Crimean-Congo Hemorrhagic Fever with Acute Subdural Hematoma, Mauritania, 2012

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To the Editor: Crimean-Congo hemorrhagic fever (CCHF) was first described in Crimea in 1944 and in the Congo in 1969. Since then, many cases in humans have been reported from different regions (1–3). The disease is transmitted to humans through the bite of an infected tick or by direct contact with blood or tissue from infected humans and livestock. We report an unusual case of acute subdural hematoma secondary to CCHF.

A 58-year-old man, a shepherd, was admitted to Centre Hospitalier National (Nouakchott, Mauritania) on July 2, 2012, with fever and epistaxis. One week earlier, he had fever, nausea, and vomiting. Without biologic confirmation of the infection, his doctors treated him for malaria. His leukocyte count was 3,200 cells/mm³ (reference range [RR] 4,000–10,000 cells/mm³), hemoglobin level was 10.6 g/dL (RR 14.0–17.5 g/dL), and platelet count was 22,000/mm³ (RR 200,000–400,000 cells/mm³). His aspartate aminotransferase level was elevated to 162 IU/L (RR 8–30 IU/L), and his alanine aminotransferase level was elevated to 162 IU/L (RR 8–30 IU/L), and his alanine aminotransferase level was elevated to 162 IU/L (RR 8–30 IU/L). His aspartate aminotransferase level was elevated to 162 IU/L (RR 8–30 IU/L), and his alanine aminotransferase level was elevated to 162 IU/L (RR 8–30 IU/L), and his alanine aminotransferase level was elevated to 162 IU/L (RR 8–30 IU/L). 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CCHF in which the patient died of complications following surgical intervention for cerebral hemorrhage (8).

Death from CCHF usually occurs after 5–14 days of illness (1,8,9). The basic pathogenesis of CCHF virus at the molecular level is complex and not well defined. Endothelial cells, immune response, virus load, and coagulation cascade play major roles in the disease pathogenesis. Blood and endothelium appear to be the target tissues of the disease (9). The coagulation cascade becomes activated over 24–48 hours; however, thrombin becomes activated and promotes edema formation and further disruption of the integrity of the blood–brain barrier. The edema formation starts when erythrocytes in the hematoma begin to lyse and its degradation products are deposited into the brain parenchyma, initiating a potent inflammatory reaction (10).

Although surgery remains the first choice for the treatment of acute subdural hematoma, some patients may benefit from conservative management with careful monitoring. This report highlights the value of an early diagnosis of CCHF and neuroimaging for severe cases when brain hemorrhage is suspected.

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Use of Plasma Therapy for Severe Fever with Thrombocytopenia Syndrome Encephalopathy

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To the Editor: The central nervous system (CNS) manifestations of severe fever with thrombocytopenia syndrome (SFTS) include apathy, seizure, muscular tremor, and coma (1,2); however, the mechanism underlying CNS manifestations in SFTS is not clear. Deng et al. reported that illness of 15 (13%) of 115 patients with SFTS met the case definition for suspected encephalitis (1). However, they did not present any straightforward evidence of CNS invasion by SFTS virus (SFTSV). Cui et al. similarly reported that encephalitis developed in one fifth of 538 patients with SFTS (2). They found evidence of SFTSV by isolating the virus from the cerebrospinal fluid (CSF) in 1 of 2 patients with SFTS whose CSF was obtained, but they did not mention CSF pleocytosis (2). We report a case of SFTS-associated encephalopathy, without pleocytosis and with normal CSF protein and glucose levels, that was confirmed by real-time reverse transcription PCR of the CSF. The patient was treated with experimental plasma exchange followed by convalescent plasma therapy.

During 2015, a 62-year-old woman who had a history of treated tuberculous meningitis 10 years earlier was admitted to a tertiary hospital in Seoul, South Korea (Republic of Korea), with a 5-day fever, myalgia, and headache. On hospital day (HD) 2, CSF examination revealed 1 leukocyte/mm³, protein 35 mg/dL (reference 9–58 mg/dL), glucose 74 mg/dL (reference 45–80 mg/dL), and CSF/blood glucose ratio 0.66 (reference 0.50–0.80). No bacteria or fungi were isolated from CSF. On HD 4, her headache worsened, and she displayed confused verbal responses and lacked orientation of time and place. No focal neurologic signs were observed. On HD 5, magnetic resonance imaging of the brain indicated no additional abnormalities of the parenchyma and extra-axial structures except for a focal parenchymal defect in the right midbrain that had been discovered as a

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