LYME DISEASE (CASE REPORT)

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Abstract: *Lyme disease is a disease caused by Borrelia Burgdorferi, a spirochete which is transmitted through the bite of a tick. Unrecognized in primary stage, the disease may progress to subsequent consequences producing lesions of the skin, joints, cardiovascular system, nervous system-paralysis of cranial nerves, meningoradiculoneuritis, meningitis, chronic encephalitis, paraparesis, fibromyalgia, chronic fatigue syndrome. We present a patient with neurological impairment (neuroborreliosis) with one year evolution, treated with multiple neurological, antidepressant and anxyoltic medication, with progressively unfavourable evolution in the absence of the etiological treatment.*

Keywords: *Borrelia Burgdorferi, neurologic involvement, treatment*

Rezumat: *Boala Lyme, este o afecţiune determinată de Borellia burgdorferi, spirochetă transmisă prin muşcătura de căpuşă, care nerecunoscută ăn stadiul primar, poate evolua cu consecinţe la distanţă prin afectare tegumentară, articulară, cardiovasculară, neurologică complexă- paralizii de nervi cranieni, meningoradiculonevrite, meningite, encefalite cronice, parapareze, fibromialgie, sindrom de fatigabilitate cronică. Prezentă cazul unei paciente cu afectare neurologică (neuroborelioză), cu evoluţie de un an, etichetată şi multiplu tratată cu medicaţie neurologică şi anxiolitică, antidepresivă, cu evoluţie progresiv nefavorabilă în absenţa tratamentului etiologic.*

Cuvinte cheie: *Borelia burgdorferi-afectare neurologică-tratament*

INTRODUCTION

Lyme disease is caused by a spirochete, Borrelia, with 3 species: B.burgdorferi, garinii and afzelii, transmitted by tick bites. The container consists of mammals with bacteremia -deer, ruminants, dogs, mice or birds, which transmit the germ to the ticks *Ixodes ricinus, damini, pacificus,* rarely to the gadfly or flies. In humans, 85% of cases are caused by tick in the nymph stage; only 15% is associated with adult tick bites. Approximately 1% of bites can develop into Lyme disease, with 3 stages of evolution:

- clearance without the appearance of clinical manifestations but with positive serology
- symptoms by direct invasion in early stage, the migratory erythema with positive serology
- presence of B. burgdorferi in the tertiary stage causes an immune response responsible for symptoms related to different organs, such as arthritis induced by immunological mechanism, production of proinflammatory cytokines and intraarticular immune complexes, associated with genetic factors HLA-DR4, HLA - DR2.

In Europe, B. burgdorferi garinii is associated with neurological manifestations while B. burgdorferi afzelii is associated with skin damage - acrodermatitis chronica atrophicans.

Worldwide presence of Lyme disease is estimated at 69/100 000 cases, affecting mainly the age groups 5-9 years and 60-69 years, 76% in the white race, more frequently in the masculine gender, adult women.Age group 20-24 years is the least affected.

Clinical Lyme disease develops in 3 stages: localized, disseminated and persistent. The first two stages are the preserve of early infection, while stage three occurs in maximum one year after the onset of the infection.

In the **first stage** characteristic is chronic migratory erythema, **secondary stage** is characterized by arthritis, paralysis of cranial nerves, atrioventricular block and fatigue. In **stage three** continue arthralgias, occurring myelitis, chronic encephalitis, paraparesis, fibromyalgia.

Migratory erythema lasts 2-3 weeks and may be associated with pruritus or local inflammatory phenomena; in 20% of cases observed relapses.

Other clinical manifestations described in the primary stage: multiple skin elements similar to erythema migrans without being involved more tick bites, fever, lymphadenopathy, myalgias, flu-like clinical syndrome. After the disappearance of migratory erythema, remain asthenia and diffuse myalgias.

Intermittent **inflammatory arthritis** may involve one or many joints with migratory character at 1-2 days; the most commonly affected are knees and elbows.

Stage II installs approximately 6 months after the chronic migratory erythema and may regress without treatment within a week; relapses are possible even after 10 years. Destructive chronic arthritis may occur rarely in the absence of therapy.

Paralyses of cranial nerves are the most common neurological manifestations of Lyme disease, 50% of children having bilateral facial nerve paralysis.

Meningoradiculoneuritis (Bannwarth syndrome) is dominated by radicular pain, increased during the night; meningitis is moderate severity, with CSF pleiocytosis dominated by mononuclear.

Heart disease occurs most frequently with fever and cardiac syncope due by intermittent atrioventricular block reversible in about 7 days, rarely being required temporary pacemaker.

Chronic neuropathy is another manifestation described in the literature, which is characterized by paresthesia, radicular pain, even without sensitivity changes or motor deficit.

Meningoencephalitis is manifested by disorders of sleep and memory, moderate or severe intensity, associated with ataxia, spastic paresis and cognitive dysfunction.

Even after proper therapy of Lyme disease, some people may develop **fibromyalgia** and **chronic fatigue syndrome**.

Other issues rarely described: borrelial **lymphocytoma** of the earlobe met in the stage 2, skin atrophy known as **acrodermatitis chronica atrophicans**.

Differential diagnosis of Lyme disease is made with other aseptic meningitis, fibromyalgia, arthritis of other etiologies, systemic lupus erythematosus.

