Phosphorus trichloride (PCI₃)

Information and recommendations for doctors at hospitals/emergency departments

- Before approaching the patient, the first responder must make sure that he does not risk exposing himself to phosphorus trichloride.
- Reacts with water to form hydrogen chloride. Patients exposed only to hydrogen chloride gas (boiling point -85°C, -121°F, respectively) do not pose a significant risk of secondary contamination. Patients whose clothing or skin is contaminated with phosphorus trichloride can cause secondary contamination of rescue and medical personnel by direct contact or through off-gassing hydrogen chloride.
- Liquid phosphorus trichloride causes skin burns/redness and pain. May lead to formation of blisters.
- Hydrogen chloride is rapidly corrosive when it comes in contact with moist tissue such as the eyes, skin, and upper respiratory tract causing eye irritation, coughing, chest pain and dyspnea. Swelling of the throat and accumulation of fluid in the lungs (shortness of breath, cyanosis, expectoration, coughing) may occur.
- There is no antidote to be administered to counteract the effects of phosphorus trichloride/hydrogen chloride. Treatment consists of supportive measures.

1. Substance information	 Phosphorus trichloride (PCl₃), CAS 7719-12-2 Synonyms: phosphorus trichloride, phosphoric chloride, PCL3 At room temperature phosphorus trichloride is a colorless to slightly yellow liquid with a sharp or pungent odor. On exposure to air dense white vapor is formed, due to condensation with atmospheric moisture. In contact with moisture, it forms hydrogen chloride. The vapor formed is corrosive. Phosphorus trichloride is available as an aqueous solution or gas (hydrogen chloride). Phosphorus trichloride is used as a precursor for production of pesticides, gasoline additives, plasticizers, dyes, and textile finishing agents.
2. Routes of exposure	
Inhalation	Most exposures occur by inhalation . Phosphorus trichloride's odor and upper respiratory irritant properties generally provide adequate warning of hazardous concentrations. Phosphorus trichloride is heavier than air and may cause asphyxiation in poorly ventilated, low-lying, or enclosed spaces. Reacts with water/moisture to form hydrogen chloride.
Skin/eye contact	Direct contact with phosphorus trichloride/hydrogen chloride on wet or moist skin causes severe chemical burns. It is poorly absorbed through the skin.
Ingestion	Ingestion is uncommon in occupational settings but may be aspirated. Aqueous solutions cause severe corrosive injury if ingested.
3. Acute health effects	
Respiratory	Hydrogen chloride exposure usually causes sore throat and coughing . Rapid development of respiratory distress with chest pain, dyspnea, laryngospasm and pulmonary edema (shortness of breath, cyanosis, expectoration, coughing) may occur. Pulmonary injury may progress over several hours. Hydrogen chloride poisoning may cause respiratory failure.
Dermal	Deep burns of the skin and mucous membranes may be caused by contact with concentrated phosphorus trichloride/hydrogen chloride; disfiguring scars may result. Contact with low concentrations cause burning pain, redness, inflammation, and blisters.
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Ocular	Low concentrations cause burning discomfort, spasmodic blinking or
Ocular	involuntary closing of the eyelids, redness, and tearing. Corneal burns, cataracts and glaucoma may occur from exposure to high concentrations.
Dose-effect relationships	Dose-effect relationships are as follows:
Phosphorus trichloride conc.0.2 ppm-0.5 ppm-	Effect TLV (ACGIH) STEL (ACGIH)
25 ppm -	IDLH
Hydrogen chloride concentration0.067-0.134 ppm5 ppm10 ppm10-50 ppm50 ppm50 ppm	Effect No change in respiratory pattern No organic damage Irritation; work undisturbed Work difficult; throat irritation after short exposure IDLH
50-100 ppm - 1,000-2,000 ppm -	Intolerable; work impossible Brief exposures dangerous; laryngospasms; lethal after a few minutes
Potential sequelae	If the patient survives the initial 48 hours after exposure, recovery is likely. After acute exposure, pulmonary function usually returns to normal in 7 to 14 days. Complete recovery is usual; however, symptoms and pulmonary deficits may persist. Airways hyperreactivity to non-specific irritants may persist, resulting in bronchospasm and chronic inflammation of the bronchi. Sequelae of the pulmonary tissue destruction and scarring may result in chronic dilation of the bronchi and increased susceptibility to infection. Chronic or prolonged exposure to hydrogen chloride gas or vapor has been associated with abnormal pulmonary function, chronic bronchial inflammation, nasal ulceration, skin and eye inflammation, and corrosion of the teeth.
4. Actions	
Recuer self-protection	In response situations that involve exposure to potentially unsafe levels of hydrogen chloride (see below), pressure-demand. Self-contained breathing apparatus and chemical protective clothing is recommended. Patients exposed only to hydrogen chloride gas do not pose a significant risk of secondary contamination. Patients whose clothing or skin is contaminated with liquid phosphorus trichloride can secondarily contaminate other people by direct contact or through off-gassing hydrogen chloride.
Decontamination	Patients exposed only to hydrogen chloride gas who have no evidence of skin or eye irritation do not need decontamination. All others require decontamination. Patients who are able and cooperative may assist with their own decontamination. If the exposure involved liquid phosphorus trichloride and if clothing is contaminated, remove and double-bag the clothing. Assure that exposed or irritated eyes have been irrigated with plain water or saline for at least 20 minutes, and that the pH of the conjunctival fluid has returned to normal (7.0). If not, continue eye irrigation during other basic care and transport. If eye irrigation is impaired by blepharospasm, one to two drops of oxybuprocaine 0.4% may be instilled into affected eyes to allow adequate irrigation. Remove contact lenses if present and easily removable without additional trauma to the eye.
Initial treatment	Assure that exposed skin and hair have been flushed with plain water for at least 15 minutes. If not, continue flushing during other basic care and transport. Protect eyes during flushing of skin and hair. Therapy will be empiric; there is not a specific antidote to be administered to counteract the effects of hydrogen chloride. The following measures are recommended if the exposure dose is

10 ppm of hydrogen chloride or greater (depending on time exposed), if symptoms, e. g. eye irritation or pulmonary symptoms have developed, or if no exposure dose can be estimated but exposure has possibly occurred:

- Administration of oxygen
- Administration of 8 puffs of beclomethasone (800 μg beclomethasone dipropionate) from a metered dose inhaler.

