The Natural History of Corrosive Gastritis

Report of Five Cases

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ACCIDENTAL OR SUICIDAL ingestion of corrosive substances has been reported sporadically[1-9] and the effects on the upper gastrointestinal tract have been well documented in patients and in the experimental animal.[10, 11] These studies have demonstrated that the esophagus bears the brunt of corrosive alkali ingestion, whereas the stomach, and particularly the pyloric antrum, is preferentially involved after the swallowing of corrosive acids. Concomitant esophagogastric ulceration and fibrosis occurs in 10–20%[6, 9] of corrosive injuries and, in these, alkalies are most frequently the cause. Corrosive gastritis has been reported following the ingestion of many different substances,[1, 2, 6, 8, 9, 12] but the presence of hydrochloric acid in a variety of easily accessible occupational fluids renders it the most frequent causative agent.

The clinical, radiological, and pathologic features of corrosive gastritis have been described in moderate detail, but conclusions have usually been drawn from single case reports. In this paper, detailed case reports of 5 patients will be presented; the clinical, laboratory, radiological, and surgical aspects will be considered, and the natural history of the disease discussed. The surgical results will be considered together with those of an additional 3 cases of corrosive gastritis excluded from the series because of insufficient clinical data.

CASE REPORTS

Case 1

A 20-year-old assistant garage mechanic was admitted with a 5-week story of ulcer-type dyspepsia complicated by brisk hematemesis. The onset was unusual in that the ill-

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ness started abruptly with severe, burning epigastric pain followed by vomiting. The nature and severity of symptoms remained unchanged until 5 days before admission when the pain became more persistent and intense, and the vomitus blood-stained. Two brisk hematemeses necessitating blood transfusion prompted transfer to this hospital. His past history was of interest in that he had been on irregular medication for epilepsy for 5 years and that he was subject to about one epileptic fit a month. Additional history became available after his barium studies; garage associates remembered that he had had an epileptiform seizure immediately prior to the onset of symptoms, and that he had swallowed a corrosive cleaning fluid containing hydrochloric acid during the postictal state. The patient denied any knowledge of this.

On examination he appeared to be in good general condition with a temperature of 99°F. and a normal pulse rate and blood pressure. There was slight pallor of the mucosa, and a single waxy exudate was noted in the left fundus. Deep tenderness was elicited in the upper abdomen. Laboratory investigations showed the following: hemoglobin, 10 gm.%; packed cell volume, 31%; E.S.R., 50 mm./hr. (Westergren); white blood count, 6500 cells; W.R., negative; serum proteins, 6.3 gm.% (albumin 3.2 gm. and globulin 3.1 gm.); blood urea, 32 mg.%; serum bilirubin, alkaline phosphatase and flocculation tests, normal. Barium meal revealed an annular constricting lesion in the pyloric antrum with ulceration in the region of the angulus (Fig. 1 and 2). The stomach emptied rapidly, and the duodenum showed dilatation in its second and third parts. Although carcinoma of the stomach was regarded as the most likely diagnosis, alternative possibilities were considered in view of the patient's age. It was at this stage that the history of suspected ingestion of hydrochloric acid was elicited. Gastroscopy was arranged before this de-

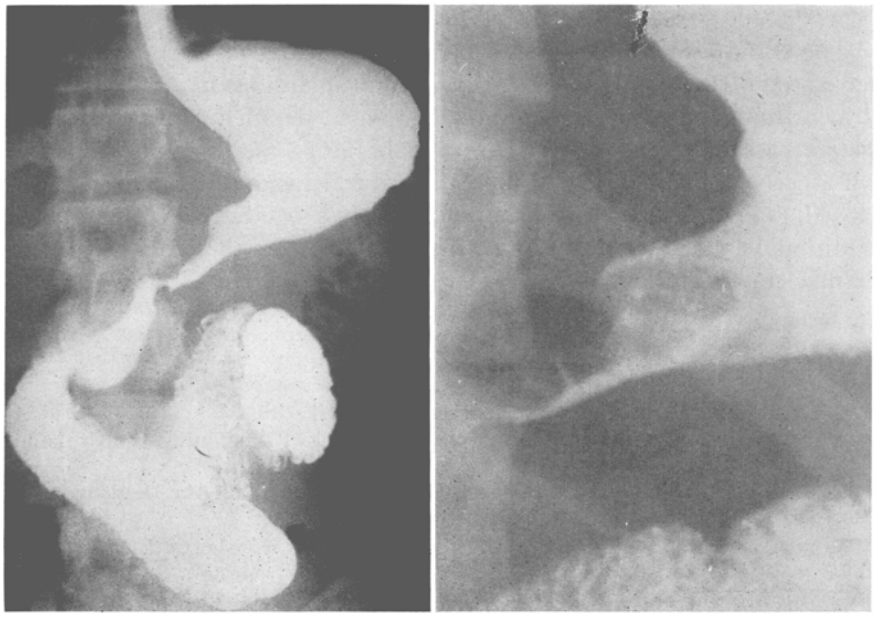

Fig. 1 (*left*). Barium meal film 5 weeks after corrosive ingestion in Case 1. **Fig. 2** (*right*). Filling defect of antrum. (Film taken during same barium study as in Fig. 1.)

