

Hemiplegia in posterior cerebral artery strokes

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Hemiplegia is an unusual presenting feature of posterior circulation strokes. We report five cases who presented with hemiplegia and in whom CT scans revealed evidence of infarcts in posterior cerebral artery territory.

Key words: stroke; hemiplegia; posterior cerebral artery

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Occlusion of the posterior cerebral artery (PCA) has been known to produce a wide variety of manifestations but hemiplegia has not been observed to be prominent (1). It has been postulated that the involvement of pyramidal tracts leading to hemiplegia can occur at the level of internal capsule, corona radiata or cerebral peduncle. We report five such cases who presented with hemiparesis and PCA infarcts on CT at our centre over a five year period.

Case histories

Patient 1 was a 62-year-old man who developed right-sided paresthesia which was followed by, a few minutes later, significant right-sided hemiparesis. Sensorium, speech, and cranial nerves were unaltered and both ocular fundi were normal. On examination he was able to lift the right upper and lower limbs against gravity and felt touch and pin prick less on the right side. Over the next week hemiparesis progressed mildly and he could barely hold the limbs against gravity. Since then the deficit remained static and at discharge two weeks later no change was noted.

Non-enhanced CT scan done at admission showed a discrete hypodense infarct in the posterior limb of the left internal capsule extending into the adjoining thalamus (Fig. 1).

Patient 2 was 22-year-old, right-handed man who presented with a two-day-old, isolated, significant left pure motor hemiparesis. He had no evident risk factor for stroke.

A non-enhanced CT study of the head on the second day showed an evolved infarct in the right cerebral peduncle and postero-medial thalamus (Fig. 2).

The deficit significantly improved and the patient was ambulant at discharge a week later. He was later lost to follow up.

Patient 3 was a 38-year-old, dextral man who presented with left-sided hemiparesis which evolved over 10 h. This was associated with mild right occipital headache and occasional vomiting. There was no alteration of sensorium, visual agnosia, impairment of memory or ataxia. Clinical examination revealed significant left hemiparesis and a left macular sparing homonymous hemianopia. No risk factors for cerebro-vascular diseases could be found.

Non-enhanced CT study done two days after the ictus showed a well-defined infarct involving the posterior limb of right internal capsule, hippocampus, adjacent temporal and occipital lobes. The adjoining thalamus was characteristically spared (Fig. 3).

The deficits were found to have remained static on clinical review two months later.

Patient 4 was a 28-year-old man who was admitted with status epilepticus while under follow-up for recurrent seizures for the past two years. The seizures were never associated with an aura, prodrome or precipitating factors and were apparently generalised in nature from onset. On few occasions weakness of the left upper limb was noted on recovery from the seizure. This would resolve spontaneously

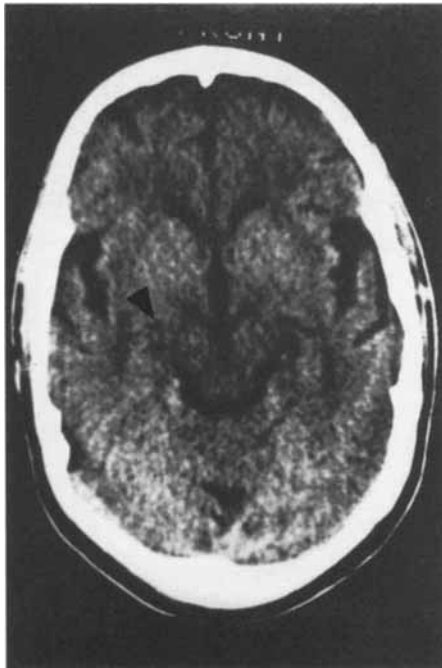


Fig. 1. Discrete infarct in the posterior limb of the left internal capsule extending into the adjoining thalamus.

