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Post-Myocardial Infarction Complications During the COVID-19 Pandemic – A Case Series



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ABSTRACT

We report 4 cases of post myocardial infarction complications due to the delay in presentation during COVID-19 era. We highlighted the need for auscultating the chest for early diagnosis. Through this case series, we urge to raise awareness among cardiac patients to access healthcare despite the fear of COVID-19.

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1. Case #1

1.1. Medical history

A 68-year old female presented on 04/06/2020 with presyncope. A week ago, she developed right shoulder pain with radiation to her arm but did not seek medical attention due concerns for COVID-19 exposure. Initial vital signs on presentations were blood pressure of 143/38 mmHg, heart rate 82 bpm and saturating 95% on 4 L nasal canula.

1.2. Investigations

Electrocardiogram (EKG) was concerning for a posterior myocardial infarction (Fig. 1.1). Laboratory results showed troponin I of 4.42 ng/mL, serum lactic acid 8.4 mmol/L. COVID-19 PCR was negative. Bedside transthoracic echocardiogram showed a hyperdynamic left ventricle (LV), a large hemopericardium with signs of tamponade, and basal to mid posterior wall akinesis (Video 1).

1.3. Management

She subsequently became hypotensive and was started on norepinephrine drip after 2 L saline infusion. She was emergently taken to the cardiac catheterization laboratory where coronary angiography revealed a 100% occlusion of an inferior distal branch of a large second obtuse marginal (Fig. 1.2). Table 1 show the hemodynamics. An intra-aortic balloon pump (IABP) was placed. Due to concern for potential ventricular wall rupture secondary to late presenting posterior MI, patient was taken to the operative room emergently and underwent a subxiphoid pericardial window with evacuation of 700 mL of old clotted blood.

1.4. Follow-up

Patient's hemodynamic status improved immediately. Norepinephrine and IABP were weaned off. Post-operative TTE showed LV ejection fraction (LVEF) of 35–40% with akinesis of the posterior wall. Pt was eventually discharged home on 04/14/2020.

2. Case #2

2.1. Medical history

A 72-year-old female presented on 04/15/2020 at a peripheral hospital with acute onset of weakness, shortness of breath followed by a

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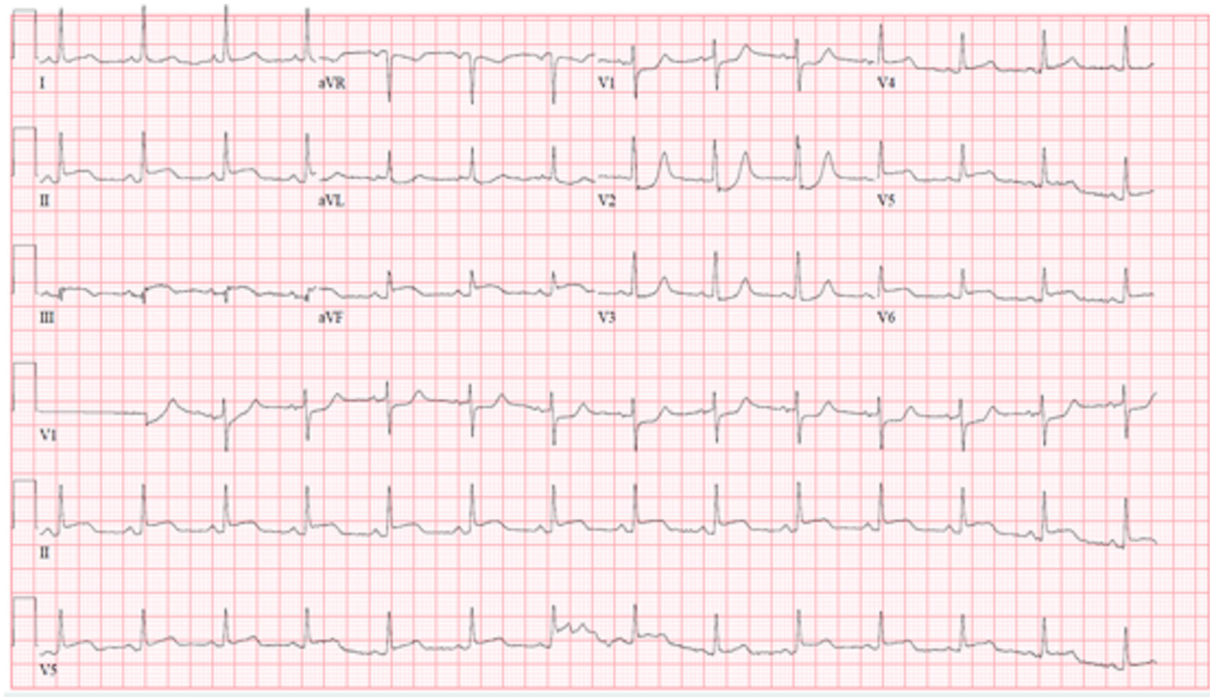


