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Tit for Tat: A Case Series of Hollow Viscus and Vascular Compressions

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Abstract

Background Abdominopelvic vascular and hollow viscus compression syndromes present unique challenges in radiological diagnosis, often requiring thorough exclusion of other pathologies. Recognizing these rare syndromes is essential for accurate differential diagnosis and timely treatment, reducing morbidity and mortality. This case series aims to share notable experiences in identifying unusual compression syndromes through radiological findings.

Case Reports Case 1: A 22-year-old male presented with postprandial epigastric discomfort and reflux, which was diagnosed as duodenal compression due to the mid portion of the third segment of the duodenum being compressed between the superior mesenteric vein and the aorta.Case 2: A 47-year-old male with left loin pain revealed hydroureteronephrosis caused by smooth narrowing/kinking at the level of the fourth lumbar vertebra, leading to obstruction and renal dysfunction. Case 3: A 15-year-old female with left lower limb swelling and pain was diagnosed with venous thrombosis in the left common iliac, external iliac, and common femoral veins due to giant fecal impaction.

Discussion These cases underscore the significance of recognizing vascular compression syndromes that mimic common clinical conditions. Case 1 highlights the rare compression of the duodenum, which may be mistaken for gastroesophageal reflux disease (GERD). Case 2 illustrates how vascular kinking can obstruct the urinary tract and cause hydroureteronephrosis. Case 3 emphasizes the role of fecal impaction in leading to venous thrombosis, a potentially overlooked complication. Prompt imaging and differential diagnosis are critical for proper management.

Conclusion These cases provide valuable insights into the recognition and management of abdominopelvic vascular and hollow viscus compression syndromes. Understanding these rare conditions allows for accurate diagnosis, timely treatment, and improved patient outcomes. The findings contribute to the body of knowledge, aiding healthcare professionals in managing such complex clinical presentations.

Keywords
- hollow viscus compressions
- SMA syndrome
- SMV syndrome
- testicular vein syndrome
- vascular compressions

Introduction

Abdominopelvic vascular and hollow viscus compression syndromes encompass a spectrum of conditions where either blood vessels compress the hollow viscera or vice versa. This can lead to diverse symptoms and pose diagnostic challenges, particularly when they manifest incidentally in

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asymptomatic individuals or with atypical symptoms. Examples include Wilkie's syndrome, ureteropelvic junction obstruction, portal biliopathy (compression of hollow viscera by adjacent blood vessels), and Dunbar syndrome, nutcracker syndrome, and May–Thurner syndrome (compression of vessels by adjacent structures).[1] These syndromes may arise due to various factors such as anatomical anomalies, rapid weight loss, surgical alterations, or congenital abnormalities.

The diagnosis of these syndromes often requires advanced imaging techniques such as contrast-enhanced computed tomography (CECT) or ultrasound, which can reveal compression of structures and aid in establishing the underlying cause. However, due to their rarity and diverse presentations, these syndromes may go undiagnosed or misdiagnosed, leading to delays in appropriate management.

Cases

Case 1

A 22-year-old male presented with complaints of postprandial epigastric discomfort, fullness, indigestion, and reflux for the past 12 months. His symptoms were relieved spontaneously a few hours after a meal. Upper gastrointestinal (GI) endoscopy did not reveal any significant finding. The patient's symptoms were not relieved by proton-pump inhibitors and other antireflux medications. CECT abdomen (ABD) was done and showed the compression of mid portion of D3 segment of the duodenum between superior mesenteric vein (SMV) and its venous tributaries and aorta. The stomach and D1 and D2 segments of the duodenum appeared distended. ►**Fig. 1A** shows CECT-ABD (venous phase) axial sections at the level of D3 segment. ►**Fig. 1B** shows CECT-ABD (venous phase) oblique coronal section of the SMV and its tributaries. To demonstrate the functional status of this mechanical compression, we proceeded with barium meal study, which showed the distension of second and proximal third parts of the duodenum with a persistent filling defect at the mid portion of D3 segment likely due to extrinsic compression. ►**Fig. 2A** shows right anterior oblique projection of contrast-filled C-loop of the duodenum with extrinsic compression at mid portion of D3. ►**Fig. 2B** shows supine anteroposterior projection of partial hold-up of contrast in D2 and proximal D3 segments with a persistent filling defect at mid D3. Hence, the diagnosis of superior mesenteric artery (SMA)-like syndrome (SMV syndrome) was made.

