Introduction

Vitreous surgical procedures have been performed to relieve vitreoretinal tractions or adhesions to facilitate reattachment of a detached retina and to reduce retinal odema. The level of difficulty of vitreous surgery depends on presence or absence of posterior vitreous detachment and the degree of adhesion between vitreous body and the retina ***(Sakuma T, 2005).***

In particular, diseases such as proliferative diabetic retinopathy, macular hole, and proliferative vitreoretinopathy are associated with pathologic changes at the vitreoretinal interface induced by anomalous posterior vitreous detachment ***(Gass JDM. 1995).***

The techniques and instruments for vitreous surgery have greatly improved in recent years. However, the surgical removal of the vitreous cortex is still difficult in some patients and carries the risk for complications such as retinal breaks, retinal detachment, and retinal nerve fiber damage ***(Sebag J. 1987).***

Therefore, it would be helpful to have a biochemical agent that could cleave the vitreoretinal interface selectively without damaging the retina. if the vitreous can be liquefied or if enzymes, either alone or in combination with vitrectomy, can weaken adhesion of the vitreous to the retina, these changes would decrease the risk for surgical complications. Several enzymes have been explored for this purpose ***(Gandorfer A et al., 2004).***

In addition, other agents changing the osmolarity of the vitreous in the past have also been suggested to be able to manipulate the vitreous and create either more liquefied vitreous or perhaps even posterior vitreous separation. The issues of enzymatic assembly must be considered a recombinant versus autologous enzymatic agents. All of these biochemical pathways have as their common goal to potentially

manipulate either central vitreous collagen or the vitreoretinal interface. Enzymes have been suggested as adjunctive therapy to vitreous surgery and have included chondroitinase, hyaluronidase, dispase, and plasmin enzyme ***(Harbour JW et al., 1996)***, and microplasmin ***(Nagai N et al., 2003).***

Pharmacological vitreolysis, also known as enzymatic vitrectomy utilizing autologous plasmin, to facilitate the peeling of the vitreoretinal interphase in pathologies such as macular hole, macular epiretinal membranes, proliferative vitreoretinopathy in the course of premature retinopathy, proliferative diabetic retinopathy and / or tractional retinal detachment ***(Gandorfer A. 2007).***

Plasmin is a protease involved in fibrinolysis. Its enzymatic action affects laminin and fibronectin, both located between the posterior vitreous cortex and the retinal internal limiting membrane and are thought to be the molecules mainly responsible for firmly adhering both surfaces ***(Li X, 2002).***

Enzymatic vitreolysis, which for years has held the promise of minimally invasive solutions for surgical problems in vitreoretinal diseases such as diabetic retinopathy and macular hole. Enzymatic vitreolysis is envisaged to augment or even replace standard mechanical vitrectomy, over which it presents important advantages. It offers lower operative risks, less surgeon time, and lower costs ***(Cullinane AB, 2000).***

Nevertheless. As the limits of conventional vitrectomy are being approached vitreoretinal surgeons continue to look forward over the next years to new generation of therapies with vitreolytic enzymes ***(Trese MT, 2000).***

Aim of the work

The aim of the study is to discuss enzymes that have been suggested as adjunctive therapy to vitreous surgery regarding Their preparations, applications, and their potential side effects and toxicity.