Disseminated Tuberculosis Presenting as Hemobilia, Successfully Treated by Arterial Embolization

D Das*, SK Mandal*, D Majumder*, BK De**

Abstract
Tuberculosis, specially disseminated tuberculosis, involves the liver frequently. Focal hepatic tuberculosis with local hemorrhage has been reported. We report on a twenty-one year female with disseminated tuberculosis presenting with initially non-localisable massive upper gastrointestinal bleeding, subsequently found to have pancreatitis, right sided pleural effusion and hemobilia which was treated successfully.

INTRODUCTION
We report a case of disseminated tuberculosis presenting with initially non-localisable massive upper gastrointestinal bleeding following upper abdominal pain, subsequently found to have pancreatitis, right sided pleural effusion, hemobilia due to leaking hepatic artery aneurysm and also generalised tonic clonic seizure during antitubercular therapy, with ring enhancing lesion in brain on CT scan.

CASE REPORT
SC, a 21 years old medical student, presented with pre-admission history of low-grade fever for ten days, for which she was taking nimesulide and amoxycillin. On eighth day of fever she had a bout of severe pain in abdomen and coffee ground vomiting. Nimesulide was stopped and omeprazole was started. On tenth day she again had a bout of severe upper abdominal pain radiating diffusely and to the back. Subsequently she had hematemesis and malaena. Upper gastrointestinal endoscopy showed presence of superficial gastric erosions with no active bleeding visible. Other reports available were hemoglobin 8.6 gm/dl, total bilirubin 2.6 mg/dl, plasma albumin 2.9 gm/dl, plasma globulin 4.8 gm/dl, AST 314 IU/L, ALT 314 IU/L, alkaline phosphatase 354 IU/L, serum amylase 6377 IU/L, serum lipase 2660 IU/L, LDH 777 IU/L, PT and APTT within normal values; serology for hepatitis B, A and C were negative as well as a negative ANF test. Pleural fluid study revealed a total cell count of 453 cell/cumm, with 75% mononuclear cells, LDH 1178 IU/L and on Ziehl-Neelsen staining of centrifuged deposit acid fast bacilli were visualised. Post-aspiration chest radiology did not reveal any appreciable parenchymal lung lesion or hilar lesions. Abdominal sonology showed a bulky pancreas with no evidence of cysts or focal edema, distended gall bladder with viscous sludge and enlarged liver with altered echotexture, and mild pleural effusion only on right side.

On third day after admission she suddenly developed severe upper abdominal pain, followed by severe sweating and pallor. She passed a large volume of malaena stool and her blood pressure fell to 90/50 mm of Hg (hemoglobin level fell to 5.6 gm/dl). She was resuscitated and repeat endoscopic evaluation after stabilisation did not show any active source of bleeding but there was old blood clot in second part of duodenum. Contrast CT scan of the abdomen revealed focal dilatation of intra-hepatic bile ducts in the upper part of the right lobe of liver with contrast enhancement adjoining the dilated ducts. There was focal calcified spots scattered in the liver. The pancreas was also bulky without signs of necrosis or focal edema.

She again suffered a bout of severe pain in abdomen and malaena next morning and a repeat endoscopy just after the episode of pain finally revealed bleeding through the ampulla of Vater. She underwent selective coeliac arteriography, which showed a leaking aneurysm of the pleural effusion. No other significant findings were detected. The patient was started on standard regimen for pancreatitis and two units of blood were transfused. Investigations showed hemoglobin 10.2 gm/dl, total bilirubin 2.6 mg/dl, plasma albumin 2.9 gm/dl, plasma globulin 4.8 gm/dl, AST 314 IU/L, ALT 314 IU/L, alkaline phosphatase 354 IU/L, serum amylase 6377 IU/L, serum lipase 2660 IU/L, LDH 777 IU/L, PT and APTT within normal values; serology for hepatitis B, A and C were negative as well as a negative ANF test. Pleural fluid study revealed a total cell count of 453 cell/cumm, with 75% mononuclear cells, LDH 1178 IU/L and on Ziehl-Neelsen staining of centrifuged deposit acid fast bacilli were visualised. Post-aspiration chest radiology did not reveal any appreciable parenchymal lung lesion or hilar lesions. Abdominal sonology showed a bulky pancreas with no evidence of cysts or focal edema, distended gall bladder with viscous sludge and enlarged liver with altered echotexture, and mild pleural effusion only on right side.
rupture due to tubercular lesion of the extrahepatic portion of the hepatic artery in a case of disseminated tuberculosis. Initial presentation with low-grade fever, high ESR, normal leukocyte count and raised serum amylase and lipase suggested a viral infection with pancreatic involvement. The clinical findings of mild icterus and tender hepatomegaly also suggested a viral hepatitis. Female sex, young age and high ESR also suggested a possible vasculitic or connective tissue disorder etiology. The negative ANF test and the demonstration of acid fast bacilli in pleural fluid settled the diagnosis of tuberculosis. The negative serology for the common hepatotropic viruses and the altered liver function results in a case of tuberculosis did point to tubercular liver involvement. Hepatic tuberculosis is known to cause deranged liver function test results with low albumin, raised globulin, raised aminotransferases and a marked increase in alkaline phosphatase.

