BRIEF REPORT

Unrelated Neurologic problems mimicking Relapse of Multiple Sclerosis

Multiple sclerosis (MS) is a heterogeneous disease with various clinical signs and symptoms. Most of its lesions occur in ill-defined neuroanatomical sites; the lesions most often do not cause any signs and symptoms. MS may present with paraparesia, seizure and peripheral facial palsy with variable incidence rates.[1-3]

MS is indeed a clinical entity and its diagnosis may be mistaken with other neurological diseases causing similar clinical pictures; peripheral facial nerve palsy, seizure and paraparesia may be easily diagnosed as MS. In this way, it is not unlikely to overlook other neurological diseases and deprive the patient from receiving an appropriate treatment.

Herein, we present three known cases of MS with choleostatoma, cavernous hemangioma, and ossification of ligametum of flavum (OLF) presented with peripheral facial nerve palsy, seizure and paraparesia, respectively. In these patients, the neurological manifestations of other diseases were considered as MS manifestations.

Case 1: A 30-year-old man with progressive weakness and paresthesia in lower limbs from two years before. After 5–6 months, he developed weakness and right-sided paresthesia. Blurred vision in his right eye followed by his left eye and diplopia were other clinical presentations. From one year before, he had developed left-sided facial nerve paresis. He had a history of left middle ear infection since long years before.

Neurological examination (NE) revealed left-sided Marcus Gunn, left sixth cranial nerve palsy, right internuclear ophtalmoplegia (INO), left peripheral facial nerve palsy, right-sided hemiparesia (4/5), right-sided sensory loss, upward plantar reflex in right side and absent abdominal reflex. Imaging study with positive oligoclonal band (OCB) and results of visual evoked potential (VEP) and exclusion of collagen vascular disease (CVD), definitely confirmed the diagnosis of MS. MRI revealed that he had choleostatoma in his left ear (Fig. 1).

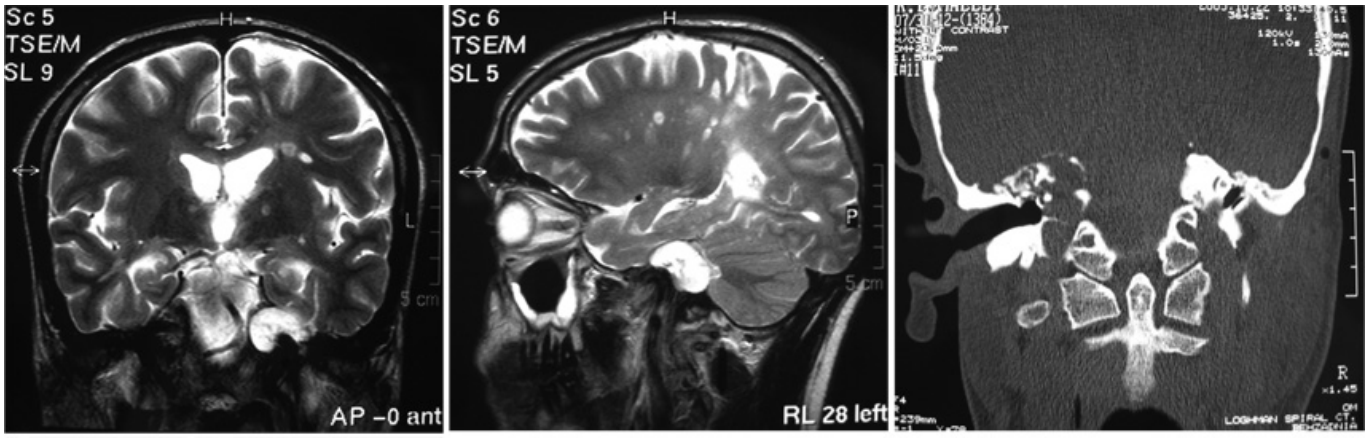
In this patient, although he had clinical course and several signs and symptoms of MS, presentation of peripheral facial nerve palsy was attributed to MS, until a careful physical examination and MRI revealed a choleostatoma The patient was treated surgically and discharged with interferon for treatment of his MS.

Case 2; A 43-year-old man developed right leg weakness eight years before followed by left leg weakness four years before. Then, he developed weakness of both legs so that from five months before he could walk only with crutches. He referred to us with the compliant of feet paresthesia, urinary frequency and urgency, history of vertigo, uncontrolled walking and blurred vision in both eyes. Since last year, he was treated with lamotrigine because of recurrent generalized tonic clonic (GTC) seizure. In neurologic examination, we found left Marcus Gunn pupil, hyperreflexia in his four limbs, lower limbs weakness (3/5), bilateral Babinski's sign and absence of abdominal reflex. He could not stand and walk without assistance.

