

Imaging features of vascular compression in abdomen: Fantasy, phenomenon, or true syndrome

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Abstract

Vascular structures in the abdomen can compress or be compressed by adjacent structures. Classic imaging findings of vascular compressions, including median arcuate ligament syndrome, superior mesenteric artery syndrome, nutcracker syndrome, portal biliopathy, May-Thurner syndrome, and ureteropelvic junction obstruction will be discussed here. It is important to correlate imaging findings and clinical data to identify asymptomatic vascular compression which requires no treatment, intermittent vascular compression with nonspecific or vague clinical manifestation, and the subset of patients with true syndromes who will benefit from treatment.

Key words: Imaging of vascular compression; imaging of vascular compression syndrome; vascular compression syndrome

Introduction

Vascular structures in the abdomen can compress or be compressed by adjacent structures. Such compressions may be incidentally found and be asymptomatic or may result in clinical syndromes. Intermittent vascular compression by anatomic abnormalities are more common than vascular compression syndromes. It is important to avoid over diagnosis or treat patients who are incidentally detected to have anatomical factors that predispose to vascular compression but are otherwise asymptomatic.^[1] Hence, the diagnosis of vascular compression syndromes should be made on the basis of both clinical and radiologic features. Although many of these conditions were discussed many years ago, they remain poorly understood. In some circumstances, they can be associated with significant

morbidity and poor treatment outcome if left untreated and unrecognized. Moreover, these conditions disproportionately affect the young and otherwise healthy patients who are most likely to derive long-term benefit from effective and prompt treatment.^[2] Imaging options allow precise definition of abnormal anatomy and prediction of hemodynamic significance, providing a roadmap prior to treatment.^[3] Doppler ultrasound is in general the preferred first test, with a major role in young patients to reduce radiation exposure and display real-time imaging findings on the hemodynamic significance of vascular compression in particular nutcracker syndrome and median arcuate ligament syndrome;^[4] but the major drawbacks are operator dependency and large body habitus. Multidetector computed tomography (CT) is the imaging of choice to illustrate vascular compression in abdomen due to

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its superior contrast, temporal and spatial resolution, speed, and noninvasiveness compared to conventional angiography. In addition, multiplanar reformation (MPR) can be performed in various planes, thereby providing an important imaging details that is specified and customized to the unique vascular anatomy of the patient. Magnetic resonance imaging (MRI) and angiography may replace CT in any patient who has had a previous allergic reaction to iodinated contrast agent and provide additional information to help diagnose vascular compression.

In this review, we discuss the broad spectrum of vascular compression in abdomen, including median arcuate ligament syndrome, superior mesenteric artery syndrome, nutcracker syndrome, portal biliopathy, May-Thurner syndrome, and ureteropelvic junction obstruction. The classic imaging features of each condition are described in detail, and clinical features and treatment options are highlighted.

Median Arcuate Ligament Syndrome

Median arcuate ligament is a fibrous arch or sling at the T12-L1 level that unites the diaphragmatic crura on either side of the aortic hiatus. Normally, this ligament courses superior to the origin of celiac artery.

Median arcuate ligament syndrome (MALS) or celiac artery compression syndrome was first described by Harjola in 1963.^[5] This ligament may cross over the proximal segment of the celiac artery in 10-24% of the patients with no symptoms.^[6] Most celiac artery compressions actually do not produce symptoms, probably due to collateral supply from the superior mesenteric artery. However, in some of these cases, the ligament may actually compress the celiac artery, compromising blood flow and causing symptoms. It typically occurs in young patients (20-40 years of age) and is more common in thin women. "Food fear" or postprandial abdominal pain, vomiting, and weight loss are the classic clinical symptoms. Some patients may reveal audible mid-abdominal bruit, which varies with respiration.^[7] During expiration, the median arcuate ligament compresses the celiac artery usually approximately 5 mm from its origin at the abdominal aorta. During inspiration, the celiac artery descends to the abdominal cavity as the lungs expand, and this compression is often relieved with a more vertical and caudal orientation of celiac artery. Therefore, persistent compression on celiac artery by median arcuate ligament on inspiration may be shown in severe cases [Figure 1] and isolated compression of celiac artery during expiration may not be clinically significant. However, this syndrome relies on a combination of both radiographic and clinical features.

Classic imaging features

Doppler ultrasound can be used as the first line in screening for MALS. The major advantages of Doppler ultrasound

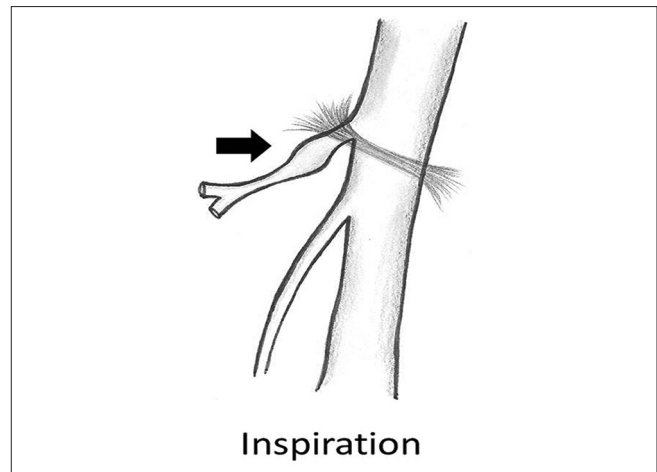


Figure 1: Drawing of sagittal orientation of median arcuate ligament syndrome in severe case. Celiac artery compression and poststenotic dilatation of celiac artery (arrow) are shown on inspiration in severe case

