Acute kidney injury caused by COVID-19 in a patient with Crohn’s disease treated with adalimumab

COVID-19, caused by SARS-CoV-2, has become a global threat in a matter of months. In particular, Italy has been one of the most affected areas worldwide. Data regarding the clinical picture and clinical course of COVID-19 are still lacking, especially in non-Asian populations. COVID-19 usually causes an influenza-like syndrome, including cough, fever, sore throat, muscle pain, and in some cases a more severe clinical picture, including interstitial pneumonia with acute respiratory distress syndrome, which may be fatal. Acute kidney failure has been reported in a substantial proportion of patients with COVID-19, but little is known regarding the mechanisms leading to kidney injury. Besides fluid depletion, which is expected to be common, a direct damage caused by the virus could be potentially responsible for kidney injury. In fact, as it has been recently hypothesised, SARS-CoV-2 may reach the kidney, as well as other organs, via viral sepsis. In turn, this process might be favoured by a defective immune response towards the virus, as in the case of patients treated with immunomodulating drugs, including biological therapies. The impact of COVID-19 in patients suffering from inflammatory bowel disease, particularly those treated with biological agents, is poorly characterised, and data are still emerging.

We here describe the unique clinical course of a COVID-19 patient with Crohn’s disease (CD) under biological therapy who was admitted to our internal medicine unit for acute kidney injury. A 25-year-old man has been suffering from CD since the age of 18, when he underwent a colonoscopy that showed multiple ulcers at the terminal ileum, caecum and transverse colon, with a substenotic ileocecal valve (Montreal classification A2L3B1). After a short course of budesonide, adalimumab was commenced in 2014 and continued as maintenance therapy at a dose of 40 mg every other week, achieving and maintaining remission since then. The last follow-up ileocolonoscopy (October 2019) showed no signs of endoscopic and histological activity (Simple Endoscopic Score-CD 1). Except for CD, his medical history was unremarkable. The patient presented to our gastroenterology outpatient clinic in March 2020 for a routine appointment. The patient lived in a nearby town where the first COVID-19 Italian outbreak started earlier in February. He had no fever, cough and dyspnoea and did not complain of any gastrointestinal symptom. Abdomen, chest and heart physical examination was unremarkable, and there was no sign of peripheral oedema. His arterial blood pressure was 180/90 mm Hg, and oxygen saturation was 99%, while he was breathing ambient air. Routine blood tests revealed mild normocytic anaemia and marked increase of serum creatinine (384.5 µmol/L, estimated glomerular filtration rate 0.3 mL/s/m², according to the Chronic Kidney Disease Epidemiology Collaboration equation). Before admission to the ward, according to the recent reorganisation of our hospital due to COVID-19 outbreak, he underwent nasopharyngeal swab for detecting SARS-CoV-2, which turned out to be positive on reverse transcriptase PCR assay. In particular, total nucleic acids were extracted from the samples (200 µL)
using the QIAsymphony instrument with QIAsymphony DSP Virus/Pathogen Midi Kit following the manufacturer’s instructions (QIAGEN, Hilden, Germany). Specific real-time PCR targeting RNA-dependent RNA polymerase and E genes were used to detect the presence of SARS-CoV-2. On admission, the patient developed dry cough and mild fever (maximum peak 37.9°C), without respiratory failure, and a chest X-ray showed mild lung interstitial thickening. Adalimumab was then discontinued (last dose was given 5 days prior to hospital admission), and on the basis of laboratory tests showing hypoalbuminaemia, albuminuria and hypercholesterolaemia (table 1), a nephrotic syndrome was diagnosed. A kidney biopsy was performed, revealing acute tubular necrosis, with loss of brush border and vacuolar degeneration (figure 1A), accompanied by moderate, patchy interstitial fibrosis (involving about 50% of the interstitium), with no clear evidence of vasculitis. The pattern of fibrosis was patchy and striped. The advanced stage of the renal disease did not allow recognition of features typical of a specific glomerulonephritis.

The authors argue whether the use of adalimumab might have promoted a favourable outcome, with particular regard to lung involvement. A growing body of evidence indicates that COVID-19 is actually a systemic infection with multiorgan involvement, at least in a subset of patients. Theoretically, anti-TNFα agents might lead to a more favourable COVID-19 course, as it may counteract the cytokine storm which is typically seen during this infection. However, whether the reduced immunosurveillance caused by biological agents may favour dissemination of SARS-CoV-2 to other organs still needs to be ascertained.

To conclude, from a clinical point of view, this case report underlines the importance of considering acute kidney injury as the only presentation of COVID-19. SARS-CoV-2 may cause a direct kidney injury, possibly via viral sepsis. Even if preliminary data seem to support the use of biological agents in patients with inflammatory bowel disease, the safety of anti-TNFα agents and other biologics in patients with COVID-19 should be further investigated.

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