showed the extracted lipid to be an accumulation of triglyceride esters which mostly consisted of oleate and to a lesser degree of palmitate and stearate (2.5 mg versus 0.16 mg in non-affected control tissue).

COMMENT

This patient presented with a lipid-rich lesion similar to the so called paraffin granuloma which represents an inflammatory reaction to exogenous lipid.[7] Usually, exogenous lipid gets access into the tissue in the form of ointments used in or in close vicinity to the eye.[7 8] In contrast with paraffin, however, the triglyceride esters that were analysed in our patient are not usually present in ophthalmic ointments. Triglycerides are rather a constituent of naturally occurring lipids and can be expected—for example, in fat necrosis after trauma. Our patient did not exhibit any features of a pre-existing lipomatous lesion such as, for example, a lipodermoid; moreover, there was no history of trauma or mechanical irritation. Thus, the most likely explanation for the presence of a lipogranuloma remains a "complication" from the treatment of her dacryocystitis 30 years ago. Various lipid based substances have been used for rinsing of, and instillation into, the canaliculus or lacrimal sac. These ointments, however, are usually also based on paraffin or Vaseline.[5–9] Other lipid based materials have been employed as contrast material for viewing the lacrimal passage. One of the substances that has been commonly used for contrast dacryocystography is Lipiodol[10], an iodised poppy seed oil which is a characteristic mixture of glyceric esters of various fatty acids including mainly oleic, linoleic, linolenic, palmitic, and stearic acid (information from Byk Gulden, Konstanz). Thus, the lipid composition of Lipiodol corresponds remarkably well to the mixture that was analysed in our specimen. The iodine present in the original substance can be expected to have been removed and transferred to the thyroid, and, with endogenous fat and ointments exhibiting somewhat different components, there is convincing evidence that Lipiodol can indeed be regarded as the initiating agent.

Similar problems after instillation of other lipid based substances into the lacrimal drainage system[5–9] and one case of a granulomatous inflammation initiated by the application of a lipid based contrast medium to the orbit[3] have been reported but the time between the original "insult" and the development of an inflammatory reaction was always much shorter. This suggests that, in our case, a minor injury to the canaliculus or lacrimal sac might have occurred, allowing only a very small amount of lipid based material to reach the surrounding tissues and cause a self propagating inflammatory process. As triglycerides are much more similar to human body fat than paraffin, one could speculate that this might further help to explain the unusually long time lapse between the primary application and the clinically relevant granulomatous reaction seen in our patient.

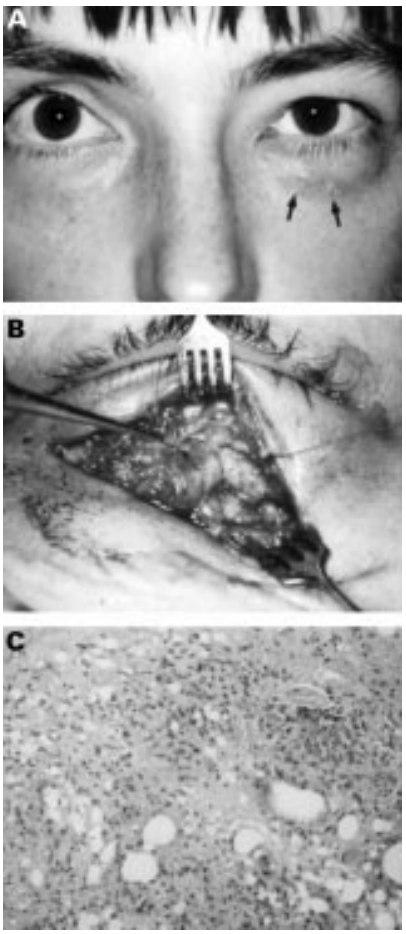

Figure 1 (A) Clinical appearance at first presentation, showing a red subcutaneous swelling (indicated by arrows) in the left medial canthus extending into the lower lid. The left upper eyelid also appears somewhat oedematous. (B) Intraoperative appearance of the lesion, revealing yellowish lipid-like tissue of firm consistency. Note that the tumour is infiltrating the surrounding tissues without evidence of a capsule or pseudocapsule. (C) Histology shows connective tissue with numerous lipid vacuoles of different sizes that are mostly surrounded by multinucleate giant cells. Note also the dense chronic inflammatory cell infiltrate. No genuine orbital fat is seen in this section. (Paraffin section, haematoxylin and eosin, ×170).

