Echocardiographic Diagnosis of Pseudoaneurysm of the Left Ventricle

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SUMMARY
The echocardiographic features of postinfarction pseudoaneurysm of the left ventricle are described for the first time. Because ultrasound allows the detection of soft-tissue structures in a manner not possible with other diagnostic techniques, the left ventricular wall can be visualized separating the left ventricular cavity from the sacular aneurysm which is delineated by pericardium and/or extracardiac tissue. In addition to these anatomic findings, relevant qualitative hemodynamic data can also be obtained.

Echocardiography seems to be a safe and specific method for the diagnosis of left ventricular pseudoaneurysm. It is suggested that echocardiography should be used in the incipient phase of pseudoaneurysm formation to detect subacute cardiac rupture.

LEFT VENTRICULAR ANEURYSMS following myocardial infarction may be either true or false. Most common are true aneurysms where the wall is formed by elements of the infarcted myocardium or its tissue replacement. On the other hand, false or pseudoaneurysms are retained by pericardium or extracardiac tissue and communicate with the left ventricle. They may result either from cardiac rupture with localized hemopericardium by an adherent pericardium or from subacute rupture with gradual and/or episodic bleeding and formation of localized hematoma. Thus, instead of being immediately fatal, some cases of cardiac rupture may be compatible with survival and seem to be amenable to surgical cure, even in the acute phase of myocardial infarction.

Therefore, a safe and accurate diagnostic method for early clinical detection is desirable.

Following the acute period a typical pseudoaneurysm may develop. Reasoning from anatomic-pathological findings, it has been suggested that pseudoaneurysms should be distinguishable from the true type with angiography by the demonstration of a small channel to a sacculating aneurysm in a patient with previous myocardial infarction. However, only a limited number of cases are reported in literature where this diagnosis could be made with confidence.

The patient described in this paper demonstrates that ultrasound may provide a safe and accurate diagnostic method to visualize pseudoaneurysms because of its unique capability to detect soft-tissue structures. In this patient, successful surgical surgery was performed ten months after myocardial infarction complicated by cardiac rupture.

Case Report
A 51-year-old male without previous medical history was admitted to his local hospital on December 12, 1973 with an acute inferolateral myocardial infarction. From his file it was learned that during the first days of hospitalization, he was restless and anxious so that sedation was needed. The third day of hospitalization, precordial pain recurred associated with central venous congestion, some cardiac enlargement on the chest X-ray and persistence of ST-segment elevation on the ECG, and it was thought that this resulted from pericarditis. With conservative treatment, the clinical course was uneventful, no arrhythmias were noted and he was discharged from the hospital on January 19, 1974.

On May 10, 1974 he was rehospitalized for cardiac failure. At that time, cardiac enlargement suspected of a developing lateral aneurysm was found on the chest X-ray. Treatment with restriction of sodium intake, diuretics and digoxin relieved his symptoms.

On September 6, 1974 he again presented with clinical signs of cardiac failure and was then referred to our department. Physical examination showed a fast regular heart rate of 100 beats/min and arterial blood pressure of 130/80 mm Hg. Venous congestion was present but no peripheral edema was noted. A precordial paradoxical impulse was detected. By auscultation a gallop sound and a grade II/VI holosystolic murmur with maximal intensity at the axilla were heard. The lungs were normal at auscultation. The electrocardiogram (fig. 1) showed regular sinus rhythm at 100 beats/min with normal P waves and P-R interval. Q waves were present in leads II, III, aVF, V₅ with a tall R wave in V₁. Flat T waves were seen in leads I, II, III, aVF with ST elevation in V₅. This ECG was interpreted as showing an old infero-posterolateral myocardial infarction. The posterior-anterior chest X-ray taken at his first visit at our outpatient department is shown in figure 2. An enlarged cardiac silhouette with a large lateral bulge relieving the costal border was found. The differential diagnosis of such a film includes a true LV aneurysm, a pseudoaneurysm, loculated

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Figure 1

Electrocardiogram of the patient described. Sinus rhythm and Q waves in leads II, III, aVF, V6 and tall R wave in V1 are indicative of an old infero-posterolateral myocardial infarction. Persistent ST elevation in V6 is compatible with ventricular aneurysm.

Pericardial fluid or even pleural effusion. However, insomuch as the previous chest roentgenograms showed a progression of the lateral bulging, and in view of the clinical history, the diagnosis of a large postinfarction LV aneurysm was made.

