CASE REPORT

Coronary Artery and Graft Spasm after Off-pump Coronary Artery Bypass Grafting

We report a case of a 52-year-old man with severe coronary artery and graft spasm after triple-vessel off-pump coronary artery bypass grafting. Emergent coronary angiography was performed to identify the location and severity of the spasm. Intracoronary injections of several vasodilators failed to relieve the spasm. Observational treatments including intra-aortic balloon pump and inotropic drugs to increase coronary flow were performed until the spasm resolved. The patient recovered and was discharged. A follow-up coronary angiography revealed patent native coronary artery and bypass grafts without evidence of residual spasm. (Jpn J Thorac Cardiovasc Surg 2005; 53: 109–113)

Key words: coronary artery spasm, coronary artery bypass grafting

Toshihiro Fukui, MD, Shuichiro Takanashi, MD, and Yasuyuki Hosoda, MD.

Perioperative coronary artery spasm is known to cause myocardial infarction, severe arrhythmia, cardiac arrest, and death.[1] Hence it has been suggested that early diagnosis, which includes catheter angiography coupled with aggressive management, are necessary in this setting.[2,3] Intracoronary and intravenous vasodilator infusion have been recognized to reduce coronary artery spasm immediately.

We describe here a patient undergoing off-pump coronary artery bypass grafting (OPCAB) who experienced a massive coronary artery and graft spasm that could not be resolved with intracoronary vasodilator infusion. The spasm improved with intra-aortic balloon pumping (IABP), positive inotropic support, and intravenous vasodilator infusion.

Case

A 52-year-old man was admitted to our institution with unstable angina pectoris. He had a history of an inferior myocardial infarction in 1996. One month before admission, he experienced angina more frequently and was scheduled to undergo cardiac catheterization. Coronary angiography revealed a 90% left main trunk stenosis and a 75% stenosis of the posterolateral branch of the right coronary artery. During left coronary artery angiography, he developed chest pain with ST segment depression in the precordial leads on electrocardiogram (ECG). These events were resolved by an intracoronary injection of nitroglycerin, and coronary artery spasm was not observed at that time. Because of the high-grade lesion of left main trunk and unstable anginal symptoms, we performed emergency coronary artery bypass grafting. We used an off-pump technique and total arterial coronary revascularization for the isolated coronary bypass surgery.

After median sternotomy, bilateral internal thoracic arteries were dissected as *in-situ* grafts and the radial artery graft was harvested from his left forearm. All arterial grafts were harvested in skeletonized fashion with an ultrasonic scalpel (Harmonic Scalpel, Ethicon Endosurgery, Cincinnati, OH, USA). A commercially available heart positioner and stabilizer were utilized to support the cardiac position and to expose target vessels. A bloodless field was obtained with a CO_2 blower and snaring silastic loops proximal to the anastomotic sites. The left internal thoracic artery (ITA) was anastomosed to the left anterior descending artery, and the radial artery was sequentially anastomosed to the obtuse marginal branch of the circumflex artery and the posterolateral branch of the right coronary artery. The proximal end of the radial artery graft was anastomosed

From Department of Cardiovascular Surgery, Shin-Tokyo Hospital, Chiba, Japan.

Received for publication June 16, 2004.

Accepted for publication November 4, 2004.

Address for reprints: Toshihiro Fukui, MD, Department of Cardiovascular Surgery, Shin-Tokyo Hospital, 473-1 Nemoto, Matsudo, Chiba 271-0077, Japan.

to the distal end of the right ITA, as a lengthened graft. Intravenous nitroglycerin and nicorandil were administered during the operative period and no ST-T changes were observed. The patient's temperature was kept no lower than 36°C during the operation. After complete hemostasis was achieved, the patient was returned to the intensive care unit (ICU).

