizavimas

Atliktas vieno centro skerspjūvio prospektyvinis tyrimas, kuris vyko Vilniaus universiteto ligoninės Santaros klinikų (VUL SK) Dermatovenerologijos centre nuo 2021 m. kovo iki 2023 m. birželio mėn.

Įtraukimo kriterijai:

- Tiriamasis gali perskaityti, suprasti ir pasirašyti informuoto sutikimo formą prieš bet kokias tyrimo procedūras
- Suaugę vyrai ir moterys, 18-70 m.
- Supūliavusio hidradenito diagnozė
- Tiriamasis gali dalyvauti suplanuotuose vizituose

Neįtraukimo kriterijai:

- Tiriamojo atsisakymas dalyvauti tyrime
- Nepilnamečiai asmenys
- Subjektas negali suprasti protokolo procedūrų ir su jomis sutikti
- Nėščios arba krūtimi maitinančios moterys
- Asmenys, sergantys aktyvia onkologine liga

Dalyvauti tyrime buvo kviečiami SH sergantys pacientai atvykę į VUL SK Dermatovenerologijos centrą įprastai sveikatos priežiūrai gauti. Pacientai pasirašė informuoto sutikimo dalyvauti biomedicininiame tyrime formą. Pacientai buvo įvertinti pagal Europos pūlingo hidradenito fondo (angl. *European Hidradenitis Suppurativa Foundation*, EHSF) registro klausimyno gaires (I priedas).

Duomenų rinkimas buvo sutelktas į demografinius duomenis, tokius kaip amžius, lytis, kūno masės indeksas (KMI), išsilavinimas, odos fototipas, plaukų struktūra, gretutinės ligos, uždegiminių ligų šeimos anamnezė, pažeidimo vieta ir klinikiniai SH potipiai, pagal *Canoui-Poitrine* kriterijus (195). Buvo įvertinti rūkymo įpročiai, jų ryšys su SH sunkumu (pagal Hurley stadiją), amžius diagnozuojant SH, ligos trukmė, nustatytos klaidingos diagnozės. Skausmo intensyvumas buvo matuojamas naudojant vizualinę analoginę skalę (VAS), o nerimas buvo vertinamas 0–10 balų skalėje. Taip pat dokumentuoti ankstesni ir dabartiniai gydymo būdai. Klinikiniai rodikliai, tokie kaip mazgų, abscesų ir fistulių skaičius, IHS4 (angl. *International Hidradenitis Suppurativa Severity Scoring System*), dermatologinis gyvenimo

kokybės indeksas (DGKI) ir VAS skausmo balai buvo fiksuoti tiek prieš gydymą, tiek jo metu.

2.2. Atitiktis bioetikos reikalavimams

Tyrimui gautas bioetikos leidimas Vilniaus regiono biomedicininių tyrimų etikos komitete (Nr. 2021/2-1310-793). Bioetikos leidimas buvo atnaujintas 2023 m. birželio 28 d. (Nr. 2023-LP-55) (III priedas), kad būtų įtraukti pakeitimai, susiję su duomenų ir odos biopsijos mėginių siuntimu už ES ribų. Tyrimo protokolas atitinka 1975 m. Helsinkio deklaraciją, peržiūrėtą 2013 m. Visi tiriamieji buvo informuoti apie tyrimą ir pasirašė informuoto sutikimo formas.

2.3. Rinkti duomenys

Demografiniai duomenys:

- Amžius, lytis, etninė kilmė, odos fototipas, plaukų struktūra
- Šeiminė, profesinė padėtis, išsilavinimas

Gyvenimo anamnezė:

- Gretutinės ligos: aknė, psoriazė, uždegiminė žarnų liga, pilonidinė cista, depresija, artritas
- Metabolinės ligos: dislipidemija, išeminė širdies liga, cukrinis diabetas, smegenų kraujagyslių ligos, pirminė arterinė hipertenzija, metabolinis sindromas
- Šeiminė anamnezė
- Vartoti ir vartojami vaistai
- Rūkymo istorija
- Alkoholio ir narkotikų vartojimas
- Alergijos

SH anamnezė:

- Amžius, kai atsirado pirmasis mazgas
- Amžius, kai pacientas pirmą kartą kreipėsi į gydytoją
- Amžius diagnozuojant SH
- Laikas, per kurį buvo apsilankyta pas dermatovenerologą po šeimos gydytojo konsultacijos
- Specialistas, diagnozavęs ligą
- Klaidingos diagnozės

- Ligos atkryčių skaičius per pastaruosius metus, vidutinė ligos atkryčio trukmė
- Pūlinių/mazgų (naujų arba paūmėjusių) skaičius per pastarąsias 4 savaites
- Pūlinių/mazgų skausmas nuo 0 iki 10, skausmingų dienų skaičius per pastarąsias 4 savaites, skausmo tipas
- Ligos progresavimas nuo jos atsiradimo pradžios
- Nebuvimas darbe dėl SH, ligos įtaka profesinei karjerai
- Ligą paūminantys veiksniai

Komplikacijos:

- SH sukeltos sveikatos problemos
- Hospitalizacija dėl SH ligos, hospitalizacijų skaičius, dienų skaičius

Gyvenimo kokybė:

- DGKI
- Nerimo balas (nuo 0 iki 10)
- Ligos įtaka miegui ir elgesiui nuo 0 iki 10

Gydymas:

- Vietiniai antiseptikai
- Vietiniai antibiotikai
- Sisteminiai antibiotikai
- Kitas sisteminis gydymas
- Biologinė terapija
- Lazerio terapija
- Chirurgija

Fizinė apžiūra:

- Pažeidimų skaičius
- Bendras uždegiminių pažeidimų skaičius
- Kiti susiję odos pažeidimai
- Kraujospūdis
- Liemens apimtis
- *Hurley* stadija: I, II, III
- IHS4 balas
- Subklinikinis ligos tipas
- Svoris, ūgis, KMI

2.4. Duomenų apibrėžimai ir transformacijos

IHS4 įvertis

SH sunkumą galima įvertinti naudojant IHS4 – patvirtintą įrankį, kurį sukūrė EHSF. IHS4 balas nustatomas uždegiminių mazgų skaičių padauginus iš 1, abscesų skaičių iš 2 ir drenuojančių fistulių skaičių iš 4. Suskaičiavus bendrą balą, SH sunkumas priskiriamas prie lengvo (≤3 balai), vidutinio sunkumo (nuo 4 iki 10 balų) arba sunkus (≥11 balų) (196).

Dermatologinis Gyvenimo Kokybės Indeksas

DGKI yra savarankiškai pildomas, paprastas ir patogus klausimynas, kurio vidutinis užpildymo laikas yra 126 s. Jį sudaro 10 klausimų, susijusių su pacientų suvokimu apie odos ligų įtaką skirtingiems jų gyvenimo aspektams per praėjusią savaitę. Jis patvirtintas 16 metų ir vyresniems pacientams. DGKI klausimai skirstomi į 6 grupes: simptomai ir jausmai (1 ir 2 klausimai), kasdienė veikla (3 ir 4 klausimai), laisvalaikis (5 ir 6 klausimai), asmeniniai santykiai (8 ir 9 klausimai), darbas ir mokykla (7 klausimas) ir gydymas (10 klausimas), kiekvieno klausimo maksimalus įvertis – 3 balai. Anketos lietuvių kalba pavyzdys pateiktas II priede.

Supūliavusio hidradenito klinikinis atsakas (angl. *Hidradenitis Suppurativa Clinical Response*, HiSCR).

Pacientų, kuriems taikoma biologinė terapija, gydymo efektyvumas vertinamas naudojant HiSCR skalę. Ši skalė buvo sukurta kaip priemonė įvertinti klinikinį atsaką į gydymą. Siūlomas atsako į gydymą apibrėžimas (pasiektas HiSCR) reiškia, kad bendras abscesų ir uždegiminių mazgų skaičius sumažėja bent 50 proc. nuo pradinio lygio (197).

Odos biopsija

Kartu su klinikiniais duomenimis, tiriamieji taip pat sutiko atlikti (3–5 mm) odos biopsijas, paimtas iš SH pažeidimų ir sveikai atrodančios odos 3–4 cm atstumu nuo pažeidimo. Gautos biopsijos buvo fiksuotos 10% formalino tirpale ir apdorotos Valstybiniame patologijos centre, kur buvo įterptos į parafino blokus ir saugomos būsimam naudojimui.

2.5. Statistinė analizė

Statistinė analizė atlikta naudojant *MS Excel* 2021 ir *IBM SPSS* 26.0, naudojant aprašomąją kokybinių ir kiekybinių kintamųjų statistiką. Grupių

homogeniškumas buvo įvertintas naudojant Chi kvadrato testą (x^2). Duomenų paskirstymo normalumas buvo įvertintas naudojant Šapiro-Vilko (angl. *Shapiro-Wilk*) kriterijų. Normaliai pasiskirsčiusiems duomenims buvo taikomas suporuotų mėginių T testas, o neparametriniai duomenys buvo analizuojami naudojant Vilkoksono (angl. *Wilcoxon*) kriterijų. Nepriklausomų grupių palyginimui buvo naudojami Mano ir Vitnio U (angl. *Mann-Whitney U*) ir Kruskalo-Voliso (angl. *Kruskal-Wallis*) kriterijai, o kokybiniams parametrams buvo naudojami Chi kvadrato (x^2) arba Fišerio tikslieji (angl. *Fischer's exact test*) testai. Skirtumai buvo laikomi statistiškai reikšmingais, kai p reikšmės buvo mažesnės nei 0,05 ($p<0,05$).