Echocardiographic Study

The echocardiographic study was performed with the ECHOcardioVISOR 01 with both the conventional and the multiscan facilities. M-mode scans were made using an Aerotech 0.5 inch transducer focused at 7.5 cm and recorded on photographic paper with a Honeywell Visicorder 1856. A sector scan from the aorta toward the apex of the left ventricle is shown in figure 3. The study begins with the beam passing through the aorta (Ao) and the left atrium (LA) which occupies the echo-free space posteriorly. During recording the beam is angled towards the left ventricle (LV). The mitral valve is shown to be in continuity with the posterior aortic wall. The interventricular septum (IVS) angles anteriorly indicating a dilated LV outflow tract. Both the anterior and posterior mitral valve leaflets (aMVL and pMVL) are recorded throughout the cardiac cycle but their amplitude of movement is diminished. This is thought to reflect the reduced blood flow through the valve and hence a low cardiac output. The LV cavity is slightly dilated (6 cm). While the IVS shows a normal motion pattern, the posterior wall (LVPW) is hypokinetic at the level of the mitral valve. Moving the sound beam further towards the apex shows that the internal LV dimension abruptly diminishes. This is due mainly to an anterior deflection of the LVPW instead of a progressive tapering off as seen normally. There is clearly an echo-free extracardiac space. In view of the clinical history, it is likely that this is a false or

Figure 2

Posterior-anterior chest roentgenogram demonstrating cardiac enlargement and a large mass adjacent to the border of the left ventricle which was interpreted as a postinfarction left ventricular aneurysm.
pseudoaneurysm which compresses the LV, giving it in turn its peculiar shape. The internal end-diastolic dimension of the LV at the level of the pseudoaneurysm is 3 cm. The cardiac walls near the apex move normally in systole. However, the flat motion of the posterior wall echoes during diastole (square root sign) is suggestive of constriction as a result of tamponade or of compression by a space-occupying mass. In case of a true aneurysm, the myocardium or its tissue replacement should form the outer wall of the aneurysm whereas in this case the wall appears to be formed by the pericardium and/or extracardiac tissues. The echoes seen in the area where epicardium and pericardium separate were interpreted as clot formation. The fact that the pseudoaneurysm is sonolucent indicates that it is filled with fluid, which makes an open communication with the LV most likely.

The multiscan sagittal cross-sectional cardiac image along the long axis of the LV is shown in figure 4. A globular echo free space (black instead of white as on M-mode tracings) can be seen behind the LV in an infero-postero-lateral location. This is also obvious in the M-mode scan. The peculiar shape of the LV by the compression of its medial and apical portion can be appreciated.

Cardiac catheterization and angiography were then proposed. However, as the patient responded to medical treatment and as there was reluctance to accept the echocardiographic diagnosis, this procedure was delayed until December 11, 1974.

Catheterization and Angiography

Right and left heart catheterization data are summarized in table 1. Most prominent are the elevated right and left ventricular diastolic pressures and the low effective cardiac output (cardiac index 2.2 L/min), which is also reflected by the low mixed venous oxygen saturation (44%). The LV pressure curves showed a dip-plateau configuration with elevated initial diastolic pressure (16 mm) typical for external cardiac compression as was anticipated from the echographic motion pattern of the LV.

Cineangiography in the right anterior oblique projection showed an enlarged LV with akinesis of the inferoposterior wall. The measured minor axis was 8.2
normal LV shape invalidates the standard area-length formula,14 which assumes an ellipsoid of revolution to calculate intracardiac volumes, the calculated total cardiac output from the LV angiogram (4.5 L/min) is falsely high. This implies also that the tidal volume between LV and aneurysm (i.e., the angiographic calculated total volume minus the effective forward cardiac output) is also less than calculated. Left ventricular angiograms in the left anterior oblique projection were also performed. Although the diagnosis of pseudoaneurysm was anticipated, several injections were necessary before the communication between the LV and the pseudoaneurysm, which arose from the posterior wall, could be visualized. A representative frame is shown in figure 5. There is a communication between the LV and the pseudoaneurysm which is smaller than the largest diameter of the pseudoaneurysm. Note also the striking similarity of this frame with the ultrasonic cross sectional image of figure 4, although the planes which are visualized are different. In fact, the cross section from the base to the apex of the heart seen on the ultrasonic image is perpendicular to the superimposed angiographic image. Coronary arteriography revealed complete proximal obstruction of the left circumflex coronary artery and multiple lesions of less than 50% in both the right and left anterior descending coronary arteries.

