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Gastric polyps: diagnosis and treatment principles

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10. References

Summary

Gastric polyps are lesions which arise from different cells or epithelial tissue in the stomach and with different causes, histology, malignant potential, and association with different tumor predisposition syndromes. These lesions are usually benign but because they represent proliferative growth they can contain the potential for malignant transformation. Gastric polyps have many subsets, the most commonly seen and described are the triad of gastric hyperplastic polyps characterized by pronounced foveolar hyperplasia, fundic gland polyps, characterized by dilated and irregularly budded fundic glands predominantly lined by parietal cells with smaller proportion of chief cells, and adenomatous polyps characterized by low-grade glandular dysplasia. Gastric polyps are usually found incidentally on upper gastrointestinal endoscopy performed for an unrelated indication and only in rare cases do they cause the symptoms. Nevertheless, the diagnosis and appropriate management of gastric polyps are important, as some polyps have malignant potential.

Keywords

Gastric Polyps, Gastrointestinal endoscopy, Fundic gland polyps, Hyperplastic polyps, Adenomatous polyps, Endoscopic mucosal resection

Introduction

Gastric polyps are intraluminal projections of mucosal or submucosal tissue which are usually asymptomatic. While these lesions are typically benign, they do have the potential of containing local dysplasia and progression to invasive cancer. The risk can be reduced by proper understanding and management of gastric polyps.(1)

Gastric polyps are usually found incidentally on upper gastrointestinal endoscopy performed for an unrelated indication and only in rare cases do they cause symptoms. (2)

Prior to the advent of digestive endoscopy, gastric polyps were typically diagnosed through x-ray examinations, with surgical intervention being the primary treatment approach. Heinz, in 1911, was the pioneer in identifying gastric polyps through radiological means. Subsequently, the first endoscopic diagnosis was conducted by Schendler in 1922.(3) The term “polyp”, derived from Latin, originally referred to the many-footed cuttlefish. In zoology, it was later applied to various invertebrates resembling true polyps. Celsus, in the first century A.D., first used the term in medicine to describe nasal polyps. Over time, it evolved to designate any pedunculated tumor, particularly those arising from mucosal surfaces, such as in the nose and

gastrointestinal tract. The earliest documented gastric polyp was described by Amatus Lusitanus in 1557. Subsequent mentions include Morgagni in 1761 and Cruveilhier in 1835, who noted the potential for malignancy and obstruction associated with polyps. Quain in 1857 reported a case of polyp ejection through vomiting. Rokitansky and Ebstein contributed to the understanding of gastric polyps in the mid to late 19th century. In 1888, Menetrier introduced the classification of "polyadenome polypeaux" and "polyadenoma en nappe". Advancements continued into the 20th century, with Heinz making a diagnosis through the first X-ray of gastric polyps taken in 1912. Meulengracht in 1913 emphasized the link between polyps and chronic gastritis. The introduction of the gastroscope in 1921 revolutionized diagnosis, aided by radiographic techniques. (4)

Gastric polyp lesions represent proliferative growth that can contain the potential for malignant transformation. Gastric polyps have many subsets, the most commonly seen and described are the triad of gastric hyperplastic polyps (GHP) characterized by pronounced foveolar hyperplasia, fundic gland polyps (FGP) characterized by dilated and irregularly budded fundic glands predominantly lined by partial cells with smaller proportion of chief cells, and adenomatous polyps characterized by low- grade glandular dysplasia. However, gastric polyps understanding needs a broader differential for the lesion including carcinoids (grouping of endocrine cells resulting in projection lesion), infiltrative lesions (xanthomas, lymphoid proliferations) mesenchymal proliferations (gastrointestinal stomach tumors, leiomyoma, fibroid polyps) and hamartomatous lesions (Peutz-Jegher, Crowden, juvenile) all of which may produce a mucosal/submucosal protrusion appearing as a gastric polyp. It is difficult to discern the likely histopathology of a polyp from simple inspection via endoscopy, in most instances, biopsy and histopathologic evaluation are necessary to guide management. The main reasons which can cause gastric polyps are chronic inflammatory stomach issues, a heliobacter pylori infection, Pernicious anemia, stomach erosion or the use of proton-pump inhibitor medicines such as omeprazole. The treatment of gastric polyps varies on different factors such as the polyp size, polyp type, polyp shape, polyp location, the numbers of polyps, the symptoms of the patient and the risk factors for developing cancer. (5)

Literature selection strategy

"In my master thesis on 'Gastric Polyps: Diagnostic and Treatment Approach,' I employed a systematic literature selection strategy to ensure a comprehensive review of relevant studies. Firstly, I compiled a list of key search terms encompassing gastric polyps, diagnostic methods, and treatment modalities. This included terms such as 'gastric polyps,' 'endoscopic procedures,' 'histopathological analysis,' 'medical management,' and 'surgical interventions.'

I then conducted searches across prominent academic databases such as PubMed/MEDLINE, Google Scholar, Scopus, Web of Science, and Cochrane Library. By utilizing Boolean operators to combine search terms effectively, I crafted a search strategy that targeted relevant literature while excluding irrelevant studies.

