The probability of middle cerebral artery MRA flow signal abnormality with quantified CT ischaemic change: targets for future therapeutic studies

P A Barber, A M Demchuk, M D Hill, J H Warwick, Pexman, M E Hudon, R Frayne, A M Buchan

Objective: In this study we define the probability of vascular abnormality in the middle cerebral artery (MCA) territory according to the extent of ischaemic change seen using computed tomography (CT). We assessed the sensitivity and specificity of the hyperdense middle cerebral artery (HMCA) and the “dot” sign using magnetic resonance angiography (MRA).

Methods: Patients presenting with ischaemic stroke had a CT scan (<6 h) prior to MRI (<7 h). A quantitative CT scoring system (ASPECTS) was applied to CT and diffusion weighted images (DWI) at baseline and follow up (24 h) by five independent observers. The presence of HMCA and the MCA “dot” sign was also evaluated. An expert reader assessed the 3D time of flight (TOF) MRA in the anterior circulation for areas of decreased vascular signal in the MCA territory, with an absent signal taken to represent severely reduced or absent flow.

Results: A total of 100 consecutive patients had baseline CT and MR scans. The median NIHSS was 9. The median CT ASPECTS was 8 and equalled the median DWI ASPECTS. There were a total of 10 HMCA and 19 MCA “dot” signs, with four patients having both HMCA and “dot” signs. A total of 47 MRA flow signal abnormalities were observed in the anterior circulation.

Conclusions: In the absence of accessible neurovascular imaging, the extent of CT ischaemia (ASPECTS) is a strong predictor of vascular occlusion. The CT hyperdense artery signs have a high positive predictive value but low negative predictive value.
For each non contrast examination the collapsed maximum intensity projection and source images were reviewed. The reader detailed the vascular abnormalities by the vascular signal intensity in the following extra- and intracranial vessels: the left and right internal carotid artery (ICA), the basilar artery (BA), the anterior cerebral arteries (ACAs), the left and right main stem of the middle cerebral artery segments (M1) and the MCA branch division (M2) regions of the MCA, and the left and right main posterior cerebral artery (PCA) segments.

**STATISTICAL ANALYSIS**

Data are reported using descriptive statistics. Baseline and follow up CT and DWI ASPECTS values were derived as the median of the results of the five observers. Comparisons of proportions, sensitivity and specificity, relative risks, and confidence intervals were assessed using 2×2 tables and exact methods. The presence of either M1 or M2 MRA flow signal abnormality was used to perform the sensitivity calculation. Logistic regression was used to adjust for baseline stroke severity in the assessment of the association between ASPECTS and arterial occlusion. Functional outcome was dichotomised into independence (mRS 0–2) and dependence or death (mRS 3–6).

**RESULTS**

A total of 100 consecutive patients were enrolled into the study, of whom 69% were male, and the mean (SD) age was 68 (13.9) years. The median NIHSS was 9 (interquartile range 3–16). A total of 39 patients received tPA: 33 patients received intravenous tPA alone, and six received a combined approach of intravenous tPA followed by intra-arterial thrombolysis into the angiographically defined thrombus.16 Of the patients 60% were independent (modified Rankin score 0–2) at 3 months (two patients were lost to follow up at 3 months). There were nine deaths, of which two were due to tPA-related fatal intracerebral haemorrhages. The median baseline CT ASPECTS was 8, which was the same as the median baseline DWI ASPECTS. A total of 67% (95% confidence interval (CI) 0.59 to 0.78) of the patients had CT ischaemic change (ASPECTS ≤9), while 79% (95% CI 0.70 to 0.87) of the DWI scans identified areas of hyperintense signal (DWI ASPECTS ≤9). Six patients (6%) had evidence of posterior circulation ischaemia on baseline DWI with or without coincident anterior circulation stroke. The mean (SD) time from symptom onset to CT was 117 (31) min compared to 219 (80) min to MR imaging (a mean (SD) difference of 102 (51) min, p = 0.0002). On CT, there were 10 hyperdense MCA signs and 19 MCA “dot” signs (four patients had both hyperdense MCA and “dot” signs) (fig 1). There were a total of 47 MRA flow signal abnormalities in the anterior circulation, of which 14 involved the ICA, either with M1 (five) or M2 (seven) alone, or both M1 and M2 (two). A total of 18 MRA abnormalities involved M1 (without ICA involvement), and 15 M2 MRA abnormalities (without involvement of either ICA or M1). The sensitivity and specificity of the hyperdense MCA and MCA “dot” sign are presented in table 1.

