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# Laparoscopic Duodenojejunostomy for Superior Mesenteric Vein Syndrome Associated with Nutcracker Phenomenon: The First Case Report

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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**Conflict of interest:** None declared

**Patient:** Male, 33
**Final Diagnosis:** Superior mesenteric vein syndrome
**Symptoms:** Epigastric pain
**Medication:** —
**Clinical Procedure:** Laparoscopic duodenojejunostomy
**Specialty:** Surgery

**Objective:** Rare co-existance of disease or pathology

**Background:** Duodenal compression between the superior mesenteric vessels and aorta or its branches is a rare disease in which the angle between the superior mesenteric vessels and aorta becomes acute, resulting in duodenal obstruction. Reduction in retroperitoneal fat due to several debilitating conditions is considered to be the cause of the decreased angle between the 2 vessels. Nutcracker phenomenon is the asymptomatic compression of the left renal vein (LRV) between the aorta and the superior mesenteric artery.

**Case Report:** We report the case of a 33-year-old man who presented with postprandial abdominal pain, mainly at the epigastric region, colicky in nature, without radiation, accompanied by nausea, postprandial vomiting, and loss of weight. Computed tomography (CT) of the abdomen showed duodenal compression between the SMV and the right common iliac artery, which has never been reported before. Laparoscopic duodenojejunostomy was performed.

**Conclusions:** Vascular compression of the duodenum presents with manifestations of proximal small bowel obstruction, which may have chronic, intermittent, or acute symptoms. Diagnosis is difficult due to the lack of knowledge of this rare disorder. Most of these symptoms can be present in other diseases, and symptoms sometimes do not correspond with imaging findings. Therefore, for a better outcome, the clinician should have a high index of suspicion and should be able to exclude other causes with similar manifestations.

**MeSH Keywords:** Duodenal Obstruction • Mesenteric Veins • Renal Nutcracker Syndrome

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## Background

Vascular compression of the duodenum was first described by Rokitansky in 1861, but it was due to the superior mesenteric artery (SMA) [1,2]. The syndrome usually develops after rapid weight loss. The most characteristic symptoms are postprandial upper-abdominal fullness with pain, belching, and bilious emesis. It commonly results from increased pressure at the junction between the third and fourth part of the duodenum between the aorta and the superior mesenteric vessels [3]. As the diagnosis is generally challenging, it is mandatory to rule out other causes that lead to duodenal obstruction. This syndrome can present with an acute onset, but the patient usually has a long-term history of symptoms [2]. The initial treatment is conservative, consisting of gastrointestinal decompression, correction of electrolyte abnormalities, and nutritional support using either nasojejunal feeding or total parenteral nutrition (TPN) [4]. The surgical management of superior mesenteric vessels (SMVs) syndrome is indicated in medically resistant cases. The surgical options are Strong's procedure, gastrojejunostomy, and duodenojejunostomy [5]. Laparoscopic duodenojejunostomy is the surgical option of choice. The 5-year success rate of duodenojejunostomy is over 90% [6].

## Case Report

A 33-year-old asthenic man with a BMI of 14 kg/m$^2$ presented with the complaint of recurrent abdominal pain, epigastric pain fullness, vomiting, and weight loss. His body mass index decreased from 18 kg/m$^2$ to 14 kg/m$^2$ in the last 6 months, without any apparent medical cause for this BMI. The pain was colicky in nature for 6 months. It was precipitated by eating and was relieved after attacks of bilious vomiting. He had no change in his bowel movements. His medical and surgical history was negative, with no history of allergies. He had been previously admitted at another hospital and received conservative treatment in the form of gastrointestinal decompression via nasogastric tube, intravenous fluid, correction of electrolyte disturbances, and enteral nutrition for 10 days. After what, he was referred to our hospital.

