characterised by a normal epidermis overlying a dermis that contains randomly arranged mature striated muscle fibers associated with varying amounts of mesenchymal elements such as adipose tissue, collagen and blood vessels. Herein we report a mesenchymal hamartoma with rhabdomyomatous features occurring in the orbit.

Case report

A 2-year-old boy was referred to the clinic with a history of right unilateral axial proptosis presenting on the fourth day of life with no useful vision ipsilaterally. Two orbital biopsies had been performed, the first being unsuccessful, and the second showing haphazardly arranged skeletal muscle fibers and fibrous tissue. On examination, he had no light perception OD, ptosis, fixed extraocular movements, and a pale atrophic optic disc. Ultrasound features included a solid, well-outlined highly reflective mass that was indenting the globe. Magnetic resonance imaging with gadolinium of the brain and orbit, at 5 months, revealed a diffuse infiltrative retrobulbar intraconal lesion. A repeat scan 18 months later, revealed an increase in the size of the lesion and the orbit (fig 1). The initial differential diagnoses included mesenchymal hamartoma, benign soft-tissue neoplasm, and granular cell tumour.

Due to the loss of vision, relentless increase in the size of the lesion, orbit, and superior orbital fissure, the patient underwent an eyelid and conjunctival sparing exenteration with lateral orbitotomy and a dermis fat graft.[1] During surgery, the tumour felt firm and solid. The case was complicated by cerebrospinal fluid (CSF) leak one day postoperatively originating from the superior orbital fissure even though temporalis muscle with Tisseal® was used to patch the fissure intraoperatively. A lumbar drain inserted for 1 day resolved the CSF leak with no further complications. Histologic features are displayed in fig 2.

Two months later, the patient was fitted with an ocular prosthesis with good cosmetic outcome. He will undergo ptosis surgery in the future.

Comment

RMH was first described as striated muscle hamartoma in 1986 by Hendrick et al[2] Since then, 30 cases of RMH have been recognized and reported in the literature.[3–8] It is commonly seen in infants or young children and appears at birth. Clinically it presents as a firm, flesh-coloured, nontender, polypoid, solitary subcutaneous lesion, at or near the midline, without prominent change in size. Specific congenital anomaly syndromes associated with RMH include Goldenhar and Delleman syndromes.[3]

In our case the histology was similar to that described for RMH; however it occurred in the orbit rather than the usual dermal and subcutaneous region. To our knowledge, this is the first such tumour reported in a non-subcutaneous site.

The possibility of a benign Triton tumor was also considered but in these lesions the nerve and muscle tissue is intimately admixed as the muscle fibers are thought to have been entrapped within the nerve sheaths during development. As the main component of this tumour was skeletal muscle, with lesser contributions by peripheral nerve and adipose tissue, we are presenting this case as a unique occurrence of an orbital mesenchymal hamartoma with rhabdomyomatous features.

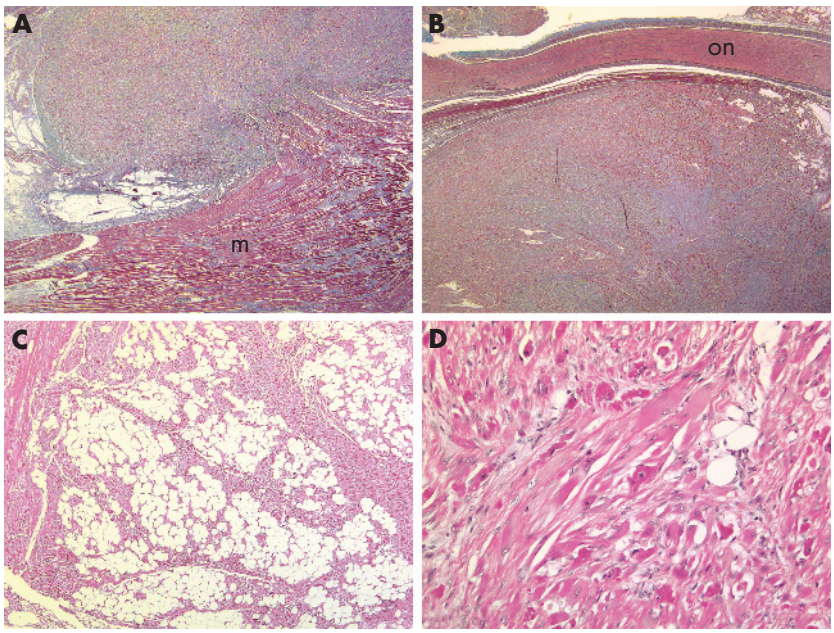

Figure 2 (A) The mass was composed predominantly of disorganized skeletal muscle which surrounded and infiltrated the extraocular muscles (m) (Trichrome stain, ×4), (B) compressed the optic nerve (on) (Trichrome stain, ×4) and, (C,D) infiltrated the adipose tissue (H&E stain, ×10, ×40). Adipose tissue and peripheral nerves were admixed with the muscle tissue in varying amounts throughout the lesion. We diagnosed this lesion as a hamartoma on the basis of presentation shortly after birth with slowly progressive growth associated with expansion of the orbit and its histologic resemblance to normal skeletal muscle. We used these features to differentiate it from a rhabdomyosarcoma which would have had rapid growth and be histologically composed of small, undifferentiated cells with little cytoplasm, a high mitotic rate and would lack an intimate admixture with adipose tissue and peripheral nerves.

