



A SARS-Cov2-negative corona victim

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Sirs:

A 48-year-old male was admitted to the specialized COVID-emergency department of a large tertiary hospital on March 27, 2020. The emergency doctor had been dispatched to the home of the patient due to an emergency call. The patient suffered from dyspnea for approximately 7 days. A short episode of an upper airway infection and a contact to a confirmed SARS-Cov2 positive individual was reported. The emergency physician found the patient in acute and severe distress because of dyspnea and thoracic pain. The patient was conscious (Glasgow Coma Scale 15), hypotensive (RR 80/60 mmHg) and tachycardic (120 bpm). Oxygen saturation (SaO₂) was 70% despite oxygen insufflation via a nasal cannula which prompted immediate intubation on the scene. During transport, repetitive boli of norepinephrine were required to maintain blood pressure. The chest X-ray on admission showed confluent infiltrations in the right and left lung suggestive of a viral pneumonia (Fig. 1).

The laboratory findings on admission are depicted in Fig. 2. As reported for patients with COVID-19, the patient showed low lymphocyte count, elevated neutrophil/

lymphocyte ratio (7.88) and elevated serum concentrations for d-dimers, ferritin and high sensitive troponin (hs-cTn) (495 ng/l). Apart from leukocytosis (19.6/nl), sepsis markers (CRP, Procalcitonin) were in the normal range. An echocardiogram performed by the emergency room staff was reported as “normal”. The ECG on admission showed sinus rhythm without ST-elevation (Fig. 3). In view of these findings, acute respiratory failure (ARDS) due to COVID-19 pneumonia and consecutive circulatory failure was proposed as the primary diagnosis.

Within few hours, the patient developed multi-organ failure. After transfer to the ICU, the respiratory (PaO₂/FiO₂ ratio 74 mmHg) and hemodynamic situation aggravated rapidly necessitating ventilation in prone position and high dose vasopressor support. Anuric kidney failure, lactate acidosis (pH 7.11; Lactate 6.7 mmol/l; base excess – 13 mmol/l) and severe hyperkalemia (7.4 mmo/l) prompted hemofiltration. A rise of hs-cTn (3.470 ng/l) corroborated ongoing myocardial damage. Central venous O₂-saturation was 73%. Repeated ECG tracings showed no ST-elevations. At this point, potential myocarditis was added to the primary differential diagnosis.

Echocardiography was repeated by a cardiologist revealing a decline in systolic function (LVEF 40%), a large hypokinesia of the lateral ventricular wall and a floating structure on the mitral valve with moderate mitral valve insufficiency. However, invasive coronary angiography was prohibited due to the clinical situation.

The first swab test for SARS-Cov2 was negative. Due to the patient’s history and the clinical presentation, the rapid deterioration in the situation was still considered to be caused by respiratory failure due to COVID-19. A second swab test came in negative for SARS-Cov2 as well as influenza A and B.

The hemodynamic situation of the patient deteriorated despite maximal catecholamine therapy and the patient died on day 3 after admission. The autopsy showed coronary calcifications and a large type 1 myocardial infarction due to a thrombotic occlusion of the atherosclerotic circumflex coronary artery and rupture of a papillary muscle (Fig. 4).

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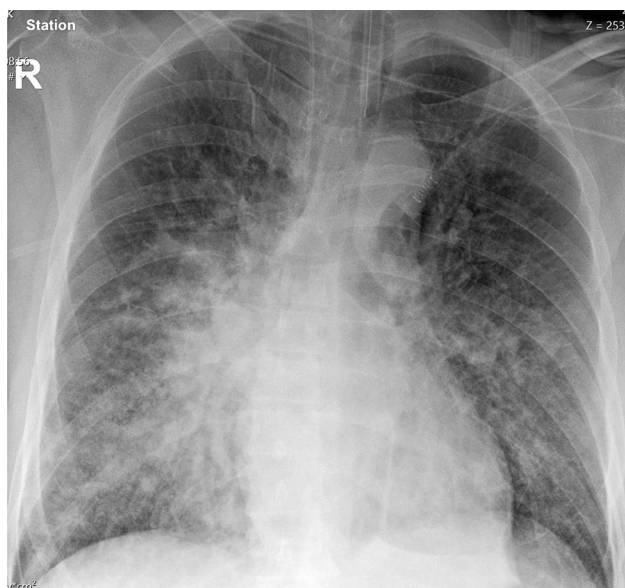