Case 2

A 47-year-old male presented with complaints of left loin pain for 4 weeks. The pain was intermittent in nature and was not relieved by nonsteroidal anti-inflammatory drugs. No history of renal/ureteric calculi was given by the patient. No recent fever episodes as alleged by the patient. The patient was referred for ultrasound kidney, ureter, and bladder (US-KUB) and the study showed moderate left hydronephrosis and proximal hydroureter. ►**Fig. 3** shows US image (longitudinal view) of hydronephrosis of the left kidney. ►**Fig. 4** shows US image (longitudinal view) of the proximal left hydroureter. No proximal ureteric or left

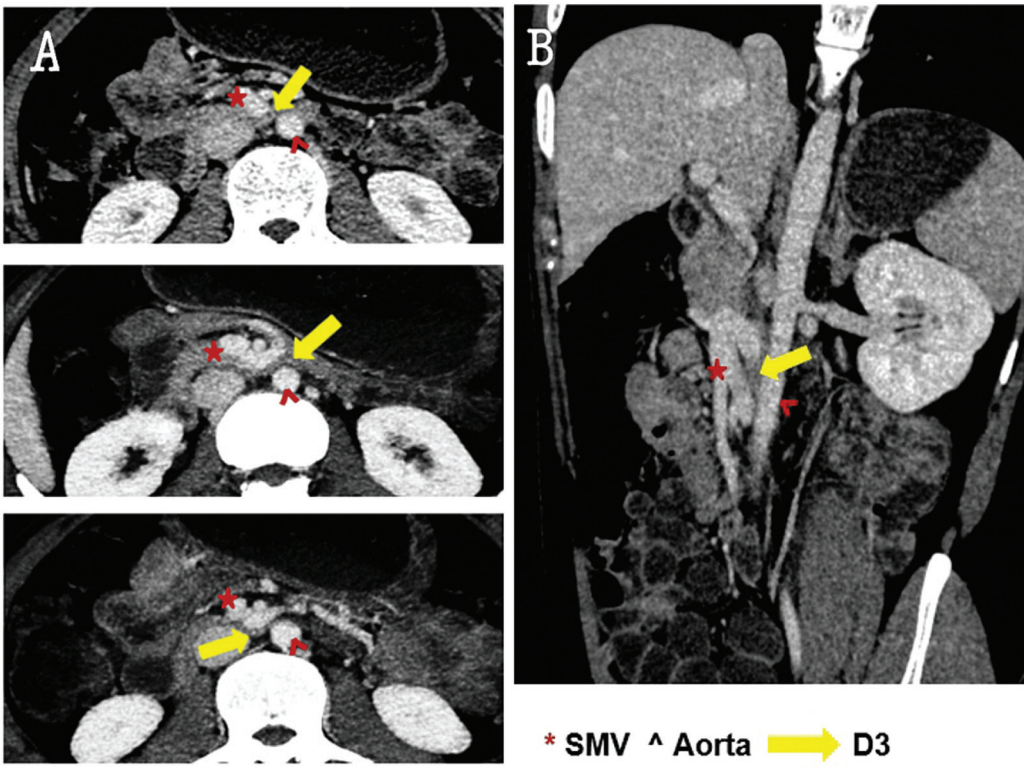

Fig. 1 (A) CECT-ABD (venous phase) axial sections at the level of D3 segment. (B) CECT-ABD (venous phase) oblique coronal section showing SMV and its tributaries. CECT ABD, contrast-enhanced computed tomography abdomen; SMV, superior mesenteric vein.

