

A New Diagnosis of Multiple Sclerosis in a 29-Year-Old Former Collegiate Basketball Player With Initial Symptoms of Recurrent Bell's Palsy: A Case Report

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Abstract

Multiple sclerosis (MS) is a demyelinating disorder of the central nervous system, characterized by immune-mediated destruction of myelinated axons, which leads to notable physical disability. Although presence among active athletes is extremely low, it can cause considerable lifestyle changes for those athletes affected as they transition into life after competitive athletics. We describe a 29-year-old former collegiate women's basketball player in whom MS was diagnosed during a visit for recurrent evaluation of Bell palsy. Subsequent neurology consult and magnetic resonance imaging led to the confirmation of the diagnosis of MS, and the patient began treatment for relapsing-remitting MS. During visits of what appear to be straightforward neurological symptoms, it is imperative to collect a full neurologic history and perform a detailed physical examination to determine whether a more systemic disease process could be at fault, which may subsequently lead to earlier detection and treatment of MS.




Introduction

Multiple sclerosis (MS) is an immune-mediated, demyelinating disease that affects the central nervous system—leading to considerable physical disability.¹ MS currently affects more than two million people worldwide according to the National Multiple Sclerosis Society, and current estimates state that more than 400,000 individuals are affected in the United States.¹ Recent data show that MS affects more women than men by a two-to-one ratio.² Patient age at diagnosis is traditionally between 20 to 50 years.¹

Numerous hypotheses exist as to which individuals get MS, but most experts agree that it is a combination of genetic and environmental factors. Genetic factors comprise mainly ethnicity and sex, with current research directed toward specific single-nucleotide polymorphisms and histocompatibility complexes.^{3,4} Environmental factors include geography and vitamin-D factors (eg, the closer that patients live to the equator, the less likely they are to get MS); additionally, higher levels of vitamin D are thought to be protective against MS.⁵⁻⁷

The diagnostic criteria for MS have gradually changed. For the 2010 revision to the McDonald criteria, the goal was to simplify the ability to show lesions in space and time with the help of magnetic resonance imaging (MRI) (Figure 1). MS is typically divided into four categories: relapsing-remitting, secondary progressive, primary progressive, and progressive-relapsing, with relapsing-remitting being the most common criterion.⁸

Treatment of MS is typically divided into acute attacks and disease-modifying agents. Acute attacks are treated with a burst of corticosteroids, whereas disease-modifying agents (including glatiramer acetate, interferon, and natalizumab) are used in attempt to reduce frequency of relapse.⁹ We describe diagnosis of MS in a former collegiate athlete who initially had presented with symptoms of Bell's palsy. The patient was informed that the data concerning her case would be submitted for publication, and she provided verbal consent.

  		
Diagnosis of MS requires elimination of more likely diagnoses and demonstration of dissemination of lesions in space (DIS) and time (DIT)*		
CLINICAL (ATTACKS)	LESIONS	ADDITIONAL CRITERIA TO MAKE DX
2 or more	Objective clinical evidence of ≥ 2 lesions or objective clinical evidence of 1 lesion with reasonable historical evidence of a prior attack	None. Clinical evidence alone will suffice; additional evidence desirable but must be consistent with MS
2 or more	Objective clinical evidence of 1 lesion	DIS; OR await further clinical attack implicating a different CNS site
1	Objective clinical evidence of ≥ 2 lesions	DIT; OR await a second clinical attack
1	Objective clinical evidence of 1 lesion	DIS OR await further clinical attack implicating a different CNS site AND DIT; OR await a second clinical attack
0 (progression from onset)		One year of disease progression (retrospective or prospective) AND at least two of: DIS in the brain based on ≥ 1 T2 lesion in periventricular, juxtacortical or infratentorial regions; DIS in the spinal cord based on ≥ 2 T2 lesions; or positive CSF

* Folman et al. Diagnostic criteria for multiple sclerosis: 2010 revisions to the McDonald Criteria. *Ann Neurol* 2011;69:292-302. * See reverse for DIS and DIT

Figure 1. The revised diagnostic criteria in 2010 for multiple sclerosis, used by the National Multiple Sclerosis Society. (Diagnostic criteria. National Multiple Sclerosis Society website. <http://www.nationalmssociety.org/For-Professionals/Clinical-Care/Diagnosing-MS/Diagnosing-Criteria>. Accessed February 4, 2017).

