Journal of Integrative Neuroscience

J. Integr. Neurosci. **2022**; 21(2): 069
https://doi.org/10.31083/j.jin2102069

Case Report

Delayed-onset subdural hematoma after mild head injury with negative initial brain imaging

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Academic Editor: Charles J. Prestigiacomo
Submitted: 28 April 2021 Revised: 22 July 2021 Accepted: 22 July 2021 Published: 23 March 2022

Abstract

Mild head injuries are commonly encountered in the neurosurgical field and emergency room (ER). The usual step is to discharge if the mental status of the patient is good and the initial brain computed tomography (CT) findings are normal. Here, we report a rare case of an 82-year-old male patient who developed delayed-onset bilateral subdural hematoma five weeks after a mild head injury. He was not on anticoagulant or antiplatelet therapy. The initial CT scan on the day of injury and magnetic resonance (MR) imaging performed seven days after the injury did not reveal any intracranial pathology or skull fracture. However, he presented with severe headaches and an unsteady ataxic gait five weeks later. Brain CT revealed bilateral subdural hematoma compressing the lateral ventricles with a midline shift to the right side. The possible pathophysiological mechanisms underlying this uncommon entity are discussed with a review of the relevant literature.

Keywords: Computed tomography; Delayed; Magnetic resonance image; Subdural hematoma; Trauma

1. Introduction

Traumatic head injury is one of the most common reasons for neurosurgical consultations in the emergency room (ER). The usual step is to discharge if the initial brain computed tomography (CT) is normal and the patients do not show any abnormal neurological findings [1]. However, a few patients may develop delayed-onset intracranial bleeding [2]. In general, Glasgow Coma Scale (GCS) is used when evaluating a patient's level of consciousness after traumatic head injury. The GCS consists of three objective tests, eye (E), verbal (V), and motor (M) response, with the lowest score being 3 points and the highest score being 15 points [3]. GCS is a very useful score to determine the prognosis of a patient according to the level of consciousness. A GCS of 7 or less is considered to be a coma, and indicate severe injury. Most cases of delayed-onset traumatic intracranial hemorrhages occur in patients with medical comorbidities related to coagulation disorders who are taking anticoagulant or antiplatelet agents [4]. Therefore, delayed-onset intracranial hemorrhage in patients without coagulation disorders or related risk factors is rarely reported and poorly understood. Here, we report the case of a patient with delayed-onset bilateral subdural hematoma (SDH) who did not show abnormal findings on the initial radiological CT on the day of injury and magnetic resonance imaging (MRI) performed seven days after the mild head injury. He had no coagulation disorders or medical comorbidities related to the bleeding tendency.

2. Case presentation

An 82-year-old man was admitted to the emergency room (ER) for nausea and dizziness after a mild head injury after slipping and falling in the bedroom while putting on his pants. He was dazed, but there was no loss of consciousness or posttraumatic amnesia. His vital signs were stable, and neurological examination findings were within normal limits (GCS 15-E4V5M6). An initial CT scan of the brain showed no signs of skull fracture or intracranial hemorrhage (Fig. 1A). He had no past history of medical comorbidities. The results of routine laboratory tests, including platelet count, prothrombin time, and activated partial thromboplastin time, aspartate transaminase (AST), alanine transaminase (ALT) and serum albumin were within normal limits. The patient was not on antiplatelet or anticoagulant therapy. He was discharged from the ER on the day of the injury.

He revisited the neurology department seven days later for persistent dizziness and mild headache. To rule out delayed hemorrhage or other intracranial pathologies, a neurologist performed MRI, which did not show any intracranial lesion or mass effects (Fig. 1B). He was discharged with no neurological deficits. However, he reported five weeks after the first visit to the ER for aggravated dizziness, continuous headache, and unsteady gait that had started five days earlier. He had no history of any other head injuries.

