

A report on pneumonia-induced ventriculitis with intraventricular abscess

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To the Editor: Ventriculitis, also known as ependymitis, ventricular empyema, pyocephalus, or pyogenic ventriculitis, is the inflammation of the ependymal lining of the cerebral ventricles. The inflammation of the ventricular drainage system is usually secondary to bacterial infection of the cerebrospinal fluid. The morbidity associated with ventriculitis has been reported in up to 45% of patients.^[1] Generally, neurosurgical patients or patients with head trauma are particularly at risk of developing ventriculitis, when these patients are treated with cerebrospinal fluid shunts or drains, implantation of intrathecal infusion pumps, or implantation of deep brain stimulation hardware.^[2] Clinically, most patients with ventriculitis present with fever, deterioration of the conscious level, and dyspnea.^[3] Delayed in the diagnosis and treatment will lead to severe intracranial infection, serious neurological impairment, and death. Most cases of ventriculitis have been reported in the newborns and infants, with bacteria being the most common pathogens, although several cases of ventriculitis secondary to *aspergillus fumigatus* infection have been described.

A 59-year-old man had several past medical comorbidities, including a 15-year history of hypertension, gout, chronic renal impairment, a four-year history of type-2 diabetes mellitus, a one-year history of thyroid carcinoma treated with surgery, and a three-month history of ischemic optic neuropathy managed with oral hormone therapy. He initially presented to a local hospital, complaining of a month history of fever, chills, and cough. The highest recorded body temperature was 39.0 °C. Physical examination revealed no significant findings. The blood investigation showed the followings: white blood cell (WBC) count $22.41 \times 10^9/L$, neutrophilic granulocyte percentage (N) 79.5%, hemoglobin (Hb) 43 g/L, platelet (Plt) $182 \times 10^9/L$, creatinine (Cr) 424 $\mu\text{mol/L}$, albumin (ALB) 2 g/L, procalcitonin (PCT) 0.3 ng/mL, negative blood and sputum culture. Computerized tomography (CT) of the thorax was performed, which showed a nodule in the upper lobe of the right lung, suggestive of an

inflammatory lesion. The patient was given a blood transfusion. He continued to have intermittent pyrexia despite 2 weeks of anti-inflammatory therapy with cefoperazone sulbactam. A chest CT [Figure 1A] was therefore repeated, which demonstrated that the previously seen nodule had transformed into a thick-walled cavitating lesion. The laboratory results showed that plasma (1-3)-beta-D-glucan >600 ng/L, galactomannan 0.64 $\mu\text{g/L}$, cluster of differentiation (CD) 4⁺/CD8⁺ ratio 0.55, and T spot-tuberculosis was negative. Given the findings above, the patient was thought to have a pulmonary fungal infection and was treated with voriconazole. However, he failed to respond following a week course of antifungal therapy that he continued to have pyrexia. The patient was then transferred to our hospital for further management. On clinical assessment, his temperature was 38.4 °C, blood pressure 144/73 mmHg, pulse rate 86 beats/min, and oxygen saturation 94% on room air. Laboratory results were as follows: WBC $11.02 \times 10^9/L$, N 88.9%, Hb 92 g/L, Plt $289 \times 10^9/L$, PCT 2.56 ng/mL, Cr 655 $\mu\text{mol/L}$, further blood and sputum cultures were negative. Upon treatment with piperacillin-tazobactam, his pyrexia had improved. The patient then developed a seizure 3 weeks after treatment in our hospital. The brain CT did not show any significant finding, but the plain and enhanced brain Magnetic Resonance Imaging (MRI) [Figure 1B-1C] was suggestive of ependymitis with intraventricular abscess.

A lumbar puncture was therefore performed, which showed that the cerebrospinal fluid pressure was 260 mmH₂O, cerebrospinal fluid was slightly muddy, the fluid analysis demonstrated WBC $950 \times 10^6/L$, red blood cell (RBC) count $100 \times 10^6/L$, protein 1.84 g/L, blood glucose 1.3 mmol/L, Cl 109 mmol/L. Though the culture of the fluid did not yield any growth, the High Throughput Sequencing of the cerebrospinal fluid showed evidence of *aspergillus fumigatus*. Following a month's course of voriconazole, the patient's pyrexia resolved. A repeated lumbar puncture was performed, which showed that the cerebrospinal fluid pressure was 190 mmH₂O with fluid analysis demonstrated WBC $0 \times 10^6/L$ and RBC $0 \times 10^6/L$.

