

Coronary Artery–Left Ventricle Fistula Complicating Balloon Angioplasty

A Case Report

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ABSTRACT

The authors describe a coronary artery fistula complicated balloon angioplasty. The proximal left anterior descending coronary artery was dilated, but a septal branch was occluded by thrombus. Angioplasty was used on the septal branch, but a pseudoaneurysm communicating with the left ventricle occurred. Follow-up angiography revealed spontaneous closure of the fistula.

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Introduction

Coronary artery perforation is a rare but serious complication of percutaneous coronary interventions.¹⁻⁴ Perforation of the epicardial coronary artery potentially leads to acute cardiac tamponade, requiring pericardiocentesis or surgery.^{3,4} In this report, we describe a unique complication consisting of a septal branch perforation during balloon angioplasty that caused a left anterior descending coronary artery (LAD)-left ventricular fistula. No adverse effects associated with the fis-

tula occurred, and the fistula spontaneously closed within 5 months.

Case Report

A 75-year-old man was admitted to our hospital in November 1997 for reevaluation of coronary artery disease. He had suffered from an acute posterolateral myocardial infarction in 1991. At that time, coronary angiography revealed a subtotal occlusion of the mid-portion of the right coronary artery (RCA), a 50% stenosis of the proximal LAD, and a total occlusion of the proximal

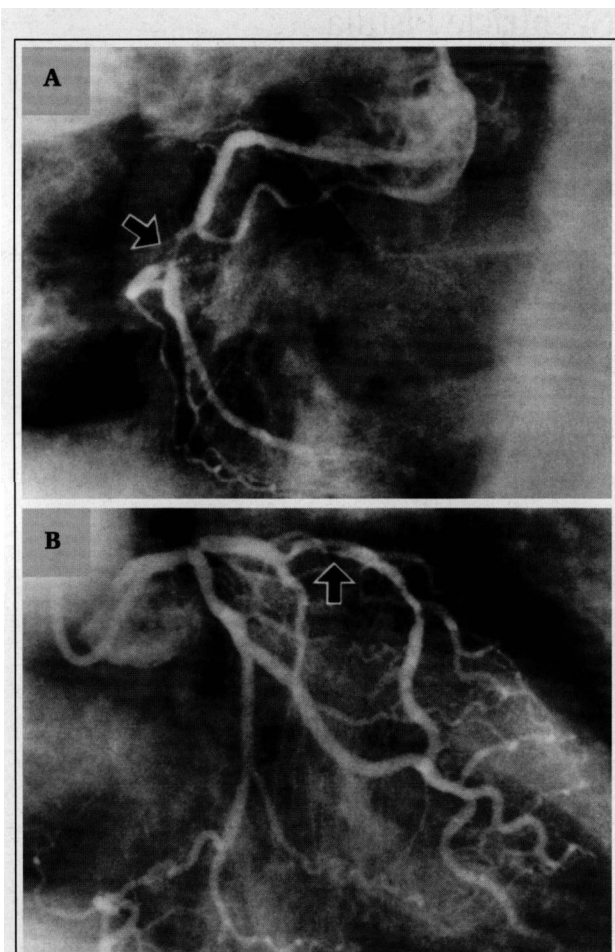


Figure 1. Right (A) and left (B) coronary angiograms prior to the fistula formation. **A.** Left anterior oblique view. A 99% stenosis (arrow) is present in the mid portion of the right coronary artery. **B.** Right anterior oblique view. A severe stenosis (arrow) is present in the proximal left anterior descending coronary artery just distal to the first septal branch.

