Massive Cerebellar Infarction: “Conservative” Management

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SUMMARY Eleven patients with large cerebellar infarctions were admitted recently to our service. Eight of them showed evidence of hydrocephalus on the CT scan examination. Five were treated with controlled external ventricular drainage and six were managed conservatively. One death, most likely due to progressive brainstem infarction, occurred. The outcome was favorable in the other patients. It is suggested that prompt treatment of the acute obstructive hydrocephalus may obviate the need for posterior fossa decompression in patients with massive cerebellar infarction.

MASSIVE CEREBELLAR INFARCTION is the result of an occlusion of the vertebrobasilar trunk or one of its branches and is associated with high mortality and disability rates. Most of the authors recommend urgent suboccipital craniectomy and resection of the swollen and/or hemorrhagic cerebellar tissue as the treatment of choice. However, this procedure is not free of serious complications, since it has to be performed in elderly people with compromised circulatory systems which comprise the vast majority of the patients with cerebellar infarction.

The rapid or progressive deterioration with coma and death can be due to 1) associated extensive brainstem infarction; 2) direct compression of the brainstem by the swollen cerebellar tissue in association with tonsilar or upward transtentorial herniation; or 3) total or partial obliteration of the 4th ventricle with displacement and/or kinking of the aqueduct resulting in obstructive hydrocephalus. The above factors of course may occur in combination or isolated.

During the recent years we have encountered eleven patients with large cerebellar infarctions. Five of them were treated with controlled external ventricular drainage (CEVD). The other six received conservative, medical management. The clinical course and outcome of these eleven patients is reported and the suggestion is made that early use of CEVD may eliminate the need for a suboccipital craniectomy in most of the patients with massive cerebellar infarction.

Patients

During the period from December 1979 to March 1982 eleven patients with massive cerebellar infarctions were admitted to our unit. The criterion for inclusion in this study was a cerebellar infarction large enough to cause at least partial obliteration of the IV ventricle on the CT scan. Five patients were treated with CEVD and six improved with conservative management. Dexamethasone was given to 2 patients of the first and 5 patients of the second group. Ten patients made a satisfactory recovery and returned to independent daily living activities. One patient progressively deteriorated despite CEVD and reduction of cerebellar swelling and hydrocephalus as evidenced by repeated CT scanning. Clinically, this patient developed signs of brain stem infarction and died 3 days after admission. The clinical course of each patient is summarized in table 1. Four illustrative cases are discussed in detail.

Illustrative Cases

Case 1

This 81 year old man presented with dizziness of sudden onset followed by persistent vomiting. On admission he was fully conscious and alert, well oriented and his speech was normal. He complained of some difficulty in swallowing. He had unsurpassed horizontal nystagmus on lateral gaze in either direction. Over the next 24 hours he deteriorated, first becoming drowsy and disoriented with course horizontal nystagmus on lateral gaze and ataxic movements in his right limbs. His speech became feeble and markedly dysarthric. He then required noxious stimuli to be roused. His right pupil became slightly smaller than the left and he developed a right 6th nerve palsy. The CT scan showed a low density mass lesion in the right cerebellar hemisphere compressing the 4th ventricle with dilation of the lateral ventricles (fig. 1). He was given 4 mg dexamethasone intravenously which was repeated every 6 hours over the next 48 hours followed by gradual reduction in dosage. In addition 500 cc of 20% mannitol was infused over a 24 hour period. His condition improved and within 12 hours he became alert. During the next few days his pupils became equal, extraocular movements were full and his nystagmus disappeared. He was up and discharged within 2 weeks after admission. Neurological examination 6 months after admission revealed mild dysmetria of his right arm.