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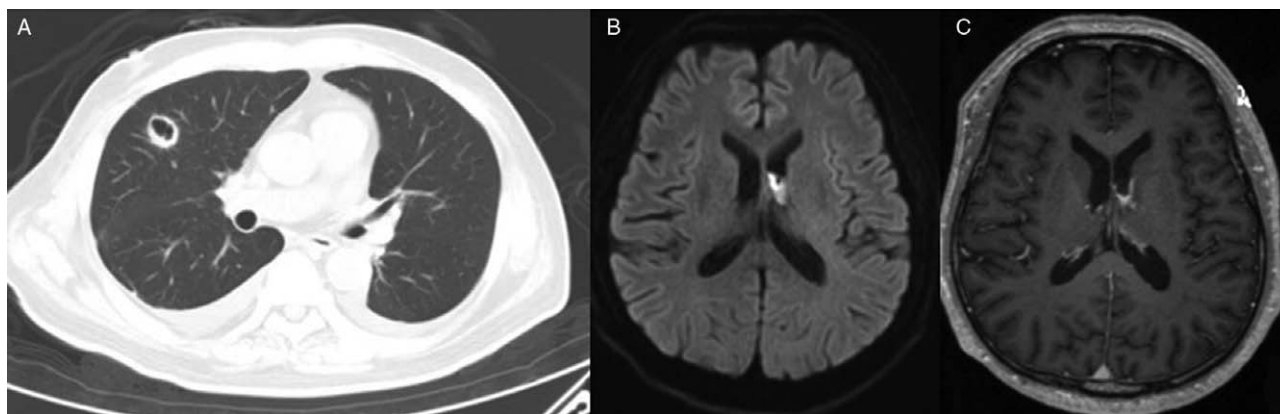


Figure 1: Chest computed tomography and brain Magnetic Resonance Imaging of the patient. (A) shows a thick-walled cavity in the upper lobe of the right lung. (B) is diffusion-weighted imaging which shows a patchy diffuse restricted lesion in the left ventricle. (C) is enhanced brain magnetic resonance imaging which shows partial marginal enhancement of the lesion.

The blood investigations showed a normal level of C-reactive protein (CRP), plasma (1-3)-beta-D-glucan, and galactomannan. The patient was subsequently transferred to the neurosurgical team, where a cranial operation was performed and this confirmed the presence of an abscess in the left lateral ventricle, but the culture did not yield any growth. Following the operation, the patient continued to be treated with voriconazole with added ceftriaxone and had no recurrence of sepsis since with resolved cavitating lung lesions.

Encephalic aspergillosis, which accounts for 5% of the infections of the central nervous system, is caused by aspergillosis invading into the central nervous system. Most cases of encephalic aspergillosis are secondary to the hematogenous spread of pulmonary aspergillosis, but a few have been reported to be directly spread from paranasal sinuses.^[4] *Aspergillus fumigatus* is the most common pathogen causing invasive aspergillosis, particularly in the immunosuppressed patients who are neutropenic or are treated with long-term glucocorticoid. It is associated with a higher mortality rate compared with other types of aspergillosis.^[5] Our patient was immunosuppressed, having had several significant chronic comorbidities and was on oral glucocorticoid therapy. He did not respond to the antifungal treatment well and continued to have pyrexia with the development of cavitating lesions later in the lung. The possibility of intracranial infection was only considered when he developed seizures, as he did not have other associated symptoms such as headache, vomiting, disturbance of consciousness, or nuchal rigidity. In this instance, the brain CT was not diagnostic, though the MRI was helpful and showed the abnormality that led to lumbar puncture being performed. The culture of blood, cerebrospinal fluid, and abscess from the ventricle had no diagnostic yield in this case. The diagnosis of *aspergillus fumigatus* was only confirmed following the High Throughput Sequencing of cerebrospinal fluid, and the fungus was most likely originated from the lung given the patient's initial presentation and the presence of cavitating lung lesion.

For immunosuppressed patients who present with sepsis and fail to respond to standard antimicrobial treatment, the possibility of occult and rare focus of infection should

be considered early. In this context, there should be a low threshold for serial clinical assessments, repeated investigations, and multidisciplinary involvements to allow early diagnosis and appropriate treatment instituted to improve clinical outcomes.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient/or his guardian has/have given his consent for his their images and other clinical information to be reported in the journal. The patient or his guardian understand that his and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

Conflicts of interest

None.

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