The efficacy of SURGIFLO® in management of sub-arachnoid hemorrhage after cerebello-pontine (CP) angle schwannoma surgery- A Case report

Case Report

Kunal Kumar Sharma[1], Prashant Dhatwalia[2], Biplav Singh[3], Suresh Kumar Thakur[4]

Department of [1]Neuroanesthesia cell, [2]Anesthesia, [3]Neurosurgery, [4]Radiodiagnosis Neuroradiology Cell, Indira Gandhi Medical college and hospital, Shimla, Himachal Pradesh, India

ABSTRACT

Background: Subarachnoid hemorrhage (SAH) as a complication after cerebello-pontine (CP) angle surgery has rarely been described in literature. We intended to cite the preceding scenario leading to this event and its successful management based on anesthetic and surgical directed approach.

Case presentation: A middle aged female patient presenting with complaints of hearing loss, vertigo and swaying gait underwent surgery for cystic vestibular schwannoma under cranial nerve monitoring. The anesthetic maintenance was solely based on entropy guided propofol infusion. Intra-operatively the patient suffered hemorrhage during the tumor decompression, which was eventually stopped after usage of SURGIFLO. Post-operatively, the neuro-imaging revealed SAH. It was managed in ICU on the lines of prevention of vasospasm and patient was subsequently discharged from the institute.

Conclusions: SURGIFLO is effective in control of intra-operative grade 3 bleed. Nimodipine usage is indispensible for management of vasospasm. Maintenance of hemodynamics during the crisis plays critical role towards a good outcome of the patient following the surgery.

Key Words: Case report, CP angle schwannoma, nimodipine, SAH, SURGIFLO, vasospasm.

Received: 22 November 2023**, Accepted:** 22 January 2024

Corresponding Author: Kunal Kumar Sharma, MD, Department of Neuroanesthesia Cell, Indira Gandhi Medical College and Hospital, Shimla, Himachal Pradesh, India, **E-mail:** kunaal_kumar@yahoo.com .

ISSN: 2090-925X, 2024, Vol.16, No. 2

BACKGROUND

Cerebello-pontine (CP) angle lesions present as a syndrome consequent of mass effect in the CP cistern[1]. During the intracanalicular stage, the symptoms progress from high frequency sensorineural hearing loss, tinnitus, vertigo and disequilibrium. Once the tumor grows out from the porus acusticus to 2 cm or more in size, the auditory symptoms worsen and facial hypoesthesia starts alongwith headache, due to trigeminal nerve compression. Thereafter, the CP cistern starts to get filled, this leads to compression of brainstem, facial nerve, cerebellum and lower cranial nerves. Finally, the lesion will cause shift of fourth ventricle. This leads to obstructive hydrocephalus and subsequent features of raised ICP. Sub-arachnoid hemorrhage (SAH) as a complication of CP angle surgery has rarely been described in literature[2]. If appropriate management is not undertaken, it will result in postoperative vasospasm and neurological deficits. In the following case we describe the successful management of this chaos.

CASE PRESENTATION

This case report adheres to the CARE checklist. The patient was a 63 year old female, who presented with the chief complaints of - (i) impaired hearing on right ear for 7 months; (ii) vertigo for 18-20 days; (iii) unsteadiness in gait for 16 - 18 days, without any diurnal variation; (iv) nausea. Her physical examination revealed gaze evoked horizontal nystagmus towards right. Dix-Hallpike test revealed bilateral vertigo. Rhomberg sign was also present. Weber test lateralized to left ear, and the Rinne test was positive (normal) on both sides. Therefore she had sensorineural hearing loss (SNHL) on right side. The pre-operative facial nerve examination revealed an assessment of House-Brackman [3] grade 1 (Table 1). Examination of visual acuity revealed vision to be 6/24 on right side and 6/36 on left side with aid which improved to 6/18 and 6/24 on respective sides, with the use of pin hole. On fundus examination, clear media with optic cup : disc ratio was 0.4-0.5 was seen on right side, in contrast to hazy media

DOI: 10.21608/ASJA.2024.250457.1011

with ratio 0.5 on left side. The finger to nose test and the heel shin test were impaired on right side. Alogwith impaired dysdiadochokinesia on the right side. The patient had a swaying, broad based gait. Examination of sensory system only revealed impairment in proprioception.

