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Delayed Symptomatic Subdural Hematoma Following an Initially Normal CT Head

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Debra Bakerjian, PhD, APRN, RN; Ryan Martin, MD, FCNS; Patrick Romano, MD, MPH and Garth Utter, MD, MSc for this Spotlight Case and Commentary have disclosed no relevant financial relationships with ineligible companies related to this CME activity.

Learning Objectives

At the conclusion of this educational activity, participants should be able to:

- Describe the indications for head imaging after a mild traumatic brain injury.

- Describe the risk factors for subdural hematoma expansion.
- Describe what symptoms should prompt ordering repeat head imaging following a mild traumatic brain injury.
- Understand how cognitive bias related to concussion can lead to a missed diagnosis and worsening neurological symptoms.

The Case

A man in his mid-50's presented to the hospital for a persistent headache after a sledding injury with his son 4 days earlier. He was not wearing a helmet and sledded head-first into a tree. There was no loss of consciousness and no vomiting or confusion, but the headache did not improve after four days. His past medical history was notable only for hypertension managed with amlodipine. Vital signs were normal, and a templated neurologic examination was documented as normal. No blood tests were performed, but a non-contrast computed tomography (CT) scan of the head was read as normal. Standard aftercare instructions for head injury were given.

About three weeks later, the patient saw his primary care physician (PCP), still complaining of ongoing headache that sometimes kept him up at night, for which he was using over-the-counter ibuprofen, as well as some forgetfulness and concentration difficulties as he tried going back to work. He reported also taking sertraline for a history of depression, low-dose aspirin and vitamins for preventive reasons. The vital signs and physical exam were again normal. He was diagnosed with post-concussive syndrome; no specific investigations or treatments were recommended. However, about two weeks later, the patient's wife insisted that he go back to the hospital for worsening cognition plus increased use of ibuprofen for headache. Head CT showed a large frontal subdural hematoma with midline shift. He was taken urgently to the operating room, where craniotomy was performed and the hematoma was evacuated, showing at least 3 different blood consistencies suggesting three different ages of hemorrhage.

The Commentary

By Ryan Martin, MD, FNCS and Kiarash Shahlaie, MD, PhD, FAANS, FCNS

Initial evaluation for brain injury often includes CT of the head, especially given the ubiquitous presence of CT scanners in most, if not all, acute care centers. In the setting of mild traumatic brain injury (mTBI), defined as a Glasgow Coma Scale (GCS) of 13-15, head CT has a sensitivity ranging from 73% to 99% and a specificity of 4% to 59% for identifying intracranial hemorrhage.[1,2]

Among patients with mTBI and initial loss of consciousness, amnesia to the event, or witnessed confusion or disorientation, the decision to pursue acute brain imaging may be guided by the Canadian CT Head Rule (CCHR), which recommends head CT if any of the following signs are present: GCS <15 after two hours from injury; suspected open, depressed, or basilar skull fracture; two or more episodes of vomiting; age 65 years or older; amnesia for 30 minutes or more before impact; or a dangerous mechanism (i.e., pedestrian struck by motor vehicle, occupant ejected from motor vehicle, or fall from >3 feet or down >5 stairs).[3] When performed properly, the CCHR has 100% sensitivity for patients requiring neurosurgical intervention and those with clinically important brain injury.[3] A similar decision rule has been developed and validated for

children who present with GCS of 14-15 after blunt head trauma.[4,5] In this case, the patient did not present immediately after his injury, but if he had, the ED team would likely have interpreted sledding head-first into a tree without a helmet as a mechanism of similar danger as ejection from a motor vehicle. However, another widely used decision rule for blunt head trauma (with or without neurologic abnormalities at presentation) does not consider the mechanism of injury, focusing only on findings absent in this case: suspected skull fracture; scalp hematoma; any neurologic deficit; GCS < 15; abnormal behavior; coagulopathy; persistent vomiting; and age 65 years or older.[6]

While clear guidelines suggest when to image patients with acute mTBI, no such guidelines exist regarding indications for repeat imaging, even in those patients with intracranial hemorrhage or high-risk features. In case series, patients who present with an initial head CT showing intracranial hemorrhage after mTBI *and who do not* decompensate neurologically rarely develop meaningful hemorrhagic expansion requiring neurosurgical intervention.[7-9] Based on these series, routine repeat imaging in neurologically stable patients with mTBI may not be necessary and may represent a cost-saving opportunity.

