Comments and illustrations of the WFUMB CEUS liver guidelines: Rare vascular pathology, part II

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Abstract

In this second part of the topic the hepatic pseudoaneurysm, hepatic infarction, and pylephlebitis are discussed as acute and potentially life-threatening hepatic vascular diseases. The focus is on their appearance on B-mode ultrasonography, duplex ultrasonography, and contrast-enhanced ultrasonography. Zahn's pseudo infarction is an important differential diagnosis to wedge-shaped hepatic infarction in this context. Knowledge of the data should help raise awareness of these rare findings, to come up with relevant differential diagnoses in the corresponding clinical situation, to interpret the ultrasound images correctly and thus to initiate the appropriate diagnostic and therapeutic steps in time.

Keywords: hepatic artery pseudoaneurysm; liver infarction; pylephlebitis; Zahn´s pseudo infarction; Ultrasonography

Introduction

The World (WFUMB) and European Federation for Ultrasound in Medicine and Biology (EFSUMB) has developed guidelines for the indication and use of contrast-enhanced ultrasound (CEUS) for the evaluation of focal liver lesions [1-5]. Improved detection and characterization of common focal liver lesions (FLL) are the main topics of these guidelines. In recent years, conventional ultrasound (US) and CEUS features of less common FLL have been described in detail.

In this current paper series on comments and illustrations of the WFUMB and EFSUMB CEUS liver guidelines, we aim to summarize the US and CEUS features of very rare FLL where there are limited reports and figures published in order to create a library of these rare lesions. Hepatic artery pseudoaneurysm, liver infarction and pylephlebitis are acute life-threatening conditions. The outcome and possibly also the survival of the affected patient depends on the high competence of the examiner with fast correct diagnosis and target-oriented further intervention. Hepatic artery pseudoaneurysm and hepatic infarction may have common precipitating factors and may be interrelated. In both conditions, precipitating fac-

tors include surgery on the hepatobiliary system and pancreas and interventions on the biliary tract. Pylephlebitis is also a life-threatening situation. If portal venous gas is detected, a complex search must be carried out in the abdomen for an underlying cause, the mesenteric vessels must be carefully examined, and the small intestine wall must also be inspected for air pockets.

In this second part, acute vascular diseases are discussed [6]. This is a comprehensive presentation of acute situations such as hepatic artery pseudoaneurysm, liver infarction, pylephlebitis (with and without hepatic portal venous gas), and Zahn's pseudo infarction. Budd-Chiari syndrome is discussed elsewhere.

Hepatic artery pseudoaneurysm

A pseudoaneurysm of the hepatic artery is caused by a leakage of the vessel wall with blood flowing into an adjacent cavity. This is not a true aneurysm, but a false aneurysm or pseudoaneurysm that can be life threatening. The cavity communicates with the artery and is under high pressure with higher risk of rupture in comparison to true aneurysms (76 % vs. 3 %) [7].

Spontaneous aneurysms are very rare. Among 233 patients with visceral artery aneurysms observed over a 10-year period, 11% (22/196) of all true aneurysms and 42% (16/38) of all pseudoaneurysms originated from the hepatic artery [7]. A total of 17.5% hepatic artery pseudoaneurysms had a history of pancreaticoduodenectomy or biliary tract interventions [7]. A hepatic artery pseudoaneurysm may arise from mechanical, infectious/ inflammatory, or thermal alteration of the artery.

Hepatic artery pseudoaneurysm can develop following liver transplantation [8], but may occur with a frequency between 0.06% and 0.6% also after (laparoscopic) cholecystectomy [9-12]. Non-surgical reasons include blunt abdominal trauma [13], acute pancreatitis [14,15], and very rare infectious disease of the liver such as amoebic abscess [16-18]. Liver biopsy is a safe method, but rarely, the development of a pseudoaneurysm is also possible [19]. The development of hepatic artery pseudoaneurysm after biliary stenting has also been described [20,21].

Another rare etiology of hepatic pseudoaneurysm is percutaneous transhepatic cholangiography drainage (PTCD). Arterial complications including pseudoaneurysm may be caused by creation of access via needle puncture, bougienage, change of external drains, and other interventions. Large series report an incidence of arterial complications of PTCD of 0.6-1.8% with a higher risk in left hepatic access routes [22]. In chronic pancreatitis, a peripancreatic artery adjacent to an acute necrotic collection, walled-off pancreatic necrosis or a pseudocyst may be eroded by a combination of pancreatic enzyme digestion and mechanical injury. A recent meta-analysis reported a pooled incidence of visceral pseudoaneurysm in patients with chronic pancreatitis of 0.03% and in acute pancreatitis of 0.05% [23]. The risk is higher in acute necrotizing pancreatitis. In two large cohorts, incidence of visceral artery pseudoaneurysms was reported to be as high as 4.3% and 6.4%, respectively [24,25]. Interventional treatment of necrotic collections especially using lumen-apposing metal stents increases the probability of severe bleeding complications [25]. However, in the majority of cases, splenic artery and gastroduodenal artery are involved; hepatic artery pseudoaneurysm is a very rare occurrence in pancreatitis [23-25]

Sudden onset of pain with a circulatory reaction are typical presentations of pseudoaneurysms. However, these may be discovered incidentally on imaging in asymptomatic individuals. In a meta-analysis of hepatic artery pseudoaneurysms after laparoscopic cholecystectomy, 85% had hemobilia, 19.9% hematemesis, 9.9% jaundice, 9.9% abdominal pain, 5.0% anemia, and 5.0% melena. Mostly the right hepatic artery was involved, but can also include the cystic artery [10]. 80% of patients become symptomatic during the first 2 months after laparoscopic cholecystectomy [12].

Imaging

Pseudoaneurysms can mimic a solid or cystic lesion owing to the thrombotic reaction (fig 1). The thrombotic area is sometimes very large, and the aneurysm may be difficult to detect, particularly if color Doppler US is not utilized or a CT scan is performed without contrast enhancement. Even with contrast, the diameter of the pseudoaneurysms may be underestimated owing to the non-enhancement of the thrombotic part of the pseudoaneurysm. Duplex sonography shows pulsatile inflow and outflow into the pseudoaneurysm. In larger pseudoaneurysms, pulsations may already be apparent on B-scan ultrasonography. Sonographic detection of a hepatic artery pseudoaneurysm depends on size, location, and examiner experience. With appropriate history and clinical symptoms of pain, obstructive jaundice, hemobilia, or signs of gastrointestinal bleeding, hepatic pseudoaneurysm should be considered and specifically excluded. The classic presentation of a rupture of hepatic pseudoaneurysms into the bile duct (pain, jaundice and haemobilia) is called a "Quincke triad".

