BASF Chemical Emergency Medical Guidelines

Chloroacetyl chloride ($ClCH_2COCl$) C 1

Information and recommendations for doctors at hospitals/emergency departments

- **Patients exposed only to chloroacetyl chloride gas do not pose a significant risk of secondary contamination. Patients whose clothing or skin is contaminated with liquid chloroacetyl chloride or solvents containing chloroacetyl chloride can secondarily contaminate rescue and medical personnel by direct contact or through off-gassing chloroacetyl chloride.**
- **Chloroacetyl chloride is a lacrimator and a severe pulmonary irritant. Because of its hydrolysis in the alveoli, serious pulmonary effects and, therefore, symptoms of toxicity may be delayed up to 24 hours. Signs of pulmonary edema (shortness of breath, cyanosis, expectoration, cough) may appear hours after toxic exposures.**
- **There is no antidote to be administered to counteract the effects of chloroacetyl chloride. Treatment consists of supportive measures.**

1. Substance information

Chloroacetyl chloride ($ClCH_2COCl$), CAS 79-04-9
Synonyms: chloroacetic chloride, CAC
Chloroacetyl chloride is a colorless, water-white liquid at room temperature with a melting point of –22 °C and a boiling point of 106°C. It has a sharp and pungent odor. Chloroacetyl chloride is hydrolyzed slowly by moisture to form chloroacetic acid and hydrochloric acid.

Chloroacetyl chlorides are used as an intermediate in the manufacture of many chemicals including adrenalin, diazepam, chloroacetophenone, chloroacetate esters and chloroacetic anhydride.

2. Routes of exposure

Inhalation

Most exposures occur by inhalation or by skin/eye contact. Chloroacetyl chloride´s odor may provide insufficient warning of hazardous exposure that can occur even at low concentrations. Chloroacetyl chloride is heavier than air and may travel along the ground.

Skin/eye contact

Chloroacetyl chloride can cause irritation and burns of the skin and the eyes. Dermal absorption may occur.

Ingestion

Accidental ingestion of chloroacetyl chloride may occur and may cause irritation of the mouth, throat and stomach.

3. Acute health effects

Chloroacetyl chloride exposure usually causes skin, eye, nose, throat, and pulmonary irritation. **Irritating effects immediately after exposure might be severe and delayed pulmonary damage, primarily edema, may occur as late as 24 hours after exposure.** Chloroacetyl chloride poisoning may cause respiratory and cardiovascular failure.
If the skin is wet or moist, contact with chloroacetyl chloride gas can cause irritation and redness of the skin.
High gas concentrations may cause tearing and conjunctival erythema of the eye. Eye contact with liquid chloroacetyl chloride may result in clouding of the eye surface and delayed perforation. High gas concentrations may cause tearing and conjunctival erythema of the eye. Eye contact with liquid chloroacetyl chloride may result in clouding of the eye surface and delayed perforation.

Dose-effect relationships Dose-effect relationships are as follows:

Chloroacetyl chloride		Effect
0.023 ppm	-	odor barely detectable
0.05 ppm	-	8 hr time weighted average (TWA) (ACGIH)
0.15 ppm	-	15 min short term exposure limit (STEL) (ACGIH)
0.05 ppm		ERPG-1 (AIHA)
0.5 ppm	-	ERPG-2 (AIHA)
10 ppm	-	ERPG-3 (AIHA)

The **TWA** is the concentration for a conventional 8-hour workday and a 40-hour workweek to which it is believed that nearly all workers may repeatedly be exposed to , day after day, without adverse effects.
ERPG-1 is the maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing other than mild transient health effects or perceiving a clearly defined, objectionable odor. **ERPG-2** is the maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing or developing irreversible or other serious health effects or symptoms which could impair an individual's ability to take protective action. **ERPG-3** is the maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing or developing life-threatening health effects.

Potential sequelae If the patient survives the initial 48 hours after exposure, recovery is likely. Sensitivity to irritants may persist, causing bronchospasm and chronic inflammation of the bronchi. Pulmonary tissue destruction and scarring may lead to chronic dilation of the bronchi and increased susceptibility to infection.

4. Actions

Decontamination Patients exposed only to chloroacetyl chloride gas do not pose a significant risk of secondary contamination. Patients whose clothing or skin is contaminated with liquid chloroacetyl chloride or solvents containing chloroacetyl chloride can secondarily contaminate other people by direct contact or through off-gassing chloroacetyl chloride. Patients who are able and cooperative may assist with their own decontamination. If the exposure involves liquid chloroacetyl chloride or solvents containing chloroacetyl chloride and if clothing is contaminated, remove and double-bag the clothing.
Assure that exposed skin and hair have been flushed with plain water for at least 20 minutes. If not, continue flushing during other basic care. Protect eyes during flushing of skin and hair.
Assure that exposed or irritated eyes have been irrigated with plain water or saline for at least 20 minutes. If not, continue eye irrigation during other basic care.
Remove contact lenses if present and easily removable without additional trauma to the eye.

Initial treatment Therapy will be empiric; there is no antidote to be administered to counteract the effects of chloroacetyl chloride.
The following measures are recommended if the exposure dose is ERPG-2 or greater, if 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 chloroacetyl chloride were in contact with the skin or eyes 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 immediately consult an ophthalmologist.
Note: Any facial exposure to liquid chloroacetyl chloride should be considered as a serious exposure.

Further evaluation and treatment

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 should be observed for a minimum of 24 hours and reexamined frequently before confirming the absence of toxic effects.
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) CPAP-therapy (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.

Patient release/ follow-up instructions

Clinically asymptomatic patients exposed to a concentration of **less than ERPG-II** (depending on the period of time exposed) **as well as patients who 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 chloroacetyl chloride exposure.
b) Information and recommendations for patients with follow-up instructions are provided verbally and in writing.
c) The physician is comfortable that the patient understands the health effects of chloroacetyl 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 24 hours.
f) Exposure to cigarette smoke should be avoided for 72 hours; the smoke may worsen the condition of the lungs.

Patients with an exposure of **ERPG-2 or above** who have a **normal examination and no signs or symptoms of toxicity after observation for 24 hours** may be discharged from the emergency department in the following circumstances:

a) The evaluating physician is experienced in the evaluation of individuals with chloroacetyl chloride exposure.
b) Even if there has not been clinical deterioration, the patient's chest X-ray should be repeated prior to release. The patient should not be released if any degree of pulmonary edema is demonstrated.
c) Information and recommendations for patients with follow-up instructions are provided verbally and in writing.
d) The physician is comfortable that the patient understands the health effects of chloroacetyl chloride and the provided follow-up instructions.
e) 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.
f) Heavy physical work should be precluded for 24 hours.
g) Exposure to cigarette smoke should be avoided for 72 hours; the smoke may worsen the condition of the lungs.

Patients who have eye injuries should be reexamined in 24 hours.
Post-discharge spirometry should be repeated until values return to the patient's baseline values.

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 in assessing the condition and managing the treatment of patients exposed to chloroacetyl chloride. 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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