Letters

favour fistula as the cause of dysphasia rather than chronic epidural haematoma. Besides, the sudden progression of neurological signs 14 days after injury may be attributed to acute epidural haematoma due to ruptured pseudoaneurysm. This was the presentation in over half the reported cases of pseudoaneurysm, particularly in those in whom neurological deterioration occurred at an average of 11 days after the time of initial injury. Coincident existence of an arteriovenous malformation and mycotic aneurysm of the peripheral cerebral artery were considered as the differential diagnosis. However, in the absence of preoperative history of meningitis, cardiac disease and epilepsy, these possibilities were considered unlikely. Besides, both of these pathologies of this patient showed evidence of trauma to the adjacent bone, dura or cerebral tissue, thus implicating trauma as their sole cause.

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A case of Parkinsonism following striatal lacunar infarction

Sir: The concept of vascular Parkinsonism is a controversial one. Critchley first introduced the concept in 1929 when he proposed that Parkinsonism due to vascular disease could be distinguished from idiopathic Parkinson’s disease on the basis of clinical and pathologic criteria. However, he did not present individual case data, either clinical or pathologic, to demonstrate convincingly how to distinguish these two conditions or to exclude other recognised causes of Parkinsonism. Subsequently, other authors have questioned the existence of vascular Parkinsonism as a distinct entity, based on a lack of clinical and pathological evidence. Nevertheless, there are authorities who accept vascular Parkinsonism as a recognisable condition, generally associated with chronic hypertension and clinical evidence of multiple small lacunar infarctions.

A diagnosis of a vascular aetiology of Parkinsonism in a particular case would be acceptable if the following criteria were met: (1) the Parkinsonian signs were acute in onset; (2) the signs showed spontaneous improvement; (3) other known causes of Parkinsonism were excluded; (4) pathological examination failed to show either neuronal loss in the substantia nigra or Lewy bodies, but did show vascular lesions affecting the corpus striatum. In the absence of histopathologic evidence, vascular Parkinsonism cannot be diagnosed with certainty, but it can be considered the probable diagnosis if the clinical criteria are met, and there is neuroradiologic evidence of striatal infarction. We recently encountered an example of probable vascular Parkinsonism, which supports belief in the existence of this condition.

This 37-year-old left-handed man with a 4-year history of untreated hypertension had sudden onset of weakness of the right arm and leg, slurred speech and right perioral numbness. His speech quickly returned to normal and he regained most of his strength in two hours. He denied difficulty with comprehension or formulation of language. He had no history of diabetes mellitus, heart disease or cigarette smoking. He had never taken antipsychotic drugs. On examination his blood pressure was 180/100 mm Hg. He had diminished facial expression. Cranial nerves were intact. There was mild rigidity of the neck, more pronounced rigidity in the right arm and leg, very slight rigidity of the left arm and leg. Rapid successive movements were diminished and slow on the right side and less so on the left side. Strength was normal. Deep tendon reflexes were brisk bilaterally, more so on the right. A Babinski response was present on the right side. Sensory examination was normal. He walked without swinging his right arm. There was postural instability with retropulsion. Cerebellar testing was normal. CT scan (fig) showed generalised mild to moderate atrophy, bilateral putaminal lucencies and a right cerebellar hemispheric lucency. A four-vessel cerebral angiogram was normal. Cerebrospinal fluid was normal. The level of homovanillic acid (HVA) in the cerebrospinal fluid was 41 ng/ml; the mean value for Parkinson’s disease patients in our laboratory is 25.3 ± 12.3 ng/ml.

He improved gradually over the next 10 days in hospital. After discharge he slowly improved further without any anti-parkinsonian medication, with residual rigidity on his right side. Three months after discharge he noted sudden weakness of all extremities and slowing of his gait. Ten minutes later he noted numbness of his right foot and right hand. All these symptoms resolved in about one half hour. His blood pressure was 150/100 mm Hg. There was no change in his neurological state. A repeat CT scan showed no new lesion. By the next day he was less rigid.

Several features of this case make the diagnosis of vascular Parkinsonism possible.

References


Fig CT scan showing bilateral putaminal lucencies (arrows) with a larger one on the left and moderate generalised atrophy.
The young age of the patient, appearance of his symptoms after a stroke, spontaneous improvement unrelated to treatment, lacunae seen on CT within the basal ganglia, and a relatively high level of HVA in cerebrospinal fluid, higher than usually seen in idiopathic Parkinson's disease, are in favour of this diagnosis. Other causes of Parkinsonism, such as normal pressure hydrocephalus or neuroleptic treatment were excluded.

While earlier reported cases of vascular Parkinsonism represent additional probable examples of this condition, they are open to question in a number of respects. Hughes and others described a 39-year-old hypertensive man who developed a Parkinsonian mask following a stroke, and who had bilateral caudate infarcts revealed at necropsy. It is not clear from the report if the patient had Parkinsonian features beyond facial masking. Tolosa and Santamaria reported three elderly men, two of whom were hypertensive, who had the insidious onset of Parkinsonism and who had infarcts of the basal ganglia revealed by CT scan. Although these cases are likely to be valid examples of vascular Parkinsonism, the insidious onset at a late age makes it difficult to definitely exclude the coincidental occurrence of idiopathic Parkinson's disease with what could have been silent basal ganglia infarctions. The concept of vascular Parkinsonism is supported by the study of Parkes et al who noted higher CSF HVA levels in patients with vascular Parkinsonism, diagnosed by clinical criteria, as compared with patients with idiopathic Parkinson's disease.

Our patient, and those cited above, represent probable examples of lacunar cerebrovascular disease, and there was associated hypertension in most cases. It is this type of cerebrovascular disease, rather than large vessel atherosclerotic disease, which has been associated with vascular Parkinsonism. Although Critchley titled his paper “Arteriosclerotic Parkinsonism” he emphasised the presence of lacunes on pathological examination. There is no evidence that multiple cerebral infarcts on the basis of diffuse atherosclerotic disease cause Parkinsonism, and it is this unsubstantiated relationship which has been the subject of many rebuttals of the concept of vascular Parkinsonism.

Although we presume our patient represents an example of vascular Parkinsonism, which is distinguished from idiopathic Parkinson's disease, we suspect that Parkinsonism is rarely vascular in aetiology. The rarity of this condition is indicated by the paucity of well-documented examples of it in the literature, and by its infrequency in a large series of Parkinsonian patients. The authors thank Dr Lucien Cote for the determination of the patient's CSF HVA level.

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