Metastatic prostate cancer presenting as diplopia with regression of signs with hormone manipulation

Introduction

This article presents a case of metastatic prostate cancer presenting as diplopia which was initially referred to the ophthalmologists. Initial imaging suggested a pituitary tumour. The subsequent diagnosis of the primary tumour is described as being prostatic in origin. There was regression and complete resolution of eye signs with hormonal therapy alone which has not been reported previously.

Discussion

Worldwide, carcinoma of the prostate is the fourth most commonly diagnosed cancer in men and it is the leading cancer diagnosis in men in the United States of America (Reiter and deKernion, 2002). Often at presentation the cancer will have already metastasized, usually to the axial skeleton. There may be only minimal lower urinary tract symptoms. In this case the patient underwent two computed tomograms (CTs) because an examination with bone windows is required to assess the bony involvement at the skull base. Magnetic resonance imaging (MRI) was also performed since it is better for evaluating intracranial soft tissue invasion while CT is superior for assessing bony destruction. Both scans provided complementary information (Ishida et al, 2002).

Figure 1. Computed tomogram revealing extensive bony destruction.

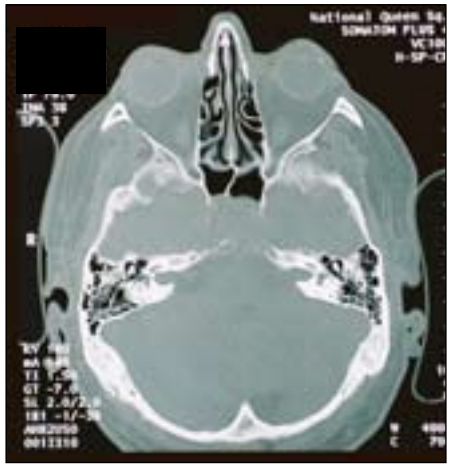
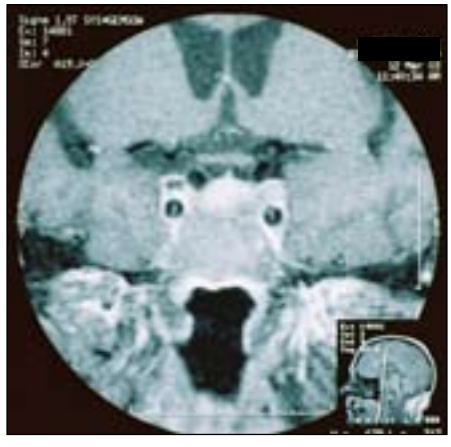


Figure 2. Magnetic resonance image showing invasive pituitary tumour.



Cranial nerve deficits resulting from metastatic carcinoma of the prostate have been described in the literature with complete or partial resolution of signs with radiation therapy (Seymore and Peeples, 1988; McAvoy et al, 2002). However, there is little written about the use of hormone manipulation in this setting. This is surprising since prostate cancer is extremely responsive to hormonal therapy. This case demonstrates regression of the tumour after hormone manipulation and illustrates that skull base irradiation is not always required in contrast to recommendations from previous authors (Seymore and Peeples, 1988).

Conclusions

This case illustrates that any elderly man presenting with unclear symptoms possibly resulting from a secondary tumour should also be assessed for prostate cancer among the various possible primary malignancies. It also shows that it is possible to reverse the effect of cranial nerve lesions caused by metastatic prostate cancer with hormonal therapy and this may spare the patient the more invasive treatment of irradiation to the skull base. BJHM

Case Report

A 73-year-old man presented with sudden onset diplopia. He was referred to the ophthalmology team who thought he had a VIth nerve palsy. He subsequently underwent computed tomography (CT) (*Figure 1*) and magnetic resonance imaging (*Figure 2*). These images revealed a mass occupying and expanding the sella with extension inferiorly to the sphenoid sinus. The clivus was almost completely replaced by tumour. The cavernous sinuses were involved bilaterally but the optic chiasm was not involved. It was thought that these finding were consistent with an unusual invasive pituitary tumour and surgical excision biopsy was undertaken. The histology was consistent with metastatic carcinoma of the prostate.

His prostate-specific antigen (PSA) taken post-histology was 382 μg/litre and a bone scan confirmed metastases in the ribs, thoracic and lumbar spine, sacrum, left sacroiliac joint, ilium, ischium and pubis. He was subsequently started on luteinizing hormone-releasing hormone analogues which brought about a resolution of his diplopia. Follow-up CT of the head revealed regression of the skull base mass. At 18-month follow-up he remains well with no visual impairment, the PSA having decreased to >0.2 μg/litre.

Ishida H, Mohri M, Amatsu M (2002) Invasion of the skull base by carcinomas: histopathologically evidenced finding with CT and MRI. *Eur Arch Otorhinolaryngol* **259:** 535–9

McAvoy CE, Kamalarajab S, Best R, Rankin S, Bryars J, Nelson K (2002) Bilateral third and sixth nerve palsies as early presenting signs of metastatic prostatic carcinoma. *Eye* **16:** 749–53

Reiter RE, deKernion JB (2002) Epidemiology, etiology and prevention of prostate cancer. In: Walsh PC, Retik AB, Vaughan ED, Wein AJ eds. *Campbell's Urology* 8th edn. Saunders, Philadelphia: 3003–16

Seymore CH, Peeples WJ (1988) Cranial nerve involvement with carcinoma of prostate. *Urology* **31:** 211–13

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