

Case Report

# Bilateral caudate nucleus infarcts: A case report of a rare complication following endoscopic resection of a tuberculum sellae meningioma

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## Abstract

**Background:** We present a rare complication of bilateral caudate infarcts and necrosed nasoseptal flaps after endoscopic transsphenoidal resection of tuberculum sellae meningioma. This case highlights the importance of early and accurate diagnosis and treatment of a postoperative cerebrospinal fluid (CSF) leak and associated bacterial meningitis, and reviews any existing guidelines regarding its management.

**Case Description:** A 54-year-old otherwise healthy man presented with progressive bitemporal hemianopsia. Magnetic resonance imaging of the head revealed a large, homogeneously enhancing sellar and suprasellar mass consistent with a meningioma. An endoscopic endonasal transsphenoidal approach was performed to resect the tuberculum sellae meningioma. The patient developed basal bacterial meningitis secondary to a CSF leak, requiring repair on two separate occasions. At the time of both repairs, there was evidence of necrosis of the nasoseptal flaps used for the repairs. Soon after the diagnosis of meningitis, the patient developed bilateral caudate infarcts.

**Conclusion:** This report discusses the possible underlying etiologies for the bilateral caudate infarcts and necrosed flaps including bacterial meningitis with associated local vasospasm of nearby vessels resulting in infarction. This case emphasizes the importance of concise management of postendoscopic CSF leak and discusses the guidelines regarding antimicrobial therapy and the management of lumbar drains.

**Key Words:** CSF leak, endoscopic transsphenoidal surgery, infarct, meningioma, meningitis

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## INTRODUCTION

The endoscopic endonasal transsphenoidal technique is routinely used to resect sellar and suprasellar lesions. Patient pain and discomfort can be reduced when undergoing endoscopic transsphenoidal resection compared to an open cranial procedure.<sup>[15]</sup> Complications from an endoscopic approach can occur and can be

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divided into anatomical (oronasofacial, sphenoid sinus, intrasellar, suprasellar, and parasellar) and endocrinological complications (anterior and posterior pituitary dysfunctions).<sup>[8]</sup> Contemporary published endoneurosurgery studies report rates of cerebrospinal fluid (CSF) leaks between 1.2–6.0%, predominantly for pituitary adenomas.<sup>[2,7,8]</sup> An expanded endoscopic endonasal approach for meningiomas is associated with higher rates of CSF leak.<sup>[10]</sup> Postoperative meningitis has been reported to range 0.4–2.2%.<sup>[2,7]</sup> We report a rare complication of a CSF leak with bacterial meningitis post endoscopic transsphenoidal resection of a tuberculum sellae meningioma, leading to bilateral basal ganglia infarcts, predominantly of the caudate heads. This case highlights the importance of early and accurate diagnosis and treatment of postoperative CSF leak and associated meningitis and reviews the current guidelines regarding its management.

## CASE DESCRIPTION

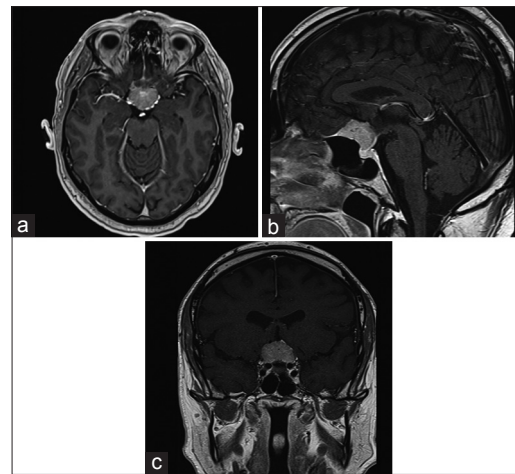
A 54-year-old, right-handed gentleman presented with a 2-year history of progressive bitemporal hemianopsia, more severe on the left. Visual acuity was 20/25-2 in both eyes, with eccentric fixation. There was no relative afferent papillary defect. The remainder of the neurological examination was within normal limits. The patient was otherwise healthy, a nonsmoker, and on no medications. There was no history of vascular disease or vascular risk factors.

