possible due to rapid hemodynamic stabilization and adequate diagnostic evaluation using echocardiography.

Two case reports have been published showing delayed and nonfatal cardiac tamponade after acupuncture. In Japan, the permanent embedment of acupuncture needles in the musculature represents a common acupuncture technique to relieve pain. As a complication, traumatic cardiac tamponade was caused in a 69-year-old woman after migration of needles from a distant part of the body via the venous route and penetration of the right ventricle. A similar pathogenetic mechanism of slow migration over years and, ultimately, penetration of the pulmonary artery was suspected in a 52-year-old man who presented with cardiac tamponade a few years after a broken acupuncture needle had not been removed.5

A case of a fatal cardiac tamponade in a 40-year-old woman that occurred immediately after acupuncture was reported by Halvorsen et al.6 Their precise workup revealed mechanical injury of the right ventricular wall and a congenital foramen in the lower third of the sternum as a crucial factor.

Our case report documents a nearly fatal complication of acupuncture after insertion of a needle into the sternum at the level of the fourth and fifth intercostal space, which represents an acupuncture point called Ren 17. In the critical analysis of the cause of this dramatic adverse event, one has to consider either a lack of anatomic knowledge or an incorrect application of the procedure. The acupuncture needle may have been inserted in a perpendicular direction in an emaciated patient.1,8 Although the involved acupuncturist had gained extensive experience for over years and, ultimately, penetration of the pulmonary artery was suspected in a 52-year-old man who presented with cardiac tamponade a few years after a broken acupuncture needle had not been removed.3

A case of a fatal cardiac tamponade in a 40-year-old woman that occurred immediately after acupuncture was reported by Halvorsen et al.6 Their precise workup revealed mechanical injury of the right ventricular wall and a congenital foramen in the lower third of the sternum as a crucial factor.

We describe the case of a heavy marijuana and tobacco smoker who presented with progressive exertional dyspnea of 2 months' duration, and bilateral nodular lung infiltrates. Examination of the lung fields was normal, and lung function tests showed mild airflow obstruction with moderately reduced gas transfer. BAL returned green-black fluid consisting predominantly of macrophages laden with carbon pigment. Thoracoscopic lung biopsy showed miliary necrotizing granulomata with an alveolar exudate of carbon-laden macrophages within macroscopically blackened lung. The differential diagnosis of pulmonary granulomata in this patient is discussed.

**Key words:** granuloma; marijuana; pulmonary; tobacco

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**Necrotizing Pulmonary Granulomata in a Marijuana Smoker**

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We describe the case of a heavy marijuana and tobacco smoker who presented with progressive exertional dyspnea of 2 months’ duration, and bilateral nodular lung infiltrates. Examination of the lung fields was normal, and lung function tests showed mild airflow obstruction with moderately reduced gas transfer. BAL returned green-black fluid consisting predominantly of macrophages laden with carbon pigment. Thoracoscopic lung biopsy showed miliary necrotizing granulomata with an alveolar exudate of carbon-laden macrophages within macroscopically blackened lung. The differential diagnosis of pulmonary granulomata in this patient is discussed.

Key words: granuloma; marijuana; pulmonary; tobacco

**M**arijuana is the most commonly used illicit drug in Western societies. In Australia, it is used regularly by 11% of the adult population, with even higher rates of regular use among adolescents.1,2 The most common route of administration is by inhalation, which is convenient, rapidly absorbed, and has good bioavailability.3 Marijuana smoke is not benign, however, being similar in composition to tobacco smoke, except for the absence of nicotine and the presence of cannabinoids. Marijuana smoke also results in more tar and carbon monoxide being delivered to the lungs than is delivered by tobacco smoke.4,5 This occurs because marijuana smokers typically inhale more deeply and with longer breath-holding times compared to tobacco smokers and therefore facilitate deposition of carbon particles in the alveoli.4 This type of breathing maneuver may also account for the increased risk of spontaneous pneumothorax seen in marijuana smokers.6

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The most common respiratory side effects noted by marijuana smokers include cough, dyspnea, bronchitis, and nasal congestion. Although the long-term effects of habitual marijuana smoking have not been completely established due to a lack of long-term longitudinal human data, it is thought that chronic marijuana smokers develop chronic bronchitis and airflow limitation with similar frequency to that seen in tobacco smokers.7

As the prevalence of marijuana smoking is not declining, and indeed is rising in adolescents, we believe it is important to highlight the potential hazards of habitual marijuana smoking.

Case Report

A 23-year-old woman presented to our emergency department with progressive exertional dyspnea, and bilateral lung infiltrates on chest radiology. This occurred on a background of long-standing heavy marijuana and tobacco smoking, and previous IV opioid abuse. The patient had been asymptomatic until 2 months prior to presentation, when she developed slowly progressive exertional dyspnea. At presentation, she was unable to walk at her usual pace and had trouble completing heavy housework, corresponding to a Medical Research Council dyspnea grade of 2.8 She also reported a morning cough, but was not producing any sputum. There was no history of night sweats, fevers, or weight loss, and the patient was not diabetic. There was no history of significant occupational exposure that would account for her pulmonary symptoms. She was known to have mild episodic asthma not requiring regular inhaled corticosteroid use.