cisive history became available. Under suitable premedication and local pharyngeal anesthesia, a Wolf-Schindler gastroscope was advanced into the stomach without difficulty. The body of the stomach was abnormal and showed diffuse hyperemia, extensive superficial ulceration and, on the greater curvature, thick edematous folds. Mucosal atrophy was not evident, and the appearance of the lesion did not suggest a carcinomatous process. The gastroscope was advanced without force and under direct vision to examine the pyloric antrum. The visualization of pulsating blood vessels clearly indicated that the stomach had been perforated, and the latter was confirmed by the absence of liver dullness on percussion, and by the presence of air under the diaphragm on an erect X-ray of the abdomen. It was of interest that the patient was completely unaware of this turn of events, and that he remained asymptomatic until operation; his pulse remained normal throughout.

As surgery was considered inevitable for the stenotic process in the antrum, a partial gastrectomy was carried out within 4 hr. of the perforation. An oblique slit-like perforation was found in the posterior wall of the stomach at the level of the proximal vas brevia; the stomach was firmly adherent to the liver, transverse mesocolon, and anterior abdominal wall and spleen; the body of the stomach was thick-walled and edematous and narrowed to a fibrosed and shrunken antrum and pylorus; the proximal part of the duodenum was dilated and the periesophageal tissues were thickened and inflamed; a large penetrating gastric ulcer tethered the lesser curvature of the stomach to the undersurface of the liver. The patient made an uneventful recovery.

Macroscopically the resected portion of the stomach showed an intense serosal inflammatory response with multiple plastic and fibrotic adhesions. Diffuse hyperemia and numerous superficial ulcerating areas virtually replaced the normal mucosal surface of the pyloric antrum, lesser curvature, and angulus; the deep penetrating ulcer was just distal to the incisura. Cicatricial narrowing of the antrum was marked, and the pylorus barely admitted a small probe. The mucosa in the vicinity of the greater curvature of body was hyperemic and edematous, with large succulent folds and only slight superficial ulceration. The perforation had occurred in this thickened and edematous region. Histology did not reveal any evidence of neoplasia.

Case 2

A 36-year-old laborer was admitted with a 5-hr. history of severe abdominal pain. He had returned home in an inebriated state and inadvertently drank about 4 oz. of paint remover from a wine bottle in which it was kept. The fluid on analysis was found to contain virtually pure 7.5 N hydrochloric acid. Severe abdominal pain, a burning sensation in the mouth and throat, vomiting and sweating developed immediately. Realizing his error, he drank a large amount of milk in an attempt to neutralize the corrosive fluid. The pain became excruciatingly severe and radiated through to the back, and the vomitus now contained altered blood.

He was dehydrated and in extreme pain when admitted to hospital. The pulse rate was 85 per minute; temperature, 96°F., and blood pressure, 110/80. The oro-pharynx showed no abnormality. Bowel sounds were propulsive, and diffuse epigastric tenderness and guarding was elicited. Intravenous fluids were administered over the next 3 days, during which time his pulse rate increased to 105 per minute, and the temperature to 100°F. The pain diminished somewhat in intensity, but flatulence became a distressing symptom. Repeated small hematemeses occurred from the eighth to the eleventh days. Food aggravated the pain and was almost invariably followed by vomiting. On admission his

hemoglobin was 12 gm.%; white blood count, 9500. The serum albumin was 3.5 gm.% and serum globulin, 2.6 gm.%. Serum bilirubin, alkaline phosphatase, and flocculation tests were within normal limits. Serum electrophoresis showed a reduced albumin with normal globulin components. An augmented histamine test carried out 3 weeks after the onset of symptoms revealed a histamine-fast achlorhydria; 81 ml. of clear mucoid gastric juice were aspirated during the 2-hour period of the test, but the lowest *p*H was only 7.3. Barium meal examination, done on the seventeenth day, showed a constricting lesion in the pyloric antrum with mucosal ulceration and a gastric ulcer in the region of the angulus (Fig. 3). The radiologist, unaware of the history of corrosive ingestion, reported an "annular carcinoma of the antrum with ulceration."

Laparotomy on the thirtieth day showed a shortened, contracted stomach with serosal congestion most marked in the region of the lesser curvature, angulus, and pyloric antrum. Perigastric adhesions were absent. The antral wall was thickened and firm, and the appearance resembled that of an infiltrating carcinoma. A Polya gastrectomy was carried out, and the patient made an uneventful recovery.