Both ASPECTS value (fig 2) and baseline NIHSS score predicted the presence of an intra- or extracranial occlusion. The relative risk of an occlusion on 3D TOF with a baseline NIHSS≥10 was 2.22 (95% CI 1.39 to 3.56). After adjusting for baseline stroke severity, for each decrement of one ASPECTS point, the odds of an occlusion involving the internal carotid artery, M1 or M2 branches rose 2.7 fold (95% CI 1.8 to 4.1). Alternately, when the baseline CT ASPECTS was less than or equal to 7, the probability of occlusion on subsequent MR angiography was 0.88 (95% CI 0.72 to 0.97) for a relative risk of occlusion of 3.27 (95% CI 2.2 to 5.0). No interaction was...
detected between ASPECTS value and baseline NIHSS score (likelihood ratio test, \( p = 0.70 \)), meaning the ability of ASPECTS to predict the MRA flow signal abnormality is not dependent on the NIHSS score.

**DISCUSSION**

Neurovascular imaging can assist in the appropriate selection of stroke patients for thrombolysis.\(^19\) Patients without occlusions receiving treatment with thrombolysis are possibly exposed to risk without obvious potential benefit. Results from the PROACT-II study suggest that, among patients with confirmed intracranial occlusion, the time window for useful intervention is longer than 3 h following the onset of symptoms.

In this cohort of consecutive patients stroke severity (median NIHSS 9) was not dissimilar to that of patients enrolled in either the ECASS II or ATLANTIS B thrombolysis clinical trials.\(^4\),\(^5\) Yet only 47\% of cases in this study had demonstrable MRA flow signal abnormalities in the MCA vascular territory. The most striking observation from this study was that ASPECTS predicts MRA flow signal abnormality independently of the NIHSS score. As the ASPECTS value decreased the probability of an MRA flow signal abnormality increased such that all cases with an ASPECTS of 5 and below had an MRA flow abnormality. When the CT scan was normal (ASPECTS 10) the prevalence of MRA flow abnormality in the anterior circulation was small (11\%). Both ASPECTS and NIHSS were independent predictors of occlusion, and therefore, may be important clinical indices in predicting vascular occlusion. The relationship between NIHSS and MRA flow signal abnormalities has been previously reported.\(^20\)

![Figure 1](A) A baseline CT scan performed on a 64 year old male patient who presented with acute dysphasia and right sided weakness, 2 h into the symptoms. A hyperdense MCA “dot” sign (arrow) suggests a thromboembolic occlusion of the M2 branch artery. There are also early CT ischaemic changes in the posterior insula and posterior temporal lobe. Incidentally there is an old right PCA stroke. [B] MRA confirming a flow signal abnormality suggesting either occlusion or slow flow in the M2 branch. The patient received intravenous tPA, but unfortunately was functionally dependent at 3 months.

The HMCA sign has been associated with severe neurological deficit, extensive infarction in the MCA territory, and poor outcome\(^1\) and correlates well with MCA occlusion based on angiography.\(^12\),\(^13\) The recently described MCA “dot” sign\(^11\) correlates with distal MCA occlusion identified by

<table>
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<th>Sensitivity and specificity of the hyperdense middle cerebral artery (HMCA) and MCA “dot” sign using 3D TOF MRA as the gold standard</th>
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Two patients had both M1 and M2 flow signal abnormalities.
conventional angiography. We now report the sensitivity and specificity of these signs using 3D TOF MRA as the gold standard. In accordance with previous angiographic studies,21 the sensitivity of both these signs is in the poor to moderate range, but both have very high specificity, confirming that when present they have a high positive predictive value for thromboembolic occlusion.

