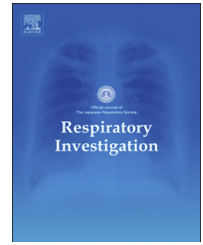




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Case report

Scedosporium aurantiacum brain abscess after near-drowning in a survivor of a tsunami in Japan

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ABSTRACT

Many victims of the tsunami that occurred following the Great East Japan Earthquake on March 11, 2011 developed systemic disorders owing to aspiration pneumonia. Herein, we report a case of tsunami lung wherein *Scedosporium aurantiacum* was detected in the respiratory tract. A magnetic resonance image of the patient's head confirmed multiple brain abscesses and lateral right ventricle enlargement. In this case report, we describe a potential refractory multidrug-resistant infection following a tsunami disaster.

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Abbreviations: CNS, central nervous system; CT, computed tomography; BAL, bronchoalveolar lavage; MRI, magnetic resonance imaging; CSF, cerebrospinal fluid; M, male; F, female; PCR, polymerase chain reaction; VRCZ, voriconazole; MCFG, micafungin; L-AMB, liposomal amphotericin B; FLC, fluconazole; PMX-DHP, polymyxin B-immobilized fiber column direct hemoperfusion

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1. Introduction

Several survivors of the tsunami in northeastern Japan developed lung and brain abscesses caused by *Scedosporium* spp. [1,2]. Central nervous system (CNS) infections secondary to *Scedosporium* spp. can occur in near-drowning individuals whereupon large inoculums of the fungi are aspirated into the respiratory tract and reach the CNS through hematogenous spreading. In this report, we describe a case of a female patient who acquired lung and CNS *Scedosporium* infection following a near-drowning aspiration event during a tsunami. We focus on the difficulties in establishing the correct diagnosis and choosing the best therapeutic approach.

2. Case

A 68-year-old Japanese woman who had been washed away by a tsunami was rescued and sent to a coastal clinic. She was gasping and short of breath after coughing paroxysms, and was transferred to a neighboring hospital owing to low oxygen saturation (67% on 10 L/min of supplemental oxygen) as determined by pulse oximetry. At the hospital, she stated that she underwent insulin therapy for diabetes, and a physical examination upon admission revealed a body temperature of 33.5 °C, blood pressure of 92/46 mmHg, and a regular pulse of 75 beats per minute. There was no evidence of trauma or fractures anywhere on her body. Lung auscultation revealed coarse crackles in both lungs. Her heart sounds were normal. She was confused, but a neurologic examination yielded negative findings. The laboratory findings were as follows: white blood cell count, 4200 cells/ μ L (reference range, 4000–9000 cells/ μ L); hemoglobin, 12.9 g/dL (reference range, 12.0–15.0 g/dL); platelets, 85,000 cells/ μ L (reference range, 13.0–34.0 cells/ μ L); aspartate aminotransferase, 2140 IU/L (reference range, 8–40 IU/L); alanine aminotransferase, 748 IU/L (reference range, 5–35 IU/L); lactate dehydrogenase, 1160 IU/L (reference range, 121–226 IU/L); and a negative C-reactive protein test. Arterial gas analysis showed severe hypoxemia (pH, 7.25; PaCO₂, 45; and PaO₂, 41 Torr) even though 10 L/min of supplemental oxygen was being administered. A chest radiograph revealed diffuse infiltration in both lung fields. On the basis of these findings, the patient was diagnosed with aspiration pneumonia due to near-drowning. The patient was treated with 0.5 g of meropenem intravenously twice a day and steroid pulse therapy. Eight hours after admission, her body temperature was 38.2 °C and her oxygen saturation, measured by pulse oximetry, was 92% on 4 L/min of supplemental oxygen. On hospital day 7, she had a body temperature of 37.4 °C, a good appetite, and was stable without supplemental oxygen. The aspartate aminotransferase, alanine aminotransferase, and lactate dehydrogenase levels also normalized; however, the C-reactive protein level had risen to 10.2 mg/dL (reference range, 0–0.40). A chest radiograph showed decreased infiltrates on both sides. On hospital day 16, the patient was found to still have a body temperature of 37.5 °C; therefore, the treatment was switched from meropenem to 2 g of piperacillin intravenously twice a day. On hospital day 26, chest computed tomographic (CT)