Regarding the clinical course, imaging study, cerebrospinal fluid (CSF) analysis (positive OCB), VEP results and exclusion of CVD, we concluded that the patient was a definite case of MS. MRI of patient revealed multiple CH lesions in brain (Fig. 2).

Although seizure with a prevalence of 3% may occur in MS clinical course, the cause of seizure in this patient was CH. With delineating the cause of seizure, he was treated by immunosuppressive therapy and discharged.

Case 3; A 44-year-old woman with recurrent attacks of weakness and paresthesia in her limbs and history of vertigo, uncontrolled gait and blurred vision since 18 years before referred to our center. She had exacerbation of weakness and spasticity in her lower limbs since 3–4 years before. In neurologic examination, we found spastic paraparesia ($3^{+}/4$), inability to walk without assistance, bilateral Achilles clonus, Babinski's sign and a sensory level at T_{10}. CSF analysis did not have pleocytosis; however, OCB was positive. She was a definite case of MS, with her special clini-



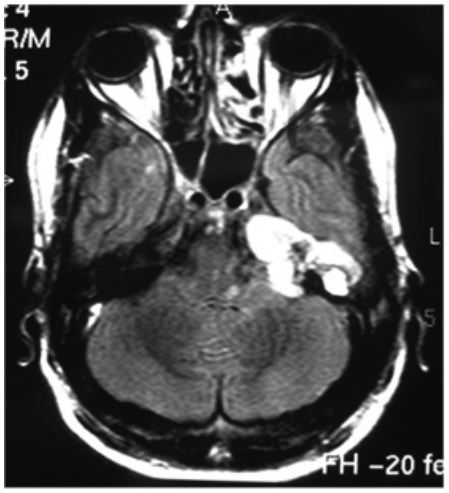


Fig. 1. 30-year old man with progressive weakness and paresthesia in lower limbs since 2 years before. Now, he has left peripheral facial nerve palsy.

A and B. Sagital and coronal T2-W image showing deep white matter hypersignal lesion due to MS plaques.

C. Coronal CT of petrous bone shows extensive destructive changes in left petrous due to cholesteatoma.

D. Axial T1-W image with Gd shows enhancement in the left petrous bone.

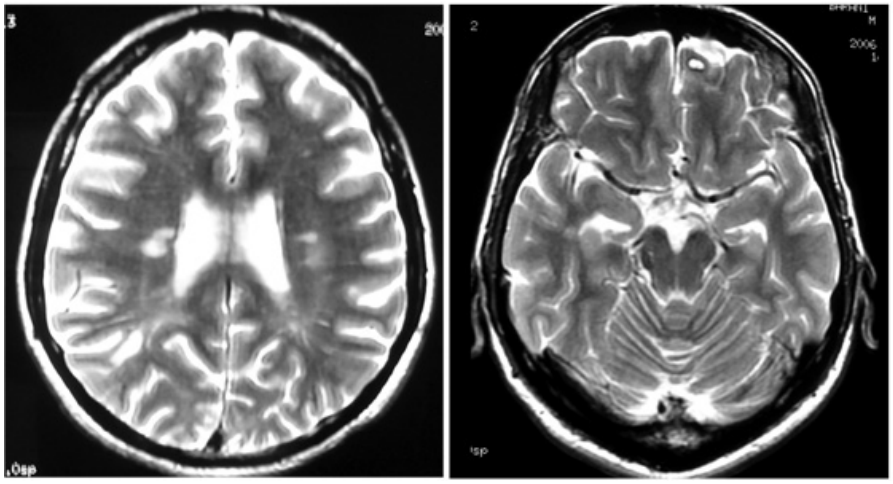


Fig. 2. A,: A 43–year old man, known case of MS, already with presentation of generalized tonic colonic seizure.

