IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 23, Issue 5 Ser. 8 (May. 2024), PP 21-23
www.iosrjournals.org

Pml Mimicking Ms Revealing Hiv Infection

Abdellaoui Walid, Louanchi Malika, Ait-Oukaci Wassila, Toubal Nadia, Sadibelouiz Mustapha, Ait-Kaci-Ahmed Mahmoud
Neurology Department, Mostaganem Hospital, University Of Mostaganem
Neurology Department, Ibn Sina Hospital, University Of Annaba
Neurophysiology Department, Ait Idir Hospital
Neurology Department, Ait Idir Hospital, University Of Algiers

Abstract:
Background*: It is an infectious demyelinating pathology of the central nervous system due to the reactivation of JC virus (present in a latent state in 80% of adults) or exceptionally other viruses (BK,SV40).It is clinically characterized by the subacute onset of variable neurological semiology. without spontaneous remission.*
PML has only been described almost exclusively in a context of deficiency of cellular immunity. (mainly in HIV positive people). The management of the latter consists of restoring the Immune function.
Clinical case report: *We report the case of a patient aged 49, single, cook, with ATCD: chronic alcoholism, glaucoma. 3 months before hospitalization: rapidly progressive cognitive disorders, focal deficit, balance disorders, aphasic disorders.*
Conclusion: *The occurrence of PML in patients with suspected MS in the absence of immunosuppressive treatment remains possible. Involvement of the U-shaped fibers should suggest the diagnosis of PML.*
Key Words*: Progressive multifocal leukoencephalopathy (PML), Multiple sclerosis (MS), HIV infection.*

Date of Submission: 11-05-2024 Date of Acceptance: 21-05-2024

I. Introduction

Progressive multifocal leukoencephalopathy (PML) is a rare but often fatal CNS pathology caused by reactivation of the JC VIRUS. The characteristics of PML have expanded considerably since the beginning of the HIV epidemic, with the monoclonal antibodies natalizumab, efalizumab and rituximab used for the treatment of multiple sclerosis, psoriasis, hematological malignancies, Crohn's disease and rheumatic diseases, have been associated with PML.

The human polyomavirus JC virus, JC virus (JCV) is well known to cause progressive multifocal leukoencephalopathy(PML) [1].

The human polyomavirus JC virus (JCV) is well known to cause progressive multifocal leukoencephalopathy (PML) [1], an often fatal brain disease resulting from lytic infection of glial cells in severely immunocompromised patients. Here we review novel features of PML and recently discovered clinical entities resulting from neuronal JCV infection, which should aid clinicians in their differential diagnosis of patients with central nervous system (CNS) disorders.

PML is a demyelinating disease of the CNS occurring in the setting of severe immunosuppression. Before the HIV era, PML remained a relatively rare disease observed in a few immunocompromised patients, including people with hematologic malignancies, organ transplant recipients, and people with chronic inflammatory diseases. The prevalence of PML increased significantly during the AIDS epidemic, where up to 5% of AIDS patients developed the disease. PML-related mortality has also increased. PML can affect patients treated with immunomodulatory drugs for autoimmune diseases, including those treated with natalizumab for multiple sclerosis and Crohn's disease [6] [7], rituximab for lupus [8] and efalizumab for psoriasis [9] .

Presenting symptoms may vary and include weakness, sensory deficit, hemianopia, cognitive dysfunction, aphasia, or impaired coordination and walking. usually the optic nerves or spinal cord are not affected. It should be noted that 18% of patients with PML may experience seizures 48 (demyelinating lesions are adjacent to the cortex).

MRI is the test of choice to diagnose PML, CT and MRI can be used. Affected brain lesions are generally detected at the white matter level and do not correspond to specific vascular territories. On CT the lesions appear hypodense, while MRI shows areas of hyperintensity on T2-weighted and FLAIR (fluid attenuated inversion recovery) images and hypointensity on T1-weighted images. Often, several lesions are present in the same patient and are frequently located in the subcortical hemispheric white matter or in the

cerebellar peduncles. Lesions can also be found in the basal ganglia or the thalamus, where the myelinated fibers reside. Classic PML lesions are devoid of edema, mass effect or contrast enhancement.

Although a cellular immune response directed against JCV is beneficial in classic PML, rapid overall recovery of the immune system is not always favorable. Indeed, this can trigger immune reconstitution inflammatory syndrome (IRIS).

Unlike classic PML, PML-IRIS lesions may show contrast enhancement on MRI, due to local inflammation and breakdown of the blood-brain barrier. This inflammation can be associated with cerebral edema, swelling, mass effect and, in the most extreme cases, cause brain involvement and death 80. Contrast enhancement in PML lesions can be detected on the MRI in HIV-positive and HIV-negative patients with IRIS.

