

Remote effect of optic tract trauma in the occipital lobe

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ABSTRACT

Traumatic brain damage can result in severe visual impairments including hemianopia. Lesions correlated with hemianopia can be located in any part of the retrochiasmatic optic pathway. However, traumatic lesions of the optic tract are relatively rare. We present a case with posttraumatic left homonymous hemianopia who had signal intensity change at the ipsilateral optic tract on MR imaging and ipsilateral occipital hypoperfusion on SPECT imaging.

Key words: • visual pathways • brain injuries
• magnetic resonance imaging

Traumatic brain injury can result in severe visual problems including hemianopia. The lesions causing hemianopia can be located at any portion of the retrochiasmatic optic tract. However, traumatic optic tract lesions are rare. In this presentation featuring a patient who has developed homonymous hemianopia, with lesions in the optic tract and a normal ipsilateral occipital lobe on MR imaging, we aim to discuss the possible causes of ipsilateral occipital hypoperfusion that was observed in single photon emission computed tomography (SPECT) examination.

Case report

A 22-year-old female patient had a previous head trauma from the frontal region six months previously. The complaints of the patient within 20 days following the trauma were amnesia, confusion, and bilateral loss of vision. The patient was immediately transferred to a trauma center and the initial computed tomography (CT) examination was normal except for a minimal right frontal subdural hematoma. A follow-up CT examination was completely normal. The ophthalmologic examination that was performed in the first week revealed bilateral homonymous hemianopia, being more prominent on the left. The neurological examination that was performed on day 21 was normal except for mild confusion. The confusion resolved completely in time. In the electroencephalography performed in the first month, diffuse slow waves were observed and these progressively improved in the follow-up examinations with final normalization. The patient was discharged after two months of hospitalization and was back at work.

Because of ongoing complaints, however, she was referred to our hospital for further examination. MR imaging that was obtained on the fourth month revealed a mild signal increase in the right optic tract on T2-weighted and fluid attenuated inversion recovery (FLAIR) sequences (Figure 1). Right optic radiation and the occipital lobe were normal (Figure 2). Three-dimensional (3D) time-of-flight (TOF) magnetic resonance angiography (MRA) examination directed at intracranial arteries was normal (Figure 3). Two days after MR imaging examination, SPECT was performed with a double-head gamma camera after the administration of 20 mCi ^{99m}Tc-hexamethylpropylene amine oxime (HMPAO) and demonstrated the hypoperfusion of the right occipital lobe. Symmetrical relevance areas were marked on both occipital lobes and lesion-contralateral radioactivity ratio (L/K ratio) was calculated. Mean L/K ratio of the occipital lobe was 0.7 (Figure 4). Two weeks of a corticosteroid therapy regimen was initiated. There was significant improvement in the perimetric analysis that was repeated after the treatment.

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Received 25 April 2003; revision requested 31 October 2003; revision received 21 November 2003; accepted 05 December 2003.

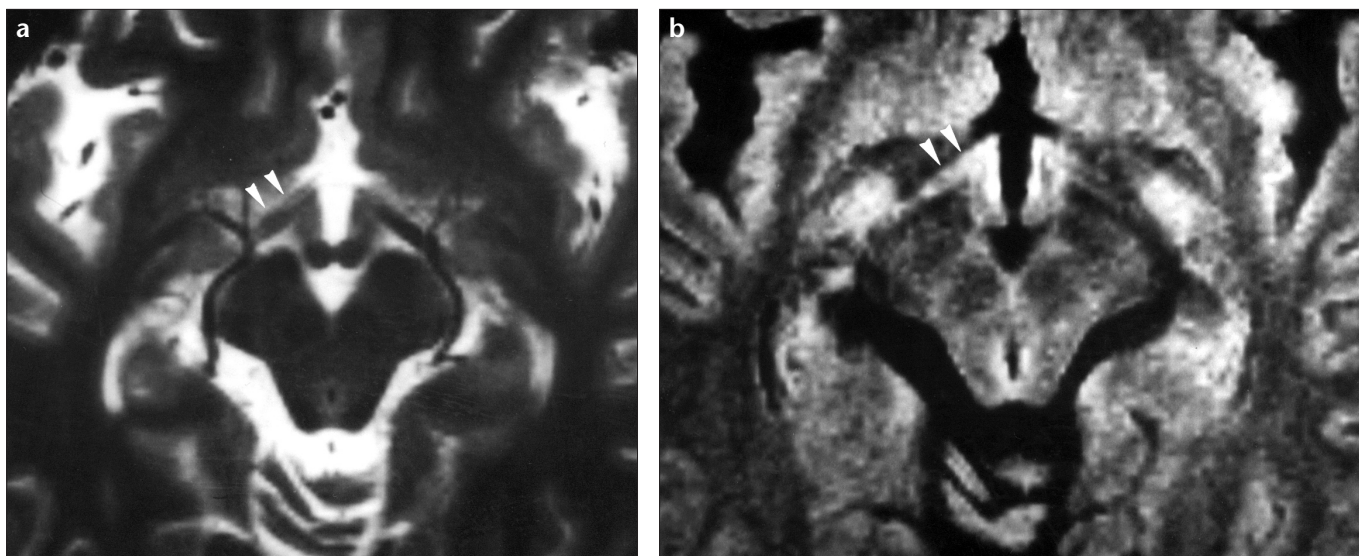


Figure 1. a, b. Axial T2-weighted (a) and FLAIR (b) MR images reveal a mild hyperintensity on the right optic tract (arrows). The lesion is more conspicuously observed on the FLAIR image with contrast.

