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Case Reports

Managing Thrombosis and Hemorrhage in a Man with Myocardial Infarction and Traumatic Hemopericardium with Cardiac Tamponade

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A 79-year-old man had an out-of-hospital acute ST-segment-elevation myocardial infarction with cardiac arrest. Cardiopulmonary resuscitation performed by a bystander resulted in traumatic hemopericardium. We discuss the patient's case, highlight the challenges of managing simultaneously life-threatening thrombosis and hemorrhage, and present our conclusions regarding the patient's eventual death. ***(Tex Heart Inst J 2021;48(3):e207308)***

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Caring for patients who have multiple pathologic conditions can be challenging when the optimal treatment for one disease process is contraindicated by the presence of another disease process. Especially during emergencies, physicians must make difficult and complex decisions to treat one condition and risk exacerbating the other—for example, when a patient has active thrombosis or a predisposition to clotting in addition to active hemorrhaging or a tendency to bleed. We report the case of a patient who presented with simultaneous ST-segment-elevation myocardial infarction (STEMI) and traumatic hemopericardium with cardiac tamponade, a combination that has not been well described in the medical literature.

Case Report

In September 2019, a previously healthy 79-year-old man was revived from cardiac arrest after a bystander performed cardiopulmonary resuscitation (CPR). He was transported by ambulance to our emergency department. On examination, the patient was confused and unable to report symptoms. His initial blood pressure was 94/65 mmHg; heart rate, 105 beats/min; respiratory rate, 28 breaths/min; and oxygen saturation, 99% on a non-rebreather face mask. He was alert but disoriented and agitated. His pupils were equal, round, and reactive. He could move all extremities (which were cool to the touch) and had no focal neurologic deficits. He was using accessory muscles of respiration but had clear lung sounds. His cardiac sounds were distant, and his jugular veins were distended.

The patient's admission electrocardiogram showed evidence of STEMI (Fig. 1). However, given his unusual clinical presentation and ongoing instability with hypotension and hypoxia, we decided to stabilize him in the emergency department rather than in the cardiac catheterization laboratory. He was intubated and placed on a mechanical ventilator, and a central line for intravenous fluids and vasopressors was inserted. A focused bedside echocardiogram showed hypokinesis of the anterolateral and inferolateral left ventricle (LV) and a moderate pericardial effusion with associated thrombus (Fig. 2). The working diagnoses were STEMI and hemopericardium with cardiac tamponade.

Because several causes of hemopericardium would necessitate surgical repair,[1] we consulted our heart team. On approval from the cardiac surgeon, we moved the patient to the catheterization laboratory. Diagnostic angiograms showed thrombotic occlusion of the mid left circumflex coronary artery (LCx) (Fig. 3A), critical stenosis in

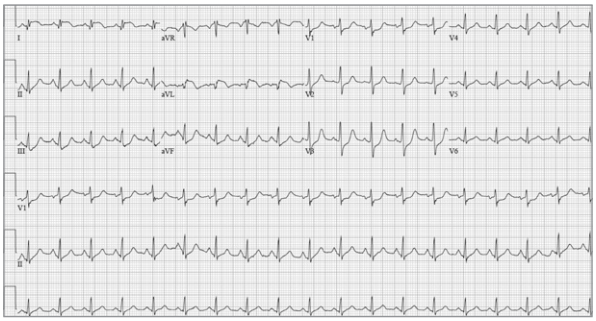

Fig. 1 *Electrocardiogram shows sinus tachycardia, ST-segment elevation in leads I and aVL, and ST-segment depression in leads III, aVF, and V_1 through V_4, suggesting acute myocardial infarction of the lateral, and possibly posterior, left ventricle.*

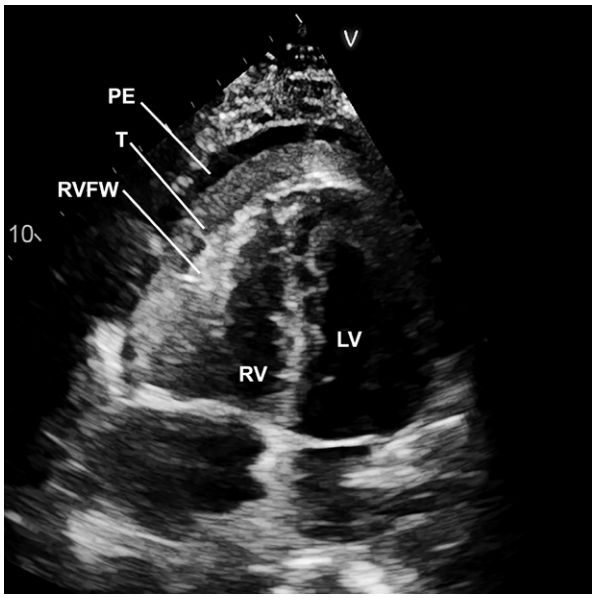

Fig. 2 *Baseline transthoracic echocardiogram (apical 4-chamber view) shows mobile thrombus (T) within the pericardial effusion (PE), and anterolateral hypokinesis.*

