Case Report

Incidental Massive Pericardial Effusion Diagnosed by Myocardial Perfusion Imaging

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ABSTRACT
A report on an unusual case of pericardial effusion and tamponade that was found incidentally on myocardial perfusion imaging. This was later confirmed by echocardiography and subsequently treated with pericardiocentesis. Two-dimensional echocardiography is still the “gold standard” for diagnosing pericardial effusion. Nuclear cardiac imaging will probably never have a primary role in the diagnosis of pericardial effusion. However, it may be helpful when the diagnosis of pericardial effusion has not been considered and when this condition is suggested by nuclear imaging findings. The echocardiogram underestimated the amount of pericardial effusion compared to myocardial perfusion imaging in this case, and in contrast to previous published reports. Further, prospective studies need to focus on the sensitivity and specificity of sestamibi nuclear scans in the qualitative and quantitative assessment of pericardial effusions.

INTRODUCTION
Myocardial perfusion interpretation may be difficult because of tracer uptake variations. Likewise, incidental-finding criteria varies from report to report. A number of well-known incidental findings, such as lymphoma, parathyroid adenoma, lung, breast and thyroid carcinoma, have been reported during myocardial perfusion imaging, but not massive pericardial effusion.

We report an incidental massive pericardial effusion causing tamponade found during routine myocardial perfusion imaging. An echocardiogram performed the following day demonstrated a moderate pericardial effusion. Finally, pericardiocentesis was performed and yielded 800 cc of sanguineous fluid.
CASE REPORT

A 73-year-old woman presented to the emergency department with shortness of breath and chest pressure. She had intermittent substernal chest pressure, and back and shoulder discomfort for 4 days that became more constant and worsened on the day of admission. She stated that she could not sleep well because of shortness of breath. There was no pain radiation to the neck, jaw, or arms, and there was no abdominal pain, nausea or vomiting, fever, chills, cough, diaphoresis, or palpitations. Treatment with acetaminophen improved her shoulder pain, but not her chest discomfort.

The patient had a past medical history of chronic atrial fibrillation, transient ischemic attacks, dyspnea, and pulmonary hypertension with the most recent pulmonary artery pressure estimated at 103 mm Hg, two months earlier. There was nothing in the patient’s history regarding the etiology of pulmonary hypertension. Her medications included coumadin, digoxin, multiple vitamins, vitamin E, lorazepam, clonazepam and fosinopril sodium tablets.

On admission her blood pressure was 161/72 mm Hg, pulse 65/min, respiratory rate 18/min, temperature 97°C, and O2 saturation 97% on room air. On physical examination she appeared somewhat anxious, but otherwise healthy and in no acute distress. The neck veins could not be evaluated due to neck obesity. She had course breath sounds bilaterally with no crepitations. The heart sounds were distant. There was a soft systolic ejection murmur. The abdomen was benign without hepatosplenomegaly. There was mild lower extremity bilateral pitting edema. There was no pulsus paradoxus identified by either the internist or cardiologist. A 12-lead electrocardiogram revealed no significant change in her atrial fibrillation with nonspecific ST-T wave changes without evidence of low voltage or electrical alternans.

The chest radiograph showed cardiomegaly with bilateral hilar infiltrates and prominent interstitial markings (figure 1A). On the same day, the radiologist had access to the previous chest radiograph (figure 1B). He thought there was now considerably more “water-bottle” configuration of the heart, suggesting either increasing cardiac dilatation or pericardial effusion.

The patient was admitted to rule out myocardial infarction and was treated with oxygen, furosemide and coumadin. An electrocardiogram was repeated the next morning. A myocardial perfusion study was ordered the morning after admission. During the resting portion of the rest-stress protocol a large pericardial effusion was seen on the raw images (figure 1C). The ordering physician was immediately made aware of this finding. The myocardial perfusion scan was normal and the left ventricular ejection fraction was calculated at 73% (figure 1D). Later that day the patient had a transthoracic echocardiogram that showed a moderate to large pericardial effusion with findings consistent with impending cardiac tamponade along with moderate to severe pulmonary hyper-tension (figure 2). Transmitral Doppler flow, done in conjunction with a respirometer, showed variation in E wave velocity in excess of 25%. This was considered evidence for “impending” cardiac tamponade. A subsequent pericardiocentesis yielded 800 cc of sanguineous and turbid fluid. There were no tumor cells by cytology. A follow-up echocardiogram showed no residual pericardial effusion. Two days later a repeat chest radiograph showed improvement in the size of the myocardial silhouette. The remaining hospital course was uneventful.

DISCUSSION

An unusual case of an unsuspected large pericardial effusion and tamponade found on a myocardial perfusion scan subsequently confirmed by two-dimensional echocardiography and treated with pericardiocentesis is presented. There are few case reports about the role of both thallium and technetium-99m-based myocardial perfusion imaging in diagnosing incidental pericardial effusion and the useful information it can provide in diagnosing this condition. Some investigators discussed the role of thallium imaging in patients with pericardial effusions, while pericardial effusion was detected in two case reports. Pericardial effusions were found using sestamibi for myocardial perfusion and parathyroid imaging. The echocardiogram underestimated the amount of pericardial effusion compared to myocardial perfusion imaging in our case and in contrast to the previous published reports.

Echocardiography is the most common and sensitive method of detecting pericardial effusions. However, computerized tomography and magnetic resonance imaging scans are commonly performed procedures that could detect incidental pericardial effusions, but are usually not done for this purpose. A chest radiograph is insensitive to small effusions, but as in this case, may be useful in detecting large effusions.

Although an incidental finding, pericardial effusion can be diagnosed by myocardial perfusion imaging. Myocardial perfusion imaging is, therefore, useful in diagnosing incidental pericardial effusions, and it is important that physicians be aware of this imaging modality that is capable of identifying large effusions. However, it should not replace echocardiography because it remains the “gold standard” for diagnosing or ruling out pericardial effusions.

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Figure 1.
A: Chest radiograph on day of admission shows a “water-bottle” shaped heart (see arrows) with cardiac enlargement and prominent vascular markings. No evidence of interstitial edema or pleural effusion was seen.
B: Previous chest radiograph 3 years prior showing cardiomegaly and prominent vascular markings (see arrows).
C: Myocardial perfusion using Tc-99m sestamibi. Raw data show a large halo around the heart (see arrow).
D: Myocardial Tc-99m sestamibi perfusion images show normal myocardial perfusion, right ventricular prominence with increased wall intensity and thickness, and septal wall flattening. In addition, the short axis images also have a surrounding “halo” from the pericardial effusion (see arrow).
REFERENCES


