Original Research Article

Clinical profile of spontaneous cerebellar hemorrhage - An original article

E.A. Ashok Kumar[1]*, P Nikitha[2], P. Jijiya Bai[3]

[1]Professor, Department of General Medicine, Maheshwara Medical College and Hospital, Chitkul(V), Patancheru(M), Sangareddy, Telanganan, India
[2]Intern, Department of General Medicine, Mallareddy Institute of Medical Sciences, Suraram, Ranga Reddy, Telangana, India
3Professor, Department of Pathology, Malla Reddy Medical College For Women, Jeedimetla, Telangana, India
*Corresponding author email: **ashokedla@gmail.com**

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Abstract

Spontaneous cerebellar hemorrhages are rare when compared to cerebral hemorrhages. They are characterized by occipital headache, repeated vomiting, and ataxia of gait. In mild cases there may be only gait ataxia. There may be Dizziness or vertigo, paresis of conjugate lateral gaze towards the side of the lesion, forced deviation of the eyes to the opposite side, or an ipsilateral sixth nerve palsy, blepharospasm, and skew deviation. Dysarthria and dysphagia may occur. The patient often becomes stuporous and then comatose from brainstem compression or obstructive hydrocephalus; immediate surgical evacuation before brainstem compression occurs, may be lifesaving. The introduction of computed tomography (CT) scan, had brought about a sea change in diagnosis and management. The most significant prognostic factors determining the outcome at one month were the grade of quadrigeminal cistern obliteration on the initial CT scan and the Glasgow Coma Scale on admission. The introduction of drugs like nimodepine made all the difference in the outcomes. We hereby share our experience in managing 12 cases of spontaneous cerebellar hemorrhage.

Key words

Spontaneous cerebellar hemorrhages, GCS, Hematoma Volume, Ventricular Extension, Nimodepine.

Introduction

Cerebellar hemorrhages are rare when compared to cerebral hemorrhages. Symptoms usually develop over several hours. They are characterized by occipital headache, repeated vomiting, and ataxia of gait. Dizziness or vertigo is the prominent feature. There may be paresis of conjugate lateral gaze toward the side or an ipsilateral sixth cranial nerve palsy. Blepharospasm, involuntary closure of one eye, ocular drooping, and skew deviation, dysarthria and dysphagia may occur. No Babinski sign until hemorrhage dissects into the ventral brainstem. Patient may become comatose if compression of brainstem occurs [1].

Grading of cerebellar hemorrhages

The cerebellar hemorrhages are graded into 5 grades according to severity of symptoms [2] (**Table - 1**).

Grade I: indicates cerebellar signs without disturbance of consciousness and or progressive neurological deficits, and size of hematoma less than 50 mm without acute hydrocephalus.
Grade II indicates disturbance of consciousness.
Grade III reveals disturbance of consciousness (stupor-semicoma), and size of hematoma less than 50 mm with acute hydrocephalus.
Grade IV reveals severe disturbance of consciousness (semicoma), and size of hematoma less than 50 mm with massive ventricular hemorrhage.
Grade V exhibits deep coma, and more than 50 mm diameter of hematoma [2].

The introduction of computed tomography (CT) scan, had brought about a sea change in diagnosis and management of cerebellar hemorrhage [3].

Calculation of the hematoma size

The size of the hematoma was assessed by calculating both the maximal transverse diameters and the volume. The volume of the hematoma was measured by calculating the area occupied by the hyper dense hematoma on each slice of the CT images and multiplying the area by the thickness of each slice [4].

Grading the degree of compression

The quadrigeminal cistern was identified and the degree of compression was graded by using the method described by Taneda, et al. [5], as follows:
Grade I, normal appearance of the quadrigeminal cistern;
Grade II, compression of the quadrigeminal cistern;
Grade III, complete obliteration of the quadrigeminal cistern.

Management

The patients of Grade I should be treated by conservative therapy. The patients of Grade II, Grade III, and Grade IV should be managed surgically. Surgical treatment for Grade V is not advisable [2]. The level of consciousness may be a better guide in management than the size of the clot [3]. The most significant prognostic factors determining the outcome at one month were the grade of quadrigeminal cistern obliteration on the initial CT scan and the Glasgow Coma Scale on admission [6].