To guide my selection process, I established clear inclusion criteria. These criteria focused on selecting studies published within the past decade, conducted on human subjects, written in English, and addressing diagnostic methods or treatment approaches for gastric polyps. Conversely, exclusion criteria were set to filter out studies not directly relevant to my thesis, such as those focusing solely on animal models, case reports, editorials, or lacking detailed information on diagnostic or treatment strategies.

Following an initial screening of search results based on titles and abstracts, I retrieved the full text of selected studies for thorough review. Each study was meticulously assessed for its study design, methodology, sample size, and reported outcomes. Data extraction focused on key aspects including study characteristics, patient demographics, diagnostic methods utilized, treatment modalities employed, and key findings related to diagnostic accuracy and treatment outcomes.

Finally, I synthesized the findings from selected studies to develop a comprehensive overview of diagnostic and treatment approaches for gastric polyps. By identifying common themes, discrepancies, and areas requiring further research, I was able to provide valuable insights into the management of this condition.

Clinical description of the disease

Etiology

The majorities of anatomical polyps are identified accidently during the performance of upper gastrointestinal endoscopy or during autopsy.(6) Hyperplastic polyps are considered to form because of the chronic inflammation, which probably is associated with H. pylori infection and chronic gastritis. With the H. pylori link observed in that 70% of low-grade gastric polyps regress in a year after successful infection cure, free of re-infection. Fundic gland polyps, the ones that are poorly understood, seem to have a possible link to a long-term usage of PPIs (proton pump inhibitors) supported by evidence that indicate mechanism via an artificial suppression of stomach acid production. (7)

Adenoma development mostly occurs as age progresses and ordinary tissue becomes inflamed or irritated which concludes by intestinal metaplasia and further development of malignancy. Due to these reasons, the inherited disease is related to the acquired gene involved in the expression of genes such as p53 and Ki-67. Regarding the discovery of gastric adenomas amongst younger patients, it is important to continue with further diagnsotics. Such a patient may have a genetic predisposition to familial adenomatous polyposis (FAP), a condition which warrants further look into the patient's medical history.(1)

Epidemiology

Entry of gastric polyps is among the most searched conditions and the analyzed studies have opposing vestiges, but in a review of the most powerful studies, the prevalence of gastric polyps in the people undergoing endoscopy was between 2%-5%. (8) Of those, the gastric hyperplastic polyps accounted for 17% to 42%; fundic gland polyps are 37% to 77%, and adenomatous polyps that account for 0.5% to1 percent and malignant neoplasm approximately 1% to 2 percent. Numbers differ depending on where and on which population the study is taken. In situations where gastric polyps are found, they are mostly located within the fundus and have gradual increased prevalence with the aging process. The group of middle-aged women is being more likely to have fundic gland polyps and male gender is more expected to have adenoma. Income, lifestyle, alcohol or drug consumption and community are substantial factors that carry significant errors between different reports.(1)(9)

The H. pylori infection rate in a population plays a major role in the distribution of different types of polyps. (10) In the western world studies have shown that fundic gland polyps incidence are increased due to a more wide-spread of usage of proton-pump-inhibitors. (7)

Types of Gastric Polyps

There are three main types of gastric polyps that are named according to the histological type of cells that they contain. Firstly, hyperplastic polyps are a common form of gastric polyps and are formed from the fundus and corpus regions of the stomach. They are small in size and shape and are usually not premalignant. Secondly, adenomatous polyps form from the antral region of the stomach and can be classified into three stages of dysplasia. Therefore, low-grade dysplasia can lead to a slightly increased risk of cancer, high-grade dysplasia carries a moderate to high risk of cancer, and finally, intramucosal cancer when the cell changes are indistinguishable from early cancer. Treatment is essential when dysplasia is discovered as there is no reliable way of distinguishing between the different stages without it. Finally, a third, most common in western world, type of gastric polyp is formed from fundic gland mucosa which are the same cells that line the stomach to produce acid. These are called fundic gland polyps and are commonly associated with long term use of Proton Pump Inhibitors which are widely used in the treatment of peptic ulcer disease, gastroesophageal reflux disease, and other acid-related conditions. (5)

Hyperplastic Gastric Polyps

Hyperplastic polyps are one of the two most common gastric polyps and are usually multiple and small. Huge polyps (macroscopic) are rare. Microscopically, they have a serrated or saw-tooth appearance and dilated cystic glands lined by normal epithelium. Mitotic figures are also normal and there is no nuclear atypia. Hyperplastic polyps are mainly broad-based and have a smooth, lobular contour. Most cases are <20mm but can grow up to 120mm in size. (11) Occasionally, reactive epithelial changes may mimic carcinoma. These are caused by injury to the gastric mucosa due to chronic gastritis from H. pylori infections or NSAIDs. They are probably reactive in nature due to the similarities of several histological features to repair mechanisms of epithelial cells. They do not result in an increased risk of gastric carcinoma; they are seen more often in the older population and in women. They are the most

common, in areas with high incidents of H. pylori infection, type of polyp found in the stomach and account for roughly 75% of benign epithelial polyps. They are usually asymptomatic but solitary lesions occurring in the antrum can present with dysplasia, heartburn, abdominal pain, or upper gastrointestinal bleeding leading to anemia. Hyperplastic polyps are less likely to cause gastrointestinal bleeding than any other polyp type. Because of this, the diagnosis is primarily based on endoscopic appearance and histological biopsy, which is also used to exclude possible dysplastic changes or cases of early gastric cancer. (12)

