CASE REPORT

Pulmonary embolism caused by liver abscess with isolated venous thrombosis in an inferior right hepatic vein—a common and yet easily overlooked variant

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Introduction

We describe an unusual case of an isolated inferior right hepatic venous thrombosis caused by a liver abscess in the right lobe. Extension of the thrombosis along the inferior right hepatic vein into the retro-hepatic inferior vena cava (IVC) was noted. Further dislodgement with septic pulmonary emboli in the segmental branches of the right upper lobar pulmonary artery was observed.

Case report

A 33-year-old-woman with good past health presented to our hospital with a 2-week history of fever and 4-day history shortness of breath. There were no other chest symptoms, bowel or urinary symptoms. She had no recent travel history. There was no history of oral contraceptive intake. Apart from fever and mild tachycardia, physical examination was unremarkable. Laboratory findings on admission revealed deranged liver function with elevated parenchymal and ductal enzymes (alanine aminotransferase 284 U/l, aspartate aminotransferase 169 U/l, alkaline phosphatase 165 U/l, gamma-glutamyl transferase 109 U/l, and total bilirubin 27 μmol/l). She was found to have desaturation with an SaO2 of 95% on 2 l/min O2. Investigation of arterial blood gasesshowed type I respiratory failure (pH 7.36, elevated pO2 of 8.5 kPa, and decreased pCO2 of 3.5 kPa, decreased actual bicarbonate of 14.7 mmol/l, and base excess of −29 mmol/l). White cell count was normal. Platelet count was low (39 £ 109/l). The clotting profile was mildly deranged with a prolonged prothrombin time (14.2 s) and International Normalized Ratio (INR) (1.2).

Urgent ultrasound examination revealed two heterogeneous 3 cm hypoechoic masses in the right lobe of liver (Fig. 1), which were suspicious of liver abscesses. Doppler examination of the hepatic veins and IVC were not performed. Contrast-enhanced computed tomography (CT) examination of the abdomen was performed using multi-slice CT (General Electric Lightspeed, Milwaukee, WI, USA) on the same day to confirm the nature of the liver mass. This was performed with a slice thickness of 5 mm, pitch 3 and table speed of 11.25 mm/rotation. Images were acquired in the portal venous phase of enhancement. CT confirmed two heterogeneously enhancing liver abscesses close to each other in segment VII of the right lobe (Fig. 2). There was thrombosis of the inferior right hepatic vein (Fig. 3) adjacent to the liver abscess. The whole inferior right hepatic vein was distended and thrombosed. Extension of the thrombosis along this vein into the retro-hepatic IVC was noted. The corresponding draining segment, segment VI, showed patchy hypo-enhancing areas in the portovenous phase (Fig. 4). The right, middle and left hepatic veins were all patent (Fig. 5). On the following day, the patient had persistent hypoxia and type I respiratory failure, and there was clinical suspicion of pulmonary embolism. Contrast-enhanced multi-slice CT pulmonary angiography was thus performed with 2.5 mm slice thickness, pitch 6 and a table speed of 7.5 mm/rotation. Non-ionic contrast medium (120 ml Iopamiro 370 Bracco, Milan, Italy) was given at a rate of 4 ml/s by power injector (Nemoto, Japan). The timing of scanning was determined by SmartPrep software (General Electric Company) with the region of interest at the main pulmonary artery. Several saddle-shaped filling defects compatible with emboli were seen in the segmental lobar arteries in the right upper lobe (Fig. 6), confirming the presence of septic pulmonary emboli. Doppler study of the deep veins of both lower limbs showed no evidence of venous thrombosis. Aspiration of the liver abscess under real-time ultrasound guidance was performed and the culture yielded growth of Klebsiella pneumoniae.

The patient was put on intravenous cefuroxime (Zinacef) and oral metronidazole (Flagyl). Intrahepatic heparin anticoagulation was started, which was later changed to Enoxaparin with Warfarin. Her fever subsided and her blood gases and platelet count returned to normal. Her condition improved and she recovered uneventfully.

Discussion

An inferior right hepatic vein is not uncommon.1–3 It

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is the most common hepatic venous variant and is said to occur in 10–31% of normal subjects.\textsuperscript{1–3} Ultrasound demonstrated this venous variant in 10–18% of normal subjects.\textsuperscript{2,3} In a recent study of potential liver donors and recipients using multidetector CT,\textsuperscript{1} an inferior right hepatic vein was detected in 31% of the 107 patients. Most of them had a single inferior right hepatic vein and 12% ($n = 4$) had two. The prevalence of this venous variant was probably underestimated in the past. Thus it is important to recognize this type of hepatic venous variant in CT studies of the liver. To the best of our knowledge, this is the first reported case of isolated venous thrombosis involving the inferior right hepatic vein.

Figure 1 Ultrasound examination of the liver shows a hypoechoic mass lesion in the right hepatic lobe (arrowheads).

Figure 2 Contrast-enhanced CT confirms the presence of two liver abscesses (arrows) in the right hepatic lobe with a small thrombus within the retro-hepatic IVC (arrowhead).

Figure 3 Serial contiguous contrast-enhanced CT images showing the thrombosed inferior right hepatic vein (arrowheads).
Acute onset venous thrombus can be echo free on B scan ultrasound examination and can be unrecognizable if colour or power Doppler is not used. Contrast-enhanced multi-detector CT should be the imaging technique of choice for suspected cases of venous thrombosis of the inferior right hepatic vein. It clearly demonstrates the filling defect in the inferior right hepatic vein and further delineates any extension into the IVC. Moreover, the patchy heterogeneous hypoenhancing area in segment VI in the portovenous phase, as a result of venous congestion, could be clearly demonstrated.

CT pulmonary angiography could be performed in the same setting if there is clinical suspicion of septic emboli to the pulmonary arteries.

Cheng et al. reported the size of the inferior right hepatic vein in normal subjects to be 1-8 mm with an average of 4.6 mm. Three to 12.6% of the inferior right hepatic veins are larger in calibre than the right hepatic vein. In our patient, the size of the right hepatic vein was 7 mm and the thrombosed inferior right hepatic vein was 4 mm. The thrombosed inferior right hepatic vein was in close proximity to one of the liver abscesses, which was likely to be the nidus causing the inflammatory change and the venous thrombosis. The inferior right hepatic vein might be more vulnerable to thrombosis due to its small size as compared with the right hepatic vein or IVC. Despite its small size, venous thrombosis of the inferior right hepatic vein can cause pulmonary embolism when the thrombosis extends to the retro-hepatic IVC. A case of undetected pulmonary embolism in a patient with hepatocellular carcinoma, who presented with sudden death, has been reported. We postulate that pulmonary embolisms caused by a hepatic tumour or abscess could involve this venous structure and because of its small size could go undetected.

In conclusion, an inferior right hepatic vein is not an uncommon venous variant. Isolated thrombosis of an inferior right hepatic vein could cause pulmonary embolism in patients with a liver abscess or tumour. Thrombosis of this common venous
variant should not be overlooked. Contrast-enhanced multidetector CT should be the imaging technique of choice.

References


