Acta Scientific Neurology (ISSN: 2582-1121)

Volume 4 Issue 9 September 2021

Case Series

Postoperative Intracranial Remote Haemorrhages: An Experience from 2 Tertiary Care Hospitals

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Received: January 15, 2021

Published: August 19, 2021

Abstract

Postoperative haemorrhages (POH) after any neurosurgical procedure can be seen at surgical site or at virgin sites away from operative field, more commonly known as remote haemorrhages. Cerebellum is the most common site of remote POH while supratentorial remote POH are very sparsely described in literature. usually benign in their clinical course but sometime can condemn patients to devastating morbidity and mortality. Due to the rare reporting, natural history of remote POH and its exact pathophysiology is not very well known that can put clinicians in a jumbled position. Multiple theories have tried to explain its genesis, of which most widely accepted are "Cerebellar sag theory" and "Drain theory". Other theories that lack good supportive evidence blames surgical position, sudden decompression of intraaxial lesion, preoperative coagulation defects and cryptic vascular malformations as plausible culprits for remote POH. Their treatment is usually based on severity of clinical presentation and can range from observation to need of surgical evacuations.

In this report we present our experience with identification of two cases with remote POH, case 1 is a first in world literature report describing a combined remote supratentorial and cerebellar POH and case 2 describes a classical remote cerebellar haemorrhage. Both had benign clinical course and were managed conservatively. We also reviewed the literature and discussed the theories behind its pathophysiology in general and pertaining to our experience.

Keywords: Cerebellar Sag Theory; Postoperative Haemorrhages (POH); Drain Theory

Introduction

"Surgeons must be very careful When they take the knife! Underneath their fine incisions Stirs the Culprit-Life!" [1] an enthralling quote that describes the uncertainty of complications associated with any surgical procedure. One such grim complication of neurosurgery is postoperative haemorrhage (POH) that can condemn patient to long-term morbidities and mortality. Radiologically diagnosed POH after any neurosurgical intervention can be divided as surgical site POH (incidence of 10.8% - 50%) [2] and remote POH that seen in virgin sites away from operative field. Remote cerebellar haemorrhages (RCH) after supratentorial or spinal surgeries are most commonly described pattern of remote POH with incidence of 0.08% - 6% [3] while supratentorial POH after infratentorial craniotomies has an incidence rate of 0.06%

[4]. Remote Supratentorial Haemorrhage (RSH) after supratentorial craniotomies is an extremely rare entity with exact incidence not known. Due to scarce description of remote POH in literature; natural history of such disease, its pathophysiology and treatment protocols are not uniform to be practiced by clinicians ubiquitously. Here, we describe our experience with two cases of remote POH from two different tertiary care centre in India.

Case Reports

Case 1: RSH with RCH after Supratentorial craniotomy for intra-axial low-grade glioma

A 48-year-old male presented to neurosurgical OPD at Yashoda hospital, Secunderabad with left sided focal motor seizure with sequential tonic posturing of left lower limb followed by left upper limb and face with post ictal Todd's palsy without any prodrome, aura or postictal residual symptoms. Based on semeiology a diagnosis of focal aware seizure localizing to right precentral gyrus was made. Patient was evaluated with MRI brain which revealed an intra-axial mass lesion Hypointense on T1WI, Hyperintense on T2WI, with no suppression in inversion recovery sequence and no enhancement on post gadolinium imaging located in medial part of motor cortex and extending anteriorly to Supplementary motor area (Figure 1A-1D). suggestive of Low-Grade Glial lesion. As lesion was in eloquent area functional MRI was done which revealed BOLD signals of left lower limb in a thin cortical rim medial to the lesion (Figure 2). There was no predisposing factor for POH in preanesthetic check-up (i.e., no history of coagulopathy with platelets counts of 1.8 lakhs and normal PT/APTT/INR, normal hepatic or renal dysfunction, no h/o postural headaches or other symptoms of intracranial hypotension and no perioperative use of antiplatelets or anticoagulants).

