Descemet’s Stripping-automated Endothelial Keratoplasty for symptomatic thioridazine deposits in the cornea

Masoumeh Mohebbi, Hassan Hashemi, Alireza Mahmoudi, Pasha Anvari

Abstract:
The aim of this study was to use Descemet’s Stripping-automated Endothelial Keratoplasty (DSEA) as a novel treatment to remove thioridazine corneal deposits. A 53-year-old female presented with a 3-month history of visual loss and glare. She had been taking thioridazine (100 mg/day for 1 year) for a psychiatric disorder. Dense yellowish-brown deposits had developed in the posterior stroma. Thioridazine was discontinued, and she was switched to fluoxetine. One year after discontinuation of thioridazine, her symptoms and signs did not resolve. Standard DSEA was performed on her left eye. Two weeks after DSEA, an anterior subcapsular cataract was detected in the same eye. Phacoemulsification and intraocular lens implantation were performed after 3 months. The left cornea became completely clear after DSEA, and the patient’s best-corrected visual acuity improved from 20/40 to 20/20 at 1 month after cataract surgery. All-visual symptoms such as glare and halos improved postoperatively. We suggest that DSEA can be used as a novel treatment to reduce vision problems caused by thioridazine-induced corneal deposits.

Keywords:
Corneal deposits, descemet’s Stripping-automated Endothelial Keratoplasty, thioridazine

Case Report
A 53-year-old female with a 3-month history of visual loss, glare, and halos was referred to our clinic. The patient’s medical history included a psychiatric disorder with symptoms of myalgia, restlessness, and anxiety, for which she had been taking thioridazine (100 mg/day) for the past year. She had no history of other medications, and her past and family histories were noncontributory. Her best-corrected visual acuity (BCVA) was 20/40 OU. A quality of vision (QoV) questionnaire score, completed by the patient, showed that vision impairment had considerably impacted her QoV.

A slit-lamp examination demonstrated yellowish-brown deposits in the cornea. A 3-month history of visual loss and glare was noted. The patient’s symptoms did not improve with discontinuation of thioridazine. Standard Descemet’s Stripping-automated Endothelial Keratoplasty (DSEA) was performed on her left eye. Two weeks after DSEA, a subcapsular cataract was detected. Phacoemulsification and intraocular lens implantation were performed after 3 months. The left cornea became completely clear after DSEA, and the patient’s best-corrected visual acuity improved from 20/40 to 20/20 at 1 month after cataract surgery. All-visual symptoms such as glare and halos improved postoperatively. We suggest that DSEA can be used as a novel treatment to reduce vision problems caused by thioridazine-induced corneal deposits.

Keywords:
Corneal deposits, descemet’s Stripping-automated Endothelial Keratoplasty, thioridazine

How to cite this article: Mohebbi M, Hashemi H, Mahmoudi A, Anvari P. Descemet’s stripping-automated endothelial keratoplasty for symptomatic thioridazine deposits in the cornea. Taiwan J Ophthalmol 2017;7:53-5.
deepest stromal layers and endothelium in both eyes [Figure 1a-c]. Anterior segment optical coherence tomography (AS-OCT, Visante OCT; Carl Zeiss Meditec, Dublin, CA, USA) confirmed that the depth of the drug deposits was confined to the deep-stromal layers [Figure 2a].

There was no significant cataract present in either eye. Her intraocular pressures were 14 mmHg OD and 16 mmHg OS and were measured using a Goldmann applanation tonometer. Shallow anterior chambers were observed in both eyes (2.3 mm based on Pentacam; Oculus Optikgerate GmbH, Wetzlar, Germany). The patient’s dilated fundus examination was normal. No extrapyramidal side effects were observed or reported.

After consulting a psychiatrist, thioridazine was discontinued and substituted with fluoxetine. At 1-year follow-up, the patient’s symptoms and signs had not yet been resolved.

