Chemical and Nuclear Agents

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PERSPECTIVE

Casualties involving chemical and radiologic or nuclear agents have traditionally been associated with military armed conflicts. However, over the past 25 years, with the increased production and distribution of industrial chemicals, as well as the escalating threat of terrorist use of weapons of mass destruction, management of casualties from chemical, biological, radiologic, nuclear, and high-yield explosive events has increasingly become the responsibility of the EP.

Initially approved in 1999 and revised in 2006, the American College of Emergency Physicians (ACEP) issued a clinical policy statement that recognized the risk posed by accidental or intentional release of chemical and nuclear hazardous material (HAZMAT).

In another clinical policy statement, the ACEP also encouraged EPs to assume a primary role in the medical aspects of planning, management, and patient care during disasters, including those involving chemical, radiologic, and nuclear agents.

BASIC PRINCIPLES OF MANAGING CONTAMINATED PATIENTS

Management of casualties from chemical and radioactive agents can be complicated by one or more factors, such as the number of patients, type of agent or agents involved, severity of the exposure, and the availability of pharmaceutical and human resources. A current and accurate hazard vulnerability analysis is essential for an optimal response to a specific identified hazard. However, because no facility can prepare for every possible factor and scenario, adherence to several basic principles may facilitate effective management of contaminated patients.

COMMUNICATION AND MOBILIZATION

Most events involving chemical or radioactive agents are rapidly identified by emergency services. Notification of health care facilities of a HAZMAT incident and casualties must also take place rapidly because 50% to 80% of the acute casualties will arrive at the closest health care facilities within 90 minutes following an event. Some casualties may leave the scene under their own power and go to a nearby health care facility, even before other patients arrive via emergency medical services.

KEY POINTS

- Decontamination should take place immediately, before initial treatment and evacuation of exposed patients.
- For patients exposed to nerve agents, atropine and pralidoxime chloride should be administered rapidly.
- Treatment of blast injuries and emergency medical conditions should precede specific treatment of radiation exposure.

EPIDEMIOLOGY

Tens of thousands of chemicals are manufactured, transported, and used every day. The 1984 Bhopal, India, disaster revealed the dangers posed by chemical agents. A 2008 U.S. Department of Health and Human Services database of 14 states reported more than 15,000 chemical-related events and over 4500 casualties. Since World War I, chemical agents have also been used intentionally on civilian and military personnel, most recently in Japan in 1994-1995, in Russia in 2002, and in Iraq in 2007. Of the 13 categories of chemical agents recognized by the U.S. Centers for Disease Control and Prevention, the four principal categories are nerve, vesicant, blood, and pulmonary agents (Box 137.1).

Detonation of an atomic bomb over Hiroshima, Japan, in 1945 heralded the evolution of a new hazardous agent, radioactive material. Showcased by the 1987 accidental exposure of cesium 137 in Goiânia, Brazil, the threat from radiologic material has continued to increase with the proliferation of medical devices and radiation therapy. Though reduced by the end of the Cold War, the threat from nuclear agents continues to persist in light of the accidents at Chernobyl, Ukraine, in 1986 and Tokaimura, Japan, in 1997, as well as the acknowledgment that dozens of nuclear devices are missing.

Management of casualties from chemical and radioactive agents can be complicated by the types of agents and exposure, in addition to specialized logistic, safety, and security issues. Emergency physicians (EPs) must be familiar with the basic principles of managing contaminated patients and initial treatment of the principal chemical and radioactive agents.
Concise and accurate communication of prehospital events is also crucial for an effective response. Collection of pertinent strategic details can guide mobilization of appropriate resources, including activation of an emergency management plan and the incident command system, consultation with material safety data sheets and HAZMAT experts, compilation of antidotes and medical equipment, and preparation of triage, decontamination, and personnel protection equipment.

**DECONTAMINATION**
Decontamination should take place immediately, before initial treatment and evacuation of exposed patients. Although decontamination is usually completed at the scene before transportation, exposed and potentially contaminated patients may go on their own to nearby health care facilities. In addition to requiring primary decontamination and triage, these patients may secondarily contaminate existing patients and medical personnel and thus create additional casualties and diminish the response by the affected facility.