Prognosis

The worst prognosis related to patients with an additional intraventricular hemorrhage and men aged between 50 and 70 years with an admission GCS of less than 7 and occluded cisterns. Early loss of consciousness, additional ventricular bleeding, brain stem extension of the hemorrhage, occluded perimesencephalic cisterns and severe systemic disease, hypertension, diabetes mellitus, liver cirrhosis from alcohol abuse with resulting coagulation disorders were the main factors which led to unfavorable outcome [7]. The important factors related to prognosis of cerebellar hemorrhage are 1. level of consciousness, 2. size of hematoma on CT scan, and 3. massive ventricular hemorrhage resulting in obstruction of the ventricular system [2].

Decision making

Decision making in the management of spontaneous cerebellar hematomas requires careful consideration and interpretation of the various criteria. The configuration of the fourth ventricle on CT scans is a useful sign for selection of the appropriate surgical procedure, compared with estimation of the size of the hematoma and/or evaluation of the quadrigeminal cistern [4]. The patients in a good neurological status on admission (Glasgow Coma Scale 13–15) with a small hematoma (<3 cm) should be treated conservatively. In all patients with large hematomas (>3 cm), including those in a good neurological status, and in all comatose patients, surgical evacuation is indicated [8].

Medical treatment

Calcium channel blockers also known as calcium antagonists block the "slow channel" of calcium influx which reduces the contraction of smooth and cardiac muscle, but does not affect skeletal muscle. It is thus theorized that the abnormal contraction of vascular smooth muscle that may contribute to vasospasm may be mitigated by the administration of calcium channel blockers. Oral nimodipine, a dihydropyridine calcium channel blocker, is the only drug to reduce the incidence of Delayed Cerebral Ischemia (DCI) and the risk of poor outcome in an evidence based manner [9, 10].

Aim of the study

To study the clinical profile of cerebellar hemorrhages

Materials and methods

All cases of cerebellar hemorrhages, admitted to Gandhi Hospital, Musheerabad, were studied retrospectively in Department of Medicine, between May 2002 and April 2011. All the cases were confirmed by CT scan brain.

Exclusion criteria

- All traumatic cerebellar hemorrhages were excluded.
- All drug induced (all anticoagulants) cerebellar hemorrhages were excluded.

Inclusion criteria

- Only non traumatic spontaneous cerebellar hemorrhage cases were included.
- All age group, both males and females were included.
- Both normotensives and hypertensives were included.

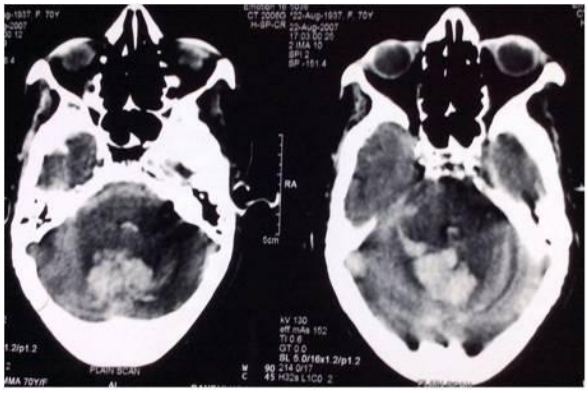
After fulfillment of both exclusion and inclusion criteria, a total number of 12 cases were considered for the study. 10 patients were male and 2 patients were female. Out of 12 cases, 10 cases were normotensive, and only 2 cases were hypertensive. Risk factors for cerebellar hemorrhage were excluded like smoking, alcohol consumption, cocaine use, bleeding diathesis, head injury, etc. by history, clinical examination, and laboratory investigations. Hypertension was present in 2cases. Neurological assessment was done by Glasgow Coma Scale Score (GCSS) and hematoma volume was measured on CT scan, in this method, the estimated volume of the hematoma is half the product of A, B, and C, where A. is the greatest diameter of the hemorrhage on the CT scan, B. is the diameter perpendicular to A, and C. is the number of slices showing hematoma multiplied by the slice. All patients were treated with Nimodipine apart from routine symptomatic treatment.

Results

A total number of 12 Cerebellar hemorrhage cases were admitted to Gandhi Hospital, Musheerabad. Age ranging from 30 to 70 years. 10 were male and 2 were female. 2 were hypertensive and 10 were normotensive.10 survived, 2 died out of 12 Cerebellar hemorrhage cases. Out of 2 hypertensive cases, death occurred in both cases; out of 10 normotensive cases, no death occurred and all of them survived. Two patients had a hematoma volume larger than 50 mm in normotensive group. In two deaths, the hematoma volume was more than 50 mm, both were in hypertensive group and they had Glasgow coma scale of score <13. The two deaths were associated with an additional

intraventricular hemorrhage. These two patients came very late to the hospital, hence surgery could not be contemplated. Cerebral angiogram studies were not done in all the cases, hence we could not know the etiological causes. All patients were treated conservatively with Nimodipine (**Figure – 1, 2**) (**Table – 2**).