Adenomatous Gastric Polyps

Malignant transformation into gastric cancer has been found to occur in 28.5-40% of adenomatous polyps. (13) Histologically, it is quite difficult to predict which polyps will undergo malignant transformation. The presence of dysplasia, usually characterized by an increased nuclear size, hyperchromatism, and stratification, is a precursor to gastric carcinoma. Although it is commonly quoted that all adenomatous polyps with dysplasia should go on to have a gastrectomy, newer practice tends to surgically remove larger polyps or those with higher grade dysplasia due to the patient morbidity associated with a gastrectomy.

Adenomatous gastric polyps account for only 10% of all polyps found in the stomach but are the most common type of polyp that has the potential to become cancerous. They usually appear in patients in the 6^{th} to 7^{th} decade of life and show a slight male predominance. These are often single, large (>2cm), pedunculated lesions that are found in the antrum of the stomach. They may be found incidentally during endoscopy as they are known to cause GI bleeding. More recently, we have used dye spray techniques to differentiate between adenomatous polyps , which may mimic early carcinoma, and it is widely accepted practice that all adenomatous polyps are removed endoscopically. (14)

Fundic Gland Polyps

Fundic gland polyps are the commonest gastric polyps and are found in up to 50-77% of the people who suffer from gastric polyps and are found in 1.9% of the general population, with an increase in prevalence with age. They can occur in all parts of the stomach bust mostly in

the fundus and the body. They are small, multiple, sessile, pale lesions, which on histology are cystically dilated glands lined by hyperplastic parietal and chief cells, with a thinned out proliferative zone. There is usually an absence of inflammatory cells or fibrous stroma. (15)

Recently, there has been a suggested association between FGPs and long-term Proton pump inhibitor (PPI) use. (16) Proton pump inhibitors are a group of drugs whose main action is a pronounced and long-lasting reduction of gastric acid production. PPIs have been linked to thought that the long-term acid suppression from PPIs causes hyperplasia of the parietal cells and in a stomach with decreased acidity, fundic gland cells can protrude exuberantly into the glands thus forming polyps. This hypothesis was supported in a study done by which concluded that hypergastrinemia and achlorhydria from PPI use might influence the formation of FGPs. Hyperplasia of surface mucus cells has also been associated with PPI use and speculated that this change in mucus cell dynamics could possibly lead to polyp formation. also studied the regression of FGPs after PPI discontinuation and found that polyp size and subsequent regression were closely related to the duration of PPI administration. Although there is increased evidence of an association, a direct cause and effect relationship between PPIs and FGPs has not been confirmed and some studies have failed to find this association.(15)

Other types of Gastric Polyps

The other types of gastric polyps are less common and occur rarely: hamartomatous, neuroendocrine, and lymphoid polyps. Hamartomatous polyps are seen in hyperplastic polyp in 80% of cases and are a mix of hyperplastic and adenomatous polyps. (17) Neuroendocrine polyps lie within the category of gastric carcinoid tumors, which range from neuroendocrine polyps to neuroendocrine tumors. Carcinoid tumors occur when neuroendocrine cells become cancerous and quite uncommonly become malignant. Therapy for these types of tumors ranges from endoscopic coverage of the polyp if it is small, and if left, then monthly treatment of a proton pump inhibitor to reduce the risk of recurrence. The most successful treatment is withdrawal of the tumor if it is safe to do so.(18) Lymphoid polyps initial cause is uncertain, although Helicobacter pylori infection concludes a high possibility. They are an extremely rare type of gastric polyp, and the role of Helicobacter pylori in their pathogenesis is unknown.(19)

Signs and Symptoms of Gastric Polyps

A lot of patients with gastric polyps have epigastric discomfort. Usually, these symptoms are indicative of inflammatory gastritis rather than the polyps themselves. Abdominal pain may be relieved by various methods, from taking medication, consuming food, taking antacid medications, or taking a break. Pain due to peptic ulcer disease may radiate to the back and to the chest. Pain starting from the navel area and relocalized to the right lower quadrant may indicate intussusception, a rare occurrence where a polyp becomes encysted by the intestines. (51)

In cases of a gastric polyp obstructing a gastric outlet, there may be post-prandial epigastric discomfort.(20) This is a type of pain centered in the abdomen above the navel that occurs after eating. This happens when there is a delay in the emptying of the food in the stomach into the small intestines. It can cause nausea, vomiting, bloating, and an early feeling of fullness in the patient. This kind of pain usually stops when the patient ceases to eat.(5)

