Case Report

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Pituitary apoplexy following endoscopic nasal surgery: A case report

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Abstract

Pituitary apoplexy develops as a consequence of acute haemorrhage and/or infarction in a pre-existing pituitary adenoma. Typical symptoms include sudden onset headache, visual acuity/field defects, and ocular palsies. We report a male patient with a known pituitary macroadenoma who underwent a right-sided endoscopic nasal surgery. Preoperatively, thickening of sphenoid mucosa was seen in computed tomography and magnetic resonance imaging. The patient developed pituitary apoplexy postoperatively. The presented report indicates that in patients with a pituitary adenoma, nasal surgery – like any other kind of surgery – is a possible precipitating factor for pituitary apoplexy. Isolated thickening of sphenoid mucosa is associated with pituitary apoplexy. It may also precede an apoplectic event.

Keywords

Pituitary apoplexy, precipitating factor, nasal surgery, polypectomy, sphenoid sinus

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Introduction

Pituitary tumour apoplexy is a rare clinical syndrome which develops as a consequence of acute haemorrhage and/or infarction in a pre-existing pituitary adenoma, typically macroadenoma, causing rapid enlargement of the pituitary tumour, thus compressing the adjacent structures.[1,2] Typical symptoms of the syndrome include sudden onset headache, visual acuity/field defects, ocular palsies, altered mental state, nausea, vomiting, and endocrine dysfunction.[2] It may occur spontaneously, but precipitating factors, such as a surgical procedure, are identified in 20%–40% of cases.[3]

Case report

Ethical approval to report this case was obtained from the Ethics Committee of Pirkanmaa Hospital District. Written informed consent was obtained from the patient for his anonymized information to be published in this article.

A 48-year-old male visited the otorhinolaryngology outclinic complaining of intense right-sided nasal obstruction that had lasted for several months. He had been prescribed intranasal steroids, but they had not relieved the symptom. Ten years earlier, he had been diagnosed with hypogonadotropic hypogonadism and had been using testosterone gel since then. At that time, the sella magnetic resonance imaging (MRI) was normal, with no evidence of pituitary adenoma. During the previous year, he had also experienced increasing tiredness, and 1 month earlier had visited an endocrinologist again. Laboratory results indicated central hypothyreosis and mild hyperprolactinaemia. Thyroxin therapy was initiated. A new sella MRI showed sellar macroadenoma, which proved to be nonfunctional. The tumour had expanded the sella in all directions, and the diameter of the tumour was 18 mm × 15 mm × 18 mm at this time. There was also mucosal thickening on the floor and lateral wall of the sphenoid sinus (Figure 1(a) and (b)). At the time, the patient also met a neurosurgeon, who decided not to operate on the macroadenoma yet, because the patient did not have any problems with vision or other symptoms related to the size of the tumour.

In a nasal endoscopy, a large polypoid mass arising from the direction of the sphenoethmoidal recess on the right side was found. This mass blocked the right choana, but otherwise, the right side of the nasal cavity – like the left side – was completely normal. In sinonasal computed tomography

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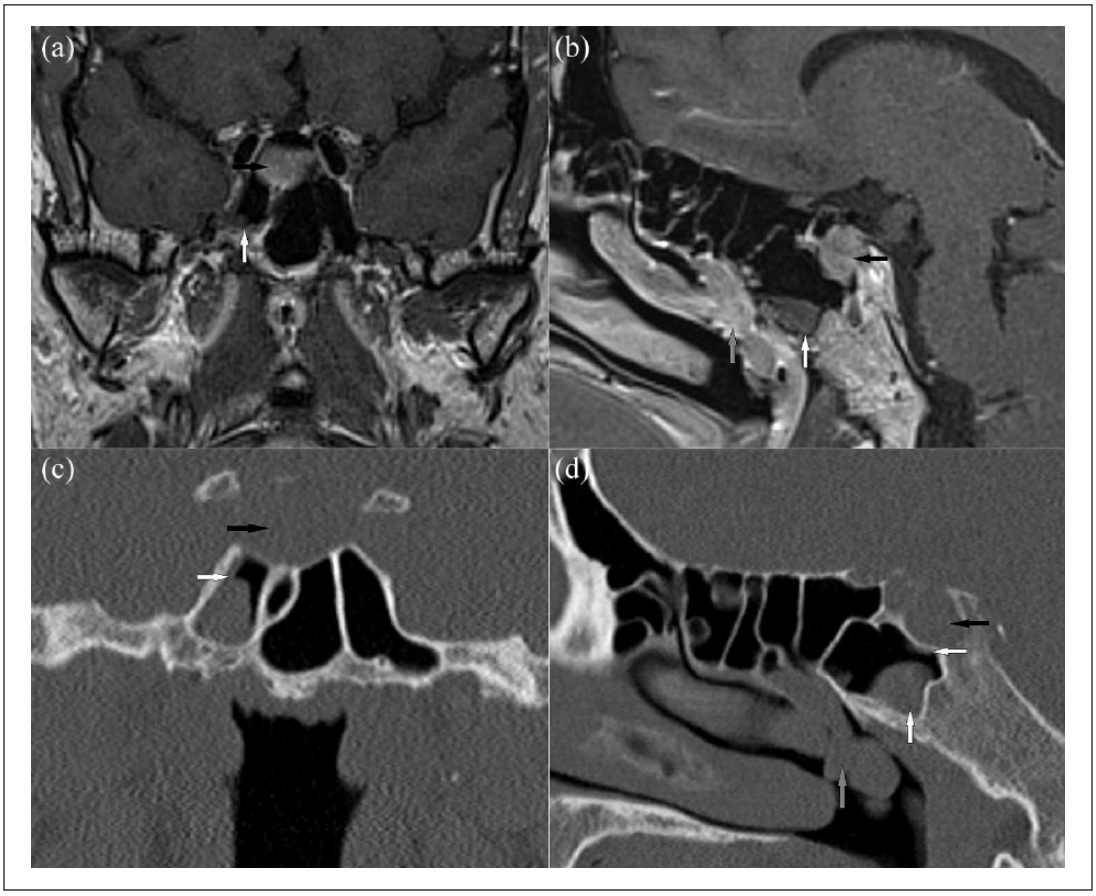

