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COVID-19 presented as acute kidney injury with secondary myocardial damage

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ABSTRACT

The most common manifestations of the 2019 novel coronavirus disease (COVID-19) include fever, cough, dyspnea. Nevertheless, many atypical forms of presentation might be present, delaying a correct diagnosis.

Acute kidney injury (AKI) is one of the important complications of COVID-19, occurring in 0.5–7% of cases and in 2.9–23% of ICU patients. The exact mechanisms by which COVID-19 induces AKI in different clinical settings is still a matter of debate.

We present the case of a 53-year old woman, without any prior renal pathology, admitted to a Cardiology Department for atypical thoracic pain and oligo-anuria, without respiratory symptoms, who was diagnosed with SARS-CoV-2 infection. The patient had a significant rise in high-sensitivity cardiac troponin (from 304 ng/L to 889 ng/L in one hour) and mild systolic dysfunction (LVEF 45%), which led to the initial misdiagnosis of an acute myocardial infarction. Blood tests confirmed the diagnosis of acute kidney injury (creatinine 8.8 mg/dL in two different samples). She received hydro-electrolytic rebalancing treatment, with good clinical and biological evolution. To our knowledge this is one of the first reports, that highlights the existence of myocardial injury secondary to acute kidney injury caused by SARS-CoV-2 infection, in a patient without respiratory symptoms.

Introduction

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pandemic has affected more than 25 million people from over 200 countries, claiming over 847.000 lives (by August 30th, 2020) [1]. Acute kidney injury (AKI) is one of the important complications, occurring in 0.5–7% of cases and in 2.9–23% of ICU patients [2]. There are only a limited number of cases reported with patients positive for SARS-CoV-2, presenting with AKI and no respiratory symptoms.

Case report

We report the case of a 53-year old woman, hotel maid, with novel coronavirus disease 2019 (COVID-19) presented as AKI. The patient was admitted initially to a Cardiology Department of an Emergency Hospital due to discrete thoracic pain and biological suspicion of myocardial infarction.

The patient had no symptoms until 2 days before admission, when oligo-anuria developed. She had nausea and had been vomiting. She reported no respiratory symptoms or fever. Smell and taste were preserved. The patient had a history of untreated hypertension, hyperlipidemia and was a smoker. Her renal function was priorly normal. She did not drink alcohol or use illicit substances. She took no medication at home.

On examination, the temperature was 36.2 °C, blood pressure 140/80 mm Hg, heart rate 80 beats per minute, respiratory rate 15 breaths per minute, and oxygen saturation 91% in ambient air. Body-mass index was 31 kg/m^2. Pulmonary auscultation was normal. Mucous membranes were dry. Urine appearance was cloudy and the output was approximately 150 mL in the last 12 h.

Hemogram showed normal hemoglobin and white blood cells count (10.7×10^3) and mild monocytosis, both percentual (12.3%) and in absolute value (1.3×10^3). Blood creatinine was 8.8 mg/dL in two different samples. Blood urea nitrogen (BUN) was 239 mg/dL. She also had hyperkalemia (6.9 mmol/L) and hyponatremia. There was no hepatic cytolysis. Uric acid levels were very high (20.4

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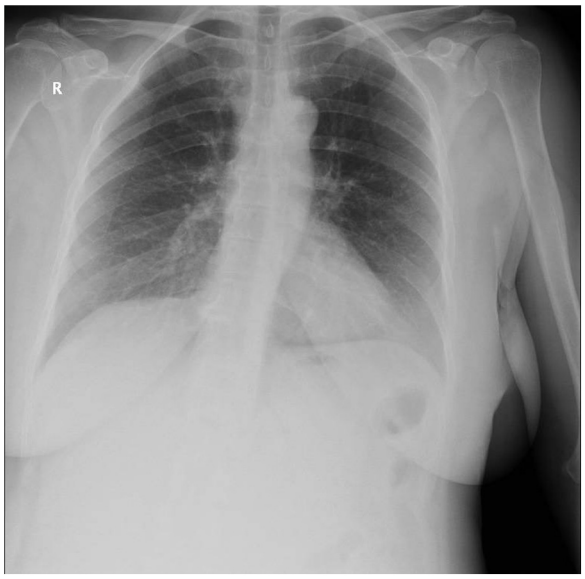

Fig. 1. Chest X-ray without signs of COVID-19 pneumonia.

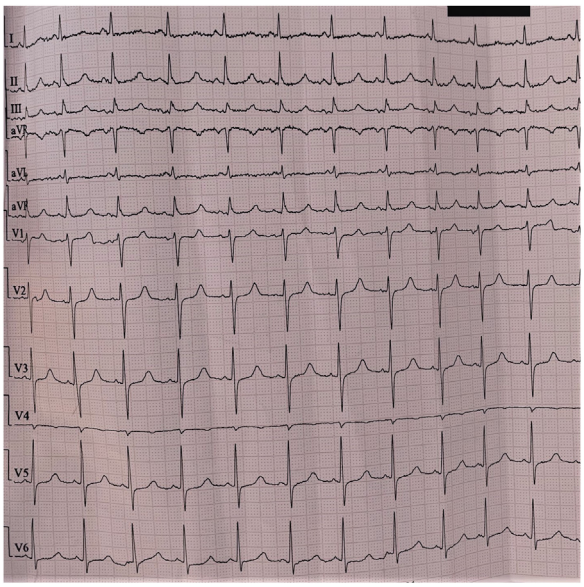

Fig. 2. ECG without signs of myocardial ischemia/necrosis.

