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Multiple Sclerosis Variants Case Series and Literature Review

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1. Abstract

Marburg type, Balo's concentric sclerosis and tumefactive MS represent special variants of multiple sclerosis, with a clinical picture characterized by clinically severe, quickly changing symptoms; also involving unusual neuron pathologic changes. Marburg type often features giant tumor liked demyelination plaques for example.These types recently can be diagnosed *in vivo*, with complementary clinical and laboratory examination and MRI follow up. However, in some patients a favorable clinical course has been described, the worse prognosis of the most patients directs a treatment with intense immunosuppression.

2. Keywords: Balo's concentric MS; Marburg; Tumefactive MS

3. Introduction

Multiple sclerosis is a chronic autoimmune, inflammatory neurological illness of the Central Nervous System (CNS). MS aggression the myelinated axons in the CNS, break down the myelin and the axons to vary degrees sclerosis (MS) [1].

Women are twice as likely to be affected as men, and onset typically occurs between the ages of 20 and 40 years. Many researches have provided that genetic factors is severely related to MS via different gene study [2].

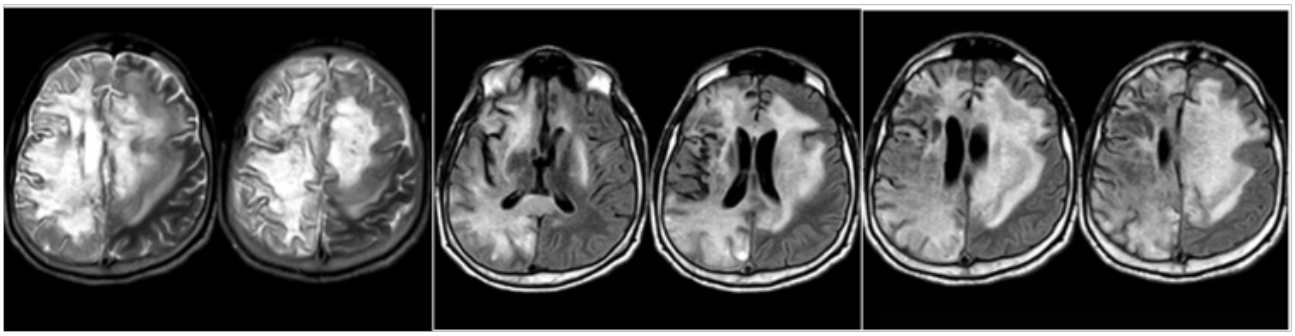
4. Case 1: A Case of Marburg MS

A 30-year-old female patient presented with sudden onset of focal fits, headache and blurring vision. Her GCS was 6/15 with right sided hemiparesis. Laboratory studies were done (Liver, renal functions and CBC) and revealed normal results.

Patient underwent Magnetic Resonance Imaging (MRI) of brain which showed large bilateral ill-defined infiltrative lesions are seen on both sided of the brain affecting centrum semiovale,they display high SI on both T2WI and FLAIR images,after contrast injection they showed no enhancment. The mass associated with moderate edema.The mass and surrounding edema exerted mass effect in the form of compression of related both lateral ventricles (Figure 1).

Cerebrospinal Fluid (CSF) analysis was done which revealed normal sugar and cell count, but slight increase in protein level (85 mg/dl).

A severe fulminant demyelinating disease diagnostic Marburg's type of MS was created based on clinical, radiological, and laboratory examination, as well as the disease's abrupt, severe start and quick development.The patient began taking 1000 mg of methyl prednisolone per day as a high dose. However, there was no improvement after that, the doctor recommended intravenous immunoglobulin (IV Ig), and after one month, the symptoms gradually subsided.



A B C

Figure 1 A,B,C: Axial,T2WI, FLAIR and post contrast MRI revealed a large bilateral ill defined infiltrative lesions are seen affecting centrum semiovale on both sided of the brain,They display high SI on both T2WI and FLAIR images,after contrast injection they showed no enhancment. They associated with mass effect and edema, both of them exerts mass effect in the form of compression of bodies of both related lateral ventricles.

