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Synchronous Deep White-Matter Haemorrhages after Endoscopic Transsphenoidal Pituitary Macroadenoma Resection: Case Report and Literature Review

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ABSTRACT

Background: Intracranial haemorrhagic complications post endoscopic transsphenoidal surgery (TSS) are rare, and remote intracerebral haemorrhage (RICH) is an exceptionally infrequent occurrence. Furthermore, disseminated haemorrhages within the deep midline white matter are remarkably uncommon. We report a case of a patient who developed synchronous, multifocal disseminated haemorrhages following endoscopic partial resection of a massive pituitary macroadenoma. **Case summary:** A 75-year-old woman with a massive sellar–suprasellar pituitary macroadenoma compressing both the optic nerves and the chiasm, underwent endoscopic endonasal tumour debulking, followed by autologous skull base reconstruction. She had normal vital signs and was neurologically stable immediately post-operative but later developed acute left sided hemiparesis and a marked decline in consciousness approximately 30 hours post-operatively. **Investigations:** Neuroimaging (30 hours post-operatively) revealed disseminated haemorrhages within the deep white matter and the cerebellum, along with small cerebral cortical infarcts, reflecting the multifocal nature of the injury. Cerebrospinal fluid (CSF) analysis post-deterioration showed markedly elevated protein (1,424 mg/dL; measured by spectrophotometry), a white cell count of 84 cells/μL (80% polymorphs), and normal glucose. Opening pressures were documented as normal. **Management:** The patient received supportive neurocritical care, scheduled lumbar drainage for CSF rhinorrhoea (which started on the fourth day after surgery), and broad-spectrum antibiotics. **Outcome:** Despite maximal therapy, the patient's condition progressed to brain death, confirmed on post-operative day 10. **Conclusion:** Deep white matter disseminated haemorrhages after TSS are exceptionally rare. Potential etiologies include cerebral vasospasm, venous congestion secondary to dysregulated CSF dynamics, and microembolic events. This case highlights the importance of timely post-operative neuroimaging in any patient demonstrating neurological deterioration following TSS, with recognition of this imaging pattern as a potential complication.

Keywords: Transsphenoidal surgery (TSS), pituitary macroadenoma, remote intracerebral haemorrhage

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INTRODUCTION

Endoscopic transsphenoidal surgery (TSS) is currently a widely accepted minimally invasive approach for treating pituitary and parasellar pathology. Compared with older microscopic techniques, it is associated with lower morbidity and a more favourable overall complication rate [1,2]. Expected complications include CSF leakage, diabetes insipidus, post-operative

meningitis, intrasellar haemorrhage, and other local surgical problems [2,3]. Remote intracerebral haemorrhage (RICH), defined as bleeding that occurs at a site distant from the operative field is exceptionally rare, and when described, most often involves the cerebellum or superficial cortical regions [1,4,5].

Deep midline white matter haemorrhages, involving structures such as the corpus callosum, fornix, or deep internal venous territories, are commonly reported in settings of trauma, hypertensive crises, diffuse axonal injury, or other non-surgical insults [6]. The deep midline venous system may be particularly vulnerable in elderly patients, potentially predisposing them to such complications [15,16]. An informal literature review (PubMed, Google Scholar; keywords: remote haemorrhage, transsphenoidal, corpus callosum, fornix; up to December 2024) revealed no prior reports of synchronous haemorrhages in these specific deep midline structures alongside the cerebellum after TSS. This case thus presents a unique pattern of post-operative remote intracerebral haemorrhage, and we discuss plausible pathophysiological mechanisms supported by the existing literature.

CASE PRESENTATION

Patient information: A 75-year-old, right-handed woman with no documented comorbidities (ASA class II) presented with a two-year history of bitemporal headaches and progressive visual blurring. She was not taking any regular medications, including antiplatelets or anticoagulants.

