

CASE REPORT

Acquired right coronary artery fistula draining to the right ventricle: angiographic documentation of first appearance following reperfusion after acute myocardial infarction, with subsequent spontaneous closure

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Most coronary artery fistulae are congenital in origin but have been reported to be acquired as complications of chest trauma, coronary angioplasty, or rupture of a coronary artery aneurysm. This is the first angiographic documentation of a coronary fistula acquired after myocardial infarction that subsequently closed spontaneously during follow up

been reported as complications of chest trauma, coronary angioplasty, or rupture of a coronary aneurysm. We describe a patient who developed a fistula between the distal posterior descending branch of the right coronary artery and the right ventricle after recanalisation of the thrombotically occluded coronary artery.

CASE PRESENTATION

A 45 year old man was admitted to hospital with severe chest pain of recent onset. The ECG showed an acute posterior myocardial infarction with extension to the lateral precordial leads. Thrombolysis with intravenous front loaded tissue plasminogen activator was initiated. Because of persistent chest

Coronary artery fistula is a rare condition in which a communication exists between a coronary artery and a cardiac chamber or systemic vein. Most fistulae are congenital in origin. Acquired coronary artery fistulae have

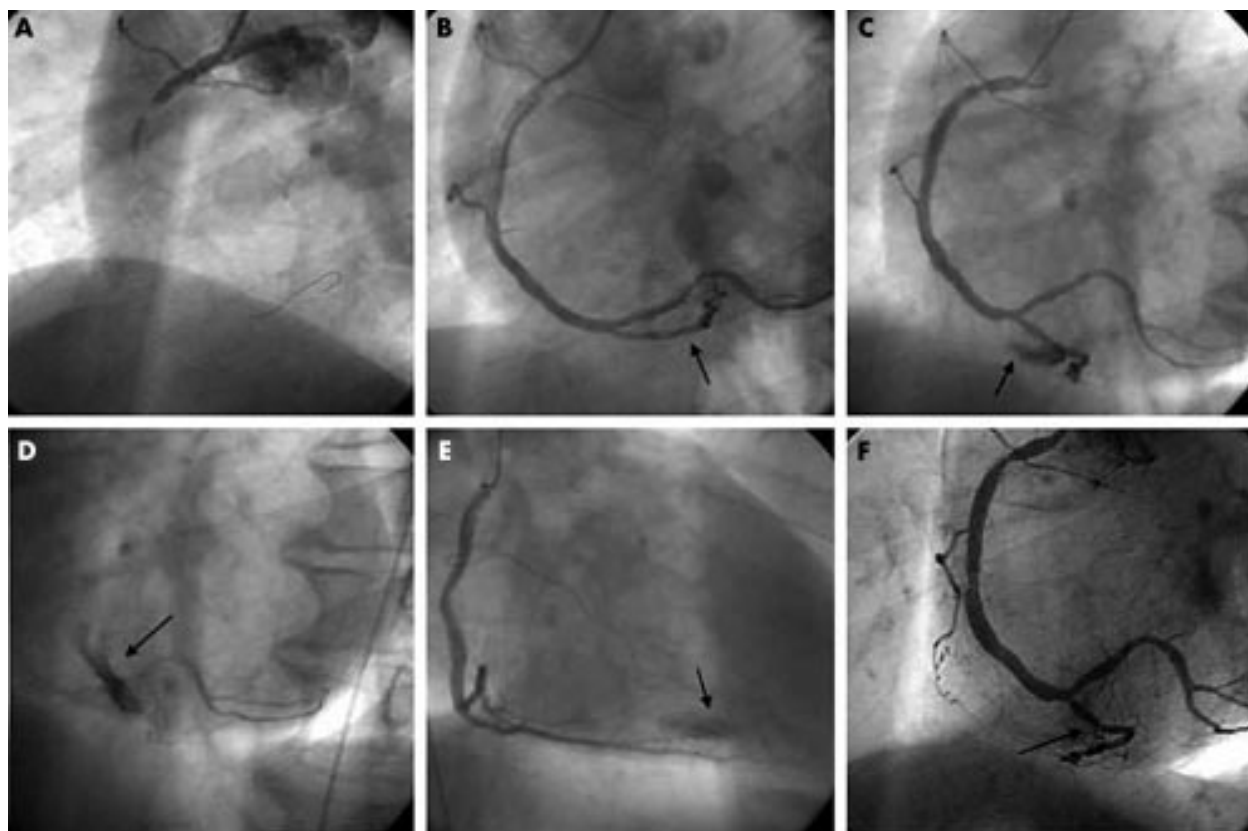


Figure 1 (A) Guide wire perforation of the right coronary artery occlusion. (B) Complete reperfusion after stenting. The posterior descending coronary artery is intact (arrow). (C) Control angiogram after six months showing a fistula to the right ventricle (arrow). (D) Late phase of the angiogram delineating the fistula. (E) Right anterior oblique projection of the fistula. (F) Angiogram 18 months later showing spontaneous closure of the fistula.

pain, ongoing ST segment elevation, and hypotension, emergency coronary angiography was performed.

