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CKJ REVIEW

Nutcracker syndrome in 2026: a nephrologist-oriented diagnosis and management

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ABSTRACT

Nutcracker syndrome (NCS) refers to symptomatic compression of the left renal vein (LRV), most commonly between the aorta and superior mesenteric artery (anterior nutcracker) or, less frequently, between the aorta and vertebral column in the presence of a retro-aortic LRV (posterior nutcracker). This venous entrapment elevates renal venous pressure and promotes drainage through the gonadal and pelvic venous networks. Clinical manifestations typically include haematuria, left flank or abdominal pain, orthostatic proteinuria, and features of gonadal or pelvic venous congestion such as varicocele. Diagnosis relies on careful clinical evaluation, exclusion of alternative aetiologies, and targeted imaging modalities including Doppler ultrasonography, computed tomography, magnetic resonance angiography, venography, and intravascular ultrasound. Despite increasing recognition of NCS, standardized diagnostic criteria and imaging thresholds remain lacking. Management strategies range from conservative treatment to endovascular interventions and open or laparoscopic surgical procedures. Optimal care requires a multidisciplinary approach involving urology, radiology, nephrology, and vascular surgery to ensure accurate diagnosis and individualized treatment planning.

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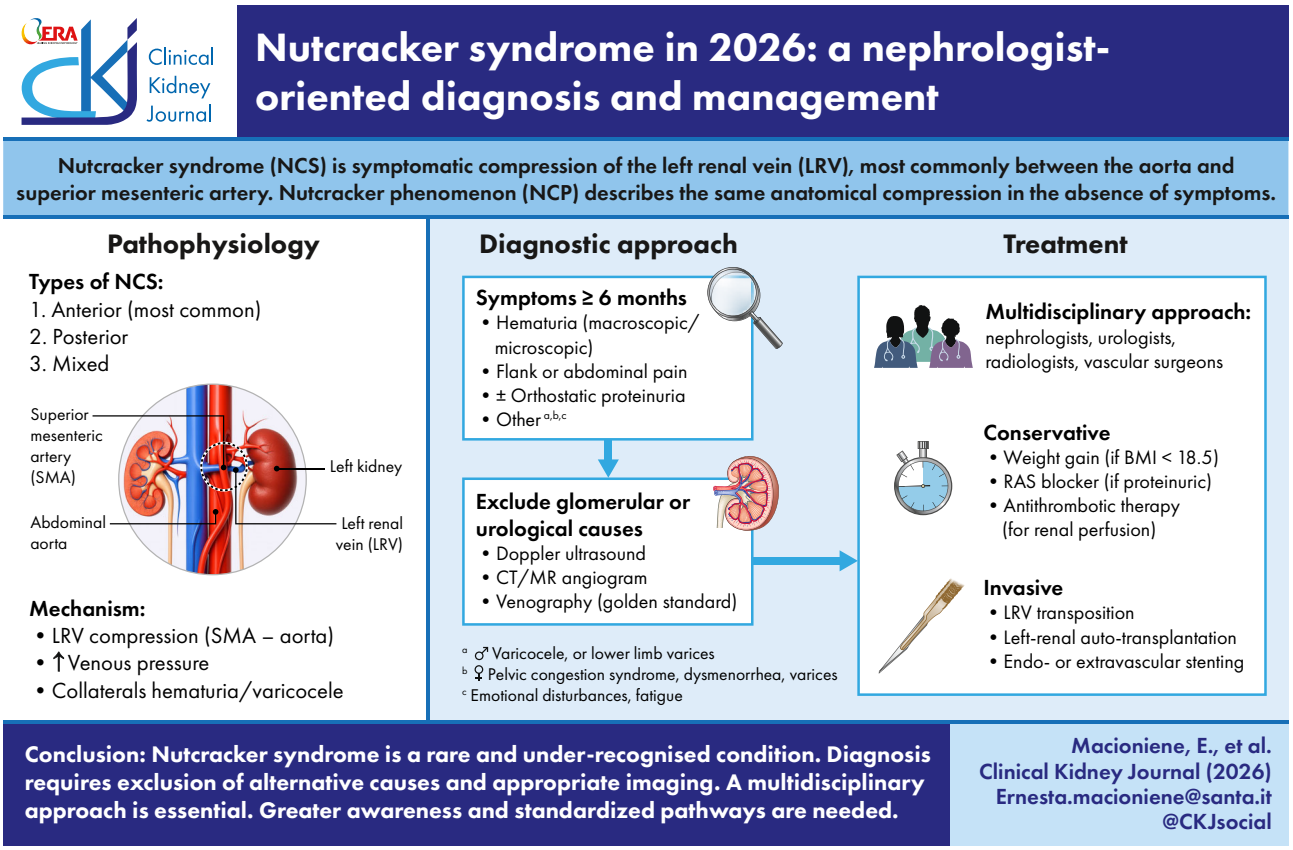
GRAPHICAL ABSTRACT



Keywords: left renal vein compression, nutcracker phenomenon, nutcracker syndrome

DEFINITION

The nutcracker phenomenon (NCP) and nutcracker syndrome (NCS) are two distinct entities. NCS refers to symptomatic compression of the left renal vein (LRV), most commonly between the aorta and superior mesenteric artery (anterior nutcracker) or, less frequently, between the aorta and vertebral column in the presence of a retro-aortic LRV (posterior nutcracker). The NCP describes the same anatomical compression in asymptomatic individuals [1, 2].

