Cutaneous chemical burns: assessment and early management

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Background

Chemical burns are common and may cause significant physical, psychological, social and economic burden. Despite a wide variety of potentially harmful chemicals, important general principles may be drawn in the assessment and initial management of such injuries. Early treatment of chemical burns is crucial and may reduce the period of resulting morbidity.

Objective

This article reviews the assessment and management of cutaneous chemical burns.

Discussion

Assessment of the patient should be rapid and occur in conjunction with early emergency management. Rapid history and primary and secondary survey may be required to exclude systemic side effects of the injury. Depth of wound assessment is difficult given that necrosis caused by various chemicals can continue despite cessation of exposure. Early management should be conducted with consideration of clinician’s safety, and appropriate precautions should be taken. Excluding specific situations and chemical exposure, copious irrigation with water remains the mainstay of early management. Referral to a centre of higher acuity may be required for expert evaluation.

Keywords

burns, chemical; wounds and injuries; skin

Burns are a common trauma that affects up to 1% of the Australian population and may be associated with significant physical, psychological, social and economic burden. Chemical burns represent 3–5% of all burns-associated admissions. Despite the small proportion, chemical burns account for 30% of burns-associated death, most commonly occurring as a result of chemical ingestion. Given the nature of injury, hospitalisation tends to be prolonged and healing is delayed.

Many substances that are freely available in the community, either occupational or domestic items, have the potential to cause chemical burns. The immediate availability and poor labelling of these substances has accounted for an increase in unintentional chemical burns. Assault and suicidal attempts account for the remaining cases of chemical burns. The affected population is generally evenly distributed but an increase in paediatric chemical burns has been previously documented. Areas affected tend to include the face, eyes and extremities. As such, the scope of this review is limited to the assessment and management of cutaneous chemical burns. Ocular burns should be urgently referred to an appropriate ophthalmic service.

Pathophysiology and types of chemicals

The pathological end result of chemical burns, regardless of the type of chemical, is consistent with changes occurring during thermal burns. The external toxic stimulus causes denaturation of biological proteins and thus renders them physiologically
inactive. This inactivation of essential
proteins results in cell death. Thermal
burns tend to cause rapid coagulation
of protein due to protein crosslinking.
By contrast, chemical burns cause
denaturation of physiological proteins
through six different processes including
reduction, oxidation, corrosion, vesication,
dessication and protoplasmic poisoning.5,7
It should be noted that many chemicals
cause injury through combinations of
these processes.

Chemical agents can also be classified
on the basis of the induced chemical
reaction that the agent initiates.
Such classification may be useful for
consideration of early management
options. Chemical agents may be
classified into one of these categories
despite slight variations in the resulting
clinical sequelae.

- Acids: act as proton donors in the
  biological system. Acid injury causes a
coagulative necrosis of the superficial
tissue.
- Bases: chemicals are proton acceptors
  and tend to have greater capability
  of producing injury.28 These agents
  produce heat via reactions with fats,
  extract water from surrounding tissue
  and result in liquefactive necrosis (Figure 1).
  Such necrosis allows penetration deep to the superficial
  wound and continues to cause injury
despite initial removal of the insult.9
- Organic solutions: cause injury by
dissolving the lipid membrane, which
results in disruption of physiological
processes.
- Inorganic solutions: cause injury by
denaturation mechanisms as outlined
above.

Assessment

1. Personal protection equipment: it is
vital that the treating clinician wears
protective clothing to prevent injury
(eg gloves, safety goggles).
2. Primary and secondary survey: as with
any clinical presentation, the patient
must be stabilised using principals
of primary survey. This should be
completed in a rapid and systematic
approach.
   a. Airways: ingestion of chemicals,
      particularly alkali agents, may
      result in upper airways obstruction.
      Stabilisation of airways and urgent
      medical support is required.
   b. Breathing: special considerations
during chemical injury include the
      exclusion of inhalation injuries,
      particularly for aerosol chemicals or
      smoke.10 Such patients frequently
      require ventilatory support and thus
      early referral should be sought prior
to clinical deterioration.
   c. Circulation: smaller chemical burns
      infrequently cause cardiovascular
collapse. Occasionally, severe
      metabolic disturbances may result
      from chemical absorption and thus
      monitoring and stabilisation may be
      required.
3. History: a rapid history should
   be taken simultaneously during
   primary survey and initial care of
   the cutaneous burn. It is vital that
   such assessment does not delay the
   initiation of immediate treatment.
   Information regarding comorbidities
   and medication may be useful.
   Information regarding the chemical
   injury is important, particularly if the
   patient requires transfer to a higher
   acuity service. Pertinent information
   includes: insulting agent (and the
   associated mechanism of injury),
   phase of the chemical (gas, liquid
or solid), concentration, quantity,
duration of cutaneous contact, extent
of penetration and initial emergency
management.7

