ANNALES ACADEMIAE MEDICAE SILESIENSIS

ACCEPTED MANUSCRIPT

Ann. Acad. Med. Siles. (Online) 2026; DOI: 10.18794/aams/221931

Case report

Thrombosis of the left renal vein in the course of vascular anomaly – posterior nutcracker syndrome: Case report

Małgorzata Wojtaluk[1], Weronika Mucha[1], Rafał Donderski[2], Wioleta Stolarek[2], Przemysław Ratajczak[3]

[1]Students' Scientific Club at the Department of Nephrology, Hypertension, and Internal Medicine, Nicolaus Copernicus University in Toruń, Ludwik Rydygier Collegium Medicum in Bydgoszcz, Poland
[2]Department of Nephrology, Hypertension, and Internal Medicine, Nicolaus Copernicus University in Toruń, Ludwik Rydygier Collegium Medicum in Bydgoszcz, Poland
[3]Department of Radiology and Diagnostic Imaging, Nicolaus Copernicus University in Toruń, Ludwik Rydygier Collegium Medicum in Bydgoszcz, Poland

Address for correspondence:
Małgorzata Wojtaluk
Studenckie Koło Naukowe
Katedra Nefrologii, Nadciśnienia Tętniczego i Chorób Wewnętrznych
Szpital Uniwersytecki nr 1 im. dr. A. Jurasza
ul. Curie Skłodowskiej 9, 85-094 Bydgoszcz
e-mail: gosiawojtaluk02@gmail.com

Received: 24.02.2026, Revised: 28.04.2026, Accepted: 16.05.2026, Published: June 2026

Publisher: Medical University of Silesia, Katowice, Poland

ABSTRACT

Nutcracker syndrome is a vascular anomaly, of which the posterior variant characterized by the compression of the left renal vein between the aorta and the spine, is extremely rare. We present a case of a 40-year-old female patient with acute lumbar pain subsequently diagnosed with posterior variant of nutcracker syndrome complicated by left renal vein thrombosis and suspected renal infarction. Treatment involved an endovascular intervention - mechanical thrombectomy and balloon angioplasty – as well as anticoagulant therapy, successfully restoring flow in the left renal vein. The paper discusses current diagnostic and therapeutic trends in this rare anatomical variant.

KEYWORDS

renal vein thrombosis, posterior nutcracker syndrome, vascular angioplasty

INTRODUCTION

Nutcracker syndrome is a rare vascular anomaly involving compression of the left renal vein. There are two types: anterior, in which the left renal vein is compressed between the aorta and the superior mesenteric artery, and posterior, in which compression occurs between the aorta and the spine. While more commonly diagnosed in women presenting with chronic pelvic or back pain, the posterior variant may also manifest through periodic episodes of macroscopic or microscopic hematuria and orthostatic proteinuria, although cases of nephrotic proteinuria have also been described in its course. In addition, gonadal or spermatic reflux may occur, promoting the development of varicocele [1,2,3]. Some patients experience orthostatic hypotension (dizziness, weakness) and chronic pelvic congestion, causing pain radiating to the buttocks and back of the thigh. In advanced cases, chronic fatigue syndrome is observed as well, resulting from venous hypertension and impaired blood flow through the left renal vein [3].

Diagnostic imaging of this phenomenon includes ultrasound examination with Doppler ultrasound (DUS), computed tomography angiography (CTA) and magnetic resonance angiography (MRA). The most accurate method, considered the gold standard, remains venography with intravascular ultrasound (IVUS) and phlebography with measurement of the pressure gradient in the renal veins and superior and inferior vena cava [4]. A characteristic feature of the posterior variants is that at the level of compression, there is a shortening of the aorto-vertebral distance, which is the smallest measured gap between the aorta and the vertebral body [2,5]. In younger patients with mild symptoms, including minor hematuria, conservative treatment is preferred, with careful

observation of patients due to the frequent possibility of spontaneous remission of symptoms. In more advanced cases, endovascular procedures or surgical treatment are used [2].

