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Corrosive Acid (Vixol®) Ingestion, Spectrum of Injury and Management

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Abstract: **A total of 10 patients with definite history of intentional corrosive acid ingestion, during the past 12 years at Taksin Hospital, were reported focussing on the spectrum of injury, recognition of the deep injury and its managemant. Most of the cases (9 out of 10 patients) were esophageal injury. Isolated involvement of esophagus and stomach were present in 3 and 1 patients, respectively. In 6 patients both were damaged. These findings are contrary to the general belief that the esophagus is spared in acid ingestion. The common site of esophageal injury was at 20 and 30 cm. from incisor and just above esophagogastric junction. Periodic dilatation of esophagus may require more than 2 years in many cases and the outcome of these patients must depend on the compliance of patients and their families. Such compliance problem was overcome after the use of "Soft & Self balloon dilatation" in Taksin Hospital and esophageal reconstructive surgery were avoided in case of multiple sites or long segment stricture. False tract that was recognised during dilatation responded well to conservative treatment. The treatment of choice for pyloric stenosis is Billroth I gastrectomy.**

Swallowing of corrosive substances, either accidentally or intentionally, is one of the leading cause of severe injuries to the upper gastrointestinal tract. Accidental swallowing occurs mainly in children. The suicidal adult who intentionally ingests a large volume of corrosive material is more likely to suffer serious injury than a child who erroneously swallows it.

Alkaline corrosives are the most commonly implicated. Commonly are sodium hydroxide (lye), potassium hydroxide and drain cleaner (a mixture of both). The spectrum of injury to the upper gastrointestinal tract has been well studied[1,2].

Strong acids are also capable of producing severe injuries when swallowed. They induce a coagulation-type necrosis by precipitation of proteins, forming an impermeable layer that prevents penetration into the deeper layers of tissues. Gastric lesions are also resulted with greater frequency[3]. The most frequently swallowed acids are hydrochloric, sulfuric, nitric and oxalic acids.

The suicidal or intentional ingestion of strong acids is not uncommon in Thailand. Vixol® (22% hydrochloric acid W/W) are sold over the counter as toilet cleaner, and the Vixol pink® with less irritated odour are widely used and easily accessed.

The purpose of this report is to review our experience with Vixol pink® ingestion at Taksin Hospital during the past 12 years, focussing on the spectrum of injury to the upper gastrointestinal tract, its management and outcome.

Materials and Methods

A total of 10 patients with definite history of Vixol

pink· ingestion were treated at surgical department between May 1985 and June 1997. All had early evaluation and treatment at medical department or at other hospitals. They were referred to surgical department in the second week or later because of its complication. There were 2 men (age 22 and 23 yr.) and 8 women (age range 14-29 yr.). The exact volume of corrosive acid consumed was difficult to ascertain in each case, but apparently 50-100 ml. was ingested. Ingestion was intentional for attempted suicide in all of them.

Results

Dysphagia or odynophagia and vomiting were the two most frequent problem that referred the patients to surgical department.

The assessment of extent of injury to the upper gastrointestinal tract was made with fiberoptic or videoscope and x-ray imaging (barium swallowing and GISM)

The timing to come to surgical department, findings, complications and management were shown in Table 1.

Of the 10 patients, the stomach and esophagus were both damaged in 6 patients, pyloric stenosis occurred in 7 patients with associated duodenal injury in one patient. Isolated involvement of the esophagus and stomach was present in 3 and 1 patient, respectively. The most serious injuries involved esophagus, pylorus and duodenal, with confined perforation of fundus of stomach.

Those with pyloric stenosis (Figure 1) underwent Billroth I gastrectomy except Case 6 who had extensive adhesions of posterior wall of stomach to pancreas and Case 9 who had positive anti HIV and both of them received gastrojejunostomy.

All patients with esophageal stricture were managed with periodic dilatation (with Eder-Puestow's dilator). The patients with short stricture, only narrow ring or less than 2 cm long as (Cases 2 and 3) responded well to periodic dilatation with complete epithelization after 6 months follow up. Of those with stricture (more than 2 cm. long) and usually with marked cicatrization (Cases 1,5,7 and 9), required frequent periodic dilatation extended to more than 2 years (Figure 2).

