Patient

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Chlorine gas

What is chlorine gas injury?[1]

Chlorine gas has several uses in water purification, sanitation, and industrial applications. However, it is a toxic inhalation hazard agent. Inhalation of chlorine gas, based on the concentration and duration of the exposure, causes a spectrum of symptoms, including lacrimation, rhinorrhoea, bronchospasm, cough, dyspneoa, acute lung injury, and death. Survivors develop signs of pulmonary fibrosis and reactive airway disease. Despite the use of chlorine gas as a chemical warfare agent since World War I and its known potential as an industrial hazard, there is no specific antidote.

Nature of chlorine[2]

Chlorine exists as a gas at normal temperature and pressure. The gas is pressurised and cooled to liquid form for storage and shipping. When released, it rapidly forms a yellow-green gas that stays close to the ground and spreads rapidly. Chlorine gas is not flammable but it can react explosively with other chemicals such as turpentine and ammonia. It can be recognised by its pungent, irritating, bleach-like odour which usually provides warning of exposure.

Routes of chlorine exposure[3] [4] [5]

Accidental exposure

Chlorine is widely used in industry. It is used as a bleaching agent in the manufacture of paper and cloth, in the manufacture of pesticides, rubber, PVC and solvents, in drinking water and in swimming pool water for purification and as part of the sanitation process for industrial waste and sewage. In the UK over 1.6 million tons of chlorine are produced annually.

Most acute exposures are now from industrial spills, chemical mixing errors and industrial accidents, and these have led to injuries and deaths. Chronic exposure to lower levels has occurred in workplace and public spaces such as swimming pools. Domestic exposure has often resulted from the mixing of chlorine bleach with acidic washing agents such as acetic, nitric and phosphoric acid.[6] [7]

Deliberate poisoning

Chlorine was first used as a weapon during World War I.[8] It was initially lethal to large numbers of troops at Ypres in 1915. Respiratory protection was swiftly developed but did not protect most civilians. At one point it was so prevalent on the Western front that respirators were developed for horses and carrier pigeons. Its use in war was subsequently banned by consensus at the United Nations (UN).[9]

The widespread use of chlorine in industry means that production and storage cannot be banned, even in regimes where chemical weapons have been surrendered. Therefore, it is available to those seeking to weaponise it. Its re-emergence has been widely reported in conflict zones, including Sri Lanka, Iraq and Syria, and many incidents have been verified by the UN.

Chlorine gas has been used recently in besieged urban areas, where it seeps down into cellars and drives the population out into the open. Its widespread storage and transportation also make it susceptible to terrorist attack, leading to spills, as has occurred several times in Iraq. Doctors in and beyond conflict zones therefore need to be aware of the management of chlorine gas exposure.

Chlorine toxicity[4]

Humans can detect very low levels of chlorine gas. The threshold concentration for detection of the odour is around 0.2 parts per million (ppm). Immediate symptoms relate to concentration:

- At 1-3 ppm, there is mild mucous membrane irritation of eyes, nose and throat that can usually be tolerated for about an hour.
- At 5-15 ppm, there is moderate mucous membrane irritation. The sensation is of irritating exposure.
- At 30 ppm there is chest pain, shortness of breath, and cough.

- At 40-60 ppm, toxic pneumonitis and/or acute pulmonary oedema can develop, together with upper airway obstruction, abdominal discomfort and even oesophageal perforation.
- Concentrations above 400 ppm are usually fatal over 30 minutes.
- Above 800 ppm, fatality ensues within a few minutes.
- Liquid exposure causes corneal burns and ulceration, and dermatitis with blistering.
- Chronic low-level exposure typically causes skin and mucous membrane irritation and, typically, chronic respiratory symptoms.

Acute chlorine gas exposure in civilian incidents presents with acute respiratory features and irritation of the eyes and throat. The development of pulmonary oedema or acute respiratory distress syndrome (ARDS) is relatively rare with civilian exposure when compared to military experience in the First World War.[10]

Pathophysiology[11]

Chlorine gas is classed as a pulmonary agent or choking agent (others include phosgene, diphosgene and chloropicrin). Once inhaled, it diffuses into the respiratory epithelium, where most damage is initiated by its dissolution into hydrochloric and hypochlorous acids. Further damage occurs with activation of inflammatory cells and subsequent release of oxidants and proteolytic enzymes. Hydrochloric and hypochlorous acids also target the epithelia of the ocular conjunctivae, leading to acid injury to the eye.[12]

The intermediate water-solubility of chlorine means it is mostly absorbed in the conducting compartment of the respiratory tract, from the nose to the level of the bronchi, and higher exposure is needed to cause alveolar damage. High concentrations of chlorine (800 ppm or more) cause mixed airway and alveolar damage. Where alveolar damage occurs its contribution to the clinical picture is usually less pronounced than that of upper airway damage, with a high prevalence of signs of obstructive impairment. There is also evidence of damage to respiratory/bronchial smooth muscle, although this appears to be reversible.[13]

Chlorine gas injury symptoms and presentation

The upper airways and eyes are irritated at low levels of exposure. At higher levels, the nasopharynx and larynx are injured. At very high levels, alveolar damage occurs rapidly. Pulmonary oedema is the most significant life-threatening effect.

