eye. Orbital MRI confirmed a subconjunctival mass extending from the superior limbus to the intraconal area in such a way that it encircled the optic nerve (Fig. 1). Biopsy and immunohistochemical staining confirmed mucosa-associated lymphoid tissue (MALT) lymphoma. We diagnosed the case as a primary ocular adnexal lymphoma (OAL) on the grounds that systemic investigations were negative.

According to the REAL (Revised European-American Classification of Lymphoid Neoplasms) classification, MALT lymphomas are extranodal marginal zone B cell lymphomas.[1] They have an indolent course and are usually localized to the original site, as in our case. These lymphomas can be differentiated with immunohistochemical staining.[2] Our case was CD-20 positive and CD-5 and CD-10 negative, which are the key characteristics of MALT lymphomas.

Chronic antigenic stimulation can lead to the formation of MALT lymphoma. Gastric MALT lymphoma is related to *Helicobacter pylori* colonization.[3] Some environmental factors, such as air pollution and plant dusts, might have played a role in our patient's condition. OAL may sometimes be misdiagnosed as chronic conjunctivitis, blepharitis, or strabismus.[4] A thorough eyelid and conjunctival examination may preclude misdiagnosis.[2] Akpek et al.[5] reported MALT lymphomas in chronic inflammatory ocular diseases.

Our case had been diagnosed as glaucoma because of high intraocular pressure, glaucomatous optic atrophy, and consistent visual field loss. The misdiagnosis might have arisen from inattention to any other causes. Our case is unique in terms of the development of rapid optic cupping and a retrobulbar mass, which had been overlooked for a long time. Although orbital lymphomas are not rare, an initial manifestation with glaucoma is an unusual property of lymphomatous lesions. In an extensive review of 5 case presentations by Coupland et al.,[1] no case was reported to have presented initially with glaucoma.

Advanced optic cupping in our case could be the result of either the mass effect of lymphoma on the optic nerve or high intraocular pressure. However, we were not able to determine which of these factors was responsible for the optic cupping. The complex presentation of OALs can make the diagnosis difficult in some challenging cases, as it was in our case. This possibility should be kept in mind in order to reach the correct solution in ambiguous situations.

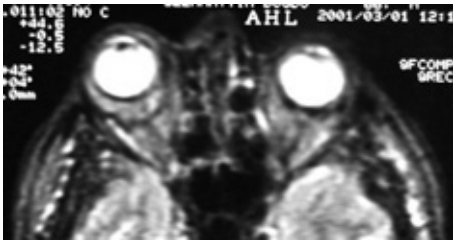

Fig. 1—On MRI the right eye is exophthalmic, and the globe wall is thickened diffusely (posterior wall 10 mm). There is no rectus muscle or optic nerve involvement.

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Basal laminar drusen and soft drusen have similar glycan composition

This case report describes the glycan composition of (small) basal laminar drusen (BLD) and (large) soft drusen using lectin histochemistry. Lectins have a high affinity for specific oligosaccharides. Because the lectin specificities for saccharides are known, the saccharide composition of both types of drusen can be inferred from their lectin-binding patterns.

Ocular tissue was obtained from a patient who died from end-stage renal failure. Prior to her demise she had undergone fluorescein angiography, which confirmed the presence of numerous small uniform drusen that fluoresced brightly in the arterial phase of the angiogram. In addition, there were larger, typical drusen, which fluoresced in the later phase (Fig. 1A). Histologic examination of the retina was undertaken post mortem on appropriate sections stained with periodic acid-Schiff. Drusen less than 75 μm were considered BLD. Other features characteristic of

BLD are uniform size and clustered distribution. Larger, more amorphous drusen were considered to be soft drusen.

Detailed oligosaccharide analysis of the drusen was undertaken using a panel of 20 biotinylated lectins (Table 1). Lectin binding was revealed with an avidin peroxidase/diaminobenzidine-cobalt revealing system, as described previously.[1] Histologic examination revealed that both the smaller (<75 μm), clustered, uniform BLD-type drusen and the larger (>75 μm), soft drusen had identical lectin-binding patterns (Fig. 1B).