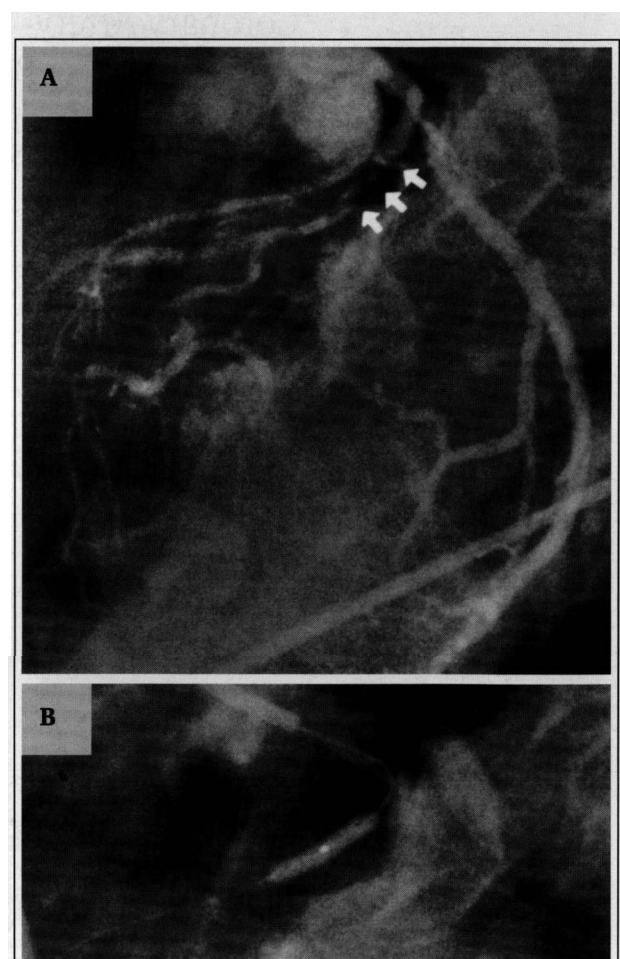


Figure 2. Emergent coronary angiograms (cranial left anterior oblique view). **A.** A stenosis with a filling defect caused by thrombus in the proximal left anterior descending coronary artery (arrows). **B.** The stenotic lesion was dilated with a 3.0-mm balloon.

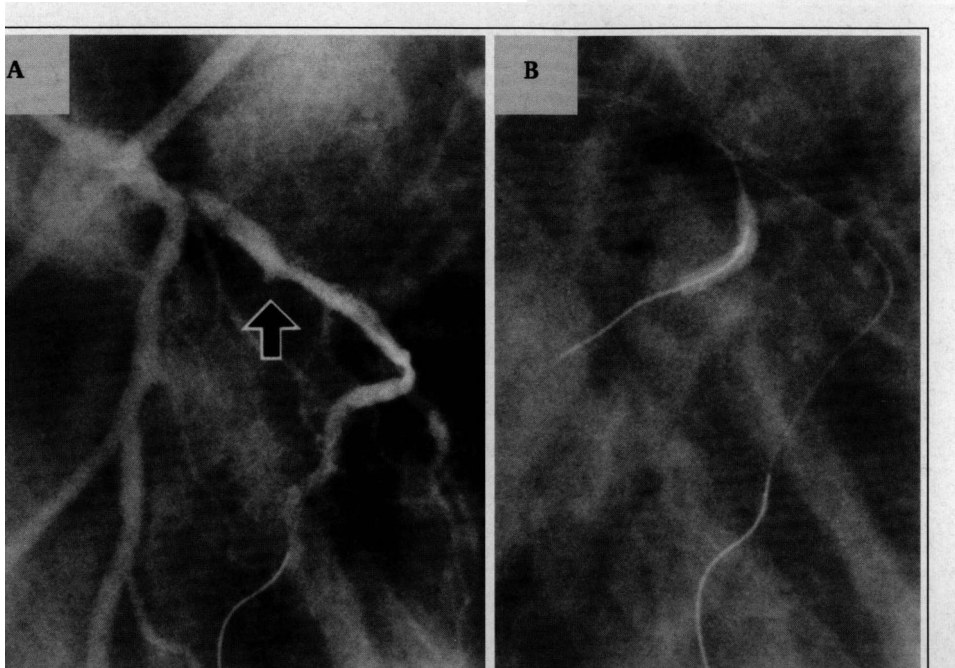


Figure 3. Left coronary angiograms before (**A**) and during (**B**) balloon angioplasty of the first septal branch (caudal right anterior oblique view). **A.** Although the stenosis of the left anterior descending coronary artery was dilated, a part of the thrombus occluded the major septal branch (arrow). **B.** Another guide wire was advanced into the septal branch and the branch was dilated with a 2.5-mm balloon at 5 atm.

left circumflex coronary artery (LCx). Emergent coronary balloon angioplasty of the infarct-related LCx lesion was successfully performed. Because of repeated restenosis of the LCx lesion, the patient underwent balloon angioplasty twice in the following 2 years. During this period, angioplasty of the RCA lesion was attempted, but we were unable to navigate the tight lesion with the balloon catheter. Good collateral blood flow from both the LAD and the LCx protected the inferior wall from myocardial ischemia. The patient had hypercholesterolemia and diabetes mellitus, which were well controlled, and he has been free from symptoms for the past 4 years.

