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Isolated Gastric Outlet Obstruction - Sequelae of Corrosive Ingestion in Paediatric Age-Group: Literature Review

Charu Tiwari*, Shalika Jayaswal, Kiran Khedkar*, Deepa Makhija*, Pankaj Dwivedi*, Hemanshi Shah*****

*Registrar, **Associate Professor, ***Professor & Head, Dept of Paediatric Surgery, TNMC & BYL Nair Hospital, Mumbai Central, Mumbai, Maharashtra. India.

Abstract

Background: Accidental corrosive ingestion is common in the paediatric age group. Severity may vary from no injury to a fatal outcome [1]. However, isolated gastric outlet obstruction (GOO), though rare, is a well-known complication of corrosive ingestion. We present five such cases. *Cases' Summary:* The mean age of presentation was 5.4 years. There were 4 males and 1 female. The average time duration between ingestion of the corrosive and presentation was 40 days. The common presenting symptoms were abdominal pain and non-bilious vomiting mixed with food particles seen in all five patients. Endoscopy was done in four patients - all four patients had antral and pyloric thickening. The scope could be negotiated across the pylorus only in one patient with difficulty. It could not be negotiated in the other three patients. Retrocolic, isoperistaltic gastrojejunostomy was done in four patients and pyloroplasty was done in one patient. Post-operative recovery was uneventful in all patients. *Conclusion:* Corrosive ingestion is an important cause of significant morbidity, especially in developing countries. Early diagnosis and appropriate endoscopic and/or surgical intervention usually lead to a good outcome.

Keywords: Ingestion; Corrosives; Gastric Outlet Obstruction; Total; Partial; Children.

Introduction

Accidental corrosive ingestion is a common problem in the paediatric age group [1]. In children, alkalies are the ingested agents in about 80% of cases [1, 2, 3]. Severity may vary from no injury to a fatal outcome [1].

Isolated gastric outlet obstruction (GOO), though rare, is a well-known complication of corrosive ingestion [1]. There is a paucity of such reports in literature and most reports are in adults in which various management modalities with varied outcomes are discussed [1]. The management of such injuries is challenging.

We report five cases of gastric outlet obstruction following accidental corrosive ingestion in children.

Cases' Summary

Demographic Details

Five children with corrosive ingestion and clinical features of GOO and their management are described. The mean age of presentation was 5.4 years (range-3 years to 6 years). There were 4 males and 1 female. The time duration between ingestion of the corrosive and presentation was 40 days (range: 20 days to 60 days).

Clinical Features

The common presenting symptoms were abdominal pain and non-bilious vomiting mixed with food particles seen in all five patients. The female child presented with severe dehydration, acidosis

Corresponding Author: Hemanshi Shah, Professor & Head, Dept of Paediatric Surgery, TNMC & BYL Nair Hospital, Mumbai Central, Mumbai, Maharashtra. India. Pin: 400008.
E-mail: hemanshisshah@gmail.com

and electrolyte imbalance along with these symptoms. Visible peristalsis was seen in four patients.

Management and Outcome

All patients were admitted. Fluid and electrolyte balance was corrected. An upper gastrointestinal tract contrast (water soluble) study was done. Endoscopy (oesophago-gastro-duodenoscopy) was done in four patients. All five patients had grossly dilated stomach on contrast study. There was small streak of dye going distally across the pylorus in two patients (Figures 1, 2 and 3). Three patients had complete GOO (Figure 4).

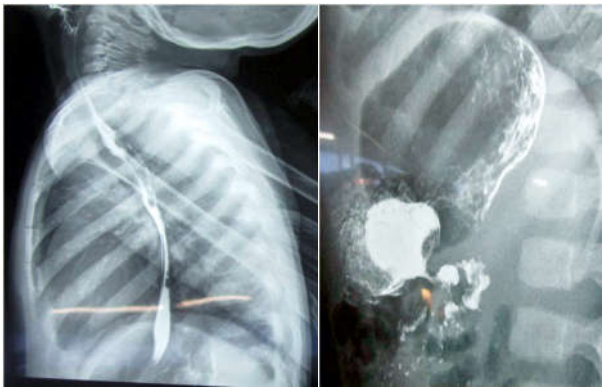

Fig. 1 and 2: Upper gastrointestinal contrast X-rays (Lateral and Antero-posterior) of a patient showing partial Gastric Outlet Obstruction.

