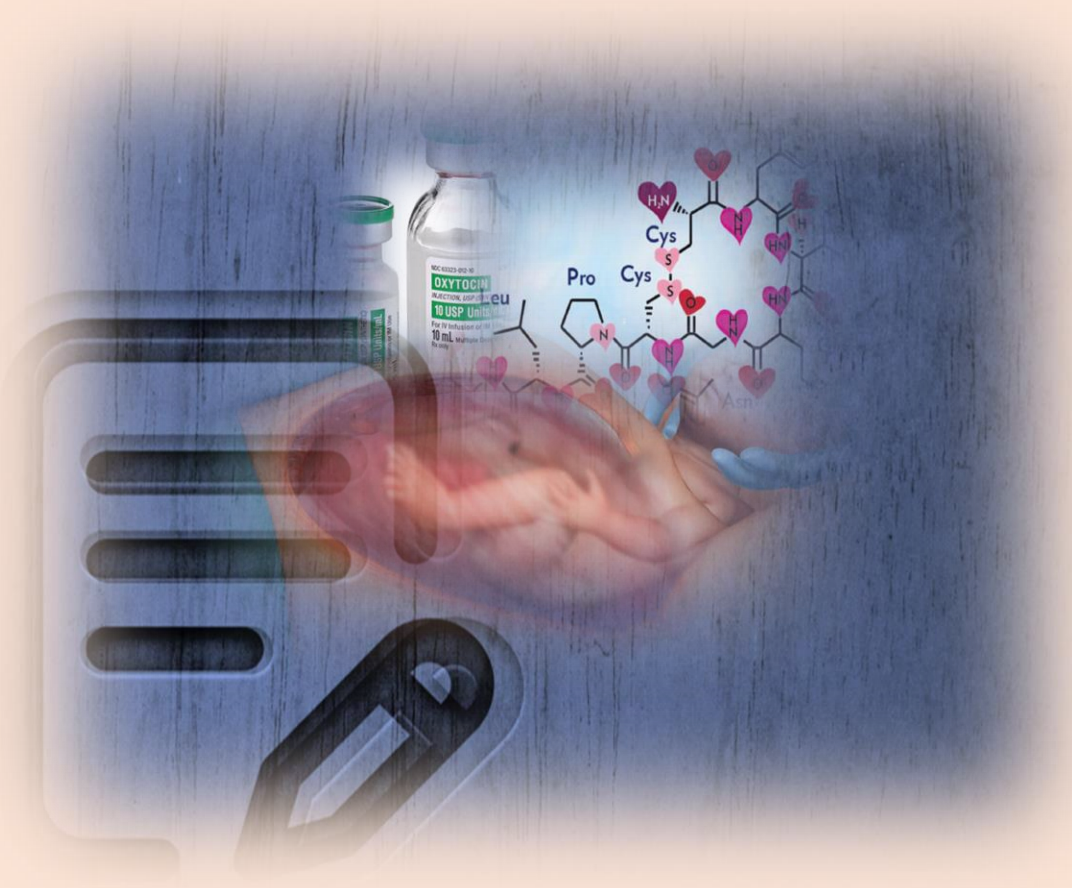
The Scientific Journal of Medical Scholar

SJMS

Volume 1, Issue 5, September 2022

https://realpub.org/index.php/sjms

Online ISSN: 2833-3772



The Scientific Journal of Medical Scholar

Available online at Journal Website
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Subject (Medicine)

Case Report

Complete Heart Block in a Patient with COVID-19 Pneumonia

Azhar Al-Shaibany [1]; Shah Shabahat [1]; Aqeel Alameer [2]

[1] Department of Medicine/Endocrinology unit, Our Lady's Hospital, Navan, Co Meath, Ireland.
[2] Department of Radiology, Ninewells hospital and Medical School, Dundee, United Kingdom.

Article information

Submitted: July, 7th, 2022

Accepted: August, 30th, 2022

Published: September, 1st, 2022

DOI: **10**.55675/sjms.v1i5.19

Citation: Al-Shaibany A, Shabahat S, Alameer A. Management of Unstable Per-trochanteric Fracture in Old Patient by Primary Prosthetic Replacement. SJMS 2022; 1 (5): 141-145. DOI: 10.55675/sjms.v1i5.19

ABSTRACT

There are multiple reports linking acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and arrhythmias (mainly bradyarrhythmias). The sinus tachycardia is commonly seen in patients with SARS-CoV-2. However, some SARS-CoV-2 patients develop heart block which might consequently lead to cardiac arrest.

