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CASE REPORT
Cardiovascular imaging

Purulent pericarditis caused by methicillin-sensitive *Staphylococcus aureus* in an immunocompetent adult after COVID-19 pneumonia: a case report

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Background SARS-CoV-2 has been implicated in many cardiac pathologies, manifesting mainly as acute. However, acute purulent pericarditis is exceedingly rare in the antibiotic era. Though, few studies have associated it with long-COVID, prompt recognition and treatment are crucial.

Case summary A 61-year-old immunocompetent woman presented with a left lower limb pitting oedema 1 month after COVID-19 pneumonia. Following clinical, laboratory, and imaging work-up, the patient was found to have deep vein thrombosis of the anterior and posterior tibial and gastrocnemius veins. Owning to persistent sinus tachycardia, an additional work-up was performed, which revealed a large pericardial effusion. Pericardiocentesis drained the frank pus, and subsequently, empirical antibiotics therapy was initiated. Pericardial fluid cultures showed methicillin-sensitive *Staphylococcus aureus* (MSSA). Following the antibiotic treatment with cloxacillin 6 × 2 g IV for 6 weeks, the patient fully recovered.

Discussion Herein, we report a rare case of bacterial pericarditis caused by MSSA 1 month after COVID-19 pneumonia. Additionally, this condition may arise as a result of immunosuppressive treatment with glucocorticoids during and after COVID-19 pneumonia. However, the causal association has not yet been confirmed.

Keywords Purulent pericarditis • Pericardiocentesis • *S. aureus* pericarditis • Post-COVID syndrome • Long-COVID • Case report

ESC Curriculum 2.2 Echocardiography • 6.6 Pericardial disease

Learning points

- Purulent pericarditis is nowadays a very rare, but serious bacterial infection, and if left untreated usually has a lethal outcome.
- In the long-COVID era, one must maintain a high suspicion for cardiac complications in patients with a history of COVID-19.

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Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has been implicated in cardiac pathologies, manifesting predominantly as acute disease. Recently, there are sporadic case reports of cardiac pathologies (including mild myopericarditis and cardiac tamponade) occurring as long-COVID complications.[1,2]

The short- or long-term consequences of immunosuppressing medications (glucocorticoids) used in the treatment of COVID-19 may arise, leading to autoimmune and inflammatory diseases.[3]

Since acute purulent pericarditis is hardly encountered, most doctors never discover this condition during their careers. Historically, the main risk factors have been bacterial chest infections. However, recently, we have witnessed a growing list of conditions identified as potential predisposing factors, among which immunosuppression also occurs.[4,5]

This report presents a rare case of acute purulent pericarditis caused by methicillin-sensitive *Staphylococcus aureus* (MSSA) in a 61-year-old immunocompetent Caucasian female 1 month after the onset of COVID-19 pneumonia.

Timeline

8 May 2021	Patient was admitted to the hospital due to COVID-19 bilateral pneumonia.
22 May 2021	Patient was discharged from COVID-19 unit on low-dose oral methylprednisolone.
29 June 2021	Emergency department visit, deep vein thrombosis was diagnosed.
30 June 2021	Pulmonary angiography CT excluded pulmonary embolism but showed a large pericardial effusion and small bilateral pleural effusion.
1 July 2021	Pericardiocentesis was performed (800 mL of fluid), macroscopic pus was evacuated, and empiric antibiotic treatment (vancomycin, ceftriaxone) was initiated.
2 July 2021	Pericardial effusion cultures grew methicilin-sensitive *Staphylococcus aureus*, change of the antibiotics to cloxacillin 6 × 2 g IV.
15 July 2021	Diagnostic pleurocentesis was perfomed; 20 mL of fluid was sent to cytology; no malignant cells were found.
21 July 2021	X-ray showed a complete resolution of bilateral pleural effusion.
22 July 2021	Patient was dischared after 6 weeks of IV antibiotic treatment.
3 September 2021	Office follow-up with the patient reporting no symptoms of heart failure. Echocardiography showed compete resolution of pericardial effusion.

Case report

A 61-year-old woman presented to the hospital emergency department with left-sided lower limb pitting oedema. She reported 4 days of progressive worsening oedema and month-long exercise intolerance with malaise, but without resting dyspnoea, chest pain, fever, chills, or syncope.

