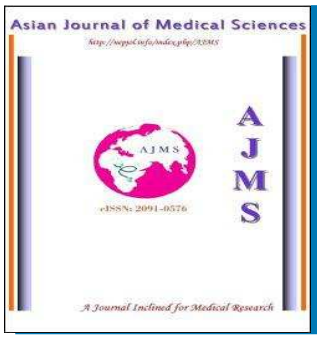

ACUTE GASTRIC NECROSIS IN A FOURTEEN-YEAR OLD CHILD

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"Early recognition of risk factors and prompt surgical intervention is the key to management of acute gastric necrosis."

ABSTRACT

Acute gastric necrosis is a rare entity of several possible causes. Progression of the disease is usually fulminant and diagnosis is made during surgery only. A-14 year child with acute onset abdominal pain and vomiting and with features of shock presented to the hospital. Rapid progression of surgical abdomen, hemorrhagic drainage in nasogastric tube and presence of gas in the portal vein could be a strong indicator of gastric necrosis. Recognizing the risk factors and prompt surgical intervention is the key to management.

Key words: Gastric, Necrosis

INTRODUCTION

Gastric necrosis is a rare condition with many etiologies that can be attributed to a patient's lifestyle or underlying morbidities or a result from a postoperative complication. The onset of symptoms is generally catastrophic and surgical therapy must be emergent and aggressive because mortality is substantially elevated for delayed diagnosis. Diagnosis of gastric ischemia is often delayed because of its rarity, but it may be considered in any patient presenting with surgical abdomen with no obvious cause. Once gastric necrosis is suspected, treatment needs to be undertaken immediately.

CASE REPORT

A 14-years-old-girl was referred to our hospital from a private clinic. History revealed sudden onset of severe abdominal pain since last 24 hours duration, starting from upper abdomen. Pain was associated with multiple episodes of vomiting (vomitus could not be characterized by the family members).

On examination in the emergency department; general condition of the patient was poor. Heart rate was 110 per minute, blood pressure of 60/40 mm/Hg and was afebrile. Oxygen saturation was 66% without supplemental oxygen. Immediate resuscitation was started and high-flow oxygen via mask given. After 1 hour, Spo_2 improved to 92%, BP recorded of 124/79 mm/Hg and HR - 90 per minute. Foley's catheter was inserted and 50 ml of concentrated urine was produced in next two hours.

The past medical history was noncontributory to the current medical problem. On examination: Abdomen was grossly distended and diffusely tender; there was mild guarding all over the abdomen. No visible peristalsis was noted. Bowel sound was audible on auscultation. Nasogastric tube was inserted which drained copious amount of hemorrhagic fluid. Serum electrolytes revealed sodium- 141.6mmol/l and potassium- 4.37mmol/l; hemoglobin 15.2 mg/dl; total leucocytes count 26500/mm^3, neutrophils 78% and lymphocytes 18%.

Ultrasound of abdomen revealed grossly distended bowel loops with mild ascites and air in the portal vein. Ultrasound impression was gastric or sigmoid volvulus.

X-ray of abdomen showed large fundic gas (Fig 1). Loops of small bowel and large bowel were found to be within normal.

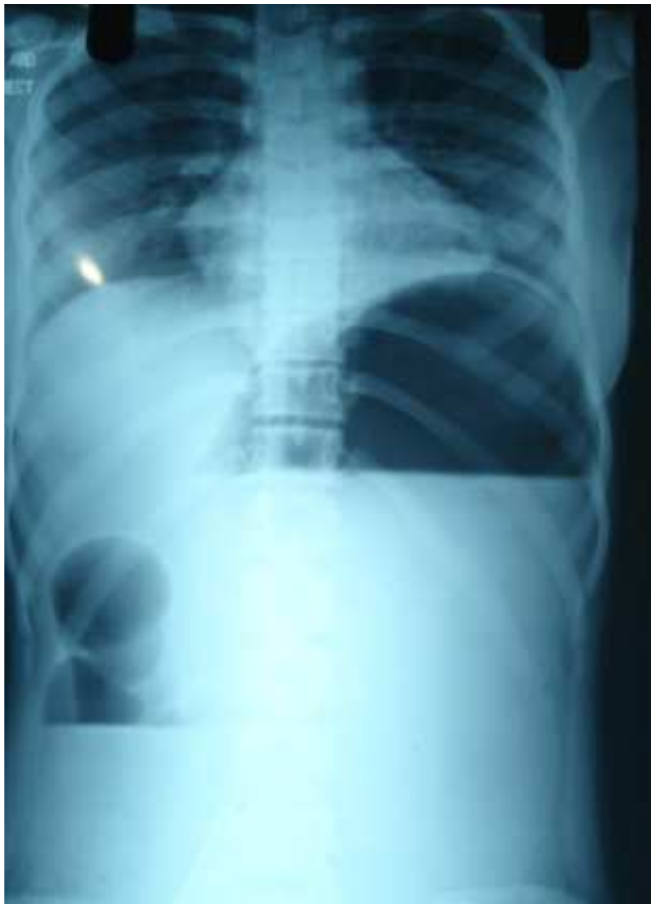

Figure1.

