

# A Case of Crimean Congo Hemorrhagic Fever Complicated With Pararenal Abscess

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## Abstract

**Introduction:** Crimean-congo hemorrhagic fever (CCHF) is a tick-borne zoonotic disease caused by Nairovirus of the Bunyaviridae family. It affects multiple systems and case fatality rate may vary between 5% - 30%. It also causes several complications. Here we presented a pararenal abscess case complicating CCHF, never reported before.

**Case Presentation:** A 49-year-old male dealing with livestock husbandry was admitted to Ankara Training and Research Hospital with three days of complaints including fever, malaise, headache, myalgia, nausea, vomiting, diarrhoea and hematuria. Laboratory findings (thrombocytopenia and elevated alanine transaminase (ALT) and aspartate transaminase (AST)) were suggesting CCHF and he was hospitalized. The diagnosis was confirmed by CCHF reverse transcriptase-polymerase chain reaction (RT-PCR) and IgM positivity. Although his clinical condition was improving, he developed right flank pain on the fourth day. The computerized tomography showed abscess (pararenal 44 mm × 17 mm). With appropriate antibiotherapy and percutaneous drainage, his clinical and laboratory findings regressed.

**Conclusions:** Unexpected complications may be seen during the course of CCHF. Patients must be closely followed up.

**Keywords:** Abscess, Crimean-Congo, Complications, Hemorrhagic Fever Virus

## 1. Introduction

Crimean-congo hemorrhagic fever (CCHF) is a tick-borne zoonotic disease caused by the Nairovirus of the Bunyaviridae family. After its first description in Crimea in 1944, because of similarities to the causative agent isolated at Congo, it was renamed as CCHF. CCHF has been reported in more than 30 countries in parts of Africa, Eurasia and the Middle East. Turkey has experienced the largest epidemic since 2002. The virus is mainly transmitted to humans via a bite from the Hyalomma genus ticks or by direct contact with blood or tissues of infected humans or viremic animals and causes severe diseases in human beings, with a reported mortality rate of 3 - 30% (1-3). A wide range of animals such as sheep, cattle, goat, ostrich, and many wild animals may become viremic, but without the apparent sickness; they also play an important role in disease transmission. In endemic regions, people living in rural areas and involved in livestock, animal husbandry, veterinarians, slaughterhouse workers, picnickers, soldiers, and also healthcare workers are at risk. The incubation period varies between 1 - 9 days, but may be longer depending on the transmission way and the amount of

viruses (4, 5). The clinical features are characterized by sudden-onset fever, myalgia, headache, fatigue, nausea, vomiting, and diarrhoea. Hematological findings may change from thrombocytopenia and petechial rash to massive bleedings. Several complications like visceral bleedings, infections, compartment syndrome and even hypertension may accompany the disease (6-8).

We had a CCHF case complicated with pararenal abscess, which had not been reported as a complication of CCHF before. Our aim was to draw attention that unexpected complications may develop during the course of CCHF.

## 2. Case Presentation

A 49-year-old male dealing with livestock husbandry from a village of the north-eastern part of Central Anatolia admitted to Ankara Training and Research Hospital in Ankara, Turkey, with complaints of fever and fatigue in 2011. His complaints had started three days before the admission, with fever, malaise, headache, myalgia, nausea, vomiting, diarrhoea and hematuria. He had no disease or drug history. At the initial examination, his

