Ocular complications of diabetes after cataract extraction

Cataract extraction is commonly required in diabetics since there is an increased prevalence of cataract in diabetes. These cataracts also tend to occur at an earlier age.[1-3] It is therefore necessary that ophthalmologists become familiar with aspects of cataract extraction peculiar to diabetics.

Peroperatively it is recognised that there is a higher incidence of pigment dispersion and fibrinous reaction in the anterior chamber, together with the development of posterior synechiae,[4] as well as an increased risk of capsule rupture and vitreous loss,[5] and these increased risks should be borne in mind when listing patients for surgery. It is encouraging, however, to note that there appears to be no detrimental effects on the corneal endothelium following cataract surgery in diabetes,[6] and this is confirmed by animal and postmortem studies.[7] A posterior chamber intraocular lens (IOL) is the commonest and most satisfactory way to correct aphakia in diabetics, but the problem arises as to how best to correct aphakia if posterior capsule rupture and vitreous loss preclude the insertion of a posterior chamber IOL. Anterior chamber IOLs are likely to exacerbate the tendency to an increased inflammatory reaction in the anterior chamber, and the subsequent development of posterior synechiae are likely to compromise pupillary dilatation and so lead to poor visualisation of the fundus with the obvious implications in the assessment of diabetic retinopathy. It is perhaps best simply to perform an adequate anterior vitrectomy with particular attention being paid to the removal of capsule remnants, and to decide how best to correct the aphakia at a later date. Owing to the increased inflammatory reaction in diabetics, it may well be that heparin coated IOLs are appropriate here, but their value is yet to be proved. The alternative of correcting aphakia with a contact lens is, in addition to the usual disadvantages, a particular problem in diabetics where the epithelium is less securely attached to the underlying basement membrane resulting in an increased frequency of recurrent corneal epithelial breakdown.[8] The necessity of a good fundal view and the possibility of laser treatment should always be borne in mind when considering cataract surgery in diabetics, and hence the IOL used should have as large an optic as possible. The current tendency, especially with phakoemulsification, to perform a small capsulorhexis with a small optic IOL should be avoided since this may well compromise a satisfactory view of the more peripheral retina as the capsule thickens.

The effect of cataract surgery on the progression of ocular neovascularisation in diabetes was the subject of an editorial in this journal two years ago[9] and little more need be said about this other than to note that even though extracapsular extraction and IOL insertion are much safer than intracapsular cataract extraction, a close watch on the retinopathy is required in the first few months of the postoperative period since in one series half of all patients with 'quiescent' treated proliferative retinopathy went on to require further laser photocoagulation.[10]

It is important to consider whether cataract surgery per se might contribute to progression of non-proliferative diabetic retinopathy and there is now increasing evidence that not only may this occur, but also that background diabetic retinopathy may develop following cataract extraction when no retinopathy was noted before.[2 11-13]

Although it has been suggested that the presence and type of posterior vitreous detachment may have an effect on macular oedema in diabetics,[14 15] these findings are confusing to interpret with a 'syneretic' type of aging posterior vitreous detachment tending to confer a protective effect against the development of macular oedema in older patients, whereas a non-syneretic posterior vitreous detachment occurring in younger patients (under 50) is thought to be specifically associated with diabetes and to contribute to macular oedema.

The main features involved in the progression of retinopathy following cataract surgery are likely to be inflammatory in origin and whatever the stimulus the retinal capillaries appear to mount a pathological response to surgery with resulting disruption of the blood-retinal barrier.[11] This would be very much in keeping with an increased anterior segment inflammatory reaction noted following cataract surgery. The paper by Menchini *et al* in this issue looks in detail at the development of cystoid macular oedema following extracapsular cataract extraction with IOL insertion in diabetics without retinopathy and compares this with non-diabetics. A significant increase in the development of cystoid macular oedema is seen in diabetics after 1 year compared with non-diabetics as shown on fluorescein angiography.

These results are remarkably similar to those of Pollack[12] with both papers finding, at 1 year postoperatively, signs of cystoid macular oedema on fluorescein angiography in 25% of patients with diabetes but without retinopathy at the time of surgery compared with none in the non-diabetic eyes in Menchini's paper. While none of Menchini's diabetics developed diabetic retinopathy, even on fluorescein angiography, the development of angiographic cystoid macular oedema indicates a breakdown in the blood-retinal barrier which, were it to be added to an existing background retinopathy, might well be expected to convert this to overt diabetic maculopathy. Bearing this in mind it behoves ophthalmologists performing cataract surgery in diabetes to pay particular attention to the assessment of any retinopathy developing or progressing in the postoperative period, particularly since nowadays there is an increased tendency to reduce the number of postoperative visits and to discharge patients early following cataract surgery. It is important to examine such patients carefully postoperatively to prevent these problems being missed in the early stages.

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