https://doi.org/10.15360/1813-9779-2026-3-2729

Minimally Invasive Management of Superior Mesenteric Artery Syndrome with Concurrent Nutcracker Phenomenon: Case Report

Miloslav Mišánik[1], Marek Smolár[1]*, Martin Grajciar[1], Diana Musová[1], Lukáš Spevák[1], Ján Janík[1], Beata Drobná Sániová[2], Juraj Miklušica[1]

[1] Clinic of General, Visceral and Transplant Surgery, Jessenius Faculty of Medicine, Martin, Comenius University Bratislava, 2 Kollárova Str., 03601 Martin, Slovak Republic
[2] Clinic of Anaesthesiology and Intensive Care Medicine, Jessenius Faculty of Medicine, Martin, Comenius University Bratislava, 2 Kollárova Str., 03601 Martin, Slovak Republic

For citation: *Miloslav Mišánik, Marek Smolár, Martin Grajciar, Diana Musová, Lukáš Spevák, Ján Janík, Beata Drobná Sániová, Juraj Miklušica.* Minimally Invasive Management of Superior Mesenteric Artery Syndrome with Concurrent Nutcracker Phenomenon: Case Report. *Obshchaya Reanimatologiya = General Reanimatology.* 2026; 22 (3): 41–47. https://doi.org/10.15360/1813-9779-2026-3-2729 [In Engl.]

***Correspondence to:** Marek Smolár, marino.smolar@gmail.com

Summary

Superior mesenteric artery (SMA) syndrome is a rare cause of high duodenal obstruction.

We report a case of an asthenic male (BMI 17.9 kg/m²) presenting with acute bowel obstruction.

Methods. Multiphase CT demonstrated marked pre-stenotic dilation of the D2 of the duodenum (max 8.8 cm) and compression of the D3 between the aorta and SMA, with an aortomesenteric angle of 11–12° and distance of 7 mm. A morphological, asymptomatic nutcracker phenomenon was present without hematuria. CT-suggested colitis was ruled out endoscopically and histologically. After consultation with vascular surgeon and shared decision-making, the patient opted for enteral bypass. The patient's preoperative anaesthesiology assessment revealed no contraindications to general anaesthesia for the planned laparoscopic procedure. A laparoscopic laterolateral, anisoperistaltic duodenojejunostomy was performed using a linear stapler (minimal blood loss; operative time 76 minutes) with a perianastomotic drain.

Results. Postoperatively, the nasogastric tube was removed on POD2, oral intake resumed from POD3, and the drain removed on POD4. Transient rise in CRP/leukocytosis on POD2 (negative presepsin) was managed empirically with ampicillin/sulbactam (Clavien–Dindo II). Patient was discharged on POD6 (7-day stay). At three months follow-up he remained symptom-free with objective nutritional gain (BMI + 2.3 kg/m²).

Conclusion. This case supports laparoscopic duodenojejunostomy as a safe and effective definitive option in hemodynamically stable patients with CT-quantified SMA obstruction. A concomitant asymptomatic nutcracker phenomenon does not require vascular intervention nor alter operative strategy.

Keywords: superior mesenteric artery syndrome; SMA; Wilkie's; duodenal obstruction, aortomesenteric angle, nutcracker phenomenon, laparoscopic duodenojejunostomy

Conflict of interest. The authors declare no conflict of interest.

Acknowledgments. The authors of the case report would like to thank the patient for his willingness to cooperate and for his consent to share medical information for the purposes of the educational process and for the writing of publications.

Information about the authors:
Miloslav Mišánik: https://orcid.org/0009-0000-8924-6645
Marek Smolár: https://orcid.org/0000-0002-7381-5513
Martin Grajciar: https://orcid.org/ — no ORCID
Diana Musová: https://orcid.org/0009-0004-7008-4323
Lukáš Spevák: https: /orcid.org/ — no ORCID
Ján Janík: https://orcid.org/ — no ORCID
Beata Drobná Saniova: https://orcid.org/0009-0009-4453-4446
Juraj Miklušica: https://orcid.org/0009-0003-6442-4064

Introduction

Superior mesenteric artery (SMA) syndrome (Wilkie's syndrome) is a rare cause of high duodenal obstruction resulting from compression of the third (D3) portion of the duodenum between the aorta and the overlying SMA [1, 2].

