Steroids are commonly used for a wide variety of ophthalmic disorders and generally have a very high safety profile when used in the short term. The potential for ocular steroids to cause ocular hypertension or glaucoma is widely known by eye-care professionals and cases of visual loss due to steroid-induced glaucoma continue to occur, particularly in patients who are not monitored or who self-medicate. Because this vision loss is preventable, it is important that all ophthalmic practitioners consider when steroid use is appropriate, which patients are at risk of developing steroid-induced glaucoma, and how to monitor, manage or refer patients with possible steroid-induced glaucoma.

Steroids are an important and commonly prescribed treatment for a diverse range of inflammatory eye disorders including dry eye, allergic eye disease, inflammation following eye surgery, uveitis, diabetic macular oedema and many others.

Steroids can cause a rise in intraocular pressure (IOP) in susceptible individuals. Topical steroid use for six weeks causes an increase in IOP of >5 mmHg in approximately 20 per cent of the population and >15 mmHg in five per cent. Following an intravitreal steroid injection, about 40 per cent of patients develop a significant IOP elevation. Individuals who develop ocular hypertension in response to steroid use are termed ‘steroid responders’. The rise in IOP is believed to be due to increased aqueous outflow resistance associated with steroid-induced structural and biochemical changes in the trabecular meshwork.

Who is at risk of steroid-induced glaucoma?
A steroid-induced rise in IOP may not be associated with optic nerve damage or vision loss, in which case it is termed steroid-induced ocular hypertension. Patients with healthy optic nerves can tolerate higher IOPs and longer periods of ocular hypertension before their nerves develop sufficient damage to cause vision loss.

If the IOP rise is high enough or sustained for long enough, then optic nerve damage may occur, leading to steroid-induced glaucoma. Patients who have pre-existing glaucomatous optic nerve damage are at an elevated risk of vision loss from steroid-induced glaucoma. Most steroid-induced glaucoma is caused by steroids administered via eye-drops, periocular or intravitreal injections. It can also occur when steroids are given orally, in topical skin cream, and even following the use of nasal or oral inhaled steroids for the treatment of hay fever or asthma. The likelihood of an IOP rise occurring with a particular route of administration, starting from most likely to least likely is intravitreal > periocular > topical > systemic.

Steroids with more potent anti-inflammatory effects are more likely to cause an elevated IOP, occurring more rapidly and to a greater level of IOP than weaker steroids. The likelihood of an IOP rise occurring with a particular steroid, from most likely to least likely is dexamethasone 0.1% > prednisolone 1.0% > fluorometholone 0.1% > hydrocortisone 0.5%.

Recognised risk factors for developing steroid-induced ocular hypertension or glaucoma include patients with open angle glaucoma and their first-degree relatives, high myopes, old age or age younger than six years, patients with traumatic angle recession, and patients with a high baseline IOP.

How to monitor patients for potential steroid-induced glaucoma
All patients being treated with ocular steroids should have a baseline IOP and optic disc assessment, especially those with specific risk factors for steroid-induced glaucoma, for example, patients with open angle glaucoma, high myopes, children and cataract.

Patients should be warned about the risk of elevated IOPs that may lead to steroid-induced glaucoma if left unmonitored. The importance of complying with follow-up arrangements should be continually emphasised to the patient.

Common clinical scenarios in which a steroid-induced IOP rise occurs are when topical steroids are used to treat post-operative inflammation following cataract or other eye surgery; when topical steroids are used to treat anterior uveitis; and when periocular or intravitreal steroids are used to treat macular oedema associated with diabetic retinopathy, retinal vein occlusions or cataract surgery.
The timing of the IOP rise varies with the potency, dose and route of steroid administration as well as individual patient factors. A rise in IOP usually occurs within the first few days or weeks after starting steroid treatment, thus all patients on steroids should have their IOP and optic nerves monitored typically within two weeks, or sooner if the patient is at high risk, after starting treatment. Occasionally, the IOP rise can occur only after several months so it is important to monitor IOP regularly as long as steroid treatment is continued.

Steroid-induced glaucoma causes the same optic disc and visual field changes as those associated with primary open angle glaucoma. Patients with suspected steroid-induced glaucoma should have visual field testing possibly combined with objective assessment of the optic disc and retinal nerve fibre layer, for example, using optical coherence tomography. Visual field assessment can be difficult because many patients on steroids have impaired visual function due to their underlying eye disease.

**Treating steroid-induced ocular hypertension and glaucoma**

The decision regarding when and how to treat steroid-induced ocular hypertension or glaucoma requires sound clinical judgement, taking into account the condition being treated, individual patient factors, the severity of pre-existing optic nerve damage and the level of IOP rise. A rise in IOP from 14 mmHg to 19 mmHg in a patient with no risk factors for glaucoma and healthy optic nerves may be monitored conservatively, whereas a patient with pre-existing open angle glaucoma, documented visual field loss and a rise in IOP from 15 mmHg to 29 mmHg should be managed aggressively to reduce the IOP and prevent progressive optic nerve damage. If the IOP is very elevated, for example >30 mmHg, in the absence of glaucomatous damage, treatment is still needed because the eye may be at risk of developing a central retinal vein occlusion.

Stopping the steroids usually causes the IOP to fall back to baseline levels but the IOP can remain elevated in some patients, particularly those who have received steroid treatment for several months. If continued steroid use is deemed to be clinically necessary or the IOP remains elevated despite stopping the steroids, then the treatment options are essentially the same as for primary open angle glaucoma. Drug treatment is often initiated with topical beta-blockers or prostaglandin analogues. Contraindications to beta-blockers such as cardiovascular or bronchial disease should be excluded. Prostaglandin analogs are effective in patients with diabetic macular oedema and uveitis but may be associated with a risk of macular oedema and increased inflammation. Other topical hypotensive drops include alpha-agonists and carbonic anhydrase inhibitors. If topical treatment is inadequate, oral acetazolamide, laser trabeculoplasty or surgery may uncommonly be required.

**Conclusion**

All patients treated with steroids for eye disease are at risk of transient ocular hypertension or possibly steroid-induced glaucoma. Part of any management and treatment plan should include taking into consideration the risk factors for each individual patient, particularly if there is a history of glaucoma. Thereafter, patients require careful monitoring for signs of steroid induced ocular hypertension. Monitoring and intervention, if required, are the key factors to prevent steroid-induced ocular hypertension from developing into steroid-induced glaucoma.

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**Correction**

The article ‘Lessons to learn about pterygium’ published on pages 10 to 12 of the December 2012 issue of Optometry Pharma carries the subheading ‘Chemotherapy necessary but risky’. The subheading was incorrect. The author, Professor Lawrie Hirst notes that ‘use of chemotherapy for pterygium surgery is not only unnecessary but potentially sight-threatening’.

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