Journal of AIDS & Clinical Research
ISSN: 2155-6113

de Oliveira et al., J AIDS Clin Res 2016, 7:11
DOI: 10.4172/2155-6113.1000625

Case Report Open Access

Limbic Encephalitis and Chronic Inflammatory Demyelinating Polyradiculopathy 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

JVJ, 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 foto reagents pupils, ocular motility without changes, speech and language preserved. Presented distal hypoesthesia in four members associated with tetraparesis strength grade IV in the proximal and distal muscle and osteotendinous hypoactive reflexes. No signs of meningeal irritation.

In the first evaluation showed the following clinical parameters: BP: 110/70 mmHg, HR: 86 bpm, temperature 36.1°C, Sat O_2: 95%, glucose: 113, diuresis and evacuation. During etiological investigation requested dosage of vitamin B12, folic acid, ANA, rheumatoid factor, cpk, cryoglobulins, aldolase, ldh, tgo, tgp, anti-receptor antibody acetylcholine, rheumatologic antibodies: normal; Brain MRI showed areas of hyperintensity in bilateral mesial temporal region, suggesting 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 global clinical picture initiated antiretroviral therapy with tenofovir,

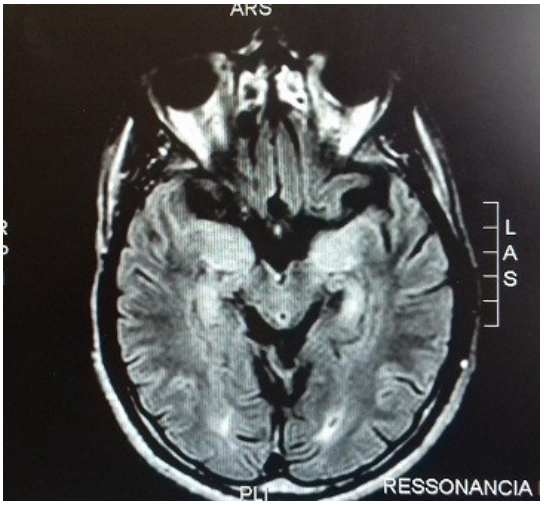

Figure 1: Sequence axial T2/FLAIR showing areas of hyperintensity in the region of bilateral mesial temporal lobe structure.

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Received September 11, 2016; **Accepted** October 05, 2016; **Published** October 12, 2016

Citation: de Oliveira FTM, Júnior FPPL, Esteves A, Modenesi CA (2016) Limbic Encephalitis and Chronic Inflammatory Demyelinating Polyradiculopathy at HIV Seroconversion Stage. J AIDS Clin Res 7: 625. doi: 10.4172/2155-6113.1000625

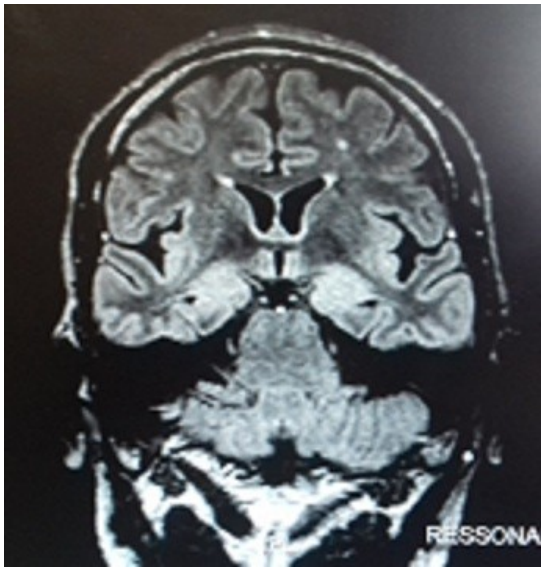

Figure 2: Sequence axial T2/FLAIR showing areas of hyperintensity in the region of bilateral mesial temporal lobe structure, notably the hippocampus.

lamivudine and efavirenz. The patient developed the neurological stability, maintaining use of corticosteroids and oral antiretroviral therapy follow-up with neurology, infectious diseases and urology.

