

LYME NEUROBORRELIOSIS

MILOVANOVIĆ A*, MILOVANOVIĆ J*, OBRENOVIĆ SONJA**, MILOVANOVIĆ ANĐELA***,
SIMONOVIĆ P****, ČEMERIKIĆ D*, TAČEVIĆ Z*, PETRONIĆ IVANA*, GRAJIĆ M*,
KEKUŠ DIVNA***** and POPEVIĆ M*

*School of Medicine, Belgrade, Serbia; **University of Belgrade, Faculty of Veterinary Medicine, Belgrade, Serbia; ***Clinical Center of Serbia, Belgrade; ****Ministry of Health, Belgrade, Serbia; *****High Medical School, Zemun, Serbia

(Received 3rd September 2010)

Lyme borreliosis (LB) is a multisystemic zoonotic disease which in humans can involve the skin, joints, heart and/or nervous system.

In this study a total of 11 patients with clinical manifestations have been assessed at the Institute for Occupational Health. Evaluation of the patients was done in order to determine their working capability and further professional orientation. Patients were of different gender, age, education and profession. They fulfilled at least two of the three criteria: tick infestation data (epidemiological criteria), central and/or peripheral neurological symptoms (clinical criteria) and a positive serological finding.

*Diagnosis was done upon classical clinical criteria: electro-myeloneurography (EMNG) analysis, neurological impairments, electroencephalography (EEG), computer tomography (CT) and/or magnetic resonance imaging (MRI). IgM and IgG antibodies against *B. burgdorferi* were determined by commercial ELISA kits.*

IgM antibodies were recorded in the serum of 4 (44.4%) and IgG in 6 (66.7%) patients.

Electro-myeloneurography findings of the upper and lower limbs were positive in 5 (83.3%), electroencephalography in 4 (66.6%) of the 6 observed patients and CT was positive in 4 (36.4%) of the 5 observed patients.

The study has established that in patients with neuroborreliosis (NB) the capability to carry out intellectual tasks, as well as responsible duties is impaired due to poor memory. Patients suffering from peripheral neuropathies are not fit to withstand longterm walks, weight lifting and carrying or any other form of physical stress.

Key words: neuroborreliosis, ELISA, occupational capacity

INTRODUCTION

Lyme borreliosis is a multisystemic antropozoonotic disease of man and some animal species. It is characterized by a polymorph clinical picture,

unpredictable course and a tendency to relapse. A number of organs can be affected, most often the skin, joints and the nervous system (Pfister *et al.*, 1994; Stanek and Strle, 2003). In human and veterinary medicine it is a relatively new clinical entity, described for the first time in 1975 (Steer *et al.*, 1977) in Lyme (Connecticut – USA). The cause of LB is *B. burgdorferi sensu lato*, at the beginning considered to be one species, later on described as 13 species within this complex (Wang *et al.*, 1999).

Results of epidemiological and acarological studies described that *B. burgdorferi* in nature is maintained between the ticks as vectors and animals as hosts – reservoirs.

The major reservoirs, vectors and sources of *B. burgdorferi* infection are ticks of the genus *Ixodes*, i.e. in Europe *Ixodes ricinus*. The link between borreliosis and *Ixodes* ticks was confirmed by the first isolation of *B. burgdorferi* from a tick in 1981. One year later, *B. burgdorferi* was isolated from skin, liquor and human blood samples (Burgdorfer *et al.*, 1982; Steere *et al.*, 1983).

Up to date, *B. burgdorferi* has been isolated (or its presence determined) in over 40 animal species in Europe. At the same time, the presence of specific antibodies was established in a much larger number of animal species (Gern *et al.*, 1998). Birds, particularly migratory seabirds, can transport the ticks (*I. uriae*) over very long distances and thus distribute borreliae worldwide (Olsen *et al.*, 1995).

Current studies have shown that only 3 species from the *B. burgdorferi* s.l. complex are pathogenic for man and some animal species, and these are: *B. burgdorferi sensu stricto*, *B. afzelii* and *B. garinii*. All three species are registered in Europe and Serbia (Wang *et al.*, 1999; Milutinović *et al.*, 2008).