The term BLD is used to describe numerous, small, round, discrete drusen that are slightly raised and tightly packed, and that stain early in the fluorescein run, giving a "starry sky appearance." They are more obvious on fluorescein angiography than on fundoscopy. Originally believed to be associated with a better visual prognosis and less frequent development of choroidal neovascularization than soft drusen, BLD were described as diffuse thickening of the basement membrane of the retinal pigment epithelium (RPE) with internal nodularity; this is distinct from the soft drusen characteristic of age-related macular degeneration (AMD), where there is focal RPE detachment. Gass et al.[2] suggested that the early choroidal fluorescence of BLD may be caused by nodular transparent hyaline thickening of the basement membrane, as opposed to the cloudy amorphous deposits that delay fluorescence in soft drusen. In contrast, Russell et al.[3] showed that BLD did not differ from soft drusen in terms of location, structure, and composition; BLD contained the same proteins and carbohydrates as drusen in AMD. However, the only lectins used in their study were from *Arachis hypogaea* with and without pretreatment with neuraminidase and *Ricinus communis* agglutinin 1. We used a comprehensive panel of lectins that would cover all the common saccharide sequences present in mammals, and showed that drusen-like lesions of varying sizes had identical lectin-binding patterns, indicating a similar, if not identical, saccharide composition. We postulate that the difference in clinical and fluorescein appearance between BLD and soft drusen is probably due to differing lipid content; hydrophilic BLD-type drusen are composed of phospholipids,[4] whereas hydrophobic drusen that fluoresce late may contain higher proportions of neutral lipids.

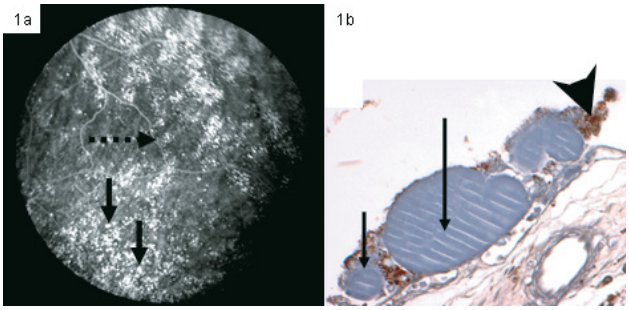

Fig. 1—(A) Fluorescein angiogram showing both small, tightly packed basal laminar drusen (dashed arrow), as well as larger, more typical soft drusen (solid arrows). (B) The lectin MPA (*Maclura pomifera* agglutinin) binds drusen (arrows) of varying sizes with equal intensity (bluish grey staining in contrast to brown retinal pigment epithelium). Arrowhead indicates retinal pigment epithelium (original magnification × 600).

Table 1—Intensity of lectin binding to drusen

Lectin	Source	Major specificity	Intensity of binding
Mannose			
Con-A	*Canavalia ensiformis*	αD-man,α-D-Glc, terminal or α1,2 N-linked sequences, high mannose, intermediate and small complex structures	2
PSA	*Pisum sativum*	α-D-man in nonbisected bi/tri antennary complex N glycan	2
LCA	*Lens culinaris*	α-D-man, similar but not identical to PSA	2
e-PHA	*Phaseolus vulgaris* (erythroagglutin)	Bisected bi/tri-antennate complex N glycan	2
l-PHA	*Phaseolus vulgaris* (leukoagglutinin)	Tri/tetra-antennate nonbisected complex N glycan	0
N-acetyl galactosamine			
SBA	*Glycine max*	Terminal GalNAcα1-	0
DBA	*Dolichos biflorus*	GalNAcα1,3(Fuc1,2)Galβ1,4GlcNAc Galactose	0
AHA	*Arachis hypogaea*	Galβ1,3GalNAcα->Galβ1,4GlcNAcα1	0
Jacalin	*Artocarpus integrifolia (Jacalin)*	Galβ1,3GalNAc-, Galα1,6-	0
MPA	*Maclura pomifera*	Galβ1,3GalNAcα1->GalNAcα1-	2
BSA-1B_4	*Bandeiraea simplicifolia*	Galα1,3Galβ1,4GlcNAcβ-	0
ECA	*Erythrina cristagalli*	Galβ1,4GlcNAc-	0
CTA	*Erythrina corrallodendron*	Galβ1,4GlcNAc-(multiple)	0
N-acetyl glucosamine oligomers			
WGA	*Triticum vulgaris*	$(-4GlcNAc\ \beta1,4GlcNAc\beta1-)_n$,$-(-Gal\beta1,4GlcNAc\beta1-)_n$	3
STA	*Solanum tuberosum*	β1,4GlcNAc oligomers	1
DSA	*Datura stramonium*	GlcNAcβ1,4GlcNAc-	3
Fucose			
UEA-1	*Ulex europaeus*	Fucα1,2Galβ1,4GlcNAc-	0
LTA	*Tetragonolobus purpureas*	α-L-fucosyl terminals	0
Sialic acid			
SNA	*Sambucus nigra*	Neu5Acα2,6Gal/GalNAc-	0
MAA	*Maackia amurensis*	Neu5Acα2,3Galβ1	0