The patient had a history of arterial hypertension, diabetes mellitus type 2, and diabetic nephropathy. She had suffered a stroke 4 years previously, which resulted in mild left-sided hemiparesis. One month before the admission, she was diagnosed with bilateral COVID-19 pneumonia and was subsequently treated with non-invasive respiratory support (continuous positive airway pressure [CPAP] mask), glucocorticoids, and low molecular weight heparin. No central lines or urinary catheters were inserted, and she was discharged with a low dose of oral methylprednisolone (32-24-16-8 mg per oral (PO), 7 days per dose).

On arrival, the patient had a blood pressure of 135/85 mmHg, heart rate of 100/min, respiratory rate of 16/min, SpO_2 of 94%, and body temperature of 36.6°C. Physical examination did not reveal jugular venous distention or murmurs or rubs; however, she exhibited oedema of the left lower limb (4 cm increase in circumference compared to the right lower limb). The electrocardiogram (ECG) showed sinus tachycardia, low QRS voltages in the precordial ECG leads, slight electrical alternans, and no ST segment changes (*Figure 1A, B*). Laboratory results were significant for leucocytosis of 11×10^9 cells/L (normal range $3.4–9.7 \times 10^9$ cell/L), neutrophils were 69.8% (normal range 44–72%), lymphocytes were 23.2% (normal range 20–46%), elevated C-reactive protein of 24.4 mg/L (normal range <5.0 mg/L), negative procalcitonin, and elevated D-dimers of 6036 mcg/L (normal range <550 mcg/L). Due to elevated D-dimer values and unilateral lower limb oedema, a colour Doppler analysis of the lower limb veins was performed. Non-compressive masses were observed in the anterior and posterior tibial and gastrocnemius veins. The diagnosis of deep vein thrombosis was confirmed, and the patient received anticoagulation therapy. Chest radiography revealed an enlarged cardiac silhouette and dilated mediastinum; however, no further evaluation was performed at that time (*Figure 2*).

After confirming the diagnosis, the patient was admitted for overnight observation and follow-up. Owing to the persistent sinus tachycardia present during rest, a computerized tomography (CT) pulmonary angiogram (CTPA) was performed excluding the pulmonary emboli. It revealed a hypodense pericardial collection, suggestive of a large circumferential pericardial effusion (50 mm) (*Figure 3*).

Small bilateral pleural effusion and pulmonary consolidation have also been described. Transthoracic echocardiography confirmed a large pericardial effusion; however, no fibrin strands were observed. The ejection fraction of the left ventricle was estimated as 55%, and no major valvular pathologies or valvular vegetation were seen. While we observed a 20% variation in the transmitral inflow pattern, no collapse of the right heart chambers was noted (*Figure 4*).

The following day, the laboratory results were negative for troponin I antibodies; however, N-terminal probrain natriuretic peptide (NT-proBNP) was elevated to 1600 pg/mL (normal range <125 pg/mL). The patient was haemodynamically stable during the examination. As cardiac magnetic resonance (CMR) was unavailable in our hospital, only pericardiocentesis was performed, which showed macroscopic purulent effusion, and more than 800 mL was drained. Immediately after drainage, we started empirical antibiotic treatment with intravenous vancomycin (15 mg/kg twice daily, bodyweight 65 kg) and ceftriaxone (2 g once daily). Analysis of the pericardial fluid revealed an exudate (pH 8.0, leucocytes 105×10^9/L, glucose 1.4 mmol/L, LDH 11758U/L, proteins 51 g/L). Cultures of the aspirated pericardial fluid showed MSSA. Therefore, we switched the antibiotic therapy to cloxacillin 6 × 2 g IV for 6 weeks.

Interestingly, four blood cultures remained negative, and the patient had no history of intubation, chest trauma, intrathoracic procedures, or central venous lines. A myocardial biopsy was not performed. The cytology was negative for malignancy. Urinary cultures also showed MSSA.

After 6 weeks of the intravenous antibiotic regimen, the patient had a full recovery and was scheduled for a follow-up 3 months after discharge. At the 3-month mark, the patient did not report any symptoms of heart failure. Echocardiography showed complete resolution of the pericardial effusion, the left ventricular ejection fraction was normal, and no valvular or other pathologies were recorded.