Clinical findings: Neurological examination revealed a Glasgow Coma Scale (GCS) score of 15, with no focal motor or sensory deficits. Visual acuity was limited to finger counting at one metre bilaterally; formal perimetry was not performed, and fundoscopy showed no papilledema. She had no cognitive changes either.

Investigations: A post-contrast CT scan and Magnetic resonance imaging (MRI; 1.5T) sequences (sagittal/coronal T1-weighted post-contrast, axial T2-weighted, and FLAIR) demonstrated a 3.0 × 3.1 × 4.7 cm sellar–suprasellar mass with superior displacement and kinking of the optic chiasm and erosion of the sellar floor (Figures 1 & 2). An endocrine work-up revealed a mildly elevated prolactin level (537 mIU/L, reference range: 102–496 mIU/L), consistent with stalk effect; a full anterior pituitary panel (ACTH, cortisol, TSH, fT4, IGF-1, LH, FSH) was within normal limits. Baseline coagulation profile (platelets, PT/INR, aPTT), full blood count, electrocardiogram, and echocardiogram were unremarkable.

Management

A binostril endoscopic endonasal TSS was performed. Neuronavigation, microdoppler, and intraoperative ultrasound were not utilised. Tumour debulking was performed piecemeal with preservation of the normal gland. No intraoperative CSF leak was noted. Skull base reconstruction was performed in a multilayered fashion: autologous thigh fat graft, followed by fascia lata and a vascularised nasoseptal flap. For nasal tamponade, a 14Fr Foley catheter balloon was filled with 12 mL of sterile saline (estimated pressure 15–20 mmHg) and left in place for 48 hours, which is a common practice for supporting complex multilayer reconstructions, especially those that use vascularised flaps. This period aims to ensure initial graft adherence and stabilisation of the nasoseptal flap, thereby mitigating the risk of early cerebrospinal fluid (CSF) leakage. No intraoperative complications were noted, and there was no obvious excessive CSF leak or difficulty in reconstruction.

A lumbar drain was placed prior to the start of the procedure (institutional protocol). Opening pressures were not recorded at the time of placement. The drain was intended primarily for prophylaxis against intraoperative high-flow cerebrospinal fluid (CSF) leaks, postoperative CSF rhinorrhea, and communicating hydrocephalus. Notably, no CSF drainage was required during the operation. The drain remained clamped throughout the procedure to prevent inadvertent drainage, as confirmed by frequent monitoring.

Estimated blood loss was 100 mL, with no transfusion required. Operative duration was 6 hours. Peri-operative antibiotic (cefazolin) and steroid coverage (dexamethasone 4mg q6h) were administered. Vasopressors were not required, and mean arterial pressure (MAP) was maintained between 80 and 100 mmHg.

Pathology Findings Histopathological examination confirmed a pituitary neuroendocrine tumour (pituitary adenoma). The specimen exhibited

monomorphic tumour cells arranged in nests and sheets. The cells had uniform nuclei with finely stippled chromatin, inconspicuous nucleoli, and moderately abundant cytoplasm. No necrosis was identified, and mitotic activity was scarce, consistent with a low proliferative index.

Outcome

The patient was admitted to the intensive care unit (ICU). Her immediate postoperative GCS was 12 (E3V4M5), which remained stable for over 24 hours. Vital signs (all normal, including mean arterial pressure) and neurological observations (hourly GCS, pupils, and limb power) were charted. Complete hourly documentation was unavailable for retrospective review. An initial non-contrast head CT (performed 24 hours post-operatively; 3 mm slices) showed only expected post-resection changes. Hypopituitarism was considered; a stat dose of 100 mg of intravenous hydrocortisone was given, followed by 50 mg every 12 hours for five days. Low-molecular-weight heparin for venous thromboembolism prophylaxis was withheld until after the deterioration event. No vascular imaging was pursued at this stage, as there was a low index of suspicion for a vascular event based on the initial post-operative CT findings.