Figure – 1: MRI of typical cerebellar hemorrhage.



All Cases were given broad spectrum antibiotic (inj. Ampicillin 500 mg IV 6th hourly, Inj. Flagyl 500 mg IV 8th hourly and Inj. Gentamycin 80 mg IV 12th hourly), Inj. Mannitol 100 mg IV 8th hourly, tablet Nimodipine 30 mg, 2 tablets, 8th hourly, proper care and physiotherapy. The size of the hematoma and level of consciousness were the only significant finding associated with deaths.

Figure – 2: MRI of typical cerebellar hemorrage with hematoma volume >50 mm.

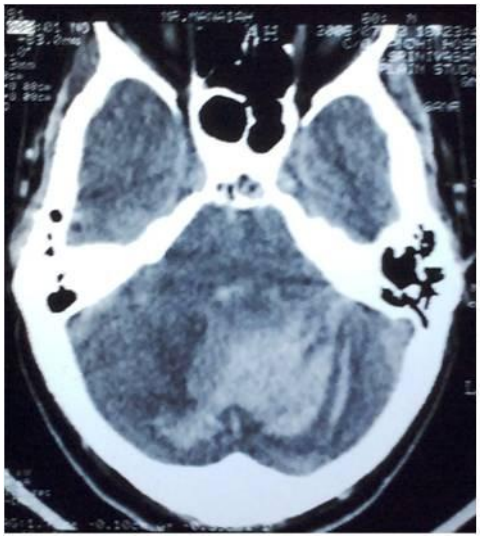


Table - 2: Showing patients characteristics in the study.

Showing patients characteristics in the study					
		Hematoma Volume	GCS score	Ventricular extension	Deaths
Male (10)	Hypertensive(1)	> 50 mm	< 13	1	1
	Normotensive(9)	< 50 mm	> 13	0	0
Female(2)	Hypertensive(1)	> 50 mm	< 13	1	1
	Normotensive(1)	< 50 mm	> 13	0	0

Discussion

Cerebral hemorrhage is a less frequent cause of stroke, than cerebral thrombosis and it is more often thought fatal. It is usually attributed to small vessel disease and the most common sites of hemorrhage are the basal ganglia, internal capsule, cerebellum and pons [11]. Cerebral hemorrhage is mainly of two types A. Primary intra cerebral hemorrhage: where hypertension is common cause and amyloid angiopathy common in normotensives. B. Secondary intra cerebral hemorrhage: where vascular malformations and other risk factors for intra cerebral hemorrhage are common [11, 12]. Although hypertensive intra cerebral hemorrhage remains the most common form of intra cerebral hemorrhage, underlying vascular abnormalities should always be considered in appropriate circumstances, because of high risk of recurrent hemorrhage and available treatment options [13, 14, 15].

Yoshida N, et al. [2], studied 56 patients with hypertensive cerebellar hemorrhage diagnosed by CT scan. The 35 male and 21 female patients ranged in age from 24 to 84 years, 39 of them were operated on. The important factors related

to prognosis of cerebellar hemorrhage were level of consciousness, size of hematoma on CT scan, and massive ventricular hemorrhage resulting in obstruction of the ventricular system. They classified the patient with cerebellar hemorrhage into 5 grades. The prognosis of all of 9 patients in Grade I was good, Eleven out of 13 patients (85%) in Grade II and all of 9 patients in Grade III were alive. Fourteen out of 19 patients (74%) in Grade IV and all of 6 patients in Grade V expired in spite of operation. They advocated that the patients of Grade I should be treated by conservative therapy. The patients of Grade II, Grade III, and Grade IV should be managed surgically. They did not advise surgical treatment for Grade V [2].

van der Hoop RG, et al [3], reviewed the records of 22 patients with spontaneous cerebellar hemorrhage. Nine patients died, four after operation (ventricular shunting in one, clot evacuation in one, both procedures in two patients). The five other patients were considered inoperable, because they showed signs of compression of the caudal brainstem. It is improbable that any of these five could have been saved by immediate ventricular drainage, advocated by some as the only treatment. Thirteen patients were treated conservatively and recovered, although four had a hematoma larger than 3 cm. All these patients had a Glasgow coma scale score of 11 points or more. The level of consciousness may be a better guide in management than the size of the clot [3].

