

Bilateral facial nerve palsy in the course of neuroborreliosis in children-dynamics, laboratory tests and treatment

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Abstract

Purpose: Presentation of four patients with bilateral peripheral facial nerve palsy as a clinical manifestation of neuroborreliosis in children – diagnostic, treatment and prognosis.

Material and methods: In 2002-2004 in The Chair and Department of Developmental Neurology, 24 children from the Wielkopolska region were admitted with diagnosis of borreliosis. Among all the children with borreliosis, confirmed by serologic examination, 4 (16.7%) demonstrated bilateral peripheral facial palsy (PFP). We investigated the presence of IgM class and IgG class specific antibodies in the sera and cerebrospinal fluid (CSF) of 4 patients with bilateral PFP. (Detected by immunoenzymatic methods – ELISA.)

Results: Before the occurrence of PFP all the children manifested unspecified systemic symptoms such as headaches, muscle and articulation pains, weakness and in two cases a mood depression. At first all patients demonstrated elevated IgM antibodies and proper levels of IgG antibodies. Control tests administered within 2-14 months later reduction of antibodies was indicated. Two patients demonstrated significant pleocytosis in CSF test, (without the meningeal symptoms). All children were treated with physiotherapeutic procedures and were administered antibiotic intravenously.

Conclusions: PFP is one of the most frequent neurological symptoms of borreliosis in children. In case of acute PFP

and especially the bilateral form of PFP, neuroborreliosis is the most probable diagnosis. All children reported PFP at one side first and after several weeks the paresis of the facial nerve on the opposite side usually appeared. The clinical state of children started to improve after the introduction of physiotherapy and this process usually lasted several months.

Key words: neuroborreliosis, bilateral peripheral facial nerve palsy, neuroborreliosis in children.

Introduction

Acute onset of peripheral facial nerve palsy (PFP) in children is frequently the result of neuroinfection [1,2]. Examination conducted in the 90's by Cook et al. showed that borreliosis was found to be the etiological factor of PFP in 50% of examined children [2]. The results are confirmed by other examinations [3,4]. It is important to remember that PFP may be the only [3,5, 6] or the dominant symptom of borreliosis in children [6,7]. PFP may occur bilaterally, especially in children [6].

Borreliosis, known also as Lyme disease is evoked by Gram-negative Spirochaetes [3,5,7-12] – *Borrelia burgdorferi* [8,10]. The increase of morbidity is the highest among the zoonotic diseases, between 2001-2002 it rose up to 40% in the USA [13].

In Poland, average index morbidity of borreliosis was 2.3/100000 in 1998 [7]. Precise data about morbidity in particular regions of Poland are unknown. Percentage of infected ticks is highest in endemic north-eastern regions (5-58%) [7,12].

On the basis of genetic examinations *Borrelia burgdorferi* Spirochaetes were divided into 11 subtypes [10], out of which 3 are considered to be pathogenic for a human being: *Borrelia burgdorferi sensu stricto*, *Borrelia garinii*, *Borrelia afzeli* [8,10,14]. The differences in geographical occurrence of particular Spirochaetes subtypes were observed: *Borrelia burgdorferi* dominates in the USA, whereas *garinii* and *afzeli* subtypes

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Table 1. Clinical manifestations in patients with serologically confirmed neuroborreliosis

Clinical manifestations	Number	[%]
Headaches	11	45.8
Fatigability, general weakness	8	33.3
Syncope	6	25
Unilateral facial nerve palsy	5	20.8
Osteomuscular pain	5	20.8
Bilateral facial nerve palsy	4	16.7
Dysmnnesia, concentration disorder	4	16.7
Cervicofacial pain on paresis site n. VII	4	16.7
Mood depression	3	12.5
Irritability	2	8.3
Augmentation of cervical and submandibular nodes	2	8.3
Erythema migrans	1	4.2
Inflammation of many articulations	1	4.2
Unpleasant feeling of heart palpitation	1	4.2
Neuronitis vestibularis	1	4.2
Unspecified abdominal, nuchal, leg pain	1	4.2
Dysgeusia	1	4.2
Low tones hypersensitivity	1	4.2

appear mainly in Europe [8,11,14,15], which were not detected in the USA [16]. It was also noticed that *Borrelia garinii* is characterized by affinity to the nervous system and is responsible for the majority of neurological symptoms [6-8,15]. There were also described single cases of parallel infection by two subtypes of *Borrelia* in a patient with bilateral PFP [17].

