Pylephlebitis of a variant mesenteric vein complicating sigmoid diverticulitis

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ABSTRACT

Pylephlebitis - suppurative thrombophlebitis of the portal and/or mesenteric veins - is a rare complication of abdominal infections, especially diverticulitis. It can lead to severe complications such as hepatic abscess, sepsis, peritonitis, bowel ischemia, etc., which increase the mortality rate. Here we present a case of suppurative thrombophlebitis of the inferior mesenteric vein, as a complication of sigmoid diverticulitis. The epidemiology, clinical and radiological features as well as treatment strategies are discussed. We also review the anatomy of the mesenteric vein given its anatomic variation in the present case and how this anatomic knowledge might influence the operative approach should surgery be necessary.

CASE REPORT

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A 60-year-old woman presented with a 7-day history of crampy abdominal pain associated with fever, chills, night sweats, lack of appetite, nausea and vomiting. On admission, the abdomen was soft, bowel sounds were normal, but rebound tenderness was noted in both lower quadrants. Laboratory findings included a normal leukocyte count, alanine aminotransferase 55 U/L (normal, <50), alkaline phosphatase 190 U/L (<130), and C-reactive protein 301 mg/L (<10). Computed tomography (CT) showed a contained sigmoid perforation in the setting of diverticulitis (Figure 1) and thrombosis of the inferior mesenteric vein. The venous wall showed subtle contrast enhancement and intraluminal air. There was stranding of the surrounding fatty tissue and localized lymphadenopathy (Figure 2). An anatomic variant of the inferior mesenteric vein was present, i.e. drainage into the

superior mesenteric vein 6mm distal to the portal confluence (Figure 3 and 4). Blood cultures grew Escherichia coli, susceptible to multiple antimicrobial agents. Because the patient was clinically stable, a conservative therapeutic approach with intravenous antibiotics and anticoagulation was taken. Over the following days, however, abdominal pain worsened, the fever persisted, and leukocytosis developed. A follow-up CT showed hepatopetal extension of the thrombosis with involvement of the superior mesenteric vein and increasing stranding of the fat tissue adjacent to the sigmoid colon (Figure 5). Left hemicolectomy was performed. Intraoperatively and histologically the diagnosis of acute diverticulitis with pylephlebitis of the inferior mesenteric vein was confirmed (Figure 6). The postoperative course was uneventful and the patient was discharged on oral antibiotics and low molecular weight heparin.

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DISCUSSION

Epidemiology and pathogenesis

Rudolf Virchow is often quoted of having been first, in 1856, to characterize three factors contributing to the pathogenesis of venous thrombosis, often referred to as the "Virchow triad" [1]: (1) alterations in blood flow (stasis), (2) vascular endothelial injury, and (3) alterations in the constitution of blood (hypercoagulable state). Pylephlebitis refers to septic (synonyms: suppurative or infectious) thrombosis of the portal vein and/or its branches [2, 3]. The inferior mesenteric vein alone is affected in 6% of cases of mesenteric vein thrombosis [4]. Predisposing factors include coagulation disorders, portal hypertension (e.g. due to cirrhosis), malignancy, postoperative dehydration, sepsis, and trauma [4-9] - all these factors may contribute to Virchow's famous triad. During infection the thrombosis might be mediated via the release of thrombogenic factors in the setting of inflammation and/or bacterial invasion [4, 8, 10, 11].

Symptoms of pylephlebitis are often non-specific including fever, chills, abdominal pain, nausea, abdominal tenderness and elevated blood markers of inflammation, such as leukocytosis and elevated C-reactive protein [2, 3, 6, 8, 12, 13]. In advanced stages with infection of the portal vein and liver abscess jaundice has been reported [3, 6, 12].

Underlying conditions in patients with pylephlebitis include diverticulitis (30%, mostly sigmoid), appendicitis (19%), inflammatory bowel disease (6%), pancreatitis (5%), infectious enteritis (4%), bowel perforation, and malignancies (6%) [3, 4, 6-8, 13]. Although diverticulitis is the most common cause of pylephlebitis, pylephlebitis is a rare complication of diverticulitis (3%) [14].

No age predilection has been reported, thus pylephlebitis can be seen in all age groups, from neonates to adults [3, 8].

