

# Magnetic Resonance Imaging Features of Solitary Inflammatory Brain Masses

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## ABSTRACT

Brain lesions in inflammatory diseases may present as solitary masses, prompting a biopsy. We present neuroimaging and histologic findings in five patients with solitary, inflammatory, demyelinating mass lesions located in the supratentorial white matter and gray-white junction. The patients presented with seizures, focal neurologic signs, and neuroimaging findings that indicated the possibility of a neoplasm. Computed tomography (CT) revealed enhancing, single hypodense lesions associated with mild or no mass effect. On MRI, the lesions were hypointense on T1-weighted images and hyperintense on T2-weighted images. Variable patterns of enhancement were noted on CT and MRI, including homogeneous/patchy ( $n = 3$ ) and ring/nodular ( $n = 1$ ) enhancement. There was no evidence of calcification or hemorrhage. Biopsies revealed a leukoencephalitis and demyelination, with varying degrees of demyelination among the cases. The syndrome, relating to a solitary lesion, was corticosteroid-sensitive, and it ultimately stabilized. Extensive longitudinal evaluations failed to reveal multiple sclerosis, infection, or neoplasm. Solitary inflammatory lesions add to the differential diagnosis of large, supratentorial, solitary space-occupying lesions noted on CT and MRI scans.

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The finding of an enhancing brain mass lesion on computed tomography (CT) or magnetic resonance imaging (MRI) studies often prompts a histologic diagnosis. Although biopsy may lead to a diagnosis of neoplasia, non-neoplastic lesions also are found. Inflammatory, demyelinating lesions rarely are indistinguishable from neoplasms on neuroimaging studies.<sup>1-16</sup> We present MRI, CT, and pathologic findings in five patients harboring solitary inflammatory, demyelinating lesions of the supratentorial white matter and gray-white junction, in whom the initial clinical and neuroimaging findings were consistent with a neoplasm.

## Materials and Methods

Computed tomography and MRI scans were reviewed (RB) and surgical specimens were examined (DNL) of five patients initially seen at Massachusetts General Hospital from 1977 to 1989. Magnetic resonance imaging studies, at 1.0 T or 1.5 T, included conventional spin echo T1-weighted images (T1-WI) and T2-weighted images (T2-WI). Histologic stains included H and E, Luxol fast blue, Bodian silver impregnation, and glial fibrillary acid protein.

