Corrosive-Induced Pyloric Obstruction with Oesophageal Sparing: A Case Report

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ABSTRACT

We report an unusual case of isolated gastric outlet obstruction following ingestion of corrosive liquid by a 25 year old male banker who attempted suicide. He presented with features of gastric outlet obstruction three months following intentional ingestion of car battery fluid of concentrated sulphuric acid in an attempt to take his own life. Prior to this, patient had been on suspension from office as a result of some problems he had with his employers. He however had no prior history of psychiatric illness or peptic ulcer disease. Barium meal confirmed antro-pyloric stenosis with dilated proximal stomach and normal oesophagus. The patient underwent nutritional rehabilitation with supplemental parental and oral high calorie, high protein diet to regain some weight and offered restorative gastro-jejunostomy to by-pass the stenosis. He recovered uneventfully and had remained stable on follow up visits. Acids usually cause more damage to the stomach than the oesophagus but in this case the oesophagus escaped any form of injury thereby highlighting the relative resistance of the oesophageal mucosa to acid ingestion.

Keywords: Corrosive, Gastric, Outlet, Obstruction

INTRODUCTION

Isolated corrosive gastric outlet obstruction with oesophageal sparing is rare.[1] The usual picture with acid ingestion is extensive gastric damage with associated varying degrees of oesophageal injury, while alkalis tend to cause more damage to the oesophagus than the stomach[2,3]. Acids of clinical importance include sulphuric acid, hydrochloric acid, nitric acid, acetic and hydrofluoric acids, while alkalis include potassium and sodium hydroxide, sodium hypochlorite and ammonia. Accidental corrosive ingestion is more common among children especially in developing countries where proper labeling remains a challenge[4]. Adults on the other hand commonly ingest corrosives deliberately as a form of self- harm or attempted suicide as in the index patient[5]. Most patients have history of psychiatric illness in form of depression, post-traumatic stress disorder or schizophrenia.[5] Injuries in adults tend to be severe due to deliberate ingestion of large quantities of corrosives[5]. The presentation may be immediate or late depending on extent of injury. At presentation, most patients are too weak to withstand immediate operative intervention due to severe dehydration and malnutrition. Immediate damage can be assessed by endoscopy or barium meal to accurately determine the extent of injury and map out treatment plans[5]. Barium meal may show pyloric stenosis from mucosal edema or hyperaemia and may also show ulcer craters from superficial or deep ulcerations. Hawkins et al proposed an endoscopic grading of oesophageal corrosive injury as follows: Grade 0= Normal examination, Grade I= Mucosal edema or hyperaemia, Grade IIA= Superficial localized ulcerations, friability and blisters, Grade IIB= Grade IIA plus circumferential ulcerations, Grade III= Multiple deep ulcerations with areas of necrosis[6]. Treatment depend on the type and extent of injury. Mild injuries (grades 0 to IIA) are amenable to balloon dilatation with or without intralesional injection of steroids while the most severe cases (grades IIB to III) would require surgery[6,7,8].

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CASE PRESENTATION

The patient was a 25 year old banker who was referred to the surgical out-patient department of our hospital with three month history of persistent, post-prandial, non-bilious, projectile vomiting following deliberate ingestion

of car battery fluid of concentrated sulphuric acid in an attempt to commit suicide. Prior to this, patient had been on suspension from office as a result of problems he had with his employers. Patient had initial treatment from the referring private hospital where he earlier presented with features of upper gastro-intestinal bleeding. There was no history of dysphagia, cough or epigastric pain. Patient had no prior history of peptic ulcer disease. Examination revealed a pale, dehydrated and emaciated young man with bilateral pitting pedal edema. The chest was clear clinically, the epigastrium was full, with positive succussion splash. There was no visible peristalsis, no palpable abdominal masses and no hepato-splenomegaly. A diagnosis of corrosive induced gastric outlet obstruction was made. Barium meal revealed a contracted distal stomach with severe antro-pyloric stenosis. The proximal stomach appeared dilated while the oesophagus appeared normal. Laboratory investigations revealed a packed cell volume of 28%, white cell count of $3.1x10^{9}/l$ and platelet count of $278x10^{9}/l$. Total protein was 5.0g/dl, Albumin 3.6g/dl. Retroviral and Hepatitis B screening were non-reactive. The serum sodium was 128mmol/l, potassium, 2.8mmol/l, chloride, 92mmol/l, bicarbonate, 22mmol/l and urea, 5.5mmol/l while creatinine was 0.9mg/dl. As part of pre-operative work-up, patient was rehydrated with intra venous crystalloids to correct the electrolyte derangement. He had one pint of blood transfused and nutritionally rehabilitated with parenteral and oral high protein, high calorie diet to regain some weight. He was offered a simple, open, ante-colic gastro-jejunostomy. Operative findings included a thick, fibrotic gastric antrum with severe pyloric stenosis. The proximal stomach was grossly dilated but the intra-abdominal oesophagus appeared normal. There were no surrounding fibrous adhesions or regional lymphadenopathies. Other abdominal viscera were normal. The post-operative course was uneventful and he was discharged after nineteen days on admission. Subsequent follow up visits showed appreciable weight gain and gradual return to pre-disease state.

