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Recurrent Syncopal Episode and Undetectable Vitamin B12

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Abstract Syncope is a common cause of hospital admissions, with a high prevalence found in people 10-30 years of age, and in those 70 years and older. The condition often requires an extensive yet unrevealing workup. Vitamin B12 deficiency, however, is rarely thought to be the primary root of recurrent syncope. We had a case of vitamin B12 deficiency without findings of anemia, which presented as a recurrent syncope in an otherwise relatively young healthy man.

Keywords: recurrent syncopal episode, undetectable vitamin B12

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1. Case Report

A previously healthy 45 year old man presented to the emergency department with dizziness that resulted in an unwitnessed fall with loss of consciousness, without remembering the sequence of events, consistent with a syncopal episode. He admitted to the presence of lightheadedness prior to passing out. On admission his blood pressure (BP) measured when he was supine ranges from 140/80 to 190/110. However he was positive for orthostasis with BP sitting 160/98 and standing 131/90, pulse sitting 106 and standing 118. He appeared euvolemic with moist mucous membrane.

Neurological examination was unremarkable. His tachycardia prompted initiation of treatment with losartan 50 mg and metoprolol tartrate 12.5 mg. Labs revealed no significant findings except for an elevated MCV (100.7) without anemia. He reported moderate alcohol use of 2-3 12-ounce glasses of beer, 3-4 times a week and denied any restrictive diet. Electrocardiogram and telemetry monitoring were unremarkable and echocardiogram revealed no structural abnormalities except for borderline left ventricular wall thickness and a diastolic filling pattern. The patient was then discharged home.

He returned after two days with a witnessed syncopal episode. Orthostasis was again noted to be positive with BP sitting 135/91 and standing 107/83, pulse sitting 118 and standing 136. Neurological examination was unremarkable with intact cranial nerves, with grossly intact motoric and sensory functions, heel-to-shin and finger-to-nose testing were normal and deep tendon reflex +2 bilaterally. Antihypertensives were held due to

concerns about the potential for treating his BP too aggressively. A workup of his orthostatic hypotension (OH) was initiated. Plasma renin activity, total catecholamine, epinephrine, norepinephrine, urine total metanephrines, normetanephrine, 24-hours metanephrines, TSH and morning cortisol were within normal limits. Elevated MCV was again noted with a Hgb of 13 and high RDW of 14.7. Vitamin B12 level was undetectable (< 159 pg/ml). He was given intramuscular cobalamin injection followed by oral supplementation and was sent home. On outpatient follow up 1 month later, he reported resolved symptoms. Repeat orthostatic BP in the office was sitting 156/100 and standing 142/94.

2. Discussion

Orthostatic hypotension (OH) is one of the common causes of syncope, defined as a drop of systolic BP of 20 mmHg or diastolic BP of 10 mmHg within 3 minutes of standing compared with sitting or supine position. It commonly affects the elderly and the frail (as high as 20%), however in younger patients without volume depletion, the most common cause is chronic autonomic failure. [1] In this case, the presence of inappropriate tachycardia and hypertension with OH raised a possibility of autonomic dysfunction involvement. There are many common identifiable causes of OH, including volume depletion, drugs and alcohol, endocrine abnormalities (adrenal insufficiency, hypothyroidism, hypoaldosteronism, pheochromocytoma), or cardiovascular conditions (anemia, heart failure, arrhythmia, valvular abnormalities) which often require an extensive and costly workup. [1] Sometimes amidst the rigorous workups, clinicians may overlook the obvious, as in our patient who had

macrocytosis, prompting workup for vitamin B12 levels and diagnosis of deficiency.

Vitamin B12 deficiency usually presents with hematological (macrocytic and megaloblastic anemia), neurological or psychiatric manifestations (dementia, Neurologic manifestation is due to depression). demyelination of neurons of the central nervous system (CNS). The most widely known parts that are affected are the dorsal column and corticospinal tract. This leads to subacute combined degeneration, resulting in abnormal vibratory sensation, symmetric paresthesias, peripheral neuropathy, numbness and gait problems/ataxia. [2] With the potential to affect any part of the CNS, it has been reported in several studies that vitamin B12 deficiency could cause hemodynamic and autonomic abnormalities, likely secondary to the involvement of small sympathetic postganglionic fibers that supply peripheral resistance vessels and the degeneration of sympathetic unmyelinated fibers. [3] There is also a case series of refractory orthostatic hypotension which presented with minimal symptoms of vitamin B12 deficiency and had rapid improvement of symptoms after vitamin B12 repletion. [4]

This case highlights the fact that vitamin B12 deficiency may manifest without the finding of anemia, with macrocytosis being the only clue. It also may be overlooked as a cause of orthostatic hypotension and recurrent syncope in otherwise young, healthy patients.

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