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Case Reports

Delayed Treatment of Acute Myocardial Infarction With Ventricular Septal Rupture Due to Patient Fear During the COVID-19 Pandemic

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During the coronavirus disease 2019 (COVID-19) pandemic, more patients are presenting with complications late after acute myocardial infarction. We report the case of a 71-year-old man who delayed seeking medical care for 2 weeks, despite progressive shortness of breath, cough, and tactile fever, for fear of contracting COVID-19 in the hospital. Clinical and echocardiographic evaluation revealed a ventricular septal rupture secondary to acute myocardial infarction. The patient underwent urgent cardiac catheterization, followed by successful saphenous vein grafting to the left anterior descending coronary artery and open surgical repair of the ventricular septal rupture with a bovine pericardial patch. This case highlights a potential long-lasting negative effect that the COVID-19 pandemic will have on the care-seeking behavior and health of patients with acute cardiovascular disease. ***(Tex Heart Inst J 2021;48(3):e207356)***

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The coronavirus disease 2019 (COVID-19) pandemic has placed great strain on healthcare systems around the world. It has also affected the care-seeking behavior of patients and their perceptions of healthcare facilities, especially among patients who may already be prone to stress, such as those with coronary artery disease.[1] Early in the COVID-19 pandemic, presentations for ST-segment-elevation myocardial infarction in the United States decreased by an estimated 40%,[2,3] while in New York City the number of out-of-hospital cardiac arrests increased 6- to 10-fold.[4] Fear of infection has discouraged some patients with potentially life-threatening cardiovascular conditions, such as acute myocardial infarction (AMI) and its associated complications, from seeking treatment.[5] Social distancing and isolation from family and friends have also made recognition of alarming cardiovascular symptoms by a second party more difficult.[6] Meanwhile, early presentation and timely reperfusion remain key to the effective treatment of AMI. We report the case of an elderly man who presented with a ventricular septal rupture (VSR) late after AMI.

Case Report

In March 2020, a 71-year-old man presented at his pulmonologist's office in New York City with a 2-week history of progressive shortness of breath, cough, and tactile fever. His medical history included chronic obstructive pulmonary disease (COPD); hyperlipidemia; anxiety disorder; and until 15 years previously, a 20-pack-year history of smoking. The patient, a practicing physician, had delayed seeking care because of concerns about contracting COVID-19 during the recent outbreak in the region. He reported that his symptoms worsened with activity and were not relieved by his usual COPD medications (fluticasone propionate/salmeterol and albuterol), but that the symptoms were not accompanied by chest pain, nausea, or vomiting. His pulmonologist immediately referred him to our hospital's emergency department (ED) for evaluation.

Upon arrival at the ED, the patient was hypoxic and had a respiratory rate of 30 breaths/min, a blood pressure of 150/115 mmHg, and a heart rate of 124 beats/min. Supplemental oxygen (4 L/min) was given through a nasal cannula to maintain an oxygen saturation level of 92%. Physical examination revealed an elevated jugular venous pressure, a grade III holosystolic murmur across the precordium, bibasilar rales, and mild (1+) pitting edema on both ankles. Laboratory values were as follows: sodium, 132 mEq/L (reference range [RR], 136–145 mEq/L); creatinine, 1.31 mg/dL (RR, 0.7–1.2 mg/dL); aspartate aminotransferase, 114 U/L (RR, 8–33 U/L); alanine transaminase, 91 U/L (RR, 4–36 U/L); troponin T, 1.630 ng/mL (RR, <0.03 ng/mL); N-terminal pro-brain natriuretic peptide, 8,539 pg/mL (RR, <125 pg/mL); white blood cells, 14.27 × 10^3/μL (RR, 4.5–11 × 10^3/μL); platelets, 263 × 10^3/μL (RR, 150–450 × 10^3/μL); lymphocytes, 9%; monocytes, 4%; segmented neutrophils, 87%; procalcitonin, 0.06 ng/mL (RR, <0.15 ng/mL); low-density-lipoprotein cholesterol, 139 mg/dL (RR, <100 mg/dL); hemoglobin A1c, 6.2% (RR, <6.4%). Arterial blood gas analysis during the administration of supplemental oxygen revealed the following: pH, 7.499; PCO_2, 19 mmHg; PO_2, 72.1 mmHg; HCO_3, 14.6 mmol/L; oxygen saturation, 94%.

