

## Multifocal Hepatohilarar Hydatidosis Complicated by Double Portal and Caval Compression: Contribution of Multimodal Imaging

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### Abstract

Hepatic hydatidosis is the most common location of cystic echinococcosis, but its vascular complications remain rare. We report the case of a 61-year-old man with chronic epigastric pain and positive hydatid serology. Doppler ultrasound and MRI revealed multifocal hepatic hydatidosis with predominance of hepatohilar, consisting of multivesicular formations with daughter vesicles. Hepatohilarar lesions led to marked extrinsic compression of the portal trunk, which remained permeable, associated with periportal shunts, dilatation of the superior mesenteric vein and splenomegaly. Concomitant compression of the inferior vena cava was also observed, without thrombosis. Cholangio-MRI did not show biliary obstruction. This case illustrates an unusual vascular complication of hepatohilarar hydatidosis and highlights the complementary role of Doppler and MRI in distinguishing extrinsic compression, thrombosis and intravascular extension.

**Keywords:** Cystic Echinococcosis; Hepatic Hydatidosis; Hepatic Hilum; Portal Vein; Inferior Vena Cava; Portal Hypertension; MRI; Doppler

### Introduction

Cystic echinococcosis, mainly caused by *Echinococcus granulosus*, remains endemic in several regions, particularly in the Maghreb. The liver is the main site of involvement. Imaging is decisive in recognizing the morphological signs of the disease, specifying its extent and looking for its complications [1-4]. While biliary and infectious complications or rupture are well known, vascular involvement is much rarer. It can be caused by extrinsic compression, thrombosis or, exceptionally, intravascular hydatid extension [1,3,5]. We present a case of multifocal hepatohilarar hydatidosis complicated by simultaneous compression of the portal trunk and the inferior vena cava (IVC), without thrombosis, with signs of portal repercussions.

### Clinical Observation

A 61-year-old man, with no particular reported pathological history, was consulting for epigastric pain that had been evolving for several months. The hydatid serology was positive. There was no fever, jaundice, pruritus or digestive hemorrhage. Other biological data were not available.

### Imaging data

#### Ultrasound and doppler

Abdominal ultrasound showed multiple hepatic and hepatohilarar cystic formations, multilocular and multivesicular, with numerous daughter vesicles achieving a “honeycomb” appearance, very suggestive of hydatidosis. Doppler showed a significant focal reduction in the caliber of the portal trunk in contact with the lesions. The portal flow remained identifiable and no intraluminal material was visualized. Periportal/porto-caval venous leads were associated with splenomegaly.

#### Hepatobiliary MRI

MRI confirmed multifocal involvement of segments I, II, III, VII and VIII. The main liver lesions measured 43 × 30 mm in segment VII and 51 × 27 mm in segment VIII. At the hepatic hilum, several multivesicular formations were individualized, two of which measured 53 × 40 mm and 50 × 39 mm. Their architecture with daughter vesicles confirmed the hydatid nature of the lesions.

Hepatohilarar formations exerted a mass effect on the vascular pedicle. The portal trunk measured 12.8 mm upstream, 4.73 mm at the compression zone, and 11.4 mm downstream. Caliber recovery outside the compressed zone, absence of intraluminal material, and persistence of portal patency favored extrinsic compression rather than thrombosis or intravascular invasion.

The IVC was also compressed: its caliber increased from 20.5 mm upstream to 7.13 mm at the compression level, then to 21.8 mm downstream. Dilation of the superior mesenteric vein, measured at about 14.5 mm, as well as splenomegaly at 14.3 cm completed the signs of portal repercussions.

The cholangio-MRI showed fine intrahepatic bile ducts and an undilated main bile duct, measured at about 5.6 mm, with no identifiable obstruction. The radiological diagnosis was that of multifocal hepatohilarar hydatidosis complicated by a double extrinsic, portal and caval vascular compression, without thrombosis or obstructive biliary complications.

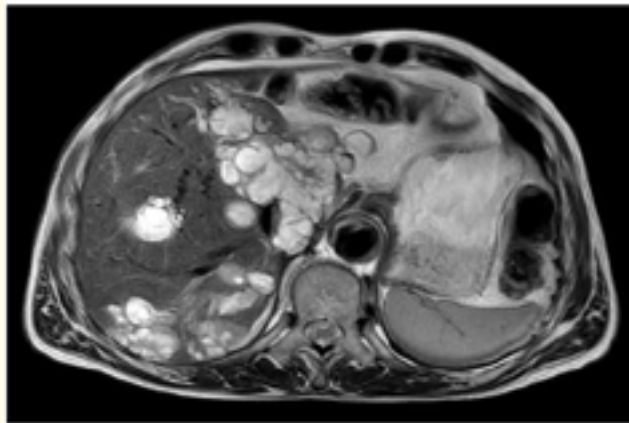


**Figure 1:** T2-weighted axial MRI showing multiple multivesicular hepatic and hepatohilarar cystic formations containing daughter vesicles, achieving a “honeycomb” appearance.



