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Sir,

Persistent unilateral myopia following blunt trauma

Myopia after blunt ocular trauma is usually transient, resolving within weeks of the injury, and may be caused by ciliochoroidal oedema and thickening of the crystalline lens.[1] This condition is distinct from bilateral accommodative spasm occurring after closed head injury, which may involve one or more elements of the near synkinesis and has variable periods of recovery.[2,3] Here, we report a case of unilateral traumatic myopia that had not resolved 3 months after blunt ocular injury.

Case report

A 28-year-old woman had been struck in the left eye by hard, baseball-sized ball and presented with ocular pain and reduced vision. Her unaided acuities were 20/20 OD and counting fingers OS, but the injured eye improved to 20/30 with a $-6.50\,\text{sph}/-0.50\,\text{cyl}\times 90^\circ$. Cycloplegic retinoscopy and refraction revealed 20/30 vision OS with a $\text{plano}/-0.50\,\text{cyl}\times 90^\circ$ correction. The pupils were equal and reactive to light and accommodation. The anterior segments were clinically normal. The intraocular pressures were 10 mmHg OD and 12 mmHg OS, and the fundi were normal. Axial length was 24.18 mm in the right eye and 24.05 mm in the left. The anterior chamber depth was 2.85 and 2.94 mm and lens thickness was 3.68 and 4.60 mm in the right and left eyes, respectively.

After 3 months, the refractive findings were unchanged, and she was prescribed cyclopentolate 1% daily.

Discussion

Closed head trauma may cause bilateral accommodative spasm in young adults, which may be permanent.[2] In addition, blunt ocular injury may cause a bilateral pseudo-myopia,[4] which may be secondary to ciliary oedema and spasm.[1] In our patient, the increased lens thickness was probably due to the induced accommodation. Given that the anterior chamber depth was greater in the injured eye, it is unlikely that the induced myopia was caused by anterior displacement of the lens iris diaphragm. To our knowledge, this form of traumatic myopia has not been previously reported to last more than several weeks. In this case, the pseudo-myopia was still present 3 months after the injury. Symptoms were relieved by cycloplegics.

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Sir,

Plexiform neurofibroma masquerading as a persistent chalazion—a case report

Neurofibromatosis Type 1 (NF1) is an autosomal dominant disorder that commonly presents with

ophthalmic lesions.[1] We would like to present a case of a patient with previously undiagnosed NF1 presenting with upper eyelid plexiform neurofibroma masquerading as a persistent chalazion. To our knowledge, this is the first such case to be reported.

Case report

A 7-year-old Filipino boy was referred to the eye department with a 1-year history of swelling and a lump at the lateral end of the right upper eyelid. On examination, initial impressions were of two palpable nodules of which the appearances were found to be consistent with a diagnosis of chalazion. He had incision and curettage of the lesions under general anaesthesia 4 months later. No samples were sent for histology and he was discharged. However, he re-presented 1 year later with persistent chalazion. On examination, there was persistent swelling of the right upper eyelid (Figure 1a) with two mobile firm lumps underneath the skin extending from the lid to the lateral orbital rim. An excisional biopsy under general anaesthesia was carried out. Intraoperatively, the diagnosis of plexiform neurofibroma was suspected and later was confirmed on histology. A general physical examination revealed *café au lait* spots (Figure 1b) and Lisch nodules. These with the presence of plexiform neurofibroma fulfilled the criteria for the diagnosis of NF1. There was no family history of this condition.

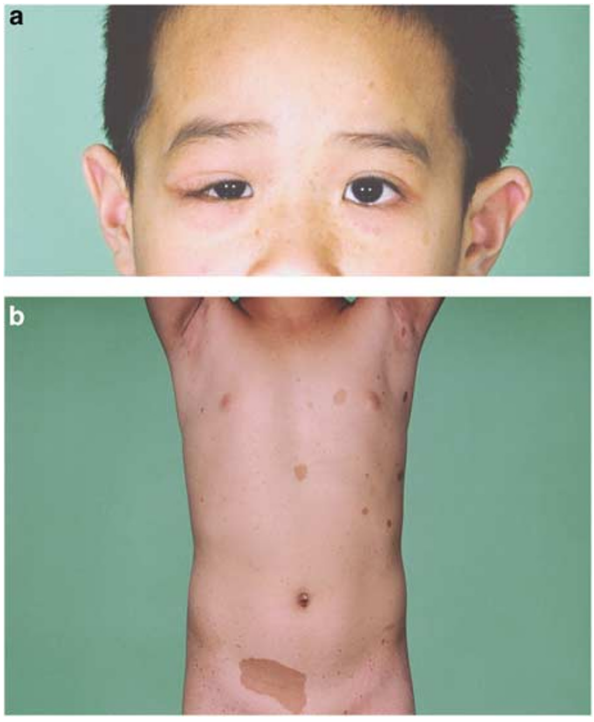

Figure 1 (a) Right upper eyelid swelling, (b) *café au lait* spots.

Comment

NF1 is categorized as a neurocristopathy (ie, a disorder that primarily affects tissues derived from the neural crest) and has a birth incidence of between 1 in 2500 and 1 in 3000 and a prevalence of 1 in 5000. The complete list of criteria for the diagnosis of NF1 is summarized in Table 1.