DSEA K was performed on the left eye. Post operative AS-OCT indicated a well-adhered graft [Figure 2b]. Despite shallow anterior chamber, no elevated intraocular pressure was noted postoperatively. Two weeks after DSEA K, a significant anterior subcapsular cataract was observed in the same eye and vision decreased to 20/200 [Figure 3a]. Phacoemulsification and intraocular lens implantation were performed 3 months later. Pupilloplasty was performed to repair the temporal iris atrophy and corectopia secondary to manipulation of the iris from previous surgery.

In the early postoperative period, a negligible amount of the drug deposit was found in the deep stromal layer of the cornea (DSEA K interface) that disappeared after 1 month. The left cornea had become completely clear 6 months postoperatively [Figure 3b]. BCVA of the patient’s left eye improved to 20/20 at 1 month after phacoemulsification, and the ocular symptoms of glare, halos, and blurred vision considerably improved when the right eye was closed. The QoV questionnaire score of 17 showed a significant improvement in the patient’s QoV. At the final visit, 18 months after initial encounter, corneal deposits and vision in the untreated eye were not different from the first visit. The patient later asked to have her right eye operated on.

**Discussion**

Thioridazine, an antipsychotic phenothiazine derivative, has been extensively used at high doses in the past. Concerns regarding thioridazine-induced cardiotoxicity prompted the restriction of its prescription to the second-line therapy. Furthermore, thioridazine has been associated with ocular toxicity, particularly retinopathy and corneal changes. Although the exact mechanism remains to be completely elucidated, the binding of thioridazine to melanin may explain the corneal and lenticular deposits, which are most likely time and dose dependent.

Our patient had been treated with a dosage of 100 mg/day thioridazine for only 1 year. Discontinuing thioridazine and switching to another drug did not reverse the patient’s symptoms. One factor that may have accelerated the rapid progression of corneal haziness may have been the patient’s occupation as an agricultural worker and the excessive exposure to sunlight. Although the mechanism of phototoxicity is not well understood, studies have shown that cytotoxicity could result from light-induced drug...
decomposition.[6] The majority of the deposits were scraped off during the DSAEK procedure. The negligible residues that disappeared postoperatively might have been eliminated by phagocytic activity of the grafted endothelium and corneal keratocytes.

Cataract following DSAEK is not uncommon.[7] In phakic endothelial keratoplasty, a combination of factors such as surgical manipulation, air tamponade at the time of surgery, and use of postoperative steroid medications leads to cataract formation.[7]

We used DSAEK as a novel treatment to remove drug-induced deposits from the deep corneal stroma and endothelium. Based on our case report, DSAEK shows promise as an effective method to reduce vision problems caused by corneal deposits from thioridazine administration.

Financial support and sponsorship
Nil.

Conflicts of interest
The authors have no conflicts of interest to declare.

References
1. Shah GK, Auerbach DB, Augsburger JJ, Savino PJ. Acute thioridazine retinopathy. Arch Ophthalmol 1998;116:826-7.
2. Davidorf FH. Thioridazine pigmentary retinopathy. Arch Ophthalmol 1973;90:251-5.
3. Rasmussen K, Kirk L, Faurbye A. Deposits in the lens and cornea of the eye during long-term chlorpromazine medication. Acta Psychiatr Scand 1976;53:1-6.
4. McAlinden C, Pesudovs K, Moore JE. The development of an instrument to measure quality of vision: The Quality of Vision (QoV) questionnaire. Invest Ophthalmol Vis Sci 2010;51:5537-45.
5. Buszman E, Rózanska R. Interaction of thioridazine with ocular melanin in vitro. Acta Pol Pharm 2003;60:257-61.
6. Hull DS, Csukas S, Green K. Chlorpromazine-induced corneal endothelial phototoxicity. Invest Ophthalmol Vis Sci 1982;22:502-8.
7. Chaurasia S, Ramappa M, Sangwan V. Cataract surgery after descemets stripping endothelial keratoplasty. Indian J Ophthalmol 2012;60:572-4.