Nuclear bilateral Bell’s palsy and ageusia associated with *Mycoplasma pneumoniae* pulmonary infection

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This case report describes a case of nuclear bilateral Bell’s palsy and ageusia associated with *Mycoplasma pneumoniae* infection. Magnetic resonance imaging evidenced T₂-weighted hyper-intense protuberantial lesions. Such topography leading to a nuclear palsy contrasts with previously reported infectious diplegia involving only peripheral facial nerves, and has not yet been described in the spectrum of *M. pneumoniae* post-infectious neurological manifestations.

Introduction

Neurological complications occur in approximately 5% of patients hospitalized for pneumonia due to *Mycoplasma pneumoniae* (Ponka, 1980). Encephalitis and meningoencephalitis are the most frequent complications within the wide heterogeneous spectrum of *M. pneumoniae* neurological manifestations (Koskiniemi, 1993). Conversely, bilateral Bell’s palsy (BBP) due to *M. pneumoniae* infection has been reported in only four cases and without any magnetic resonance imaging (MRI). In concordance with observations in other infectious diplegia, due to Varicella-zoster virus, Herpes simplex virus and Epstein–Barr virus (EBV) (Morgan et al., 1995; Ramsey & Kaseff, 1993; Volter et al., 2004), peripheral facial nerve involvement has been suggested. This report describes what we believe to be the first case of nuclear BBP and ageusia due to *M. pneumoniae* with normal cerebrospinal fluid (CSF) and confirmed by protuberantial lesions on MRI.

Case report

A 49-year-old man was hospitalized in our department for fever and non-productive coarse cough. No history of allergy to antibiotic-drugs, particularly beta-lactamin, was recorded. At the time of admission (day 0), he had fever up to 39.7°C. Pulmonary auscultation and neurological examination results were normal. No peripheral lymphadenopathy was noted. Chest X-ray and thoracic CT (computed tomography) scan showed isolated pulmonary left higher lobe infiltrate. Lymphopenia (1400 per mm³) and inflammatory syndrome [50 mg L⁻¹ C-reactive protein (CRP)] were evidenced on routine blood tests.

Intravenous cefotaxime was initiated on admission (day 0), without any clinical improvement 72 h later. Given this absence of clinical improvement, cold agglutinin antibodies, which are highly associated with *M. pneumoniae* pneumonia (Cherry, 1993) when positive, were searched for and found to be negative. Ofloxacin (200 mg twice a day) was then started (day 3). The fever disappeared within 48 h after the ofloxacin switch, which was delivered for a total of 3 weeks. Meanwhile, CRP returned to a normal value.

A right facial palsy occurred on the ninth day of admission (on the sixth day of ofloxacin treatment). Twenty-four hours later (day 10), BBP and right predominant lingual hypogeusia were observed, associated with a bilateral erythematous maculopapular palmar rash (Fig. 1a). Concomitantly, the monocyte and total lymphocyte cell counts rose to 4200 per mm³ and 2000 per mm³, respectively, with a thrombocytosis at 630,000 per mm³ without any recurrence of CRP elevation or fever. CSF analysis (day 9) was normal, with no detection of intrathecal immunoglobulin synthesis and negative bacterial cultures. Herpes simplex virus, EBV and cytomegalovirus serologies and amplification (PCR) were negative in CSF and blood plasma samples. No specific signs of polyradiculoneuritis were observed on an electromyogram. A cerebral CT scan was normal but cerebral MRI revealed T₂-weighted hyper-intense lesions in the protuberantial area (Fig. 1b).

In addition to ofloxacin, the patient received intravenous steroid and aciclovir between day 10 and day 15. A first serum sample at the time of admission was negative for *M. pneumoniae* antibodies but a sample 2 weeks later was positive, with a titre up to 300 on a specific complement-fixing antibody test (de Ory et al., 2004). PCR for *M.
pneumoniae and a specific complement-fixing antibody test of CSF were negative. Aciclovir and intravenous steroid were discontinued given the absence of Herpes simplex virus detection in CSF and confirmation of M. pneumoniae seroconversion, steroids having not been found to be significantly effective in neurological M. pneumoniae complications (Koskineni, 1993).

In addition to other infectious causes, autoimmune diseases were inquired into, but aetiological investigations, such as antinuclear and anti-neutrophil cytoplasmic antibodies, were negative. Salivary gland histology was normal.

The palmar rash and haematological disorders spontaneously resolved within 5 days, while the patient was still receiving the ofloxacin course. After 4 months, the patient recovered slowly and partially from his diplegia with a persistent right hemi-ageusia. Concomitant cerebral MRI showed a complete resolution of the protuberantial lesion. The patient still complained of hemi-ageusia at 3-year follow-up.

Discussion

The occurrence of BBP gives rise to full investigation, in order to eliminate aetiologies such as sarcoidosis, Wegener granulomatosis, Guillain–Barre syndrome, diabetes mellitus, and tumoural and infectious diseases (herpes zoster, EBV, HIV, Lyme disease and syphilis), none of which were found in our patient. Central nervous system manifestations due to M. pneumoniae have been classified into two distinct patterns, early and late post-infection complications (Narita & Yamada, 2001). Thus, this BBP is more likely to be considered as part of the late post-infection pattern due to the time interval following the M. pneumoniae pneumonia (>7 days), and the negative CSF M. pneumoniae PCR and culture (Narita & Yamada, 2001). Its pathogenesis remains unclear, but the autoimmune hypothesis rather than a direct invasion (Abramovitz et al., 1987) would be the most plausible. Indeed, the delay observed between the pulmonary infection and the onset of neurological signs (Koskineni, 1993), associated with a concomitant maculopapular palmar rash (Cherry, 1993) and hypermonocytosis occurring after 7 days of ofloxacin treatment, is more likely to indicate an autoimmune cause, rather than an allergic or infectious origin.

Four cases of BBP due to M. pneumoniae have been previously reported, without MRI investigation (Ernster, 1984; Klar et al., 1985; Montalban et al., 1986; Morgan et al., 1995). Cerebral MRI has been of significant relevance in the diagnosis of unilateral or bilateral infectious Bell’s palsy. Enhancement of the geniculate ganglia or the facial nerve, in its labyrinthine or horizontal and/or descending segments, has been described (Ramsey & Kaseff, 1993; Tien et al., 1990). In contrast to what has been previously reported in infectious BBP, our patient’s cerebral MRI revealed only protuberantial lesions, which could explain the BBP (nuclear palsy), as well as the gustative failure, given the localization of the protuberantial solitarus nucleus. The initial clinical feature associated with the T2-weighted hyper-intense lesions in the protuberantial area on cerebral MRI, as well as the complete resolution on the 4-month MRI, strengthen the infectious hypothesis rather than the fortuitously concomitant thromboembolic event. In our case report, MRI was sensitive enough to point to the precise localization of the nuclear lesion, which was not the classical one usually reported in infectious BBP (Ramsey & Kaseff, 1993). Thus, MRI imaging helped in the diagnosis and subsequent management of our patient.

In conclusion, we believe that we describe here the first case of nuclear BBP and ageusia due to M. pneumoniae infection for which a cerebral MRI was performed, showing protuberantial area involvement and leading to the diagnosis of nuclear palsy. This topography has not yet been reported in infectious diplegia and may be part of the broad spectrum of M. pneumoniae neurological features. Screening for M. pneumoniae infection should not be omitted in diplegia involving peripheral or nuclear facial nerve.

References


