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ORIGINAL COMMUNICATION

Differential diagnosis of peripheral facial nerve palsy: a retrospective clinical, MRI and CSF-based study

Julia Zimmermann[1] · Sarah Jesse[1] · Jan Kassubek[1] · Elmar Pinkhardt[1] · Albert C. Ludolph[1]

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Abstract

Background Facial nerve palsy is the most common cranial nerve disorder. There is no consensus on a single diagnostic tool deemed as the 'gold standard' for distinguishing between idiopathic (Bell's palsy) and symptomatic causes. The diagnosis is one of exclusion and most often made on physical examination. In the present study, we describe the etiological background of peripheral facial palsy in $N=509$ patients and evaluate the relevance of cerebrospinal fluid (CSF) analysis and magnetic resonance imaging (MRI) in differential diagnosis.

Methods We carried out a retrospective data analysis of 509 patients with the clinical diagnosis of peripheral facial palsy admitted to our emergency unit between January 2006 and January 2017. All patients were seen clinically; their CSF was analyzed and MRI was performed.

Results Of $N=526$ patients with isolated facial palsy, 17 patients were excluded because they did not consent to CSF analysis. Of the remaining $N=509$ patients, 383 patients (75.2%) were diagnosed with idiopathic facial palsy. In the remaining 126 patients (24.8%), the following etiologies for facial palsy could be found: Ramsay-Hunt-Syndrome ($N=34$), Lyme Neuroborreliosis ($N=32$), other viral/bacterial central nervous system (CNS) infections ($N=8$), neoplasias ($N=18$), autoimmune disease ($N=12$), otogenous processes ($N=6$), or other etiologies ($N=16$). Analysis of the CSF showed 85% sensitivity for Ramsay-Hunt-Syndrome and 100% for Lyme Neuroborreliosis and other viral/bacterial CNS infections. CSF analysis proved a reliable diagnostic tool for identifying these subgroups. MRI with contrast compounds, as performed in 409 patients, was the most important tool in diagnosing neoplasias (88% sensitivity) and otogenous processes (83% sensitivity). MRI with contrast-enhancing compounds did not reveal additional information concerning inflammatory facial nerve lesions when performed the same day as hospital admission.

Conclusions Although peripheral facial palsy was predominantly idiopathic (75.3%) in our cohort, the disease was caused in approximately 25% of the patients by factors which require specific treatment. In the present study, CSF analysis proved to be the leading method for the diagnosis of Ramsay-Hunt-Syndrome, Lyme Neuroborreliosis, and other CNS infections. These subgroups made up approximately 15% of our cohort. To detect these subgroups reliably, routine use of CSF analysis in peripheral facial palsy may be advisable, whereas MRI proved to be useful for exclusion of otogenic and neoplastic processes with a sensitivity of 83% and 88%. We found that the use of MRI with contrast-enhancing compounds does not provide additional diagnostic information on the day of hospital admission. Hence, the potential benefits of routine use of MRI in patients with facial nerve palsy should be weighed against health care cost factors.

Keywords Facial nerve palsy · Facial paralysis · Etiologies · Cerebrospinal fluid · MR-imaging · Ramsay-Hunt-Syndrome · Neuroborreliosis

Introduction

According to the literature, among all peripheral facial nerve palsies, idiopathic or Bell's palsy (BP) constitutes the majority (60–75%) of cases [1–3]. The underlying etiopathogenesis remains unknown, although some authors suggest the reactivation of herpes simplex virus or autoimmunological

✉ Albert C. Ludolph
albert.ludolph@rku.de

[1] Department of Neurology, University of Ulm, 89081 Ulm, Germany

processes against peripheral myelin as factors [4, 5]. The incidence of BP ranges from 20 up to 37.7:100.000/per year with equal distribution of both sexes and the affected side of the face [1, 2, 6–8]. Apart from BP, there are heterogeneous but specific etiologies that cause symptomatic peripheral facial palsy, including infections, such as Ramsay-Hunt-Syndrome or Lyme Neuroborreliosis, neoplastic or otogenic lesions, autoimmune diseases, and trauma [1, 3].