5. Case 2: A Case of Tumefactive MS

A 35-year-old woman presented with blurred vision, numbness, and weakness of the right side. She had no previous medical disorders. Brain MRI was done one week after admission demonstrating hyper intense lesion in the deep periventricular white matter reaching a diameter of approximately 2.5 cm in diameter on T2WI and FLAIR. Gadolinium MRI contrast sequences revealed an open ring-enhancing lesion in the left mesencephalon region facing the cortical surface and the lesion showed restricted diffusion (Figure 2).

MRI findings were suggestive of active demyelinating lesions, and after patient treated with corticosteroid therapy, there were gradual decrease of clinical symptoms.

6. Case 3: A case of concomitant MS and Balo's concentric sclerosis.

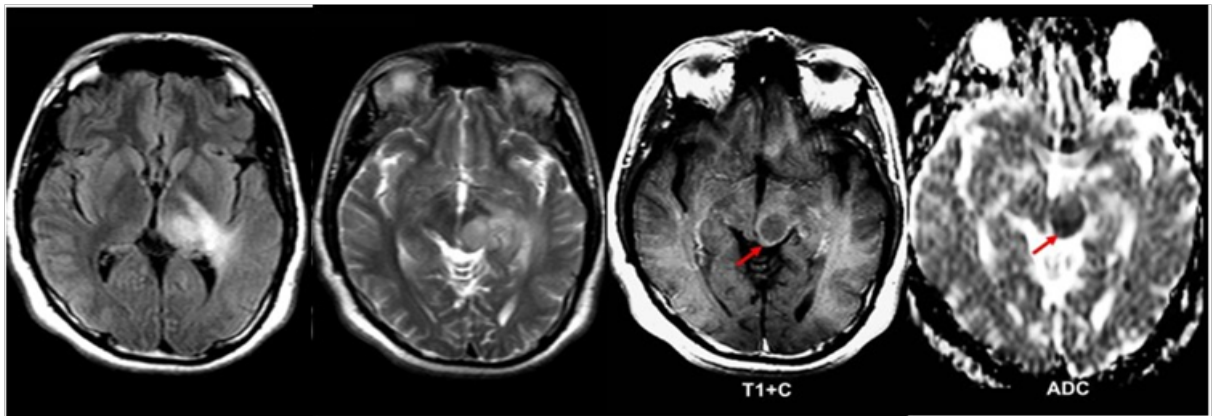
Male patient of 35-year-old presented with focal seizures,loss of concentrations and limited movements. He suffers from hypertension and take concor 5mg/day as a treatment, no other medical disorders were found.

Laboratory studies were done and revealed normal liver, renal functionsand CBC results. CSF analysis was done and revealed positive Oligoclonal Bands (OCBs) and C-reactive protein was normal (<2.50 mg/dl.).

Brain MRI and cervical spine were done, they showed heterogeneous lesion in the left deep white matter of parietal cerebral hemisphere, and displaying alternating iso intense and hypo intense concentric rings on T1WI and high SI on T2WI and FLAIR images with minimal enhancement pattern on the administration of gadolinium contrast with concentric ring features in keeping with BCS (Figure 3 A,B).

