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Nosocomial herpes simplex encephalitis: A challenging diagnosis

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Summary Herpes simplex encephalitis (HSE) is a rare disease, but it is the most common form of sporadic encephalitis. HSE is transmitted through direct contact and developing nosocomial HSE is rarely reported in the literature. Nosocomial HSE is difficult to diagnose due to its non-specific clinical features. In this article, we present a case of nosocomial HSE that was responsible for grave consequence. We also explore its causes, outcome, and give recommendations to avoid such fatal occurrence. We stress on strict adherence to the standard precautions and preventive control measures.

Introduction

Herpes simplex encephalitis (HSE) is the most common form of sporadic encephalitis with a global incidence of 1-4/1,000,000 population per year. The disease is challenging to diagnose and carries a bad prognosis with high morbidity and mortality rates if treatment was not given or delayed [1]. The disease can be caused by a primary herpes simplex virus type 1 (HSV-1) infection, reactivation of the latent virus in the sensory ganglia or brain parenchyma, or invasion of the central nervous system from the olfactory tract. Nosocomial HSE is extremely rare and can be responsible for grave consequences [2]. In this article, we report a case of nosocomial HSE and explore its causes.

Case report

A 55-year-old female patient, known case of multiple medical problems including diabetes,

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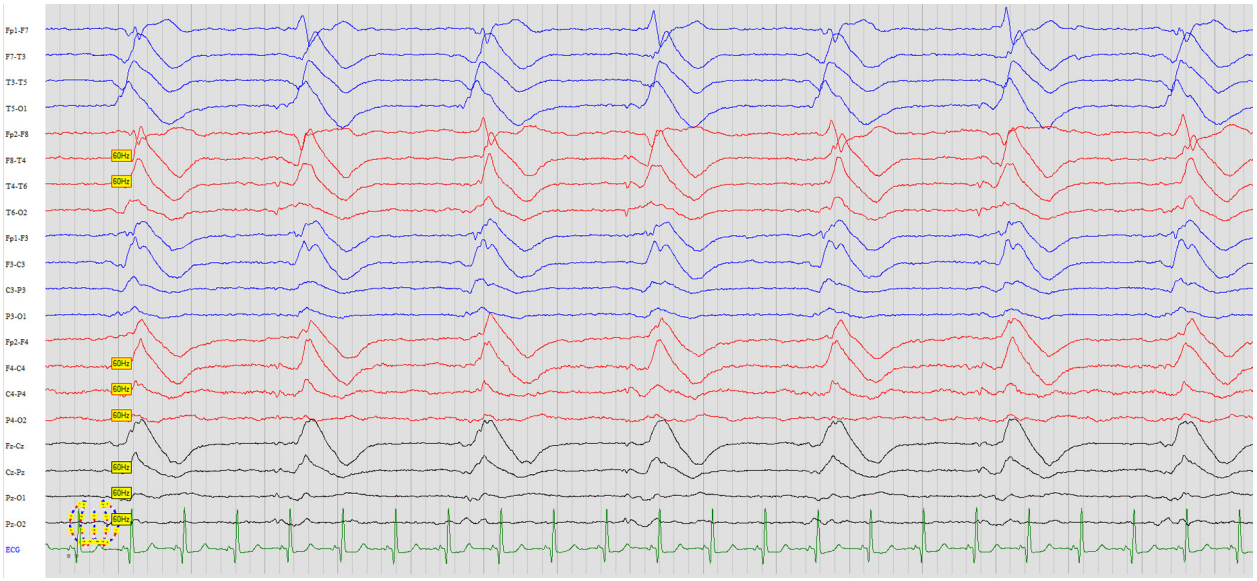

Figure 1 EEG showing classical triphasic waves with burst suppression pattern.

hypertension, hypothyroidism, and chronic kidney disease with baseline serum creatinine of 150 μmol/L, presented with fever, shortness of breath, and dysuria. She is also known case of bipolar disorder on anti-psychotic medications. She was admitted through the emergency room with urosepsis, septic shock, and congestive heart failure. She was resuscitated, started on broad spectrum antibiotics, and admitted to the intensive care unit (ICU). Despite aggressive management, she developed cardiac arrest and cardiopulmonary resuscitation (CPR) was initiated followed by intubation and mechanical ventilation. She improved both clinically and biochemically (acid-base balance and renal functions) over the course of five days with extubation. Two days later she was shifted to the ward with Glasgow Coma Scale (GCS) of 15/15 and normal mental status and neurological examination. Her cardiac functions normalized and repeated full septic screen came back negative. Four weeks later, she developed a tonic–clonic seizure with decreased level of consciousness and dropping in blood pressure. She was started on norepinephrine up to 7 mcg, loaded with phenytoin and continued on maintenance dose of phenytoin. Brain magnetic resonance imaging (MRI) was normal. The patient was started empirically on meropenem, vancomycin, colistin and anidulafungin. Several electroencephalograms (EEGs) were done, which initially showed a diffuse encephalopathic process, and eventually a burst suppression pattern was observed (Fig. 1). She was also started on lacosamide and levetiracetam. Lumbar puncture was done, and the patient was started empirically on intravenous acyclovir 10 mg/kg every 8 h. Cerebrospinal fluid (CSF) analysis revealed high opening pressure, lymphocytic pleocytosis, normal glucose, highly elevated protein, and positive HSV-1 PCR. A repeat MRI revealed high signal intensity on T2-weighted and fluid-attenuated inversion recovery (FLAIR) which were extensive involving temporal and frontal lobe and basal ganglia (Fig. 2). A repeat MRI showed extensive pseudolaminar necrosis with worsening of the previously observed changes of HSE (Fig. 3). Despite receiving three weeks of acyclovir treatment, unfortunately, the patient developed hospital-acquired pneumonia, septic shock and died 70 days post admission.

Discussion

Two distinct types of herpes simplex virus are recognized. In children and adults, the most common cause of sporadic encephalitis is herpes simplex virus type 1 (HSV-1). While in neonates, especially in the first month of life, the infection is caused by herpes simplex virus type 2 (HSV-2) [3]. Despite the fact that HSE is rare, one-third of the world population is infected with the herpes simplex virus. Primary infection typically occurs in the oropharyngeal mucosa of children and adolescents. Through retrograde axonal transport, the virus reaches the trigeminal ganglia where colonization is established. Encephalitis occurs by either extension from the olfactory tract or trigeminal ganglia into the parenchyma or through reactivation in the trigeminal ganglia with subsequent spread to the temporal and frontal lobe [1].

The onset of HSE is typically acute or subacute with a prodrome of upper respiratory or

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