The level of harm is influenced by victim factors (age, current lung health, presence of bronchospasm response, exertional state and metabolic rate, history of smoking) and environmental factors (intensity and duration of exposure, quality of ventilation in the space in which exposure occurs). Greater exposure is associated with greater potential harm.[12]

Immediate symptoms[2]

This depends on the level of exposure. During or immediately after exposure to dangerous concentrations of chlorine, the following signs and symptoms are typical:

- **Eyes**: burning pain, watering, redness, blurred vision. Chlorine dissolves on the surface of the eye to produce acidic eye injury.
- **Skin**: burning pain, redness, and blisters on the skin if exposed to gas. Skin injuries similar to frostbite can occur if exposed to liquid chlorine.
- **ENT**: burning sensation in the nose, throat and eyes. Irritation of the larynx by very large concentrations can cause sudden laryngeal spasm or oedematous obstruction, which can be fatal.
- **Pulmonary**: coughing with substernal aching, chest tightness, shortness of breath, and wheeze. These may appear immediately if high concentrations of chlorine gas are inhaled, or they may be delayed if low concentrations of chlorine gas are inhaled. Pulmonary oedema may occur early but is more commonly delayed for a few hours. Haemoptysis may occur (it is more usually a feature of vesicant gas poisoning). Early cyanosis carries a near-hopeless prognosis.
- **Gastroenterological**: nausea and vomiting, oesophageal perforation at high levels.
- **Neurological**: headache, disorientation.

These symptoms are not specific for chlorine; many are also features of exposure to other chemical agents such as phosgene and tear gas, and to some neurological agents. For those exposed, the most obvious clues to chlorine as the causative agent are the characteristic smell of chlorine and the sight of the yellow-green, dense gas at ground level.

Latent symptoms [11] [12]

The presence and speed of development of pulmonary oedema depend on exposure intensity. Patients present with worsening respiratory distress. If pulmonary oedema is to develop, it usually does so within 6-24 hours, although after very high exposure it can develop in minutes (with extremely poor prognosis). Oedema fluid, usually frothy, is secreted from the bronchi, and may leak from the mouth and the nostrils.

- The most prominent symptom is dyspnoea, with or without chest tightness.
- Patients may become cyanosed.
- The high volume of plasma-derived oedema fluid in the lungs (up to one litre per hour) may lead to hypovolaemia and hypotension.
- Early hypoxaemia has a poor prognosis.

Fatalities [12]

At very high levels of exposure, death occurs in minutes to hours from respiratory failure, hypoxaemia, hypovolaemia, acute respiratory obstruction, alveolar destruction or a combination of these. Acute pulmonary hypertension, pulmonary vascular congestion, and burns of the upper and proximal lower airways contribute. Hypoxia and hypotension indicate a poor prognosis, as does development of pulmonary oedema within four hours of exposure.

In survivors, resolution commences within 48 hours.

Chronic low-level exposure

Chronic exposure to relatively low levels of chlorine gas tends to cause chronic low-level symptoms - particularly:

- Acne and skin redness.
- Eye irritation - red eyes, tearing, blepharospasm.

- ENT irritation: chronic sore throat, rhinorrhoea, hypersalivation, stridor.
- Persistent cough and wheeze, typically a chronic obstructive pulmonary disease (COPD)-like presentation which may show some reversibility.
- Tooth corrosion.
- Vague chest pain with reduced exercise tolerance.
- Pulmonary oedema may develop and, sometimes, haemoptysis.

Repeated exposure to chlorine in the pool has been postulated as the cause of an excess of asthma among swimmers.[14] In atopic adolescents, the risk factor of allergic rhinitis and asthma appears to be dose-dependently augmented by chlorinated swimming pool attendance.[15]

Differential diagnosis[11]

The symptoms of chlorine gas exposure are nonspecific, although the smell and sight of the gas are diagnostic if the patient can give a history. Other possible causes of similar symptoms include:

Other pulmonary irritant agents

- Phosgene gas is distinguished by its odour of newly mown hay. Although its effects are similar to chlorine, it is less soluble and therefore reaches the alveoli in greater proportion, making it more lethal.
- Diphosgene is similar to phosgene, with similar effects.
- Chloropicrin (nitrochloroform) is a carcinogenic pesticide used in World War I which is less lethal but particularly causes vomiting and eye irritation, causing those affected to remove respiratory protection.
- Disulfur decafluoride (SF5) has a sulfur dioxide-like odour. It is more poisonous than phosgene but it tends not to produce eye irritation.