A very dangerous condition is the pseudocystic appearance of the pseudoaneurysm after acute pancreatitis. Drainage of the pseudoaneurysm into the stomach or duodenum can thus be fatal. Therefore, a color Doppler investigation is mandatory before endoscopic pseudocyst drainage. If a pseudoaneurysm of the hepatic artery is di-
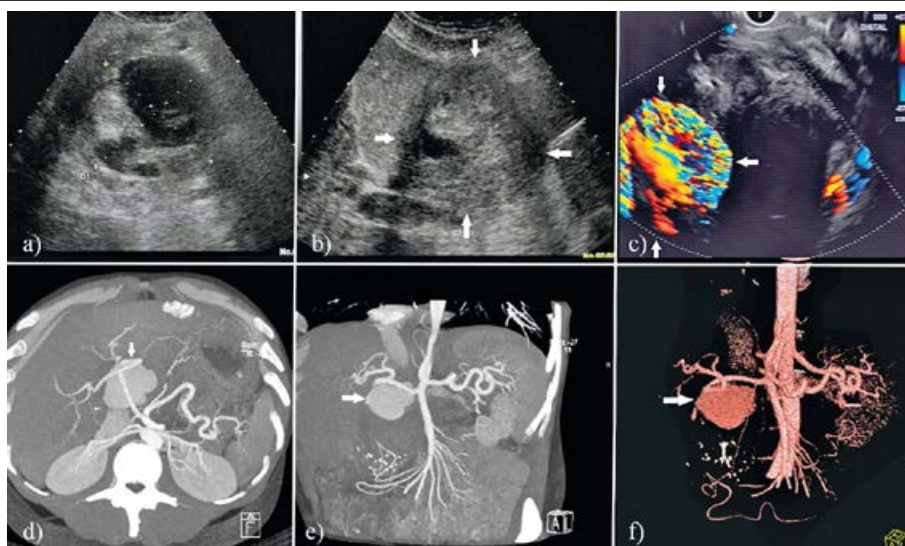
Fig 1. Pseudoaneurysm of the hepatic artery on transabdominal ultrasound (note the cystic appearance) (a) in the close proximity to the left liver lobe (b). Endoscopic ultrasound appearance on color Doppler mode; the hepatic artery is on the very left of the picture with the rupture and the false lumen next to it (c). CT angiogram of the hepatic artery with pseudoaneurysm (d) and in transversal reconstruction (e). 3-D-reconstruction of the angiogram (f). Image source of CT angiogram: Dr. H.-J. Hald, Helios Hospital Meiningen, Radiology

agnosed, an acute intervention is necessary owing to the threat of rupture [26]. Depending on the size and location of the pseudoaneurysm, different approaches can be used. Stenting, coiling, or embolization of the aneurysm—usually using angiographic methods—are the treatments of choice. However, emergency procedures such as percutaneous puncture with hemostatic substances or even the same procedure using endoscopic US have been reported [27]. In very large aneurysms, surgery to reconstruct the hepatic artery may be necessary.

Liver infarction

Liver infarction is a serious, although rare, vascular complication. It is rare because the liver is supplied with blood via two vascular systems. The development of a hepatic infarction is not only related to the arterial system, but also to the portal venous system and systemic circulatory injuries [28]. There are various pathogenetic factors: iatrogenic postoperative injuries of the hepatic artery, systemic diseases involving the hepatic artery and its branches, coagulation disorders but also the creation of a transjugular intrahepatic stent shunt (TIPS) or transarterial chemoembolization (TACE) procedure.

To understand the specificity of liver infarcts compared to other organs, it is important to understand the anatomical features. The arterial blood supply also takes place in different ways. In addition to the hepatic artery, there are connections from the coeliac trunk and the superior mesenteric artery and its branches. It is rare for all these vessels to be interrupted and occluded at the same time. [29]. However, if both the hepatic artery and the portal vein or their respective branches are occluded, the risk of liver infarction increases considerably [30].

In the study of Seeley et al, there were 19 liver infarctions in 700 autopsies diagnosed. The authors concluded that liver infarctions might be more frequent than clinically assumed. Ten autopsies had arterial occlusion, three of these patients had combined arterial and portal venous occlusion, four had only portal vein thrombosis without arterial occlusion, and five patients had no vascular occlusion. In three cases, massive hepatic infarction was the main cause of death. One of these cases had an arterial occlusion owing to a dissecting aneurysm of a branch of the hepatic artery. The second patient had venous thrombosis and the third had no vascular occlusion. In 16 cases, the hepatic infarction was not the cause of death but concomitant disease. An important cause of infarction was obstruction of the hepatic artery, especially distal to the origin of its gastroduodenal branch [31].

In the autopsy study of 20 patients with liver infarcts by Saegusa et al, 85% of the cases had previous circulatory insufficiency. In this study, the authors concluded that not only impaired blood flow in the portal system is serious for the development of liver infarction, but that a reduction in blood flow to the liver owing to circulatory insufficiency is an additional potential risk factor [32].

An infarction in a cirrhotic liver is a rare event. The rarity of liver infarcts is explained by the presence of collateral vessels within the liver [31].

The clinical symptoms of liver infarction are variable: right-sided upper abdominal discomfort, chest pain, fever, nausea and vomiting and liver laboratory elevations. Patients may also remain mild or asymptomatic [28,33-35]. In rare cases, liver infarction precipitates acute liver failure with high mortality rates [34].

The iatrogenic causes of liver infarction include damage of the right hepatic artery and portal vein associated with laparoscopic cholecystectomy, but also bile duct, liver and pancreatic surgery [36]. The risk of infarction is more severe with ligation of the hepatic artery distal to the junction of the right gastric artery [29].

In the study of Shindo et al, 19% of all patients undergoing pancreaticoduodenectomy developed hepatic infarction, with hepatic artery and celiac trunk resection being the most important risk factors [37]. Liver infarctions resulting from endovascular treatment of bleeding events after pancreaticoduodenectomy were described in 12.5% [38].

Systemic causes include eosinophilic granulomatosis with polyangiitis [39] and also polyarteritis nodosa [29, 30]. Vasculitis can cause irregular wall changes and stenosis of the hepatic arteries and thus can occasionally lead to hepatic infarction.

Hepatic infarction can complicate a HELLP syndrome (hemolysis, elevated liver enzymes and low platelets) and cause death in 16% of the women [40-42].

Systemic lupus erythematosus (SLE) and antiphospholipid syndrome with increased risk of thrombosis may be associated with an enhanced incidence of hepatic infarction in women during pregnancy or after delivery [43,44]. Severe bleeding postpartum may in turn cause hypercoagulable status with disseminated intravascular coagulation and lead to hepatic infarction [45]. Bacterial endocarditis, left ventricular thrombus and thrombosis of a liver metastasis from a bile duct carcinoma were described on autopsy as causes of hepatic artery thrombosis [29].

TIPS is performed in patients with liver cirrhosis to treat refractory ascites or variceal bleeding. TIPS can lead to hypoperfusion in other portal and hepatic veins and therefore, can contribute to liver infarction [28]. However, it is not known how often ischemia in the liver occurs after TIPS in a way that is clinically relevant [34]. The development of acute liver failure together with liver infarction after TIPS implantation has also been described as a rarity [46]. Other possible mechanisms for liver ischemia after TIPS are hepatic arterial problems such as thrombosis, pseudoaneurysms or arteriovenous fistulae, usually related to needle punctures during the procedure or rarely, to extrinsic compression of the hepatic artery by the stent [34]. Arterial occlusion can be poorly tolerated in patients with portal hypertension, as they are more dependent on the arterial circulation.

Lopera et al performed contrast-enhanced CT in 374 patients with TIPS. Of these, 10 patients had focal ischaemia or infarction in the liver. The percentages of liver infarction were 11–40% (mean, 21%) of the total liver volume. Thrombosis of branches of the PV was a common imaging finding after the procedure (anterior branch, 19%; posterior branch, 51%; left PV, 11%). Associated thrombosis of the hepatic veins was also very common [34]. The combination of portal and hepatic vein thromboses was a risk factor for the development of ischemia after TIPS creation [34].

TACE has traditionally been considered to be contraindicated in cases of portal venous tumor thrombosis, owing to the risk of hepatic infarction and further deteriorate liver function [47]. In the case of TACE and the presence of a transjugular shunt or TIPS, several risk factors are compounded [48].

