A 15-year-old male patient with no previous medical history was brought to the emergency room after an assault. He was mentally confused, and his vital signs were as follows: blood pressure, 80/40 mm Hg; heart rate, 120 bpm; respiration rate, 22 breaths per minute; and body temperature, 36.5°C. The patient suffered multiple contusions all over his body with no evidence of fracture or bleeding. On auscultation, a subtle holosystolic murmur was heard at the left lower parasternal border. A chest x-ray demonstrated mild cardiomegaly, and an ECG showed sinus tachycardia with complete right bundle-branch block (Figure 1). Laboratory test results were as follows: serum creatinine phosphokinase, 5057 IU/L (normal range: 22–269 IU/L); creatinine phosphokinase-MB, 272.6 ng/mL (normal range: 0–7.2 ng/mL); troponin-I, >50 ng/mL (normal range: 0–0.3 ng/mL); serum myoglobin, >1200 ng/mL (normal range: 0–154 ng/mL); and urine myoglobin, >4000 ng/mL (normal range: 0–10 ng/mL). Transthoracic echocardiography revealed a moderate tricuspid regurgitation (TR) with a flail septal leaflet caused by chordae rupture (Figure 2 and Movie I in the online-only Data Supplement). However, no sign of heart failure or chamber enlargement was identified. Computed tomography of the chest and abdomen showed no remarkable findings. We continued monitoring for cardiac injury and treatment for traumatic rhabdomyolysis with vigorous fluid resuscitation to reduce the complications of myoglobin-associated renal failure.

Two weeks later, his general condition had gradually improved, and transthoracic echocardiography still revealed moderate TR. Notably, akinetik wall motion abnormality was newly detected at the inferior wall of the left ventricular base compared with the previous echocardiography (Figure 3A and 3B and Movie II and III in the online-only Data Supplement). To assess traumatic coronary injury, a coronary angiogram was performed, and his coronary arteries were normal (Figure 4). Because of the possibility of myocardial stunning or myocardial infarction, we planned conservative care and serial assessment of left ventricular function. Surgical correction of the flail tricuspid valve was deferred because of the patient’s good hemodynamic tolerance and the fact that he was still in the growing period. At the 2-month follow-up, although the patient remained free of symptoms, cardiac magnetic resonance imaging to evaluate myocardial viability revealed a perfusion defect and delayed enhancement along the inferior and inferoseptal wall of the left ventricular base, suggesting a transmural infarction in the right coronary artery (Figure 5A and 5B and Movie IV in the online-only Data Supplement). Over 2 years of follow-up, transthoracic echocardiography revealed akinesia at the inferior wall of the left ventricular base and a moderate TR without chamber enlargement.

The leading causes of blunt cardiac injury are motor vehicle accident and sports injury. This case introduces a traumatic TR and concomitant acute myocardial infarction caused by direct fist blows to the chest. Injury of the tricuspid valve apparatus results from acutely increased intracardiac pressure by sudden compression because the right ventricle is situated anteriorly and immediately behind the sternum. In traumatic TR management, some authors recommend earlier surgical intervention to facilitate valve repair because injured tricuspid valves become thicker and fibrotic changes occur over time. However, traumatic TR by chordal rupture and leaflet tear shows a more insidious course than papillary muscle rupture, and this patient maintained good hemodynamic tolerance during 2 years of follow-up without progression of TR and right ventricular dysfunction. Because he is still in the growth period, we chose close follow-up with serial echocardiographic assessment.

In the first stage, we overlooked the possibility of traumatic myocardial infarction because of concomitant musculoskeletal injury and rhabdomyolysis, which may also contribute to cardiac enzyme elevation and an abnormal ECG combined with electrolyte imbalance. After identifying normal coronary artery, we considered that the newly developed myocardial akinesia resulted from myocardial stunning or infarction. However, cardiac magnetic resonance imaging eventually demonstrated a transmural infarction along the territory of the right coronary artery.

The left anterior descending artery is the most commonly affected coronary artery after blunt cardiac injury. In addition, the right coronary artery is located in a vulnerable anatomic position, as well as the tricuspid valve. Although determining the precise mechanism of myocardial infarction in patients with normal coronary arteries is challenging, acute coronary spasm...
or spontaneous clot lysis after blunt chest trauma was the likely causative event of myocardial infarction in this patient.

This case emphasizes the importance of serial echocardiographic assessment early after blunt chest trauma to monitor structural cardiac injuries. In addition, increased awareness of coronary artery injury accompanying other multiple injuries is necessary for a timely diagnosis and prompt treatment to prevent permanent myocardial damage.

Disclosures

None.

References


Figure 1. ECG on admission shows sinus tachycardia with complete right bundle-branch block.

Figure 2. Transthoracic echocardiography reveals a flail septal leaflet of the tricuspid valve with moderate tricuspid regurgitation.
Figure 3. Akinetic wall motion abnormality at the inferior wall of the left ventricular base is newly detected at follow-up echocardiography (2 weeks later, B) compared with initial echocardiography (on admission, A).

Figure 4. Coronary angiogram shows normal coronary arteries.

Figure 5. Cardiac magnetic resonance imaging reveals a perfusion defect (A) and delayed enhancement (B) along the inferior and infero-septal wall of the left ventricular base, suggesting transmural infarction in the right coronary artery.
Combined Traumatic Tricuspid Regurgitation and Acute Myocardial Infarction After Fist Blows to the Chest
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Movie Legend


Movie 2. Regional left ventricular function is normal at initial echocardiography. Best viewed with Windows Media Player.

Movie 3. Follow-up echocardiography after two weeks newly detects akinetic wall motion abnormality at the inferior wall of the left ventricle base. Best viewed with Windows Media Player.

Movie 4. Cine-magnetic resonance imaging indicates akinetic inferior wall of the left ventricular base Best viewed with Windows Media Player.