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CASE REPORT

Trauma

Development of a delayed chronic subdural hematoma 2 months after mild traumatic brain injury with a normal initial brain computed tomography: A case report

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Abstract

This is a case involving the development of a delayed chronic subdural hematoma 2 months after a minor head injury with normal clinical neurological findings and brain computed tomography at initial presentation. An 84-year-old man visited the emergency department (ED) after a minor head trauma. The patient complained of dizziness and vomiting 8 hours after an injury. He was not on an antiplatelet or anticoagulant. He did not have any abnormal findings during neurological examination, and brain computed tomography did not show any intracranial pathology or skull fractures. He was admitted to the ED short-stay ward for observation and was discharged asymptomatic and stable 12 hours later. However, he presented 2 months later with dizziness and unsteady gait. He was asymptomatic within those 2 months. At the ED, his brain computed tomography showed a large right chronic subdural hematoma, compressing the right lateral and third ventricles, with a 1.2 cm midline shift, subfalcine and uncal herniations, and early hydrocephalus. An emergency burr-hole evacuation was performed. He was discharged without neurological deficit 3 days later. Emergency physicians attending to patients with normal neurologic examination and initial brain computed tomography after suffering a mild traumatic brain injury should be vigilant for a chronic subdural hematoma should the patient re-present for evaluation subsequently. The attending physician may be biased as patients could have symptoms attributed to postconcussion syndrome that may overlap with symptoms of chronic subdural hematoma. Unsteady gait and ataxia are uncommon clinical signs of postconcussion syndrome and should prompt the physician to consider a repeat brain computed tomography.

KEYWORDS
burr hole, chronic subdural hematoma, surgical decompression, traumatic brain injury

1 | INTRODUCTION

The most common cause of subdural hematoma (SDH) is head trauma, with the majority attributed to motor vehicle accidents, falls, and assaults.[1]

Factors such as being elderly, history of chronic alcohol abuse, and previous traumatic brain injury (TBI) predispose to significant cerebral atrophy, which leads to a higher risk of SDH from trivial head injury or whiplash injury in the absence of physical impact.[2,3] The risk of SDH is also increased with the use of antiplatelet agents, direct oral anticoagulants, or vitamin K antagonist.[4,5]

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Patients with coagulation disorder taking anticoagulant or antiplatelet therapy have been documented to present with delayed intracranial hematoma.[6] Most of these described cases are delayed traumatic intraparenchymal or extradural hematomas.[6-8] However, the development of delayed acute SDH or delayed chronic SDH in patients without coagulation disorder or risk factors is rare and poorly understood.

This case report describes a patient with delayed chronic SDH on second brain computed tomography (CT) who did not show radiological or clinical abnormality at initial presentation. This patient was not on antiplatelet or anticoagulant therapy.

2 | CASE REPORT

An 84-year-old man presented to the emergency department (ED) for a minor head injury after he slipped and fell at ground level while trying to put on his pants after showering. There was a direct impact of his occiput against the wall. The patient was in a daze momentarily, without a loss of consciousness. He recovered spontaneously. There was no alcohol use. At 8 hours postinjury, he presented to the ED with vomiting and dizziness. There was no clinical abnormality on neurologic examinations. His vital signs were stable and Glasgow Coma Scale (GCS) was 15 (E4V5M6) and he had bilateral equal pupils. His past medical history included hyperlipidemia, glaucoma, and benign prostatic hypertrophy. He was not on antiplatelets or anticoagulants. Performed brain CT did not show any bony fracture, intracranial lesion, or mass effects (Figure 1). This was confirmed by the neuroradiologist. Laboratory tests were normal, including hematologic indexes and coagulation profile. He was admitted to the ED short-stay unit for observation. He was discharged asymptomatic and stable with no neurological deficit 12 hours later.

He re-presented 2 months later to the same ED for dizziness and unsteady gait leading to another fall. The mechanism of the second fall was exactly like the first, but there was no direct head impact the second time. In the preceding 2 months postdischarge from the first ED visit, he remained asymptomatic. At the ED, his vital signs were again stable and GCS was 15 (E3V4M5) and he had bilateral equal pupils. There was, however, broad-based, high-stepping ataxic gait on clinical examination. Brain CT performed immediately showed a large right chronic SDH compressing the right lateral and third ventricles with a 1.2 cm midline shift, subfalcine and uncal herniations, and early hydrocephalus (Figure 2). Laboratory tests were again normal, including hematologic indexes and coagulation profile. The patient was immediately referred to neurosurgery. Emergency decompression with dual burr-holes was performed 2 hours later. Brain CT performed 48 hours postsurgery showed a reduction of mass effect, no hydrocephalus, and no new intracranial haemorrhage (Figure 3). The patient was discharged on the third postoperative day with a GCS score of 15 and an absence of neurologic deficit. Despite a delay in diagnosis, the patient experienced no postoperative morbidity or sequelae.