Case Report

A 29-year-old former collegiate basketball player presented to our sports-medicine clinic with concern for Bell's palsy. She had reported that this was the second occurrence of Bell's palsy; she had experienced a similar episode about a year earlier, which resolved after a 5-day treatment of oral corticosteroid. She reported the inability to fully close her left eye since the onset of symptoms, about 7 to 10 days before presentation to our clinic. She also reported some fullness in her right ear. She did not mention any hyperacusis, loss of taste, numbness, tingling of her extremities, or diffuse weakness. She did report some mild blurry vision in her left eye along with some lateral gaze disturbance, but she attributed this to her inability to close the eye fully and said that this was similar to her last bout of Bell's palsy 1 year earlier. Additionally, the patient did not have any recent viral infection of the upper respiratory system or other viral prodrome.

On physical examination, results of the Head, Eye, Ear, Nose and Throat test and neurological examination were remarkable for inability to raise the left eyebrow. Her left eye could only be covered with full, maximal effort. Upon smiling, the left side of her mouth was noted to not raise as much as the right side to produce an asymmetrical smile. Additionally, the patient was noted to have end-gaze nystagmus on both eyes.

The patient was discharged from the clinic with a 5-day, 60-mg prednisone burst for Bell's palsy treatment; however, owing to presence of nystagmus, we consulted our colleagues in neurology for additional recommendations. Neurologists agreed with the current treatment plan and diagnosis because of the lack of other systemic symptoms, but they advised to have a low threshold to get an MRI owing to concern for mass, a tumor of the central nervous system, cavernous sinus aneurysm, or MS.

At 1 week after the initial visit to our clinic, the patient returned for scheduled follow-up. Her facial palsy was improved, but she continued to report lateral gaze disturbance and occasional diplopia, which worsened during lateral gaze. She noted these symptoms the most while playing pick-up basketball and during kickboxing class. While playing basketball, she also suffered from an unusual sluggishness and "couldn't move normally." She did not report any history of diplopia or blurry vision during periods of raised internal temperature. We again discussed the case with neurologists, who recommended to further assess the patient's symptoms with an MRI, magnetic resonance angiography, and additional blood examinations (ie, tests for complete blood count; C-reactive protein levels; erythrocyte sedimentation rate; chemical substance levels; liver function; human immunodeficiency virus; treponema; and antinuclear antibody count). Because of concern for cavernous sinus

aneurysm, the patient was instructed to not work out or do any activities that would raise her blood pressure in the interim.

Results of laboratory studies were remarkable for a mildly elevated creatinine count to 1.16; total bilirubin count of 1.3 (direct, 0.3); and positive antinuclear antibody count, with a titer of less than 1:40. An MRI was obtained on the night of her second clinic visit (Figures 2A and 2B). The image revealed an abnormal moderate amount of scattered areas of cerebral and brainstem T2 prolongation, with distribution and formation suggestive of demyelinating plaques of MS. Multiple areas of T2 prolongation showed postcontrast enhancement. Differential consideration included collagen vascular disease and vasculitis, although this was thought less likely.

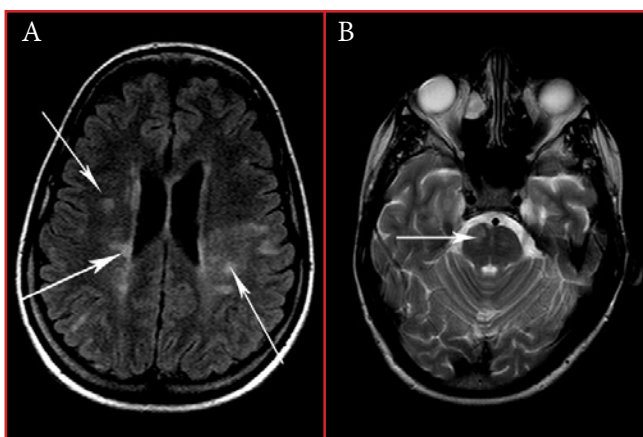


Figure 2. T2-weighted magnetic resonance imaging at 1 week after initial presentation. A) Abnormal moderate amount of scattered areas of cerebral T2 prolongation, with distribution and morphological features (arrows) suggestive of demyelinating plaques of multiple sclerosis. B) Specific lesion (arrow) of T2 prolongation in the pons, which may explain the patient's symptoms of palsy during lateral gaze.