van Loon J et al [6], reviewed a series of 49 consecutive patients with spontaneous cerebellar hematoma, treated according to a standardized management protocol. Seventeen patients were managed conservatively, 30 underwent ventricular drainage, and in six patients the hematoma was evacuated. The most significant prognostic factors determining the outcome at one month were the grade of quadrigeminal cistern obliteration on the initial CT scan and the Glasgow Coma Scale on admission. Patients with normal cisterns had a good outcome, and only needed (temporary) ventricular drainage in case of hydrocephalus. Patients with totally obliterated cisterns had a bad outcome irrespective of treatment. In the patients with compressed cisterns, it is suggested that evacuation of the hematoma might improve outcome; treatment of hydrocephalus alone is insufficient in many cases in this group [6].

Cerebellar hemorrhages may enter ventricles and can cause an acute life-threatening hydrocephalus; therefore such hematomas should be operated on. Subcortical hematomas, which are usually not associated with hypertension and may be due to tumor or vascular malformation, should as a rule be operated on. Carotid angiography is necessary for most supratentorial hematomas to exclude the presence of aneurysm or arteriovenous malformation. Secondary hematomas from ruptured arterial aneurysm should be operated on as urgently as traumatic intracranial hematomas if the patient's level of consciousness is deteriorating and if there is severe neurological deficit. Hematomas due to arteriovenous malformation must sometimes be evacuated as an emergency measure if the patient is unconscious, and the malformation should be excised if technically possible. The operation should preferably be postponed to the second week after the bleeding if the patient's level of consciousness is not deteriorating, since the malformation is more easily excised after the brain edema has subsided. Hematomas associated with anticoagulant treatment should be evacuated if the hematoma is expansive and if the patient is unconscious or somnolent but the results are not very good. Hematomas of hemophiliacs should be evacuated, and these patients need an appropriate replacement therapy [7].

Donauer E ,et al [16], carried out a prospective study for the treatment of cerebellar hemorrhage in a non-selected group of 33 patients. All of them required intensive care respectively with intensive supervision.

The following management protocol has been established.

I. Cases with small hemorrhage, in good clinical condition, without hydrocephalus and/or occlusion of the basal cisterns: intensive supervision, operative intervention only if they deteriorate into one of the following groups.

II. Cases with hydrocephalus — even if not yet pronounced — but without occluded basal cisterns and without major tonsillar herniation: pressure monitored ventricular drainage, which opens at 15 mm Hg and thus prevents higher CSF pressure developing.

III. a): Cases with large hematoma, occluded basal cisterns and/ or tonsillar herniation, but without severe general risk factors, as a first step: pressure monitored ventricular drainage; as a second step, if they do not improve soon after the normalization of the ventricular pressure: open surgical evacuation of the hematoma, which also decompresses the posterior fossa. If present and possible, causative vascular malformations may be dealt with at the same session.

b): Same intracranial situation, but patients with severe general risk factors: pressure monitored ventricular drainage only.

IV. Cases with causative aneurysm or angioma, who initially had been treated conservatively or by ventricular drainage: secondary operation of the vascular malformation after stabilization of the general condition.

Overall mortality was 33%. For the 13 men, the initial average Glasgow Coma Scale (GCS) grade was 9.2 and the median hematoma diameter 3.7 cm. For the 20 women, the corresponding figures were 10.4 GCS and 3.4 cm. Of the 13 men, 6 died (mortality 46%); Of the 20 women, 5 died (mortality 25%). The worst prognosis related to patients with an additional intraventricular hemorrhage and men aged between 50 and 70 years with an admission GCS of less than 7 and occluded cisterns. Early loss of consciousness, additional ventricular bleeding, brain stem extension of the hemorrhage, occluded perimesencephalic cisterns and severe systemic diseases like diabetes mellitus, liver cirrhosis from alcohol abuse with resulting coagulation disorders, were the main factors which led to unfavorable outcome [16].

Ramez W, et al. [4], developed a treatment protocol which was prospectively applied for the management of 50 consecutive cases of cerebellar hematomas. The appearance of the fourth ventricle, adjacent to the hematoma, on computed tomographic scans was divided into three grades (normal, compressed, or completely effaced). The degree of fourth ventricular compression was correlated with the size and volume of the hematoma and the presenting Glasgow Coma Scale (GCS) score. The hematoma was surgically evacuated for all patients with Grade III compression and for patients with Grade II compression when the GCS score deteriorated in the absence of untreated hydrocephalus. Patients with Grade I or II compression were initially treated with only ventricular drainage in the presence of hydrocephalus and clinical deterioration.