Fig. 1.1. Electrocardiogram (EKG) of Case #1 showing posterior myocardial infarction.

mechanical fall. Prior to her presentation, patient reported four-day history of intermittent shortness of breath with exertion. Vital signs were stable except tachycardia. Physical exam was notable for a holosystolic murmur heard at the left lower sternal border and lungs were clear to auscultation.

2.2. Investigations

Initial EKG showed remote inferior infarct and anterolateral ST segment elevations (Fig. 2.1). Laboratory studies showed initial troponin I of 49.2 ng/mL, brain natriuretic peptide 2660 pg/mL, serum lactic acid of 7.3 mg/L, white blood count (WBC) 34,000/mL. COVID-19 PCR was negative.

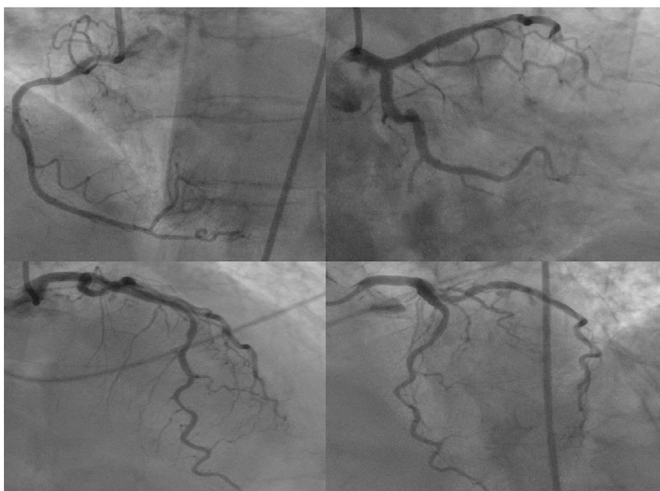


Fig. 1.2. Angiogram of Case #1 showing occlusion of right posterolateral branch of right coronary artery.

2.3. Management

Patient was given aspirin, ticagrelor and heparin and was transferred to the tertiary care center cardiac catheterization laboratory emergently. Coronary angiography revealed serial 70% and 99% stenoses of the mid left anterior descending artery (LAD) (Fig. 2.2). Ventriculography showed LVEF 30% with antero-apical and apical akinesis and evidence of a distal ventricular septal rupture (VSD). Hemodynamics are shown in Table 1. An IABP was inserted for hemodynamic support and cardiac surgery were consulted for VSD repair. TTE obtain showed LVEF 45% with distal LAD territory hypokinesis to akinesis with a distal non-restrictive VSD with left to right shunting (Video 2). Patient underwent a VSD repair with a polytetrafluoroethylene (PTFE) over collagen-impregnated Dacron Hemashield patch with intra-operative echocardiogram showing residual small restrictive VSD. Post operatively, patient developed worsening cardiogenic shock requiring vasopressor and inotropic support over a period of 2 weeks. Repeat TTE after 2 weeks showed persistent leak around the repair site.

2.4. Follow-up

Given worsening hemodynamics, patient underwent a percutaneous closure of VSD using a 16 mm Amplatzer® post infarct VSD occluder after sizing it. Follow up TTE showed persistent left to right shunt suggestive of VSD despite it appearing relatively smaller than before. Patient's clinical status continued to deteriorate with increasing need for hemodynamic support and evidence of multiorgan failure over the course of next week. Care was withdrawn by the family after another week of supportive management and patient expired on 05/12/2020.

