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Case Report

Acute Coronary Spasm Following Pelvic Fracture, Bleeding, and Shock in a Trauma Patient

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We report a case of a patient with severe pelvic fracture who showed concurrent ST elevation on electrocardiogram. A 70-year-old man incurred an unstable pelvic fracture from a motorcycle accident. On admission, he was hemodynamically unstable, and massive transfusion and noradrenaline were administered immediately. Although ST elevation was present in leads II, III, aV_F, V5, and V6, cardiac function was preserved; thus, trans-arterial embolization and external fixation for pelvic fracture were given priority. Four days after the injury, he suffered a cardiac arrest, and coronary angiography revealed that the cause of ST elevation and cardiac arrest was coronary vasospasm. Physicians should be aware that pain-related stress and platelet activation as well as use of noradrenaline in severe trauma cases can induce coronary vasospasm.

Key words: coronary spasm angina, noradrenaline, severe trauma, ST elevation, treatment strategy

Rapid identification of the injury site, and hemostatic treatment are critical in the treatment of severe trauma patients with unstable hemodynamic condition. Rapid diagnosis and treatment are also important for patients with acute coronary syndrome. When these conditions occur concurrently, the patient's prognosis can be affected by the treatment prioritization, for which the physicians' judgment may be the only guide. We experienced a case of concomitant pelvic fracture and ST elevation on electrocardiogram (ECG), suspected as acute coronary syndrome, and report our therapeutic strategies, physiological mechanisms of development, and outcomes, including perspectives from the literature.

Informed consent for publication was obtained from the patient.

Case Report

A 70-year-old man was injured in a collision with a car while riding his motorcycle.

His only significant medical history was prostate hypertrophy, and he was taking no daily medications. There was no mention of heart disease in his past medical history.

Pre-hospital care. Cold sweats and signs of shock were noted when the flight doctor encountered the patient. He had no complaints of chest pain, but had pelvic pain, suggesting hemorrhagic shock due to pelvic fracture. He arrived at our hospital by helicopter 1 h 20 min after the injury.

Patient condition upon hospital arrival. His airway was open, but reduced breathing sounds were observed, and SpO_2 was 95% with a 6-L oxygen mask. He had cold sweats and wet skin, his blood pressure

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was 57/41 mmHg, and his pulse was 117 beats/min. Focused assessment with sonography for trauma (FAST) showed echo free space in the left paracolic groove. Plain radiographs of the pelvis showed anterior-posterior compression-type unstable pelvic fracture. The Glasgow Coma Scale was E4V5M6.

The blood examination results are shown in Table 1. Metabolic acidosis and hypocoagulability, hyperfibrinolysis, and elevated myocardial markers were observed.

After initiating blood transfusion therapy and endotracheal intubation, contrast-enhanced computerized tomography (CT) showed a pelvic ring fracture (Fig. 1A) and splenic injury, with no extravasation of the contrast medium around the pelvis (Fig. 1B). Subsequently, an ECG revealed ST elevation in leads II, III, aVF, V5, and V6 (Fig. 2A). However, the echocardiography showed no asynergy of the wall and confirmed overall good cardiac function (Fig. 2B). Since cardiac function was preserved, the possibility of myocardial ischemia due to hemorrhagic shock was considered, and trans-arterial embolism (TAE) was prioritized for hemostasis.

TAE, pelvic external fixation. Ruptures were observed in the right iliolumbar, superior gluteal and obturator arteries; thus, occlusion of these arteries was performed. External fixation was selected for the pelvic fracture. Following TAE, the patient's ECG (five hours after the original) became normal (Fig. 2C) and the patient was admitted to the intensive care unit.

Post-admission course. Intubation and mechanical ventilation were continued. In the 24 h after injury, 16 units of red blood cells, 18 units of fresh frozen plasma, and 10 units of platelets were infused. At midnight on day 4 of hospitalization, a sudden cardiac arrest occurred. Only the P wave remained on the monitor. Return of spontaneous circulation was achieved with chest compression and 1 mg of adrenaline within 2 min. Asynergy was suspected in the inferior wall, but cardiac contractility was preserved, and ECG findings suggested recovery after ischemia. Coronary spasm angina was suspected, and nicorandil and nitroglycerin were administered. In addition, transcutaneous pacing was put on standby. Coronary angiography (CAG) was performed, and the right coronal artery #3 showed 50-75% stenosis, but it improved to 0% stenosis after coronary injection of 100 μg of