Guidelines for the use of appropriate diagnostic tools to distinguish between symptomatic and idiopathic cases are inconsistent [9–11]. The present large study of $N = 509$ patients presents data of descriptive nature on peripheral facial palsy as well as the results of CSF analysis in each participant. We further analyze the results of magnetic resonance imaging (MRI) in $N = 493$ patients, 409 of whom received a contrast agent. Our study demonstrates the diagnostic impact of CSF analysis and provides a more nuanced view of MRI studies in peripheral facial palsy.

Methods

This study retrospectively evaluates the discharge reports from patients with the clinical diagnosis of peripheral facial palsy, who were admitted to our hospital between January 2006 and January 2017. All 526 patients were examined by a board-certified neurologist and all but 17 received a complete CSF examination. The 17 patients who did not receive a full CSF exam were excluded. A complete medical history was taken, including comorbidities, such as diabetes mellitus or arterial hypertension. In addition, chart notes were analyzed regarding recurrent palsies or pregnancy.

This study was performed in compliance with ethics committee standards at the University of Ulm.

Lumbar puncture was carried out in 509 patients. Analysis of the cerebrospinal fluid (CSF) included white cell count (per µl), total protein (mg/l), lactate (mmol/l), CSF/serum albumin ratio ($\times 10^{-3}$), oligoclonal IgG bands, and antibody indices to varicella-zoster virus (VZV), herpes simplex virus (HSV 1 and 2), tick-borne encephalitis virus, and Borrelia Afzelii in combination with measurement of CXCL13 (cut-off > 300 pg/ml). Specific antibody indices (e.g., AI = (CSF/serum ratio IgG borrelia)/(CSF/serum ratio total IgG)) were classified positive when ≥ 1.5.

MRI was performed on the day of hospital admission (1.5 T MR scanner Magnetom TIM Symphony, Siemens, Erlangen, Germany) and was equipped with a 12-channel head coil. The standard MRI protocol consisted of transversal diffusion-weighted imaging (DWI) with apparent diffusion coefficient (ADC), transversal T1-, T2-, and proton density-weighted (PD) turbo spin echo sequences, a transversal T2-star fast low angle shot gradient echo sequence, and a coronar fluid-attenuated inversion recovery (FLAIR) sequence. An additional T1-weighted sequence with contrast agent (gadoteridol in a dose of 0.1 mmol/kg body weight) was administered in 409 of 493 patients.

The standard clinical workup of patients with facial nerve palsy in our clinic includes MRI as the standard imaging modality to use its superior sensitivity for pathology of brain tissue alterations and for the delineation of the cranial nerval structures themselves. In the case of the diagnosis or even suspicion of an otogenic process (such as otitis media, malignant otitis externa, mastoiditis, cholesteatoma or tumors, e.g., of the mastoid or parotid gland), an additional computed tomography scan was performed due to its higher sensitivity to osseous processes like associated bone destruction. All these patients also received a consultation by a specialist in otorhinolaryngology.

Diagnostic criteria for the etiological subgroups were as follows:

- Idiopathic: no identifiable cause for the paralysis
- Ramsay-Hunt-Syndrome: positive PCR to VZV in CSF *or* positive antibody-index to VZV *or* typical herpetic efflorescence in the external auditory canal
- Lyme neuroborreliosis: positive antibody-index to Borrelia in CSF or positive CXCL13
- Neoplasia: intracerebral, intrathecal, or meningeal neoplasia with anatomical neighborhood to the facial nerve or its nucleus *or* the presence of tumor cells in CSF *or* neoplasia of the mastoid or parotid gland
- Viral/bacterial CNS infection other than HSV, VZV, and Borrelia: pleocytosis > 20 leukocytes/µL without detection of causative pathogen
- Autoimmune: patients with multiple sclerosis, Guillain–Barré-Syndrome, Miller-Fisher-Syndrome *or* autoimmune encephalitis
- Otogenous: patients with otitis media *or* malignant otitis externa *or* mastoiditis *or* cholesteatoma
- Uncommon etiologies: other rare causes of peripheral facial palsy, e.g. Brucellosis, Sarcoidosis.

