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Managing cerebrospinal fluid leak and its sequential dynamic morbidities after acoustic neuroma surgery: What can we learn?

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Abstract

Acoustic neuroma surgery has a significant morbidity rate despite its relatively low mortality rate. We want to share our unique experience in managing a 55-year-old male who experienced multiple consecutive complications such as recurrent cerebrospinal fluid otorrhea, tension pneumocephalus, dural venous sinus thrombosis, intracranial haemorrhage and extensive deep venous thrombosis after acoustic neuroma resection via translabyrinthine approach. Nonetheless, the patient recovered well with no residual deficit and no evidence of tumour recurrence. Herein, we reviewed the related literature on acoustic neuroma surgeries and postoperative complications. We elucidate the underlying pathophysiology and the possible relations with our case. We also underline the important lessons that could be learnt from this complicated case.

Keywords

acoustic neuroma; venous thrombosis; cerebrospinal fluid; hearing loss; magnetic resonance imaging, computed tomography.

Introduction

Acoustic neuroma (AN) is a benign intracranial, extra-axial tumour commonly arises from the Schwann cell sheath of the eighth cranial nerve vestibular division. It is a slow-growing tumour that accounts for 80% of cerebellopontine angle (CPA) tumours. Management of AN can be conservative such as watchful sequential monitoring with magnetic resonance imaging (MRI), stereotactic radiation therapy or surgical excision. The indications for surgical excision are a large size tumour, a rapidly growing tumour with mass effect, and a tumour in a young patient who is considered for surgical hearing preservation surgery [1]. Three common surgical approaches are via translabyrinth, retrosigmoid and middle cranial fossa.

Case Report

A 55-year-old male with unknown past medical history was complaining of worsening right-sided tinnitus for three weeks in addition to the pre-existing right-sided hearing loss for two years. The patient neglected the problem until the worsened tinnitus caused a disturbance in his daily life. Pure tone audiometry revealed right severe mixed hearing loss and left mild mixed hearing loss. A gadolinium-enhanced MRI demonstrated a right CPA lesion measuring 19 millimetres (mm) X 18.4 mm X 16 mm with evidence of widened right internal auditory canal (IAC) (Figure 1A,1B).

