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Recurrent Syncope in A Patient with Subdural Hygroma: A Case Report

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ABSTRACT

This case report highlights the importance of recognizing recurrent syncope as a clinical manifestation of a Subdural Hygroma (SDHy). SDHy is a collection of Cerebrospinal Fluid (CSF) within the subdural space that does not contain blood. The presentation of SDHy is typically, asymptomatic. However, in some cases, it can alter a patient's consciousness, resulting in recurrent syncopal episodes. Clinicians should maintain a high index of suspicion for SDHy when a patient encounter presents as such. Historically, and to date, the management of SDHy has remained controversial, i.e., whether to treat conservatively or with more aggressive surgical drainage. ***This case report features the presentation of*** an elderly female suffering from repeated syncopal attacks. Extensive neurologic and cardiac workups revealed no focal neurologic deficit or positive finding other than SDHy.

INTRODUCTION

SDHy is a collection of Cerebrospinal Fluid (CSF) within the subdural space (underlying the dura mater and overlying the arachnoid mater) that does not contain blood. The presentation of SDHy is typically asymptomatic. However, in some cases, it can produce a mass effect on the cerebrum, reducing blood flow to the brain parenchyma and altering consciousness, resulting in recurrent syncopal episodes. Historically, and to date, the management of SDHy has remained controversial, i.e., whether to treat conservatively or with more aggressive surgical drainage.[1] SDHy may present with myriad symptoms, including but not limited to headaches, seizures, focal neurologic deficits, and mental status changes. Controversy related to the management approach is primarily because the vast majority of patients with SDHy remain asymptomatic. Nonetheless, neurosurgical consultation is warranted if a patient exhibits any sign indicative of a mass effect.[2] ***Herein,*** we report the case of a 79-year-old woman with a past medical history of essential hypertension, hyperlipidemia, and Gastroesophageal Reflux Disease (GERD), who presented to the hospital with the complaint of repeated fainting. Upon presentation to the hospital, an

Electrocardiogram (ECG) showed a normal sinus rhythm without ST segment or T wave changes and normal PR and QRS intervals. Cardiac and neurologic workups were unremarkable, apart from the finding of SDHy on CT imaging of the brain. In this report, we emphasize the importance of coordinated multidisciplinary management between different specialties, namely internists, neurologists, cardiologists, and radiologists, to appropriately address the diagnostic challenges related to the workup, diagnosis, and management of a patient with SDHy. The patient featured in this case report gave informed consent and is aware that there are no patient-identifying details in the text or images submitted.

CASE DESCRIPTION

A 79-year-old Hispanic female with a past medical history of ***essential hypertension, hyperlipidemia, GERD,*** and a past surgical history of bilateral total knee replacements, presented to the hospital with the complaint of repeated fainting (resulting in multiple traumas). The patient reported that she most recently fainted while attempting to move from seated to standing. A family member who witnessed the syncopal episode said the patient was unconscious for a few seconds before fully recovering. The patient denied prodromal symptoms, including lightheadedness, dizziness, diaphoresis, chest pain, and palpitations. She also denied any situational relationship, such as fainting while exerting herself, coughing, or urinating. The patient reported having numerous similar episodes in the past. She denied any history of a bleeding diathesis or malignancy. Upon physical examination, blood pressure was measured at 158/89 mmHg supine and 150/90 mmHg standing (indicating no orthostatic hypotension). Heart rate was 80 beats per minute with regular rate and rhythm; temperature: 98.0°F (oral); SpO2: 99% on ambient air. Cardiovascular, pulmonary, and abdominal examinations revealed no abnormalities apart from mild tenderness over the thoracic and lumbar spine and skin abrasions on the bilateral knees secondary to her fall. A neurological examination demonstrated that her mental status was normal (Glasgow Coma Scale: 15/15 points). She was also determined to have normal sensation, reflexivity, range of motion, and strength. Gait and cranial nerve tests were also normal. Babinski sign was absent bilaterally. Positive neurologic signs found on her exam included the Hoffman sign (involuntary flexion of the first and second distal phalanx in response to flicking the nail of the third or fourth digit) and the Palmomental sign (contraction of the mentalis muscle of the lower lip in response to stroking the ipsilateral palm). Both signs were present bilaterally. The patient was admitted to inpatient service with telemetry monitoring to continue the workup of her recurrent syncope. An X-ray of the patient's chest revealed an unremarkable cardiac silhouette (Figure 1). Initial ECG showed a regular, normal sinus rhythm without ST segment or T wave changes and normal PR and QRS intervals (Figure 2). Transthoracic echocardiography showed mild concentric Left Ventricular Hypertrophy (LVH), no sign of structural or functional abnormalities of the left and right ventricles, normal Left Ventricular Ejection Fraction (LVEF), which was measured at 60%, moderate diastolic dysfunction, and no pericardial effusion (Figure 3). ***CT of the brain without contrast showed*** a bilateral, frontoparietal, chronic subdural hygroma without evidence of acute intracranial hemorrhage or midline shift of the brain (Figure 4). ***CT of the cervical, thoracic, and lumbar spine showed*** multilevel spondylotic changes, including intervertebral disc space narrowing and mild dextroscoliosis. No acute displaced fractures or other sign of trauma

