Review

Med Ultrason 2024, Vol. 26, no. 2, 178-186
DOI: 10.11152/mu-4287

Comments and illustrations of the WFUMB CEUS liver guidelines: Peliosis hepatis and porphyria

Kathleen Möller[1], Oliver Hoffmann[2], Ehsan Safai Zadeh[3], Christian Görg[4], Christian Jenssen[5], Michael Hocke[6], Chiara de Molo[7], Carla Serra[7], Yi Dong[8], Xin Wu Cui[9], Adrian Lim[10], Klaus Dirks[11], Siegbert Faiss[1], David Srivastava[2,12], Christoph F. Dietrich[2]

[1]Sana Hospital Lichtenberg, Medical Department I/Gastroenterology, Berlin, Germany, [2]Department Allgemeine Innere Medizin, Kliniken Hirslanden, Beau Site, Salem und Permanence, Bern, Switzerland, [3]Department of Biomedical Imaging and Image-Guided Therapy, Medical University of Vienna, Vienna, Austria, [4]Center of Ultrasound Diagnostics, University Hospital Giessen and Marburg, Philipps University Marburg, Baldingerstraße, 35033 Marburg, Germany, [5]Krankenhaus Märkisch-Oderland, Department of Internal Medicine, Strausberg, Germany; Brandenburg Institute for Clinical Ultrasound (BICUS) at Medical University Brandenburg, Neuruppin, Germany, [6]Medical Department, Helios Klinikum Meiningen, Germany, [7]Interventional, Diagnostic and Therapeutic Ultrasound Unit, Department of Medical and Surgical Sciences, IRCCS, Azienda Ospedaliero-Universitaria Sant'Orsola Malpighi Hospital, Bologna, Italy, [8]Department of Ultrasound, Xinhua Hospital Affiliated to Shanghai Jiaotong University School of Medicine, Shanghai, China, [9]Department of Medical Ultrasound, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China, [10]Imaging Department, Imperial College Healthcare NHS Trust, Charing Cross Hospital, London, UK, [11]Rems-Murr-Klinikum, Winnenden, Germany, [12]Inselspital, University Hospital of Bern, Switzerland

Abstract

In this series of articles on comments and illustrations of the World Federation for Medicine and Biology (WFUMB) guidelines on contrast-enhanced ultrasound (CEUS), the topics on very rare focal liver lesions (FLL) are discussed. This article describes the diverse changes of focal liver lesions in peliosis hepatis and the typical changes in porphyria. Although the focus is on the appearance on ultrasound and CEUS, the clinical context is always considered. While peliosis may be a surprising finding on puncture, lesions in porphyria cutanea tarda may be typical visual diagnoses that obviate the need for biopsy. If only you knew. This article aims to sharpen the clinician's eye. It provides knowledge of the clinical presentation and US and CEUS imaging of peliosis hepatis and porphyria.

Keywords: peliosis hepatis; porphyria; imaging; ultrasonography; CEUS

Introduction

The appearance of the most common benign and malignant liver tumors on ultrasound (US) and contrast-enhanced ultrasound (CEUS) is well known and reported in detail in the guidelines of the World Federation for Ultrasound in Medicine and Biology (WFUMB) [1-5].

It is usually not difficult to diagnose typical liver lesions such as typical haemangiomas or metastases on CEUS. However, problems may arise when rare lesions do not enter the differential diagnosis in the mind of the examiner when characterizing a lesion, owing to their poorly known or described imaging features on US and CEUS. For a large number of rare focal liver lesions (FLL), data already exist. Examples are nodular regenerative hyperplasia [6], inflammatory pseudotumour [7], peliosis [8-10], sarcoidosis [11-14], cholangiocellular adenoma [15], cystadenoma and cystadenocarcinoma

Received 09.06.2023 Accepted 17.07.2023
Med Ultrason
2024, Vol. 26, No 2, 178-186
Corresponding author: Prof. Dr. med. Christoph F. Dietrich
Department Allgemeine Innere Medizin (DAIM)
Kliniken Beau Site, Salem und Permanence,
Hirslanden, Bern, CH-3036 Bern, Switzerland
Phone: +41 764408150
E-mail: c.f.dietrich@googlemail.com

[16], hemangioendothelioma [17,18], hepatocellular carcinoma (HCC) in the non-cirrhotic liver [19], fibrolamellar hepatocellular carcinoma [20,21], mixed HCC and cholangiocellular carcinoma [22], very small HCC (<10 mm) [23], sarcoma [24], tuberculosis [25,26], hydatid cysts [27-30], alveolar echinococcosis [28], schistosomiasis [31,32], ascariasis [33, 34], fasciolosis [35], clonorchis and opisthorchis [36], toxocariasis [37], bacillary angiomatosis [38], amyloidosis with spontaneous hemorrhage [39], as well as rare FLLs in pediatric patients [40,41].

In addition, there are data on CEUS for various entities and clinical situations [42-47].

This current article continues this and describes the liver lesions of peliosis and porphyria. An overview of disease patterns is also provided but the main focus is on the appearance on US and CEUS.

Peliosis

Peliosis hepatis (PH) is a rare benign disease histologically characterized by a proliferation of the sinusoidal hepatic capillaries with blood-filled cystic cavities of various sizes and irregular shapes [48]. It was first described by E. Wagner in 1861 [49]. In 1916, W. Schoenlack defined the term "peliosis" from the Greek word *pelios*, which means "reddish" or "bluish") [50]. The pathogenesis and etiology of PH remains unclear, and it remains unidentified in 20-50% of all patients [51,52]. The cause may be secondary to an altered venous outflow tract with the consequence of damage to the walls of the sinusoids, dilatation of the sinusoids and dilation of the central vein of the hepatic lobule [53]. Peliosis hepatis is often associated with chronic wasting diseases. The diagnosis has been reported in patients suffering from tuberculosis, immunosuppressed patients [54-57], HIV [58-60], haemato-oncological diseases [61-65], but also in the context of pregnancy [8], oral contraceptives [8,66], anabolic steroids [64], azathioprine and chemotherapeutic agents.

