PYLORIC STENOSIS CAUSED BY INGESTION OF CORROSIVE AGENTS : A CASE REPORT

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ABSTRACT

Corrosive or caustic is a group of chemical which has the capacity to cause tissue injury on contact by a chemical reaction.Ingestion of a substance that causes a chemical reaction with mucosal tissue and injures the gastrointestinal and respiratory tract is caustic ingestion. Accidental ingestion of caustic agents may cause devastating injury in children. Eighty percent of corrosive poisoning occurs in children below five years. Children are particularly susceptible to the accidental exposure to such substances due to inadequate parental supervision and careless storing of these chemicals at homes. Pyloric stenosis or gastric outlet obstruction is a well-known complication of caustic acid ingestion. The stenosis mostly occurs at the pylorus and antrum due to the pooling of the corrosive agent and the reflex pyloric spasm of the stomach after the ingestion. Typically corrosive acid ingestion leads to local reaction, oesophageal damage and gastric injury in that order. Isolated injury to the stomach resulting in pyloric stenosis is very rare, accounting to as little as 3,8% of all the cases of corrosive ingestion as reported in literature. A proper management including surgery is needed in this case to treat a child with pyloric stenosis caused by ingestion of corrosive agents. Conclusion: stricture pylori is late sequele complications caused swallowed corrosive substances and has a good prognosis with proper governance

***Keyword** : Pyloric stenosis, accidental ingestion, corrosive agents.*

ABSTRAK

Korosif atau kaustik adalah sekelompok bahan kimia yang memiliki kapasitas untuk menyebabkan cedera jaringan jika kontak dengan reaksi kimia. Penyumbatan zat yang menyebabkan reaksi kimia dengan jaringan mukosa dan melukai saluran cerna dan pernapasan adalah menelan kaustik. Penelanan agen kaustik yang tidak disengaja dapat menyebabkan cedera parah pada anak-anak. Delapan puluh persen keracunan korosif terjadi pada anak-anak di bawah lima tahun. Anak-anak sangat rentan terhadap paparan yang tidak disengaja terhadap zat-zat tersebut karena pengawasan orang tua yang tidak memadai dan penyimpanan bahan kimia ini secara sembarangan di rumah. Stenosis pilorus atau obstruksi saluran keluar lambung adalah komplikasi umum dari konsumsi asam kaustik. Stenosis sebagian besar terjadi di pilorus dan antrum karena penyatuan agen korosif dan spasme refleks pilorus pada perut setelah menelan. Biasanya konsumsi asam korosif menyebabkan reaksi lokal, kerusakan esofagus dan cedera lambung dalam urutan itu. Cedera terisolasi pada perut yang menyebabkan stenosis pilorus sangat jarang terjadi, terhitung 3,8% dari semua kasus konsumsi korosif seperti yang dilaporkan dalam literatur. Penatalaksanaan yang tepat termasuk pembedahan diperlukan dalam kasus ini untuk merawat anak dengan stenosis pilorus yang disebabkan oleh konsumsi agen korosif. Kesimpulan: striktur pylori merupakan komplikasi sekuel lanjut yang disebabkan oleh zat korosif yang tertelan dan memiliki prognosis yang baik dengan tata kelola yang baik.

Kata kunci: *stenosis pilorus, accidental ingestion, agen korosif.*

Introduction

Corrosive or caustic is a group of chemical which has the capacity to cause tissue injury on contact by a chemical reaction. They most commonly affect the gastrointestinal tract (GIT), respiratory system and eyes. Acids and alkalis are the

two primary types of agents most often responsible for caustic ingestion.[1]

Accidental ingestion of caustic agents may cause devastating injury in children. These account for a large number of accidental and intentional poisonings. Eighty percent of corrosive poisoning occurs in children below five years.[1] Children are particularly susceptible to the accidental exposure to such substances due to inadequate parental supervision and careless storing of these chemicals at homes.[2]

Pyloric stenosis or gastric outlet obstruction is a well-known complication of caustic acid ingestion. The stenosis mostly occurs at the pylorus and antrum due to the pooling of the corrosive agent and the reflex pyloric spasm of the stomach after the ingestion. Typically corrosive acid ingestion leads to local reaction, oesophageal damage and gastric injury in that order. Isolated injury to the stomach resulting in pyloric stenosis is very rare, accounting to as little as 3,8% of all the cases of corrosive ingestion as reported in literature.[3]

