OLGU SUNUMU/CASE REPORT

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May Covid-19 Disease Cause Renal Infarction in Young People without Comorbidity?

Covid-19 Hastalığı Komorbiditesi Olmayan Gençlerde Böbrek Enfarktı Yapabilir Mi?

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ABSTRACT

Coronavirus disease 19 (COVID- 19) is a life threatening contagious infection caused by coronavirus 2 (SARS-CoV-2). Hypertrombotic state and endotheliopathy occurring in COVID-19 infection may affect numerous organs as well as influence renal tubular and endothelial cells, causing ARI that is a rarely seen condition. This reveals the significant extent of microvascular and endothelial damage caused by the virus. Similarly to the above mentioned definition, cases of ARI induced by COVID-19 in elderly and/or patients with comorbidity prone to thrombosis have been reported in the literature. Herein, we present a case of ARI secondary to COVID-19 that was developed in a patient without typical COVID-19 symptoms and comorbidity.

Keywords: covid-19, acute renal infarction, endotheliopathy

ÖZ

Coronavirus Disease 19(COVİD- 19) hastalığı, yaşamı tehdit eden, koronavirüs 2'nin (SARS-CoV-2) neden olduğu bulaşıcı bir enfeksiyondur. COVID-19 enfeksiyonunda oluşan hipertrombotik durum ve endotelyopati bir çok organı etkileyebildiği gibi böbrek tübüler ve endotel hücrelerini de etkileyerek nadir görülen bir durum olan akut renal enfarkta sebep olabilir. Bu durum virüsün meydana getirdiği mikrovasküler ve endotel hasarının ne derece önemli boyutta olduğunu gözler önüne sermektedir. Literatürde, yukarıdaki tanıma benzer şekilde, tromboza yatkın yaşlı ve/veya komorbiditesi olan hastalarda COVİD-19 hastalığı ile indüklenen akut renal enfarkt vakaları bildirilmiştir. Bizde burada tipik covid 19 belirtisi ve komorbiditesi olmayan COVİD-19'a sekonder gelişen akut renal enfarkt olgusunu sunduk.

Anahtar Kelimeler: covid-19, akut böbrek enfarktı, endotelyopati

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INTRODUCTION

Coronavirus Disease 19 (COVID- 19), which is still affecting the whole world is caused by a single-chain, positive-polarity enveloped RNA virus belonging to Coronaviridae family (1). Since the transmission occurs more commonly via droplets, the lung is the most often affected organ. However, all organs and systems may be involved through Angiotensin Converting Enzyme (ACE2) receptors (2). Primarily endothelial cells are damaged by direct effect of the virüs. The resultant endotheliopathy leads to the development of inflammation and edema. The levels of cytokines and antiphospholipids increase. The amount of coagulation factors produced by the liver increases and a prothrombotic state is developed in which an especially extrinsic coagulation cascade is activated. This can be noticed with elevated levels of some laboratory parameters including Fibrin Degradation Products, Fibrinogen and D-Dimer, mild PT and PTT prolongation and a mild decrease in platelet counts. Thrombosis developing secondary to hypercoagulation state has been shown in autopsy series up to 58% (3).

Acute renal infarction (ARI) is a rare disease with frequent delays in diagnosis, about which most of the available information is synthesized from case reports (4). Since it is rarely seen, case reports consist of populations including a small number of cases. ARI often develops in the presence of an underlying thromboembolic risk factor, especially like atrial fibrillation. Herein we share acute renal infarction developed secondary to COVID-19 in a young male patient who had no additional risk factors and typical COVID-19 findings.

