Case Report

Post operative 'pituitary apoplexy' in giant pituitary adenomas: a series of cases

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This is report on four cases of giant pituitary tumors that developed postoperative pituitary apoplexy after they had undergone a partial tumor resection. Three patients had undergone surgery by a transsphenoid route and one patient underwent surgery by transcranial route. Postoperative CT scan showed hemorrhagic expansion of residual tumor mass. All the four patients were re-explored transcranially and hemorrhagic swelling of the tumor was observed. In three cases, the tumor had swollen to a size greater than twice the original tumor. All the four patients had a fatal outcome.

Key words: Giant pituitary tumor, apoplexy, transsphenoid surgery

Introduction

The phenomenon of postoperative pituitary apoplexy following partial resection of a pituitary adenoma has been described by Goel et al in 1995.[1] In this report we present our experience with four such cases encountered by us in the last 8 years. The high degree of mortality associated with this phenomenon and the implications in pituitary tumor surgical philosophy is discussed[1].

Case History

In the last 8 years, 890 cases of pituitary tumors were operated at our centre. Among them, 134 patients had giant pituitary adenomas (more than 4 cm in largest extent by definition).[2] Four of these patients developed post operative pituitary apoplexy.

Case 1

A 50-year-old male was admitted with mild headache and progressively decreasing vision in both eyes for 2 years. His visual acuity in both eyes was 6/24 with right temporal field cut. He had hypopituitarism at the time of presentation. Due to a large suprasellar and parasellar components of the tumor (Figure 1 A), the patient was operated by transcranial route. The patient worsened following surgery and became unconscious. Postoperative CT scan showed hemorrhagic swelling of the residual tumor and hypodense area in the brainstem and hypothalamus [Figure 1 B]. Patient was re-operated by a transcranial route, and a large tumor with evidence of intratumoral hemorrhage was identified. Despite re-operation and radical resection of the lesion, the patient continued to worsen in his neurological status and he died on third day following surgery.

Case 2

A 30-year-old male, presented with one-year history of headache and longstanding history of acromegalic features with GH level of 15.5 ng/ml. Visual acuity was 6/24 in left eye and 6/6 in right eye with bi-temporal hemianopia. He underwent a trans-sphenoidal surgery and partial resection of a giant pituitary tumor [Figure 2A]. Following surgery, the patient remained well for six hours, but then worsened in sensorium. CT scan showed swelling of residual tumor with evidence of

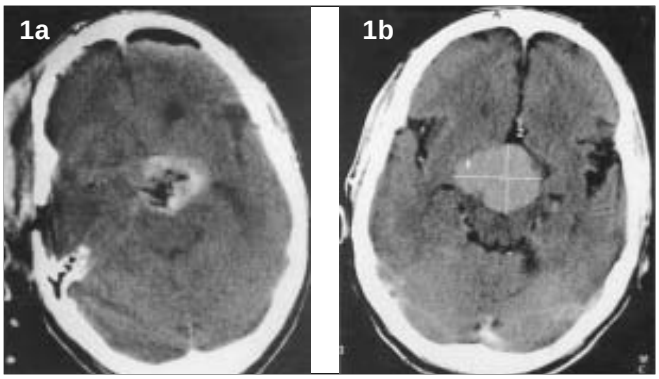

Figure 1: Contrast enhanced CT (CECT) scan showing a giant, homogenously enhancing suprasellar tumor with right parasellar extension, without hydrocephalus (1a). Post operative CT scan showing residual tumor swelling with bleed (1b).

