# Nosocomial transmission of Crimean-Congo hemorrhagic fever in a health care worker, Fars Province, Iran

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### ABSTRACT

**Background**: Crimean-Congo hemorrhagic fever (CCHF) virus causes a severe hemorrhagic syndrome in humans with fatality rate up to 50%. Its transmission to humans is through the bite of Ixodid ticks or by contact with blood or tissues from infected livestock.

**Patient**: By a nosocomial transmission of Crimean-Congo hemorrhagic fever (CCHF), a health care worker was infected in December 2008 due to a re-emerging outbreak of CCHF in Fars province, Iran. After admission of probable CCHF cases in a local hospital, one of the nurses contributed in taking care of the patients was infected with CCHF, though it seems that she had not had direct contact with blood and secretions of CCHF patients. The laboratory detected anti-CCHF virus IgM antibody through specific ELISA and also the CCHF virus genome in her serum by real-time and gelbased RT-PCR. She was improved by an alert and on time clinical diagnosis and treatment.

Conclusion: We recommend that in outbreaks of CCHF, care to prevent airborne transmission should be kept in mind.

**Keywords**: Crimean-Congo hemorrhagic fever; Health care worker; Nosocomial. (Iranian Journal of Clinical Infectious Diseases 2011;6(1):47-50).

## INTRODUCTION

Crimean-Congo hemorrhagic fever (CCHF) virus is a tick-born virus (genus Nairovirus, family Bunyaviridae) with negative-sense, singlestranded, three segmented RNA genome (1), causes a severe hemorrhagic syndrome in humans with fatality rate up to 50% (2-5). The CCHF virus transmission to humans is through the bite of Ixodid ticks (mostly Hyaloma genus) or by contact with blood or tissues from infected livestock (6). In addition to zoonotic transmission, CCHFV is one of the rare hemorrhagic fever viruses which is able

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to cause nosocomial outbreak in hospitals especially among health care workers, because CCHFV can be spread from person to person (7, 8). Nosocomial transmission is well described in reports from Pakistan, Iraq, United Arab Emirates, South Africa (8), and Iran (9); and in all of them contact with the blood and secretions of the patients with CCHF is reported.

We report a nurse who had no history of contact with blood and body secretions of the CCHF patients, but developed CCHF nosocomially. This report justified nosocomial transmission between health care workers, although previously published papers showed the other routes in Iran as well (10-13).

# CASE PRESENTATION

In December 2008, we encountered with an outbreak of CCHF infection in Banarooyeh village near the Jahrom (a city in Fars province in the south of Iran). In this outbreak, six patients with suspicious signs and symptoms of CCHF were admitted in Peymanieh hospital in Jahrom during 17 to 20 December 2008. CCHF was confirmed in 4 cases by serological (IgM and IgG) and molecular analysis which were performed by the National Reference Laboratory in Pasteur Institute of Iran.

The patients were hospitalized and received antiviral (Ribavirin) and supportive therapy but unfortunately one of them died. These patients had history of contact with tissue and blood of an infected livestock (a 2 month cattle) around the day of Muslim Famous Eid (religious ceremony), holy Eid Ghorban (Eid-al-Adha) (14).

The interest case was a 26 years old nurse who had contributed to take care of the mentioned patients. One week after the admission of the aforementioned CCHF patients on 27 December 2008, she referred to the clinic with suffrage of chills, myalgia, headache, and fever (flu-like syndrome). She was admitted and routine laboratory tests were achieved. These tests were CBC with differential, liver enzymes (ALT: alanine transaminase, AST: aspartate aminotransaminase), PT (prothrombin time) and PTT (partial thromboplastin time). All tests were within normal limits, however, the body temperature was 39.5°C continuously, and pulse rate was 120 beats/min. The patient was conscious and had not any neurological symptoms. Physical examination revealed clear lungs but a rather tender abdomen on palpation, especially on epigastric region, liver and spleen were not palpable.

In December 29, platelet count decreased from  $184000/\mu$ l to  $36000/\mu$ l. ALT and AST levels raised (52 U/L and 138 U/L, respectively) and PTT was prolonged to 64 seconds.

On day 3, she was still febrile. She had severe abdominal pain and tenderness. Abdominal ultrasonography revealed the presence of free fluid in the abdominal cavity, suggestive of hemoperitoneum.

