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Effect of Intrastromal Injection of Suramin in Treatment of Corneal Angiogenesis in a Rabbit Model

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Introduction: Suramin (Sigma-Aldrich, EUA) is an antineoplastic drug that has multiple potential mechanisms of action, including *in vitro* and *in vivo* inhibition of VEGF, bFGF, IGF-1, PDGF, TGF- β and kinase C protein [1, 2].

Purpose: To analyze the effect of intrastromal administered Suramin on experimentally induced corneal neovascularization (NV) in a rabbit model.

Methods: NV was induced by silk 6.0 suture in peripheral cornea from 8 New Zealand rabbits and were randomly distributed into three groups:

Control Group (n=4): Received intrastromal injection (30G) of Balanced Saline Solution (BSS®) 14 days after injury.

Suramin Group 1 (n=2): Received 8mg/0,2mL intraestromal injection (30G) of Suramin 14 days after injury.

Suramin Group 2 (n=2): Received 4mg/0,2mL intraestromal injection (30G) of Suramin 14 days after injury.

Standardized biomicroscopic photographs were taken at days 7, 14, 21 and 28. NV areas were processed and morphometrically analyzed by Image J 1.31v software (Wayne Rasband at the Research Services Branch, National Institute of Mental Health, Bethesda, MD, USA).

Quantitative and qualitative analysis between the NV area growth and/or regression among each group was made.

Results:

The majority of silk 6.0 suture-induced corneal NV was superficial.

0,2 mL intrastromal injection was little, thus not filling entire cornea.

Intracameral injection of Suramin occurred accidentally in one rabbit of the Group 2. The animal showed no different ocular effects than the others.

All the animals presented progressive increase in the NV area along the 28 days follow up, though in different degrees. Average NV area at D28 was largest in Group 2 followed closely by Group1. Control group surprisingly featured conspicuous smaller areas. (Table 1, Graphic 1).

The NV area of the D14 was considered as 100% and Graphic 2 shows relative progression of NV along time.

Qualitative analysis revealed a decrease in neovascular branching and density at D28 in all groups (Fig. 1).

In addition, loss of corneal bright and a whitish intrastromal deposit were

observed in all rabbits after the injection. Those alterations persisted through the whole studied period. The opaque white deposits however turned translucent along the time (D28). (Fig. 1)

Conclusions: Although Suramin has proved to inhibit corneal NV in rats[1] and in rabbits [3] we found different results in this pilot study. Intrastromally injected Suramin may maximize or even yield an intense NV process in comparison to control. We speculate that such unexpected results may be caused either by a direct action of the drug in the corneal stroma or by secondary injection related inflammation.