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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.¹ Recognition of its prevalence and clinical features has been made possible by the advent of CT.^{2,3} We describe a patient with a large traumatic basal ganglia haematoma with slight neurological signs and complete recovery.

A 15 year old right handed young woman sustained a 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 retroanterograde 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 bilateral random 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

Figure CT scan showing right basal ganglia haemorrhage

carotid angiogram did not show a vascular lesion.

Traumatic basal ganglia haematoma is a rare (3%) complication of severe closed head injury, occurring mainly in the young,^{2,3} the proposed underlying mechanism is shearing of an anterior choroidal or lenticulostriate artery due to the violent acceleration-deceleration brought about by a high velocity injury.¹⁻³ 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.^{2,3} In one large series patients with a traumatic basal ganglia haematoma had a poor prognosis² but cases with a favourable outcome have been reported.³ Basal ganglia vascular lesions that do not involve the internal capsule may be asymptomatic,⁴ and subcortical vascular lesions of the dominant hemisphere may bring about only aphasic disturbances⁵ or even be clinically silent.⁶ Small basal ganglia haemorrhages in the non-dominant hemisphere may not be associated with the typical cognitive and behavioural syndrome (left neglect, visuospatial impairment etc).³ 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 haematoma.

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

Pure sensory stroke (PSS) usually results from a lacunar infarct in the sensory nucleus of the thalamus;¹ however, ischaemic and haemorrhagic lesions with various locations have also been reported.¹⁻³ We studied a patient with PSS in whom an aneurysm of the rostral basilar artery was disclosed by CT scan and MRI.

On the day of admission a 78 year old, right handed man suddenly developed three brief episodes of numbness and unpleasant dysaesthesia on the right side of the body. He had no headache, stiff neck, dizziness, or visual symptoms. He was in good health except for arterial hypertension, which was well controlled with medication. Neurological examination performed during one of the episodes showed that he was conscious, well oriented, and aware of his disorder. There was loss of temperature and pain sensation affecting the left side of the body including the face. Touch, vibration, position sensation, graphaesthesia, and stereognosis were normal, and no other neurological deficits were found. A few minutes later the symptoms resolved spontaneously, and the neurological examination showed no objective sensory disturbances. Speech and language and other neuropsychological functions 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 basilar aneurysm (figure, top). MRI 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, which appeared slightly atrophic (figure, middle). MRI disclosed hyperintense images within the aneurysm, suggesting a clot inside its lumen (figure, bottom). Both CT scan and MRI 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 six months later showed a normal neurological examination, and the patient reported that no other similar disturbances had occurred.

The neurological disorders in this patient meet the established criteria for transient ischaemic neurological deficit (TIA) as they resolved within a few minutes after onset. Both CT scan and MRI showed a saccular aneurysm of the rostral basilar artery without 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 the vascular supply of the thalamus or of the upper midbrain explains the symptoms and signs presented.¹⁻³ Asymptomatic aneurysms

Figure Top—contrast-enhanced CT scan showing round area of increased density in upper interpeduncular fossa. Middle—MRI (Sagittal, T2 weighted) showing aneurysm extending from upper pons to inferior aspect of third ventricle compressing left cerebral peduncle. Bottom—MRI (Axial, T2 weighted) shows hyperintense images within aneurysm suggesting clot inside its lumen.

have been found at necropsy or during neurological evaluation for other reasons. In our case the association of ischaemic attacks with an aneurysm in the appropriate vascular distribution without any evidence of other abnormalities of the CNS strongly suggests a causal relation rather than an incidental finding. Transient neurological disorders resembling TIAs or reversible ischaemic neurological deficits have been described in patients with intracranial unruptured aneurysms, but although an ischaemic process was suspected in all cases, the exact pathophysiological mechanisms of these disturbances remain obscure.⁴ Focal neurological events have been attributed to the compressive effects of large intracranial aneurysms. The abrupt onset of symptoms may occur because enlarging aneurysms acutely obstruct a penetrating branch of the basilar artery within the confines of the aneurysm. A mechanism of direct compression on the adjacent cerebral structures seems unlikely in our patient as he showed transient

sensory disturbances closely resembling an ischaemic process.

Intra-arterial embolism of an aneurysmal sac thrombosis is considered a possible cause of transient ischaemic deficit or completed stroke in patients with unruptured, intracranial aneurysms.^{4,5} In our case the demonstration by MRI of intraluminal thrombotic material inside the aneurysm gives support to the view that thrombi may have dislodged from the clot and embolised into the distal vessels.⁷ Alternatively the thrombosis process may extend from the aneurysm and involve the lumen of an artery arising from the basilar artery and so cause ischaemia. On the other hand, it seems unlikely that platelet embolisation from an extracranial source was the cause because the patient had no stenosis or ulceration in these vessels; neither a cardiac source, systemic hypotension, nor underlying conditions predisposing to hypercoagulability were identified.

