Afr J Med. med Sci (1990) **19**, 105–110

A case of pituitary apoplexy

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Summary

Pituitary apoplexy, an uncommon syndrome, presented in a 40-year-old Nigerian with severe impairment of vision and persistent headaches over a 4-month period. Surgical decompression successfully restored his vision. This case is presented with a review of the literature and discussed as the first case seen in this unit since it started 5 years ago.

Résumé

L'apoplexie pituitaire, un syndrome rare, s'est présenté dans un Nigérien de 40 ans avec un gros affaiblissement de la vue et maux de tête persistant surpassant une période de 4 mois. La décompression chirurgicale avec succès lui redonne sa vue. Ce cas est présenté avec une revue de la littérature et c'est le premier cas depuis le commencement du département il y avait 5 ans.

Introduction

The dramatic symptoms and clinical signs as well as the pathological findings that accompany sudden haemorrhage into the pituitary gland make the term 'pituitary apoplexy' most appropriate for this uncommon clinical syndrome. Although Bailey, a New York City neurologist of outstanding repute was the first to recognize and report a typical case of pituitary apoplexy in 1898 [1], Bleibtreu [2], however, is generally given this accolade. Since then there have been many case reports in the literature because of the variation in pathological findings in each case [3–11]. Pituitary apoplexy commonly occurs in a pre-existing pituitary adenoma which suddenly undergoes infarction with swelling, and haemorrhage or acute bleeding into the tumour. Less commonly, spontaneous haemorrhage and infarction may occur in a non-adenomatous pituitary gland and produce similar clinical results. Prompt and accurate diagnosis allows optimal results from surgical intervention. The availability of high technology in the use of computerized tomography (CT) scan and nuclear magnetic resonance scan have further enhanced early detection. Surgical treatment has changed to microsurgical approaches to the tumour since 1969 when Hardy reported his first successful removal of hormonally active microadenoma with preservation of normal pituitary function via the trans-sphenoidal route [12]. Although high technology facilities are not yet available to the neurosurgical unit of the Jos University Teaching Hospital (JUTH) the first case of pituitary apoplexy seen here was successfully treated surgically and is presented for discussion.

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Case report

A 40-year-old male Nigerian was admitted into the neurosurgery unit on 16th February 1988 because of increasing severity of headache and rapid deterioration in vision (Fig. 1). He had enjoyed good health in the past. There was no recent history of trauma, diabetes or thyroid disease. He was hypertensive and had been on diuretics. Examination revealed a healthy looking right-handed patient who was fully conscious and well orientated. He was not dysphasic; BP 130/80, PR 72/min regular, good volume. Both pupils were equal and reacted briskly to light. He had a full range of eye movements. Visual fields showed bi-temporal

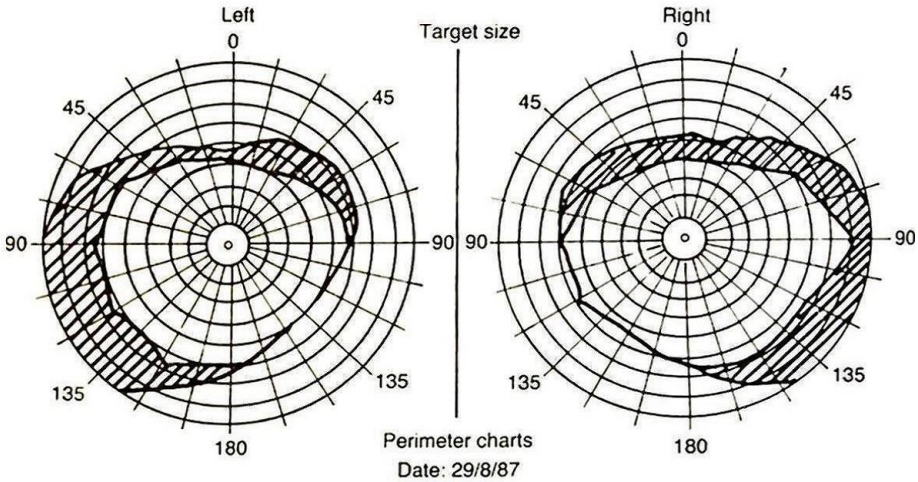

Fig. 1. Peripheral visual fields showing a small loss.

haemianopsia (Fig. 2). Fundoscopy showed a normal left eye. The right eye had a pale disc with increased pressure in the central retinal artery. The other cranial nerves were intact.

The cardio-respiratory system, thyroid, abdomen and body hair were normal. There was no neck stiffness. He had normal tone and reflexes. He had a full range of movement and power in all four limbs. Chest X-ray and electrocardiogram were normal. Skull X-ray (Fig. 3) showed an expanded sella with erosion of the clinoids. It was not possible to perform a carotid angiogram. A diagnosis of pituitary apoplexy was made and surgical decompression was necessary to salvage vision and relieve the headache. He was started on parenteral dexamethasone which was continued both intra- and post-operatively. On 19th February 1988 a right frontotemporal craniotomy was performed. A large haemorrhagic tumour was seen in the pituitary fossa region. It was evacuated with a pituitary forceps and the optic chiasm was seen to be lax on completion of the procedure. Histology showed a pituitary adenoma with extensive areas of necrosis. The tumour consisted of infiltrating nests and cords of neoplastic cells arranged around thin walled vascular channels that gave a sinusoidal pattern. Many haemosiderin-laden macrophages were seen. The overall picture was that of an invasive chromophobe adenoma with pituitary apoplexy (Fig. 4).

