

Brainstem Infarction Following Resection of a Cerebellopontine Angle Epidermoid Cyst with Normal Intraoperative Neurophysiological Monitoring: A Case Report

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Case Report

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Abstract

A 33-year-old man with untreated hypertension underwent gross-total resection of a cerebellopontine angle epidermoid cyst that completely encased the bilateral vertebral and basilar arteries. Intraoperative brainstem auditory evoked potentials and somatosensory evoked potentials remained entirely normal throughout the 8-hour-20-minute procedure. Immediately after surgery, the patient developed a fixed, dilated left pupil with absent light reflex. Emergent CT excluded haemorrhage, but subsequent MRI confirmed acute ventral brainstem infarction. This case **demonstrates for the first time that catastrophic ischaemic complications can occur after CPA epidermoid cyst resection despite uneventful intraoperative neurophysiological monitoring**, highlighting a critical blind spot of current monitoring technology and underscoring the essential value of postoperative pupillary examination.

Introduction

Cerebellopontine angle (CPA) epidermoid cysts are rare, slow-growing benign tumours that account for 40–5 % of intracranial epidermoids ^[1, 2]. Although gross-total resection is the standard treatment, the tumour frequently encases the vertebrobasilar system and adheres tightly to the ventral brainstem, rendering surgery technically demanding and associated with substantial morbidity. Reported complication rates include new cranial nerve dysfunction, aseptic meningitis, intracranial infection, cerebrospinal fluid leakage and intracranial haemorrhage. Postoperative brainstem infarction is a rare but devastating complication, with only isolated cases described in the literature ^[3].

We report a CPA epidermoid cyst case that developed immediate postoperative brainstem infarction despite entirely stable intraoperative neurophysiological monitoring. To our knowledge, only a handful of cases have described ischaemic stroke directly attributable to epidermoid cysts, either by tumour compression ^[1, 2] or by postoperative vasospasm ^[3]. However, none have documented an event occurring immediately after surgery with completely uneventful intraoperative monitoring ^[5]. The case carries a critical teaching message

a completely normal monitoring run does not guarantee freedom from ischaemic complications in high-risk tumour surgeries. This observation has direct implications for preoperative counselling and postoperative surveillance.

Case Report

A 33-year-old man presented with a 15-day history of intermittent dizziness. He had untreated hypertension for three years. Neurological examination and routine laboratory tests were normal.

Preoperative magnetic resonance imaging (MRI) showed an irregular lesion in the interpeduncular, ambient, suprasellar and prepontine cisterns, hypointense on T1-weighted imaging (Fig. 1A), hyperintense on T2-weighted imaging (Fig. 1B), and with marked restricted diffusion on diffusion-

weighted imaging (DWI) (Fig. 1C)—features diagnostic of an epidermoid cyst. The tumour completely encased the bilateral vertebral and basilar arteries and adhered tightly to the ventral brainstem, indicating an extremely high surgical risk.

After thorough informed consent, a senior neurosurgeon performed a standard retrosigmoid craniotomy. Intraoperative neurophysiological monitoring (IONM), including brainstem auditory evoked potentials (BAEPs) and somatosensory evoked potentials (SSEPs), remained stable throughout the procedure with no significant changes. The tumour was dissected from the encased vessels and brainstem under the operating microscope and resected piecemeal, achieving gross-total resection (Fig. 2). Total operative time was 8 hours and 20 minutes.

Immediately after surgery, the patient was noted to have a fixed, dilated left pupil measuring 4.5 mm with absent direct and consensual light reflexes. The right pupil measured 2.5 mm with variable size but preserved reactivity. Emergency head computed tomography (CT) excluded intracranial haemorrhage. Mechanical ventilation and vasodilator therapy (nimodipine) were initiated.

On postoperative day 1, the patient remained somnolent. Neurological examination: left pupil 4.5 mm with no light reflex, right pupil 2.5 mm with normal reactivity, right-sided hemiparesis (upper limb power 0/5, lower limb 5/5), and normal left-sided motor function. MRI/DWI confirmed acute infarction in the ventral brainstem (Fig. 3). Vasodilator therapy and cerebral protective measures were continued.