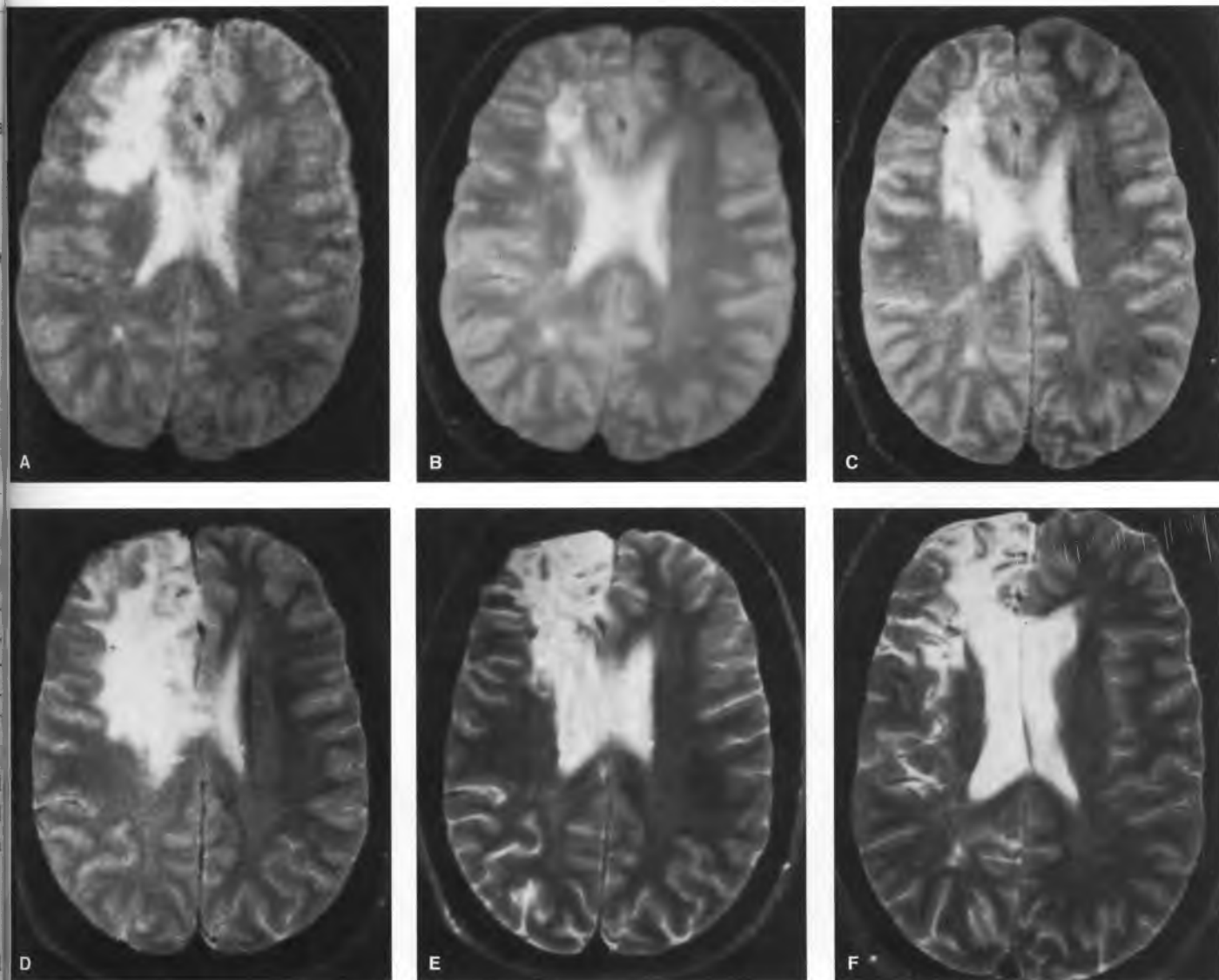
## Results

### Case Reports

**Patient 1:** A 19-year-old woman developed generalized seizures and a mild left hemiparesis. Computed tomography revealed a hypodense mass in the right frontal subcortical white matter, extending to the corpus callosum (Table 1). On MRI, the lesion was hypointense on T1-WI, hyperintense on T2-WI (Figs 1A, 2A), and showed patchy, intense enhancement (Fig 2B). Cerebrospinal fluid (CSF) findings are presented in Table 2. A brain biopsy revealed demyelination of white matter with axonal preservation,

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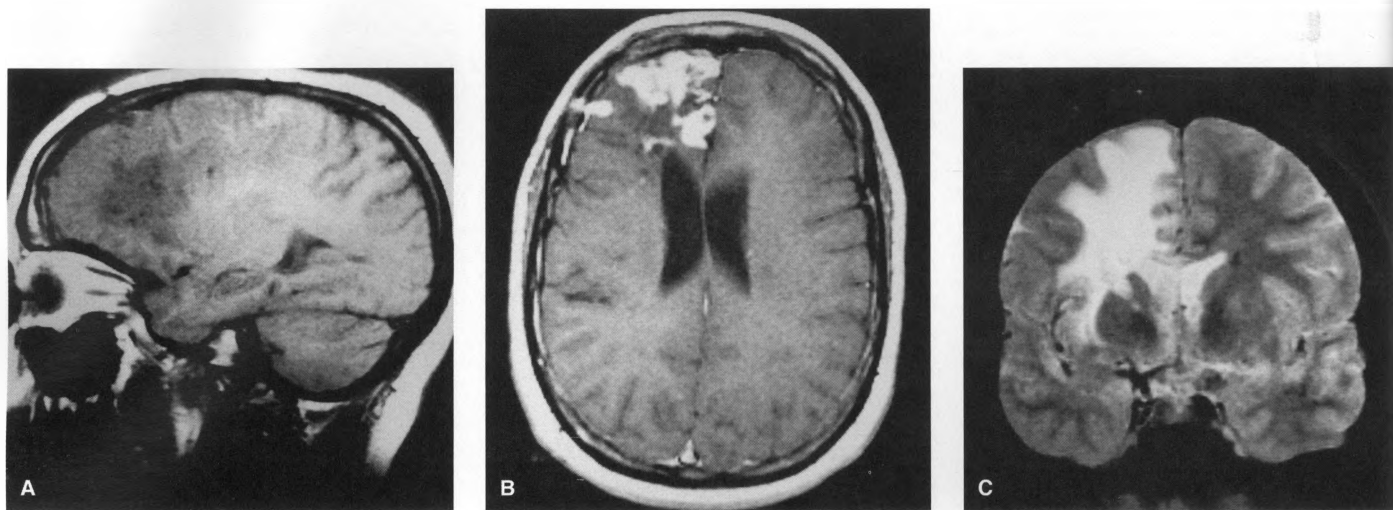


