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Review

Hepatic hematoma: A challenging, emergency disease

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ABSTRACT

Background: Hepatic hematoma (HH) consists of a collection of blood and clots inside the liver, with various etiologies: traumatic, pre-existing macro/micro vascular disease, previous diagnostic/interventional procedures, and open or laparoscopic operations. Clinically, it may go unnoticed when its signs are mild or non-specific; otherwise, it may be followed by challenging emergency complications.
Methods: Our experience, although limited, allowed to study some pathophysiological features and role of diagnostic and therapeutic procedures.
Results: Abundant hepatic vascularization is a predisposing factor for HH. Its progressive expansion may be promoted by a self-feeding mechanism of compression, ischemia and necrosis of the surrounding liver parenchyma. Differently, high internal tension may cause a parietal tear, while a large HH, due to mass effect, may produce a secondary hepatic compartment syndrome through compression on the inferior vena cava, or a sectoral 'Budd-Chiari-like' syndrome, characterized by downstream portal venous congestion.
Conclusions: HH requires a careful diagnostic study, preferably by CT or MRI, especially when complicated by rupture, internal hemorrhage, haemobilia, hepatic compartment syndrome or acute infection. Treatment varies from a percutaneous drainage and selective vascular embolization, to surgical parietal fenestration and hepatic resection.

Introduction

Hepatic hematoma (HH) can be defined as an intrahepatic collection of blood, clots, necrotic and inflammatory cells, which during its evolution can become surrounded by a fibrous capsule, even calcified [1]. It can originate from intrahepatic aneurysms, or pseudo-aneurysms, which fissure and rupture. Mainly today, it can complicate hepatic interventional procedures, such as biopsy, arterial embolization, TIPS, ERCP, percutaneous biliary drainage, and various laparoscopic or open surgical operations [2]. Otherwise, it may follows vasculitis or other microvascular diseases, typically HELLP syndrome, with a mechanism of endothelial damage in the portal venules or hepatic sinusoids, causing multiple microhemorrhagic foci, which subsequently coalesce into a full-blown hematoma [3,4]. Today, the diagnosis of this rare disease is facilitated by cross-sectional imaging tools such as Ultrasound (US), Computed Tomography (CT) and Magnetic Resonance Imaging (MRI). Our aim is to alert surgeons to this pathology, and to indicate general rules of treatment.

Pathophysiology

Firstly, the onset of HH is facilitated by the abundant liver vascularization, consisting of a triple vasculature, one arterial, and two venous, afferent and efferent. In particular, the rich network of small arteries, just below Glisson's capsule, explains its frequent peripheral localization within the liver parenchyma [5,6]. Secondly, the HH expands in proportion to the strength of its blood supply, until it compresses the surrounding liver parenchyma, which becomes ischemic and necrotic (Fig. 1, A and B) [2]. This process may also involve the diaphragm, paving the way for a dangerous HH expansion within the mediastinum or right hemithorax [7]. Thirdly, the absence of an adequate fibrous capsule, as in its early stage, and an increased internal tension, by Laplace law, can directly cause its rupture, preferentially where it protrudes from the liver surface. Fourthly, a large HH can complicate with a sectorial hepatic compartment syndrome, compressing adjacent portal vein branches and/or hepatic vein roots. A blood flow, reduced in its inflow or hindered in its outflow, becomes an additional factor in promoting necrosis in the surrounding liver parenchyma and increasing the HH size. Fifthly, elective compression in hepatic veins and subsequent congestion of the corresponding portal vein branches may also generate a "sectorial Budd-Chiari like syndrome", given the absence of suitable pre-