After liver transplantation, most liver infarctions occur due to vascular typical complications [49,50]. Vascular complications in the early postoperative period occur in approximately 9% of liver transplanted patients. Complications involving the hepatic artery include thrombosis, stenosis and pseudoaneurysms. Another possible complication is the splenic artery steal syndrome [8,51-53]. They are the most common cause of graft loss.

Hepatic artery thrombosis after liver transplantation (HAT) has been reported in the literature in 4-11% of adults and in up to 40% of children [51]. Thrombosis of the hepatic artery led to liver infarction in 3% of adults and 12% of children [54]. Eighty-five percent of hepatic ischemia or infarction was secondary to untreated thrombosis or stenosis in the hepatic artery [51]. Portal vein complications after liver transplantation include portal vein thrombosis (PVT) and portal vein stenosis (PVS) with an incidence ranging from 1% up to 12.5% [8]. Liver transplant patients are at increased risk of hepatic infarction owing to loss of collateral pathways of the hepatic vascular system.

Other causes of liver infarction such as liver abscess [55,56], Clostridium difficile infection [57], acute pancreatitis [58] and sickle cell disease [59] have also been reported.

Imaging

The localization of hepatic infarction is frequently described in the right hepatic lobe [28,33,36,39,45,49,60], but can also occur in the left hepatic lobe [48] and may be multifocal particularly in the peripheries of both lobes [43,49,55,61]. Localization to the caudate lobe has also been reported [62]. Hepatic infarctions after pancreatoduodenectomy were most frequently seen in liver seg-
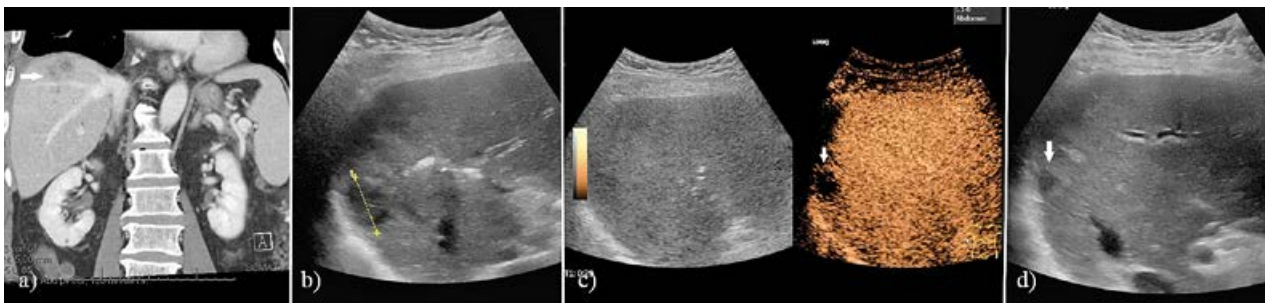
Fig 2. A 76 y/o male. Post choledochojejunostomy for choledocholithiasis. Owing to a complicated postoperative anastomosis, access to the bile ducts was percutaneous with PTCD placement of external-internal drains. During the recovery course, hemobilia developed, due to a small pseudoaneurysm of the hepatica propria artery. This was treated endovascularly with coils. Postinterventional CT showed a wedge-shaped hypodense area in the subdiaphragmatic parts of the liver (a), (Source of the CT image: Prof. Dr. Sven Mutze, Dr. Christopher Nauck: Sana-Hospital Berlin-Lichtenberg, Institute of Radiology). The tip of the lesion points in the direction of the coils. B-mode US showed a small oval echo-poor area subcapsular subdiaphragmatically in the right liver lobe (calipers) (b), avascular in CEUS (arrow) (c). In contradistinction to an abscess, no hyperenhancing capsule or of the surrounding areas were present. The wedge-shaped character of the lesion developed during the regressive phase (arrow) (d).

ment VII [37]. However, a previous study demonstrated that hepatic infarction caused by intraoperative damage to the hepatic artery and celiac artery commonly occurs in the left lobe [63].

Parenchymal lesions secondary to hepatic infarcts may be apparent on B-mode US. However, the diagnosis of an ischemic injury on B-mode US alone is difficult in many cases, especially immediately after the acute event [64]. Typically, they are described as wedge-shaped, well-circumscribed or rounded hypoechoic regions and maybe located in the periphery or centrally [49].

Lev-Toaff et al [43] reported the varied appearance of hepatic infarcts on US. Liver infarcts are expected to be wedge-shaped and peripheral according to the vascular branches. However, this was rarely the case. Mostly, the infarct areas were oval or round and distributed over the liver. The appearance of infarct areas on US was also dependent on the duration of their existence. Initially, many were hypoechoic. As they progressed, they became more sharply defined or confluent. Even liquefied areas may appear and gas bubbles. There are a wide range of other hypoechoic lesions from which infarct areas must be differentiated, including tumors and abscesses [35].

Analogous to splenic infarction [65], a so-called "Bright band sign" has been described in the liver [49]. The liver infarction cases of Whang et al showed echogenic bands within the infarcted portions of the liver on US, possibly reflections from the intact portal triads [49]. The authors suggested that the "Bright band sign" may a useful gray-scale US feature for liver infarction. These bright stripes may indicate the still intact architecture of the acinar zone surrounding the portal triads. However, the authors themselves estimate that further studies are still needed to assess the sensitivity and specificity of this sign [49].

Duplex sonography is used to assess flow in the hepatic artery, portal veins, and hepatic veins. The underlying etiology of a hepatic infarction i.e. occlusion or stenosis of the hepatic artery, an arterial pseudoaneurysm or vascular thrombosis can thus be diagnosed.

Von Herbay et al investigated the performance of CEUS in infarctions of various parenchymal organs. Whereas an ischemic zone was only visible on B-mode scan in a little more than half the cases, with CEUS, it was detected in all organs [64]. In one patient with ischemic areas in the liver, this was delineated as a non-enhanced area on CEUS. However, the definite diagnosis of ischemia was only established on a CT scan, which showed partial thrombosis of the coeliac trunk [64].

Typical characteristics of hepatic infarction in CEUS is non-enhancement in the arterial, portal venous and late vascular phases. The borders are smooth and unlike, for example, abscesses, there is no increased enhancement at the periphery [9,66] (fig 2). Anechoic lesions without enhancement in all vascular phases may also be seen with abscesses and liquefying hematomas. The entire history and paraclinical history are essential to aid the differential diagnosis. Abscesses often have a hyperenhancing capsule.

The 2012 WFUMB-EFSUMB guidelines and recommendations on the applications of CEUS to the liver [2,67] still recommend performing CEUS after liver transplantation for suspected liver infarction. This is to exclude perfusion abnormalities in the liver. In addition, fluid accumulation and collections can be detected and evaluated [9]. In the updated WFUMB and EFSUMB Guidelines 2020, this recommendation was not specifically formulated [4,5]. We also refer to the history of ultrasound in Europe and the first guidelines on CEUS [68,69].

CEUS can also be used to delineate more accurately vascular thromboses and confirm stenoses when duplex examination is inconclusive [8,9]. In most publications, the diagnosis of hepatic infarcts was made with contrast-enhanced CT (CE-CT). Typically, infarcted areas in the liver are nonenhanced in all phases even with this imaging method. The ischemia is usually diagnosed as a triangular perfusional defect on contrast-enhanced images. Depending on the localization at the periphery or centrally, the lesion may be wedge-shaped or oval [34,37, 56].

In a retrospective study including 19 patients with arterially related liver infarction, CT scans showed the infarct in variable appearances: hypodense lesions in the liver parenchyma that were triangular, round or oval, peripheral or central, sharply defined or irregularly configured. There were also isolated air reflections which were not secondary to infection or an abscess [70].

