Subepicardial aneurysm is a rare complication of acute myocardial infarction (AMI) and is associated with the risk of spontaneous rupture. We present two patients with subepicardial aneurysms detected after 1 day and 6 weeks after AMI. Both lesions were successfully treated by patch repair. (Ann Thorac Cardiovasc Surg 2010; 16: 291–293)

Key words: coronary disease, myocardial infarction

Introduction

A subepicardial aneurysm is one of the critical complications following acute myocardial infarction (AMI); others include free wall rupture, ventricular septal perforation, and papillary muscle rupture. Although the morphological characteristic of subepicardial aneurysm is abrupt interruption of the myocardium without epicardial damage, the lesion can reportedly progress to an external expansion of subepicardial aneurysm, which has the risk of spontaneous rupture. However, the mechanisms of progression of the lesion and the clinical course of this complication are not well understood. We present two cases of subepicardial aneurysm. One was detected at one day post-AMI, and the other 6 weeks post-AMI. Both patients underwent patch repair.

Case 1

A 74-year-old man was admitted as a result of severe dyspnea. Electrocardiography showed ST elevation on leads II, III, and aVF, and we therefore suspected AMI. Coronary angiography revealed total occlusion of the right coronary artery (RCA) (#1) and 75% stenosis of the left circumflex artery (LCX) (#13). The RCA was recanalized by percutaneous catheter intervention (PCI) under intracoronal balloon pump (IABP) support. The next day, two-dimensional echocardiography disclosed an interruption of the inferior wall myocardium (1.3 × 1.1 cm) without external bulge (Figs. 1A and 1B). IABP assist was required for 2 weeks because of severe cardiac failure, and morphological alteration was observed on echocardiography. Elective surgery was performed 6 weeks after the onset. The surface of the infarcted area appeared to be scarred with a thin wall, and the limb was sufficiently robust to anchor the patch. Patch plasty with equine pericardium and concomitant coronary artery bypass grafting (CABG) for #14 was performed. Weaning from cardiopulmonary bypass was straightforward without IABP support. The postoperative course was uneventful.

Case 2

A 65-year-old man was transferred to our hospital as a result of severe dyspnea. Electrocardiography revealed ST elevation on leads II, III, and aVF, indicating AMI. Coronary angiography demonstrated 25% stenosis of the left anterior descending artery (#6) and total occlusion of the RCA (#3) and LCX (#13). The RCA and LCX were recanalized by PCI under IABP support, which was gradually withdrawn after 4 days. Three weeks after the onset, PCI was performed for the RCA (#4AV) without...
IABP support. Six weeks after the onset, two-dimensional echocardiography revealed a large aneurysm (4.9 × 3.6 cm) originating from a narrow orifice (2.6 × 2.4 cm) on the posterior wall of the left ventricle (LV) (Figs. 2A and 2B). Consequently, urgent surgery was performed. The aneurysm was opened, and the firm lip of the orifice was identified. The root of the anterolateral papillary muscle was located in close proximity to the lip. The firmness of the lip of the orifice facilitated stable anchoring of the equine pericardial patch, and concomitant CABG for #7 and #14 was performed. The postoperative course was uneventful.

Comment

A subepicardial aneurysm is a rare complication following AMI. It is characterized morphologically by abrupt interruption of the myocardium without epicardial damage and is associated with a risk of spontaneous rupture. Although a progression of external expansion of subepicardial aneurysm, which would be the anatomical indicator of rupture, requires urgent surgery, appropriate timing for surgical treatment remains unclear if no external bulge has been formed.

When a subepicardial aneurysm is detected in the early phase of AMI, intensive medical therapy is recommended for patients with severe cardiac failure. Surgical correction should be planned after recovery from acute myocardial damage (around 4−6 weeks) in the absence of the external expansion of subepicardial aneurysm. However, considerable attention should be paid to morphological alteration of a subepicardial aneurysm during medical therapy. It has been reported that a subepicardial aneurysm detected after 10 days following AMI is associated with the risk of rupture, though mean interval from the onset of AMI to rupture was 8 weeks. In our first case, we controlled cardiac failure medically for 6 weeks and sequentially observed anatomy of the subepicardial aneurysm by echocardiography during treatment. An interval of 6 weeks from the onset of AMI, which would be sufficient for the aneurysmal orifice to become firm enough for anchoring sutures, is thought to be optimal surgical timing in terms of myocardial recovery and reduced surgical risk.

Most documented cases of subepicardial aneurysms found in the late phase of AMI had already undergone external expansion of subepicardial aneurysm and were successfully repaired by patch plasty. With this time course, cardiac function should have recovered from acute myocardial damage, and an urgent operation would be indicated to avoid rupture. Even if external expansion does not occur, surgical correction is necessary because of possible progression. In such situations, patch plasty is among the appropriate surgical procedures when the orifice has a narrow neck and a firm myocardial lip. This was why we performed an urgent repair of a subepicardial aneurysm by patch plasty in our second case.

Optimal surgical timing for repair of subepicardial aneurysms depends on the interval from the onset of AMI to the operation, which is associated with preoperative
cardiac function and stability of ventricular suture lines. Serial echocardiography should be performed to detect the formation of external expansion of a subepicardial aneurysm, which requires an urgent operation.

References


Fig 2. Two-dimensional echocardiography six weeks after the onset of AMI in case 2 patient. Apical long axis (A) and short axis (B) views showed the aneurysmal formation originating from myocardial interruption in the posterior wall between the root of the anterolateral papillary muscle and the mitral valve. The narrow orifice (2.6 × 2.4 cm) is indicated by a double arrow (↑↑).