Chemical casualties — recognition and management

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Abstract

Poisoning with chemical agents was once thought to be confined to the battlefield. However, over the past decade there has been an increase in the use of chemical weapon agents and toxic industrial chemicals as weapons of terror. As well as use during conflict, these poisons have been used in other attacks with deadly effects. These agents require particular treatments that fall out with standard medical practice to reduce harm and prevent contamination of medical treatment facilities. The risk of a mass casualty incident with a deliberate or accidental release is a possibility.

Keywords Antidotes; chemical weapons agents; nerve agents; toxic industrial chemicals; toxins

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Learning objectives

After reading this article, you should be able to:

- perform an initial assessment on any patient presenting with suspecting poisoning due to chemical weapons or toxic chemicals
- recognize the clinical features of the different types of chemical agents
- consider the need for decontamination for chemical casualties

Introduction

Chemical casualties and poisonings result from exposure to various hazardous substances. The threat from toxic industrial chemicals (TICs) and chemical warfare agent (CWA) exposure is increasingly relevant in both civilian and military operational contexts, with an overlap in the context of asymmetric non-conventional warfare and insurgent activities. Recent notable cases include the chlorine truck bombs in Baghdad (2007), gas attacks on girls' schools in Afghanistan (2010 onwards), the use of nerve agents and chlorine in Syria (2013 onwards), the use of VX to assassinate Kim Jong-nam in Malaysia (2017), and the Novichok attack in Salisbury (2018).

The attribution of these threats can be considered on a spectrum from occupational exposure to TICs, transport accidents, suicide attempts, to deliberate release of CWAs. Overlap between TICs and CWAs exist, related to the 'threshold of intent' to cause harm. Some chemicals may have legitimate industrial uses (such as cyanide, chlorine, organophosphate pesticides), but may also be used intentionally to harm.[1]

Exposure to TICs and CWAs have the potential to cause mass casualties, and some agents (particularly military chemical weapons) have exceedingly high toxicity. Rapid assessment of affected patients and timely initiation of specific definitive treatment is vital for good outcomes.[2]

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Assessment of the chemical casualty

An environmental or chemical hazard exposure incident should be suspected if there are unusual or unexplained symptoms such as altered vision, headache, chest tightness, non-thermal burns, eye pain, excessive secretions or dyspnoea. Additionally, if there are multiple patients with similar non-traumatic symptoms and signs, or symptoms involving incident response or prehospital staff then a chemical hazard should be considered. Traumatic injuries may also present simultaneously in a chemical casualty, and the presence of a known chemical exposure should not distract from prioritized management of trauma.

Rapid assessment of clinical features is important to identify possible agents, and to determine appropriate specific management. The type of chemical agent can be identified by toxidromes and can be generally classified by mechanism of action into the following groups: *pulmonary; vesicant; asphyxiant;* and *nerve*.

Pulmonary agents produce respiratory irritant and corrosive features affecting the lungs. Vesicant agents produce irritant and corrosive features that are specific to the route of exposure (i.e. skin, ocular, respiratory), as well as systemic effects in cases of severe exposure (sulphur mustard can produce bone marrow failure and immunosuppression, hydrofluoric acid burns results in hypocalcaemia and arrythmias). Asphyxiant agents produce a rapid 'knockdown' effect resulting from inhibition of cellular aerobic respiration. Nerve agents and organophosphate poisons produce a cholinergic toxidrome from inhibition of acetylcholinesterase (AChE). These agent classes and examples are shown in Table 1.[1,2]

Environmental cues

Scene assessment from bystanders or prehospital teams may provide information on route of exposure, or features such as smells. There are certain distinctive smells that are suggestive of TICs or CW agents:[1]

- cyanide — bitter almonds
- sulphur mustard — garlic or horseradish
- lewisite — geraniums
- hydrogen sulphide — bad eggs
- chlorine — swimming pool
- cyanide — bitter almonds.

