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Original Research Article

The degree of damage in the peripheral facial nerve palsy in children depending on the cause: The role of Lyme neuroborreliosis



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ABSTRACT

Introduction: Symptoms of peripheral facial nerve paralysis occur in patients of all age groups. Etiology of this disease is very diverse.

Aim: The aim of this work is to estimate facial nerve damage in relation to presumable cause, including cases in which Lyme neuroborreliosis has been confirmed by laboratory testing.

Material and methods: Thirty-three patients with symptoms of facial nerve paralysis were examined from January to September 2012 in the Department of Pediatric Neurology of the Provincial Specialist Children's Hospital in Olsztyn. Facial nerve function was classified on a three-stage scale created by the authors of this work. Full neurological examination was performed in all patients. Laboratory tests for Lyme-specific antibodies IgG and IgM by enzyme-linked immunosorbent assay has also been performed.

Results and discussion: In the study group, diagnosis of Lyme neuroborreliosis was confirmed in nine patients. Analysis shows that the stage of facial nerve damage in the group with Lyme disease was significantly heavier and that percentage of mild paralysis was significantly higher in the group of patients in whom Lyme disease has not been confirmed.

Conclusions: The role of neuroborreliosis among the infectious factors ought to be emphasized. It seems significant that in patients with confirmed Lyme disease the stage of facial nerve damage was considerably deeper and the recovery was slower.

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1. Introduction

Facial nerve, as one of the most important of the cranial nerves, has a very complex structure and course, which implies a diverse symptomatology of its damage. It consists of a motor root and the part described under the name of intermediate nerve, which contains sensory (afferent) and parasympathetic (secretory) fibers. Motor fibers that reach facial mimic muscles arise from the facial nerve nucleus located between the dorsal pons and medulla oblongata.^{1–3} Symptoms of peripheral facial nerve palsy occur in patients of all age groups, beginning from the neonatal period. Etiology of palsies is very diverse.⁴

The most common unilateral facial nerve damage is defined as idiopathic peripheral facial paralysis – Bell's palsy, which represents 75% of facial nerve paralyzes. Currently, it seems that even these cases have a viral etiology and are often preceded by exposure to cold.^{1,3,5,6}

Recently increasing attention is paid to *Borrelia burgdorferi* infection as a potential cause of the facial nerve paralysis. In case of *B. burgdorferi* infection there are probably two mechanisms responsible for the development of peripheral neuropathies. The first results from the direct interaction between the spirochete and the nerve cell, which leads to its damage, while the second is associated with stimulation of the immune response against this pathogen. Reports in the literature describe the T- and B-cell autoreactivity against endogenous neural structures, which in the presence of 41 kDa *B. burgdorferi* flagellin may elicit inflammatory mediators.^{4,7} Inflammatory and angiopathic lesions, perivascular infiltrates, partially with thrombosis of the vasa nervorum are responsible for the axonal damage of peripheral nerves. Axonal neuropathies, in contrast to direct nerve damage, are less responsive to antibiotic therapy.^{4,7,8}

Secondary causes include inter alia, congenital anomalies, such as Mobius syndrome, trauma, including iatrogenic trauma during surgical procedures, neoplasms, particularly cerebellopontine angle tumors or parotid tumors, and infections.^{1,3,5}

2. Aim

The aim of this work was to assess the degree of facial nerve damage in relation to the presumable cause, with particular reference to cases of elevated antibody titers against *B. burgdorferi* in serum and cerebrospinal fluid (CSF) findings indicating neuroborreliosis.

3. Material and methods

In the period between the beginning of January and the end of September 2012 in the Department of Neurology of the Provincial Specialist Children's Hospital in Olsztyn, 33 patients with symptoms of peripheral facial nerve palsy were examined. Study group consisted of 10 boys (30.3%) and 23 girls (69.7%) aged 4–18 years. Median age was 8.5 years. Facial nerve function was classified on a scale created by the authors. In the

study, visual determination of each separate function was performed: wrinkling the forehead, closing the eyelids, drooping of the corner of the mouth, whistling and grinning. Each of this functions was given a specific number of points (on a scale of 0–3), where 0 points – no damage of the motor function, 1 point – minimal damage that becomes visible by mimic movements with modest asymmetry of the face, 2 points – moderate damage characterized by dysfunction visible also at rest with distinct muscle weakness and facial muscle movements still present with great effort, and 3 points – severe damage to the function with great asymmetry of the face and lack of movements even with great effort.

Corneal reflex was also evaluated. The presence of corneal reflex was given 0 points, uncertain reflex was assessed as 1 point, and lack of reflex as 2 points. In such an examination, the maximum number of points indicating a severe damage was 17 points.

Dependent on the number of points obtained on examination, patients were divided into three groups:

- 1st degree of damage (mild): this group included patients in whom facial nerve function on the scale described above ranged from 1 to 10 points,
- 2nd degree of damage (moderate): range of points from 11 to 13,
- 3rd degree of damage (severe): range of points from 14 to 17.

For all patients a thorough medical history was obtained, that included presumable etiology of facial nerve palsy, particularly tick bite within the preceding six months and potential rash at the site of the tick bite.

Laboratory tests for serum *B. burgdorferi*-specific IgG and IgM antibodies detected by immunoenzymatic method were performed, and in case of positive results, by Western Blot assay. Lumbar puncture was also performed to determine concentrations of *B. burgdorferi*-specific IgG and IgM antibodies in CSF in all patients with elevated serum antibody titers. Pleocytosis and elevated protein concentrations in the CSF were also analyzed.

4. Results

In the examined group of patients it was found that facial nerve paralysis occurs more frequently on the left side and in girls.

Patients, in whom no potential cause of facial nerve palsy was found, constituted a group of 15 subjects (45.5%). Other possible causes in the examined group of patients might have included: multiple sclerosis (2 patients), herpes (1 patient), temporal bone fracture (1 patient), acute otitis media (1 patient), history of chemotherapy for leukemia (2 patients), electrocution (1 patient), and Epstein-Barr infection (1 patient), making a total of 9 cases (27.3%). Elevated serum *B. burgdorferi*-specific IgG and IgM antibodies concentrations were found in the 9 patients (27.3%) (Fig. 1 and Table 1). After obtaining guardian consent, these children had lumbar puncture performed, which confirmed elevated IgM and IgG antibody titers in the CSF in 4 patients. Pleocytosis and elevated protein concentrations in the CSF were found in 2 cases (Table 2).

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