A Case of Syncope with Elevated C-Peptide Levels in a 22-Year Old Male: A Case Report

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Abstract

There are multiple causes of syncope, which is a short loss of consciousness and muscle strength, characterized by a fast onset, short duration, and spontaneous recovery. The exact cause of syncope in young adults is usually difficult to figure out. This case report describes syncope in a young adult with multiple intermittent high C-peptide levels upon testing.

Keywords: Syncope; Muscle strength; Down’s syndrome

Case Report

A 22 year old white male with Down’s syndrome presented to a family medicine clinic with increased intermittent episodes of syncope. These episodes lasted approximately 3-5 minutes and the patient awoke without recall. The patient presented with two main complaints, seizures and syncope frequently over the last two years. The syncope and seizure occurs simultaneously (Figure 1). The seizures were consistent with epilepsy and he was given Keppra 750 milligrams an anti-convulsant that controlled these symptoms initially. However, the syncope episode came back again. Laboratory evaluations showed an elevated C-Peptide 3.25 to 12.20 ng/mL over the last two years (0.8-3.1 normal range) with each episode of syncope, Insulin level 9 uIU/mL (<23 normal range), Insulin autoantibody <0.4 U/mL (<0.4 normal range), hemoglobin A1c 5.7 (<5.7 normal range), Prolactin levels 5.0 ng/mL (<23 normal range), Calcium 9.6 mg/dL (8.6-10.3 normal range), Parathyroid hormone, intact 24 pg/mL (14-64 normal range) and his CBC and CMP were also within normal range. The cardiology evaluation was unremarkable.

In detail the Syncope is multifactorial and requires a broad approach to diagnosis which involves taking an in depth history, reviewing medications, a thorough physical exam, blood pressure measurement, electrocardiogram and neurological exam. A syncope protocol was used to determine if hospitalization is required. Further testing was done due to the etiology being unclear. Cardiology evaluation revealed a systolic ejection murmur II/VI and normal LV systolic function with no significant valvular abnormalities by prior Transthoracic Echocardiogram (TTE) Electrocardiogram shows a normal sinus rhythm, his heart rate was 79 beats per minute and RSR in V1. Magnetic Resonance imaging (MRI) of the brain in multiple planes was performed. The study demonstrated no evidence of mass or mass-effect (Figure 2). The ventricular system is normal and there is no evidence of extra-axial fluid. There are no areas of abnormally increased or decreased signal intensity nor any abnormally enhancing lesions that were identified. The midline structures are unremarkable ruling out neurological causes. Computed Tomography (CT) of the abdomen with and without contrast was performed using 5mm helical images were made from the dome of the diaphragm to the aortic bifurcation following oral contrast administration. Resultant images were viewed on soft tissue window settings. The lung bases are clear. The liver, spleen, pancreas, gallbladder, adrenal glands and kidneys are unremarkable. There is no intra-abdominal or retroperitoneal adenopathy. Opacified bowel loops in the upper abdomen are unremarkable. There is no ascites. There is no evidence of an abdominal wall hernia revealing an unremarkable CT of the abdomen. C-Peptide measurements were elevated greater than 3.1 ng/mL (0.80-3.1 normal range) with each episode of syncope and normal laboratory levels of glucose, calcium, parathyroid hormone, insulin level, prolactin level and a complete blood count.

Insulin and C-Peptide are secreted in response to all insulin secretagogues. Although proinsulin may have some mild hypoglycemic action, C-peptide has no physiological function. Originally, the unit

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insulinoma, an insulin secreting tumor of the islets of Langerhans even with a negative Brain MRI and abdominal Computerized Tomography Scan. These tumors can appear at any age and generally have symptoms of episodic disorientation, somnolence, personality changes, amnesia and loss of consciousness due to the production of excessive insulin resulting with a hypoglycemia event. The hypoglycemia induced intracellular signal change, which eventually triggered these seizure episodes. This patients’ syncope was due the combination of hypoglycemia and subsequent seizure due to a recurrent central nervous system dysfunction. This patients symptoms are still unresolved and has been referred to Endocrinology for further evaluation of his continued hypoglycemia, and elevated C-peptide levels for a probable insulinoma. Patient was also referral to neurology for management of his seizure disorder.

References