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Case Report

Gastric outlet obstruction without esophageal involvement: A late sequelae of acid ingestion in children

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ABSTRACT

Accidental corrosive ingestion is not a rare occurrence in children because of easy access to strong household cleansers. Gastric injury as a predominant finding following acid ingestion in pediatric age group is less widely known with a few cases being reported in Indian literature. We report two such cases of gastric outlet obstruction following accidental ingestion of dilute acid. Both children have typical clinical presentation just 4-6 weeks after the ingestion. Upper GI endoscopy and barium meal studies confirmed the diagnosis. Early surgical intervention resulted in a satisfactory recovery. Both patients are doing well on follow up.

KEY WORDS: Acid Ingestion, gastric outlet obstruction

INTRODUCTION

Corrosive injury to stomach without esophageal involvement is rare entity in children. A review of recent literature suggests that acid and alkali ingestion should no longer be considered collectively as corrosive injury due to a significant dissimilarity in their mode of action, site of injury, sequelae, early, and late management.[1] The ingestion of alkali primarily results in oropharyngeal and esophageal injury whereas acids tend to spare the esophagus and primarily results in gastric injury.[1-4] Absence of overt clinical and radiological signs at the time of acid ingestion usually results in a delayed diagnosis in such cases.[1,2] Upper GI endoscopy thus remains the diagnostic modality of choice and is also useful in planning the time and the type of surgical intervention, which remains the treatment of choice in such children.[5-7] To the best of our knowledge, corrosive pyloric obstruction in children has been discussed rarely in Indian literature till date. Thus, we report to such cases with the aim to highlight the role of early upper GI evaluation and surgical intervention in the management of such children with acid ingestion.

CASE REPORTS

Case 1

A 3-year-old boy was referred with complaint of persistent vomiting for the last 4 days. The vomiting used to occur 12-18 hour after taking meal and vomitus contained foul smelling undigested meal. There was a history of accidental ingestion of H_2So_4 on full stomach by the child 40 days ago, when child had symptoms of increased salivation, vomiting, decreased oral intake, and abdominal pain. Patient was managed conservatively at a primary health center and was discharged after 7 days. Patient remained asymptomatic during the intervening period except for anorexia and weight loss noticed by the parents. On physical examination, patient was mildly dehydrated, poorly nourished, with a normal vital signs and mild anemia. Oropharyngeal, abdominal, and chest examination revealed no abnormality. Laboratory investigations revealed Hb of 9 gm%, TLC 6000 mm^3, serum albumin 3 gm%, mild hypokalemia and a normal coagulation profile. Upper GI endoscopy suggested pyloric stenosis with a normal esophagus [Figure 1]. Patient was taken up for surgery after initial resuscitation aimed at the correction of fluid and electrolyte imbalance, nutritional support, antibiotic prophylaxis, and gastric lavage. Laparotomy revealed adhesions in the region of pylorus and pyloric antrum, a contracted and firm pylorus and distal pyloric antrum. The rest of the stomach was dilated and normal in consistency. There was partial obliteration of pyloric lumen with the presence of viable mucosa. Heineke-Mikulicz pyloroplasty was performed. Post-operatively, the recovery was uneventful. After

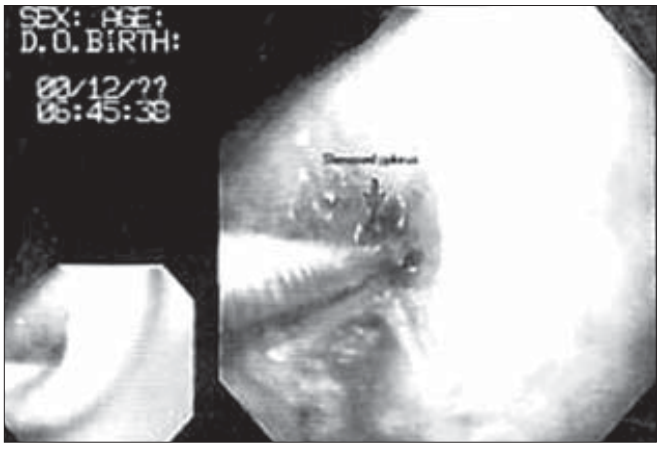

Figure 1: Upper GI endoscopy showing stenosed pylorus

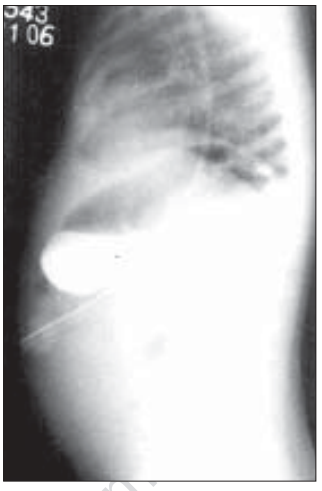

Figure 2: Contrast roentgenogram showing a normal esophagus with features suggestive of gastric outlet obstruction

eighth post-operative day, the patient started accepting orally and is doing well on follow up for the last 4 months.

