INTERNAL MEDICINE

□ CASE REPORT □

A Destabilized Case of Stable Effort Angina Pectoris Induced by Low-dose Adenosine Triphosphate

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Abstract

A 79-year-old man was diagnosed with sudden deafness. He had previously experienced a suspected episode of angina pectoris. At a local hospital, after 500 mg of hydrocortisone and 80 mg adenosine triphosphate (ATP) were administered, he became aware of chest discomfort. An electrocardiogram revealed serious ST-segment depressions. He was diagnosed with a non-ST elevated myocardial infarction (NSTEMI). Emergency coronary angiography revealed triple vessel disease, and the lesion was successfully stented. The mechanisms whereby the stable effort angina pectoris destabilized in this case were thought to include a reduction of the local blood flow because of an ATP product and probable thrombus formation in response to the administered steroids.

Key words: unstable angina pectoris, adenosine triphosphate

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Introduction

Adenosine triphosphate (ATP) is a nucleoside triphosphate that is used in cells as a coenzyme. ATP products are used for both the treatment of arrhythmias and inner ear disorders, but they can also induce myocardial ischemia. We herein present a rare case of stable effort angina pectoris that became destabilized after the administration of a low-dose ATP product.

Case Report

A 79-year-old man visited an otorhinolaryngology clinic because of the chief complaint of sudden left-sided hearing loss. He was diagnosed with sudden deafness (SD) (1) and admitted to a general hospital. His body height was 155 cm and body weight was 49.1 kg. In medical examinations, there were no problems except for the left-sided hearing loss. The serum triglyceride level was 69 mg/dL. Although he had experienced chest compression and pain with exertion and was suspected to have effort angina pectoris by his regular doctor, no examinations had been carried out because of the patient's reluctance to do so. His cardiac risk factors included hypertension, dyslipidemia, a history of brain infarction and a current smoking status. His medications were aspirin 100 mg o.d., nifedipine 40 mg o.d., doxazosin 2 mg o.d., telmisartan 40 mg o.d., furosemide 10 mg o.d., pravastatin 10 mg o.d., teprenone 50 mg v.d.E. At the general hospital, 500 mg hydrocortisone and 80 mg adenosine triphosphate (ATP) were administered via an intravenous drip over a 3-hour period (ATP; 0.44 mg/min). These treatments were adequate as reported previously (2, 3). After initiating the intravenous drip, he became aware of chest discomfort. This symptom disappeared 20 minutes later. An electrocardiogram (ECG) obtained after the administration of the medications revealed serious ST-segment depressions in the II, III, aVF and V2-V6 leads (Fig. 1). All cardiac enzyme levels were normal, except for those of troponin-T. His blood pressures pre and post intravenous administration of ATP were 178/94 mmHg and 133/72 mmHg, respectively. The heart rates changed from 78 beats per minute (bpm) to 96 bpm. Thus, he was suspected to have ischemic heart disease and was transferred to our institution. His blood pressure was 156/83 mmHg, and heart rate was 78 bpm. Although no significant ST-segment changes in ECG were ob-

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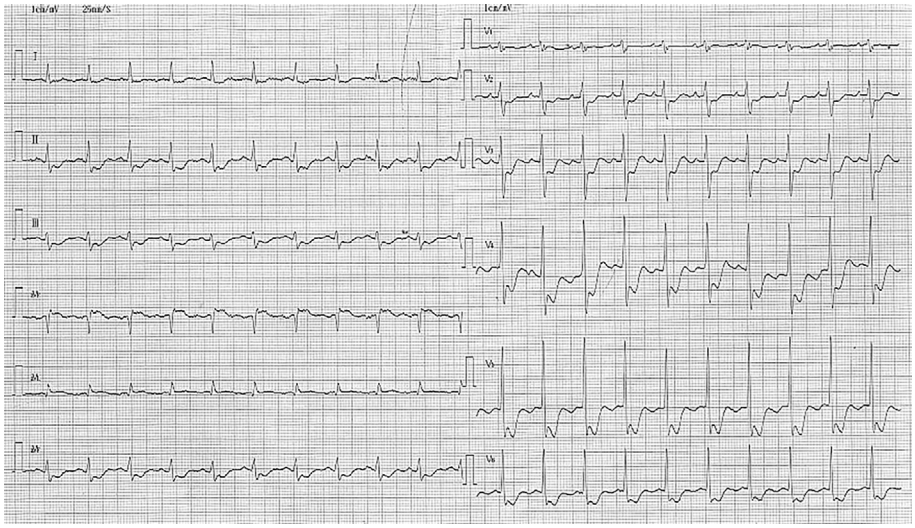