Results

Etiology

Of 509 patients, a specific cause for facial nerve palsy was detected in 126 patients. 383 patients were diagnosed with idiopathic facial palsy. The distribution of underlying etiologies is shown in Table 1.

The subgroup ‘neoplasia’ is characterized by various types of tumors: 50% of palsies were caused by leptomeningeal spread of breast cancer, prostate carcinoma, non-small cell lung carcinoma or cancer of unknown primary (CUP). These findings underline the importance of precise

Table 1 Proportions of etiological subgroups in peripheral facial nerve palsy

Total	$N=509$	100%
Idiopathic (Bell's palsy)	$N=383$	75.25%
Ramsay-Hunt-Syndrome	$N=34$	6.68%
Lyme Neuroborreliosis	$N=32$	6.29%
Neoplasia	$N=18$	3.54%
Other viral/bacterial CNS infection	$N=8$	1.57%
Autoimmune	$N=12$	2.36%
Otogenic process	$N=6$	1.18%
Other etiologies	$N=16$	3.14%

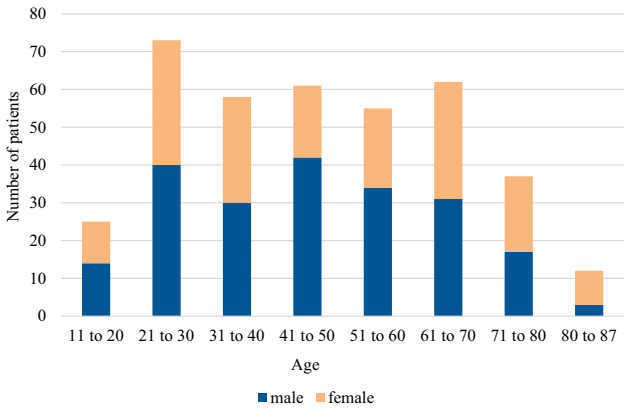

Fig. 1 Age distribution of patients with idiopathic facial palsy

history taking, including all currently existing as well as former illnesses. The remaining patients had uncommon causes of facial nerve palsy, such as cutaneous or cerebral metastases ($N=2$), lymphoma and leukemia ($N=2$), cholesteatoma ($N=1$), carcinoma of the parotid gland ($N=1$), oncocytoma of the parotid gland ($N=1$), or vestibular schwannoma ($N=2$). It is noteworthy that tumors of the cerebellopontine angle accounted for facial palsy in only two cases. The subgroup 'other etiologies' was found to be equally heterogeneous: here, the underlying causes for palsy were HIV-associated meningitis ($N=2$), Brucellosis ($N=1$), tick-borne encephalitis ($N=1$), suspected schistosomiasis ($N=1$), parotitis ($N=1$), Millard–Gubler syndrome ($N=1$), brainstem ischemia affecting the nucleus nervi facialis ($N=1$), neurosarcoidosis ($N=1$), residual paralysis after incomplete recovery ($N=2$), and iatrogenic interventions ($N=3$). Two patients were diagnosed with diabetic mononeuropathy presenting hemoglobin A1c levels higher than 10.0% as a manifestation of insufficient diabetes treatment. The subgroup 'autoimmune' is represented by patients with Guillain–Barré-syndrome ($N=4$), Miller-Fisher-syndrome ($N=2$), multiple sclerosis ($N=2$), and autoimmune encephalitis ($N=4$). Six patients were diagnosed with an otogenic process (otitis media ($N=1$), malignant otitis externa ($N=1$), and mastoiditis ($N=4$)), while other possible etiologies like cholesteatoma were not observed in our series. All these patients showed no osseous destruction in computed tomography and no CSF pleocytosis.