The magnetic resonance image (MRI) revealed post-contrast altered signal intensity with lesion at right CP angle of 2.6 x 5 x 4.5 cm being hypointense on T1 and hyperintense on T2 image with incomplete suppression on fluid attenuated inversion recovery sequence (FLAIR) Figure 1. The diffusion weighted image (DWI) sequence revealed solid components exhibiting diffusion restriction. On susceptibility weighted image (SWI) sequence, multiple internal foci suggestive of micro hemorrhages with calcified foci were observed. The lesion was abutting and displacing right middle cerebellar peduncle, pons, medulla, right cerebellar hemisphere and basilar artery to contralateral side. This led to effacement of fourth ventricle thereby causing an upstream dilatation. The cerebral angiography revealed bilateral posterior cerebral artery (PCA) and basilar arteries to be normal.

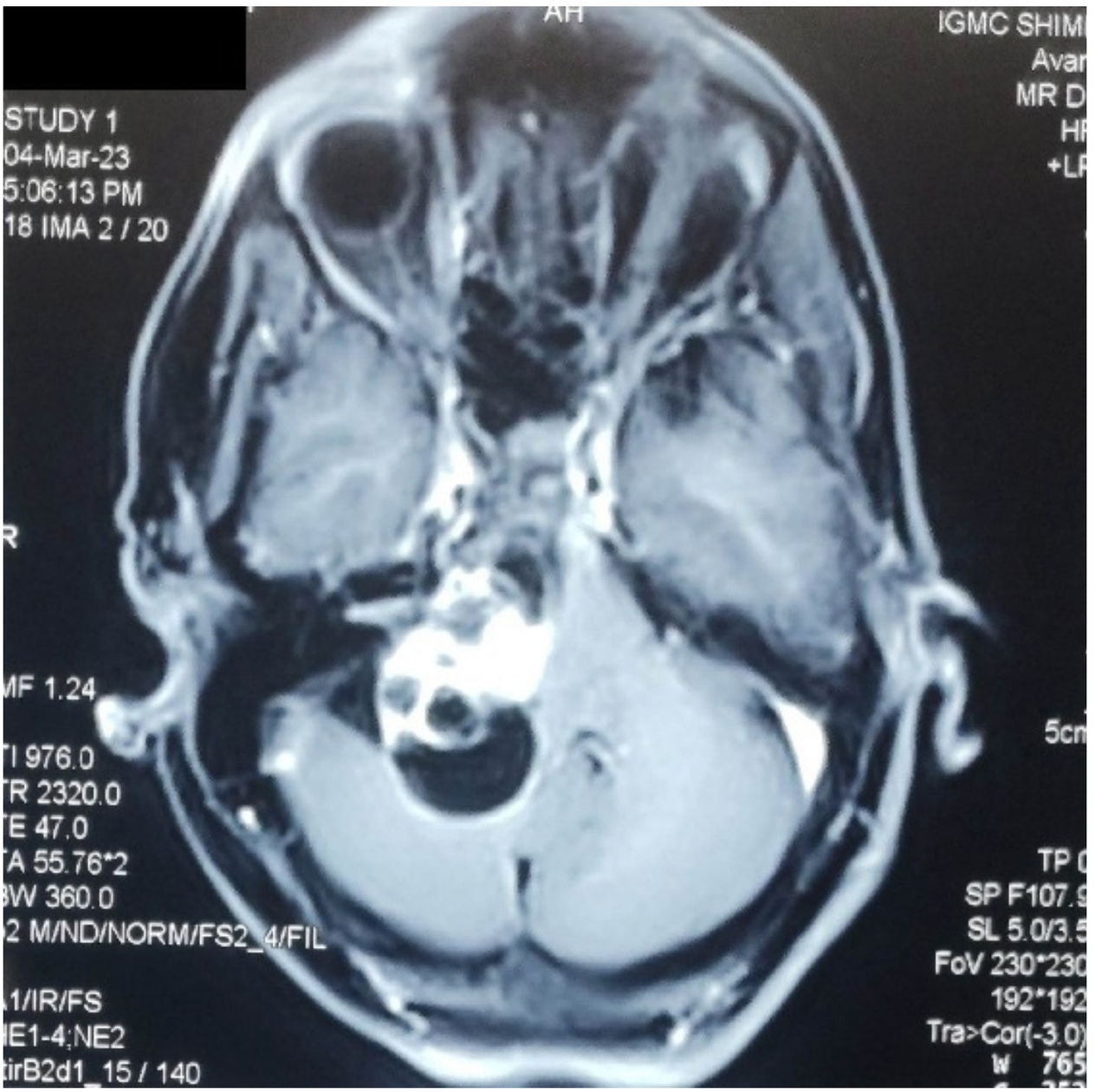

Figure 1: T2 axial scan depicting vestibular schwannoma at right CP angle.