MRA provides anatomical and physiological evidence of flow through the major collateral pathways, and allowed non invasive correlation of the CT vascular signs. Conventional angiography remains the gold standard for assessing the collateral pathways and is consistent with anatomical studies.26 But the risk of inducing stroke in this particular patient group may be similar to or higher than the complication rate in the Asymptomatic Carotid Surgery Trial of 1%.24 Time of flight angiography allows the arteries of the circle of Willis to be examined by the endogenous contrast of flow-related spin enhancement and a flow related abnormality can indicate either an absence of flow or slow flow. The rationale for the use of MRA as the gold standard was its safety (cf formal cerebral angiography), and avoidance of radiation and contrast bolus as with CTA. In addition its accuracy is probably superior to other techniques such as transcranial Doppler.27 In a study comparing TOF MR angiography of carotid stenosis, flow void artefacts represent severe carotid stenosis when compared to formal angiography.28 The major drawback of this method is the depiction of the distal intracranial vessels which may account for some of the under-reporting of distal MCA branch abnormalities in this study.

Previous data support the concept that stroke patients can be selected on the basis of stroke severity and quantified CT ischaemia.4 6–8 28 There is mounting evidence that the extent of ischaemia is very important in determining response to thrombolysis. This concept would require further validation before it impacts patient care but a systematic approach to the assessment of non contrast CT with ASPECTS may provide a useful surrogate in future thrombolysis trials.

New CT techniques such as CT angiography, CT perfusion, and post contrast and blood pool analysis are being increasingly evaluated in clinical practice: the latter has been shown to increase the visualisation of ischaemic areas by 40%.29 However, the functional outcome following tPA administration is time dependent,30 and additional contrast enhanced CT techniques, as with MRI, may delay initiation of treatment with no additional benefit.

ACKNOWLEDGEMENTS
The authors thank Kathryn Werdal and Andrea Cole-Haskaynes for administrative aid in organising films for review.

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This study was supported by the Alberta Foundation for Health Research. Dr Barber was supported by the Canadian Institute of Health Research, the Heart and Stroke Foundation of Canada, and the Alberta Heritage Foundation for Medical Research. Dr Hill was supported by the Heart and Stroke Foundation of Alberta, NWT, Nunavut and the Canadian Institutes for Health Research. Dr Demchuk was supported by the Alberta Heritage Foundation for Medical Research and the Canadian Institute of Health Research. Dr Fryayne was supported by Heart and Stroke Foundation of Canada and the Alberta Heritage Foundation for Medical Research. Dr Buchan was supported by the Heart and Stroke Foundation of Canada, and the Alberta Heritage Foundation for Medical Research.

Competing interests: none declared

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J Neurol Neurosurg Psychiatry 2004 75: 1426-1430
doi: 10.1136/jnnp.2003.029389

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Possible transcallosal seizure induction by paired pulse transcranial magnetic stimulation in a patient with frontal lobe epilepsy

Seizure induction by high frequency transcranial magnetic stimulation (TMS) has been reported in normal subjects and by single pulse TMS close to the epileptic focus in patients with epilepsy.

Case report
We report an 18 year old patient with right frontal lobe epilepsy due to paramedian focal cortical dysplasia (FCD). The patient's usual seizure semiology consisted of a somatosensory aura of the left hand followed by a tonic seizure of the left arm which evolved to a bilateral asymmetrical tonic seizure without loss of consciousness. In the two years preceding the study (see below) he had rare night-time seizures only. His antiepileptic medication consisted of levetiracetam 500 mg, phenobarbital 25 mg, and carbamazepine 1600 mg daily.

Presurgical videoelectroencephalogram (video-EEG) monitoring, interictal EEG showed right frontotemporal spikes. Ictal EEG revealed seizure patterns with a right frontal onset. Magnetic resonance imaging (MRI) showed FCD in the right superior frontal gyrus extending into the right precentral gyrus (fig 1A). Neurological examination was normal.

Transcranial magnetic stimulation
The patient participated in a TMS study using a protocol described previously to evaluate intracortical excitability of both motor cortices (M1). The study was approved by the local ethics committee, and the patient gave written informed consent.

We used a focal 70 mm figure of eight coil connected to two magnetic stimulators via a BiStim module (Magstim Company, Dyfed, UK). Surface electromyography (EMG) was recorded from the contralateral abductor digitii minimi muscle (ADM) of the hand.