3 | DISCUSSION

Mild TBI is head injury with GCS score between 13 and 15. Most patients with mild TBI have an uneventful recovery. Mild TBI is a complex pathophysiologic process, with symptoms largely reflecting a functional disturbance rather than a structural injury identifiable on standard neuroimaging. Of patients with mild TBI, 6%–8% have injuries on CT ranging from subarachnoid hemorrhage, subdural hemorrhage, epidural hemorrhage, cerebral contusions, and intraparenchymal hemorrhage to axonal injury such as oedema and petechiae hemorrhage. Less than 1% will require neurosurgical intervention.[9,10]

In the ED, the initial evaluation of minor head trauma is directed at the diagnosis of intracranial injury and the identification of patients requiring neurosurgical intervention. The Canadian CT Head Rule is a highly sensitive clinical decision rule for the use of CT in patients with minor head injuries. The rule allows emergency physicians to order head CT based on strong evidence and provides consistent management without affecting the patients' outcomes.[10] At first presentation, brain CT was done for this patient as his Canadian CT Head Rule score included 2 high-risk features of vomiting and age. His brain CT was normal. This was not unusual because this patient did not have additional high-risk features of chronic alcohol use, underlying coagulation disorder, or antiplatelet or anticoagulant use.

The mechanism of delayed intracranial hemorrhage is not well studied, with some postulates involving weakness of the vessel walls, an increase in venous pressure, and congestion after trauma, aggravating these previously injured extraaxial and intracerebral veins, resulting in hemorrhage.[11]

Although delayed presentation of acute or subacute SDH after minor head trauma is well documented, the development of chronic SDH after a normal CT is rare with only a few reports.[12] Historically, chronic SDH is considered a result of head trauma, causing tearing of bridging veins traversing from the brain to the draining dural-venous sinuses.[13,14] However, recent evidence suggests more complex interrelated processes involving membrane formation, angiogenesis, fibrinolysis, and inflammation. The characteristic membrane surrounding chronic SDH acts as a source of fluid exudation and hemorrhage, with angiogenic stimuli leading to the creation of fragile blood vessels within the membrane walls while fibrinolytic processes prevent clot formation resulting in continued hemorrhage. The inflammatory cells and markers within the membranes and subdural fluid propagate an inflammatory response that stimulates ongoing membrane growth and fluid accumulation.[15]

Studies from Asia have postulated that chronic SDH may be preceded by persistent traumatic subdural effusion.[16-18] The subdural effusion could be very thin and at the boundary between detectable and undetectable by head CT and may not be pointed out by a general neuroradiologist.[19] Head CT performed on the day of injury often showed normal findings or thin subdural effusions of 6 mm or less, whereas CT performed 1–7 days after trauma mainly showed thick subdural effusion. A subdural effusion that precedes chronic SDH may be near the limit of detection by CT immediately after