Operative finding: grossly distended stomach, occupying most of the abdominal cavity and stomach wall was very thin and it was blue to black in colour with some areas of hemorrhagic patches. The distal part of the antrum looked viable and there was no bleeding to cut from rest of stomach. All other organs including greater omentum were

normal. Pulse deficit of mesenteric vessels was absent. All vessels supplying the stomach, including left gastric, were normal. Some amount of hemorrhagic ascites was noted. After complete evaluation of abdominal cavity total gastrectomy with Rouxeny esophagojejunostomy was performed.

Postoperative course was uneventful. Nasogastric tube was self removed by patient on day five. Barium swallow was done on day eight, which was normal (Fig. 2) and the patient was gradually promoted to soft diet. Superficial infection of surgical site occurred, which healed after few days.

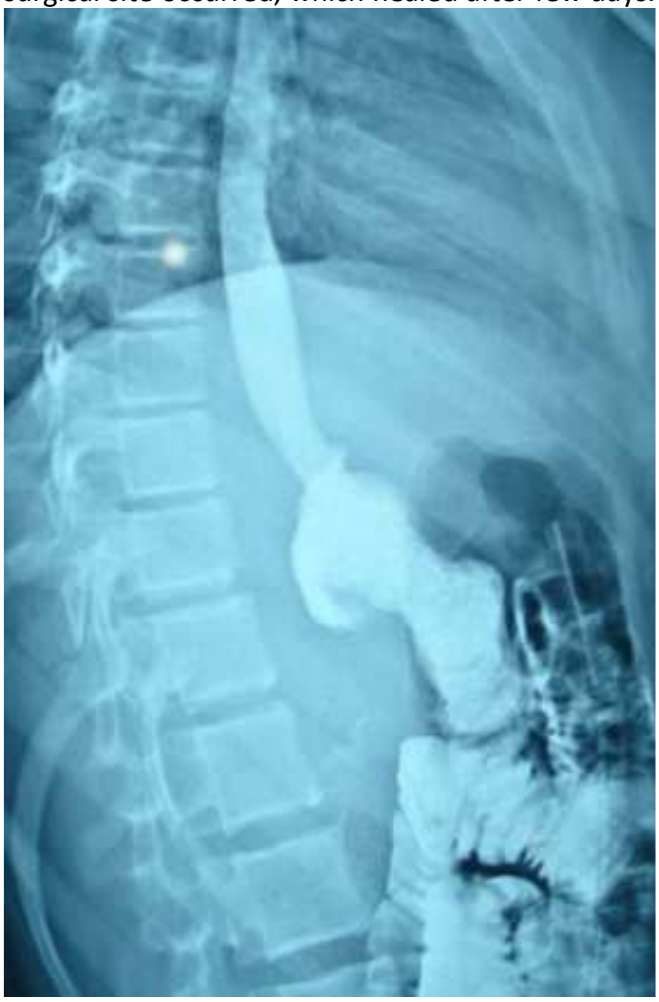

Fig. 2
Patient discharged on 12th post operative day.