The common sites of esophageal stricture are usually at about 20 cm and 30 cm from incisor or both sites in the same patient as (Cases 1, 7 and 9). At 20 cm from incisor site the injury was more intense with marked cicatrization and long segment or with multiple strictures (Cases 7 and 9, Figure 4). The false tract and perforation were accidentally made during dilatation in Cases 5,7 and 9 (Figure 4) due to tortuous tract and cicatrization of the stricture but all recovered unevenfully after conservative and supportive treatment. Gastrostomy was necessary in difficult cases (Cases 7 and 9) for nylon thread splinting (mouth to gastrostomy) to ease dilatation with Eder-Puestow's dilator as a rail-road tract.

The Soft and Self dilatation with the balloon of Foley catheter (Figure 5) was developed and used in Cases 1,7 and 9 after facing the neccessity of frequent dilatation and very anxious mood of the patient on standard dilatation with Eder-Puestow's dilator. The patients accept this self manuveure and the interval for esophagogastroscopy with standard dilatation can be postponed to longer than every 2 or 3 months.

The most extensive injury in case 6; confined perforation of fundus of stomach, necrosis of lower end of esophagus, pylorus with first part duodenum stenosis underwent total gastrectomy with feeding jejunostomy and later with Colon bypass operation (Esophagoileostomy at neck with jejunocolic and side to side jejunojejunostomy). Some slight stricture at esophagoileostomy developed postoperatively but responded well to Eder-Puestow's dilator and later with Soft and Self balloon dilatation. However, the

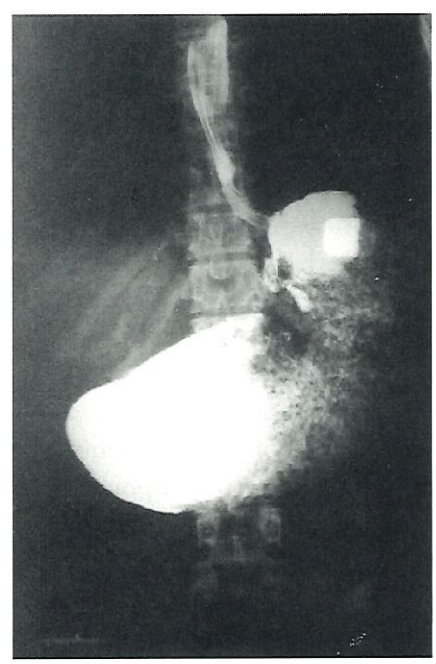


Fig. 1 Case 10: Upper GI series showing pyloric stenosis 6 weeks post injury.

Table 1 Clinical features, stricture sites and results of management in cases of corrosive acid ingestion.

Case	Sex/ Age	Time Post Injury	Findings	Operation	Complications	Results
1	F/29	2 yrs.	stricture at 20 cm.	- previous gastrectomy - dilatation 1 yr - *soft & self balloon dilatation*	nil	- satisfactory
2	M/23	6 months	stricture at EG junction	dilatation, 2 sessions	nil	- satisfactory
3	F/17	1 month	- linear ulcer at EG junction - pyloric stenosis	- Billroth I gastrectomy - dilatation, 2 sessions	nil	- satisfactory
4	F/16	1 month	- stricture at 25 cm.	nil	-	- against advice
5	M/22	6 months	- post treatment from many hospitals - status post gastrojejunostomy - stricture at 40 cm.	- dilatation multiple sessions	- false tract (recovered with conservative treatment)	lost to follow up
6	F/23	1 month	- stricture at 15-17 cm. - multiple ulcers along esophagus - rigidity of stomach with superficial ulcer - pyloric stenosis	- total gastrectomy with feeding jejunostomy - colon bypass (esophagoileostomy, jejunocolic and side to side jejunojejunostomy) - *Soft and self balloon dilatation*	- necrosis of lower end of esophagus and fundus of stomach with confined perforation - postopertive stricture of esophagoileostomy	- lost to follow up
7	F/14	3 weeks	- stricture at 20 and 30 cm. - scaring posterior wall of stomach - pyloric stenosis	- gastrostomy and feeding jejunostomy with gastrojejunostomy - dilatation, multiple sessions (>2 yrs.) via splinting as rail - road tract - *Soft & Self balloon dilatation*	false tract (recovered with conservative treatment)	- lost to follow up until times re-admitted in cachetic state - recovery well
8	F/21	4 weeks	- stricture at 20 and 30 cm. - pyloric stenosis	- exploratory thoracotomy and repair of esophagus with pleural flap - Billroth I gastrectomy - dilatation, multiple sessions (> 1 yr.)	dilatation with tear at 30 cm., 8 cm. long	- satisfactory after18 months dilatation
9	F/15	1 day	- hoarseness of voice stricture at 20 & 30 cm. with multiple ulcers in lower 1/3 - pyloric stenosis	- Billroth I gastrectomy - dilatation >2 yrs. - *soft & self balloon dilatation*	false tract (recovered with conservative treatment)	- lost to follow up until readmitted in cachetic state - recovered well
10	F/21	1 month	- pyloric stenosis - Anti HIV +	- Gastrojejunostomy	nil	satisfactory

