Hemobilia as a Result of Coagulopathy

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Citation

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Abstract

Hemobilia, upper gastrointestinal tract bleeding originating from within the biliary tract, has become a widely understood and more commonly reported disorder. The first description of hemobilia was in 1654 and is credited to Francis Glisson. (1) The classic triad of hemobilia consisting of right upper quadrant (RUQ) abdominal pain, jaundice, and upper gastrointestinal tract hemorrhage was described by Quincke in 1871. (2) However, the term “hemobilia” was not coined until 1948 when Sandblom published a paper entitled “Hemorrhage into the Biliary Tract Following Trauma: Traumatic Hemobilia.” (3) With advancing medical expertise it has become evident that the classic triad of hemobilia only occurs in about 22% of cases. (4) Most cases occur as a result of accidental or iatrogenic blunt or penetrating trauma. It is also described in patient with cholelithiasis, acalculous inflammatory hepatobilary disease, vascular disorders, and neoplasms. (5, 6, 7) We present a case of hemodialysis arteriovenous fistula bleeding complicated by cholangitis and hemobilia.

CASE REPORT

A 79-year-old African American male was admitted to the hospital with right upper quadrant abdominal pain and nausea. He denied fever, chills, bright red rectal bleeding, or melena. His past medical history was significant for end-stage renal disease requiring hemodialysis, diabetes mellitus, peripheral vascular disease, coronary artery disease, Wolff-Parkinson-White syndrome, St. Jude mechanical aortic valve replacement requiring warfarin, and recent methicillin-resistant staphylococcus aureus bacteremia.

Physical examination showed a well nourished male in no distress but with sinus tachycardia, persistent hypotension, icterus, murmur produced by the mechanical valve, and diffuse abdominal tenderness to palpation with voluntary guarding in the right upper quadrant. Dry blood was also present around the hemodialysis arteriovenous fistula but no acute bleeding was observed.

Laboratory evaluation was significant for AST 218 unit/L, ALT 177 unit/L, alkaline phosphatase 576 unit/L, total bilirubin 5.3 mg/dL, conjugated bilirubin 5.0 mg/dL, albumin 2.1 gm/dL, INR 16.9, WBC 31,300/mcL, Hgb 10.0 gm/dL, MCV 90.3 fL, and RDW 17.5 %. Escherichia Coli was cultured from the blood. Computed tomography of the abdomen and pelvis revealed gallbladder distension, gallbladder wall edema, intraluminal sludge, cholelithiasis, choledocholithiasis, and common bile duct(CBD) dilatation with a diameter of 14-15 mm. Intravenous broad spectrum antibiotic therapy was commenced and he was given vitamin K and transfused with fresh frozen plasma. On day 2 of his admission the patient developed melena and the hemoglobin decreased to 5.4 gm/dL requiring blood transfusion.

The clinical picture of leukocytosis, right upper quadrant abdominal pain, jaundice, the computed tomography findings, and blood cultures consistent with gastrointestinal flora were consistent with a diagnosis of cholangitis for which an endoscopic retrograde cholangiopancreatography (ERCP) was planned. With the new onset of melena, severe anemia, and hemodynamic instability the procedure was delayed; however, the patient required immediate treatment of cholangitis.

Instead, the patient underwent percutaneous transhepatic cholangiography with placement of a biliary drain. The cholangiogram showed filling defects within the gallbladder and cystic ducts, morphologically consistent with blood clots. The drain returned bloody fluid. (Figure 1) With evidence of blood clots in the gallbladder lumen and cystic ducts, it was suspected that the CBD obstruction could have resulted from a blood clot rather than a gall stone. At this point hemobilia was also entertained as the cause of melena and ERCP was believed to be the ultimate diagnostic modality.