The infection is evoked by a bite of tick of *Ixodes* family infected by pathogenic *Spirochaetes* [3,7-9,11,14,15,18].

Antibodies of IgM class, detected by immunoenzymatic methods (ELISA) appear within 2-4 weeks [6,11,12,14] reaching the peak within 6-8 weeks [6,12,14], and after 4-6 weeks antibodies of IgG class occur [6,11,14] staying present for many years [6].

The examination by western blot is used to eliminate the false positive results and to interpret the borderline values [12, 14,19]. In the USA the principle of „two-step protocol” has been introduced, while the first test ELISA is confirmed by the result obtained by western blot [20].

Borreliosis is the multisystem disease [3,7,12,21] occurring in 3 stages and involving dermatological, cardiological, neurological and rheumatological types [3,12].

Neuroborreliosis is the nervous form of borreliosis. It was described about 25 years ago for the first time [18]. The premature symptoms of neuroborreliosis are: cerebrospinal meningitis, PFP, other cranial nerves inflammation and radiculoneuritis [8,21]. Almost 10% of the patients with borreliosis manifest the neurological symptoms [8,22]. In Europe neuroborreliosis is diagnosed more frequently than in the USA in relation to *Borrelia garinii* neurotropism [6-8]. In the course of neuroborreliosis alterations in the cerebrospinal fluid (CSF) such as cytosis with the prevalence of mononuclear cells [6,10,23,24] and the increase of protein concentration [6,10,24] usually occur after 3 weeks of the disease [21].

Material and methods

In 2002-2004 in The Chair and Department of Developmental Neurology Poznań University of Medical Sciences Poland 24 children from the Wielkopolska region were admitted with diagnosis of borreliosis. Among all the children with borreliosis, confirmed by serologic examination, 9 (37.5%) manifested PFP out of whom 4 (16.7%) demonstrated bilateral PFP. The *Tab. 1* presents the clinical manifestations of the observed patients.

Results

The *Tab. 2* encloses clinical manifestations and dynamics of pathological symptoms in 4 patients with bilateral facial nerve paresis.

Patient I

Fifteen-year-old patient was diagnosed due to bilateral facial nerve palsy. A few months after the stay in forest areas the sudden onset of the frontal left-sided pain appeared. Consequently, progressive paresis of innervated muscles through the left facial nerve occurred which successively spread over the muscles of facial expressions starting with the forehead downward to the lower parts of the face. Unspecified general symptoms were presented in the *Tab. 2*. Oral doxycycline treatment (100 mg/day) was given during 21 days, with no clinical improvements. However, a few days after the treatment was stopped, the paresis of the right facial nerve appeared which also was initiated with the frontal region pain on the right side. Serologic examination of blood serum confirmed the diagnosis of neuroborreliosis (*Tab. 3*). The girl was treated with an intravenous antibiotic: 2g of ceftriaxone was given daily for 2 weeks. Prednison was given in decreasing doses for 25 days. During electrostimulation VII nerves procedures and Solux irradiation therapy, galantamine was given subcutaneously for 30 days. After pharmacological treatment and a series of electrostimulation slow improvement has been obtained, leading to total regression of bilateral facial palsy (*Tab. 2*). Five years before the onset of bilateral facial paresis the patient complained of the left facial nerve paralysis. No serologic examination was conducted for borreliosis. The paresis disappeared after the period of 6 weeks after the administration of antibiotic (amoxicillin with clavulonic acid), prednison, galantamine, B-vitamins and facial massage.

