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Case Report Open Access

Unexplained Dysphasia and Disorientation as Signs of Ictal Activity in a Patient with Secondary Progressive MS: MRI Findings

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Abstract

A 42-year old woman with a 14 year history of secondary progressive multiple sclerosis developed new onset aphasia and disorientation. Magnetic Resonance Imaging (MRI) showed sequelae of ictal activity on diffusion- and perfusion-weighted MRI (hippocampal hyperintensity with a reduced apparent diffusion coefficient and spatially matching hyperperfusion). The epileptic origin was confirmed by c (EEG); under antiepileptic treatment aphasia improved over 3 days and the previous clinical disability level was reached, while the EEG normalised gradually. Isolated signal change restricted to the hippocampal area may provide a diagnostic clue to the underlying pathophysiology of confusional syndromes and help to identify non-convulsive status epilepticus.

Keywords: Dysphasia; Magnetic resonance imaging; Electro-encephalography; Confusional syndromes; Electro-encephalography; Magnetic resonance imaging

Introduction

A high percentage of MS patients develops secondary progressive MS after an initial relapsing-remitting disease course. This stage of MS is characterised by persistent neurological disability mostly limitations of locomotion but it may include all functional systems. Due to the reduced mobility and other functional limitations complications like infections are more likely to occur in this stage of MS. In case of new symptoms an acute relapse and new inflammatory activity as part of the MS needs to be considered. Also worsening of pre-existing neurological deficits may occur in the context of febrile infections are frequently encountered. For this reason all possible origins in particular non-neurological complications need to be considered in case of new symptoms. We report important diagnostic information that can help to diagnose a common possible complication that may occur in MS patients.

Case Report

A 42-year old woman with a 14 year history of secondary progressive multiple sclerosis (MS) and a recent expanded disability status scale (EDSS) score of 7.0 was brought to the emergency room by her husband in the late afternoon because of new onset aphasia and disorientation, that he had first noticed since in the morning of the same day. Focal myoclonic activity of the right arm was observed, which stopped after administration of 2 mg diazepam. Magnetic resonance imaging (MRI) of the brain was performed including Fluid Attenuated Inversion Recovery (FLAIR) sequences to demonstrate chronic MS lesions. Furthermore vascular and metabolic sequences were performed in order to detect either ischemic or metabolic acute changes to explain the acute clinical changes. FLAIR images showed MS related chronic pathology with extensive T2-hyperintense white matter lesions and signs of MS brain atrophy, but no contrast enhancing lesions were present, that would have pointed to new inflammatory activity. There was a high number of chronic white matter lesions and confluent subcortical abnormality on both T2- and T1-weigthed MRI. Both medial temporal lobes were strongly affected by white matter lesions and hippocampal T2-hyperintense tissue changes bilaterally. In addition, on diffusion weighted- and dynamic contrast enhanced perfusion-weighted MRI (DWI resp. PWI) there were remarkable left hippocampal signal changes unrelated to the chronic MS pathology [1]. There was left hippocampal hyperintensity on the DWI in combination with a reduced apparent diffusion coefficient (ADC) and spatially matching signs of hyperperfusion on time-to-peak (TTP) maps. The combination of increased perfusion in the presence of a DWI hyperintensity and a reduction of the ADC are typical sequelae and specific findings of ictal activity [2]. The epileptic origin of symptoms was confirmed by EEG 1.5 hours after MRI showing a left temporal regional slowing with epileptic discharges. On antiepileptic treatment aphasia improved gradually over the following 3 days and the previous clinical disability level was reached, while the EEG normalised gradually (Figure 1).

