

Disasters that develop in the liver due to hydatid cyst: a case report

Desastres que se desarrollan en el hígado debido a un quiste hidatídico: caso clínico

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Abstract

Hilar cavernous transformation is the formation of venous structures rich in collateral around the portal vein. Portal vein thrombosis is a rare entity. Although there are many reasons for its etiology, few cases have been reported secondary to hydatid cysts in the liver. Here, we present a 24-year-old patient with complaints of abdominal pain and swelling. Her CT and MRI scans show cholelithiasis with portal vein thrombosis and hilar cavernous transformation due to giant hydatid cyst compression in the lateral liver sector.

Keywords: Cavernous transformation. Hydatid cyst. Portal vein thrombosis. Gallbladder.

Resumen

La transformación cavernosa hilar es la formación de estructuras venosas ricas en colaterales alrededor de la vena porta. La trombosis de la vena porta es una afección poco frecuente. Aunque existen muchas razones en su etiología, se han descrito pocos casos secundarios a quiste hidatídico en el hígado. Aquí se presenta el caso de una paciente de 24 años con quejas de dolor abdominal e hinchazón. La tomografía computarizada y la resonancia magnética mostraron colelitiasis con trombosis de la vena porta y transformación cavernosa hilar por compresión del quiste hidatídico gigante en el sector lateral del hígado.

Palabras clave: Transformación cavernosa. Quiste hidatídico. Trombosis de la vena porta. Vesícula biliar.

Introduction

Hydatid cyst is a zoonotic disease caused by Echinococcus parasites. It is an endemic disease worldwide, especially in the Middle East, Asia, India, and South America¹. While it is frequently located in the liver (75%) and lung, it is rarely seen in the spleen, pancreas, and intraperitoneal space². It is generally

asymptomatic unless it causes complications. Intra-biliary or intraperitoneal ruptures of the cyst are common complications and can cause serious consequences such as urticaria and anaphylaxis³.

Here, we present a case that settled in the lateral liver sector and completely covered the lobe, eliminating the lateral sector and causing thrombosis and cavernous collateral development by compressing the portal vein.

