GASTRIC STRICTURE FOLLOWING ZINC CHLORIDE INGESTION

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SYNOPSIS

This paper describes the clinical course of a 26 year old woman who swallowed a solution containing 35% zinc chloride and 0.5% methanol. The initial injuries were similar to that seen in patients who had ingested the more commonly available 11% zinc chloride solution used in soldering. She showed no central nervous system involvement. Inspite of the severity of the upper pharyngeal corrosion as evidenced by sloughing of the mucous membrane and by the presence of subcutaneous emphysema of the upper chest wall and neck, there were no residual strictures of the pharynx or oesophagus. Only the stomach became severely contracted. She required corrective surgery to relieve the gastric obstruction.

INTRODUCTION

Studies by the Draize technique in rabbits of acute toxicity of the various zinc salts reveal that the chloride salt is the most corrosive. (1) In these studies, early death after gastric intubation with a solution of zinc chloride shows general precipitation of the stomach lining. Reports in the literature of acute zinc chloride toxicity are usually related to the ingestion of zinc soldering fluxes, (2, 3) — an 11% solution of zinc chloride. Symptoms and signs of corrosion are mild with the ingestion of an 11% solution of zinc chloride. We report here a patient who swallowed about 10mls of correction fluid which contains 35% zinc chloride and 0.5% methanol.

CASE REPORT

YCG, a 26 year old Chinese lady, was admitted to us with the history that she had swallowed about 10mls of a correction fluid (OCE — correction fluid; contents 35% zinc chloride and 0.5% methanol). She was employed as a clerk in a multinational company and had of late been under psychiatric care for reactive depression. She also had a past history of suffering from bronchial asthma.

At the time of admission the patient was conscious and alert. There was evidence of small areas of first degree burns on the upper lip. There was gross oedema of the oral mucosa and pharynx. The pharnyx was in addition inflammed with sloughing of the mucous membrane. The patient did not complain of pain but she had lost her voice and was unable to swallow her saliva. Her abdomen was soft to palpation with only minimal tenderness in the epigastrium. She also passed some loose stools.

A gastric lavage was performed with normal saline using a nasogastric tube. A later attempt to pass an oesophagoscope to determine the extent of the corrosion was not possible because of pain. She was commenced on treatment with intravenous hydrocortisone 200mgm at 8 hourly intervals, and maintained on intravenous fluids. Aminophylline was added to relieve the bronchial spasms of the asthmatic attack that had developed. Ampicillin was given intramuscularly as prophylaxis against infection. Intravenous cimetidine was also used prophylactically.

Laboratory investigations revealed a haemoglobin of 17.8gm/dl and a total white cell count of 28,800/ul. Urine microscopy contained 5-6 red cell in the field, with albumin at +, and the presence of urobilinogen. Liver function studies: total protein 7.0 gm/DL, albumin 4.6 gm/Dl, bilirubin 0.9 mg/DL, Alkaline phosphatase 80 u/L (normal 32 — 105 u/L), SGPT 16 u/L (normal 9 — 36 u/L), and SGOT 31 u/L (normal 15 — 33 u/L). Serum zinc estimated on the third hospital day was 90 ug/DL (normal 55 — 150 ug/DL). Serum electrolytes, blood urea, blood gases were all within normal range.

On the third hospital day the patient was found to have subcutaneous emphysema in the upper chest wall and the neck. There was no pneumothorax or pneumomediastinum. She was still troubled by bronchial asthma although this was only mild.

On the fifth hospital day, the subcutaneous emphysema was noticed to have cleared partially. The patient was still breathless and this was due more to inspiratory stridor than to bronchial asthma. She was still unable to swallow her saliva and had not regained her normal voice. She had a bout of malena. Her blood pressure remained at 110/50, but her haemoglobin level was now 11.8gm/dl.

On the ninth hospital day, the patient was in a fair state. She was coughing and was still unable to swallow her saliva. She was now able to speak and able to take small feeds. However, she began suddenly to vomit fresh blood. The haemetemesis persisted for two days with decreasing amounts. Her blood loss was corrected with multiple blood transfusions and intravenous cimetidine was used. Her PT and aPTT were both normal. She began to make adequate recovery after this episode of haematemesis. Her main problems were the persistence of the inability to swallow her saliva and coughing. She could, however, on occasions, manage only small amounts of feeds and frequently vomited that too.