Patient II

Nine-year-old patient was treated in hospital due to bilateral facial palsy. The boy lives with parents in a farm located near the forests. The patient manifested the paresis of innervated muscles through the right facial nerve, preceding for about 4 weeks the occurrence of left facial nerve palsy. Unspecified systemic symptoms had appeared before the neurological signs occurred (*Tab. 2*). The boy was treated with an intravenous antibiotic: 1g of ceftriaxone was given daily for 2 weeks. Procedures of electrostimulation VII nerves and galantamine were applied for 20 days. Serologic blood plasma tests and examination of cerebrospinal fluid (CSF) confirmed the diagnosis of neuroborrelio-

Table 2. The case history and the physical examination and dynamic of pathological changes in patients with bilateral facial palsy

Case description	Patient I	Patient II	Patient III	Patient IV
Age in years	15	9	14	8
Sex	F	M	F	M
Stay in forests	+	+	+	+
Tick /medical history/	-	-	+	+
Site after tick bite	-	-	+	+
Erythema migrans	-	-	-	-
Borrelial lymphocytoma	-	-	-	-
Bony and muscular pain	+	+	+	+
Bilateral lid tremor	+	-	-	-
Headaches	+	+	+	+
Vertigo	+	-	-	-
Irritability	-	-	-	+
Fatigability, weakness	+	+	-	+
Mood depression	-	+	-	-
Dysgeusia	-	-	-	-
Low tone hypersensitivity	-	-	-	-
Facial and cervical pains of paresis site n. VII	+	-	-	-
Unspecified abdominal, nuchal, leg pain	-	-	-	-
Enlargement of cervical and submandibular lymphatic nodes	-	-	+	-
Time interval between facial nerves palsy	About 1 month	About 1 month	About 2 weeks	3 days
Start of regression of facial nerve palsy	After 14 days of therapy	After 6 days of therapy	After 14 days of therapy	After 30 days of therapy
Total regression of symptoms left side	After 2 months	After 3 weeks	After 2 months	After 2 months
Total regression of symptoms right side	After 3 months	After 6 months slight paresis	After 4 months	After 2 months

sis (Tab. 3). Control serological test, which was performed after treatment, revealed slow regression of IgM and IgG antibodies titre in blood plasma and CSF. Decreasing of specific antibodies correlated with gradual improvement of clinical condition of the patient (Tab. 2).

Patient III

Fourteen-year-old patient was admitted to hospital and treated due to bilateral facial palsy. In summer of 2002 when the girl stayed in forest areas, she noticed ticks on her body twice. It was thoroughly removed in both cases. After the period of 2 months, at the time of upper respiratory tract infection and after a stressful situation, there was a sudden onset of innervated muscle paresis through the right facial nerve, which was followed by the left PFP within 2 weeks. Before the appearance of facial nerve paresis the patient manifested a series of general symptoms presented in the Tab. 2. Serologic blood plasma test confirmed the diagnosis of borreliosis (Tab. 3). The girl was treated with an intravenous antibiotic: 2g of ceftriaxone was given daily for 3 weeks. Prednisone was given in decreasing doses for 23 days. Solux irradiation therapy was applied for 20 days. After the pharmacological treatment and physiotherapy, slow improvement of neurological condition occurred, but there appeared bilateral spasms of muscles of facial expressions, provoked by face cooling.

Patient IV

Eight-year-old patient was diagnosed of the recurrent facial nerve palsy. The left PFP occurred 5 years earlier, which after the introduction of pharmacological-rehabilitation treatment subsided. The second episode of facial palsy, right-sided this time, occurred 3 years later. The symptoms totally disappeared after the introduction of anti-inflammatory treatment. In summer 2004 the boy was bitten by a tick and few weeks later, among the unspecified general symptoms, the sudden onset of bilateral facial nerve paresis was recorded within 3 day-period (Tab. 2). Serologic tests of blood plasma and CSF confirmed the diagnosis of neuroborreliosis (Tab. 3). The child was treated with two antibiotics. Oral doxycycline treatment (100 mg/day) was given during 21 days, with no clinical improvements. Therefore intravenous ceftriaxone treatment (2g/day) was given during 22 days. Series of bilateral facial nerve electrostimulation and facial massage were applied for 20 days. Slow clinical improvement was confirmed by blood and CSF serological test (Tab. 3).

