

Are your MRI contrast agents cost-effective?

Learn more about generic Gadolinium-Based Contrast Agents.



AJNR

MR Imaging of the Brain in Lymphomatoid Granulomatosis

Ukihide Tateishi, Satoshi Terae, Akihiko Ogata, Yutaka Sawamura, Yasuhiro Suzuki, Satoru Abe and Kazuo Miyasaka

This information is current as of April 29, 2024.

AJNR Am J Neuroradiol 2001, 22 (7) 1283-1290
<http://www.ajnr.org/content/22/7/1283>

MR Imaging of the Brain in Lymphomatoid Granulomatosis

Ukihide Tateishi, Satoshi Terae, Akihiko Ogata, Yutaka Sawamura, Yasuhiro Suzuki, Satoru Abe, and Kazuo Miyasaka

BACKGROUND AND PURPOSE: Clinical diagnosis of lymphomatoid granulomatosis (LG) of the brain, in patients without skin or chest lesions, usually is difficult because of the non-specific neurologic manifestations, laboratory data, and CT appearance. Our aim was to characterize the MR appearance of LG of the brain.

METHODS: We retrospectively reviewed the MR images in four patients (35 to 72 years old) with histologically confirmed LG of the brain.

RESULTS: On T2-weighted images, we noted diffuse hyperintense lesions in the cerebral white matter bilaterally (n = 3), in the brain stem and cerebellar hemisphere (n = 1), and patchy hyperintense lesions the brain stem (n = 2). On contrast-enhanced T1-weighted images, we observed multiple punctate or linear enhancements residing along the perivascular space (n = 4), nodular enhancements (n = 2), ringlike enhancements (n = 1), and a large, enhanced mass (n = 1). All patients had multifocal lesions.

CONCLUSION: Although the MR appearance of LG of the brain varies, multiple punctate or linear enhancements that reside along the perivascular space suggest LG.

Lymphomatoid granulomatosis (LG) is an uncommon multisystem disease characterized by multifocal “angiocentric angiodestructive lymphoreticular proliferative and granulomatous lesions” (1). LG involves the lungs most frequently, followed by the skin and brain (2). Neurologic symptoms may be the initial or only manifestation of the disease, but they are nonspecific. Laboratory findings usually are unremarkable (2). The reported radiologic appearance of LG of the brain, mostly using CT, has varied and has been nonspecific. Thus, the diagnosis of LG of the brain, without lung or skin involvement, usually is delayed or made only at autopsy. In this report, we characterize the MR appearance of the LG of the brain through review of imaging data from four local cases and those in the literature.

Methods

We retrospectively reviewed four cases of LG in the brain from our case records. The patients were three men and one woman, ranging in age from 35 to 72 years (mean, 60 years). At diagnosis, three of the patients had presented with spastic gait, and one had presented with polyradiculoneuritis, skin eruptions, and spastic gait (Table). This latter patient (case 3) also had pulmonary lesions. The other three patients had no evidence of LG lesions outside the central nervous system. LG was confirmed histologically in all cases by means of either open or stereotactic brain biopsy.

All tissues were fixed in 10% neutral formalin and embedded in paraffin. Five-micrometer sections were stained with hematoxylin-eosin and lymphocyte surface markers. We followed the avidin-biotin-peroxidase complex procedure, using antibodies to CD20 (a cytoplasmic antigen in B-lymphocytes) and to CD3 (a marker for T-lymphocytes).

MR imaging data were collected from three institutions. The interval between initial presentation and MR imaging ranged from 1 week to 7 years (Table). MR images were obtained with 1.5-T scanners. We obtained axial images of 4- to 6-mm thickness in the following pulse sequences: pre- and postcontrast T1-weighted spin-echo sequences with imaging parameters of 540–600/14–20/1–2 (TR/TE/excitations) and T2-weighted fast spin-echo sequences with imaging parameters of 3500–4500/93–96/1–2 (TR/TE_{eff}/excitations) with a field of view of 23 × 23 cm and an image matrix of 256 × 256. Each patient also underwent MR imaging after intravenous administration of 0.1 mmol/kg of gadopentetate dimeglumine or gadoteridol. The images were evaluated by two neuroradiologists.

On T2-weighted images, we described the location of abnormalities in signal intensity and categorized them as patchy or diffuse hyperintense areas, infarctlike, suggestive of a mass lesion, perifocal edema; or suggestive of hemorrhage. Enhancement was divided into parenchymal and meningeal. Pa-

Received October 9, 2000; accepted after revision March 13, 2001.

From the Departments of Radiology (U.T., S.T., K.M.), Neurological Medicine (A.O.), and Brain Surgery (Y.S.), Hokkaido University School of Medicine; First Department of Medicine (Y.S.), Asahikawa Medical College; and Department of Radiology (S.A.), Sapporo Azabu Neurosurgical Hospital, Hokkaido, Japan.

Address reprint requests to Ukihide Tateishi, MD, Department of Radiology, Hokkaido University School of Medicine, N-15 W-7, Kita-ku, Sapporo, Hokkaido 060-8638, Japan.

