fluid (CSF) because of loculation or morphological derangement, or both. Diminished CSF flow in these regions would account for the relative increase in signal intensity, as compared to the remainder of the central thecal sac. This finding supports recent observations regarding the role of decreased CSF resorption in this disorder [12]. Abnormal resorption may be secondary to local inflammatory changes from underlying arachnoiditis.

The management of cauda equina syndrome of ankylosing spondylitis is uncertain. Surgical intervention has been unsuccessful or has resulted in further neurological disability in several instances [2, 3, 6]. Recent reports have advocated surgery when CSF collections cause mechanical symptoms [9, 12]. Consideration of surgery should be reserved for those instances in which neural compression clearly has caused symptoms. The presence of arachnoiditis and the poor results following surgery in other series, and reports of spontaneous improvement and stabilization in this disorder, demand a cautious approach [6, 7, 13].

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Sensory Level and Parietal Lobe Hemorrhage

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ABSTRACT

A 39-year-old woman with a right sensory level at T3 involving touch, pain, temperature, vibration, and position was found to have a small hemorrhage in the superior part of the postcentral gyrus. This case points out an unusual clinical presentation of parietal lobe lesion and provides clinicoanatomical correlation of the somatotopic organization of the sensory cortex. Furthermore, it confirms that sensory disturbance with a segmental level suggestive of a spinal cord lesion can be caused by a parietal lesion.

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A sensory level is classically associated with a spinal cord lesion. The unusual presentation of a patient with a misleading thoracic sensory level associated with a parietal lesion is discussed.

Case Report

A healthy 39-year-old woman woke up 1 week after ambulatory septoplasty with the feeling of a cold right leg. She also felt numbness and pins and needles on her right hemibody up to the right shoulder. Her right arm and face were spared. When she stood up, she noticed gait difficulty, mostly related to the absence of feeling the floor under her right foot.

The patient had no headache and no history of head trauma, loss of consciousness, seizures, hypertension, or change in mental state.

On physical examination 5 hours later, functioning of the cranial nerves was within normal range. No meningeal sign or Lhermitte sign was present. Tendon reflexes were 3+ on the right and 2+ on the left and the plantar response was indifferent on both sides. Motor examination revealed normal findings, but the right leg was unsteady on the Barré maneuver. The patient was unsteady standing on the right leg. On sensory examination, there was a sensory deficit to all modalities (touch, pain, temperature, vibration, and posture) with a clear level at T3 on the right shoulder. The right arm and face as well as the left side of the body were spared. She was slightly dysmetric on the right on the heel-to-knee maneuver, but there was no ataxia in the upper extremities. Findings on general examination were normal and included a blood pressure of 140/90 mm Hg,

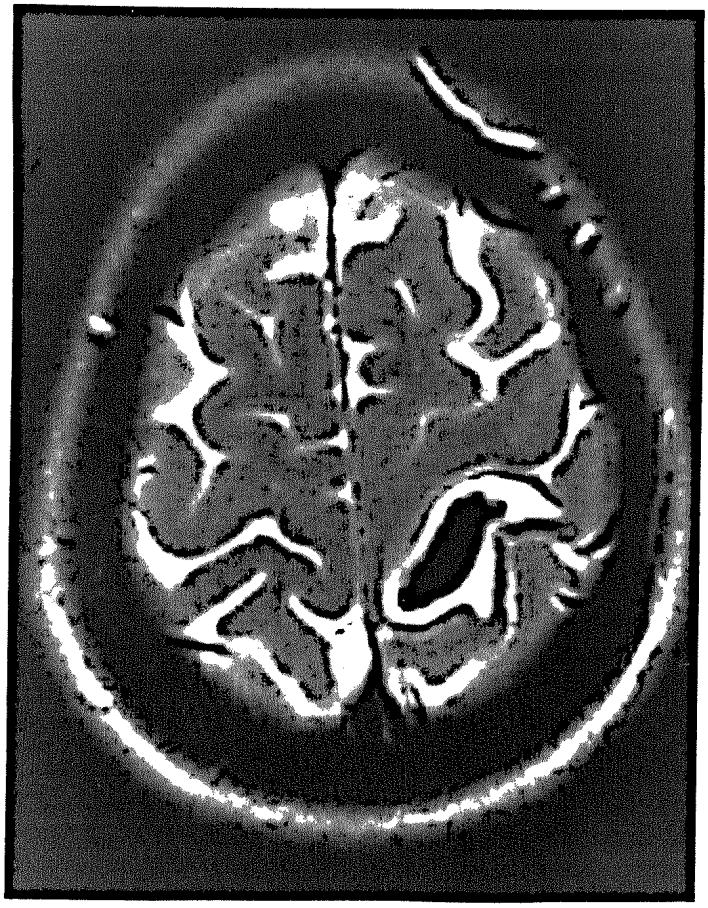

Fig 1. MRI showing a superficial lesion 1 × 2 cm in area in the superior part of the postcentral gyrus, moderately hypointense on T2-weighted image and surrounded by a hyperintense ring of less than 1 cm in diameter, suggestive of hemorrhage.

