separated from the host, and nits hatch in 7–10 days, careful sealing of fomites in plastic bags for 2 weeks can also be effective.

Lastly, pubic lice in children may be an indication of sexual abuse, and it is of interest that the patients discussed herein were reported to have shared a bed with an uncle on several occasions. There has been a resurgence of pubic louse infestation from increased sexual activity in the adolescent population, and associated venereal diseases have been detected in a large percentage of involved subjects.[7]

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Sir,

Rapid bilateral sequential visual loss secondary to optic canal metastases in prostatic carcinomatosis

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Stage D III metastatic prostatic carcinoma is usually an indolent disease most frequently affecting the pelvis, lumbar vertebrae, ribs, and the skull convexity.[1] In contrast, metastases to the skull base and orbit are less common. We present the clinical and imaging findings and discuss the management of a case of rapid, sequential, painless loss of vision secondary to prostatic metastases to the optic canals. The rapidity of bilateral progression and the complete lack of orbital and ophthalmic signs are unusual.

Case report

A 64-year-old Caucasian male presented to eye casualty with a 1-week history of painless sudden central loss of vision, followed by total loss of vision in his left eye. He had biopsy-proven metastatic prostatic adenocarcinoma diagnosed 2 years previously. His metastases were to long bones without skull involvement for which he had received irradiation. He was on maintenance nonsteroidal anti-inflammatory agents and oral morphine for bone pain. He was pale but not clubbed. His left visual acuity was hand movements compared to 6/6 in his fellow eye. On examination, he demonstrated a left relative afferent pupillary defect. Confrontational visual fields confirmed a full right field with an extinguished left field. He had pulsatile and nontender temporal arteries, full ocular movements, no proptosis, no lid fullness, and white nonchemotic globes. Applanation tonometry and anterior slit-lamp examination were normal. Fundoscopy revealed normal fundi and optic discs. Haematological screening revealed normochromic anaemia, elevated erythrocyte sedimentation rate of 102 mm/h, and a C-reactive protein of 44, thought to be consistent with chronic metastatic disease. His prostate-specific antigen level was elevated at 326.6 ng/ml. Fluorescein angiography excluded delayed vascular filling. After 1 week, acuities were hand movements in the right eye and no light perception in the left eye. The right nasal optic disc edge was now slightly swollen. A left temporal artery biopsy was undertaken, as temporal arteritis was thought to be a possibility, 1 day after initiation of 1 g intravenous methylprednisolone. The biopsy was normal. Same day computerised tomography, however, showed distorted middle cranial fossa architecture, specifically a destructive soft tissue mass at the left orbital apex involving the lesser wing of the sphenoid with destruction of the lateral margin of the sphenoid sinus and clinoid and extension into the sinus cavity. The proximal portion of the left optic nerve was thickened and distorted. Additionally, there was expansion and sclerosis of the right lesser wing of the sphenoid with resultant narrowing of the optic canal. The trabecular pattern of the clivus was coarsened (Figure 1). He received further pulsed intravenous steroid over 3 days, followed by oral prednisolone with bisphosphonate cover. He underwent palliative

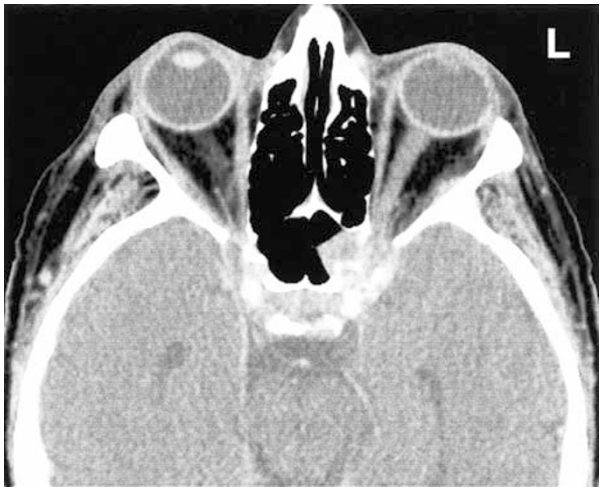

Figure 1 Noncontrast enhanced axial CT image demonstrating bilateral optic canal and middle cranial fossa changes.

radiotherapy to the region without clinical success. He died 3 months following presentation.

Comment

Ophthalmic involvement from prostatic adenocarcinoma includes spread to the uvea, orbit and pituitary region.[1–6] Optic canal involvement is rare. The present case illustrates the unusual presentation of bilateral canal dissemination with rapid optic neuropathy. The short interval between fellow optic nerve involvement, the lack of orbital and ophthalmic signs, the young age of our patient, and the short lifespan are distinctive. The optic neuropathy was thought to be secondary to compression from deformity of the optic canals. Parenchymal nervous tissue or meningeal infiltration is much less common, but it may also present with similar signs.[1] It is now well recognised that prostatic dissemination to bone often displays a mixed osteoblastic and lytic tendency.[3,5] Treatment of prostatic metastases to the orbit is palliative and does not alter survival. Testosterone determines the rate of tumour growth in most cases; consequently, hormonal manipulation has been shown to alter the course of systemic disease.[7] Traditional androgen blockade modalities such as luteinizing hormone-releasing hormone (LH-RH) agonists (leuprolide or goserelin) were not felt to be appropriate as the relative chemical hypophysectomy can cause an initial testosterone surge, which results in acute tumour volume increase.[2] This could have further compromised the optic nerves. Interestingly, pretreatment with antiandrogens (chlormadinone acetate, cyproterone acetate or flutamide) may prevent this initial flare phenomenon.[8] A treatment with gonadotropin-releasing hormone antagonists (buserelin or abarelix) provides a rapid medical castration and potentially avoids the testosterone surge characteristic of LH-RH agonists.[9] The only palliative options available in our patient were high-dose systemic steroids to reduce local perineuronal swelling, direct irradiation to the bony optic canals, or gonadotropin-releasing hormone antagonists. The prognosis, once orbital metastases occurs, has been reported as 4–26 months.[2,4,10] The present case illustrates the difficult management of advanced disseminated prostatic disease to the posterior orbit and an unusual pattern of bilateral bony disruption of the optic canals.

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