CASE REPORT

We present the case of a 40-year-old female patient who was urgently admitted to the Department of Nephrology, Hypertension and Internal Disease because of severe, escalating pain in the left lumbar region lasting approximately 12 hours, accompanied by nausea, anxiety, and restlessness. Aside from a 10-year history of chronic smoking (10 cigarettes daily) and the use of oral contraceptives, the patient's medical history was otherwise unremarkable.

Physical examination on admission revealed: the patient's general condition was stable. Blood pressure: 136/65 mmHg, regular heart rate of about 80 beats/min. No additional murmurs over the heart. Normal breath sounds over the lungs. Abdomen was soft, moderately painful on the left side. The peritoneal signs were negative. There were normal bowel movements. There was a positive Goldflam's sign on the left side. There was no peripheral edema. Vital signs at the admission were as follows: temperature: 36.4°C, weight: 70 kg, height: 165 cm, BMI: 25.71, oxygen saturation: 96%.

An abdominal ultrasound undertaken upon admission revealed abnormalities in the left kidney: significant enlargement and swelling of the left kidney. In addition, fluid was found in the retroperitoneal space. A Doppler ultrasound scan revealed an image suggesting impaired blood flow from the narrowed left renal vein with the presence of thrombosis. Laboratory parameters performed in the following days of the patient's stay are presented in Table I.

Table I. Laboratory results on admission, one day and three days after endovascular interventions, and on discharge

Parameter (reference values)	The day of admission	The day after the intervention	3 days after the intervention	The day of discharge
Hb (g/dl) (11.2–15.7)	14.7	11.4	9.0	10.4
WBC (10^3 /μL) 3.98–10.04	17.18	16.84	10.48	7.20

PLT (10^3 /μL) 132–370	323	179	203	572
RBC (10^6 / μL) 3.93–5.22	4.94	3.65	3.04	3.31
CRP (mg/dl) < 5.0	19.61	248.89	230.20	10.89
Na (mmol/l) (136–145)	137.6	132.6	137.5	139.4
K (mmol/l) (3.5–5.1)	3.6	3.7	3.3	4.7
Creatinine concentration (mg/dl) enzymatic method (0.55–1.02)	1.20	0.87	0.90	0.83
eGFR (ml/min) CKD-EPI 2021 (>60.00)	56.90	83.25	79.81	88.62
D-dimers (ng/ml) <500	–	–	4991	2413

Hb – hemoglobin; WBC – white blood cells; PLT – platelets; RBC – red blood cells; CRP – C-reactive protein; Na – sodium; K – potassium; eGFR – estimated glomerular filtration rate.

Urinalysis showed erythrocyturia and leukocyturia, while a subsequent computed tomography (CT) scan confirmed critical narrowing of the left renal vein due to compression by the aorta, characteristic for posterior nutcracker syndrome (Figure 1).

Fig. 1. Computed tomography scan showing the left renal vein in a retroaortic position and an image of an enlarged left kidney

In addition, CT revealed fluid in the retroperitoneal space and around the kidney, as well as hypoplasia of the left branch of the portal vein, which could have been a congenital anomaly or the result of previous thrombosis. During the diagnostic process, a hematoma of the left adrenal gland was found, which was monitored in subsequent follow-up examinations.

During hospitalization, laboratory tests were performed to identify other potential causes of thrombosis. Systemic lupus erythematosus and systemic vasculitis were excluded based on normal immunological profiles, including c-ANCA (PR3) <2.3 CU (ref. <20.0 CU), p-ANCA (MPO) <3.20 CU (ref. <20.00 CU). Antinuclear antibody (ANA) was negative at 37.20 AU/ml (ref. <40,00), and complement components C3 and C4 remained within normal limits (C3: 1.15 g/l; ref. 0.79–1.52, C4: 0.18; ref. 0.16–0.38). Primary antiphospholipid syndrome as a cause of thrombosis was excluded, with lupus anticoagulant absent and negative titers for anticardiolipin antibodies: IgG 3.1 PL-IgG-U/ml (ref. <12 PL-IgG-U/ml), IgM <2.0 PL-IgM-u/ml (ref. <12 PL-IgM-U/ml) and IgA <2.0 PL-IgA-u/ml (ref. <12 PL-IgA-U/ml). Thrombophilia was further cross-referenced with normal levels of Protein C – 92.% (ref. 70.0–140.0) and Protein S – 90.7% (ref. 54.7–123.7). Moreover, selected tumor antigens such as alpha-fetoprotein, carcinoembryonic antigen (CEA), cancer antigen 19-9 (CA19-9) were not elevated.