During the immediate five weeks after his discharge after the first ER visit, he remained symptomatic and com-

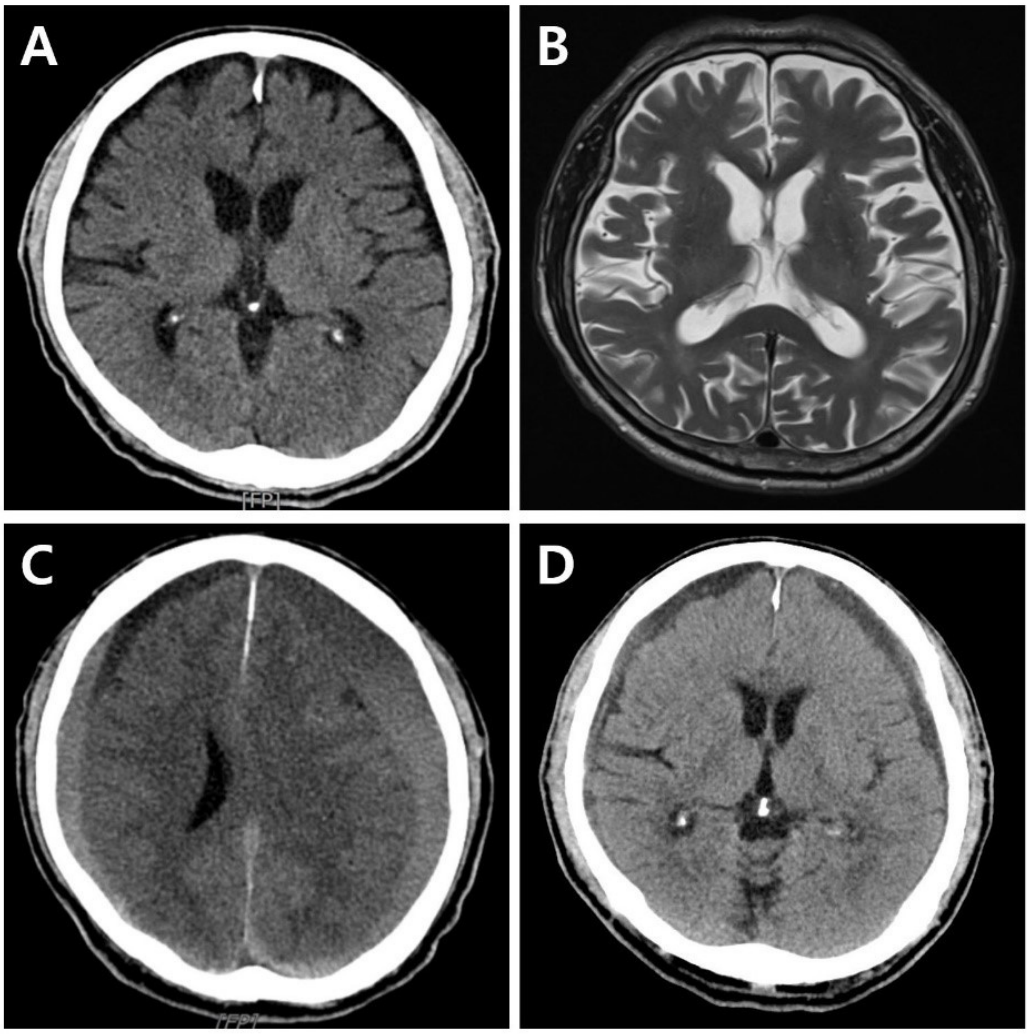

Fig. 1. Brain images of the patient. (A) Brain computed tomography scan obtained on the day of injury shows no intracranial pathology. (B) T2-weighted axial image obtained seven days after the mild head injury shows no intracranial bleeding. (C) Brain computed tomography scan obtained five weeks after the head injury demonstrates bilateral chronic subdural hematoma (SDH) with midline shifting. (D) Brain computed tomography scan obtained 2 months after burr-hole trephination reveals residual SDH in both frontoparietal areas but improved midline shifting.

plained of persistent dizziness and headache. At the ER, his vital signs were restabilized, and his GCS score was 15 (E4V5M6). However, clinical examination showed a broad-based low-stepping ataxic gait. Brain computed tomography (CT) showed bilateral subdural hematoma compressing the lateral ventricles with a midline shift to the right (Fig. 1C). Simultaneous hematoma evacuations through burr-hole trephination with 5-L catheters were performed under local anesthesia. He fully recovered, and his unsteady gait improved immediately after the hematoma was evacuated. He was discharged on the seventh postoperative day with no neurological deficits. Follow-up CT 2 months after the burr-hole trephination showed a reduction of the hematoma with improved midline shifting despite some residual SDH (Fig. 1D).

