Abstract:
We report on a 9-year-old healthy boy with a left main coronary artery arising from the right sinus of Valsalva coursing between the aorta and pulmonary artery who experienced a myocardial infarction after a syncopal episode during exercise. The high risk of sudden death associated with this anomaly during or immediately after vigorous activity makes immediate diagnosis and surgical intervention paramount. Diagnosis was made by transthoracic echocardiogram and confirmed by autopsy.

Keywords:
Coronary artery anomaly; echocardiography; myocardial infarction; left main coronary artery

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Exertional Syncope and a Congenital Cardiac Anomaly

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A 9-year-old previously healthy Hispanic boy presents after experiencing a witnessed syncopal episode lasting several minutes while running in gym class. He reports feeling lightheaded before the episode and denies any prior syncope. He had 3 episodes of nonbloody/nonbilious emesis after the event but no diarrhea or fever. Two days prior he developed generalized malaise, nausea, and anorexia, although he felt well enough to go to school. He reports chest pain after the syncope but not before the episode. The patient's mother has not noticed increased work of breathing, shortness of breath, palpitations, orthopnea, cyanosis, cough, or congestion. There are no ill contacts.

He was first taken to a community hospital emergency department (ED) by prehospital providers, where he was noted to be in respiratory distress with blood pressures in the 80s/50s. His oxygen saturation was in the 70s on room air, and he was placed on supplemental oxygen via a nonrebreather face mask (NRBFM). A chest radiograph was concerning for bilateral infiltrates. He received a dose of ceftriaxone and 3 intravenous fluid boluses of 0.9% saline before transfer to our facility for further care. En route, he had mild respiratory discomfort with the NRBFM and was less interactive, but his vital signs were within normal limits for age.

Review of systems is negative except for shortness of breath, nonspecific abdominal pain, and headaches on and off for the last 2 days, frontal in location and pounding in nature. His medical history is significant for a febrile seizure at 1 year of age. His mother denies prior surgeries. His birth and developmental histories are noncontributory. His immunizations are up to date by report, and he has no known allergies to medications. He lives with his mother, her fiancée, and 3 siblings. He is in the third

grade and gets mostly As. He enjoys playing sports and has many friends at school. He denies drug use, smoking or alcohol, and sexual activity. There is no family history of sudden death, drowning, or single car accidents.

On physical examination, his temperature is 36.8°C, pulse is 147 beats per minute, respiratory rate is 30 breaths per minute, and blood pressure is 88/60 mm Hg. His oxygen saturation is 99% on an NRBFM, and he weighs 28 kg. He is awake and anxious with his eyes closed, responding appropriately and following commands. His head is normocephalic and atraumatic, and his extraocular movements are intact. His pupils are equal, round, and reactive to light. His conjunctivae are clear, and there is no discharge. His oropharynx is clear, lips are pale, and he has moist mucus membranes. His neck is supple, and there is no meningismus. He has decreased breath sounds at the left lower lobe with fine crackles at both bases. He has intercostal retractions with no Kussmaul respirations. He is tachycardic, and no murmurs, gallops, or rubs are appreciated. Peripheral and central pulses are strong. His capillary refill time is less than 2 seconds. His abdomen is soft, nondistended, but tender to palpation in both the suprapubic and left lower quadrants. His liver edge is palpated 3 cm below the right costal margin. He exhibits no guarding and no peritoneal signs. He has no focal neurologic deficits and normal tone and strength. He is Tanner stage I. There is no clubbing, cyanosis, or edema. He has full range of motion in all extremities. His skin has no rashes or lesions.

Laboratory workup in the ED includes a venous blood gas with electrolytes, lactate, hemoglobin, and coagulation studies. These are unremarkable except for a slight acidosis, with a pH of 7.27 with base excess of minus 7. An electrocardiogram (ECG) and a chest radiograph are also performed. The ECG shows ST depression in leads II, III and aVF and ST elevation in leads V_3 and V_4. The radiograph demonstrates diffuse bilateral parenchymal and interstitial opacities with relative sparing of the costophrenic angles most consistent with pulmonary edema (Figures 1 and 2).

The patient is triaged to a major resuscitation room. He has progressively worsening respiratory distress and is placed on Bilevel positive airway pressure (BiPAP) and started on furosemide. Cardiology is consulted, and a transthoracic echocardiogram shows decreased shortening fraction of 10% to 15% (normal 36%) and ejection fraction of 23% to 33% (normal 66%), severe tricuspid regurgitation, and mitral regurgitation, with moderately decreased biventricular function worse at the left ventricular apex. The patient is admitted to the cardiac intensive care unit (CICU) for further management where the diagnosis is made on hospital day 2.

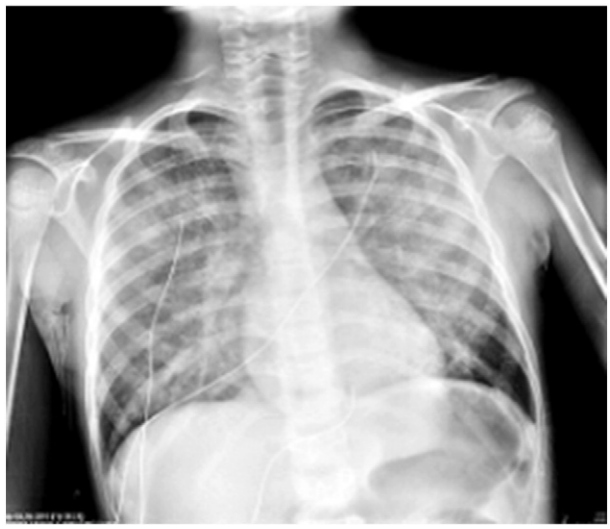

Figure 1. First chest xray of patient on admission to the ED.

DIFFERENTIAL DIAGNOSIS

Syncope is defined as a sudden, brief loss of consciousness associated with loss of postural tone from which recovery is spontaneous.[1] Approximately 15 to 25% of children and adolescents will experience syncope in their lifetime.[2] It accounts for 1% of hospital admissions and 3% of ED visits.[3] Most causes are benign with more than 70% attributed to vasodepressor syncope. Cardiac syncope accounts for 2 to 6% of pediatric cases and can be associated with an increased risk of death.[3] Albeit difficult, the goal of the emergency care provider is to appropriately select out those particular individuals with life-threatening etiologies among the larger collective of patients presenting with benign syncope[21]. These include conditions such as brain tumors, arrhythmias, or toxins (eg, carbon monoxide poisoning) (Table 1).

A detailed history, physical, and a thorough evaluation of an ECG is key in distinguishing benign vasovagal syncope and syncope caused by underlying lethal cardiac etiologies. In a retrospective study of almost 500 children diagnosed with syncope, 21 of 22 with a cardiac etiology were correctly identified by a combination of history, physical examination, and ECG.[4] Historical features to consider are the child's position and the activity during the episode. Syncope with prolonged upright posture or after exercise can be associated with vasovagal etiologies, whereas syncope during exercise ("mid-stride") is of cardiac etiology until proven otherwise. A trigger such as an

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