over CT are that it is noninvasive and does not expose patients to radiation. In addition, it can give the imaging details of celiac artery during both phases of respiration and during sitting and erect positions [Figure 2].^[8] A flow velocity measurement on Doppler ultrasound performed at compressed or narrowed segment of celiac artery reveals variation of peak systolic velocity (PSV) during respiration with a marked increase during expiration in PSV to greater than 200 cm/s. A greater than 3:1 ratio of PSV in the celiac artery in expiration compared with the PSV in the abdominal aorta just below the diaphragm is another useful criterion to diagnose MALS.^[4,9] CT angiography plays a major role in establishing diagnosis. Owing to the superior spatial resolution of CT, the median arcuate ligament may be detectable, a thickness of more than 4 mm is considered abnormal [Figure 3].^[10] Celiac artery compression may not be appreciated on the axial plane. Sagittal plane is necessary to visualize median arcuate ligament and proximal portion of the celiac artery. Focal narrowing with hooked appearance is the hallmark which can help distinguish this condition from other etiologies of celiac artery stenosis such as atherosclerosis [Figure 4A]. Severe compression persists during inspiration. Other findings include poststenotic dilatation and collateral vessels such as pancreaticoduodenal arcade from superior mesenteric artery. MRI can illustrate perivascular inflammation or fibrosis around the celiac artery in chronic cases [Figure 5].

Management

The treatment options for MALS include surgical or laparoscopic division of the median arcuate ligament to restore normal blood flow in the celiac artery [Figure 4B].^[11] A surgical division of the fibrous ligament was classically performed in noncomplicated cases. However, complex surgical procedures such as vascular reconstruction of the celiac axis, aortoceliac bypass, and reimplantation of the celiac artery may be required in some patients.^[12]

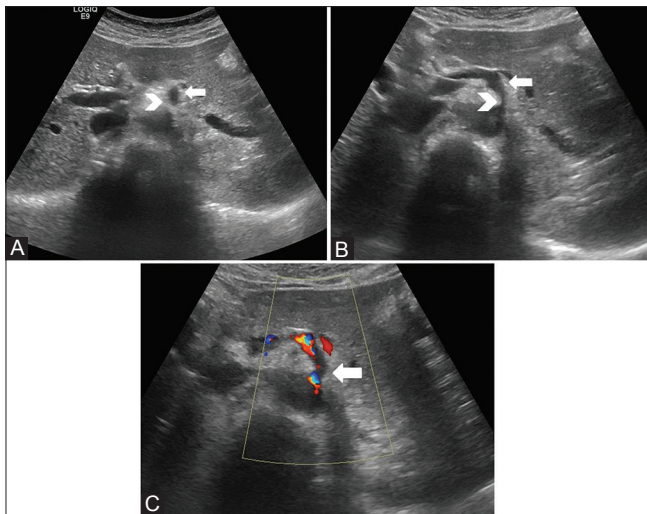


Figure 2 (A-C): Celiac artery compression. (A) Gray scale ultrasound during expiration shows thickened hyperechoic median arcuate ligament (arrowhead) compressing celiac artery (arrow). (B) Gray scale ultrasound during expiration, slightly lower than (A) shows focal narrowing (arrowhead) and poststenotic dilatation of celiac artery (arrow). (C) Doppler ultrasound on upright position and inspiration, the celiac artery descends further in the abdominal cavity, resulting in more vertical orientation and relief of compression (arrow)

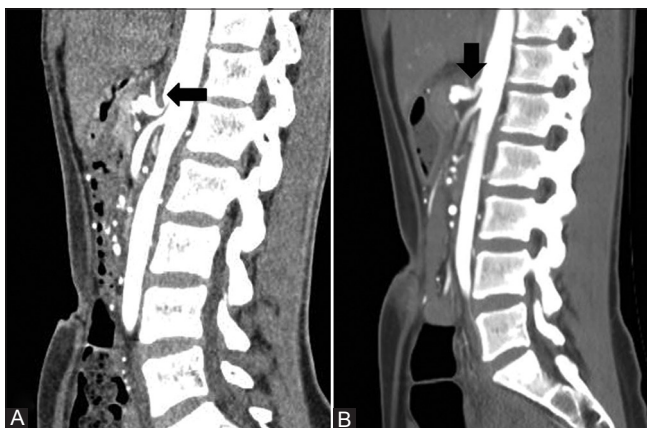


Figure 4 (A and B): A 28-year-old female with postprandial abdominal pain. (A) Contrast-enhanced sagittal CT angiography of abdomen shows thickened median arcuate ligament (arrow), kinking proximal celiac artery with hooked appearance and poststenotic dilatation. (B) After laparoscopic division of the median arcuate ligament shows lumen patency with acceptable mild residual stenosis at proximal celiac artery (arrow)

Superior Mesenteric Artery Syndrome

Superior mesenteric artery (SMA) syndrome is an uncommon intestinal obstruction caused by extrinsic compression of the third part of the duodenum due to constriction of the angle between SMA and abdominal aorta. It is more frequent in woman and young adults. There is some debate regarding this syndrome because the relationship between anatomical findings and clinical symptoms is not well established. Risk factors include rapid weight loss which decreases fatty pouch between SMA and aorta, corrective scoliosis surgery which

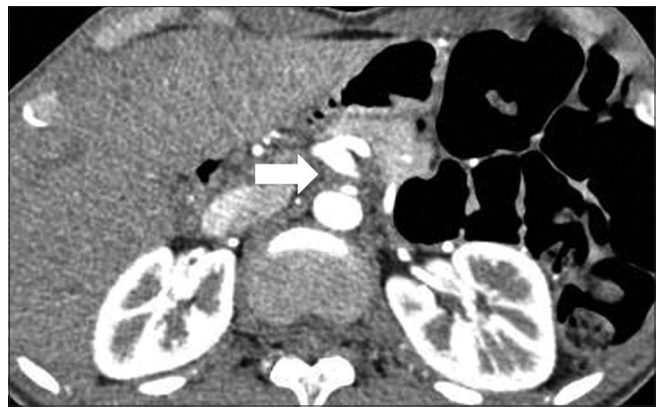


Figure 3: Contrast-enhanced axial CT angiography shows compression of origin of celiac artery by band of median arcuate ligament (arrow)