Our initial differential diagnosis included viral and bacterial respiratory tract infection and pneumonia, as well as suspected COVID-19 infection; AMI with possible mechanical complications or decompensated heart failure; and COPD exacerbation.

A 12-lead electrocardiogram showed sinus tachycardia (124 beats/min); right bundle branch block; ST-segment elevation in leads II, III, and aVF; and ST-segment depression in leads V_4 through V_6 (Fig. 1A). A chest radiograph showed ill-defined bilateral interstitial opacities (consistent with atypical pneumonia or edema) and mild cardiomegaly (Fig. 1B).

Respiratory viral tests were negative for influenza A and B and for respiratory syncytial virus. Results of reverse transcription polymerase chain reaction (RT-PCR) testing for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) were unavailable (at the time, RT-PCR turnaround time at our hospital was 2 to 3 days). An urgent transthoracic echocardiogram (TTE) revealed moderately reduced left ventricular (LV) function (ejection fraction, 40%), LV inferior and inferoseptal wall akinesis, right ventricular (RV) dilation, severely reduced RV function, and RV free wall akinesis (Fig. 2). The TTE also showed mid-to-basal septal rupture (Fig. 3) and left-to-right shunting (Fig. 4), findings consistent with a VSR. The color-flow Doppler images also showed mild mitral regurgitation, but no evidence of papillary muscle or chordae rupture. The estimated pulmonary artery systolic pressure was 55 mmHg.

Droplet and contact precautions were implemented

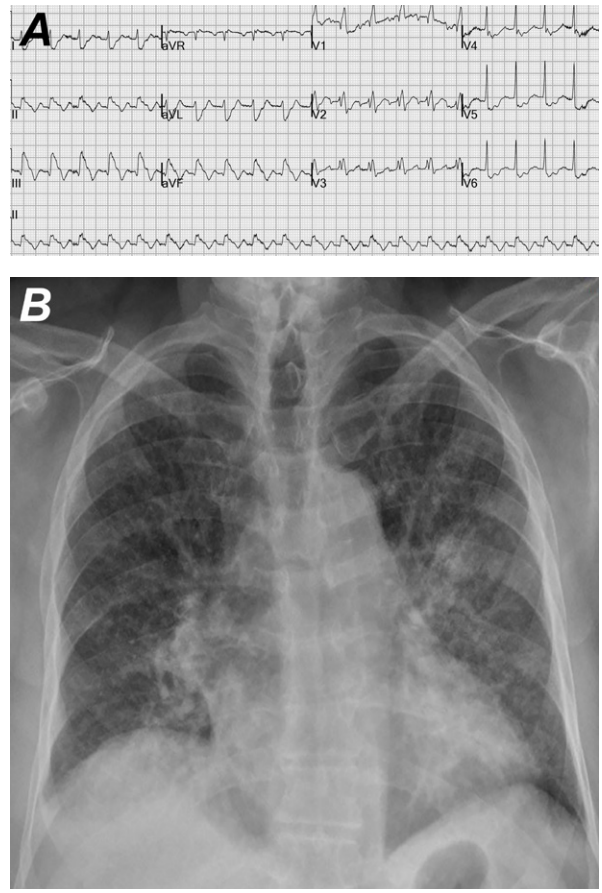
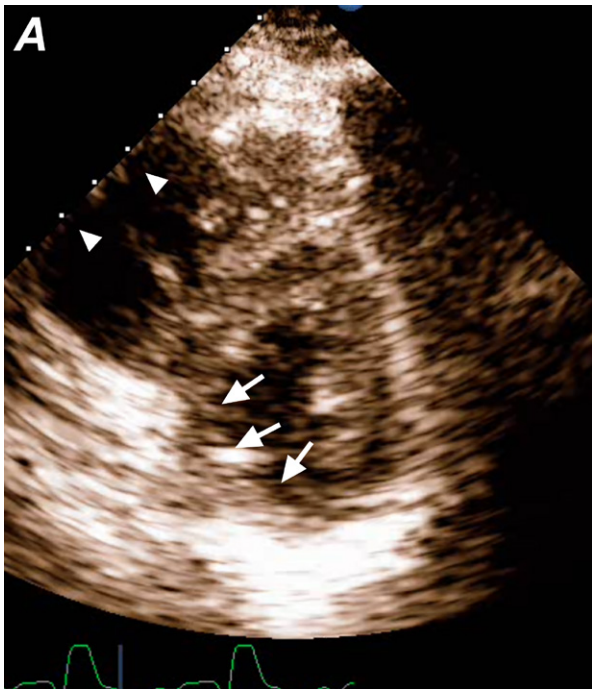