### Imaging

Magnetic resonance imaging (MRI) of the head revealed a well-defined enhancing mass in the sella turcica and suprasellar cistern measuring  $2.6 \times 2.7$  cm, with an enhancing dural tail extending along the planum sphenoidale. The pituitary gland and infundibulum were displaced posteriorly. The optic chiasm was significantly displaced superiorly and draped over the superior aspect of the mass. Furthermore, the anterior communicating artery, bilateral A1 segments, and bilateral A2 segments were superiorly displaced. Imaging characteristics were most consistent with a tuberculum sellae meningioma [Figure 1].

### Surgical procedure

Endocrinological investigations did not reveal any signs of pituitary insufficiency. Taking into account the progressive visual changes, the patient elected to undergo endoscopic transsphenoidal resection. The patient was given 2 g cefazolin (Ancef) 30 minutes prior to the surgery. The surgical approach to sella was performed by an otolaryngologist in conjunction with a neurosurgeon. A posterior nasal septectomy was performed to allow binasal access. The face of the sphenoid and intersphenoidal septations were removed and an accessory approach through the superior 1/3 of the clivus was also



**Figure 1: Radiographic appearance of a sellar and suprasellar meningioma in the current patient (a) Axial T1 MRI with gadolinium showing large homogeneously enhancing suprasellar mass. (b) Sagittal T1 MRI with gadolinium showing sellar/suprasellar mass with enhancing dural tail along planum sphenoidale. (c) Coronal T1 MRI with gadolinium showing superior displacement of optic chiasm**

created to maximize access and to facilitate mobilization of the pituitary gland during the case. The tumor was removed by a combination of suction and gentle traction. A gross total resection was achieved. The A1, A2, anterior communicating arteries, optic chiasm, and pituitary stalk were visualized and preserved during the surgery. Intraoperative CSF leak was repaired in a layered fashion consisting of gelfoam, fat, and fascia graft with a dura matrix overlay. A vascularized nasoseptal flap, fashioned at the beginning of the case, was positioned over the repair. Lastly, gelfoam soaked with tranexamic acid and fibrin glue (Tisseel) was laid down into the nasal cavity supported by an inflated Foley catheter. A lumbar drain was inserted postoperatively and discontinued 5 days later. Pathology confirmed the diagnosis of a grade I meningioma, meningothelial subtype.

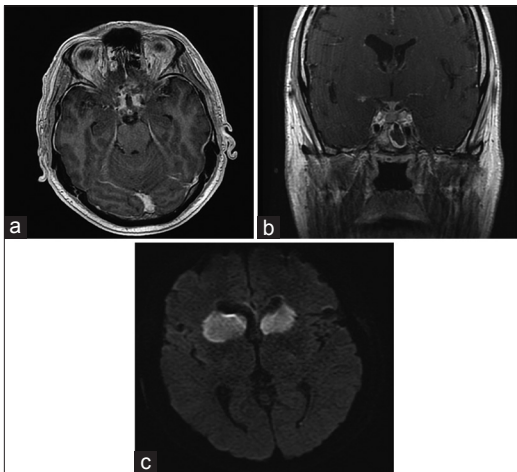
### Postoperative course

The patient's vision and endocrinological status remained stable. Approximately 10 days post surgery, the patient developed a CSF leak and a lumbar drain was reinserted. The patient was brought back to the operating room for repair of the CSF leak. The nasoseptal flap was found to be necrosed up to the proximal arterial supply with surrounding purulent material, which was sent for microbiology and pathology. The flap geometry had not been twisted or under undue tension to account for the necrosis. The repair site was taken down to the dura, which appeared infected from the nasal flap. A muscle graft from the left thigh was then transposed intracranially into the dural defect and dura matrix layered above, followed by a new vascularized inferior turbinate flap secured in place with gelfoam and fibrin glue (Tisseel). A Foley catheter maintained gentle pressure at the repair site and was deflated on postoperative day 1. Microbiological

culture from the septal flap was positive for *Haemophilus influenzae*. Pathology confirmed necrotic tissue of the septal flap with prominent acute inflammation and bacterial aggregates. CSF sent from the lumbar drain had a glucose count of 2.7 mmol/L, protein 977 mg/L, and a nucleated cell count  $708 \times 10^6/L$ .

Within a day of the repair, the patient developed an abrupt severe headache and increasing confusion without obvious evidence of meningismus. MRI of the head revealed extensive leptomeningeal enhancement, surrounding the optic nerves and chiasm, into the Sylvian fissures and encasing the infundibulum [Figure 2a]. Diffusion-weighted MRI revealed bilateral caudate head infarcts, more extensive on the right with involvement of the adjacent capsular white matter and right anterior inferior lentiform nucleus infarct [Figure 2b]. MR angiography revealed constricted left and right A1 segments, Sylvian M1 segments, and the basilar artery [Figure 3]. The A2 branches were well seen. There was no evidence of subarachnoid hemorrhage, abscess, or thrombosis.

