



A rare ocular manifestation of mycoplasma pneumoniae infection

Une manifestation oculaire rare d'une infection à mycoplasma pneumoniae

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ABSTRACT

Introduction: Ocular manifestations of Mycoplasma Pneumoniae infection are rare. We present a case of Mycoplasma Pneumoniae infection revealed by a recurrent retro-bulbar optic neuritis.

Case description: A 38-year-old woman, initially treated for a typical retro-bulbar optic neuritis of the right eye with corticosteroid therapy at high doses, presented for a recurrent decreased vision in the same eye, associated with blepharospasm and conjunctival hyperemia. The etiological assessment revealed a recent Mycoplasma Pneumoniae infection. The patient was treated with corticosteroids and fluorquinolones. Her visual acuity improved to 20/20 and the other symptoms disappeared. She did not develop any recurrence during follow-up.

Conclusions: Management of atypical optic neuritis in a young adult requires consideration and serologic testing for Mycoplasma Pneumoniae especially in endemic regions.

Key words: Mycoplasma pneumoniae, recurrent, retro-bulbar optic neuritis

RÉSUMÉ

Introduction: Les manifestations oculaires de l'infection à Mycoplasma Pneumoniae sont rares. Nous présentons un cas d'infection à Mycoplasma Pneumoniae révélé par une névrite optique rétro-bulbaire récidivante.

Description de cas: Une femme de 38 ans, initialement traitée pour une névrite optique rétro-bulbaire typique de l'œil droit par une corticothérapie à fortes doses, s'est présentée pour une baisse récurrente de la vision du même œil, associée à un blépharospasme et une hyperhémie conjonctivale. Le bilan étiologique a révélé une infection récente à Mycoplasma Pneumoniae. La patiente a été traitée par des corticoïdes et des fluorquinolones. Son acuité visuelle s'est améliorée à 20/20 et les autres symptômes ont disparu. Elle n'a pas développé de récurrence au cours du suivi.

Conclusions: La prise en charge de la névrite optique atypique chez un jeune adulte nécessite un examen et des tests sérologiques pour Mycoplasma Pneumoniae, en particulier dans les régions endémiques.

Mots clés: Mycoplasma pneumoniae, récurrente, névrite optique rétro-bulbaire

INTRODUCTION

Mycoplasma pneumoniae (*M. pneumoniae*), an atypical pathogen, mainly affects the respiratory tract. It is the most frequently encountered germ in atypical pneumopathies with a prevalence of 22.7% (1). However, it may have extra-pulmonary manifestations, the most common of which are neurological (2,3). Ocular manifestations, other than conjunctivitis, are rare (4).

Aim:

To report the case of a 38-year-old woman who presented with recurrent unilateral retro-bulbar optic neuritis (RBON) secondary to *M. pneumoniae* infection.

CASE REPORT

A 38-year-old woman, with no particular pathological history, consulted for sudden onset decreased vision

and pain in the right eye (RE). The initial examination of the RE showed a best corrected visual acuity (BCVA) of 12/20 with a relative afferent pupillary defect, a normal ocular motility, a calm anterior segment, a calm vitreous and a normal fundus. The examination of the left eye (LE) was unremarkable with a BCVA of 20/20 and a normal fundus (Fig1). Goldmann visual field (VF) showed narrowing of the isoptera with exclusion of the blind spot at the RE (Fig2a). Visual evoked potentials were normal (Fig3). Fluorescein angiography found no abnormalities (Fig1). The usual biological examinations were normal, ruling out in particular any inflammatory syndrome, liver or kidney damage, or blood count disorder. The diagnosis of typical optic neuritis was retained. We completed with a neurological examination that turned out to be normal and by a cerebrospinal magnetic resonance imaging (MRI) which was without abnormalities. The patient was treated with high-dose corticosteroid therapy according to the ONTT (Optic neuritis treatment trial) protocol:

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methylprednisolone 1 g IV qd for three days followed by oral prednisone 1 mg/kg qd for 11 days. The evolution was marked by the disappearance of the pain and by the improvement of the BCVA in the RE to 16/20 on the fifth day of the treatment.

