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CASE REPORT

Remote cerebellar hemorrhage: Report of 2 cases and review of literature

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ABSTRACT

Remote cerebellar hemorrhage (RCH) is an extremely rare and potentially devastating complication of supratentorial and spinal surgeries. While there are numerous postulates explaining the patho-physiology behind this phenomenon, including the most popular CSF over drainage theory, the exact cause for the same is still largely unknown. In this report, we present 2 cases of remote cerebellar hemorrhage encountered following 2 different surgical procedures. One patient had preceding pterional craniotomy for ruptured anterior communicating artery aneurysm while the other one developed RCH after placement of EVD. Both of them had history of poorly controlled hypertension, contrary to most reports where hypertension has not been found to be commonly associated with it. Moreover, while most cases have been reported to occur following supratentorial craniotomies and spinal surgeries, one of our patients developed the same after placement of the EVD, which, to the best of our knowledge, has not been reported earlier.

Key words: Craniotomy, external ventricular drain, remote cerebellar hemorrhage

Introduction

While hematoma at operative site is not uncommon in neurosurgery, spontaneous cerebellar hematoma remote from the site of surgery is extremely rare.[1,2] This potentially devastating complication has been described after supratentorial and spinal surgeries. While there are numerous postulates explaining the patho-physiology behind this phenomenon, these are, at best, speculative and none of these is established as yet. Outcome is mainly dependent on the degree of bleed and can range from complete recovery to even death. Here, we share our experience with 2 cases of RCH and present a brief review of the literature.

Case Reports

Case 1

A 71-year-old lady with history of poorly controlled hypertension was brought to the emergency room with complaints of sudden onset of loss of consciousness 12 h earlier with no improvement in sensorium since then. There was no history of seizures, decreased movements on one side of the body, or trauma. On examination she was unconscious, not opening her eyes to pain, and only localizing to deep central pain with both upper limbs. Her pupils were bilaterally equal and sluggishly reacting to light. Non-contrast computerized tomogram (NCCT) revealed a hematoma in the left cerebellar hemisphere measuring about 3 × 4 cm with effacement of the fourth ventricle and compression of the brainstem [Figure 1a] and hydrocephalus. She was operated emergently and a left sub occipital craniectomy was performed and the hematoma was evacuated. Postoperatively the patient was electively ventilated and after 6 h could localize pain with both her upper limbs. Postoperative NCCT showed a small residual hematoma in the left cerebellar hemisphere with operative site edema. The 4th ventricular effacement was persistent and ventriculomegaly had increased in comparison to the preoperative scan [Figure 1b]. Hence, external ventricular drain (EVD) was inserted to allow temporary CSF diversion. The EVD was placed in the right Kocher's point using a twist drill, 1 cm anterior to the coronal suture and 2 cm lateral to the midline. The right frontal horn was hit at a depth of 4 cm in one attempt and clear CSF was drained. However, soon after the EVD placement, the patient stopped moving her limbs to pain, and her pupils became pin pointed with no reaction to light. The respiratory effort that she had developed on the ventilator also ceased. An emergency NCCT brain revealed a large hematoma in the left cerebellar hemisphere with compression of the brainstem and

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hemorrhage within the substance of the brainstem extending from the pons to the midbrain supratentorially [Figure 1c and 1d]. The patient showed no further neurological improvement and died on the fifth postoperative day.

Case 2

A 50-year-old lady with poorly controlled hypertension presented to our emergency department with abrupt onset of severe holocranial headache accompanied by recurrent vomitings 3 days prior to admission. On examination, she was found to be drowsy and disoriented with evidence of meningism. There was spontaneous movement in all 4 limbs. Non enhanced CT scan of the head revealed thick subarachnoid hemorrhage in the anterior interhemispheric fissure without any evidence of infarction or hydrocephalus [Figure 2a]. CT angiography was done keeping the possibility of aneurysmal rupture in mind. CTA revealed an anterosuperiorly directed saccular aneurysm arising from the anterior communicating artery. The left A1 was dominant and the aneurysm fundus was directed to the right side [Figure 2b]. So, the patient was taken up for surgical clipping of the aneurysm via left pterional craniotomy. The intraoperative findings were consistent with preoperative CT angiography. The aneurysm was clipped uneventfully with a 6 mm straight clip. The patient was extubated on the morning following surgery after overnight elective mechanical ventilation. However, due to persistent drowsiness and worsened motor response (M4), CT head was repeated which revealed significant operative site edema with midline shift and subfalcine herniation. It also revealed a hematoma in the left cerebellar hemisphere with blood in the cerebellar folia but without any 4th ventricular effacement or hydrocephalus [Figure 2c]. Hence, she was planned for urgent surgical decompression of the edematous left frontal lobe but due to deranged APTT, she could be taken up for surgery only after correction of the same. Following decompressive lobectomy with expansive duroplasty, the patient recovered gradually and was discharged in a satisfactory condition.

