Traumatic Intramyocardial Dissection With Anterior Papillary Muscle Avulsion and Severe Mitral Regurgitation

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INTRODUCTION

Intramyocardial dissection (IMD) of the left ventricle (LV) is a rare cardiac emergency that may result from an acute myocardial infarction, percutaneous coronary intervention, and, less commonly, chest trauma.1 We present a unique case of an IMD with simultaneous avulsion of the papillary muscle without frank rupture, leading to severe acute mitral regurgitation (MR), in a 35-year-old pregnant woman who sustained blunt chest trauma after a motor vehicle collision (MVC).

CASE PRESENTATION

A 35-year-old pregnant woman (8/40 weeks) with a history of a seizure disorder was transferred to our tertiary care facility for a mitral valve replacement (MVR) due to severe MR following an MVC. The patient was a restrained driver, and airbags were deployed. On arrival to the referring facility, the patient was fully alert and oriented with only a complaint of neck pain. There was no external evidence of bruising or swelling to the chest on examination. An initial electrocardiogram revealed sinus tachycardia. A computed tomography (CT) scan of the chest from the referring facility noted a small circumferential pericardial effusion and bilateral pulmonary contusions. The patient subsequently developed premature ventricular contractions and nonsustained ventricular tachycardia for which amiodarone and lidocaine infusions were initiated. Upon arrival to our facility, a transesophageal echocardiogram (TEE) confirmed the avulsed anterolateral papillary muscle. This, along with the avulsed anterolateral papillary muscle, was resected (Figure 1A–C, Videos 1-3).

The patient was emergently taken for an MVR. Intraoperative two-dimensional TEE, midesophageal 4-chamber view at 33° demonstrates a large mobile echogenic mass within the mid LV cavity that prolapses into the left atrium in systole and suggests an avulsed anterolateral papillary muscle. The global LV systolic function is moderately reduced with a thin and hypokinetic apical segment.

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findings, diagnosis of LV IMD with likely avulsion of the anterolateral papillary muscle was made (Figure 1A–C, Videos 1-3).

The patient was emergently taken for an MVR. Intraoperative transesophageal echocardiography (TEE) confirmed the avulsed anterolateral papillary muscle that prolapsed into the left atrium in systole (Video 4) that was not visualized on the initial TTE. During the surgical procedure, the anterior leaflet was grasped and moved into the left atrium, confirming prolapse of a large mass of papillary muscle. This visible mass was still partially tethered within the LV. The anterior leaflet was explanted, exposing the LV cavity and enabling the identification of the few remaining attachments of the papillary muscle. Upon further evaluation of the endocardial surface of the apex of the LV, there was a large bare area where the dissected myocardium was visualized. This, along with the avulsed anterolateral papillary muscle and MV, was resected (Figure 2). A 29 mm Carbomedics
mechanical MVR was implanted. A postsurgical TTE showed a normal functioning mechanical MV with a low normal LV ejection fraction of 50% and a small remnant of dissected myocardial tissue in the LV apex. The patient did well postsurgical resection of the dissected myocardium and anterolateral papillary muscle along with MVR. The patient was discharged in good condition on postoperative day 8. The patient was on warfarin and then transitioned to enoxaparin during her pregnancy and successfully delivered a healthy baby at 38 weeks via cesarean section.

DISCUSSION

Blunt chest trauma resulting in an IMD with simultaneous papillary muscle avulsion and severe MR is rare, although it has been reported as a postmyocardial infarction late complication. The extensive myocardial dissection led to myocardial thinning and hypokinesis of the involved segments. Severe MR resulted from avulsed anterolateral papillary muscle leading to systolic bowing of the anterior MV leaflet and decrease in coaptation length (reduction in closing force of the valve).

An IMD is caused by dissection of the spiral myocardial fibers separated by thrombotic hemorrhagic channels thus creating a neocavity in the myocardium without full rupture. It also can also occur following septal channel perforation during percutaneous intervention of chronic total occlusion via retrograde approach, or in a case of revascularization of a chronically infarcted coronary vessel. Differential diagnoses of IMD include intramyocardial hematoma (IDH), intracardiac thrombosis, pseudoaneurysms, and prominent ventricular trabeculations. Echocardiographic findings of IDH include separation of LV muscle by hemorrhagic channels. The endocardium and epicardium remain intact, but the hematoma is entrapped within the myocardium. Diagnosis of IDH is based on the presence of 3 or more of the following signs: (1) the formation of at least 1 neocavitation within the tissue with an echolucent area, which expands during systole and shrinks during diastole; (2) thin and mobile endomyocardial border that surrounds the involved myocardial defect; (3) Doppler evidence of the continuity between dissecting hematoma and one of the ventricular cavities; (4) communication between the 2 ventricles; (5) partial or complete spontaneous retraction of the dissection flap; (6) presence of ventricular septal rupture, papillary muscle, or chordal rupture. This may be surrounded by an extensive area of infarction of the involved coronary artery. We consider our case to be an IMD without hematoma as there was no visualization of clotted blood within the neocavity seen between the 2 myocardial layers on imaging or on gross surgical specimen. With pseudoaneurysm, there is full rupture of the myocardial
wall contained within the pericardium, with an outpouching of the LV segment with a narrow neck and systolic expansion. Valvular injury in MVC is related to sudden deceleration and transfer of kinetic energy to the chest. Closed atrioventricular valves during systole are most susceptible to damage, causing rupture of the valve, chordae tendinea, and papillary muscle. In our case, IMD along with avulsed anterolateral papillary muscle led to systolic bowing of anterior MV leaflet and severe MR. Avulsed anterolateral papillary muscle was seen prolapsing into the left atrium on intraoperative TEE. However, no frank papillary rupture was seen as described in other cases of blunt chest trauma.

There are limited data describing the management of IMD. In one case series of IMDs, 10% (1/10) of those treated medically survived past 30 days, as opposed to 100% of those treated surgically, suggesting better prognosis with surgical management. Factors such as the anatomic features of the dissection, clinical presentation, and comorbidities should be considered during management decisions of myocardial dissection. In one case report, a patient was surgically treated 6 weeks following the initial injury to allow the torn myocardial tissue to fibrose before the operation as the patient was clinically stable. Our patient was asymptomatic; however, given the severe MR, they were considered high risk for progressive clinical deterioration. Surgical management was pursued due to severe MR in the setting of the avulsed papillary muscle. Optimal treatment in this case was MVR with resection of dissected myocardium and avulsed papillary muscle.

**CONCLUSION**

Isolated IMD is a rare cardiac emergency from blunt chest trauma that carries a high mortality rate. The association of IMD with papillary muscle avulsion and severe MR is exceedingly uncommon. A combination of a high index of suspicion and appropriate diagnostics including a TTE is essential to identifying this emergent condition.

**SUPPLEMENTARY DATA**

Supplementary data to this article can be found online at https://doi.org/10.1016/j.case.2022.06.009.

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