Acta Neurol Belg (2012) 112:409–411
DOI 10.1007/s13760-012-0089-y

LETTER TO THE EDITOR

The relevance of electroencephalogram as a follow-up test in Hashimoto encephalopathy course after corticosteroids therapy

José Luiz Pedroso · Marcos Knobel · Orlando G. Barsottini · Luis Otávio Caboclo · Elias Knobel

Received: 26 February 2012 / Accepted: 15 May 2012 / Published online: 5 June 2012

Introduction

Hashimoto encephalopathy (HE) has been recognized as a syndrome of encephalopathy, and high serum levels of antithyroid antibody concentrations that are responsive to corticosteroid therapy [1]. Few studies have demonstrated neurophysiological improvement after treatment with corticosteroids in HE patients. Herein, we describe a patient diagnosed with HE with clear improvement in electroencephalogram (EEG) aspects after high doses of corticosteroids.

Case report

A 88-year-old woman presented to our hospital with 3-week history of progressive mental confusion and memory impairment. Neurological examination showed disorientation, cognitive impairment and memory dysfunction. She quickly developed lowering of consciousness. All general blood exams and serologic tests were normal. Brain magnetic resonance imaging (MRI) disclosed general atrophy. Cerebrospinal fluid (CSF) demonstrated normal cell count and moderate high levels of proteins: 76 mg/dl (normal range less than 35 mg/dl). Tireoglobulin antibody level was elevated (843 IU/ml) and thyroperoxidase antibody was normal. Other immunologic tests and levels of general antibodies resulted normal. The first EEG recording, while the patient was drowsy, disclosed diffuse slowing of background rhythms and very frequent triphasic waves, more marked over anterior regions and presenting periodic character (Fig. 1). Diagnosis of HE seemed likely. Methylprednisolone 500 mg daily for three consecutive days was started, and the patient presented a clear cognitive improvement. An EEG performed 7 days after the first exam, after treatment with intravenous steroids, showed a significant improvement of the background slowing and disappearance of triphasic waves, which correlated with clinical improvement (Fig. 2). At this moment, the patient was awake, and there was a remarkable improvement in disorientation, cognitive impairment and memory dysfunction. The lack of evidence for exogenous poisoning or general metabolic diseases, the normal cell count in CSF making the diagnosis of encephalitis unlikely, and clear neurological improvement after corticosteroids therapy allowed us to perform the diagnosis of HE.

Discussion

The term HE or steroid responsive encephalopathy associated with autoimmune thyroiditis (SREAT) is used by medical community to describe a syndrome with variable cerebral symptoms in patients with high serum levels of antithyroid antibody concentrations [1]. The clinical spectrum of HE is variable and two subtypes are described: a vasculitis type, characterized by multiple stroke-like episodes, and a diffuse progressive type, characterized by dementia and psychiatrics symptoms. Both forms may be accompanied by stupor or coma, tremor, seizures, or myoclonus [1]. The diagnosis is based on the following

J. L. Pedroso (✉) · O. G. Barsottini · L. O. Caboclo
Department of Neurology, Universidade Federal de São Paulo, Rua Botucatu, 740, São Paulo, SP 04.023-900, Brazil
e-mail: zeluizpedroso@yahoo.com.br

J. L. Pedroso · M. Knobel · O. G. Barsottini · L. O. Caboclo · E. Knobel
Hospital Israelita Albert Einstein (HIAE), São Paulo, Brazil

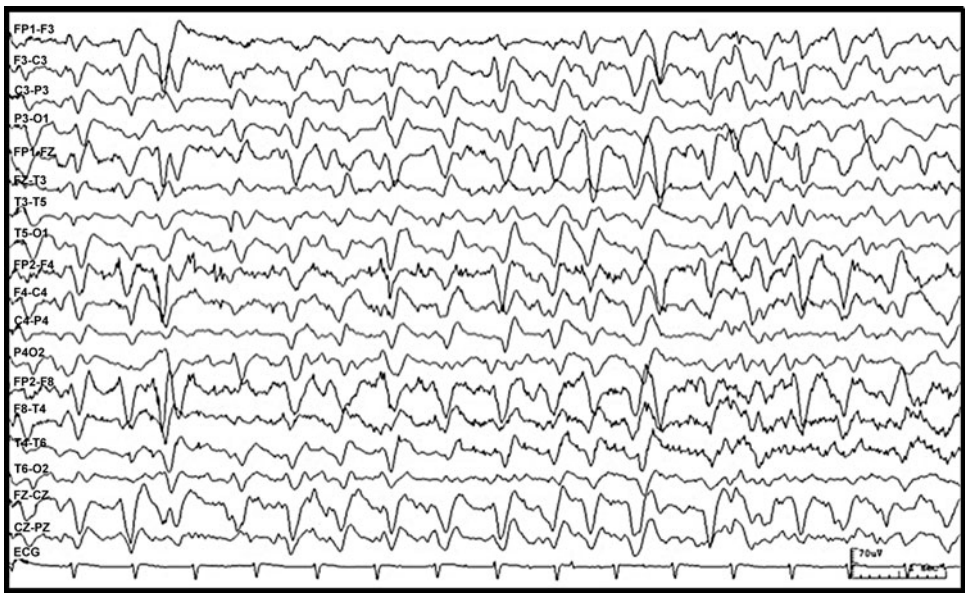

Fig. 1 Wake electroencephalogram recording showing diffuse slowing of background rhythms and very frequent triphasic waves, marked over anterior regions and presenting periodic character

