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Acute esophageal injury and strictures following corrosive ingestions in a 27 year cohort

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ABSTRACT

Purpose: We aimed to determine the incidence of esophageal strictures in corrosive ingestions and potential predictors of severe injury.**Basic procedures:** This was a retrospective cohort study of corrosive ingestions from a toxicology unit (1987–2013) with telephone follow-up at least 1 y post-ingestion. Clinical data and investigations were obtained from a toxicology admission database. The primary outcome was esophageal stricture. Other outcomes included in-hospital mortality, endoscopy grade and early complications.**Main findings:** There were 89 corrosive ingestions; median age, 31 y [1–87 y; 46 females], including 13 strong alkalis (pH > 12), 8 strong acids (pH < 2), 29 domestic bleaches, 30 other domestic products, 6 non-domestic products and three unknown. Three patients died in hospital within 24 h (phenol, sodium azide, HCl). Two developed strictures (both strong alkalis): one had complete esophageal destruction; another developed a stricture after 25 d (inpatient grade 2A endoscopy). 24 patients were asymptomatic and discharged without complication. 65 patients were symptomatic (4 catastrophic injuries). 61 reported sore mouth/throat (50), abdominal pain (21), chest pain (17), dysphagia (13); 28 had an abnormal oropharyngeal examination. 25/61 symptomatic patients underwent inpatient endoscopy: normal (3), grade 1 (5), grade 2 (15) and grade 3 (2). Of 88 patients, 12 died (3 inpatients, 9 unrelated), 28 couldn't be contacted and 48 were contacted after 1.7–24 y, including two with strictures. Five couldn't be interviewed (normal endoscopy (1), no dysphagia (3) and stroke (1). 4/41 interviewed reported dysphagia but no objective evidence of stricture.**Principal conclusions:** All inpatient deaths and severe complications were apparent within hours of ingestion, and occurred with highly corrosive substances. One delayed stricture occurred, not predicted by inpatient endoscopy.

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1. Introduction

Corrosive ingestions are a common presentation to emergency departments and can cause life-threatening injuries within hours of ingestion, such as esophageal perforation. Patients who survive are at risk of developing esophageal strictures, which can lead to dysphagia causing vomiting, regurgitation, aspiration and nutritional deficiency. They usually require endoscopic dilation and sometimes surgery [1].

An important issue in the emergency department is the initial determination of who is at risk of complications from corrosive injuries. This aims to allow patients at low risk to be discharged early without further investigation. Conversely patients at risk of early complications should be admitted and investigated. Acute complications present early and require immediate intervention. In adult patients a number of studies

[2–4] suggest that patients ingesting strong acids or alkalis and/or demonstrating major clinical findings on admission will develop early complications such as perforation and delayed complications such as strictures [3,5]. However, there is less information on the frequency and severity of strictures in these patients [5,6].

The specific predictors of stricture formation remain unclear. Numerous variables contribute to the degree of esophageal injury, including pH, volume, concentration and viscosity of the substance. Endoscopy is commonly used to investigate corrosive injuries, but it remains unclear how useful it is in predicting esophageal strictures. A recent review suggested that endoscopy may help in predicting the later development of a stricture [7]. Computed tomography (CT) has been suggested as a non-invasive alternative to endoscopy [8,9], but there is limited evidence that CT predicts delayed esophageal strictures [7].

This study aimed to determine the incidence of esophageal strictures following corrosive ingestions in a toxicology unit in the developed world, as well as attempt to identify potential risk factors or early features that predict stricture formation.

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2. Methods

2.1. Study design and setting

We undertook a retrospective cohort study of patients admitted to a tertiary toxicology service following corrosive ingestion. The study was undertaken in an urban hospital with approximately 30,000 annual emergency presentations. The tertiary toxicology unit services a population of over 500,000 people. Every poisoned patient is reviewed daily by the toxicology unit and treatment determined by the medical toxicologist. The study was approved by the Institutional Ethics Committee.

All poisoning presentations, including corrosive ingestions, have data collected prospectively during their hospital admission using a pre-formatted admission sheet. This includes demographic details, clinical effects, complications, investigations and treatment [10]. Data are then entered into a relational database (Microsoft Access) within a week of admission by trained research staff. Each entry is also reviewed by a medical toxicologist.

2.2. Selection of participants

Patients who presented following a corrosive exposure between January 1987 and December 2013 were identified by searching the toxicology database. Only patients with corrosive ingestions were included – topical, inhalational and parenteral exposures were excluded.

2.3. Data collection and processing

Data was extracted regarding the initial admission from the toxicology database. This included age, sex, hospital length of stay (LOS), previous deliberate self-harm or psychiatric admission, intention of the ingestion (deliberate, accidental), symptoms (oropharyngeal pain, chest pain, abdominal pain and dysphagia), documented signs of oropharyngeal injury, investigations (endoscopy) and any treatment. Endoscopic findings were classified according to the Zargar criteria (Supplementary Table 1). All esophagoscopies were flexible and done by the attending gastroenterologist.

