

# Left trisectionectomy and supra-hepatic caval reconstruction with vascular prosthesis for chronic Budd-Chiari syndrome caused by hepatic alveolar echinococcosis

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*To the Editor:* Hepatic alveolar echinococcosis (AE) is a lethal infectious disease caused by the larval stage *Echinococcus multilocularis* (*E. multilocularis*) and exhibits low prevalence in endemic areas with high morbidity and mortality.<sup>[1]</sup> AE lesions invading the hepato-caval confluence, including main hepatic veins and retro-hepatic inferior vena cava (IVC), may result in Budd-Chiari Syndrome (BCS), which was a severe complication of AE presented with abdominal pain, ascites, and hepatomegaly.<sup>[2]</sup> In this study, we reported a case of chronic BCS caused by hepatic AE and summarized the therapeutic method of left trisectionectomy and supra-hepatic IVC replacement with prosthetic grafts.

A 57-year-old man from *E. multilocularis* endemic area was admitted to our department with the chief complaint of right upper quadrant pain and ascites. Pre-operative abdominal computed tomography (CT) demonstrated that 10 × 10 × 8 cm AE lesion, located in the left and right anterior liver lobe, had invaded the supra-hepatic IVC [Figure 1A], which was further confirmed through magnetic resonance imaging [Figure 1B]. Venous angiography showed that the cramped section of supra-hepatic IVC extended into the right atrium, and collateral circulation was already established [Figure 1C]. According to the World Health Organization Informal Working Group on Echinococcosis PNM (parasite, neighboring organ and tissue invasion, metastases) classification system, the staging of this patient was P4N0M0 and his liver function was Child grade B.

After a thorough assessment by multidisciplinary teamwork, a step-by-step resection strategy was imple-

mented. First, most AE lesion was removed through left trisectionectomy, and the lesion only invading IVC was remained. Second, we blocked IVC on both sides, and hemodynamics was still stable due to adequate collateral circulation. Then, the invaded IVC was resected, followed by reconstruction of IVC with a vascular prosthesis. Finally, the diaphragm was repaired with a patch, which was not completely closed to avoid pericardial effusion [Figure 1D–1F]. Post-operative pathological hematoxylin & eosin staining confirmed the diagnosis [Figure 1G and 1H]. The operation procedure lasted 8 h, among which vascular reconstruction took up 40 min, and the blood loss was about 500 mL without blood transfusion. The patient recovered well, and no vascular thrombosis or other major complications were observed during the 4-year follow-up period [Figure 1I and 1J].

BCS caused by hepatic AE was a rare clinical setting,<sup>[2]</sup> and hepato-caval reconstruction, transjugular intra-hepatic portosystemic shunt placing, and even liver transplantation have been used as treatment options.<sup>[3]</sup> In this study, we adopted the strategy of hepatectomy and artificial blood vessel replacement for the treatment of BCS.

Reconstruction of the supra-hepatic IVC is a great challenge for surgeons.<sup>[4]</sup> In this case, the whole supra-hepatic IVC was fibrotic and stenotic, and the defect area was too large to be repaired by autologous vessel graft. If allogeneic blood vessels were introduced, the application of lifelong immunosuppressants was inevitable, which may increase the risk of AE recurrence. Therefore, we consider that artificial vascular graft was the best option in spite of

