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Sir,

Relapsing reversible posterior leukoencephalopathy syndrome

Reversible posterior leukoencephalopathy syndrome (RPLS) is characterized by headache, altered mental functioning, seizures, and visual loss, and has been recognized in a variety of conditions, including hypertension, eclampsia, treated with immunosuppressive and cytotoxic drugs, etc.^{1,2} We report an RPLS patient with relapsing course and abnormal electroencephalogram (EEG).

Case Report

This 9-year-old girl was diagnosed with acute lymphoblastic leukemia (ALL) and admitted for allogenous bone marrow transplantation. On admission, her blood pressure was 127/85 mmHg. On the 15th day of induction therapy (including methotrexate, prednisolone, cytosine, L-asparaginase, and vincristine), acute hypertension (156/98 mmHg) was noted. On the following day, she suffered from sudden onset of severe visual loss and gait imbalance.

Ophthalmological examination revealed a visual acuity of counting fingers in each eye. Both pupils reacted to light briskly. Slit lamp examination and ophthalmoscopy examination were unremarkable. Acute cerebral blindness was impressed. Brain magnetic resonance imaging (MRI) demonstrated hyperintensities in bilateral occipital lobes and left frontal lobe on T2-weight images, diffusion-weighted images (DWI) and apparent diffusion coefficient (ADC) maps (Figure 1). Under the impression of RPLS, all drugs except prednisolone were discontinued. Her blood pressure was normalized by administration of nifedipine 20 mg per day. The vision improved to 20/40 in both eyes within 3 days. However, on the 23rd day of induction, the visual acuity deteriorated to hand motion in both eyes, and elevation of blood pressure to 126/92 mmHg was also noted. Repeated MRI T2-weighted images still showed



Figure 1 Brain MRI on first episode of vision loss demonstrated gyriform hyperintensities in bilateral occipital lobes and left frontal lobes on T2-weight images (top), diffusion-weighted images (middle, b = 1000) and apparent diffusion coefficient maps (bottom). These findings confirmed the diagnosis of reversible posterior leukoencephalopathy syndrome.



Figure 2 T2-weight image on relapse of vision loss still showed hyperintensities in bilateral occipital lobes, only to a lesser degree.

hyperintensities in bilateral occipital lobes, only to a lesser degree (Figure 2). An EEG showed abnormal sharp wave complex arising from both occipital regions. The vision recovered with more strictly controlled blood pressure by increasing nifedipine to 50 mg per day. She received allogenous bone marrow transplantation 2 weeks later with smooth course. At 2 months after the onset of RPLS, the follow-up visual acuity was 20/20 in each eye. Follow-up MRI showed complete resolution. The patient is now in complete remission state without any neurologic sequelae.

Comments

Reversible posterior leukoencephalopathy syndrome (RPLS) is a clinical and radiographic syndrome of importance to ophthalmologists because it may present with acute bilateral visual loss of cortical origin. With correct management, as the name implies, the visual loss is reversible and if the cause is not recognized the visual loss can be permanent. In the evaluation of cerebral blindness, DWI, and ADC maps are invaluable in differentiating vasogenic oedema of RPLS from cytotoxic oedema due to an ischaemic stroke.³ Prompt diagnosis avoids potentially dangerous invasive procedures such as thrombolytic therapy.

RPLS have been reported in ALL patients treated with L-asparaginase or high-dose methotrexate therapy.^{4,5} Our patient developed RPLS on induction chemotherapy, when elevation of blood pressure was also noted. All drugs except prednisolone were discontinued, and blood pressure was normalized with nifedipine, and vision

improved. However, the symptoms relapsed during discontinuation of these suspected drugs. Meanwhile, elevation of blood pressure was noted. It was, therefore, deduced that acute hypertension rather than L-asparaginase or methotrexate was the culprit of RPLS in this case. Besides, relapse in RPLS has rarely been reported. Our patient suffered from relapse, which corresponded with elevation of blood pressure. Though the blood pressure only increased to 126/92 mmHg on relapse, it might be still high for a posterior cerebral circulation that had already given up autoregulation.

Previous reports showed that patients with RPLS exhibited no pathogenomic EEG findings or only slowing in the parietal and occipital areas.⁶ In contrast, the EEG in our patient showed abnormal sharp wave complex arising from both occipital regions, in terms of partial seizure with epileptiform discharge, though no frank seizure was noted.

In summary, acute hypertension rather than L-asparaginase or methotraxate was considered the culprit of RPLS in our patient. The course fluctuated with unstable blood pressure. The EEG showed abnormal sharp wave complex arising from occipital regions.

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Sir,

Refractive lens exchange combined with pars plana vitrectomy to correct high myopia

Thank you for the interesting discussion concerning our pilot study.

As described none of the patients had retinal problems postoperatively. Vitreous body status was not taken into account as inclusion criteria. However, we can report that nine eyes had a pre-existing posterior vitreous detachment (PVD) preoperatively and five eyes needed a PVD induced during vitrectomy. Actually, inducing a PVD during surgery is a risk factor for retinal tear formation.¹ However, only undetected or improperly managed retinal breaks lead to postoperative retinal detachment. The assumption is that the surgeon is experienced in surgery of the anterior and posterior segment of the eye.

We are familiar with the study of Suzuki *et al*² and Bilinska *et al.*³ The spread between predicted and actual refractions was -0.05 ± 1.18 D in the combined surgery group and $+0.05 \pm 1.32$ D in the cataract surgery group. The actual refractive errors in the combined surgery group were found to shift toward myopia when compared with the controls.² However, the actual refractive errors in the combined surgery group showed nearly no spread between predicted and actual refraction $(-0.05 \pm 1.18 \text{ D})$. In contrast, the cataract surgery group showed a hyperopic shift.

We measured a mean postoperative refraction of -0.7 ± 1.6 D. As slight myopia was targeted as postoperative result (-0.5 to -1.0 D; using the IOLPC-5-formula by Haigis).⁴ Thus, we found no myopic shift between predicted and actual refractions for our group with combined surgery. We had no group with patients undergoing RLE without PPV for comparison of the shift between combined surgery and cataract surgery alone.

None of the eyes in our study needed a gas tamponade following vitrectomy. We therefore did not expect to find a myopic shift due to gas tamponade, which can press the intraocular lens forward.

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Sir,

Refractive lens exchange combined with pars plana vitrectomy to correct high myopia

We read with interest the article by Uhlmann and Wiedemann,¹ and congratulate the authors on their