His vital signs were normal. An abdominal examination found no significant clinical findings. Routine blood tests showed hypokalemia (K: 3.2 mEq\L) and hyponatremia (Na: 129 mEq\L). Urine examination was normal. Ultrasonography (USG) of the abdomen was normal. Contrast-enhanced computed tomography (CECT) showed that the aortic bifurcation was high, at the level of the upper border of the third lumbar vertebra (Figure 1), with compression of the junction of the third and fourth part of the duodenum between the superior mesenteric vein and the right common iliac artery, with mild duodenal dilatation (Figure 2). The left renal vein was compressed between the superior mesenteric artery and the aorta (nutcracker phenomenon) (Figure 3). Upper GIT endoscopy was done and showed dilatation of the second part of the duodenum, with gastritis and duodenitis (Figure 4). The patient was admitted to the surgical ward. A nasogastric tube was inserted for decompression, correction of electrolyte abnormalities was done, and he received TPN for 2 weeks before surgery. Informed consent was then obtained from the patient for a laparoscopic duodenojejunostomy. Intraoperatively, we found duodenal compression of the third part of the duodenum (Figure 5). After mobilization of the third part of the duodenum, side-to-side duodenojejunostomy

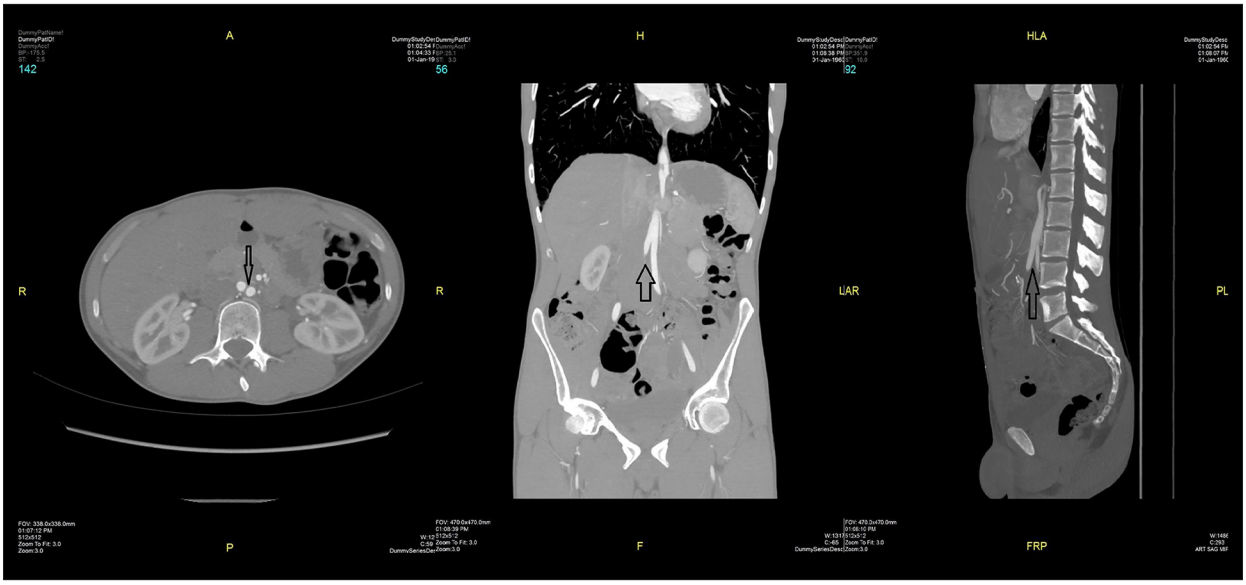


**Figure 1.** Computed tomography of the abdomen showed high bifurcation of the aorta at the upper border of the third lumbar vertebra (arrow) from axial, coronal, and sagittal views (from left to right).