Figure 1. **Electrocardiogram after the initiation of an adenosine triphosphate product.**

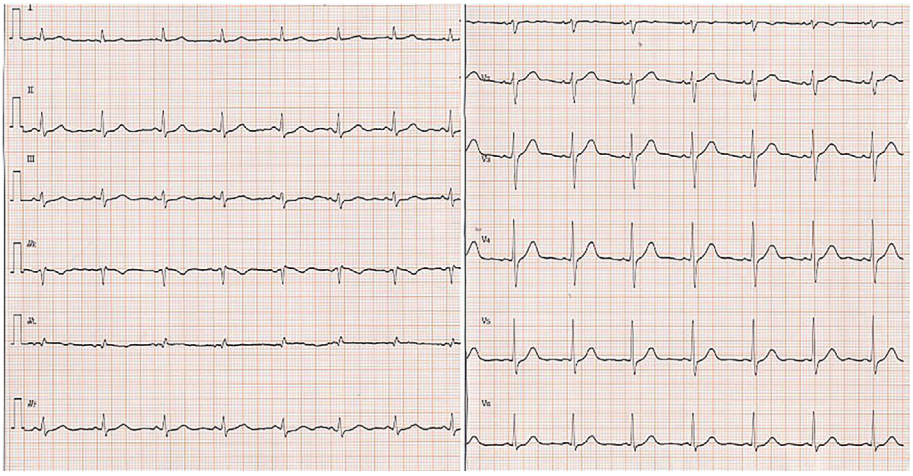

Figure 2. **Electrocardiogram performed at our emergency department.**

served at our emergency department (Fig. 2), his troponin-T levels were high. His echocardiogram results showed no wall motion abnormalities. Considering these findings, he was diagnosed with non-ST elevated myocardial infarction (NSTEMI) and underwent emergent coronary angiography (CAG) because his symptoms persisted. CAG revealed 75%, 90%, and 75% stenosis of his distal-right coronary artery (Fig. 3A), proximal-left anterior descending coronary artery (LAD), and mid-left circumflex coronary artery (Fig. 3B-3D), respectively. The causative lesion was identified in the LAD based on the severity of the stenosis in the LAD. Subsequently, sequential balloon expansion was performed, and the lesion was successfully stented (Fig. 3E).

The treatment of his SD was continued in our hospital after percutaneous coronary intervention using steroids, however, his hearing loss did not recover, and thus further treatments were planned to be performed at the former hospital.

Discussion

The detailed mechanism regarding how the extracorporeal administration of ATP products affect SD remains unknown, and ATP is one of most frequently orally or intravenously administered medications (3).

Holton et al. searched for compounds that were associated with vasodilation after sensory nerve reverse conduction stimulation and found a vasodilation effect in a neurotransmitter in the dorsal horn and reported that the possible causative agent was ATP. However, this study was published over 60 years ago (4).

ATP products can induce vasodilation effects because of the direct effects of ATP or one of its metabolites, adenosine. A coronary artery that has stenotic lesions can provoke transient myocardial ischemia via a relative blood flow reduction in response to an ATP product. Thus, ATP products are used to examine the coronary flow reserve to achieve maximal hyperemia (ATP; 150 μg/kg per minute) (5). In the

