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F J Lexa and R I Grossman

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MR of Sarcoidosis in the Head and Spine: Spectrum of Manifestations and Radiographic Response to Steroid Therapy

Frank J. Lexa and Robert I. Grossman

PURPOSE: To evaluate the role of MR in the diagnosis and treatment of patients with neurosarcoidosis. **METHODS:** The MR studies of 24 patients who satisfied stringent criteria for the diagnosis of sarcoid were retrospectively reviewed. All patients had signs and symptoms referable to the head and/or spine. The majority, 17 patients (71% of the total), were examined at least once with gadolinium enhancement. Fifteen of 24 patients (63%) underwent serial examinations during steroid therapy. **RESULTS:** A wide spectrum of findings was noted: white matter and periventricular high signal intensity on long-repetition-time/long-echo-time sequences, mimicking multiple sclerosis (11 patients); leptomeningeal enhancement (11 patients); brain parenchymal mass (seven patients)—six demonstrated enhancement, one did not receive contrast; lacrimal gland mass (three patients); hydrocephalus (three patients); enlarged ventricles, apparently atrophic (one patient); periventricular enhancement (three patients); extraaxial mass, mimicking meningioma (two patients); chiasmal enhancement or swelling (one patient); enhancing nerve roots (two patients); enlarged pituitary stalk (two patients); pontine infarct (one patient); and enhancing parenchymal spinal cord mass (three patients). Partial or complete resolution of the radiographic abnormality occurred in 13 of 15 cases (87%), which paralleled clinical improvement. No response was detected in the remaining two. Abnormal enhancement was the finding that was most responsive to steroid therapy, with response seen in nine of 10 patients with leptomeningeal enhancement, in six of six patients with enhancing brain parenchymal masses, in three of three patients with enhancing cord masses, and in all three patients with periventricular enhancement. **CONCLUSIONS:** 1) MR shows a spectrum of protean central nervous system abnormalities associated with neurosarcoidosis. 2) This high sensitivity for associated abnormalities aids in differentiating central nervous system sarcoid from the many diseases that it can mimic. In particular, enhancement was a useful clue to the diagnosis in 15 of 17 cases in which it was used (88%). 3) MR demonstrates regression of central nervous system abnormalities during steroid therapy, in particular abnormal meningeal, periventricular, and parenchymal enhancement.

Index terms: Sarcoidosis; Head, magnetic resonance; Spine, magnetic resonance; Steroids

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Sarcoid is a multisystem disease of unknown cause with a worldwide distribution. In the North American population it disproportionately afflicts patients of West African descent. There is a female predominance. Sarcoid is usually diagnosed between the ages of 20 and 40 years (1–3). Although the reported incidence is highly variable, a large series using mass screening by chest radiography gave an estimated incidence of 20 per 100 000 (4). Involvement of virtually every organ system has been reported. Boeck is credited for the initial description of the disease based on the dermatologic manifestations of sarcoid with histologic correlation (5). In 1905, Winkler described the first reported case of central nervous system (CNS) sarcoidosis (6–8). Clinical CNS involvement is estimated to occur in approximately 5% of cases (1, 2) with some smaller subseries reporting higher rates up to 16% (3). In a large series, approximately 3% of patients had clinical symptoms of CNS disease; of those who

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From the Neuroradiology Section, Department of Radiology, Hospital of the University of Pennsylvania, Philadelphia.

Address reprint requests to Frank J Lexa, MD, Neuroradiology Section, Department of Radiology, Hospital of the University of Pennsylvania, 34th and Spruce Streets, Philadelphia, PA 19104.

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died and came to autopsy, 14% had pathologically proved CNS involvement (9). Another autopsy study reported a 16% rate of CNS involvement (10), again implying a significant rate of subclinical CNS disease.

Computed tomography and magnetic resonance (MR) have a primary role in the detection of CNS involvement (11–20). The use of contrast enhancement in computed tomography further increases sensitivity and has documented response to steroid therapy (18–20). Gadopentetate dimeglumine enhancement during MR seems to be very sensitive for detection of meningeal and parenchymal involvement (21–30).

The wide spectrum of potential findings in patients with CNS sarcoidosis makes this unusual entity a mimic of many more common diseases of the CNS. This retrospective review of the MR findings in 24 documented cases of CNS sarcoid emphasizes the diverse spectrum of CNS findings that this disease may demonstrate on MR and the responsiveness of these findings to steroid therapy in a subgroup examined with serial MR studies.

Materials and Methods

We reviewed MR studies of patients with proved CNS sarcoid. All MR was done at 1.5 T. Enhancement was performed with intravenous gadopentate dimeglumine (0.1 mmol/kg) (Berlex, Wayne, NJ). A spin-echo sequence used short-repetition-time (TR) sequences (400–800/10–30/2 [TR/echo time (TE)/excitations]) before and after gadolinium. Section thickness varied from 3 to 5 mm with a skip from 0 (interleaved) to 2.5 mm. The long-TR sequences used a multiecho technique with 2000–3000/20–35,80–90/0.5–2, with 5-mm-thick sections and 0- to 2.5-mm skip.

Because the diagnosis of neurologic sarcoidosis demands two criteria, a compatible clinical picture of a multisystem disease and histologic confirmation of sarcoid tissue (31), the following criteria were applied in order to avoid inclusion of unproved cases or cases in which a confounding additional disease process could explain the findings: 1) We required histologic proof of sarcoid involvement either directly in the CNS when available or in another organ system. Lymph node biopsy was the usual confirmatory source of tissue. 2) Positive chest radiographic findings, gallium scanning, angiotensin converting enzyme levels, and Kveim testing were used as supportive evidence. 3) All patients included in this report had both clinical symptoms and radiographic findings referable to the CNS involvement. 4) Any additional known disease processes that could explain the findings were grounds for exclusion. 5) Patients who did not meet all of the above criteria were excluded. Because the majority of patients had serial examinations during therapy, the response to treatment with steroids could be evaluated. The clinical response to therapy and the improvement or resolution of radiographic findings could be compared. All of the index studies were reinterpreted by two neuroradiologists in a consensus, nonblinded fashion in order to confirm the reported findings.