Discussion

All cases of bilateral PFP included circumstances where the tick from *Ixodes ricinus* family could have bitten our patients. These ticks are carriers of *Spirochaetes*. Only in two cases the

Table 3. Results of laboratory tests in patients with bilateral facial palsy

Type of examination	Patient I	Patient II	Patient III	Patient IV
I. Serologic test of blood serum for borreliosis				
IgM (Test IDEIA TM IgM)	0.630 (+)	1.220 (+)	1.650 (+)	3.690 (+)
IgG (Test IDEIA TM IgG)	0.110 (-)	0.590 (+/-)	0.070 (-)	0.500 (+/-)
II. Serologic test of blood serum for borreliosis				
	After 2 months	After 4 months	After 4 months	After 18 days
IgM (Test IDEIA TM IgM)	0.40 (-)	0.620 (+)	1.120 (+)	2.770 (+)
IgG (Test IDEIA TM IgG)	0.060 (-)	0.410 (+/-)	0.210 (-)	0.110 (-)
III. Serologic test of blood serum for borreliosis				
			After 14 months	
IgM (Test IDEIA TM IgM)	~~~~~	~~~~~	0.820 (+)	~~~~~
IgG (Test IDEIA TM IgG)	~~~~~	~~~~~	0.050 (-)	~~~~~
I. CSF examination				
Pleocytosis	1.3/ul	135.5/ul	1.7/ul	454/ul
Protein concentration	19 mg/dl	54 mg/dl	20 mg/dl	59 mg/dl
IgG concentration	2.48 mg/dl	~~~~~	1.75 mg/dl	9.14 mg/dl
IgG index	0.58	~~~~~	0.75	0.64
Serologic test of CSF for borreliosis				
Lyme IgG and IgM(LYT)	TV negative	~~~~~	~~~~~	~~~~~
IgM (Test IDEIA TM IgM)	~~~~~	3.570 (+)	(-)	3.560 (+)
IgG (Test IDEIA TM IgG)	~~~~~	1.810 (+)	(-)	0.780 (+)
II. CSF examination				
	~~~~~	After 2 weeks	~~~~~	After 17 days
Pleocytosis	~~~~~	50.7/ul	~~~~~	18.0/ul
Protein concentration	~~~~~	19 mg/dl	~~~~~	16 mg/dl
<b>Serologic test of CSF for borreliosis</b>				
IgM (Test IDEIA TM IgM )	~~~~~	3.360 (+)	~~~~~	3.340 (+)
IgG (Test IDEIA TM IgG)	~~~~~	1.280 (+)	~~~~~	0.080 (-)
Neuroimaging examination	MR normal	MR normal	CT normal	MR and CT normal

* CSF – cerebrospinal fluid; * MR – Magnetic Resonance; * CT – Computing Tomography

patients noticed the tick on the skin. None of the children reported dermal symptoms such as erythema migrans, which is the most frequent dermal form 85% [8], or occurring in 5% lymphocytoma [11]. The dermal manifestation is the most often diagnoses and is evaluated for 60-80% all cases of borreliosis [11]. However 1/3 of cases do not report any dermal changes, which significantly hinder the proper diagnosis [12]. Literature indicates the most frequent neurological symptoms of borreliosis such as headaches, paresthesia, PFP and radicular symptoms [25,26]. There are also reports that tension headaches are the only symptoms of neuroborreliosis [27]. Belman et al. report that 96 children manifested PFP as the most frequent neurological symptom [28]. In case of acute PFP and especially the bilateral form neuroborreliosis is the most probable diagnosis [29,30]. In our group of 24 children headaches appeared in 45.8% and PFP in 37.5 % (Tab. 1).

Before the occurrence of PFP all the children manifested unspecified systemic symptoms such as headaches, muscle and articulation pains, fatigue, weakness, excessive sleepiness and in two cases a slight mood depression. The occurrence of non-

specific symptoms in the first stage of the disease is more characteristic for adults than for children [24]. All children reported peripheral paresis of the facial nerve at one side first and after 2-4 weeks the paresis of the facial nerve on the opposite side appeared. None of the children manifested the dysgeusia or low tone hypersensitivity. In one case the above mentioned sequence was preceded by the PFP (5 years earlier), however, diagnostics for borreliosis wasn't administered. All children were tested serologically of blood serum for borreliosis. The application of serologic tests is