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Outcomes of emergent embolisation of ruptured hepatocellular carcinoma in a western population

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AIM: To evaluate the authors' experience with interventional radiological management of tumour rupture in hepatocellular carcinoma (HCC) in a Western population.

MATERIALS AND METHODS: A retrospective review was performed of all consecutive patients treated at a single institution with transcatheter embolisation for ruptured HCC between 2000 and 2013. Patient age, sex, aetiology of liver disease, degree of underlying liver dysfunction, and clinical presentation were assessed. Embolisation was performed in a selective fashion when possible. Success, complications, and survival were assessed.

RESULTS: Twenty-three patients were treated with embolisation for ruptured HCC. Of these patients, nine, nine, and five patients were Child–Pugh Class A, B, and C respectively. Embolisation was successful in 22 patients; one patient remained haemodynamically unstable and transfusion dependent despite embolisation. No major complications occurred. Median survival time was 260 days and the 30 day and 1 year survival rates were 83% and 45%, respectively. Child–Pugh class B or C ($p = 0.04$), Model for End-Stage Liver Disease score greater than 10 ($p = 0.04$), lobar embolisation ($p = 0.04$), and presence of portal vein thrombosis ($p = 0.01$) were significantly associated with worse prognosis.

CONCLUSION: Transcatheter embolisation is effective at controlling haemorrhage in patients with ruptured HCC. Although major procedural complications are low, embolisation should proceed with an understanding of poor prognosis in patients with decompensated liver disease. Superselective embolisation is associated with improved prognosis and should be performed when feasible.

Introduction

Hepatocellular carcinoma (HCC) is the third leading cause of cancer-related deaths worldwide, resulting in approximately 600,000 deaths annually.[1] The incidence of HCC in Western countries continues to rise, likely due, in part, to hepatitis C infection and non-alcoholic steatohepatitis, and

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the annual incidence of HCC in the United States increased by approximately 80% over the last 20 years.[2–4]

Tumour rupture with associated haemorrhage is a catastrophic complication of HCC. Although its incidence is difficult to assess, it is the third leading cause of death overall behind liver failure and tumour progression in patients with HCC.[5] Tumour features associated with rupture include large size and peripheral location with protrusion beyond the liver margin (Fig 1).[6–9]

Emergent partial hepatectomy may provide definitive therapy in low surgical risk patients if complete resection is possible.[5,10–14] Unfortunately, most patients lack the functional reserve to tolerate surgery and the disease is surgically unresectable at the time of rupture. In such cases, management options include conservative treatment and transcatheter embolisation with or without staged hepatectomy. Transcatheter embolisation has been shown to be effective at achieving haemostasis in the acute setting[15–17] as well as stabilising patients for staged hepatectomy.[18,19] However, previous studies examining emergent catheter-based management of ruptured HCC focus on populations in which HCC is most commonly related to hepatitis B and the presentation of HCC more frequently precedes liver decompensation.[20,21] Patients with compensated liver disease free of portal hypertension are more often tolerant of surgical resection or aggressive transcatheter embolisation. The efficacy and safety of embolisation in the Western population is less well characterised and the benefits of treatment in cases of decompensated liver cirrhosis remain controversial.[22–24]

Materials and methods

A retrospective study was conducted at a single institution by querying the hospital's patient information system to identify all patients who underwent treatment with transcatheter embolisation for ruptured HCC. This was collected into a database in compliance with regulations of the Health Insurance Portability and Accountability Act and approved by the hospital's institutional review board.

Initial clinical presentation, blood counts, blood chemistry, coagulation profiles, and cross-sectional imaging were assessed. Unstable haemodynamic condition was defined as hypotension (systolic blood pressure <90 mmHg) and tachycardia (heart rate >100 beats per minute) at presentation. Child–Pugh and Model for End-Stage Liver Disease (MELD) scores were calculated using presenting laboratory values and clinical features.

The decision to perform transcatheter embolisation was made jointly by hepatology, hepatobiliary surgery, and interventional radiology. Antibiotic prophylaxis was provided, and the procedures were performed under moderate sedation or general anaesthesia. After diagnostic angiography of the superior mesenteric artery and coeliac trunk with a 4 or 5 F catheter, selective catheterisation of the hepatic arteries supplying the tumour was performed with a 2.8 F coaxial microcatheter (Progreat, Terumo Medical Corporation, Somerset, NJ or Renegade HI-FLO, Boston Scientific, Natick, MA, USA). Superselective arterial catheterisation (Fig 2) was performed if possible. If multiple feeding arteries to a tumour were present, embolisation was performed proximally in order to cover the entire perfused territory. Embolisation was performed with a variety of embolic agents. The performing interventional radiologist chose the type and amount of embolic agent at the time of the procedure. Intended procedural end-point was stasis or near-stasis of all tumour-feeding vessels and region of rupture. After delivery of the embolic agent, post-embolisation angiogram was performed. Success of embolisation was defined as cessation of angiographic extravasation, stabilisation of haematocrit without further need for transfusion, and haemodynamic stability. Procedural complications were recorded and overall survival was assessed for each patient.

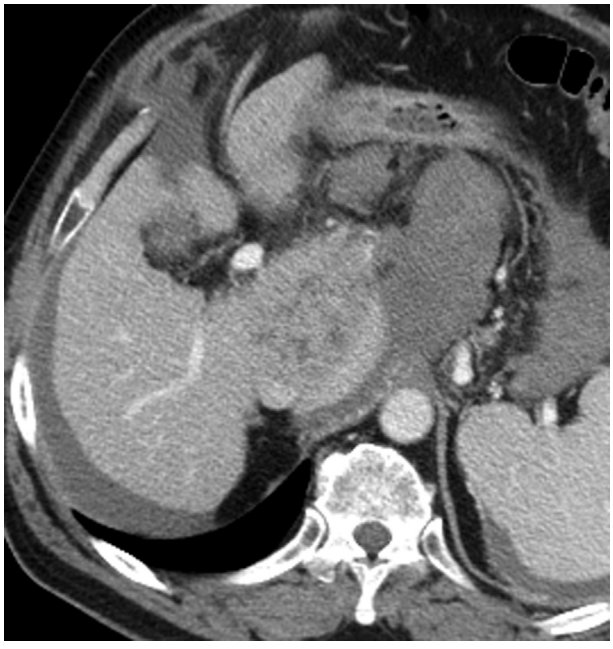

Figure 1 Contrast-enhanced axial CT shows a large ruptured subcapsular caudate lobe tumour with associated haemoperitoneum.

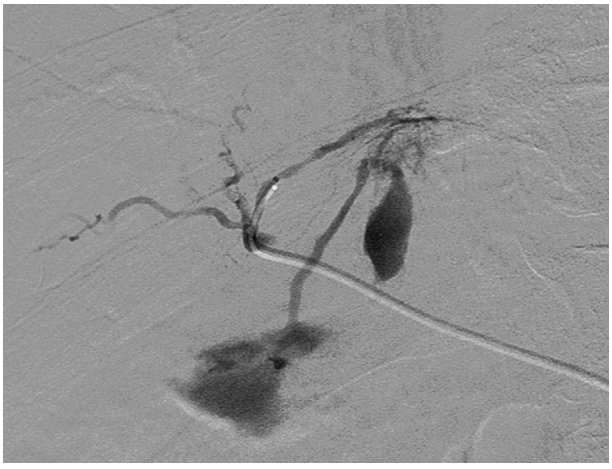

Figure 2 Superselective angiography of the ruptured caudate lobe tumor shown in Fig 1 shows gross active extravasation. Embolisation was performed from this location with gelfoam and coils.