perature followed by severe respiratory failure that required mechanical ventilation within 3 weeks of onset. The diffuse infiltration of NK-LGL into the interstitial space was most probably the cause of the respiratory failure. Although infiltrations to other organs, including liver and spleen, are common findings, this report is the first (to our knowledge) to demonstrate the infiltration of LGL into the lungs with the manifestation of nodular shadows on a chest radiograph and severe dyspnea.

REFERENCES

Persistent Asthma After Inhalation of a Mixture of Sodium Hypochlorite and Hydrochloric Acid*

Dominique Deschamps, M.D.; Paul Soler, Ph.D.; Nicole Rosenberg, M.D.; Frédéric Baud, M.D.; and Pierre Gervais, M.D.

Chlorine gas inhalation can lead to temporary mucous membrane irritation, pulmonary edema, and transient bronchospasm. Existence of respiratory sequelae is debated. We report a case of asthma, persisting 2 years after the inhalation of a mixture of sodium hypochlorite and hydrochloric acid. Bronchial histologic findings and transmission electron microscopy examinations showed uncommon abnormalities supporting irritation for cause of this noninmunologic asthma.

(CHEST 1994; 105:1895-96)

Chlorine gas inhalation can cause temporary mucous membrane irritation, pulmonary edema, and tran-

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The first attack of asthma happened 44 days after the accident and then twice a month, whenever she attempted mild physical exercise. With continuous treatment of inhaled steroids, attacks were less frequent, the mild bronchial obstruction remained unchanged, but bronchial hyperreactivity worsened to 140 µg of methacholine. Two years after the accident, the patient still suffers from attacks.

Chest x-ray film and CT scan results were normal. Bronchial histologic examination was performed before treatment was started with inhaled steroids. Histologic studies showed epithelial destruction, partial or total according to location but only a slight inflammatory response with sparse lymphocytes and no eosinophils. Examination by transmission electron microscopy showed signs of injury of the epithelial cells (decreased density of cytoplasm, vacuolization of the organelles) and thickening of the subepithelial connective tissue, with tangle of collagen fibers. There was no thickening of the basal lamina and no alteration of tight junctions or of cilia (Fig 1). In some places, the epithelial destruction was nearly complete, leaving occasional basal cells still attached to the basal lamina (Fig 2).

**DISCUSSION**

As far as we know, this is the first case published of asthma that persisted years after inhalation of a mixture of sodium hypochlorite and hydrochloric acid, a common domestic accident.

Subepithelial fibrosis commonly is observed in asthma. However, the lack of eosinophils is unusual and surprising, since the patient was atopic. The onset of asthma after the accident and the histologic abnormalities support irritation and caustic injuries (and not inflammation) as a primary hypothesis for this reactive airway dysfunction syndrome, persistent asthma after inhalation of an irritant product. The epithelial destruction could impede release of neutrophils with bronchodilator effects, leading to persistent asthma. The product responsible is probably chloride formed when mixing sodium hypochlorite and hydrochloric acid.

Asthma due to individuals to exposure chemicals usually occurs through immunologic mechanisms in individuals exposed to low concentrations for a long period. But short accidental exposure to high concentrations of irritant gas also may lead to asthma. Our report indicates that mixing sodium hypochlorite and hydrochloric acid is another cause of this reactive airway dysfunction syndrome. Bronchial histologic studies may be useful for the diagnosis when the patient is atopic.

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**Surgical Repair of Esophageal Perforation in Cirrhotic Patients With Varices**

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A 51-year-old woman, a known alcohol abuser, had sclerotherapy for esophageal varices from portal hypertension. A perforation of the distal esophagus, diagnosed several days later, could not be closed primarily at thoracotomy due to extensive bleeding. The method of "exclusion and diversion in continuity" was modified by ligating the esophagogastric junction with absorbable suture over a tube stent. The perforation healed and patency of the esophageal lumen was demonstrated 2 weeks later. This alternative life-saving procedure may be useful in chronic esophageal perforation, especially in cirrhotic or otherwise debilitated patients.

**Controversy surrounds the treatment of esophageal perforations. This condition is best managed surgically, with some exceptions dictated by the size and location of the perforation, the time elapsed, and the patient's condition. Procedures include drainage, with primary repair if possible, and buttressing the repair. Celestin tubes, stents, or T-tubes are advocated without wide acceptance. Resection with gastroesophageal replacement in patients with underlying esophageal disease has also been proposed.

In 1974, Urschel and coworkers introduced "exclusion and diversion in continuity," which involved ligation of the gastroesophageal junction to prevent reflux and a cervical esophagostomy to control oral secretions. The perforation was closed primarily, the mediastinum was drained, and a gastrostomy was constructed. This requires a second operation to reconstruct the distal esophagus later. Popovsky modified this procedure by ligating the distal esophagus with a nonabsorbable suture and thus avoided the need for a second operation. We present a further modification of this procedure.

**CASE REPORT**

A 51-year-old woman with a history of alcohol abuse was admitted to the hospital emergently with crampy upper abdominal pain, coffee ground emesis, loose tarry stools, generalized weakness, and recent weight loss. Physical examination revealed epigastric tenderness without rebound, normal bowel sounds, hepatomegaly, and spider angiomas. On panendoscopy, varices in the distal esophagus were identified as the source of bleeding and were sclerosed. Two days later, panendoscopy and sclerotherapy were repeated. Two days after the last sclerotherapy treatment, the patient complained of acute right-sided chest pain and shortness of breath. Her temperature was 38.3°C and her WBC count was 13,400/mm³. A chest radiograph revealed bilateral varices.*

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**Surgical Repair of Esophageal Perforation in Cirrhotic Patients (Clouse et al)**