**Fig 1.** Patient 1. Serial axial MRI (T2-WI); hyperintense lesion. (A) 4/86 (onset): Right frontal periventricular white matter and gray-white junction lesion. (B) 7/86: Reduction in lesion size after corticosteroid therapy. (C) 2/87: Increase in lesion size. (D) 11/87: Further increase in lesion size. (E) 5/90: Reduction in lesion size after corticosteroid therapy. (F) 4/93: Atrophic lesion with ex-vacuo dilatation of the lateral ventricle.

perivascular lymphocytes, scattered microglia, parenchymal macrophages and reactive astrocytes. Three months later, corticosteroid therapy led to lesion size reduction (Fig 1B). Nineteen months after presentation, the mass progressively expanded (Figs 1C, D; 2C). Repeat biopsy revealed similar changes, including moderate numbers of chronic inflammatory cells, diffuse, incomplete demyelination with axonal and oligodendroglial preservation, and myelin-laden macrophages. Management with prednisone (80 mg/day) led to lesion size reduction (Fig 1E) without a change in seizures and hemiparesis. An open biopsy revealed similar changes. The lesion regressed during the next 6 years, becoming atrophic (Fig 1F), and nonenhancing (not shown). A frontal lobectomy (performed for seizure control) revealed similar findings. The patient's clinical

status and neuroimaging studies remained stable during the next 3 years.

**Patient 2:** A 66-year-old woman developed left hemiparesis, headaches, and ataxia 6 months after an illness characterized by adenopathy, fatigue, and an elevated Epstein-Barr virus viral capsid antigen antibody titer (1:5120). Computed tomography (not shown) revealed a diffusely enhancing right frontal periventricular white matter lesion associated with mild mass effect (Table 1). On MRI (not shown), the lesion spared the cortex; it was hypointense on T1-WI, hyperintense on T2-WI, and irregularly enhancing (not shown). Cerebrospinal fluid findings are presented in Table 2. An open biopsy revealed perivascular lymphocytes, reactive astrocytes, scattered microglia, parenchymal



**Fig 2.** Patient 1. Other MRI studies. (A) Frontal lesion, hypointense on sagittal non-contrast T1-WI. (B) Post-contrast axial T1-WI. Patchy intense enhancement. (C) Coronal T2-WI. Lesion progression, extensive involvement of the subcortical white matter.

mal macrophages, and demyelination with relative axonal and oligodendroglial preservation. Management with prednisone (40 mg/day) led to clinical improvement and lesion size reduction (not shown). Over the next 8 years, there was no recurrence of the mass.

**Patient 3:** A 54-year-old man developed a generalized seizure. Computed tomography revealed an enhancing lesion in the frontal lobe, exerting minimal mass effect (not shown). On MRI, the mass was located in the frontal white matter and gray-white junction; it was hypointense on T1-WI, hyperintense on T2-WI, and irregularly enhancing (Figs 3A, B). A stereotactic biopsy revealed demyelination of white matter with perivascular lymphocytes, reactive astrocytosis, prominent perivascular and parenchymal lipid-laden macrophages, scattered microglia, and focal areas of necrosis. Cerebrospinal fluid findings are presented in Table 2. Despite a transient corticosteroid response (Fig 3C), enlargement of the mass followed 2 months later on MRI (Fig 3D). Two repeat biopsies, including an open biopsy, revealed similar changes; results of electron microscopic and culture studies for fungi, parasites, viral inclusion bodies, or other microorganisms were unremarkable. The lesion ultimately became atrophic and nonenhancing (not shown). Seven years after presentation, the clinical status and neuroimaging studies remained stable.

