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**Peliosis hepatis: Personal experience and literature review**

Crocetti D *et al*. Peliosis hepatis

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**Abstract**

Peliosis hepatis (PH) is a disease characterized by multiple and small blood-filled cysts within the parenchymatous organs. PH is a very rare disease, more common in adults, and when affects the liver, it comes to surgeon attention only in extremely urgent situation after the lesion’s rupture with the resulting hemoperitoneum. We report the case of a 29-year old woman affected by recurring abdominal pain. Computed tomography scan showed a hepatic lesion formed by multiple hypodense areas, which showed an early acquisition of the contrast during the arterial phase. By the way, it returned hysodense with the remaining parenchyma during the late venous phase. We decided on performing a liver resection of segment VII avoiding biopsy for safety reasons. The histopathological examination confirmed the diagnosis of focal PH. PH should always be considered in the differential diagnosis of hepatic lesion. Clinicians discussed over the possible causes and issues related to the differential diagnosis in addition to the appropriate therapeutic approach. The fortuitous finding of a lesion, potentially compatible with PH, requires elective surgery with diagnostic and therapeutic intents. The main aim is to prevent the risk of a sudden bleeding that, in absence of properly equipped structures, may have a fatal outcome.

**Key words:** Peliosis hepatis; Hemoperitoneum; Haemorrhagic hepatic cysts; Surgical treatment; Liver mass

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**Core tip:** We report the case of a 29-year old woman affected by recurring abdominal pain. Computed tomography scan showed a hepatic lesion formed by multiple hypodense areas, which showed an early acquisition of the contrast during the arterial phase. We perform a liver resection of segment VII avoiding biopsy for safety reasons. The histopathological examination confirmed the diagnosis of focal hepatic peliosis. Surgery was successful and the patient had a good recovery.

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**INTRODUCTION**

Peliosis hepatis (PH) is a disease characterized by multiple, small blood-filled cysts within the parenchymatous organs. Peliosis is a very rare disease and it comes to surgeon attention just after the rupture, even spontaneous, of the lesion resulting in a massive hemoperitoneum. In this particular case, PH acquires the role of potentially fatal disease[1-5].

**CASE REPORT**

Our experience refers to a 29-year old woman who was hospitalized at the Hepato-Pancreato Biliary Surgery Unit at Rome “Regina Elena” Cancer Institute because of recurrent abdominal pain even though it was discontinuous over the years. The pain started from the back and became chronic even though in 2006 she was subjected to a laparoscopic cholecystectomy performed for gallbladder stones without any postoperative complications.

The patient came to our Hospital for persistent symptoms after cholecystectomy, characterized by epigastric pain spread to shoulder blade area. Past medical history was negative for other kind of abdominal diseases. The ultrasound did not show the presence of hepatic masses, except for the presence of biliary sand. The patient did not report chronic use of drugs, such as steroids or oral contraceptives, antibiotics, oestrogens or tamoxifen. The past medical history was negative for infection with Rickettsia, hepatitis, HIV virus, tuberculosis and other diseases of the hematopoietic system. Abdominal ultrasound showed multiple low echogenicity liver cysts areas in the right lobe in addition to some other high echogenicity areas, mainly located in the seventh segment (total diameter = 4 cm). These features suggested the presence of multiple abscesses or a newly formed process of indeterminable nature. A contrast computed tomography (CT) was also performed: the lesion appeared made by multiple low attenuation areas, and an early acquisition of contrast (Figure 1) with centrifugal progression was noticed during the arterial phase. The whole parenchyma appeared so much homogeneously hysodense (similarly to large vessels) during the late venous phase that the same lesion appeared indistinguishable.

The patient was also subjected to magnetic resonance imaging (MRI) examination both with and without contrast. In T2-weighted sequences, the lesions were hyperintense as the remaining parenchyma with multiple high signal spots (Figure 2); in T1-weighted sequences the lesions were hypointense. (Figure 3). As in CT, an early enhancement was found. CT and MRI images did not guide the diagnosis for any of the hepatic lesions most commonly identified. The only plausible hypothesis seemed to be that of a haemangioma, even though the presence of necrotic areas of persistent attenuation requires a differential diagnosis with abscesses, hematomas, and liver tumours.