Repeat imaging in patients with initial negative head imaging is even more controversial. The published data have largely been limited to case series of elderly patients on antiplatelet or anticoagulation therapy, a population felt to be a high-risk for hemorrhagic conversion (i.e. developing an intracranial hemorrhage after an initially negative CT head) and expansion. Borst and colleagues[10] reported that 0.9% of 1377 patients developed intracranial hemorrhage on subsequent imaging, but none of these events was associated with neurological deterioration or neurosurgical intervention. Similarly, Flaherty and colleagues reported similar rates of hemorrhagic conversion after an initial normal head CT among patients not taking (0.7%) versus those taking antithrombotics (0.6%).[11] Mourad and colleaguesdescribed 0.5% risk of hemorrhagic conversion within 24 hours among elderly patients taking direct oral anticoagulants.[12] While these conversion rates are low, early identification may be important in patients taking antiplatelet and anticoagulation medications, as persistent antithrombotic use is a risk factor for delayed hemorrhagic expansion that may contribute to neurological deterioration.[13,14]

Delayed Acute Subdural Hematoma

Despite the very low risk in these case series, symptomatic subdural hematomas have been reported to occur after initially negative head CTs, as in the patient described in this report. This process has been termed delayed acute subdural hematoma (DASH).[15-22] The pathophysiology of DASH is not well understood, but it is largely seen in elderly patients who are taking antiplatelet and anticoagulant medications. Neurological deterioration occurs over days to weeks. Classically, acute subdural hematoma has been attributed to rupture of bridging veins across the dura, with elderly patients more susceptible due to brain atrophy.[23,24] However, other hypotheses have been proposed, including osmotic gradients, angiogenesis, and chronic inflammation.[23,24] An intriguing hypothesis regarding the development of DASH is its apparent association with subdural hygromas or effusions, which are often overlooked or considered normal findings on initial head CT reports.[17,21] Subdural hygromas are collections of clear cerebrospinal fluid under the dura mater of the brain and are more likely to be seen in the elderly population, given the presence of atrophy. Olivero and colleagues described 37 patients who underwent surgery for chronic subdural hematomas, 7 of whom had normal initial imaging except for a subdural hygroma, which was invariably the site of the subsequent hematoma.[21] They and others hypothesize that the hygroma is

caused by traumatic arachnoid membrane tears that initiate an inflammatory response in the subdural space, leading to new blood vessels (i.e., neovascularization) that ultimately bleed and cause DASH. [17,21]

It is also possible that DASH represents progression of a “missed” subdural hematoma that was radiographically occult or missed as a false-negative interpretation. In a series described by Amrhein and colleagues,[25] the sensitivity and specificity of a non-enhanced axial CT scan after trauma were 75.7% and 94.3%, respectively, with a false negative rate of 24.3%. These authors found that reformatting axial CT scans to include coronal and sagittal images improved sensitivity and specificity to 88.3% and 98.3%, respectively, reducing the false negative rate to 11.7%. This study suggests that a significant minority of small acute subdural hematomas may be missed, and a subset of these missed hematomas may progress and result in a diagnosis of DASH. In this scenario, of course, a false negative CT interpretation would result in a treatment plan that does not include measures aimed at prevention and early diagnosis of hematoma expansion (routine repeat imaging study, low threshold for delayed imaging if symptoms progress, avoidance of antiplatelet and/or anticoagulating medications).