LV = left ventricle; RV = right ventricle; RVFW = right ventricular free wall

Supplemental motion image is available for Figure 2.

the mid left anterior descending coronary artery (LAD) (Fig. 3B), and moderate stenosis in the right coronary artery (Fig. 3C).

Pericardiocentesis was performed through the subxiphoid approach, 670 mL of frank blood was evacuated, and a pigtail drainage catheter was left in place. The patient's vital signs immediately improved; however, within 5 minutes, recurrent and refractory hypotension developed. Of note, flow from the pericardial drain was minimal despite flushing of the catheter to ensure its patency; this suggested that shock had been caused by STEMI. Suspecting LV failure, we placed an Impella CP heart pump (Abiomed, Inc.), and the patient's blood pressure returned to normal. Because the pericardial bleeding appeared to have stopped, we decided to

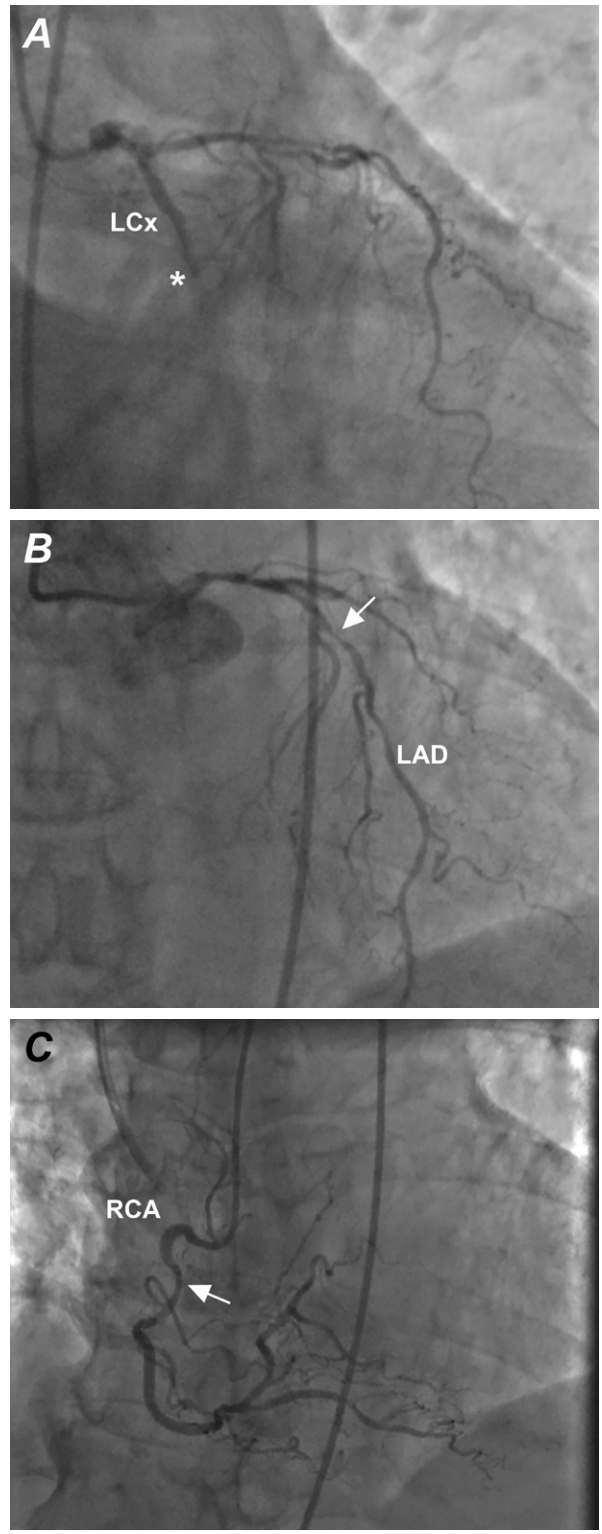

Fig. 3 *Baseline angiograms of the left coronary artery in the **A**) caudal and **B**) cranial views show occlusion (asterisk) of the left circumflex coronary artery (LCx) and stenosis (arrow) of the left anterior descending coronary artery (LAD). **C**) Baseline angiogram (cranial view) shows moderate stenosis (arrow) in the right coronary artery (RCA).*

Supplemental motion images are available for Figure 3A, 3B, and 3C.

perform percutaneous coronary intervention (PCI). Up to this point, the patient had been given oral aspirin but no other antithrombotic therapy. We administered heparin (target activated clotting time, 285 sec), and placed stents in the LCx and LAD without complication (Fig. 4). Clopidogrel was administered through an orogastric tube, and the patient was transferred to intensive care.

