Indian Journal of
Applied Radiology

Open Science Publications

Volume 11, Issue 1 - 2025

www.opensciencepublications.com

Compressed and Overlooked: A Radiologic-Clinical Overview of Abdominal Vascular Compression Syndromes

Research Article

Akhil. M. Kulkarni[1,2], Pranavi Kondaveeti[2], and Suhasini Vittal Rao[1]*

[1]*Consultant fetal medicine specialist, Davangere scan centre, Davangere, Karnataka, India.*

[2]*Department of Radiology, SSIMS & RC, Davangere, Karnataka, India.*

***Corresponding author:** Suhasini Vittal Rao,Consultant fetal medicine specialist, Davangere scan centre, Davangere, Karnataka, India, E-mail: drsuhasini2010@gmail.com

Article Information: **Submission: 16/06/2025; Accepted: 05/08/2025; Published: 06/08/2025**

Abstract

Abdominal vascular compression syndromes constitute a collection of infrequent but clinically important conditions caused by external compression of abdominal vessels or by vascular structures exerting pressure on hollow organs. Key syndromes include Median Arcuate Ligament Syndrome (MALS), Nutcracker Syndrome (NCS), Superior Mesenteric Artery (SMA) syndrome, SMA-like syndrome, Retrocaval Ureter & Portal Biliopathy. These conditions are often underrecognized, as they can present with vague abdominal or urinary symptoms, while many affected individuals remain symptom-free. This review focuses on essential CT imaging findings and clinical correlations that assist in diagnosing symptomatic patients.

Keywords: Vascular Compression Syndromes; MALS; Nutcracker Syndrome; SMA Syndrome; SMA Like Syndrome; Retrocaval Ureter; Portal Biliopathy

Introduction

Abdominal vascular structures may be subjected to compression by surrounding anatomical elements, or conversely, may compress adjacent hollow organs. The resulting symptoms tend to be nonspecific, leading frequently to diagnostic delays or misdiagnoses. Although these syndromes have been recognized for several decades, they remain insufficiently understood. Untreated cases risk significant complications. Because these conditions span multiple specialties, they present substantial diagnostic challenges, underscoring the need for heightened clinical awareness and recognition [1].

Anatomical variants or morphological changes predisposing to vascular compression are sometimes incidentally detected in asymptomatic patients undergoing imaging for unrelated reasons. Therefore, clinical correlation is critical to avoid overdiagnosis, and imaging findings alone should not be the basis for diagnosis [1].

Non-invasive imaging techniques like ultrasound, computed tomography (CT), and magnetic resonance imaging (MRI) are useful in identifying vascular compression syndromes affecting the abdomen and pelvis. Of these, contrast-enhanced CT is typically the modality of choice when clinical suspicion arises, owing to its ability to clearly depict vascular structures and their anatomical relationships with surrounding tissues [1,2].

This review outlines the underlying pathophysiology and clinical manifestations of various abdominal vascular compression syndromes, emphasizing key CT imaging findings that assist in establishing a precise diagnosis.

Median arcuate ligament syndrome

Median Arcuate Ligament Syndrome (MALS), also referred to as Dunbar syndrome or celiac artery compression syndrome, was first described by Harjola in 1963 [1,3]. This condition arises from a

ISSN: 2581-3919

fibrous arch known as the median arcuate ligament, which connects the diaphragmatic crura at the T12–L1 vertebral level and usually courses above the origin of the celiac artery [4]

In 10–24% of asymptomatic individuals, this ligament crosses anteriorly over the proximal celiac artery [5]. While many such compressions are clinically silent, a subset results in sufficient arterial stenosis to impede blood flow and provoke symptoms [4].

Clinical features

This syndrome most often affects individuals between 20 and 40 years of age, typically women with a lean body build [6]. The main symptoms are chronic epigastric discomfort after meals, nausea, and weight loss-signs of intermittent compression of the celiac artery [1]. Nonetheless, nearly 85% of those with this anatomical pattern do not exhibit symptoms, and findings are often discovered incidentally during CT imaging for unrelated issues [1,7,8] (Figure 2).

CT imaging findings

Multidetector CT (MDCT), due to its high spatial resolution, enables clear visualization of the median arcuate ligament—thickness beyond 4 mm is considered abnormal [8] (Figure 1B). Since axial images may not reveal the full extent of compression, sagittal reconstructions are essential to assess both the ligament and celiac artery origin. A distinctive hooked or "J-shaped" narrowing at the origin of the celiac artery (Figure1A) serves as a key imaging sign distinguishing MALS from atherosclerotic changes. The compression is often persistent, even during inspiration. Additional findings may include post-stenotic dilatation and development of collateral vessels such as the pancreaticoduodenal arcade from the superior mesenteric artery [9].

Management

Symptomatic cases of MALS are usually treated by surgically releasing the median arcuate ligament, sometimes accompanied by celiac ganglion removal or vascular bypass procedures. These interventions generally lead to long-term relief from symptoms [10].