On the twenty-fourth day of admission, a barium swallow was performed. With the first bolus, barium was seen to be aspirated into the trachea and then into the left bronchus. The oesophagus was satisfactorily delineated and its mucosal pattern was normal. There were no strictures seen. As the entry of the barium into the respiratory tract could imply scarring or severe inflammation in the pharynx leading to abnormal deglutition, or even the presence of a fistula, a direct laryngoscopy was done to view the upper pharynx. No muscosal ulcerations were seen. The only abnormality was moderate oedema of the ary-epiglottic folds of the supra-glottic part of the larynx. Both vocal cords were normal. It did not seem that the amount of swelling could cause the overspill of barium into the larynx. As even drinking water caused the patient to cough at the examination of the larynx, the possibility of a fistula was considered. She was continued on steroid therapy and at repeat laryngogoscopy two weeks later, the ary-epiglottic folds were only mildly oedematous. Both the vocal cords were normal and mobile. Oesophagoscopy at this time showed that the oesophagus was normal. The stomach was not clearly seen because of food debris but was noted to be contracted. She remained comfortable taking small feeds orally and through a Ryles tube. She still complained of mild epigastric discomfort. There was occasional vomiting of feeds but generally she was able to retain small liquid meals.

On the forty-fifth hospital day, she began to complain of vomiting of all her feeds. She was unable to even retain 75 mls of the liquid diet. There was no epigastric discomfort. Oesophagoscopic examination showed that the stomach was contracted and the oesophagoscope could not be passed beyond 50 cm. The barium meal revealed a long smooth stricture involving the entire body of the stomach except for the fundal region and the antrum (Fig. 1). There were no fistulae. The oesophagus and the duodenum were normal.

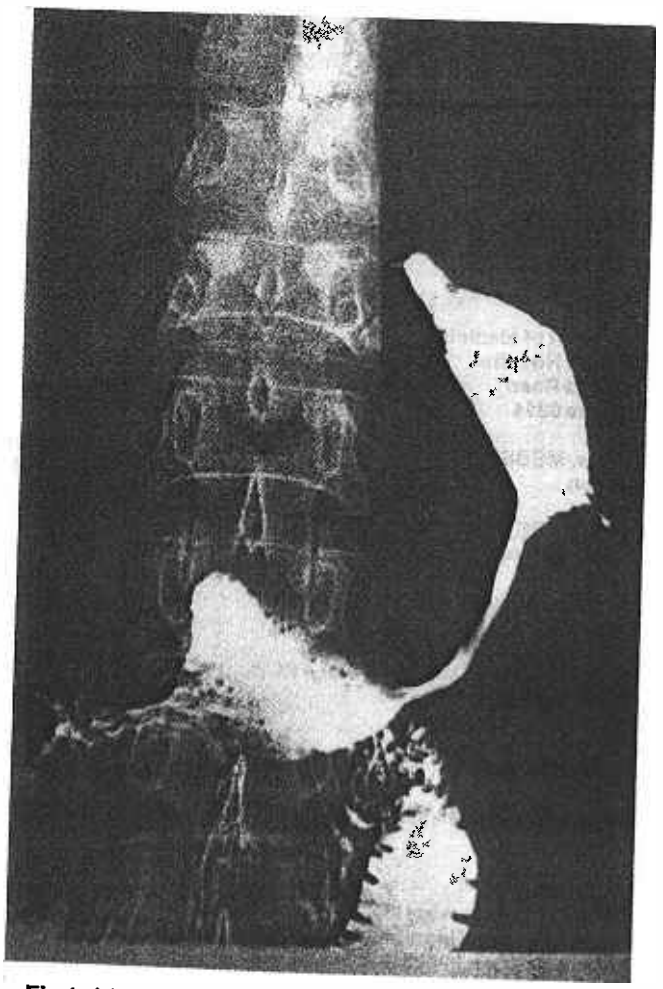

Fig 1. A barium meal picture of the stomach showing the long smooth stricture involving the entire body of the stomach.

