True and False Left Ventricular Aneurysms
Propensity for the Latter to Rupture

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SUMMARY
Two cases are described of sudden death resulting from rupture of small chronic false aneurysm of the left ventricle with secondary hemopericardium. The cases support the point that rupture of chronic false left ventricular aneurysm tends to occur. This appears to be contrary to the potential for chronic true aneurysms of the left ventricle which tend not to rupture. Rupture of true left ventricular aneurysm in its developing stage may, however, occur.

Additional Indexing Words:
Sudden coronary death
Rupture of heart
Hemopericardium

ANEURYSMS OF THE LEFT VENTRICLE usually result from myocardial infarction. Two types are recognized, the true and false, each complications of transmural infarction. The true aneurysm develops by a gradual bulge of the involved portion of the wall. The process results from removal of infarcted tissue and concomitant moulding of the bulge. The final result is an aneurysm with a wide mouth. The wall of the true aneurysm is comprised primarily of fibrous tissue among which elements of the original wall may be identified (figs. 1 and 2). Commonly, left ventricular aneurysms, whether true or false, contain mural thrombi which are constant potentials for systemic embolus (fig. 3).

Classically, the true aneurysm is relatively large, frequently six to seven centimeters in diameter. In exceptional cases, true aneurysms may be smaller (fig. 4).

The false or pseudoaneurysm is a consequence of rupture of the ventricular wall but with containment of the resulting hematoma. With time, the periphery of the hematoma becomes organized into fibrous tissue among which no elements of the cardiac wall are present. Characteristically, the mouth of a false aneurysm is narrow compared with the width of the fundus (fig. 5).

The functional effects of true aneurysms are shared by false aneurysms. Each exhibits paradoxical motion, a feature which wastes energy expended by the functioning muscle of the left ventricle.

An important difference, however, exists with regard to the potential for rupture of true aneurysm as compared to false aneurysms.

In the early stages of the development of a true aneurysm, about two to three weeks after the onset of the underlying myocardial infarction, rupture through the center of the aneurysm may, as an uncommon event, occur (fig. 6). When, however, the fibrous stage of a true aneurysm has appeared, the potential for rupture is, in our experience, a very rare phenomenon.

This situation stands in marked contrast to false aneurysm. These may not only rupture during their early stages of development but, in sharp contrast to true aneurysm, may rupture after the established fibrous stage is reached.

While rupture of a large false aneurysm may be taken as an understandable event, it is of interest that even the small false aneurysm may rupture. To emphasize the latter point, we relate experience with two cases of rupture of relatively small false aneurysms of the left ventricle.

In each of the cases, sudden and unexpected death had occurred. Were no autopsy done, it is likely that death in each case would have been attributed to an
True aneurysm of left ventricle in anterior apical region. a) Exterior of heart in lateral view shows bulge of aneurysm (between arrows). b) Interior of left ventricle. Aneurysm develops as gradual thinning of the ventricular wall. The bases of the papillary muscles are involved by scarring of healed infarction.

True aneurysm of posterior left ventricular base. a) Exterior of heart viewed from left side. The aneurysm (between arrows) protrudes from the posterior basal wall. b) Interior of left ventricle. The aneurysm is related to the posteromedial papillary muscle (P.). c) Low power photomicrograph of the left ventricular wall and the aneurysm. The latter contains residual elements of the cardiac wall as is classical for a true aneurysm. (Elastic tissue stain; × 1.)
True aneurysm of anteroseptal region of left ventricle. A mural thrombus is contained. This is a classical situation in ventricular aneurysm, whether true or false.

Small true aneurysm of posterolateral wall of left ventricle. a) Gross specimen shows the aneurysm related to the posteromedial papillary muscle (P.). b) Photomicrograph. The wall of the aneurysm contains elements of the cardiac wall. (Elastic tissue stain; X 1).

Two examples of large false left ventricular aneurysms. a) A small ostium (between arrows) leads from the left ventricular cavity into a large, fibrous-walled sac. Discoloration along upper aspect of the sac represents hemorrhage resulting from rupture of the false aneurysm. b) A large false aneurysm communicates with the cavity of the left ventricle (L.V.). Abrupt loss of muscular continuity exists at the ostium of the aneurysm. The spur in the aneurysm may represent an old site of rupture of the false aneurysm.
Figure 6
Rupture of developing true aneurysm of left ventricular wall. a) Interior of left ventricle. At the center of a hollowed out area, representing a developing left ventricular aneurysm, is a perforation (between arrows). b) Exterior of heart. The site of rupture is indicated between the arrows. Hemopericardium was the cause of death. c) Photomicrograph of the infarcted wall showing a picture of removal of tissue consistent with an age of infarction of about two weeks. (H & E, × 75.)