Nutcracker Syndrome

Nutcracker Syndrome refers to the entrapment of the left renal vein (LRV), most commonly between the superior mesenteric artery and the aorta, known as the "anterior nutcracker" variant. Less frequently, compression occurs between the aorta and the vertebral column when the LRV follows a retro-aortic or circum-aortic course—termed the "posterior nutcracker" [1].

It primarily affects otherwise healthy, thin women in their third and fourth decade of life. The anterior variant is more common, whereas the posterior variant (Figure 5) is rare and its clinical relevance remains unclear [10].

Clinical features

Nutcracker syndrome often presents in young to middle-aged adults, with women being more frequently affected. Because diagnostic criteria are not well standardized, diagnosis is frequently missed or delayed. Symptoms result from increased venous pressure in the LRV and may be exacerbated by physical activity. Manifestations range from microscopic haematuria to visible haematuria leading

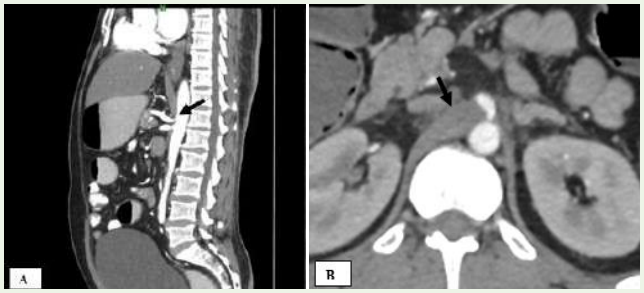

Figure 1A and B: A 49-year-old man presenting with abdominal pain after meals. (A) Sagittal contrast-enhanced CT angiogram of the abdomen reveals a prominent median arcuate ligament (black arrow), causing a hooked deformity and narrowing at the proximal celiac artery with post-stenotic dilatation. (B) Axial contrast-enhanced CT angiogram demonstrates the celiac artery origin being compressed by the thickened median arcuate ligament (black arrow).

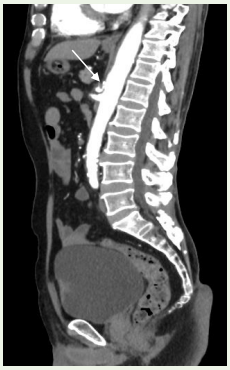

Figure 2: A 77-year-old man evaluated for haematuria and diagnosed with bladder carcinoma (white arrow). Incidentally noted on sagittal contrast-enhanced CT angiography is a kinked proximal celiac artery with a hooked contour and associated post-stenotic dilatation.

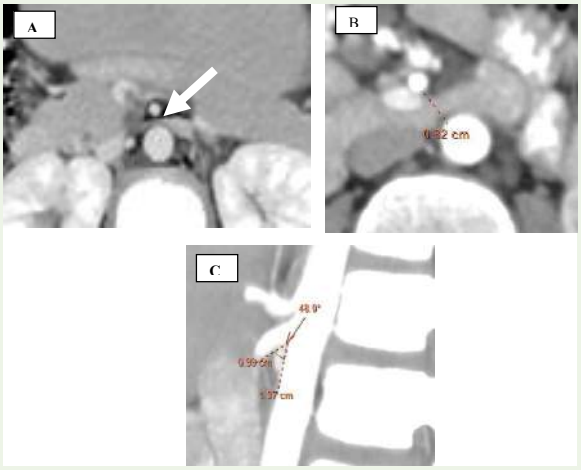

Figure 3 A-C: A 17-year-old male presenting with swelling in the left scrotal region. (A) Axial contrast-enhanced CT image demonstrates the characteristic "beak sign" (white solid arrow) due to narrowing of the left renal vein. (B) Axial CT showing a reduced aortomesenteric distance (AMD) measuring approximately 8 mm. (C) Sagittal contrast-enhanced CT reveals a sharply decreased aortomesenteric angle (AMA) of around 48°.

Citation: Akhil MK, Pranavi K, Suhasini VR. Compressed and Overlooked: A Radiologic-Clinical Overview of Abdominal Vascular Compression Syndromes. Indian J Appl Radiol. 2025;11(1): 216.

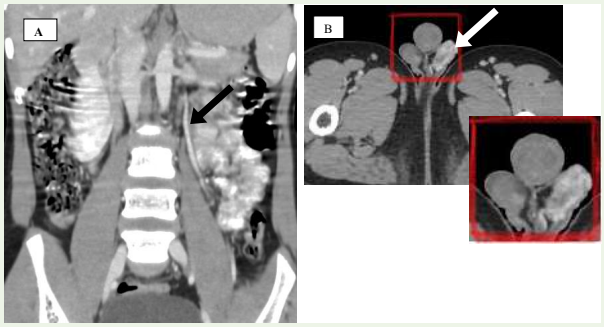

Figure 4 A, B: (A) Coronal contrast-enhanced CT image displays a prominently dilated left testicular vein (black arrow). (B) Axial contrast-enhanced CT scan of the scrotum reveals a cluster of tortuous, enhancing veins within the left scrotal sac (white solid arrow), consistent with a left-sided varicocele.