It can be considered that between species there is a certain difference in organotropism. Thus, *B. burgdorferi* s.s. is often associated with changes on the joints, *B. afzelii* causes skin lesions and *B. garinii* changes on the CNS (van Dam *et al.*, 1993).

Lyme borreliosis can manifest itself in three stages: early localized LB, disseminated early LB and disseminated late LB. Lyme borreliosis manifests as a CNS and/or peripheral nervous system disease. Clinical manifestations are not pathognomonic. Most often it develops during the second stage of LB in the form of radiculitis, neuritis, meningitis and encephalitis. The most common clinical sign of NB is meningoradiculoneuritis (Garin – Bijadoux – Bannwarth syndrome) which develops four months after infection. Late NB usually develops six months after infection. It is a rare condition and it can be in the form of chronic lymphocytic meningitis and chronic encephalomyelitis with concurrent peripheral neuropathy and acrodermatitis chronica atrophicans. In the liquor lymphocytic pleocytosis and intrathecal antibody synthesis are often present and *B. burgdorferi* can be isolated from the liquor (Kristoferitch, 1991; Strle *et al.*, 2006).

Lyme neuroborreliosis manifests itself in 10 – 15%, or even as reported 30% cases (Pachner *et al.*, 1998; Cimmino, 1998).

Diagnosis of NB can be set only according to strict diagnostic clinical criteria and laboratory tests. According to the recommendations given by the Centre for Disease Control (CDC, 1995) specific laboratory LB diagnostics implies the

detection and isolation of *B. burgdorferi* in the samples or serological testing. Isolation of *B. burgdorferi* is difficult due to the small number of bacteria present in the tissues and body fluids. Isolation is a long lasting process (from 4 to 6, sometimes even 12 weeks), thus is not considered as a routine laboratory diagnostic procedure.

In clinically suspected cases the recommendation is to prove the presence of antibodies in the blood serum, or cerebrospinal fluid (CSF) indirect immunofluorescence assay (IFA), or enzyme-linked immunosorbent assay (ELISA). In the case of a positive result, Western blot as the confirmative test is recommended. If the immunoblot is negative the reactive ELISA or IFA will probably have been a false-positive. Interpretation of serological test results must always be done in context with clinical data. In stage I (erythema migrans) only 20%-50% of patients are seropositive for IgM and/or IgG antibodies (Asbrink *et al.*, 1985; Hansen and Asbrink, 1989). IgM antibodies usually prevail. An exception might be the immune response against some primarily *in vivo* expressed antigens (Bacon *et al.*, 2003). In stage II (acute neuroborreliosis) seropositivity (IgM and/or IgG antibodies) increased to 70%-90% (Hansen *et al.*, 1988; Wilske *et al.*, 1993). In principle, patients with early manifestations may be seronegative especially in the case of short duration of symptoms. Serological follow up is recommended and in the case of neurological symptoms the CSF/serum index should be determined. Six weeks or more after the onset of symptoms, 100% of the patients with stage II neuroborreliosis were seropositive (Hansen *et al.*, 1988). In the case of late disease (stage III, acrodermatitis chronica atrophicans and arthritis) IgG antibodies are detectable in all tested patients (Hansen and Asbrink 1989; Wilske *et al.*, 1993). The presence of specific antibodies does not prove the presence of disease; a positive antibody test may also be due to clinical or subclinical infections in the past. Since IgM and IgG antibodies to *B. burgdorferi* may persist in the serum for years after clinical recovery, serology has no role in measuring the response to treatment. The more nonspecific the symptoms, the lower is the predictive value of a positive serological test. Seropositivity in the normal healthy population varies with age and increased outdoor activities (Wilske, 2003).

MATERIAL AND METHOD

Our study included 11 patients. In all patients tests toward Lyme borreliosis were conducted in regional health centers, and all were referred to hospital for further diagnosis, therapy, rehabilitation and work ability evaluation. Diagnosis was made by clinical criteria: neurological disorders, CT and/or MRI findings, and by testing for specific antibodies.

Assessment of borrelia antibodies

The IgM and IgG antibodies were measured using a commercial Lyme borreliosis ELISA kit (Dade Behring, Germany).