Patients were contacted for follow up and the medical record reviewed to obtain information on patient outcomes, including whether there were any subsequent re-presentations, death, further esophageal investigations or symptoms of stricture. Living patients were notified of the study by mail and then contacted by phone after two weeks. Up to five attempts were made to contact the patient by phone. Once contacted, patients were asked about any further symptoms or investigations for strictures. They were then evaluated using the Mayo Dysphagia Questionnaire [11]. If an endoscopy had been performed after discharge the results were obtained with patient permission.

Substances were classified according to their pH, with strong acids (pH < 2) and strong alkalis (pH > 12) considered as discrete groups due to their association in the literature with more severe gastroesophageal injury [12]. The remaining agents were classified into domestic products (reasonably expected to be found in a home) and non-domestic products (intended for industrial or commercial use). Bleach was considered as a separate category due to the frequency with which it was ingested and it rarely being reported to cause significant injury [1]. All chart reviews and phone interviews were conducted by a single investigator.

2.4. Outcomes

The primary outcome was the development of an endoscopically confirmed stricture within two years of ingestion. Secondary outcomes included symptoms of esophageal injury, development of a stricture at any time, in-hospital mortality, all-cause mortality, inpatient endoscopy results (classified using Zargar's criteria [13]), hospital LOS, and the presence of dysphagia symptoms on the Mayo questionnaire.

2.5. Analysis

Continuous variables are reported as median values with interquartile ranges (IQR) and/or ranges. All analyses were done in GraphPad Prism (Version 6 for Windows, GraphPad Software, San Diego California USA, www.graphpad.com).

3. Results

During the 27 y study period there were 120 admissions for corrosive exposure out of a total of 17,266 toxicology admissions [10]. Thirty one were not ingestions (14 inhalational, 11 injected, and six topical). This left 89 admissions in 88 patients with one patient presenting twice (Fig. 1). The median age was 31 y (range: 1–87 y; IQR: 20–45 y) and 46 patients (52%) were female. Sixteen ingestions were unintentional (18%). Fifty one patients (57%) had a separate psychiatric admission in their medical record and 43 (48%) had another presentation for deliberate self-harm. Two admissions were young children with minor injuries due to unintentional ingestions.

Corrosive substances ingested were strong alkalis in 13 (15%), strong acids in eight (9%), domestic bleach in 29 (37%), other domestic products in 30 (34%), non-domestic products in six (7%) and unknown in three (3%). The median LOS was 1 day (range: 0–66 d; IQR 1–2 d; Table 1).

3.1. Clinical outcomes

Twelve patients died during the study period. Three deaths occurred during the first 24 h of hospital admission as a direct result of highly corrosive ingestions. A 42 y old male ingested 80% phenol and had severe corrosive injury of the gastrointestinal tract, and developed hypotension, hypothermia, metabolic acidosis and died 24 h post-ingestion. An 81 y old male ingested 32% HCl and developed severe corrosive injury of the stomach and duodenum, metabolic acidosis, perforated intestine, hypotension and died 8 h post-ingestion. A 32 y old male ingested sodium azide and presented with a decreased level of consciousness and severe metabolic acidosis, then developed intractable hypotension and died about 5 h post-ingestion. Only the 81 y old man had an endoscopy, with a grade 2b esophageal injury, but severe injury to the stomach. In the other nine patients who died, the cause and time of death was not related to their ingestion (Supplementary Table 2).

There were two endoscopically confirmed strictures that both presented early. The first was diagnosed during the initial admission in a 38 y old man who presented 24 h after ingestion of a commercial cleaner (strong alkali). He had a grade 3B esophageal injury on endoscopy and eventually required a colonic conduit operation because of complete esophageal destruction. The second patient was a 48 y old woman who accidentally ingested one mouthful of a commercial cleaning fluid (strong alkali). She had a 2A injury at endoscopy during her initial admission but did not have any dysphagia in hospital. Follow-up barium swallow 18 days post-ingestion showed a stricture which was confirmed endoscopically and dilated. She was never symptomatic from this stricture and had no subsequent dilation.

Twenty four patients were asymptomatic (27%) (Fig. 2). Twenty two of these were discharged without endoscopy, and two others had a normal esophagus on endoscopy. Sixty five patients were symptomatic, including the four with catastrophic injuries (three died and one required a colonic conduit). Of the remaining 61 reporting symptoms, 50 had a sore mouth or throat, 21 had abdominal pain, 17 had chest pain and 13 reported dysphagia (Table 2). Twenty eight patients had an abnormal oropharynx on examination.

Twenty nine of 89 admissions underwent endoscopy during the initial admission. Of these 25 were performed within 24 h, two between 24 and 48 h post-ingestion and two >48 h post-ingestion. Nine of the 29 had a high grade esophageal injury (grade 2B or above) (Fig. 2 and Table 3). One of these developed a stricture but already had a

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