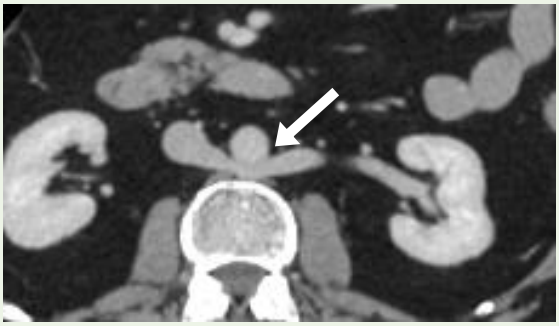

Figure 5 Axial contrast-enhanced CT of a 33-year-old woman demonstrates a retro-aortic course of the left renal vein (white arrow), an incidental finding consistent with the posterior variant of Nutcracker Syndrome.

to anaemia, orthostatic proteinuria, and pain in the left flank due to passage of clots. Reflux into the gonadal vein may lead to left-sided varicocele in males and varicose veins in the pelvis or vulva in females [1].

CT imaging findings

On axial CT, the "beak sign" is seen as abrupt narrowing of the LRV at the point where it passes between the SMA and aorta, forming an acute angle (Figure 3A) [11,12]. Sagittal reconstructions help assess the aortomesenteric angle (AMA), which normally ranges from 38°–56°; angles under 35° are suggestive of the syndrome [13,14]. Affected individuals also demonstrate a reduced aortomesenteric distance (AMD), typically narrowed from the normal 10–28 mm to 2–8 mm [1,15] (Figure 3B), (Figure 3C). A pre- to post-compression LRV diameter ratio exceeding 2.25 has been shown to yield 91% sensitivity and specificity for diagnosis [16]. CT may also reveal dilated gonadal veins and pelvic varicosities, although it cannot assess blood flow velocity or direction [11] (Figure 4A), (Figure 4B).

Management

Management of mild or asymptomatic cases is conservative, focusing on observation. More severe cases require intervention to relieve venous obstruction and hypertension. Surgical options include LRV transposition, external venous stenting, bypass procedures, renal auto-transplantation, or nephrectomy [1].

Superior Mesenteric Artery Syndrome

Superior Mesenteric Artery (SMA) syndrome, first documented by Rokitansky in 1842, results from compression of the third part of the duodenum between the SMA and the aorta, leading to duodenal obstruction [1,13].

A hallmark diagnostic feature of SMA syndrome is the narrowing of the angle between the SMA and the aorta, which normally measures between 38° and 65°. In affected individuals, this angle is significantly reduced. Likewise, the aortomesenteric distance at the level where the duodenum crosses is typically reduced from its normal range of 10–28 mm [13,17,18].

Clinical features

Patients typically show signs of upper gastrointestinal obstruction such as abdominal pain following meals, nausea, vomiting, and unintended weight loss—symptoms linked to delayed gastric emptying. Other potential causes must be excluded, and diagnosis relies on identifying the anatomical abnormality using imaging studies [19].

Imaging findings

On CT, the AMA is decreased (6°–22°) and the AMD reduced (2–8 mm) (Figure7A), (Figure 7B). Characteristic findings include gastric and proximal duodenal dilation (Figure 6A), (Figure 6B) with abrupt narrowing at the compression site. CT helps localize the obstruction and exclude other causes. Importantly, decreased AMA and AMD without symptoms do not suffice for diagnosis [20, 21, 22].

Management

The first-line approach involves conservative therapy, including fluid resuscitation, electrolyte correction, and nutritional support-often through nasojejunal feeding. Patients are advised to consume small, frequent meals and may benefit from positional adjustments like lying prone or in the left lateral decubitus position. If non-surgical methods fail, operative options such as duodenojejunostomy, gastrojejunostomy, or division of the ligament of Treitz with bowel derotation (Strong's procedure) may be considered [1,13,21].

Superior mesentery artery-like syndrome

SMA-like syndrome is a newly described condition that mimics the presentation of SMA syndrome but results from compression of the duodenum by vascular structures other than the SMA and aorta. These may include mesenteric or retroperitoneal arteries and veins [23].

Clinical features

This syndrome clinically mimics SMA syndrome and carries similar risks if untreated [23].

Imaging findings

On imaging, compression of the third portion of the duodenum may be observed between multiple anterior (mesenteric) and posterior vascular structures. Anatomical variations in the suspensory ligament of the duodenum may contribute to this phenomenon. CT scans may reveal either duodenal compression leading to obstruction (Figure 8A), (Figure 8B) or isolated vascular compression without evidence

Citation: Akhil MK, Pranavi K, Suhasini VR. Compressed and Overlooked: A Radiologic-Clinical Overview of Abdominal Vascular Compression Syndromes. Indian J Appl Radiol. 2025;11(1): 216.