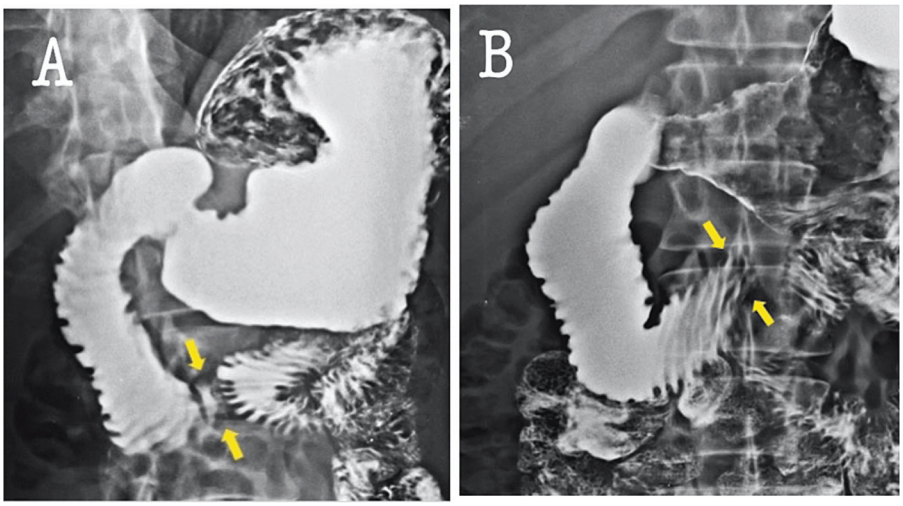

Fig. 2 (A) RAO projection displaying contrast-filled C-loop of duodenum with extrinsic compression at mid portion of D3. (B) Supine anteroposterior (AP) projection displaying partial hold-up of contrast in D2 and proximal D3 segments with persistent filling defect at mid D3. RAO, right anterior oblique.

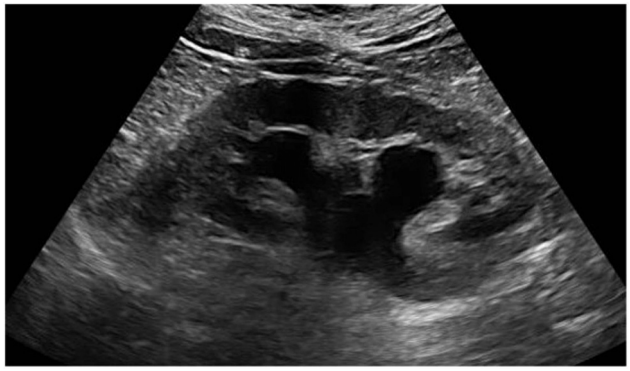

Fig. 3 US abdomen image (longitudinal view) showing hydronephrosis of left kidney. US, ultrasound.

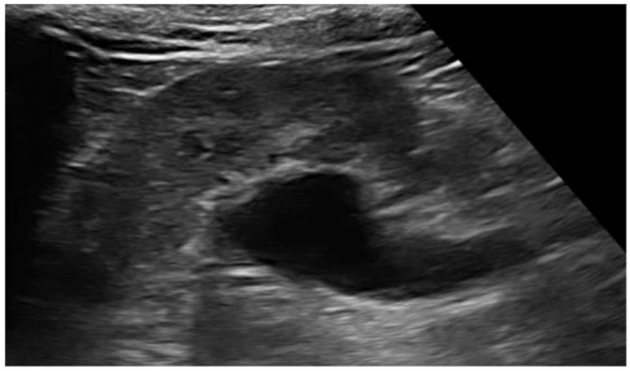

Fig. 4 US abdomen image (longitudinal view) showing proximal left hydroureter. US, ultrasound.

vesicoureteric calculus was noted. No obvious cause for hydroureteronephrosis (HUN) was detected in the study. So, we proceeded with CECT-KUB, which showed left moderate HUN up to the testicular vein crossover at the level of L4 vertebra with smooth narrowing/kinking. The portion of the ureter distal to the crossover was unremarkable. The testicular vein at the crossover appeared pristine and was of

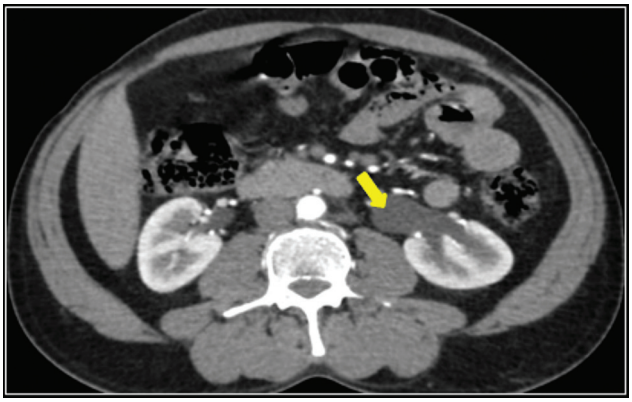

Fig. 5 CECT-KUB (corticomedullary phase) axial section at the level of renal pelvis showing left HUN. CECT-KUB, contrast-enhanced computed tomography kidney, ureter, and bladder; HUN, hydroureteronephrosis.