Regardless of the physiopathological mechanisms, the case reported shows that the PSS syndrome can occur secondarily to a basilar aneurysm and provides another example of the many potential aetiologies of lacunar syndromes.

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Ectopic parasellar pituitary adenoma with subarachnoid seeding

There are very few descriptions of an ectopic supra- or parasellar pituitary adenoma in a patient with a normal intrasellar pituitary gland. In one case the tumour originated in the pars tuberalis,¹ in another in the parasellar region,² and in a third case the tumour arose either in the sphenoid or in the pars tuberalis.³ We report a further case of ectopic parasellar pituitary adenoma which was complicated by subarachnoid seeding 13 years after initial treatment by craniotomy, radiotherapy and systemic chemotherapy.

In 1975 a 45 year old man who had been impotent for one and a half years was found to have a left sided oculomotor paresis. Radiographs showed a normal pituitary fossa and paranasal sinuses. Orbital venography, carotid angiography and basal cisternography suggested a left parasellar lesion. The patient's hormonal iodine concentration was low (0.23 m mol/L; normal range 0.24–0.48), his serum prolactin concentration was not determined. Craniotomy disclosed two mucoid tumours. One was pea-sized, seemed to infiltrate into the left oculomotor nerve near its entrance into the cavernous sinus, and was

removed incompletely. The other tumour was bean-sized and located under the left optic nerve; this tumour was removed completely. The pathologists who were consulted had difficulty in making a definite diagnosis: reticulosarcoma and malignant epithelial tumour were suggested. An extensive search did not detect tumours in other sites. The patient was treated with radiotherapy followed by systemic chemotherapy (12 courses of cyclophosphamide and vincristin). After whole brain irradiation up to 23.50 Gy treatment coned down to (para)sellar areas was given up to 60 Gy. Special attention was paid to include the suprasellar area for up to 3 cm, and the whole of the sphenoid; the clivus was only partly irradiated. In 1979 a CT of the brain revealed nothing abnormal.

In 1988, the patient was found in an unkempt condition and was admitted in a delirious state and with neck rigidity. Body temperature was 34.7°C. T4, FTA index and T3 uptake were 65 nmol/l (N = 80–150), 55 nmol/l (N = 70–170) and 0.66 nmol/l (N = 0.95–1.20) respectively. Serum TSH was normal. CSF protein was 20 g/l, cell count was normal and cytological examination of the fluid did not identify tumour cells.

The patient's condition improved with general supportive treatment and levodopa and dexamethasone. CT and MRI of the brain and spinal cord identified a mass located anterolateral to the pons and medulla oblongata, mainly on the right side and extending down to the sixth cervical vertebra. Intraspinal lesions were also present in the high and mid thoracic regions. To obtain a tissue diagnosis, stereotactic biopsies were taken under local anaesthesia. Smears made during this procedure suggested a pituitary adenoma. The patient died suddenly on the third postoperative day.

At necropsy the immediate cause of death appeared to be aspiration of food. No other abnormalities were found outside the central nervous system. Grossly as well as microscopically no abnormalities were found in the para- and suprasellar region, pituitary fossa or sphenoid sinuses.

Mucoid tumour tissue was found in the lesions shown by CT and MRI. Histopathologically, the tumour consisted of sheets of medium-sized epithelial cells with round or ovoid nuclei surrounded by faintly stained agranular cytoplasm. Mitotic figures were rare and nuclear polymorphism was inconspicuous. Immunoreactions for keratin and NSE were positive, and for S-100 weakly positive. Very few cells reacted positively for chromogranin A. Between 10–25% of the cells reacted strongly for prolactin, less than 10% for growth hormone. Ultrastructural examination of stereotactic biopsy showed a few 100–200 nm dense granules, often surrounded by a membrane (figure). The histological appearance of the specimens obtained from 1975 and 1988 were identical; the paraffin blocks from 1975 were not available for further study. The diagnosis therefore was of an ectopic parasellar pituitary adenoma, with a normal intrasellar pituitary producing leptomeningeal seeding.

The coexistence of an ectopic supra- or parasellar pituitary adenoma with a normal intrasellar pituitary gland is not surprising in view of the recognition by Hori⁴ of "aberrant" adenohypophyseal cells in the leptomeninges surrounding the pituitary stalk and infundibulum. These cells were found in all foetuses examined and in 75% of adult necropsies. In the present case, between