Magnetic resonance imaging

Patients were examined also by using a high-field magnet (1.5 Magnetom, Siemens) with T₂ and T₁ sequences (TR 2500, TE 90 and TR 600, TE 15). Gadolinium enhancement was also used. Axial, coronal and sagittal planes were imaged.

Work ability

Work ability was evaluated by standard criteria for work ability evaluation defined by occupational health regulations.

RESULTS

Our study was conducted on 3 males (27.3 %) and 8 females (72.7 %). All patients previously reported a tick infestation. The examinees belonged to the 19 to 67 years age group, with an average age of 44.09 years.

Results of the diagnostic procedures performed on patients with clinical signs of neuroborreliosis are shown in Table 1.

Table 1. Results of diagnostic procedures performed on patients with clinical signs of neuroborreliosis

Patient No	Proteins CSF	IgM serum	IgG serum	EMNG	EEG	CT
1	+	-	+	+	/	/
2	+	-	+	/	/	/
3	+	+	+	+	/	/
4	/	+	+	+	-	/
5	/	+	+	/	/	+
6	+	/	/	/	+	+
7	+	/	/	/	+	+
8	/	-	-	-	+	/
9	/	-	-	+	-	-
10	/	-	-	+	/	/
11	+	+	+	/	+	+

+ Positive; - Negative; / not determined.

The protein level in CSF was positive in all tested patients, which was 54.5 % of the total examined number. Other patients for some reason were not tested.

By immunoenzyme testing IgM and IgG antibodies were examined in the blood serum of nine patients in which due to the history of tick infestation and clinical symptoms NB was beforehand diagnosed. Two patients were not tested.

IgM antibodies were present in 4 (44.4%) and IgG in the sera of 6 (66.7%) tested patients. Four patients concurrently presented IgM and IgG antibodies and 2 presented IgG only.

Out of the 6 tested patients electromyeloneurography results of the upper and lower limbs were positive in 5 (83.3%).

Electroenceelography results were positive in 4 (66.6%) out of the 6 tested patients.

Computerized tomography results were positive in 4 (80.0%) and negative for one patient.

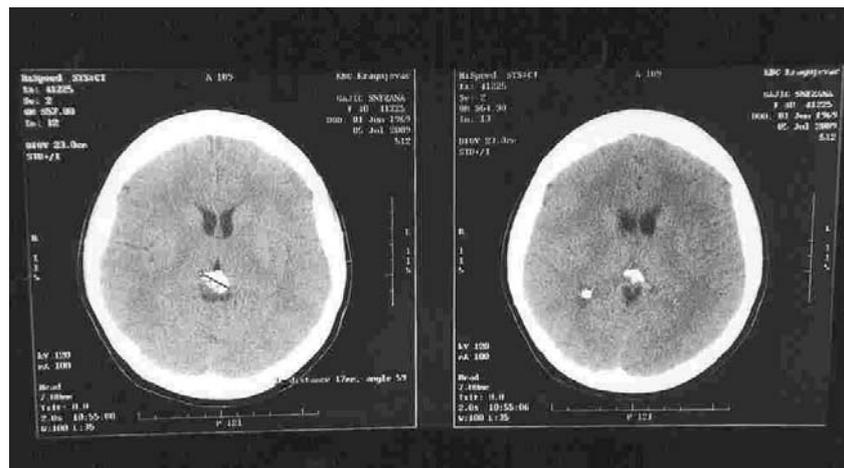


Figure 1. Calcified changes in the area of pineal regions, which is characteristic for neuroborreliosis

Based upon the performed analysis it was determined that three patients with diagnosed neuroborreliosis had positive EEG and CT findings with concurrent increase in CSF proteins. One of the three patients tested positive for serum IgM and IgG antibodies.

DISCUSSION

Diagnosis of NB is based mainly on epidemiological data and clinical diagnostic criteria, as well as on the knowledge of the possibilities of existing laboratory tests (Prasad and Sankar, 1999).

The isolation of *B. burgdorferi* on nutritive media is the best evidence of an active infection, especially in patients without distinctive clinical signs, as well as in serologically negative patients. Sadly, isolation is a very demanding and time consuming procedure and is carried out in specially equipped laboratories (Wilske, 2003).