Results

Twenty-four patients with proved CNS sarcoid underwent a total of 65 MR examinations at this institution over 7 years. There were 15 women and 9 men. Sixteen of 24 were of West African ancestry (67%). Ages at initial MR examination ranged from 21 to 75 years with a mean of 44 years, the latter reflecting probably both an age bias from our referral population and the greater age of patients with long-standing involvement. Four patients had MR findings limited to the spinal cord; two had combined findings in the brain and cord; and the remainder had findings limited to the head. Seventeen had at least one gadolinium-enhanced examination. Fifteen of the 24 (63%) had serial imaging during steroid therapy.

The spectrum of MR findings in our patient cohort is given in Table 1. The most common abnormality seen was abnormal high signal intensity on long-TR sequences in the periventricular regions and deep white matter (Figs 1 and 2),

TABLE 1: Spectrum of MR findings in 24 patients with CNS sarcoid

MR Finding	Number of Patients with Finding
White matter, periventricular and periaqueductal high signal on long-TR/long-TE sequences	11
Leptomeningeal enhancement	11
Brain parenchyma enhancing mass	6
Brain parenchyma mass, no contrast administered	1
Pontine infarct	1
Hydrocephalus	3
Enlarged ventricles, atrophic	1
Periventricular enhancement	3
Lacrimal mass	3
Extraaxial mass	2
Enlarged pituitary stalk	2 (one with enhancement)
Chiasmal enhancement or swelling	1
Masticator mass	1
Enhancing, clumped nerve roots	2
Enhancing parenchymal cord mass and hyperintensity on long-TR/long-TE sequences	3
Parenchymal cord mass, no contrast administered	1

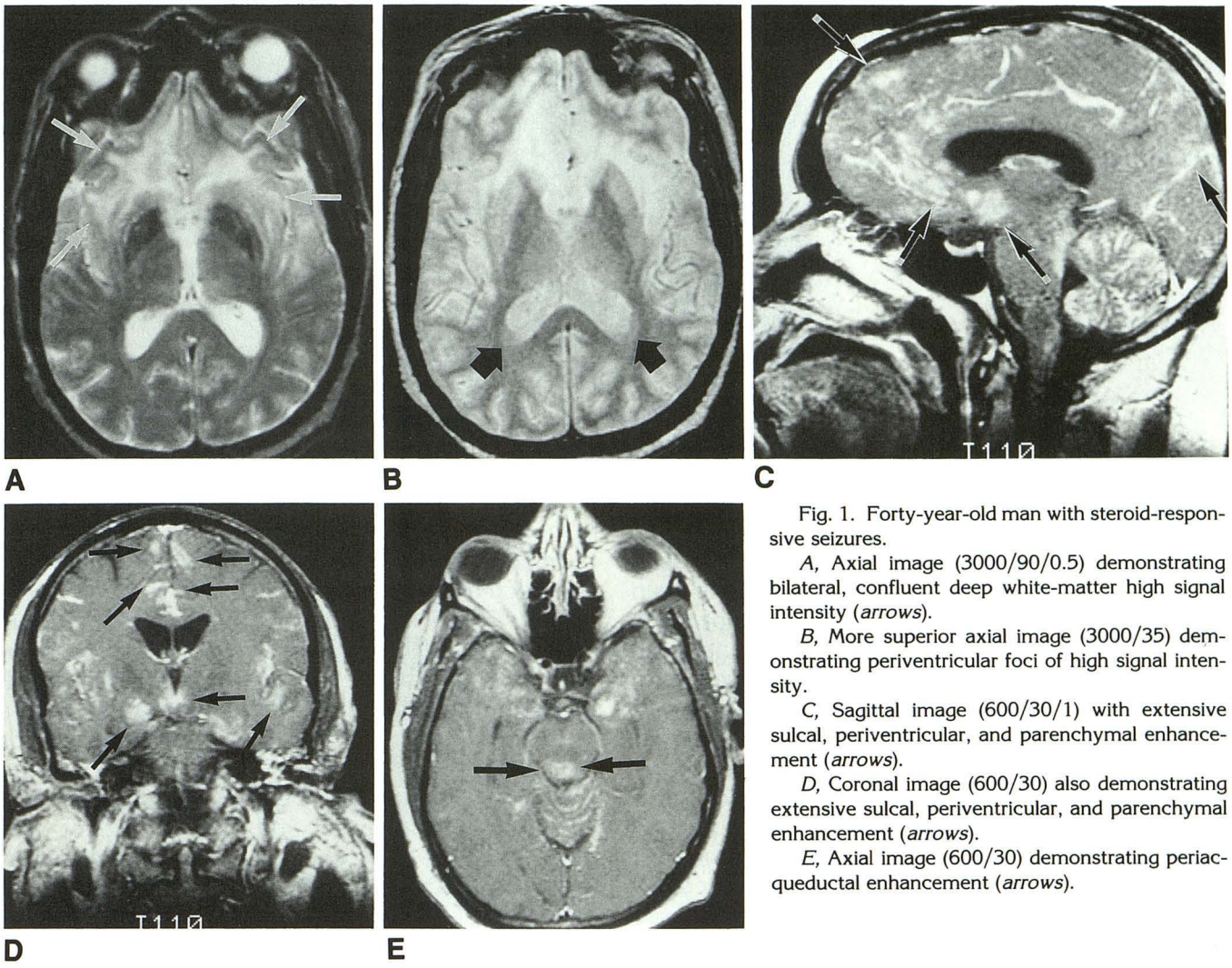


Fig. 1. Forty-year-old man with steroid-responsive seizures.

A, Axial image (3000/90/0.5) demonstrating bilateral, confluent deep white-matter high signal intensity (*arrows*).

B, More superior axial image (3000/35) demonstrating periventricular foci of high signal intensity.

