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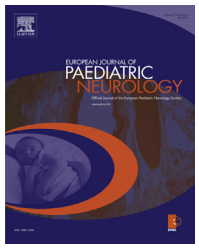

Original article

Cortical thickness at the time of the initial attack in two patients with paediatric relapsing–remitting multiple sclerosis

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ABSTRACT

Background: Multiple sclerosis (MS) is a chronic demyelinating disease of the central nervous system with a low incidence in the paediatric population; cortical atrophy is often striking, even in the early stages of the disease. Evidence of cortical thinning in childhood MS is scant.
Aims: This study aimed to assess cortical thickness in paediatric patients during the initial attack of remitting–relapsing MS.
Methods: We report two cases of remitting–relapsing MS, with initial attacks at 12 and 16 years of age. We analysed brain cortical thickness (CTh) in these patients and compared these data to the CTh of a control group comprised of six 12-year-old females and six 16-year-old males.
Results: Both cases exhibited a total brain CTh significantly below that of the control group. This difference was also observed when analysing the CTh of all lobes except the left parietal lobe in one of the cases.
Conclusions: Cortical atrophy is already present at the time of onset of MS. Studies with larger patient populations that have a more homogenous clinical presentation could identify the time of onset of cortical atrophy and use this parameter as a prognostic and/or treatment marker of MS.

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1. Introduction

Multiple sclerosis (MS) is a chronic demyelinating disease of the central nervous system that is rare in children (0.51/100,000 person-year).[1–3] Between 3 and 10% of individuals with MS are children under the age of 16 years.[4–7]

MS affects cortical and subcortical white and gray matter of the central nervous system.[8–12] Neocortical atrophy is prominent in MS, and synaptic loss is particularly striking. These features may independently contribute to the expression of neuronal disease in MS patients.[13–16]

Cortical atrophy can appear in the early stages of MS.[13,15] Cortical thinning is an early, diffuse phenomenon in adult patients who receive an early diagnosis of MS based on clinical signs in the initial stages of the disease[13,15,17–19]; however, few studies evaluated cortical thickness (CTh) and atrophy in childhood-onset MS.[8,12,20]

The purpose of this study is to report two paediatric clinical cases with prospective follow-up that meet the diagnostic criteria for the remitting–relapsing form of MS.[2,21,22] These cases presented distinct cortical atrophy, which was already discernible at the time of the initial attack.

2. Clinical cases

2.1. *Clinical case 1*

A 12-year-old female with no personal or family history of note was admitted to the hospital due to acute onset of facial asymmetry and diplopia. At admission, the neurological examination revealed left facial paralysis, which was associated with paralysis of the left VI cranial nerve, with no additional findings of note.

Upon admission, a cranial computerized tomography was performed with normal findings. Cerebral magnetic resonance revealed the presence of at least 4 demyelinating infratentorial lesions: 2 in the left cerebellar hemisphere, 1 in the pons, and 1 in the mid-brain. There were more than 20 supratentorial lesions; the most significant of these lesions were as follows: 7 in the periventricular white matter, 5 pericallosal lesions, 3 in the corpus callosum, 4 in the centrum semiovale, and 7 bilateral juxtacortical, frontoparietal lesions (Fig. 1A). The magnetic resonance of the spinal cord was normal.

The study also included the following tests[23]: blood count, basic metabolic studies, sedimentation rate, PCR, liver function tests, serum B12 and folate levels, proteinogram, angiotensin-converting enzyme, and immunological (i.e., antinuclear antibodies, TSH, antiDNA anti-Sm, anti RNP, anti SS-A, and anti SS-B) and virological (i.e., serum agglutination test for *Brucella*, *Borrelia*, HIV, and Epstein Barr titres) studies, which were all normal.

The cerebrospinal fluid analysis revealed discrete hyperproteinorraquia (480 mg/L), with the presence of oligoclonal bands.

The ophthalmological examination was normal. The study using visual evoked potentials revealed a discrete p100 latency in both eyes (p100 right at 121 ms and left at 118 ms).

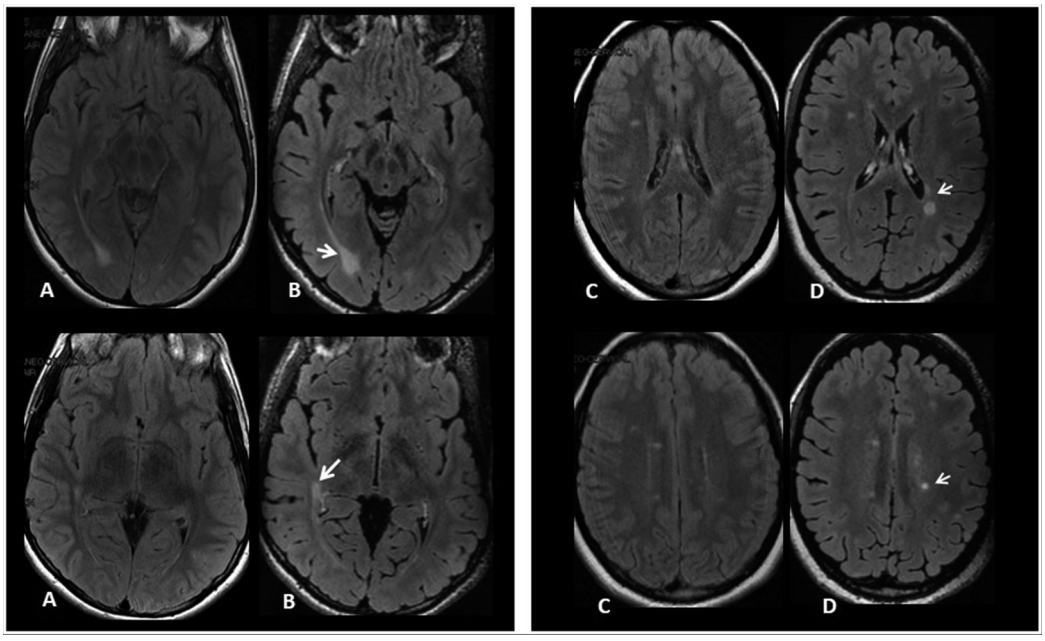

Fig. 1 – Axial T2-weighted FLAIR images from the clinical cases. Clinical case 1 (A–B). (A) Small hyperintense lesions located in the periventricular white matter adjacent to the right occipital horn and the right ventricular atrium, which are compatible with demyelinating lesions. (B) Control MRI performed two years later. Increased lesion size (indicated by white arrows) due to reactivation or confluence of new lesions. Clinical case 2 (C–D). (C) Hyperintense lesions located in the periventricular white matter and in paracallosal areas perpendicular to the greater ventricular axis, which are characteristic of a multiple sclerosis-type demyelinating disease. (D) Control MRI performed two years later. Appearance of new lesions (indicated by white arrows), indicating progression.