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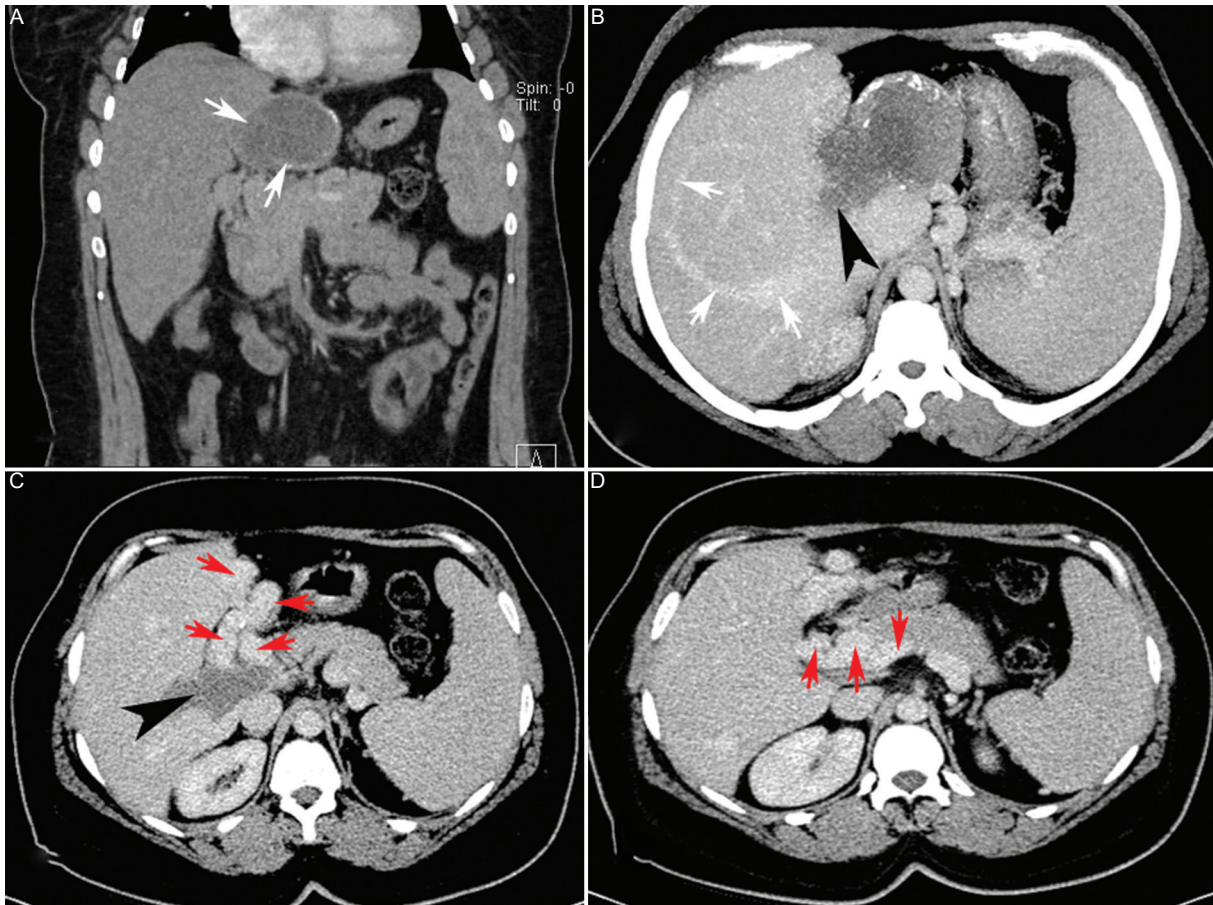


Figure 1. Top row: **A:** The coronal CT shows a giant hydatid cyst (Arrows) filling the lateral sector. **B:** the axial section shows intrahepatic venous-venous collaterals (Arrows) extending toward the right hepatic vein can be seen as a result of portal vein thrombosis due to the cyst extending to the hilus (Black arrowhead). Bottom row: **C:** The axial CT shows the part of the hydatid cyst extending (Black arrowhead) to the portal hilus and pressing the portal vein, and periportal venous collaterals with phlebolite calcification (red arrows) on its wall. **D:** this section shows that the splenic venous flow is poured into collaterals (red arrows) at portal confluence level. Venous collaterals inside the gallbladder wall.

Case

A 24-year-old female patient applied due to abdominal pain and swelling for the last 3 months. The patient, who did not have a history of alcohol use and medication, had not had abdominal surgery before. On physical examination, there was tenderness in the epigastric region. In laboratory examination, hydatid cyst serology was 1/320 positive, and biochemical parameters, coagulation factors, and liver enzymes were within normal limits. In abdominal ultrasonography and computed tomography, a high-density lesion, measuring 80 × 75 mm and extending to the hilus, filling the left lateral region of the liver, was consistent with the Gharbi-Stage 4 hydatid cyst (Fig. 1A and B). The cyst was compressing the portal vein. There were intrahepatic collaterals and shunts in the right lobe

due to portal vein thrombosis (Fig. 1B). There were dilated cavernous structures in the subhepatic area, extending from the falciform ligament to the hilus (Fig. 1C). The patient with portal vein thrombosis did not have cirrhosis and acid but had mild splenomegaly (Fig. 1D). There were also stones in the gallbladder. The patient was started on 2 months of albendazole and prepared for the operation. The patient with aberrant and tortuous veins in the perihilar area was operated on by pre-operative blood preparation. The 80×75 mm hydatid cyst wall filling the lateral liver sector and extending to the hilus was opened, and the daughter vesicles and germinative membrane were excised (pericystectomy). The cyst cavity was washed with a hypertonic NaCl solution. Afterward, the visceral peritoneum was opened to free the gallbladder. The hilar region and Calot triangle were attempted to