**Patient 4:** A 37-year-old woman developed a right hemiparesis. A CT scan revealed a nonenhancing, supratentorial hypodense lesion (Fig 4A) in the periventricular white matter and gray-white junction. On MRI, it was hypointense on T1-WI and hyperintense on T2-WI, without mass effect or abnormal enhancement (Table 1). Cerebrospinal fluid findings are presented in Table 2. An open biopsy revealed demyelination of white matter with relative axonal and oligodendroglial preservation,

perivascular lymphocytes, reactive astrocytes, scattered microglia, and myelin-laden macrophages. Over the next 2 years, worsening hemiparesis and seizures coincident with enlargement of the mass partially responded to corticosteroids. Over the next 17 years, seizures and hemiparesis persisted. Serial MRI scans revealed encephalomalacia in the area of the prior mass, resulting in ex-vacuo dilatation of the lateral ventricle (Figs 4B, C). No new lesions appeared, and two repeat biopsies were unrevealing.

**Patient 5:** A 43-year-old woman developed a generalized seizure. A head CT revealed a homogeneously-enhancing hypodense lesion without mass effect (Figs 5A, B) in the occipito-parietal lobar white matter and gray-white junction, extending to the cortex. On MRI, it was hypointense on T1-WI, hyperintense on T2-WI, and homogeneously enhancing (Figs 5C-E). A stereotactic biopsy revealed abnormal white matter with perivascular lymphocytes, reactive astrocytes, scattered microglia, and mild demyelination with relative axonal and oligodendroglial preservation. A repeat biopsy revealed similar changes. Management with prednisone (40 mg/day) led to clinical improvement and lesion size reduction on MRI (not shown). Eight years after presentation, with clinical stability, MRI revealed a nonenhancing atrophic lesion (Fig 5F).

**Summary of Clinical Material:** Our five patients, ages 1 to 66, developed seizures and/or hemiparesis, secondary to solitary inflammatory mass lesions that regressed over months to years. Corticosteroid therapy was associated with improvement in each case. After 7-17 years, none of the patients showed clinical evidence for a second central nervous system lesion, active infection, neoplastic disease, or multiple sclerosis.

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Table 1. Summary of Neuroimaging Findings

Patient	Age/Sex	CT		MRI			Other CT/MRI Features			
		Unenhanced	+ C	T1	T2	+ C	Mass Effect	Ca+	Hemor	Location
1	19/F	Hypodense	Ring, Nodular	↓	↑	Nodular	None	None	None	F, Perivent; White, G-W jn > cortex
2	66/F	Hypodense	Irreg	↓	↑	Irreg	Mild	None	None	F, Perivent; White, G-W jn
3	54/M	Hypodense	Homo, Irreg	↓	↑	Homo, Irreg	Mild	None	None	F, Lobar; White, G-W jn > cortex
4	37/F	Hypodense	None	↓	↑	N/A	None	None	None	F-P-O, Perivent; White, G-W jn
5	43/F	Hypodense	Homo	↓	↑	Homo	None	None	None	P-O, Lobar; White, G-W jn > cortex

↓ = decreased signal; ↑ = increased signal (vs. gray matter); +C = postcontrast enhancement; Ca+ = calcification; F = frontal; G-W jn = gray-white junction; Hemor = hemorrhagic component; Homo = homogeneous; Irreg = irregular; N/A = not available; O = occipital; P = parietal; Perivent = periventricular; White = supratentorial white matter.

**Neuroimaging Findings:** Neuroimaging features indicated the possibility of a neoplasm (Table 1). Computed tomography revealed a solitary hypodense lesion associated with mild to no mass effect. On MRI, the lesions were hypointense on T1-WI and hyperintense on T2-WI. Enhancement was homogeneous/irregular (n = 3) or ring/nodular (n = 1). There was no evidence of calcification, hemorrhage, cystic change or increased vascularity. All of the lesions were located in the supratentorial white matter. Three lesions abutted the lateral ventricles; the other two were lobar. Ultimately, the lesions became atrophic and nonenhancing on MRI.