mg/dL). Creatine kinase (CK) was 13-times higher than laboratory upper limit (2607 U/L) and Creatine kinase-MB (CK-MB) was 3.7-times higher than laboratory upper limit (93 U/L). There was an increase in high-Sensitivity Cardiac Troponin (hs-cTnI) from 304 ng/L to 889 ng/L in one hour. NT-proBNP was mildly elevated (301 pg/mL). Coagulation was normal and D-dimers were negative. Inflammation biomarkers were mildly elevated: erythrocyte sedimentation rate 28 mm/h, C-reactive protein (CRP) 2.2 mg/dL, ferritin 337 μg/dL. Procalcitonin was negative. Viral markers for hepatitis and for HIV infection were negative. The urinalysis showed proteinuria, the presence of blood and no leukocytes in the urine (qualitative analysis). Blood gas analysis showed compensated metabolic acidosis, with a mild increase in the blood lactate (2.16 mmol/L).

Due to the epidemiological context and to limit in-hospital spread of the SARS-CoV-2 infection, all patients were isolated and tested for the virus upon hospital admission. Results of the real-time PCR were available after aproximately 12 h.

Chest X-ray showed accentuation of the interstitial pattern bilaterally with the appearence of fibrosis and stasis (Fig. 1).

Abdominal sonography showed no pathological findings and kidneys with normal dimensions.

Serial electrocardiograms showed no patological changes (Fig. 2).

Transthoracic echocardiography showed normal heart dimensions, mild systolic dysfunction (LVEF 45%) without segmental kinetics alteration, no significant valvular disease, no intracardiac masses and no pericardial effusion.

The patient received hydro-electrolytic rebalancing treatment, with improvement of the urine output (1000 mL/12 h) and a decrease in the creatinine blood levels (5.31 mg/dL), BUN (235 mg/dL) and potassium blood levels (3.53 mmol/L). After receiving the result of the RT-PCR, the patient was transferred to the COVID unit of an infectious disease hospital.

The COVID unit performed a thoracic CT scan, without pathological findings suggestive for SARS-CoV-2 pneumonia. Hydro-electrolytic rebalancing treatment, as well as betablocker (50 mg metoprolol daily), simple antiplatelet treatment and low-dose statin (20 mg atorvastatin daily) were continued. The patient remained hospitalized for 11 days, until the RT-PCR test for the SARS-CoV-2 was negative. During hospitalization, creatinine (0.73 mg/dL), potassium (3.79 mmol/L) and sodium (135 mmol/L) levels returned to normal. Also troponin levels returned to baseline: 115 pg/mL at 24 h, 84.03 pg/mL at 36 h, 15 pg/mL at 10 days.

Repeated urinalysis showed microproteinuria (50 mg/dL) and glycosuria (300 mg/dL), but no other pathological finding.

The patient remained hemodynamically and respiratory stable, slowly regained diuresis and had no other symptoms until discharge.

Discussion

COVID-19 affects many organs, but the exact mechanism by which it attacks the kidney is still unclear [3]. Both intrinsic renal mechanisms (such as thrombotic vascular processes, viral mediated tubular cell injury, and glomerulonephritis), as well as extrinsic mechanisms (fluid depletion, multi-organ failure, and rhabdomyolysis) have been reported.

The entrance of the virus in an organ is conditioned by the presence of the angiotensin-converting enzyme 2 (ACE2), to which the spike (S) protein of SARS-CoV-2 binds, as well as of cellular transmembrane serine proteases (TMPRSSs) that cleave the S protein. Co-expression of ACE2 and TMPRSS occurs in podocytes and proximal straight tubule cells [4].

Also, high levels of circulating inflammation mediators determined by viral infection can cause endothelial dysfunction, microcirculatory derangement, and tubular injury [5].

Case reports with kidney biopsy have suggested acute tubular necrosis (ATN) as the most common cause of AKI, probably because endothelial cells have a high expression of ACE-2, target of COVID-19 [6]. Collapsing glomerulopathy has also been reported as a presentation form of COVID-19 even in the absence of severe respiratory disease [7,8].

SARS-CoV-2 activates the Renine-Angiotenin-Aldosterone System (RAAS), which contributes to the development of cardiovascular injury after AKI in the SARS-CoV-2 infection [9].

AKI is one of the important complications of COVID-19 and is frequently associated with cardiac damage [10]. Up to 43% of patients have proteinuria and 11% hematuria [7]. Regardless of the pathogenic mechanism underlying AKI, it can be associated with a rise in myocardial biomarkers, including necrosis markers. This can lead to misdiagnosis of a potential acute coronary syndrome.

Our case illustrates that even a significant rise in high sensitivity troponin should rather be considered as a consequence of

myocardial injury secondary to AKI in the setting of SARS-CoV-2 infection, than the expression of myocardial necrosis. Moreover, our case illustrates the various ways in which the novel coronavirus disease can manifest, frequently without any pulmonary signs or symptoms.

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Competing interests

None declared.

Ethical approval

Not required.

Authors' contributions

All authors contributed equally in drafting and writing the manuscript. All authors have approved the present submission.

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