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Waldenstrom's Macroglobulinema Presenting as Syncope

Ramy Sedhom[1]

[1] Rutgers Robert Wood Johnson Medical School

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Abstract

Syncope is a common complaint usually secondary to neurologic, cardiovascular, or orthostatic causes. However, rare etiologies are possible, implicating a great importance to history, physical examination, and interpretation of laboratory results and diagnostic workup. Waldenstrom?s Macroglobulinema (WM) is a B-cell Lymphoma, hallmarked by an overproduction of IgM. Neurologic manifestations of WM include visual or auditory disturbances, headache, confusion, dizziness, vertigo, stroke and rarely, syncope. Neurologic presentations are a result of hyperviscosity or direct infiltration of malignant cells into the CNS. We present a case of Waldenstrom?s Macroglublinema associated syncope.

Index terms—

1 I. Introduction

yncope is a common presenting problem evaluated by clinicians in internal medicine. Syncope associated hospitalizations have a high cost on our health care system. Neurally mediated syncope is implicated in the majority of cases, followed by cardiovascular disease and orthostatic hypotension. The work-up for syncope is often not helpful. The importance of a good history cannot be overstated, as it often leads the clinician to the most probable diagnosis. Waldenstrom's Macroglobulinemia is a lymphoplasmacytic malignancy that secretes IgM. Patients with WM may have neurologic manifestations either from hyperviscosity or related to direct infiltration of lymphoplasmacytic cells into the CSF. This is well described as the Bing Neel syndrome. Neurologic manifestations include visual and auditory disturbances, headache, confusion, dizziness, vertigo, and rarely syncope or stroke.

2 II. Case Report

A 71-year-old man presented with syncope. For 3 months, he was evaluated several times by his primary care provider, and in urgent care facilities, for episodes of both syncope and pre-syncope. Review of symptoms was positive for fatigue, nausea, visual floaters, and hearing loss. He reported several falls in the past six months. He reported that symptoms prior to his falls were sudden, without a preceding prodrome. He felt unsteady with positional changes. He denied vertigo or dizziness. He was prescribed meclizine, by several providers, without any benefit. On the day of admission, he reported a near syncopal event while watching a football game. He sustained no injuries from his fall. His grandson, who witnessed it, denied any jerking or twitching movements. The patient's grandson denied any confusion and the patient was lucid immediately following the event. During his prior episodes, he was not urinating, defecating, or straining. He denied palpitations, diaphoresis, or chest pain. He reported a 40-pound weight loss in the six months preceding this current hospitalization. Several months prior, he suffered a lower gastrointestinal bleed. He was not on any blood thinners and denied taking ibuprofein or over the counter medications. A colonoscopy during that hospitalization demonstrated internal hemorrhoids. He had no evidence of malignancy on either upper or lower endoscopy. Past medical history included diabetes mellitus, hypertension, hyperlipidemia, chronic kidney disease, and iron deficiency anemia. His home medications included lisinopril, metformin, iron tablets, atorvastatin, and meclizine. He lived with his wife and was completely independent for all activities of daily living. She was his only sexual partner. He smokes cigarettes but denies alcohol use. He has no history of cardiac disease, heart failure, or arrhythmia.

On physical exam, his vital signs were heart rate of 84 bpm, blood pressure of 147/81 mmHg, respiratory rate of 16, and temperature of 98.5 °F. Orthostatic vital signs were normal. Cardiac exam revealed regular rate and rhythm without murmurs or gallops. Abdominal, pulmonary, vascular, skin, and lymph node examination was normal. His neurological exam revealed normal strength, and intact cranial nerves, without sensory deficits. His gait was within normal limits. Romberg testing was normal. Lab data was notable for a hematocrit 25.6 %, hemoglobin 8.0 g/dL, MCV 93.6 fL, BUN 30 mg/dL, creatinine 1.6 mg/dL, glucose 256 mg/dL, calcium of 9.3 mg/dL, total protein of 9.7 mg/dL, albumin of 2.4 mg/dL, and a mild lactic acid of 4.6 mg/dL. Urinalysis was positive for evidence of proteinuria. The remainder of laboratory testing was within normal limits. EKG revealed sinus tachycardia without any acute ST changes or history suggestive of prior coronary disease.

Magnetic resonance imaging of the brain and neck did not show any significant stenosis. Carotid Doppler was normal and transthoracic echocardiogram did not reveal any wall motion abnormalities, a normal

3 III. Discussion of Differential Diagnosis

Orthostasic hypotension as a result from his anemia and prior gastrointestinal bleeding was considered initially as a potential diagnosis. However, further history and review of prior records revealed that prior episodes of syncope preceded the admission for gastrointestinal bleeding. In addition, his admissions for reported gastrointestinal bleeding never required blood transfusions and his hemoglobin levels were always stable. Cardiac examination and imaging narrowed the differential diagnosis, as there was no indication of aortic stenosis or a clinically significant outflow obstruction. Neurologic examination was only helpful in ruling out diagnosis, but was otherwise not helpful in leading to a more probable cause. The weight loss was of great interest to the medical team. It was not typical to elicit or consider in the acute presentation of syncope. It assisted in making an alternative diagnosis.

Looking closer at some of his laboratory results, we inquired for a unifying diagnosis that can further explain his renal insufficiency, proteinuria, anemia, elevated lactic acid, and protein-albumin gap. Though he had presented to multiple institutions with an elevation of his baseline creatinine, it was never worked up at any of the outside institutions. Telemetry testing was performed numerous times and was always normal. Orthostatic hypotension was ruled out during initial vital signs and also was never positive at any of his prior visits. We decided to investigate his elevated protein/ albumin gap, which was found in the setting of anemia and kidney injury, in an elderly male. A major concern and an important diagnosis to consider was multiple myeloma.

His completed work-up included serum protein electrophoresis (SPEP), serum immunofixation and free light chain testing. SPEP demonstrated the presence of an IgM kappa monoclonal band, with an elevated IgM level at 8090 mg/dL. The patient's underwent bone marrow biopsy and results were consistent with lymphoplasmacytic lymphoma. Serum viscosity was elevated at 9.2 centipoises. Following review of his clinical story, bone marrow biopsy, and cytogenetics, a diagnosis of Waldenström's macroglobulinemia was made. The patient first tolerated plasmapheresis followed by chemotherapy. At 6-month follow-up, his symptoms had resolved, and he had no further episodes of syncope.

4 IV. Conclusions

Syncope work-up should be highlighted by a detailed history and physical exam, and not driven by imaging or diagnostic testing. In our patient, renal insufficiency, proteinuria, anemia, elevated lactic acid level, and protein/albumin gap, suggested an atypical cause of syncope. Waldenström's macroglobulinemia is associated with elevated IgM levels. IgM, a pentamer, if elevated, can cause symptoms related to hyperviscosity. In our patient, it explained his neurologic symptoms. Common neurologic syndromes in the literature include vertigo, hearing loss, changes in vision, and ataxia. Headache, altered mental status, stroke and seizures, have also been described. Rarely, hyperviscosity can cause syncope. Hyperviscosity is a clinical diagnosis, and treatment should be started prior to the return of test results. This case highlights the importance of clinical thinking in the workup of syncope.

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