Due to the above reasons nowadays serological tests are used for routine NB diagnosis. In patients suffering from suspected NB it is imperative to determine the presence of antibodies in the serum and CSF. This is most often achieved by ELISA. If the ELISA test results to be positive in the serum and/or CSF

a confirmation is required (due to the possibility of false positive results) by Western blot which confirms the specificity of the finding.

In a number of patients suffering from NB only intrathecal antibodies are formed. As a result the antibody index (AI) is defined. AI is given by the relationship between the quantity of antibodies present in the CSF and in the serum. Determination of CSF antibodies is crucial for those patients with only intrathecal antibody synthesis, with no serum antibodies (Buniks and Barbour, 2002).

The diagnostic value of serology depends on the humoral immune response of the infected host and of the characteristics of the applied tests (Smismans *et al.*, 2006). Serological tests in LB diagnostics are not standardized yet and they must be interpreted in the light of the available epidemiological and clinical data. The absence of antibodies in the serum or liquor can be the consequence of missing immune activation, suppression of humoral immunity, binding of antibodies into immune complexes or concurrent infections (Pachner *et al.*, 1998). False negative results during the early stages of clinical NB can be the result of a slow synthesis of antibodies, thus in order to confirm the diagnosis paired serum and liquor samples should be tested in a period of 4 to 6 weeks.

The inflammation syndrome in the CSF is more common in patients suffering from meningitis and/or encephalitis (75%) compared to cases of myelitis or radiculitis (49%) (Pal *et al.*, 1998). Patients with extracutaneous LB almost always have diagnostic serum antibodies to *B. burgdorferi* except for some patients with early seventh nerve palsy or occasional patients who have antibodies in CSF only (Tugwell *et al.*, 1997). Some authors (Oksi *et al.*, 1998) consider the presence of the inflammation syndrome in the CSF to be the compulsory criteria for the diagnosis of NB. The importance of immune mediated episodes in the diagnosis of LB shows the almost regular presence of *B. burgdorferi* immune complexes in the early stages of the disease in both seropositive and seronegative patients before treatment. Hence, after therapy in treated patients this finding is missing (Schutzer *et al.*, 1999). According to some studies in order to establish the efficiency of the treatment determination of IgG antibodies against flagellar antigens can be used (Panellus *et al.*, 1999).

Molecular methods, mainly PCR, can be used for the detection, genotyping and taxonomic classification of *B. burgdorferi*, however they are not a routine procedure for the diagnosis of NB. In unclear cases PCR can be crucial in proving the infection in serologically negative patients and in proving the success of the administered therapy, as well as in differentiating chronic LB and post-Lyme syndrome (Oksi *et al.*, 1999). This is of significance as patients suffering from NB can experience discomfort due to residual damages, immune mediated disorders, psychogenic disorders or other diseases (Pavlović, 1998). Real-time PCR based on the detection of OspA *B. burgdorferi* gene is positive in only 50% patients suffering from NB (Gooskens *et al.*, 2006).

A positive serological finding, as well as positive PCR results after a successful treatment can persist for a number of years, thus being the cause of unnecessary therapy. The success of the treatment is estimated upon achieved normal neurological status and pleocytosis in the liquor. As serum and

intrathalaeal antibodies can persist in the patient for a very long time their follow-up is irrelevant for the determination of the success of the treatment (Pfister and Rupprecht, 2006).

Brain magnetic resonance (MR) is a noteworthy diagnostic procedure, specially due to the fact that in neuroborreliosis patients it significantly correlates to the neurologic finding.

The direct action of borellia on the oligodendroglial cells can result in demyelination and possible immune mediated reactions. The distribution of demyelinated lesions is subcortical. Dot-like alterations in the white brain mass can be registered during Lyme – encephalopathy, as well as hypodense areas which correspond to vasculitis and ischemia (Fallon, 2000). In some patients with involvement of the CNS non specific changes in the white mass (Pal *et al.*, 1998).

When we analyze the work ability evaluation of patients with Lyme disease, it is important to know the degree of disease invasion, as well as if NB developed with the primary disease. If it has, usually those patients are no more capable for any kind of intellectual work, as well as for jobs in relations with moral and material responsibility, because of difficulties with memory and cognition. If the changes are on the peripheral nerves, and this was confirmed by electro-myoneurography, then they are not capable for jobs that involve long standing, long walking, picking and carrying weight, and any kind of intensive physical labor.