Its estimated population incidence is low, most commonly reported at approximately 0.013–0.3%. Diagnosis relies on the clinical findings and contrast-enhanced CT with assessment of the aortomesenteric angle and the aortomesenteric distance [1, 2].

The nutcracker entity denotes compression of the left renal vein between the aorta and the SMA. The nutcracker phenomenon is a morphological, often incidental finding without symptoms, whereas the nutcracker syndrome represents the symptomatic form with hematuria and/or venous congestion [3, 4].

Coexistence of SMA syndrome with a nutcracker entity is rare and has been reported mainly in single case reports [5–7].

Here, we present SMA syndrome with a con-

current asymptomatic nutcracker phenomenon managed by laparoscopic duodenojejunostomy. The case report was prepared in accordance with the SCARE criteria [8].

Patient consent

The authors of the case study obtained a written consent from the patient to publish this case study and to use the medical information and visual aids.

Ethics Commission

The approval of the ethics committee was not necessary as the article is a completely anonymized case study and no experiments were performed. A clear written consent was obtained from the patient regarding the photographs and information being published.

Case report

An asthenic man (BMI 17.9 kg/m²) was evaluated in the Emergency Department for acute ileus presenting with sudden epigastric pain, recurrent vomiting, and cessation of flatus. His history included hospitalization in 2013 for abdominal pain, then assessed as duodenoparesis with esophageal dysmotility. From that time until the current episode he tolerated solid food and maintained a stable weight without significant loss. On admission, abdominal distension was present with maximal tenderness in the epigastrium and absent bowel sounds. Laboratory tests showed leukocytes 19.6×10^9/L and CRP 11 mg/L, with mild hyponatremia. Initial X-ray/ultrasound confirmed a sub-ileus/ileus pattern. A subsequent multiphase abdominal CT demonstrated marked prestenotic dilatation of the D2 duodenum (up to 8.8 cm) and compression of the D3 segment between the aorta and the superior mesenteric artery (SMA), with an aortomesenteric angle of 11–12° and an aortomesenteric distance of 7 mm (Fig. 1). Segmental colitic changes of the colon described on CT (suspected IBD) were later excluded by pancolonoscopy during differential work-up. Incidentally, the CT also revealed the morphological picture of an asymptomatic nutcracker phenomenon (compression of the left renal vein

