Ventricular tachycardia in a disseminated MDR-TB patient: a case report and brief review of literature

Hui Li1, Ran Li1, Jiuxin Qu1, Xiaomin Yu1, Zhixin Cao2, Yingmei Liu1, Bin Cao (✉)1,a

1 Department of Infectious Diseases and Clinical Microbiology, Beijing Chao-Yang Hospital, Beijing Institute of Respiratory Medicine, Capital Medical University, Beijing 100020, China; 2Department of Respiratory Medicine and Critical Care, Beijing Chao-Yang Hospital, Beijing Institute of Respiratory Medicine, Capital Medical University, Beijing 100020, China

© Higher Education Press and Springer-Verlag Berlin Heidelberg 2014

Abstract Although significant breakthroughs have been achieved in tuberculosis management, we still encounter numerous difficulties in diagnosis and treatment of the disease. Additionally, a new challenge, multidrug-resistant tuberculosis (MDR-TB) with unspecific clinical presentation, often results in delayed diagnosis. In this paper, we reported a case of disseminated tuberculosis with rare presentation of ventricular fibrillation, which proved resistant to both isoniazid and rifampicin. A review of literature showed that ventricular fibrillation or tachycardia in tuberculosis patients with pericarditis or myocarditis has been sporadically reported in the past, but none has been conducted involving patients with MDR-TB infections.

Keywords tuberculosis; MDR-TB; ventricular tachycardia

Introduction

Ventricular tachycardia or fibrillation in tuberculosis patients has been intermittently reported, but not in severe disseminated multi-drug resistant tuberculosis (MDR-TB) patients who responded well to reserve anti-tubercular drugs. We revealed a case of severe disseminated MDR-TB with a rare presentation of ventricular fibrillation. Mycobacterium tuberculosis had infected almost every organ of the body, including the brain, lung, heart, liver, spleen, bone and joint. After treatment with electrical defibrillation, anti-arrhythmia agents, and a 5-drug anti-tuberculosis regimen, the female patient achieved a stable condition.

Case presentation

On June 19, 2012, a 22-year-old, previously well female college student was transferred to our hospital with a history of arthralgia for 1.5 years. She also suffered from fever (Tmax 41 °C), cough, and progressive dyspnea for 1.5 months. Rheumatoid arthritis was suspected 1.5 years previously when she consulted a doctor regarding pain in her distal interphalangeal joints, which was later excluded due to negative results for related factors, including rheumatoid factor, anti-citrullinated peptide, anti-keratin antibodies and anti-perinuclear factor. Six months before admission, her right ankle became swollen with severe pain, and the swelling portion burst spontaneously with dark bloody secretion after 2 months of treatment with traditional Chinese medicine. Unfortunately, the thick, bloody secretion garnered little attention from the doctor, and it was not tested for TB and other pathogens. Enhanced computed tomography (CT) revealed several small nodules in the right lung and enlarged mediastinal lymph nodes, as well as enlarged liver and spleen accompanied by well-defined ovoid low-density lesions (Fig. 1). However, she opted out of further examination, and definite diagnosis was not made at that time.

Roughly one month before admission to our hospital, the patient began to suffer from fever, cough with yellow sputum, and shortness of breath on exertion, which were treated as symptoms of common cold. During treatment, her condition suddenly deteriorated, with the patient experiencing palpitation and orthopnea. Then, she was hospitalized in a tertiary hospital in Beijing for acute left heart failure. CT scan indicated multiple nodules in both lungs with pleural effusion accompanied by brain abscess and bony destruction of right ankle (Fig. 2A to 2C).

Diagnosis of infectious endocarditis was presumed. However, her condition deteriorated despite treatment with
potent, broad-spectrum antibiotics and cardiovascular drugs. Particularly, her heart function noticeably declined, with the left ventricular ejection fraction decreasing from 59\% to 22\%. She even had two episodes of ventricular fibrillation (May 19, 2012) and recurrent ventricular tachycardia (electrocardiogram in Fig. 2E and supplementary Fig. 1). Echocardiography showed decreased motion of both ventricles without any vegetation, and repeated blood culture showed negative results. Therefore, infectious endocarditis was excluded from consideration.

On June 18, 2012, the patient was transferred to our hospital. At admission, her blood pressure was 86/40 mmHg, with heart rate of 150 to 160/min and with fever at 39 °C. Systolic murmurs in the mitral valve were observed, and the tricuspid valve and decreased breath sounds in the right lung could be noted. In addition, her liver and spleen were evidently enlarged, but no edema was seen in her lower extremities. A review of her history did not reveal the use of corticosteroids and other immunomodulators. Both her parents were healthy, and no previous or current history of tuberculosis was noted in her close contacts. Chest CT scan showed multiple nodules in both lungs (Fig. 2D). Hematological analysis revealed mild anemia and normal white blood cell count. Biochemical investigation yielded the following results: elevated alkaline phosphatase (ALP); lactate dehydrogenase (LDH) with decreased albumin; and troponin C (CTnI) levels in 48 h of serial monitoring during the worst condition of her heart function (ranging from 0.00 ng/ml to 0.04 ng/ml), which were within the normal range. The patient tested negative for HIV antibody. Pathogenic examination found acid-fast bacilli and positive TB-PCR in the sputum specimens collected on three days (June 19, 22, 23) (Fig. 3A). Pulmonary tuberculosis was suspected, and combined anti-tuberculosis therapy (isoniazid, rifampicin, ethambutol, and pyrazinamide) was provided. After four weeks of treatment, she still suffered from high fever and paroxysmal ventricular tachycardia. During hospitalization, she was afflicted with seizures, gastrointestinal bleeding, recurrent heart failure, and tachycardia. On July 20, sputum and bone marrow cultured positive for M. tuberculosis (Fig. 3B, 3C).

Gene mutations of rpoB and katG codon 315 were detected by new GenoType MTBDRplus assay [1] (Hain Lifescience GmbH, Nehren, Germany), which were responsible for rifampin and isoniazid resistance, respectively. Disseminated MDR-TB was subsequently diagnosed, and treatment was changed to reserve anti-tuberculosis drugs (amikacin, protionamide, pyrazinamide, ethambutol, and moxifloxacin), which yielded a satisfactory result. By August 3, 2012, ventricular tachycardia disappeared, the patient’s temperature returned to normal, and she was discharged in a stable condition on the 5-drug anti-tubercular regimen. Two months later, the enlargement of the liver and spleen was slightly reduced, and echocardiography showed fibrosis changes in the left ventricular wall (Fig. 4). Her heart condition evidently improved, with the left ventricular ejection fraction normalizing to 66\%.

Discussion

Since streptomycin, the first anti-tuberculosis drug, was introduced into clinical practice by Waksman 60 years ago, great breakthroughs have been achieved in tuberculosis management. Nearly 6 million people’s lives have been saved over the past 15 years. However, the disease has not been completely conquered: tuberculosis continues to infect and even kill millions of people every year in the new century [2]. Due to inadequate treatment, a new challenge to the management of the disease is posed by multidrug-resistant tuberculosis.

Given the lack of effective and rapid laboratory diagnostic capacity for tuberculosis, especially for MDR-TB, most patients have been severely sick and already infected many others by the time they were treated [2]. MDR-TB is a serious challenge worldwide, especially in low-income countries [3]. A national survey in China revealed that 5.7\% new cases and 25.6\% previously treated cases were MDR-TB [3]. Due to its unspecific clinical presentation, disseminated tuberculosis exerts more difficulty in differential diagnosis. About 33\% to 88\% cases of all disseminated tuberculosis found at autopsy.