

Acute Hemorrhagic Cardiac Tamponade as Presenting Findings of Transmural Myocardial Infarction in an Adult Patient

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To the Editor: A 70-year-old female was admitted for severe precordial pain. Electrocardiograph on admission showed ST-segment elevation myocardial infarction (STEMI) with inferior wall, examination showed cardiogenic shock, noninvasive blood pressure (NIBP) 50/30 mmHg (1 mmHg = 0.133 kPa), and cardiac troponin I positive. She was diagnosed as acute myocardial infarction (AMI) with cardiogenic shock. Fluid resuscitation and inotropic agents were administered immediately, and systolic blood pressure increased to about 90 mmHg. The patient was transferred to catheterization laboratory immediately after administration of a loading dose of aspirin and clopidogrel. Angiography revealed occlusion at the left distal circumflex artery and culprit vessel [Figure 1a]. Percutaneous coronary intervention (PCI) was performed successfully [Figure 1b], and the intro-aortic balloon was implanted considering the intraoperative cardiogenic shock.

Her hemodynamic state deteriorated again (peripheral oxygen saturation 83%, NIBP 60/42 mmHg) when returned to the ward. Considering that the culprit vessel was too small to have a significant influence, echocardiography was performed immediately, which revealed a moderate pericardial effusion with the diastolic collapse of cardiac chambers [Figure 1c]. Acute pericardial tamponade was diagnosed. Echo-guided pericardiocentesis was performed immediately, and 60 ml nonclotting blood was extracted. Cardiac output increased from 1.7 to 7 L/min rapidly. Ultrasound showed no pericardial effusion, and all chambers were completely filled [Figure 1d]. NIBP increased to 126/63 mmHg. Systemic heparinization was maintained and activated clotting time fluctuated between 180 and 200 s. Repeated ultrasound showed no more pericardial effusion. Drainage catheter and intra-aortic balloon pump (IABP) were withdrawn on the 3rd and 5th day, respectively. The patient was discharged on day 13 with a diagnosis of acute hemorrhagic pericardial tamponade (AHPT) secondary to STEMI. Dual antiplatelet treatment was continued after discharge, and echocardiography at 3 months showed no pericardial effusion.

AHPT after infarction rarely occurs, which was reported only in 1% of STEMI treated with thrombolysis,^[1] often within the first 24 h.^[2] To our knowledge, very few reports have been published on AHPT after the onset of STEMI without thrombolysis, especially as the present findings. The predictors of cardiac tamponade in STEMI patients

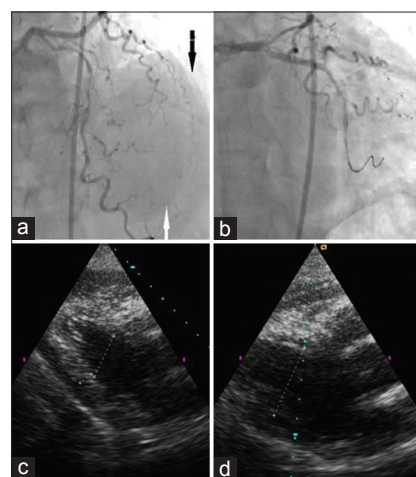


Figure 1: The evolutions of coronary angiography and echocardiography results. (a) coronary angiography revealed triple-vessel lesions, occlusion at the left distal circumflex artery and signs of “radiolucency” suspected (shown as black and white arrows); (b) angioplasty of culprit vessel; (c) before pericardiocentesis: Left ventricular end-diastolic diameter = 26 mm, an echo-free space at apex in diastole of 19.9 mm; (d) after pericardiocentesis: Left ventricular end-diastolic diameter = 42 mm, an echo-free space at apex disappeared (long-axis view).

include lateral site, increasing age, increasing number of leads involved, lack of reperfusion therapy, and late hospital admission.^[3] The mechanism of AHPT may be related to diffuse hemorrhagic pericarditis after infarction,^[2] as the patient had high white blood cell count ($21.09 \times 10^9/L$) and neutrophil percentage (90.5%) on admission, which, however, became normal within 72 h. AHPT may also be related to the rupture of microvessels at the infarction site,

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but no thoracic exploration was performed to confirm this suspicion in our case. Dressler syndrome is not considered, as it usually occurs between 1 week and several months after the onset.

It remains unknown whether systemic anticoagulation with heparin during IABP support aggravates AHPT. In our case, the dynamic changes of effusion and vital signs were repeated to monitor on day 1 and no increase in effusion was observed even under standard anticoagulation intensity.

In conclusion, AHPT is a rare but catastrophic complication following AMI. Prompt echocardiography and pericardiocentesis may save lives.

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Conflicts of interest

There are no conflicts of interest.

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