Absent Uptake on Hepatobiliary Scintigraphy in Hepatic Lobar Infarction from Portal Vein Occlusion in Cholangiocarcinoma

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Hepatic infarction is an uncommon entity because of the dual blood supply to the liver. We report a case in which multimodalities demonstrate infarction of the left lobe of the liver secondary to left portal vein occlusion by an invasive cholangiocarcinoma. A ^{99m}Tc-DISIDA hepatobiliary scan showed complete absence of activity to the left of the gallbladder fossa. The differential diagnosis of absent hepatic activity on a hepatobiliary scan must include hepatic infarction.

Key Words: hepatic infarct; portal vein occlusion; cholangiocarcinoma; hepatobiliary scintigraphy

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Historically, hepatic infarcts were felt to occur solely secondary to hepatic arterial compromise (*1–4*). The dual blood supply to the liver makes hepatic infarction uncommon. We report a case in which multi-modalities demonstrate infarction of the left lobe of the liver, patent hepatic arterial supply and occlusion of the left portal vein secondary to an invasive cholangiocarcinoma. A ^{99m}Tc-di-isopropyl iminodiacetic acid (DISIDA) hepatobiliary scan demonstrated absent hepatic uptake to the left of the gallbladder fossa. Absent hepatic uptake secondary to hepatic infarct has not previously been described.

CASE REPORT

A 38-yr-old previously healthy male was admitted with a two day history of increasing right upper quadrant pain. Laboratory studies revealed an elevated total bilirubin, alkaline-phosphatase, SGOT and SGPT. An abdominal ultrasound performed to exclude cholecystitis demonstrated sludge in the gallbladder without gallstones or gallbladder wall thickening. Hypoechogenicity was noted diffusely throughout the left lobe of the liver. A ^{99m}Tc-DISIDA hepatobiliary scan revealed prompt hepatic uptake to the right of the gallbladder fossa, excretion of activity into the biliary system and filling of the gallbladder. At 70 min, hepatic uptake was persistently absent to the left of the gallbladder fossa and no intestinal activity was visualized (Fig. 1A). Intravenous contrast-enhanced abdominal CT performed two days later demonstrated diffuse low attenuation throughout the left lobe of the liver, irregular bands of enhancing tissue located in a central periportal distribution in the left lobe of the liver, low attenuation in the left portal vein consistent with occlusion and intrahepatic ductal dilatation within the left lobe of the liver (Fig. 2A and B). Celiac arteriography demonstrated patent hepatic arteries and neovascularity in the central periportal regions of the left and right hepatic lobes. CT arterioportography showed occlusion of the left portal vein and absent perfusion of the left, caudate and anterior segment of the right lobes of the liver (Fig. 2C).

On the basis of imaging findings, infarction of the left hepatic lobe secondary to a periportal neoplasm invading and occluding the left portal vein was suspected. Percutaneous biopsy of the periportal region of the left lobe of the liver yielded cholangiocarcinoma. The patient was discharged pending outpatient work-up

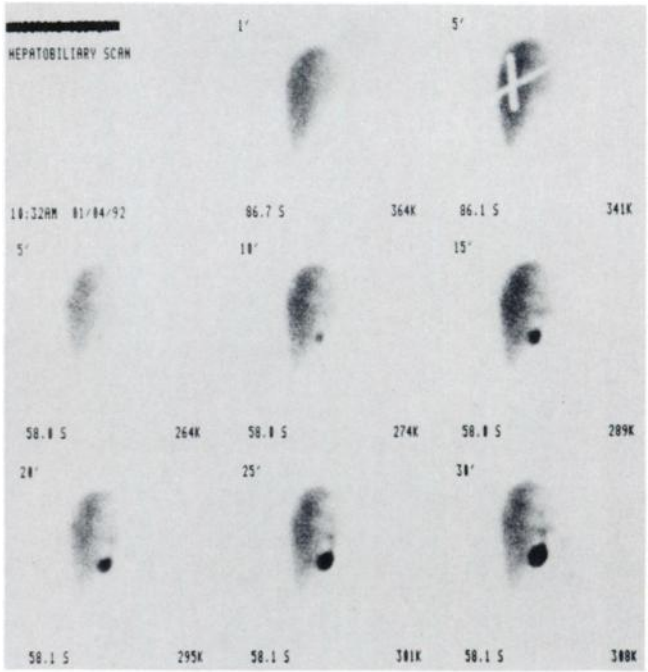

FIGURE 1. Technitium-99m-DISIDA hepatobiliary scan shows no hepatic activity to the left of the gallbladder fossa secondary to hepatic infarction. Bowel activity is absent secondary to biliary obstruction by cholangiocarcinoma.

