Bilateral optic neuropathy after headache A

In adults bilateral optic neuropits is most commonly seen with demyelinating disease. An acute form exists in which both eyes are affected simultaneously. There exists a similar neuritic bilateral optic neuropathy (ASBON) behaves differently from that associated with demyelinating disease and is characterized by a good visual prognosis and a low incidence of subsequent bilateral optic disease.1,2 Here we describe a case of ASBON after hepatitis A infection.

A 22 year old female nurse presented in October 1992 with a three week history of malaise, lethargy, and recurrent febrile pain, followed by the onset of jaundice. A full infectious screen detected hepatitis A specific IgM antibody only. One week later she developed right eye acuity which was exacerbated by movement. After a further week the visual acuity in her right eye deteriorated, followed two days later by her left eye. On presentation to the eye department, the visual acuity was 1/60 in the right eye and 6/18 in the left eye. There was a right relative afferent pupillary defect and colour vision was impaired in both eyes. Fundal examination showed bilateral asymmetric optic discs with right disc more swollen. Automated examination of the visual field showed diffuse field loss in both eyes. Apart from the presence of jaundice, general medical and neurological examination was normal.

Over the next three days the visual acuity fell to "no perception of light" and "perception of light" in the right and left eyes respectively. Head and orbit CTs showed no abnormality. MRI of the head did not show any white matter lesions and views of the optic nerves were normal. Visual evoked potentials showed prolonged latencies in both eyes, with averaged P2 latencies of 139 ms on the right and 121 ms on the left. Auditory and somatosensory potentials were normal. Lumbar puncture showed clear CSF, with an opening pressure of 10-5 cm H2O. The CSF protein was 0.20 g/l, CSF IgG was 0.03 g/l, and there were no white cells or oligoclonal bands. Treatment with intravenous methylprednisolone and oral prednisolone was started.

Twelve days after the initiation of treatment the visual acuity had improved to "count fingers" and 6/5 in the right and left eyes. After one month the acuity had improved further to 6/18 and 6/5 respectively, and after four months to 6/12 and 6/5. One year after the onset of symptoms the patient had developed no further ocular or neurological symptoms. There was, however, temporal and bilateral optic disc swelling and a persistent afferent pupillary defect.

ASBON occurs more often in children, and has been described after immunisation and after several viral and bacterial infections. Cases have been described in adults after fevers and also after chickenpox.3 It is questionable, given the relatively good visual prognosis in adults with ASBON whether both eyes with intravenous and oral steroids was indicated in this patient. Given the profound loss of vision, we thought that steroids were indicated to hasten visual recovery.4 Whereas the visual improvement begins quickly, it continued for over six months after the cessation of treatment. A previous report of patients with unilateral optic neuritis has shown that even those with profound visual loss initially may recover good vision, and this was the case here.5

In any case of adult bilateral optic neuritis the diagnosis of multiple sclerosis must be considered. Given the normal CSF protein and the absence of further neurological symptoms after more than one year of follow up, we believe that multiple sclerosis is most unlikely but that hepatitis A was the causative factor.

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Eyelid opening apraxia in focal cortical degeneration

Tyrrell and associates recently described a novel neurodegenerative syndrome in which patients develop progressive restriction of speech output and orofacial apraxia.6 All patients initially retained normal intellectual capacity. Positron emission tomography showed striking bifrontal hypometabolism, providing a functional neuroanatomical correlate to this clinical picture. Or a considerable orofacial apraxia, the patients described by Tyrrell et al appeared not to show abnormal ocular or eyelid movement.

We encountered a patient who had a visually identical syndrome of progressive speech loss and orofacial dyspraxia. Whereas initial manifestations likely resulted from left frontal lobe dysfunction, she eventually developed pronounced lid opening apraxia, possibly reflecting progression of disease to homologous regions of non-dominant frontal cortex.

A 72 year old woman initially noted difficulty selecting and pronouncing words during telephone conversations in 1991. Her spouse noted that one of the earliest signs was a change in the way her mouth moved when she kissed. Despite a speech disturbance, the patient noted no dysphagia and recalled no other neurological symptoms. The problems gradually advanced to the point that she could only utter single, poorly pronounced words. Nevertheless, she understood spoken language and retained complete expressive communication through writing. Her memory also remained intact. By 1993, the patient's speech initiation movement and her gait became slow and unsteady. No tremor or adventitious movements were ever witnessed. Within 12 months, the patient experienced difficulty
Sumatriptan and daily headache.

