International Journal of Recent Advances in Multidisciplinary Topics
Volume 7, Issue 9, September 2026
www.ijramt.com | E-ISSN: 2582-7839 | RESAIM Publishing | www.resaim.com

Crimean-Congo Hemorrhagic Fever Without Hemorrhagic Manifestations: A Case Report

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Abstract*: Background: Crimean-Congo hemorrhagic fever (CCHF) is a potentially life-threatening viral infection caused by Crimean-Congo hemorrhagic fever virus (CCHFV). The virus is primarily transmitted through the bite of infected Hyalomma ticks or through contact with the blood or tissues of infected animals, particularly during slaughter. Human-to-human transmission may also occur following exposure to the blood or other body fluids of infected individuals. Although severe CCHF may be associated with hemorrhagic manifestations, disseminated intravascular coagulation, multiorgan failure, and death, overt bleeding is not an obligatory feature of the disease. Case Presentation: A 32-year-old man presented with a 2-day history of high-grade fever, photophobia, and neck pain associated with watery diarrhea. He worked in a city abattoir with regular occupational exposure to sheep and cattle and had not traveled outside the United Arab Emirates during the preceding year. On examination, he was hemodynamically stable and had a maculopapular rash over the trunk. His Glasgow Coma Scale score was 15/15. There was no neck rigidity, lymphadenopathy, jaundice, or clinical evidence of bleeding. Initial laboratory investigations revealed leukopenia, marked thrombocytopenia, and mildly elevated liver transaminases. His platelet count continued to decline during hospitalization. Given the epidemiological exposure and the combination of acute febrile illness and progressive thrombocytopenia, CCHF was suspected. Both CCHF serology and reverse-transcription polymerase chain reaction (RT-PCR) were positive, confirming the diagnosis. The patient received supportive treatment and oral ribavirin. His clinical condition and hematological parameters subsequently improved, and he was discharged in stable condition without developing hemorrhagic manifestations. Conclusion: CCHF should be considered in patients presenting with an acute febrile illness and thrombocytopenia, particularly when there is a history of occupational exposure to livestock or potential exposure to infected ticks. The absence of overt hemorrhage does not exclude the diagnosis. Early recognition, laboratory confirmation, appropriate infection-control measures, and timely supportive management are essential for optimizing outcomes and reducing the risk of secondary transmission.

***Keywords*: Crimean-Congo hemorrhagic fever, Crimean-Congo hemorrhagic fever virus, thrombocytopenia, RT-PCR, ribavirin, livestock exposure, Hyalomma, viral hemorrhagic fever.**

1. Background

Viral hemorrhagic fevers (VHFs) comprise a group of febrile illnesses caused by viruses belonging to several different viral families. These include filoviruses, such as Ebola and Marburg viruses; arenaviruses, such as Lassa virus and New World arenaviruses; nairoviruses, including Crimean-Congo hemorrhagic fever virus; and flaviviruses, including yellow fever virus [1].

Crimean-Congo hemorrhagic fever (CCHF) is a widespread zoonotic viral disease caused by CCHFV, an enveloped, negative-sense RNA virus belonging to the genus *Orthonairovirus* in the family *Nairoviridae*. The virus is primarily transmitted to humans by infected *Hyalomma* ticks. Infection can also occur through direct contact with the blood or tissues of infected animals, particularly during and immediately after slaughter. Consequently, abattoir workers, farmers, veterinarians, and other individuals with occupational exposure to livestock are at increased risk [2]-[5].

The disease was first recognized in the Crimean Peninsula in 1944, when an outbreak occurred among Soviet soldiers and was initially described as Crimean hemorrhagic fever. In 1956, a similar virus was isolated from a patient in the Belgian Congo. Subsequent investigations demonstrated that the two viruses were identical, leading to the current designation, Crimean-Congo hemorrhagic fever virus [3], [5].

The clinical spectrum of CCHF is broad, ranging from asymptomatic or mild infection to severe disease characterized by hemorrhage, shock, multiorgan failure, and death [6]. The disease usually begins abruptly with fever, headache, myalgia, dizziness, neck pain, photophobia, gastrointestinal symptoms, and generalized malaise. Nausea, vomiting, diarrhea, abdominal pain, conjunctival injection, and pharyngitis may also occur [7]-[9].

The early prehemorrhagic phase is often nonspecific and may resemble other infectious diseases. In severe cases, hemorrhagic manifestations may subsequently develop, including petechiae, ecchymoses, epistaxis, gingival bleeding, hematemesis, hemoptysis, melena, hematuria, and other mucosal or gastrointestinal bleeding. Severe disease may be associated with hepatic dysfunction, renal impairment, disseminated intravascular coagulation (DIC), acute respiratory distress syndrome (ARDS), circulatory shock, multiorgan failure, and death [10], [11].

The pathogenesis of severe CCHF is complex and involves endothelial dysfunction, dysregulation of the host immune

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response, and abnormalities of coagulation and vascular permeability [10], [11]. Importantly, however, clinically apparent hemorrhage is not present in every patient. Therefore, the absence of bleeding should not exclude CCHF in a patient with a compatible epidemiological history and characteristic laboratory abnormalities.

