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Nutcracker syndrome – pathophysiology, diagnosis and treatment options: a literature review

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Abstract:

Introduction and purpose: Nutcracker syndrome (NCS) is a rare disorder resulting from renal venous hypertension as a consequence of external compression on the left renal vein (LRV). Because of its non-specific symptoms, it still presents as a diagnostic and therapeutic challenge. In the current literature there is no consensus on clear diagnostic and therapeutic pathway. We conducted a review of the literature to gather and systematize knowledge about this disease in order to describe its etiology, diagnostics and treatment options.

A brief description of the state of knowledge: NCS is a disorder that should be considered in the differential diagnosis of abdominal conditions causing left flank pain, hematuria and proteinuria. Nowadays, the most common first-line diagnostic technique is Doppler ultrasound examination but, in most cases, it must be confirmed by computed tomography, magnetic resonance imaging or invasive venography. In some patients suffering from NCS, withdrawal

of symptoms can be achieved by conservative treatment but sometimes it is insufficient. The most frequent surgical treatment method for this syndrome is LRV transposition, sometimes combined with great saphenous vein autograft. Other invasive techniques include left gonadal vein transposition, kidney autograft, nephrectomy or endovascular treatment.

Summary: Although NCS has been known for a long time, widely accepted diagnostic and treatment criteria are yet to be developed. Physicians can use several treatment options, but the choice often depends on individual clinical presentation and preference. Many issues, for example role of aspirin or renal auto transplantation, still require extensive research.

Key words: nutcracker syndrome, left renal vein compression.

Introduction and purpose

Nutcracker syndrome (NCS) is an ailment consisting of symptoms which result from extrinsic compression of the left renal vein (LRV), leading to unilateral venous hypertension. Asymptomatic obstruction of LRV is a relatively common incidental radiological finding. However, if the occlusion causes significant blood flow disruption, it presents with non-specific chronic or recurrent symptoms and still proves to be a diagnostic and therapeutic challenge. Different medical centers opt for different treatments, based on their individual experience and preference. The purpose of our article is to gather the latest knowledge and present it comprehensively, describing a complete picture of disease, starting from the normal anatomy, through diagnosis, and ending with treatment modalities.

In order to create this article on 09.03.2025 we conducted a review of literature using PubMed, Google Scholar and Elsevier. Two independent authors (B.R. and K.S.) performed a search using terms such as „Nutcracker syndrome” and „left renal vein occlusion”. Any disagreements about inclusion were solved by discussion and consultation with third person (N.K.). Additional references were identified using bibliographies of chosen articles.

Description of state of knowledge

Normal anatomy:

Aorta is the main artery of the human body. It enters the abdominal cavity from the thorax through the diaphragm at Th12 level. In the abdominal cavity, it runs slightly to the left of the median line, along the anterior surface of the lumbar spine. Among the visceral branches of the abdominal aorta are, going from the top down: the visceral trunk (Th12 level), the superior mesenteric artery (SMA, L1 level), the inferior mesenteric artery (L3 level) [1].

Due to the location of the inferior vena cava (IVC) on the right side of the aorta, LRV must cross the abdominal aorta between its anterior surface and SMA to connect with IVC [2]. This spatial relationship of the vessels is the most common, while an anatomical variant (found in 0.8-8.2% cases) is the left renal vein courses posteriorly to the aorta [2, 3, 4, 5].

Anatomical and other causes of nutcracker syndrome

Anterior (‘classic’) nutcracker syndrome is the most popular variant of this disease, in which symptoms are caused by compression of LRV between abdominal aorta and SMA [6]. The nomenclature distinguishes nutcracker phenomenon (there are features of compression on LRV on radiographic imaging) and nutcracker syndrome (when symptoms resulting from nutcracker phenomenon are present) [5, 6, 7]. Nutcracker phenomenon, as a radiologic presentation, can be found incidentally during imaging performed due to other indications or symptoms [6, 7].

The SMA usually originates from the aorta at an angle close to 90°. In case of its reduction to 35° or less and decrease in aortomesenteric distance (which in physiological conditions varies between 10 and 34 mm), additional pressure can be caused on LRV (nutcracker phenomenon or nutcracker syndrome) or on the thrid portion of the duodenum (SMA syndrome). [7, 8].

