Corrosives

Paul I Dargan

Abstract

Acids and alkalis cause severe corrosive damage on contact with tissues. Topical exposure causes severe pain, blistering and ulceration. The most important aspect of management is decontamination with water until skin pH is normal. Inhalational exposure results in sore throat, cough, wheeze and respiratory distress. Hoarseness or stridor necessitates an urgent anaesthetic assessment and perhaps endotracheal intubation or tracheostomy. Ingestion causes severe pain, oropharyngeal burns and vomiting. In severe cases, dysphagia, drooling of saliva and oesophageal or gastric perforation occur with mediastinitis/peritonitis, shock and a high mortality rate. Patients with shock or clinical/radiographic features of perforation should be referred for urgent surgical assessment. Drooling of saliva, severe pain, oropharyngeal burns, dysphagia, vomiting and stridor are indications for endoscopy to grade severity of injury and guide management. Patients with Zargar grade 1 injury can be given a trial of oral fluids. Those with grade 2a–3a injury at endoscopy should be admitted for observation and kept nil by mouth; those with grade 3b injury should undergo urgent computed tomography to assess depth of necrosis and need for urgent surgery. If injury is grade 2a or higher, patients should be followed up after discharge as there is a risk of long-term strictures.

Keywords Acid; alkali; corrosive; decontamination; endoscopy

Introduction

Acids and alkalis are found in a variety of household and industrial chemicals, they can cause severe corrosive damage on contact with tissues. Exposure can occur by topical contact with the skin or eyes, inhalation or ingestion – the clinical pattern of toxicity depends on the route of exposure.

The risk associated with acid/alkali exposure is dependent on the volume of solution the individual is exposed to, the concentration of the solution and, in particular, the strength of the acid/alkali in the solution. The strength of an acid/alkali is determined by its $pK_{a/b}$ – the pH at which it is half dissociated in aqueous solution. Stronger acids/alkalis have a lower pK_a/pK_b and are therefore ionized to a greater extent in aqueous solution. They have a more extreme pH (lower pH for acids, higher pH for alkalis) – examples include hydrochloric acid (pH 1.1 in 0.1 M solution), sulfuric acid (pH 1.2), sodium phosphate (pH 12.0) and sodium hydroxide (pH 13.0). Weaker acids/alkalis have a higher $pK_{a/b}$, so there is an equilibrium between the parent and ionized form and they have a lesser impact on pH. Examples include carbonic acid (pH 3.8 in 0.1 M solution) and sodium bicarbonate (pH 8.3).

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The risk of severe corrosive injury is greatest with exposure to acids with pH <2 and alkalis with pH >11–12.[1,2] Generally, industrial products have a more extreme pH because they are more likely to contain concentrated solutions of stronger acids/alkalis. The risk of severe corrosive injury is therefore greater with exposure to industrial products containing acids or alkalis.

Common inorganic acids include hydrochloric, hydrofluoric, nitric, phosphoric and sulfuric acid; common organic acids include acetic, lactic and trichloroacetic acid. Hydrochloric acid is used in many industrial processes, sulfuric acid is found in car batteries, and many anti-rust products contain a mixture of phosphoric and hydrochloric acid. Hydrofluoric acid is used in metal processing and glass etching. Weaker solutions of inorganic and/or organic acids are found in a variety of household products including products to remove limescale and lavatory cleaners. Sodium hydroxide is found in industrial products and drain cleaners, and many household products, including dishwasher detergent, denture cleaners and oven cleaners, contain alkalis. Household bleach generally contains dilute solutions of the weak alkali sodium hypochlorite, and therefore exposures to household bleach are less likely to cause significant corrosive injury than exposures to other household products or to industrial alkali products.

Epidemiology

There are limited data on the prevalence of corrosive exposures. In England and Wales, it is estimated that there are approximately 15,000 exposures per year.[3] In 2012, US poisons centres took 193,802 enquiries relating to exposures to cleaning substances (7.2% of all exposures) and 39,729 enquiries relating to chemical exposures (1.4%); specific data are not provided on corrosive ingestions, but it is likely that a proportion of these involve corrosives.[4] The age of individuals presenting with corrosive ingestions typically follows two peaks: children aged 2–5 years who ingest household products unintentionally, and adults who more commonly ingest products intentionally to cause themselves harm.[5,6]

Mechanisms of toxicity

Dissociated hydroxide (OH^-) ions within alkaline products penetrate the tissue surface to cause liquefaction necrosis; this tissue penetration can result in corrosive effects spreading deep into the tissues, resulting in protein dissolution, cell membrane destruction, fat saponification, transmural thrombosis, necrosis and ulceration.[2,7]

Free protons (H^+) within acid products cause direct corrosive damage to the tissues, resulting in coagulative necrosis.[7] Unlike alkalis, acids have poor tissue penetration, and therefore the greatest effects are seen within the epithelium; however, concentrated and/or strong acids can cause deeper tissue damage. The coagulative necrosis results in oedema, erythema and mucosal sloughing; more severe injury is associated with desiccation, eschar formation, necrosis and ulceration.