was noted. ***Duplex ultrasound of the bilateral carotid arteries*** (Figure 5) showed no occlusion or hemodynamically significant stenosis. The laboratory workup, including Complete Blood Count (CBC), Comprehensive Metabolic Panel (CMP), coagulation profile, cardiac troponin-I, high-sensitivity C-Reactive Protein (CRP), lactic acid, and COVID-19 tests were within normal limits. The patient was closely monitored throughout her hospital stay. Vital signs were recorded every four hours and remained within normal limits. Serial ECGs and 72-hour telemetric recordings showed no evidence of cardiac arrhythmia. Given that the patient's cardiac and neurologic workups were otherwise negative, we attribute the recurrent syncopal episodes to the radiologic findings of SDHy. After a thorough case discussion between internal medicine, neurology, and radiology teams, the patient was deemed safe to be discharged home. The patient was counseled for regular follow-up with her primary care physician and consideration for brain MRI and surgical intervention if the syncopal episodes persist.

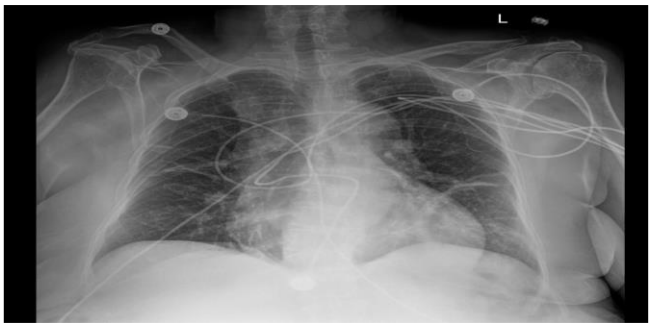

Figure 1: Chest X-ray: showing normal cardiac silhouette and no cardiopulmonary evidence of disease.

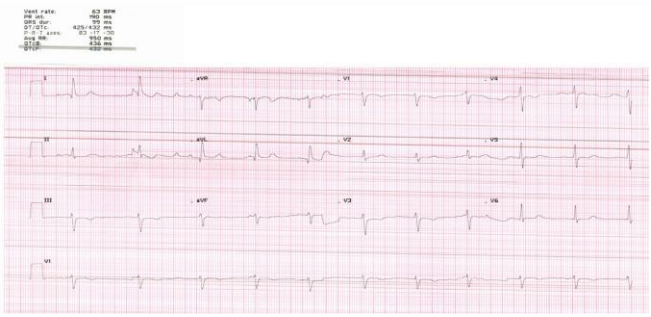

Figure 2: ECG showing normal sinus rhythm.

