Correspondence

Retinal detachment

Sir, I read with interest the article by Ho and Tolentino.1 The probable causal element of the 14 detachments reported post capsulotomy is not emphasised in this article. It would be nice to know how and when the posterior capsulotomies were done in relation to the retinal detachments. As others have reported, and our experience supports, neodymium: YAG laser posterior capsulotomy can be associated with rhegmatogenous retinal detachment.2

In response to the authors’ statement in the concluding paragraph, ECCE (and PC-IOL) probably does minimise the incidence of postoperative retinal detachment, but posterior capsulotomy probably increases the likelihood of same.

DONALD L HALL
2751 Virginia Avenue,
Shreveport,
Louisiana 71103,
USA

References

Sir, Extracapsular cataract extraction (ECCE) and posterior chamber intraocular lens (PC-IOL) implantation are becoming the procedure of choice among cataract surgeons. Many surgeons performing this type of surgery on a regular basis would share the same concern and interest with Dr Hall about the causal relationship of posterior capsulotomy and retinal detachment.

As stated in our article,3 we were not the primary surgeons for any of the capsulotomy operations, and the nature of a retinal referral practice made it difficult to obtain details of the circumstances surrounding the capsulotomies in all 14 cases in our report. However, in four of six cases (case 3, 7, 10, and 11), the retinal detachment was detected within two weeks of the posterior capsulotomy. The temporal relationship in these cases would lead one to suspect that the detachment might have been precipitated by the capsulotomy. Furthermore, in cases 10 and 11 vitreous gel was observed to herniate anteriorly through the capsulotomy around the IOL into the anterior chamber. Although the preoperative finding of vitreous gel in the anterior chamber in front of the IOL has been reported to be associated with a poor prognosis,2 the retinal detachments in both of these cases were successfully repaired.

The pathogenetic mechanism of pseudophakic retinal detachment following posterior capsulotomy is still uncertain. In the two cases where there was forward movement of the vitreous gel into the anterior chamber one might speculate that the anterior displacement of the vitreous mass would increase peripheral vitreous traction on the vitreous base resulting in retinal breaks.4 But in the majority of our cases, and as observed by others,2 the absence of anterior movement of the vitreous in front of the iris plane would suggest an alternative mechanism for the detachment. It is generally difficult to separate visually the anterior hyaloid face from the opacified posterior capsule under the operating microscope or the slit-lamp biomicroscope. Consequently, the anterior hyaloid face is frequently violated during the posterior capsulotomy procedure. It is possible that the broken anterior hyaloid surface allows anterior movement of proteins and macromolecules from the vitreous gel, resulting in fluid shifting within an already syneryotic vitreous cavity, leading to increased peripheral retinal traction and break formation. Even with an intact anterior hyaloid surface a rent in the posterior capsule disrupts the physical barrier between the anterior and posterior segments of the eye similar to that of the aphakic eye after intracapsular lens extraction. The loss of this barrier may facilitate diffusion of hyaluronic acid, a stabiliser of the vitreous gel, into the anterior chamber,5,6 and this situation is manifest clinically as collapse of the vitreous gel.

We share Dr Hall’s impression that ECCE and PC-IOL probably minimise the incidence of postoperative retinal detachment, and posterior capsulotomy probably increases the likelihood of same. We await a well conducted prospective study to confirm our suspicion.

PATRICK C HO
Faculty of Medicine,
Chinese University of Hong Kong

References
3 Ho PC, Tolentino FI. The role of vitreous in aphakic cystoid macular edema: a review. Am Intraocular Implant Soc J 1982; 8: 258–64.

