

Reversibility of Acute Demyelinating Lesions in relapsing-remitting Multiple Sclerosis

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Multiple Sclerosis (MS) is a demyelinating disease of the central nervous system (CNS) of presumed autoimmune etiology and characterized by a relapsing-remitting course¹. Brain magnetic resonance imaging (MRI) has emerged as the most sensitive investigation to detect demyelination in MS^{2,3}. Furthermore, acute relapses of MS have been associated with breakdown of the blood-brain barrier (BBB)⁴ seen as contrast enhancement on brain MR(T1-weighted images⁵. Recently, we encountered a case of an acute relapse in a patient with known MS. Treatment with high-dose intravenous methylprednisolone (IVMP) resulted in clinical recovery as well as resolution of brain MRI abnormalities. This patient represents a striking case of complete reversibility of acute demyelinating lesions seen on brain MRI scans.

Case Report

A 30 year old woman with a history of relapsing-remitting MS for several years presented with acute dysarthria, weakness, difficulty in walking and headache of four hours duration. Prior to the onset of symptoms she had been stable for several months. Her only medication was baclofen. Vital signs were within normal limits and general physical examination was also normal. Neurological examination was pertinent for mild dysarthria, right hemiparesis, hyperreflexia (more prominent on the right) with bilateral Babinski signs and spastic mild vibratory loss in both distal lower extremities and impaired coordination on the right. She required assistance to ambulate. She was admitted with the diagnosis of acute relapse of MS. Blood counts and routine chemistries were normal. A brain MRI scan with and without contrast was obtained. A well defined area of increased signal intensity adjacent to the anterior horn of left lateral ventricle on axial T2-weighted image was seen (figure 1A)