Peliosis has also been reported in association with Bartonella species infection [56,60]. Bartonella henselae and Bartonella quintana cause the so-called bacillary peliosis in patients with AIDS.

Most commonly, Peliosis occurs in the liver but can also affect the spleen, bone marrow, lungs, abdominal lymph nodes, and other organs [59].

In most cases, the lesions are asymptomatic incidental findings on conventional imaging [67,68]. However, intrahepatic hemorrhage, liver rupture, and hemoperitoneum or acute liver failure have also been described [51,63,65,69-71].

In clinical practice, accurate preoperative diagnosis of PH may impact the immediate management of patients and prevent unnecessary surgery or biopsy [51].

Imaging

Imaging findings of focal PH may vary depending on the size, pathological presentation, and stage of the lesion [51,72]. It is typically difficult to make a clear distinction of PH from other hypervascular lesions through imaging studies alone [52]. Most cases are detected incidentally as a hypervascular tumor on cross-sectional imaging (CT, MRI, or US).

It has non-specific features on grey-scale abdominal US and can present as a homogeneous, hypoechoic lesion in patients with fatty livers, a hyperechoic lesion in those with healthy livers or a heterogeneous lesion if complicated by hemorrhage [51,73]. It is worth bearing in mind that any hepatic lesions with a hypoechoic rim must be suspicious for metastases [9,74].

With color Doppler US, it is possible to detect intralesional flow which typically has low-resistive indices on spectral Doppler US [75].

A series on the appearances of peliosis on US and CEUS in 24 patients has been reported by Dong et al [68]. These lesions were solitary in 70.8% and multiple in 29.2% [68]. On B-mode US, lesions were usually heterogeneously hypoechoic with well-defined margins but irregular shapes. There was no displacement of surrounding structures [68]. Color Doppler flow signals were detected in 41.7% [68]. On CEUS, in the arterial phase, mild heterogeneous enhancement was seen in 83.3%, centrifugal hyperenhancement in 12.5%, and isoenhancement in 16.7%. After 1 minute in the portal venous phase, 87.5% of the lesions were slightly hypoenhanced and washed out in the late phase [68]. While late-phase hyperenhancement is a typical feature of benign lesions in noncirrhotic livers [76,77], late-phase hypoenhancement of benign lesions are rarely found [4,5]. These exceptions include inflammatory infiltrations and pseudotumors.

Hypervascularized liver metastases and cholangiocellular carcinomas can be distinguished from PH based on their early and distinct washout. However, liver metastases of neuroendocrine tumors are an exception. A weak and late washout is indicative of HCC, but ancillary features of liver cirrhosis will usually be apparent.

There were three patients (12.5%) with phlebectatic variant of hepatic peliosis who had a highly enhanced area in the central part during the arterial phase, which then progressively spread centrifugally to the periphery during the portal venous and late phases [68]. The phlebectatic type of hepatic peliosis is due to aneurysmal dilatation of the central vein [78,79]. In contrast to

FNH, centrifugal enhancement in the portal venous and late phases was described here [68]. A similar contrast behavior with arterial centrifugal fill in and late phase enhancement has been reported by Loizides et al [9]

Gronlykke et al described multiple lesions with peripheral ring enhancement in the early arterial phase and centripetal filling, then centripetal enhancement and hyperenhancement in the late phase [8]. Contrast enhancement characteristics similar to metastases with peripheral hyperenhancement with central but not peripheral washout in the late phase has also been described in another case report [74].

The various observed appearances of peliosis lesions in the CEUS are listed in Table I.

Focal PH should be suspected when an incidentally detected focal liver lesion reveals no specific US features that clearly favor a diagnosis of common tumor-like hepatic lesions [81]. The lack of a mass effect would further support this diagnosis. The CEUS enhancement patterns of focal PH may also vary with any underlying diseases and the various stages of the blood components [72].

The characteristics of the disease in abdominal US, CT, MRI, angiography, and FDG-PET/CT have been previously described [72,82].

On unenhanced CT, peliotic lesions usually appear as multiple areas of low attenuation. However, they may show hyperattenuation to the liver parenchyma, presumably related to hemorrhage [51]. On contrast-enhanced CT, peliotic lesions may be hypoattenuating to the liver parenchyma. However, they tend to become increasingly isoattenuating over time. In addition, some lesions may also have areas of increased attenuation [51]. Variants with centripetal and centrifugal enhancement have been described [51,78,83].

The signal intensity of lesions on MRI largely depends on the duration and status of the blood component. On T2 weighted imaged, peliotic lesions are usually hyperintense and hyperintense on T1w which may indicate blood. Hepatic peliosis causes persistent enhancement on delayed phase images [51,64,65,72,83,84]. However, isointense and hypointense lesions have also been reported [51]. On T1-weighted images post contrast injection, peliotic lesions usually show centrifugal enhancement patterns and rarely centripetal [51,83].

Ultimately, the definitive diagnosis of hepatic peliosis can only be confirmed histopathologically. Dilated sinusoidal spaces and hemorrhagic dilated spaces within the liver parenchyma are typical but this requires adequate tissue sampling which can be difficult owing to the dilated sinusoidal spaces involved. In addition, biopsy in peliosis is associated with a high risk of bleeding [85]. The aspirates can also be examined for Bartonella species especially in cases without predisposing factors and corresponding serology for Bartonella antibodies in the blood can also be carried out.