On postoperative day 4, the right pupil measured 3.5 mm with sluggish reactivity. By postoperative day 7, both pupils were equal, round and reactive (2.5 mm). On postoperative day 10, the patient developed fever (39.2°C). Cerebrospinal fluid (CSF) analysis confirmed meningitis, which was successfully treated with vancomycin and meropenem over 12 days.

The patient was discharged on postoperative day 31. At discharge, he was awake and alert, with equal and reactive pupils, right upper limb power 3/5, right lower limb power 5/5, and normal left-sided function. Postoperative histopathological examination with haematoxylin and eosin staining showed stratified squamous epithelium with laminated keratin debris, confirming the diagnosis of epidermoid cyst (original magnification $\times 200$) (Fig. 4). Written informed consent was obtained from the patient.

Discussion

1. Mechanism of ischaemic injury:

In this case, the tumour completely encased the vertebrobasilar system, requiring extensive dissection within the narrow CPA corridor. The immediate postoperative onset of pupillary abnormality without haemorrhage on CT strongly supports an ischaemic mechanism. Two mechanisms likely contributed: (1) **perforator injury**—the brainstem receives its blood supply from small perforating arteries originating from the posterior wall of the basilar artery and the anterior inferior cerebellar artery (AICA). These delicate vessels may be encased by tumour capsule, and during dissection, even meticulous

microsurgical technique can result in vasospasm, traction injury or local thrombosis, leading to focal brainstem infarction without damaging larger, identifiable vessels. (2) **Vasospasm**—mechanical manipulation of the vertebrobasilar system may induce sustained vasospasm, reducing distal perfusion pressure to critical thresholds. Interestingly, most reported vasospasm after CPA epidermoid cyst resection occurs 4–14 days postoperatively ^[3]. **However, Housley et al. ^[4] recently described a case of severe vertebrobasilar vasospasm immediately following iatrogenic rupture of a posterior fossa epidermoid cyst, demonstrating that acute intraoperative manipulation can indeed trigger catastrophic vasospasm.** The immediate onset in our patient suggests that perforator injury may be the dominant mechanism, with vasospasm playing a secondary or synergistic role.

2. The critical blind spot of intraoperative neurophysiological monitoring

This case carries a stark clinical warning: BAEPs and SSEPs remained entirely normal throughout a nearly 8.5-hour procedure, yet the patient sustained a clinically significant brainstem infarction. Why did monitoring fail to provide warning? BAEPs assess the functional integrity of the auditory pathway (cochlear nucleus → superior olivary complex → inferior colliculus → medial geniculate body → auditory cortex), while SSEPs assess the dorsal column-medial lemniscal pathway. The blood supply of these pathways derives from long circumferential and perforating arteries that do not completely overlap with the perfusion territory of perforators supplying the ventral brainstem (corticospinal tract, oculomotor nucleus, reticular formation). Consequently, **an ischaemic event confined to the ventral brainstem may leave BAEPs and SSEPs entirely unchanged. This phenomenon has been previously observed in a case reported by Higashino et al. ^[3], where postoperative symptomatic cerebral vasospasm occurred after uneventful resection of an epidermoid cyst, yet intraoperative monitoring had not provided warning. Our case extends this observation to brainstem infarction.** More fundamentally, evoked potentials require substantial neural dysfunction—typically ischaemia reaching the threshold of conduction block—to generate an alarm. Ischaemia limited to a small perforator territory may cause a focal infarction without disrupting the functional integrity of the larger white matter tracts assessed by these modalities. Surgeons must therefore recognise that **normal IONM does not equal perfusion safety**, especially when tumours encase major arteries and their perforating branches.

3. The value of simple pupillary examination

In our patient, the first sign of catastrophe was a fixed, dilated left pupil detected immediately after surgery. This bedside finding, combined with contralateral hemiparesis (localising to midbrain or oculomotor nerve), preceded any imaging confirmation and directly prompted emergency CT and subsequent MRI. Pupillary examination requires no equipment, is repeatable and yields instantaneous results. It should be incorporated into the routine postoperative monitoring protocol for all posterior fossa and CPA surgeries.

