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Migraine: Evaluation by MR

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Twenty-four patients clinically diagnosed as having migraine (17 of the classic or common type and seven of the complicated type) were evaluated on a 0.5-T or 0.6-T superconductive MR imaging unit with the objective of detecting associated parenchymal lesions. Thirteen (54%) of the patients had normal MR studies. Eleven (46%) of the patients (seven with classic or common and four with complicated migraine) showed well-defined lesions with prolonged T2 signal intensity. The lesions associated with classic migraine were focal and predominantly distributed in the periventricular white matter, bilateral in four and unilateral in three. In the group with complicated migraine, larger cortical abnormalities similar to infarcts were seen in three patients and multiple bilateral focal white matter lesions were seen in one. Almost all the lesions were evident only on T2-weighted studies; a few exhibited hypointense signal intensity on T1-weighted studies. The focal periventricular white matter lesions were not necessarily associated with neurologic deficits, but the cortical lesions were.

Our study indicates that parenchymal changes are frequently associated with migraine and that MR may well be the screening and diagnostic method of choice for their detection and evaluation.

Headache is a very common problem, affecting the majority of individuals in Western cultures. Of these, approximately 15–20% suffer from migraine type headaches [1]. Classic migraine, the most common type, is clinically recognized as a form of severe, usually unilateral recurrent throbbing headache, which is preceded by visual symptoms (the "aura"), and responds to ergotamine [2]. Similar headaches without visual symptoms are referred to as common migraine. Migraine associated with neurologic deficits, which are usually transient, is referred to as complicated migraine, a much less common entity [2].

The reported findings in migraine from numerous imaging techniques (CT, angiography, positron emission tomography) have been variable (see Discussion). MR imaging has emerged as a diagnostic tool with superb sensitivity in detecting early and subtle alterations in brain parenchyma, prompting us to use this method to evaluate patients with migraine.

Materials and Methods

Twenty-four patients, eight males and sixteen females, ages 15–55 years, clinically diagnosed as having migraine, had MR studies of the brain on a Picker 1.0-T superconductive MR unit* operating at 0.5 T (21 patients) or a Technicare 0.6-T superconductive MR unit† (three patients). One patient was scanned on both units. Seventeen had clinically determined classic or common migraine, and seven had complicated migraine. All patients had MR studies of the brain using 2500/100 (TR/TE) T2-weighted 10-mm spin-echo images in the axial and coronal planes and 600/40 T1-weighted 5-mm spin-echo images in the sagittal plane. Images had 256 × 256 matrixes and were obtained from a 30-cm field of view.

One patient was scanned within 24 hr of onset of symptoms of dysphasia and had follow-up scans 3 weeks and 4 months later; the other patients were studied at random. The

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* Vista 2055, Picker International, Highland Heights, OH.

† Teslacon I, Technicare Corp., Solon Springs, OH.

location and signal intensity of lesions were noted on both T1- and T2-weighted studies. Attempts were also made to correlate the parenchymal changes neurologically.

Results

Seven of the 17 patients with common or classic migraine and four of the seven patients with complicated migraine had abnormal MR findings (see Table 1). The findings consisted of focal regions of increased signal intensity on T2-weighted studies (Fig. 1). In the patients with classic or common migraine, the lesions were seen exclusively within white matter (unilaterally in three and bilaterally in four), while three of the patients with complicated migraine had lesions involving the cerebral or cerebellar cortex unilaterally. In a patient who had an MR within 24 hr of onset of dysphasia, T2-weighted images showed regions of increased signal intensity in the left centrum semiovale and insular cortex, as well as small foci of

increased signal intensity in the left cerebral white matter (Fig. 2). CT was normal. This patient was treated with nifedipine and recovered. A follow-up MR study obtained 3 weeks later showed an increase in the size of the lesions in the insular cortex and frontal operculum despite the resolution of the neurologic deficits. Of the other six patients with complicated migraine, one had multiple tiny foci of increased signal in the cerebral white matter bilaterally, one had increased signal in the cerebral cortex consistent with an infarct (Fig. 3), and one had involvement of the cerebellar cortex (Fig. 4). The remaining three had normal MR findings. The cortical abnormalities appropriately correlated with the patients' neurologic deficits, which all resolved within a few days after the migrainous attacks. The smaller white matter lesions seen in patients with classic or common migraine could not be accounted for neurologically.

Discussion

Complex and variable etiologic considerations and observations have evolved from numerous studies in the past on patients with migraine. The most widely accepted hypothetical basis for migraine headache is that initial vasoconstriction of extra- and intracranial arteries takes place, leading to ischemia (clinically manifested by the "aura" and neurologic deficits), followed by reactive vasodilatation and hyperemia resulting in the development of headache [3-5]. Numerous factors are thought to account for the vascular events, including the presence of circulating vasoactive substances such as serotonin [6] and histamine [7], sensitivity to ingested substances such as nitrites [8], and inherent abnormalities of the autonomic nervous system in patients with migraine [9].

