CASE REPORT

Hyperinsulinism in a child presenting with cardiac ischemia and bradycardia

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A 5-year-old boy referred to our service with suspected sinus node dysfunction. In addition to the arrhythmia, he had moderate mitral valve regurgitation and depressed ventricular function during a hypoglycemic episode. Cardiac abnormalities resolved with glucose infusion. We believe that hypoglycemia was responsible for the cardiac manifestations and it should be considered in unexplained rhythm disturbances or ischemia.

Keywords: Hypoglycemia, Bradycardia, Myocardial ischemia

A five-year-old boy was referred to our cardiology service with suspected sinus node dysfunction. His symptoms started five months prior with episodic attacks of palpitation, abdominal pain, chest discomfort, nausea, cold extremities, sweating, and altered level of consciousness. During evaluation in the referring hospital, these symptoms were associated with sinus tachycardia followed by severe sinus bradycardia. There was no significant past medical history, and no history of medication ingestion, or remarkable family history of similar problems. Physical examination was unremarkable with normal growth parameters.

A 24-h ambulatory ECG recording was placed at the initial evaluation. During monitoring, the patient developed a typical episode and presented to the emergency room with an altered level of consciousness. Severe sinus bradycardia (45 beats/min) on the ambulatory ECG coincided with the episode onset followed by sinus tachycardia and 7 mm ST segment depression on the rhythm strip (Fig. 1). His blood pressure at the time was 100/70 mmHg. Blood sugar and cardiac enzyme levels were not obtained. Echocardiogram in the emergency room revealed the presence of moderate mitral valve regurgitation and depressed ventricular function with an ejection fraction of 24% (Fig. 2, panel A). Serum glucose was not measured at presentation; however, after initial management with IV fluid and glucose, the heart rate returned to normal, the patient regained consciousness, and this coincided with the ST segment having normalized on the ambulatory ECG monitoring, which was analyzed later. The patient was admitted to the intensive care unit for observation and, due to the severe ST segment changes observed in the 24-h ECG recording, coronary angiograms were performed, excluding coronary anomalies. Blood sugar and cardiac enzyme levels were both normal 12 h after

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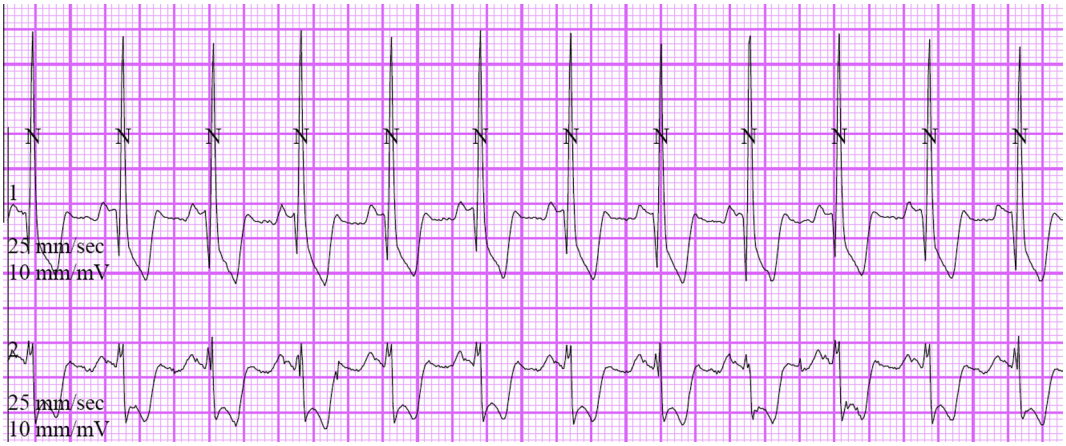

Figure 1. ST segment depression associated with sinus tachycardia during a hypoglycemic episode recorded on a 24-h ambulatory ECG.

