Case Report

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Traumatic Isolated Interhemispheric Fissure Subdural Hematoma Under Anticoagulant Therapy in an Elderly Patient: A Case Report

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Received: 21 August 2024; **Revised:** 27 October 2024; **Accepted:** 03 November 2024; **Published:** 16 November 2024

Abstract

Acute subdural hematomas are often encountered over the convexity of the cerebral hemisphere, but the localization of the interhemispheric fissure is rare. Interhemispheric fissure subdural hematoma (IHSDH) is usually accompanied by subdural hematoma, intracerebral hemorrhages, while its isolated form is extremely rare. They are usually benign due to the fact that they are usually treated conservatively and are not directly correlated with an increase in intracranial pressure. In cases which develop under the use of antiplatelets or anticoagulants, it is recommended to discontinue the drug for a while so that the hematoma does not grow in size. There is no clear answer in which patients the drug will be discontinued and how long the use will be interrupted.

Keywords: ***Acute interhemispheric fissure subdural hematoma, acute subdural hematoma, intracerebral hematoma, head trauma.***

Introduction

Subdural hematoma usually occurs from venous origin. It is caused by rupture of the bridging veins that extend from the surface of the brain to the dural sinuses. Most subdural hematomas are localized over lateral cerebral convexities [1]. IHSDH can be seen with subdural hematoma and intracerebral hematomas, but its isolated form is extremely rare. Approximately 80-90% of the hematomas are in the occipital lobe. Although it is usually traumatic, it can also occur spontaneously in elderly patients using antiaggregants or anticoagulants [2]. Hematoma is mostly detected by brain computer tomography(CT), brain magnetic resonance imaging(MRI) is more sensitive for thin subdural hematoma or IHSDH. Antiplatelet and anticoagulant drugs are usually discontinued in the presence of hematoma because of the risk of increasing the size of the hematoma and potentially increasing intracranial pressure [3]. There are no guidelines for the management of antithrombotic drugs in patients with IHSDH. Further studies are needed.

Case Report

A 74-year-old female patient was brought to the emergency department due to a fall while in nursing home. She had a known history of dementia, atrial fibrillation and essential hypertension. She was taking rivaroxaban 15 mg/day, memantine 10 mg/day, quetiapine 50 mg/day and perindopril 5 mg/day. When she was brought to the emergency department, her glasgow coma scale was 10. Neurological examination revealed no lateralizing findings. There was a 2 cm open skin wound in the frontal region. Brain CT was performed due to the low score on the glasgow coma scale, which developed after the trauma. A subdural hematoma measuring 6.7 mm was detected posteriorly in the interhemispheric fissure [**Figure-1**]. There is no skull fracture detected. Any aneurysms, venous malformations or other pathologies were excluded on brain angiography imaging. After 4 hours, a control tomography was performed, no increase in the size of the subdural hematoma was detected. There was no indication for the operation by neurosurgery. She was evaluated by ultrasonography and elbow tomography due to the fact that she had subcutaneous ecchymosis on her left arm. No fracture was detected on the elbow tomography. On ultrasound, a hematoma measuring 5 centimeters was detected, starting from the middle section of the arm in the proximal direction and extending to the middle section of the forearm in the distal direction. Upper extremity elevation, cold compress therapy and paracetamol were applied. Rivaroxaban used for atrial fibrillation was discontinued for 4-6 weeks according to cardiology recommendations. Treatment was continued with a low dose of low molecular weight heparin. The glasgow coma scale score increased to 14 at the follow-up of the patient. The patient's subcutaneous ecchymosis was significantly reduced. 10 days later, she was discharged due to the fact that the subdural hematoma was resorbed on the control CT [**Figure-2**].

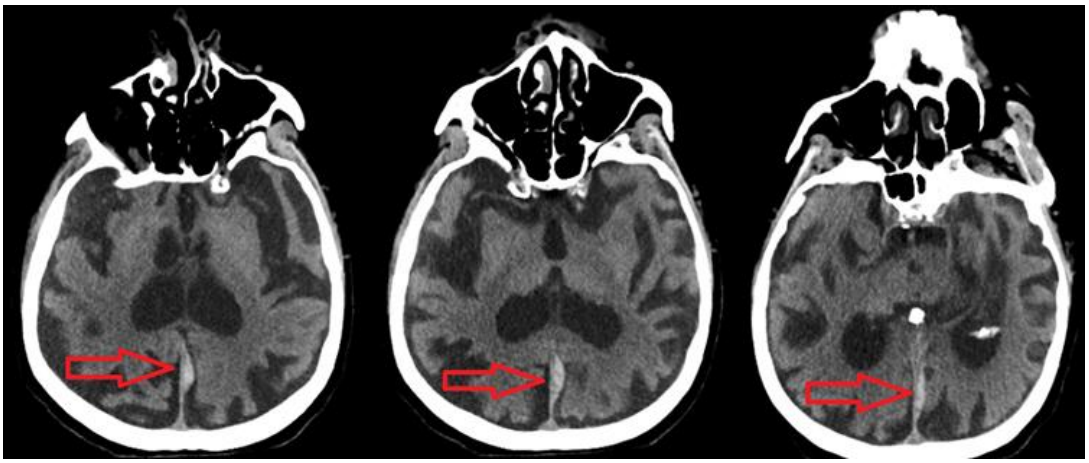

Figure-1: Acute interhemispheric fissure subdural hematoma (red arrows) in brain CT

