A Case of Variant Angina and Myocardial Infarction 6 Months after Successful Bypass Surgery

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SUMMARY
A 61-year-old man with variant angina underwent bypass surgery to the left anterior descending artery (LAD) which had a 90% narrowing in the proximal segment. The postoperative course was favorable, but 6 months after surgery, the calcium antagonist, diltiazem, with which the patient had been continuously treated since surgery, was stopped because of hepatitis. Immediately after discontinuation of the calcium antagonist, the patient had an acute anterior myocardial infarction. An angiogram demonstrated a patent graft and an anteroapical infarction.

The infarction is thought to have been caused by a severe, prolonged spasm of the LAD distal to the graft or diffuse spasm of the LAD throughout its entire length.

Thus, after bypass surgery calcium antagonists should be given continuously to patients with variant forms of angina pectoris.

Additional Indexing Words:
Calcium antagonist    Coronary spasm

RELATIVELY unfavorable results of bypass surgery have been reported in patients with variant angina.1)–3) Graft occlusion, perioperative myocardial infarction and persistence of angina are more frequent in patients with variant angina than in those with effort angina. In some cases with persistent angina, postoperative angiograms reveal patent grafts.2)–4)

The present report describes an interesting case with variant angina who had a myocardial infarction 6 months after successful graft surgery.

CASE REPORT
A 61-year-old man, a known hypertensive, noticed precordial squeezing on effort in November 1980. Since then, the symptom had occurred several
times a month, predominantly on effort and occasionally during sleep. These attacks worsened in the beginning of February 1981, when they developed on very mild effort early in the morning. He was admitted for evaluation in February 1981.

On physical examination, the blood pressure was 190/100 mmHg and the arterial pulse was regular. The heart was not enlarged. There were no significant cardiac murmurs, but a fourth sound was clearly audible. Electrocardiograms were normal when he was free of chest discomfort. An electrocardiogram recorded during a spontaneous attack showed marked ST
Fig. 2. Angiogram done in February 21, 1981. A, The left coronary arteriogram, before intervention, shows a 99% narrowing in the LAD proximal to the 1st septal perforator. A delay in filling is observed in the LAD. B, During an episode of chest pain induced by atrial pacing, the LAD is completely occluded. C, After administration of nitroglycerin, the narrowing of the LAD is 90%. No delay of filling is seen. D, The right coronary artery has no significant lesions. E and F, The left ventricle shows normal contraction.

elevation in leads I, aVL and V_1-5. ST elevation was also observed in the same leads during an attack provoked by an exercise test (Fig. 1).

Routine laboratory determinations, including serum cholesterol, triglycerides and uric acid, and glucose tolerance test were normal.

The first coronary arteriography was performed on February 21, 1981 using Sones’ method. Before intervention, the proximal segment of the left anterior descending artery (LAD) showed a 99% narrowing (Fig. 2A). Atrial pacing was performed at a rate of 100 per minute. About 30 sec after beginning pacing, ST elevation in monitor lead V_5 and a sensation of mild chest squeezing developed. The pacing was stopped but the chest squeezing and ST elevation did not disappear until 0.3 mg of nitroglycerin was administered sublingually. During this attack, the 99% narrowing in the LAD became a complete occlusion (Fig. 2B) and, after sublingual nitroglycerin, the narrowing was about 90% of the luminal diameter (Fig. 2C). The right coronary artery (RCA) and the left circumflex artery (CX) were normal (Fig. 2A–D).
Fig. 3. Electrocardiogram 4 weeks after bypass surgery. The T wave is inverted in leads V₅ and V₆ but no significant changes in the QRS complex are present.
Fig. 4. Angiogram 4 weeks after the surgery. A, The saphenous vein graft is complete and the entire length of the LAD is opacified. The proximal 90% narrowing is also visible with retrograde filling, indicating no change in the narrowing compared with the findings prior to surgery. B and C, The left ventricle is normal, showing no changes as compared with Fig. 2.

Left ventriculography, performed after coronary arteriography, showed vigorous and symmetrical contraction of all segments (Fig. 2E and 2F).

Although it was thought that his symptoms might be almost completely controlled with medication including calcium antagonists and nitrates, it was also considered that, if the 90% narrowing of his extraordinarily long LAD progressed to a complete occlusion, a large potentially fatal anterior infarction might arise. Thus, it was recommended that he underwent aorto-coronary bypass surgery.