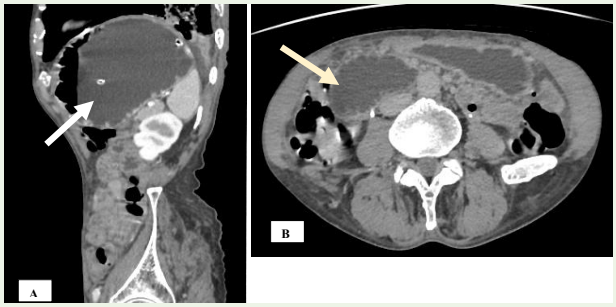

Figure 6 A, B: A 76-year-old man presenting with postprandial abdominal discomfort, vomiting, and a history of weight loss. (A) Sagittal contrast-enhanced CT of the abdomen shows a markedly distended stomach (white arrow). (B) Axial contrast-enhanced CT angiogram reveals dilatation of the first and second portions of the duodenum (yellow arrow) due to compression of the third part by the superior mesenteric artery and aorta.

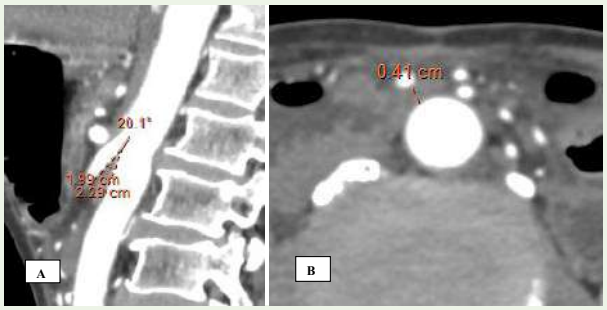

Figure 7 A and B: (A) Sagittal contrast-enhanced CT demonstrates a sharply reduced angle between the superior mesenteric artery and aorta (AMA ~ 20°). (B) Axial contrast-enhanced CT image shows a decreased aortomesenteric distance (AMD) measuring approximately 4 mm.

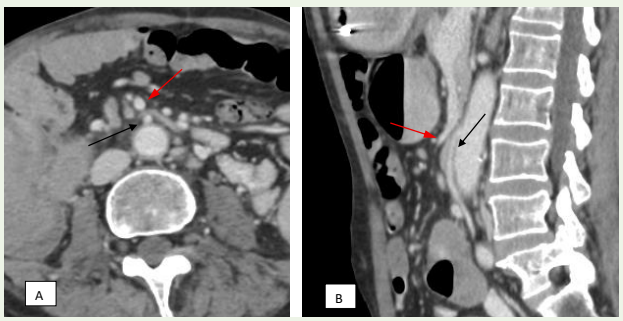

Figure 8 A,B: A 50-year-old man presenting with abdominal pain and vomiting. (A & B) Axial and sagittal contrast-enhanced CT images of the abdomen show compression of the third portion of the duodenum by the ileocolic vein (red arrow) and the inferior mesenteric artery (black arrow), resulting in gastric overdistension due to stagnant intraluminal contents—findings consistent with SMA-like Syndrome.

of obstruction (Figure 9A) (Figure 9B). CT imaging is particularly helpful in identifying the precise location of the duodenum and ruling out alternative causes of obstruction [21, 24].

Management

Management begins conservatively, focusing on nutritional support and hyperalimentation to restore retroperitoneal fat.

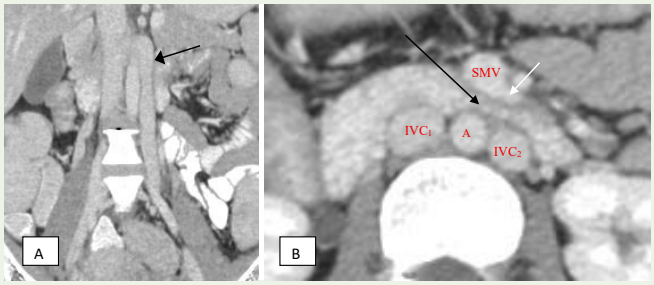

Figure 9 A,B: A 29-year-old woman with complaints of indigestion, vomiting, and weight loss. (A) Coronal contrast-enhanced CT scan of the abdomen demonstrates a duplicated inferior vena cava (IVC) on the left side (black arrow). (B) Contrast-enhanced axial CT of abdomen shows compression of 3rd part of duodenum (black arrow) by aorta (A), duplicated IVC (IVC_2) and SMV & its jejunal tributary (black arrow) at the level of L3 vertebra with no signs of bowel obstruction—SMA-Like Syndrome.

Surgery (gastrojejunostomy or duodenojejunostomy) is considered if symptoms persist [23, 25].