C, Sagittal image (600/30/1) with extensive sulcal, periventricular, and parenchymal enhancement (*arrows*).

D, Coronal image (600/30) also demonstrating extensive sulcal, periventricular, and parenchymal enhancement (*arrows*).

E, Axial image (600/30) demonstrating periaqueductal enhancement (*arrows*).

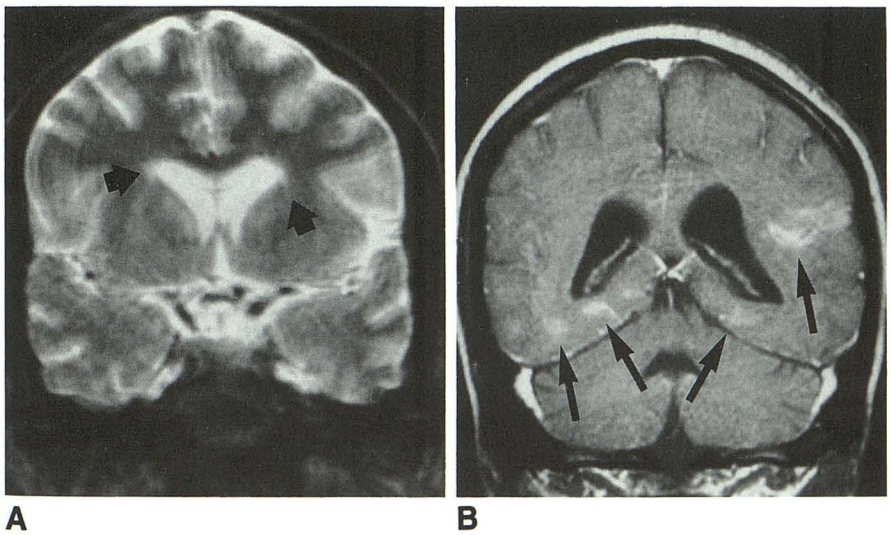


Fig. 2. Twenty-seven-year-old man with fluctuating mental status, steroid-responsive.

A, Coronal image (3000/90/1) showing periventricular high-signal-intensity foci.

B, Coronal enhanced image (700/30/1) demonstrating deep parenchymal enhancement (*arrows*).

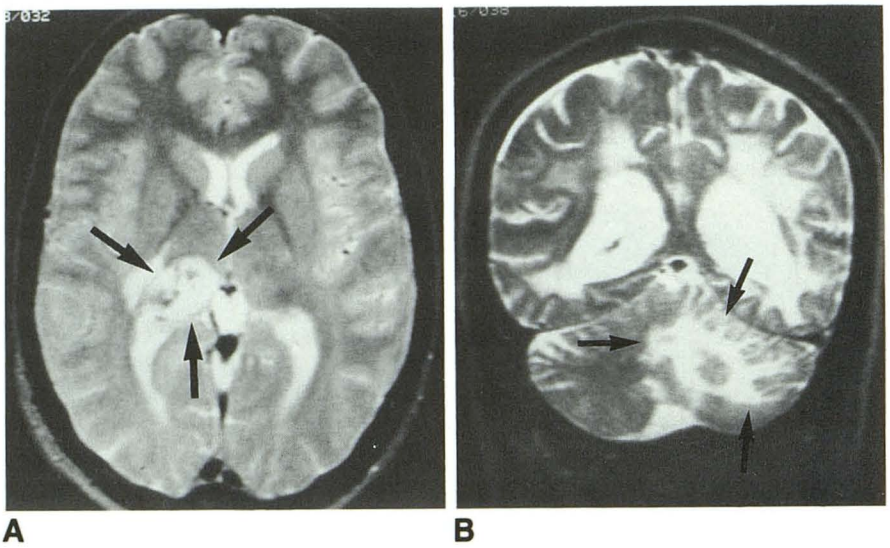


Fig. 3. *A*, Twenty-year-old woman with weakness. Axial (2500/80/2) image shows a parenchymal mass in the right hemisphere adjacent to the ventricular surface (*arrows*).

B, Sixty-year-old man with a 30-year history of sarcoid now presenting with dysarthria, change in mental status, and weakness. Coronal (3000/80/2) image demonstrates a left cerebellar mass (*arrows*).

seen in 11 of the 22 patients who had diagnostic brain studies (50%), simulating the lesions seen in multiple sclerosis. Eleven of the 17 patients (65%) who received gadolinium demonstrated significant meningeal enhancement, often with associated cortical enhancement (Fig 2), and in one case with an associated infarct in the subjacent pons.

The next most common abnormality was a brain parenchymal mass (Figs 3A and 3B). Of the seven patients who had parenchymal masses, six received gadolinium; all masses enhanced in a patchy, irregular fashion. Hydrocephalus was seen in three patients with one demonstrating acqueductal stenosis without evidence of a mass, one showing periaqueductal abnormal high signal intensity on long-TR/long-TE sequences with enhancement, and the last demonstrating communicating hydrocephalus. One patient had increased ventricular volume with an appearance suggesting atrophic change rather than obstruction.

Periventricular enhancement was noted in three patients. Two patients had intracranial, extraaxial masses that mimicked meningiomas (Figs 4 and 5); three patients had lacrimal masses. Two patients demonstrated enlargement of the pituitary stalk (Fig 6). One patient had enlargement of the optic chiasm. Two demonstrated enhancing, clumped nerve roots (Fig 7). Three had enhancing spinal cord masses; a fourth had a spinal cord mass but did not have a contrast-enhanced examination.

Gadolinium enhancement provided useful clues to the diagnosis in 15 of the 17 patients in which it was used (88%). Leptomeningeal and additional areas of parenchymal involvement not suspected from the noncontrast images were seen. These occurred along Virchow-Robin spaces and deep parenchyma and in a periventricular distribution. The circumstances in which it did not provide additional utility were: 1) in a patient with focal meningeal involvement (Figs 4A and 4B), in whom enhancement was noted but did not alter the preoperative impression of a potential meningioma; and 2) in a patient who was scanned after receiving intensive steroid therapy, in whom patchy white matter lesions were seen on the precontrast images, but no abnormal enhancement was seen.

