

Short report

Pure dysarthria due to anterior internal capsule and/or corona radiata infarction: a report of five cases

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SUMMARY Five cases with a sudden onset of dysarthria in the setting of hypertension are presented. No case had limb weakness or other neurological deficits. Computed tomographic scan demonstrated a small low density lesion in the anterior part of the internal capsule or the adjacent corona radiata. All cases showed a good recovery from dysarthria within two to four weeks.

Since computed tomographic scan (CT scan) was introduced, small infarcts have attracted the interest of many neurologists, for the various symptoms produced. In a recent review, Fisher¹ summarised the lacunar syndromes; however, the site of the lesion responsible for the pure dysarthria syndrome, one of the lacunar syndromes he originally described, remains uncertain. In this report, we present five cases with pure dysarthria, in which a small low density lesion was demonstrated by CT scan. In all instances, the lesion was in the anterior part of the internal capsule or the adjacent corona radiata.

Case reports

Case 1

A right-handed 52 year old man with untreated hypertension for one year, presented on 1 May 1984, because he had difficulty in speaking since 29 April. On examination his blood pressure was 170/110 mm Hg; he had impaired articulation with slurred speech with nasal features, but no dysphagia. Weakness of the tongue was not detected. Soft palate movements were well preserved. No facial palsy. There was neither weakness nor ataxia in his limb movements. Tendon reflexes were normal and symmetrical. Plantar responses were flexor; however, the left palmomental reflex was positive. CT scan performed on 7 May, 9 days after the onset, revealed a small low density area at the right genu of the

internal capsule (fig a). His symptom had completely resolved 2 weeks later.

Case 2

A right-handed 59 year old housewife suddenly could not speak fluently in late July, 1984. She had been suffering from bradycardia with atrial fibrillation and had been treated with an artificial pacemaker for 18 months. She had also received anti-hypertensive medication. She was referred to us on 17 August. Neurological examination revealed moderate dysarthria, which she believed was gradually improving. Other cranial nerves were normal. Power in the extremities, gait, coordination, deep reflexes and sensory functions were all normal. Her blood pressure was 150/100 mm Hg. CT scan showed a small low density area in the left internal capsule and the adjacent corona radiata (fig b). At the end of August, her speech almost recovered.

Case 3

A right-handed man aged 51 years could not speak clearly on 1 April 1984. He was known to have had hypertension for 10 years. When he presented on 3 April, he was only complaining of dysarthria. There was possible but not definite right facial palsy. Other cranial nerves were intact. There was no abnormal finding in the extremities and in the trunk. His hypertension was poorly controlled, blood pressure 220/120 mm Hg. A small ill-defined low density spot was revealed in the anterior part of the left corona radiata by CT scan performed on 4 April. His dysarthria had nearly disappeared two weeks later. Six months later, the low density spot was again found at the same place with a clearer outline than before (fig c).

Case 4

A right-handed man aged 53 years suddenly noted dysarthria on the morning of 5 September 1984. Neurological examination revealed no abnormality except for moderate dys-