<u>Anatomy</u>

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The inferior mesenteric vein (IMV) emerges from small veins that drain the sigmoid colon. It runs medial to the descending colon gathering its draining veins within the mesocolon in the left anterior pararenal space. Here it is usually located lateral to the superior and inferior mesenteric artery and anterior to the left ureter and gonadal veins (Figure 3). It passes behind or lateral to the duodenojejunal junction. Finally it drains either into the splenic vein, the confluence of splenic vein and superior mesenteric vein, or into the superior mesenteric vein (SMV) [15-17]. Krumm et al analyzed 916 abdominal CT-scans to document anatomic variations of the IMV [17]. These authors found that the IMV typically drains into the splenic vein (approx. 40%), into the portal confluence (approx. 30%), or into the SMV (approx. 20%) (Figure 4) these anatomic details were best seen on coronal images. This knowledge is important for gastrointestinal surgery, e.g. transplantation planning, pancreatic cancer surgery, etc. Another interesting point in this study was the small distance to the portal venous system from where the IMV drained either into the splenic vein (1.66cm) or into the SMV (0.75cm), respectively [17]. In the present case this latter distance is 0.6cm. Such anatomic knowledge is also important for color

duplex sonography; the examiner has to take care to visualise the portal confluence and its surroundings including the draining vessels. Otherwise thrombosis, cancerous invasion, etc. might be missed.

Diagnosis and differential diagnoses

On plain abdominal radiographs suppurative mesenteric vein thrombosis is not detectable. In our case the thrombosis was associated with intraluminal gas on abdominal CT, which was suggestive of suppurative thrombophlebitis. Intraluminal gas in the portal venous system may indicate an inflammatory illness but is mostly associated with an ischemic cause [18]. Thus it is not specific for thrombosis. In addition, extrahepatic intraluminal gas on plain films may be difficult to diagnose. Bear et al described a case of a patient who underwent barium enema with intravasation of barium into the IMV in the setting of IMV thrombosis, which was initially attributed to a coloureteral fistula [19]. Intrahepatic gas may be easier to diagnose on plain radiographs than extrahepatic gas, but needs to be distinguished from biliary air.

Color duplex sonography has the advantages of using neither radiation nor contrast; using this method, IMV thrombosis may be suggested by a flow defect, dilatation (normal IMV diameter: 0,5 cm [17]) or absent compressibility of the vein. The utility of duplex sonography is often limited by the presence of overlying bowel gas [6, 7].

On unenhanced CT or MRI images the diagnosis of thrombosis is difficult to make [7]. Findings indirectly suggestive of IMV thrombosis include dilatation of the vein [4], perivascular inflammation or/and lymphadenopathy. With contrast media the filling defect due to the thrombosis can be directly seen on CT / MRI as a central hypodense / hypointense thrombus compared to the peripheral hyperdense / hyperintense contrast media [4, 15]. Differential diagnosis in this constellation includes mixing artifact (contrasted blood mixing with non-contrasted blood). This usually occurs in the inferior vena cava [20] and would not be associated with the other imaging findings described above.

Prognosis

Before 1990 the mortality rate of patients with pylephlebitis was approximately 75%. Improved diagnostic imaging methods and therefore, more rapid institution of broad spectrum antibiotics could be the reason for the lower mortality rate of approximately 25% observed in reports published after 1990 [7, 8, 12]. The cause of death is typically sepsis, followed by peritonitis, bowel ischemia, intestinal bleeding, or rupture of the portal vein [6, 8]. Therefore, early detection of mesenteric thrombosis may have important prognostic implications. Complications may include thrombosis extension and/or septic emboli spreading to the portal vein and its intrahepatic branches with subsequent hepatic abscesses [6, 7, 11-13]. Such hepatic complications may radiologically be suggested by unopacified intrahepatic branches and central or peripheral zones of low attenuation secondary to a decreased intrahepatic blood flow [7, 11].

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<u>Treatment</u>

As with suppurative thrombophlebitis involving other veins, pylephlebitis typically is accompanied by bacteremia. Blood cultures have been positive in 77% of patients in one series [8] and in 88% of patients in another series [3]. Blood cultures most often have grown Bacteroides fragilis, followed by Escherichia coli (as in our case), viridans streptococci, Proteus mirabilis and Klebsiella pneumoniae [3, 6, 8, 11-13]. Empirical therapy should therefore be given with broadspectrum antibiotics targeting gram positive, gram negative as well as anaerobic bacteria [2, 6, 12].