II. Clinical Case

Patient aged 49, single, professional cook, with a history of chronic alcoholism and glaucoma, who consulted for balance problems when standing and walking, as well as rapidly progressive cognitive disorders. The symptomatology evolved for 3 months before hospitalization. On hospitalization, the neurological examination revealed oculomotor paralysis, central facial paralysis, osteotendinous reflexes abolished in all 4 limbs, fluctuating disorders of consciousness.

The brain MRI showed a T2 hypersignal and flair in the range of the sustentorial white matter, subcortically, touching the U-shaped fibers at the level of the bilateral, asymmetrical on temporoparietal regions, and subtensorial taking the middle cerebellar peduncles and the protuberance, without effect or contrast enhancement.EEG showed theta waves on temporo-parietal regions. Positive HIV serology .JC PCR in CSF not available.

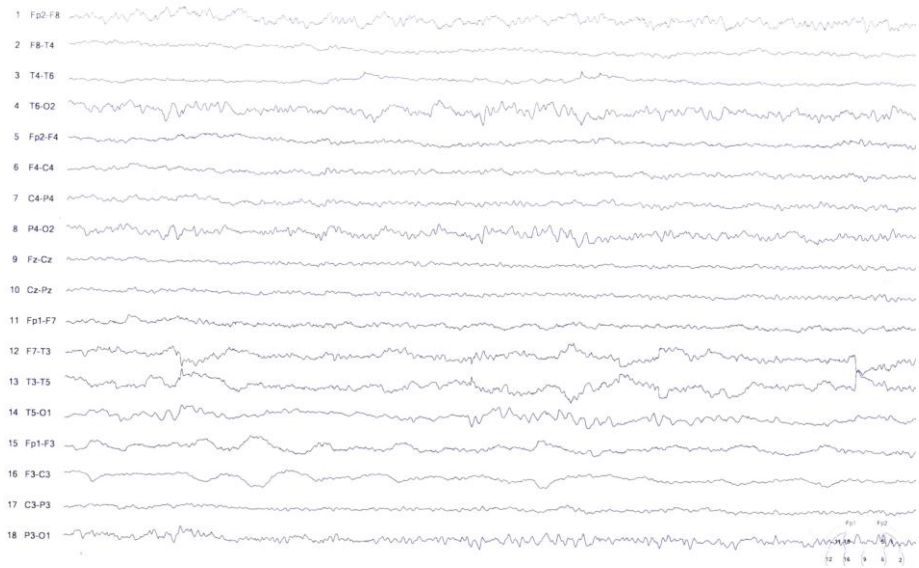

Figure 1. EEG, theta waves on temporo-parietal regions.

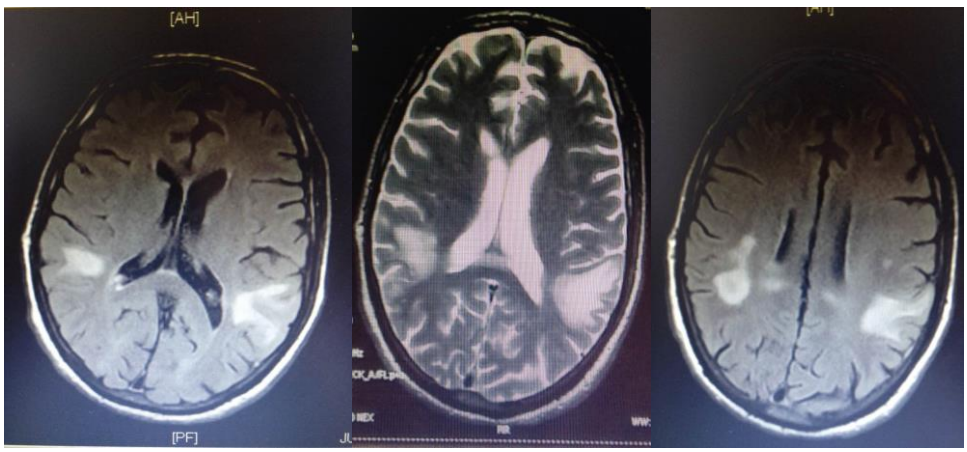

Figure 2. Brain MRI, T2 hypersignal and flair in the range of the sustentorial white matter, subcortically ,touching the U-shaped fibers at the level of the bilateral, asymmetrical on temporoparietal regions.

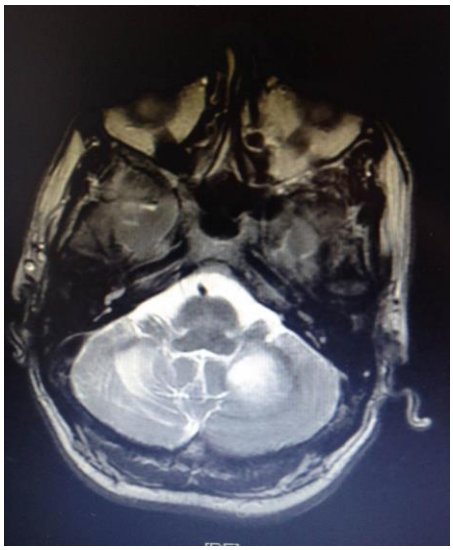

Figure 3. Brain MRI, T2 hypersignal in the range of the subtensorial white matter taking the middle cerebellar peduncles and the protuberance, without effect or contrast enhancement.