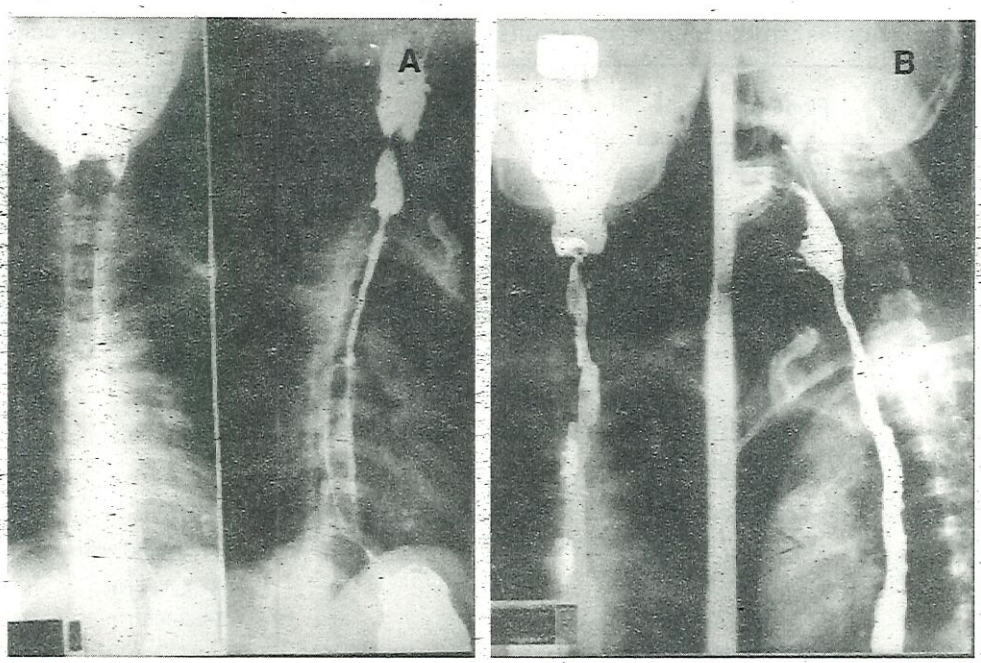


Fig. 2 Case 9 : Barium swallowing examination showing
A. Strictures at 20 and 30 cm with marked cicatrization.
B. Post periodic dilatation and later with Soft & Self balloon dilatation.



Fig. 3 Case 7 : Barium swallow examination showing long segment stricture.

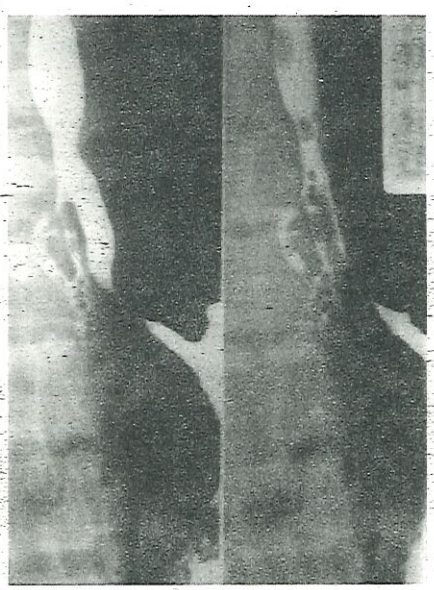


Fig. 4 Case 5 : Barium swallow showing a false tract.

patient was lost of follow up 6 month post colon bypass operation.