Dong et al concluded that hepatic lesions without a mass effect demonstrating mild heterogeneous arterial hyperenhancement with washout in the very late portal

Table I. Peliosis hepatis appearance in the CEUS.

Autor	n	Arterial phase	Portalvenous phase	Late Phase
Schuldes 2011 [74]	n = 1 Multiple lesions	Peripheral hyperenhancement	Isoenhancement	Central but not peripheral hypoenhancement / central washout
Xu 2011 [80]	n =2	Homogeneous hyperenhancement	Isoenhancement	Hypoenhancement
		Homogeneous isoenhancement	Subsequent washout in the portal venous or late phase (not specified)	Subsequent washout in the portalvenous or late phase (not specified)
Gronlykke 2013 [8]	n = 1 Multiple lesions	Peripheral ring enhancement in the early arterial phase with centripetal nonglobular fill in		Homogeneous enhancement for larger lesions
		Homogeneous enhancement for small lesions		Slight central washout in 1 lesion after 6 min
Loizides 2017 [9]	n = 1	central enhancement in the early arterial phase with a centrifugal filling		Homogeneous enhancement
Dong 2021 [68]	n = 24	Heterogeneous hyperenhancement 83.1 % Isoenhancement 16.7 %	Hypoenhancement 87.5 %	Hypoenhancement 87.5 %
		Centrifugal hyperenhancement 12.5 %	Hyperenhancement 12.5 %	Hyperenhancement 12.5 %

n, number of cases

venous phase (after 1 minute) on CEUS are characteristics of hepatic peliosis [68]. However, these characteristics alone can be difficult to distinguish from other well-vascularized lesions and tumors. Hepatic peliosis should be considered in the differential diagnosis when the clinical context is not suggestive of malignancy or infection. The decision to biopsy should be made very responsibly because of the high risk of bleeding. In the case series by Dong et al, needle biopsy was performed with 18-G or 20-G needles in 41.7% of hepatic peliosis cases, with no mention of complications. All other patients underwent surgery, highlighting the difficulty in excluding suspected malignancy [68]. In individual cases, the lack of histologic assessment by needle aspiration may lead to a hemihepatectomy [86].

In asymptomatic patients with incidentally discovered liver lesions that show hyperenhancement in the late phase, a benign lesion can be assumed, and biopsy may not be necessary. The situation is different in symptomatic patients or patients with a history of malignancy in whom the focal liver lesion shows hypoenhancement in the portal venous and late phases. In these circumstances, the lesion should be assumed malignant until proven otherwise.

Treatment

Therapeutic strategies include treatment of the underlying disease, if possible. In the case of small asymptomatic findings, it is possible to wait. In the case of larger, complicated findings with hemorrhage, symptomatic or unclear findings that do not allow malignancy to be ruled out, surgical resection would be indicated.

Imaging assessment it thus highly important but usually requires a high degree of experience to make a confident diagnosis of peliosis conservatively (fig 1).

Porphyria

Porphyrias predominantly include hereditary metabolic disorders of heme biosynthesis, which are diagnosed and differentiated through specific biochemical patterns of porphyrins and porphyrin precursors found in urine, feces, and blood. Each type of porphyria is the result of a specific enzyme alteration in the pathway. The liver appears to be a central organ in the pathophysiology of porphyrias. Acute and non-acute, as well as erythropoietic and hepatic porphyrias can be subdivided according to etiology and clinical presentation [87-90].

Acute hepatic porphyrias (AHP) include: acute intermittent porphyria (AIP), porphyria variegata (VP), hereditary coproporphyria (HCP), and and 5-aminolevulinic acid dehydrase defect porphyria (ALAD-DP; Doss porphyria). These forms are characterized by an overproduction of presumably neurotoxic porphyrin precursors, the onset of which leads to intermittent, col-

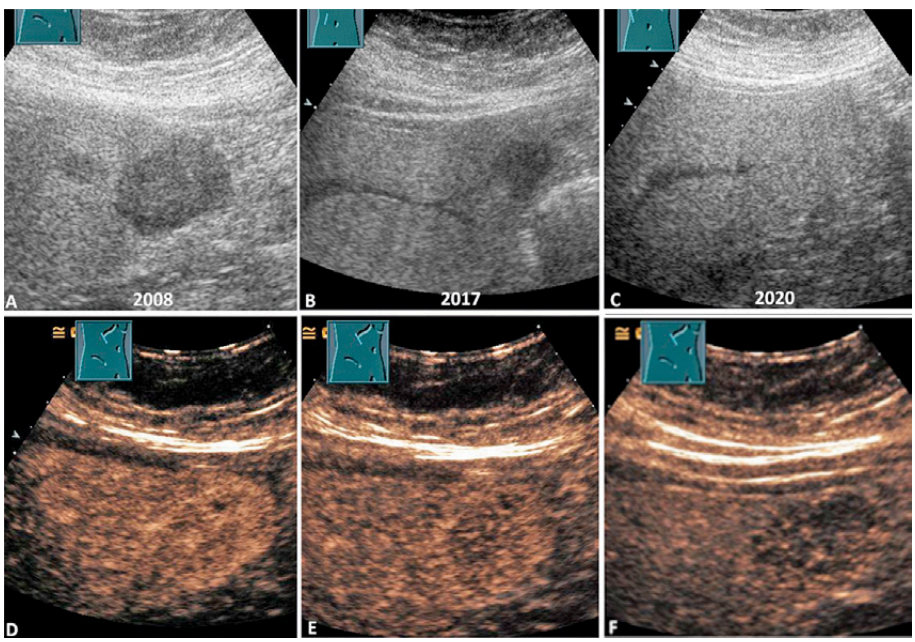

Fig 1. Peliosis. A 47-year-old female patient with incidental hypoechoic hepatic lesions. Visualization of the lesions on B-mode ultrasound in 2008 (A), 2017 (B), and 2020 (C). On contrast-enhanced ultrasound, the lesion showed mild arterial hyperenhancement after 30 s (D), with increasing hypoenhancement after 2 min (E) and 3 min (F). An ultrasound-guided biopsy was performed, and the diagnosis of peliosis was confirmed histologically.

