

Small cerebellar strokes may mimic labyrinthine lesions

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SUMMARY Thirty nine cases of cerebellar infarct and haemorrhage were seen over a period of 3 years. Of these, 69% had no impairment of consciousness. Six cases had nystagmus and gait ataxia as their only abnormal signs. Small cerebellar strokes may present with only vertigo, unsteady gait, and unidirectional nystagmus, thus mimicking labyrinthine lesions. Care in managing vertigo attacks in patients with risk factors for cerebrovascular disease appears to be warranted.

Until the advent of computed tomography, cerebellar infarction and haemorrhage were thought to be rare, and were seldom diagnosed in life.^{1,2} Although now better recognised, recent reviews³ nevertheless continue to emphasise the more severe cases. We review our experience in the last 3 years, both to document that cerebellar haemorrhage and infarcts are not rare and also to emphasise that small cerebellar infarction and haemorrhage may mimic more benign lesions.

Cases

Nineteen patients (11 males, eight females age 43-80 years) with cerebellar infarction, and 20 patients (eight males, 12 females age 45-73 years) with cerebellar haemorrhage were seen in the University Medical Unit, Queen Mary Hospital from January 1981 to December 1983. All diagnoses were confirmed on CT scan, 48.6% of which was performed within the first 3 days, and 86.6% within the first 9 days. Fifteen of the infarct cases, and 12 of the haemorrhage cases had no disturbance of consciousness during the course of their illness. This group of 27 patients had fairly similar features of vertigo, nystagmus, nausea and vomiting, unsteady gait and limb ataxia. Headache was infrequent, as also were limb weakness and cranial nerve abnormality (table 1). The vertigo was spontaneous and lasted for more than 5 minutes at onset in all cases,

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although brief vertigo of seconds duration precipitated by head movements, occurred later in three patients. Nystagmus was unidirectional in six cases of infarct, and three cases of haemorrhage. This was in the same direction as the direction of fall in two cases, and opposite in three cases. In the other four cases the patients had no consistent direction of falling. The direction of nystagmus did not lateralise the lesion well, corresponding in four of the unidirectional cases and not corresponding in five cases. Six cases had nystagmus and gait ataxia as their only abnormal signs (table 2). However, careful examination revealed history of dysarthria in one, and presence of upgaze nystagmus in another. Most of the patients in this group resumed normal walking within a few days.

Twelve other cases had disturbance of consciousness. Of these 12 cases, three cerebellar haemor-

Table 1 *Symptoms and signs of benign cerebellar stroke*

	<i>Infarct</i>	<i>Haemorrhage</i>
<i>Patient number</i>	15	12
<i>Symptoms</i>		
Abnormal mental state	0	0
Headache	1	3
Vertigo	8	9
Unsteadiness	12	11
Dysarthria	3	2
nausea/vomiting	9	10
<i>Signs</i>		
Gait ataxia	15	12
Limb ataxia	8	9
Nystagmus	8	5
Limb weakness	1	2
Hearing disturbance/tinnitus	2	0
Ocular movement abnormality	3	3
Other cranial nerve abnormality	0	3
Sensory loss	1	0

Table 2 *Minimal feature cerebellar stroke*

	Sex Age (yr)	Clinical feature	CT finding
1	M/60	Vertigo, nausea, gait ataxia, left beating nystagmus, transient left hearing defect	Right cerebellar infarct
2	M/56	Vertigo, nausea, gait ataxia, nystagmus to right	Left cerebellar infarct
3	M/59	Vertigo, nausea, gait ataxia	Right cerebellar infarct
4	M/65	Transient dysarthria and vomiting, gait ataxia	Right cerebellar infarct
5	F/55	Nausea, vomiting, vertigo, nystagmus on left gaze and upgaze, gait ataxia	Left cerebellar infarct
6	F/72	Vertigo, right beating nystagmus, gait ataxia	Right cerebellar haemorrhage

rhage cases required operation, one refused surgery and died suddenly on the 17th day. Of the infarction cases, one died before surgery could be performed and one died with brainstem infarction. The others did not need surgery.

