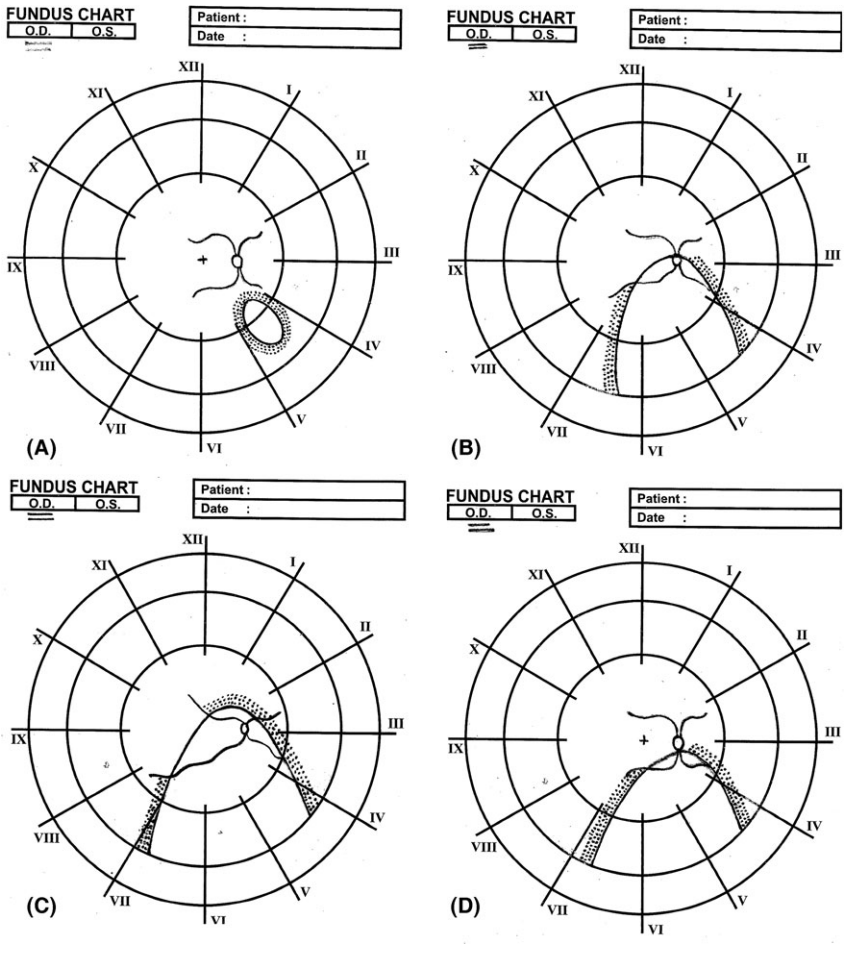

Fig. 1. (A) For isolated coloboma with normal surrounding retina outside arcade (Ida Mann type 5), the coloboma is entirely surrounded by three rows of the laser. (B) Cases in which the upper margin of coloboma is touching the superior border of the optic disc (Ida Mann type 2), laser spots are applied starting from nasal to the optic disc. Then nasal margin is lasered. Temporally laser is performed inferior to the presumed inferotemporal vascular arcade. (C) Cases in which the upper margin of coloboma is superior to the optic disc (Ida Mann type 1 fundal coloboma), laser spots are applied initially along the superior margin of coloboma and then continued along the whole nasal margin. Temporal margin is lasered inferior to the presumed inferotemporal vascular arcade and superiorly up to superotemporal arcade sparing the macula. (D) In type 3 coloboma, margins of coloboma are lasered sparing the area within the temporal vascular arcade and nasally up to 0.5 mm from the disc.

acuity during follow-up, which ranged from a minimum of 3 years to a maximum of 17 years (median 5 years). A similar benefit of PLP has been reported in a study in which RRD developed in 2.9% eyes which underwent PLP in contrast to 24.1% eyes that did not (Uhumwangho & Jalali 2014). Lack of a control group and selection bias due to the hospital setting are the major limitations of our study. Due to retrospective nature of the study, we cannot predict the incidence of RRD. We recommend PLP for FC as in our cohort of over 300 eyes this simple prophylactic procedure helped in maintaining vision with a very low rate of development of RRD. Further randomized prospective long-term studies may provide more evidence to support our recommendation.

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Correspondence:
Dr. Rohan Chawla
Department of Retina and Uvea, Dr. Rajendra Prasad Centre for Ophthalmic Sciences
All India Institute of Medical Sciences (AIIMS)
Room 488
New Delhi - 110029
India
Tel: +911126593188
Fax: +911126588919
Email: dr.rohanrpc@gmail.com

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Accidental intralenticular dexamethasone intravitreal implant with the resolution of macular oedema in central retinal vein occlusion

Solmaz Abdolrahimzadeh,[1] Pasquale Plateroti,[2] Fabio Scarinci[3] and Andrea Maria Plateroti[1]

[1]Ophthalmology Unit, Department of Sense Organs, Umberto I Polyclinic, University of Rome 'Sapienza', Rome, Italy; [2]Ophthalmology Unit, St. Andrea Hospital, NESMOS Department, University of Rome 'Sapienza', Rome, Italy; [3]G.B. Bietti Eye Foundation for Study and Research in Ophthalmology, Rome, Italy