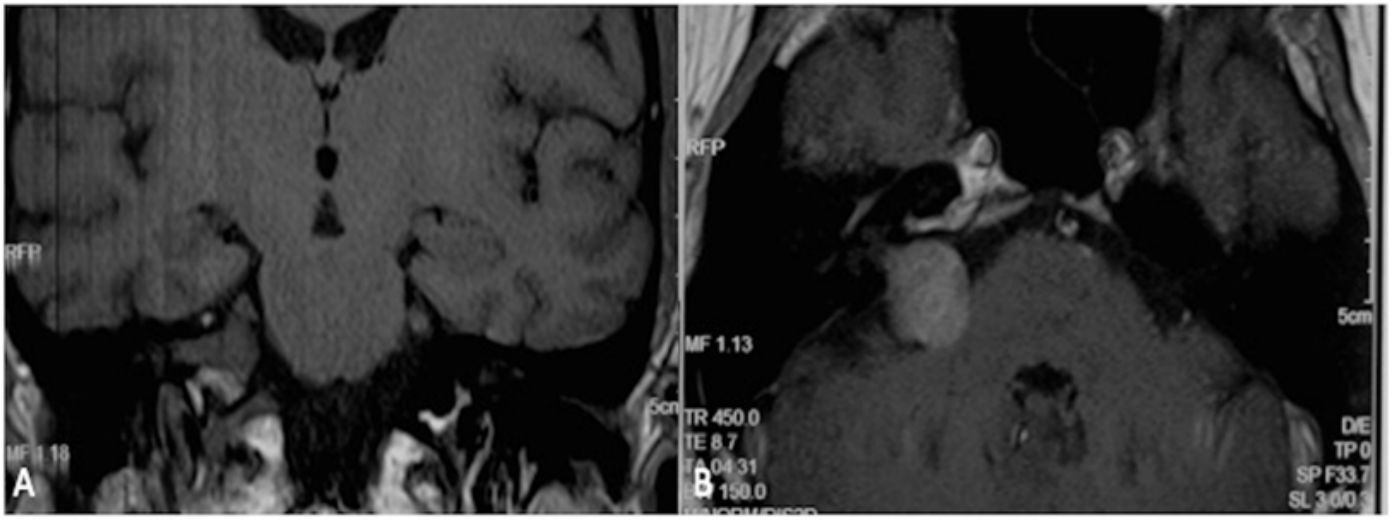

Figure 1: MRI of the cerebellopontine angle (CPA) on coronal T1W (A) and axial T1W post-contrast (B). An enhancing lesion in the right CPA extends into the right internal auditory canal with an expansion of the acoustic meatus consistent with acoustic neuroma.

The patient underwent near-total excision of AN with facial nerve preservation via translabyrinthine approach. A 20 mm X 20 mm tumour was excised, leaving only a few mm due to the adherence to the facial nerve. Temporalis fascia and abdominal fat graft were used to pack the mastoid cavity. The anteriorly based flap was sutured in a watertight manner followed by mastoid bandage application. He was given intravenous (IV) ceftriaxone as a meningitis prophylaxis antibiotic, analgesics, and anti-straining medications postoperatively.

The patient developed right facial nerve palsy grade II (House – Brackmann) and right cerebrospinal fluid (CSF) otorrhea on the second day post-operation. Otherwise, there was no collection at the surgical wound or CSF rhinorrhoea. Conservative treatment was adopted by commencing oral prednisolone 0.5 mg/kg/day, re-application of mastoid bandage and close observation.

After one week of persistent CSF otorrhea, right ear examination under anaesthesia and myringoplasty were performed. The external auditory canal (EAC) was packed with silk strips and gel foam to cover the slit-like perforation on the tympanic membrane. The patient was observed conservatively despite still having recurrent CSF leak.

The patient started to complain of throbbing frontal headache two weeks later. MRI revealed pneumocranium at anterior bilateral frontal lobes, bilateral temporal lobes, interhemispheric fissure, and the basal cisterns. A tract communicating between the right CPA, the right middle ear cavity and EAC indicate the site of CSF leak. Evidence of dural sinus thrombosis (DST) involving right sigmoid, transverse, superior

sagittal sinuses and the right proximal internal jugular vein (IJV) (Figure 2A-2E). The neurology team was consulted, and subcutaneous low molecular weight heparin (LMWH) with oral warfarin bridging was initiated. The patient was kept rest entirely in bed and hydrated with dextrose 5% saline IV drip.

Unfortunately, the patient's condition deteriorated three days after the commencement of the anticoagulant, and he had a sudden loss of consciousness. An urgent computed tomography (CT) brain scan de-