3. REZULTATAI

3.1. Tiriamųjų charakteristikos

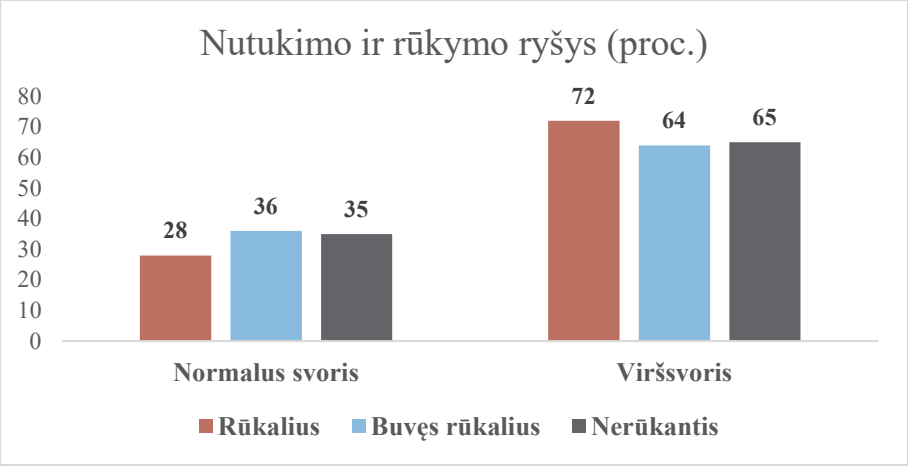
Tyrime dalyvavo 49 pacientai, iš kurių 57,14 proc. (N=28) buvo vyrai ir 42,86 proc. (N=21) moterys. Vidutinis tiriamųjų amžius – 39,91±13,665 metai, vidutinis KMI – 28,44±6,142, vidutinė juosmens apimtis – 91,85 cm, antsvorio turėjo 30,61 proc. (N=15) pacientų, o nutukę buvo 36,73 proc. (N=18) (**1 lentelė**).

1 lentelė. Vidutinis KMI, juosmens apimtis, svoris ir antsvorio rodiklis.

Kriterijai	**Pacientai (n=49)**	***P* reikšmė**
Vidutinis KMI kg/m2 Moterų KMI kg/m2 Vyrų KMI kg/m2	28,44±6,142 28,12±5,819 28,68±6,142	0,372
Liemens apimtis, cm Moterys, cm Vyrai, cm	91,85±16,806 85,90±19,944 96,32±12,619	0,077
Svoris, kg Moterys, kg Vyrai, kg	87.93±21.025 80.04±24.340 93.85±16.497	<0,001
Antsvoris, n, (%)	33 (67,35%)	

Statistinės koreliacijos tarp lyties ir KMI kintamųjų nebuvo. Nustatyta statistiškai reikšminga koreliacija tarp nutukimo ir rūkymo būklės kintamųjų ($r=0,673$, $p<0,01$). 72 proc. antsvorio turinčių asmenų rūkė, o normalaus svorio grupėje – tik 28 proc. 64 proc. antsvorio turinčių asmenų anksčiau buvo rūkaliai, o tik 36 proc. normalaus svorio asmenų patenka į šią kategoriją. 65

proc. antsvorio turinčių asmenų buvo nerūkantys, o normalaus svorio grupėje – 35 proc. (**1 pav**.).



1 pav. Rūkymo ir nutukimo ryšys tarp SH pacientų.

51,02 proc. (N=25) tiriamųjų turėjo aukštąjį išsilavinimą. Ligos sunkumas nepriklausė nuo plaukų struktūros (p=0,467) ar odos fototipo (p=0,631). Duomenys pasiskirstę pagal normalumo kreivę, grupės vienalytės (**2 lentelė**).

2 lentelė. SH sergančių pacientų charakteristikos: tiriamųjų pasiskirstymas pagal amžių, lytį, išsilavinimą, odos fototipą, plaukų struktūrą ir profesinę padėtį.

Kriterijai	**Pacientai (n=49)**	***P* reikšmė**
Amžius, metai		
Vidurkis±SD	39,91±13,665	0,565
Mediana (diapazonas)	39,24 (18,73)	
Lytis		
Moterys, n, (proc.)	21 (42,85)	0,378
Vyrai, n, (proc.)	28 (57,14)	
Išsilavinimo lygis		
Pradinis išsilavinimas, n (proc.)	7 (14,28)	
Vidurinis išsilavinimas, n (proc.)	17 (34,69)	
Aukštasis išsilavinimas, n (proc.)	25 (51,02)	
Profesinis statusas, n, (proc.)		

Kriterijai	Pacientai (n=49)	*P* reikšmė
Dirba/Studentas Bedarbis Nedarbingumas Pensininkas	41 (83,67) 2 (4,08) 5 (10,20) 1 (2,04)	
Odos fototipai, n, (proc.)		
I II III IV	15 (30,61) 19 (38,77) 13 (26,53) 2 (4,08)	
Plaukų struktūra, n (proc.)		
Tiesūs Banguoti Garbanoti	32 (65,30) 13 (26,53) 4 (8,16)	

Vidutinė ligos pradžia buvo 25,71±13,743 metų, pacientai kreipėsi į gydytoją vidutiniškai 28 metų amžiaus, o vidutinis laikas iki diagnozės buvo 5,2 ±7 607 metai. Moterims diagnozė buvo nustatyta žymiai vėliau nei vyrams: vidutiniškai po 6,5 metų, palyginti su 4,2 metais, atitinkamai (p=0,01) (**3 lentelė**).

3 lentelė. Ligos pradžios amžius, medicininės priežiūros pradžia ir laikas iki diagnozės nustatymo.

Kriterijai	Pacientai (n=49)	*P* reikšmė
Ligos pradžia (metai±SD)	25,71±13,743	0,425
- Moterys	26,61±13,93	0,865
- Vyrai	25,03±13,743	0,243
Pradėta medicininė priežiūra (metai±SD)	28,22±14,000	0,391
- Moterys	27,57±14,204	0,452
- Vyrai	28,71±14,003	0,841
Laikas iki diagnozės (metai±SD)	5,2±7,607	0,201
- Moterys	6,5±7,748	<0,001
- Vyrai	4,2±7,607	0,540

70,2 proc. pacientų (N=33) anksčiau buvo nustatyta klaidinga diagnozė; 51,51 proc. (N=17) tiriamųjų buvo diagnozuotas furunkulas (**4 lentelė**). 87,75 proc. (N=43) pacientų SH diagnozavo gydytojas dermatovenerologas.

4 lentelė. Dažniausios klaidingos diagnozės.

Klaidinga diagnozė, N, proc.	Pacientai (n=49)
Iš viso	33 (70,2)
Furunkulas	17 (51,51)
Opa	4 (12,12)
Abscesas	3 (9,09)
Aknė	2 (6,06)
Alergija; Cukrinio diabeto komplikacija; Mikozė; Limfadenitas; Pilonidinis sinusas; Folikulitas; Psoriazė	1 (3,03)

3.2. Gretutinės ligos

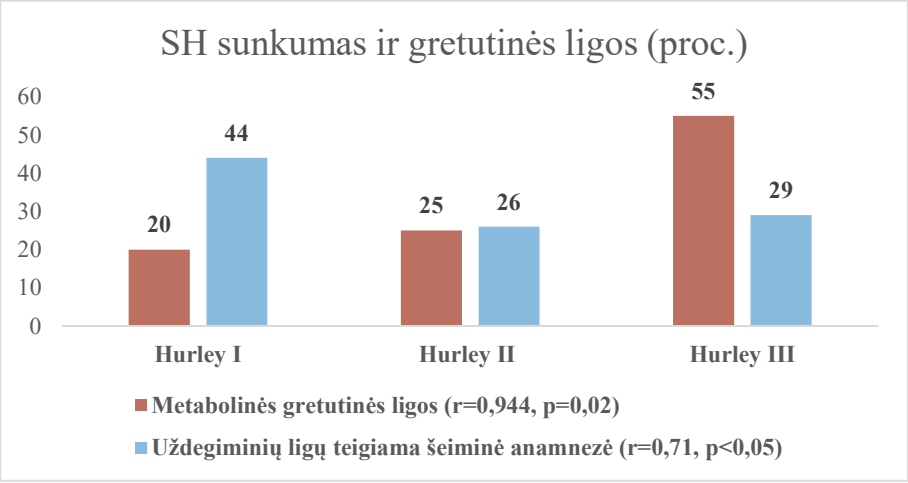
Nustatyta, kad 34,69 proc. (N=17) tiriamųjų turėjo sunkią aknę, o 64,70 proc. (N=11) iš jų – aknė vis dar tęsėsi; tačiau koreliacijos tarp aknės ir Hurley stadijos (r=0,088) ar SH sunkumo (r=0,091) nenustatyta. 61,22 proc. (N = 30) pacientų sirgo gretutinėmis ligomis, tokiomis kaip psoriazė, uždegiminė žarnyno liga, sąnarių ligos, metabolinė liga, dislipidemija ir hipertenzija (**5 lentelė**).

