Haemorrhage into pituitary adenomas

S. MOHANTY, P. N. TANDON, A. K. BANERJI, AND
B. PRAKASH

From the Department of Neurosurgery, All India Institute of Medical Sciences, New Delhi, India

SUMMARY Seventy cases of pituitary adenoma were analysed for evidence of haemorrhage. It was present in 18 cases. These are classified into four groups, acute, acute on chronic, chronic, and late sequelae of pituitary apoplexy, and an example of a patient in each group is given. The diagnosis and management are discussed.

Acute haemorrhage in a pituitary adenoma, classically manifesting as 'pituitary apoplexy', is now a well recognised condition but it is relatively uncommon. In contrast, microscopic or macroscopic haemorrhage in the adenoma, especially the large ones, is probably more frequent than is commonly recognised. Muller and Pia (1953) found 19 instances of haemorrhage in a series of 270 pituitary adenomas. In a given case, the patient may give a history of one or more subacute episodes before a final acute one or else both old and recent bleeding may be found at necropsy or operation (Uihlein *et al.*, 1957).

Patients

Pituitary adenomas constituted 7.5% of the verified intracranial space-occupying lesions treated in the Department of Neurosurgery, All India Institute of Medical Sciences. Seventy cases of pituitary tumours were analysed for evidence of haemorrhage. For this purpose the clinical history and operative findings were reviewed and the biopsy material was re-examined. Evidence of varying degrees of haemorrhage was observed in 18 cases (25.7% of the total cases reviewed). On the basis of the clinical, operative and histopathological findings, cases could be classified into four groups (Table 1).

Case reports

CASE 1

RB (339/71), a 46 year old male, was admitted with headache and vomiting for six days followed by loss of vision, for four days. He was found to be apathetic and irritable. Optic disc margins were blurred. Both pupils were dilated and fixed.

Accepted 30 March 1977

Table 1 Four groups of patients with haemorrhage into pituitary adenomas

Group	Number of cases
1. Acute—pituitary apoplexy (initially presenting as an acute illness)	3
2. Acute on chronic—known cases of pituitary adenoma with sudden or rapid deterioration of symptoms	3
3. Chronic—unsuspected haemorrhage encountered at operation	11
4. Late sequelae of apoplexy	1

Meningism was present. Plain radiographs of the skull showed an enlarged sella turcica. A right carotid angiogram (Fig. 1) revealed suprasellar extension of a sellar mass. A right frontal osteoplastic craniotomy disclosed the suprasellar part of the tumour lifting the optic nerve and the chiasma. Thick chocolate coloured fluid was aspirated from the tumour. Biopsy revealed a necrotic tumour, the viable fragments being typical of chromophobe adenoma. The patient made an uneventful recovery. He was able to count fingers at a distance of three metres on the second day after the operation, and at a later follow-up was asymptomatic with no visual field defect.

Comments In this case the presentation was acute with predominant CNS symptoms associated with rapid visual loss. Radiological investigation localised the site and the nature of the lesion. Early surgery under steroid cover proved beneficial and life-saving.

CASE 2

MO (6840/72). A 54 year old male, was admitted with the history of gradual diminution of vision in both eyes, headache, and impaired libido for two

Fig. 1 Carotid angiogram. AP view showing large suprasellar mass.

years. Nine days before admission he had severe headache and occasional vomiting with rapid loss of vision in the right eye. He was moderately obese with sparse hair over the chest. With the

right eye there was no perception of light. With the left eye finger counting was possible at 250 mm, with loss of the temporal field of vision. There was primary optic atrophy in both eyes. Plain radiographs of the skull revealed a J-shaped sella turcica with erosion of the dorsum sellae. A right carotid angiogram showed elevation of the A₂ part of the anterior cerebral artery. Right frontal craniotomy revealed a large, soft, necrotic, haemorrhagic suprasellar and sellar tumour with a thin capsule. Intratumoral decompression was done. The biopsy sample was reported as chromophobe adenoma with areas of haemorrhage. The patient had CSF rhinorrhoea for 12 days after operation which stopped spontaneously. Before discharge he had perception of light in the right eye and finger counting at 500 mm in the left eye.

Comments Longstanding visual deterioration associated with headache and impaired libido was suggestive of a pituitary tumour. Aggravation of symptoms was caused by haemorrhage (Fig. 2). Timely decompression improved the neurological status.

CASE 3

MD (CR No. 13058), a 30 year old female, had amenorrhoea for three years, headache, gain in weight, intolerance of heat, and coarsening of features with progressive visual impairment for eight months. Examination revealed acromegalic features, visual acuity R6/36, L6/24, bitemporal visual field defect, bilateral primary optic atrophy, and right 6th nerve paresis. Plain radiographs of the skull revealed an enlarged sella turcica. Right carotid angiography showed a suprasellar lesion.