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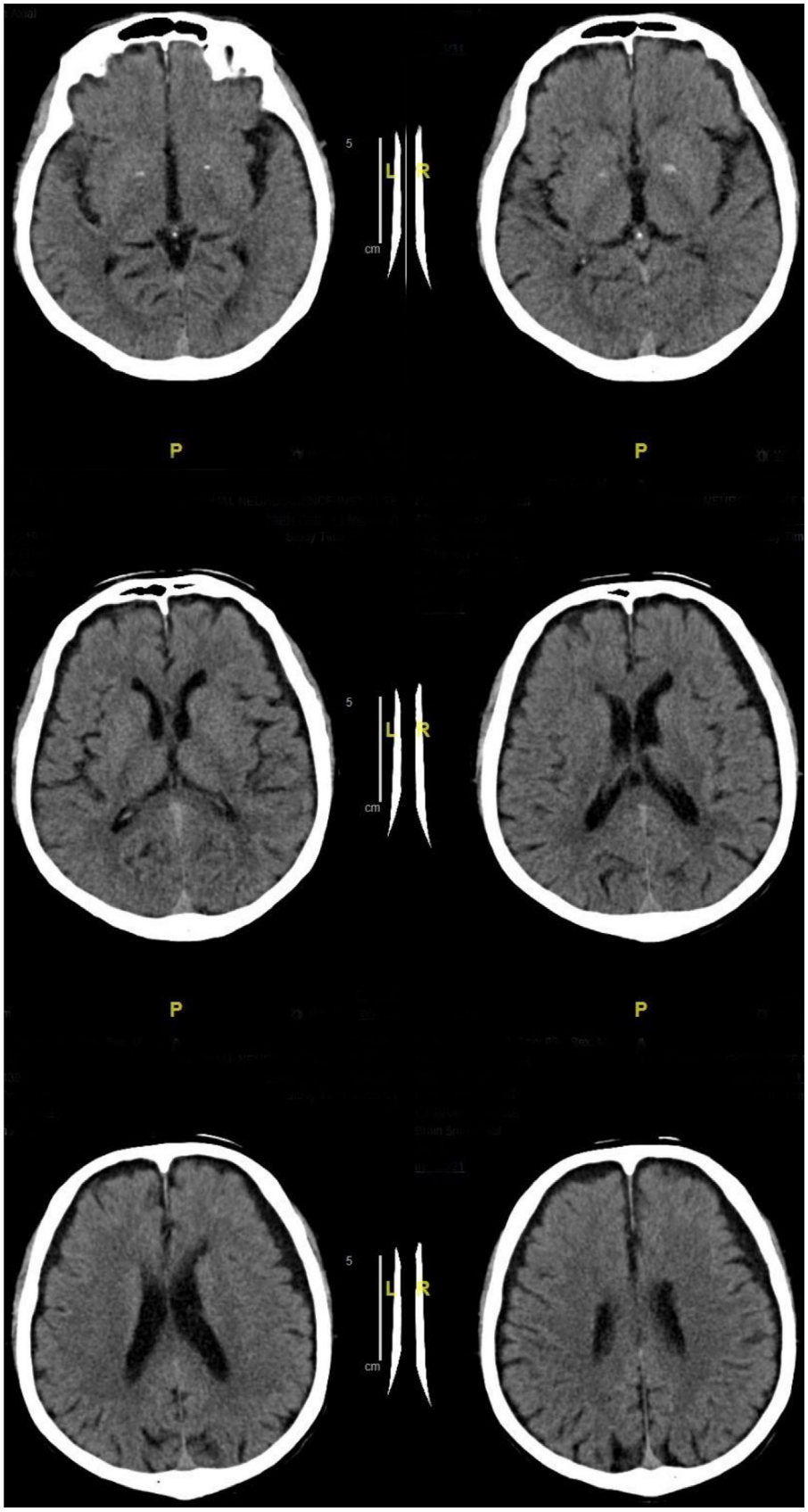

FIGURE 1 Brain computed tomography at first emergency department visit. L, left; R, right; P, Posterior

