

Access this article online

Quick Response Code:



Website:

www.e-tjo.org

DOI:

10.4103/tjo.tjo_50_20

Bilateral corneal edema in an alcoholic male

Peng-Yu Lee¹, Wei-Yi Chou², Chih-Chien Hsu^{2,3,4*}, Pei-Yu Lin^{2,3}, Ko-Hua Chen^{2,3}

Abstract:

Pseudophakic bullous keratopathy and Fuchs' endothelial dystrophy are the two most common causes of corneal edema after cataract surgery. We report a 61-year-old alcoholic male with bilateral corneal edema that improved after his alcohol abstinence. He had uneventful bilateral cataract surgery 3 years ago and blurred vision in both eyes developed for weeks. As he had no history of endothelial dystrophy, the treatment for viral endotheliitis was used initially yet in vain. We asked him to stop alcohol and adjusted his psychiatric drugs, but he lied about stopping drinking. The corneal edema progressed, and finally, he underwent penetrating keratoplasty in his left eye 1 year later. During hospitalization for surgery, alcohol withdrawal syndrome was noted because he could not drink alcohol in our hospital. After he quit drinking for months, corneal edema in the right eye disappeared. Our case highlights that alcoholism can result in corneal edema, and stopping drinking is necessary in these patients.

Keywords:

Alcohol, cataract surgery, corneal edema, endothelial cell

Introduction

The corneal endothelium plays a significant role in regulating hydration of the corneal stroma and maintaining its transparency.^[1,2] Patients with corneal edema can have pain and vision impairment due to fluid accumulation arising from endothelial disorders, inflammation, ocular surgery, trauma, or toxins.^[3-5] Long-term alcohol consumption has been linked to various ophthalmic consequences. Some studies proposed that the amount of drinking and the nutritional status might contribute to eye diseases.^[6-9] However, corneal edema and endothelial changes related to ethanol intake are less commonly investigated. Herein, we report a case of alcohol-associated corneal edema, which improved after alcohol abstinence in Taiwan.

Case Report

A 61-year-old male patient complained of sudden blurred vision in both eyes

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: WKHLRPMedknow_reprints@wolterskluwer.com

for weeks. His medical history included diabetes mellitus, hypertension, hyperlipidemia, anxiety, depression, and alcoholism. He took sitagliptin, glimepiride, empagliflozin, amlodipine, fenofibrate, pregabalin, venlafaxine, and lorazepam to control his underlying problems. As for ocular history, he underwent bilateral cataract surgery without complications 3 years ago. On examination, the best-corrected visual acuity (BCVA) was 6/60 and 6/20 in the right and left eye, respectively. Intraocular pressure was 8 mm Hg in both eyes. Slit-lamp examination revealed bilateral corneal edema with striations. There were no significant keratic precipitates (KPs) on the endothelium despite the edematous stroma [Figure 1]. Anterior segment optical coherence tomography showed increased corneal folds and central corneal thickness (CCT) was 737 and 688 μ m. Endothelial cell count (ECC) was not measurable due to corneal swelling. Topical antiviral drugs, topical steroids, and 5% NaCl eye drops were thus prescribed because herpetic

How to cite this article: Lee PY, Chou WY, Hsu CC, Lin PY, Chen KH. Bilateral corneal edema in an alcoholic male. Taiwan J Ophthalmol 2021;11:175-8.

¹Department of Medical Education, Taipei Veterans General Hospital, Taipei, ²Department of Ophthalmology, Taipei Veterans General Hospital, Taipei, ³Faculty of Medicine, National Yang-Ming University School of Medicine, Taipei, ⁴Institute of Clinical Medicine, National Yang-Ming University, Taipei, Taiwan

*Address for correspondence:

Dr. Chih-Chien Hsu,
No. 201, Sec. 2, Shipai Road, Beitou District, Taipei City 11217, Taiwan.
E-mail: chihchienym@gmail.com