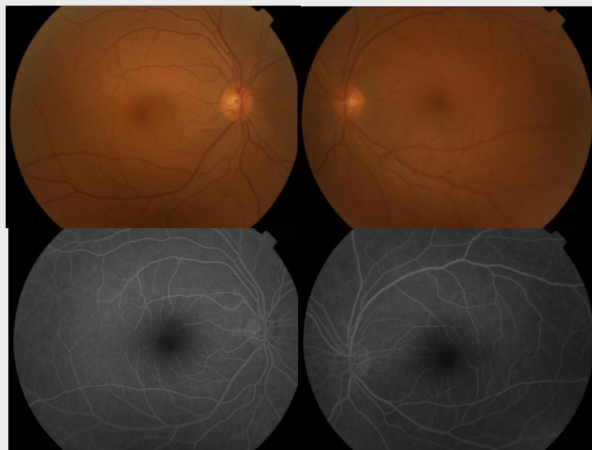


Figure 1. Fundus photos and fluorescein angiography: absence of optic disc edema or other retinal lesions

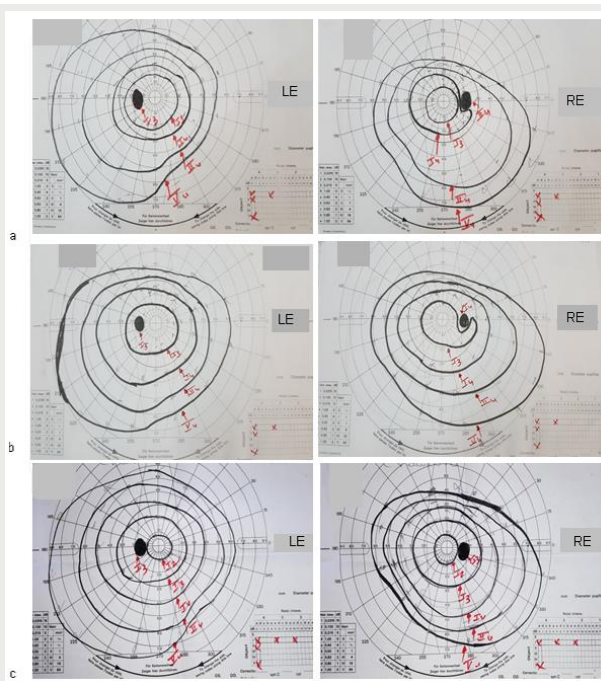


Figure 2. Visual fields
a. During the first episode: narrowing of the isoptera with exclusion of the blind spot in the right eye
b. During the second episode: same aspect
c. After treatment: marked improvement

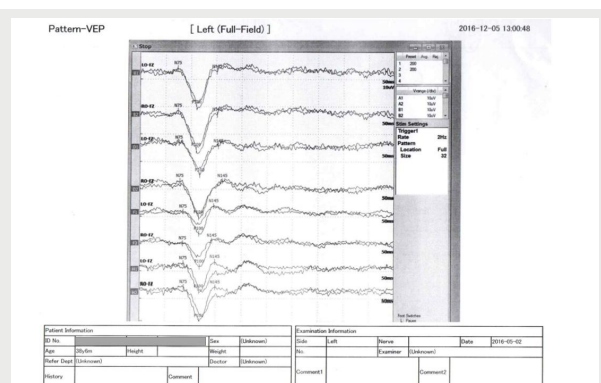


Figure 3. Visual evoked potentials: Normal morphology responses with correct P 100 wave latencies on both sides

A week later, the patient reconsulted for a decreased vision in the same eye. Examination of the RE revealed a BCVA of 6/20, a blepharospasm, a preserved ocular motility, a conjunctival hyperemia, a calm anterior segment, a calm vitreous and a normal fundus (Fig4a). A second VF performed revealed the same results as those of the first (Fig2b) and the orbito-cerebral MRI was without abnormalities. A more exhaustive etiological assessment was started. Complete blood count, urea, creatinine, fasting glucose and transaminases were normal. The sedimentation rate was 23 mm in the first hour. Serologies of Bartonella henselae, Human Immunodeficiency Virus, Cytomegalovirus, Epstein Barr virus, Parvovirus B19 and West Nile virus were negative. The intradermal tuberculin test was negative. Anti-optic neuromyelitis, anti-neutrophil cytoplasmic, antinuclear, anti-tissue and anti-neuronal antibodies were absent. The notion of a neglected respiratory infection preceding the first episode was found and the serologies of the atypical pathogens objectified a recent infection by *M. pneumoniae*: first serology revealing positive immunoglobulins M (IgM) at 42.3 and positive immunoglobulins G (IgG) and second serology 15 days later finding positive IgM at 66.4 and an increase in IgG level of four times. The diagnoses of conjunctivitis with blepharospasm and RBON secondary to *M. pneumoniae* infection were retained. The patient received a second course of corticosteroids associated with systemic fluoroquinolone treatment for 15 days. The evolution was marked by the disappearance of the blepharospasm and the conjunctival hyperemia and by the improvement of the BCVA to 20/20 (Fig4b).

An ophthalmological examination five months later found a BCVA of 20/20 in both eyes and the VF showed a marked improvement (Fig 2c). No recurrence was noted during the following four years.