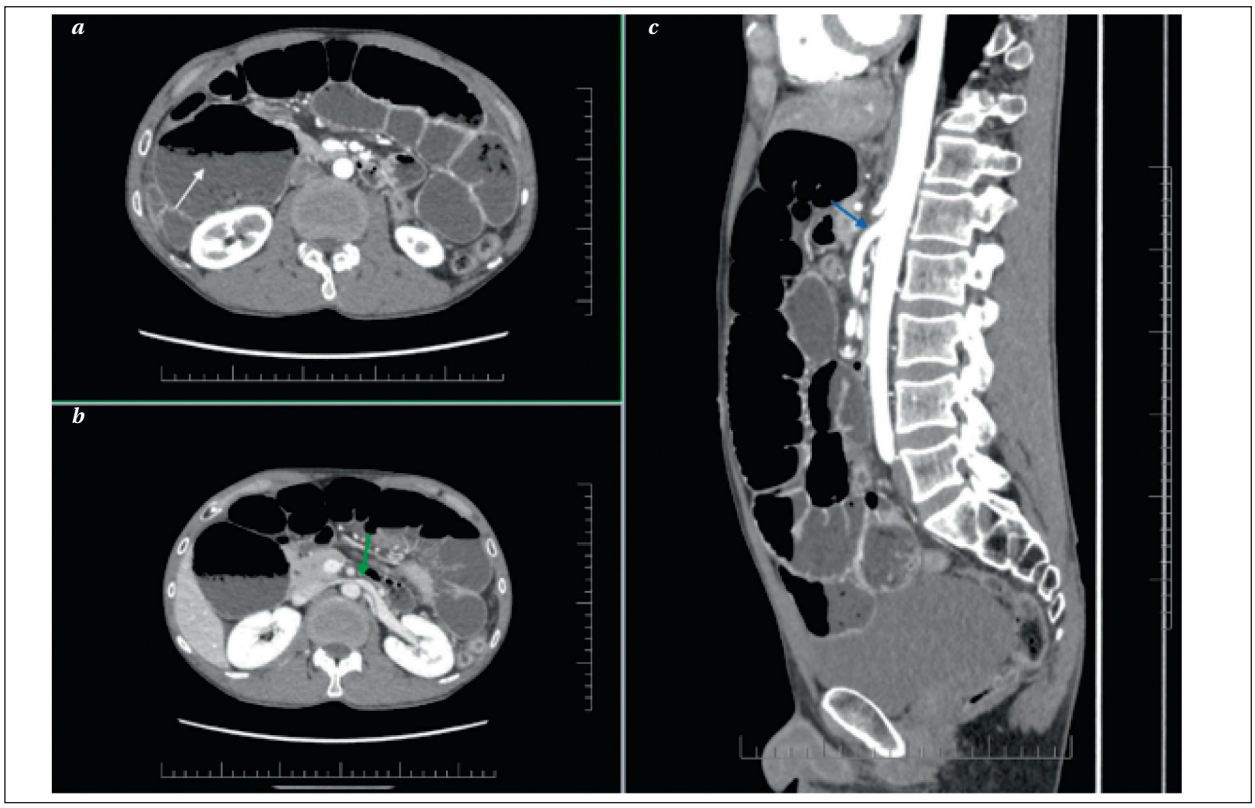


Fig. 1. CT findings in SMA syndrome with concurrent nutcracker phenomenon.
a — axial image — marked pre-stenotic dilation of D2 of the duodenum (max 8.8 cm, arrow); *b* — axial image — pre-stenotic dilation of the left renal vein with tapering at the aortomesenteric space (asymptomatic nutcracker phenomenon, arrow); *c* — sagittal image — narrow aortomesenteric angle ~11–12° with the transition point in D3 (arrow). Source: author.

Table. Objective parameters, reference thresholds and clinical interpretation. Patient data: present case; reference thresholds per [1, 2]; nutcracker definition per [3]; BMI per WHO [9].

Parameter	Patient value	Reference values / thresholds	Interpretation
Aortomesenteric angle (CT)	11–12°	Physiologic ~28–65°; suspicious for SMA ⩽ 22° [1, 2]	Strongly supportive of SMA
Aortomesenteric distance (CT)	7 mm	Physiologic ~10–28 mm; suspicious for SMA ⩽ 8 mm [1, 2]	Strongly supportive of SMA
D2 (second portion) dilation, max diameter	8.8 cm	In SMA typically marked proximal duodenal dilation (often >3–4 cm) [1,2]	High-grade obstruction proximal to D3
BMI	17.9 kg/m²	< 18.5 kg/m² = underweight (predisposing factor for SMA) [9]	Asthenic habitus — predisposition for AMS
Nutcracker phenomenon (left renal vein)	present; no hematuria	Phenomenon = morphologic LRV compression without symptoms; Syndrome = with hematuria/ venous congestion [3]	Asymptomatic finding; does not change acute management
Endoscopy/histology for IBD	not confirmed	—	CT 'colitic' changes without endoscopic/histologic correlation

Note. Published «normal» ranges for aortomesenteric angle/distance vary slightly; the thresholds ⩽ 22° and ⩽ 8 mm are among the most commonly cited and clinically used [1, 2].

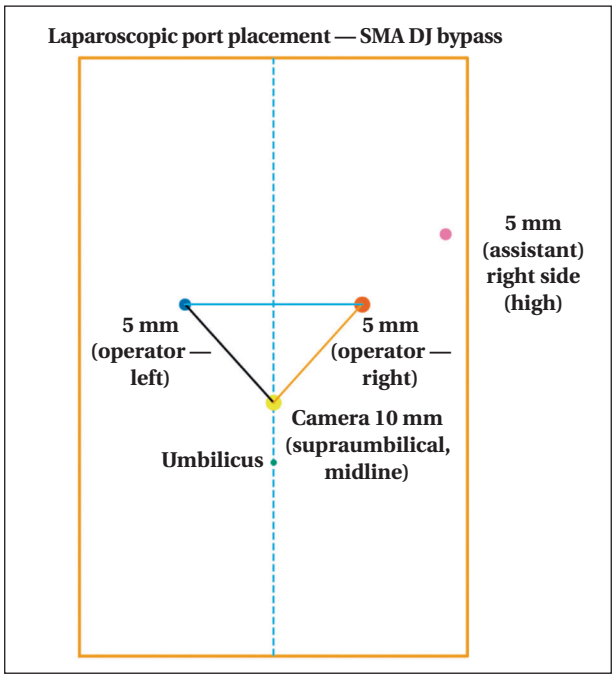


Fig. 2. Port Placement for laparoscopic DJ bypass.
Source: author.

between the aorta and SMA) without clinical manifestations. The relevant measurements and threshold values are summarized in Table.