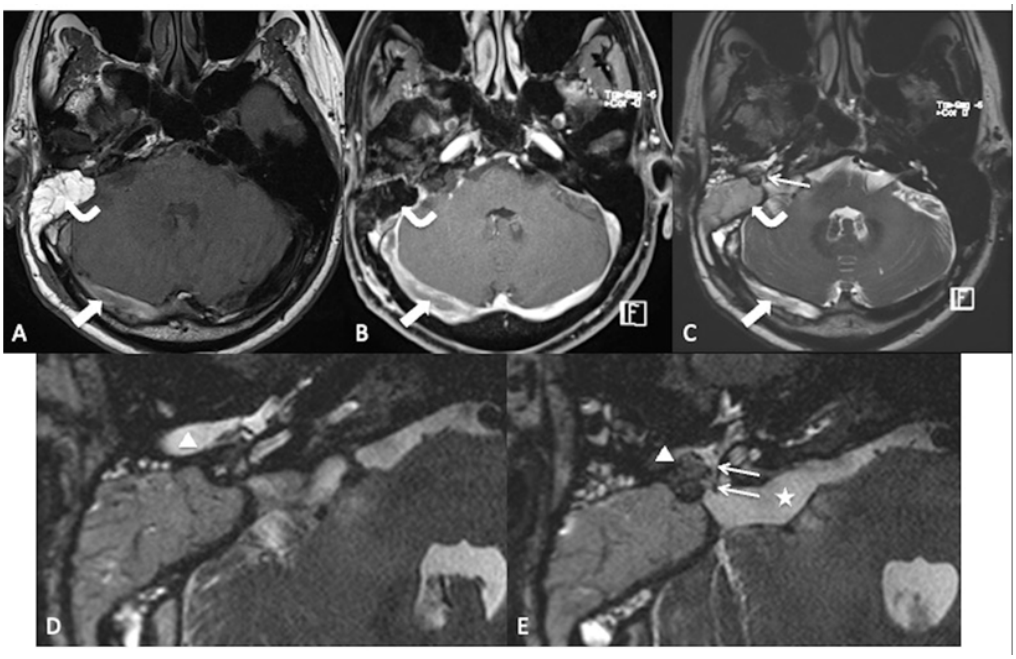

Figure 2: MRI of the brain on T1W (A), T1W fat-saturated with contrast (B), T2W (C) and magnified view of the right CPA on CISS3D (D) & (E). The mastoid cavity is packed with fat (curved block arrow). On the CISS3D, the right external auditory canal and middle ear cavity are fluid-filled (triangle) with fluid tracking posteriorly communicating with the CPA cistern (star) through a small tract (arrow). The right transverse sinus has lost its normal flow void on T2W, with a hyperintense signal on T1W and a filling defect on T1W post contrast consistent with dural sinus thrombosis (block arrow).

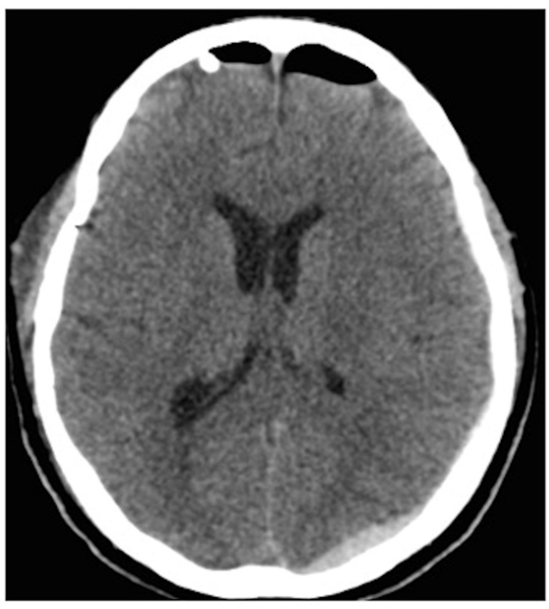

Figure 3: CT brain demonstrates pneumocephalus and left subdural bleed.

monstrated worsening pneumocranium with mass effect on the bilateral frontal lobes with intraventricular extension. A newly developed subdural haemorrhage (SDH) and subarachnoid haemorrhage (SAH) were seen at the frontoparietal region and the left parietal region, respectively (Figure 3). Oral warfarin was immediately withheld. Emergency ventriculocisternostomy, external ventricular drainage (EVD) and right ear packing with Fogarty catheter were performed. Haemorrhagic CSF was observed during right subdural drain insertion. Postoperatively, the patient was kept in cerebral protection for 24 hours.

