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The subject matter was previously presented at the meeting of American Society of Cataract and Refractive Surgery, 2008.

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Sir,

Virtual assessment and glaucoma shared care

We read with interest the report by Bourne *et al*.[1] Our attention was particularly drawn to the use of a virtual glaucoma assessment to support the community optometrists and provide quality assurance.

Many glaucoma management decisions are based on clinical findings, optic discs, visual fields, corneal thicknesses, and intraocular pressures (IOP) that are straightforward. Expert opinion is required however for a significant minority of cases. Unfortunately, deciding which patients require this additional input may be challenging. We also use a virtual clinic assessment to augment the triage of new patients who have been seen by 'in-house' ophthalmic nurse practitioners.[2] The decision-making remains the responsibility of the consultant, who reviews the clinical data, in the absence of the patient and oversees initial management. Our feasibility study has confirmed virtual assessment to be a reliable method of obtaining an accurate working diagnosis showing good agreement with experienced ophthalmologists.

In common with Bourne *et al*[1] we found issues with disc image interpretation in some patients. For us this was largely due to disc size (for both small and large discs), extensive peripapillary atrophy, and abnormalities of shape (tilting). Such disc images may be difficult to interpret in isolation, but this need not mean they cannot be reviewed on a virtual basis through serial images over time. We too found subtle variations in IOP around 21 mm Hg between practitioners that would tend to suggest the allocation of patients to different parts of the glaucoma care pathway. However, we would argue that this variation reflects not the accuracy of the optometrists, but rather the need to base IOP-governed decisions on a series of values. Given the demands of the recent NICE guidelines for glaucoma and ocular hypertension,[3] schemes for referral refinement and review of patients deemed stable, which utilise resources in the professions allied to medicine, are timely and appropriate. The use of a virtual clinic environment to allow expert advice and quality assurance also has much to recommend it.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Immunohistochemical analysis of internal limiting membrane by confocal microscopy in a case of stage 4 idiopathic macular hole

There are only rare reports showing the presence of α-smooth muscle actin (α-SMA)staining-positive cells in internal limiting membrane (ILM) specimens removed during idiopathic macular hole surgery, and in these studies, specimens have been evaluated by means of light or scanning electron microscopy.[1–3] To the best of the authors' knowledge, identification and localisation of α-SMA microfilaments by confocal microscopy in the ILM surrounding the borders of a macular hole have not been previously described.

Case report

A 68-year-old woman underwent vitrectomy for stage 4 idiopathic macular hole. Preoperative evaluation of the macular hole biomicroscopically and imaging with optical coherence tomography showed the absence of an epiretinal membrane (Figure 1b). Triamcinolone–acetonide was used for visualising the posterior cortical

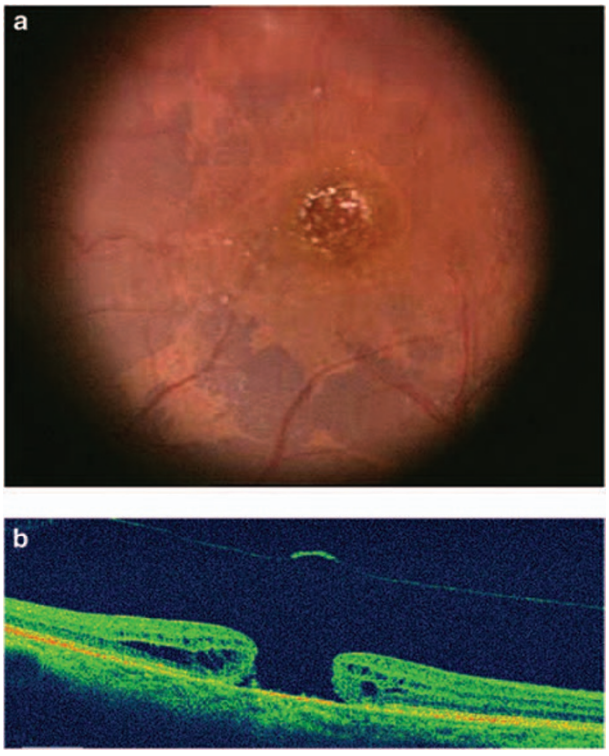

Figure 1 Imaging of the macular area. (a) Intraoperative visualisation of the macular hole and the internal limiting membrane (ILM) after staining with Brilliant blue dye. Note that the area around the edge of the macular hole is not stained blue, suggesting the presence of cells covering the ILM even in the absence of a formed epiretinal membrane (ERM). (b) Macular optical coherence tomography shows a full-thickness macular hole and confirms the absence of any ERM.