Deterioration Event

Approximately 30 hours post-operatively, the patient acutely developed left-sided hemiparesis (MRC grade 2/5) and a rapid decline in consciousness to a GCS of 8 (E1V2M5). Blood pressure at onset was 146/84 mmHg. An urgent non-contrast CT (second post operative CT scan, performed at 30 hours post-op) revealed multifocal, punctate to confluent haemorrhages involving the body and splenium of the corpus callosum, the columns of the fornix, and the deep periventricular white matter bilaterally. In addition, a small 2 mm haemorrhage was noted in the left cerebellar hemisphere. There were also small acute cortical infarcts in the right frontal and parietal lobes, consistent with an embolic pattern of injury. No significant mass effect or midline shift was observed. (Figure 3)

CSF Studies & CSF Rhinorrhoea

CSF analysis post-deterioration revealed: RBC 120/μL, xanthochromia present, WBC 84/μL (80% polymorphs), protein 1,424 mg/dL (spectrophotometric assay), and glucose 3.8 mmol/L (serum glucose 5.6 mmol/L). Gram stain and subsequent culture were negative. The serum–CSF albumin quotient was not available, limiting assessment of blood–CSF barrier dysfunction in the context of markedly elevated CSF protein. Opening pressure was 12 cmH_2O. On post-operative day 4, the patient developed right-sided CSF rhinorrhoea. Scheduled lumbar drainage was initiated at 20 mL every 4 hours (total 120 mL/24 h), which controlled the leak. The drain was removed on postoperative day 9.

Management, Outcome, and Brain Death Declaration

Advanced vascular imaging (e.g., computed tomography angiography (CTA)) was not performed due to resource constraints. No specific anti-vasospasm therapy (e.g., nimodipine, induced hypertension) was instituted. Management consisted of mechanical ventilation ($PaCO_2$ target 4.5–5.0 kPa), neuroprotective measures, and broad-spectrum antibiotics (ceftriaxone 2g q12h) for suspected meningitis. Intracranial pressure (ICP) monitoring was not available. Despite maximal supportive care, she progressed to brain stem death. Brain death was confirmed on post-operative day 10 via two separate clinical examinations 6 hours apart, demonstrating absent brainstem reflexes and apnoea ($PaCO_2$ >8.0 kPa).

A detailed neuropsychological assessment was precluded by her rapid decline; however, the involvement of midline structures such as the corpus callosum is associated with deficits in interhemispheric transfer, memory, and executive function [13,14]. Immediate family members were adequately apprised of the guarded prognosis and signed a do-not-resuscitate form.

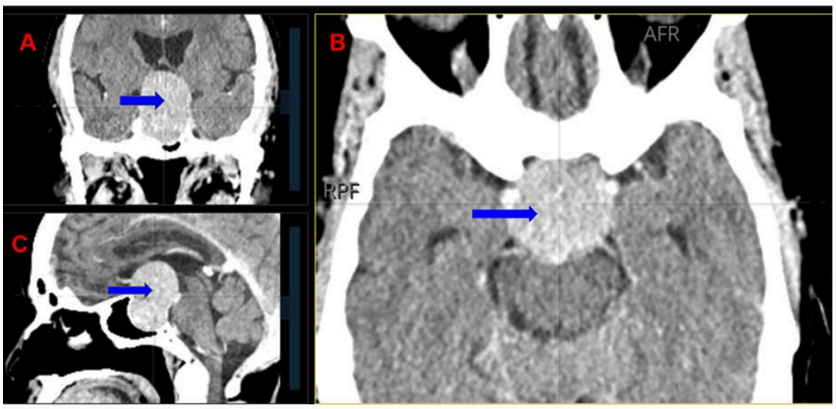


Figure 1. Pre-operative (3-mm slice) post-contrast CT images demonstrating a sellar–suprasellar mass (blue arrows) with superior extension causing upward displacement of the third ventricle. (A) Coronal image at the level of the uncus showing the well-defined lesion. (B) Axial image at the level of the cerebral peduncles. (C) Midline coronal image further delineating the mass and its suprasellar extension.