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**Table 1**

*Right and Left Ventricular Catheterization Data of Patient with Postinfarction Pseudoaneurysm of the Left Ventricle*

<table>
<thead>
<tr>
<th>Site</th>
<th>Pressure (mm Hg)</th>
<th>O₂ Saturation (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Systolic</td>
<td>Diastolic</td>
</tr>
<tr>
<td>RA</td>
<td>17/11</td>
<td></td>
</tr>
<tr>
<td>RV</td>
<td>85/12–20</td>
<td></td>
</tr>
<tr>
<td>PA</td>
<td>85/45</td>
<td></td>
</tr>
<tr>
<td>Wedge</td>
<td>40/28</td>
<td></td>
</tr>
<tr>
<td>Ao</td>
<td>126/92</td>
<td></td>
</tr>
<tr>
<td>LV systolic</td>
<td>126</td>
<td></td>
</tr>
<tr>
<td>LV diastolic</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>dp/dt (LV)</td>
<td>1140</td>
<td></td>
</tr>
<tr>
<td>Vmax</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Eff. CI</td>
<td>2.2</td>
<td></td>
</tr>
</tbody>
</table>

**Angiographic data** (Heart rate, 100 beats/min)

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Normal</th>
<th>Normal</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDV</td>
<td>166</td>
<td>20 ml</td>
<td>70 ml</td>
<td>10 ml</td>
</tr>
<tr>
<td>ESV</td>
<td>120</td>
<td>9.5 ml</td>
<td>6 ml</td>
<td>10 ml</td>
</tr>
<tr>
<td>SV</td>
<td>46 ml/beat</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total CI</td>
<td>4.6 L/m²/min</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: RA = right atrium; RV = right ventricle; PA = pulmonary artery; Ao = aorta; LV = left ventricle; Eff. CI = effective cardiac index; EDV = end-diastolic volume; ESV = end-systolic volume; SV = stroke volume; Total CI = total left ventricular cardiac index.

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**Figure 5**

*Selected frame of the left ventricular (LV) angiogram in the left anterior oblique projection. The diameter of the mouth, between the LV and the aneurysm (PA) is smaller than the largest diameter of the globular aneurysm which is typical for a pseudoaneurysm (PA).*
Subsequently, the patient was operated upon and a large pseudoaneurysm communicating with the LV at the posterobasal region was found (fig. 6). The diameter of the opening was 2 cm. No papillary muscles were found within the pseudoaneurysm but much thrombotic material was present. The defect was closed and the pseudoaneurysm partially resected. Except for some respiratory problems, the postoperative course was uneventful.

At the time of discharge from the hospital, two weeks after the operation, no clinical signs of cardiac failure were present and the holosystolic murmur had disappeared.

A postoperative echocardiogram was recorded on January 30, 1975, which showed some apparent changes (fig. 7). The pseudoaneurysm was no longer present but there was a small posterolateral echo-free space at the level of the mitral valve ring (2 cm). Such a finding is a frequent postoperative feature.

Compression of the heart in an anterior direction has been relieved since the septum is now at a distance of 5 cm from the chest wall, instead of 2.5 cm preoperatively. The standard left ventricular (LV) dimension at the tips of both mitral valve leaflets is slightly

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Figure 6
Photograph taken at the time of operation showing the opened pseudoaneurysmal sac and the ventricular communication consisting of multiple small holes (arrows).

Figure 7
Postoperative echocardiogram. Because of the removal of the pseudoaneurysm, the left ventricle (LV) is more posteriorly positioned and the interventricular septum (IVS) is 5 cm from the anterior chest wall instead of 2.5 cm preoperatively. However, there is a small posterolateral echo-free space at the level of the mitral valve ring. This is not an unusual postoperative finding and may result from a loculated pericardial effusion. The standard LV internal dimension at the level of the tips of the mitral valves is smaller, most probably as a result of LV shape restoration. Ao = aorta; LA = left atrium; aMVL = anterior mitral valve leaflet; RV = right ventricle; LVPW = left ventricular posterior wall.
smaller (5 cm instead of 6 cm), most probably as a result of restoration of normal LV shape. Some abnormalities remain at the level of the papillary muscles which were also present preoperatively.

