References

- 1 Potter MJ, Berson EL. Diagnosis and treatment of gyrate atrophy. Int Ophthalmol Clin 1993; 33(2): 229–236.
- 2 Marano F, Deutman AF, Pinckers AJ, Aandekerk AL. Gyrate atrophy and choroidal neovascularization. *Arch Ophthalmol* 1996; **114**(10): 1295.
- 3 Chen CJ, Furr P. Bilateral keratoconus in a patient with gyrate atrophy and hyperornithinemia. *Am J Ophthalmol* 1983; **95**(5): 705–706.
- 4 Feldman RB, Mayo SS, Robertson DM, Jones JD, Rostvold JA. Epiretinal membranes and cystoid macular edema in gyrate atrophy of the choroid and retina. *Retina* 1989; 9(2): 139–142.
- 5 Judson PH, Yannuzzi LA, Macular Hole, Ryan SJ (eds). *Retina*. Mosby, Inc.: Singapore, 2001, pp 1196–1197.
- 6 Banker AS, Freeman WR, Azen SP, Lai MY, The Vitrectomy for Macular Hole Study Group. A multicentred clinical study of serum as adjuvant therapy for surgical treatment for macular holes. *Arch Ophthalmol* 1999; **117**: 1499–1502.
- 7 Ezra E, Gregor ZJ, Moorfields Macular Hole Study Group Report No. 1. Surgery for idiopathic full-thickness macular hole. *Arch Ophthalmol* 2004; **122**: 224–236.

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Sir,

Acute bilateral simultaneous PION after ectopic pregnancy-related haemorrhage

Acute bilateral simultaneous posterior ischaemic optic neuropathy (PION) is a rare visual complication of ectopic pregnancy-related haemorrhage. Only one other patient with acute visual loss related to haemorrhage from an ectopic pregnancy has been described.¹

Case report

This healthy 37-year-old gravida 4 para 3 woman presented with acute bilateral simultaneous severe visual loss 1 day after her exploratory laparotomy, left salpingectomy, bilateral tubal ligation, and total abdominal hysterectomy for her ruptured cornual ectopic pregnancy. Prior to her surgery she had 20/20 vision. Intra-operative haematocrit was 19.3% (38-46% normal), intraoperative blood pressure readings ranged from 110/50 to 155/82 mmHg, and heart rate ranged from 58to 110 beats/min. She received a total of 6100 ml of crystalloid solution and her total estimated blood loss was 4000 ml. Postoperative haematocrit was 25.2% (38-46% normal) at 1 h, 18.2% (38-46% normal) at 6 h, and 15.6% (38-46% normal) at 12 h. Postoperative blood pressure readings ranged from 100/50 to 132/62 mmHg. On postoperative day one, she developed acute bilateral simultaneous visual loss associated with no headache or ocular pain. Her vision was NLP OU. Pupils were both 8 mm and nonreactive and round to light. Intraocular pressures were 17 mmHg OD and 18 mmHg OS. Extraocular motility was normal. Both optic discs appeared normal without any evidence of edema. Head CT scan was normal and MRI of the brain and orbits with gadolinium 3 days later did not reveal any infarcts or optic nerve pathology. CSF analysis was also normal.

After 1 month, her visual acuity improved to 20/200 OD and 20/40 OS. Humphrey central 24-2 visual field testing revealed that she had generalized depression OD and peripheral constriction OS. She had a right relative afferent pupillary defect and a red–green colour defect. Extraocular motility was normal. Diffuse disc pallor was greater in the right optic disc than in the left. P100 latencies were increased at 122 ms OD and 117 ms OS (97–115 ms normal); P100 amplitudes were decreased at 1.1 μ V OD and 1.7 μ V OS (3–15 μ V normal). The electroretinogram (ERG) was normal.

Comment

Visual loss is an uncommon but well-known complication of severe hypotension and anemia, most often occurring in the perioperative period.² Decreased blood supply to pial arteries derived from the ophthalmic artery leads to ischaemia of the posterior optic nerve.³ The pathogenesis of ischaemic optic neuropathy is unclear, but is believed to be related to optic nerve hypoxia or ischaemia in severe anaemia, with or without arterial hypotension.⁴ Hayreh⁴ noted that visual loss was not necessarily prevented in his patients who received immediate and adequate blood transfusion. He hypothesized that release of angiotensin, epinephrine, and vasopressin secondary to increased sympathetic activation could have caused vasoconstriction of the posterior optic nerve circulation resulting in posterior optic nerve ischaemia.

The diagnostic criteria for PION in this report are based upon those put forth by Buono *et al*⁵ which includes: (1) an acute decrease in visual acuity, visual field, or both; (2) an ipsilateral relative afferent pupillary

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defect, unless there is bilateral symmetrical involvement or a preexisting contralateral optic neuropathy when the pupils are sluggish or nonreactive to light. (3) documentation of a normal optic disc at the onset of visual deficit; (4) exclusion of other identifiable causes of visual deficit, including retinal and glaucomatous problems, and other causes of optic neuropathy, such as compression, demyelination, or inflammation with neuroimaging; (5) an abnormal VEP. (6) a normal ERG. (7) development of optic disc pallor within 4 – 8 weeks of onset of visual loss.

Unlike the patient described by Chun and Levin¹ who had nonarteritic anterior ischaemic optic neuropathies (NAION) after ectopic pregnancy-related haemorrhage, the patient in this report presented with acute bilateral PION. The optic discs were initially normal without any oedema and then became diffusely pale about 1 month later. Her normal MRI of the brain, normal ERG, and abnormal VEP helped confirm that her visual loss was a result of optic nerve lesions. The patient described by Chun and Levin¹ experienced minimal visual recovery, but the patient in this report improved to 20/200 OD and 20/40 OS with residual visual field defects after 4 weeks of onset.

This case report illustrates the unusual complication of bilateral PION associated with haemorrhage from a cornual ectopic pregnancy. This type of visual loss does not necessarily lead to legal blindness and occasionally may improve spontaneously. Acute bilateral PION should be included as a possible cause of visual loss in this clinical setting.

References

- Chun DM, Levin DK. Ischemic optic neuropathy after hemorrhage from a cornual ectopic gestation. *Am J Obstet Gynecol.* 1997; **177**(6): 1550–1552.
- 2 Sadda SR, Nee M, Miller NR, Biousse V, Newman NJ, Kouzis A. Clinical spectrum of posterior ischemic optic neuropathy. *Am J Ophthalmol* 2001; **132**: 743–750.
- 3 Isayama Y, Hiramatsu K, Asakura S, Takahashi T. Posterior ischemic optic neuropathy. I. Blood supply of the optic nerve. *Ophthalmologica* 1983; 186: 197–203.
- 4 Hayreh SS. Anterior ischemic optic neuropathy. VIII. Clinical features and pathogenesis of post-hemorrhagic amaurosis. *Ophthalmology* 1987; **94**(11): 1488–1502.
- 5 Buono LM, Foroozan R, Savino PJ, Danesh-Meyer HV, Stanescu D. Posterior ischemic optic neuropathy after hemodialysis. *Ophthalmology* 2003; **110**(6): 1216–1218.

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