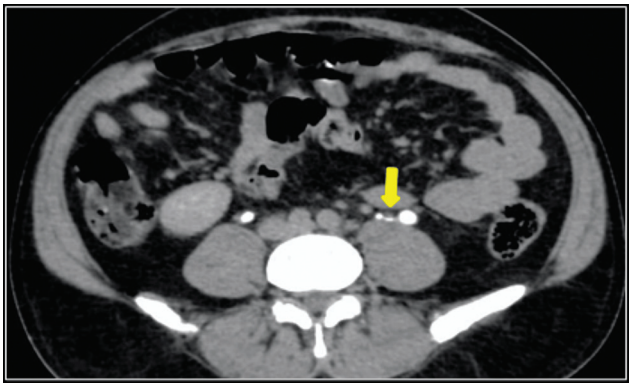

Fig. 6 CECT-KUB (excretory phase) axial section at the level of left testicular vein crossover. CECT-KUB, contrast-enhanced computed tomography kidney, ureter, and bladder.

normal caliber (3.2 mm). ►**Fig. 5** shows CECT-KUB (corticomedullary phase) axial section at the level of renal pelvis showing left HUN. ►**Fig. 6** shows CECT-KUB (excretory

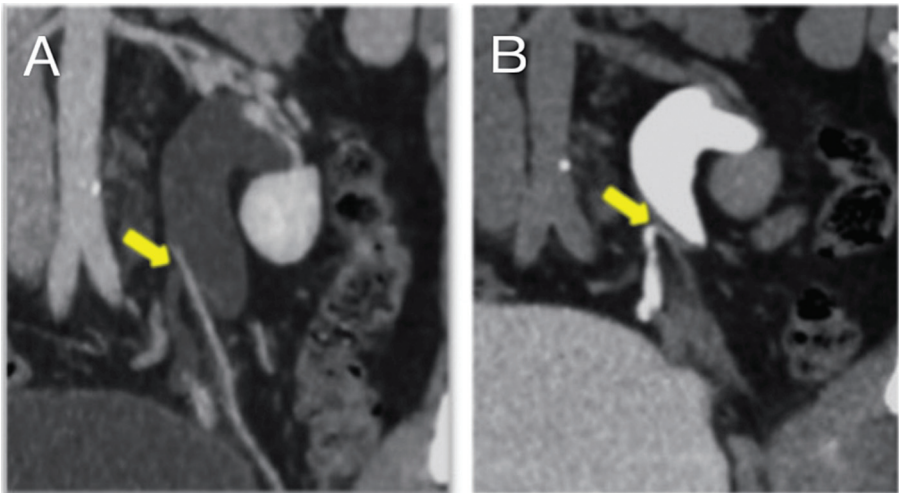

Fig. 7 CECT-KUB (venous and excretory phases) coronal section at the level of left testicular vein crossover. CECT-KUB, contrast-enhanced computed tomography kidney, ureter, and bladder.

phase) axial section at the level of the left testicular vein crossover. ►**Fig. 7** shows CECT-KUB (venous and excretory phases) coronal section at the level of the left testicular vein crossover.

Case 3

A 15-year-old normal female child with no developmental abnormalities presented with complaints of left lower limb swelling and pain predominantly on the proximal aspect for the past 4 days. The patient had history of chronic constipation, which was gradually progressive in nature along with on-and-off episodes of abdominal pain. All the initial blood investigations including the coagulation profile were within normal limits. We performed left lower-limb venous Doppler study in the patient, which showed echogenic thrombus within the lumen of visualized portions of common femoral vein extending into the superficial femoral vein (►**Fig. 8**). Neither color flow nor spectral wave pattern could be demonstrated in those veins. The abovementioned veins were incompressible and showed no flow augmentation on distal compression. Since we were not able to arrive at the cause of deep vein thrombosis in the otherwise healthy child and also due to her complaints of abdominal pain and constipation, we proceeded with CECT-ABD study, which showed us gross dilatation of sigmoid colon and rectum with significant fecal impaction measuring approximately $41 \times 16 \times 15.5$ cm extending up to the level of left hemidiaphragm, compressing and causing thrombosis of the left common iliac, external iliac veins, and visualized portions of left common femoral vein. ►**Fig. 9** shows CECT-ABD (arterial phase) coronal section showing gross fecal impaction in