General principles of
management

After primary survey and initial rapid
assessment, the following care outlines
the general principles for managing acute
chemical burns.11
1. Removal of the chemical: the duration
   of skin contact is the key determinant
   of injury severity.12,13 Thus, prompt
   removal of chemical contact is
   mandatory.
   a. This should be performed rapidly
      and generally requires removal of
      contaminated clothing at the scene
      of injury.14 Initially, residues or dust
      should be brushed off the skin.
   b. Irrigation should then be performed
      with warm water under a tap with
      appropriate drainage to prevent
      further injury. Care should be
taken to ensure the wash off does
      not occur across unaffected skin.
   c. Neutralisation of chemicals is
      contentious but is generally not
      indicated because of the risk of
      further heat production and thus
      continuing injury. Several neutralising
      agents have shown some benefit,9
      but irrigation with plain water
      remains the most efficacious,
      accessible and cost-effective
      treatment.12,15–17
2. Complete wound evaluation: the
   microcirculation of the wound
   is evaluated by pinprick test for
   pain and capillary return time.18,19
   Assessment regarding the depth
   of the chemical burn is notoriously
difficult, as burns may be deceptively superficial. The difference in surface temperature between the affected and unaffected skin may assist in depth assessment. Re-assessment should be done at regular intervals as this may provide information about injury progression. As a general rule, unless the observer can be absolutely sure, chemical burns should be considered deep dermal of full-thickness until proven otherwise.

a. Chemicals causing liquefactive necrosis, typically basic solutions, may cause continuing necrosis dispute removal of agent. Caution should be practised in such situations and expert opinion may be required.

b. Debridement of blisters and non-viable tissue is advocated as early as possible via surgical or non-surgical approaches.

3. Systemic toxicity: the insulting chemical injury or subsequent treatment may produce systemic changes that require assessment and intervention.

a. Metabolic disturbances: the most common disturbance is acid-base imbalance. Monitoring blood gases through venous sampling may be necessary to ensure metabolic stability.

b. Electrolyte disturbances and associated sequel: various chemicals may cause biochemical disturbances. As such, patients may require biochemical analysis on admission to higher acuity centres. For example, hydrofluoric acid (HFA) may cause hypocalcaemia and resulting cardiac arrhythmia.

c. Hypothermia: may occur from the prolonged duration of wound lavage. Water temperature should be maintained as close to body temperature as possible.

4. Referral: Given the difficulty in assessing injury extent and depth, caution is generally advised. Chemical burns should be treated as full-thickness burns until proven otherwise.

a. Unless full thickness burns can be explicitly excluded by the treating physician, referral to secondary or tertiary centres is required for formal assessment by specialist services. Full-thickness chemical burns may require admission for surgical debridement and grafting of non-viable tissue.

b. Ocular chemical injury is beyond the scope of this review, but generally requires urgent ophthalmic review.

Specific agents

Management of specific chemical agents is complex and is generally advised in the emergency department following early management. Current Australian guidelines have been previously published. Table 1 outlines common domestic products and the harmful agents they contain, with more comprehensive lists easily accessible online.

- Cement: is a common cause of chemical burns. The main injury-causing agent in wet cement is calcium oxide and resulting hydroxyl ion. Cement has multiple mechanisms of action, but predominantly can be classed as an alkali. Injury is insidious, usually presenting several hours after injury.
- Tar: in liquid form, tar is superheated and classically causes deep thermal and chemical burns. If not removed promptly, tar cools and causes liquefactive necrosis and adheres to skin. Adherent tar should not be removed in the pre-hospital setting and urgent referral is required as surgical debridement may be necessary. Various household items such as baby oil, mineral oils and butter may aid in tar removal.
- Hydrochloric and sulphuric acids: burns caused by these agents are among the most commonly treated chemical burns. Common household goods contain moderate concentrations of such agents or their immediate precursors. On contact, these agents donate protons and cause coagulative necrosis.