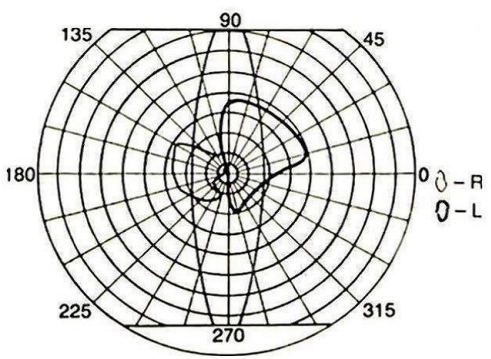

Fig. 2. Bitemporal haemianopsia in the same patient pre-operatively.

Post-operatively he developed a partial right-sided third cranial nerve paresis that recovered fully. He also developed a mild diabetes insipidus which has settled. Figure 5 shows the expansion of his visual fields prior to discharge to an out-patient clinic. The patient returned to his job and has been followed up in the clinic for 6 months.

Discussion

Only 6.8% of all patients with pituitary adenoma develop clinical manifestations of pituitary

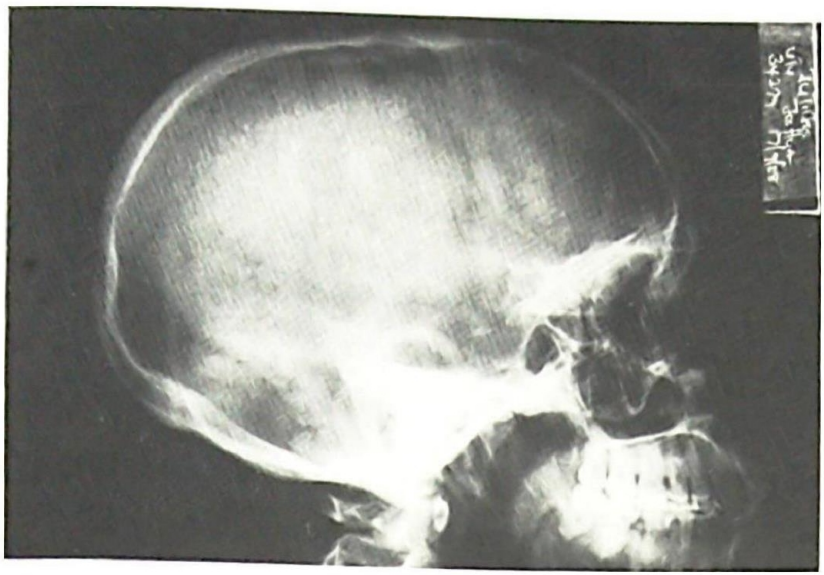

Fig. 3. Radiograph of the skull, showing expanded sella with erosion of the clinoids.

apoplexy [13]. Earle and Dillard estimated that about 1% of chromophobe adenomas present as pituitary apoplexy [14]. Haemorrhage with or without necrosis occurs either macroscopically or microscopically in 10–28% of chromophobe adenomas [9, 13, 15–17]. Pituitary apoplexy occurs most commonly in untreated cases and frequently in previously undiagnosed cases of pituitary adenomas, although a pre-existing endocrinopathy is present in at least 30% [18]. Pituitary tumours may be prone to infarction because of the local vascular anatomy. Expansion of the sellar contents due to an adenoma may include the blood supply especially with the added effect at the diaphragmatic notch where the portal vessels hug the pituitary stalk [18]. The ischaemia that occurs in the anterior lobe and the effect of the tumour increase the sellar contents and the acute signs of pituitary apoplexy ensue. Associated factors in the pathogenesis of pituitary tumour infarction are not well understood and contribute to the acute deterioration of an already compromised tumour blood supply.

Pituitary apoplexy may present with headache, visual deterioration or ophthalmophegia or both, meningeal irritation and evidence of extrasellar spread. A major differential diagnosis is subarachnoid haemorrhage from rupture of an aneurysm or presence of an intrasellar or parasellar aneurysm that has deformed the sella. In 7% of cases with pituitary adenomas a co-existing aneurysm has been found [19]. Migraine, optic neuritis, stroke, partial cilio-retinal artery block, myocardial infarction, meningitis, encephalitis, cavernous sinus thrombosis, pituitary abscess or subarachnoid haemorrhage from a ruptured arteriovenous malformation are other differential diagnoses to be considered. Delay in treatment may result in blindness, or death may be the outcome in untreated pituitary apoplexy, the precise clinical cause of which is unpredictable. Pituitary apoplexy has not been shown to have a preference for any type of hormone-secreting pituitary adenoma. Histological classifications of these tumours have been augmented by immunochemical staining techniques which have shown that as many as 50% of chromophobe adenomas produce a hormone [20–22]. Only two types of apparently non-secreting tumours are known: (i) the oncocytomas, which are thought to be transformed epithelial cells without endocrine potential and, (ii) the null-cell adenoma which have an as yet unidentified secretory product.

