Coronary Spastic Myocardial Injury Presenting in the Emergency Room: A Case Report

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We describe a 57 year old man with an acute coronary spastic Myocardial injury who suffered severe chest pain and developed life-threatening ventricular fibrillation. Following defibrillation, both the ventricular fibrillation and the hyperacute T wave disappeared immediately and subsequent serum levels of cardiac enzymes were normal. Subsequent coronary angiography revealed atherosclerotic changes in all coronary arteries, with a discrete eccentric stenosis of 80% in the proximal first diagnonal branch of the left anterior descending coronary artery. Percutaneous transluminal coronary angioplasty was performed. The patient recovered uneventfully.

***Key words:* coronary spastic myocardial injury, ventricular fibrillation, defibrillation**

Introduction

It has been shown that coronary spasm superimposed on atherosclerotic stenosis can be a cause of coronary thrombus formation that leads to acute myocardial infarction[1]. However, the mechanisms of coronary spasm in the development of sudden cardiac death remain uncertain, as it has been reported that the infarct size in these patients is relatively small and that spontaneous coronary reperfusion usually occurrs in the early stages of myocardial infarction[2].

We describe a patient with coronary spastic myocardial injury who later developed ventricular fibrillation. Upon defibrillation, the ventricular fibrillation together with the electrocardiographic hyperacute T wave reversed immediately, and subsequent serum levels of cardiac enzymes were normal.

Case Report

A 57-year-old man arrived at our emergency room with acute substernal chest pain accompanied by heavy perspiration and nausea for 40 minutes. His past medical history was unremarkable. He smoked one pack of cigarettes per day and did not use alcohol.

Physical examination on admission revealed a blood pressure of 140/90 mmHg, pulse 85 per minute, respirations 16 per minute, and temperature of 36°C. There were bilateral crackles in the basal region of the lungs. The heart rhythm was regular and a grade 2 to 3/6 systolic murmur was heard at the apex with radiation to the left axilla. The remainder of the physical examination was normal.

Complete blood count and blood chemistry were normal. Creatine kinase and CKMB were 130 and 39 IU/L at admission; at 6 hours, 138 and 18 IU/L; at 12 hours, 146 and 20 IU/L, at 18 hours, 161 and 19 IU/L; and at 36 hours, 49 and 11 IU/L, respectively. LDH isoenzyme levels were normal.

Accepted for publication: December 24, 2001
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Chest X-ray films showed cardiomegaly and increased pulmonary vascularity. The electrocardiogram showed hyperacute T waves in I, aVL, and V1 to V5 precordial leads and ST segment depression in the inferor leads, with Q waves in V1 to V3 (Fig. 1). An echocardiogram revealed an akinetic septum and hypokinetic anterior wall with an ejection fraction of 35%.

Soon after admision to the emergency room, the patient suddenly developed ventricular fibrillation as shown on the bedside heart monitor. Immediate defibrillation of 200 J was given. The patient's rhythm converted to sinus rhythm immediately after the defibrillation and, surprisingly, the former electrocardiographic hyperacute T wave and ST segment changes also recovered concomitantly.

Because of recurrent anginal pain, the patient underwent coronary angiography. It revealed atherosclerotic changes in all coronary arteries, with diffuse luminal irregularity, and a discrete 80% eccentric stenosis in the proximal first diagonal branch of the left anterior descending coronary artery (Fig. 2). Percutaneous transluminal coronary angioplasty was performed. Follow-up echocardiograms showed mild hypokinesis over the anteroseptum with preserved ventricular systolic function (ejection fraction of 52%). The patient recovered uneventfully.

Discussion

Our patient presented in the emergency room with severe chest pain and electrocardiographic hyperacute T waves, which were reversed immediately following defibrillation. Myocardial infarction was subsequently ruled out on the basis of persistently normal serum CKMB levels. However, there was coronary spastic myocardial injury as revealed by the Q wave and relatively high CKMB level at admission, as well as akinesis of the septum and hypokinesis of the anterior wall with an ejection fraction of 35%.

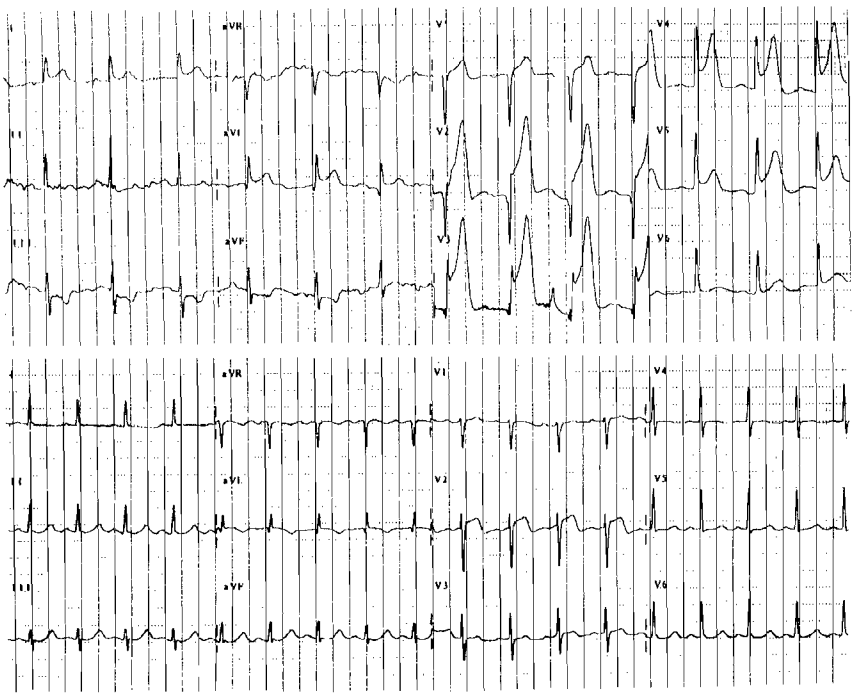

Fig. 1 Electrocardiogram performed on admission showing hyperacute T waves in I,aVL,V1 to V5 precordial leads and ST segment depression in the inferior leads with Q waves in V1 to V3 (top). Electrocardiogram after defibrillation shows sinus tachycardia with non-specific ST-T changes (bottom).