© American Society of Neuroradiology

Summary of MR findings in patients with LG of the brain

Case (No.)	Age (y)/ Sex	Symptoms at Onset	Symptoms at Presentation	Interval between MR Examination and Onset	MR Findings		
					High Intensity on T2-weighted Image		Peritumoral Edema
					Patchy	Diffuse	
1	72/M	Spastic gait	Paraparesis	1 week	Bilateral cerebral WM, cerebellum, brain stem	(-)	(+)
2	58/M	Spastic gait	Paraparesis	7 years	(-)	Bilateral cerebral and cerebellar WM, brain stem	NA
3	56/F	Polyradiculoneuritis, skin lesions, spastic gait	Left facial nerve palsy, nystagmus, dysarthria	4 years	(-)	Bilateral cerebral WM	(+)
4	35/M	Spastic gait	Chorea-athetosis, dementia, anisocoria, apraxia, rigidity, spasticity	10 months	Brain stem	Bilateral cerebral WM	NA

Note.—WM indicates white matter; NA, not applicable; PR, partial regression; RT, radiation therapy; Rec, recurrence.

enchymal enhancement was categorized as no enhancement, solid, ringlike, nodular, punctate, or linear. Nodular enhancements were defined as round enhancements 3–9 mm in diameter; punctate enhancements, as dotlike enhancements <3 mm in diameter. In cases of linear enhancement, we also described whether the enhancement appeared to reside along the intramedullary vessels.

Results

MR imaging of the brain showed multifocal abnormalities in all four cases (Table). On T2-weighted images, three patients showed diffuse hyperintense areas in the cerebral white matter bilaterally, and this abnormality was observed in the brain stem ($n = 2$) and cerebellar white matter bilaterally ($n = 1$). One patient also showed patchy hyperintense areas in the brain stem (case 1 [Fig 1]). On postcontrast T1-weighted images, two patients showed multiple punctate and linear enhancements (cases 2 and 4 [Figs 2 and 3]), one showed multiple punctate enhancement (case 1), and one showed multiple linear enhancements (case 3). The punctate and linear enhancements seemed to reside along the intramedullary vessels. Nodular enhancement was seen in two patients. Two patients showed a mass lesion; a solid mass accompanied by necrosis in one case (case 1 [Fig 1]) and two ringlike enhanced masses in the other (case 3 [Fig 4]). There were no infarctlike lesions or hemorrhages. Meningeal enhancement was not present.

Microscopic examination revealed LG in all four cases. All patients showed perivascular polyclonal lymphoid cell infiltrates and angiodescriptive fea-

tures. The infiltrate was composed of mononuclear cells admixed with histiocytes and plasma cells. Lymphoid cells with atypical nuclei were seen in two patients (cases 2 and 3 [Figs 2E and 4G]). The lymphoid infiltrates were composed predominantly of the T-cell phenotype in two patients (cases 1 and 3) and were composed of both T- and B-cell phenotypes in two patients (cases 2 and 4).

All patients received pulsed and maintenance corticosteroid treatment. The steroid therapy was followed by cyclophosphamide in one patient (case 2) and by radiation therapy in the other three patients (cases 1–3). In all patients, the lesions became smaller or disappeared with treatment. One patient with acute onset (case 1) died from recurrence 15 months after radiation therapy. Another patient (case 3) showed a relapsing-remitting clinical course.

Discussion

Lymphomatoid granulomatosis was first described in 1972 as an angiocentric, angiodescriptive lymphoproliferative, and granulomatous disease that predominantly affects the lung (1). The most commonly involved extrapulmonary sites are the skin and the nervous system; however, any organ system may contain the lesions typical of LG (1).

The clinical manifestations of LG generally consist of chest symptoms such as cough and shortness of breath, systemic complaints such as fever, weight loss, or malaise. Frequently, skin lesions (a raised erythematous rash or nodules) appear si-

Extended.

MR Findings (Extended)					
Mass Lesion	Contrast-enhanced T1WI	Other	Histopathologic Findings	Therapy	Follow-up
Heterogeneous (high + iso) intensity from splenium to parietal white matter	Intensely enhancing mass with small necrosis, multiple punctate enhancements	Subdural hematoma	T cell predominant-LG	Pulsed steroid and maintenance RT	Rec at 4 months after RT, died at 19 months
(-)	Multiple nodular, punctate, and linear enhancements	Mild cerebellar atrophy, multiple punctate enhancements in spinal cord	T + B cell LG	Pulsed steroid and maintenance RT	PR at 24 months
Heterogeneous (high + low) intensity at left middle cerebellar peduncle, heterogeneous (high + iso) intensity at right cingulate gyrus	Ringlike enhancing lesions at left middle cerebellar peduncle and right cingulate gyrus, linear enhancement in pons	...	T + B cell LG	Pulsed steroid and maintenance, cyclophosphamide, and RT	Rec at 5 months, PR 8 months after radiation for Rec
(-)	Multiple nodular, punctate and linear enhancements	Brain atrophy	T cell predominant-LG	Pulsed steroid and maintenance	PR at 14 months

