week after the first injection patient started noticing improvement in her night vision. Serum vitamin A level rose to 0.85 μml/l. Her post-treatment (9 weeks after first vitamin A injection) electrodiagnostic tests showed marked improvement in retinal function. Flash ERG responses under both scotopic and photopic conditions had returned to normal (Figure 1 and Table 1). Our patient continues to remain symptom free with vitamin A injection supplements.

Comment

Vitamin A deficiency is well documented in patients suffering from chronic pancreatic insufficiency (alcoholic pancreatitis, cystic fibrosis) and in intestinal bypass surgery involving jejunum, ileum, and colon.[4–7] However, night blindness due to vitamin A deficiency in relation to pancreatic neoplasm is probably rare and to our knowledge there are only a couple of case reports in medical literature.[2,8]

Vitamin A (retinol) is ingested mainly as retinyl esters and a range of carotenoids, the most abundant of which β carotene is cleaved in the enterocyte to retinaldehyde and then reduced to retinol. Carotenoids are obtained mainly from green vegetables and vitamin A (retinol) from animal products. Vitamin A is mostly absorbed in the upper small intestine. Since hydrolysis is necessary to liberate retinol before it can be absorbed, pancreatic insufficiency may be associated with vitamin A deficiency.[9]

Vitamin A is necessary for the synthesis of visual pigments of rods and cones. In our case, scotopic ERG was affected to a greater extent than photopic ERG indicating more marked involvement of rods over cones. This differential susceptibility to vitamin A deficiency is well documented in previous reports.[5,6]

If night blindness occurs in a patient with a pancreatic neoplasm one should suspect vitamin A deficiency. Another possibility is CAR although there are no reports suggesting its association with pancreatic malignancy and also the electrophysiological findings in CAR are different from vitamin A deficiency-related night blindness.[10] The useful investigations in this setting are serum vitamin A level, antiretinal antibodies, and electrodiagnostic tests. Night blindness due to vitamin A deficiency responds very well to vitamin A supplements.

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S Appaswamy[1], D De Silva[1], A Tyagi[2], LE Grocott[2], PD Heath[2] and T Gillow[1]

[1]Department of Ophthalmology
North Staffordshire Royal Infirmary
Stoke On Trent
ST4 7LN, UK

[2]Department of Clinical Neurophysiology
North Staffordshire Royal Infirmary
Stoke On Trent, ST4 7LN, UK

Correspondence: S Appaswamy
Tel: +44 2476 224055
Fax: +44 2476 844119
E-mail: drappaswamy@doctors.net.uk

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Sir,

Conjunctival necrosis and bleb leakage secondary to an adherent conjunctival foreign body

A case of conjunctival necrosis and bleb leakage secondary to an adherent conjunctival foreign body is presented.

Case Report

A 72-year-old gentleman presented to ophthalmic causality complaining of irritation watering

photophobia, and fall of vision in his left eye for 7 days. He was known to have open-angle glaucoma and had undergone a trabeculectomy to both eyes 5 years ago. There was no history of trauma.

His best-corrected visual acuity was 6/5 in the right and 6/12 in the left eye. There was a cystic conjunctival bleb superiorly in the right eye. A foreign body was adherent to the conjunctival bleb in his left eye. The anterior chamber was shallow and Seidl's test was positive (Figure 1). The intraocular pressure was 2 mmHg. Removal of the foreign body with refashioning of the conjunctival flap was performed under local anaesthesia. The adherent foreign body along with the necrotic tissue was sent for histopathology. Histology showed a seed husk with an underlying granulation tissue. On the first postoperative day, his visual acuity was 6/9 in the left eye, a deep anterior chamber, and an intraocular pressure of 12 mmHg.

The patient was seen in the clinic 4 weeks following surgery when his visual acuity was 6/6-2 in the left eye with a well-formed conjunctival bleb. His anterior chamber remained deep with an intraocular pressure of 18 mmHg (Figure 2).

Discussion

Trabeculectomy is the surgical procedure of choice in patients with uncontrolled glaucoma. It is however known to be associated with a number of early and late complications. The most frequently encountered early complications are hyphaema, shallow anterior chamber, hypotony, wound leak, and choroidal detachment. Late complications include bleb leak, cataract, visual loss, and an encapsulated bleb.[1]

Spontaneous bleb leak may be due to late-onset transconjunctival oozing or a point leak of aqueous humour.[2] The risk of late-onset focal bleb leakage increases following trabeculectomy with Mitomycin C

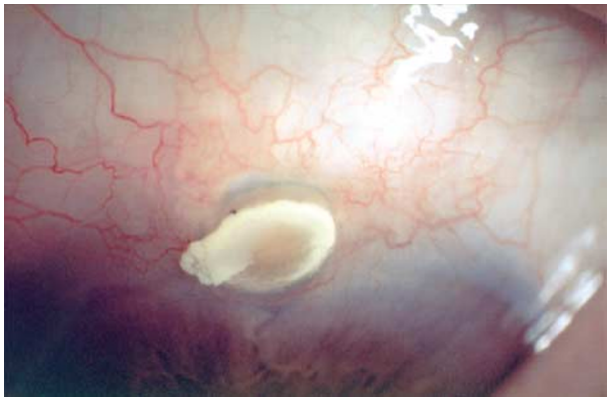

Figure 1 Photograph showing an embedded foreign body.

