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## Frequent detection of *Mycoplasma pneumoniae* in Bell's palsy

Received: 13 May 2003 / Accepted: 12 August 2003 / Published online: 24 October 2003  
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**Abstract** The cause of Bell's palsy (BP) remains unknown despite various hints to an infectious etiology. *Mycoplasma pneumoniae* is a common pathogen of the respiratory tract causing pharyngitis, tracheobronchitis or pneumonia. Neurological complications are the most frequent extrapulmonary manifestation. So far, only a few case reports suggested an association between cranial nerve palsy and *M. pneumoniae* infection. Patients with a BP who were admitted to the Department of Otorhinolaryngology or Neurology of the University of Wuerzburg between 2000 and 2002 were tested serologically for the presence of antibodies against *Borrelia burgdorferi*, herpes viruses (HSV-1/2, VZV) and *M. pneumoniae*. The diagnosis of mycoplasmal infection was made when at least one of the following criteria was met: a threefold rise or more in the titer of antibody of *M. pneumoniae* in paired sample or a microparticle agglutination assay (MAG) of  $\geq 1:40$  and the detection of IgA and/or IgM antibodies in the acute phase serum. Ninety-one consecutive patients could be included. Fifteen patients showed a reactivation of a VZV ( $n=12$ ) or of a HSV-1 ( $n=3$ ) infection. In six cases the immunoblot revealed specific antibody bands for *B. burgdorferi*. In 24 patients (26.4%) a seroconversion of *M. pneumoniae* could be detected. Only two patients complained of mild respiratory symptoms. According to our results, *M. pneumoniae* is fre-

quently associated with Bell's palsy. Thus, a routine screening for this pathogen, even in the absence of respiratory symptoms, is necessary.

**Keywords** Facial palsy · Bell's palsy · Etiology · Infectious agents · *Mycoplasma pneumoniae*

### Introduction

So far the etiology of Bell's palsy (BP) is not completely understood [2, 4, 23, 33]. Clinical and epidemiological data point to an infectious origin, which might trigger an immunological response inducing a facial neuropathy [22]. Various infectious agents have been linked to facial palsy, such as the herpes simplex and varicella zoster viruses (HSV and VZV), mumps and the rubella virus, cytomegalovirus and HIV as well as *Borrelia burgdorferi* [5, 23, 16, 32]. The association of facial palsy and the reactivation of a dormant VZV infection was recognized a long time ago. Even in the absence of herpetic skin lesions, reactivation of VZV can be demonstrated in up to 29% (zoster sine herpette) [14]. More recently, the herpes simplex virus has been implicated in the pathogenesis of Bell's palsy [34]. Inoculation of HSV-1 DNA in the tongue or the auricle of mice led to the development of facial palsy [37]. The presence of HSV-1 DNA could be demonstrated in 11 of 15 geniculate ganglia of unselected cadavers by in situ hybridization [13]. Moreover, Murakami et al. identified the HSV-1 genome in the perineural fluid of 11 out of 14 patients with a presumed Bell's palsy using polymerase chain reaction [24]. Nevertheless, in the majority of cases investigations failed to establish a definitive etiology of acute peripheral facial palsy [33].

*Mycoplasma pneumoniae* (MP) is an important pathogen of upper and lower respiratory tract infections. CNS manifestations – mainly meningitis and encephalitis – are the most frequent extrapulmonary complications following mycoplasmal infection [18]. Only a few case reports pointed to an association between an isolated cranial nerve palsy and a mycoplasmal infection [11, 19, 39].

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The aim of this investigation was to study in a large population the role of *Mycoplasma pneumoniae* infection in the pathogenesis of Bell's palsy with regard to other well-known agents causing BP, such as *Borrelia burgdorferi* and the herpes viruses.

## Subjects and methods

We serologically tested all consecutive patients who presented with an acute peripheral facial palsy at the Department of Otorhinolaryngology or Neurology at the University of Wuerzburg between 2000 and 2002. The severity of the facial palsy was evaluated according to the grading scale published by Stennert in 1977 [36].

The search for MP antibodies was carried out in two steps. A microparticle agglutination test (MAG) was used as a screening procedure (Serodia-Myc II Kit, Fujirebio, Tokyo). In case of a titer of  $\geq 1:40$ , an immunoblot was performed to detect circulating serum-specific IgM, IgG and IgA antibodies (Western Blot Genzyme, Virotech GmbH Rüsselsheim, Germany). This Western Blot used different *Mycoplasma*-specific surface-expressed proteins of the strain M 129 such as the P1, P90, P40 and P30 adhesin proteins as well as the P65 and P100 membrane proteins. The diagnosis of a mycoplasma infection was made when at least one of the following criteria was met: a threefold or greater rise in titer of antibody to MP in paired sample or an antibody titer of  $\geq 1:40$  and the presence of IgA and/or IgM antibodies in the acute phase serum.