Figure 7
Rupture of small false aneurysm of left ventricle in a 77-year-old woman (case 1). a) Exterior of heart. Protruding from the left ventricular wall is an aneurysm which has ruptured leading to hemopericardium. b) Sections through left ventricular wall and the false aneurysm. Characteristically, there is an abrupt loss in continuity of ventricular muscle at the ostium of the false aneurysm. c) Low power photomicrograph of the portion shown in rectangle in b. There is scarring of the left ventricular wall and abrupt interruption. The wall of the false aneurysm is formed by fibrous tissue and is continuous with the left ventricular wall. (Elastic tissue stain; × 4.)
Rupture of small false aneurysm in a 70-year-old man (case 2). a) Lateral wall of left ventricle showing two false aneurysms. The upper lies between arrows; the lower is partly out of view. Its ostium contains the probe. P. = posteromedial papillary muscle. b) Lateral wall of left ventricle sectioned at a deeper level than in a showing the upper aneurysm (between arrows), as well as the lower aneurysm. The latter had ruptured. c) Low power photomicrograph of the upper of the two false aneurysms. There is abrupt loss in continuity of the scarred left ventricular wall. The false aneurysm is walled by fibrous tissue. (Elastic tissue stain; × 4.) d) Low power photomicrograph of the larger of the two aneurysms, the one that had ruptured. The features are similar to those shown of the upper aneurysm in c. Of additional interest is the fact that in the wall of the aneurysm at this plane of section is a coronary artery (C.). At this level of section, hardly more than the coronary artery forms the wall of the false aneurysm. (Elastic tissue stain; × 3.)
"electrical" event. In each, the autopsy showed that a small false aneurysm of the ventricle had ruptured leading to fatal hemopericardium. Also, while there were pathologic signs of healed myocardial infarction (leading to false aneurysm), no history of antecedent myocardial infarction was obtained.

**Case Reports**

**Case 1**

A 77-year-old woman had evidently been well until she was found dead under a hair dryer. The autopsy showed an extensive hemopericardium resulting from rupture of a false aneurysm of the lateral aspect of the left ventricle. No pericardial adhesions were present. The aneurysm, which measured about 2.0 cm in greatest dimension, was situated in the basal aspect of the left ventricle near the base of the anterolateral papillary muscle. In addition to extensive coronary atherosclerosis, there was a healed myocardial infarction surrounding the ostium of the aneurysm. The wall of the aneurysm was composed of a thin layer of fibrous tissue which did not contain any elements of the original cardiac wall and fulfilled the definition of a false aneurysm (fig. 7).

**Case 2**

A 70-year-old man had considered himself in good health. Five years before his death, he had consulted a physician for a boil but had not sought medical attention since that time. On the day of his death, he arose in apparently good health. Shortly after eating breakfast, he was found unresponsive on the dining room floor. When medical assistance came, he was pronounced dead.

The significant findings at autopsy were confined to the heart and pericardium. The latter was distended with partly clotted blood and showed no adhesions. The source of hemopericardium was a ruptured aneurysm of the posterolateral wall of the left ventricle. The coronary arteries showed widespread, extensive atherosclerosis. In the posterolateral aspect of the left ventricle were two aneurysms separated by scarred cardiac muscle. Each of the aneurysms exhibited a similar appearance. At the mouth of each there was an abrupt loss in continuity of the ventricular wall. In this area, scarred myocardium was continuous with the thin fibrous wall of the aneurysm which did not contain any elements of the cardiac wall. The features of each of the aneurysms fulfilled the characteristics of a false aneurysm of the left ventricular wall (fig. 8).

The upper of the two aneurysms was the smaller, measuring about 2.0 cm in greatest dimension. The lower of the two aneurysms was the larger and had ruptured. It is of passing interest that the section obtained for histologic examination was cut along the course of a related coronary artery. At this plane of section, the wall of the aneurysm was made up only of the artery and a small amount of related fibrous tissue (fig. 8d).

**Comment**

The two cases to which specific reference was made have considerable similarity. In each, relatively small false aneurysms had resulted from myocardial infarction which was apparently silent in nature. That the false aneurysm had been present for many months or years is probable, as judged by the pathologic features.

The cases portray the point that false aneurysms of the left ventricle, even when small, have a propensity to rupture. This feature appears not to be shared by chronic true aneurysm of the left ventricle but rupture of a true aneurysm in its developing stage may, in an occasional patient, rupture.

False aneurysms of the left ventricle are accepted as sequelae of rupture of the ventricular wall complicating myocardial infarction. Rupture usually results in a rapidly accumulating hemopericardium and death. Only in exceptional cases does the hematoma, which extends through the full thickness of the myocardium, become contained, thereby setting the stage for organization of the periphery of the hematoma and formation of a chronic false aneurysm. In some cases, containment of the hematoma following ventricular rupture seems to be accomplished by adhesion of the parietal pericardium to the site of rupture. In our cases 1 and 2 no such adhesion was present so that this explanation for containment of the hematoma does not apply. It is of interest that in case 2 the section taken through one of the false aneurysms shows a coronary artery in its wall. From this observation, one must conclude that, at least at this level, the hematoma had not extended through the epicardium.

Of additional interest with regard to the two specific cases cited is that they represent unusual examples of sudden, unexpected death. It is probable, had autopsies not been done, that the death of the man (case 2) might have been considered a classical case of sudden electrical death from coronary disease. The case of the woman (case 1) might have been considered an example of accidental electrical death related to hair drying.

**References**


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