Bilateral Isolated Facial Paralysis Due to Lyme Disease

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Abstract: Lyme disease, a tick-borne infection caused by the spirochete Borrelia burgdorferi, is a multisystem disease most commonly affecting the skin, joints, nervous system, or heart. Acute neurologic abnormalities occur in 15 to 20% of the patients and neuropathy, particularly facial paralysis, lymphocytic meningitis and motor and sensory radiculoneuritis are the most common manifestations. However, facial paralysis is an objective manifestation of acute neuroborreliosis as it occurs during the early disseminated phase of the infection. The incidence of facial paralysis was reported as 4.5% but isolated facial paralysis with or without other clinical manifestations such as headache, neck pain, stiff neck or throat pain due to borreliosis is extremely rare. A previously healthy 46-year-old male developed bilateral facial nerve paralysis. The neurological examination was normal except the bilateral facial nerve palsy. Serological investigation of CSF and serum revealed positive Enzyme-linked Immunosorbent Assay (ELISA) for Ig M and Ig G antibodies to B. burgdorferi antigens. An antibiotic regimen consisting of ceftriaxone 2 g/day for six weeks was initiated. The patient was fully recovered at the end of first month with full regression of serological and CSF laboratory findings. In this current case, we report a patient with sole bilateral facial nerve paralysis due to Lyme disease.

Key words: Facial paralysis, borreliosis

INTRODUCTION

Lyme Disease (LD) is a multi system bacterial infection caused by the spirochete Borrelia burgdorferi. Nerve conduction defects (weakness/paralysis of limbs, loss of reflexes, tingling sensations of the extremities, peripheral neuropathy), severe headaches, stiff neck, meningitis, cranial nerve involvement (e.g. change in smell/taste; difficulty chewing, swallowing, or speaking; hoarseness or vocal cord problems; facial paralysis-Bell's palsy; dizziness/fainting; drooping shoulders, inability to turn head, light or sound sensitivity; change in hearing; deviation of eyeball (wandering or lazy eye), drooping eyelids), stroke are common manifestations of the disease. Facial paralysis is an objective manifestation of acute neuroborreliosis as it occurs during the early disseminated phase of the infection[1-3].

The incidence of facial paralysis was reported as 4.5% but isolated facial paralysis with or without other clinical manifestations such as headache, neck pain, stiff neck or throat pain due to borreliosis is extremely rare[4-9].

CASE

A previously healthy 46-year-old male developed bilateral facial nerve paralysis. The neurological examination was normal except the bilateral facial nerve palsy. The patient defined no systemic disease and also he was on no medications. He did not recall a tick bite, skin rash, viral type illness, or joint symptoms. He did not define nausea, vomiting, headache or fever. He had no recent weight loss or gain.

On physical examination the patient was normal except bilateral facial paralysis. Signs of bilateral facial paralysis were prominent (Fig. 1 and 2). There were no signs of meningeal irritation or skin lesions. On neurological examination the patient was fully oriented and had a fluent speech. Magnetic resonance imaging of the brain was done and evaluated normal (Fig. 3). A lumbar puncture was done and Cerebrospinal Fluid (CSF) was harvested for further investigation. The leukocyte count was 110 cells/mm³ (lymphocytes 72%, monocytes 28%), glucose 50 mg dL⁻¹ and protein 62 mg dL⁻¹. The complete blood count, liver and renal function tests and serum electrolytes, including calcium, potassium, phosphate and sodium were in normal ranges. Serological investigation of CSF and serum revealed positive Enzyme-linked Immunosorbent Assay (ELISA) for Ig M and Ig G antibodies to B. burgdorferi antigens. An antibiotic regimen consisting of ceftriaxone 2 g/day for six weeks was initiated. Neurological and laboratory examinations were repeated every week and patient’s facial paralysis

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Fig. 1 and 2: Facial paralysis was prominent in the patient recessed in 3 weeks time (Fig. 4). The patient was fully recovered at the end of first month with full regression of serological and CSF laboratory findings. Ig M reactivity at ELISA disappeared, however Ig G reactivity persisted but the response did not expand. On a follow up 8 months after the initiation of antibacterial chemotherapy, patient was fully recovered from facial palsy.

DISCUSSION

Lyme disease is a complex multisystem infection caused by *Borrelia burgdorferi* and is the most common vector-borne disease in the United States of America and Europe. Lyme disease usually begins with the pathognomonic skin lesion, erythema migrans generally appears at the site of a tick bite and in seven to ten days *B. burgdorferi* spreads hematogenously. Most of the patients skip the characteristic erythema migrans phase and this disables the early diagnosis of the disease. Thus, the skin lesions of Lyme disease show a variation from erythema migrans, lymphocytoma cutis to Acrodermatitis chronica atrophicans. However, hematogenous spread of the spirochete is the main cause of the neurologic, cardiac and rheumatologic manifestations of the disease. Systemic complaints in patients with Lyme disease are
more common in USA than in Europe, as a result of
different types of virulent genospecies. The most frequent
symptoms of Lyme disease include fatigue (54%), myalgia
(44%), arthralgia (43%), headache (42%), fever and/or
chills (39%) and stiffness of the neck (35%)\cite{9,10}. In this
case report, a Lyme disease case with isolated bilateral
facial paralysis was reported. As state above Lyme
disease has a vast variety of clinical manifestations.
However, this case has a unique clinical manifestation
because of isolated bilateral facial nerve palsy without
other clinical signs.

