Right Ventricular Free Wall Rupture Complicating Anterior STEMI

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ABSTRACT

Ventricular free wall rupture is a rare but devastating complication. We report right ventricular free wall rupture complicating anterior ST-segment elevation myocardial infarction caused by a wrap-around left anterior descending coronary artery. In acute cardiac tamponade, a rapid and systematic evaluation of the likely source of bleeding is paramount to prevent disastrous outcomes. (Level of Difficulty: Advanced.) (J Am Coll Cardiol Case Rep 2022;4:770–774) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 56-year-old Caucasian woman was admitted to our tertiary center (Freeman Hospital, Newcastle upon Tyne, UK) for primary percutaneous coronary intervention (PCI) for an acute anterior myocardial infarction (MI). She had chest pain and a witnessed out-of-hospital cardiac arrest. She underwent immediate bystander cardiopulmonary resuscitation and successful cardioversion from ventricular fibrillation (VF) on paramedic arrival. She was then intubated and brought to our center. Her blood pressure was 90/60 mm Hg, her heart rate was 116 beats/min, her body temperature was 36.2 °C, and pulse oximetry oxygen saturation (SpO2) was 98%. She had normal heart sounds with no murmurs. The patient had a past medical history of multiple sclerosis.

INVESTIGATIONS

A 12-lead electrocardiogram (ECG) confirmed anterior ST-segment elevation myocardial infarction (STEMI) (Figures 1A and 1B). The patient was accepted for primary PCI, and emergency angiography was performed through the right radial artery approach using a 6-F sheath. The left main (LM) coronary artery and the right coronary artery (RCA) were engaged with an EBU 3.5 guide catheter (Medtronic) and a Judkin’s Right (JR) 4...
diagnostic catheter, respectively. The RCA was small, codominant, and unobstructed (Video 1). The left coronary system was dominant, with a severe distal LM bifurcation lesion and an occluded mid-left anterior descending (LAD) coronary artery (Video 2).

The patient was administered aspirin, prasugrel, and heparin, and PCI of the LAD artery was then performed. The occlusion was crossed, and an aspiration catheter was used to aspirate the thrombus, with immediate restoration of Thrombolysis In Myocardial Infarction (TIMI) flow grade 3 revealing a wrap-around LAD artery (Figure 2). The lesion was then stented with a 3.5 × 28 mm drug-eluting stent and optimized with a 3.5 × 20 mm noncompliant balloon, with an excellent result (Video 3). The LM artery stem was then treated with a T-stenting technique 4.0 × 15 mm into the LAD optimized with a 5 × 12 mm noncompliant balloon and 3.5 × 12 mm into the left circumflex artery, with an excellent final result (Video 4). Throughout PCI, the patient’s hemodynamics were stable, with a blood pressure of 145/75 mm Hg.

At the end of the procedure and before removing the patient from the catheter laboratory table, her systolic blood pressure dropped to 90 mm Hg. An immediate bedside transthoracic echocardiogram was performed and showed a significant circumferential pericardial effusion with signs of cardiac tamponade and a suggestion of right ventricular (RV) free wall leak on color Doppler imaging (Figure 3, Videos 5 and 6).

DIFFERENTIAL DIAGNOSIS

The differential diagnosis included coronary perforation, ventricular perforation, ventriculoseptal defect, ventricular free wall rupture, papillary muscle rupture, and acute aortic dissection.

(A and B) Electrocardiogram showing normal sinus rhythm and ST-segment elevation in leads II, III, aVF, V2, V3, V4, V5, and V6.
Emergency pericardiocentesis was performed, and an intra-aortic balloon pump was inserted, which temporarily improved the hemodynamics. Reaccumulation of the pericardial effusion occurred rapidly; therefore, autotransfusion of aspirated blood through a femoral venous line was commenced. The patient remained in refractory hypotension despite continuous drainage and autotransfusion.

A repeat coronary angiogram showed no evidence of coronary artery perforation, and there was no obvious left ventricular leak or wall rupture on left ventricular angiography. Bubble contrast transthoracic echocardiography with agitated saline was used to confirm the suspicion of RV free wall rupture, with bubbles seen to emerge into the effusion from the same location as the jet seen on color Doppler imaging (Figure 4, Video 7). This finding was corroborated with right-sided heart catheterization and a RV angiogram demonstrating a pulsatile jet of contrast from the right ventricle into the pericardium. Thereafter, the patient was stabilized by aggressive fluid resuscitation, blood and platelet transfusions, repeated pericardial drainage, and autotransfusion to maintain hemodynamics.

