

Metal Fume Fever

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A 35-year-old welder developed metal fume fever after working two hours longer than usual and with a new metal. Metal fume fever is an acute illness of short duration produced by many metals. Symptoms are of sudden onset, four to six hours, and include rigors, high fever, profuse sweating, weakness and muscle aches. Some metals cause pulmonary involvement as in this case. Therapy involves steroids to reduce respiratory tract injury, oxygen to correct hypoxemia, and aminophylline to treat bronchospasm. Dula DJ: Metal fume fever. *JACEP* 7:448-450, December, 1978.

fever, metal fume; poisoning, inhalation

INTRODUCTION

Exposure and inhalation of metal fumes is an occupational risk that produces respiratory tract as well as systemic signs and symptoms. We report here the case of a welder who became ill from metal fumes inhalation, known as metal fume fever.

CASE REPORT

This patient, a 35-year-old welder, developed chest tightness and chills three hours after finishing welding. Fifteen minutes later, he developed a non-productive cough, became dyspneic, and felt acutely ill. He had welded two hours longer than usual and was working with a new metal, but he could not recall its composition. The patient denied being symptomatic after welding before and any prior lung disease. He was a nonsmoker.

On physical examination, the patient appeared acutely ill, seemed dyspneic and was coughing and diaphoretic. Vital signs were pulse, 100/minute; respiratory rate, 28/minute; blood pressure, 140/90 mm Hg; temperature orally, 39 C (102.2 F). Chest examination revealed scattered wheezes but was otherwise clear. He had a macular erythematous rash on his face and neck, and his face looked flushed. The remainder of the examination was within normal limits. The electrocardiogram (ECG) revealed a sinus tachycardia. A chest x-ray film showed a haziness of the left upper lobe and a left lower lobe infiltrate (Figure 1). Hemoglobin was 15.4 gm; white blood cell count (WBC), 14,800/cu mm with 77% neutrophils, 15% bands and 7% monocytes. Sodium was 139; chloride, 77; carbon dioxide, 26; potassium, 3.8; blood urea nitrogen (BUN), 23 and blood sugar 135. Arterial blood gases (ABG) on room air were pH 7.45; pCO₂, 34; pO₂, 68; HCO₃, 24 and ABGs on 100% O₂ were pH, 7.43; pO₂, 346; pCO₂, 36; HCO₃, 24, with a calculated shunt of 15%.

He was admitted to the intensive care unit where ABGs were monitored and nasal oxygen was given. Within 24 hours his fever had lysed, his respiratory symptoms were gone and repeat chest x-ray film was normal.

DISCUSSION

Metal fume fever is an acute illness of short duration that occurs when

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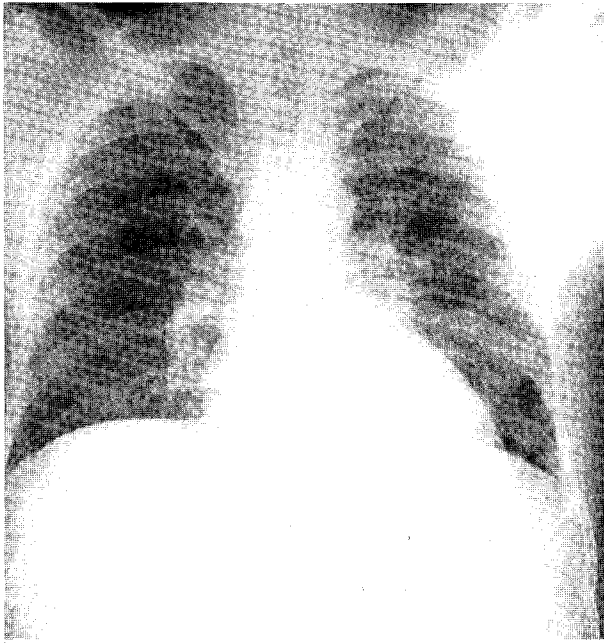


Fig. 1. Chest x-ray film on admission.

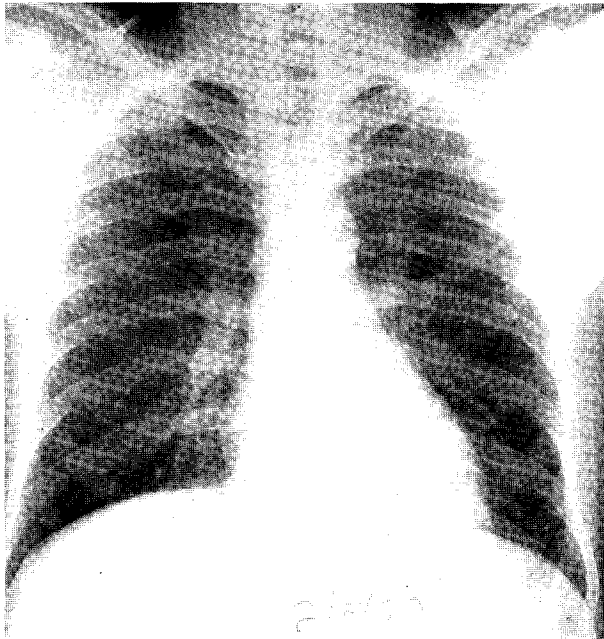


Fig. 2. Chest x-ray film 24 hours after admission.