Discussion

Forty percent of homonymous hemianopia cases involve the occipital lobe, 30% the parietal lobe, 25% the temporal lobe, and 5% the optic tractus and the lateral geniculate nuclei (LGN). Visual field defects originate from canicular optic nerve injury in 62% of the cases following closed head traumas, regardless of the presence of optic canal fractures (1, 2). Anterior marginal tears, retrobulbar, and chiasmatic damage are observed to a lesser degree. Optic tract injuries are very rare in closed head traumas (1).

Visual field examination reveals important information as to the localization of the damage, yet wrong localization is also a possibility. Furthermore, visual field examination does not provide information about the nature and the extent of the primary pathology. For patients in whom a lesion in any part of the optic tract is suspected, neuroradiological examinations are warranted. In closed head traumas, CT without contrast is the first choice for radiological examination. Emergency CT examination in our patient was normal, other than a mild subdural hematoma. MR imaging examination, however, demonstrated that the lesion responsible from hemianopia was located on the right optic tract. In SPECT examination, however, the right occipital lobe that was observed to be normal on MR imaging demonstrated mild hypoper-

fusion. Discrepancies, though limited in number, have been reported in the literature between the results of examinations like MR imaging and CT when compared with the results of functional examination techniques such as SPECT and PET. Silverman et al. have compared SPECT and MR imaging examination results in seven patients having visual loss due to different etiologies (cerebral ischemia, carbon monoxide intoxication, status epilepticus, and Alzheimer disease) (3). In all of these patients, MR imaging examination was either normal or revealed lesions that were smaller than those demonstrated by SPECT. Bosley et al. have demonstrated abnormal PET findings in a patient having migraine-like sight disturbances while he had normal CT results (4). MR imaging is not generally sensitive to functional changes and, in certain visual field defects, it cannot fully delineate the related pathological changes. SPECT, on the other hand, is sensitive to functional changes for which anatomical imaging techniques have low sensitivity.

To the best of our knowledge, ours is the first case presentation reporting occipital hypoperfusion secondary to optic tract damage. There are limited numbers of functional imaging studies in the literature performed on patients with visual disturbances. Celesia et al. have demonstrated decreases in regional blood flow in bi-hemispheric posterior parieto-occi-

tal fields in patients who have developed cortical blindness secondary to ipsilateral cerebrovascular events (5). PET examination demonstrated hypometabolism secondary to ischaemic lesions in the optic tracts of patients who have homonymous hemianopsia or quadrantanopia (4). Bosley et al. have shown increases in the cerebral metabolic rate of the occipital cortex harboring the infarct, whereas there were moderate decreases in this rate in the occipital cortex of patients having optic radiation infarcts. Similar to the findings of Bosley et al., the optic tract rather than the optic radiation was affected in our case and regional flow rate of the right occipital cortex was 30% lower compared to that of the contralateral cortex. We evaluated this finding as a remote effect.

Kiyosawa et al. claimed that if the cerebral metabolism of the affected cortical fields was measured as being less than 65% of the healthy contralateral side, this should be evaluated as the complete infarct of the ipsilateral visual cortex (6). They reported that remote effects should be considered in case of having measurements more than 65%. Remote effect due to neuronal degeneration (antegrade or retrograde) is a well known phenomenon (7, 8). We think that the minimal increase in the perfusion of the right occipital cortex of our case was secondary to degeneration. There are three possible explanations to support this idea: first, the absence of

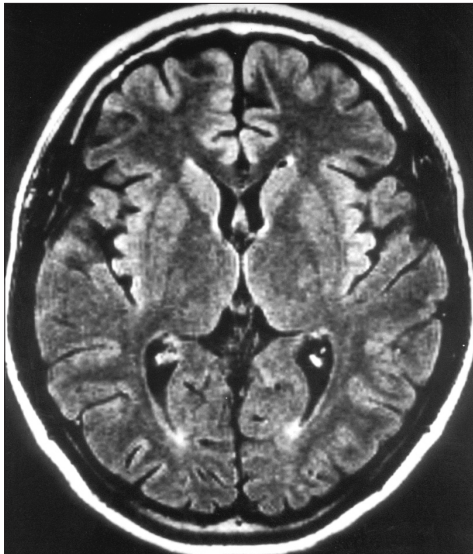


Figure 2. Optic tracts and occipital cortex are observed as normal on the FLAIR MR sequence.