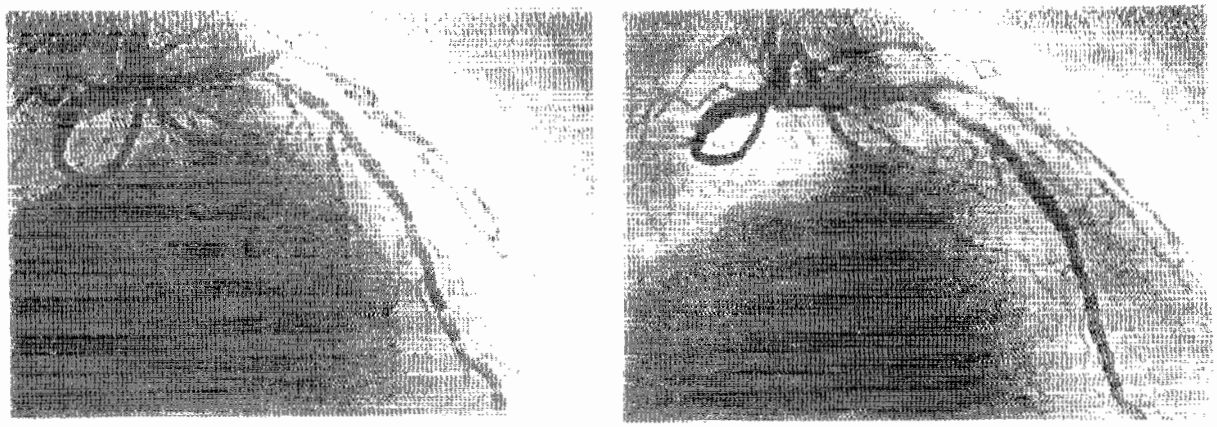

Fig. 2 Coronary arteriograms before (left) and after (right) percutaneous transluminal coronary angioplasty of the diagonal branch of the left anterior descending coronary artery.

Patients with possible acute myocardial infarction in whom myocardial infarction is subsequently ruled out may have transient coronary occlusion, coronary spasm, or other manifestations of unstable angina. In our patient, acute myocardial infarction was impressed by the presence of persistent chest pain and hyperacute T wave upon admission. The patient developed ventricular fibrillation which was reversed by defibrillation. Suprisingly, the hyperacute T wave also disappeared following the defibrillation and myocardial infarction was subsequently ruled out. There are three interesting observations. First, although it is generally believed that the infarct size caused by coronary spasm is relatively small and spontaneous coronary reperfusion occurs in the early stages of myocardial infarction[2], ventricular fibrillation and sudden cardiac death may indeed develop[3-5]. Secondly, myocardial ischemia is expected to produce changes in wall motion almost immediately, even before electrocardiographic changes. Following reversal of ischemia, however, the electrocardiographic changes occur earlier than the resolution of wall motion abnormalities, as was depicted in our patient, probably as a result of stunning. Thirdly, the concomitant disappearance of hyperacute T waves immediately following defibrillation suggests that the defibrillation per se probably influences the dynamic interaction between atherosclerosis, platelet aggregation and coronary spasm.

The coronary arteriograms clarified the clinical picture in our patient. They showed atherosclerotic changes in all coronary arteries, with diffuse luminal irregularity, and a discrete eccentric stenosis in the diagonal branch of the left anterior descending coronary artery. The findings in this case therefore indicate the involvement of coronary spasm in the left anterior descending coronary artery in the development of acute myocardial injury, and the subsequent unstable angina was attributed to the coronary occlusion in the diagonal branch. Our case clearly demonstrated a typical course of coronary spastic myocardial injury. Moreover, its surprising immediate resolution following defibrillation deserves further elucidation.

Long-acting nitrates and calcium antagonists are the mainstay of therapy for variant angina and are extremely effective in preventing coronary artery spasm. Our patient was discharged in stable condition and followed up at our outpatient department. He was maintained with 20 mg isosorbide mononitrate and 90 mg diltizem once daily for his variant angina as well as prevention of recurrent attacks.

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急性冠狀動脈痙攣引發心肌傷害：病例報告

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一位57歲男性急診病患，有胸痛及心電圖呈現超急性T波現象，突然出現心室顫動。電擊治療後心電圖回復正常，而心肌激酶亦正常。此病患診斷爲急性冠狀動脈痙攣引發心肌傷害及心室顫動，於急診室電擊後回復正常。心導管檢查發現diagonal branch冠狀動脈有狹窄，經氣球擴張術後症狀改善出院。

關鍵詞：急性冠狀動脈痙攣引發心肌傷害，心室顫動，去纖維顫動術

接受刊載：90年12月24日
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