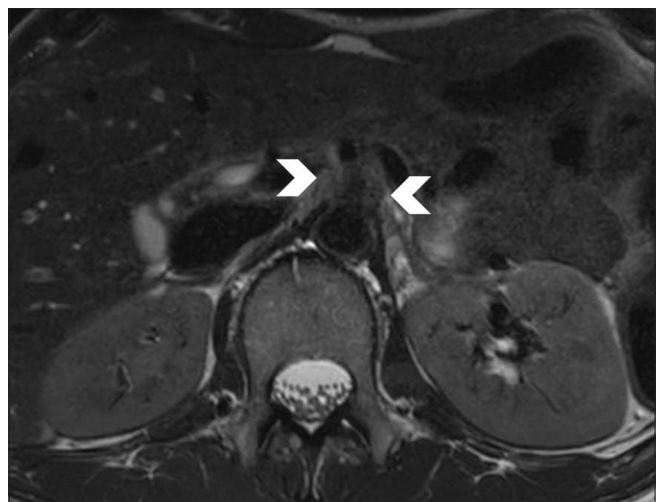


Figure 5: Median arcuate ligament syndrome. Axial T2-weighted image shows ill defined soft tissue thickening surrounding celiac artery (arrowheads) representing perivascular inflammation and fibrosis

causes lengthening of the spine, and hip or body cast that applies external abdominal pressure. These conditions are postulated to decrease the aortomesenteric angle (AMA) and the aortomesenteric distance (AMD).^[13,14] The normal AMD is typically 10-28 mm and is measured at the level of the horizontal part of the duodenum as it travels between the abdominal aorta and SMA.^[15,16] This syndrome is more frequent in young females. Symptoms resulting from duodenal obstruction include abdominal pain, vomiting, and weight loss. Classically, the pain may be relieved by lying in prone or left lateral decubitus position. All other conditions of duodenal obstruction, including intrinsic causes such as benign or malignant tumors, and extrinsic causes such as pancreatitis, mesenteric mass, or aortic aneurysm must be ruled out with customized imaging modalities before a diagnosis of SMA syndrome is established.^[17]

Classic imaging features

Barium examination can be performed to diagnose SMA syndrome. It shows vertical band of external compression

at the third part of the duodenum with proximal gastric and duodenal dilatation [Figure 6A]. Other findings on barium include reverse barium flow or antiperistaltic flow of barium proximal to the site of obstruction and relief of obstruction when the patient is in a prone or left lateral decubitus [Figure 6B and C].^[18] However, CT is the preferred imaging modality as it allows measurement of AMA and AMD. In SMA syndrome, both parameters are reduced, with values of 6-22 degrees and 2-8 mm, respectively [Figure 7A and B].^[19] Associated findings on CT include a dilated stomach and duodenum up to the aortomesenteric angle followed by abrupt narrowing as the duodenum passes underneath the SMA. Additional value of CT is to show the exact anatomic location of the duodenum and excluding other causes of obstruction.^[20] Reduction in AMA and AMD on CT findings with no clinical signs and symptoms is not sufficient to diagnose SMA syndrome.

Management

Nasogastric tube placement to decompress the obstruction is the initial conservative treatment and also to provide enteral feeding. Surgery is indicated in case of failed conservative treatment. Duodenojejunostomy is the surgical option to relieve the obstruction, with good results being reported in 79-100% of the cases.^[18]

Nutcracker Syndrome

Nutcracker syndrome or renal vein entrapment syndrome is the compression of the left renal vein as it passes in

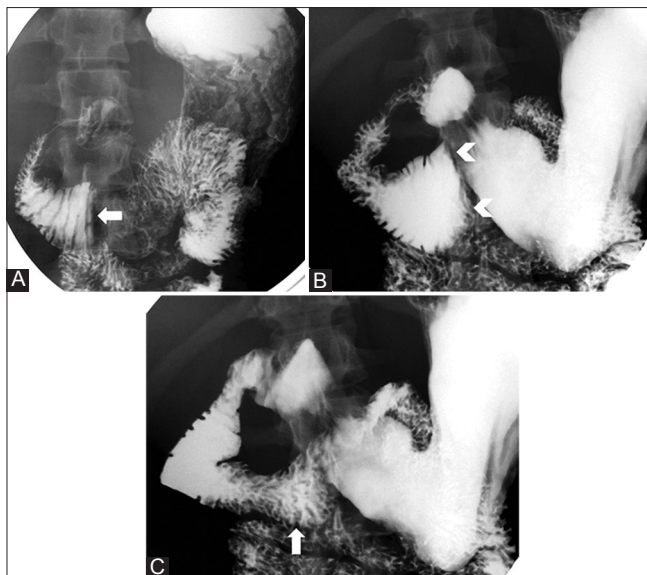


Figure 6 (A-C): A 34-year-old female with weight loss. (A) Barium examination shows vertical band of extrinsic compression (arrow) on the mid horizontal part of duodenum caused by superior mesenteric artery with proximal duodenal dilatation. It is not relief on prone position (not shown). A 32-year-old-female with abdominal pain shows dilatation with abrupt vertical compression at the third part of duodenum (arrowheads in B) and relief of obstruction when the patient is in a prone position (arrow in C)

the narrow angle between SMA and the abdominal aorta (anterior nutcracker). Steep or narrow angulation of the SMA relative to the aorta resulting in decreased aortomesenteric distance can predispose to anterior nutcracker syndrome.^[21] Therefore, anterior nutcracker syndrome and SMA syndrome may simultaneously occur. Other predisposing factors including rapid weight loss with loss of retroperitoneal fat, and abnormally high course of the left renal vein have also been postulated to cause left renal vein compression.^[22] However, this type of anatomy does not always produce clinical symptoms and may present as a normal variation. Therefore, the term nutcracker syndrome should be reserved for patients with both characteristic clinical symptoms and nutcracker anatomy.^[23] No consensus exists as to which clinical manifestations are severe enough to be aware of this syndrome. However, in cases where compression is detected but there is no clinical syndrome, it is possibly termed nutcracker phenomenon.

