Pulmonary Acute Lesions After Caustic Exposure

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ABSTRACT

Although lesions related to chemical burns concern digestive tract first, pulmonary damage can also be observed, in cases of inhalation or secondary to gastric aspiration. Pulmonary lesions after caustic exposure are non-specific. Multiple factors may influence the pattern of lesions, including the nature of the caustic substance, the duration of contact, the amount of the substance encountering the tissue and the length of post-ingestion survival. Significant complications of caustic ingestion such as chemical pneumonitis can develop in the first days or later. This article summarizes the most frequent pulmonary lesions according to the corrosive agent (gas, solid and liquid caustic, arsenic).

Introduction

Caustic ingestion and inhalation continues to be a significant problem world-wide especially in developing countries and particularly in children under 6 years. Ingestion and skin contact are the most commonly encountered means of exposure in chemical burn cases1. Accidental ingestion occurs most often in infants and toddlers, but it is less common in adults in whom chemical ingestion is often deemed to be suicidal behaviour2. Digestive corrosive burns, generally reaching the upper digestive tract, can be categorized by depth, from superficial erosion to full-thickness necrosis3,4,5. Acids cause severe lesions of the stomach, mainly in the pyloric region, leading to strictures, but generally spare the esophagus6,7. Microscopic lesions after acid exposure show that mucosa is first concerned, with extensive local venous thrombosis, interstitial edema, haemorrhage and inflammatory infiltrate, possible perforation when the whole wall is affected by coagulative necrosis8. Alkalis deeply penetrate into tissues, typically, esophagus is the prime target of alkali injury2. If great quantities of caustic substance are drunk, the duodenum may be involved9. Microscopic lesions may have a similar aspect as those found after acid ingestion. Complications of caustic exposure can involve chemical pneumonia, secondary to inhalation or to aspiration.

The presented short review aims at examining main pulmonary lesions after caustic exposure in both lethal and non-lethal cases.

Review

The aggressiveness of a caustic agent depends on several factors10-13, especially the nature and concentration of the product, duration of contact, amount of the substance that touches the
skin, respiratory tract or gastrointestinal tract, length of exposure, regional epidermal or mucosal properties, penetrability of the chemical, physicochemical properties and speed of action.

Lesions related to chemical burns have been studied through case reports, clinical analyses and autopsy series. They generally concern digestive tract and more rarely lungs.

Severe caustic ingestion can cause critical tracheobronchial burns characterized by necrotic lesions. Those lesions constitute a severe complication, leading in some cases to death. In case of an ulcerating or pre-splitting aspect appearing in localised necrotic lesions, the treatment can consist in a tracheobronchial plasty in emergency.

Contrary to digestive damage, there is no specific pulmonary lesion between acid and bases described in literature.

Gases and toxic industrial or household fumes attack the lung. At sufficient dosage these aggressive agents have first of all a "suffocating" action as well as a caustic and corrosive action. Pulmonary oedema is the main lesion, due to a direct action on the bronchial epithelium and an indirect action by disturbance of surfactant metabolism. It is mainly an interstitial oedema, which can be secondarily endo-alveolar. Differential diagnosis is not always easy to achieve. The possibility of burns of the respiratory pathways (blast) must be considered. These elements can moreover be associated with lesions due to toxic inhalation. In case of survival, fibrosis can later develop.

After heavy ammonia (a weak base) poisoning, an irritating and caustic gas, the intensity of the lesions and the mortal risk are proportional to the quantity of gas per m³ of air. The most recent lesions, which appear during the first three days, consist of a sharp lung oedema sometimes associated with a pulmonary emphysema. Later on, the main lesion is a pneumopathy (pulmonary infection, bronchiectasis, pulmonary fibrosis).

Acute inhalation toxicity of lithium combustion aerosols was studied in rats. Respiratory difficulty, perioral and perinasal encrustation were observed. The most prominent lesions are necrotizing laryngitis and ulcerative rhinitis. Pulmonary lesions represented a secondary extension of the upper respiratory tract lesions rather than a primary manifestation of lithium toxicity. These lesions are often accompanied by areas of squamous metaplasia, and, in some cases, a suppurative bronchopneumonia or aspiration pneumonia, probably secondary to the laryngeal lesions.

In 2006, Mattos and al. observed the effects of solid corrosive soda after ingestion. If esophagus was the first internal organ concerned, damage to the pulmonary parenchyma and trachea due to this base occurred at 33.66% concentration after 10 minutes, whereas 1.83% concentration was sufficient for esophageal epithelial necrosis. According to this study, higher amount of caustic is necessary to damage pulmonary tract, as compared with the digestive tract.