Fig. 1 ***A****) 12-lead electrocardiogram obtained at presentation shows sinus tachycardia; right bundle branch block; ST-segment elevation in leads II, III, and aVF; and ST-segment depression in leads V_4 through V_6.* ***B****) Chest radiograph (anteroposterior view) shows ill-defined bilateral interstitial opacities.*

while awaiting the patient's RT-PCR test results for SARS-CoV-2. The patient was given azithromycin intravenously to treat suspected bacterial pneumonia; 325 mg of aspirin orally, 80 mg of atorvastatin orally, and heparin intravenously according to institutional guidelines to treat AMI; and 40 mg of furosemide intravenously to treat pulmonary edema. Nevertheless, the patient's condition deteriorated rapidly and progressed to respiratory failure and cardiogenic shock.

The patient was taken urgently to the cardiac catheterization laboratory, where he was sedated and intubated endotracheally. He then underwent left- and right-sided heart catheterization and intra-aortic balloon pump (IABP) insertion. Coronary angiograms revealed multivessel coronary artery disease including severe (90% to 95%) stenosis of the proximal and mid left anterior descending coronary artery (LAD); chronic total occlusion of the mid left circumflex coronary artery; severe (95%) stenosis of the mid right coronary artery (RCA); and subtotal occlusion (>99%) of the distal RCA before the bifurcation of the right posterolateral branch and posterior descending coronary artery



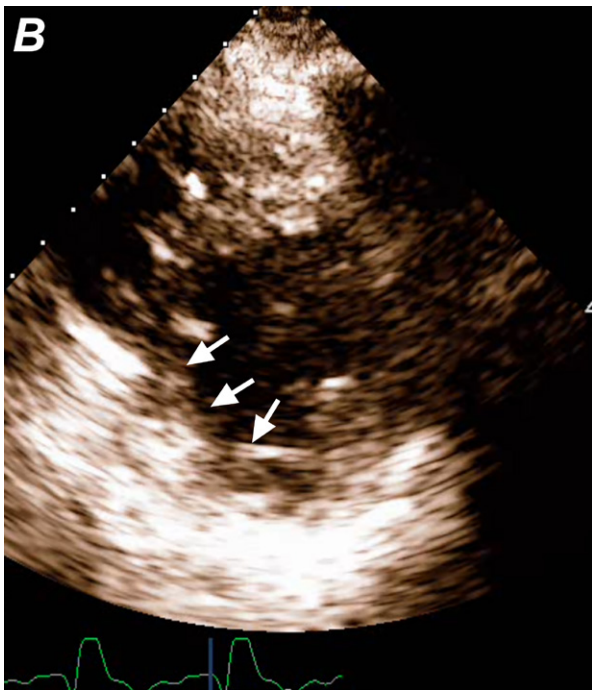


Fig. 2 *Transthoracic echocardiograms obtained in **A**) systole and **B**) diastole (parasternal short-axis mid-cavity view) show a dilated right ventricle with an akinetic free wall (arrowheads) and a contracted left ventricle with akinetic inferior and inferoseptal wall segments (arrows).*

Supplemental motion image is available for Figure 2.