For 5 years, the patient had been a heavy marijuana user, smoking 2 to 3 g of marijuana a day via a water pipe. The marijuana she smoked was illicit, and she was unaware of the conditions under which it was grown, or any details about its drying and preparation. In addition, she smoked 5 to 10 tobacco cigarettes per day, and had used IV heroin for a short period 2 years prior to this presentation. There was no history of crack cocaine use.

The patient had cutaneous varicella 6 months prior to presentation. At that time, she had widespread skin lesions but no respiratory or neurologic symptoms. She is a single mother of four children and keeps a dog and a cat, but has never kept birds.

Physical examination was unremarkable, except for a resting tachypnea with a respiratory rate of 24 breaths/min. The patient’s temperature was 36.9°F. Auscultation of the lung fields was normal, and there was no cyanosis or clubbing. Cardiovascular examination was normal, with no evidence of endocarditis. There was no hepatomegaly or signs of chronic liver disease, and no evidence of recent use of illicit IV drugs.

The chest radiograph showed a bilateral lung infiltrate, with diffuse small nodules evenly distributed throughout both lungs. High-resolution CT scanning of the chest confirmed the presence of bilateral diffuse lung nodules, 1 to 4 mm in diameter (Fig 1). A full blood examination was normal with no eosinophilia. The erythrocyte sedimentation rate was not elevated, and serum calcium and angiotensin-converting enzyme levels were normal. Antinuclear antibody was present at a titer of 1:160 with a nucleolar pattern; however, double-stranded DNA and antineutrophil cytoplasmic antibodies were negative. She had chronic hepatitis-C infection but normal liver function tests and had not received treatment for this. HIV and hepatitis-B serologies were negative. Mantoux testing (10-U purified protein derivative) and serum aspergillus precipitins were negative. Sputum microscopy and culture for bacteria, acid fast bacilli, fungi and viruses were negative.

Pulmonary function tests demonstrated trivial airflow obstruction: FVC, 3.62 L (88% predicted); FEV1, 2.65 L (79% predicted); and FEV1/FVC, 76% with no acute bronchodilator response.9 Carbon monoxide transfer factor was mildly reduced at 19.2 CO/min/mm Hg (74% predicted).10

![Figure 1. High-resolution CT chest scan revealing diffuse nodules present throughout both lungs.](image1)

![Figure 2. BAL fluid with blackened particulate matter.](image2)

![Figure 3. Necrotizing granuloma containing histiocytes and multinucleated giant cells (hematoxylin-eosin, original × 135).](image3)
The patient underwent fiberoptic bronchoscopy that revealed generalized tracheobronchitis but no other endobronchial abnormality. BAL fluid contained green-black pigmented material (Fig 2) and had a cell count of 85% macrophages, 13% neutrophils, and 2% lymphocytes. Most of the alveolar macrophages were laden with carbon pigment. A transbronchial biopsy could not be performed, as the patient became hypotensive and bronchoscopy had to be abandoned. Subsequently, a video-assisted thoracoscopic biopsy of the right lung was performed. At surgery, the right lung was blackened with miliary white nodules over the entire lung surface. Microscopy of lung sections showed numerous well-circumscribed necrotizing granulomata, which contained histiocytes and scattered multinucleated giant cells (Fig 3). There were no acid-fast bacilli, or birefringent foreign materials seen within the granulomata, and no evidence of vasculitis. A single fungal element was seen on one section only within the lung parenchyma (Fig 4). Elsewhere there was a florid alveolar exudate of alveolar macrophages that appeared to coat the alveolar membrane (Fig 5). Culture of lung tissue was negative for bacteria, acid-fast bacilli, fungi, and viruses.

The patient discharged herself from hospital prior to any treatment. She has not attended for further follow-up, and we have been unable to investigate her water pipe.

**DISCUSSION**

This case demonstrates a number of abnormalities in lung function and histology related to chronic marijuana and tobacco use. Some of these changes have been previously described; however the finding of diffuse necrotizing granulomata is unusual, and most previous reports of blackened BAL fluid have occurred in crack smokers.