TMS commenced over the left M1 contralaterally to the epileptic focus with the coil placed over the M1 hand area. First, motor thresholds (RMT, AMT) and cortical induced silent period at an intensity of 110% RMT were evaluated. Next, paired pulse TMS (conditioning stimulus set at 38% of maximum stimulator output; second stimulus 60% of stimulator output) was started on the left M1 with a train of paired pulses with ISI 2, 3, 10, and 15 ms in a random order.

After 65 stimuli, the patient noticed that his habitual somatosensory aura of the left hand followed by myoclonic jerks of the left forearm (mainly biceps brachii muscle and forearm flexor muscles) was triggered by either stimulus, contralateral to the epileptogenic zone but ipsilateral to the cortical stimulation. The jerks were triggered by both single and paired stimuli at all ISI and rapidly involved both arms. These motor phenomena were different from the typical seizure semiology. EMG recordings of the ADM showed movement artefacts 63–75 ms after the MEP (fig 1B). The TMS was immediately interrupted, which aborted the myoclonus at once.

The TMS data of the left hemisphere showed increased motor thresholds, prolonged cortical induced silent period, markedly decreased intracortical inhibition, and increased facilitation compared with 20 controls (percentiles of the patient's measures within the control group: >99% for ISI 2 and >99% for motor thresholds and ISI 15, >90% for ISI 10, and >85% for ISI 3).

After the TMS experiment, the patient was again free of daytime seizures until the last follow up visit six months later.

Transcallosal seizure induction by paired pulse TMS
In patients with epilepsy, all reported cases of seizure induction by TMS have occurred during ipsilateral stimulation and near to the epileptic focus. Therefore, it has been assumed that direct stimulation of the epileptogenic tissue was required to trigger a seizure. We used a focal coil placed over the left M1 hand area more than 5 cm away from the midline. Thus, it is unlikely that the right epileptogenic zone or other cortical areas of the present patient was stimulated directly, and we assume that an indirect transcallosal activation of the epileptogenic zone provoked the aura. The latency of 65–75 ms of the myoclonic jerks after the MEP may reflect a polysynaptic pathway in addition to a direct transcallosal connection of both M1. It is still not clear whether involvement of additional cortical areas such as the ipsilesional and contralateral sensory cortices or basal ganglia contributed to the seizure provocation. Despite the patient's statement that the jerks were not volitional, this cannot be completely ruled out. The preceding somatosensory aura, however, represented a typical seizure semiology. We hypothesise that transcallosal activation of the epileptic focus was promoted by the increased excitability of M1, which was due to the underlying FCD. This, in turn, led to the aura and ictal changes in M1 excitability facilitating TMS driven myoclonic jerking. It has been previously reported that FCD is intrinsically epileptogenic and promotes reflex seizures. There is a possibility that ipsilateral pathways of movement activation could underlie our observations. In a child with extensive cortical dysplasia, TMS of the unaffected hemisphere evoked MEPs in both ADM muscles implying bilateral corticospinal connections from one cortex. Histological studies on severe brain damage in early development have revealed collateral sprouting into denuerated areas of cortex or spinal cord. Ipsilateral activation under maximum muscle contraction has been observed in healthy volunteers and in patients with acute stroke. Our patient, however, presumably had congenital but circumscribed FCD, no motor deficits, and was investigated at rest. This and the fact that his habitual somatosensory aura occurred before the myoclonic jerks strongly argue against the activation of ipsilateral corticospinal tracts. Activation of a silent mirror focus in the left hemisphere with subsequent spread to the right is also unlikely because exclusively right sided ictal and interictal epileptiform discharges were recorded during the video-EEG monitoring.

