Abstract

Bannwarth’s syndrome is a tick-transmitted neurological disease caused by spirochetes of the Borrelia burgdorferi group. Neurological manifestations of the disease occur after skin erythema and include: neuritic pain, lymphocytic pleocytosis without headache and sometimes cranial neuritis. We present the case of a man who complained of a neurological syndrome without evidence of tick bite and concurrent manifestation of the infection, for whom serological analysis only revealed the infection after testing repetitive specimens. We discuss the need to start early therapy when clinical manifestations are suggestive of the disease in endemic areas.

Keywords

Meningoradiculitis · Cranial nerve paralysis · Cerebrospinal fluid pleocytosis · Borrelia burgdorferi

Introduction

Bannwarth’s syndrome, commonly known as neuroborreliosis, is a neurological complication resulting from infection caused by the spirochete Borrelia burgdorferi [1]. The disease includes neuritis pain, meningoradiculitis, cranial nerve neuritis and lymphocyte pleocytosis of the cerebrospinal fluid (CSF) [2].

Neurological symptoms usually develop several weeks to months after skin lesions, known as erythema chronicum migrans (ECM), which occur at the site of the tick bite [3].

Here we report the case of a man who developed multiple cranial nerve involvement in Bannwarth’s syndrome without a history of tick bite and skin manifestations, whose serum proved positive for B. burgdorferi antigens only after testing repeated specimens.

Case report

In December 2005, a previously healthy 81-year-old clergyman began to complain of back pain subsequently involving his right leg, which had onset after lifting a weight. A few days later, the pain became more severe and was associated with a loss of sensitivity in the right leg and inability to walk, despite analgesic therapy.

Twenty days later, the patient developed a peripheral left facial palsy, followed after three more days by diplopia and ptosis.

On neurological examination the patient appeared alert, collaborative and well oriented. He was unable to walk and stand. He presented marked weakness of the right leg and was only able to bend his foot, which was reported to have diminished tactile sensitivity. Passive mobilisation of the leg evoked back pain. All deep tendon

reflexes were absent and cranial nerve examination revealed left third and seventh nerve palsy.

Laboratory blood tests indicated an increase in inflammatory indices with leukocytosis; repeated tests showed a progressive increase in values. The following tests were negative: tumour markers (AFP, NSE, CYFRA, CEA, CA19.9, PSA), complementary factor assay, circulating immunocomplex assay, organ- and non-organ-specific autoantibodies, anti-neuronal antibodies and IgM and IgG against CMV, HSV 1-2, toxoplasmosis, *B. burgdorferi* (enzyme immunoassay (EIA) commercial test, performed both on serum and CSF), and serology for mononucleosis and tick-borne encephalitis.

The patient underwent total brain and spinal magnetic resonance imaging with contrast, and thoracic-abdominal CT scan with contrast, with normal results.

Electromyography showed signs of anatomic denervation, particularly marked in L3–L4 and right L5 radicular territory, while it was unremarkable in the other muscular districts explored in the left lower extremity and upper extremities. Sensitive potentials were normal.

The patient underwent lumbar puncture about one month after onset of symptoms. Investigation of the CSF, which appeared clear and colourless, yielded protein and glucose levels of 150 mg/dl and 53 mg/dl, respectively. Tests identified 336 mononuclear cells. Isoelectrofocusing revealed damage to the blood–brain barrier and an increase to 0.75 in the IgG index, with intrathecal synthesis of IgG and CSF oligoclonal bands.

Continuation of analgesic therapy permitted complete control of the painful symptoms and physiatric therapy led to an improvement in strength deficit in the lower extremities. A skin biopsy was performed, which ruled out the presence of vasculitis.

Ten days after the first CSF test, the patient underwent a second lumbar puncture, which disclosed a reduction in cell count (178 mononuclear elements/mm³) with high protein levels (135 mg/dl). The search for HSV 1 and 2, CMV, EBV, VZV, toxoplasmosis and enterovirus in the CSF by PCR of DNA proved negative. Despite being originally negative, EIA and Western blot (WB) tests were repeated on the patient’s serum and CSF to search for anti-Borrelia

![Fig. 1a WB on CSF: IgM bands. Bands observed: p41 Fla, p39 BmpA, 18/p14, p58 (arrows). Following the manufacturer’s instructions bands of intensity equal or superior to the cut-off value are considered positive: for IgM positivity at least one of the following bands should present: p39, 25/OspC, Osp17/21, p41 (strong). At least two of the following bands presents marked intensity: p83/100, p58, p43/45, 34, 31, p30, 29, p21/22, 19, p14/18. b WB on CSF: IgG bands. Bands observed: 75, 60, p41 Fla, p39 BmpA, 18/p14 (arrows). Following the manufacturer’s instructions, bands of intensity equal or superior to the cut-off value are considered positive: for IgG positivity at least two of the following bands should present: p83/100, p38, p43/45, p39, 34, 31, p30, 29, 25/OspC, p21/22, Osp 17/21, 19, p14/18](image)