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1 Boynton JR, Searl SS, Heimer JL, *et al.* Eyelid oleogranulomas caused by petroleum jelly injection. *Arch Ophthalmol* 1988;**106**:550–1.
2 Cornblath WT, Dotan SA, Trobe JD, *et al.* Varied clinical spectrum of necrobiotic xanthogranuloma. *Ophthalmology* 1992;**99**:103–7.
3 Eifrig DE. Lipid granuloma of the orbit. *Arch Ophthalmol* 1968;**79**:163–5.
4 Hanssens M, Poelman J. Sclerosing lipogranuloma of the orbit. *Bull Soc Belge Ophtalmol* 1988; **227**:35–46.
5 Crehange JR, Arouette J, Mouly R, *et al.* Vaselinomes palpebraux apres injections medicamenteuses dans les voies lacrymales. *Bull Soc Ophtalmol Fr* 1970;**70**:1202–6.
6 Newcomer VD, Graham JH, Schaffert RR, *et al.* Sclerosing lipogranuloma resulting from exogenous lipids. *Arch Dermatol* 1956;**73**:361–72.
7 Witschel H, Geiger K. Paraffin induced sclerosing lipogranuloma of eyelids and anterior orbit following andonasal sinus surgery. *Br J Ophthalmol* 1994;**78**:61–5.
8 Moolchandani J, Kazim M, Farber M, *et al.* Eyelid lipoid granuloma following topical ointment application. *Ophthalmic Plast Reconstr Surg* 1990;**6**:133–5.
9 Struck HG, Tost F. Die "Tränenwegsplombe"—Indikation und Risiken. *Aktuelle Augenheilkd* 1992;**17**:153–6.
10 Kassel EE, Schatz CJ. Lacrimal apparatus. In: Som PM, Curtis HA, eds. *Head and neck imaging*. 3rd ed. St Louis: Mosby, 1995:1129–83.

Treatment of subhyaloid haemorrhage with intravitreal tissue plasminogen activator and C_3F_8 gas injection

EDITOR,— Subhyaloid haemorrhage can be caused by a variety of retinal disorders, such as age related macular degeneration, proliferative diabetic retinopathy, Valsalva retinopathy, macroaneurysm, and trauma. Nd:YAG laser membranotomy has been used for the rapid clearing of premacular haemorrhage, but complications such as retinal or choroidal haemorrhage and retinal hole formation were reported with the use of Nd:YAG laser. If the patient has cataract or media opacity, effective and precise laser delivery would be difficult.[1]

Hassan *et al* reported that intravitreous tissue plasminogen activator (tPA) and C_3F_8 injection effectively displaced the subretinal haemorrhage.[2] Furthermore, it has been recently reported that intravitreal tPA and SF_6 promote the clearing of premacular subhyaloid haemorrhages in shaken and battered baby syndrome.[3]

We treated a patient with subhyaloid haemorrhage by intravitreal tPA and C_3F_8 injection without any complications. YAG laser membranotomy failed because the patient's pterygium and cataract hindered proper contact lens application and caused laser beam scattering.

CASE REPORT

A 75 year old female patient visited our clinic because of sudden visual loss in her right eye 45 days earlier. Visual acuity was counting fingers at 20 cm in the right eye and 20/100 in the left. There were pterygia in both eyes, and her lenses showed cortical opacity. On fundus examination, a round dark red haemorrhage with a convex surface covering the right macula was noted (Fig 1). There was a fluid level in the upper part of haemorrhage and its preretinal location was confirmed by fluorescein angiography. There was neither posterior vitreous detachment nor a hole in the posterior hyaloid. Indocyanine angiography showed an arterial macroaneurysm in the superotemporal vascular arcade in the right eye.

Since the subhyaloid haemorrhage was thick, we first tried Nd:YAG laser membranotomy. However, this failed because her pterygium hindered proper contact lens application, and YAG laser was not able to be precisely focused on the anterior surface of the

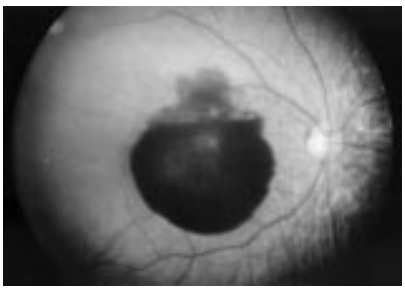

Figure 1 Fundus photograph of the right eye shows a dark subhyaloid haemorrhage centred at the fovea. The fluid level in the upper part of the haemorrhage suggests subhyaloid space location. Lens opacity makes the fundus hazily visualised.