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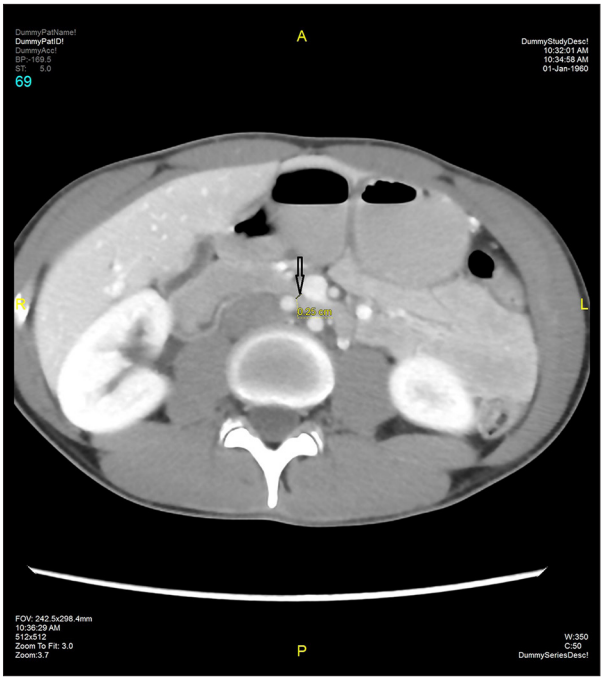


**Figure 2.** Computed tomography of the abdomen showed compression of the third part of the duodenum between the SMV and the right common iliac artery (arrow), with distance between vessels of 0.25 cm.

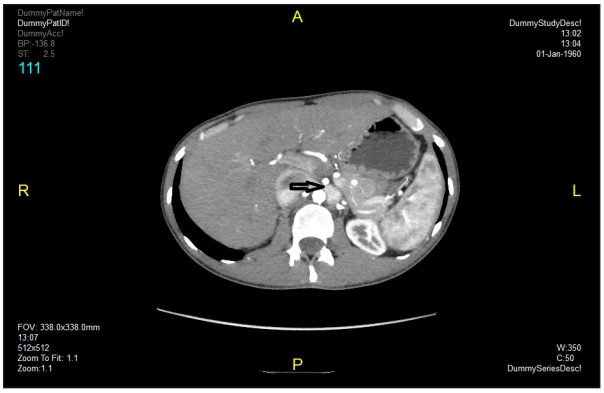


**Figure 3.** Computed tomography of the abdomen showed compression of the left renal vein between the superior mesenteric artery (SMA) and the aorta (arrow).

(Figure 6) was done using an Endo-GIA stapler. His recovery was uneventful, and he was discharged home 4 days after surgery. During this period, a CT (computed tomography) of the abdomen with oral contrast was done to check the anastomosis, which showed no evidence of contrast leak (Figure 7), and, based on this result, oral feeding was started. Daily laboratory investigations produced normal results (Hb: 14 mg\dl, WBCs: 4 $\times 10^9$/L, K: 4.4 mmol/L, Na: 135 mmol/L), so we tapered the TPN. The hospital stay was 18 days. Follow-up after 1 year showed complete resolution of his upper GI symptoms,