"Portal vein penetration sign" was observed in half of all patients with liver infarction after pancreatoduodenectomy. This sign revealed penetration of the portal vein with contrast enhancement in the infarcted area [37]

Treatment of infarction of the liver depends directly on the etiology. Anticoagulation was not generally performed and was dependent on the cause and clinical situation described.

Zahn's pseudo infarction

In Zahn's pseudo infarction, a wedge-shaped hyperemia zone of the liver parenchyma occurs because of the occlusion of a peripheral portal vein branch. It is not a true arterial infarction. The base of the wedge-shaped area is towards the liver surface. In the acute phase, the base of the wedge may be exophytic because of the congestion from the thrombotic changes in the portal vein branches or their compression from a tumor. Usually, a reduction in portal venous blood flow is compensated by increased arterialization which does not occur in these cases. Other causative factors may include a reduction in arterial perfusion (although the hepatic artery is intact), arterial hypotension and existing venous congestion with disrupted venous outflow can occur [71,72]. Such well-defined hyperemic areas in the liver were first described by Zahn in 1897 (Zahn, F.W. (1897). Verh. Dtsch. Naturf. Aertze). In the acute stage, sinusoidal congestion occurs, followedby parenchymal atrophy in the later stage [72]. Hepatocellular necrosis is not typical in Zahn's pseudo infarction. However, in the case of a massive arterial perfusion failure, centrilobular necrosis can also occur [72]. In addition to portal branch thrombosis owing to thrombotic events in the mesenteric and por-

tal venous circulation, splenectomy and severe cardiac insufficiency were reported [72,73]. Another case report described Zahn's pseudo infarction adjacent to a transjugular portosystemic shunt [74].

Imaging

Consequently, according to the pathological description, Zahn's pseudo infarct appears on B-mode US as a wedge-shaped lesion at the edge of the subcapsular region in the liver. One would expect hypoechogenicity. However, if stasis of blood occurs as described patho-anatomically, it is conceivable that the lesion may also be hyperechoic on US similar to diffuse non-liquid parenchymal hemorrhage. Feeding vessels can be visualized in duplex US. In the case of a wedge-shaped area at the liver periphery, not only should the arterial inflow be checked, but thrombosis of a small portal vein branch need to be specifically searched. Regarding pathogenic factors, thrombotic changes in the portal venous and splanchnic system should be sought, as well as any adjacent hepatic lesions and specifically in the presence of liver cirrhosis, hepatocellular carcinoma. On CEUS, wedge-shaped hypoenhancement can be expected in the presence of disrupted arterial perfusion and thrombosis of the supplying portal venous branch (fig 3, fig 4).

Pylephlebitis and gas within the portal vessels

Pylephlebitis is thrombosis and thrombophlebitis of the portal vein secondary to sepsis. It occurs because of severe inflammation in the abdomen or pelvis typically within the drainage areas of the mesenteric veins extending to the portal vein. Portal vein thrombosis alone does not correspond to pylephlebitis. The most serious and life-threatening etiology is small bowel infarction with small bowel gangrene. Septic portovenous embolization and consecutive liver abscesses are a complication of pylephlebitis. Possible causes include sigmoid diverticulitis, complicated appendicitis, infectious or chronic inflammatory bowel disease, pancreatitis and potentially any septic or bacterial associated abdominal infection. In severe inflammatory abdominal disease, pylephlebitis should be considered, especially if liver enzymes are abnormal [75,76].

In up to 77%, pathogens may be detected in blood culture or aspirates. In most cases, this was multiple pathogens and are typically Bacteroides, Escherichia coli, and Streptococcus species [75,77]. Pylephlebitis begins in the venous drainage area of the infected focus. The inflammation may then extend to the connected veins in the drainage area and finally affect the portal vein.

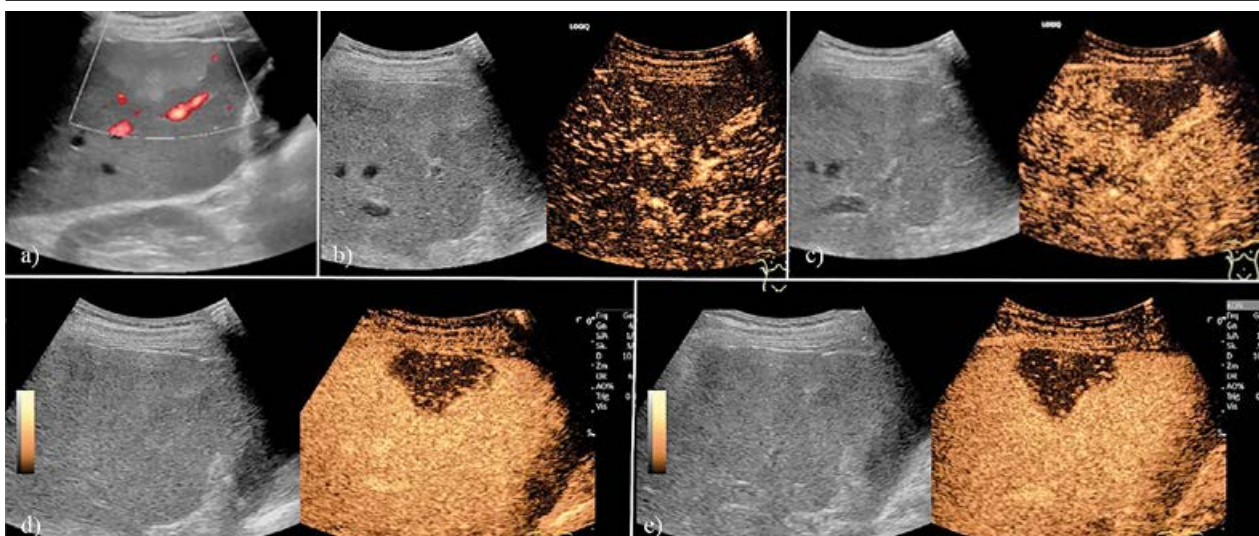A critical situation is gas within the portal vessels (hepatoportal venous gas, HPVG) (fig 5). This was con-

Fig 3. A 86 y/o male with Zahn´s - pseudo infarction, abdominal complaints and history of diffuse large B-cell non-Hodgkin's lymphoma. More than 10 years before, a partial resection of the small intestine had been performed owing to a small intestinal ileus. B-mode US showed a flat slightly round hyperechogenic subcapsular focal liver lesion in the right liver lobe (a). A hemangioma was suspected. CEUS showed a wedge-shaped non-enhanced mass in the arterial (b, c), portal venous (d) and late phase (e). The tip of the wedge points to the hepatic hilus. The findings did not correspond to a haemangioma. The anamnesis clarified that a mesenteric vein thrombosis was the cause of the small intestinal ileus. The findings were thus considered to be the residuum of an old pseudo infarction using follow up.
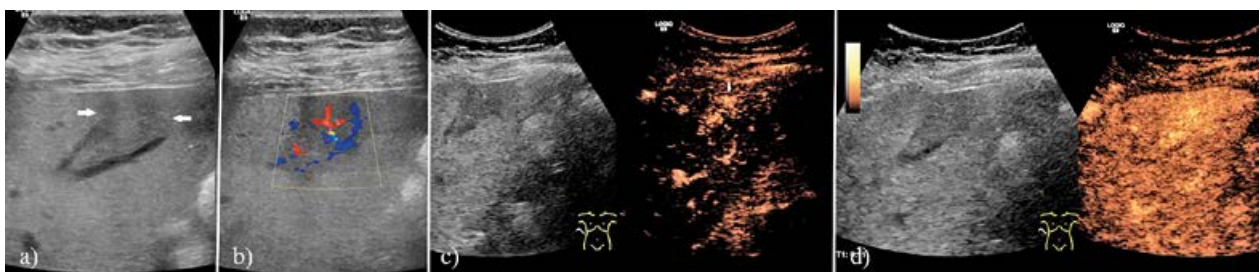