In a subset of 15 patients who were followed serially with MR examinations while on steroid therapy (Table 2), there was a similar distribution of abnormalities. The majority of patients exhibited more than one abnormality. Partial or complete resolution of one of the principle abnormalities during steroid therapy was seen in 13 of the 15 (87%). Five of 13 demonstrated a mixed response with clearing of one abnormality; another was unresponsive. Two patients showed no evidence of improvement in any of the detected abnormalities.

As in the larger group of patients, abnormal leptomeningeal enhancement and high-intensity signal foci on long-TR/long-TE sequences in white matter and in periventricular regions were the two most common abnormal findings, with leptomeningeal enhancement seen in 10 of 15 patients (67%) and abnormal white matter and periventricular signal seen in nine of 15 (60%). The abnormal meningeal enhancement showed a response to steroids with partial or complete

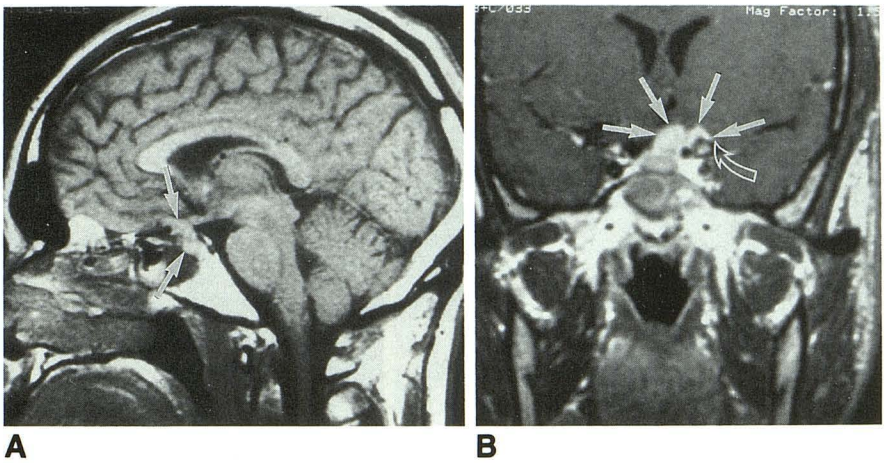


Fig. 4. Thirty-two-year-old man with acute loss of vision in the left eye.

A, Sagittal (600/10/1) image showing an extraaxial mass simulating a planum sphenoidale meningioma (*arrows*).

B, Coronal enhanced image (600/26) showing superior and lateral extent of the mass (*closed arrows*) with development of a dural "tail" sign (*open arrow*).

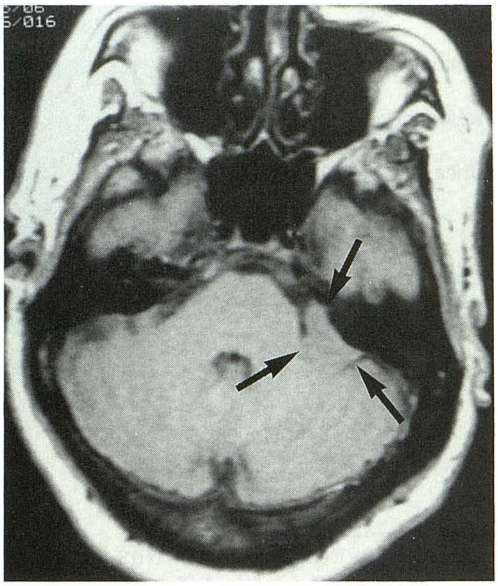


Fig. 5. Sixty-four-year-old woman. Axial image (600/20/1) of extraaxial mass simulating cerebellopontine angle meningioma (*arrows*).

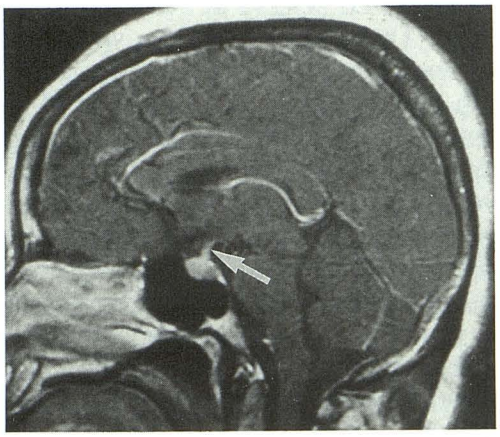


Fig. 6. Fifty-year-old woman. Sagittal enhanced (600/30/2) image showing enlarged, enhancing pituitary stalk (*arrow*).

