# Asystole in a COVID-19 patient without systemic illness: a case report

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#### Abstract

There is growing evidence that patients with severe systemic illness from coronavirus disease 2019 (COVID-19) are at risk for developing a variety of cardiac arrhythmias. Less is known about patients with milder symptoms. Here, we report on the case of a 62-year-old male, admitted to the hospital following an episode of syncope, who experienced multiple episodes of cardiac arrest due to asystole lasting up to 30 seconds. History revealed a recent asymptomatic COVID-19 infection, and recurrent episodes of prolonged asystole necessitated permanent pacemaker placement. To our knowledge, this is the first report of an asymptomatic COVID-19 patient experiencing prolonged asystole. Cardiac arrhythmias in asymptomatic or oligosymptomatic COVID-19 patients may be underestimated.

## INTRODUCTION

As coronavirus disease 2019 (COVID-19) continues to spread and mutate into new and increasingly infectious strains, it remains vital for researchers to continue to improve their understanding of the pathophysiology of symptoms caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection. While the virus' effects on the lungs and respiratory system are well described, less is known about the mechanisms of morbidity and mortality in other bodily systems.

There is increasing evidence that SARS-CoV-2 infection can lead to cardiovascular complications and arrhythmias [1, 2]. The most common arrhythmias appear to be sinus tachycardia and atrial fibrillation, though bradyarrhythmias have also been described [1]. While medical literature frequently notes arrhythmia is a concern for patients with systemic illness, less is known about patients with milder symptoms. Here we present the case of an asymptomatic COVID-19 patient who developed a life-threatening arrhythmia.

## CASE REPORT

A 62-year-old white male with a history of type 2 diabetes and hypertension was brought to the emergency department after a motor vehicle accident. While driving the car, the patient had a sudden loss of consciousness.



Figure 1. Chest X-ray. Chest X-ray at admission showing clear lungs bilaterally.

He denied preceding chest pain, shortness of breath, palpitations, vision changes, lightheadedness, incontinence, prior syncopal episodes or history of seizures. On admission, blood pressure was 134/72 mmHg and heart rate was 80 beats per minute. Initial laboratory results were remarkable for leukocytosis (20500/mm<sup>3</sup>), elevated lactate (2.8 mmol/l) and creatine kinase (854  $\mu$ /l). Electrolytes, serial troponin-I, creatine kinase myocardial band and alcohol levels were within normal limits. Chest X-ray showed no infiltrates (Fig. 1). Initial

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Figure 2. Asystole telemetry. Telemetry (A, C) and heart rate trend (B, D) during hospitalization on Day 2 and Day 3. The Day 2 telemetry (A) shows the detail of an asystole episode (from red circle) without any escape beats for 29.5 seconds. The Day 2 heart rate (B) trend shows episodes of sinus arrest with prolonged pauses without a preceding change in heart rate (red arrows). On Day 3 (after temporary pacing wire placed), the telemetry (C) shows episode of asystole requiring pacing.

electrocardiogram demonstrated normal sinus rhythm, normal intervals and no evidence of hypertrophy or ischemic changes. The echocardiogram showed normal chamber sizes, left ventricular ejection fraction of 65% and no wall motion abnormalities.

During the first 24 hours of admission, the patient experienced three episodes of cardiac arrest due to asystole lasting up to 30 seconds. Telemetry showed sinus arrest and asystole without preceding changes in heart rate, PR intervals, or escape beats (Fig. 2). Repeat troponin-I tests were negative. Further history revealed that the patient had a positive reverse transcription polymerase chain reaction (RT-PCR) screening test for SARS-CoV-2 8 days prior but had experienced no viral symptoms. Repeat RT-PCR SARS-CoV-2 testing during this admission was positive. Thyroid stimulating

**Table 1.** Timeline of patient's hospitalization and initialfollow-up appointment

Time	Progress
Day –8	Patient screened positive for COVID-19
Day 1	Patient presented to emergency room
Day 2	Three episodes of asystole. Cardiology consulted. Emergent temporary pacemaker wire placed
Day 3	14 episodes of asystole requiring pacing
Day 5	Temporary wire lost capture
Day 6	Permanent pacemaker implanted with continued pacing overnight
Day 7 Day 19	Patient discharged from the hospital Follow-up appointment showing pacing burden of <0.1%
Day 7 Day 19	Patient discharged from the hospital Follow-up appointment showing pacing burden of <0.19

hormone was normal. Borrelia burgdorferi antibodies were negative. Cardiac contusion was ruled out by negative troponin and normal echocardiogram. An urgent temporary pacing wire was placed, and concurrent left heart catheterization showed non-obstructive coronary artery disease. The patient had 14 additional episodes of asystole lasting 20-30 seconds requiring pacing in the first 24 hours after wire placement (Fig. 2). While the initial plan was to assess for myocarditis with cardiac magnetic resonance imaging (MRI) during this admission, the patient was deemed too unstable for the examination. Within 48 hours of placement, the temporary wire failed to capture. Therefore, he underwent permanent pacemaker implantation the following day (Table 1). During a follow-up appointment 14 days after, the pacing burden was <0.1%. The patient was scheduled for an outpatient cardiac MRI but did not present to the visit and was subsequently lost to followup.

## DISCUSSION

Arrhythmia is an increasingly recognized symptom of COVID-19, with one cohort study noting a 1% incidence of bradyarrhythmias in 700 hospitalized patients [2]. Currently, most described arrhythmias occurred among critically ill patients, leading some experts conclude that arrhythmias and cardiac arrests are consequences of systemic illnesses rather than SARS-CoV-2 infection [2]. We report a unique and severe form of bradyarrhythmia in an asymptomatic patient with COVID-19. We performed a thorough workup and did not find any usual causes of asystole.

To date, there are few reports of COVID-19 patients experiencing prolonged asystole. One report described an 83-year-old woman with COVID-19 pneumonia and elevated troponin-I of 0.76 ng/ml [3]. She developed sinus arrest with pauses up to 5.3 seconds along with tachyarrhythmias. Another report described a young male with acute lung injury due to COVID-19 requiring mechanical ventilation who developed 4- to 8-second episodes of asystole [4]. The third report described a severely ill patient with COVID-19 with episodes of prolonged asystole up to 20 seconds on Day 14 of his hospitalization [5]. This patient developed asystole in the setting of posterior myocardial infarction and required extracorporeal membrane oxygenation due to hypoxia. A fourth report detailed a previously healthy 55-yearold female with systemic illness who notably developed asystole lasting up to 90 seconds [6]. To the best of our knowledge, this is the first report of a healthy and asymptomatic patient with COVID-19 experiencing such prolonged asystole.

Our case highlights that prolonged asystole can occur in any COVID-19 patients regardless of their symptoms and therefore is not a consequence of systemic illnesses. We propose that SARS-CoV-2 infection may induce neurocardiogenic syncope with predominantly chronotropic inhibition [7] or directly affect the entire cardiac conduction system including cardiac pacemakers, sinus node, atrioventricular node and His–Purkinje system resulting in a combination of sinus arrest and the absence of escape beats [8, 9]. We hope this case will increase awareness of this potentially deadly cardiac complication even among non-hospitalized asymptomatic COVID-19 populations.

Further research and case documentation in the literature will help illicit a better understanding of the pathophysiology and natural history of this complication from infection.

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None.

# CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest to declare.

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## ETHICAL APPROVAL

No approval was required for this submission.

# CONSENT

Written informed consent was obtained from the patient for the publication of this case report. A copy is available for review by the editorial staff of this journal.

# **GUARANTOR**

Joseph S. Needleman

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