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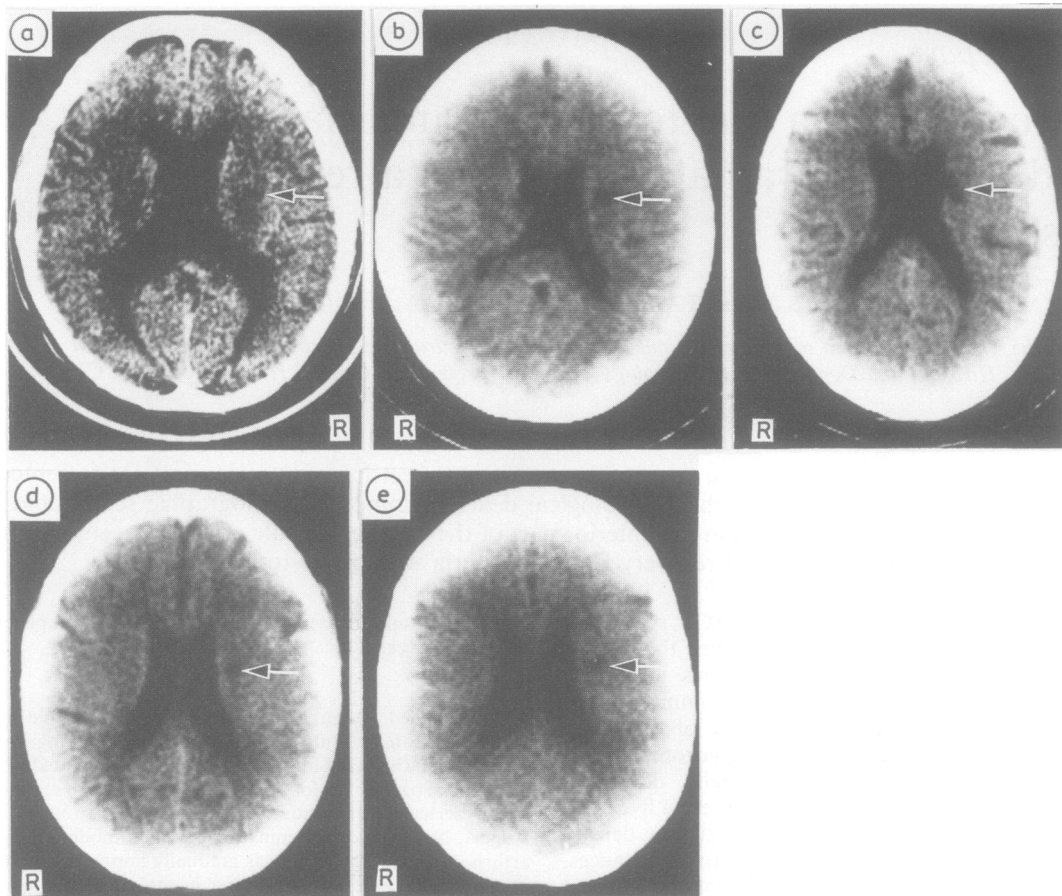


Fig 5 CT scans in case 1(a), 2(b), 3(c), 4(d) and 5(e). The arrows indicate small low density spots in the anterior part of the internal capsule or the adjacent corona radiata.

arthria. His blood pressure was 140/102 mm Hg. CT scan, carried out on 12 September showed a poorly distinguished low density spot in the anterior part of the left corona radiata, which was seen as a clear low density lesion eight months later (fig d). Normal articulation was restored within 2 weeks of the onset of dysarthria.

Case 5

A right-handed 56 year old housewife suddenly noted dysarthria in the morning of 11 November 1984. She had been treated for diabetes mellitus and hypertension since 1980. On examination, only dysarthria was noted in her cranial nerve region. She had no weakness in the extremities. Coordination was normal. Ankle jerks were absent. No sensory signs were found. Her blood pressure was 180/80 mm Hg. CT scan five days after the onset showed a small low density area in the anterior part of the internal capsule and the adjacent corona radiata of the left hemisphere. The lesion was seen as a well-defined low density spot at the same place 5 months later (fig e). Two weeks after the onset, her dysarthria had

considerably improved. Four weeks later her speech was normal.

Discussion

The common neurological deficit in all five patients was dysarthria of sudden onset, which showed very good recovery within 2 to 4 weeks. To our knowledge, the lesion responsible for pure dysarthria syndrome has not been identified. No pathological verification so far has been obtained. Fisher considered that the basis pontis was a likely site of the lesion, because neither CT nor angiography had demonstrated any abnormalities. However, in all our five cases, CT scan demonstrated a small low density lesion in the anterior part of the internal capsule or the adjacent corona radiata. This finding strongly suggests that there is a close relationship between the pure dysarthria syn-

drome and a lacune in the anterior part of the internal capsule or the adjacent corona radiata.

The anterior part of the corona radiata and the internal capsule mainly consists of frontopontine fibres and anterior thalamic radiations.² Hence damage to those fibres could play an important role for the development of pure dysarthria. Case 1, in which the lesion was located in the genu, more posteriorly than in the other cases, revealed positive palmomental reflex on the contralateral side of the lesion. The corticobulbar tract happened to be minimally involved in this particular case.

All the cases described here were right handed.

Four of the five had a lesion in the left side, the dominant hemisphere. This suggests that the lesion in the dominant hemisphere might be responsible for the development of pure dysarthria; however, case 1 had a lesion in the right non-dominant hemisphere. Hence, the relationship between the syndrome and the dominance of the hemisphere remains uncertain.

References

- 1 Fisher CM. Lacunar strokes and infarcts: a review. *Neurology* 1982;**32**:871-6.
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