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As the COVID-19 pandemic drags on, where have all the STEMIs gone?

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As the COVID-19 pandemic drags on, where have all the STEMIs gone?

To the Editor,

In the midst of current pandemic individuals with all sorts of medical ailments are afraid to venture into health-care settings and risk contracting Coronavirus disease 2019 (COVID-19). Irrespective of the inherent thromboembolic risks associated with COVID-19, this puts patients at high risk of cardiac complications.

A 36-year-old male was brought to the emergency department (ED) after being found unresponsive at home. History was limited, given his condition. He was last seen normal over 15 h ago. He had no significant past or family medical history. He denied any use of alcohol or smoking. In the ED, he was febrile (101.2F°) and hypotensive 78/59 mm Hg. His oxygen saturation (SaO2) was 78%. Laboratory investigations revealed leukocytosis (16,760/uL), elevated d-dimer of 48,648 ng/mL (normal < 0.50 ng/ml) and serum troponin-T (TnT) of 1.86 ng/L, which peaked to 44.4 ng/L (normal < 0.10 ng/L). Electrocardiogram revealed ST-segment elevation in the anteroseptal leads (V2-V4). (Fig. 1) Transthoracic echocardiography (TTE) showed extensive septal, anterior and apical akinesia with an apical left ventricular (LV) thrombus and a reduced ejection fraction of 35%. (Fig. 2) He was intubated for hypoxia and was started on norepinephrine infusion for hypotension. His chest x-ray and computed tomography showed multifocal infiltrates. He received aspirin, clopidogrel, atorvastatin and a heparin infusion for a presumed subacute ST-segment elevation myocardial infarction (STEMI). The following day his nasal swab test for real-time polymerase chain reaction (RT-PCR) returned positive for acute respiratory syndrome coronavirus-2 (SARS-CoV-2). He was treated with an 80 mg single dose of tocilizumab and hydroxychloroquine 400 mg twice a day on the first day followed by 200 mg twice a day for the next 4 days. Post-extubation, he revealed that he had been having substernal chest pain for

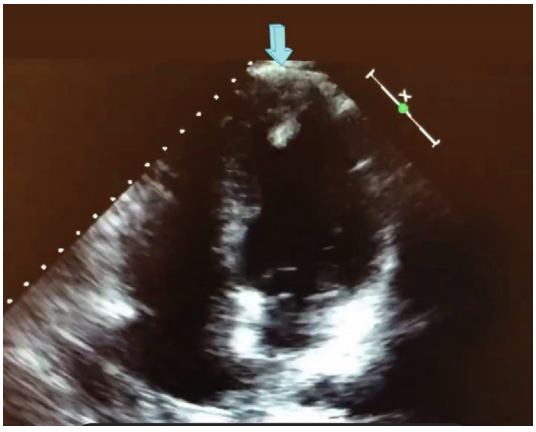

Fig. 2. TEE, apical view showing an apical thrombus (arrow).

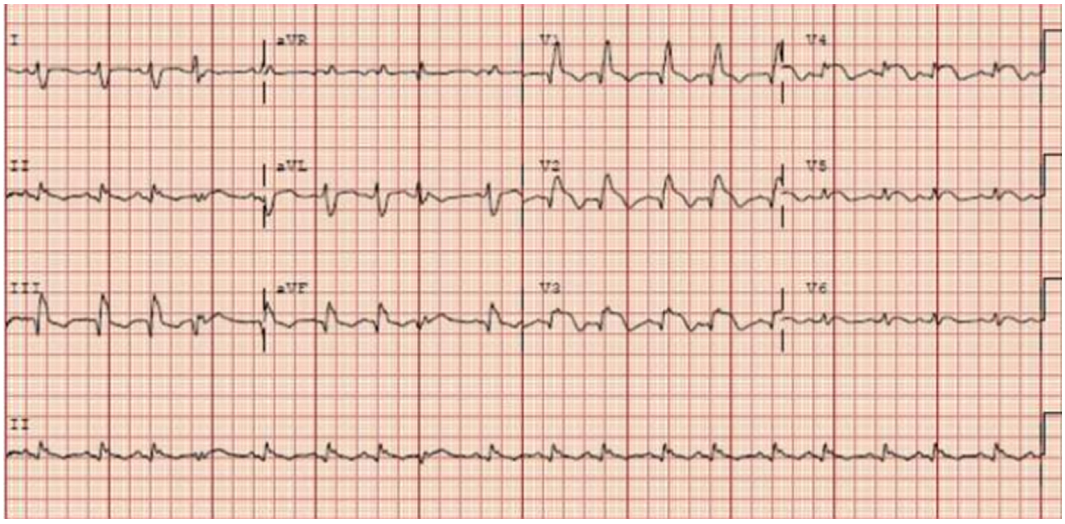

Fig. 1. EKG showing ST-elevation in leads v2-v4 indicating anteroseptal wall myocardial infarction.