T Catarci, G L Lenzi, R Cerbo and C Fieschi

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doi: 10.1136/jnnp.58.4.508

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established scientists from other areas

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logy) into research on schizophrenia and bipolar
disorder as well as to provide sup-

port for innovative research by scientists

already in the field whose funding sources

are limited. Applicants are invited from all

stages of career development.

Awards are for one or two years. They

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involving human subjects and up to $50 000

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be used for salaries, supplies, and equip-

ment, but it is the policy of the Stanley

Foundation not to pay indirect costs for

administration of the award. In 1995, 49 applications

were funded out of a total of 220 received.

Deadline for receipt of applications is 1

March 1996. The 4 page application con-

sists of a brief synopsis of the pro-

ject, a budget, and a list of current and

pending sources of funding. Notification of

awards is made in June and funding to

award recipients begins in August.

The research award applications are

reviewed by a professional selection commit-

tee.

Requests for applications and questions

should be directed to: Research Awards

Coordinator, Stanley Foundation Research

Awards Program, c/o NAMI, 200 North

Glebe Road, Suite 1015, Arlington, VA

22203-3734, USA. Tel (703) 524-7600; fax

(703) 524-9094

Sixth Meeting of the European

Neurological Society June 8-12 1996

Netherlands Congress Centre, The

Hague, The Netherlands.

Administrative Secretariat ENS 1996, c/o

AKM Congress Service, PO Box, 4003

Basel, Switzerland, Tel ++41 61 691 51 11,

Fax: ++41 691 81 89.

British Neurosurgery Research Group

Meeting together with the North

American Research Society of

Neurological Surgeons, 1996.

This joint meeting will be held in Newcastle

upon Tyne, 23–25 May 1996.

For further information contact: Professor

A David Mendelow, Newcastle General

Hospital, Westgate Road, Newcastle upon

Tyne NE4 6BE, UK.

World Federation of Neurosurgical

Societies

Awards to young neurosurgeons.

The World Federation of Neurosurgical

Societies will give five awards to young neu-

rosurgeons for the best papers submitted

for presentation at the XI International

Congress of Neurological Surgery to be held

in Amsterdam, Netherlands 6-11 July 1997.

This will be open to all neurosurgeons born

after 31 December 1961. Each award will

consist of an honorarium of US $1500, a

certificate for the Congress. The papers will

be judged by a committee and must contain

original, unpublished work on basic research

or clinical studies related to neurosurgery.

Young neurosurgeons Committee,

Department of Neurological Surgery,

University of Florida Medical Center,

PO Box 100265; 1600 SW Archer Road

Gainesville, Florida 32610 USA.

The submission should be accompanied

by a supporting letter from the head of the

candidate's neurosurgical department.

The last date for submission is 1 October 1996.

Announcement from the British Neuro-

psychiatry Association: 1996 meetings

The 1996 Winter meeting—a joint meet-

ing with The British Neuropsychological

Society—will be held on Friday 19

January at the London Zoo. “Disorders of

reasoning and perception” is the theme of

the morning session and there will be pre-

sentation of short scientific papers and single

case videos by members of both associations

in the afternoon.

The 1996 Summer meeting will be held

on 14–16 July at Robinson College,

Cambridge. It will include topics on neuro-

development, language, and the presenta-

tion of short scientific papers and single case

vectors by members. The Association’s AGM

will be held on 16 July.

For further details of these meetings please

contact: Sue Gerratt, Administrative

Assistant, BNPA, 17 Clocktower Mews,

London NW 1 7BB. Telephone/Fax: 0171 226

5949.

For details of membership of the BNPA, which

is open to medical practitioners in psychiatry,

neurology, and related clinical neuroscience,

please contact: Dr Jonathan Bird, Secretary

BNPA, Burden Neurological Hospital, Stoke

Lane, Stapleton, Bristol, BS16 1QT.

Telephone: 0117 701212 ext 2925/2929 or

Sue Gerratt at the address given above.

CORRECTIONS


The reference to Osborne et al should be BMJ 1994;308:113.


In table 2 (bottom line) the mean R2 index (range) in the third EMG subclass should be 31 (28-37).