Case Report

A case of paroxysmal homonymous hemianopsia: Uncommon presentation of nonconvulsive status epilepticus

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ABSTRACT

Paroxysmal homonymous hemianopsia (HH) is uncommon presentation of epilepsy. We demonstrate a rare case of paroxysmal HH that was diagnosed by magnetic resonance (MR) arterial spin-labeling (ASL). A 82-year-old woman presented with abrupt onset of isolated visual field abnormality without convulsive epilepsy at 16 days after a traumatic head injury. Diffusion weighted and MR-ASL obtained on admission revealed hyperintensity and hyperfusion in the right temporo-occipital cortex. Nonconvulsive status epilepticus was suspected. The patient was treated with oral levetiracetam and the symptoms resolved in 3 days. Paroxysmal HH should be considered in patients who present with simple partial epilepsy, and MR-ASL imaging may assist in the differential diagnosis of these patients.

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Introduction

A variety of symptoms have been associated with seizure; however, persistent pure visual defect has been rarely reported [1,2]. Because the tempo of onset of seizure mimics that of occipital stroke, an accurate diagnosis is important to avoid the interventions associated with ischemic stroke. Here, we demonstrate a rare case of paroxysmal homonymous hemianopsia (HH) following mild traumatic brain injury (TBI) and suggest the utility of arterial spin-labeling (ASL) resonance imaging in its diagnosis.

Abbreviation: ADC, apparent diffusion coefficient; aSDH, acute subdural hematoma; ASL, arterial spin labeling; CBF, cerebral blood flow; CT, computed tomography; DWI, diffusion weighted imaging; EEG, electroencephalogram; FLAIR, fluid-attenuated inversion recovery; HH, homonymous hemianopsia; IMZ, [123]I-iomazenil; MR, magnetic resonance; SPECT, single photon emission computed tomography; TBI, traumatic brain injury.

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Case report

An 82-year-old woman was admitted to the Emergency Department after being hit by a motor vehicle when crossing the street by bicycle. She has well controlled hypertension, but had no previous history of ischemic event, epilepsy, or persistent atrial fibrillation. On admission, her Glasgow Coma Scale score was 15 and no focal neurological abnormality was noted. Computed tomography (CT) revealed traumatic subarachnoid hemorrhage and thin acute subdural hematoma (aSDH) in the right temporo-occipital and parieto-occipital regions, respectively. Magnetic resonance (MR) imaging performed 9 days after admission revealed brain contusions in the right temporo-occipital lobe and deep white matter, and thin aSDH on fluid-attenuated inversion recovery (FLAIR) imaging. There was no diffusion restriction on diffusion weighted imaging (DWI). Confrontation visual field test revealed no abnormality. Fifteen days after admission, the patient was discharged from hospital without neurological deficits but was returned to our hospital by ambulance following abrupt onset of abnormal visual field symptoms on the day of discharge.

On readmission, the patient did not have any alteration in mental state or convulsive epilepsy. CT showed no enlargement of the subdural hematoma or new mass sign in the brain parenchyma. Emergency MRI performed 4 hours after the abrupt onset of abnormal visual field symptoms revealed cortical hyperintensity in the right temporo-occipital region on FLAIR (Fig. 1A), and DWI revealed corresponding a hyperintensity in this area (Fig. 1B) with slightly low ADC value (Fig. 1C). MR angiogram showed no steno-occlusive arteries (Fig. 2). Subsequent ASL imaging revealed hyperperfusion of cerebral blood flow (CBF) in the right temporo-occipital region (Fig. 1D). All MR images were acquired with a GE Optima MR450W 1.5T MRI scanner. Labeling with pulsed continuous ASL, data collection was performed using 3D fast spin echo. ASL sequence parameters are post label delay = 1500 (short), 2500 (long) msec, TR = 4640 msec, FOV=240 mm, slice thickness = 4mm. IMZ-SPECT images were acquired the day after seizure onset. Late images showed a decrease in tracer uptake in the right cerebral hemisphere compared with the contralateral hemisphere at the corresponding region of hyperintensity on FLAIR images, indicating reduction in cortical benzodiazepine receptor binding potential (Fig. 3A). Those findings enabled differentiation of simple focal epilepsy from ischemic stroke. Nonconvulsive status epilepticus was suspected as an exclusion diagnosis, and oral levetiracetam (1000 mg/day) was given immediately after MRI acquisition. Goldmann visual field examination on the day of readmission

Fig. 1 – MRI acquired on readmission. FLAIR (A) and DWI (B) image shows hyperintensity at the convexity underlying a subdural hematoma (white arrows) that corresponds with an area. Note ADC map (C) shows slightly low and ASL image (D) shows hyperperfusion of CBF in the right temporo-occipital region.