Figure 1. (a and b) Enhanced T1-weighted MRI scan 4 months before the acute stage of the pituitary apoplexy and (c and d) CT scan 3 months before the acute stage of the pituitary apoplexy.
Pituitary macroadenoma (black arrows), right sphenoid sinus mucosal thickening (white arrows), and a polypoid mass blocking the right choana (grey arrows) are seen.

(CT), a soft tissue mass was seen in the sphenoethmoidal recess protruding towards the choanae. Mucosal thickening was seen in the sphenoid sinus posterolaterally. There were no signs of an evident skull base defect, but the cortex of the sella was narrow, indicating pressure erosion (Figure 1(c) and (d)). The possibility of an intranasal tumour, such as an inverted papilloma, was considered. A neurosurgeon recommended the removal of the nasal polyp before the possible forthcoming transsphenoidal pituitary surgery.

Three months later, the patient underwent right-sided endoscopic microdebrider-assisted polypectomy. The polyp, which originated from the mucosa above the right sphenoid ostium, was completely removed. Small sphenotomy was also carried out.

In the recovery room after the operation, the patient complained headache, but was discharged after a neurologist's examination. Two days later, the patient came to the hospital again complaining of diplopia and worsened problems with visual acuity, especially in the left eye. In the neurological examination, the patient's eye movements were disconjugated, and there was mild ptosis in the right eye, indicating oculomotorius paresis. A defect in the temporal visual field of the left eye was also detected. The patient also had a fever, and his C-reactive protein (CRP) level was 147. In an MRI, the tumour had expanded, pushing the chiasma and especially the left opticus upwards and compressing the right oculomotorius nerve laterally. A haemorrhagic component was detected inside the tumour. There was also mucosal oedema in the sphenoid sinus (Figure 2(a) and (b)). A surgical decompression with a craniotomy approach was carried out by neurosurgeons, and the haemorrhagic tumour was removed. In addition, a permanent medical therapy with hydrocortisone 50 mg twice a day was instituted.

Postoperatively, the patient's right eye had become blind as a new symptom, and he still had a defect in the temporal visual field of the left eye. He had also developed anosmia as another new symptom. The patient also had increased urination and hypernatremia referring to diabetes insipidus. Permanent desmopressin therapy was started, and as a result, the urination as well as the serum natrium levels normalized during the following days. Thyroxin therapy was continued with the previous dose and testosterone delivery was changed from transdermal to intramuscular injection between 3 months. Six months after the operation, the patient still had