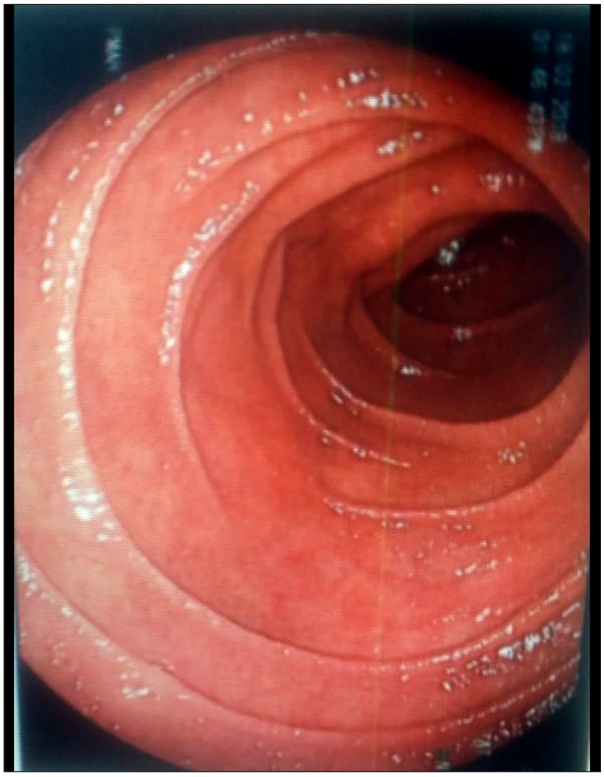

**Figure 4.** Upper gastrointestinal endoscopy showed mild dilatation of the duodenum with duodenitis.

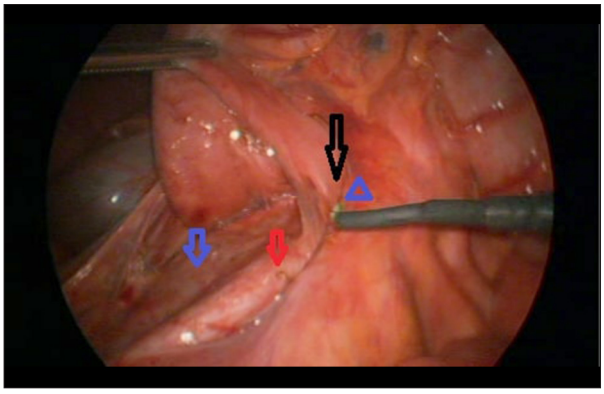

**Figure 5.** Intraoperative finding of the compression of the third part of the duodenum between the superior mesenteric vein (head of arrow) and the right common iliac artery (red arrow). Inferior vena cava (IVC) (blue arrow).

he was able to eat full meals without vomiting, and he gained weight (BMI was 18 kg/m$^2$).

## Discussion

The abdominal aorta is the part of the aorta that starts at the aortic opening of the diaphragm, which lies at the level of the



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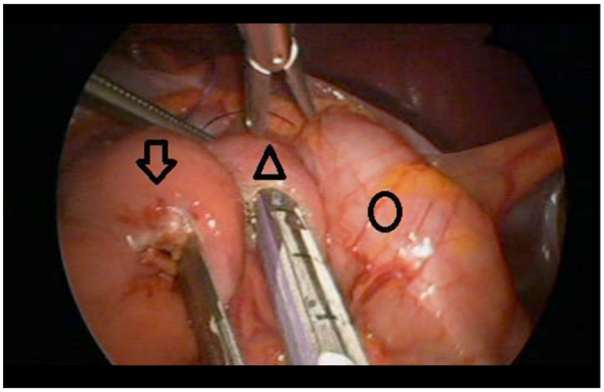

**Figure 6.** Anastomosis between the third part of the duodenum (head of arrow) and the proximal jejunum (arrow). Transverse colon (circle).

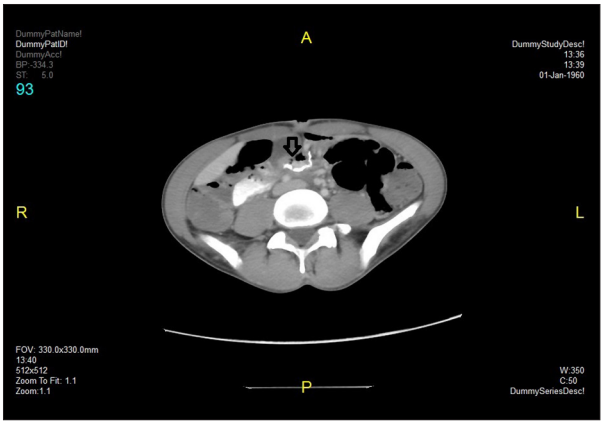


**Figure 7.** Computed tomography (CT) of the abdomen after surgery, showing intact anastomosis (arrow) without evidence of leak.

twelfth lumbar vertebra. It descends in front of the lumbar vertebrae, ending at the lower border of the fourth lumbar vertebra by dividing into 2 common iliac arteries. The abdominal aorta is similar to the thoracic aorta; it has visceral and parietal branches [1]. In our case, the abdominal aorta was divided at a higher level, at the upper border of the third lumbar vertebra.

