

Subclinical pericarditis long after SARS-CoV-2 infection: A case report

Anna Giulia Falchi,¹ Sergio Leonardi,² Giovanni Cappa,³ Stefano Perlini¹

¹*Emergency Department, IRCCS Policlinico San Matteo Foundation, Pavia;* ²*Coronary Care Unit and Laboratory of Clinical and Experimental Cardiology, IRCCS Policlinico San Matteo Foundation, Pavia;*

³*Emergency Medicine Postgraduate Training Program, University of Pavia, Italy*

Abstract

We describe a rare complication of COVID-19 long after infection in a 76-years-old man presented to the Emergency Department with dyspnea and palpitations. A 12-lead Electrocardiogram (ECG) showed sinus tachycardia PR depression in the inferior leads associated with an apparent pseudo ST elevation. In the absence of elevation of inflammatory indices, considering the lack of symptoms neither NSAIDs nor colchicine were prescribed, and the patient was referred for clinical follow-up. After ten days ECG documented initial reduction of the widespread concave STE and PR depression, and the 1-month follow-up visit, the patient was asymptomatic with unremarkable physical examination, and a 12-lead ECG showed almost complete

normalization of the ST and PR segments. Although pericardial involvement after COVID-19 infection has been already described, the incidence of subclinical pericarditis has not and may have implications for the monitoring of patients with uncomplicated COVID-19 infection managed as outpatients.

Introduction

Cardiac complications of COVID-19 infection are emerging in the current pandemic, although few descriptions are available in those who are managed as outpatients.¹ Notably, SARS-CoV2 can cause long-lasting illness, and the term *long COVID* describes the disease in people who continue reporting symptoms several weeks after the infection. Whether COVID-19 will become a chronic or permanent condition in some patients, and/or whether SARS-CoV-2 infection might generate a new autoimmune disease (or autoimmune-like) remains unknown.²

Case Report

A 76-year-old white man without previous history of cardiovascular disease entered the Emergency Department (ED) with dyspnea associated with palpitations. He denied other symptoms, and in particular chest pain. The patient referred a previous admission 6 weeks prior, due to COVID-19 infection with initial signs of interstitial pneumonia, followed by clinical remission in 4 weeks. Since the nasopharyngeal swab SARS-CoV-2 RNA test was negative 8 days before, quarantine was terminated. He reported long-term fatigue and paroxysmal nocturnal dyspnea. He was a non-smoker and he denied any substance abuse. His clinical history reported: primary systemic hypertension, Chronic Obstructive Pulmonary Disease (COPD), previous bladder cancer.

Upon arrival at the ED, physical examination revealed blood pressure of 100/60 mmHg, heart rate of 120 bpm, oxygen saturation of 96% while breathing room air, and body temperature of 36.6 °C (he remained afebrile during the subsequent clinical course).

Clinical examination

Valid heart sounds without any murmur, normosphygmic peripheral arterial pulses, absence of peripheral edema and jugular distension, normal bilateral vesicular murmur.

Arterial gas analysis

pH of 7.46, oxygen partial pressure of 86.3 mmHg, carbon dioxide partial pressure of 33.1 mmHg with PiO₂/FiO₂ rate 435 mmHg.

ECG

A 12-lead Electrocardiogram (ECG) (Figure 1) showed sinus tachycardia at 120 bpm, with PR depression most evident in the

Correspondence: Anna Giulia Falchi, Emergency Department, IRCCS Policlinico San Matteo, Piazzale Golgi 19, 27100 Pavia, Italy.
Tel.: +39.0382.502039.
E-mail: a.falchi@smatteo.pv.it

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Consent for publication: The patient gave his written consent to use his personal data for the publication of this case report and any accompanying images.

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inferior leads associated with an apparent pseudo ST elevation. This finding was evident when the isoelectric line was identified as the TP segment (see red line). An ECG performed in the previous month was normal.

Chest radiography findings were unremarkable. Blood tests revealed normal levels of markers of myocyte injury [high-sensitivity troponin T level of 5 ng/L (double check), URL 49 ng/L], normal value of C-Reactive Protein (CRP) levels (0,57 mg/dL), normal blood cell counts. Transthoracic Echocardiogram (TTE) confirmed normal structure and function, with normal pericardium and no pericardial effusion. A nasopharyngeal swab specimen sample was repeated, yielding negative results for SARS-CoV-2.

Subclinical pericarditis was suspected. In the absence of inflammatory indices elevation, and considering the lack of symptoms and the presence of renal insufficiency, neither NSAIDs nor colchicine were prescribed. The patient was referred for clinical

follow-up at interval of ten days, in order to evaluate the clinical evolution. The absence of other causes for pericardial effusion and the history of recent COVID-19 infection with initial signs of interstitial pneumonia rose the suspicion of a viral etiology. It has to be underscored that the clinical manifestations in the post-acute period after COVID-19 are largely unknown, considering that many of these features can resolve with time and their prevalence therefore depends on the evaluation moment.