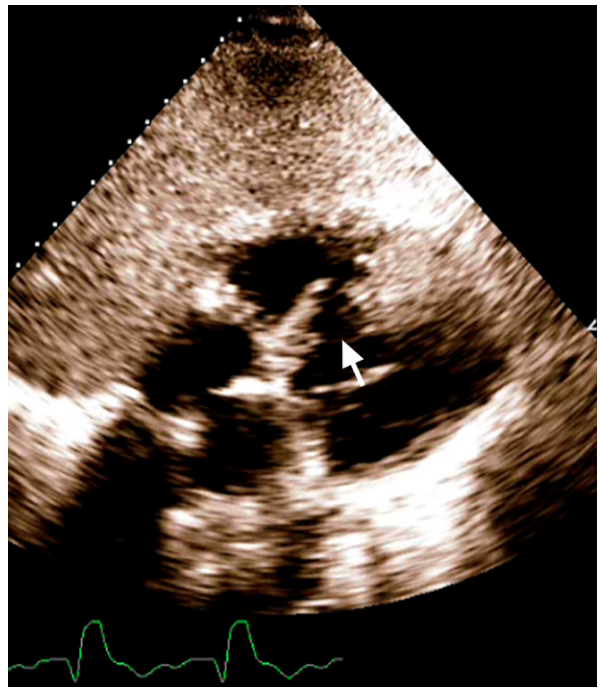


Fig. 3 *Transthoracic echocardiogram (subcostal view) shows a ruptured interventricular septum (arrow).*

Supplemental motion image is available for Figure 3.

(Fig. 5). We considered the RCA lesions the likely cause of the AMI. Contrast filling of the RV seen on a left ventriculogram was consistent with a VSR. Right-sided heart catheterization revealed a mean right atrial pressure of 24 mmHg, an RV pressure of 72/21 mmHg, a pulmonary artery pressure of 65/26 mmHg (mean, 41 mmHg), a pulmonary capillary wedge pressure of 22 mmHg, and an LV end-diastolic pressure of 25 mmHg. Oximetry revealed an oxygen saturation step-up (from 62.7% in the right atrium to 88.7% in the RV and 85.6% in the PA), with a calculated Qp/Qs ratio of 1.9.

After discussions among the cardiac care team, the patient's family, and his pulmonologist, the patient underwent emergency bypass with a saphenous vein graft to the LAD and open repair of the VSR with a bovine pericardial patch. The single-vessel bypass approach was used to protect the LAD territory and to minimize on-pump time. The patient's postoperative course was complicated by vasoplegia and RV failure, which necessitated substantial hemodynamic support with an IABP, inotropic agents, and vasopressors. A test for SARS-CoV-2 on postoperative day 2 was negative.

The patient was prescribed low doses of a β-adrenergic receptor blocker on postoperative day 10 and an angiotensin-converting enzyme inhibitor on postoperative day 12, and he tolerated both well. He was discharged from the hospital on postoperative day 14 and continued taking aspirin and atorvastatin. At his 2-month telehealth follow-up visit, the patient was doing well and maintaining a self-managed physical therapy program. At his last follow-up visit in April 2021, the patient remained well.