Case 2

A 5-year-old girl presented with foul smelling vomitus of ingested food taken 12-18 hours before and decreased oral intake for the last 3 days. There was a history of accidental ingestion of dilute H_2So_4 (battery fluid) 6 weeks back when patient got treatment from a local hospital and remained asymptomatic thereafter. On examination, patient appeared to be mildly dehydrated, moderately nourished, hemodynamically stable, and clinically anemic. Oropharyngeal, abdominal, and chest examination revealed no abnormality. On laboratory investigation, patient had Hb of 8 gm%, TLC 8000 mm^3, mild hypokalemia, albumin of 3 gm% and a normal liver function test. Chest X-ray revealed no abnormality. Barium studies of upper GI tract revealed gastric outlet obstruction with a normal esophagus [Figure 2]. Upper GI endoscopy revealed stenosis in the region of pylorus and pyloric antrum. After pre-operative preparation patient was taken up for laparotomy. Intra-operative findings revealed fibrosis and contraction of pylorus and distal pyloric antrum. Rest of the stomach was dilated and normal in consistency. Duodenum and small gut were normal. Heineke-Mikulicz pyloroplasty was performed. Post-operative period remained uneventful and patient was discharged in a satisfactory condition. For the last 8 months, she is asymptomatic and gaining weight.

DISCUSSION

In 1828, Robert reported the first case of corrosive induced pyloric stenosis,[1] Ciftci *et al*. reported gastric outlet obstruction in 5% of cases following corrosive ingestion.[8] Gastric outlet obstruction is more common following acid ingestion although an incidence of 20% has been reported following ingestion of alkalis.[1]

Acids being commonly available in household items as toilet cleaners (sulfuric acid (H_2So_4), hydrochloric acid (HCl)), antirust compounds (oxalic acid ($H_2C_2O_4$), HCl), battery fluid (sulfuric acid (H_2So_4)), etc. makes them easily accessible to the kids, thus resulting in high incidence of accidental ingestion by children.[1,3] The clinical manifestations and thus the surgical implications of acid ingestion depends upon several factors including the surrounding circumstances, alike the nature, quantity and concentration of the agent, physical state, duration of exposures and post-prandial state of the child patient.[3] Due to contact burns and bitter taste, concentrated acids are usually vomited out thereby producing a little damage to esophagus and stomach.[2] Gastric injuries usually follows ingestion of dilute acids.[1] Once ingested the acids due to low viscosity and low specific gravity have a rapid transit which results in a superficial action on the resistant squamous epithelium of the esophagus.[1,2,8] The resulting pylorospasm induced by the presence of acid in stomach results in a maximum damage to pylorus and pyloric antrum.[3] A diffuse injury results following its ingestion in empty stomach.

The pathological changes vary widely. Grossly, stomach appears to be firm, contracted, and nodular. The lumen may be completely obliterated. In most of the cases, the only change is scarring of the pyloric antrum or the pyloric ring with surrounding adhesions. Microscopically, the acute phase is characterized by edema, inflammation, and thromosis of sub-mucosal vessels resulting in local necrosis and gangrene. Early reparative phase follows by the end of 1st week and is characterized by chronic inflammatory cell infiltration. Scar formation usually start from third week and is completed by sixth week.[1] The resulting fibrosis and adhesions formation during this phase results in obliteration of the lumen thus resulting in the characteristic manifestation of gastric outlet obstruction. More diffuse injury with resulting fibrosis

and destruction of nerve plexus results in squamous metaplasia and altered motility of the stomach.[1]

Clinically patients presents with severe local burning of lips, mouth, and pharynx immediately following the ingestion.[1,2] Nausea, vomiting, anorexia, and hematemesis usually continue for 24-48 hrs.[1-3,8,9] Patient usually responds to conservative treatment in the acute phase, although anorexia persists for one to two weeks. After a latent period of 4-6 weeks, patient again presents with complaints of persistent vomiting, early satiety and post-prandial fullness.[1,2,8,10] Vomiting, rapid loss of weight, and decreased oral intake remain the most notable features in children. The latent period of even 5-6 years has been reported in literature.[1]

Evaluation of upper GI tract by endoscopy confirms the diagnosis, the extent and the severity of injury.[3,5] Upper GI contrast studies too remains a useful diagnostic tool in patients with associated esophageal stricture.[9] Early surgical intervention remains the treatment of choice with the aim to restore normal passage for food from stomach to small intestine. The type of surgery depends upon the patient's character, ability, and judgment of the surgeon and above all, the findings at laparotomy.[1] As experienced in the present case partial obstruction with moderate mucosal injury usually responds to pyloroplasty. A number of complication reported with gastrojejunostomy makes partial gastrectomy with Billroth I reconstruction remains the procedure of choice in patients with complete luminal obstruction, deep ulceration, eschar formation, and non-viable mucosa.[6,7] A recent review of literature suggests that endoscopic balloon dilatation with or without intra-lesional steroid injection proves to be useful in a few cases but the long-term results are still awaited.[3,11]

In present cases, both patients presented with typical, clinical manifestations of gastric outlet obstruction, and had a normal esophagus on upper GI endoscopy and contrast studies. An early surgical intervention resulted in a satisfactory recovery in both the cases thus avoiding the repeated attempts at endoscopic dilatation, which is usually not feasible in developing countries like ours due to scarcity of resources, facilities and the low socio-economic status.

As antral-pyloric strictures and gastric outlet obstruction develops even in the absence of overt clinical and radiological signs of potentially severe upper GI tract pathology we recommend a thorough upper GI endoscopic evaluation in all children with acid ingestion with the aim to have satisfactory recovery by early surgical intervention.

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