A repeat CT brain scan on the following day showed reducing bifrontal pneumocephalus with residual SAH. He suffered from persistent CSF otorrhea, requiring mastoid exploration for middle ear and Eustachian tube (ET) packing three weeks post the EVD insertion. Intraoperatively there was a collection of the CSF in the middle ear draining from the root of zygoma air cells. The minor defect from the tegmen was repaired with muscle, tissue glue and fat. Fat was used to pack middle ear and ET. Redo-myringoplasty was performed using the overlay technique and rosette ear packing inserted. The rosette ear packing was removed in the clinic two weeks later, and a 0-degree oto-endoscopy showed the graft in situ.

A contrasted MRI was repeated eight weeks post AN resection demonstrated imperceptible evidence of CSF leak and resolved DST with a small filling defect within the right medial transverse sinus. The CSF otorrhea eventually resolved with complete bed rest and avoidance of straining activities. The initiation of oral warfarin was deferred due to hematoma at the abdominal fat graft donor site. However, the patient developed left leg oedema three weeks later with evidence of deep venous thrombosis (DVT) involving the left external iliac vein, left common femoral vein, left superficial femoral vein and left popliteal vein. The oral warfarin with heparin bridging regime was resumed, and the patient tolerated the medication without any undesired adverse effects.

Finally, after two months of hospitalization, he was discharged with long term oral warfarin and aftercare advice. The oral warfarin was successfully terminated two years later without recurrent DST or DVT. During his routine appointment six years after the surgery, he did well with no recurrence of CSF leak, DVT or residual neurological deficit.

Discussion

Since the first successful surgical removal in 1894 by Sir Charles Ballance, AN surgery has evolved tremendously; the mortality rate has decreased from 80% to less than 0.5% [2-4]. Complete tumour resection, preservation of neurological function and averting postoperative complications serves as the new cornerstone for AN surgery attributed to the advent of operative microscope and the employment of modern neuroanaesthetic practice in neurotology microsurgery. Nevertheless, there are still unavoidable numbers of postoperative morbidities reported in the literature [2-4].

The most-reported complication is CSF leak which may present as rhinorrhea or otorrhea. The incidence ranges from 2.8% to 63% [3-5]. Aznmi MN et al. had proposed a few salient intraoperative steps to prevent the occurrence of postoperative CSF leak. First, by the application of bone wax to the apical and perifascial air cells. Second, by watertight dural closure to separate posterior cranial fossa from the mastoid cavity. Third, by obliteration of the mastoid cavity and ET technique [6]. In our case, all appropriate measures were taken to reduce the risk of postoperative CSF leak. Middle ear access via post tympanotomy and

antrum was secured together with mastoid cavity using a few layers of abdominal fat strips and temporalis fascia. ET obliteration was not a common practice in our centre. Nevertheless, this step can be adopted in refractory cases. [7]

CSF leak is commonly managed with a stepwise approach beginning with conservative treatments, followed by lumbar drainage and finally by surgical repair. The conservative measures include applying the mastoid bandage, bed rest, and avoiding straining activities. Fortunately, most of the patients that received conservative treatment had recovered well [6]. A lumbar drain is an adjunct therapy to conservative treatments in patients with elevated CSF pressure. The drainage must be cautiously monitored not to exceed 20-25 ml/hour (standard CSF production rate) to avoid building a differential pressure across the foramen magnum, leading to transhiatal brain herniation. Surgical repair is reserved for the refractory leak or in the case with a high risk of meningitis [6].

Excessive CSF drainage also may lead to the development of pneumocephalus. However, there is still a paucity of data reporting pneumocephalus as the consequence of CSF otorrhea following AN surgery, especially without lumbar drainage insertion. Violation of the dura lining with pressure gradience between the intracranial space and the extracranial environment are the essential mechanism for the development of pneumocephalus. The leaking dura provides a connection where air may enter the cranium during straining activities or passively from the aerated mastoid ear cells. The air entering the cranium will accumulate, subsequently increasing the intracranial pressure and quickly closing the dural defect, causing air entrapment in the cranium, known as the ball-valve effect [8]. If unaddressed earlier, this condition may progress to tension pneumocephalus [8]. Headache is the most typical presenting symptom of tension pneumocephalus, as in our case, followed by other various symptoms such as loss of consciousness, aphasia, vertigo, visual changes, and paralysis [8,9]. Patients with pneumocephalus should be replenished with adequate hydration to substitute the severe depletion of intracranial fluid. Then, adefinitive surgical decompression such as burr hole or ventriculocisternostomy should take place. In our case, it was quite hard to quantify the flow rate of the CSF leak as the lumbar drain was deemed not required during the initial operative procedure.