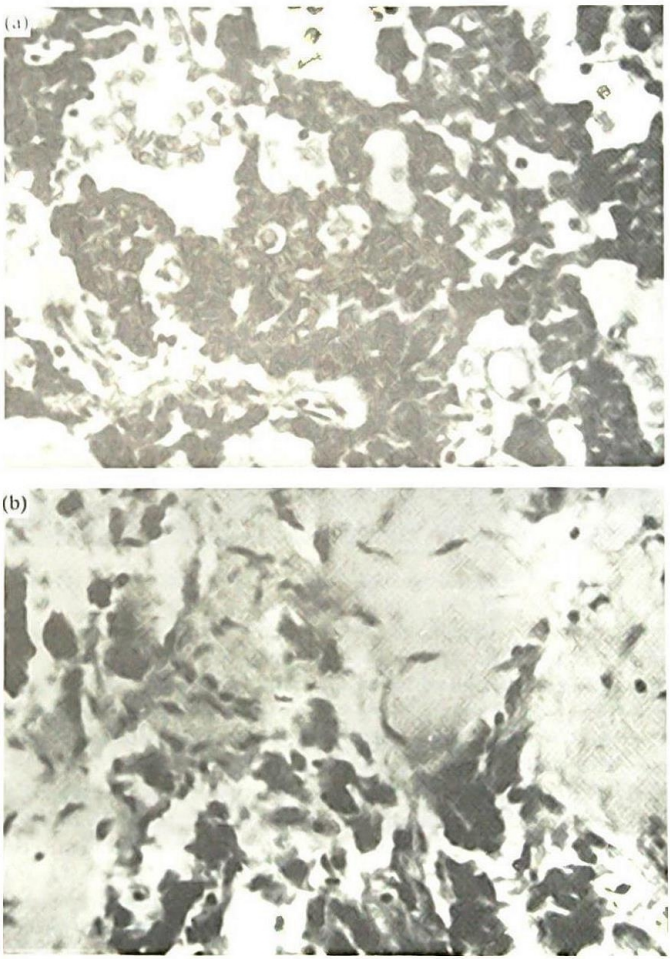

Fig. 4. Invasive chromophobe adenoma. (a) Nests and cords of neoplastic hyperchromatic chromophobe cells arranged around vascular channels. The latter contains numerous erythrocytes. H&E (×175). (b) Nests and clusters of neoplastic chromophobe cells infiltrating the fibrillar pars nervosa. The latter is the lightly stained zone, H&E (×175).

In our patient a diagnosis of pituitary apoplexy was based on operative findings, clinical features and skull radiographs. Surgical decompression was performed and the two optic nerves were visualized and preserved at craniotomy. Clinically this was grade II, i.e. a tumour of more than 10 mm in an enlarged sella. Histology showed extensive areas of necrosis and haemosiderin-laden macrophages in a chromophobe adenoma that had bled before. Unclotted blood aspirated from the tumour confirmed a more recent bleeding episode. Further immunocytochemical staining for pituitary hormones was not done. Steroid replacement therapy was continued.

A 12.7% recurrence rate for pituitary adenomas has been described by Ciric *et al.* [23]. They showed that the greatest number occurred 4–8 years post-operatively. Post-operative radiotherapy appeared to play a greater role than the completeness of tumour removal in prevention of recurrences. In those patients in whom radiotherapy was withheld a tumour recurrence rate of 21% was recorded.

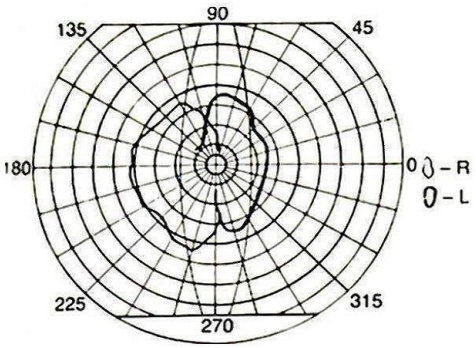

Fig. 5. Visual fields prior to discharge home after surgical intervention.

Conclusion

A case of pituitary apoplexy was treated by craniotomy and evacuation of tumour. The patient returned to work and has been followed up. He has shown no clinical evidence of tumour recurrences.

Acknowledgments

Mrs P. Ovuiro and Mrs T. Jay provided the secretarial services, and the Photographic and the Graphics Sections of the Medical Instructional Technology Unit of the University of Jos produced the pictures and figures. Mr P. Arthur was kind enough to translate the summary into French. We are grateful to them all.

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(*Accepted* 28 *November* 1988)