After resuscitation, an ERCP was performed to evaluate the

cause of melena and to look for a possible obstructing common bile duct stone resulting in the above symptoms; however, at ERCP blood was seen emitting from the Ampulla of Vater confirming the diagnosis of hemobilia was observed, and the procedure was terminated. (Figure 2)

Over the following 48 hours bleeding resolved with stabilization of hemoglobin. The biliary drainage tube was flushed and drained multiple times with saline. Repeat cholangiogram showed improvement in the appearance of the gallbladder which contained a residual single, small thrombus. The common bile duct was patent and there was no evidence of ductal thrombus or stones. His overall status improved during the following days and he was able to tolerate a regular diet prior to his discharge from hospital.

Figure 1

Figure 1 (initial cholangiography during placement of percutaneous biliary drain showing dense, inhomogeneous tissue with multiple filling defects which was consistent with blood and blood clots)

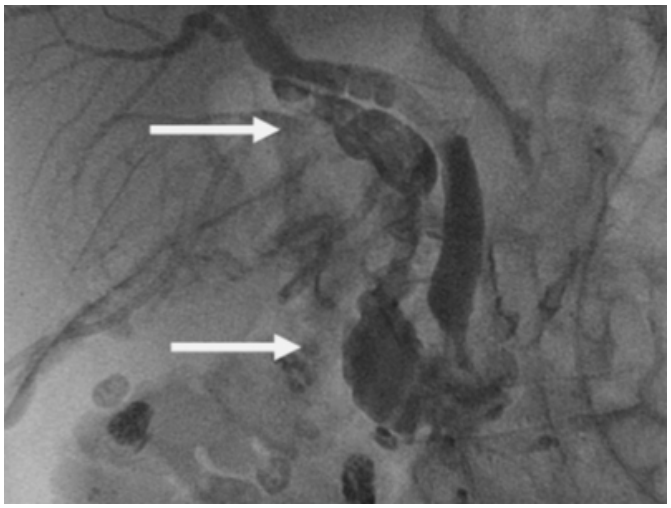
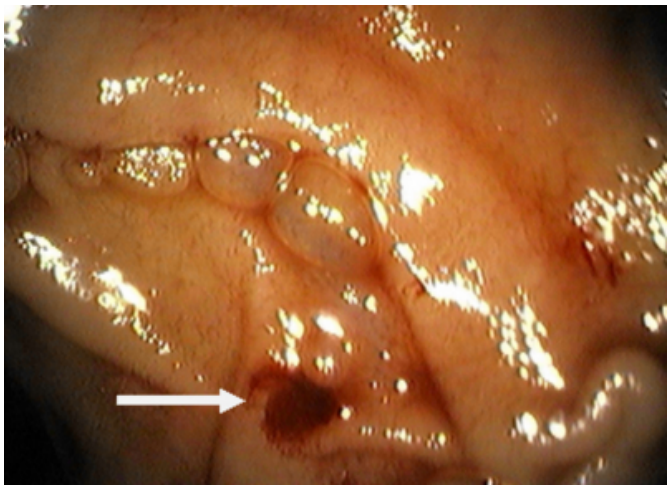


Figure 2

Figure 2 (Blood at Ampulla of Vater)



The patient had a complicated hospital course. He was originally thought to have cholangitis as a result of choledocholithiasis. After he was evaluated for the additional complication of gastrointestinal bleeding a diagnosis of hemobilia was made when blood was noted to be oozing from the Ampulla of Vater at ERCP. It then became apparent that the obstruction within the common bile duct was a result of a blood clot rather than a stone, and this resolved with cholecystostomy placement, flushing, and draining.

DISCUSSION

After extensive literature review and evaluations by various gastroenterologists, this case drew a large amount of interest as hemobilia appeared to have been a result of a coagulopathy from warfarin therapy. Previous reports of hemobilia have been accounted for by trauma, cholelithiasis, acalculous inflammatory disease, vascular disorders, or neoplasms. By history and our medical work-up the patient did not have any of these etiologies. We believe the patient's coagulopathy, secondary to warfarin therapy, was the cause of the hemobilia, which lead to the ensuing findings.

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