CASE REPORTS

Bilateral optic neuropathy and white dot syndrome following a mycoplasmal infection

M Cordonnier, L E Caspers-Velu, C Jacquemin, C Van Nechel, M Tombroff

Little has been written about *Mycoplasma pneumoniae* and ophthalmology. It is mentioned as an agent that may cause conjunctivitis.^{1,2} Salzmann *et al*³ recently stressed the fact that *Mycoplasma pneumoniae* should be considered in the differential diagnosis of any febrile illness that is accompanied by uveitis or optic disc oedema. The current report describes a case of bilateral optic disc swelling and choroidal lesions occurring after a mycoplasmal infection.

Case report

A 52-year-old woman, without previous medical history, was admitted on 1 December 1991 for sudden blindness in the left eye being preceded 24 hours before by the same event in the right eye. This blindness was accompanied by photopsias. The patient also complained of headache, numbness, and paraesthesia of all four limbs. She did not drink, or smoke, and took no medication on a regular basis. She had just recovered from a flu-like illness that lasted 8 days during which she took minicycline, amoxycillin, and aspirin. Three days before the loss of vision, she had returned to work, although she still had a slightly productive cough.

Physical examination was unremarkable, included pulmonary and ear, nose, and throat examination. Neurological examination showed very brisk myotatic reflexes of the four limbs. There were no meningeal signs. Besides bilateral blindness and pupillary areflexia, other cranial nerves were unaffected. The fundus showed bilateral hyperaemic and blurred papillae, without any haemorrhage or exudate.

SUCCESSIVE BLOOD AND CEREBROSPINAL FLUID FINDINGS

Two lumbar punctures revealed an increased level of protein (71 mg/100 ml on 2 days of December and 79 mg/100 ml 1 month later) with no cells. Protein electrophoresis (agar and electrofocusing) was normal as was glucose concentration. Blood analysis showed no inflammatory component. There were no LE cells or antinuclear antibodies. Immunoglobulins were normal. Protein electrophoresis and immunoelectrophoresis were normal. Circulating immune complexes were found (fivefold the normal maximal level). Early and late viral and bacteriological tests were negative for infectious mononucleosis, herpes simplex, cytomegalo-

virus, influenza A and B, para-influenza, syncytial respiratory virus, enterovirus and adenovirus, HIV, HBS, syphilis, psittacosis, ornithosis, *Legionella*, *Chlamydia*, *Yersinia*, and *Borrelia*. However, on 2 days of December – that is, 2 weeks after the flu-like illness, the anti-*Mycoplasma* antibody titre was 1/320, and the cold haemagglutinin titre was 1/128. One month later, the anti-*Mycoplasma* antibody titre was higher than 1/1280. This level stayed unchanged in March and June 1992. The cold haemagglutinin titre dropped to 1/8 at the beginning of January 1992 and to 1/2 in March 1992.

Methanol was absent in blood. A mitochondrial DNA study did not show any mutation responsible for Leber optic neuropathy.

RADIOLOGICAL FINDINGS

The chest x ray was normal. Cerebral magnetic resonance imaging performed the day after admission showed multiple hyperintense foci bilaterally and symmetrically in the supratentorial white matter, away from the ventricles. All of them were enhanced by contrast. They were observed every month until May 1992. At that time, cerebral magnetic resonance imaging had returned to normal.

OTHER INVESTIGATIONS

Lower limb somesthetic evoked potentials showed bilateral slowing of conduction velocity in December 1991. Acoustic evoked potentials were normal at that time. Six months later, the somesthetic evoked potentials had returned to normal.

OPHTHALMIC FINDINGS

Throughout 10 months of follow up, visual acuity was reduced to no light perception in the right eye. In the left eye, visual acuity developed very slowly from no light perception to slight perception of hand movements at 50 cm.

Bilateral papilloedema was initially described, without haemorrhage or exudate. Biomicroscopic examination 1 month after admission found the initial optic atrophy but a normal retinal and vitreous aspect. Between the third and the fourth month after admission, biomicroscopic examination revealed bilateral optic atrophy and disclosed retinal thickening, involv-

Neuro-Ophthalmology
Unit, Hôpital Erasme,
Cliniques Universitaires
de Bruxelles, Brussels,
Belgium
N Cordonnier
C Van Nechel

Hôpital E Cavell,
Brussels, Belgium
Ophthalmology
Department
L E Casper-Velu

Radiology Department
C Jacquemin

Internal Medicine
Department
M Tombroff

Correspondence to:
Dr M Cordonnier.

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Figure 1 Red-free picture of the left fundus (4 months after the acute illness): white spots are visible in mid-periphery.

Figure 2 Fluoroangiographic picture of the left posterior pole: multiple hyperfluorescent spots.

Figure 3 Left fundus (7 months after the acute illness): yellow-white choroidal spots in mid-periphery.

Figure 4 Red-free photograph of the right fundus: tiny white spots are disseminated on the posterior pole.

ing both posterior poles as well as white spots in the middle periphery at the level of the choroid (Fig 1). Fluorescein angiography revealed multiple punctate early hyperfluorescence spots (Fig 2), without cystoid pattern. Seven months after admission, these yellow-white spots were still more numerous in both mid peripheries (Fig 3). Very tiny white spots were also disseminated on both posterior poles (Fig 4). The retinal thickening was not apparent. Fluoroangiography revealed early mottled hyperfluorescence of the whole posterior pole and mid periphery (Figs 5A, 5B) accentuating later (Figs 5C, 5D).

The visual evoked potentials were flat. Electroretinography performed 5 months after admission was normal. Electro-oculography was impossible owing to absence of adequate fixation.