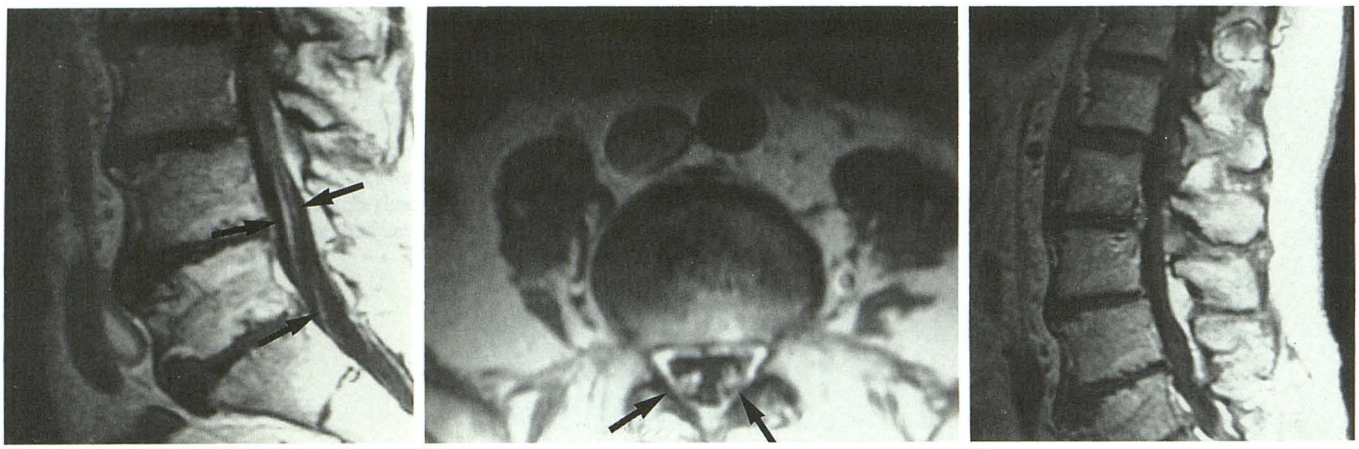
resolution in nine of 10 patients (90%). Periventricular enhancement was seen in three patients; all showed a steroid response with two returning to normal and one decreasing in magnitude. Conversely, the abnormal signal intensity on T2-weighted sequences in white matter and periventricular tissue was unresponsive to steroid treatment in seven of nine patients observed (78%). One patient showed complete resolution, and the remaining patient demonstrated some reduction in the magnitude of the finding. In three patients with cord enhancement and abnormal signal intensity on long-TR/long-TE images, the enhancement responded to steroid therapy in all three, but the abnormal signal intensity showed a response in only one. The pontine infarct did not change during steroid therapy.

In the subgroup that was followed serially, brain parenchymal enhancing masses were seen in six patients. All of these showed a response, with four completely resolving and two showing definite improvement. The enlarged optic chiasm returned to normal on steroids. Two patients with abnormal nerve root enhancement were followed; both showed partial resolution (Figs 7A-7D).

In the course of our review, we also encountered seven patients with proved systemic sarcoid and neurological symptoms attributed to sarcoid, but who had negative MR studies (three performed with gadolinium enhancement and four without).

Discussion

CNS sarcoid remains a difficult diagnosis because of its rarity and protean manifestations. Historical reports and reports using modern neuroimaging techniques have emphasized a variety



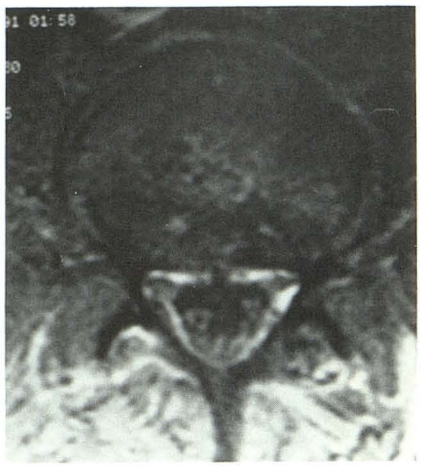


Fig. 7. Sixty-five-year-old man.

A, Pretreatment sagittal enhanced image (400/12/1) demonstrating extensive enhancement of the lumbosacral nerve roots (*arrows*).

B, Pretreatment axial enhanced image (600/12) demonstrating extensive enhancement of the lumbosacral nerve roots (*arrows*).

C, Postcontrast sagittal image (800/12) after steroid therapy showing reduction in enhancement.

D, Axial enhanced posttreatment image (800/20) showing reduced enhancement.

of presentations of neurosarcoid, including leptomeningitis, mass lesions either parenchymal or extraaxial, hydrocephalus, atrophy, cranial neuropathy, chiasmal syndrome, hypothalamic/pituitary dysfunction, spinal cord parenchymal mass, cauda equina syndrome, lumbosacral nerve root mass, and granulomatous angiitis (2, 3, 7, 9, 11–77).

Our experience documents a similar wide spectrum of MR abnormalities in sarcoid of the head and spine, especially white matter disease, periaqueductal involvement, and abnormal enhancement of CNS parenchyma and meninges. Depending on the presentation, this makes sarcoid an especially important differential consideration with regard to multiple sclerosis, meningioma, tuberculous meningitis, normal pressure hydrocephalus, and vasculitis. Multiple sclerosis remains a particular problem given the age of the cohort affected and the similarity of the white matter lesions (12, 14, 15), which constituted one of the two most common abnormalities identified in this series (average age of this subgroup was 45 years, not significantly different than the overall study group). Enhancement patterns were useful for distinguishing sarcoid from multiple sclerosis (Figs 1 and 2).

Of the patients in this report, only one (Fig 1) had documented seizure activity as a presenting symptom. This patient's course was particularly severe whenever steroids were tapered, consistent with the report by Krumholtz et al (78) that seizures are associated with a more progressive form of the disease.

Several reviews have reported the utility of gadopentetate dimeglumine for making the diagnosis of neurosarcoid (21–30). Leptomeningeal enhancement in the MR study aids in the discrimination of sarcoid from multiple sclerosis (25). In a similar manner, leptomeningeal enhancement and periventricular or white matter disease also seem to be useful ancillary findings when an intraaxial or extraaxial mass is discovered, as was the case in 13 of 24 patients. Our experience

TABLE 2: Response of MR findings to steroid therapy in 15 patients followed with serial imaging