The value of anticoagulant therapy is controversial [3, 6, 11, 12] since successful resolution of pylephlebitis has been reported with or without anticoagulation [2, 6, 8]. Baril et al suggested that patients with pylephlebitis and a hypercoagulable state, e.g. protein S deficiency [5], should be anticoagulated [9]. Plemmons et al indicated that anticoagulation might benefit patients by preventing liver abscesses by decreasing septic embolization to the liver from infected thrombi [3]. Kanellopoulou et al analyzed 100 cases of pylephlebitis in the literature and found that patients with anticoagulation therapy more often had a favourable outcome compared to patients who were treated with antibiotics alone [8]. These authors observed a significantly lower mortality rate, a higher recanalization rate, and a lower rate of development of portal hypertension in patients who also received anticoagulant therapy [8].

Suppurative thrombophlebitis is typically considered an indication to treat the underlying infectious process surgically [6, 12], particularly if intestinal or mesenteric ischemia occurs, or when conservative medical therapy fails [2, 6].

TEACHING POINT

Pylephlebitis is rare and usually occurs as a complication of a common intra-abdominal infection, e.g. diverticulitis or appendicitis. The associated mortality rate is approximately 25%. Therefore, assessment of the intra-abdominal veins should routinely be done when interpreting abdominal CT images; the clot causes a central filling defect and a peripherally contrasted vein on enhanced images.

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FIGURES

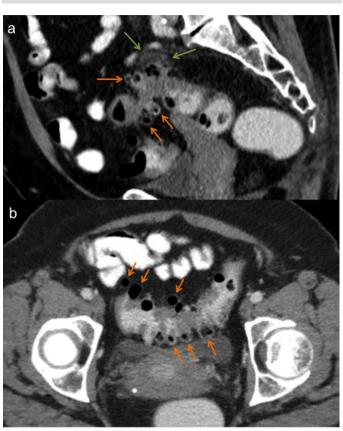


Figure 1: 60-year-old woman with pylephlebitis of the inferior mesenteric vein due to sigmoid diverticulitis. Sagittal (a) and axial (b) images of the initial abdominal CT show multiple diverticula of the sigmoid colon (orange arrows) as well as a surrounding inflammatory reaction (green arrows). Images obtained by a multidetector scanner; Protocol: 120 kV, with a max. of 230 miliamperes; slice thickness, 2.5 mm axial and 3mm sagittal; intravenous contrast medium iomeprol 400 mg/mL; total contrast dose, 80 ml; oral and rectal contrast medium megluminioxitalamat 300 mg iodine/mL; total contrast dose, 30 mL

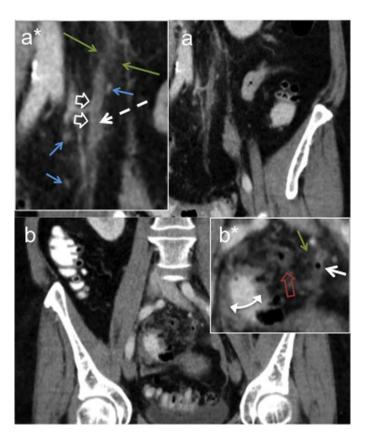


Figure 2: 60-year-old woman with pylephlebitis of the inferior mesenteric vein due to sigmoid diverticulitis. Coronal images (a and a*) of the initial abdominal CT show pylephlebitis with a central hypodense filling defect (dashed arrow) and peripheral hyperdensity (open arrows), lymphadenopathy (blue arrows) and inflammatory changes (green arrows), in the setting of diverticulitis seen in (b and b*) with wall thickening of the sigmoid colon (double arrow), a diverticulum (red open arrow) and gas in the thrombosed inferior mesenteric vein (white arrow) with surrounding edema (green arrow). Images obtained by a multidetector scanner; Protocol: 120 kV, with a max. of 230 miliamperes; slice thickness, 3 mm; intravenous contrast medium iomeprol 400 mg/mL; total contrast dose, 80 mL; oral and rectal contrast medium megluminioxitalamat 300 mg iodine/mL; total contrast dose, 30 mL