III. Discussion

The diagnosis of PML was made on the basis of the data clinical, and imaging. PML revealed HIV infection.The clinic is non-specific[10], an isolated and progressive focal neurological disorder is usually found with a good radio-clinical correlation: Hemi-deficiency: sensory > motor (upper > lower),Visual disorders (amaurosis, hemianopia), Cerebellar syndrome, Cognitive disorders. MRI is much superior to CT for diagnosis[12][13],Lesions are found: subcortical white matter, bilateral and asymmetric ,Which respect the gray matter and the marrow ,Topography: parieto-occipital -frontal -infra-tentorial. In T1++ hyposignal (because of demyelination). In T2 hyperintensity, without mass effect +++, classically without enhancement after injection of gadolinium.

Search for JC virus in CSF by PCR but positivity,inconstant (Se:80% Sp:95%°). Brain biopsy -Post mortem examination: Only diagnosis with certainty [11] [12].

IV. Conclusion

The occurrence of PML in patients with suspected MS in the absence of immunosuppressive treatment remains possible. Involvement of the U-shaped fibers should suggest the diagnosis of PML.

References

[1]. Koralnik Ij. Progressive Multifocal Leukoencephalopathy Revisited: Has The Disease Outgrown Its Name? Ann Neurol. 2006 Aug;60(2):162–73.

[2]. Van Assche G, Van Ranst M, Sciot R, Dubois B, Vermeire S, Noman M, Et Al. Progressive Multifocal Leukoencephalopathy After Natalizumab Therapy For Crohn's Disease. N Engl J Med. 2005 Jul 28;353(4):362–8.

[3]. Kleinschmidt-Demasters Bk, Tyler Kl. Progressive Multifocal Leukoencephalopathy Complicating Treatment With Natalizumab And Interferon Beta-1a For Multiple Sclerosis. N Engl J Med. 2005 Jul 28;353(4):369–74.

[4]. Du Pasquier Ra, Koralnik Ij Inflammatory Reaction In Progressive Multifocal Leukoencephalopathy: Harmful Or Beneficial? J Neurovirol. 2003;9(Suppl 1):25–31.

[5]. Huang D, Cossoy M, Li M, Choi D, Taege A, Staugaitis Sm, Et Al. Inflammatory Progressive Multifocal Leukoencephalopathy In Human Immunodeficiency Virus-Negative Patients. Ann Neurol. 2007 Jul;62(1):34–9.

[6]. Polman Ch, O'connor Pw, Havrdova E, Et Al. A Randomized, Placebo-Controlled Trial Of Natalizumab For Relapsing Multiple Sclerosis. N Engl J Med 2006;354:899–910

[7]. Ghosh S, Goldin E, Gordon Fh, Et Al. Natalizumab For Active Crohn's Disease. N Engl J Med 2003;348:24–32.

[8]. Kapoor T., Mahadeshwar P., Hui-Yuen J., Quinnies K., Tatonetti N., Gartshteyn Y., Guo C., Geraldino-Pardilla L., Askanase A.D.Prevalence Of Progressive Multifocal Leukoencephalopathy (Pml) In Adults And Children With Systemic Lupus Erythematosus. Lupus Sci. Med. 2020;7:E000388. Doi: 10.1136/Lupus-2020-000388.

[9]. Namita Kothary, Ida-Lina Diak, Allen Brinker, Shewit Bezabeh, Mark Avigan, Gerald Dal Pan. Progressive Multifocal Leukoencephalopathy Associated With Efalizumab Use In Psoriasis Patients. J Am Acad Dermatol. . 2011 Sep;65(3):546-551.

[10]. Stankoff.B, Tourbah.A, Taoufik.Y, Leucoencéphalopathie Multifocale Progressive. Emc Neurologie 2010; 17-066-A- 68.

[11]. Tan.C, Koralnik.I, Progressive Multifocal Leucoencephalopathy And Other Disorders Caused By Jc Virus: Clinical Features And Pathogenesis. Lancet Neurol 2010; 9:425-37

[12]. Gheuens S, Pierone G, Peeters P. Progressive Multifocal Leukoencephalopathy In Individuals With Minimal Or Occult Immunosuppression, J Neurol Neurosurg Psychiatry 2010; 81:247- 254.

[13]. Zaidan M, De Broucker. Leucoencéphalopathie Multifocale Progressive:Aspects Radiologiques. Rev Neurol Fmc 2009