3. Case #3

3.1. Medical history

A 53-year-old male presented on 05/30/2020 with 5-day history of progressively worsening chest pain, shortness of breath and

Table 1
Characteristics of case series patients.

Case	Age (years)	Sex	Relevant comorbidities	Symptoms and signs	Hemodynamics	Length of hospitalization	Time from symptom onset to first medical contact	Final diagnosis
1	68	F	Hypertension Diabetes mellitus	Lightheadedness Pre-syncope Shoulder pain	LVEDP 20 mmHg RA 18 mmHg PA 32/19 (24) mmHg PCWP 18 mmHg Fick's CO/CI - 4.6 L/min/2.8 L/min/m ²	10 days	7 days	Ventricular free wall rupture
2	72	F	Hypertension Hyperlipidemia Chronic kidney disease Neurofibromatosis	Shortness of breath Weakness Fall	LVEDP 30 mmHg RA 14 mmHg PA 43/24 (31) mmHg PCWP 28 mmHg CO/CI 7.8 L/min/ 4.2 L/min/m ² Qp/Qs - 2.5	27 days	4 days	Ventricular septal rupture
3	53	M	Hypertension Hyperlipidemia Obstructive sleep apnea	Chest discomfort Shortness of breath Diaphoresis	LVEDP 36 mmHg RA 10 mmHg PA 41/21 (32) mmHg PCWP 16 mmHg CO/CI 2.4 L/min, 1.2 L/min/m ² Qp/Qs - 2.6	1 day	5 days	Ventricular septal rupture
4	72	F	Hypertension	Shortness of breath	LVEDP 30 mmHg RA 10 mmHg PA 32/12 (19) mmHg PCWP 11 mmHg CO/CI 2.5 L/min, 1.3 L/min/m ² Qp/Qs: 2.8	14 days	7 days	Ventricular septal rupture

LVEDP – left ventricular end diastolic pressure. RA - right atrium. PA – pulmonary artery. CO – cardiac output. CI – cardiac index. Qp – pulmonary blood flow. Qs – systemic blood flow. Qp/Qs – shunt fraction. F – female. M – male.

diaphoresis. Vital signs were stable. Physical examination was notable for a holosystolic murmur with radiation to the apex.

3.2. Investigations

EKG showed sinus tachycardia, with ST segment elevations in the inferior leads and reciprocal depressions in the lateral leads (Fig. 3.1). Laboratory studies were significant for troponin I of 6.56 ng/mL and negative COVID-19 PCR.

3.3. Management

Patient was given aspirin, ticagrelor and heparin and transferred emergently to the tertiary care center cardiac catheterization laboratory. Coronary angiography revealed a 100% proximal RCA occlusion with left to right collaterals (Fig. 3.2). Ventriculogram showed an LVEF of 35% and distal ventricular septal rupture. Table 1 shows hemodynamics. TTE showed EF of 65–70% with large apical muscular ventricular septal defect with left to right shunting (Video 3). Patient