The resected stomach measured 9 cm. along the lesser curvature and 16 cm. along the greater curvature. The mucous membrane of the acid-bearing area appeared well preserved, while the antral portion showed a complete loss of mucosal surface covered by a thin, membranous inflammatory exudate. A small portion of recognizable mucosa along the lesser curvature was edematous. The antral wall was markedly thickened, this being maximal distally and extending proximally for 4.5 cm. The submucosal edema was up to 1.0 cm. in thickness, and presented a gelatinous appearance (Fig. 4). Histology

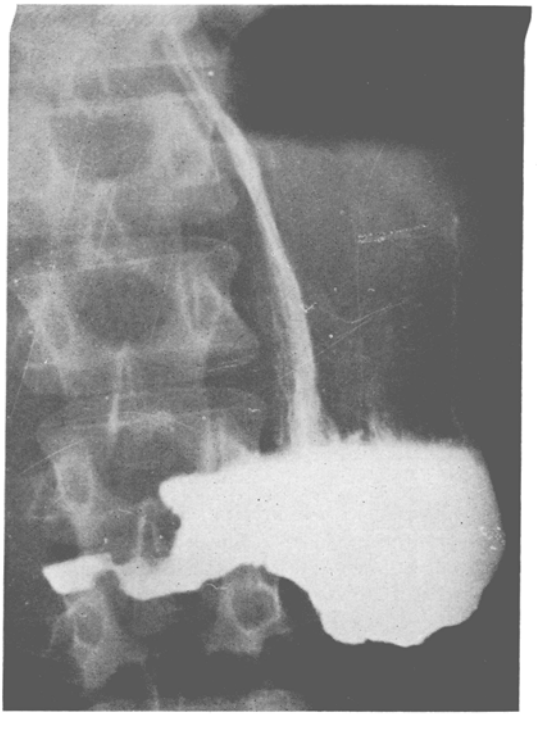

Fig. 3. Barium meal film 17 days after corrosive ingestion in Case 2.

showed the antral mucosa to be virtually absent. The submucosa was markedly edematous, with areas of excessive vascularity covered by inflammatory exudate. The lymph nodes showed reactive change.

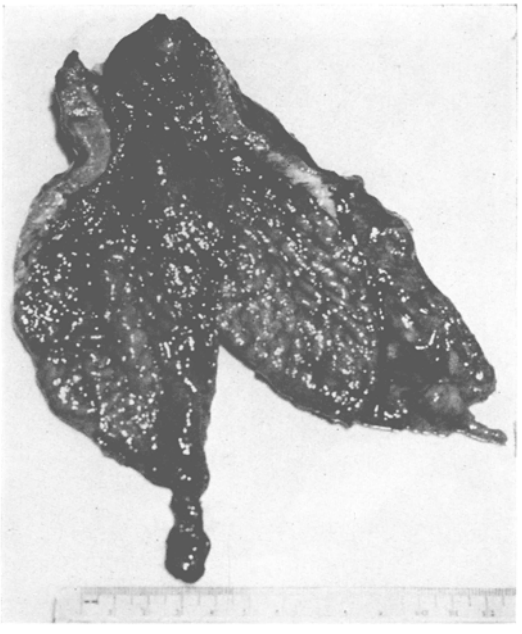

Fig. 4. Partial gastrectomy specimen from Case 2. Note gross submucosal thickening, canal region, and markedly thickened longitudinal antral fold of mucosa.

Case 3

A 19-year-old girl drank about 6 oz. of dilute hydrochloric acid, used as a cleanser before soldering, in a phase of transient depression during an argument between her parents. Abdominal pain and vomiting developed almost immediately, and she was admitted to a psychiatric ward. Her temperature was 99.5°F.; pulse, 80 per minute; and blood pressure, 120/70. Sodium bicarbonate and calcium carbonate were given after admission. Vomiting and abdominal pain were still present when she left hospital 6 days later. A barium meal carried out on the fourteenth day because of severe postprandial pain and repeated hematemeses, showed a "tube-like stomach" (Fig. 5). The duodenum was dilated and showed an air-fluid level. The patient did not return until 9 weeks later, however, by which time she had lost 30 lb. in weight; persistent vomiting and epigastric pain had been present for a month, and she had recently had a further hematemesis.

Examination revealed an introverted, distressed girl with obvious weight loss. Epigastric tenderness was present. The knee and ankle reflexes could not be elicited. Urine examination was negative for protein. The hemoglobin was 11 gm.%; E.S.R., 33 mm./hr. (Westergren); and white blood count, 9500. The serum albumin was 4.7 gm.%; serum globulin, 2.3 gm.%; and the serum bilirubin, alkaline phosphatase, and flocculation tests were within normal limits. The Wasserman reaction was negative. Barium meal revealed diffuse narrowing of the distal half of the stomach with rigidity and minimal irregularity. The appearance suggested fibrotic contraction of the distal half of the stomach almost to

the point of total occlusion. Peristalsis in the proximal part of the stomach was vigorous, and a large portion of the barium had left the stomach at the end of 2 hr. The duodenum was again dilated, and an air-fluid level was present.