Case 2

This 56 year old man suddenly collapsed at home three days prior to admission. He did not lose consciousness but was dizzy and vomited repeatedly. He was admitted to the local hospital for three days but gradually became drowsy and confused for which he was transferred to our unit. His blood pressure was 210/100 mm Hg. He was drowsy but could be aroused with repeated verbal stimuli. His speech was dysar-
thric and he was disoriented. About 6 hours after admission the drowsiness increased and his speech became soft and extremely slurred. Horizontal nystagmus was noted on lateral gaze to either side as well as vertical nystagmus on upward gaze. His breathing then became intermittently ataxic though he remained responsive with tactile stimuli. A CT scan showed a large right cerebellar infarction with obliteration of the 4th ventricle and moderate dilatation of the 3rd and lateral ventricles (fig. 2). This patient was given 4 mg dexamethasone intravenously every 6 hours and monitored closely over the next 24 hours. His condition remained unchanged for about 6 hours and then there was progressive improvement. Within 12 hours he was alert and well oriented. His speech was still slurred but the nystagmus had disappeared. He was up and around within a few days and was discharged 11 days after admission when the neurological examination revealed mild dysmetria of his right arm. A repeat CT scan during the recovery period showed some resolution of

### TABLE 1  Clinical Course of 11 Patients with Cerebellar Infarctions

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Presenting symptoms</th>
<th>Presenting signs</th>
<th>CT findings</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (S.P.)</td>
<td>71, F</td>
<td></td>
<td>vertigo, dysarthria</td>
<td>impairment of (L)</td>
<td>large (L) cerebellar</td>
<td>steroids</td>
<td>normal</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>ataxia, vomiting</td>
<td>conjugate gaze,</td>
<td>hemisphere hypodense lesion</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>for one day</td>
<td>left sided dysmetria</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 (M.S.)</td>
<td>66, F</td>
<td></td>
<td>unsteadiness for 2</td>
<td>bilateral nystagmus</td>
<td>large (R) cerebellar</td>
<td>CEVD for 3 days</td>
<td>gradual deterioration and death 3 days after admission despite decrease of the mass effect on the CT scan</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>months, headaches,</td>
<td>tremor, drowsiness</td>
<td>infarction with mass effect and mild hydrocephalus</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>vertigo, vomiting</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>for one day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 (G.R.)</td>
<td>45, F</td>
<td></td>
<td>ataxia, nausea for 3</td>
<td>stupor, mild neck</td>
<td>large (L) cerebellar</td>
<td>steroids</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>days, vomiting,</td>
<td>stiffness, nystagmus, impaired (L)</td>
<td>infarction obstruction of 4th ventricle and hydrocephalus</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>headache, drowsiness for one day</td>
<td>conjugate gaze</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 (E.L.)</td>
<td>72, F</td>
<td></td>
<td>vertigo, nausea for 2</td>
<td>nystagmus, incoordination of (R) limbs</td>
<td>large (R) cerebellar</td>
<td>steroids</td>
<td>slight unsteadiness and gait ataxia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>days</td>
<td></td>
<td>infarct with mass effect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 (A.W.)</td>
<td>49, M</td>
<td></td>
<td>headache, vomiting, vertigo, photophobia for one day</td>
<td>nystagmus on (R) lateral gaze</td>
<td>hemorrhagic (R) cerebellar infarction with mass effect and moderate hydrocephalus</td>
<td>steroids CEVD</td>
<td>completely independent mild (L) hemiparesis</td>
</tr>
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<tr>
<td>6 (L.S.)</td>
<td>61, M</td>
<td></td>
<td>slurred speech, vom­</td>
<td>drowsy, paresis of (R) lateral gaze, (R) dysmetria, impaired sensation of (L) side of face</td>
<td>large (R) cerebellar infarct with mass effect and moderate hydrocephalus</td>
<td>steroids</td>
<td>slightly slurred speech, some unsteadiness</td>
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<td></td>
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<td>iting, obtundation after cardiac catheterization</td>
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<tr>
<td>7 (W.B.)</td>
<td>57, M</td>
<td></td>
<td>unsteady gait,</td>
<td>(L) arm dysmetria, dysarthria, (L) Horner’s syndrome, (R) horizontal nystagmus, weak gag reflex</td>
<td>large (L) cerebellar infarction</td>
<td>observation</td>
<td>slight dysarthria and dysmetria (L) arm independent</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>slurred speech,</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>headache, drowsiness for 3 days</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 (S.T.)</td>
<td>81, M</td>
<td></td>
<td>dizziness, vomiting</td>
<td>unsustained horizontal nystagmus, ataxia of (R) limbs</td>
<td>large (R) cerebellar infarct with hydrocephalus</td>
<td>steroids Mannitol</td>
<td>mild dysmetria of (R) arm</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>and dysphagia for 1 day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 (O.S.)</td>
<td>56, M</td>
<td></td>
<td>dizziness, vomiting and confusion for 3 days</td>
<td>drowsy, disorientation and dysarthric</td>
<td>large (R) cerebellar infarct and hydrocephalus</td>
<td>steroids</td>
<td>mild dysmetria of (R) arm</td>
</tr>
<tr>
<td>10 (H.N.)</td>
<td>72, M</td>
<td></td>
<td>headache, nausea, vomiting and unsteadiness for 24 hours</td>
<td>drowsy, bilateral horizontal and vertical nystagmus, incoordination of (L) limbs</td>
<td>(L) cerebellar infarct with hydrocephalus</td>
<td>CEVD for 4 days</td>
<td>minimal dysmetria of (L) arm</td>
</tr>
<tr>
<td>11 (D.T.)</td>
<td>76, F</td>
<td></td>
<td>headache, vomiting and dizziness for 12 hours</td>
<td>deeply comatose, small and minimally reactive pupils, depressed corneal reflexes, absent ice-water caloric response</td>
<td>large (R) cerebellar infarct with hydrocephalus</td>
<td>CEVD for 5 days</td>
<td>normal</td>
</tr>
</tbody>
</table>
CEREBELLAR INFARCTION/Khan et al