The patient was scheduled for right retro-mastoid sub occipital craniotomy (RMSOC) and resection of the lesion under facial, glossopharyngeal, hypoglossal nerve monitoring. After securing adequate venous access, the patient underwent a stable anesthetic induction with fentanyl 120 µg iv, propofol 160 mg iv, lidocard 60 mg iv. This was followed by endotracheal intubation and placement of Entropy sensor on forehead. Facial nerve, trigeminal nerve, glossopharyngeal nerve and hypoglossal nerve monitoring was done using the medtronic nerve integrity monitoring system by subdermal needle electrodes placed at orbicularis oculi, orbicularis oris, masseter, mentalis, tounge and soft palate Figure 2. A stimulus intensity of 0.05 mA resulted in response amplitude of 260 µV. Therefore in order to avoid any detrimetal effect of anesthetics on latency and amplitude of evoked potentials, the maintenance of anesthesia was done using Oxygen and Air (50:50) mixture alongwith propofol infusion and intermittent boluses of fentanyl.

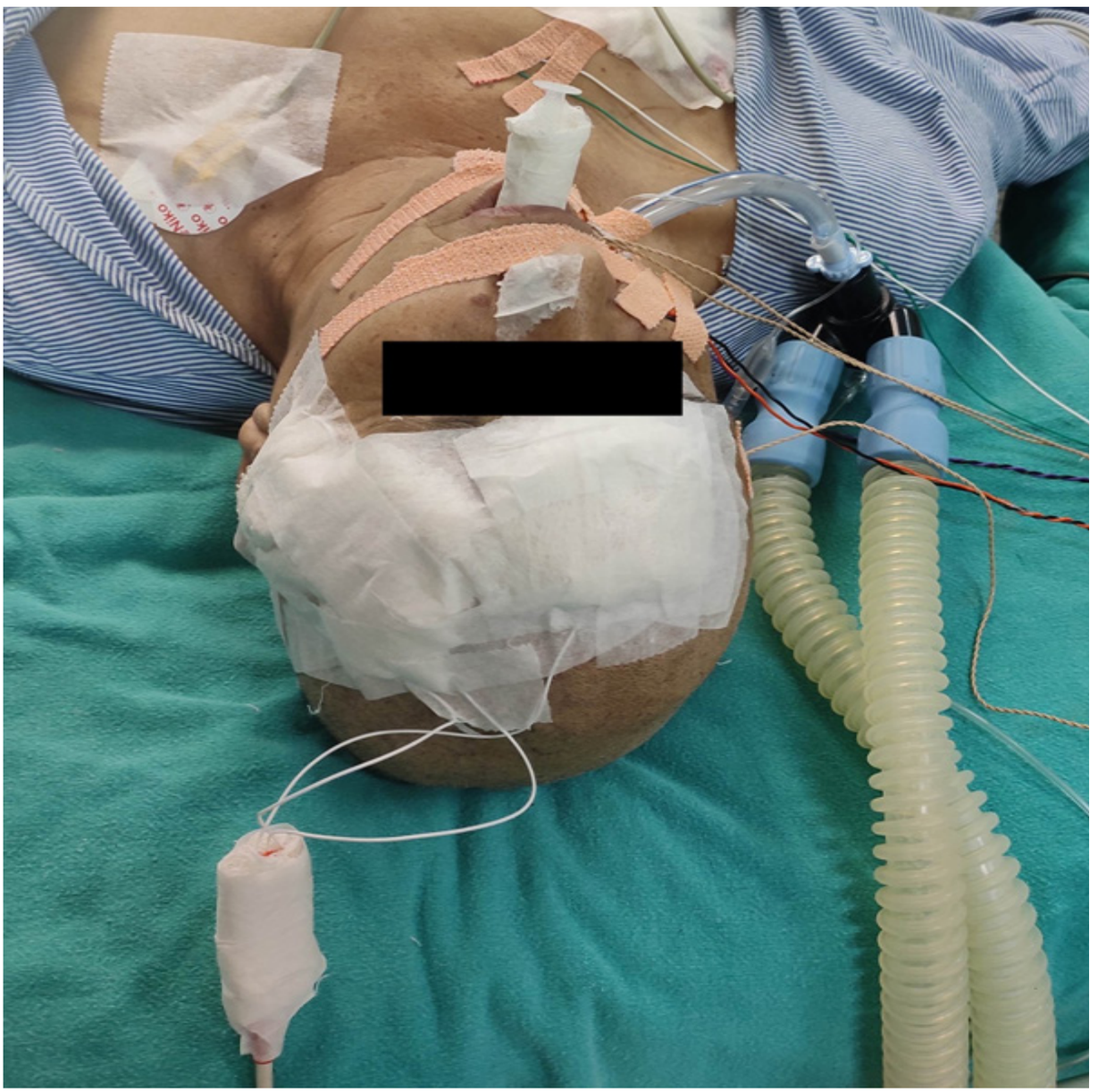

Figure 2: T2 Needle electrodes placed at the facial muscles for intraoperative neuromonitoring.

The patient was positioned in park bench position due to better hemodynamic stability in this position and lesser incidence of air embolism. Mayfield pins were applied. A retromastoid suboccipital incision from right mastoid to inion was made. An external ventricular drain (EVD) was placed via right Frazier's point by neurosurgeon in order to drain 40 ml of cerebrospinal fluid (CSF). The flap was raised in layers and retromastoid sub-occipital craniotomy was done. The dura was incised and adequate exposure was observed. Right cerebellar