



An Exceptional Case of Budd-Chiari Syndrome Secondary to a Hepatic Hydatid Cyst

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Abstract

Budd-Chiari syndrome (BCS) is an uncommon but serious disorder caused by hepatic venous outflow obstruction, leading to hepatomegaly, ascites, portal hypertension, and liver failure. Secondary forms of BCS due to extraluminal compression from space-occupying lesions such as tumors, abscesses, or hydatid cysts are particularly rare.

We report the case of a 56-year-old woman with no prior comorbidities who presented with abdominal pain, ascites, and dyspnea. Imaging and serology revealed a large hepatic hydatid cyst causing compression and collapse of the retrohepatic inferior vena cava (IVC) and hepatic veins, leading to secondary BCS. She received conservative management with albendazole and anticoagulation, but declined surgical cyst resection. Unfortunately, she experienced a sudden cardiorespiratory arrest three weeks after discharge, most likely due to complications of the cyst such as rupture with anaphylaxis or cardiac compression.

This case highlights the importance of considering hydatid cysts in the differential diagnosis of BCS, especially in endemic regions, and emphasizes early recognition and appropriate management to prevent fatal outcomes.

Keywords: Budd-Chiari Syndrome; Hepatic Venous Outflow Obstruction; Echinococcus granulosus; Hydatid Cyst; Inferior Vena Cava Compression

Introduction

Budd-Chiari syndrome (BCS) is defined as hepatic venous outflow obstruction, regardless of the underlying cause, location, or mechanism [1]. Its estimated prevalence is approximately 1 in 100,000 individuals, with a higher frequency reported in developing countries [2]. BCS is classified into primary (due to intraluminal thrombosis of hepatic veins or IVC) and secondary (resulting

from extrinsic compression or invasion by lesions such as tumors, abscesses, or cysts).

While myeloproliferative disorders represent the most common etiology in Western populations, parasitic infections remain an underrecognized cause in endemic areas [3]. Hepatic hydatid disease, caused by *Echinococcus granulosus*, is still prevalent in Mediter-

anean countries, the Middle East, South America, and Central Asia [4]. Humans act as accidental intermediate hosts by ingesting parasite eggs from contaminated food or water, leading to cyst development predominantly in the liver (50–70%) and lungs [5]. Although often asymptomatic, complications may arise from cyst enlargement, rupture, infection, or compression of adjacent structures.

Rarely, hydatid cysts may compress the retrohepatic IVC or hepatic veins, triggering secondary BCS [6]. Such cases are seldom reported but represent important diagnostic considerations in endemic areas. Here, we describe an unusual case of secondary BCS caused by a hepatic hydatid cyst compressing the IVC and hepatic veins, illustrating the diagnostic and therapeutic challenges associated with this condition.

Case Presentation

A 56-year-old woman, with no significant past medical history, presented with abdominal pain lasting 15 days, progressive abdominal distension, and dyspnea for six days. She also reported non-bilious vomiting three to four times per day for the past week.

On examination, she had bilateral pitting edema of the lower limbs and dilated collateral veins on the posterior abdominal wall. Abdominal inspection revealed distension, and palpation showed tenderness in the right hypochondrium along with hepatomegaly extending 8 cm below the right costal margin. Other systemic examinations were unremarkable. Vital signs were stable (BP 138/75 mmHg, HR 87 bpm, normal JVP).

Laboratory findings revealed anemia (Hb 9.6 g/dL), elevated liver enzymes (AST 311 U/L, ALT 254 U/L), and hypoalbuminemia (2.5 g/dL).

Imaging

Contrast-enhanced CT of the abdomen showed a large cystic hepatic lesion with daughter cysts and calcifications, consistent with a hydatid cyst. There was compression and collapse of the retrohepatic IVC and hepatic veins, moderate ascites, and mild bilateral pleural effusion (Figure 1).

Serology



Figure 1: Giant hepatic hydatid cyst with cardiac and inferior vena cava compression. Axial contrast-enhanced computed tomography (CT) scans demonstrate a large, well-defined hypodense cystic lesion (asterisk) in the right hepatic lobe. The cyst causes significant mass effect, with upward displacement and compression of the right cardiac chambers (yellow arrow) and compression with lack of opacification of the inferior vena cava (blue arrow).

Hydatid serology (IgG antibodies) was positive. Ascitic fluid analysis revealed a serum-ascites albumin gradient (SAAG) of 1.9 g/dL, with negative cytology and sterile cultures.

Management

The patient was started on albendazole, therapeutic paracentesis, and anticoagulation with subcutaneous heparin (5000 IU every six hours). Surgical excision of the cyst was strongly recommended; however, the patient declined hospitalization and surgery. She was discharged against medical advice.

Outcome

Three weeks later, the patient suffered an unexpected cardiorespiratory arrest and died. The cause was presumed to be either cyst rupture with anaphylactic shock or direct cardiac compression from the expanding cyst.

Discussion

BCS remains a rare but serious clinical entity. In Western countries, hematological disorders such as myeloproliferative syndromes are the leading cause [7], whereas in developing regions, infections, particularly parasitic diseases, represent important contributors [8].

In this case, secondary BCS was attributed to extrinsic compression of the IVC and hepatic veins by a hydatid cyst, a phenomenon rarely documented in the literature [9]. Hydatid disease, caused by *E. granulosus*, is endemic in sheep-rearing regions. Cysts usually remain asymptomatic until complications arise, such as rupture, infection, or compression of vital structures [10].

The pathophysiology of secondary BCS due to hydatid cysts involves mechanical obstruction of venous outflow, resulting in sinusoidal congestion, hepatomegaly, ascites, and progressive liver dysfunction [11]. In our patient, ascites, abdominal pain, hepatomegaly, and dyspnea reflected the hemodynamic consequences of venous obstruction.

Diagnosis relies on imaging and serology. Ultrasonography, CT, and MRI can demonstrate the cystic lesion, daughter cysts, calcifications, and vascular compression [12]. Positive serology for *Echinococcus* further supports the diagnosis.

Treatment of hepatic hydatid cysts includes:

- Medical therapy with albendazole to inhibit parasite metabolism.
- Percutaneous procedures such as PAIR (puncture, aspiration, injection of scolicidal agents, re-aspiration).
- Surgical resection, which remains the definitive management, especially in complicated cases [13].

In this case, although medical therapy was initiated, refusal of

surgery prevented definitive management. The sudden death likely resulted from rupture-induced anaphylaxis or compression of the IVC and right atrium, both recognized complications of large hydatid cysts [14].

This case emphasizes the need for early surgical intervention in complicated hepatic hydatid cysts and highlights the fatal risk of delayed or conservative-only management.

Conclusion

This case illustrates a rare but critical presentation of secondary Budd-Chiari syndrome due to a hepatic hydatid cyst. Clinicians should maintain a high index of suspicion for hydatid disease in patients presenting with unexplained hepatic venous outflow obstruction, especially in endemic regions. Early diagnosis and timely surgical intervention are vital to prevent catastrophic complications such as rupture, anaphylaxis, or sudden death. Multidisciplinary collaboration between radiologists, hepatologists, and surgeons is essential to optimize patient outcomes.

Disclosures

Human subjects: Consent was obtained or waived by all participants in this study.

Conflicts of Interest

The authors declare no conflicts of interest.

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Author Contributions

- **Manuscript design and drafting:** Ajertil Abderrazzak
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