

Deep vein thrombosis in Crimean-Congo hemorrhagic fever: a rare clinical situation

Kırım-Kongo kanamalı ateşinde derin ven trombozu: nadir bir klinik durum

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ABSTRACT

Crimean-Congo hemorrhagic fever is an acute, tick-borne viral disease and potentially fatal by affecting multiple organ systems. Bleeding is one of the severe complications of the disease; however, thromboembolic complications have received less attention. Here, we report a 41-year-old female patient, developed deep vein thrombosis after diagnosed with Crimean-Congo hemorrhagic fever.

Keywords: Crimean-Congo hemorrhagic fever, deep vein thrombosis, venous thromboembolism

ÖZ

Kırım-Kongo kanamalı ateşi akut, kenelerden bulaşan ve multipl organ sistemlerini etkileyerek ölümcül seyredabilen viral bir hastalıktır. Hastalığın en önemli komplikasyonlarından birisi kanama olmasına rağmen tromboembolik komplikasyonlara da dikkat etmek gerekmektedir. Bu yazımızda Kırım-Kongo kanamalı ateşi tanısı aldıktan sonra derin ven trombozu gelişen 41 yaşındaki bayan hastayı sunmayı amaçladık.

Anahtar Kelimeler: Kırım-Kongo kanamalı ateşi, derin ven trombozu, venöz tromboembolizm

INTRODUCTION

Crimean-Congo hemorrhagic fever is a viral disease and transmitted via Hyalomma ticks or direct contact with the blood of infected humans or domestic animals. The most common clinical signs are hemorrhage and fever. Endothelial dysfunction plays a role in hemorrhagic manifestations. At the same time, increased coagulation and platelet adhesion due to endothelial dysfunction lead to thromboembolism, although rarely (1). Close clinically follow-up and appropriate therapy considering bleeding-co-

agulation balance are important in the patients who develop venous thromboembolism while diagnosed with CCHF.

CASE REPORT

Previously healthy 41-year-old female patient admitted to the emergency department with complaints of nausea, vomiting, abdominal pain, and hemoptysis, for 2 weeks. In her detailed history, it was learned that she was exposed to tick-bite one month ago. Physical examination revealed ecchymosis in

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the arms and abdomen and laboratory findings revealed elevated liver enzymes, prolonged INR and aPTT, and low platelet count. The patient was isolated and followed up in an intensive care unit and was diagnosed with Crimean-Congo hemorrhagic fever (CCHF). While the patient was under support therapy for CCHF, the swelling was detected in her right leg on the seventh day of hospitalization. Acute thrombus was seen in the right common and superficial veins and popliteal vein with venous doppler ultrasound. Platelet count was 60000/mm³ at the diagnose time and because of the relatively short half-life that leads to control of the probable bleeding and provide neutralization, standard heparin treatment -4x1cc-, with aPTT follow up was started. Lower extremity elevation and compression therapy were also performed. On the third day of the standard heparin therapy, the patient's symptoms were regressed and platelet count was over 100000/mm³. Then, standard heparin therapy was stopped and enoxaparin sodium 6000 IU, two times a day was started. Warfarin sodium 5 mg a day was added on the second day of low molecular weight heparin (LMWH) therapy.

Eighteen days after hospitalization, the patient was discharged with warfarin sodium for three months, without any complication. The recanalization in deep venous structures was revealed in the control ultrasound after one month.

DISCUSSION

Crimean-Congo hemorrhagic fever is a mortal disease caused by a Nairovirus involve in the family of Bunyaviridae (2). The most characterized symptoms are; fever, vomiting, myalgia, elevated liver enzymes, nausea, headache and hemorrhagic manifestations ranging from mucocutaneous bleeding to life-threatening massive hemorrhage. Mortality rates due to the disease range from 3% to 30% (3). Bleeding may occur up to 90% of the patients (4). Endothelial cells, hepatocytes, and mononuclear phagocytes are the major targets of the disease (5). While the endothelial cells are infected by the virus, this results in a wide range of vascular effects that lead to changes in vascular permeability or hemorrhage (1). Besides this, dysfunction of endothelin, during hemorrhagic fever may lead to thromboembolism. Andersen et al. (6) defined the association between hemorrhagic fever with renal syndrome and myocardial infarction, stroke and venous thromboembolism.

Sijjeel et al. (7) studied the probable mechanisms of deep venous thrombosis in Dengue hemorrhagic fever. Venous thromboembolism consists of deep vein thrombosis and pulmonary embolism may occur in isolation or as a complication of other diseases or

procedures (8).

Endothelial damage contributes to hemostatic failure by stimulating platelet aggregation and degranulation with consequent activation of intrinsic coagulation cascade may play a role in the mechanism of venous thromboembolism (2). In addition, increased coagulation and platelet adhesion due to endothelial damage may lead to venous thromboembolism (6). The imbalance between pro-coagulant and anti-coagulant factors enhances the chances of deep vein thrombosis.

However, the low platelet count, prolonged activated partial thromboplastin time and prothrombin time and decreased fibrinogen levels, venous thromboembolism may occur in CCHF.

In conclusion, although rarely seen in clinical situations, venous thromboembolism must be kept on the mind in patients with CCHF.

ETHICAL STATUS

Institution approval was obtained.

CONFLICTS OF INTEREST

The authors declare that there are no conflicts of interest or supporting funder/organization.

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