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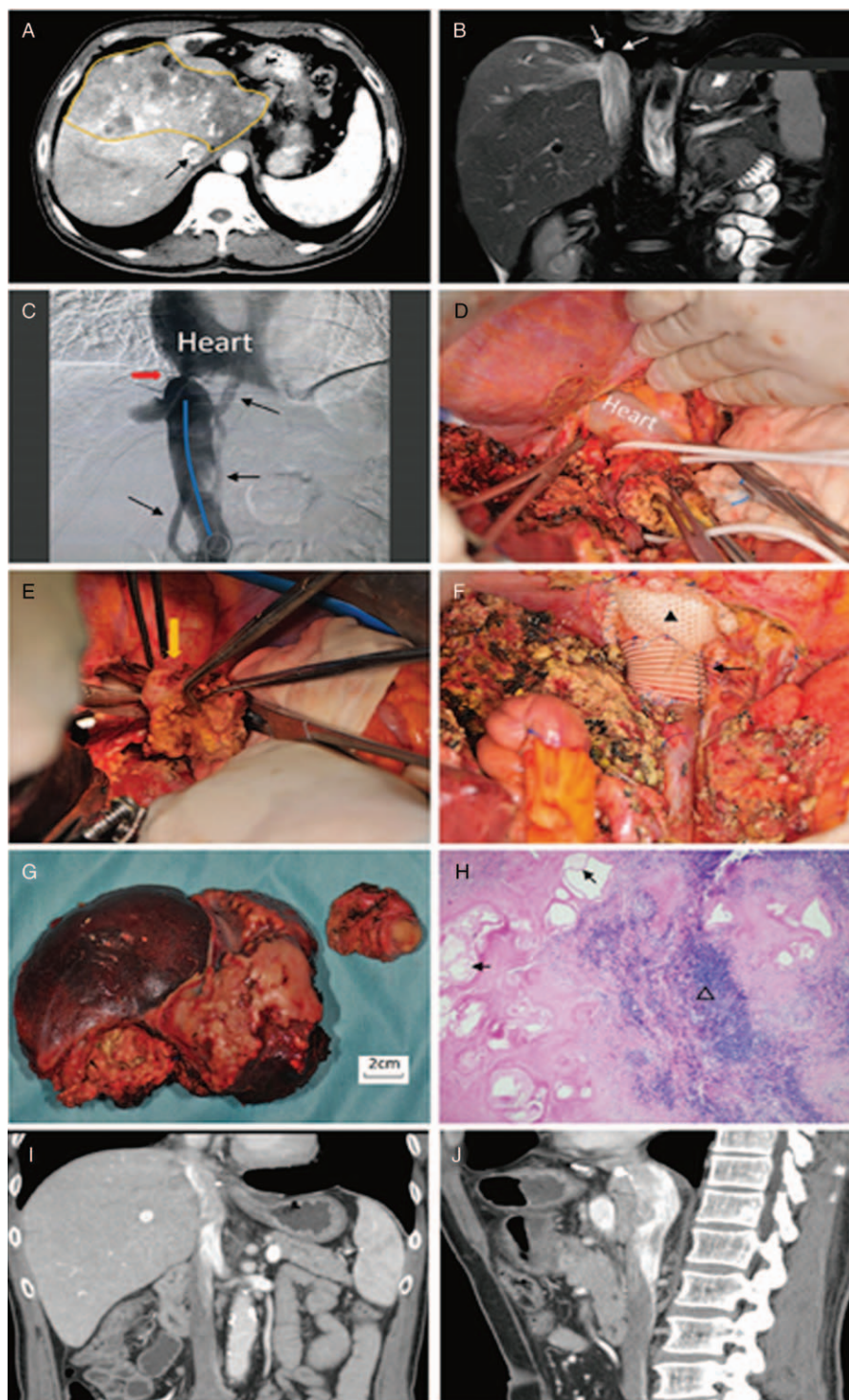
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**Figure 1:** Representative images and post-operative pathological results. (A) Abdominal CT showing hepatic AE lesions. (B) Abdominal MRI revealing round-shaped vascular thrombus at supra-hepatic level of IVC (arrows). (C) Venous angiography detecting blood block beneath the right atrium (red arrow), IVC (blue line) and collateral circulation (black arrows). (D–F) Intra-operative imaging demonstrating operation process, AE lesion in the IVC (yellow arrow), and vascular prosthesis used to replace supra-hepatic IVC (black arrow) and the sticking patch (triangle). (G) Specimens of AE lesions resected from the liver (bigger) and the supra-hepatic IVC cavity (smaller). (H) HE staining indicating peri-lesion lymphocyte infiltration (triangle) and laminated layers of the parasite (back arrows; original magnification  $\times 40$ ). (I and J) Post-operative CT showing IVC patency after the operation. AE: Alveolar echinococcosis; CT: Computed tomography; HE: Hematoxylin & eosin; IVC: Inferior vena cava; MRI: Magnetic resonance imaging.

possible concerns, including the formation of thrombosis or obstruction of the vascular graft. Luckily, post-operative monitoring showed no signs of AE recurrence, vascular obstruction, or thrombosis.

In conclusion, BCS caused by hepatic AE is an extremely rare clinical phenomenon, and radical resection with R0 margin may offer the best chance for AE patients with long-term disease-free survival. The experience might

provide treatment modalities for secondary BCS caused by hepatic AE.

### **Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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### **Conflict of interest**

None.

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