Although most manifestations of LB resolve spontaneously without treatment, antibiotics may hasten the resolution and prevent disease progression. In patients with arthritis, clinical recovery typically coincides with antibiotic therapy (often combined with a non-steroidal anti-inflammatory drug) (Steere *et al.*, 1994; Nocton *et al.*, 1994), as well as with physical therapy. Patients with carditis and neurological disorders also tend to do well, though some do have residual deficits such as mild seventh nerve palsy after treatment (Logigian *et al.*, 1990; van der Linde *et al.*, 1993).

Address for correspondence:

Prof. Aleksandar Milovanović, m.d., PhD
Institute of Occupational Health of Serbia, School of Medicine
Deligradska 29
11000 Belgrade
Serbia
E-mail: milalex@eunet.rs

REFERENCES

1. Asbrink E, Hovmark A, Hederstedt B, 1985, Serologic studies of erythema chronicum migrans Afzelius and acrodermatitis chronica atrophicans with indirect immunofluorescence and enzyme-linked immunosorbent assays, *Acta Derm Venereol*, 65, 509-14.
2. Bacon RM, Biggerstaff BJ, Schriefer ME, Glimore RD Jr, Philipp MT, Steere AC *et al.*, 2003, Serodiagnosis of Lyme disease by kinetic enzyme-linked immunosorbent assay using recombinant V1sE1 or peptide antigens of *Borrelia burgdorferi* compared with 2-tiered testing using whole-cell lysates, *J Infect Dis*, 187, 1187-99.
3. Bunikis J, Barbour AG, 2002, Laboratory testing for suspected Lyme disease, *Med Clin North Am*, 86, 311-40.

