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NEW LEFT BUNDLE BRANCH BLOCK IN A PATIENT WITH COVID-19- CASE REPORT

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Contribution
AJK conceived the idea and AJK, FAB, HJM, and HJM collected data and drafting and finalized the manuscript.

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ABSTRACT

As more cardiovascular manifestations of COVID-19 are experienced, the body of knowledge related to the effect of COVID-19 on the cardiovascular system is expanding.

We are presenting a unique case of acute coronary syndrome in a COVID-19 positive patient, who presented with an ECG consistent with left bundle branch block.

The patient was considered for a percutaneous intervention versus fibrinolytic therapy. However, the patient returned to a normal QRS with no ischemic features. His troponin I was raised but an echocardiogram showed a normal ejection fraction without any regional wall motion abnormality. A watchful approach in COVID-19 cases may be a reasonable option.

Keywords: COVID-19, left bundle branch block, Case report, Echocardiogram, Conservative therapy.

INTRODUCTION

The association between COVID-19 and pathology in the cardiovascular system is unclear. Patients with SARS CoC2 can develop different symptoms related to the heart.[1] We describe a patients revealing left bundle branch block as manifestation of acute coronary syndrome in COVID-19 patients, which requires a conservative approach.

The patient managed with pharmacologic therapy and with spontaneous normalization of ECG. The issue of ACS-like consequences of COVID-19 is a hot-topic for current cardiology physicians. We describe the clinical presentation of patients, with spontaneous disappearance, and remind to follow the AHA/ACC Consensus Statement guidelines when in doubt about the balance between risks and benefits of an invasive coronary revascularization.[2]

CASE REPORT

A 56 years-old-male patient, diagnosed with diabetes mellitus, hypertension, chronic kidney disease stage II, and chronic obstructive airway disease, was diagnosed with COVID-19, presented with dysuria and frequency associated with a loose motion. On admission he had stable vital signs and his examination was unremarkable. He was managed with supportive therapy. He already used aspirin and was prescribed enoxaparin 80 mg OD. During his admission, he developed classical angina pain, retrosternal with radiation to his arm and associated with sweating. His pain was not related to breathing or coughing. No heartburn reported. On clinical examination, his pulse was 87 sinus rhythm, blood pressure 145/83, normal jugular venous pressure. He had normal first and second heart sound with fourth heart sound. Bilateral coarse crepitation noted, mainly on the right side.

The immediate ECG indicated a new left bundle branch block (LBBB) (Figure 1). He was immediately transferred to the Cardiac Care Unit (CCU) and he received a loading dose of ticagelor 180 mg OD, atorvastatin was on 80 mg once daily, Metoprolol increased to 100 mg once daily and continued on Nifedipine retard 30 mg once daily. A Chest X-ray showed the development of a peripheral area of airspace opacification in the right mid zone, with pulmonary congestion (Figure 2).

Figure 1: ECG showing new onset left bundle bransh block

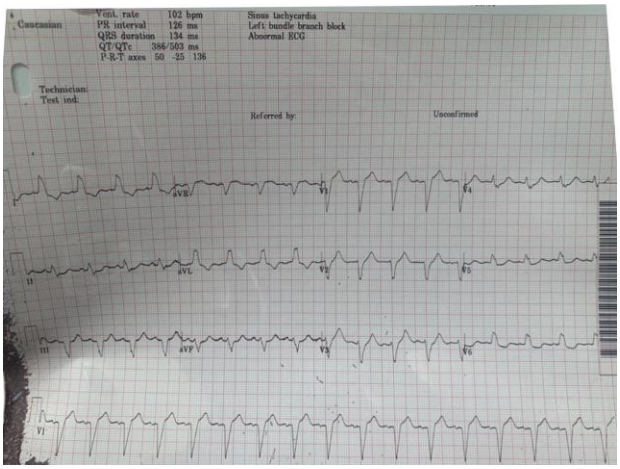
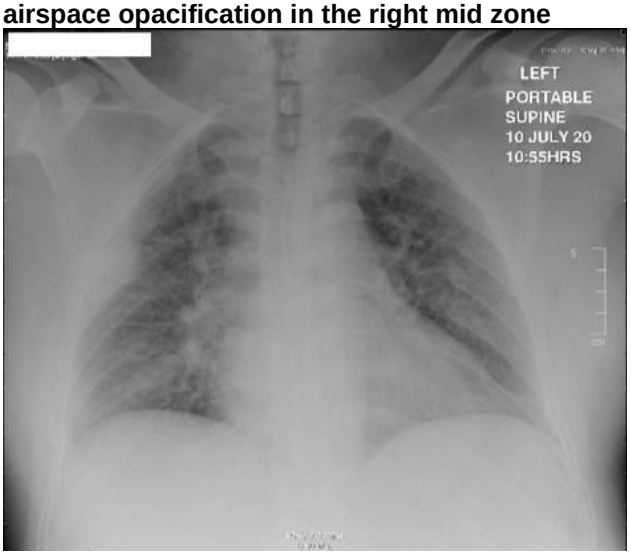


