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Case Report

A case of chronic subdural hematoma demonstrating the epileptic focus at the area with sulcal hyperintensity on fluid-attenuated inversion recovery image

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ABSTRACT

Although the sulcal hyperintensity on fluid-attenuated inversion recovery (FLAIR) images is detected in some chronic subdural hematoma (CSDH) cases, its clinical significance remains determined. A 77-year-old man with symptomatic CSDH presented with generalized tonic-clonic seizures at 9 days after surgery. ^{123}I-iomazenil -single photon emission computed tomography revealed transient reduction in cortical benzodiazepine receptors binding potential at the region corresponding to that of the sulcal hyperintensity on FLAIR images. Findings of sulcal hyperintensity on FLAIR imaging under the CSDH may have a relation with the cause of epileptic seizure.

Introduction

Epileptic seizures are one of the major complications associated with chronic subdural hematoma (CSDH), either in the pre- or postoperative period [1]. To date, alcohol abuse, change of mental status, and density of hematoma on computed tomography (CT) have been suggested as risk factors for epileptic seizures in patients with CSDH, excluding detectable intraparenchymal lesions on neuroimaging [2]. As a specific ligand for neuronal-type benzodiazepine receptors, ^{123}I-iomazenil (IMZ) is used as a tracer in single photon emission CT (SPECT) to detect original epileptic foci more clearly than tracers of cerebral blood flow such as

Abbreviation: CSDH, chronic subdural hematoma; CT, computed tomography; IMZ, ^{123}I-iomazenil; BRBP, benzodiazepine receptor binding potential; SPECT, single photon emission computed tomography; FLAIR, fluid-attenuated inversion recovery; CSF, cerebrospinal fluid; MRI, magnetic resonance imaging.

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^{123}I-iodoamphetamine [3]. IMZ-SPECT is associated with neural integrity in the cerebral cortex, and a reduction in cortical benzodiazepine receptor binding potential (BRBP) thus indicates cortical neural damage [4,5]. However, images obtained within 23 minutes after tracer administration (early images) also correlate with brain perfusion images [6].

Sulcal hyperintensity on fluid-attenuated inversion recovery (FLAIR) images has been reported in patients with abnormal cerebrospinal fluid, such as those with meningitis, meningeal carcinomatosis, and subarachnoid hemorrhage [7]. Also, the frequent association with the dirty cerebrospinal fluid sign suggests an increase in the blood pool and a small amount of protein leakage [7]. However, the relationship between epileptogenesis and this radiological finding in CSDH patients remains unknown.

We report a case of CSDH with symptomatic epilepsy 9 days after burr hole surgery, demonstrating transient reduction in uptake of cortical BRBP, corresponding with the region showing sulcal hyperintensity on FLAIR images.

Case report

A 77-year-old man presented with progressive gait disturbance and left-side hemiparesis. He had no history of major traumatic head injury. CT showed right-sided CSDH but no other lesions. Single burr hole drainage was performed under local anesthesia on the day of admission. The preoperative neurological deficit disappeared and the patient was discharged from our hospital 7 days after surgery. However, he was brought back to our hospital by ambulance with generalized tonic-clonic seizures on postoperative day 9. CT images showed no enlargement of hematoma, but did show a new mass sign in brain parenchyma. Magnetic resonance imaging performed 6 hours after the seizure revealed sulcal hyperintensity at the convexity underlying the hematoma on FLAIR image (Fig. 1A,B), but T1-, T2-, T2*-, and diffusion-weighted imaging revealed no abnormal findings in the brain parenchyma (Fig. 1C-E). The patient was treated with levetiracetam at 1000 mg/day to prevent further seizure activity and was free from seizures without side effects on the day after readmission, although postictal left hemiparesis (Todd's palsy) and confusional state continued after seizure onset. IMZ-SPECT was performed 2 days after seizure onset (Fig. 2). Scanning was started immediately (early images) and at 180 minutes (delay images) after intravenous injection of 167 MBqIMZ; the duration of each scan was 20 minutes. On early images, tracer uptake was increased in the right cerebral hemisphere compared with the contralateral cerebral hemisphere. On delayed images, tracer uptake was relatively