Removal of contaminated clothing can eliminate 70% to 90% of HAZMAT. Once completed, patients should shower—or be showered if incapacitated—with copious amounts of tepid water. Several adjuncts, such as hypoallergenic liquid soap, may be helpful. Other adjuncts, including hard brushes and dilute additives such as bleach, are unlikely to provide additional benefit and, in some scenarios, could be harmful. Contaminated clothing and special items such as valuables and firearms should be labeled and securely contained to prevent accidental or continued secondary contamination, as well as for possible forensic analysis during a HAZMAT or criminal investigation.

**SECURITY**
Security personnel at health care facilities should be engaged promptly because their primary responsibility is to protect existing patients, staff, and the health care facility from contamination and distraction from avoidable crowds by controlling all access points to the facility. This responsibility may entail limiting visits from arriving friends, family, and other third parties, as well as sequestering contaminated patients.

Security personnel should control traffic flow to and within the facility. Incoming patients should be directed to designated triage and decontamination sites, and the arrival of hospital personnel should be expedited. Patients awaiting further evaluation and disposition may be directed to secondary triage, holding, and treatment areas. Security personnel should coordinate with law enforcement and government agencies, safeguard valuables, and maintain the chain of custody of firearms and forensic evidence.

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**BOX 137.1 Categories of Chemical Agents**

<table>
<thead>
<tr>
<th>Categories</th>
<th>Examples</th>
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<tbody>
<tr>
<td>Biotoxins</td>
<td>Metals</td>
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<tr>
<td>Blister agents/vesicants</td>
<td>Nerve agents</td>
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<tr>
<td>Blood agents</td>
<td>Organic solvents</td>
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<tr>
<td>Caustics</td>
<td>Riot control agents</td>
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<tr>
<td>Choking/pulmonary agents</td>
<td>Toxic alcohols</td>
</tr>
<tr>
<td>Incapacitating agents</td>
<td>Vomiting agents</td>
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<tr>
<td>Long-acting anticoagulants</td>
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Nerve agents are potent anticholinesterases that can be divided into three categories: quaternary ammonium alcohols, carbamates, and organophosphates. Quaternary ammonium alcohols are used as disinfectants. Carbamates are found in household insecticides. Also in insecticides, some organophosphates have been designed for use as warfare agents. The G series consists of tabun (GA), sarin (GB), soman (GD), and cyclosarin (GF). The V series, which includes VE, VG, VM, and VX, was developed 20 years later and is more potent and persistent, which means that it is not removed as easily.

Absorbed through inhalation and skin contact, nerve agents irreversibly bind and inactivate acetylcholinesterase (AChE) receptors, thereby leading to excessive accumulation of acetylcholine and overstimulation of nicotinic and muscarinic receptors. Though dose dependent, the effects from inhalation begin within seconds and peak within minutes, whereas the effects from skin exposure may take minutes to hours to become evident.

**PRESENTING SIGNS AND SYMPTOMS**
The earliest symptoms from inhalation are rhinorrhea and dim vision secondary to miosis, and those from skin contact are anxiety and fasciculations. With greater exposure, the classic symptoms of a cholinergic crisis become apparent. Hyperactivity of muscarinic receptors leads to lacrimation and salivation, bronchoconstriction, and vomiting and diarrhea. Hyperactivity of nicotinic receptors produces diaphoresis, tachycardia, hypertension, and muscle weakness. With severe or prolonged exposure, end-organs fatigue and fail, and convulsions, incontinence, flaccid paralysis, and apnea ensue.