Fig 4. A 56 y/o, female. Upper abdominal discomfort. B-mode US demonstrates a wedge-shaped subcapsular hypoechoic area (a), suggestive of infarction or Zahn's pseudo infarction. Duplex US shows a single wheel spoke-like area in the center of the wedge (b). CEUS shows early arterial radicular flooding of a FNH (c). Still in the early arterial phase, the wedge-shaped area is more contrasted than the surrounding area (d). Thus, infarction or Zahn's pseudo infarction were excluded. An arterioportal fistula in the setting of a very small FNH was likely.

sidered a particularly severe form of the disease, in which small bowel gangrene must be excluded. Liebman et al still [78] described a proportion of intestinal gangrene of 73% and mortality of 75% with HPVG [78]. Kinoshita et al reported 182 cases of hepatoportal venous gas [79], the overall mortality was 39%. The most common cause of HPVG was bowel wall necrosis. At 43%, this affected almost half of all cases and 75% of these patients died. Dilatation of the gastrointestinal tract of various etiologies and intra-abdominal abscesses were also not uncommon (12% and 11% respectively), and should be at the top of the differential diagnoses.

Less frequent were ventricular ulcers, chronic inflammatory bowel diseases (ulcerative colitis and Crohn's disease), endoscopic interventions or the presence of abdominal tumours. Mortality rates were intermediate for a number of other conditions: 30% for patients with abscesses, 25% for peptic ulcers, and 21% for dilatation of the digestive tract. Fortunately, there were no deaths in cases of inflammatory bowel disease, pancreatitis, cholangitis, hepatitis with HPVG. Here, the prognosis of HPVG seems to be better [79]. The data is consistent with that of a large single institution series of cases presenting with HPVG in an Emergency Department. Overall, mortality of the 50 HPVG patients was 56%, varying between none in patients without shock or intestinal pneumatosis and 84% in patients with shock and intestinal pneumatosis. Both conditions were significant predictors of a worse outcome with an Odds Ratio of 17 for shock and 5 for intestinal pneumatosis [80].

According to our experience and the data, gas in the portal venous system is not necessarily associated with fatality if the underlying cause is suitably treated. Air is thought to enter the capillary veins from the intestinal lumen either by a disrupted epithelial barrier or by increased intraluminal pressure. In this way, mucosal lesions in Crohn's disease and ulcerative colitis can lead to air in the portal vein, as can the performance of a colonoscopy [81-83].

Liver enzymes are usually elevated in pylephlebitis. In case of bowel wall necrosis, LDH and lactate are also increased. In addition, C-reactive protein (CRP) is elevated depending on the underlying etiology.

Imaging

The purpose of imaging is to diagnose pylephlebitis, the cause of septic abdominal disease and complications. On B-mode US, the portal vein and its branches are filled with hypoechoic material. As a result of acute thrombosis, the lumen may be dilated. Thrombosis of the mesenteric veins and splenic vein may also develop and thus a lack of flow on Duplex US lacks. Ultrasound Assessment will be hampered in the presence of small bowel ileus. As a rule, CT is performed in these critically ill patients.

Gas bubbles in the portal system appear as small echogenic reflections with small dorsal reverberation artifacts. Portal venous gas must be differentiated from aerobilia (air in the bile ducts). HPVG continuously moves the liver periphery, while aerobilia is present in the bile duct branches and segmental branches. Portal venous gas is not only visible in the main portal vessels but the small gas reflections can be seen seen diffusely throughout the liver parenchyma. This is a result of the air expanding with the blood flow into the smallest portal venous vessels and sinusoids.

CEUS can be helpful in the evaluation of thrombosis but also septic complications such as liver abscesses (fig 6). Another possible indication is the early detection of
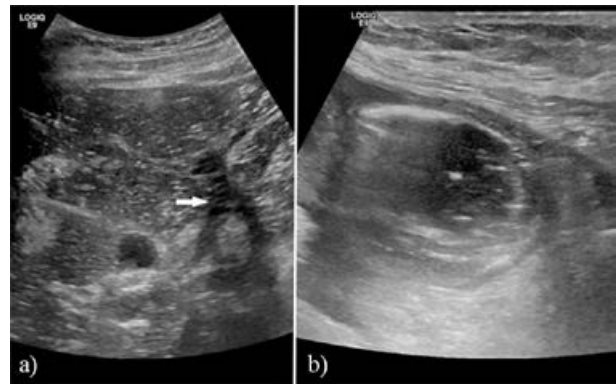

Fig 5. Pylephlebitis. A 66 y/o female presented with vomiting and abdominal pain owing to small bowel obstruction secondary to peritoneal metastatic ovarian cancer. B-mode US shows multiple lenticular air reflections diffusely distributed throughout the liver, including in the portal vessels such as the pars umbilicalis visible here (arrow) (a). Pneumatosis intestinalis could not be proven with certainty but could not be excluded either (b). In a highly palliative situation, conservative therapy was carried out with the insertion of a 20 CH outflow PEG. The patient was cared for in the palliative ward for several weeks. Overinflation of the gastrointestinal tract and stomach can also lead to air in the portal vein.

acute mesenteric ischemia and transmural intestinal infarction when the thrombosis extends into the superior mesenteric vein [84]. In this case, mesenteric ischemia is caused by SMV thrombosis. CEUS can also be used to verify nonenhancement of gangrenous intestinal bowel loops in cases of acute mesenteric arterial occlusion [85-88].

Treatment of patients with HPVG and in particular indications for emergency surgery should be directed to the underlying disease. The high mortality rate in HPVG associated with intestinal necrosis must be considered. Treatment is with antibiotics. Common pathogens are gram-negative aerobes and anaerobes as well as streptococci. Anticoagulation is beneficial [89,90]. Despite this,
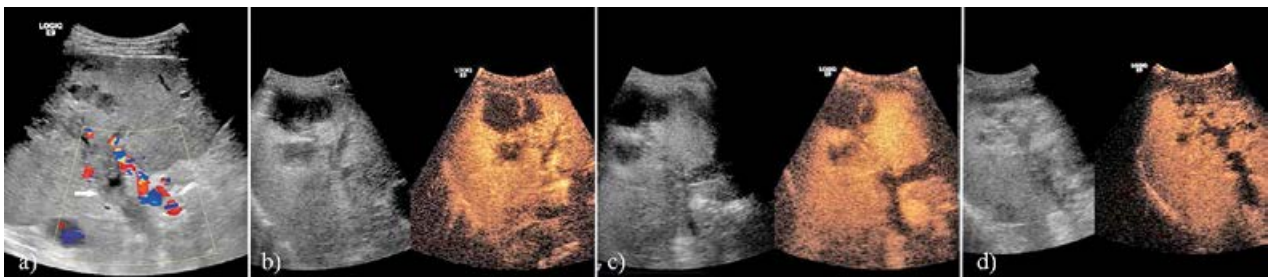

Fig 6. A 50 y/o, male, acute pancreatitis with exudates, splenic vein thrombosis, peripancreatic pseudocysts, fever and elevated inflammatory parameters. B-mode and duplex sonography show portal vein thrombosis and hypoechoic liver lesions (a). On CEUS, lesions are avascular and not enhanced in the arterial phase (b), portal venous phase (c), and late phase (d). The portal vein is not perfused. The interpretation leaves room for various differential diagnoses. The cause of the inflammation may be peripancreatic infected pseudocysts, infected lesions in the liver, or pylephlebitis. These avascular liver lesions, in turn, may be a consequence of tryptic lesions or correspond to abscesses or infarcts owing to portal vein thrombosis.

a mortality rate of 11% was reported in a study of 95 patients with pylephlebitis [75].

