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Case Report

Fatal pulmonary disease and encephalic complication in a man with HSV-1 Infection: A case report

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ABSTRACT

Herpes simplex virus type 1 (HSV-1) is associated with a large spectrum of pathologies i.e. pulmonary diseases. Although it has often been isolated from the lower respiratory tract of immunocompetent or immunosuppressed patients undergoing prolonged mechanical ventilation (MV), its causative role in serious lung infections is still unclear. Here we report the case of a 44-year-old man presenting seizures that followed an acute respiratory illness that occurred during hospitalization. The detection of HSV-1 DNA in bronchoalveolar lavage (BAL), in spinal fluid, and in blood samples, supported the evidence of a disseminated viral infection that strengthens the hypothesis of herpetic pneumonia as a possible triggering cause of neurological complications and fatal outcome. This observation draws attention to the opportunity of introducing tests for the detection of HSV-1 into the diagnostic protocols for such patients. In fact, adequate diagnostic tools would favor early diagnosis and correct therapy to HSV-1 that could reduce the possibility of either encephalic complications or the rate of mortality in critical long-term patients affected by respiratory pathologies who need assisted ventilation.

1. Why this case is important

The pathologies of the lower respiratory tract are various and microorganisms are certainly one of their prime causes. The herpes simplex viruses, especially HSV-1, are rarely considered in the diagnostic protocols adopted in the management of intensive care patients suffering from serious respiratory diseases. This is probably because the pathogenetic role of these viruses in such pathologies has been considered poor and, thus, consolidated epidemiological studies are not available. HSV-1 is a ubiquitous human pathogen in adults [1]. Less common manifestations such as meningitis, encephalitis [2,3], hepatitis [4–6] and pneumonitis [7] can occur both in children and adults. After the first contact, the virus establishes a lifelong latent infection in the neuronal district, resulting in reactivation due to various triggering factors. Because of its capability of inducing persistence, it is unclear if HSV-1 is the primary cause of multi-organ failure or if its detection in clinical samples is due to its reactivation because of the deficit of the immune system or other underlying diseases; mechanical ventilation (MV) can be considered one of these triggering factors. To underline the relevant role of HSV-1 reinfection in critical patients, the case of a severe respiratory disease in an adult intensive care patient infected by HSV-1 is described. During hospitalization in the Intensive Care Unit (ICU), the virus was first detected in the bronchoalveolar lavage (BAL) and subsequently in cerebrospinal fluid (CSF). The patient died after 18 days.

2. Case report

2.1. Case description

A 44-year-old male was hospitalized in February 2012 in our Radiotherapy Unit because of rhinopharynx cancer. He was then referred to the ICU of the same hospital because of the sudden appearance of a severe respiratory disease and generalized seizures. Then the patient underwent prolonged MV, showed persisting fever (38–39 °C) and worsening symptoms. No

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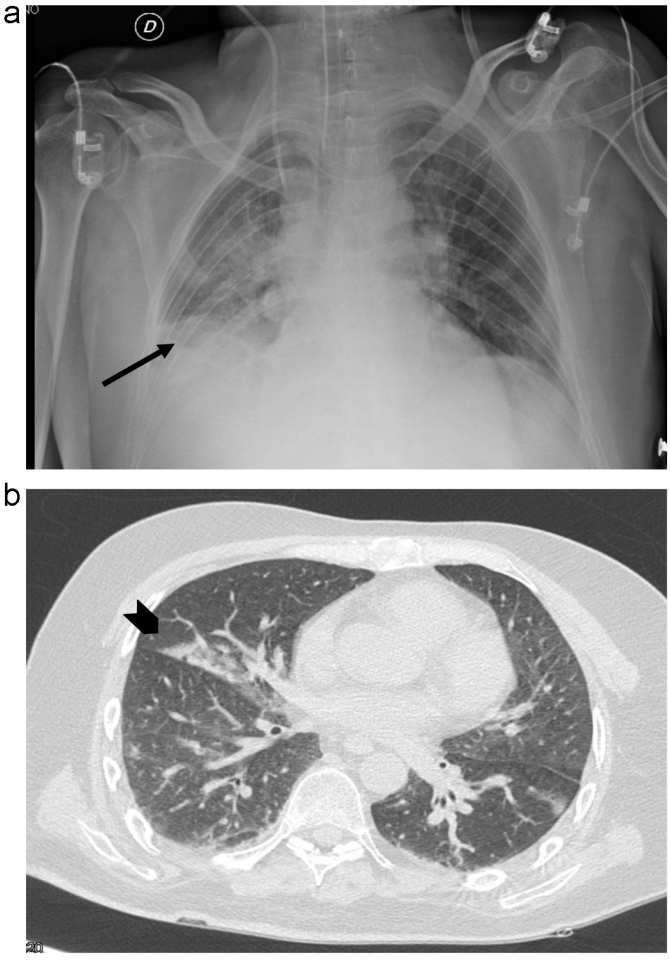

Fig. 1. (a) Chest RX shows a non-homogeneous right lung consolidation; a homolateral pleural effusion was also observed (arrow). (b) Ground-glass opacities and scattered areas of consolidation were found on the CT image (arrowhead).