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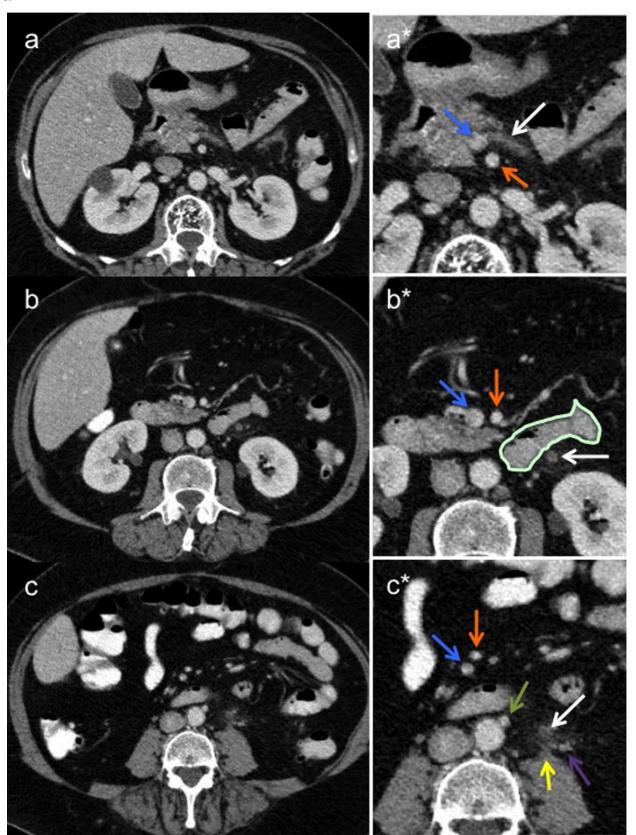


Figure 3: 60-year-old woman with pylephlebitis of the inferior mesenteric vein due to sigmoid diverticulitis. Initial abdominal computed tomography showing the anatomy of the inferior mesenteric vein (white arrow) compared to other abdominal structures on axial images. The inferior mesenteric vein (a and a*) shows, in this case, a variant drainage into the superior mesenteric vein (blue arrow). (b and b*) It passes behind the duodenojejunal junction (light green). (c and c*) The inferior mesenteric vein is located laterally to the superior (orange arrow) and inferior mesenteric artery (green arrow) and anterior to the left ureter (yellow arrow) and gonadal veins (purple arrow). Note the inflammatory stranding surrounding the inferior mesenteric vein due to the thrombosis, especially in c and c*. Images obtained by a multidetector scanner; Protocol: 120 kV, with a max. of 230 miliamperes; slice thickness, 2.5 mm; intravenous contrast medium iomeprol 400 mg/mL; total contrast dose, 80 mL; oral and rectal contrast medium megluminioxitalamat 300 mg iodine/mL; total contrast dose, 30 mL