Abnormality	Number Observed	Number Showing A Response	Number Showing No Response
Leptomeningeal enhancement	10	9 responded, with complete resolution in 4, and 5 showing reduced abnormal enhancement	1
White matter and periventricular high signal	9	2 (1 completely resolved, 1 partially resolved)	7
Brain parenchyma enhancing mass	6	All six responded, with complete resolution in 4 and 2 showing decreased size and abnormal enhancement	0
Hydrocephalus	2	1 showed improvement on steroids, the second improved but was also shunted in the same interval making the response equivocal	0
Periventricular enhancement	3	2 completely resolved, 1 showed a significant decrease	0
Chiasmal swelling and enhancement	1	1, completely resolved	0
Enhancing nerve roots	2	2, both of which showed a significant reduction	0
Enhancing spinal cord masses	3	3, enhancement resolved in one and showed significant reduction in the other two. The abnormality on long-TR/long-TE images, however, showed only slight improvement in 1 of 3	0
Abnormal high signal in cord on long TR, enhancement unknown	1	0	1
Pontine infarct	1	0	1

differs from some other reports in that leptomeningeal enhancement is not limited to the basal meninges but clearly can be a more diffuse process. One of the patients with an enhancing extraaxial mass showed a "dural tail sign," consistent with the experience of others (71) that this sign can be found in a variety of dural-based processes. Eleven of 24 patients (46%) had intraaxial masses of the neuraxis, simulating primary or secondary neoplasia. The nature of enhancement reported in the literature for parenchymal masses has varied from homogeneous to the patchy irregular pattern seen in this series. In all of these lesions more than one focus of abnormality was seen. In addition, all of these patients had ancillary findings of leptomeningeal enhancement, long-TR/long-TE signal abnormalities, or both, which should help suggest the diagnosis of sarcoid.

Overall, in 15 of 17 patients who received intravenous gadolinium the enhanced images provided additional information that supported the diagnosis of sarcoid. The two exceptions involved a patient in which an area of meningeal involvement remained the solitary abnormality on the postcontrast images (Figs 4A and 4B) and a patient treated intensively with steroids before enhanced MR imaging who had abnormalities on the precontrast images but no abnormal enhancement.

In the four patients with ventricular enlargement, it was possible to suggest sarcoid as a cause in three on the basis of ancillary findings. The remaining patient had aqueductal stenosis, did not receive gadolinium, and had no identifiable additional findings. Two patients in this study had extraaxial masses that were indistinguishable from meningiomas, one simulating a planum sphenoidale meningioma, the other a meningioma of the cerebellopontine angle.

We encountered seven sarcoid patients with neurologic symptoms referable to the CNS who had normal MR studies (three with enhancement,

four without). Although this is a small group, it is interesting to consider what factors may lead to this situation. All had well-documented sarcoidosis before their first MR examinations. Several had been treated with steroids before that first examination because the diagnosis of neurosarcoid had been confirmed. This was not the case with many of the other patients reported here, in that for many the definitive diagnosis of sarcoid was not made until after they had had at least their first MR examinations. Given our observations regarding the responsiveness of many MR abnormalities, one possibility is that treatment before MR imaging may have led to a false-negative MR appearance, especially on the enhanced studies. Second, gadolinium enhancement was necessary for demonstrating abnormalities in many of the cases reported here; this may in part explain the lack of findings in the four unenhanced examinations. Last, the possibility exists that even under ideal circumstances there may be symptomatic lesions for which current MR technology remains insensitive. This is supported by the report of Miller et al (12) showing that three of 21 symptomatic patients examined at 0.5 T (noncontrast) had normal studies.

Finally, we were able to document steroid response, confirming the experience of others (17, 21, 23, 27–29, 40, 62, 66) with angiography, nuclear scanning, computed tomography, and MR. Steroids remain the mainstay of therapy (79), and MR provides an excellent means for assessing therapeutic response. Abnormal enhancement of the leptomeninges and parenchymal and periventricular/subependymal regions seems to be a useful marker of response to therapy, with partial or complete resolution in all but one of our cases. Conversely, abnormalities of the deep white matter and periventricular regions that are seen on long-TR/long-TE sequences seem to be insensitive to steroid effects in the patients studied, with only one patient showing complete clearing, one showing partial improvement, and the remaining seven showing no improvement. This may reflect a distribution of gadolinium into areas of active inflammation that are both steroid responsive and have some potential for reversibility. However, nonenhancing areas that are abnormal on long-TR/long-TE imaging may reflect regions of permanent damage without active inflammation, and therefore little potential for reversal. The two cases studied that did show some response may have had a component of reversible disease such as edema.

In conclusion: 1) MR is capable of showing a spectrum of protean abnormalities in CNS sarcoid. 2) Detection of associated abnormalities may allow discrimination of sarcoid from the gamut of diseases that it mimics. In particular, enhanced imaging proved useful in 88% of patients in which it was used. 3) MR is capable of showing response to steroid therapy for CNS sarcoid.

References

1. Siltzbach LE, James DG, Neville E, et al. Course and prognosis of sarcoidosis around the world. *Am J Med* 1974;57:847–852
2. Delaney P. Neurologic manifestations in sarcoidosis: review of the literature with a report of 23 cases. *Ann Intern Med* 1977;87:336–345
3. Mayock RL, Bertrand P, Morrison LE, Scott JH. Manifestations of sarcoidosis. *Am J Med* 1963;35:67–89
4. James DG, Neville E, Castairs LS. Bone and joint sarcoidosis. *Semin Arthitis Rheum* 1976;6:53–81
5. Boeck C. Multiple benign sarkoid of the skin. *J Cutan Genitourinary Dis* 1899;17:543–550
6. Winkler M. Beitrag aur grage der sarkoide (Boeck). *Arch Dermatol Syph* 1905;77:3–24
7. Walker AG. Sarcoidosis of the brain and spinal cord. *Postgrad Med J* 1961;37:431–436
8. Salvesen HA. The sarcoid of Boeck, a disease of importance to internal medicine. *Acta Medica Scandinavica* 1935;86:127–151
9. Ricker W, Clark M. Sarcoidosis: a clinicopathologic review of three hundred cases, including twenty-two autopsies. *Am J Clin Pathol* 1949;19:725–749
10. Aszkanazy CL. Sarcoidosis of the central nervous system. *J Neuropathol Exp Neurol* 1952;11:392–400
11. Lewis R, Wilson J, Smith FW. Diabetes insipidus secondary to intracranial sarcoidosis confirmed by low-field magnetic resonance imaging. *Magn Reson Med* 1987;5:466–470
12. Miller DH, Kendall BE, Barter S, et al. Magnetic resonance imaging in central nervous system sarcoidosis. *Neurology* 1988;38:378–383
13. Kumpe DA, Rao CVGK, Garcia JH, Heck AF. Intracranial neurosarcoidosis. *J Comput Assist Tomogr* 1979;3:324–330
14. Hayes WS, Sherman JL, Stern BJ, Citrin CM, Pulaski PD. MR and CT evaluation of intracranial sarcoidosis. *AJNR Am J Neuroradiol* 1987;8:841–847
15. Smith AS, Meisler DM, Weinstein MA, et al. High-signal periventricular lesions in patients with sarcoidosis: neurosarcoidosis or multiple sclerosis? *AJNR Am J Neuroradiol* 1989;10:485–490
16. Ketonen L, Oksanen V, Kuuliala I. Preliminary experience of magnetic resonance imaging in neurosarcoidosis. *Neuroradiology* 1987;29:127–129
17. Bahr AL, Krumholz A, Kristt D, Hodges III FJ. Neuroradiological manifestations of intracranial sarcoidosis. *Radiology* 1978;127:713–717
18. Kendall BE, Tatler GLV. Radiological findings in neurosarcoidosis. *Br J Radiol* 1978;51:81–92
19. Brooks BS, El Gammal T, Hungerford GD, Acker J, Trevor RP, Russell W. Radiologic evaluation of neurosarcoidosis: role of computed tomography. *AJNR Am J Neuroradiol* 1982;3:513–521
20. Leeds NE, Zimmerman RD, Elkin CM, Nussbaum M, LeVan AM. Neurosarcoidosis of the brain and meninges. *Semin Roentgenol* 1985;20:387–392