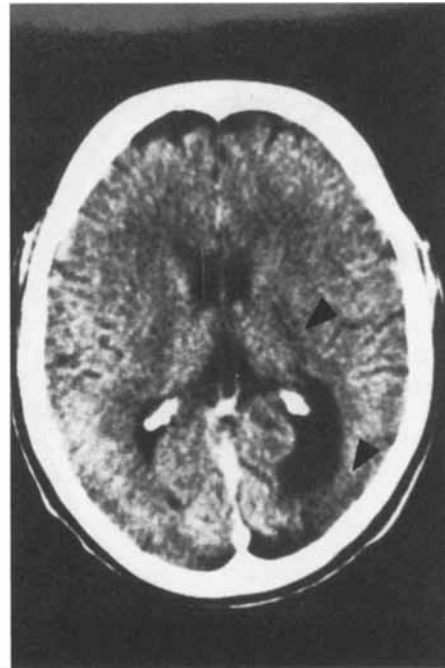


Fig. 3. Infarct of the posterior limb of the right internal capsule, hippocampus, adjacent temporal and occipital lobes. The adjacent thalamus is characteristically spared.

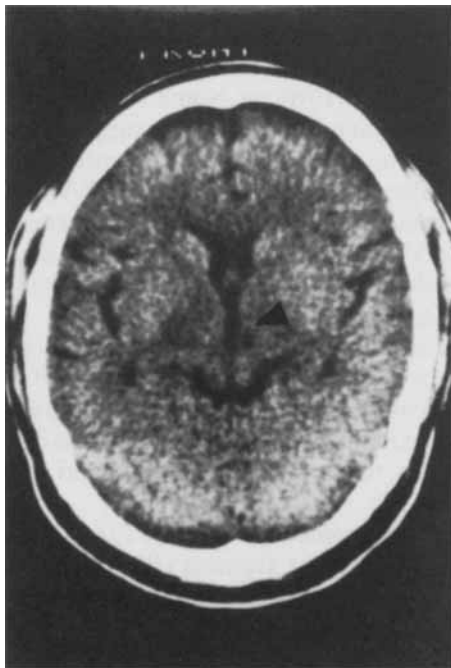


Fig. 2. Evolved infarct in the right cerebral peduncle and postero-medial thalamus.

over the next three to four days. On examination he had left hemiparesis with left homonymous hemianopia. He had no neurocutaneous markers or subcutaneous nodules and both ocular fundi were normal. X-ray chest revealed evidence of healed pulmonary tuberculosis.

Enhanced CT head study at admission revealed an old infarct in the posterior parietal and occipital lobes associated with a hypodensity in the genu of the right internal capsule, extending into the adjacent retrolentiform region and the geniculate body. The occipital horn and the trigone were also dilated (Fig. 4). He was treated with parenteral diphenylhydantoin initially which was reduced to a maintenance dose at discharge. He had no seizure during the hospital stay and at discharge his neurological status remained unchanged. He was subsequently lost to follow up.

Patient 5 was a 62-year-old man who presented with sudden, painless right-sided field defect and weakness of the right half of the body. On examination he had a macular sparing homonymous hemianopia and significant right hemiplegia. No parietal and frontal lobe signs were evident on examination.

CT scan done two weeks after the ictus revealed a well-defined hypodensity affecting the medial and inferior part of the left temporal lobe and extending into the left occipital lobe (Fig. 5). During hospital stay deficit remained static. At review one month later no change was noted.

Discussion

Hemiparesis in posterior cerebral infarcts has been focused upon previously and various sites of involvement of the pyramidal tracts implicated. Johansen (2) first suggested involvement of the motor fibres in

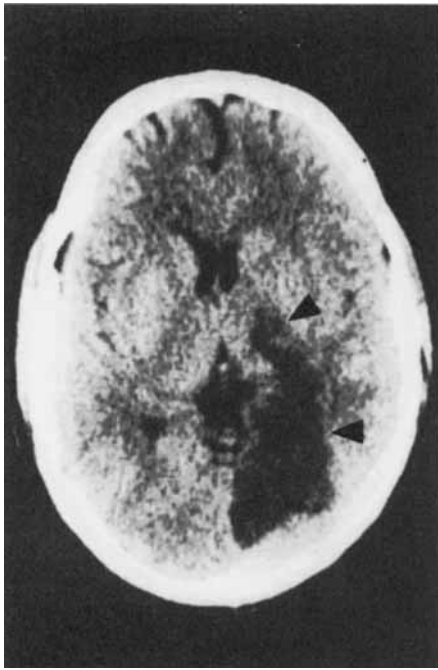


Fig. 4. Enhanced CT scans shows old infarct in the posterior parietal and occipital lobes associated with hypodensity in the genu of the right internal capsule.