TREATMENT

During the acute phase, intravenous prednisolone was given (100 mg/day) for 10 days and then gradually tapered off until the end of March. Erythromycin was added during the second week of treatment (1.5 g/day) for 11 days. Because no visual improvement occurred, a third treatment was tried at the beginning of January: 400 mg/kg/day intravenous immunoglobulin (Sandoglobin) for 1 week. This was done twice at 3 week interval, and led to a slight improvement in the visual acuity of the left eye (the patient started to see shadows instead of no light perception).

Comment

Neurological complications of mycoplasmal infection are rare. Ponka⁴ found 4.8% of central nervous system manifestation in hospitalised patients with serologically verified *Mycoplasma pneumoniae* infection.

Clyde^{5,6} suggested several criteria that should be met in proving the relationship between neurological syndromes and mycoplasmal infections: epidemic period of mycoplasmal infection, appropriate age of the patient (greatest incidence between 5–40 years, while any age group may be involved), appropriate season (more common in autumn and early winter), compatible clinical illness (febrile tracheo-bronchitis, influenza-like syndrome, or atypical pneumonia), and serological findings compatible with mycoplasmal infection (fourfold or greater rise in specific antibody titre between properly timed acute and convalescent phase sera; fourfold or greater rise or fall in cold haemagglutinins titre). This case fulfils the criteria for linking the neurological disease to *Mycoplasma pneumoniae*.

– There is presently in Europe a *Mycoplasma* epidemic that started in 1991.⁷

– This patient had, during the autumn, an influenza-like syndrome that is compatible with a mycoplasmal infection.

– Neurological symptoms appeared within 14 days of the acute illness.

– There was a more than fourfold rise in anti-*Mycoplasma* antibody titre in paired serums made at 1 month intervals, the first serum being taken 2 weeks after the influenza-like syndrome.

Fig 5A

Fig 5B

Fig 5C

Fig 5D

Figure 5 Early and late fluoroangiographic picture of the right posterior pole (A-C) and peripapillary area (B-D).

Moreover, there was an elevated titre of cold haemagglutinins 2 weeks after the influenza-like syndrome and a more than fourfold fall 4 weeks later.

The description of an optic neuropathy after mycoplasmal infection is not new and we have found several such cases in the published reports (eight to 19), most of them having optic disc swelling. According to the literature, the consequences may vary from irreversible blindness to complete recovery. As in our case, the optic neuropathy usually accompanies other neurological findings (meningoencephalitis, peripheral and/or cranial nerve neuropathy, cerebellar ataxia, transverse myelitis). The pathogenesis of central nervous system involvement is still unknown but is presently regarded as an immunological response to extraneural infection: *Mycoplasma* infection may induce the development of antibodies for normal tissues and *Mycoplasma* itself influences the activity of T and B lymphocytes.²⁰

There are several entities that have been characterised as essentially presenting at some time with multiple white dots in the fundus, usually in the deeper layers of the posterior segment. Nussenblatt²¹ gathers these entities together under the name of 'white dot syndrome'.

They include multiple evanescent white dot syndrome (MEWDS), multifocal choroiditis and panuveitis, acute retinal pigment epithelitis, acute posterior multifocal placoid pigment epitheliopathy (APMPPE), and subretinal fibrosis and uveitis syndrome.

None of these entities seems to correspond to our case: unlike in MEWDS, the white dots of our case are choroidal rather than deep retinal and are not evanescent. The other entities have obviously different ophthalmoscopic and fluoroangiographic features and we will not consider them in detail. The delayed appearance (3 months after admission) of these white choroidal dots and retinal thickening is surprising. The corticosteroid treatment and the immunological disorder caused by *Mycoplasma* may have favoured another infection that was clinically silent but induced a white dot syndrome. On the other hand, both choroidal dots and retinal thickening appeared when steroid dosage was very low (10 mg prednisolone every other day) and 1 month after intravenous immunoglobulin treatment was over. If these fundus anomalies are related to some immunological disorder induced by *Mycoplasma* itself, steroids may have protected the eye from the early development of this white dot syndrome. Intravenous immuno-

globulin therapy may also have played a protective role through its immunoregulatory effect.²²

Conclusion

This case reaffirms that *Mycoplasma pneumoniae* should be considered in the differential diagnosis of an optic neuropathy preceded by a febrile illness. Moreover, it suggests that *Mycoplasma* may play a role in the induction of a white dot syndrome.

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Orbital masquerade: hyperthyroidism and cavernous haemangioma of the orbit

Martin L Leib

Orbit and Plastics Service, the Edward S Harkness Eye Institute, Columbia-Presbyterian Medical Center and the College of Physicians and Surgeons, Columbia University, New York, USA
M L Leib

Correspondence to: Martin L Leib, MD, Director, Orbit and Plastics Service, The Edward S Harkness Eye Institute, Columbia-Presbyterian Medical Center, 635 West 165th Street, New York, New York 10032, USA.

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Unilateral exophthalmos is a challenging clinical problem, and hyperthyroidism, statistically, is one of the most common causes in the adult population.

The possibility of other orbital inflammations or neoplasms coexisting with hyperthyroidism requires emphasis. In this report a case is presented where the patient had a history of chemical hyperthyroidism and cavernous haemangioma of the orbit previously unrecognized.

Case report

A 32-year-old woman presented on 15 September

1982 with a several week history of intermittent blurred vision in her right eye.

Her ocular history included variable exophthalmos in the right eye that was associated previously with documented hyperthyroidism of 3 years' duration. She had received a short course of propylthiouracil and propranolol, and is presently euthyroid.

On ophthalmic examination, her best corrected visual acuity was 20/20+2 right eye and 20/15-1 left eye. The pupils were normal and the extraocular movements were full. She was orthophoric at distance and had 6 prism dioptres of exophoria at near. No diplopia could be elicited.

Orbital examination revealed mild resistance