required in all cases of borreliosis, excluding primary dermal changes [10,31]. It is not indicated to test serologically all the patients bitten by a tick [31]. At first all patients demonstrated elevated IgM antibodies and proper levels of IgG antibodies. After 2-14 months, control serological tests revealed slow reduction level of antibodies in the blood serum and CSF. In all cases the examination of CSF was administered. However, American College of Physicians in 1997 defined no need to determine the level of antibodies in CSF in patients with symptoms of borreliosis [32]. The positive results of blood serum are considered to be satisfactory [32].

However, it is advised to administer the serologic tests of CSF in case of borreliosis suspected [33,34]. Boys manifested elevated antibodies of IgM and IgG, which after two weeks of treatment indicated the tendency of slow decrease. There were not any serologic or biochemical changes of CSF detected in girls. They do not exclude the diagnosis of neuroborreliosis since they may be negative in the chronic form of the disease [35]. Two patients demonstrated significant pleocytosis in CSF test, however, neither of them manifested the meningeal symptom. It is important to remember that because of the lack of the possibility to detect treponema in standard examinations [12,21] there are index substances being searched to correlate with the inflammatory process [21]. Cytokines [21,36] and apoptosis processes [36] are considered to be characteristic because of their role in pathogenesis of borreliosis. Determination of cytokines levels may become the effective method of disease course monitoring and effectiveness of antibiotic therapy [21].

Pathophysiology of PFP in borreliosis is not satisfactorily explained [3]. It is not clearly known if the lesion of facial nerve is caused directly by the treponema invasion or the effect of immunological processes [3,36,37]. There are reports underlying the presence of infiltration changes in nuclei and roots of VII nerve [38]. It is considered that more frequent occurrence of neuroborreliosis with spread over the cranial nerves in children is correlated with the localization of the bite in the region of the neck [24,39]. Similar conclusions were reached by Eiffert et al., investigating this phenomenon in animals [39].

There are not universal courses of borreliosis treatment in the available literature. However, it is considered that ceftriaxon administered intravenously for a period of 14 days in a dose of 2 g/24 hours should be applied in neuroinfection evoked by treponema (children below 12 years of age 50-100 mg/kg/24 hours) [6,8-10,14,18,19,40-44], for 6 weeks maximum [14]. The clinical status of children started to improve after the introduction of physiotherapeutic treatment.

Frequent occurrence of borreliosis and neuroborreliosis in the population of children from Wielkopolska region is possibly correlated with unsatisfactory social consciousness in relation to the prevention of tick bites, the lack of repellents application, protective dresses during walks in forests where these Arachnoidea live. The basic steps to be undertaken while staying in forests are body protection against the direct contact with ticks [6,8,9,14,40] and application of repellents against stinging and sucking insects [6,9,14]. It is necessary to emphasize that after forest walks examination all the child's body is urgent in order to remove 'any' ticks since treponema transmission takes place within the period of 24-48 hours [6,7]. Urgent removal of the tick is the elementary method of prevention and these procedures may obviate part of Spirochaetes infection [6,8-10,14].