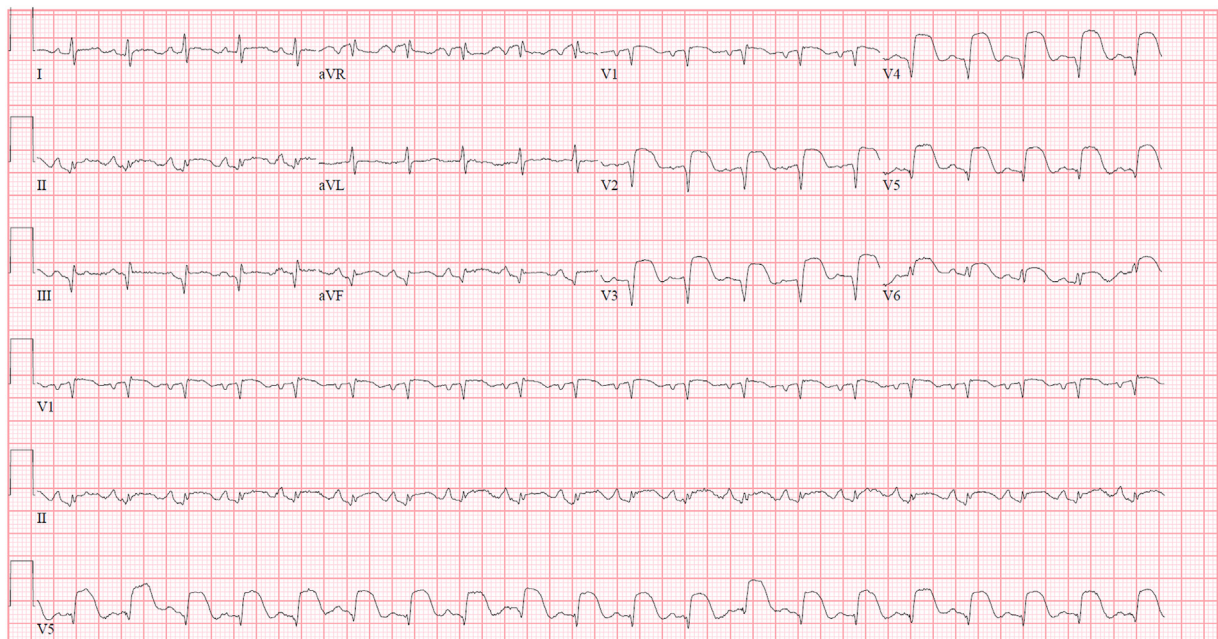


Fig. 2.1. Electrocardiogram (EKG) of Case #2 showing remote inferior infarct and acute anterolateral ST elevation infarction.

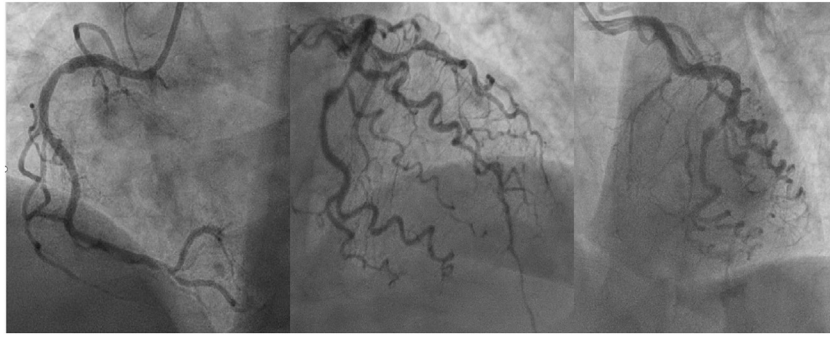


Fig. 2.2. Angiogram of Case #2 showing serial 70% and 99% stenoses of the mid left anterior descending artery.