After ten days, concomitant with general clinical improvement, CRP was in the normal range, as well as TSH (0.33 mcU/L). ECG documented initial reduction of the widespread concave STE and PR depression (Figure 2). At the 1-month follow-up visit, the patient was asymptomatic with unremarkable physical examination, blood pressure was 110/70 mmHg, and heart rate was 75 beats per minute. A TTE was repeated showing normal structure and function, and absence of pericardial effusion. A 12-lead ECG

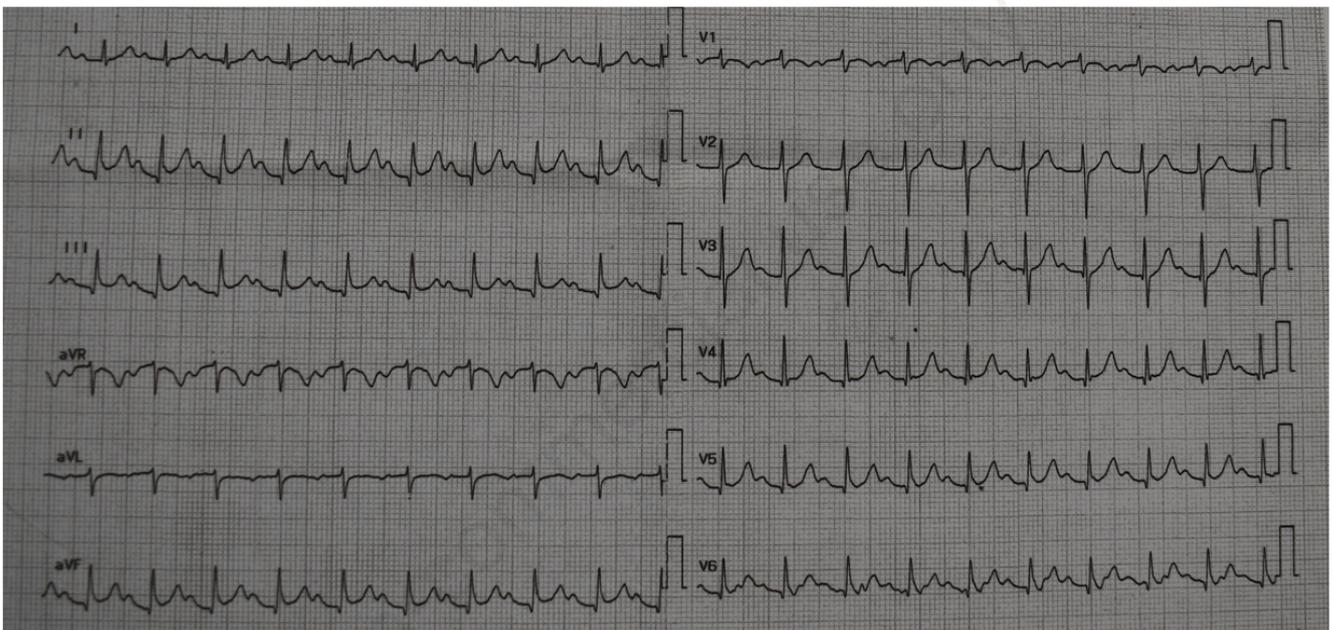


Figure 1. The 12-lead electrocardiogram showing sinus tachycardia at 120 bpm, with PR depression most evident in the inferior leads associated with an apparent pseudo ST elevation.

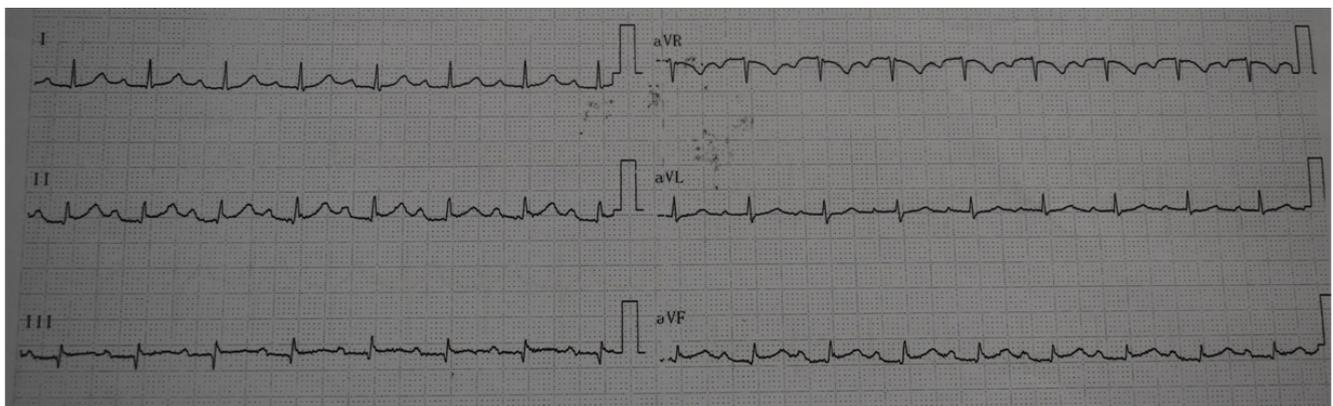


Figure 2. The ECG after ten days documenting the initial reduction of the widespread concave ST and PR depression.