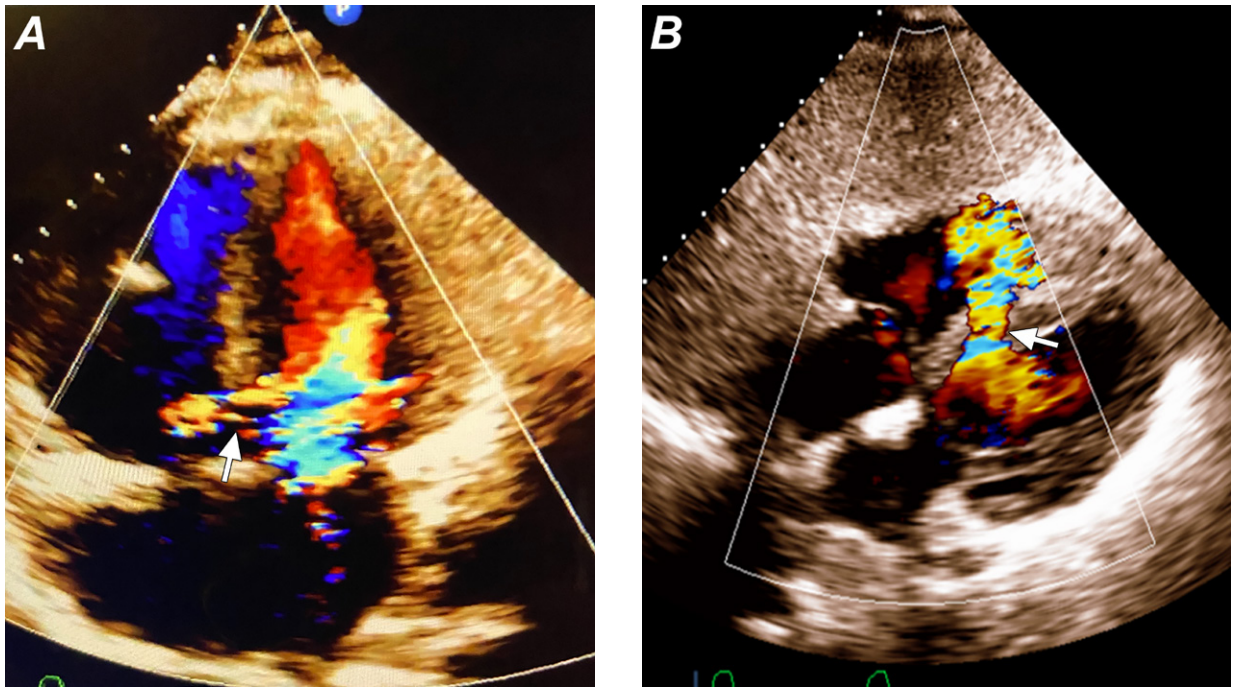


Fig. 4 *Transthoracic echocardiograms (color-flow Doppler mode) in **A**) apical 4-chamber and **B**) subcostal views show left-to-right shunting through a ventricular septal rupture (arrows).*

Supplemental motion image is available for Figure 4B.

