CLINICAL VIGNETTE

Delayed Acute Subdural Hematoma

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Case Presentation

A 91-year-old female suffered a ground level fall and presented to the emergency department (ED). Past medical history includes hypertension, hyperlipidemia, coronary artery disease and type 2 diabetes. She reported head trauma and loss of consciousness lasting 20 seconds. Home medications included aspirin, losartan and atorvastatin. Her initial vitals signs included T 36.3 C, blood pressure 212/110 mmHg, heart rate 79/min, respiratory rate of 22 per minute, and room air oxygen saturation of 98%. Weight was 68.6 kg with BMI of 27.6 kg/m^2. On physical examination, she was alert and fully oriented, without any focal neurologic motor or sensory deficits. Laboratories included normal complete blood count (CBC), and basic metabolic panel (BMP), and liver function tests (LFTs).

She had a normal neurologic exam, with Glasgow Coma Status (GCS) 15. She remained hypertensive with a systolic blood pressure of 170 to 180 mmHg and was thought to have a postconcussive headache and associated nausea. CT brain and cervical spine were unremarkable for acute intracranial pathology. On hospital day two, acute hypotonic hyponatremia developed with serum sodium of 121. She did not have any neurologic changes on exam or complain of new symptoms. Nephrology was consulted and the patient was started on hypertonic saline, closely monitored serum sodium. Sodium chloride tablets were also started due to concern for syndrome of inappropriate diuretic hormone (SIADH). On hospital day three, she developed an acutely worsening headache and drowsiness. She was somnolent on exam, and hypertensive with a systolic blood pressure of 180 to 200 mmHg. She was given intravenous hydralazine and oral amlodipine. An urgent CT brain without contrast was obtained, which showed an acute right sided subdural hematoma without significant mass effect or midline shift and trace subarachnoid hemorrhage along the bilateral cerebral convexities. Neurosurgery recommended immediate transfer to the neuro intensive care unit (ICU) for possible surgical intervention. The patient had been receiving aspirin 81 mg daily, and she was given DDAVP 30 mg/kg intravenously, 1 unit of platelets for emergent reversal of antiplatelet effects. She was started on intravenous nicardipine infusion to maintain strict blood pressure control between 90 to 140 mmHg. Her neurologic status was closely monitored in the neuro ICU with continuous electroencephalogram (EEG) and transcranial doppler ultrasound. She was started on levetiracetam for anti-epileptic prophylaxis and nimodipine to reduce cerebral vasospasm. Repeat CT and neurologic exam remained stable, and she did not require surgical intervention. Her hyponatremia and hypertension remained controlled and she was discharged home.

Discussion

Ground level falls are generally thought of as a low impact mechanism of injury. However, elderly patients presenting with fall-related injuries are at increased risk of mortality and intracranial injury. Age alone remains an independent risk factor.[1] In addition, the use of aspirin and anticoagulation is also increasing in the geriatric population and may impact outcomes in ground level fall injuries. Elderly patients fall more often than their younger patients. Forty-three percent of all falls occur in patients 65 and older.[1] Among fall-related injuries, traumatic brain injury increases mortality risk in elderly patients, especially those found with GCS less than 15.[2,3] Mild traumatic brain injury is common, found in 80 percent of all hospital admissions due to head injuries. Mild traumatic brain injury (TBI) is defined as initial GCS of 13 to 15, loss of consciousness less than 20 minutes, absence of focal neurological deficits, and post traumatic amnesia less than 24 hours.[4] Despite a normal GCS level following a ground level fall, one study reported 20 percent of elderly patients with intracranial hemorrhage.[2] Therefore, it is important for clinicians to maintain a high index of suspicion for intracranial hemorrhage when caring for elderly patients with mild TBI presenting to emergency department or admitted to the hospital.

Acute subdural hematoma (ASH) is easily diagnosed with brain computed tomography (CT) as an extra-axial hyperdense crescent mass between the dura the brain parenchyma.[5] Delayed acute subdural hematoma (DASH) is a rare clinical condition that is defined as an ASH that is not apparent on the initial CT scan but appears on a follow up CT scan.[6] The incidence of DASH is unknown. The OCTOPUS Study, a multicenter randomized controlled trial of 2,602 patients presenting to the ED with mild TBI compared obtaining an immediate brain CT scan to admission for medical observation, found that rates of mortality and severe loss of function at 3-month follow up were

similar between groups.[7] They reported none of the patients with normal findings on the immediate CT had later complications. There literature review included more than 62,000 patients for cases in which an early CT was normal and then the patient deteriorated within 2 days. They found only a few cases of early adverse outcomes after normal CT and GCS 15 on initial presentation.[8] The OCTOPUS authors support immediate CT as a safe, cost-effective way to triage ER patients with mild TBI for admission.

Nevertheless, cases of DASH have been reported.[4,9,10] Shabani et al performed a literature review of DASH and reported it occurs mainly in middle-aged to elderly patients taking anticoagulation or antiplatelet therapy. Neurologic deterioration occurs within in 24 hours in 70 percent of cases.[10] For this group of patients, the authors recommend admission and observation. The mechanism for DASH remains unknown. One hypothesis is that the bridging veins or small perisylvian arteries are ruptured, while the patient is hypotensive. Subsequent fluid resuscitated leads to the delayed development of DASH. Some believe that delayed clinical deterioration can occur in elderly patients with ASH because volume loss and atrophic changes in old age create a larger subdural space, which may initially compensate the expanding hematoma and edema prior to neurological deterioration.[5]

Conclusion

Although rare, DASH should be suspected in elderly patients taking anticoagulation or antiplatelet therapy presenting with mild TBI who develop acute neurologic deterioration in the days following a negative initial brain CT.

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