5 lentelė. SH pacientų demografiniai duomenys ir gretutinės ligos.

Kriterijai	**Pacientai (n = 49)**	***Hurley* stadijos:**	
Baltaodžiai, n, proc.	49 (100)	**I,** n, proc.	25 (51,02)
Metabolinės gretutinės ligos:		**II,** n, proc.	12 (24,48)
Hipertenzija, n, proc.	10 (29,4)	**III,** n, proc.	12 (24,48)
Dislipidemija, n, proc.	6 (12,24)	**Kitos gretutinės ligos:**	
Cukrinis diabetas, n, proc.	3 (6,12)	Depresija, n, proc.	8 (16,32)
Metabolinis sindromas, n, proc.	1 (2,04)	Sąnarių skausmas, n, proc.	6 (12,24)
Uždegiminės ligos:			
Aknė, n, proc.	17 (34,69)	Psoriazė, n, proc.	4 (8.16)
Pilonidinė cista	10 (29,4)	Uždegiminė žarnų liga	3 (6.12%)

18,36 proc. tiriamųjų (N=9) buvo teigiama SH anamnezė šeimos anamnezėje. Nustatyta vidutinė teigiama koreliacija tarp uždegiminių ligų šeiminėje anamnezėje (69,38 proc. (N=34)), įskaitant aknę, psoriazę, uždegimines žarnų ligas ir sąnarių ligas, bei SH sunkumo pagal Hurley stadiją ($r= 0,71$, $p<0,05$). 30,6 proc. (N=15) pacientų turėjo gretutinių ligų, susijusių

su širdies ir kraujagyslių ligomis, o 60 proc. (N=9) iš jų buvo teigiama šeiminė uždegiminių ligų anamnezė. Lyginant SH sunkumą, nustatyta stipri statistiškai reikšminga koreliacija (r=0,944, p=0,02) tarp metabolinių gretutinių ligų ir *Hurley* stadijos, 55,0 proc. (N=10) iš jų buvo nustatyta *Hurley* III stadija (**2 pav.**).



2 pav. Ryšys tarp *Hurley* stadijos ir gretutinių metabolinių ligų bei teigiamos uždegiminių ligų šeimimės anamnezės.

3.3. Rūkymas

36,73 proc. (N=18) tiriamųjų buvo aktyvūs rūkaliai, o 22,44 proc. (N = 11) – buvę rūkaliai, vidutiniškai surūkantys 26,56 pakelio per metus. 40,81 proc. (N=20) dalyvių buvo nerūkantys. Tarp 12 *Hurley* III stadijos pacientų, 91,66 proc. (N=11) buvo rūkaliai; tarp šių dviejų kintamųjų nustatyta statistiškai reikšminga koreliacija (r=0,659, p<0,01). Pasiskirstymas tarp rūkančiųjų ir nerūkančiųjų, turinčių skirtingą *Hurley* stadiją, taip pat buvo statistiškai reikšmingas (p<0,01). Rūkančiųjų vyrų yra statistiškai daugiau nei moterų (p<0,01): 33,33 proc. (N=7) moterų ir 78,57 proc. (N=22) vyrų.

3.4. Klinika

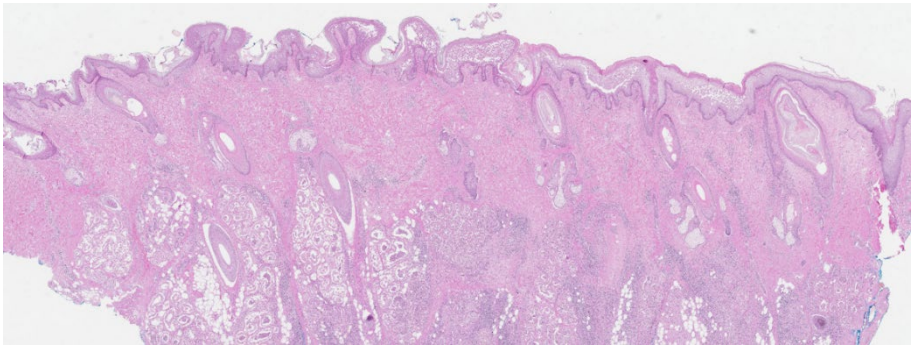
75,51 proc. pacientų (N=37) buvo pakitimų pažastų srityje, 59,18 proc. (N=29) kirkšnių srityje, 28,57 proc. (N=14) gaktos srityje ir 26,53 proc. (N=13) kitose srityse (**6 lentelė**). Tarp vyrų ir moterų reikšmingų skirtumų nebuvo. 88 proc. (N=16) nutukusių asmenų turėjo odos pažeidimų kirkšnių srityje.

6 lentelė. Vyrų ir moterų SH pažeidimų lokalizacijų pasiskirstymas.

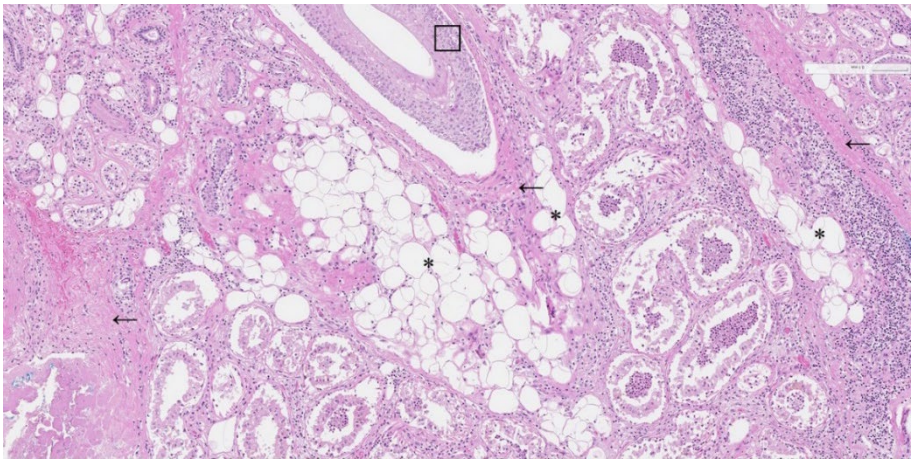
Pažeidimų lokalizacija	Visi pacientai (N=49)	Vyrai (N=28)	Moterys (N=21)	*p*-reikšmė
Pažastys, n, proc.	37 (75,51)	20 (71,42)	17 (80,95)	0,295
Kirkšnys, n, proc.	29 (59,18)	18 (64,28)	11 (52,38)	0,042
Gakta, n, proc.	14 (28,57)	8 (28,57)	6 (28,57)	0,395
Kitos sritys	13 (26,53)	7 (25,00)	6 (28,57)	0,467
SH pažeidimų tipai				
Pažastų sritis:				
Abscesai, n, proc.	7 (18,91)	4 (20)	3 (17,64)	0,271
Mazgai, n, proc.	21 (56,75)	9 (45)	12 (70,58)	0,249
Fistulės, n, proc.	9 (24,32)	5 (25)	4 (23,52)	0,543
Kirkšnių sritis:				
Abscesai, n, proc.	7 (24,13)	5 (27,77)	3 (27,27)	0,042
Mazgai, n, proc.	15 (51,72)	9 (50)	7 (63,63)	0,284
Fistulės, n, proc.	7 (24,13)	4 (22,22)	1 (9,09)	0,031
Gaktos sritis:				
Abscesai, n, proc.	4 (28,57)	2 (25)	1 (16,66)	0,038
Mazgai, n, proc.	8 (57,14)	4 (50)	4 (66,66)	0,782
Fistulės, n, proc.	2 (14,28)	2 (25)	1 (16,66)	0,081
Kitos sritys (veidas, smakras, pilvas, krūtys):				
Abscesai, n, proc.	6 (46,15)	4 (57,14)	2 (33,33)	0,246
Mazgai, n, proc.	5 (38,46)	3 (42,85)	3 (50)	0,291
Fistulės, n, proc.	2 (15,38)	0 (0)	1 (16,66)	0,823

3.5. SH pacientų histologiniai radiniai

Histologiškai ištyrus SH ankstyvuosius pažeidimus, nustatoma folikulinė hiperkeratozė, folikulinio epitelio hiperplazija ir perifolikulitas (**3 pav**.). Dermoje yra mišrus uždegiminių ląstelių infiltratas, kartais besitęsiantis į poodinį sluoksnį. Gali būti neutrofilinių abscesų, kurie dažnai jungiasi prie cistų ir sinusų takų, išklotų plokščiu epiteliu, kurie atsiveria į odos paviršių. Šiose cistose ir sinusiniuose taktuose paprastai yra laminuoto keratino, taip pat gali būti plaukų folikulų. Maždaug 25 proc. atvejų stebimas granuliuotas audinys su retkarčiais randamomis milžiniškomis svetimkūnio ląstelėmis. Kai kuriais atvejais uždegimas gali apimti apokrinines liaukas. Taip pat, aplink folikulų plyšimo vietas yra stebimas fibrozinis audinys (**4–6 pav.**).