HISTORICAL CONTEXT

The LRV entrapment was first anatomically defined in 1937 by an anatomist J. Grant: 'the LRV, as it lies between the aorta and superior mesenteric artery, resembles a nut between the jaws of a nutcracker' [3]. Thirteen years later, El-Sadr and Mina described the clinical impact of LRV compression between the aorta and superior mesenteric artery [4]. In 1971, Chait and co-authors mentioned renal vein compression and its impact on the ureter indentation seen in radiology images, however the term 'nutcracker syndrome' was first used by Schepper 1 year later [5, 6].

PATHOPHYSIOLOGY OF NUTCRACKER SYNDROME

In most patients with NCP (~85%–90%), the vein is squeezed in its normal position between the aorta and the superior mesenteric artery (anterior nutcracker) (Fig. 1). Less commonly, compression occurs between the aorta and the spine when the LRV takes an abnormal retroaortic course (posterior nutcracker) (Fig. 2) [2, 7]. Rare forms involve an aberrant right renal artery (RRA) or a combination of mechanisms. The problem usually begins when the space between the aorta and superior mesenteric artery becomes too narrow. In healthy adults, the aortomesenteric angle is 38°–65° and the distance is 4–18 mm. When the angle falls below 25°–35° or the distance is ≤3–5 mm, the LRV can be significantly compressed [2, 7, 8]. This narrowing is more likely in people who are very thin (low retroperitoneal and mesenteric fat), have an exaggerated lumbar lordosis, a low-lying left kidney (renal ptosis), or experience rapid height gain in adolescence, pregnancy, or major weight loss.

HOW THE COMPRESSION AFFECTS BLOOD FLOW

High pressure in the renal veins backs up into the small vessels inside the kidney, even though arterial blood supply remains normal. This leads to congestion, reduced effective perfusion, and a state of relative renal ischemia.

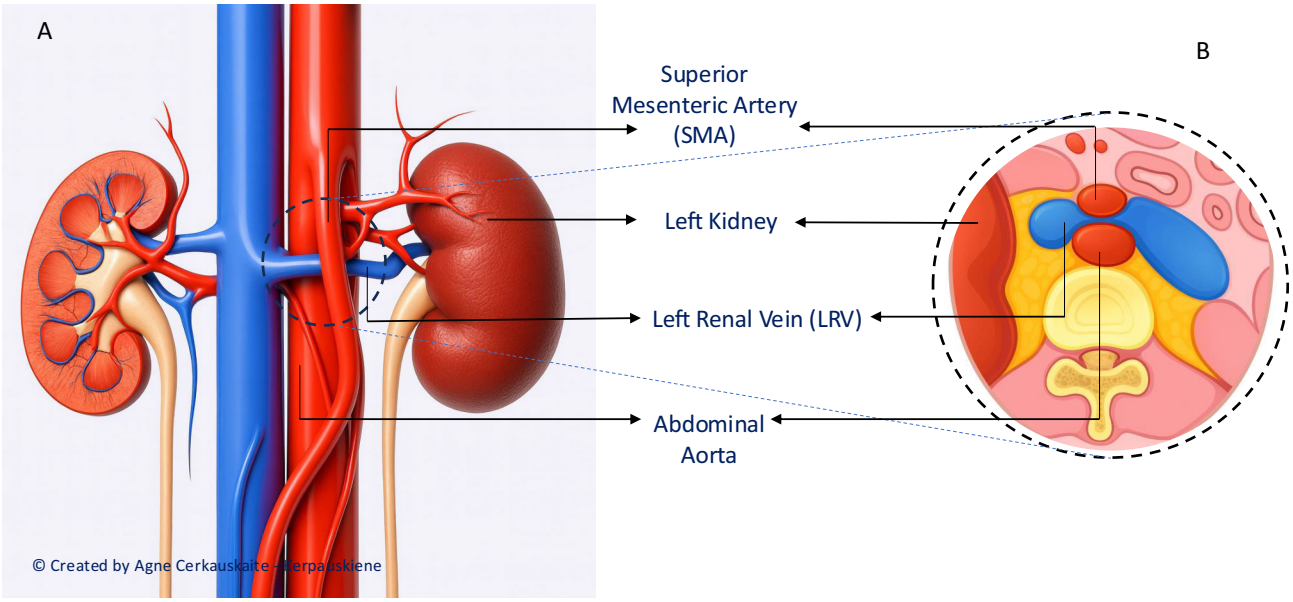


Figure 1: Pathophysiology of nutcracker syndrome (NCS). Schematic frontal (A) and axial (B) views of the abdominal vasculature illustrating anterior NCS. Narrowing of the fat-filled aortomesenteric space results in compression of the left renal vein between the abdominal aorta and the superior mesenteric artery, leading to renal venous hypertension and collateral circulation through the gonadal and pelvic veins.

