Commotio cordis and ventricular pseudoaneurysm

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A case of an eight-year-old boy who experienced commotio cordis with the development of myocardial infarction and a ventricular pseudoaneurysm is described. Progressive enlargement of the aneurysm resulted in distortion and compression of the overlying coronary arteries, causing myocardial ischemia.

Key Words: Aneurysm; Arrhythmia; Myocardial infarction; Pediatrics; Ventricles

The present report describes the case of an eight-year-old boy who experienced commotio cordis with the development of myocardial infarction and a ventricular pseudoaneurysm. Progressive enlargement of the aneurysm resulted in distortion and compression of the overlying coronary arteries, causing myocardial ischemia. The patient's family gave consent for the publication of the present report.

CASE PRESENTATION

A previously healthy eight-year-old boy was playing on an indoor soccer field when a goal net fell on him. The net was of lightweight construction but had the added weight of several other children when it struck him over the left precordium. He reported feeling palpitations before losing consciousness. He recovered spontaneously 20 s to 30 s later and was able to walk home. He had no noticeable bruising or tenderness. Three hours after the injury, he developed chest and abdominal pain, vomiting and diaphoresis. At the local emergency department, electrocardiogram (ECG) changes and a dramatic rise in troponin levels to 75 µg/L (levels lower than 0.5 µg/L indicate an unlikelihood of myocardial infarction) led to the diagnosis of anterior myocardial infarction. Echocardiography revealed akinesis of the anterior ventricular septum with a left ventricular (LV) ejection fraction (LVEF) of 28%. He spent 17 days in the hospital on bed rest, and received atenolol and lisinopril. He underwent exercise testing before he was discharged and tolerated 7 min of a standard Bruce exercise protocol without symptoms or ECG changes. His LVEF at the time of discharge was 48%, and the anteroapical region of the LV was still dyskinetic but not overtly aneurysmal.

Three days following discharge, the patient developed chest pain at rest. The initial examination revealed that the patient was alert and not distressed. His heart rate was 88 beats/min and regular, with a blood pressure of 100/50 mmHg, a respiratory rate of 25 breaths/min, an oxygen saturation of 99% in room air, normal pulses and perfusion, an abnormal systolic impulse palpable in the third left intercostal space, normal heart sounds with no murmurs or rubs, normal breath sounds and no hepatosplenomegaly. His ECG showed sinus rhythm with T wave inversion in lead I and ST segment depression in leads V3 to V6. Echocardiography revealed anteroapical LV dyskinesis and an LVEF of 25%. There was no pericardial fluid. His troponin T level was elevated at 0.6 µg/L. He was admitted to the intensive care unit for observation.

The following day he underwent cardiac catheterization and angiography, which revealed an aneurysm of the anterior wall of the LV (Figure 1). The aneurysm was distorting the midportion of the left anterior descending coronary artery and first diagonal branch, resulting in compression of long segments of these vessels during ventricular

Une commotion cérébrale et un pseudoanévrisme ventriculaire

On décrit le cas d'un garçon de huit ans qui a souffert d'une commotion cérébrale accompagnée d'un infarctus du myocarde et d'un pseudoanévrisme ventriculaire. L'hypertrophie progressive de l'anévrisme a provoqué une distorsion et une compression des artères coronaires sus-jacentes, responsables d'une ischémie myocardique.

Figure 1) Left ventriculogram profiling a pseudoaneurysm of the anterior wall

systole (Figures 2 and 3). He was transferred to the regional centre for pediatric cardiac surgery.

Intraoperatively, a pseudoaneurysm was identified under the left anterior descending artery (Figure 4). The neck of the aneurysm measured 2 cm × 3 cm and bordered the LV outflow tract in the region of the aortomitral continuity. It was repaired with a dacron patch. Histology of a segment of the aneurysm wall demonstrated fibrous tissue with no myocardial elements, confirming the diagnosis of a pseudoaneurysm.

His postoperative course was uneventful. A technetium-99m gated wall motion study performed eight days postoperatively showed that a substantial region of the anteroapical LV was noncontractile, corresponding to the location of the patch. The LVEF was 29%.

The echocardiogram eight days postoperatively revealed similar wall motion abnormalities, a dilated LV and an LVEF of 44%. His cardiac examination findings at discharge were normal.

DISCUSSION

Acquired ventricular aneurysms and pseudoaneurysms are rare phenomena with an estimated prevalence of 0.05% (1). Causes include coronary

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Figure 2) Selective left coronary angiogram in diastole showing some attenuation of the left anterior descending coronary artery and the first diagonal branch

Figure 3) Selective left coronary angiogram in systole showing complete compression of the left anterior descending coronary artery and the first diagonal branch by the expansion of the pseudoaneurysm

artery disease with transmural myocardial infarction (particularly in the adult population), previous cardiac surgery and endocarditis with abscess. Trauma, both penetrating and blunt, accounts for up to 7% of cases (2,3). Blunt chest trauma may cause myocardial damage by a number of means including the direct effect of the impact on the myocardium, and by compression, thrombosis, spasm or dissection of the coronary arteries (4). The damage may be exacerbated by hypoperfusion or hypoxia in the setting of multiple traumas. Arrhythmia-induced hypoperfusion that occurs with commotio cordis, as described in the present case, may also exacerbate the myocardial damage.

There is, not uncommonly, a delay in the diagnosis of aneurysm formation, and patients may present with sudden death weeks to months after their initial injury due to rupture of a previously undiagnosed aneurysm (5). Even cardiac ventriculography may not show aneurysm formation in the early post-trauma phase, suggesting that these defects are an evolving pathological process involving ongoing myocardial damage, the application of forces to weakened tissues or both over time. Magnetic resonance imaging, computed tomography

Figure 4) Intraoperative photograph showing the left anterior descending coronary artery coursing over the surface of the pseudoaneurysm. Photograph courtesy of Dr C Soder (IWK Health Centre, Halifax, Nova Scotia)

and angiography may unveil the presence of aneurysms (1,2), although currently, most are diagnosed by transthoracic echocardiography. This modality has significant limitations in terms of user dependence, echocardiographic windows and patient compliance. Even with close clinical monitoring and repeated echocardiographic surveillance, aneurysm formation may be overlooked; this may have been a factor in the present case.

More likely, the rapid development of the pseudoaneurysm may explain the patient's presentation with a large aneurysm less than one week after echocardiography showed only dyskinesia but no aneurysm. It is this rapid enlargement that may explain the high risk for rupture of aneurysms once they develop and provides support for the current recommendation for surgical repair of these defects (2,3).

Ventricular pseudoaneurysms are associated with a mortality rate approaching 50%. This is primarily due to rupture but may also be due to thromboembolism or ventricular arrhythmia (2). Compression of adjacent structures may also be problematic. This was seen in the present case, in which coronary artery compression led to the development of symptoms of myocardial ischemia.

CONCLUSION

The present case of a large ventricular pseudoaneurysm forming weeks after blunt chest injury provides a cautionary tale for those managing pediatric myocardial trauma and infarction. Pseudoaneurysms can develop rapidly. Even close follow-up of cases known to be at risk for the development of ventricular aneurysm and rupture may miss this rapidly developing complication. Tremendous diligence to reassessment, consideration of alternative imaging modalities and a very high index of suspicion are warranted over many weeks. It may also be said that sporting nets are not best suited for use as climbing frames!

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