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# Tourette syndrome and multiple sclerosis: a case report

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Tourettism and tics have rarely been reported in multiple sclerosis (MS).<sup>1-5</sup> Tourette syndrome (TS) is a complex neuropsychiatric disease of uncertain etiology not previously reported in MS.<sup>1-5</sup> Here, we present a patient with TS who was later diagnosed with MS.

In 2013, a 29-year-old male was referred for possible motor neuron disease after developing left arm fasciculations, numbness, and weakness for 5 years. The patient agreed to the discussion and publication of this case, and his anonymity was guaranteed. Electromyography revealed restricted fasciculations and re-innervation in the left arm, with normal nerve conduction studies. He was diagnosed with TS: multiple motor and vocal tics (head turning, blinking, throat clearing, humming), coprolalia, and obsessive compulsive disorder. His neurological exam revealed left proximal arm paresis (4/5) with proximal fasciculations, decreased left biceps and triceps reflexes, atrophy, and no sensory deficits. He was started on risperidone for tics and obsessive compulsive disorder, with good response. Tics began at 10 years of age and

progressed over the years. His maternal grandfather had tics and a history suggestive of TS.

Cervical spine magnetic resonance imaging revealed a cervical demyelinating lesion (Figure 1A).

The fasciculations and left arm weakness improved over the next months. In 2016, he experienced bilateral leg weakness. Brain magnetic resonance imaging revealed slowly progressive subcortical white matter lesions (including brainstem plaques), characterizing dissemination in time and space (Figures 1B and C). The cerebrospinal fluid protein and cell count were normal, with no oligoclonal bands. Anti-aquaporin 4, ANA, P and C-ANCA, RF, anti-SSA and SSB, anticardiolipin, and lupus anticoagulant were negative. The patient was diagnosed with MS and started on glatiramer acetate (Copaxone<sup>©</sup>) sc 20 mg/day in December 2017. Risperidone (1 mg/day) and clonazepam (1 mg/day) were continued to control tics and obsessive compulsive disorder. In 2019, he developed insomnia, worsening tics, irritability, and depression, and was started on sertraline 25 mg/day and an increased dose of risperidone (1 mg BID). As of his last outpatient visit to our clinic (January 2023), no further MS relapses have been reported after glatiramer treatment began. Glatiramer had no effect on TS symptoms, which were well controlled with risperidone, clonazepam, and sertraline.

To our knowledge, this is the first case to simultaneously fulfill all diagnostic criteria for TS in the DSM 5/ ICD-10 and the latest McDonald MS criteria. Tics have been rarely reported in MS and, among the 5 reported cases of tics and/or tourettism (features of TS that do not meet all the criteria), the movement disorders were considered secondary to MS disease activity, i.e., MS plaques affecting the basal ganglia. Our patient had no basal ganglia demyelinating plaques. Tics and tourettism in MS have been attributed to involvement of the corticostriatal-thalamocortical circuit and/or basal ganglia.

The phenomenology underlying the 5 other reported MS cases with tics was quite variable, but only one mention of tourettism was made, coincidentally in a Brazilian patient. Both cases of MS patients with tics were reported in the USA, and these patients had serious behavioral disorders. One of them was diagnosed with Asperger syndrome, pedophilia, and seizures; the tics were the least striking symptom. The other had serious issues with criminal behavior and substance abuse. The 2 patients from Italy had secondary progressive MS. Finally, the Brazilian patient with Tourettism had phonic tics following diagnosis of MS.

In summary, our patient is the first report of an association between TS and MS. No basal ganglia plaques were identified. Further studies are necessary to determine whether this association is coincidental or due to a common genetic and/or (less likely) immunological basis. In classic autoimmune disorders, there is ample evidence of genetic susceptibility – at least in part – related to human leukocyte antigen subtypes. However, in TS, there is evidence that both autoimmune and genetic factors may play a significant role, indicating the need for more elaborate and collaborative studies in this field.

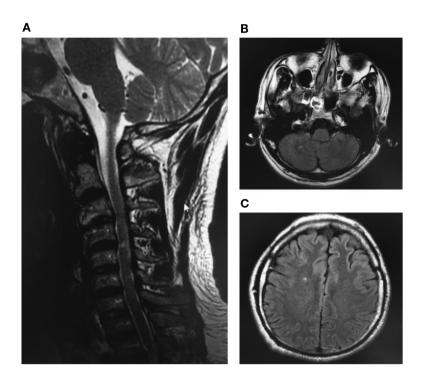


Figure 1 Brain and cervical spine neuroimaging of a patient with Tourette syndrome and multiple sclerosis. A) Flair image of the cervical spinal cord showing the demyelinating lesion near the C5 level; B) and C) latest brain magnetic resonance imaging depicts scattered bilateral subcortical and brainstem white matter lesions.

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