bulge was noted, therefore the right CP angle was approached from medial side with traction of cerebellar lobe. The greyish capsule of the cystic component of schwannoma was pierced and the accumulated collection was drained. The cerebellar-medullary cistern was opened and CSF was drained. Intraoperatively, the surgeon had to constantly apply suction in order to suck out blood and CSF. On visual examination, the solid component of schwannoma was pearly white, highly vascular, ovoid in shape and well delineated from surrounding parenchyma. It was approached using cavitatory ultrasonic aspirator (CUSA). This decompresssion of the schwannoma was stopped when a sudden spurt of blood was noticed. This resulted in blood loss of around 900 ml, it was of grade 3 as per the Lewis blood loss assessment scale[4] Table 2. Since the conventional methods of hemostasis had failed, the team decided to use SURGIFLO® haemostatic matrix. The most likely cause of the bleed was breach in the petrosal vein. This bleed decreased to grade 1 after the application of SURGIFLO® Figure 3. The hemodynamics during this period were managed with crystalloids and packed red blood cells. The neurosurgeons performed a subtotal resection and partial cerebellar lobectomy. Duroplasty was done using occipital galeal flap. The wound was closed in layers. The EVD was connected to the bag and antiseptic dressing was done. The patient was shifted to ICU. Vancomycin and metronidazole were started empirically by the neurosurgeons after the end of surgery and continued for 15 days and 9 days respectively. The patient was kept sedated overnight under propofol infusion. In morning, the sedation was turned off for patient assessment.