After consultation with vascular surgery, the diagnosis of Wilkie's syndrome was confirmed, operative options and risks were explained. SMA transposition was proposed, but the patient preferred an intestinal bypass. Following preoperative optimization (hydration, proton-pump inhibitor, LMWH prophylaxis, nutritional support; internal medicine/anaesthesiology assessment: ASA II), we performed a laparoscopic latero-lateral, anisoperistaltic, stapled duodenojejunostomy. General anaesthesia was administered in accordance with the requirements for a laparoscopic procedure (patient intubation, inhalational anaesthesia, and analgesia with opioids plus sedatives). Port configuration: a 10 mm camera port supraumbilically on the midline, above it, for triangulation, a 5 mm (left, operator) and a 10 mm (right, operator) port and a separate 5 mm assistant port to the right above the triangulation (Fig. 2). A drain was left near the anastomosis. The procedure was completed without intraoperative complications with minimal blood loss, an operative time 76 minutes and no need for conversion.

POD1: CRP 113 mg/L, leukocytes 16×10^9/L. POD2: leukocytes rose to ~20×10^9/L and CRP to 306 mg/L; presepsin was negative, therefore empiric antibiotic therapy with ampicillin/sulbactam (Unasyn) was initiated, with subsequent improvement and decline of inflammatory markers (Clavien–Dindo II). The nasogastric tube was removed on POD2. Oral refeeding began on POD3 with stepwise advancement (liquids to soft diet) and emphasis on small, frequent, high-energy/high-protein meals, complemented by oral nutritional supplements. Estimated requirements were calculated using a pragmatic weight-based method (ideally from ideal body weight): ~25–30 kcal/kg/day (≈105–126 kJ/kg/day; 1 kcal = 4.184 kJ) with ~1.2–1.5 g protein/kg/day [10]. Electrolytes (phosphate, potassium, magnesium) were monitored during refeeding according to local protocol. If oral intake remained insufficient (e. g., < 60% of the target), escalation to enteral feeding distal to the anastomosis (or short-term parenteral nutrition) was considered. The drain was removed on POD4. The patient was discharged on POD6 (total hospital stay of 7 days) with a structured nutritional plan for home focused on ongoing weight gain (small frequent energy-

dense meals) including a prescription for high-energy, high-protein oral nutritional supplements (e. g., 2–3 servings daily) in addition to meet the calculated targets. At 3-month outpatient follow-up he reported good dietary tolerance without dyspeptic symptoms and weight gain, with BMI increased by 2.3 kg/m².

Discussion

Superior mesenteric artery (SMA) syndrome (Wilkie's syndrome) is a rare vascular variant in which a narrowed aortomesenteric angle and reduced aortomesenteric distance lead to compression of the D3 portion of the duodenum between the aorta and the SMA. It typically occurs in asthenic patients with low BMI, after rapid weight loss, or in the presence of anatomic predispositions [1, 2, 11–13].

In our case, the clinical picture of high-grade obstruction correlated with the CT findings (aortomesenteric angle 11–12°, distance 7 mm) and marked prestenotic dilation of D2, meeting the most commonly reported diagnostic criteria for SMA syndrome [1, 2, 11, 13].

Diagnosis relies on contrast-enhanced CT with multiplanar reformations (MPR). The aortomesenteric angle is measured on a sagittal plane aligned to the SMA axis, the aortomesenteric distance on a coronal plane at the D3 level, with additional assessment of the extent of oral duodenal dilatation. Although published ranges vary slightly, thresholds of ⩽ 22° and ⩽ 8 mm are among the most frequently cited and clinically used [1, 2, 11–13]. Because the aortomesenteric angle and distance vary with posture and respiration, standardizing measurement conditions (phase, MPR alignment) is important. In our case we used portal-venous phase with MPR aligned to the SMA axis and at the D3 level [11, 12]. Our diagnostic measurements and visual correlates (angle 11–12°, distance 7 mm, D2 dilatation) are shown in Fig. 1 and summarized against reference thresholds in Table [1, 2, 9, 11–13].

