

# Characteristic and management of Sars-Cov-2 pneumonia without Remdesivir in patient with chronic kidney diseases and sarcomatous endothelial carcinoma

Weimer LE<sup>1\*</sup>, Cattari G<sup>2</sup>, Binelli A<sup>1</sup>, Fanales-Belasio E<sup>3</sup>, Poddighe AF<sup>2</sup> and Sensi F<sup>2</sup>

<sup>1</sup>National Center for Global Health, Istituto Superiore di Sanità, Rome, Italy

<sup>2</sup>SSD Lungodegenza-ASSL Sassari-ATS Sardegna, Italy

<sup>3</sup>Department of Infectious Diseases, MIPI, Istituto Superiore di Sanità, Rome, Italy

## Abstract

The SARS-CoV-2 pandemic has already infected more than 98 million people worldwide and resulted in 2.1 million deaths. Though originally described as a respiratory virus, SARS-CoV-2 has now been shown to have multiorgan involvement.

Chronic kidney disease (CKD) has emerged as a risk factor for adverse outcomes. The virus can cause renal involvement, and severe renal dysfunction is more common among patients with chronic comorbid conditions, especially patients with chronic kidney disease.

Angiotensin-converting enzyme 2 (ACE2) has been proven to be the major receptor of SARS-CoV-2 in kidneys, suggesting that ACE2-related changes may be involved in renal injury during the infection.

For patients with SARS-CoV-2 infection, renal injury by either direct infection or systemic effects, including host immune clearance and immune tolerance disorders, endothelium-mediated vasculitis, thrombus formation, glucose and lipid metabolism disorder, increased serum creatinine, variable degrees of proteinuria, hematuria, and radiographic abnormalities of the kidneys and hypoxia.

Here, we report an Italian patient with Chronic kidney diseases, hypertension and sarcomatous endothelial carcinoma responding to alternative therapy with regression of pneumonia caused by Sars-Cov-2 without Remdesivir for nephrotoxicity.

## Background

The SARS-CoV-2 pandemic has already infected more than 98 million people worldwide and resulted in 2.1 million deaths [1]. Though originally described as a respiratory virus, SARS-CoV-2 has now been shown to have multiorgan involvement [2].

Chronic kidney disease (CKD) has emerged as a risk factor for adverse outcomes.

The virus can cause renal involvement, and severe renal dysfunction is more common among patients with chronic comorbid conditions, especially patients with chronic kidney disease.

Angiotensin-converting enzyme 2 (ACE2) has been proven to be the major receptor of SARS-CoV-2 in kidneys, suggesting that ACE2-related changes may be involved in renal injury during the infection [3].

For patients with SARS-CoV-2 infection, renal injury by either direct infection or systemic effects, including host immune clearance and immune tolerance disorders, endothelium-mediated vasculitis, thrombus formation, glucose and lipid metabolism disorder, increased serum creatinine, variable degrees of proteinuria, hematuria, and radiographic abnormalities of the kidneys and hypoxia.

Here, we report an Italian patient with Chronic kidney diseases, hypertension and sarcomatous endothelial carcinoma responding to alternative therapy regression of pneumonia caused by Sars-Cov-2 without Remdesivir for nephrotoxicity.

## Description

Our Italian patient 84-year-old, woman, developed asthenia, anosmia, ageusia, myalgia, dyspnea, fever, cough, and tightness on 13 December 2020. In the Hospital she was admitted immediately after computed tomography (CT) imaging of her chest showed multiple and bilateral ground-glass opacities located in both subpleural spaces. Nasopharyngeal swab specimens were collected to detect severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) nucleic acid. The swab specimens were tested by real-time reverse transcriptase-polymerase chain reaction; a positive result was received 2 days later on 15 December 2020. Our patient was diagnosed with COVID-19, and not recommended treatment with Remdesivir for nephrotoxicity. She received 400 mg of moxifloxacin I.V daily for 3 days; O<sub>2</sub> Therapy; methylprednisolone three i.v. boluses of 200 mg; Tocilizumab was given in a single i.v. 400 mg dose; prophylactic enoxaparin was prescribed (she no presented thrombotic events).

**\*Correspondence to:** Weimer LE, National Center for Global Health, Istituto Superiore di Sanità, Rome, Italy; E-mail: liliana.weimer@iss.it

