Case Report

Transmural myocardial ischemia due to slow coronary flow

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Abstract Slow coronary flow phenomenon (SCFP) is an angiographic observation characterized by delayed distal vessel opacification in the absence of significant epicardial coronary disease. Only limited studies have been focused on the etiologies, clinical manifestations and treatment of this unique angiographic phenomenon. In our case report, we described an 85-year-old man who came with significant ST segment elevation in leads V₁-V₄ and V₃R-V₅R without increase in myocardial enzyme. The patient also developed respiratory failure requiring intubation and mechanical ventilation. Coronary angiography revealed only mild atherosclerosis without spasm or thromboembolic occlusion. Slow flow was seen in all coronary arteries, especially in the left anterior descending and right coronary arteries. This case speculated that transmural myocardial ischemia with ST segment elevation might be resulted from slow coronary flow. Transmural myocardial ischemia can occur owing to abnormalities of the coronary microcirculation. (J Geriatr Cardiol 2007;4:182-185.)

Key Words slow coronary flow phenomenon, ST segment elevation, transmural myocardial ischemia, coronary microcirculation

Case presentation

An 85-year-old male patient was sent to the emergency room because of "chest pressure for 4 hours". The electrocardiogram (ECG) showed 0.1-0.2mV ST elevation in V₁-V₃ leads. At the same time, the patient showed sign of respiratory failure and passed out. The patient was quickly intubated and put on mechanical ventilation. The arterial blood gas showed a pH of 7.20, PO₂ of 54mmHg, and PCO₂ of 105mmHg. Past medical history showed that the patient had 30 years history of chronic bronchitis. In the last 9 years, the patient developed recurrent dyspnea after exertion, relieved by rest or nitroglycerin (NTG). He had no history of hypertension, and diabetes. He smoked 12 cigarettes per day for 40 years and stopped smoking 10 years ago. He did not drink alcohol. On examination, this is an elderly patient, comatose, intubated on ventilator. His temperature, pulse, respiratory rate and blood pressure were within normal limit. There were no jaundice and no palpable superficial lymph nodes. His lips were mildly cyanotic. His chest exam showed symmetric bilateral respiratory movements and positive hyperresonance on percussion. Lung auscultation revealed scattered rhonchi and a few rales in both lower lung fields. The cardiac border was normal. No pathologic cardiac murmur could be heard. No clubbing, no edema of both lower extremities. The neurological examination showed that the patient was comatose. Serial ECGs showed further 0.2-0.5mV ST elevation in V₁-V₄ leads and 0.1-0.2mV ST elevation in V₃R-V₅R leads (Fig.1). The preliminary diagnoses were (1) acute anterior wall myocardial infarction, (2) right ventricular infarction, (3) respiratory failure,(4) loss of consciousness (R/O cerebro-vascular accident).

After admission to the intensive care unit, the blood pressure fell to 80/50mmHg and increased to 100/60mmHg with fluid infusion and low dose dopamine (3μg/kg/min). Other medications included intravenous NTG 20μg/min, oral aspirin, clopidogrel 75mg and subcutaneous enoxaparin 2000 IU/0.2ml Q12h. Serial myocardial enzymes such as CK, CK-MB, and cTnl were within normal range. The echocardiography showed dilated right ventricle and outflow tract. Pulmonary arterial systolic pressure was 40mmHg. Left ventricle ejection fraction (LVEF) was 55%. Left ventricle diastolic function decreased. Chest X-ray showed increased bilateral pulmonary markings and old lesions in both upper lungs Fig.2. On the second day of hospitalization the patient regained consciousness, and still experienced chest discomfort. He also experienced severe dyspnea, tachycardia, hypertension after bowel movement. The physical exam at that time showed increased rales and wheezes at the bases of the lungs. Acute left ventricular decompensation was suspected. Inotropes, diuretics vasodilators and sedation were given to relieve the symptoms. On the third day of hospitalization, blood gas analysis had improved but ST segment elevation still persisted. Coronary angiography was performed and revealed normal left main, mild atherosclerosis near the second diagonal branch of the left anterior descending (LAD) artery, a myocardial bridging near the third diagonal branch with 50% stenosis,
no epicardial coronary spasm or thromboembolic occlusion, no pathological change in the left circumflex (LCX) or right coronary artery (RCA). It displayed slow flow in all coronary arteries, especially in the LAD and RCA. Nitroglycerin 100μg was infused to the coronary arteries. There was no improvement in slow flow. After the coronary angiography, the ST segment elevation returned to baseline (Fig.3). The patient was given further treatment including intravenous papaverine and carnitine and the chest discomfort was relieved. The patient was extubated 7 days later. Long-term prescription included oral diltiazem 30mg TID, trimetazidine 20mg TID, losartan 25mg QD and simvastatin 10mg QD. At two months follow up, the echocardiogram showed LVEF 70% and PASP 40-45mmHg. The final diagnoses were (1) chronic bronchitis, (2) obstructive emphysema, (3) coronary heart disease, with possible coronary microcirculation dysfunction.