Hydrogen peroxide, an acid molecule, which is found in numerous products, in particular household products, causes toxicity via three main mechanisms: corrosive damage, oxygen gas formation and lipid peroxidation. Ingestion of hydrogen peroxide may cause irritation of the gastrointestinal tract with nausea, vomiting, haematemesis and foaming at the mouth; the foam may obstruct the respiratory tract or result in pulmonary aspiration. Although most inhalational exposures cause little more than coughing and transient dyspnea, inhalation of highly concentrated solutions of hydrogen peroxide can cause severe irritation and inflammation of mucous membranes, with coughing and dyspnea. Shock, coma and convulsions may ensue and pulmonary oedema may occur up to 24-72 hours post exposure.

Paraquat (trivial name, corresponding to N,N′-dimethyl-4,4′-bipyridinium dichloride (systematic name), is an organic compound with the chemical formula [(C₆H₇N)₂]Cl₂) used as herbicide. Heavy exposure to paraquat usually results in death, either due to gastrointestinal caustic lesions, shock, and acute respiratory distress syndrome or related to the progressive development of pulmonary fibrosis associated with refractory hypoxemia. If early administration of an antioxidant therapy, including deferoxamine and acetylcysteine may be useful, associated with measures that prevent digestive absorption or enhance elimination to limit systemic toxicity in potentially fatal paraquat poisoning, respiratory damage can be irreversible. The main prognostic factors appear to be the route of administration. Inhalation of paraquat aerosols and/or contamination of skin with the herbicide seems to have better outcome than ingestion, in particular according to the amount of poison. Above 50 mg/kg, patients die of circulatory failure within 72 h; between 35 and 50 mg/kg, a progressive pulmonary fibrosis occur.

Exposure to arsenic, notably through drinking water from arsenic-affected areas of Bangladesh and India, has been described in Asian population. The mechanism remains unclear; however, earlier human and animal studies indicate deposition and permanent structural changes in the lung epithelium as a result of arsenic exposure. Animal studies showed tissue inflammation and increased morphologic changes in the lung with increasing arsenic exposure.

Main characteristics of these corrosive agents are summarized in table 1.
In our previous study\(^{28}\), we showed that many organic and nonorganic agents can cause chemical pneumonitis. The presented microscopic findings, although generally unspecific\(^{29,30,31}\), often showed intra-alveolar edema, activated macrophages, congestion and pulmonary inflammation. Hyaline membranes and capillary haemorrhages can also be found in this regard, as observed after lye ingestion. These are not specific to the type of agent and can be caused by a large variety of potential toxic insults\(^{32}\), such as infections, high concentrations of oxygen, toxic inhalants, drug toxicity, radiation and many other ingestants including kerosene, denatured rapeseed oil and pararquet. Moreover, the tracheo-bronchial tree was the site of mild burns, with lesions similar to those seen on the skin. We suppose that respiratory burns can result either from gastric acidity or from the direct chemical effects of the caustic agent. The severity of chemical-induced respiratory damage depends on the nature and amount of the inhaled agent\(^{33}\).

We also pointed out that pulmonary lesions may have developed during the aspiration of the agent from the stomach. Alternatively, patients may not have ingested enough agent and/or the agent, particularly alkalis, may not have been aggressive enough to cause respiratory lesions.

**Conclusions**

Pulmonary damage after caustic exposure are rare, except in cases of gas or vapor exposition or as a result of gastric aspiration. They frequently consist in epithelial necrosis, intra-alveolar edema, activated macrophages, congestion and pulmonary inflammation. With time, fibrosis can be a serious complication, leading to chronic respiratory disease.

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**Conflict of Interest**

The authors declare that they have no conflict of interest.

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**Table 1 : Respiratory tract and lung lesions according to the nature of the agent.**

<table>
<thead>
<tr>
<th>Agent</th>
<th>Gases</th>
<th>Lithium combustion</th>
<th>Paraquat</th>
<th>Arsenic</th>
<th>Hydrogen peroxide (acid)</th>
<th>Ammonia (base)</th>
<th>Solid caustic soda (base)</th>
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<tr>
<td>Main lesions</td>
<td>Interstitial and endo-alveolar edema +/- epithelial burns</td>
<td>Necrotising laryngitis, ulcerative rhinitis, secondary pulmonary lesions (squamous metaplasia, suppurative bronchopneumonia, aspiration pneumonia)</td>
<td>Pulmonary irritation. Later : pulmonary fibrosis</td>
<td>Inflammation, increased morphologic changes in lung tissue</td>
<td>Severe irritation and inflammation of mucous membranes, pulmonary edema</td>
<td>First days : pulmonary oedema +/- pulmonary emphysema</td>
<td>Epithelial necrosis of the trachea and the lung at high concentration.</td>
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**References**