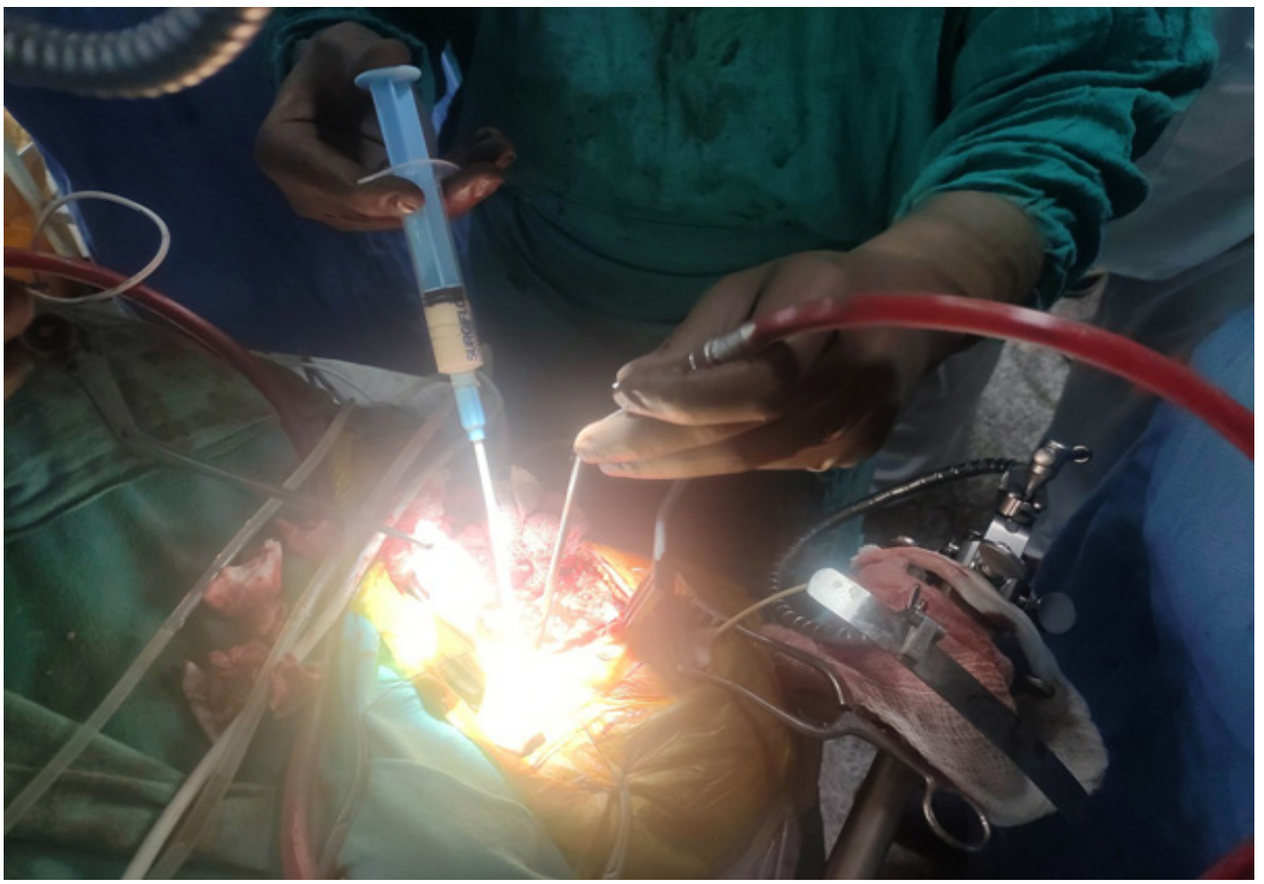

Figure 3: Application of SURGIFLO for control of bleed.

The postoperative CT brain revealed SAH in the basal cisterns Figure 4, therefore she was started on oral nimodipine 60 mg every 4 hours in view of preventing vasospasm. Subsequent neuroimaging and clinical examination did not reveal any deterioration of neurological status further. Therefore she was extubated and shifted out from the ICU to neurosurgery ward. On postoperative day 6, she developed obstructive hydrocephalus for which left medium pressure ventriculo-peritoneal (MPVP) shunt was placed via Keen point. The surgery was uneventful and the subsequent post-operative period was uneventful. The patient discharged from the institute after 20 days with a GCS of 15 and no limb deficits.