CASE REPORT

A boy,JA, 4years and 9 month old came to emergency department of Dr. M. Djamil Hospitalwith chief complain Reccurent vomit since 1week before admission. Children was ingested of accu water ± 20 days before admission, amount was± 1/4cup and then he vomit mixed with blood. Previously, the child was treated in Padang Panjang Distric Hospital for 4 days. During the treatment hewas still often vomit mixed with blood for 1 day, but after treatment he was taken home in stable condition.Child also complaining about abdominal pain intermittently since ± 20 days before admission especially in epigastrium region. There was no history of abdominal trauma. Child had vomit again since ±1 weeks before admission, frequency 4 - 5 times/day, the amount was 5 tablespoons until a quarter of glass/times, especially after eating. Vomit was not mixed with blood accompanied by pain in swallowing. Sometimes ½-1 hour after eating a child would spit back some food.No fever, breathlessness, seizure and chest pain.There was no decrease of body weight. Mixturition and defecation was within normal limit.

Patient isthe first child of two siblings, caesarean delivery assisted by obstetrician, aterm,with birth body weight 2800 grams, body length 49 cm, directly cried. Basic immunization wascomplete, BCG scar was present. Booster and non PPI immunizations does not exist. History of growth and development was normal. Hygiene and sanitation was good.

On physical examination Patient look moderately ill, alert, blood pressure 100/70 mm Hg, pulse rate 100 times per minute, respiratory rate 24 times per minute,body temperature 36,9°C. No pallor, edema, jaundice and cyanosis. Body weight 16 kg, (<P50 curve CDC-NCHS 2000), body height 101 cm (<P50 curve CDC-NCHS 2000). Genetical height potential (GHP): 154-171 cm. (the patient's body height between GHP). Weight for age (W/A) 94%, height for age (H/A) 97%, weight for height (W/H) of 100%, the impression is well-nourished. Skin was warm, turgor return quickly.No enlargement of lymph nodes. Head round symmetrical. Black hair, didn't easily fall out. The eyesdidn't look sunken, conjunctiva was not pale, sclera was noticteric, pupil was isokor with diameter of 2 mm, light reflex was positive normal. Tonsils T1-T1 was hyperemic, posterior pharyngeal wallwas hyperemic. There was no ulcus in mucous of the mouth.Lip was wet. JVP was 5-2 cmH2O. On lung examination, normochest, symmetrical, no retraction, breath sounds vesicular, there was no rales and wheezing. Heart: regular rhythm, no murmur. Abdomen: no distension, there was tenderness in epigastrium, liver and spleen not palpable, turgor was return fast, percussion timpani, bowel sound was normal. Genitalia: no abnormality, pubertal status A1P1G1.

Acral was warm, no cyanosis, no edema, capillary refilling was good, normal physiological reflexes and there is no pathological reflexes.

Laboratory finding Haemoglobin 13,1 g/dl, leukocytes 6,600/mm^3, differential count 0/1/0/54/37/8, platelets 546,000/mm^3.sodium: 135 mmol/L, potassium: 3,8 mmol/L, calcium: 11 mg/dl, random blood glucose 95 mg/dl The impression within normal limits. Urine and feces within normal limits.

Based on those finding, patient was diagnoses suspect esophagitis and corrosive gastritis due to acid ingestion (accu water) with differential diagnosis suspect acute gastritisor peptic ulcer.

Patient was treated with Total parenteral nutrition IVFD D 12,5% + electrolyte 47/hours + aminofuchsin ped 5% 6 cc/hours, Ranitide (IV) 2 x 15 mg, Omeprazole (IV) 1 x 15 mg, Ondancentron (IV) 3 x 2 mg and plan to perform abdominal xray and endoscopy. The result of esophagogastroscopy waspyloric stenosis, they found cicatrix in prepyloric.

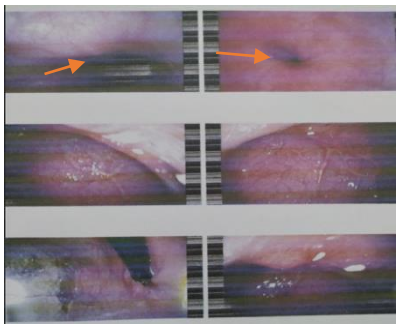

Figure 1. endoscopy result. Red sign ( ) show pyloric stenosis

On 8th day during inwarded, pediatric surgeon performed abdominal x-ray with air insertion 100 cc by nasogastric tube (NGT). With result partial obstruction on pylori duodenal suspicious pylori stenosis. The child was planned to performed bypass gastro duodenostomy operation.