Riot-control agents

Tear gas and CS gas produce severe tearing, along with burning sensation and pain, predominantly in the eyes, upper airways, mucous membranes, and skin. The distinctive odour of chlorine is absent. CS gas produces in addition profuse coughing, disorientation, difficulty breathing, and vomiting. However, it does not cause pulmonary oedema at the levels seen in crowd-control situations.[16]

Nerve agents

These cause the production of watery secretions as well as respiratory distress. Other characteristic effects, such as muscle twitching and miosis, help distinguish them from chlorine.

Vesicants

These blistering agents, such as mustard gas, usually produce a delayed respiratory toxicity of the central airways. Vesicant inhalation severe enough to cause dyspnoea typically causes signs of airway necrosis, often with pseudomembrane formation and partial or complete upper airway obstruction. Pulmonary damage usually manifests as haemorrhage rather than oedema.

Investigations[11]

Investigations are of limited value in the immediate care of exposed patients, although some have predictive value in determining severity of outcome.

CXR

Radiological changes may lag behind clinical changes by days, so the chest radiograph may be of limited value, particularly if normal. Hyperinflation suggests toxic injury of the smaller airways with alveolar air trapping. Perihilar infiltrates suggest pulmonary oedema secondary to alveolar-capillary membrane damage. Atelectasis is common.

Arterial blood gases

Both central and peripheral pulmonary damage may produce hypoxia. Low PaO_2 or $PaCO_2$ are early nonspecific warnings of pulmonary oedema. Normal arterial blood gas values at 4-6 hours are predictive of non-lethal outcome. High $PaCO_2$ suggests bronchospasm.

Pulmonary function tests
Peak expiratory flow rate may decrease soon after a massive exposure and helps to assess both degree of airway damage and effect of bronchodilator therapy.

Triage of chlorine gas-exposed patients[12]

In the field, rapid triage of patients may be necessary. It is done according to clinical condition and available treatment:

- **Immediate**: this category is used for patients with pulmonary oedema only if intensive pulmonary care is immediately available; otherwise, they are 'expectant'.
- **Expectant**: patient has pulmonary oedema, cyanosis and/or hypotension. A patient with these signs within four hours of exposure is not expected to survive without immediate, intensive medical care including artificial ventilation.
- **Delayed**: patient is dyspnoeic without objective signs and should be observed closely and re-triaged hourly. If the patient is recovering, discharge 24 hours after exposure.
- **Minimal**: patient is asymptomatic with known exposure and should be observed and re-triaged every two hours. If the patient remains asymptomatic 24 hours after exposure, it is safe to discharge them. If exposure is doubtful and the patient is asymptomatic 12 hours following possible exposure, discharge can be considered.

Chlorine gas injury treatment and management[1] [4] [12]

No antidote exists for chlorine exposure. Treatment is supportive. Various therapies have seemed promising, based on animal studies or case reports. However, these recommendations have been based on low-level quality data.[17]

At the site of exposure, pre-hospital support and stabilisation involve removing victims from the source of chlorine exposure and giving supplemental oxygen. Administering inhaled beta-agonists to control bronchospasm may be considered. Standardised protocols for triaging victims may help to prioritise treatment plans.

Terminating exposure

The casualty should be physically removed from the hazardous environment or, if this is not possible, given respiratory protection. Removal from the source of the poisoning includes removal of contaminated clothing and contact lenses. Decontamination of liquid agent on clothing or skin is essential.

Resuscitation

- **Supplemental humidified oxygen**: is ideally administered via a device generating intermittent or continuous positive pressure.
- **Intubation with or without ventilatory assistance**: may be required. Establishing an airway is especially important in a patient with hoarseness or stridor; they may have impending laryngeal spasm and require intubation. Establishing a clear airway also aids in interpretation of auscultatory findings.
- **Maintain haemodynamic stability**: crystalloid or colloid is equally effective. Intravascular volume should be carefully monitored. There is a danger of hypotension induced by pulmonary oedema or positive airway pressure. Vasopressors may help as a temporary measure until fluids can be replaced.