icky abdominal pain as well as pain in the back and extremities, paresthesia and paralysis, vigilance disorders, seizures, hallucinations, and cardiovascular symptoms. Hyponatremia may occur secondary to inadequate ADH secretion (i.e., Schwartz-Bartter syndrome). Additional photosensitivity occurs in VP and HCP. Liver damage does not usually accompany AHP. Crucial to the diagnosis of AHP is the more than 4-fold increase in urinary excretion of 5-aminolevulinic acid (ALA) in ALAD-DP and of ALA and porphobilinogen (PBG) in all other acute porphyrias [87-89,91,92].

Non-acute hepatic porphyrias are porphyria cutanea tarda (PCT) and hepatoerythropoietic porphyria (HEP) [87-89].

Non-acute erythropoietic porphyrias include erythropoietic protoporphyria (EPP), X-linked protoporphyria (XLP), and congenital erythropoietic porphyria (CEP, M. Günther [93]).

In non-acute porphyrias, the accumulation of porphyrins in the liver and skin leads to photosensitivity and occasionally severe liver damage. The most common clinical forms of porphyrias are PCT, AIP, and EPP [87-89].

The cause of PCT is a sporadic or hereditary reduction in the activity of the uroporphyrinogen decarboxylase (Uro-D) in the liver. In this regard, PCT can be subdivided into several types as follows [87-89,94-96]:

- ***Type I:*** Sporadic or acquired form. The enzyme activity of hepatic Uro-D decreases during the active disease state. However, type-I PCT is only induced by external factors such as iron, drugs, estrogen, nicotine, chemicals, hepatitis C, HIV, and lupus erythematosus. Alcohol is one of the most important causative agents.
- ***Type II:*** Hereditary form of PCT with a family history; an autosomal dominant disorder with mutations on the Uro-D gene. However, most of the gene carriers do not display symptoms.
- ***Type III:*** Toxic form, characterized by its sporadic nature with a family history.
- ***Type IV:*** Hepatoerythropoietic porphyria (HEP), caused by a homozygotic defect on Uro-D.

In PCT, skin changes such as photosensitivity, bullae formation, and skin hyperpigmentation are observed, along with liver damage. Hepatic involvement is common. Most patients have abnormal liver function tests or exhibit only mild elevation of aminotransferase levels. Fatty liver degeneration and iron overload are noted in almost all cases. Liver cirrhosis and HCC may be induced by PCT, depending on the degree of liver involvement. However, decompensated cirrhosis is very rare [94]. Increases in porphyrin precursors and porphyrins may be secondary in patients without porphyria but with liver diseases, iron deficiency, and lead intoxication [87].

Porphyrias and increased risk of hepatocellular carcinoma

Patients with AIP are at increased risk for developing HCC without pre-existing chronic liver disease, especially liver cirrhosis. In addition, a rarer association exists between HCP, VP, and HCC that concerns symptomatic patients as well as latent carriers [90,97-101]. Kauppinen et al reported HCC as the cause of death in 9% of patients with AHP [97]. Innala et al conducted a screening over 15 years on AIP carriers in Sweden. They discovered that carriers over the age of 55 have a 100-fold increased risk of developing HCC. The authors recommend annual US screening for HCC in AIP gene carriers from the age of 50 and as long as therapeutic options are applicable with regard to concomitant diseases and age [101]. Deybach et al extend this screening recommendation to all AHP gene carriers, including those of AIP, VP, and HCP [90]. PCT and HCC are equally implicated. However, in this context, both HCC and PCT appeared because of an underlying liver disease such as chronic alcohol abuse, hepatitis C and B, or hemochromatosis, each contributing to fibrosis and cirrhosis. Currently, patients with PCT can benefit from etiopathogenetic treatments that reduce liver damage and halt the progression to liver cirrhosis.

Imaging

In AHP, the task of US is the differential diagnosis of acute abdominal pain and the screening for HCC. Meanwhile, with PCT, B-mode US may reveal hypoechoic round liver lesions, which exhibit the distinctive feature of a hyperechoic rim. The center of the lesions may be hyperechoic or isoechoic to varying degrees, and the hepatic vessels are not affected by the lesions. The lesions have no angioneogenesis and are isoenhanced on CEUS. If porphyria is already known, these lesions are indicative of liver involvement. If no porphyria is known to date, it makes sense to carry out a corresponding serological and urine diagnosis. Differential diagnosis are fat distribution disorders. In the case of PCT, it is important to assess the degree of liver damage during patient management, especially in cases of elevated liver values. B-mode US, elastography for fibrosis assessment, Doppler US for vascular assessment, and CEUS for liver lesions are available for this purpose. In the case of liver cirrhosis, screening with regard to HCC should be conducted (fig 2).