*Borrelia burgdorferi* (Bd) was tested according to the guidelines of the Deutsche Gesellschaft für Hygiene und Mikrobiologie [15] using a commercially available enzyme-linked immuno sorbent assay (ELISA) followed by an IgG and IgM immunoblot in positive cases (Biotest AG, Dreieich). Criteria for an acute *Borrelia* infection was the detection of at least three specific lanes in the IgG and of two specific lanes in the IgM immunoblot.

Herpes virus diagnostics involved the search for specific VZV- and HSV-IgG and IgM antibodies in the ELISA (Enzygnost, Dade Behring, Marburg, Germany) or the detection of virus genome in a throat swab of the involved side by PCR (for HSV according to Pohl-Koppe and for VZV modified from Puchhammer-Stockl) [28, 29]. Either a significant change of more than twofold in IgG antibody levels or the detection of virus genome was considered as evidence for infection with VZV or HSV.

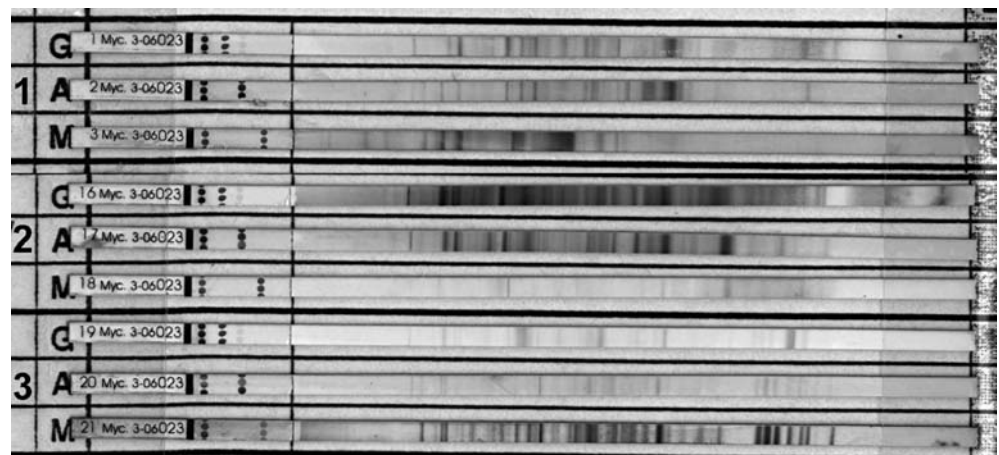
## Results

Ninety-one patients were included in this study. The MAG was positive in 35% (32/91). In total 24 of these 32 pa-

tients also showed a characteristic antibody pattern in the Western Blot (Fig. 1). In 14 cases the immunoblot revealed *Mycoplasma*-specific IgM antibodies; in 10 cases with negative or slightly positive IgM response the diagnosis of an acute infection with MP was established by the presence of IgA antibodies. Whereas in total the mean age was 44.6 years (range of 4–85 years), patients with a MP infection were younger (mean 33 years, range 12–69 years). The gender ratio (man: woman) was 14:10. Ten suffered from a palsy on the left, 12 on the right side, one woman of a recurrent alternating palsy and one man of a bilateral palsy. According to the grading scale of Stennert 16 patients showed a Stennert grade of 5 or less, 8 of more than 5 [36]. The medical history revealed only in two cases mild respiratory symptoms 2 weeks before admission to the hospital. No patient had signs of pneumonia. Cerebrospinal fluid (CSF) specimens were available for 13 of the 24 *Mycoplasma*-positive patients. All patients had normal or slightly elevated cell counts and protein levels except for two patients with clinical signs of an encephalitis, one of them with a concomitant VZV infection. The differential cell count of the CSF revealed a predominance of lymphocytes in ten cases.

The facial palsy improved in all 24 cases except three patients, who developed severe neurologic complications during treatment. One patient had to be admitted to the Department of Neurology because of a cerebellitis 10 days after the onset of the acute peripheral facial palsy. Serologic evidence of both *Mycoplasma* and VZV infection was found. A second patient suffered from a severe meningoencephalitis with elevated CSF cell counts (1180 cells/ $\mu$ l) and protein levels (75 mg/dl). VZV was identified by PCR in CSF. The third patient was admitted to the ENT Department with a facial palsy on the left side (Stennert grade 4). Three days later, the severity of the palsy increased to 9, and he developed palsy on the opposite side. All serological examinations including the search for *Borrelia burgdorferi*, HSV1–2, VZV, EBV, CMV, FSME and HIV1–2 were negative. IgA-specific antibody bands for MP could be detected in the blood and in the cerebrospinal fluid as well. Isolation of the pathogen from the CSF was not possible. Beside these two already mentioned patients

