Moderate Pericardial Effusion Early After Myocardial Infarction

Left Ventricular Free Wall Rupture Until Proven Otherwise

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Pericardial effusion (PE) associated with myocardial infarction (MI) is considered to be associated with anterior ST-segment elevation MI (STEMI), with large infarcts, and when heart failure is present. When PE is associated with cardiac tamponade, it is usually due to cardiac rupture, hemorrhagic pericarditis, or aortic dissection involving the right coronary artery. However, the majority of PE does not cause hemodynamic compromise, and a small to moderate PE may often be viewed as a benign reaction to the MI. In 1986, Galve et al demonstrated in a study of 138 consecutive patients with acute MI that 28% of patients had a PE. The study was meticulously performed with repeated echocardiographic evaluations, and there were no cases of cardiac tamponade. However, these data may no longer be valid because the treatment of acute MI has changed dramatically since 1986. In a large study in the modern era of reperfusion from 25 French hospitals including 908 patients with MI, a PE was detected in only 6.6% of the patients. Patients had an echocardiogram performed at admission and again at discharge, and 0.8% developed left ventricular free wall rupture (FWR). This incidence of FWR is similar to that of a larger registry that did not search meticulously for rupture.3

Acute FWR is one of the most common causes of mortality in acute MI. The diagnosis is most often made by the presence of sudden electromechanical dissociation followed by death if left untreated. Some patients survive for several hours, allowing time for diagnosis and intervention. Textbooks of pathology often describe rupture as a late event, but large systematic series have shown that rupture is most common on day 1.4

In this issue of Circulation, Figueras and colleagues report the hitherto largest series of patients with PE after acute STEMI. During a period of 18 years, data on presence of PE were collected in 4446 consecutive patients in a single center. An early echocardiogram was obtained routinely in all patients (after 1992). Patients are presented according to hospital, year of MI, and primary PCI. The study provides no direct insight into the optimal timing of echocardiography but strongly advocates early screening to identify high-risk patients in whom much closer surveillance is warranted. Whether this should be done as an early focused clinical presentation with cardiac tamponade (with or without electromechanical dissociation) or according to size of PE. PE was graded into small or moderate on the basis of a parasternal short-axis view by simply summing anterior and posterior effusion measured in end-diastole. This revealed a small PE (5 to 9 mm) in 218 patients and a moderate (≥10 mm) effusion in 228 patients. The authors clearly demonstrate that a moderate PE after STEMI bears a grave prognosis, with a day-30 mortality of almost 43%.

Even in the absence of electromechanical dissociation or cardiac tamponade, mortality was twice that of patients without moderate PE. The cause of death in patients with electromechanical dissociation or tamponade was almost exclusively left ventricular FWR occurring within 5 days of STEMI. Few baseline characteristics predicted PE, although there was an expected overrepresentation of anterior infarcts. In addition, the data demonstrated a clear association between poor ST-segment resolution and presence of PE and especially with tamponade or electromechanical dissociation.

Thus, incomplete recanalization of infarct-related coronary artery appears to be a pathophysiological link to hemorrhagic PE and threatening rupture. The study was largely historical, with a very low utility (<20%) of percutaneous coronary intervention (PCI). This does not reflect the management of STEMI in many centers in 2010, where PCI is the preferred method to achieve reperfusion. One advantage of primary PCI is a faster and more complete recanalization of infarct-related artery with more rapid and complete ST-segment resolution.6 This could possibly influence the prevalence of PE and could potentially lower the risk of FWR. In addition, a high frequency of coronary intervention may also introduce a bias. PCI may cause dissection, rupture, or perforation of epicardial vessels that may cause PE. In addition, the use of temporary pacemakers is possibly higher in an interventional population, which also may be associated with right ventricular free wall perforation that may be unnoticed during the procedure. Thus, whether the data from the study of Figueras et al can be translated directly to a group of patients in whom primary PCI is the preferred method to achieve reperfusion is debatable.

Interestingly, the echocardiograms were obtained early after admission, and the detection of PE is based on a simple assessment of PE size in the parasternal short-axis view, with an arbitrary definition of moderate PE being ≥10 mm. The study provides no direct insight into the optimal timing of echocardiography but strongly advocates early screening to identify high-risk patients in whom much closer surveillance is warranted. Whether this should be done as an early focused...
study to assess left ventricular function and PE with a later more comprehensive echocardiogram or as an early comprehensive study should depend on local logistics. The same authors have presented data from a subset of these patients demonstrating that a minor PE within the first days after MI may be a harbinger of later manifest FWR. No data on additional imaging were presented in the present study by Figueras et al. One might speculate that in the subset of patients with moderate PE (5% of the population), magnetic resonance imaging could possibly provide further insight into area at risk and transmurality of infarcted myocardium.

Although detection of a moderate PE early after MI should raise the concern of myocardial rupture, other differential diagnoses should be considered in the acute setting, including aortic dissection and perimyocarditis. Angiography and absence of typical rise and fall in cardiac markers often suggest the diagnosis of perimyocarditis. However, benign early pericarditis after MI may be difficult to differentiate. A recent study demonstrated that 31 (4%) of 743 patients undergoing primary PCI for acute STEMI developed pericarditis, most with PE within 4 days of MI. No cases of myocardial FWR was seen, only 1 case of late pericarditis (Dressler syndrome) was seen, and development of early pericarditis did not affect long-term outcome.

The study by Figueras et al elegantly demonstrates how measurement of hematocrit in pericardial fluid provides important additional insight into the cause of PE. A diagnostic pericardial puncture with aspiration of 5 to 10 mL of pericardial fluid can usually be done safely with ultrasound guidance. If the hematocrit level is high, rupture should be considered the cause. If measurement of hematocrit in the pericardial fluid is performed days later, a decline in hematocrit seems ascribable to the formation of a blood clot and the dilution of the supernatant fluid.

The study by Figueras et al suggests that clinicians should look for PE early, and if moderate PE is present, they should suspect that FWR may be present or evolving. If the PE is hemodynamically compromising, the suspicion of FWR is augmented, especially if electromechanical dissociation occurs. However, the study does not tell us how to react when a moderate PE is present, but, when one considers the severe prognosis of early to moderate PE, close surveillance and an aggressive therapeutic approach seem warranted. Optimally, the patients should be observed in a cardiac intensive care unit with placement of an arterial line, with close echocardiographic surveillance, in an institution with cardiothoracic surgical backup. The present data also advocate pericardial puncture and drainage to better determine the cause of PE and guide therapy. Thus, pericardial drainage should be considered in patients with moderate PE. In the study, 19 patients were operated on for FWR, but these patients did not appear to do better than patients who did not undergo surgery. However, this may be due to a severe bias because acutely ill patients may be sent to surgery more often. It is disturbing that a moderately high number of patients with a moderate PE died suddenly of electromechanical dissociation, and future studies may guide us in methods to refine the diagnosis of evolving FWR and to determine when to treat surgically.

Conclusion

Lessons on ways in which to detect PE and to react to PE will need to be derived from patients’ series. With the uncertainty of such an approach, the present study of Figueras et al, combined with other available studies, indicates that (1) early echocardiography to detect PE can detect a population with a high risk of death and who deserve close monitoring to detect FWR and, it is hoped, to allow time to react before sudden death; (2) when the PE in a parasternal short-axis view exceeds 10 mm, the risk of a FWR is particularly high; and (3) measurement of hematocrit in PE is valuable for detection of FWR.

Disclosures
None.

References

Key Words: Editorials ■ acute myocardial infarction ■ pericardial effusion ■ prognosis ■ rupture
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