The patient was diagnosed with bacterial meningitis and was treated with vancomycin and ceftriaxone to cover for *H. influenzae* and coagulase-negative *Staphylococcus*. The lumbar drain was discontinued on day 5. Six days after the first repair, the patient underwent a second CSF leak repair due to experiencing rhinorrhea when in a dependent position. Similar to the previous repair, the intranasal turbinate flap was completely necrosed; however, the rest of the repair remained virtually intact. Adjustment of the muscle graft stopped the leak.



**Figure 2: MRI head post resection of sellar/suprasellar meningioma. (a) Axial T1-weighted MRI with gadolinium showing extensive leptomeningeal enhancement around the base of the brain and postsurgical packing with no evidence of residual tumor, and (b) coronal T1-weighted MRI with gadolinium showing enhancement extending along the optic nerves and infundibulum. (c) T2-weighted trace, acquired from diffusion-weighted MRI, highlighting infarcts within the caudate head bilaterally, more extensively present on the right**

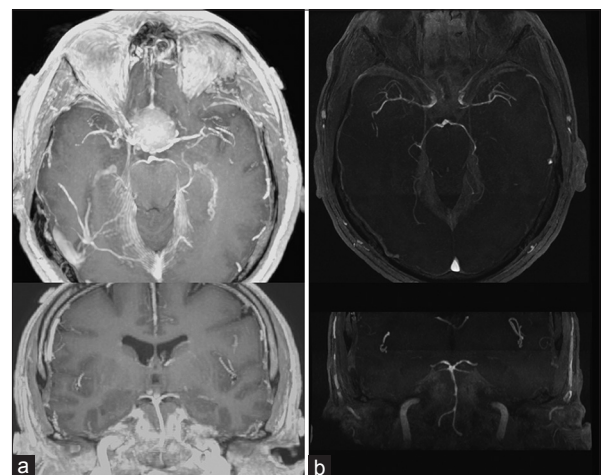
Tisseel and Gelfoam were again used to overlay the wound.

The patient was discharged home without any obvious neurological deficits related to the bilateral caudate infarcts. During interview with the family, the only changes noted were a subtle change in personality, impaired short-term memory, and the observation that the patient infrequently initiated speech. At the 6-month follow-up, the patient had no evidence of pituitary insufficiency and improved visual fields.

## DISCUSSION

Bilateral caudate nucleus infarcts are relatively rare incidents. Behavioral abnormalities, movement disorders, language disturbances, and memory loss may occur, however, prognosis is favorable as 60% of patients fully recover.<sup>[13]</sup> The blood supply to the head of the caudate shows individual variation. The caudate nuclei can receive dual blood supply from the middle cerebral artery (MCA) and anterior cerebral artery (ACA), although classically, is supplied by the medial lenticulostrates including the recurring artery of Heubner.<sup>[1]</sup> This artery commonly branches from the ACAs that run along the A1 segments, and was compromised in our patient as evidenced by constricted A1 segments on the patient's MR angiogram, whereas the A2 branches were well visualized. The risk of infarct is generally dependent on the adequacy of collateral blood supply, cardiac output, blood pressure, and intracranial pressure. Although ipsilateral caudate infarcts are seen following surgery for anterior communicating aneurysms, the occurrence of bilateral caudate infarcts is rare following neurosurgical procedures.

The intimate temporal relationship between this patient's bilateral caudate nucleus infarcts and diagnosis of basal meningitis points to the etiology of this vaso-occlusive