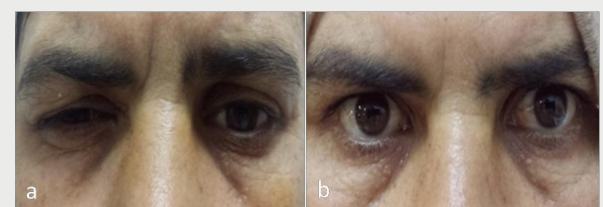


Figure 4.
a. Blepharospasm and conjunctival hyperemia in the right eye
b. Regression of blepharospasm and conjunctival hyperemia in the right eye after treatment

DISCUSSION

M. pneumoniae is an atypical pathogen that primarily causes respiratory tract infection. The presumption is clinical but the diagnostic confirmation is biological based on the elevation of serum immunoglobulin levels (2). The most common extra-pulmonary complications of *M. pneumoniae* are neurological such as meningitis, meningoencephalitis, myelitis, and peripheral neuropathy. In most cases, respiratory illness, usually mild, precedes or coincides with the neurological findings. The interval between the onset of respiratory symptoms and neurologic manifestations has a range of 2–14 days. Respiratory infection can be completely asymptomatic (5). Typically, ocular involvement secondary to *M. pneumoniae* is limited to conjunctivitis. Other manifestations have been reported such as paralysis of the 3rd or 6th cranial nerve, homonymous hemianopsia, nystagmus, anterior uveitis, but very rarely optic neuritis (4). Our patient presented two

manifestations: conjunctivitis and optic neuritis. Optic neuritis following *M. pneumoniae* infection is a rare pathology that mainly affects children. In adults, only five cases have been reported in the literature (table 1) (2,4,6-8). Four patients had respiratory and neurological

symptoms, bilateral involvement and collapsed visual acuity. For our patient, this is the second case of unilateral RBON with relatively good visual acuity and no associated neurological signs.

Table 1. Bibliographic review of optic neuritis secondary to *Mycoplasma pneumoniae* infection in adults

Authors	Age (years)	Sex	Respiratory symptoms	Neurological symptoms	Unilateral or bilateral involvement	Initial visual acuity	Ocular fundus	MRI
Ginestal and al (2004) [2]	69	M	Present	Frontal headache	B	RE : 2/70 LE : 2/200	Optic disc edema	Normal
Milla and al (1998) [3]	37	F	Present	Frontal headache	B	RE : HM LE : LP	Optic disc edema	Focal left periventricular lesion
Pfausler and al (2002) [5]	53	M	Present	Tetraparesis with loss of deep sensation	B	No LP	Not mentioned	Normal
Choi and al (2017) [6]	20	F	Absent	Absent	U, RE	RE : 8/20	Normal	Enhancement of right optic nerve
Baheerathan and al (2016) [7]	39	F	Present	Guillain-Barré syndrome	B	No LP	Optic disc edema	Enlargement of both optic nerves with gadolinium enhancement
Our case	38	F	Present	Absent	U, RE	RE : 6/20	Normal	Normal

M: male; F: female; B: bilateral; U: unilateral; RE: right eye; LE: left eye; LP: light perception; HM: hand motion; MRI: magnetic resonance imaging

The physiopathological mechanisms of the extrapulmonary and in particular neurological manifestations of *M. pneumoniae* infection have been widely reported in the literature. Direct invasion by the pathogen and the post-infectious immune reaction are the main mechanisms involved (9). Pfausler B and al (6), in their study detected anti-myelin and anti-neural tissue antibodies. Their elimination by plasmapheresis may have improved neurological symptoms, and the authors thus suggested an autoimmune mechanism. Bitnun and al (10), in a large series, identified the *M. pneumoniae* antigen in the cerebrospinal fluid and/or in the throat of patients with at least 5-7 days of respiratory symptoms, and they concluded that this infection can have a cytotoxic effect on the respiratory epithelium facilitating blood invasion by *M. pneumoniae*. However, the coexistence of respiratory and visual symptoms and high levels of anti-*M. pneumoniae* antibodies highlights the possibility that the two infectious and autoimmune phenomena are concomitant, a possibility also reported by Bae JW and al. (11). Knowledge of the pathophysiological mechanisms is very important for the therapeutic choice and this infectious and immune intricacy could justify corticosteroid and antibiotic therapy association and thus explain the initially partial improvement of our patient under corticosteroids alone.

CONCLUSION

In conclusion, the etiological diagnosis of atypical optic neuritis in a young adult must include serology for *Mycoplasma pneumoniae*, especially in endemic countries. The case of our patient shows the interest of combining corticosteroids and antibiotics for the treatment of this type of optic neuritis.

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