

# Caution urged in steroid treatment for laser retinal injuries

**Dermot McGrath  
in Monte Carlo**

STEROID therapy to treat traumatic laser retinal injury should be used with caution in order to avoid potentially severe complications with wound healing, according to an American researcher.

In a presentation here at the 5th International Symposium on Ocular Pharmacology and Therapeutics (ISOPT), Stephen T Schuschereba PhD, said that he had set out to test the hypothesis that treatment with the glucocorticoid methylprednisolone would reduce short-term secondary injury from laser retinal injury as well as improve long-term outcomes.

"No evidence-based therapeutic regimens have been developed for traumatic laser retinal injury, but most clinical interventions currently involve the use of glucocorticoid therapy. The purpose of this study was to determine if high-dose steroid use is justified for medical management of traumatic laser retinal injuries. We concluded that in severe laser-induced retinal trauma, the immunosuppressive effects of high dose methylprednisolone therapy contributed to a variety of untoward wound healing outcomes, thereby suggesting caution in its use to treat similar injuries in humans," Dr Schuschereba said.

Dr Schuschereba, who works at the U.S. Army Medical Research Department in San Antonio, Texas, said that primary laser-induced retinal damage such as photo-thermal and photo-mechanical processes induced the onset of early stage secondary damage such as oedema, ischaemia-reperfusion injury, lipid peroxidation and inflamma-

tion. These in turn can lead to late stage tertiary damage such as scarring, scar remodelling and neovascularisation, which can contribute to retinal traction, retinal hole formation, detachments, continued tissue degeneration and vision loss.

Although damage events responsible for tissue degeneration and vision loss are becoming better understood, Dr Schuschereba pointed out that no effective or results-based therapeutics can be confidently recommended for therapy to spare vision.

In the study, 37 New Zealand Red rabbits were either dosed with methylprednisolone sodium succinate about 20 minutes before laser irradiation or were left

untreated. Dosing with methylprednisolone was tapered from 30 mg/kg/day to 10 mg/kg/day for five consecutive days. A multi-line argon laser was then used to produce retinal injuries near haemorrhaging levels. A variety of funduscopy and histologic assessments were made from 10 minutes to six months after injury.

Fluorescein angiography showed that control lesions stopped leaking at three days post injury, but methylprednisolone-treated lesions leaked for two to four days longer. After one month, methylprednisolone-treated lesions increased in area while controls became reduced.

Histologic analysis showed no effect on reduction of neutrophils (PMN) in methylprednisolone-treated lesions over controls at three hours. After 24 hours, retinal PMN values in hemorrhagic lesions of the methylprednisolone group were significantly elevated ( $p < 0.05$ ) while monocyte/macrophage counts were reduced ( $p < 0.05$ ) compared to control.

After just four days, retinal lesions treated with methylprednisolone showed suppressed cell proliferation and lack of cell filling-in for lost retinal elements. There was also evidence of early stage retinal hole development in all lesions types, leading to definitive retinal hole development at one

month followed by extensive scarring in both the retina and the choroid at six

months. Furthermore, he said that the inhibition of retinal glial cell proliferation by methylprednisolone contributed to retinal holes that resulted around one month after treatment. After six months, increased chorio-retinal scarring occurred, and bearing all these points in mind, methylprednisolone therapy to treat laser-induced retinal injuries should be used with due caution, he added.

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Putting the data into perspective, Dr Schuschereba said that the key findings of the study included the fact that the blood-retinal barrier is delayed by methylprednisolone, suggesting suppression of RPE cell activity.

It was noteworthy also that the acute inflammatory response after trauma – leading to secondary damage – was not curtailed by methylprednisolone and that debris clearance in the wound may be impeded by the steroid due to suppression of macrophage activity.