Giant middle cerebral aneurysm presenting as hemiparkinsonism

Sir: Aneurysms with a diameter greater than 2.5 cm are designated "giant" and are uncommon, comprising only 5% of all intracranial aneurysms. They usually present either with subarachnoid haemorrhage or progressive visual loss and headache due to their frequent proximity to the carotid and ophthalmic arteries. They may however present as a direct result of their space occupying effect and usually this produces a lateralised weakness or sensory disturbance. I report here a case of a giant middle cerebral aneurysm presenting with hemiparkinsonism.

A 39 year old teacher had a 7 month history of difficulty writing and manipulating a pen. His writing had become small, particularly when using a blackboard at school and at the end of sentences. There was a tremor of the right hand at rest and also at certain angles when doing precise tasks such as shaving or holding a glass. He had noticed increasing difficulty in using the right arm with incoordination of rapid finger movements. He complained that the right arm would hang by his side and that he needed to use the left arm more than previously. He had noticed an absence of right arm swing with walking. In the 3 months before his admission, his right leg had begun to drag and he had noticed that he had generally slowed down. Daily tasks would take twice as long as before he became ill.

There was no significant past medical or family history. He smoked twenty cigarettes per day and had up to four pints of home brewed beer each day.

On examination he walked without swinging the right arm. His cranial nerves were normal. There was a tremor of the right hand (4 to 6 Hz) in the resting position. There was mild cogwheel rigidity of the right arm with normal power. Tone was similarly increased in the leg though power was reduced in a pyramidal distribution. Fine movements of the right hand were impaired as were rapid movements. His reflexes were brisk on the right side with a right extensor plantar response.

A CT head scan showed an extensive area of mixed attenuation in the left temporal lentiform region with mass effect and shift of the mid-line. After injection of contrast, there was a central area of increased enhancement (fig). Carotid angiography showed a left middle cerebral artery bifurcation giant aneurysm mainly containing thrombus.

The patient subsequently underwent a left fronto-temporal craniotomy with excision of a giant middle cerebral aneurysm. Following surgery, he made an excellent recovery with complete resolution of the extrapyramidal features. However, there was a residual mild right hemiparesis.

Giant aneurysms have a pleomorphic pattern of clinical presentation. In Bull's series of 22 cases, nine had visual disturbance and a similar number had headaches. Only one case presented with tremor in one arm with associated hemiparesis but no other extra pyramidal features. Similarly in other series, no case of an extrapyramidal syndrome has been described.

In our case, the aneurysm led to Parkinsonism and the initial erroneous diagnosis of Parkinson's disease, though the associated mild right pyramidal signs argued against this diagnosis. Presumably capsular compression, together with the involvement of the basal ganglia on that side, led to the combined pyramidal and extrapyramidal syndrome.

Tumours may produce Parkinsonism either by pressure on the basal ganglia or more rarely direct infiltration. Removal of these neoplasms may result in improvement.

Structural lesions of the basal ganglia also produce hemidystonic syndromes. Most of these cases have been due to haemorrhages, infarction or tumours, and none were secondary to aneurysm.

The original CT scan of our patient suggested that the mass lesion with mixed attenuation was likely to be a tumour, possibly a glioma or meningioma. The left carotid angiogram demonstrated the aneurysm containing thrombus at the bifurcation of the left middle cerebral artery. Although most neurosurgeons do request angiography before exploration of unusual lesions on the CT scan, this case demonstrates the need for caution before proceeding to biopsy or removal. This is particularly important with the increased use of stereotactic surgery for biopsy of deep-seated intracerebral mass lesions. At surgery the aneurysm was extremely large and filled with thrombus. This was in contrast to the angiogram and the CT scan appearance, where most of the lumen did not fill. The displacement of vessels on the angiogram and the CT scan appearance demonstrated the actual size of the aneurysm. The excellent response to surgical removal of the aneurysm was most satisfying, though whether he will be predisposed to developing lateralised Parkinsonism with time is uncertain. The possibility that a lowered dopamine reserve is present in these patients who develop Parkinsonism with a structural lesion remains unanswered.

I am very grateful to Dr Ralph Ross Russell for his permission to report on this case. Professor L Symon performed the neurosurgical operation.

Michael Gross
The National Hospital for Nervous Diseases, Queen Square, London WC1N 3BG, UK

References


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Fig. Enhanced CT scan showing an extensive area of mixed attenuation in the left temporal lentiform region with mass effect.
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M Gross

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