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doi: 10.1136/bjo.2006.099747

Accepted 4 August 2006

Informed consent has been obtained from the patient for the publication of their details.

Competing interests: none.

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Ecstasy induced acute bilateral angle closure and transient myopia

A case of ecstasy (3,4-methylenedioxymethamphetamine (MDMA)) misuse in a previously emmetropic healthy man, who presented with acute bilateral angle closure and transient myopia after 2 weeks of consumption, is reported. Ultrasound biomicroscopy revealed bilateral ciliochoroidal effusions suggesting the mechanism of the adverse event. The episode resolved spontaneously. Ecstasy misuse needs to be considered as a possible cause in patients presenting with acute angle closure with choroidal effusion when no other known class of prescription drugs can be implicated.

MDMA, known as ''ecstasy,'' has become increasingly popular as a recreational drug over

the past several years.[1] There has been an associated increase in reports of presumed ecstasy-related deaths and severe adverse effect (UN World Drug Report, 2004) (http://www.unodc.org/unodc/world_drug_report_2004.html).

Case report

A 39-year-old man presented with painless progressive decrease of vision in both eyes over a period of 2 days to his optometrist. The intraocular pressure (IOP) was recorded as 32 mm Hg in both eyes and he was referred to an ophthalmologist. He was emmetropic previously. On examination, he had bilateral myopic refraction and IOP of 40–41 mm Hg in both eyes. Bilateral Neodymium-DOPED yttrium aluminium garnet peripheral iridotomies (PI) were performed for presumed bilateral acute angle closure. Despite patent PI, IOP remained high with closed angles. Topical treatment included brimonidine 0.2% and timolol maleate 0.5% twice a day. He was referred to a tertiary centre for further management.

There had been no similar episodes. There was no significant ocular or medical history. He was not on any prescribed systemic or ocular medication. Repeated enquiries about medication or drug use revealed a single episode of ecstasy misuse about 2 weeks before the onset of symptoms. At presentation his visual acuity was 6/30 in the right eye and 6/90 in the left eye, improving to 6/12 in both eyes with a refractive correction of −5.00 DS. The corneas were clear; pupils were sluggish but not dilated or fixed. Both anterior chambers were uniformly shallow (fig 1A). The iridotomies were patent. His IOP was recorded as 28 and 23 mm Hg in the right and left eyes, respectively. Gonioscopy revealed bilateral closed angles not opening with indentation (fig 1C). Oral acetazolamide 250 mg four times a day was initiated. Dilated fundus examination revealed macular folds in both eyes and there was no obvious evidence of retinal elevation. Examination with 78 D lens showed normal optic nerves. Ultrasound biomicroscopy (UBM) revealed 360° ciliochoroidal effusion, closed angles and ciliary body and scleral thickening (fig 2A). B-scans showed choroidal thickening and effusion in both eyes (fig 2B). A scan revealed axial length of 23 mm in both eyes.

Four days later, vision improved to 6/6 in the right eye (−2.00 DS) and 6/9 in the left eye (−3.00 DS) without further intervention. IOP was 12 mm Hg in both eyes. Medications were withdrawn subsequently. Anterior chamber was deep and gonioscopy revealed open angles up to scleral spur in both eyes after 10 days of initial onset of symptoms (fig 1B,D). Serial UBM showed gradual resolution of the supraciliary effusion. After 3 months, his vision was noted to be 6/6 without any refractive error. Rest of the ocular examination and UBM were normal.