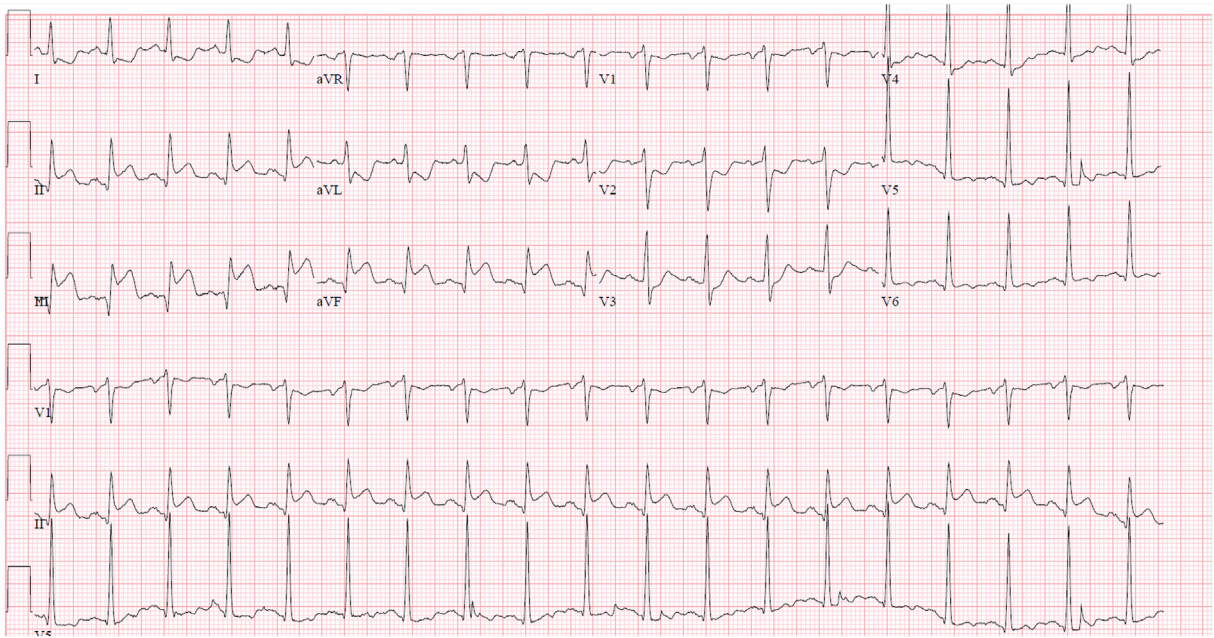


Fig. 3.1. Electrocardiogram (EKG) of Case #3 showing acute inferior ST elevation infarction.

was taken emergently for surgical repair of VSD with 2 Dacron patches. While taking the patient off cardiac bypass, patient developed significant bleeding from tears in the inferior wall of left ventricle.

3.4. Follow-up

Due to persistent bleeding, patient expired while in the operation room.



Fig. 3.2. Angiogram of Case #3 showing proximal RCA occlusion with left to right collaterals.

4. Case #4

4.1. Medical history

A 72-year-old female presented to a peripheral hospital with 2 days of shortness of breath. Patient reported symptoms of chest pain 1 week prior to arrival. Her initial vitals were unstable. She was transferred to tertiary care center for further care.

4.2. Investigations

COVID PCR was negative. Upon arrival, the patient had severe lactic acidosis (ABG = pH 7.05 mmHg, pO₂ 134 mmHg, pCO₂ 18, bicarbonate 5 meq/L, lactic acid >12 mg/dL), was on BiPAP but was still following commands. Shortly after arrival, an IABP was placed at bedside and she was emergently intubated in lieu of poor oxygenation. During this process, the patient had a pulseless electrical activity arrest requiring several rounds of cardiopulmonary resuscitation before achieving return of spontaneous circulation. TTE (Fig. 4.1) showed a serpiginous mid-apical infero-septal VSD; LVEF 40–45% with regional wall motion abnormalities consistent with recent anterior MI.

4.3. Management

The patient was brought to the catheterization lab urgently and was found to have a severely stenotic LAD artery with evidence of thrombus. The patient underwent successful stenting of the LAD artery with two

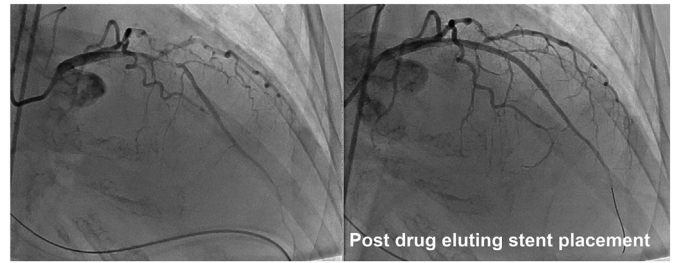


Fig. 4.2. Coronary angiogram of Case #4 showing pre and post stenting of left anterior descending artery stenosis.

drug eluting stents aided by aspiration thrombectomy (Fig. 4.2). This improved the flow from TIMI 0 to TIMI 2. At this stage, due to severe cardiogenic shock, the patient was supported with TandemHeart® percutaneous left ventricular assist device. Table 1 shows hemodynamics. After a week of care, patient underwent percutaneous closure of the VSD with 25 mm Amplatzer® Cribriform device. Transesophageal echocardiography showed no significant shunting thereafter (Fig. 4.3).

4.4. Follow-up

Unfortunately, the hospital course was complicated by sepsis and multi-system organ failure, the patient expired approximately 2 weeks after VSD closure device implantation.