CASE REPORT

A 30-year-old male patient presented to the emergency service of our hospital with the complaint of flank and abdominal pain that started 2 days ago. His medical history was unremarkable and the patient had no accompanying systemic disease. General status of the patient who had no history of trauma, was moderate and he was conscious, oriented and cooperative. On physical examination, there was voluntary defense in the abdominal right upper quadrant, no rebound finding was observed and the costovertebral angle tenderness on the right side was positive. Vital findings were stable during admission. Laboratory parameters at the time the first presentation were found as WBC: 20.400 mcL, Hg: 14.8 g/dL, platelets: 208.000 mcL, lymphocytes count:1870 /mL, blood urea nitrogen: 14.77 mg/dL, creatinine:1.05 mg/dL, AST:65 u/L , ALT:43 u/l , Na:144 mmol/L , K:4.35 meq/L, amylase: 36 u/L, direct bilirubin: 0.22 mg/dL, total bilirubin: 0.66 mg/dL, Ca:8.72 mg/dL, Cl:107.9 mmol/L, and complete urine analysis:12 leukocytes, 2 leukocytes, nitrite (-). Electrogram of the patient showed sinus rhythm. Because pain could not be explained with whole abdomen ultrasonography, unenhanced and iv enhanced whole abdomen computed tomography (CT) was ordered. On CT sections, a parenchymal area of 46 mm that did not show hypodense enhancement was observed in the lower pole of the right kidney (Figure 1). This image was evaluated as acute renal infarction. Whereas, the left kidney was normal. No other feature that could cause the right colic pain was observed on the abdominal CT.

The patient who was considered to have acute renal infarction following the initial evaluations was assessed by the departments of nephrology and interventional radiology. The department of interventional radiology reported that the common renal artery was open, there may be an infarction developed secondary to vasculitis, which involved the segmental artery, and that no emergency intervention was. The department of nephrology recommended control by nephrology outpatient clinic with follow-up of renal function and blood pressure. Upon pain of the patient continued and WBC was

elevated, a nasopharyngeal smear sample was taken from the patients and he was interned in the urology service for further investigation and treatment.

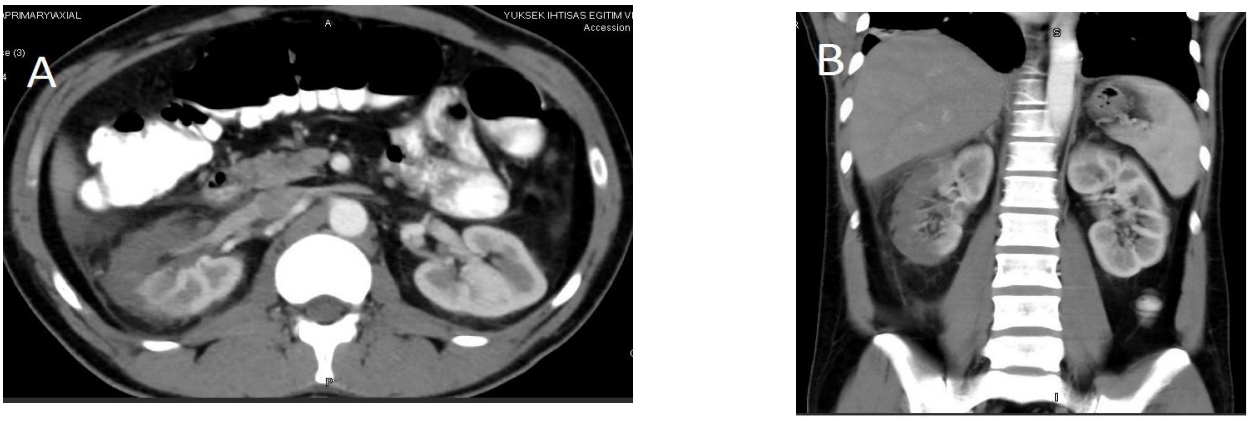

Figure 1 A: Axial section, B: Coronal section. Posterior and lateral part of the right kidney, hypodense, non-enhancing parenchyma view.

Since the nasopharyngeal smear sample tested positive with Polymerase Chain Reaction (PCR) test, treatment and follow-up of the patient was initiated with favipiravir as 2x1600 mg loading dose mg and 2x600 mg maintenance, 4000 i.u sc low-molecular weight heparin and iv 1g 2x1 ceftriaxone. Postero-anterior chest X-ray of the patient showed no consolidation area. After it was learned that the patient was SARS-CoV-2 positive, biochemical analysis was reported as WBC: 10.250, INR:0.94 ku/L, PT:11.5 sec, aPTT:20.4 sec, CRP: 123 mg/L, Ferritin: 269 ng/mL, LDH:858 u/L, Troponin t:3.01 pg/mL, Fibrinogen:900 mg/dL, and D-dimer:2.23 g/mL. No bacterial growth was observed in the urine culture.

