

an arteriovenous malformation involving the caudate nucleus contralateral to the direction of neck rotation (8), lacunar infarction in the contralateral putamen (9), traumatic infarctions in the caudate nucleus and putamen (structures involved in case 1) (10,11), and a unilateral lesion in the centrum semiovale (12). The case of one patient with torticollis associated with MS and bilateral MRI lesions in the pallidum was described (1), but in the majority of cases the location of the demyelinating lesion was not stated (2,3,13). Plant et al. (6) reported a case of MS with spasmodic torticollis and a midbrain lesion. We also found a lesion at the level of pontomesencephalic junction in case 1. The abrupt onset in case 1 would somewhat favor an association with MS; in case 2, the remissions of spasmodic torticollis in time-locked fashion with two consecutive remissions of MS is suggestive of the same association.

Some patients with spasmodic torticollis have complete or partial remissions of their symptoms (14). In case 2, however, the two episodes of transient torticollis occurred over only 7 months and correlated with the clinical remissions of MS. However, MRI in this patient revealed only nonspecific diffuse changes. In conclusion, although the association of MS and spasmodic torticollis in particular may usually be coincidental (1), the clinical correlations in case 2 suggest that MS lesions might also be the pathologic substrate of the involuntary movements.

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Painful Tonic Spasms and Pure Motor Hemiparesis Due to Lacunar Pontine Infarct

Epileptic seizures are well-recognized manifestations of cerebrovascular disease (1). Paroxysmal repetitive involuntary movements have been described with hemodynamic carotid artery transient ischemic attacks (2,3), and clonic movements with limb posturing may result from vertebrobasilar occlusions (4). Lacunar infarcts rarely cause epileptic seizures (1), but can give rise to chorea, hemiballismus (5), or dystonia (6). Paroxysmal tonic spasms, most commonly seen in multiple sclerosis, are brief recurrent spasms that readily respond to treatment with carbamazepine (7). Tonic spasms typical of those seen in multiple sclerosis have been reported as being caused by lacunar infarcts involving the putamen (8) and the pons (9). We report the case of a second patient with a pure motor hemiparesis and recurrent painful tonic spasms involving both hemiparetic limbs due to contralateral pontine lacunar infarction.

Case Report

A 57-year-old right-handed man with insulin-dependent diabetes mellitus, ischemic heart disease, and hypertension was transferred to our center for investigation. Eleven days previously, he woke with weakness affecting his left arm, leg, and face. This developed over the following 2 days into a complete hemiplegia, and he complained of a sensation like "running water" over his left arm and leg. He noticed paroxysmal painful spasms affecting his left arm and leg that were typically preceded by a dull occipital headache and nausea. There were no obvious precipitating factors, and the episodes lasted up to 2 min. During these episodes, he retained consciousness but became anxious. Afterward, he noted increased weakness of his arm and leg, which would subside over the next few hours. On examination between attacks, his higher mental function was normal. He was slightly dysarthric, but reported no dysphasia. He had a left upper motor neuron facial weakness, but the remainder of the cranial nerves were normal. There was flaccid weakness affecting his left arm and leg, with no voluntary movement (grade 0) in the left arm and moderate pyramidal weakness (grade 3-4) in the left leg. His reflexes were brisk on the left, but both plantar responses were flexor. Sensation was intact.

During each spasm, his left arm was adducted to his chest and flexed at the elbow, wrist, and fingers. His left leg was extended at the hip and knee, and there was plantar flexion at the ankle. Attempted movement of his arm exacerbated the severe pain, and movement of his leg was impossible due to intense muscle spasm. The patient remained fully conscious throughout, with normal movement of his right-sided limbs, but he became anxious. One episode lasted longer than usual and,

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after 5 min, he was given 10 mg of intravenous diazepam, which rapidly relieved the spasm. Immediately afterward, there was increased weakness of his left side, with hyperreflexia and sustained clonus at the ankle and an extensor plantar response. On review 12 hours later, the hyperreflexia had diminished and the left plantar was equivocal. Apart from the duration, previous episodes had been identical.

Investigations revealed a normal full blood count, urea and electrolytes, clotting, and erythrocyte sedimentation rate; a *Treponema pallidum* hemagglutinin test was negative. The findings on computed tomographic brain scan were normal, but magnetic resonance imaging revealed a lacunar infarct in the right ventral pons (Fig. 1) and several small scattered lesions in the region of the left basal ganglia. The findings on magnetic resonance angiogram were normal. A carotid duplex ultrasound showed mild (30%) bilateral internal carotid artery stenosis but good flow in both distal internal carotid arteries. Transcranial color Doppler revealed normal middle, anterior, and posterior cerebral artery flow velocities. The patient was administered carbamazepine 100 mg twice daily, which reduced the frequency of spasms, and the left hemiparesis gradually improved. Following discharge, the patient continued to experience brief spasms occasionally despite increasing carbamazepine to a dose of 200 mg twice daily.

