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Mult Scler 2008; 14; 248 originally published online Jan 21, 2008;
DOI: 10.1177/1352458507082617

The online version of this article can be found at:
http://msj.sagepub.com/cgi/content/abstract/14/2/248

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SHORT REPORT *Multiple Sclerosis* 2008; **14**: 248–251

Brain magnetic resonance imaging findings in acute relapses of neuromyelitis optica spectrum disorders

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We studied cranial magnetic resonance imaging (MRI) lesions in three women with acute attacks of recurrent longitudinally extensive transverse myelitis (r-LETM), recurrent-optic neuritis (r-ON) and r-LETM-CNS. Neuromyelitis Optica -immunoglobulin (IgG) antibody was positive in all cases. Brain MRI (1.5 Tesla) was performed according to protocol from consortium MS centre. We described the cranial lesions in brain MRI of acute relapses. These lesions were different from MS, most had an asymptomatic course which disappeared with time, protocol from consortium of MS centre criteria for brain MRI and seropositivity of NMO-IgG are useful tools for differentiate acute lesions of NMO/MS. *Multiple Sclerosis* 2008; **14**: 248–251. http://msj.sagepub.com

Key words: neuromyelitis optica spectrum disorders; recurrent longitudinally extensive transverse myelitis; recurrent optic neuritis

Introduction

Several clinical forms of neuromyelitis optica spectrum disorders (NMO-SD) have been observed: monophasic, relapsing-NMO (R-NMO), recurrent longitudinally extensive transverse myelitis (r-LETM) and recurrent-optic neuritis (r-ON) [1].

Two studies have shown brain magnetic resonance imaging (MRI) abnormalities in 60– 65.5%, but only in R-NMO cases that were found to be stable at the moment of the studies [2,3].

Brain MRI findings and their course in acute relapses of r-LETM and r-ON are not known.

The objective of this study was to report the serial brain MRI findings in three cases during acute relapses of NMO-SD.

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Received 19 May 2007; accepted 27 June 2007

10.1177/1352458507082617

Methods

Three cases with acute relapses of NMO-SD with brain MRI lesions were selected as follows:

1. r-LETM: Two or more acute relapses of: recurrent transverse myelitis without overt visual involvement and spinal cord MRI with lesion(s) extending over three or more vertebral segments
2. r-ON: Recurrent bilateral or unilateral ON that led to severe vision loss without overt spinal cord involvement
3. r-LETM-CNS: r-LETM associated with symptoms of the central nervous system (CNS) and,

all the above criteria should have absence of systemic diseases or neoplasm and NMO immunoglobulin G antibody (NMO-IgG) seropositivity detected by immunohistochemistry using an avidin-biotin technique on paraformaldehyde-fixed frozen sections of rat brain as described previously [4].

All cases had evoked potentials (EP), non-organ specific autoantibodies/sarcoid and CSF studies.

Serial Brain/spinal MRI imaging studies were performed on a 1.5 Tesla MRI system according to the protocol of the consortium of multiple sclerosis centers [5].

Results

Case 1. A 30 year-old female with history of three relapses of ON. She had a new ON relapse. Somatosensory EP, spinal cord MRI and complementary tests of viruses/non-organ-specific antibodies and CSF were normal. NMO-IgG antibody were positive.

Brain MRI in the acute relapse showed an enlarged right parietal white matter lesion >5 mm and another -1 mm- in the left hemisphere. Lesions diminished in size until they gradually disappeared (Figure 1).

Case 2. A 39-year old woman presented two cervical LETM attacks. Serological tests for viruses/non-organ-specific, visual EP (VEP) and CSF were normal. NMO-IgG detection antibody was positive. Serial MRI showed confluent lesions in cervical spinal cord (Figure 1). Brain MRI was normal. She had a third relapse with cervical-thoracic LETM and manifested episodes of hiccup/nausea and dystonia. Spinal cord MRI, in the acute episode, showed increased lesions in cervical and thoracic levels. Brain MRI evidenced numerous lesions (Figure 1). T1 hypointense lesions were not detected. Three months later brainstem and corpus callosum lesions diminished and periventricular disappeared (Figure 1).

