Cerebellar Infarction: Natural History, Prognosis, and Pathology

Richard A.L. Macdonell, MBBS, Renate M. Kalnins, MBBS, BMedSci, FRCPA, and Geoffrey A. Donnan, MD, FRACP

Using clinical and computed tomography (CT) criteria, an analysis of 2,000 consecutive stroke unit patients from 1977 to 1984 revealed 30 patients with cerebellar infarction. The case fatality rate was 23%, higher than for any other location of brain infarction studied over the same period. Death was most often due to concomitant brainstem infarction. Obstructive hydrocephalus occurred in 4 patients (13%), and in 2 cases diagnosis, facilitated by urgent CT scanning, allowed early surgical intervention that was life saving. Patients who survived the acute phase were followed for an average of 21 months, and over that time 22% sustained further brainstem infarction, representing a 13% stroke rate per year. Over the latter 3 years of the clinical study, an autopsy survey revealed 11 cases of cerebellar infarction that had been clinically unrecognized. None of these died as a direct result of their infarction. Mechanisms of infarction inferred from autopsy included in situ thrombosis, embolism, watershed, and lacunar infarction, with in situ thrombosis being the most common. We conclude that the case fatality rate of cerebellar infarction is greater than of any other form of brain infarction, but it may be reduced by prompt recognition of those patients who will benefit from surgical decompression.

In survivors, a high risk of subsequent hindbrain stroke exists. More attention needs to be paid to this entity in terms of early diagnosis and prevention of subsequent stroke. (Stroke 1987;18:849–855)

Subjects and Methods

Data from the Austin Hospital Stroke Unit (Melbourne, Australia) from 1977 to 1984 were reviewed to establish the frequency of presentation of cerebellar infarction to a secondary and tertiary referral center. The Austin Hospital is such a center as 1 of 5 University teaching hospitals in Melbourne, Australia (population approximately 3.5 million). Over the study period, there were 1,997 referrals to the unit, of which 295 (14.8%) were cerebrovascular events related to the posterior circulation.

Thirty patients were diagnosed on clinical grounds as suffering from acute cerebellar infarction. Twenty-two were confirmed by CT scan and an additional 2, who were not scanned, were confirmed by postmortem examination. Of the remaining 6 patients, 2 were not scanned, and 4 had normal CT scans performed 0–11 (mean 4) days after the onset of symptoms, which were not repeated. None of these 6 patients were autopsied.

Follow-up of the 23 survivors was by outpatient review (18 patients) or by discussion with their local physician (5 patients). All histories and CT scans were reviewed by the authors to confirm the original diagnoses.

During the study period, case fatality rates for cerebellar hemorrhagic, lacunar, and brainstem infarction were compared with that of cerebellar infarction. During the latter 20 months of the clinical study, a concurrent analysis of 500 consecutive autopsies was undertaken to establish the prevalence of cerebellar infarction in selected postmortem examinations and to suggest pathophysiologic mechanisms of infarction.

Results

Clinical Features

The series of 30 patients with cerebellar infarction were of mean age 66 (range 45–80) years; 10 were women and 20 men. The presenting symptoms are shown in Table 1. Dizziness and/or vertigo was by far the most common symptom. Of the 24 patients (80%) with this symptom, 9 described true rotational vertigo. Thirteen patients (43%) had symptoms in which brainstem structures could be implicated. Three patients had visual symptoms suggesting coincident ischemia of the...
Symptom Table 1. Symptoms and Signs of Cerebellar Infarction in 30 Patients at Presentation

<table>
<thead>
<tr>
<th>Symptom</th>
<th>No. patients</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dizziness and/or vertigo</td>
<td>24</td>
<td>80</td>
</tr>
<tr>
<td>Truncal ataxia</td>
<td>23</td>
<td>77</td>
</tr>
<tr>
<td>Nausea and vomiting</td>
<td>19</td>
<td>63</td>
</tr>
<tr>
<td>Dysarthria</td>
<td>18</td>
<td>60</td>
</tr>
<tr>
<td>Headache</td>
<td>12</td>
<td>40</td>
</tr>
<tr>
<td>Diplopia</td>
<td>5</td>
<td>17</td>
</tr>
<tr>
<td>Limb weakness</td>
<td>4</td>
<td>13</td>
</tr>
<tr>
<td>Facial anesthesia</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>Visual disturbance</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>Dysphagia</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Limb anesthesia</td>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>

