Mortality following Posterior Fossa Infarct: Clinical Course and Prognosis: Case Report

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Abstract:
Infarction of the cerebellum is rare compared to cerebral infarct and mortality following cerebellar infarct is usually high. Their clinical manifestations are very diverse and often unspecfic, meaning that this entity is often mistaken for other more benign conditions. Treatment groups differed regarding the level of consciousness, signs of mass effect in imaging and signs of brainstem involvement. The overall risk for poor outcome depended on the level of consciousness after clinical deterioration. The vascular territory involved did not affect outcome. Medical or surgical management in cerebellar infarction still remain controversy. Surgical intervention (ventricular drainage or decompressive craniotomy) may be necessary in patients with cerebellar infarction if mass effect develops. We report our experience with the management of 2 patients with posterior fossa infarcts.

Key words: Posterior fossa infarct, hydrocephalus, consciousness, prognosis, surgery, mortality.


Introduction:
Infarction of the cerebellum is uncommon, representing approximately 1.5% of strokes.¹,² In 11% to 25% of all cerebellar infarcts ischemic edema becomes space occupying within the posterior fossa, causing brain stem compression and obstructive hydrocephalus.³-⁶

Disturbance of consciousness and secondary brain stem signs develop in hours to days. Three stages of clinical deterioration due to the increasing infratentorial mass can be differentiated.⁷ In the early stage the patient shows signs and symptoms attributable to cerebellar dysfunction. In the intermediate stage consciousness is slightly impaired, and signs of brain stem compression occur. In the late stage the patient becomes stuporous or comatose with posturing and cardiovascular/respiratory instability. Mortality is high in this stage, but there is widespread agreement that neurosurgical intervention by external ventricular drainage and/or decompressive craniotomy improves prognosis significantly.⁸

Nevertheless, there is still controversy regarding the timing and strategy of surgery. Mortality from strokes involving the cerebellum is generally agreed to be high. Cerebellar haemorrhage has been stated to be fatal in as many as 75% of patients and infarction in the distribution of the vertebrobasilar system was fatal within a week of onset in 27% of patients.⁹-¹¹ The recognition of the full spectrum of clinical presentation in cerebellar infarction has occurred since the advent of computed tomography (CT) and magnetic resonance imaging (MRI).¹² These techniques have made possible the diagnosis of minimally symptomatic
patients as well as the delineation of patterns of clinical-anatomic evolution.\textsuperscript{13,14}

\textbf{Report of Cases:}

\textbf{Case 1.}

A 46 year old man had been in good health, with known case of hypertension for 6 months and diabetes mellitus for 4 years under oral medications. The patient presented with multiple episode of vomiting for 5 days, headache for 4 days, and altered level of consciousness for 1 day. On examination on day of admission his GCS was $E_3V_2M_5(10)$, pupils were 3mm bilateral equal and reactive to light .His vitals were pulse-100b/min and regular,BP-160/100mm Hg, temperature –normal. He was conscious but restless and confused. There was no neck stiffness. Both optic discs were blurred on fundoscopic examination. There was no facial weakness. His speech was slurred. Motor and sensory system examination couldn’t be assessed be properly due to poor conscious level. Tendon reflexes were diminished throughout, and both plantar responses were extensor.

MRI of brain T2WI showed hyper-intense lesion occupying left cerebellar hemisphere with mass effect and effacement of fourth ventricle and compression of ipsilateral cerebral peduncle (Fig:1).

Following admission, mannitol and dexamethasone was started. Next day pt.’s condition was improved with GCS –$E_4V_5M_6(15)$,Pulse-88b/Min & BP-140/90 mm Hg.

On 2\textsuperscript{nd} day of admission, patient’s consciousness level deteriorated with fall in GCS-$E_1V_2M_3(6)$,pupil-bilateral reactive 3mm, pulse-124 b/min,BP-180/100 mm Hg. External ventricular drain (EVD) was placed on emergency basis but there was no neurological improvement following EVD placement. So the patient underwent posterior fossa decompression through sub-occipital craniectomy,durotomy and evacuation of devitalized brain tissue on following day. The entire left cerebellar hemisphere was swollen, pale, and soft, with loss of normal convolutional markings. The lateral one third to one half of the left cerebellar hemisphere was resected. Microscopic examination during operation showed acute necrosis of the cerebellar tissue with acute inflammatory changes in the meninges and in the walls of some arteries and veins, suggesting reaction to necrosis. After the operation, he was then transferred to ICU for ventricular support. The patient unfortunately expired on 3\textsuperscript{rd} post-operative day

\textbf{Case 2.}

A 70 year old man with known case of hypertension for 10 years under oral medications presented with headache and neck pain for 1 month and sudden severe headache 7 days back and multiple episode of vomiting for 7 days. On query,he gave history of one episode of convulsion which was tonic-clonic type. On examination on day of admission, his GCS was $E_4V_2M_6(15)$, pupils were 4 mm bilateral equal and reactive to light .His vitals were pulse-90 b/min and regular,BP-140/80 mm Hg, temperature –normal. There was no neck stiffness. Fundoscopic examination of both eyes reveals normal. Motor and sensory system examination revealed normal findings.

