
Information and recommendations for
doctors at hospitals/emergency departments

- Patients whose clothing or skin is contaminated with nitric acid can cause secondary contamination of rescue and medical personnel by direct contact or by release of nitric acid vapor or fumes.
 - Nitric acid and its vapor or fumes are rapidly corrosive when they come in contact with tissues such as the eyes, skin, and upper respiratory tract causing irritation, burns, coughing, chest pain and dyspnea. Laryngospasm and pulmonary edema (shortness of breath, cyanosis, expectoration, cough) may occur.
 - Ingestion of nitric acid can cause severe corrosive injury to the lips, mouth, throat, esophagus, and stomach.
 - There is no antidote to be administered to counteract the effects of nitric acid. Treatment consists of supportive measures.
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1. Substance information

Nitric acid (HNO₃), CAS 7697-37-2

Synonyms: hydrogen nitrate

At room temperature nitric acid is a colorless to yellow or brownish-red liquid with a choking odor. The color is due to the release of oxides of nitrogen, especially nitrogen dioxide, into the air upon exposure to light. Depending on environmental factors the vapor or fumes of nitric acid may actually be a mixture of various oxides of nitrogen and nitric acid, even at temperatures well below the boiling point of 83°C (181°F). Nitric acid may be formed in photochemical smog from the reaction between nitric oxide and hydrocarbons.

Nitric acid itself is nonflammable, but it can increase the flammability or cause the spontaneous combustion of other materials. It is soluble in water.

Nitric acid is used in the manufacture of fertilizers, gunpowder and explosives, pesticides, dyestuffs, and pharmaceuticals, especially in the manufacture of organic and inorganic nitrates. It is also used for etching and cleaning of metals, and electroplating.

2. Routes of exposure*Inhalation*

Nitric acid's odor and irritant properties generally provide adequate warning of acutely hazardous concentrations.

Skin/eye contact

Direct contact with liquid nitric acid or concentrated vapor or fumes on wet or moist skin causes severe chemical burns. Nitric acid is poorly absorbed through the skin.

Ingestion

Ingestion of nitric acid can cause severe corrosive injury to the lips, mouth, throat, esophagus, and stomach.

3. Acute health effects*Respiratory*

Nitric acid exposure usually causes dryness of the nose and throat, and coughing. Inhalation of very high concentrations may result in laryngospasm and eventually in obstruction of the airways and death. Development of respiratory distress with chest pain, dyspnea and pulmonary edema (shortness of breath, cyanosis, expectoration) may occur after a delay of up to 24 hours.

Hematologic

Only after high-dose exposure methemoglobinemia may result, but usually to an extent that does not require treatment. For further information on methemoglobinemia see BASF Chemical Emergency Medical Guideline for ANILINE.

<i>Gastrointestinal</i>	Epigastric pain, nausea, and vomiting may occur. In cases of ingestion diffuse corrosive mucosal injury can involve the entire intestinal tract.
<i>Renal</i>	Acid-base imbalance and acute renal failure may occur.
<i>Dermal</i>	Deep burns of the skin and mucous membranes may be caused by contact with concentrated nitric acid; sometimes yellowing of the skin results. Contact with less concentrated nitric acid vapor or fumes can cause burning pain, redness, and inflammation.
<i>Ocular</i>	Severe eye burns with clouding of the surface, perforation of the globe, and ensuing blindness may occur from exposure to liquid nitric acid. Low concentrations of vapor or fumes cause burning discomfort, spasmodic blinking or involuntary closing of the eyelids, redness, and tearing.
<i>Potential sequelae</i>	Skin, eye, and mucous membrane damage caused by chemical burns may be irreversible, e. g. gangrene, blindness, or narrowing of the esophagus. After inhalation, complete recovery is usual; however, symptoms and pulmonary deficits may persist. Permanent restrictive and obstructive lung disease may occur from bronchiolar damage. Pulmonary tissue destruction and scarring may result in chronic dilation of the bronchi and increased susceptibility to infection. Yellow discoloration or erosion of teeth can occur from prolonged exposure.

4. Actions

<i>Self-protection</i>	Patients whose clothing or skin is contaminated with nitric acid can secondarily contaminate other people by direct contact or through nitric acid vapor or fumes.
<i>Decontamination</i>	All patients exposed to nitric acid require decontamination. Patients who are able and cooperative may assist with their own decontamination. If the exposure involved liquid nitric acid 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. 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.
<i>Initial treatment</i>	Therapy will be empiric; there is no specific antidote to be administered to counteract the effects of nitric acid. The following measures are recommended if the airborne exposure concentration is 10 ppm or greater, if symptoms, e. g. eye irritation or pulmonary symptoms have developed, or if no exposure concentration 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 β_2-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 nitric acid 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.

Note: Any facial exposure to liquid nitric acid should be considered as a serious exposure.

In case of ingestion of nitric acid, **do not induce emesis. If signs or symptoms of esophageal irritation or burns are present, consider endoscopy to determine the extent of the injury; in severe cases surgical intervention should be considered if gastrointestinal necrosis or perforation is suspected.**

Only if a large dose has been ingested less than 30 minutes before evaluation of the patient's condition and if a perforation can be excluded, consider immediate gastric lavage with a small-bore tube.

*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. Arterial blood gases and methemoglobin concentrations should be used to assess for the presence of acidosis and methemoglobinemia in symptomatic patients.

Evidence of pulmonary edema - hilar enlargement and ill-defined, central-patch infiltrates on chest radiography - **is a late finding that may occur as late as 24 hours 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 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 gases 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 10 ppm (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 nitric acid or irritant pulmonary exposures.
- 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 nitric acid.
- d) Site physician is informed, 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.

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 nitric acid. 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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