A less common variant is posterior NCS, in which LRV courses retroaortically. In this case, the LRV is obstructed between the abdominal aorta and the spine. The literature also describes variants of LRV with a circum-aortic course, another subtype of NCS [9, 10].

Other, rarer causes of this condition include pregnancy, pressure on the LRV by swollen soft tissues (e.g. pancreatic neoplasm, adjacent lymphadenopathy), severe lordosis or lumbar osteophytes, decreased retroperitoneal and mesenteric fat, aortic aneurysm, and overarching testicular artery [5, 6, 9, 11]. There are also reports about NCS coexisting with IgA vasculitis

(Henoch-Schönlein purpura), idiopathic hypercalciuria with nephrolithiasis, membranous nephropathy, and IgA nephropathy [12, 13].

Epidemiology and appearance

The first reports about left renal vein compression between the aorta and the SMA date back to 1972, when De Schepper described two cases of patients with NCS symptoms and detected obstruction in renal venography [14]. The asymptomatic nutcracker phenomenon was described earlier, in 1937, by Grant [11].

NCS can occur at any age, ranging from childhood to elderliness, especially in second decade of life and in middle-aged adults [5, 10]. Many authors highlight that the real incidence of NCS remains unknown, however it is believed that due to a high percentage of oligosymptomatic patients, many cases are never diagnosed [9, 10].

Symptoms and clinical features

LRV occlusion generates increased venous blood pressure inside the vessel. The pressure exerted on thin-walled veins results in miniature ruptures and bleeding into the renal collecting system [15]. Moreover, the existence of vascular channels that communicate with calyceal cavity have been discovered. It is suggested that this phenomenon is responsible for their formation [16]. Many authors believe these mechanisms explain ~~of~~ unilateral macroscopic hematuria from the left ureteral orifice which may be seen in cystoscopy. Patients usually report this symptom as recurrent and painless.

Apart from hematuria, all these mechanisms lead to symptoms such as left flank or pelvic pain, anemia, orthostatic proteinuria, autonomic dysfunction, pain associated with gonadal vein syndrome, varicoceles in males (as gradual vein occlusion leads to extensive collateral drainage – mainly via the left gonadal vein [15]), and pelvic congestion syndrome in females [10, 11, 12, 13, 17]. Some patients report exacerbation of symptoms while standing or walking [13]. The retroaortic and circum-aortic course of LRV (‘posterior’ NCS) results in symptoms similar as classic, or anterior, NCS [9].

It is important to remember that nutcracker syndrome symptoms may ~~be~~ resemble those of other causes of abdominal pain, for instance nephrolithiasis (manifesting as renal colic), endometriosis, or renal pelvis vascular malformation. Therefore, physicians should remember about this condition while performing differential diagnosis [13].

Diagnosis and diagnostic methods

Due to of the high variability of symptoms, their recurrent character, the proper diagnosis of NCS is often challenging. Over the years, various centers and researchers have developed their own methods for both diagnosing and treating the disease. The most recent study summarizing the conclusions of an expert panel about NCS diagnose and treatment is an article by Heilijgers et al. (January 2025), known as the 'Delphi consensus'. In our study, we relied on various articles and sources to create the most comprehensive clinical picture of NCS by gathering all available information and experiences.

Diagnosis

Available scientific sources agree that the diagnosis of NCS requires presence of appropriate clinical symptoms, confirmation of their cause, LRV compression, through radiological examination, as well as the exclusion of other potential causes of symptoms, particularly hematuria [18, 19]. Some authors additionally suggest that compression-related symptoms should persist for at least six months [20].

Diagnostic methods

Traditionally, NCS was diagnosed in patients who presented with gross hematuria using LRV catheterization and pressure gradient measurement. Nowadays, non-invasive techniques such as Doppler ultrasound, computed tomography or magnetic resonance imaging are used as first-line methods. Their advantage is the possibility to assess various anatomical structures and parameters, for instance LRV diameter (in distal and proximally sections) or spatial resolution between abdominal aorta and the SMA [21].

