CLINICAL VIGNETTE

Chronic Subdural Hematoma in an Elderly Patient: Diagnosis and Management Overview in Primary Care Setting

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Introduction

With increase in the aging population, many diseases have become more prevalent. Chronic subdural hematoma (CSDH) is expected to double by 2030.[1] CSDH is a common neurosurgical event in elderly patients, with mean age of 76.8 years old.[2] The risk increases with anticoagulation or antiplatelet therapy. In patients over age of 70 years, the CSDH has been identified as a sentinel event for underlying systemic pathology, with one year mortality similar to hip fractures.[3] Surgical treatment is the gold standard; however, given the multiple chronic medical conditions in the elderly, a conservative approach maybe valuable and beneficial in certain situations. Nonsurgical measures can include the "wait and watch/ wait and scan" approach or medications such as corticosteroids, tranexamic acid, mannitol, platelet activating factor receptor antagonist or statin therapy. Strong evidence based studies for nonsurgical treatment of CSDH are still lacking. In this vignette, the rule for imaging in the setting of traumatic brain injury and treatment options for CSDH will be reviewed.

Case

A 69 year old male with history of hypertension and chronic hepatitis B presented for evaluation six days after a fall from a ladder. He was working on a retaining wall, lost his balance and slid down a ladder about 4-5 feet from the ground. He bumped the back of his head and landed on his left side. He denies loss of consciousness, and did not seek immediate care. Other than a mild tender bump on the back of his head and a contusion around his right eye with mild swelling, he denied any headache, blurry vision, dizziness, nausea, vomiting or confusion. He also denied any neurologic changes since the incident. He was not taking any anticoagulants or aspirin and his only medication was valsartan for blood pressure control.

On exam, vital signs were BP 143/89, pulse 81, afebrile, O2 sat 98%. Patient was AAO x3 and in no acute distress. Physical exam was significant for ecchymosis around the right eye with minimal tenderness. His eye exam showed EOMI with normal visual fields. There was a small soft bump noted at the left posterior parietal region with minimal tenderness and fluctuance. Cranial nerves were intact with normal reflex, sensation and motor strength. He had normal finger to nose and heel to shin test. No Romberg sign. No pronator drift and normal gait.

With a normal neurologic exam and absence of any symptoms of concussion, he was instructed to monitor for any neurologic change and sent home. A week later, the patient traveled abroad for 2 months. He had a medical exam done outside of the USA per family's request. MRI of brain, a month after the fall, was positive for a symmetrical bilateral frontoparietal subdural hematoma without midline shift. He was evaluated by local neurosurgeon and was recommended conservative treatment with observation because he was asymptomatic. Repeat CT a month later showed unchanged, persistent hematoma. He returned home and followed up with his PCP. He continued to deny any headache, mental status and neurologic changes. However, his spouse noticed that his gait had become unsteady and he appeared to be more off balance. A repeat CT showed larger mixed density, subacute on chronic bilateral subdural hematoma that is 21mm vs. 19 mm previously. There was a new, slight rightward midline shift of 2mm. Neurosurgery recommended burr hole craniotomy. Patient refused the surgery but

agreed to follow up with serial imaging studies. Two weeks later, a repeat CT of brain showed diminished size of bilateral fronto-parietal mixed density subdural hematomas decreased from 21 mm to 15 mm. There was diminished mass effect and resolution of the midline shift. Another repeat CT 1 month later continue to show mild decrease in size of the bilateral subdural hematomas. Clinically, the patient had regained his balance and his gait was no longer unsteady. He remained off aspirin and will continue to follow up with the neurosurgeon with another MRI brain scheduled in the near future.

