Journal Homepage: - www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI: 10.21474/IJAR01/23199
DOI URL: http://dx.doi.org/10.21474/IJAR01/23199

RESEARCH ARTICLE

WHEN EPILEPSY REVEALS MULTIPLE SCLEROSIS: A CASE REPORT OF AN ATYPICAL PRESENTATION

Akkaoui F, Taouilr, Ferjouchia Z, Jilla M and Belfkih R

1. Department of Neurology, CHU Mohammed VI Tanger, Morocco.

Manuscript Info

Manuscript History
Received: 4 February 2026
Final Accepted: 8 March 2026
Published: April 2026

Key words:-
Multiple sclerosis; focal epilepsy; temporal lobe; cortical involvement; EEG; MRI

Abstract

Temporal lobe epilepsy is an uncommon manifestation of multiple sclerosis (MS), particularly in patients with cortical involvement. We report the case of a 24-year-old woman presenting with focal epileptic seizures evolving for one year, characterized by behavioral arrest, head and eye deviation, and automatisms, with suspected nocturnal generaliz ed seizures. Interictal neurological examination was normal.Electroenc ephalography revealed a left temporal epileptogenic focus. Brain MRI demonstrated white matter lesions fulfilling the McDonald criteria, along with left temporal cortical and juxtacortical lesions.Cerebrospinal fluid analysis showed a type 2 oligoclonal band pattern with intrathecal IgG synthesis, while extensive etiological investigations were unremark able.Despite treatment with carbamazepine, seizure control remained incomplete. Fingolimod therapy was initiated, leading to a marked reduction in seizure frequency after three months. This case highlights the importance of considering demyelinating disease in young patients with focal epilepsy, particularly in the presence of cortical lesions, and suggests a potential role of inflammation in epileptogenesis.

Introduction:-

Multiple sclerosis (MS) is a chronic inflammatory disease of the central nervous system characterized by demyelination disseminated in time and space. Its clinical manifestations are heterogeneous, most commonly including motor, sensory, and visual deficits.Epilepsy is a less frequent neurological manifestation of MS, although its prevalence is higher than in the general population. It is more commonly observed in patients with cortical or juxtacortical lesions, suggesting a potential role of cortical involvement in epileptogenesis. However, the relationship between MS and epilepsy remains incompletely understood, and the causal link between demyelinating lesions and seizures is not clearly established. In some cases, epilepsy may represent the initial manifestation of the disease, particularly in young patients with cortical or juxtacortical brain lesions.We report the case of a young woman with left temporal lobe epilepsy revealing asymptomatic MS, with strong clinico-electro-radiological correlation and favorable evolution under disease-modifying therapy.

Corresponding Author:-Akkaoui F.
Address: -Department of Neurology, CHU MohammedVI Tanger, Morocco.

Case Presentation :-

A 24-year-old woman with no prior medical history presented with focal epileptic seizures evolving for approximately one year, initially treated with carbamazepine with partial response.

Seizure semiology included:-

- behavioral arrest with staring,
- head and eye deviation to the right,
- stereotyped automatisms consisting of right-hand rubbing,
- episodes lasting a few minutes with spontaneous recovery,
- nocturnal events suggestive of bilateral tonic-clonic seizures, suspected due to postictal urinary incontinence.

Interictal neurological examination was strictly normal.Electroencephalography revealed a left temporal epileptogenic focus.Brain magnetic resonance imaging demonstrated white matter lesions as well as left temporal cortical and juxtacortical lesions, fulfilling the McDonald criteria for multiple sclerosis.Cerebrospinal fluid analysis showed a type 2 immunological profile with intrathecal IgG synthesis.An extensive etiological workup excluded infectious, tumoral, and alternative autoimmune causes.The diagnosis of multiple sclerosis associated with left temporal lobe epilepsy was established.

Management and Outcome:-

The patient was initially maintained on antiseizure medication (carbamazepine), with persistent seizures.

A disease-modifying therapy with fingolimod (Gilenya) was initiated.After three months of treatment, a significant reduction in seizure frequency was observed compared to the period under antiseizure therapy alone.No clinical relapses of MS were reported during follow-up, and neurological examination remained normal.