Fig. 2 Microphotograph showing an area of haemorrhagic necrosis in the tumour. H and E $\times 200$.

On exploration, there was a solid tumour with a cyst containing dark brown fluid and necrotic tissue. Recovery was uneventful. Before discharge the visual acuity had improved to 6/18. Biopsy showed both acidophilic and chromophobe pituitary cells with areas of haemorrhage.

Comments A small haemorrhage in the tumour may become encysted without evidence of acute deterioration in the condition of the patient, and may be discovered only at surgery. The abducens palsy indicates parasellar spread of the tumour.

CASE 4

KCK (594/69), 26 year old male, noticed unusual growth in stature for 10 years. Four years before admission he had an attack of severe headache, neck stiffness, and vomiting which was immediately followed by diminution of vision in the right eye. Since then there had been slow progressive deterioration of vision. Still later he developed severe diabetes mellitus and required more than 120 units of insulin per day. He had features of gigantism and hypogonadism. Vision in the left eye was 6/6 and in the right eye finger counting at 1 metre. He had bitemporal hemianopia. The right fundus showed primary optic atrophy. Plain radiographs of the skull showed an enlarged sella turcica with undercutting of the anterior clinoid process but a pneumoencephalogram and angiogram did not reveal any suprasellar extension of tumour; on the contrary the suprasellar air shadow dipped into the sella. In view of the severe diabetes it was decided to perform pituitary stalk section. At operation dense adhesions were found around

the right optic nerve with xanthochromic discolouration of the arachnoid mater of the cisterna chiasmatica and the olfactory tract. The sella turcica was empty. The pituitary stalk was sectioned, and biopsy of the excised arachnoid mater showed thickening with presence of haemosiderin laden cells. He made an uneventful recovery with perceptible improvement in vision in the right eye. His insulin requirement was markedly reduced, and six months after the operation he was fully controlled on small doses of oral antidiabetic drugs.

Comments It is reasonable to conclude that the acute episode of headache, vomiting, neck stiffness and visual deterioration four years before admission was due to haemorrhage in the tumour which escaped into the subarachnoid space, resulting in the pathological changes seen in the arachnoid mater of the chiasmatic cistern. The haemorrhage practically destroyed the tumour resulting in an 'empty sella'.

The clinical features in each of the four groups described above are summarised in Table 2.

Discussion

Bleibtreu (1905) was the first to describe haemorrhage within a pituitary tumour. The incidence of haemorrhagic adenoma varies (Muller and Pia 10%. Poppen 20%, Dastur and Pandya 18%). In the present series of 70 patients, 18 had evidence of haemorrhage, an incidence of 27.7%. This may possibly be related to the large size of tumours commonly seen by us.

Table 2 Clinical features in each of the four groups

Onset	Visual loss	Meningeal signs	Ocular palsy	Endocrine insufficiency
1. Acute	Rapid to sudden	Common	Common	Absent
2. Acute on chronic	Rapid	Rare	Occasional	Present
3. Chronic	Slow	Absent	Rare	Present
4. Acute episode in past	Rapid during the episode followed by impairment or deterioration	Absent	Absent	Present

Table 3 Correlation between clinical groups and histological findings

Clinical findings	Histology		
	Chromophobe	Acidophilic	Necrosis
1. Cases presenting with apoplexy	2	1	Marked
2. Known cases of pituitary adenoma with sudden or rapid deterioration of symptoms	2	2	Moderate
3. Unsuspected haemorrhage	10	1	Occasional
4. Late sequelae of apoplexy			Evidence of old haemorrhage. Haemosiderin deposit

Table 4 Results of treatment

	Mortality	Improved	Unchanged	Worsened
1. Case presenting with apoplexy	Nil	1	1	Nil
2. Known cases of pituitary adenoma with sudden or rapid deterioration of symptoms	Nil	2	Nil	1
3. Unsuspected haemorrhage encountered at operation	2	7	1	1
4. Late sequelae of apoplexy (empty sella)	Nil	1	Nil	Nil

Subarachnoid haemorrhage is a common mode of presentation (Brougham *et al.*, 1950; List *et al.*, 1952; Ramamurthi, 1970). In two of our three patients who had apoplectic presentation, lumbar puncture revealed subarachnoid haemorrhage. Furthermore, evidence of old subarachnoid haemorrhage was noticed at operation as xanthochromic pigmentation of arachnoid mater in the chiasmatic region in one case.