In the USA vaccines containing OspA antigen have been introduced [6,8-10,40]. However, difference of *Borrelia* types in Poland may result in its ineffectiveness [14].

## Conclusions

1. PFP is one of the most frequent neurological symptom of borreliosis in children. In case of acute PFP and espe-

cially the bilateral form of PFP neuroborreliosis is the most probable diagnosis.

2. All children reported PFP at one side first and after several weeks the paresis of the facial nerve on the opposite side usually appeared.
3. The clinical state of children started to improve after the introduction of physiotherapy.
4. Abatement of PFP symptoms usually lasted several months.

## References

1. Grundfast KM, Guarisco JL, Thomsen JR, Koch B. Diverse etiologies of facial paralysis in children. *Int J Pediatr Otorhinolaryngol*, 1990; 19: 223-9.
2. Cook SP, Macartney KK, Rose CD, Hunt PG, Eppes SC, Reilly JS. Lyme disease and seventh nerve paralysis in children. *Am J Otolaryngol*, 1997; 18: 320-3.
3. Vazquez M, Sparrow SS, Shapiro ED. Long-term neuropsychologic and health outcomes of children with facial nerve palsy attributable to Lyme disease. *Pediatrics*, 2003; 112: 93-7.
4. Siwula JM, Mathieu G. Acute onset of facial nerve palsy associated with Lyme disease in a 6 year-old child. *Pediatr Dent*, 2002; 24: 572-4.
5. Vorstman JA, Kuiper H. [Peripheral facial palsy in children: test for Lyme borreliosis only in the presence of other clinical signs]. *Ned Tijdschr Geneesk*, 2004; 148: 655-8.
6. Franz JK, Krause A. Lyme disease (Lyme borreliosis). *Best Pract Res Clin Rheumatol*, 2003; 17: 241-64.
7. Duszczyk E, Karney A, Kowalewska-Kantecka B, Gryglicka H. Borreliosis in children – clinical manifestation, diagnosis and treatment. *Med Wieku Rozwoj*, 2003; 7: 49-56.
8. Hengge UR, Tannapfel A, Tyring SK, Erbel R, Arendt G, Ruzicka T. Lyme borreliosis. *Lancet Infect Dis*, 2003; 3: 489-500.
9. Singh SK, Girschick HJ. Lyme borreliosis: from infection to autoimmunity. *Clin Microbiol Infect*, 2004; 10: 598-614.
10. Stanek G, Strle F. Lyme borreliosis. *Lancet*, 2003; 362: 1639-47.
11. Hercogova J. Lyme borreliosis. *Int J Dermatol*, 2001; 40: 547-50.
12. Fiegler J. Borelioza z Lyme (krętkowica kleszczowa). *Przew Lek*, 2001; 5: 86-9.
13. Hamlen R. Lyme borreliosis: perspective of a scientist-patient. *Lancet Infect Dis*, 2004; 4: 603-4.
14. Serafin M. Borelioza – choroba przenoszona przez kleszcze. *Śłużba zdrowia*, 2000; 57-60.
15. Halperin JJ. Nervous system Lyme disease. *J Neurol Sci*, 1998; 153: 182-91.
16. Stanek G, Gray J, Strle F, Wormser G. Lyme borreliosis. *Lancet Infect Dis*, 2004; 4: 197-8; discussion 198-9.
17. Oksi J, Marjamaki M, Koski K, Nikoskelainen J, Viljanen MK. Bilateral facial palsy and meningitis caused by *Borrelia* double infection. *Lancet*, 1995; 345: 1583-4.
18. Wokee JHJ, Vanneste JAL. Neuroborreliosis. *Practical Neurology*, 2004; 4: 152-61.
19. Blaauw AA, Rijpkema SG, Kuiper H, Bijlsma JW. Lyme disease: who should be tested and treated, and how? *Neth J Med*, 1997; 51: 154-62.
20. Reed KD. Laboratory testing for Lyme disease: possibilities and practicalities. *J Clin Microbiol*, 2002; 40: 319-24.
21. Kondrusik M, Swierzbinska R, Pancewicz S, Zajkowska J, Grygorczuk S, Hermanowska-Szpakowicz T. Evaluation of proinflammatory cytokine (TNF-alpha, IL-1beta, IL-6, IFN-gamma) concentrations in serum and cerebrospinal fluid of patients with neuroborreliosis. *Neurol Neurochir Pol*, 2004; 38: 265-70.
22. Duszczyk E, Kowalik-Mikolajewska B. *Borrelia burgdorferi* infection in children. *Przegl Epidemiol*, 2001; 55: 511-5.