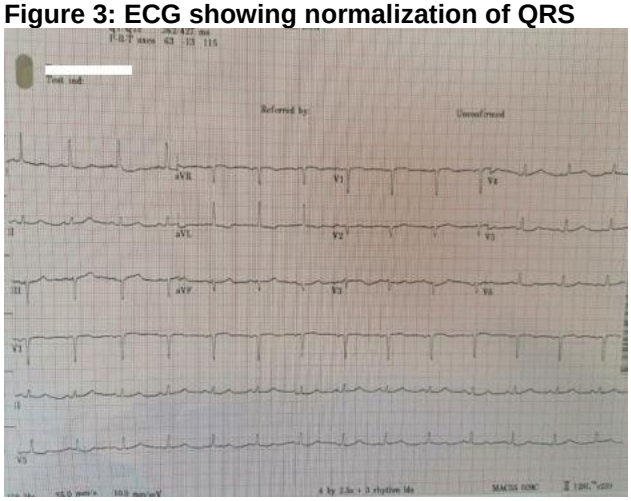
Figure 2: Chest X-ray showing peripheral area of airspace opacification in the right mid zone



His laboratory parameters are summarized in Table 1. When he arrived at CCU, his pain subsided. The team considered his management options in terms of taking him to the catheter laboratory with the risk to the staff and contrast induced nephropathy versus fibrinolytic therapy. The echocardiogram did not show any regional wall motion abnormality and his left ventricle function was normal. A repeat ECG

indicated that the LBBB returned to normal sinus rhythm (Figure 3).

Figure 3: ECG showing normalization of QRS



His medications were later changed to subcutaneous heparin due to a decline in the creatinine clearance. His Troponin I declined as shown in Table 1. He remained pain free and was discharged with an outpatient non-invasive assessment for residual ischemia.

Table 1: Showing the laboratory results

	During ACS	Baseline	Reference value
D-Dimers	0.40	0.24	0.0- 0.5 mg/L
Creatinine	269	254	62.0 - 115.0 umol/L
Troponin I	936	421	0.020 - 0.060 ug/L
ESR	120	24	0 - 15 mm/Hr
Procalcitonin	0.11	0.11	0.00 - 0.10 ng/ml
LDH	182	167	85 – 227
INR	1.1	1.1	0.9 - 1.2
WBC	5.4	5.6	4.0 - 10.0 10^9/L
Lymphocyte	1.92	1.01	1.5-4.0 10^9/L
Platelet Count	224	203	150 - 450 10^9/L
Hemoglobin	8.6	8.8	13.0 - 17.0 g/dL
C-reactive protein	18.8	5.2	0.00 – 5.0 mg/L
Fibrinogen	2.9	4.3	2 - 4 g/L
Glucose	9.7	7.0	-

DISCUSSION

Literature describes a wide spectrum of cardiac involvement in COVID-19.[1] A transient ST elevation was recognised in two cases, but no report has yet been published regarding a new LBBB.[3,4] The knowledge regarding this rapidly spreading virus is growing and different observations may improve our management strategy.

Pros and cons of a percutaneous intervention versus fibrinolytic therapy were discussed as he had both renal impairment and anemia, in addition to the concern about the potential risk for the cardiac catheterization laboratory and personnel.

Cardiac involvement in COVID-19 is complex. Although it is a single case, it highlights the importance of a careful assessment of possible cardiac involvement in the clinical decision making of the patient affected by COVID 19 disease.

It also highlights that the pathology is complex as well as the hypercoagulable state of the disease. The current patient was using both aspirin and low molecular weight heparin but still developed acute coronary syndrome. Different pathologies have been described. Microthrombi is a major factor associated with the different degrees of direct endothelial or vascular injury, coronary spasm, plaque rupture, hypoxic damage or cytokine storm.[5]

The autopsy series at the Mount Sinai Hospital demonstrated blood clots in the lungs, in addition to multiple organs, such as the brain, kidney, and liver.[5,6] These findings reflect the extensive endothelial damage, which correlate with the activation of the coagulation cascade and the elevation of inflammatory blood markers. Immune system activation, in addition to immunometabolism alterations may result in plaque instability, contributing to the development of acute coronary events. However, a type 2 myocardial infarction, related to the respiratory infection in the context of anemia, is still a possibility.[5]

A watchful approach, and the use of other diagnostic modalities such as an echocardiography, may be beneficial. It should be noted however, that 50% of the patients with COVID-19, have echo abnormalities.[2,7]

In Conclusion: A left bundle branch block is another manifestation of acute coronary syndrome in COVID-19 patients, which requires a conservative

approach. The complexity of the disease surpasses the use of antiplatelet and heparin therapy. Research related to an appropriate anticoagulation method is warranted.

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