

Multiple Central Nervous System Lesions Associated With Rheumatoid Arthritis

A 51-YEAR-OLD WOMAN suffering from 20 years of rheumatoid arthritis (RA) had noticed difficulties in walking 2 months earlier. Neurological examinations of the patient disclosed hyperactive deep-tendon reflexes, abnormal bilateral reflexes, and motor weakness of her left upper and lower extremities. Blood analysis demonstrated a highly elevated rheumatoid factor (241 IU/mL), and anticardiolipin antibody was not found. Examination of the cerebrospinal fluid (CSF) demonstrated pleocytosis (205 cells/mm³), and the CSF protein level was markedly elevated to 316 mg/dL. The patient's cranial T2-weighted (Figure 1A) and flair magnetic resonance imaging (MRI) (Figure 1B) investigations revealed high-signal lesions. These abnormalities were revealed as slight T1 hypointensity signals similar to those of the gray matter without any gadolinium-diethylenetriaminepentaacetic acid enhancement. Magnetic resonance angiography did not demonstrate any vascular changes. Cervical MRI showed high-signal lesions in the T2-weighted images (Figure 1C). Based on these findings, we diagnosed the patient as suffering from encephalomyelopathy associated with RA. After the steroid treatment, the CSF abnormalities have resolved and most of the

abnormal signal intensities on MRI have disappeared (Figure 2). The patient's clinical manifestations also showed marked improvement.

COMMENT

The MRI abnormalities exhibited a symmetric distribution and appeared to have the tendency to involve and spread through the white matter bundles. The patient's mild neurological impairment suggested that these abnormal MRI signals did not necessarily represent a destruction of neural transmission such as demyelination or axonal damage, but instead probably indicated reversible changes such as vasogenic edema. Diffusion-weighted MRI examinations did not show any signal changes, and therefore cytotoxic edema was unlikely. Demyelinating disorders were also considered unlikely, because both the oligoclonal band and the myelin basic protein tested negative, and gadolinium-MRI did not demonstrate apparent parenchymal enhancement. All of these results strongly supported the conclusion that vasogenic edema was the major pathological deterioration in our case. Central nervous system involvement in RA is actually rare,^{1,2} even though meningeal involvement with pachymeningitis has occasionally been reported. In conclusion, this is an extremely rare case of encephalomyelopathy associated with active RA.

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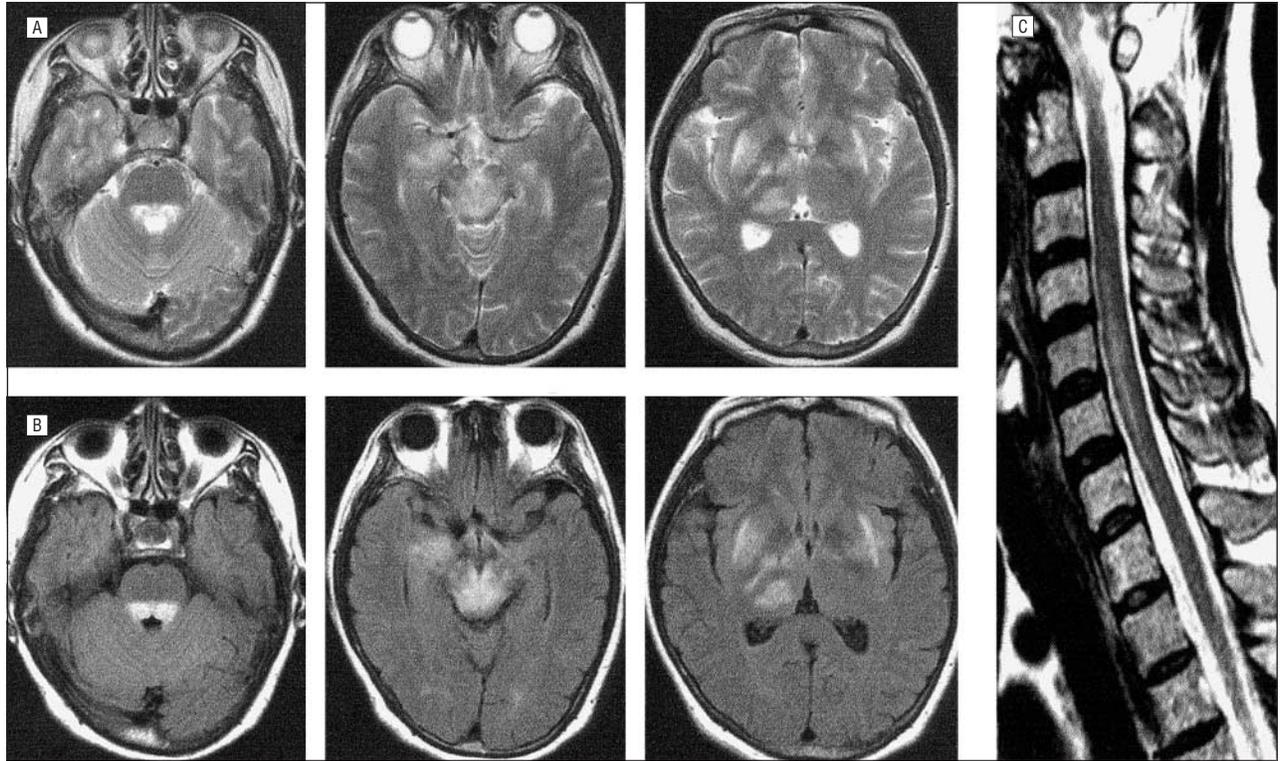