multaneously with the lung lesions. Neurologic symptoms also are common, present in 30% of cases, and usually are seen with chest or systemic complaints (2). Neurologic symptoms may be the initial or only manifestation of the disease, however. Such symptoms frequently are nonspecific and may consist of headache, seizures, hemiparesis, ataxia, blindness, deafness, cranial-nerve palsies, altered consciousness, or dementia (2-4). All of our cases had an initial manifestation of the disease in the central nervous system, it was localized in the brain in all but one patient (case 3), and all patients had spastic gait. In case 3, multiple pulmonary nodular lesions were seen on chest radiography, but this patient had no respiratory symptoms.

Histologically, LG is characterized by a polymorphic lymphoid infiltrate of atypical mononuclear cells, small lymphocytes, plasma cells, and histiocytes. Most lymphocytes exhibit the T-cell phenotypic pattern. Granulomas may form and infiltrate the meninges, vessels, and parenchyma (11). Neurologic involvement may reflect direct infiltration of cranial and peripheral nerves by the perivascular lymphoid infiltrates or thrombosis and infarction from vasculitis (13).

The pathogenesis of LG remains to be elucidated. Some investigators consider LG to be a reactive or "preneoplastic" process, possibly in reaction to antigen stimulation, such as that induced by the Epstein-Barr virus (16, 17). Others consider LG an angiocentric variant of T-cell lymphoma (14), because a certain proportion of patients with LG de-

velop non-Hodgkin's lymphoma (1, 2). Lipford et al (18) showed that LG represents a spectrum of T-cell proliferation from lack of atypia to frank angiocentric lymphoma. Histologic subclasses have been proposed by Jaffe et al (19).

In our series, perivascular granulomatous infiltrates of lymphoid cells in the biopsy specimens correlated with enhanced lesions on MR images. Because the biopsy specimens were small, we could not completely correlate the MR imaging and histopathologic findings. We speculate, however, that multiple punctate and linear enhancements, which appear to reside along the medullary vessels on MR imaging, represent abnormal perivascular tissue and affected walls of small vessels, and nodular enhancements represent granuloma formation. We also speculate that the hyperintense areas on T2-weighted images, other than peritumoral edema, represents reactive or ischemic changes including vacuolization, reactive and ischemic gliosis, and ischemic demyelination caused by vascular stenosis and thrombosis (20, 21).

The appearance of LG of the brain on CT varies but most often shows intraparenchymal lesions, which may be unifocal or multifocal and supra- or infratentorial in location. These lesions may appear as solid or ringlike, enhanced masses with or without edema, or nonconfluent, low-density areas of white matter without enhancement. They may have associated leptomeningeal thickening or enhancement. Occasionally, they appear as intraparenchymal hemorrhage, cerebral infarction, extraparenchymal mass lesions, or aneurysm (3-11). Cerebral

