Intraocular injection of Depomedrone

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SUMMARY A case of inadvertent intraocular injection of Depomedrone (methylprednisolone acetate) and its management by vitrectomy is discussed, with a review of previously reported cases.

The administration of steroids by retrobulbar or orbital floor injection has been established as an effective¹ and safe² procedure. Accidental perforation of the globe and intraocular injection of a large bolus of steroids is an unusual but well documented complication and is known to carry a poor prognosis for vision.³ Here we report on the outcome and the effects on the retinal function of a patient who suffered an accidental intraocular injection of Depomedrone (methylprednisolone acetate).

Case report

CLINICAL COURSE

A 65-year-old female patient was referred to Moorfields Eye Hospital after an inadvertent injection of 0.7 ml of Depomedrone into the right vitreous cavity eight hours previously. She had been receiving regular retrobulbar injections of Depomedrone (methylprednisolone acetate) as a treatment for macular oedema. That day the injection was performed via the upper nasal fornix and was apparently uneventful. The patient did not experience any undue discomfort but did notice immediate blurring of vision. She returned to the referring ophthalmologist six hours later as the vision in the right eye had failed to improve during the course of the day.

The patient was known to have been a high myope and 10 years previously had undergone bilateral intracapsular cataract extractions. The last recorded visual acuity was 6/18 in the right eye and 6/60 in the left eye.

On admission to hospital the patient's right visual acuity was perception of light only. Vision in the left eye was reduced to the level of counting fingers due to severe myopic degeneration. The conjunctiva of the right eye was injected, but the cornea was clear.

There was a marked right afferent pupillary defect. The intraocular pressures were 0 mmHg in the right eye and 14 mmHg in the left. Large white deposits were present on the iris and in the anterior chamber angle. The vitreous was cloudy, with large white clumps throughout the anterior gel obscuring the view of the right retina.

The left eye was aphakic, and there was extensive myopic degeneration of the posterior pole.

Ultrasound examination of the right eye suggested a detached vitreous gel, and the retina was apparently attached. In view of the potential posterior

Fig. 1 The artist's impression of the operative findings at vitrectomy following the removal of the central portion of the opaque posterior hyaloid face. A discreet mass of the drug is layered over the posterior pole. Note the small deposit of the drug under the superonasal retinal vein, presumed site of ocular penetration. (The fibro-optic light probe and the infusion terminal have been omitted from the painting.)

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segment complications, pars plana vitrectomy was performed on the same night.

At operation Depomedrone was removed by suction from the anterior chamber and by vitrectomy from the posterior segment. The drug was found to be layered over the posterior pole and a small collection of subretinal Depomedrone was found in the superonasal quadrant (Fig. 1). The vitreous cavity was reconstituted with Hartmann solution and a subconjunctival injection of cephaloridine was given.

On the first postoperative day the eye was comfortable, but the visual acuity was hand movements only. There was stromal and epithelial oedema of the cornea, but this cleared during the following three days. The right relative afferent pupillary defect which was present at first diminished gradually, becoming equivocal by the third postoperative day.

The retina remained attached throughout the postoperative period, and at no time was there any clinical evidence of epiretinal membrane proliferation. Fluorescein angiography performed at the end of the first postoperative week and again one month later did not show leakage from retinal capillaries or macular oedema.

Over the next four weeks the visual acuity improved to 6/36, and it remained so during the subsequent 12 months.

ELECTRORETINOGRAPHY

In addition to the clinical follow-up serial electroretinography was performed during the immediate postoperative period and thereafter at two-monthly intervals. We used Arden's modified method as described elsewhere.⁴

The amplitudes of the b wave were reduced in each eye. This reduction was thought to be consistent with the bilateral myopic degeneration. With the bright stimulus the recording from the operated eye showed a rise in the voltage of the b wave from 80 μ v to 180 μ v in the first 10 days, and over the next five months

the voltage ranged from 175 μ v to 280 μ v. There was no significant change in the peak implicit time. The b wave amplitude of the fellow eye was 180 μ v immediately after the operation, and thereafter it remained in the range 200 μ v to 240 μ v.

The b wave amplitudes of the two eyes were compared, and the resulting ratio showed the recovery of the affected eye (Fig. 2).

Discussion

Periocular injection of methylprednisolone acetate has been used as a treatment of intraocular inflammation for over 20 years.⁵ The recognised complications of such injections include glaucoma, posterior subcapsular lens opacities, scarring of Tenon's capsule, paralysis of extraocular muscles, atrophy of subcutaneous tissues of the inferior orbital rim, and inadvertent intraocular administration.^{6,7}

To our knowledge only 12 other cases of inadvertent intraocular injection of methylprednisolone have been recorded so far.^{3,8-11} Most were managed conservatively and the majority suffered serious complications: five developed complex retinal detachment, one an extensive preretinal membrane, and another ascending optic atrophy. A recent report suggests vitrectomy as a useful alternative to conservative management,⁹ and the visual result in our case showed that early surgery can offer a better prognosis in this type of injury.

It was interesting to correlate the return of the visual function with the results of the serial electrophysiological tests. While we are aware that intraocular procedures such as vitrectomy may decrease electroretinographic responses, the profound depression of the wave form in our case was in excess of that usually observed after 'simple' vitrectomy without intraocular tamponade.¹² Rather, direct retinal toxicity of one or more components of the drug was more likely to have produced such marked electrophysiological changes.

Each ml of Depomedrone contains: methylprednisolone acetate 40 mg; polyethylene glycol 4000 29 mg; sodium chloride 8.7 mg; myristyl-gamma-picolinium chloride 0.19 mg; water for injection; and the pH is 5.6-6.5.

The dosage and toxicity of steroids for intraocular use in the treatment of intraocular infections and proliferative retinopathy is well documented.^{13,14} However, the suggested safe dose of triamcinalone for intraocular use is 20 times less than the equivalent dose of methylprednisolone injected into the eye of our patient.^{15,16}

Of the other constituents polyethylene glycol is a detergent and is known to be capable of producing arachnoiditis and pachymeningitis in humans.^{17,18}

Fig. 2 The ratio of the amplitude of the b wave of the injured eye was compared with that of the fellow eye. The ratios are presented as graded results over a period of eight months.

Myristyl-gamma-picolinium chloride is a germicidal as well as a detergent substance and is considered safe in concentrations up to 1 in 1000¹⁹. In Depomedrone its concentration is only 1 in 5000. The low pH of Depomedrone may have also contributed to the depression of retinal function.

Thus we consider that in cases such as ours the immediate removal of the drug by vitrectomy achieved not only an early visual rehabilitation but also minimised its long-term toxic effects on retinal function.

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