4. Comparison with the literature

A PubMed search (1960–2025) revealed only sporadic reports of ischaemic cerebrovascular events following CPA epidermoid cyst resection. Yilmazlar et al. ^[2] reported two cases of brainstem stroke associated with epidermoid tumours, but both were due to tumour compression rather than surgery. Bhaskar et al. ^[1] described a similar case of epidermoid causing ischaemic stroke in the brainstem. More recently, Higashino et al. ^[3] reported delayed cerebral vasospasm following uneventful resection of an epidermoid cyst, and Dib et al. ^[5] documented a complicated case with immediate postoperative ischaemic cerebrovascular accident. However, none of these cases explicitly documented that intraoperative BAEPs and SSEPs remained entirely normal throughout the procedure. To our knowledge, no previous report has combined all three features

(1) immediate postoperative brainstem infarction, (2) tumour encasement of the vertebrobasilar system, and (3) completely uneventful intraoperative neurophysiological monitoring. This case fills a documentation gap and provides a crucial teaching point.

5. Clinical implications

Based on this case, we propose several practical recommendations: **(1) Preoperative counselling:** In high-risk patients with tumour encasement of the vertebrobasilar system, the consent discussion must explicitly include the risk of postoperative brainstem ischaemia/infarction, with the specific warning that this complication may occur even if IONM shows no abnormality. **(2) Intraoperative adjuncts:** When dissecting tumour capsule adherent to the brainstem and its perforator arteries, consider using indocyanine green (ICG) video-angiography or micro-Doppler ultrasonography to assess vessel patency in real-time. Avoid excessive traction, maintain stable mean arterial pressure, and minimise thermal injury during coagulation near perforator origins. **(3) Postoperative surveillance:** Establish a standardised pupillary assessment protocol (every 15–30 minutes until full awakening). Any newly developed anisocoria or pupillary abnormality should trigger: (i) emergency head CT to exclude haemorrhage; and (ii) immediate institution of vasodilator therapy, cerebral protection and optimisation of cerebral perfusion pressure (target mean arterial pressure $\geq$ 90 mmHg in suspected ischaemia).

6. Limitations

This is a single case report, precluding conclusions about incidence. Intraoperative ICG angiography was not available at our institution, so direct visualisation of perforator compromise remains inferential. Long-term functional follow-up data are still being collected. **Future studies should incorporate intraoperative ICG angiography or micro-Doppler to better define the incidence of perforator compromise, as suggested by recent case reports ^[4].**

Conclusion

Brainstem infarction can occur after gross-total resection of a CPA epidermoid cyst despite entirely normal intraoperative BAEPs and SSEPs. This catastrophic complication likely results from perforator injury and/or severe vasospasm. **Normal IONM does not guarantee freedom from ischaemic complications** when tumours encase the vertebrobasilar system. Postoperative pupillary examination is a simple, critical bedside tool for early detection of brainstem injury and should be routine in all posterior fossa surgeries. Prompt vasodilator therapy and aggressive management of secondary complications are essential for optimising outcomes.

Declarations

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Conflict of interest: The authors declare no conflict of interest.

Informed consent: Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Author contributions

C.L. and J.C. conceptualized and designed the study. C.L. curated the data, wrote the original draft, and prepared the visualization. E.C. performed the investigation, developed the methodology, provided resources, validated the findings, and reviewed and edited the manuscript. K.S. conducted the formal analysis, managed the software, and reviewed and edited the manuscript. J.C. supervised the project, administered the study, acquired funding, and reviewed and edited the manuscript. All authors read and approved the final manuscript.