Discussion

The clinical manifestations of acid ingestion and, therefore, the surgical implication depend upon several factors. The circumstances surrounding the ingestion are important. Since acid causes immediate pain in the oropharynx when ingested accidentally, the agent is usually expelled rapidly and the actual amount swallowed may be minimal or none. Conversely, significant injury was noted in the esophagus and stomach with little or no oropharyngeal burns when acids were ingested intentionally. As the volume ingested was large, therefore, the presence of injury about the lips and oral cavity indicates only that a corrosive agent

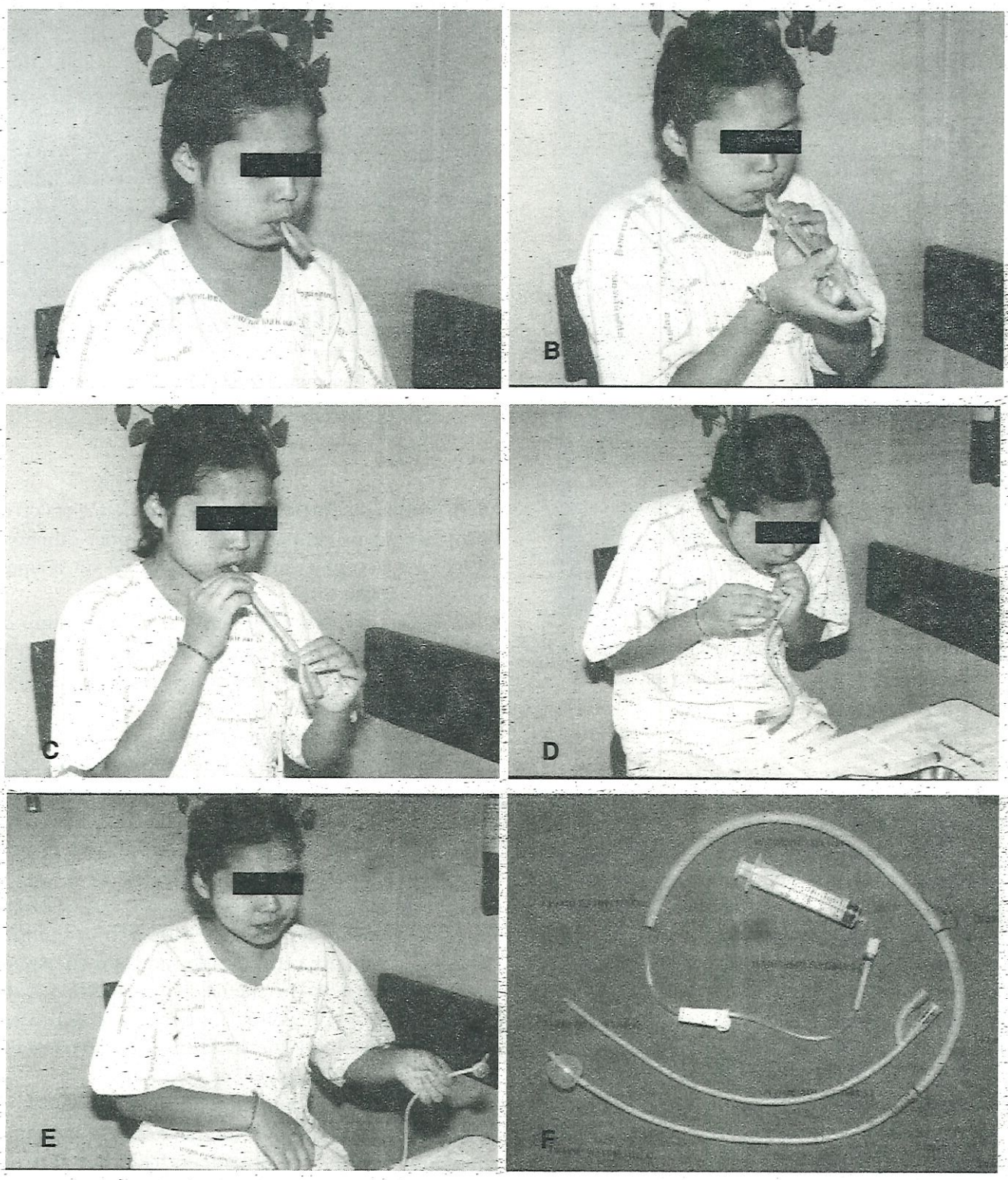


Fig. 5 Photographs showing Soft & Self balloon dilatation's procedure.

