Case Report

Coronavirus disease 2019 (COVID-19) and simultaneous acute anteroseptal and inferior ST-segment elevation myocardial infarction

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Abstract

Coronavirus disease 2019 (COVID-19) is a recently recognised pandemic spreading rapidly from Wuhan, Hubei, to other provinces in China and to many countries around the world. The number of COVID-19-related deaths is steadily increasing. Acute ST-segment elevation myocardial infarction (STEMI) is a disease with high morbidity and mortality rates, and primary percutaneous coronary intervention is usually recommended for the treatment. A patient with diabetes mellitus and hypertension for five years was admitted to the emergency unit with symptoms of fever, cough and dyspnoea. These symptoms were consistent with viral pneumonia and a COVID PCR test was performed, which tested positive three days later. The patient had chest pain on the eighth day of hospitalisation. On electrocardiography, simultaneous acute inferior and anterior STEMI were identified. High levels of stress and increased metabolic demand in these patients may lead to concomitant thrombosis of different coronary arteries, presenting with two different STEMIs.

Keywords: COVID-19, acute anterior wall myocardial infarction, acute inferior wall myocardial infarction

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Coronavirus disease 2019 (COVID-19) is a recently recognised pandemic spreading rapidly from Wuhan, Hubei, to other provinces in China and to several countries throughout the world.¹ The number of COVID-19-related deaths is steadily increasing.¹ Acute ST-segment elevation myocardial infarction (STEMI) is a disease with high morbidity and mortality rates, and primary percutaneous coronary intervention (PPCI) is usually recommended for the treatment.² Co-existence of cardiovascular disease (CVD) is common in patients with COVID-19 and is related to an increased mortality rate. In this case report, we present a patient who was diagnosed with simultaneous acute anteroseptal and inferior STEMI while he was receiving treatment for COVID-19.

Case report

Our patient (a 55-year-old male, body mass index 26 kg/m²) with diabetes mellitus (DM) and hypertension (HT) for five years was admitted to the emergency unit on 25 March 2020 with symptoms of fever, cough and dyspnoea. He had a history of stable angina pectoris and 2.5 years earlier had had a stent implantation in his left anterior descending (LAD) artery. He had no chest pain on admission.

He was pre-diagnosed with COVID-19 after a thorough physical examination and work-up, including a thoracic computed tomography (CT) scan. There was ground-glass opacity on the CT scan. Azithromycin and oseltamivir treatment were started for COVID-19. His COVID PCR test was positive on 28 March and he was diagnosed with COVID-19. Hydroxychloroquine was added to the treatment regime.

On the morning of 2 April, he experienced chest pain with an increase in dyspnoea. As the level of oxygen saturation decreased, he was intubated. On electrocardiography (ECG), simultaneous acute inferior and anterior STEMI were identified (Fig. 1). It was decided to proceed with the PPCI.

All necessary precautions were taken for viral protection. All laboratory staff wore N95 masks and protective goggles with single-use coveralls with an opening for the face. Additionally,



the performing physician and the technician wore sterile singleuse laboratory coats.

On coronary angiography, the stent in the mid LAD was found to be patent, however it was occluded totally after the stent, and the right coronary artery (RCA) had subtotal occlusion. Initially, a Partner sirolimus-eluting stent of 2.75×15 mm was placed in the mid LAD (Fig. 2). After stent implantation, some blood flow was observed beyond the occlusion, however, a thrombotic total occlusion occurred in the distal LAD. Repeated dilatations were performed on this site, however no blood flow was observed. Two Boston Scientific Rebel bare-metal stents of 4.0×24 mm and 4.0×16 mm were directly implanted (Fig. 2) into the RCA.

In our hospital we have two catheterisation units. The laboratory used for the intervention was sterilised with disinfectant solutions and ultraviolet light and closed for 48



Fig. 2. A, C. Anglographic views of the feit anterior descending artery before and after primary percutaneous coronary intervention. B, D. Anglographic views of the right coronary artery before and after primary percutaneous coronary intervention.



hours. The patient was taken into the intensive care unit. After the intervention, we observed resolution of ST-segment elevations on his ECG (Fig. 3). On a transthoracic echocardiogram (TTE), the ejection fraction was observed at 45% with apical akinesia. In his control TTE performed on 6 April, we observed similar findings after the intervention. The patient was discharged as healthy on 21 April.

Discussion

COVID-19 is a pandemic spreading over approximately 208 countries/regions. By 6 April 2020, more than 1 287 742 patients had become infected worldwide and it had caused the death of more than 70 000 people.