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Figures

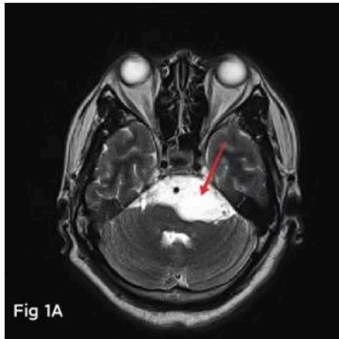
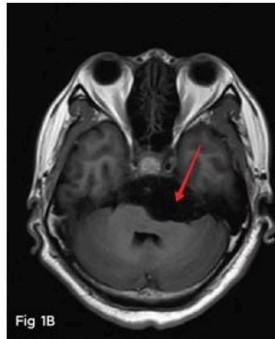
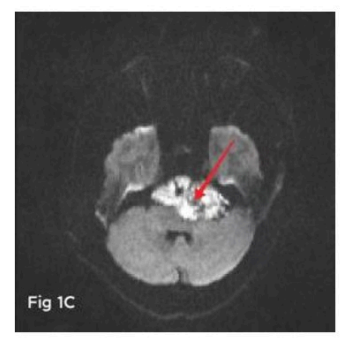


Figure 1

Preoperative magnetic resonance imaging (MRI) showed an irregular lesion in the interpeduncular, ambient, suprasellar and prepontine cisterns, hypointense on T1-weighted imaging (Fig. 1A), hyperintense on T2-weighted imaging (Fig. 1B), and with marked restricted diffusion on diffusion-weighted imaging (DWI) (Fig. 1C)

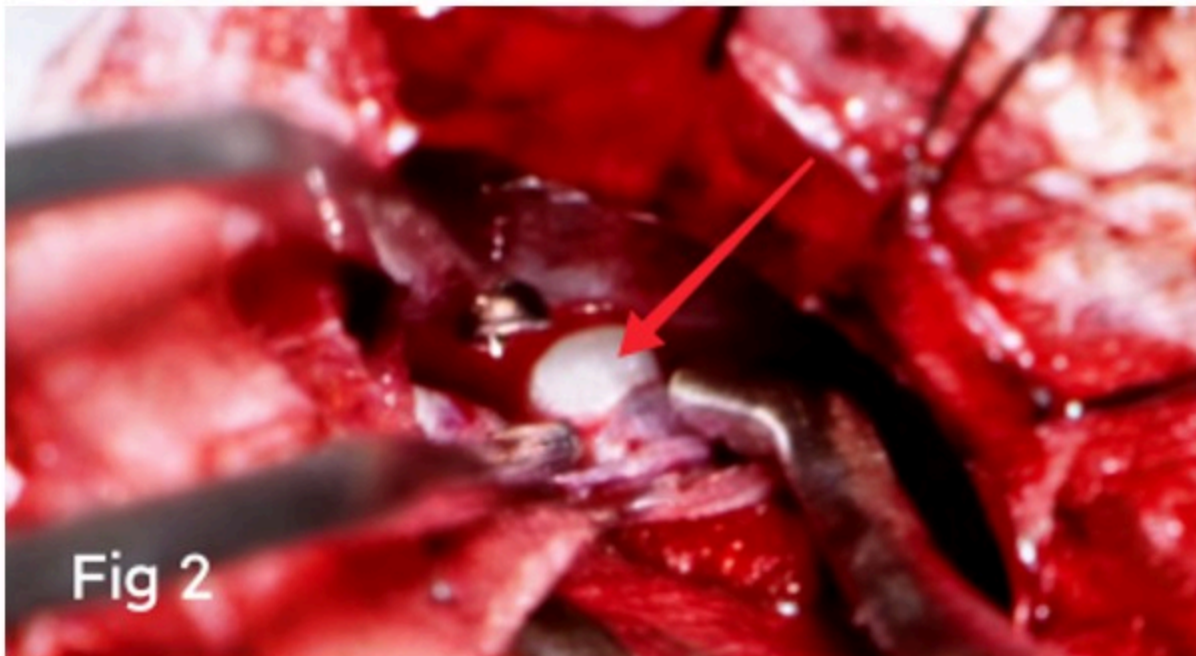


Figure 2

Intraoperative photograph. The typical pearly-white appearance of the epidermoid cyst is visible in the cerebellopontine angle after retrosigmoid exposure. The tumour was carefully dissected from the encased vessels and completely excised.