Changes in motor cortex excitability
Our patient's higher motor thresholds compared with controls are very likely due to his ion channel blocking anticonvulsant medication.
The loss of intracortical inhibition and increased intracortical facilitation in the left hemisphere contralateral to the epileptic zone may reflect synaptic reorganization of the ipsilesional and contralesional motor cortices. These distant functional cortical changes associated with malformations of cortical development have also been described previously.6 The prolongation of the cortical induced silent period seen in the present patient may be independent of the phenobarbital intake and confirms similar findings from previous studies as a remote effect of FCD on the motor cortex in untreated patients with cortical dysgenesis.7

Discussion

Hashimoto’s encephalopathy (HE) is a steroid responsive disorder characterised by high titres of anti-thyroid antibodies. The original description of this condition was in an established case of Hashimoto’s thyroiditis where the patient developed focal neurological deficits and coma.1 Clinical presentation includes episodic confusion, myoclonus, seizures, and stroke-like episodes.2 Females are more affected than males (3:6:1), with a mean age of onset of 41 years. The hallmark of HE is its response to steroids, improving within a few hours to days.1 The titres of anti-thyroid antibodies may independently influence the severity of the clinical presentation.2 Fewer than 100 cases of HE have been reported in the literature. Generalized and hypothroidism can be associated with the disorder, but the majority of patients are euthyroid. Although steroid responsiveness is the rule, additional immunosuppressive therapy, in the form of azathioprine and cyclophosphamide has been tried in a minority of cases.8 The immunopathological basis of this syndrome has been compared to a relapsing form of acute disseminated encephalomyelitis.9 Although reversible MRI findings have been described in HE,2 neuroradiographic changes (except for isolated patchy uptake on isotope scans) is usually normal in most cases.3 Cerebral angiography has been found to be normal in several cases of HE, unlike in many other cerebral vasculitides.4-5 Thyroid autoantibodies can co-exist with several other forms of autoimmune encephalomyelitis, but the normal MRI scan, the initial dramatic response to steroids, and negative autoantibodies for most other common vasculitides, tends to favour the diagnosis of HE in our case. Steroid responsive encephalopathy associated with Hashimoto’s thyroiditis is an alternative proposed name for this condition,6 but the majority of cases have normal thyroid function, leaving “Hashimoto’s encephalopathy” a universally accepted term. A recent literature review of 85 patients with encephalopathy associated with anti-thyroid antibodies suggests that the combination of encephalopathy, high serum anti-thyroid antibody concentrations, and...
responsiveness to glucocorticoid therapy seems unlikely to be due to chance. The initial meningoencephalitic type presentation of our patient in 1987 was probably the first manifestation of HE in view of clinical findings and laboratory data (Mild CSF pleocytosis is not unusual in HE). There was a delay of 14 years before the diagnosis was first established, in spite of several hospital admissions. The initial relapses after diagnosis responded well to steroids, confirming the diagnosis of HE. Whether the current episode was precipitated by the sudden withdrawal of oral steroids or the chest infection itself, for which they were prescribed, is unclear.

Our patient illustrates the possibility of steroid resistance in an established case of HE and the need to consider further immunomodulatory therapy. Intravenous immunoglobulins are a safe, convenient, and effective treatment in such circumstances.

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doi: 10.1136/jnnp.2004.049395

Competing interests: none declared

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Spontaneous lobar haemorrhage in CADASIL

CADASIL is an autosomal dominant form of arteriopathy, primarily affecting cerebral vessels, and predominantly caused by point mutations in the Notch3 gene on the short arm of chromosome 19. Affected individuals develop subcortical strokes and cognitive deficits in their 50s and 60s. Brain magnetic resonance imaging (MRI) shows large areas of leukoencephalopathy and multiple subcortical lacunar infarcts. Small arteries and capillaries are characterised histologically by a non-atherosclerotic, non-amyloid angiopathy with accumulation of granular osmiophilic material (GOM) within the smooth muscle cell basement membranes and extracellular matrix. While CADASIL is considered a primarily ischaemic form of vascular dementia, microhaemorrhages have recently been reported in 31% of symptomatic Notch3 mutation carriers, suggesting that structural fragility of the arterial walls may lead to leakage of haem products. Lobar haemorrhage in the absence of other risk factors for haemorrhage has previously been reported in one patient with CADASIL. Here we report a second case.

Case report

A 56 year old man who had been diagnosed with multiple sclerosis six years earlier was admitted to the hospital with an acute change in mental state. He had collapsed at home and was unresponsive when rescue arrived. In the emergency room he had a depressed level of consciousness and difficulty following commands, with paucity of speech, dysarthria, and hypophonia. There was no evidence of head trauma. His blood pressure was 100/63 mm Hg and his temperature was 36.1°C.