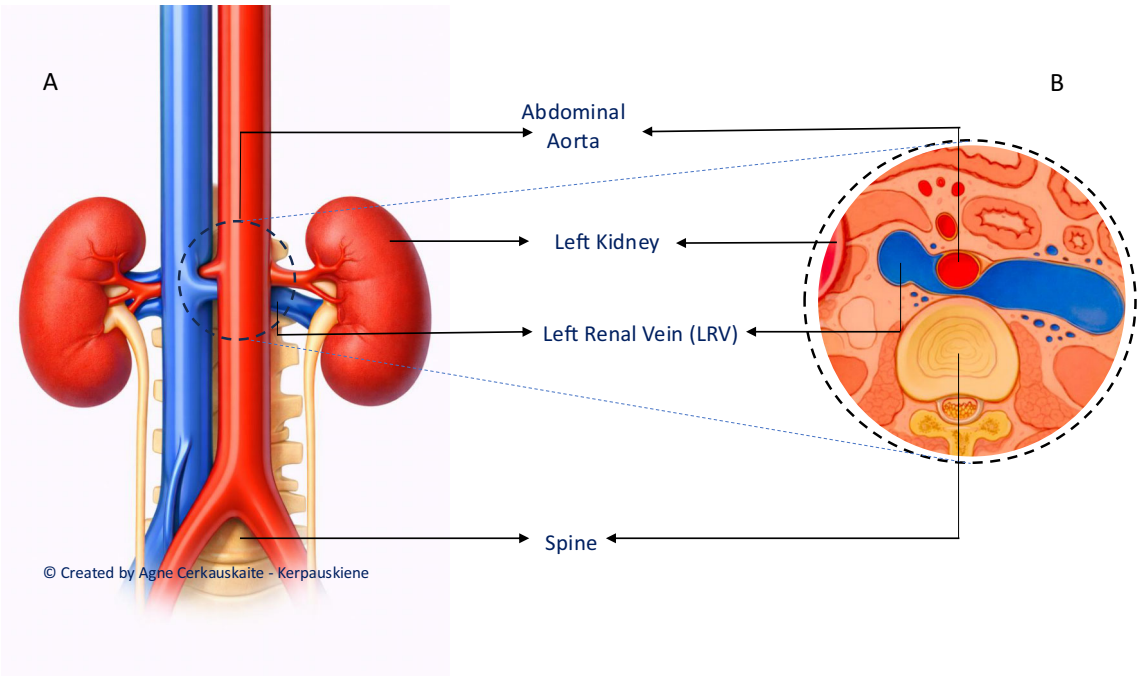


Figure 2: Pathophysiology of posterior nutcracker syndrome (NCS). Schematic frontal (A) and axial (B) views of the abdominal vasculature illustrating posterior NCS. In the presence of a retroaortic left renal vein, the vessel is compressed between the abdominal aorta and the vertebral column, leading to renal venous hypertension and collateral circulation through the gonadal and pelvic veins.

The squeezed vein creates a pressure difference between the LRV and the inferior vena cava. Normally this gradient is less than 1 mmHg; in symptomatic patients it is usually ≥3 mmHg (often 4–15 mmHg), and many experts now consider >2 mmHg abnormal [2, 9]. Computer simulations of blood flow (computational fluid dynamics) show that the tighter the compression, the faster blood rushes through the narrow segment, producing turbulence, high wall stress, and a marked rise in pressure upstream of the blockage [9, 10, 11]. The result is left renal venous hypertension with relative low pressure downstream in the vena cava.

However, well-developed collateral veins can partly relieve pressure on the kidney, explaining why many people with the same anatomical narrowing never develop symptoms (NCP).

EPIDEMIOLOGY

Although symptomatic NCS is considered rare, the asymptomatic NCP is increasingly recognized through advanced imaging modalities. Recent large imaging studies (e.g. computed tomography (CT) angiography series) have substantially refined our understanding of the prevalence and variability of the anatomical phenomenon.

In a cohort of 324 asymptomatic kidney-donor candidates evaluated with high-definition CT angiography, 30.5% demonstrated an aortomesenteric angle $<41^{\circ}$, 15.3% exhibited the 'beak sign', and 9.8% had a beak angle $\geq 32^{\circ}$, indicating some degree of the LRV compression. However, only 0.7% fulfilled the criterion of a high diameter ratio ($\geq$4.9), suggesting that severe compression consistent with hemodynamically significant NCP is uncommon [12]. Similarly, Grimm and co-authors reported that 23% of 99 potential renal transplant donors showed 50%–70% LRV stenosis on CT angiography [13]. In a more recent study, Sun *et al.* identified the RRA as an alternative compressive structure of the LRV, noting that RRA-induced compression may even surpass the classic SMA-aorta variant in frequency among certain populations [14]. These findings collectively suggest that the conventional anatomical definition of the 'nutcracker' configuration may warrant expansion.

The true prevalence of NCS remains poorly quantified. In a study of 1125 contrast-enhanced abdominal CT scans, a retroaortic LRV was observed in 1.6% of females and 1.7% of males [15]. Notably, 77% of affected males had a varicocele confirmed by Doppler ultrasonography, which is more consistent with NCS than with NCP. In a Korean kidney-referral cohort assessed via Doppler ultrasonography, NCP and NCS were diagnosed approximately 30% and 15%, respectively [16]. These findings imply that while NCS may reach a prevalence of up to 15% in selected clinical populations, its global population prevalence is unknown.

Regarding sex distribution, several studies report a female predominance in NCS, although this trend is not universal, and asymptomatic NCP does not appear to exhibit clear sex predilection [17–20]. Domenico *et al.* reported that most NCS cases were women aged under 50 [21]. Another study noted that males tended to be diagnosed younger (mean ~23.6 years) than females (mean ~29.3 years) [22]. Kuklinsky *et al.* mention that symptomatic patients are most often in their 20s–30s, with a possible secondary 'peak' among middle-aged women [2].

CLINICAL PRESENTATION OF THE NCS

The NCS characteristic clinical symptoms may vary between studies; however, the most common clinical presentation includes haematuria (ranging from 13% to 100% of patients), flank or abdominal pain, pelvic pain, and varicocele [7]. Other features, such as proteinuria, abnormal uterine bleeding, nausea, and anaemia, were also reported to be associated with NCS [7].

Haematuria develops when pressure causes tiny veins around the renal fornices to swell and eventually rupture into the collecting system. Haematuria is typically microscopic but may be macroscopic (gross) in some cases. On cystoscopy the bleeding is seen only from the left ureter, and the red blood cells have a normal shape (non-glomerular, isomorphic) [23, 24].