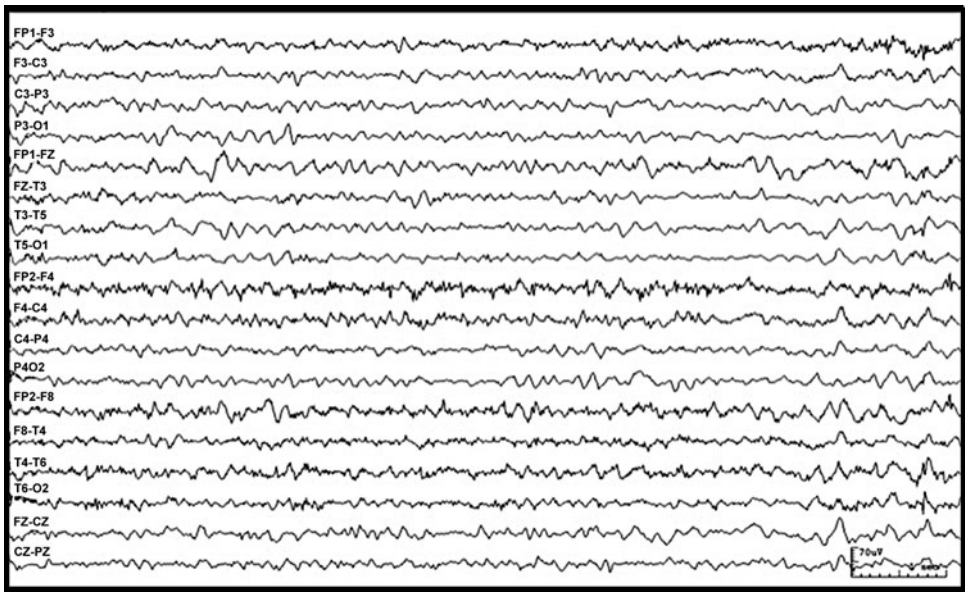

Fig. 2 Electroencephalogram performed 7 days after the first exam, and 4 days after treatment with methylprednisolone, showing marked improvement of background rhythms and disappearance of triphasic waves

three findings: clouding of consciousness, with reduced attention, or cognitive function; no CSF evidence of bacterial or viral infection; high serum levels of antithyroid microssomal, antithyroid peroxidase, or antithyroglobulin antibodies [1, 2]. Brain MRI can disclose mesial temporal lobe lesions or multiple subcortical ischemic areas, or can be normal. In general, EEG abnormalities include diffuse slow wave abnormalities that may correlate with the degree of severity of the underlying encephalopathy [3]. Other abnormalities may be found is some cases, such as triphasic waves and focal epileptiform discharges [4, 5].

Usually, EEG findings in most symptomatic patients include mild to severe generalized slow wave abnormalities [4, 5]. Several reports have highlighted the presence of more localized findings on the EEG, with no correspondence in brain MRI. It has been suggested that focal disturbances is mediated by auto-immune complex deposition [4, 5]. Triphasic and atypical triphasic waves are also frequently found. Epileptiform discharges and PLEDS are uncommon in patients with HE, but are also reported. Improvement in clinical and EEG findings at the same time has been described in some previous data [4]. Also, EEG is useful in excluding other conditions such as Creutzfeldt-Jakob disease or status epilepticus in patients with rapidly evolving encephalopathy [4].

In summary, our report reinforces the idea that EEG is extremely useful in monitoring or following HE patient's course after corticosteroids therapy.

Conflict of interest We have no conflict of interest.

Ethical statement Full consent was got from the patient for the case report publication.

References

1. Chong JY, Rowland LP, Utiger RD (2003) Hashimoto encephalopathy: syndrome or myth? Arch Neurol 60:164–171
2. Shaw PJ, Walls TJ, Newman PK, Cleland PG, Cartlidge NE (1991) Hashimoto's encephalopathy: a steroid-responsive disorder associated with high anti-thyroid antibody titers—report of 5 cases. Neurology 41:228–233
3. Forchetti CM, Katsamakis G, Garron DC (1997) Autoimmune thyroiditis and a rapidly progressive dementia: global hypoperfusion on SPECT scanning suggests a possible mechanism. Neurology 49:623–626
4. Schauble B, Castilho PR, Boeve BF, Westmoreland BF (2003) EEG findings in steroid-responsive encephalopathy associated with autoimmune thyroiditis. Clin Neurophysiol 114:32–37
5. Rodriguez AJ, Jicha GA, Steeves TD, Benarroch EE, Westmoreland BF (2006) EEG changes in a patient with steroid-responsive encephalopathy associated with antibodies to thyroperoxidase (SREAT, Hashimoto's encephalopathy). J Clin Neurophysiol 23:371–373