DISCUSSION

Acute gastric necrosis is thought to be rare because of the stomach's abundant blood supply, which includes five major direct arterial Trindade EN, Von Diemen V and Trindade MR reported a case of acute gastric necrosis and perforation in a 13 years old child [1]. In adults gastric volvulus and vascular causes are defined, but in children the cause is mostly obscured. Turan M, Sen M, Canbay E, Karadayi K, Yidiz E reported a case of acute gastric dilation and necrosis in a mentally retarted 18 years old boy, following a heavy meal [2]. Stomach has many major sources (right and left gastric, right and left gastroepiploic, and short gastric) and numerous minor and collateral sources [3]. Schein and Saadia showed that complete gastric wall vascular filling can be achieved in cadavers with only one patent major artery [4]. Somervell found he could achieve gastric necrosis in animal models only after ligation of the right and left gastric arteries, right and left gastroepiploic arteries, and 80% of collaterals [5]. Babkin and colleagues noted that ligating most of the arterial supply to dogs' stomachs did not result in ischemia; however, both venous and arterial occlusion produced infarction in every case [6]. Cohen reported three cases of complete gastric necrosis and one case of partial gastric necrosis from a series of 23,836 autopsies in Los Angeles County General Hospital over a 12-year period. Cohen determined that all four patients not only had mesenteric arterial occlusions but also profound venous congestion secondary to right-sided heart failure. Diagnosis of gastric ischemia is often delayed because of its rarity. Initially, patients may have symptoms of mild epigastric tenderness, vomiting, or diarrhea that rapidly progress to acute peritonitis, septic shock, and death. The preliminary diagnosis may be supported by CT scan findings of pneumoperitoneum, gastric pneumatosis, and portal venous gas. Actual diagnosis is often made at laparotomy or autopsy. In our patient, the first investigation was ultra-

-sound. Ultrasound showed distended stomach and air in the portal vein. In association with USG report, finding of distended stomach in plain abdominal x-ray with no abnormal distension of bowel loops and hemorrhagic drainage from nasogastric tube were strong positive signs of gastric necrosis. Based on these findings rather than going for a CT scan and wasting few precious hours, we opted for early laparotomy. In our case, pulse in the left gastric artery was perceivable. When massive dilatation occurs, ischemia is presumably due to venous insufficiency: 14 mm/Hg of pressure is sufficient to impair venous outflow and intraluminal quantity of more than 3 L of fluid can distend the stomach to that pressure, although chronic distension of more than 15 L has been described in eating disorders (polyphagia, bulimia). Rupture can occur with intragastric pressures of more than 120 mm/Hg (corresponding to approximately 4 L of fluid), but pressure can reach these values even after cardiac compression during the course of resuscitation [7]. Atonicity of the stomach can also lead to massive distension when food intake is reprised, and this happens in case of anorexia nervosa or electrolyte imbalance. Mechanical factors can be implied in gastric dilatation, like bowel obstruction or pyloric stenosis,[8] and infectious causes (necrotizing gastritis) have been reported, generally involving immunocompromised patients (diabetes, AIDS, neoplasia)[9,10]. Physiopathologic theories have been debated in the past: one advocated upper esophageal sphincter relaxation (due to debilitation or anesthesia) with consequent aerophagia and gastric dilatation as a cause. Atonic theory (muscular atrophy during prolonged starvation does not support rapid refeeding), superior mesenteric artery syndrome (vascular compression of the third duodenal portion) or atonic conditions caused from regional alterations (in pancreatitis, ulcer and other abdominal inflammations) are other supposed etiologies. A consequence in events is postulated by Abdu: the first step should be mucosal necrosis, followed by full-thickness involvement of the gastric wall and perforation [11]. Clinically, emesis might be the initial symptom, but events can precipitate suddenly to shock. Physical findings are abdominal distension and tympanism, with tenderness and peritonitis in case of perforation. Plain abdominal films and CT scan are useful in the diagnosis as they can demonstrate gastric distension and free air. Decompression with nasogastric tube is mandatory as the first therapeutical act, followed by immediate surgery. Necrosis might be partial (mostly in the lesser curve due to vascular supply) or involving the full organ. If a diagnosis of gastric ischemia is being considered, resuscitation and intravenous antibiotics should be initiated immediately, followed by an emergency exploratory laparotomy. Resection of a necrotic stomach is required, with total gastrectomy if necessary. One-stage resection with esophagojejunostomy has been recommended and was performed in this case. Alternatives include resection with cervical esophagostomy for proximal diversion or resection and placement of an esophageal drain. Though we did not put in our patient, a jejunal feeding tube should be placed according to some authors. Revascularization of acute thromboses or embolism is necessary if either is found to be the etiology. Diagnosis and treatment must be expeditious, because mortality rates for gastrectomy due to acute ischemia are high, ranging from 50% to 80% [12-14].

CONCLUSION

Although gastric ischemia is rare, it is important to recognize the risk factors. Disease or insult to both the arterial inflow and venous outflow is necessary for the condition to occur. Progression to necrosis leads to peritonitis and sepsis. Urgent laprotomy is

warranted, and the diagnosis is made at operation. Treatment should consist of resection and feeding jejunostomy may required. Increased awareness of this rare entity and its risk factors may lead to more prompt diagnosis and an increased chance for patients' survival.

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