

Bilateral Cortical Blindness Caused by Tentorial Herniation due to Brain Tumor

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Two patients, one with glioblastoma multiforme (GM) in the right thalamus and the other with meningioma at the right frontal convexity, had suffered bilateral cortical blindness after transtentorial herniation. On one of those patients, bilateral cortical blindness had occurred due to acute obstructive hydrocephalus caused by GM and on the other patient, cortical blindness had developed after acute hemorrhage from meningioma. Bilateral occipital lobes of those patients showed signal change on the brain magnetic resonance image (MRI). There were no ophthalmologic abnormalities on funduscopy and ophthalmologic examination. After recovery of consciousness, cortical blindness was detected in both patients, and during gradual recovery period, visual function was slowly recovered. The pattern of visual evoked potential (VEP) at 7 weeks and 12 weeks after herniation was normalized gradually. Cortical blindness due to herniation was reversible, even though the high signals of bilateral visual cortex still existed on MRI 16 months later in case 2.

KEY WORDS : Bilateral · Cortical blindness · Brain herniation · Brain tumor.

Introduction

Cortical blindness was the term initially coined by Marquis in 1933 to describe patients with visual loss but normal pupillary reactions and a normal outcome on ophthalmologic examination^{2,5,18,20,22}. It has been occurred after bilateral lesions of the calcarine cortex^{5,22}. There may

be bilateral homonymous hemianopsia due to optic tract, radiation or occipital lobe involvement and bilateral central scotomas due to bilateral occipital lobe lesions^{2,5,22}. The most common cause of cortical blindness is cerebral vascular disease⁵. The suggested causes are emboli, profound hypotension, anemia, and infarction of watershed areas in the parietal or occipital lobe^{2,5}. Unilateral homonymous hemianopsia due to transtentorial herniation sometimes occur but, bilateral homonymous hemianopsia is rare. We experienced two cases of bilateral cortical blindness with severely reduced visual acuity after transtentorial herniation by brain tumors.

Case Report

Case 1

The 54-year-old woman was presented with headache, nausea and vomiting. The subjective hemiparesis on left side started 5 days before admission. The brain MRI study revealed a round mass at the right thalamus causing compression of the third ventricle and dilatation of the right lateral ventricle. The mass was slightly enhanced on gadolinium-enhanced MRI (Fig. 1). She became stuporous

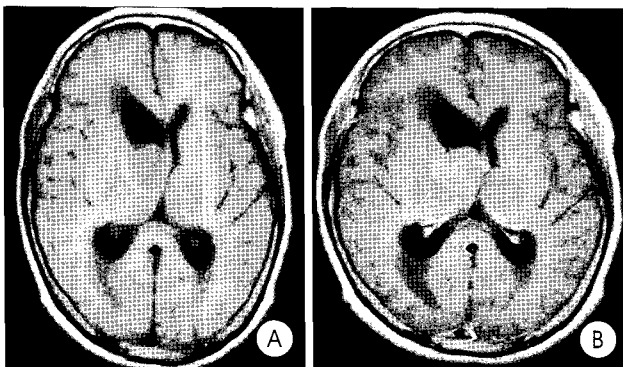


Fig. 1. A, B : Preoperative pre- and gadolinium-enhanced magnetic resonance image (MRI). A : Pre-enhanced MRI showing a 2.5 x 3 x 2 cm sized round mass lying within the right thalamus near the foramen of Monro causing compression of the third ventricle and enlargement of the right lateral ventricle. B : Gadolinium-enhanced MRI showing that the mass is faintly enhanced.