**Fig. 1** Examples of an immunoblot searching for *Mycoplasma pneumoniae* antibodies (G IgG antibodies, A IgA antibodies, M IgM antibodies); patient 1 showed the presence of anti-*Mycoplasma pneumoniae* IgG, IgA and IgM antibodies; in patient 2 immunoblot revealed only a positive IgG and IgA response and in patient 3 a positive IgM, but negative IgG and IgA response



**Table 1** Results of the mycoplasma positive patients ( $n=24$ ) (MAG microparticle agglutination assay; + positive, (+) slightly positive, 0 negative; immunoassay: detection of IgG, IgA and IgM antibodies, clinical presentation, mixed infection with HSV-1 or VZV)

No.	MAG	IgG	IgA	IgM	Clinical presentation	Mixed infection
1	1:40	(+)	+	+		
2	1:40	(+)	+	(+)	Respiratory s.	
3	1:160	+	+	0		
4	1:40	+	+	+		
5	1:1280	+	(+)	+	Respiratory s.	
6	1:160	(+)	+	+		
7	1:40	(+)	+	(+)		
8	1:80	+	+	0		
9	1:40	+	+	+		
10	1:40	+	(+)	+		
11	1:80	+	+	(+)	Meningitis	
12	1:160	(+)	0	+		

**Table 2** Results of the mycoplasma positive patients ( $n=24$ ) (MAG microparticle agglutination assay; + positive, (+) slightly positive, 0 negative; immunoassay: detection of IgG, IgA and IgM antibodies, clinical presentation, mixed infection with HSV-1 or VZV)

Nr	MAG	IgG	IgA	IgM	Clinical presentation	Mixed infection
13	1:80	+	+	(+)		
14	1:160	(+)	0	+		
15	1:160	+	+	(+)		
16	1:80	+	+	(+)		
17	1:640	+	+	0		HSV-1
18	1:160	(+)	+	+	Meningitis	VZV
19	1:80	(+)	+	+	Meningitis	VZV
20	1:40	+	+	+		
21	1:80	(+)	(+)	+		
22	1:40	+	+	+		
23	1:160	+	+	+		
24	1:80	+	+	(+)		

with a mixed infection of VZV and MP, a third patient had serological evidence for a concomitant infection with HSV-1. Tables 1 and 2 summarize the serological and clinical data of the mycoplasma positive patients.

Antibodies against *Borrelia burgdorferi* could be found in 6 of the 91 patients (6.6%). Serological evidence for a reactivation of herpes virus was established in 13 patients ( $n=12$  VZV,  $n=1$  HSV1/2). Additionally, HSV-1 DNA could be amplified in a throat swab in two cases by PCR. Fifty percent of all the patients with a VZV infection did not show skin lesions at the onset of the facial palsy; in two cases herpetic vesicles appeared several days later. Two patients suffered from a herpetic eruption on the auricle without serological evidence of a reactivation of zoster virus. Mixed infection with *Borrelia burgdorferi* ( $n=2$ ) or *Mycoplasma pneumoniae* ( $n=3$ ) was detected in five cases.

## Discussion

In 1938 Reimann firstly described a tracheobroncho-pneumonia in seven patients that was so different from the

classical bacterial pneumonia that he proposed this disease as a new etiologic entity caused by an unknown infectious agent [30]. It wasn't until 1961 that Chanock could clearly show that *Mycoplasma pneumoniae* is the pathogen of this so-called atypical pneumonia [8].

*Mycoplasma* is a pleomorphic organism living as a parasite on the surface of epithelial cells. Because of the absence of a cell-wall, they are resistant to cell-wall-active antibiotics such as penicillin and cephalosporin. For humans, only *M. pneumoniae*, *M. hominis* and *Ureaplasma urealyticum* are pathogenic [7]. Although 20 to 45% of all clinical pneumonia are caused by mycoplasma, only 3% of all patients suffering from a serologically proven mycoplasma infection develop pneumonia. Seventy-seven percent only show minor respiratory illnesses such as pharyngitis or tracheobronchitis and even 20% are asymptomatic [9, 38].

Laboratory diagnosis of mycoplasmal infection is mainly based on serological tests because the organism is difficult to isolate [17, 31]. The complement fixation test, which is most widely used, lacks specificity because of cross-reaction. The micro particle agglutination assay (MAG) and the enzyme-linked immunosorbent assay (ELISA) are more specific and sensitive, but require paired sera for diagnosis. We used a Western immunoblot technique, which is based on different *Mycoplasma*-specific surface-expressed antigens. Nevertheless, IgM response can be missing during acute infection in more than 22%, especially in re-infection and in the elderly [35, 38]. Thus, a search for IgA antibodies is also important. In our study, diagnosis of an acute MP infection was done in 10 of the 24 patients with a negative or slightly positive IgM response by the presence of IgA antibodies. A correlation with the patients' age as described in the literature could not be found.