Immediately after the operation, the patient was hemodynamically stable on low-dose dopamine (3 μ/kg/min) and norepinephrine (0.05 μ/kg/min). Intravenous nitroglycerin and nicorandil were injected continuously after the operation. Blood pressure was 100/50 mmHg, cardiac output was 4.9 l/min, pulmonary artery pressure was 23/10 mmHg, and the ECG was normal. Blood gas analysis revealed that pH was 7.38, pO_2 was 143 mmHg and pCO_2 was 37 mmHg with mechanical ventilator support. The patient was gradually awakened for weaning from mechanical ventilator about 1 hour after the operation. At that time, the patient coughed heavily and blood pressure fell to 50/30 mmHg and pulmonary artery pressure rose to 36/14 mmHg. Sedation with propofol was initiated because of his unstable hemodynamic condition. Blood pressure reached only 80/40 mmHg despite several doses of inotropic agents (8 μ/kg/min of dopamine and 0.1 μ/kg/min of norepinephrine) after this event. ECG revealed ST-segment depressions in I, aVL, V1–4 leads and ST-segment elevations in II, III, aVF leads. This hemodynamic deterioration and ECG changes suggested coronary artery spasm; therefore, we administered diltiazem (0.5 μ/kg/min). The patient's condition did not improve and he was transferred for angiographic examination. Coronary angiography demonstrated a severe diffuse spasm of the native coronary artery and bypassed grafts (Fig. 1). Several vasodilator agents (nicorandil, verapamil, papaverine, and nitroglycerin) were injected into the bilateral ITA and native coronary artery. Spasm of all vessels did not improve. An IABP

