

Bilateral Optic Neuritis in Pregnancy

M S Suraiya, MS(Ophthal), B Norazlina, MS(Ophthal), C Carmen, MS(Ophthal), M Muhaya, PhD

Department of Ophthalmology, Universiti Kebangsaan Malaysia, Jalan Yaacob Latif, Bandar Tun Razak, 56000 Cheras, Kuala Lumpur

Summary

A 25-year old primigravida at 11-weeks period of amenorrhoea presented with bilateral optic neuritis following Varicella Zoster viral (VZV) infection. She was serologically positive for systemic lupus erythematosus but negative for virus. The exact pathogenesis of the patient's severe optic neuritis, adduction and neurological deficit was unknown. The initiation of high dose steroids for optic neuritis was a big clinical dilemma in a pregnant patient with viral infection. The patient was treated with high dose steroids after three days of commencement of antiviral treatment. At 6 months after presentation, her visual acuity in the right eye was 6/36 with perception to light in the left.

Key Words: Optic neuritis, SLE, Varicella, Steroid, Pregnancy

Case report

A 25-year old primigravida at 11 weeks period of amenorrhoea, was referred to the Ophthalmology Clinic, Hospital Universiti Kebangsaan Malaysia with the complaint of painless loss of vision in both eyes, five days prior to presentation. This was preceded by a two week history of vesicular eruptions on the left side of the chest. She also experienced progressive weakness and numbness of both lower limbs but no bowel or urinary incontinence. She had chicken pox at the age of twelve.

Her visual acuity was counting fingers at 1 foot in the right eye and hand movement in the left eye. She had left exotropia and limitation of adduction in both eyes but no abduction nystagmus. Pupillary reactions were sluggish in both eyes with a relative afferent pupillary defect in the left eye. Anterior segment examination was normal. Ophthalmoscopic examination revealed bilateral hyperaemic and markedly swollen optic discs with peripapillary splinter haemorrhages and venous dilatation and tortuosity. (Figure 1) Both maculae and peripheral retinae were normal. The vitreous was clear bilaterally. Confrontation visual field test demonstrated

bilateral central scotoma, denser in the left eye. There were multiple dried zoster lesions in the left eighth thoracic dermatome. The muscle power of both lower limbs was reduced to grade 3-4/5. The lower limb reflexes were absent.

CT scan of the brain excluded any space-occupying lesion. She was diagnosed as having bilateral optic neuritis secondary to VZV infection with associated Gullain-Barre Syndrome in view of her neurological deficit of the lower limbs.

Investigations revealed a normal haemoglobin and total white count. The sedimentation rate was very high at 115mm/hour. Virologic studies for herpes were negative but she tested positive for anti-nuclear antibody and anti-double stranded DNA. Anticardiolipin antibody and lupus anticoagulant were negative. Visual evoked potentials were markedly reduced and delayed in both eyes. The patient however refused lumbar puncture.

The patient was started on intravenous Acyclovir 500mg 8 hourly for one week followed by oral Acyclovir 800mg

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Corresponding Author: Department of Ophthalmology, Universiti Kebangsaan Malaysia, Jalan Yaacob Latif, Bandar Tun Razak, 56000 Cheras, Kuala Lumpur

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5 times daily for 10 days. Intravenous Methylprednisolone 250mg 6 hourly was started 3 days after commencement of the antiviral treatment, followed by oral Prednisolone at 1mg/kg/day for 11 days and maintained at 20mg/day.

Her condition gradually improved with treatment. The right visual acuity improved to 6/60 within the next six weeks but the left acuity did not. It remained unchanged thereafter. However, the eye movements recovered within two months. The optic discs swelling resolved

leaving pale discs (Figure 2). Direct pupillary reactions were still sluggish with a residual left relative afferent pupillary defect. She regained full power in both lower limbs. Her pregnancy progressed well. She gave birth to a healthy baby girl at term via Caesarean section for foetal distress and flexed breech. When last seen, her best corrected visual acuity was 6/36 and N12 for near. She is still under follow-up with the Ophthalmology and Neurology team and maintained on oral Prednisolone 20mg daily for her SLE.