Ultrasound examination

Doppler ultrasonography (DUS) should be performed as the first diagnostic method. Due to its non-invasiveness and high accessibility, it is primarily used to measure diameter and peak velocity flow (PVF) in hilar and proximal sections of LRV. Studies suggest a hilar-to-aorto-mesenteric diameter ratio greater than 4.2-4.7 is predictive of NCS. Its sensitivity and specificity are reported to range between 78-100% and 90-100% respectively [13, 17, 22].

Physiological PVF (with no external compression) in proximal LRV ranges from 10 to 20 cm/s. In nutcracker phenomenon (slight compression without clinical consequences), PVE is 40-50 cm/s. In symptomatic NCS, PVF usually exceeds 100 cm/s at the compressed proximal (aorto-mesenteric) section of LRV [21].

DUS is considered as the optimal method for follow-up (e.g., to check stent patency or detect signs of reocclusion), and the first post-operative control examination should be performed within 6 weeks after surgery [20].

Computed tomography (CT)

CT with 3D reconstruction is a reliable diagnostic method. Unlike DUS, CT and MRI provide a detailed spatial resolution of abdominal structures and help ~~to~~ clarify the presence of compression and collateral circulation [13, 18].

A characteristic finding in CT imaging is the 'beak sign'. It is a triangular shape of narrowing of the left renal vein at the aortomesenteric portion, suggesting a severe form of obstruction. Its sensitivity (91.7%) and specificity (88.9%) significantly help in the diagnostic process [17].

Magnetic Resonance (MR)

MR is also a valuable diagnostic method for this syndrome. The findings observed on MRI are like those seen in CT images. The main advantage of MR over CT and venography is lower radiation exposure for patient [17]. Like CT, MRI should always be performed before the surgical intervention.

Venography

The most reliable method to confirm LRV occlusion and secondary renal vein hypertension is renal venography combined with measurement of the pressure gradient between LRV and IVC. This technique also reveals excessive blood flow in collateral vessels, such as gonadal or adrenal veins. Gradient pressures exceeding 2-3 mm Hg also suggest the diagnosis [15, 17]. The development of non-invasive techniques has reduced the need for this test, especially in children. However, in unclear cases [9] and in patients requiring further invasive

intervention, it remains the gold standard for confirming the diagnosis of NCS and implementing appropriate surgical treatment [13, 18, 22].

Treatment methods

There are many techniques and methods of treating NCS. Depending on the patient's profile, the severity of symptoms, and risk-benefit ratio, the final strategy should be chosen individually for each patient.

Conservative treatment is considered by many experts as the first-line option, especially in patients with mild symptoms or microscopic hematuria. Moreover, many sources emphasize the importance of weight gain in underweight patients (BMI < 18.5 kg/m^2) [20, 22, 23]. Reed et al. reported decrease in tension on the LRV with symptoms resolution in 30% of patients treated conservatively, highlighting the importance of increasing BMI when it is too low [24].

The effectiveness of non-invasive treatment is particularly emphasized in patients younger than 18 years of age. In this group, there is a higher chance of spontaneous resolutions of symptoms, estimated at 76.2%. Moreover, symptoms did not reoccur for several consecutive years of follow-up [23, 25].

The routine use of low-dose aspirin after an NCS diagnosis (to optimize kidney perfusion), as well as the use of angiotensin-converting enzyme inhibitors (to improve orthostatic proteinuria), remains questionable. There is no clear consensus on this form of conservative treatment [9, 20, 23, 25].

If the conservative treatment is unsuccessful (e.g., if symptoms worsen or recure, particularly gross hematuria or persistent orthostatic proteinuria), invasive treatment is indicated, ranging from endovascular interventions to complex (open or laparoscopic) surgical procedures. Before undertaking surgical treatment, it is essential to carefully analyze the imaging studies to identify the causes, correlate them with the symptoms, and select the appropriate intervention technique [15]. There is insufficient data to determine the better approach - open or laparoscopic surgery [20].