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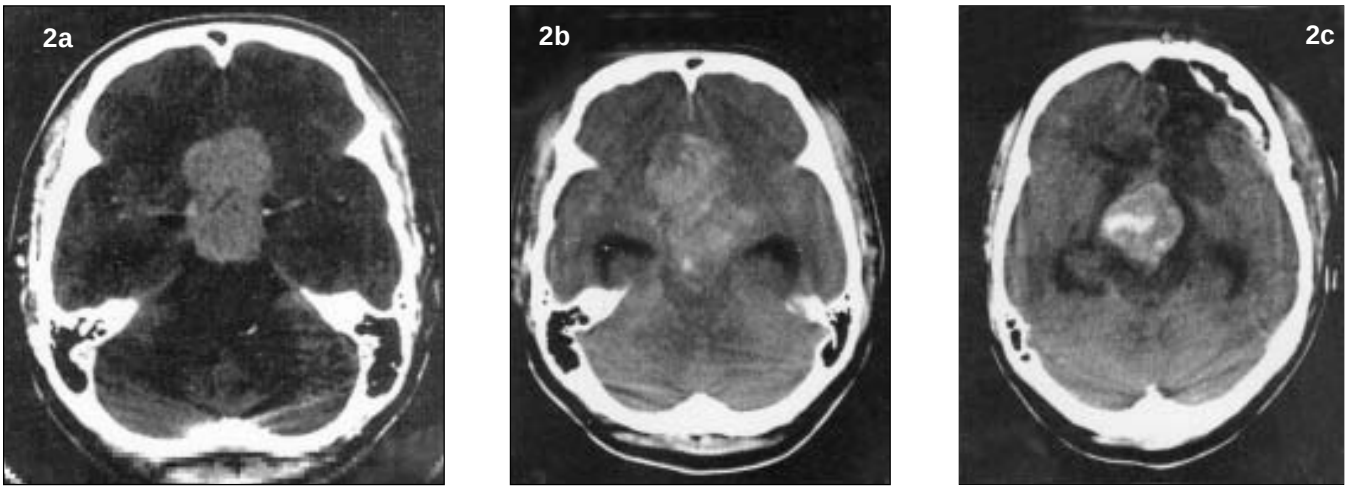

Figure 2: CECT showing a giant sellar and suprasellar tumor with sub-frontal extension and homogenous contrast enhancement (2a). NCCT head after first surgery showing swelling of residual tumor with evidence of multiple punctuate hemorrhages (2b), with hydrocephalus. NCCT after second surgery showing residual tumor with hemorrhage and multiple cerebral infarcts (2c).

bleed [Figure 2B]. There was associated hydrocephalus. The patient was operated by a transcranial sub-frontal route. A large residual tumor with clear evidence of intra-tumoral bleed was identified and radically resected. The patient did not show any significant improvement following the re-exploration. Another CT scan showed persistence of the residual tumor and the hemorrhage. The patient was operated for the third time, and radical decompression of the tumor was carried out. The patient had a stormy post operative course, developed diabetes insipidus and multiple cerebral infarcts were identified on CT scan (Figure 2C). He died 20 days later.

Case 3 and Case 4

A 35 year old female patient and a 19 year old male patient having giant pituitary tumor had almost identical clinical course as seen in our first two cases.

Discussion

Pituitary apoplexy is a rare and life threatening condition characterized by headache, visual loss, opthalmoplegia, and altered mental status caused by sudden hemorrhage or infarction of the pituitary gland.[3] Massive swelling and hemorrhage in a pituitary tumor or the phenomenon of 'postoperative pituitary apoplexy' following a subtotal or a partial resection of giant pituitary adenomas was described by Goel et al in 1995.[1] Our literature search did not reveal any other report of a similar clinical finding, and it appears that probably this complication is under-reported.

All the four patients in our series and in the series reported by Goel et al,[1] the patients had giant pituitary tumors. There was postoperative worsening in the neurological status in all the four reported cases. There was no evidence of alteration of coagulation parameters. Case 1, 2 and 4 had deterioration in sensorium and Case 3 developed third nerve paresis along with visual decline. Despite the decompression of the haemorrhagic tumor during the re-operations, all the patients had a stormy post operative course and subsequently died.

Various causes have been postulated for pituitary apoplexy. Some authors have proposed that a rapidly growing adenoma that outstrips its blood supply may lead to ischemic necrosis of the gland.[4] Others propose direct compression of the pituitary infundibulum by an expanding mass, thus compromising the blood flow from the portal vessels, resulting in necrosis of the entire gland with hemorrhage as a secondary occurrence. Sudden release of tumor vessels from the internal carotid artery due to reduced tumor burden and compromise of the venous drainage of the tumor during surgery have also been proposed.[1] Tumor manipulation during surgery, swelling and subsequent compression of the hypophyseal arteries causing hemorrhagic necrosis is a plausible explanation in our cases. However Cardoso and Peterson[5] did not agree with the above theory since angiographic studies show that pituitary adenomas derive their blood supply from inferior hypophyseal arteries and not from superior hypophyseal arteries, which get compressed with the impaction of the enlarging tumor against the diaphragmatic notch.

Lath and Rajshekhar reported a patient who died of massive cerebral infarct due to direct compression of the internal carotid artery caused by pituitary apoplexy.[6] Hypothalamic disturbances could be an important cause of mortality in such cases and at least one of our cases (Case 2) had evidence of hypothalamic dysfunction.

It has been proposed that a radical tumor resection must be attempted during the first surgery for giant pituitary adenomas [1,2]. Our experience and that of others suggest that surgery for giant pituitary adenomas is a formidable surgical challenge.[1,7]

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