Physical examination at admission revealed a heart rate of 66 beats per minute and a blood pressure of 126/76 mm Hg. An electrocardiogram showed normal sinus rhythm with a prominent R wave in lead V_1 and inverted T wave in leads I and aVL. Although results of a treadmill exercise test were negative, results of exercise thallium myocardial scintigraphy revealed anteroapical and inferior wall ischemia. Coronary angiography

revealed a 90% stenosis of the mid portion of the RCA (Figure 1A) and a patent LCx. A severe stenosis of the proximal LAD was noted just distal to the first septal branch (Figure 1B). Collateral blood flow from the LCx to the RCA was also demonstrated. Based on these findings, staged angioplasty for the RCA and the LAD lesion was scheduled.

During hospitalization, unstable angina developed refractory to intravenous nitroglycerin and heparin with ST segment depression in leads V_2 – V_5 . Emergent coronary angiography revealed progression of the LAD lesion to 99% stenosis with associated thrombus formation (Figure 2A). Although the lesion was successfully dilated with a 3.0-mm balloon catheter (Figure 2B), part of the thrombus was displaced into the first septal branch and occluded the artery (Figure 3A). Intractable chest pain with ST segment elevation in leads V_1 – V_4 developed. To recanalize the septal branch, a floppy guide wire was used to advance a 2.5-mm balloon catheter into the septal branch, and the balloon was inflated twice at 5 atm

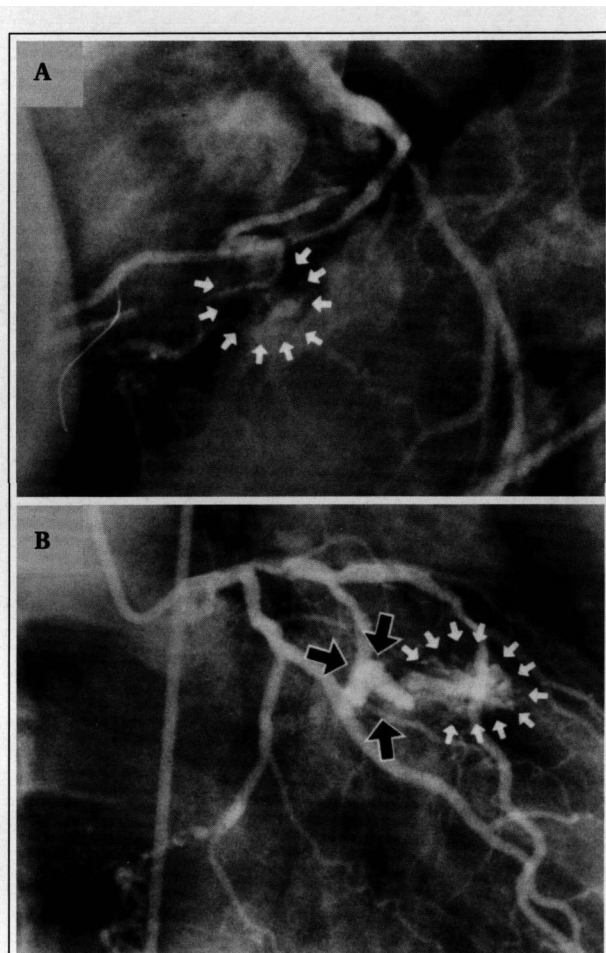


Figure 4. Left coronary angiograms revealing the coronary-left ventricular fistula. **A.** Cranial left anterior oblique view. Although the septal branch is recanalized, extravasation of contrast material into the septal wall occurs (white arrows). **B.** Right anterior oblique view. A pseudoaneurysm of the septal branch is present (black arrows) and contrast material poured into the left ventricle (white arrows). A 3.0-mm AVE gfx™ stent was deployed into the proximal left anterior descending coronary artery without closing the septal branch.

(Figure 3B). The chest pain and the ST segment elevation disappeared after the second balloon inflation. However, coronary angiography revealed extravasation of contrast material into the septal wall as well as into the left ventricular cavity, indicating a LAD-left ventricular fistula (Figure 4A). Pulmonary arterial pressure, measured with a Swan-Ganz catheter, was normal, and no step-up in the oxygen saturation was noted in any right-sided chambers.