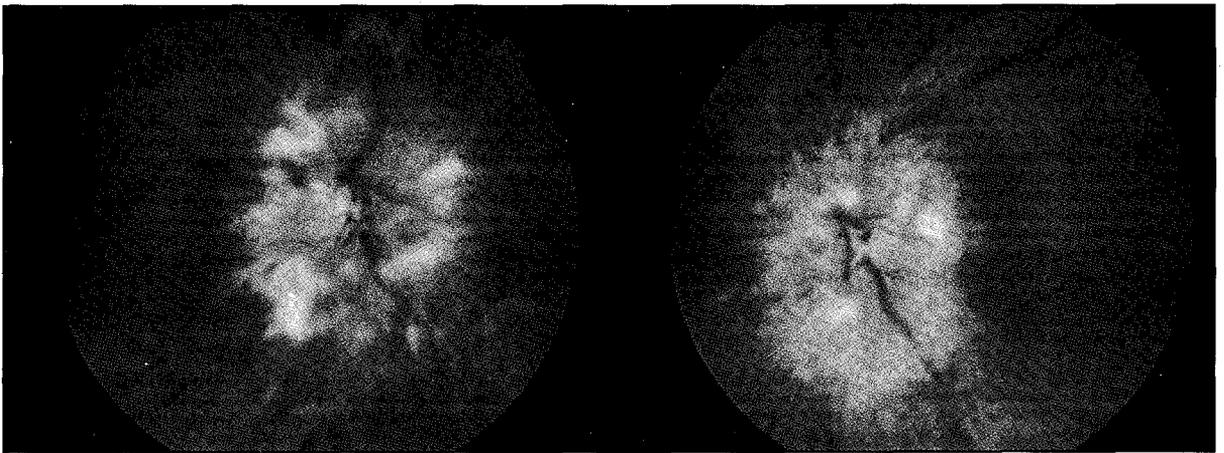


Fig 1: Fundus photos of the right and left eye at presentation showing markedly swollen optic disc with peripapillary splinter haemorrhages and dilated and tortuous veins.

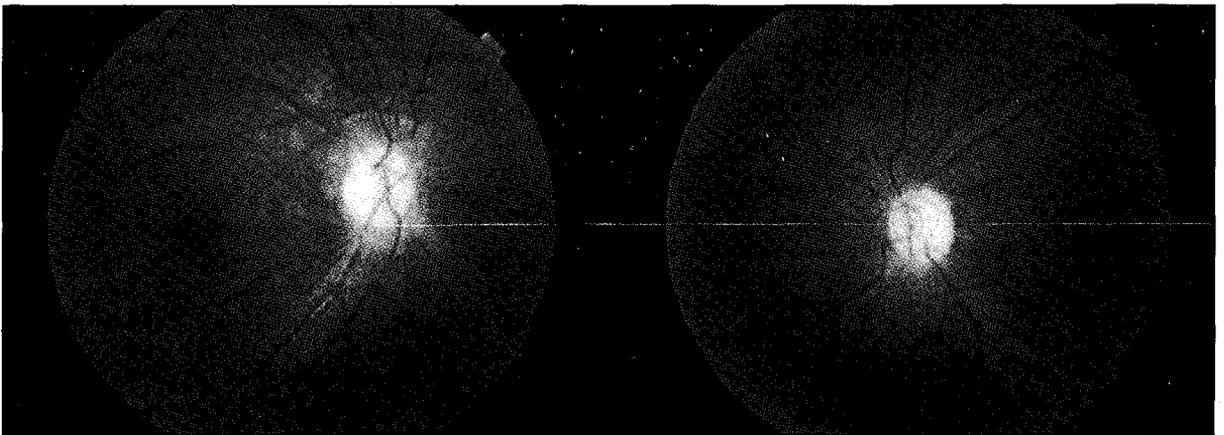


Fig 2: Fundus photos of the right and left eye taken 2 months later showing optic atrophy.

Discussion

This unique case illustrates the problem of making an exact diagnosis and the initiation of treatment in a pregnant lady with bilateral papillitis associated with VZV infection. She tested positive for SLE serologically, without any systemic features or criteria for this condition. There were no ocular manifestations of SLE present.

Optic neuritis is well documented but an unusual complication of VZV reactivation¹. It usually occurs weeks to months after the onset of skin lesions. Optic neuritis has been reported following Herpes zoster ophthalmicus, following dermatomal rash other than the ophthalmic division of trigeminal nerve and also in those without dermatomal rash.

Optic neuritis is a very rare complication of SLE, occurring in 1% of patients². Antiphospholipid antibodies which is responsible for venous and arterial thrombotic process leading to vaso-occlusive phenomenon is seen in 10% of SLE patients with optic neuritis. In these patients bilateral optic neuritis is more frequent than unilateral and is usually associated with poor visual outcome.

In both VZV infection and SLE, two pathogenesis of optic neuritis has been postulated either ischaemia as evidenced by features of ischaemic papillitis in fluorescein angiography or demyelinating optic neuropathy.

Adduction deficit may range from central involvement to local insult affecting the extraocular muscles. In this

patient, it may be explained by a local myositis of the muscle close to the inflamed optic nerve, or by the inflammation of the muscular branch supplying the muscle as it travels alongside the optic nerve, or demyelination of the midbrain although there are no other features of midbrain dysfunction. These may result in complete recovery of function and may occur in both VZV infection and SLE. The Guillan-Barre syndrome in this patient is caused by the VZV infection.

We concluded that our patient's SLE was triggered by her pregnancy which then lowers her immune response and thus reactivated the VZV infection. Apart from delayed initiation of therapy the poor visual outcome is also contributed by the ischaemic nature of her optic neuritis, which was reflected in the loss of amplitude in her VEP.

The treatment of VZV-related optic neuritis consists of intravenous Acyclovir 10-15mg/kg/day in 3 divided doses for 7 to 10 days. In immunocompromised patients the recommended dose is 15-30mg/kg/day for 10 days or longer with systemic steroids². The treatment given to this patient was adequate for her body weight.

It was a dilemma to initiate therapy for this patient. The nature of papillitis could either be immunogenic as part of SLE which would be controlled with steroids, or it could be infective secondary to VZV where steroids would predispose to disseminated VZV infection. Furthermore the possibility of adverse reactions of systemic steroid and antiviral agent to both mother and unborn fetus was high. However, in this patient there was no evidence of steroidal effect on the child.

References

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