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Manic Episode in an Acute Disseminated Encephalomyelitis

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Abstract **Case Report**

Acute disseminated encephalomyelitis (ADEM) is a demyelinating inflammatory disease. Corticosteroids are its main treatment. This case report presents a manic episode after taking corticosteroids during the treatment of ADEM. A 23 years old woman,that presented two month ago an episode of 40°C fever, fatigue and headache. The radiology imaging concluded to an ADEM. After taking corticosteroid she presented a manic episode which needed decreasing in corticosteroids, and the introduction of Olanzapine and Lamotrigine with a good evolution.

Keywords: ADEM, corticoids, manic episode.

I- INTRODUCTION

Acute disseminated encephalomyelitis (ADEM), is an acute, rapidly developing, auto-immune inflammatory process that occurs in the central nervous system. It has neurological and psychiatric signs and its treatment is based on corticosteroids.

Corticosteroids, along with their anti-inflammatory, antalgic and immunosuppressive properties, are known to have side effects that affect several systems, including the neuropsychic system.

The objective of our work is to illustrate by a clinical vignette the occurrence of psychiatric signs in a patient diagnosed with ADEM without a psychiatric history (in particular bipolarity), and who presented a manic episode during corticosteroid treatment.

II- CLINICAL VIGNETTE

This is a 23-year-old woman, married, with no history of mood disorders, and who presented 2 months ago, a fever at 40 C, asthenia and headache, which led to hospitalization in medical structure, symptomatic treatment was given, the patient improved after 25 days, without a diagnosis of etiology.

Then, the evolution was marked by the reappearance of asthenia, headache, drowsiness, psycho-motor slowdown, isolation and sad mood.

The clinical examination did not found neurological abnormalities, and the blood test were also normal.

The CSF (cerebrospinal fluid) was clear, a glycorrachy at 0.71g/l (blood glucose 1.04 g/l), protein 0.19g/l, leukocytes: 4/mm3, hematites: 1/mm3, and germs were absent.

The radiological exploration was in favor of an ADEM according to the images of the brain MRI (brain magnetic resonance imaging): bilateral lenticulo-caudal ranges, symmetrical, in hyposignal T1, hypersignal T2 flair, discreetly raised after injection of Gadolinium. An MRI of the control brain after a week showed the same lesions.

The conduct of the treating neurologist consisted of the prescription of injectable corticosteroids and then relayed via the oral route. the evolution was marked by improvement, especially asthenia and headaches.

Ten days after the start of the degression of corticotherapy, the patient presented a table made of agitation, insomnia, irritability, euphoria, obscenity, logorrhea, with an attempt to defenergize in a playful setting.

When the patient was taken to the psychiatric emergency, we introduced olanzapine, lamotrigine,

lorazepam and levopromazine, with a rapid release of corticosteroids.

In an effort to minimize the risk of teratogenicity in a patient of reproductive age, lamotrigine was used as a mood stabilizer.

III- DISCUSSION

1- Generalities

Acute disseminated encephalomyelitis (ADEM) is an acute, rapidly evolving autoimmune process that occurs in the central nervous system. ADEM is characterized by demyelination of the brain and spinal cord. Its annual impact is estimated at 1 in 125,000 to 250,000 people.

The disease occurs more frequently in men than in women (male/female ratio of 1.3/1), and more often seasonally in winter and spring.

The risk factors for developing ADEM are genetic factors, clear skin pigmentation, exposure to infectious agents or recent vaccination: in 50% to 75% of cases, ADEM is associated with a previous infection or vaccination and the majority of cases follow a viral or bacterial infection that may be up to 60 days old [1].

It has a sudden start with a rapidly progressive progression, and is characterized by multifocal neurological symptoms requiring early hospitalization [1].

The clinical picture may include fever, headache, fatigue, discomfort, nausea and vomiting, but these are non-specific symptoms.

In about 20% to 52% of adult cases, there is an altered mental state (encephalopathy) that may be associated with irritability, confusion, psychosis, drowsiness, or even coma.

Motor and sensory deficits may be present, such as paraparesis, tetraparesis. Patients may also have brain stem deficits such as dysarthria or oculomoter dysfunction, or other neurological abnormalities such as convulsions, meningism, ataxia, aphasia, nystagmus, and even increased intracranial pressure or extrapyramidal signs [1].

Brain MRI is the imaging of choice for scanning ADEM, showing hyper-intense lesions in T2. Lesions are usually not viewed on T1-weighted sequences, and larger lesions may appear as hypodensities [1].

In 50-80% of patients with ADEM, CSF abnormalities are found, these abnormalities may include lymphocytic pleocytosis and a slightly elevated CSF protein (less than 70 mg/dL). More specifically, it is often observed that patients with ADEM have high levels of basic myelin protein in cerebrospinal fluid (CSF) [1].