Approximately 3 hours later, the patient suddenly became hypotensive and began hemorrhaging from the pericardial drain. During initial resuscitation, we administered fluids, multiple vasopressors, and protamine, and followed a massive transfusion protocol. The surgeon initiated emergency extracorporeal membrane oxygenation (ECMO) and created a pericardial window, after which the patient's condition stabilized. Of note were the decreases in his serial hemoglobin concentrations: hospital arrival, 14.1 g/dL; after PCI, 11.6 g/dL; and during this acute decompensation, 5 g/dL.

During the next week, we stopped dual antiplatelet therapy (DAPT) and began intermittent transfusion of blood products and maintenance of organ perfusion with use of pharmacologic support. Impella and ECMO support were continued. The patient's brain, kidney, and liver functions had remained intact for 11 days. Echocardiograms showed a large pericardial hematoma and severely depressed biventricular function (Fig. 5).

On hospital day 8, the surgeon performed a sternotomy and removed extensive pericardial thrombus, substantially improving the patient's cardiac contractility. In addition, actively oozing abrasions of the anterior aspect and a perforation of the inferior aspect of the right ventricle (RV) were repaired with use of absorbable hemostatic pads and an adhesive sealant.

The patient's overall condition improved during the next few days. The Impella device provided a flow rate of 2.8 L/min at performance level P6, and we began to slowly wean the patient from ECMO support. Invasive hemodynamic data on hospital day 13 included a systemic arterial pressure of 92/74 mmHg, a right atrial pressure of 8 mmHg, a pulmonary artery pressure of 24/12 mmHg, and a mixed venous oxygen saturation of 86%. However, later that day, the patient's condition rapidly deteriorated; he had multiple episodes of polymorphic ventricular tachycardia, followed by refractory hypotension, profound lactic acidosis, and multiorgan failure. The family decided to institute comfort measures and discontinue support. The patient died instantly, and the family declined a request to perform an autopsy.

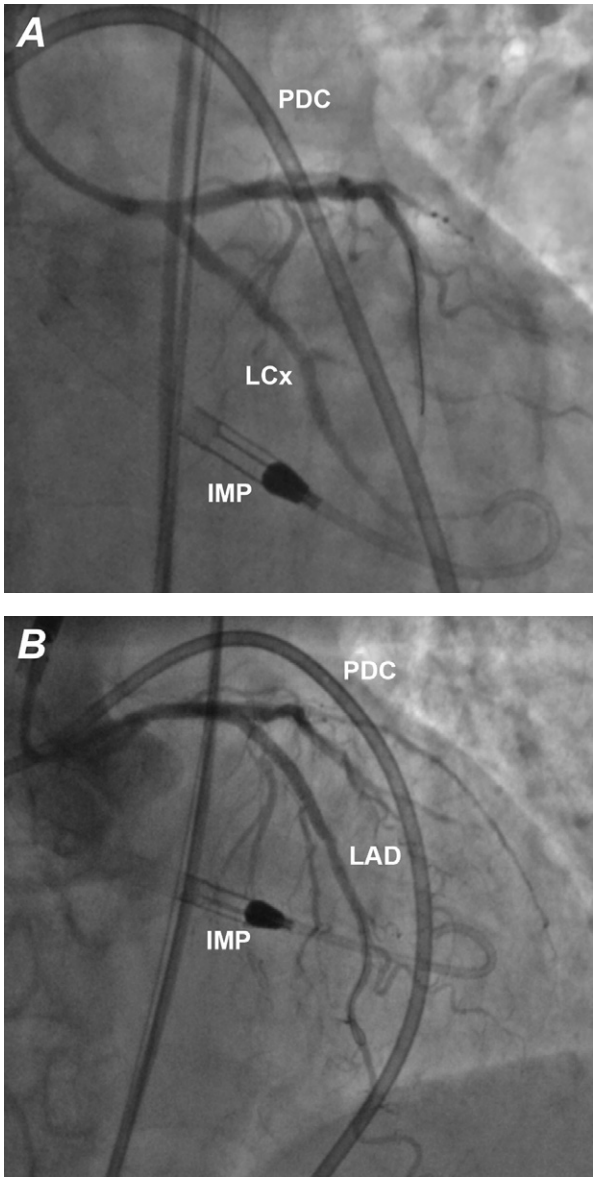

Fig. 4 *Completion angiograms show stents in the **A**) left circumflex coronary artery (LCx) and **B**) left anterior descending coronary artery (LAD).*

IMP = Impella CP pump; PDC = pigtail drainage catheter

Supplemental motion images are available for Figure 4A and 4B.