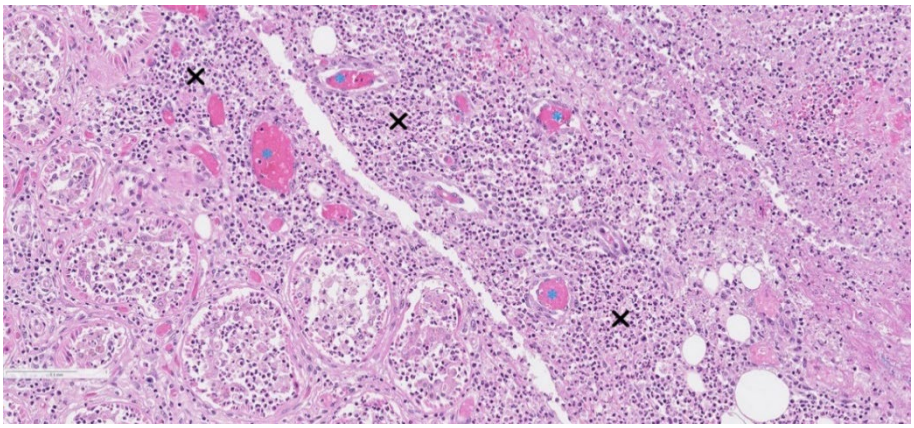


3 pav. SH histopatologija: ryški folikulinė hiperkeratozė, folikulų hiperplazija ir perifolikulitas.



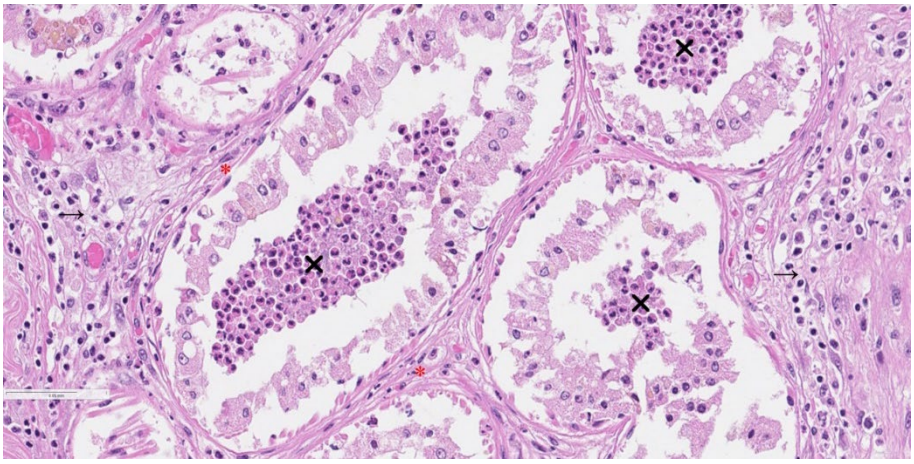
4 pav. SH histopatologija: išsiplėtę ir plyšę plaukų folikulai su keratinu ir uždegiminiu infiltratu. Uždegimininės ląstelės, ypač aplink plaukų folikulus, tai rodo nuolatinį uždegiminį atsaką.

Adipocitai (*). Uždegimas plinta į poodinį audinį, dėl kurio gali atsirasti skausmingų, gilių pažeidimų, būdingų SH. Granuliacinis audinys ir fibrozė (→). Tai rodo gijimą ir randų formavimąsi, dažnai stebimą lėtiniais ir pasikartojančiais ligos atvejais. Plauko folikulo epitelis (□).



5 pav. SH histopatologija: intensyvus uždegiminis infiltratas visoje dermoje.

Uždegiminis infiltratas (✕) apima neutrofilus, limfocitus, plazmos ląsteles ir histiocitus, rodančius ūminį ir lėtinį uždegimą. Gali atsirasti dėl audinių pažeidimo, kurį sukelia lėtinis uždegimas ir abscesų susidarymas. Kai kurios apokrininės liaukos yra išsiplėtusios dėl uždegiminių ląstelių infiltracijos, o tai yra būdingas SH radinys. Staziniai kapiliarai (*).



6 pav. SH histopatologija: išsiplėtusios apokrininės liaukos užpildytos uždegiminėmis ląstelėmis.

Uždegiminis infiltratas, daugiausia sudarytas iš neutrofilų (✕). Tai rodo ūminį uždegimą, dėl kurio liaukose gali susidaryti mikroabscesai. Neutrofilinė infiltracija yra aktyvaus, pūlingo uždegimo požymis. Liaukų epitelinis sluoksnis yra edemiškas ir deskvamuotas (*). Liaukas supanti stroma edemiška, o uždegiminės ląstelės infiltravusios į jungiamąjį audinį (→).

3.6. Klinikiniai SH potipiai

Visų pacientų SH fenotipas buvo nustatytas naudojant *Canoui-Pouitrine* ir kt. kriterijus (195). Nustatėme, kad 40,81 proc. pacientų (N = 20) turėjo pažastų-krūtų (angl. *axillary mammary*) fenotipą, 34,69 proc. (N = 17) folikulnį (angl. *follicular*) fenotipą ir 24,48 proc. (N = 12) sėdmenų (angl. *gluteal*) fenotipą. Folikulinis fenotipas buvo labiau paplitęs tarp nerūkančių pacientų, palyginus su pažastų-krūtų fenotipu, ir labiau paplitęs tarp *Hurley* III stadijos tiriamųjų nei sėdmenų ir pažastų-krūtų fenotipai (**7 lentelė**).

7 lentelė. Klinikiniai SH potipiai ir pasiskirstymas tarp tiriamųjų.

	Pažastų – krūtų fenotipas (N=20)	**Folikulinis fenotipas (N=17)**	**Sėdmenų fenotipas (N=12)**	***p*-reikšmė**
Lytis				
Moterys, n, proc.	9 (45)	7 (41,17)	5 (41,66)	0,651
Vyrai, n, proc.	11 (55)	10 (58,82)	7 (58,33)	0,743
Rūkymo statusas				
Rūkalius, n, proc.	12 (60)	7 (41,17)	5 (41,66)	<0,001
Nerūkantis, n, proc.	8 (40)	10 (58,82)	7 (58,33)	
***Hurley* stadija**				
I, n, proc.	13 (65)	6 (35,29)	6 (50)	0,231
II, n, proc.	5 (25)	3 (17,64)	4 (33,33)	0,482
III, n, proc.	2 (10)	8 (47,05)	2 (16,66)	<0,001

3.7. Skausmas

Vidutiniškai tiriamieji turėjo 6,17±6,98 skausmingų dienų per pastarąsias 4 savaites. Vidutinis skausmo intensyvumo balas pagal VAS skalę buvo 5,60±3,36. Moterys nurodė, kad jų skausmo intensyvumo balų vidurkis buvo 5,42 balo, vyrai – 5,74 (**8 lentelė**). 30,61 proc. (N = 15) tiriamųjų patyrė nuolatinį skausmą, o 69,38 proc, (N = 34) - periodinį. Vyrai statistiškai reikšmingai dažniau skundėsi protarpiniu skausmu (p=0,003). 24,48 proc. (N=12) tiriamųjų teigė kad SH paūmėjimą sukelia mechaninis spaudimas ar trintis.

8 lentelė. SH sukeltas skausmas: skausmingų dienų skaičius per pastarąsias 4 savaites ir vidutiniai VAS balai.

