CASE REPORT

Corrosive - Induced Gastric Outlet Obstruction Without Oesphageal Involvement: A Case Report

N. Ali MBBS, FWACS, **U.E. Eni** MBBCh, FWACS
Dept of Surgery, College of Medical Sciences, University of Maiduguri
and University of Maiduguri Teaching Hospital, Maiduguri, Nigeria.

ABSTRACT

Background: *The objective of this paper is to report an unusual case of isolated gastric outlet obstruction following corrosive ingestion.*

Method: *A case report of a 28-year old female seen by the authors. The literature on gastric outlet obstruction following ingestion of corrosives is reviewed briefly.*

Results: *Features of worsening gastric outlet obstruction were found in this 28-year old female five months after ingestion of hydrochloric acid. There was an antecedent history of depressive illness. The upper gastrointestinal barium contrast radiographs showed a normal oesophagus and proximal stomach. The distal stomach was however scarred, contracted with severe antropyloric stenosis. She underwent nutritional rehabilitation with high protein diet and made an uneventful recovery after a gastrojejunostomy.*

Conclusion: *This case suggests a relative resistance of the oesophagus to corrosive acids as reported in the literature. The stomach, however, is more susceptible to acids causing burns with subsequent cicatrisation around the antrum and pylorus.*

KEY WORDS: *Corrosives; Gastric outlet; Obstruction; Oesophageal noninvolvement.*

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INTRODUCTION

Upper gastrointestinal tract injury caused by ingestion of corrosive substances, accidentally or deliberately (as a suicide attempt) has been extensively studied[1]. It is associated with oesophageal burns and resultant oesophageal stricture in 6 to 63% of patients depending on the nature of corrosive agents. However, gastric outlet obstruction (GOO) without oesophageal involvement, a sequelae of corrosive acid ingestion, is not as common[2]. The corrosive agents include hydrochloric acid, nitric and sulphuric acid, which tend to affect the stomach more, and alkalis like potassium and sodium hydroxide which affect mainly the oesophagus. Other corrosive agents include lye, detergents and formalin. The typical patient is a child or an adult (some with psychiatric problems) who has ingested corrosives accidentally or deliberately. In the adults the injuries are frequently more serious because they are intentional, with larger volumes of ingested corrosive. The presentation may be immediate or delayed depending on the extent of injury. Fibreoptic endoscopy and upper gastrointestinal contrast radiographs done within 24-36 hours of injury give a good assessment of the extent of injury and may help suggest treatment. Hawkins and colleagues[3] classified lesions endoscopically into four grades: 0, I, II and III. In their series most cases of grades 0-IIa did not require surgical treatment. The moderate to severe groups (groups IIb-III) however, required feeding jejunostomy and subsequent surgery. We report a rather unusual case of isolated gastric outlet obstruction following corrosive ingestion together with a brief literature review.

CASE REPORT

JFJ, a 28 year old Nigerian female school teacher (with a background depressive illness) presented to our surgical unit with a 10 month history of persistent post-prandial vomiting, easy satiety and marked weight loss. These symptoms followed deliberate ingestion of hydrochloric acid. The vomiting had become projectile and more in intensity five months prior to presentation. There was no dysphagia, cough or epigastric pain.

Physical examination revealed a pale and wasted young woman with bilateral pedal oedema. There was no peripheral lymphadenopathy. The epigastrium was full, with positive succussion splash and visible peristalsis that moved from left to right. There was no hepatosplenomegaly and no palpable abdominal masses. A clinical diagnosis of corrosive-induced gastric outlet obstruction (GOO) was made.

Laboratory investigations revealed a packed cell volume of 24%, and serum sodium, potassium, chloride, bicarbonate, and urea of 130, 2.8, 100, 20 and 13 mmol/L respectively. She had total serum protein of 58.0g/L and albumin of 26g/L. The liver function tests were normal and the retroviral (HIV) screening and hepatitis B surface antigen (HBsAg) were negative.

Upper gastrointestinal barium contrast radiographs demonstrated a completely normal

Correspondence to: **Dr. U.E Eni**

oesphagus (Fig. 1). The distal stomach was however scarred and contracted, with severe antro-pyloric stenosis (Fig. 2).

The patient was worked up for laparotomy, which was performed after one month, during which time she received three units of whole blood and nutritional rehabilitation with high protein diet. This nutritional build-up was possible after repeated gastric lavages and resting the stomach.

The operative findings were diffuse fibrous adhesion in the supracolic compartment especially in the right sub-hepatic space. The stomach was thickened and scarred with severe pyloric stenosis. Other abdominal viscera were grossly normal. She had an anterior gastrojejunostomy. The postoperative course was uneventful and she was discharged on the 14th day to be followed up at the surgical out patient clinic.

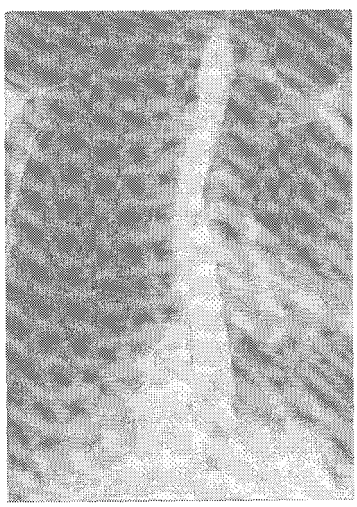

Figure 1.
An upper gastrointestinal barium contrast radiograph showing a completely normal oesophagus and upper stomach

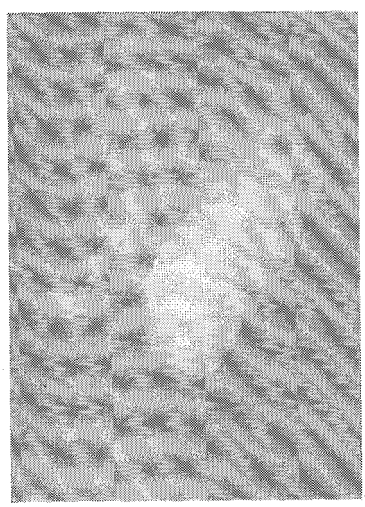

Figure 2.
A Barium meal of the same upper gastrointestinal series showing a deformed, scarred and contracted stomach with severe antro pyloric stenosis.