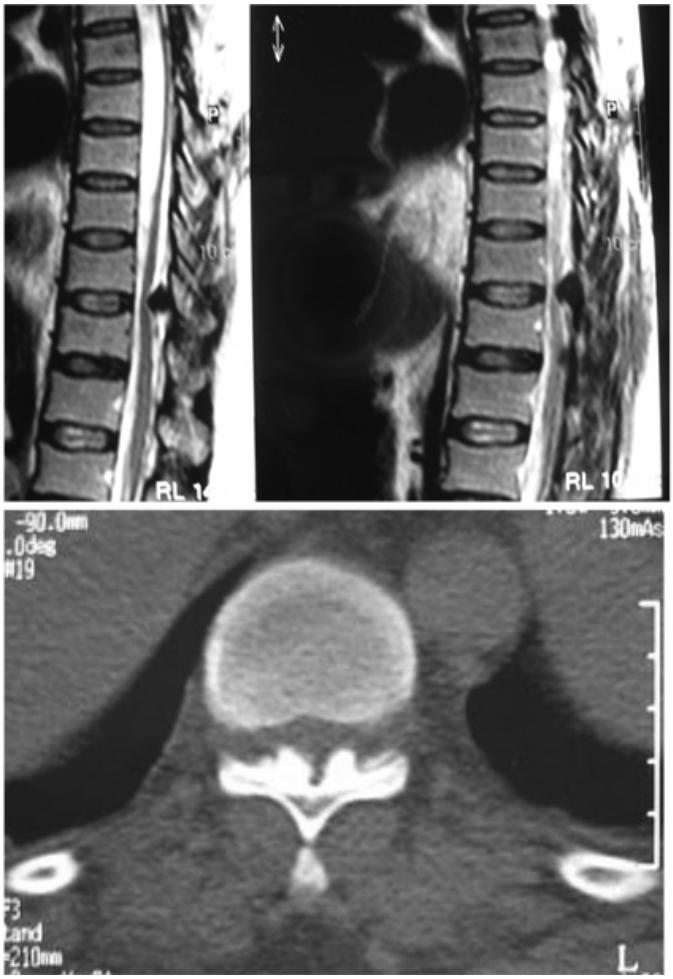
A. Axial T2W image show multiple periventricualr hypersignal lesion.

B. Axial T2W image show a hypersignal lesion surrounding with hyposignal rim in frontal lobe which could be a cavernous hemangioma.

cal course and imaging study (Fig. 3).

In this patient who had more than 18-year history of MS, attribution of paraparesia to MS was the best judgment at first. However, work up for a progressive paraparesia (without recurrent attacks of the lower limbs weakness) with a new MRI from thoracic spine, revealed that ossification of ligamentum flavum (OLF) at T_9-T_{10} level of the spine is indeed the causative factor. Although the patient was in category of SPMS, finding another lesion for explanation of her progressive spastic paraparesia gave her enough hope to continue her MS treatment and to accept receiving appropriate surgical treatment for her OLF.

In conclusion, whenever a patient with MS develops a new signs or symptoms, we should not overlook the necessary paraclinical and radiological evaluations, because, like any persons, other neurological diseases can also occur in those with MS.



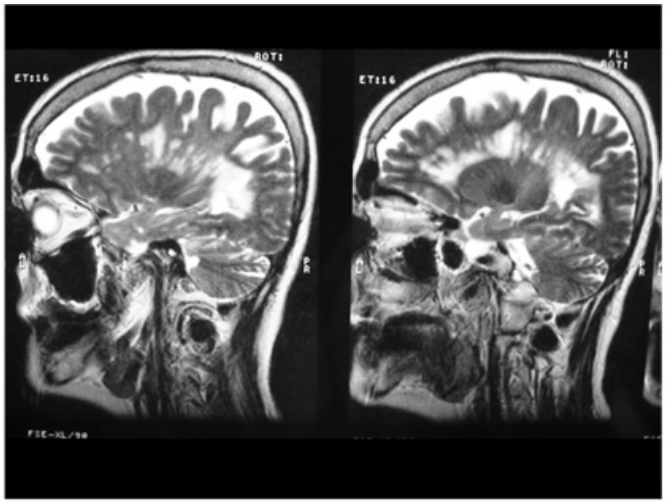


Fig. 3. A 44–year old woman with recurrent attacks of weakness and paresthesia in limbs and history of vertigo, uncontrolled gait and blurred vision since 18 year ago and exacerbation of weakness and spascity of lower limbs from 3-4 years ago.

A, Sagital T2W image of brain show deep white matter hypersignal lesion.

B. Sagital T1W and T2W of thorasic spine show OLF in T9-T10 level with posterior cord compression .

C. Axial CT scan of T9-T10 level show a ossification of ligamentum flavum associated with canal stenosis.

Treatment of this new neurologic disease can seriously affect the insight of our patient to his/her clinical course, management and prognosis.

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