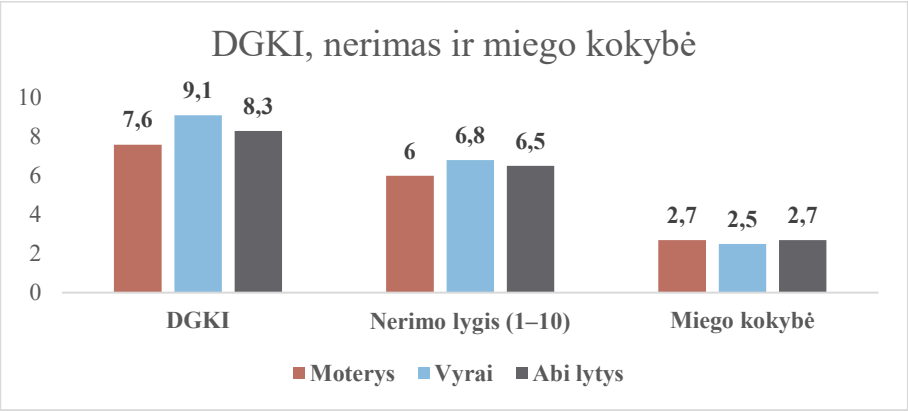
Kriterijai	Visi pacientai (N=49)	Moterys (N=21)	Vyrai (N=28)	p-reikšmė
Skausmingos dienos per pastarąsias 4 savaites (±SD)	6,17±6,98	6,61±7,06	5,92±6,85	0,005
Vidutinis VAS balas (±SD)	5,60±3,36	5,42±3,34	5,74±3,36	0,003
Nuolatinis skausmas, n, proc.	15 (30,61)	8 (38,09)	7 (25)	0,062
Protarpinis skausmas, n, proc.	34 (69,38)	13 (61,90)	21 (75)	0,003

3.8. Ligos našta ir gyvenimo kokybė

36,73 proc. (N=18) pacientų buvo hospitalizuoti dėl SH, vidutiniškai 19,5 dienas; iš jų 66,66 proc. (N=12) buvo vyrai ir 61,11 proc. (N=11) sirgo III *Hurley* stadija. 26,53 proc. (N=13) visų tiriamųjų pripažino, kad SH turėjo įtakos jų profesinei karjerai. 24,48 proc. (N=12) pacientų neteko darbo dėl SH. Per 6 mėnesius pacientai vidutiniškai praleido 20,5 darbo dienos. Iš jų 76,92 proc. (N=10) turėjo aukštąjį išsilavinimą.

Vidutinis pacientų nerimo lygis buvo 6,5±2,586 balai iš 10. Vyrai dėl ligos nerimauja dažniau, savo nerimo lygį įvertino vidutiniškai 6,84 balais, o moterys - 6,04 balais (p=0,014). Amžius koreliavo su nerimo lygiu – kuo jaunesnis pacientas, tuo didesnis nerimo balas (r=0,231, p=0,002). Ligos poveikį miego kokybei pacientai įvertino vidutiniškai 2,68±3,060 balų iš 10. 77,55 proc. (N=38) savo nerimo lygį pažymėjo >5, visų šių pacientų miego kokybė buvo blogesnė nei tiriamųjų, kurių nerimo balas < 5 (p=0,02).

Vidutinis pacientų DGKI tyrimo pradžioje buvo 8,30±7,461 balai (**7 pav**.). Iš viso 32,65 proc. (N=16) pacientų DGKI buvo >10, iš jų 62,5 proc. (N=10) buvo vyrai. Iš viso 93,75 proc. pacientų (N=15) KMI buvo >25 (p<0,01), o jų nerimo lygis buvo 7,5 balo.



7 pav. SH pacientų dermatologinė gyvenimo kokybė, nerimo lygis ir miego kokybė.

3.9. Gydymas

Prieš ištyrimą pacientai vartojo įvairius vaistus nuo SH, kurie pateikti **9 lentelėje**.

9 lentelė. Ankstesnis SH sergančių pacientų gydymas.

Vaistai/gydymas	**Pacientai (N=49)**	**Efektyvumas (N, proc.)**
Vietinis gydymas		
Antiseptikai, n, proc.	29 (59,18)	Dalinis (N=28, 96,55) Jokio (N=1, 3,44)
Antibiotikai, n, proc.	46 (92,03)	Dalinis (N=44, 95,65) Jokio (N=1, 2,17) Visiškas (N=1, 2,17)
Gliukokortikosteroidų injekcijos į bėrimus, n, proc.	10 (20,40)	Dalinis (N=7, 70) Jokio (N=2, 20) Visiškas (N=1, 10)
Sisteminis gydymas		
Nesteroidiniai vaistai nuo uždegimo, n, proc.	5 (10,20)	Dalinis (N=4, 80) Jokio (N=1, 20)
Doksiciklinas, n, proc.	31 (63,26)	Dalinis (N=24, 77,41) Jokio (N=4, 12,90) Visiškas (N=3, 9,6)
Rifampicinas + Klindamicinas, n, proc.	17 (34,69)	Dalinis (N=15, 88,23) Visiškas (N=2, 11,76)
Klindamicinas, n, proc.	10 (20,40)	Dalinis (N=9, 90) Visiškas (N=1, 10)

Vaistai/gydymas	Pacientai (N=49)	Efektyvumas (N, proc.)
Penicilinas + Klavulaninė rūgštis, n, proc.	5 (10,20)	Dalinis (N=2, 40) Jokio (N=3, 60)
Retinoidai, n, proc.	7 (14,28)	Dalinis (N=5, 71,42) Jokio (N=1, 14,28) Visiškas (N=1, 14,28)
Adalimumabas, n, proc.	14 (28,57)	Dalinis (N=12, 85,71) Visiškas (N=2, 14,28)
Intervencijos		
Incizija ir drenažas, n, proc.	24 (48,97)	Dalinis (N=14, 58,33) Visiškas (N=10, 41,66)

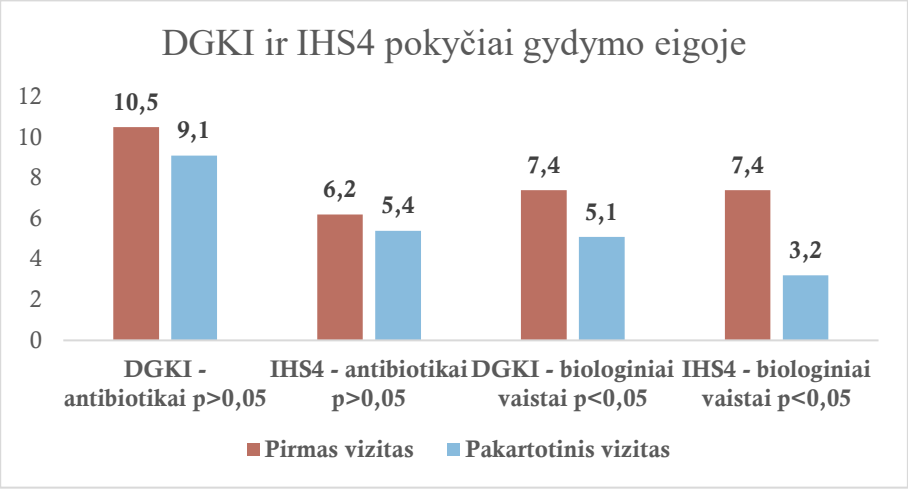
Po pirminio pacientų įvertinimo dažniausiai buvo skiriami šie vaistai, pateikti **10 lentelėje**.

10 lentelė. Dažniausiai skiriami gydymo būdai SH sergantiems pacientams.

Vaistai/gydymas	Pacientai (n=49)
Vietinis gydymas	
Antiseptikai, n, proc.	49 (100)
Klindamicinas, n, proc.	44 (89,79)
Gliukokortikosteroidų injekcijos į bėrimus, n, proc.	1 (2,04)
Sisteminis gydymas	
Rifampicinas + Klindamicinas, n, proc.	8 (16,32)
Doksiciklinas, n, proc.	17 (34,69)
Klindamicinas, n, proc.	7 (14,28)
Retinoidai, n, proc.	3 (6,12)
Biologinė terapija	
Adalimumabas, n, proc.	11 (22,44)
Sekukinumabas, n, proc.	5 (10,20)
Intervencijos	
Incizija ir drenažas, n, proc.	3 (6,12)

32,65 proc. (N=16) pacientų buvo gydomi biologine terapija, 65,30 proc. (N=32) vartojo sisteminius antibiotikus, 14,89 proc. (N=7) vartojo ir biologinius, ir sisteminius antibiotikus, o 44,68 proc. (N=21) buvo gydomi tik vietiniais vaistais. Pacientų, vartojusių biologinius vaistus, vidutinis DGKI tyrimo pradžioje buvo 7,4, sisteminius antibiotikus vartojusių – 10,5, o kitų – 7. Po 6 mėnesių nuo pirminio įvertinimo ir vaistų skyrimo buvo atliktas pakartotinis pacientų ištyrimas. Biologine terapija gydomų pacientų vidutinis

IHS4 gydymo pradžioje buvo 7,38, o pakartotinio vizito metu – 3,22 (p<0,05). Tuo tarpu pacientų, nevartojančių biologinių vaistų, pradinis IHS4 balas buvo 6,21, pakartotinio vizito metu – 5,42 (p>0,05) (**8 pav**.). Nustatėme, kad 29,78 proc. (N=14) pasikartojančių uždegiminių pažeidimų per paskutines 4 savaites nesiskyrė tarp pacientų, vartojusių biologinius ar sisteminius antibiotikus.



8 pav. Sisteminio SH gydymo įtaka DGKI ir IHS4 balams.