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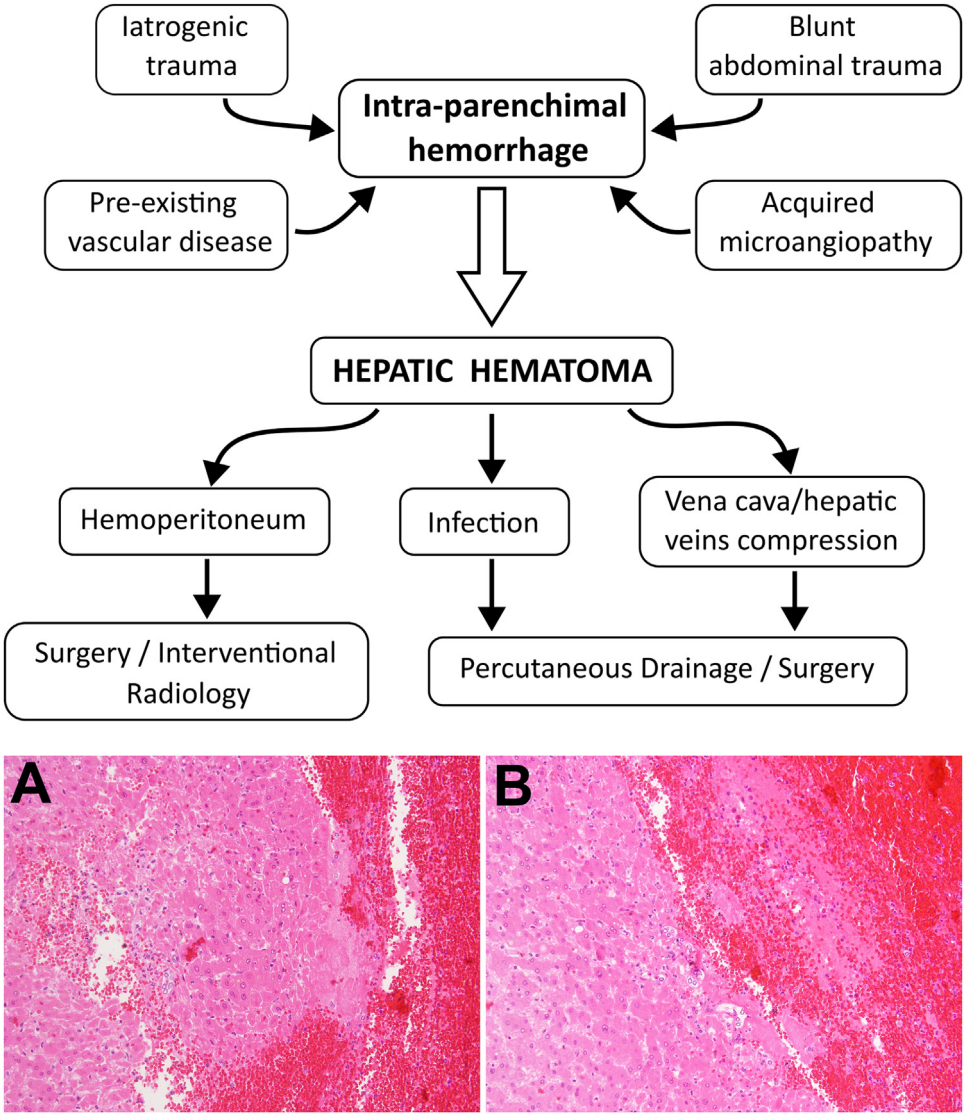

Fig. 1. Hepatic hematoma: pathophysiological schema. Histology (A and B) shows a recent hepatic hematoma developing towards the contiguous ischemic and necrobiotic parenchyma (Hematoxylin-Eosin stained, 200X).

existing collaterals between proximal portal vein branches, or with hepatic veins roots. However, in the medium term, this effect may be mitigated by expanded pre-existing accessory hepatic veins, usually located in the sub-glissonian space, or by the enlarged collaterals with adjacent portal vein branches, typically of the caudate lobe, directly and with a lower pressure inflowing into the inferior vena cava. Both these compensatory mechanisms can be promoted by Vascular Endothelial Growth Factors, released from the hypoxic areas of the adjacent liver parenchyma [8,9]. Sixth, a large HH, typically in the right lobe of the liver, by mass effect, may directly compress the retro-hepatic segment of the inferior vena cava, obstructing the systemic venous return [10,11]. Seventh, a hemorrhage within an HH may cause a haemobilia, having as an associated pathogenetic element a proximal bile duct directly injured, or involved by a mechanism of parietal erosion, giving rise to an internal biliary fistula. This complication is favored by the low-pressure present in all intrahepatic veins and bile ducts. In fact, it preferentially affects HH, located high up in the hepatic dome, lacking a fibrous capsule and subjected to the diaphragm movements and their suction action during the breathing expiratory phase. Eighth, an HH, as a closed space containing also necrotic material, can turn into a hepatic abscess after translocation of intestinal microbes; it can further complicate with secondary cholangitis, promoted by contaminated lymphatic or blood microchannels (Fig. 1).

Diagnostic and interventional procedures

It should be emphasized that, despite this chain of severe events, an HH may remain silent or paucisymptomatic for a long time, and be discovered incidentally, or misdiagnosed as a simple cyst. At its onset, it appears anechoic on US, hypo-attenuating on CT scan, and strongly hyperintense on T2-weighted MRI or cholangiographic sequences. It usually has regular contours, with a biconvex, biconcave, or "quarter-moon" shape, depending on its peripheral or more central location within the liver, with blood or clots, but no parietal vegetations inside (Fig. 2). Knowledge of previous invasive procedures, surgical operations or liver diseases may facilitate a precise diagnosis. Otherwise, a HH may be discovered through its own complications. Firstly, in case of acute abdomen, sometimes associated with a critical anemia, a fast US may reveal warning signs, such as an intrahepatic mass and a peri-hepatic effusion. When confirmed by CT imaging, unequivocally demonstrating a ruptured HH, they indicate an emergency laparotomy. Secondly, active arterial bleeding within the HH cavity, directly discovered by CT imaging, points to a direct

