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NEPHROLOGY - LETTER TO THE EDITOR

Acute kidney injury in COVID-19: a case-report

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Editor,

A 81-year-old-Caucasian- female with a history of rheumatoid arthritis in chronic treatment methotrexate and metilprednisolone showed fever and chills partially responsive to antibiotic therapy, and dry cough. Since the persistence of the symptoms, she underwent an oropharyngeal swab for SARS-CoV-2-RNA, resulted positive and was hospitalized at our COVID-19 unit. A thoracic CT scan showed bilateral interstitial pneumonia with ground glass opacities. At the admission, the patient didn't need oxygen therapy, and PaO2/FiO2 at haemogasanalysis was 319. Her clinical parameters and lab tests were in the normal range (Table 1), except for mild anemia and high D-dimer. A therapy with lopinavir/ritonavir, hydroxychloroquine, enoxaparine was started. During the observation, at day 4 from hospitalization, she experienced progressive dyspnoea and hypoxemia despite a respiratory support with Venturi Mask (FiO_2 24/28%). Suspecting pulmonary embolism (PE), a thoracic angio-CT scan was performed, resulted negative for PE, but suggestive of an interstitial involvement more severe than the previous CT. Adjunctive therapy with tocilizumab and metilprednisolone was started. During the following days, the patient developed hypotension, oliguria and impaired renal function, with the lower glomerular filtration rate (GFR) of 16 mL/min, corresponding to a serum creatinine (SCr) of 2.7 mg/dL, 3 days after the angio-CT scan (day 7 from hospitalization, Table 1).

So, a contrast induced nephropathy (CIN) was suspected, and intravenous hydration therapy started. The patient did not drink any water, and also presented clinical signs of dehydration. Urinalysis was normal. She showed a restoration of a normal diuresis and improvement of renal function (SCr and GFR), although never as normal as before the admission. She also developed unexplained anemia and thrombocytopenia, initially interpreted as related to enoxaparine, but more probably related to COVID-19.

After 30 days of hospitalization, and two swab test resulted negative for SARS-CoV2, the patient was discharged in a good respiratory and clinical condition.

Analyzing the possible causes of acute kidney injury (AKI), we can reasonably consider it as a CIN, for the onset of the renal function impairment occurred 3 days after intravenous contrast administration [1]. Rheumatoid arthritis (RA) or other causes of renal damage, as glomerulonephritis, seem to be unlikely, respectively due to the good control of RA and to the absence of worsening hypertension, proteinuria or hematuria caused by an acute glomerulonephritis.

The progression of renal damage cannot be attributed to the radiocontrast agent only: in fact, despite the restoration of a normal diuresis and the improvement of kidney function, SCr and GFR never returned to pre-hospitalization values, as we could usually expect in a CIN treated properly, and without a previous history of chronic kidney disease [2].

Furthermore, marked hypotension and the simultaneous deterioration of respiratory function suggested COVID-19 infection may have played a role [3, 4]. Increasing of LDH, D-Dimer and thrombocytopenia suggests a high inflammatory response to the infection. We can suppose that inflammation and immune response played an important role in the development of AKI, along with CIN [5–7].

The present clinical case suggests that during COVID-19 AKI may occur because of different causes that the physician should identify for a good management.

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Table 1 Biochemical data at admission and at day 7 of hospitalization

	At admission	Day 7 after admission
Haemoglobin	11.8 g/dL	9.1 g/dL
White blood cells	4.46×10^3/mm^3	3.44×10^3/mm^3
Platelets	132×10^3/mm^3	248×10^3/mm^3
Serum creatinine	0.8 mg/dL	2.7 mg/dL
Urea	49 mg/dL	103 mg/dL
Na^+	137 mEq/L	133 mEq/L
K^+	4.3 mEq/L	3.8 mEq/L
Lactate dehydrogenase	376 U/L	354 U/L
D-dimer	33 µg/mL—55×	23 mcg/mL—38×

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