CASE REPORT

Haemodynamic collapse in a patient with acute inferior myocardial infarction and concomitant traumatic acute spinal cord injury

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SUMMARY

A 71-year-old man suddenly collapsed and went into cardiopulmonary arrest. The cardiopulmonary resuscitation attempt succeeded in restoration of spontaneous circulation. The initial 12-lead electrocardiogram showed inferior acute myocardial infarction (AMI). The patient was initially diagnosed as having cardiogenic shock associated with inferior AMI. In spite of early coronary revascularisation, bradycardia and hypotension were sustained. After termination of sedation and extubation, he was found to have a quadriplegia and diagnosed with a cervical spinal cord injury (SCI). Therefore, the patient was finally diagnosed with neurogenic shock caused by acute cervical SCI due to the traumatic injury preceded by loss of consciousness complicating inferior AMI. We should recognise that SCI has unique haemodynamic features that mimic those associated with inferior AMI, but requires very different treatment.

BACKGROUND

Complications of inferior acute myocardial infarction (AMI) include high-degree atrioventricular block, sinus bradycardia, hypotension and right ventricular (RV) infarction, which can cause haemodynamic collapse. These complications can be prevented or minimised by primary percutaneous coronary intervention (PCI) and are generally self-limited, but can occasionally lead to prolonged haemodynamic instability. On the other hand, cardiovascular complications are well-recognised in the acute phase of spinal cord injury (SCI) and require prompt medical attention to avoid neurological and haemodynamic compromise, morbidity and death. Especially, loss of supraspinal control of the sympathetic nervous system can cause cardiac dysfunction and haemodynamic instability in patients with a severe SCI at cervical or above the sixth thoracic (T6) vertebra.¹ The predominance of vagal activity results in severe hypotension and cardiac arrhythmia including persistent bradycardia, which are common components of the phenomena known as neurogenic shock. Thus, SCI has unique haemodynamic features that mimic those associated with inferior AMI, but requires very different treatment.

CASE PRESENTATION

A 71-year-old man required emergency medical service system because he suddenly collapsed on July 2012. His wife heard a loud noise and then

found that he was lying on his stomach on the floor with nasal bleeding, sweating and lightheadedness. He was a non-smoker and did not have a family history of ischaemic heart disease, but had been diagnosed with hypertension and is receiving antihypertensive treatment. He had experienced frequent chest pains on exertion over the past 7 days. When the ambulance crew got in contact with him, his systolic blood pressure was 70 mm Hg and heart rate was 40 bpm. In the ambulance, he went into cardiopulmonary arrest due to severe bradycardia and cardiopulmonary resuscitation (CPR) was immediately started by the ambulance crew. After arrival in our hospital, the CPR attempt succeeded in the restoration of spontaneous circulation following tracheal intubation and an intravenous administration of epinephrine and atropine. Initial arterial blood gas analysis on an FiO₂ of 1.0 revealed a pH of 7.28, PaCO₂ of 29.0 Torr, PaO₂ of 289.0 Torr and a bicarbonate level of 13.6 mmol/L. Laboratory data demonstrated serum aspartate aminotransferase (AST) 364 IU/L, alanine aminotransferase (ALT) 119 IU/L, lactate dehydrogenase (LDH) 833 IU/L, creatinase (CK) 1530 IU/L, troponin T 7.17 ng/mL, blood urea nitrogen (BUN) 27 mg/dL, creatinine 2.3 mg/dL and potassium 3.9 mEq/L. The initial 12-lead ECG after the restoration of spontaneous circulation showed ST segment changes which are compatible with the early stages of inferior AMI with complete atrioventricular block (CAVB), but there were no acute ST segment elevations in the right precordial leads (figure 1). An echocardiography immediately after resuscitation revealed preserved left ventricular (LV) function with mild inferior wall hypokinesis and normal RV systolic function without any signs of mechanical complications by conventional visual assessment. A whole body CT scan was performed, and any sign of major traumatic bleeding or non-cardiac causes of ST elevation including acute aortic dissection and subarachnoid bleeding were not detected. Therefore, he was finally diagnosed with acute inferior AMI, and intravenous heparin with 200 mg of aspirin and 300 mg of clopidogrel through nasogastric tube was administered. On the way to cardiac catheterisation laboratory, he fell into cardiac arrest followed by severe bradycardia again. Because the effects of intravenous administration of atropine were transient, temporary transvenous right ventricular pacing was immediately started. Subsequent emergent coronary

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Figure 1 A 12-lead ECG immediately after successful cardiopulmonary resuscitation.

