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Delayed symptomatic cerebral vasospasm after transsphenoidal resection of pituitary adenoma

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Surgical intervention is a primary treatment modality in functional pituitary adenomas, with the exclusion of prolactinomas. Usually it is performed with minimally invasive endoscopic endonasal transsphenoidal surgery (EETS). Although, surgery for pituitary lesions is considered a safe procedure [1], pituitary dysfunction is the main cause of life-threatening complications in the postoperative period. In invasive tumours, intraoperative vascular or cranial nerve injury, or postoperative infectious complications should be considered in the differential diagnosis of neurological deterioration, as in other types of skull base lesions.

We present a case of a patient initially diagnosed with Cushing's disease related to invasive macroadenoma that was managed by EETS followed by delayed symptomatic postoperative cerebral vasospasm.

A 23-year-old male patient with signs of Cushing's disease and imaging appearance of pituitary macroadenoma with suprasellar extension (Fig. 1) was admitted to the Endocrinology Department. The patient presented with a progressive left eye temporal visual field deficit, weight gain, hypertension, abdominal and axillary striae, as well as insulin resistance, dyslipidaemia, hepatic steatosis, and features of secondary osteoporosis of the spine in the laboratory and imaging workup. Dynamic hormonal tests confirmed adrenocorticotropin dependent hypercortisolism, and the patient was referred for surgical treatment.

Upon admission to the Neurosurgery Department, neurological examination did not reveal any abnormalities beside left eye visual field deficit (confirmed with perimetry), and the patient was scheduled for EETS for tumour resection. The procedure was performed with bi-nostril, 4-hand technique. Due to suprasellar extension of the lesion the transplanum transtuberculum approach was used. The intraoperative tumour appearance was characteristic of macroadenoma. Due to the invasive character of the lesion, the arachnoid was opened, and a high-flow cerebrospinal fluid (CSF) leak

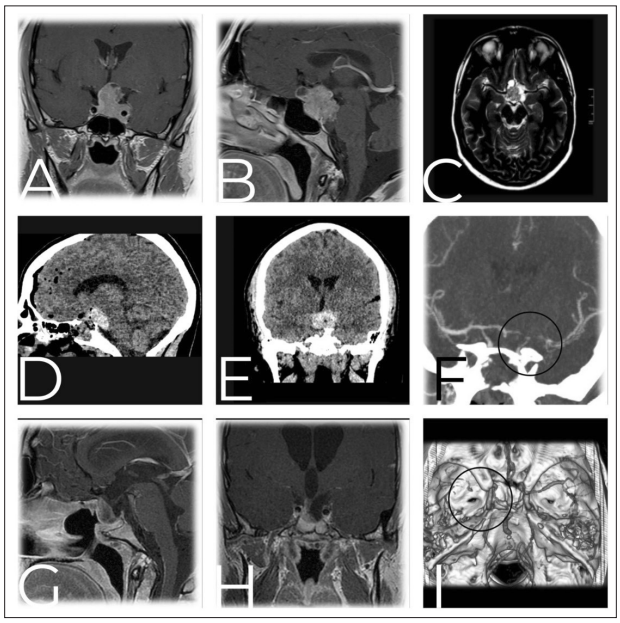

Figure 1. A–C. *Preoperative T1-weighted magnetic resonance imaging (MRI) with contrast enhancement — coronal (***A***) and sagittal (***B***), T2-weighted axial (***C***) images of large sellar-suprasellar tumour.* **D, E.** *Postoperative non-contrasted CT with blood clots in the resection cavity.* **F, I.** *Coronal computed tomography angiography (CTA) scan and CTA 3D reconstruction showing narrowing of the left internal carotid artery, left posterior cerebral artery, M1 segment of the left middle cerebral artery, and A1 segment of the left anterior artery.* **G, H.** *Long-term follow-up imaging presenting proper tumour resection*

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was observed. The tumour did not encase the cavernous sinus internal carotid artery, but suprasellar dissection included manipulation of the supraclinoid internal carotid artery (ICA) and basilar artery bifurcation. Multilayer reconstruction with the use of the nasoseptal Hadad flap was performed. Postoperative histopathological and immunohistochemical testing confirmed the diagnosis of an adrenocorticotropin-producing pituitary adenoma. Immediate postoperative non-contrasted head computed tomography (CT) was unremarkable apart from blood clots within the resection cavity in the interpeduncular cistern. Over the following days, the patient remained neurocognitively stable, without new neurological deficits or CSF leak, with subjective improvement of left eye vision and mild vertigo upon standing up, which resolved on the third postoperative day (POD 3). The patient was discharged on POD 7.

The patient was readmitted on POD 11 due to cognitive deterioration, confusion, speech difficulties, and weakness of the right upper and lower extremities. Physical examination revealed somnolence, speech dysfunction indicative of mixed aphasia, upper and lower right-sided hemiparesis, and decreased ability to perform tasks. Electrolyte and hormonal abnormalities were excluded. The patient received 100 mg of hydrocortisone intravenously without any improvement and underwent non-contrasted head CT and CT angiography (CTA). While head CT did not reveal any abnormalities, CTA showed narrowing of the left supraclinoid ICA, left posterior cerebral artery, A1 segment of the left anterior cerebral artery, and the M1 segment of the left middle cerebral artery. Based on the clinical presentation and imaging findings, the patient was diagnosed with symptomatic delayed cerebral ischaemia related to vasospasm of the left ICA. The patient was immediately started on intravenous nimodipine infusion and maintained in induced hypervolaemia.

Over the next days, the patient remained alert and oriented with improving motor and speech deficits. On the eleventh day after readmission, the patient was diagnosed with SARS-CoV-2 infection and was isolated for 10 days. Throughout the isolation period he remained haemodynamically stable, without significant disturbances. On the POD 20, after completing the isolation, the patient was discharged with a complete resolution of neurological and neurocognitive deficits. Long-term follow-up imaging revealed proper tumour resection without ischaemic changes.

Cerebral vasospasm is a complication typical in neurosurgical practice for subarachnoid haemorrhage of aneurysmal origin (aSAH). Its occurrence after pituitary tumour resection or other skull base lesions is a rare event [2] with an incompletely understood aetiopathology [3]. The management is similar to that in aSAH-attributed vasospasm [4], as there have been no designated treatment protocols established for tumour resection-related cerebral vasospasm. The diagnosis includes ruling out other conditions that can produce delayed neurological deterioration and utilization of angiographic imaging techniques. In milder cases, standard therapy involves oral or intravenous use of calcium channel blockers (nimodipine) along with induced hypertension, hypervolaemia, and/or haemodilution (Triple H — therapy), which improves cerebral blood flow and perfusion. In the case of a more severe clinical course or failed response to standard therapy, endovascular angioplasty or selective intraarterial vasodilator administration might be necessary [5]. In our case prompt diagnosis and intravenous nimodipine infusion with controlled hypervolaemic therapy brought a complete recovery to preoperative cognitive and neurological baseline.

In conclusion, not only pituitary insufficiency can lead to postoperative life-threatening complications. Cerebral vasospasm following pituitary tumour resection is a rare complication with a significant impact on postoperative mortality and long-term morbidity. Increasing awareness of this condition may prevent delays in diagnosis and management, potentially limiting the formation of infarctions and serious unfavourable outcomes.

Ethics statement
Agreement from Bioethics Committee not required.

Author contributions
M.S., T.D., M.G., T.G. — collecting data, writing manuscript; P.K. — supervision, writing manuscript

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Conflict of interest
None declared.

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