Condition after surgery there was no fever, shortness of breath, seizures, vomiting and bleeding. Pain at the incision

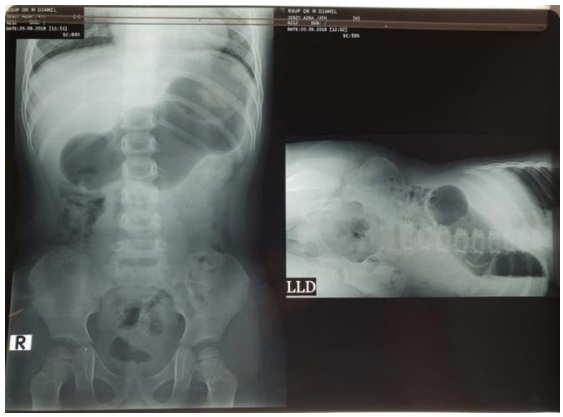

Figure 2. Abdominal X-ray after given 100 cc air via NGT

was present. Urinationwas in sufficient quantities, diuresis 3,6 cc/kg/hr. Moderately ill, conscious, pulse rate110 times per minute, respiratory rate 22 times per minute, body temperature 36,5ºC, conjunctiva was not anemic, sclera was noticteric. There was no abnormalityfound on heart and lungs, no abdominal distension, surgical woundbandages was closed, bleedingdid not exist, Extremities was warm with good perfusion.

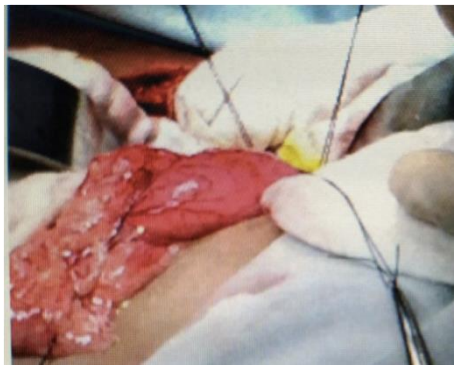

Figure 3. Bypass Gastroduodenostomy Operation

Patient inwarded in PICU for one day. Five day after surgery, Patient was tried to drink liquid meal step by step with good tolerance. 10 days after surgery, patient allowed to go home.

DISCUSSION

We present a case of a 4 year 9 month old boy with pylory stenosis due to caustic acid ingestion (car battery water/ accu water). Diagnose was made based on

history of ingestion of accu water, non billous vomiting especially after eating. On physical examination we didn't found sign and symptom of dehydration. Vomiting and decreased oral intake remain the most notable features after acid burns in children. Nausea, vomiting, anorexia and hematemesis usually continue for 24-48 hours. Patient ussually respons to conservative treatment in the acute phase, although anorexia persist for one to two weeks. After latent period of 4-6 weeks, patient presents again with complaints of persistent vomiting, early satiety and post-prandial fullness.[4,5] In our patient we found vomit mixed food at the first time hospitalized and resolve with symptomatic therapy.

Vomit was reccurent 2 weeks after the incident and he has difficulty to eat but didn't have any dehydration symptomps. Shukla was reported a case in 4 children with history of ingestion toilet cleaner (hydrocloric acid). They was treated with conservatively teraphy for 3 to 6 weeks, but three patient presented non bilious vomit after each feed, decreased of body weight and dehydration after that.[6]

Endoscopy is an effective technique for determining the presence of esophageal and gastric damage and to avoid unnecessary treatment in patients with no or mild injury.[7]In this case, we performed endoscopy and turn out that its really helpful to diagnose patient accurately. In our case we found stenosis in pyloric.

Gastric outlet obstruction has an incidence of 5%, mainly in the prepyloric area, where prolonged contact with the antral mucosa due to pyloric spasms and to resulting pooling of the caustic agent in this region usually results in stricture in more than 60% of patients.[8]

Pyloric stenosis without esophageal involvement is rare but it is a well-known complication of corrosive acid ingestion. There is a tendency of acids to lick the esophagus and bite the pyloric antrum. The esophagus is spared because of the relative resistance of the squamous epithelium to acid and shorter contact time due to rapid transit of the acidic corrosive agent. Once in the stomach, the acid tends to follow the natural curve of the lesser curvature to pool in the antrum. This pooling is aggravated by reflex pylorospasm, therefore the gastric burns mostly occur in the anthrum and pylorus.[9] In our patient, there was pain in swallowing. On physical examination, we found hyperemic in tonsil and posterior pharyngeal wall. Literature said that depending on the injured area, pain may arise in the oropharynx.[2] This may caused odynophagia in this patient and hyperemic in physical examination.