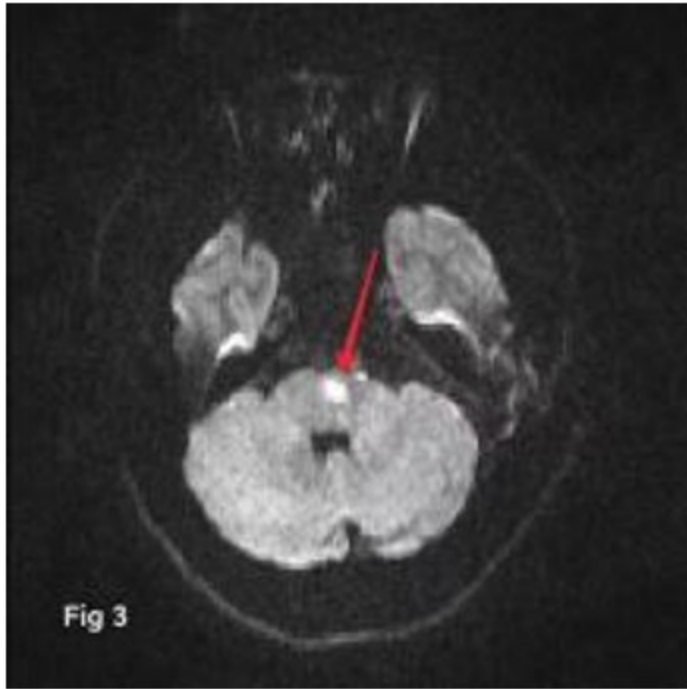


Figure 3

Postoperative brain MRI. DWI showing acute infarction in the ventral brainstem (arrow).

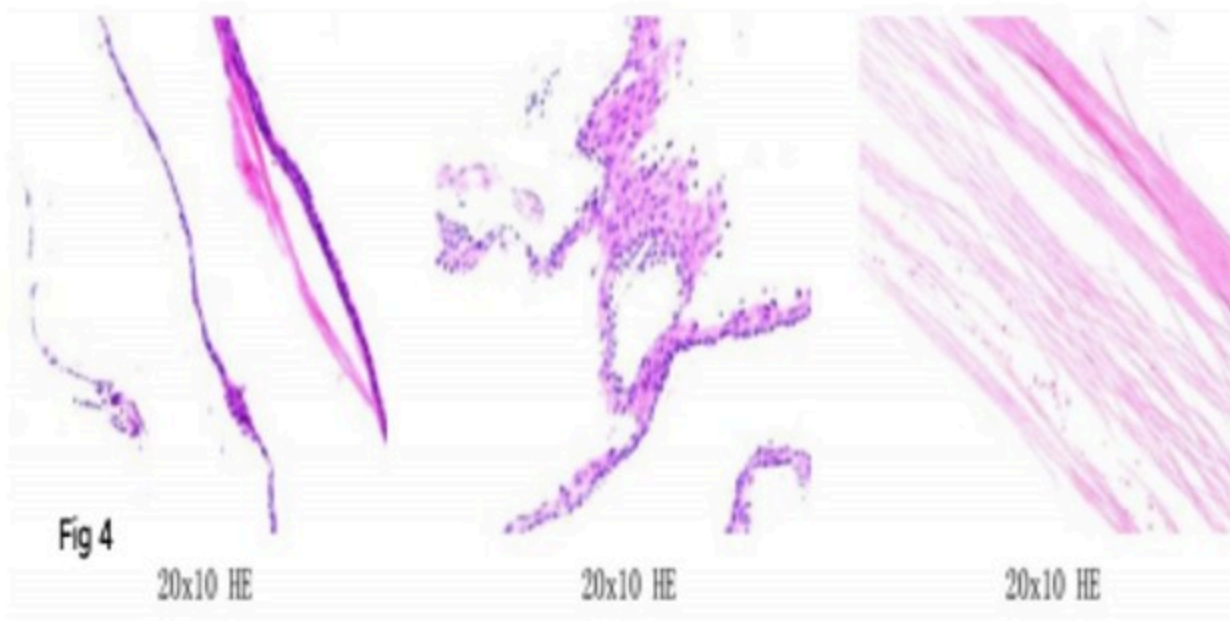


Figure 4

Postoperative histopathological examination. Haematoxylin and eosin staining showing stratified squamous epithelium with laminated keratin debris, confirming the diagnosis of epidermoid cyst (original magnification $\times 200$).