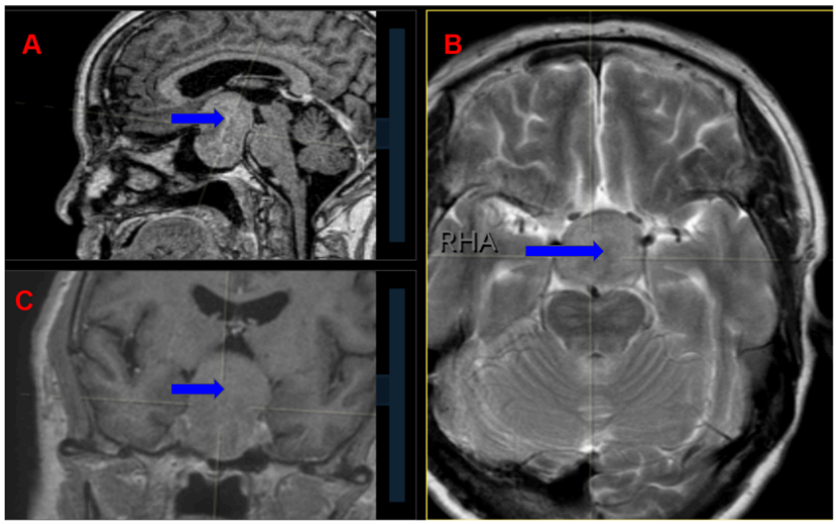


Figure 2. Illustrates the pre-operative MRI of the patient's brain, highlighting a sellar/suprasellar tumor (blue arrows). The midline sagittal T1-weighted post-contrast image (A) demonstrates a mass in the sellar and suprasellar region displacing the third ventricle. The axial T2-weighted image at the level of the midbrain (B) shows a well-circumscribed sellar lesion, while the coronal T1-weighted post-contrast image at the level the root of zygoma (C) depicts the tumor's extent in the coronal plane.

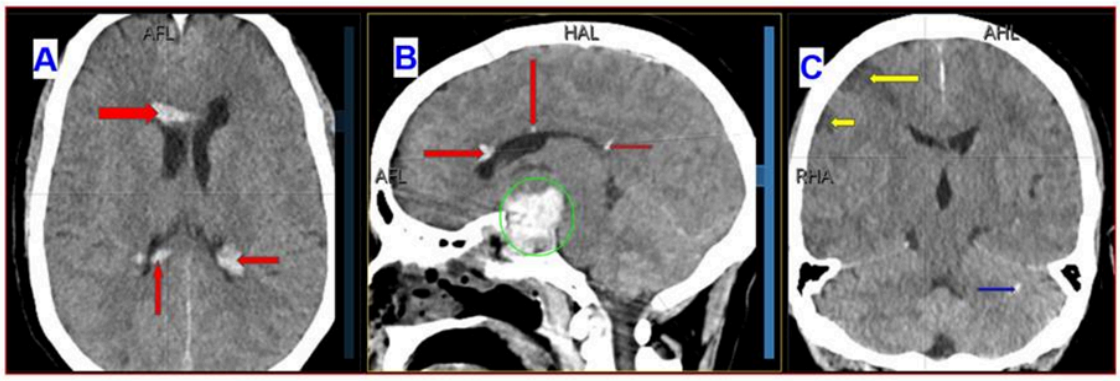


Figure 3: Post-operative non-contrast CT (3-mm slice) scan following deterioration in GCS. Images A and B demonstrate disseminated white matter haemorrhages (red arrows). The green circle in Image B highlights expected post-operative changes in the tumor bed following partial resection. Image C shows parietal cortical infarcts (yellow arrows) and a tiny left cerebellar intraparenchymal haemorrhage (blue arrow).