Laboratory diagnosis can be established by detection of CCHFV RNA using reverse-transcription polymerase chain reaction (RT-PCR), detection of specific antibodies using enzyme-linked immunosorbent assay (ELISA), antigen detection, or virus isolation [12]. During the early phase of illness, RT-PCR may be particularly useful because detectable antibody responses may not yet have developed. Laboratory specimens from suspected CCHF cases require appropriate biosafety precautions because of the risk of laboratory-associated infection.

There is currently no universally approved specific antiviral therapy for CCHF. Management is primarily supportive and includes careful monitoring of hemodynamic status, renal function, oxygenation, hematological parameters, and coagulation abnormalities, with appropriate replacement of blood components when clinically indicated [13]. Ribavirin has been used off-label for the treatment of CCHF, particularly in some endemic countries; however, evidence regarding its clinical efficacy remains controversial, and randomized clinical trial evidence is lacking [13]-[15]. Current WHO guidance continues to emphasize early intensive supportive care, while acknowledging uncertainty regarding the efficacy of ribavirin.[16]

We report a case of laboratory-confirmed CCHF in a young adult with significant occupational exposure to livestock who presented with fever, photophobia, neck pain, diarrhea, and progressive thrombocytopenia but did not develop clinically apparent hemorrhagic manifestations. This case emphasizes that CCHF should remain an important differential diagnosis in patients with compatible epidemiological exposure and laboratory findings even in the absence of overt bleeding.

2. Case Presentation

A 32-year-old man was admitted to the medical ward with a 2-day history of high-grade fever and photophobia associated with neck pain. His illness was also accompanied by watery diarrhea. He denied abdominal pain. He worked at a city abattoir and had regular occupational exposure to sheep and cattle. He had not traveled outside the United Arab Emirates during the preceding year.

Because of the combination of fever, photophobia, and neck pain, the patient was initially admitted with a provisional diagnosis of viral meningitis.

On physical examination, the patient was hemodynamically stable, with a Glasgow Coma Scale score of 15/15. A maculopapular rash was noted over the trunk. There was no neck rigidity, lymphadenopathy, or jaundice. Abdominal examination revealed a soft, non-tender abdomen. Respiratory and cardiovascular examinations were unremarkable. There was no clinical evidence of bleeding from the skin, mucous membranes, gastrointestinal tract, urinary tract, or other sites.

Initial laboratory investigations demonstrated leukopenia, marked thrombocytopenia, and mildly elevated serum liver transaminases. During hospitalization, the platelet count continued to decline. Despite the marked thrombocytopenia, the patient did not develop petechiae, ecchymoses, epistaxis, gingival bleeding, gastrointestinal bleeding, hematuria, or other clinically apparent hemorrhagic manifestations.

In view of the patient's occupational exposure to livestock and the combination of acute febrile illness, leukopenia, progressive thrombocytopenia, and elevated liver enzymes, a viral hemorrhagic fever was considered. CCHF was included prominently in the differential diagnosis because of the epidemiological setting.

CCHF testing was subsequently performed. Both CCHF-specific serology and RT-PCR were positive, confirming acute CCHFV infection.

The patient was managed with supportive care, including close clinical and laboratory monitoring. The decision to administer ribavirin was considered in the context of the available evidence and local clinical practice. Although the efficacy of ribavirin in CCHF remains uncertain, it has been used in endemic settings, including Turkey and other countries in the region [13], [14]. Following discussion of the potential benefits and limitations of treatment, oral ribavirin was initiated according to the recommended regimen used at the treating institution.

The patient subsequently demonstrated progressive clinical improvement accompanied by recovery of his hematological parameters. No hemorrhagic manifestations developed during the hospital stay. He was discharged home in stable clinical condition.

3. Diagnostic Assessment

The initial differential diagnosis included viral meningitis and other acute infectious causes of fever with thrombocytopenia.

The combination of progressive thrombocytopenia, leukopenia, mild transaminitis, occupational exposure to livestock, and the epidemiological setting raised clinical suspicion for CCHF. Laboratory confirmation was obtained by positive CCHF serology and positive RT-PCR for CCHFV.

The absence of overt bleeding did not exclude the diagnosis. In this case, the diagnosis was established during the early clinical course, before the development of hemorrhagic manifestations.

4. Therapeutic Intervention

The patient was managed with supportive care and close clinical and laboratory monitoring.

Because of the confirmed diagnosis of CCHF, appropriate infection-prevention and control precautions were implemented to minimize the risk of healthcare-associated transmission.

Oral ribavirin was initiated according to the treatment regimen used at the treating institution. The patient subsequently demonstrated clinical and hematological improvement.

Because ribavirin efficacy in CCHF remains uncertain, the

patient's improvement should not be interpreted as evidence of treatment efficacy. Rather, the clinical outcome occurred in the setting of early diagnosis, supportive care, and administration of ribavirin.

5. Clinical Course and Outcome

During hospitalization, the patient's platelet count initially continued to decline. Despite severe thrombocytopenia, he did not develop clinically apparent hemorrhagic manifestations.