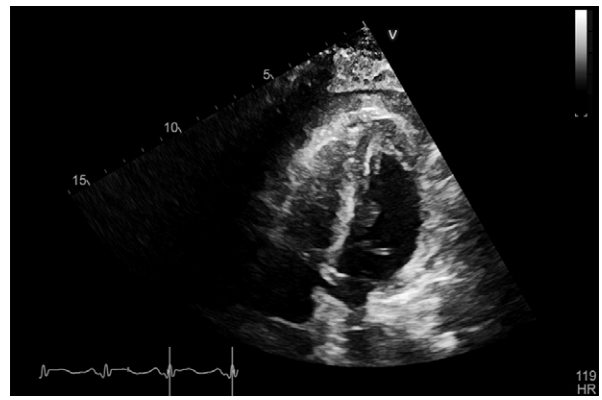

Fig. 5 *Transthoracic echocardiogram (apical 4-chamber view) shows a small residual pericardial effusion with organized hematoma and biventricular dysfunction.*

Supplemental motion image is available for Figure 5.

Discussion

Even without autopsy findings, we can explain what happened in this case. The acute presentation began with plaque rupture in the LCx, resulting in STEMI and cardiac arrest, probably due to a ventricular tachyarrhythmia. Chest compressions perforated the RV, causing blood to accumulate rapidly in the pericardium. Perforation from CPR can be distinguished from spontaneous myocardial rupture in that the latter typically affects the LV.[2,3] After pericardiocentesis, the bleeding seemed to be controlled, but heparin and clopidogrel administration disrupted the temporary hemostasis and caused severe repeat bleeding. Surgical evacuation of the pericardium enabled transient clinical improvement. However, the absence of DAPT for 13 days probably led to LAD and LCx stent thrombosis, cardiogenic shock, and death.

In most reports of STEMI and hemopericardium, bleeding is attributed to thrombolytic therapy[4] or spontaneous myocardial rupture,[5] and it typically occurs hours to days after the initial presentation and use of antithrombotic therapy. This case was especially challenging because we had to manage the thrombotic and hemorrhagic processes concurrently from the start.

Of note, bedside echocardiography is not routinely used in straightforward cases of STEMI; however, we decided to use it because of the patient's ongoing hypotension, hypoxia, and altered mental status. It enabled us to identify blood within his pericardium and to alter the entire course of care.[6] In addition, we recognized the potential need for surgery[1] and collaborated with a cardiac surgeon from the start.

In retrospect, initial surgery may have afforded a better outcome. First, it probably would have enabled early, definitive treatment of the RV injuries, as well as an opportunity to perform coronary artery bypass grafting (CABG) and to mitigate the need for DAPT. Second, whereas elevated pericardial pressure with cardiac tamponade from hemopericardium counteracts ongoing blood loss, pericardiocentesis eliminates this protective effect and can result in further bleeding, which is why surgery is usually preferred in this situation.[7]

Another consideration in our case is the role of PCI. Although one year of DAPT is recommended for all patients after acute coronary syndrome, independent of the treatment strategy,[8] the risk of abrupt vessel closure within the first 14 days (thus warranting DAPT) is greatest after stenting, more so than after plain old balloon angioplasty,[9] CABG, or medical therapy. Therefore, had we selected one of these management options, we could have avoided DAPT, decreasing the risks of the bleeding 3 hours after stenting and the fatal thrombosis on hospital day 13. Furthermore, had PCI been performed only on the LCx (the infarct-related vessel) and not the LAD, the thrombotic event would have affected only one vascular territory, and the patient might have survived. A large meta-analysis[10] revealed higher mortality rates in patients with STEMI who had multivessel PCI versus culprit-vessel-only PCI; however, most of the clinical trials in the analysis excluded patients who were in shock, so the conclusions cannot be applied to our patient's case.

Managing concomitant bleeding and clotting is challenging. In the case of STEMI and hemopericardium with cardiac tamponade, performing surgery to identify and to correct the source of bleeding, in conjunction with CABG if indicated, is superior to combined pericardiocentesis and PCI.

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