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Case Report

A case of acute and late coronary events after blunt chest trauma
Attention to the late onset angina

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ABSTRACT

Mechanisms of acute myocardial infarction caused by traumatic coronary artery injury have been reported. However, late-onset coronary artery stenosis associated with trauma is less well known. We experienced a case in which acute myocardial infarction of the right coronary artery occurred at the time of blunt chest trauma (BCT) caused by a traffic accident and an increase in coronary artery stenosis in the left anterior descending artery (LAD) branch about 1 year later. A comparison of a volume-rendering image created from enhanced-contrast computed tomography at the time of trauma and coronary angiography revealed that the trauma site and the stenotic lesion in the LAD were in very close proximity, suggesting to us that traumatic coronary artery injury without flow limitation may have developed into high-grade stenosis in the LAD 1 year later. In this case we were able to demonstrate a causal relationship between BCT and delayed coronary artery stenosis. After BCT, it is necessary to be aware of the possibility of delayed coronary artery stenosis even if coronary injury is absent in the acute phase.
<**Learning objective:** Careful follow up for the onset of angina pectoris is necessary to prevent coronary events after blunt chest trauma.>

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Introduction

Cardiac injury caused by blunt chest trauma (BCT) is a serious condition that can often be fatal. In particular, heart failure caused by damage to the myocardium, coronary arteries, and aorta, with blood leakage involved in acute cardiac tamponade, and rupture of valves and the ventricular/atrial septal walls can lead to cardiac shock [1]. Pathological autopsy or autopsy imaging have revealed cardiac chamber ruptures in 36–65% of deaths by BCT [2,3]. Even when there is no appearance of trauma, ventricular arrhythmia due to *commotio cordis* after BCT can be fatal [4,5].

Because the coronary arteries run along the surface of the heart, they are susceptible to damage from an external force [6]. The coronary arteries most often injured by BCT are the left anterior descending artery (LAD), followed by the right coronary artery (RCA), and, less often, the left circumflex branch [6,7]. If an external force damages the coronary arteries, even if there is no blood leakage, there may be coronary dissection, inner membrane tear, thrombus formation, and spasm. Acute myocardial infarction (AMI) caused by traumatic injury of a coronary artery may not be difficult to diagnose by electrocardiogram or cardiac echocardiography; however, mild coronary artery injury can occur without obstruction or obvious stenosis and may not be noticed even with coronary angiography (CAG).

Case report

A 51-year-old man was admitted to our hospital after being involved in a traffic accident. His car had collided head-on with a

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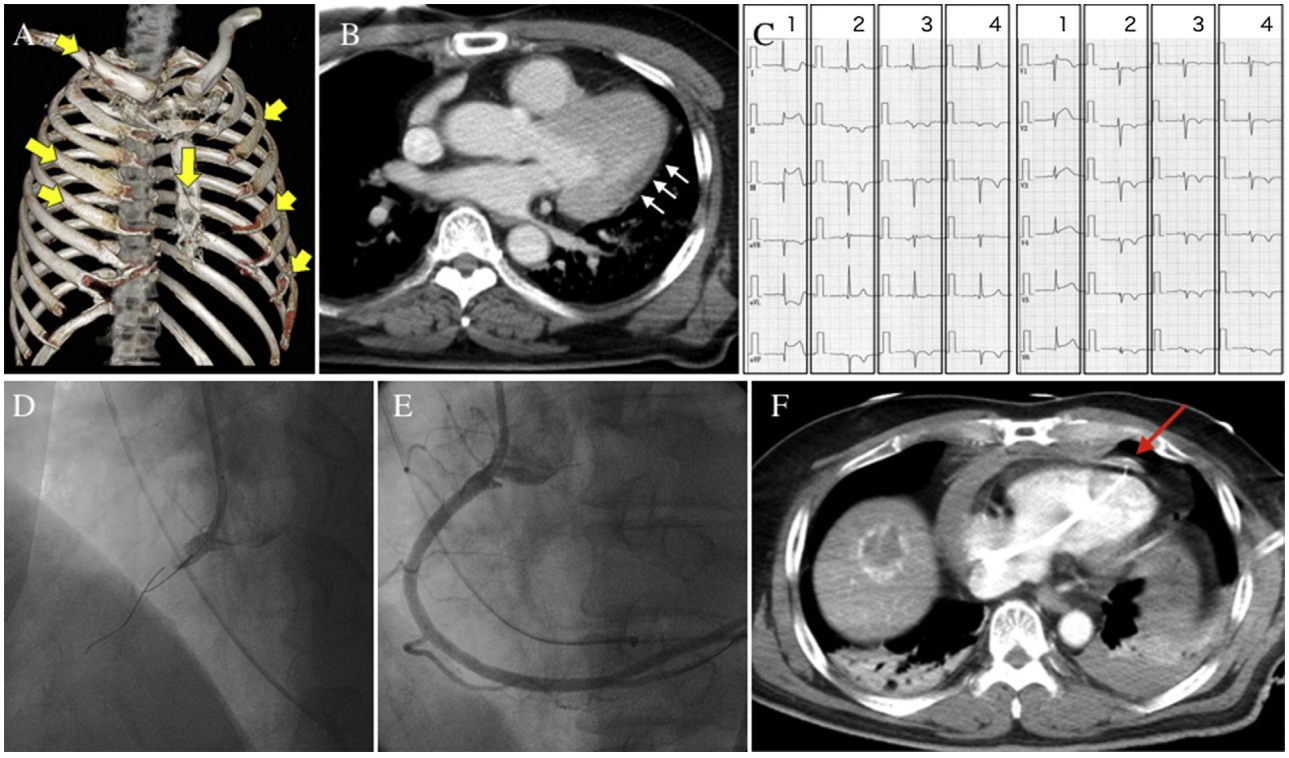