The cardiothoracic surgical team was contacted and accepted the patient for emergency repair of the RV free wall rupture. The patient was transferred to the operating room in a peri-arrest situation. Cardiac output was lost just before emergent sternotomy, and immediate return of spontaneous circulation was obtained on pericardial decompression.

A single defect was found in the acute margin of the right ventricle (Figure 5), with associated bruising extending for 7 cm, without obvious evidence of RV free wall hematoma. The bleeding point was closed with a horizontal mattress sutures using 4.0 polypropylene (Prolene, Ethicon) reinforced with Teflon (Chemours) pledges. The patient had a further episode of VF, which reverted back to sinus rhythm by internal DC cardioversion. She was weaned from bypass without incident and was transferred to the cardiac critical care unit.

On day 8, repeat transthoracic echocardiography revealed good left ventricular function, impaired RV function with moderate tricuspid regurgitation, and no evidence of pericardial collection. On day 20, she was successfully discharged to her local hospital for a period of rehabilitation.

**DISCUSSION**

To our knowledge, this is the first report of RV free wall rupture complicating anterior STEMI as a result...
of thrombotic occlusion of a wrap-around LAD artery, with early diagnosis and successful management. The occurrence of RV infarction secondary to left coronary artery occlusion was previously reported in a retrospective examination of 107 autopsies from patients with coronary heart disease. In this study, RV infarction was found to have almost similar frequency in both anterior and posterior infarctions (64% vs 66%, respectively).

The right ventricle is supplied by the RV marginal branches of the RCA. Thus, most RV infarcts result from occlusion of the RCA proximal to the origin of the major RV branches. In general, the occurrence of RV infarction is usually expected in association with inferior infarction of the left ventricle because the RCA usually supplies not only the inferior (and sometimes inferolateral) wall of the left ventricle but also the RV free wall. However, the RV wall rarely undergoes transmural MI; this is the reason that RV free wall rupture is much rarer than left ventricular rupture, with only few previous reports of isolated RV free wall rupture.

RV myocardial infarcts secondary to anterior coronary artery territory involvement are associated with a significant and higher in-hospital morbidity and mortality compared with those infarcts secondary to RCA territory involvement. Adverse outcomes are usually related to significant hemodynamic compromise and electrical complications, which occur in approximately 50% of affected patients. However, the long-term prognosis is generally good for those patients who survive.

In our patient, the coronary anatomy showed a small, codominant, nonobstructed RCA with a dominant left coronary system and an occluded wrap-around anterior descending artery (LAD). This apical LAD artery extension possibly serves as the PDA and perfuses the inferior wall, hence supplying the RV wall. This may explain the RV infarction and the subsequent RV wall rupture secondary to anterior STEMI, although the occurrence of this complication is rare.

Pericardial effusion with cardiac tamponade is a well-recognized complication in the setting of
primary PCI for STEMI. This can be caused by bleeding from the coronary arteries or from a cavity. Ventricular perforation may result from free wall rupture from the infarction itself, or very rarely, it may be iatrogenic. Although the pericardial effusion is rapidly identifiable with 2-dimensional trans-thoracic echocardiography, identification of the source of bleeding requires a systematic evaluation of the likely culprits, by performing coronary angiography to exclude coronary perforation, followed by detailed echocardiography, correlated with left and right ventriculography.

This case also highlights the usefulness of bubble contrast echocardiography to confirm and localize an RV free wall rupture. This technique is readily applicable to emergency situations and has been previously described. In the absence of shunts, intravenous bubble contrast is specific to the right side of the heart, with bubbles breaking down in the pulmonary capillary bed. Observation of intrapericardial bubbles immediately post-injection is highly suggestive of RV perforation or rupture, and we were able to localize the defect of the free wall. Furthermore, this technique is safe, and it takes little time to perform (9 mL saline and 1 mL air agitated rapidly through a 3-way tap).

**FOLLOW-UP**

The patient made an uneventful recovery and was discharged home approximately 1 month from the index event. A repeat echocardiogram at 3-month follow-up showed preserved left ventricular systolic function and only mildly impaired RV systolic function. No intracardiac shunts or obvious pericardial effusion signs were found.

**CONCLUSIONS**

Ventricular free wall rupture is a rare complication of anterior STEMI as a result of LAD occlusion. However, it has to be considered and ruled out in a timely and systematic approach to identify a likely bleeding culprit in acute cardiac tamponade, to prevent disastrous outcomes.

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**KEY WORDS** acute coronary syndrome, bubble echocardiography, tamponade, ventricular fibrillation

**APPENDIX** For supplemental videos, please see the online version of this article.