References


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Can migraine cause multiple segmental cerebral artery constrictions?

Sir: Before computed tomography (CT) scanning, angiography was extensively used in the investigation of patients with migraine, in particular those with complicated forms of migraine. It became clear that in between attacks cerebral angiograms were normal except for the low incidence of structural lesions such as arteriovenous malformations.1 Likewise during attacks of migraine most angiograms are normal2 arguing against Wolff’s suggestion that the aura in migraine is due to intracranial artery vasospasm.3 However, many authors have pointed out that intra-arterial contrast medium can provoke migraine and many of the limited number of reports of cerebral arterial changes during migraine attacks were induced this way.4,5 These reports mainly mention two different patterns, firstly the failure to visualise one or more of the major intracranial arteries6 and secondly a transient proximal constriction most commonly of the infraclinoid portion of the internal carotid artery.7,8 Recently there have been three reports which raised the possibility that multiple separate narrowed arterial segments can also be found in migraine.9-12 We present a further case of migraine with such angiographic abnormalities. The possible significance and differential diagnosis is discussed.

A 41 year old right handed Causasian accounts clerk was admitted to hospital for investigations of menorrhagia. Five years prior to admission she developed attacks of migraine. These occurred two to three times a year and consisted of a unilateral throbbing headache, lasting 6 hours associated with photophobia, phonophobia and recurrent vomiting. During her attacks she suffered from a tingling sensation in the face and both hands. Her father suffered from classical migraine. She also had a history of 17 years intermittent bloody diarrhoea, associated with recurrent bilateral irritative mouth ulcers, and joint pains affecting ankles and elbows. Her bowel disorder had never been investigated but was thought to be ulcerative colitis. A barium enema during her admission was normal and a rectal biopsy showed non-specific proctitis.

After 2 weeks in hospital undergoing gynaecological and gastroenterological investigations she developed sudden severe bifrontal headache, recurrent vomiting, and sensation of flashing lights in both eyes. A numbness in her face and hands and profuse sweating. These severe symptoms persisted for 3 days. Initial examination showed marked stiffness but no Kernig’s sign, normal fundi, no focal neurological deficits but a blood pressure of 210/100 mm Hg. A subarachnoid haemorrhage was suspected. CT scan was initially reported as showing evidence of subarachnoid blood over the convexity of the cortices with no evidence of intraventricular or intracerebral bleeding. On review the scan was subsequently felt to be normal. However, four vessel angiography was requested in response to the initial scan report. This revealed no aneurysms but instead showed multiple regions of narrowing affecting mainly the anterior cerebral arteries (fig).

A subsequent lumbar puncture was unremarkable; the pressure was 225 mm CSF, there were 3 white blood cells/mm3, 7/1 red blood cells/mm3, protein was 0-32 g/l and sugar was 4-4 mmol/l. The erythrocyte sedimentation rates were normal (13, 16 and 17 mm/hour). Other routine blood tests were normal including antinuclear factor, although her rheumatoid factor was positive at 1:40.

The patient’s headache improved but fluctuated over a 7 day period. In view of the possibility of a vasculitic disorder the patient was started on prednisolone 60 mg/day. The headaches continued to...
improve although despite being on a decreasing dose of prednisolone she had bouts of headache twice a month and during two such attacks suffered right sided weakness and dysphasia lasting 4 hours. Propranolol treatment was started with some decrease in the frequency of her attacks. The angiographic appearances are compatible with arterial spasm, vessel wall oedema or vasculitis. The current case probably suffers from ulcerative colitis which is not associated with cerebral vasculitis. Furthermore there was no evidence of active collagen disease at the time of the investigated attack of headache as indicated by the normal erythrocyte sedimentation rate and antinuclear factor. The unremarkable rectal biopsy and barium enema showed that her colitis was quiescent. However, without arterial biopsies of the appropriate segments it is impossible to completely exclude vasculitis although the nature of the present episode and her previous headaches favour the idea that this was migraine.

Serdaru et al described a patient with transient aphasia, hemianopia, headache and a seizure in whom an initial angiogram showed multiple distal segmental narrowings which were not present in a repeat study 2 weeks later. A temporal artery biopsy failed to show any inflammatory changes despite its narrowed angiographic appearance. The authors discussed the diagnosis of benign isolated cerebral vasculitis (also called cerebral post-partum angiopathy) but point out the lack of any histological reports and conclude that their case could well have been due to migrainous vasospasm. Uzawa et al described somewhat similar angiographic appearances in a woman 4 weeks post-partum who developed severe headache and vomiting in whom a temporal artery biopsy was also normal. They too attribute the changes to migraine. Garnic et al showed segmental narrowings in a case of atypical cluster headache affecting proximal portions of the middle and anterior cerebral arteries.