Demography and epidemiology

Idiopathic palsy (Bell's palsy, BP) proved to be the predominant etiology in peripheral nerve palsy. It occurred slightly more often in men than in women (55%:45%) with a mean onset of 47 years (range 11–87; SD 18.9). A detailed age distribution of patients with idiopathic palsy is shown in Fig. 1. 54.8% of patients developed BP between October and March, with a peak arising in January. 27 of BP patients (7.1%) had suffered from facial paralysis previously. In 14 cases (3.7%), the condition arose during pregnancy or postpartum. Both sides of the face were equally affected (right side: 193, left side: 189, bilateral: 1).

Patients showing facial palsy due to neoplasia had a higher mean age (62.5 years; SD 15.5) compared to all other groups. Aside from facial palsy, 45% of these patients showed no further accessory symptoms, such as tinnitus or other cranial nerve disorders. As expected, patients suffering from Lyme Neuroborreliosis were admitted to the hospital only within the months from June to January, with a peak in July. Three of these 32 patients presented with bilateral palsy. 84.4% of the affected patients in this subgroup were male. Notably, of all affected patients, only 34.4% could recall being bitten by a tick in the recent past.

CSF analysis

CSF analysis was performed in 509 patients. Independently of the underlying etiology, 27.1% of this sample showed at least one abnormality in the CSF. In symptomatic facial palsy ($N=126$), the proportion of patients with abnormalities amounted to 73%. The abnormalities in these samples included:

- pleocytosis > 4 leukocytes/µL
- positive antibody-index to HSV, VZV, or Borrelia or positivity of CXCL13
- oligoclonal IgG.

Table 2 indicates the detailed proportions of patients with at least one of these abnormal findings. Under these criteria, CSF analysis showed 100% sensitivity in patients with Lyme Neuroborreliosis and other viral/bacterial CNS infections, and 85% sensitivity in patients with Ramsay-Hunt-Syndrome. These subgroups made up approximately 15% of our cohort and were provided with antiviral or antibiotic therapies.

Table 2 Proportions of patients within the etiological subgroups showing at least one of the following in CSF: pleocytosis, positive antibody-index to HSV, VZV, Borrelia or CXCL13, oligoclonal IgG in CSF

Total	$N=509$	138 (27.11%)
Idiopathic (Bell's palsy)	$N=383$	46 (12.01%)
Ramsay-Hunt-Syndrome	$N=34$	29 (85.29%)
Lyme neuroborreliosis	$N=32$	32 (100.00%)
Neoplasia	$N=18$	10 (55.56%)
Other viral/bacterial CNS infection	$N=8$	8 (100.00%)
Autoimmune	$N=12$	5 (41.66%)
Otogenous process	$N=6$	0 (0.00%)
Other etiologies	$N=16$	8 (50.00%)

Table 3 displays the proportion of patients within the etiological subgroups showing pleocytosis, increased CSF/serum albumin ratio (as a marker for the integrity of the blood–brain barrier) and isolated oligoclonal IgG.

MRI analysis

MRI was performed on 493 patients; a subgroup of 409 patients received contrast agents. In 61 patients, a facial palsy-related pathological finding could be revealed in MRI with all of them showing contrast-enhancement as one sign of structural pathology in these cases.

Table 4 exhibits the proportion of patients within the etiological subgroups who showed contrast-enhancement along the facial nerve, the mastoid, the parotid gland, the meninges, or other structures with special proximity to the nerve or its nucleus. Only 6.02% of patients with idiopathic facial palsy showed enhancement along the facial nerve. Notably, only a fraction of patients with Ramsay-Hunt-Syndrome, Lyme Neuroborreliosis, or other viral/bacterial CNS infections displayed pathologic contrast-enhancement. For neoplasia, MRI with contrast agents showed a sensitivity of 88.2%.