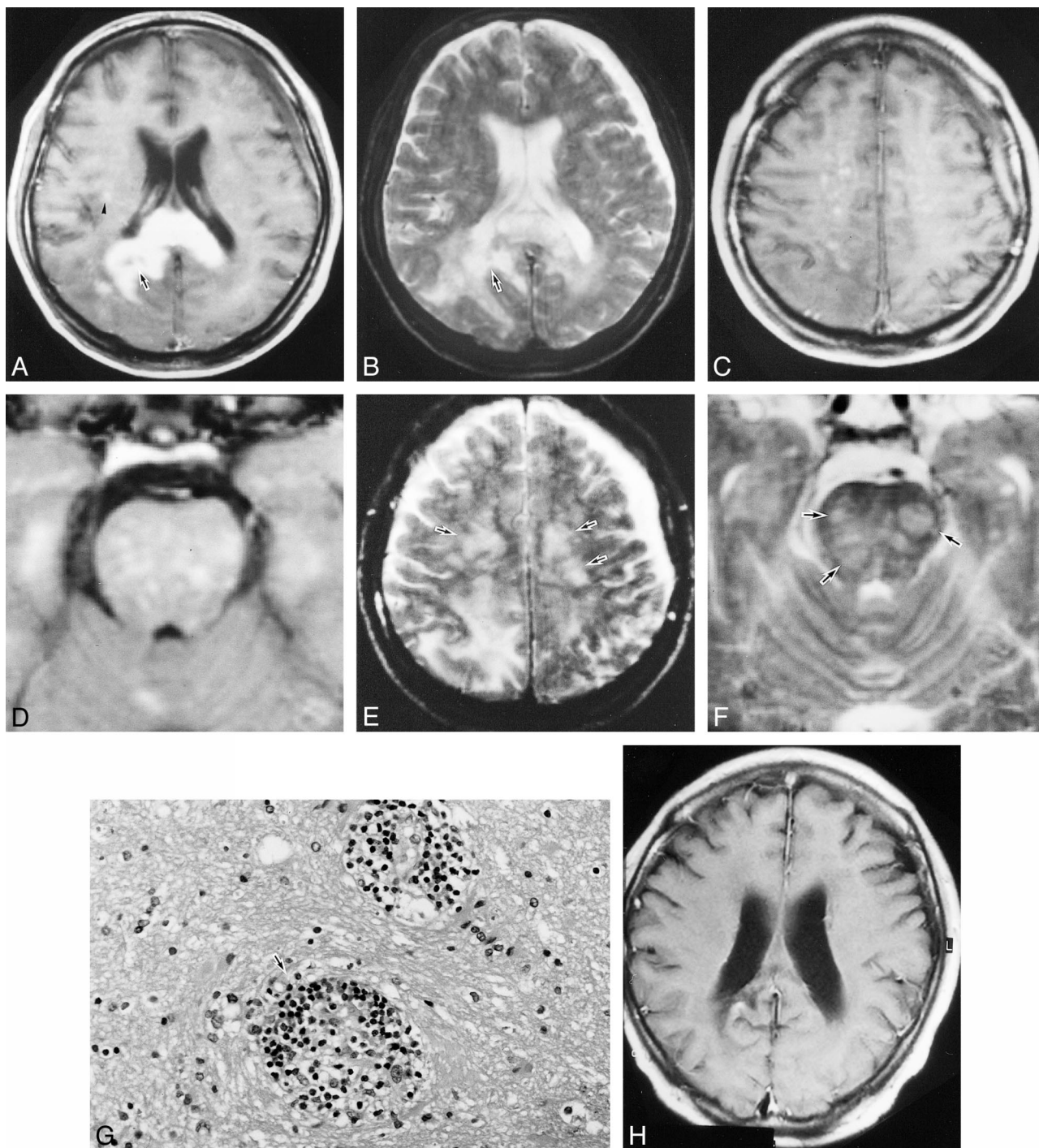


FIG 1. Case 1.

A, Contrast-enhanced T1-weighted axial image (spin-echo sequence with parameters 600/14 [TR/TE]) shows an enhanced lesion from the splenium of the corpus callosum extending to the right parietal white matter. There is a small area of necrosis within the mass (*arrow*). Nodular and multiple punctate enhancements are seen in the white matter (*arrowhead*).

B, T2-weighted image (fast spin-echo sequence with parameters of 4000/96 [TR/TE]) shows a hyperintense mass (*arrow*) and surrounding edema.

C and D, Contrast-enhanced T1-weighted images show multiple punctate enhancements in the pons and cerebral white matter bilaterally.

E and F, T2-weighted axial image shows patchy hyperintense areas in the pons and cerebral white matter bilaterally (*arrows*).

G, Stereotactic biopsy specimen from the right parietal lobe shows small lymphoid cells, microglia, and macrophages, which were prominent in the perivascular space (*arrow*).

H, Contrast-enhanced T1-weighted image 6 months after radiation therapy shows that the mass has almost disappeared.

