
CASE REPORT

Splenic Metastasis of Hepatocellular Carcinoma with Spleen Rupture

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ABSTRACT

Splenic metastasis is uncommon, and splenic rupture secondary to metastasis of hepatocellular carcinoma (HCC) is even rarer. We report on a 37-year-old man who underwent an emergency splenectomy for splenic rupture secondary to metastasis of HCC. Differentiation between pathological and idiopathic atraumatic splenic rupture is important. In patients with splenic rupture without evidence of trauma, underlying splenic abnormality should be suspected. Splenic lesions may be obscured on initial imaging during an acute setting.

Key Words: Carcinoma, hepatocellular; Neoplasm metastasis; Splenic rupture

中文摘要

肝細胞癌脾臟轉移與脾破裂

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脾臟轉移癌不常見，肝細胞癌脾臟轉移導致脾破裂更為罕見。本文報告一名37歲男子接受緊急脾切除術治療由肝細胞癌轉移導致脾破裂。區別病理性與特發性無創脾破裂很重要。在脾破裂患者無創傷跡象的情況下，應懷疑潛在的脾臟異常。在緊急情況的最初成像，脾臟腫瘤可能被遮蔽。

INTRODUCTION

Despite its vascular nature, splenic metastasis is uncommon and usually occurs when there is already substantial systemic metastasis. Based primarily on autopsy series, the prevalence of splenic metastasis ranges from 0.3% to 7.3%.^{1,2} Nonetheless, the exact prevalence is uncertain due to different inclusion criteria and limited reported series. A local retrospective review

over a 25-year span yielded only 92 such cases, with 1.1% of splenectomy specimens containing metastatic tumour.² The low prevalence could be due to (1) the acute angle of the splenic artery, (2) lack of afferent lymphatic channels, and (3) anti-tumour activity of the rich lymphoid tissue. Splenic metastasis is usually asymptomatic, but its symptoms include splenomegaly, abdominal pain, systemic symptoms and, rarely, splenic rupture.³

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Submitted: 6 Jul 2016; Accepted: 18 Oct 2016.

Disclosure of Conflicts of Interest: All authors have disclosed no conflicts of interest.

CASE REPORT

In January 2015, a 37-year-old man presented to Pamela Youde Nethersole Eastern Hospital with dizziness and vague left-sided back/abdominal pain. He had a history of chronic hepatitis B infection, alcoholic liver cirrhosis, hepatocellular carcinoma (HCC), and portal vein thrombosis. In 2010, he had undergone open right hemi-hepatectomy elsewhere; detailed pre-operative staging, operative findings, and follow-up imaging were not available. He had received an abdominal massage 10 days prior to presentation. During initial assessment, he was alert but with low blood pressure (58/38 mm Hg) and a heart rate of 53 beats per second. There was tenderness over the left abdomen with no bruising.

Emergency contrast-enhanced computed tomography (CT) showed gross haemoperitoneum, collapsed inferior vena cava, and hyperenhancing bowels suggestive of a shock complex (Figure 1). The mildly enlarged spleen (14.8 cm) showed diffuse hypoenhancement with perisplenic sentinel clots and active contrast extravasation at the splenic hilum. There was also evidence of prior right hemi-hepatectomy with hypertrophy of the liver remnant thrombosed portal vein with adjacent cavernous transformation. The splenic vein was also thrombosed. No hypervascular HCC was identified in the liver parenchyma.