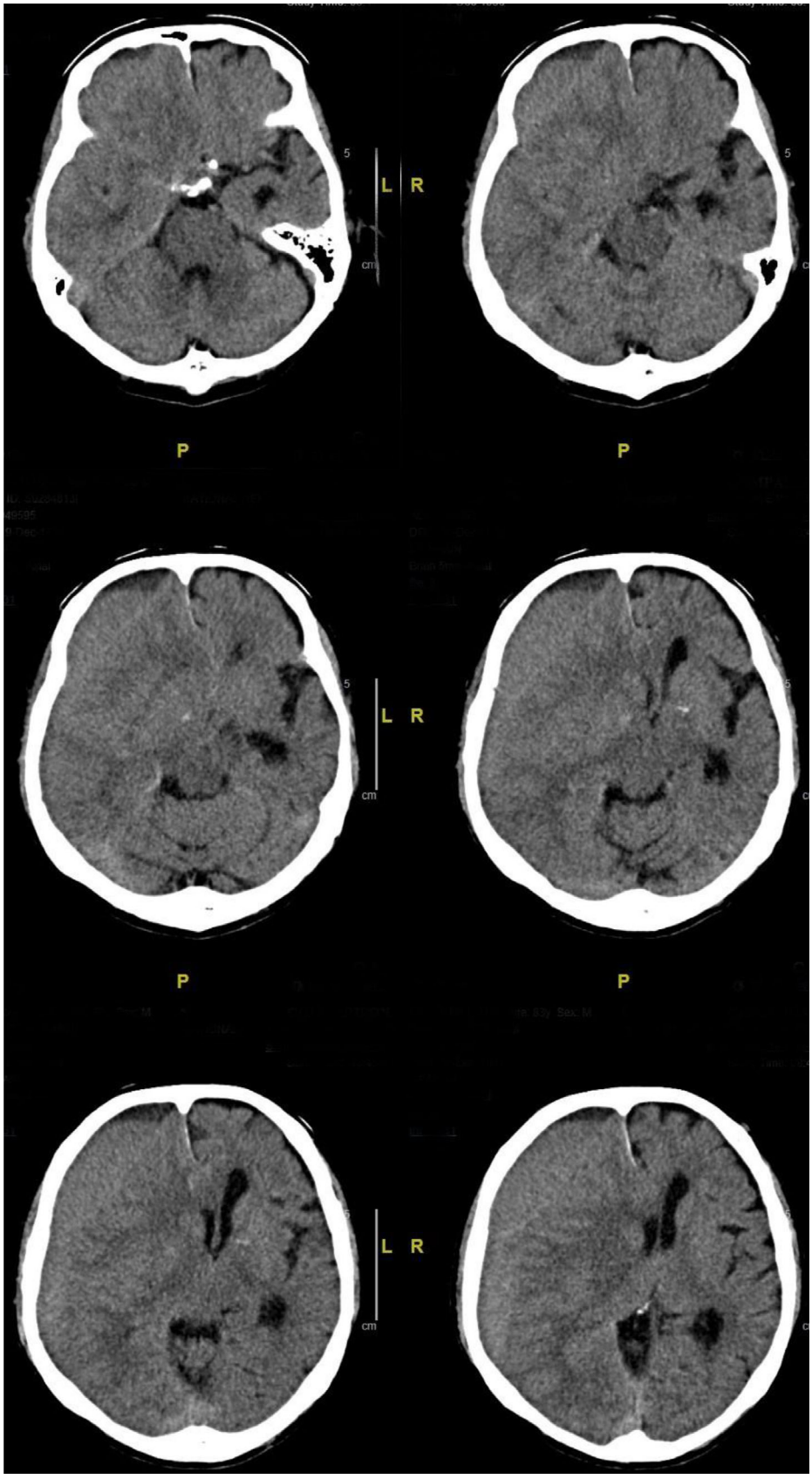

FIGURE 2 Brain computed tomography at second emergency department visit. L, left; R, right; P, Posterior

