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Radial arterial thrombosis in COVID-19: A case report

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ABSTRACT

Thrombosis due to hypercoagulable state is an important cause of morbidity and mortality in coronavirus disease 2019 (COVID-19). . Increased D-dimer levels are an important marker of the presence and risk of thrombosis. In this report, we present that a 59-year-old male patient developed thrombosis in the distal radial arteries despite normal D-dimer level. The patient was treated with enoxaparin, iloprost infusion, and cilostazol. This case should lead us to be very careful that people diagnosed with COVID-19 with normal D-dimer levels may also have thrombosis.

Keywords: COVID-19, Radial artery, Thrombosis

1. INTRODUCTION

Coronavirus disease 2019 (COVID-19) spread all over the world in a short time after the first cases were reported from China in December 2019 [1] [1]. So far, COVID-19 has caused approximately 96 million cases and over 1.8 million deaths worldwide as of January 2021 [2]. The COVID-19 clinical spectrum ranges from asymptomatic to severe manifestations that are sepsis, acute respiratory distress syndrome (ARDS), coagulopathy, and death [1-3]. COVID-19 causes arterial and venous thrombosis with the hypercoagulable state, which is an elevation in D-dimer, prothrombin time, and fibrinogen levels [4,5].

In COVID-19, according to current literature, patients with arterial thrombosis were usually male, older, and have accompanying comorbidities [6-8]. COVID-19 related cumulative incidence of thrombotic events has been reported as 49% including 37% venous, and 3.8% of arterial thrombosis in critically ill intensive care unit (ICU) patients [3]. To date, arterial thrombosis associated with COVID-19 has occurred in many vessels such as the aorta, carotid artery, central nervous system arteries, coronary, superior mesenteric, jejunal, splenic, renal, lower, and upper extremity arteries [6-8].

In this report, we describe an acute radial arterial thrombosis in a patient receiving prophylactic anticoagulation.

2. CASE REPORT

A 59-year-old male patient with a history of hypertension and chronic obstructive pulmonary disease was admitted to our emergency department with cough and shortness of breath. On physical examination, he was hemodynamically stable and had polypnea (30 cycles per minute with 90% of blood oxygen saturation on room air). The Rox index (blood oxygen saturation/Fraction of inspired oxygen x respiratory rate) of the patient was 4.62. In initial laboratory studies, severe lymphopenia (500 x10^3/μL), elevated C reactive protein (CRP) (69.3 mg/L) and hyperfibrinogenemia (627 mg/dL) were seen. Other blood tests result such as leukocyte count, D-dimer level, prothrombin time, activated partial thromboplastin time and platelet count revealed normal limits (Table I). Nasopharyngeal swab for COVID-19 was positive. Thorax computed tomography (CT) was performed and compatible with COVID-19 pneumonia (CO-RADS – 5 [9]-scattered localized ground glass infiltration

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areas in both lungs and occasionally accompanying consolidated areas) (Figure 1). Based on the COVID-19 treatment policy of our hospital, the patient initiated on favipiravir, and prophylactic enoxaparin (4.000 IU once daily) on admission. As the saturation level was below 92, dexamethasone, and oxygen supportive treatment was started. The patient had severe COVID-19 pneumonia based on the COVID severity index [10]. Convalescent plasma treatment was given three times to the patient whose respiratory distress and oxygen requirement increased during the follow-up. On the 11th day of the hospital admission and the diagnosis of COVID-19, the cardiovascular surgery department was consulted when there was blue-discoloration and heat loss in the 3rd and 4th fingers of the right hand (Figure 1). In the laboratory tests performed on the day of thrombosis, the lymphocyte count increased (800 x10^3 /µL), fibrinogen level decreased (273 mg/dl), and platelet count, CRP, prothrombin time, activated partial thromboplastin time, and D-dimer levels were measured as normal. Sinus rhythm was seen on the electrocardiogram, there were no ischemic changes. The patient has no history of arterial intervention. In upper extremity CT angiography, major vascular structures were normal, digital arteries of the right 3rd and 4th fingers were found to be severely occluded of the level of proximal interphalangeal joint (Figure1). As advised by the consultant; the medical treatment was arranged as 1mg/kg twice daily enoxaparin, warming the right hand, administering intravenous iloprost infusion for 5 days, and additional cilostazol (phosphodiesterase inhibitor) twice daily. The cilostazol treatment was planned to continue for at least three months. Surgical embolectomy was not considered. After the iloprost infusion was completed, the fingers of the patient returned to normal color (Figure 1). After 1 month of hospitalization, the patient was discharged after resolution of his pulmonary symptoms and signs.

