CASE REPORT

Cardiopulmonary Arrest Caused by Coronary Spasm after Coronary Vasodilator Withdrawal during the Peri-operative Period of Gastrectomy

Manabu Kurabayashi¹, Kaoru Okishige¹, Mitsutoshi Asano¹, Hidetoshi Suzuki¹, Tsukasa Shimura¹, Shinsuke Iwai¹, Nobutaka Kato¹, Kensuke Ihara¹, Hideshi Aoyagi¹ and Mitsuaki Isobe²

Abstract

Calcium antagonists, nicorandil and long-acting nitrates are highly effective for preventing coronary spasm. The withdrawal of coronary vasodilators, especially calcium antagonists, is risky in cases of vasospastic angina. We herein present a case of cardiopulmonary arrest that occurred due to coronary spasm triggered by the discontinuation of coronary vasodilators during the peri-operative period of gastrectomy. Vasospastic angina patients who are not able to take oral coronary vasodilators in the peri-operative period should be maintained on a parenteral vasodilator until they are able to take them orally. Physicians should also be aware of the possible development of nitrate tolerance in patients on prolonged nitrate therapy.

Key words: vasospastic angina, cardiopulmonary arrest, coronary vasodilator withdrawal, peri-operative period

(Intern Med 52: 81-84, 2013)
(DOI: 10.2169/internalmedicine.52.8918)

Introduction

Calcium antagonists are highly effective for preventing coronary spasm, and are considered the drugs of first choice for the treatment of vasospastic angina (1, 2). Nicorandil and long-acting nitrates are similarly effective as such treatment. However, these drugs are usually prescribed orally when used for the purpose of prevention of coronary spasm. This may lead to problems when oral medications are discontinued, such as during the postoperative period following digestive tract surgery. We herein present a case of cardiopulmonary arrest that occurred due to coronary spasm that was triggered by the discontinuation of oral coronary vasodilators during the peri-operative period of gastrectomy.

Case Report

A 66-year-old male with a medical history of vasospastic angina was admitted to our institute for the purpose of surgical treatment of gastric cancer. He had experienced an episode of chest oppression and syncope about 15 years prior to this presentation, and had undergone coronary angiography at another hospital, which revealed no significant stenosis, but demonstrated spasm of the left coronary artery after intracoronary injection of acetylcholine. Because the spasm of the left anterior descending artery and chest pain did not resolve spontaneously within five minutes and nitroglycerin was injected into the left coronary artery, acetylcholine was not injected into the right coronary artery. His syncope was considered to have been provoked by a lethal arrhythmia due to coronary spasm, and was diagnosed as vasospastic angina. He was treated with amlodipine at a dose of 2.5 mg

¹Division of Cardiology, Yokohama City Minato Red Cross Hospital, Japan and ²Department of Cardiovascular Medicine, Tokyo Medical and Dental University, Japan

Received for publication September 3, 2012; Accepted for publication September 30, 2012

Correspondence to Dr. Manabu Kurabayashi, kurabayashi.card@yokohama.jrc.or.jp
once daily, nicorandil at 5 mg three times daily, and a transdermal nitroglycerine patch (25 mg) once daily (before sleep). He had remained free from angina at the time of his admission for surgery.

The patient took his last dose of amlodipine, nicorandil and nitroglycerin the morning of the operation. He underwent a total gastrectomy under general anesthesia. From the time of the operation until postoperative day (POD) 3, intravenous nicorandil was administered, and no adverse events were noted. On POD3, the intravenous nicorandil was discontinued, and the use of the transdermal nitroglycerine patch (25 mg, before sleep) was restarted by the surgeon. A stomach X-ray examination on POD7 revealed an anastomotic leak preventing the restarting of any oral medications.

At 9 AM on the morning of POD10, the patient complained of cold sweats and dizziness. Ten minutes later, he collapsed, and his electrocardiograph monitor showed ventricular fibrillation. Cardiopulmonary resuscitation was performed immediately, including electrical defibrillation, endotracheal intubation, four intravenous injections of adrenaline (1 mg every 3-5 minutes) and intravenous injection of 300 mg of amiodarone. However, a return of spontaneous circulation was not achieved despite twenty minutes of resuscitation effort (Fig. 1). The patient was then put promptly on a percutaneous cardiopulmonary support system thirty minutes after cardiopulmonary arrest. After the application of the percutaneous cardiopulmonary support system, hemodynamic stability was regained followed by a return of spontaneous circulation. After resuscitation, we performed emergency cardiac catheterization, which showed no significant stenosis of the coronary artery (Fig. 2) and near normal left ventricular contraction. We thus ruled out acute coronary syndrome as the cause of the patient’s cardiopulmonary arrest.