vitreous. After vitrectomy, the ILM was stained with Brilliant blue (BB) dye, then peeled off using intraocular forceps. The part of the ILM around the edge of the macular hole was not stained (Figure 1a). Two separate ILM specimens were obtained during surgery; the first (specimen A) around the macular hole and the second (specimen B) 1-disc diameter away from the macular hole up to the temporal vascular arcades and the optic disc.

The ILM specimens were studied immunohistochemically using confocal microscopy after labelling with antibodies to α-SMA. ILM specimen A was stained positive for α-SMA (Figure 2a–c), whereas specimen B failed to stain positive for α-SMA (Figure 2d).

Comment

Vitreofoveal traction has been proposed as the main mechanism for early stage (1 and 2) idiopathic macular hole formation.[4] Cellular migration and proliferation with secondary contraction on the ILM might lead to a further progression of the macular hole even after posterior vitreous detachment has occurred (stages 3 and 4) and keep the macular hole open, with stage 4 macular holes having the largest area of cellular migration around the macular hole.[5] Our case clearly shows that cells on the ILM around stage 4 macular hole contain bundles of actin microfilaments and therefore

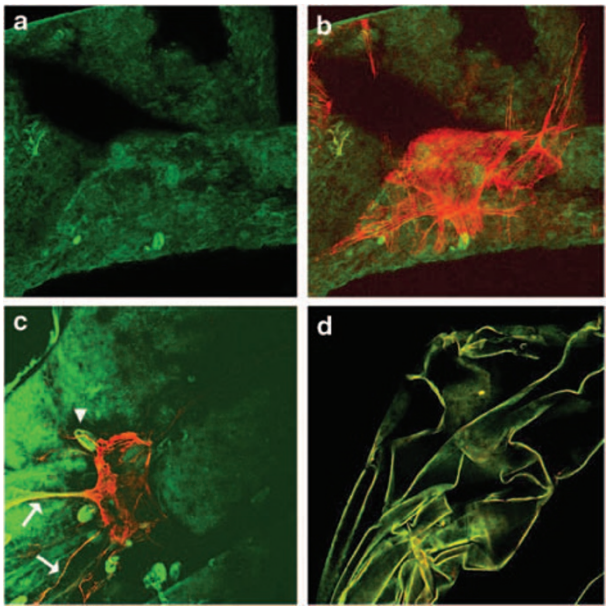

Figure 2 Confocal microscopy imaging of the internal limiting membrane (ILM) in a stage 4 idiopathic macular hole. ILM peeled around the macular hole (specimen A). (a) Fibronectin is an extracellular glycoprotein that is distributed throughout the ILM of the human retina. After immunostaining with antibodies to fibronectin, the ILM acquired a homogenous nonspecific staining (green) that was used as a marker of ILM surface. (b) α-smooth muscle actin (α-SMA), a contractile intracellular protein arranged in microfilaments, is a marker of myofibroblasts. α-SMA is coloured red. Colocalisation image showing simultaneously the ILM (in green) and bundles of actin microfilaments (in red) after immunostaining with antibodies to both fibronectin and α-SMA. Note the dense network of interweaving α-SMA microfilaments. (c) Part of the same ILM specimen showing characteristic red-coloured actin microfilaments (white arrows) on its surface (coloured green). Note also the presence of some grains of triamcinolone–acetonide (arrowhead) deposited directly on the surface of the ILM. (d) Confocal microscopy imaging of the ILM (specimen B) peeled 1–2 disc diameters away from the macular hole. Both antibodies to fibronectin and α-smooth muscle actin (α-SMA) have been used. Note that this ILM specimen does not contain any α-SMA filaments as it only stains in green.

have contractile properties. The presence of cells should be suspected when the ILM staining around the macular hole fails to stain with BB, even when OCT does not show any ERM, and should guide the surgeon in favour of ILM removal. These cells may arise from retinal Müller cells that have the capacity to change their phenotype to transdifferentiate into myofibroblast-like cells and express α-SMA.[6]

