Sir,

Pubic lice effectively treated with Pilogel

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Phthiriasis palpebrarum is the most common eyelid infestation caused by *Phthirus pubis* (pubic lice), sometimes referred to as crab lice.

Case report

Two brothers, aged 8 and 3 years, presented to the casualty with a 3-week history of itchy red eyes. They had no previous ocular history and were generally fit and healthy. They lived with their parents and one other brother, who had no ocular complaints.

The visual acuity of the older boy was 6/6 and that of the younger boy was not recordable.

On examination, lice and nits were noted on the eyelashes of both patients, and microscopic examination subsequently confirmed these as pubic lice (Figure 1).

The children were managed with Oc. Pilogel 4% applied twice daily to the lashes, leading to elimination of the lice.

Comment

Phthiriasis palpebrarum, is the most common cause of eyelid infestation, and is caused by *Phthirus pubis* (pubic lice). *Phthirus* are 2 mm long and have a broad-shaped, crab-like body. Their thick, clawed legs make them less mobile than the *Pediculus* species, but enable them to infest areas where the adjacent hairs are within their grasp (eyelashes, beard, chest, axillary region, pubic region).[1] They rarely infest the scalp.

Pediculosis palpebrarum is an eyelid infestation caused by *Pediculus humanus corporis* (body lice) or *Pediculus humanus capitus* (head lice). *Pediculus* species are 2–4 mm long and typically infest the hair. Infestation of the cilia, however, is rare.

Ocular signs and symptoms include the following: bilateral ocular itching, irritation, visible lice and nits, visible erythematous lesions resulting from louse bites, reddish-brown deposits on the lashes (louse faeces) secondary blepharitis, follicular conjunctivitis, and marginal keratitis.

Adult females lay eggs on the hair shafts, which are resistant to mechanical and chemical removal. They lay as many as 26 eggs (up to 3 eggs a day),[1] which hatch every 7–10 days. The average lifespan of adult lice is less than a month, and they die within 24–48 h if removed from their hosts. They interbreed freely within different species. Crowded conditions and poor personal hygiene may be reasons for infestation.

A number of treatment options are available. These include trimming or plucking of eye lashes,[2,3] traumatic amputation, cryotherapy,[4] argon laser photocoagulation,[5] fluorescein 20%,[5] physostigmine 0.25%,[6] lindane 1%,[7] petroleum jelly,[8] yellow mercuric oxide ointment 1%,[8] malathion drops 1% or malathion shampoo 1%,[9] and pilogel 4%.

The exact mechanism of action of pilocarpine 4% gel is not yet known. It could be attributed to its direct cholinergic action of depolarising the effector cell, causing paralysis of the lice, or because of direct pediculocidal action or even the smothering effect of the gel.

Pilocarpine 4% gel is cheap and easily available and has much less side effects than indirectly acting cholinergic agonists like physostigmine or organophosphorous compounds, as they have a longer duration of action than pilocarpine.

These patients require follow-up for 7–10 days, and education regarding transmission to avoid interpersonal contact until completely cured.

Laundering of potential fomites (eg towels, pillow covers, sheets, hats) at a temperature exceeding 131°F for more than 5 min kills the eggs, nymphs, and mature lice. Since adult lice cannot survive more than 48 h if

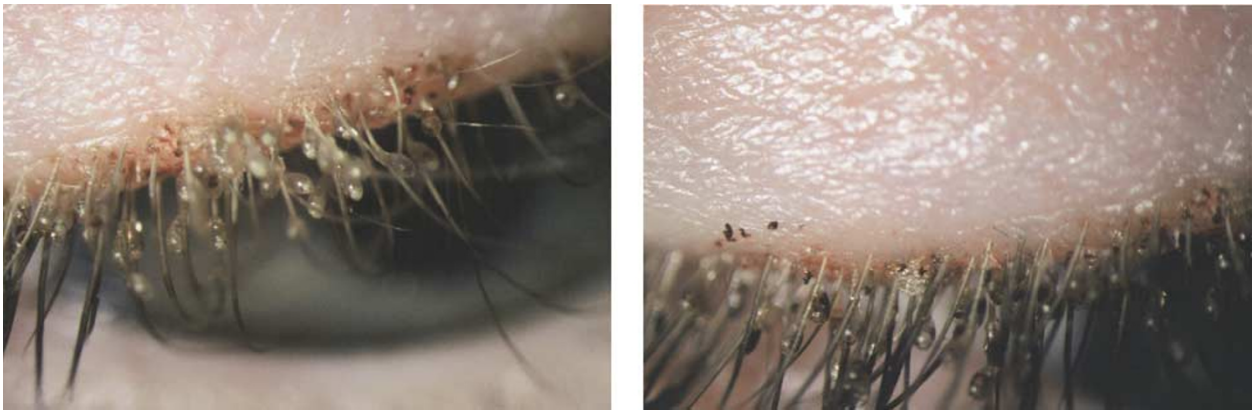

Figure 1 Lice and nits noted on the eyelashes.

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