the superior-anterior aspect of the right frontal lobe white matter as well as a microhaemorrhage in the right parietal region (fig 1B). The area of haemorrhage was hypointense on T2 (fig 1C) and isointense on T1 weighted sequences (fig 1D), consistent with acute haemorrhage. There was no MRI evidence of a cavernous haemangioma, arteriovenous malformation, or tumour. Magnetic resonance angiography was not done.

A brain biopsy of the right frontal lobe done on the seventh hospital day showed degeneration of small and medium sized arteries. Vessel walls were thick and hyalinised in the grey matter, white matter, and meninges. PAS staining was positive and the muscular coat of the large vessels revealed degenerative changes. Electron microscopy showed the granular osmiophilic material characteristic of CADASIL. *Notch*3 gene testing revealed a R133C mutation in exon 4, consistent with the diagnosis of CADASIL. The patient remained normotensive throughout his hospital stay. On the fifth hospital day he developed aspiration pneumonia requiring mechanical ventilation. He died eight days later as a result of this pneumonia.

Comment

This is the second report of spontaneous cerebral haemorrhage in a patient with CADASIL. In 1977, Sourander and Walinder reported a 29 year old man with hereditary multi-infarct dementia on anticoagulants, with a large haemorrhage in the right hemisphere.[5] This family was thought to be one of the first with CADASIL; however, recent testing for *Notch3* mutations in the family has not confirmed that diagnosis.[6] In 1992, Baudrimont *et al* reported a case of massive left cerebral haematoma involving the caudate nucleus, internal capsule, and thalamus in a 40 year old normotensive woman who was a member of a large CADASIL family. She had no known history of other risk factors for haemorrhage.[7]

The index patient in this report had no evidence of coagulopathy and no history of previous hypertension, cerebral haemorrhage, or anticoagulant therapy. The patient could have experienced a haemorrhagic contusion related to a closed head injury during his unwitnessed fall before admission, but there was no evidence of trauma on physical examination or on head CT. On MRI there was no evidence of a cavernous haemangioma, arteriovenous malformation, or neoplasm. Necropsy was not carried out.

Ultrastructural analysis of small arteries in human postmortem brain and skin in patients with CADASIL shows breakdown of the arterial wall cytoarchitecture, which may help explain the propensity for microhaemorrhages.[8] The first *notch3* transgenic mouse shows early widening of the subendothelial and intra-smooth-muscle spaces in the vascular smooth muscle cells, denoting weakening of the arterial wall and increasing susceptibility to micro- and macrohaemorrhages.[9]

This case report supports the growing evidence for both ischaemia and haemorrhage in a variety of small artery diseases including amyloid angiopathy and CADASIL.[10 11] Clinicians may need to consider the possibility of haemorrhage when evaluating new events and deciding on treatment for stroke prevention in patients with CADASIL.

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Urinary retention caused by a small cortical infarction

The cortical representation of micturition is speculated to reside in the medial frontal lobes.[1 2] Lesion pathology, however, varies from acute stroke to a neoplasm, and there is not necessarily a small, distinct lesion.[2] We report a case of urinary retention in which the main presenting symptom is thought to have been caused by a small cortical infarction.

Case report

One morning, a 66 year old, right handed man had difficulty urinating. He had no history of voiding difficulty, diabetes mellitus, injury to the lower urinary tract, or neurological disease. Digital rectal examination and ultrasonography of the prostate detected no enlargement. Urinalysis showed no haematuria or pyuria. He was not taking any medications that cause voiding dysfunction. There was no urinary incontinence, but he had difficulty in voiding even though he felt the bladder was full. At that time, he also had difficulty in lifting his left arm and leg and so was brought to our hospital. Neurological examination in the emergency room found no weakness, and he was sent home. Later, he experienced urinary retention and visited the emergency room again. His post void residual urine volume was 350 ml, and an urinary catheter was inserted. At that time the patient was alert, and his cranial nerves were intact. Limb muscle strength was normal. Sensory examination was unremarkable. Tendon reflexes were normal in all four limbs. Tandem gait and standing on one foot were difficult. He had normal bladder sensation but difficulty in urinating. Drip infusion pyelography revealed no abnormality in the upper urinary tract or the form of the bladder. Filling cystometry showed stable detrusor with normal bladder sensation, whereas acontractile detrusor was noted in the voiding phase. He could void only with strain, having a peak flow rate of 5.0 ml/s and a voided volume of 135 ml. Diffusion weighted MRI, performed on the day of onset, showed a small, distinct, high intensity signal, and T1 weighted imaging showed a low signal in the right caudal part of the anterior cingulate gyrus, indicative of an infarct in the acute stage (fig 1A and C). No definitive infarct was observed elsewhere. MR angiography showed no occlusion or stenosis of the intracranial vessels. An electrocardiogram was normal. Transthoracic echocardiograms showed no abnormal findings. The urinary catheter was withdrawn 3 days after admission, and he had no subsequent difficulty with urination. His gait returned to normal about the same time.