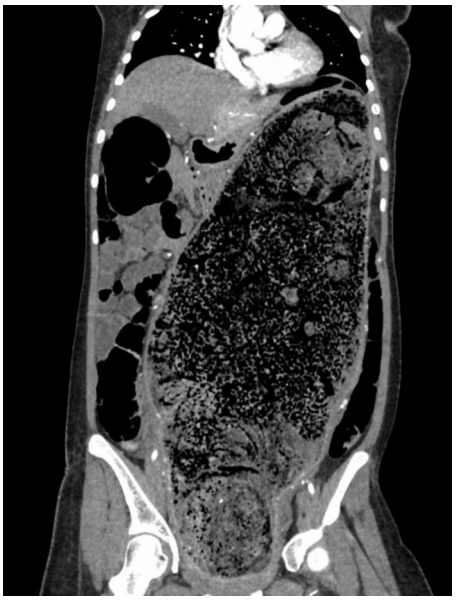

Fig. 8 Left lower-limb venous Doppler study in the patient, which showed echogenic thrombus within the lumen of visualized portions of common femoral vein extending into the superficial femoral vein.

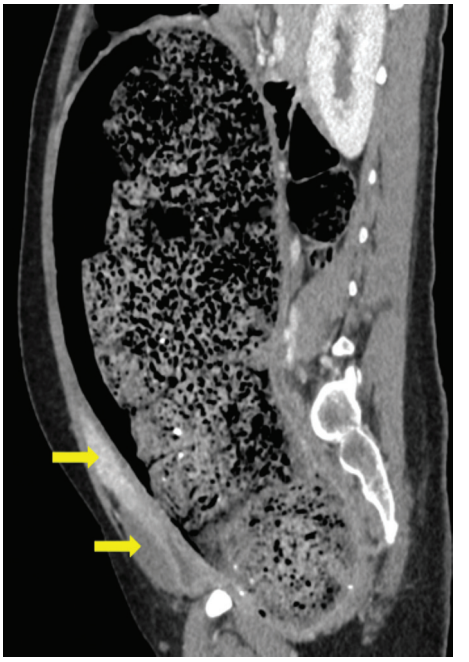

Fig. 9 Contrast-enhanced computed tomography abdomen (CECT-ABD) (arterial phase) coronal section displaying gross fecal impaction in the rectum and sigmoid colon.

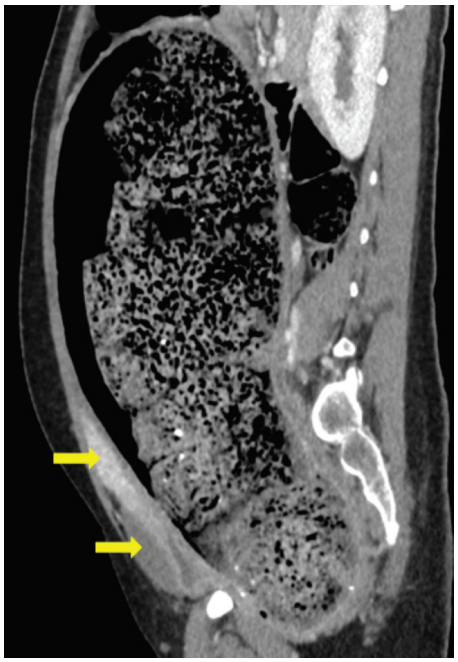

Fig. 10 Contrast-enhanced computed tomography abdomen (CECT-ABD) (venous phase) sagittal section showing pressure effect on the urinary bladder and uterus.

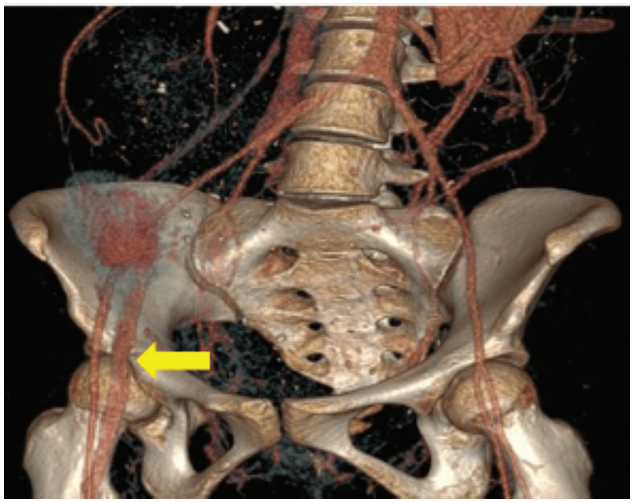

Fig. 12 Contrast-enhanced computed tomography abdomen (CECT-ABD) (venous phase) volume rendering image displaying nonvisualization of left common iliac, external iliac, and common femoral veins and normal appearing veins on the contralateral side.