Laparotomy was eventually carried out 16 weeks after the onset of symptoms. A tight

Fig. 5. Barium meal film 14 days after corrosive ingestion in Case 3.

stricture was present 1½–2 in. proximal to the pylorus, and considerable edema of the greater omentum was noted. The stomach wall proximal to the stricture was friable. A Billroth I gastrectomy was performed. The postoperative course was complicated by breakdown of the anastomoses and peritonitis, and death occurred on the fourth day. Necropsy showed, in addition, marked friability of the greater curvature of the gastric remnant.

Case 4

A 32-year-old laborer was admitted to hospital vomiting altered blood after an attempted suicide. He had swallowed spirits of salts (hydrochloric acid) and, in addition, had stabbed himself in the abdomen with a screwdriver.

Sloughing of the mouth and tongue was seen on admission, and the stab wound of the abdomen was situated below the left costal margin. His pulse was 108 per minute; blood pressure, 150/80; and hemoglobin, 15 gm.%. An emergency laparotomy revealed a laceration of the liver due to the penetrating injury and slight bruising of the surface of the stomach; the laceration was sutured.

Although his general condition improved, the patient experienced great difficulty in swallowing; this progressed to almost complete dysphagia. A jejunostomy was therefore

carried out on the fifth day. At operation the stomach was found to be grossly swollen and edematous, especially along the lesser curve. A Lipiodol swallow, done after a brisk hematemesis on the nineteenth day, showed that the opaque medium passed into the esophagus and stomach without delay. The stomach contained excess fluid, but emptied within 20 min. An esophagoscopy was attempted, but the esophagus could not be dilated beyond the first few centimetres.

Esophageal dilatation was carried out over the next 3 months, but a barium meal performed just before the end of this period showed an almost complete obstruction at the lower end of the esophagus (Fig. 6). The 6-hr. film showed barium retention in the esophagus and virtual occlusion of a small, scarred, and contracted stomach (Fig. 7). The presence of these findings despite prolonged esophageal dilatations prompted surgical intervention; an esophagogastrectomy was carried out almost 6 months after admission. The postoperative coure was complicated by an anastomotic leak and peritonitis on the fourteenth postoperative day, confirmed at necropsy 2 days later.

Case 5

A 36-year-old foreman in a power station was admitted in a semiconscious state after a suicide attempt. He admitted drinking 10 gm. of potassium hydroxide used as an electrolyte in a hydrogen-generating cell. The past history revealed that he had a depressive illness requiring hospitalization 5 months previously and that he had suffered a relapse prior to this suicide attempt. Oral, pharyngeal, and retrosternal discomfort occurred

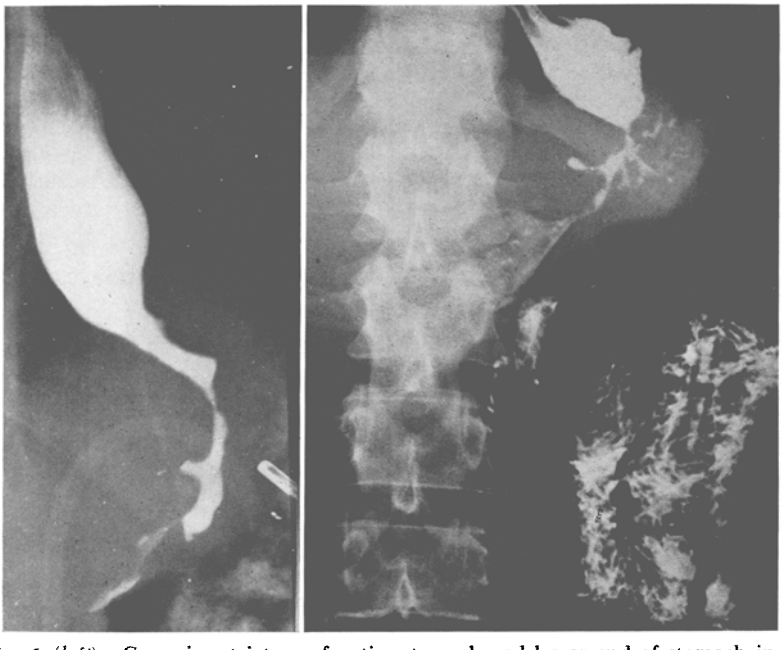

Fig. 6 (*left*). Corrosive stricture of entire stomach and lower end of stomach in Case 4. **Fig. 7** (*right*). Six-hour film showing barium retention in esophagus and scarred and shrunken stomach.

immediately on swallowing the alkali and this was followed within a few seconds by severe, burning epigastric pain and vomiting of blood-stained material. A polythene nasogastric tube was passed on admission and the gatric contents aspirated. Six days after admission he vomited an esophageal cast.