The patient had a saphenous vein graft to the LAD on March 8, 1981. His postoperative course was uneventful. Postoperatively he was given diltiazem, a calcium antagonist at a dose of 240 mg a day. A second arteriogram performed on April 8, 1981 demonstrated a patent graft and a normal left ventricle (Fig. 4). All native coronary arteries were unchanged as compared with the preoperative angiogram, and the severely narrowed LAD, shown by retrograde opacification in Fig. 4A, was not occluded. The electrocardiogram showed T wave abnormalities which were considered to be a result of the surgery (Fig. 3).

Since the operation, he had been asymptomatic until September 1981, when he displayed anorexia and jaundice. He was diagnosed as having serum hepatitis based on elevated serum transaminase and bilirubin. The
Fig. 5. Electrocardiogram in February 1982. A QS pattern and inversion of the T wave are seen in leads V₃-V₅. The R wave in lead V₆ is decreased. Inferior leads show a QS pattern.
hepatitis had a rather prolonged course, so the family doctor stopped all drugs including diltiazem. In a few days, the patient experienced severe chest pain and was admitted to a hospital. He was diagnosed as having had an acute anterior myocardial infarction because of newly developed Q waves in the precordial leads and elevated serum creatine phosphokinase. There was no cardiogenic shock, congestive heart failure, or serious arrhythmias. The recovery from hepatitis was complete as of January 1982.

The patient felt occasional mild chest squeezing again as of December 1981. The third coronary arteriography was performed on February 5, 1982. The electrocardiogram recorded in February 1982 showed a marked left axis deviation and QS pattern in the inferior leads and in leads V₃ through V₅ (Fig. 5). The graft remained patent and the LAD was occluded at the site of the previous severe narrowing. RCA and CX showed no changes. Left ventriculography showed systolic outward movement (dyskinesis) in the anteroapical segment with a filling defect in the apex, which was thought
to be a mural thrombus. Also found was an absence of systolic movement (akinesia) in the apical half of the inferior segment, which was supplied by the apical twig of the very long LAD instead of the relatively small RCA (Fig. 6). Thus, it was considered that the QS pattern in the inferior leads, as seen in Fig. 5, was based on this inferior extension of the anteroseptal infarction.

After the third angiogram diltiazem was again begun, and the patient has been asymptomatic up to the present on a diltiazem dosage of 240 mg a day.

**DISCUSSION**

There have been many reports on the value of aortocoronary bypass in cases with a variant form of angina.\(^1\)\(^–\)\(^7\) Except for a few reports,\(^6\)\(^,\)\(^7\) investigators have considered that the postoperative course is not as good as in cases with classic angina.\(^1\)\(^–\)\(^3\) Thus, perioperative infarction, perioperative death, and recurrence of chest pain are more frequent following bypass surgery in these cases than in those with effort angina. In these reports, postoperative angiograms showed occlusion of the graft and/or native coronary arteries.\(^1\)\(^,\)\(^2\)

However, some authors reported that postoperative angina occasionally occurs in the presence of patent grafts.\(^3\)\(^–\)\(^5\) Among them, Bertrand et al\(^5\) reported 2 cases with postoperative spontaneous angina whose angiograms demonstrated patent grafts and coronary spasm distal to the site of graft anastomosis.

The present case experienced a typical myocardial infarction 6 months after successful surgery and persistence of chest pain despite the patent graft. Although the third arteriogram, obtained after the infarction, showed closure of the native LAD at the site of the previous severe narrowing, this occlusion could not have been the cause of the infarction because blood flow to the anterior wall was thought to be sufficient through the widely patent graft. Since the patient had been completely free of chest pain since the graft surgery until discontinuation of the calcium antagonist, it would be reasonable to conclude that, after the discontinuation of diltiazem, a severe spasm occurred in the LAD and thus caused the anterior infarction. The spasm might have occurred distal to the graft, as in the cases reported by Bertrand et al, or, over the entire length of the LAD, because a spasm at the site of the 90% narrowing by itself could not provoke ischemia or infarction in the presence of the patent graft. On the other hand, it is also possible that the spasm occurred in the graft itself as well as in the LAD (either in the proximal segment or in the distal segment) resulting in severe ischemia of the anterior wall and myocardial infarction. Many authors have reported cases of myocardial infarc-
tion without significant coronary narrowing, and an extraordinarily long spasm was thought to be the cause of the infarction in these cases.8)–13)

Thus, it is suggested that, in the present case, a spasm of long duration provoked by the sudden discontinuation of diltiazem, caused the infarction.

Thus, as Endo et al2) indicated, calcium antagonists must be administered continuously in cases with coronary spasm even if the bypass surgery is deemed successful.

**References**