Orthostatic proteinuria and flank pain—two classic orthostatic symptoms often appear or worsen when the patient is upright. Standing narrows the aortomesenteric angle (because gravity pulls the intestines downward), which abruptly increases left renal venous pressure. This heightened venous hypertension is responsible for both symptoms. Flank pain is left-sided, dull or aching, caused by acute distension of the renal capsule and congestion of the renal parenchyma. It may be the dominant complaint in adolescents and adults [2, 23]. However, atypical right-sided symptoms, including flank pain, can occur due to altered venous drainage patterns, such as cross-filling through lumbar or renolumbar collaterals that propagate pressure or congestion to the right side [25]. Both symptoms typically improve or disappear when the patient lies down, reflecting the postural dependence of the venous compression.

Proteinuria develops due to the pressure surge stimulating renal baroreceptors and chemoreceptors, triggering local release of norepinephrine and angiotensin II. This leads to preferential efferent arteriolar vasoconstriction, a transient rise in glomerular capillary pressure, and reversible disruption of the filtration barrier. In many children and young adults, the orthostatic proteinuria resolves completely with ACE-inhibitor or angiotensin-receptor-blocker therapy [18, 24, 26].

Proteinuria and haematuria are common signs of immunoglobulin A (IgA) nephropathy—the most common primary glomerulopathy, therefore it can mimic the clinical characteristics of NCS [27, 28]. Several published case reports describe the coexistence of IgA nephropathy and NCS, with some noting resolution of haematuria after interventions that relieve renal vein compression [29, 30]. These findings suggest that patients with biopsy-proven IgA nephropathy might not undergo further investigation, leading to missed diagnoses of NCS in some cases.

Pelvic congestion syndrome (PCS) is another of the most frequent conditions associated with NCS, especially in women [23, 31, 32]. Retrograde transmission of elevated LRV pressure into the left ovarian vein leads to pelvic venous dilatation, varicosities, and chronic pelvic pain, usually accompanied by dyspareunia, dysmenorrhea, or postural symptom fluctuations [33, 34]. Physiological imaging studies have demonstrated that mesoaortic compression of the LRV can directly produce both outflow obstruction and reflux that are characteristic of PCS [2, 31].

In men, NCS frequently manifests as left-sided varicocele (20%–40% of cases) [31, 35, 36]. The elevated venous pressure within the vein is transmitted to the left gonadal vein and pampiniform plexus, resulting in dilated, tortuous scrotal veins [2, 35, 36]. Several studies highlighted that NCS should be considered in patients presenting with recurrent or atypical varicocele, particularly when accompanied by haematuria, flank pain, or imaging evidence of LRV entrapment [2, 35, 37]. Rarely, varicocele may be the sole clinical manifestation of NCS, underscoring the importance of including LRV compression in the diagnostic work-up [35–37].

NCS also is linked to ovarian vein dysfunction, including ovarian vein reflux syndrome [33, 38]. Persistent retrograde flow into the gonadal venous system can contribute not only to pelvic varicosities but also to vulvovaginal symptoms and other manifestations of chronic venous insufficiency [33, 38]. Radiologic case series have documented findings such as dilated ovarian, arcuate uterine, and pelvic varices in patients ultimately diagnosed with concurrent NCS, illustrating how gonadal venous involvement can guide targeted interventions such as embolization [33, 38].

Furthermore, NCS can coexist with other vascular compression syndromes [38, 39]. Wilkie syndrome (superior mesenteric artery syndrome) shares a similar anatomical basis—reduction of aortomesenteric angle—and has been reported alongside NCS, suggesting parallel compression phenomena within the same anatomical compartment [2, 39]. May-Thurner syndrome, median arcuate ligament syndrome, and other 'entrapment' disorders have also been described in association with NCS, form-

ing part of a broader constellation of venous compression syndromes [2, 39]. Such overlaps may intensify venous stasis, exacerbate symptoms, and complicate management strategies, necessitating a comprehensive vascular evaluation [39–41].

Secondary hypertension is a less common but important clinical sign. NCS can rarely be associated with increased blood pressure due to impaired sodium excretion ability of the kidney, renal ischemia and hypoxia secondary to elevated LRV pressure, and renal chemoreceptor and baroreceptor response to altered glomerular haemodynamics, inducing impulses to the hypothalamus to increase the release of vasoconstricting agents [42]. In 5%–15% of cases, the kidney senses low perfusion and releases renin, activating the renin–angiotensin system. Additional sympathetic nervous system activation can produce true, sometimes postural or treatment-resistant hypertension [2, 23].

Collectively, these associated conditions reflect the diverse downstream effects of LRV hypertension and collateral formation [21, 23]. Recognition of these relationships is essential, as the presence of PCS, varicocele, ovarian vein reflux, or overlapping vascular compression syndromes may be the primary driver of a patient's symptoms and may influence the need for renal vein-directed therapy, gonadal vein intervention, or combined medical approaches [41, 43, 44].

LONG-TERM OUTLOOK

The natural history and the exact prognosis of NCS remains unclear, as long-term, high-quality studies are limited [45]. In some patients—particularly children and adolescents—the condition may represent a transient phenomenon rather than a fixed syndrome [2, 23]. As body mass and mesenteric/retroperitoneal fat increase with growth, the aortomesenteric angle may widen and decrease compression of the LRV [2, 23], allowing conservative management for at least one or two years [46].

