

Spontaneous left main coronary artery spasm and intravascular ultrasonography findings

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SUMMARY

Vasospasm causes an abrupt and temporary occlusion in coronary arteries in patients with normal coronary arteries. Nonetheless, vasospasm of the left main coronary artery is unusual and can be spontaneous or iatrogenic. We herein report a very rare cause of temporary spasm of LMCA, which resulted in vasospastic angina and anginal chest pain. Intravascular ultrasonography should be considered as a good diagnostic tool in revealing the exact etiology of occlusions such as vasospasm or thrombosis.

Key words: Intravascular ultrasonography, left main coronary artery, vasospasm

ÖZET

Spontan sol ana koroner arter spazmı ve intravasküler ultrasonografi bulguları

Vasospazm, normal koroner arterleri olan hastaların koroner arterlerinde ani ve geçici bir tıkanmaya yol açabilir. Bununla beraber, sol ana koroner arterin vazospazmı pek sık değildir ve bu durum spontan veya iyatrojenik olabilir. Bu yazıda sol ana koroner arterin vazospastik anjina ve anjinal göğüs ağrısına neden olan çok nadir ve geçici bir nedeni sunulmuştur. İnvasküler ultrasonografi, vazospazm veya tromboz gibi tıkanıklıkların etiolojisini göstermede iyi bir tanısal araç olarak düşünülmelidir.

Anahtar kelimeler: İnvasküler ultrasonografi, sol ana koroner arter, vazospazm

Introduction

Coronary artery spasm is one of the important etiologic factors in patients with chest pain. Vasospasms cause an abrupt and temporary decrease in the diameter of coronary artery. The duration and location of the spasm are important, especially when it is located in a critical site such as left main coronary artery (LMCA) (1). Vasospasm of LMCA is rare. If the spasm is prolonged, it may lead to acute coronary syndromes and be fatal. Although most of the vasospasms of LMCA are iatrogenic (catheter induced), spontaneous spasm of LMCA is relatively uncommon and the mechanism is not known exactly. Moreover, intravascular ultrasonography (IVUS) findings may be elucidatory in some selected cases. We herein report a very rare cause of temporary spasm of LMCA, which resulted in vasospastic angina and anginal chest pain.

Case report

A 62-year-old smoker woman without a history of any cardiovascular disease was admitted to the hospital because of typical chest pain. Her chest pain had started 24 hours ago while resting. An admission 12-lead electrocardiogram (ECG) showed ST elevation in lead aVR and ST depression in all leads (Figure 1A). Vital parameters were within normal ranges. Standard biochemical tests, cardiac enzymes and troponin T were within normal limits. She was diagnosed as acute coronary syndrome, and then transferred to the coronary intensive care unit. She was treated with intravenous infusion of nitrate and heparin at recommended doses. Her chest pain was relieved after the administration of medications and ST segment changes disappeared on follow up ECG after 10 minutes (Figure 1B). There were no regional wall motion abnormalities in two-dimensional echocardiographic examination. Elective coronary angiography

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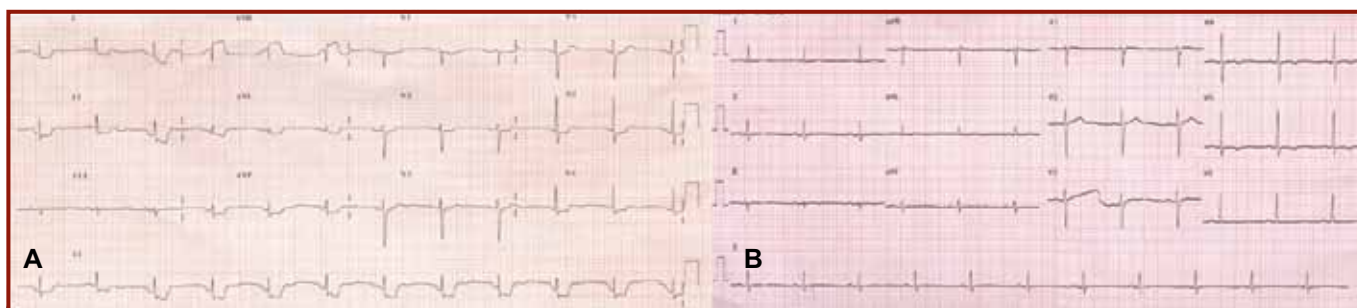


Figure 1. 12-lead electrocardiograms at hospital admission (A), and on follow-up (B)

was performed and did not reveal any atherosclerotic changes in the coronary arteries. Subsequently, an intravascular ultrasonographic (IVUS) examination was performed to detect whether there were any ruptured and/or partially thrombotic atherosclerotic plaques, which might be responsible for the development of acute coronary syndrome.

In the IVUS examination (40 MHz Atlantis SR Pro, Boston Scientific, USA), there were minimal atherosclerotic changes with negative remodeling (Remodeling Index: 0.90) in the distal segment of LMCA, but there was no ruptured and/or partially thrombotic plaque in LMCA. However, there was no atherosclerotic disease in the proximal segment of the LMCA (Figure 2). We suggested a provocation test with an intracoronary injection of acetylcholine to reproduce

the clinical syndrome, but the patient rejected this test because of the risks of the procedure. Then, we presumed that LMCA spasm resulted in vasospastic angina and led to anginal chest pain in our patient on the basis of ECG findings. Medical treatment with calcium channel blocker (diltiazem 120 mg tb 1x1), isosorbide-5-mononitrat (20 mg 2x1), and aspirine (100 mg 1x1) were given. She was advised to stop smoking. The patient was free of anginal chest pain under these medications on follow-up control after six months.