Discussion

Tonic spasms are brief, frequent, intensely painful episodes in which the limbs on one side adopt a tonic posture. Typically, this involves adduction of the shoulder; flexion of the elbow and wrist; and a tetanic posture, extension, or clenching of the fingers. The legs are commonly extended at the hips and knees, with plantar flexion at the ankle. The spasms are most com-

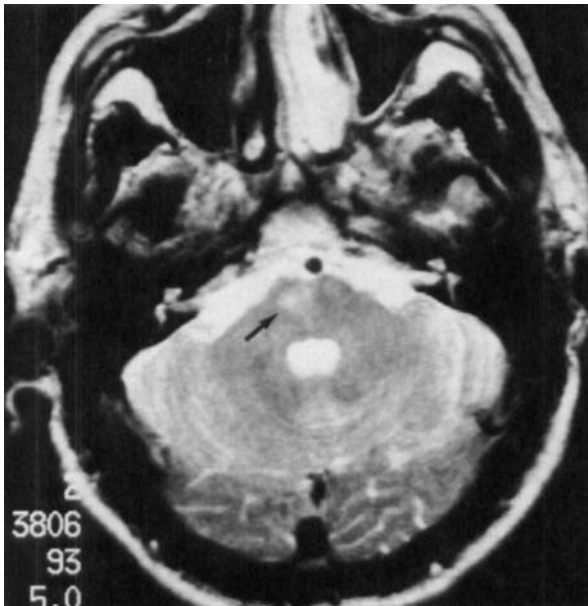


FIG. 1. Magnetic resonance image at the level of the exit of the facial nerve showing high signal lesion (arrow) involving the right ventral pons, consistent with lacunar infarction.

monly seen as a paroxysmal symptom of multiple sclerosis (7); may be triggered by movement, sensory stimulation, or hyperventilation; and usually resolve after a few weeks (7). Kaufman et al. (9) recently reported a patient with a mild motor hemiparesis caused by a brainstem infarct and frequent extensor spasms of the left leg lasting 2–3 seconds and recurring every 30 seconds. Spasms were precipitated by attempts to move and tended to be more common at night. The spasms in our patient were more prolonged, typically lasting 1–2 min but on at least one occasion >5 min, and clinically similar to the tonic spasms seen in multiple sclerosis. During one prolonged spasm, our patient was noted to be hyperventilating. It is unclear whether this precipitated the spasms, but prolonged hyperventilation might have exacerbated his attacks, as occurs in patients with multiple sclerosis (7).

As in the case reported by Kaufman et al. (9), tonic spasms were preceded by a dull occipital headache and nausea. Following each spasm, there was increased weakness, a marked increase in tone, and sustained clonus at the ankle. This is similar to the Todd's paresis that may follow partial seizures, but it also has been noted following tonic spasms due to demyelination of the contralateral cerebral peduncle in the context of multiple sclerosis and associated with functional impairment of corticospinal pathways (10). Epilepsy seems an unlikely mechanism for the spasms seen in our patient, because acute lacunar, subcortical, and brainstem infarcts are not usually associated with epileptic seizures (1). The patient remained fully conscious throughout the episode and was fully conversant. Unfortunately, we were unable to obtain an electroencephalogram during the episodes, but epileptiform activity was not detected during tonic spasms in the patient reported by Kaufman et al (9). Episodes similar to tonic spasms have resulted from epileptic seizures arising from lesions on the medial surface of the hemisphere (11), but, in our patient, the responsible infarct was in the right ventral pons. Various repetitive movements, including limb posturing and head jerking, and clonic movements resembling seizures have been reported in association with brainstem and cerebellar signs due to occlusion of the vertebrobasilar arteries (6). In contrast to the patient reported here, the clonic movements in these patients are contralateral to the hemiparesis (6); in addition, magnetic resonance angiography did not reveal any evidence of vertebrobasilar occlusion, making this unlikely in our patient.