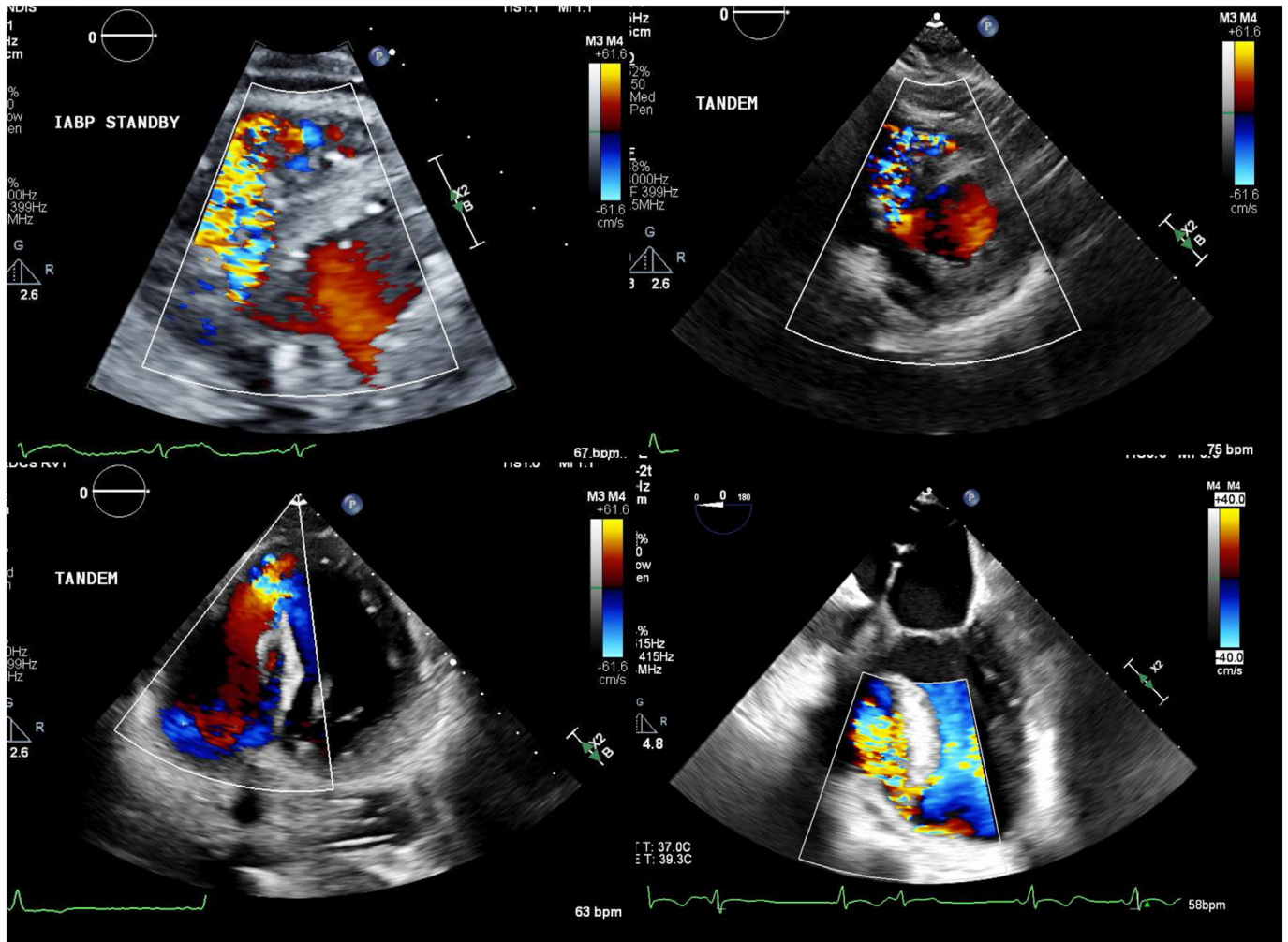


Fig. 4.1. Transthoracic echocardiogram of Case #4 showing serpiginous mid-apical infero-septal ventricular septal rupture.