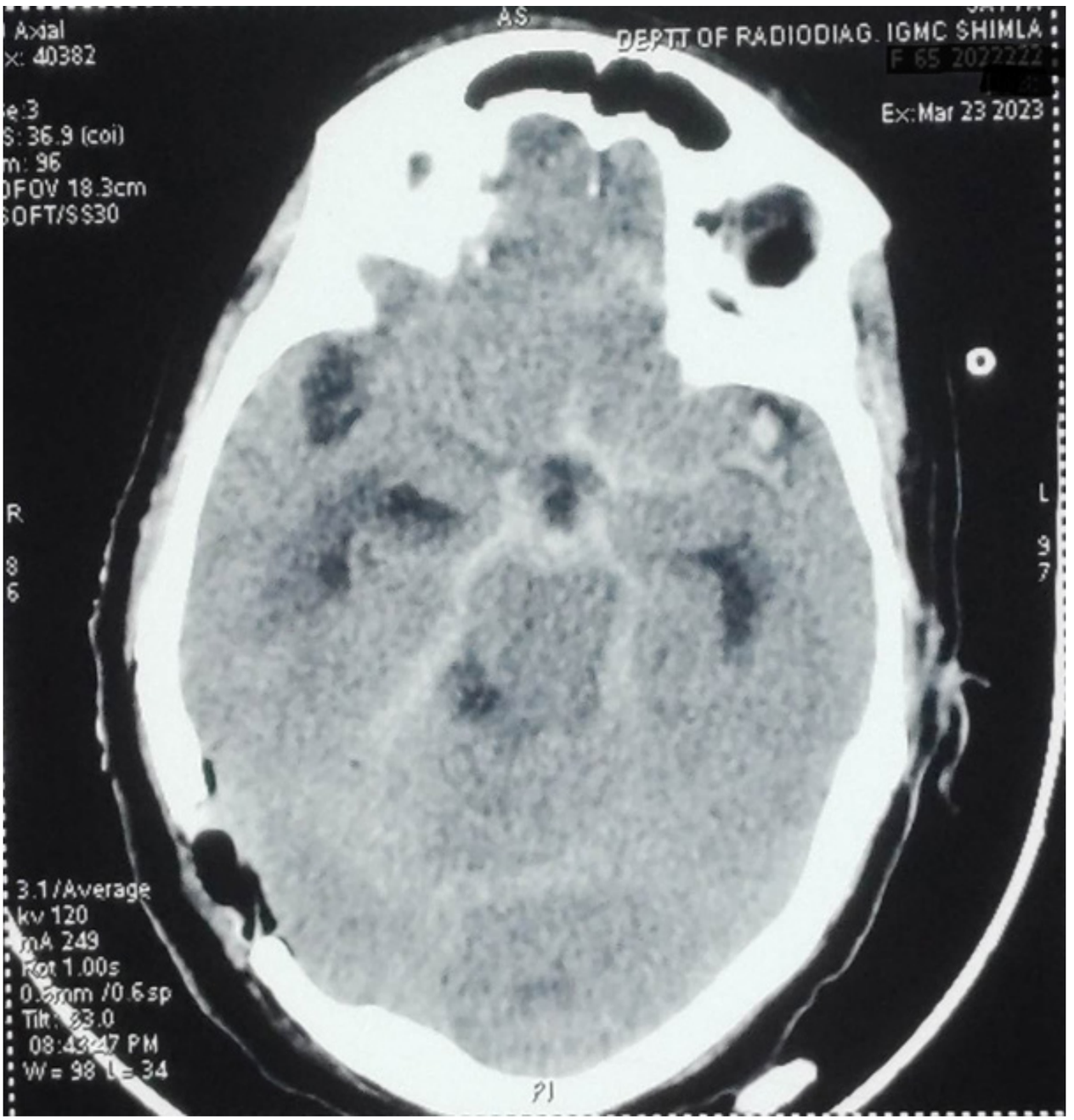

Figure 4: Post-operative axial CT brain slice depicting SAH.

Discussion: Postoperative SAH after CP angle surgery has been rarely described in litreature. In an analysis by Lazard *et al.*,[5] of 72 patients, the authors noted thrombo-embolic complications in 2 patients, hematomas at CP angle in 3 % of operated patients, meningitis in 8 patients, CSF leaks in 12 patients, distended cutaneous flaps in 38 cases. But they did not find any complication of SAH in their analysis.

An incidentally detected aneurysmal rupture in a patient scheduled for vestibular schwannoma resection, poses its own set of catastrophic implications. SAH can lead to reduction in cerebral blood flow (CBF < 250 ml/min ~ 18 ml/100g/min), whereby global or regional cerebral ischemia develops. The cerebral arterial oxygen supply (DaO_2) is inter-twined with the provision of nutrient to the neurons vide the equation $DaO_2 = CaO_2$ x CBF; where CaO_2 is the oxygen content in the arterial blood. The cerebral ischemia leads to subsequent cessation of glial and neuronal cell function[6]. The EEG becomes isoelectric[7] at CBF of ~16 ml/100g/min, whereas the evoked potentials lapse[8] at a CBF of ~12 ml/100g/min. Therefore it becomes imperative to maintain the optimal cerebral perfusion during this crisis scenario. However, the pre-operative MR angiography of the present case had not revealed any presence of posterior circulation aneurysm. Therefore it could not have been the cause of SAH in the present case.