CNS manifestations, occurring at an incidence of 1:1000 patients, are the most frequent extrapulmonary complication following mycoplasmal infection [20]. The spectrum of clinical presentation is wide: encephalitis, meningitis, myositis, cerebral vasculitis as well as Guillain-Barré-like syndrome or coma have been reported [1, 27, 20, 31]. The underlying pathogenic mechanism is still uncertain. Several hypotheses have been postulated, including the direct invasion of the nervous system, the production of a neurotoxin or an autoimmune mechanism [27]. Histopathologically, perivascular infiltration, edema, microthrombi and signs of demyelination have been described at autopsy [12]. Examination of the cerebrospinal fluid (CSF) usually shows slightly elevated protein levels with cell counts in the normal range [10, 38].

Whereas to date the association between encephalitis and mycoplasma infection is accepted, so far only five case reports deal with peripheral facial palsy and a simultaneous *Mycoplasma* infection. In 1984, Ernster reported a bilateral facial paralysis in a 35-year-old man who had suffered from a flu 2 weeks before admission to the hospital [10]. Beside an elevated antibody titer against MP, also evidence for a VZV and a CMV reactivation was found. A bilateral palsy and a complement fixation reaction of 1:128 was also found by Klar in a 1-year-old infant 4 weeks

after a febrile illness [19]. In 1992, Morgan published the case of a 44-year-old man suffering from an unilateral facial weakness and a serologically proven MP infection without pulmonary symptoms [23]. Three years later, Morgan described a 7-year-old girl with pneumonia and a concomitant bilateral facial palsy [22]. In 1999, Papaeangelou encountered a 4-year-old child with a left facial palsy [26]. Eleven days before the child was admitted to the hospital because of a progressive respiratory distress. X-ray of the lung revealed bilateral interstitial infiltration. Serological studies showed positive reaction in the IgG and IgM ELISA and an elevated complement fixation test of 1:1024.

To our knowledge this is the first report about a high incidence of MP infections in patients with Bell's palsy. As screening for mycoplasma antibodies is routinely done only in cases with pulmonary symptoms, the number of *Mycoplasma* infections might be higher than recognized so far [7, 25, 38]. The absence of respiratory symptoms in neurologic complications following mycoplasma infection is not a rare event [25]. In a study of neurological diseases associated with MP infection, Thomas found that only one of 13 patients had pneumonia [38]. Similar observations have been reported by Lerer and by Bitnun [21, 3]. Only two of our patients suffered from a preceding respiratory illness.

There is no consensus regarding to the therapy for central nervous complications caused by mycoplasma infections [3, 20, 26, 38]. However, most authors suggest the use of corticosteroids and tetracycline or erythromycin. In vitro, nitric oxide production triggered by MP in glial cells could be inhibited by glucocorticoids [6]. In our study all patients were treated with prednisolone (Soludecortin® H 250 mg/day for 3 days, followed by 1 mg/kg body weight for 1 week), valacyclovir (Valtrex® 3×1000 mg/day for 7 days) and additionally with cefotaxime (Claforan® 2×1–2 g/day) or ceftriaxone (Rocephin® 1×2 g/day) when bacterial infection was suspected. In case of a proven infection with MP, antibiotic treatment was changed either to erythromycin or doxycycline. Interestingly, patients with mycoplasma antibodies were younger (median age of 23 years) in comparison to the study population in total (median age of 40 years). Moreover, three of the five published cases involved small children.

Mixed bacterial or viral infections concomitant to MP infection have been reported frequently in the literature [3, 7, 10, 20]. Bitnun detected co-infection with one pathogen or more (including respiratory viruses and herpes viruses) in 9 of 20 children with MP infection. The underlying immunological mechanisms are not yet understood, but a mycoplasma-induced nonspecific immune activation has been supposed [3]. We observed reactivation of herpes viruses in 3 of the 24 mycoplasma-positive patients (VZV  $n=2$ , HSV-1  $n=1$ ).

## Conclusion

The incidence of *Mycoplasma* infection in patients with Bell's palsy is higher than previously has been recognized

(26.4%), especially in younger patients. An infection with the well-known pathogens such as *Borrelia burgdorferi* or herpes zoster is less frequent. Thus, each patient presenting with a peripheral facial palsy of unknown origin should be tested for antibodies against *Mycoplasma pneumoniae*, regardless of whether they have signs of respiratory illness or not. In case of positive infection, effective antibiotic treatment should be applied.

**Acknowledgements** We thank R. Martinez, MD, Department of Neurosurgery, University of Dresden, for helpful discussions.

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