Submission: 02-05-2020

Accepted: 03-07-2020

Published: 07-10-2020

endotheliitis was suspected initially. However, we did not see any KPs on the endothelium, and we discontinued topical antiviral drugs weeks later. His corneal edema improved but did not totally subside 2½ months later with topical steroid and 5% NaCl drops [Supplementary Figure 1]. We asked him to stop drinking alcohol and to adjust his psychiatric drugs because alcoholism-induced and psychiatric drug-related corneal edema had been documented. He claimed to have reduced alcohol ingestion; however, his corneal edema still progressed, especially in the left eye, after treatment for 6 months [Supplementary Figure 2]. ECC at central cornea was not assessable but at paracentral cornea was 1042 and 1014 cells/mm² in the right and left eye, respectively. Penetrating keratoplasty (PK) in the left eye was eventually indicated 1 year later since BCVA dropped to 6/30 in the left eye. During hospitalization for PK, hallucination and hand tremors were noted, and the psychiatrist suspected alcohol withdrawal syndrome. At that point, we realized that he had never stopped drinking until there was no access to alcohol in our hospital. Treatment for the withdrawal syndrome started and continued after discharge. Two weeks after his operation in the left eye, his corneal edema still persisted in the right eye [Figure 2a and b]. However, after 2 months of alcohol cessation, his corneal edema in the right eye disappeared. Figure 2c and d showed the corneal condition in the right eye after 1 year of alcohol cessation. His BCVA advanced to 6/12 and 6/15, CCT was 469 µm and 518 µm, ECC was 1412 and 2695 cells/mm², coefficient of variation was 60% and 42%, and hexagonality was 47% and 43% in the right and left eye, respectively.

Discussion

This case highlights a rare presentation of bilateral corneal endothelial dysfunction and extensive cell loss due to chronic alcohol use. Shiono *et al.* and

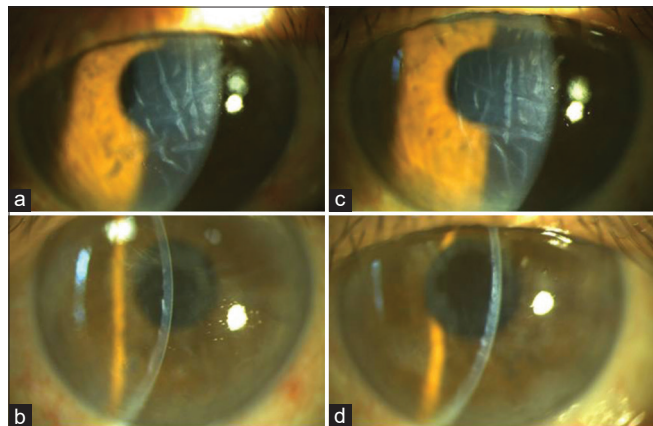


Figure 1: Severe corneal edema with striation in both eyes (a and b: Right eye; c and d: Left eye)

Ranjan *et al.* ever reported the similar corneal changes after acute alcohol intake and concluded that alcohol could induce temporary endothelial deterioration and cell damage.^[10,11] Another study by Olsen and Olsen noted a statistically significant increase in corneal thickness following ethanol ingestion.^[12] Sati *et al.* further evaluated endothelial alterations in patients with alcohol dependence syndrome (ADS). Significant differences in terms of CCT and ECC were seen between ADS patients and control group, as well as before and after abstinence in ADS group.^[13] Our patient had been drinking six large cans of beer per day, approximately 150 g of ethanol, for 30 years. According to Diagnostic and Statistical Manual of Mental Disorder-V, he met the criteria for alcohol use disorder and alcohol withdrawal syndrome. His liver enzymes remained within the normal range, whereas Stage I fatty liver was evaluated by sonography. Our case report corresponded to Sati *et al.* and supported their hypothesis for endothelial alterations in patients with chronic alcohol use, particularly dependence.