Fig. 1 Chest X-ray on admission

		Reference range
Leukocytes	19.6/nl	(3.7-10.1)
Thrombocytes	506/nl	(150-361)
Lymphocytes	10.8%	(18-45%)
Neutrophil/Lymphocyte ratio	7.88	
D-Dimer	3.84 mg/l	<0.55 mg/l
hs-Troponin	495 ng/l	<14 ng/l
NT-proBNP	2185 pg/ml	<85.8 pg/ml
LDH	237 U/l	<225 U/l
Creatinin	1.7 mg/dl	<1.2 mg/dl
GFR	47 ml/min	> 60 ml/min
Ferritin	424 µg/l	27-365 µg/l
Potassium	4.4 mmol/l	(3.5-5.3 mmol/l)
Sodium	137 mmol/l	(135-148 mmo/l)

Fig. 2 Laboratory results on admission

The 48-year-old patient without known significant comorbidities died from a type 1 myocardial infarction of the circumflex artery that was complicated by ischemic mitral valve insufficiency due to rupture of a papillary muscle. Due to the massive efforts to avoid infections of hospital patients and staff with SARS-Cov2 and the subsequent shifts in the organization of emergency care and the attention of the medical staff involved, the pathogenesis of the patient was only recognized post-mortem. Despite the very important measures to contain SARS-Cov2, it remains key to follow established clinical pathways. Importantly, cardiologists are important for the full assessment of hemodynamically compromised patients.

The novel coronavirus SARS-Cov2 and its clinical sequelae (so called coronavirus disease—COVID-19) has a wide spectrum of symptoms from asymptomatic causes to flu-like symptoms and finally pneumonia with acute respiratory distress syndrome [1]. Due to the clinical cause of COVID-19, the disease shares many of the initial symptoms with life-threatening cardiovascular diseases such as heart failure, myocardial ischemia and myocardial infarction and the virus might also involve the myocardium itself [2]. In patients with COVID-19, myocardial injury often develops [3, 4] and is associated with poor outcomes [5] as cardiovascular comorbidity load is high [6]. Furthermore, viral infections and pneumonia are associated with an increased risk for subsequent cardiovascular disease and myocardial infarction [7].

This case is an unfortunate example of a SARS-Cov2-negative corona victim, in whom all attention was placed on the possibility of COVID-19 infection. This case also highlights the need for emergency strategies to identify and treat life-threatening cardiovascular disease mimicking COVID-19 symptoms or coexisting in the presence of COVID-19. Physicians should pay great attention to cardiac symptoms and not lose focus on life-threatening cardiovascular disease, though it can sometimes hardly be distinguished from a COVID-19 infection, one of the major challenges nowadays.