Tests were also ordered for genetically determined causes of thrombosis (Factor V Leiden mutation, prothrombin gene mutation) and assessment of anti-citrullinated protein antibodies (ACCP), anti-GBM, and the ANA/ENA BLOT basic profile. No abnormalities were detected. On this

basis, thrombosis of the left renal vein was considered to be part of the clinical picture of posterior nutcracker syndrome, rather than a separate disease entity.

In view of the confirmed thrombosis of the left renal vein and renal perfusion disorders, mechanical thrombectomy and balloon angioplasty were initiated urgently in the Radiology Department by an interventional radiologist. A vascular catheter (CAT 8, Penumbra) was inserted through the right jugular vein. Three catheter passes were performed, which allowed the removal of thrombi, restoring partial flow in the left renal vein. Despite this, a follow-up phlebography showed critical narrowing of the vessel secondary to compression by the aorta, which required further intervention.

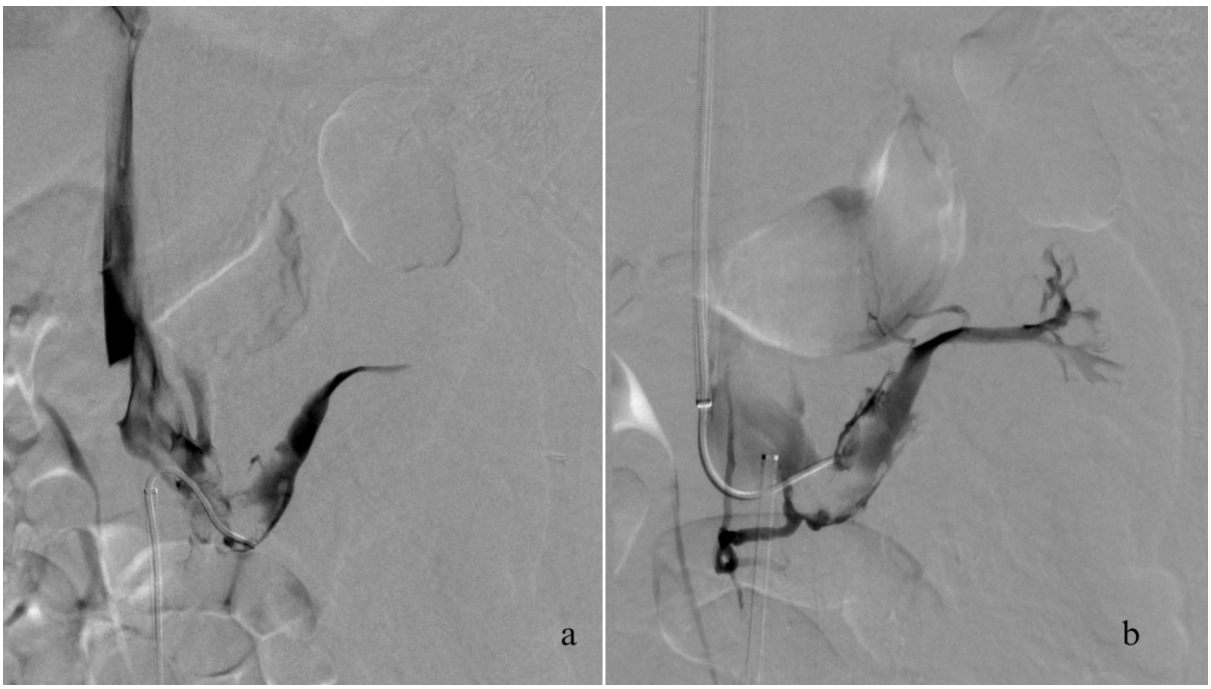

Fig. 2. Images from phlebography performed before thrombectomy (a) and after mechanical thrombectomy (b)

Subsequent to thrombectomy, balloon angioplasty was implemented to achieve mechanical dilation of the compressed renal vein. A 16 × 40 mm balloon was used, which was inserted under fluoroscopic guidance and then gradually inflated. The procedure improved blood flow from the kidney, although the compression of the vein by the aorta was not completely eliminated. The stenosis was most likely structural in nature and required further monitoring and conservative therapy.