In adults, however, the course is more variable and often chronic, predisposing to LRV thrombosis and progressive kidney dysfunction [36]. However, true kidney failure due to NCS is exceptionally rare when nutcracker is the only problem [2, 23]. Massive haematuria, requiring transfusion, and significant proteinuria have been reported [2, 47]. Moreover, insufficient venous outflow from the left gonadal vein can cause venous congestion and dilatation, resulting in left-sided varicoceles in men and PCS in women, causing infertility [36]. Worsening of coexisting glomerular diseases such as IgA nephropathy was reported and correcting the venous obstruction dramatically improved proteinuria and haematuria [48]. Although life-threatening outcomes are rare, quality-of-life impairment can be significant [49]. The clinical trajectory is unpredictable, therefore prognosis must be individualized, and patients with significant or progressive symptoms may eventually require intervention [50].

In summary, NCS is best understood as a pathophysiological cascade in which mechanical entrapment of the LRV produces functional stenosis, sustained venous hypertension, renal parenchymal congestion, and a variety of downstream local and systemic effects. Modern imaging and modelling show that symptoms depend less on how narrow the vein looks on imaging and more on measurable disturbances of flow and pressure [10, 11]. This explains the wide variation in presentation and the occasional difficulty in deciding when treatment is truly needed.

DIAGNOSIS

The diagnosis of NCS is based on a stepwise approach that includes clinical suspicion (characteristic signs and symptoms) persisting for more than 6 months and an imaging test after excluding other potential causes (Table 1) [7]. Doppler ultrasound is non-invasive, simple and the first choice diagnostic tool for screening patients and supporting a NCS diagnosis. It has a reported sensitivity of 69%–90% and specificity of 89%–100% and allows measuring the blood flow velocity in the LRV [7, 44, 50]. A peak systolic velocity ratio ≥4.2–5.0 between the aorto-superior mesenteric and hilar LRV segments is considered a diagnostic criterion [50]. Despite advantages, Doppler ultrasound has limitations (results depend on patient positioning and technical aspects) and therefore should not be used as a single diagnostic test for NCS [7, 44, 50, 51]. For a more definitive diagnosis, cross-sectional contrast-enhanced imaging is required.

CT/magnetic resonance imaging (MRI) angiography enables the evaluation of LRV compression between the superior mesenteric angle and the aorta or between the aorta and the spine (Fig. 3) [7, 40, 52]. MRI angiography is comparable to CT angiography in evaluating the aorto-superior mesenteric artery angle; however, its clinical utility is limited by cost and long scanning time [51]. A normal angle between the aorta and superior mesenteric artery is considered between 45° and 90°, however there is no consensus that values below 30° definitively confirm the diagnosis [7, 23]. Although, many other diagnostic criteria and cut-off values are still widely used and may support the diagnosis, they also lack sufficient significance for diagnosis NCS [7, 44, 50–52].

Although imaging studies (CT/MRI, CT/MRI angiography) provide valuable information and help support the diagnosis in symptomatic patients, as well as exclude other potential causes and assist in surgical planning, more invasive contrast-enhanced testing is often required to objectively measure pressure gradients and confirm the diagnosis [52]. Venography and intravascular ultrasound in diagnosing NCS are considered by some authors to be the gold standard and confirmatory imaging procedure; however, consensus is lacking [7, 44].

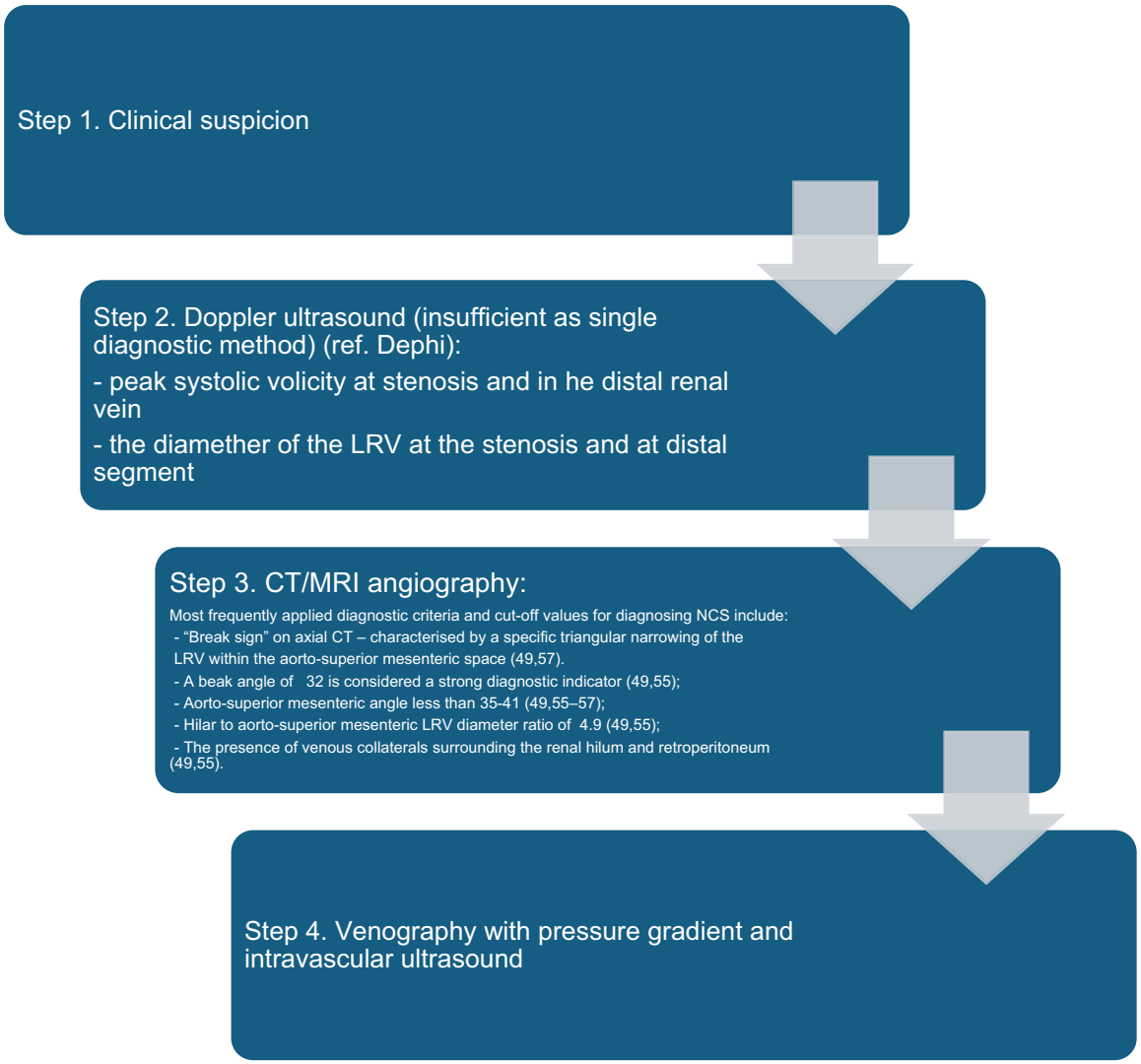
Contrast venography is performed with reno-caval pressure gradient monitoring. Pullback pressure gradient ≥1 mmHg supports the diagnosis; however, results may vary depending on patient positioning, hydration, and collateral circulation [52, 53]. As no consensus is present on pathological pressure gradient threshold, some authors use a pressure gradient of ≥2 mmHg, or even ≥3 mmHg to confirm the diagnosis of NCS, however, normal values do not definitively exclude the condition [21, 40, 44, 52]. Intravascular ultrasound demonstrates a high specificity of 90% for diagnosing NCS, compared with 62% for contrast-enhanced venography and allows evaluation of the severity of LRV stenosis in NCS patients [40, 52].