**Laboratory Testing:** Extensive CSF (Table 2) and other laboratory investigations did not disclose an infectious origin, neoplastic disease, multiple sclerosis, or other autoimmune origin.

**Pathologic Findings:** Each of the lesions showed an inflammatory process associated at least focally with demyelination. Perivascular lymphocytes, reactive astrocytosis, scattered microglia, myelin-laden parenchymal macrophages and relative axonal and oligodendroglial preserva-

tion was noted. Small, focal areas of necrosis were noted in only one patient (Patient 3). Vascular inflammation, hemorrhage, microglial nodules, inclusion bodies, neoplasia, and microorganisms were not present. Immunoperoxidase staining identified the perivascular lymphocytes to be predominantly T-cell (Patients 1, 3).

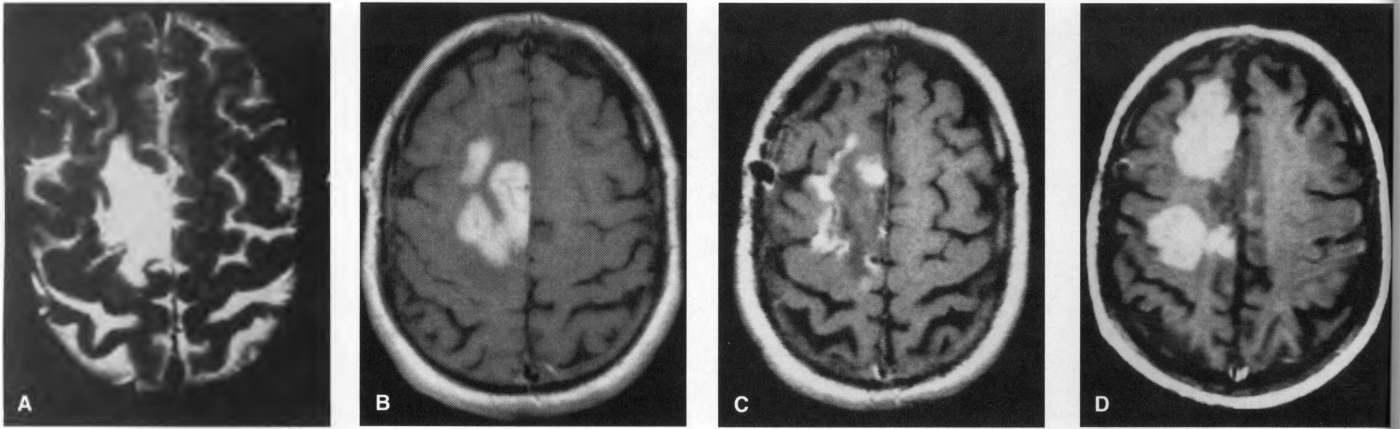
### Discussion

Our patients presented with focal cerebral signs and symptoms, including seizures and hemiparesis, attributable to solitary supratentorial brain mass lesions that were identified on MRI and CT scans. The neuroimaging findings indicated the possibility of a neoplasm, prompting a biopsy; however, an inflammatory, demyelinating process was revealed. The lesions involved the periventricular or lobar subcortical white matter with extension to the corticomedullary junction. They were diffusely hypodense on CT scans, hypointense on T1-WI, and hyperintense on T2-WI, probably reflecting the inflammatory demyelinating nature of the lesions. Enhancement was seen in nearly all of the lesions on CT and MRI scans, with patterns that ranged from diffuse/homogeneous to irregular/ring-like. The abnormal contrast enhancement in these lesions most

Table 2. Summary of CSF Findings

Patient	WBC/ (% differential)	RBC	Glucose (mg/dl)	Protein (mg/dl)	Negative Studies
1	4 (100L)	<100	55	49	Cytology, Bact, Fung, TB, Obands, IgG, Crypt, VDRL, Lyme, Encephalitis panel
2	14 (100L)	<100	67	38	Cytology, Bact, Fung, TB, Obands, IgG, Crypt, VDRL, Lyme, Encephalitis panel
3	2 (52M, 44L, 4N)	<100	80	51	Cytology, Bact, Fung, TB, Obands, IgG, Crypt, VDRL, Lyme
4	25 (84L, 16M)	<100	63	26	Cytology, Bact, Fung, TB, Obands, IgG, Crypt, VDRL, Encephalitis panel
5	N/A	N/A	N/A	N/A	N/A

