*Review Article*

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# Spontaneous Cerebellar Hemorrhage: Guidelines for Management

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**Abstract**

Spontaneous cerebellar hematomas occupy a special place among all intracranial hemorrhages because of the risk of sudden deterioration due to either acute hydrocephalus or brain stem compression. These hematomas also have a significant incidence of delayed worsening, necessitating careful observation in conservatively treated patients. Unlike supratentorial hemorrhages, there is not much controversy that timely surgical evacuation improves survival and hence it is mandatory for the physician manning the Emergency to know the guidelines for conservative and surgical management.

**Keywords:** Cerebellum; External Ventricular Drain; Hydrocephalus, Hypertension; Vermis; Suboccipital Craniotomy.

## Introduction

10%–15% of all spontaneous intracerebral hemorrhages occur in the cerebellum [1]. It occurs most frequently from the 5th to the 8th decades of life[1]. It occurs more in males than females. While the commonest cause is hypertension [1] (60%-90% cases), other causes include AV malformations, aneurysms, tumor bleed, coagulopathies etc.

Usually, these hemorrhages start near the dentate nucleus [2] and then spread through the ipsilateral hemisphere. However they may extend across the vermis to the opposite side. They may extend into the cerebellar peduncles or rupture into the fourth ventricle, but do not commonly involve the brainstem. Headache, nausea, vomiting, vertigo, 6th nerve palsy or conjugate gaze paresis, ataxia, slurred speech are common presenting features. There may also be neck pain and stiffness due to tonsillar herniation or associated subarachnoid hemorrhage and also obtundation and varying degrees of coma extensor posturing, and hemodynamic or respiratory instability if hydrocephalus or brain stem compression supervenes.

### *Delayed Deterioration: An Unsuspected Danger*

50% of patients with cerebellar hemorrhage show clinical worsening [3] which usually starts 3 days after bleed. Worsening is associated with high mortality (25%–100%) regardless of treatment and deterioration can occur unpredictably, even in patients who appear to have reached a clinical plateau. This is the rationale of advising surgical treatment for the majority of patients. 50% of patients who remained alert and relatively stable for 2 days degenerated into coma over the course of the next several days, and a disconcerting 25% of patients who remained awake for 7 days subsequently deteriorated in the series by Ott et al [3]. This can happen either due to increase in hematoma size or mass effect from increasing perilesional edema. Obstructive hydrocephalus due to either intraventricular extension

or by effacement of the fourth ventricle, is another cause of clinical deterioration.

Brainstem compression, upwards herniation through the tentorial incisura or downward tonsillar herniation through the foramen magnum is the anatomical substrate associated with worsening.

*Basic Management Guidelines*

The basics in medical management of cerebellar bleeds hemorrhage are similar to those of supratentorial hemorrhages. Patients must be observed in a critical care setting with periodic neurological assessment. Coagulation deficits need to be corrected and as many of these patients are elderly they are often on antiplatelets as well. Platelet transfusions, Vitamin K, fresh frozen plasma and other blood products may be required. Normoglycemia and normotension (hypertension to be controlled with a reversible, titratable agent) should be established. Oxygenation must be maintained and hypercapnia avoided. Endotracheal intubation may be necessary to protect airway in patients with a decreased level of consciousness. Mannitol may be given in patients with tight posterior fossa (effaced cisterns and 4$^{th}$ ventricle). Bradycardia may need to be treated with atropine.

Though most cases of spontaneous cerebellar hemorrhage are the result of hypertension, they may be secondary to vascular lesions. Presence of subarachnoid blood, calcification, prominent vascular structures, or edema out of proportion to the size and young age of the patient must prompt the emergency physician to urgently ask for a CT angiogram or DSA (digital subtraction angiogram).

Patients at risk of worsening suddenly include those with hematoma diameter greater than 30 mm, blood pressure greater than 200 mm Hg, presence of brainstem distortion, intraventricular hemorrhage, hydrocephalus, medial location of clot and obliteration of quadrigeminal cistern due to upward herniation.

*Guidelines for Surgery*

It is important to improve patient selection for surgery, identify those who can be managed medically and also those where intensive therapy is likely of no use. However guidelines for surgery vary from centre to centre.

As a rule of thumb, a patient who is awake and has a Glasgow Coma Scale score of 14 or greater with a small hemorrhage (< 30 mm diameter) and with no hydrocephalus may be managed medically with close monitoring and surgical intervention may be required if his clinical condition deteriorates.

Likewise a person who is deeply comatose and without brainstem reflexes with a large midline hemorrhage has a poor prognosis and supportive care without surgery is the only indicated therapy.

The grey area is in patients who are in the middle of these extremes. American Heart Association/ American Stroke Association guidelines [4,5] previously gave a high-level recommendation for surgical removal of hematoma smaller than 30 mm in patients who are deteriorating neurologically or have radiological/clinical brain stem compression or hydrocephalus. However a specific size recommendation is lacking in recent recommendations. Similarly no guideline exists on location of clot (medial vs lateral) as a determinant of surgery. Likewise the preferred type of surgery (ventriculostomy or suboccipital craniectomy and posterior C1 arch removal with clot evacuation and duraplasty) is debatable. While the former is easier technically and has less operative time and blood loss it has the risks of upward herniation (can be eliminated by keeping the bag elevated at 15-20 cms above 3$^{rd}$ ventricle height [6]) and is more of a temporizing measure. It also does not address the mass effect the clot is causing on the brain stem. Suboccipital craniotomy with clot evacuation acts by directly decreasing the raised posterior fossa pressure but has risks of increased blood loss, postoperative CSF leak, infection etc.

Kirrilos et al [7] proposed a management paradigm based on appearance of the fourth ventricle: Grade I (normal size and configuration), Grade II (partially compressed and shifted), and Grade III (completely obliterated). Craniectomy and clot evacuation was recommended for all patients with Grade III compression and for patients with Grade II compression with deteriorating GCS score in the absence of untreated hydrocephalus. Patients with Grade I or II compression are to be initially treated using only ventricular drainage if they developed hydrocephalus and clinical deterioration and stable Grade I and II patients are to be managed conservatively.

While most outcome prediction is based on preoperative GCS and brain stem reflexes, Taneda et al [8] classified the appearance of the quadrigeminal cistern into 3 groups: Grade I (normal), Grade II (compressed), and Grade III (absent) and found good outcomes were reported in 88%, 69%, and 0% of Grade I, II, and III cases, respectively. The predictive value of

the cisternal compression was later confirmed by van Loon et al [9] who found that patients with totally obliterated cisterns had poor outcomes regardless of treatment.

Finally, there are no prospective, multicentre, randomized controlled trials to guide treatment. However, the potential value of neurosurgical intervention remains well established.

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