The prevalence of CVD is higher among those with COVID-19, and myocardial injury occurs in more than 7% of patients due to the infection (22% of them critically ill).³ COVID-19 infection can directly affect the cardiovascular system and the presence of CVD also facilitates COVID-19 infection.⁴ The Chinese Centre for Disease Control and Prevention reported in the recently published largest case series in mainland China that the overall mortality rate was 2.3% (1 023 deaths in 44 672 confirmed cases); however, the mortality rate increased up to 10.5% in patients with a history of CVD.⁵

STEMI is still a significant underlying factor for increased morbidity and mortality rates all around the world, although there has been a decrease in incidence and an increase in survival rates recently.⁶ Thrombotic occlusion occurs in a coronary artery at the site of a ruptured or eroded plaque and it leads to STEMI.⁶ Characteristic symptoms, and changes in the ECG are the basis for diagnosis, and elevated cardiac enzymes subsequently confirm the diagnosis.⁶ If it is performed by experienced specialists timeously, mechanical reperfusion via PPCI is superior to fibrinolytic therapy.⁶ For patients with COVID-19, there are two possible pathophysiological mechanisms: (1) type-I MI caused by anxiety-induced catecholamine discharge, prothrombotic system activation triggered by severe inflammatory activation, and plaque rupture, and (2) type-II MI caused by decreased oxygen delivery due to acute inflammation, respiratory failure and hypoxia.^{7,8}

Moreover, Huang *et al.* stated that high concentrations of interleukin-1 β , interferon- γ (IFN- γ), IFN- γ -inducible protein 10 and monocyte chemoattractant protein-1 could be observed in COVID-19 patients, thereby leading to activated T-helper-1 cell responses.^{9,10} It was also suggested that invasion by the virus of angiotensin converting enzyme-II, which is abundantly present in myocytes and vascular endothelial cells and is also the binding site of the coronavirus, may be the direct cause of cardiac involvement.⁹

In a published study, it was reported that a total of 40% of patients with positive test results for COVID-19 had cardiovascular or cerebrovascular disease and 7% had the possibility to develop acute cardiac injury. In case reports, on the other hand, the first causes of admission to hospital due to COVID-19 were heart failure, acute MI, myocarditis and sudden cardiac arrest.¹⁰⁻¹² Shi *et al.* found that 19.7% of patients experienced cardiac injury, and the report showed for the first time that cardiac injury was independently related to an increased risk of mortality in patients with COVID-19 infection.¹³

It was also reported that in a significant number of patients with COVID-19 infection, levels of high-sensitivity cardiac troponin were increased.¹⁴ In a retrospective analysis of 191 patients hospitalised due to COVID-19, it was observed that levels of troponin were increased in more than 50% of patients who died.¹⁴ In other words, increasing levels of troponin in patients presenting with COVID-19 infection is an important indicator of mortality.¹⁴

Li *et al.* reported in a meta-analysis of six studies including 1 527 patients that the prevalence of CVD in patients with COVID-19 was 16.4%.¹⁵ The prevalence of CVD was higher in patients who required intensive care than those who did not require it.¹⁵ It was also reported that at least 8% of the patients were troponin positive.¹⁵

In their analysis, Guo *et al.* showed that while in-hospital mortality rates of intensive care patients who were without CVD and had normal troponin levels was 7.62%, it was 69.44% in patients with known CVD and high troponin levels.¹ In a study evaluating 187 patients, troponin elevation, which is an indication of myocardial injury, was observed in 52 (27.8%) patients, and while the mortality rate was 59.6% in patients with high troponin levels.¹

For the first time, acute MI was identified at the autopsy of a 53-year-old woman with chronic renal failure in Jinyintan Hospital (data not published; obtained via personal communication with a pathologist from the Chinese Academy of Science).¹⁴

In this study, we present a case of MI with anterior and inferior ST elevation on ECG, with the sudden onset of chest pain and increasing dyspnoea while receiving treatment for COVID-19. These patients may be hypoxic due to pneumonia, and both catecholaminergic discharge due to stress and plaque rupture with prothrombotic system activation induced by inflammatory activation related to COVID-19 may develop and result in STEMI. Moreover, in these patients, increased thrombophilia and arterial and venous embolisms are observed.

Our case presented with HT and DM, however, there was no history of chest pain or dyspnoea. On the fourth day of hospitalisation, sudden onset of chest pain with ST-segment elevation in the anteroseptal and inferior leads on ECG were observed. The Turkish Society of Cardiology (TSC), in its recently published expert opinion report, recommended thrombolytic therapy as the first option during STEMI.⁴ They also recommended PPCI in cases of wide anterior wall infarcts.⁴ We considered PPCI in our patient because of ST elevations in the V1-6 and inferior leads on ECG, in agreement with the recommendations of the TSC. PPCI was performed with coronary angiography for the LAD and right coronary arteries.