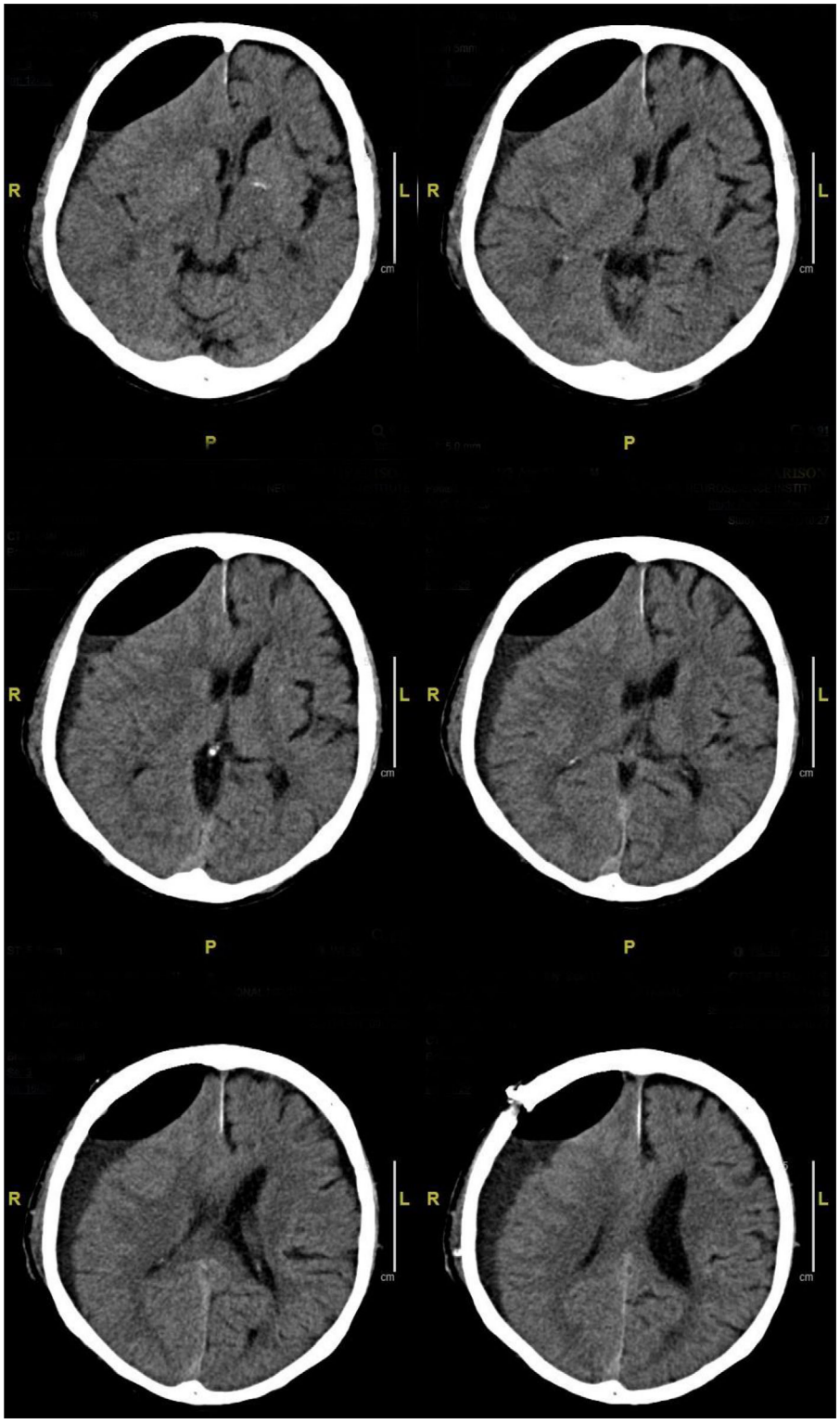

FIGURE 3 Brain computed tomography at 48 hours postoperation. L, left; R, right; P, Posterior

injury but becomes more apparent from the day after injury.[20] This patient had brain CT on the day of injury (8 hours postinjury). Although his brain CT was read by the neuroradiologist, there could be a possibility that any lesion was undetectable at that point.

As many as 30% of patients discharged from ED with mild TBI continue to have symptoms 3 months postinjury, known as postconcussive syndrome.[21] Patients with postconcussion syndrome may experience a range of symptoms during the next few days, such as difficulty concentrating, dizziness, or trouble falling asleep.[22] These symptoms can be

part of the normal healing process, and most go away over time without any treatment. Patients should be given advice to return immediately to the ED if they have worsening or severe headache, loss of consciousness, increased vomiting, or increasing confusion, seizures, weakness, or numbness. Patients should get plenty of rest and sleep and return gradually and slowly to usual routines. They should avoid alcohol and activities or sports that are physically demanding or require a lot of concentration. An appointment should be arranged for patients to attend the neurosurgery head injury clinic for follow-up.

Postconcussive symptoms of headache, dizziness, sleep disturbance, and nausea overlap with the symptoms of the rare cases of delayed intracranial hemorrhage. This couples with a clinical bias of an initial normal brain CT and normal neurologic examination and presents the emergency physician with a difficult challenge and potentially a delayed diagnosis. Fortunately for this patient, he had ataxia and an unsteady gait on second presentation, which is an uncommon symptom of postconcussive syndrome. Gait assessment could be a useful clinical marker to determine whether a patient needs a second brain CT for re-evaluation.

4 | CONCLUSION

Chronic subdural hemorrhage is not uncommon. However, delayed chronic subdural hemorrhage after a minor head trauma in which initial CT scans and neurologic examinations were normal is rare. The routine use of CT evaluation in the immediate post-traumatic period does not completely eliminate the possibility of a delayed intracranial hemorrhage. There could be symptoms overlapping with postconcussion syndrome. The onset of new neurologic signs, specifically ataxia in this patient, led to a correct diagnosis after a repeat CT scan. Although this phenomenon is uncommon, emergency physicians must be vigilant for this possibility in the face of persistent or delayed post-traumatic symptoms even if an initial CT scan is normal.

CONFLICT OF INTEREST

The author declares no conflicts of interest.

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