Percutaneous biopsy was not performed because the mass was located in the liver and because of the remarkable lesions’ vascularization.

Due to the variety of pathologies, among which cancer, that may determine a biliary duct dilatation with pain and/or segmental Caroli's disease, and the impossibility to establish or exclude the presence of PH in the differential diagnosis, we decided to operate the patient fully removing the lesion throughout the resection of segment VII with diagnostic and therapeutic intent.

The histopathological examination confirmed the presence of haemorrhagic cyst cavities in the liver parenchyma, with size from less than one to several millimetres in diameter, and excluded the presence of neoplastic cells.

The histopathological features were useful for the diagnosis of the focal PH (Figures 4 and 5).

**DISCUSSION**

PH was first described in 1861 by E. Wagner. In 1916, W. Schoenlack denominated it peliosis from the Greek word “pelios” that means “reddish”, “bluish”[6-8].

Its pathogenesis remains uncertain, although the triggering event that produces the dilation of sinusoids might be due to an altered outflow with the consequent damage of the sinusoids’ walls and the dilatation of the central vein of the hepatic lobule[9,10]. A pathogenesis determined by the hepatocellular necrosis with the subsequent formation of blood-filled cavitiesis also supposed[10,11].

PH could have a focal development within liver parenchyma, even though more often it is widespread[6,12]. Even the possibility of hepatic rupture varies in percentage considering the different sites of the disease: on the right liver in 75% of the cases; on the left liver in 11% of the cases; on both in 14% of the cases[1].

Although most commonly identified in the liver, the same process may occur in the spleen, bone marrow, lymph nodes, and more rarely in lungs, stomach, intestine, parathyroid, pancreas, pituitary gland and kidneys[2,5,6,7,13]. Its incidence does not vary according to the gender and a greater frequency is observed among adults, even though it can be found in paediatric age too[11,14,15].

PH pathogenesis remains uncertain, but Pan *et al*[14] divided the etiological factors between: (1) factors related with drugs assumption; (2) factors related with autoimmune mechanisms; and (3) factors related with infectious cases.

During the course of time, many drugs have been associated with PH onset, such as steroids, oral contraceptives, tamoxifen, methotrexate, thiopurine, azathioprine and iron chelators, in addition to several toxins, such as arsenic or thorium. Even alcohol consumption facilitate the onset of PH since it reduces the amount of glutathione that would have a protective and detoxifying effect for sinusoidal endothelial cells, especially during the metabolism of immunomodulatory drugs. As we already said[7,14,16], patients who take this kind of drugs are more likely to develop PH[7,8,17,18].

A severe risk is present also in case of association between neoplastic diseases (such as hepatocellular carcinoma), in particular myeloproliferative disease or in those diseases involving the lymphatic system[13,14,19,20,21], and Castleman's disease[13,22]. Some authors have reported some cases of association between arteritis, telangiectasia and intestinal lymphangiectasia[10,23,24] and cases of portal circulation hypertension in not transplanted patients[25], myopathy tubular[26,27], cystic fibrosis[2] and celiac disease[13].

Some other authors have described associations between PH and leprosy, tuberculosis or sifilidis, or more in general, infectious cases of HIV[2,8,11,13,28,29], such as the bacillus infection of Rickettsia family (*Rochalimaea* *henselae* and *R. quintana*)[2,6,13,30]. In 20%-50% of the cases it is not possible to identify any comorbidity.

Imaging studies, (CT angiography; ETG and MRI angiography), even though were carried out through a hepatospecific contrast in hepatic sites of the disease, did not allow to clearly diagnose PH[11,30-37]. The differential diagnosis in hepatic localizations is made with adenomas, haemangioma, focal nodular hyperplasia, Caroli's disease, multiple abscesses or metastatic adenocarcinomas[3,11,20,32,38,39]. Lesions may show a variable diameter from few millimetres up to more than 4 cm[5,38,40].