Budd-Chiari-Syndrome

Hepatic vein thrombosis resulting in the development of severe congestion, hepatomegaly, and ascites and is referred to as Budd-Chiari syndrome (BCS). The cause is an occlusion in the drainage area of the hepatic veins to the inferior vena cava. The development of portal vein thrombosis is also possible. In the course, fibrosis and cirrhosis of the parenchyma develop in Budd-Chiari syndrome [91-93].

Fibrotic bridges may form between hepatic veins and portal veins "venoportal fibrosis". Another variant is "venocentric cirrhosis", in which fibrotic bridging between hepatic veins and portal veins is minimal [93]. Many livers develop large regenerative nodules (LRN) that histologically resemble focal nodular hyperplasia. LRNs occur in the majority of BCS livers as well as in other diseases with severe vascular occlusion, including portalvenous obstruction, and cirrhosis. Large regenerative nodules have a single branched arterial supply. LRNs rely on adequate venous drainage, usually into the hepatic veins. Portal veins are almost always absent in LRNs [93]. Budd Chiari-syndrome (BCS) have been described and illustrated in separate papers [92].

Conclusions

With a thorough and systematic examination of all hepatic vessels on B-mode and Doppler sonography, it is possible to diagnose stenoses, thromboses and pseudoaneurysms. CEUS is helpful in the differentiation between vascular from tumor causes of thrombosis and provides assessment of the liver parenchyma. It is important, in combination with anamnestic data and the ultrasonic appearances, to include the acute clinical scenario to formulate a cohesive differential diagnoses and to search for these systematically. In the hands of an experienced clinician, CEUS can be used to detect infarcted portions of the liver parenchyma early and classify them based on the typical wedge-shaped avascular appearance. Infarct or pseudo-infarct areas can be differentiated from areas with different enhancements because of arterioportal shunts. Pylephlebitis requires a complex sonographic approach to the various abdominal organs, the mesenteric veins and the small and large intestine.

The current overview provides the examiner with the theoretical knowledge and description of sonomorphological, Doppler ultrasonographic and CEUS findings to achieve the correct diagnosis early which in turn will positively influence the outcome of the patient.

References

1. Dietrich CF, Averkiou M, Nielsen MB, et al. How to perform Contrast-Enhanced Ultrasound (CEUS). Ultrasound Int Open 2018;4:E2-E15.
2. Claudon M, Dietrich CF, Choi BI, et al. Guidelines and good clinical practice recommendations for contrast enhanced ultrasound (CEUS) in the liver--update 2012: a WFUMB-EFSUMB initiative in cooperation with representatives of AFSUMB, AIUM, ASUM, FLAUS and ICUS. Ultraschall Med 2013;34:11-29.
3. Claudon M, Dietrich CF, Choi BI, et al. Guidelines and good clinical practice recommendations for Contrast Enhanced Ultrasound (CEUS) in the liver - update 2012: A WFUMB-EFSUMB initiative in cooperation with representatives of AFSUMB, AIUM, ASUM, FLAUS and ICUS. Ultrasound Med Biol 2013;39:187-210.
4. Dietrich CF, Nolsoe CP, Barr RG, et al. Guidelines and Good Clinical Practice Recommendations for Contrast-Enhanced Ultrasound (CEUS) in the Liver-Update 2020 WFUMB in Cooperation with EFSUMB, AFSUMB, AIUM, and FLAUS. Ultrasound Med Biol 2020;46:2579-2604.
5. Dietrich CF, Nolsoe CP, Barr RG, et al. Guidelines and Good Clinical Practice Recommendations for Contrast Enhanced Ultrasound (CEUS) in the Liver - Update 2020 - WFUMB in Cooperation with EFSUMB, AFSUMB, AIUM, and FLAUS. Ultraschall Med 2020;41:562-585.
6. Möller K, Tscheu T, De Molo C, et al. Comments and illustrations of the WFUMB CEUS liver guidelines: rare congenital vascular pathology. Med Ultrason 2022, doi: 10.11152/mu-3879
7. Pitton MB, Dappa E, Jungmann F, et al. Visceral artery aneurysms: Incidence, management, and outcome analysis in a tertiary care center over one decade. Eur Radiol 2015;25:2004-2014.
8. Ma L, Lu Q, Luo Y. Vascular complications after adult living donor liver transplantation: Evaluation with ultrasonography. World J Gastroenterol 2016;22:1617-1626.
9. Ren X, Luo Y, Gao N, Niu H, Tang J. Common ultrasound and contrast-enhanced ultrasonography in the diagnosis of hepatic artery pseudoaneurysm after liver transplantation. Exp Ther Med 2016;12:1029-1033.
10. Machado NO, Al-Zadjali A, Kakaria AK, Younus S, Rahim MA, Al-Sukaiti R. Hepatic or Cystic Artery Pseudoaneurysms Following a Laparoscopic Cholecystectomy: Literature review of aetiopathogenesis, presentation, diagnosis and management. Sultan Qaboos Univ Med J 2017;17:e135-e146.
11. Deziel DJ, Millikan KW, Economou SG, Doolas A, Ko S-T, Airan MC. Complications of laparoscopic cholecystectomy: A national survey of 4,292 hospitals and an analysis of 77,604 cases. The American Journal of Surgery 1993;165:9-14.

12. Lampropoulos C, Markopoulos G, Tsochatzis S, et al. Symptomatic pseudoaneurysms following laparoscopic cholecystectomy: Focus on an unusual and dangerous complication. J Minim Access Surg 2021;17:450-457.

13. Mahmood I, Kasim M, El-Menyar A, et al. Late development of giant hepatic artery pseudoaneurysm following abdominal trauma due to tire blast: case report and literature review. J Surg Case Rep 2021;2021:rjab564.

14. Saito A, Fayad N. An Unusual Cause of Obstructive Jaundice and Acute Pancreatitis: Visceral Artery Pseudoaneurysm. Cureus 2021;13:e14307.

15. Nam HS, Noh MH, Han JE, et al. Left Hepatic Artery Pseudoaneurysm Caused by Acute Pancreatitis. Med Princ Pract 2017;26:192-194.

16. Tacconi D, Lapini L, Giorni P, Corradini S, Caremani M. Pseudoaneurysm of the hepatic artery, a rare complication of an amebic liver abscess. J Ultrasound 2009;12:49-52.

17. Kargl S, Breitwieser J, Gitter R, Pumberger W. [Hepatic artery pseudoaneurysm following blunt abdominal injury]. Unfallchirurg 2012;115:1120-1122.

18. Shiozawa K, Watanabe M, Ikehara T, et al. Right hepatic artery pseudoaneurysm complicating acute pancreatitis: a case report. Med Princ Pract 2013;22:402-404.

19. Bishehsari F, Ting PS, Green RM. Recurrent gastrointestinal bleeding and hepatic infarction after liver biopsy. World J Gastroenterol 2014;20:1878-1881.

20. Park JY, Ryu H, Bang S, Song SY, Chung JB. Hepatic artery pseudoaneurysm associated with plastic biliary stent. Yonsei Med J 2007;48:546-548.

21. Kim JH, Kim DJ, Kwon CI. Pseudoaneurysmal Rupture of the Hepatic Artery Caused by a Biliary Plastic Stent. Am J Gastroenterol 2021;116:1371.

