MINI REVIEW

# Subretinal membranes of proliferative vitreoretinopathy

Subretinal membranes (SRMs), also known as retroretinal bands or membranes, are a well recognised complication of rhegmatogenous retinal detachment and as such are part of the spectrum of proliferative vitreoretinopathy (PVR).[1] Proliferative vitreoretinopathy SRMs are of two main types. The majority tend to form diffuse cell sheets which do not interfere with retinal attachment and over which, in the absence of contractile epiretinal proliferation, the retina may be reattached with scleral buckling procedures alone.[2] The second group of PVR SRMs embodies taut membranes or bands (often in an annular configuration) which raise the neuroretina and impede conventional retinal detachment surgery. This second, problematic type of SRM occurs in 13% of patients with PVR[3] and usually requires microsurgical division. An internal approach to tensile SRMs, such as described by Machemer in 1980,[4] may enable retinal reapposition and, incidentally, provide specimens for research.

Laboratory investigations provide some information on the pathobiology of proliferative vitreoretinopathy SRMs. Subretinal membranes in PVR are cellular or fibrocellular in composition and most are avascular. Diffuse sheets of subretinal cells which do not obstruct retinal apposition ('simple SRMs') are usually glial and contain little or no extracellular material. By contrast taut SRMs are fibrocellular, and up to 95% of the cells are retinal pigment epithelial in origin, while the extracellular component includes fibrin and collagen types I to IV.

Of particular interest is the source of the tension in problematic subretinal membranes. Since the SRMs do not appear to contain elastin, it is thought that the traction is generated by contractile retinal pigment epithelial cells with some characteristics of myofibroblasts.[5] However, myofibroblast-like cells are not always present in tensile SRMs, though the absence of such cells might relate to delayed removal of the membranes.[6] Nevertheless SRMs probably develop over many months, and so the absence of myofibroblastic cells in some specimens is mysterious if such cells are the origin of the traction.

Whatever the mechanism(s) of the tension in subretinal membranes in cases of PVR, there is no pharmacological means to counter it in vivo, and therefore the management of patients with problematic SRMs relies on vitreoretinal surgery. However, when a surgical approach to SRMs is required, the visual outcome is frequently disappointing, with only a 20% chance of a restoration of ambulatory vision.[3] Thus the prevention of PVR membrane formation, by a combination of early surgical and drug intervention in retinal detachment,[7] is an important research avenue in which carefully planned antiproliferative drug regimens may come to have a major role.

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