Ribavirin (30mg/kg as loading dose and 15mg/kg every 6 hours as therapeutic dose), IVIG (1gr per Kg body weight), and prednisolone (1mg/kg) on day 3<sup>rd</sup> (30 December) and supportive therapies (such as platelets and fresh frozen plasma) were administered. Meanwhile, according to NECVHFs (National Expert Committee on Viral Hemorrhagic Fevers) protocol, her first blood sample (10mL each) was collected (30 December) and immediately centrifuged in the local laboratory, and sent to Laboratory of Arboviruses and Viral Hemorrhagic Fevers (National Reference Laboratory: Pasteur Institute of Iran) for laboratory confirmation (serological and molecular detection) (12). On day 5<sup>th</sup> (1 January 2009), the patient developed petechia and purpura over her extremities and trunk, specially, around the injection sites.

On day  $6^{th}$  (2 January 2009), the platelet count was lower than 30,000/µl and the patient developed epistaxis, gingival bleeding and also melena in spite of platelet transfusion (8 units every 24 hours). In the same day, she was menstruated and her bleeding was profuse, hence, we administered progesterone, too. During the next days, hemoglobin level decreased mandating packed cell transfusion.

Pulmonary hemorrhage was a severe complication that was developed on the 6<sup>th</sup> day with sudden severe dyspnea and bloody sputum, while chest X-ray demonstrated new patchy infiltration all over the lungs, nevertheless, it was controlled by supportive management. In January 4, according to NECVHFs protocol, the second sample has been sent to the National Reference Laboratory. Finally, she was discharged in a good condition on day 13<sup>th</sup> (9 January 2009).

In the laboratory, serum was analyzed by specific ELISA method (IgM and IgG) and viral RNA genome of the virus detected by real-time and gel-based RT-PCR (10,12,15). In serologic laboratory tests, the first sample was negative for IgG and IgM, but molecular analysis was positive. However, IgM antibody revealed positive for the second sample.

Our patient believed that she had not any direct contact with blood and secretions of the patients and it was unbelievable and strange for her to be affected by CCHF. Indeed, she was referring to the patients' rooms (including the CCHF patients) everyday, and distributing their drugs. She had this responsibility during the days when the CCHF patients were hospitalized. She was not responsible for IV or IM injecting drugs, therefore, it seems that she has not had any contact with blood of the patients with CCHF.

## DISCUSSION

Nosocomial transmission can be reduced by precise health care management and, in case of exposure, providing prophylactic treatment to health-care workers is useful (16). For example, in Pakistan, three nosocomial cases of CCHF were treated with oral ribavirin for 10 days, and they made a complete recovery (17,18). It has been postulated that the ignorance of necessary precautions and an insufficient number of isolation rooms may contribute to nosocomial outbreaks of CCHF in developing countries (19,20). In a study conducted in Iran, 3.87% of health care workers who has had exposure with the CCHF patients tested positive for anti CCHF-IgG (21). Papa et al believed that no tertiary case is detected due to CCHF nosocomial outbreaks (22) but Mardani et al described a case of CCHF as a tertiary case (9). Although, the risk for nosocomial transmission in overcrowded hospitals, where basic hygiene measures are not followed, was high (23), it should imply that early laboratory and clinical diagnosis is vital in terms of treatment of patients, prevention of nosocomial infections and reduction of mortality rate (24). Some of the papers suggest that simple barrier methods and care in provision of CCHF cases may prevent transmission of this infection in health care workers (24). However, current VHF management guidelines recommend caution with body fluids such as excreta, vomit, sweat and saliva, especially for patients with respiratory symptoms or in latter stages of illness characterized by vomiting, diarrhea, hemorrhage, and shock. The recommended safety measures include barrier precautions, isolation, gloves, gowns, face-shields, and goggles with side shields when contacting the patient or soiled environmental surfaces. As this nurse is infected without a history of contact with blood and body secretions of the CCHF patients, the previous notes which do not believe in airborne transmission of CCHF (25) may be in doubt; although this postulate needs more study. Therefore, we recommend that in outbreaks of CCHF, care to prevent airborne transmission should be kept in mind.

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