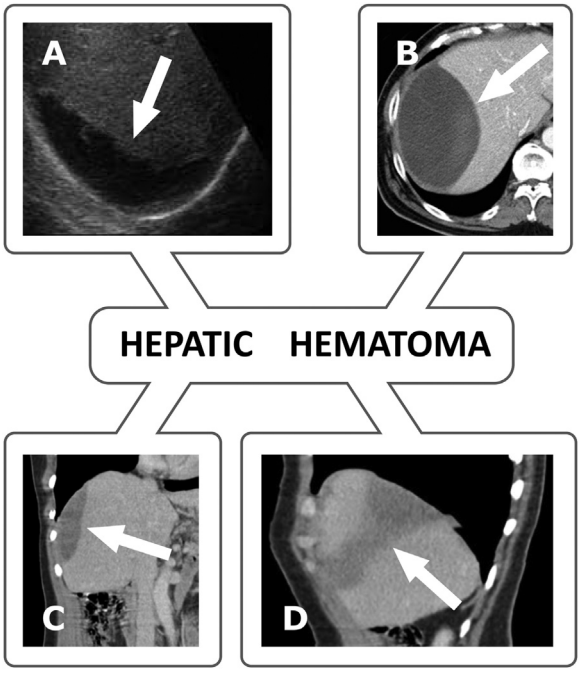

Fig. 2. Hepatic Hematoma images (arrows): **A**) Subcapsular hematoma, "crescent moon" shaped, in the liver right lobe (US scan). **B**) Sub-capsular biconvex hematoma (CT axial scan); **C)** Upper right lobe hepatic lenticular hematoma (CT coronal scan); D): Right lobe biconcave hematoma (CT sagittal scan).

selective embolization. Thirdly, a secondary hemobilia can be detected by CT angiography, demonstrating contrast medium stagnating inside the HH, or clots within enlarged intrahepatic bile ducts [12]. More diagnostically challenging is the rare venous hemobilia, where the bleeding source originates at a lower pressure from a portal vein branch or hepatic vein root. Usually, it can be demonstrated during the portal phase of a CT angiography with the same characteristic signs. Moreover, MRI cholangiography may demonstrate an associated internal biliary fistula through a paradoxical reflux of the contrast medium into the HH cavity [13]. Today, this complex pathology can be treated with a selective embolization, more challenging in the case of venous hemobilia, needing catheterization of a portal vein branch or of a hepatic vein root, respectively through a trans-hepatic US-guided approach, or the jugular vein, eventually with a Cone-beam CT approach [14]. The subsequent treatment of the biliary fistula, if persistent after an internal biliary stenting, may require a hepatic resection. Fourthly, an HH, complicated by an abscess, can be recognized on CT imaging by gas bubbles inside; an urgent procedure, consisting of transcutaneous drainage, represents the first option, postponing a more radical surgical treatment in case of recurrence, or when a better general condition is achieved.

Complications and surgical indications

The HH complications rate is difficult to be quantified, given the absolute rarity of this disease, with single cases reported in the medical literature. Moreover, in the absence of specific and validated guidelines, those applied to liver trauma can be used as landmarks [15]. An uncomplicated HH, especially if developed inside the liver parenchyma, and <3 cm in diameter, can be managed conservatively. Otherwise, in case of progressive increase in size, or bulging from the hepatic surface, an open or laparoscopic treatment becomes indicated. It usually consists of a parietal fenestration, or resection of the 'salient dome', and in a subsequent omentoplasty. In case of HH complete intrahepatic development, a corresponding resection of the surrounding liver parenchyma layer can become necessary [5,15]. These procedures, inspired by hydatid cyst surgery, are less invasive than a total peri-cystectomy and are well suited for HHs with still plastic walls and suitable for secondary remodeling [16,17]. On the contrary, in the case of a rigid fibrous capsule, the risk of a residual cavity and recurrent pathology would indicate a more extensive subtotal peri-cystectomy, performed following the dissection plane, easily found just outside the HH. Otherwise, mainly in the short or medium term, acute complications may occur, to which some general principles of treatment can be applied. First of all, in case of HH rupture, an emergency laparotomy, either open or laparoscopic, allows to treat peritonitis, drain the liver cavity, and perform a partial parietal resection. This solution, although invasive, is safer and more radical than a simple transcutaneous drainage [1]. Secondly, an arterial hemorrhage within an HH, or causing secondary haemobilia, is now subject to an urgent selective embolization. Third, in cases of HH complicated by an abscess, a direct transcutaneous drainage represents the most direct emergency treatment, as an alternative to the a more invasive surgery, consisting of a parietal fenestration or partial resection. This general policy combines two principles of early treatment and 'damage control' and seems particularly appropriate in unstable patients. It should be considered that, after a non-invasive treatment, the residual cavity is at risk of the same recurring or of a different pathology. Therefore, under elective conditions, several solutions can be considered, such as HH fenestration or segmental parietal resection, total peri-cystectomy, and a hepatic mono/bi-segmentectomy, in order to include the proximal bile ducts involved in an internal biliary fistula or haemobilia [5].

Conclusions

Overall, we regard HH as a multifaceted pathology, often followed by several complications, including urgent ones. It deserves to become part of the surgeon's diagnostic and therapeutic armamentarium, especially in the current era of advanced diagnostic/interventional radiological procedures. Moreover, its pathophysiology demonstrates mechanisms active in other liver diseases, but without an equivalent direct impact. Looking forward, the proposed elements, derived from single case studies, can be used as landmarks for larger multi-centric research.

Declaration of Competing Interest

The authors declare no competent interest.

Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.soda.2023.100084.

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