Angiography showed no narrowing of the small left coronary artery. The dominant right coronary artery contained a proximal obstruction with faint penetration of contrast material (fig 1A, video sequence 1). Neither antegrade nor retrograde perfusion of the distal right coronary artery was observed. A guidewire was placed across the occlusion followed by primary implantation of a 4.0 mm AVE stent (Advanced Cardiovascular Systems, Temecula, California, USA), which initiated immediate and rapid reperfusion of the artery (fig 1B, video sequence 2). There were no signs of distal embolisation of thrombus. However, contrast material in the distal septal part of the interventricular posterior right coronary artery persisted after washout but did clear almost completely before the next injection of contrast material (fig 1C, video sequence 3). There were no extravasations of contrast material into the ventricular cavum. Transient third degree atrioventricular block developed but resolved after atropine administration. During the next few days, the patient developed pericarditis with a small pericardial effusion but he nevertheless finally recovered uneventfully.

After six months, the patient was readmitted for coronary angiography because of recurrent atypical chest pain. Left ventriculography showed an area of decreased contractility of the inferior wall resulting from the prior infarction. Global ejection fraction was 45%. The right coronary artery was patent without any intimal hyperplasia in the stented coronary arterial segment. However, angiography showed a newly developed fistulous communication between the posterior descending branch of the right coronary artery and the right ventricular chamber (fig 1D,E, video sequences 4 and 5). Dye emptied rapidly through a coarsely meshed network of arterioventricular communications. The location of the fistula corresponded to the area of delayed contrast clearance observed immediately after reperfusion (fig 2). Left coronary artery angiography displayed no pathologies. Medical treatment was recommended. However, 18 months later the patient was readmitted because of reoccurrence of chest pain. Embolisation of the fistula with 0.0018 inch fibred platinum coils was considered. Surprisingly, repeat angiography of the right coronary artery showed spontaneous closure of the fistula (fig 1F, video sequences 6 and 7).

DISCUSSION

Although there are several reports of congenital fistulae originating from coronary arteries, an acquired fistula to the right side of the heart appears to be an extremely rare phenomenon. Most reported fistulae involving an infarcted wall segment drained to the left ventricle. The cause of an infarct associated fistula is controversial. Congenital arteriosystemic communications associated with a coronary steal syndrome can some-

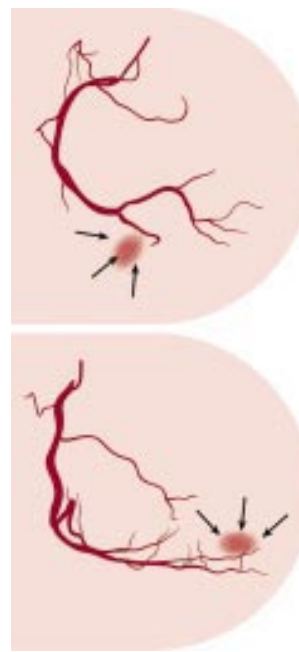


Figure 2 Schematic representation of the right coronary artery illustrating the fistulous communication (arrows) between the posterior descending right coronary artery and the right ventricular chamber.

times be responsible for ischaemic complications. Because of this, a congenital fistula may be causally related to myocardial infarction and be coincidentally discovered at the time of cardiac catheterisation. However, serial coronary angiography excluded a congenital fistula and clearly documented the acquired nature of the fistula in our patient. Ruptures of localised micronecrosis of the subendocardium due to destruction of the microvasculature probably resulted in drainage to the right ventricular cavity. However, the spontaneous closure of the fistula strongly suggests a functional cause. Therefore, it is more likely that the communication evolved from extensive collateralisation and reopening of the thebesian veins. To our knowledge, this is the first angiographic documentation of a coronary fistula acquired after myocardial infarction that subsequently closed spontaneously during follow up.

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