Ultrasound scan may show a pseudo-cystic lesion of the hepatic parenchyma, which may correspond to venous lakes that are commonly described by the histopathological examination[5]. Even the condition of the hepatic parenchyma determines the sonographic features of PH that appears hyperechoic, in presence of steatosis, and hypoechoic in a normal parenchyma. Through Doppler examination it is possible to show the intra- or perilesional vascularity[11,32,37]. Some authors have also reported the use of ultrasound with contrast; anyway an effective higher sensitivity was not emphasized[37,38]. Angiography examination shows multiple hypervascularized nodules during the late arterial phase and the enhancement is more pronounced during the parenchymal phase and persists during the venous phase[1,32]. CT images may show small lesions between few millimetres and 1-4 cm in diameter[5].

These lesions appear as multiple areas with low signal. Their aspect depends on the degree of thrombosis or haemorrhage inside the cavity, since they can appear with variable density (although more often hypodense) and contain some calcifications[2,7,11,32].

In early arterial phase, they may appear hyperdense and then they become hysodense with the remaining parenchyma[5,32]. The dimensions of the cavities can have a density comparable to that of vessel or they can even appear without *enhancement* when thrombosed[5,24,32].

CT scan cannot detect lesions smaller than 1 cm in diameter, and this method does not appear to be more sensitive compared with the ultrasound in PH diagnosis[6,24]. On the other hand MRI examination represents the gold standard in radiological diagnosis for this disease[32], especially if it is combined with the use of a hepatospecific contrast medium[30].

The imaging varies considerably depending on the blood component of the lesion[1,3,5,8]. Usually lesions are never exophytic[32]; in T2 weighted-sequences may appear hyperintense lesions compared with the surrounding parenchyma with high signal multiple spots, that are attributable to haemorrhagic necrosis[5,8,24]. In T1 weighted-sequences different kind of lesions can be detected: hypo, hyso or hyperintense, that employ an enhancement at the time of contrast medium administration. This enhancement is generally centrifugal (as for the CT), but can also be centripetal[5].

In T1 acquisitions with fat suppression, during the late stage, and after the use of gadobenate dimeglumine, a strong contrast with ramifications due to the vascular component is observed[24,32]. The acquisition of the contrast medium during the late phase, can sometimes suggest a differential diagnosis with haemangioma. The doubt can only be solved after the surgery[3,4]. For the differential diagnosis with proliferative lesions, the use of PET allows us to exclude a high metabolic activity of the lesion[39].

Some authors claim that, due to the high risk of bleeding, perform an open biopsy is essential in order to realize the differential diagnosis under intraoperative ultrasound supervision[2,6,13,31,34,40]. The macroscopic examination shows that peliotic lesions in the cutting section present haemorrhagic cyst cavities of various sizes (from < 1 mm to several centimeters) with features of "Swiss cheese"[1,3]. From the histopathological point of view, the differential diagnosis is used in order to diversify the sinusoids’ dilatation from Budd-Chiari syndrome, which determines a venous congestion of the liver due to a vascular occlusion[1,4,41].

Yanoff *et al*[42] described microscopically two different types of PH: the "parenchymal peliosis", that consists of irregular cavities that are surrounded neither by the sinusoidal cells nor by fibrous tissue, and the "peliosis flebectasica" characterized by spherical regular cavities coated by endothelium and/or fibrosis[3,5,32].

Even though the blood-filled cavities not always have endothelial coverage, it has been observed that it is quickly reconstituted. The continuity and the new breaking of the sinusoids’ endothelium do not constitute a real classification parameter[5], in this manner it is more adequate to describe such circumstances as temporally different aspects of the same pathology[33].

PH does not show a well-defined evolution. It can get worse asymptomatically and represent an accidental finding that occurs during investigations over other diseases or on the autopsy table[2,32]. Some authors claim that PH could be associated to liver failure clinical features with finding of hepatomegaly, portal hypertension, cholestasis and, more rarely, in cirrhotic patients positive for HCV[6,32,43] or, in cases of rupture of the lesions with hemoperitoneum, hemodynamic decompensation with lethargy and abdominal pain[1,3,6,32]. In other cases, especially when PH occurs at very young age, the disease shows important effects compression with stenosis of the vena cava[44].