Also tubercular pancreatitis has been reported from patients of Indian subcontinent origin. Liver biopsy was not done due to lack of consent. The episodic nature of the pain in abdomen, followed by upper gastrointestinal bleeding, failure to find a source of bleeding, icteric tinge and associated pancreatitis suggested the diagnosis of hemobilia. CT scan of the abdomen almost confirmed the diagnosis as also provided evidence of scattered hepatic calcification, commonly found in cases of hepatic tuberculosis. Finally the third endoscopy clenchd the diagnosis of hemobilia. Though a histological proof was not established, the most probable cause of the hepatic artery aneurysm and its rupture into biliary channel in this case was caseous necrosis due to tuberculosis. The disseminated nature of the disease in this patient strongly favours the tubercular etiology of the aneurysm.

We were fortunate in being able to timely resuscitate, diagnose and treat the young lady successfully. Selective

**DISCUSSION**

Trauma, (spontaneous and iatrogenic viz. hepatobiliary surgery, liver transplantation, biopsy), cholelithiasis, lupus vasculitis and associated pyogenic liver abscess are the leading causes of hemobilia reported worldwide. Both pulmonary and extra-pulmonary tuberculosis frequently involve the liver (25-66% and upto 80% cases, respectively). The granulomas of tuberculosis have the tendency to coalesce and focal cystic or tumour-like lesions with focal hemorrhage have been documented. However hemobilia as a complicating feature of tuberculosis has, to the best of our knowledge, been reported only in one case, where autopsy revealed hepatic artery rupture due to tubercular lesion of the extrahepatic portion of the hepatic artery in a case of disseminated tuberculosis.

Initial presentation with low-grade fever, high ESR, normal leucocyte count and raised serum amylase and lipase suggested a viral infection with pancreatic involvement. The clinical findings of mild icterus and tender hepatomegaly also suggested a viral hepatitis. Female sex, young age and high ESR also suggested a possible vasculitic or connective tissue disorder etiology. The negative ANF test and the demonstration of acid fast bacilli in pleural fluid settled the diagnosis of tuberculosis. The negative serology for the common hepatotropic viruses and the altered liver function results in a case of tuberculosis did point to tubercular liver involvement. Hepatic tuberculosis is known to cause deranged liver function test results with low albumin, raised globulin, raised aminotransferases and a marked increase in alkaline phosphatase. Also tuberculosis pancreatitis has been reported from patients of Indian subcontinent origin. Liver biopsy was not done due to lack of consent.

The episodic nature of the pain in abdomen, followed by upper gastrointestinal bleeding, failure to find a source of bleeding, icteric tinge and associated pancreatitis suggested the diagnosis of hemobilia. CT scan of the abdomen almost confirmed the diagnosis as also provided evidence of scattered hepatic calcification, commonly found in cases of hepatic tuberculosis. Finally the third endoscopy clenchd the diagnosis of hemobilia. Though a histological proof was not established, the most probable cause of the hepatic artery aneurysm and its rupture into biliary channel in this case was caseous necrosis due to tuberculosis. The disseminated nature of the disease in this patient strongly favours the tubercular etiology of the aneurysm.

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coeliac arteriography is the preferred mode of diagnosis of hemobilia, which is often missed on endoscopy, as in our case due to the episodic nature of the bleeding. Transcatheter embolization of the culprit artery is now the gold standard of therapy for most cases.¹

**REFERENCES**


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**Announcement**

Physicians interested in HIV / AIDS CARE

**Send Brief CV to JAPI Box. No. 4**

JK Mehta Clinic
Commissariat Bldg., 3rd Floor,
231 DN Road,
Mumbai 400 001.
Tel. : (022) 2611719