Vascular Compression of the Ureter—Retrocaval Ureter

The ureter may rarely follow a retrovascular path, such as retroiliac or retrocaval routes, which can lead to compression [26]. Retrocaval ureter is a congenital anomaly caused by abnormal embryological development of the inferior vena cava (IVC) [27]. Under typical development, the intrarenal segment of the IVC originates from the right supracardinal vein, which lies dorsal to the ureter, maintaining the ureter's lateral position. However, in retrocaval ureter, the ventral subcardinal vein fails to regress, causing the ureter to pass posterior to and encircle the IVC during embryogenesis. This aberrant course results in its compression between the IVC and vertebral column, eventually leading to progressive hydronephrosis [28].

Clinical features

Clinically, patients often report right flank pain, although many remain asymptomatic [28].

Imaging findings

Imaging distinguishes two types:

Type 1: The ureter crosses behind the IVC near L3 with a characteristic "S" or "fish-hook" deformity and hydronephrosis in over 50% of casesalso known as the low loop retrocaval ureter (Figure 10) (Figure 11)

Type 2: The retrocaval segment crosses at the renal pelvis with a sickle-shaped curve and mild hydronephrosis, which is less common [29].

Management

Management is tailored based on symptom intensity and renal function status. For mild, asymptomatic cases, watchful waiting may be appropriate. In symptomatic individuals, surgical correction through ureteroureteral reanastomosis anterior to the IVC—along with removal of the retrocaval segment—is commonly performed and typically produces favorable results [30].

Citation: Akhil MK, Pranavi K, Suhasini VR. Compressed and Overlooked: A Radiologic-Clinical Overview of Abdominal Vascular Compression Syndromes. Indian J Appl Radiol. 2025;11(1): 216.

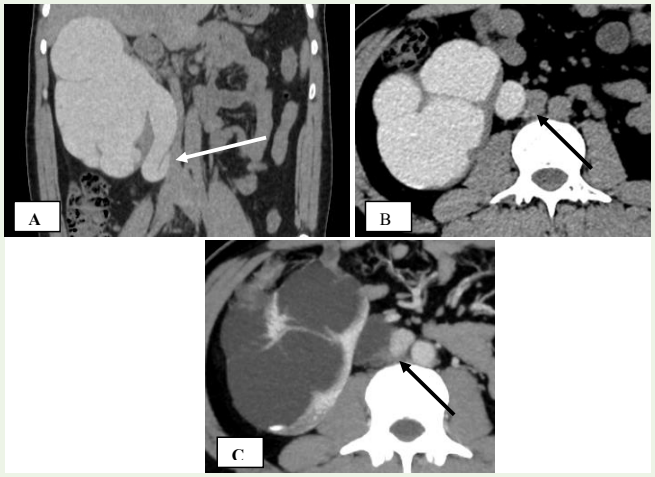

Figure 10 A, B, C: A 26-year-old man with repeated episodes of right flank pain. (A) Coronal reconstructed image from the excretory phase of contrast-enhanced CT IVU reveals right-sided hydronephrosis and a characteristic 'fish-hook' appearance of the upper ureter caused by its retrocaval path (white arrow). (B) and (C) Axial CT images during excretory and nephrogenic phases show the right ureter coursing posterior to the IVC (black arrow).

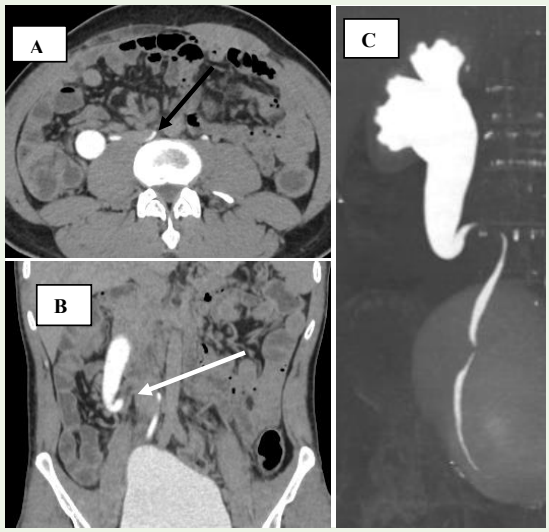

Figure 11 A 28-year-old man presenting with recurrent right flank pain. (A) Axial CT image during the excretory phase demonstrates the retrocaval trajectory of the right ureter (black arrow). (B) Coronal reconstructed image from the excretory phase reveals hydronephrosis with the upper ureter forming a 'fish-hook' configuration due to its retrocaval route (white arrow). (C) A 3D-rendered CT IVU image highlights the same finding—right-sided hydronephrosis and 'fish-hook' shaped ureter consistent with a retrocaval course.

Portal Biliopathy

Portal biliopathy refers to a collection of abnormalities affecting the bile ducts and gallbladder, typically seen in individuals with extrahepatic portal vein obstruction or portal hypertension [31].

