Surgery is advisable where possible because of the high incidence of rupture (up to 50%) in untreated cases. Surgical treatment includes ligation, revascularization with graft or primary anastomosis, oblitative aneurysmorrhaphy, endovascular embolization and, lately, transluminal endovascular stent insertion. The overall mortality of unruptured aneurysm is less than 5% but increases to 40% to 60% if rupture occurs.2

The reactive thrombocytosis seen in our patient is an indicator of bleeding,4 but can also be seen in inflammation and neoplastic conditions.

References


Correspondence to: Dr Rakesh, Flat 21, Staff Residences, Glenfirrty Close, Leicester LE3 9QJ, UK; E-mail: Rakesh203@sol.com.

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Hepatic failure with flutamide

R R KACKAR, H G DESAI

Department of Gastroenterology, Jaslok Hospital and Research Center, 15 Dr G Dehebnuk Marg, Mumbai 400 026

A 41-year-old woman, who was on flutamide for hair loss for 3 months, presented with deep jaundice. She developed hepatic encephalopathy but gradually recovered after discontinuing flutamide. Flutamide can cause fatal toxic liver injury and hence should be used with close monitoring of liver profile. [Indian J Gastroenterol 2003;22:149-150]

Key words: Drug-induced hepatotoxicity

Flutamide is used in the treatment of prostate cancer, hirsutism and hair loss. The major side effects are hepatotoxicity and diarrhea.1

A 41-year-old lady, dental surgeon, was admitted with history of loss of appetite, fever and vomiting for 2 days, followed by progressive jaundice for three weeks, and pedal edema and distention of abdomen for one week prior to admission. There was no history of jaundice, GI bleed, altered senso-

rium, or surgery or blood transfusion in the past. She was on flutamide 250 mg twice a day for 3 months for loss of hair. The drug was omitted on appearance of jaundice. She became drowsy (grade I coma) about 15 days after admission but regained consciousness in the next 48 hours.

Investigations: Blood count, serum creatinine, blood sugar, urine and stool examination were normal. Liver profile: AST 900 U/L, ALT 1380 U/L (normal <40), serum alkaline phosphatase 183 U/L (<150), proteins 5.3 g/dL (albumin 2.3), prothrombin time 58 s (control 16), INR 4.9, serum bilirubin 2.3 mg/dL which increased to 24.4 mg/dL (direct 16.1). Serological markers for acute viral hepatitis (IgM anti-HAV, IgM anti-HEV, IgM anti-HBC, HBsAg and anti-HCV) were negative.

Gamma globulin level, antinuclear antibodies, antismooth muscle antibodies and perinuclear antineutrophil cytoplasmic antibodies were normal. Serum copper oxidase was marginally low (0.15 OD; normal 0.2 to 0.5), K-F ring was negative; 24-hour urinary copper excretion (71 µg/day) and serum copper (78 µg/dL) were normal.

Ultra声ography showed small liver with coarse echotexture and moderate ascites, raising suspicion of chronic liver disease. Ascitic fluid showed cell count of 42 cells per mm³, protein 0.9 g/dL, and no malignant cells. Isotope scan showed normal-sized spleen with no increased uptake. Esophagogastroduodenoscopy showed no varices.

The patient was shifted to the UK for possible liver transplantation, but recovered there on supportive management.

Flutamide is a nonsteroidal anti-androgen that is converted to 2-hydroxyflutamide. It is a competitive inhibitor of binding of dihydrotestosterone to androgen receptors. The incidence of flutamide-induced liver injury is 1%-5%.2

The drug causes hepatotoxicity by several mechanisms. It binds to androgen receptors and increases the bioavailability of dihydrotestosterone and testosterone, which produces cholestatic hepatitis. The drug also causes immunoinflammatory type of reaction documented by peripheral cosinophilin.3 The likelihood of flutamide hepatotoxicity is enhanced by hypotension of any cause. Transient decrease of oxygen supply to subclinically flutamide-damaged hepatocytes causes cytochrome P450 (3A and 1B)-mediated formation of electrophilic metabolites; flutamide also exerts inhibitory effect on mitochondrial respiration and ATP formation.3 Simvastatin has also been reported to potentiate the drug hepatotoxicity.3