Bact = routine bacterial culture; Crypt = cryptococcal antigen; CSF = cerebrospinal fluid; Encephalitis panel [31]; Fung = fungal culture; IgG = IgG Index (CSF/serum); L = lymphocytes; Lyme = antibodies to *Borrelia burgdorferi*; N = neutrophils; N/A = not available; Obands = oligoclonal bands; RBC = erythrocytes per high power field; TB = mycobacterial culture; VDRL = syphilis; WBC = white blood cells per high power field.

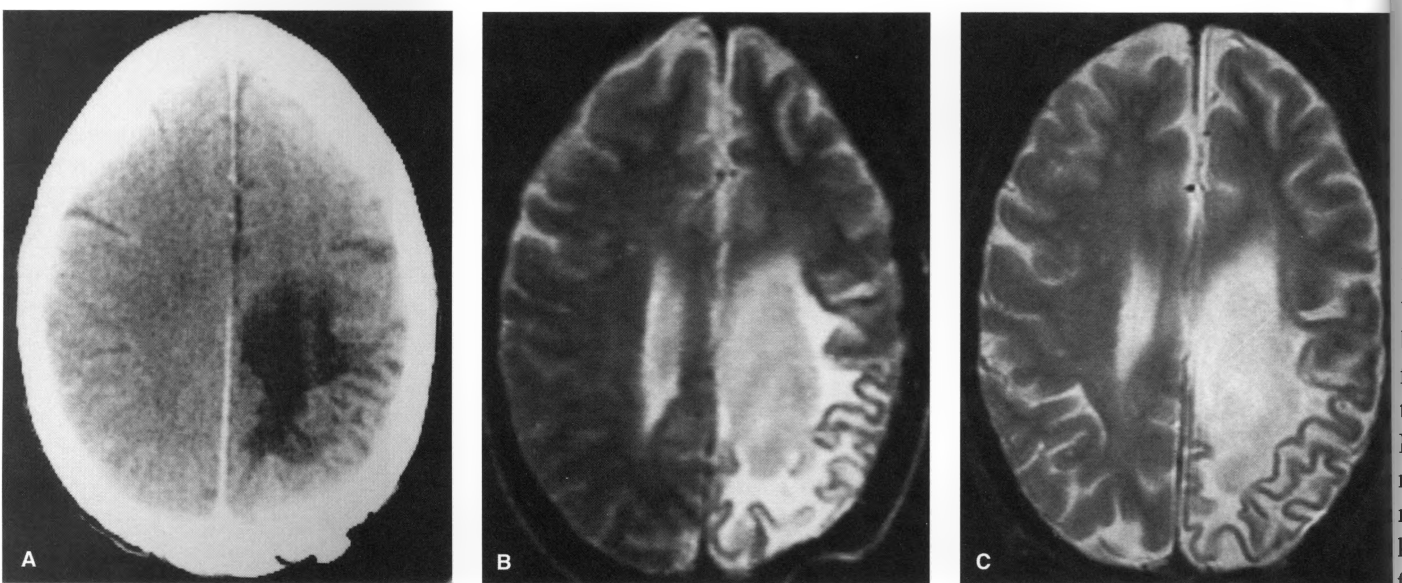


**Fig 3.** Patient 3. Serial axial MRI. (A, B) Initial presentation. Posterior frontal mass involving the lobar white matter and gray-white junction, hypointense on T1-WI (not shown), homogeneously hyperintense on T2-WI (A); homogeneous and irregular enhancement (B). (C) Three months after presentation. Corticosteroid therapy led to a marked reduction in the amount of enhancement. (D) Five months after presentation. Clinical deterioration. Recurrent, intense areas of enhancement in the dorsal frontal lobe and new areas of enhancement in the anterior frontal lobe.

likely reflects compromise of the blood-brain-barrier caused by active inflammation. Consistent with this hypothesis, the abnormal enhancement correlated with clinical worsening and resolved with corticosteroid therapy or clinical remission. Ultimately, the lesions decreased in size, became atrophic and failed to enhance. The initial neuroimaging findings of a single large enhancing supratentorial mass lesion raised the suspicion for a neoplasm, prompting a biopsy. Solitary inflammatory lesions should be added to the differential diagnosis for lesions with similar clinical and neuroimaging findings.