Figure 1. T2-weighted (A) and flair high-signal intensities (B) widely involved right thalamus, bilateral putamen, internal capsules, external capsule, midbrain brain, and dorsal part of pons. C, T2 high-signal intensity area was revealed at the C4-C7 level with slight cord swelling.

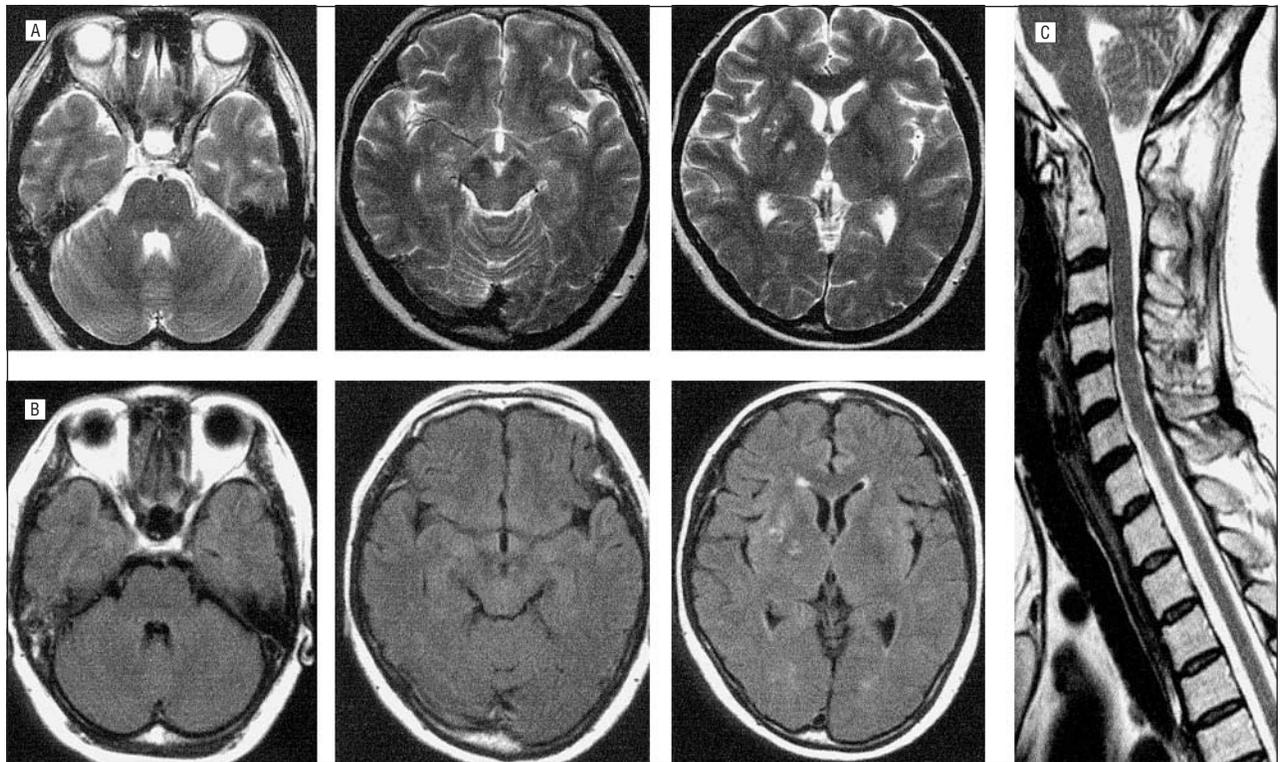


Figure 2. After the steroid treatment, most of the T2 (A) and flair (B) high-signal lesions have disappeared. C, Previous T2 high-signal lesions in the cervical cord have also disappeared.