The condition arises primarily through two mechanisms: first, by direct mechanical pressure on the biliary tract from a portal cavernoma (Figure 12) and second, through peribiliary fibrosis triggered by inflammation or ischemia following thrombosis in the small venous channels within the bile duct wall [4, 19, 32]. A portal cavernoma is a network of dilated collateral veins surrounding the common bile duct, resulting from cavernous transformation. It is composed of paracholedochal veins (also known as the plexus of Petren) and epicholedochal veins (plexus of Saint), both of which run along the bile duct wall [33].

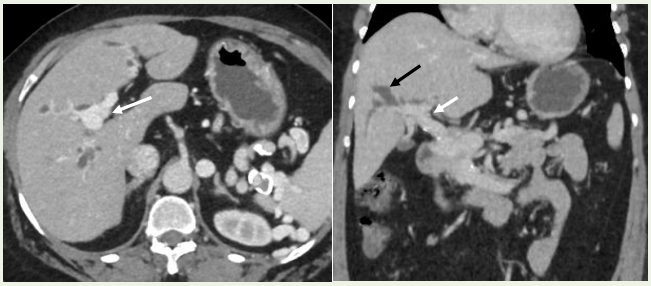

Figure 12 A 55-year-old woman diagnosed with portal hypertension. (A) Axial contrast-enhanced CT image reveals cavernous transformation of the portal vein (white arrow). (B) Coronal contrast-enhanced CT scan shows dilated biliary ducts (black arrow) secondary to external compression from the portal cavernoma (white arrow), indicative of portal biliopathy.

There are three main types of portal biliopathy: varicoid, fibrotic, and mixed. The varicoid form is due to compression and distortion of the bile duct by large external collaterals (paracholedochal veins), while the fibrotic form is associated with thickening and narrowing of the bile duct caused by intramural compression from smaller collateral veins (epicholedochal veins) [4].

Clinical features

Around 70–100% of individuals who exhibit radiologic features of portal biliopathy do not initially experience symptoms [32]. Only a small subset presents with clinical signs such as prolonged cholestasis or jaundice [4, 19, 31].

Imaging findings

Contrast-enhanced CT is typically performed to rule out alternative causes of biliary dilation, especially since portal biliopathy can resemble malignant distal bile duct strictures, such as those from pancreatic cancer or cholangiocarcinoma [34]. The most characteristic radiologic sign, apart from detecting a portal cavernoma, is the abrupt bending or "kinking" of the common bile duct, usually due to external pressure from enlarged paracholedochal veins [35].

Management

While the majority of cases are asymptomatic and do not need active intervention, patients who develop symptoms—especially from biliary strictures—can be treated effectively using endoscopic approaches such as balloon dilatation, stenting, or performing a sphincterotomy [36].

Conclusion

Although often underrecognized, abdominal vascular syndromes constitute an important and frequently overlooked clinical entity. Their presentations can vary widely—from subtle, nonspecific symptoms to severe and obvious clinical signs—necessitating

Citation: Akhil MK, Pranavi K, Suhasini VR. Compressed and Overlooked: A Radiologic-Clinical Overview of Abdominal Vascular Compression Syndromes. Indian J Appl Radiol. 2025;11(1): 216.

	Cause	Clinical Features	CT Findings	Treatment
Median Arcuate Ligament Syndrome (MALS)	Compression of the celiac artery by the median arcuate ligament	Postprandial upper abdominal pain, nausea, weight loss	Hooked contour of celiac artery without atherosclerosis; post-stenotic dilatation; prominent collaterals; pancreaticoduodenal artery aneurysms	Embolization of aneurysms; surgical decompression (ligament release, celiac ganglionectomy); revascularization or bypass procedures
Nutcracker Syndrome (NCS)	Compression of the left renal vein between the aorta and SMA (anterior) or between aorta and spine (posterior)	Hematuria, pelvic varices, gonadal vein reflux	"Beak" sign of LRV; aortomesenteric angle <35°; aortomesenteric distance 2–8 mm	Conservative if mild; surgical options include LRV or gonadal vein transposition, vein patch, LRV stenting, or reimplantation
Superior Mesenteric Artery Syndrome	Duodenal compression between the SMA and aorta	Post-meal abdominal pain, weight loss, nausea, vomiting	Narrow aortomesenteric angle (<22°); distance <8 mm; proximal duodenal dilatation	Nasogastric decompression in mild cases; duodenojejunostomy, gastrojejunostomy, or ligament of Treitz division with bowel derotation for severe or refractory cases
Superior Mesenteric Artery Like Syndrome	Duodenal compression by vessels other than the SMA and aorta	Post-meal abdominal pain, weight loss, nausea, vomiting	Vascular compression of 3rd part of duodenum causing obstruction or isolated compression without obstruction	Conservative in mild cases- nutritional support and hyperalimentation; Surgery -gastrojejunostomy or duodenojejunostomy in severe cases
Vascular Compression of the Ureter—Retrocaval Ureter.	Compression of the ureter when it takes a retro-vascular course—retrocaval.	Right Flank pain, Hematuria	Type 1: The ureter crosses behind the IVC with a characteristic "S" or "fish-hook" deformity and hydronephrosis Type 2: The retrocaval segment crosses at the renal pelvis with a sickle-shaped curve and mild hydronephrosis	Surgical ureteroureteralreanastomosis anterior to the IVC with excision of the retrocaval segment
Portal Biliopathy	Compression of biliary ducts by portal cavernoma	Jaundice, cholestasis, cholangitis.	Dilated bile ducts; tortuous portal collaterals compressing common bile duct; "scalloping" or irregular ductal margins	Endoscopic interventions such as balloon dilatation, stent placement, or sphincterotomy