The works of Duvernoy[9] have described the venous drainage of the cerebellum from superior and inferior cerebellar surfaces via vermian and hemispheric veins. The superior vermian veins thereafter drain into pre-central vein, great cerebral veins and confluens sinuum. The inferior vermian veins drain into the confluens sinuum. The superior hemispheric veins reach the tentorium cerebelli and drain into transverse and superior petrosal sinuses. The inferior hemispheric veins drain into the transverse sinus. The veins on the anterior cerebellar surface drain into the anterior cerebellar vein, which via the superior petrosal vein drains into the superior petrosal sinus. Its rupture during the decompression of the tumor was the most likely cause of hemorrhage. The trickling of this blood into the fourth ventricle resulted in its spread subsequently to the third ventricle and the basal cisterns, and it even extended up to bilateral sylvian fissures. Fortunately, this bleed did not spread further into the surface of brain and cortical sulci.

This bleed was stopped with SURGIFLO® hemostatic matrix, which is a topical hemostatic agent that contains thrombin and porcine derived gelatin granules. On its application at the area of bleeding, the granules swell up and shield blood flow. This provides a stable matrix via tamponade effect[10]. Compounded thrombin then facilitates forming a fibrin clot around the matrix that results in hemostasis. SURGIFLO® itself can cause infection, thrombosis and even postoperative hemorrhage[11]. However, such issues were not observed with its usage in the present case, because its application only reduced the grade of bleed from 3 to zero. Postoperatively, the patient was afebrile. Her postoperative WBC count was 15800 /µl which resolved to 12600 /µl over next 9 days.

We addressed the concern of vasospasm by prescribing oral nimodipine 60 mg every 4th hourly. Nimodipine is a second generation 1, 4-dihydropyridine calcium channel blocker, which relives the vasospasm by blockade of voltage gated calcium channels resulting in dilatatory effect on arterial smooth muscle, thereby reliving vasospasm. It is the only FDA approved agent and has a half life of 9 hours. The meta-analysis by Hao *et al.*,[12] of thirteen randomized clinical trials and 1727 patients, observed that nimodipine significantly decreased poor outcome, mortality and cerebrovascular stroke. Mackenzie *et al.*,[13] had proposed a dosage regimen of 30 mg oral nimodipine every 2 hourly in view of patients who were hypotensive. The works of Ott *et al.*,[14] showed that intra-arterial nimodipine infusion is efficacious and safe treatment for symptomatic CVS. However, our patient was already on the improving trend, therefore initiating this treatment was not deemed necessary for the current case. Likewise, the usage of milrinone, fasudil, clazosentan, magnesium or atorvastatin was not deemed necessary for the current case as she had already improved on oral nimodipine.

Hydrocephalus develops due to the combined effect of silting of blood cells after inter-ventricular hemorrhage (IVH) into the choroidal plexus epithelium and inflammatory state of choroidal plexus after IVH and surgery[15, 16]. This alters the normal physiology of the cilia, thereby impeding the absorption of CSF and leading to hydrocephalus. The obstructive hydrocephalus in the present cased was managed by MPVP shunt insertion via Keen point. No further complications were observed, therefore the patient was discharged from the institute in a stable condition.

CONCLUSIONS

Incidental hemorrhage is a vexing issue during neurosurgeries. SAH carries the dread of post-operative vasospasm. The usage of SURGIFLO® was life saving intervention in this case as other conventional means did not suffice. Nimodipine usage is definitely an indispensible arrow in the quiver of the neurointensivist.

LIST OF ABBREVIATIONS

- **CaO_2:** arterial blood oxygen.
- **CARE:** CAse REport.