Chemical agent types, examples, and features

Class of agent	Clinical features	Examples	Remarks
Pulmonary	Eye pain Airway irritation Pulmonary oedema	Chlorine, Phosgene, Ammonia	Irritation of respiratory tract Also referred to as choking agents
Vesicant	Eye pain Erythema Blistering	Sulphur mustard, Lewisite, Phosgene oxime	DNA damage or chemical burns Also referred to as blistering agents
Asphyxiant	Rapid onset Hypoxemia Respiratory arrest	Cyanides, Hydrogen sulphide, Methaemoglobin formers	Inhibition of aerobic respiration Also referred to as blood poisons
Nerve	Miosis Weakness Bronchospasm Bronchorrhoea Bradycardia Seizures	Tabun (GA), Sarin (GB), Soman (GD), Cyclosarin (GF), VX, Novichok	Inhibition of acetylcholinesterase Also includes organophosphate pesticides (cholinergic toxidrome)

Table 1

Persistency of agent

It is relevant to consider the physical properties of the agent as patients may still be contaminated at presentation to hospital, and at risk of ongoing exposure. Exposure to inadequately decontaminated patients may also result in secondary cases, including hospital staff. Contamination may be external or internal and may involve wounds. The persistency of an agent determines the extent of decontamination required. Gases, volatile liquids or vapours are non-persistent or have low persistency. Less volatile liquids and particulate or dry material are more persistent and require more extensive decontamination.

Management

The management of chemical casualties and patients includes decontamination and a rapid assessment followed by supportive and definitive treatments. Staff should ensure they are wearing appropriate personal protective equipment if the patient has not been decontaminated. The general approach should be aligned with a rapid ABCDE assessment with resuscitative interventions. Supportive treatments include oxygen, fluids and analgesia if indicated, and prophylactic broad-spectrum antibiotics for penetrating trauma. Definitive treatment for CBRN patients includes specific antidotes, antibiotics and surgery.

Decontamination

The extent of decontamination required is dependent on the physical property of the hazard, summarized in Table 2.[1,3] These actions should ideally be performed by prehospital at the scene but may need to be performed on arrival at the hospital if decontamination is incomplete or not performed (for example, where there is a lack of resources, the patient is unstable, or there is a major medical incident).

The affected casualty should be removed from the environment, moved to a ventilated area and clothing removed. This should remove most gas and vapour hazards. Liquid-type hazards should first undergo dry decontamination with an adsorbent material ('blot-bang-rub', using materials such as Fuller's earth, paper towels or microfibre pads), followed by wet decontamination ('rinse-wipe-rinse', with copious amounts of warm water). Eyes should also be irrigated. Solid and particulate hazards should undergo immediate wet decontamination. If there are also open wounds, then wound decontamination should be considered.

Decontamination guidelines

Type of hazard	Clinical features	Remarks
Gas	Move to ventilated area	Remove clothing if patient is non-ambulatory
Vapour	Remove clothing	No further decontamination necessary
Liquid	Remove clothing, dry decontamination (apply adsorbent material), wet decontamination	Wet decontamination can be deferred if ambulatory
Solid or particulate	Apply face mask to casualty, remove clothing, immediate wet decontamination	Consider wound decontamination

Table 2

Treatment overview for cyanide exposure

Drug	Dose	Remarks
Sodium thiosulphate (IV)	Adults and children aged 12 years or over: 12.5 g IV over 10 min Children (under 12 years of age): 500 mg/kg (0.8 ml/kg of 50%, 1.6 ml/kg of 25%)	Can give in cases of possible cyanide exposure
Dicobalt edetate [Kelocyanor®] (IV)	Adults and children aged 12 years or over: 20 ml of 1.5% dicobalt edetate solution (300 mg) over 1 min followed by 50 ml of 50% glucose Children (under 12 years of age): 4 mg/kg over 1 min followed by 2 ml/kg bolus of 10% glucose	For confirmed cyanide exposure only
Hydroxocobalamin [Cyanokit®] (IV)	Adults and children aged 12 years or over: 5 g over 15 min; repeated once, if necessary, over 15 min to 2 h Children (under 12 years of age): 70 mg/kg (not exceeding 5 g) over 15 min; repeated once, if necessary, over 15 min to 2 h	For confirmed cyanide exposure only Give in dedicated IV line

Table 3

Definitive treatment

Pulmonary: Give supplemental oxygen if required and consider inhaled salbutamol and steroids. If there are respiratory symptoms, perform ABG, CXR, PEF, and repeat if necessary. In severe cases, ventilation may be required. Patients should be admitted for 24 h for observation if symptoms persist beyond period of exposure, or if there is pre-existing respiratory disease. Complications include ARDS and secondary infection.

