REFRACTORY OCULAR HYPERTENSION SECONDARY TO INTRAVITREAL INJECTION OF TRIAMCINOLONE ACETONIDE

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ABSTRACT

Purpose: to report a case of severe ocular hypertension occurring as a complication after a single intravitreal injection of triamcinolone acetonide for the treatment of a diabetic cystoid macular edema.

Methods: interventional case report.

Results: a 63-year-old pseudophakic diabetic woman developed a severe and relatively sudden IOP increase to 50 mm Hg one month after receiving a single 4-mg intravitreal injection of triamcinolone acetonide for a chronic progressive macular cystoid edema. Previously the patient who did not develop corticosteroid-induced glaucoma secondary to her cataract surgery was treated with topical beta-blockers for a mild chronic bilateral ocular hypertension. A deep sclerectomy had to be performed in emergency to avoid optic nerve damage and allowed to successfully control the IOP with a 5 month follow-up. Concomitantly visual acuity could be increased from 0.05 before the intravitreal injection to 0.4.

Conclusions: Although unfrequent in the literature, this observation confirms the risk of occurrence of a severe ocular hypertension after intravitreal injection of triamcinolone. A close monitoring of IOP is mandatory after intravitreal injection, especially in patients with altered trabecular function. This potentially devastating complication has to be weighed up with the benefices of intravitreal injection of triamcinolone for improving visual acuity in patients with clinically significant diabetic macular edema.

RÉSUMÉ

Objectif: le but de cette étude est de montrer la survenue possible d'une hypertension oculaire sévère dans le décours d'une injection unique d'acétate de triamcinolone administrée dans le cadre du traitement d'un œdème maculaire cystoide diabétique.

Méthodes: description d'un cas clinique.

Résultats: nous rapportons l'observation d'une patiente de 63 ans, diabétique, pseudophaque qui avait développé une hypertension oculaire importante et assez soudaine à 50 mm Hg et ce 1 mois après une injection unique d'acétonide de triamcinolone effectuée dans le cadre d'un œdème maculaire cystoide chronique évolutif. La patiente n'avait pas développé d'hypertension oculaire sur les corticoides locaux qu'elle avait reçus dans le décours de son intervention de cataracte et était traitée au préalable par un collyre bêta-bloquant pour une hypertension oculaire simple modérée. Une sclérectomie profonde a dû être réalisée en semi-urgence afin d'éviter une atrophie glaucomateuse de la papille optique et a permis de contrôler avec succès la tension oculaire avec un recul de 5 mois. Parallèlement, l'acuité visuelle centrale est passée de 0.05 avant l'injection à 0.4 au décours de celle-ci.

Conclusions: En dépit de son caractère relativement exceptionnel, cette observation confirme le risque de survenue d'une hypertension oculaire sévère après une injection intravitréenne de triamcinolone. Un suivi très rapproché de la PIO est indiqué après toute injection intravitréenne de triamcinolone, surtout chez les patients présentant une fonction trabéculaire altérée. Cette complication aux conséquences potentiellement graves doit être mise en balance avec les bénéfices de l'injection intravitréenne de triamcinolone chez des patients porteurs d'un œdème maculaire diabétique chronique réfractaire au traitement médical et au laser.

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KEY WORDS

Glaucoma, intraocular pressure, intravitreal triamcinolone, diabetic macular edema

MOTS-CLÉS

Glaucome, pression intra-oculaire, injection intravitréenne, triamcinolone, maculopathie exsudative diabétique.