This syndrome is most frequently seen in healthy and thin women in the third and fourth decade of life. There are two variants of nutcracker syndrome – anterior nutcracker and posterior nutcracker. The anterior nutcracker or mesoaortic compression of the left renal vein is more common. The posterior or pseudonutcracker syndrome is rare and defined as the compression of the retroaortic left renal vein between the vertebral body and aorta. However, the clinical significance of pseudonutcracker syndrome is still unclear.^[24]

The most common clinical manifestation is hematuria which can vary from mild to severe, as the left renal vein is compressed at mesoaortic angle which results in left renal vein hypertension and formation of high pressure thin-walled varices around the renal pelvis, which tend to rupture at the calyceal fornices. Other manifestations include nonspecific abdominal pain, varicoceles in men or pelvic congestion, or ovarian vein syndrome in women.^[25]



Figure 7 (A and B): Superior mesenteric artery syndrome. (A) Contrast-enhanced sagittal CT shows steep angulation of superior mesenteric artery with short AMD and narrowed AMA (arrow). (B) Contrast-enhanced sagittal CT in normal subject shows normal AMD and AMA (arrow)

Classic imaging features

Doppler ultrasound is a noninvasive imaging method to diagnose nutcracker syndrome with a sensitivity and specificity of 78% and 100%, respectively.^[26] A significantly increased ratio of peak systolic velocity at the point of renal vein compression to peak systolic in the hilar renal vein has been found in patients with nutcracker syndrome compared with controls.^[27,28] However, advanced CT or MRI with multiplanar images clearly depict the beak sign (abrupt narrowing or compression of the left renal vein between aorta and SMA) [Figure 8A], dilatation of left gonadal vein [Figure 8B], or pelvic varices and left renal vein variations such as retro-aortic type (in posterior



Figure 8 (A and B): A 62-year-old male with abdominal pain. (A) Contrast-enhanced axial CT shows evidence of beak sign (arrow) which is the marked narrowing of left renal vein between superior mesenteric artery and tortuous abdominal aorta. (B) Contrast-enhanced coronal CT shows dilated left gonadal vein (arrow) caused by left renal vein entrapment

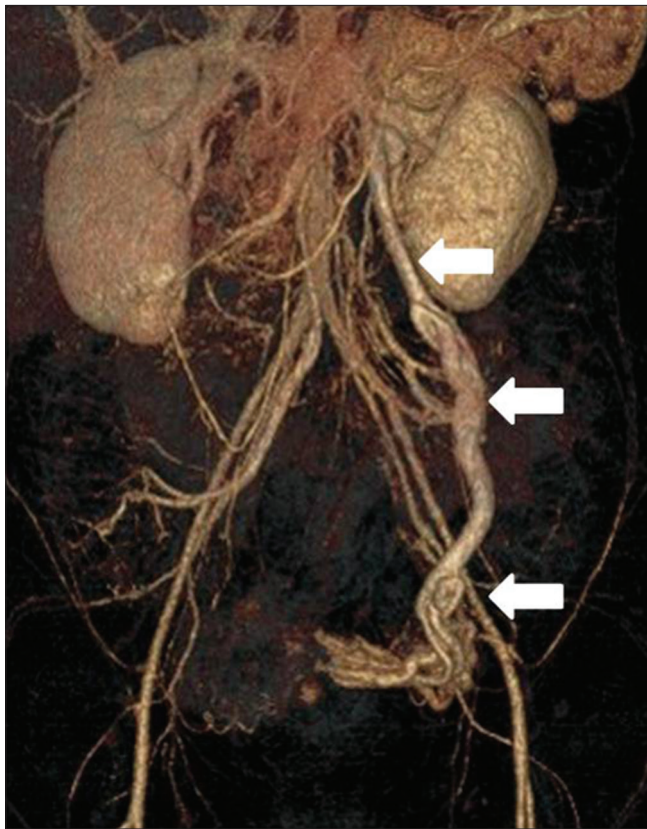


Figure 10: Anterior nutcracker syndrome. Volume rendering technique shows moderately dilated left gonadal vein (arrows) and pelvic varices

nutcracker syndrome) [Figure 9]. MPR images or volume rendering technique better depicts the course of dilated gonadal vein [Figure 10]. Demonstration of left renal vein compression at imaging in the absence of clinical symptoms and varices should be termed nutcracker phenomenon, and not nutcracker syndrome. CT also plays the key role to differentiate the various causes of left renal vein compression such as retroperitoneal tumors, retroperitoneal fibrosis, or lymphadenopathy [Figure 11]. However, the gold standard of diagnostic test is retrograde venography, which allows measurement of renocaval pressure gradient and contrast mapping of the dilated gonadal vein and the periureteral and pelvic collateral veins. In normal individuals, the pressure gradient between left renal vein and IVC is less than 1 mm Hg. A gradient of more than 3 mmHg has been reported to be hemodynamically significant in nutcracker syndrome.^[29]

Management

There is no definite guideline about which clinical manifestations are sufficiently enough to warrant appropriate management. Gross hematuria, severe pain, and renal failure may be the indications for surgical treatment,^[22] ranging from a left renal vein bypass to the more conventional approach of nephrectomy. Nowadays,