3.9.1. Adalimumabo tyrimas

Adalimumabą vartojančių pacientų demografiniai duomenys

Iš viso į šią tyrimo dalį buvo įtrauktas 21 pacientas, kuriam diagnozuotas vidutinio sunkumo ar sunkus SH, iš kurių 8 moterys (38,1 proc.) ir 13 vyrų (61,9 proc.), kurių amžiaus vidurkis buvo 42,9±14,1 metai. Vidutinis pacientų KMI buvo 30,33±7,13, 8 pacientams (38,1 proc.) buvo antsvoris, o 8 (38,1 %) pacientai – nutukę (**11 lentelė**).

11 lentelė. Adalimumabą vartojančių pacientų demografinės charakteristikos.

Kriterijai	**Pacientai (N=21)**
Lytis	
Moterys, n, proc.	8 (38,1)
Vyrai, n, proc.	13 (61,9)
Vidutinis amžius, metais, (±SD)	42,9 (±14,1)
Vidutinis KMI kg/m^2 (±SD)	30,33 (±7,13)
Normalus (18.5–24.9), n, proc.	5 (23,8)
Viršsvoris (25.0–29.9), n, proc.	8 (38,1)
Nutukimas (≥30.0), n, proc.	8 (38,1)

Adalimumabą vartojančių pacientų klinikinės charakteristikos

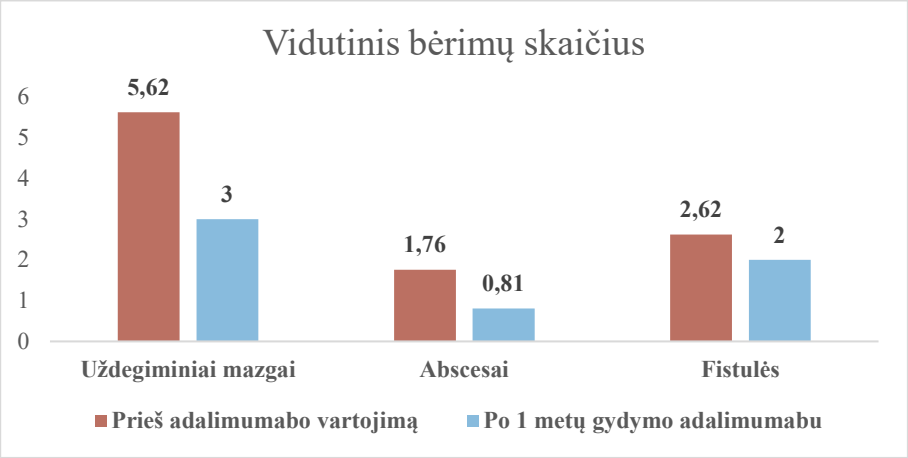
13 asmenų (61,9 proc.), buvo priskirti *Hurley* III stadijai. Vidutinė ligos trukmė – 15,48±12,83 metų. Visi (100 proc.) pacientai anksčiau buvo gydomi sisteminiais antibiotikais, iš kurių 14 (66,7 proc.) anksčiau buvo gydomi chirurginiu būdu. Vidutinis pradinis IHS4 balas buvo 19±10,78, daugumai pacientų buvo sunkus SH. Pradinis DGKI balo vidurkis buvo 15,76±7,73, o tai reiškia labai didelį ligos poveikį asmens gyvenimo kokybei. Vidutinis skausmo intensyvumas pagal VAS pradžioje buvo 6,69±1,59, o tai rodo, kad pacientai patyrė vidutinio sunkumo skausmą (**12 lentelė**).

12 lentelė. Pradinės klinikinės adalimumabą vartojančių pacientų charakteristikos.

Kriterijai	**Pacientai (N=21)**
***Hurley* stadija, n, proc.**	
II	8 (38,1)
III	13 (61,9)
Vidutinė SH trukmė, metais (±SD)	15,48 (±12,83)
Ankstesnis sisteminis antibiotikų vartojimas, n, proc.	21 (100)
Ankstesnis chirurginis SH gydymas, n, proc.	14 (66,7)
Bėrimų skaičius	
Vidutinis uždegiminių mazgų skaičius (±SD)	5,62 (±4,12)
Vidutinis abscesų skaičius (±SD)	1,76 (±2,63)
Vidutinis fistulių skaičius (±SD)	2,62 (±1,86)
Vidutinis IHS4 įvertis (±SD)	19 (±10,78)
Vidutinis (4–10), n, proc.	7 (33,3)
Sunkus (≥11), n, proc.	14 (66,7)
Vidutinis DGKI balas (±SD)	15,76 (±7,73)
Vidutinis VAS įvertis (±SD)	6,69 (±1,59)

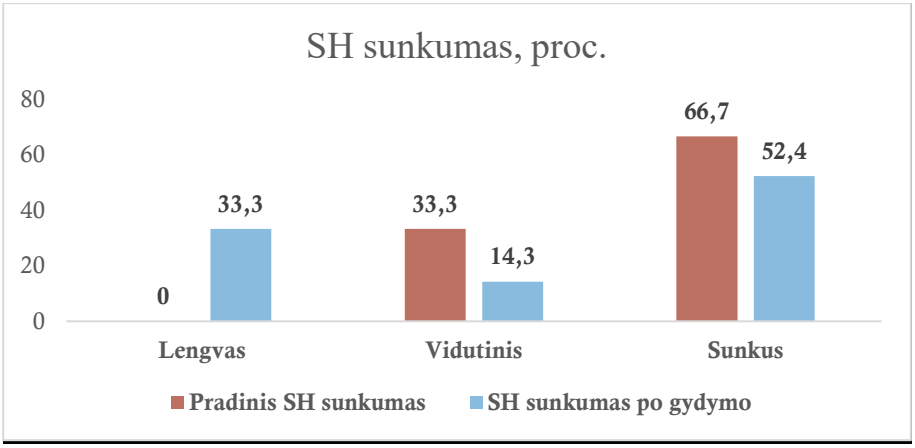
Po vienerių metų gydymo adalimumabu 10 (47,62 proc.) pacientų pasiekė HiSCR. Po vienerių metų gydymo buvo pastebėtas reikšmingas visų SH pažeidimų sumažėjimas, palyginti su pradiniu skaičiumi ($p<0,05$). Vidutinis uždegiminių mazgų skaičius sumažėjo nuo 5,62±4,12 iki 3±3,46,

vidutinis abscesų skaičius sumažėjo nuo 1,76±2,63 iki 0,81±1,4, o vidutinis fistulių skaičius sumažėjo nuo 2,62±1,86 iki 2,9±1,9 (**9 pav.**).



9 pav. Vidutinis uždegiminių mazgų, abscesų ir fistulių pažeidimų skaičius prieš adalimumabo vartojimą ir po 1 metų gydymo.

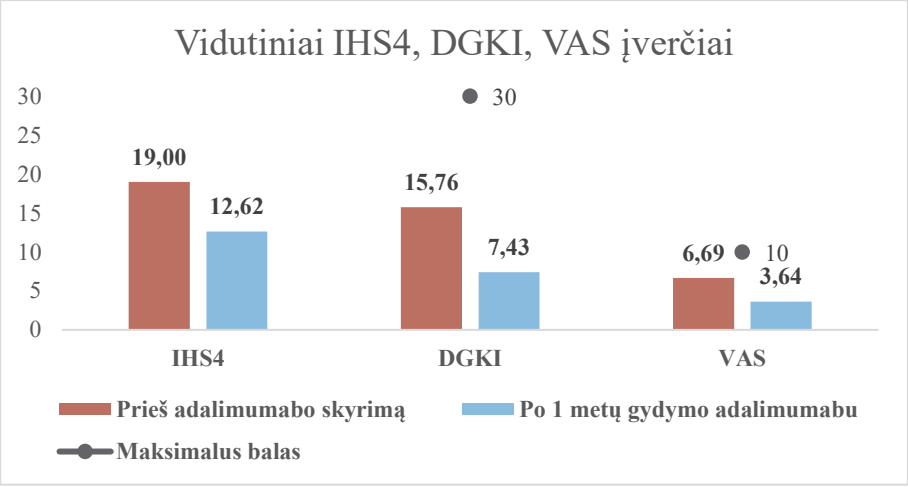
Prieš vartojant adalimumabą, 14 pacientų (66,7 proc.) buvo sunkus SH pagal IHS4 įvertį, po gydymo sunkus SH išliko 11 pacientų (52,4 proc.). Pažymėtina, kad iš pradžių nebuvo pacientų, kuriems buvo lengvas SH, tačiau po vienerių metų gydymo 7 pacientai (33,3 proc.), kuriems buvo vidutinio sunkumo ar sunki SH forma, perėjo į lengvo SH kategoriją (**10 pav**.).



10 pav. SH sunkumo kategorijų pasiskirstymas prieš gydymą adalimumabu ir po 1 metų gydymo.

Uždegiminių pažeidimų skaičiaus sumažėjimas atitiko IHS4 įverčio sumažėjimą. IHS4 balo sumažėjimas po 1 metų gydymo adalimumabu buvo

statistiškai reikšmingas, palyginti su pradiniu lygiu (p=0,001). Vidutinis IHS4 balas nuo 19±10,78 sumažėjo iki 12,62±11,13. Taip pat statistiškai reikšmingai pagerėjo pacientų gyvenimo kokybė pagal DGKI balus (p<0,001). Prieš gydymą vidutinis DGKI balas buvo 15,76±7,73, o po vienerių metų gydymo sumažėjo iki 7,43±7,76. Pradinis vidutinis VAS balas nuo 6,69±1,59 statistiškai reikšmingai sumažėjo iki 3,64±2,65 (p<0,001) (**11 pav**.).