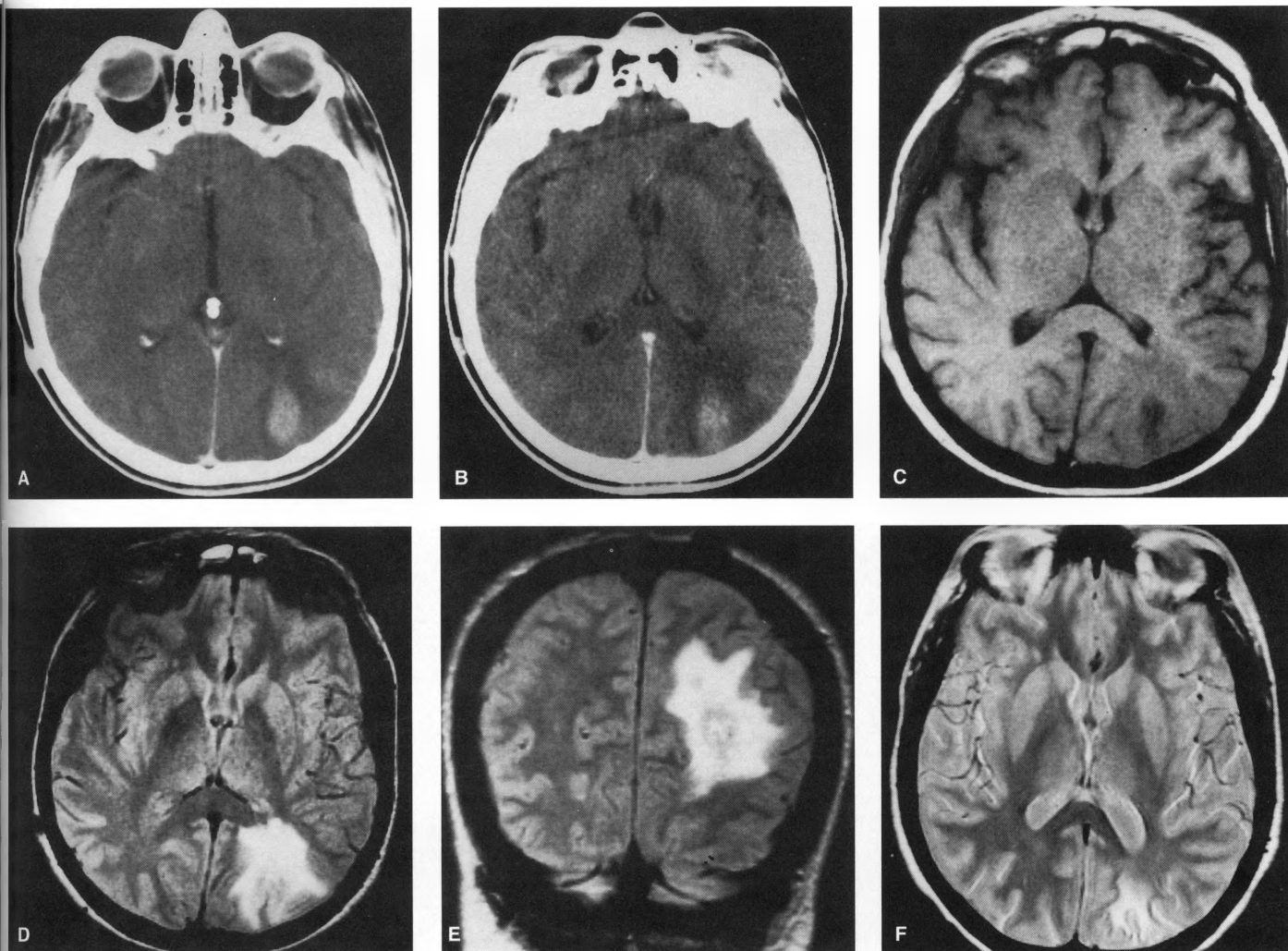
These lesions bear neuroimaging similarities to those of Kepes,<sup>16</sup> whose 28 of 31 patients developed tumor-like, single, solitary, corticosteroid-responsive demyelinating