4. Burgdorfer W, Barbour AG, Hayes SF, Benach JL, Grunwaldt E, Davis JP, 1982, Lyme disease—a tick-borne spirochetosis, *Science*, 216, 1317-9.
5. Centers for Disease Control and Prevention, 1995, Recommendations for test performance and interpretation from the Second National Conference on Serologic Diagnosis of Lyme Disease, *Morb Mortal Wkly Rep*, 44, 590-1.
6. Cimmino MA, 1998, Relative frequency of Lyme borreliosis and of its clinical manifestations in Europe, European Community Concerted Action on Risk Assessment in Lyme borreliosis, *Infection*, 26, 298-300.
7. Fallon BA, 2000, Review of Lyme Neuroborreliosis, Medscape coverage of: 13th International Scientific Conference on Lyme Disease and other Tick-borne Disorders, Medscape Portals, Inc.
8. Gern L, Estrada-Pena A, Frandsen F, Gray J, Jaenson T, Jongejan F et al., 1998, European reservoir hosts of *Borrelia burgdorferi* sensu lato, *Zentbl Bakteriell Parasitenkd Infektkrankh*, Hyg Abt, 287, 196-204.
9. Gooskens J, Templeton KE, Claas EC, van Dam AP, 2006, Evaluation of an internally controlled real-time PCR targeting the ospA gene for detection of *Borrelia burgdorferi* sensu lato DNA in cerebrospinal fluid, *Clin Microbiol Infect*, 12, 894-900.
10. Hansen K, Asbrink E, 1989, Serodiagnosis of erythema migrans and acrodermatitis chronica atrophicans by the *Borrelia burgdorferi* flagellum enzyme-linked immunosorbent assay, *J Clin Microbiol*, 27, 545-51.
11. Hansen K, Hindersson P, Pedersen NS, 1988, Measurement of antibodies to the *Borrelia burgdorferi* flagellum improves serodiagnosis in Lyme disease, *J Clin Microbiol*, 26, 338-46.
12. Kristoferitch W, 1991, Neurological manifestations of Lyme borreliosis: Clinical definition and differential diagnosis, *Scand J Infect Dis*, 77(suppl), 64-73.
13. Loggjian EL, Kaplan RF, Steere AC, 1990, Chronic neurologic manifestations of Lyme disease, *N Engl J Med*, 323, 1438-44.
14. Milutinović M, Masuzawa T, Tomanović S, Radulović Ž, Fukui T, Okamoto Y, 2008, *Borrelia burgdorferi* sensu lato, *Anaplasma phagocytophilum*, *Francisella tularensis* and their coinfections in host-seeking *Ixodes ricinus* ticks collected in Serbia, *Exp Appl Acarol*, 45, 171-83.
15. Nocton JJ, Dressler F, Rutledge BJ, Rys PN, Persing DH, Steere AC, 1994, Detection of *Borrelia burgdorferi* by polymerase chain reaction in synovial fluid from patients with Lyme arthritis, *N Engl J Med*, 330, 229-34.
16. Oksi J, Kalimo H, Marttila RJ, Marjamaki M, Sonninen P, Nikoskelainen J et al., 1998, Intracranial aneurysms in three patients with disseminated Lyme borreliosis: cause or chance association? *J Neurol Neurosurg Psychiatry*, 64, 636-42.
17. Oksi J, Marjamaki M, Nikoskelainen J, Viljanen MK, 1999, *Borrelia burgdorferi* detected by culture and PCR in clinical relapse of disseminated Lyme borreliosis, *Ann Med*, 31, 225-32.
18. Olsen B, Duffy DC, Jaenson TG, Gylfe A, Bonnedahl J, Bergstrom S, 1995, Transhemispheric exchange of Lyme disease spirochetes by seabirds, *J Clin Microbiol*, 33, 3270-4.
19. Pachner AR, Schaefer H, Amemiya K, Cadavid D, Zhang WF, Reddy K et al., 1998, Pathogenesis of neuroborreliosis – lessons from a monkey model, *Wien Klin Wochenschr*, 110, 870-3.
20. Pal E, Barta Z, Nagy F, Wagner M, Vesceil L, 1998, Neuroborreliosis in county Baranya, Hungary, *Funct Neurol*, 13, 37-46.
21. Pfister HW, Wilske B, Weber K, 1994, Lyme borreliosis: basic science and clinical aspects, *Lancet*, 343, 1013-6.
22. Pfister HW, Rupprecht TA, 2006, Clinical aspects of neuroborreliosis and post-Lyme disease syndrome in adult patients, *Int J Med Microbiol*, 296, 11-56.
23. Prasad A, Sankar D, 1999, Overdiagnosis and overtreatment of Lyme neuroborreliosis are preventable, *Postgrad Med J*, 75, 650-6.
24. Panelius J, Seppala I, Granlund H, Nyman D, Watilberf P, 1999, Evaluation of treatment responses in late Lyme borreliosis on the basis of antibody decrease during the follow-up period, *Eur J Clin Microbiol Infect Dis*, 18, 621-9.
25. Pavlović D, 1998, Dijagnostika Lajmske neuroborelioze, *Srp Arh Celok Lek*, 126, 119-24.