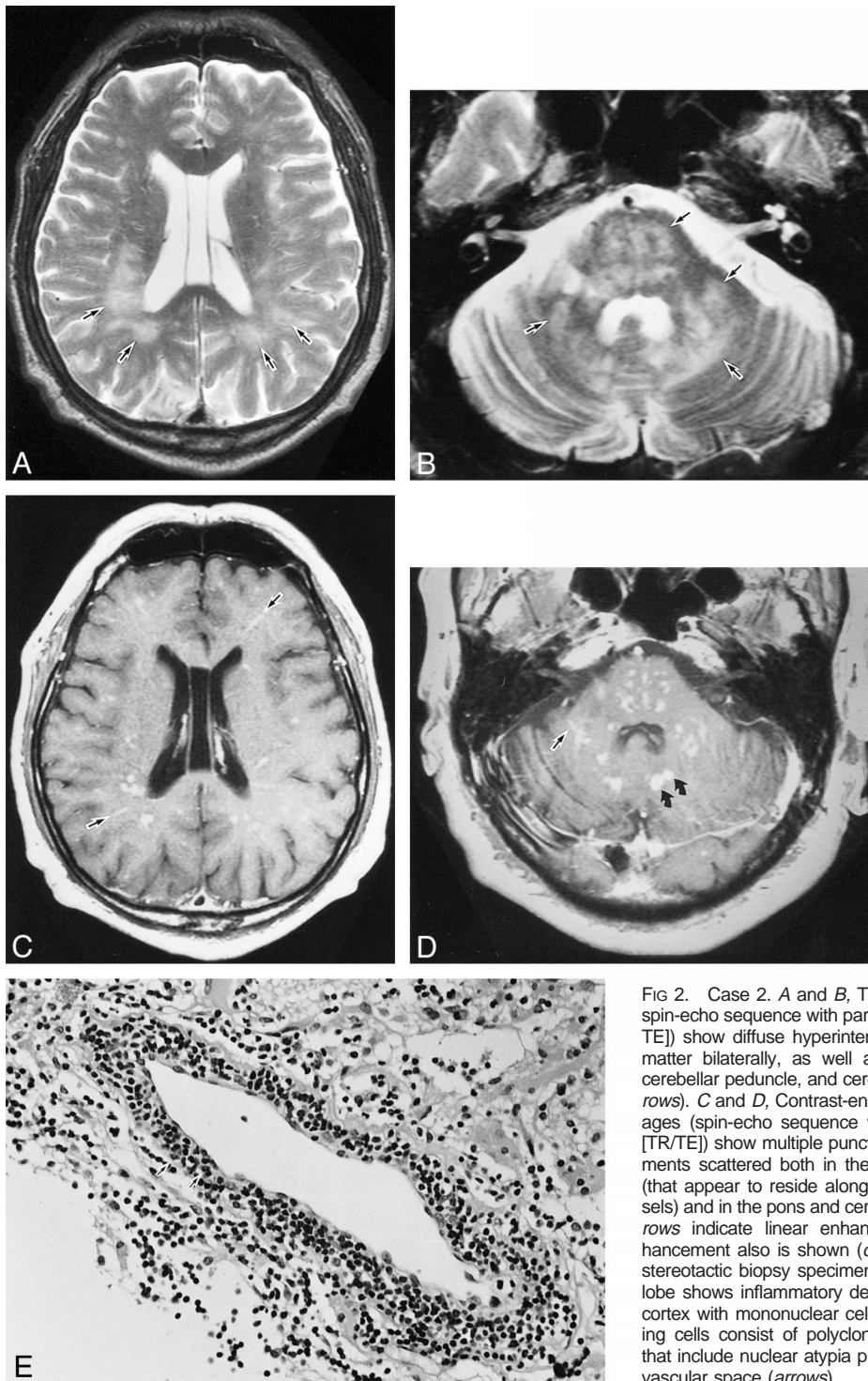


FIG 2. Case 2. *A* and *B*, T2-weighted images (fast spin-echo sequence with parameters of 4500/96 [TR/TE]) show diffuse hyperintense lesions in the white matter bilaterally, as well as in the pons, middle cerebellar peduncle, and cerebellar hemispheres (*arrows*). *C* and *D*, Contrast-enhanced T1-weighted images (spin-echo sequence with parameters 600/14 [TR/TE]) show multiple punctate and linear enhancements scattered both in the white matter bilaterally (that appear to reside along the intramedullary vessels) and in the pons and cerebellar hemispheres (*arrows* indicate linear enhancements). Nodular enhancement also is shown (*curved arrows*). *E*, Brain stereotactic biopsy specimen from the right occipital lobe shows inflammatory destruction of the cerebral cortex with mononuclear cell infiltration. The infiltrating cells consist of polyclonal small lymphoid cells that include nuclear atypia predominantly in the perivascular space (*arrows*).

angiography may show findings consistent with vasculitis (12).

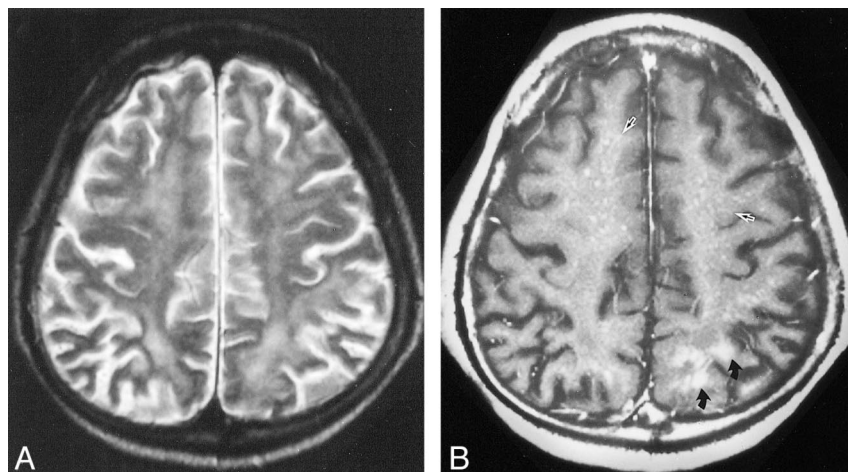
We found 15 case reports in the English-language literature that describe findings on MR imaging for LG of the central nervous system (3, 4, 6, 10, 13, 21–27). Thirteen of these cases had intracranial lesions, two had intraorbital lesions, and two had intraspinal lesions. Of the 13 patients with intracranial lesions, 11 had intraparenchymal lesions (3–7, 9, 10, 13, 21, 24, 26), one had supra-

tentorial extraaxial masses (27), and one had a cavernous sinus mass and meningeal thickening (22). The intraparenchymal lesions often were multifocal but unifocal lesions were observed in two cases. These lesions were found in supratentorial or infratentorial locations; the affected areas were the cerebral white matter with or without extension into the adjacent cortical gray matter, basal ganglia, cerebellum, or brain stem. The white matter lesions were located in the periventricular, deep, or sub-

FIG 3. Case 4.