Discussion

Hematoma in and around the operative site is a common occurance in neurosurgical practice and more often than not, denotes inadequate intraoperative hemostasis. However, hemorrhage at a site distant from the area of surgery is very rare.

Remote cerebellar hemorrhage (RCH) has been defined as hemorrhage into the cerebellar parenchyma as the result of a neurosurgical intervention carried out at an anatomically unrelated area away from it.[1,2]

It is a rare but potentially life threatening complication of supratentorial and spinal surgeries. The estimated incidence of RCH following supratentorial surgery is around 0.3 to0.6 %[3] whereas the same after spinal surgeries is not exactly known.

RCH is most common between the ages of 30 and 60 years, though it has been reported in patients as young as 10 and as old as 83 years. The most common presenting symptom of RCH is decreased level of consciousness. Other common symptoms may include motor deficits, gait ataxia, and prolonged awakening from anesthesia. Some cases are asymptomatic and found incidentally on postoperative CT or MR imaging[4]. RCH seems to be located bilaterally (~53.5%) just as often as unilaterally (~46.5%)[5] Co-affection of the vermis has frequently been reported, although a mere affection of the vermis is seldom seen.[6]

Various supratentorial and spinal surgeries are complicated by this rare complication. These include craniotomies for aneurysm surgery, temporal lobectomy,[2] craniotomy and evacuation of acute subdural hematomas,[7] craniotomy for tumor excision, and spinal surgeries with intended or inadvertent duratomies. There are reports of RCH after seemingly innocuous procedure like burr-hole evacuation of chronic subdural hematoma.[8] However, to the best of our knowledge, there is no report of an extensive rebleed into brainstem and expansion of the cerebellar hematoma

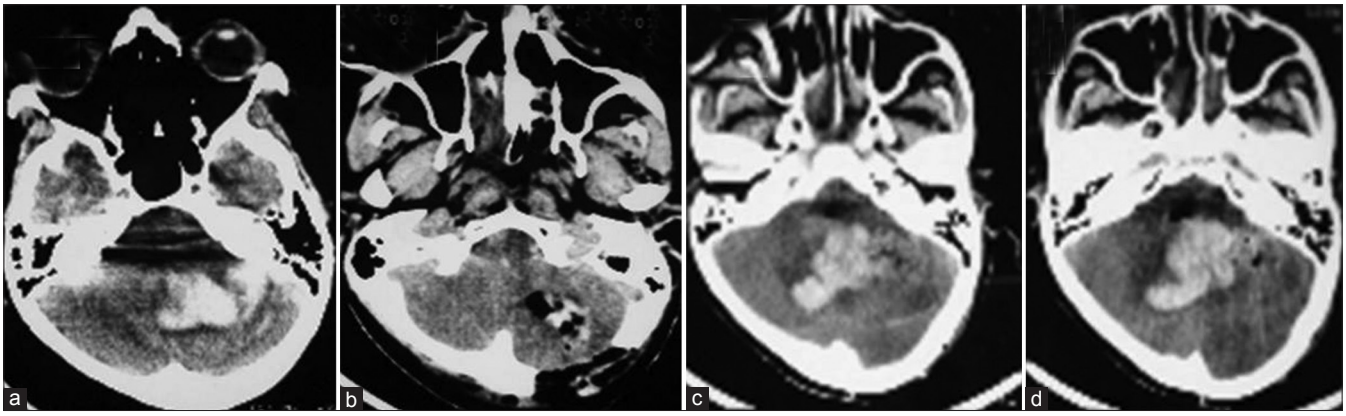

Figure 1: (a) Plain CT head showing left cerebellar hematoma; (b) Postoperative CT head showing near complete hematoma evacuation; (c and d) Increase in size of cerebellar hematoma and extensive bleed involving the brainstem and cerebellar peduncle. Postoperative CT head showing near complete hematoma evacuation

