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Case Report

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Balo's Concentric Sclerosis Mimicking Cerebral Tuberculoma

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Balo's concentric sclerosis (BCS) is considered a rare variant of multiple sclerosis, which often mimics an intracranial neoplasm or abscess. We report the case of a 21-year-old woman presenting with BCS while undergoing treatment for pulmonary tuberculosis. Initial brain magnetic resonance imaging (MRI) findings were similar to those for cerebral tuberculoma, multiple metastases, or abscesses. However, the pathognomonic concentric sclerosis characteristic of BCS was seen on MRI. The antemortem confirmatory diagnosis of BCS was made by follow-up MRI and a brain biopsy. It is suggested that BCS should be included in the differential diagnosis of cerebral tuberculoma, especially in developing countries with a high prevalence of tuberculosis.

Key words: Balo's concentric sclerosis, multiple sclerosis, corticosteroids, cerebral tuberculoma

INTRODUCTION

Balo's concentric sclerosis (BCS) is a rare and recognized variant of multiple sclerosis (MS) [1]. Most cases of BCS show acute encephalopathy with a fulminant course that can rapidly become fatal. However, the advent of magnetic resonance imaging (MRI) has changed this, and long-term survival has been reported with BCS [1-3]. Imaging features pathognomonic for BCS include alternating rings of demyelinated and myelinated white matter seen on T2-weighted images and concentric ring enhancement on T1-weighted images with gadolinium enhancement [1, 4]. In the earlier stage, many other neurological diseases can mimic BCS because it presents with diverse features and brain MRI findings are not prominent in that period. In Korea, there has been at least one case report, which described proton magnetic resonance spectroscopy (1H-MRS) findings similar to those in multiple sclerosis [5]. Here, we describe the case of a woman who presented with BCS while undergoing therapy for pulmonary tuberculosis.

CASE REPORT

A 24-year-old woman presented with dysarthria and bilateral upper extremity weakness for 3 days. She did not complain of headache, dizziness, nausea, fever, or chills. She had been taking anti-tuberculous medication for pulmonary tuberculosis and tuberculous lymphadenitis over the prior 2 months. On neurological examination, she was alert and well oriented. Cranial nerve examination revealed central type facial palsy of the left side and dysarthria; other cranial nerve functions were normal. The muscle power of the right and left upper extremities were both Medical Research Council (MRC) grade 4. The muscle power of the lower extremities was normal. Deep tendon reflexes were hyperactive in the bilateral upper extremities, without definite

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pathological reflexes. Sensory function and autonomic nervous system functions, including micturition and defecation, were normal.

Screening tests for infectious diseases including serum antibodies to human immunodeficiency virus, herpes simplex virus type 1 and 2, varicella zoster virus (VZV), Epstein-Barr virus, cytomegalovirus,

