Ruptured mycotic aneurysm during a computed tomogram brain scan

Sir: Early diagnosis and treatment of mycotic aneurysms may reduce the high mortality associated with this condition. In developing countries the incidence of rheumatic fever and bacterial endocarditis is still high and has a significant morbidity and mortality. The neurologic manifestations of bacterial endocarditis are well-known, and a high rate of rupture of mycotic aneurysms frequently has been reported. A unique case is reported showing spontaneous rupture of a mycotic aneurysm during computed tomogram (CT) brain scanning.

An 18-year-old girl with known rheumatic valvular disease presented in June 1981 with features of bacterial endocarditis. Klebsiella species was isolated on blood culture and she was treated with Tobramycin and Cephalosporin intravenously for five weeks. She responded well except for two episodes of headache during that period.

In November 1981 she was re-admitted with severe photophobia, neck stiffness, headache, right-sided weakness and pyrexia. Lumbar puncture revealed raised protein, low sugar and numerous red cells. The patient was treated for subarachnoid haemorrhage with sedation and bed rest. The provisional diagnosis was mycotic aneurysm. Three days later when the localising signs on the right side and the severe headache had improved a CT scan was performed. On arrival in the radiology department the patient was fully conscious and orientated. The pre-contrast CT scan showed a high density in the deep parietal lobe adjacent to the posterior limb of the internal capsule on the left side. This was thought to represent the site of initial bleeding due to aneurysm formation (fig A). The surrounding low-density on the left side at the level of the third ventricle was in keeping with ischaemia related to vascular spasm. One hundred ml of Conray 420 (sodium iothalamate 70%) was given intravenously over a few minutes. Shortly thereafter there was an acute deterioration in the patient’s condition with coma and Cheyne-Stokes breathing. The post-contrast CT scans obtained showed extensive high-density in the subarachnoid space, cisterns and third ventricle in keeping with an acute haemorrhagic episode (fig B). With conservative treatment marked improvement was noted. Three weeks later the patient died suddenly following a massive intracerebral bleed.

Spontaneous rupture of aneurysms during angiography has been reported previously. The majority showed extravasation of the opaque medium into the subarachnoid space or cerebral substance. A few cases showed ventricular opacification as well. Many authors have considered contrast leakage during angiography to be co- incidental. Goldstein and Hoff suggested that the sudden increase in intra-arterial pressure during angiography was the cause of the rupture. The increased arterial blood pressure has been attributed to the mechanical effect of a high injection pressure followed by a local vasodilatory effect of the contrast medium. This mechanical pressure effect cannot be incriminated in our patient as the contrast was injected intravenously via an antecubital vein.

Mycotic aneurysms nearly always are due to bacterial endocarditis and may involve any artery in the body. In the absence of endocarditis the commonest causes of a bacteraemia are lung and bone infections. Although there is a high incidence of neurological manifestations with bacterial endocarditis (33%, 39%). only 2–3% of these patients develop mycotic aneurysms. Most mycotic aneurysms occur in patients under 40 years of age. In 17-6% the aneurysms are multiple. The most familiar clinical presentation is the patient with known endocarditis who suffers a subarachnoid or intra-cerebral haemorrhage. Mycotic aneurysms typically lie peripherally in the cerebral arteries rather than near the circle of Willis. Angiography gives precise information regarding the size, location and degree of spasm related to the aneurysm. The smallest intracranial aneurysm visualized by CT is about 5 mm in diameter. This is improved with contrast enhancement. When CT was performed within 5 to 7 days after the bleeding episode, subarachnoid blood has been detected in 95–100% of cases. The blood distribution can suggest the location of the aneurysm. Some authors conclude that patients with bacterial endocarditis who develop sudden severe headache, focal neurological signs or symptoms, or seizures should undergo serial cerebral angiography.

References


Levodopa induced chorea in Meige syndrome

Sir: Meige syndrome is an adult-onset focal dystonia which affects the face and jaw causing blepharospasm and painful oromandibular spasms.¹ No histological or neurochemical abnormalities have been found, but some clinical pharmacological studies point towards a striatal dopamine preponderance.² We report here the occurrence of levodopa-induced peak dose chorea in a patient with Meige syndrome.

A 44-year-old right-handed woman with no antecedent medical, psychiatric or drug history and no family history of neurological illness, developed writer’s cramp shortly after her promotion to supervisor of a typing pool in 1972. Her disabilities were initially mild, but gradually increased to encompass other dexterous tasks such as unscrewing bottle tops and holding a badminton racket. In October 1981 she resigned from her job because she was finding it too mentally taxing and within two weeks, intermittent blepharospasm had developed. One month later her mother, who had a long history of depression, committed suicide and within days of this, intermittent yawning movements and tonic jaw spasms also appeared. Over the next three months symmetrical involuntary facial dystonia became increasingly distressing. At times she was unable to read, type or watch television without developing intense blepharospasm; bright lights were another powerful trigger. The jaw contractions on the other hand, were adversely affected by emotional upsets. Her personality and temperament were normal and there were no overt features of depression. In addition to paroxysms of uncontrollable sustained eyelid closure, facial spasms and trismus, the platysma and right-sided neck and shoulder muscles were occasionally involved causing neck torsion and shoulder elevation. Posture of the right arm was normal at rest and when outstretched, but the limb adopted a typical dystonic posture when writing. Cogwheel rigidity was detectable at the right wrist on synkinesis and there was a decreased right arm swing when she walked. Normal investigations included cerebrospinal fluid examination, electroencephalography and computed tomography. Treatment was begun with levodopa (100 mg) in combination with benserazide (25 mg) administered in two divided daily doses. The dose was increased over ten days to 100 mg of levodopa eight-hourly in combination with benserazide (25 mg) in two divided daily doses. After three weeks, chorea appeared in the fingers of the right hand and rapidly spread to involve the whole of the right arm. The right-sided painful stiffness was abolished, but the dystonic facial and jaw spasms increased in severity and levodopa was discontinued. The drug-induced chorea then subsided rapidly over forty-eight hours, but she became markedly depressed and improvement in her dystonia occurred only slowly over several weeks. Her right-sided rigidity was subsequently alleviated by benhexol 2 mg twice daily without benefit to the dystonia.

The occurrence of levodopa-induced dyskinesias provides circumstantial evidence implicating dysfunction of the basal ganglia in focal dystonias. The modest aggravation of the facial spasms would also be in keeping with the notion of a relative dopaminergic excess in the corpus striatum. It is of interest that, in contrast, dopamine receptor agonists have been reported to be of benefit in both Meige syndrome³ and levodopa-induced end-of-dose focal dystonia in Parkinson’s disease.⁴

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