Sign
Cerebellar dysfunction
  Lateralized | 21 | 70 |
  Truncal     | 20 | 67 |
  Nystagmus   | 16 | 53 |
  Small pupils| 10 | 33 |
  Ocular movement disorder | 8 | 27 |
  Abnormal gag         | 8 | 27 |
  Drowsy            | 7 | 23 |
  Facial weakness   | 4 | 13 |
  Facial hypoesthesia| 4 | 13 |
  Unconscious       | 4 | 13 |
  Hemiplegia/monoplegia | 2 | 7 |
  Diplopia          | 2 | 7 |
  Tongue deviation on protrusion| 1 | 3 |
  Hemi/mono paresthesia | 1 | 3 |

posterior cerebral artery territory. Headache was present in 40% of the cases. The delay from the onset of symptoms until admission varied from 0 (during an angiogram) to 6 days. Twenty-two patients were seen within 24 hours.

Signs at presentation are also shown in Table 1. In 8 cases, no cerebellar signs could be elicited, thus making clinical diagnosis of cerebellar infarction difficult; 4 of these patients were unconscious. Urgent CT scanning provided the diagnosis in 2 of these 4 patients, demonstrating hydrocephalus. The other 2 cases did not have a CT scan because CT was not available when these patients were admitted in 1978. In these 2 cases, the diagnosis was based on the preceding history and was later confirmed by postmortem examination. Cerebellar infarction was confirmed by CT in 2 of the other 4 cases without lateralizing cerebellar signs.

Seven patients were noted to be drowsy on admission. CT scanning did not show brainstem compression or hydrocephalus in any of these patients.

Localizing signs in the form of ipsilateral ataxia were found in 21 patients (70%). In 10 patients (33%), there was a history of progression and worsening of symptoms from the time of the original insult. A relatively lucid period of 24–36 hours before decline of conscious state was seen in all cases of brainstem compression and hydrocephalus. When brainstem infarction was the cause of loss of consciousness, the interval varied between 0 (immediately) and 3 days after the onset of symptoms.

Ten patients (33%) had a history of transient symptoms related to ischemia in the vertebrobasilar territory. These symptoms occurred from 10 weeks to 1 day (mean 39 days) prior to the definitive cerebellar infarct.

The prevalence of major risk factors for cerebrovascular disease is shown in Table 2. In addition, 15 patients had histories of cardiac disease (14 ischemic, 1 valvular), and 7 patients were assessed to be in atrial fibrillation on clinical grounds at presentation. Eight patients had carotid bruits, and 3 had subclavian bruits.

Radiologic Findings
CT scans. The first scan was performed within 24 hours of symptoms in 11 patients; cerebellar infarction was confirmed in 6. Three of the 5 patients with normal CT scans were scanned again using i.v. contrast 7–10 days later; in each case cerebellar infarction was confirmed.

Fourteen patients were scanned 1–10 (mean 5) days after onset of symptoms and 1 patient at 20 days; cerebellar infarction was confirmed in 13 of the 15.

Size of the infarcts varied. One involved a small area of the vermis alone, presumably due to infarction in the distal superior cerebellar artery territory (Figure 1); some almost totally occupied one hemisphere on a CT slice. The 4 patients who developed hydrocephalus presented 24–72 hours after the onset of symptoms, and CT showed infarction of most of one cerebellar hemisphere with shift and compression of the fourth ventricle (Figure 2). In 14 cases, infarction was clearly unilateral and in 5 cases bilateral; 1 case showed multiple areas of infarction (Figure 3), which was later confirmed histologically (Figure 4).

Arteriograms. Six studies of the vertebrobasilar arteries were performed. In all except 1 case, the arteriogram demonstrated significant abnormalities. These

Table 2. Cerebrovascular Disease Risk Factors for Cerebellar Infarcts

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>No. patients</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>17</td>
<td>57</td>
</tr>
<tr>
<td>Diabetes</td>
<td>5</td>
<td>17</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 20/day</td>
<td>8</td>
<td>27</td>
</tr>
<tr>
<td>&lt; 20/day</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>Nonsmoker</td>
<td>9</td>
<td>30</td>
</tr>
<tr>
<td>Ex-smoker</td>
<td>4</td>
<td>13</td>
</tr>
<tr>
<td>Unknown</td>
<td>7</td>
<td>23</td>
</tr>
</tbody>
</table>

Hypertension and diabetes were considered risk factors if patients were taking regular treatment for these disorders at admission.
abnormalities included 1 case of subclavian stenosis proximal to the vertebral artery, 2 cases of vertebral origin stenosis on the side ipsilateral to the infarct, 1 case with irregularities and narrowing of the vertebral artery, and 1 case of vertebral artery ectasia. In no case was occlusion of either the vertebral, posterior inferior cerebellar (PICA), or basilar artery demonstrated.