Fig. 5. Infarct in the medial and inferior part of the left temporal lobe extending into the left occipital lobe.

the corona radiata or internal capsule in a CT-study. Evidence of midbrain infarct was provided by Caplan in an autopsy study (3). Homel et al. showed mid brain involvement on MRI and CT as did Tomlison et al. on radionuclear scanning (4). The commonest site of involvement is possibly the regions supplied by the perforators of the posterior cerebral artery.

The five cases we present here includes three young patients with no evident risk factors for cerebrovascular disease. One of the two patients over fifty years was a known hypertensive (Case 5). Only one of the five patients had pure motor hemiplegia. In one of the cases (Case 2) the initial impression was a that of a middle cerebral artery stroke. None of the patients had any other associated infarcts.

Attempting to correlate the neurological deficits with the infarcts on CT we have categorized patients into two broad groups. The first group (Patients 1, 2) consisted of patients who had hemiplegia without field defects whereas those in the second group had hemiplegia with field defects. Before attempting to correlate the symptomatology with the CT features the distribution of the basal perforating arteries should be reviewed. Foix & Hillemand (5) described the paramedian and circumferential arteries originating from the rostral basilar artery supplying the anterior and anterolateral midbrain upto the substantia nigra. The thalamotuberal artery (TTA) which phylogenetically arises from the posterior communicating artery can be considered to arise from the

most proximal segment of the posterior cerebral artery. The distribution of the thalamotuberal artery to the anterior pole of the thalamus and hypothalamus is well defined. However, some thalamotuberal artery radicles have been shown to supply the anterior segment of the posterior limb of the internal capsule.

The thalamoperforating arteries (TPA) arise from the peduncular segment (P1) of the posterior cerebral artery (PCA) and supply a wide area which includes the anterior part of the posterior thalamus, hypothalamus, subthalamus, substantia nigra, red nucleus, oculomotor nucleus and nerve. The short circumflex arteries also arise from the P1 segment and run a short course around the midbrain before terminating in branches which supply the corticospinal tract in the cerebral peduncle and substantia nigra.

The thalamogeniculate arteries (TGA) have their origin in the ambient segment (P2) of the PCA and enter the brain between the medial and lateral geniculate bodies and then supply the thalamus.

The posterior choroidal arteries have their origin from the P2 segment of the posterior cerebral artery. The medial posterior choroidal artery is thought to supply the postero medial and dorso-medial portions of the thalamus. The lateral posterior choroidal artery is distributed to the pulvinar, dorsolateral thalamus, lateral geniculate body, crus, commissural body and part of the anterior column of the fornix.

The two groups of symptom complex may be explained by occlusions at various discrete segments of the PCAs. In the first group of patients with isolated

Table 1.

| Sl. no. | CT lesion | Neurological deficit | Site of occlusion |
|---------|--|--|---------------------------------|
| 1. | Int. capsule and lateral | Hemiplegia and hemianaesthesia | TTA (P Comm) & TGA (P1 segment) |
| 2. | Cerebral peduncle & post-med. thalamus | Pure motor hemiplegia No hemianesthesia | TTA (P Comm) & TGA (P1 segment) |
| 3. | Int. capsule Hippocampus Temporal & occipital lobes | Hemiplegia & hemianopia | P2 and P3 segments |
| 4. | Int. capsule lat. geniculate body, Retrolentiform region | Hemiparesis & hemianopia | P2 and P3 segments |
| 5. | Inferior and medial temporal lobe | Hemiparesis & hemianopia | P2 and P3 segments |

hemiparesis the block involves the P1 segment which can explain the discrete infarct in the posterior limb of the internal capsule. If the P2 segment is also included in the lesion the adjacent hippocampus and trigone are also involved. This is seen in Patient 3. The involvement of the P2 segment along with the quadrigeminal segment of the PCA (P3 segment) would lead to infarction of the occipital cortex explaining the occurrence of field defects with hemiparesis (Table 1).

The pure motor symptoms can be explained by either involvement of the perforators from the P1 or P2 segment of the posterior cerebral artery. Presence of sensory symptoms would implicate the P1 segment whereas involvement of the third nerve or substantia nigra would suggest involvement of the P2 segment. The visual cortex would be affected when the P3 segment is also involved.

A reliable diagnosis of hemiparesis due to PCA infarcts is possible by detailed clinical examination and current generation CT scanners though acute infarctions limited to the crura may be missed occasionally due to limitations of computerized tomograms.

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