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Case report

ST elevation myocardial infarction caused by coronary slow flow: Case report and brief review of the literature

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ABSTRACT

Coronary Slow Flow Phenomenon (CSFP) is an angiographic phenomenon in which vessel opacification is delayed without any evidence of obstructive epicardial coronary disease. We aim to present, in this paper, extremely slow coronary flow along with its severe clinical manifestation. A 47-year-old male patient was admitted to our emergency department with ST elevation myocardial infarction caused by coronary slow flow. Oral Acetylsalicylic acid, nebivolol and atorvastatin therapy successfully resulted in complete resolution of his symptoms during the 18-month observation.

Introduction

The Coronary Slow Flow Phenomenon (CSFP), an angiographic phenomenon first described by Tambe et al. in 1972, refers to the slow movement of contrast agents in the coronary arteries without significant stenosis or myocardial bridge.[1] The Thrombolysis in Myocardial Infarction (TIMI) frame count, a reproducible index of coronary flow, functions as the quantitative measurement of coronary slow flow.[2] More specifically, it is a numerical representation of cine frames which are necessary for the contrast agents to reach a pre-specified distal coronary artery landmark. The process is continuous with a correction through normalization of the left anterior descending artery (LAD). The corrected TIMI frame count (CTFC) is the division of the absolute TIMI frame count in the LAD by 1.7. The CSFP phenomenon is characterized by CTFC >2 standard deviations from normal published range (21 ± 3).[2] It has been reported that 1–5.5% of patients who undergo coronary angiography experience CSFP.[3] However, the pathophysiology of CSFP has not been comprehensively discerned. In this paper, we present a rare case of STEMI caused by coronary slow flow, which might help us to understand better the CSFP.

Case

A 47-year-old man who complained of chest pain spreading towards his shoulder, the pain having begun nearly an hour prior, was admitted into our emergency department. His blood pressure was 110/75 mm Hg, his heart rate was 52 beats/min, and his cardiopulmonary examination was normal. His electrocardiogram showed ST elevation in leads V1–4 (Fig. 1A). Then, he was immediately taken to the laboratory for coronary angiography. There was no significant stenosis, coronary vasospasm or myocardial bridge in coronary angiography. Further, intracoronary nitro-glycerine fusion was performed to exclude vasospastic angina. However, no change in ST elevation was observed. On the other hand, advanced-degree slow flow was observed in the LAD and right coronary artery (RCA). The TIMI frame-count method was used to measure the degree of slow flow. The CTFCs were observed to be 52 frames for the left anterior descending coronary artery (Fig. 2A) and 35 frames for the RCA (Fig. 2B). After about 30 min, the patient's chest pain had disappeared completely; ST elevation improved (Fig. 1B); and Troponin I was slightly elevated (Troponin I 0.24 μg/L (normal range, 0.010–0.023 μg/L)). In terms of lipid profile, the findings were as follows: total cholesterol: 182 mg/dL, LDL: 109 mg/dL, HDL: 35 mg/dL, and TG: 192 mg/dL. Other biochemical values were normal, his body mass index was in the normal range, and there were no cardiac risk factors other than smoking. Two-dimensional echocardiography showed normal left ventricle function and no wall motion abnormalities. Acetylsalicylic acid 300 mg, nebivolol 5 mg and atorvastatin 20 mg were started. The patient was discharged without chest pain after four days with a prescription of acetylsalicylic acid 100 mg, nebivolol 5 mg and atorvastatin 20 mg. There was no complaint reported during the 18-month period after discharge.

Discussion

Recurrent chest pain is a commonly observed symptom among patients with CSFP.[4] Although it is rare, CSFP may also lead to life-threatening situations such as ST elevation myocardial infarction

