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Commentary

Retina - A New Look at Treating Severe Diabetic Retinopathy

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Abstract

Diabetes has been increasing both in incidence and prevalence throughout the world. New trends in nutrition and an easy going fast food behaviour, as well as genetic factors play a role in this increment.

Diabetic retinopathy, as a consequence, is more frequent in the office, and patients come due to low visual acuity secondary either to macular edema or complications of proliferative diabetic retinopathy, the first leading the retina specialist to treat the patient with laser, intraocular injections and the latter will oftenly end up leading the patients to the operating room, consequent to, usually, vitreous hemorrhages, retinal detachments and others.

Some patients are not treated adequately and consequently are diagnosed as severe proliferative diabetic retinopathy, and clinical treatment may not work and even after intraocular injections of antiangiogenics, develop the entity called Crush Syndrome, meaning they respond badly to intraocular injections, their retinopathy gets worse in less time than usual and many patients have to be taken right away towards surgical treatment.

Tie 2 is a tyrosine kinase receptor and in combination with vascular endothelial growth factor (VEGF) therapy make the outcome of diabetic retinopathy better. Drugs are also used for that purpose, and act as anti-VEGF, modulating the response of the patient with diabetic retinopathy and the above complications mentioned. The aim of this investigation is to propose a switch from clinical to surgical treatment when the patient develops this imbalance in the disease homeostasis, being diagnosed as Crush Syndrome.

Keywords: *Diabetes; Diabetic Retinopathy; Vitreoretinal Surgery; Laser Photocoagulation; Vitrectomy; Intraocular Tamponades; Gas; Silicon Oil*

Diabetes may lead to diabetic retinopathy. Some factors may accelerate this process, and get the disease worse in no time so as to end up in Crush Syndrome, causing severe vision loss.

Since gestation, angiogenesis relies on the Tie pathway for the vascular formation and homeostasis, thus avoiding inflammation, keeping the vessels in a normal state.

In a normal condition, an angiopoietin 1 with is a Tie 2 ligand, activates Tie 2 after binding to it, keeping the eye rid of vascular malformations such as new malformed vessels.

Since angiopoietin 2, which is not a Tie 2 ligand, not causing Tie 2 receptor activation, and that may cause inflammation and vascular imbalance, with vascular leakage and malformations. In bad controlled patients with severe diabetic retinopathy, Crush Syndrome may ensue and a prompt approach must be considered, to avoid complications, to mention blindness.

How can this be explained?

Crunch Syndrome is usually a fast manifestation of treatment for severe diabetic retinopathy and happens very fast, usually after intraocular injections of antiangiogenics for the disease. Some intraocular drugs may have an unknown effect that worsens the disease more rapidly, as compared to others. Usually, the candidate for the complication is a poorly controlled diabetic, has fibrovascular proliferations (with severe diabetic retinopathy), and retina traction with imminent or already diagnosed tractional retinal detachment.

We propose an approach as to submit the patient to vitrectomy after crunch syndrome is detected, in this way avoiding blindness and controlling the disease.

Modern vitrectomy machines with high cutting rates, together with the advantage of small gauge approach usually give the patient a good benefit from the surgery, if this is a decision taken in due time. The results may be promising and surveillance of the patient is necessary with regular visits and follow ups to guarantee a good result.

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