- **CBF:** Cerebral blood flow.
- **cm:** centimeter.
- **CP:** Cerebello-pontine.
- **CUSA:** cavitatory ultrasonic surgical aspirator.
- **DaO_2:** cerebral arterial oxygen.
- **DWI:** Diffusion weighted imaging.
- **EEG:** electro-encephalogram.
- **FDA:** Food development authority.
- **FLAIR:** Fluid attenuation inversion recovery.
- **g:** gram.
- **ICU:** Intensive care unit.
- **iv:** intravenous.
- **IVH:** Inter-ventricular hemorrhage.
- **mg:** milligram.
- **min:** minute.
- **ml:** milliliter.
- **MPVP:** medium pressure ventriculo-peritoneal.
- **PCA:** Posterior cerebral artery.
- **SAH:** Subarachnoid hemorrhage.
- **SNHL:** sensorineural hearing loss.
- **SWI:** Susceptibility weighted imaging.
- **T1:** longitudinal relaxation time.
- **T2:** transverse relaxation time.
- **µg:** micro gram.
- **µV:** micro volt.
- **WBC:** white blood cells.

CONFLICTS OF INTEREST

There are no conflicts of interest.

REFERENCES

1. Bingas B, Cotsou S. Cerebello-pontine angle-syndrome with uncommon aetiology (a case report). Neuroradiology.1972;3:165-6.
2. Memari F, Hassannia F, Abtahi SH. Surgical Outcomes of Cerebellopontine angle Tumors in 50 Cases. Iran J Otorhinolaryngol. 2015;27(78):29-34.
3. House JW, Brackmann DE. Facial nerve grading system. Otolaryngol Head Neck Surg. 1985;93(2):146-7. doi: 10.1177/019459988509300202. PMID: 3921901.
4. Lewis KM, Li Q, Jones DS, Corrales JD, Du H, Spiess PE, et al. Development and validation of an intraoperative bleeding severity scale for use in clinical studies of hemostatic agents. Surgery 2017;161:771-81.
5. Lazard D.S, Tosello M., Bozorg-Grayeli A. et al. Early complications and symptoms of cerebellopontine angle tumor surgery: a prospective analysis. Eur Arch Otorhinolaryngol 2011;268:1575–1582.
6. Lee RHC, Lee MHH, Wu CYC, Couto E Silva A, Possoit HE, Hsieh TH, Minagar A, Lin HW. Cerebral ischemia and neuroregeneration. Neural Regen Res. 2018;13(3):373-385.
7. Foreman B, Claassen J. Quantitative EEG for the detection of brain ischemia. Crit Care. 2012 (12);16(2):216.
8. Baron JC. Perfusion thresholds in human cerebral ischemia: historical perspective and therapeutic implications. Cerebrovasc Dis. 2001;11(1):2-8.
9. Duvernoy HM. The human brain stem and cerebellum: surface, structure, vascularization and three-dimensional sectional anatomy with MRI. Springer, New York 1995.
10. Vyas KS, Saha SP. Comparison of hemostatic agents used in vascular surgery. Expert Opin Biol Ther. 2013;13(12):1663-1672.
11. Danker W 3rd, Aggarwal J, Kelkar SS, Marston XL, Gao X, Johnston SS. Real-World Clinical and Economic Outcomes Associated with Surgiflo® vs Floseal in Cardiovascular Surgeries in the US. Clinicoecon Outcomes Res. 2022;14:129-138.
12. Hao G, Chu G, Pan P, Han Y, Ai Y, Shi Z, Liang G. Clinical effectiveness of nimodipine for the prevention

of poor outcome after aneurysmal subarachnoid hemorrhage: A systematic review and meta-analysis. Front Neurol. 2022;13:982498.

13. MacKenzie M, Gorman SK, Doucette S, Green R. Incidence of and factors associated with manipulation of nimodipine dosage in patients with aneurysmal subarachnoid hemorrhage. Can J Hosp Pharm. 2014;67(5):358-65.

14. Ott S, Jedlicka S, Wolf S, Peter M, Pudenz C, Merker P, Schürer L, Lumenta CB. Continuous selective intra-arterial application of nimodipine in refractory cerebral vasospasm due to aneurysmal subarachnoid hemorrhage. Biomed Res Int. 2014;2014:970741.

15. Chen S, Luo J, Reis C, Manaenko A, Zhang J. Hydrocephalus after Subarachnoid Hemorrhage: Pathophysiology, Diagnosis, and Treatment. Biomed Res Int. 2017;2017:8584753.

16. Yang Y, He J, Wang Y, Wang C, Tan C, Liao J, Tong L, Xiao G. Targeting choroid plexus epithelium as a novel therapeutic strategy for hydrocephalus. J Neuroinflammation. 2022;19(1):156.