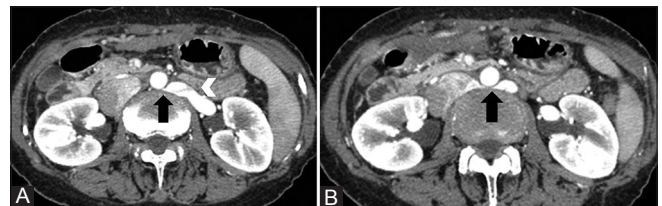


Figure 9 (A and B): Posterior nutcracker syndrome. (A) Contrast-enhanced axial CT shows beak sign (arrow) at retroaortic left renal vein and dilatation of left renal vein (arrowhead). (B) Contrast-enhanced axial CT shows entrapment of the retroaortic left renal vein between aorta and vertebral column (arrow)

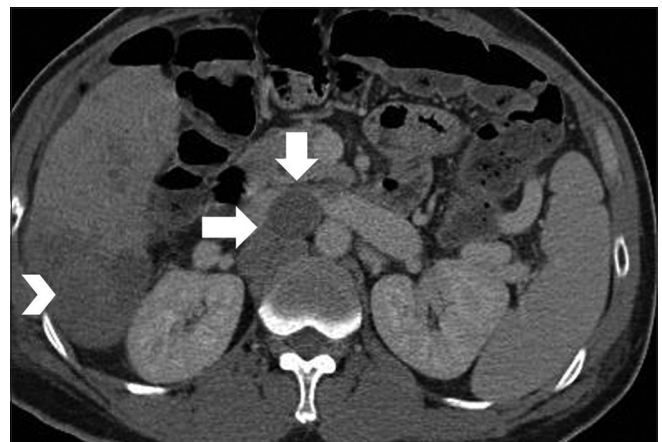


Figure 11: A 49-year-old male with left leg swelling. Contrast-enhanced axial CT shows multiple aortocaval nodes (horizontal arrow) causing left renal vein compression (vertical arrow). Liver mass is also seen (arrowhead). Surgery confirmed cholangiocarcinoma

endovascular stent placement has gained popularity with good outcomes.

Portal Biliopathy

Portal biliopathy is a relative new diagnostic terminology that refers to abnormalities of the bile duct and gallbladder in patients with extrahepatic portal vein obstruction or portal hypertension.^[30] Two postulated mechanisms of biliary abnormalities are external compression by collateral veins or portal cavernous transformation and peribiliary fibrosis due to inflammatory or ischemic changes.^[31] Studies have reported that changes in the bile duct occur in 81-100% of the patients, although only 5-30% have obstructive jaundice.^[32] The average duration since diagnosis of portal cavernous transformation was made to biliary symptoms was 8 years.^[31] The portal cavernous transformation composed of two venous systems including the paracholedochal veins, which run parallel to the ductal wall, and the epicholedochal veins, located on the surface of the bile duct.^[33,34] Shin *et al.* classified the portal biliopathy on imaging into three types – varicoid, fibrotic, and mixed.^[30] The varicoid type is the compression and distortion of bile duct by large collateral veins (paracholedochal veins). The fibrotic type shows thickened, narrowed enhanced bile duct that results from compression of smaller intramural collateral veins (epicholedochal veins).

Classic imaging features

Gray scale and color Doppler ultrasound are considered as the first imaging modality to evaluate portal cavernous transformation.^[35] Classic signs include decreased caliber of portal vein and multiple anechoic tubular structures with vascular flow corresponding to paracholedochal collateral veins in hepatoduodenal ligament and porta hepatis.^[36] An increased flow in hepatic artery may be seen, representing a compensatory mechanism to the reduced portal flow. Luminal stenosis of the common bile duct due to extrinsic compression by portal cavernous transformation as well as proximal bile duct dilatation may also be seen by ultrasound. However, ultrasound has limitation in demonstration of common bile duct in some patients. In addition, irregularities caused by the dilatation of epicholedochal veins usually cannot be observed by ultrasound because its caliber is less than 1 mm. Direct cholangiography has been performed to make a diagnosis of this infrequent condition^[37,38] but being replaced by CT and MRI. Findings on direct cholangiography include segmental upstream dilatation, caliber irregularity, and extrinsic compression on the bile duct due to collateral veins, have been called “pseudocholangiocarcinoma sign” because they mimic a cholangiocarcinoma spreading along the bile duct.^[34] CT and MRI with MRCP are helpful noninvasive imaging methods to make a diagnosis, giving a better delineation of both collateral veins and biliary duct. The imaging findings include biliary dilatation, biliary

strictures and collateral veins with wavy appearance of bile duct [Figure 12]. Findings may mimic primary sclerosing cholangitis and cholangiocarcinoma.

Management

The majority of these patients are asymptomatic and do not require any intervention. However, symptomatic cases such as those with bile duct stricture can be treated by endoscopic procedures such as balloon dilatation, stent placement, and sphincterectomy.^[39] The endoscopic management should be carefully performed due to the large venous collaterals surrounding bile ducts. These venous collaterals are risk factors for hemobilia; however, if this occurs, is usually mild and needs conservative treatment in most cases.^[40]

May–Thurner Syndrome

May–Thurner syndrome (MTS) or Cockett syndrome is an uncommon vascular compression that is defined as compression of the left common iliac vein between the right common iliac artery anteriorly and the fifth lumbar vertebral body posteriorly. This compression may cause symptoms or be an incidental finding. Recent information indicates that compression of the left common iliac vein at the point of crossover may be present in 66% of the general population with no symptoms.^[41] However, the true prevalence of MTS is not known.

This syndrome typically affects young women in the age group of 20-40 years. The result of this vascular compression by arterial pulsation of the left common iliac artery is local extensive intimal proliferation causing venous thrombosis in the left common iliac vein and left femoral vein [Figure 13]. Because typical risk factors for deep venous thrombosis include postsurgery, pregnancy, and oral contraceptive



Figure 12 (A-C): A 41-year-old male with portal hypertension. (A) Contrast-enhanced axial CT shows gross portal cavernous transformation (arrow). These multiple varices or paracholedochal veins cause extrinsic compression to common bile duct (arrowhead). (B) Contrast-enhanced axial CT at superior level shows dilatation of intrahepatic bile ducts (arrow). (C) Contrast-enhanced coronal CT shows wavy appearance of the common bile duct (arrows)

pill, MTS maybe overlooked, especially if patients have those risk factors.