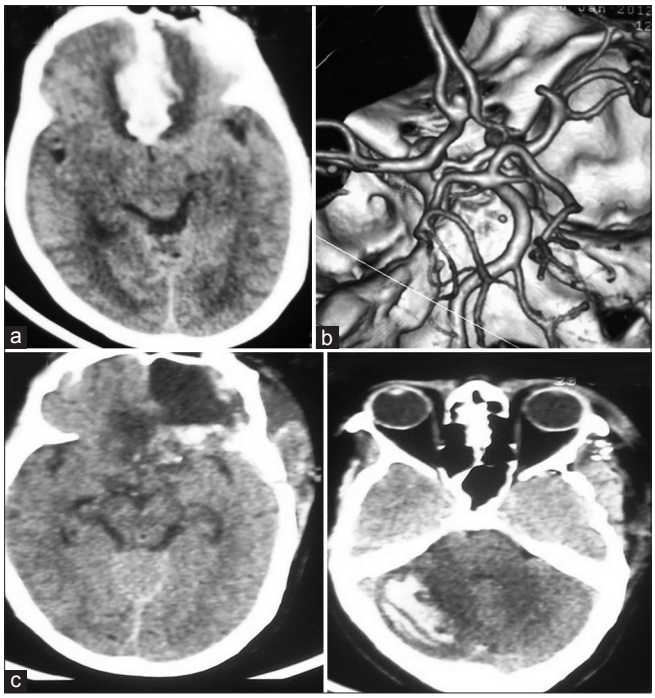

Figure 2: (a) Thick SAH involving the anterior interhemispheric fissure with perifocal edema seen involving bilateral basi-frontal lobes- suggestive of Fisher Grade 4 hemorrhage, (b) Reconstructed CT angiogram showing an antero superiorly directed a-comm. Aneurysm with dominant left A1 and hypo plastic right A1, (c) Postoperative CT of head showing evidence of left frontal lobectomy with RCH. It is seen involving the upper part of the right cerebellar hemisphere

following external ventricular drainage of CSF like observed in our first patient.

However, many believe that the underlying lesion for which the supratentorial craniotomy is performed is unrelated to spontaneous cerebellar hemorrhage.[9] What is believed to be more important is that these procedures are associated with significant CSF drainage with attendant brain shifts, mainly downward displacement of the cerebellum which is responsible for the RCH.[2]

Interestingly, it has been observed that RCH tends to be more common following surgery for unruptured aneurysm.[10] This is thought to be so because in presence of acute SAH, there is effacement of CSF spaces and hence CSF loss during craniotomy is lesser than expected. Whereas the CSF loss is more in unruptured aneurysms due to patent CSF spaces and hence more is the risk of RCH. However, one of our patients developed RCH inspite of having Fischer grade 4 SAH with considerable cerebral edema.

As far as the bleeding pattern in RCH is concerned, classically it occurs in the sulci of superior surface (tentorial surface) of one or both cerebellar hemispheres giving the classic radiologic sign know as the Zebra sign.[11] Additionally, intraparenchymal bleed especially in the upper part of the cerebellar hemisphere is also known to occur.

The timing between operations, onset of symptoms, drained volume of CSF, and the diagnosis of RCH in many cases have not been reported. Most often it occurs during the first 10 h (46%). However, the development of RCH after more than 40 h is not uncommon (17%)[5]

There are a multitude of postulates explaining the pathophysiology of this phenomenon. Chadduck [12] postulated that sudden rise in blood pressure might cause an increased gradient between intravascular pressure and CSF pressure, and thus induce hemorrhage into the cerebellar parenchyma. Andrews and Koci [13] speculated that RCH was nothing but hemorrhagic conversion of an infarct resulting from transient kinking, spasm or traction of superior cerebellar artery. While most others believe that drainage of large volumes of CSF due to whatever reason in the peroperative period leads to downward cerebellar displacement with consequent shearing away of bridging cerebellar veins between the tentorium and superior cerebellar surface.[5]