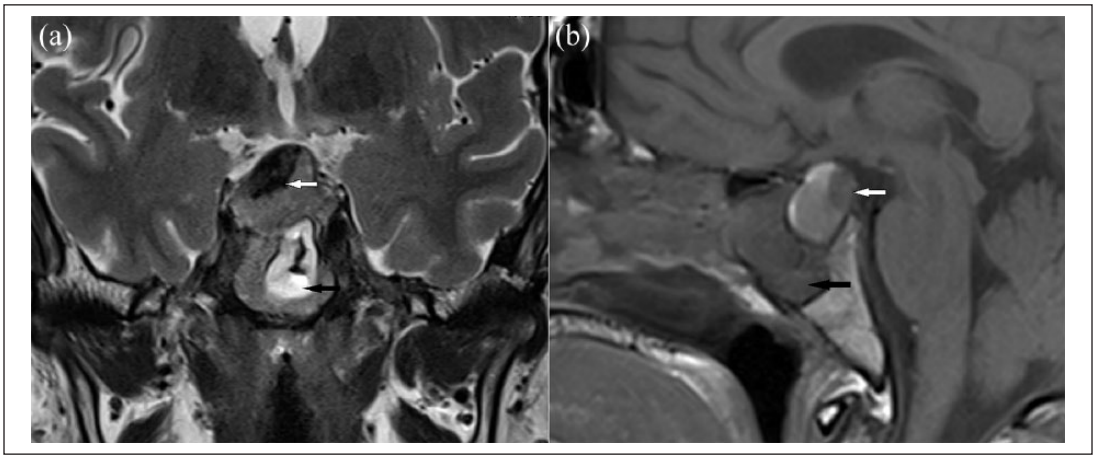

Figure 2. (a) T2-weighted coronal view MRI sequence from the posterior part of the sphenoid sinus and (b) T1-weighted sagittal view MRI sequence from the level of the nasal septum demonstrating haemorrhage in a pituitary macroadenoma (white arrows) and mucosal oedema and in the right sphenoid sinus (black arrows).

Table 1. Precipitating factors of pituitary apoplexy.

Hypertension/hypotension
Major surgery
Coronary artery bypass grafting/stenting
Other types of surgery
Anticoagulation
Clotting disorder
Myocardial infarction
Dynamic endocrine stimulation testing
Oestrogen therapy
Dopamine agonist therapy
Head trauma
Traumatic injury with shock
Radiotherapy
Pregnancy

the visual defects and anosmia, which are likely to be permanent.

Discussion

Pituitary adenomas are vulnerable to haemorrhage and necrosis, possibly because they outgrow their blood supply or because tumour expansion causes ischemia by compressing infundibular or superior pituitary vessels against the sellar diaphragm.[3] The inner features of the tumour may also explain the tendency to bleed. Adenomas have higher metabolism, limited angiogenesis, and reduced vessel density compared to the normal pituitary gland tissue. As a result, the tumour is particularly prone to acute ischemia or spontaneous infarction by any event that acutely changes the balance between tumour perfusion and tumour metabolism.[4] Possible precipitants related to pituitary apoplexy are summarized in Table 1.[2,4]

Optimal management of acute pituitary apoplexy is somehow controversial. Some authors recommend early transsphenoidal surgical decompression for all patients, and others choose a conservative approach for some patients, especially those without visual acuity or field defects and with normal consciousness.[3] In surgical treatment, the transsphenoidal approach is almost always considered better option than craniotomy.[2,3] In the present case, the patient was feverish and the CRP level led to the suspicion of postoperative infection in the sphenoid sinus. This was the reason why neurosurgeons, after a serious consideration, chose craniotomy instead of the transsphenoidal approach. Regarding the patient's vision, the outcome was very unfortunate.

We found only one previously reported case where pituitary apoplexy followed endoscopic sinus surgery.[5] A patient with a known pituitary macroadenoma underwent an endoscopic middle meatal antrostomy and ethmoidectomy for chronic rhinosinusitis with polyps as a preparatory step for safe transsphenoidal access to the pituitary fossa. The followed pituitary apoplexy was successfully treated with emergency endoscopic transsphenoidal resection of the tumour.

Thickening of sphenoid sinus mucosa seen in an MRI or CT scan is associated with the acute stage of pituitary apoplexy and should not be thought as originating from sinusitis. Venous congestion caused by obstruction of the out-to-in transsellar venous flow, which is attributable to a sudden increase in intrasellar pressure, is thought to be a possible mechanism of the thickening.[6] The finding of thickened sphenoid sinus mucosa seems to correlate with a more severe pituitary apoplexy with worse endocrinological and visual outcomes.[7] Agrawal et al. described a case where a patient with acute onset headache had thickened sphenoid sinus mucosa and a sellar mass without evidence of haemorrhage in CT and MRI scans. Three days later, a second MRI demonstrated sphenoid mucosal thickening and a small haemorrhage in the pituitary. They concluded that sphenoid sinus mucosal thickening may also be a precedent and suggestive

of an apoplectic event.[8] In the present case, both CT and MRI scans demonstrated thickening of sphenoid sinus mucosa on the right side – without thickening of other sinus mucosa – 3 and 4 months before the acute stage of the pituitary apoplexy took place. Macroadenoma had expanded the sella in all directions, and the cortex of the sella was narrow, indicating pressure erosion. Therefore, it is likely that the pressure effect of the macroadenoma was related in some way to the sphenoid mucosal thickening even before the actual pituitary apoplexy occurred.

Conclusion

In patients with a pituitary adenoma, nasal surgery – like any other kind of surgery – is a possible precipitating factor for pituitary apoplexy. When a patient is known to have pituitary macroadenoma and elective surgery is planned, the risk of pituitary apoplexy should be considered. If nasal surgery is planned to a patient with pituitary macroadenoma, the need for simultaneous transsphenoidal surgical removal of the tumour should be evaluated. Isolated thickening of the sphenoid mucosa is a finding associated with the acute stage of pituitary apoplexy. Sphenoid sinus mucosal thickening may also be related to the pressure effect of the macroadenoma itself and precede an apoplectic event, thus supporting the case for early neurosurgical intervention.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical approval

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Informed consent

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