

## Short report

# Hemiataxia and crural hemiparesis following capsular infarct

E DE RENZI,\* P NICHELLI,\* G CRISI†

*From Clinica Neurologica\* and Istituto di Radiologia† Università di Modena, Modena, Italy*

**SUMMARY** Two patients presenting with hemiataxia and hemiparesis on the same side are described. Both had CT scan evidence of an infarct located in the posterior limb of the contralateral internal capsule.

The association of cerebellar ataxia and pyramidal signs involving the limbs of the same side was first described by Fisher and Cole<sup>1</sup> in 1965, who tentatively located the lesion in the posterior limb of the internal capsule. Subsequently ataxic hemiparesis has been reported in six patients with lesion in the basis pontis contralateral to the involved limb,<sup>2-5</sup> in a patient with a ventrolateral midbrain lesion<sup>6</sup> and in three patients showing CT scan evidence of an infarct of the posterior limb of the internal capsule.<sup>7-9</sup> We report two more cases of ataxia and homolateral crural paresis. In both patients a lesion located in the posterior limb of the internal capsule was identified on the CT scan.

### Case 1

A 45-year-old man was admitted to the wards after the sudden onset of a right hemiparesis. On neurological examination he was alert and oriented. Speech and cranial nerves were normal. There was slight right-sided weakness of the peroneal muscles with minimal weakness of the extensor muscles of the right arm. Tendon reflexes on the right were slightly brisker than those on the left and the right plantar response was extensor. On standing with the feet together the patient fell to the right. There was a severe dysmetria on both finger-to-nose and heel-to-knee tests. Rapid alternating movements were slow and clumsy on the right side. Sensory functions and visual fields were normal. Weakness and ataxia increased in a step-wise fashion in the next two days. Weakness was greater in the proximal muscles of the upper limb and in the distal muscles of the lower limb. At the end of the fourth week of illness the right arm had completely recovered its strength,

but both upper and lower right extremities were still dysmetric and the right lower limb was still moderately weak particularly in the hamstrings and peroneal muscles. Four and a half months after the onset of disease, ataxia persisted with only minimal improvement, while weakness of the left hamstrings had almost completely recovered.

CT scan one day after admission revealed a small decreased density area in the left posterior limb of the internal capsule with possible involvement of the adjacent thalamus. One month after the onset of illness CT scan (Fig 1a) showed a clear-cut hypodensity area in the two middle quarters of the posterior limb of the left internal capsule. The lesion extended upward from a midthalamic to a high thalamic level, but spared the corona radiata and that most superior part of internal capsule which is immediately adjacent to corona radiata.

### Case 2

A 55-year-old man, with a long history of hypertension and diabetes, suddenly noticed dragging of his left leg and loss of motor power and tingling of his left arm. On admission the patient was alert and fully oriented. Visual fields were intact. There was moderate left central facial weakness and a gross left hemiparesis which in the upper limb, mainly affected the proximal muscles. Tendon reflexes were slightly brisker on the left side. The left plantar response was extensor. Light touch and joint position sense were slightly impaired on the left. At that time tests of coordination could not be carried out, because of the severe motor weakness. Computed tomography revealed a low density area occupying a great part of the posterior limb of the internal capsule with involvement of the most lateral part of the thalamus. The corona radiata also was involved (Fig 1b). The patient's paralysis began to improve after two weeks, and after a month the muscle power of arm and forearm flexors and extensors had recovered to the point of permitting finger-to-nose and finger-to-finger testing. The latter manoeuvre was carried out holding the examiner's thumb at the same horizontal level as the patient's hand, so that no movement of the shoulder muscles, which were still weak, was required. These tests dis-

Address for reprint requests: Dr Ennio De Renzi, Clinica Neurologica Via del Pozzo 71, 41100 Modena, Italy.