During the past few years, crystalline cortisone in intravitreal injection has revealed to be a therapeutic possibility for numerous oedematous, proliferative and neovascular intraocular diseases. These include refractory diabetic cystoid macular edema, macular edema and choroidal neovascularization from age-related macular degeneration, long-standing pseudophakic cystoid macular edema or secondary to central retinal vein occlusion as well as chronic uveitis, proliferative diabetic retinopathy and proliferative vitreoretinopathy (2,6,7, 9-11,12,17-19,22-25,32,35,37,39,40,49). Intravitreal triamcinolone acetonide (TA) has also been suggested for treating ischemic ophthalmopathies and resistent cystoid macular edema secondary to birdshot retinochoroidopathy, serpiginous choroiditis, and idiopathic juxtafoveal telangiectasis (1,26,31,34). Finally, it has proved to be useful as angiostatic therapy in eyes with iris neovascularization and to be effective for treating pre-phthysical ocular hypotony (15,16, 21,41).

So far, endophthalmitis, cataract formation and ocular hypertension are the more significant reported ocular side effects attributed to this form of treatment (22,27,42,45,48).

We report herein the unusual clinical course of a patient who developed a severe and intractable ocular hypertension in the next few weeks following an intravitreal injection of 4 mg triamcinolone acetonide for a refractory progressive diffuse diabetic macular edema.

CASE REPORT

A 63-year old woman with a 10-year history of diabetes mellitus treated with insulin and oral metformine chlorhydrate (Glucophage®) was referred to us with a clinically significant macular edema caused by a non proliferative diabetic retinopathy.

She was also suffering from a mild asthma and an unsteady systemic hypertension despite multiple treatment. Moreover, she had been receiving betaxolol bid in both eyes for a mild ocular hypertension for 5 years. No case of glaucoma was known in her family.

During admission, her best corrected visual acuity was 0.6 in RE and 0.5 in LE, Parinaud 2 and 3 respectively in RE and LE. Anterior biomicroscopy revealed cortical lens opacities in both eyes. Applanation tonometry was 22 mm Hg in both eyes with betaxolol. On fundus examination, the appearance of the optic discs was in the normal limits. A diffuse exudative macular diabetic retinopathy was observed in both eyes, mostly in the right eye and confirmed in fluorescein angiography.

As cataract was impeding the completion of macular photocoagulations, an uncomplicated phakoemulsification was performed with implantation of a posterior chamber lens in both eyes with six months interval. During the first postoperative month, the IOP measurements remained within the reference range in both eyes despite administration of topical steroids.

Macular grid photocoagulations were completed 9 months after cataract surgery in both eyes.

Eighteen and 12 months respectively post cataract surgery on LE and RE, visual acuity was 0.2- in RE with -1.50 -1.00 60° and 0.4+ in LE with -0.50 -0.75 160°, Parinaud 8 and 4 with addition of +3.00 D. IOP measurements were 16 and 17 mm Hg in respectively RE and LE with betaxolol. A persistent diffuse macular edema was confirmed in ophthalmoscopy, fluorescein angiography and optical coherence tomography (OCT) in the right eye.

Therefore the patient was offered a transconjunctival intravitreal injection of 4 mg of crystalline triamcinolone acetonide (Kenacort®) in 0.1 ml of Ringer solution in her right eye.

During the following eight months, visual acuity rapidly improved from 0.1 to 0.3-0.4. On the meantime, fluorescein angiography showed a significant regression of the macular edema and of the hard exudates in the posterior pole. Central macular thickness measured by optical coherence tomography decreased by 34% two months after the injection but increased again moderately five months after the

injection. In ophthalmoscopy, cristals of cortisone had disappeared out of the vitreous cavity at 4-month follow-up.

However as early as one month after the injection, IOP gradually increased to reach 50 mm Hg at 3 month follow-up and then persisted at a level ranging between 30 and 40 mm Hg despite maximally tolerable antiglaucomatous treatment combining Cosopt® and acetazolamide 750 mg per day. In slit lamp examination, the anterior chamber was clear. In gonioscopy, the iridocorneal angle was open with a mild trabeculodysgenesis.