brain masses. However, pathologically, the masses showed prominent demyelination with associated inflammation. In contrast, the current cases were characterized more by inflammation, resembling a leukoencephalitis, with the associated demyelination being less prominent and with oligodendroglia being preserved. Other white matter inflammatory demyelinating disorders occasionally resemble neoplasms on MRI or CT.<sup>1-15</sup> For example, patients with multiple sclerosis may develop large plaques, leading to biopsy.<sup>1-10</sup> These lesions share many of the neuroimaging and pathologic features of the present cases.<sup>2,3,5,8</sup> However, our patients clearly did not meet the diagnostic criteria for multiple sclerosis.<sup>17</sup> A second lesion was not found by longitudinal evaluation. Acute disseminated en-



**Fig 4.** Patient 4. CT: (A) Nonenhancing, hypodense lesion involving the periventricular, subcortical white matter and gray-white junction. MRI: (B) Eight years after presentation. Encephalomalacia within the area of the prior mass, associated ex-vacuo dilatation of the lateral ventricle. (C) Thirteen years after presentation. Stable lesion.

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**Fig 5.** Patient 5. CT: (A, B) Initial presentation: Left occipito-parietal homogeneously enhancing lesion without mass effect (hypodense on non-contrast images, not shown). MRI: (C-E) Initial presentation: Lobar white matter and gray-white junction lesion, extending to the gray-white junction; (C) Hypointense on T1-WI, homogeneously enhancing (not shown). (D, E) Hyperintense on T2-WI (proton density) (D = axial; E = coronal); hyperintensities resemble a peritumoral vasogenic edema pattern. (F) Nine years after presentation: Smaller, atrophic lesion; ex-vacuo ventricular dilatation; nonenhancing (not shown).

cephalomyelitis (ADEM) may show neuroimaging features indistinguishable from a neoplasm.<sup>8,13,14</sup> While pathologic similarities do exist between ADEM lesions and our cases,<sup>8</sup> the clinical course and neuroimaging findings are not consistent with typical ADEM; multiple intracranial lesions are typically noted on MRI.<sup>18</sup> Our cases bear histologic similarities to a leukoencephalitis that may follow Epstein-Barr virus infection.<sup>19,20</sup> One of our patients (Case 2) developed serologically confirmed Epstein-Barr virus infection 6 months before the onset of the neurologic symptoms. However, the clinical course and neuroimaging findings of post-Epstein-Barr virus leukoencephalitis are not similar to our cases.<sup>20</sup> Bacterial forms of leukoencephalitis, such as Whipple's disease<sup>21-24</sup> or Lyme disease,<sup>25</sup> can be excluded by the neuroimaging and pathologic findings. Rasmussen's chronic encephalitis is similar clinically to the current cases.<sup>26,27</sup> However, this

entity can be excluded by the pathologic<sup>26</sup> and neuroimaging<sup>27-29</sup> findings.

Similarities exist between our cases and a recent series of focal, corticosteroid-responsive, inflammatory demyelinating, contrast-enhancing brain masses.<sup>30</sup> However, each of these patients developed new, separate, symptomatic brain lesions several months later that were diagnosed as primary central nervous system lymphoma. This entity is excluded by the longitudinal follow-up failing to reveal lymphoma in our cases. In addition, many of these "sentinel lesions of lymphoma" were located in deeper brain regions, such as the brain stem, internal capsule, and hypothalamus, unlike the current cases.

The etiology of these unusual, solitary, supratentorial, inflammatory demyelinating, space-occupying lesions remains speculative. The significant improvement after corticosteroid therapy, followed by the ultimate regression of

the masses, suggests that this syndrome may represent one or more inflammatory disorders, such as a solitary form of a syndrome similar to ADEM or multiple sclerosis or an undetermined insidious infection. The clinical and pathologic findings favored a diagnosis of inflammatory leukoencephalitis; however, the initial neuroimaging findings indicated the possibility of a neoplasm. Because of the nonspecific neuroimaging and pathologic features and an uncertain etiology, this syndrome may be referred to simply as "leukoencephalitis."

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