Discussion

Vasospasm is defined as a decrease in the diameter of coronary arteries and plays an important role in the generation of variant angina even in patients with

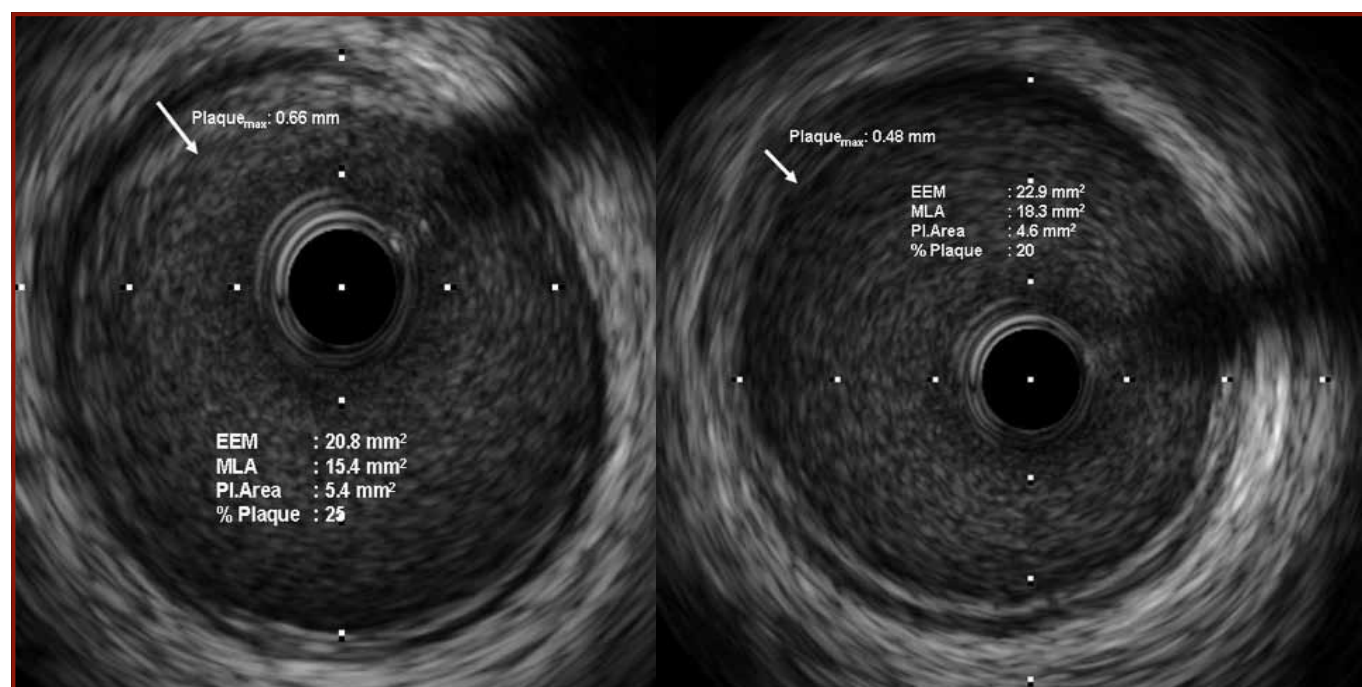


Figure 2. Intravascular ultrasonographic examination of left main coronary artery at the time of coronary angiography. Distal segment of the left main coronary artery (A) showed minimal atherosclerotic disease with negative remodeling (Remodeling index: 0.90) according to proximal segment of the left main coronary artery (B)

EEM: external elastic membrane, MLA: minimal lumen area

normal coronary arteries. Spasm of coronary arteries may be spontaneous or iatrogenic. Iatrogenic spasm of coronary arteries is often catheter-induced during coronary angiography. Spontaneous vasospasm of coronary arteries is an unusual cause of variant angina. Although the precise mechanism has not been determined, several reasons have been suggested for predisposition to variant angina by causing coronary vasoconstriction. These are overactivity of parasympathetic nervous system, reduced sympathetic activity, a deficiency in nitrite oxide production, hypomagnesemia, hyperinsulinemia, vitamin E deficiency and cocaine use (2,3).

Variant angina can be diagnosed by ECG changes occurring during resting chest pain and disappearing after the administration of nitroglycerin. In the present case, LMCA spasm was diagnosed by the ECG finding. Also, coronary angiography could be performed in variant angina to distinguish patients who had atherosclerotic coronary disease and normal coronary arteries. In our case, coronary angiography showed normal anatomy of the coronary arteries. IVUS examination at the time of coronary angiography may help identify the exact coronary anatomy (4). There are limited data about IVUS findings of the spasm site of coronary arteries. Early atherosclerosis and high incidence of negative remodeling were shown at the site of focal vasospasm, even in the absence of angiographically normal coronary arteries (2,5). There was minimal atherosclerosis at the site of

focal vasospasm in our patient's IVUS examination. In the absence of angiographically significant coronary artery disease, pharmacological and non-pharmacological provocative tests can be used to provoke coronary spasm and confirm the diagnosis of variant angina. Among these, intracoronary injection of acetylcholine is the most preferred method.

In conclusion vasospasm of LMCA is a rare but potentially life-threatening condition. IVUS examination at the time of coronary angiography may be helpful and guides the physicians to make an exact decision.

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