The ADC decrease observed after excitatory overactivation in status epilepticus both in patients and animal models is due to metabolic changes. Prolonged ictal activity is known to increase glucose utilization, the increase of which is not adequately matched by the enhanced blood flow [3,4]. As a result, blood flow-metabolism uncoupling leads to a reduction of high-energy adenosine phosphates and tissue hypoxia, thereby stimulating anaerobic glycolysis. The generation of lactate increases tissue osmolality which, in turn, contributes to the reduction of water mobility due to failure of energy dependent ion exchange and/or shrinkage of the extracellular space by water shifts into the intracellular compartment. It is assumed, that the observed regional hyperperfusion serves as a compensatory mechanism, yet insufficient to prevent the stimulation of anaerobic glycolysis due to the prolonged ictal activity. As a consequence of prolonged ictal overactivation compromised energy metabolism and a rise of lactate, ADC reductions may develop. This mechanism and these MRI findings are not specific to MS, but do develop as response to the ictal activity. Many different underlying pathologies have been reported to be the potential epileptic trigger. This includes brain neoplasms, cerebral venous sinus thrombosis and acute or chronic ischemic lesions [5].

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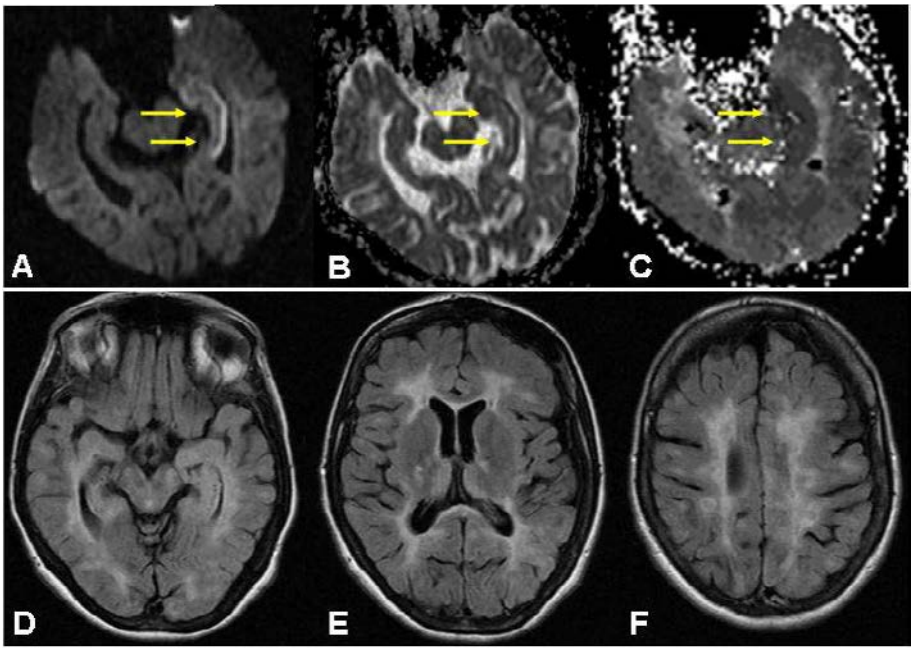

Figure 1: FLAIR images (D-F) show extensive chronic hyperintense periventricular and juxtacortical MS lesions. In (D) it is demonstrated that the periventricular area of the temporal horns of the lateral ventricles is strongly affected by hyperintense lesions including involvement of both hippocampi. Diffusion-weighted MRI (A) demonstrates high signal in the left hippocampus (yellow arrows) with corresponding low signal in the apparent diffusion coefficient map (B), and low signal intensity in perfusion-weighted MRI on the time-to-peak (TTP) map (C) indication hyperperfusion.

Discussion and Conclusion

Although MS is characterised primarily by white matter pathology lesions in the gray matter have been known for a long time as shown by pathology studies [6]. Most cortical lesions are leuco-cortical, reaching from the white matter into to the adjacent gray matter and can be readily detected by double inversion recovery MRI [7]. For this reason it is not surprising that patients with MS have an approximately threefold increased risk of seizures and a cumulative risk of 3.1% over 15 years' disease duration of developing epilepsy [8,9]. In many cases the etiology of seizure associated symptoms may be apparent, especially if tonic-clonic activity is observed initially [10]. However, in case of prolonged confusional states - as in this case - MRI findings can help to reach a definite diagnosis of ictal activity. For clinicians, isolated signal change limited to the hippocampal area may thereby provide a diagnostic clue to the underlying pathology of complex partial status epilepticus [11].

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