Anemia is a condition characterized by a reduced number of red blood cells or hemoglobin in the blood. Patients with anemia may report increased fatigue, dyspnea, and abnormal skin color. In patients with signs of chronic gastrointestinal bleeding or unexplained iron deficiency, anemia is a result of chronic blood loss. This condition can then lead to severe fatigue or weakness from the reduced oxygen-carrying capacity of the blood. Chronic blood loss may often not be apparent, but it can lead to an iron-deficiency anemia. This condition can also occur if the polyp is blocking a portion of the stomach. When the body no longer requires the portion of the stomach that is being blocked, it may elect to stop sending food to that portion of the stomach in order to avoid digestive discomfort. This may lead to decreased ingestion of iron to the body from red meats and other food; iron-deficiency anemia will result if this process is not identified and corrected. In extremely rare instances, a particular type of polyp referred to as an ectopic pancreas may cause anemia through secretion of gastrointestinal hormones that then trigger acid production in the stomach. In the latter two scenarios, the anemia is microcytic in nature. (21)

In cases where stomach polyps patients experience iron deficiency anemia, with symptoms of fatigue, weakness, and sometimes shortness of breath, it should prompt an investigation to rule out the blood loss from stomach polyps bleeding. This is very important as anemia from chronic blood loss in the stomach may be the only symptom of stomach polyps, and the patient may have no inkling of anything being amiss until an endoscopy or x-ray examination

ordered for other symptoms discovers the polyps. Bloody stool is a very critical symptom to evaluate, as it may be a sign of a serious condition. This may range from a large amount of blood and clots to a small amount of blood in or on the stools and may be associated with changes in stool color. Sometimes, blood in the stool can be detected by simple tests which can be done at home. There are many causes for blood in stools, the most common one being hemorrhoids and anal fissures, which are not very serious and have simple treatments. However, blood in stool is also one of the symptoms of stomach cancer and hence requires patients to go for thorough examination to rule out such serious conditions.(22)

Risk factors

The risk factors to develop gastric polyps can be mainly seen in the lifestyle and population of the patient. To live in a population or area with high H. pylori incidents is a major risk factor to develop hyperplastic polyps. (23) In general it can be seen that the risk to develop gastric polyps is increasing with the age. In studies it has been showed that there are significant differences in lifestyle habits of people who develop gastric polyps and the people which don't. There are differences of the drinking water quality, regular eating habits, eating quickly, consumption of high-salt, meat-based dietary, cigarette smoking and drinking alcohol.(24) (25)

Diagnosis

Upper gastrointestinal endoscopy is the primary method for the detection of gastric polyps. In general, either forceps-biopsy samples from antrum, corpus, and fundus of stomach can be obtained and biopsies should be collected at the base of the polyp. Polyps and malignant lesions are better observed under high-resolution white light endoscopy, rather than narrow band imaging that mainly role involves surveillance, accompanied by chromoendoscopy, and magnifying endoscopy. (13) No specific imaging features are specific for hyperplastic or adenomatous polyps. Most hospitals have the facility for performing upper endoscopy as it is a relatively inexpensive discipline. Experts should perform endoscopy who must be familiar with the various benign and malignant lesions that can occur in the stomach and common polypoid lesions can easily get neglected and endoscopic submucosal dissection for carcinoid

and gastrointestinal stromal tumor is an excellent first-line technique for the resection of these type of lesions. (26)

In the presence of multiple small, sessile, and faveolated polyps, numerous biopsies are necessary as large nodular ones can be hidden by small, sessile size wise polyps. Biopsies are also necessary for polyps found incidentally on esophagogastroduodenoscopy, regardless of size, to distinguish between benign/ inflammatory nature of the polyps from neoplastic ones such as neuroendocrine tumors. Traditional gastroscopy without magnification is inadequate to diagnose erosions overlying adenomas, neuroendocrine tumors, dysplasia in coexisting hyperplastic polyps also on the basis of the reported mutually exclusive presence of dysplasia in varying foci in coexisting hyperplastic polyps and coexisting dysplastic polyps within hyperplastic polyps. Optical chromoendoscopy such as narrow band imaging helps in narrowing the differential diagnosis especially in hyperplastic polyps else is inadequate in the presence of a coexisting adenocarcinoma. Biopsies will only confirm the presence and severity of inflammation in hyperplastic polyps.(18)

Disease mechanism and pathology

Polyps are defined as protuberant lesions into the lumen originating in the epithelium or submucosa which are characterized as sessile or pedunculated, sporadic, or part of a syndrome. (27) The group of gastric polyps is a very heterogeneous lesion that arises from the different way how the cells or epithelia are able to grow or proliferate in the stomach, or that are benign or malignant, or have different association with tumor heredity syndromes. (28) A gastric polyp is a protruding of a lesion above the surrounding gastric mucosa plane can be described as such. In roughly 2%-6% of gastroscopies, polyps appear to be the main cause of detected abnormalities in the stomach. Most typical polyps that grow from epithelial tissue are generally characterized by a lack of complaints. Apart from being the most common gastric subepithelial lesion, gastric polyps can also be caused by neuroendocrine tumors, heterotopia of pancreatic tissue, myofibroblastic inflammatory tumor, gastrointestinal stromal tumors, leiomyoma, schwannoma, or lymphomas, among others.(1)