Neurological manifestations of Lyme disease were
seen in up to 10% of untreated patients. However, chronic
neuroborreliosis generally needs at least several weeks to
display clinical signs after the initiation of hematological
spirochete spread. The neurological manifestations of
neuroborreliosis are peripheral neuropathy (chronic
axonal neuropathy), paresthesias, radicular pain,
encephalopathy (typically subacute or chronic, subtle
memory and cognitive dysfunction) and encephalomyelitis
(unifocal or multifocal inflammatory disease)\cite{9,10}. On the other hand, in Europe the frequency of
neuroborreliosis seems higher, potentially due to the
greater neutropism of \textit{B. garinii}\cite{9}. Involvement of
cranial neuropathy or neuritis to disease may be the main
complaint of the most patients in the early stages of the
disease\cite{9}. In this case, bilateral facial paralysis was the
one and only clinical expression of the disease. The
differential diagnosis of facial nerve paralysis was the
most important issue for treatment and follow up of the
patient. In this case, Bell’s palsy, Ramsey Hunt’s
syndrome, trauma, other infectious disease and malignant
disease were other possible causes of facial nerve palsy.
Bell’s palsy was ruled out as it is generally demonstrates
unilateral facial paralysis. The Ramsey Hunt’s syndrome
due to presumably to herpes zoster of the geniculate
ganglion, consist of a facial palsy associated with a
vascular eruption in the external auditory canal, other
parts of the cranial integument and mucus membrane of
the oropharynx. Often the eight cranial nerve is affected
as well, causing vertigo and deafness. The Ramsey
Hunt’s syndrome ruled out as there was not any eruption
and other nerve involvement. Other infectious and
malignant diseases were also ruled out with normal
computed tomography and magnetic resonance
imaging\cite{9,10}.

The diagnosis of erythema migrans in locations
demic for Lyme borreliosis is purely clinical\cite{9}. Under
these conditions, laboratory testing is neither necessary
nor recommended. Culture of \textit{B. burgdorferi} from
specimens in early erythema migrans, acrodermatitis
lesions, less often from plasma and cerebrospinal fluid
enables a definitive diagnosis. In the late phase of the
disease, Polymerase Chain Reaction (PCR) technique is
very accurate to isolate spirochete from joint fluid\cite{9}.
Furthermore, monoclonal antibody staining and PCR
techniques can be used to detect DNA sequence specific
for \textit{B. burgdorferi} in clinical specimens, but this technique
cannot distinguish between live and dead organisms.
Moreover positive results may persist after clinical cure.
However in such cases like our case clinical manifestation
may vary and serological investigation was the only tool
for diagnosis\cite{9}. That’s why ELISA test and detection of
lg M and G antibodies were chosen to investigate the
\textit{B. burgdorferi} infection. These two tests may be
considered as complementary tests which are supporting
the others results. On the other hand, these tests cannot
be used for screening of the effective therapy as
antibodies against \textit{B. burgdorferi} flagella antigens
decrease in such a long period of time\cite{9}.

The pathophysiologic mechanism of infection
related facial nerve dysfunction is still uncertain.
Hypothesized mechanisms include an inflammatory
process affecting the nerve within the subarachnoid
space (the mechanism postulated for acute facial nerve
paralysis associated with other bacterial infections, such
as pyogenic meningitis and syphilis) or direct infection of
the geniculate ganglion or of the facial nerve itself, with
or without swelling of the nerve in the bony fallopian
canal (the mechanism presumed to occur with certain
viruses, such as herpes agents)\cite{9}. We couldn’t find any
pathologic finding neither in computed tomography nor
magnetic resonance imaging studies of head. The only
pathological finding in the physical examination was
bilateral facial paralysis. According to the marked
elevation of the white blood cells in the CSF, an ongoing
inflammation process could be taken into account.
Nevertheless, after the termination of the medication
and regression of the symptoms, the elevation of white blood
cell count in the CSF markedly decreased to normal
levels. This strong correlation and exact timing supports the
inflammation hypothesis.

REFERENCES
345: 115-25.
2. Hemmige, U.R., A. Tannapfel, S.K. Tyring, R. Erbel,
3. Shortland, L.I., M.A. Mastroianni, D.L. Choo,
Y. Szymko-Bennett, L.G. Dally, A.T. Pikus, K.
Sedleski and A. Marques, 2003. Audiologic
manifestations of patients with post-treatment Lyme
disease syndrome. Hear, 24: 508-517.