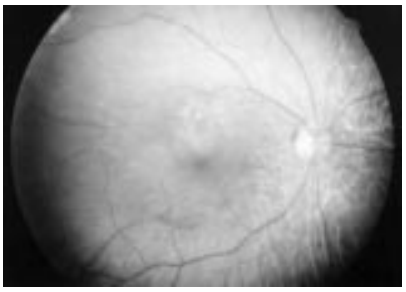

Figure 2 Fundus photograph of right eye 2 months after treatment. The subhyaloid haemorrhage has completely cleared.

subhyaloid membrane owing to the poorly applied contact lens and cataract. Therefore, we decided to perform intravitreal tPA and C_3F_8 injection under topical anaesthesia. Twenty minutes after injecting intravitreal 0.1 ml of 25 µg/0.1 ml tPA (total dose of 25 µg), 0.5 ml of 100% C_3F_8 was injected into the vitreous cavity. A paracentesis was done to decrease the intraocular pressure. The patient was told to maintain the face down position for 2 weeks. Three days after the injection, the subhyaloid haemorrhage was displaced by the gas bubble out of the macular region. The haemorrhage slowly decreased in size over 2 weeks, and then markedly decreased. After 2 months, the subhyaloid haemorrhage had completely cleared (Fig 2). Her vision in the right eye increased to 20/70 on her last visit.

COMMENT

Although the subhyaloid haemorrhage was somewhat old and very thick, it was rapidly displaced out of the macular region within 3 days. We suggest that tPA worked to lyse the blood clot. The vision of 20/70 may be attributed to cataract and retinal damage caused by the subhyaloid haemorrhage.

Tissue plasminogen activator and C_3F_8 injection seems to be an alternative way to clear the subhyaloid haemorrhage especially when the patient has media opacity or when there is a problem with contact lens application for laser.[4 5]

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1 Gabel VP, Bringruber R, Gunther-Koszuka H, *et al*. Nd:YAG laser photodisruption of hemorrhagic detachment of the internal limiting membrane. *Am J Ophthalmol* 1989;**107**:33–7.
2 Hassan AS, Johnson MW, Schneiderman TE, *et al*. Management of submacular hemorrhage with intravitreous tissue plasminogen activator injection and pneumatic displacement. *Ophthalmology* 1999;**106**:1900–6.
3 Conway MD, Peyman GA, Recasens M. Intravitreal tPA and SF_6 promote clearing of premacular subhyaloid hemorrhages in shaken and battered baby syndrome. *Ophthalmic Surg Lasers* 1999;**30**:435–41
4 Adel B, Israel A, Friedman Z. Dense subhyaloid hemorrhage or subinternal limiting membrane hemorrhage in the macula treated by Nd:YAG laser. *Arch Ophthalmol* 1998;**116**:1542–3.
5 Iijima H, Satoh S, Tsukahara S. Nd:YAG laser photodisruption for preretinal hemorrhage due to retinal macroaneurysm. *Retina* 1998;**18**:430–4.

Blunt trauma in Best's vitelliform macular dystrophy

EDITOR,—Recently, the association of Best's vitelliform macular dystrophy (BVMD) with mutations in chromosome 11 has been reported, and candidate genes that may be affected by these mutations have been identified.[1 2] However, the role of these genes in retinal and retinal pigment epithelium (RPE) function is not clear. In addition, factors that determine the progression of the vitelliform foveal lesion leading to impairment of visual acuity in patients with BVMD are not understood. We present a case in which blunt trauma was associated with deterioration of visual acuity and macular scar formation in a patient with BVMD.

CASE REPORT

A 14 year old male presented to our clinic after being hit in his right eye by a fist 40 days earlier. He complained of reduced visual acuity in this eye since the trauma. He had a history of good and equal visual acuity in both eyes until the trauma occurred, and ocular and systemic history were unremarkable. He was the fourth son among eight children (two males and six females), and the patient was unaware of any significant ocular diseases in his family. Other family members were not available for our examination.