It has been suggested that hyperventilation due to pain might cause vasoconstriction and explain the changes in cerebral blood flow and angiography in migraine. This is unlikely to be relevant to the present case both because angiography was carried out under general anaesthesia and also because hypocapnoea produces generalised and not focal changes.

Similar appearances have been reported after head injuries and in association with a phaeochromocytoma in the absence of subarachnoid bleeding. Head injuries can lead to transient neurological deficits akin to those seen in migraine but the angiograms in the post traumatic cases described by Oka et al were normal.

It is interesting that in three of the four cases described with multiple constricted segments the angiogram was carried out during the headache phase. If these appearances were due to vessel wall oedema it would be compatible with Moskowitz's hypothesis that the headache may be related.

Fig. Lateral view of left internal carotid angiogram showing multiple short areas of arterial narrowing. Those illustrated at higher magnification (A and B) are indicated with an arrowhead.
to the release of vasoactive peptides from trigeminal sensory perivascular fibres.\textsuperscript{18}

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References


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Occlusion of the basilar artery in a 7 year old boy

Sir: Occlusion of the basilar artery is uncommon in childhood; a review of the literature disclosed only 29 cases documented in children up to 16 years old;\textsuperscript{1–11} there was a clear male dominance.\textsuperscript{6} The most frequent cause for occlusion of the basilar artery in adults is arteriosclerosis,\textsuperscript{4,12} while in children the reported cases have been associated with congenital malformations,\textsuperscript{3,2} embolism,\textsuperscript{9,8} arthritis,\textsuperscript{7,11} cervical injuries,\textsuperscript{1,4,7–10} and idiopathic causes.\textsuperscript{1,4,7–10} We describe a 7 year old boy with occlusion of the basilar artery verified by angiography and a locked-in state in whom had had previous craniocervical injury.

A 7 year old boy was admitted to our service because of sudden headache, vomiting and lethargy. Two weeks earlier he had sustained injuries to the head and cervical region, while being violently shaken against a tree trunk by an older child, resulting in malaise and drowsiness lasting a few hours but without loss of consciousness. Ten days before admission he had unsteady gait and cerebellar signs in the right limbs lasting a few minutes, accompanied by headache and vomiting. On admission his temperature was 38°C, blood pressure 120/70 mm Hg and pulse rate 116/min. He had decerebrate rigidity, oculeocephalic deviation toward the left and spastic tetraparesis with bilateral Babinski sign. The pupils were symmetrical with normal reactions. The corneal reflexes were present. He was able to blink voluntarily, reacting to verbal commands by moving the eyes up and down. There was involuntary downward jerking of the eyes with slow upward drift (ocular bobbing), and sucking mouth movements without uttering words and his facial expression at times mimicked crying, either spontaneously or after stimuli. The child was admitted to the intensive care unit. The remainder of the physical examination, cardiology studies, lumbar puncture and laboratory analyses including clotting tests, antinuclear factor tests and syphilis tests all were normal, as were chest films and cranial and spine films. The EEG revealed a slowed basic rhythm of low voltage and occasional bursts of bilateral delta waves in the frontal and occipital areas. A CT scan showed areas of decreased density at the level of the left middle cerebellar peduncle, left lateral half of the pontine and left cerebellar hemisphere. Angiography showed normal vertebral arteries and a well filling supranumerary branch originating in the right subclavian artery. A complete proximal occlusion of the basilar artery was observed (fig). Left carotid arteriography revealed partial and irregular filling of the distal portion of the basilar artery through the left posterior communicating artery.

Treatment was begun with heparin sodium i.v. and because of worsening in ventilation, tracheostomy was performed and a volumetric respirator connected. Three weeks later the patient still opened and closed his eyes, responded to visual stimuli, breathed spontaneously and could perform voluntary proximal movements with the upper limbs, but did not speak or respond to commands and no contact could be established. Four weeks after admission the patient died because of massive haemorrhage as a complication of the tracheostomy. No post-mortem examination could be performed.

Fig Complete occlusion of the basilar artery at its proximal third (arrow). Left vertebral artery is normal.