In COVID-19 patients, the incidence of CVD together with multiple cardiovascular risk factors is high. It is difficult to evaluate chest pain in these patients as they are isolated in intensive care units and the number of intubated patients is high. Moreover, ECG and TTE are performed less often due to strict isolation of these patients. In retrospective analysis, troponin was determined to be positive in approximately half of the deceased patients. With co-morbid coronary artery disease and positive troponin results, these patients were identified as a group with a significantly higher mortality rate.

Conclusion

Serial ECG monitoring and performing echocardiography as required in these patients in the intensive care unit could help to diagnose STEMI or non-STEMI accurately. A true diagnosis of MI may lead to the administration of appropriate treatment and lowering of mortality rates. On the other hand, a diagnosis of MI during an autopsy suggests that the reported rate of MI diagnosis is lower than the actual rate. High levels of stress and increased metabolism in these patients may lead to thrombosis of several coronary arteries with the concurrent occurrence of two different STEMIs.

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Aspirin use best for those with high coronary calcium, low risk of bleeding

An X-ray test, commonly used to assess hardening of the arteries, could help doctors decide whether the benefits of taking aspirin to prevent a first heart attack or stroke outweigh the risks of bleeding from its use, UT Southwestern research suggests. The findings could give doctors and patients more concrete guidelines for making this important decision.

Due to its anti-clotting properties, aspirin is widely prescribed as a preventative measure to patients who have already had cardiovascular events, such as a heart attack or ischaemic stroke. However, aspirin's role in primary prevention – averting first heart attacks and strokes – has been unclear, explains study leader Dr Amit Khera, professor of internal medicine.

After decades of commonly prescribing aspirin for primary prevention, recent guidelines from the American Heart Association (AHA) and American College of Cardiology (ACC) recommend more select use for those with the highest risk of cardiac events due to the increased risk of bleeding.

'We used to say for aspirin, generally yes, occasionally no,' Khera says. 'With these new guidelines, we've flipped that on its head and are saying that we should not use aspirin for most people in primary prevention.'

However, he adds, 'it's been unclear how to select which patients might still benefit most from aspirin therapy, taking into account the risk of bleeding. We need tools to find that sweet spot where aspirin is most beneficial and offsets the associated risks,' he says. In the study, Khera and his colleagues looked to a diagnostic test – coronary artery calcium (CAC) scanning – to see if it could help doctors make this important decision. CAC scanning, a CT scan that scores the amount of calcium that lines the heart's arteries, is commonly performed to detect hardening of the arteries and risk of a heart attack or stroke.

The researchers gathered data from the Dallas Heart Study, an ongoing study that tracks the development of cardiovascular disease in more than 6 000 adults in Dallas County. Initially, participants were invited to three visits for the collection of health and demographic information, laboratory samples and various imaging studies, including CAC scanning. These volunteers were then followed for 12 years on average to track those who had heart attacks, died from heart disease, or had a non-fatal or fatal stroke – medical problems collectively called atherosclerotic cardiovascular disease – and/or who had a bleeding event that caused hospitalisation or death.

The researchers used data from 2 191 participants with a mean age of 44 years who had CAC scans and follow-up information available. About 57% were female and 47% were black.

Overall, about half of the participants had a CAC score of 0, suggesting little to no calcium build-up in their arteries. About 7% had a CAC score of more than 100, suggesting heavy calcium build-up. The rest had values in the middle (1–99).

When Khera and his colleagues examined the rates of atherosclerotic cardiovascular disease (ASCVD) and bleeding in the study group, they found that both events increased in a graded fashion as CAC scores rose. However, when they used statistical modelling to see how many of the ASCVD events may have been prevented by aspirin use – based on values gleaned from a recent meta-analysis that informed the AHA and ACC guidelines – they found that aspirin's benefits only outweighed its risks for those with CAC scores above 100. For this group, the risk of ASCVD was about 15-fold and the bleeding risk about three-fold of those with a CAC score of 0.

Yet, this effect only held true for those whose inherent risk for bleeding was already low, Khera says, meaning that in practice, as mentioned in the guidelines, if someone has had prior significant bleeding episodes, risk factors for bleeding, or was on medications that increase bleeding, they should not take aspirin for primary prevention regardless of their CAC score.

Together, Khera says, the findings reinforce new guidelines suggesting that aspirin for primary prevention is only appropriate for select patients and that CAC scanning can help doctors and patients make that decision.

'Aspirin use is not a one-size-fits-all therapy,' says Khera, who holds the Dallas Heart Ball chair in hypertension and heart disease. 'CAC scanning can be a valuable tool to help us tailor care to help more patients avoid a first heart attack or stroke.'

Source: Medical Brief 2020