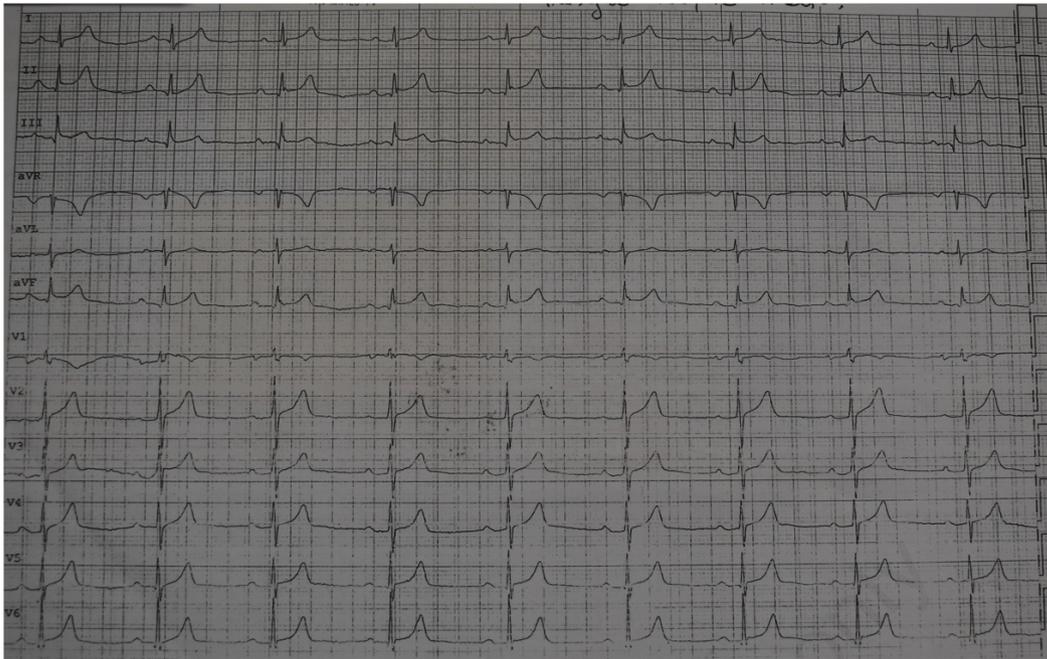


Figure 3. The 12-lead ECG after ten days showing almost complete normalization of the ST and PR segments.

showed almost complete normalization of the ST and PR segments (Figure 3).

Acute pericarditis is the most common disease of the pericardium and is responsible for 0.2% of chest pain-related hospitalizations, with an incidence two times higher in men than in women. Acute pericarditis is usually self-limiting, although it recurs in up to 30% of cases. In developed countries, viruses are the most common causes, while Tuberculosis (TB) is the most common cause in the world and in developing countries, often associated with Human Immunodeficiency Virus (HIV) infection. In developed countries the most common causes of pericarditis are idiopathic or viral (42-49%). It is commonly diagnosed in viral infections, including coxsackie, enterovirus, herpes simplex, cytomegalovirus, H₁N₁, respiratory syncytial virus, parvovirus B19, influenza, varicella, HIV, rubella, echovirus, hepatitis B and C and finally for SARS-CoV-2.³⁻⁷ It is not always possible to define the responsible virus, and the viruses responsible in a given patient may be different genotypes of the same virus or different coexistent viruses.

A post-acute COVID-19 syndrome was detected in a half of COVID-19 survivors, not limited to severe acute COVID19 patients, and with no identified predictors. Amenta *et al.*⁸ suggest that the post-acute period for COVID-19 starts 3 weeks after symptom onset. Also, they propose classifying post-acute manifestations into 3 categories: residual symptoms that persist after recovery from acute infection; organ dysfunction persisting after initial recovery; and new symptoms or syndromes that develop after initial asymptomatic or mild infection. A possible hypothesis for our patient is that after COVID-19 infection, virus-mediated damage to pericardial tissues keeps on, although with normal inflammation indices, probably by induction of pro-inflammatory cytokines, not included in our assessment. Although pericardial involvement after COVID-19 infection has been already

described, the incidence of subclinical pericarditis has not and may have implications for the monitoring of patients with uncomplicated COVID-19 infection managed as outpatients.

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