be dissected. It was observed that the area between the gallbladder and its surrounding visceral peritoneum was surrounded by large vascular structures that appeared as a result of cavernous transformation. There was bleeding in all areas at the time of dissection. Cholecystectomy was performed by carefully connecting the cavernous structures. In the histopathological examination of the specimen (Fig. 2), it was observed that the cystic-looking structures around the gallbladder were thick-walled vascular cavernous collateral areas. The patient was discharged on the 6th post-operative day. The patient was followed up on the 6th month without any problems.

Discussion

Hydatid cysts can apply pressure, disrupt the functions of organs, and present with very different clinical symptoms. Thrombosis and cavernomatosis are rarely seen as a result of compression of the hydatid cyst into the portal vein^{4,5}. According to the literature, we found six cases that developed hydatid cyst cavernous transformation.

During chronic obstruction occurring in the portal vein, physiological mechanisms are activated for the decompression of blood reaching the liver, and new collaterals are formed around the vein. This process can be within days or months in patients with venous thrombosis. In the case of our patient, it is understood that the blood coming to the liver is attempted to decompress through the collateral veins and shunts in the intrahepatic, subcapsular, pericholecystic, pericholedocal, and falciform ligaments, starting from the left portal vein. These collaterals, which serve as secondary pathways, expanded over time and turned into the cavernous structure. In our patient, it was thought that the hydatid cyst destroyed the left lateral sector; thrombosis developed as a result of the compression of the cyst extension over the segment five or eight to the portal vein; and due to the slow progression, it did not show any clinical signs with collaterals, especially intrahepatic and subcapsular. Cirrhosis, thrombophilic disorders, pancreatitis, and cholecystitis are involved in the etiology of portal vein thrombosis. Protein S, protein C, and antithrombin III values were also within normal limits in our patient. There were also no known as liver cirrhosis and previous intra-abdominal inflammatory events. In the literature, portal vein thrombus cases secondary to the liver cyst and very few cases with cavernoma have been reported^{5,6}. These occur due to compression of the cyst located in the hilus or obstruction following the invasion of parasites in the



Figure 2. Specimen shows gallbladder margin (Arrows) and pericholecystic cavernous formation (Stars).

cyst directly into the portal⁶. It often occurs in the form of periportal, pericholedocal, and pericystic cavernous. In this case, a giant hydatid cyst extending from the lateral sector to the hilus was shown as the cause of the transformation of cavernous secondary to portal vein thrombosis. The patient had stones in the gall bladder due to the compression of dilated venous structures at the hilus's level into the extrahepatic biliary tract, and the biliary stasis formed. It is a condition that requires surgical treatment due to serious and fatal complications such as hydatid cysts of the liver, spontaneous rupture, portal vein thrombosis, and anaphylaxis common in endemic regions³. Periportal cavernoma disrupts the Calot triangle anatomy and causes unexpected bleeding that causes mortality and morbidity during the case⁷. In our patient, the indication for surgical treatment was symptomatic gallstones and a hydatid cyst that atrophied the lateral sector. During the operation, a careful surgical technique was applied, taking into consideration both the giant cyst in the liver and its complications, as well as the perihilar and pericholecystic high-density venous collaterals.

Conclusion

It should be remembered that hydatid cysts may have different comorbidities depending on where they are

compressed, and there are many known complications. In cases where cholelithiasis and hydatid cysts coexist, the development of portal vein thrombosis and cavernous transformation should be kept in mind. Hilar dissection and surgical procedures involve difficulties in the presence of cavernous transformation.

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Conflicts of interest

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Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this study.

Confidentiality of data. The authors declare that they have followed the protocols of their work center on the publication of patient data.

Right to privacy and informed consent. The authors have obtained the written informed consent of the patients or subjects mentioned in the article. The corresponding author is in possession of this document.

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