26. Schutzer SE, Coyle PK, Reid P, Holland B, 1999, *Borrelia burgdorferi* – specific immune complexes in acute Lyme disease, *JAMA*, 282, 1942-6.
27. Smismans A, Goossens VJ, Nulens E, Bruggeman CA, 2006, Comparison of five different immunoassays for the detection of *Borrelia burgdorferi* IgM and IgG antibodies, *Clin Microbiol Infect*, 12, 648-55.
28. Stanek G, Strle F, 2003, Lyme borreliosis, *Lancet*, 362, 1639-47.
29. Steere AC, Malawista SE, Snyderman DR et al., 1977, Lyme arthritis: an epidemic of oligoarticular arthritis in children and adults in three Connecticut communities, *Arthritis Rheum*, 20, 7-17.
30. Steere AC, Grodzicki RL, Kornblatt AN, Craft JE, Barbour AG, Burgdorfer W et al., 1983, The spirochetal etiology of Lyme disease, *N England J Med*, 308, 733-40.
31. Steere AC, Levin RE, Molloy PJ et al., 1994, Treatment of Lyme arthritis, *Arthr Rheum*, 37, 877-88.
32. Strle F, Ruzic-Sabljic E, Cimperman J, Lotric-Furlan S, Maraspin V, 2006, Comparison of findings for patients with *Borrelia garinii* and *Borrelia afzelii* isolated from cerebrospinal fluid, *Clin Infect Dis*, 43, 704-10.
33. Tugwell P, Dennis DT, Weinstein A et al., 1997, Clinical guideline 2: laboratory evaluation in the diagnosis of Lyme disease, *Ann Intern Med*, 127, 1109-23.
34. van Dam AP, Kuiper H, Vos K, 1993, Different genospecies of *Borrelia burgdorferi* are associated with distinct clinical manifestations of Lyme borreliosis, *Clin Infect Dis*, 17, 708-17.
35. van der Linde MR, Ballmer PE, 1993, Lyme carditis. In: Weber K, Burgdorfer W, Schierz G, eds. *Aspects of Lyme borreliosis*. Berlin: Springer-Verlag, 131-45.
36. Wang G, van Dam AP, Schwartz I, Dankert J, 1999, Molecular typing of *Borrelia burgdorferi* sensu lato: taxonomic, epidemiological, and clinical implications, *Clin Microbiol Rev*, 12, 633-53.
37. Wilske B, Fingerle V, Herzer P, Hofmann A, Lehnert G, Peters H et al., 1993, Recombinant immunoblot in the serodiagnosis of Lyme borreliosis. Comparison with indirect immunofluorescence and enzyme-linked immunosorbent assay, *Med Microbiol Immunol (Berl)*, 182, 255-70.
38. Wilske B, 2003, Diagnosis of Lyme borreliosis in Europe, *Vector Borne Zoonotic Dis*, 3, 215-27.

LAJM NEUROBORELIOZA

MILOVANOVIĆ A, MILOVANOVIĆ J, OBRENOVIĆ SONJA, MILOVANOVIĆ ANĐELA,
ČEMERIKIĆ D, TAČEVIĆ Z, PETRONIĆ IVANA, SIMONOVIĆ P, GRAJIĆ M,
KEKUŠ DIVNA i POPEVIĆ M

SADRŽAJ

Lajm boreliozna je multisistemska oboljenja, iz grupe zoonoza koje kod ljudi može zahvatiti kožu, zglobove, srce i/ili nervni sistem. Istraživanjem je obuhvaćeno 11 bolesnika sa kliničkim manifestacijama neuroborelioze koji su ispitivani u Institutu za medicinu rada Srbije u cilju ocene radne sposobnosti i dalje profesionalne orijentacije. Ispitani su bolesnici različite starosti, pola, nivoa obrazovanja i različitih zanimanja koji su ispunili minimalno dva od tri kriterijuma i to: podatak o ubodu krpelja (epidemiološki kriterijum), ispoljavanje centralnih i/ili, perifernih neuroloških simptoma (klinički kriterijum) i pozitivan serološki nalaz. Dijagnoza neuroborelioze je postavljena na osnovu klasičnih kliničkih kriterijuma: neuroloških ispada, analize elektro-mioneurografije (EMNG), elektroencefalografije (EEG), kompjuterske tomografije (CT) i/ili magnetne rezonance (MRI). Ispitivanje

prisustva antitela IgM i IgG klase u krvnom serumu prema *B. burgdorferi* vršeno je komercijalnim ELISA testom. Antitela IgM klase registrovana su u serumu četiri (44,4%), dok su IgG antitela registrovana kod 6 (66,7%) ispitanih pacijenata. Nalaz elektro-mioneurografije gornjih i donjih ekstremiteta je bio pozitivan kod pet (83,3%), nalaz elektroencefalografije kod četiri (66,6%) od šest ispitanih pacijenata, dok je nalaz CT bio pozitivan kod 4 (36,4%) od pet ispitanih pacijenata. Sprovedenim ispitivanjem je utvrđeno da je kod bolesnika sa razvijenom neuroboreliozom smanjena sposobnost za bilo koju vrstu intelektualnog rada, kao i za poslove koji su povezani sa moralnom i materijalnom odgovornošću zbog problema sa pamćenjem. Kod bolesnika sa perifernim neuropatijama postoji nesposobnost za poslove koji uključuju dugotrajno stajanje i hodanje, dizanje i nošenje tereta, kao i bilo koju vrstu fizičkog rada.