Table 4 Patients showing contrast-enhancement in MRI

	Total number of patients	Patients with contrast-enhancement
	$N=409$	61 (14.91%)
Idiopathic (Bell's palsy)	$N=299$	18 (6.02%)
Ramsay-Hunt-Syndrome	$N=25$	4 (16.00%)
Lyme Neuroborreliosis	$N=29$	5 (17.24%)
Neoplasia	$N=17$	15 (88.24%)
Viral/bacterial/CNS infection	$N=7$	2 (28.57%)
Autoimmune	$N=11$	5 (45.45%)
Otogenic processes	$N=6$	5 (83.33%)
Other etiologies	$N=15$	6 (40.00%)

Discussion

'Bell's Palsy', named after the Scottish anatomist Sir Charles Bell, accounts for 60–75% of all cases of unilateral facial paralysis [1–3]. The etiopathogenesis is still unknown, although viral infections or autoimmunity had been suggested as possible pathomechanisms [5]. In 1972, McCormick suggested HSV-1 as a possible causative agent for BP [12]. Murakami et al. supported this hypothesis in 1996 when they detected the genome of HSV-1 in 11 of 14 samples of endoneurial fluid or the posterior auricular muscle in patients with BP [4]. However, Stjernquist-Desatnik et al. found HSV-1 DNA in muscle biopsies of only 5% in their sample ($N=20$) [13]. The authors claimed that the replication of the virus may already have descended by the outbreak of palsy [13]. By contrast, Linder et al. argued that the mere detection of the viral genome alone does not suffice to show a causal connection between the pathogen and BP [14].

Various authors report an annual BP incidence of 20–37.7/100.000 [1, 6, 7]. There seems to be no notable

Table 3 Proportions of patients within the etiological subgroups showing pleocytosis, increased CSF/serum albumin ratio and oligoclonal IgG in CSF

	Pleocytosis	Increased CSF/serum albumin ratio	Oligoclonal IgG
Total ($N=509$)	111 (21.81%)	174 (34.18%)	70 (13.75%)
Idiopathic (Bell's palsy) ($N=383$)	28 (7.31%)	98 (25.59%)	19 (4.96%)
Ramsay-Hunt-Syndrome ($N=34$)	26 (76.47%)	15 (44.12%)	12 (35.29%)
Lyme Neuroborreliosis ($N=32$)	32 (100.00%)	30 (93.75%)	26 (81.25%)
Neoplasia ($N=18$)	9 (50.00%)	8 (44.44%)	3 (16.67%)
Other viral/bacterial/CNS infection ($N=8$)	8 (100.00%)	5 (62.50%)	0 (0.00%)
Autoimmune ($N=12$)	1 (8.33%)	5 (41.66%)	4 (33.33%)
Otogenous process ($N=6$)	0 (0.00%)	2 (33.33%)	0 (0.00%)
Other etiologies ($N=16$)	7 (43.75%)	11 (68.75%)	6 (37.5%)

gender predominance [1, 8, 15]. In contrast, the amount of right- and left-sided palsies seems to be balanced [1, 2, 8]. Similar to the results in the existing literature which report recurrence rates of 6.8–12.9% in BP, we observed more than one episode of paralysis in 7.1% of our patients ($N=27$) [1, 8, 16].

BP can occur at any age but shows a peak of incidence at the age of 10–40 or 15–45 years, respectively [1, 17]. In our study, the average age of onset was 47 years. Similar results have been reported by other groups, who calculated a mean age of onset of 43.7 and 45 years [18, 19]. As in Spengos et al., we report a peak incidence of BP in January [15]. Notably, the disease shows a higher incidence (43/100.000) in pregnant women [20]. Our data fully support previous epidemiologic findings in BP by other groups.

Besides BP, additional underlying etiologies in peripheral facial nerve palsy exist and these are related to viral agents, traumatic lesions, autoimmune disorders and otogenous as well as neoplastic processes [1, 3]. Even the Coxsackie virus, adenovirus, human herpes virus-6, mycobacterium tuberculosis, human immunodeficiency virus, as well as measles, rubella, and mumps viruses are regarded as causative pathogens [3, 21–25]. Relevant to our data, we found rare cases of peripheral facial nerve palsy in Brucellosis and in tick-borne encephalitis.

