NEUROBORRELIOSIS PRESENTING AS GUILLAIN-BARRE SYNDROME : A CASE REPORT

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Introduction :

Neuroborreliosis is a tick-born disease caused mainly by the spirochetes Borrelia burgdorferi. The most common presentation in children is headache, aseptic meningitis, and unilateral facial palsy. Other neurologic manifestations include radiculitis, neuropathies, plexopathies, and transverse myelitis. In rare instances the clinical symptoms and signs present like Guillain-barré syndrome (GBS), a demyelinating radiculoneuropathy attributed to autoimmune involvement of nerve roots and peripheral nerves. A clear distinction between neuroborreliosis and GBS is sometimes difficult.

We discuss the case of a 9-year-old boy who presents symptoms of GBS with suspicion of neuroborreliosis.

Case report :

Symptoms – Clinical exam

- Acute lower limb pain with weakness
- Blurred vision
- Areflexia
- Absence of meningial signs
- Absence of cranial nerve palsies

Biological investigation

- Lyme serology : IgM at threshold value, IgG psitive (206,4 UA/ml)
- CSF analysis : WBC = 2/mm³, Total protein = 1272 mg/L, gamma-globulins = 17,6%, glucose = 57mg/dL, IgG borrelia = 35,6 UA/ml , intrathecal secretion index = 14
- Anti-ganglioside antibodies : negative

Electromyological findings :

- prolongation of distal motor conduction of the right common peroneal and right posterior tibial nerves
- Decelaration of the speed of motor conduction of the right peroneal nerve
- Elongation of F waves latences of the different examinated nerves
- Absence of Hoffmann's reflexe for the posterior tibial nerves
- Spreading of distal sensitive responses of sural nerve

Treatment :

Intravenous immunoglobulins 1g/kg/day over 2 days and Ceftriaxone 100mg/kg/day during 21 days

Evolution :

Three days after the start of the treatment the patient begun to recover. A complete resolution of his neurological deficits was observed three weeks after.

Discussion :

The first diagnosis was a GBS because of the clinical symptoms, the high level of proteins in the CSF and the electromyographic findings.

However, the Lyme serology was also positive in the blood and neuroborreliosis was confirmed by the intrathecal secretion of antibodies. Yet, there was no pleiocytosis associated which is atypical in a neuroborreliosis.

In doubt, a double treatment was started and the clinical evolution was favorable.

Thereafter, we complete the medical assessment with anti-ganglioside antibodies assay. The result was negative therefore pleading against a GBS.

Thus, the physiopathological course and the final diagnosis were unclear : neuroborreliosis leading to a GBS or neuroborreliosis with a polyradiculoneuropathy.

Only a few similar cases have been described in the literature. Some are in favour of the hypothesis that Borrelia Burgdorferi may provoke an autoimmune response causing a GBS. Other observations consider that neuroborreliosis can be atypical and mimic GBS syndrome. In our case, the mechanism is uncertain since the patient recovered after receiving the two treatments, immunoglobulins and ceftriaxone.

Conclusion :

Our case demonstrates an active Borrelia infection in the context of a typical clinical presentation of GBS.

In conclusion, this case shows that it is important to perform Lyme serology when we are in front of a patient with GBS symptoms to offer a more suitable treatment.