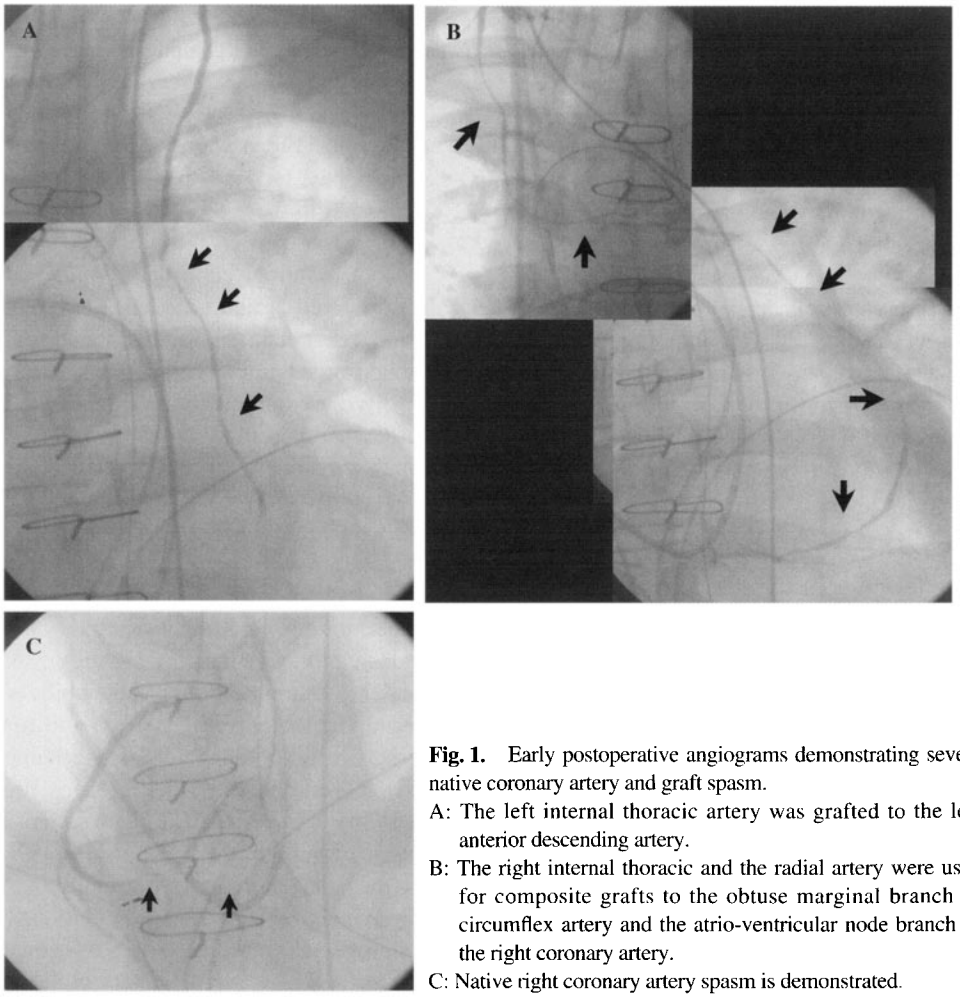

Fig. 1. Early postoperative angiograms demonstrating severe native coronary artery and graft spasm.
A: The left internal thoracic artery was grafted to the left anterior descending artery.
B: The right internal thoracic and the radial artery were used for composite grafts to the obtuse marginal branch of circumflex artery and the atrio-ventricular node branch of the right coronary artery.
C: Native right coronary artery spasm is demonstrated.

