An unusual aetiology for a not-so-unusual movement disorder

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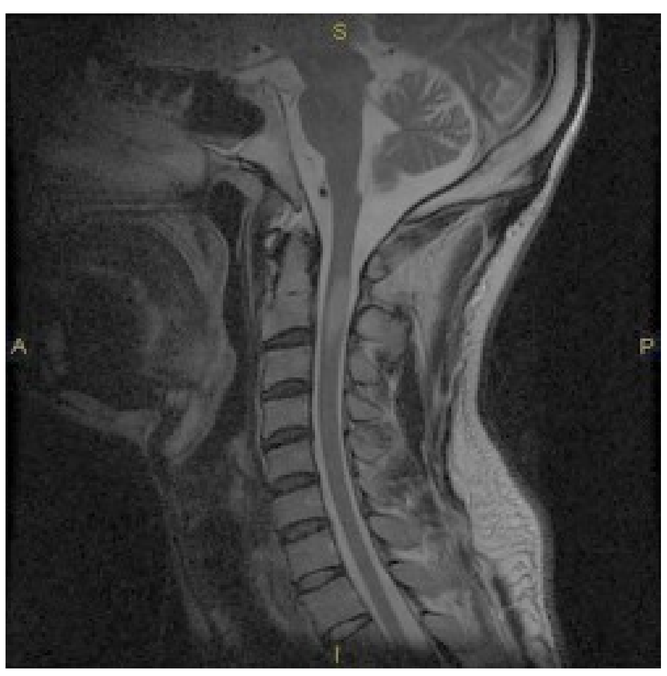
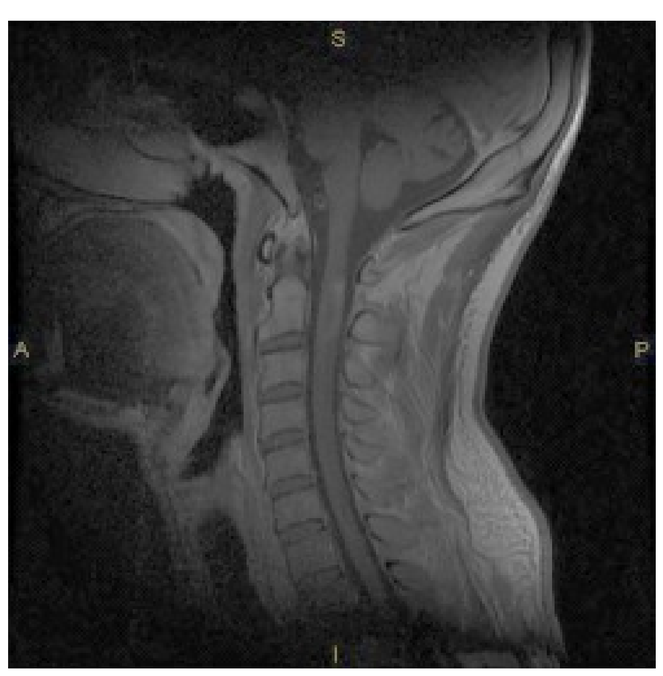
Background. *Spinal lesions are rarely investigated as cause of movement disorders and the underlying pathogenetic mechanisms are not well known yet. The most claimed hypotheses to explain such an association are: ephaptic transmission between damaged neurons of corticospinal tracts and ascending pathways, interaction between spinal interneurons, alteration of cell membrane and its ion channels. The aim of our report is to focus the role of spinal pathology in dystonic postures and other movement disorders.*

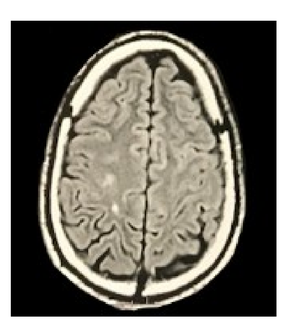
Case report. *A 32-year-old man, baker, no family history for neuropsychiatric diseases, complained subacute onset of sneaky numbness of both hands, with slight impairment of sensibility. He consulted a neurologist who didn't find any objective deficit and recommended a spinal MRI, speculating a radicular pathology. The numbness progressively worsened and, within less than one month, weakness of the four limbs appeared. He was admitted to the neurological Department.*
Neurologic examination showed dystonic postures of both hands with irregular, slow, non-stereotyped finger movements, which prevented voluntary actions. Arm strength wasn't really impaired.

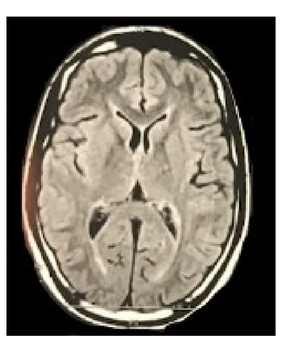
- *Brain and spinal cord MRI showed multiple supratentorial T2-hyperintense areas in cerebral hemispheres, not involving basal ganglia, and a gadolinium-enhancing lesion involving spinal posterior columns at C2 level (Figure 1). MRI lesions were suggestive of Multiple Sclerosis (MS).*
- *A spinal tap revealed a slight WBC increase and the presence of intrathecal IgG synthesis. Further instrumental and biochemical examinations showed negligible results except for visual evoked potentials, which were altered. Anti AQP4 antibodies were negative.*

The patient received intravenous high dose corticosteroids and pregabalin up to 900 mgs/day. Since pregabalin was ineffective, carbamazepine (up to 200 mgs/day) was introduced.
Our patient gradually and substantially recovered; the benefit was maintained on long term follow-up. One year from onset dystonic postures almost totally disappeared and hand movements weren't clumsy anymore. He keeps on taking carbamazepine. MS modifying therapy has been initiated after six months from the first evaluation because of dissemination in time on new MRI scan. We consider the C2 lesion to be responsible for our patient's movement disorder.









Conclusions. Very few reports describe the role of spinal pathology in paroxysmal dystonias and they mainly concern Neuromyelitis Optica patients often during recovery from a myelitis episode. Recognition of movement disorders of spinal origin in inflammatory diseases, such as MS, is important because of the good response to appropriate therapy (carbamazepine, phenytoin, acetazolamide) in add-on to high dose steroids.

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