DST is a relatively uncommon sequela of CPA tumour surgery. Sigmoid and transverse sinuses are the most frequently affected sinuses due to their proximity to the CPA area [10]. Hence, it is extremely rare to encounter extensive dural venous thrombosis with the IJV involvement following translabyrinthine microsurgery. Shaikh N et al. reported a novel case of multiple dural venous sinuses and right IJV thrombosis caused by tension pneumocephalus following frontal lobe mass tumour resection [9]. The similarity with our case could enlighten us of this unique occasion. It is propounded that increased intracranial pressure by tension pneumocephalus causes an impaired venous flow of the dural sinuses, thus predispose them to thrombosis [9].

The management of DST remains a dilemma despite ubiquitous successful treatment plans proposed in the literature. The variability in the report is likely confounded by the heterogenicity of the causative diseases, patient's health status, physician's competency and institutional facilities in the reporting centres. Brahimaj BC et al. advocated clinical monitoring in asymptomatic patients and reported resolution of the thrombus without initiation of the anticoagulant agent [11]. Conversely, a cohort study by Moore J

et al. manifested a safe and favourable outcome with early initiation of anticoagulant therapy in cerebral venous sinus thrombosis cases [10]. However, after the commencement of systemic anticoagulant, the patients should be cautiously monitored to avoid the unwanted side effects of the treatment and provide a prompt necessary treatment should it happen. A Cochrane study by Coutinho J et al. concluded about 3-6% risk of intracerebral haemorrhage and 3% risk of extracerebral haemorrhage associated with anticoagulant treatment [12]. The haemorrhage further complicates the pre-existing problem and delays further active intervention. Medel R et al. advocated mechanical thrombectomy and local fibrinolysis as adjuncts to systemic anticoagulants for a symptomatic and deteriorating patient [13]. Our patient was initially hydrated to promote cerebral blood flow and circulation. We decided to initiate anticoagulant therapy after considering multiple locations of thrombosis with subsequent vigilant monitoring. Unfortunately, he developed SDH and SAH following anticoagulant treatment.

The peculiar complication in our case is the extensive extracranial venous thrombosis involving the left iliac, femoral and popliteal vein. To our knowledge, this is the novel case report of extensive systemic venous thrombosis following the translabyrinthine approach for AN resection. AN has demonstrated a low procoagulant propensity for venous thromboembolism (VTE) events, thus underscore the above mentioned rarity [14]. The determining risk factors in our patients are the advanced age, the usage of steroids and prolonged bed rest, in addition to the deterrence of the anticoagulant treatment due to the intracranial haemorrhage [14]. A study by Vanek VW showed a 48% reduction of postoperative VTE when intermittent pneumatic compression (IPC) devices are utilized compared to LMWH [15]. Thus, IPC should be considered in patients with high VTE risk but are contraindicated for anticoagulant therapy.

Conclusion

In ensuring a successful and safe acoustic neuroma surgery, profound knowledge with competent surgical skills of the attending surgeons are paramount to reduce risks of postoperative complications and provide the appropriate management should the complications occur. Latent channels of CSF must be identified or secured at the initial setting to reduce the risk of a leak. Intermittent pneumatic compression devices should be considered in patients undergoing major surgery who require complete rest despite a normal body mass index. Multidisciplinary collaboration is equally crucial, especially in the presence of dynamic postoperative complications.

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