The origin of macular cysts in diabetic maculopathy

Macular oedema is a non-specific pathological response to a wide range of ocular disorders. If the oedema is severe and prolonged cysts form at the fovea and in the perifoveal neuroretina. Lamellar hole formation and central visual loss are the common result. Histopathologically, the oedema first accumulates between the inner nuclear and outer plexiform neuroretinal layers. Fluid filled cysts then form in Henlé's (outer plexiform) layer. In severe cases the entire retinal thickness in involved and the fovea is no longer identifiable.

Cystoid macular oedema (CMO) associated with diabetic maculopathy has been attributed to inner retinal capillary changes causing ischaemia. However, some eyes with marked macular capillary changes do not develop CMO and can retain reasonable vision. This suggests that retinal capillary abnormalities are not the only contributor to CMO in diabetics. Coexisting changes in the retinal pigment epithelium, choroid, vitreous, or systemic circulation may be necessary for CMO to occur. In some conditions, such as pseudophakic CMO or where the causative intraocular inflammation can be treated, the cysts can resolve with improvement in vision. In inherited retinal pigment epithelial disease acetazolamide, presumably through an effect on pigment epithelium, can cause the oedema to dry up.

Cystoid changes associated with diffuse diabetic macular oedema or neglected focal diabetic maculopathy frequently have a poor prognosis and a disappointing response to photocoagulation. Only one good prospective study has shown a satisfactory response to photocoagulation for diffuse diabetic macular oedema. CMO associated with very poor glycaemic control and/or renal failure may improve with correction of these metabolic disturbances, but this is not always the case.

Diabetic maculopathy remains a major cause of severe visual handicap and a serious public health problem. This is confirmed by two recent publications. Clarke et al reviewed blind registration data and found that 72% of all registrations were due to visual loss from diabetic maculopathy. The 15th report of the Wisconsin Epidemiological Study of Diabetic Retinopathy reported on the incidence of macular oedema in a cohort of diabetics followed for 10 years; 20-1% of the younger onset group, 25-4% of the older onset group on insulin, and 13-9% of the older onset group not on insulin developed macular oedema. Hyperglycaemia is undoubtedly a major factor in the pathogenesis of this condition. This was confirmed by the Diabetes Control and Complications Trial. In this trial the incidence of macular oedema in younger onset diabetics was 23% less in the tightly controlled group compared with the group receiving conventional insulin therapy. Other factors may be relevant particularly in older onset diabetics who are most susceptible to visual loss from macular oedema.

The paper by Arend et al in this issue of the BJO (p 628) compares macular capillary changes in diabetics with and without CMO. The authors used fluorescein angiographic images from a scanning laser ophthalmoscope to calculate the macular capillary blood velocity (CBV), the perifoveal intercapillary area (PIA), and the size of the foveal avascular zone (FAZ) in non-diabetic controls, as well as in diabetics with and without CMO. Not surprisingly there was a marked difference in all these variables between the diabetic eyes and the non-diabetic control eyes. However, there was no real difference in the CBV, the PIA, or the size of the FAZ, between diabetics with and without CMO. This suggests that inner retinal ischaemia due to capillary changes is unlikely to be the cause of CMO. The groups were carefully controlled for duration of diabetes, HbA1c concentrations, and diastolic blood pressures so systemic factors are unlikely to be relevant.

If, as this information suggests, inner retinal capillary changes and ischaemia are not the main causes of CMO, changes in the vitreous or the retinal pigment epithelium and choroid may be relevant.

Vitreous traction has been implicated in aphakic CMO when vitreous is adherent to the surgical section. Although the role of vitreous in proliferative diabetic retinopathy has been investigated extensively its possible contribution to diabetic CMO has received little attention. One paper by Nasrallah et al found that older diabetics without macular oedema were more likely to have a detached vitreous than those with macular oedema. The difference was statistically significant but the numbers in the oedema group were small.