Discussion

Slow coronary flow phenomenon (SCFP) is an angiographic observation characterized by angiographically normal or near-normal epicardial coronary arteries with delayed dye opacification of the distal vasculature. It was first described by Tambe in 1972. In that report, the coronary arteriogram of 6 patients with angina revealed normal coronary arteries but a strikingly slow flow of the contrast through the major vessels. Further studies indicated that the SCFP patients were usually male, had a history of tobacco abuse, and typical angina on exertion. Usually a positive exercise test was commonly observed in these patients. SCFP is defined as corrected TIMI frame count (CTFC) greater than 2 standard deviations (SD) from normal published range for that particular vessel. This phenomenon can occur in any coronary artery as in single or multiple vessels at a time. Slow flow was seen more frequently in the LAD than in the LCX or RCA. The exact pathophysiologial mechanisms of SCFP remain uncertain. Small vessel dysfunction has been typically implicated since its first description. Microvascular resistance remains the important factor involved in regulating myocardial perfusion. Increased microvascular resistance may account for the slow coronary flow. The evidence of disease in the small vessels comes from the histopathological examination of endomyocardial biopsy specimens in SCFP patients, including endothelial thickening due to cell edema, capillary damage and reduced luminal diameter of the small vessels. Hemodynamic studies have shown that there is no increase in coronary blood flow after such stimuli as exercise, atrial pacing or intracoronary vessel dilator in some SCFP patients. It shows that the abnormal vasodilator function and decreased coronary blood flow reserve may play an active role in the SCFP. The endothelial dysfunction has also been proposed as the development factor for slow coronary flow. The endothelial dysfunction may cause imbalance in the endothelium-mediated vessel active substance and damage the vessel regulatory function. Intra-vascular ultrasound study has shown the extended massive calcification and atherosclerosis along the epicardial arteries in SCFP patients, which suggests that slow coronary flow be a form of early phase of atherosclerosis. In addition, microvascular spasm, platelet function disorder, and inflammation have also been documented to be involved in the pathogenesis of slow coronary flow.

In our report, the patient had clinical chest discomfort with typical ST segment elevation on the ECG. However, there were no changes on serial ECG tracings, no rise in serum cardiac markers and no regional wall motion abnor-

![Fig 1. First electrocardiogram after admission, It showed 0.2-0.5mV ST elevation in V1-V4 leads and 0.1-0.2mV ST elevation in Vr-V5R leads.](image-url)
mality on echocardiogram. All these results ruled out the diagnosis of acute myocardial infarction. The coronary angiogram showed no spasm which ruled out variant angina pectoris too. In the past, transmural myocardial ischemia associated with ST segment elevation was generally attributed to acute epicardial coronary artery obstruction, for example thromboembolic occlusion, epicardial coronary spasm, and coronary air embolus. But there were case reports about transmural ischemia induced by slow coronary flow. In this case, only mild atherosclerosis without severe obstructive change was observed in the epicardial coronary artery. It was characterized by delayed distal vessel opacification. The finding suggests that transmural myocardial ischemia without epicardial coronary artery obstruction can occur owing to abnormalities of the coronary microcirculation or small vessel disease. We assume that long-term hypoxia owing to chronic obstructive pulmonary disease affects endothelial function and coronary microcirculation which leads to the slow flow, and so cause transmural myocardial ischemia under stress. On the other hand, intra-thoracic positive pressure may affect the venous return and cardiac output in patients on mechanical ventilation. It is not certain whether slow coronary flow has relation to mechanical ventilation which deserves further discussion.

There are limited studies about the treatment of slow coronary flow. According to previous reports, the calcium T-channel blocker and dipyridamole may have clinical benefits. In this case, the patient had been asymptomatic on treatment including anti-platelet, ameliorated coronary ischemia, improved coronary microcirculation and myocardial metabolism. ST elevation had reversed to baseline.

SCFP has distinct angiographic feature and clinical manifestations which are different from other usual syndrome. Therefore, some scholars would like to call it as a new syndrome “slow coronary flow syndrome”. Previous studies had indicated that SCFP had close relation to the structure and function of coronary microcirculation. With the development of interventional technology, this phenomenon has been more and more recognized in clinical work. Further investigations are required to elucidate the etiologies, clinical manifestations and treatment of SCFP in the future.
References


