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A case of acute bacterial pericarditis in a COVID-19 patient

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Dear Sir,

We herein report a case of a COVID-19 patient with acute bacterial pericarditis. A 32-year-old man without comorbidities and cardiovascular risk factors presented with three days of fever and right-sided non-pleuritic chest pain. At 18 days prior to presentation, he had been seen for fever, sore throat and myalgia; a nasopharyngeal swab for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) polymerase chain reaction (PCR) testing had returned positive.

On examination at the emergency department, he was febrile at 38.3°C, with mild epigastric and right upper abdominal quadrant tenderness. Examination of the heart and lungs was unremarkable. Laboratory investigations showed leucocytosis (white blood cell count 10.6 [4.0–9.6] × 10^9/L), neutrophilia (neutrophil count 7.83 [1.90–6.60] × 10^9/L), thrombocytopenia (platelet count 147 [150–360] × 10^9/L), raised lactate dehydrogenase (978 [270–550] U/L), C-reactive protein 49.8 (0.0–5.0) mg/L, procalcitonin 2.57 (0.00–0.05) ug/L and troponin-I 110 (0–19) ng/L, which peaked at 172 ng/L five hours later. Electrocardiography was unremarkable (Fig. 1a). Chest radiography showed right lung lower zone airspace opacities (Fig. 1b). As the patient became increasingly tachycardic, he was transferred to the intensive care unit (ICU). Urgent transthoracic echocardiography (TTE) showed good ejection fraction, no regional wall motion abnormality and a 0.6-cm rim of pericardial effusion (Fig. 1c). The patient developed mild hypoxia, and arterial blood gas on 1 L/min of supplemental oxygen showed respiratory alkalosis and a PF (PaO_2/FiO_2) ratio of 268. He was treated for bacterial pneumonia. The next morning, the patient was more tachycardic despite a fluid infusion, and his right upper abdominal quadrant pain had intensified. Computed tomography of the thorax and abdomen showed a pericardial effusion and bilateral pleural effusions with no evidence of acute cholecystitis or calculus (Fig. 1d).

A few hours later, he was noted to be diaphoretic, tachypnoeic at 45 breaths per minute, and tachycardic at 150 beats per minute (Fig. 2a). This necessitated endotracheal intubation and central venous line insertion in anticipation of the need for a vasopressor infusion. Another urgent TTE revealed rapid enlargement of the pericardial effusion, with a maximum inter-pericardial separation of 1.8 cm (Fig. 2b) and evidence of right atrial collapse despite a plethoric inferior vena cava. Pericardiocentesis drew 100 mL of haemoserous fluid. The pericardial fluid analysis showed the following: nucleated cells 94 cells/uL, neutrophils 2%, lymphocytes 38%, lactate dehydrogenase 636 U/L and protein 48 g/L. A Gram stain of the pericardial fluid showed Gram-positive cocci, and a culture grew *Staphylococcus hominis* (*S. hominis*). Acid-fast bacilli smear and PCR tests for SARS-CoV-2, tuberculosis, enterovirus and adenovirus were negative. Cytology did not show malignant cells. Four sets of blood cultures grew *Staphylococcus lugdunensis* (isolated from aerobic bottle, taken from peripheral vein), mixed coagulase-negative staphylococci (CoNs) [isolated from aerobic bottle, taken from peripheral vein], *Staphylococcus epidermidis* (isolated from aerobic bottle, taken from right radial arterial line), *Enterobacter cloacae* (isolated from both bottles, taken from central line) and *Staphylococcus epidermidis* (isolated from anaerobic bottle, taken from central line). A HIV screen was negative. The patient was stable enough to be extubated the next day. He received one week of vancomycin and meropenem with good clinical response, followed by two weeks of intravenous cefazolin and one week of oral ciprofloxacin. The pericardial drain was removed after five days, with cumulative fluid drainage of 1,600 mL. A repeat TTE 18 days later confirmed the resolution of the pericardial effusion. We concluded that the *S. hominis* positive culture from the pericardial fluid was unlikely to be a contaminant, given the positive pericardial fluid Gram stain, compatible clinical and echocardiographic features, and response to treatment.

To our knowledge, this is the first reported case of acute bacterial pericarditis complicating SARS-CoV-2 infection in Singapore. The patient presented again during the third week following confirmation of SARS-CoV-2 infection with non-respiratory symptoms. His chest radiograph on previous admission showed no consolidation or pleural effusion. The early ICU admission and urgent bedside diagnostics prevented an impending cardiovascular collapse from cardiac tamponade. He improved rapidly within 24 hours of pericardiocentesis and antibiotics.