general condition was average, body temperature was 38°C, blood pressure was 100/60 mmHg, heart rate was 100 beats/minute, and examination of the abdomen revealed a tick on his skin. The tick was removed. The laboratory analysis showed the following: serum white blood cell (WBC) count: 4500 mm<sup>3</sup> (N: 4.800 - 10.800/mm<sup>3</sup>), haemoglobin: 11.5 g/dL (N: 13 - 17 g/dL), thrombocyte count: 23.000 mm<sup>3</sup> (N: 150000 - 400000/mm<sup>3</sup>), erythrocyte sedimentation rate: 33 mm/h (N:0 - 20 mm/hours), C reactive protein (CRP): 1.85 mg/dL (N:0 - 5 mg/dL), serum aspartate aminotransferase (AST): 165 IU/L (N:10 - 38 IU/L), alanine aminotransferase (ALT): 231 U/L (N:10 - 41 U/L), lactate dehydrogenase (LDH): 760 U/L (N:240 - 480 U/L), creatinine phosphokinase (CPK): 764 U/L (N: 20 - 200 U/L), activated partial thromboplastin time (aPTT): 42 seconds (N: 26 - 35 seconds). He showed hematuria in urine analysis. He lived in an endemic region; all the clinical and laboratory findings were indicative of CCHF. He was hospitalized and his serum samples were sent to a reference laboratory (Virology Reference and Research Laboratory of Public Health Institute of Turkey) for CCHF IgM, IgG and real time polymerase chain reaction (RT-PCR) tests. On the second day of admission, his diagnosis was confirmed by CCHF RT-PCR and IgM positivity. He was treated with oral ribavirin (initially 2 g, then 4 × 1 g/day for four days and 4 × 500 mg for six days) and platelet transfusions. Complete blood count and biochemical tests were performed on a daily basis. All the biochemical analyses

were performed by auto-analyser and complete blood counts were performed by automatic hemocounter at central laboratory of our hospital. As his clinical and laboratory findings related to CCHF were improving, he complained about right flank pain on the fourth day and fever again. His peripheral white blood cell count was 10.200/mm<sup>3</sup> with polymorphonuclear cell dominance. His abdomen ultrasonography revealed hyper-echogenicity in the right pararenal space; abscess or hematoma could not be differentiated; so, abdominal computed tomography (CT) was requested. The CT showed right pararenal abscess (44 mm × 17 mm). Ceftriaxone and metronidazole treatment were initiated and he was consulted by an urologist. Other causes of pararenal abscess were investigated as well. There was growth neither in blood cultures taken during the fever period nor in urine culture. Ten days after the admission, his biochemical tests and thrombocyte levels were normal (Table 1), but his temperature was still high despite being under antibiotherapy; so, CT was repeated and showed that the abscess size had increased (pararenal 195 mm × 150 mm). Percutaneous drainage was performed and approximately 300 mL of purulent material was drained. No microorganism was detected with Gram staining and there was no bacterial growth on the culture of pus material. We continued the antibiotic therapy. The abscess disappeared completely and the patient was discharged in a healthy condition after 40 days of hospitalization.

**Table 1.** Main Laboratory Findings of Patient According to Days

| Tests                                    | First Day (Admission) | Fourth Day (Abscess Detection) | 10th Day (Abscess Drainage) | 30th Day (Discharge) | N               |
|--|-----------------------|--------------------------------|-----------------------------|----------------------|-----------------|
| White Blood Cells/mm <sup>3</sup>        | 4500                  | 10200                          | 16000                       | 6400                 | (4000 - 10000)  |
| Thrombocytes/mm <sup>3</sup>             | 23000                 | 90000                          | 206000                      | 250000               | (150000-400000) |
| Alanine Aminotransferase, IU/L           | 231                   | 90                             | 36                          | 30                   | (10 - 41)       |
| Aspartate Aminotransferase, IU/L         | 165                   | 104                            | 74                          | 35                   | (10 - 38)       |
| Creatinine Phosphokinase, U/L            | 764                   | 146                            | 146                         | 150                  | (10 - 200)      |
| Lactate Dehydrogenase, U/L               | 760                   | 447                            | 299                         | 256                  | (240 - 480)     |
| Activated Partial Thromboplastin Time, s | 42                    | 32                             | 25                          | 25                   | (26 - 35)       |