Case 3. A 63 year-old woman with history of two thoracic/cervical attacks of LETM. Serological tests for viruses/non-organ-specific tests, CSF and VEP were normal. NMO-IgG detection antibody was positive. Spinal cord MRI demonstrated a cavitation-like lesion from cervical spinal cord to medulla. Brain MRI demonstrated periventricular and corpus callosum hyperintense lesions, but their morphology was not oval, ovoid nor perpendicularly orientated. Some subcortical/deep white matter lesions were also observed. A new axial brain MRI three months after, demonstrated a slight reduction of periventricular and corpus callosum lesions (Figure 1).

Discussion

We described three cases with clinical forms of NMO-SD with acute relapses. Serial Brain MRI demonstrated lesions that decreased or disappeared gradually. The evolution of the lesions in two cases was asymptomatic.

Case 1 with r-ON, brain MRI studies in the acute phase of the last relapse demonstrated two enlarged deep white matter lesions and both gradually disappeared.

The only abnormality described in brain MRI of r-ON is optic nerve high signal abnormalities which enhance the optic nerves [6]. However, brain MRI lesions observed during the acute phase of relapses in this case, indicate that in r-ON there can also be impairment outside the optic nerve but with an asymptomatic course. As to differential diagnosis of such lesions with MS, brain lesions did not fulfil Barkhof *et al.* [7] criteria as there were only two subcortical/deep white matter lesions. NMO-IgG antibody was present.

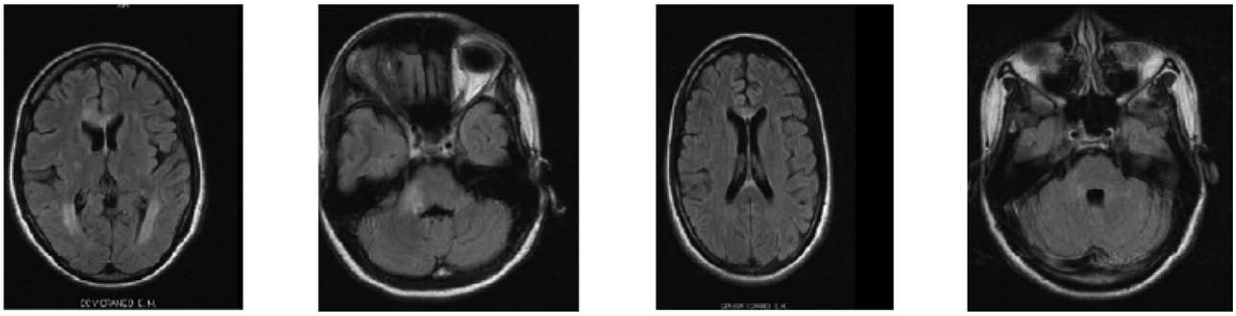
Case 2 had r-LETM/CNS with dystonia and hiccup/nausea episodes. Brain MRI performed in the acute phase demonstrated: periventricular, corpus callosum, but not oval, ovoid or perivenular as in MS lesions, four subcortical/deep white matter hyperintense and two extensive lesions in the brainstem. The characteristics of the brain MRI lesions in this case have been described in R-NMO [3]. In addition, NMO-IgG antibody was positive.

Three months later brain MRI showed a diminishment of brain stem/corpus callosum lesions, periventricular lesions disappeared and only some subcortical/deep white matter lesions remained.

