Prothrombotic course in COVID-19 patients with acute myocardial infarction: Case series

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INTRODUCTION

This case series explores the link between SARS-CoV2 infection and atypical myocardial infarction. COVID-19 is caused by the SARS-CoV2 virus and can lead to hyperactivity of the immune system and a cytokine storm, resulting in endothelium damage and hypercoagulability [1]. We suggest that COVID-19 may predispose individuals to acute myocardial infarction (AMI) [2, 3]. This study aimed to present atypical cases of myocardial infarction associated with COVID-19.

METHODS

We reviewed data from patients who underwent coronary angiography between March 2020 and December 2021. Four patients who met the inclusion criteria for acute coronary syndrome with prothrombotic lesions and positive COVID-19 status were enrolled.

Despite a negative PCR test of one of our patients, we decided to present this case because the clinical presentation was similar to others.

The approval of the bioethical committee was not required for this study.

Case 1

A 52-year-old male presented with chest pain and dyspnea. Coronary angiography revealed narrowing of the left anterior descending artery (LAD) and acute occlusion in the proximal section of the circumflex artery (LCx). Cardiac arrest during the procedure resulted in the patient's death. The COVID-19 PCR test was positive.

Case 2

A 57-year-old male experienced severe chest pain with ST-segment elevation on ECG. Coronary angiography showed critical lesions in the diagonal branch and LAD artery. During the procedure, the patient experienced cardiac arrest but was resuscitated. The COVID-19 PCR test was negative, but it might have been a false negative due to difficulties during resuscitation.

Case 3

A 75-year-old male with a history of myocardial infarction was admitted with posterior wall myocardial infarction and cardiogenic shock. Coronary angiography revealed occlusions in multiple vessels. Despite cardiopulmonary resuscitation (CPR), the patient died. The COVID-19 PCR test was positive.

Case 4

A 43-year-old male, one month after COVID-19 infection, was admitted for cardiac arrest. Coronary angiography showed occlusions in multiple vessels. The patient died despite percutaneous coronary intervention.

RESULTS AND DISCUSSION

This case series included four male patients at a median age of 54.5 years. Three patients tested positive for COVID-19. All patients died rapidly on admission to the hospital. On admission, 3 of 4 patients did not present signs of upper respiratory infection, their COVID-19 status was not known, and ST-segment elevation myocardial infarction (STEMI) was the first manifestation of COVID-19. In 2 cases, cardiogenic shock was observed. Multiple culprit lesions were revealed on coronary angiography, which in general is a rare occurrence in STEMI patients but is more common in COVID-19 cases. Elevated proinflammatory and prothrombotic status in COVID-19 patients may contribute to these thrombotic complications. Scientists from

Table 1. Laboratory data

	Case 1	Case 2	Case 3	Case 4
CRP, mg/l	27.7	0.9	4.7	1.0
D-dimer, ng/ml	1557	–	1359	6705
PLT, 10^3/µl	268	153	143	287
Troponin I, ng/ml	>250	0.559	30.442	1.109
NT-proBNP, pg/ml	3110	207	5016	486

Abbreviations: CRP, C-reactive protein; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PLT, platelet count

the United Kingdom detected a higher incidence of multi-vessel thrombosis and a tendency for a higher thrombus burden in COVID-19 patients [4].

The common characteristics of our patients were multiple culprit lesions and rapid death. Simultaneous multiple coronary artery thrombosis in the general population of STEMI patients is uncommon and occurs in 4.8% of patients who underwent PCI [5]. On the other hand, an autopsy series reported, that in patients, who died from AMI, up to 50% had thrombotic occlusion in more than one epicardial coronary artery [6]. This discrepancy may be caused by the fact that sudden cardiac death may occur in patients with diffuse thrombosis [5]. In all 4 cases, there were lesions in the LAD. Three of 4 patients had lesions in the LCx, 2 of 4 patients had lesions in the RCA, and 1 of 4 in the diagonal branch. One of our patients developed a stent thrombosis.

Rescue intervention did not lead to a positive outcome, and the patients died. That outcome may have resulted from a very severe inflammatory response, which is the trigger for the blood coagulation cascade and interaction between the SARS-CoV2 virus and angiotensin-converting enzyme receptors in the heart [1, 7]. Similarly, in a case report from Italy, in a patient with diffuse artery thrombosis, resistance to medical therapy was observed. The authors of the report confirmed the hypothesis that high proinflammatory and prothrombotic status of COVID-19 patients is the main factor triggering diffuse coronary thrombosis [8]. Other data suggested that a combination of hypercoagulation, inflammation, endothelial injury, and thrombus or plaque rupture have an influence on thrombotic complications [9]. All our patients had elevated high-sensitivity cardiac troponin I (TnI). However, there are clear differences in high troponin I in our cases. Two patients had a slight increase in TnI, the third patient had a moderate increase, and in the case of the fourth, TnI was significantly higher than in others (Table 1). Moreover, a multicenter Polish study showed a decreased number of hospital admissions due to fear of COVID-19 during the pandemic, which may have resulted in unfavorable clinical outcomes and increased the risk of AMI complications [10].

Overall, this case series provides valuable insights into potential cardiovascular complications associated with COVID-19, highlighting the need for awareness of thrombotic risks during the course of the disease and further research. However, it is essential to note that this study is limited by its small sample size and lack of a control group. Further investigations with larger cohorts are needed to establish a more robust link between COVID-19 and atypical myocardial infarction.

Article information

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