Jaundice generally commences 1-3 months after initiation of flutamide treatment; AST and ALT levels increase to more than 6 times normal.4 Ursodeoxycholic acid administration may be beneficial.6

References
Acute necrotizing gastritis

SATISH B DHARAP, GEETA GHAG, ARUNDHATI BISWAS
Department of Surgery, L T M Medical College and L T M G Hospital, Sion, Mumbai 400 022

A 17-year-old man presented with signs of peritonitis. Laparotomy revealed gangrene of the stomach without obvious cause. The patient underwent total gastrectomy with esophago-jejunal anastomosis with formation of jejunal pouch. Bacterial culture of the peritoneal fluid grew Strept. pyogenes and E. coli. The patient was discharged on day 21 after a stormy postoperative course. [Indian J Gastroenterol 2003;22:150-151]

Key words: Gastrectomy, stomach gangrene

Gangrene of the stomach is a rare catastrophic event that is often fatal. Causes are embolization of atherosclerotic plaque, thrombosis of major arterial supply, occlusion of gastric vessels by therapeutically injected foreign bodies, psychogenic polyphagia resulting in massive gastric dilatation, ingestion of caustic materials, intrathoracic herniation of the stomach through the diaphragm, volvulus of the stomach, and necrotizing gastritis caused by organisms.

We report a man with stomach gangrene that appeared to be caused by severe necrotizing infection with no known portal of entry.

A 17-year-old man presented during the period of Ramzan fasting with sudden onset of abdominal distention and pain with associated nausea and vomiting. On examination the patient was febrile, dehydrated, had tachycardia of 140/min, with systolic blood pressure of 90 mmHg. Abdomen was tender, guarded and rigid, with rebound tenderness all over and absent bowel sounds. Abdominal paracentesis was positive from all four quadrants and revealed foul smelling brownish hemorrhagic fluid. X-ray abdomen showed no gas under the diaphragm or air-fluid level.

With a clinical diagnosis of peritonitis, exploratory laparotomy was done after initial resuscitation. As the peritoneum was opened, there was an obnoxious smell. The proximal two-third of the stomach was slate gray in color with normal-looking pylorus. There was about 500 ml of brown hemorrhagic, foul-smelling fluid in the peritoneal cavity. No obvious perforation was noted. The diaphragmatic domes were normal and there was no evidence of volvulus of the stomach. Major gastric arterial pulsations were well felt but the end-vessels supplying the stomach were thrombosed. Total gastrectomy with Roux-en-Y esophago-jejunal anastomosis with the formation of jejunal pouch was done.

The patient needed ventilatory support for 10 days. He developed cerebral malaria, which was treated with artesunate, and pelvic abscess, which was tapped under ultrasonographic guidance. He was discharged on postoperative day 21.

The specimen revealed gangrenous, slate gray, thinned out stomach with loss of rugal folds in the proximal two-third. There was a sharp line of demarcation between necrotic and healthy tissue at the pylorus (Fig). When opened along the greater curvature, the fundus and body looked devitalized, with thrombosed vessels and sloughing of the mucosa; the lesser curve and antrum appeared uninvolved. Microscopic examination showed hemorrhages with intensive lymphocytic and eosinophilic infiltration in all major vessels of the stomach, with necrosis and congestion.

Peritoneal fluid grew Strept. pyogenes and E. coli on culture, sensitive to cefotaxime and amikacin.

The abundant and anastomotic nature of vascular supply of the stomach makes gangrene very rare. In the present case the finding of gangrene of the stomach was an operative surprise. Pulsatile gastric arteries, his young age, and absence of cardiac disease made vascular accident unlikely. Both the domes of the diaphragm were normal, ruling out strangulation of contents of a diaphragmatic hernia as the cause of gastric gangrene. No twisting of the stomach was noted; that ruled out volvulus. There was no history of swallowing any corrosive substance.

Fig: Stomach showing greyish necrotic fundus and body. Antrum and lesser curvature are spared