Discussion

The subdural space is between the dura and arachnoid maters. There is a layer of dural border cells in the space with bridging veins transversing through the cells and subdural space. Head trauma can result in injury to these bridging veins with bleeding into the subdural space. In patients with brain atrophy, as in elderly or alcoholic, the arachnoid layer is further separated from the dura mater as the brain volume decrease and further stretch the bridging veins making them more easily torn, even with minor trauma. In addition to trauma, low CSF fluid pressure or intracranial hypotension due to CSF leakage either spontaneously or from lumbar puncture or VP shunt, can lead to rupture of the veins due to traction on the anchoring structure because the brain loses buoyancy.[4] The insult in the subdural space can trigger both hematoma and complex repairing response to heal the injured tissue. It leads to proliferation of the dural border cells and formation of granulation tissue with collagen fibers. These local inflammatory processes can induce angiogenic reactions that promote neovascularization in the subdural space. The fragile new capillaries can break easily and result in further bleeding into the hematoma causing enlargement and subsequent complications.[5]

Chronic subdural hematoma (CSDH) is an old collection of blood and blood breakdown product in the subdural space. It can result from prior acute subdural hematoma or from subdural hygroma with bleeding from micro capillaries.[4] In the acute SDH, if the hematoma is not evacuated surgically or completely resorbed by the body, it becomes chronic. CSDH can occur over 2 weeks after the trauma. A hygroma is a collection of CSF. Hematoma can result from hygroma if there is a leak from new vessel that was formed. The main complication for enlarging CSDH is the mass effect on the brain structure. Risk factors that can contribute to CSDH includes advanced age, use of anticoagulation therapy, history of falls, minor head injury, chronic alcohol abuse, epilepsy, hemodialysis, low intracranial pressure states, bleeding diatheses, meningioma, dural metastases, to name a few.[4-5]

There is a wide spectrum of clinical presentation for CSDH. The symptom onset and progression can vary from days to weeks and it can be transient or fluctuating.[6] Patient may have minimal symptoms. On the other hand, it can mimic stroke or rapidly progressive dementia. The CSDH symptoms may not become evident until weeks after the initial injury. Patient can present with multiple symptoms including gait disturbance and falls, limb weakness, cognitive decline, nonspecific deterioration, speech impairment or acute confusion.[2] Glasgow coma scale can range from 3-15. Because of the wide range of presentations, the diagnosis of SDH is not as clear cut if patient presented without history of head trauma and/or on anticoagulation therapy. In addition to history and physical evaluation with focus on neurologic assessment, imaging is key in diagnosing SDH and determination of treatment option. CT is most widely used due to its speed and availability. The acute SDH presents as high-density crescentic collection while chronic SDH may appear as hypo-dense or iso-dense lesion. However, if patient has bilateral iso-dense subdural hematoma without midline shift, the diagnosis can be missed.

In patients with history of head trauma, there are two criteria that can be used to determine the need of CT scan. The criteria are the Canadian CT head rule and the New Orleans criteria. The Canadian criteria rule states CT is needed if patient has any of the following after head trauma which are GCS < 15 within 2 hours after injury, suspected open or depressed skull fracture, any signs of basilar skull fracture, >2 episodes of vomiting, >65 years of age, amnesia > 30 minutes, dangerous mechanism of impact, i.e. fall

from >3 feet or > 5 stairs. In addition, any patient with seizure, neurologic deficit and patients on anticoagulation with trauma should have CT evaluation.[7] The New Orleans criteria are simpler. Patients with GSC < 15 and has headache, vomiting, age > 60, drug or alcohol intoxication, persistent amnesia or visible trauma above clavicle need CT evaluation.[7]