Treatment of retinal detachment

Sir, I have read with interest Gilbert and McLeod’s7 paper on the preliminary drainage of subretinal fluid in the treatment of retinal detachment. Just for the record, and for further information of those primarily concerned with retinal surgery, I wish to point out that the indications, advantages, complications, and technique of that procedure have been extensively dealt with by me in a previous report.2 Ophthalmic Clinic, A URRETS-ZAVALÍ
Univ. Nacional de Córdoba,
Casilla de Correo 301,
Córdoba 5000,
Argentina

References
3 Ho PC, Tolentino FI. The role of vitreous in aphakic cystoid macular edema: a review. Am Intraocular Implant Soc J 1982; 8: 258–64.
Correspondence

References


Sir, Your correspondent rightly draws attention to his excellent paper describing the merits of preliminary drainage of subretinal fluid, followed by intravitreal saline injection, in the management of bullous retinal detachments. However, the main purpose of the paper on D-ACE surgical sequence was to popularise preliminary fluid drainage in the context of the use of intravitreal air tamponade (which thus achieves initial break-closure and allows low-profile scleral buckling to be employed for subsequent maintenance of break-closure). Whenever intravitreal injection is contemplated in order to restore ocular volume after drainage of subretinal fluid, I would submit that there are few instances wherein fluid injection is to be preferred over air injection, thus denying the patient the benefits of internal break tamponade.

As I have recently discovered, details of D-ACE surgical sequence are also to be found in Schepens' textbook.1 Moorfields Eye Hospital, City Road, London EC1V 2PD

David McLeod

Reference


Gonococcal conjunctivitis

Sir, Gonococcal infection is a potentially blinding disease in African adults as shown by Schwab and Tizazu.1 These authors describe an epidemic of gonococcal keratoconjunctivitis that caused severe damage in 23 out of 32 (72%) infected eyes. They should be congratulated for bringing this problem to the attention of the medical community, but unfortunately they fail to recommend an adequate therapeutic approach for gonococcal conjunctivitis in order to prevent eye damage in the future.

In recent years there has been an increase of gonococcal conjunctivitis in adults and neonates in most of sub-Saharan Africa.2 With the advent of gonococcal strains which are either less sensitive to penicillin (chromosomal resistance) or totally resistant to this antibiotic (penicillinase-producing Neisseria gonorrhoeae = PPNG), severe complications are more and more encountered in referral clinics. Partly responsible for this situation are the dispensaries and peripheral health units who continue to treat gonococcal conjunctivitis with topical antibiotics and/or low dose systemic penicillin, despite this problem of resistance. Schwab and Tizazu, who considered this possibility, apparently rejected it based upon their microbiological findings. They reported that five strains tested with disc diffusion were sensitive to penicillin. Microbiological support being far from optimal in developing countries, the strains could not be tested for penicillinsase production. The possibility of plasmid resistance (PPNG strains) or chromosomal resistance among a number of their isolates therefore cannot be excluded.

In Rwanda we recently observed several cases of adult gonococcal keratoconjunctivitis comparable to the cases described in Malawi. Since the first description of a PPNG strain in this country in 1982, the prevalence has now risen to 60% in Kigali, the capital.3 Consequently our standard treatment, following recent WHO recommendations,4 consists of spectinomycin 2 g or cefotaxime 1 g intramuscularly in a single dose plus 10 days of topical 1% tetracycline ointment. With this treatment conjunctivitis subsides in 24 to 48 hours and progression of complications is immediately aborted. In Malawi some cases went on to severe eye damage although receiving high doses of penicillin, which could be indicative of causative strains resistant to this antibiotic.

Once penicillin resistant strains emerge, a switch in treatment for gonococcal conjunctivitis from penicillin to spectinomycin or one of the newer cephalosporins should be considered. Health authorities and clinicians alike often resist this change, arguing that the large majority of strains remain sensitive to penicillin and that these newer antibiotics are more expensive and less readily available. But in countries which do not have an effective gonorrhoea control programme, once introduced the PPNG strains increase exponentially and two to three years later account for up to 70% of circulating gonococci.5 The implementation of the new treatment recommendations is then an absolute necessity, otherwise gonococcal conjunctivitis again becomes a blinding disease in those countries.

Department of Ophthalmology, PHILIPPE KESTELYN Centre Hospitalier de Kigali, BP 979, Kigali, Rwanda

Epidemiology and Community Medicine, ANDRE MEHEUS University of Antwerp, Belgium

References


Sir, Drs Kestelyn and Meheus correctly point out a few of the difficulties inherent in dealing with a potentially destructive ocular infection in a developing country where medications, manpower, and laboratory support systems are limited.