CASE REPORT

Metal fume fever presenting as aseptic meningitis with pericarditis, pleuritis and pneumonitis

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Background Metal fume fever (MFF) is a well-known complication of zinc oxide fume inhalation. Prompt recognition of this condition is essential for the proper medical management of this self-limited disease.

Aim To present a unique and unusual case of MFF.

Results Our patient is a 25-year-old male welder who had MFF and presented with aseptic meningitis with pericarditis, pleuritis and pneumonitis. To our knowledge, this is the first case of MFF presenting with these signs and symptoms.

Conclusions MFF can present with a systemic inflammatory response causing a multi-organ serositis. Our case highlights the utmost importance of obtaining an occupational history on all our patients, even if they are critically ill.

Key words Meningitis; metal fume fever; occupational lung disease; pericarditis; toxic inhalation.

Introduction

Metal fume fever (MFF) is an acute, self-limiting, influenza-like illness that occurs most commonly in welders. This condition typically presents with fever, chills, cough, dyspnoea, headache, myalgias and malaise within 4–12 h of exposure to zinc, iron or copper oxide fumes [1]. We present a case of MFF in a 25-year-old male who presented with aseptic meningitis with pericarditis, pleuritis and pneumonitis. To our knowledge, this is the first case of MFF associated with inflammation of the meninges, pericardium and pleura to this degree of severity.

Case report

Our patient was a 25-year-old male welder with well-controlled, mild intermittent asthma who presented in early December 2003 to another hospital with recent onset of headache, neck pain, myalgias, fever and chills. Prior history revealed that after returning to work from a long absence, his job involved demolition of a large car manufacturing plant along with other flame cutters cutting galvanized steel. Our patient and 10–12 other welders were working in an area directly above where galvanized steel was being cut. The ventilation was poor, and he inhaled a substantial amount of fumes without wearing a respirator, causing him and several of his co-workers to cough. That night, he developed a headache and stiff neck, which worsened by the next morning. Over the counter ibuprofen provided slight relief. Five of his co-workers also complained of headaches and stiff necks, and some of them also took ibuprofen with relief. He returned to the work site the following day and cut galvanized steel conduit-containing copper wires. Blue smoke was released after the conduit was cut, and he became dyspnoeic. He was also coughing and complained of throat irritation and a metallic taste in his mouth and had to suspend his welding for 1 h. After finishing his work, his symptoms progressed to diffuse myalgias, headache, neck stiffness and difficulty in breathing. On the way home from his work site, he developed orthopnoea. These symptoms worsened significantly, which prompted him to seek medical attention.

On evaluation, he was found to be febrile (38.5°C), tachycardic (114 beats/min) and tachypnoeic (28 breaths/min). Neck rigidity was present. Chest radiography was clear. A lumbar puncture was performed and showed only a mild lymphocytic pleocytosis. Arterial blood gas on room air was pH 7.39, PaCO₂ 45 mmHg and PaO₂ 76 mmHg. He was diagnosed with aseptic meningitis and admitted to the hospital for further evaluation and intravenous antibiotic therapy. Computed tomography
(CT) of the chest revealed a left lower lobe infiltrate with several nodular densities.

Transthoracic echocardiography revealed a small pericardial effusion. His symptoms and oxygenation worsened despite this therapy, and he was transferred to our institution for further work up and management. Upon arrival at our intensive care unit, the patient was complaining of an intense, sharp, anterior pleuritic chest pain. He was afebrile but very tachycardic (>150 beats/min) and tachypneic (30–40 breaths/min). On 100% FIO\textsubscript{2} administered via reservoir mask, his oxygen saturation was 92%. Physical examination revealed neck rigidity, maxillary sinus tenderness, coarse breath sounds with crackles bilaterally and a pericardial friction rub. Laboratory examination demonstrated a mild leukocytosis (10.2 thousand/µl), erythrocyte sedimentation rate of 56 mm/h, and lactate dehydrogenase of 561 U/l. Arterial blood gas on 100% FIO\textsubscript{2} revealed a pH of 7.47, pCO\textsubscript{2} of 28 mmHg and pO\textsubscript{2} of 55 mmHg. Anti-nuclear antibody, influenza A/B antigen, urinary Histoplasma antigen, HIV antibody and double-stranded DNA antibody were all negative. CT of the chest was repeated, and it showed development of bilateral consolidations, bilateral pleural effusions and an enlarged heart with a large pericardial effusion (Figure 1). Transthoracic echocardiography confirmed the presence of a large, free-flowing pericardial effusion. Blood, urine, sputum and viral cultures were taken, and the patient was started on intravenous broad-spectrum antibiotics, nebulized bronchodilators, intravenous diuretics and intravenous corticosteroids.

