**INTRODUCTION**

Multiple sclerosis (MS) is an inflammatory and neurodegenerative disease of the central nervous system (CNS) characterized by myelin loss, axonal pathology and progressive neurocognitive impairment (1). Recent studies have shown a significant comorbidity between migraine and MS. Patients with migraine have a higher risk of developing MS than non-migraineurs, with MS patients being more than twice as likely to report migraine than controls (2). Migraine has been reported as a presenting symptom of the onset of MS with a higher frequency (3). Status migrainosus as clinical overtue symptom of multiple sclerosis must be considered as exceptional.

Here, we report the first case of an Italian patient that showed as presenting symptom of the onset of multiple sclerosis a status migrainosus in the setting of a demyelinating lesion in the caudal brainstem.

**CASE REPORT**

In October 2012, a 37 year-old woman was admitted to the emergency department of our hospital with a severe left sided fronto-temporal throbbing headache, associated with severe nausea and photo/phonophobia, lasting 3 days. A positive family history for migraine was reported and her personal history was typical for migraine without aura, according to ICHD-II diagnostic criteria, with 1-2 attacks per month since adolescence. The neurological examination at admission was normal, the computed tomography scan of the brain did not show any focal lesions. The referred symptoms fulfilled ICHD-II version criteria for episodic migraine without aura (4). She was therefore treated with oral NSAIDS and was discharged. After 10 days, due to persistence of the headache, the patient was newly admitted at the emergency department. A diagnosis of status migrainosus was made according the ICHD-II criteria and the patient was hospitalized. On neurological examination, a superficial hypoaesthesia involving all the three branches of left trigeminal nerve was evidenced. A brain magnetic resonance imaging (MRI) with angioMRI interestingly showed an enhancing T2 hyperintense lesion extending from caudal brainstem to C1 level (Figure 1 and 2). Brain MRI also showed multiple areas of abnormal T2 signal without contrast enhancement in subcortical and deep white matter of both frontail and parietal lobes. Immune-rheumatologic and metabolic screening resulted normal. At cerebrospinal fluid examination oligoclonal bands were detected. Hence, diagnosis of Clinical Isolated Syndrome was performed. The patient was treated with methylprednisolone i.v. 1 g/day for 10 days. The subject was asymptomatic for migraine after 5 days of i.v. therapy, whereas the trigeminal hypoaesthesia gradually disappeared in less than a month. After one-year follow up, neuroradiological examination showed a new active demyelinating lesion in the spinal cord (C4 level). Based on the established McDonald criteria, a diagnosis of multiple sclerosis was made. After 2 years, the patient is still asymptomatic for both migraine and other neurological dysfunctions. To date, she does not receive any disease-modifying therapy.

**REFERENCES**