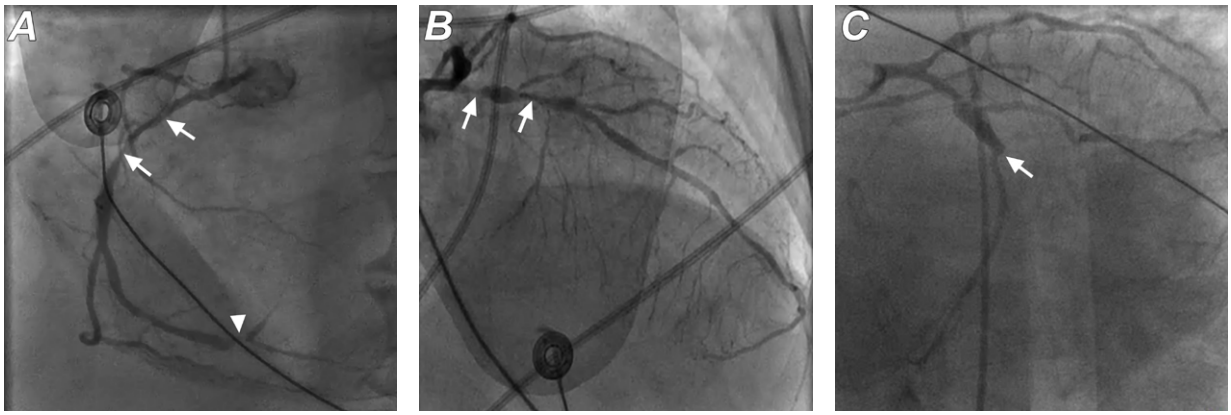


Fig. 5 *Coronary angiograms show **A**) severe stenosis in the mid right coronary artery (RCA) (arrows) and subtotal occlusion of the distal RCA (arrowhead) (left anterior oblique view), **B**) severe stenosis in the proximal and mid left anterior descending coronary artery (arrows) (right anterior oblique/cranial view), and **C**) chronic total occlusion of the distal left circumflex coronary artery (arrow) (anteroposterior/caudal view). In **A**), note the location of the subtotal occlusion of the distal RCA (arrowhead) just before the bifurcation of the right posterolateral branch and posterior descending coronary artery, with Thrombolysis In Myocardial Infarction (TIMI) 1 flow through the culprit lesion.*

Discussion

Early presentation and timely reperfusion are necessary for the effective treatment of AMI. In this case, however, the patient waited 2 weeks before seeking medical treatment for what turned out to be symptoms of AMI complicated by a VSR; he did not seek immediate treatment because he had no classic symptoms of angina pectoris or AMI and because he feared contracting COVID-19 in the hospital. Only after presenting at his pulmonologist's office and the ED was his dangerous condition diagnosed.

As the COVID-19 crisis has evolved, our hospital has implemented and maintained a policy to reduce the risk of nosocomial COVID-19 and address the related concerns of healthcare workers and patients. We require that one senior, experienced member of the care team interview and thoroughly examine and evaluate each

patient suspected of having COVID-19 at presentation. In this case, and in accordance with this policy, initial examination and evaluation of the patient by the senior fellow on the cardiac care team and by the ED attending physician contributed to timely recognition of the mechanical complications of AMI.

The regional wall motion abnormalities, VSR, and hemodynamic instability revealed by clinical and diagnostic TTEs dictated urgent invasive evaluation. However, the patient's respiratory distress and suspected COVID-19 necessitated special precautions. He was intubated endotracheally before cardiac catheterization to reduce the potential risk of virus aerosolization and staff exposure. His case was managed as if he had COVID-19 because prompt diagnosis at our hospital during New York City's COVID-19 outbreak of March 2020 was limited by an RT-PCR turnaround time of 2 to 3 days. His intubation before cardiac catheterization and the use of appropriate personal protective equipment by our cardiac catheterization and cardiac surgery teams were consistent with subsequent recommendations proposed by several organizations for urgent cardiac procedures during the COVID-19 pandemic.[7-9]

Ventricular septal rupture is a rare mechanical complication of AMI, resulting from ischemic necrosis and consequent rupture of the interventricular septum. Patients with AMI who are older are at increased risk of VSR, as are those who present late or who undergo delayed, unsuccessful, or no reperfusion therapy.[10,11] The prognosis of VSR after AMI is poor. Early diagnosis requires heightened clinical awareness, including recognizing harsh pansystolic murmurs, noting evidence of pulmonary congestion, and carefully evaluating the results of early imaging studies, such as TTE in color-flow Doppler mode. Affected patients often present with cardiogenic shock and respiratory failure that necessitate mechanical circulatory support with an IABP, extracorporeal membrane oxygenation, or an LV assist device as a bridge to transplant. Determining the best approach to revascularizing affected coronary arteries and definitively treating the VSR—whether by percutaneous coronary intervention and closure device treatment, or by coronary artery bypass grafting and open repair—is best done by a cardiac care team.[10]

Conclusion

The COVID-19 pandemic has posed unique challenges in evaluating and treating patients with severe cardiac conditions. Timely recognition and treatment of potential fatal mechanical complications of AMI require prompt clinical and echocardiographic diagnosis, as well as systematic protocols for appropriately selecting patients for invasive evaluation and treatment and mitigating healthcare worker exposure. Educating the general public about appropriate care-seeking behaviors and symptom recognition is essential.

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