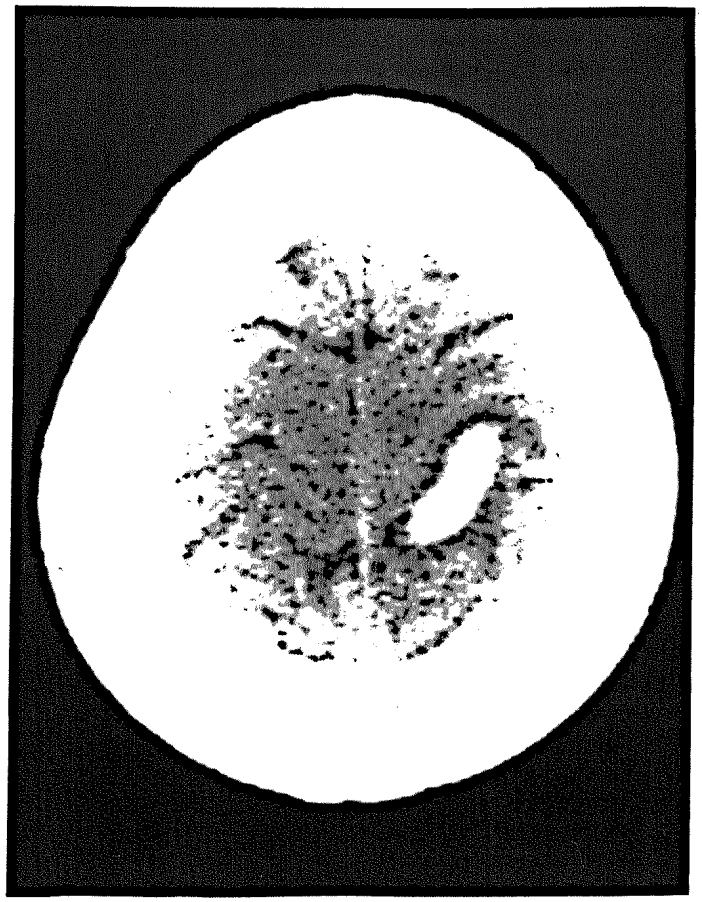

Fig 2. CT showing the same lesion as in Figure 1. Note the absence of calcifications, which supports the hypothesis of a hemorrhage (calcifications are often seen in benign tumors [e.g., oligodendroglioma]).

heart rate of 90/min, and temperature of 37°C.

The complete blood cell count, blood chemistry results, and chest x-ray appearance were normal. Magnetic resonance imaging (MRI) of the spine the day after the onset was normal, but MRI of the head showed a superficial lesion 1 × 2 cm in area in the superior part of the left postcentral gyrus, moderately hypointense on T2-weighted images and surrounded by a hyperintense ring of less than 1 cm in diameter, suggesting acute hemorrhage (Fig 1). Cerebral computed tomography (CT) supported this hypothesis (Fig 2). A left carotid arteriogram performed 8 days after onset failed to demonstrate any arteriovenous malformation. There was no cortical somatosensory evoked response to stimulation of the right leg. The other evoked potentials were normal.

The cerebrospinal fluid (CSF) was normal. Blood and CSF serology for Lyme disease and syphilis, rheumatoid factor, and antinuclear antibodies were negative.

Comment

To the authors' best knowledge this is the second report of a sensory deficit suggestive of a spinal lesion but due in fact to a postcentral stroke [1]. Sensory loss caused by parietal lesions usually predominates in the upper limb and distally, does not reach the midline, and may not have a sharp demarcation [2, 3]. Some areas (e.g., the perioral, periocular, and perianal areas) may be spared. The present case is unusual because it involved the lower limb and totally spared the upper limb and the face, with a T3 sensory level at the shoulder.

Other presentations of sensory loss in patients with a parietal lesion include pseudothalamic syndrome resulting from a superficial lesion in the territory of the anterior parietal artery [4, 5] and pseudoradicular sensory impairment [2, 6, 7], as well as other patterns of sensory defect (e.g., confined to the face, circumoral, or on the radial side of the hand) [2].