A, T2-weighted image (fast spin-echo sequence with parameters of 3500/96 [TR/TE]) shows diffuse hyperintense areas in the cerebral white matter bilaterally. Cerebral sulci are enlarged bilaterally, indicating cerebral atrophy.

B, Contrast-enhanced T1-weighted image (spin-echo sequence with parameters of 600/14 [TR/TE]) shows multiple punctate and linear (arrows) enhancements in the white matter bilaterally and nodular enhancement in left parietal lobe (curved arrows).



cortical regions. On T2-weighted images, these lesions were most frequently hyperintense relative to the brain parenchyma, with small foci, round, globular, infarctlike, or masslike lesions with or without associated edema or mass effect. In two cases the lesion showed low signal intensity centrally (necrosis with hemorrhage) with peripheral hyperintense edema, and in one case the lesion showed mixed intensity. On T1-weighted images, these lesions were hypointense relative to the brain parenchyma. As for the intraparenchymal lesions, contrast-enhanced T1-weighted images showed ringlike enhancements in three patients and multiple punctate and linear enhancements in one patient (6, 9, 21). Serial MR imaging findings were described for four patients (4, 21, 22, 26); the lesions increased in size or number after variable follow-up periods.

In this study, ringlike enhancement was observed in one patient, and multiple linear or punctate enhancements were observed in all four patients. Although the literature has described the findings of contrast-enhanced T1-weighted images for only three patients with brain lesions, these enhancement patterns were in accord with those of the previous reports. In case 1, a large, enhanced, solid mass lesion was observed in the splenium of the deep white matter of the right parietal lobe. The stereotactic biopsy specimen was not obtained from this lesion but from a nodular, enhanced lesion at the subcortical white matter of the right parietal lobe. We speculated that the mass was lymphoma associated with or evolved from LG. We consider the multiple punctate and linear enhancements on MR imaging to be characteristic of LG because they most likely represent perivascular tissue and walls of small vessels affected by the disease. The punctate or linear enhancements are not diagnostic for the disease, however, because these can occur in other diseases, including sarcoidosis, primary angiitis of the central nervous system, other granulomatous angiitis (such as Churg-Strauss syndrome), and intravascular lymphomatosis (8, 28). Nonetheless, the possibility of LG should be considered if these findings are observed. Ringlike en-

hancement was seen in one patient in our series and in three cases in the literature (two patients with brain lesions and one patient with a spinal cord lesion) (6, 10, 25). It is known that ringlike enhancement can be seen in various diseases, such as metastatic tumors, glioblastomas, abscesses, and acute demyelinating plaques in multiple sclerosis. Among these diseases, ringlike, enhanced brain lesions in LG may especially mimic metastatic tumors, and differentiation may be quite difficult without histologic examination.

On T2-weighted images, we noted patchy or diffuse hyperintense areas other than peritumoral edema in the cerebral white matter in all patients in our study. Two patients also showed hyperintense areas in the brain stem and cerebellar hemisphere. We speculate that the hyperintense areas represent reactive or ischemic change caused by the disease; however, this finding is nonspecific, and it is difficult to suggest LG on the basis of T2-weighted images alone.

The prognosis of LG is poor, especially in LG of the brain, and its treatment is controversial. Treatment with various cytotoxic agents, corticosteroids, and radiotherapy have been described (14). Among these, the combination chemotherapy of cyclophosphamide and corticosteroid has been reported as a strategy for long-term remission (15). All of our cases received pulsed and maintenance steroid therapy; three patients also received radiation therapy, and one received the combination of steroid and cyclophosphamide therapy. All patients showed partial remission immediately after treatment, but two patients later had recurrence of disease. Although the number of patients is limited, our data suggest that acute onset of the disease predicts poor prognosis, whereas gradual onset or progression predicts a better prognosis after therapy.

Conclusion

The clinical diagnosis of LG is difficult when the lesion is confined to the brain. Although radiologic findings in LG of the brain are quite variable, the