Conclusion

Liver lesions in porphyria have characteristic US and CEUS features., and should not be misclassified as liver

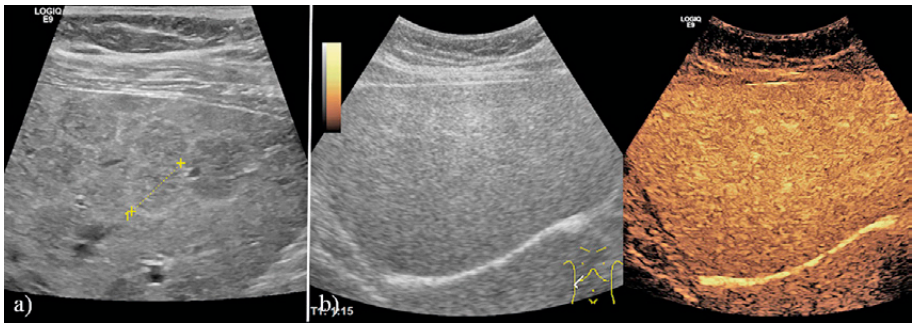

Fig 2. Porphyria (no biopsy, unchanged in control one year later), a 68-year-old female patient. Multiple nodules in the liver were found incidentally. The patient had no complaints, normal liver enzymes, no signs of cirrhosis on sonography and 2D-shear wave elastography. CT and MRI found no signs of malignancy. A control one year later did not show any change. Multiple hypoechoic foci up to 2 cm with a characteristic hyperechoic rim in both liver lobes with no distortion of the hepatic veins (a). On CEUS, homogeneous contrast enhancement in the arterial as well as in the portal-venous phase and no washout in the late phase (b). The foci are possibly caused by deposits of intermediate products of heme synthesis.

malignancy/metastases, which will cause undue worry to patients. In contrast, the imaging features of hepatic peliosis on US and CEUS are not only varied and confusing, but may also be suggestive of a malignant tumor.

Peliosis is a benign lesion. However, it can occur in various consumptive situations, in immunocompromised patients and tumor therapy. In this context, any new liver lesion is suspicious for tumor or metastases. The sonographic appearance is very diverse. Peliosis is non-vascular infiltrating and non-displacing, often hemorrhagic. Typical features in CEUS are a heterogeneous hyperenhancement in the arterial phase with a weak washout to the end of the portal venous phase, making differentiation from malignancy impossible.

In this case, US-guided biopsy may be indicated. Contrary to reports of increased bleeding risk, no complications occurred in the current work by Dong et al [68]. Another typical appearance is the phlebectatic variant with centrifugal enhancement in the arterial and portal venous phase. Although this has a pathognomic enhancement pattern, the occurence of this lesion is less common [68]. In the case of a planned biopsy, appropriate follow-up should be performed if the risk of bleeding is still considered to be increased. The pathologist should also be informed of the possible diagnosis of peliosis. Biopsies show sinusoidal dilatation without typical tissue cylinders.

Patients with PCT can show typical liver lesions characterized by a hyperechoic rim. The lesion itself can be isoechoic to the surrounding area, even slightly hyperechoic without vessel distortion. The lesions show no neoangiogenesis and are isoenhanced on CEUS. In this context, a liver biopsy is not necessary. If no porphyria is known to date, appropriate serological and urine diagnostics would be recommended. Patients with PCT, who often have underlying liver disease, need to be screened for the development of HCC. Patients with AIP have an increased risk of HCC even without liver disease. Therefore, these patients must also be monitored.

For the differential diagnostic assessment of liver lesions, it is important to consider and include the entire clinical context. The clinical data are important for deriving a suspected diagnosis from the US image. US images can be typical, such as the liver lesions in PCT or the phlebectatic variant of peliosis in CEUS. If there is even the slightest suspicion of peliosis, post-biopsy surveillance should be particularly intensified. Owing to the characteristic imaging features and combined with supporting clinical findings, biopsy should not be required for the diagnosis of porphyria lesions.

Knowledge of these ultrasound images should help to instigate appropriate further management.