On physical examination the pulse was 100 per minute, and the blood pressure, 110/80. Mild excoriation of the lips and tongue was present. The abdomen was soft and nontender. The heart and lungs were normal. Results of the relevant investigations were: Hb, 13.8 gm.%; P.C.V., 42; W.B.C.'s, 12,360; E.S.R., 60 mm./hr.; serum albumin, 3.5 gm.%; serum globulin, 2.1 gm.%; liver function tests, normal. The electrophoretogram showed a moderate diminution in albumin and a low gamma globulin concentration; the $\alpha 1$, $\alpha 2$, and β globulins were normal. An augmented histamine test done on the tenth day yielded 185 cc. of clear gastric juice. Although basal achlorhydria was present the response to maximal histamine stimulation produced an output of 3.3 mEq. "free" acid per hour, and a "total" acid output of 7.0 mEq./hr. Barium examination after 5 days showed that the esophageal mucosa was destroyed, but no obstruction to the passage of the barium was encountered; the body and antral region of the stomach was rigid, narrowed, and ulcerated on the lesser curvature (Fig. 8). The second part of the duodenum was dilated and an air fluid level was present. Pyloric holdup was apparent radiologically one month after admission, and an organic stricture of the pyloric region was evident when the barium study was repeated one month later (Fig. 9). Altered blood was vomited through the polythene nasogastric tube before the latter study, and upper abdominal discomfort and frequent vomiting of gastric contents through the tube developed soon after. Weight loss continued, and it became obvious that the progressive pyloric stenosis would require surgical intervention. Laparotomy carried out 3 months after admission revealed thickening and fibrosis of the distal inch of the prepyloric antrum to a point of almost complete stenosis; the obstruction was short-circuited with a posterior gastroenterostomy performed, and the polythene nasogastric tube was left in position.

Further barium studies and esophagoscopy showed narrowing of the distal 15 cm. of the esophagus and, since this lesion was also progressive, the first stage of an esophageal bypass using transverse colon was carried out 6 months after admission. Further reconstruction of the esophagus is planned.

The salient features in the 5 patients with corrosive gastritis are collected in Table 1.

Four of the patients were admitted to hospital within a few hours of swallowing the corrosive, and the fifth was transferred from another hospital shortly after. Acute oropharyngeal discomfort was present in 3 patients (Cases 2, 4, and 5), 2 of whom developed dysphagia (Cases 4 and 5). This necessitated a feeding jejunostomy in one (Case 4) and feeding through a nasogastric tube in another (Case 5). Oropharyngeal discomfort was not noted in 2 of the 4 who had swallowed hydrochloric acid (Cases 1 and 3). The sudden onset of severe epigastric pain and vomiting almost immediately after the ingestion of the corrosive was perhaps the most arresting clinical feature; the vomitus was frequently blood-streaked. Thereafter the pain became more or less persistent, aggravated by food and eased to some extent by vomiting or alkalis. Four of the 5 patients had one

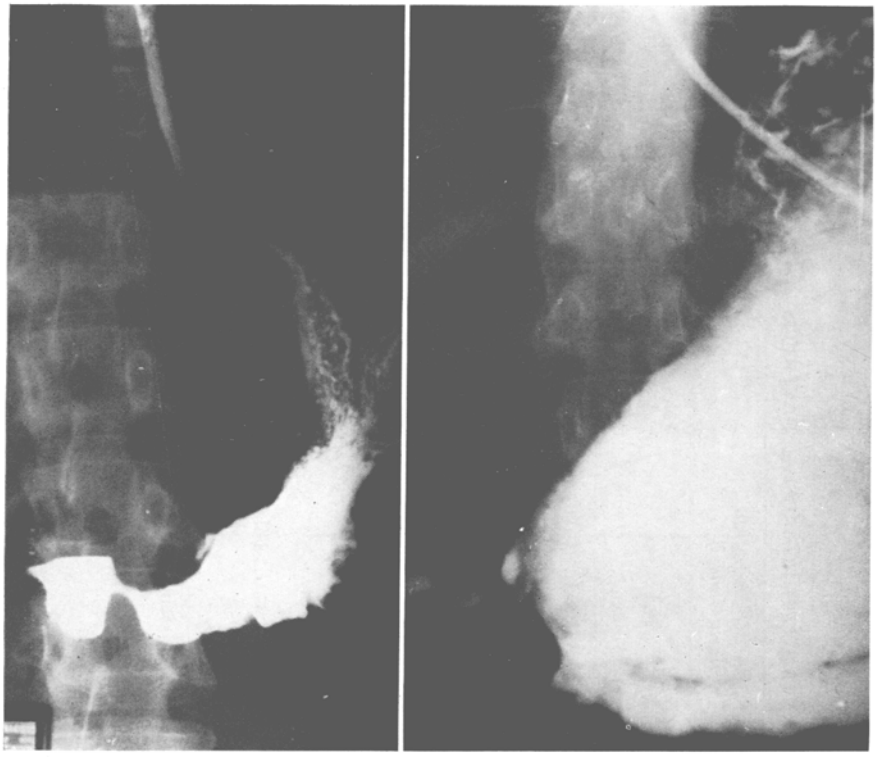

Fig. 8 (*left*). Barium meal film 5 days after corrosive ingestion in Case 5. **Fig. 9** (*right*). Barium meal film 2 months after that of Fig. 8, showing gross pyloric stenosis.