Table 1 Laboratory data on emergency room arrival

Biochemistry			Normal range
AST (U/L)	60	H	8–38
ALT (U/L)	42		4–44
γ-GTP (U/L)	47		16–73
CK (U/L)	1,084	H	56–244
CK-MB (U/L)	61.8	H	0–15
High sensitive Troponin I (pg/mL)	107.2	H	<26.2
Amylase (U/L)	117	H	41–112
Urea nitrogen (mg/dL)	18.8		8–20
Creatinine (mg/dL)	1.17	H	0.6–1.1
Sodium (mEq/L)	135		135–145
Potassium (mEq/L)	5.0	H	3.3–4.8
Chloride (mEq/L)	101		97–107
Calcium (mg/L)	8.2	L	8.8–10.2

Hematology			
WBC (/μL)	20,390	H	3,500–9,000
RBC ($\times 10^4/\mu L$)	383	L	410–550
Hemoglobin (g/dL)	12.7	L	13.4–17.4
Hematocrit (%)	38.5	L	39.8–51.8
Platelet ($\times 10^4/\mu L$)	19.7		12–35

Coagulation			Normal range
PT-INR	1.01		0.9–1.1
APTT (sec)	22.8		20–40
Fibrinogen (mg/dL)	144	L	200–400
FDP (μg/mL)	68.3	H	0–10
D-dimer (μg/mL)	31.4	H	0–1

Blood gas analysis (O2 10 L/min)			
pH	7.198	L	7.35–7.45
HCO_3 (mmol/L)	19	L	20–26
SBE (mmol/L)	−7.7	L	−3–3
pCO_2 (mmHg)	50.8	H	35–45
pO_2 (mmHg)	471	H	75–100
Lactate (mmol/L)	6.2	H	0.5–2.0

AST, aspartate aminotransferase; ALT, alanine aminotransferase; γ-GTP, γ-glutamyl transpeptidase; CK, creatine kinase; CK-MB, creatine kinase MB; PT-INR, prothrombin time international normalized ratio; APTT, activated partial thromboplastin time; FDP, fibrin degradation product; SBE, serum base excess. H, high; L, low.

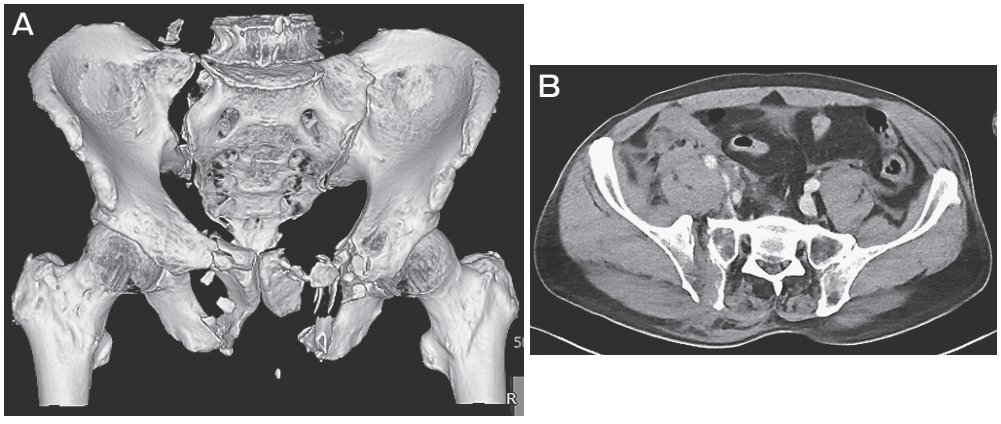

Fig. 1 Computed tomography image at the time of injury. (**A**) Right sacroiliac joint dislocation fracture and bilateral pubic fracture were revealed, and the patient was diagnosed with unstable pelvic fracture. (**B**) Hematoma formation was observed surrounding the right pelvic fracture, but extravasation was not evident.

