Hepatic artery aneurysm: a rare case of obstructive jaundice with severe hemobilia

George Peter, Rooby Shaheer, Premalatha Narayanan, Kattoor Ramakrishnan Vinayakumar
Government Medical College, Thiruvananthapuram, Kerala, India

Hepatic artery aneurysms (HAA) account for nearly one fifth of all visceral artery aneurysms [1]. The incidence of hepatic artery aneurysm has been on the rise due to the increasing numbers of imaging studies and hepatobiliary procedures being performed. The classical presentation of Quincke's triad, comprising abdominal pain, obstructive jaundice and hemobilia, has been reported in only one third of the cases [2]. While the vast majority of cases remain asymptomatic, those which present clinically are the ones which rupture, which have an estimated mortality of 40% [3].

A 73-year-old lady, with no significant comorbidities, presented with complaints of right upper quadrant dull aching abdominal pain and progressively increasing jaundice associated with pruritus of 3 months duration, with an episode of melena 2 weeks back. On evaluation she had deep icterus, hepatomegaly and hepatic bruit. Her liver function tests showed total bilirubin / direct bilirubin: 22.9/17.3 mg%; aspartate aminotransferase / alanine aminotransferase: 386/380 U/L; and alkaline phosphatase: 821 IU/L. Her hemogram, renal function tests, urine routine and electrolytes were within normal limits. Viral markers for hepatotropic viruses were negative. Contrast-enhanced computerized tomography of the abdomen with angiogram demonstrated aneurysmal dilatation of common hepatic artery (Fig. 1). The proximal fusiform dilatation measured 12.7x52.6x13.3 mm. The distal saccular aneurysm measured 57.4x53.6x50.3 mm with a rind of thrombus within. There was marked narrowing of the common bile duct at this region with moderate bilateral intrahepatic biliary radicle dilatation.

On the second day of admission, she developed high-grade fever with chills and rigors with neutrophilic leukocytosis. A side viewing endoscopy was performed which revealed blood spurting from the ampulla of Vater with multiple blood clots. Endoscopic retrograde cholangiogram with biliary stenting was deferred in view of active hemobilia and the patient was planned for immediate surgical repair. On the same day she developed massive hematemesis and succumbed to the illness.

Notwithstanding the rarity, HAA is reported to be the second commonest visceral artery aneurysm second to splenic artery aneurysm. In the past, mycotic aneurysms accounted for most HAAs but atherosclerotic aneurysms have emerged as the most common cause in the present era. Less frequent causes are polyarteritis nodosa, tuberculosis, Marfan syndrome and diagnostic instrumentation [4,5].

No definite risk factors have been established which predict the tendency for HAA rupture, but non-atherosclerotic origin and multiple lesions have been cited as potential risk factors [3]. Once HAA ruptures, the patient manifests hemobilia, which may range from mild to severe [6]. The therapeutic options available include coil embolization of the aneurysm, stenting across the main trunk and embolization of the common hepatic artery or open surgical repair with reconstruction [7,8].

In conclusion, HAAs are an extremely rare cause of obstructive jaundice and hemobilia. Symptomatic HAAs are associated with a poor prognosis if the diagnosis is delayed. We also believe that HAAs which present with an index bleed, as in our case, warrants urgent management strategies, as they rupture in no time, resulting in fatal hemobilia.

References


Department of Medical Gastroenterology, Government Medical College, Thiruvananthapuram, Kerala, India

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Correspondence to: Dr George Peter, Senior Resident, Department of Medical Gastroenterology, Super specialty block - 3rd floor (SSB - 3), Government Medical College, Thiruvananthapuram 695 011, India, Tel.: +91 85 479 67352, e-mail: georgepeter23@gmail.com

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