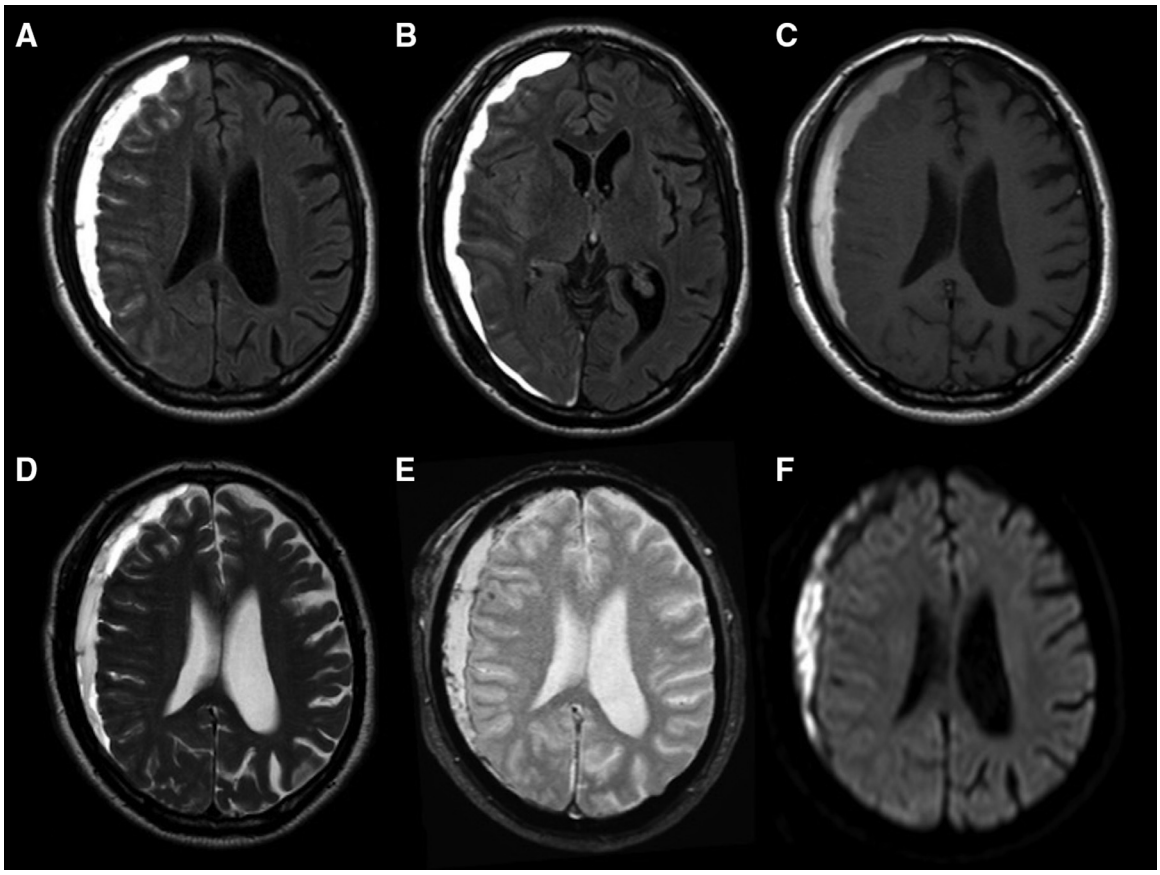

Fig. 1 – MRI obtained 6 hours after seizure onset. Fluid-attenuated inversion recovery (FLAIR) image at the level of the body of the lateral ventricle shows sulcal hyperintensity at the convexity underlying the hematoma (A), but the FLAIR image at the level of the anterior horn of the lateral ventricle shows no sulcal hyperintensity (B). T1-, T2-, T2*- and diffusion-weighted imaging revealed no abnormal findings in the brain parenchyma (C-F).