**TREATMENT**
Atropine and pralidoxime chloride (2-PAM chloride) should be administered rapidly. When intubating patients, use of succinylcholine should be avoided. Atropine, a competitive cholinergic blocking agent, can mitigate muscarinic symptoms and should be administered every 5 to 10 minutes until
Vesicant agents are highly penetrative oily substances that induce blister formation and include mustard agents and organic arsenicals. Mustard has a wide range of effects on the eyes, skin, lungs, nervous system, and bone marrow. It has been produced and stockpiled by multiple countries. Lewisite is an arsenical often combined with mustard, smells like geraniums, and is associated with renal failure and hepatic necrosis.

Absorbed through inhalation and topical contact, mustard agents are alkylating substances that damage DNA and nucleic acid synthesis. The mechanism of toxicity for lewisite is not known. Symptoms are dependent on the agent, as well as the severity and route of exposure. The effects of lewisite begin within seconds to minutes. DNA damage from mustard agents begins within minutes, but some symptoms may not be manifested for hours.

**Presenting Signs and Symptoms**

For mustard agents, the earliest symptoms are irritation and burning of exposed areas, such as the eyes and upper airway. Subsequent symptoms include pruritus and erythema of the skin, especially warm, moist locations such as the axillae and groin. With higher doses or prolonged exposure, shortness of breath, bulla formation, and corneal damage develop. Vomiting, diarrhea, bone marrow suppression, and seizures are associated with severe exposure and imply a poor prognosis.

Exposure to lewisite also produces eye, lung, and skin symptoms, which unlike those with mustard agents, begin immediately. With severe exposure, increased capillary permeability can lead to substantial loss of intravascular volume and end-organ damage.

**Treatment**

Unless completed within minutes of exposure, decontamination will not prevent tissue and DNA damage. It can, however, reduce or prevent ongoing exposure and secondary contamination. Only lewisite has a specific antidote, the chelating agent British antilewisite (BAL). BAL can be toxic itself, may contain peanut oil, and thus should not be given to patients with peanut allergy.

Additional treatment is primarily supportive. Eye injuries should be managed with lubrication and ophthalmologic antibiotics. Respiratory injuries may require oxygen, bronchodilators, and ventilatory support. Skin injuries should be treated similar to thermal burns, including wound care, tetanus prophylaxis, analgesia, and intravenous fluids. However, although fluid loss from mustard agents is less than that associated with thermal burns, lewisite casualties may require aggressive fluid replacement.

**BLOOD AGENTS**

**Pathophysiology**

Blood agents include cyanide and arsenic-based chemicals. Absorbed through inhalation, ingestion, and topical exposure, blood agents disrupt the mitochondrial cytochrome oxidase complex, thereby inhibiting intracellular oxygen use and aerobic metabolism. Cyanide is the most well-known blood agent and is often associated with a pungent odor described as bitter almond. Cyanide is widely used in many industries, such as mining and plastic manufacturing, and can be released during the combustion of numerous natural and synthetic material. It has also been identified as a likely agent for use by terrorists.

**Presenting Signs and Symptoms**

The earliest symptoms begin within seconds to minutes and may include dyspnea, dizziness, nausea, and anxiety. With greater exposure, generalized weakness, diaphoresis, cyanosis, and hypotension may be observed, and with severe or prolonged exposure, dysrhythmias and seizures may progress rapidly to respiratory failure and death.

**Treatment**

Treatment should be initiated immediately and presumptively based on prehospital information and clinical suspicion.
because diagnostic tests are not readily available. Traditionally, definitive treatment has been administration of the three components of the cyanide antidote kit: amyl nitrite, sodium nitrite, and sodium thiosulfate. Hydroxocobalamin, a precursor of vitamin B<sub>12</sub>, has gained acceptance as another antidote because it can be used for prehospital management and has better safety and side effect profiles.28-30

**PULMONARY AGENTS**

**PATHOPHYSIOLOGY**

Pulmonary agents include phosgene and chlorine. Absorbed through inhalation and topical contact, pulmonary agents irritate and cause an inflammatory reaction of the peripheral and central airways. Phosgene is widely used in many industries, released during the combustion of foam plastics, and associated with the smell of newly cut grass. Chlorine is used widely in manufacturing and for purifying water and has its own distinctive odor. It was the first chemical warfare agent and has recently been involved in several large-scale disasters and terrorist attacks.20,31