A subtotal gastrectomy with gastro-jejunostomy was performed and the patient remains in fair condition. A repeat barium meal six weeks after corrective surgery showed no later stricture formation in the pharynx, oesophagus or the fundus of the stomach.

Pathology report of stomach:

The stomach received was already opened along the lesser curve which measured 4 cm. The greater curve was 8 cm long. The stomach was small and the size of the lumen was markedly reduced.

A false diverticulum-like structure was seen in the proximal half as a result of instrumentation. The mucosa of the proximal portion was ulcerated and haemorrhagic. A few rugae were seen in the pylorus.

The muscle layer was thickened especially at the pylorus whose lumen was greatly narrowed.

Microscopically there were areas of ulceration which extended down to the upper muscle layers. The base of the ulcer was composed of vascular granulation tissue with much plasma cell infiltrate. Stretches of the mucosa showed regenerative glands.

The submucosa was thickened by fibrosis and showed a patchy infiltrate of plasma cells, lymphocytes and a few polymorphonuclear leucocytes.

The muscle layer was hypertrophic and thickened nerve bundles were present between the muscle layers.

DISCUSSION

In the late 1950 and early 1960s, in Singapore, it was common for caustic soda to be used in suicide attempts. (4) The corrosive action of caustic soda caused acute inflammation and fistula formation such as oesophago-bronchial fistula, oesophago-pleural fistula, and subsequent strictures of the oesophagus and the stomach, which often required corrective surgery. Fortunately in 1962, legislation was passed banning the sales of caustic soda for use in homes. This Act led to the total disappearance of caustic soda being used in suicide attempts. Today, washing detergents are very commonly used in suicide attempts. (5) But the attempt at suicide of this patient with a solution that contains 35% zinc chloride highlights the availability of a new corrosive that has the potential to cause as much problem as caustic soda. The chloride salt of zinc is the most toxic of the commonly occurring zinc compounds. (1) Accidental or suicidal ingestion of zinc chloride solutions has usually been associated with solder flux (11% zinc chloride solution). This results in mild burns of the pharynx with sloughing of the mucous membrane and ulcer formation. This patient ingested correction fluid which is a solution containing 35% zinc chloride and 0.5% methanol. She had evidence of first degree burns on her lips, excessive oedema of the oral pharynx with sloughing of the mucous membrane. On the third day of her hospital stay she was noticed to have subcutaneous emphysema of the anterior chest wall and the neck, but not pneumothorax or pneumomediastinum. This could have been the result of perforation of the oral pharynx from the corrosive action of the zinc chloride or consequent upon trauma (gastric washout and oesophagoscopy) to friable pharyngeal tissues. The initial difficulty this patient experienced with swallowing of saliva, water and the barium of the first barium meal examination, which showed the tracking of barium down to the left main bronchus, was fortunately not associated with any fistulae.

Serum zinc levels were reported to be high in the patients described by Potter (2) and Chobanian. (3) Both their patients were lethargic on admission. In this patient, serum zinc was taken for estimation on the third day. The level was within normal limits for our laboratory. There was no central nervous system involvement. The patient was alert on admission. Recent studies suggest that corticosteriods reduce serum zinc levels. (6) Our patient was commenced on intravenous hydrocortisone as she had bronchial asthma that required to be relieved at admission, and this may have lowered the serum zinc levels and thus prevented the onset of central nervous system depression. However, the therapeutic implications of corticosteriod in zinc chloride intoxication have not been defined. Calcium disodium edate is recommended as the chelating agent in the management of raised serum zinc levels. (7) It was not used in this patient. Gastric lavage should have been avoided in this patient who showed so much of the corrosive effects of zinc chloride ingestion, to avoid creating fistulae. The subcutaneous emphysema could have been the result of trauma to a friable pharynx.

