Cerebellar Hemorrhage and Infarction

Roberto C. Heros, M.D.

IN LARGE AUTOPSY SERIES at Boston City Hospital, Bellevue, and the University of Washington Hospital, the incidence of cerebellar hemorrhage was 0.27%, 0.44%, and 0.7%, respectively. Cerebellar infarcts uncomplicated by brain stem infarction are about two-thirds as common as cerebellar hemorrhages. In several clinical series of spontaneous intracerebral hemorrhages, the cerebellum was the site of the hemorrhage in approximately 10% of the cases. Both cerebellar hemorrhage and infarct occur most frequently in the fifth through the eighth decades and about as frequently in males as in females.

Etiology

Some two-thirds of all spontaneous cerebellar hemorrhages occur in hypertensive patients. Arteriovenous malformations, blood dyscrasias, trauma, neoplasms, anticoagulants, and aneurysms account for the rest. In younger patients arteriovenous malformations, trauma, and blood dyscrasias are the usual causes of cerebellar hemorrhages.

Emboli are implicated in about 50% of cerebellar infarcts. The source of the emboli is most frequently the heart, and as expected, there is an increased incidence of ischemic heart disease and atrial fibrillation in these patients. About 40% of patients with cerebellar infarction are hypertensive.

In a recent review of the subject, we found several cases of cerebellar infarct due to traumatic vertebral artery occlusion. Chiropractic manipulation of the neck was the most common causative factor.

Pathology

Cerebellar hemorrhages seem to occur most frequently in the area of the dentate nucleus. From this site they can spread to involve most of a cerebellar hemisphere and occasionally cross the midline to involve the other side. Not infrequently, they extend into the cerebellar peduncles, and they commonly rupture into the fourth ventricle. Only rarely do they directly involve the brain stem, but the stem is frequently deformed by pressure from the clot on the cerebellum and fourth ventricle.

About 85% of symptomatic cerebellar infarcts occur in the territory of the posterior inferior cerebellar artery (PICA), in the inferior, medial portion of the hemisphere, they are grossly hemorrhagic about 25% of the time. Occlusion of the intracranial portion of the vertebral artery or bilateral vertebral occlusion is found in about 50% of the cases, and occlusion of the PICA is found in about 30%.

Clinical Features and Pathophysiology

Although McKissock had emphasized some of the important features of the syndrome of cerebellar hemorrhage in his 1960 series, he felt that the clinical picture was too protean and the neurological signs were not specific enough to establish a clinical diagnosis without laboratory and radiological investigations. In 1965 Fisher and coworkers were the first to insist that the diagnosis of cerebellar hemorrhage should be made strictly on the basis of clinical criteria without resorting to time-consuming radiological investigations. The subsequent experience of this group as well as that of others confirms this opinion.

This clinical picture of cerebellar infarction has also been studied in detail and will be reviewed together with the syndrome of cerebellar hemorrhage.

The clinical features of early, intermediate, and late stages are summarized in table 1. Of course, patients may be seen only at a very late stage or may quickly lapse into a moribund state without showing an orderly progression of symptoms.

Early Stage

Initially, the symptoms produced by both cerebellar hemorrhages and infarcts are related to destruction and compression of the cerebellum itself or to the subarachnoid hemorrhage resulting from rupture of the hematoma into the subarachnoid space. Thus, it is not surprising that dizziness, nausea, vomiting, headache, lack of balance, and difficulty walking are the most common presenting symptoms. Although the onset is abrupt in most patients with cerebellar hemorrhage, it may be more gradual, occurring over a period of several hours or even days. Many patients with cerebellar infarction run a subacute course, and some of these patients have premonitory signs or transient ischemic attacks (TIA) before the onset of their stroke.

When examined at this stage, patients are alert or irritable and sometimes confused. The most striking sign is truncal ataxia, which might be expected since both processes involve the deeper portions of the cerebellum including the vermis. It may be said that if the patient can stand and walk normally, he does not have a significant cerebellar hemorrhage or infarct. Frequently, the truncal ataxia is so severe that the patient cannot even sit to be examined. This finding alone in an alert patient complaining of headache and dizziness of sudden onset should strongly suggest the diagnosis of cerebellar hemorrhage or infarct. Nystagmus, usually horizontal and rapid in the direction of gaze, is another common finding at this stage and, together with the dizziness and occasional vertigo and nausea, suggests involvement of the flocculonodular complex and the vestibular connections of the cerebellum. Apparentataxiaisreflects involvement of the more lateral portions of the cerebellum and is frequently found at this stage, although its absence should never preclude a diagnosis. The same applies to some degree of ataxic speech or dysarthria.