### 3. Discussion

CCHF is an acute, infectious illness that can cause multiorgan failure and death. The first cases of CCHF was described in Tokat (a city in the Black Sea Region of Turkey); since then, a great epidemic is continued in some parts of Turkey (2, 9). Although there are some other transmission ways, CCHF occurs most frequently among agricultural workers through the bite of an infected tick. Our case was also in an endemic region and he was a husbandman. He had not noticed tick on his abdomen. His complaints were fever, myalgia, headache, fatigue, nausea, vomiting, and diarrhoea and were compatible with CCHF. He did not have any illness or drug story. With his typical epidemiological history as well as clinical laboratory findings (thrombocytopenia, elevated ALT, AST, CK, LDH, aPTT), we did not have any difficulty to make presumptive diagnosis (1, 5). The CCHF diagnosis of the patient was confirmed by PCR and serology tests. Supportive treatment and ribavirin were given to the patient, as described in the literature (3, 5, 10). There are four periods in the course of typical CCHF cases: incubation, prehemorrhagic, hemorrhagic, and convalescence. The incubation period may vary between 2 - 9 days depending on the contact type. The prehemorrhagic period is about one week and symptoms are nonspecific and may mimic several infectious and noninfectious diseases. The hemorrhagic period generally develops at 3 - 5 days from the disease onset (5). Hemorrhagic manifestations, changing from epistaxis to several visceral bleeding, may be seen in 48% of patients (9). Thrombocytopenia, increased aPTT and hematuria were detected as hematological manifestations in this case. All the hematological findings subsided on the fourth day, but fever rose again and we detected pararenal abscess.

The main complications of CCHF are hematological complications. Most of the deaths are results of massive hemorrhagia, disseminated intravascular coagulation, and shock (3). Some other complications like hemophagocytosis and infections may be seen during the course of the diseases (11). The convalescence period begins 10 days after the onset of symptoms and may be longer. Although recovery is complete in CCHF, the long-term effect of the disease is not very well known. As the cases have increased around the world, some miscellaneous complications of CCHF are being reported. Compartment syndrome and orchitis are among some of the complications of CCHF (6, 12). It can be complicated in many different ways such as acalculous cholecystitis and intraabdominal abscess according to a previous report (13). Furthermore, it is reported that clinical conditions simulating acute appendicitis can be seen in the course of CCHF (14). We detected pararenal abscess in the patient with CCHF. This is an unexpected complication and was not previously reported.

The pathogenesis of CCHF is still not completely understood. Endothelium and mononuclear phagocyte system cells are the virus's main target (3, 15). Endothelial invasion and vascular endothelial damage are the main pathologi-

cal findings. Proinflammatory cytokines, chemokines and other mediators released from infected cells play major roles in the pathogenesis of CCHF and may lead to vascular permeability changes, systemic inflammation, and widespread intravessel coagulation (15-17). As with Ebola hemorrhagic fever, the suppression of type 1 interferon response and the attack of immune cells like dendritic cells and macrophages, as well as inefficient initial immune response may contribute to the pathogenesis (18). Diffuse endothelial damage, thrombocytopenia, immunosuppression, and coagulation dysfunction may predispose to complications seen during the course of the disease.

Perirenal abscesses are located in the perinephric space between the renal capsule and Gerota's fascia. They commonly occur either as a result of the rupture of an adjacent renal abscess or the hematogenous spread of infection. Although *Staphylococcus aureus* is the most common pathogen of the hematogenous infection, Gram-negative microorganisms are the most common cause of pararenal abscesses resulting from adjacent ruptured renal abscesses (19). In this case, we could not find any focus leading to pararenal abscess. All the cultures obtained from the patients were negative. This was the weakness of this case report. Because of the nosocomial transmission of CCHF, routine abdomen ultrasonography was not a part of our initial patient's examination. For this reason, we did not have a knowledge whether a hematoma was there or not at the admission. This was the second weakness of our case report. A hematoma might be developed initially due to thrombocytopenia and/or vascular damage; then, bacteria might settle there; thus, abscess might exist. Beside these, mucosal and endothelial damage might lead to bacterial translocation. The disability of the immune system is also likelihood at the development of infections. When we consider all these regarding the pathogenesis of CCHF and absence of any underlying disease, it strongly suggests that the development of pararenal abscess in our patient was a complication of CCHF. Thinking of the probable pathogens, we started antibiotics empirically and gave ceftriaxone and metronidazole to the patient parenterally. We were not able to detect the bacterial agent after the drainage of abscess because of antibiotic usage.

In conclusion, miscellaneous and unexpected complications may occur in patients with CCHF during the course of disease, even after the clinical findings due to diseases are subsided. For this reason, physicians must be aware of the possibility of encountering to unexpected complications and patients with CCHF must be followed up closely. Each distinct complaint must be evaluated carefully.

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## Footnote

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