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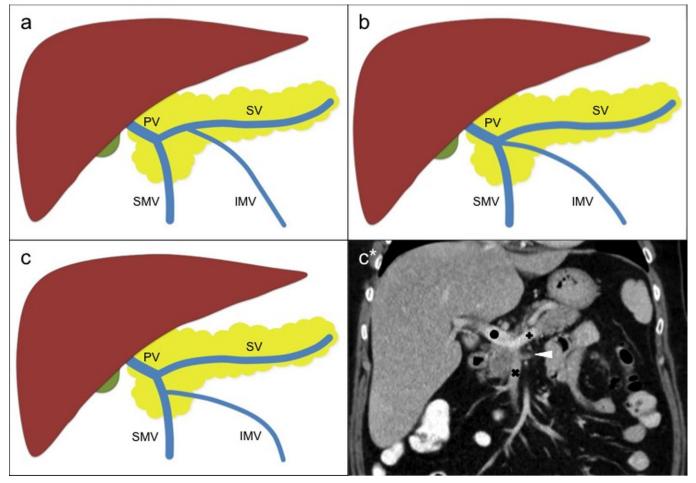
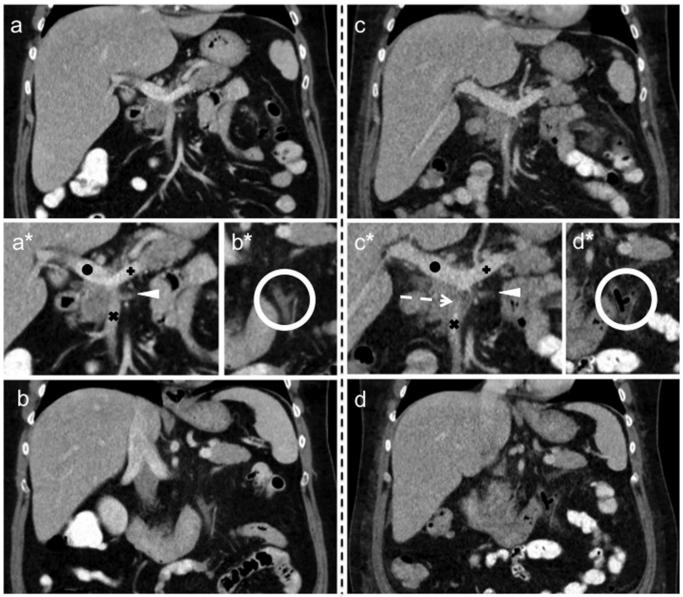


Figure 4: Anatomic variants of the inferior mesenteric vein (IMV): (a) the IMV most often drains into the splenic vein (SV); (b) sometimes it drains into the portal confluence; (c) rarely drainage of the IMV into the superior mesenteric vein (SMV) can be found. Coronal computed tomography image (c*) shows the third possible variant of the IMV (white arrowhead) draining into the SMV (x) 6mm distal to the portal confluence in a 60-year-old woman with pylephlebitis of the inferior mesenteric vein due to sigmoid diverticulitis (dot: portal vein; plus: SV).

Images obtained by a multidetector scanner; Protocol: 120 kV, with a max. of 230 miliamperes; slice thickness, 3 mm; intravenous contrast medium iomeprol 400 mg/mL; total contrast dose, 80 mL; oral and rectal contrast medium megluminioxitalamat 300 mg iodine/mL; total contrast dose, 30 mL

All illustrations prepared by Anna L. Falkowski.

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Figure 5: 60-year-old woman with pylephlebitis of the inferior mesenteric vein due to sigmoid diverticulitis. In the initial CT (coronal images, left column: a and a^{*}, b and b^{*}) there was no thrombosis or intraluminal gas seen in the veins of the upper abdomen; (a and a*) shows the variant influx of the inferior mesenteric vein (white arrowhead) into the superior mesenteric vein (x). The splenic vein (+) and the portal vein (dot) are also shown. Images obtained by a multidetector scanner; Protocol: 120 kV, with a max. of 230 miliamperes; slice thickness, 3 mm; intravenous contrast medium iomeprol 400 mg/mL; total contrast dose, 80 mL; oral and rectal contrast medium megluminioxitalamat 300 mg iodine/mL; total contrast dose, 30 mL Follow-up abdominal computed tomography (coronal images, right column), obtained four days after the initial CT; (c and c*) shows progression of mesenteric thrombosis (dashed arrow) of the inferior mesenteric vein (white arrowhead) into the superior mesenteric vein (x); (d and d*) shows the drainage of a vein adjacent to the left colonic flexure (circle) into the inferior mesenteric vein, with intraluminal gas present - note the progression compared to (b and b*). Images obtained by a multidetector scanner; Protocol: 120 kV, with a max. of 153 miliamperes; slice thickness, 3 mm; intravenous contrast medium iomeprol 400 mg/mL; total contrast dose, 80 mL; oral and rectal contrast medium megluminioxitalamat 300 mg iodine/mL, total contrast dose, 30 mL