or more brisk hematemeses from 8 days to 6 weeks after the corrosive ingestion; the fifth (Case 5) vomited blood-stained material on admission and again after 5 weeks. The vomiting became more persistent and obstructive in type within 2–6 weeks in 3 patients (Cases 2, 3, and 5); one of the remaining patients (Case 1) was operated on before the development of obstructive vomiting, and the other (Case 4) had a feeding jejunostomy carried out on the fifth day because of esophageal strictures; a subsequent barium meal showed almost complete pyloric stenosis, and he did in fact vomit gastric contents through his tube after about 6 weeks. Weight loss was prominent in 4 of the 5 patients.

The hypoalbuminemia and hypogammaglobulinemia in Case 5 were investigated by administering 20 μc of I^{131} albumin intravenously and measuring the radioactivity of bidaily aliquots of gastric juice on a scintillating counter for 7 days. The fall-off in the blood was also determined by measuring blood radioactivity every second day. The I^{131} albumin was adminis-

TABLE 1. DATA ON 5 PATIENTS WITH CORROSIVE GASTRITIS

Case No.	Sex & age	Corrosive	Reason for ingestion	Admission (time after onset)	Involvement			X-ray determination (Barium meal)			Hematemesis (time after onset)	Serum albumin (gm.%)	E.S.R. (mm./hr.)	Surgery	
					Mouth	Esoph.	Stomach	Time (after onset)	Stomach*	Duod.				Time (after onset)	Operation
1	M 20	HCl	Postictal state	5 wk.	−	−	+	5 wk.	A, L.C.U.	Dilated	4 wk.	3.2	50	6 wk.	Polya Gy.†
2	M 36	HCl	Inebriated	1 day	±	−	+	17 days	A, L.C.U.	—	Adm.; 8–11 days	3.5		4 wk.	Polya Gy.†
3	F 19	HCl	Suicidal	1 day	−	−	+	2 wk. 11 wk.	A A	Dilated fluid level	10–12 days; 6 wk.	4.7	33	16 wk.	Billroth I Gy.†
4	M 32	HCl	Suicidal	1 day	+	+	++	3 wk. 18 wk.	N E		Adm.; 19 days			5 days 22 wk.	Jejunostomy Esoph.-Gy.†
5	M 36	KOH	Suicidal	1 day	+	+	+	5 days 1 wk. 2 wk.	A, L.C.U. P.S. P.S.	Dilated fluid level	Adm. 5 wk.	3.5	60	3 wk. 6 wk.	G-E‡ Esoph. by-pass

*Key: A represents constricting lesion distal part stomach; P.S., grossly dilated stomach; N, normal; and L.C.U., lesser curve ulceration; E, entire stomach involved.

†Gastrectomy.

‡Gastroenterostomy.

tered 11 days after the onset of the corrosive gastritis. A relatively constant gastric excretion of radioactivity varying from 1.2 to 2.0% of the administered dose per day was calculated from the aliquots, it being assumed that gastric secretion was of the order of 1500 cc. per day. The fall-off in blood

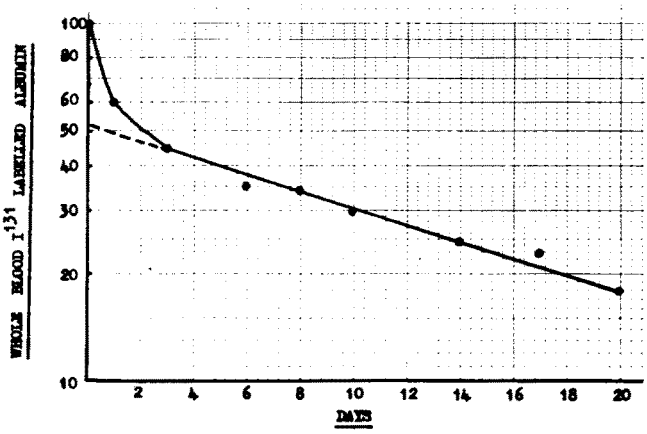

Fig. 10. Disappearance curve of I^{131}-labeled albumin from whole blood in Case 5 (corrosive gastritis).

radioactivity shown in Fig. 10 was more rapid than in control subjects studied here.[29]

The augmented histamine test was carried out in 2 patients, one of whom showed a histamine-fast achlorhydria (Case 2). The other, Case 5, failed to secrete acid under basal conditions and showed an impaired response to maximal histamine stimulation; the posthistamine response was 3.3 mEq. "free" acid in the hour. Both patients secreted excessive amounts of mucus.