**Figure 2:** Extrinsic compression of the portal trunk: 12.8 mm upstream (A), 4.73 mm at the compression (B) and 11.4 mm downstream (C).



**Figure 3:** Extrinsic compression of the IVC: 20.5 mm upstream (A), 7.13 mm at the compression (B) and 21.8 mm downstream (C).



**Figure 4:** Portal repercussions: periportal Doppler leads (A), dilatation of the superior mesenteric vein (B) and splenomegaly at 14.3 cm (C).

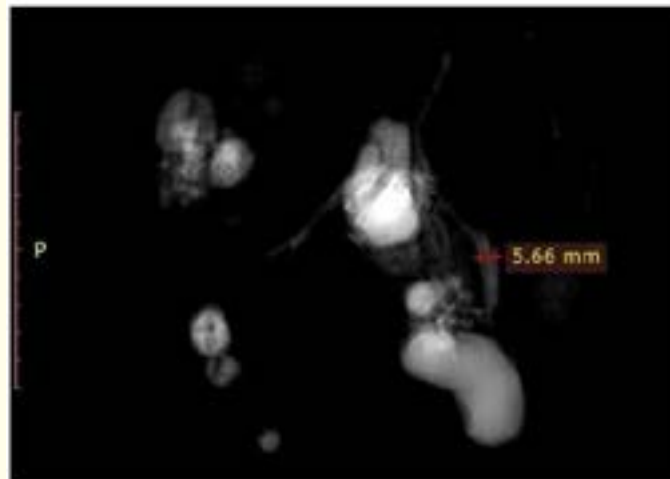


**Figure 5:** Cholangio-MRI showing undilated bile ducts; main bile duct measured at approximately 5.6 mm.

## Discussion

Cystic echinococcosis is a parasitic zoonosis caused mainly by *Echinococcus granulosus*. It remains endemic in many farming regions, including North Africa, the Middle East, Central Asia, China and parts of South America. Human contamination occurs accidentally through ingestion of parasitic eggs, most often as part of the dog-herbivore-human cycle. The liver is the main filter after the intestinal passage and is thus the most common location of the disease, ahead of lung involvement [1-4,8].

The course is usually slow and may remain asymptomatic for several years. The symptoms depend mainly on the site, volume and number of cysts as well as the existence of complications. In hepatic locations, the most frequent manifestations are pain or heaviness of the right hypochondrium or epigastrium, sometimes associated with nausea or vomiting. Jaundice, fever, allergic reaction or manifestations of portal hypertension should be investigated for a biliary or infectious complication, rupture or vascular repercussions. In our patient, the presentation was not very specific, limited to chronic epigastric pain, without jaundice or infectious syndrome, despite multifocal hepatohilarar involvement with significant vascular repercussions.

Radiologically, imaging is central to the positive diagnosis, staging and therapeutic strategy of cystic echinococcosis. Ultrasound is the first-line examination and identifies the main morphological elements: cystic wall, detached membranes, daughter vesicles and multivesicular architecture. The WHO-IWGE ultrasound classification, based on cyst activity, distinguishes between active, transitional and inactive forms and is directly involved in the therapeutic choice. In our observation, the multivesicular appearance with many daughter vesicles was strongly suggestive of an active form [1-4].

CT and especially MRI complement ultrasound in complex forms. MRI offers excellent characterization of cystic content, daughter vesicles and membranes and allows precise mapping of extension. In hepatohilarar locations, it is particularly useful for analyzing relationships with the hepatic pedicle, inferior vena cava and bile ducts. The cholangio-MRI looks for cystic bile communication, obstruction or dilation of the bile ducts [3,4].

Vascular complications are much rarer than biliary or infectious complications. Three mechanisms must be distinguished: extrinsic compression, vascular thrombosis and, exceptionally, direct extension of the hydatid material into the vascular lumen [3,5]. This distinction is important because it modifies the management. In our case, the portal trunk showed a major focal reduction in caliber, going from 12.8

mm upstream to 4.73 mm at the level of compression, then recovering a caliber of 11.4 mm downstream. The absence of intraluminal material, the persistence of portal permeability and the recovery of the caliber after the compressed area argued in favor of pure extrinsic compression rather than thrombosis or portal invasion.