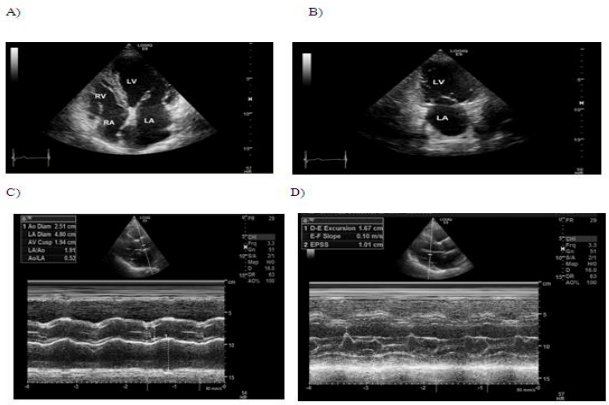

Figure 3: Transthoracic Echocardiography (TTE) showing normal findings on: A) 4 chamber view B) 2 Chamber view C) M mode on aortic valve showing normal aortic valve opening D) M-Mode of the Mitral valve shows normal opening and closure.

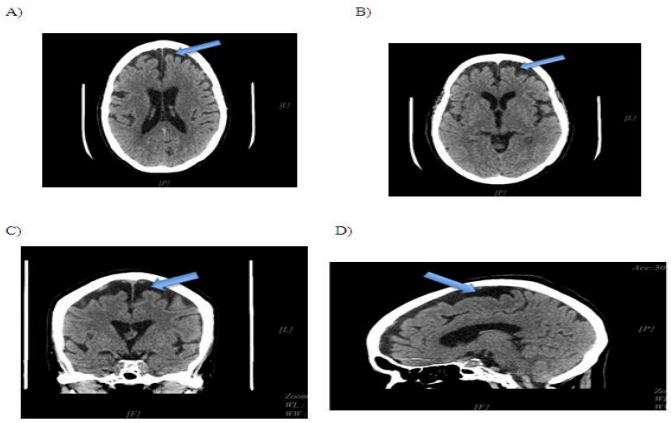

Figure 4: Brain Computed tomography Without Contrast Agent **A-D)** showing a left frontoparietal chronic subdural hygroma, with no CT evidence of acute intracranial hemorrhage, no midline shift, or mass effect.

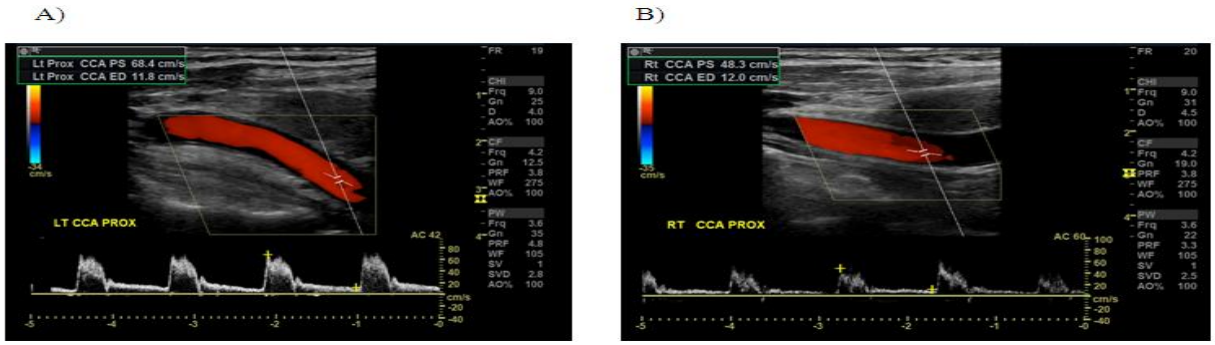

Figure 5: Examination of the common Carotid Artery (CCA). A) Carotid Duplex showing left Common carotid non stenotic, B) Carotid Duplex Right Common Carotid, non-stenotic