The retinal pigment epithelium forms the outer blood-retinal barrier so retinal pigment epithelial dysfunction and/or choroidal ischaemia might allow oedema to persist and cysts to form. Experimental streptozotocin induced diabetes in animals causes a breakdown in the outer blood-retinal barrier which can be demonstrated by tracer studies using electron microscopy. The improvement in CMO in inherited retinal pigment epithelial disease following treatment with acetazolamide confirms that retinal pigment epithelial dysfunction can cause CMO. However, acetazolamide has not been shown to be of value in CMO associated with retinal capillary disorders. Fluorescein
angiography even with a scanning laser ophthalmoscope provides little information about chorioidal and choriocapillaris blood flow. This is particularly so in CMO where the thickened retina masks the choriocapillaris completely. It is tempting to speculate that CMO only occurs when retinal pigment epithelial function is seriously and permanently compromised by chorioidal ischaemia. This would explain why diffuse CMO does not respond to grid photocoagulation. Chronic CMO is more common in older onset diabetics. This could be as a result of the choroid in such patients being more ischaemic than in young patients. Severe CMO can occur in young diabetics after extensive panretinal photocoagulation or as part of pro-proliferative retinopathy. However, it usually clear spontaneously in such cases possibly because of an underlying healthy choriocapillaris.

Hopefully, indocyanine green angiography with a scanning laser ophthalmoscope will provide information on chorioidal blood flow in diabetics to allow these questions to be answered in the near future.

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Visual impairment in children

Significant visual impairment in children has long been overlooked and underestimated in terms of prevalence, personal impact, and the social and economic implications. The publication in 1992 by the World Health Organisation (WHO) of 'Prevention of childhood blindness' attempted to estimate the extent of the problem and target the issue of prevention. This is a critical issue, as it is estimated that up to 75% of childhood blindness is preventable or curable. Without access to medical and surgical treatment, the majority of these children will remain with impaired vision. It is imperative that children have access to special education and (re-)habilitation programmes to alleviate the effects of visual deprivation.

The WHO estimates the number of blind children in the world to be 1-5 million with half a million new cases of childhood blindness each year. Based on this figure of prevalence of blindness, Eckstein and colleagues, in a paper in this issue of the BJOP (p 633), have calculated that this accounts for 75 million years of blindness. To the figure of 1-5 million had to be added those children whose vision is impaired to the extent that they are classified as having low vision. From the 1994 WHO update of country statistics on blindness and low vision, 'Available data on blindness', there are typically three times as many people with low vision as there are blind.

There are difficulties in obtaining accurate data on the prevalence and causes of visual impairment in children. This has been demonstrated in Britain where the survey conducted by the Royal National Institute for the Blind found that the registration figures have grossly underestimated the prevalence of visual impairment in both children and adults. An approach often used is to survey schools for the blind to establish data for a given area or country. It is more practical to travel to a limited number of sites to examine substantial numbers of children in schools for the blind than to travel to either a large number of schools where a few children are integrated or to visit homes or villages in a population based survey. Care must be taken with generalising data obtained from schools for the blind to the whole country, as the population of the schools is unlikely to be representative of all children with impaired vision. The numbers attending schools for the blind are usually relatively small with greater numbers of students in 'integrated' education and, in developing countries, many or most children may not even attend any school. There are also likely to be children with impaired vision in other special schools for the disabled. The eligibility criteria for enrolment in a school will also influence the degree of impairment of its students and there may also be an unrepresentative sample of the causes of visual impairment, age range, ethnic groups, health, and socioeconomic status within a country or region. Any changes in educational policy and practice will make it difficult to compare data over a period of time.

Although vision screening to establish prevalence data on blindness and low vision can be carried out in the community by health workers, it is more difficult to collect information on the causes of visual impairment. This can only be determined after an ophthalmic examination. Strategies for prevention need to be based on knowledge of the causes of impaired vision. The survey reported in this issue, which was conducted in Sri Lankan schools for the blind, used the WHO/PBL eye examination record for children with blindness and low vision form. This useful new form records comprehensive information on each child as to the extent and cause of impaired vision.

References