Other Investigations

EEG. Fifteen patients had an electroencephalogram (EEG); 11 had a normal EEG, and 1 had evidence of slowing in the theta and delta range diffusely at the time of cerebellar infarction. The other 3 patients had nonspecific focal slow wave abnormalities.

Cardiac. Twenty-seven patients had an electrocardiogram (ECG) within 24 hours of admission; 15 patients (56%) demonstrated abnormalities including old myocardial infarction (6 patients), recent infarction (1 patient), and atrial fibrillation (3 patients). Three patients had a 24-hour cardiac Holter monitor, and all 3 demonstrated abnormalities (ventricular ectopics, ventricular tachycardia, and sporadic atrial fibrillation). Four cardiac echocardiograms were performed, and none of these demonstrated a source of embolus.

Figure 1. A small infarct in the left cerebellar vermis.

Figure 2. Infarction of the left cerebellar hemisphere causing compression of the fourth ventricle.
Prognosis

Short term. In the hospital, 17 patients (57%) deteriorated due to complications related to cerebellar infarction. These consisted of 4 patients who developed brainstem compression and hydrocephalus due to swelling of their infarct, and 13 who developed features suggestive of brainstem infarction or ischemia.

In 3 of the 4 cases complicated by hydrocephalus, the posterior fossa was decompressed. Two of these 3 patients survived, whereas the other died, not having regained consciousness. The fourth patient with hydrocephalus had a cardiorespiratory arrest while in the CT scanner and could not be resuscitated.

Five additional patients died. In 2 cases, the CT scan showed cerebellar infarction without hydrocephalus, and the cause of death was thought to be brainstem infarction, which was confirmed by postmortem examination in 1 case. Two patients died without having a CT scan. At postmortem examination, both were found to have sustained brainstem infarction, and there was no evidence of hydrocephalus. There was 1 incidental death due to an acute myocardial infarction in a patient recovering from cerebellar infarction. The case fatality rate, therefore, was 23%. A parallel study of other forms of cerebral infarction over the same period showed that this rate was higher than for any other subgroup of cerebral infarction (Table 3).

Nine patients developed brainstem signs such as poor gag and swallow without CT evidence of brainstem compression. This was believed to represent brainstem infarction due to extension of thrombus.

The outcome for all patients at time of hospital discharge is shown in Table 4. The 2 patients who were decompressed and survived were able to return home using walking aids.

Long term. All 23 surviving patients were followed for periods varying from 1 to 84 (mean 21) months. During this period, 5 patients (22%) suffered a hindbrain ischemic stroke, and 2 of the 5 died. Over the follow-up period, this represents a 13% per year stroke rate. Two of the remaining 3 patients with later brainstem stroke were severely disabled, requiring nasogastric feeding and in 1 case permanent tracheostomy. Hindbrain ischemic events of a transient nature were reported in an additional 4 patients.

Recurrent symptoms of hindbrain ischemia occurred
at times varying from 1 day to 23 months (mean 5.3 months) after the original insult. Five patients had single events, whereas 4 had multiple attacks. Six patients were treated with long-term prophylactic aspirin following their cerebellar infarct; none of these 6 had recurrent events during the follow-up. One patient who had an episode of verteobasilar ischemia subsequent to the cerebellar infarct was begun on aspirin and had no further events.

Neuropathology

Cases of cerebellar infarction reviewed pathologically included those from the clinical series of 1977–1984 (5 cases) together with all cases found among routine autopsies from late 1982 to 1985. The latter group comprised 16 cases in a total of 500 consecutive autopsies, a rate of 3.2%. The 2 groups combined gave a total of 19 cases examined. Histologic evidence of infarction and, where applicable, of vascular thrombosis was obtained in each case. Only 8 (42%) of the 19 cerebellar infarcts examined had been clinically evident. The distribution of the infarcts is listed in Table 5 and the underlying vascular pathology for most cases in Table 6.

Acute cerebellar infarction. From the clinical series, autopsies were performed on 4 of the 7 patients who died during the acute phase following symptomatic cerebellar infarction. In 3 patients, death was due to concomitant brainstem infarction; the fourth had an acute myocardial infarction and died following a cardiac arrest. Two of the 3 patients who died as a result of their stroke had sustained infarction of the posterior inferior portion of one cerebellar hemisphere (Figure 5) plus brainstem infarction as a result of acute thrombosis of the PICA. The third patient sustained bilateral superior cerebellar and brainstem infarction due to basilar artery atheroma and superimposed thrombus; only in this latter case was there evidence of hydrocephalus. In no case was the primary infarct hemorrhagic. One case with PICA infarction showed focal hemorrhagic softening on the superior surface of the cerebellum consistent with an area of herniation.