One of the most recent attempts to standardise the diagnostic strategy for NCS was the Delphi consensus, published in 2025. The agreement was not reached regarding a single gold standard method and imaging cut-off values. However, it is agreed that in suspected NCS, initial evaluation should include one cross-sectional imaging modality and one functional imaging modality. If the results are positive, further diagnostic assessment should be performed using venography with pressure gradient measurement and intravascular ultrasound [7]. The suggested diagnostic approach to NCS is shown in Fig. 4.

TREATMENT

Treatment options for NCS vary from conservative to endovascular techniques and more invasive surgical interventions [7, 50]. Conservative management is based on widening the aorto-superior mesenteric angle and regression of

Table 1: A stepwise approach to the diagnosis of NCS.



renal vein hypertension, most commonly achieved through weight gain with proliferation of visceral adipose tissue or development of venous collaterals. This approach is typically the first treatment option, especially applied for individuals younger than 18 years old, with well-tolerated, mild or non-specific symptoms, or patients who do not meet sufficient diagnostic criteria for NCS [50, 53].

Possible medical management for NCS includes RAAS inhibitors (for arterial hypertension control and orthostatic proteinuria reduction) and aspirin (for renal perfusion improvement) [23, 50, 53].

Surgical interventions are indicated when conservative management is ineffective [44]. According to the 2025 Delphi consensus, surgical treatment was considered as superior to endovascular procedures due to the significant risk of stent migration [7]. LRV transposition is the standard and mostly preferred open surgical method for anterior and posterior NCS [23, 51, 53]. This technique involves repositioning the LRV more distally on the inferior vena cava to relieve aorto-superior mesenteric compression and is highly effective, with symptom resolution in approximately 90% cases [40]. However, reintervention rate ranges from 27% to 30% [50].

Left-renal auto-transplantation is another invasive surgical option, associated with a higher risk of postoperative complications such as graft loss, bleeding or bowel injury [44, 49, 50]. Nevertheless, this approach is used in patients who have undergone previous interventions and have not reached clinical improvement [23, 50]. Less frequently performed interventions include transposition of the superior mesenteric artery, nephropexy with removal of renal varices, and transposition of the left gonadal vein to the inferior vena cava or left common iliac vein [50]. Nephrectomy is considered only as a last resort, reserved for patients with persistent haematuria despite multiple prior interventions [23].

Minimally invasive techniques, including laparoscopic or robot-assisted laparoscopic surgery for LRV transplantation or renal auto-transplantation, are increasingly adopted due to faster recovery times [44]. Extravascular stenting, performed laparoscopically and placed external to the LRV, is another minimally invasive option [50, 54]. Published series report favourable