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. Faust D, Fellbaum C, Zeuzem S, Dietrich CF. Nodular regenerative hyperplasia of the liver: a rare differential diagnosis of cholestasis with response to ursodeoxycholic acid. Z.Gastroenterol. 2003;41:255-258.
7. Schuessler G, Fellbaum C, Fauth F, et al. [The infammatory pseudotumour -- an unusual liver tumour]. Ultraschall Med. 2006;27:273-279.
8. Gronlykke L, Tarp B, Dutoit SH, Wilkens R. Peliosis hepatis: a complicating finding in a case of biliary colic. BMJ Case Rep 2013;2013.
9. Loizides A, Glodny B, Zoller H, et al. Contrast enhanced ultrasound of a rare case of Peliosis hepatis. Med Ultrason 2017;19:114-116.
10. Dong Y, Wang WP, Lim A, et al. Ultrasound findings in peliosis hepatis. Ultrasonography 2021;40:546-554.
11. Tana C, Schiavone C, Ticinesi A, , et al. Ultrasound imaging of abdominal sarcoidosis: State of the art. World J Clin Cases 2019;7:809-818.
12. Tana C, Silingardi M, Dietrich CF. New trends in ultrasound of hepatosplenic sarcoidosis. Z Gastroenterol 2015;53:283-284.
13. Hirche TO, Hirche H, Cui XW, Wagner TO, Dietrich CF. Ultrasound evaluation of mediastinal lymphadenopathy in patients with sarcoidosis. Med Ultrason 2014;16:194-200.
14. Tana C, Dietrich CF, Schiavone C. Hepatosplenic sarcoidosis: contrast-enhanced ultrasound findings and implications for clinical practice. Biomed Res Int 2014;2014:926203.
15. Ignee A, Piscaglia F, Ott M, Salvatore V, Dietrich CF. A benign tumour of the liver mimicking malignant liver disease--cholangiocellular adenoma. Scand.J.Gastroenterol. 2009;44:633-636.
16. Dong Y, Wang WP, Mao F, et al. Contrast enhanced ultrasound features of hepatic cystadenoma and hepatic cystadenocarcinoma. Scand J Gastroenterol 2017;52:365-372.
17. Klinger C, Stuckmann G, Dietrich CF, et al. Contrast-enhanced imaging in hepatic epithelioid hemangioendothelioma: retrospective study of 10 patients. Z Gastroenterol 2019;57:753-766.
18. Dong Y, Wang WP, Cantisani V, et al. Contrast-enhanced ultrasound of histologically proven hepatic epithelioid hemangioendothelioma. World J Gastroenterol 2016;22:4741-4749.
19. Dong Y, Wang WP, Lee WJ, et al. Contrast-Enhanced Ultrasound Features of Histopathologically Proven Hepatocellular Carcinoma in the Non-cirrhotic Liver: A Multicenter Study. Ultrasound Med Biol 2022;48:1797-1805.
20. Dong Y, Wang WP, Mao F, et al. Imaging Features of Fibrolamellar Hepatocellular Carcinoma with Contrast-Enhanced Ultrasound. Ultraschall Med 2020.
21. Fu T, Ding H, Xu C, Zhu Y, Xue L, Lin F. Imaging findings of fibrolamellar hepatocellular carcinomas on ultrasonography: A comparison with conventional hepatocellular carcinomas. Clin Hemorheol Microcirc 2021;77:49-60.
22. Dong Y, Teufel A, Trojan J, Berzigotti A, Cui XW, Dietrich CF. Contrast enhanced ultrasound in mixed hepatocellular cholangiocarcinoma: Case series and review of the literature. Dig Liver Dis 2018;50:401-407.
23. Dong Y, Teufel A, Wang WP, Dietrich CF. Current Opinion about Hepatocellular Carcinoma <10 mm. Digestion 2021;102:335-341.
24. Trojan J, Hammerstingl R, Engels K, Schneider AR, Zeuzem S, Dietrich CF. Contrast-enhanced ultrasound in the diagnosis of malignant mesenchymal liver tumors. J.Clin.Ultrasound 2010;38:227-231.
25. Dong Y, Jurgensen C, Puri R, et al. Ultrasound imaging features of isolated pancreatic tuberculosis. Endosc Ultrasound 2018;7:119-127.
26. Barreiros AP, Braden B, Schieferstein-Knauer C, Ignee A, Dietrich CF. Characteristics of intestinal tuberculosis in ultrasonographic techniques. Scand.J.Gastroenterol. 2008;43:1224-1231.
27. Dietrich CF, Douira-Khomsi W, Gharbi H, et al. Cystic echinococcosis, review and illustration of non-hepatic manifestations. Med Ultrason 2020;22:319-324.
28. Dietrich CF, Douira-Khomsi W, Gharbi H, et al. Cystic and alveolar echinococcosis of the hepatobiliary tract - the role of new imaging techniques for improved diagnosis. Med Ultrason 2020;22:75-84.
29. Brunetti E, Tamarozzi F, Macpherson C, et al. Ultrasound and Cystic Echinococcosis. Ultrasound Int Open 2018;4:E70-E78.
30. Dietrich CF, Mueller G, Beyer-Enke S. Cysts in the cyst pattern. Z.Gastroenterol. 2009;47:1203-1207.
31. Richter J, Botelho MC, Holtfreter MC, et al. Ultrasound assessment of schistosomiasis. Z Gastroenterol 2016;54:653-660.
32. Richter J, Azoulay D, Dong Y, et al. Ultrasonography of gallbladder abnormalities due to schistosomiasis. Parasitol Res 2016;115:2917-2924.
33. Dong Y, Mao F, Cao J, et al. Differential diagnosis of gallbladder ascariasis debris: the added value of contrast enhanced ultrasound with high frequency transducer. Med Ultrason 2018;20:413-419.
34. Dietrich CF, Sharma M, Chaubal N, et al. Ascariasis imaging: pictorial essay. Z Gastroenterol 2017;55:479-489.
35. Dietrich CF, Kabaalioglu A, Brunetti E, Richter J. Fasciolosis. Z Gastroenterol 2015;53:285-290.
36. Dietrich CF, Atkinson NSS, Lee WJ, et al. Never seen before? Opisthorchiasis and Clonorchiasis. Z Gastroenterol 2018;56:1513-1520.
37. Dietrich CF, Cretu C, Dong Y. Imaging of toxocariasis. Adv Parasitol 2020;109:165-187.
38. Braden B, Helm B, Fabian T, Dietrich CF. [Bacillary angiomatosis of the liver, a suspected ultrasound diagnosis?]. Z.Gastroenterol. 2000;38:785-789.
39. Barreiros AP, Otto G, Ignee A, Galle P, Dietrich CF. Sonographic signs of amyloidosis. Z.Gastroenterol. 2009;47:731-739.