Other less popular mechanisms suggested to contribute to this remote cerebellar hemorrhage include hypertension, occult arteriovenous malformation (AVM) bleed, deranged bleeding parameters or jugular venous obstruction from extreme head rotation during patient positioning at surgery.[2,5,9,14] While evaluating factors contributing to RCH, intra operatively elevated systolic blood pressure and history of aspirin intake were found to be significant.[5]

Coagulation parameters may have an indirect effect on cerebellar hemorrhage; these certainly do not seem to be major contributor. Marquardt *et al.*,[14] were not able to find any correlation between coagulation parameters and RCH; they proposed that abnormal blood coagulation could be a contributor of RCH. Friedman *et al.*,[2] found that use of ASA within 7 days of surgery did increase the risk of RCH, yet they reported that there were no differences between control and RCH patients' coagulation parameters. Out of our 2 patients, coagulation parameter was normal in one whereas it was deranged in the other. However, we consider that deranged coagulation parameter was probably not the cause in our patient because had it been the culprit, the patient ought to have bled in the operative site as well.

Peri-operative hypertension is also a known predisposing factor for RCH[2] and was present in both of our patients. However, Marquardt *et al.*,[14] found only one patient to have hypertension among their series of 9 patients and concluded that there was no relationship between history of hypertension or preoperative hypertension and RCH. Similarly, in their series consisting of 7 cases, Amini *et al.*,[9] did not encounter even a single patient with hypertension.

As far as the management is concerned, small hemorrhages without any clinical evidence of raised ICP or brainstem

compression can be managed medically with serial imaging to document resolution of the hematoma. On the other hand, larger hematomas with significant mass effect in the posterior fossa need urgent surgical intervention either in the form of CSF diversion or expanded sub occipital craniectomy with hematoma evacuation and duraplasty. General measures in the form of blood pressure control and correction of coagulopathies are also very important and go side by side in the management of such patients.[5, 9,15]

The outcome in patients with RCH varies significantly and seems primarily to depend on the extent of bleeding, its intracerebellar component, the underlying disease, the amount of time before action is taken and further complications.[5] In cases with blood only in the sulci of the upper cerebellum, severe complications or serious permanent cerebellar defects are seldom observed. Nevertheless, the greater the extent of intracerebellar hemorrhage, the greater is the risk of acute obstructive hydrocephalus and associated complications. The outcome is generally good with more than 50% of all cases having either complete recovery or with only mild residual neurological symptoms, while death occurs in ~10 to15%[5] of the cases.

As far as the prevention of RCH is concerned, based on the existing knowledge of the underlying mechanisms, certain measures will definitely go a long way in prevention of this potentially fatal complication. These include good peroperative blood pressure control, evaluation and correction of coagulopathies, attention to details during surgery like avoidance of rapid drainage of CSF, avoidance of unintended durotomy, replacement of isotonic saline before watertight dural closure, etc.

The case number 1 might give an impression of bleed at the operative site but this wasn't the case. This is so because postoperative scan showed near total evacuation of hematoma and the patient deteriorated suddenly after placement of external ventricular drain. This not only led to acute expansion in hematoma size but also extensive brainstem bleed. With coagulation parameters within normal range, first postoperative CT showing near total evacuation of the hematoma and entire event following placement of EVD, we were left with no doubt regarding the diagnosis of RCH in this case. This case is also probably the first report of RCH following EVD placement. This case also underscores the importance of gradual decompression of the ventricles when EVD is placed.

The case number 2 is a classical case of RCH as described in literature. However, as already mentioned, in this case, RCH occurred following surgery for ruptured aneurysm. This was contrary to the belief that ruptured aneurysm provides a relative immunity as far as RCH is concerned.[10]

Conclusion

RCH is a rare but potentially life threatening complication of supratentorial and spinal surgeries. The exact pathophysiologic mechanism behind this is not exactly known; however, over drainage of CSF with attendant brain shift appears to be a very important and consistent factor in its genesis. Although rare, RCH must be considered in patients with unexplained neurological deterioration after supratentorial or spinal surgery. Timely diagnosis may prevent the dreaded sequelae of RCH. Management needs to be individualized. Small cerebellar hematomas can be managed medically, but larger lesions that cause significant mass effect in the posterior fossa needs surgical intervention. Most cases are self limiting and are associated with good outcome.

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