After the procedures, the patient was closely monitored, with blood samples taken every 4-6 hours. During hospitalization, anticoagulant therapy was initiated, first with low molecular weight heparin (LMWH) – Enoxaparin 2 × 80 mg s.c., followed by dabigatran at a dose of 2 × 150 mg daily. The decision to maintain full anticoagulation despite the confirmed anemia was associated with

the risk of potential bleeding, but was justified by the imaging diagnosis of thrombosis of the left renal vein and a relatively stable hemoglobin level.

In addition, during the period of intervention procedures, owing to elevated inflammation markers, including C-reactive protein (CRP) and procalcitonin (PCT), broad-spectrum antibiotic therapy was administered. Blood and urine cultures were negative.

During hospitalization, the patient developed secondary anemia from blood loss due to an adrenal hematoma, intravascular procedures, and retroperitoneal bleeding. After 30 days, she was discharged in good condition with anticoagulant therapy and follow-up imaging orders. She was referred to a nephrology clinic. Renal scintigraphy was scheduled to assess perfusion and excretory function, providing an objective evaluation of renal performance following these vascular complications.

DISCUSSION

The reported case of a 40-year-old female patient represents a significant contribution to the knowledge of posterior nutcracker syndrome, which, due to its anatomy, poses an unique diagnostic challenge. Although there is no specific information on the prevalence of this anomaly, the retro-aortic position of the left renal vein is described in the literature in 1.84% of the population [6]. Owing to the extreme rarity of this condition, clinical data regarding its management remain scarce.

In the presented case, mechanical thrombectomy and balloon angioplasty undertaken by an interventional radiologist were used. The procedures were successful. A follow-up contrast-enhanced CT scan of the abdomen and pelvis carried out 2 months after discharge from the hospital showed recanalization of the left renal vein and normal contrast agent excretion into the urine. In addition, compared to previous examinations, a reduction in the left adrenal hematoma was observed.

The diagnosis of nutcracker syndrome is based on clinical assessment and imaging tests. Doppler ultrasonography is the recommended first-line test, but its results may vary depending on the patient's position and technical conditions. In cases of inconclusive results, CTA and MRA should be used, which allow for accurate anatomical assessment but do not enable dynamic analysis of blood flow. Although the aortic-vertebral distance can be measured via ultrasound or multi-channel CT, the lack of a defined precise cut-off point currently prevents this parameter from enabling an unambiguous diagnosis of this syndrome [2].

Presently, despite being invasive, phlebography with pressure gradient measurement and IVUS are the diagnostic gold standards. However, overlapping pressure values between healthy individuals and patients can complicate result interpretation [4].

Once a diagnosis is established, the management strategy must be carefully tailored to the patient's age and the severity of clinical manifestations. In mild cases, particularly among patients under 18, a conservative approach is preferred. This strategy relies on the natural development of intra-abdominal tissue to alleviate venous pressure, potentially supplemented by analgesics, anticoagulants, or angiotensin-converting enzyme inhibitors. Lifestyle changes are important as well, including avoiding prolonged sitting and intense physical activity, which can exacerbate symptoms [4].

If severe symptoms persist, such as recurrent hematuria leading to hemodynamic instability and anemia, surgical intervention is indicated. Endovascular stenting is hindered by unfavorable anatomical conditions – the direct proximity of the aorta to the vertebral bodies restricts the available vascular space [7]. Left renal vein transposition is used as the primary method, involving its resection from the junction with the inferior vena cava and reimplantation at a distal site free from spinal compression. This procedure carries risks of venous thrombosis, hemorrhage, and paralytic ileus. An alternative is the more invasive renal autotransplantation, specifically into the iliac fossa, which requires new vascular and ureteral anastomoses under conditions of prolonged organ ischemia. Venous bypasses, notably those involving the saphenous vein, are used less frequently due to the risk of thromboembolic complications and mesenteric ischemia [4].

