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REMOTE CEREBELLAR HEMORRHAGE. A CASE REPORT AND REVIEW OF LITERATURE

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ABSTRACT Remote cerebellar hemorrhage (RCH) is an extremely rare complication of supratentorial surgeries. There are numerous theories explaining the patho-physiology behind this phenomenon, including the CSF over drainage theory, but the exact cause is still unknown. In this report, we present a case of remote cerebellar hemorrhage, encountered following removal of supratentorial craniopharyngioma without loss of large CSF volume. RCH can be an unusual finding on routine postoperative imaging studies and should not be mistaken for more common causes of bleeding such as hemorrhagic infarction, coagulopathy or cortical vein occlusion. Cerebellar hemorrhage in the typical setting can be identified as RCH and it does not require more extensive or invasive evaluation.

KEYWORDS : Remote Cerebellar Hemorrhage, Supratentorial Craniotomy, Craniopharyngioma

INTRODUCTION

Remote cerebellar hemorrhage (RCH) has been defined as bleeding within the cerebellar parenchyma, a rare compli cation that can occur after neurosurgical intervention.[1] Most of the postoperative intracranial haemorrhages occur around the operative site. Spontaneous cerebellar hematoma remote from the site of surgery is an extremely rare complication. Cerebellar haemorrhage after supratentorial craniotomy is the most commonly described pattern of remote haemorrhage. Perioperative hypertension, head positioning, lumbar drainage of cerebrospinal fluid (CSF), and postoperative epidural drainage have each been proposed to be the cause of remote cerebellar haemorrhage.[2] Outcome mainly depends on the degree of bleed and can range from complete recovery to even death. RCH is an incidental finding on standard radiological imaging modalities done as a part of post- operative workup in patients who undergo these supratentorial surgeries.

Here, we report a case of RCH that occurred following removal of craniopharyngioma without loss of a large volume of CSF and present a brief review of literature.

CASE REPORT

A, 13-year-old girl, presented with complaints of progressive diminution of vision in the eyes for 3 months and occasional headaches. Ophthalmic examination revealed vision of 6/12 in the left eye and 6/24 in the right eye, with optic atrophy of bilateral eye. Multiplanar MRI brain was done. MRI revealed large lobulated T1/T2 hyperintense lesion, extending from base of skull in the region of basisphenoid, pituitary sella, suprasellar cistern up to the frontal horn of lateral ventricle, measuring ~7.4 x 7.8 x 7.0 cm (antero-posterior, transverse, cranio-caudal). Posteriorly lesion extends up to the tectum of midbrain, causing widening of interpeduncular fossa and mild compression of aqueduct, resulting into dilatation of bilateral lateral and 3rd ventricle. Laterally lesion extends into bilateral sylvian fissure (left more than right), compressing the adjacent brain parenchyma. Anteriorly lesion extends into base of anterior cranial fossa, compressing the frontal lobe and optic chiasma. Pituitary gland was compressed by the mass. There was no evidence of diffusion restriction/ fat component within. Few T2 hyperintense areas were seen at the periphery of mass on right side – likely peripherally trapped CSF/ cystic component. Mass was encasing the M1 segment of right MCA, M1, and M2 segment of left MCA and bilateral A1 and A2 segment. On post contrast study, few nodular enhancing areas were seen within – likely mural component. (Image1) Vision of the right eye recovered completely 3 days postoperatively. Histopathological exami nation of the excised mass showed features suggestive of craniopharyngioma. Postoperatively, the level of consci ousness remained unchanged from the preoperative level and no new neurological deficits were detected.

A postoperative MRI scan was done on the 10th post-operative (post-op) day, revealed large post-op cavity seen in left bilateral basifrontal, sella, suprasellar region, interped uncular fossa and left temporal region. There was small enhancing linear and nodular component seen in right lateral and posterior aspect of post op cavity, measuring 13x 7 mm and 9 x 10 mm respectively, representing residual tumour. Multiple areas of blooming seen within post op site, bilateral cerebral hemisphere. Also, there was a well-defined T1/T2 hyperintense lesion measuring 14 x 10 mm seen in right cerebellar hemisphere showing peripheral blooming on SWAN sequence (Image 2). These findings were consistent with remote right cerebellar bleed. On follow-up, she was doing very well, without any new neurologic deficits.