As complementary tests in selected cases, an upper GI contrast study (barium transit) or EGD may be considered to document the transition zone and exclude an intraluminal obstruction [11, 12, 14].

The differential diagnosis of high duodenal obstruction includes annular pancreas, congenital membranes/webs, malrotation, Crohn's disease, and neoplasms, which can mimic SMA syndrome [11–13].

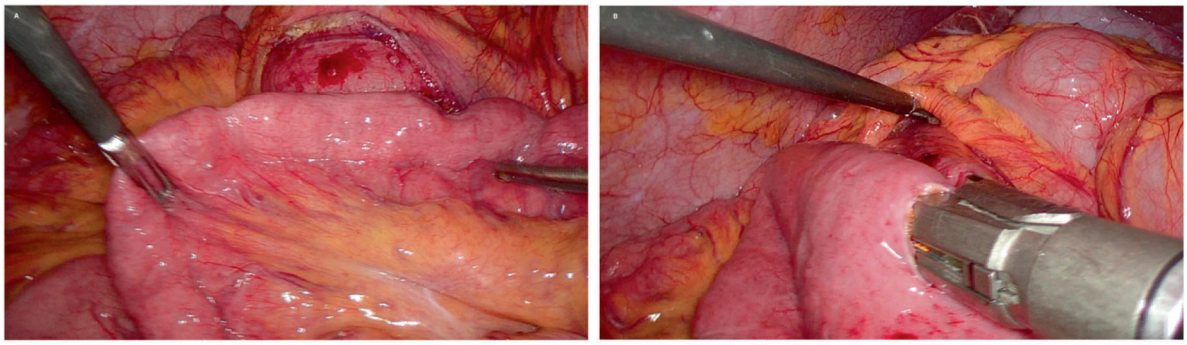
A concomitant nutcracker entity (compression of the left renal vein between the aorta and the SMA) represents a separate spectrum of vascular anomalies. The phenomenon denotes an isolated morphologic finding without clinical manifestations, whereas the syndrome is a symptomatic condition with hematuria, lumbar pain, or signs of venous congestion [3, 4]. Coexistence of SMA syndrome and a nutcracker entity has been reported in single case reports, and the two diagnoses are linked by a narrowed aortomesenteric angle [5–7]. In our case, this was an asymptomatic nutcracker phenomenon without hematuria that did not affect therapeutic decision-making focused on relieving the duodenal obstruction (Fig. 1, *b*).

Diagnostic ambiguity at the initial evaluation of our patient is also noteworthy. The index CT described segmental colitic changes (suspected IBD), which were ruled out by pancolonoscopy and histology. The dynamics of ileus, bowel distension, and reactive mucosal changes can mimic inflammatory bowel disease. Endoscopic verification with biopsy is therefore essential to avoid inappropriate treatment.

Management of SMA syndrome is stepwise. In selected patients, conservative therapy may be initiated (nutritional intervention aimed at restoring the fat pad within the aortomesenteric angle, postprandial positioning, prokinetics, and, if needed, enteral feeding distal to the obstruction). Conservative management works best with shorter symptom duration and lesser dilatation. In practice, a time-limited trial is undertaken with clear nutritional targets (weight/angle increase) and lifestyle measures [1, 2, 11–13]. Persistent symptoms, marked dilatation, or recurrences indicate surgery [1, 2, 11–13]. Because conservative management had failed to provide sustained symptom relief, surgical bypass was considered appropriate in our patient.

Nutritional rehabilitation is central in SMA syndrome because it targets the underlying mechanism: low BMI and loss of retroperitoneal/mesenteric fat reduce the aortomesenteric angle and distance, thereby aggravating duodenal compression, while weight restoration may rebuild the protective «fat cushion» and contribute to symptom improvement. Accordingly, conservative management when feasible is typically built around nutritional therapy (together with postural measures, prokinetics and decompression) and should be guided by explicit, objectively monitored goals focused on weight restoration [1, 2, 11–13].

