



## LYME DISEASE - PECULIAR ASPECTS - CASE REPORT

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**Abstract.** Lyme disease is caused by *Borrelia burgdorferi* and it manifests as a localized infection of the skin (erythema migrans), followed by heart, joints and nervous system involvement. Seventh nerve palsy is by far the most common. The authors present the case of a 19-year-old female who developed low grade fever, aseptic meningitis and facial palsy and was admitted to the Clinical Hospital of Infectious Diseases and Pneumophysiology "Dr. Victor Babeş" Timișoara. Diagnosis of Lyme borreliosis was established by serologic testing (ELISA) that revealed high titers of IgM and IgG. Clinical outcome was good after treatment with ceftriaxone. Changes of biological features, diagnosis and differential diagnosis difficulties and peculiar aspects of evolution are presented.

In the presented case the incubation period was longer than usual, the chronic migratory erythema was absent and the patient remembered the tick bite very late. The clinical course was dominated by symptoms of upper respiratory tract infection, serous meningitis and Bell palsy. Lyme disease must be kept in mind in the differential diagnosis of aseptic meningitis and facial palsy as it can be the first and only sign of disease.

**Keywords:** aseptic meningitis, facial palsy, Lyme disease

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### Introduction

Lyme disease (borreliosis) is an emerging infectious disease caused by at least three species of bacteria belonging to the genus *Borrelia*: *Borrelia burgdorferi sensu lato* is the main cause of Lyme disease in the United States, whereas *Borrelia afzelii* and *Borrelia garinii* cause most European cases. The disease is caused by a spirochete (identified by Willy Burgdorfer only in 1982) which is transmitted through the bite of an infected adult tick or nymph (genus *Ixodes*).[1,2]

The incidence of Lyme disease in a given geographical area depends on the simultaneous presence of the pathogen, of arthropods, of different species of mammals (large and small, wild and domestic), insectivorous birds (especially for larvae and nymphs), reptiles. *Ixodes ricinus* can parasite 148 species of mammals, 149 species of birds and 20 species of reptiles. The infectious ability of the adult tick is in direct relation with the time (in hours and days) it is attached to the skin. The migration of the spirochete from the digestive tract to the salivary gland requires an interval of 1-3 days.[3]

Symptoms usually occur 2-10 weeks following infection. Lyme disease (LD) manifests itself as a localized infection of the skin (erythema migrans) (EM), followed by multiple spirochete reactions on the skin and body parts such as the heart, joints

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and nervous system. Seventh nerve palsy is by far the most common. Aseptic meningitis is relatively common, occurring in as many as 15% of untreated patients bitten by the Ixodes tick and in 30% of LD cases.[4]

#### The disease has three stages:

- the 1<sup>st</sup> stage (early localized infection) occurs in 3-32 days (mean 7 days) after the time of the tick bite and appears as a migratory erythema on any part of the body associated with general symptoms suggesting an acute infection of viral etiology (fever, chills, headache, fatigue, myalgia, photophobia, migratory arthritis, cough),
- the 2<sup>nd</sup> stage of the disease (early disseminated infection) follows marrow or lymphatic dissemination of the pathogen; the lesions of EM enlarge and multi-systemic manifestations develop, especially cardiac, neurological, musculoskeletal symptoms and signs, with a duration from several weeks to several months (Bell's palsy, meningitis, encephalitis, radiculoneuritis) and
- the 3<sup>rd</sup> stage (late persistent infection) develops after a latency period of months or years (polyneuropathy, chronic encephalomyelitis, paraplegia, cognitive impairment).[5,6,7] This division into three evolutionary stages is arbitrary, since scrolling through these sequential stages is not mandatory. The most appropriate delineation is that of early Lyme disease (localized infection, early disseminated) and late (recurrent, persistent or chronic infection for at least 1 year).[8]

LD is clinically diagnosed based on symptoms,

objective physical findings (such as EM, arthritis, serous meningitis or facial palsy), a history of possible exposure to infected ticks and serological blood tests. Serologic testing for Lyme disease is complex and timing is extremely important. Seroconversion may take several weeks in patients infected with the spirochete, so early seronegativity is to be expected.[9]

Antibiotics are the primary treatment for Lyme disease; the most appropriate antibiotic treatment depends on the patient and the stage of the disease. The antibiotics of choice are doxycycline (in adults), amoxicillin (in children), erythromycin, cefotaxime and ceftriaxone, with treatment lasting 14 to 28 days.[10]

#### Case report

A 19-year-old female student was hospitalized three times in the Clinical Hospital of Infectious Diseases and Pneumophysiology "Dr. Victor Babeş" Timișoara in 2009. She had been in good health until 11 days before the first admission, when low grade fever, chills and headache developed.