Intermediate Stage

As the mass effect increases, hydrocephalus results from compression of the fourth ventricle or rupture of the...
TABLE 1  Clinical Features of Cerebellar Hemorrhage and Infarct

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Signs</th>
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<td>Truncal ataxia</td>
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<td>Nyctagmus</td>
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<td>Nausea</td>
<td>Appendicular ataxia</td>
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<td>Vomiting</td>
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<td>B. Intermediate Stage</td>
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<tr>
<td>Irritability</td>
<td>Pseudo-VI palsy</td>
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<td>Confusion</td>
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<td>Drowsiness</td>
<td>Gaze paresis</td>
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<td>Babinski signs</td>
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<td>Peripheral facial palsy</td>
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<td>Horner's syndrome</td>
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<td></td>
<td>Mild hemiparesis</td>
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<td>Small pupils reactive to bright light</td>
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<td>C. Late Stage</td>
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<tr>
<td>Stupor</td>
<td>Pinpoint pupils</td>
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<td>Coma</td>
<td>Ataxic respirations</td>
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<td>Posturing</td>
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<td>Cardiovascular instability</td>
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</table>

When a patient presents in the intermediate clinical stage with drowsiness, a sixth nerve palsy or a gaze paresis, and a peripheral facial paresis; with a history of ataxia, headache, nausea, vomiting of abrupt onset, and without a major hemiparesis, a cerebellar hemorrhage or a large cerebellar infarct is present, and no other diagnosis can be entertained. The diagnosis is more difficult when the patient presents during the early stage without signs of brain stem compression or during the late stage in deep coma. During the early stage a patient presenting with sudden onset of headache, dizziness and nausea, and a stiff neck may be thought to have a subarachnoid hemorrhage secondary to a ruptured aneurysm or arteriovenous malformation. Patients with aneurysmal hemorrhage frequently lose consciousness transiently at the onset and have a telltale initial, transient hemiparesis or monoparesis. Also, their headache is usually very severe, and photophobia is a prominent symptom. Frequently, they pace the floor, holding their head in an effort to relieve the headache. This would be impossible with a cerebellar hemorrhage because of truncal ataxia. Another frequently entertained diagnosis in a patient with cerebellar hemorrhage into the ventricle. At this stage the patient may become quite confused and agitated or drowsy, or periods of drowsiness and agitation may alternate. Early compression of the dorsal portion of the brain stem results in ipsilateral sixth nerve paresis. In the early stages this paresis of voluntary lateral gaze in one eye can be overcome with caloric stimulation, a condition that Fisher called “pseudo-VI paresis.” Later, ipsilateral gaze paresis develops from compression of the horizontal gaze centers. The eyes may be forcibly deviated to the opposite side; this paresis cannot be overcome by caloric stimulation. At this stage some degree of ipsilateral “peripheral-type” facial paresis is almost always present because of the compression of the facial colliculus. Babinski signs, first ipsilaterally and then bilaterally, are present at this stage. Sometimes a Horner’s syndrome is seen because of compression of the sympathetic pathways running from the hypothalamus through the dorsal brain stem. A very mild hemiparesis may be detected at this stage, but it is never severe until quite late. The pupils in this intermediate stage are usually small, again from interference with the descending sympathetic pathways.

Ott and his coworkers suggested that the triad of signs — appendicular ataxia, ipsilateral gaze palsy, and peripheral facial palsy — should strongly suggest the diagnosis of cerebellar hemorrhage; at least two of these signs were present in 73% of their 26 patients.

Late Stage

If the process continues unchecked, massive brain stem compression or extension of the hemorrhage into the brain stem can occur. Patients quickly pass from a stuporous condition into a comatose state, begin to posture, and eventually become decerebrate. Pinpoint pupils are usually found at this stage, but they will react to bright light because, although the descending sympathetic control from the hypothalamus to the cervical cord is lost, the parasympathetic control of

the pupils in the midbrain is preserved until late. Ataxic respirations appear and are often followed rapidly by cardiovascular instability and apnea from medullary involvement.

Lateral Medullary (Wallenberg) Syndrome

As mentioned previously, a partial or complete lateral medullary syndrome frequently complicates the clinical picture of cerebellar infarction. Indeed, it is not uncommon for patients with a typical Wallenberg’s syndrome to have some degree of infarction of the cerebellum, which is usually asymptomatic. Only when the cerebellar infarction is massive does it assume primary clinical importance with surgical implications. The signs and symptoms of infarction in the part of the medulla usually supplied by the PICA are summarized in Table 2. Fisher et al. have presented an excellent discussion of this syndrome.