DISCUSSION

In this case report, we call attention to a unique presentation of an elderly female suffering from repeated syncopal attacks. Extensive neurologic and cardiac workups revealed no focal neurologic deficit or positive finding other than SDHy on CT imaging of the brain. Whether the patient's syncopal episodes represent the cause of the SDHy (as a result of trauma sustained during a fall) or, conversely, represent an effect of the SDHy (due to increased intracranial pressure leading to cerebral hypoperfusion) is quite unclear. In routine cases of syncope, neurologic imaging (CT or carotid Duplex) is generally avoided, especially in the absence of neurologic deficits. However, a history of significant trauma mandates such workups, as with this case.The underlying cause of SDHy can be idiopathic or a result of trauma-related phenomena (Acute vs. chronic). In most cases, SDHy is determined to be driven by chronic

subdural hematomas.[1] SDHys commonly occur in the elderly when there is a history of trauma,[3] following neurologic procedures, or in children diagnosed with Central Nervous System (CNS) infections. While the majority of SDHys are small and asymptomatic, they can be large enough to mimic other pathologies by presenting with a wide range of symptoms, including headaches, focal neurologic deficits, seizures, or alterations in mental status.[1] In this case, the patient presented with a history of recurrent syncopal attacks. Subsequently, the patient had negative cardiac and neurologic workups. On CT imaging, SDHy is visualized as a crescentic density in the subdural space and typically does not extend beyond sulci or exhibit a mass effect. Additionally, with CT imaging, SDHy can be visualized along the supratentorial cerebral convexities (as seen in this case) or, rarely, in the posterior fossa. The differential diagnosis of SDHy should include chronic subdural hematoma and cerebral atrophy. Cerebral atrophy results in involutional change, widening of the cortical sulci and gradual enlargement of the ventricles, and a pronounced cortical thinning resembling SDHy.[4] Such differentiation can be made possible by gadolinium-enhanced brain MRI, an imaging modality not performed in this case, which may be a limitation. The workup for our patient's syncopal episodes was negative, apart from the radiographic evidence of SDHy. There is a paucity of literature reporting on SDHy as related to recurrent syncopal attacks. To our knowledge, only one case has been reported, by Bruner and colleagues, [2] reporting on a situational syncopal attack (coinciding with cough). The patient was a 75-year-old male with no significant comorbidities apart from CT imaging of the brain showing SDHy. In our case, the workup was extensive, and the recurrent syncopal attacks were not attributed to hypotension (neither reflex nor orthostatic) or any cardiac cause (arrhythmia, valvulopathy, or structural abnormality). We can argue that it would have been advisable to investigate further with the use of an implantable loop recorder (indicated to be Class I (i.e. useful and indicated) for patients with recurrent syncope of uncertain origin according to the ACC/AHA/HRS Guidelines for the Diagnosis and Management of Syncope,[5] yet, in our patient, there was no concern for arrhythmia as evidenced by numerous benign ECG and telemetric readings throughout the hospital course. The underlying mechanism for either SDHy or subdural hematoma to cause recurrent syncope may be related to the proposed mechanism of a temporary increase in Intracranial Pressure (ICP), which subsequently impedes blood flow to the cerebrum, resulting in a transient loss of consciousness. Such a temporal association may better explain reflex or situational syncope associated with cough or vagal maneuvers. Per ACC guidelines, [5] the routine workup for syncope usually does not involve neurologic imaging with CT, MRI, or carotid Duplex and is reserved for those patients with neurologic deficits. Given this patient's otherwise negative workup and the consensus between cardiology, internal medicine, neurology, and radiology services, it was decided that her syncopal episodes were likely brought on by increased intracranial pressure causing reduced cerebral perfusion. Managing SDHy is controversial as most patients do not experience symptoms. However, if there are findings that indicate a mass effect, neurosurgical consultation is warranted to assess if there is a need for surgical drainage.[2]

In Summary, this case emphasizes the importance of coordinated multidisciplinary management between specialties, namely internists, neurologists, cardiologists, and radiologists, to appropriately address the diagnostic challenges related to the presentation of a patient with SDHy.

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