Note: 0 = no binding; 1 = weak binding; 2 = moderate binding; 3 = moderately strong binding; and 4 = intense binding.

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Exudative retinal detachment following photodynamic therapy for retinal capillary hemangioma

Many therapeutic modalities have been applied to retinal capillary hemangiomas over the past decades. In 2002, the use of photodynamic therapy (PDT) with verteporfin began to emerge.[1] We describe a case of marked exudative response to PDT treatment.

A 16-year-old male was referred by his optician for an abnormality in his right fundus. He had a best-corrected logMAR acuity of 0.12 OD and 0.00 OS. The right fundus showed extramacular hard exudates, shallow subretinal macular fluid, and a pair of dilated retinal vessels at the posterior pole leading to a 3.4 mm fleshy, pink-orange retinal lesion in the superotemporal quadrant (Fig. 1). A diagnosis of solitary retinal capillary hemangioma was made. PDT with verteporfin was carried out uneventfully according to standard Treatment of AMD with Photodynamic Therapy Study protocol, using a spot size of 4600 μm. The following day, the patient noticed severe visual reduction and when reassessed a week later, the logMAR acuity in the affected eye was 1.2. Funduscopy revealed increased macular edema and hard exudates; subretinal hemorrhage and intraretinal edema around the lesion; and exudative retinal detachment involving the temporal and inferior retina (Fig. 2). The subretinal fluid completely resolved after 5 weeks and acuity improved to 0.8. Vision remained stable 3 months later and macular findings became less marked (Fig. 3).

The use of PDT in peripheral retinal hemangiomas has been documented in various case reports.[1–3] Aaberg et al.[2] described exaggerated cicatricial reactions in 3 patients and Szabó et al.[3] encountered a transient increase in subretinal fluid under the macula in a single case. In all cases, reduction of tumour activity was successful.

There are 2 theories that may explain the increased exudation from retinal hemangioma following PDT that was seen in our case. First, it could be increased leakage from the vascular lesion, a response known to occur with laser photocoagulation and cryotherapy. Alternatively, PDT can affect the choroidal vasculature, causing ischemia and even shutdown of the choriocapillaris or retinal capillaries with breakdown of the vascular barrier and significant subretinal fluid exudation, a response seen in PDT treatment of choroidal neovascularization.[4] Fluorescein angiography and (or) indocyanine green angiography would have been helpful in determining the cause of this complication but was not carried out. It is uncertain as to whether the size and location of the hemangiomatous lesion or the degree of pre-existing exudation have a bearing on the response to treatment with PDT alone.

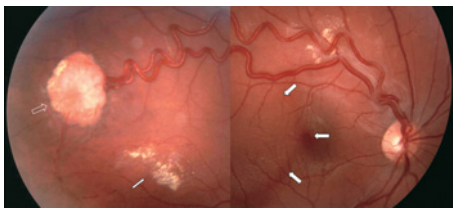

Fig. 1—Composite image of right fundus showing lesion (hollow arrow), exudation (thin white arrow), and nasal extent of subretinal fluid just involving macula (3 thick white arrows).

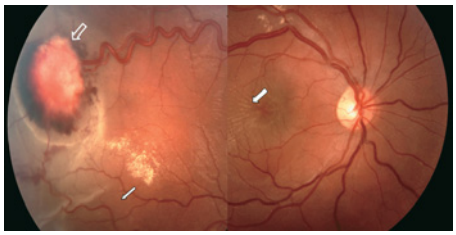

Fig. 2—Composite image of right fundus 1 week after photodynamic therapy showing increased subretinal fluid at the macula (thick white arrow), subretinal hemorrhage and intraretinal edema around the lesion (hollow arrow), and exudative retinal detachment involving the temporal and inferior retina (thin white arrow).