Discussion

In the acute stage of a cerebral vascular accident, the presenting symptom often is urinary retention due to detrusor areflexia,[3] but patients who have this problem usually have a major stroke with severe neurological deficits.

To the best of our knowledge, this is the first report in the English literature of urinary retention, although temporary, caused by a small cortical infarct as shown by diffusion weighted MRI.

Various cortical areas are activated during voiding because a network of brain regions is necessary for voiding modulation.[4] The locations of the primary cerebrum cortical areas for voiding and storage are speculated to be separate, the former being at the para-central lobule.[2] A PET study found normal micturition to be associated with activation of the middle frontal gyrus, superior frontal gyrus, superior precentral gyrus, thalamus, and the caudal part of the anterior cingulate gyrus in the left hemisphere.[5] Another recent PET study showed that increased brain activity related to increasing bladder volume was located in the bilaterally mid-cingulate cortex, while that related to decreased urge to

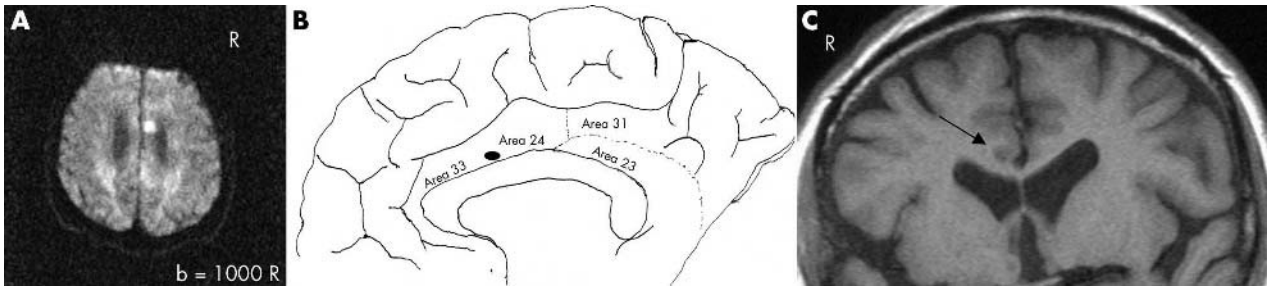

Figure 1 Diffusion weighted MRI. (A) The axial section shows a small high signal in the right caudal part of the anterior cingulate gyrus. (B) The closed ellipse denotes the lesion in the cortical map (map modified from Brodmann, 1909). (C) In the T1 weighted MRI procedure, the coronal section shows a low intensity signal in the right caudal part of anterior cingulate gyrus (arrow). (Imaging condition: a negative tilt of −20° to the orbitomeatal line.)

void was bilaterally in a different portion of the mid-cingulate gyrus.[4]

Although the infarct in our patient was located in the caudal part of the anterior cingulate gyrus, it was on the right side, nearby the region activated in the PET study.[4] SPECT showed increased blood flow in the right medial frontal area, indicative that urinary retention was due to ''decreased urge to void'', and decreased flow in the right medial parietal lobe, which might explain the gait disturbance, in light of the essentially normal sensory examination. Unfortunately, a PET scan was not available in our hospital (Kameda Medical Center). Because there has been no report of an isolated lesion of the cingulate gyrus causing hemiparesis, these brain imaging studies indicate that the left hemiparesis, which disappeared within a half day of onset, could have been due to a transient ischaemic attack.