Figure 1 (A and B). CT scan showing low density mass lesion in right cerebellar hemisphere compressing the 4th ventricle with dilatation of lateral ventricles.

Figure 2. CT scan showing large right cerebellar infarction with obliteration of the 4th ventricle and dilatation of 3rd and lateral ventricles.

Figure 3. CT scan showing partial resolution of cerebellar swelling with a patent 4th ventricle and reduction in hydrocephalus.

Case 3

This 72 year old man was admitted to hospital because of suboccipital headache, nausea, vomiting and unsteadiness for approximately 24 hours duration. He was drowsy but his speech was normal. He had horizontal nystagmus on lateral gaze in either direction and vertical nystagmus with upward gaze. Minimal incoordination was noted in his left arm and leg. Tendon reflexes were normal and plantar responses were flexor. CT scan showed a left cerebellar infarction. Over the next 24 hours the patient’s condition deteriorated. He became comatose with occasional decerebrate movements to painful stimuli. Pupils were small and reacted feebly to light. His eyes were fixed in conjugate deviation to the right and his corneal reflexes were depressed bilaterally. There was moderate symmetrical hyperreflexia (fig. 4). An emergency twist drill hole was made in the right frontal area and the right lateral ventricle was tapped with external ventricle drainage against a differential pressure of 25 cm H₂O. He became conscious within an hour and was able to
obey simple commands. During the next 24 to 48 hours he became alert, well oriented and his speech returned to normal. Cranial nerve function returned to normal but he had minimal incoordination on his left side. External ventricular drainage was subsequently readjusted to a differential pressure of 15 to 20 cm H$_2$O and removed 4 days later. A third CT scan demonstrated less swelling of the left cerebellar hemisphere with a patent 4th ventricle and some resolution of the hydrocephalus (fig. 5). The patient was discharged a few weeks later and is back to his previous activities. Follow-up examination 1 year later revealed minimal dysmetria of the left arm.