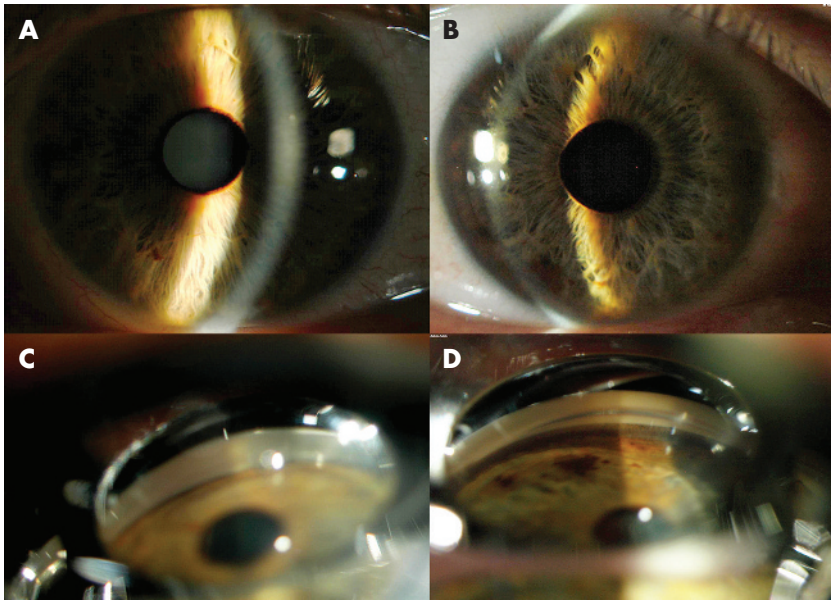

Figure 1 Slit-lamp images. (A, B) Day 4 and day 10. (C, D) Gonioscopy images showing closed angles (day 4) and open angles (day 10).

Comment

Acute onset bilateral angle closure and/or transient myopia has been reported as an after effect for many drugs, including selective serotonin uptake inhibitors, tricyclic antidepressants, sulfonamides, tetracycline and some diuretics.[2 3] The postulated mechanisms by which supraciliary effusions produce angle closure glaucoma and transient myopia have already been described.[4] Fluid movement in choroidal effusion could be related to drug-induced membrane potential changes or a possible idiosyncratic reaction. The acute myopia can probably be explained by the forward displacement of the lens caused by supraciliary effusion, although ciliary body swelling and lens thickening could also play a role. This angle closure resolves spontaneously and PI is not indicated.

MDMA has major effects on serotonin (5-hydroxytryptamine) pathways. As a synthetic amphetamine derivative, it increases the release of monoamine neurotransmitters (serotonin, noradrenaline and dopamine) and inhibits the reuptake of serotonin.[5] It influences pupillary diameter inducing mydriasis and depressing pupillary reaction to light.[6–8] The weak anticholinergic or mydriatic effects of serotonergic drugs are enough to precipitate an acute angle-closure episode by a mechanism similar to that of the tricyclic antidepressants.[7 9] Serotonergic innervation in the eye and the presence of serotonin and serotonin receptors in the aqueous humour and ciliary body have been reported.[8 10] Although the precise mechanism is unknown, the supraciliary effusions in this patient could be evidence of the serotonergic effects of ecstasy.

There has been only one report of ecstasy-induced acute bilateral angle closure in combination with marijuana. However, the mechanism of angle closure was not directly attributed to ecstasy alone nor was there any documentation of choroidal effusion.[9]

The late effect of the drug in our patient can be explained by the fact that ecstasy could have induced a gradual rise in post-synaptic levels of serotonin via desensitisation of the feedback systems that control the rate limiting enzyme in 5-hydroxytryptamine synthesis.[10] We could not confirm whether the patient had predisposing narrow angles as he had bilateral PI when presenting to us.

In conclusion, ecstasy misuse needs to be kept in mind as a differential diagnosis for drug-induced acute onset angle closure and transient myopia.

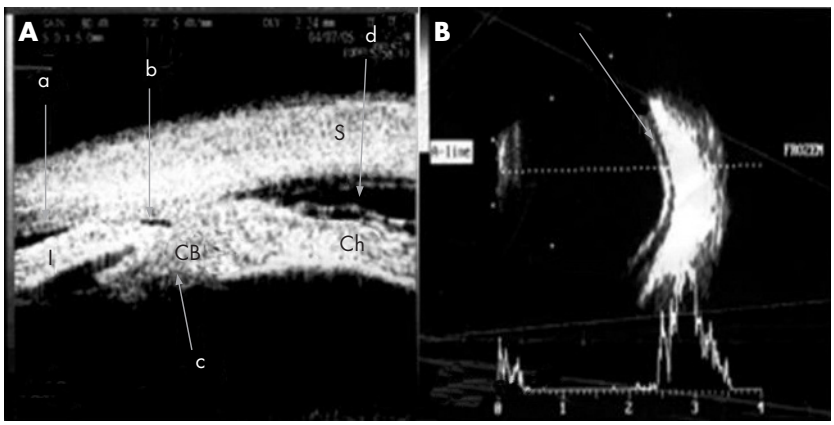

Figure 2 (A) Ultrasound biomicroscopy image showing (a) shallow anterior chamber , (b) closed angle, (c) ciliary body swelling, and (d) ciliochoroidal effusion. I, iris; CB, ciliary body; S, sclera; Ch, choroid. (B). B-scan image showing choroidal effusion.

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doi: 10.1136/bjo.2006.099986

Accepted 1 September 2006

Competing interests: None.