Urinary symptoms disappeared 3 days after admission, probably because the cortical neuron network compensated by providing a functional alternative to the lesion damaged by the infarct. This is similar to the condition of urinary incontinence after cerebral infarction, as is well documented. The laterality of the lesion in this patient differs from that in a previous PET study[4] which showed bilateral activation in the cingulate gyrus. Because this report cites only a single case, its applicability is limited. Additional lesion studies of patients with micturition disturbance due to small cortical infarcts should help to identify the anatomical cerebral structures involved in voiding.

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BOOK REVIEW

Neuropsychiatry and behavioural neurology explained

Edited by A J Mitchell. Published by Saunders, London, 2004, £69.00 (hardback), pp 500. ISBN 0-7020-2688-3

This is an ambitious project for a single author; the whole of neuropsychiatry explained using an up to date, evidence based review of the literature, and in a format that is designed to be attractive to read. There are numerous figures, boxes, lists with bullet points, and ''clinical pointers'' to break up the text.

Although aimed particularly at liaison and old age psychiatrists, this book will have wide appeal and be of interest to neurologists. They will be able to quickly access clinically relevant discussion of the neuropsychiatric sequelae of common neurological disorders. The core sections of the book, on dementia and delirium, neuropsychiatric treatments, and the psychiatric complications of neurological diseases, are excellent. The discussion is practical and to the point. The reader is not stifled with references strewn in the text. They must therefore have confidence in the assertions of the author; I am confident that we are being offered accurate information. But at times the style feels a little pedantic; for example, those of us who dared to believe that alcohol might cause depression are put firmly in our place. Another quibble I have is the value of some of the lists/classifications which were of uncertain provenance. We are, for example, given lists suggesting difference aetiologies for chorea versus athetosis, but some would be sceptical of the value in splitting choreoathetosis. Many classifications are based on neuroanatomical models of neuropsychiatry that need to be treated with caution.

The book strays into biological psychiatry, and a later section is devoted to understanding how neurological disorders result in neuropsychiatric symptoms, but this does cause a problem because some of the discussion of the neuropsychiatric sequelae of a particular disorder may not be found in the index chapter on that disorder, but in this later section. For example, the only discussion of suicide following head injury in the chapter on head injury is a single misleading sentence indicating that suicide accounts for 10% of head injury deaths. Yet, easily missed, 300 pages later, in the chapter on the neurological origins of suicide, is a more complete account of the relationship.

Overall, however, this book is a significant achievement. A large amount of material has been made readily accessible. There are no lacunae and the length of discussion of each disorder is proportionate to its importance. The book is to be trusted and recommended. One interesting innovation is a list of support groups and useful websites in the appendix. Neurologists and psychiatrists and their trainees have good reason to buy this book.

S Fleminger

CORRECTIONS

doi: 10.1136/jnnp.2004.047118corr1

Wood J M, Worringham C, Kerr G, *et al*. Quantitative assessment of driving performance in Parkinson's disease (*J Neurol Neurosurg Psychiatry* 2005;**76**:176–80). SD instead of SE was inadvertently inserted during the publication process in tables 2, 3, and 4. The p values are unaffected.

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Barber PA, Demchuk AM, Hill MD, *et al*. The Probability of middle cerebral artery MRA flow signal abnormality with quantified CT ischaemic change: targets for future therapeutic studies (*J Neurol Neurosurg Psychiatry* 2004;**75**:1426–30). The following errors appeared in this article:

(1) The median CT ASPECTS and DWI ASPECTS quoted in the article were both 8. These are incorrect and should be CT ASPECTS 9 and DWI ASPECTS 8;
(2) Sixty-six per cent (95% CI 0.56–0.75) of the patients had CT ischaemic change, while 81% (95% CI 0.72–0.88) of the DWI scans identified areas of hyperintense signal (not 67% and 79% quoted in the article);
(3) In figure 2 the numbers in parentheses on the x axis were incorrect. The correct numbers for each ASPECTS value are 10 (34), 9 (21), 8(12), 7 (11), 6 (12), and 5 (10).