Differential Diagnosis

When a patient presents in the intermediate clinical stage with drowsiness, a sixth nerve palsy or a gaze paresis, and a peripheral facial paresis; with a history of ataxia, headache, nausea, vomiting of abrupt onset, and without a major hemiparesis, a cerebellar hemorrhage or a large cerebellar infarct is present, and no other diagnosis can be entertained. The diagnosis is more difficult when the patient presents during the early stage without signs of brain stem compression or during the late stage in deep coma.

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hemorrhage or, more commonly, with cerebellar infarct is acute labyrinthitis. With labyrinthitis, patients have prominent nystagmus and, when ambulating, they tend to fall towards the affected side. Patients with cerebellar disease also tend to fall to the side of the lesion, and while the nystagmus is usually more prominent towards the affected side, but it tends to change direction with changes of gaze which does not occur in labyrinthitis.

When seen in the late stages, patients with cerebellar hemorrhage and infarction present a more difficult differential diagnosis. Intracerebral hemorrhage is often suspected in patients who experience an apoplectic event and lapse rapidly into coma. However, in thalamic and putaminal hemorrhage, hemiplegia occurs early and usually before the patient becomes comatose. With cerebellar hemorrhage isolated hemiplegia is not seen, and hemiparesis occurs late when other signs of brain stem compression are obvious. In pontine hemorrhage the patient rapidly becomes comatose and quadriplegic. Oculomotor paresis occurs before deep coma in putaminal hemorrhage and usually not at all in cerebellar hemorrhage. In thalamic hemorrhage pinpoint pupils will not react to bright light as the pupils usually do in cerebellar hemorrhage. Dysconjugate gaze with the ipsilateral eye adducted and deviated downwards is common in thalamic hemorrhage. Fisher pointed out that an intracerebral hemorrhage into the anterior portion of the frontal lobe might cause a problem in differentiation because hemiparesis may be mild or absent and the eyes may deviate to one side. However, this deviation can be overcome with caloric irrigation, which is not the case when the forced deviation is caused by brain stem compression. In addition, patients with frontal lobe hemorrhage will show frontal lobe signs such as grasping and sucking reflexes.

**Diagnostic Tests**

Until recently, a lumbar puncture was a standard early diagnostic test for patients with a history of headache of sudden onset and without a major hemiparesis or papilledema, and not surprisingly, most patients with cerebellar hemorrhages had a lumbar puncture. A few clearly deteriorated and died shortly after this test, as might be expected in patients who already had some degree of tonsillar herniation. The procedure was not definitive because a negative spinal tap did not rule out a small or medium-sized cerebellar hemorrhage or a cerebellar infarct of any size. Since the advent of the computerized tomographic (CT) scan, most clinicians feel that a spinal tap is contraindicated in cases of suspected cerebellar hemorrhage or infarct. The CT scan usually makes it unnecessary to perform a lumbar puncture, which adds little information, has few therapeutic implications, and carries some risk to the patient. Ventriculography also has no place in the modern diagnosis of cerebellar hemorrhage and infarcts since it offers no information that could not be obtained by a CT scan.

Angiography was another diagnostic test used frequently before the CT scanner appeared. Carotid angiograms may show hydrocephalus and nonspecific signs of impaired circulation in the posterior fossa. Vertebral angiography usually shows an avascular cerebellar mass and, in cases of cerebellar infarct, occasionally reveals the appropriate vascular lesion. Nowadays, if a CT scan shows a cerebellar hemorrhage, angiography is usually unnecessary, but it may still be useful in the rare patient with a normal or questionable CT scan who is not in acute danger and in whom a cerebellar infarct is strongly suspected. In such a patient a vertebral angiogram may show changes that could support the diagnosis of infarction of the cerebellum. Also, angiography is useful in patients with a cerebellar hemorrhage in whom an arteriovenous malformation or tumor is suspected because of young age, absence of hypertension, or other clinical factors.

CT scanning is the diagnostic procedure of choice in all patients suspected of having a cerebellar hemorrhage or infarct and who are not deteriorating rapidly enough to necessitate immediate surgery. A good CT scan will reveal all cerebellar hemorrhages of clinical significance. Most CT scans of cerebellar infarctions will show either a low density area or at least indirect signs of a cerebellar mass effect, such as hydrocephalus and displacement or obliteration of the fourth ventricle. It should be emphasized, however, that a cerebellar infarct may be isodense when scanned, either because it is too early to show significant changes or because the infarct, in becoming hemorrhagic, is passing through a stage of equal density to surrounding brain tissue.