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Ophthalmomyiasis externa caused by *Dermatobia hominis* in Florida

Infestation of humans or other vertebrates by dipterous fly larvae is called myiasis. Larvae can infest several parts of the body, most commonly cutaneous tissue. Occasionally, infestation of ocular tissue could occur resulting in internal ocular or external (conjunctival or eyelid) ophthalmomyiasis.[1–5] The most common species causing external ophthalmomyiasis in the US is the sheep botfly, *Oestrus ovis*.[6] The human botfly, *D hominis*, is the primary cause of cutaneous myiasis in Central and South America, but only rarely causes external ophthalmomyiasis.[1 2 5]

Several case reports of external ophthalmomyiasis owing to *D hominis* occurring in the US exist in the literature; however, every case reports a history of recent travel to tropical American countries.[1] To our knowledge, there have been no previously identified cases of external ophthalmomyiasis secondary to *D hominis* originating in the US.

Case report

A 5-year-old girl presented with a 10-day history of pain and swelling of her left upper eyelid. Her symptoms began while at the beach in Fort Walton Beach, Florida, USA. She had recently been examined by an external ophthalmologist who reportedly saw a larva protrude from an aperture in the eyelid. Ocular examination revealed an excoriated, erythematous area within 2 mm of the left upper lid margin (fig 1). On closer examination, a tiny aperture producing a clear discharge was noted within the lesion. The patient was taken to the operating room for exploration. A single larva was identified and removed in total.

On gross examination, the larva measured 5.5 mm×1 mm (fig 1, inset). Microscopic examination revealed a larva with a broad rostral end exhibiting two rows of backward-directed, thorn-shaped spines (fig 2). Based on its size, shape and surface characteristics, the larva was identified as a first-stage larva of the fly *D hominis*.

Comment

D hominis is the most common cause of cutaneous myiasis. However, external ophthalmomyiasis encompasses <5% of all cutaneous sites.[1 5] The human botfly is not indigenous to North America. However, there are several dozen reports in the literature of *D hominis* ophthalmomyiasis occurring in the US among those who have travelled to Central and South America. A recent review by Denion and co-workers[2] describes eight of nine cases of external ophthalmomyiasis owing to *D hominis* originating in tropical American countries. One case presumably originated in New York[3]; however, diagnostic inaccuracy was suspected given the lack of defining features required to identify this species.

Reports of myiasis caused by *D hominis* are appearing more frequently because of increasing international travel.[1 2] Currently, a history of travel to or residency in a tropical American country is needed to raise clinical suspicion of dermatobiasis.[2] Pathological analysis and identification of the larva and therefore appropriate diagnosis cannot be achieved if the species is not first suspected. We are presently unaware of previous reports of external ophthalmomyiasis caused by *D hominis* found in the US that do not include a history of foreign travel. Thus, our case, originating in Fort Walton Beach, could imply migration of a species. Furthermore, Fort Walton is located at a latitude of 30.4 N, which is just north of what is considered tropical America (18 S to 25 N).[5] Consequently, Fort Walton's subtropical climate (www.britannica.com) could be compatible with life for *D hominis*, especially in light of changing global temperatures.[7] Therefore, it is plausible that if the species were brought into the area by travellers from endemic regions, the botfly could have been able to survive and adapt to this climate. Certainly, there needs to be more cases to support these theories. This case highlights the need to recognise this species as an aetiological agent causing external ophthalmomyiasis in cases originating in North America.

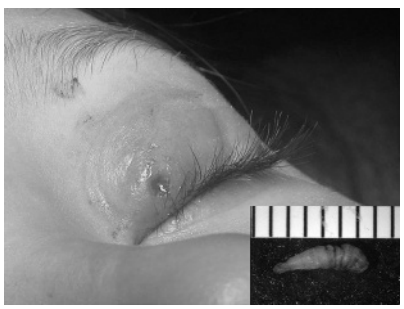

Figure 1 External ophthalmomyiasis. Note the tiny aperture producing clear discharge superior to the left upper lid margin. Inset: macroscopical view of human botfly larva, *Dermatobia hominis*. The rostral end of the larva is covered with several rows of thorn-like spines.

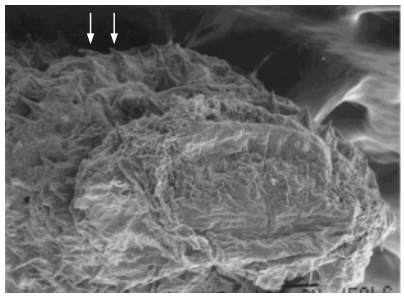

Figure 2 Scanning electron micrograph of *Dermatobia hominis* showing the numerous backward-directed spines (arrows).

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doi: 10.1136/bjo.2006.100776

Accepted 2 September 2006

Competing interests: None.

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