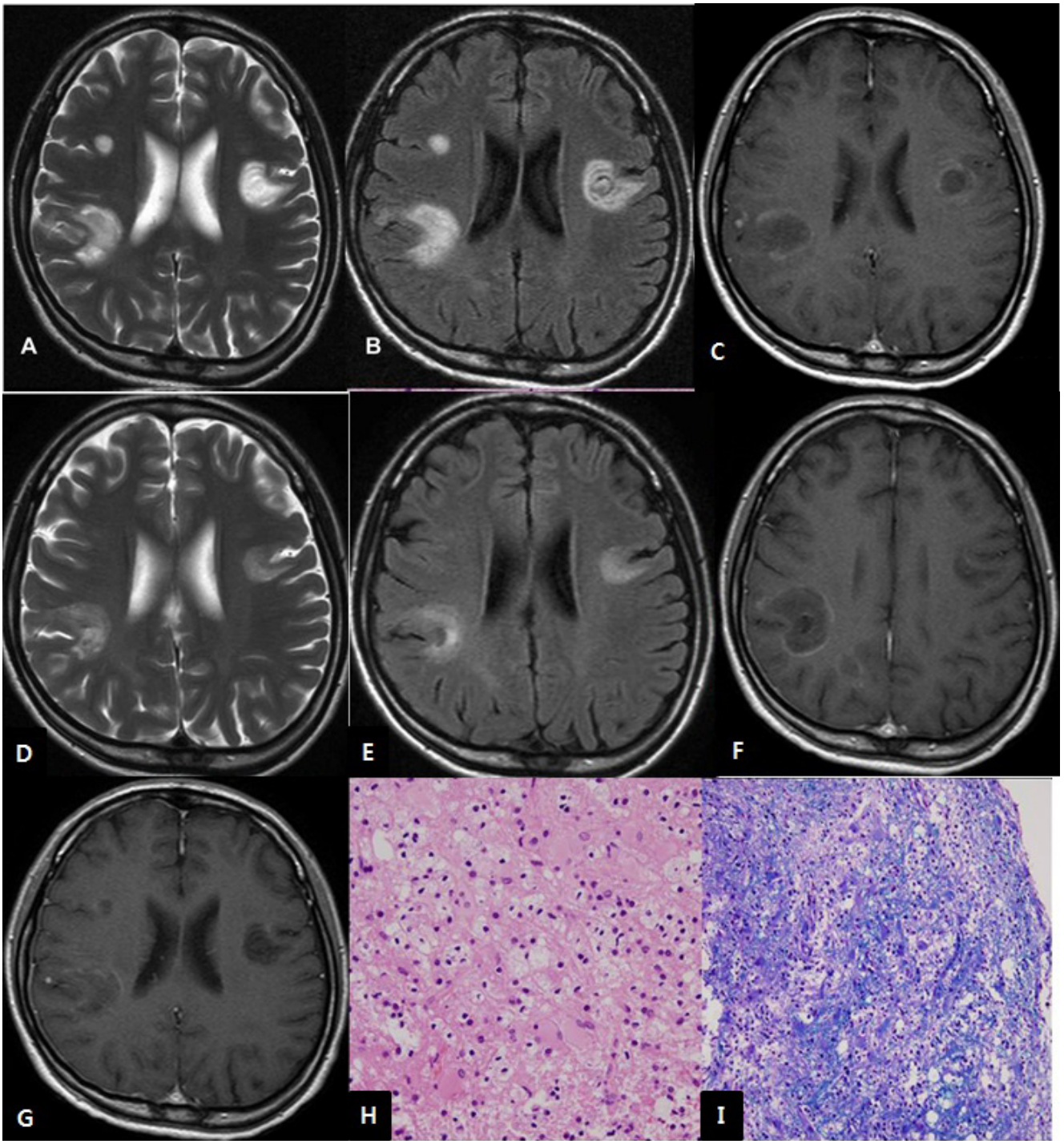

Fig. 1. Brain MRI. (A~C) Three weeks after the onset of symptoms, T2-weighted (A), FLAIR (B) and enhanced T1W1 (C) images show a more distinctive lamellate pattern. (D~G) Five months after the initial presentation, T2-weighted (D), FLAIR (E), and enhanced T1W1 (F, G) images show that the size of the lesions is d`iminished and the lamellate pattern has become indistinct. (H, I) Microscopic examination of brain biopsy, showing heavy infiltration of foamy histiocytes with some lymphocytes (H) (Hematoxylin & Eosin staining, ×400). Luxol fast blue staining reveals scattered loss of the myelin sheath (I) (×100). These findings are suggestive of a demyelinating disease.

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Borrelia burgdorferi, and *Toxoplasma gondii* were unremarkable. The adenosine deaminase level in serum was 21 IU/L (normal range: 5.3~17.8 IU/L). Other routine serological tests were normal. Cerebrospinal fluid (CSF) examination showed mild protein elevation (88.3 mg/dL) without pleocytosis. There were no oligoclonal bands in the CSF, and the IgG index was 0.41. No acid-fast bacillus (AFB) was isolated. Visual evoked potentials, brainstem auditory evoked potentials, and somatosensory evoked potentials were normal.

Initial brain MRI revealed multiple mass-like lesions with ring enhancement and peripheral edema. Some lesions showed multiple concentric rings with high signal intensity around them on T2-weighted and fluid-attenuated inversion recovery (FLAIR) images. However, these findings were not prominent (Fig. 1A, 1B). A stereotactic brain biopsy revealed findings suggestive of a demyelinating disease (Fig. 1H, 1I). The staining of brain tissue for AFB was negative. There was no evidence of neoplasm, granuloma, or abscess. A nested PCR test for Mycobacterium tuberculosis in the biopsied tissue was negative. With intravenous dexamethasone treatment (16 mg/day for 18 days), neurological deficits were normalized. The multiple lamellate high signal lesions on T2-weighted and FLAIR images became more prominent in repeat brain MR imaging performed 3 weeks later, though the, peripheral edema and gadolinium enhancement were decreased. This lamellate concentric pattern disappeared in repeat brain MRI performed 5 months later (Fig. 1D, 1E). No new lesions were detected. There were not any lesions in the brainstem and the spine MRI was not under consideration. The patient did not experience any symptoms or signs of relapse, and neurological deficits were not detected during the follow-up examination 51 months later.

DISCUSSION

BCS is a rare demyelinating disease. Its histopathological hallmark is an alternating lamellar pattern of demyelinated and well-preserved white matter [1]. Traditionally, the diagnosis of BCS is made by postmortem pathological features, and the clinical course of BCS frequently presents as an acute or subacute encephalopathy. Therefore, BCS has been considered fulminant and rapidly fatal. With the advent of MRI techniques, however, early diagnoses can be made, and treatments can be initiated. Thus, some cases of BCS have recently been reported to have a good therapeutic outcome [1-4, 6, 7]. MRI findings of BCS show concentric ring patterns on T2-weighted images with enhancement on T1-weighted images, and these findings reflect pathological features of BCS that are characterized by concentric lesions of alternating demyelinated and myelinated bands in the white matter [6]. More recently, an MRS study was performed. Proton spectroscopy may show a high choline peak and a low N-acetylaspartate peak, similar to acute MS plaques [5]. However, the concentric pattern is not always observed if the MR imaging is not performed early in the course of the disease [8]. Patients usually present with symptoms of acute or subacute onset, which progress over a period of weeks to months, suggesting a space-occupying lesion in the brain. Clinical differential diagnoses includes neoplasms, abscesses, sarcoidosis, and cerebral tuberculoma [4]. Although cerebral tuberculoma is a rare condition, it is highly prevalent in developing countries. Tuberculosis with CNS involvement accounts for 10~15% of all tuberculous infections [9].

In the present case, the patient had pulmonary tuberculosis and was treated with anti-tuberculous medication. The clinical presentation of weakness and sensory changes led to the initial suspicion of a cerebral tuberculoma. However, MRI showed multiple mass lesions with ring enhancements and a subtle lamellate pattern in some of them. Therefore, a stereotactic brain biopsy was needed for the differential diagnosis, and it revealed findings suggestive of a demyelinating disease without evidence of a tuberculous granuloma, sarcoidosis, abscess, or neoplasm. The diagnosis of BCS was finally made based on these typical MR and pathological findings. The follow-up MR images acquired 3 weeks later at the same level showed a more prominent lamellate pattern. It was thought that cytotoxic edema was decreased and thus the outer demyelination became more prominent.

In conclusion, it is suggested that BCS should be included in the differential diagnosis of cerebral tuberculoma in developing countries with a high prevalence of tuberculosis, especially when brain MRI shows findings atypical for tuberculoma.

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