**Course and Prognosis**

Before CT scanning became widely available, estimates of the mortality rate of untreated, symptomatic cerebellar hemorrhage ranged from 65 to 80%. It is becoming obvious that many small cerebellar hemorrhages detected by CT scanning result in only mild symptomatology and have a relatively benign natural history, but there is no question that once a cerebellar hemorrhage leads to stupor and signs of brain stem compression, the fatality rate approaches 100% without treatment. Several authors have commented on the unpredictable course of cerebellar hemorrhage. Some patients present in deep coma and die rapidly. Others show a gradual but relentless course over a period of several days. More importantly, some patients who are awake or only moderately drowsy and seem to be stable or to have reached a clinical "plateau" can deteriorate suddenly, becoming afebrile and soon dying. Ott and coworkers found that 50% of patients who remained awake and relatively stable for two days went on to deteriorate into coma sometime over the next several days. Only 25% of patients who remained awake for seven days deteriorated.

Although small, asymptomatic cerebellar infarcts are not rare at autopsy, the mortality rate of symptomatic, untreated, cerebellar infarct has been estimated at about 50%. Once signs of brain stem compression appear, the mortality rate approaches 80%. The clinical course of symptomatic cerebellar infarction is more predictable than that of cerebellar hemorrhage and, in general, evolves at a slower rate. In fatal cases death usually occurs between the third and sixth days, but once obtundation or stupor develops, death usually occurs in 6 to 30 hours in untreated cases.
Treatment and Results

Balance in 1906 first reported the successful removal of a cerebellar hemorrhage. Several other cases were reported during the first half of the century. In 1960 McKissock reported 34 cases of cerebellar hemorrhage treated surgically. Nine of these patients were treated by ventricular tapping or drainage; all nine died. In some of these patients, there was sudden deterioration after ventricular decompression which was attributed to upward herniation. Of the 14 patients treated by suboccipital craniectomy with resection of the hematoma, nine survived. The quality of survival of these patients was good; most had normal intellectual capacity and exhibited only some ataxia and occasional, mild brain stem signs. McKissock's study and a subsequent study by Richardson pointed out the danger of using ventricular drainage alone as the primary treatment for cerebellar hemorrhage. Excision of the hematoma by craniotomy was established as the treatment of choice. The report by Fisher and colleagues suggested that acute cerebellar hemorrhage is a lethal condition which should be treated surgically with the same urgency as subdural and extradural hematomas. In that classic paper they recommended that once the diagnosis is made, which should be done strictly on clinical grounds, craniectomy and evacuation of the hematoma should be carried out immediately in all patients except those who are alert, show flexor plantar responses, and are stable clinically.

Ott and colleagues reviewed a series of 50 patients from the Massachusetts General Hospital. Two-thirds were conscious on admission. The diagnosis was made exclusively on clinical grounds in the majority of the patients. Nine patients who were in good condition remained stable and survived without surgery. Twenty-eight patients had suboccipital craniectomy with evacuation of the hematoma. The mortality rate was 17% for patients who were responsive at the time of surgery and 75% for those who were unresponsive. The proportion of alert patients who deteriorated into a comatose state after various intervals was compared with the operative mortality rate once the patient became comatose. This comparison led to the conclusion that operative intervention is warranted during the first week of the illness in all patients with cerebellar hemorrhage. Only after the eighth day did the predicted mortality rate of the untreated awake patient seem better than the potential operative mortality rate in such patients.

In 1956 Fairburn and Oliver, Lindgren, and Murphy were the first to point out that acute cerebellar infarction constituted a neurosurgical emergency. Most authors agree that acute, symptomatic cerebellar infarction, like cerebellar hemorrhage, should be treated by suboccipital craniectomy with resection of the grossly necrotic tissue. A few mild cases have been treated successfully by simple ventricular drainage. The surgical results appear similar to those in cerebellar hemorrhage, but there is no reported series large enough to give meaningful statistical data.

When cerebellar hemorrhage or infarct is suspected, a CT scan should confirm the diagnosis except when the patient is deteriorating rapidly. A vertebro angiogram is performed only in stable patients with suspected cerebellar infarction. Immediate suboccipital craniectomy and excision of the hematoma or infarcted tissue are performed if there is neurological deterioration and in all patients who have clinical signs of brain stem compression or who have a large hematoma, massive infarction, or hydrocephalus by CT scan. Several reports confirm our experience that deeply comatose patients can be restored to a useful life if their brain stem compression is relieved promptly.

Conclusion

Patients with cerebellar hemorrhage and infarction usually present with a well-defined clinical syndrome that should be recognized by the neurologist or neurosurgeon. CT scanning is an excellent confirmatory test but may not be diagnostic in some cases of cerebellar infarction. Once the diagnosis is made, emergency suboccipital craniectomy with evacuation of the hematoma or infarct should be carried out in all patients except in those who are alert and stable clinically with no signs of brain stem compression and who have a small hemorrhage or infarct without hydrocephalus by CT scanning. Deep coma of recent onset is not a contraindication to surgery since some of these patients will be restored to a nearly normal life with prompt surgical relief of brain stem compression.

References

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