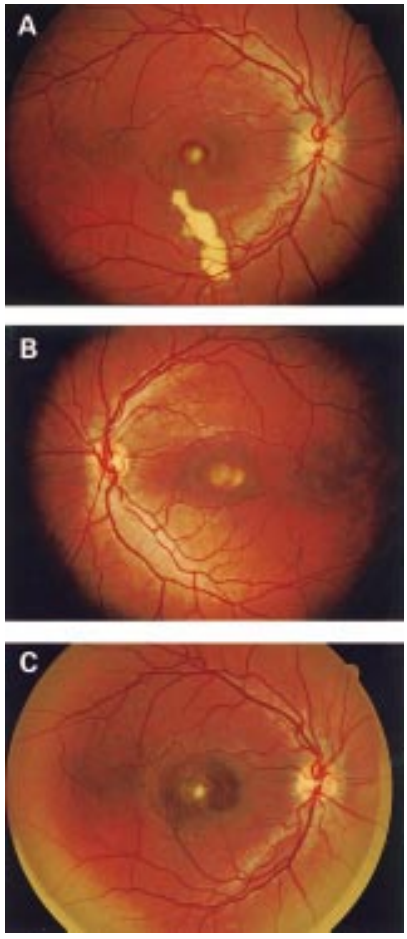

Figure 1 Colour fundus photographs of a patient with Best's vitelliform macular dystrophy. On initial examination (40 days after blunt trauma), the right eye had a foveal scar with remnants of perifoveal haemorrhage, and subretinal yellowish material (A). The left eye showed a typical vitelliform lesion (B). One month later, the remnants of haemorrhage and the yellowish subretinal material had been absorbed, and mild RPE changes were noted in this area in the right eye. The other findings remained unchanged (C).

Best corrected visual acuity was 6/24 and 6/7.5 in the right and left eye respectively. Anterior segments and intraocular pressures were normal. Funduscopy of the right eye revealed a foveal scar with remnants of subretinal haemorrhage around the fovea. Yellowish subretinal material was seen extending centrally from the lower temporal arcade. In this area, two parallel pigmented lines that may represent choroidal ruptures were noted (Fig 1A). The left eye showed a vitelliform foveal lesion (Fig 1B). The optic disc and peripheral retina were normal in both eyes.

ISCEV standard electro-oculography (EOG) and electroretinography (ERG) were performed. The EOG showed severely reduced light peak to dark trough ratios of 120% and 100% in the right and left eye, respectively (lower limit of normal 180%). The full field photopic cone ERG response, as well as the scotopic rod and mixed cone-rod responses, were normal. The patient could not discriminate the colours on the Farnsworth D-15 test in his right eye, while the left eye showed a deuteranopic defect.

On the basis of these findings, we diagnosed blunt trauma that resulted in subretinal haemorrhage and foveal scar formation in a patient with BVMD. On follow up examination 1 month later, the visual acuity remained unchanged. However, the haemorrhage surrounding the foveal scar as well as the yellowish subretinal material in the posterior pole of the right eye had absorbed, and mild RPE changes were noted (Fig 1C). Fluorescein angiography at this time showed staining of the foveal scar in the right eye with hypoflu-

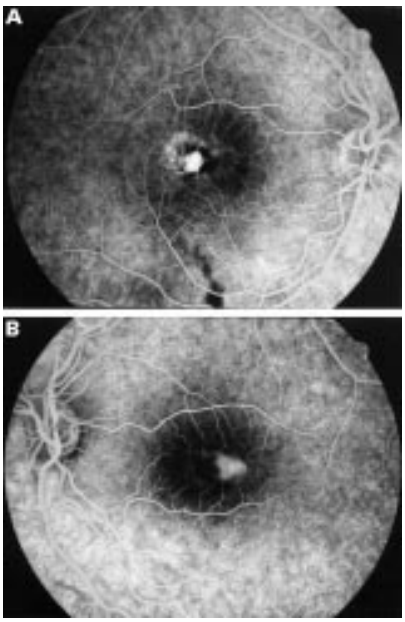

Figure 2 Fluorescein angiography in BVMD approximately 2½ months after trauma to the right eye, taken on the same day as Figure 1C. Late staining of the foveal scar with hypofluorescence in the area corresponding to the absorbed subretinal material is seen in the right eye (A). Late staining of the vitelliform foveal lesion is noted in the left eye (B).