Investigators using positron emission tomography have demonstrated decreased regional glucose metabolism in the preheadache phase, which rose but remained below resting levels during the headache phase [10]. Oxygen inhalation studies have revealed decreased blood flow and diminished oxygen extraction in regions of migraine infarction as demonstrated on CT [11]. Xenon-133 blood studies have substantiated the occurrence of decreased cortical blood flow

TABLE 1: Distribution of Patients by Age and Gender

	Positive Study		Negative Study	
	Age	Gender	Age	Gender
Under 40 years old	28	F	18	M
	31	F ^a	20	M ^a
	38	F	27	F
	38	F	27	F
	39	M ^a	30	F
			32	F
			34	F
			35	M
			36	F
			40	F ^a
Over 40 years old	41	M	42	M ^a
	43	F ^a	44	M
	44	F	55	M
	47	F ^a		
	51	F		
Total	11		13	

^a Neurologic deficits.

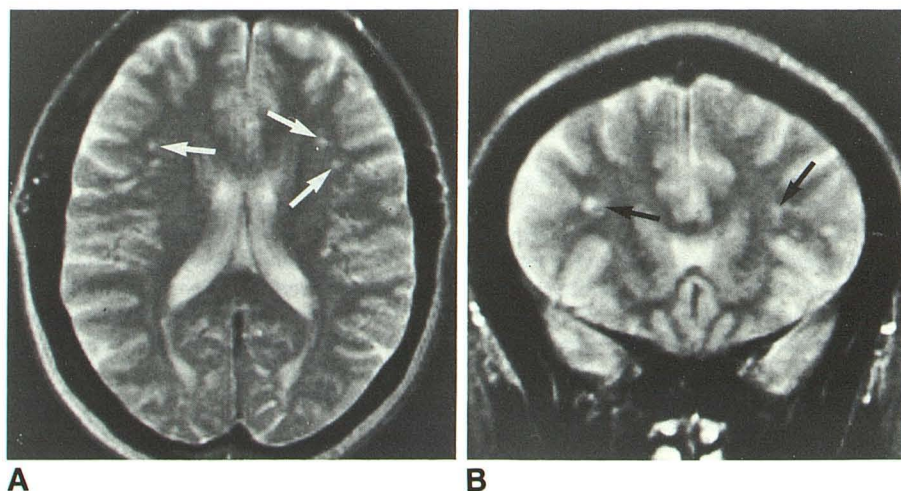


Fig. 1.—A and B, Axial (A) and coronal (B) T2-weighted MR images of 44-year-old woman with classic migraine. Multiple small foci of abnormally increased signal intensity are present in cerebral white matter bilaterally (arrows).

Fig. 2.—31-year-old woman with migraine who experienced a sudden onset of difficulty speaking.

A and B, MR study performed within 24 hr of the attack shows focal regions of increased signal in left centrum semiovale (*arrow, A*), frontal white matter, and insular cortex (*arrows, B*). There is also diffusely increased signal involving the sylvian and frontal cortex (*arrowheads, B*).

C, Follow-up MR image 3 weeks later shows that the abnormality in the sylvian and frontal cortex is more pronounced (*arrowheads*) despite clinical resolution of symptoms.

D, 4 months later, cortical abnormality is still faintly visible in sylvian region (*arrow*). Lesion in centrum semiovale was unchanged.

