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H-C Lin[1,2], P-L Lu[3] and C-H Chang[1,4]

[1]Department of Ophthalmology, Kaohsiung Medical University Hospital, Kaohsiung, Taiwan
[2]Department of Ophthalmology, Yuan's General Hospital, Kaohsiung, Taiwan
[3]Department of Internal Medicine, Kaohsiung Medical University Hospital, Kaohsiung, Taiwan
[4]Department of Ophthalmology, Kaohsiung Municipal Hsiao-Kang Hospital, Kaohsiung, Taiwan
E-mail: ophchang@yahoo.com.tw

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Sir,

Intravitreal bevacizumab (Avastin) causing acute glaucoma: an unreported complication

We report a case where intravitreal bevacizumab, an antivascular endothelial growth factor (VEGF) agent caused an acute intraocular pressure (IOP) rise in an ocular hypertensive patient. This complication, to our knowledge, has not been reported before.

Case report

A 75-year-old Caucasian, with ocular hypertension and using guttae latanoprost and brinzolamide, was found to have classic juxtafoveal choroidal neovascularisation (CNV). After a written informed consent, the patient underwent intravitreal bevacizumab injections (1.75 mg/0.07 ml) at monthly intervals. Vision was checked immediately after each injection (at least counting fingers) to confirm ocular perfusion. Three days after the fourth injection, the patient developed corneal oedema with the IOP rising to 56 mmHg. He had a quiet anterior chamber and vitreous, and gonioscopy showed an open angle. The IOP was initially controlled on maximal medical treatment including oral acetazolamide. Acetazolamide was stopped after 3 weeks. Eleven weeks post-injection, the IOP is controlled on guttae latanoprost, cosopt (dorzolamide–timolol fixed combination), and apraclonidine.

Comment

Intravitreal bevacizumab, increasingly being used for CNV[1] and retinal vascular disorders,[2] is believed to be extremely safe with no association reported with glaucoma.

IOP elevation following an intravitreal injection can be explained by several theories. A temporary vitreous volume increase causes an IOP spike. Studies with pegaptanib, another anti-VEGF, have shown such a spike normalizes within one hour.[3]

Drug-induced trabeculitis is unlikely in the absence of anterior chamber inflammation which generally accompanies viral trabeculitis.[4]

A probable explanation is the blockage of the trabecular meshwork in an ocular hypertensive patient by bevacizumab, a 148 kDa full-length antibody. Mordenti *et al*[5] have shown the clearance of the high-molecular weight antibody from the vitreous is slow (half-life 5.6 days) with the internal limiting membrane acting as a barrier, and it also diffuses from the vitreous to the anterior chamber. Hence, the drug might have accumulated in the trabeculum increasing the aqueous outflow resistance causing the IOP to rise acutely. Now, this effect has lasted for three months; long after the drug is cleared from the vitreous.

In conclusion, bevacizumab should be used with caution in patients with glaucoma or ocular hypertension. Further studies are required to identify any predisposing risk factors in patients susceptible to developing acute glaucoma following intravitreal bevacizumab injection.

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A Jalil, C Fenerty and S Charles

Manchester Royal Eye Hospital, Manchester, Lancashire, UK
E-mail: assadjalil@hotmail.com

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