Diffuse non specific paracentral mild defects related to retinal photocoagulations and mild opacification of the posterior lens capsule were detected in automated perimetry. Due to the development of early glaucomatous change in the right optic nerve head and to the systemic side effects with acetazolamide, a deep sclerectomy was carried out at 4 month post-injection. Surgery was associated with a reticulated hyaluronic acid (SKgel®) implant and a subconjonctival injection of mitomycin C (0.2 mg/ml) at the end of the procedure. The peroperative and early postoperative course were uneventful. Five months after filtering surgery, a diffuse cystic bleb was present whereas IOP was 13 mm Hg without topical treatment in RE and 16 mm Hg with betaxolol bid in LE.

COMMENT

Steroids administered topically or systemically are well known to be associated with a rise of IOP in 18% to 36% of the general population but in 46 to 92% in patients with POAG (47). This has been shown to be related to the type and dose of steroid and to the route of administration.

Triamcinolone acetonide (TA) is a potent relatively insoluble synthetic glucocorticoid with similar mechanisms of action to those of naturally occurring glucocorticoids. It has been used in the treatment of ocular inflammation by peribulbar or sub-Tenon's injections for decades. Because of the highly variable intraocular levels of triamcinolone injection delivered by this route, the incidence of raised IOP in posterior sub-Tenon's triamcinolone injections in patients with posterior and intermediate uveitis is variable, ranging from 0% to 30% (13,29,33,44)

Based on experimental studies as well as on clinical and pathologic observations, Machemer, Peyman and other searchers found that direct intravitreal injection of a corticosteroid suspension of TA allows to deliver the drug to its target tissues in a more direct fashion. At the same time, it avoids most of extraocular side effects and has no toxicity to the intraocular tissue (3,8,30,46). In addition, the use of crystalline form of cortisone provides with intraocularly available cortisone for a considerably longer period than the single injection of soluble cortisone which is washed out of the eye within 24 hours after intraocular application (43). Owing to its potent antiphlogistic and antiangiogenic effects and its ability to reduce breakdown of the blood-retinal barrier, crystalline cortisone in intravitreal injection revealed to be a therapeutic possibility for treating various intraocular neovascular, edematous and proliferative diseases (22,36,46).

In most studies referring both to a single intravitreal injection of 4 mg or 25 mg of TA, the incidence of secondary ocular hypertension ranges from 3 to 83%.

This secondary ocular hypertension is known to be transient with a duration rarely exceeding 6 months and to be easily controlled with topical medications (Table 1). The risk of developing a severe ocular hypertension is considered to be much higher with more than one injection. As suggested by J.K.Challa, a second or a third injection should not be administered, at least within 4 months of the first injection (7). J.Jonas, who has a large expertise on this procedure and used higher dosages of 25 mg of TA, had mentioned this complication in about 50%, most often after about 1-2 months. He found that the rise of IOP was reversible about 6 months after the injection and usually controlled by topical antiglaucomatous medications, without development of a major damage to the optic nerve (23,27). Unlike all expectation, Jonas and associates did not find a significant relation between a secondary rise of IOP and a preoperative POAG, as well as the presence of a diabetes. He also concluded that the risk of developing an IOP rise after a second intravitreal TA injection appeared to be very low if the IOP had remained within normal limits

Table 1: *Steroid-induced secondary ocular hypertension after intravitreal TA injection: literature data.*