The patient was referred to a neurology clinic for management of suspected MS. At that visit, the patient reported intermittent numbness of her left calf and left side of her trunk for the past 5 months, which were not reported during her initial visit for Bell's palsy. Her scan was reviewed in detail by the neurologist, who noted that the patient had a lesion on the left side of her pons, near the origin of her sixth and seventh cranial nerves. The presence of the lesion was consistent with the noted lateral gaze palsy, associated diplopia on lateral gaze, and facial nerve paresis. Diagnostic criteria was noted to have been met for MS on the basis of the "MRI, age, response to steroids, and duration of symptoms."

The patient recently underwent treatment with use of Rebif (interferon beta-1a) and vitamin D. At her most recent follow-up, she reported an increase in baseline

left-lower extremity pain and "hotness" during her exercise class. The neurologist recommended that she have an ice-cold beverage with her to prevent a rise in her core body temperature which is known to exacerbate symptoms of MS.

Discussion

Because MS affects the central nervous system, the disorder can result in severe limitations to physical activity.¹ Although studies have shown benefits from exercise in regards to a patient's "overall functional capacity and psychological well-being" in the presence of MS, the type of exercise should be discussed and exposure to heat should be limited.¹⁰

The current case fits a typical description of a patient with MS. Our patient was a woman aged 29 years, with an initial attack responding to a burst of steroids and a relapse about 1 year after. Perhaps a more thorough evaluation at her initial clinic visit could have elicited the report of left lower extremity numbness as indicated when she saw the neurologist. However, the sports-medicine physicians correctly suspected a more systemic process; thus, neurologists were consulted immediately. Although recurrent Bell's palsy may occur, it is very rare. In one of the largest studies involving Bell's palsy, it was determined that only 7% of patients had a recurrence, and mean time to recurrence was 10 years.¹¹

As such, the findings of this case can serve as a reminder that patients who present with symptoms of Bell's palsy, especially a recurrent symptom, should get a full neurological examination, with specific focus on the timing of symptoms in relation to activity; and an increase in core body temperature.

Funding

The authors received no financial support for the research, authorship, and publication of this article.

Conflict of Interest

The authors report no conflicts of interest.

References

1. Noonan CW, Williamson DM, Henry JP, et al. The prevalence of multiple sclerosis in 3 US communities. *Prev Chronic Dis* 2010;7(1):A12.
2. Who gets MS? (Epidemiology). National Multiple Sclerosis Society website. <http://www.nationalmssociety.org>

- org/What-is-MS/Who-Gets-MS. Accessed February 4, 2017.
3. Nischwitz S, Müller-Myhsok B, Weber F. Risk conferring genes in multiple sclerosis. *FEBS Lett* 2011;585(23):3789-97. doi: 10.1016/j.febslet.2011.03.037.
 4. Yeo TW, De Jager PL, Gregory SG, et al. A second major histocompatibility complex susceptibility locus for multiple sclerosis. *Ann Neurol* 2007;61(3):228-36.
 5. Kampman MT, Brustad M. Vitamin D: a candidate for the environmental effect in multiple sclerosis-observations from Norway. *Neuroepidemiology* 2008;30(3):140-6. doi: 10.1159/000122330.
 6. Munger KL, Levin LI, Hollis BW, Howard NS, Ascherio A. Serum 25-hydroxyvitamin D levels and risk of multiple sclerosis. *JAMA* 2006;296(23):2832-8.
 7. Islam T, Gauderman WJ, Cozen W, Mack TM. Childhood sun exposure influences risk of multiple sclerosis in monozygotic twins. *Neurology* 2007;69(4):381-8.
 8. McDonald WI, Compston A, Edan G, et al. Recommended diagnostic criteria for multiple sclerosis: guidelines from the International Panel on the diagnosis of multiple sclerosis. *Ann Neurol* 2001;50(1):121-7.
 9. Compston A, Coles A. Multiple sclerosis [published erratum in: *Lancet* 2002;360(9333):648]. *Lancet*. 2002 Apr 6;359(9313):1221-31.
 10. Crutchfield KE. Managing patients with neurologic disorders who participate in sports activities. *Continuum (Minneapolis)* 2014;20(6 Sports Neurology):1657-66. doi: 10.1212/01.CON.0000458968.40648.6f.
 11. Pitts DB, Adour KK, Hilsinger RL Jr. Recurrent Bell's palsy: analysis of 140 patients. *Laryngoscope* 1988;98(5):535-40.