increased awareness and a collaborative, multidisciplinary approach to diagnosis and management. Advances in imaging modalities, particularly high-resolution CT and magnetic resonance angiography (MRA), have improved early detection, thereby facilitating timely intervention and enhancing patient outcomes. Despite these technological improvements, abdominal vascular syndromes are still commonly misdiagnosed or diagnosed late, due to their complex clinical manifestations and overlap with more prevalent abdominal diseases. Clinicians and radiologists alike should consider these entities in the differential diagnosis when assessing patients with unexplained abdominal pain, weight loss, or similar symptoms. A thorough grasp of the underlying vascular anatomy, associated pathophysiological mechanisms, and key imaging findings is essential for ensuring effective patient care.

Disclosure: The authors have no conflicts of interest to disclose. None of the authors received outside funding for the production of this original manuscript and no part of this article has been previously published elsewhere.

References

1. Lamba R, Tanner D, Sekhon S, McGahan J, Corwin M, et al. (2014) Multi detector CT of vascular compression syndromes in the abdomen andpelvis. Radiographics 34: 93-115.
2. Eliahou R, Sosna J, Bloom AI (2012) Between a rock and a hard place:clinical and imaging features of vascular compression syndromes.Radiographics32: E33-E49.
3. Harjola PT(1963) A rare obstruction of the coeliac artery: report of a case. Ann Chir Gynaecol Fenn 52: 547-550.
4. Srisajjakul S, Prapaisilp P, Bangchokdee S (2017) Imaging features of vascular compression in abdomen: Fantasy, phenomenon, or truesyndrome. Indian J Radiol Imaging 27: 216-224.
5. Horton KM, Talamini MA, Fishman EK (2005) Median arcuate ligamentsyndrome: Evaluation with CT angiography. Radiographics 25: 1177-1182.
6. Dunbar JD, Molnar W, Berman FF, Marble SA (1965) Comparisonof the celiac trunk and abdominal angina. Am J Roentgenol 95: 731-744.
7. Fong JK, Poh AC, Tan AG, Taneja R (2014) Imaging findings and clinicalfeatures of abdominal vascular compression syndromes. AJR Am JRoentgenol 203: 29-36.
8. Heo S, Kim HJ, Kim B, Lee JH, Kim J, et al. (2018) Clinical impact ofcollateral circulation in patients with median arcuate ligament syndrome. DiagnIntervRadiol 24:181-186
9. Jimenez JC, Harlander-Locke M, Dutson EP (2012) Open and laparoscopic treatment of median arcuate ligament syndrome. J VascSurg56: 869-873.
10. Barnes RW, Fleisher HL, Redman JF, Smith JW, Harshfield DL, et al. (1988) Mesoaortic compression of the left renal vein (the so-called nutcracker syndrome): Repair by a new stentingprocedure. J VascSurg 8: 415-421.
11. Ananthan K, Onida S, Davies AH (2017) Nutcracker syndrome: an update oncurrent diagnostic criteria and management guidelines. Eur J VascEndovascSurg 53:886-894.
12. Kim KW, Cho JY, Kim SH,Yoon JH, Kim DS, et al (2011) Diagnostic value of computedtomographic findings of nutcracker syndrome: Correlation with renalvenography and renocaval pressure gradients. Eur J Radiol 80: 648-654
13. Merrett ND, Wilson RB, Cosman P, Biankin AV (2009) Superior mesenteric arterysyndrome: diagnosis and treatment strategies. J GastrointestSurg 13: 287-292.
14. He Y, Wu Z, Chen S, Tian L, Li D, et al (2014) Nutcracker syndrome-How well do weknow it? Urology 83:12-17.
15. Warncke ES, Gursahaney DL, Mascolo M, Dee E (2019) Superior mesentericartery syndrome: a radiographic review. AbdomRadiol (NY) 44: 3188-3194.