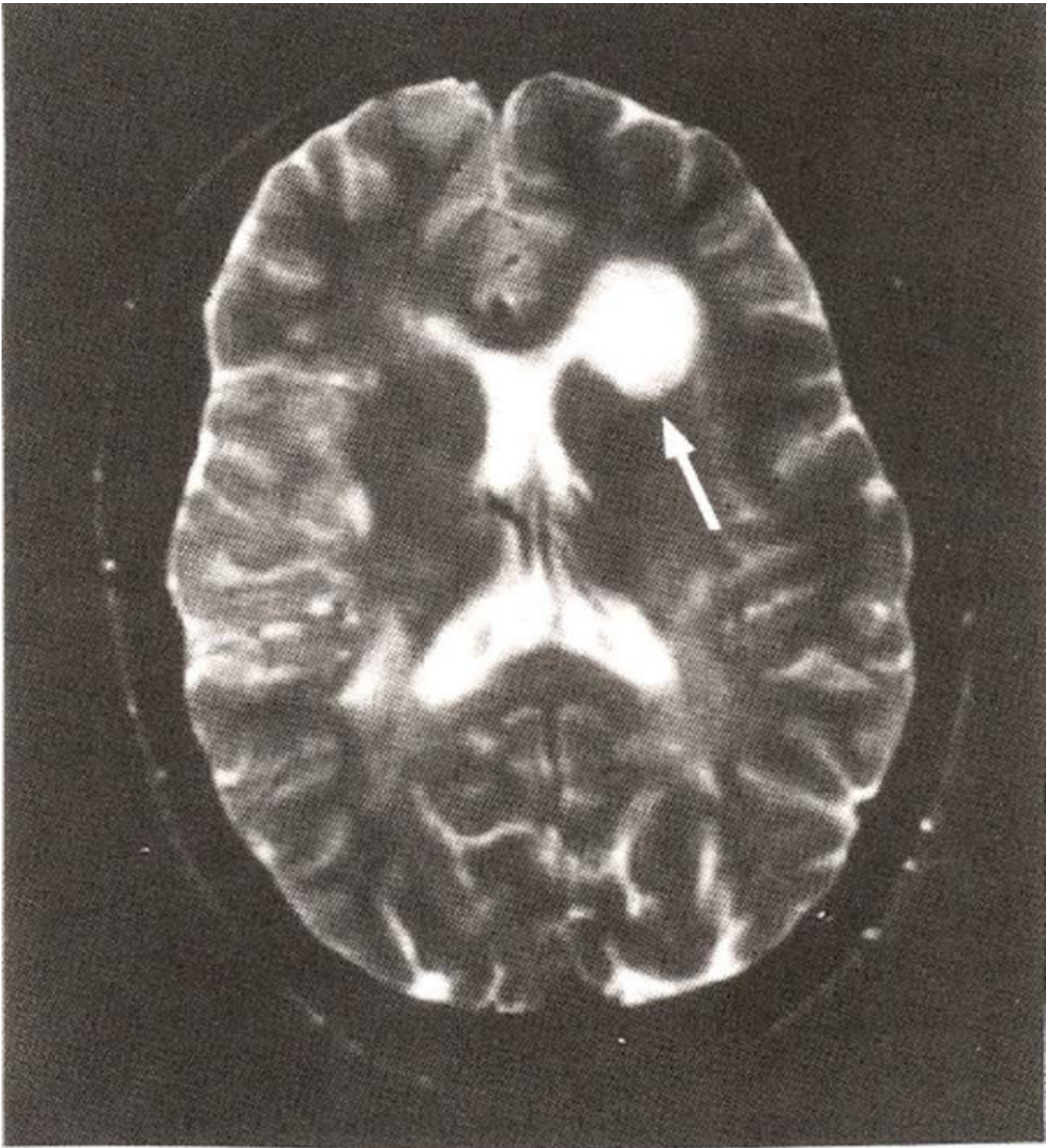


Figure 1A: Brain MRI showing a well defined area of high signal intensity (arrow) demonstrating a ring-like enhancement (arrow).
with a ring like contrast enhancement seen on axial T1 -weighted image (figure 1 B).