11 pav. Vidutiniai IHS4, DGKI ir VAS balai prieš adalimumabo vartojimą ir po vienerių gydymo metų.

KMI ir chirurginio gydymo įtaka

Reikšmingo ryšio tarp KMI kategorijos (normalus, antsvoris ir nutukimas) ir HiSCR pasiekimo (p=0,350) nebuvo (**13 lentelė**). HiSCR pasiekusių vidutinis KMI buvo 32,17±8,66, o nepasiekusių šio klinikinio balo vidutinis KMI buvo 28,65±5,27.

13 lentelė. KMI kategorijos ir HiSCR pasiekimo ryšys.

	Pasiektas HiSCR (n = 10)	**Nepasiektas HiSCR (n = 11)**	**Iš viso (n = 21)**	***p*-reikšmė**
Normalus KMI, n, (proc.)	3 (60)	2 (40)	5	0.,50
Viršsvoris, n, (proc.)	5 (62,5)	3 (37,5)	8	
Nutukimas, n,(proc.)	2 (25)	6 (75)	8	

Nebuvo pastebėta statistiškai reikšmingų skirtumų pradinio IHS4 įverčio (p=0,928), pradinio DGKI balo (p=0,232) ir pradinio VAS balo (p=0,316) tarp pacientų, kurių KMI normalus, ir turinčių antsvorį ar nutukimą. Didžiausios vidutinės IHS4 ir VAS reikšmės buvo pacientų, kurių KMI normalus, (atitinkamai 20,4±13,01 ir 7,4±1,08); tačiau didžiausias vidutinis DLQI (19,25±7,23) buvo antsvorį turinčių pacientų grupėje (**14 lentelė**).

14 lentelė. Pradinių vidutinių IHS4, DGKI ir VAS įverčių pasiskirstymas tarp KMI grupių.

	Normalus KMI	**Viršsvoris**	**Nutukimas**	***p*-reikšmė**
Vidutinis IHS4 (±SD)	20,4 (±13,01)	19,75 (±13,18)	17,38 (±7,52)	0,928
Vidutinis DGKI (±SD)	12,8 (±7,95)	19,25 (±7,23)	14,13 (±7,68)	0,232
Vidutinis VAS (±SD)	7,4 (±1,08)	6,13 (±1,71)	6,81 (±1,69)	0,316

Taip pat nesiskyrė pacientų, pasiekusių HiSCR, santykis tarp tų, kuriems anksčiau buvo atliktas chirurginis gydymas, ir tų, kuriems nebuvo taikytas chirurginis gydymas (p=0,659) (**15 lentelė**).

15 lentelė. Ryšys tarp pacientų, kuriems buvo atliktas arba netaikytas chirurginis gydymas, ir HiSCR pasiekimas.

	Pasiekė HiSCR (n = 10)	**Nepasiekė HiSCR (n = 11)**	**Iš viso (n = 21)**	***p*-reikšmė**
Ankstesnis chirurginis gydymas, n, (proc.)	6 (42,86)	8 (57,14)	14	0,659
Netaikytas chirurginis gydymas, n, (proc.)	4 (57,14)	3 (42,86)	7	

Prieš gydymą adalimumabu buvo reikšmingas IHS4 balų skirtumas (p=0,001) tarp pacientų, kuriems anksčiau buvo atlikta operacija (vidutinis balas 23,86±9,4), palyginti su tais pacientais, kuriems anksčiau nebuvo atlikta SH operacija (vidutinis balas 9,29±5,53). Statistiškai reikšmingų pradinių DGKI (p=0,585) ir VAS balų (p=0,4) skirtumų tarp pacientų grupių, kurioms buvo atlikta ir neatlikta SH operacija nenustatyta. Vidutinis DGKI ir VAS balų

vidurkis buvo didesnis pacientams, kuriems anksčiau nebuvo taikytas operacinis gydymas dėl SH (**16 lentelė**).

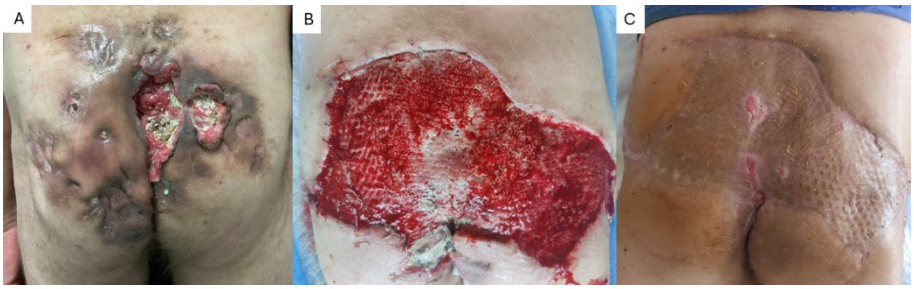
16 lentelė. Pradinių vidutinių IHS4, DGKI ir VAS įverčių pasiskirstymas tarp pacientų, kuriems buvo atliktas chirurginis gydymas, ir tų, kuriems nebuvo atliktas chirurginis gydymas.

	Taikytas chirurginis gydymas	**Netaikytas chirurginis gydymas**	***p*-reikšmė**
Vidutinis IHS4 (±SD)	23,86 (±9,4)	9,29 (±5,53)	0,001
Vidutinis DGKI (±SD)	15,5 (±8,92)	16,29 (±5,12)	0,585
Vidutinis VAS (±SD)	6,46 (±1,82)	7,14 (±0,9)	0,4

3.10. Klinikinė patirtis

Ilgai negydomas odos ir gleivinių uždegimas ilgainiui gali progresuoti į piktybinį procesą, dažniausiai – plokščiųjų ląstelių karcinomą. Vienas iš tyrime dalyvavusių pacientų 16 metų sirgo sunkiu SH, kuris pastaruosius 5 metus buvo gydomas 40 mg adalimumabu kas savaitę. Nepaisant stabilios ligos gydant anti-TNF, 2023 m. pacientas patyrė atkrytį. Fizinio ištyrimo metu nustatyti nauji pūlingi bėrimai, difuzinė infiltracija ir opos kryžkaulio bei tarpvietės srityse. Liga atitiko **Hurley** III stadiją (**12A pav**.).

Dėl paciento būklės pokyčių adalimumabas galiausiai buvo nutrauktas. Dėl daugybei vaistų atsparios *Acinetobacter baumanii* ir labai padidėjusių uždegiminių žymenų (C reaktyvusis baltymas – 76,82 mg/l) taikyta kombinuota intraveninė antibiotikų terapija. Biopsija iš opos krašto patvirtino gerai diferencijuotą plokščiųjų ląstelių karcinomą (SCC), kurios didelės rizikos žmogaus papilomos viruso (ŽPV) testas buvo neigiamas. Kompiuterinės tomografijos (KT) vaizduose matyti kryžkaulio pažeidimas su infiltracija, odos pažeidimas ir kirkšnies limfadenopatiją. Įtartinas židinys dešiniojo plaučio S6 segmente rodė galimą metastazę. Pacientą konsultavo daugiadisciplininė komanda. Buvo pradėtas spindulinės terapijos kursas, siekiant sumažinti naviko dydį, po kurio buvo atlikta plati ekscizija, dėl kurios atsirado 20x20 centimetrų audinio defektas (**12B pav**.) Atlikta rekonstrukcinė operacija, kartu pritaikyta vakuuminio uždarymo (VAC) sistema, etapinis debridementas, žaizdų drėkinimas ir tvarsčiai. Odos persodinimas buvo atliktas keturis kartus. Po 6 mėnesių atlikta kontrolinė KT neparodė jokio ligos progresavimo. Pacientas buvo atidžiai stebimas kas 3 mėnesius, odos transplantatas beveik visiškai padengė defektą (**12C pav**.), tačiau pacientas mirė praėjus 10 mėnesių po operacijos dėl nežinomų priežasčių.



12 pav. A – pūlingi bėrimo elementai, difuzinė infiltracija ir išopėjimas kryžkaulio ir perianalinėje srityje. B – plati ekscizija su reikšmingu audinio defektu, po kurios atliekama pirmoji odos persodinimo procedūra. C – kontrolė po 4 mėnesių po paskutinės odos persodinimo procedūros.