In addition, 4 acute cerebellar infarcts (hours to days old) that had not been clinically recognized were found among the unselected autopsy group, a rate of 0.8% in the 500 autopsies studied. In all 4 cases, the patient had been extremely ill due to other conditions, and in no instance was there significant local verteobasilar disease. One of these 4 patients had an intravascular coagulopathy, and another had a mural thrombus in the heart as a likely embolic source. Death in all 4 cases was due to the antecedent condition rather than cerebellar infarction. The location of infarction in this group was not the common PICA site. In 2 cases, multiple small infarcts in both hemispheres were present; an additional case had patchily hemorrhagic infarction of the superior portion of one cerebellar hemisphere, whereas in the final case the infarct, although unilateral, was thought to be of the watershed type.

Old cerebellar infarction. Four clinically evident cases were examined at autopsy some years after their original presentation; 2 had unilateral PICA infarcts, whereas 2 had unilateral superior infarctions. Seven old infarcts were found incidentally among the autopsy series (rate 1.4%). Of these 7, only 2 were single infarcts in the PICA location; 2 were in the superior portions of the hemispheres, 1 case had multiple random infarcts, 1 was a watershed infarct, and the final case was a lacunar infarct.

The lacunar infarct was 8 mm in maximal dimension and was associated with local small vessel thickening.

Table 3. Case Fatality Rates (1977–1984) for All Forms of Cerebral Infarction

<table>
<thead>
<tr>
<th>Location of infarction</th>
<th>Fatality rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral hemisphere</td>
<td>12.5%</td>
</tr>
<tr>
<td>Lacunar</td>
<td>1.2%</td>
</tr>
<tr>
<td>Brainstem</td>
<td>17%</td>
</tr>
<tr>
<td>Cerebellar</td>
<td>23%</td>
</tr>
</tbody>
</table>

Table 4. Early Outcome Following Cerebellar Infarction in 30 Patients

<table>
<thead>
<tr>
<th>Outcome</th>
<th>No. patients</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>No residual signs</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>Mild ataxia</td>
<td>10</td>
<td>33</td>
</tr>
<tr>
<td>Requiring walking aids</td>
<td>6</td>
<td>20</td>
</tr>
<tr>
<td>Full nursing care</td>
<td>5</td>
<td>17</td>
</tr>
<tr>
<td>Death</td>
<td>7</td>
<td>23</td>
</tr>
</tbody>
</table>

Table 5. Distribution of Pathologically Confirmed Cerebellar Infarction in 19 Cases

<table>
<thead>
<tr>
<th>Vascular territory involved</th>
<th>No. cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posterior inferior cerebellar artery (PICA)</td>
<td>7</td>
</tr>
<tr>
<td>Superior cerebellar artery</td>
<td>7</td>
</tr>
<tr>
<td>Multiple random sites</td>
<td>3</td>
</tr>
<tr>
<td>White matter lacune</td>
<td>1</td>
</tr>
<tr>
<td>Bilateral watershed (PICA, superior cerebellar territory)</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 6. Inferred Pathogenesis of Infarcts Confined to Territory of the PICA or Superior Cerebellar Arteries in 14 Cases

<table>
<thead>
<tr>
<th>Location of infarct</th>
<th>Inferred pathogenesis</th>
<th>No. cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posterior inferior cerebellum</td>
<td>Ipsilateral vertebral artery atheroma and thrombosis</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Bilateral vertebral and PICA atheroma and thrombosis</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Severe ipsilateral PICA atheroma</td>
<td>1</td>
</tr>
<tr>
<td>Superior cerebellum</td>
<td>Basilar artery atheroma and thrombosis</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Embolism from heart</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Uncertain</td>
<td>2</td>
</tr>
</tbody>
</table>

PICA, posterior inferior cerebellar artery.

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of the hypertensive type. The case of watershed infarction showed bilateral narrow zones of infarction occurring at the boundaries between the territories supplied by the superior cerebellar artery and the PICA. Local vascular atheroma was mild, and no embolic source was identified at autopsy. The patient had suffered from hypotensive complications occurring perioperatively some weeks previously.