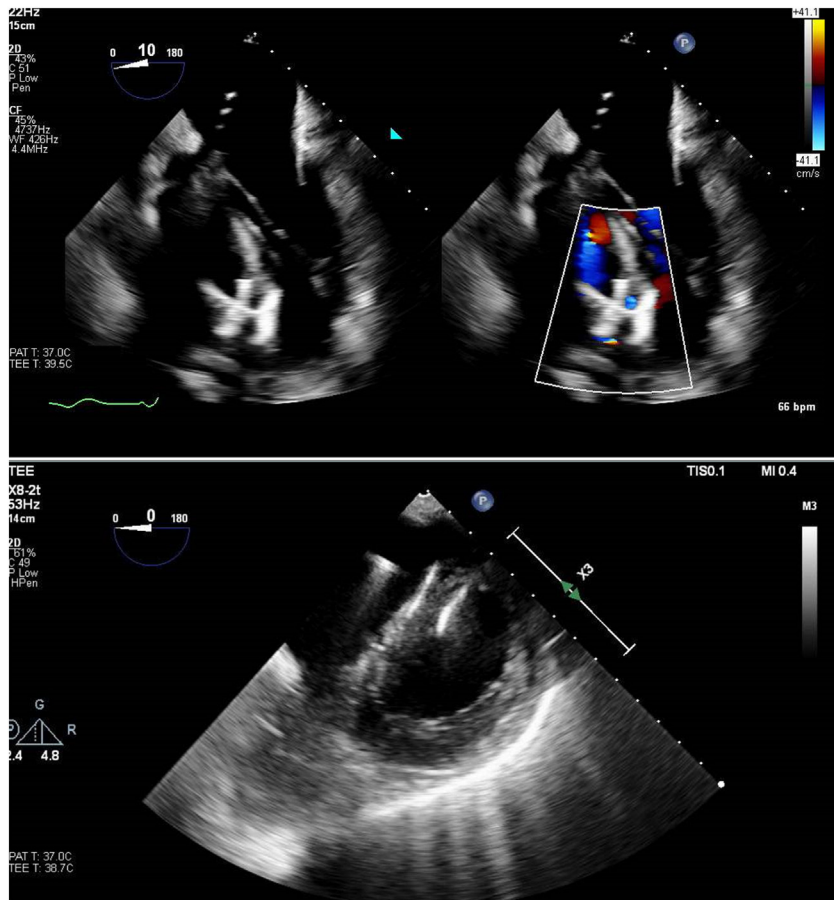


Fig. 4.3. Transesophageal echocardiography of Case #4 showed no significant shunting after ventricular septal rupture repair.

5. Discussion

The novel coronavirus 2 (SARS-CoV2), is a viral disease that leads to severe respiratory syndrome termed coronavirus disease 2019 (COVID-19) that has affected more than 10 million people and has killed more than half a million worldwide [1]. Several measures including social distancing and #stayhome campaigns are being run. However, due to COVID-19, nearly half of the public (48%) has delayed their health care as found by Kaiser Family Foundation poll [2]. In the pre percutaneous coronary intervention era, the incidence of free wall rupture was 6%, papillary muscle rupture was 2% and ventricular septal defect was 2% [3]. The incidences of all these complications decreased to 0.2% in the post PCI era and is rarely witnessed in areas where PCI is readily available [4,5].

An important aspect of this case series was that there was already a suspicion of VSD in two of these cases because of the physical examination finding of murmur prior to arrival to the cardiac catheterization lab. It is important to auscultate these patients prior to bringing to the catheterization laboratory as it may impact the follow up management. Though a small case series, this rapid increase in post MI complications cases in less 2 months at these institutions highlights the “unmeasured” toll and impact of COVID-19 on patients during this pandemic. This further highlights the crucial role that cardiologists could play in educating patients to recognize and seek medical help for their cardiovascular symptoms.

6. Conclusion

Post MI complications have high morbidity and mortality. Early identification may impact the outcomes and patients should be

encouraged to seek health care for their cardiovascular symptoms during COVID pandemic.

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Declaration of competing interest

The authors have nothing to disclose. This manuscript is an original effort that has not been submitted or is under consideration for publication elsewhere. All co-authors have contributed substantially to the manuscript preparation and are wholly responsible for the manuscript content without any associated conflicts of interest or financial incentives.

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