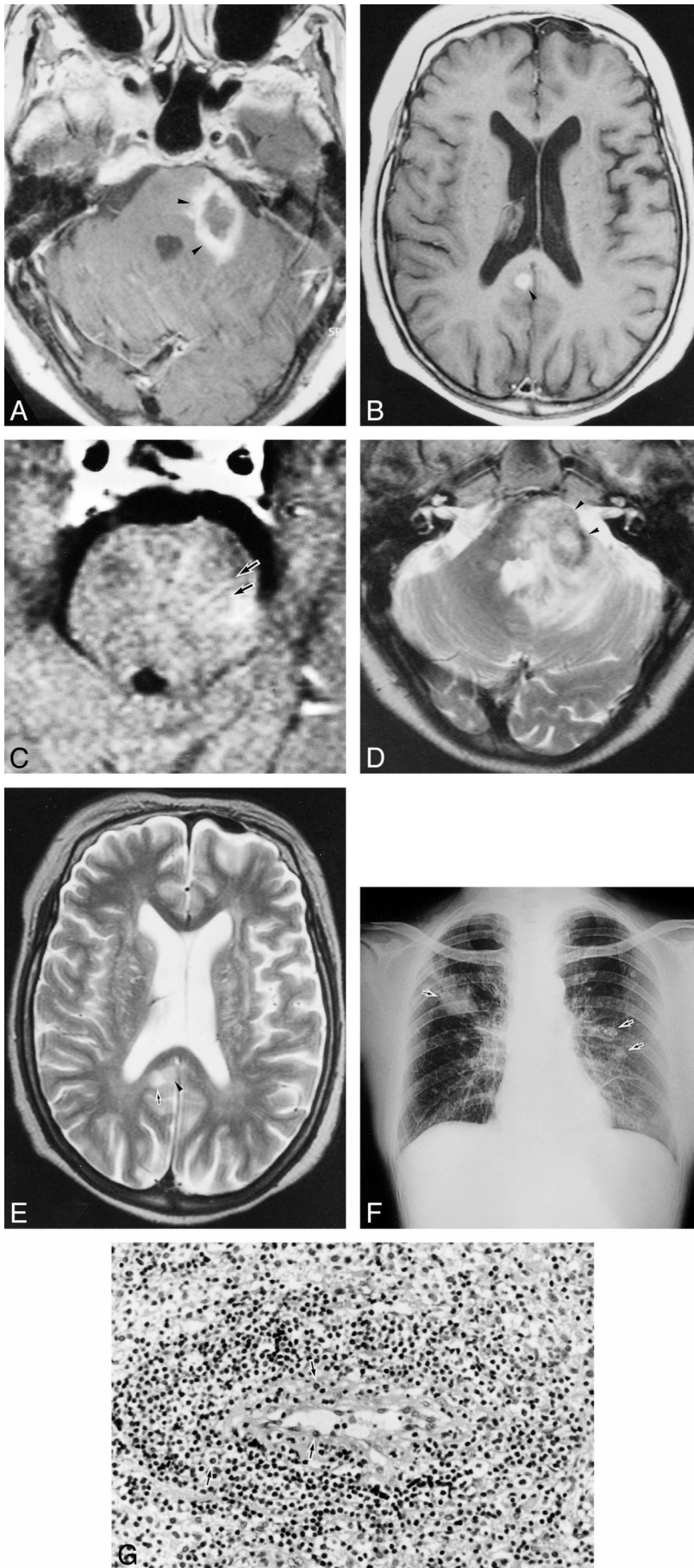


FIG 4. Case 3.
 A and B, Contrast-enhanced T1-weighted images (spin-echo sequence with parameters of 600/14 [TR/TE]) show ringlike enhancements in the left middle cerebral peduncle and pons and in the right cingulate gyrus (arrowheads).
 C, The slices of 4-mm thickness show linear enhancements in the pons (arrows).
 D and E, T2-weighted MR images (fast spin-echo sequence with parameters of 4500/96 [TR/TE]) show that the ringlike, enhanced lesions are hypointense to isointense with gray matter (arrowheads). There are hyperintense areas surrounding these lesions, suggesting perifocal edema (arrow).
 F, Chest radiograph shows multiple nodular shadows in both lung fields (arrows).
 G, Open brain biopsy specimen from the left middle cerebellar peduncle shows perivascular lymphoid-cell infiltration with partial nuclear atypia (arrows).

presence of multiple punctate or linear enhancements that reside along the perivascular space on MR imaging suggests LG or other diseases affecting the vascular wall or perivascular space, such as sarcoidosis, primary angiitis of the central nervous system, and other granulomatous angiitis. Although less specific for LG, when ringlike enhancement or meningeal thickening and enhancement is observed on MR images, the possibility of LG should be considered in an appropriate clinical setting.

Acknowledgment

We sincerely thank Dr. Hitoshi Aizawa, First Department of Medicine, Asahikawa Medical College and Professor Kazuo Nagashima and Assistant Professor Hiroshi Nishihara, Department of Second Pathology, Hokkaido University School of Medicine, for their invaluable and kind guidance of this study.

