Traumatic basal ganglia haemorrhage with slight clinical signs and complete recovery

A traumatic basal ganglia haemorrhage is a rare but serious complication of head injury. Recognition of its prevalence and clinical features has been made possible by the advent of CT. We describe a patient with a large traumatic ganglia haemorrhage with slight neurological signs and complete recovery.

A 15 year old right handed young woman sustained left frontotemporal injury in a motorcycle accident. Witnesses reported a short loss of consciousness (lasting a few seconds) accompanied by a sudden and brief extensor “stiffening” of all limbs and followed by a short confusional state (a few minutes). On admission to the emergency department an hour later she was awake and fully orientated and reported retrograde amnesia of a few minutes duration. General physical and neurological examinations were normal, as were X ray pictures of skull, chest, and cervical spine, routine laboratory investigations and EKG. The next day she was still alert and cooperative, but complained of diffuse, moderate to severe, band-like headache. She had a very slight weakness of her left lower facial muscles. Her EEG showed a drowsy pattern (flattening with 4-7 Hz low voltage waves, with inverted arousal reaction) without clear cut abnormalities. Two days later a repeat EEG showed right temporo-frontal 1-3 Hz high voltage waves, spreading mainly to the ipsilateral hemisphere. A brain CT scan showed a medium sized haemorrhage surrounded by a slight oedema in the anterior half of the right lentiform nucleus, with a slight compression of the frontal horn of the lateral ventricle and displacement of the anterior limb and genu of the internal capsule and the head of caudate nucleus (figure). Over the following days the facial weakness disappeared completely. A repeat CT ten days later showed a reabsorption of the haemorrhage. The EEG had reverted to normal. A right carotid angiogram did not show a vascular lesion.

Traumatic basal ganglia haematomas (3%) complication of severe closed head injury, occurring mainly in the young, but the proposed underlying mechanism is shearing of an anterior choroidal or lentilostratate artery due to violent acceleration-deceleration brought about by a high velocity injury. In almost every case the haemorrhage is accompanied by the usual pathological features of severe head injury—for example, diffuse axonal injury, multiple contusions, and epidural or subdural haematomas. In one large series patients with a traumatic basal ganglia haematomata had a poor prognosis but cases with a favourable outcome have been reported.

Basal ganglia vascular lesions that do not involve the internal capsule may be asymptomatic, and subcortical vascular lesions of the dominant hemisphere may bring about only aphasic disturbances or even be clinically silent. Small basal ganglia haemorrhages in the non-dominant hemisphere may not be associated with the typical cognitive and behavioural syndromes (left neglect, visuospatial impairment etc.). The interest of the present case lies in its favourable outcome. Although an early CT examination was not performed, we suggest that the early absence of neurological and EEG abnormalities reflected a slow development of the haematomata.

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6 Radin A. Asymptomatic intracerebral haemato-

Transient pure sensory strokes in patient with aneurysm of rostral basal artery

Pure sensory stroke (PSS) usually results from a lacunar infarct in the sensory nucleus of the thalamus; however, ischaemic and haemorrhagic lesions with various locations have also been reported. We studied a patient with PSS in whom an aneurysm of the rostral basal artery was disclosed by CT scan and MRA.

On the day of admission a 78 year old, right handed man suddenly developed three brief episodes of numbness and unpleasant dysesthesia on the right side of the body. He had no headache, stiff neck, dizziness, or visual symptoms. He was in good general health. We were able to induce his disorder. There was loss of temperature and pain sensation affecting the right side of the body including the face. Touch, vibration, position sensation, graphesthesia, and stereognosis were normal, and no other neurological or behavioural symptoms. A few minutes later the symptoms resolved spontaneously, and the neurological examination showed no objective sensory disturbances. Speech and language and other cranial nerves were normal.

General physical examination was unremarkable and laboratory studies showed normal results. Electroencephalogram, somatosensory, brainstem auditory, and visual evoked potentials were also normal. The CT scan showed a round area of contrast enhanced density in the region of the interpeduncular fossa, with the CT features of a rostral aneurysm (figure, top). MRA confirmed the presence of an aneurysm extending from the upper pons to the inferior aspect of the third ventricle without affecting the thalamus and compressing the left cerebral peduncle. A slightly stenotic (figure, middle). MRA disclosed hyperintense images within the aneurysm, suggesting a clot inside its lumen (figure, bottom). Both CT scan and MRA did not show any abnormality of a focal nature in the brainstem, internal capsule, basal ganglia, or cerebral hemispheres. A digital venous angiogram showed no stenosis or ulceration in the carotid or basilar arteries. Reassessment performed later showed a normal neurological examination, and the patient reported that no other similar disturbances had occurred.

The neurological disorder in this patient met the established criteria for transient ischaemic neurological deficit (TIA) as they resolved within a few minutes after onset. Both CT scan and MRA showed a saccular aneurysm of the rostral basilar six months any other pathological change elsewhere in the brain. Therefore the precise vascular territory affected cannot be identified, but on the basis of the aneurysm location either a vascular supply of the thalamus or of the upper midbrain explains the symptoms and signs presented. Asymptomatic aneurysms