Case Reports

Coronary Arterial Wall Disruption and Intramural Hematoma in a Patient With Coronary Spastic Angina

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Summary

Acute myocardial infarction is sometimes complicated in patients with coronary spastic angina. The mechanisms are known to be plaque rupture and thrombosis induced by spasm, and reduced coronary flow due to prolonged spasm. We describe the case of a 45-year-old woman with coronary spastic angina who had a complication of an acute myocardial infarction. A specimen obtained with thrombectomy was the disrupted coronary artery wall accompanied by massive intramural hemorrhage. The cause of the acute myocardial infarction was thought to be an embolism of the coronary arterial wall that was disrupted by spasm and intramural hemorrhage. (Int Heart J 2012; 53: 68-71)

Key words: Coronary spastic angina, Intramural hemorrhage, Acute myocardial infarction

A 45-year-old woman was admitted to our hospital because of a chest pain attack. She had no previous medical history. Five months before admission, she had experienced a chest pain attack at rest. An electrocardiogram (ECG) conduct-

Figure 1. A: Electrocardiogram, which was recorded during a chest pain attack 5 months before admission, showed ST segment elevation in leads II, III, aVF, and V1-6. B: Coronary angiograms conducted immediately after the attack revealed no significant stenosis.

Coronary spastic angina is also implicated in the progression of coronary atherosclerosis. The mechanisms of the progression are intimal hyperplasia due to intimal injury and intramural hemorrhage.1-3) Experimental studies have shown that intramural hemorrhages are induced at spastic sites by repetitive coronary spasms.2,3) However, it is difficult to show intramural hemorrhages in clinical settings. We describe here a patient with coronary spastic angina whose severe spasm led to intramural hemorrhage and coronary arterial wall disruption.

CASE REPORT

A 45-year-old woman was admitted to our hospital because of a chest pain attack. She had no previous medical history. Five months before admission, she had experienced a chest pain attack at rest. An electrocardiogram (ECG) conduct-

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ed during the attack showed ST segment elevation in leads II, III, aVF, and V1-6 (Figure 1A). The chest pain was relieved with sublingual administration of nitroglycerin. Coronary angiography performed immediately after the attack showed no significant stenosis (Figure 1B). We made a diagnosis of coronary spastic angina. Diltiazem was prescribed, and her adherence was good. Eight days before admission, her chest pain recurred and was relieved by 4 sublingual doses of nitroglycerin. The duration of the chest pain was 60 minutes. Five hours before admission, a chest pain attack occurred at rest and was relieved following sublingual administration of 3 doses of nitroglycerin. Two hours before admission, chest pain recurred, and it could not be relieved with sublingual administration of nitroglycerin. She was transferred to the emergency room of our hospital by ambulance. Upon arrival, the chest pain was still present. Her blood pressure was 122/68 mmHg. An electrocardiogram showed ST segment elevation in leads I, aVL, and V4-6 (Figure 2). Nitroglycerin and nicardipine were administered intravenously, but they were not effective. Emergent coronary angiography showed total occlusion at the mid segment of the large obtuse marginal artery (Figure 3A). Thrombectomy was performed, and small pieces of reddish and whitish material were obtained (Figure 4A). There were two lesions at the proximal portion of the obtuse marginal artery; one exhibited 25% stenosis, and the other exhibited 50% stenosis. Thrombolysis in Myocardial Infarction (TIMI) 3 flow was achieved (Figure 3B). Intravascular ultrasound showed focal coronary dissection and an echolucent zone in the coronary wall at the proximal site of the obtuse marginal artery (Figure 5). The coronary intervention was finished only with thrombectomy. The amount of diltiazem was increased, and nicorandil was added. Serum creatine kinase levels were increased to 1217 IU/L. On the fifth hospital day, a chest pain attack occurred at rest, and an electrocardiogram showed ST segment elevation in leads II, III, and aVF. The chest pain was relieved with sublingual nitroglycerin, intravenous nitroglycerin, and intravenous nicardipine. Because of the prolonged chest pain attack and electrocardiogram findings that were different from admission, we performed emergent coronary angiography. The coronary angiogram revealed that there were no changes in the lesions at the proximal portion of the obtuse marginal artery, and that spastic

Figure 2. Electrocardiogram, which was recorded on admission, showed ST segment elevation in leads I, aVL, and V4-6.