3. Discussion

Recently, the GCS is widely used to assess the state of consciousness in general patients with reduced consciousness. Usually, 3 to 8 points are defined as severe, 9 to 12 points are moderate, and 13 to 15 points are defined as mild traumatic brain injury (TBI) [3]. Most patients with mild TBI have a good recovery, and less than 1% require neurosurgical intervention [5,6]. In the ER, the sequential evaluation of a minor head injury is dependent on the neurologic condition of the patients and directed at the diagnosis of intracranial pathology and the identification of patients with medical comorbidities requiring neurosurgical intervention. Usually, the common practice is to discharge if the findings of the initial brain CT or MRI are normal. However, in rare cases, delayed intracranial hemorrhage may occur, especially in patients receiving antiplatelet or

anticoagulant therapy. The exact mechanisms underlying delayed intracranial hemorrhage are not well-documented, but vessel wall weakening, increased venous pressure, and congestion have been postulated [4].

Although delayed-onset acute SDH after mild head injuries is well-documented, the development of subacute or chronic SDH after the initial normal CT or MRI is rare; there have only been a few reports [7,8]. Historically, subacute or chronic SDH is regarded as a result of head injury causing tearing of bridging veins. As the cerebral veins cross the subdural space, they have little supporting structure, and are therefore more vulnerable to brain injury at this point. Subdural bridging veins are stretched and traversed in elderly patients with brain atrophy. Recent studies have suggested complex interrelated processes underlying membrane formation, angiogenesis, fibrinolysis, and inflammation during the development of delayed-onset SDH [7,9]. The membrane surrounding the subdural effusion acts as a source of fluid exudation and hemorrhage, and angiogenic stimuli drive the development of weakened blood vessels within the membrane walls while fibrinolytic processes limit clot formation, which results in persistent hemorrhaging [9].

The role of preceding traumatic subdural effusion in the development of chronic SDH has attracted attention [10]. Subdural effusion may be very thin and undetectable by brain CT on the day of injury, whereas CT obtained 1–7 days after a head injury may show a thick subdural effusion. However, the brain CT of our patient on the day of injury showed no intracranial pathology, and MRI did not reveal a thick subdural effusion seven days after the mild head injury. Approximately 30% of patients discharged from the ER with mild head injuries continue to have symptoms that usually last for several months; this characterizes the well-known post-concussion syndrome [11].

Various symptoms, including headache, dizziness, sleep disturbance, and fatigue, may be experienced, and they can be regarded as part of the normal healing process after head injury. Therefore, post-concussive symptoms may be confused with symptoms of delayed intracranial hemorrhage. An initial normal brain CT and follow-up MRI showing no significant abnormalities or deficits may lead to the neglect of other clinical symptoms by neurosurgeons or neurologists and a delay in diagnosis. Fortunately, our patient had obvious gait disturbance, which is a rare symptom of post-concussive syndrome that can be useful for identifying patients requiring further evaluation.

4. Conclusions

Although rare, neurosurgeons and neurologists should consider delayed-onset SDH based on clinical symptoms, even if the findings of initial CT and follow-up MR imaging are normal. Especially, even if the patient don't have a predisposition to cause delayed intracranial hemorrhage (liver cirrhosis, coagulation disorder, or taking anticoagulants), it can be slowly occured in the elderly patient. Therefore, in the case of elderly patient, it is important to check and observe the neurological condition frequently through short-term follow-up visits, even if the initial brain images are normal finding after mild head injury.

Abbreviations

ALT, alanine transaminase; AST, aspartate transaminase; CT, computed tomography; ER, emergency room; GCS, Glasgow Coma Scale; MR, magnetic resonance; SDH, subdural hematoma; TBI, traumatic brain injury.

Author contributions

Research design—HGK and SWK; Data collection and analysis—HGK and SWK; Computational studies and manuscript writing—HGK and SWK. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

Written informed consent was obtained from the patient for publication of this case report and the accompanying images.

Acknowledgment

Not applicable.

Funding

The present study was supported by grants from the Clinical Medicine Research Institute at Chosun University Hospital (2019).

Conflict of interest

The authors declare no conflict of interest.

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