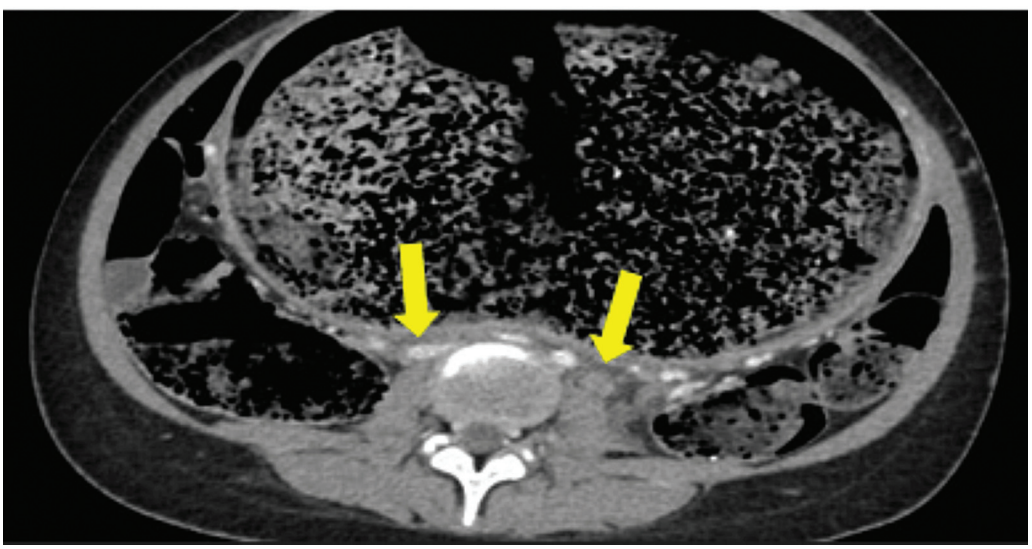

Fig. 11 Contrast-enhanced computed tomography abdomen (CECT-ABD) (venous phase) axial section showing thrombosed left common iliac vein.

the rectum and sigmoid colon. ►**Fig. 10** shows CECT-ABD (venous phase) sagittal section showing pressure effect on the urinary bladder and uterus. ►**Fig. 11** shows CECT-ABD (venous phase) axial section showing thrombosed left common iliac vein. ►**Fig. 12** shows CECT-ABD (venous phase) volume rendering image showing nonvisualization of the left common iliac, external iliac, and common femoral veins and normal appearing veins on the contralateral side.

Discussion

The first case is a case of SMA-like syndrome (SMV syndrome). The risk factors for SMA-like syndromes include various causes of megaduodenum-like systemic sclerosis, dermatomyositis, systemic lupus erythematosus, diabetes mellitus, amyloidosis, and chronic idiopathic intestinal pseudo-obstruction (Ogilvie's). Other risk factors include malrotation of midgut, congenitally shortened suspensory ligament of the duodenum (high placed insertion), rapid and severe weight loss, iatrogenic alteration of anatomy (GI surgeries), surgical correction of scoliosis, and chronic external compression (hip spica cast). Only eight such cases have been reported in the literature.[2–5] Five out of these eight cases were duodenal compressions between SMV and inferior vena cava. One case was due to postsurgical alterations in anatomy, while another was due to an anomalous venous anatomy. There is only one case in literature similar to ours, which was between the SMV and aorta.[5] The distribution of cases is represented in ►**Fig. 13**. Treatment options include

