9 Weiss A, Martinez C, Greenwald M. Microphthalmos with cyst: clinical presentations and computed tomographic findings. *J Pediatr Ophthalmol Strabismus* 1985; **22**: 6–12.
10 Nowinski T, Shields JA, Augsburger JJ, DeVenuto JJ. Exophthalmos secondary to massive intraocular gliosis in a patient with a colobomatous cyst. *Am J Ophthalmol* 1984; **97**: 641–642.

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Sir,

Should we discontinue tamoxifen in a patient with vision-threatening ocular toxicity related to low-dose tamoxifen therapy?

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Tamoxifen, a triphenylethylene nonsteroid oestrogen antagonist, has been widely used as an adjuvant postoperative therapy of oestrogen receptor-positive breast cancer. Its ocular toxicities, such as retinopathy, keratopathy, optic neuritis and cataract, have been reported since 1978, and tend to occur in patients who have a higher total dose and longer treatment.[1,2] These complications seldom cause significant visual impairment and, except for crystalline retinopathy, are reversible upon discontinuation of tamoxifen. We report a breast cancer patient who, despite the presence of vision-threatening ocular toxicity of low-dose tamoxifen therapy, made a full visual recovery of the left eye after cataract surgery without tamoxifen discontinuation.

Case report

A 45-year-old woman, a breast cancer patient with a history of receiving modified radical mastectomy in 1999, complained of progressive visual loss in her left eye for 5 weeks. Before her first presentation, she had received a cumulative dose of 1.86 g tamoxifen (20 mg/day) as postoperative adjuvant therapy for 3 months. Examination revealed a best-corrected visual acuity (BCVA) of RE 6/8.6 and LE 6/60. Anterior segment examination disclosed asymmetrical central posterior subcapsular opacity of both eyes, which was more severe in the left eye. No corneal opacity existed, and intraocular pressure and colour sensation were normal in both eyes. Funduscopic examination was bilaterally unremarkable. Electroculography (EOG) showed a subnormal Arden ratio: 180% in the right eye and 151% in the left eye. Flash electroretinography (ERG) for the maximal combined response disclosed slightly decreased amplitudes of both the cone and rod response in both eyes. Except 4-dioptre myopia, she did not have any ocular disease in the past. Her vision was 6/6 with myopic glasses before the initiation of tamoxifen treatment. A family history of ocular disease also did not exist. Although these ocular abnormalities were considered to be related to tamoxifen, the treatment persisted and the dosage was still 20 mg/day for fear of recurrence of breast cancer.

After 8 months, the BCVA deteriorated to RE 6/12 and LE 4/60. She decided to receive phacoemulsification and posterior chamber intraocular lens implantation of the left eye. Her BCVA of LE returned to 6/6 after cataract surgery. Then 4 months later, the acuity of LE went back to 6/15. After Nd:YAG capsulotomy, her vision returned to 6/6 again. However, the right eye acuity continued to get worse. A year after the cataract surgery, the BCVA of RE deteriorated to 6/30 while LE remained 6/6. The central posterior subcapsular opacity of the right eye became denser (Figure 1). The specific whirling corneal opacity and crystalline retinal deposit still did not exist (Figure 2), but the Arden ratio decreased to 119% in the right eye and 124% in the left eye. Amplitudes of ERG did not worsen any further in both eyes. We inferred that the impaired vision of the right eye resulted from the progressively denser posterior subcapsular lens opacity. The low-dose tamoxifen treatment was kept on to meet its standard regimen.

Comment

Low-dose tamoxifen ocular toxicity is well documented and its incidence was reported to be 6.3 and 12% in two

Figure 1 Central posterior subcapsular lens opacity of the right eye displayed by retroillumination.

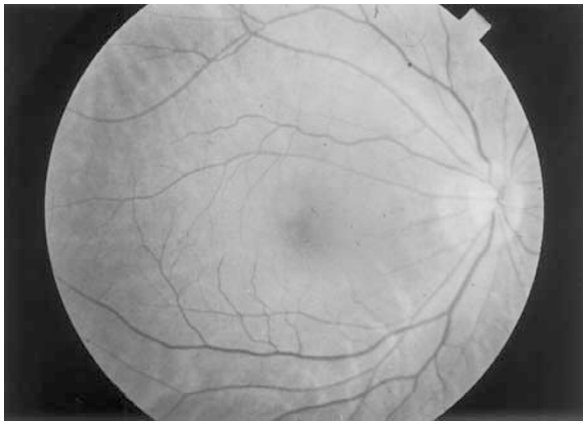

Figure 2 No apparent crystalline retinal deposit in the posterior pole.

prospective studies.[2,3] The common toxic effects included inner retinal crystalline deposition, macular oedema, whorl-like corneal opacities, posterior subcapsular lens opacities, optic neuritis and affected EOG.[1–5] Although the mechanism of tamoxifen ocular toxicity is not well understood yet, most of these side effects are reversible upon discontinuation of tamoxifen treatment and seldom lead to significant visual disturbance.

The temporal and dose–effect relationship between tamoxifen therapy and ocular abnormalities in our patient appear to be more than coincidental and suggested that ocular toxicity was due to tamoxifen. Although we cannot explain why our patient developed asymmetrical posterior subcapsular lens opacities after taking a small dose (a total less than 1.86 g) in a short period of time, her cataracts had indeed become denser along with the tamoxifen therapy and her vision had deteriorated proportionately. The Arden ratio also kept on decreasing.