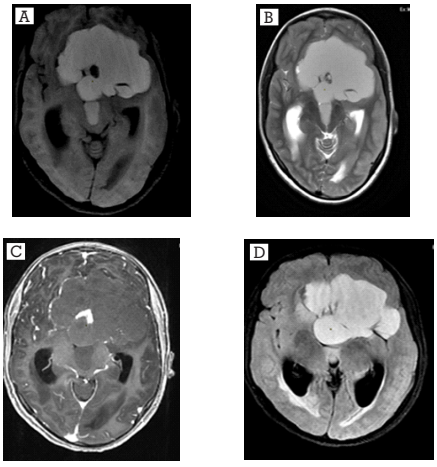

Image 1. Axial T1-weighted (A), T2-weighted(B), post contrast (C), and FLAIR (D) images show a large supratentorial craniopharyngioma with non-communicating hydrocephalus

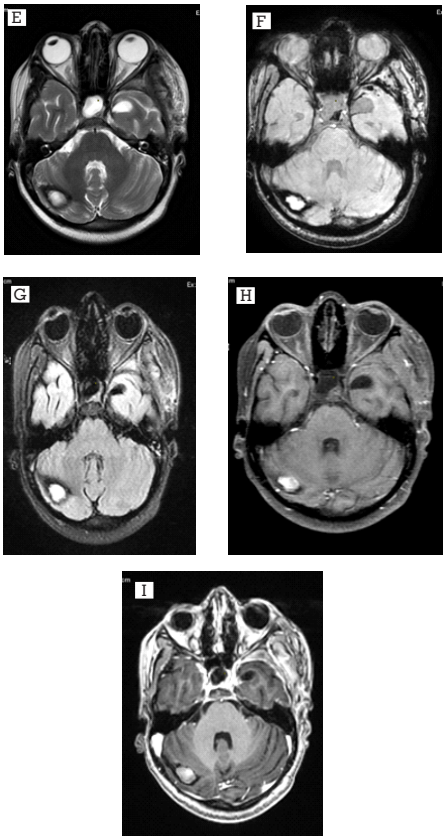

Image 2. Axial T2-weighted (E), FLAIR (F), and gradient– (G) images show a left cerebellar hemisphere haemorrhage with mild mass effect and minimal adjacent oedema. (H& I) No enhancement seen in the post contrast T1-weighted MR image.

DISSCUSSION

RCH has been defined as haemorrhage into the cerebellar parenchyma as the result of a neurosurgical intervention carried out at an anatomically unrelated area away from it.[3,4] RCH is a less recognized complication of supratentorial craniotomies, with an incidence of about 0.08-0.6%.[2] RCH comm only occurs 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. Various surgeries complicated by this complication include craniotomies for temporal lobectomy, aneurysm surgery, tumor excision, craniotomy and evacuation of acute subdural hematomas, and spinal surgeries with intended or inadvertent durotomies. There are a few reports of RCH after procedure like burr-hole evacuation of chronic subdural hematoma.[5] The bleeding in RCH classically occurs in the sulci of superior surface (tentorial surface) of one or both cerebellar hemispheres giving the classic radiologic sign known as the Zebra sign.[6]Intrapa renc hymal bleed in the upper part of the cerebellar hemisphere can also occur.

The timing between operations, drained volume of CSF, onset of symptoms 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%).[9]

The most common presenting symptom of RCH is impaired consciousness. Other common symptoms include gait ataxia, motor deficits and prolonged awakening from anesthesia.[5] However; some cases are asymptomatic and are incidentally diagnosed on postoperative neuroimaging. MRI findings in RCH include feature of haemorrhagic infarct, causing T1 hypo-isointense signals and T2/FLAIR hypo or hyperintense signals depending whether the scan was done in acute or hyperacute stage. No diffusion restriction is seen. Blooming is seen on the SWI/GRE sequence.

There are many postulates explaining the pathophysiology of this phenomenon. Chadduck postulated that acute elevation blood pressure might cause an increased gradient between intravascular pressure and CSF pressure, and thus induce haemorrhage into the cerebellar parenchyma.[7] Andrews and Koci speculated that RCH was haemorrhagic conversion of an infarct resulting from transient kinking, spasm or traction of superior cerebellar artery.[8] While others believe that drainage of large volumes of CSF in the preoperative period leads to downward cerebellar displacement with consequent shearing away of bridging cerebellar veins between the tentorium and superior cerebellar surface.[9] Other less popular mechanisms include hypertension, deranged bleeding parameters, occult arteriovenous malformation (AVM) bleed or jugular venous obstruction from extreme head rotation during patient positioning at surgery and history of aspirin intake were found to be significant.

Most of the cases of RCH are treated conservatively except few of them which require the extra ventricular drainage and/or decompression surgery. The outcome is usually good with more than 50% of cases having either complete recovery or with only mild residual neurological symptoms, while death occurs in 10 to15% of the cases.[9]

On the basis of existing knowledge of the underlying mechanisms, few measures may help in prevention of this potentially fatal complication. These include good preop era tive blood pressure control, attention to details during surgery like avoidance of rapid drainage of CSF, evaluation and correction of coagulopathies, replacement of isotonic saline before water tight dural closure, avoidance of unintended durotomy etc.[10]

CONCLUSION

RCH is a rare complication which may have a devastating outcome if left undiagnosed and untreated. Radiologist knowledge of these entities is very important, because their proper, early identification can promote interventions aimed at their correction and at alleviating the associated symptoms.

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