

A Case of Neuroborreliosis Mimicking Guillain-Barré Syndrome

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ABSTRACT

Lyme disease is a zoonosis that arises from *Borrelia burgdorferi* spp belonging to the Spirochaetales family, transmitted by Ixodes-type ticks. In the course of the disease, the heart, skin, nervous and musculoskeletal system may be affected. Central nervous system involvement, defined as neuroborreliosis, may be similar to Guillain-Barré syndrome (GBS), an immune-mediated acute neuropathy. In this article, a case that was followed up in the neurology clinic with GBS due to facial paralysis, muscle weakness and widespread muscle pain was shared. Neuroborreliosis was considered in the differential diagnosis of the patient whose clinical findings did not improve due to the presence of tick contact in history, and the diagnosis was confirmed by clinical and laboratory findings. In this case, it was emphasized that neuroborreliosis should be kept in mind in the differential diagnosis of the GBS.

INTRODUCTION

Lyme disease is a zoonosis that is transmitted by Ixodes-type ticks, and its causative pathogen is *Borrelia burgdorferi*. Musculoskeletal system, heart and central nervous system may be affected in the course of the disease.^[1]

Neurological involvement, which can be seen in 10% of Lyme disease patients, is called neuroborreliosis. Neuroborreliosis is an acute disease and often develops within a few weeks after the onset of Lyme disease. It is often observed in the form of painful meningo radiculoneuritis (Garin-Bujadoux-Bannwarth syndrome) and facial paralysis in adults.^[2,3]

Headache cranial nerve involvement, meningo radiculitis and meningismus are frequently encountered symptoms and signs in neuroborreliosis. When the disease becomes chronic, changes in consciousness, paresis, extrapyramidal symptoms, psychosis, paralysis, and vasculitis can be observed.^[4] These signs and symptoms resemble those of

the GBS, also known as acute inflammatory demyelinating polyneuropathy. In the literature, there are few cases of Lyme disease in the pediatric and adult age group that is similar to GBS.^[5,6]

According to the Centers for Disease Control and Prevention (CDC), clinical signs and symptoms and tick contact history are at the forefront in the diagnosis of Lyme disease. Laboratory tests must be used correctly and performed with verified methods. Laboratory tests are not recommended for patients without symptoms specific to Lyme disease. In the diagnosis of Lyme disease, a two-step test procedure using the same serum sample is recommended in both steps. In the first stage, enzyme immunoassay (EIA) or immunofluorescent assay (IFA) is performed, and if one of these two tests gives a positive or uncertain result, the second step is taken. The second step is the Western blot (WB) test. If signs and symptoms last shorter than 30 days, the WB test should be performed with *Borrelia burgdorferi* IgM and IgG tests,

and if they persist longer than 30 days, then only *Borrelia burgdorferi* IgG and WB tests should be performed. Results are considered positive only if EIA/IFA and WB are both positive.^[7]

Lymphocytic pleocytosis in the cerebrospinal fluid is an expected early-stage finding in the diagnosis of neuroborreliosis (CSF). *Borrelia burgdorferi* IgM and/or IgG antibodies may not be present in CSF, but CSF specific IgG should be detectable in all patients after the onset of symptoms.^[8]

Here, a patient with a diagnosis of neuroborreliosis is presented while being followed up with a preliminary diagnosis of GBS.

CASE REPORT

A sixty-one-year-old female patient had increased muscle weakness, widespread body pain for one month, and difficulty in closing his eyes for a week, and she had frequently applied to orthopedics and physical therapy outpatient clinics. In the physical examination of the patient who was admitted to the neurology clinic with these complaints showed that she was healthy with body temperature: 36 °C, TA: 130/60 mmHg, and pulse rate: 7/min.

Apart from bilateral facial paralysis and hepatomegaly, her physical examination findings were unremarkable. Some laboratory test results were as follows: WBC: 11.9 K/ μ (4800–10800), ALT: 23 U/L (N: 0–35), AST: 24 U/L (N: 0–35), CK: 178 U/L (N: 0–145), creatinine: 0.8 mg/dl (N: 0.51–0.95), CRP: 0.3 mg/dl (N: 0–3.5), sedimentation 12 mm/hour. Empirical intravenous immunoglobulin (IVIG) therapy was initiated in the neurology clinic with a preliminary diagnosis of GBS, and any pathology was not detected in cranial imaging. The results of CSF examination of the patient, whose electromyography (EMG) was compatible with early demyelinating polyneuropathy were as follows: 350 leukocytes/mm³ (80% lymphocytes), protein: 172 mg/dl, glucose: 80 mg/dl, simultaneous blood glucose: 147 mg/dl. When the anamnesis of the patient was obtained in detail, it was learned that there was a tick contact during her residence for two months in the Black Sea Region. The diagnosis was confirmed by determining *B. burgdorferi* IgM and IgG positive results in serum and CSF together with the WB test, and *B. burgdorferi* IgM and IgG positivities in CSF. Ceftriaxone (1x2 g IV) was started for the treatment of neuroborreliosis. The patient was discharged after 28 days of treatment, after regression of her facial paralysis and complete resolution of myalgia and muscle weakness. The patient's consent was obtained for this study.