According to the literature, tamoxifen was stopped in visually symptomatic tamoxifen-treated patients and most of them experienced an improvement in their vision after discontinuation.[6] However, a comparison between randomised trials of different tamoxifen durations (2 *vs* 5 years) showed that a more prolonged treatment conferred further survival benefits in breast cancer patients.[7] Our patient had suffered from progressive visual loss 7 weeks after the initiation of low-dose tamoxifen treatment. We decided against stopping tamoxifen because of the fear of recurrent breast cancer, and it turned out that her vision improved immediately after cataract surgery. Tamoxifen might induce cataracts after a short period of ingestion and, during this period, cataract surgery could help to regain the vision without discontinuing the drugs.

It is an interesting question as to whether tamoxifen should be discontinued to reverse the decreased vision in patients who received the treatment for less than 2 years. Moreover, if discontinued, how great would the impact be on the survival rate? With the greater widespread use of tamoxifen, we think this question merits further investigation.

References

1 Kaiser-Kupfer MI, Lippman ME. Tamoxifen retinopathy. *Cancer Treat Rep* 1978; **62**: 315–320.
2 Noureddin BN, Seoud M, Bashshur Z *et al*. *Eye* 1999; **13**: 729–733.
3 Palvidis NA, Petris C, Braissoulis E *et al*. Clear evidence that long-term, low dose tamoxifen treatment can induce ocular toxicity. *Cancer* 1992; **69**: 2961–2964.
4 Gorin MB, Day R, Costantino JP *et al*. Long-term tamoxifen citrate use and potential ocular toxicity. *Am J Ophthalmol* 1998; **125**: 493–501.
5 Kuchenbecker J, Pump-Schmidt C, Olbricht S *et al*. Electrophysiological studies in breast carcinoma patients with tamoxifen retinopathy. *Ophthalmologe* 2001; **98**(1): 81–85.
6 Nayfield SG, Gorin MB. Tamoxifen-associated eye disease: a review. *J Clin Oncol* 1996; **14**: 1018–1026.
7 Swedish Breast Cancer Cooperative Group. Randomized trial of two versus five years of adjuvant tamoxifen for postmenopausal early stage breast cancer. *J Natl Cancer Inst* 1996; **88**: 1543–1549.

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Sir,

The ocular hazards of egg throwing
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We present three cases of ocular injury sustained as a result of egg throwing. The raw egg can act as a substantial missile, resulting in significant ocular trauma. The ocular dangers of egg throwing need to be highlighted and addressed.

Case reports

Case 1 A 16-year-old female was walking on the sidewalk when a car stopped and an egg was thrown at her left eye. Her vision was 20/80 unaided (UA) and 20/40 pinhole (PH). She had lid ecchymoses, numerous subtarsal foreign bodies and multiple corneal abrasions. She had a dense fibrinous anterior uveitis, which eventually settled on topical steroids and she made a good visual recovery.

Case 2 A 51-year-old pedestrian was struck by an egg thrown from a passing car. On presentation, she was in severe pain and was vomiting. Her visual acuity was perception of light (PL). She had substantial periocular ecchymoses and lacerations with infraorbital anaesthesia. Her cornea had a partial thickness laceration. There was a hyphaema associated with a significant iridodialysis (Figure 1). Her intraocular pressure was elevated at 32 mmHg. A CT of orbits confirmed a fracture of her orbital floor with no muscle entrapment. She was managed conservatively and at 5-month follow-up her BCVA was 20/200. Her intraocular pressure was controlled with topical antiglaucomatous medication.

Case 3 A 15-year-old schoolboy had a raw egg thrown at him in the schoolyard. The egg broke when it struck his left eye. His left visual acuity on presentation to casualty was counting fingers (CF). He had ecchymoses of his lids, subconjunctival haemorrhages and a 5 mm hyphaema. His intraocular pressure was medically controlled and his final visual acuity at 3 months was 20/20 UA.

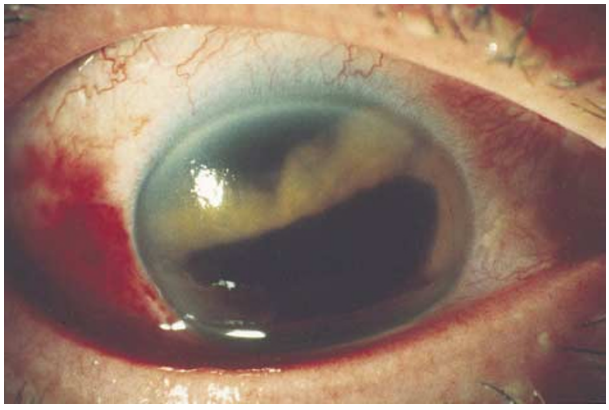

Figure 1

Comment

Assault accounts for 22% of ocular trauma admitted to hospital.[1] The most common cause of ocular injury in children is a thrown missile.[2] Although raw egg throwing may sound like a minor offence, the ocular and orbital consequences can be severe as described in our case reports.

An egg, like a squash ball, fits into the orbital cavity. Relatively little force is therefore dissipated to the orbital rim, the egg transferring most of its kinetic energy directly to the globe on impact. The result is a severe blunt injury to the eye.

Egg throwing may occur as a random act of violence, but also commonly occurs in the schoolyard on birthdays and holidays. Two of our patients were minors. Egg throwing is a common practice at Halloween, and the public needs to be educated as to the potential ocular dangers of egg throwing.[3]

References

1 Desai P, MacEwen CJ, Baines P, Minassian DC. Epidemiology and implications of ocular trauma admitted to hospital in Scotland. *J Epidemiol Community Health* 1996; **50**(4): 436–441.
2 Niiranen M, Raivio I. Eye injuries in children. *Br J Ophthalmol* 1981; **65**(6): 436–438.
3 Fiore P, Wagner R. Halloween hazards ocular injury from flying eggs. *N Eng J Med* 1988; **319**(17): 1159.

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