The patient did not develop respiratory symptoms, and saturation values were normal during hospitalization. Upon complaints of the patient regressed after 5-day treatment, he was advised therapeutic anticoagulants and 14-day quarantine at home and discharged.

DISCUSSION

Hyperthrombic state and endotheliopathy caused by COVID-19 combined with underlying conditions such as atrial fibrillation, coronary artery disease, diabetes mellitus, previous vascular surgery and hypertension, increase the risk of developing acute renal infarction, which is an extremely rare situation. Studies in the literature have reported cases of acute renal infarction induced by COVID-19 disease in patients prone to thrombosis and/or having comorbidity, similar to the definition above (5). This situation reveals the extent of microvascular and endothelial damage caused by the virus. Herein, in order to provide contribution to the literature, we present a case of acute renal infarction developed secondary to COVID-19, in a young patient who had no typical COVID-19 symptoms and comorbidity.

COVİD-19 predisposes to a thrombotic state by affecting both arterial and venous systems in severe patients. This predisposition may lead to several conditions such as Deep Vein Thrombosis (DVT), venous thromboembolism, pulmonary embolism, and arterial thromboembolism (2). As a result, many organs are influenced, although the most commonly seen prothrombotic event is pulmonary embolism. Infarctions resulting from thromboembolisms have been reported in extrapulmonary organs such as the brain,

intestines, spleen extremities and even isolated gallbladder (2,6,7). Mortality significantly increases with these infarctions especially when the brain is involved and the infarcts are multiple (2).

Progression of renal involvement is highly affected negatively especially in severe COVID-19 patients. This effect can be explained by several ways. First, SARS-CoV-2 directly causes acute tubular necrosis and peritubular lymphocyte infiltration. Furthermore, the extent of tubular damage increases by the virus triggering Membrane Attack Complex (C5b-C9) and increasing CD68 macrophage count. In addition, hypoperfusion related renal tubular damage is also added as a result of cytokine storms developed due to acute inflammation. All of these increase the risk of developing acute renal failure. Acute renal failure is an independent risk factor for mortality in COVID-19 patients (8). In our patient, renal functions were not influenced initially and at follow-up. This can be attributed to the absence of comorbidities and good renal reserve of the patient. Conversely, renal functions and urinary output may be affected and even dialysis may be needed in patients with a transplanted kidney or immunosuppressed patients (9).

Among the laboratory parameters, elevated WBC at the time of first admission of our patient and elevated lactate dehydrogenase at the second sampling were remarkable. Rarely seen acute renal infarction often presents with acute-onset flank pain, nausea and vomiting. Oliguria is not a very expected finding if there is no bilateral embolism or solitary kidney. Slightly elevated blood pressure may accompany as a result of the renin dependent mechanism. Elevated white blood cells as well as high levels of creatinine in bilateral cases may be seen. The presence of microscopic hematuria, which was seen in our patient, has been reported up to 72% in case series in the literature (10). However, it should be remembered that the absence of microscopic hematuria may indicate a greater infarction area. Elevated lactate dehydrogenase just as in myocardial infarction is another significant laboratory finding, which was also observed in our patient. If there is no suspected infarction in another organ, elevated lactate dehydrogenase is sensitive, but is not specific in terms of acute renal infarction (4).

As far as we could see in the literature, there was no young patient with cardiac disease, rheumatological disease or a history of malignancy and typical COVID-19 findings that predispose to thrombosis. Therefore, I think that the case we presented here is important in this context.

CONCLUSION

In conclusion, we believe that infarction of solid organs such as kidney must be ruled out with enhanced whole abdomen CT in all patients presenting with flank and abdominal pain, even if the patient had no typical COVID-19 symptoms, considering that there may be a hyperthrombotic or endotheliopathy complication caused by COVID-19.

ETHICAL STATEMENT

Informed consent was obtained once the risks were discussed with the patient in details. This case report did not require ethical approval from our institute. The patient gave permission for the publication of details of his case. His anonymity has been preserved.

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