Sensory Stroke Caused by a Small Cortical Infarct in the Middle Cerebral Artery Territory

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SUMMARY A patient with the sudden onset of a pure sensory deficit involving cortical modalities in his left hand and foot is described. CT scan suggested the lesion responsible for the deficit was a small cortical infarct in the middle cerebral artery territory, involving the middle part of the post-central gyrus and interrupting thalamo-cortical pathways in the corona radiata.

IN 1965, Fisher1 coined the term “pure sensory stroke” (PSS) for the sudden onset of a unilateral isolated sensory deficit involving the face, arm and leg. He attributed this syndrome to a lacunar infarct in the ventroposterior nucleus (VPN) of the thalamus. More recently, examples of this clinical syndrome caused by thalamic hemorrhage and by infarct or hemorrhage in the corona radiata have been documented.2-3 Pure sensory transient ischemic attacks associated with occlusive vascular disease of the internal carotid, middle cerebral or posterior cerebral arteries are well known,4 but a PSS caused by a cortical infarct has not yet been reported. We have had the opportunity to observe a PSS involving the left hand and foot, which proved on CT scan to be caused by a small cortical infarct in the territory of the right middle cerebral artery.

Report of a Case

A 50-year-old, white, right-handed man, with no history of systemic hypertension, had undergone a bilateral sympathectomy for peripheral occlusive disease two years before admission to the Hôpital de la Salpêtrière. Two days prior to admission (PTA) he suddenly developed numbness in his left thumb, index and middle fingers: they felt as if they had “gone to sleep” or were frozen. He could move his fingers normally but dropped small objects held in his left hand. One day PTA, on awakening, he experienced numbness in the distal part of his left foot and clumsiness while putting his slipper on his foot. On the day of admission, examination revealed that his muscular strength was good and deep tendon reflexes were normal and equal; plantar responses were flexor. Pinprick, light touch, heat and cold, vibration were appreciated perfectly. However, fine movements of the left fingers and toes were inaccurately perceived, and stereognosis, graphesthesia and two-point discrimination were severely impaired. Visual fields were full to confrontation. Copying and drawing were normal. His blood pressure was 150/100 mm Hg, equal in both arms. The right tibial pulse was not palpable. No neck bruit was heard. Clinical examination of the heart and ECG showed no abnormalities. These data were consistent with a diagnosis of PSS and, because the patient had no history of hypertensive disease, treatment with calcium heparin (5000 U I x 3/day) was initiated without delay. CT scan (fig. 1 A) performed on the 17th day after the clinical onset. The latencies of somatosensory evoked potentials (SSEP) produced by stimulation of the median and tibial nerves, were performed 30 and 56 days after the clinical onset. The latencies of the thalamocortical components: N 19 P 22 (recorded from a scalp electrode over the parietal sensory cortex were normal.
F. Gurel

CT scans performed after contrast injection 17 days (A) and 2 months (B) after the clinical onset

Figure 1

Discussion

In a patient with PSS, a recent cortico-subcortical infarct in the territory of the middle cerebral artery was demonstrated by CT scan. The infarct was presumably caused by embolization from an atheromatous and stenotic internal carotid artery. The CT scan abnormalities were consistent with an infarct of the same date as the clinical symptoms. A simultaneous thalamic infarct cannot be totally ruled out by the normal appearance of the thalamic region on CT scan, nor by the normal latencies of the short-latency somatosensory EPS. This hypothesis, however, is very unlikely.

The sensory deficit in the hand was of a cortical type, i.e.: distal with a "pseudoradicular" distribution and characterized by exclusive loss of joint position sense, stereognosis, graphesthesia and two-points discrimination. The CT scan data are consistent with an infarct involving the middle part of the post-central gyrus which, according to Penfield, is the site of sensory projections from the opposite hand. This area is vascularized by the anterior parietal artery, a branch either of the central artery (as cerebral angiography showed in the case reported here), or of the trunk of the middle cerebral artery, or rarely of the posterior parietal artery. The superior and inferior parts of the post central gyrus have a different blood supply. Accordingly, the limited sensory deficit in the hand can be explained by embolization to the anterior parietal artery.

The involvement of the foot is more difficult to explain. The most plausible explanation would be that the same cortical infarct also interrupted the corresponding sensory pathways between the thalamus and paracentral lobule in the corona radiata. These fibers appear to be supplied by cortical perforating arteries.

This hypothesis is congruent with isolated reports that an infarct in the anterior parietal artery territory was found responsible for contralateral hemianesthesia. Somatotopic identification of thalamocortical pathways has remained obscure but the data presented here indicates that the sensory fibers corresponding to the distal part of the foot might be located more laterally than the fibers corresponding to the rest of the leg. Lastly, the normal latencies and decreased amplitude of the thalamocortical (N19 — P22) EPS components are consistent with a pure cortico-subcortical lesion.

In a recent survey of PSS, Fisher reaffirmed his earlier belief in the predominance of thalamic lesions when there is a contralateral sensory deficit, involving the face, arm and leg. He also stated that, when only one or two parts of the side are involved, the localization of the lesion might be questionable. However,
Figure 2. Short-latency somatosensory evoked potentials performed 30 days after the clinical onset. Upper: stimulation of the right median nerve; amplitude of the N19-P22 components = 4.40 V. Lower: stimulation of the left median nerve; amplitude of the N19-P22 components = 1.25 V. Quite similar results were observed on the 56th day.

Discrete infarcts of the VPN of the thalamus have been documented with sensory deficits individually involving the face and hand; the mastoid region, arm and leg; the face; the arm; the leg; the oral cavity; the peribuccal area and forearm; and the peribuccal area and radial edge of forearm. On the other hand, sensory deficits involving face, arm and leg ("pseudo-thalamic") have been described in partial middle cerebral artery territory infarcts. It is, therefore, not possible to locate the lesion from the physical findings alone.

Fisher believed that the lesion responsible for PSS was a lacunar infarct, usually due to lipohyalinotic occlusion related directly to hypertensive disease. However, in one pathological case, the lesion consisted of a small hemorrhage found in the posterior limbs of the internal capsule. Of three other cases of PSS, one was related by CT scan to a thalamic hemorrhage; a second, to a lacunar infarct located in the thalamocortical projection area; and a third to a small thalamic infarct associated with occlusive disease of the posterior cerebral artery.

In the case here, PSS was due to a small infarct in the cortical territory of the middle cerebral artery caused, presumably, by embolization from an atheromatous stenosis of the internal carotid artery. Therefore it seems that PSS might not be a specific manifestation of a lacunar lesion in the thalamus. In this way it is comparable to the syndrome of pure motor hemiplegia which cannot be reported as a manifestation only of capular lesions infarction. Assessment of patient, with the syndrome of PSS should be the same as for any other minor stroke, and should include CT scan, doppler, ECG and ancillary investigations to verify the topographical and etiological diagnosis.

References
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C Derouesné, J L Mas, F Bolgert and P Castaigne

doi: 10.1161/01.STR.15.4.660

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231  
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:  
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