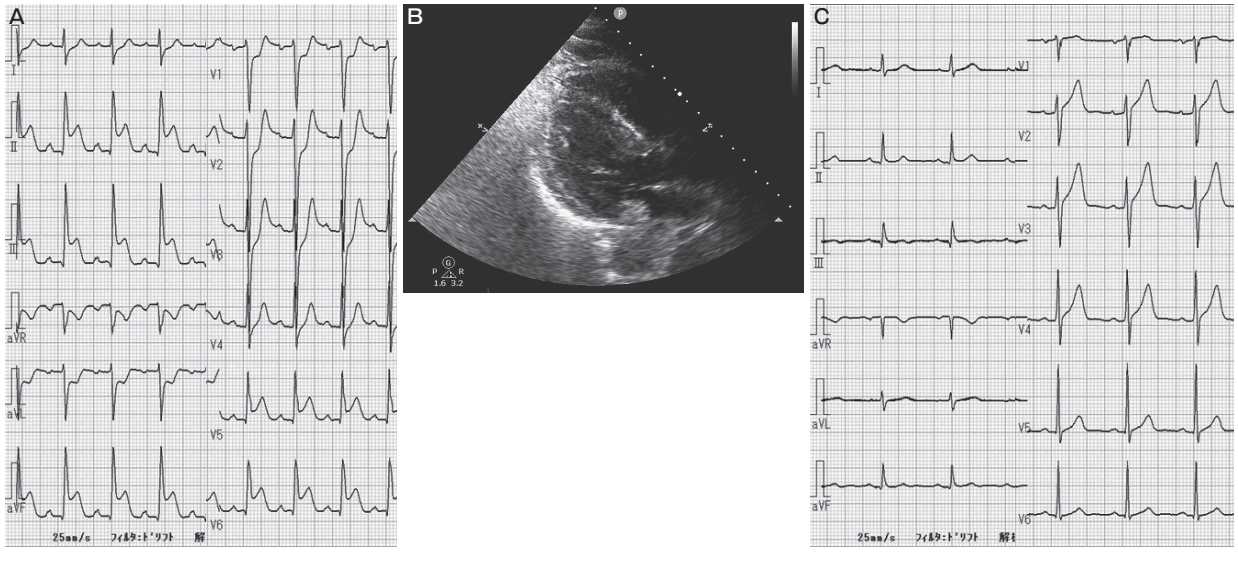

Fig. 2 Electrocardiogram changes and echocardiography. (**A**) Electrocardiogram (ECG) at the time of injury: ST elevations of II, III, aVF, V5, and V6 were observed. (**B**) No asynergy of the wall, and good cardiac function were observed. (**C**) ECG after trans-arterial embolism: Sinus rhythm with no ST elevation and any other abnormalities were observed.

nitroglycerin (Fig. 3A, B). Based on these findings, the patient was finally diagnosed with coronary spasm angina accompanying severe pelvic fracture.

On hospital day 5, internal fixation with screws and anterior spinal instruments was performed for the pelvic fracture. On day 7, heparin was administered and nicorandil, benidipine hydrochloride, and isosorbide mononitrate were started by oral administration. No angina attacks were observed thereafter. The patient recovered well and was transferred to a rehabilitation hospital on day 53.

Discussion

The prevalence of concomitant acute coronary syndromes in severe trauma patients is unclear. Among 77,745 non-cardiac surgeries in a survey study, 42 cases (9 myocardial infarction, 4 fatal arrhythmia, 29 unstable angina), or 0.054%, had cardiac events, 18 of which were coronary spastic events [1]. Although it is impos-

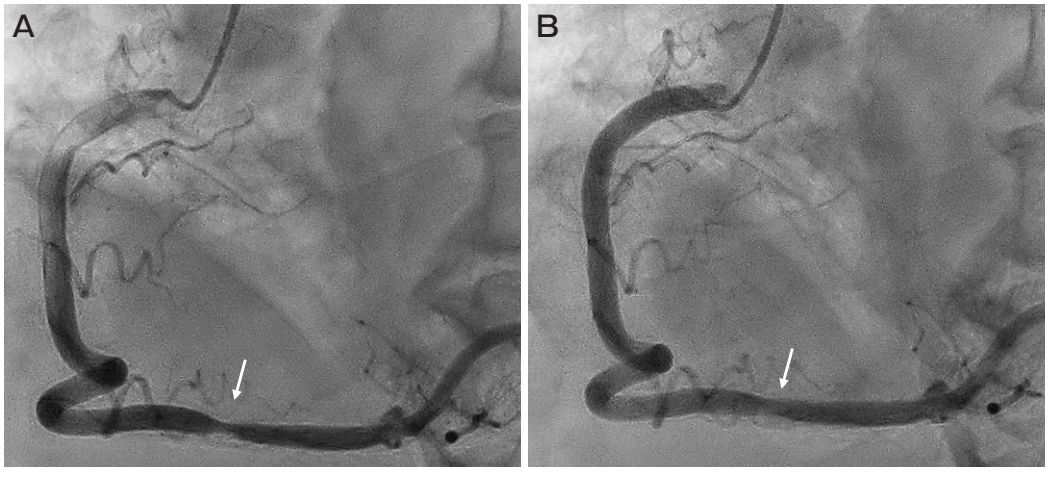

Fig. 3 Coronary angiography findings. (A) Before treatment: 50–75% stenosis in right coronal artery #3. (B) After administration of nitroglycerin 100 μg coronary injection: change to 0% stenosis.