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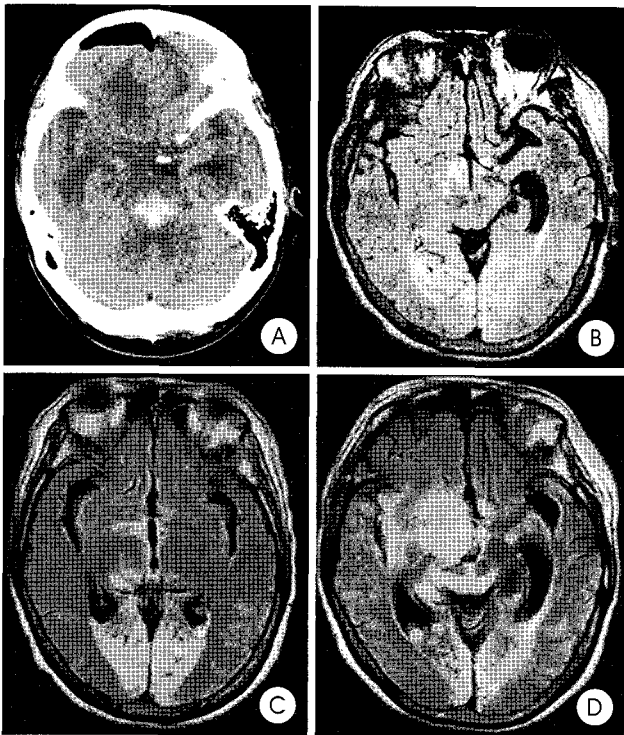


Fig. 2. A : On 3rd hospital day, brain CT shows pontine hemorrhage. B : On 8th hospital day, preoperative axial FLAIR MRI showing high signal change in the bilateral occipital lobes. C : On 24th postoperative day, FLAIR MRI still shows high signal area. D : Bilateral occipital lesions are decreased on the postoperative 5th month.

by obstructive hydrocephalus on next day and computed tomography (CT) revealed further enlargement of ventricle size and increase of edema. After the emergency extraventricular drainage (EVD) her consciousness was recovered within 24 hours. However, the patient's consciousness suddenly deteriorated to comatose state again and the brain CT showed the pontine hemorrhage (Fig. 2A). On neurologic examination, she had severe quadriplegia and limitation of extraocular eyeball movements. Five days later, brain MRI study showed high signals in occipital lobes bilaterally on FLAIR sequence (Fig. 2B). She could understand what her family wanted to say but still existed quadriplegia was persisted with like locked-in-syndrome type appearance. The thalamic tumor was removed via contralateral transcallosal approach on 9th hospital day. Afterwards, the visual acuity was about perception to light with anisocoria in all quadrants postoperatively. There were no ophthalmologic abnormalities on funduscopy and ophthalmologic examination. We performed visual evoked potential (VEP) to assess cortical blindness at three-month intervals. Abnormal findings on the VEP simultaneously were improved and visual acuity was improved as to finger counting (Fig. 3A, B). Brain MRI still showed high signal lesions in the bilateral occipital lobes on FLAIR images but the extent of high signal lesions was decreased (Fig. 2C, 2D).

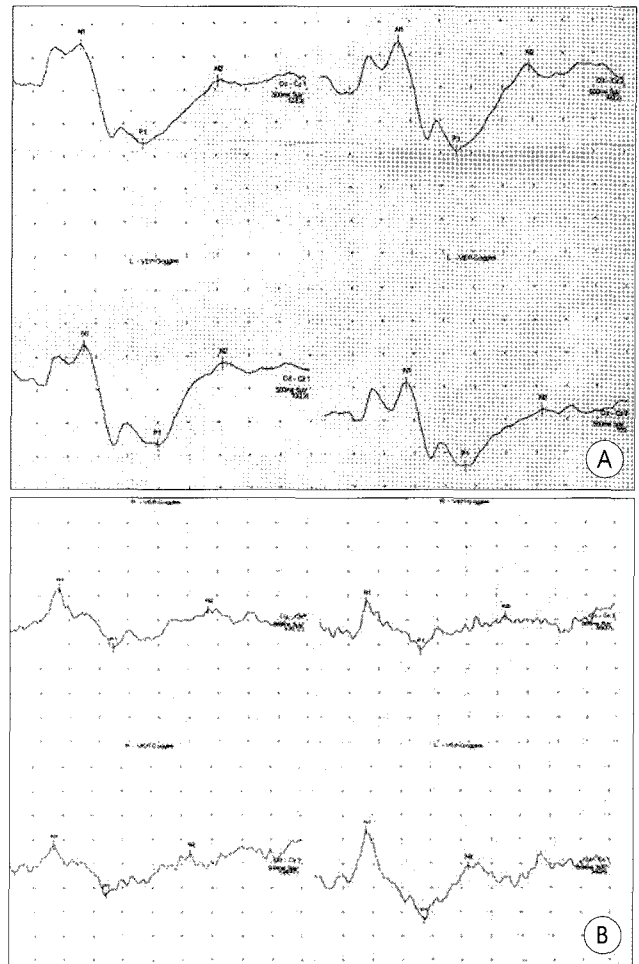


Fig. 3. A, B : Visual evoked potentials (VEP) obtained on 50th postoperative day and performed 3 month after 1st VEP. The VEP pattern shows an improvement and the latency of each peak is normalized 3 month later.