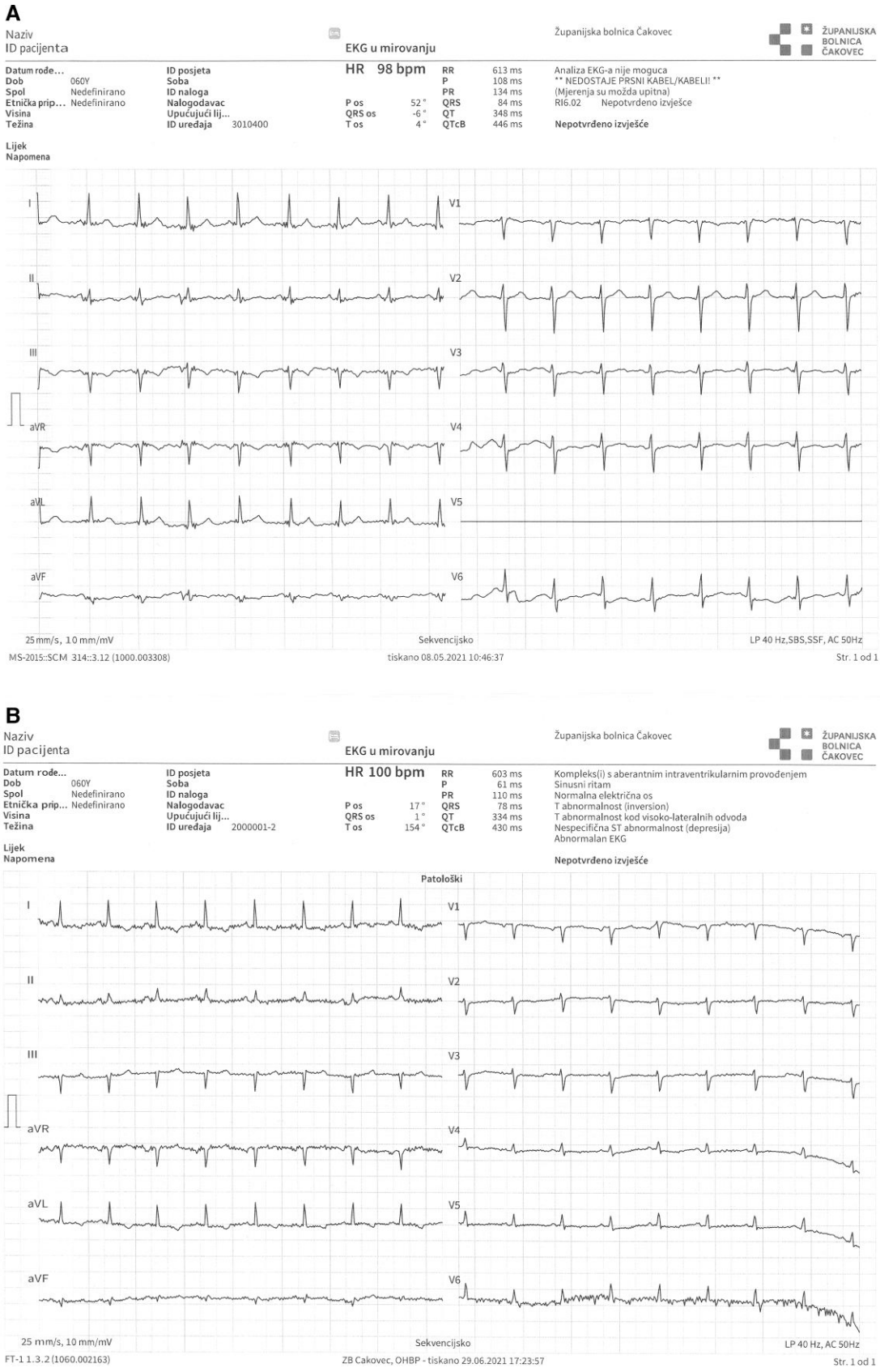

Figure 1 (*A*) ECG of the patient at admission for COVID-19 pneumonia, lead V5 is missing, (*B*) ECG of the patient at admission 1 month after COVID-19 pneumonia. ECG shows sinus tachycardia, low QRS voltage in precordial leads, and slight electrical alternans.