Overlap with Acute Concussion

Most often, patients who develop DASH are initially diagnosed with concussion, a disease process with overlapping symptoms that may mask the signs and symptoms of a developing subdural hematoma. Acute concussion symptoms are often myriad, but include headache, diplopia, dizziness, nausea and emesis, mood changes, insomnia or hypersomnolence, short-term memory impairment, poor concentration, and fatigue.[26,27] For around 90% of patients, concussion symptoms gradually improve and resolve by three to four weeks after injury.[26,27] Symptoms may last longer, particularly in patients with prior concussion, personal or family history of migraine, or pre-injury mood disorder or learning disability.[27] However, even in this population of patients with persistent symptoms, acute symptoms tend to improve over the first several weeks. Red flags to monitor for after concussion include worsening headache, new focal neurological deficits, repeated or worsening emesis, and changes in consciousness.[26] The patient in this case reported persistent headaches about three weeks after the initial head CT. Of particular interest, the headaches kept him up at night. It is unclear whether his headaches were worse when supine, but nocturnal headaches can be the presenting sign of elevated intracranial pressure from a mass lesion, such as a brain tumor or worsening intracranial hemorrhage, as in this case. Such symptoms should prompt a repeat head CT even in the absence of neurological deficits.

Critique of Patient Care

The acute management of this patient at the time of his first presentation seems appropriate. The initial head CT four days after his injury was reportedly negative for bleeding; therefore, it was reasonable to discharge the patient home with appropriate aftercare instructions. Although a false negative interpretation is possible, there was no clear indication to instruct the patient to discontinue aspirin or avoid other non-steroidal anti-inflammatory drugs. The lesson to be learned in this case is in follow-up care, which as noted above, is not guided by high-grade evidence.

We do not know if a missed subdural hematoma and/or subdural hygroma was present on the initial head CT, but it is our practice to repeat head imaging upon outpatient follow-up in persistently symptomatic

patients (e.g., headaches that are not improving) if any subdural lesion was initially identified. In addition, we obtain repeat imaging in persistently symptomatic patients with risk factors for hemorrhagic expansion, which include older age, hypertension, and use of antiplatelet and/or anticoagulant medications.[13,14] This patient had two such risk factors, including his hypertension and daily concomitant use of aspirin and ibuprofen. A non-improving headache more than three weeks after injury in the setting of these risk factors should have prompted repeat imaging. In addition, as mentioned earlier, nocturnal headaches that keep patients awake at night should prompt repeat imaging given the concern for elevated intracranial pressure from a mass lesion. In this case, we suspect that imaging was not obtained by the PCP as his neurological examination was documented as normal and his difficulties with concentration and memory were attributed to the prior concussion (a cognitive bias sometimes described as "anchoring" or "premature diagnostic closure"). However, a normal neurological examination does not rule out a significant intracranial mass lesion, especially if the lesion is growing slowly, as in DASH, which allows time for the brain to adapt to tissue displacement and modestly increased intracranial pressure without developing focal neurological deficits. If the expanding mass lesion is left untreated, the patient will eventually develop focal neurological deficits, often with devastating consequences.

Lastly, repeat imaging is always indicated in a patient complaining of worsening symptoms after recent trauma – a cognitive forcing strategy to avoid anchoring bias by seeking an underlying causative explanation for a patient's signs, symptoms, and laboratory and imaging data. After this patient's follow-up with his PCP, the patient had worsening headaches (as indicated by his increased ibuprofen use) and worsening cognitive deficits. Concussion should not typically lead to symptoms that worsen over time and patients should always be advised to seek medical attention immediately if symptoms are progressing. In this case, the patient did not seek medical help for another two weeks, which probably contributed to further SDH expansion and permanent neurological deficits.

Conclusion

A high index of suspicion for new intracranial hemorrhage is warranted in patients with persistent or worsening symptoms after the diagnosis of concussion even when initial head imaging is interpreted as negative for hemorrhage; this is especially true in the older population, in the setting of antiplatelet or anticoagulation use, or in the presence of a subdural hygroma on initial head imaging. Patients should always be counseled after head injury to seek medical advice immediately if there is any worsening of symptoms. This presentation should prompt new imaging to rule out an expanding hematoma that may contribute to long-term disability.

Take-Home Points

- Delayed acute subdural hematoma is a rare phenomenon that can develop in patients with head injury whose initial brain imaging is negative for intracranial hemorrhage
- Risk factors include older age, antiplatelet or anticoagulation use, and the presence of subdural hygromas on initial brain imaging
- Repeat brain imaging may be warranted in patients with these risk factors and persistent symptoms; head imaging is always indicated in the setting of worsening symptoms.

- Nocturnal headaches that keep patients from sleeping should prompt brain imaging to rule-out a mass lesion.

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