The superior mesenteric artery (SMA) is one of the major branches of the aorta. It arises from its anterior aspect about 1 cm below the origin of the celiac trunk at the level of the disc between the first and the second lumbar vertebrae [1]. The superior mesenteric vein (SMV) is the vein that drains the blood from the small bowel; it lies slightly anterior and on the right side of the SMA. It has 2 tributaries at the level of the mesentery of the transverse colon: the gastrocolic vein (trunk of Henle) and middle colic vein [2]. In our case, there was no variation in the origin or the course of the SMA, except for increased pressure in the third part of the duodenum between the SMV and the right common iliac artery, which is extremely rare.

The left renal vein drains blood from the left kidney. It is 2–3 times longer than the right renal vein; it passes anterior to the aorta and ends in the inferior vena cava (IVC) [3]. In our case, the left renal vein was entrapped between the SMA and the aorta, leading to the nutcracker syndrome.

The angle between the aorta and the superior mesenteric vessels is preserved by several structures, including the left renal vein passing in front of the aorta, the uncinate process of the pancreas, retroperitoneal fat, and lymphatics. The superior mesenteric vessels pass over the third part of the duodenum, which is anchored within the aortomesenteric angle by the Treitz ligament. The standard distance between the SMA and the aorta is 10–28 mm, and the angle between them is 38–65 degrees [4,5].

Duodenojejunostomy was first described as a treatment for vascular compression of the duodenum by Bloodgood in 1907 [6]. The first duodenojejunostomy was done by Stavely in 1908 and was open [7]. In 1921, Wilkie reported that duodenojejunostomy was the treatment of choice for superior mesenteric artery syndrome (cast syndrome) and described it as chronic duodenal ileus, which is considered now is a misnomer [8].

The compression of the duodenum on its third part resulting from loss of retroperitoneal fat reduces the angle between the superior mesenteric vessels and aorta. There are several precipitating factors that lead to the reduction in this angle, including significant weight loss, which results from malabsorption syndrome, malignancy, acquired immune deficiency syndrome (AIDS) [9–11], burns [12], trauma [13], morbid obesity surgery [14–17], spinal cord injury, paraplegia, [18], drug abuse [19], prolonged bed rest, and anorexia nervosa [20–23].

The clinical presentation of vascular compression of the duodenum may be acute (e.g., after surgery) or chronic with progressive symptoms. In both scenarios, the symptoms are due to the proximal small bowel obstruction. The presentation depends on the degree of obstruction; patients with a mild degree of obstruction may present with only epigastric pain, mainly after a meal (postprandial abdominal pain), while patients with a marked degree of obstruction may develop nausea, bilious vomiting, and weight loss [24].

The pain may improve when the patient is lying in prone, lateral decubitus, or knee-chest positions because these positions release the pressure from the mesentery and superior mesenteric vessels, increasing the angle between the vessels and the aorta or common iliac vessels [25], as seen in our patient.

Delayed diagnosis can lead to serious complications, including electrolyte disturbances, emphysematous gastritis with gas in the portal vein, gastric perforation, and formation of



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gastric and duodenal bezoar, which can lead to gastric outlet obstruction [26,27].

The diagnosis of vascular compression of the duodenum is difficult because most symptoms are nonspecific and diagnosis based mainly on the exclusion of other causes of epigastric pain with small bowel obstruction. Upper gastrointestinal study with oral contrast will usually reveal significant delay in contrast passage from the duodenum to the distal small bowel, with sharp cutoff of contrast at the third part of the duodenum, in addition to dilatation of the stomach and the duodenum, with retention of contrast for long periods [24]. Upper endoscopy is essential to rule out other causes of duodenal obstruction.