Benjamin (1929) reported a case of a woman aged 63 years with a haemorrhage into the tumour without blood appearing in the CSF. Microscopically the tumour was a haemorrhagic adenoma. As seen in the present series haemorrhage may remain totally restricted to the tumour, and may even become encysted in a much larger number of cases than those with subarachnoid haemorrhage. Such cases may present either with acute exacerbation of a well-established disease or may be a chance finding at surgery. Haemorrhagic adenoma producing intracerebral clot (Dastur and Pandya, 1970) and middle cerebral artery occlusion (Sehnitker and Lehnher, 1952) or prechiasmal interaction have not been seen in our series.

Preoperative radiotherapy is usually blamed for haemorrhage in a pituitary adenoma. (Brougham *et al.*, 1950; Uihlein *et al.*, 1957), but in the present series none of the patients had received prior irradiation. None of Cushing's patients with haemorrhagic cyst had received deep X-ray therapy previously.

Necrosis and haemorrhage are well known features in neoplasms that outstrip their blood supply. They occur in gliomas and metastatic tumours. The high incidence of haemorrhage in our series is probably directly related to the large size of the tumours. Locke and Tyler (1961) found a thrombosed vein during necropsy and postulated haemorrhagic infarction as the cause of tumour haemorrhage in their patient.

Brougham *et al.* (1950) thought necrosis and haemorrhage to be more common in eosinophilic adenomas but many of the patients with haemorrhage in pituitary adenoma reported in the literature did not have clinical evidence of acromegaly. Likewise in our experience the

tendency to haemorrhage and necrosis is directly related to the size and vascularity of the tumour rather than to the type of adenoma.

The evolution of the clinical picture in these cases naturally depends upon the severity and course of the haemorrhage. While haemorrhage is seen in a much larger number of pituitary adenomas, the classical 'pituitary apoplexy' is seen in only a small percentage of cases, only three out of 18 in the present series.

In these latter cases the haemorrhage is massive, generally becomes subarachnoid, and the clinical course is acute and often stormy, characterised by impairment of consciousness, blindness, and ophthalmoplegia. Emergency surgical decompression is required for most of these patients. Gratifying return of vision is possible even after total blindness. In contrast to this are patients in whom the haemorrhage is small, without any clinical indication of its occurrence or existence. The diagnosis in this group of cases becomes obvious only at surgery. They constitute the larger group of cases of haemorrhagic adenomas. These are not of much therapeutic or prognostic significance, neither is there any evidence to suggest that this type of lesion predisposes to 'pituitary apoplexy'. Between these two extremes are cases which we have classified as acute on chronic. Prompt surgical intervention in this group, usually characterised by rapid deterioration of vision in a patient known to be harbouring a pituitary adenoma, reverses the visual loss quickly. The fourth group of haemorrhage in an adenoma destroying the tumour, is the most rare.

References

- Benjamin, J. E. (1929). Pituitary tumour with fulminating symptoms. *Journal of the American Medical Association*, **92**, 1755-1758.
- Bleibtreu, L. (1905). Ein Fall von Akromegalie (Zerstörung der Hypophysis durch Blutung). *Münchener Medizinische Wochenschrift*, **52**, 2079-2080.
- Brougham, P., Heusner, A. P., and Adams, R. D. (1950). Acute degenerative changes in adenomas of the pituitary body with special reference to

- pituitary apoplexy. *Journal of Neurosurgery*, **7**, 421–439.
- Dastur, H. M., and Pandya, S. K. (1971). Haemorrhagic adenomas of pituitary gland, their clinical and radiological presentation and treatment. *Neurology (India)*, **19**, 4–12.
- List, C. F., Williams, J. R., and Balyeat, G. W. (1952). Vascular lesions in pituitary adenomas. *Journal of Neurosurgery*, **9**, 177–187.
- Locke, S., and Tyler, H. R. (1961). Pituitary apoplexy. *American Journal of Medicine*, **30**, 643–648.
- Muller, W. I., and Pia, H. W. (1953). Zur Klinik und Aetiologie der Massenblutungen in Hypophysenadenome. *Deutsche Zeitschrift für Nervenheilkunde*, **170**, 326–336.
- Poppen, J. L. (1966). Chromophobe pituitary adenoma. Current concepts of diagnosis and treatment. *Postgraduate Medicine*, **40**, 396–461.
- Ramamurthi, B. (1970). Chromophobe adenomas. *Neurology (India)*, **18**, 65–74.
- Sehnitker, M. R., and Lehnert, H. B. (1952). Apoplexy in a pituitary chromophobe adenoma producing the syndrome of middle cerebral artery thrombosis. A case report. *Journal of Neurosurgery*, **9**, 210–213.
- Uihlein, A., Balfour, W. M., and Donovan, P. F. (1957). Acute hemorrhage into pituitary adenoma. *Journal of Neurosurgery*, **14**, 140–151.