Our findings suggest that ILM may serve as a scaffold for cellular migration and proliferation and contribute to the pathogenesis of stage 4 macular hole formation. Surgical peeling of the ILM in stage 4 macular holes even without evidence of an epimacular membrane would allow the removal of contractile myofibroblasts with consequent elimination of any tangential tractional forces around the macular hole, thus leading to its successful closure. This is in accordance with a large study showing that ILM peeling is of particular benefit for obtaining anatomical hole closure in stage 3 and 4 macular holes.[7]

Conflict of interest
The authors declare no conflict of interest.

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Sir,

Ocular complications in Mulvihill–Smith syndrome

Mulvihill–Smith is an ageing syndrome with short stature, skin nevi,[1] immunodeficiency, genital abnormalities, hearing loss, and diabetes.[2,3] Reported ophthalmological findings include astigmatism, myopia, endothelial dystrophy, keratoconus,[4] cataract,[5] amblyopia, and allergic conjunctivitis. We emphasize the unreported features in this ninth case in the literature.

Case report

A 29-year-old Japanese woman presented with decreased vision and grittiness OU. The previously reported descriptive features of this syndrome, such as progeroid appearance, microcephaly, short stature, diffuse multiple pigmented nevi on the face and trunk (Figure 1a and b), sleep disorder, sensorineural deafness, high-pitched voice, non-insulin-dependent diabetes, history of recurrent otitis media, and pulmonary infections, allowed us to diagnose the syndrome in our patient. Although immunodeficiency is a feature of this syndrome, IgG, IgA, IgM levels, PHA stimulation test, and lymphocyte subpopulation analysis were unremarkable. The new, previously unreported systemic features in the medical history of our case include severe shingles, retrolingual swelling, which proved to be a granuloma on biopsy, pseudopapillary cystic pancreatic tumour and a 2-cm right cerebellar mass (Figure 1c) found incidentally on a head MRI that remained constant in size over 3 years, and low serum Mn-SOD levels measured on two occasions (248 and 150 ng/ml, respectively; normal value: 402 ng/ml). The new unreported ocular findings include dry eye disease evidenced by elevated tear evaporation rates (6.7×10^{-7} g/cm^2/s) and low Schirmer test levels (<5 mm) OU, band keratopathy, bilateral posterior subcapsular cataracts, and meibomian gland disease evidenced by lid telangiectasia, orifice closure, and marked meibomian gland drop-out compared with normal individuals in lid transillumination

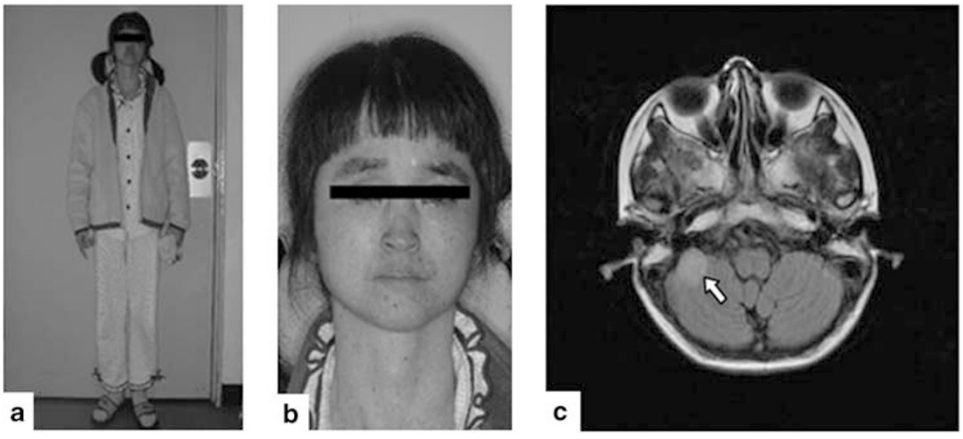

Figure 1 (a) Note the short stature. (b) Progeroid facies, multiple facial pigmented nevi, microcephaly, and mild micrognathism. (c) Magnetic resonance image (MRI) of the patient's cerebellum showing a 2 cm right cerebellar mass (white arrow).