Patients with severe clinical respiratory symptoms (e.g. bronchospasms, stridor) should be treated as follows:

a) Nebulization of adrenaline (epinephrine): 2 mg adrenaline (2 ml) with 3 ml NaCl 0.9% and inhale through a nebulizer mask.

b) Administration of a ß2-selective adrenoceptor agonist, e.g., four strokes of terbutaline or salbutamol or fenoterol (one stroke usually contains 0.25 mg of terbutaline sulfate; or 0.1 mg of salbutamol; or 0.2 mg of fenoterol); this may be repeated once after 10 minutes. Alternatively, 2.5 mg salbutamol and 0.5 mg atrovent may be administered by nebulizer mask.

If inhalation is not possible, administration of terbutaline sulfate (0.25 mg to 0.5 mg) subcutaneously or salbutamol (0.2 mg to 0.4 mg over 15 minutes) intravenously.

c) Intravenous administration of 250 mg methylprednisolone (or equivalent steroid dose).

Patients with clinical signs of a toxic lung edema (e.g. foamy sputum, wet crackles) should be treated as follows:

- a) Start CPAP-therapy (Continuous Positive Airway Pressure Ventilation).
- b) Intravenous administration of 1000 mg methylprednisolone (or an equivalent steroid dose) is recommended.

Intubation of the trachea or an alternative airway management should be considered in cases of respiratory compromise. When the patient's condition precludes this, consider cricothyrotomy if equipped and trained to do so.

Note: Efficacy of corticosteroid administration has not yet been proven in controlled clinical studies

If hydrogen chloride was in contact with the skin, chemical burns may result; treat as thermal burns: adequate fluid resuscitation and administration of analgesics, maintenance of the body temperature, covering of the burn with a sterile pad or clean sheet.

After eye exposure chemical burns may result; treat as thermal burns. Immediately consult an ophthalmologist.

To the standard intake history, physical examination, and vital signs add pulse oximetry monitoring and a PA chest X-ray. Spirometry should be performed. Routine laboratory studies should include a complete blood count, blood glucose and electrolyte determinations.

Evidence of pulmonary edema - hilar enlargement and ill-defined, central-patch infiltrates on chest radiography - is a late finding that may occur 6 to 8 hours or later after exposure. The chest X-ray is typically normal on first presentation to the emergency department even with severe exposures.

Patients who have possible exposure or who develop serious signs or symptoms should be observed for a minimum of 24 hours and

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Further evaluation and treatment

reexamined frequently before confirming the absence of toxic effects. Delayed effects are unlikely in patients who have minor upper respiratory symptoms (mild burning or a slight cough) that resolve quickly.

If oxygen saturation is less than 90 % or if it appears to drop, immediately check arterial blood gasses and repeat the chest X-ray. If blood gasses begin to show deterioration and/or if the chest X-ray begins to show pulmonary edema start oxygen supplementation. In case of worsening clinical signs (especially tachypnea >30/min with a simultaneous decrease of the partial pressure of carbon dioxide) CPAPtherapy (Continuous Positive Airway Pressure Ventilation) should be started within the first 24 hours after exposure.

In case of a pulmonary edema fluid intake/output and electrolytes should be monitored closely. Avoid net positive fluid balance. Central line or Swan-Ganz catheterization might be considered, to optimize fluid management.

As long as signs of pulmonary edema are present, intravenous administration of methylprednisolone (or an equivalent steroid) should be continued in intervals of 8-12 hours.

Prophylactic antibiotics are not routinely recommended but may be used based on the results of sputum cultures. Pneumonia can complicate severe pulmonary edema.

If required, provide adequate pain treatment, e.g. 10 mg morphine sulfate intravenously.

Clinically asymptomatic patients exposed to a concentration of **less than 10 ppm hydrogen chloride** (depending on the period of time exposed) as well as patients who have a **normal clinical examination** and **no signs or symptoms of toxicity** may be discharged after an appropriate observation period in the following circumstances:

- a) The evaluating physician is experienced in the evaluation of individuals with hydrogen chloride exposure.
- b) Information and recommendations for patients with follow-up instructions are provided verbally and in writing. Patients are advised to seek medical care promptly if symptoms develop or recur.
- c) The physician is comfortable that the patient understands the health effects of hydrogen chloride.
- d) Site medical is notified, so that the patient may be contacted at regular intervals in the 24-hour period following release from the emergency department.
- e) Heavy physical work should be precluded for up to 24 hours.
- f) Exposure to cigarette smoke should be avoided for 72 hours; the smoke may worsen the condition of the lungs.

Patients who have serious skin or eye injuries should be reexamined in 24 hours.

Post discharge spirometry should be repeated until values return to the patient's baseline values.

Patient release/ follow-up instructions In this document BASF has made a diligent effort to ensure the accuracy and currency of the information presented but makes no claim that the document comprehensively addresses all possible situations related to this topic. This document is intended as an additional resource for doctors at hospitals/emergency departments in assessing the condition and managing the treatment of patients exposed to phosphorus trichloride. It is not, however, a substitute for the professional judgement of a doctor and must be interpreted in the light of specific information regarding the patient available to such a doctor and in conjunction with other sources of authority.

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