The presence of downgaze paresis is required for the diagnosis, but is not necessarily present in the early stages of the disease, so that at this stage other neurological findings such as hyperreflexia, extensor plantar responses and a more rapid clinical course may make one suspect PSP rather than idiopathic PD. The limitation of upgaze was presumed to be due to bilateral involvement of the fronto-mesencephalic pathways for voluntary upgaze. This case showed evolution from a clinical picture compatible with PD through to one suggesting possible PSP, but with a suspiciously rapid clinical course. The MRI scan demonstrated a multifocal astrocytoma that masqueraded as PSP.

Contralateral selective saccadic palsy after a small haematoma in the corona radiata adjacent to the genu of the internal capsule

Contralateral saccadic palsy with ipsilateral conjugate deviation of the eyes is usually attributed to lesions involving the frontal eye field (FEF) or connections with that area coursing through the internal capsule. The lesions are usually so large that they cause obtundation and contralateral hemiparesis. Pathological confirmation of the lesions confined to the FEF or its connections has not been reported. We report a case of contralateral selective saccadic palsy with neither cluing of consciousness nor limb weakness after a very small haematoma in the corona radiata adjacent to the genu of the internal capsule.

A 50 year old woman developed acute dysarthria whilst in hospital for a broken leg. After defaecation, she had developed speech disturbance, immediately followed by right facial drooping. Blood pressure was 160/100 mm Hg and pulse 90 minutes. On neurological examination, she was alert and cooperative. Her eyes deviated upwards and to the left but she was able to follow slowly moving targets in either horizontal direction. Optokinetic testing with targets moving to the patient’s right evoked a normal nystagmus response but the eyes deviated tonically into an eccentric leftward position with the target moving to the left. She had minimal right lower facial weakness and mild parietal dysarthria. She complained of difficulty in swallowing liquids, and palatal and pharyngeal weakness was present on the right. There was no deviation of the tongue on protrusion. Facial and buccal sensation were normal. Motor, sensory and cerebellar functions were normal in the limbs except for the following signs of subtle corticospinal damage: when the fingers were stretched out voluntarily, abduction of the fifth finger (a digitii quinti sign of Alter*) and adduction and flexion of the first metacarpal (a hollow hand sign of Garcin*) were noted on the right side. Muscle strength of the legs was probably normal. Tendon reflexes were normal with flexor plantar responses.

A CT scan revealed a round hyperdensity, of approximately 7 mm diameter, located in the left corona radiata adjacent to the genu of the internal capsule, suggesting a small haematoma (fig). Her oculomotor disorder disappeared within two weeks. Two months after the stroke, neurological examination was normal.

Our patient presented contralateral selective saccadic palsy and contralateral supranuclear facio-palato-pharyngeal paresis, but no weakness of the tongue and limbs. According to recent anatomical studies in monkeys, the major pathway from the frontal eye field descends in or slightly anterior to the genu of the internal capsule near the caudate head. The very restricted lesion of our patient suggests that the descending pathway from the FEF in humans may pass through the genu of the internal capsule in parallel with the corticobulbar tract.