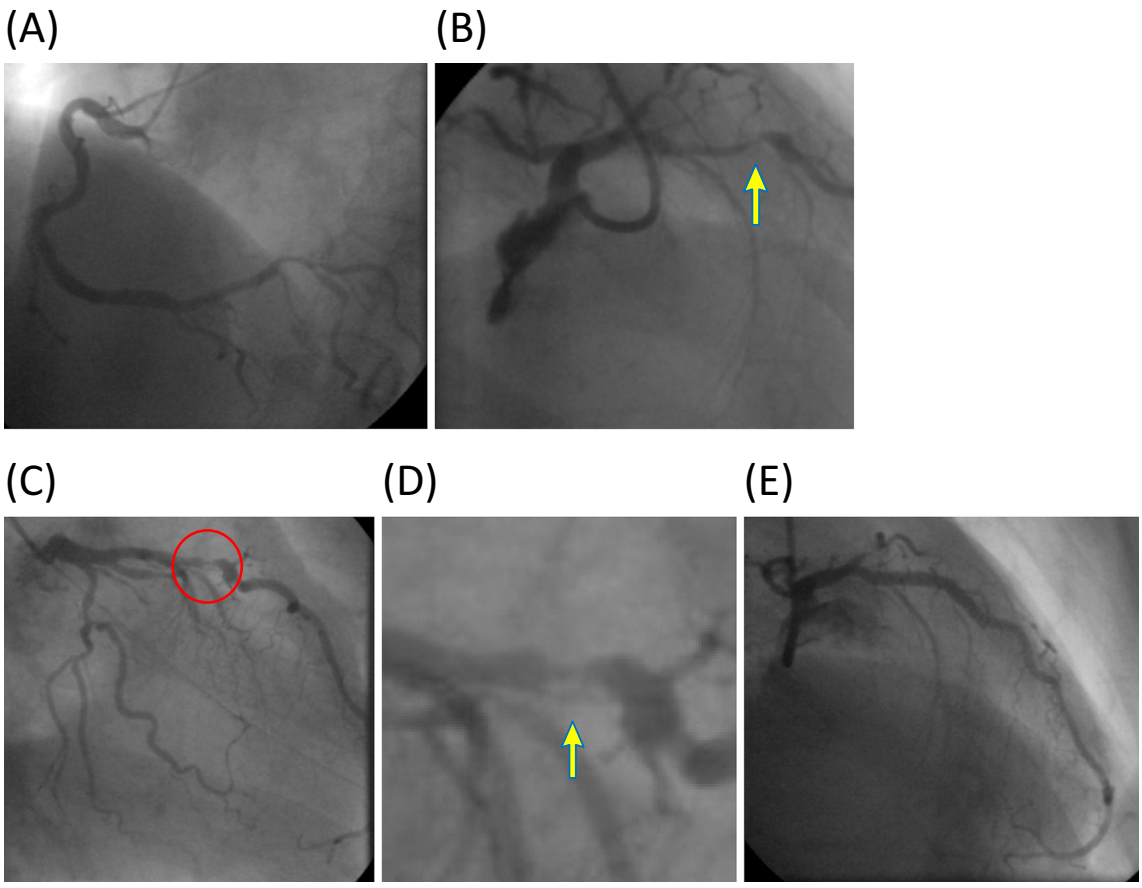

Figure 3. **(A) The image shows 75% stenosis of the distal-right coronary artery. (B) The image shows 90% stenosis of the proximal-left anterior descending coronary artery (arrow). (C) The image shows 90% stenosis of the proximal-left anterior descending coronary artery and 75% of the mid-left circumflex coronary artery. The red circle was enlarged in (D). (D) The image shows 90% stenosis of the proximal-left anterior descending coronary artery (arrow). (E) Post-stent deployment, which achieved excellent results.**

present case, it was difficult to definitively determine whether hyperemia occurred and if the effects of an ATP product only cause plaque destabilization.

The mechanism whereby the stable effort angina pectoris destabilized in the present case was thought to be due to the local blood flow reduction induced by ATP and the probable thrombus formation induced by steroids, which were included in the SD therapy. It is essential that otolaryngologists and emergency physicians be aware of such a case. We should also confirm the presence of a suspected angina pectoris episode prior to the use of an ATP product. Generally, regarding the mechanism of thrombus formation, it has been reported that the steroid hormone increases the blood clotting ability (6). Although thrombus formations should have been confirmed by utilizing intravascular ultrasound (IVUS) or optical coherence tomography (OCT), at the time that this case was encountered neither IVUS nor OCT were covered by the Japanese health insurance system.

In conclusion, we herein reported, to the best of our knowledge, the first case in which stable effort angina pectoris progressed to a NSTEMI in response to treatment with a low-dose ATP product and steroids.

The authors state that they have no Conflict of Interest (COI).

Acknowledgement

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