40. Chiorean L, Cui XW, Tannapfel A, et al. Benign liver tumors in pediatric patients - Review with emphasis on imaging features. World J Gastroenterol 2015;21:8541-8561.
41. Schreiber-Dietrich DG, Leuschner I, Tannapfel A, et al. [Primary liver tumours in childhood]. Z Gastroenterol 2015;53:1267-1275.
42. Moller K, Tscheu T, De Molo C, et al. Comments and illustrations of the WFUMB CEUS liver guidelines: rare congenital vascular pathology. Med Ultrason 2022;24:461-472.
43. Ignee A, Moller K, Thees-Laurenz R, et al. Comments and illustrations of the WFUMB CEUS liver guidelines: Rare focal liver lesions - infectious (bacterial). Med Ultrason 2023, doi: 10.11152/mu-4065.
44. Zander T, Zadeh ES, Moller K, et al. Comments and illustrations of the WFUMB CEUS liver guidelines: Rare focal liver lesion - infectious parasitic, fungus. Med Ultrason 2023, doi: 10.11152/mu-4091.
45. Moller K, Safai Zadeh E, Gorg C, et al. Prevalence of benign focal liver lesions and non-hepatocellular carcinoma malignant lesions in liver cirrhosis. Z Gastroenterol 2023;61:526-535.
46. Moller K, Safai Zadeh E, Gorg C, et al. Focal Liver Lesions other than Hepatocellular Carcinoma in Cirrhosis: Diagnostic Challenges. J Transl Int Med 2022;10:308-327.
47. Moller K, Braden B, Culver EL, et al. Secondary sclerosing cholangitis and IgG4-sclerosing cholangitis - A review of cholangiographic and ultrasound imaging. Endosc Ultrasound 2023;12:181-199.
48. Zafrani ES, Cazier A, Baudelot AM, Feldmann G. Ultrastructural lesions of the liver in human peliosis. A report of 12 cases. Am J Pathol 1984;114:349-359.
49. Wagner E. Fall von Blutcysten der Leber. Arch Heilkd 1861;2:369-370.
50. Schoenlank W. Ein Fall von Peliosis hepatis. Virchows Archiv für pathologische Anatomie und Physiologie und für klinische Medizin 1916;222:358-364.
51. Iannaccone R, Federle MP, Brancatelli G, et al. Peliosis hepatis: spectrum of imaging findings. AJR Am J Roentgenol 2006;187:W43-52.
52. Iwata T, Adachi K, Takahashi M. Peliosis Hepatis Mimicking Malignant Hypervascular Tumors. J Gastrointest Surg 2017;21:1095-1098.
53. Wanless IR, Huang W-Y. 12 - Vascular disorders. In: Burt AD, Portmann BC, Ferrell LD, eds. MacSween's Pathology of the Liver (Sixth Edition). Edinburgh: Churchill Livingstone, 2012; 601-643.
54. Izumi S, Nishiuchi M, Kameda Y, et al. Laparoscopic study of peliosis hepatis and nodular transformation of the liver before and after renal transplantation: natural history and aetiology in follow-up cases. J Hepatol 1994;20:129-137.
55. Degott C, Rueff B, Kreis H, Duboust A, Potet F, Benhamou JP. Peliosis hepatis in recipients of renal transplants. Gut 1978;19:748-753.
56. Ahsan N, Holman MJ, Riley TR, Abendroth CS, Langhoff EG, Yang HC. Peloisis hepatis due to Bartonella henselae in transplantation: a hemato-hepato-renal syndrome. Transplantation 1998;65:1000-1003.
57. Kim SB, Kim DK, Byun SJ, et al. Peliosis hepatis presenting with massive hepatomegaly in a patient with idiopathic thrombocytopenic purpura. Clin Mol Hepatol 2015;21:387-392.
58. Scoazec JY, Marche C, Girard PM, et al. Peliosis hepatis and sinusoidal dilation during infection by the human immunodeficiency virus (HIV). An ultrastructural study. Am J Pathol 1988;131:38-47.
59. Leong SS, Cazen RA, Yu GS, LeFevre L, Carson JW. Abdominal visceral peliosis associated with bacillary angiomatosis. Ultrastructural evidence of endothelial destruction by bacilli. Arch Pathol Lab Med 1992;116:866-871.
60. Gazineo JL, Trope BM, Maceira JP, et al. Bacillary angiomatosis: description of 13 cases reported in five reference centers for AIDS treatment in Rio de Janeiro, Brazil. Rev Inst Med Trop Sao Paulo 2001;43:1-6.
61. Wakabayashi T, Onda H, Tada T, Iijima M, Itoh Y. High incidence of peliosis hepatis in autopsy cases of aplastic anemia with special reference to anabolic steroid therapy. Acta Pathol Jpn 1984;34:1079-1086.
62. Xiong WJ, Hu LJ, Jian YC, et al. Focal peliosis hepatis in a colon cancer patient resembling metastatic liver tumor. World J Gastroenterol 2012;18:5999-6002.
63. Choi SK, Jin JS, Cho SG, et al. Spontaneous liver rupture in a patient with peliosis hepatis: a case report. World J Gastroenterol 2009;15:5493-5497.
64. Battal B, Kocaoglu M, Atay AA, Bulakbasi N. Multifocal peliosis hepatis: MR and diffusion-weighted MR-imaging findings of an atypical case. Ups J Med Sci 2010;115:153-156.
65. Dave YA, Gupta A, Shah MM, Carpizo D. Liver haematoma as a presentation of peliosis hepatis. BMJ Case Rep 2019;12:e226737.
66. Hung NR, Chantrain L, Dechambre S. Peliosis hepatis revealed by biliary colic in a patient with oral contraceptive use. Acta Chir Belg 2004;104:727-729.
67. Levin D, Hod N, Anconina R, Ezroh Kazap D, Shaco-Levy R, Lantsberg S. Peliosis Hepatis Simulating Metastatic Liver Disease on FDG PET/CT. Clinical nuclear medicine 2018;43:e234-e236.
68. Dong Y, Wang WP, Lim A, et al. Ultrasound findings in peliosis hepatis. Ultrasonography 2021;40:546-554.
69. Kim EA, Yoon KH, Jeon SJ, et al. Peliosis hepatis with hemorrhagic necrosis and rupture: a case report with emphasis on the multi-detector CT findings. Korean J Radiol 2007;8:64-69.
70. Wang SY, Ruggles S, Vade A, Newman BM, Borge MA. Hepatic rupture caused by peliosis hepatis. J Pediatr Surg 2001;36:1456-1459.
71. Cimbanassi S, Aseni P, Mariani A, Sammartano F, Bonacina E, Chiara O. Spontaneous hepatic rupture during pregnancy in a patient with peliosis hepatis. Ann Hepatol 2015;14:553-558.
72. Kim SH, Lee JM, Kim WH, Han JK, Lee JY, Choi BI. Focal peliosis hepatis as a mimicker of hepatic tumors: radiological-pathological correlation. J Comput Assist Tomogr 2007;31:79-85.

