Limbic Encephalitis and Chronic Inflammatory Demyelinating Polyradiculoneuropathy at HIV Seroconversion Stage

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Abstract

Limbic encephalitis is an inflammatory condition that affects the central nervous system with more involvement of the limbic system anatomical regions. The Chronic Inflammatory demyelinating polyradiculoneuropathy (CIDP) is an immune-mediated inflammatory disease of the peripheral nerve. Through literature review, it seems that simultaneous involvement by these two diseases in HIV seroconversion is rare. This case study and literature review shows us how is important heterogeneous clinical presentations and early diagnosis in the presented pathologies.

Keywords: Limbic encephalitis; Chronic Inflammatory Demyelinating Polyradiculoneuropathy; HIV

Introduction

Limbic encephalitis is an inflammatory condition that affects the central nervous system with more involvement of the limbic system anatomical regions. Most pathological studies show inflammatory infiltrates also at a close distance from the limbic system, particularly when the limbic encephalitis is paraneoplastic. In such cases, a careful clinical evaluation often reveals signs and symptoms involving other areas of the nervous system (central or peripheral) that may remain discrete or become more prominent and symptomatic than the symptoms of limbic involvement [1,2].

Case Report

JV, 54, the patient followed up with medical staff by chronic diarrhea, for about three months. He had personal antecedents of secondary syphilis treated with penicillin Benzathine about 20 years since the beginning of the current frame. Made use of levofloxacin without improvement, had negative on infectious causes research (including hepatitis and HIV). Colonoscopy showed no significant changes excluding the gastroenterology hypothesis of irritable bowel syndrome. There are 30 days of entry into the Ready Relief and the patient initiated behavioral change framework and seizures associated with delusions of grandeur, personality change (more emotional) and overall weakness, initially assigned to weight loss by diarrhea frame. Suitable hospital for Neurology team. Patient neurological examination revealed vigil, disoriented in time, but walked in space. Isochoric and fotoreagents pupils, ocular motility without changes, speech and suitability for the diagnosis of limbic encephalitis within the clinical context (Figures 1 and 2). Electromyography of 4 members was conclusive of chronic inflammatory demyelinating polyradiculoneuropathy. CSF 2 cells, 98% of lymphocytes, protein 89 (normal up to 40), glycorrhachia 56 (blood glucose 90), negative cultures, PCR negative for viral causes, VDRL, cysticercosis negative immunology, negative neoplastic cell search. No clinical evidence of joint involvement, collagen diseases, polychondritis. Electroencephalogram unchanged. Realized new rapid HIV test, which resulted in positive (CD4: 426, viral load of 774), confirmed by test Western Blotting. Held pulse intravenous therapy with methylprednisolone 1 g for 5 days, the patient improved leading confirmed by test Western Blotting. Held pulse intravenous therapy with methylprednisolone 1 g for 5 days, the patient improved leading confirmed by test Western Blotting.

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The Human Immunodeficiency Virus (HIV) is an agent that can cause severe neurological damage, including limbic encephalitis and Chronic Inflammatory Demyelinating Polyradiculopathy (CIDP). The pathogenesis of HIV associated with CIDP and Guillain-Barré Syndrome (GBS) is incompletely understood. Possible mechanisms include direct HIV neurotoxicity or autoimmunity. Several features favour a possible immune mechanism. Neural histology supports an antibody-mediated process and high titres of autoantibodies to myelin sheath glycosphingolipids are found in the serum of patients. The presence of anti-ganglioside antibodies at low CD4 counts suggests that abnormal immunoregulation in HIV may precipitate a paradoxical rise in autoantibodies, resulting in neuropathies. In the initial stages, neurological weakness is rapidly progressive, involving respiratory muscles in 25% of cases. These patients may require mechanical ventilation. After a disease nadir, usually 2-4 weeks (Guillain-Barré syndrome) or 8-12 weeks (CIDP) after onset of symptoms and a variable plateau phase, recovery occurs over a period of weeks to months. Mortality varies between four and 15% and 20% remain disabled at one year despite treatment. Outcomes in HIV-positive patients are equivalent to those of HIV-negative patients. Early treatment with corticosteroids, plasmapheresis or IVIg therapy have equal efficacy in reducing the proportion of patients requiring ventilation [14-18].

**Conclusion**

Analyzing the clinical evolution of the patient and observing that it had serology HIV negative before clinical worsening and serology HIV positive after the onset of symptoms, we considered the possibility of limbic encephalitis and CIDP be associated with seroconversion phase of the HIV virus (Table 1). Due to the wide range of manifestations...
and heterogeneity of presentations of neurological involvement by HIV, sometimes within the same neurological syndrome group, it is important reports like this presented and a review of the events cited as a way to always remind neurologists, infectologists and general clinicians of this diagnostic possibility, because not always therapeutic possibilities are the same, but there is a certain consensus that the earlier identification and treatment, the better the prognosis for these patients. Also according to evolution, it should stratify the need of a therapy with high effectiveness antiretroviral penetration in the central nervous system [14,15,19,20].

References

Table 1: Neurological complications secondary to HIV seroconversion.

<table>
<thead>
<tr>
<th>Central Nervous System</th>
<th>Peripheral Nervous System</th>
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<tr>
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<td>Bell’s palsy</td>
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<td>Guillain Barré Syndrome</td>
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