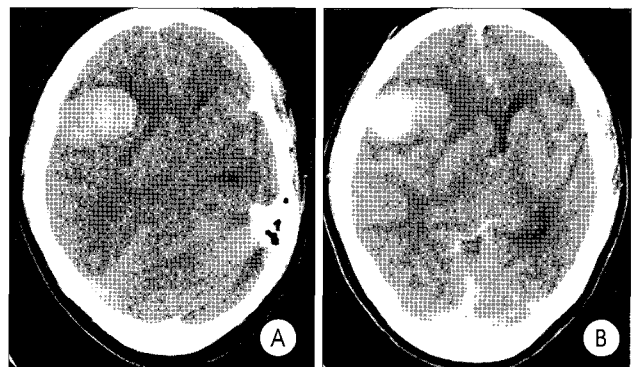


Fig. 4. A, B : Preoperative pre-enhanced and enhanced CT. A : Pre-enhanced CT showing a 2.5 x 3 x 2 cm sized high density round mass lying on the right Sylvian fissure and midline shifting to the left due to severe cerebral swelling. B : Enhanced CT shows strong enhancement.

Case 2

The 48-year-old woman complained of severe headache with a 2.5 x 3 x 3 cm sized enhanced high density mass that suggested hemorrhage inside tumor around the right Sylvian fissure on the brain CT. She was progressively deteriorated

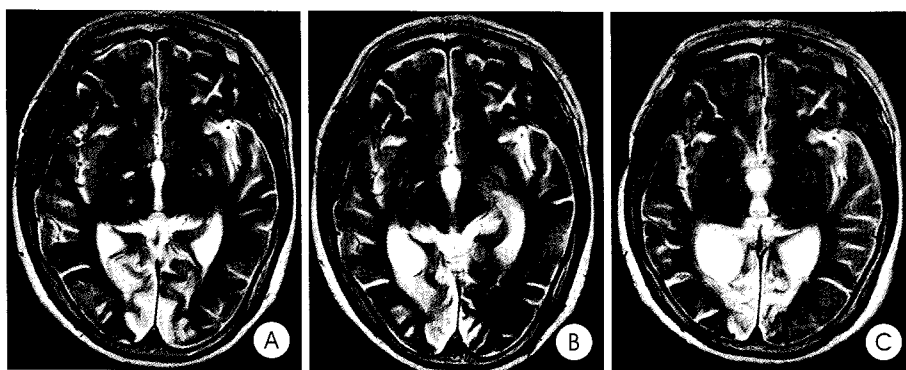


Fig. 5. A : On 8th hospital day, preoperative axial T2-weighted MR image shows high signal change in the bilateral occipital lobes. B : On 36th postoperative day, T2-weighted MR image still shows high signal area. C : Bilateral occipital lesions are decreased on the postoperative 16th month.

as stuporous mentality and developed left hemiparesis after admission (Fig. 4A, B). The surgery was done on next day. The tumor revealed as atypical meningioma, which was composed of meningothelial cells forming whorls with nuclear pleomorphism, increased cellularity and mitosis. On 24th postoperative day, her consciousness was recovered as nearly alert but, she still suffered from visual disturbance. She complained glimmer visual accuracy. Examination of visual fields with Goldmann perimeter revealed normal findings. Eye movements in all directions were normal with the pupils reactive to light and fundoscopic examination was also normal. However, around one month after surgery, her visual function completely recovered without glimmer visual accuracy. Axial T2-weighted MR image on the postoperative 36th day and 16th month showed still high signals in both occipital lobes (Fig. 5).