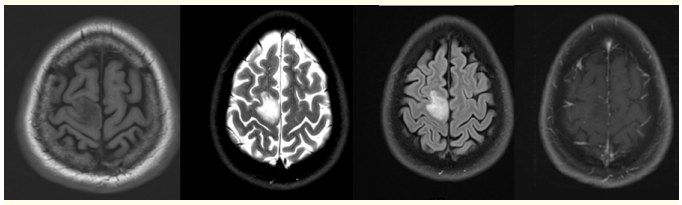

Figure 1: Intraaxial lesion hypointense on T1 (A), Hyperintense on T2(B), no inversion on FLAIR (C), no enhancement on contrast scan (D) suggestive of low grade glial lesion.

Figure 2: Left lower limb signals seen medial to lesion on Functional MRI seen as BOLD signals (blue arrows).

Patient underwent intraoperative neural monitoring guided Awake craniotomy with patient lying supine and head fixed on Mayfield three pin skull clamp in neutral position for maximal safe resection of lesion.

Intraoperatively patient developed right lower limb paresis. The margins of resection cavity were stimulated with bipolar stimulator and motor potential was recorded in posterior aspect of cavity using 4mA current. Cavity was irrigated with warm saline and further resection was not attempted. Total operative time was 5 hours and 30 minutes with good control of perioperative blood pressure. Postoperative Noncontrast CT scan of brain was performed which revealed bilateral curvilinear cerebellar haemorrhages with intervening hypodense normal cerebellar folia (Figure 3A) along with distant supratentorial small specks of bleed in virgin cortex peripheral to resection cavity outside margins of our craniotomy (Figure 3B and 3C). Postoperatively patient recovered from right hemiparesis by day 3 and there were no deficits localising to the site of RSH and RCH.

Figure 3: Curvilinear hemorrhages in bilateral cerebellar folia (A), supratentorial hemmorhages in virgin subcortical white matter peripheral to resection cavity (B and C).

Case 2: RCH after supratentorial craniotomy for sphenoid wing meningioma

45yrs old lady visited to neurosurgery OPD at Medical Trust Hospital, Kochi with history of one episode generalised tonic clonic seizure. She was evaluated with CT brain which showed a moderate sized hyper dense mass on left sphenoid area with mass effect. MRI with contrast was done which was suggestive of left sided sphenoid wing meningioma (Figure 4). There was no predisposing factor for POH in preanesthetic check-up (as described in case 1) and Patient underwent left fronto-temporal craniotomy with head fixed on Mayfield skull clamps rotated 20-25 degrees to right with mild extension with malar prominences being the highest point for excision of meningioma. Intraoperatively arachnoid plane was maintained all around except for medial aspect where the plane was ill defined, which led to cerebrospinal fluid (CSF) leak. Basal dura was infiltrated with tumour which was coagulated and dura was reconstructed with artificial Dural graft and a Simpson's grade 2 excision of meningioma was achieved. Total operative time was 4 hours with good control of perioperative blood pressure. Postoperatively MRI with contrast showed no residue but to our surprise we noticed multiple haemorrhaging foci at superior aspect of cerebellum (Figure 5). Patient had no symptom related to this and showed no neurological worsening and got discharged on post op day 5.

Figure 4: (A, B): Axial and coronal post contrast MRI scans showing Well defined extraxial lesion noted in left fronto-temporal region with homogenous contrast enhancement with Dural tail suggestive of Lateral sphenoid wing meningioma.

Figure 5: Postoperative day 1 scan (A) showing total excision of meningioma, (B) showing remote cerebellar haemorrhage as curvilinear blooming in cerebellar folia on susceptibility weighted imaging.

Discussion

Remote haemorrhages are rare post neurosurgical complication. Though the term sounds new to many, it was reported as early as in 1937 by Van gehuchan., *et al*. and later demonstrated by Konig., *et al*. in 1988 [5]. They are most commonly observed following supratentorial craniotomies done for aneurysm and temporal lobectomies [6] but can be seen after spinal surgeries too.

