Case Report

Crimean-Congo hemorrhagic fever: An emerging threat for the intensivist

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Abstract

We present the case of a 55-year-old female, who presented with 15 days of fever with rash, pancytopenia, and altered behavior. She was investigated for routine causes of fever with rash and multi organ dysfunction and treated for the same. As she tested negative for all routine causes of such an illness and did not show improvement to therapy, she was investigated for Crimean-Congo hemorrhagic fever and tested positive for the same. She was started on ribavirin, but eventually succumbed to her illness. This disease has rarely been reported from the Northern India and we need to have high clinical suspicion for this deadly disease so that appropriate therapy can be started in time for the patient and prophylaxis given to all inadvertently exposed.

Keywords: Crimean-Congo hemorrhagic fever, hemorrhagic fever, pancytopenia

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Introduction

Crimean-Congo hemorrhagic fever (CCHF) virus is an enveloped RNA virus of the family Bunyaviridae (genus *Nairovirus*) and causes severe viral hemorrhagic fever. Although it is primarily an animal disease, sporadic cases and outbreaks of CCHF affecting, humans do occur. CCHF outbreaks constitute a threat to public health because of its epidemic potential, high case fatality, the potential for nosocomial outbreaks, and difficulties in treatment and prevention.

Case Report

A 55-year-old, previously healthy female with significant domestic cattle handling, hailing from a rural area around Moradabad, Uttar Pradesh, presented with 15 days history of high grade fever with chills. The patient was admitted to a local hospital near her village, where she was treated with broad spectrum antibiotics and antimalarials. During the course of the hospital stay, the patient developed progressively worsening pancytopenia and developed bleeding from the oral cavity, epistaxis and developed a fine red macular rash all over her body. Around the 14th day of her illness, she developed incoherent speech and breathing difficulty and was referred to our center. On presentation, the patient was febrile (39.4°C), dehydrated and had severe pallor. She was oriented to time, place, and person, but was very irritable and had lost bladder and bowel control. Investigations showed platelet count of 10,000/cmm, Hb of 4.8 g/dl, white blood cell 980/cmm, lactate dehydrogenase 34,000 IU/L, serum creatinine 2.4 mg/dl, blood urea 201 mg/dl, serum bilirubin 3.6 mg/dl (predominantly indirect fraction), Na 133 meq/l, K: 5.4 meq/l, prothrombin time 23 s, and the International Normalized Ratio of 3.4. The chest X-ray was suggestive of bilateral alveolar infiltrates and minimal bilateral pleural effusions and ultrasound abdomen revealed mild ascites with hepatosplenomegaly. Dengue, malaria, scrub typhus, and leptospira serology tested negative. The patient was treated with oxygen,

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noninvasive ventilation, intravenous (IV) fluids, and blood products and was started on IV ceftriaxone, artesunate, and oral doxycycline. As her clinical picture matched with the clinical presentation of CCHF and her condition deteriorated despite appropriate therapy, blood samples were sent to the National Centre for Disease Control, New Delhi for CCHF serology testing. She tested positive for CCHF disease specific genes by reverse transcription-polymerase chain reaction (RT-PCR). The patient was started on oral ribavirin 1000 mg every 6 h (via naso - gastric tube). Over the next 2 days, the patient developed worsening multi organ failure and succumbed to her illness. Around 20 hospital staff were inadvertently exposed to the patient, of which 16 had close contact with her or had handled blood and urine samples. All were started on ribavirin prophylaxis as per the recommended doses. The drug was poorly tolerated by most and had to be stopped prematurely as most could not tolerate the prescribed dose. Contact tracing was carried out, but thankfully none of the family members developed the disease. One of the hospital staff were she was admitted prior to our unit, developed a similar illness, which resolved with supportive management. We do not know about the CCHF seropositivity of that person.

Discussion

CCHF, is highly contagious and is associated with high mortality, ranging from 10% to 50%.[1] The virus which causes CCHF is a *Nairovirus*, which is transmitted by ixodid ticks in animals. Humans acquire the virus from direct contact with blood or other infected tissues from livestock having viremia, or they may become infected from a tick bite. The majority of cases have occurred in those involved with the livestock industry, such as veterinarians, agricultural, and slaughterhouse workers. CCHF can be transmitted from one infected human to another by contact with infectious blood or body fluids.[2]

Clinical and laboratory features of patients with CCHF mimic the most prevalent diseases, such as dengue, malaria, and scrub typhus which are commonly encountered in routine practice. As CCHF is usually not considered as the primary cause of fever with rash and pancytopenia in our patients, it is often diagnosed late. This leads to a higher mortality and exposes more health care workers to the disease. Classical clinical features include high grade fever with chills, headache, myalgia, generalized rash, and bleeding.[3] Over the next few days, the patient may experience neurological features, such as mood swings, and may become agitated. After 2–4 days, the agitation may be replaced by mental obtundation and altered sensorium.

Laboratory diagnosis of a patient with a clinical history compatible with CCHF can be made during the acute phase of the disease by using the combination of detection of the viral antigen (ELISA antigen capture), viral RNA sequence (RT-PCR) in the blood or in tissues collected from a fatal case and virus isolation. Later in the course of the disease, in people surviving, antibodies can be found in the blood.[3] General supportive therapy and maintenance of vital parameters is the mainstay of patient management in CCHF. Blood component replacement and hydration are usually required. Ribavirin has been used in the treatment of established CCHF infection with apparent benefit.[4] Both oral and IV formulations seem to be effective.

Patients with suspected or confirmed CCHF should be isolated and cared for using barrier nursing techniques. Specimens of blood or tissues taken for diagnostic purposes should be handled using universal precautions. Hospital equipment and body wastes should be safely disposed of using appropriate decontamination procedures.

If strict precautions are not taken, there is a risk of nosocomial spread of infection and it is imperative that adequate infection control measures be observed to contain the infection. Healthcare workers who have had contact with tissue or blood from patients with suspected or confirmed CCHF should be followed up with daily temperature and symptom monitoring for at least 14 days after the putative exposure.

Outbreaks of CCHF have been reported from India and other regions of the South East Asia earlier[5] and have been associated with high mortality. Most of these cases, like our patient, were diagnosed late, as CCHF was not initially considered as a differential diagnosis. CCHF should be considered in patients with compatible clinical and laboratory features, so that early ribavirin therapy may be started, and appropriate control measures are taken to prevent nosocomial and community spread of the disease.

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Conflicts of interest

There are no conflicts of interest.

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