Radiological Features

Esophageal involvement was a prominent feature in the patient who had swallowed potassium hydroxide (Case 5) and in one of the 4 patients who had taken hydrochloric acid (Case 4).

The radiological features of the stomach were remarkably similar in 4 of the 5 patients; diffuse narrowing and rigidity of the distal half of the stomach, maximal in the pyloric region, was the rule by the end of the second or third week, and 3 of the 4 also had evidence of ulceration of the lesser curvature in the region of the angulus. Repeat barium studies in 2 of these 4 patients revealed that the lesion had progressed to virtually complete pyloric stenosis by the end of 11 weeks. In the fifth patient (Case 4) the stomach emptied normally on the nineteenth day, but appeared small,

scarred, and contracted and its lumen almost completely occluded at the end of the fourth month.

Dilation of the second and third parts of the duodenum was present in three of the five patients, and a fluid level was noted in the dilated portion of the duodenum in two of them (Cases 3 and 5).

Surgical Treatment

The intractable epigastric pain and liability to frank hematemesis coupled with the relentless progression of the lesion to virtually complete pyloric stenosis made surgery mandatory in all our patients. Perforation of the stomach during gastroscopy necessitated an emergency gastrectomy in one (Case 1), but surgery was carried out in the others only after conservative treatment had obviously failed to influence the natural history of the disease.

Associated esophageal involvement necessitated feeding through a nasogastric tube in one patient (Case 5) and a feeding jejunostomy in another (Case 4). Both patients required surgery for the subsequent development of esophageal and pyloric stenosis.

A standard Polya partial gastrectomy was carried out in 2 patients (Cases 1 and 2), a Billroth I in one (Case 3) and an esophagogastrectomy in another (Case 4). A gastroenterostomy and subsequent reconstructive surgery of the esophagus was carried out in one patient (Case 5) with both pyloric and esophageal stenosis. Two of the 3 patients whose case histories were omitted because of insufficient clinical data had a Polya partial gastectomy carried out for antral stenosis; a total gastrectomy was performed in the third, who presented 5 months after swallowing hydrochloric acid with strictures of the cardiac, mid-body, and pyloric regions of the stomach. Two of the 8 patients developed anastomotic leaks.

DISCUSSION

The clinical features, radiologic appearances and natural history of the 5 cases of corrosive gastritis presented in this paper are in keeping with previous reports of the condition. The suicidal intent in 3 of our patients clearly indicates that easily accessible corrosive cleaning fluids are still resorted to despite the availability of more sophisticated and less unpleasant methods of attempting suicide. The inadvertent swallowing of cleaning fluids containing hydrochloric acid in the two remaining patients, one during a postictal state and the other while inebriated, underlines the importance of careful enquiry for a possible history of corrosive ingestion in patients with clinical or radiological features of corrosive gastritis.

The present study has demonstrated that the clinical picture is frequently as dramatic as the disturbance which prompted ingestion of the corrosive. Severe epigastric pain and retching at the onset is followed by a period of intractable ulcer-type dyspepsia associated with frequent vomiting of blood-stained gastric contents and, at times, frank hematemeses and melaena; obstructive vomiting and weight loss eventually supervene, with the development of pyloric stenosis. The radiological and pathologic features of corrosive gastritis are also fairly uniform, irrespective of the particular corrosive ingested. The pyloric antrum and incisura show maximal involvement and the lesion progresses inexorably to antral stenosis. The tendency for acid corrosives to "lick the esophagus and bite the pyloric antrum" as opposed to the tendency of alkali corrosives to "bite the esophagus and lick the pyloric antrum" has been confirmed.

The selective localization of corrosive gastritis to the antrum and lesser curvature has been investigated in the experimental animal by Testa,[10] who showed that strong corrosives descend the lesser curvature along the magenstrasse, bathe the angulus, and accumulate in the antrum, and suggested that the presence of the corrosive in the antrum excites tetanic prepyloric spasm resulting in the bulk of the acid being concentrated in this site. Extensive superficial ulceration of the antrum invariably occurs, and edema, ulceration, and cicatrization of the angulus or lesser curvature proximal to it develop not infrequently. Laparotomy in Case 1 revealed that the lesser curve ulcer had penetrated deeply into the liver, but it is of interest that the radiological appearance of an ulcer in Case 2 was almost certainly due to gross thickening of the mucosal folds in the region.

Difficulty in distinguishing the radiological appearance of corrosive gastritis from carcinomatous infiltration of the antrum has been stressed in the past,[14–16] particularly in those patients in whom a history of corrosive ingestion is lacking. Gray and Holmes[17] drew attention to the fact that a number of patients presenting with pyloric obstruction due to corrosive ingestion had forgotten or were unaware of the original incident, as illustrated by Case 1. This erroneous diagnosis may also be made on clinical grounds since weight loss is prominent, the E.S.R. elevated, and achlorhydria or marked hypochlorhydria the rule; indeed the gastric lesion may appear cartilaginous[18] and resemble neoplastic infiltration at laparotomy. The intense submucosal edema in Case 2 simulated a mucoid carcinoma even on naked-eye inspection of the lesion.