In the patient reported here the sensory loss involved all modalities. This is unusual (although already described [8]) because in parietal lesions the sensory deficit usually predominates on proprioception with relative sparing of the epicritic sensory modalities [9]. It is interesting that 3 months after the hemorrhage occurred, vibration and postural sensation testing showed clear improvement but the response to touch and pain remained impaired, with a level at the nipple. The lesion in this patient was located in the upper part of the postcentral gyrus, well in accordance with the somatotopic organization of the cortex [10, 11].

Visual field defect and neuropsychological dysfunction are sometimes present in parietal strokes, and their absence may also suggest a spinal cord lesion [12].

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Partial Resolution of Calcifications in Cerebral Infarcts in a Patient with Lupus

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ABSTRACT

A patient with lupus had calcified cerebral infarcts, with partial resolution of the calcification over time. Such reductions in the number and size of calcifications may contribute to an underestimation of the frequency of calcium deposition in cerebral infarcts.

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Calcification of cerebral infarcts has been reported, but has been considered rare [1]. This reports calcification within cerebral infarcts in a patient with lupus, which partially resolved 9 months later.

Case Report

The patient is a 43-year-old woman with a 17-year history of systemic lupus erythematosus (SLE), manifested by a positive antinuclear antibody (ANA) titer (1:640), a positive anti-DNA titer, renal involvement (by biopsy), arthritis, and rash. In October 1990 she noted swift onset of memory problems, not further described. On December 22, 1990, she and her husband noted sudden onset of her dragging the right leg and calling the children by incorrect names; however, she did not see a physician. At least one subsequent episode, with the sudden onset of slurred speech, occurred. She presented on January 21, 1991, with a moderate spastic right hemiparesis, motor perseveration, misnaming of simple objects, and mild slurring of speech. She had a history of one spontaneous abortion in the second trimester (three normal pregnancies), mitral valve prolapse, nonspecific ST-T changes on electrocardiography, and mild hypercholesterolemia (250 mg/dl). She was taking 20 mg of prednisone daily.

A brain computed tomography (CT) scan on January 29, 1991, was remarkable for an extensive area of low density, with calcifications in the periventricular white matter of the left hemisphere (Figs A, B). The differential diagnosis included opportunistic infection, neoplasm, or calcified cerebral infarct. A chest radiograph appeared normal. There was no systemic evidence of opportunistic infection. Cerebral angiography revealed no tumor blush and a large ulcerated plaque at the origin of the right internal carotid artery with a 75% stenosis and mild atherosclerotic disease at the origin of the left internal carotid artery. Magnetic resonance imaging (MRI) revealed periventricular leukomalacia of the left frontal lobe, consistent with a cerebral infarct. The electroencephalogram was normal.

The patient gradually improved. Repeat MRI on May 20, 1991, revealed left periventricular encephalomalacia, now with a dilated left lateral ventricle. Repeat CT on October 31, 1991, demonstrated a marked reduction in the size and number of the calcifications and enlargement of the left lateral ventricle (Figs C, D). Neurological examination on November 11, 1991, revealed a very mild spastic right hemiparesis with subtle errors in speech, mainly with hesitancy on naming tasks.

Discussion

Strokes are a well-known complication of SLE [2]. The presence of calcifications in the cerebral infarcts in this patient led to an expanded differential diagnosis, including opportunistic infections and a calcified neoplasm. The clinical course and the sequential radiographic studies support a diagnosis of cerebral infarcts. Idiopathic basal ganglia calcifications have been reported in 2 patients with SLE [3, 4] in whom clinical correlations included chorea in one and seizures in the other.

The temporal profile of calcifications within cerebral infarcts is unknown. In one patient calcification of a cerebral infarct was detected 18 days following a stroke [1], with the hyperintensity on CT initially interpreted as hemorrhagic transformation. On autopsy 4 days later (22 days following the stroke) diffuse calcium deposition was present within a nonhemorrhagic infarct. Calcifications have been seen within embolic cerebral infarcts in rats [5], with the earliest calcification found 15 days following infarction. Retraction of cerebral infarcts has also been noted in the rat [5], resulting in ventricular dilatation similar to that seen in this patient. Although calcifications have been seen years following cerebral infarcts [6], the sequential CT scans in this patient suggest that the number and size of the calcifications may decrease with time. Perhaps the calcium phosphate salts can dissolve or be partially removed by macrophages.

The frequency of calcifications of cerebral infarcts is unknown. Increased uptake of technetium 99m, which occurs in areas of calcium accumulation [7], has been seen commonly in cerebral infarcts