Following initiation of supportive management and oral ribavirin, his general clinical condition improved and his hematological parameters progressively recovered. He remained hemodynamically stable and did not develop shock, DIC, ARDS, renal failure, or other documented major complications.

The patient was discharged home in stable condition.

6. Discussion

CCHF is an important zoonotic viral disease with a wide geographical distribution across Africa, the Middle East, Asia, the Balkans, and southeastern Europe. The virus is primarily transmitted by infected *Hyalomma* ticks, while direct exposure to infected animal blood or tissues represents another important route of transmission. Slaughterhouse workers are therefore considered an important occupational risk group [2]-[5], [16], [17].

Human infection may occur following a tick bite or crushing of an infected tick against exposed skin. Transmission may also occur through percutaneous or mucosal exposure to infected animal blood or tissues, particularly during slaughter. Human-to-human transmission has been well documented, particularly in healthcare settings following exposure to blood or body fluids of infected patients [18].

The incubation period varies according to the route of infection. Following a tick bite, symptoms generally develop within a few days, whereas infection following exposure to infected blood or tissues may have a somewhat longer incubation period [16].

The clinical course of CCHF is traditionally described as an incubation period followed by a prehemorrhagic phase, a hemorrhagic phase in severe cases, and a convalescent phase in survivors. The initial symptoms are nonspecific and may include fever, headache, myalgia, dizziness, neck pain, photophobia, nausea, vomiting, diarrhea, and abdominal pain [16].

Thrombocytopenia and leukopenia are common laboratory abnormalities and may provide an important diagnostic clue, particularly when accompanied by elevated hepatic transaminases. However, these findings are not specific to CCHF and must be interpreted in the context of the patient's epidemiological exposure and clinical presentation.

The present case is notable because the patient had laboratory-confirmed CCHF despite the absence of overt hemorrhagic manifestations. The initial presentation with fever, photophobia, neck pain, and diarrhea was nonspecific and led to an initial consideration of viral meningitis. The subsequent recognition of severe and progressive thrombocytopenia, leukopenia, mild transaminitis, and occupational exposure to livestock prompted consideration of CCHF.

This case therefore illustrates an important diagnostic principle: the term "hemorrhagic fever" should not lead clinicians to require visible bleeding before considering CCHF. Hemorrhagic manifestations are more characteristic of severe disease, but their absence does not exclude infection. WHO clinical descriptions similarly recognize that CCHF may initially present with nonspecific febrile and gastrointestinal symptoms before hemorrhagic manifestations develop in severe cases [16].

CCHF is also important from an infection-control perspective. Early recognition is essential not only for appropriate patient management but also for preventing healthcare-associated transmission. Strict infection-prevention and control measures, appropriate isolation, personal protective equipment, and safe handling of blood and other body fluids are essential when CCHF is suspected [16].

CCHF is not commonly recognized as an endemic disease in the United Arab Emirates; however, historical cases and outbreaks have been reported in the region. A notable outbreak occurred in Dubai in 1979, with secondary cases among healthcare workers [19]. CCHF has also been reported in neighboring and regional countries, including Oman, Saudi Arabia, Kuwait, Iran, Pakistan, India, and Iraq [17], [20].

The treatment of CCHF remains primarily supportive. Early recognition and intensive supportive care, including appropriate fluid management, monitoring of organ function, and replacement of blood components when clinically indicated, are important components of management [13], [16].

The role of ribavirin remains controversial. Earlier observational studies, particularly from Turkey, suggested potential benefit, while experimental and clinical evidence has remained inconsistent [14], [15]. WHO currently states that there is considerable uncertainty regarding the efficacy of ribavirin because of limited clinical evidence [16].

In the present case, ribavirin was administered after discussion of its potential role, in conjunction with supportive care. The patient subsequently improved clinically and hematologically. However, because this is a single case without a comparator, improvement cannot establish the efficacy of ribavirin. The favorable outcome is more appropriately interpreted as an example of recovery following early diagnosis and supportive management with ribavirin administered as an adjunctive treatment.

The principal lesson from this case is that clinicians should maintain a high index of suspicion for CCHF when confronted with acute febrile illness and unexplained thrombocytopenia in a patient with occupational exposure to livestock, even when hemorrhagic manifestations are absent.

7. Conclusion

CCHF should be considered in patients presenting with acute high-grade fever and thrombocytopenia, particularly when there is a history of occupational exposure to livestock or possible exposure to *Hyalomma* ticks.

The absence of overt hemorrhagic manifestations does not exclude CCHF. Early recognition based on the combination of clinical features, epidemiological exposure, and characteristic laboratory abnormalities can facilitate timely laboratory confirmation and appropriate infection-control measures.

Supportive care remains the cornerstone of management. Although ribavirin was administered in this case and the patient subsequently improved, the efficacy of ribavirin remains uncertain and should not be inferred from the outcome of an individual case.

Early diagnosis and appropriate infection-prevention measures are also essential to reduce the risk of healthcare-associated transmission.

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