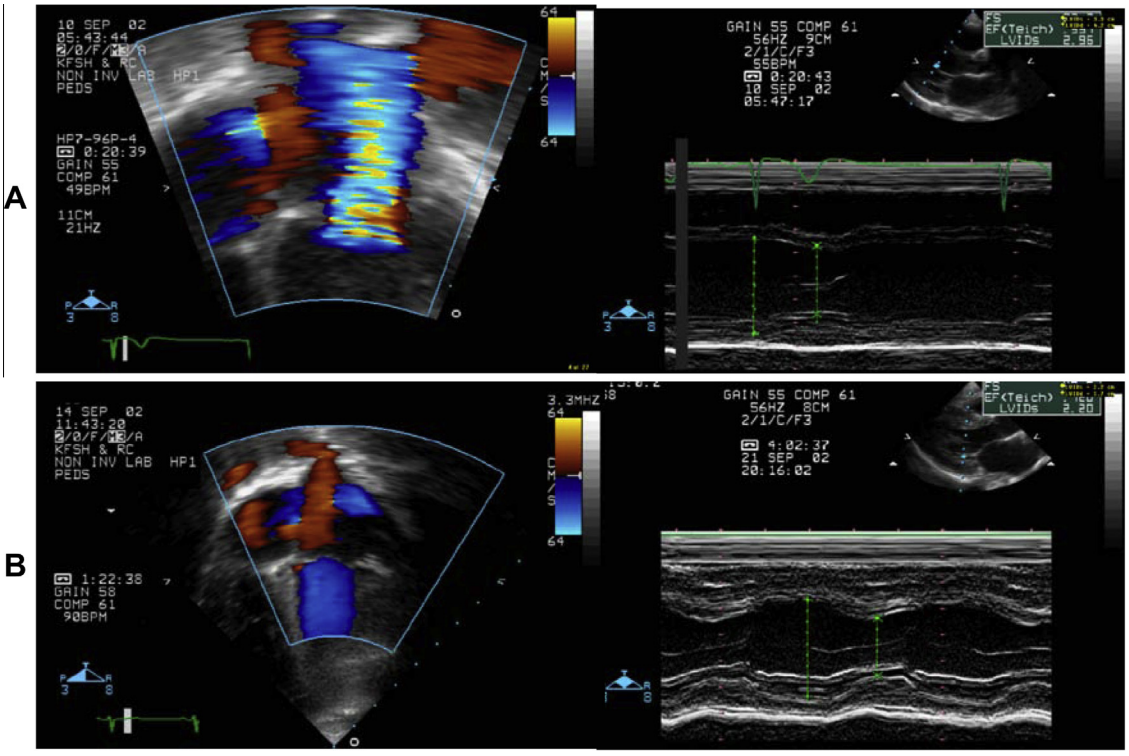

Figure 2. Color flow Doppler study across the mitral valve and M-mode at presentation (panel A) showing moderate mitral regurgitation and ejection fraction of 24%, and after correction of hypoglycemia (panel B) showing competent mitral valve and normal ventricular function with ejection fraction of 72%.

presentation. A repeat echocardiogram a few days after admission showed normal cardiac function with improved mitral regurgitation (Fig. 2, panel B). While in the hospital under monitoring, the patient developed another episode with the same ECG changes and bradycardia. Based on the symptoms and previous response to IV fluids, hypoglycemia was suspected as an etiology for the presenting symptoms. During the episode, a blood sample obtained showed hypoglycemia of 1.3 mmol/L (critically low random glucose <2.2 mmol/L) with elevated random insulin level (166 pmol/L). A dose of glucagon administered intravenously during the episode corrected the blood glucose level, alleviated the patient's symptoms and reversed the electrocardiogram changes. This confirmed the diagnosis of hyperinsulinemia, and the patient was started on diazoxide prior to discharge. While on therapy the patient became asymptomatic. The child was followed as a case of persistent hyperinsulinemic hypoglycemia of infancy (PHHI, formerly called nesidioblastosis) based on