4. IŠVADOS

a) SH dažnai prasideda 20-ųjų viduryje, tiksli diagnozė nustatoma pradėjus 5,2 m. nuo simptomų pradžios, dažnai dėl klaidingos diagnozės, ypač moterims. *Šie rezultatai sutampa su ankstesniais tyrimais, kurie taip pat pabrėžia ligos pradžią ankstyvame suaugusiojo amžiuje ir diagnozės uždelsimą dėl klaidingų diagnozių.*

b) Daugelis pacientų turi antsvorio arba yra nutukę, o rūkymas yra susijęs su didesniu ligos sunkumu. *Šios išvados sutampa su ankstesniais tyrimais, kurie patvirtina, kad nutukimas yra dažnai nustatomas SH sergantiems pacientams, o rūkymas yra svarbus rizikos veiksnys, pabloginantis ligos sunkumą.*

c) Trečdalio pacientų DGKI >10 b., įrodant reikšmingą poveikį gyvenimo kokybei, ypač vyrams. Jaunesni pacientai turi didesnį nerimo lygį. *Nors neigiamas SH poveikis gyvenimo kokybei yra gerai dokumentuotas, mūsų tyrimas prideda naują įžvalgą, kad ši liga labiau neigiamai veikia vyrų gyvenimo kokybę nei moterų, o jaunesni pacientai patiria didesnį nerimą.*

d) Šeimos uždegiminių ligų ir metabolinių gretutinių ligų istorija yra susijusi su didesniu SH sunkumu. Pažeidimai dažniausiai paveikia pažastų ir kirkšnių sritis, nepriklausomai nuo lyties. *Lietuvoje iki šiol nebuvo nagrinėtas teigiamos šeiminės anamnezės ir metabolinių gretutinių ligų ryšys su SH sunkumu, suteikiant naujų įžvalgų apie galimus genetinius ir metabolinius rizikos veiksnius.*

e) Adalimumabas sumažina uždegiminius pažeidimus ir pagerina gyvenimo kokybę, maždaug 50 proc. pacientų pasiekia HiSCR. Ankstesnė SH operacija yra susijusi su didesniu ligos sunkumu. *Šie rezultatai sutampa su įvairiuose tyrimuose pateiktais duomenimis apie adalimumabo veiksmingumą ir taip pat akcentuojant būtinybę tobulinti SH gydymo metodus.*

5. PASIŪLYMAI IR TĘSTINUMAS

- Ankstyva SH diagnozė yra labai svarbi siekiant pagerinti pacientų gydymo rezultatus. Būtina didinti kitų Lietuvos sveikatos priežiūros specialistų žinomumą apie SH, kad būtų galima laiku nustatyti diagnozę ir skirti savalaikį gydymą. Tai galima pasiekti vykdant profesinį švietimą ir visuomenės informavimo kampanijas, padedančias dermatovenerologams ir kitiems specialistams ankstyvoje stadijoje atpažinti SH ir nukreipti pacientus atitinkamam gydymui.
- Pagrindinių dviejų rizikos veiksnių – nutukimo ir rūkymo – koregavimas itin svarbus geresnei ligos kontrolei. Nauji nutukimo gydymo metodai gali padėti kontroliuojant SH glaudžiai bendradarbiaujant su endokrinologais, dietologais ir nutukimo chirurgais.
- Ankstyvas biologinės terapijos naudojimas gali atlikti pagrindinį vaidmenį stabdant SH sergančių pacientų ligos progresavimą. Ankstyvoje stadijoje pradėtas biologinis gydymas gali padėti kontroliuoti uždegimą ir užkirsti kelią sunkių pažeidimų ir komplikacijų vystymuisi, galiausiai pagerinant paciento gyvenimo kokybę ir sumažinant intensyvesnių intervencijų poreikį.
- Kombinuotas požiūris, taikant ir biologinį, ir chirurginį gydymą, gali duoti geresnių rezultatų gydant SH, ypač pacientams, sergantiems pažengusia liga. Biologiniai vaistai gali sumažinti uždegimą ir kontroliuoti ligos aktyvumą, o chirurginės procedūros gali pašalinti esamus pažeidimus, abscesus ir sinusinius takus, taip prisidedant prie visapusiškesnio ligos valdymo.
- SH centro sukūrimas Lietuvoje galėtų sutrumpinti laiką iki diagnozės didinant ligos žinomumą. Centre galėtų būti taikoma tarpdisciplininė pacientų priežiūra pasitelkiant modernų konservatyvų ir chirurginį gydymą, svorio mažinimą, psichinės sveikatos gerinimą, taip mažinant ligos sukuriamą naštą.
- Nuolatiniai tyrimai ir naujų vaistų kūrimas yra būtini siekiant patenkinti pacientų, sergančių SH, poreikius. Nauji gydymo būdai, kurie veiktų įvairius ligos patofiziologijos aspektus, galėtų suteikti papildomų galimybių pacientams, kuriems nėra teigiamo atsako į esamą gydymą.

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- Gečaitė, A. Gedminaitė, T. Raudonis, J. Grigaitienė. Gonorrhoea and chlamydia clinical presentation, diagnosis and treatment: VUL SH DVC experience. Public Health, 2019; 2(85): 46-55
- T. Raudonis, R.A. Vankeviciute, A. Lideikaite, A.G. Grigaityte, J. Grigaitiene. Contact Sensitisation in Patients with Chronic Leg Ulcers. Advances in skin and wound care, 2019; 32(12):558-562
- T. Raudonis, A. Gliebutė, A.G. Grigaitytė, Ž. Lukošiūnaitė, J. Grigaitienė. A Six-Year Analysis of Biological Therapy for Severe Psoriasis in a Lithuanian Reference Centre of Dermatovenereology. Medicina, 2020; 56(6):275.
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- Ulianskaite G, Timinskaite F, Raudonis T. Severe pityriasis rubra pilaris complicated with Kaposi's varicelliform eruption and cutaneous MRSA infection case report. Heliyon. 2024 Jun 27;10(13):e33750. doi: 10.1016/j.heliyon.2024.e33750. PMID: 39040271; PMCID: PMC11261859.
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- R. Matulionytė, M. L. Jakobsen, V. I. Grecu, J. Grigaitiene, T. Raudonis, L. Stoniene, M. Olteanu, L. de la Mora, D. Raben and A. K. Sullivan. Increased integrated testing for HIV, hepatitis C and sexually transmitted infections in health care facilities: results from the INTEGRATE Joint Action pilots in Lithuania, Romania and Spain. BMC Infect Dis 2021, 21(Suppl 2):845
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- Jokubaitė J, Janušonytė E, Raudonis T. Dermatological indicator condition guided testing for HIV: experience of a reference dermatology center. J Dtsch Dermatol Ges. 2025 Mar 30. doi: 10.1111/ddg.15680. Epub ahead of print. PMID: 40159788.

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PUBLICATION LIST

I. Raudonis T, Šakaitytė A, Vileikis TP, Černel V, Gancevičiene R, Zouboulis CC. Demographic Data, Risk Factors, and Disease Burden of HS Patients in Lithuania at a Reference Center. Healthcare (Basel). 2024 Sep 14;12(18):1849. doi: 10.3390/healthcare12181849. PMID: 39337190; PMCID: PMC11431364. IF *Impact factor 2.8*
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II. Raudonis T, Šakaitytė A, Vileikis TP, Černel V, Gancevičienė R, Zouboulis CC. Comorbidities, Clinical Presentation, Subtypes, and Treatment of HS Patients in Lithuania. J Clin Med. 2024 Jul 3;13(13):3900. doi: 10.3390/jcm13133900. PMID: 38999466; PMCID: PMC11242771. *Impact factor 4.9*
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III. Šakaitytė A, Česnavičiūtė I, Raudonis T. Assessing the Role of Adalimumab in Treating Hidradenitis Suppurativa: Findings from a Retrospective Study at a Reference Center. Clin Pract. 2024 Aug 27;14(5):1696-1706. doi: 10.3390/clinpract14050135. PMID: 39311285; PMCID: PMC11417930. *Impact factor 1.7*
Personal role in the publication: study concept, data analysis oversight, manuscript editing and review

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Personal role in the publication: direct patient treatment and supervision, manuscript concept, manuscript editing and review

PRESENTATION LIST

I. Raudonis T. Biologics for inflammatory skin diseases. LDVD international conference Dialogues in Dermatovenereology 2023. 4 MAY 2023. Vilnius, Lithuania.

II. Raudonis T. Hidradenitis suppurativa: demographic data, diagnosis, clinical presentation, medical and surgical treatment. LDVD international conference Dialogues in Dermatovenereology 2023. 4 MAY 2023. Vilnius, Lithuania.

III. Raudonis T., Cernel V., Ganceviciene R., Zouboulis C.C. Analysis of HS patient demographics and clinical data in Lithuania. *ePoster*. 12th EHSF Conference. 8–10th FEB 2023. Florence, Italy.

IV. Raudonis T., Sakaityte A., Vileikis T., Cernel V., Ganceviciene R., Zouboulis C.C. HS patient demographics and clinical data from Lithuania. *ePoster*. 13th EHSF Conference. 7–9th FEB 2024. Lyon, France

V. Sakaityte A., Cesnaviciute I., Raudonis T., Ganceviciene R. Adalimumab efficacy in the management of hidradenitis suppurativa data from Lithuania. *ePoster*. 14th EHSF Conference. 12–14th FEB 2025. Vilnius, Lithuania

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