Leucocytosis occurs several hours after exposure to zinc oxide fumes (metal fume fever). (8) In this patient, the initial white cell count of 28,800/cmm, confirms that a rapid leucocyte response occurs also in zinc chloride ingestion. The presence of microhaematuria may have been the result of acute tubular necrosis and interstitial nephritis as with all heavy metal poisionings. Abdominal pains may be due to pancreatic damage suggested by elevated amylase and lipase values. (8) These enzymes were not estimated in this patient. Her epigastric pains were mild and vague. Moore (9) reported the first case of gastrointestinal bleeding in a patient who was on zinc sulphate for acne. Our patient had no acute gastric haemorrhage, although she initially had some diarrhoea. The severe haemetemesis and malena on the ninth hospital day was most probably due to secondary haemorrhage of gastric corrosion.

But perhaps the most unusual feature in this patient is the amount of gastric cicatrization without associated pharyngeal or oesohageal stricture. In rats, early death after gastric intubation with zinc chloride shows precipitation of the stomach lining. (1) This patient developed severe haemetemesis on the ninth day. Although oesophagoscopy was not done then to elucidate the cause, it could be presumed that this was a haemorrhage from sloughing of a damaged mucosa or from ulcerations in the stomach. A prophylactic H_2- inhibitor was used but did not prevent the ulceration due to zinc chloride. Microscopy of the portion of the stomach that was removed showed areas of ulceration which extended to the upper muscle layer.

Oesophagoscopy as early as the twenty-fourth hospital day already showed evidence of contraction of the stomach. At surgery, it was found to be severely strictured and resembling a shrunken tube. Only a small cuff of normal stomach remained at the cardia. The stomach was small and its size was markedly reduced. At histology, the submucosa was thickened by fibrosis. The muscle layer was hyperthrophic and especially at the phylorus it was thickened greatly narrowing the lumen. (Fig. 2) Experimental studies by Spain and colleagues in 1950, first suggested that early administration of cortisone produced an anti-inflammatory effect that would inhibit fibroplasia in wound healing. (10) Studies also suggested that steroid therapy should be instituted immediately after corrosive injury of the oesophagus. However, this form of treatment is discouraged if the patient is seen later than 48 hours following injury.

This patient was admitted to us approximately four hours after ingestion of the solution of zinc chloride, and inspite of the early administration of hydrocortisone, albeit for the relief of bronchial asthma, and its continued use, the gross stricture of the stomach could not be prevented. Perhaps, the 35% zinc chloride and 0.5% methanol solution ingested by this patient,

Fig 2. - The specimen of the stomach showing severe stricture and hypertrophy of the muscle layers.

caused severe damage to the stomach because it acted longer on the gastric mucosa and its wall than in the pharynx and oesophagus.

When last reviewed, three months after corrective surgery, this patient remained well and had increased her weight. She was due to return to work.

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REFERENCES

1. Hehir RM, Gaskin JR, et al: Toxicology of zinc soldering compounds. J Toxicol Appl Pharmacol 1964; 6: 349.
2. Potter JL: Acute zinc chloride ingestion in a young child. Ann Emerg Med 1981; 10: 267-9.
3. Chobanian SJ: Accidental ingestion of liquid zinc chloride: Local and systemic effects. Ann Emerg Med 1981; 10: 91-3.
4. Seow LJ. Personal communication, 1984.
5. Chee YC, Teo LH: Self poisoning: A study of female patients hospitalized in a general medicine department in one year. Sing Med J 1984; 25 (4): 240-3.
6. Flyan A, Pories WJ, Strain WH, et al: Rapid serum zinc depletion associated with corticosteroid therapy. Lancet 1971; 2: 1169-72.
7. Dresbach RH. Management of poisoning in Handbook of Poisoning, 9th ed 1977: 86.
8. Murphy J: Intoxication following ingestation of elemental zinc. JAMA 1970; 212: 2119-20.
9. Moore R: Bleeding gastric erosion after oral zinc sulphate. Br Med 1978; 1: 754.
10. Spain DM, Malomert N, Haber A: The effect of cortisone on the formation of granulation tissue in mice. Am J Pathol 1950; 26: 710.
11. Knox WG, Scott JR, Zintel HA, et al: Bouginage and steroids used singly or in combination in experimental corrosive oesophagitis. Ann Surg 1967; 166: 930-41.