Discussion

We observed along the sequentially clinical follow-up that a syndrome with main symptoms of limbic encephalitis, often evolve with multifocal neurological dysfunctions as encephalomyelitis, with chance of involvement, including the dorsal root ganglion, as happens in many patients with antibodies anti-Hu or with concomitant involvement with the peripheral nervous system, as in HIV infections (the case presented). In other situations, the clinical picture of limbic encephalitis is apparently masked or less evident clinically, due to the symptoms with increased cortical involvement, reducing the level of consciousness, or seizures (sometimes refractory) [1,3]. In rare cases, the simultaneous involvement of the central and peripheral nervous system (mainly acute and chronic polirradiculoneurites or polyneuropathy) occur concomitantly. In these cases, we must always remember the systemic conditions, such as metabolic and infectious diseases, and even in the face of a negative screening, remember chance of neurological manifestations during HIV seroconversion stage.

While the limbic encephalitis is an inflammatory disease of the central nervous system, 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 pathologies in HIV-infected patients is rare. The CIDP characterizes by mononuclear cell infiltrates and segmental demyelination associated with macrophage activation with both humoral and cellular components of the immune system. CIDP presents in two main ways or as an idiopathic disease or associated with changes in immune system, associated with systemic diseases such as paraproteinaemias, systemic erythematosus lupus, inflammatory bowel disease, sarcoidosis and lymphoma [4-6].

The Human Immunodeficiency Virus (HIV) is an agent that promotes disruption of the immune system and its functional impairment is mainly characterized by the inefficiency of infected CD4+ T lymphocytes. The virus is also neurotropic and neurovirulent and thus has a range of events that harm the brain, spinal cord and peripheral nerves [6]. The spectrum of HIV manifestations associated with the peripheral nervous system is quite extensive and includes distal sensory polyneuropathy, acute and chronic polyradiculoneuropathy (CIDP), plexopathies, mono neuritis (including cranial) and diffuse multiple mononeuropathies. Clinically, CIDP in HIV-infected patients seems being similar to patients without HIV [6,7].

In work done by Valcour et al. it was found that the HIV penetration in the central nervous system (CNS) occurs at an early stage during the primary infection in the periphery, as shown by findings of HIV RNA in cerebrospinal fluid, determined in up to 8 days after exposure to HIV virus and during the first phase (Fiebig I) infection. These results are consistent with those found in animal models, demonstrating that the early viral invasion of the CNS occurs in humans after exposure to mucous membranes. The inability to detect HIV RNA in 3 subjects CSF leads us to think that there must have some variability in HIV penetration time in the CNS. It can point out that there are viral and host factors influencing the early establishment of viremia in the CNS. Such findings may well prove differences of involvement with HIV in central and peripheral nervous system in different populations studied so far, as well as to the length of involvement or manifestation of the first clinical signs and symptoms [8].

Since the first reports associated with the Acquired Immunodeficiency Syndrome, various neurological manifestations of acute HIV infection reported in both central nervous system (aseptic meningitis, stroke, encephalitis by *Varicella zoster*, persistent pleocytosis, injuries Multiple-like Sclerosis acute encephalopathy and more recently limbic encephalitis) and in peripheral nervous System (Guillain-Barret syndrome - GBS, CIDP, Polymyositis, brachial plexopathy, cranial nerves neuropathy, mono neuritis multiplex, myasthenia gravis) [9-13].

The pathogenesis of HIV associated with CIDP and 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 Barret 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

Central Nervous System	Peripheral Nervous System
Aseptic meningitis	Bell´s palsy
Meningoencephalitis	Guillain Barré Syndrome
Transverse myelitis	Brachial neuritis
Limbic encephalitis	Polymyositis
Multiple Sclerosis-like	CIDP

Table 1: Neurological complications secondary to HIV seroconversion.

and heterogeneity of presentations of neurological involvement by HIV, sometimes within the same neurological syndromic 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].

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