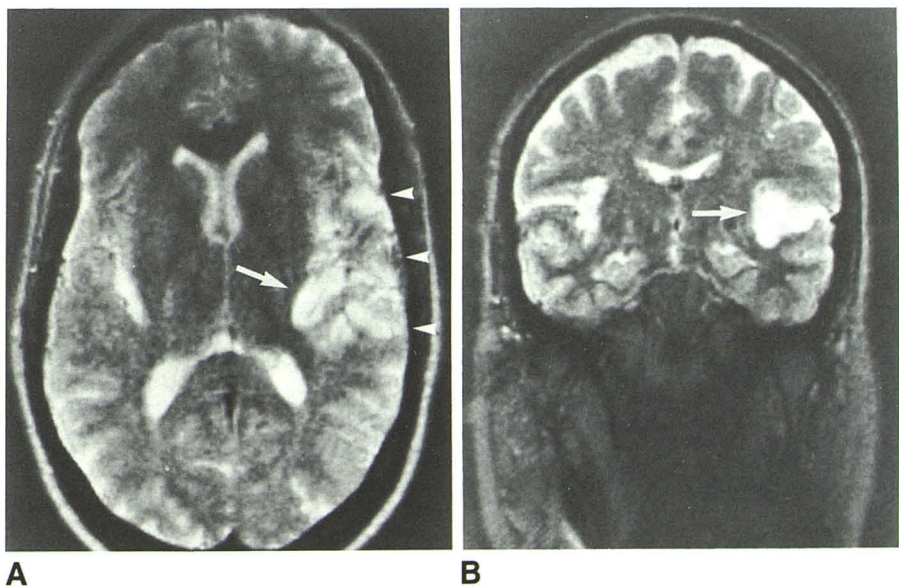
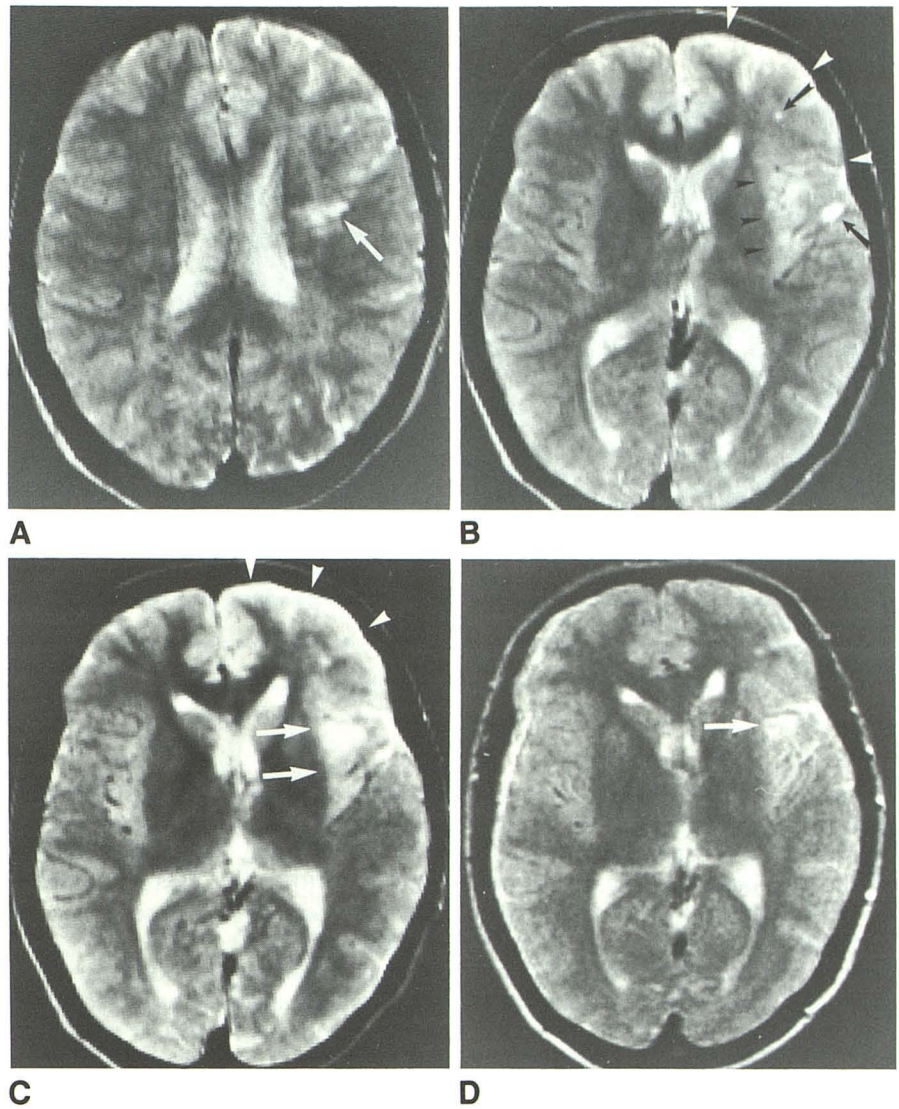


Fig. 3.—**A and B,** 43-year-old woman with classic migraine who presented with aphasia and right hemiparesis. MR image obtained several days later demonstrates increased signal in left cerebral cortex (*arrows, A and B*) centered around insula (*arrowheads, A*).