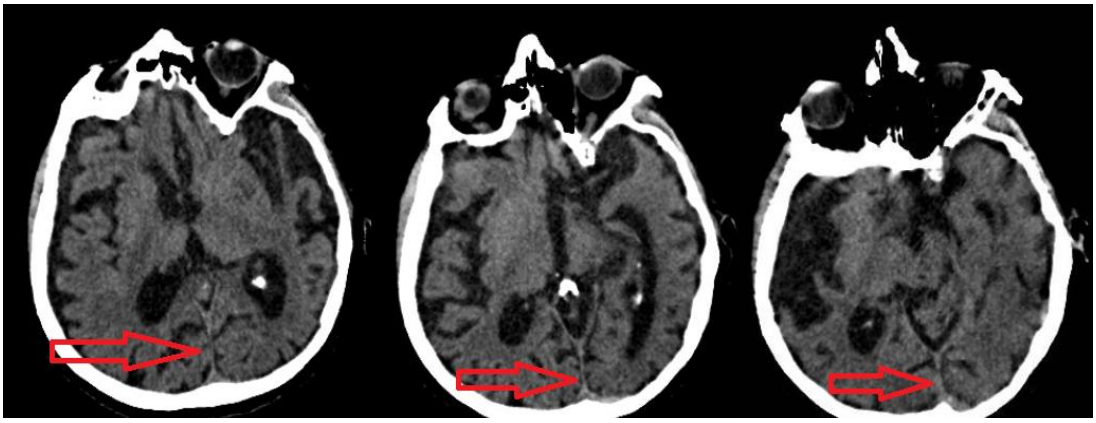

Figure-2: Acute interhemispheric fissure subdural hematoma was resorbed (red arrows)

Discussion

Subdural hematoma is usually caused by blood filling between dura and arachnoid membrane after stretching and tearing of bridging veins or vessels. Most subdural hematomas are localized over lateral cerebral convexities, but subdural blood can also collect on the medial surface of the hemisphere, between the tentorium and the occipital lobe, between the temporal lobe and the skull base or posterior fossa. The elderly with cerebral atrophy and alcohol abusers are predispose to subdural hematoma [4,5].

Subdural hematomas can be classified into subtypes according to whether they are caused by trauma or not. In a retrospective cohort study in which 27,000 patients were evaluated, it was reported that 71% of the patients were traumatic and 29% were non-traumatic [6]. The incidence increases with aging. Coagulopathies, alcohol abuse, anticoagulant therapy are predisposing factors. Aneurysm, cerebral venous malformations, tumor, post-surgical and intracranial hypotension are among the rare causes [7].

IHSDH is a rare type of subdural hematoma due to the unusual localization. It usually occurs in the elder patients with bleeding disorders and accounts 6% of traumatic subdural hematomas [8]. In most patients, bleeding occurs in the occipital lobe. Although brain CT is usually sufficient for the diagnosis of thick subdural hematoma, brain MRI is more sensitive for thin IHSDH that cannot be detected on tomography [9,10].

Contrary to expectation, the mortality rates of patients with IHSDH hematoma were reported to be lower than those with acute subdural hematoma. IHSDH does not correlate with increase in intracranial pressure due to its localization and the fact that most patients can be treated conservatively are factors that reduce mortality. For this reason, IHSDH can be considered a benign variant of acute subdural hematoma [11,12]. Although it is mentioned as a benign form of acute subdural hematoma, the patients' glasgow coma scale score and mean hematoma thickness are among the poor prognostic factors [13].

The use of antiplatelet or anticoagulant therapy has increased in the elderly population due to comorbid conditions. There is no definitive guide on how to manage the medication in patients with acute IHSDH, which develops under the use of these antithrombotic drugs. In the presence of subdural hematoma in patients with atrial fibrillation, previous ischemic stroke, and previous myocardial infarction, patients face embolic events if antithrombotic drugs are discontinued. The use of antithrombotic drugs is considered an obvious risk factor for the growth of a small acute subdural hematoma, but it is not known how much the subdural hematoma will expand, whether it will increase intracranial pressure. Lavrov and Rappaport retrospectively analyzed the experience of conservative treatment of patients with acute subdural hematoma. Of the 21 patients who used antiaggregants or anticoagulants, 20 reported that surgical drainage was performed due to chronic subdural hematoma. The hematoma thickness of these patients was higher than those who did not undergo surgical drainage [14]. We discontinued rivaroxaban, but continued low-dose low molecular weight heparin therapy to protect the patient from cardiovascular risks. We detected almost complete resorption in the control computer tomography imaging, which was evaluated 10 days later.

Acute IHSDH can be accompanied by subdural hematoma, intracerebral hemorrhages, subarachnoid hemorrhages, ventricular hemorrhage and pneumocephalus, but its isolated form is very rare. Most patients are treated conservatively, usually because of spontaneous resolution of the hematoma. Surgical treatment may be preferred if the hematoma is enlarged, the clinic worsens, paraplegia develops, the hematoma volume is more than 40 mL, and the hematoma thickness is more than 15 mm [15].

Conclusion

Isolated IHSDH is rarely seen due to unusual localization. Thin hematomas may not be obvious on CT scan compared to prominent thick hematomas, in which case brain MRI is helpful. It has less mortality and worse outcome than acute subdural hematomas. A low score on the Glasgow coma scale and thick hematomas have a worse prognosis. Although antiplatelet and anticoagulant therapy seems to cause enlargement of even small hematomas, more studies are needed for treatment strategies.

Declarations

Ethical Approval

As per international standard or university standard written ethical approval has been collected and preserved by the author.

Study Funding

The authors received no financial support for the research, autorship, and/or publication of this article.

Competing of Interest

The authors declare that they have no known competing financial or personal relationships that could have appeared to influence to work reported in this paper.

Consent

As per international standard or university standard, patient's written consent has been collected and preserved by the authors.

Competing Interests

Authors have declared that no competing interests exist.

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