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Metastatic prostatic adenocarcinoma presenting as complete ophthalmoplegia from pituitary apoplexy

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Pituitary apoplexy is a rare presentation of pituitary disease. A case resulting in sudden bilateral complete ophthalmoplegia due to a prostatic metastasis is described for the first time. The pathogenesis of this condition and relevant literature are discussed.

Case report

An 81-year-old West Indian man presented with a 4 day history of severe headache and sudden visual loss on the left. He reported polydipsia but no nausea or vomiting. His ophthalmic history included advanced open angle glaucoma blinding his right eye, previous left cataract extraction complicated by removal of the implant, and right branch retinal vein occlusion. Thirty two years earlier he had undergone a bilateral orchidectomy for prostatic carcinoma.

On examination he was afebrile, oriented but inattentive and had complete bilateral ophthalmoplegia and complete ptosis. Best visual acuities were light perception right eye and 3/24 left eye. Corneal sensation was absent but there were no other cranial nerve or long tract signs. He was normotensive with a pulse of 54 and there were no other signs of endocrine disease. Endocrine function tests confirmed panhypopituitarism and cranial diabetes insipidus (cDI).

A bone scan revealed widespread metastases and high resolution contrast computed tomography scan of the head demonstrated destruction of the pituitary fossa by a solitary enhancing mass eroding inferiorly into the sphenoid sinus and posteriorly through the dorsum sella to reach the pons. There was both supra and parasellar extension (Fig 1A and B).

A provisional diagnosis of pituitary apoplexy

Fig 1A

Fig 1B

Figure 1 Metastatic deposit demonstrated by contrast enhanced high resolution computed tomography. Horizontal (A), and sagittal sections with vertical reconstruction (B), demonstrating a solitary enhancing deposit filling the fossa and expanding superiorly, laterally, and posteriorly.

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Figure 2 Prostatic adenocarcinoma: microglandular pattern of monomorphic cells with pleomorphic nuclei and extensive necrosis completely replacing the normal pituitary architecture.

arising from infarction of a non-functioning adenoma was made. The patient's visual acuity deteriorated despite high dose dexamethasone and he underwent trans-sphenoidal resection of the tumour.

At operation a radical resection of the tumour was undertaken. The consistency of the tumour was haemorrhagic with evidence of old blood indicative of previous bleeds within the lesion. The diagnosis of pituitary apoplexy was confirmed. Postoperatively the patient made an uneventful recovery and underwent a course of fractionated radiotherapy. There was no improvement of his visual acuity or eye movements.

Histology showed that metastatic prostatic adenocarcinoma with extensive necrosis had completely replaced the normal pituitary architecture (Fig 2).

Comment

The sudden swelling of a pituitary tumour due to ischaemic necrosis or infarct was first described as pituitary apoplexy by Brougham *et al.*¹ The typical history is one of sudden severe headache and vomiting coupled with signs of meningism and visual loss or ophthalmoplegia.

Ophthalmoplegia is present in up to 6% of patients with pituitary adenoma. Patients with sella metastases commonly present with cDI whereas this occurs in under 2% of patients with a pituitary adenoma.² Although well described as a presenting sign of pituitary apoplexy the occurrence of complete bilateral ophthalmoplegia

resulting from pituitary apoplexy due to a prostatic metastasis has not been previously reported in English language publications.

Prostatic carcinoma classically metastasises to bone, lung, and liver³; brain metastases are seldom evident antemortem,⁴ particularly as a presenting feature.⁵ Surveys of several large series of patients with prostatic carcinoma find the incidence of pituitary metastases to be between 1 and 6%.^{6,7} Several factors including the failure to examine pituitary tissue microscopically at post mortem⁸ and the high incidence of cerebral vascular events in this age group may, however, lead to underreporting.

The route of pituitary metastases involves several mechanisms. The paravertebral venous plexus draining the prostate gland has been proposed as the primary route for parenchymal brain metastases.⁹ Varkarakis *et al.*¹⁰ in a multi-step model advocate a cascade involving intermediate sites, accounting for the late presentation of central nervous system metastases and thus the invariably widespread disease and short survival of these patients.¹¹

Although pituitary apoplexy is a rare cause of ophthalmoplegia it has a characteristic clinical picture which is often misdiagnosed leading to subsequent delay in management.

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