**Figure 3: MR angiogram of the intracranial vessels (a) preoperatively and (b) postoperatively, showing narrowing of basal arteries postoperatively including A1 segments, M1 segments, distal ICA, and basilar artery**

event. MRI revealed basal cisternal infection with extensive leptomeningeal enhancement. There was visible local narrowing on MR angiogram of the A1 segments likely impacting on flow within the medial lenticulostriates as well as small cortical perforators, resulting in the infarcts. There was also narrowing of the M1 Sylvian segments and mild narrowing of the basilar artery; however, there was likely enough collateral blood supply distal to these to prevent any more extensive infarcts. In the context of cerebral inflammation or bacterial meningitis, many different factors influence cerebral blood flow including inflammatory hyperemia, mean cerebral blood flow velocity, increased intracranial pressure, arterial CO<sub>2</sub>, and mean arterial pressure among others. Cerebral inflammation in bacterial meningitis has been associated with reactive vasospasm of cerebral arteries and arterioles, effectively reducing the downstream blood flow.<sup>[4]</sup> A case reported in 1966 established a link between bacterial meningitis and cerebral vasospasm and featured *H. influenzae* as the virulent organism,<sup>[11]</sup> as in our patient. Due to the inflammatory response to bacterial meningitis, imbalances between leukocyte-orchestrated vasoconstricting and vasodilating agents have been implicated in the pathogenesis of cerebral vasospasm.<sup>[6]</sup> Bacterial meningitis secondary to CSF leak is an uncommon but known complication of endoscopic transsphenoidal surgery.<sup>[2,7]</sup>

An initial review of this case focused on the use of the anti-fibrinolytic agent tranexamic acid during the meningioma resection operation. Tranexamic acid is used in patients with major life-threatening bleeding and has many off-label uses including dental procedures, perioperative bleeding control in cardiac and spinal surgery, epistaxis, blood conservation in hip or knee arthroplasties, caesarean sections, and transurethral prostatectomies.<sup>[3]</sup> A warning of increased risk for cerebral thrombosis and deep vein thrombosis was initially associated with this drug.<sup>[3]</sup> However, upon exploration of randomized controlled trial data from a diverse group of surgical and nonsurgical studies, no investigation showed an increased risk of vaso-occlusive events in relation to tranexamic acid administration. Trauma trials including the MATTERs and CRASH-2 showed no increased risk.<sup>[12,14]</sup> A traumatic brain injury trial utilizing tranexamic acid showed no significant increase in thromboembolic events.<sup>[17]</sup> Multiple trials and meta-analyses of tranexamic acid use within the orthopedic and spinal surgical specialties also failed to show an increased risk of vaso-occlusive events.<sup>[18,19]</sup> As such, there seems to be no obvious rationale suggesting tranexamic acid use as the causative factor for this patient's necrosed nasal flaps or even bilateral caudate infarcts.

Necrosed vascularized nasal flaps have been reported in the literature, although it is not clear why tissue viability was lost in this case. Prior radiation to the nasopharynx or paranasal sinuses is a risk factor for flap necrosis,

whereas septal spurs and iatrogenic injury can lead to intraoperative injury of the flap.<sup>[5]</sup> Our patient was healthy with no history of radiation, a nonsmoker, and without any evidence of intraoperative injury or improper geometry to the flap. The initial necrosed flap showed clear signs of bacterial infection, which was confirmed by microbiological analysis; *H. influenzae* was cultured. However, this organism is considered normal flora for a large proportion of the population and it is unclear why this commensal resulted in basal meningitis in this case.

In cases of intraoperative CSF leakage, meticulous reconstruction of the sellar floor is indicated to prevent postoperative fistula formation. The routine insertion of a lumbar drain in patients in whom an intraoperative CSF leakage has occurred has been shown in one study to significantly reduce the incidence of postoperative meningitis.<sup>[16]</sup> Risk factors for meningitis include age, expanded endoscopic approaches for complex tumors with intraoperative CSF leaks, postoperative CSF leak, abnormal preoperative body mass index (BMI), among others.<sup>[9]</sup> In expanded endonasal surgeries, there may be an increased risk of developing meningitis secondary to gram-negative rods in addition to nosocomial bacteria.<sup>[9]</sup> Few guidelines exist within the use of prophylactic antibiotics in patients undergoing endoscopic sinus surgery and there have been no randomized control trials. It is important that an appropriate preoperative and potentially a postoperative antibiotic regimen covers this broad spectrum of organisms in this setting.

In conclusion, this patient experienced two rare postoperative complications following a successful tuberculum sellae meningioma transsphenoidal endoscopic resection, namely bilateral caudate nucleus infarctions and recurrent necrosed nasoseptal flaps. The most likely etiology is acquired bacterial basal meningitis from a CSF leak, resulting in reactive vasospasm of local arteries. This case reinforces the importance of the early identification of a postoperative CSF leak and early identification and treatment of meningitis to avoid these potential complications.

### Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Nil.

### Conflicts of interest

There are no conflicts of interest.

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