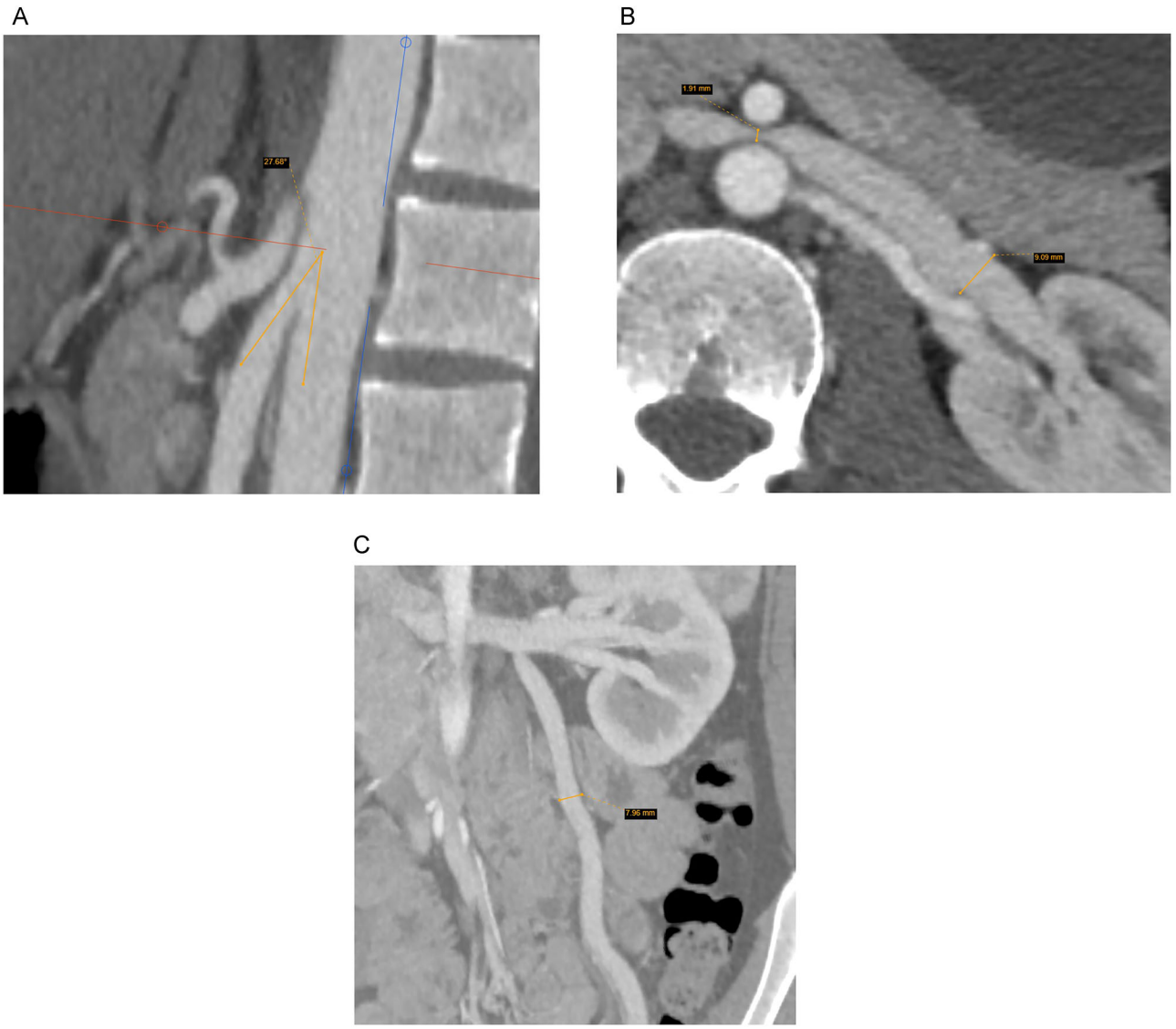


Figure 3: Computed tomography images of the nutcracker phenomenon. (A) The angle (yellow lines) between theaorta and superior mesenteric artery. (B) Left renal vein compression between the aorta and supresior mesenteric artery (short yellow line): the diameter of LRV is 1,91 mm at the site of compression and 9,09 mm at the prie-compression area. (C) Left gonadal vein dilatation and reflux (yellow line).

outcomes with symptom resolution in over 70% cases, and stent migration has been reported in some cases, but remains an uncommon complication [54].

The popularity of endovascular interventions is increasing due to their minimally invasive nature, rapid recovery and have been shown to reduce symptoms in 72%–100% of patients [50, 54]. However, no stents are specifically designed for NCS, which may result in complications such as stent migration, embolization, restenosis, and thrombosis [40, 44, 54]. Reintervention is required in roughly 5% of cases, with stent migration into the inferior vena cava or right atrium. Techniques to surgically fix the stent or hybrid approaches combining endovascular and laparoscopic methods have been described to reduce migration risk, although they add procedural complexity and risks [50, 54, 55].

Although the Delphi consensus recommends LRV transposition as first-line therapy, no universally accepted treatment algorithm exists due to limited and heterogeneous evidence. Recent systematic reviews indicate a trend toward minimally invasive and endovascular approaches, including endovascular stenting, renal auto-transplantation, gonadal vein transposition, LRV transposition, and hybrid techniques, with increasing use of laparoscopic and robotic methods [44, 50].

KNOWLEDGE GAPS AND FUTURE DIRECTIONS

Despite increasing awareness of NCS as a clinically relevant venous compression disorder, high-quality evidence to guide diagnosis, management and follow-up remains limited, particularly in nephrology practice [7, 21, 23, 44]. The incidence of NCS appears to vary with age [12], while its true prevalence and natural history are uncertain because most data come from small, heterogeneous case series, and LRV compression is frequently detected as an incidental radiological finding, especially in children and young adults [21, 23, 44]. Inconsistent use of the terms ‘nutcracker phenomenon’ and ‘nutcracker syndrome’ further