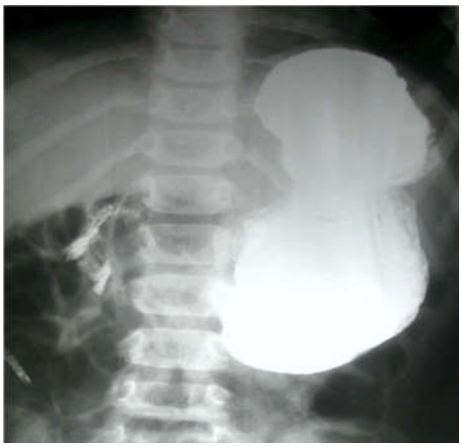

Fig. 3: Upper gastrointestinal contrast X-ray of a patient showing total Gastric Outlet Obstruction

At endoscopy, all four patients had antral and pyloric thickening. The scope could be negotiated across the pylorus only in one patient with difficulty. It could not be negotiated in the other three patients.

Exploratory laparotomy was done in all patients. One patient had only pyloric thickening and pyloroplasty was done. Four patients had grossly dilated stomach with thickened and stenosed pylorus and antrum. A retrocolic, isoperistaltic gastrojejunostomy was done . Post-operative recovery was uneventful in all patients. Orals were gradually started at fifth post-operative and patients were discharged after one week. At follow-up, all patients are aymmptomatic and are gaining weight.

Discussion

Both acids and alkalies act as corrosives after ingestion and produce considerable and progressive damage to the upper gastrointestinal tract [4]. The nature of the agent, the amount and concentration ingested, the amount of food already present in the stomach at the time of ingestion and the intention (suicidal or accidental) of ingestion are the factors affecting the degree of mucosal injury [4, 5]. Presence of a pre-existing comorbid condition compounds the damage [4]. The burden of morbidity caused by the corrosive substances is more in developing countries because of the easy availability of these agents as items of household use which are not under regulatory control [4].

Human exposure to these corrosive agents is usually either accidental or suicidal, the circumstances being different in paediatric and adult populations [4]. Accidental ingestion of corrosive substances is seen most common in young children (80%) [4]. Children under 5 years of age are curious by nature and unaware of the dangers of these toxic agents [1]. Hence, they are more prone to accidental ingestion of these corrosives [4]. This coupled with easy availability of corrosive substances in the form of cheap toilet cleaners which is kept in empty bottles of mineral water is a common cause of such incidences in developing countries like India [4]. Isolated pyloric and antral stenosis without oesophageal injury, though uncommon has been reported. There are only few reports in literature which highlight the management of corrosive stricture of pylorus and antrum of the stomach in children [5, 6].

There is a common belief that alkali ingestion causes severe esophageal damage and limited gastric injury due to the buffering action of acid [7]. However, pyloric stenosis has recently been reported to occur with corrosive alkali ingestion [1,3,7,8,9]. There is a well known tendency of acids "to lick the esophagus and bite the pyloric antrum" [5]. The lower viscosity and specific gravity of corrosive acids when compared to liquid alkalis, lead to their rapid transit through the esophagus and the damage primarily occurs in the antrum and pyloric region of the stomach

[5, 10]. This rapid transit is coupled with antral spasm which causes pooling of the corrosive consequently causing more damage to the antrum [5]. The stomach, with its columnar epithelium is more susceptible to corrosive damage when compared with oesophagus which has a more resilient squamous epithelium [5, 11]. As suggested by Nuutinin et al, acids cause burn injuries more often than alkalies which more often develop into scars as a result of coagulation necrosis of the tissue in contact [1]. With a severe injury to the stomach, gastric outlet obstruction (GOO) may occur as early as three weeks or as late as 10 weeks [7].