The portal resonance was objectified by the presence of venous leads periportals, dilatation of the superior mesenteric vein and splenomegaly. Portal hypertension secondary to hepatic hydatidosis is rare but already described [6]. The additional particularity of our observation was the concomitant compression of the IVC, whose caliber decreased from 20.5 mm to 7.13 mm on contact with the lesions before normalizing downstream. Compression or caval obstructions secondary to hepatic hydatid cysts have been reported [7]. The association of portal and caval compression, both without thrombosis, underlines the unusual nature of this presentation.

Conversely, no obstructive biliary complications were found. Despite hepato-hilar, the intrahepatic bile ducts remained thin and the main bile duct measured about 5.6 mm. This dissociation between a significant vascular impact and the absence of biliary obstruction is another interesting element of the observation.

Therapeutically, the management of cystic echinococcosis is individualized and is based on the stage of the cyst, its size, its location, its complicated nature or not, as well as on the available expertise. The main options are antiparasitic treatment with benzimidazoles, mainly albendazole, percutaneous techniques such as PAIR (puncture-aspiration-injection-reaspiration), surgery and, for certain uncomplicated inactive lesions, watch and wait. The therapeutic choice must therefore be guided by imaging and does not rely on a single approach [2-4].

Surgery remains a major part of complicated forms, especially when there is a significant mass effect, a biliary complication, a rupture, an infection, a high-risk anatomical location or a vascular complication. Recent WHO recommendations generally consider surgery to be the treatment of choice for complicated liver cysts when the necessary expertise is available. In a hepatohilarar disorder such as that of our patient, the proximity of the portal trunk, the IVC and the bile ducts requires precise pre-therapeutic planning. In particular, imaging must define vascular permeability, look for thrombosis or intravascular extension and specify bile ratios. Percutaneous treatments are not interchangeable with surgery and their indication depends in particular on the morphological stage of the cyst and the absence of certain complications. Albendazole can be used alone in certain selected forms or in combination with interventional treatment depending on the clinical context and the stage of the cyst.

In our observation, the available data do not specify the treatment finally performed.

Finally, the prognosis of hepatic cystic echinococcosis is generally favourable when the diagnosis is made before the onset of severe complications and management adapted to the stage is possible.

However, it depends on the number and location of the lesions, their activity, vasculobiliary ratios, complications and the possibility of complete treatment. Complicated hepatohilarar locations are more delicate because of the risk of compression or damage to vascular and biliary structures.

Recurrence remains possible after treatment and warrants prolonged clinical and radiological follow-up. Ultrasound is generally the main monitoring tool, supplemented by CT or MRI in deep, multiple or anatomically complex forms. According to the WHO, the postoperative mortality reported for cystic echinococcosis averages about 2.2%, while recurrence after surgery is observed in about 6.5% of cases. These figures concern cystic echinococcosis as a whole and should not be interpreted as specific to hepatohilarar forms with vascular compression.

In our case, the preservation of the patency of the portal trunk and the IVC, the absence of thrombosis and the absence of biliary obstruction represent favorable anatomical elements. Nevertheless, the already constituted portal impact and the double vascular compression justify specialized management and close follow-up.

## Conclusion

Hepatohilarar hydatidosis can exceptionally lead to major vascular complications due to mass effect. In this observation, imaging showed severe extrinsic compression of the portal trunk with signs of portal repercussions, associated with compression of the IVC, the two remaining axes permeable and without thrombosis. Doppler ultrasound and MRI are complementary to characterize the vascular mechanism, eliminate intraluminal extension and specify bile ratios before management.

## Bibliography

1. Pedrosa I, *et al.* "Hydatid disease: radiologic and pathologic features and complications". *Radiographics* 20.3 (2000): 795-817.
2. Zalaquett E, *et al.* "Imaging of hydatid disease with a focus on extrahepatic involvement". *Radiographics* 37.3 (2017): 901-923.
3. Greco S, *et al.* "Complications of hepatic echinococcosis: multimodality imaging approach". *Insights into Imaging* 10.1 (2019): 113.
4. Calame P, *et al.* "Role of the radiologist in the diagnosis and management of the two forms of hepatic echinococcosis". *Insights into Imaging* 13.1 (2022): 68.
5. Ertan G, *et al.* "Hydatid disease of the liver with portal vein invasion mimicking portal vein thrombosis". *Arab Journal of Gastroenterology* 20.1 (2019): 50-52.
6. Papadimitriou J, *et al.* "Portal hypertension due to hydatid disease of the liver". *Journal of the Royal Society of Medicine* 83.2 (1990): 120-121.
7. Rajagopal KV and Bishwas R. "Hydatid cyst of the liver presenting as an inferior vena cava obstruction". *Journal of Clinical Ultrasound* 30.2 (2002): 114-116.
8. Das CJ, *et al.* "Abdominal hydatid disease: role of imaging in diagnosis, complications, and management". *Abdominal Radiology (NY)* 50.11 (2025): 5591-5606.

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