fumes from metals, heated above their melting point, are inhaled. Many metals can cause the syndrome, but the most common are aluminum, antimony, cadmium, iron, magnesium, nickel, selenium, silver and tin.^{1,2} Exposure is most common in occupations involving the metal industry, brass and zinc foundries, galvanizing, chrome plating, welding and metal grinding² (Table 1). The condition was first recognized by Potissier in 1822¹ and through the years has been called Brazier's disease, Spelter shakes, Brass chills,

Zinc chills, Welder's ague, Copper fever, Monday fever, and Foundry fever.¹

Symptoms of the disease are of sudden onset, usually within four to eight hours of exposure, and begin with a sense of thirst and metallic taste in the mouth.³ Later, rigors, high fever, profuse sweating, weakness and muscle aches occur.⁴ Pulmonary symptoms and signs may be sparse but some metals, particularly cadmium, beryllium, manganese, antimony, cobalt, osmium and bauxite, cause severe chemical pneumonitis

in addition to fever and the other symptoms described.^{2,3} The condition seems to occur most frequently in those without previous exposure to the fumes or in workers returning to work on Monday after having the weekend off — hence the name, Monday fever.¹ When pulmonary involvement is severe, symptoms are the result of airway irritation and parenchymal injury producing cough and shortness of breath.³ Physical examination may reveal rales and wheezing.³ The chest x-ray film may be normal initially, but often a pat-

Table
AGENTS AND INDUSTRIES INVOLVED IN METAL FUME FEVER

Agents	Industry
Most Common: Zinc, Copper, Magnesium. Less Common: Cadmium, Iron, Manganese, Nickle, Selenium, Tin, Anti- mony, Mercury, Cobalt, Beryllium, Silver, Vanadium.	Zinc and brass foundries; electric furnaces to melt metals; galvanizing; chrome plating; weld- ing; metal grinding; electroplat- ing; manufacture steel alloys.

tern of hazy infiltrates or pulmonary edema develops within 12 to 24 hours after exposure.⁴ ABGs will show hypoxemia, depending on the degree of pulmonary injury, and there may be a leukocytosis, often as high as 18,000 cu mm.¹ The diagnosis is entirely based on the history of exposure to metal fumes as no specific test for the condition exists.^{3,4}

The pathology found in the respiratory tract injury is the result of the irritative effects of the toxic fumes on the bronchi and parenchyma of the lungs.³ Studies after inhalation of toxic gases such as cadmium and zinc show inflammatory changes in the bronchial mucosa and edema and inflammation of the alveolar walls.^{1,3}

Therapy

The course of the disease is usually benign with spontaneous resolution of symptoms in 24 to 48 hours. However, if exposure is prolonged or if the material is particularly noxious (as is cadmium), acute pneumonitis can result and the condition may be fatal.^{2,4,6}

Patients who present in a toxic state with dyspnea, pulmonary infiltrates and hypoxemia, need immediate care while in the emergency

department. Steroids, intravenously, in high doses are probably effective in reducing the amount of respiratory tract injury, especially after cadmium inhalation.^{2,4} Oxygen should be administered to correct hypoxemia, and bronchospasm can be treated with aminophylline intravenously and steroids.⁴ The use of antibiotics is of little value and should be reserved for possible secondary bacterial infection.³

Difficulties arise in managing patients with equivocal exposure to noxious fumes. Even though they may be only mildly symptomatic when first seen, acute pulmonary edema can develop with alarming rapidity.³ The type of fume exposure, patient symptoms, physical examination, chest x-ray films, and ABGs are helpful in determining those with a significant exposure. Performing shunt studies on patients (breathing 100% oxygen) exposed to toxic gases is a more sensitive test for assessing pulmonary injury than ABGs on room air.^{5 p61-62} We deliver 100% oxygen by having the patient breathe through a mouthpiece with a Collins double J valve (which allows inspired air to come only from the closed system) connected to a 5 liter anesthesia bag filled with oxygen, running 15 liters per minute. The patient breathes for 15 minutes, making

sure the lips are tight with no leaks, and keeping the nose clipped shut. ABGs are then drawn and the percent shunt is estimated by assuming that a normal pO₂, while breathing 100% oxygen, would be 650 mm Hg and for each 100 mm below this value, one can assume a 5% shunt. Normal physiologic shunt is usually about 5%.^{5 p61-62}

In the case presented in this paper, the pO₂ on room air was 68, while the shunt done on 100% oxygen revealed a pO₂ of 346 or a shunt of approximately 15%.

CONCLUSION

Misdiagnosis of this entity as bacterial pneumonia or influenza can be disastrous, since steroid therapy may be lifesaving in patients with significant chemical pneumonitis.^{1,4,6 p523} Patients who seem well but have had a significant exposure to a toxic fume may need to be observed for 24 hours, while, in those with obvious respiratory distress, steroids and other modes of respiratory support should be instituted in the emergency department.

Use of shunt studies is a more sensitive test to detect pulmonary injury and may be a helpful test in patients with equivocal changes in chest x-ray films or ABGs.

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