sible to determine with certainty the frequency of their occurrence in trauma situations, cardiac events can cause severe problems in trauma patients.

There is no guideline for treating severe trauma patients with ST elevation on ECG. Serious consideration should be given based on the urgency and severity of each individual case. Joseph *et al.* [2] reported five orthopedic trauma patients with acute coronary syndrome, noting that it is important to determine whether hemodynamically unstable conditions in severe trauma patients derive from cardiac dysfunction or the trauma itself. Multidisciplinary intervention is desirable to appropriately discern and treat such cases. In this case, there were interventions from the emergency, cardiology, orthopedics and radiology departments. We concluded that the cause of the hemodynamic unstable condition was bleeding from the pelvic fracture; therefore, we prioritized hemostasis and the patient underwent TAE.

The cardiac event in this case was coronary spasm angina, but ST-elevation myocardial infarction (STEMI) and takotsubo cardiomyopathy were also included in the differential diagnosis as cardiac events associated with ST elevation [3]. There have been several reports of acute myocardial infarction associated with blunt chest trauma [4-6]. Coronary artery dissection and rupture of coronary artery plaque due to blunt chest trauma have been reported as causes. In this case, we could not diagnose STEMI or takotsubo cardiomyopathy because echocardiography showed no evidence of wall motion abnormalities such as asynergy. However, the presence of splenic injury might have suggested the possibility of blunt chest trauma, and the patency of the coronary artery should have been evaluated early.

Performing routine ECG in critical trauma care for patients with no or unknown chest symptoms is controversial. However, in critical trauma patients with ECG ST elevation, in addition to checking for pericardial fluid retention with FAST, it is also necessary to evaluate cardiac function and asynergy.

Coronary spastic angina is induced by a variety of stimuli that act on coronary artery smooth muscle receptors. In addition to stimulation due to abnormal autonomic nerve function [7], the release of vasoconstrictive hormones such as noradrenaline [8] by the sympathetic nervous system and the release of serotonin by platelet activation have the direct effects of coronary artery constriction [9,10]. In trauma patients, the sympathetic nervous system is activated due to pain-related stress, platelets activate in response to bleeding, and the action of directly administered noradrenaline, which is used to maintain blood pressure, can thus be complex, potentially resulting in the development of vasospastic angina. In this patient, noradrenaline was used for his unstable condition at primary treatment and in the intensive care unit, and it is possible that these combined effects caused coronary spasm angina. We were unable to find any report describing an association between trauma and coronary spastic angina; however, it is noteworthy that the abovementioned hyper-constrictive conditions are often all present in

severely injured patients. Given the reversibility of his coronary stenosis, it is thought that their combination might have triggered coronary spasm angina in our patient.

In the present case, the hemodynamic condition became stable after TAE on the first day, and ST elevation disappeared from subsequent ECGs. A cardiac arrest might have occurred on day 4 after the injury since the attack was observed without special therapeutic intervention. Coronary spasm attacks usually occur at rest, such as at night or in the early morning. The diurnal variation in the endothelium-dependent diastolic response to nitric oxide is thought to be a factor [11]. Although it is impossible to confirm, it is believed that the patient suffered cardiac arrest from a severe coronary spasm attack at rest during the night. It is highly possible that this could have been avoided if the cause of ST elevation had been diagnosed from CAG on the first day, and prophylactic coronary dilators had been administered. Early detection of the cause of ST elevation in patients with severe trauma is therefore critical, and routine examination of ECG for cardiac abnormalities in trauma cases can be justified on this account.

Conclusion

Echocardiographic assessment of cardiac function and asynergy proved important in the management of a case of severe pelvic trauma. Physicians should be aware that the use of noradrenaline in the context of trauma patients with pain-related stress and platelet activation may induce coronary spasm angina.

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