LRV transposition

In the vast majority of centers, the first-line surgical treatment method is LRV transposition. It is a highly effective technique that requires either minilaparotomy or laparoscopy approach. Antithrombotic or antiplatelet therapy is highly recommended after the

procedure. After tissue mobilization and identification of all structures, the LRV is clamped and transposed as distally as possible to the left lateral aspect of inferior vena cava. In case of 'posterior' NCS, the LRV is moved to the anterior side of the abdominal aorta. Some surgeons additionally perform kidney cooling during the surgery [17, 20, 25, 26].

In some cases, it is necessary to modify the procedure and use an autograft from the great saphenous vein. When persistent compression has caused permanent distortion of the vein, transposition alone may not be sufficient to reduce the pressure gradient. In such cases, additional patch venoplasty is performed to widen the lumen of the vessel and help prevent restenosis. Another potential issue is when the LRV is too short, and transposition puts excessive tension on its walls. In these situations, the great saphenous vein can be used to create a saphenous vein cuff. Alternatively, the great saphneous vein can be used to create bypass around the obstructed section of LRV without transposing the vein itself [24, 25, 27].

Left gonadal vein (LGV) transposition

A method of vascular transposition that proceeds without disrupting the LRV structure is transposition of the LGV. The method is particularly recommended for patients suffering from pelvic symptoms. It involves excision of the LGV and its reimplantation into the IVC or the left common iliac vein [17, 26, 28]. This technique is recommended by the 'Delphi consensus' as an alternative to LVR transposition [20].

By using this method, a collateral pathway for venous blood outflow from the kidney is created, resulting in relief of pelvic congestion and gonadal venous hypertension, regardless of the course of the LRV (anterior, posterior, or atypical) [25]. As with LRV transposition, antiplatelet therapy is used postoperatively as a part of a long-term protocol [20, 28].

SMA transposition

This technique involves resecting SMA and reanastomosing its distal end to abdominal aorta below the LRV. This technique permanently eliminates compression, but there is a risk of bowel ischemia intraoperatively. After the procedure, an antiplatelet agent is ordered as a precaution of thrombosis [29].

Kidney surgery

Renal autotransplantation is a highly invasive and complicated procedure which involves resection of left kidney (open, laparoscopic, or robotic-assisted [30]) and its transplantation to the ipsilateral or contralateral iliac fossa [25]. Some authors claim that, in their experience, patients who undergo this procedure require fewer reinterventions, compared to other surgical methods, and report fewer reoccurrences of symptoms. However, there is no consensus on the safety of this technique, therefore further research is required [20, 31].

We found some case reports that describe less common treatment options, such as total nephrectomy or kidney transplantation from an unrelated donor [32]. In general follow-up of surgical treatment shown good results (87-90% efficiency) in small groups of patients with varying observation periods [27, 33, 34].

Endovascular

The implantation of a stent to prevent LRV stenosis is commonly reported in the literature, however due to the risk of stent migration, it is not considered as a primary option. Additionally, there is a lack of long-term follow-up, so open or laparoscopic surgery is recommended as first [15, 20, 27].

Conclusions

Nutcracker syndrome is a condition that can produce non-specific symptoms over a long period, proving to be an essential part of the differential diagnosis of symptoms such as recurrent hematuria, proteinuria, or left flank pain. Doppler ultrasound examination is sufficient for the initial diagnosis of the syndrome, but further imaging studies are often necessary. Conservative treatment should be considered in the management process, but in many cases surgical interventions, such as left renal vein, left gonadal vein transposition, or kidney autotransplantation, are required. Despite numerous studies, issues such as the role of aspirin, follow-up duration, and the safety and efficacy of renal autotransplantation remain to be further investigated.

Disclosure:

Authors' contribution

Conceptualization: BR and KS;

Methodology: BR and NK;

Software: BR and JMM;

Check: AD, MJ and JMM;

Formal analysis: NK;

Investigation: BR and KS;

Resources: MJ and KS;

Data curation: AD;

Writing - rough preparation: BR and NK;

Writing - review and editing: KS, MJ and JMM;

Visualization: AD and MJ;

Supervision: KS;

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