DISCUSSION

1. RICH after TSS—what is known

Remote intracerebral haemorrhage (RICH) following transsphenoidal surgery (TSS) is rare, with most reported cases involving the cerebellum or superficial cortical regions rather than deep structures. Early series and case reports describe cerebellar haemorrhage occurring within hours to a few days post-operatively, often associated with CSF loss and altered intracranial pressure dynamics [1,11]. More recent reports confirm that RICH can also occur after endoscopic skull base procedures, though the overall incidence remains low and the spectrum is heterogeneous [3].

Timing is variable but typically early (within 24–72 hours), consistent with our patient's deterioration at 30 hours. Reported distributions predominantly include cerebellar and lobar haemorrhages; deep midline involvement is not emphasised in existing TSS literature [1,3,11]. Importantly, vasospasm has also been described after pituitary surgery, usually presenting in a delayed fashion (often several days post-operatively) and associated with arterial narrowing on imaging [4,5,10]. Outcomes in reported RICH cases range from full recovery to severe disability or death, depending on haemorrhage burden and associated ischaemia [1,11].

Taken together, the literature supports that RICH after TSS is uncommon, typically early, and most often cerebellar—making the deep, synchronous, multifocal pattern in this case unusual but not entirely outside the broader spectrum of post-operative intracranial haemorrhagic complications.

2. Why deep midline structures?

The predilection for deep midline involvement in this case likely reflects the unique vascular anatomy of these regions. The corpus callosum, fornix, and periventricular white matter are supplied by small perforating arteries and drained by the internal cerebral venous system, which is relatively low-pressure and susceptible to outflow obstruction.

These territories are particularly vulnerable to haemodynamic perturbations, including venous congestion and microvascular dysregulation. In elderly patients, age-related vascular fragility and reduced compliance may further increase susceptibility to haemorrhagic transformation under stress conditions [6,15,16].

While most reported RICH cases involve the cerebellum, the same underlying principles—venous vulnerability and pressure disequilibrium—may extend to deep midline structures. However, this extrapolation should be made cautiously, as direct evidence for this distribution in TSS is lacking.

3. Mechanisms in this case

The radiological pattern—deep haemorrhages with concurrent cortical infarcts—strongly suggests a multifactorial process.

(a) CSF dynamics and venous congestion

Classically, RICH has been linked to excessive CSF loss causing intracranial hypotension, downward brain displacement, and tearing or congestion of veins [7,8,11]. This mechanism is well described in spine surgery, particularly following durotomy [7]. However, extrapolation to TSS must be cautious.

In our case, the lumbar drain remained clamped intra-operatively, no overt CSF leak was identified, and post-deterioration opening pressure was normal. These findings argue against significant early intracranial hypotension. Two potential mechanisms may explain this phenomenon, including occult intra-operative CSF loss that was not appreciated during reconstruction, as well as an early post-operative microleak preceding the clinically evident CSF rhinorrhoea, which manifested on post-operative day 4.

Thus, rather than sustained hypotension, a transient disturbance in CSF equilibrium may have contributed to venous congestion in vulnerable deep venous territories. The later need for scheduled CSF drainage supports an underlying CSF dynamic instability, though it likely represents a secondary rather than primary driver.

(b) Cerebral vasospasm (large artery vs perforator)

Cerebral vasospasm after TSS is recognised but uncommon, with reported cases demonstrating large-artery narrowing on angiographic imaging and typically delayed onset [4,5,10]. In contrast, our patient deteriorated at 30 hours, which is

earlier than most classical vasospasm presentations.

A key distinction should be made between large-artery vasospasm, which is typically detectable on CTA or MRA, and perforator or microvascular vasospasm, which may not be captured on routine vascular imaging.

The presence of small cortical infarcts alongside deep haemorrhages raises the possibility of diffuse vasculopathy affecting both cortical and perforating vessels. Elevated CSF protein may reflect blood-product–mediated or inflammatory endothelial dysfunction contributing to this process [10].