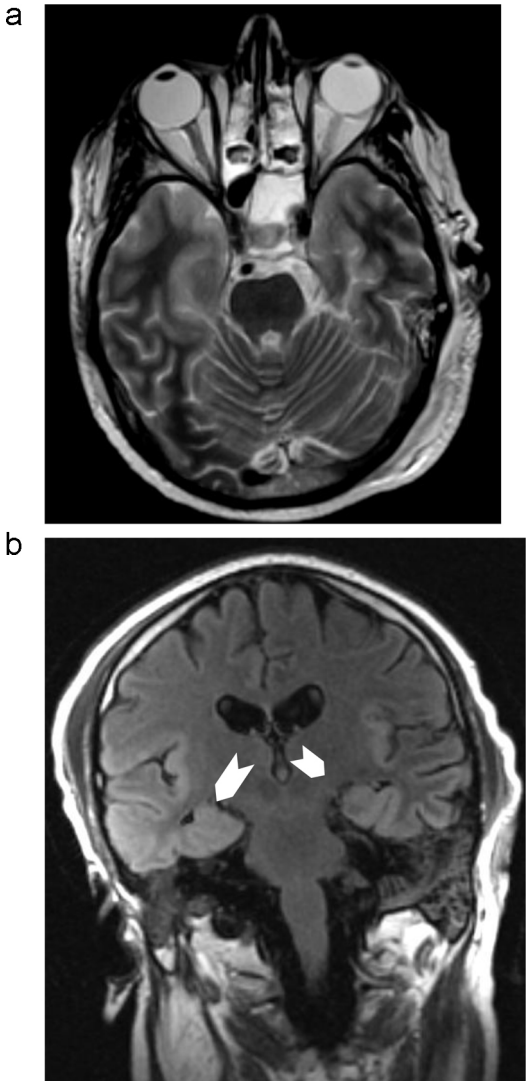

Fig. 2. Axial T2-weighted image (a) and coronal FLAIR sequence (b) show high signal intensity in the cortex of both temporal lobes (arrowheads on coronal image).

clinical macroscopic herpetic lesions were observed. An acute lower viral respiratory tract infection was diagnosed, but specific therapy was introduced when the positivity was revealed in the first spinal liquid sample. No previous significative respiratory or cardiovascular pathologies were reported. His clinical history revealed that he was symptomatic for HSV-1 infection with a frequency of about one episode/year. The blood cell count detected 12,600 white blood cells/mm^3 (93% neutrophils, 4% lymphocytes, 3% monocytes), 3,610,000 red blood cells/mm^3 and not relevant procalcitonin levels. Three chest radiographs within one week were performed. The patient had a non-homogeneous right lung consolidation on the first two; pleural effusion was also observed (Fig. 1a). A multidetector CT scan was performed to better investigate the involvement of the lung: ground-glass opacities and scattered areas of consolidation were found, without any imaging findings suggestive of a specific type of pulmonary infection (Fig. 1b). The third chest radiograph showed a slight improvement. Moreover, the patient was submitted to encephalic Magnetic Resonance (MR) that revealed various suffering areas such as the medial side of the temporal lobe, hippocampal zone, ethmoidal cavities and frontobasal brain; T2-weighted images and Flair (Fluid Attenuated Inversion Recovery) sequences showed a diffuse and symmetrical high signal intensity in the cortex of both temporal lobes (Fig. 2a and b). BAL and CSF specimens were collected as shown in Table 1. His general conditions worsened progressively until he died after 18 days of hospitalization. BAL

Table 1
Real-time PCR results to HSV-1 according to the type of specimen and timing.

	February 21	February 23	February 28	March 6
BAL fluid[d] (Ct/copies/ml)	[b]Positive (36/<500)	Not done	[b]Positive ($22/3 \times 10^6$)	Positive ($25/1 \times 10^6$)
Blood (Ct/copies/ml)	Positive (>40/<500)	Positive ($33/2 \times 10^3$)	Not done	Negative
[a]CSF (Ct/copies/ml)	Negative	[c]Positive ($29/2 \times 10^5$)	[c]Positive (36/<500)	Negative

BAL, Bronchoalveolar lavage.
[a] Cerebral spinal fluid obtained during a surgical procedure. The real-time PCR was performed on two separate aliquots processed independently.
[b] Respiratory viruses and other herpes viruses were negative.
[c] Encephalitic viruses were negative.
[d] Ct (cycle value) cutoff threshold: <35, quantitative result; >35, qualitative result; sensitivity, 10 copies/reaction.