Both procedures (left vein transposition and autotransplantation) can also be performed laparoscopically. This approach serves as a less invasive alternative to classic open surgery, providing the patient with a shorter recovery time while maintaining the same efficacy in bypassing the site of vascular compression [4].

In recent years, endovascular techniques such as stent implantation have gained popularity, as they can reduce venous compression and improve blood flow [4,8]. However, in this case too, we must take into account the possible risks of stent migration, stent thrombosis, or stent restenosis [8].

Although minimally invasive techniques are associated with a lower risk of complications and faster recovery, the choice of a specific method depends on the location and degree of vein compression, as well as the individual anatomical conditions of the patient. After surgical interventions, it is important to monitor patients regularly to assess the effectiveness of the

procedure and identify any complications, such as thrombosis or deterioration of kidney function [4].

In the presented patient, mechanical thrombectomy, although technically difficult due to the unusual anatomy of the vessels, allowed blood flow to be restored. Balloon angioplasty proved effective in dilating the narrowed section of the vein, although it did not completely eliminate the compression caused by the aorta. This case illustrates the need to consider more permanent treatments, such as stenting or reconstructive surgery, in cases of persistent compression. More recently, left renal vein stenting has emerged as a less invasive option, though evidence regarding its long-term efficacy remains insufficient [9].

Authors' contribution

Study design – M. Wojtaluk, W. Mucha, R. Donderski

Data collection – W. Stolarek, P. Ratajczak, R. Donderski

Manuscript preparation – M. Wojtaluk, W. Mucha, R. Donderski

Literature research – M. Wojtaluk, W. Mucha, R. Donderski

Final approval of the version to be published – M. Wojtaluk, W. Mucha, R. Donderski, W. Stolarek, P. Ratajczak

REFERENCES

1. Onka B, Khouchoua S, Yehouenou TRT, Jerguigue H, Latib R, Omor Y. Nutcracker syndrome: A rare cause of chronic pelvic pain and left back pain. Radiol Case Rep. 2021;16(8):2025–2030. doi: 10.1016/j.radcr.2021.05.007.

2. Gozzo C, Farina R, Foti PV, Iannace FA, Conti A, Pennisi I, et al. Posterior nutcracker syndrome: a case report. J Med Case Rep. 2021;15(1):42. doi: 10.1186/s13256-020-02617-0.

3. Kurklinsky AK, Rooke TW. Nutcracker phenomenon and nutcracker syndrome. Mayo Clin Proc. 2010;85(6):552–559. doi: 10.4065/mcp.2009.0586.

4. Ananthan K, Onida S, Davies AH. Nutcracker Syndrome: An Update on Current Diagnostic Criteria and Management Guidelines. Eur J Vasc Endovasc Surg. 2017;53(6):886–894. doi: 10.1016/j.ejvs.2017.02.015.

5. Burger JA, Becker LA, Li Z, Wang Z, Schmidt H, Schömig F, et al. The aortic-vertebral distance is more associated with axial plane deformities than coronal and sagittal deformities in idiopathic scoliosis patients of Lenke types I and II. Eur Spine J. 2025;34(2):593–601. doi: 10.1007/s00586-025-08663-w.

6. Kuzan TY, Kuzan BN, Telli TA, Tüney D. Evaluation of the frequency of left renal vein variations in computed tomography and its relationship with cancer development. Folia Morphol. 2020;79(4):793–798. doi: 10.5603/FM.a2019.0137.

7. Chansakaow C, Orrapin S. Posterior Nutcracker Syndrome Caused by Abdominal Aortic Aneurysm: A Case Report. Vasc Spec Int. 2023;39:13. doi: 10.5758/vsi.230036.

8. Park JH, Lee GH, Lee SM, Eisenhut M, Kronbichler A, Lee KH, et al. Posterior nutcracker syndrome – a systematic review. Vasa. 2018;47(1):23–29. doi: 10.1024/0301-1526/a000670.

9. Avgerinos ED, Saadeddin Z, Humar R, Salem K, Singh M, Hager E, et al. Outcomes of left renal vein stenting in patients with nutcracker syndrome. J Vasc Surg Venous Lymphat Disord. 2019;7(6):853–859. doi: 10.1016/j.jvsv.2019.06.016.