Hemobilia Associated with Hepatic Artery Aneurysms: Scintigraphic Detection with Technetium-99m-Labeled Red Blood Cells

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Biliary tract bleeding is an unusual cause of upper gastrointestinal hemorrhage. Rupture of hepatic artery aneurysms is one of the least common etiologies of hemobilia. Both cholescintigraphy and \(^{99m}\text{Tc}\)-labeled blood cell scintigraphy are useful in the diagnosis of this rare disorder. The combination of focal obstruction on cholescintigraphy and intermittent visualization of the major bile ducts on red blood cell scintigraphy should suggest the diagnosis of hemobilia and prompt an angiogram to determine the cause.


In his classic 1973 treatise, Sandblom attributed the first description of biliary tract hemorrhage to Francis Glisson in 1654, and attached the term “hemobilia” to this group of disorders in 1948 (1). Less than 10% of the 545 cases reviewed by Sandblom were of vascular origin (1). Only 23 of the 80 cases of hemobilia caused by rupture of hepatic artery aneurysms reported by Harflatis and Akin in 1977, were diagnosed pre-operatively. Twenty-nine were diagnosed postmortem, attesting to the high mortality of this disease and the difficulty of its diagnosis (2). In general, the treatment of hemobilia is surgical (3); however, increasing interest is being given to the embolization of some cases of hemobilia through angiographic techniques (4,5). Nuclear medicine techniques are becoming the accepted first line of radiologic/scintigraphic diagnosis in cases of gastrointestinal (GI) bleeding in which the source is not revealed by clinical means (6–10,21). This paper reports the findings on cholescintigraphy and technetium-99m-\(^{99m}\text{Tc}\) labeled red blood cell scintigraphy in the diagnosis of a case of hemobilia, and on angiographic embolization as successful treatment of the disorder.

CASE REPORT

A 32-yr-old man was admitted for the evaluation of epigastric pain, and blood was discovered in his stool. An upper GI series and endoscopy failed to reveal any source of upper GI bleeding. Colonoscopy was negative for lower intestinal bleeding. An oral cholecystogram failed to visualize the gallbladder after two successive doses, but real-time ultrasound demonstrated a normal appearing gallbladder and bile ducts. A biliary scan using 5 mCi (185 MBq) of \(^{99m}\text{Tc}\)-labeled disofenin revealed excellent uptake of the tracer by the liver and prompt passage of the agent into the bowel. However, the gallbladder failed to visualize on 4- and 24-hr delayed images. In addition, two focal areas of increased tracer accumulation within the right and left lobes were seen at 4 hr (Fig. 1). The prolonged retention of the radiopharmaceutical within the liver conformed to the expected anatomic outline of the right and left lobes, as determined by a prior radiocolloid liver scan. On serial imaging, the abnormal areas failed to migrate or conform to the body wall. The patient's total bilirubin fluctuated between 0.6–2.0 mg/dl, with the upper limits of normal 1.0 mg/dl. In view of the patient's worsening abdominal pain, and the positive cholescintigram, an exploratory laparotomy was performed. At operation, no gallstones were found, but the gallbladder was filled with clotted blood, as were the cystic and common bile ducts. A routine cholecystectomy was performed. After discharge, the patient continued to suffer intermittent bouts of right upper quadrant pain associated with melena.

The continued intestinal blood loss necessitated the transfusion of a total of six units of packed red blood cells over a
period of several weeks to maintain a hematocrit of 30%. A sulfur colloid bleeding study was performed during a period of suspected active bleeding using 10 mCi (370 MBq) of $^{99m}$Tc-labeled sulfur colloid and imaging according to the technique described by Alavi and Ring (10). This study was negative. On labeled red blood cell study using 25 mCi (925 MBq) of $[^{99m}$Tc]red blood cells labeled by the modified in vivo technique described by McKusick et al. (11) an obliquely oriented collection of activity appeared in the region of the liver at 1 hr, conforming to the expected location of the major bile ducts (Fig. 2). This activity was visible on two successive 5-min images and was not visible for the remaining 30 min of the study. On 4-hr delayed imaging, an extensive collection of activity was seen within both the small and large bowel (Fig. 3). An angiogram revealed multiple hepatic artery aneurysms (Fig. 4) as the source of the patient’s GI bleeding and hemobilia. Angiographic embolization was performed and the patient’s right upper quadrant pain and GI bleeding resolved. A follow-up biliary scan was normal, except for the absence of the gallbladder, without persistence of the tracer in the hepatic parenchyma. A repeat angiogram postembolization showed successful occlusion of the aneurysms. The patient had no recurrence of GI bleeding during 18 mo of follow-up.