Plexiform neurofibroma is pathognomonic of NF1, and often appears within the first 2 years of life followed by cutaneous neurofibromas. Pathologically, a plexiform neurofibroma represents diffuse involvement of a long nerve segment and its branches with tortuous expansion, and its gross appearance has been described as a 'bag of worms'. This lesion frequently involves the trigeminal or upper cervical nerves and occurs less commonly in eyelids compared to the trunk area. Plexiform neurofibroma occurs in only 10% of patients with NF1.[2] Plexiform neurofibromas need to be monitored frequently because 5 % develop into malignant peripheral nerve sheath tumours. The most serious problem is the development of glaucoma in the eye ipsilateral to the plexiform neurofibroma. This develops in upto 50% of patients.[2]

In conclusion, a recent report stated that a number of different benign, premalignant, and malignant conditions may clinically masquerade as a chalazion.[3] Hence, we would like to emphasize the importance of histological sampling when there is recurrent or persistent chalazion. Plexiform neurofibroma needs to be borne in mind as a possible differential diagnosis of a chronic orbital or lid swelling. This case illustrates the importance of general physical examination, even in a

Table 1 Criteria for diagnosis of NF1: fulfilling at least two of seven criteria makes a diagnosis of NF1

- Six or more *café-au-lait* spots that are greater than 1.5 cm in postpubertal individuals or 0.5 cm or larger in prepubertal individuals
- At least two neurofibromas of any type or at least one plexiform neurofibroma
- Freckling in the axilla or groin (Crowe's sign)
- Optic glioma
- At least two Lisch nodules (benign iris hamartomas)
- A distinct bony lesion including sphenoid wing dysplasia or thinning of the long bone cortex
- A first-degree relative with NF1

Source: The National Institute of Health Consensus Development Conference on neurofibromatosis (1987).

busy eye clinic. In this case, considerable delays in establishing diagnosis could have been avoided.

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Sir,

Recurrent branch retinal vein occlusion with factor V leiden mutation

The normal homeostasis between procoagulant and anticoagulant activities in the body is a delicate balance of a large number of plasma factors. Although symptomatic retinal venous occlusion (RVO) is a common disorder among population 65 years or older, recurrent RVO is less frequently encountered. Recurrent central RVO due to factor V mutation has been reported in the literature.[1,2] Here we report a case of recurrent branch retinal vein occlusion in a patient with mutation of clotting Factor V gene.

Case report

A 60-year-old white male was urgently referred from the optician with complaint of sudden impairment of vision in the left eye. The presenting visual acuity was 6/5 in the right eye and 6/18 in the left eye. Ocular examination revealed an infero-temporal branch retinal vein occlusion with macular oedema in the left eye. Assessment of risk factors revealed him to be a hypertensive on regular antihypertensive medication. As a part of initial work-up for venous occlusion, we asked for full blood count, glucose estimation, and lipid profile evaluation—all of which came as normal. He was advised regular blood pressure check-up and we started him on Aspirin 75 mg, once daily. Fundus fluorescein angiography carried out 4 months later showed small areas of capillary dropout along the blocked vessel with resolving macular oedema. No neovascularisation was noted on the disc or elsewhere. Subsequent follow-up showed spontaneous resolution of macular oedema, well-developed collateral circulation, and visual acuity in the left eye improved to 6/5. He was discharged from specialist care. The patient was referred back to us about 18 months later with similar complaints in the left eye. The presenting visual acuity was 6/5 in the right eye and 6/12 in the left eye. This time he was found to have supero-temporal branch vein occlusion in the left eye with macular oedema. His blood pressure was well under control with felodopine and he was taking aspirin regularly. We were unable to elicit any evidence of thromboembolic disease on past history or systemic examination. We ordered repeat full blood count, glucose, and lipid estimation as well as tests for anticardiolipin antibody, ANCA, Clotting profile, plasma homocysteine level and plasma electrophoresis. All the above investigations came as normal except slightly prolonged activated partial thromboplastin time (APTT). A carotid doppler scan did not reveal any stenosis on either side. He was referred to the haematologist and a thrombophilic screen was performed. The result showed modified activated protein C (APC) resistance consistent with Factor V Leiden mutation. Considering the increased risk of future thrombo-embolic events, he was started on warfarin prophylaxis. The patient is asymptomatic presently with vision of 6/5 in both eyes. Repeat fluorescein angiogram showed scattered areas of capillary dropout, well-developed collaterals, no macular oedema and no neovascularisation. There have been no further thrombo-embolic episodes during the last 18 months of follow-up.

Comments

Retinal venous occlusion (RVO) is the second most common cause of reduced vision due to retinal disease with branch vein occlusion being about 2–3 times more common than central venous occlusion. More than half of the patients of RVO are over 65 years. Numerous acquired and hereditary risk factors have been identified for venous thrombosis. The main acquired risk factor for RVO is hypertension and it is more commonly associated with branch vein occlusion. Younger patients with central RVO need to be investigated regarding use of oral