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Editor,

The dexamethasone intravitreal implant Ozurdex (Allergan, Inc Irvine, CA) is composed of a matrix of dexamethasone and a biodegradable copolymer of lactic and glycolic acids

(Lambiase et al. 2014). The implant allows the release of 700 g of dexamethasone, providing peak doses at 2 months of injection with a slower release for up to 6 months. It is currently used in macular oedema following branch or central retinal vein occlusion (CRVO), non-infectious posterior uveitis, diabetic macular oedema and psuedofakic macular oedema (Lambiase et al. 2014). The implant is injected 3.5 mm to 4 mm posterior to the limbus into the mid-vitreous with a muzzle velocity of 0.8 m/s (Meyer et al. 2012). Among the complications of intravitreal injection due to touch or penetration of adjacent anatomic structures with the needle tip are retinal detachment in 0.013% and lens damage in 0.009% of cases. (Meyer et al. 2010, 2011).

A 62-year-old woman with a 2-week history of CRVO in the left eye and accidental injection of an intravitreal dexamethasone implant into the lens 2 days previously in a private practice presented to our observation. Best-corrected visual acuity (BCVA) was 20/40. Slit lamp examination showed a circumscribed area of cataract formation around the point of penetration of the implant in the posterior capsular area of the lens (Fig. 1A). Fundus examination showed CRVO with cystoid macular oedema. Surgical removal of the crystalline lens and implant was advised, but the patient refused surgery. At 1 month of follow-up, BCVA was 20/20 although the patient complained of disturbed vision due to the position of the implant in the visual axis (Fig. 1B). At 8 months of follow-up, the circumscribed lens opacity had not expanded and there were no ophthalmoscopic signs of CRVO or optical coherence tomography (OCT) evidence of cystoid macular oedema. Anterior segment imaging with rotating Scheimpflug camera combined with a Placido disc system (Sirius, CSO Italy) showed the dexamethasone implant impaling the posterior lens capsule for approximately one-fifth of its length with the rest protruding in the vitreous chamber (Fig. 2). The study was conducted in accordance with the Tenets of the Declaration of Helsinki, and the patient provided consent to the publication of the present case.

In cases of accidental injection of the dexamethasone implant in the crystalline lens, cataract formation

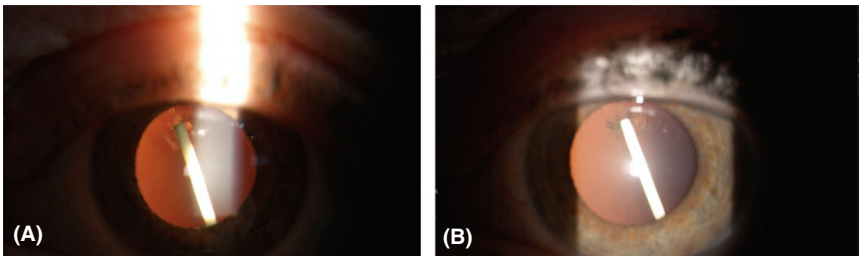

Fig. 1. Slit lamp photographic image of the dexamethasone implant in the lens 2 days (A) and 8 months after accidental injection (B).

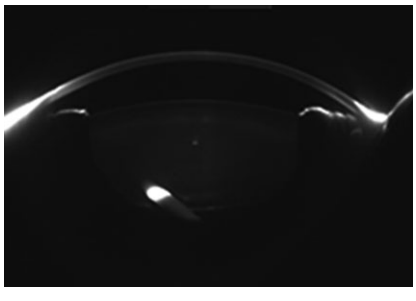

Fig. 2. Anterior segment Pentacam image showing penetration of the dexamethasone implant in the posterior capsular area of the lens.

followed by removal of the lens has been reported (Coca- Robinot et al. 2014). Inefficacy of treatment in these cases has been attributed to the insufficient release of the drug due to the different biochemical properties of the lens with respect to the vitreous (Coca-Robinot et al. 2014). In the case reported herein, macular oedema resolved, presumably because the dexamethasone implant was only partially inside the lens and protruded into the vitreous cavity providing sufficient therapeutic levels of dexamethasone. Furthermore, the intralenticular position of the lens could have given an additional slow release of dexamethasone as there were no ophthalmoscopic signs of CRVO or OCT evidence of cystoid macular oedema at 8 months of follow-up, which is beyond the reported 6-month release profile of dexamethasone in the vitreous cavity (Lambiase et al. 2014). Cataract progression was not observed over the entire follow-up period. This could have been because lens wounds of small size can heal spontaneously due to the proliferation of subcapsular epithelium which seals the wound before the intralenticular passage of ions and fluid, which is the cause of cataract progression (Fagerholm & Philipson 1979).

To our knowledge, this is the first report in the literature of the intralenticular retention of a dexamethasone implant with resolution of macular oedema and no progression of cataract over 8 months of follow-up.

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Correspondence:
Andrea Maria Plateroti
Ophthalmology Unit
Department of Sense Organs
Azienda Policlinico Umberto I
University of Rome 'Sapienza'
viale del Policlinico 155, Rome 00161
Italy
Tel: +3906 49975348
Fax: +3906 49975304
Email:andrea.plateroti@gmail.com