**PRESENTING SIGNS AND SYMPTOMS**

Symptoms begin within minutes and may start with eye irritation, rhinorrhea, coughing, and dyspnea. With greater exposure, skin irritation, vomiting, and shortness of breath may be observed, and with severe exposure, blistering and pulmonary edema may develop.32

**TREATMENT**

Treatment is primarily supportive. Eye injuries should be managed with ophthalmologic antibiotics and follow-up. Skin injuries should be treated similar to thermal burns, including wound care, tetanus prophylaxis, and analgesia. Respiratory injuries may require oxygen, bronchodilators, and endotracheal intubation and ventilatory support. Corticosteroids are recommended, and nebulized lidocaine and sodium bicarbonate may be beneficial.31

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**Table 137.2 Types of Blast Injuries**

<table>
<thead>
<tr>
<th>TYPE</th>
<th>MECHANISM</th>
<th>EXAMPLES/EFFECTS</th>
</tr>
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<tbody>
<tr>
<td>Primary</td>
<td>Direct pressurization</td>
<td>Rupture of tympanic membranes, lungs, viscera</td>
</tr>
<tr>
<td>Secondary</td>
<td>Projectiles</td>
<td>Penetrating trauma from fragments</td>
</tr>
<tr>
<td>Tertiary</td>
<td>Secondary trauma</td>
<td>Structural collapse, being thrown by the blast wind</td>
</tr>
<tr>
<td>Quaternary</td>
<td>Other</td>
<td>Burns, radiation, hazardous materials</td>
</tr>
</tbody>
</table>


**PRESENTING SIGNS AND SYMPTOMS**

Within a few hours of exposure to greater than 1 Gy, three acute radiation syndromes may develop. Hematopoietic syndrome results in pancytopenia and a predisposition to infection, bleeding, and poor wound healing. Gastrointestinal syndrome results in abdominal cramping, vomiting, and diarrhea. Early intractable vomiting and bloody diarrhea imply a poor prognosis. Cerebrovascular syndrome results in confusion, ataxia, and seizures.37

**TREATMENT**

After blast injuries and emergency medical conditions are treated, several radiation exposure-specific treatments should be discussed and initiated under the guidance of radiation safety, nuclear medicine, and hematology consultants. Any necessary surgeries should be performed within the first 24 to 36 hours before patients become immunologically incompetent. Cytokine therapy and the administration of chelating or blocking agents, such as potassium iodide and Prussian blue, should be considered.37

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**Radiologic and Nuclear Agents**

**PATHOPHYSIOLOGY**

Radiation, regardless of type, causes injury through the production of charged water molecules and ionization of DNA. Because of its cellular effects, radiation can affect every organ, especially the hematopoietic, gastrointestinal, and central nervous systems.

Irradiation and external contamination by a radiologic agent may occur as a result of the surreptitious placement of a radiation emission device or detonation of a dirty bomb.3,34 A dirty bomb involves the use of conventional explosives to disperse radioactive material, such as iodine 131. After decontamination, treatment of blast injuries should precede treatment of radiologic injuries because immediate death from the radiation is unlikely35,36 (Table 137.2). If free of particulate matter and shrapnel, patients are unlikely to pose a significant threat to other patients and medical personnel.

Irradiation and external contamination by a nuclear agent may occur during transportation of HAZMAT or a nuclear reactor accident. Though an unlikely scenario, detonation of a nuclear device would involve catastrophic levels of radiation.
Follow-Up, Next Steps in Care, and Patient Education

Patients with the mildest of exposure to a chemical or radiologic or nuclear agent may be eligible for discharge after confirmation of the agent and consultation with an appropriate HAZMAT expert. However, because the majority of chemical and radiologic or nuclear agents are associated with prolonged symptoms and delayed effects, most patients should be admitted. Patient disposition should also be coordinated with responding law enforcement and government agencies for possible interview during a HAZMAT or criminal investigation.

SUGGESTED READINGS


REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.