DISCUSSION

In our case, the patient suffered from myalgia starting a few weeks after tick contact in the rural area, lasting for a month and accompanied by neurological symptoms in the last week. For this reason, he had applied to the physiotherapy and orthopedics outpatient clinic and received

treatment. In the literature, as in our case, it is seen that these cases frequently apply to orthopedics and physical therapy outpatient clinics due to bodily pains.^[8]

Although frequent increases in CRP and sedimentation rates are expected in Lyme disease, these values of our case were found to be within normal limits.^[8]

Neuroborreliosis may have symptomatology similar to GBS. Cases of neuroborreliosis mimicking GBS have been described in the literature. In most of these cases, there is a history of IVIG treatment before being diagnosed with Lyme disease.^[8,9]

The EMG of our case was compatible with early demyelinating polyneuropathy. Data obtained by EMG and CSF analysis are valuable in the differential diagnosis of GBS. Axonal type involvement and demyelination of motor nerves can occur in EMG. CSF findings are also important in the diagnosis of GBS, and protein concentration in CSF increases, the number of mononuclear cells is normal or decreases (<50/mm³). In the first week of the disease, CSF findings are generally within normal limits.^[10] In our case, moderately increased cell count and high protein levels were detected in CSF.

Lyme disease is responsive to tetracyclines, most of the penicillins, many of the second and third-generation cephalosporins and macrolides. Lyme disease is unresponsive to the first-generation cephalosporins, rifampicin and some fluoroquinolones. In the presence of erythema chronicum migrans, oral doxycycline, amoxicillin, cefuroxime axetil and phenoxymethylpenicillin, and as an alternative, azithromycin is being used for 14 days.^[11]

Dose and duration of antibiotherapy used depending on the stage of the disease and organ tissue involvement vary. Starting treatment at an early stage can prevent further progression of the disease.

While oral treatment regimens are often preferred in the early stages, parenteral treatments are prominent in the central nervous system, cardiac system and joint involvement.^[12] Since the central nervous system was involved in our case, the treatment was completed to 28 days with IV ceftriaxone administered at twice-daily doses of 1 g.

CONCLUSION

In our country, Lyme disease is endemic, especially in the Black Sea Region. Tick contact should be questioned in patients with findings of central nervous system involvement, and neuroborreliosis should be kept in mind in the differential diagnosis.

Informed Consent

Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

Peer-review

Internally peer-reviewed.

Authorship Contributions

Concept: Z.Ş.Ç.; Design: G.K.; Supervision: A.K.; Fundings: K.G.G.; Materials: P.Ö.; Data: A.S.Ö.; Analysis: Ş.Ç.; Literature search: S.Ö.; Writing: Z.Ş.Ç.; Critical revision: L.N.A.

Conflict of Interest

None declared.

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Guillain Barre Sendromunu Taklit Eden Bir Nöroborelyoz Olgusu

Lyme hastalığı, ixodes türü kenelerle bulaşan, Spirochaetales ailesi içinde yer alan *Borrelia burgdorferi* cinsi bakterinin neden olduğu zoonozdur. Hastalığın seyrinde kalp, deri, sinir ve kas-iskelet sistemi etkilenebilir. Nöroborelyoz olarak tanımlanan santral sinir sistemi tutulumu, immün ilişkili akut nöropati olan Guillain Barre sendromu (GBS) ile benzerlik gösterebilir. Bu yazıda fasial paralizi, kas güçsüzlüğü ve yaygın kas ağrısı nedeniyle GBS düşünülerek nöroloji kliniğinde takip edilen bir olgu paylaşıldı. Klinik takibinde bulgularında düzelme olmayan hastanın öyküsünde kene temasının olması nedeni ile ayırıcı tanıda nöroborelyoz düşünülmüş, klinik ve laboratuvar bulguları ile tanı doğrulanmıştır. Bu olguda GBS'nin ayırıcı tanısında nöroborelyozun akılda tutulması gerektiği vurgulanmıştır.

Anahtar Sözcükler: Kene; Lyme; zoonoz.