Case 4

This previously healthy 76 year old woman developed suboccipital headache, nausea, vomiting and dizziness approximately 12 hours prior to admission to her local hospital. She continued to deteriorate over the next 24 hours and was subsequently transferred to our unit when she was noted to be comatose and not responding to painful stimuli. The pupils were small and reacted minimally to light. The corneal reflexes were depressed and ice water caloric responses were absent. She had bilateral symmetrical hyperreflexia with extensor plantar responses. A CT scan showed a large right cerebellar infarction associated with moderate...
obstructive hydrocephalus (fig. 6). Shortly after this investigation, her respirations became irregular and she had periodic spontaneous decerebrate movements. Emergency right frontal twist drill hole and external ventricular drainage were established and maintained against a differential pressure of 20 cm H$_2$O. Within 30 minutes after this procedure she became fully conscious and could obey simple commands. Her respirations returned to normal. Twenty-four hours later she had mild dysmetria of the right arm and leg. The external ventricular drainage was subsequently readjusted to a differential pressure of 15 to 20 cm H$_2$O and discontinued 5 days later. A repeat CT scan revealed less cerebellar swelling with some resolution of the hydrocephalus and a patent 4th ventricle (fig. 7). This patient was discharged from hospital, returned to her routine and was without neurological deficit when re-assessed 6 months later.

Discussion

In 1938, Germain and Morvan, $^5$ first described the clinical course of a 32 year old man who died from a right cerebellar infarction. Two further papers appeared in 1956, one by Fairburn and Oliver, $^6$ and the other by Lindgern, $^7$ reporting a total of 5 cases of cerebellar infarction, each presenting with the clinical manifestations of a rapidly expanding posterior fossa mass lesion. In following years, a number of clinical reports have been published. $^1$, $^8$-$^{25}$ Most authors recommend immediate surgical decompression with removal of the swollen infarcted cerebellar tissue or when there is progressive decrease in the state of consciousness and increasing signs of brain stem compression in spite of medical therapy.

The real incidence of cerebellar infarction with brain stem dysfunction is not known. In 1975, Sypert and Alvord$^{28}$ reported an incidence of acute cerebellar infarctions of 1.1% in an adult autopsy population of 5,495 cases. About half of the cases were complicated by other life threatening central nervous system lesions, mostly acute massive brain stem infarctions.

The clinical features of cerebellar infarction cover a wide spectrum mimicking symptoms and signs from an acute labyrinthitis to a rapidly expanding posterior fossa mass lesion with brain stem and cerebral dysfunction. Attempts to classify the patterns of the clinical picture were made recently. $^{26}$-$^{27}$ Obviously the crucial feature, determining the type of treatment and the timing of surgery, is alteration of the mental state and the level of consciousness.

The mortality for patients with progressive brain dysfunction approaches the level of 80% if the condition is left untreated. $^1$ The current treatment of choice, posterior fossa craniectomy with resection of the infarcted tissue carries a high mortality, 28%, in elderly, patients with artherosclerotic vascular disease. $^1$ External ventricular drainage has been recommended by Geraud et al. $^{18}$ (Case 3), and Greenberg et al. $^{29}$ (Cases 1 and 2) and is supported by the good results of our cases. The only death in our series occurred despite reduction of the cerebellar swelling on the CT scan, presumably because of further brain stem infarction. Autopsy was not allowed by the family.

Factors favouring this approach are: 1) external ventricular drainage, although not risk-free, is a much less hazardous procedure compared to the direct removal of the infarcted tissue. It can be performed within a few minutes on the ward without general anesthesia; 2) the most important cause of the clinical deterioration appears to be obliteration and displacement of the 4th ventricle and/or the aqueduct resulting in acute hydrocephalus rather than pressure effect on the brain stem by the swollen cerebellar hemisphere. In the study by
Sypert and Arvold\textsuperscript{28} histological evidence of brain stem damage by compression was lacking, and 3) the natural history of an infarction is one of reduction in size after an initial phase of swelling. According to Feigin and Budzilovich\textsuperscript{29} in early stages infarcted tissues are swollen occupying a volume greater than that of the same tissues prior to undergoing necrosis. If the infarct is large, this swelling may cause narrowing of an adjacent ventricle. The swelling begins to recede, perhaps 1 to 2 weeks after onset and later, perhaps 3 to 4 weeks, the infarct comes to occupy a volume less than that of the original tissues. According to McCall and Fletcher,\textsuperscript{31} the infarct ceases to increase in size after four to five days. If the draining system compensates for the hydrocephalus over several days, spontaneous recovery of the patency of the CSF pathways will follow with resolution of the hydrocephalus. Since there are no microscopical distinguishing features between cerebral and cerebellar infarction it is safe to accept these assumptions on the temporal profile of the cerebellar infarction, which are also in agreement with descriptions of the time of maximal swelling following sudden, massive encephalomalacia.\textsuperscript{32}