Discussion

Left ventricular false or pseudoaneurysms are thought to be rare because rupture of the free cardiac wall is generally considered rapidly fatal. Nevertheless, they were seen in nine of 40 cases with cardiac rupture after myocardial infarction studied by Van Tassell and Edwards. This high incidence probably resulted from a process of selection (referred cases of special interest). However, both the fact that pseudoaneurysm is one possible consequence of cardiac free wall rupture and the fact that the incidence of free wall rupture is as high as 5 to 10% after myocardial infarction may suggest that the true occurrence is higher than hitherto suspected.

Observations that rupture of the myocardium is not an immediate fatal event and may be clinically evident for some time prior to death, are also in favor of this hypothesis. Indeed, London and London, Lautsch and Lanks, and Van Tassell and Edwards, when correlating pathologic findings with clinical courses, found that prolonged periods of precordial pain often resulted from slow leakage of blood into the pericardial sac prior to clinically evident cardiac tamponade and death or, infrequently, survival and pseudoaneurysm formation. This points to the importance of having an accurate and reliable technique for the prompt diagnosis of this complication at the bedside, since acute surgical intervention may be possible. Echocardiography may fulfill this role more authoritatively since it has been demonstrated that its sensitivity allows the detection of as little as 15 ml of pericardial fluid (or blood). Once a pseudoaneurysm has formed, its high risk of rupture, as opposed to the infrequent rupture of true aneurysms, makes a specific and definitive diagnosis imperative. However, since a pseudoaneurysm closely mimics a true aneurysm in all aspects (history, clinical features, ECG and chest X-ray), cineangiography has been employed to provide a diagnosis. As the adequate visualization of the pseudoaneurysm, particularly the communication with the LV cavity, is largely dependent upon the care of the examination and the proper projections during filming, it is not surprising that only a few cases are reported in literature where the diagnosis of postinfarctional pseudoaneurysm was made with confidence. In fact, posttraumatic pseudoaneurysms and those developing following cardiac surgery are more frequently diagnosed correctly, since under these circumstances the diagnosis is anticipated.

The echocardiographic study of the patient described in this paper suggests that this technique is perhaps more accurate and certainly a safer method for the diagnosis of pseudoaneurysm. Although the echocardiogram obtained from this patient resembles the patterns obtained from patients with localized pericardial effusion, the final diagnosis must be established by consideration of the patient’s history, physical examination, clinical course, etc. The history, starting with a myocardial infarction located at the place where the lateral bulge on chest X-ray appeared, suggests an aneurysm rather than localized effusion. In addition we have never seen a localized effusion of this size. The smaller localized echo-free space as seen postoperatively (fig. 7) is more characteristic for loculated effusions.

One can hardly expect any one technique to be diagnostic but echocardiography should be an important part of the work-up of such a patient. In this instance, it proved decisive. The unique possibilities of echocardiography result chiefly from its ability to visualize soft tissues in differentiating such cardiac and extracardiac tissues as myocardium and pericardium. Another advantage is the display of these structures in a direct and positive way rather than the shadow image of these structures obtained with angiographic techniques. Furthermore, since the structures are superimposed in depth, the angle of X-ray beams is often critical. This may result in a false negative diagnosis of pseudoaneurysm when the neck or site of perforation is not properly oriented with the X-ray beam in the projection chosen. It is also important to stress that echocardiography provides information regarding the hemodynamic state of the heart. In this particular patient, a qualitative hemodynamic assessment could be made in addition to the anatomic diagnosis.

Echocardiography also added information about the LV shape and thus placed the quantitative angiographic data in a true perspective. A limitation of currently used echo systems is their beam width. This limits the ready visualization of ventricular wall defects, such as the perforation in this case.

It is concluded from this case report that echocardiography can provide detailed information about cardiac anatomy and the hemodynamic state. It should be employed in all patients in whom a true or false LV aneurysm is suspected prior to the use of more invasive methods. In some cases, the latter methods may be unnecessary as they will not yield additional information and their associated risk may thus be avoided.

References


Echocardiographic diagnosis of pseudoaneurysm of the left ventricle.
J Roelandt, M Brand, W B Vletter, J Nauta and P G Hugenholtz

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