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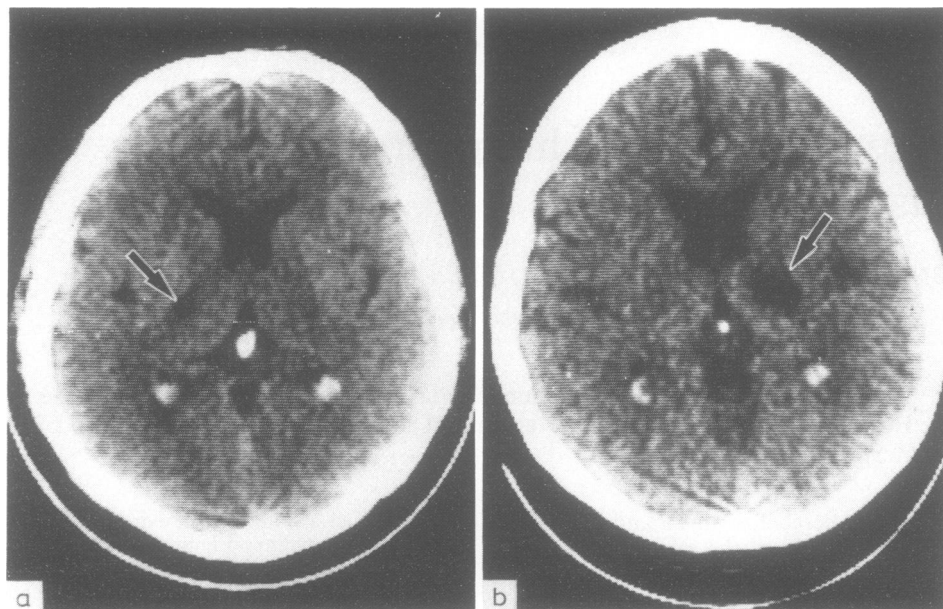


Fig 1 (a) Case 1: 2nd CT scan (one month after onset of disease) shows a lesion in the posterior limb of the left internal capsule, (b) Case 2. CT scan (8 days after onset of disease) shows a low density area in the posterior limb of the right internal capsule encroaching upon the thalamus.

closed a gross wavering ataxia, which did not change with the eyes closed and could not be explained by weakness. There was also exaggerated rebound on the same side and, when sitting on the bed with his feet dangling, the patient slowly toppled to the left. Heel-to-knee test could not be performed owing to the weakness of the left limb.

### Discussion

Both patients had a lesion in the superior portion of the internal capsule, which encroached upon the lateral thalamus, marginally in case 1, more extensively in case 2. The location of the infarcts corresponds to that shown by CT scan in analogous cases, of which three were reported in detail<sup>7-9</sup> and three were mentioned in a recent paper<sup>10</sup> devoted to lacunar infarcts. Also the finding that paresis was more marked on the lower limb appears to be typical of this kind of capsular infarcts.

As suggested by Fisher and Cole,<sup>1</sup> the mechanism whereby hemiataxia follows a capsular lesion is probably the interruption of the cortico-pontine-cerebellar connections. Their origin and course has recently been the subject of a number of studies. These have shown that neither Arnold's bundle, nor Türk's bundle, which are reported by many anatomical textbooks<sup>11-13</sup> to be the main constituents of these pathways, are implicated in the transmission of information from the cerebral cortex to the cerebel-

lum, since both tracts terminate above the pontine level.<sup>14-16</sup> By studying areas of degeneration in the pontine nuclei of monkeys which had undergone cortical excisions, Brodal<sup>17</sup> was able to demonstrate that cortico-pontine fibres originate predominantly from area 4, areas 3, 1 and 2, area 5 and parts of the visual cortex representing the peripheral visual field. The contribution from area 6 and 7 is less substantial, and only a minor contingent of fibres comes from the prefrontal and temporal cortex. Part of the cortico-pontine connections are collaterals of pyramidal fibres, while the majority have an independent origin in approximately the same areas giving rise to the cortico-spinal pathway. It is, therefore, suggested that they do not run separately, but adjacent to, or even intermingled with the pyramidal tract in the caudal sector of the posterior limb of the internal capsule, a hypothesis already advanced in the monkey.<sup>14</sup>

In addition to the involvement of cortico-pontine fibres, the capsular lesion may produce cerebellar symptoms by encroaching upon the ventro-lateral (VLo) nucleus of the thalamus or its efferents to the motor cortex, which run in the medial part of the posterior limb of internal capsule.<sup>18</sup> This nucleus receives fibres from the contralateral dentate nucleus and is connected with the motor cortex, thus entering as a component of the afferent limb of the cortico-cerebellar loop. Its damage has been

reported to produce cerebellar symptoms, mainly in the form of intention tremor,<sup>19</sup> although a more destructive injury of this nucleus and of the neighbouring ventralis anterior nucleus abolishes the tremor, as shown by stereotaxic surgery findings.<sup>20</sup>

It may prove to be extremely difficult to estimate the extent to which the cerebellar symptomatology consequent to capsular infarcts is contingent upon damage of cortico-pontine or thalamo-cortical connections. At any rate its occurrence is likely to be a common event, which is usually obscured by the concomitant hemiparesis and can only be detected when the latter abates. Even so, there is the risk that hemiataxia is overlooked by the examiner, who may feel hesitant to diagnose it in the presence of motor weakness. The CT scan detection of a posterior capsular infarct should alert neurologists to carefully test cerebellar functions.

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