Tobacco smoking alone can cause an accumulation of pigmented macrophages in alveolar spaces, as described with respiratory bronchiolitis-associated interstitial lung disease. However, marijuana smoking has also been shown to increase respiratory tract inflammation and alveolar macrophages in BAL fluid, an effect that was additive to that of tobacco smoking. It is unclear which component of marijuana smoke induces this exudate of alveolar macrophages, but as the macrophages are carbon laden, the increased tar and carbon retention associated with marijuana smoking may be the cause. A similar exudate of alveolar macrophages has been shown in primates exposed to 12 months of daily marijuana smoke inhalation, and in a human postmortem series of heavy marijuana smokers. In the latter series, all 13 individuals had a dose-related alveolar infiltrate of pigmented macrophages similar to that seen in this case (Fig 5). This exudate of alveolar macrophages contributes to the reduction in single-breath carbon monoxide diffusing capacity seen with chronic marijuana smoking and seen in our patient.

Blackened BAL fluid has been described principally in association with crack cocaine smoking; however, this patient had no history of crack cocaine use and it is a very uncommon illicit drug in Australia. This patient smoked marijuana predominantly via a water pipe that does not filter particulate-phase smoke toxins such as tar from marijuana smoke. In addition, smoking marijuana via a water pipe can result in a buildup of carbonaceous residue within the water pipe, similar to that seen with crack smoking. This residue is believed to be responsible for the blackened BAL fluid seen in crack smokers; but water pipes, if used correctly, do not allow inhalation of particulate matter from marijuana smoke. This makes the cause of the blackened BAL fluid in our patient unclear. However, it is most likely due to a combination of tar and particulate matter inhaled with marijuana smoke, incorrect water pipe use, or use of a homemade water pipe without a consistent water seal.

Tobacco smoking alone has not been associated with granulomatous inflammation in humans. Respiratory bronchiolitis-associated interstitial lung disease is the only interstitial lung disease thought to be directly related to tobacco smoking. However, granulomatous inflammation has been shown to occur in primates exposed to marijuana smoke. The granulomata seen in these primates generally had birefringent material or foreign bodies within them. In our patient, there were no birefringent materials or foreign bodies within the granulomata, so it is unlikely that talc or starch granules inadvertently injected with IV opioid abuse caused the granulomata.

Although persistent pulmonary granulomata have been described following varicella pneumonia, this is unlikely to have caused the granulomata in this case. Our patient did not have symptoms of lung involvement at the time of varicella infection, and the granulomata lacked the fibrous
capillary usually seen surrounding the granulomata in post varicella granulomatous lung disease. Necrotizing sarcoi
d granulomatosis can cause lesions very similar to those seen
in our patient. This is unlikely, however, given that the
patient had no systemic symptoms of sarcoidosis, normal
erthrocyte sedimentation rate and angiotensin-converting
enzyme level, and no evidence of vasculitis involving
arteries or veins, as would be expected in necrotizing
sarcoi
d granulomatosis.

Infection is a common cause of pulmonary granulomata,
and is potentially involved in the pathogenesis of our case.
Mycobacterial infection is unlikely, given the negative Mantoux and sputum and lung cultures. Bacteria have
been shown to contaminate marijuana, with Gram-nega-
tive bacilli being the most common organisms. Despite
this, pulmonary bacterial infection from marijuana smok-
ing is not a clinical problem, which probably relates to
killing of bacteria during combustion of marijuana. Mari-
juana is often contaminated with fungal spores that are
respirable in marijuana smoke from either cigarettes or
water pipes. These spores do not appear to be inactivated
during the combustion of marijuana and are not filtered
from marijuana smoke by water pipes.

The most common fungi are Aspergillus and Mucor species, but many other fungal species such as Thermoactinomyces, Mi-
cropyllospora, Penicillium, and Rhizoporus contaminate
marijuana. Fungi inhaled with marijuana smoke are
known to cause disease in immunocompromised individ-
uals. Invasive pulmonary aspergillosis has been described
in immunocompromised individuals smoking marijuana.

In a series of immunocompetent marijuana smokers, 11 of
21 had serum precipitins to aspergillus, compared to 1 of
10 control subjects. The clinical significance of this is not
clear; however, it is clear that fungi contaminate marijuana
and are inhaled with marijuana smoke, leading to potential
fungal infection or hypersensitivity to inhaled fungi. In
our case, serum precipitins to aspergillus were not detected,
and there was only a single fungal element seen in the lung
on biopsy. We were unable to culture fungus from lung
biopsy specimens, and the patient was not significantly
immunocompromised. Although we have been unable to
conclusively demonstrate the presence of fungal infection
in our patient, we believe fungal infection or hypersensi-
tivity to inhaled fungi are the most likely cause of the
necrotizing granulomata seen in our case.

This case highlights some of the pulmonary hazards of
chronic marijuana smoking. To our knowledge, this case
represents the first reported demonstration of necrotizing
granulomata in the setting of heavy marijuana smoking.
Even though the exact cause for these remains unclear, it
is likely that these granulomata are a result of chronic
marijuana smoking and perhaps the inhalation of a con-
taminant such as a fungus with the marijuana smoke. This
etiolo
gy should be considered in other patients who are
chronic marijuana smokers with radiologic changes of
diffuse pulmonary nodules.

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