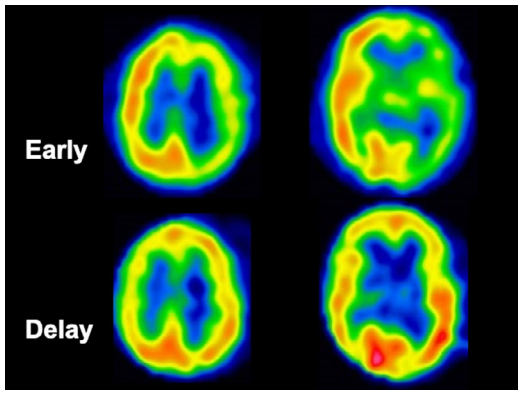

Fig. 2 – ^{123}I-iomazenil single photon emission computed tomography images 2 days after seizure onset. Early images (upper row) reveal an increase in tracer uptake in the right cerebral hemisphere compared with the contralateral hemisphere. Delayed images (lower row) reveal a decrease in tracer uptake in the right cerebral hemisphere compared with the contralateral hemisphere, corresponding to the region of sulcal hyperintensity on fluid-attenuated inversion recovery images.

decreased in the right cerebral hemisphere at the level of the body of the lateral ventricle, corresponding with the region of sulcal hyperintensity on FLAIR images, despite the symmetric uptake evident at the level of the anterior horn of the lateral ventricle where no sulcal hyperintensity was seen on FLAIR images. The neurological deficit resolved completely 1 week after seizure onset. Sulcal hyperintensity on FLAIR images disappeared over the course of 6 weeks (Fig. 3). Increases in cerebral blood flow and decreases in cortical BRBP in the right cerebral hemisphere on IMZ-SPECT also normalized over these 6 weeks (Fig. 4). We diagnosed epilepsy on the basis of clinical symptom presenting a partial seizure with secondary generalized and with normal laboratory data at the onset of the seizure. Therefore, electroencephalography was not necessarily performed for this patient. The patient has been followed without either recurrence of seizure or any neurological deterioration.

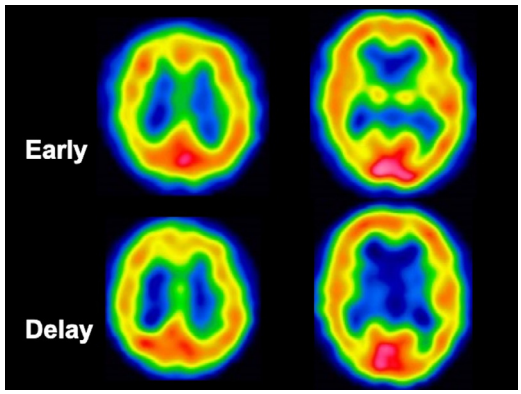

Fig. 4 – ^{123}I-iomazenil single photon emission computed tomography images 6 weeks after seizure onset, showing disappearance of laterality.

Discussion

We have reported here a case of CSDH with symptomatic epilepsy 9 days after burr hole drainage, demonstrating transient reduction in uptake of cerebral BRBP, corresponding geographically with the region of sulcal hyperintensity on FLAIR images.

Interestingly, uptake of BRBP was decreased only in the area corresponding to the sulcal hyperintensity on FLAIR images taken 2 days after seizure onset. Low uptake of BRBP subsequently showed gradual normalization over 6 weeks after seizure onset, suggesting transient suppression of neuronal activity due to epileptic discharges.