Fig. 1. (A) VR image created from E-CT. Yellow arrows indicate the fracture site on the thorax. (B) E-CT image before we performed CAG and PCI. White arrows indicate infarcted left ventricular inferior wall which was not enhanced by contrast medium. (C) Image of electrocardiogram recorded at the emergency room when he visited (1), after 6 months of BCT (2), after 14 months of BCT, and before PCI for LAD (3) and after 2 years of BCT (4). (D) Image of right coronary angiography which indicates total occlusion of the RCA ostial site and shows the IVUS probe inserted in the false lumen and the second guide wire passing through the true lumen. (E) Final coronary angiography image after implantation of a bare-metal stent in segment 1 of the RCA. (F) E-CT image performed immediately following PCI. Red arrow indicates the leakage point of the contrast medium on the epicardium.
VR, volume-rendering; E-CT, enhanced-contrast computed tomography; PCI, percutaneous coronary intervention; CAG, coronary angiography; BCT, blunt chest trauma; LAD, left anterior descending coronary artery; RCA, right coronary artery; IVUS, intravascular ultrasound.

heavy-duty truck, and his right tibia, right fibula, right clavicle, sternum, and several ribs were fractured. From the distribution of the fracture sites on his thorax (Fig. 1A), it was inferred that the fractures were caused by compression from his seat belt. Enhanced-contrast computed tomography (E-CT) revealed no dissection of the ascending aorta; no bleeding in the thorax, including cardiac tamponade; and non-enhancement of the inferior left ventricular wall (Fig. 1B). Electrocardiogram (ECG) showed bradycardia with junctional rhythm and ST-segment elevation in inferior and precordial leads (Fig. 1C-1). Emergency CAG showed total occlusion with an irregular stump at the ostium of the RCA, and, except in segment 6 of the LAD, where there was 25% stenosis (Fig. 2A), there was no significant stenosis in the other coronary artery branches.

We diagnosed the patient with AMI caused by localized traumatic dissection in the ostium of the RCA. We decided to treat it by percutaneous coronary intervention (PCI). It was difficult to pass a 0.004-in. coronary guidewire into the true lumen of the dissected RCA. We inserted an intravascular ultrasound (IVUS) probe into the false lumen (Fig. 1D) and found an entrance to the true lumen. After the guidewire passed through the true lumen, we succeeded in revascularizing the RCA by implanting a bare-metal coronary stent (Fig. 1E). However, the patient developed shock state one hour after PCI. A second E-CT, performed during the shock state, revealed cardiac tamponade and hemothorax caused by extravasation from the epicardium (Fig. 1F) despite the first E-CT examination which performed on the patient's arrival had revealed no bleeding from the epicardium. These events were considered delayed bleeding related to the use of unfractionated heparin (8000IU i.v.) during the PCI. Emergency surgical hemostasis was performed and a critical situation was avoided. His blood pressure was in the normal range, HbA1c, low-density lipoprotein cholesterol level, and high-density lipoprotein (HDL) cholesterol level were 5.8%, 95 mg/dl, 26 mg/dl respectively without medical treatment and he had no smoking history. That is to say, he had no coronary risk factors except for low HDL cholesterol level. He continued oral administration of aspirin 100 mg/day, clopidogrel sulfate 75 mg/day, enalapril maleate 5 mg/day, and rabeprazole sodium 10 mg/day. One month later, we stopped clopidogrel sulfate. After multidisciplinary treatment for approximately two months, he was able to return to his usual daily activities.

A second CAG after 6 months was performed, there was no restenosis in the RCA. However, the 25% lesion in segment 6 of the LAD, noted on the previous CAG (Fig. 2A), had progressed to approximately 50% (Fig. 2B). One year after the BCT, he began feeling a chest squeezing sensation and shortness of breath on exertion. Fourteen months after BCT, we performed a third CAG and found 90% stenosis in segment 6 of the LAD (Fig. 2C). We therefore diagnosed exertional angina pectoris and planned PCI. We performed IVUS examination and found high echoic intimal hyperplasia around the stenotic lesion which was assumed to be fibrous plaque poor in lipid components (Fig. 2D).We succeeded the PCI with the implantation of everolimus drug-eluting stent (Fig. 2E), following which the patient's chest pain completely disappeared.

Was there an association between BCT and the patient's progressive stenotic lesion in the LAD? To answer this, we created a volume-rendering (VR) coronary image from E-CT without ECG

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