References

- Liebow A, Carrington C, Friedman P. **Lymphomatoid granulomatosis.** *Hum Pathol* 1972;3:457-558
- Katzenstein AL, Carrington CB, Liebow AA. **Lymphomatoid granulomatosis: a clinicopathologic study of 152 cases.** *Cancer* 1979;43:360-373
- Kerslake R, Rowe D, Worthington BS. **CT and MR imaging of CNS lymphomatoid granulomatosis.** *Neuroradiology* 1991;33:269-271
- Kleinschmidt-DeMasters BK, Filley CM, Bitter MA. **Central nervous system angiocentric, angiodestructive T-cell lymphoma (lymphomatoid granulomatosis).** *Surg Neurol* 1992;37:130-137
- Kapila A, Gupta KL, Garcia JH. **CT and MR of lymphomatoid granulomatosis of the CNS: report of four cases and review of the literature.** *AJNR Am J Neuroradiol* 1988;9:1139-1143
- George JC, Caldemeyer KS, Smith RR, Czaja JT. **CNS lymphomatoid granulomatosis in AIDS: CT and MR appearances.** *AJR Am J Roentgenol* 1993;161:381-383
- Simard H, LeBlanc P. **Radiotherapy: an effective treatment of cerebral involvement by lymphomatoid granulomatosis.** *Chest* 1993;103:650-651
- Shoemaker EI, Lin ZS, Rae-Grant AD, Little B. **Primary angiitis of the central nervous system: unusual MR appearance.** *AJNR Am J Neuroradiol* 1994;15:331-334
- Bhagavatula K, Scott TF. **Magnetic resonance appearance of cerebral lymphomatoid granulomatosis.** *J Neuroimag* 1997;7:120-121
- Hamilton MG, Demetrick DJ, Tranmer BI, Curry B. **Isolated cerebellar lymphomatoid granulomatosis progressing to malignant lymphoma.** *J Neurosurg* 1994;80:314-320
- Simon RH, Adeles M, Farber NJ, Grunnet M, Brennan TG Jr. **Lymphomatoid granulomatosis with multiple intracranial lesions: case report.** *J Neurosurg* 1981;55:293-298
- Sackett JF, ZuRhein GM, Bhimani SM. **Lymphomatoid granulomatosis involving the central nervous system: radiologic-pathologic correlation.** *AJR Am J Roentgenol* 1979;132:823-826
- Smith AS, Huang TE, Weinstein MA. **Periventricular involvement in CNS lymphomatoid granulomatosis: MR demonstration.** *J Comput Assist Tomogr* 1990;14:291-293
- Sordillo PP, Barbara E, Koziner B, et al. **Lymphomatoid granulomatosis: an analysis of clinical and immunologic characteristics.** *Cancer* 1982;49:2070-2076
- Fauci AS, Haynes BF, Costa J, Katz P, Wolff SM. **Lymphomatoid granulomatosis: prospective clinical and therapeutic experience over 10 years.** *N Engl J Med* 1982;306:68-74
- Ilowite NT, Fligner CL, Ochs HD, et al. **Pulmonary angiitis with atypical lymphoreticular infiltrates in Wiskott-Aldrich syndrome: possible relationship of lymphomatoid granulomatosis and EBV infection.** *Clin Immunol Immunopathol* 1986;41:479-484
- Veltri RW, Raich PC, McClung JE, Shah SH, Sprinkle PM. **Lymphomatoid granulomatosis and Epstein-Barr virus.** *Cancer* 1982;50:1513-1517
- Lipford EH Jr, Margolick JB, Longo DL, Fauci AS, Jaffe ES. **Angiocentric immunoproliferative lesions: a clinicopathologic spectrum of post-thymic T-cell proliferations.** *Blood* 1988;72:1674-1681
- Jaffe ES. **Pathologic and clinical spectrum of post-thymic T-cell malignancies.** *Cancer Invest* 1984;2:413-426
- Anders KH, Latta H, Chang BS, Tomiyasu U, Quddusi AS, Vinters HV. **Lymphomatoid granulomatosis and malignant lymphoma of the central nervous system in the acquired immunodeficiency syndrome.** *Hum Pathol* 1989;20:326-334
- Ng P, Dwyer R, Hughes A, Despas P. **Lymphomatoid granulomatosis: case report and review of the literature.** *Australas Radiol* 1997;41:57-62
- Brazis PW, Menke DM, McLeish WM, et al. **Angiocentric T-cell lymphoma presenting with multiple cranial nerve palsies and retrobulbar optic neuropathy.** *J Neuroophthalmol* 1995;15:152-157
- Forman S, Rosenbaum PS. **Lymphomatoid granulomatosis presenting as an isolated unilateral optic neuropathy: a clinicopathologic report.** *J Neuroophthalmol* 1998;18:150-152
- Collins S, Helme RD. **Lymphomatoid granulomatosis presenting as a progressive cervical cord lesion.** *Aust NZ J Med* 1989;19:144-146
- Herderschee D, Troost D, de Visser M, Neve AJ. **Lymphomatoid granulomatosis: clinical and histopathological report of a patient presenting with spinal cord involvement.** *J Neurol* 1988;235:432-434
- Petrella TM, Walker IR, Jones GW, Leber B. **Radiotherapy to control CNS lymphomatoid granulomatosis: a case report and review of the literature.** *Am J Hematol* 1999;62:239-241
- Paspala AB, Sundaram C, Purohit AK, Immaneni D. **CNS involvement by lymphomatoid granulomatosis in a 12-year-old boy: a case report.** *Surg Neurol* 1999;51:258-260
- Liow K, Asmar P, Liow M, et al. **Intravascular lymphomatosis: contribution of cerebral MRI findings to diagnosis.** *J Neuroimaging* 2000;10:116-118