Past medical history included chronic obstructive pulmonary disease, prostate resection for prostate cancer, and a history of nicotine and alcohol dependence. He had no history of hypertension, diabetes mellitus, or coagulopathy. His drug treatment included ipratropium, ranitidine, methylprednisolone, and albuterol. His mother, now deceased, had been diagnosed as having multiple sclerosis and had migraines with aura, stroke-like symptoms, and dementia. He had eight siblings, three with headaches and one with recent transient ischaemic events.

Computed tomography (CT) of the head in the emergency department showed an area of high attenuation in the right frontal lobe consistent with acute haemorrhage (fig 1A). There was no evidence of trauma on head CT. Gradient echo MRI sequences of the brain done on hospital day 2 showed a 2×2.5 cm area of haemorrhage in

Figure 1 (A) Non-contrast computed tomography of the head done in the emergency room showing an area of high attenuation in the right frontal lobe consistent with acute haemorrhage. The other panels show non-contrast magnetic resonance imaging done on hospital day 2. (B) Gradient echo sequence demonstrating a 2×2.5 cm area of haemorrhage in right frontal lobe, a microhaemorrhage in the right parietal region, and extensive white matter disease. (C, D) The area of haemorrhage is hypointense on T2 and isointense on T1 weighted imaging, consistent with an acute haemorrhage.
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 iso-
tense on T1 weighted sequences (fig 1D), consistent with acute haemorrhage. There was no MRI evidence of a cavernous haem-
angioma, 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 cali-
nised 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. Notch3 gene test-
ning 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, Sourour and Wallinder reported a 29 year old man with hereditary multi-infarct dementia on anticoaguulants, with a large haemorrhage in the right hemi-
sphere.1 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.2 In 1992, Baadrumont et al reported a case of massive left cerebral haematoma involving the cau-
date 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.3 The index patient in this report had no
evidence of coagulopathy and no history of previous hyperextension, cerebral haemor-
rhage, or anticoagulant therapy. The patient could have experienced a haemorrhagic con-
tusion related to a closed head injury during his unvouched 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 haem-
angioma, 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 microhaemor-
rhages.4 The first Notch3 transgenic mouse shows early widening of the subependymal and intra-smooth-muscle spaces in the vascu-
lar smooth muscle cells, denoting weakening of the arterial wall and increasing susceptibility to micro- and macrohaemor-
rhages.5 This case report supports the growing evidence for both ischaemia and haemorrhage in a variety of small artery diseases includ-
ing amyloid angiopathy and CADASIL. Clinicians may consider the possibility of haemorrhage when evaluating new events and deciding on treatment for stroke preven-
tion in patients with CADASIL.

Acknowledgements
Supported by grants P20 RR015578 and K08 MH001487.

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doi: 10.1136/jnnp.2004.042564

Competing interests: none declared

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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.3 9 Lesion pathology, however, varies from acute stroke to a neoplasm, and there is not necessarily a small, distinct lesion.4 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 mellis-
ti, or 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 dysfunc-
tion. 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 he was brought to the hospital. Neurological examination in the emergency room found no weakness, and he was sent home. At that time the patient was alert, and his cranial nerves were intact. Limb muscle strength was normal. Sensory examination was normal, except for some proprioceptive deficit in the left hand. 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 urinary tract, and there was no evidence of a tumour 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 with strain, having a peak flow rate of 5.0 mls 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 posterior aspect 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 stenosis of the intracranial vessels. An elec-
trocardiogram was normal. Transthoracic echocardiograms showed no abnormal find-
ings. The urinary catheter was withdrawn 3 days after admission, and this was followed by 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,5 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.7 The locations of the primary cerebral cortical centres for voiding and storage are speculated to be separate, the former being at the para-central lobule.7 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.7 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

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void was bilaterally in a different portion of the mid-cingulate gyrus. 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. 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 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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doi: 10.1136/jnnp.2004.050542

Competing interests: none declared

References

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.)

BOOK REVIEW

Neuropsychiatry and behavioural neurology explained


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 stilled 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 aetiology for chorea versus athetosis, but some would be sceptical of the value in splitting choreathetosis. 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


doi: 10.1136/jnnp.2003.029389corr1

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).