Medical history a critical step in metal fume fever diagnosis

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Abstract

Metal fume fever is an acute, severe occupational syndrome usually in welding maneuvers and despite protective rules, sporadic cases are recorded by medical departments. Welding is an important occupational activity because a fraction by 0.2% to 2.0% of the working population from industrialized countries has been reported to be engaged in welding. We report a case of a 42-year-old male presenting with metal fume fever, 10 days after oxycutting galvanized steel work. Primary care physicians can early detect patients with metal fume fever but identification of an inhalation syndrome requires an understanding of the etiology and an adequate occupational history. History and the physical exam are one of the easiest ways to set up an early diagnosis with the lowest health costs of metal fume fever. More important early recognition can well-timed a more directed management treatment approach. (Revista de Medicină de Urgenţă, Vol. 3, Nr. 1: 42-44)

Keywords

metal fume fever, zinc oxide, metal oxides, welding, respiratory symptoms

Introduction

Environmental control measures have diminished markedly the incidence of this occupational exposure, but sporadic cases continue to be reported by medical departments [1]. Metal fume fever (MFF) is a professionally acquired disease during or after inhalation of metal oxide fumes [2]. It can also be rarely determined by the exposures to magnesium and copper oxide fumes [3]. The incidence of MFF seems to be undervalued, the diagnosis being overlooked due to the influenza-like symptoms. For example, in the United States is estimated a minimum of 1500–2000 cases of MFF each year [3].

Case report

A 42-year-old man, with no past medical history, cigarette smoker and drinker, presented to the medical depart-

ment for fever, shaking chills, myalgia, intense fatigue, nasal congestion, dry cough and headache. The patient was well until 10 days prior to admission, when he developed the symptoms of upper respiratory infection that got better to the end of the day. Social history revealed the electrician and welding galvanized steel occupation from 1980. Fever recurrence determined the patient to come to the hospital. Admission physical examination showed fever – 38.4°C, blood pressure - 100/60 mmHg, underweight, normal chest examination, and no modifications to rest of examination. A chest radiography revealed no important abnormalities (Fig. 1). An ECG report: normal record. Laboratory data: blood glucose -126 mg/dL, BUN - 48 mg/dL, sodium- 132 mmol/l, aspartate transaminase - 71 U/L, lactate dehydrogenase - 193 U/L, γ-glutamyl transpeptidase - 110 U/l, platelets - 141 000/m3, fibrinogen – 482 mg/dl, peripheral neutrophil leukocytosis, urinalysis – unremarkable. Respiratory functional tests with no abnormalities. He was admitted for a provisional diagnosis of viral syndrome and treated with symptomatic therapy and intravenous hydration. Abdominal ultrasound showed no important findings. Hepatitis markers HbsAg and anti-HCV were absent. Repeated blood and urine cultures in fever peaks were negative. However the condition of patient followed same pattern in the first two days with fever – 41-42 °C, chills, intense malaise, dry cough and headache. Comprehensive medical history shown the involvement of patient in the last 10 days to an oxycutting galvanized steel work with oxide zinc fumes in closed higher spaces with no ventilation and with no protection measures. The diagnosis of MFF was confirmed by the blood zinc value of 747 μg/dL (the normal recommended exposure limit < 1000 μg/L). The symptomatic therapy associated with chelating drug - Calcium disodium ethylenediaminetetraacetate (CaNa2EDTA) - dose of 50 mg/kg/day, for 5 days, starting with the third day of hospitalizations; interrupt for 7 days, and then repeated. Rapid recovery was observed with normalization of blood glucose – 96 mg/dl, alanin transaminase - 85 U/l, aspartate transaminase - 96 U/l, INR - 1.87, reduction of fibrinogen to 460 mg/dL, and blood zinc value after 5 days of chelating therapy was 493 μg/dL. The patient was discharged with recommendation to come back after 7 days to repeat the chelating therapy.

