False aneurysm of the left ventricle

In this issue four cases are described1–4 in which the authors illustrate their experience with the very uncommon surgical challenge of false (or pseudo) aneurysm of the left ventricle. Although this condition is seen infrequently, it should be recognised and distinguished from the common type of left ventricular aneurysm. It is said to have a propensity for rupture, and may cause symptoms of heart failure. It is eminently operable if it is approached with an understanding of its anatomy and certain characteristic features.

The basis of the condition is a contained rupture of the free wall of the myocardium.5,6 Acute rupture of the myocardium is in fact quite common. Of patients dying of infarction, 17% have been found to have ruptured the heart through the infarcted area.7 Free wall rupture is four to five times more common than septal rupture,8 presumably a simple reflection of the relative volumes of myocardium at risk, but it is usually immediately fatal. These are among the cases described as “EDM” (electromechanical dissociation) when they collapse some days after infarction, with a peak incidence at 3–5 days.9 The ventricle is beating but no output is generated because of tamponade. The patients with ruptured myocardium who survive to operation in the period soon after infarction, we see as the familiar acute septal rupture, loosely known as “infarct VSD”. The UK Cardiac Surgical Register (of the Society of Cardiothoracic Surgeons of Great Britain and Ireland) records about 200 operations each year for this condition, with a mortality of 35–40%. This has changed very little over the past 20 years. Ruptured papillary muscle is also a familiar surgical condition.10

The early descriptions of false aneurysm of the ventricle are dominated by pathological descriptions from Jesse Edwards’s department.6–8 The cases were identified postmortem and included patients dying of late rupture of the false aneurysm. As a surgeon, the few cases that I see are the survivors who come to operation with the aneurysm intact. What is the true natural history and how many will rupture if not treated? The cases in the literature are so sporadic, documented in ones9–17 and twos18–20 or small series21–24 that I am very uncertain of the characteristic natural history. Without more data of the true denominator, all that can be said is that rupture is a risk3–8,14,18,20 but its true frequency is unknown. In life, false aneurysm is most often a finding in the course of investigation of a patient with ischaemic heart disease. It has been described following mitral valve replacement, presumably as a late presentation of posterior wall rupture.25 A false aneurysm may be picked up by echocardiography, or on left ventricular angiography. Occasionally they are so large that the plain chest x-ray shows a dramatic bulge. Once suspected, echocardiography is probably the most practical way of assessing the anatomy and monitoring change in size26 while magnetic resonance imaging gives unequalled anatomical information.2

In pathological terms a typical ventricular aneurysm can be regarded as a “true” aneurysm in the sense that the sac contains the three layers of the vessel wall, the endocardium and epicardium sandwiching a layer of thinned fibrous tissue that is the remnant of the left ventricular muscle. The wall of a false aneurysm consists of adherent pericardium and associated postinflammatory scar tissue, with some remnants of the epicardium. The key for the clinician is to be able to distinguish, in life, false aneurysm from the familiar left ventricular aneurysm. In contrast to the typical postinfarction aneurysm, which is in wide open continuity with the cavity of the left ventricle, the false aneurysm has a well defined neck, which is the rent in the infarct, through which the aneurysm arose. Typically this is relatively narrow and consists of stout scar tissue, in marked contrast with the tapering poorly defined zone of continuity between ventricular muscle and the usual left ventricular aneurysm. Repair is thus easy, both because the suture line is easily defined and the tissues lend themselves to secure surgical repair.

The indications for operation are debatable, and in such a rare entity each case must be considered on its merits. If surgery for coronary artery disease is indicated on the usual grounds, repair of such a defect in the wall of the ventricle is easily undertaken at the time. If there are manifestations of left ventricular dysfunction we get into the difficult discussion of whether the symptoms are likely to be alleviated by resection of the aneurysm. The phase image of a MUGA scan seems to me to be the best way to make the decision if it is difficult. However, if a substantial proportion of the left ventricular work is involved in filling and tensing a large but useless sac, its exclusion is likely to be of benefit. That has been my experience, albeit in a very small series of cases. Finally, should we consider whether repair is indicated purely to preempt rupture. That is the received wisdom in the text books.26 The argument would at least make weight in favour of surgery if an operative decision is in the balance. It is also a compelling reason for operating on an incidentally discovered aneurysm in a patient being operated on for angina. There may be a case for regular monitoring and using change in size as an indication, but there are no data available to guide us.

A final word about surgical technique—the mistake is to think in terms of dissecting out, mobilising, and then resecting the aneurysm. The word “resection” is best excluded from the operative discussion at the outset, because it indicates a dangerous and inappropriate approach to this pathological entity. The surgeon should establish bypass, gain control, prevent ejection by cardioplegia or left ventricular fibrillation, and then enter the sac.25,26 This both reduces the risk of disastrous bleeding and avoids systemic embolism.37 The neck must be defined but with no more than essential dissection. Direct suture is usually easy and, unless there has been an error in diagnosis or technique, there should be no need for foreign material such as pledgets, or the use of a patch. Any clot or debris can be tidied up, but the thin sac of pericardium can simply stay where it is.

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