How-to-do-it

Surgical repair of coronary arteriovenous fistula: a simple and useful approach to identify the fistulous communication

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Received 30 April 2001; received in revised form 3 July 2001; accepted 10 July 2001

Abstract

In repairing coronary arteriovenous fistula (CAVF), it is very important to interrupt the fistulous tract without compromise of normal coronary vessel flow. In our case, selective coronary arteriography showed that the CAVF from the left anterior descending coronary artery (LAD) was very close to the native coronary artery and had a very broad and short neck. We describe a simple and useful approach, by using both antegrade and retrograde coronary perfusion, that makes it possible to certainly protect myocardium and to clearly distinguish the normal native coronary artery from the fistulous tract.

Keywords: Coronary arteriovenous fistula; Selective coronary arteriography; Angina pectoris; Antegrade coronary perfusion; Retrograde coronary perfusion; Aneurysmal formation; Surgical repair

1. Introduction

The surgical management of coronary arteriovenous fistula (CAVF) is controversial, but its goal is interruption of the fistulous tract without compromise of normal coronary vessel flow. Here, we describe a simple and useful method to repair CAVF which helps to identify the fistulous communication exactly and prevents the injury of normal coronary artery.

2. Case description and method

The patient was a 75-year-old woman with a history of asthma. She was referred to our cardiology unit, because she had progressive angina pectoris on exertion during the previous 2 months. The electrocardiogram was normal, and chest radiographs showed only slight cardiac enlargement and slightly increased pulmonary vasculature. Physical examination revealed no continuous cardiac murmur. Selective coronary arteriography showed no significant organic stenosis, but CAVF with multiple giant aneurysmal formation (maximum diameter of 30 mm) originating from both the conus branch of the right coronary artery (RCA) and the proximal parts of the LAD draining into the main pulmonary artery (Fig. 1a). Chest computed tomographic scan also demonstrated the fistulas. Although dipyridamole thallium scintigraphy revealed no apparent ischemia, on the basis of these findings, we presumed that the cause of the angina pectoris was a coronary steal effect [1] of blood into the tortuous fistulas with multiple giant aneurysmal formation, so that surgical repair was advised.

Operation was performed through a median sternotomy. On opening the pericardium, the appearance of the fistulas was found. At first, the fistulous communication from the conus branch was directly ligated, before cardiopulmonary bypass (CPB) was established, using 5-0 monofilament suture because it was clearly identified. After systemic heparinization, the patient was placed on CPB both with separate venous return cannulas placed in the superior and inferior vena cava and with the arterial cannula from the ascending aorta. Myocardial protection by crystalloid cardioplegia was ensured antegrade, at first, but the heart did not stand still easily, and then its protection was switched retrogradely from the coronary sinus. After that, the heart promptly came to a standstill and the part of giant aneurysmal formation was incised. There was some amount of mixed thrombi consisting of both an old white one and a relatively new reddish one. After its removal, we visually confirmed the exact drainage site of fistula by infusing cold blood cardioplegia antegrade and closed it using 5-0 monofilament suture with felt. After that, cold blood cardioplegia was perfused antegrade and the preservation of native LAD was confirmed. Next, pulmonary arteriotomy was carried out, and we checked out the opening of the
fistula, which was easily visible, draining into the pulmonary artery, and closed it from its inside. Finally, the fistula that was incised and the pulmonary arteriotomy were closed. The aortic cross-clamp time was 77 min, and the CPB time was 122 min. The patient had an uneventful postoperative course and did not have any recurrence of angina.

Postoperative selective coronary arteriography revealed no vessels connecting the coronary and pulmonary arteries and complete disappearance of the aneurysms (Fig. 1b).

3. Comment

CAVF, first described by Krause in 1865 [2], is an uncommon lesion that is usually congenital and isolated [3]. Because of the lack of specific symptoms, it may remain undetected for many years. However, because of the widespread use of cardiac catheterization in the evaluation of patients with chest pain, it is likely that this anomaly has been discovered with increasing frequency. Slightly more than half arise from the RCA and in most cases drain into the right ventricle. Other drainage sites in decreasing order of frequency include the right atrium, pulmonary trunk, coronary sinus, superior vena cava, left ventricle and left atrium. Both coronary arteries, like our case, are involved in only about 5% of cases [4]. Based on a very large number of coronary arteriographies, Yamanaka and Hobbs reported the incidence of coronary artery anomalies to be 1.3%; 3.7% of these patients had multiple or large CAVFs [5]. Patients with large fistula may be associated with potentially serious sequelae such as angina pectoris due to coronary steal phenomenon [1], embolization of mural thrombi to the distal coronary bed with subsequent myocardial infarction [6], or sudden death due to its abruptly spontaneous rupture [7]. So, once a precise diagnosis is established, some surgical intervention should be taken into consideration to prevent the development of significant and potentially fatal complications [4].

Successful surgical management is dependent upon a thorough preoperative evaluation which precisely defines the anatomy and pathophysiology of the anomaly. The diagnosis, therefore, requires arteriographic demonstration of the involved coronary artery, the recipient cardiac chamber, and the exact site of communication. The surgeon must be aware of the abnormal anatomy in order to avoid accidental ligation or transection at the time of surgery. Moreover, careful delineation and preservation of myocardial perfusion is essential in preventing perioperative myocardial ischemia and infarction.

In our case, we had great difficulty in identifying the fistulous communication and had a fear of ligating LAD, because the CAVF from the LAD was very close to the native coronary artery and had a very broad and short neck (Fig. 1a). In order to solve these problems, we performed the operation under cardiac standstill establishing CPB and both antegrade and retrograde cardioplegic perfusion, which could afford good myocardial protection even in this case with coronary steal phenomenon due to the multiple giant aneurysmal formation, and which made it possible to clearly distinguish the normal native coronary artery without injury from the fistulous tract. We regard this simple supplementary means as one of the most simple and useful approaches not only to prevent myocardial ischemia but also to identify the fistulous communication.

References