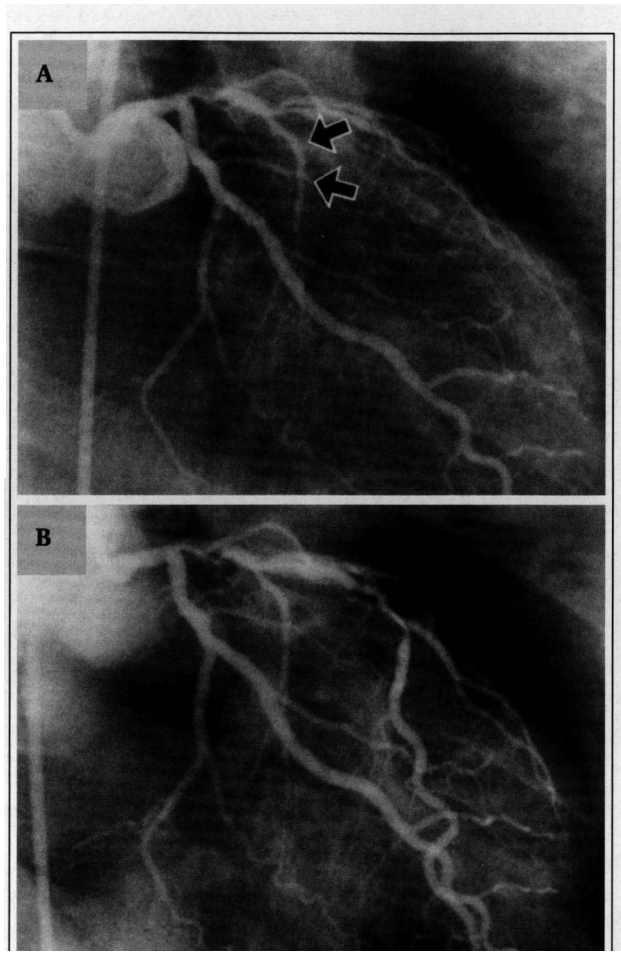


Figure 5. Left coronary angiograms obtained 5 months later (right anterior oblique view). **A.** Both the pseudoaneurysm and the fistula communicating with the left ventricle are no longer present and the first septal branch is patent (arrows). In-stent restenosis of the left anterior descending coronary artery just distal to the septal branch is present. **B.** The restenosis was dilated with a 3.0-mm balloon at 18 atm.

Because the patient's condition was stable for 30 minutes, further interventions to treat the coronary fistula were not conducted. A 3.0-mm AVE gfx™ stent (Arterial Vascular Engineering, Santa Rosa, CA) was deployed in the recoiled LAD lesion without closing the septal branch (Figure 4B). Despite sustained ST segment elevation in lead V₂ and an increase in the serum creatine kinase activity to 469 IU/L, no new Q waves developed on the electrocardiogram. Echocardi-

graphy demonstrated no left ventricular wall motion abnormalities. Coronary angiography 2 weeks later showed a patent LAD and a persistent LAD–left ventricular fistula. The RCA lesion was successfully dilated with a 3.0-mm Palmaz-Schatz stent. Exercise thallium myocardial scintigraphy showed no evidence of myocardial ischemia. The patient had no further symptoms. Coronary angiography performed 5 months later revealed complete closure of the fistula and a patent first septal branch (Figure 5A). Because in-stent restenosis was noted in the LAD, repeat angioplasty with a 3.0-mm balloon was performed with a favorable outcome (Figure 5B).

Discussion

The incidence of coronary artery perforation during balloon angioplasty is fairly low, and hemorrhage into the cardiac cavity through a perforation is exceedingly rare.¹⁻³ Ellis et al³ classified coronary perforation as three types: type I, extraluminal crater without extravasation; type II, pericardial or myocardial blush without contrast extravasation; and type III, extravasation through a frank perforation. Coronary perforation into an anatomic chamber or into the coronary sinus was classified as a subclass of type III. Non-cavity spilling type III perforations are frequently accompanied by serious cardiac events such as cardiac tamponade.^{3,4} In contrast, cavity spilling is associated with non-fatal sequelae, including the creation of a coronary artery fistula.³

There have been only three reported cases in which coronary artery rupture formed a fistula communicating with the left or right ventricle.⁵⁻⁷ Although those cases demonstrated a perforation of the proximal or mid LAD, our patient had a perforation of the septal branch. The coronary perforation in the present case appeared to arise from an inadequate insertion of the guide wire into a small side branch of the first septal branch. The subsequent insertion and inflation of the balloon catheter caused disruption of the small side branch, resulting in hemorrhage into the septal wall and communication with the left ventricular cavity. Serious complications did not occur in our patient because the fistula was a left-to-left shunt present mainly during diastole. Furthermore, coronary steal was not noted during exercise testing.

Iannone and Iannone⁷ first described a coronary artery–left ventricular fistula complicating balloon angioplasty. Although they demonstrated a dissection of the LAD that communicated with the left ventricle, the long-term outcome of the fistula was not mentioned. In our case, however, the fistula was found to have spontaneously closed within 5 months. Therefore, we conclude that emergent surgical treatment is not necessary for an iatrogenic coronary artery–left ventricular fistula if distal coronary flow is preserved.

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