Investigations on CSF findings in peripheral facial palsy are sparse. We systematically researched CSF in 509 patients and evaluated the value of this method in the differential diagnosis. In our series, an impaired CSF/blood–brain barrier (BBB) was observed in 25.6% of 383 patients with idiopathic palsy, pleocytosis was found in 7.3%. Weber et al. studied CSF samples of 59 patients with BP and found an impaired BBB in 15%, whereas pleocytosis was detected in approximately 12% [26].

Kohler et al. noted an abnormal CSF in 11% of patients with idiopathic palsy [27]. Similarly, we report a total of 12% of BP patients showing at least one abnormality (Table 2). Other authors reported a higher proportion of patients (25%) with abnormal CSF, including pleocytosis, an impaired BBB, or intrathecal synthesis of immunoglobulins [26]. Our study shows similar increases when elevated total protein and/or BBB impairment were considered. Thus, the number of patients with BP who presented with pathological CSF rose from 12 to 46%.

In summary, the CSF in BP more often resulted in pathological findings in our patients than in the remaining literature. Our data neither verify nor refute the theory of HSV as the pathogenic agent of idiopathic facial paralysis but they show an inflammatory reaction with slight meningeal involvement in at least a proportion of patients with BP. In light of these results, it should be reconsidered whether idiopathic palsy can indeed be linked to a single pathomechanism rather than a range of underlying etiologies.

The diagnosis of Ramsay-Hunt-Syndrome can be made clinically in the presence of the triad of ear pain, facial palsy, and the pathognomonic vesicles of the external auditory canal [28]. Holland and Weiner claimed that "vesiculation may not necessarily appear (*zoster sineherpete*) or may be delayed in up to half of patients" [20]. Consistent with these results, 61.8% of patients with Ramsay-Hunt-Syndrome in our study showed vesicles at the time of clinical examination. In all other patients, diagnosis was made of positive AI to VZV or positive PCR to VZV in CSF. Since patients with Ramsay-Hunt-Syndrome have a poor prognosis and, therefore, should be treated immediately with antivirals, it is essential to detect this subgroup reliably [10, 28, 29]. Some authors believe that up to 8% of patients with idiopathic palsy are, in fact, suffering from '*zoster sineherpete*' [30]. Our findings strongly support this interpretation: lumbar puncture proved to be the only adequate tool for distinguishing reliably between idiopathic palsy or '*zoster sineherpete*'. By inspection alone, up to 38.2% of patients with Ramsay-Hunt-Syndrome would have been misdiagnosed as having BP.

Kohler et al. reported abnormal CSF findings in 11 of 17 patients with Ramsay-Hunt-Syndrome [27]. In our study, 85% showed either pleocytosis, oligoclonal IgG or positive AI to VZV. In Lyme Neuroborreliosis, 100% of affected patients showed either pleocytosis, oligoclonal IgG, positive CXCL13, or positive AI to Borrelia. Under these criteria, lumbar puncture proved to be an excellent diagnostic measure to rule out herpes infection and Borrelia as causative agents.

Since facial palsy occurs in up to 10.6% of patients with Lyme disease, this etiology must be included in the differential diagnosis of peripheral facial palsy [31]. Hyden et al. report that a Borrelia diagnosis may be overlooked if a CSF examination is not performed. Three of their patients had elevated CSF titers to Borrelia but normal serum [18]. Even a precise patient history is not sufficient: similar to our results (34.4%), Bremell and Hagberg found that only 29% of affected patients recollected a tick bite in their recent past [32]. In our series, CSF analysis was the only method that proved to be reliable diagnostically, showing 100% sensitivity in Lyme Neuroborreliosis under the criteria applied for pathologic CSF.