73. Ferrozzi F, Tognini G, Zuccoli G, Cademartiri F, Pavone P. Peliosis hepatis with pseudotumoral and hemorrhagic evolution: CT and MR findings. Abdominal imaging 2001;26:197-199.
74. Schuldes M, Weickert U. [Contrast-enhanced ultrasound in suspected liver metastases]. Dtsch Med Wochenschr 2011;136:1255-1256.
75. Hocke M, Ignee A, Topalidis T, Stallmach A, Dietrich CF. Contrast-enhanced endosonographic Doppler spectrum analysis is helpful in discrimination between focal chronic pancreatitis and pancreatic cancer. Pancreas 2007;35:286-288.
76. Dietrich CF, Tana C, Caraiani C, Dong Y. Contrast enhanced ultrasound (CEUS) imaging of solid benign focal liver lesions. Expert Rev Gastroenterol Hepatol 2018;12:479-489.
77. Dietrich CF. Contrast-Enhanced Ultrasound of Benign Focal Liver Lesions. Ultraschall Med 2019;40:12-29.
78. Gouya H, Vignaux O, Legmann P, de Pigneux G, Bonnin A. Peliosis hepatis: triphasic helical CT and dynamic MRI findings. Abdom Imaging 2001;26:507-509.
79. Tsukamoto Y, Nakata H, Kimoto T, Noda T, Kuroda Y, Haratake J. CT and angiography of peliosis hepatis. AJR Am J Roentgenol 1984;142:539-540.
80. Xu HX, Xie XY, Lu MD, et al. Unusual benign focal liver lesions: findings on real-time contrast-enhanced sonography. J Ultrasound Med 2008;27:243-254.
81. Ignee A, Gebel M, Caspary WF, Dietrich CF. [Doppler imaging of hepatic vessels - review]. Z Gastroenterol 2002;40:21-32.
82. Ferrozzi F, Tognini G, Zuccoli G, Cademartiri F, Pavone P. Peliosis hepatis with pseudotumoral and hemorrhagic evolution: CT and MR findings. Abdom Imaging 2001;26:197-199.
83. Crocetti D, Palmieri A, Pedulla G, Pasta V, D'Orazi V, Grazi GL. Peliosis hepatis: Personal experience and literature review. World J Gastroenterol 2015;21:13188-13194.
84. Yekeler E, Dursun M, Tunaci A, Cevikbas U, Rozanes I. Diagnosing of peliosis hepatis by magnetic resonance imaging. J Hepatol 2004;41:351.
85. Slim R, Hachem N, Smayra V, et al. Education and Imaging. Hepatobiliary and pancreatic: peliosis hepatis associated with multiple myeloma. J Gastroenterol Hepatol 2014;29:5.
86. Bexten T, Burck I, Bechstein WO, Moench C. [Hepatic peliosis--a rare liver tumor and challenge for diagnostic investigation plus therapy]. Dtsch Med Wochenschr 2012;137:1505-1509.
87. Stolzel U, Doss MO, Schuppan D. Clinical Guide and Update on Porphyrias. Gastroenterology 2019;157:365-381 e364.
88. Stolzel U, Kubisch I, Stauch T. [Porphyrias-what is verified?]. Internist (Berl) 2018;59:1239-1248.
89. Stolzel U, Stauch T, Doss MO. [Porphyrias]. Internist (Berl) 2010;51:1525-1533; quiz 1534.
90. Deybach JC, Puy H. Hepatocellular carcinoma without cirrhosis: think acute hepatic porphyrias and vice versa. J Intern Med 2011;269:521-524.
91. Wang B. The acute hepatic porphyrias. Transl Gastroenterol Hepatol 2021;6:24.
92. Ma Y, Teng Q, Zhang Y, Zhang S. Acute intermittent porphyria: focus on possible mechanisms of acute and chronic manifestations. Intractable Rare Dis Res 2020;9:187-195.
93. Balwani M. Erythropoietic Protoporphyria and X-Linked Protoporphyria: pathophysiology, genetics, clinical manifestations, and management. Mol Genet Metab 2019;128:298-303.
94. Lee KG, Hyun JJ, Seo YS, et al. Liver cirrhosis induced by porphyria cutanea tarda: a case report and review. Gut Liver 2010;4:551-555.
95. Usta Atmaca H, Akbas F. Porphyria cutanea tarda: a case report. J Med Case Rep 2019;13:17.
96. Frank J, Poblete-Gutierrez P. Porphyria cutanea tarda--when skin meets liver. Best Pract Res Clin Gastroenterol 2010;24:735-745.
97. Kauppinen R, Mustajoki P. Acute hepatic porphyria and hepatocellular carcinoma. Br J Cancer 1988;57:117-120.
98. Andant C, Puy H, Bogard C, et al. Hepatocellular carcinoma in patients with acute hepatic porphyria: frequency of occurrence and related factors. J Hepatol 2000;32:933-939.
99. Andant C, Puy H, Deybach JC, Soule JC, Nordmann Y. Occurrence of hepatocellular carcinoma in a case of hereditary coproporphyria. Am J Gastroenterol 1997;92:1389-1390.
100. Luvai A, Mbagaya W, Narayanan D, et al. Hepatocellular carcinoma in variegate porphyria: a case report and literature review. Ann Clin Biochem 2015;52:407-412.
101. Innala E, Andersson C. Screening for hepatocellular carcinoma in acute intermittent porphyria: a 15-year follow-up in northern Sweden. J Intern Med 2011;269:538-545.