23. Grandsaerd MJG, Meulenbroeks AA. Lyme borreliosis as a cause of facial palsy during pregnancy. *Eur J Obstet Gynecol Reprod Biol*, 2000; 91: 99-101.
24. Duszczyk E. Borelioza z Lyme. *Essentia medica*, 2004; 6: 12-6.
25. Vrethem M, Hellblom L, Widlund M, Ahl M, Danielsson O, Enerudh J, Forsberg P. Chronic symptoms are common in patients with neuroborreliosis – a questionnaire follow-up study. *Acta Neurol Scand*, 2002; 106: 205-8.

26. Dotevall L, Eliasson T, Hagberg L, Mannheimer C. Pain as presenting symptom in Lyme neuroborreliosis. *Eur J Pain*, 2003; 7: 235-9.
27. Brinck T, Hansen K, Olesen J. Headache resembling tension-type headache as the single manifestation of Lyme neuroborreliosis. *Cephalalgia*, 1993; 13: 207-9.
28. Belman AL, Iyer M, Coyle PK, Dattwyler R. Neurologic manifestations in children with North American Lyme disease. *Neurology*, 1993; 43: 2609-14.
29. Gevers G, Lemkens P. Bilateral simultaneous facial paralysis-differential diagnosis and treatment options. A case report and review of literature. *Acta Otorhinolaryngol Belg*, 2003; 57: 139-46.
30. Kilic R, Ozdek A, Felek S, Safak MA, Samim E. A case presentation of bilateral simultaneous Bell's palsy. *Am J Otolaryngol*, 2003; 24: 271-3.
31. Ramsey AH, Belongia EA, Chyou PH, Davis JP. Appropriateness of Lyme disease serologic testing. *Ann Fam Med*, 2004; 2: 341-4.
32. Guidelines for Laboratory Evaluation in the Diagnosis of Lyme Disease. *Ann Intern Med*, 1997; 127: 1106-8.
33. Kuiper H. Clinical spectrum and incidence of neuro-borreliosis in the Netherlands. *Ned Tijdschr Geneesk*, 2004; 148: 670-3.
34. Albisetti M, Schaer G, Good M, Boltshauser E, Nadal D. Diagnostic value of cerebrospinal fluid examination in children with peripheral facial palsy and suspected Lyme borreliosis. *Neurology*, 1997; 49: 817-24.
35. Marszał E, Wojaczyk-Stanek K, Krawczyk W. Neuroborelioza u 16-letniej dziewczynki. *Neur Dziec*, 1992; 1: 61-4.
36. Grygorczuk SS, Pancewicz SA, Kondrusik M, Swierzbinska R, Zajkowska JM, Hermanowska-Szpakowicz T. Apoptosis in Lyme borreliosis – a preliminary study. *Med Sci Monit*, 2003; 9: 449-55.
37. Vanzielegem B, Lemmerling M, Carton D, Achten E, Vanlangenhove P, Matthys E, Kunnen M. Lyme disease in a child presenting with bilateral facial nerve palsy: MRI findings and review of the literature. *Neuroradiology*, 1998; 40: 739-42.
38. Bertrand E, Szpak GM, Pilkowska E, Habib N, Lipczynska-Lojkowska W, Rudnicka A, Tylewska-Wierzbanowska S, Kulczycki J. Central nervous system infection caused by *Borrelia burgdorferi*. Clinicopathological correlation of three post-mortem cases. *Folia Neuropathol*, 1999; 37: 43-51.
39. Eiffert H, Karsten A, Schlott T, Ohlenbusch A, Laskawi R, Hoppert M, Christen HJ. Acute peripheral facial palsy in Lyme disease – a distal neuritis at the infection site. *Neuropediatrics*, 2004; 35: 267-73.
40. Dedeoglu F, Sundel R. Emergency Department Management of Lyme Disease. *Clin Ped Emerg Med*, 2004; 5: 54-62.
41. Kaiser R. Clinical courses of acute and chronic neuroborreliosis following treatment with ceftriaxone. *Nervenarzt*, 2004; 75: 553-7.
42. Eppes SC. Diagnosis, treatment, and prevention of Lyme disease in children. *Paediatr Drugs*, 2003; 5: 363-72.
43. Dorresteyn EM, Kouwenberg JM. Facial paresis in children; consider Lyme disease. *Ned Tijdschr Geneesk*, 2001; 145: 1013-6.
44. Hashimoto Y, Takahashi H, Kishiyama K, Sato Y, Nakao M, Miyamoto K, Iizuka H. Lyme disease with facial nerve palsy: rapid diagnosis using a nested polymerase chain reaction-restriction fragment length polymorphism analysis. *Br J Dermatol*, 1998; 138: 304-9.