LETTERS TO THE EDITOR

A case of Descemet's membrane detachment during phacoemulsification surgery

Editor,—Descemet's membrane detachment (DMD) was first described by Weve in 1927. DMD can be classified as planar or non-planar, the former being less than 1 mm separation of Descemet's membrane from the stroma, the latter being greater than 1 mm separation from the stroma. Further description of each of the above is possible into a 'peripheral' type and a 'peripheral with central' involvement type—that is, for the latter, peripherally and centrally there is DMD (see Table 1).

Table 1 Classification of Descemet's membrane detachment

| Planar (<1 mm separation from the stroma) | Peripheral detachment only | Membrane, peripheral and central detachment |
| Non-planar (>1 mm separation from the stroma) | Peripheral detachment only | Combined peripheral and central detachment |

DMD can be caused by any factor which is responsible for a break in Descemet's membrane, be it surgical (cataract extraction, iridectomy, cyclodialysis, penetrating keratoplasty), or the result of tears caused by congenital glaucoma, keratoconus, keratoglobus, or trauma. In particular relation to phacoemulsification surgery, superficial scleral tunnel which enters the cornea may be associated with an increased incidence of DMD.1 Additionally, there may be an anatomical predisposition, as there have been reports of bilateral DMD after cataract extraction.2

DMD is not uncommon in cataract surgery. The incidence is reported as 2-6% and 0-5% for extracapsular and phacoemulsification surgery respectively.3,4 There are, however, no statistics available for the incidence of sight threatening DMD. Previous reports of visually disabling DMD were associated with extracapsular or intracapsular cataract extraction, peripheral iridectomy, and holmium laser thermal keratoplasty.4 In 1992 Macrì5 reported the first case of a sight threatening DMD associated with phacoemulsification; this was only noted clinically on the first postoperative day. Minkovitz et al6 subsequently described a DMD occurring 1 month after phacoemulsification surgery. We describe a case of DMD, made complicated further by severe tearing of the fragment, which occurred during phacoemulsification surgery, and discuss the difficult intraoperative decisions posed by this combination.

CASE REPORT

An 80-year-old woman presented with a gradual loss of vision in the left eye. Examination of the left eye revealed 6/60 corrected visual acuity, the presence of pseudoexfoliation, and a cataract with grade 2 nuclear sclerosis and a dense posterior subcapsular lens opacity. The patient was scheduled for routine phacoemulsification surgery. Surgery was uneventful until the irrigation/ aspiration stage when a capsular or membranous entity was suddenly noted. Initially this was thought to be an anterior capsular capsulorhexis remnant. When aspiration of this membrane was attempted, the size of the membrane was seen to increase, the reflux facility was engaged, and it was then realised that this was not a capsulorhexis remnant, but Descemet's membrane. The membrane was unrolled with the help of air. Unfortunately, owing to the previous engagement of the fragment in the aspiration port, a tear had occurred nasally and temporally in Descemet's membrane, therefore leaving almost 40% of Descemet's membrane detached from the stroma; and torn from the part of Descemet's membrane still attached to the cornea (see Fig 1). Attempts were made to reposition the DMD with Healon. Owing to the large size of the fragment, and the small size of its residual attachment to the stroma, repositioning of the DMD against the stroma with air or Healon proved impossible. The other option considered was to try to reattach the DMD to the cornea at the sites of the nasal and temporal tears and in the vicinity of the corneal entrance to the scleral tunnel, where the base of the fragment had originally commenced. This was considered unfeasible given the number of sutures required, the fact that the tear and detachment of Descemet's membrane extended right across the visual axis, and finally given the sheer size of the detachment (almost 40% of the corneal area). The fragment was excised with capsulotomycises, care being taken not to increase the detachment size during the excision of the fragment. Surgery was completed in the normal way, with endocapsular insertion of an intracocular lens and sutureless closure of the 5.1 mm scleral tunnel. On the first postoperative day the patient was noted to have a geographic distribution of corneal oedema, extending from the corneal entrance of the scleral tunnel to the central cornea (see Fig 1) — that is, it coincided with the location where the DMD (and excision of same) had occurred. Subsequently the patient underwent penetrating keratoplasty, and currently she has a corrected visual acuity of 6/12.

COMMENT

There are only two cases describing visually disabling DMD associated with phacoemulsification in the literature,5,6 in both cases the DMD was noted only postoperatively. The first case combined the use of a small 4 mm scleral tunnel, and an intraocular lens injector delivery device; however, as in our case, penetrating keratoplasty was necessary. Details relating to the operative procedure in the second case were not recounted by the author. This latter case was unusual for two reasons: (1) the DMD did not occur until 1 month after operation; (2) it had an initially progressive detachment which then spontaneously resolved without intervention.