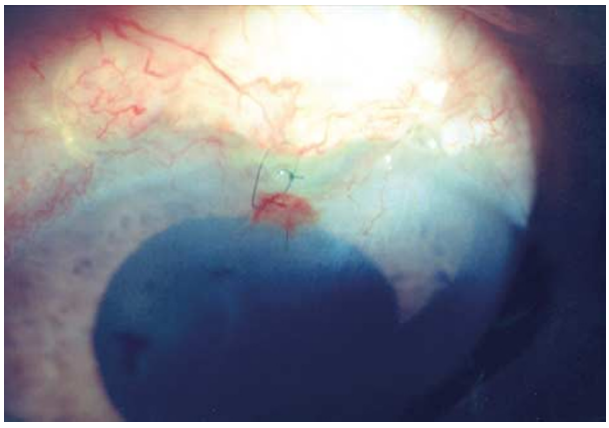

Figure 2 A deep anterior chamber following removal of foreign body and repair.

therapy.[3] Bleb leaks may in turn be associated with chronic ocular hypotony, decreased visual acuity, infection, hypotony maculopathy, corneal oedema with folds, choroidal effusion, and a persistently shallow anterior chamber, which may require intervention.[4]

Bleb leaks can be managed conservatively or surgically. Nonincisional conservative management is by aqueous suppression with lubrication or patching, bandage contact lenses, cyanoacrylate glue, or autologus blood injection. Surgical techniques are conjunctival suturing, resuturing of trabeculectomy flap, bleb revision with autologous or donor scleral grafting, bleb excision, conjunctival advancement, lyodura, and tenons patching.[5] Surgical bleb revision has a high success rate with regard to maintaining a functioning bleb and to preserving vision. When compared with nonincisional treatment, patients with late bleb leaks managed with conjunctival advancement are more likely to have successful outcomes.[6]

The case in discussion developed a bleb leak secondary to retained foreign body, adherent to the conjunctiva. Formation of foreign body granuloma and associated release of cytokines[7] lead to tissue necrosis and consequent bleb leak. This presentation of a retained conjunctival foreign body with conjunctival necrosis and bleb leak is very unusual and to the best of our knowledge has not been reported before.

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P Puri, R Saxena and ME Nelson

Department of Ophthalmology
Royal Hallamshire Hospital
Glossop Road
Sheffield S10 5 RL, UK

Correspondence: P Puri
Tel: +44 114 2630197
Fax: +44 114 2713747
E-mail: pankajpuri35@hotmail.com

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Sir,

Retinal astrocytic hamartoma with exudation

Case report

An asymptomatic 24-year-old lady was referred in 1997 following a routine optician examination with an amelanotic posterior pole lesion in her right eye. Her past medical history and family history were not contributory. Her visual acuity was 6/6 in both eyes. She was diagnosed as having retinocytoma. She was under periodic observation until October 2001 when she was referred to the Ocular Oncology Clinic for a second opinion.

Examination of the right eye showed a circumscribed yellow white retinal lesion along the superotemporal arcade (Figure 1). The lesion had fine intrinsic vessels and the feeder vessels were of normal caliber in contrast to retinoblastoma. Retinal exudation surrounding the lesion was also present. Fluorescein angiography showed intrinsic vasculature in the arteriovenous phase (Figure 2a) with late diffuse hyperfluoresence (Figure 2b). On indocyanine angiography (Figure 2c), the intrinsic vessels appeared to be of retinal origin. Blocking of choroidal fluorescence due to a deep retinal component of the lesion was also observed (Figure 2c, arrowheads). On B-scan ultrasonography, high intrinsic reflectivity indicated calcification (Figure 2d).

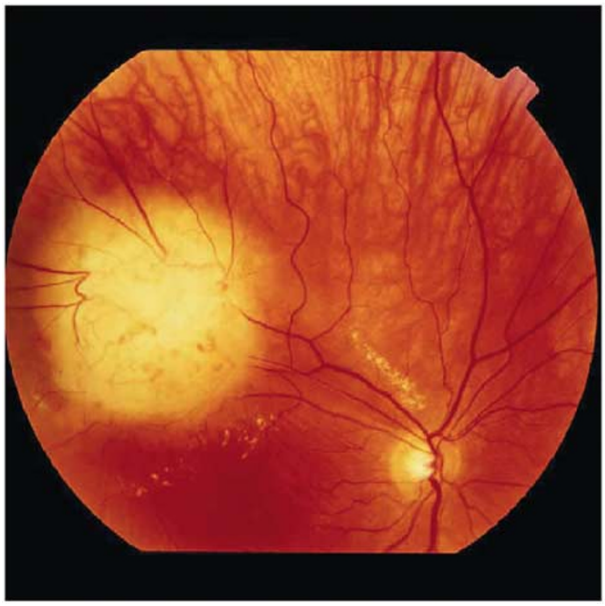

Figure 1 Fundus photograph of the right eye showing a circumscribed yellow white retinal lesion. Note retinal exudation.

Comment

The differential diagnosis of a calcified retinal mass must include retinoblastoma, retinocytoma, and astrocytic hamartoma. Remote possibilities such as combined hamartoma of the retina and retinal pigment epithelium, retinal pigment epithelial adenoma, and retinal capillary hemangioma were also considered.

A final diagnosis of a sporadic astrocytic hamartoma with exudation was made as she had none of the clinical findings associated with tuberous sclerosis complex.

Approximately 50% of patients with tuberous sclerosis complex have retinal astrocytic hamartoma with bilateral involvement in 25%.[1] They are located superficially in the retina predominantly near the optic disc. The retinal astrocytic hamartoma are generally stable with slow growth over several years or new calcification in some cases.[2,3] It is unusual for new lesions to develop in a previously normal-appearing retina[3] or for astrocytic hamartoma to demonstrate spontaneous regression.[4] Rare cases of aggressive astrocytic hamartoma leading to retinal exudation and even exudative retinal detachment have been reported.[5]

In summary, this patient developed a sporadic retinal astrocytic hamartoma with retinal exudation, in the