There are four other published cases in the literature of SARS-CoV-2-associated pericarditis manifesting within a week of respiratory symptoms.[1-4] These cases had a more fulminant course with hypoxia and haemodynamic compromise; one recovered without pericardiocentesis. Microbiology results of blood and pericardial samples were negative.

Our case suggests that myocardial and pericardial involvement can manifest as a late sequela in SARS-CoV-2 infection. It is plausible that, similar to other viral infections, the myocardial and pericardial inflammation that occurred earlier was complicated by a secondary bacterial infection. One limitation is that myocarditis cannot be confirmed in the absence of cardiac magnetic resonance imaging, histopathology and consistent electrocardiography with TTE findings.

The present case also highlights the challenges of ascertaining the clinical relevance of CoNS isolation and identification.[5] CoNS are typical opportunistic bacteria that not only colonise healthy individuals but also represent one of the major hospital pathogens with a substantial, increasing impact on human life and health.[6] There have been case reports of pneumonia caused by CoNS.[5,7,8] Bilateral pleural effusions were also noted in our patient. However, the pleural fluid was not sent for further evaluation, as bedside ultrasonography showed that there was minimal pleural fluid for aspiration. The patient's polymicrobial bacteraemia may have been acquired via a vascular (line-related) source, and some of the

isolates may represent colonisation rather than true bacteraemia. While it remains unclear, we hypothesise that his *S. hominis* pericarditis, specifically, could have arisen from a pulmonic, vascular (line-related) or cutaneous source.

In conclusion, SARS-CoV-2 associated pericarditis with superimposed bacterial infection is rare, and this case highlights the need for continued vigilance beyond the early phase of illness and to expedite the diagnostic process for optimal outcomes.

