

# Kidney Graft Loss Subsequent to Vascular Thrombosis Following Severe COVID-19 Infection – A Case Report

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**Abstract:** The coronavirus disease of 2019 (COVID-19) has pushed the world into a pandemic and global terrorism. As many people have acquired the virus and become ill, many phenomena and systemic manifestations of the disease have raised questions regarding its mechanism and long-term effects. Many studies have been conducted to understand the pathophysiology of coronavirus. Here, we describe the case of a 66 year old Saudi male who experienced pneumonia secondary to severe COVID-19 with subsequent loss of a kidney graft. In addition, we discuss the potential mechanisms underlying COVID-associated coagulopathy.

**Keywords:** kidney transplant, complications, COVID-19, thrombosis, graft loss, case report, acute kidney injury, AKI

## Introduction

The World Health Organization declared the rapidly spreading acute respiratory virus a cause of the 2020 global pandemic.<sup>1</sup> Coronavirus is a respiratory virus that originated in Wuhan, China in 2019, infecting mainly the lungs and other organs at a secondary and systematic level. It facilitates entry into cells by binding to angiotensin-converting enzyme 2 (ACE2), which is abundant on the surface of alveolar cells in the lung.<sup>2–5</sup>

Most patients present with varying degrees of flu-like symptoms, most commonly fever, cough, fatigue, and loss of taste or odor. On the other hand, a minority experience more severe diseases, such as dyspnea, chest pain, systemic complications, and death.<sup>1</sup> The pathophysiology and long-term effects of COVID-19 have yet to be completely understood and studied in depth. This report details the case of a kidney transplant recipient who developed severe COVID-19 pneumonia complicated by graft loss secondary to vascular thrombosis. This paper also discusses the possible theories of COVID-19 pathophysiology and its effect on coagulation.

## Case Report

A 66 year old Saudi male was transferred to our institution with severe COVID-19 associated pneumonia. Past medical history included hypertension, type 2 diabetes mellitus, dyslipidemia, chronic kidney disease managed with live-related donor transplantation, coronary artery disease, and heart failure with an ejection fraction of 35–40%. The patient had no history of tobacco use. There was no history of rejection episodes and baseline serum creatinine prior to his illness was 0.8 mg/dL (0.7–1.3 mg/dL).

On examination, the patient had a low-grade fever of 37.7°C, heart rate of 86 bpm, elevated respiratory rate of 24 breaths/min, blood pressure of 108/74 mmHg, and a reduced SpO<sub>2</sub> of 82%. Rhonchi, rales, and signs of fluid overload were also observed. The following blood tests were performed; platelets 181,000 mm<sup>3</sup> (140,000–400,000), serum creatinine 7.6 mg/dL (0.7–1.3), sodium 131 mEq/L (135–145), potassium 4.8 mEq/L (3.5–5.0).

Ultrasonography showed no parenchymal flow through the transplanted kidney with highly resistive Doppler waveforms of the feeding iliac artery, which was suggestive of vascular compromise. Computed tomography confirmed



**Figure 1** CT imaging showing infarcted kidney in the right lower quadrant with no sign of blood perfusion.

thrombosis in both the graft artery and vein as well as an infarcted non-enhancing transplant kidney located in the right iliac fossa (Figure 1). Renal biopsy confirmed Banff Type IIA cellular graft rejection. The patient was started on continuous renal replacement therapy, later switched to intermittent hemodialysis, and became dialysis dependent.