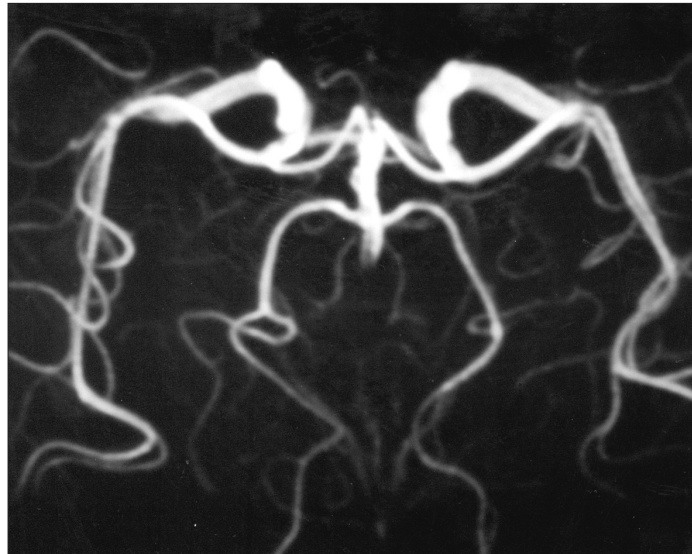


Figure 3. On 3D-TOF MRA, flow phenomenon is normal on the right posterior cerebral artery.

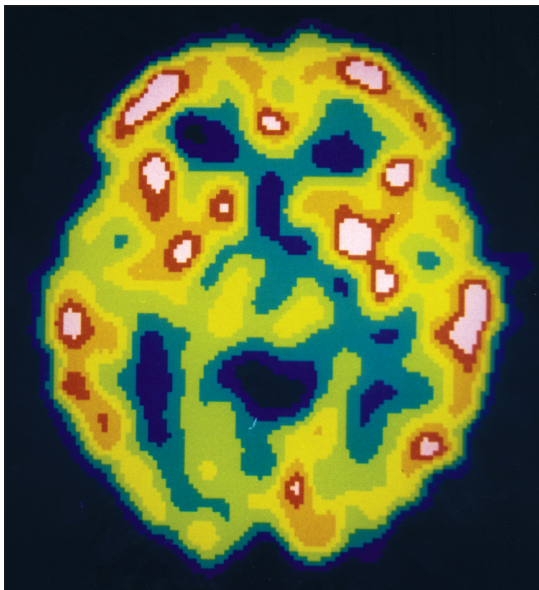


Figure 4. Mild hypoperfusion is observed on the right occipital cortex on axial SPECT examination.

morphological changes in the occipital cortex on MR imaging; second, having no findings on MR imaging that would support vascular pathologies in the areas fed by the posterior cerebral artery; third, the presence of a mild decrease in the regional blood flow rather than a moderate one that would support a primary local pathology.

In two patients having chronic neurological problems, Savoiardo et al. have demonstrated Wallerian degeneration of the optic radiation (9). In these two patients, the only pathology in relation to the Wallerian degeneration were the lesions of the ipsilateral geniculate body. The occip-

ital lobes of these patients had normal signals on MR imaging. Although the presence of T2 hyperintensity in fields far away from the primary lesion was a commonly encountered situation in cases of Wallerian degeneration, such a change was not identified in our case. Orita et al. have presented a possible explanation for this situation (10). These authors mentioned four stages of Wallerian degeneration of the pyramidal tracts based on T2-weighted sequences (10). In stage 1 (the first 0.7 month), there are no signal changes. In stage II (0.7-2 months), low signal intensity is anticipated. This is due to the increase

in the lipid/water ratio as triggered by the lipids that were released from the degraded myelin sheaths of the degenerated axons of the pyramidal tracts. In stage III (2.1-3.7 months), there are no signal changes. In stage IV (after 3.8 months), high signal intensities are observed. Our patient might have been examined during stage III. Furthermore, as has been demonstrated by the improvement in hemianopia following corticosteroid therapy, the Wallerian degeneration of the optic tract might not have involved all the nerve fibers and the resulting partial volume effect might not have been sufficient enough to be observed as signal increase in the voxels. Another explanation for the mild decrease of perfusion in the occipital cortex might be the decrease in the impulses arriving at the cortex from the optic tracts (transsynaptic inhibition). The best known example of transsynaptic inhibition is cross-cerebellar or trans-cerebral diachisis (11, 12).

This study shows that there can be changes in the cortical perfusion secondary to optic tract injuries. The absence of accompanying pathologies in MR imaging directs us to a functional change rather than a morphological one. In conclusion, SPECT examination can reveal the flow changes in the occipital cortex following a lesion affecting the optic tracts and, when evaluated together with MR imaging, can help in demonstrating remote effect phenomenon.

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