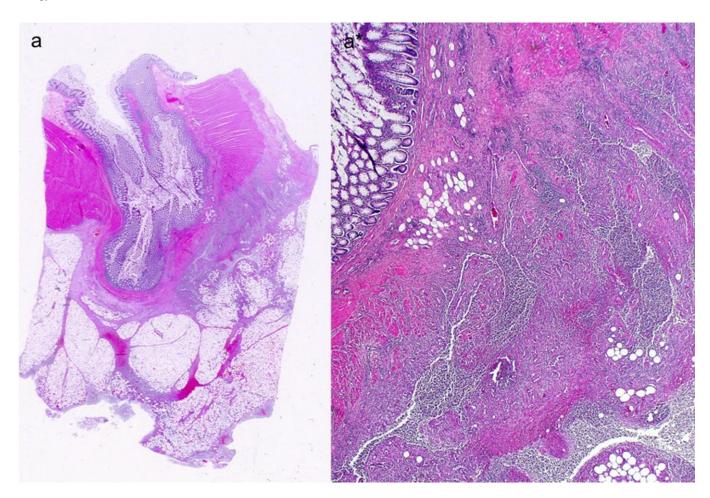


Figure 6: 60-year-old woman with pylephlebitis of the inferior mesenteric vein due to sigmoid diverticulitis. (a) Diverticulosis with acute peridiverticular inflammation (Hematoxylin and eosin stain; Cross-section original size). (a*) Magnification view (original magnification x 25) shows acute peridiverticular inflammation.

Etiology	Virchow's triad: (1) alterations in blood flow (stasis), (2) vascular endothelial injury, and (3) alterations in the constitution of blood (hypercoagulable states);	
	E.g. coagulation disorders, portal hypertension (e.g. due to a cirrhosis), malignancy, postoperative	
	dehydration, sepsis, and trauma.	
Incidence	Rare. Although diverticulitis is the most common cause of pylephlebitis, pylephlebitis is a rare	
	complication of a diverticulitis (3%) [14].	
Gender ratio	Unknown.	
Age predilection	None: from neonates to adults [3, 8].	
Risk factors	Diverticulitis (30%, mostly sigmoid), appendicitis (19%), inflammatory bowel disease (6%, e.g. Crohn's	
	disease, ulcerative colitis), pancreatitis (5%), regional enteritis (4%), bowel perforation, or even	
	malignancies (6%) [3, 4, 6-8, 13].	
Treatment	Broadspectrum antibiotics [2, 6, 12], anticoagulation (controversial) [3, 6, 12], surgery [6, 12].	
Prognosis	Mortality rate of approx. 25% after 1990 [7, 8, 12].	
Imaging findings	Central filling defect due to the thrombosis is directly seen with contrast media [4, 15].	

Table 1: Summary table of pylephlebitis

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Diagnosis	Pylephlebitis	Artifacts
X-Ray	 <i>thrombosis</i>: not detectable; <i>indirect signs</i>: gas in the vein 	• <i>non-venous gas</i> : extrahepatic (e.g. bowel perforation, coloureteral fistula), intrahepatic (e.g. biliary air, abscess)
Ultrasound	 <i>thrombosis</i>: no flow; <i>indirect signs</i>: dilated vein, missing compressibility of the vein 	 incorrect Doppler setting, reverberation artifact, overlying bowel gas artifacts
СТ	 <i>thrombosis</i>: isodense (not clearly detectable); <i>indirect signs</i>: dilated vein, perivascular inflammation and/or lymphadenopathy, gas in the vein 	• <i>mixing artifact</i> (with contrast media): heterogeneous
MRI	 <i>thrombosis</i>: T1: hyperintense, T2: hyperintense, no flow void on spin echo sequences, steady state free precessing sequence: low signal intensity of vein with susceptibility artifacts from gas inside the vessel <i>indirect signs</i>: dilated vein, perivascular inflammation and/or lymphadenopathy, 	• <i>mixing artifact</i> (with contrast media): heterogeneous
Contrast enhancement	• central filling defect and peripheral contrast	• <i>mixing artifacts</i> (see above): due to contrasted blood mixing with non-contrasted blood usually occurs at vessel confluxes

Table 2: Differential diagnosis table of imaging findings for pylephlebitis

ABBREVIATIONS

CT = Computed tomography IMV = inferior mesenteric vein MRI = Magnetic resonance images PV = portal veinSMV = superior mesenteric vein SV = splenic vein

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KEYWORDS

pylephlebitis; mesenteric thrombosis; mesenteric vein variant; inferior mesenteric vein; sigmoid diverticulitis; CT