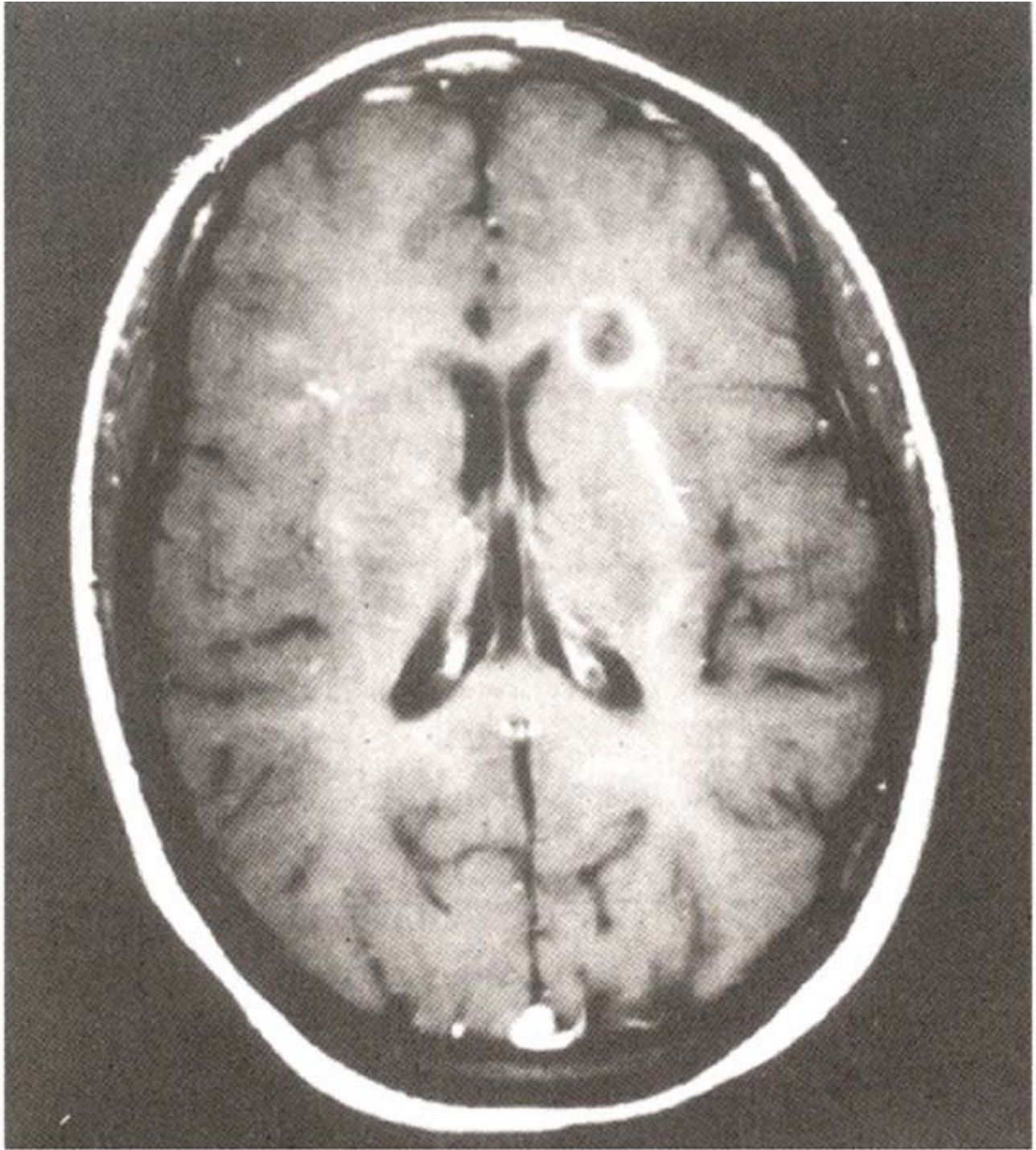


Figure 1B: Brain MRI showing a well defined area of high signal intensity (arrow) demonstrating a ring-like enhancement (arrow)

Treatment with IVMP was instituted at one gram a day for five days followed by a brief taper of oral prednisone. The patient had complete clinical recovery one week after the onset of treatment. A follow up brain MRI scan was obtained two weeks after the first scan. There was significantly diminished signal intensity seen on axial T-2 weighted image (figure 2A)

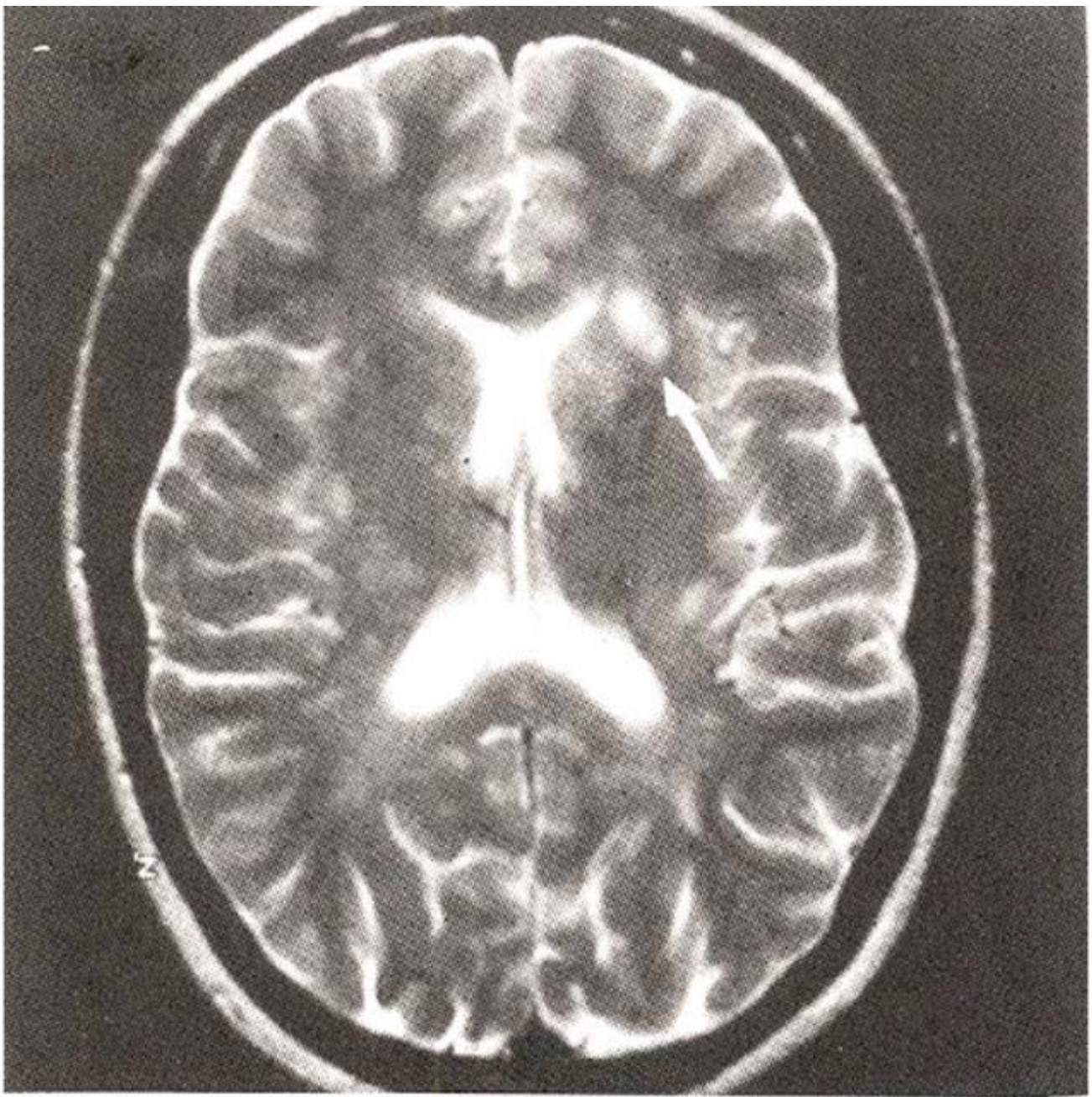


Figure 2A. Two weeks later, brain MRI reveals considerably diminished signal intensity (arrow) without any enhancement (arrow).