Fig. 2 – MR angiogram shows no steno-occlusive arteries.

Fig. 3 – Late IMZ-SPECT images at the day after seizure onset show a decrease in tracer uptake in the right cerebral hemisphere (A). Late IMZ-SPECT images disappear 1 week after the onset of abnormal visual field symptoms (B).
revealed complete left HH (Fig. 4). Electroencephalogram (EEG) obtained during the period of abnormal visual field symptoms did not show abnormal waves suggestive of epilepsy. The hemianopsia improved gradually and resolved completely 3 days after readmission. The abnormal findings on FLAIR (Fig. 5A), DWI (Fig. 5B), ADC (Fig. 5C), ASL (Fig. 5D), and late IMZ-SPECT images (Fig. 3B) disappeared 1 week after the onset of abnormal visual field symptoms.

Although the EEG showed no characteristic epileptic findings, the patient was diagnosed with post-traumatic simple partial seizure on the basis of the clinical symptoms, ASL and IMZ-SPECT findings, and normal laboratory data at onset. Post-traumatic simple partial seizure is a subtype of non-convulsive status epilepticus. The patient follow-up has been uneventful.

**Discussion**

We demonstrated a rare case of pure paroxysmal HH following mild TBI. Only seven patients, including the present patient, are reported to have presented with pure HH as the initial symptom of epilepsy [3–6], although this symptom is relatively common as an epileptic aura [7]. However, the prevalence of HH may have been underestimated because EEG might not be the most appropriate modality for detecting epileptic foci derived from a small area of cortex [8]. Of these seven cases, a definitive diagnosis of epilepsy was made by EEG in all cases except the present case. Furthermore, the time to diagnosis was longer in these previous cases (range, 2-14 days). A secondary generalized seizure was observed all but the present patient (85.7%), suggesting the delayed diagnosis of this entity with EEG and the importance of early diagnosis and treatment. Therefore, although EEG is considered the gold standard for the diagnosis of epilepsy, MR-ASL may be able to diagnose epilepsy earlier and also avoid an unfavorable course in selected patients with epilepsy.

It can be difficult to differentiate simple focal epilepsy from ischemic stroke in the emergency department because of their similar presentations and the limited utility of emergent EEG. However, as delayed treatment potential predisposes toward poor outcome, it is critical to obtain a prompt and accurate diagnosis [1]. ASL has been reported as a simple alternative to EEG that is suitable for use in emergency departments because it can differentiate seizure-associated symptoms from stroke.
by detecting an increase in CBF in affected regions during the ictal period without the use of contrast or radioactive tracer [9,10] (Table 1).

Conclusion

We presented a rare case of paroxysmal HH following mild TBI. In patients with simple partial epilepsy, MR-ASL imaging can be used as a diagnostic tool to be able to diagnosis epilepsy earlier.

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Table 1 – The summary of the patients presenting with only homonymous hemianopia as an initial symptom of epilepsy.

| References     | Age(yr)/Sex | Etiology                  | Epileptic focus | Secondary generalization | Diagnosis method | Time to diagnosis |
|----------------|-------------|---------------------------|-----------------|--------------------------|------------------|------------------|
| Spatt et al., 2000 | 83/M        | stroke                    | Mesial occipital | yes                      | EEG              | 2 days           |
| Shaw S et al., 2012 | 65/M        | osteomyelitis             | Mesial occipital | yes                      | EEG              | 2 weeks          |
| Shaw S et al., 2012 | 36/M        | hippocamal sclerosis     | Lateral occipital | yes                      | EEG              | 5 days           |
| Shaw S et al., 2012 | 35/M        | encephalitis              | Lateral occipital | yes                      | Video EEG        | NA               |
| Sawaya et al., 2014 | 53/M        | idiopathic                | Parieto-occipital| yes                      | EEG              | 10 days          |
| Siatouni et al., 2016 | 30/F        | NA                        | Lateral occipital | yes                      | EEG              | unknown          |
| Present case      | 82/F        | trauma                    | Parieto-occipital| no                       | MR-ASL           | 4 hour           |

EEG, electroencephalography, NA; not available.