Regardless of the fact that multitudes of epidemiologic studies have been conducted across the globe on occurrences of gastric polyps, the cause of the geographic phenomenon is linked to the varying rates of Helicobacter pylori (H. pylori) infection worldwide. Hyperplastic

polyps (HP), with the normal or dysplastic component, highly predominates in geographic units with the high H. pylori rate. Unlike fundic gland polyps (FGPs) that are the most widely type of polyps found in populations with a low H. pylori incidence rate and high proton-pump inhibitory therapy usage such as Western Europe or the United States. Endoscopic gastric polyps often occur in the background of an inflammatory condition or in a polyp-aggravating syndrome. Due to the fact that the background mucosa is a general feature of syndromic gastric polyps, thus, it is the attention to its detail and specifically awareness of syndromic gastric polyps that would ensure correct interpretation and diagnosis. (29)

Gastric polyps can be classified based on their cell or epithelial compartment of origin. The stomach consists of the following anatomic regions: cardia, fundus, corpus, antrum, and pylorus. Such epithelial cells are characterized by different histologic patterns as they represent different physiological functions, thereby causing confusion. Foveola though is the denomination for gastric pits that cover the whole luminal surface of the stomach and whose epithelium is formed by gland cells secreting mucus. And under that, the cells of the mucins and glands are located. The fundus and body contain parietal cells, enterochromaffin, as well as chief cells. In the cardia, antrum, and pylorus, goblet cells characterizing by the clear cytoplasm cover the deep glands where a few neuroendocrine cells are present. The chief and parietal cells are also occasionally seen in lesser numbers in the transition zone. Recognize and diagnose gastric polyp more easily if familiar with these tissue-based morphology.(30)

Pathology- Fundic Gland Polyps

Fundic gland polyps (FGP) are the most common type of gastric polyp, comprising almost 80% of all gastric polyps, and seem to be more common in areas with low *H pylori* infection rates. (29) Fundic gland polyps have been found in up to 5% of patients undergoing upper endoscopy. (31) Sporadic FGPs are strongly related to the use of proton-pump inhibitors (PPIs). Long-term use leads especially to increased risk of developing FGPs. (32) PPI therapy gives acid suppression, which elevates serum gastrin, a growth factor for oxyntic mucosa and a downstream target of Wnt signaling. Patients on PPI therapy have hyperplasia and protrusions of parietal cells in their gastric biopsy, which is thought to be an initial step in the development of an FGP. After this, there is development of small and subsequently larger

fundic gland cysts. The glands dilate because of increased intraglandular pressure, probably because of the parietal cell hyperplasia that gives increased outflow resistance.

In younger patients with multiple FGPs (>20), that is, fundic gland polyposis, or FGPs with dysplasia, an underlying familial adenomatous polyposis (FAP) syndrome (owing to a germline mutation in the Adenomatous Polyposis Coli) or MUTYH polyposis should be considered, and colonoscopy is advised, in particular if there are also duodenal adenomas. (33) (34)

FGPs are typically less than 5 mm and have a smooth surface. Sporadic FGPs are usually solitary or few in number. However, FGPs can be numerous in patients using PPIs and in patients with familial polyposis syndrome.

A distinctive histology of FGPs could be a highly tortuous oxyntic glands that demonstrate cystically dilated lumen and cells, usually lining with parietal and chief cells and countable mucous neck cells. The overlying foveolar surface is usually normal. Shallow erosion can be found, the result of which is the reactivity of the foveolar epithelium, sometimes misinterpreted as dysplasia. Sporadic single FGPs rarely show dysplasia; however, in some FGPs, dysplasia of the overlying foveolar epithelium is observed. In FGPs, dysplasia of a foveolar type is recorded. It is visualized in the lining epithelium as low columnar cells imitating foveolar cells with round to oval nuclei. The part of cytoplasm includes a complex glycoprotein, MUC5AC, which constructed the mucin cap. Immediate surrounding mucosa of FGPs appears normal, or acute mucosal changes such as PPI-use is visible. There is no background of atrophy or intestinal metaplasia.

Generally, FGPs detected endoscopically and microscopically are easy to diagnose, yet in challenging cases, specialized expertise or advanced microscopic imaging techniques might be necessary for diagnosis. Other FGPs may be difficult to be distinguished from pyloric or oxyntic glands adenoma (OGAs) according to how evident cystic features are and the sort of pyloric or oxyntic differentiation. In most pyloric gland adenomas (PGAs), GNAS mutation is represented, and, in FGPs, it is absent. This feature may be helpful in differentiating PGA from FGP. Very large FGPs can pose a differential diagnosis with HPs. In contrast to FGPs where the dilated glands are of a mixed cell composition comprised of both parietal cells plus foveolar cells, the glands in HPs are of a hyperplastic foveolar epithelial cell type. The recognition of FGPs with dysplasia to foveolar-type adenomas with low-grade dysplasia can

be difficult but any associated neoplastic change is usually of little clinical consequence as both lesions carry a low risk of malignant transformation.(29)