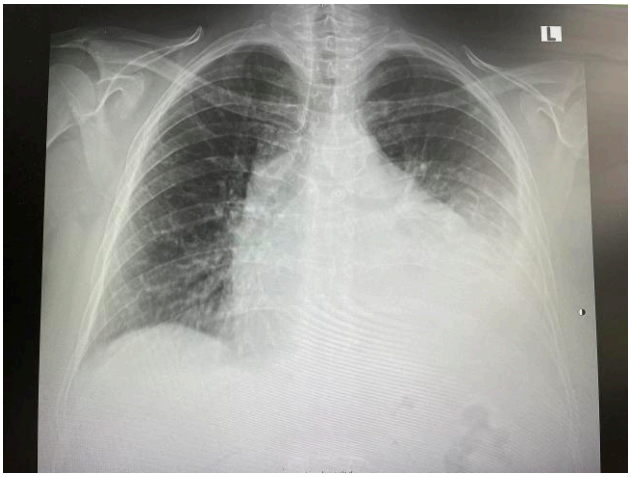

Figure 2 X-ray of the patient showing enlarged cardiac silhouette and dilated mediastinum.

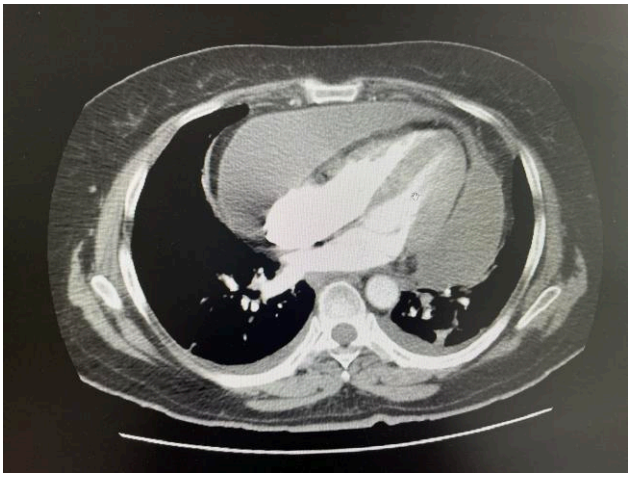

Figure 3 CT pulmonary angiogram excluded pulmonary emboli. A hypodense pericardial collection is observed that is suspicious for a large circumferential pericardial effusion (50 mm).

Discussion

To our knowledge, this rare case report is one of the few reports of acute bacterial pericarditis presenting shortly after COVID-19. The patient presented to the hospital in the fourth week after recovery from COVID-19 pneumonia. There were no typical signs or symptoms of pericarditis. Only subtle signs of pericardial effusion (sinus tachycardia and precordial low QRS voltage on ECG, enlarged cardiac silhouette, and dilated mediastinum) were present at the initial presentation in the emergency department. However, the main complaint of the patient was left lower limb swelling and month-long exercise intolerance with malaise, but no resting dyspnoea which was considered a consequence of COVID-19. Within 24 h after admission, using CTPA and echocardiography, a prompt diagnosis was made, and adequate treatment was started. Pericardiocentesis revealed macroscopic pus, but the patient was haemodynamically stable and showed no signs of sepsis. In accordance with the current guidelines for pericardial disease,[4] we initiated empiric antibiotic therapy (intravenous vancomycin and ceftriaxone). Four sets of blood cultures were negative, and only pericardial effusion and urinary cultures were positive for MSSA. After the results were obtained, targeted cloxacillin (6 × 2 g IV) treatment was initiated.

A similar case was described in Singapore 3 weeks after SARS-CoV-2 infection.[6] In the present case report, it was concluded that bacterial pericarditis (pericardial fluid culture grew *Staphylococcus hominis*) was a secondary bacterial complication of the viral disease. In contrast to our case, the patient presented with fever, mild epigastric tenderness, right upper quadrant abdominal tenderness, and suffered a rapid haemodynamic compromise. However, the causal associations remain unclear.