DISCUSSION

Our patient presented with symptoms of GOO 5 months after ingestion of hydrochloric acid. The development of GOO could be as early as 7 days or as late as 6 years[4]. This is signaled by post prandial epigastric fullness and discomfort, persistent non-bilious vomiting, visible peristalsis across the upper abdomen and a positive succussion splash. The patients are severely wasted, and often have fluid, electrolyte and acid/base imbalance. The immediate treatment is therefore resuscitative[5]. The history and typical endoscopic and radiological features [Figs. 1 and 2] confirm the diagnosis and enable definitive treatment to be planned.

Hydrochloric acid produces coagulation necrosis of the gastric mucosa and submucosa and the process may involve the entire thickness of the gastric wall, with subsequent ulceration and fibrosis. This dynamic pathophysiologic event influences the timing of surgical intervention because of the variable interval between acid ingestion and sufficient cicatrisation. The fact that most acid burns affect the stomach and concentrated alkalis the oesophagus (by liquifactive necrosis) is borne out by many studies[6,7]. This pattern of injury occurs because of the relative resistance of the oesophageal mucosa (stratified squamous epithelium) to acid and its rapid transit through the oesophagus. In the stomach, on the other hand, the severe reflex pyloric spasm in response to acid occludes its outlet causing severe burns to the distal portion. Corrosive alkalis however tend to coat the oesophagus and transit slowly therefore penetrating deeper and causing more damage to this site Nonetheless, injuries to both the stomach and the oesophagus occur with ingestion of both acids and alkalis.

The damage that follows ingestion of acid may present acutely as a perforation or necrosis of the stomach requiring emergency operative treatment or later with progressive antro-pyloric cicatrization (in about 30% of patients) causing GOO.

Our patient presented 10 months after ingestion of corrosives with established GOO, malnutrition and anaemia. Gastric lavage allowed feeding with high protein liquid diet. This together with plasma and whole blood transfusions were able to correct her serum protein derangement and anaemia before surgery.

The non-operative treatment includes endoscopic balloon dilatation with or without intralesional triamcinolone injection where the success rate was more than 80% in moderate cases[8]. Where there is severe GOO with

malnutrition the treatment of choice is surgery in the form of partial gastrectomy. Billroth I or II reconstruction is done depending on the length of available duodenum. Other surgical procedures such as pyloroplasty, Y-V advancement antroplasty and anterior gastrojejunostomy are also done depending on findings at laparotomy[9]. The timing and nature of surgery for corrosive-induced GOO is determined by acceptable nutritional status, fully developed scar tissue, grade of injury and experience of the surgical team. Notwithstanding a potential risk of malignant evolution in the scarred gastric remnant, a gastrojejunostomy was the operation of choice in our patient because of the extensive cicatrization. It is also a lesser grade of surgery compared to all forms of gastric resection with less complication.

In conclusion GOO following ingestion of corrosive substances (especially mineral acids) causes a lot of morbidity and mortality. A full assessment with fibreoptic endoscopy or barium contrast studies is not only necessary but may suggest definitive treatment. The treatment of choice is surgery and distal gastric resection, pyloroplasty and gastrojejunostomy, in that order, are preferred. Non-operative treatment via endoscopic balloon dilatation and intralesional triamcinolone injection are not as successful and may be prone to recurrence. Access to corrosive substances should be regulated in our environment by legislation.

REFERENCES

1. Maull KI, Scher LA, Greenfield IJ. Surgical implication of acid ingestion. Surg Gynaecol Obstet 1979; 148:895-8.
2. Ciftci AO, Senocak ME, Buyukpamukca N, *et al.* Gastric outlet obstruction due to corrosive ingestion: incidence and outcome. Paediatr Surg Int 1999; 15:88-91.
3. Hawkins DB, Demeter NJ, Barnett TE. Caustic ingestion: controversies in management: a review of 214 cases. Laryngoscope 1980; 90:98-109.
4. Sugawa L, Lucas CE. Caustic injury of the upper gastrointestinal tract in adults: a clinical endoscopic study. Surgery 1989; 106:802-6.
5. Mc Auley CE, Steed DL, Webster MM. Late sequelae of gastric acid injury. Am J Surg 1985; 149:412-5.
6. Tamisani AM, DiNoto C, DiRovasenda E. A rare complication due to sulfuric acid ingestion Eur J Paediatr Surg 1992; 2:162-4.
7. Collure DW. Pyloric obstruction following ingestion of corrosive acid. Ceylon Med J 1989; 34:135-7.
8. Kochhar R, Sethy PK, Nagi B, *et al.* Endoscopic balloon dilatation of benign gastric outlet obstruction. J Gastroenterol Hepatol 2004; 19:418-22.
9. Hsu C, Chen C, Hsu N, *et al.* Surgical treatment and its long- term result for caustic induced prepyloric obstruction. Eur J Surg 1997; 163: 275-9.