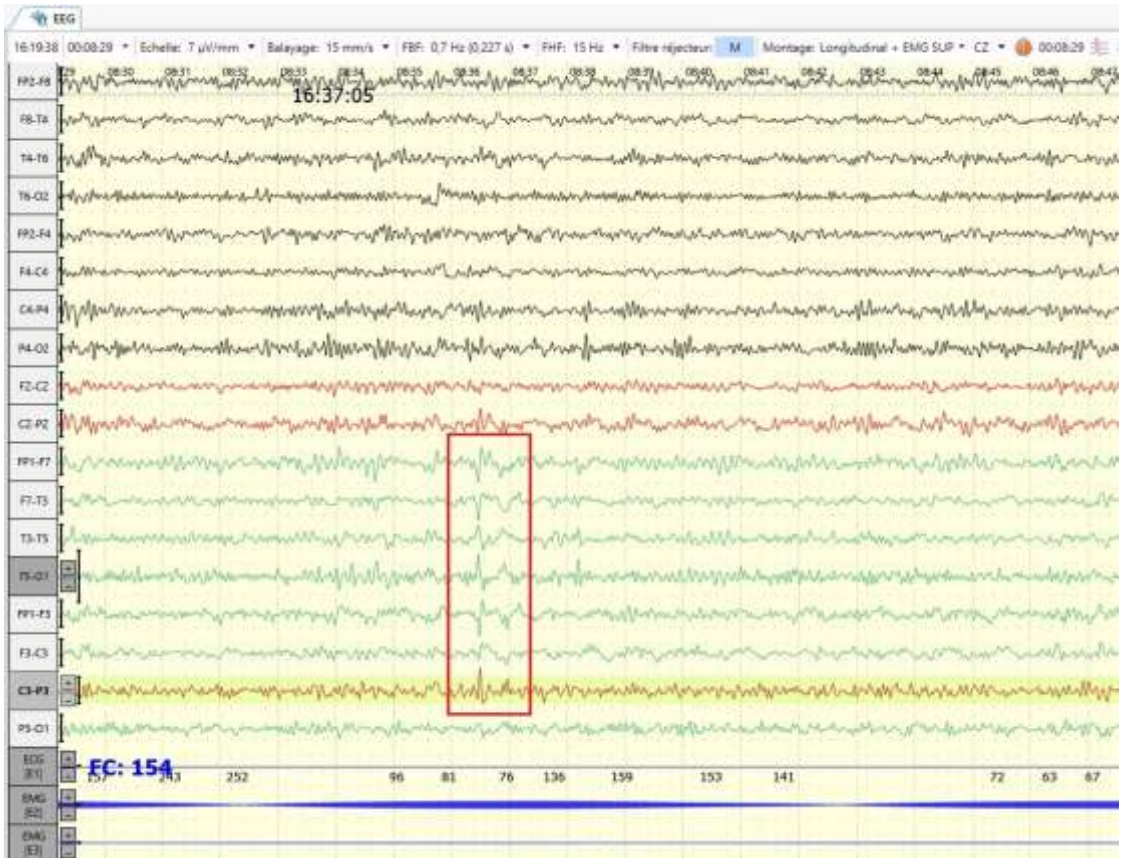

Figure 1.Interictal EEG showing left temporal epileptiform discharges with phase reversal and spread to adjacent regions

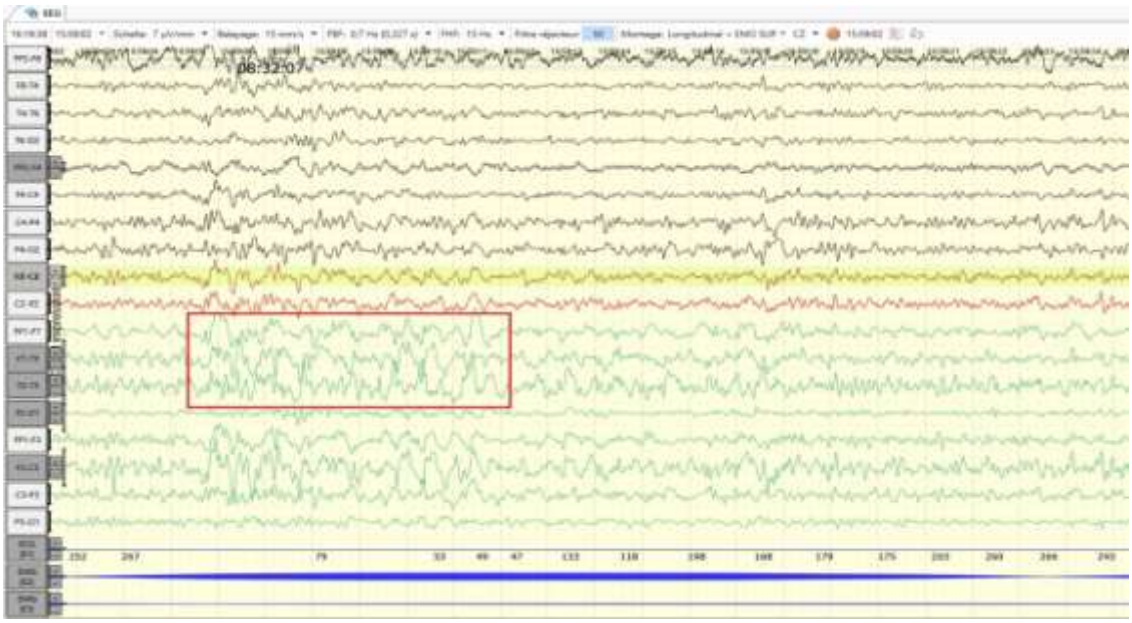

Figure 2. EEG demonstrating spike-and-wave discharges over the left temporal region.