Plain radiograph of chest and abdomen and water soluble contrast examination of the upper GI tract may be required during the acute phase to look for perforation.[10] We found gastric dilatation and there was an air in distal of gastric but in limited volume. There was distribution of bowel air until distal part of rectum. There was no air fluid level and free air with conclusion; partial obstruction on pylori duodenal suspicious pylori stenosis.

The degree of mucosal injury depends on the nature, the concentration, amount of acidic substance as well as the length of time in the stomatch, the amount of food in the stomach at the time of ingestion and the mode of ingestion.[9] Strong acids reaching the stomatch may cause perforation in 24-48 hours if a large volume and if the organ is empty. The perforation case 2 hours after ingestion in children 2 years old was reported in Turkey[11]and in children 3 years old who accidental ingestion of HCL in Pakistan.[12] Our patient suggest suffered in grade 2 or 3 injury. Based on literature patient with grade 2 or grade 3 injury has the greater risk for development complications such as strictures or perforation. [9]Nagi et al [10]also said that most patient with grade 2B and 3 injury develop strictures, while those with grades 1 and 2A injury recover without sequelae. The chances for the development of the stricture is about 10-

30% in the grade 2 injury and about 40-70% in the grade 3 injury.[13,14]

Extensive damage of the gastrointestinal tract hinder physiological nutrition in these patients. Nutrition is life-maintaining therapy in patients who cannot take food and are disposed to a risk of malnutrition. Documented effects from artificial feeding in patients intoxicated with corrosive substances are reduction of infections, reduction of predisposition to developing aspiration pneumonia, reduction of the risk for pulmonary embolism, economical reasons. The type of the artificial nutrition depends on the degree of esophageal or gastric damage seen by endoscopy. In patients with 1 and 2A degree of damage, total parenteral nutrition in the first 24–48 hours is followed by a liquid diet until the 10th day. Afterwards, food intake can be in a more liberal regimen. Some authors recommend taking liquids (liquid nutritional solutions, milk) 48 hours after ingestion if the patient can swallow his/her saliva. In patients with 2B and 3 degree of damage the so-called "esophageal rest" is recommended, that is the patient must not take food per os. During the "rest", the patient is fed by nasogastric or nasoenteral tube, gastrostoma or jejunostoma and parenterally by peripheral or central vein. This is explained by the fact that food particles enter the granulocytes of the esophageal wall and exacerbate the inflammation. Esophageal rest may last until the 10th day after corrosive ingestion or some authors say until the 15th day, that is until the first endoscopic control.[5,16]In our case, the child came in late sequele and he ate regular meal but vomit soon after eat. We suggested to ate a liquid meal with small volume and frequent times. We evaluated the tolerance intake and gave an additional calori by parenteral nutrition.

Gastric acid suppression with proton pump inhibitor and H2-antagonists are often used in corrosive burn injury as oesophagitis and gastritis are common and patients have been kept fasting.[14] This treatment has been employed in our patients in order to suppress gastric acid production and to prevent stress ulcers in the stomach. The choice of definitive surgery for corrosive gastric outlet obstruction was determined by the extent of cicatrisation of the stomatch and general condition of the patient. The main aim of surgery in corrosive pyloric stenosis is the relief of obstruction with reasonable gastric volume. For short segmen stricture in distal stomatch, stricturoplasty or pyloroplasty, either Heineke-Mikulicz type or Y-V flap can be done. In cases of severe stricture and loss of gastric volume, gastrojejunostomy was preferred. To prevent bile reflux, Roux-en-Y gastrojejunostomy was preferred. [17]

Timing of surgery is controversial, but early surgical intervention remain the treatment of choice.[6]In our patient, we performed operation on day 15th after inwarded or, day 35th after ingestion of corrosive agents.

CONCLUSIONS

This paper is based on the report of a case in which pyloric stenosis developed about 20 days after the ingestion of accu water. Corrosive acid ingestion leads to local reaction, oesophageal damage and gastric injury in that order. Isolated injury to the stomach resulting in pyloric stenosis is very rare, accounting to as little as 3,8% of all the cases of corrosive ingestion as reported in literature.

Plain radiograph of chest and abdomen and water soluble contrast examination of the upper GI tract may be required during the acute phase to look for perforation. But endoscopy is an effective technique for determining the presence of esophageal and gastric damage and to avoid unnecessary treatment in patients with no or mild injury.

Nutrition is life-maintaining therapy in patients who cannot take food and are disposed to a risk of malnutrition. Documented effects from artificial feeding in patients intoxicated with corrosive

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