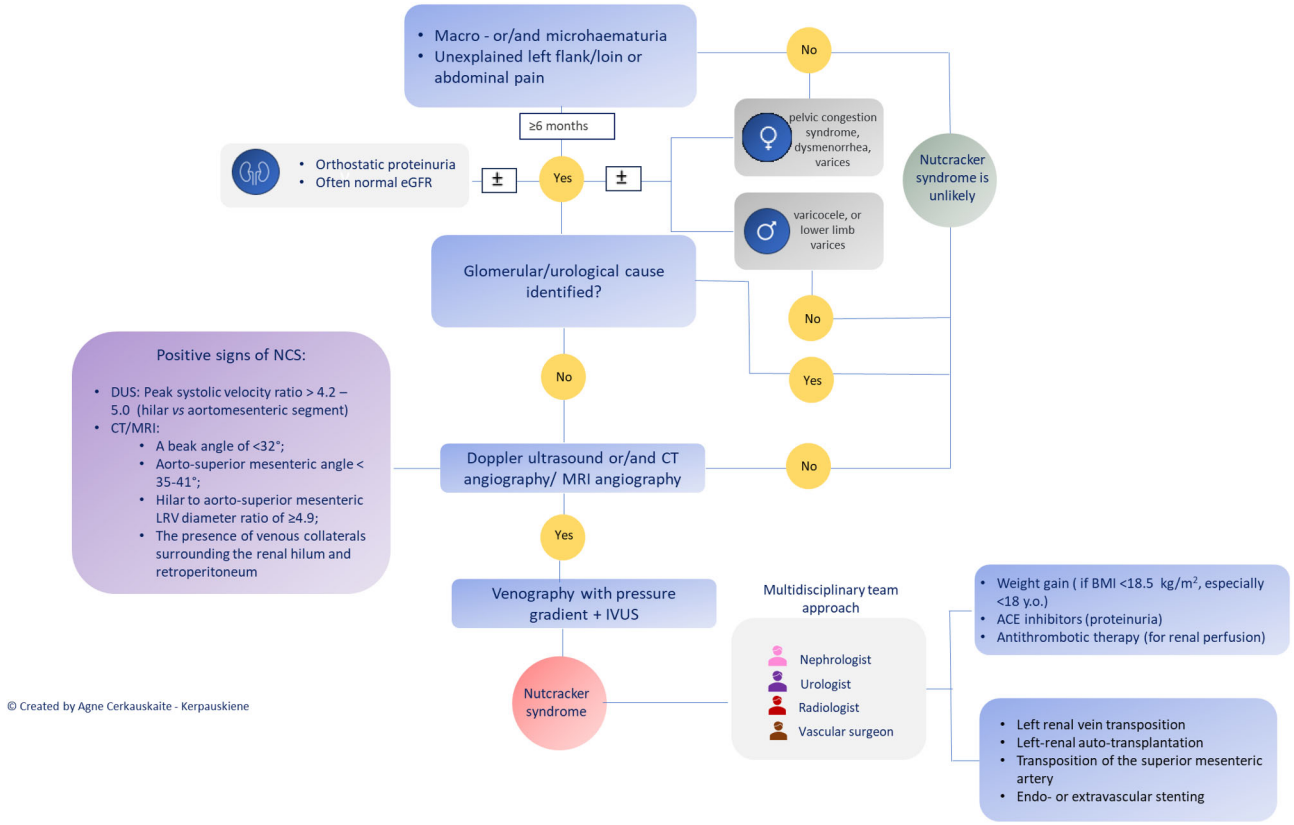


Figure 4: Diagnostic approach and treatment options of the nutcracker syndrome. Diagnosis begins with a clinical evaluation to identify typical signs and symptoms. If no glomerular or urological cause is identified, Doppler ultrasound, or CT angiography, or MRI angiography is evaluated for diagnostic criteria of the nutcracker syndrome. A multidisciplinary approach is needed for the NS treatment.

complicates interpretation and comparison of outcomes [7, 21, 23].

Although radiological evidence of LRV compression is an important element in supporting a diagnosis of NCS, validated diagnostic criteria, age-adjusted reference values and physiologically meaningful haemodynamic and imaging cut-offs are still lacking. Differentiating NCS from other pelvic venous disorders remains challenging, and further randomised studies and international data are needed to standardise imaging assessment [7]. Diagnostic gaps persist and, in particular, isolated radiological LRV compression cannot reliably guide invasive treatment [21, 23, 44]. As a result, decision-making between conservative management and intervention—including LRV transposition, renal autotransplantation and endovascular stenting—is often complicated, highlighting the need for comparative effectiveness research using standardised indications and outcome measures, ideally in multicentre prospective cohorts and, where feasible, randomised trials with adequate long- term follow-up to clarify optimal patient selection and the relative roles of conservative, surgical and endovascular strategies [7, 21, 44, 56–58]. LRV transposition appears relatively low risk in experienced centres, but its safety in specific cohorts such as young women is uncertain, and available evidence for both surgery and stenting remains largely retrospective, single-centre and short term, with endovascular series relying on non-dedicated venous stents that may not optimally match renal vein anatomy [52, 57, 59]. Recent Delphi consensus work and contemporary reviews highlight unresolved questions regarding patient selection, indications or endovascular therapy and antithrombotic strategies, and emphasise the need for dedicated stents, multicentre registries and prospective long-term comparative studies to standardise conservative, surgical and endovascular approaches in NCS [7, 44, 52].

NCS is also a rare and usually under-recognised condition, and awareness in routine nephrology practice appears limited, as most data and expert opinion originate from vascular and radiology-led series rather than nephrology cohorts [7, 21, 23, 44, 52]. Because diagnosis and treatment depend on appropriate imaging, careful exclusion of alternative renal and pelvic causes, and nuanced discussion of conservative versus invasive options, a multidisciplinary approach involving nephrologists, radiologists and vascular/angiovascular surgeons is essential [7, 21, 23, 44, 52, 56]. Future research and guideline efforts should therefore not only address methodological issues (diagnostic criteria, comparative effectiveness and long-term outcomes) but also aim to raise awareness of NCS among nephrologists and formalise multidisciplinary care pathways that can be implemented in everyday clinical practice [7, 21, 23, 44, 52].

CONFLICT OF INTEREST STATEMENT

None declared.

FUNDING

None declared.

DATA AVAILABILITY STATEMENT

No new data were generated or analysed in support of this research.

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