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CASE REPORT

Absence of neurobehavioral disturbance in a focal lesion of the left paracentral lobule

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The case of a right-handed woman with an infarcation confined to the left paracentral lobule and sparing the supplementary motor area (SMA) is reported. She presented with a right leg monoplegia and displayed no mutism. The absence of any associated neurobehavioral disturbances (mutism, forced grasping, reduced spontaneous arm activity or aphasia raises the possibility that the left SMA has discrete neurobehavioral functions.

Keywords: Medial frontal lobe – Precentral gyrus – Supplementary motor area – Transcortical motor aphasia

INTRODUCTION

Various kinds of neurobehavioral disturbances associated with left medial frontal lesions involving the supplementary motor area (SMA) have been reported. Aphasia due to damage of the left medial frontal lobe is characterized by an initial period of mutism followed by a stage of decreased verbal output and spontaneous initiation with normal articulation (Stuss and Benson, 1986). Forced grasping, compulsive manipulation of tools and decreased spontaneous limb movements have also been described (Wise, 1984; Feinberg *et al.*, 1992). However, most reported patients with a combination of these disturbances in association with left medial frontal infarctions (Rubens, 1975; Masdeu *et al.*, 1978; Racy *et al.*, 1979; Alexander and Schmitt, 1980; Goldberg *et al.*, 1981; Mori and Yamadori, 1982; Uchiyama *et al.*, 1983; Notoya *et al.*, 1985; Motomura *et al.*, 1988; Takahashi *et al.*, 1991; Maeda *et al.*, 1991; Della Sala *et al.*, 1991) have an additional contralateral leg monoparesis, indicating involvement of the medial precentral gyrus.

The left precentral gyrus has recently been considered to have additional neurobehavioral functions. Some authors (Mori *et al.*, 1989; Alexander *et al.*, 1990) attributed the articulatory disturbance of Broca's aphasia to a lesion in the lower part of the dominant precentral gyrus. Rapcsak *et al.* (1988) reported a lexical agraphia associated with an infarction confined to the middle one third of the left precentral gyrus. These lesions are immediately posterior to the classical language area, that is, Broca's area or the writing center of Exner (the foot of the inferior or middle frontal gyrus, respectively).

We now describe a patient with a focal infarction in the medial part of the left precentral gyrus, which is adjacent to the SMA, to evaluate its possible neurobehavioral functions.

CASE REPORT

A 73-year-old right-handed woman with a history of hypertension and diabetes mellitus suddenly developed a gait disturbance. On examination, 24 h later, she was alert and orientated with no emotional changes. There was a flaccid monoplegia and sensory disturbance of the right leg. Spontaneous movements of the arms were normal and grasp reflex or instinctive grasp reaction absent. No alien hand syndrome or compulsive manipulation of tools was observed. Her speech was fluent and productive with no hesitation, effortfulness, dysprosodia, articulatory disturbances or paraphasia. She could name 10 small man-made objects and 10 body parts in a prompt manner. She performed category word finding tasks satisfactorily (animal, 11/min; fish, 11/min and when given the initial letter, 8 and 9/min respectively on 2 trials). There was no abnormality in repetition, pointing or syntax comprehension. She could read sentences with no difficulty, and her writing was normal. Ideomotor apraxia was not observed. Neuroimaging showed an infarction confined to the left paracentral lobule (Fig. 1), which spared the SMA and the frontoparietal convexity.

By the 4th week in hospital, her weakness had improved. She scored 99 on the verbal IQ of the Wechsler Adult Intelligence Scale (WAIS). Assessment with the Standard Language Test of Aphasia (SLTA) (Takeda,

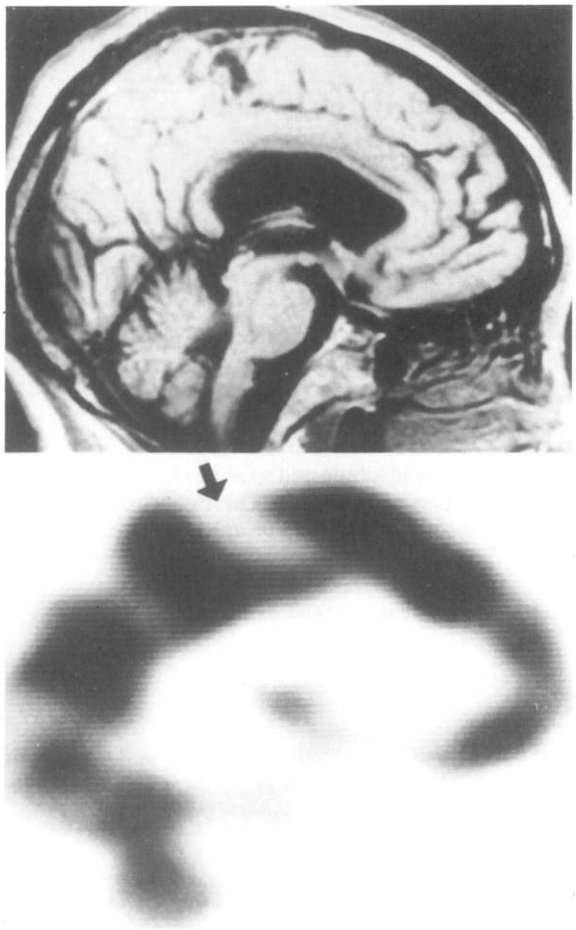

FIG. 1. A focal infarction in the left paracentral lobule can be seen on a magnetic resonance T1 weighted image (top). A sagittal image of single photon emission CT (bottom) revealed a corresponding reduced uptake of the tracer (arrow).

1977), which consists of standardized subtests of speech, comprehension, reading, writing and calculation for Japanese, revealed no signs of language disturbances. Repeated neuropsychological assessments detected no callosal disconnection syndrome.

COMMENT

The absence of aphasia and other neurobehavioral disturbances in a focal infarction of the left paracentral lobule is unusual. Several case reports in the literature, however, are consistent with our observation. Wilson (1923) reported a patient who at autopsy was found to have a left medial paracentral infarction, and whose speech "presented no abnormality". A recent series of anterior cerebral artery territory infarctions (Bogousslavsky and Regli, 1990) included a patient with a distal left ACA infarct who revealed no neuropsychological disturbances. The only neurological abnormality was a left monoplegia with sensory disturbance.

The medial and lateral parts of the precentral gyrus have discrete neuroanatomical relationships (see Wise, 1984 for review). The anterior portion of the lateral precentral gyrus belongs to the premotor cortex (Brodmann's area 6). On the other hand, the medial precentral gyrus consists of primary motor cortex (Brodmann's area 4). This may be the reason why only lateral lesions of the left precentral gyrus manifest neurobehavioral disturbances.

Language disturbances without weakness of the limbs in left medial frontal infarction suggest an intact precentral gyrus. Case 7 of Freedman *et al.* (1984) had transcortical motor aphasia (TCMA), and patient 14 of Bogousslavsky and Regli (1990) had initial mutism followed by TCMA associated with perseveration and impaired conflictual tasks. Computed axial tomography of both patients indicated lesions involving the left SMA. These findings support a double dissociation, that is, the lesion confined to the left SMA reveals a characteristic language disturbance without motor weakness, and a focal lesion of the left medial precentral gyrus shows right lower monoplegia without aphasia. Further clinico-pathological studies are needed, however, to confirm this putative neurobehavioral function of the left SMA.

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