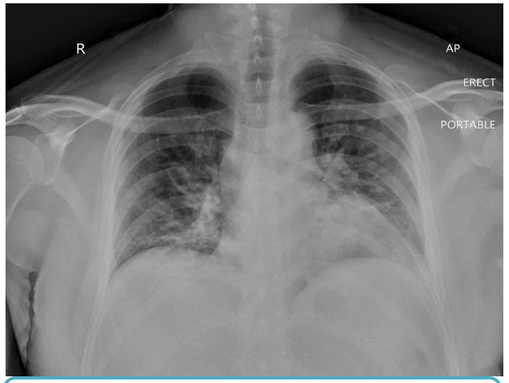
A Puzzling Presentation of COVID-19: Cardiac and Renal Failure Without Overt Respiratory Failure.

G Madders, M Murphy, H Tuite
Department of Infectious Disease , University Hospital Galway

Background

The new Coronavirus-disease-19 (COVID-19) caused by Severe Acute Respiratory Syndrome Coronavirus2 (Sars-CoV2) has led to a world health emergency. As the name of the virus would suggest, this disease preferentially targets the lungs but other organ involvement has been widely described. Direct viral injury via Angiotensin converting enzyme 2 (ACE2), immune dysregulation and hypercoagulability are thought to be involved in disease pathogenesis(1). This case demonstrates the multiorgan impact of COVID-19 and may represent a case of cytokine mediated cardiac and renal dysfunction.

Fig 1: Chest Xray showing bilateral lower lobe infiltrates



The Case

A 37-year-old male presented with persistent fevers, lethargy and myalgia. He was mildly hypoxic requiring 2litres of oxygen. A swab was positive for COVID-19. Chest X-ray showed bilateral lower lobe infiltrates in keeping with atypical pneumonia. (Figure1). Bloods revealed evidence of hyperinflammation with a CRP of 114 and a Ferritin of 66.

Due to a persistent tachycardia, an echocardiogram was carried out which showed an ejection fraction (EF) of 10- 15%. Interestingly, there was no troponin elevation or electrocardiogram abnormality, and the patient was euvolemic. Shortly after admission, he became oliguric with a rapidly progressive AKI. The aetiology of this was unclear as he had no overt hypotension and was not on any nephrotoxins. He commenced haemodialysis and gradually recovered renal function over the coming weeks with supportive cares.

In the differential, we considered a COVID-induced myocarditis and a Takutsubo cardiomyopathy. The factors that pointed away from these were the normal troponin making a myocarditis unlikely and the global rather than regional hypokinesis on echo.

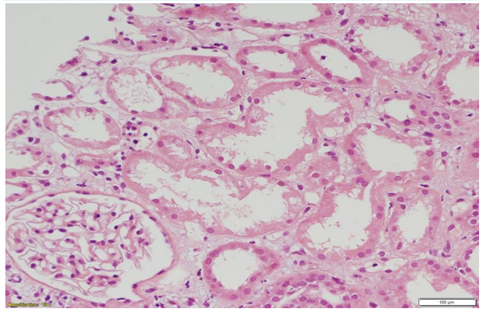
In terms of the differential for severe AKI, we considered whether direct renal injury secondary to SarsCoV2 or a microangiopathic process secondary to COVID could be responsible. Finally, we proposed that an exaggerated immune response to COVID resulting in a cytokine-mediated tubular injury could explain the acute AKI. We concluded that a renal biopsy was necessary to aid diagnosis. (Figure 2)

Renal biopsy showed tubular injury without evidence of microangiopathy. Electron microscopy out-ruled direct viral cytopathy and it was felt that ATN may have been a result of cytokine mediated injury. Serial echo revealed an improvement in EF before discharge. Cardiac MR definitively excluded myocarditis and showed changes in keeping with a post-infectious cardiomyopathy.

Conclusion

This case encapsulates the multiorgan impact of Covid-19 and highlights that severe disease can spare the lungs. Cell infectivity by SarsCoV2 depends on the expression of ACE2 receptors. One distinct mechanism of injury to consider is cytokine-mediated (2). Cytokine-mediated myocardial depression syndrome as well as cytokine-mediated renal tubular injury were described in this case. These are important entities to recognize as they are potentially reversible but can have long term consequences. Further studies are necessary to better understand disease pathology and its chronic complications so we can prepare for what could be another facet of the pandemic.

Fig 2: H&E stain showing dilatation of proximal tubules and flattening of tubular epithelium without glomerular injury.



References

1. Daniel Batlle et al “Acute Kidney Injury in COVID-19: Emerging Evidence of a Distinct Pathophysiology”, JASN July 2020, 31 (7) 1380-1383
2. Li Yang et al “COVID-19: Immunopathogenesis and Immunotherapeutics”, Sig Transduct Target Ther 5, 128 (2020).