Vesicant: Rapid wet decontamination is the mainstay of management. In mustard exposure, symptoms may be delayed. Monitor FBC in mustard exposure as immunosuppression can occur. Specific treatments for Lewisite include dimercaprol (British Anti-Lewisite) at 3 mg/kg IM 4-hourly for 2 days, 6-hourly on day 3, then 12-hourly for the next 10 days (alternatives include succimer and Unithiol). Blisters may be deroofed, but caution should be taken as the blister fluid may contain arsenic. Hydrofluoric acid burns should be treated with calcium gluconate, both topically and systemically if indicated. Fluid therapy is likely to be required if there are large surface area blisters or burns.

Asphyxiant: The main treatment for asphyxiant exposure (such as cyanides or hydrogen sulphides) is to remove from the toxic environment and give oxygen therapy. Antidotes may not be

Treatment overview for nerve agents and organophosphate exposure

Drug	Dose	Remarks
Anticholinergic		
Atropine (IV/IM/IO)	Adults and children aged 12 years or over: 2—4 mg Children (under 12 years of age): 50—75 μg/kg Repeat every 5 minutes until 'atropinizsed'	Increase doses by serial doubling to achieve reversal and clinical endpoint
Oxime		
Pralidoxime (IV)	Loading dose: 30mg/kg (∼2 g in adults) over 30 minutes Continuation: IV infusion at 8 mg/kg/hr (∼0.5 g/h in adults) and continue for 12—24 hours	If there is deterioration on withdrawal, restart the pralidoxime infusion or switch to obidoxime
Benzodiazepines		
Diazepam (IV)	10—20 mg in adults; 0.1—0.3 mg/kg in children	Benzodiazepine choice depends on local guidelines. Give before oxime therapy if seizures are present
Lorazepam (IV)	4 mg in adults; 0.1 mg/kg in children	
Midazolam (IV)	5—10 mg in adults; 0.05—0.16 mg/kg in children	

Table 4

Treatment overview for chemical casualties

Class of agent	Treatment overview
Pulmonary	Oxygen, bronchodilators, inhaled steroids, ventilation with PEEP
Vesicant	Wet decontamination and irrigation, deroof ruptured blisters, fluid therapy if large surface area affected. Lewisite (arsenicals) – consider BAL (first line), DMSA, or DMPS. Hydrofluoric acid – calcium gluconate
Asphyxiant	Oxygen and supportive measures. Cyanide – consider sodium thiosulphate, dicobalt edetate (followed by IV glucose), hydroxocobalamin, or sodium nitrite. Methaemoglobinaemia – methylene blue
Nerve	Atropine IV boluses to be repeated with serial doubling until bronchospasm, bronchorrhoea, and bradycardia are reversed. Following administration of atropine, pralidoxime (or alternative such as obidoxime) should be given. Control convulsions with benzodiazepines. May require ventilation

Table 5

required if patients recover spontaneously. Metabolic acidaemia can be corrected with sodium bicarbonate IV. Cyanide poisoning can be treated with IV sodium thiosulphate 12.5 g, or in severe cases dicobalt edetate (Kelocyanor®) 300 mg followed by 50 ml of 50% glucose or alternatively hydroxocobalamin (Cyanokit®) 5 g over 15 min. Other options include prehospital inhaled amyl nitrite, or IV sodium nitrite 300 mg IV over 10 min (contraindicated in trauma or smoke inhalation). A summary of drugs for treating cyanide exposure are shown in Table 3. Severe hydrogen sulphide exposure may result in methaemoglobinaemia which can be treated with methylene blue at 1–2 mg/kg IV.

Nerve: Immediately administer IV bolus of atropine with an aim to reverse bronchospasm (clear lungs), bronchorrhoea (minimal secretions) and bradycardia (heart rate great than 80/min and adequate blood pressure). Increase repeat doses by serial doubling (2 mg, 4 mg, 8 mg, 16 mg, etc.) to achieve reversal of symptoms – extremely high doses of atropine may be required (over 1.5 g has been recorded). Pralidoxime IV 2 g should be given to reactivate inactivated AChE. Obidoxime is more effective in tabun exposure. Seizures should be treated with benzodiazepines. Patients may require prolonged ventilation. A summary of drugs for treating nerve agents and organophosphate exposure are shown in Table 4 and Table 5 provides an overview of treatments for chemical casualties. ◆

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FURTHER READING

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