angiography revealed a total occlusion of the mid portion of the right coronary artery proximally to the acute marginal branch (figure 2, top left). There were ectatic and 50% stenotic lesions in the proximal left anterior descending coronary artery (figure 2, top right). The patient underwent primary PCI with a bare-metal stent for right coronary artery lesion and achieved thrombolysis in myocardial infarction grade 3 angiographic flow (figure 2, bottom left).² The time to recanalisation was 3 h after symptom onset. The maximum CK level was 1993 IU/L. In spite of early coronary revascularisation, CAVB, bradycardia and hypotension were sustained. He was therefore forced to undergo critical care including sedation, mechanical ventilation, RV pacing, administration of catecholamines and large amount of fluid infusion (figure 3). Quantitative echocardiography on hospital day 2, revealed unchanged LV systolic function with ejection fraction of 51% by biplane modified Simpson's method, normal RV systolic function with fractional area change of 41% and no valvular insufficiency. In addition, pulsed Doppler-derived stroke volume was 67 mL and cardiac output was 5.4 L/min despite low-blood pressure with 66/49 mm Hg under the RV pacing, indicating low peripheral vascular resistance. These echocardiographic findings did not suggest that this patient had cardiogenic shock. On day 3, CAVB and bradycardia disappeared, but he still required catecholamines and fluid infusion for the treatment of the refractory hypotension. On day 4, he was found to have a quadriplegia after termination of sedation and extubation. Neurological examinations by a neurologist revealed severe paresis of bilateral elbow extension, bilateral finger flexion and extension and incomplete paresis of bilateral elbow flexion, wrist flexion and extension, bilateral lower limbs flexion and extension. Careful examinations of his head revealed a slight trace of a bruise on his forehead. There were no cervical spine fractures on cervical X-ray CT imaging. The cervical spine MRI revealed severe cord compression because of cervical spondylosis associated with high-signal intensity in the cord of C4 through C7 segments on T2-weighted images (figure 2, bottom right), which strongly indicates acute central cervical SCI. Therefore, he was finally diagnosed with neurogenic shock caused by acute cervical SCI due to the traumatic injury preceded by the loss of consciousness complicating inferior AMI. He required continuous catecholamine infusion for more than 2 weeks due to supine and orthostatic hypotension. He was also found to have a neurogenic bladder and refractory constipation.

Since rehabilitation was started with the use of the hard neck collar a week after the onset, the paralyses gradually improved except the bilateral upper limb movements. Follow-up echocardiography findings did not change and he was transferred to the rehabilitation hospital 4 weeks after the onset of SCI.

OUTCOME AND FOLLOW-UP

Along with a complete recovery of orthostatic hypotension within 6 weeks, he was able to stand up from the sitting position. Although he was able to walk for about 50 m without walking aids after 4 months of rehabilitation management, his motor power of the upper limb only slightly improved.

DISCUSSION

Heart block and RV infarction are the main cause of cardiogenic shock in patients with inferior AMI.³ High-degree heart block occurs in about 8% of patients with inferior AMI.⁴ The heart block can be caused by the atrioventricular node ischaemia and high-vagal tone (Bezold-Jarisch reflex). The Bezold-Jarisch reflex is caused by a marked increase in vagal afferent discharge elicited by left ventricular stimuli through chemoreceptors and mechanoreceptors. This reflex causes a bradycardia and vasodilation, resulting in lowering of the blood pressure and heart block in some patients.^{4 5} RV infarction in inferior AMI can also cause cardiogenic shock and is an independent predictor of in-hospital mortality. It is important to recognise that ST-segment elevation in right precordial leads is transient and may be absent in patients with RV infarction. These cardiac complications associated with inferior AMI can be minimised by primary PCI and are generally self-limited, but can occasionally lead to prolonged haemodynamic instability. Therefore, the patient was initially diagnosed as having cardiogenic shock associated with inferior AMI based on the clinical features. However, he required continuous catecholamine infusion longer than we expected despite successful primary PCI. Finally, he was diagnosed with acute central cervical SCI that caused prolonged bradycardia and hypotension after the termination of sedation and extubation. The autonomic nervous system is composed of the sympathetic and parasympathetic nerve, which work in opposition to each other. Sympathetic outflow is disrupted from acute cervical SCI resulting in unopposed vagal tone. This leads to the neurogenic shock, characterised by bradycardia and hypotension from decreased peripheral vascular resistance and

Figure 2 An emergent coronary angiography on the right (top left) and left coronary arteries (top right), and right coronary artery immediately after primary percutaneous coronary intervention (bottom left). The cervical spine MRI (bottom right).

cardiac output. In an epidemiological survey of SCI in Japan from January 1990 to December 1992, traffic accidents and sports accidents were more frequent causes of injury among

young people, while falls to the ground from an upper level or the same level occurred more often in elderly people.⁶ Because of high prevalence of cervical spondylosis, elderly patients will

Figure 3 Clinical course after successful cardiopulmonary resuscitation.

often incur SCI without fracture of cervical vertebrae as a result of a relatively minor traumatic event such as falls from the same level.⁷ Acute central cervical SCI was first described by Schneider *et al* in 1954 as characterised by disproportionately more impairment of the upper than the lower extremities, bladder dysfunction and varying degrees of sensory loss below the level of the lesion.⁸ In the present case, cervical spine MRI revealed severe cord compression because of cervical spondylolysis. Although prompt and accurate diagnosis was difficult because cardiopulmonary arrest and sedation after successful CPR masked quadriplegia due to SCI, a systematic retrospective review of the clinical manifestations lead to a final diagnosis of acute cervical SCI due to the minor traumatic injury preceded by loss of consciousness complicating inferior AMI.

Learning points

- ▶ Patients with acute myocardial infarction (AMI) frequently have syncope or consciousness disturbance following haemodynamic collapse, therefore, acute traumatic spinal cord injury (SCI) can occur together with AMI.
- ▶ SCI has unique haemodynamic features that mimic those associated with inferior AMI, but requires very different treatment.
- ▶ Evaluation of SCI together with invasive haemodynamic monitoring during the early hospital days should be performed in patients with prolonged and unexplained shock after AMI.

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Competing interests None.

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