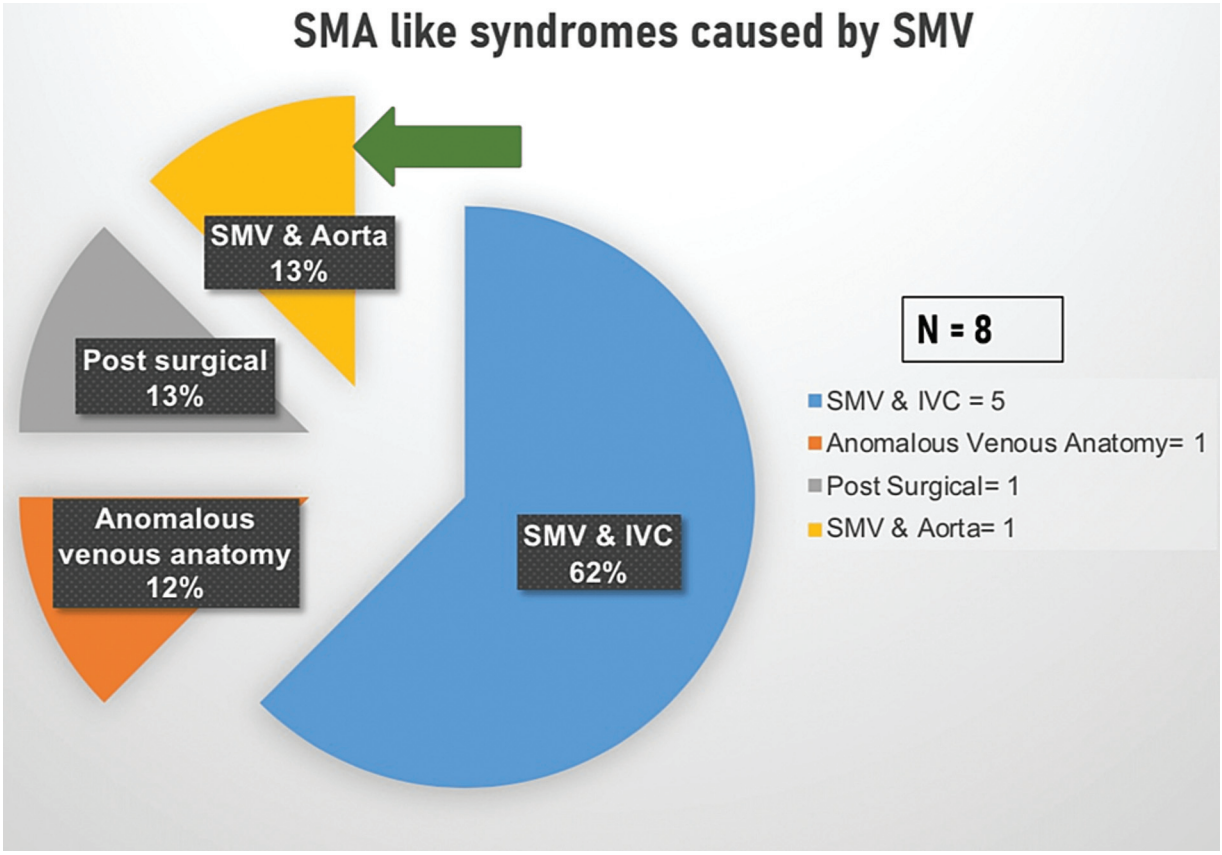

Fig. 13 Distribution of various SMA-like syndromes caused by SMV. IVC, inferior vena cava; SMA, superior mesenteric artery; SMV, superior mesenteric vein.

Table 1 Comparison of all reported cases of testicular vein syndrome (including current case [Hamidi et al 2017[8]])

Cases	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9
Publication	Mellin et al 1974	Krestwoki et al 1977	Lassing et al 1978	Meyer et al 1992	Ugurel et al 2005	Gupta and colleagues 2011	Tiwari et al 2011	Hamidi 2017	Current case
Age	28 y	20 y	Young adult	42 y	54 y	37 y	55 y	27 y	47 y
Site	Right	Left	Left	Right	Right	Left	Right	Left	Left
Level	L4	L2/L3 disc	NA	L3	L3	L3	L3	L2/L3 disc	L4

Abbreviation: NA, not available.