Clinical manifestations include acute or chronic unilateral left leg swelling, pain, and skin discoloration. Serious complications include pulmonary embolism, iliac vein rupture, and phlegmasia cerulea dolens.^[42] Phlegmasia cerulea dolens is an uncommon sequel of extensive thrombosis of ileofemoral vein that is characterized by acute limb ischemia, which can develop gangrene.

Classic imaging features

Ascending venography has been traditionally performed for definitive diagnosis. This venography illustrates the venous compression and allows hemodynamic assessment by pressure gradient measurement.^[43] However, it is an invasive test and may cause postprocedural phlebitis. Doppler ultrasound has a limitation to detect abnormalities of the iliac vein which commonly obscured by bowel gas. Therefore, conventional venography and Doppler ultrasound are currently replaced by multidetector CT or MR venography. The important diagnostic CT or MRI findings are extrinsic compression of the left common iliac vein at the crossover point [Figure 14A], presence of venous collaterals crossing the pelvis to join contralateral vein [Figure 14B], and venous thrombosis (if present). The left common iliac vein compression is best seen in the axial plane. Furthermore, CT or MRI can help exclude other causes of venous compression such as pelvic mass or adnexal mass.

Management

MTS should be treated only when it produces symptoms. Surgical repair of MTS was done previously, however, with advancement of technology less invasive endovascular treatment has gained popularity [Figure 14C].^[44]

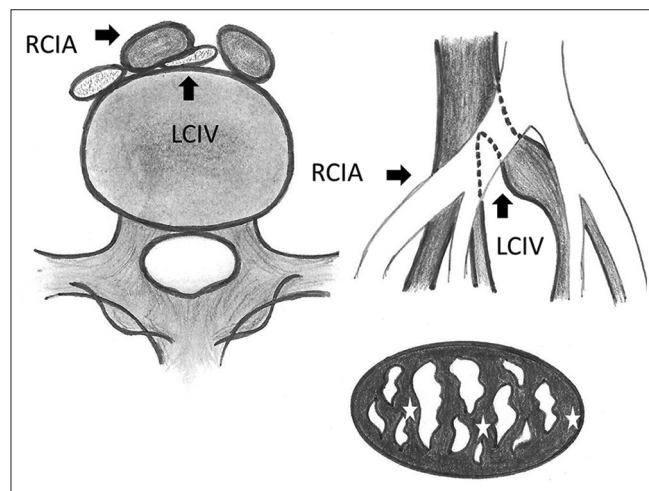


Figure 13: Drawing of May-Thurner syndrome. Left common iliac vein (LCIV) is compressed by right common iliac artery (RCIA). Endothelial damage causes deposition of collagen and web in the lumen (stars) of LCIV

Ureteropelvic Junction Obstruction by Crossing Vessel

Ureteropelvic junction (UPJ) obstruction is the most common cause of hydronephrosis with an incidence of 1 in 1000-1500.^[45] It is caused by intrinsic or extrinsic etiology. Muscular defect resulting in aperistaltic dysplastic segment is the common intrinsic cause while aberrant vessel crossing anteriorly to the UPJ or proximal ureter is the most common extrinsic cause of UPJ obstruction. Posterior crossing vessel is less commonly found.

The functional significance of crossing vessels at the location of UPJ obstruction is controversial. Although crossing vessels have been implicated as the cause of UPJ obstruction, they more commonly aggravate rather than produce the obstruction. Some reports have found crossing vessels incidentally in more than 70% of cases with no UPJ obstruction.^[46] Differentiation between incidental crossing vessels and those that appear to cause obstruction is recommended. At present, no imaging modality can make this distinction. However, preoperative detection of aberrant crossing vessel is still important because it changes the surgical plan to reduce vascular complications such as hematoma, arteriovenous fistula, and pseudoaneurysm.

Classic imaging features

CT with three-dimensional reconstructions can clearly depict vascular anatomy of the kidney and crossing vessels at the location of UPJ obstruction (the crossing vessel that contacts with the most stenotic point of UPJ) [Figure 15].

Management

Open pyeloplasty has been traditionally performed for the surgical correction for UPJ obstruction, however, it

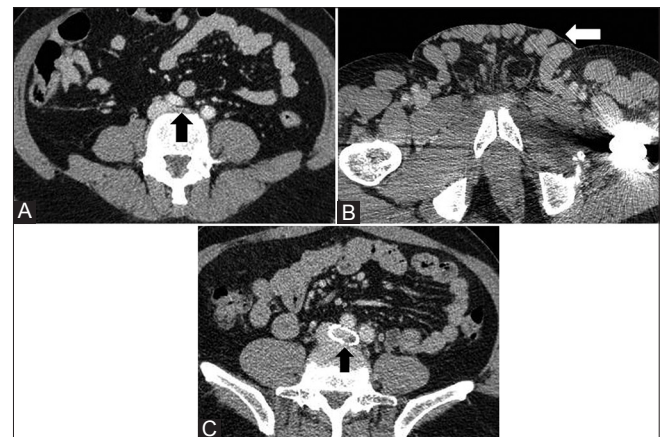


Figure 14 (A-C): A 70-year-old male with left leg pain. (A) Contrast-enhanced axial CT shows marked compression of left common iliac vein between right common iliac artery and vertebral body. Noted slit like left common iliac vein (arrow). (B) Contrast-enhanced axial CT shows pelvic venous collaterals (arrow). (C) Contrast-enhanced axial CT shows stent placement with patent lumen of left common iliac vein (arrow). (Image courtesy: Dr. Jitladda Wasinrat)