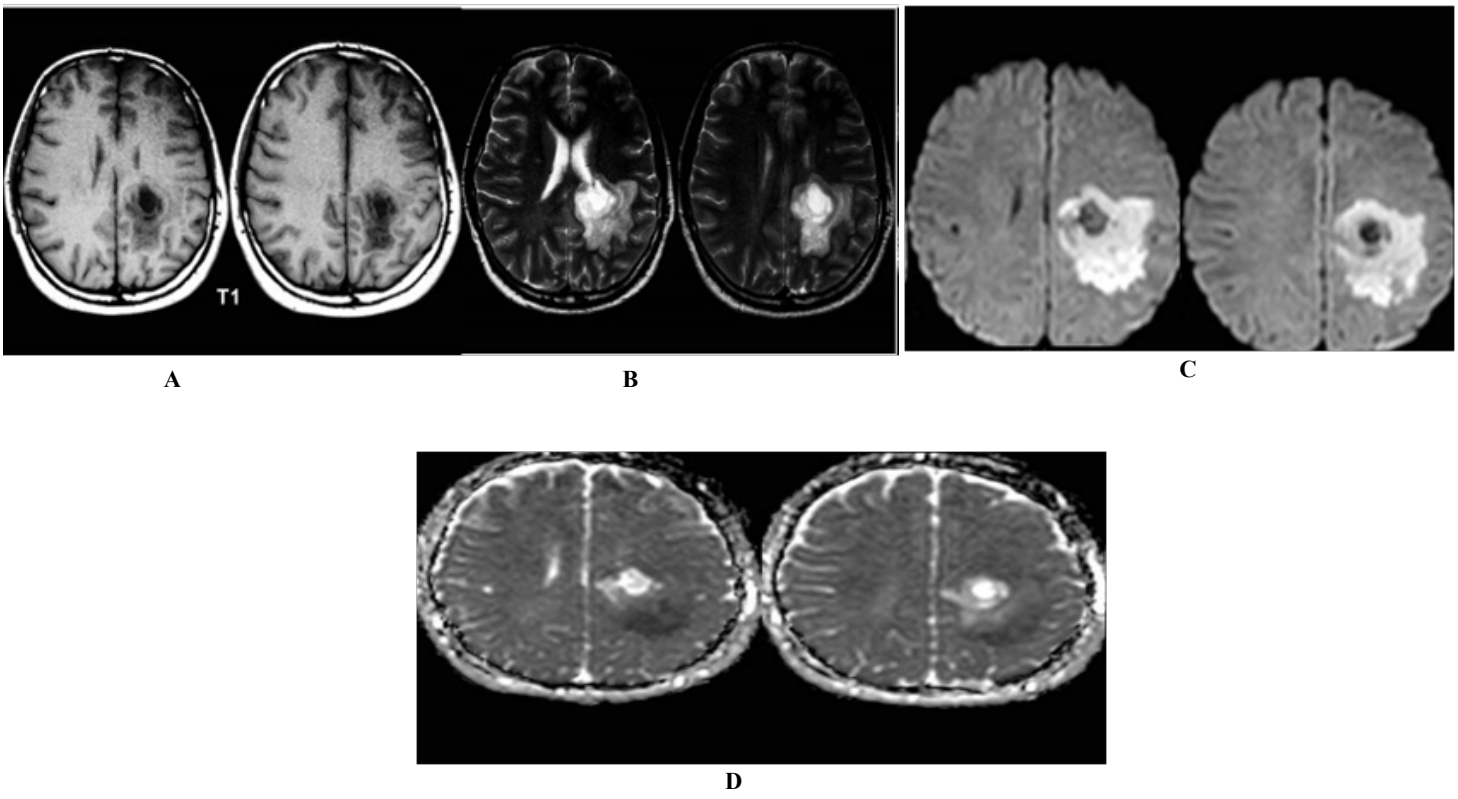
On DWI, it showed high SI on DWI and restricted diffusion on ADC. MR spectroscopy showed elevated choline peak in most of the regions with reduction in the NAA. Cho/Cr ratio was 2.55 (Figure 3C-4).Cervical spine MRI revealed abnormal high SI plaques in short segment of cervical spinal cord on T2WI (Figure 5).

Steroids and immunosuppressive medications were used in combination as a beginning of treatment. High-dosage steroids (intravenous methylprednisolone 1gm daily for 5 days, followed by a maintenance dose of oral prednisolone) were the first course of treatment. After two months, many symptoms gradually improved, but movement limitations persisted. Additionally, follow up MRI imaging showed decrease in the previously described lesion and surrounding edema, but follow up MRI images were not available.