The history of the disease included a series of acute episodes followed by remissions over a period of 29 days. The acute episodes were characterized by low grade fever, chills, headache, low tolerance to effort, dysphagia, nasal obstruction, reduced appetite. Remissions with duration of 1 to 3 days were characterized by almost complete disappearance of the symptoms.

During the first clinic admission (23-26.06.2009), the physical exam revealed: poor general condition, low grade fever (37.9°C), obstruction of the nose, reduced tolerance to effort, normal skin color,

|                   | The first admission to our clinic | The second admission to our clinic | The third admission to our clinic |
|-------------------|-----------------------------------|------------------------------------|-----------------------------------|
| Hb g%             | 11.9                              | 11.5                               | 12.3                              |
| Ht %              | 36                                | 35                                 | 37                                |
| L/mm <sup>3</sup> | 4990                              | 4670                               | 9080                              |
| FL %              | N43 L47 M8 E2                     | N60 L33 M5 E2                      | N88 L11 M1 E1                     |
| ESR mm/h/2h       | 5/15                              | 5/10                               | 10/20                             |
| Fb g‰             | 3.68                              | 3.8                                | 5.9                               |
| TGP U/l           | 9                                 | 8                                  | 25                                |
| TGO U/l           | 10                                | 7                                  | 12                                |
| glucosis mg%      | 98                                | 98                                 | 123                               |
| BUN mg%           | 27                                | 25                                 | 29                                |
| Creatinine mg%    | 0.78                              | 0.66                               | 0.52                              |
| PT g%             | 7.53                              | 6.95                               | 7.24                              |
| ELFO %            |                                   |                                    | 59-4-10-8-19                      |

Table I. The biological work-up

normally colored pharynx, heart and lung auscultation was normal, liver, spleen within normal limits, without meningeal signs or symptoms. The biological work-up is shown in table I.

Frontal sinuses x-ray: normal transparency. Neurological exam: within normal limits. Ophthalmological exam: normal fundus.

Treatment with acetaminophen iv was started. The evolution was favorable, the patient presented no fever after 24 hours of hospitalization and complete remission of the symptoms. She requested to be discharged. Discharge diagnosis: upper respiratory tract viral infection.

The second admission (01-08.07.2009), on the 18<sup>th</sup> day of illness was determined by the recurrence of the headache that was localized on the right side, influenced general condition and low grade fever (37.5°C). The physical exam was normal and the biological changes are shown in table I. Although the CT of the head (30.06.2009) revealed only bilateral chronic maxillary sinusitis, lumbar puncture was done and showed clear CSF (table II). Depletion and anti-inflammatory treatment associated with group B vitamins were started. The evolution was favorable, she presented no fever after 72 hours of hospitalization, and we witnessed again the complete remission of the symptoms. She was discharged with the diagnosis of aseptic meningitis.

The third admission (14-29.07.2009), on the 32<sup>nd</sup> day of illness (transfer from Neurology) was



**Figure 1.** U.M. Bell's palsy

Reevaluation of clinical history in the context of the newly created medical data, revealed the presence of a tick bite, 43 days prior to the onset of the disease.

Immunological examination for *Borrelia burgdorferi* (ELISA) showed: IgM 65.71IU/ml (> 11 positive titer), IgG 120.73IU/ml (> 11 positive titer).

CSF: Bb IgM antibodies negative, IgG 12IU/ml (> 11 positive).

Treatment with ceftriaxone 4g/day, for 14 days, was started along with depletion, dexamethasone and group B vitamins.

The evolution was favorable, with the regression of facial palsy and of the other symptoms. The values of control lumbar puncture (24.07.2009) are presented in table II.

| CSF                   | The first lumbar puncture (2.07.2009) | The second lumbar puncture (14.07.2009) | The third lumbar puncture (24.07.2009) |
|-----------------------|---------------------------------------|---|--|
| Aspect                | clear                                 | clear                                   | clear                                  |
| Pandy                 | +                                     | ++                                      | +                                      |
| Proteins g%           | 0.92                                  | 1.12                                    | 0.26                                   |
| Glucosis mg%          | 51                                    | 55                                      | 57                                     |
| Chlorine g%           | 7.19                                  | 7.01                                    | 7.25                                   |
| Cells/mm <sup>3</sup> | 98                                    | 225                                     | 90                                     |
| Lymphocytes %         | 100                                   | 100                                     | 100                                    |
| Microbial flora       | absent                                | absent                                  | absent                                 |
| Culture               | sterile                               | sterile                                 | sterile                                |
| BK directly           | negative                              | negative                                | negative                               |

**Table II.** CSF dynamics

imposed by the appearance of right peripheral facial paresis (figure 1). The physical exam showed: influenced general condition, low grade fever (37.5°C), heart and lung auscultation were normal, without any abdominal masses, discrete nuchal rigidity. The biological changes are shown in table I.