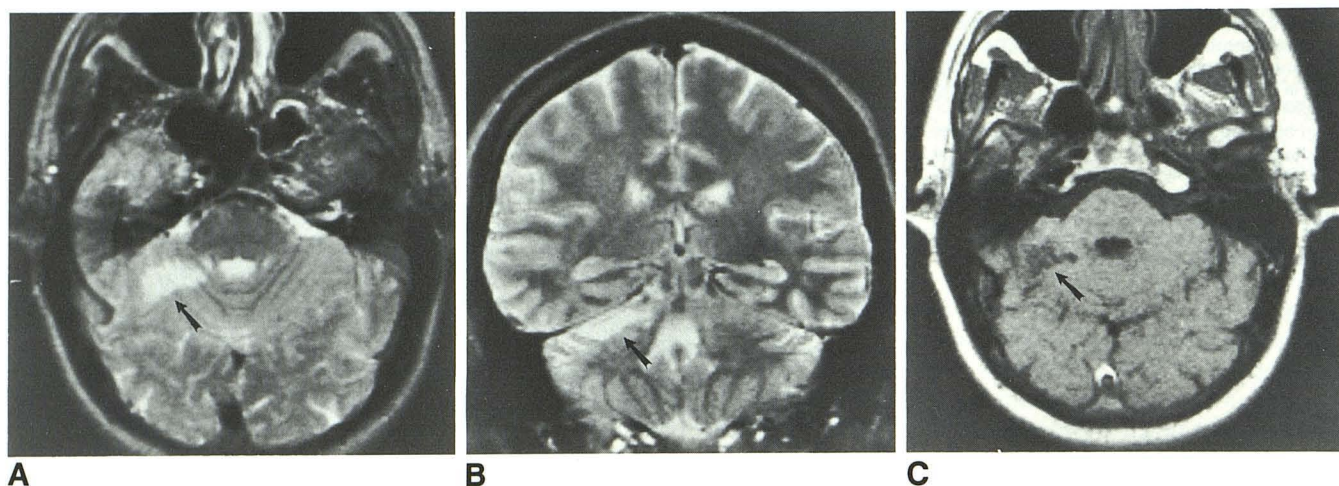


Fig. 4.—39-year-old man who had several episodes of severe headache associated with right-sided weakness, ataxia, and slurred speech. He was diagnosed as having vertebrobasilar migraine.

A and B, MR obtained several months after last episode shows abnormally increased signal in right cerebellar hemisphere on T2-weighted images (arrow, A and B).

C, T1-weighted image shows abnormality as a region of decreased signal (arrow).

and postischemic hyperemia with increased cerebral blood flow on the symptomatic side in classic migraine [12, 13]. Angiography has occasionally demonstrated vascular spasm or dilatation, but in most cases has been normal [14–16].

The frequency of parenchymal abnormalities associated with migraine is indeterminate. Reported CT observations on patients with migraine have ranged from all normal in a large series of 435 patients [17] to findings of cerebral edema and infarctions [14, 16, 18, 19]. The most consistent finding was an increased incidence of atrophy [20–22].

In our series, MR showed parenchymal abnormalities in 41% of patients with classic or common migraine and in 57% of patients with complicated migraine. These consisted mainly of focal lesions with prolonged T2 signal intensity that were distributed exclusively in white matter in those with common or classic migraine. These small lesions were not associated with overt neurologic dysfunctions. Cortical lesions resembling infarcts were seen in some patients with complicated migraine in regions appropriately correlating with the patients' neurologic deficits.

Any process that increases the water content of a tissue, thereby prolonging its T2 relaxation time, will result in increased signal intensity in the affected region on MR images with long TR and TE (T2-weighted) pulse sequences. In white matter, increased water content may be due to either loss of the hydrophobic myelin (demyelination) or increased interstitial water (edema, hydrocephalus) [23].

Patchy foci of increased signal intensity in white matter is a fairly common normal finding in the elderly, in whom an incidence of 20–30% has been reported [24]. However, they are rarely found in normal individuals under 45 years old [25, 26]. These lesions have been attributed to ischemic demyelination and lacunar infarcts [25–28]. Kirkpatrick and Hayman [29] examined pathologic preparations of 15 brains of clinically healthy subjects ages 52–72 years to determine the fre-

quency and nature of white matter lesions. They found white matter lesions in 12 (80%) of the 15. These included atrophic periventricular demyelination in eight, two white matter infarcts, four vascular malformations (three telangiectases and one capillary hemangioma), and three small ventricular diverticula.

The white matter lesions encountered in patients with migraine resemble those of multiple sclerosis and the periventricular white matter lesions of small vessel atherosclerotic disease (subcortical arteriosclerotic encephalopathy). However, the dominant clinical feature in our patient population is headache, which is not described as a common presenting manifestation of multiple sclerosis [30] and arteriosclerotic cerebrovascular disease [28, 31, 32]. Furthermore, even limiting our observations to patients under 40 years old to reduce the overlap with age-related white matter lesions, we still find an incidence of 27% in patients with the classic or common type of migraine and 67% for those with complicated migraine.

We could not offer histologic correlation for the visualized lesions nor can we infer their cause. However, our study demonstrated a high frequency of white matter and cortical changes in patients with migraine and indicates that MR may well be the diagnostic technique of choice for detecting them.

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