## Discussion

The COVID-19 virus infects the body through the respiratory system. It binds to ACE2 receptors on the surface of type II alveolar lung cells. These receptors are also found in many cells in the body, including the vascular endothelium and multiple renal cell types.<sup>2–5</sup> The renal system is believed to be particularly vulnerable because of its porous nature.<sup>6,7</sup>

Several studies have reported that coronaviruses injure the kidneys via both direct and indirect mechanisms. After the virus infects the pulmonary system, it gains access to the vascular system and eventually infects the vascular endothelial cells. Additionally, ACE2 receptors are found in the kidney cells. Once the virus enters the bloodstream and reaches the renal system, it directly infects the kidney. One study confirmed the presence of coronavirus in the renal glomerular cells, renal tubular epithelium, and renal podocytes.<sup>2,6</sup>

It has been reported that the coronavirus can overactivate the complement system, resulting in a more aggressive approach to eradicate the virus compared to other pathogens.<sup>2,3</sup> The virus stimulates the release of excessive pro-inflammatory mediators in a state known as cytokine storm. Cytokine storms have been linked to disease severity, hypoperfusion, the development of acute kidney injury, systemic inflammatory response syndrome, shock, and death.<sup>2,3</sup> Specifically, they bind to Toll-Like Receptors (TLRs) on the surface of cells, which stimulate the release of interleukin-1 (IL-1), interleukin-6 (IL-6), and tumour necrosis factor- $\alpha$  (TNF- $\alpha$ ). In turn, they increase the expression of ICAM-1, P-selectin, VCAM-1, MCP-1, and IL-8. These factors release reactive oxygen and nitrogen species, which are pro-inflammatory in of themselves.<sup>8,9</sup> They further contribute to thrombogenesis by stimulating platelet activation and aggregation, promoting neutrophil extracellular traps (NETs), and increasing the release and expression of tissue factors by monocytes and macrophages.<sup>10</sup> NETs are pro-coagulants, further promoting a hypercoagulable intravascular state, trapping and activating platelets.<sup>3,6,10</sup> On the other hand, oxidative stress can cause damage on a cellular and molecular level. They have been associated with injury to renal tissue directly, or to surrounding tissue which ultimately affects the kidneys. Reports show that reactive oxidative species can cause podocyte damage, reduced GFR, endothelial damage, and faster progression of CKD.<sup>11</sup>

Overall, the end result of the stated above processes results in positive feedback from each other, with the majority of released cells contributing to thrombogenesis through different pathways.<sup>3,10</sup> Additionally, blood viscosity increases in such inflammatory states, which can lead to further aggregation of coagulation factors in the endothelium, leading to stasis. This can lead to impaired oxygen delivery, poor nutrition, and vasoconstriction. These effects are more likely to

occur in the smaller vessels and capillaries.<sup>3</sup> It is believed that the complement and coagulation systems collaborate and further stimulate each other in patients with COVID-19, creating an aggressive cycle of increasing coagulation.

COVID-associated coagulopathy differs from other coagulation diseases. As such, it can easily be missed when a coagulation profile is performed. Several studies have reported that elevated D-dimer levels are the most common laboratory abnormality. Additionally, D-dimer levels are associated with poor prognosis, high risk of mechanical ventilation, disease severity, and death. Few studies have reported the involvement of other parameters, such as elevated Von Willebrand Factor, elevated fibrin degradation products, elevated Factor VIII, normal or mild platelet reduction, and prolonged prothrombin time.<sup>2–5,10</sup> In contrast, a study focusing on the complement system suggested that elevated C3a, C3/C3a ratio, and sC5b-9/C3 ratio on admission increased the risk of severe disease and thrombotic microangiopathies, with C3a being the most consistent abnormal parameter that correlates with severe disease.<sup>6</sup> Further studies are required to confirm the specificity and sensitivity of these parameters.

## Conclusion

In conclusion, this case highlights COVID-19's pro-thrombotic effects, which can affect the kidneys among other organs and may lead to graft loss in renal transplant recipients. Increasing awareness in the medical community is essential to allow for early intervention and improve patient outcomes.

## Acknowledgments

This study was approved by the IRB Review Board of Johns Hopkins Aramco Healthcare. An informed written consent was obtained from the patient for publication. The authors acknowledge the use of JHAH facilities for the research data utilized in this article. Opinions expressed in this article are those of the authors and not necessarily those of JHAH.

## Disclosure

The authors declare no conflicts of interest in this work.

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