

# An Unusual Manifestation of Severe Caustic Injury

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## ABSTRACT

**Knowledge of the sequelae of caustic ingestion is of relevance to staff of the Emergency Room, Intensive Care Unit, surgical and gastroenterology services. It poses a considerable management problem and may result in life-threatening complications such as visceral perforation. This case report demonstrates an unusual and previously unreported manifestation of caustic injury.**

**Keywords:** caustic, complication, diaphragm, gastric, perforation

## CASE REPORT

A 33-year-old domestic worker from the Philippines, after experiencing auditory hallucinations, swallowed an unknown quantity of Chlorox® and was brought to the Emergency Room. She sustained caustic injury to the face, neck and anterior chest wall due to vomiting. There was inflammation and severe ulceration of the pharynx and vocal cords. She was intubated for airway protection but was allowed to breathe spontaneously. Intravenous fluids and dexamethasone 4 mg every eighth hourly was given. There was no abnormality on systems examination. Initial chest X-ray and blood investigations were normal.

Total parenteral nutrition was instituted two days later via a right internal jugular line. Endoscopy was not performed due to severe hypo-pharyngeal involvement. After five days she developed a low grade fever. Blood cultures and chest X-ray were normal. The central venous line became dislodged and was re-inserted in the left internal jugular vein. By day seven (post-ingestion), the fever continued and a moderate size right pneumothorax was found on repeat chest X-ray. This was tolerated for approximately twelve hours when oxygen desaturation became apparent (oxygen saturation 85% - 90%). Examination revealed only pneumothorax.

Urgent chest CT scan revealed pneumothorax with bilateral pleural fluid. There was no mediastinal collection. A dilute barium swallow via a naso-oesophageal tube did not demonstrate oesophageal perforation. Chest tube insertion revealed bilateral haemothoraces. A naso-oesophageal tube was passed to the stomach and

blood stained aspirate was drained. Bronchoscopy revealed upper tracheal erythema only.

Emergency surgery was planned for suspected oesophageal perforation. Intra-operative endoscopy revealed mild, diffuse erythema of the oesophagus and a gastric fundal perforation. Thoracotomy revealed a blood stained empyema (which was subsequently drained) and excluded oesophageal and apical pleuro-pulmonary injury. Laparotomy revealed a gastric fundal perforation and adjacent left diaphragmatic perforation. Fundectomy was performed and the diaphragm was repaired.

The post-operative course was complicated by metabolic acidosis and seizure activity which were managed in the intensive care by intravenous bicarbonate, broad spectrum antibiotics and phenytoin. Total parenteral nutrition was continued. After a long convalescent period, she was discharged and returned to the Philippines.

## DISCUSSION

Caustic alkaline agents cause prompt liquefactive necrosis while acids produce coagulative necrosis. The spectrum of injury includes superficial mucosal involvement to transmural injury with perforation, resulting in the involvement of adjacent organs including the duodenum, pancreas, gall bladder, spleen, liver, aorta, the tracheo-bronchial tree and non-perforating injury to the diaphragm<sup>(1-3)</sup>. It is thought that acids may spare the oesophagus and cause gastric injury, though it is known that severe gastroesophageal injury may occur with either strong or weak alkaline or acid substances<sup>(4)</sup>. Sodium hypochlorite, considered a weak alkali, had devastating results in our patient.

Emergency endoscopy, within 24 - 48 hours of ingestion may have prognostic value, as patients with severe injury are more likely to suffer adverse short or long term complications<sup>(5,6)</sup>. This was not done in our patient due to the severity of the hypopharyngeal and upper airway injury, a contraindication to gastrointestinal instrumentation<sup>(7,8)</sup>.

The role of steroids is controversial in the management of caustic ingestion. Once considered valuable<sup>(9)</sup> and used in our patient, they are not recommended by others because of the lack of efficacy and the potential danger due to immunosuppression<sup>(10)</sup>.

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Emergency surgery is indicated for signs of perforation, peritonism, acidosis, continued bleeding or endoscopic evidence of extensive "black necrosis" of the oesophagus or stomach<sup>(1,2)</sup>. In our patient, the fundal perforation was not associated with signs of peritonism, possibly due to perforation of the adjacent diaphragm with spillage of gastric contents into the thorax rather than the peritoneal cavity. The pneumothorax led us to investigate for oesophageal or tracheobronchial perforation though it reflected gastric and diaphragmatic perforation. Diaphragmatic involvement has been reported in widespread intraperitoneal caustic injury<sup>(1)</sup>. However, perforation and resultant surgical intervention were not required.

The pneumothorax was contralateral to the site of the diaphragmatic perforation. It was possibly caused by air tracking along the pleural space from left to right, after the initial diaphragmatic perforation. The central line was a potential cause. However, at thoracotomy there was no evidence of central line associated pleural or lung injury.

Mortality rates of 23% – 50%<sup>(1,2,11-13)</sup> include patients with severe caustic injury from surgical series. Predictors of higher mortality include: ingestion of more than 60 mLs of strong alkali<sup>(12)</sup>, age greater than 50 years and signs of peritonism, shock or acidosis<sup>(13)</sup>. Despite significant complications experienced by our patient, she made a successful recovery after intensive multidisciplinary management.

We have reported a new manifestation of visceral perforation complicating caustic ingestion. As it occurred without the typical signs of visceral perforation, early recognition in future requires knowledge of the entity and a high index of suspicion.

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