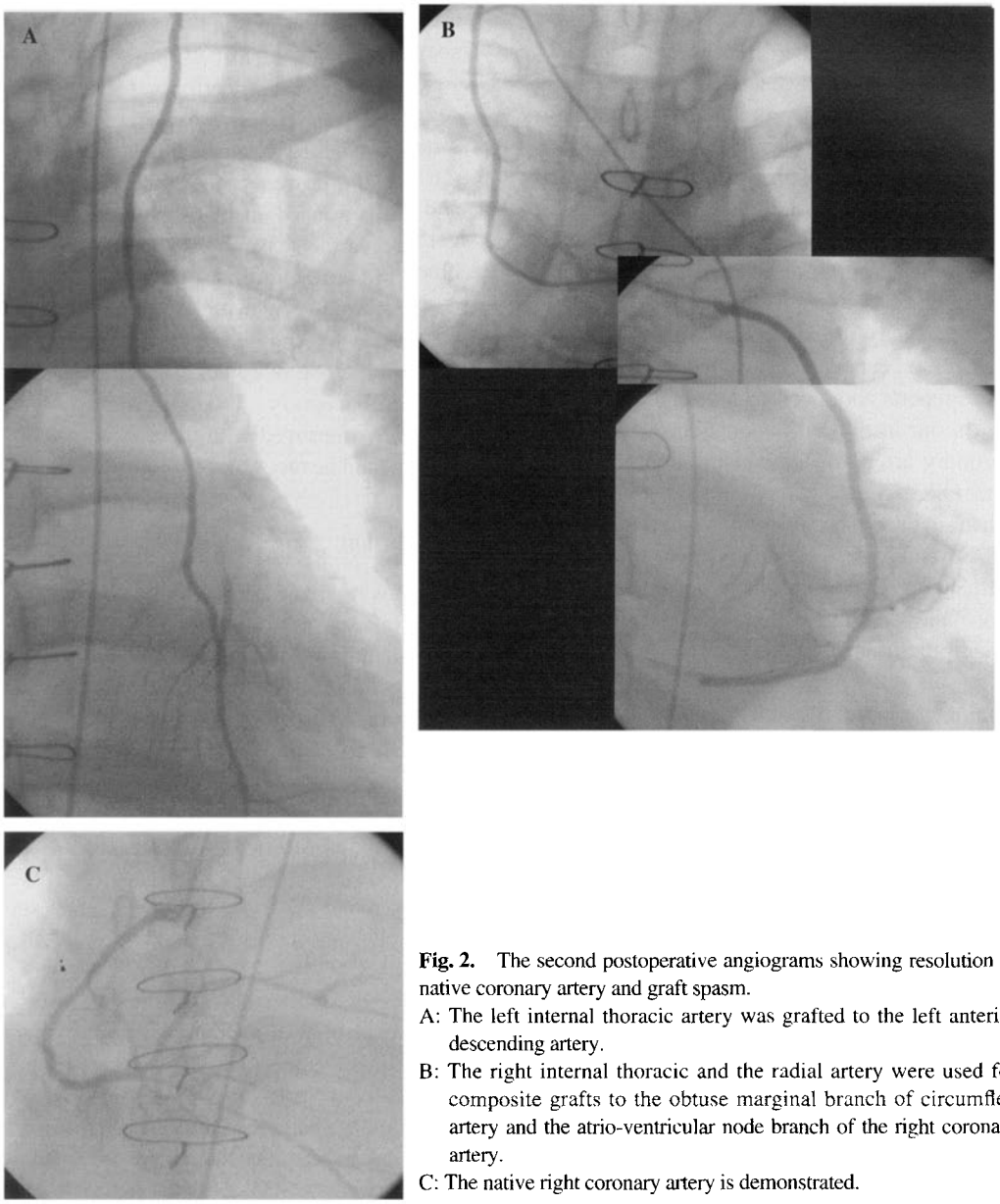

Fig. 2. The second postoperative angiograms showing resolution of native coronary artery and graft spasm.
A: The left internal thoracic artery was grafted to the left anterior descending artery.
B: The right internal thoracic and the radial artery were used for composite grafts to the obtuse marginal branch of circumflex artery and the atrio-ventricular node branch of the right coronary artery.
C: The native right coronary artery is demonstrated.

was inserted into the right femoral artery and he returned to the ICU. We decided to observe these conditions until the spasm resolved. Blood pressure was maintained at 90/60 mmHg with adrenergic drugs (epinephrine at 0.1 μ/kg/min, norepinephrine at 0.3 μ/kg/min, and dopamine at 5 μ/kg/min), vasodilator drugs (nitroglycerin, nicorandil, and diltiazem) and IABP. The serum concentration of creatine phosphokinase isoenzyme (CK-MB) elevated to 935 U/l. Because hemodynamic improvement was gradually seen, adrenergic drugs were tapered over 4 days with the exception of dopamine at 3 μ/kg/min. IABP was removed on the fifth postoperative day (POD) and his cardiac output was 5.3 l/min. Sedation was stopped and the patient was easily weaned from mechanical ventilation at that time. Oral nitroglycerin, nicorandil, and diltiazem were started and intravenous vasodilator drugs were discontinued gradually. His subsequent course was uneventful. Coronary arteriography performed again on POD 14 demonstrated that native coronary artery and bypass grafts were patent without evidence of residual spasm (Fig. 2). Left ventricular function was preserved; the ejection fraction was 64% at the time of discharge.

Discussion

Coronary artery spasm is now recognized to potentially occur in the early postoperative period that may lead to sudden circulatory collapse or death.[1] Hence, immediate diagnosis and aggressive treatment must be performed in these situations.[2,3] The incidence of perioperative coronary artery spasm causing hemodynamic collapse after coronary artery bypass grafting using cardiopulmonary bypass was reported to be approximately 1%.[4] But the incidence of coronary artery spasm after OPCAB is unknown. To the best of our knowledge, there were only two case reports about coronary artery spasm after OPCAB.[5,6] In our institution, this patient was the first case of coronary artery spasm causing hemodynamic collapse after OPCAB.