According to a recent systematic literature review, cardiac manifestations secondary to COVID-19 are heterogeneous.[1] Most described cases of cardiac involvement are present in the acute phase of COVID-19.[7] With the success of COVID-19 vaccination programs, the incidence of acute COVID-19 has decreased. However, given the high number of people who have had COVID-19 and have recovered, we face a significant number of long-COVID patients who present with

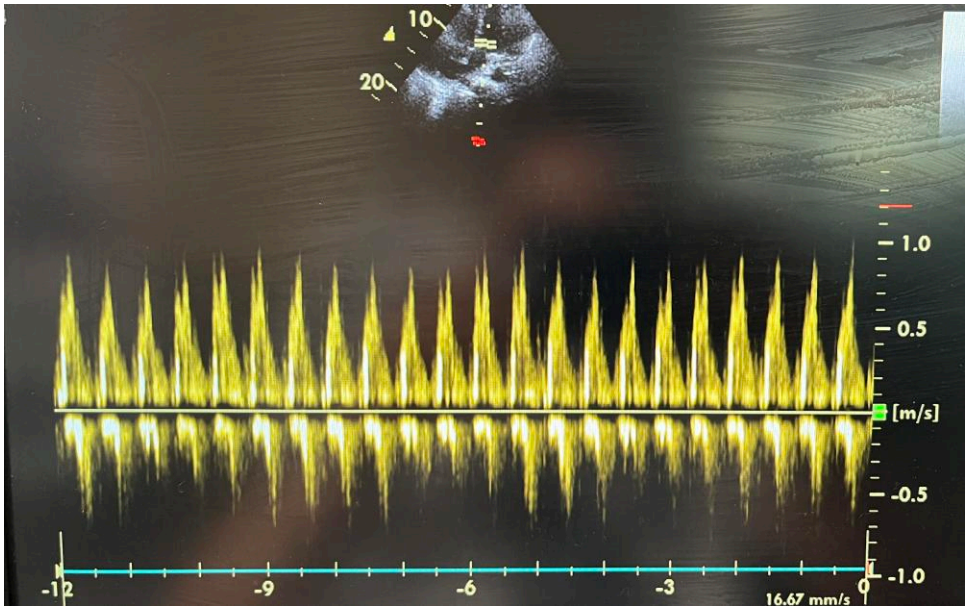

Figure 4 Echocardiography shows 20% variations in transmitral inflow.

persistent symptoms months after COVID-19 infection. Unfortunately, little is known about the cardiovascular manifestations that occur after clinical and virological recovery from SARS-CoV-2 infection.[8] Carubbi *et al.* recently described a case series where seven patients developed pericarditis after a median of 20 days of clinical and virological recovery from SARS-CoV-2 infection without a specific identifiable cause.[2] They associated the occurrence of the disease with ongoing inflammation sustained by the persistence of viral nucleic acids, without viral replication in the pericardium.

In conclusion, this report describes the first case of acute purulent pericarditis caused by MSSA 1 month after COVID-19 pneumonia. This case study highlights a high possibility of cardiac complications in patients with a history of COVID-19. As discussed here, the clinical presentation may not be straightforward. In this case, transient immunosuppression and post-COVID status might have triggered acute purulent pericarditis. However, a direct association remains unclear. Further studies are needed to gain a better clinical and molecular understanding of COVID-19 and cardiac complications.

Lead author biography

Tea-Terezija Cvetko (born Kasnik) was born in Dobrna (Slovenia). After finishing Medical Faculty in Ljubljana (Slovenia), she started working as a junior doctor at General Hospital Ptuj (Slovenia). In 2018, she started specializing in cardiology at County Hospital Cakovec (Croatia) and Sisters of Charity Hospital Zagreb (Croatia). She did a rotation at Hermann Memorial Heart and Vascular Institute in Houston, Texas, USA, and 1 month of Echocardiographic training at Vienna General Hospital, Austria. Her main areas of interest are non-invasive cardiology (advanced echocardiography and cardiac magnetic resonance).

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports*.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: Written consent was obtained from the patient for data collection, including echocardiography, and was used for publication. This case report was approved by the Local Ethics Committee under COPE guidelines.

Conflict of interest: None declared.

Funding: None declared.

Data availability

The data underlying this article are available in the article and in its online supplementary material.

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