Supportive care

- **Rest**: is essential for patients suspected of having inhaled any agent that might cause pulmonary oedema. Physical exertion may shorten the latent period and increase severity of respiratory symptoms.
- **Prevent or treat bronchospasm**:
  - Inhaled beta-adrenergic agents are indicated for patients with signs of airway obstruction - eg, wheeze, reduced breath sounds, increased respiratory rate, cough. [18]
  - Early administration of steroids may reduce the risk of pulmonary oedema and is also indicated for bronchospasm. Parenteral administration is preferred, as inhaled routes may result in inadequate distribution to damaged airways. Methylprednisolone 1000 mg, or its equivalent, is given during the first day and tapered throughout the symptomatic period. [12] [19]

- **Assisted ventilation** - continuous positive airway pressure (CPAP) and/or positive end-expiratory pressure (PEEP): reduce the complications of pulmonary oedema:
    - CPAP is spontaneous ventilation with a positive airway pressure maintained throughout the whole respiratory cycle. It may exacerbate hypotension by decreasing thoracic venous return.
    - PEEP maintains airway pressure above atmospheric at the end of expiration and can also be used with spontaneous breathing.
    - If the patient is intubated, protective lung ventilation is recommended, as for acute lung injury. This is low tidal volume ventilation of around 6 mL/kg predicted (not actual) body weight, which is physiologically normal for a healthy person. Low tidal volume ventilation reduces ventilator-associated lung injuries such as hyperinflation, alveolar rupture, pneumothorax and the release of inflammatory mediators. The associated hypercapnia may also have directly beneficial effects.[20]
- **Suction**: is helpful for profuse pulmonary secretions.
- **Diuretics**: have limited value and may predispose the patient to hypotension.
- **Antibiotics**: are not indicated without evidence of an additional infectious process.

Prognosis[11] [12]

Acute exposure

- In survivors, improvement typically begins from 48 hours.
- Most people with mild-to-moderate exposure recover fully in 3-5 days, although some develop chronic problems such as reactive airway disease.
- Smoking and pre-existing lung conditions like asthma increase the risk of long-term complications.[2]
- Those who survive acute severe chlorine inhalation and pulmonary oedema usually recover fully, although residual obstructive-reactive symptoms are more likely.

Chronic exposure

- Long-term sequelae of chronic low-level exposure include increased airways reactivity, chronic bronchitis and recurrent wheezing. This is more severe among those who are older, have smoked and/or have pre-existing chronic lung disease.
- Irritant-induced asthma is sometimes seen after acute occupational exposure. It resembles COPD with a reversible element but it comes on within 24 hours of exposure.[4]

Advice in case of chlorine exposure[2] [11]

- Leave the area where the chlorine is settling/dispersing and get to fresh air. This is highly effective in reducing exposure. If outdoors, go to higher ground if possible, as chlorine is heavier than air and will gather in lower spots.
- If the chlorine release was indoors, get out of the building.
- If exposed, remove clothing. The Centers for Disease Control and Prevention (CDC) advise that you wash your entire body with soap and water, then seek medical care as quickly as possible. Clothing that has liquid chlorine on it should be removed urgently. These items should be cut off the body instead of pulled over the head. If possible, seal the clothing in a plastic bag and seal the first bag in a second plastic bag. Do not further handle the plastic bags.
- If helping other people remove their clothing, avoid touching contaminated areas and be as quick as possible.
- If eyes are burning or vision is blurred, rinse your eyes with plain water for 10-15 minutes. Remove contact lenses before doing so and then discard them. Do not put them back into your eyes. Spectacles can be washed with soap and water, then re-worn.
- If you have swallowed (ingested) chlorine, do not induce vomiting or drink fluids. Seek medical attention straightaway.

History[9] [21]

Gas was envisaged as an effective tool to draw soldiers out of their trenches so as to attack them with conventional weapons. It was first used on 22 April 1915, when 160 tons of chlorine gas drifted slowly over the French trenches, where within minutes it killed more than 1,000 soldiers, injuring approximately 4,000 more. Its effect on morale was equally substantial, and as the war continued other toxic gases like sulfur mustard and phosgene were also used, to devastating effect.

After World War I some claimed that poison gas was a humane weapon, as it did not kill in the numbers that machine guns and artillery did, and there was fierce debate. However, poison gas, including chlorine gas, is now classified as a weapon of mass destruction and is banned by the UN Chemical Weapons Convention.[22]

Dr Mary Lowth is an author or the original author of this leaflet.

Further reading

- Chlorine: health effects, incident management and toxicology; GOV.UK

Authored by: Dr Mary Elisabeth Lowth, FRCGP	Peer Reviewed by: Dr Krishna Vakharia, MRCGP	
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