Positive diagnosis suggested by epidemiological data (yet absent in over 60% of cases) and clinical findings is confirmed by laboratory examinations: cultures of skin biopsy, serological tests, titer of IgM and IgG antibodies by ELISA, with subsequent confirmation by Western blot.(1)

In the absence of therapy patients with Lyme disease continues to produce IgM, while IgG present. In patients with neurological symptoms, intrathecal specific antibodies can be investigated.(2)

The treatment of Lyme disease not requires hospitalization of patients, excluding those with BAV and risk of cardiac syncope, neuroborreliosis.

The Infectious Diseases Society of America recently made significant changes in the treatment of Lyme disease.

An effective therapeutic measure to prevent disease is a single dose of 200 mg doxycycline in the first 72 hours after the tick bite.(3,4)

For stage I, of erythema migrans, therapy is administered orally (doxycycline for patients over 8 years, except pregnant women, 3g/day amoxicillin, erythromycin, cefuroxime) for 30 days.

For **joint manifestations, paralysis of cranial nerves** it is recommended oral therapy for 30 days; in case of persistent symptoms parenteral ceftriaxone is recommended; if the patients do not respond to this therapy may benefit from hydroxychloroquine.(5,6)

Neuroborreliosis is treated with ceftriaxone, 14 days for radicular pain or 30 days with doxycycline 200 mg/day, respectively 28 days for encephalitis, encephalopathies.(7)

Fibromyalgia, far from being resolved by antibiotics, requires administration of ceftriaxone 2g/day, 30 days, followed by administration of doxycycline 200mg/day, 60 days.

Not recommended for repeat IgM antibodies, which may persist after antibiotic administration.

We present a case of neuroborreliosis whose clinical course made difficult differential diagnosis of neurological and psychiatric disorders, with unfavourable evolution in the absence of etiologic treatment.

CASE REPORT

We present the case of a female patient aged 36 years, which was hospitalized in the Neurology Clinic Tg-Mures for a severe vertiginous syndrome onset of one year, inconstant motor deficit of the left lower limb, burning at the whole body, more pronounced in the scapulohumeral region, insomnia. The clinical, laboratory and imaging evaluations were made and the diagnosis established was conversion disorder with elements of somatization, anxious major depressive syndrome, the patient being treated with gabapentin and anxiolytics. In parallel, the Borrelia burgdorferi serology was performed, which showed IgG equivocal and IgM positive, 1,6.

The patient was known with hypothyroidism (in hormonal treatment), mastoiditis operated and was recently diagnosed with major depressive syndrome for which she received treatment with Venlafaxine, Tranxene, Gabaran.

Clinical examination revealed: general condition influenced, normally coloured skin, dry mucous membranes, adipose tissue-underrepresented, BMI 16.82 kg/m2, legs muscle pain, joint pain, difficulty walking, rhythmic cardiac sounds, HR 80/ min, BP 90/60 mmHg, tendon hyperreflexia, left plantar reflex indifferent, positive Babinski's sign in right, paresis of left crural nerve, tremor of upper limb, without clinical signs of meningeal irritation.

Paraclinic investigations were normal: WBC 7610 / mm ^{3}, Er 4.47 million/mm ^{3,} Hb 13.7 g/dl, Ht 40.9%, MCV 91.5 fl, MCH 30.6 pg, MCHC 33.5 g /dl, Platelets 189000/mm ^{3,} N61.8%,Ly 25.4%, 10.9% M10,9%, B 0.1%, E1,8%, blood glucose 87 mg / dl, urea 26 mg / dl, created 0.72 mg / dl, ESR 28 mm / h, fibrinogen 240 mg / dl, CRP 4.7 mg / l, HDL-C 50 mg / dl, GOT 34 U / l, GPT 44 U / l, amylase 60 U / L, alkaline phosphatases 64 U /L, GGT 38 U /L, cholesterol 269 mg /dl, TG 300 mg /dl, lipids 995 mg / dl, Na 138 mEq / L, K 4.4 mEq /L, Fe 98µg / dl, Ca 4.3 mEq /L, Mg 1.86 mEq / L, Ca^{2+} 2.36 mEq / L.

Brain magnetic resonance, performed native, showed small single demyelinating gap, of 5-6 mm, located paraventricular left deep in the subcortical white matter, without focal areas of edema, mass effect or blood degradation products supra- and infratentorial. Ventricular system – symmetrical, without dimensional changes. Pituitary gland and midline structures - normally positioned and configured. Inflammatory-fluid looking

area, heterogeneous, of about 19 mm located at the left mastoid and middle ear. Inflammatory mucosa in the lower part of both maxillary sinuses.

CONCLUSION

Small single demyelinating gap subcortical paraventricular in left. Left mastoiditis. Bilateral maxillary sinusitis.

VC radiograph showed rotating of the lumbar vertebral bodies with dextroscoliosis and narrowing of L5-S1 space and the **chest radiograph** showed pleural right costophrenic recess thickness.

Anamnesis, (a year and half ago the patient describing the presence of tick bites), clinical and laboratory data, positive IgM serology confirmed the diagnosis of neuroborreliosis, fibromyalgia, paresis of left crural nerve, severe depressive syndrome, conversion disorder with somatization elements.

The evolution was favourable; the patient received treatment with ceftriaxone for 30 days, corticosteroids, group B vitamins, antifungal prophylactic with improving ataxic syndrome, fibromyalgia and left crural paresis. Currently the patient is treated with doxycycline 200 mg /day with good digestive tolerance that allows the continued use for 60 days.

The case presented, far from being singular, is a warning to neuropsychiatric aspects of Lyme disease interpreted as independent diseases, with unpredictable developments in the absence of etiologic treatment, reason for which IgM and IgG serology for B burgdorferii should be made routine in areas considered endemic for Lyme disease, as is the Sibiu County.

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