An indirect effect of covid-19 on the heart: A case report

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Abstract
Covid-19 virus has been recognized to develop major cardiac complications. The indirect effect of the virus though, in terms of fear for hospital admissions, constitutes a greater threat. In this case we present a 69-year old male patient who suffered from a myocardial infarction that has not been given the proper attention due to the fear of in-hospital contact with covid patients. The result was the delayed revascularization and eventually the development of heart failure. This case presents the full range of covid-19 affection to the heart and raises the public awareness for not underestimating symptoms suggesting life-threatening conditions.

Keywords
Acute myocardial infarction, covid-19, heart failure, case report

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Avoiding cardiovascular complaints and symptoms due to fear of COVID 19 transmission can lead to irreversible cardiac damage and more severe consequences.

Background
The global Severe Acute Respiratory Syndrome coronavirus (SARS – CoV2/COVID-19) pandemic, which broke out in the city of Wuhan in China in December 2019, has led to a collateral damage in the form of the missed diagnosis of some life-threatening cardiovascular disorders. We present here a case of a 69-year old male patient who was admitted into our center with symptoms of decompensated heart failure as a result of an extensive anterior myocardial infarction. The patient underestimated his symptoms and he was advised not to go to the hospital to avoid direct contact with SARS-CoV2 infection.

In this unprecedented pandemic, as healthcare professionals committed to providing the patient tailored, most appropriate and accurate approach in each different individual; our main goal should be to preclude patients from going to the hospital needlessly; nevertheless, it is paramount to enhance their level of awareness regarding serious symptoms that could be clue to life-threatening conditions.

Case presentation
A 69-year-old male patient with history of hypertension, dyslipidemia and smoking presented to our outpatient department with progressive shortness of breath over the last 5 days. The symptoms started with an epigastric pain a week earlier, yet his family

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advised him against going to hospital assuming that the symptoms are more likely gastrointestinal or anxiety related. During the following days, his symptoms progressively worsened and he started to develop exertional dyspnoea and orthopnoea respectively. On admission, his clinical examination revealed a pathologic S3 sound and rales bilaterally to the lungs, ECG demonstrated anteroseptal pathological Q waves with normal (Figure 1) and transthoracic echocardiography (TTE) revealed severely reduced left ventricular (LV) systolic function with an estimated ejection fraction of around 30–35%, akinetic LV apical segments, severely hypokinetic anterior segments with an accompanying lobulated apical LV thrombi protruding into LV cavity: a larger one measuring 4×2 cm, and a smaller one measuring 1.5×1.5 cm (Figure 2). The TTE and ECG findings were consistent with a recent extensive anterior wall myocardial infarction. The patient was hospitalized due to pulmonary congestion and concomitant renal insufficiency and was scheduled for a coronary angiogram.

After cautious optimization over the first day, the patient was totally asymptomatic and hemodynamically stable. On the next day, a coronary angiogram demonstrated a critical stenosis in the right coronary artery (RCA) and a total occlusion of the left anterior descending artery (LAD). Our interventional cardiology team decided for total revascularization of the patient. After the interventions, he was started on warfarin with a target International Normalized Ratio (INR) level of 2–3 and the patient was discharged home few days later on warfarin, dual antiplatelets and heart failure medication. At one-month follow up he remained asymptomatic and control TTE revealed dissolution of the LV thrombus without a significant LV systolic function improvement, thus an an Implantable Cardioverter Defibrillator (ICD) insertion was planned.

**Discussion and conclusions**

Although COVID-19 primarily affects the respiratory system, cardiovascular involvement may also occur due to the suggested pathophysiological role of the Renin Angiotensin System/Angiotensin Converting Enzyme 2 (RAS/ACE2) in the disease process. SARS-CoV2 has been shown to have a direct impact on endothelial function, therefore microvascular dysfunction or thrombosis caused by hypercoagulability can result in myocardial ischemia and related myocardial infarction. Moreover, immune system activation and proinflammatory cytokine release may result in plaque instability, bringing along acute coronary syndromes (ACS). In addition, a significant increase in cardiac output would be needed due to respiratory failure and hypoxia caused by COVID-19 which may result in Type II myocardial ischemia.

It is unfortunate that the COVID-19 pandemic has led to a compromise in the timely reperfusion of STEMI patients as we experienced in this case. Although global healthcare services provision and delivery are currently focused on the COVID-19
pandemic, we must be aware of the collateral damage which could happen if we don’t deliver other life-saving evidence-based treatments to our patients with cardiovascular diseases.¹

According to an extensive worldwide survey by the European Society of Cardiology (ESC), there is a major decline (more than 50%) in the number of hospital admissions and thus treatments for all subtypes of ACS, since the beginning of the COVID-19 pandemic.²

This observation could be explained by several patient and system-related factors. The most common factors that prevented patients from seeking life-saving treatments in the hospitals are the strict ‘stay-at-home’ instructions, as well as the overall public fear of being exposed to the coronavirus. Moreover, ACS-related symptoms such as chest discomfort and dyspnea may overlap with the symptoms of COVID-19 pneumonia and therefore it could be misinterpreted as an acute respiratory infection. Regardless of the causes, the reduced number of ACS patients admitted and treated may be accompanied by a significant increase in the early and late infarction-related morbidity and mortality.³,⁴

A recent survey found a 28% increase in life-threatening complications among patients with ACS during the pandemic. It is obvious that the risk of dying of a heart attack is much greater than that of SARS-CoV2 infection complications. Moreover, cardiac mortality can largely be preventable if patients with myocardial infarction are provided timely and accurate treatment.⁵ Delayed presentation with late reperfusion is often associated with an increased risk of myocardial disruption including free wall rupture, ventricular septal defect, acute papillary muscle tearing and LV thrombus formation giving rise to a high mortality and morbidity risk. Our case reflects the ambiquity in the mind of people regarding hospital admission in emergent, life threatening conditions and depicts the complexity and importance of managing ACS and STEMI during the COVID-19 pandemic. We are strongly of the opinion that governments, medical societies, heart foundations and social media have a pivotal role, not only to develop strategies to inform patients of the increased contagion risk due to unnecessary hospital admissions but also to guide public clearing up the symptoms of life threatening emergent conditions. It is important to ensure that a safe environment is provided for patients who are admitted to the hospital in case of a cardiovascular emergency amidst the COVID-19 crisis and disinformation and confusion is avoided.

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