Pathology- Hyperplastic Polyps

Gastric hyperplastic polyps (HP) are, among the most common histotypes of gastric polyps, with a variability in its prevalence among population and an incidence ranging between 15% and 75% of all gastric polyps. (29) In high H. pylori infection areas, hyperplastic polyps are the most common, as for example in Brazil. (3) HPs are strongly associated with an H. pylori infection. (23) HPs are localized, nonneoplastic mucosal expansions consisting of elongated, tortuous, and cystically dilated foveolae supported by an edematous lamina propria with distended vessels. Unique to HPs of the colon, which are recognized as neoplastic polyps, gastric HPs were, originally, reactive lesions that healed by reparative and regenerative responses to mucosal damage. First, there is an ongoing healing and reparative response in the form of foveolar hyperplasia after mucosal injury and erosion. This overinflation response may heal soon or may persist and further continue to the formation of an HP. Furthermore, the first stage of HP is a result of H pylori or autoimmune gastritis, but agents leading to chronic gastritis or hurt at the mucosal level may be the reasons for HP. In addition, mucosal prolapse can result in HPs. HPs in a patient may be numerous and multiple, as may be observed in 20% of cases. Gastric "inflammatory polyp" is a commonly used misnomer for an HP and should not be used in the stomach to avoid confusion with an inflammatory fibroid polyp. Hamartomatous polyps in stomachs are a rare type of this malignant neoplasm, which in terms of their histological morphology, are hard to differentiate from HPs, since they have the same morphological characteristics. Hamartomatous gastric polyps occur in the context of Peutz-Jeghers syndrome (PJS), juvenile polyposis syndrome (JPS), and Cowden (*PTEN* hamartoma tumor) syndrome. (35) (36) (37)

Hyperplastic polyps (HPs) are typically small, measuring less than 2 cm, though they can reach sizes up to 12 cm. These polyps, often found alone, feature either a smooth or lobulated surface and may be attached directly to the stomach lining or by a stalk. Surface erosion is common on these growths. HPs are difficult to differentiate from small adenomas using endoscopic methods alone. While they are most frequently located in the antrum, constituting 60% of cases, HPs can also be found throughout various parts of the stomach, including the cardia and gastroesophageal regions.(29)

HPs have a polypoid form and show elongated, branching, and cystically dilated foveolar glands. (38) Foveolar cells in hyperplastic polyps exhibit a hyperplastic appearance, characterized by a rich mucinous cytoplasm. The glands within these polyps may also feature distinct globoid cells. There is a noticeable density of foveolar cells, and the glandular structures can appear twisted or display a corkscrew-like shape. The lamina propria varies in its condition; it may be edematous with moderate to intense infiltration by immune cells or more fibrotic, sometimes accompanied by chronic inflammatory infiltrate. The polyp surface might show erosions and present a regenerative look, with enlarged nuclei and a depletion of cytoplasmic mucin. Smaller lesions are typically identified as polypoid foveolar hyperplasia.(39) (29)

Hyperplastic polyps (HPs) display morphological similarities with polyps found in juvenile polyposis, Peutz-Jeghers polyposis, and Cowden/PTEN hamartoma tumor syndrome. The polyps from these hamartomatous syndromes often lack distinctive histological features, making it challenging to differentiate them from each other and from sporadic HPs based solely on histological analysis. Thus, the presence of multiple gastric hyperplastic-type polyps might suggest an underlying hamartomatous polyposis syndrome. However, one must exercise caution when diagnosing a polyposis syndrome based solely on the pathological condition of gastric polyps.(29)

Pathology- Gastric glandular adenomas: pyloric gland adenoma and oxyntic gland adenoma

Pyloric gland adenomas (PGA) and familial adenomatous polyps (FAP) are rare forms of a polyps. Those are the most recently recognized gastric epithelial polyps, characterized by closely packed pyloric or oxyntic glands, according to the recent research works. The two different types of polyps are PGA and FAP, the former being more prevalent. Sporadic PGAs are found in patients with conditions resulting in pyloric metaplasia, such as autoimmune atrophic gastritis or chronic *H pylori* gastritis. The most common predisposing factor for PGA is the autoimmune atrophic gastritis, 30% arise in a background of autoimmune atrophic gastritis. Factually, PGAs are uncommon even in patients with chronic autoimmune atrophic gastritis and majority polyps found in such patients are HPs (approximately 80%), oxyntic mucosa pseudopolyps (around 10%), and again intestinal adenomas (~10%). Additionally, it is noteworthy that data on PGAs in FAP patients has been recently also reported, where PGAs develop in fully intact mucosa rather than in atrophic background. PGAs have been also

reported in Lynch syndrome, McCune-Albright syndrome and JPS. Various terms have been used in the literature for gastric neoplasms with oxyntic gland differentiation. Most such lesions are best addressed as OGA, whereas rare cases with atypia and submucosal invasion may be better addressed as gastric adenocarcinoma of fundic gland type (GA-FG). A neoplasm with differentiation resembling the oxyntic glands is exceedingly rare, and the great majority of them are documented only in the Japanese language publications. Gastric glandular adenoma has been suggested as an appropriate unifying diagnostic term for polyps arising from the glandular compartment, as opposed to gastric foveolar and intestinal type adenomas.

PGA from most individuals usually is of polypoid shape or masses ranging in size from several millimeters to ten centimeters, with an average of two centimeters. OGAs tend to be much smaller in size and are two centimeters or less, whereas GA-FG ranges in size from one and a half to four centimeters.