From a practical standpoint, energy and protein targets can be set pragmatically at approximately 25–30 kcal/kg/day (≈ 105–126 kJ/kg/day) and 1.2–1.5 g protein/kg/day (preferably using ideal body weight in underweight patients), with early re-establishment of oral intake supported by high-energy/high-protein oral nutritional supplements [10, 15]. If oral intake remains inadequate, enteral feeding distal to the obstruction (e. g., nasojejunal/jejunal feeding) should be considered, while parenteral nutrition is reserved for intolerance or failure of enteral strategies [10, 15]. In markedly underweight patients or after prolonged poor intake, cautious caloric escalation with monitoring and correction of phosphate, potassium and magnesium (and con-



a *b*

Fig. 3. Intraoperative views — duodenojejunostomy.
Note. *a* — jejunum aligned and approximated to the third part of the duodenum at the intended laterolateral anisoperistaltic anastomosis site; *b* — linear stapler positioned intraluminally across the duodenum and jejunum immediately before firing. After construction the enterotomies were oversewn in two layers. Source: author.

sideration of thiamine supplementation according to protocol) is recommended to mitigate refeeding syndrome risk [16]. Even after definitive surgical bypass, continuation of a structured nutritional plan after discharge (energy-dense small frequent meals, oral supplements, and dietitian follow-up) helps secure sustained weight restoration, which was also observed at follow-up in our patient.

Among operative options, duodenojejunostomy (DJ), preferably laparoscopic, is considered the first-line method, as it reliably bypasses the site of compression, provides rapid relief of obstruction, and has a favorable postoperative profile compared with open surgery. The surgical principle of the DJ bypass is illustrated in Fig. 3 [1, 2, 11–13, 17].

Alternatives to the intestinal bypass include Strong's procedure (division of the ligament of Treitz with derotation of the duodenum), which may fail when the duodenum is firmly fixed, and gastrojejunostomy, which does not address the obstruction at D3 and carries risks of bile reflux and blind-loop syndrome. Therefore, it is used only exceptionally [1, 2, 11–13].

Laparoscopic DJ shows high success rates and low morbidity in published series. Long-term outcomes confirm sustained improvement in symptoms and BMI, although patients with concomitant motility disorders may have a poorer prognosis [11, 12, 17]. In our case, given the marked D2 dilatation, unequivocal CT quantification, and patient preference, laparoscopic DJ was chosen. The procedure was performed with minimal blood loss, an operative time of 76 minutes, and no need for conversion. The postoperative course corresponded to a Clavien–Dindo II stage, consistent with common, pharmacologically manageable complications after minimally invasive visceral procedures. Published data support the safety profile of the laparoscopic approach in this indication as well [1, 2, 17].

Transposition of the superior mesenteric artery is a less frequently used alternative in which caudal reimplantation of the SMA increases the aortomesenteric angle and relieves D3 compression. This technically demanding vascular procedure requires appropriate expertise and is considered on a case-by-case basis (e. g., after failure/contraindication of DJ/Strong's, specific anatomy, or concomitant planned vascular reconstruction). In available reviews, SMA transposition is described in small series and case reports and is not regarded as first-line because of its greater complexity compared with DJ [11–13].

When nutcracker syndrome is clinically manifest (hematuria, lumbar pain, venous congestion), options include open transposition of the left renal vein or endovascular stenting of the LRV. The choice depends on age, severity, anatomy, and institutional experience. LRV stenting is a less invasive solution with good short-term outcomes, but it requires antithrombotic therapy and surveillance for thrombosis or stent migration; long-term data are limited [3, 14, 18].

In overlap with SMA syndrome, when duodenal obstruction is treated by DJ bypass (and the patient declines vascular SMA transposition), LRV stenting can be considered for symptomatic nutcracker syndrome as a standalone or staged procedure. Rarely, regression of duodenal compression after LRV stenting has been reported due to altered anatomic relationships [5].

Limitations and strengths of our case. This is a single-case report with a 3-month follow-up and without postoperative re-measurement of aortomesenteric parameters; generalizability is therefore limited. Strengths include quantified diagnostics (angle/distance, Table 1, Fig. 2), a detailed

surgical description with a minimally invasive approach (Fig. 3), and an objective nutritional benefit (BMI + 2.3 kg/m²).

Conclusion

Minimally invasive duodenojejunostomy is a safe and effective definitive option in hemodynamically stable patients with superior mesenteric artery syndrome and clearly confirmed obstruction. Compared to alternatives (Strong's procedure, gastrojejunostomy), it reliably bypasses the site of compression and provides rapid recovery with low morbidity. Concurrent asymptomatic nutcracker phenomenon does not require vascular intervention and does not change the surgical strategy. Based on our case, we support a laparoscopic-first strategy within multidisciplinary decision-making in appropriately selected patients.

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Received 07.10.2025
Accepted 13.05.2026
Online First 08.06.2026