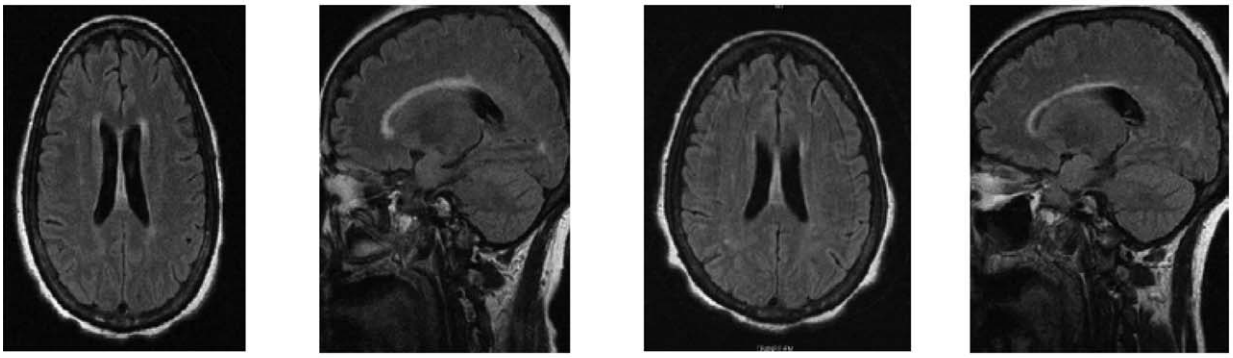
Case 3 had two episodes of r-LETM with no clinical signs of impairment, neither optic nerves nor CNS. Nevertheless, brain MRI studies in the acute phase of the last relapse evidenced eight lesions: six subcortical/deep white matter lesions, one infratentorial and periventricular, respectively, which none fulfilled MS Barkhof *et al.* [7] criteria.

In addition, NMO-IgG antibody was positive and confirmed the diagnosis of r-LETM. Three months later, these brain MRI lesions had slightly diminished.

Case 1



Case 2



Case 3

Figure 1 Case 1. Recurrent-Optic Neuritis. Brain MRI axial FLAIR in the third relapse of optic neuritis showing an enlarged lesion (>5 mm) in the deep white matter of the right parietal hemisphere. There was also another lesion (1 mm) in the white matter of the left hemisphere. Same lesion three months after the relapse as one deep white matter lesion (1 mm) in right hemisphere.
Case 2. Recurrent-longitudinally extended transverse myelitis with central nervous system symptoms. Brain MRI in the acute phase of the third relapse. Axial FLAIR. Lesion (>5 mm) located in the brainstem. Periventricular and corpus callosum hyperintense lesions, but not oval, ovoid or perpendicularly oriented in morphology. Three months after the third relapse brain MRI showed a diminishment of brain stem and corpus callosum lesions. Periventricular lesions disappeared.
Case 3. Recurrent-longitudinally extended transverse myelitis. Axial and sagital Brain MRI in acute second relapse. Periventricular confluent .and corpus callosum hyperintense lesions but not oval, ovoid or perpendicularly oriented in morphology. A new brain MRI three months after the relapse, demonstrated a slight diminishment of periventricular and corpus callosum lesions.

In summary, brain MRI findings during acute relapses in these cases with R-NMO-SD evidenced:

i) Morphologically, periventricular lesions were not oval/ovoid/ perpendicularly orientated; ii) confluent lesions could appear in periventricular white matter as well as in deep white matter and infratentorial, but they were transient; iii) these periventricular lesions disappeared and only some subcortical/deep white matter lesions remained. Serial Brain MRI lesions after acute relapses in these NMO-SD cases most had an asymptomatic course and disappeared with time.

A recent study on the pathology of brain involvement in NMO-SD, have confirmed our MRI findings which have demonstrated in one case of r-LETM-SNC that a biopsy of the temporal lobe was pathologically similar with the NMO lesion in the spinal cord [8].

In conclusion, cranial lesions in brain MRI appeared in acute relapses of NMO-SD but they were transient, different from MS and disappeared with time. Protocol for MS from Consortium of MS centres, Barkhof *et al*.[7] criteria for brain MRI and seropositivity of NMO-IgG are useful tools to differentiate acute lesions of NMO/MS.

Acknowledgement

NMO-IgG antibody was assessed support in part by a grant from Fondo de Investigaciones Sanitarios, Madrid, Spain (PI 60070; AS, FG).

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