MRI of brain T2WI showed hyper-intense lesion occupying right cerebellar hemisphere, both occipital lobe with extension to right parietal lobe (Fig :2-a,b,c).

The patient was managed with dexamethasone and was stable.

On 3\textsuperscript{rd} day of admission, hesuddenly deteriorated with drop in GCS gradually from $E_1V_1M_1(3)$ to $E_1V_1M_1(3)$ within 30 minutes. During this time, he was managed with mannitol, bolus dose of dexamethasone and prepared for emergency

\begin{figure}[h]
  \centering
  \includegraphics[width=0.4\textwidth]{fig1.png}
  \caption{MRI in the axial plane T2WI showing bright hyperintense lesion surrounded by hypo-intense signal abnormality corresponding to infarction in basal medial aspect of left cerebellar hemisphere in posterior inferior cerebellar artery territory.}
  \label{fig:1}
\end{figure}
operative procedure. But before the operation, the patient expired.

**Discussion:**
Cerebellar infarcts constitute 1.5–4.2% of cases in clinicopathological series \(^{15,16}\) and 1.9–10.5% of those in clinical series of patients with cerebral infarctions. \(^{17,18}\) A subgroup of patients with large cerebellar infarctions deteriorate after a variable interval of relatively stable deficits. \(^{19}\) The main predisposing factor for subsequent deterioration is thought to be the infarct size; however, other factors, including type of underlying vascular lesion, hemorrhagic transformation, and inadequate collateral blood flow may be involved. Patients presenting with areas of hypodensity on computed tomography (CT) extending to two-thirds of the posterior inferior cerebellar artery territory are currently considered to be at risk for subsequent deterioration. \(^{20}\)

Neurological deterioration occurs as a result of mass effect of swollen brain tissue in the posterior fossa \(^{21}\) compressing the brainstem, with associated oclusive hydrocephalus. \(^{22,23}\) Mechanisms of clinical deterioration in cerebellar infarcts are recognized and are addressed in current therapeutic guidelines. \(^{19,22,24-26}\) Although there is good evidence that surgery is required to salvage the majority of patients with imminent infratentorial herniation, patient selection criteria, type of surgery (i.e., ventriculostomy, posterior fossa craniotomy), and timing of the procedures are widely disputed. \(^{21,27}\)

Craniotomy is considered to be essential in obtunded patients with massive cerebellar infarcts, and this has therefore prohibited any controlled therapeutic trial design among these patients. \(^{28}\) Other argue that such treatment cannot be advocated in the absence of evidence from randomized controlled trials. Consequently we decided to carry out an observational design to provide prognostic factors and outcomes on the efficacy of therapeutic interventions used in patients with massive cerebellar infarcts. \(^{24,26}\)

McKissock et al. studied 34 patients with cerebellar haemorrhage: 14 who were in good condition at the time suboccipital craniectomy was carried out had a mortality of 35% 9 patients when close to death were treated by ventricular tapping or drainage, and all died. \(^{29}\) Richardson had a 40% surgical mortality, and Ott et al. reported a 17% mortality in cerebellar haemorrhage patients responsive at the time of suboccipital craniectomy, and a 75% mortality in patients who were unresponsive. \(^{30}\)

In another study of 9 patients treated with surgery for cerebellar haemorrhage, the 6 patients only stuporous at the time of surgery recovered, but 3 in coma died. \(^{31}\) The reported mortality after suboccipital craniectomy for cerebellar infarct varies from 2 deaths in 5 cases \(^{32}\) to 5 deaths in 18 patients. \(^{33}\) A review of 17 patients treated with surgery for cerebellar infarct concluded that the mortality was more than 50% if the patient was comatose and zero if the operation was done before coma occurred. \(^{34}\) Consequently, it is recommended that surgery be done early, while the patient is in good condition, and before onset of coma. No means of

**Fig.-2:**
a. **MRI in the axial plane T2WI showing bright hyperintense lesion surrounded by hypo-intense signal abnormality corresponding to infarction in basal medial aspect of right cerebellar hemisphere in posterior inferior cerebellar artery territory.**
b. **MRI in the axial plane T2WI showing bright hyperintense to hypo-intense signal abnormality corresponding to infarction in both occipital lobes in superior cerebellar artery territory.**
c. **MRI in the sagittal plane T2WI showing no significant hydrocephalus.**
differentiating between conscious patients who will do well without surgery and those who require surgery has previously been suggested, but our study shows that whether or not hydrocephalus develops determines the prognosis in these patients, and its presence should be the criterion for surgical intervention.  

**Conclusion:**

Neither surgical treatment nor medical treatment is beneficial in cerebellar infarctions either in awake/drowsy patients or in somnolent/stupor patients. Comatose patients may benefit from ventricular drainage or decompressive craniotomy; however, we found no benefit of surgical versus medical therapy in comatose patients. Level of consciousness is thus the most powerful predictor of outcome predictor. Since clinical deterioration occurs between days 2 and 4, the patient should be kept under observation at least during this period, preferably in a stroke unit or intensive care environment.

**References:**

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