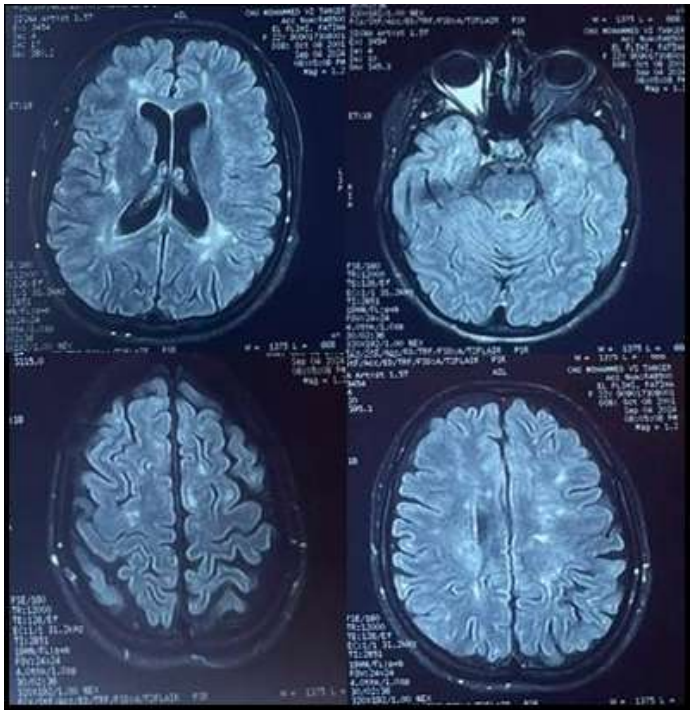

Figure 3.Brain MRI (T2/FLAIR) demonstrating white matter lesions consistent with McDonald criteria and a left temporal cortical lesion.

Discussion:-

The association between multiple sclerosis (MS) and epilepsy is increasingly recognized, with a higher prevalence of seizures compared to the general population. However, epilepsy remains a relatively uncommon manifestation of MS and is more frequently observed in patients with cortical or juxtacortical lesions [1–4].In the present case, several findings support a causal relationship between epilepsy and MS. The patient presented with left temporal lobe epilepsy, confirmed by EEG, in clear concordance with left temporal cortical and juxtacortical lesions

identified on MRI. This clinico-electro-radiological correlation strongly suggests an epileptogenic substrate related to cortical demyelinating involvement [5–7].

The pathophysiological mechanisms underlying epileptogenesis in MS remain incompletely understood. Proposed mechanisms include cortical inflammation, demyelination leading to altered neuronal conduction, and synaptic reorganization promoting neuronal hyperexcitability [2,6]. Cortical lesions, particularly in the temporal lobe, may therefore act as epileptogenic foci in certain MS phenotypes [6,7].Another notable aspect of this case is the asymptomatic nature of MS, with no identified clinical relapses and a normal neurological examination outside of seizures. This highlights that MS may be revealed by atypical manifestations such as epilepsy, particularly in young patients [3].Furthermore, the marked reduction in seizure frequency following fingolimod initiation is noteworthy. Although the exact mechanism remains unclear, it may be related to reduced central nervous system inflammation and immune modulation, suggesting a potential indirect effect of disease-modifying therapies on neuronal excitability [8].Finally, this case emphasizes the importance of a thorough etiological workup in young patients presenting with focal epilepsy, especially when cortical or juxtacortical MRI abnormalities are present.

Conclusion:-

This case illustrates an unusual presentation of multiple sclerosis revealed by left temporal lobe epilepsy associated with cortical and juxtacortical lesions. It highlights the importance of considering demyelinating disease in the etiological workup of focal epilepsy in young patients, even in the absence of interictal neurological deficits. The observed clinico-electro-radiological concordance supports a potential role of cortical lesions in MS-related epileptogenesis. Finally, the improvement in seizure frequency under fingolimod suggests a possible impact of immunomodulatory therapies on neuronal excitability, warranting further investigation.

References:-

1. Giovannoni G, Et Al.Multiple Sclerosis. Lancet. 2012;379(9822):1942–1954.Doi:10.1016/S0140-6736(12)60273-9
2. Holtkamp M. Epilepsy In Multiple Sclerosis: Clinical Features, Pathophysiology, And Treatment. Epilepsy Behav.2013.
3. Magyari M, Et Al. Frequency Of Epilepsy In Multiple Sclerosis: A Population-Based Study. Neurology. 2020.
4. Gasparini S, Et Al. Epilepsy Associated With Multiple Sclerosis: Clinical Characteristics And Prognosis. Epilepsia.2017.
5. Siva A, Et Al. Cortical Lesions In Multiple Sclerosis: Clinical Relevance And Imaging. Mult Scler. 2014.
6. Calabrese M, Et Al. Cortical Lesions And Epilepsy In Multiple Sclerosis. Brain. 2010.
7. Koch M, Et Al. Seizures In Multiple Sclerosis: Relationship With Cortical Lesions. Neurology. 2008.
8. Kappos L, Et Al. Fingolimod In Relapsing Multiple Sclerosis (Freedoms Study). N Engl J Med. 2010.