A rationale for surgical intervention was suggested by Vastine7 for DMD associated with extracapsular surgery. Small planar detachments are best observed, as many will resolve spontaneously. When, however, central or peripheral, and non-planar or scrolled detachments, require surgical intervention. The DMD can be unrolled and tamponaded against the cornea with air, sulphur hexafluoride gas, or viscoelastic; the DMD can then be secured by a reverse through and through suture technique.7

Our (intraoperative) case was particularly complex, as the DMD was complicated by the DMD occurring at XZ and YZ in Fig 1) which only failed to meet because of an intervening small apical area of residual attachment (at location Z in Fig 1). Moreover, given the type of detachment (combined non-planar), spontaneous reattachment was not considered likely. We elected, therefore, to excise the fragment in order to facilitate subsequent penetrating keratoplasty.

Apart from severe visual loss resulting from DMD, other complications can ensue. Irregular astigmatism may result owing to the formation of wrinkles in Descemet's membrane. Repair of DMD can result in instrument, and it is a removable but potentially blinding cause of postoperative corneal oedema.
Acute posterior multifocal placoid pigment epitheliopathy associated with pulmonary tuberculosis

EDITOR— The hallmark of acute posterior multifocal placoid pigment epitheliopathy (APMPE) is the appearance of multiple, ill-defined, yellow white lesions with indistinct margins that resolved spontaneously. These lesions were supposed to be due to a local immune reaction against the patient's own tissue. However, recent serological evidence has suggested that the lesions might be due to a systemic immune response against a retroviral infection.

We report a case of acute posterior multifocal placoid pigment epitheliopathy associated with pulmonary tuberculosis.

CASE REPORT

A 34-year-old woman reported to an ophthalmic casualty department with a history of progressive blurring of vision of the left eye over the previous 24 hours. Systemic inquiry was otherwise negative. There were no respiratory symptoms. She was married with an 8-month-old child, and had never smoked cigarettes. General physical examination was normal. Visual acuities were 6/60 left eye and 6/5 right eye. Multifocal lesions of pale yellow appearance with indistinct margins were seen on fundus examination of the left eye (Fig 1). These lesions were positioned within the vascular arcades with other smaller peripheral lesions. Fluorescein angiography showed early masking with late fluorescein.

The chest radiograph exhibited a round 2 cm lesion in the right middle third of the lung field. Subsequent computerized tomography showed the lesion to be in the right lower lobe. It was clearly defined with no evident calcification. The mediastinum was normal.

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Figure 1 Left fundus appearance on presentation showing multifocal pale yellow lesions with indistinct margins.

A tuberculin test (1/1000) produced 25 mm induration. There was no scar on any of the usual vaccination sites to suggest previous BCG. Haemoglobin was 14·8 g/dl, white cell count 6·2×10^9/l with normal differential count, erythrocyte sedimentation rate was 18 mm in the first hour. Serum electrolytes, creatinine, calcium, phosphate, glucose, liver function tests, albumin, and urate were normal as were serum angiotensin, anti-nuclear factor, rheumatoid factor, and syphilis serology.

Pulmonary tuberculosis was suspected and treated with isoniazid, rifampicin, and pyrazinamide. One month later she developed a productive cough. Numerous acid and alcohol fast bacilli were seen on direct microscopy of the sputum but these did not grow on culture medium during 16 weeks of incubation. The non-viability of these organisms presumably resulted from antituberculous treatment. Her lung shadow subsequently cleared in a manner typical of early tuberculosis which fully resolves with appropriate therapy.

From the respiratory aspect, her health remains excellent and she successfully completed her planned 6 month course of antituberculous therapy. Progressive pigmentation of the fundus lesions took place (Fig 2) and in this case there has been no significant improvement in vision.

COMMENT

We are not aware of any previous case published in which the clinical appearance of APMPE has been associated with other systemic asymptomatic infections, such as tuberculosis.

The alternative diagnostic label of multifocal choroiditis could, of course, be applied but in the case described it was a clinical appearance indistinguishable from APMPE which led to the systemic diagnosis.

The clinical features on presentation of our patient accord with those typical of APMPE. Our patient was young. Her visual symptoms were of rapid onset and she developed multiple posteroventral, circumscribed, flat, grey-white, subretinal lesions involving the RPE on examination by biomicroscopy and fluorescein angiography. The late features of the disorder in our patient accord with those described for APMPE but also resemble those of other white spot syndromes in which the spots are of larger size and tend to coalesce.

It is of interest that in the original description of APMPE by Gass,1 of the four cases described, two had a positive tuberculin skin test and one a family history of TB although none had overt tuberculosis. This patient's case (which has now been formally notified on the TB register) highlights that patients with the clinical appearance of APMPE warrant careful investigation for underlying tuberculosis.

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Disappearance of optomotor shunt vessels after optic nerve sheath decompression

EDITOR— Optomotor shunt vessels were described by Salzman in 1893 and have been reported in association with many ophthalmic conditions including central retinal vein occlusion, optic nerve sheath meningioma, and chronic papilloedema. The common mechanism appears to be obstruction to blood outflow in the central retinal vein which results in shunts between retinal and choroidal veins, usually at the edge of the optic disc. We report a patient with optic hydrocephalus causing chronic papilloedema associated with optociliary shunts which resolved after optic nerve