The exact influences of alcohol on cornea are still not fully understood and likely to be multifactorial. Pleomorphism with cell loss, toxic endotheliitis, and depression of endothelial pump activity were hypothesized.^[10,11] *In vitro* experiments by Grütters *et al.* observed a drop in ECC along with an increase in protein degradation in cornea stored in acetaldehyde, a metabolite of ethanol.^[14] In addition, Honey *et al.* found a good correlation between vitreous alcohol concentration and blood alcohol concentration in postmortem examinations.^[15] Hence, a higher alcohol concentration in aqueous humor leading to endothelial insults is a reasonable possibility.

In alcoholic patients, malnutrition, subsequent hypoglycemia as well as hypovitaminosis may also

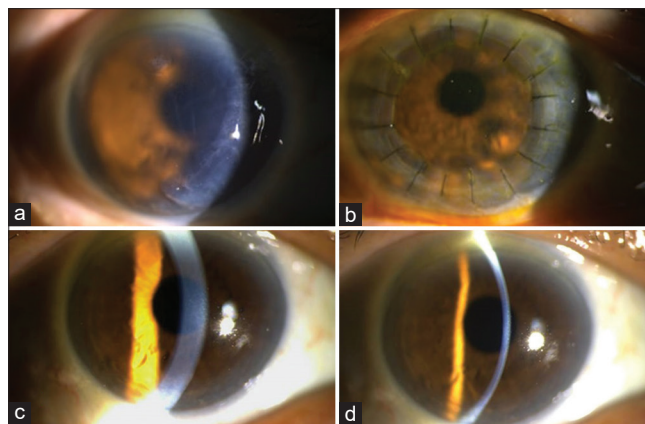


Figure 2: (a) Corneal edema with bullae in the right eye after initial treatment for 1 year. (b) Penetrating keratoplasty was done due to more severe edematous cornea in the left eye. (c and d) Corneal edema in the right eye disappeared after stopping drinking for 1 year

give rise to endothelial changes in cornea. Reduced glucose level in aqueous humor can lead to insufficient nutrition support to endothelium.^[16] On the other hand, vitamins serve as anti-oxidants in cornea and prevent apoptosis.^[17,18] Decreased vitamin level may thereby result in endothelial cell loss.

In this case, we also considered a diagnosis of viral infection or postoperative keratopathy. Anti-viral agents were given at first but with little response. Delayed-onset corneal edema from surgical trauma is possible, though it seldom happens.^[19,20] In contrast, he underwent smooth cataract surgery 3 years ago, and postoperative follow-ups did not detect any abnormality. Another differential diagnosis was ocular manifestations of systemic drugs. Several pharmacological treatments, such as amantadine and chlorpromazine, were previously recorded as the reasons for corneal edema.^[21-23] Furthermore, psychotropic agents can cause ocular complications.^[24] Interactions between alcohol and medications also concerned us.^[25] Therefore, we reviewed his prescription but found no direct relationship between his drugs and corneal haziness. We still consulted the psychiatrist to simplify his medicine in the hope of minimizing drug-drug and drug-alcohol interactions. Considering the temporal sequence of prominent resolution and clinical improvement after quitting alcohol, ethanol-induced corneal edema was the most likely etiology.

In conclusion, we present an alcoholic man with acute corneal edema, who gradually recovered after he stopped drinking. We believed transient suppression of corneal endothelial cells instead of total apoptosis occurred at first, but ultimately endothelial cells died. Although corneal endothelial cells could not regenerate, the remaining cells later regained functions following alcohol cessation. Nevertheless, cell loss and recurrence of corneal edema are highly expected in future. The toxic effects of ethanol should be attributed to multiple mechanisms. Moreover, psychotropic medications should be taken into account as these drugs are widely used in such patients.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

Acknowledgment

The authors would like to thank technicians at the Department of Ophthalmology, Taipei Veterans General

Hospital, for supporting the ophthalmic examinations in each patient.

Financial support and sponsorship

Nil.