Reiber reported that viral CNS infections usually follow a milder course than bacterial infections [33]. This is also consistent with our findings: In Ramsay-Hunt-Syndrome, 44.1% of the affected patients showed impairment of the BBB and 76.5% pleocytosis (mean leukocytes/µL: 54.7, range 0–323). The total protein was elevated in 58.8% (mean total protein mg/l: 599.3, range 261–1360). In Lyme Neuroborreliosis, pleocytosis could be detected in all affected patients (mean leukocytes/µL: 141.6, range 17–543). Impairment of the

BBB and elevated total protein was found in the CSF of 93.8% (mean total protein mg/l: 1340, range 394–4780).

In summary, CSF analysis is the most reliable method for ruling out viral or bacterial CNS infections in peripheral facial palsy. The number of leukocytes, lactate, and total protein in CSF can even be helpful in distinguishing between viral and bacterial infection. Since patients with a CNS infection (15% of our total sample) require appropriate drug management with antivirals or antibiotics, we recommend the routine use of lumbar puncture in peripheral facial palsy.

The question arises to what extent MRI with a contrast agent may help to differentiate between symptomatic and idiopathic peripheral facial palsy. For the exclusion of neoplastic and otogenous processes, MRI with contrast agents seemed to be the major diagnostic procedure, in case of pathological findings in combination with computed tomography scanning (due to its higher sensitivity to osseous processes) together with the interdisciplinary consultation by an otorhinolaryngology specialist. On the other hand, MRI with contrast agents performed on the same day as hospital admission did not provide additional information about facial nerve-related inflammatory processes. In contrast, Sartoretti-Schefer et al. and Kinoshita et al. both claim that contrast-enhancement of the labyrinthine and distal intrameatal segments are specific for facial nerve palsy [34, 35]. Enhancement of these segments was observed in 43% or 67% by Kinoshita et al. [35], but the mean time between onset of palsy and MRI was 15.4 days (range 1–81) [35]. This latency period may explain the apparent contradiction between the findings of others and those of our study. Burmeister et al. claimed that the maximum nerve swelling in peripheral facial palsy occurs within 3 weeks [36]. The fact that MRI in our investigation was performed on the day of hospital admission might explain our low rates of contrast-enhancement. We reached the conclusion that MRI with contrast agents in the very early phase of facial palsy merely helps to exclude neoplasia and otogenous processes but does not provide any further useful information regarding inflammatory facial nerve lesions. For neoplasia and otogenous process, MRI with contrast agents shows a sensitivity of 88.2% and 83.3%, and, therefore, strengthens the degree of diagnostic reliability for the neurologist and the patient. These two subgroups made up approximately 4.7% of our overall sample. Hence, the potential benefits of routine use of MRI should be weighed against health care cost factors.

Conclusion

Since idiopathic peripheral facial palsy is an exclusionary diagnosis, it is imperative that treatable causes for the disease have to be ruled out before reaching the diagnosis of BP. The heterogeneous etiology of peripheral facial palsy calls for uniform diagnostic procedures that can reliably differentiate between symptomatic and idiopathic palsy. In symptomatic palsy, 73% of affected patients showed at least one of the following abnormalities in their CSF, i.e., pleocytosis > 4 leukocytes/µL; positive antibody-index to HSV, VZV, or Borrelia, or positivity of CXCL13; oligoclonal IgG in the CSF. Hence, in the majority of symptomatic cases (approximately 25% of the overall investigated sample), CSF analysis enabled us to directly diagnose the underlying cause or at least indicated the need for further diagnostic measures. CSF analysis proved to be the major method in ruling out CNS infections, with a sensitivity of 85–100% in these subgroups. For the exclusion of neoplastic and otogenous processes, MRI with contrast agents seemed to be the major diagnostic procedure (in case of pathological findings in combination with computed tomography scanning due to its higher sensitivity to osseous processes together with the interdisciplinary consultation by an otorhinolaryngology specialist), whereas MRI with contrast agents performed on the same day as hospital admission did not provide additional information about facial nerve-related inflammatory processes.

Compliance with ethical standards

Conflicts of interest None to report for all the authors.

Ethical approval This study has been approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

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