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LETTER

Superficial thrombophlebitis in a patient with COVID 19: Heparin treatment after evaluation of D—Dimer

To The Editor,

Coronavirus Disease 2019 (COVID-19) is a respiratory tract disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-Cov-2), which first appeared on December 1, 2019 in Wuhan, China. The pandemic was declared by World Health Organization on March 11, 2020 and as of May 21, 2020, more than 5 million cases were reported worldwide and more than 300 thousand deaths occurred.[1]

The role of obstructive vasculopathy has been an essential topic of discussion in the pathogenesis of COVID-19. Endothelial damage and autoimmune mechanisms have been reported to contribute to the development of microvascular thrombosis and occlusion. Furthermore, severe COVID-19 cases have been shown to be complicated by disseminated intravascular coagulation.[2,3]

A 62-year-old man without previous medical history presented with fever (37.9°C), cough, weakness, and fatigue. The chest computerized tomography scan was compatible with viral pneumonia. The nasopharyngeal swab test for SARS-Cov-2 turned out to be positive as well. A combined therapy, including hydroxychloroquine (Day 1: 2 × 400 mg, Day 2-4: 2 × 200 mg), azithromycin (Day 1: 1 × 500 mg, Day 2-4: 1 × 250 mg), oseltamivir (2 × 75 mg for 5 days) and enoksaparin (40 mg/day) was started. On the fourth day of treatment, a painful swelling and redness occurred over the right hand and arm. The doppler ultrasound examination showed a thrombus in the lumen of a superficial vein, accompanied by inflammatory reaction of adjacent tissues, leading the diagnosis of superficial thrombophlebitis. D-dimer level was 0.82 μg/mL (reference range:0-0.0.5 μg/mL). The other blood tests including complete blood count and biochemical parameters were within normal limits. There was no history of trauma or contact exposure, and no intravenous treatment was administered from the right extremity. The involved extremity was elevated, a warm compresses were applied, and the dose of enoxaparin was doubled (2 × 40 mg/day). On the 6th day following, both thrombophlebitis and pneumonia regressed considerably, and the patient was discharged without additional complication.

Recently, many cutaneous findings associated with COVID-19 have been described in the relevant literature. Purpuric, livedoid, and thrombotic-ischemic skin lesions observed in patients with COVID 19 have been considered as the cutaneous manifestations of obstructive vasculopathy developing during the course of the disease.[4-7] Mazzotta and Troccoli[4] reported acro-ischemic lesions in a 13-year-old boy with COVID-19 while Magro et al observed purpuric lesions in three patients with severe COVID-19. Histopathology of these purpuric lesions showed thrombogenic vasculopathy, and C5b-9 and C4d accumulation. These findings suggest that, in addition to hypercoagulation, complement activation may play a role in the pathogenesis of COVID-19.[6] On the other hand, it has been speculated that structural and non-structural proteins of the SARS-Cov-2 bind to the porphyrin and create a complex, inhibiting the oxygen and carbon dioxide carrying capacity of hemoglobin. Accordingly, tissues that are not adequately oxygenated begin to be damaged and cause an intense release of cytokines. The cytokine storm caused by overproduction of proinflammatory cytokines activates the plasminogen activator inhibitor and complement system, causing endothelial damage and microvascular thrombosis.[8,9]

To the best of our knowledge, this is the first report describing superficial thrombophlebitis in a patient with COVID-19. Cutaneous manifestations observed in COVID 19 may provide further opportunity to understand the pathophysiology of the disease, suggesting new possible targets for specific treatments.

CONFLICT OF INTEREST

The authors declare no potential conflict of interest.

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