## Discussion

This case shows that, not in as seldom situations as we may think, the disease presents with „loan“ symptoms or the patient remains „silent“ for a long period of time before „speaking“. The flu-like symp-

toms of our patient reported at the first admittance, the negative personal history, the almost normal results of the first biological work-up, as well as the evolution as an apparently self-limited disease, did not impose conducting further investigations. The diagnosis of upper respiratory tract viral infection turned out to be a „loan“ diagnosis.

Recurrence of the same symptoms just a few days later, the increasing right sided headache imposed the head CT examination and, subsequently, the lumbar puncture (in the absence of meningeal signs or symptoms). The data led to the diagnosis of aseptic meningitis. It was supported by the absence of vicinity (ear, mastoid process, sinuses, brain) or distance (endocarditis etc.) suppuration, the absence of the biological syndrome of non-specific inflammation. The serous meningitis diagnosis proved to be only an intermediary step.

The appearance of right facial paresis (on the 29<sup>th</sup> day of disease) guided the reevaluation of our diagnosis, paresis being interpreted as a „signature“ of Lyme disease, in its 2<sup>nd</sup> stage.

Many times patients are unaware of a tick bite because ticks are extremely small and their bites are often painless. Generally, enzyme-linked immunoassay (ELISA) or immunofluorescent assay (IFA) and a confirmatory Western blot are used for diagnosis.

Only at last the patient admitted the tick bite 43 days earlier (2.05.2009), before the onset of the disease. She denied the existence of skin changes or on the bite site. The immunoassay tests (ELISA) confirmed the diagnosis with a very high titer of IgG and IgM antibodies against *B.burgdorferi*.

#### **The summary of the clinical course of our patient:**

- the presence of a tick bite on 2.05.2009 in the patient's history;
- the existence of a free (asymptomatic) interval of 43 days (2.05-13.06.2009);
- the presence of flu-like symptoms, the alternations of acute episodes and remissions – expression of the period of dissemination of the disease;
- the nervous location of the infection – meningeal involvement (positive physical on the 19<sup>th</sup> day of disease) and facial nerve palsy (on the 26<sup>th</sup> day of illness);
- remission obtained after correct etiological treatment (ceftriaxone 4g per day for 14 days).

#### **Clinical and evolutional peculiarities seen in our patient:**

- a longer incubation (43 days), compared with the normal accepted 3-31 days with an average of 7-14 days;
- the absence of chronic migratory erythema, viewed as a sign of localized stage disease;
- in our patient, a flu-like onset marked the transition to the dissemination of the infection. Another peculiarity was the absence of localized or generalized enlargement of lymph nodes;
- CNS impairment was present in two instances, first as aseptic meningitis, on the 19<sup>th</sup> day of illness and second as facial paralysis 7 days later. Other organs or systems (cardiac, ocular, vascular system) were not affected. Facial paresis, known as the most common neurological symptom, can sometimes be the first and only sign of disease;
- IgG and IgM antibodies present in blood in a significant high titer, were not detected in CSF. This dissociation is supported by the following terms: the presence of antibodies in the CSF and their absence in blood indicates a neuroborreliosis, their absence in the CSF, along with their presence in the blood, does not exclude this diagnosis;
- there is no complete agreement on the timing of appearance of the specific IgM and IgG antibodies. The existing assessments vary widely between 1-3 weeks for IgM and 1-3 months for IgG to 3-4 months for IgM and 5-7 months for IgG. In our patient, antibodies' synthesis was within the first period;
- based on the statistical assessment, we note the following causal relationship between the 4 genospecies of existing borreliosis in the European area and certain clinical forms: *B. garinii* and CNS, the *B. afzelii* and chronic atrophic acrodermatitis (3<sup>rd</sup> stage sign), *B. burgdorferi* ss and Lyme arthritis or other rheumatic signs/symptoms.

#### **Conclusions**

In order to define the clinical framework of Lyme disease in our country we must know the clinical manifestations of this anthropomorphic zoonosis, which explains the need for presenting such particular cases.

Many times pathology manifests as non typical forms of disease. In certain cases, time must elapse

until the pathognomonic features of an illness appear. Although apparently not linked, the patient's clinical manifestations made sense only when the new diagnostic suspicion was established.

Facial paresis, ranked as the most common neurologic symptom of Lyme disease, can sometimes be the first and only sign of disease. If there is no other evident cause of Bell's palsy, inquiries for borreliosis must be done.

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