A B C D

Figure 2A,B,C,D: Axial FLAIR,T2WI ,post contrast and ADC MRI respectively revealed a well defined left mesencephalon lesion displays high SI on FLAIR and T2WI,after contrast injection it showed incopmlete ring enhancment and restricted diffusion on ADC. It is associated with edema that exerts mass effect in the form of compression of related left lateral ventricle.



A B C

D

Figure 3A,B,C,D: Axial T1WI,T2WI,DWI and ADC of brain respectively revealed a well-defined left parietal deep white matter lesion measures about 2×2.5×2cm AP × Tr × H respectively. It displays low SI on T1WI and high SI on T2WI associated with mild oedema, after contrast injection it showed minimal enhancementgiving concentric ring appearance(peculiar appearance). DWI and ADC, revealed restricted diffusion.

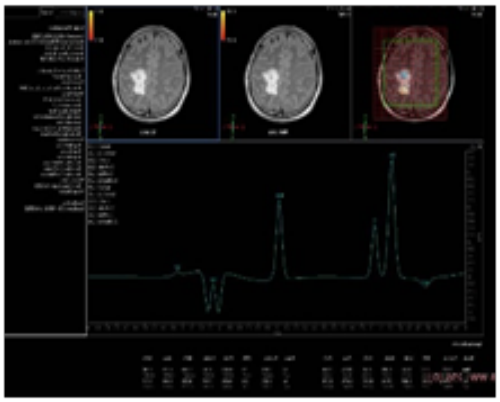
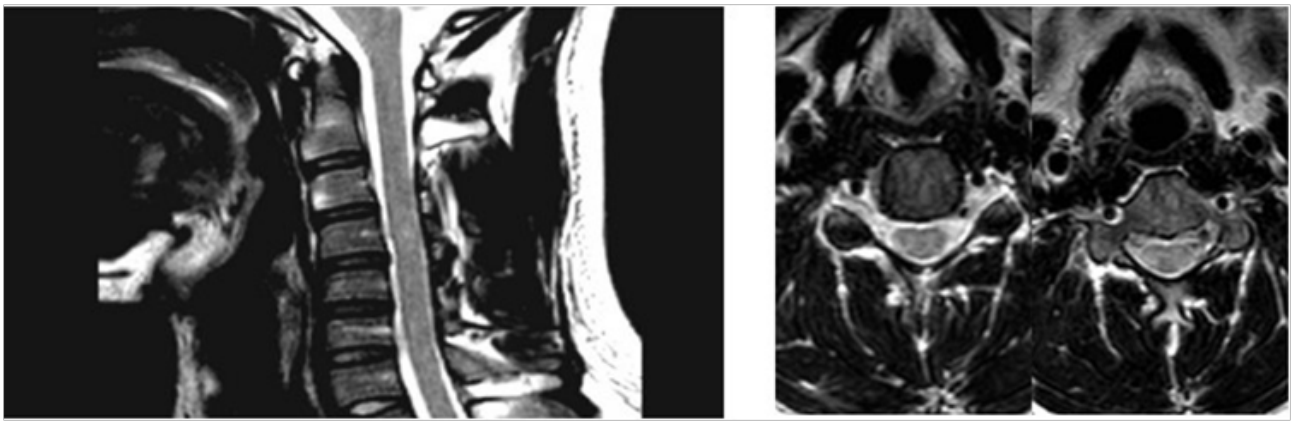


Figure 4: Multi voxel spectroscopic image of the concentric lesion shows increased choline peak and decreased N-acetyl aspartate peak. high Cho/Cr ratio (2.5).



A **B**

Figure 5A,B: Sagittal and Axial T2WI MRI cervical spine revealed abnormal high SI plaques in the short segment of cervical cord.

