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EDITORIAL

Rhegmatogenous retinal detachment: time to consider real prevention

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Ideally in medicine, to manage a disease certain criteria must be fulfilled. First, it has to be recognized that a specific condition exists and what its features are (*diagnosis*). Secondly, understanding how that condition forms (*pathogenesis*) helps in developing the optimal therapeutic approach (*treatment*, as opposed to natural history). Finally, a method should be designed to try to prevent the disease (*prophylaxis*).

It was recognized in 1920 by Gonin that in most eyes with a retinal detachment (RD) the visible etiology is a break (rhegma) in the neuroretina. The diagnosis thus became straightforward and it also alluded to the pathogenesis, marking the target for treatment: closing the break would allow retinal reattachment. The underlying pathology has been finally confirmed to be dynamic vitreoretinal (VR) traction [1], which causes break development and subsequent separation of the neuroretina from the retinal pigment epithelium (RPE). VR traction exists in every eye with a rhegmatogenous RD, whether the patient notices flashes or not.

In the 1930s, the success rate of treating an eye with an RD was below 60% [2]. The breakthrough in increasing the chance of retinal reattachment to ~80% came with the introduction of cryopexy and scleral indentation; the former addressed the retinal break by sealing it, the latter the traction force by pushing the eye wall in so as to bring the retina into contact with its foundation. With time, re-establishing this contact was hastened by injecting air (later gas) into the vitreous cavity.

The introduction of vitrectomy into the surgeon's armamentarium allowed definitive treatment of the VR traction as well as coexisting pathologies, such as vitreous hemorrhage, which previous treatment modalities could not address. By reattaching the break prior to retinopexy, vitrectomy also allowed replacement of cryopexy with laser as the sealing vehicle. As a result of all these developments (along with many others, including refinement of the vitrectomy machines), a patient presenting with a fresh RD can expect a~90% success after one surgery, and this number increases with reoperation/s.

Every physician knows, however, that prevention is preferable to cure, no matter how effective the cure is. In most patients, the risk of RD development is similar in the two eyes – exceptions include anisometropic high myopia or single-eye mechanical trauma. In the typical patient presenting with an RD, the fellow eye has an annual risk of ~4% to also develop RD [3]. The recognition of this risk naturally leads to the question of prophylaxis: should the surgeon also offer the patient, at the time of RD diagnosis in one eye, RD prevention in the fellow eye, and if yes, what kind of prevention?

The typical retinal break leads to detachment when the VR traction overcomes both the retinal resistance (so that a tear can form) and the forces keeping the neuroretina attached (mostly the centropetal fluid transport by the RPE plus the interphotoreceptor matrix) so that the retina can be pulled off its foundation. The location of most causative breaks is between the ora serrata and the equator. The prophylactic treatment must address this entire region, and not simply the sites that a "careful examination of the retinal periphery" identifies as "high risk", such as lattice degeneration: in up to 90% of the cases the break occurs in an area where the examination did not reveal an elevated risk for break and thus RD formation [4].

Cryopexy is an obvious weapon for RD prophylaxis: it has been successfully employed in retinopathy of prematurity as well as in Stickler's syndrome. However, cryopexy leads to significant morbidity and also causes intraocular inflammation, increasing the risk of the development of

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proliferative vitreoretinopathy, the major cause of failure of RD surgery.

Laser retinopexy, on the other hand, offers effective RD prevention with a minimal risk of causing complications. As explained above, the laser treatment works best when it is applied not only focally ("where there is visual pathology threatening the development of a retinal break") but 360 degrees: encircling treatment, laser cerclage.

Laser cerclage, using a pattern of a few rings, was attempted decades ago and has been deemed ineffective; given a bad name, it was subsequently abandoned. It took some time for surgeons to realize that the problem is not that "laser cerclage does not work" but that "inadequate laser cerclage does not work". (This is similar to someone claiming, after driving a Fiat 500, that "cars cannot reach a speed of 200 km/h" – just tell that to the owner of a Porsche.)

Proper laser cerclage [5] covers the entire area, 360 degrees, between the ora serrata and the equator – the Scott line, peripheral to which the retina and vitreous are inseparable, is rarely posterior to the equator, and this is where retinal breaks form. In an emmetropic eye this means~1,200 moderate burns, sparing the 3 and 9 o'clock meridians where the long ciliary nerves are found. In eyes with a very high risk of RD development, such as in Stickler's syndrome or with light pigmentation, the burns should be more intense and more tightly spaced.

Laser can de delivered during vitrectomy if proper scleral indentation is possible so that the treatment truly covers the *entire* area between the posterior border of the treated retina ("ora secunda") and the ora serrata, or via the indirect ophthalmoscope as an outpatient procedure. The risk of the procedure if the guidelines outlined above are followed, is ~1% of clinically significant macular-pucker formation. While this risk is not zero, both its incidence and severity are much lower than those of the condition it aims to prevent.

Laser cerclage, when done properly, has now been strongly proven to be both safe and highly effective in preventing RD development in the fellow eye. It should thus be offered during counseling to every patient presenting with a retinal detachment as long as the RD risk is the same as it was in the affected eye. It should be then up to the patient to decide whether to choose prophylactic laser or live with the ~4% annual risk of RD formation in the second eye.

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