RCH is the most common pattern of remote POH. They usually present in wide variety of ways, most commonly as asymptomatic patients with incidentally diagnosed haemorrhage on postopera-

tive CT/MRI, others may present with mild to moderate symptoms like prolonged awakening from anaesthesia, postural headache, dizziness, pendular tremors, gait ataxia and decreased consciousness [7]. Sometimes, stormy postoperative event like sudden fall in neurological score, apnoea and even death can occur. Vast number of theories has been put forth by various authors regarding pathophysiology of RCH. Most popular is "Cerebellar sag theory" which states that loss of CSF during surgery allows cerebellum to gravitate away from tentorium and cause stretching, occlusion and tearing of superior vermian and superior cerebellar veins causing venous infarction and overall rise in transmural venous pressure triggers its rupture and bleed [8]. Other theories suggest Transient hypertension during immediate post-operative period with procedure like extubation can increase the gradient between intravascular and CSF pressure, resulting into cerebellar sagging and occluding the superior bridging vein within the posterior fossa, thus haemorrhagic venous infarct [6,9,10]; Even primary positioning of patient is important, as extreme rotation can lead to jugular vein occlusion and increases the Transmural pressure in superior vermian vein and leads to haemorrhage. "Drain theory" states that Postoperative drain in suction leads to loss of CSF volume and creating intracranial hypotensive state ultimately leads to cerebellar sagging and rupture of vein leading to haemorrhage [5]. Superior cerebellar artery occlusion as result of compression due to brain shift during surgery leading to infarct with haemorrhagic transformation can be a cause of RCH [11]. "Coagulation theory" by Konig., *et al*. states that low dose heparin in post-operative period can lead to new haemorrhages. But this theory has fallen out of favour, as it should be the intervention site bleed rather than remote bleed [5,12]. Characteristic sign of RCH noted on imaging is "ZEBRA sign" which refers to high attenuation curvilinear haemorrhage between the low attenuation cerebellar folia, usually it is bilateral and in subarachnoid space, also pure parenchymal bleed also been reported [13]. Management of such cases is tailored according to presentation. Asymptomatic and mildly symptomatic patients can be observed clinicoradiologically while patients with massive bleeds with mass effect over 4th ventricle and brainstem compression may require urgent decompression and evacuation.

RSH on the other hand is a very rare pattern of remote POH. In scarce literature, it is most often seen after infratentorial craniotomies and events of RSH after supratentorial craniotomies is extremely rare. Amongst the speculated theories regarding its pathophysiology, surgery in sitting position is most accepted; sitting position can exacerbate CSF and blood loss which results in dramatic intraoperative brain shift and stretching of cortical draining veins which results in venous infarct and subdural haemorrhages. Sitting position can also result in intraoperative ischemia due to reduced arterial perfusion which can result in hyperperfusion haemorrhages in postoperative period once the patient is bought back to supine position [14]. Other plausible mechanism can be preoperative chronic raised ICP with sudden rapid decompression intraoperatively along with dysautoregulation can result in venous pressure surge and haemorrhage [15]. Radiological diagnosis and management of RSH follows the same algorithm as of intracranial haemorrhage (ICH) and depends upon symptoms of patient, size and location of hematoma with associated stage of intracranial herniations.

Lesson learnt from our cases

To the best of author's knowledge Case 1 is the first in world literature that shows a combined postoperative intraparenchymal punctate RSH along with RCH. Rapid decompression of intraaxial lesion might be a cause for remote POH. Due to slow growing LGG the surrounding microvenules might be under chronic compression and thus might have lost the power to autoregulate in response to gush of blood coming after decompression of lesion resulting in small punctate bleed in normal parenchyma around surgical site. Another plausible cause can be sudden decompression of tumour with collapse of cavity which can cause snapping of microvenules in the surrounding parenchyma and resultant RSH. RCH also can be explained venous compressions secondary to brain shift.

In Case 2 patient was operated on a horseshoe instead of a stable 3 pin device due to logistical reasons which may have resulted in excessive neck rotation and reduced jugular outflow, along with intraoperative arachnoid breach and coagulation of basal dura that lead to postoperative CSF loss in suction drain may have resulted in postoperative RCH.

Conclusion

Remote POH is usually benign but sometimes potential life-threatening complication seen after neurosurgical procedures. Most commonly due to venous compressions secondary to intraoperative brain shifts and CSF loss, surgical positioning and excessive CSF drainage in postoperative periods. The treatment depends

on patients' symptoms and can range from mere observation to need for emergency decompression. Thus, a differential diagnosis of remote POH should always be kept in mind in postoperative neurosurgical patients who develop symptoms unrelated to site of surgery.

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