In other cases after the interruption of the steroid therapy, or the resolution of the subsequent infections, a total regression was noticed[5,6,32]. The regression can also occur without any connection with the past medical history, especially in those cases (20%-50%) that do not permit to associate PH to any kind of etiology[30]. Even though some authors support the possibility to perform a transplantation in acute liver failure cases, or to use hepatectomy for the diagnosis and treatment of PH, surgery is more and more used in urgency case as treatment for the bleeding that may occur. At the same time, for the same issue some authors proposed embolization that may be performed by interventional radiologist[45,46] or during the operation or the laparoscopic biopsy, as we already mentioned.

The PH is a rare disease that often arises with atypical symptoms, but more often is asymptomatic and occurs in urgent situation after the spontaneous lesion’s rupture or can also be related to minor trauma. The peculiarity of our experience is that we did not diagnose it in urgent situation, but thanks to the preoperative investigations. We decided to operate by means of diagnostic and therapeutic means through which we could prevent future bleeding complications.

Patients are rarely treated with elective surgery because of the diagnostic difficulty and because none of the preoperative investigations is useful to make a certain diagnosis. As already described in literature[11,32], the ultrasound (I level investigation) shows only the presence of a hypervascularised lesion, usually hypoechoic, without further features. Even TC showed only the presence of a hypervascular lesion in early stage (Figure 1) with rapid washout of contrast, that was not useful to allow a differential diagnosis[5,32].

Even MRI with contrast material did not allow the diagnostic classification because in T2-weighted sequences, lesions were hyperintense as the remaining parenchyma with multiple high signal spots. On the contrary, in T1-weighted sequences lesions were hypointense, thus hardly distinguishable from that of the hemangiomatous lesions[4,32]. We decided not to perform a percutaneous biopsy because of the considerable vascularity of the lesion and the consequent high risk of bleeding[31,34,40].

We decide to perform an hepatic resection that, according to Samyn *et al*[6], permits to better manage the prior haemostasis, also in concordance with Fowell *et al*[38] assertions and patient’s good conditions, such as her young age and the site of the lesion in site VII (that can be easily reached in surgery). The histopathological examination on the whole mass confirmed the presence of haemorrhagic cyst cavities in the hepatic parenchyma, with size from less than one to several millimetres in diameter[3,5,32]. It also confirmed the diagnosis of hepatic peliosis (PH). In the case reported, we could not identify any past medical history element because of the nature of the lesion. In 20%-50% of the cases, in fact, it is not possible to define the PH etiology[11,24,30,31]. In addition to permit the diagnosis and the pathology therapy, the surgical intervention permitted also to prevent possible haemorrhagic events that are very frequent in patients affected by PH.

In conclusion, PH is a rare and difficult to diagnose disease. From an iconographic point of view, it is almost impossible to carry out a differential diagnosis especially during the preoperative phase, because of the presence of many other diseases with similar characteristics. The conspicuous vascularization, that is not only a PH feature, requires caution toward percutaneous biopsies. The PH is by definition an asymptomatic disease, as long as a dramatic event, such as the haemorrhage, makes it obvious. According to us, the fortuitous finding of a lesion, potentially compatible with PH, requires elective surgery with diagnostic and therapeutic intents. The main aim is to prevent the risk of a sudden bleeding that, in absence of properly equipped structures, may have a fatal outcome.

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**Figure 1 Computed tomography, arterial phase: The lesion is composed of more areas of low attenuation and during the arterial phase showed an early acquisition of the contrast.**



**Figure 2** **Magnetic resonance, T2-weighted sequences: The lesion is hyperintense as the remaining parenchyma with multiple foci of high signal.**



**Figure 3 Magnetic resonance, T1-weighted sequences: The lesion appears hypointense.**



**Figure 4 Haepatic tissue with pseudovascular pattern (without endothelium) filled of debris and eritrocytes; note the rich intratissutal capillary vessels presence.** Hematoxylin and eosin staining, magnification × 200



**Figure 5 Pseudovascular spaces associated to reactive lymphoid aggregates with macrophage cells and tissutal hystiocytes.** Hematoxylin and eosin staining, magnification × 200.