The absence of CTA, MRA, or transcranial Doppler (TCD) in this case limits diagnostic certainty. CTA/MRA could have identified proximal vasospasm, while TCD may have provided indirect evidence of flow velocity changes. Without these, vasospasm remains a plausible but unconfirmed contributor.

(c) Microembolic phenomena

The cortical infarcts are most suggestive of an embolic component. Potential intraoperative sources of microemboli include fat droplets from autologous graft harvest, air emboli introduced during irrigation, and thrombotic material generated during surgical manipulation. Microemboli may occlude distal vessels, leading to ischaemia with secondary haemorrhagic transformation, particularly in vulnerable white matter territories [12].

Preventive strategies include meticulous irrigation, minimisation of air entry, and careful handling of graft material. While direct evidence in TSS is limited, similar mechanisms have been described in other neurosurgical settings [12].

4. Practical monitoring and management implications

Several practical considerations arise from this case. Early recognition is critical, as any neurological deterioration following TSS warrants urgent neuroimaging, even when initial post-operative scans are unremarkable. Where available, vascular assessment with CTA or MRA should be considered in patients presenting with unexplained neurological deficits to evaluate for vasospasm or embolic phenomena, while transcranial Doppler may serve as a useful bedside adjunct. Mechanism-directed care is essential, as differentiating between venous, vasospastic, and embolic processes has direct therapeutic implications, such as haemodynamic augmentation in vasospasm versus supportive care in venous infarction. Additionally, careful monitoring of CSF dynamics remains important, even when lumbar drains are initially clamped, given the possibility of delayed leaks or occult disturbances.

5. Resource-limited considerations

Low-resource settings, including our institution, may limit access to advanced vascular imaging and intracranial pressure monitoring. Diagnostic evaluation often relies heavily on clinical assessment and serial CT imaging due to limited access to advanced modalities such as CTA, MRA or TCD. As a result, differentiation between vasospastic, embolic, and venous mechanisms is frequently inferential rather than confirmatory. Furthermore, therapeutic options, including targeted vasospasm management, may be restricted.

These limitations necessitate a **high index of suspicion** and early escalation of supportive neurocritical care. They also emphasise the necessity of pragmatic protocols tailored to available resources.

Limitations

This report describes a single case, limiting generalisability. The absence of vascular imaging (CTA, MRA, or TCD) precludes definitive assessment of vasospasm or embolic sources. Advanced haemodynamic modelling or computational flow dynamics was not available. Endocrine parameters at the time of deterioration were incomplete, limiting assessment of metabolic contributors. Additionally, resource constraints restricted both diagnostic and therapeutic options, which may have influenced outcomes.

Future Directions

Future work should focus on developing structured post-TSS monitoring algorithms incorporating early neurological assessment and selective vascular imaging. Multicentre registries are needed to characterise better the incidence, timing, anatomical patterns, and outcomes of RICH after TSS, particularly rare deep white matter presentations.

CONCLUSION

We present an exceptionally rare complication of endoscopic TSS for a pituitary macroadenoma: synchronous disseminated haemorrhages involving the corpus callosum, fornix, deep white matter, and cerebellum. Possible contributing factors include cerebral vasospasm, intracranial hypotension with venous congestion, and microembolic injury. The case also shows how these kinds of lesions can cause serious neuropsychological problems. Rapid recognition of neurological deterioration and timely neuroimaging are essential. Accumulation and reporting of similar cases are required to enhance understanding of risk factors and optimize management.

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Conflicts of interest: The authors report no conflicts of interest

Consent for publication: Verbal informed consent for publication was obtained from the patient's next of kin, as written consent could not be secured due to geographical distance and the circumstances surrounding the patient's death. The consent discussion was documented in the medical record, which is available for review by the editorial board upon request. All identifying information has been anonymised to protect patient confidentiality.

Author contributions: DN: Conceptualisation, literature review, data curation, manuscript writing, and final approval. DC: Surgical intervention, perioperative patient management, clinical oversight. EW: Surgical intervention, perioperative patient management, clinical oversight.

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