Figure 3. A: Coronary angiogram, which was conducted on admission, showed total occlusion at the mid segment and 25% stenosis at the proximal segment of the obtuse marginal artery. B: Coronary angiogram conducted after thrombectomy revealed two lesions at the proximal portion of the obtuse marginal artery; one exhibited 25% stenosis, and the other exhibited 50% stenosis.
lesions appeared in the posterolateral artery (Figure 5). The attack was thought to be due to a coronary spasm of the posterolateral artery. Benidipine was added. Subsequently, her hospital course was uneventful, and she was discharged on hospital day 15.

Histological examination of the specimen obtained during the thrombectomy revealed that the specimen was the disrupted coronary artery wall. The coronary artery wall was accompanied by mild thickened intima and massive intramural hemorrhage (Figures 4B, 4C). The cause of the acute myocardial infarction in our case was thought to be embolization of the coronary artery wall, which was disrupted by spasm and intramural hemorrhage.

**Discussion**

Intramural hemorrhage is one of the causes that promote progression of atherosclerosis and coronary stenosis. Intramural hemorrhage accumulates cholesterol crystals with erythrocyte fragments, foam cells, and iron deposits. Intramural hemorrhage itself is suggested to serve as an atherogenic stimulus. Intramural hemorrhage also increases intramural volume and pressure, resulting in progression of coronary stenosis.

Experimental studies have revealed the relationship between coronary spasm and intramural hemorrhage. In a pig model that had a previously provoked intimal injury by irradiation and a high cholesterol diet, repetitive severe coronary spasms of acute onset were shown to induce intramural hemorrhage. Intramural hematoma occurred mainly in the thickened intima. Coronary spasm is thought to provoke mechanical tearing of newly proliferating capillaries induced by intimal injury, leading to intramural hemorrhage. In our case, massive intramural hemorrhage was observed mainly in the media. It was different from the findings of the experiment in which intramural hemorrhage was mainly in the intima. The experimental study also showed the possibility of intramural hemorrhage from the capillaries in the media, and the case in which the hemorrhage extended from the intima to the media. In our case, massive intramural hemorrhage was not assessed because the specimen was the disrupted coronary artery wall, and not the entire vessel. Before the onset of acute myocardial infarction, the patient had frequent episodes of severe chest pain attack that required large amounts of nitroglycerin to relieve. These attacks were thought to induce intramural hemorrhage and disruption of the coronary artery wall, resulting in embolization. Intravascular ultrasound showed a defect of the coronary artery wall at the proximal site of the obtuse marginal artery, suggesting that was the site of disruption.
There are few reports that show the relationship between coronary spasms and intramural hemorrhages in clinical settings. Etsuda, et al showed intimal hemorrhage at spasm-induced sites in patients with coronary spastic angina using percutaneous coronary angioscopy. Suzuki, et al examined the coronary plaques obtained with atherectomy in patients with significant stenosis. Intimal hemorrhage was observed at the spasm-induced site. A case of coronary artery disruption due to coronary spasm and intramural hemorrhage has not been previously reported.

We did not perform an acetylcholine spasm provocation test because the diagnosis of coronary spasm was made from the patient’s clinical course, ECG findings, and coronary angiogram. It is potentially harmful to perform acetylcholine provocation testing during emergent coronary angiography in patients with acute coronary syndrome. Intense medical therapy to prevent coronary spasm is important in patients with coronary spastic angina who have severe attacks.

REFERENCES