

Gastric Outlet Obstruction Due to Isolated Pyloric Stenosis Following Corrosive Acid Ingestion: A Case Report

Authors

Dr. Shubhankar Mishra, Dr. Bhagabat Swain

Dept. of Pediatrics, M.K.C.G Medical College Berhampur, Odisha, 760004

Email: *dr.subham.scb@gmail.com*

Abstract:

Corrosive poisoning is a common thing in children .But isolated gastric outlet obstruction after one month of asymptomatic ingestion of some unknown liquid is a rare phenomenon and rarely reported. We report a case in which isolated gastric outlet obstruction is seen after one month of drinking some liquid from a battery. At the time of poisoning she was asymptomatic, but subsequently she had intractable vomiting with abnormal bowel movements.UGI endoscopy confirmed diagnosis of acquired pyloric stenosis. Early surgical management showed a satisfactory recovery.

Key Words: Gastric outlet obstruction, intractable vomiting , corrosive poisoning

INTRODUCTION:

Most of the times corrosive poisons cause severe local reactions then esophageal strictures and finally gastric injury. It's always said that "acid spares esophagus and eats pylorus." In case of alkali esophagus injury is more predominant. Still both produce severe injury to upper G I T .Very

rarely acid may cause only late complication like antral stricture and stenosis. In this type of cases diagnosis is very difficult due to no symptoms at the time of poisoning , and history is always biased. Mostly they present with intractable vomiting, altered bowel movements .An upper GI endoscopy always helps in diagnosis .This type of

cases rarely reported .We report this case to enlighten isolated GOO after an asymptomatic corrosive poisoning .

CASE REPORT:

A 5 yr female child presented with intractable vomiting for 15 days. She was apparently alright 15 days back. To start with she developed severe vomiting which was 7 to 10 times a day and the vomitus was around 150 ml in each bout, non projectile , non bilious with food particles .The frequency of stool passing was reduced to 2 to 3 times each week. Urination was decreased. No other history of fever ,jaundice ,epigastric pain haematemesis, melaena, visual disturbances ,seizure ,headache, fall etc.

About one month back there was a history of hospitalization for accidental ingestion of some unknown liquid which later found to be the liquid used in batteries/distilled water ,then admitted to the hospital for observation to rule out any unknown poisoning, but the incidence was purely asymptomatic and she was discharged after 24 hours of observation with an antacid gel and was advised for regular follow up . There was no significant anomaly in family, developmental and diet history. She was immunized according to age.BCG scar was present on left deltoid region.

On examination patient was, conscious, eyes sunken, severe lethargic ,dry tongue, skin pinch returning very slowly, not feeding well, scanty urination and was afebrile. Heart rate was 130 per min. with all other vitals normal .She had mild pallor no icterus.NG tube aspiration showed non

bilious fluid. Abdominal examinations revealed no organomegally. All other systemic examinations revealed no anomaly. After initial correction of severe dehydration and emesis, and keeping nil per orally patient was apparently asymptomatic for 3 days. But on following up repeatedly on 3rd day as patient was stable she was given some liquid diet peristalsis in epigastrium seen and was followed by vomiting.

All investigation showed HB was 9 gm%, N50 E2 L48, TLC was 9800,TPC was 2.3 lakh ESR 05 , serum sodium 139mg/l, potassium 3.4mg/l ,sr.calcium 8.8 mg/dl sr.protein 5.2 mg/dl and serum ca. was 8.8 m/dl sr.urea was 40m/dl s.creat was 1.2mg/dl SGPT was 46 and SGOT was 58 mg/dl .Urine routine and microscopy was within normal range with some phosphate crystals.The plain erect x-ray of abdomen had no anomaly and the ultrasonography of abd and pelvis no anomaly.On upper G.I endoscopy the stomach was grossly edematous ,antrum edematous and inflamed with slough covering all the walls with reduced distensibility and pylorus is grossly spastic.

DIAGNOSIS AND MANAGEMENT:

The patient was diagnosed to be a case of gastric outlet obstruction due to acquired pyloric stenosis and antral stricture most probably due to corrosive acid ingestion induced acquired pyloric stenosis . She was sent to pediatric surgery department and surgery was planned. Laparotomy revealed strictures in pylorus .Heineke Mickulitz pyloroplasty was done. Post operatively patient

was uneventful. On 8th post operative day she was taking oral liquid diet and bowel bladder movement was normal.



Fig-1(pre operative condition)



Fig-2(visible antral stricture in O.T)



Fig-3 (patient on follow up)

DISCUSSION:

The first corrosive induced antral stenosis was reported in 1828^[1]. The incidence of antral stenosis was 5% of all the corrosive poisoning^[2]. Acid injures stomach more than alkali. Pathologically the stomach is firm, contracted the lumen may be obliterated completely or incompletely. Microscopically the edema accompanied by inflammation of sub mucosal vessels causing local necrosis and gangrene. Later the reparative phase occurs which causes chronic inflammatory cell intrusion. The scar formation occurs 3rd to 6th week, which completes by fibrosis and strictures later^[2]. Acid has a tendency to lick the esophagus and bite the pylorus^[3]. It can be explained by following explanations

1: Acid has less specific gravity which causes rapid transit of acids through esophagus which causes rapid transit of acids through the esophagus so does less damage to it.

2: Antral spasm occurs more commonly after acid ingestion.

3: Squamous mucosa of esophagus is less resilient to acid than alkali^[3].

The injury also depends upon several other nature of the corrosive substance, quantity ingested, its concentration, duration of exposure, the act of swallowing, the existing condition of the tissues that came in contact with the corrosive agent^[4]. The dilute acid has more chance of doing isolated antral stenosis. According to a study of 220 children of corrosive poisoning 52 patients took acid agents. Among them only 2 had isolated pyloric stenosis. So isolated acquired pyloric

stenosis is around 3.8% of all the acid ingesting people^[5]. Vomiting ,rapid loss of weight and decreased oral intake of fluids are cardinal features of acid ingestion .But in some cases only pain in epigastrium and severe vomiting after 4-6 wk are major complaints. In some literatures a latent period of 5-6 years can be seen.

CONCLUSION:

In developing countries like india accidental ingestion of acid is very common. Sometimes patients, like this child come to pediatric emergency with history of some unknown liquid ingestion. Even if the ingestion is asymptomatic long term complication of pyloric stenosis should be kept in view. In literatures isolated pyloric stenosis is scantily mentioned. This reporting of unknown liquid ingestion with asymptomatic presentation and later isolated antral stricture will help to throw light on this type of long term complication of dilute acids.

REFERENCES:

- 1.Howard KG,Holmes CL.Pyloric stenosis caused by ingestion of corrosive substances:Report of case.Surg clin North Am 1948:28:1041-56
- 2.Sharma, S., Debnath, P. R., Agrawal, L. D., & Gupta, V. (2007). Gastric outlet obstruction without esophageal involvement: A late sequelae of acid ingestion in children. Journal of Indian Association of Pediatric Surgeons, 12(1).

3. Shukla, Ram Mohan, et al. "Pyloric and antral strictures following corrosive acid ingestion: A report of four cases." Journal of Indian Association of Pediatric Surgeons 15.3 (2010): 108.

- 4.Chibishev, A., N. Simonovska, and A. Shikole. "Post-corrosive injuries of upper gastrointestinal tract." Contributions of Macedonian Academy of Sciences & Arts 31.1 (2010).

5. Ciftci, A. O., et al. "Gastric outlet obstruction due to corrosive ingestion: incidence and outcome." Pediatric surgery international 15.2 (1999): 88-91.