**Key words:** chronic kidney diseases, Sars-Cov-2, sarcomatous endothelial carcinoma

**Received:** March 02, 2021; **Accepted:** March 08, 2021; **Published:** March 11, 2021

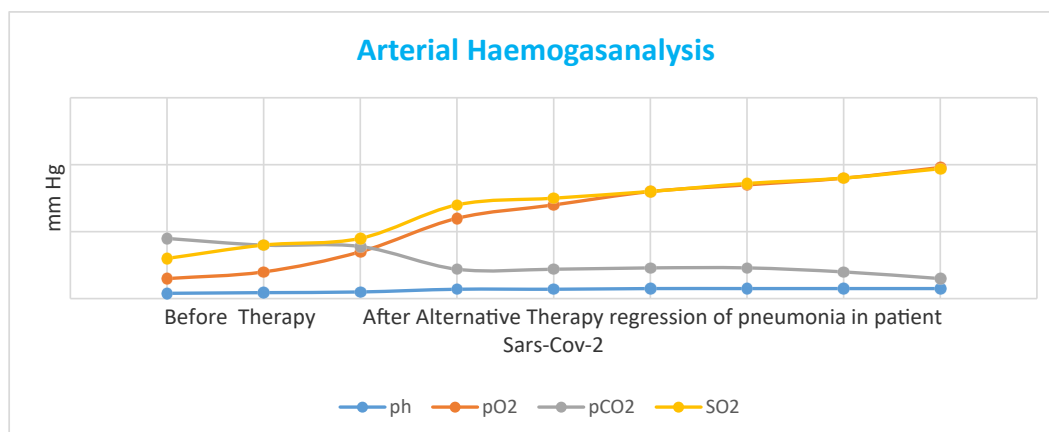


Figure 1. Arterial haemogasanalysis

Note: This figure show arterial haemogasanalysis before alternative therapy without antiviral agents for nephrotoxicity and after therapy with regression of pneumonia caused by Sars-Cov-2 in patient with chronic kidney disease.

The patient had a history of Chronic Kidney Disease (not recommended treatment with Remdesivir), which was diagnosed on 2 March 2012, previous cystectomy surgery for sarcomatous urothelial carcinoma and arterial hypertension. At that time, the patient showed minimal to moderate kidney function deterioration during admission, but the estimated glomerular filtration rate (eGFR) decline was lower than 20 mL/min; laboratory testing also showed proteinuria, hematuria, an increase of serum creatinine concentration (it is currently the most widely used screening test for the detection of renal impairment, but it has been shown to be a poor screening test, particularly in elderly patients), lymphopenia, high levels of C-reactive protein, IL-6, D-dimer, and procalcitonin were verified but without a clear pattern associated with severity.

On December 28, 2020, our patient was negative, and computed tomography (CT) have shown imaging of a complete resolution of bilateral areas of altered density a ground glass after treatment without Remdesivir (Figure 1).

## Discussion

The COVID-19 pandemic has greatly affected nephrology. Firstly, dialysis patients appear to be at increased risk for infection due to viral transmission next to an enhanced risk for mortality as compared to the general population, even in the face of an often apparently mild clinical presentation. Derangements in the innate and adaptive immune systems may be responsible for a reduced antiviral response, whereas chronic activation of the innate immune system and endothelial dysfunction provide a background for a more severe course. The presence of severe comorbidity, older age, and a reduction of organ reserve may lead to a rapid deterioration of the clinical situation of the patients in case of severe infection. Secondly, patients with COVID-19 are at increased risk of acute kidney injury (AKI), which is related to the severity of the clinical disease. The presence of AKI, and especially the need for renal replacement therapy (RRT), is associated with an increased risk of mortality. Acute kidney injury in COVID-19 has a multifactorial origin, in which direct viral invasion of kidney cells, activation of the renin-angiotensin aldosterone system, a hyperinflammatory response, hypercoagulability, and nonspecific factors such as hypotension and hypoxemia may be involved [4].

Our patient with chronic kidney diseases, hypertension and sarcomatous endothelial carcinoma responding to alternative therapy with regression of pneumonia caused by Sars-Cov-2 without antiviral agents (Remdesivir for nephrotoxicity). In addition, immunosuppressive

drugs such as cyclosporin and mycophenolic acid may be good candidates for therapeutic medicines against renal damage by SARS-CoV-2 [5,6], and specific inhibitors of IL-6 appear to be beneficial in severely infected cases.

Chronic kidney disease with or without dialysis patients should therefore be considered a risk group, both in terms of infection risk and outcome. Moreover, a high degree of clinical suspicion is needed as the clinical presentation in dialysis patients may be obscured because chronic dyspnea is common in dialysis patients whereas the febrile response may be blunted. However, in studies in which universal screening for Sars-Cov-2 was applied, a significant proportion of dialysis patients were asymptomatic positive. This allowed earlier adjustment and improved prognosis.

## Conclusion

We need prospective studies of treatment options and additional patient characteristics to further understand the variables associated with COVID-19-associated death in patients with Chronic kidney diseases. The high mortality observed in patients with this severe comorbidity highlights the importance of applying all measures to prevent infection and treat this complex situation as early as possible.

## References

1. Zhang X, Song K, Tong F, Fei M, Guo H, et al. (2020) First case of COVID-19 in a patient with multiple myeloma successfully treated with tocilizumab. *Blood Adv* 4: 1307–1310. [Crossref]
2. Gupta A, Madhavan MV, Sehgal K, Nair N, Mahajan S, et al. (2020) Extrapulmonary manifestations of COVID-19. *Nat Med* 26: 1017–1032. [Crossref]
3. Wang M, Xiong H, Chen H, Li Q, Ruan XZ. (2021) Renal Injury by SARS-CoV-2 Infection: A Systematic Review. *Kidney Dis* 7: 100–110.
4. Noris M, Benigni A, Remuzzi G (2020) The case of complement activation in COVID-19 multiorgan impact. *Kidney Int* 98: 314–322. [Crossref]
5. Kuba K, Imai Y, Rao S, Gao H, Guo F, et al. (2005) A crucial role of angiotensin converting enzyme 2 (ACE2) in SARS coronavirus-induced lung injury. *Nat Med* 11: 875–879. [Crossref]
6. Tanaka Y, Sato Y, Sasaki T (2013) Suppression of coronavirus replication by cyclophilin inhibitors. *Viruses* 5: 1250–1260. [Crossref]

**Copyright:** ©2021 Weimer LE. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.