

Incomplete trifascicular block with Mobitz type II AV block in the setting of severe COVID-19: a case and literature review

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Introduction

The novel coronavirus of 2019 (SARS-CoV-2) while primarily causing pulmonary complications, manifests cardiac complications in almost a quarter of patients. Arrhythmia (16.7%) or acute cardiac injury (7.2%) are the most common presentations. [1]

Case Presentation

A 74-year-old female with a history of diabetes, chest pain, and shortness of breath for two days. She was hypoxic to an oxygen saturation of 60% in the emergency department, requiring BiPAP to maintain saturations. Chest X-ray demonstrated bilateral hazy opacities suspicious for viral pneumonia. COVID-19 was confirmed. RBBB with LAFB was noted on admission ECG. Cardiac enzymes and BNP were within normal limits. After noting frequent pauses on telemetry, a repeat ECG was performed which demonstrated RBBB with LPFB as well as second-degree AV block (Mobitz Type II). Transcutaneous pacing pads were placed, and atropine was placed at the bedside. Cardiac enzymes remained negative. IL-6 levels were elevated at 159 pg/mL. Hydroxychloroquine was deferred due to the patient's arrhythmia and prolonged QTc. Tocilizumab was deferred due to the patient's age. The patient's oxygen requirements and mental status continued to worsen. She continued to desaturate despite maximal BiPAP therapy and eventually passed away.

Figure 1. Admission ECG demonstrating bifascicular block (RBBB with LAFB) and prolonged QTc

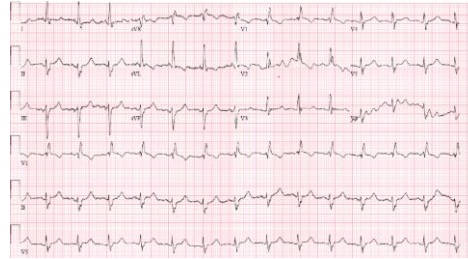
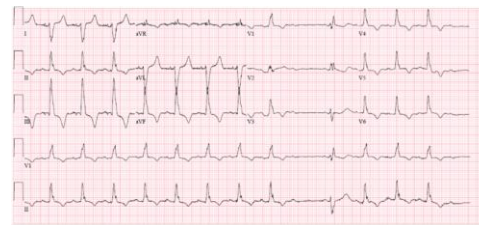


Figure 2. Subsequent ECG demonstrating altered bifascicular block (RBBB with LFPB), Second-degree AV block (Mobitz Type II) and prolonged QTc



Discussion

The diagnosis of incomplete trifascicular block was made given evidence of conducting disease in all three fascicles. Along with the presence of Mobitz Type II, this suggests disease below the level of the AV-node. [2,3] Cardiac disease in COVID-19 manifests most commonly as arrhythmia and acute cardiac injury, the etiology of which is likely multifactorial. [1] Elevated sympathetic tone causing increased myocardial oxygen consumption in critical illness can exacerbate existing CAD. Myocarditis has been suspected in several cases and studies. [4-7] Hydroxychloroquine which has seen increased use during the COVID-19 pandemic has historically been implicated in several cases of cardiomyopathy and arrhythmia in addition to its QT-prolonging effects. [8-12] Hypoxia coupled with increased myocardial oxygen consumption in the setting of COVID-19 are the likely precipitating factors of our patient's arrhythmia.

Conclusion

Cardiac involvement in COVID-19, whether caused primarily by the virus, secondary to its clinical sequelae, or even due to its treatment cannot be ignored. Further high-quality research is needed to clarify the cardiac pathophysiology. Thorough cardiac exams with electrocardiographic correlation should be performed on all patients with COVID-19. Clinicians should not hesitate to consult cardiovascular services in the event of noted abnormality.

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