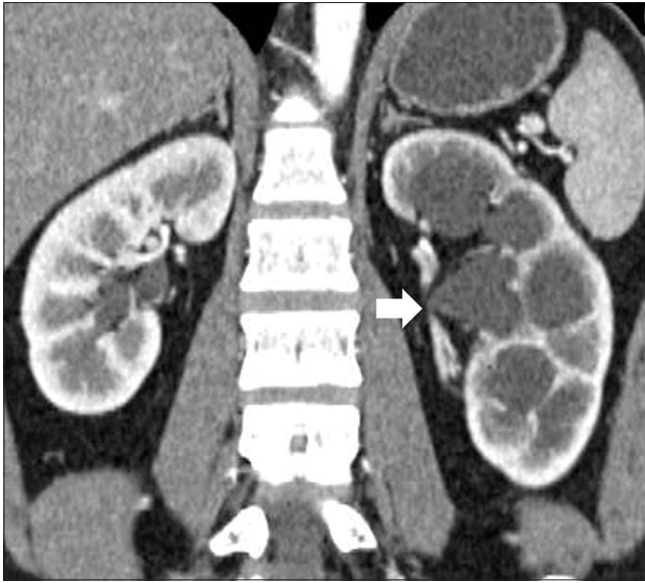


Figure 15: A 44-year-old male with left flank pain. Contrast-enhanced coronal CT shows aberrant crossing left renal vessel with extrinsic compression at left ureteropelvic junction (arrow) and moderate left hydronephrosis

is currently replaced by minimally invasive treatments such as endopyelotomy and laparoscopic dismembered pyeloplasty either transperitoneal or retroperitoneal approach.^[47]

Conclusion

Vascular compression in abdomen discussed in this review are not common and are easily missed on routine practice. Such vascular compression may be asymptomatic or may cause clinical nonspecific symptoms. A combination of clinical and radiologic features with knowledge of underlying causes that relate to anatomical compression are particularly useful to make correct diagnosis.

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Conflicts of interest

There are no conflicts of interest.

References

- Lamba R, Tanner DT, Sekhon S, McGahan JP, Corwin MT, Lall CG. Multidetector CT of vascular compression syndromes in the abdomen and pelvis. *Radiographics* 2014;34:93-115.
- Noorani A, Walsh SR, Cooper DG, Varty K. Entrapment syndromes. *Eur J Vasc Endovasc Surg* 2009;37:213-20.
- Butros SR, Liu R, Oliveira GR, Ganguli S, Kalva S. Venous compression syndromes: Clinical features, imaging findings and management. *Br J Radiol* 2013;86:20130284.
- Erden A, Yurdakul M, Cumhur T. Marked increase in flow velocities during deep expiration: A duplex Doppler sign of celiac artery compression syndrome. *Cardiovasc Intervent Radiol* 1999;22:331-2.

- Harjola PT. A rare obstruction of the coeliac artery. Report of a case. *Ann Chir Gynaecol Fenn* 1963;52:547-50.
- Horton KM, Talamini MA, Fishman EK. Median arcuate ligament syndrome: Evaluation with CT angiography. *Radiographics* 2005;25:1177-82.
- Dunbar JD, Molnar W, Berman FF, Marble SA. Comparison of the celiac trunk and abdominal angina. *Am J Roentgenol* 1965;95:731-44.
- Wolfman D, Bluth EI, Sossaman J. Median arcuate ligament syndrome. *J Ultrasound Med* 2003;22:1377-80.
- Scholbach T. Celiac artery compression syndrome in children, adolescents, and young adults: Clinical and color duplex sonographic features in a series of 59 cases. *J Ultrasound Med* 2006;25:299.
- Eliahou R, Sosna J, Bloom AI. Between a rock and a hard place: Clinical and imaging features of vascular compression syndromes. *RadioGraphics* 2012;31:E33-49.
- Duffy AJ, Panait L, Eisenberg D, Bell RL, Roberts KE, Sumpio B. Management of median arcuate ligament syndrome: A new paradigm. *Ann Vasc Surg* 2009;23:778-84.
- Dunbar JD, Molnar W, Berman FF, Marble SA. Compression of the celiac trunk and abdominal angina. *Am J Roentgenol Radium Ther Nucl Med* 1965;95:731-44.
- Sapkas G, O'Brien JP. Vascular compression of the duodenum (cast syndrome) associated with the treatment of spinal deformities. A report of six cases. *Arch Orthop Trauma Surg* 1981;98:7-11.
- Merrett ND, Wilson RB, Cosman P, Biankin AV. Superior mesenteric artery syndrome: Diagnosis and treatment strategies. *J Gastrointest Surg* 2009;13:287-92.
- Unal B, Aktas A, Kemal G, *et al.* Superior mesenteric artery syndrome: CT and ultrasonography findings. *Diagn Interv Radiol* 2005;11:90-5.
- Neri S, Signorelli SS, Mondati E, Pulvirenti D, Campanile E, Di Pino L, *et al.* Ultrasound imaging in diagnosis of superior mesenteric artery syndrome. *J Intern Med* 2005;257:346-51.
- Merrett ND, Wilson RB, Cosman P, Biankin AV. Superior mesenteric artery syndrome: Diagnosis and treatment strategies. *J Gastrointest Surg* 2009;13:287-92.
- Welsch T, Buchler MW, Kienle P. Recalling superior mesenteric artery syndrome. *Dig Surg* 2007;24:149-56.
- Konen E, Amitai M, Apter S, Garniek A, Gayer G, Nass S, *et al.* CT angiography of superior mesenteric artery syndrome. *Am J Roentgenol* 1998;171:1279-81.
- Agrawal GA, Johnson PT, Fishman EK. Multidetector row CT of superior mesenteric artery syndrome. *J Clin Gastroenterol* 2007;41:62-5.
- Menard MT. Nutcracker Syndrome: When Should It Be Treated and How? *Perspect Vasc Surg Endovasc Ther* 2009;21:117-24.
- Kurklinsky AK, Rooke TW. Nutcracker phenomenon and nutcracker syndrome. *Mayo Clin Proc* 2010;85:552-9.
- Shin JI, Lee JS. Nutcracker phenomenon or nutcracker syndrome. *Nephrol Dial Transplant* 2005;20:2015.
- Barnes RW, Fleisher HL, Redman JF, Smith JW, Harshfield DL, Ferris EJ. Meso-aortic compression of the left renal vein (the so-called nutcracker syndrome): Repair by a new stenting procedure. *J Vasc Surg* 1988;8:415-21.
- Preza Fernandes J, Amorim R, Gomes MJ, Oliveira V, Reis A, Ribeiro-Castro J. Posterior nutcracker syndrome with left renal vein duplication: A rare cause of haematuria in a 12-year-old boy. *Case Rep Urol* 2012;2012:849681.
- Takebayashi S, Ueki T, Ikeda N, Fujikawa A. Diagnosis of the nutcracker syndrome with color Doppler sonography: Correlation with flow patterns on retrograde left renal venography. *AJR Am J*