The patient was diagnosed with traumatic splenic

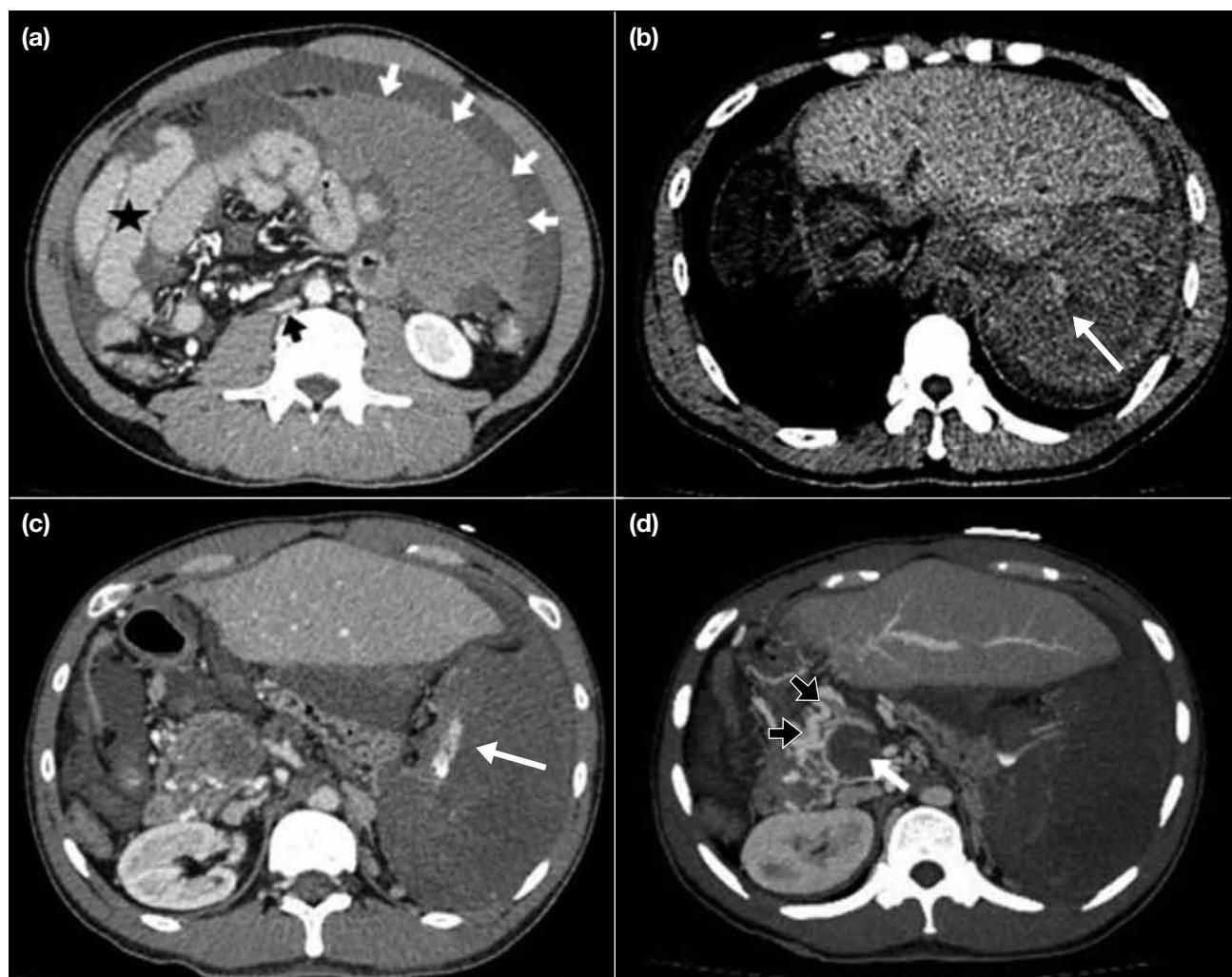


Figure 1. Contrast-enhanced computed tomography showing (a) gross haemoperitoneum (outlined by white arrows), collapsed inferior vena cava (black arrow), and hyperenhancing bowels (asterisk), suggestive of a shock complex, (b) peri-splenic sentinel clot (arrow), (c) diffuse hypoenhancement of the spleen and active contrast extravasation at the splenic hilum (arrow), and (d) thrombosed portal vein (white arrow) with cavernous transformation (black arrows).

rupture and treated with emergency splenectomy, although the clinical presentation was unusual: vague left-side back / abdominal pain without bruising, rib fracture, or other left-side visceral injury.

The specimen showed a laceration and metastatic HCC in the congested spleen measuring up to 4 x 6 x 12 cm. Venous invasion and thrombosis by the HCC near the splenic hilum was noted. Histology showed HCC with a sinusoidal pattern and marked nuclear pleomorphism and patchy positivity of tumour cells (Figure 2).

Two months later, he underwent dual-tracer positron emission tomography / CT elsewhere, and two mildly ¹¹C-acetate avid lesions in the segments IVa and II / III of liver and a hyper-metabolic lesion over the gastrohepatic space were detected, suspicious of peritoneal metastasis. He had a trial of sorafenib but

later stopped due to intolerance to the associated hand-foot syndrome. He was subsequently lost to follow-up.

DISCUSSION

Splenic metastases most commonly originate from breast, lung, gastric, colorectal, and ovarian primaries, and melanoma. The distribution of primaries appears to be associated with regional epidemiology. In Hong Kong, lung, stomach, colon, pancreas and oesophagus are the most common primaries causing splenic metastasis.²

Metastasis of HCC to the spleen is rare, accounting for only 1% of cases.⁴ Common HCC metastatic sites include lung, regional lymph nodes (peri-celiac and portahepatic nodes), and axial skeleton. The frequency of HCC metastasis correlates with the stage of the cancer. Risk factors include a large tumour load, either by size or multiplicity, and the presence of vascular invasion.