In only 1 cohort study, the higher prevalence of symptomatic epilepsy following drainage of CSDH in a patient with

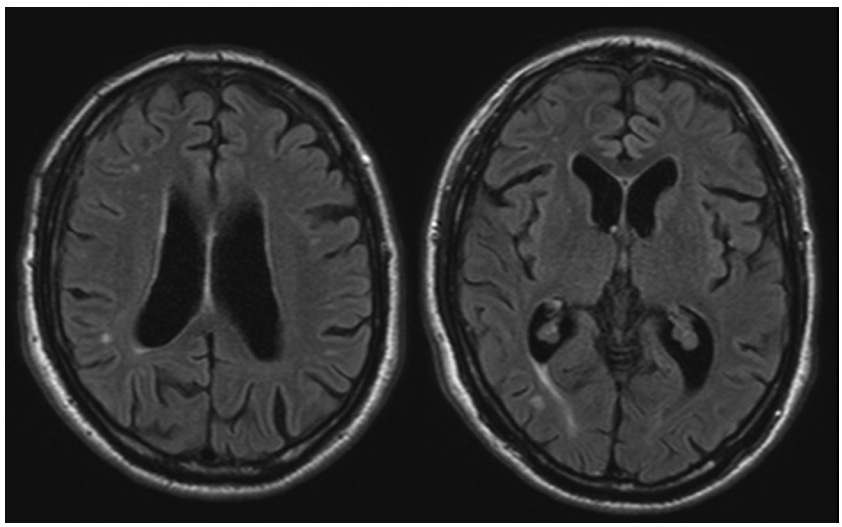

Fig. 3 – MRI images 6 weeks after seizure onset, showing disappearance of sulcal hyperintensity on Fluid-attenuated inversion recovery images.

sulcal hyperintensity on FLAIR was indicated, although no topographic relationship between epileptic focus and this sulcal finding on FLAIR image had been demonstrated [8]. The sulcal hyperintensity on FLAIR images has been reported in some pathological states, such as those with meningitis, meningeal carcinomatosis, blood collection, and a small amount of protein leakage in the subarachnoid space [7]. In the present case, meningitis or meningeal carcinomatosis could be excluded based on the uneventful clinical course following seizure treatment alone. The microscopic features of CSDH reveal many fresh erythrocytes and higher fibrinogen composition, indicating high fibrinolytic activity and recurrent hemorrhage [9,10]. Furthermore, fibrin degeneration products cause angiogenic activity in the outer membrane rather than the inner membrane of CSDH, whereas the outer membrane contains many fragile sinusoidal vessels that often are a source of repeated multifocal bleeds [11]. Therefore, since the outer membrane of CSDH has higher vascular permeability, the blood or protein content might pass through the CSDH membrane and influence the brain parenchyma to induce seizures. To the best of our knowledge, the present case offers the first demonstration of a clear relationship between epileptic focus and this sulcal finding on FLAIR images following drainage of the CSDH.

Two mechanisms can be considered. First, an epileptic discharge due to postoperative hyperemia can be proposed. Ogasawara et al reported that transient hyperemia immediately after drainage of CSDH was caused by dysautoregulation, inducing temporary agitated delirium or seizure onset [12]. However, the present case did not involve acute agitated delirium in the postoperative acute state, so sustained postoperative hyperemia seems unlikely to have induced the seizure. Second, the epileptogenic area corresponding to that with sulcal hyperintensity on FLAIR might be in an activated electrophysiological state, with the activated area exhibiting a hypermetabolic state, thereby causing compensatory regional hyperperfusion. From the perspective of radiological findings, sulcal hyperintensity on FLAIR imaging gradually disappeared over the postictal period in the present case. Furthermore, rapid neurological improvement without any agitated delirium was observed in the postoperative period. The activated electrophysiological state in sulcal hyperintensity on FLAIR images thus seemed to lead to cerebral hyperemia and seizure onset.

Conclusion

We have reported a case of CSDH in which the relationship between the epileptic focus and sulcal hyperintensity on FLAIR images was radiologically confirmed. Although further studies are required to confirm our findings, meticulous observation or prophylactic antiepileptic medication may be required for patients showing sulcal hyperintensity on FLAIR images.

Declarations of interest

None.

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