Discussions

Metal fume fever (MFF) is also termed “brass-founders’ ague” or “zinc shakes”, „Monday morning fever” and is a
professionally acquired disease defined mainly by chills, a self-limiting fever, general malaise, and myalgia during or after inhalation of metal oxide fumes [2]. It is commonly described as a systemic disorder in welders - workers that usually maneuver galvanized steel with secondary releasing of zinc oxide fumes. Therefore occupational inhalation of zinc oxide fumes occurs during zinc welding, smelting, and galvanizing, and produces a dose-dependent inflammatory response in the lung.

Onset is typically rapid, occurring within the first few hours, and resemble a flu-like illness with cough, dyspnoea, chest tightness, headache, fever, myalgia, arthralgia, sweating, and sometimes a metallic taste, nausea, vomiting, blurred vision, salivation, and a neutrophil leucocytosis. Other symptoms may also include hoarseness, sore throat, paroxysmal coughing, rapid pulse, malaise, shortness of breath, and abdominal cramps. Respiratory symptoms generally disappear in 1-4 days with usual supportive care. In some cases, there is a rapid recovery in 24 hours [3]. Non-specific neurological effects such as headache and malaise are characteristic of MFF. Chest X-ray may show transient ill-defined opacities.

Symptoms of MFF may get better to the end of day and the working week (possibly due to the development of short-term immunity) but reappear after the weekend giving rise to the term ‘Monday morning fever’. This kind of immunization is probable related to the inducing of metallothionein proteins synthesis having the role to attach heavy metals, and it may be decreased in case of fluctuating exposures [4]. Tolerance to the inflammatory effects of inhaled zinc may explain, in part, the occurrence of symptoms at lower concentrations in healthy subjects compared to those occupationally exposed. On the other hand, the symptoms may also develop at ‘safe concentrations of oxides fume levels’ [3].

In acute exposure, the zinc oxide irritates the respiratory tract but in the chronic or repeated exposure can determine the asthma, process potentate by the cigarette smoking. The pathophysiology is unclear but seems to reflect a direct toxic effect [3]. Studies demonstrated a dose-dependent increase in the bronchoalveolar lavage fluid of proinflammatory cytokines triggered by zinc oxide inhalation. Otherwise, these data supports an immunological process that is similar to ‘farmer’s lung’ and other forms of extrinsic allergic alveolitis.

Diagnosis is based on the history of exposure, chest examination and chest X-ray, and pulmonary-function tests. Evidence of possible exposure is critical, as medical history. In all cases of inhalation disorders caused by toxic smokes, the primary investigation is toward the pulmonary system. Pulmonary function tests may be modified but not specifically. Ever more, some authors are suggesting diagnostic criteria’s by using of terms as ‘possible MFF’ or ‘probable MFF’ which may be useful to the diagnostic approaching [5].

Differential diagnosis is made with any process that presents with pulmonary symptoms or signs: pneumonia, congestive heart failure, pulmonary embolism, COPD exacerbation, precipitant of noncardiogenic pulmonary oedema, myocardial infarction, pleurisy, or tuberculosis, any toxic inhalation exposure (e.g., cyanide, carbon monoxide, and other toxic fumes). In the case of pneumonic process, the investigation for chemical pneumonitis after metal fume exposure caused by cadmium, manganese, mercury, and nickel fumes is a major diagnostic step [3].

Symptomatic and supportive measures are the priority in the management of ‘metal fume fever’. Calcium disodium ethylenediaminetetraacetate (CaNa2EDTA) is the chelator of choice based on case reports that demonstrate normalization of zinc concentrations, but there are few clinical data to confirm the efficacy of this agent.

The prognosis is usually excellent with complete recovery within one to four days if exposure ceases, although there are occasional reports of on-going symptoms and signs of airways obstruction in individuals with no previous history of asthma. In case of mild-to-moderate exposures of toxic smokes, the prognostic is generally very good, with the usual outcome return to full recovery without sequel. With more severe exposures, lungs may become severely damaged and develop chronic pulmonary fibrosis. No long term complications are known.
References