Discussion

The diagnosis of cerebellar infarction had a poor prognosis in the pre-CT scan era, and from postmortem studies it was clear that the diagnosis was often overlooked.\(^2,7\) The introduction of CT has made it possible to rapidly distinguish cerebellar infarction from hemorrhage, to detect surgically reversible brainstem compression and hydrocephalus, and to identify small cerebellar infarcts.\(^10,11,15-17\) As a result, there has been a fall in the mortality due to this condition, from 50–80% before CT to 20% in our series. This is principally due to the ability of CT to detect small infaracts with a benign course which previously were not recognized.\(^18\) To a lesser degree, earlier recognition of patients who will benefit from surgery has also improved prognosis.\(^7,9-11,14\)

Three groups of patients can be recognized from their clinical course following cerebellar infarction. The first group includes those whose conscious state deteriorates within the first few hours, usually due to extension of the ischemic process to infarct the brainstem. The second group comprises those whose conscious state deteriorates 24–36 hours after the onset of symptoms, usually because of brainstem compression by the expanding cerebellum, although brainstem infarction may also occur at this stage due to propagation of thrombus. The final group includes those whose conscious state remains unimpaired throughout.\(^9\)

When cerebellar infarction is suspected, patients should have an immediate CT scan, repeated urgently if there is any deterioration in conscious state. The first scan may be normal, but hemorrhage is quickly excluded; this is not possible on clinical grounds alone.\(^19-22\) If conscious state is impaired or deteriorates, CT will help identify the cause. Brainstem infarction is associated with normal CT scan appearances, whereas brainstem compression is shown by loss of the fourth ventricle and hydrocephalus even if the infarct itself is not seen. Concomitant brainstem and cerebellar infarction is associated with a high case fatality rate (31% in our series), and there is no effective treatment. Brainstem compression, if detected early through a combination of clinical suspicion and CT scanning, may be reversed surgically. In our series, 67% of such patients who were operated on survived compared with a reported mortality rate of >80% in those managed conservatively.\(^5,7,24-27\) Posterior fossa decompression was the form of surgery in all our patients. Ventricular drainage alone has been advocated by some,\(^7,24-25\) but because it may provoke upward herniation of the cerebellum and does not relieve pressure on the brainstem, we do not advocate its use alone.\(^14,26\)

Infarction without impairment of conscious state needs to be distinguished from a benign labyrinthine disturbance or small cerebellar hemorrhage. Clinically, each may present as acute dizziness and unsteadiness of gait. Distinguishing a cerebellar lesion is difficult if lateralizing cerebellar signs are not present.\(^13,27\) The initial CT scan will exclude a small hemorrhage, but in the other two conditions CT appears typically normal. Repeating the CT scan 7–10 days later with i.v. contrast will improve the rate of detection of cerebellar infarction.\(^28\) Magnetic resonance imaging may further improve the rate of detection of cerebellar infarcts and at an earlier stage than CT.\(^18\)

Patients with clinical features suggestive of cerebellar infarction should be treated as a high risk group in the acute phase. The case fatality rate for our patients (23%) was higher than for any other infarct subgroup over the same period. Death was most commonly due to extension of the ischemic process to cause brainstem infarction (57%).

Warning symptoms of vertebrobasilar ischemia occurred in 33% of our patients prior to infarction. After recovery from infarction, 39% of surviving patients had further symptoms of ischemia in the vertebrobasilar territory; 22% of these patients sustained brainstem infarction, representing a 13% stroke rate per year.

The autopsy series demonstrated that a number of cerebellar infarcts may still escape clinical detection. It must be said that none of the autopsied patients was assessed by a neurologist, and the cause of death in all cases was not related to cerebellar infarction. These infarcts fell into 2 distinct groups. Infarcts occurring shortly before death were due to proximal sources of emboli or generalized systemic disturbances such as coagulopathy. In contrast, old infarcts were most commonly associated with local vessel atheroma and thrombosis as the presumed cause of infarction, al-
though other mechanisms were represented (watershed and hypertensive small vessel disease).39-32

The full spectrum of cerebellar infarction as diagnosed clinically, imaged on CT, and examined pathologically has been reviewed. Cerebellar infarction has a significant morbidity and mortality in the early phase. Surgical decompression may be life saving if the complications of brainstem compression and hydrocephalus are recognized early, using a combination of clinical and CT findings.

Given the high rate of subsequent hindbrain ischemic events, further studies are needed to test the efficacy of various treatment options, such as anticoagulants, when the heart is the suspected embolic source, or antiplatelet agents following cerebellar infarction.

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