In the intensive care unit, we obtained his detailed occupational exposure and noted the temporal relationship to his symptoms. Suspecting MFF, antibiotics, diuretics and corticosteroids were discontinued, and the patient was placed on 50 mg oral indomethacin three times daily. His symptoms improved within 16 h, and within 4 days his pleural effusions and infiltrates resolved almost completely (Figure 2). All cultures were negative. He was discharged in good condition, and his pericardial effusion completely resolved 1 week later. The patient’s complete recovery allowed us to deem him clinically fit to return to work, with the requirement that he always wear a respirator when welding. Pulmonary function testing after this episode was completely normal, and he remains clinically well.

Discussion

It is estimated that >1000 cases of MFF are reported each year in the USA [2]. Patients frequently complain of a sweet or metallic taste in the mouth, irritated or dry throat, intense thirst and chest pain. Clinical and radiographic presentation in MFF varies considerably [1,3–9], and the entire clinical syndrome resembles that of infection or other inflammatory process. Consequently, a detailed occupational history and high index of suspicion

![Figure 1](https://example.com/figure1.png)  
**Figure 1.** Computed tomography scan of the chest upon admission which shows pericardial effusion, bilateral pleural effusions and dense bilateral pulmonary consolidations.
are essential. There is no diagnostic test for MFF, although urinary [10] and serum [4] zinc levels may be elevated. It is of utmost importance that the presence of infection be ruled out. Treatment is supportive, consisting of bedrest, analgesics and antipyretics. Intravenous corticosteroids have been advocated [11], but this has not been studied in a randomized, controlled fashion.

Our patient had the typical signs and symptoms of MFF which developed soon after inhaling metal fumes with negative virologic and bacteriologic studies. The time course of disease was longer than is typical [1,6–7]. Nevertheless, the temporal association between inhalation of metal fumes and the development of symptoms, the dramatic improvement with indomethacin alone (he received only one dose of diuretic and corticosteroid) and the similar symptoms in other co-workers similarly exposed strongly suggest the diagnosis of MFF. Although we believe his syndrome was due to exposure to zinc oxide, we acknowledge that the patient’s presentation may have been due to a mixed exposure to a number of substances, including cadmium, nitrogen dioxide, carbon monoxide or copper oxide. Although pleuritis has been previously reported [10], this is the first report—to our knowledge—of meningitis, pericarditis and pneumonitis to this degree of severity in association with MFF.

The pathophysiology of MFF remains unclear. The hypotheses postulated include delayed hypersensitivity reaction to exposure to zinc oxide [1], antigen–antibody complex [12,13] and a direct toxic effect of zinc oxide on human tissue. This latter theory is supported by reports of obstructive lung disease [9,14–17] and skeletal muscle and myocardial injury [18] following MFF. Recent research has also elucidated the role of cytokines [2,19–21] and oxidative damage by neutrophils [2,5,19,22–25]. We postulate that the inhalation of metal fumes in our patient induced a cytokine-mediated systemic inflammatory response resulting in his clinical presentation.

As our case demonstrates a high index of clinical suspicion is essential to the diagnosis of MFF. A thorough occupational history should always be attempted, even in critically ill patients if feasible, so that potentially toxic antimicrobial and/or anti-inflammatory therapies are not given unnecessarily.

**Conflicts of interest**

None declared.

**References**