- Roentgenol 1999;172:39-43.
27. Kim SH, Cho SW, Kim HD, Chung JW, Park JH, Han MC. Nutcracker syndrome: Diagnosis with Doppler US. *Radiology* 1996;198:93-7.
 28. Park SJ, Lim JW, Cho BS, Yoon TY, Oh JH. Nutcracker syndrome in children with orthostatic proteinuria: Diagnosis on the basis of Doppler sonography. *J Ultrasound Med* 2002;21:39-45.
 29. Reed NR, Kalra M, Bower TC, Vrtiska TJ, Ricotta JJ 2nd, Gliviczki P. Left renal vein transposition for nutcracker syndrome. *J Vasc Surg* 2009;49:386-93.
 30. Shin SM, Kim S, Lee JW, *et al.*: Biliary abnormalities associated with portal biliopathy: Evaluation on MR cholangiography. *AJR Am J Roentgenol* 2007;188:W341-7.
 31. Vibert E, Azoulay D, Aloia T, Pascal G, Veilhan LA, Adam R, *et al.* Therapeutic strategies in symptomatic portal biliopathy. *Ann Surg* 2007;246:97-104.
 32. Dilawari, J.B and Chawla, Y.K. Pseudo-sclerosing cholangitis in extrahepatic portal venous obstruction. *Gut* 1992;33:272-6.
 33. Chevallier P, Denys A, Novellas S, Schmidt S, Schnyder P, Bruneton JN. Magnetic resonance cholangiography features of biliary abnormalities due to cavernous transformation of the portal vein. *Clin Imaging* 2000;30:190-4.
 34. He ZP, Fan LJ. Diagnosis and treatment of portal biliopathy. *Hepatobiliary Pancreat Dis Int* 2002;1:581-6.
 35. Nunoi H, Hirooka M, Ochi H, Koizumi Y, Tokumoto Y, Abe M, *et al.* Portal biliopathy diagnosed using color Doppler and contrast-enhanced ultrasound. *Intern Med* 2013;52:1055-9.
 36. Gulati G, Pawa S, Chowdhary V, Kumar N, Mittal SK. Colour Doppler flow imaging findings in portal biliopathy. *Trop Gastroenterol* 2003;24:116-9.
 37. De Gaetano AM, Lafortune M, Patriquin H, De Franco A, Aubin B, Paradis K. Cavernous transformation of the portal vein: Patterns of intrahepatic and splanchnic collateral circulation detected with Doppler sonography. *AJR Am J Roentgenol* 1995;165:1151-5.
 38. Chandra R, Kapoor D, Tharakan A, Chaudhary A, Sarin SK. Portal biliopathy. *J Gastroenterol Hepatol* 2001;16:1086-92.
 39. Bhatia V, Jain AK, Sarin SK. Choledocholithiasis associated with portal biliopathy in patients with extrahepatic portal vein obstruction: Management with endoscopic sphincterotomy. *Gastrointest Endosc* 1995;42:178-81.
 40. Layec S, D'Halluin PN, Pagenault M, Bretagne JF. Massive hemobilia during extraction of a covered self-expandable metal stent in a patient with portal hypertensive biliopathy. *Gastrointest Endosc* 2009;70:555-6.
 41. Kibbe MR, Ujiki M, Goodwin AL, Eskandari M, Yao I, Matsumura I. Iliac vein compression in an asymptomatic patient population. *J Vasc Surg* 2004;39:937-43.
 42. Kim YH, Ko SM, Kim HT. Spontaneous rupture of the left common iliac vein associated with May Thurner syndrome: Successful management with surgery and placement of an endovascular stent. *Br J Radiol* 2007;80:e176-9.
 43. Taheri SA, Williams J, Powell S. Iliocaval compression syndrome. *Am J Surg* 1987;154:169-72.
 44. Moudgill N, Hager E, Gonsalves C, Larson R, Lombardi J, DiMuzio P. May-Thurner syndrome: Case report and review of the literature involving modern endovascular therapy. *Vascular* 2009;17:330-5.
 45. Hu G, Luo M, Xu Y. Giant hydronephrosis secondary to ureteropelvic junction obstruction in adults: Report of a case and review of literatures. *Int J Clin Exp Med* 2015;8:4715-7.
 46. Lowe FC, Marshall FF. Ureteropelvic junction obstruction in adults. *Urology* 1984;23:331-5.
 47. Wolf JS. Laparoscopic transperitoneal pyeloplasty. *J Endourol* 2011;25:173-8.