without any enhancement on axial T-1 weighted image (figure 2B).

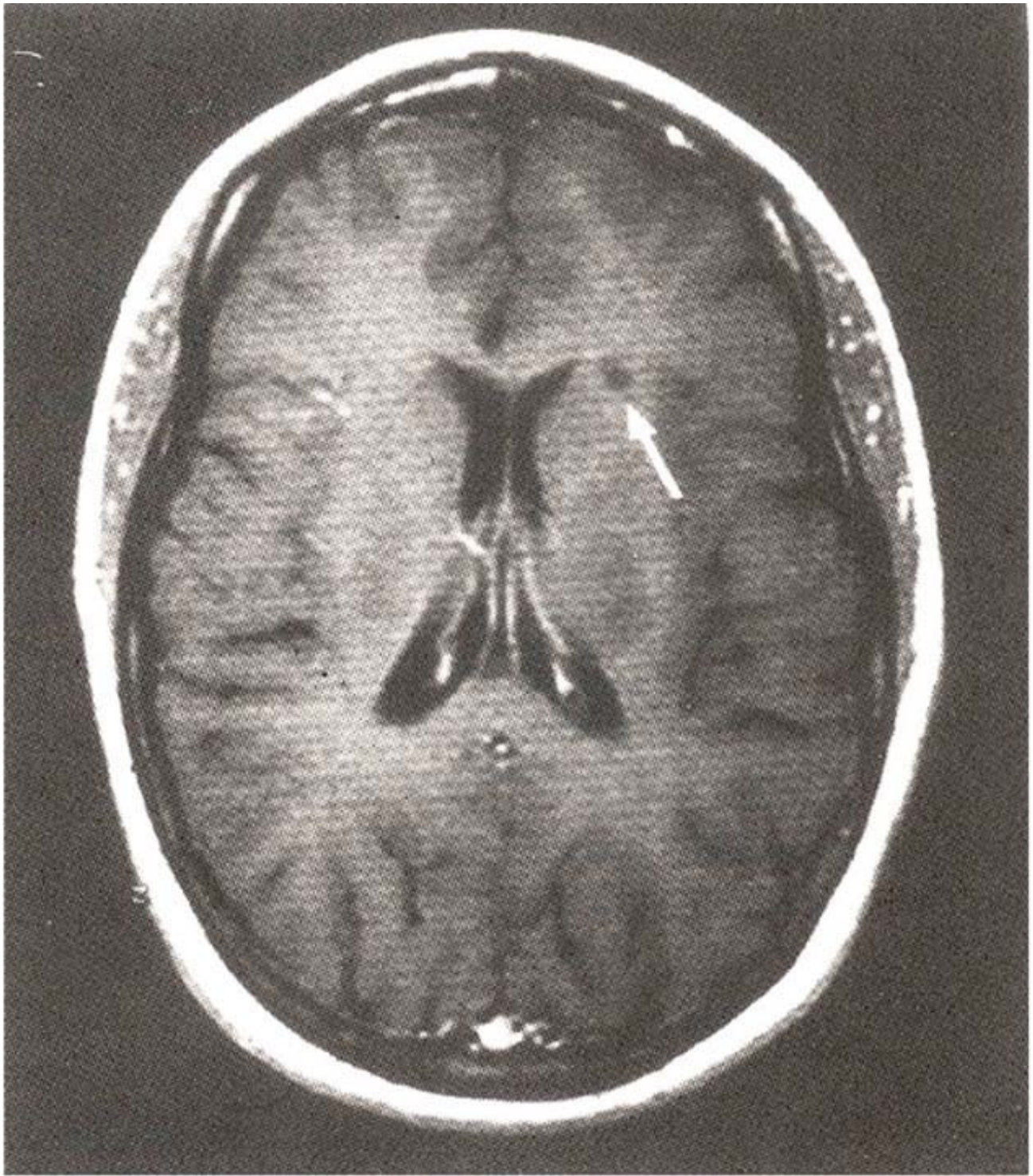


Figure 2B: Two weeks later, brain MRI reveals considerably diminished signal intensity (arrow) without any enhancement (arrow).