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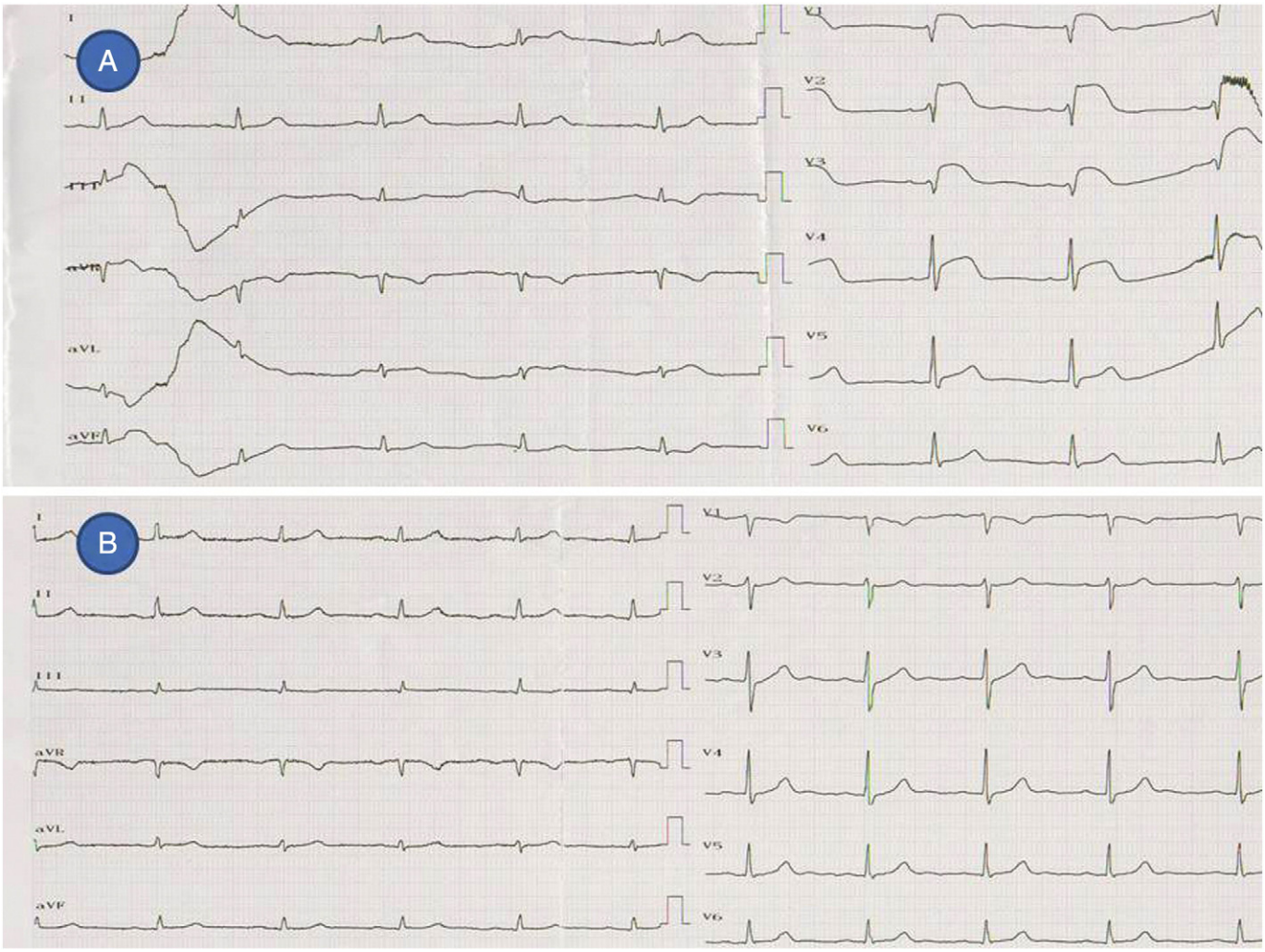

Fig. 1. The electrocardiogram shows ST elevation in leads V1–4 during presence of chest pain (A). ST elevation improved after disappearance of chest pain (B).

(STEMI), ventricular arrhythmias, and sudden cardiac death.[5–10] Recently, a patient presented as having acute coronary syndrome reportedly illustrated an association between CSFP and a new onset of intermittent left bundle branch block (LBBB).[11] Also, Sunbul et al.[12] suggested that slow coronary flow might lead to ST segment elevation in the exercise stress test.

Although different theories have been posited regarding the causes of CSFP, research has not been able to discern comprehensively its pathogenesis. In a variety of researches, small vessel disease, endothelial dysfunction, subclinical atherosclerosis, inflammation, and anatomic properties of coronary arteries have been reported as existing in association with CSFP. Among these, small vessel dysfunction is one of the most typical of the pathogenesis of CSFP.[13] In an effort to confirm this hypothesis, Beltrame et al. suggested that fibromuscular hyperplasia, medial hypertrophy, myointimal proliferation, endothelial edema, and the thickening as well as the degeneration of the coronary micro-vessels correspond with the phenomenon.[14] Therefore, it is perhaps more fitting to suggest that the coronary microcirculation might be a result of the coexistence of structural and functional abnormalities. In a variety of cases, patients with CSFP were reported to have increased levels of plasma homocysteine, increased endothelin-1 release and reduced nitric oxide bioactivity, which indicated impaired endothelial function.[3,15–18] CSFP patients have metabolic syndrome more frequently.[19] As a result of the use of IVUS technique and flow rate measurements, diffuse intimal thickening, widespread calcification along the coronary vessel wall, and non-obstructive atheromatous coronary changes were reported among patients with CSFP.[20] Based on the data presented here, it can be argued that CSFP might reflect diffuse, non-obstructive atherosclerotic disease of epicardial vessels together with microvascular disease. Moreover, elevated plasma concentration of high-sensitivity C-reactive protein and interleukin-6 were documented among CSFP patients.[21] In a similar vein, higher levels of plasma-soluble adhesion molecules such as intercellular adhesion molecule-1, vascular cell adhesion molecule-1 and E-selectin were reported to exist in relation to coronary slow flow.[22] Red cell distribution width and serum uric acid levels, among other inflammatory markers, were studied in association with CSFP patients.[23,24] In endothelial dysfunction, abnormalities in inflammatory parameters can possibly be an indicator; both of these contribute to coronary slow flow. Shao-Ping et al.[25] demonstrated the existence of CSFP in relation to higher tortuosity and more distal branches in coronary arteries. Thus, certain anatomic properties of coronary arteries could be alleged to have an effect on disturbed coronary flow and endothelial damage, therefore resulting in CSFP.

Treatment for CSFP has not yet been clearly defined, but several drugs have proven to be effective at various levels. So far, pharmacological therapy for CSFP has not been executed on a large scale. Certain small studies have presented evidence regarding the drugs. In some studies, dipyridamole, mibefradil and nitroglycerine have been used for the treatment of CSFP.[19,26] Statins present certain benefits for CSFP patients, partially owing to their anti-inflammatory properties.[27] Nebivolol not only can improve endothelial function but also can remedy symptoms considerably; therefore, the drug enhances the