The children usually present with non-bilious vomiting, post-prandial fullness followed by early satiety, decreased oral intake and rapid weight [1, 5]. Patients may present with acute complications like oesophageal perforation (<2% of cases), aspiration pneumonitis and respiratory failure [4]. In patients who present early, after stabilization, an upper gastrointestinal endoscopy is recommended to characterize the nature of injury, if it can be done between 48 and 72 hours of ingestion [4,12]. However, endoscopy is not recommended between 5 and 1 days of ingestion due to a high risk of perforation [4]. The corrosive injuries are graded endoscopically as grade 1, 2a, 2b, 3 and 4 as per the classification system proposed by Zarger et al [4,13]. It has been reported that sequelae like oesophageal and/or gastric stricture and cicatrization is more common with grades 2b (penetration to the submucosa with ulceration or whitish membranes) and 3 (transmural involvement with deep injury and necrotic mucosa) injuries [1,4, 13-16]. A contrast study is recommended to confirm the injuries in such patients and identify the complications [1]. Oesophageal injuries, strictures and cicatrization are more commonly reported as long-term sequelae; however, gastric antral and pyloric injuries causing GOO is also not uncommon in children [4,5,17]. Both alkalies and acids are known to cause GOO.

The management of GOO in such paediatric patients is controversial because of the paucity of literature [1,5,17,18]. However, early surgical intervention has been recommended as the treatment of choice with satisfactory results in various studies [1,5, 7, 20].

The various management options are feeding jejunostomy and endoscopic balloon dilatation of stricture, gastrojejunostomy with or without vagotomy, pyloroplasty, or antrectomy with Billroth I anastomosis [5, 19, 20, 21]. Each of these various procedures has its own advantages and disadvantages [5].

Pyloroplasty and endoscopic procedures like balloon catheter dilatation and intralesional steroid injection are usually indicated in cases with partial obstruction with moderate mucosal injury [1, 5, 22]. However, data of endoscopic management is lacking in children [1]. Erdogan et al has reported successful management of a partial pyloric obstruction by endoscopic balloon dilatation [1, 3]. Pyloroplasty has been reported to be safe, simple and fast procedure for partial GOO in children in many studies [1,4,8,9, 23]. However, there are reports of recurrence requiring surgical intervention [1, 8].

The two procedures commonly performed for complete GOO are gastroenterostomy and gastrectomy [1]. Ozcan et al and Chaudhary et al have reported gastrojejunostomy for complete GOO in children with good long-term results [1, 2, 24]. Gastrojejunostomy is simple, safe and usually has good outcomes in cases with poor nutrition status, extensive perigastric adhesions and unhealthy duodenum [1,5,24,25,26].

Gastric resection is usually reserved for extensive gastric cicatrization [5]. However, it becomes a major surgery in nutritionally depleted patients and has its associated morbidities [5]. Many surgeons have doubts about the risks and benefits of gastric resection in children [1]. Kaushik et al, Ciftci et al, Tekant et al and Erdogan et al have reported Billroth I procedure in children with good results [1,2,8,9,27].

The decision of additional vagotomy and a drainage procedure is usually considered taking into account the diminution of acid and pepsin production due to damage of glandular elements [5,28].

Though rare, paediatric GOO after corrosive ingestion has good outcome with early diagnosis and appropriate management.

Conclusion

Corrosive ingestion is an important cause of significant morbidity, especially in developing countries. Parent education regarding storage of household corrosives away from the children, stringent legislation to curtail unrestricted access of adults to harmful corrosive chemicals and safe packaging of these chemicals in child proof containers with correct labeling are warranted. Early diagnosis and appropriate endoscopic and/or surgical intervention usually lead to a good outcome.

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