21. Greco A, Steiner RE. Magnetic resonance imaging in neurosarcoidosis. *Magn Reson Imaging* 1987;5:15–21
22. Sze G, Soletsky S, Bronen R, Krol G. MR imaging of the cranial meninges with emphasis on contrast enhancement and meningeal carcinomatosis. *AJNR Am J Neuroradiol* 1989;10:965–975
23. Sherman JL, Stern BJ. Sarcoidosis of the CNS: comparison of unenhanced and enhanced MR images. *AJNR Am J Neuroradiol* 1990;11:915–923
24. Phillips ME, Ryals TJ, Kambhu SA, Yuh WTC. Neoplastic vs inflammatory meningeal enhancement with Gd-DTPA. *J Comput Assist Tomogr* 1990;14:536–541
25. Williams III DW, Elster AD, Kramer SI. Neurosarcoidosis: gadolinium-enhanced MR Imaging. *J Comput Assist Tomogr* 1990;14:704–707
26. Walker FO, McLean WT Jr, Elster A, Stanton C. Chiasmal sarcoidosis. *AJNR Am J Neuroradiol* 1990;11:1205–1207
27. Sauter MK, Panitch HS, Kristt DA. Myelopathic neurosarcoidosis: diagnostic value of enhanced MRI. *Neurology* 1991;41:150–151
28. Seltzer S, Mark AS, Atlas SW. CNS sarcoidosis: evaluation with contrast-enhanced MR imaging. *AJNR Am J Neuroradiol* 1991;12:1227–1233
29. Khaw KT, Manji H, Britton J, Schon F. Neurosarcoidosis—demonstration of meningeal disease by gadolinium enhanced magnetic resonance imaging. *J Neurol Neurosurg Psychiatry* 1991;54:499–502
30. Ulmer JL, Elster AD. Sarcoidosis of the central nervous system. *Neuroimaging Clin North Am* 1991;1:141–158
31. James DJ, Sharma OP. Neurological complications of sarcoidosis. *Proc R Soc Med* 1967;60:1169–1174
32. Wiederholt WC, Siekert RG. Neurological manifestations of sarcoidosis. *Neurology* 1965;15:1147–1154
33. Silverstein A, Feuer MM, Siltzback LE. Neurologic sarcoidosis. *Arch Neurol* 1965;12:1–11
34. Jefferson M. Sarcoidosis of the nervous system. *Brain* 1957;80:540–555
35. Baron B, Goldberg AL, Rothfus WE, Sherman RL. CT features of sarcoid infiltration of a lumbosacral nerve root. *J Comput Assist Tomogr* 1989;13:364–365
36. Colover J. Sarcoidosis with involvement of the nervous system. *Brain* 1948;71:451–475
37. Babu VS, Eisen H, Pataki K. Sarcoidosis of the central nervous system. *J Comput Assist Tomogr* 1979;3:396–397
38. Herring AB, Urich H. Sarcoidosis of the central nervous system. *J Neurol Sci* 1969;9:405–422
39. Cahill DW, Saleman M. Neurosarcoidosis: a review of the rarer manifestations. *Surg Neurol* 1981;15:204–211
40. Brooks Jr J, Stuckland MC, Williams JP, Vulpe M, Fowler HL. Computed tomography changes in neurosarcoidosis clearing with steroid treatment. *J Comput Assist Tomogr* 1979;3:398–399
41. Clark WC, Acker JD, Dohan FC, Robertson JH. Presentation of central nervous system sarcoidosis as intracranial tumors. *J Neurosurg* 1985;63:851–856
42. de Tribolet N, Zander E. Intracranial sarcoidosis presenting angiographically as a subdural hematoma. *Surg Neurol* 1978;9:169–171
43. Decker RE, Mardayat M, Marc J, Rasool A. Neurosarcoidosis with computerized tomographic visualization and transsphenoidal excision of a supra- and intrasellar granuloma. *J Neurosurg* 1979;50:814–816
44. Goodman SS, Margulies ME. Boeck's sarcoid simulating a brain tumor. *Arch Neurol Psychiatry* 1959;81:419–423
45. Skillicorn SA, Garrity RW. Intracranial Boeck's sarcoid tumor resembling meningioma. *J Neurosurg* 1955;12:407–413
46. Norwood CW, Kelly DL. Intracerebral sarcoidosis acting as a mass lesion. *Surg Neurol* 1974;2:367–372
47. Slavin ML, Glaser JS. Optic neuropathy and cerebral sarcoidosis. *J Clin Neuro Ophthalmol* 1983;3:259–262
48. Powers WJ, Miller EM. Sarcoidosis mimicking glioma: case report and review of intracranial sarcoid mass lesions. *Neurology* 1981;31:907–910
49. Lax F, Tabaddor K. Extraparenchymal sarcoid mass: a case report. *Neurosurgery* 1979;5:604–606
50. Lukin RR, Chambers AA, Soleimanpour M. Outlet obstruction of the fourth ventricle in sarcoidosis. *Neuroradiology* 1975;10:65–68
51. Stern B, Krumholtz A, Johns C, Scott P, Nissim J. Sarcoidosis and its neurological manifestations. *Arch Neurol* 1985;42:909–917