In addition to their direct corrosive effects, acids and alkalis can cause further tissue damage related to an exothermic reaction, causing thermal injury. The fluoride ions in hydrofluoric acid complex with calcium, and this can result in severe hypocalcaemia.[8]

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Topical exposure

Clinical features

Strong acids and alkalis cause pain (although alkaline burns can initially be painless), blistering, ulceration and penetrating necrosis.[9,10] Hydrofluoric acid and strong alkalis can produce deep blistering and ulceration that can progress over a period of many hours if treatment and decontamination is delayed; extensive burns can also result in significant fluid and electrolyte loss.[8–10]

Management

The patient should be decontaminated in a well-ventilated area. Contaminated clothing should be removed and the skin decontaminated with copious amounts of water at low pressure for at least 10–15 minutes until the pH of the skin is normal (pH 5–6 in adults and 6–7 in children); more prolonged irrigation may be required for exposures to strong concentrated acids and particularly alkalis.[9,10] The pH of the affected areas of skin should be rechecked 10–15 minutes after irrigation, and further irrigation may be required. Neutralizing chemicals should not be used as this can result in heat generation that can worsen skin damage. Patients with extensive burns (>10–15% body surface area) may require rehydration with intravenous crystalloid. Analgesia with parenteral opioids may be needed as the burns can be extremely painful. The skin blistering and ulceration should be treated as for thermal burns after decontamination; skin grafting may be required for extensive full-thickness injury.[9,10]

Hydrofluoric acid exposure can result in severe, deep burns and, in addition, significant hypocalcaemia can develop. Topical application of calcium gluconate 2.5% gel and/or subcutaneous injection of calcium gluconate 10% should be considered, and in patients with severe burns of the entire hand, intra-arterial injection of calcium gluconate 10% into the brachial artery can be considered.[8]

Inhalation

Clinical features

Inhalational exposure results in irritant effects to the eyes and nose, with sore throat, cough, chest tightness, wheeze, tachypnoea and dyspnoea. Stridor due to laryngeal oedema can occur with exposure to strong acids/alkalis, and pulmonary oedema with increasing respiratory distress, wheeze and hypoxia can develop; this may be delayed and develop over 12–36 hours after exposure with acute lung injury (ALI) in severe cases.[11,12] Respiratory effects can also occur from aspiration of ingested acids/alkalis.

Management

Contaminated clothing should be removed and patients managed supportively. Those with significant respiratory effects should undergo a peak flow, chest X-ray and arterial blood gas measurement and be treated with oxygen.[11] If wheeze is present, treat with nebulized salbutamol 2.5 milligrams, which may need to be repeated.[11,12] There is no evidence that prophylactic steroids or antibiotics are of benefit; pulmonary oedema or ALI may require continuous positive airway pressure or mechanical ventilation with positive end-expiratory pressure.[11,12] Patients with stridor should have an urgent anaesthetic assessment – endotracheal intubation or tracheostomy may be required if severe laryngeal oedema/ulceration is present.

Ophthalmic exposure

Clinical features

Initial effects include blepharospasm, lacrimation, conjunctivitis and palpebral oedema. Exposure to more concentrated and stronger acids and particularly alkalis can result in corneal ulceration and iritis; in severe cases, long-term complications including corneal opacification, cataracts and retinal atrophy can occur.[13]

Management

Contact lenses should be removed and the eye(s) irrigated with water for at least 10–15 minutes until the conjunctival pH is 7–8; longer irrigation may be required.[13] The pH should be rechecked after 10–15 minutes; further irrigation may be needed. Installation of topical local anaesthetics may be required in patients with severe pain or blepharospasm to facilitate irrigation. Fluorescein staining should be used to assess for corneal damage. Patients with severe clinical features and/or corneal damage should have an urgent ophthalmology assessment.[13]

Ingestion

Clinical features

Ingestion of strong acids and alkalis causes similar clinical features, although alkalis may be associated with greater proximal (oropharyngeal, oesophageal) injury.[14–18] Initial effects include severe pain in the mouth, throat and upper abdomen/chest. Oropharyngeal burns may be seen on inspection of the mouth, although their absence does not exclude distal injury.[14] Patients can develop vomiting, haematemesis and diarrhoea. Severe mucosal oedema can make swallowing difficult, resulting in drooling of saliva.[14] Aspiration can lead to laryngeal and airway involvement with hoarseness, stridor, airway compromise and later pneumonitis and acute lung injury.[11,12,14,19,20] Oesophageal and/or gastric perforation can occur with mediastinitis, peritonitis, shock and a high mortality rate.[19,20] Systemic effects including shock related to fluid loss or haematemesis, metabolic acidosis, electrolyte disturbances, haemolysis and disseminated intravascular coagulation can occur in severe cases.

Patients who survive an episode of severe corrosive ingestion can go on to develop stricture formation weeks to months later, leading to dysphagia and regurgitation (oesophageal stricture) and/or vomiting and easy satiation (pyloric stenosis); the risk of stricture formation is greatest in those with grade 2 and particularly grade 3 injury.[21,22] There is an increased risk of oesophageal and gastric cancer in those with severe corrosive injury and stricture formation.[23]

In addition to severe local gastrointestinal irritant effects, hydrofluoric acid ingestion can result in severe hypocalcaemia with tetany, convulsions and arrhythmias.[8]

Risk assessment and management

Patients with hoarseness, stridor or any respiratory distress should have an urgent anaesthetic assessment – endotracheal intubation or tracheostomy may be required if severe laryngeal

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