Repetitive involuntary movements described as uncontrollable jerking or shaking have been described in patients with severe contralateral carotid stenosis or occlusion. These usually take the form of rhythmic involuntary movements of one arm or leg, precipitated by standing, walking, or hyperextension of the neck, and are promptly terminated by sitting or lying down (2). They are thought to be caused by transient hemodynamic cerebral ischemia (3) and often resolve when cerebral blood flow is improved by endarterectomy or bypass surgery (2). Given the absence of hemodynamically significant carotid stenosis, this would seem an unlikely mechanism causing spasms in this patient. Painful tonic spasms have also been reported with lacunar infarcts affecting the putamen (8), but lacunar infarcts involving the basal ganglia more commonly produce hemiballismus, hemichorea (5), or dystonia (6).

Tonic spasms of multiple sclerosis usually respond to carbamazepine (7), but an incomplete response was noted in our patient, with reduced frequency of spasms. During a prolonged

episode, rapid relief was noted with intravenous administration of diazepam, as reported in an earlier case (8).

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Unilateral Postural and Action Tremor Resulting from Thalamic Toxoplasmosis in a Patient With Acquired Immunodeficiency Syndrome

Although neurologic complications of acquired immunodeficiency syndrome (AIDS) frequently manifest quite early in the course of disease, movement disorders still seem relatively rare, among which hemichorea-hemiballismus is most typical of a toxoplasmic abscess (1).

Tremor, either postural or kinetic, is an early and common finding in patients with human immunodeficiency virus (HIV⁺; 2) but is a rare manifestation of toxoplasmosis (3). Whereas in toxoplasmosis, tremor is usually associated with other movement disorders such as dystonia or myoclonus (1), isolated tremor has been reported only as a single case (3) featuring

wing-beating tremor resulting from a midbrain toxoplasmic abscess.

Case Report

A 39-year-old man had a history of alcoholism and intravenous substance abuse for >12 years. At age 31 years, serum proved positive for HIV, and herpes zoster, onychomycosis, and Sicca syndrome developed successively. After suitable treatment, he became symptom free up to November 1993, when concentration impairment became noticeable. A month later, paresthesias suddenly developed on the right side, followed by severe ipsilateral upper-limb weakness lasting a few minutes. During the following 2 weeks he had two such episodes with total recovery of muscular strength and sensation, and later he became verbally aggressive and asthenic, reporting difficulty in judging distance. In January 1994, right upper-limb paresis associated with slight ipsilateral lower-limb weakness progressively developed, culminating at 20 days in a severe right hemiparesis.

At neurologic examination, the patient was fully alert, cooperative, oriented in time and space, and deeply concerned about his condition. Ocular fundus, visual fields, and ocular movements were normal. A severe right hemiparesis with brisk right deep tendon reflexes and a right up-going toe were evident, but sensory abnormalities and meningeal signs were lacking.

A brain computed tomography (CT) scan disclosed an enhancing lesion in the left posterior thalamic region and in the posterior arm of the internal capsule, confirmed at magnetic resonance imaging (MRI) as a hyperintense T₂-weighted mass (Fig. 1) markedly enhancing with gadolinium at T₁-weighted imaging.

Laboratory findings included reduced CD4 count (160/μL) and positive immunoglobulin G (IgG) for toxoplasmosis in the serum (1/2,048), whereas VDRL, HBS Ag, anti-HBC, and serologic findings were negative. He was given zidovudine, sulfadiazine, and pyrimethamine.

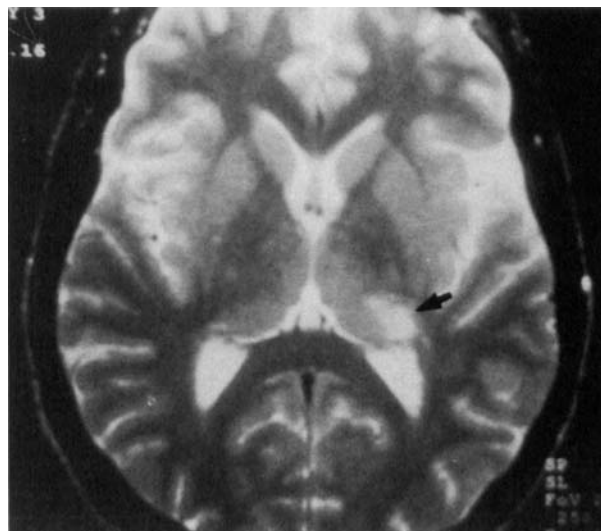


FIG. 1. T2-weighted magnetic resonance imaging scan showing hyperintense signal in the left posterior thalamic region as well as in the posterior arm of the internal capsule (arrow).

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