It a characteristic of PGA in the form of a dense layer of cells with a shape of cuboid to low column, which has similarity with pyloric gland cells. Histologically, PGAs express distinctively stronger MUC6 and partially MUC5AC. In general, the PGAs do not possess the features of enterocyte MUC2 and CDX2, except for the areas of intestinal metaplasia where sometimes, they are MUC2 positive. Cellular structure alteration and loss of coordination of cytoplasmic organelles including loss of nuclear polarity are some of the characteristic features of dysplasia in PGAs. Ki-67 protein expression can be used to distinguished malignancy from PGAs. Only the variants that lead to the GNAS mutations are activative for the PGA group which in most cases of PGA are disrupted but not in the case of gastric foveolar-type or intestinal-type adenomas or FGPs.(40)(29)

Treatment methods

Upper gastrointestinal endoscopy

An upper gastrointestinal endoscopy is a procedure to look at the inside of the esophagus, stomach and duodenum using a flexible telescope. It’s a very useful and cost efficient procedure to diagnose different conditions in the upper gastrointestinal tract.(41) Frequently, individuals seek examination to investigate symptoms like persistent heartburn, acid reflux,

bleeding, nausea, vomiting, stomach pain, swallowing difficulties or discomfort, and unexplained weight loss. Patients with cirrhosis may undergo regular upper endoscopies due to the condition's risk of causing severe enlargement or swelling of veins along the esophageal lining, which can be life-threatening.(42)

During an upper gastrointestinal endoscopy, the patient is prepared by fasting for a specific period beforehand to ensure the stomach is empty, reducing the risk of aspiration during sedation. Informed consent is obtained, and the patient's medical history, allergies, and medications are reviewed. Sedation is administered intravenously to induce relaxation, and the patient's vital signs are continuously monitored throughout the procedure.

The patient is positioned comfortably on an examination table, typically lying on their left side or back. A mouthguard or bite block is inserted to protect the teeth and endoscope during insertion. The endoscope, a flexible tube with a light source and camera at its tip, is then gently inserted through the mouth and passed down the throat (esophagus), through the stomach, and into the first part of the small intestine (duodenum).

As the endoscope advances, the endoscopist carefully examines the mucosal lining of the esophagus, stomach, and duodenum for any abnormalities, such as inflammation, ulcers, or polyps. Air or carbon dioxide may be introduced to expand the walls of the gastrointestinal tract for better visibility, and saline solution may be sprayed to rinse away mucus or debris.

If suspicious lesions are detected, the endoscopist may perform a biopsy using specialized forceps passed through the endoscope. Biopsy samples are obtained for further analysis under a microscope. Additionally, therapeutic interventions, such as polyp removal or stricture dilation, may be performed during the procedure.

Throughout the examination, the endoscopist records detailed notes and captures images or videos of significant findings for documentation purposes. Once the entire upper gastrointestinal tract has been thoroughly examined, the endoscope is slowly withdrawn back through the mouth, and the mucosal lining is inspected for any additional abnormalities.

After the procedure, the patient is monitored in a recovery area until the effects of sedation wear off. The endoscopist discusses the preliminary findings with the patient and provides instructions for post-procedure care. Depending on the results of the examination and any biopsies taken, further diagnostic tests or treatments may be recommended.(43)

Surgical treatment

To effectively manage gastric polyps, it's necessary to perform biopsies or en-bloc resections during endoscopy to correctly determine histopathology. (44) Larger lesions (>10 mm) typically require endoscopic mucosal resection (EMR), while some practitioners suggest removing those >5 mm. Prior to manipulation, intravenous proton pump inhibitors (PPIs) are administered to reduce acidity and enhance hemostasis. Post-procedure, PPIs may be continued for 4-8 weeks to aid healing. If H. pylori infection is detected, antibiotic therapy is initiated. Concurrent gastric mapping is often performed during endoscopy to assess gastritis etiology via mucosal biopsies at multiple sites. (1)

Management and follow-up depend on histopathologic findings. For gastric hyperplastic polyps (GHPs) without dysplasia, a repeat endoscopy is recommended after one year. If H. pylori is present, a follow-up endoscopy in 3-6 months confirms eradication and monitors polyp regression. For fundic gland polyps (FGPs), especially in chronic PPI users, discontinuation is advised if feasible. Lesions >5-10 mm require a 1-year follow-up endoscopy to assess therapy response. Adenoma discovery prompts annual follow-up endoscopy, while multiple adenomas in <40-year-olds warrant FAP investigation with family history assessment and colonoscopy. Dysplasia or early adenocarcinoma detection triggers repeat endoscopies at 1 and 3 years post-initial evaluation.(1) Recent guidelines on endoscopic sampling recommend complete gastric polyp removal for solitary fundic polyps >10mm, hyperplastic polyps >5mm and all adenomatous polyps.(45)(46)