22. Choi SH, Gwon DI, Ko GY, et al. Hepatic arterial injuries in 3110 patients following percutaneous transhepatic biliary drainage. Radiology 2011;261:969-975.

23. Sagar S, Soundarajan R, Gupta P, et al. Efficacy of endovascular embolization of arterial pseudoaneurysms in pancreatitis: A systematic review and meta-analysis. Pancreatology 2021;21:46-58.

24. Maatman TK, Heimberger MA, Lewellen KA, et al. Visceral artery pseudoaneurysm in necrotizing pancreatitis: incidence and outcomes. Can J Surg 2020;63:E272-E277.

25. Abdallah M, Vantanasiri K, Young S, et al. Visceral artery pseudoaneurysms in necrotizing pancreatitis: risk of early bleeding with lumen-apposing metal stents. Gastrointest Endosc 2022;95:1150-1157.

26. Chaer RA, Abularrage CJ, Coleman DM, et al. The Society for Vascular Surgery clinical practice guidelines on the management of visceral aneurysms. J Vasc Surg 2020;72:3S-39S.

27. Sidhu A, Shaik NB, Sharma M. Endoscopic and percutaneous ultrasound-guided thrombin and glue injection in a pseudoaneurysm of the right hepatic artery. Endoscopy 2020;52:E245-E246.

28. Mayan H, Kantor R, Rimon U, et al. Fatal liver infarction after transjugular intrahepatic portosystemic shunt procedure. Liver 2001;21:361-364.

29. Carroll R. Infarction of the human liver. J Clin Pathol 1963;16:133-136.

30. Haratake J, Horie A, Furuta A, Yamato H. Massive hepatic infarction associated with polyarteritis nodosa. Acta Pathol Jpn 1988;38:89-93.

31. Seeley TT, Blumenfeld CM, Ikeda R, Knapp W, Ruebner BH. Hepatic infarction. Hum Pathol 1972;3:265-276.

32. Saegusa M, Takano Y, Okudaira M. Human hepatic infarction: histopathological and postmortem angiological studies. Liver 1993;13:239-245.

33. Wang FH, Yang NN, Liu F, Tian H. Unexplained huge liver infarction presenting as a tumor with bleeding: A case report. World J Clin Cases 2020;8:2016-2022.

34. Lopera JE, Katabathina V, Bosworth B, et al. Segmental liver ischemia/infarction after elective transjugular intrahepatic portosystemic shunt creation: clinical outcomes in 10 patients. J Vasc Interv Radiol 2015;26:835-841.

35. Lev-Toaff AS, Friedman AC, Cohen LM, Radecki PD, Caroline DF. Hepatic infarcts: new observations by CT and sonography. AJR Am J Roentgenol 1987;149:87-90.

36. Wong MD, Lucas CE. Liver infarction after laparoscopic cholecystectomy injury to the right hepatic artery and portal vein. Am Surg 2001;67:410-411.

37. Shindo D, Yamamoto A, Amano R, et al. The findings of the contrast-enhanced CT and risk factors for hepatic infarction after pancreatoduodenectomy. Jpn J Radiol 2020;38:547-552.

38. Kamada Y, Hori T, Yamamoto H, et al. Fatal arterial hemorrhage after pancreaticoduodenectomy: How do we simultaneously accomplish complete hemostasis and hepatic arterial flow? World J Hepatol 2021;13:483-503.

39. Otani Y, Anzai S, Shibuya H, et al. Churg-Strauss syndrome (CSS) manifested as necrosis of fingers and toes and liver infarction. J Dermatol 2003;30:810-815.

40. El Allani L, Benlamkaddem S, Berdai MA, Harandou M. A case of massive hepatic infarction in severe preeclampsia as part of the HELLP syndrome. Pan Afr Med J 2020;36:78.

41. Sarmento SG, Santana EF, Campanharo FF, et al. Microcirculation Approach in HELLP Syndrome Complicated by Posterior Reversible Encephalopathy Syndrome and Massive Hepatic Infarction. Case Rep Emerg Med 2014;2014:389680.

42. Rolfes DB, Ishak KG. Liver disease in toxemia of pregnancy. Am J Gastroenterol 1986;81:1138-1144.

43. Sakhel K, Usta IM, Hannoun A, Arayssi T, Nassar AH. Liver infarction in a woman with systemic lupus erythematosus and secondary anti-phospholipid and HELLP syndrome. Scand J Rheumatol 2006;35:405-408.

44. Meloni C, Schreiber C, Studt JD, Kamm S, Di Chiara M, Herren T. Liver infarctions as the first manifestation of antiphospholipid antibody syndrome in pregnancy: a case report. J Med Case Rep 2022;16:103.

45. Peng IT, Chung MT, Lin CC. Severe Postpartum Hemorrhage Complicated with Liver Infarction Resulting in Hepatic Failure Necessitating Liver Transplantation. Case Rep Obstet Gynecol 2018;2018:2794374.

46. Liu GP, Zhang MY, Xu R, Sun CJ. Acute liver failure and infarction complicating TIPS placement. Radiol Case Rep 2019;14:876-879.

47. Patidar Y, Basavaraj, Mukund A, Sarin SK. Transarterial Chemoembolization in Unresectable Hepatocellular Carcinoma with Portal Vein Tumor Thrombosis: A Tertiary Care Center Experience. Indian J Radiol Imaging 2021;31:270-276.

48. Park BV, Gaba RC, Lokken RP. Liver Infarction after Drug-Eluting Embolic Transarterial Chemoembolization for Hepatocellular Carcinoma in the Setting of a Large Portosystemic Shunt. Semin Intervent Radiol 2016;33:337-341.

49. Whang G, Chopra S, Tchelepi H. "Bright Band Sign" A Grayscale Ultrasound Finding in Hepatic Infarction. J Ultrasound Med 2019;38:2515-2520.

50. Haque M, Schumacher PA, Harris A, et al. Late acute celiac and hepatic artery thrombosis with portal vein thrombosis resulting in hepatic infarction 12 years post orthotopic liver transplantation. Ann Hepatol 2009;8:396-399.

51. Quiroga S, Sebastia MC, Margarit C, Castells L, Boye R, Alvarez-Castells A. Complications of orthotopic liver transplantation: spectrum of findings with helical CT. Radiographics 2001;21:1085-1102.

52. Crossin JD, Muradali D, Wilson SR. US of liver transplants: normal and abnormal. Radiographics 2003;23:1093-1114.

53. Liu DY, Yi ZJ, Tang Y, Niu NN, Li JX. Three Case Reports of Splenic Artery Steal Syndrome After Liver Transplantation. Transplantation Proceedings 2015;47:2939-2943.

54. Torabi M, Hosseinzadeh K, Federle MP. CT of nonneoplastic hepatic vascular and perfusion disorders. Radiographics 2008;28:1967-1982.

55. Ashida A, Matsukawa H, Samejima J, et al. Liver infarction due to liver abscess. Dig Surg 2008;25:258-259.

56. Priyadarshi RN, Kumar P, Kumar R, Anand U, Shyama. Venous thrombosis and segmental hypoperfusion in amebic liver abscess: MDCT demonstration and its implications. Abdom Radiol (NY) 2020;45:652-660.

57. Mihaila RG, Sandu M. Liver infarction in a patient with Clostridium Difficile colitis. A possible connection? J Gastrointestin Liver Dis 2019;28:245-246.

58. Kulkarni A, Arif H, Veer M, Ziggas K, Kaura A, Sareen M. Hepatic infarction and acute pancreatitis: a case report and review of the literature. Clin Exp Hepatol 2018;4:43-45.