Authors	Indication	N eyes	TA dose	OHT incidence	Meds/surgery	Evolution
R.J. Antcliff 2001 (2)	uveitis	6	2 mg	16.6%	surgery	
J.K. Challa 1998 (7)	ARMD	30	4 mg	3%-75% (2d inject)	meds **surgery n=1**	transient 6 weeks
R.P. Danis 2000 (9)	ARMD	27	4mg	25%	meds	transient
B. Del Castillo 2001 (11)	uveitis	10	4 mg	20%	meds	transient
M.C. Gillies 2003 (12)	ARMD	151	4mg	41%	meds	transient
J.B. Jonas 2003 (23)	ARMD	71	25 mg	49.3%	meds **surgery n=1**	transient
J.B. Jonas 2003 (24)	CMO pseudo-phakic	5	25 mg	40%	meds	transient
J.B. Jonas 2003 (25)	diabetes	26	25 mg	34.6%	meds	transient
J.B. Jonas 2003 (27)	ARMD, diabetes	75	25 mg in 12/75 ×2	52% 1-2months	meds **surgery n=1**	transient 6 months
A.Martidis 2002 (32)	diabetic macular edema	16	4 mg	36%	meds macular edema	transient 3-6 months
C.H. Park 2003 (35)	CMO-CRVO	10	4 mg	30% previous POAG	meds **surgery n=1**	transient?
P.L. Penfold 1995 (37)	ARMD	30	4 mg	33%	meds	transient
J. M. Rakic 2003 (39)	CMO	15	4 mg	0%		
N.T. Ranson 2002 (40)	ARMD	14	4 mg	21%	meds?	transient
R.B.Wingate 1999 (48)	ARMD	113	4 mg	42%IOP rise: 32% 11%	2-4mm Hg ≥ 5mmHg > 10mmHg	?
S.Young 2001(49)	uveitis	6	4 mg	83.3%	meds	transient

after the first injection and that the risk of developing secondary ocular hypertension was comparable in both eyes in bilateral injections (27).

The mechanism of this IOP rise is not thoroughly understood but there is evidence of glucocorticoid induction of a gene promoting synthesis of a new protein/glycoprotein which is deposited in a typical manner of steroid-induced glaucoma and distinct from POAG (14,27,38,47).

As far as we know, only 4 cases of refractory ocular hypertension secondary to TA intravitreous injection and requiring surgery have been reported in the literature

Pharmakocinetic studies about intravitreal TA injection have shown that this route of administration allows to deliver a concentration of thousands of nanograms of triamcinolone to the vitreous cavity. However P.M. Beer and coll. have suggested that there could be a considerable variability both in the peak, the mean triamcinolone concentration and in the elimination half lives between subjects after a single injection of triamcinolone (5).

Therefore the expected duration of the positive treatment effect of TA may be variable from eye to eye. Measurable concentrations and persisting therapeutic effects are usually maintained in the vitreous for approximately 3-4 months in a non vitrectomized eye. However the persistence of even a trace amount up until 13 months in the vitreous cavity and until 9 months in the aqueous after the injection has been suggested to be related to the prolonged ocular hypertension occasionally seen in some patients.

In vitrectomized eyes, the elimination half life of TA being significantly reduced, the risk of secondary IOP can be expected to be lower (20,25,28,44).

Our patient developed a resistant steroid-induced secondary open angle glaucoma on the basis of a previous mild ocular hypertension which was well controlled with a single anti-glaucomatous medication. Her status in responding to corticosteroids was apparently negative and theoretically, it did not augur the occurrence of such a complication. For these reasons, the mechanism by which our patient has developed such an acute hypersensitivity to TA intravitreal injection is still questionable.

CONCLUSION

In support of our case report, we recommend that patients should be fully informed of the benefits, the most often transient effect and known side effects of intravitreal injections of steroids. Among these, the risk of developing acute resistant IOP rise appears to be higher in patients with a history of ocular hypertension or POAG

Large interindividual variations in intraocular TA pharmacokinetics, which probably lead to important variations in the IOP responsiveness to intravitreal TA injection, should be kept in mind.

Although our case report was apparently non steroid responder, we recommend to assess corticoresponsiveness in all candidates for this form of treatment by administering one drop 3 to 4 times daily of topical prednisolone acetate during at least one month before intravitreal TA injection (4). In any case, a close monitoring of IOP is mandatory after intravitreal injection during several months, especially in patients with altered trabecular function.

Eventually, if they are needed, repeated injections have to be spaced from at least minimum 4 months and IOP post re-injection monitoring has to be reinforced.

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