On contrast-enhanced CT, splenic metastasis typically shows an ill-defined, hypodense, hypoenhancing lesion, either solitary or multiple.⁵ Depending on the nature of the primary malignancy, hyper-enhancement could also be observed. In healthy patients, an incidentally discovered splenic lesion is usually benign such as haemangioma. Splenic metastasis should be suspected when the patient has a known primary malignancy with evidence of extensive visceral metastasis. Magnetic resonance imaging or positron emission tomography can be used to characterise an indeterminate splenic lesion. Splenic metastasis usually manifests as a T2 hyperintense and T1 hypointense or isointense lesion.^{6,7} The pattern of contrast enhancement on CT depends on the primary lesion, and presence of T1 hyperintensity suggests haemorrhage or secondary to melanin in cases of metastatic melanoma. On positron emission tomography, splenic metastasis appears hypermetabolic.

Atraumatic splenic rupture is uncommon.⁸ It is also known as spontaneous splenic rupture, idiopathic splenic rupture, and occult splenic rupture. It can be divided into pathological and idiopathic subtypes, with the proportions being 93% and 7%, respectively.⁸ Common causes for pathological atraumatic splenic rupture include underlying haematological conditions (e.g. leukaemia, non-Hodgkin lymphoma), active systemic infection (e.g. infectious mononucleosis, malaria), splenic artery / vein thrombosis (e.g. post-hepatectomy) and local inflammation (e.g. pancreatitis). The success rate of conservative management for

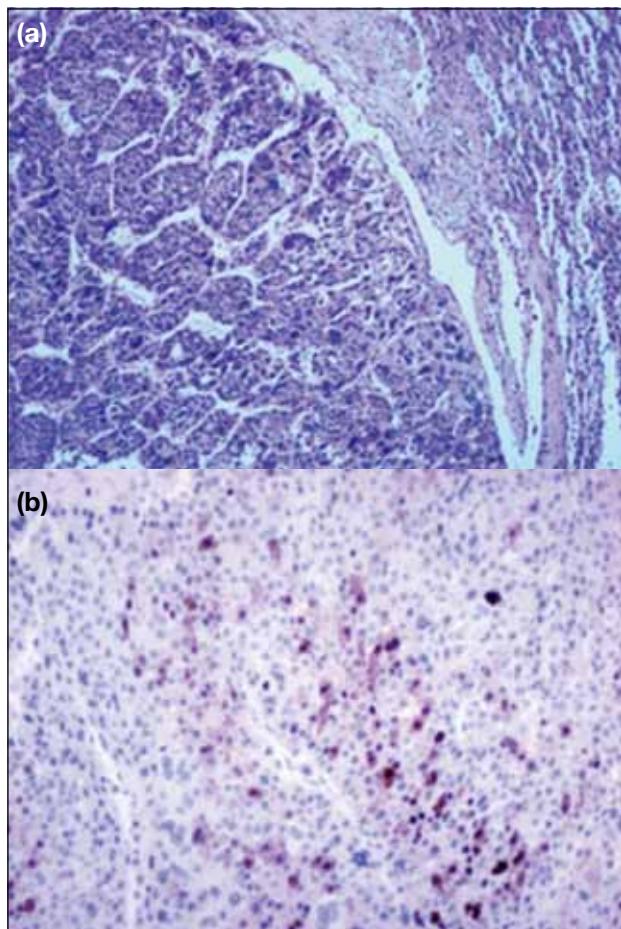


Figure 2. (a) Sinusoidal pattern and marked nuclear pleomorphism of hepatocellular carcinoma, with vascular red pulp tissue of spleen on the right side (H&E, x 100), and (b) patchy positivity of tumour cells (arginase-1, x 200).

pathological rupture (particularly caused by neoplasm) is lower than that for idiopathic rupture.⁸

Risk factors for splenic rupture include a congested and mildly enlarged spleen secondary to portal hypertension and splenic vein thrombosis (increased size and intra-splenic pressure), the hypervasculature nature of the metastatic HCC with necrosis (abnormal tissue architecture), and underlying vascular invasion near the splenic hilum (compromised capsular integrity). In our patient, the metastatic HCC was obscured on initial CT owing to generalised splenic infarct. The mechanisms of the splenic rupture include (1) initial injury by the abdominal massage, with propagation of the laceration by micro-trauma (coughing, sneezing) and (2) spontaneous, pathological atraumatic splenic rupture just prior to admission. Awareness of pathologic atraumatic splenic rupture is crucial, as underlying splenic lesions may be obscured owing to altered haemodynamics in a ruptured spleen.

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