Early intervention for confirmed subdural hematoma prevents irreversible brain injury and death due to hematoma expansion, elevated intracranial pressure and brain herniation. The management option can be separated into surgical or conservative management. Decision between these treatments is based on multiple factors. These are GCS, size of hematoma, degree of midline shift, neurologic exam with pupillary signs, clinical stability, acuity of SDH, any comorbidities and associated trauma and age.[8] If patient is symptomatic with confirmed hematoma radiologically, a surgical treatment for hematoma evacuation is warranted. Urgent surgery is warranted if clot thickness > 10 mm or midline shift > 5 mm, GCS decreased by > 2 since the injury or patient with asymmetric or fixed and dilated pupil. Surgical intervention resulted in rapid improvement in over 80% of symptomatic patient.[9] There are three different surgical techniques based on size of opening. Craniotomy with opening greater then 30mm is most effective for complicated cases. Burr hole craniotomy has smaller opening between 10-30 mm. The Twisted drill craniotomy has skull opening < 10 mm is preferred for patient with multiple comorbidities.[10]

Non-operative management of SDH can be used for stable patients, including comatose patients, with small hematomas < 10 mm in clot thickness as long as there are no clinical or CT sign of elevated intracranial pressure or brain herniation (midline Shift < 5 mm).[8] If patient is asymptomatic or has a small non-space occupying hematomas, a conservative management with careful observation and repeated follow up imaging study can be an appropriate option.[4] Often, conservative management is necessary when patients refuse surgery or have high operative risk.[10,11] In the CSDH setting, there is no current expert guideline. Patients with CSDH who develop signs of brain herniation or elevated intracranial pressure such as asymmetric or fixed and dilated pupils need urgent surgical intervention. If there is severe cognitive impairment or progressive neurologic deterioration attributed to CSDH, surgical intervention may be beneficial.

Some proposed medical management of CSDH include the use of corticosteroids, tranexamic acid, mannitol, PAF receptor antagonist and statins. These are all potential treatment options; however, no strong supporting data exist at this time. Most of these medications worked on the inflammatory process and the angiogenic reaction in the formation of hematoma. Corticosteroid suppresses the inflammatory and angiogenic factors thus decrease bleeding event.[10] The tranexamic acid has antifibrinolytic effect and increase vascular permeability in CSDH allowing reabsorption of the hematoma.[12] Mannitol as osmotic diuretic can decrease hematoma pressure thus prevents fluid moving into the hematoma capsule and avoids microvascular bleed from enlarging capsule.[11] Platelet activating factor is a potent mediator of inflammatory process that leads to growth of CSDH. The PAF receptor antagonist works at inhibiting the process, thus promoting the resolution of the hematoma.[1] Statins appear to promote angiogenesis and restrain inflammation. Atorvastatin 20 mg daily for 1-6 months led to significant reduction in hematoma size. It may be a safe and cost effective alternative treatment to surgery in elderly patients.[13]

In this patient, CT imaging of the brain should have been ordered during the initial evaluation as he met the criteria for both Canadian CT rule and New Orleans Criteria based on his age, visible facial and head trauma with fall from > 3 feet. It was possible that his chronic subdural hematoma might have worsened from his oversea travel. It was important for him to establish his care with a neurosurgeon along with frequent follow up with primary care physician to monitor his neurologic status. His repeated imaging had showed a midline shift at one point with patient exhibiting gait imbalance. Patient had refused surgical intervention and preferred to wait. He continued to stay off aspirin. His subsequent CT brain showed improvement 5 month

after the fall. His gait imbalance also resolved. His follow up visit did not show signs of increased intracranial pressure. At this point, he will continue with the conservative follow up. Patient was made aware of his increased risk of recurrent CSDH due to his risk factors.

Conclusion

The CSDH has increased incidence in the elderly given their risk factors of brain atrophy due to aging and increase risk for fall. Because the presentation of the condition can be widely variable, primary care physician should have a high index of suspicion when evaluating elderly patients, especially with history of fall. In this population, the surgical intervention might not be the most beneficial method of treatment. With emerging understanding of underlying pathophysiology, conservative medical management appears promising. More randomized controlled trials are underway. We anticipate improved guidelines and treatment options to guide the management of CSDH in this population. At this time, close follow up with repeat imaging and neurologic evaluation is critical for patient on conservative therapy for chronic subdural hematoma.

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