Computed tomography (CT) is essential in the diagnosis of vascular compression of the duodenum. It can provide information regarding diagnoses, such as angle and distance between the superior mesenteric vessels and aorta or its division, or any vascular anomalies, such as assessment of retroperitoneal and intrabdominal fat, as in our case [28]. The essential criteria for diagnosis of vascular compression of the duodenum are: obstructed duodenum with cutoff sign at the third part of it, the angle between the superior mesenteric vessels and aorta or common iliac vessels ≤25°, the distance should be less than 8 mm, and the duodenum should be fixed at a higher level by the ligament of Trietz [24,29,30].

Management of vascular compression of the duodenum includes conservative and surgical treatment. The aim of conservative treatment is the relief of obstructive symptoms and the elimination of any predisposing factors such as weight loss. Conservative treatment includes gastrointestinal decompression via nasogastric tube (NGT), correction of electrolyte disturbances, positioning of the patient in left lateral decubitus, prone, or knee-to-chest positions following a meal, and adequate nutritional support [31]. Nutritional support includes enteral feeding via a nasojejunal tube inserted distal to the third part of the duodenum. In certain circumstances, both enteral and parenteral feeding are required for better nutritional supplementation [32]. Conservative measures should be established at least 4–6 weeks before surgical treatment.

In adults with short-term history of symptoms, and in pediatric patients who present in an acute setting, initial conservative treatment with nutritional support has shown a good outcome. Nutritional support is likely to add benefit in adult patients with chronic symptoms; for these patients, surgical treatment is recommended after gastrointestinal decompression, correction of electrolyte abnormalities, and a short course of nutritional support [12,33,34].

Surgical intervention is indicated when the conservative treatment fails to alleviate symptoms, especially in patients with long history, progressive weight loss, marked dilatation of the duodenum with stasis, and complications such as peptic ulcer. The surgical options are either bypass surgery such as duodenojejunostomy or gastrojejunostomy to bypass obstruction or Strong procedure (duodenal derotation procedure), aiming for placement of the third and the fourth part of the duodenum on the right side of the superior mesenteric vessels [35].

A successful laparoscopic approach for duodenojejunostomy and Strong procedure has been reported; it provides a less invasive surgical approach [36–38]. Follow-up with a contrast study is recommended after surgery to ensure patency of the anastomosis and normal duodenal emptying. Patients should be followed regarding resolution of symptoms and weight gain.

Hmadeh et al. (2018) reported laparoscopic liberation of the duodenum and terminal ileum in a 30-year-old male patient who presented with recurrent abdominal pain; CT abdomen showed midgut malrotation and duodenal obstruction by normal displaced appendix [39].

Nutcracker phenomenon (NCP) is the asymptomatic compression of the left renal vein between the aorta and the superior mesenteric artery (SMA), which impairs the outflow of blood from the kidney, with dilatation of the distal part of the renal vein [40]. Nutcracker syndrome is the symptomatic compression of the left renal vein between the same vessels associated with the nutcracker morphologic features [41]. In our case, the patient was asymptomatic.

Peterson et al. (2017) noted an incidental anatomic finding of a celiacomesenteric trunk (CMT) type 1-b associated with compression of the left renal vein (nutcracker phenomenon) in a 91-year-old male patient who underwent cadaveric examination [42]. Furthermore, Siddiqui et al. (2017) reported 3 cases of left renal vein compression syndrome: 2 cases underwent intervention and 1 was treated conservatively [43].

There is no difference in causes, precipitating factors, clinical presentation, methods of investigation, or management between duodenal compression due to either superior mesenteric artery or vein. In our case, the compression was between the SMV and right common iliac artery, which makes this the first case reported from an anatomical point of view.

## Conclusions

Vascular compression of the duodenum is a rare disease resulting from a variety of causes that results in weight loss. Diagnosis is challenging and requires a high index of suspicion. CT of the abdomen is needed for diagnosis of the narrowing angle and decrease in distance between the compressing



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vessels, either between the superior mesenteric artery and aorta or superior mesenteric vein and right common iliac artery, as seen in our case. Duodenojejunostomy is the most commonly used surgical procedure for the management of vascular compression of the duodenum.

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**Conflicts of interest**

None.



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