Here, we presented a case of a 51-years-old male patient, with no history of cardiovascular disease. He presented to our county hospital with fever, shortness of breath, lethargy and dizziness on a background of asthma. The patient was found to have COVID-19 pneumonia (confirmed by polymerase chain reaction). The initial electrocardiogram (ECG) showed third degree heart block. The patient was subsequently treated with antibiotics, supportive care and permanent pacemaker placement. Patient was discharged home in stable condition and followed up by cardiology clinic.

Keywords: COVID-19; Arrhythmia; Pneumonia, pacemaker; SARS.

* Corresponding author
Email: azharalshaibany@gmail.com

INTRODUCTION

Several studies have linked extra pulmonary manifestations to SARS-CoV-2 infection [1]. This fact is likely due to the cumulative effects of the super inflammatory response of the body and the presence of angiotensin-converting enzyme 2 (ACE2) cellular receptor that SARS-CoV-2 uses for cell entry in major organs [2]. Of all systems affected by the virus, possibly the commonest extra-pulmonary consequences can be observed in the cardiovascular system. These complications include myocardial injury, cardiomyopathy, acute coronary syndrome, cardiogenic shock and acute cor-pulmonale [3, 4]. Data from Wuhan, China demonstrates that myocardial injury is s common finding present in 19.7% of patients and it is associated with further complications and higher mortality rates [5]. It's clearly understood that SARS-CoV-2 is responsible for multiple cardiac manifestations. The most common brady-arrhythmia associated with SARS CoV-2 reported worldwide is sinus bradycardia and complete heart block [5, 6]. SARS-CoV-2 cardiac manifestations leading to bradyarrhythmia and different degree of heart blocks that are poorly understood. Here, we discuss a case of irreversible bradycardia resulting from complete heart block in a patient with COVID-19 infection that warranted pacemaker insertion.

Description of the case:

A 59 years old male with a background history of bronchial asthma on inhalers, no known drug allergy, independent and non-smoker. No family history of any medical problems. Patient was brought in by ambulance with breathlessness, fever, feeling unwell, lethargy, poor appetite and dizziness. Patient was looking after his mother who was diagnosed with COVID 19 infection two weeks ago. On examination, Patient was pale and sweaty. Blood pressure 150/77mmHg, Pulse rate 38 beats per minute and irregular. Temperature 38.9 Celsius, O_2 saturation of 93% on room air and 98% on 2 litres of oxygen with nasal mask, respiratory rate was 22 /minute. On examination, patient had bilateral crackles more severe on the right side. Abdomen was soft, non-tender and normal neurological exam. Patient's biochemical test results are shown in table (1).

Table (1): Biochemical test results on admission

Test	Value	Normal reference range
Haemoglobin (g/dl)	13.6	13.2-16.6 for mean, and 11.6-15 for women
WBCs x 10^9/L	8.8	4.5 – 11
Platelets x 10^9/L	441	150-450
C-reactive protein (CRP) (mg/L)	58	< 1
Urea (mmol/L)	4.8	3.57 - 7.14
Creatinine (µmol/L)	73	61.9 to 114.9 for men, and 53 to 97.2 for women
Sodium (mmol/L)	128	136-145
Potassium (mmol/L)	4.2	3.6 - 5.2
D-Dimer (ng/ml)	410	< 250 ng/mL
Lactate dehydrogenase (LDH) (U/L)	273	100–190 U/L for adults
Creatine Kinase (CK) (U/L)	50	20-200
Thyroid stimulating hormone (TSH) (µIU/L)	1.07	0.4 to 4.0 mIU/L
Free T4 (pmol/L)	14.4	12-30
Glycated haemoglobin (HbA1c) (mmol/mol)	39	<48mmol/mol
Blood and urine cultures	No growth after 5 days	
COVID-19 PCR	Positive on day 1, 3 and Negative on day 7 of admission.	
pH	7.45	7.38- 7.42
PO2 (kPa)	10.1	10.5-13.5
PCO2 (kPa)	4.35	5.1- 5.6
O2 saturation	96.4 %	94-100
cHCO3 (mmol/l)	25.5	21-29
Lactate (mmol/l)	1.1	0.5- 1.0
Alanine transaminase (ALT) (U/l)	31	7 - 56
Gamma-glutamyl transferase (GGT) (U/l)	104	5-40
Alkaline phosphatase (ALP) (U/l)	60	30-120
Albumin (g/l)	32	35-52
Protein (g/l)	61	60-83
Amylase (U/l)	100	30-110
Corrected calcium (mmol/l)	2.22	2.25-2.625
Magnesium(mmol/l)	0.8	0.65 to 1.05
Phosphate (mmol/l)	0.75	1.12- 1.45
Troponin (ng/l)	17	Up to 14 ng/l
INR	1.1	0.8 – 1.1
Ferritin (ng/ml)	1533	12-300 for adult male, and 12-150 for adult female
Blood glucose (mmol/l)	6.3	4.0 - 5.4