conservative (gravitational maneuvers) such as postprandial prone knee chest/left lateral decubitus positioning, nutritional augmentation such as multiple small feedings, parenteral hyperalimentation, and surgical bypass.[3] The second case is a case of testicular vein syndrome. The risk factors include a congenitally enlarged testicular vein, thrombophlebitic testicular vein and varicocele of testicular vein. Eight cases have been previously reported in the literature.[6–8] The patient details are given in ►**Table 1**. The average age of these patients was 37.5 years. Fifty-six percent of the patients had left-sided testicular vein syndrome and 44% had right-sided testicular vein syndrome. Compression was commonly encountered at the L3 vertebral level (50%), followed by L2-L3 disc level (25%) and L5 vertebral level (25%). The treatment includes resection/transection of the vein at the crossing point ± excision and ureteroureterostomy if the ureteral segment at the crossover is atretic. Follow-up for these patients were done clinically and with intravenous pyelography between 3 and 5 months postsurgery.[8] The third case is a case of giant fecal impaction causing deep vein thrombosis. The risk factors include Hirschsprung's disease, Chagas disease, diabetic neuropathy, neuropsychiatric diseases, inflammatory and neoplastic diseases, scleroderma, anorectal malformations, chronic bedridden patients, and long-term drugs like antidepressants and opioids.[9] Multiple such cases have been reported in the literature. The common

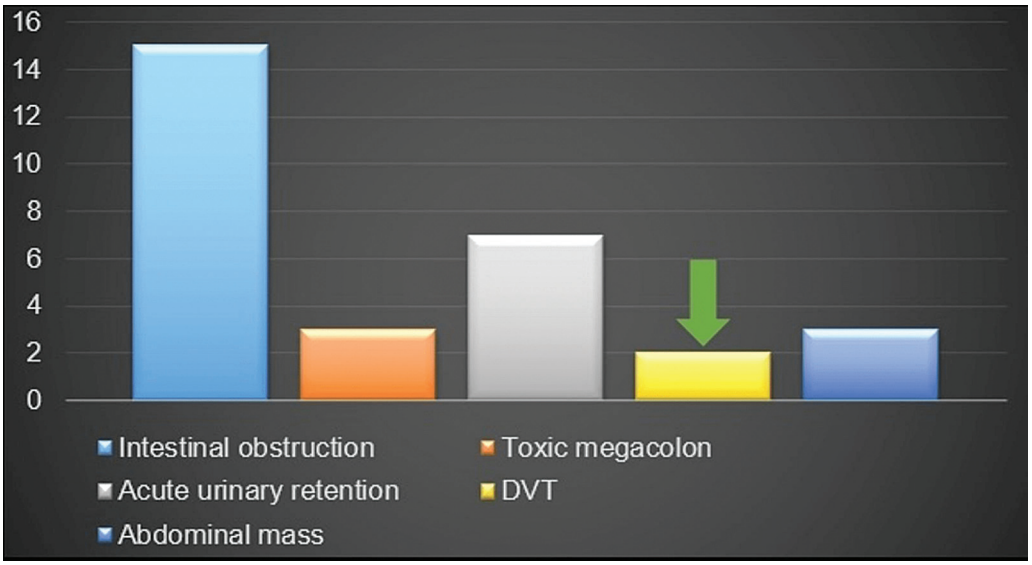

Fig. 14 Distribution of various acute presentations of fecal impaction/fecaloma. DVT, deep vein thrombosis.

acute presentations of fecal impaction so far published in the literature include intestinal obstruction,[10] followed by acute urinary retention,[11] abdominal mass,[12] and toxic megacolon, as shown in ►**Fig. 14**.[13] Deep vein thrombosis is an uncommon acute presentation of fecal impaction with the first ever reported case being a pediatric case.[14] Ours is the second such presentation reported so far. Treatment options include conservative management like laxatives, enema, and manual evacuation. Colonoscopy-guided fragmentation of fecal matter is indicated in some cases. Surgical intervention is mandated if complicated by bowel obstruction, toxic megacolon or if there is an underlying cause of Hirschsprung's disease.[15] Another interesting management that is tried is colonoscopic instillation of Coca-Cola.[16]

Conclusion

In conclusion, the intricate nature of abdominopelvic vascular and hollow viscus compression syndromes underscores the importance of vigilance and thorough evaluation in clinical practice. These syndromes, although rare, present a diagnostic challenge due to their diverse manifestations and potential for mimicking other common conditions. The cases presented here demonstrate the significance of advanced imaging techniques in establishing accurate diagnoses and guiding appropriate management strategies.

Additionally, these cases serve as valuable learning experiences, contributing to the expanding knowledge base surrounding these rare syndromes. Ultimately, a thorough understanding of abdominopelvic vascular and hollow viscus compression syndromes, coupled with a proactive approach to diagnosis and management, is essential for mitigating morbidity and mortality associated with these conditions. Continued research and clinical observations will further enhance our understanding and refine treatment approaches, ultimately improving the quality of care for affected patients.

Conflict of Interest
None declared.

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