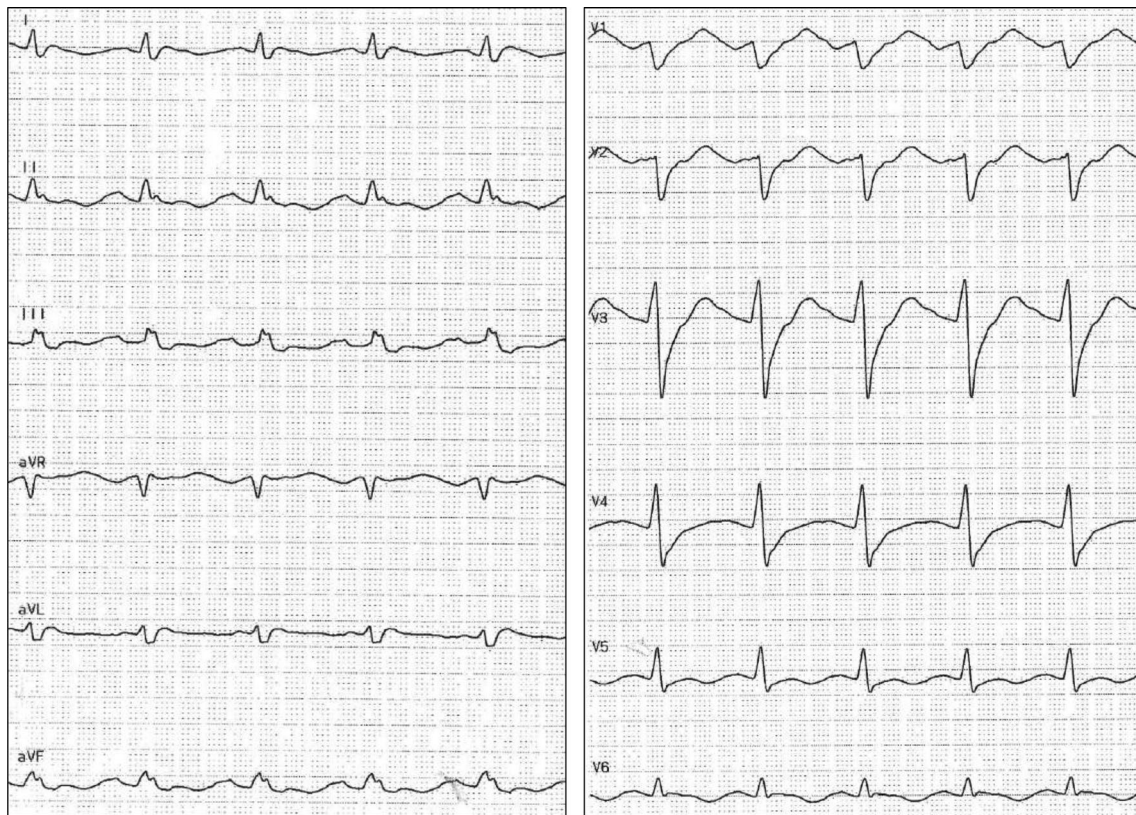


Fig. 3 Electrocardiogram on admission

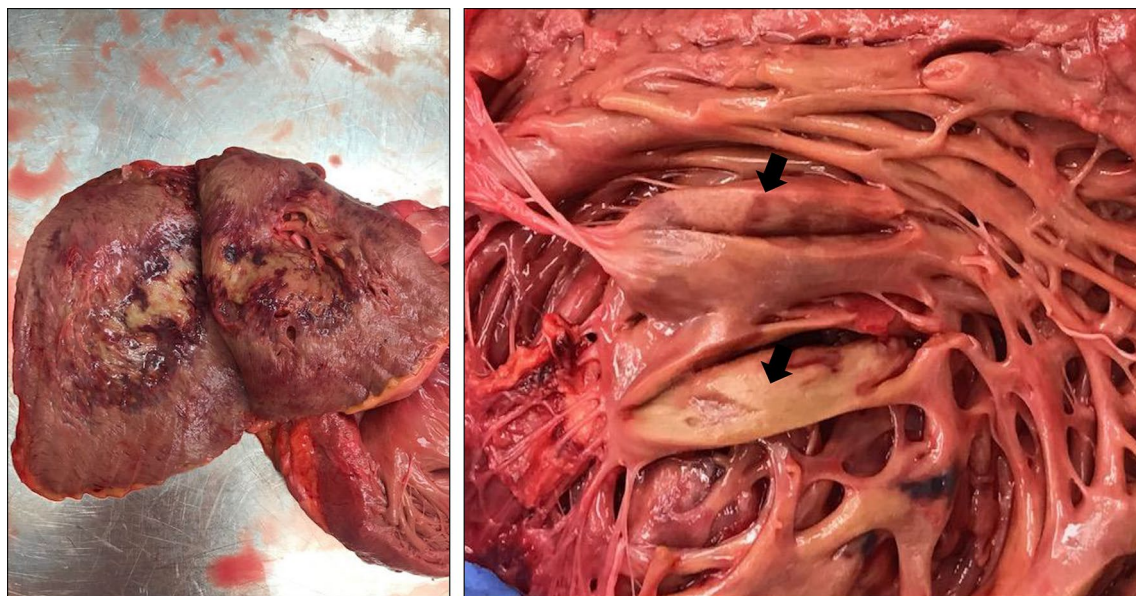


Fig. 4 Autopsy revealed acute and extended myocardial ischemia of the lateral wall of the left ventricle (left) and rupture of the ischemic papillary muscle. Arrows mark “tiger sign” of the papillary muscle indicating ischemic damage

Compliance with ethical standards

Conflict of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

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