Patient's initial ECG showed a 3rd degree heart block (Figure 1). A bedside echocardiogram showed no structural wall abnormalities, normal ejection fraction and no segmental wall motion abnormalities. Patient's chest radiograph showed findings consistent with SARS-CoV-2 pneumonia (Figure 2).

The patient was treated using the sepsis pathway and SARS-CoV-2 protocol and given antibiotics along with other supportive measures. Patient was transferred to telemetry isolation bed in CCU. After admission to CCU, patient was seen by cardiologist who advised to monitor and to keep isoprenaline infusion on standby if needed. After stabilization, patient was sent for permanent pacemaker placement. During his stay in CCU, the patient was doing well; fever subsided 3 days later, inflammatory markers trended down and no inotropic support was needed as heart rate was between 30-50 beats per minute (Figure 3).

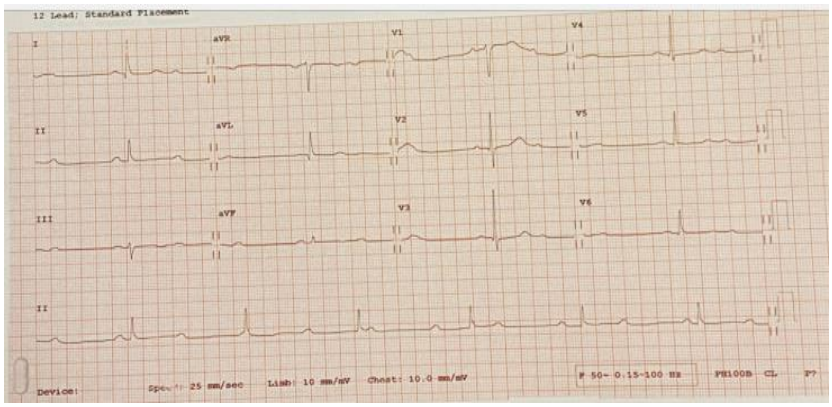

Figure (1): Patient's initial ECG

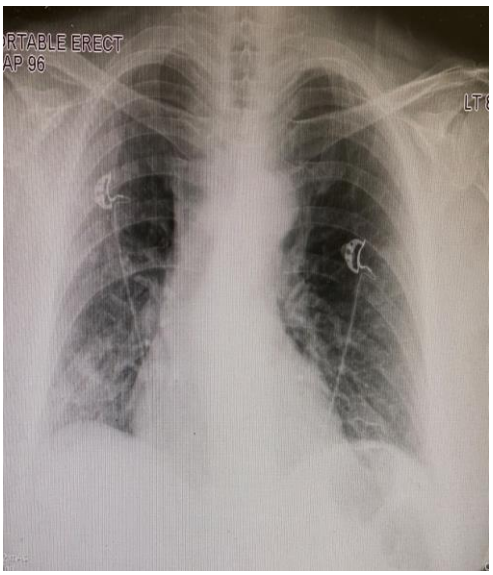

Figure (2): AP portable chest radiograph

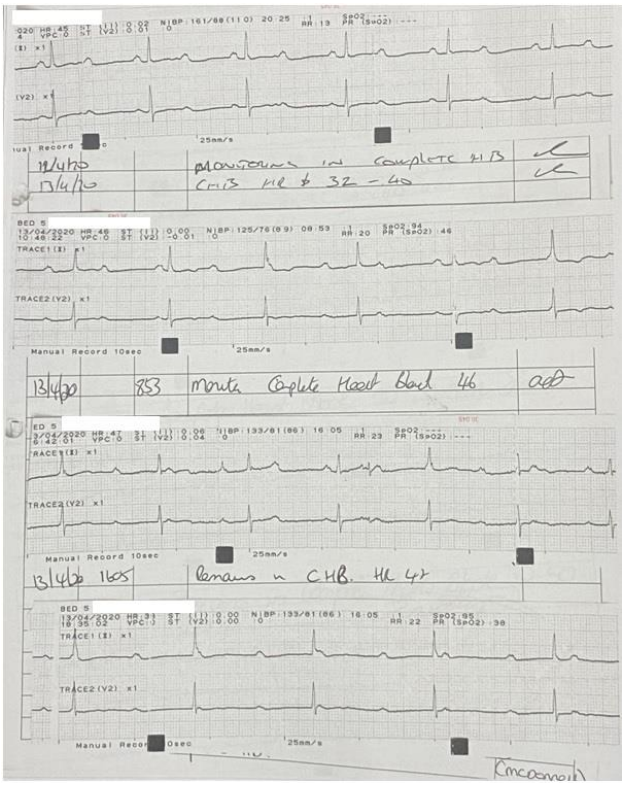

Figure (3): CCU telemetry record

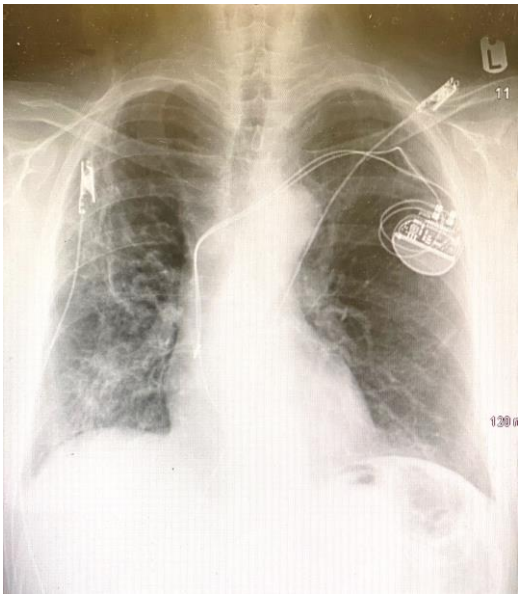

Figure (4): Chest radiograph showing PPM with no complications identified

On day 7 of admission the 3rd throat swab was negative; patient was transferred to catheterisation unit for permanent pacemaker (PPM) placement. After PPM insertion (Figure 4), patient was kept for monitoring in CCU for 24 hours with stable hemodynamic state. Heart rate stabilized after PPM (Figure 5).

Forty-eight hours later the patient was reviewed by cardiology and discharged home with follow up PPM check after 4-6 weeks. Currently the patient stable and under follow up with cardiology every 6-12 months.