7. Discussion

Acute MS of the Marburg type may be the classification for Marburg variations, which are rare, inflammatory demyelinating diseases of unknown cause that may be mostly resistant to corticosteroids and cause death or significant worsening. Either acute involvement of the lower brainstem or higher cervical cord, or severe multiple cerebral lesions, might cause death within weeks to months [1].

In our case we described a typical MRI pattern and progression of a Marburg variant of MS. Brain tumor could not be considered because of minimal gadolinium enhancement, whereas grey matter sparing suggested a possible diagnosis of leukodystrophy. However, decrease symptoms after administrations of both corticosteroid and immune suppressive disease confirm the diagnosis of Marburg variants.

It has recently been proposed that a pre-existing anomaly of myelin basic protein in is the cause of Marburg MS. On the other hand, Marburg-type demyelination might just be at the extreme, acute end of the MS clinical spectrum, perhaps due to host immune response-related variables [2]. Regarding to our case after immunosuppressive treatment we found gradual decrease of deteriorated symptoms.

Other forms of multiple sclerosis are known as "tumefactive MS." On MRI, it revealed up as a large intra-parenchymal lesion, usually with a lower mass effect. After gadolinium injection, there could be some peripheral enhancement, usually with an incomplete ring. These lesions can be distinguished from glioma and intra parenchymal abscesses, which typically show a closed-ring enhancement [3-4].

This was consistent with our findings that the diagnosis was confirmed by partial ring enhancement as well as a reduction in symptoms following corticosteroid therapy.

In general, tumefactive MS represents 1-2/1000 of cases of MS with high frequency in adult females.It is defined by the presence of single (frequent) or multiple large sized brain masses (≥ 2.0 cm in diameter) associated with perilesional edema and mass effect. The common clinical presentations of tumefactive MS include headache, cognitive abnormalities, mental confusion, impaired consciousness, aphasia, apraxia, cerebellar symptoms, visual field defects and/or seizures [5].

Balo's concentric sclerosisis characterized by one or more concentric tumefactive-like lesions that result in severe neurologic deteriorations. Initially, Balo's concentric sclerosis was identified as an acute condition. The white matter of the cerebral hemisphere has a distinctive pattern of illness in BCS, which is defined by a concentric ring of alternating bands of white matter with areas of demyelination interspersed with comparatively intact myelination [6].

Although the concentric pattern may also be visible on T1-weighted imaging, BCS lesions are easily recognized on T2-weighted images. Contrast enhancement in BCS has been reported, with bands of enhancement and non-enhancement alternating; the enhancing regions are believed to correlate to demyelination zones [6]. Transient enhancing rings were demonstrated by MRI. Contrast enhancement in BCS has been reported, with bands of enhancement and non-enhancement alternating; the enhancing regions are believed to correlate to demyelination zones [7].

About half of BCS coexists with MS and this may explain the varying expression of oligoclonal bands in CSF of patients with BCS [8-9].This agree with our study there was associations between Balo's concentric sclerosis lesion in brain and MS plaques in cervical cord and this interpreted positive oligoclonal bands in our case.

Other investigations have observed a variety of presentation patterns on MRI, including distorted rings, and even parallel and rectangular bars, even though the typical concentric lamella of demyelinating plaques alternating with areas of myelin preservation is the primary finding of BCS [10-11].

Even though BCS has a benign history, patients should be informed about the possibility of recurrence, which can happen at any point throughout treatment and follow-up. As shown in multiple sclerosis, this can develop de novo from previously uninvolved locations or from a preexisting lesion [12].

8. Conclusion

A history and physical examination that show signs of neurological degeneration are necessary for the clinical diagnosis of multiple sclerosis. Although clinical demonstration of dissemination in time and space is possible, MRI is now frequently utilized to confirm the diagnosis. Other CNS inflammatory diseases such NMOSD, ADEM, and IG antibody-related disease are included in the differential diagnosis of multiple sclerosis. Since each ailment has a different treatment strategy, it is critical to distinguish between them.

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