59. Ebert EC, Nagar M, Hagspiel KD. Gastrointestinal and hepatic complications of sickle cell disease. Clin Gastroenterol Hepatol 2010;8:483-489; quiz e470.

60. Wang MY, Potosky DR, Khurana S. Liver infarction after transjugular intrahepatic portosystemic shunt in a liver transplant recipient. Hepatology 2011;54:1887-1888.

61. Khong SY, James M, Smith P. Diagnosis of liver infarction postpartum. Obstet Gynecol 2005;105:1271-1273.

62. Anand A, Gamanagatti SR, Vaishnav M, Shalimar. Hepatic Infarction after Transjugular Intrahepatic Shunt Creation. J Vasc Interv Radiol 2021;32:1513.

63. Chincarini M, Zamboni GA, Pozzi Mucelli R. Major pancreatic resections: normal postoperative findings and complications. Insights Imaging 2018;9:173-187.

64. von Herbay A, Schick D, Horger M, Gregor M. [Low-MI-sonography with the contrast-agent SonoVue in the diagnosis of infarction of the spleen, kidney, liver and pancreas]. Ultraschall Med 2006;27:445-450.

65. Llewellyn ME, Jeffrey RB, DiMaio MA, Olcott EW. The sonographic "bright band sign" of splenic infarction. J Ultrasound Med 2014;33:929-938.

66. Fontanilla T, Noblejas A, Cortes C, et al. Contrast-enhanced ultrasound of liver lesions related to arterial thrombosis in adult liver transplantation. J Clin Ultrasound 2013;41:493-500.

67. Claudon M, Dietrich CF, Choi BI, et al. Guidelines and good clinical practice recommendations for Contrast Enhanced Ultrasound (CEUS) in the liver - update 2012: A WFUMB-EFSUMB initiative in cooperation with representatives of AFSUMB, AIUM, ASUM, FLAUS and ICUS. Ultrasound Med Biol 2013;39:187-210.

68. Merz E, Evans DH, Dong Y, Jenssen C, Dietrich CF. History of ultrasound in obstetrics and gynaecology from 1971 to 2021 on occasion of the 50 years anniversary of EFSUMB. Med Ultrason 2022.

69. Dietrich CF, Bolondi L, Duck F, et al. History of Ultrasound in Medicine from its birth to date (2022), on occasion of the 50 Years Anniversary of EFSUMB. A publication of the European Federation of Societies for Ultrasound In Medicine and Biology (EFSUMB), designed to record the historical development of medical ultrasound. Med Ultrason 2022, doi: 10.11152/mu-3757

70. Giovine S, Pinto A, Crispano S, Lassandro F, Romano L. Retrospective study of 23 cases of hepatic infarction: CT findings and pathological correlations. Radiol Med 2006;111:11-21.

71. Passmore R. LIVER: STRUCTURE AND FUNCTION. By H. Popper and F. Schaffner. New York, Toronto and London: McGraw-Hill Book Company Inc. 1957. Pp. xv + 777. £7, 10s. Quarterly Journal of Experimental Physiology and Cognate Medical Sciences 1957;42:418-419.

72. Horrocks P, Tapp E. Zahn's 'infarcts' of the liver. J Clin Pathol 1966;19:475-478.

73. Symmers W. Portal embolism following thrombosis of splenic vein, and causing infarct-like cyanotic atrophy ('Zahn's infarcts') of the liver; a complication of splenectomy performed in the course of total gastrectomy. Br J Surg 1951;38:498-502.

74. Stout LC, Lyon RE, Murray NG, Barth MH. Pseudointimal biliary epithelial proliferation and Zahn's infarct associated with a 6 1/2-month-old transjugular intrahepatic portosystemic shunt. Am J Gastroenterol 1995;90:126-130.

75. Choudhry AJ, Baghdadi YM, Amr MA, Alzghari MJ, Jenkins DH, Zielinski MD. Pylephlebitis: a Review of 95 Cases. J Gastrointest Surg 2016;20:656-661.

76. Hartpence J, Woolf A: Pylephlebitis. In: StatPearls. Treasure Island (FL), 2022.

77. Kanellopoulou T, Alexopoulou A, Theodossiades G, Koskinas J, Archimandritis AJ. Pylephlebitis: an overview of non-cirrhotic cases and factors related to outcome. Scand J Infect Dis 2010;42:804-811.

78. Liebman PR, Patten MT, Manny J, Benfield JR, Hechtman HB. Hepatic--portal venous gas in adults: etiology, pathophysiology and clinical significance. Ann Surg 1978;187:281-287.

79. Kinoshita H, Shinozaki M, Tanimura H, Umemoto Y, Sakaguchi S, Takifuji K, Kawasaki S, et al. Clinical features and management of hepatic portal venous gas: four case reports and cumulative review of the literature. Arch Surg 2001;136:1410-1414.

80. Seak CJ, Hsu KH, Wong YC, et al. The prognostic factors of adult patients with hepatic portal venous gas in the ED. Am J Emerg Med 2014;32:972-975.

81. Nelson AL, Millington TM, Sahani D, et al. Hepatic portal venous gas: the ABCs of management. Arch Surg 2009;144:575-581; discussion 581.

82. Capolupo GT, Masciana G, Carannante F, Caricato M. Hepatic portal venous gas after colonoscopy: A case report and review. Int J Surg Case Rep 2018;51:54-57.

83. Ginesu GC, Barmina M, Cossu ML, et al. Conservative approach to Hepatic Portal Venous Gas: A case report. Int J Surg Case Rep 2017;30:183-185.

84. Jiang M, Li CL, Pan CQ, et al. Nomogram for predicting transmural bowel infarction in patients with acute superior mesenteric venous thrombosis. World J Gastroenterol 2020;26:3800-3813.

85. Hamada T, Yamauchi M, Tanaka M, Hashimoto Y, Nakai K, Suenaga K. Prospective evaluation of contrast-enhanced ultrasonography with advanced dynamic flow for the diagnosis of intestinal ischaemia. Br J Radiol 2007;80:603-608.

86. Kanzaki T, Hata J, Imamura H, et al. Contrast-enhanced ultrasonography with Sonazoid for the evaluation of bowel ischemia. J Med Ultrason (2001) 2012;39:161-167.

87. Koenig G, Tantawi M, Wessner CE, Eisenbrey JR. Evaluation of Suspected Small Bowel Ischemia Using Contrast-Enhanced Ultrasound with Computed Tomography Fusion. J Emerg Trauma Shock 2022;15:60-62.

88. Hata J, Kamada T, Haruma K, Kusunoki H. Evaluation of bowel ischemia with contrast-enhanced US: initial experience. Radiology 2005;236:712-715.

89. Naymagon L, Tremblay D, Schiano T, Mascarenhas J. The role of anticoagulation in pylephlebitis: a retrospective examination of characteristics and outcomes. J Thromb Thrombolysis 2020;49:325-331.

90. Anand S, Umeh CA, Giberson C, et al. Septic Portal Vein Thrombosis, Clinical Presentation, and Management. Cureus 2021;13:e19840.

91. Zu M, Xu H, Zhang Q, et al. Review of Budd-Chiari Syndrome. J Interv Med 2020;3:65-76.

92. Wiest I, Teufel A, Ebert MP, et al. [Budd-Chiari syndrome, review and illustration]. Z Gastroenterol 2021.

93. Tanaka M, Wanless IR. Pathology of the liver in Budd-Chiari syndrome: portal vein thrombosis and the histogenesis of veno-centric cirrhosis, veno-portal cirrhosis, and large regenerative nodules. Hepatology 1998;27:488-496.