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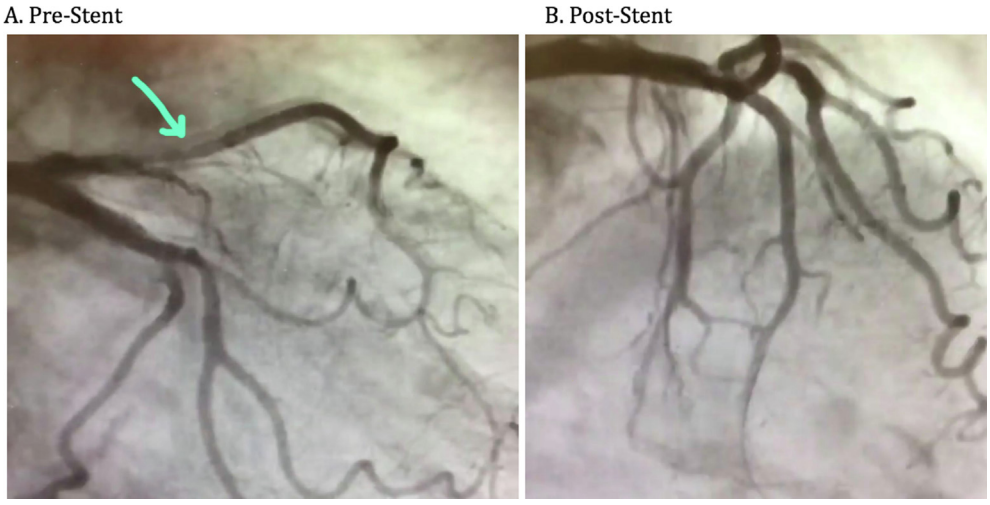

Fig. 3. A. Angiography demonstrating left anterior descending artery occlusion (arrow), B. with an adequate restoration of blood flow post-stenting.

several hours before losing consciousness but had not sought care earlier due to fear of getting COVID-19 in the hospital. He refused further workup and was discharged home with a plan to continue aspirin, clopidogrel, atorvastatin in addition to carvedilol and lisinopril post-discharge. A week later, he had an elective left-sided angiography demonstrating a 99% occlusion of the left anterior descending artery (LAD), a drug-eluting stent was deployed with an adequate restoration of blood flow. (Fig. 3) He was advised to be compliant with his medications and subsequent cardiology follow-ups.

The novel Coronavirus Disease 2019 (COVID-19) is a generation-defining global pandemic; the scale, scope and pace of which is unprecedented. Despite the high rate of cardiovascular complications, contemporary reports show that COVID-19 concerns have prompted patients to delay seeking emergent care, reducing the number of STEMI cases.[1,2] This is presumptively being attributed to patients being reluctant to go to the hospital even in dire circumstances and possibly underdiagnosis in overwhelmed ED's, the former being the case in our patient. [1]

Recently, a United States (US) model from 9 major centers observed a 38% drop in total STEMI activations.[3] This is similar to a 40% reduction noted in Spain.[4] The early Hong Kong experience demonstrated a significant delay in the time of symptom onset to the first medical encounter by about 318 min.[2] These findings are particularly concerning given that COVID-19 itself can be a cause of STEMI due to microthrombi, cytokine storm, coronary spasm, or direct endothelial injury, furthermore acute coronary syndrome occurring in COVID-19 patients has been associated with a worse prognosis. [5,6] Reluctance to visit a hospital due to fear of morbidity related to COVID-19 only adds to time delays in managing STEMI due to increased precautionary measures and accessory testing in the ED, delayed triaging, short staffing and slow activation of cardiac catheterization labs, putting patients at risk of worse clinical outcomes.[2]

The specific treatment protocols for STEMI patients with COVID-19 have been evolving. Early recommendations from the Wuhan experience included intravenous thrombolysis as first-line therapy for STEMI patients with confirmed COVID-19 given that most hospitals do not have protected cardiac catheterization labs and coronary care units for respiratory borne illnesses.[7] More recently, the Society of Cardiovascular Angiography and Intervention (SCAI) recommendations favored continuation of the current standard of care (percutaneous intervention) in COVID-19 patients, though recognize that this may change if hospital systems become further overwhelmed.[8]

In our case, the collision between COVID-19 induced STEMI and a reluctance to seek urgent care led to catastrophic complications. The reduced ejection fraction, cardiogenic shock and the early development of an LV thrombus seen in this case can be attributed to not only the late presentation but perhaps also a consequence of COVID-19 specific hypercoagulable state. Elevated D-dimer, raised troponin levels and EKG abnormalities prompted a high suspicion for ACS even in a hitherto healthy person with no conventional cardiovascular risk factors.

While remarkable public health efforts are ongoing, we advocate for increased public awareness of cardiovascular complications of COVID-19 and the need to seek early medical attention.

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Industrial Relationship/Financial Disclosure: None

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcha.2020.100550.

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