### Table 1. Common domestic agents and mechanism of injury

<table>
<thead>
<tr>
<th>Domestic item</th>
<th>Chemical agent</th>
<th>Pathological process</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>In the garage</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Batteries (car)</td>
<td>Sulphuric acid</td>
<td>Potent acid causing coagulative necrosis</td>
</tr>
<tr>
<td><strong>In the laundry</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cleaners</td>
<td>Ammonia</td>
<td>Potent alkali causing oxidation and liquefactive necrosis</td>
</tr>
<tr>
<td></td>
<td>Sodium hypochlorite</td>
<td>As previous</td>
</tr>
<tr>
<td>Bleach</td>
<td>Sodium hypochlorite</td>
<td>As previous</td>
</tr>
<tr>
<td>Pool cleaner</td>
<td>Sodium hypochlorite</td>
<td>As previous</td>
</tr>
<tr>
<td><strong>In the kitchen</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oven cleaners</td>
<td>Sodium (or potassium) hydroxide</td>
<td>Potent alkali causing oxidation and production of heat</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(exothermic)</td>
</tr>
<tr>
<td><strong>In the bathroom</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toilet cleaner</td>
<td>Precursors of sulphuric acid</td>
<td>Potent acids and alkalis as previous</td>
</tr>
<tr>
<td></td>
<td>Hypochlorite</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hydrochloric acid</td>
<td></td>
</tr>
<tr>
<td>Drain cleaner</td>
<td>Sulphuric acid</td>
<td>Potent acids and alkalis as previous</td>
</tr>
<tr>
<td></td>
<td>Sodium hydroxide</td>
<td></td>
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</tbody>
</table>
and cause coagulative necrosis of the affected tissue. Immediate irrigation is recommended. Excision of non-viable tissue should be considered early in the course of injury.

- HFA: a large proportion of the population is at risk from HFA given its widespread use in the household setting. HFA has the potential to cause significant local and systemic effects despite a small contact wound. The onset of local effects are dependent on the concentration of the HFA. The injury caused by HFA causes liquefactive necrosis and causes interruption in the surrounding cellular physiology. The injury results in hypocalcaemia and hypophosphataemia and, potentially, cardiac arrhythmia. The fluoride component is a metabolic toxin affecting nerve transmission. Haemodialysis and cation exchange resins have been reported for removal of absorbed fluoride.

- Phosphoric acids: such chemicals are found in fertilisers and explosives, thus generally causing injury in the industrial setting. White phosphorus ignites in the presence of oxygen and thus immediate removal is necessary. Particles may be identified with the aid of ultraviolet light or 0.5% copper sulphate solution and should be removed from the wound. Copious irrigation should be performed and the patient should be transported with a wet towel covering the injury. Systemic consequences including hypocalcaemia, hyperphosphataemia and cardiac arrhythmia have been previously reported.

- Alkali: act as proton acceptors and classically cause progressing injury despite the removal of the harmful agent. As discussed above, alkanes cause liquefactive necrosis, allowing progression to deeper tissues. Initially alkali burns seem superficial, but may progress to full thickness within 48–72 hours. Brushing of residues and irrigation provides early control. Referral to a centre of higher acuity and consideration for wound

Personal protection
   - Protective equipment including gloves, googles

Early management
   - Remove solid debris, carefully brush if necessary
   - Copious irrigation 30 to 120 mins as necessary

Wound evaluation
   - Assess microcirculation and sensation to gauge depth
   - Treat as full thickness until proven otherwise

Primary and secondary survey
   - Assess airway, breathing, circulation

Rapid assessment
   - Assess agent (concentration, phase and amount), location of burn, duration of insult

Systemic evaluation
   - Consider metabolic, electrolyte and thermostatic disturbances as potential sequelae of injury or treatment

Compromised
   - Support compromised system until emergency services arrive

Stable
   - Normal primary, secondary and systemic evaluation
   - Wound; full thickness explicitly excluded, burns <1% of BSA, no vital regions injured (ie. face, eyes, genitalia

Complete early management
   - Complete early management and treat as outpatient

Referral to centre of higher acuity for further assessment

Figure 2. Algorithm for the assessment and early management for acute cutaneous chemical burns
debridement should be done given the nature of continuing injury.\textsuperscript{13} Common harmful alkali agents include sodium, ammonium, calcium and potassium salts.

**Conclusion**

Chemical burns are common and may cause significant physical, psychological and economic burdens on patients. Complete rapid assessment of the cutaneous injury and clinical status of the patient is essential in establishing the need for prompt referral to centres of higher acuity. Appropriate early management is crucial in reducing the period of patient morbidity. Current guidelines suggest water irrigation is the safest, most efficacious and readily available treatment option in the early stages of care of chemical burns.

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