The performance of the EEG may show a sleep disturbance, and a focal or generalised slowdown of electrical activity [1].

2- Psychiatric Symptoms

Work by Diaz-Olavarrieta *et al.*, [2] evaluated psychiatric signs in a group of patients with ADEM or multiple sclerosis. The researchers found that 95% of the patients had psychiatric manifestations. Depression was present in 79% of patients, agitation in 40%, anxiety in 37%, irritability in 35%, apathy in 20%, euphoria in 13%, disinhibition in 13%, and hallucinations in 10% [2].

3- Treatment of ADEM

In case of meningeal signs, fever, acute encephalopathy and signs of inflammation in the blood or CSF, an empirical treatment based on acyclovir is introduced.

The basic treatment of ADEM is based on high-dose injectable glucocorticoides for immunosuppressive purposes.

In the case of non-response or insufficient response to glocucorticoides intravenous immunoglobulin, cyclophosphamide or plasma exchange may be used [1].

4- Psychiatric Side Effects of Coerticosteroids

The psychic side effects of corticosteroids are defined by the onset within eight weeks of the introduction or increase of corticosteroids and resolution of symptoms after reduction of corticosteroids without the addition of an immunosuppressive agent [3].

Rome and Braceland [4] defined four levels of psychiatric response to corticosteroid treatment:
Level 1: feeling of psychic well-being with better concentration and clarity of thought.
Level 2: insomnia, increased motor activity. Both levels involved 60% of patients.
Level 3: severe anxiety and thymic variations affects 25-30% of patients.
Level 4: concerns 5% of patients, with wide variations in mood and delusions.

A cohort of 372696 patients taking corticosteroids, followed in general medicine in Great Britain over 18 years [5] : this study showed that these patients have a 7 times higher risk of suicidal behaviour or suicide compared to the general population.

5- Risk Factors for Cortico-Induced Psychiatric Disorders

a- The Dose

The dose of corticosteroids is the most important risk factor: dose-dependent effects [6].

The Boston Collaborative Drug Surveillance Program reports a positive correlation between corticosteroid doses and incidence of disorders: an incidence of 1.3% at 40 mg/d (prednisone) and an incidence of 18.4% at doses > 80 mg/d. [7]. Psychiatric disorders generally occur in the first few weeks of corticosterotherapy [5].

b- Background of Psychiatric Disorders (Not Corticosteroid Induced)

According to reliable studies, the presence of a psychiatric history does not against indicate the use of corticosteroids, however, some works has concluded that corticosteroids should not be given to patients with a history of mood disorders [8].

Barrimi *et al*, found a significant relationship between the presence of psychiatric history and the occurrence of cortical-induced psychic disorders [6].

Authors who advocate the use of corticosteroids even if they have a psychiatric history do so with low dosages [3,4].

c- Background of Psychiatric Disorders Corticosteroid Induced

The risk of developing a psychiatric disorder is greater if there is ATCD of cortico-induced disorder in the past. The prescription of corticosteroids under these conditions must be carried out with vigilance and with a lower dosage [5]. This notion is controversial, because according to other authors, the presence of cortico-induced psychiatric history does not predispose to other similar episodes and therefore does not counter-indicate the use of corticosteroids by this patient profile [9].

d- Gender

Ling *et al*, report female predominance in cortico-induced disorders [10]. Likely due to female predominance in system diseases, which often require treatment with corticosteroid therapy. According to the study by Fardet *et al*, women may be at greater risk of suffering from cortico-induced depressive episodes and that men have more manic episodes [5].

e- Organic Diseases

Certain pathologies, such as systemic lupus erythematosus, can cause damage to the central nervous system, making it difficult to differentiate between the psychiatric disorders caused by the disease or by its treatment [3].

The lesions of the blood-meningeal barrier in some diseases of the system could expose to the risk of developing cortico-induced psychiatric disorders [3].

6- The Treatment of A Cortico-Induced Manic Episode

The key element of treatment is the gradual decrease in the dose or cessation of corticosteroids. If this is not possible because of the underlying disease or the risk of secondary adrenal insufficiency, the combination of a psychotropic drug seems to be necessary [6].

Studies have reported the efficacy of olanzapine in cortico-induced manic episodes [3].

The glucocorticoid-induced Mania appears to respond to lithium and sodium valproate. They rapidly reduce induced manic symptoms and allow continued treatment with glucocorticoids, but at lower doses, if discontinuation of glucocorticoids is not possible [6].

Isolated cases of response to risperidone, quetiapine, carbamazepine, gabapentin, or clonazepam are also reported. [3, 11].

IV- CONCLUSION

The occurrence of thymic signs during ADEM is described in the literature; the same symptoms may emerge after taking corticosteroids. The difficulty lies in the distinction between psychiatric tables due to this disease and those induced by corticosteroids, especially in new patients with no psychiatric history, particularly bipolar disorders.

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