52. Cohen JP, Lachman LJ, Hammerschlag PE. Reversible facial paralysis in sarcoidosis. *Arch Otolaryngol* 1983;109:832–835
53. Galetta S, Schatz NJ, Glaser JS. Acute sarcoid optic neuropathy with spontaneous recovery. *J Clin Neuro Ophthalmol* 1989;9:27–32
54. Rush J. Retrobulbar optic neuropathy in sarcoidosis. *Ann Ophthalmol* 1980;12:390–394
55. Kirkham TH. Neuro-ophthalmic presentations of sarcoidosis. *Proc R Soc Med* 1973;66:167–169
56. Tang RA, Grotta JC, Lee KF, Lee EL. Chiasmal syndrome in sarcoidosis. *Arch Ophthalmol* 1983;101:1069–1073
57. Ingestad R, Stigmar G. Sarcoidosis with ocular and hypothalamic pituitary manifestations. *Acta Ophthalmol* 1971;49:1–10
58. Stuart CA, Neelon FA, Lebovitz HE. Hypothalamic insufficiency: the cause of hypopituitarism in sarcoidosis. *Ann Intern Med* 1978;88:589–594
59. Vesely D, Maldonodo A, Levey G. Partial hypopituitarism and possible hypothalamic involvement in sarcoidosis: report of a case and review of the literature. *Am J Med* 1977;62:425–431
60. Meyer JS, Foley JM, Compagna-Pinto D. Granulomatous angiitis of the meninges in sarcoidosis. *Arch Neurol Psychiatry* 1953;69:587–600
61. Reske-Nielsen E, Harmsen A. Periangiitis and panagiitis as a manifestation of sarcoidosis of the brain: report of a case. *J Nerv Ment Dis* 1962;135:399–412
62. Griggs RC, Markesbery WR, Condemi JJ. Cerebral mass due to sarcoidosis. *Neurology* 1973;23:981–989
63. Kelly RB, Mahoney PD, Cawley KM. MR Demonstration of spinal cord sarcoidosis: report of a case. *AJNR Am J Neuroradiol* 1988;9:197–199
64. Nesbitt GM, Miller GM, Baker HL Jr, Ebersold MJ, Scheithauer BW. Spinal cord sarcoidosis: a new finding at MR imaging with Gd-DTPA enhancement. *Radiology* 1989;173:839–843
65. Martin N, Debroucker T, Mompoint D, Akoun J, Cambier J, Nahum H. Sarcoidosis of the pineal region: CT and MR studies. *J Comput Assist Tomogr* 1989;13:110–112
66. Reed LD, Abbas S, Markivee CR, Fletcher JW. Neurosarcoidosis responding to steroids. *AJR Am J Roentgenol* 1986;146:819–821
67. Sherman JL, Citrin CM, Barkovich AJ, Bowen BJ. MR Imaging of the mesencephalic tectum: normal and pathologic variations. *AJNR Am J Neuroradiol* 1987;8:59–64
68. Cooper SD, Brady MB, Williams JP, Pilgreen KL, Harp DL, Weissmann JR. Neurosarcoidosis: evaluation using computed tomography and magnetic resonance imaging. *J Comput Tomogr* 1988;12:96–99
69. Michotte A, Dequenne P, Jacobovitz D, Hildebrand J. Focal neurological deficit with sudden onset as the first manifestation of sarcoidosis: a case report with MRI follow-up. *Eur Neurol* 1991;31:376–379
70. Katz B. Disc edema, transient obscurations of vision, and a temporal fossa mass. *Surv Ophthalmol* 1991;36:133–139
71. Tien RD, Yang PJ, Chu PK. Dural tail sign; a specific MR sign for meningioma? *J Comput Assist Tomogr* 1991;15:64–66
72. Vazquez R, Oates E, Sarno RC, Fay J, Gale DR. Gallium-67 breast uptake in a patient with hypothalamic granuloma (sarcoid). *J Nucl Med* 1988;29:118–121
73. Allen KS, Glickstein M, Arger PH, Bilaniuk L, Levy DW. Cryptococcosis associated with sarcoidosis: CT and MR findings. *J Comput Assist Tomogr* 1988;12:420–422

74. Tonami H, Tamamura H, Kimizu K, et al. Intraocular lesions in patients with systemic disease: findings on MR imaging. *AJNR Am J Neuroradiol* 1989;10:1185–1189
75. Atlas SW, Grossman RI, Savino PJ, et al. Surface-coil MR of orbital pseudotumor. *AJNR Am J Neuroradiol* 1987;8:141–146
76. Aldrich MS, Naylor MW. Naroclepsy associated with lesions of the diencephalon. *Neurology* 1989;39:1505–1508
77. Brooks ML, Wang A, Black PM, Haikal N. Subdural mass lesion secondary to sarcoid granuloma MR and CT findings and differential diagnosis. *Comput Med Imaging Graph* 1989;13:199–205
78. Krumholz A, Stern BJ, Stern EG. Clinical implications of seizures in neurosarcoidosis. *Arch Neurol* 1991;48:842–844
79. Kerdel FA, Moschella SL. Sarcoidosis: an updated review. *J Am Acad Dermatol* 1984;11:1–19