Progression to the stage of the true antral stenosis has been reported to vary from 19 days[19] to as long as 6 years.[20] Cases 4 and 5 illustrate that pyloric stenosis may develop insidiously and remain undetected in patients suffering from corrosive stricture of the esophagus; a careful radio-

logical examination of the stomach for antral deformity or mucosal irregularity should therefore be carried out in these patients. Conversely, the finding of pyloric stenosis in a patient with a past history of corrosive stricture of the esophagus should also alert one to the possibility of its being a legacy of the initial corrosive ingestion.

Perforation of the stomach during gastroscopy in Case 1 exemplified the danger of this procedure in patients with corrosive gastritis. Gastroscopy has only rarely been carried out in patients with this condition, and we have gleaned but a single report from the literature.[21] Although the pyloric antrum, angulus, and lesser curvature are selectively involved in corrosive gastritis, the greater curvature and fundus frequently show mucosal hyperemia and submucosal edema. The perforation in our patient occurred through edematous fundic wall, and was due, in part at least, to tethering of the stomach to surrounding organs with consequent reduction in the normal mobility and distensibility of the stomach.

The association between hypoalbuminemia and giant hypertrophic gastritis has received attention in recent years.[22, 23] Albumin leakage into gastric juice in patients with giant hypertrophic gastritis has been demonstrated utilizing radioactive iodinated serum albumin[23] and radioactive polyvinyl pyrrolidone (P.V.P.) technics.[24] A similar protein-losing gastropathy has recently been shown in some patients with gastric carcinoma by means of electrophoretic studies on gastric juice.[25] The finding of low serum albumin values in 3 of the 4 patients in our series in whom data were available suggested that corrosive gastritis may constitute a further cause of protein-losing gastropathy; all were well-nourished and showed normal hepatic and renal function. The results of the labeled-albumin studies carried out in one of our patients tended to support this hypothesis and might have been more convincing had the study been commenced immediately after the acute insult. Further studies, preferably with P.V.P.,[24] are obviously necessary to confirm this observation.

Results of acid secretory studies carried out in 2 of our patients were in keeping with the experience of previous authors regarding the absence or gross diminution of both basal and stimulated acid secretion. The gross reduction in acid secretion in a lesion involving the pyloric gland area, sparing the greater part of the parietal cell mass, requires explanation. Destruction of the gastrin-producing area is unlikely to be entirely responsible for this and it is probable that functional impairment of the parietal cells,[12] analogous to that found in active gastric ulceration,[26–28] contributes to the marked reduction in acid secretion.

Radiology showed abnormal widening of the second and third parts of the duodenum in 3 of the 5 patients in the present series; a fluid level was

noted in 2 of these 3 patients. The absence of operative or necropsy evidence of corrosive duodenitis or of direct involvement of the distal portion of the duodenum by perigastric adhesions suggests that the radiological findings in these 3 patients were probably reflex in nature. This raises the possibility as to whether the presence of duodenal dilatation in other conditions may not also be due to associated disorders.

Although the initial treatment of corrosive gastritis is obviously medical, the subsequent development of pyloric stenosis makes eventual surgery mandatory in almost all patients. Early surgery is hazardous because of friability and edema of the gastric wall, and surgery even at a later stage may be difficult for the same reason. A feeding jejunostomy is often helpful to tide the patient over until elective surgery can be carried out. The type of surgery depends on the extent of the corrosive involvement, but partial gastrectomy or gastroenterostomy are usually adequate in patients with antral or pyloric stenosis. Total gastrectomy may be necessary in lesions involving the entire stomach. Associated esophageal involvement may necessitate further surgery aimed at replacing the stenosed portion of the esophagus with a jejunal or colonic bypass.

SUMMARY

Case reports of 5 patients with corrosive gastritis are presented. The corrosive was ingested accidentally in one, without the patient's knowledge in another, and with suicidal intent in 3. Hydrochloric acid in the form of cleaning fluid was the offending substance in 4 and potassium hydroxide in 1.

The natural history of the disease is outlined. The dramatic onset of dyspepsia, the liability to hemorrhage, and the relentless progression to pyloric stenosis is stressed, and the tendency of the pyloric gland area to be selectively involved is confirmed. The entire stomach was stenosed in one, and associated involvement of the esophagus was present in 2 of the patients. The radiological and clinical points of similarity between corrosive gastritis and carcinomatous infiltration of the antrum are mentioned. The danger of gastroscopy in the condition is discussed.

Attention is drawn to the finding of low serum albumin values in patients with corrosive gastritis. It is tentatively suggested that the latter may constitute another cause of protein-losing gastropathy.

The treatment of the condition is briefly considered.

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