After the patient was moved from the cardiac catheterization laboratory to the intensive care unit, his electrocardiogram recorded two hours after cardiopulmonary arrest showed ST segment elevation in leads II, III, aVF, and V4-6 (Fig. 3a). We administered intravenous isosorbide dinitrate, which promptly relieved the ST segment elevation (Fig. 3b). We therefore attributed the cause of the cardiopulmonary arrest to coronary spasm, and administered intravenous calcium antagonist, nicorandil and nitroglycerine. Over the next week, we changed the delivery method for the coronary vasodilators from intravenous to oral, while monitoring him for signs of aggravation of coronary spasm. After we restarted the same coronary vasodilators as had been used before admission, the patient was free of angina. We could not perform an acetylcholine provocation test to evaluate the efficacy of his coronary vasodilators because of the presence of renal dysfunction with a creatinine of 1.5 mg/dL. However, we performed electrocardiographic monitoring and Holter electrocardiography, which showed no significant ST-T segment abnormalities. We did not replace amlodipine with a stronger calcium antagonist because the patient had a history of hypotension as a side effect of a stronger calcium antagonist. At the time of discharge, the patient retained a slight neurological deficit, but he was free from any angina symptoms.

Discussion

Patients with vasospastic angina who have had syncope are at high risk, because their symptoms may be due to atrioventricular block, ventricular arrhythmia or asystole (3). Kishida et al. reported that 12.5% (30 of 240 patients) of vasospastic angina patients had syncope during vasospastic angina attacks, and their syncope was associated with ST segment elevation in inferior leads and serious arrhythmia (4). There have been several case reports describing life-threatening coronary spasm caused by the sudden withdrawal of a calcium antagonist (5, 6). However, many patients may be obliged to forego their oral medicines during the postoperative periods for digestive tract surgery. Our pa-
Figure 2. Coronary angiography after cardiopulmonary resuscitation revealed no significant stenosis of the left (a) and right (b) coronary arteries.

Figure 3. An electrocardiogram in the intensive care unit showed ST segment elevation in leads II, III, aVF and V3-6 (a). After intravenous injection of isosorbide dinitrate, the ST segment elevation was promptly relieved (b).

The patient was not able to take oral coronary vasodilators for several days because of a postoperative complication, and received only a transdermal nitrate patch. Ten days after calcium antagonist withdrawal and seven days after intravenous nicorandil withdrawal, he suffered aborted cardiac sudden death likely due to vasospasm. Although the patient had remained free from vasospastic angina for several years, the abrupt discontinuation of long-term amlodipine therapy and surgical stress may have provoked his coronary spasm. Because the patient had a vasospastic angina attack despite treatment with transdermal nitroglycerine, the development of nitrate tolerance (7) after the seven-day prescription might therefore have been another cause of the coronary spasm.

Nagayoshi et al. (8) reported that the prevalence of coronary spasm was infrequent, but could be a cause of cardiac trouble during the peri-operative period. Eighteen of 77,745 consecutive non-cardiac surgery patients were diagnosed as having definite vasospastic angina, and only two patients had fatal ventricular arrhythmia due to coronary spasm in the peri-operative period. Takagi et al. (9) reported that the incidence of arrhythmic events during a spontaneous vasospastic angina attack was 7.5%. Although cardiopulmonary arrest due to coronary spasm is very rare, physicians, especially those in Japan, should keep coronary vasospasm in mind as a possible diagnosis, because the prevalence of coronary vasospasm is higher in Japanese than in Caucasians (10). For example, a multi-center study found documented coronary spasm in 6% of patients resuscitated after out-of-hospital cardiac arrest of cardiac origin, which was in contrasted that with the 3% reported in a European study (9).

In conclusion, the withdrawal of coronary vasodilators, especially calcium antagonists, is risky in cases of severe vasospastic angina. Vasospastic angina patients who are not able to take oral coronary vasodilators in the peri-operative...
period should be maintained on a parenteral vasodilator until they are able to take them orally. Physicians should also be aware of the possible development of nitrate tolerance in patients on prolonged nitrate therapy, which may also lead to coronary spasm.

The authors state that they have no Conflict of Interest (COI).

References


© 2013 The Japanese Society of Internal Medicine
http://www.naika.or.jp/imonline/index.html