A. Insertion of Foley's catheter.
B. Inflation of catheter balloon.
C. Beginning of retrograde dilatation
D. Dilatation in progress.
E. Completion of dilatation.
F. Photograph of a modified Foley's catheter for stricture more than 25 cm from incisor.

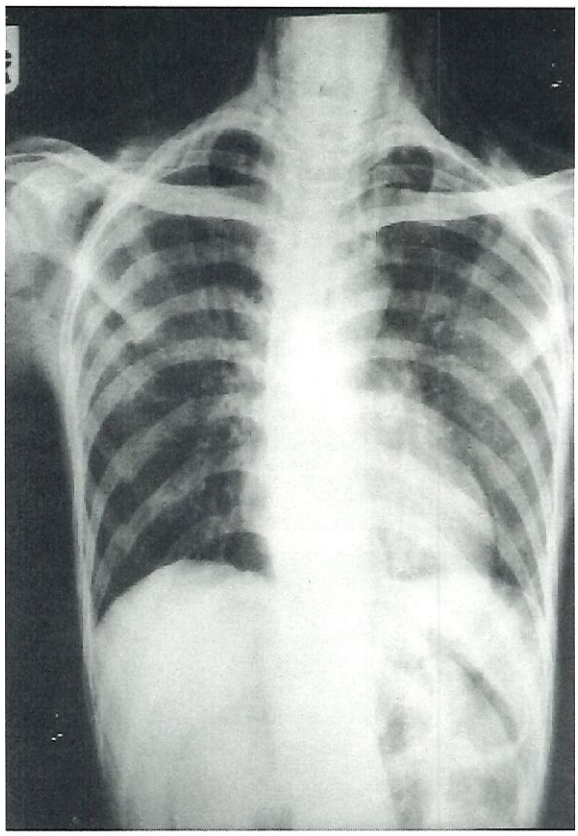


Fig. 6 Case 5 : Chest film showing subcutaneous emphysema recognized during esophagogastroscopy.

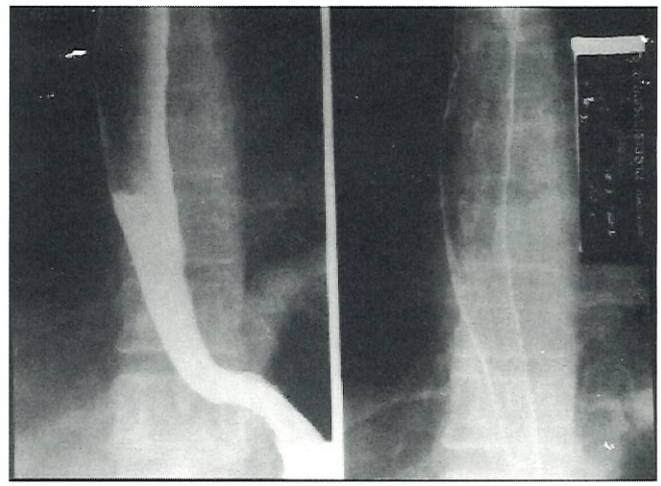


Fig. 7 Case 6 : Barium swallow showing almost complete loss of esophageal motility.

was taken orally but the extent of internal damage can not be quantitated.

The first description of pathologic findings after acid ingestion, as reported by McLanahan[4], was recorded by Robert in 1828 in a patient who swallowed sulfuric acid and died nine weeks later. The esophagus was normal but the stomach demonstrated ulceration and the pyloric area was markedly contracted. The esophageal sparing effect of acid has been attributed to rapid transit and the resistance of the esophageal squamous epithelium to acid digestions.[5] Although these description of pathologic finding after acid ingestion are well supported by many reports,[6,7] we found esophageal injury in a majority of our patients (6 out of 10 showed damages both in stomach and esophagus) and 3 patients had isolated esophageal involvement. These finding are contrary to the general belief that the esophagus is mostly spared in acid ingestion. Zargar et al[8] reported 87.8 per cent of patients with esophageal injury and Chanvitan[9] also reported 20 out of 24 patients with acid destruction in both esophagus and stomach. The incidence of corrosive esophagitis probably has been underestimated because the reports are few and based on a small number of patients[10].

Injury to the esophagus was maximum in cricopharyngeal region, the middle and lower thirds (Table 1). The injured sites correlated to the areas of relative stagnation were:-

- the cricopharyngeal area,
- the impression by the aortic arch and left bronchus,
- the area of the lower esophageal sphincter.