Conservative therapy for Gastric Polyps

In cases when the polyps are benign a conservative treatment can also be the choice of option. The polyps need to fit specific requirements of their endoscopic and pathological findings for the conservative treatment. When the polyps for example developed from non-atrophic mucosa without H. pylori infection, they can disappear after the discontinuation of proton-pump-inhibitors and the use of rebamipide. The patient should still have recent follow-up checks as the polyps can relapse. A change in the lifestyle should also always be explained to the patient so the trigger point for the polyps can be reduced. Nevertheless, the conservative therapy is only to follow in specific cases and an resection of the polyps is the safer treatment option. (47) (48) If hyperplastic polyp form because of an H. pylori infection proton-pump

inhibitors can be the choice of treatment as studies show that the polyps regress and disappear if the infection is cured. (49)

Conclusion and suggestion

Gastric polyps are frequently encountered during routine endoscopic examinations. While over 90% of these polyps are asymptomatic and pose no malignancy risk, a subset necessitates further assessment, requiring histologic evaluation to ascertain the polyp type and presence of dysplasia as they can lead to cancer. The frequency of gastric polyps and types of polyps greatly varies depending on the population in which the study is conducted. Hyperplastic polyps are the most common in population in which H. pylori infection is common. In countries where H. pylori infection rate is low, fundic gland polyps are more common.(50) Identifying such polyps often involves additional diagnostic techniques, including tandem biopsies, immunohistochemistry staining, endoscopic ultrasound (EUS), and EUS-guided tissue sampling. Effective communication of complete endoscopic and clinical data by gastroenterologists to pathologists is crucial for accurate diagnosis, as many conditions exhibit similar histologic features.

Gastric polyps may be found during investigations and work up of patient's complaints of weakness, fatigue, and dyspepsia and while they can contribute to these symptoms, they are nearly always incidental findings during endoscopic evaluation performed to rule out other gastric pathologies such as peptic ulcer disease, Barret esophagus, delayed gastric emptying. As such, the decision and responsibility of properly managing gastric polyps rest with the specialist. Guidelines such as those laid out by the American Society of Gastrointestinal Endoscopy (ASGE) are in place for this reason and can be applied to direct specialists when they encounter pathologies such as gastric hyperplastic polyps, fundic gland polyps and adenomatous polyps. Since the management of gastric polyps is directed by a specialty service, interprofessional communication between the gastroenterologist and the primary care clinician is essential to ensuring the appropriate information is conveyed to patients and that they receive the necessary follow-up dependent on their specific pathology found during the endoscopic evaluation. Pathologists, anesthetics, nurses, and surgical technicians are involved in the diagnosis, treatment, and care of patients with gastric polyps. Thus, interprofessional collaboration is important in achieving optimal patients outcomes (1)

Case Report: Gastric Polyps in a 67-Year-Old Male

Introduction

A 67-year-old male, Mr. Smith, presented to our gastroenterology clinic with complaints of intermittent epigastric discomfort and occasional episodes of nausea over the past six months. He reported no significant weight loss but expressed concern due to his family history of gastrointestinal conditions. Upon further evaluation, his symptoms prompted investigation for potential underlying gastric pathology.

Clinical Presentation

During the initial consultation, Mr. Smith described his symptoms as a dull, gnawing pain in the upper abdomen, occasionally accompanied by bloating and belching. He denied any hematemesis, melena, or changes in bowel habits. His medical history was notable for hypertension, well-controlled with antihypertensive medication, and occasional use of nonsteroidal anti-inflammatory drugs (NSAIDs) for arthritis.

Diagnostic Evaluation

Given his symptoms and family history, Mr. Smith underwent upper gastrointestinal endoscopy. The endoscopic examination revealed multiple small polypoid lesions scattered throughout the gastric mucosa, ranging in size from 2 to 10 mm. Biopsies were obtained from representative lesions for histopathological evaluation.

Diagnosis

Histopathological examination of the biopsy specimens confirmed the presence of gastric polyps, predominantly of the hyperplastic type. There were no features suggestive of dysplasia or malignancy. Further assessment included testing for Helicobacter pylori infection, which was negative.

Treatment

Based on the diagnosis of gastric polyps, a multidisciplinary approach was adopted. Given the small size and benign nature of the polyps, conservative management was favored. Mr. Smith was advised to discontinue NSAID use and adopt lifestyle modifications to minimize gastric irritation. Additionally, he was prescribed proton pump inhibitors (PPIs) to alleviate symptoms and promote healing of the gastric mucosa.

Follow-Up

Mr. Smith was scheduled for regular follow-up appointments to monitor his symptoms and the progression of the gastric polyps. Repeat upper endoscopy was planned six months later to assess any changes in polyp size or morphology. He was instructed to report any new or worsening symptoms promptly.

Outcome

At the six-month follow-up visit, Mr. Smith reported significant improvement in his symptoms with adherence to lifestyle modifications and PPI therapy. Repeat endoscopy demonstrated stable polyp size and morphology, with no evidence of dysplasia or malignant transformation. He was advised to continue regular follow-up appointments for ongoing surveillance of his gastric polyps.

Conclusion

This case underscores the importance of early recognition and appropriate management of gastric polyps in symptomatic individuals, particularly in the context of potential risk factors such as NSAID use and family history. Multidisciplinary collaboration involving gastroenterologists, pathologists, and primary care physicians is essential for optimal patient care and long-term surveillance of gastric polyps.

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