Among the entire group of cerebellar stroke, hypertension was present in 10 of the infarcts, diabetes mellitus in four. Fourteen of the haemorrhages had hypertension, and two had diabetes. Previous stroke or TIA was not recorded in any ischaemic cases, but one of the haemorrhage cases had had a previous lacunar infarct and another had had an episode of spontaneous vertigo 2 years previously.

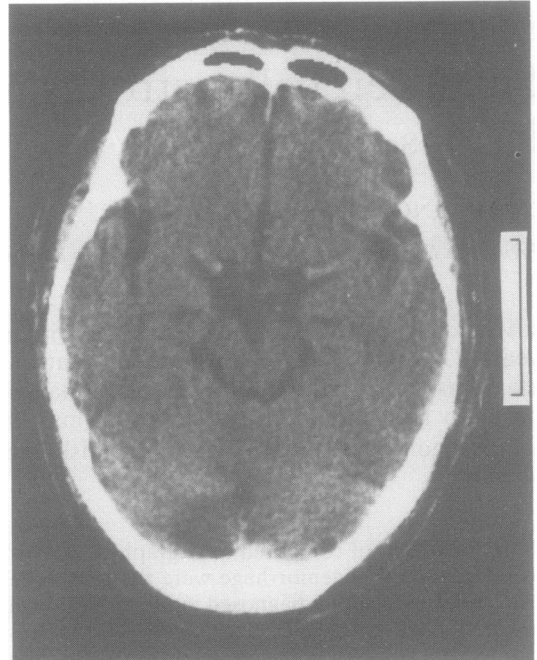
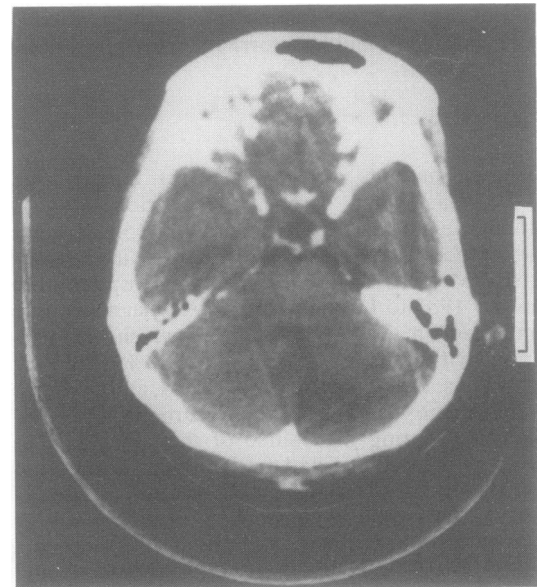
Illustrative cases

Case 1

A 60-year-old male Chinese was known to be hypertensive but not treated for 2 years. He suddenly developed on the day of admission, severe vertigo, unsteadiness, nausea and vomiting. The vertigo lasted for one hour and was accompanied by transient left hearing difficulties but not tinnitus. Afterwards, head movement would precipitate brief vertigo. There was no headache or other symptoms. On admission, he was mentally alert, with a blood pressure of 160/100 mm Hg. He had nystagmus with fast component to the right on right gaze. There was no dysarthria or limb ataxia. However, he was unable to walk except with a wide-based gait. The rest of his neurological examination was normal. CT scan demonstrated an area of decreased attenuation in the right cerebellar hemisphere (fig 1). His condition improved and he was able to walk normally in a few days.

Case 5

A 55-year-old Chinese female had been treated for diabetes mellitus for 7 years. She suddenly developed spontaneous vertigo, and vomiting on the day of admission. Subsequently, vertigo occurred when she turned to the left. On admission she had vertical nystagmus on upgaze which disappeared after a day, as well as nystagmus with fast component to the left on left gaze. There was no limb ataxia. She tended to fall to the right when she tried to

Fig 1 *Small infarct in the right cerebellar hemisphere.*Fig 2 *Small infarct in the left cerebellar hemisphere.*

walk. No other abnormality was detected. CT scan showed a left cerebellar hemisphere infarct (fig 2). The gait became normal after a few weeks.