All perforation that were recognized during esophagogastroscopy and dilatation by the presence of subcutaneous emphysema (Figure 6) responded to conservative treatment without surgical intervention. This was due to early recognization and limited spreading of infection in the mediastinum resulting from fibrosis around previously injured esophagus after corrosive ingestion. These patients were successfully dilated after the healing of perforation.

The most difficult problem in managment of stricture esophagus is the compliance of patient and their families. All of these patients had some underlying psychological problems and stress. Many cases (Cases 6-9) were lost to follow up for more than 3 months after multiple sessions of dilatation and were apparently eating well. They came back in cachetic state with weight loss of more than 10 kilograms and only some liquid could be taken. Gastrostomy was necessary for feeding and for synchronous antegrade

esophagoscopy (per oral) and retrograde esophagoscopy (per gastrostomy) to localize and splint the tract for subsequent dilatations.

The problem of patient's intolerance to dilatation was overcome after the use of soft and self balloon dilatation in our hospital. The patient could do dilatation by self at home and at least twice a day. The interval for esophagogastroscopy and dilatation was extended to every two months or more in the previous problematic cases.

In most instances, the gastric injury was greatest in the lesser curvature and in the prepyloric area. This distribution could be explained by the results in the experiment by Testa[11] in 1938. When the acid reaches the stomach, it will follow the lesser curvature to the pylorus producing severe pylorospasm and corrosive acid pooling in the pre-pyloric area where the greatest injury occurs. The extent of gastric injury appears related to volume, concentration, length of time they remain in contact with the stomach and the content of the stomach at the time of injury. More extensive gastric injury involves the submucosa and muscularis layers producing various types of deformity including antral stenosis, hourglass stricture and rigidity simulating infiltrating gastric carcinoma[12-14]. Elective surgical correction of pyloric stenosis is necessary when resulted in gastric outlet obstruction. This usually develops between the first and second month post injury[13], same as our patients in this report.

A number of surgical techniques have been used over the years to deal with pyloric stenosis :

- Dilatation and pyloroplasty were promptly discarded because of problems with restenosis

- Gastroenterostomy was considered the treatment of choice for many years and was effective in relieving the obstruction in nearly all patients. O'Donnell and co-authors[15] and afterwards Eaton and Tennekoon[16] reported the cases of squamous metaplasia and carcinoma in the stomach developing many years following gastroenterostomy for corrosive acid burn of pyloric canal.

- A limited gastric resection, preferably with a Billroth I anastomosis, can be performed safely and is curative. The lesion is totally removed and normal stomach is brought to normal duodenum, a physiologic reconstruction, and malignant degeneration[15,16] is avoid by gastric resection.

The relative sparing of duodenum in contrast to the esophagus and stomach is possibly due to pyloric spasm induced by the irritant acid in the antrum and the alkaline pH of the duodenum. Only one case (Case 6) in our record was so severely injured. Distal esophageal necrosis, fundic perforation of stomach and pyloric and first part duodenum stenoses required extensive operation of esophageal and stomach resection with colon bypass operation, but the post operative course was unevenful.

All of corrosive acid ingestion cases in this report were intentional for attempted suicide. Although the volume of ingested Vixol pink® was difficult to estimate but the deep injury were all present with bloody vomitus in the first 72 hours after injury as in cases 6,7 and 9. Barium swallowing and GISM in the early post injury period may predict the depth of injury when the esophageal motility is decreased or lost (Figure 7). It is the reliable indication of transmural injury or injury to myenteric plexus and should be closely followed up.

Conclusion

The suicidal ingestion of corrosive acid (Vixol pink, 22% Hydrochloric acid W/W) is not uncommon in Thailand. The exact volume consumed was difficult to ascertain in each case, but apparently 50-100 ml. might have been ingested. The bloody vomitus and lost or decreased peristalsis of esophagus in the first few day post ingestion are the indication of deep injury to the wall of stomach and esophagus. Treatment of choice for pyloric stenosis is Billroth I gastrectomy. Esoophageal injury occurs in most of the cases, 9 out of 10 patients, and requires a lengthy course of management. The problem of patients' co-operation and psychic trauma on periodic esophageal dilatation can be overcome by the use of "Soft & Self balloon dilatation". No esophageal reconstructional surgery is necessary in multiple strictures or long segment strictures, except in the case with esophageal necrosis. False tracts that were immediately recognized during dilatation responded well to conservative treatment.

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