scans showed pleural effusion and multiple nodular lesions in both sides (Fig. 1a), and the level of (1 \rightarrow 3)- β -D-glucan had increased to 123.4 pg/mL (reference range, 0–20 pg/mL). Fungal lung disease was suspected, and treatment with 150 mg of micafungin intravenously once a day was initiated; however, the patient then presented with a body temperature of 38.5 °C. As a result, she was transferred to our hospital 42 days after her initial visit to the previous hospital. Upon admission to our hospital, she presented with generalized fatigue and had a fever of 39 °C, blood pressure of 142/92 mmHg, and tachycardia at a rate of 112 beats per minute. Auscultation of the chest revealed some crackles in both lungs. Her heart sounds were normal, and an abdominal examination also yielded normal results. Tsunami-associated refractory pneumonia was suspected on the basis of clinical appearance. Upon admission to our hospital, normal flora was isolated from the sputum. The level of (1 \rightarrow 3)- β -D-glucan was 35.5 pg/mL. *Pneumocystis carinii* infection was suspected, and trimethoprim-sulfamethoxazole (trimethoprim 4.8 mg/kg 3 times per day) was started. On day 2 at our hospital, a CT scan of the head demonstrated normal findings, and a scan of the chest revealed bilateral smooth septal thickening, bilateral pleural effusions, and compressive atelectasis (Fig. 1b). Subsequently, Gram staining of bronchoalveolar lavage (BAL) fluid demonstrated filamentous fungi under microscopic examination collected BAL fluid from the left lower bronchus (B9); therefore, treatment with 150 mg of micafungin intravenously once a day was continued. On day 16 at our hospital, the patient became hypotensive and developed right-sided flaccid paralysis. She was therefore diagnosed as having a cerebral infarction and was treated with 30 mg of argatroban and edaravone intravenously twice a day. Owing to the increase in (1 \rightarrow 3)- β -D-glucan levels to 47.8 pg/mL, micafungin-refractory fungal infection was suspected, and the antimycotic therapy was switched to voriconazole at 400 mg twice a day. On day 29 at our hospital, she lost consciousness and developed flaccid paralysis of the left arm and leg (double hemiplegia). A neurological examination disclosed exaggerated tendon reflexes, and brain magnetic resonance imaging (MRI) showed intraventricular abscesses (Fig. 1c). Blood and cerebrospinal fluid (CSF) cultures for bacteria were negative, and the level of (1 \rightarrow 3)- β -D-glucan in the CSF was increased to 243 pg/mL on day 45 at our hospital. After informed consent was obtained from the patient, voriconazole was given in doses of 14 mg consecutively for 10 days intrathecally and the level of (1 \rightarrow 3)- β -D-glucan was found to be reduced to 18.9 pg/mL of CSF on day 54 at our hospital. A diagnosis of *Scedosporium aurantiacum* infection was confirmed on day 62 at our hospital by performing polymerase chain reaction and deoxyribonucleic acid (DNA) sequencing of the filamentous fungi obtained from the BAL specimens taken on day 2 at our hospital and cultured on Sabouraud dextrose agar. Although the *S. aurantiacum* isolates were susceptible to voriconazole, an MRI of the head taken on day 131 at our hospital confirmed multiple brain abscesses and lateral right ventricle enlargement. Because enhanced antifungal activity has been demonstrated *in vitro* against combinations of amphotericin B plus azoles [3], we decided to include amphotericin B (125 mg of liposomal amphotericin B once a day) to the antifungal regimen on day 170 at our hospital.

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