Apparently an important reason for urgent decompression was the difficulty in differentiating between cerebellar infarction and hemorrhage in the pre-CT scan era. The latter is a much more aggressive lesion. In the autopsy series of Sypert and Alvord 95\% of the cerebellar hemorrhages found were acute resulting directly in a fatal outcome, compared to 54\% of the detected cerebellar infarctions. A good quality CT scan will demonstrate all cerebellar hemorrhages of clinical significance. Cerebellar infarction on the CT scan will appear either as a low density area or iso-dense when the scan is performed early or the infarction is becoming hemorrhagic. Large hematoma can occur with an infarction which might be impossible to distinguish from frank hemorrhage.\textsuperscript{33} Practically this should be treated as a primary hemorrhage.

Richardson\textsuperscript{33} has emphasized the potential danger of upward transtentorial brain stem herniation following external ventricular drainage for cerebellar hemorrhage and possibly for massive cerebellar infarction. However, this danger is minimized when external ventricular drainage is controlled with proper adjustment of the differential height between the system and the lateral ventricle and closely monitored by repeated clinical and CT examinations.

The CT scan is now the definitive method for establishing the diagnosis of cerebellar infarction and the presence or absence of hydrocephalus. Shenkin and Zavala\textsuperscript{34} reported on a series of 55 patients with cerebellar infarcts diagnosed clinically and by CT scanning. Forty-nine patients did not have hydrocephalus of whom 44 (90\%) survived. Six patients developed hydrocephalus and became progressively obtunded. Each of these patients was treated by ventricular drainage and survived.

Eight of our 11 patients developed obstructive hydrocephalus. CEVD was established in five of these patients because of progressive neurological deteriora-

Progressive neurological deterioration in patients with cerebellar infarctions and associated hydrocephalus is very likely related to increasing intracranial pressure in the absence of brain stem infarction. The dramatic and rapid recovery after CEVD would support this hypothesis. In contrast, patients who do not develop hydrocephalus with large cerebellar infarctions fair well without ventricular drainage.\textsuperscript{34} In our patients, neurological deterioration continued unless the hydrocephalus became arrested or was treated by CEVD. Recently, Sayama et al.\textsuperscript{35} reported 12 cases of cerebellar infarction and used the presence of hydrocephalus as an important factor in deciding about posterior fossa decompression. Thus, it would appear that one of the most important determining factors in survival of patients with cerebellar infarction is whether hydrocephalus develops.

Dexamethasone was used in 7 of the 11 patients presented. Since the initial report of the use of cortisone in the acute treatment of "apoplectic stroke" by Russek, Zohman and Russek,\textsuperscript{35} numerous reports have followed on the use of steroids in cerebral infarction, although conclusions as to their benefit are often diametrically opposed.\textsuperscript{36-38} More specifically, there is no statistically significant data defining the use of steroids in acute cerebellar infarction. While brain swelling from ischemic edema is seldom life threatening in the supratentorial compartment, massive cerebellar infarction and swelling may be critical in the presence of acute obstructive hydrocephalus. It is not inconceivable that, in some cases, steroids may reduce cerebellar swelling, albeit minimal, but sufficient to prevent the development of acute obstructive hydrocephalus.

In conclusion, it is our opinion that external ventricular drainage should be tried first and that posterior fossa craniectomy should be reserved only for the cases in which the former procedure is proven ineffective.

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CEREBELLAR INFARCTION/Khan et al

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