Discussion

Descriptions of symptoms and signs in cerebellar infarction and haemorrhage in earlier necropsy series emphasised gross and dramatic deficits. Sybert and Alvoid¹ noted that 64% of their patients with cerebellar infarction had abnormal mental state, 47% ocular movement abnormality, 42% motor disturbance, and 57% had other cranial nerve abnormality. Ott *et al*² described headache, nausea, vomiting, unsteadiness in most of the patients with cerebellar haemorrhage, and only 14 of their 56 patients were alert. Seventy three per cent of the patients had limb ataxia, ipsilateral gaze palsy and peripheral facial palsy. However, later studies using computed tomography detected less dramatic presentations. Scotti *et al*⁴ described 21 cases of cerebellar infarction of which 15 had a benign course. Mental abnormality was detected at admission in only six of the cases. Ocular movement disorder was seen in six of 21 cases, facial palsy in eight, and limb weakness in eight. Ho *et al*⁵ described another seven cases of which one needed surgery. The current series documents that cerebellar infarction and haemorrhages are not rare and many present with unsteadiness, vertigo, nausea, and vomiting, with little disturbance of consciousness, cranial nerve palsy, or limb weakness. Signs of brainstem abnormality such as vertical nystagmus and diplopia may be transient and subtle. The gait ataxia can easily be blamed on labyrinthine disturbance alone. The features therefore easily mimic those of labyrinthine disturbance. This possibility of misdiagnosing cerebellar lesions for more benign peripheral disorders has previously been noted by Duncan *et al*,⁶ who described three cases of acute cerebellar infarction in the PICA territory where the patients had only positional related dizziness, nausea, vomiting and unsteadiness. The nystagmus was unidirectional in two cases. They thought that a nystagmus in the opposite direction to the falling would favour labyrinthine lesion. In our cases, however, reliance on the relationship of the direction of fall to that of the nystagmus would have led to erroneous diagnosis in three out of five cases.

Postmortem studies have shown that cerebellar infarction may not be accompanied by brainstem strokes in about 70% of cases.¹ We have noted earlier that cases of spontaneous non-positional related vertigo, who develop subsequent cerebrovascular disease, were more likely to have vertebrobasilar territory strokes than carotid territory strokes.⁷ Others have noted that there is increased risk of cerebrovascular disease associated with the less specific complaint of dizziness.⁸ The current series suggests that one possible explanation is that pure

cerebellar transient ischaemic attacks, as well as small infarcts may have no features other than vertigo, unidirectional horizontal nystagmus and unsteadiness, and therefore be misdiagnosed as benign labyrinthine lesions. Since vertigo alone is more likely to be due to labyrinthine disease, current practice rightly does not consider transient vertigo and unsteadiness *per se* as diagnostic of vertebrobasilar TIA. Nevertheless, cases of vertigo occurring in patients with risk factors for cerebrovascular disease should perhaps be investigated for this possibility more often than currently practised, especially when the vertigo is not positional related at onset, and unaccompanied by tinnitus and hearing disturbance.

Ott *et al* commented that 52% of cerebellar haemorrhage patients who were awake for 2 days later deteriorated.² In our cases about a fifth of the infarction cases did show progressive stepwise deterioration. Whether early initiation of anticoagulant therapy could prevent this deterioration is uncertain. However, Scotti *et al*⁴ had earlier concluded that no symptoms or signs differentiated cerebellar infarct or haemorrhage with certainty, except perhaps headache. Our experience would concur with this. Even among the six patients who presented with the minimal features of vertigo, nausea, nystagmus and ataxic gait, there was one case of cerebellar haemorrhage. Significant headache was so uncommon with small cerebellar haemorrhages that this symptom also becomes unhelpful. Computed tomography should therefore be used before any anticoagulation is initiated.

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