Myocardial contusion injury (MCI) may occur as a rare complication of blunt thoracic trauma, presenting with possible electrocardiogram (ECG) changes, rhythm disturbances, left ventricular systolic dysfunction, or troponin elevation. We present an 8-year-old boy with MCI after a kick to the chest by a horse and describe the use of diagnostic modalities and his clinical course.

Case Report
An 8-year-old previously healthy boy presented 2 hours after he was kicked in the chest by a horse. He was awake, alert, and oriented with normal vital signs, including a heart rate of 84 bpm. Physical examination was significant for superficial abrasions over his left chest and left chin. He sustained an incomplete distal ulnar fracture. Chest radiograph and a computed tomography scan without contrast of the chest and abdomen demonstrated extensive bilateral pulmonary contusions and a small right-sided pneumothorax.

The initial ECG demonstrated bifascicular block (right bundle branch block and left anterior fascicular block; Figure 1), a well described, albeit rare, transient conduction abnormality that can be seen secondary to myocardial edema after significant MCI.1 Troponin I level at presentation was elevated at 11.2 ng/mL (normal range, 0–0.1 ng/mL), creatine kinase - MB of 58.5 ng/mL (range, 0–4 ng/mL), and creatine kinase total of 605 U/L (normal range, 21–232 U/L). Transthoracic echocardiogram showed normal intracardiac anatomy, competent intracardiac valves, no pericardial effusion, normal proximal coronary artery origins, normal biventricular systolic function, with mild left apical inferior segmental wall hypokinesia (Movie I in the Data Supplement).

He was admitted to the intensive care unit for 48 hours of telemetry. At 24 hours after admission, the left anterior fascicular block resolved but ECG continued to demonstrate right bundle branch block (Figure 2). On day 3, the right bundle branch block resolved (Figure 3). He had no arrhythmias during his hospitalization.

The troponin I peaked at 70 ng/mL at 9 hours post injury and started to downtrend but remained elevated 36 hours after injury (11.1 ng/mL). The transthoracic echocardiogram demonstrated persistent but improving left apical inferoseptal hypokinesia.

Coronary angiography was performed and demonstrated mild thinning and hypokinesia of the apical septal segment of the left ventricle. The hypokinesia corresponded to an area of abnormal, increased enhancement in the subendocardial region on T2-weighted turbo spin echo images (Figure 4), signifying acute myocardial injury and edema. First-pass perfusion imaging was performed after contrast injection (0.2 mL/kg of gadoteridol) and did not show any perfusion defects at rest. Images using a phase-sensitive inversion recovery sequence were obtained ≈6 minutes after contrast and demonstrated abnormal myocardial hyperenhancement in the same location (Figure 5). Given CMR findings consistent with myocardial changes secondary to coronary ischemia, a coronary angiogram was performed, which showed unobstructed filling of his coronaries to the apex (Figure 6). It is possible the CMR changes were secondary to transient coronary vasospasm.

Discussion
Myocardial injury from chest trauma has been described in the pediatric literature. However, because of its rare occurrence in this age group, the medical management has not been standardized. Abnormal troponin levels and ECG on admission in patients with chest trauma can be indicative of MCI and warrant further evaluation,2 which in our patient included serial troponin and ECGs, as well as telemetry given high risk of arrhythmias.3 Although normal ECG and troponin levels at admission and 4 hours have a high negative predictive value in ruling out myocardial injury, troponin I is abnormal at presentation in a majority of patients with significant MCI2 and levels peak sooner compared with myocardial ischemia.3

Importantly, although rare, MCI can cause coronary artery injury and peritraumatic myocardial infarction, even in the absence of underlying coronary artery disease. The cause can be multifactorial, including parietal hematoma and coronary artery dissection, although in some cases it may remain unknown.4 Its rarity and variation in cause can make this diagnosis difficult. In this setting, CMR has been useful in differentiation of peritraumatic myocardial infarct from nonischemic myocardial contusion by evaluating for the presence of delayed enhancement in

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From the Division of Cardiology, Department of Pediatrics, UT Southwestern Medical Center at Dallas, and Children’s Medical Center of Dallas, TX.
Correspondence to Poonam Punjwani Thankavel, MD, Division of Pediatric Cardiology UT Southwestern Medical Center, Children’s Medical Center of Dallas, 1935 Medical District Dr, Dallas, TX 75335. E-mail poonam.punjwani@childrens.com
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the subendocardial region, which is consistent with coronary ischemia. When this pattern is visualized, coronary angiography may be necessary to exclude coronary injury.

Our patient was discharged on day 4 and restricted from strenuous physical activity. At 1-month follow-up, echocardiogram, ECG, and troponin levels were normal. He was cleared for recreational activities, but restricted from organized sports, including karate. A 6-month follow-up is scheduled with repeat echocardiogram, ECG, and an exercise test. Follow-up CMR is planned for 1 year post injury to assess for residual myocardial changes.

Disclosures

None.

References


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Figure 1. Admission 12-lead ECG demonstrating right bundle branch block and left anterior fascicular block as evidenced by the rsr' in V1, prolongation of QRS duration, and left-axis deviation.

Figure 2. Twelve-lead ECG on day after admission shows resolution of left anterior fascicular block. Right bundle branch block is still noted.
Figure 3. Twelve-lead ECG on third day of admission shows resolution of right bundle branch block.
Figure 4. Cardiac MRI (CMR) in long-axis planes. T2-weighted scan with enhanced signal intensity (arrows) in apical septal region shows myocardial edema.
Figure 5. The arrows correspond to areas of late gadolinium enhancement in the subendocardial region of the septum at the apex in the short-axis (A) and long-axis images (B).

Figure 6. Coronary angiogram demonstrates normal coronary artery filling.
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Danielle M. Moyé, Adrian K. Dyer and Poonam P. Thankavel

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SUPPLEMENTAL MATERIAL

Video 1: Parasternal short axis video on echocardiogram shows hypokinesia of inferoseptal segment.