**DISCUSSION**

Hemobilia is one of the less common etiologies of GI bleeding, and often remains undiagnosed. Sandblom has referred to the problem as the “neglected syndrome,” meaning the frequency of misinterpretation of the signs and symptoms, with associated delay in diagnosis and inappropriate treatment (1). Patients with hemobilia classically present with right upper quadrant pain, GI bleeding, and jaundice. The pain, jaundice, and bleeding are most often intermittent (12). The reported mortality, despite improvement in diagnosis and treatment, remains in the range of 10–20% (5). Trauma is the most common etiology for hemobilia, accounting for almost 50% of the cases in one series (7). “Trauma” includes surgical and other iatrogenic causes, as well as automobile accidents. Other etiologies
include parasites (mostly in the Orient), erosion by gallstone or tumor, and vascular disorders. The most common etiology for hemobilia secondary to a vascular cause is rupture of hepatic artery aneurysms (2).

Jaundice in hemobilia is caused by the presence of blood clots in the biliary tree, obstructing the normal passage of bile into the gut. Cholescintigraphy has been used with great success in the workup of right upper quadrant pain (with or without jaundice) in patients suspected of having cholecystitis (13), and has been reported to be useful in the evaluation of blunt trauma to the biliary tree (14,20,22). Prolonged diffuse retention of a hepatobiliary imaging agent within the hepatic parenchyma has been reported in a variety of disorders (15). There have been few mentions, however, in the literature concerning the appearance of focal intrahepatic obstruction on cholescintigraphy (16,17). Hemobilia has been reported as a cause for acute nonvisualization of the gallbladder on cholescintigraphy (18). This is the first case to our knowledge of hemobilia not only producing nonvisualization of the gallbladder, but also resulting in the focal obstruction of the intrahepatic biliary tree with delay in clearance of the tracer from portions of the liver. Bile leakage may mimic the intrahepatic retention of tracer seen in this patient (20). However, the failure of the activity to change in configuration with time, and the close conformation of the activity to the expected normal liver anatomy mitigated against this diagnosis.

Technetium-99m labeled red blood cells, in determining the site of GI bleeding (9), has the advantage over other scintigraphic methods that it may detect intermittent bleeding. In our patient, the [99mTc]sulfur colloid scan was negative, presumably because the patient was not bleeding at the time of injection. Progressive accumulation of the radiocolloid in the liver also makes this method unsatisfactory for the precise identification of the site of intrahepatic bleeding. There have been few reported cases of successful scintigraphic detection of hemobilia (7,19,23,24), none due to hepatic artery aneurysm rupture. While the aorta and inferior vena cava are easily visible on blood-pool scintigraphy, the portal vein is usually indistinguishable from the vascular background of the liver. The crucial diagnostic observation in our patient was the transient visualization of labeled red cells in the bile ducts, with subsequent visualization of tracer in the bowel. The aneurysms were too small to be seen.

**SUMMARY**

Hemobilia should be suspected as source of bleeding in any patient with concomitant right upper quadrant pain and jaundice. Because clinical evaluation may not point to the biliary tree as the source of blood loss, red blood cell scintigraphy may provide the first clue as to the site of bleeding.

Hemobilia as a result of ruptured hepatic artery aneurysms can be added to the list of entities diagnosable through 99mTc-labeled red blood cell scanning.

**REFERENCES**