Table I. *Laboratory results of the patient on the days of admission to the hospital, thrombosis, and the discharged from the hospital*

	Admission	Thrombosis	Discharge
White blood cell (x10^3 /L)	7000	16.100	13.800
Neutrophils (#)	6000	14.600	12.300
Lymphocytes (#)	500	800	600
Neutrophils (%)	85.9	90.6	89.7
Lymphocytes (%)	6.9	4.7	4.4
Hemoglobin (g/dL)	15	14.6	13.8
Hematocrit (%)	41.8	41.7	41.7
Mean cell volume (fL)	88.8	89.9	90.3
Platelet (x10^3 /L)	230	400	234
Lactate dehydrogenase (U/L)	673	540	291
C-reactive protein (mg/L)	69.3	1.9	0.70
Fibrinogen (mg/dL)	627	273	228
D-dimer (mg/L)	0.42	0.21	0.27
Protrombin Time (s)	16.4	14.3	13.9
INR	1.24	1.09	1.05
aPTT (s)	29.9	25.2	25.9

INR: International normalized ratio, aPTT: Activated partial thromboplastin time

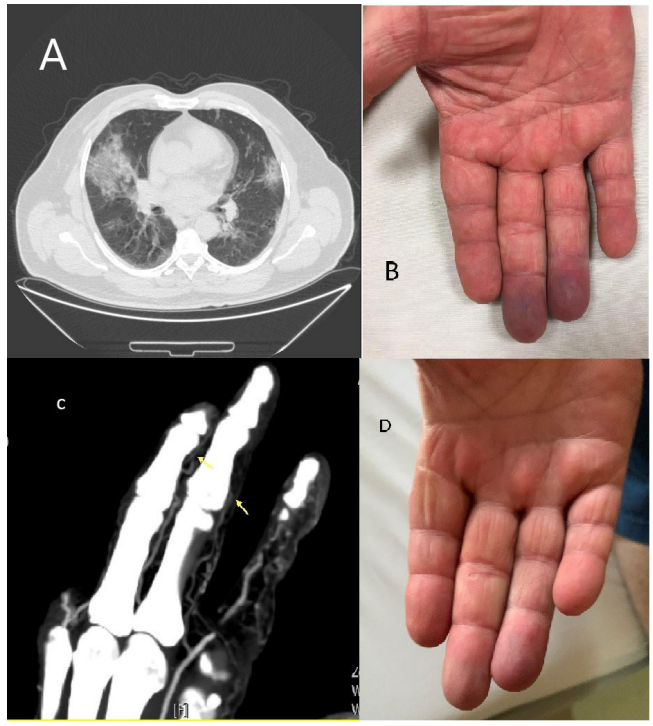

Figure 1. *Selected computed tomography (CT) of the patient. CT of the thorax (A) and upper extremity angiography (C). (B) view of the patient's right hand on the day of thrombosis, (C) an abrupt occlusion of the radial artery is evident on angiography, yellow arrow indicates the thrombosis level and (D) view of the patient's right hand on the day of discharge from the hospital.*

3. DISCUSSION

Many studies have reported a relationship between COVID-19 and hypercoagulable status [1,4,5]. Besides, elevated D-dimer levels were associated with mortality in COVID-19 patients [11]. Although, venous thrombosis is the most common, the risk of arterial thrombosis is also increased [3]. In this case, we have observed COVID-19 may have acute limb ischemia even the D-dimer level is normal. Cholesterol embolism, which will be considered in the differential diagnosis, was excluded because the major vascular vessels were normal in CT angiography.

In the case reports and series published to date, thrombosis has generally been reported in the great arteries [6-8]. In a study in the United States, thrombosis in the ulnar and radial arteries was reported only in 6.1% (3/49) patients [6]. Acute arterial occlusion is a vascular emergency, can lead to life or limb-threatening ischemia [6-8]. It may cause serious clinical findings, poor prognosis, and mortality [6,8,12,13]. In the study of Bellosta et al., only 1 patient had upper limb ischemia, and this patient died due to the development of ARDS after lower limb occlusion in the later period. [14]. Digital ischemia was observed in 2 patients in the intensive care unit, and one of these patients was discharged to the acute care rehabilitation center while the other had mortality, and concurrent venous thrombosis was also demonstrated in these two cases [15]. In another case report, a

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34-year-old female patient reported stroke and brachial artery thrombosis at in same time [16]. Considering all these, it should be kept in mind that thrombosis may be seen in other organs concurrently because of coagulopathy. In COVID-19, according to published data, patients with arterial thrombosis were male, older, and have comorbidities, like our patient [6-8]. In our case, there was thrombosis in the radial artery in a 59-year-old male patient with comorbidities. Our patient was discharged home 2 weeks after thrombosis. There was no thrombosis in another organ or extremity during follow-up.

Connolly et al., reported palmar vein thrombosis in a female patient [17]. But, this patient is a carrier of factor V Leiden mutation. On the other hand, our patient did not have a known thrombophilia condition, thrombosis occurred in his digital arteries due to COVID-19 infection.

Both the COVID-19 disease itself and the thrombotic complications elevate D-dimer levels [5,6,14]. Etkin et al., reported that all 49 patients with thrombosis had high D-dimer levels [6]. In a series of 20 patients, the mean D-dimer was 2200 ng / mL [14]. In another study with 388 patients, only one patient with subsegmental pulmonary embolism had a D-dimer level in the normal range [18]. In our case, there was not any elevation in the D-dimer level that was observed both at the time of diagnosis, during the development of thrombosis, and follow-up.

Conclusion

This case should lead us to be very careful that people diagnosed with COVID-19 with normal D-dimer levels may also have thrombosis.

Compliance with Ethical Standards

This research was conducted ethically by following per under Helsinki World Medical Association Declaration.

Patient Consent: The patient gave his consent for images and other clinical information relating to his case to be reported in a medical publication.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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