Gastric zygomycosis – an unusual cause of massive upper gastrointestinal bleed

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We report a 62-year-old man with cardiac failure and acute renal failure, who had massive hematemesis. Upper GI endoscopy showed a large gastric lesser curvature ulcer. Bilroth II gastrectomy specimen showed fungal invasion. He received amphotericin B postoperatively, and recovered uneventfully. [Indian J Gastroenterol 2004;23:146-147]

Key words: Mucormycosis, stomach

Zygomycosis refers to a group of uncommon but frequently fatal mycosis caused by fungi of class Zygomycetes. The spectrum of disease ranges from localized cutaneous to disseminated disease. Rhinocerebral and pulmonary disease are the most common forms, followed by gastrointestinal infections. Stomach is the most frequent site of involvement (67%), followed by colon (21%), small intestine (4%) and esophagus (2%). Although invasive gastrointestinal mucormycosis has been reported with fatal outcome, our patient, who presented with massive upper gastrointestinal bleed, had successful outcome after resectional surgery followed by antifungal therapy.

A 62-year-old man, a known hypertensive, was admitted in the cardiac unit with cardiac failure and acute renal failure. He was diabetic for the last seven years and had good glycaemic control on oral hypoglycemic agents. He was recovering well when after one week of hospitalization, he had sudden onset of massive upper gastrointestinal bleed with hemodynamic instability. Upper gastrointestinal endoscopy revealed a large ulcer in the body of the stomach on the lesser curvature. With a suspicion of malignancy, the patient was operated upon. After gastrectomy, multiple ulcers were seen in the body of the stomach, varying in size from 1 cm x 1 cm to 4 cm x 4 cm, with black slough covering them. Billroth II gastrectomy was performed.

Microscopic examination of the resected stomach showed broad, aseptic hypae with irregular walls and frequent perpendicular branching, foreign body giant cells and eosinophils; the hypae were invading the gastric wall as well as blood vessels. Postoperatively the patient received amphotericin B (1 mg/Kg/day) for three weeks and had uneventful recovery.

Gastrointestinal variants of zygomycosis are uncommon; most previous reports have come from South Africa with few cases from America, Europe and the Indian subcontinent. Gastric zygomycosis can be categorized into three forms: colonization, infiltration, vascular invasion. Colonization usually occurs in pre-existing gastric ulcers and is not fatal. Invasive form of gastric mycosis has variable presentation and is usually fatal. This form can either invade pre-existing peptic ulcer or can invade the stomach de novo.

The most frequent presentation of gastric zygomycosis is perforation, bleeding or epigastric discomfort. Lawson and Schmaman reported seven cases of invasive gastric mycosis, all presenting with perforated viscus, diagnosed either intra-operatively or at autopsy. Thomson et al. reported that all their seven patients with peptic ulcer disease and gastrointestinal mucormycosis, presented with complications. At upper gastrointestinal endoscopy, the ulcers usually appear malignant with rolled edges, but are usually covered with black necrotic tissue unlike the white slough of gastric ulcer. Biopsy usually reveals fungal hypae.

In the absence of complications medical management with antifungal agents is usually successful. In those with complications, resectional surgery followed by amphotericin B therapy is preferred. Non-resectional surgery carries high mortality. In one series all patients died after simple suture of perforation. Ninety percent of patients in another series underwent surgery for gastric mucormycosis and had successful outcome. Park et al. reported success with resectional surgery followed by antifungal therapy in bleeding peptic ulcer complicated by gastric mucormycosis. Increased awareness and early resective surgery in complicated gastric zygomycosis, followed by effective antifungal therapy, provides improved survival in patients with this otherwise fatal disease.

References

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Pulmonary aspergillosis in a child with hepatic failure

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Invasive aspergillosis is described more frequently as a complication of neoplastic disease and in immunocompromised patients. Hepatic failure is not a generally recognized risk for pulmonary aspergillosis. We report a 3-year-old boy who presented with hepatic failure and pneumonia and whose autopsy revealed liver cirrhosis and pulmonary aspergillosis. [Indian J Gastroenterol 2004;23:147-148]

Key words: Cirrhosis of liver

Pulmonary aspergillosis in children has been generally associated with immunocompromised state, neutropenia, broad-spectrum antimicrobial therapy, and acute organ rejection. We describe a child who presented with hepatic failure and pneumonia in whom autopsy revealed pulmonary aspergillosis.

A 3-year-old boy presented with fever for 15 days, associated with abdominal distension and pedal edema for 7 days, and seizures and altered sensorium for 4 days. He also had dark urine and gum bleeding for 3 days. There was no significant past medical history.

On examination, he was febrile and disoriented. His weight was 12 Kg and height 96 cm. He was pale and icteric. Abdominal examination revealed shrunken liver with span of 5 cm, and ascites. Respiratory system examination revealed decreased breath sounds on the right side and bilateral scattered crepitations.

Investigations: hemoglobin 5.6 g/dl, WBC count 3900/cumm (N 20%, L 64%, M 12%, B 3%, E 1%) and platelets 3000/cumm. Prothrombin time was 33.5 s (control 10.6) and INR 3.23; aPTT was 109 s (control 29.8). Total bilirubin level was 26.9 mg/dl (direct 20.8), total protein 3.5 g/dl (albumin 1.7), AST 4080 U/L, ALT 1020 U/L, and alkaline phosphatase 214 U/L. Serum ceruloplasmin was 5 U/L, serum creatinine 1.1 mg/dl, serum calcium 6.5 mg/dl, serum electrolytes normal. Malarial parasite, Widal test, blood culture, leptospirosis MAT, dengue IgM and HBsAg were negative.

Chest X-ray showed bilateral pulmonary infiltrates, more on the right side, with mild effusion. He was diagnosed to have hepatic failure with esophageal varices and bronchopneumonia, and was started on antibiotics, anti-hepatic coma measures, vitamin K, dextrose infusion, and fresh frozen plasma. He required mechanical ventilation for impending respiratory failure. In spite of the above measures he died within a few hours of admission.

Autopsy showed liver weight of 330 grams. Histology of the liver showed cirrhosis with bridging necrosis, cholestasis, and regenerative changes with fibrosis. Lungs showed foci of intra-alveolar hemorrhage, edema, and septa containing branching hyphae filaments of Aspergillus involving small and medium-sized vessels and pleura (Fig). The postmortem diagnosis was acute liver injury in a child with chronic liver disease with aspergillosis in the lungs and pleura.

Invasive aspergillosis is described more frequently as a complication of neoplastic disease and in immunocompromised patients. Hepatic failure is not a generally recognized risk factor for pulmonary aspergillosis. A few cases of pulmonary aspergillosis as a complication of cirrhosis have been reported. A review of literature revealed that 5 of the 14 previously reported cases of invasive aspergillosis in seemingly immunocompetent hosts were associated with liver disease.

Patients with fulminant hepatic failure have depressed hexose monophosphate shunt and phagocytosis-associated metabolic burst activity, which may increase their risk for severe infections.

Amphotericin B is the drug of choice for pulmonary aspergillosis. The drug may be considered in a child with hepatic failure, if aspergillosis is found in the sputum or if pulmonary infiltrates persist despite broad-spectrum antibiotics.

References

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