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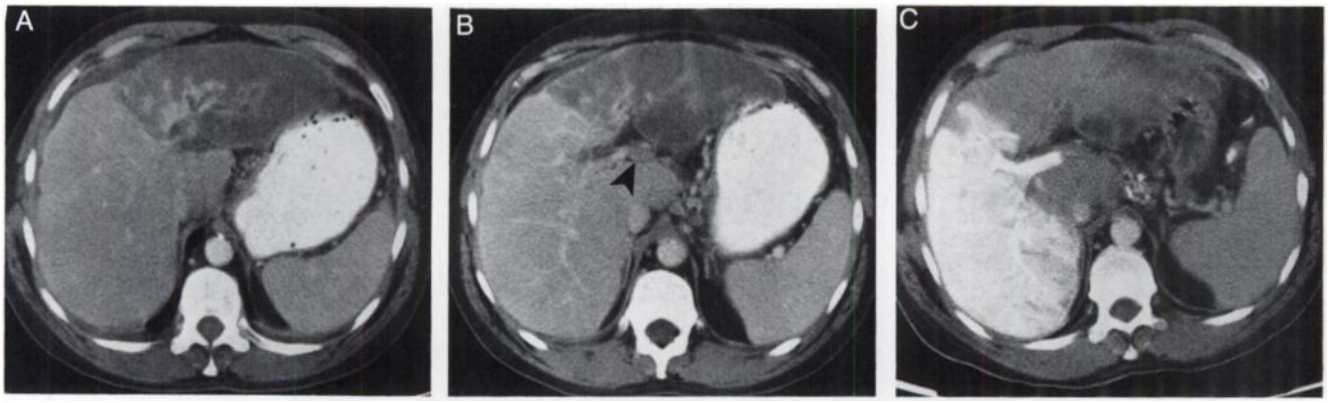

FIGURE 2. (A) Intravenous contrast-enhanced CT demonstrates hypodensity throughout the left lobe of the liver consistent with infarction. The enhancing periportal tissue proved to be cholangiocarcinoma at percutaneous biopsy. (B) Inferior CT image demonstrates the lack of enhancement of the left portal vein (arrow) consistent with portal vein occlusion. (C) CT arterial portography demonstrates left portal vein occlusion with lack of enhancement of the left lobe, caudate lobe and portions of the anterior segment of the right lobe of the liver.

for possible liver transplantation but returned in nine days with acute hepatic failure and expired within 24 hr of readmission.

DISCUSSION

It has been previously demonstrated that cholangiocarcinoma can occlude the portal veins by direct invasion or by extrinsic compression (*5–7*). Hepatic infarction, in conjunction with portal venous occlusion secondary to malignancy has been previously described at autopsy but has never been described from a radiographic or scintigraphic standpoint (*3,4*). In this case, the spectrum of findings involving multimodalities led to suspicion of the correct diagnosis and subsequent biopsy confirmation. Hepatic infarction was demonstrated as complete absence of activity on hepatobiliary scintigraphy within the left hepatic lobe. In addition to congenital malformations such as a hypoplastic or aplastic hepatic lobe, hepatic infarct should be included in the differential diagnosis of absent activity on hepatobiliary scintigraphy. Primary or secondary neoplasm would be unlikely to be demonstrated as complete absence of activity in a lobar distribution.

CT and sonographic appearance of the hepatic infarction secondary to hepatic arterial occlusion has been well described (*1–3*). Initially, hepatic infarcts were described as well-defined low attenuation areas or hypoechoic regions on CT and ultrasound, respectively. They were noted to be wedge-shaped and peripheral, analogous to the CT appearance of renal and splenic infarcts (*1*). Recent studies have found the appearance of the hepatic infarcts on CT and ultrasound far more variable and nonspecific (*2,5*). This case demonstrates typical features of hepatic infarct on CT and ultrasound. On CT, the infarction was well-defined, lobar in distribution and low in attenuation. On ultrasound, the lesion was hypoechoic. In addition, CT also demonstrated asymmetric intrahepatic biliary ductal dilatation in the affected left lobe and an enhancing periportal mass, which raised the correct suspicion of an underlying neoplasm as the cause of infarction. It is interesting that the region of hepatic infarction demonstrated on contrast-enhanced CT was much smaller than the regions of absent portal blood flow demonstrated by CT arterial portography.

Although it is unclear why hepatic infarction should result from portal vein occlusion in the presence of patent hepatic blood flow, this case clearly shows that hepatic infarction not only can occur under these circumstances but can be significant clinically, scintographically and radiographically. In the appropriate setting, differential diagnosis of absent hepatic uptake on hepatobiliary scintigraphy must include hepatic infarction.

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