The patient was discharged and continues to do well one year later.

Discussion

This case highlights the MRI changes seen during an acute demyelinating event associated with clinical

worsening as well as alteration of the BBB⁶. The role of brain MRI in detecting acute demyelination is well established² Although this patient had well established MS and had been followed at our clinic for several years, other diagnostic considerations in a patient with no previous medical history, should include cerebral abscess, primary or secondary brain tumor, toxoplasmosis, parasitic cyst, neuroborreliosis, neurosarcoidosis and tuberculosis⁷. Thus, careful consideration should be paid to the differential diagnosis of MRI abnormalities seen during an acute neurological event and follow up studies should be obtained if deemed appropriate in order to confirm the initial diagnosis. Treatment with high dose IVMP is generally considered to be the treatment of choice for an acute relapse of MS⁸. The effect on brain MRI lesions including contrast enhancement is variable though clinical recovery usually ensues⁹. Most studies show that NMP suppresses contrast enhancement in acute demyelinating lesions¹⁰ This effect is attributed primarily to decreased permeability of the BBB^{12,13}. Although, one could argue that such resolution of MRI abnormalities could also represent the natural course of relapsing—remitting MS. However, radiological resolution of acute MS plaques typically occurs over a period of at least 6 to 8 weeks¹⁴. This case highlights the abnormalities which may be seen on brain MRI during an acute relapse of MS. Furthermore, treatment with high—dose IVMP confirmed temporal correlation between clinical improvement and reversal of acute MRI changes including suppression of contrast enhancement.

References

1. McAlpine D, Compston ND, Acheson ED. Multiple sclerosis: a reappraisal, 2nd ed. London: Churchill Livingstone, 1972:214.
2. Ormerod IEC, Miller DH, McDonald WI, et al. The role of MRI in the assessment of MS and isolated neurological lesions: a quantitative study. *Brain*, 1987;110:1579-1616.
3. Miller DH. MRI: Sensitive and safe in diagnosing MS. *MRI Decisions*, 1988;2: 7-24.
4. McFarland H, Frank JA, Albert PS, et al. Using gadolinium—enhanced magnetic resonance imaging lesions to monitor disease activity in multiple sclerosis. *Ann. Neurol.*, 1992;32:758-66.
5. Smith ME, Stone LA, Albert PA, et al. Clinical worsening in MS associated with increased frequency and area of gadopentetate dimeglumine enhancing magnetic resonance imaging lesions. *Ann. Neurol.*, 1993;33 :480-89.
6. Gonzalez-Scarano F, Grossman RI, Galetta S, et al. Multiple sclerosis disease activity correlates with gadolinium-enhanced magnetic resonance imaging. *Ann. Neurol.*, 1987;21 :300-6.
7. Nelson AG: Diagnostic Neuroradiology. St. Louis, Mosby, 1994.
8. Durelli L, Cocito D, Riceio A, et al. High—dose intravenous methylprednisolone versus ACTH in the treatment of multiple sclerosis: clinical-immunologic correlations. *Neurology*, 1986;36:238-43.
9. Milligan NM, Newcombe R, Compston DAS. A double-blind controlled trial of high dose methylprednisolone in patients with multiple sclerosis: 1 Clinical effects. *J. Neurol. Neurosurg. Psychiatry*, 1987;50:511-16.
10. Burnham JA, Wright RR, Dreisbach J, et al, The effect of high-dose steroids on MRI gadolinium enhancement in acute demyelinating lesions. *Neurology*. 1991;41:1349-54.
11. Trotter JL, Garvey WE. Prolonged effects of large-dose methylprednisolone infusion in multiple sclerosis. *Neurology*, 1980;30:702-8.
12. Kermode AG, Tofts PS, Thompson AJ, et al. Heterogeneity of blood—brain barrier changes in multiple sclerosis: an MRI study with gadolinium-DTPA enhancement. *Neurology*, 1990;40:229-35.
13. Miller DH, Thompson AJ, Morrissey SP, et al. High-dose steroids in acute relapses of multiple sclerosis: MRI evidence for a possible mechanism of therapeutic effect. *J. Neurol. Neurosurg. Psychiatry*, 1992;55:450-53.

14. Capra R. Gadolinium-pentetic acid MRI in patients with relapsing—remitting MS. *Arch, Neurol.*, 1992;49:687-89.