Yours sincerely,

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FIGURES

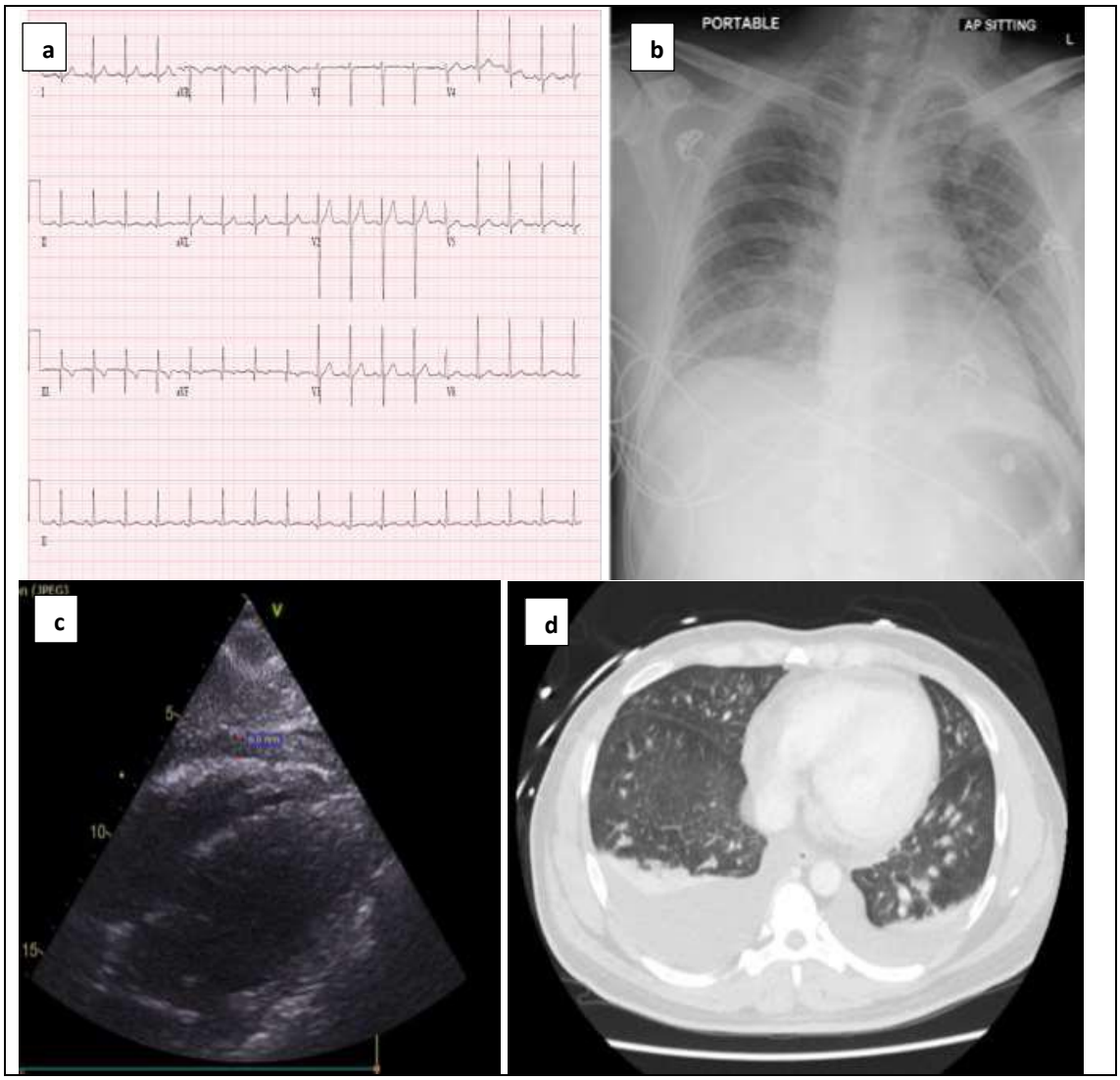

Fig. 1 (a) Electrocardiogram shows a normal sinus rhythm, similar to the patient's baseline. (b) Chest radiograph shows patchy airspace opacities over the right lung lower zones. (c) Transthoracic echocardiogram shows good ejection fraction without regional wall motion abnormality, with a 0.6-cm rim of pericardial effusion. (d) CT image shows a small pericardial effusion and bilateral pleural effusions.

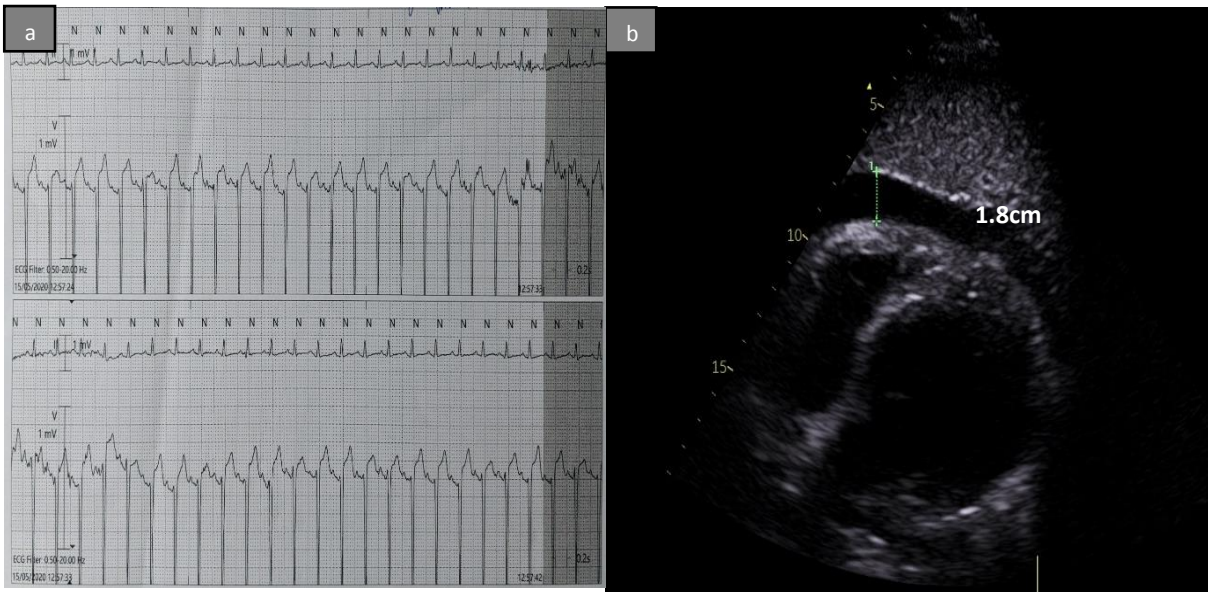

Fig. 2 (a) Telemetry image shows sinus tachycardia at 150 beats per minute. (b) Transthoracic echocardiography shows that the pericardial effusion has enlarged with a maximum intra-pericardial separation of 1.8 cm (line).