DISCUSSION

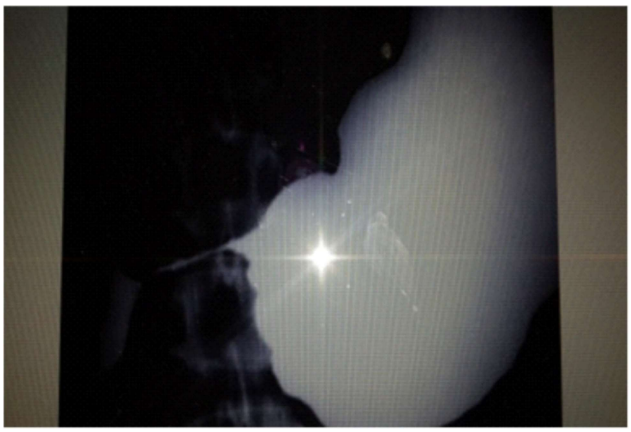

Figure 1: Barium meal of patient showing severe antro pyloric stenosis with dilated proximal stomach.

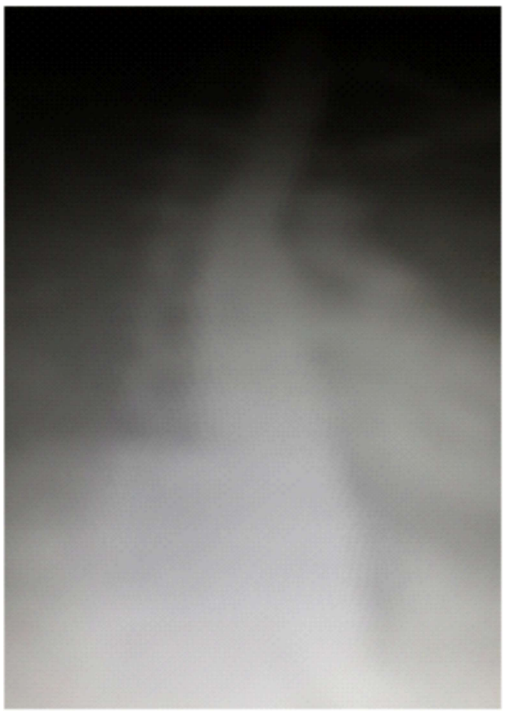

Figure 2: Contrast upper gastro-intestinal series of patient showing normal oesophagus.

The extent of injury caused by corrosive ingestion depends on its concentration, the contact time and volume[9]. While acid ingestion causes coagulation necrosis, with the coagulum limiting further tissue penetration and insult, alkalis induce liquefaction necrosis with protein dissolution and fat saponification, resulting in further tissue damage[2,3,10]. Ingestion of alkalis result in oropharyngeal and oesophageal injury whereas acids tend to spare the esophagus and cause injury to the stomach[1,5,11-13]. The oesophagus

resists acid injury partly because of its stratified squamous epithelium and because of the rapid transit of acids through it but in the stomach, acid causes reflex pyloric spasm resulting in severe burns to the pylorus and distal stomach. Alkalis on the other hand tend to coat the oesophageal mucosa and therefore transits slowly causing more injuries in the oesophagus than in the stomach[1,5,11-13]. This isolated pattern of injury is however rare as the usual picture is mixed with acid and alkali causing injury to both the stomach and oesophagus.[2,3] In the index case the oesophagus and oropharynx were spared. This was quite unusual since patient admitted to ingesting large quantity (nearly one coca cola bottle full) of the corrosive in an attempt to take his own life. This is therefore a typical case of probable rapid transit of acid through a relatively resistant oesophageal mucosa leading to isolated pyloric injury. Corrosive ingestion triggers necrosis followed by ulceration and subsequent fibrosis. The end result is extensive stenosis of affected lumen. In the stomach, the outlet obstruction could be as early as seven days or as late as six years with antro- pyloric oedema being mainly responsible for early obstruction while fibrosis is responsible for late presentation[14]. Most African and Indian patients however tend to present with early fibrotic gastric outlet obstruction due to early onset of fibrosis[5,15]. The index case presented three months after ingestion of corrosive with established gastric outlet obstruction, malnutrition and anaemia. This picture often makes prompt surgical intervention a huge challenge. The index patient was resuscitated with intravenous fluids to correct electrolyte derangement, the stomach was lavaged with nasogastric tube and he was commenced on parenteral and oral feeds to correct malnutrition. He subsequently had simple ante colic gastro-jejunostomy to by-pass the stenosed distal stomach. Other operative procedures that may be offered depending on extent of injury include pyloroplasty, partial gastrectomy with Billroth I or II reconstruction, advancement antroplasty and total gastrectomy with Roux-en-Y oesophagojejunostomy[16]. However in most cases, the patients are too weak or malnourished to withstand these major procedures and extensive cicatrization also precludes routine use of pyloroplasty as a corrective method. Even though simple by-pass is preferred by most surgeons, there is a slight chance of subsequent metaplastic changes in the stenosed gastric remnant. For this reason some surgeons advocate a more radical approach in resection of the involved stomach[3,17]. However, in most instances the operative risk far outweighs the potential risk of metaplasia and subsequent carcinoma. The first reported case of squamous metaplasia in the stomach of a patient who had ingested acid was by Howard and Holmes in 1948[11]. Since then several authors have reported similar findings[18,19]. Regular follow up of these patients is therefore important for early detection and treatment. Unfortunately, the index case was lost to follow up after six months. Mortality in corrosive injury approaches 20% but rises sharply to over 70% in cases of suicide attempt[10]. Morbidity and mortality occurs chiefly as a result of haemorrhage, perforation, severe dehydration and malnutrition.

CONCLUSION

Acid ingestion causes more damage to the stomach than the oesophagus but in this case the oesophagus escaped injury thereby highlighting the relative resistance of the oesophageal mucosa to corrosive acid.

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