Decision-making in the management of spontaneous cerebellar hematomas requires careful consideration and interpretation of the various criteria. The configuration of the fourth ventricle on CT scans is a useful sign for selection of the appropriate surgical procedure, compared with estimation of the size of the hematoma and/or evaluation of the quadrigeminal cistern. The results of the study support the management protocol. The outcomes of patients with Grade III ventricular compression and GCS scores of less than 8 at the time of treatment were poor, despite aggressive treatment. A more aggressive approach should be recommended for young patients. Simply, the protocol uses GCS scores to select patients for

surgical treatment and uses the degree of fourth ventricular compression to select the surgical procedure [4].

Cohen ZR, et al. [8], predicted of the outcome by a logistic regression model which included both the size of the hematoma and the treatment modality (surgery or conservative) as variables. The data suggest that patients in a good neurological status on admission (Glasgow Coma Scale 13–15) with a small hematoma (<3 cm) should be treated conservatively. In all patients with large hematomas (>3 cm), including those in a good neurological status, and in all comatose patients, surgical evacuation is indicated. A different hematoma size as a cut-off point for choosing surgery as the preferred therapeutic modality, as well as the indication for ventriculostomy as a temporizing measure or a substitute for surgery are yet undetermined and need further investigation in a randomized trial [8].

Remote cerebellar hemorrhage (RCH) after supratentorial craniotomy is a very rare complication and can be a life threatening and results in death. It is considered to avoid rapid loss of CSF intra-operative (i.e., avoiding excessive vacuum drainage, conservative treatment and serial CT scan [17, 18].

Brain arteriovenous malformations presenting with hemorrhage, with deep venous drainage, or associated aneurysms have 2-fold greater likelihood of a future hemorrhage. Partial treatment by embolization does not alter these risks. This natural history should be taken into account in the treatment strategy [19].

Posterior fossa AVMs present an increased risk for hemorrhage and for increased morbidity and mortality. Cases with hematoma should be operated on an urgent basis. The hematoma volume is a factor that impacts postoperative results and prognosis [20].

Nimodipineis the most extensively studied has better penetration into the brain and brain vessels. Subarachnoid and cerebral hemorrhage causes sustained vasopasm. This vasospasm causes all the clinical signs and symptoms of ischemic neurological deficits. Nimodipine is a dihydropyridine ca2+ antagonist which dilates the vasospastic cerebral arterioles and increase cerebral blood flow. Nimodipine was used in all the cases as initial medical treatment, with good results, when compared to 50% mortality in other studies where Nimodipine was not given. The STICH study shows, that in patients with spontaneous supratentorial intracerebral hemorrhage in neurosurgical units show no evidence of overall benefit with a policy of early surgery compared with initial conservative medical treatment [21].

Currently, nimodipine is the most efficacious and widely used medication in the setting of Post Traumatic Vasospasm (PTV) . Nimodipine works as a calcium channel blocker by antagonizing the effect of dihydropyridine channels in smooth muscle cells. This decreases calcium influx into cells, which then decreases smooth muscle cell contractility, ultimately preventing vasospasm from occurring [22, 23, 24].

Conclusion

The most common sites of hemorrhage are the basal ganglia, internal capsule, cerebellum and pons. Cerebellar hemorrhages are rare when compared to cerebral hemorrhages, although hypertensive intra cerebral hemorrhage remains the most common form of intra cerebral hemorrhage, underlying vascular abnormalities should always be considered in appropriate circumstances, because of high risk of recurrent hemorrhage and available treatment options.

The cerebellar hemorrhages are graded into 5 grades according to severity of symptoms. The important factors related to prognosis of cerebellar hemorrhage are 1. level of consciousness, 2. size of hematoma on CT scan, and 3. massive ventricular hemorrhage resulting in obstruction of the ventricular system. Hence,

the decision making in the management of spontaneous cerebellar hematomas requires careful consideration and interpretation of the various criteria.

The available literature suggests that the patients in a good neurological status on admission (Glasgow Coma Scale 13–15) with a small hematoma (<3 cm) should be treated conservatively. In all patients with large hematomas (>3 cm), including those in a good neurological status, and in all comatose patients, surgical evacuation is indicated.

The introduction of drugs like nimodepine made all the difference in the outcomes. We hereby share our experience in managing 12 cases of spontaneous cerebellar hemorrhage.

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