Discussion

The causes of cortical blindness are cerebrovascular disease, head trauma, cerebral swelling, seizure, infection, drug, cardiac surgery and angiography (cerebral angiography and coronary arteriography)^{2,4,5,8,9,11,18,20,22}. In 1920, Meyer¹³ first reported occipital lobe infarction caused by tentorial herniation and recognized that compression of branches of posterior cerebral artery (PCA) was the cause of these occipital lobe infarctions that were confined to the occipital cortex. Moore and Stern¹⁴ in 1938, Maltby¹² in 1942, Pevehouse et al.¹⁶ in 1960, and Hoyt⁷ in 1960 reported cases of transtentorial herniation with a homonymous hemianopsia. Sato et al.¹⁹ in 1986 reported that the incidence of occipital lobe infarction caused by tentorial herniation was 9% of their patients with tentorial herniation. They mentioned that PCA or its branches were compressed at the free margin of the tentorial aperture. However, some parts of occipital lobes that were spared ischemic insult corresponded to the area supplied by the

individual branches of PCA : the calcarine artery, posterotemporal artery, and collateral flow from PCA and middle cerebral artery, respectively. These findings support the idea that occipital lobe infarction in tentorial herniation is of arterial origin, as suggested by Sunderland²¹, Hoyt⁷, and Lindenberg et al.¹⁰. They reported that the infarcted area caused by compression of the contralateral artery was seen in three cases; there were two occipital lobes and an area around

Ammon's horn. The area around Ammon's horn was presumably involved by the occlusion of the hippocampal branches arising from PCA. Others believe that infarction is of venous origin with compression of the calcarine vein as it runs toward the great vein of Galen¹⁷. Sunderland²¹ and Lindenberg et al.¹⁰ have noted that the basal vein is compressed in the groove between the midbrain and herniated temporal lobe but Sato et al.¹⁹ described that venous congestion is probably compensated by collateral circulation. Clinical investigation using MRI demonstrated variability in clinical condition and white-gray matter difference in the territory of the occipital lobe, although infarction was confined to the gray matter by Meyer's first report¹³. The variability has been thought to be caused by the anatomical variation around the midbrain, mainly in the shape and size of the tentorial aperture, were well described by Sunderland²¹. The point where the artery is compressed against the free edge of the tentorium will vary from case to case. It is speculated that the width of tentorial aperture and variation of the PCA, rather than the anterior-posterior diameter of the aperture, play important roles; a narrow aperture between the free margins of the tentorium is apt to compress the PCA in the more proximal portion, resulting in a wide area of infarction over the occipital lobe. The patients without infarction of the medial part or inferolateral part of the occipital lobe may have a wider aperture, resulting in compression of PCA at the more distal portion, although only the posteromedial part also may be influenced by some collaterals from the middle cerebral artery¹⁹. Diagnostic methods for cortical blindness are neuroimaging (e.g. Brain CT, MRI) electroencephalogram (EEG), VEP, single photon emission computed tomography (SPECT), and functional MRI²⁵. Moseman et al.¹⁵ reported that MRI revealed within the occipital lobe areas of symmetric bilateral decreased signal intensity on T1-weighted imaging and high-signal-intensity areas on fluid-attenuated inversion recovery (FLAIR) images and T2-weighted images. During the period of cortical

blindness EEG is abnormal with absent alpha rhythm but reactive alpha rhythm is a good prognostic finding in cortical blindness⁵⁾. VEP recorded during blindness was abnormal but did not correlate with the severity of visual loss or with outcome like our case¹⁵⁾. Makino et al.¹¹⁾ supposed that the positive peak at the latency of 116.5 m sec (P 100) reflects the activation of the occipital lobe and P 200 corresponds to the visual perception. SPECT is used to investigate changes in cerebral blood flow. The functional MRI demonstrates areas of the brain that are metabolically active⁵⁾.

Aldrich et al.¹⁾ reported that bi-occipital abnormalities shown on CT scan in cortical blindness are associated with a poor prognosis. Groenendaal et al.⁶⁾ described that most patients with cortical blindness do not regain normal vision. However, there are rare cases of visual improvement. Good³⁾ reported that visual improvement may be sudden or gradual.

Bergman³⁾ reported a pattern of recovery of visual functions in cortical blindness, which he defined as blindness caused by injury of the optic pathway behind the lateral geniculate body. At first, light sense around the macular area was observed, and subsequently recognition of colors before recognition of geometric shape. Fortunately, although there were radiologically still remained abnormal high signal, the visual function of our patients was recovered but the test of color discrimination and geometric shape should be considered in these patients in order to recognize the course of prognosis because patterns of recovery may be versatile.

Conclusion

Cortical blindness caused by transtentorial herniation could be reversible by early decompressive surgery, even though radiological abnormalities persist.

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