Citation: Akhil MK, Pranavi K, Suhasini VR. Compressed and Overlooked: A Radiologic-Clinical Overview of Abdominal Vascular Compression Syndromes. Indian J Appl Radiol. 2025;11(1): 216.

16. Hangge P, Gupta N, Khurana A, Quencer KB, Albadwai H, et al. (2018) Degree of Left Renal Vein Compression Predicts Nutcracker Syndrome. J Clin Med 7: 107

17. Unal B, Aktas A, Kemal G, Bilgili Y, Güliter S, et al. (2005) Superior mesenteric artery syndrome: CT and ultrasonography findings. DiagnIntervRadiol 11: 90-95.

18. Sapkas GO, Brien JP (1981) Vascular compression of the duodenum (cast syndrome) associated with the treatment of spinal deformities. A report of six cases. Arch Orthop Trauma Surg 98: 7-11.

19. Gozzo C, Giambelluca D, Cannella R, Caruana G, Jukna A, et al. (2020) CT imaging findings of abdominopelvic vascular compression syndromes: what the radiologist needs to know. Insights into Imaging 11: 48.

20. Konen E, Amitai M, Apter S, Garniek A, Gayer G, et al. (1998) CT angiography of superior mesenteric artery syndrome. Am J Roentgenol 171:1279-1281.

21. Agrawal GA, Johnson PT, Fishman EK (2007) Multidetector row CT of superior mesenteric artery syndrome. J Clin Gastroenterol 41: 62-65.

22. Srisajjakul S, Prapaisilp P, Bangchokdee S (2017). Imaging features of vascular compression in abdomen: Fantasy, phenomenon, or true syndrome. The Indian Journal of Radiology and Imaging, 27: 216-224.

23. Andour H, Naggar A, Khatibi W, Zahi H, Imrani K, et al. (2024) Duodenal compressions from non-superior mesentery artery structures in the same patient causing superior mesentery artery-like syndrome: a rare case report. BJR Case Reports 10: uaae037.

24. Dross PE, Molavi S, Chan A, Latshaw R, Chhabra P (2019) Unusual etiologies for vascular duodenal compression mimicking the superior mesenteric artery (SMA) syndrome: The SMA-like syndrome. Journal of Gastrointestinal and Abdominal Radiology 02: 140-146.

25. Shin J, Shin PJ, Bartolotta RJ (2018) SMA-like syndrome with variant mesenteric venous anatomy. Clin Imaging 48:86-89.

26. Brito RR, Zulian R, Albuquerque J, Borges HJ (1973) Retrocaval ureter. Br J Urol 45:144-152.

27. Lautin EM, Haramati N, Frager D, Friedman AC, Gold K, et al. (1988) CT diagnosis of circumcaval ureter. AJR Am J Roentgenol 150: 591-594.

28. Perimenis P, Gyftopoulos K, Athanasopoulos A, Pastromas V, Barbalias G (2022) Retrocaval ureter and associated abnormalities. Int Urol Nephrol 33: 19-22.

29. Bateson EM, Atkinson D (1969) Circumcaval ureter: A new classification. Clin Radiol 20: 173-177.

30. Uthappa MC, Anthony D, Allen C (2002) Case report: Retrocaval ureter: MR appearances. Br J Radiol. 75: 177-179.

31. Shin SM, Kim S, Lee JW, Kim CW, Lee TH, et al. (2007) Biliary abnormalities associated with portal biliopathy: Evaluation on MR cholangiography. AJR Am J Roentgenol 188: W341-W347.

32. Moomjian LN, Winks SG (2017) Portal cavernoma cholangiopathy: diagnosis, imaging, and intervention. AbdomRadiol (NY) 42: 57-68.

33. Chandra R, Kapoor D, Tharakan A, Chaudhary A, Sarin SK (2001) Portal biliopathy. J Gastroenterol Hepatol 16:1086-1092.

34. Dhiman RK, Saraswat VA, Valla DC,Chawla Y, Behera A, et al (2014) Portal cavernoma cholangiopathy: consensus statement of a working party of the Indian national association for study of the liver. J Clin Exp Hepatol 4: S2-S14.

35. Jabeen S, Robbani I, Choh NA, Ashraf O, Shaheen F, et al (2016) Spectrum of biliary abnormalities in portal cavernoma cholangiopathy (PCC) secondary to idiopathic extrahepatic portal vein obstruction (EHPVO)—a prospective magnetic resonance cholangiopancreaticography (MRCP) based study. Br J Radiol 89: 20160636.

36. Bhatia V, Jain AK, Sarin SK (1995) Choledocholithiasis associated with portal biliopathy in patients with extrahepatic portal vein obstruction: Management with endoscopic sphincterotomy. Gastrointest Endosc 42: 178-181.

Citation: Akhil MK, Pranavi K, Suhasini VR. Compressed and Overlooked: A Radiologic-Clinical Overview of Abdominal Vascular Compression Syndromes. Indian J Appl Radiol. 2025;11(1): 216.