The mechanism of perioperative coronary artery spasm is not defined. Several causes of coronary artery spasm after coronary artery bypass surgery have been considered, such as coronary arterial wall trauma secondary to surgical manipulation, alpha-adrenergic stimulation, hypothermia, alkalosis, local increase in potassium levels, histamine release, hypomagnesemia, or release of vasospastic factors by platelets.[1,7] In our patient, a systematic factor was likely to have caused the vascular spasms, because spasms occurred in target vessels and also non-target vessels (posterior descending artery). Furthermore, spasms occurred not only in the native coronary artery, but also in the bypass grafts. Therefore, the most probable etiology of coronary artery and graft spasm in our patient was increased alpha-adrenergic stimulation. Alpha-adrenergic stimulation may have been provoked by heavy coughing under usage of low-dose dopamine (3 μ/kg/min) and norepinephrine (0.05 μ/kg/min). This stimulation may have increased the arterial wall tone and induced vascular spasm. Also, a sympathetic nervous system response post awakening from anesthesia may have induced vascular spasm.[5] Intraoperative use of cathecolamine and other vasoconstrictive agents may induce coronary artery spasm even in the case of OPCAB. Intra- and postoperative use of dopamine may not be recommended in a patient who has been diagnosed as having coronary artery spasm preoperatively like our patient. We believe that prudent use of vasoconstrictive agents and a high dose of calcium channel blocker should be administered in a patient with coronary artery spasm.

Treatment of this life-threatening complication has been well described.[1-10] Intravenous infusion of nitroglycerin and calcium channel blockers simply and quickly resolve spasms. However, in patients refractory to this treatment, coronary angiography is necessary to ascertain the location and severity of the spasm. If the spastic lesion is identified, intracoronary injection of vasodilator should be performed selectively. There are some reports that intracoronary injection of nitroglycerin and calcium channel blocker is effective.[5,6,10] In our patient, however, the intravenous administration and intracoronary injection of various vasodilators did not resolve the spasm. We determined that raising the blood pressure with inotropic drugs and increasing the coronary blood flow with IABP were the only method to resolve the spasm in our patient. Lemmer et al.[1] recommended the use of epinephrine if inotoropic support is deemed necessary. Indeed, the hemodynamic status gradually improved with these treatments over a 5-day interval and he recovered.

Conclusion

In this report we described a case of coronary artery and graft spasm after coronary artery bypass grafting. Treatment with intracoronary injection of vasodilators failed to resolve the spasm however raising the blood pressure with inotropic drugs and increasing the coronary blood flow with IABP effectively stabilized our patient.

REFERENCES

1. Lemmer JH Jr, Kirsh MM. Coronary artery spasm following coronary artery surgery. Ann Thorac Surg 1988; 46: 108–15.
2. Zaiac M, Renzulli A, Hilton CJ. Coronary artery spasm following coronary artery bypass grafting. Eur J Cardiothorac Surg 1990; 4: 109–11.
3. Paterson HS, Jones MW, Baird DK, Hughes CF. Lethal postoperative coronary artery spasm. Ann Thorac Surg 1998; 65: 1571–3.
4. Buxton AE, Goldberg S, Harken A, Hirshfield J Jr, Kastor JA. Coronary-artery spasm immediately after myocardial revascularization: recognition and management. N Engl J Med 1981; 304: 1249–53.
5. Bittner HB. Coronary artery spasm and ventricular fibrillation after off-pump coronary surgery. Ann Thorac Surg 2002; 73: 297–300.
6. Trimboli S, Oppido G, Santini F, Mazzucco A. Coronary artery spasm after off-pump coronary artery bypass grafting. Eur J Cardiothorac Surg 2003; 24: 830–3.
7. Sarabu MR, McClung JA, Fass A, Reed GE. Early postoperative spasm in left internal mammary artery bypass grafts. Ann Thorac Surg 1987; 44: 199–200.

8. Kopf GS, Riba A, Zito R. Intraoperative use of nifedipine for hemodynamic collapse due to coronary artery spasm following myocardial revascularization. Ann Thorac Surg 1982; 34: 457–60.
9. Cohen DJ, Foley RW, Ryan JM. Intraoperative coronary artery spasm successfully treated with nitroglycerin and nifedipine. Ann Thorac Surg 1983; 36: 97–100.
10. Caputo M, Nicolini F, Franciosi G, Gallotti R. Coronary artery spasm after coronary artery bypass grafting. Eur J Cardiothorac Surg 1999; 15: 545–8.