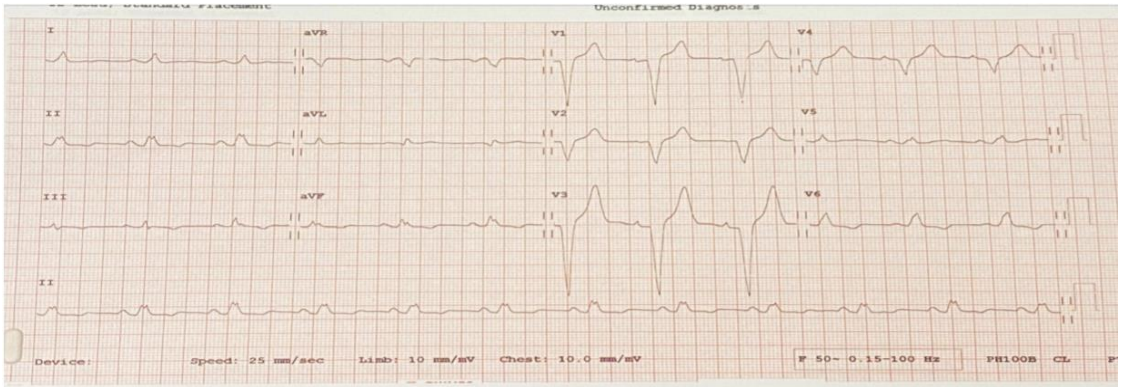

Figure (5): Post-PPM-ECG

DISCUSSION

Conductive cardiac abnormalities commonly occur in critically ill patients. Wide range of arrhythmias have been documented in SARS-CoV-2 patients and sinus bradycardia has been frequently described [6].

Atrioventricular (AV) block may account for up to 12% of arrhythmias seen in patients with SARS-CoV-2 [7]. Possible reversible causes of AV block include metabolic derangements, enhanced vagal stimulation due to pain, sleep, manipulation of the carotid sinus during usual nursing activities or side effects of medications. These multifactorial mechanisms tend to lead to intermittent atrioventricular (AV) block of the first- or second-degree. Higher-degree of AV block as described in the current case suggest a different pathological mechanisms of AV conduction disturbance [8]. Possible causes of myocardial injury could lead to AV block in patients with SARS-CoV-2 include, but not limited to myocarditis, hypoxic ischemic injury, stress (Takotsubo) cardiomyopathy, ischemic injury due to cardiac microvascular damage, coronary artery disease, right heart strain (acute cor-pulmonale) and systemic inflammatory response syndrome [5, 9-11].

The pathogenesis of dysrhythmia remains uncertain; different proposed mechanisms include increased vagal tone, direct effects of SARS-CoV-2 infection on the conduction system, or even SARS-CoV-2 myocarditis [12]. Although several etiologies of AV block related to SARS-CoV-2- have been described in patients with preserved ventricular function and/or normal cardiac biomarkers [13], it is possible that these patients have subclinical myocardial inflammation. In one such patient, cardiac magnetic resonance imaging (MRI) revealed edema of the ventricular wall suggestive of myocarditis despite no evidence clinical evidence of myocardial injury [14].

Additionally, heart block seen in the usual setting of acute infection might resolve frequently without intervention. Patients with SARS-CoV-2 and persistent AV block were directly treated with standard-of care pacemaker placement and outpatient follow-up [15]. Nevertheless, heart block in patients with SARS-CoV-2 has been pointed to be one of the poor prognostic signs in SARS-CoV-2 patients [8]**.** The treatment is still according to advanced cardiac life support (ACLS) guidelines for symptomatic bradycardia. According to the (ESC/AHA; European Society of Cardiology/American Heart Association) guidelines, if an advanced degree of heart block is found (e.g., Mobitz type II and higher) or if the bradycardia is associated with hemodynamic instability or is symptomatic, this became very unstable. Thus, temporary pacing should be performed to guard against potential deterioration with subsequent hemodynamic instability or cardiac arrest [16]. Our patient was hemodynamically stable and the AV block was irreversible, thus the patient was a candidate for permanent pacemaker.

SARS-CoV-2 pandemic has been expanding and can affect essential body systems apart from the respiratory system; it had been reported with a wide range of cardiac presentation in multiple patterns [17].

To sum up, in the absence of other metabolic and pharmacological causes of conductive heart disease in context to SARS-CoV-2, possible causes of AV block in patients with SARS-CoV-2 include myocarditis, hypoxic injury, stress cardiomyopathy, ischemic injury caused by cardiac microvascular damage or coronary artery disease, right heart strain (acute cor-pulmonale), and systemic inflammatory response syndrome. In this case, AV block may be linked to hypoxic injury or stress cardiomyopathy, ischemic injury caused by cardiac microvascular damage in view of negative troponin and normal echocardiography or subclinical myocarditis. Because the patient presented at early days of SARS-CoV-2 pandemic, cardiac MRI wasn't performed. Moreover, future extensive studies are needed to determine the relation between the two conditions.

Conflict of interest and financial disclosure

None

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SJMS

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https://realpub.org/index.php/sjms

Online ISSN: 2833-3772