Conflicts of interest

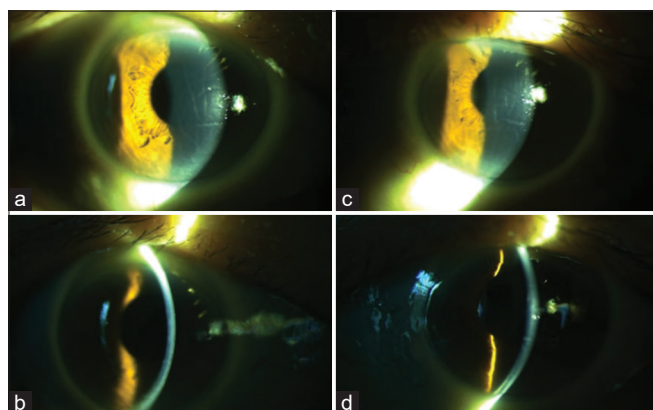
The authors declare that there are no conflicts of interests of this paper.

References

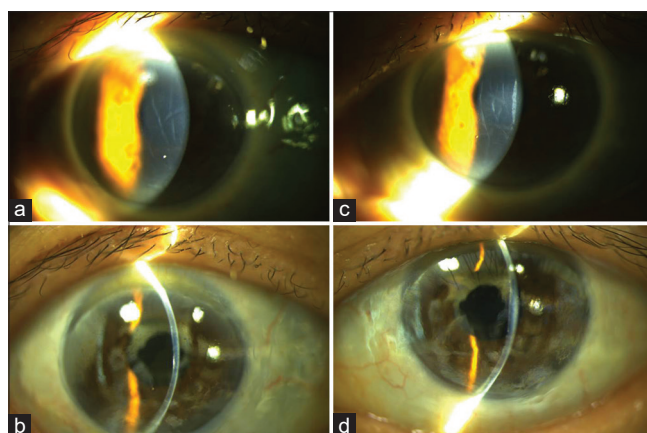
1. Tuft SJ, Coster DJ. The corneal endothelium. *Eye (Lond)* 1990;4(Pt 3):389-424.
2. Bourne WM. Biology of the corneal endothelium in health and disease. *Eye (Lond)* 2003;17:912-8.
3. Costagliola C, Romano V, Forbice E, Angi M, Pascotto A, Boccia T, *et al.* Corneal oedema and its medical treatment. *Clin Exp Optom* 2013;96:529-35.
4. Feizi S. Corneal endothelial cell dysfunction: Etiologies and management. *Ther Adv Ophthalmol* 2018;10:2515841418815802.
5. Moshirfar M, Murri MS, Shah TJ, Skanchy DF, Tuckfield JQ, Ronquillo YC, *et al.* A Review of corneal endotheliitis and endotheliopathy: Differential diagnosis, evaluation, and treatment. *Ophthalmol Ther* 2019;8:195-213.
6. Wang S, Wang JJ, Wong TY. Alcohol and eye diseases. *Surv Ophthalmol* 2008;53:512-25.
7. Moss SE, Klein R, Klein BE. Prevalence of and risk factors for dry eye syndrome. *Arch Ophthalmol* 2000;118:1264-8.
8. Castro AJ, Rodrigues AR, Côrtes MI, Silveira LC. Impairment of color spatial vision in chronic alcoholism measured by psychophysical methods. *Psychol Neurosci* 2009;2:179-87.
9. Sharma P, Sharma R. Toxic optic neuropathy. *Indian J Ophthalmol* 2011;59:137-41.
10. Shiono T, Asano Y, Hashimoto T, Mizuno K. Temporary corneal oedema after acute intake of alcohol. *Br J Ophthalmol* 1987;71:462-5.
11. Ranjan A, Murthy SI, Rathi VM, Sangwan VS. Acute bilateral toxic endotheliitis following alcohol consumption. *Ocul Immunol Inflamm* 2018;26:269-72.
12. Olsen EG, Olsen H. Influence of ethanol ingestion on the cornea. *Acta Ophthalmol (Copenh)* 1993;71:696-8.
13. Sati A, Moulick P, Shankar S, Chatterjee K, Dwivedi AK, Vazirani J. Corneal endothelial alterations in alcohol dependence syndrome. *Br J Ophthalmol* 2018;102:1443-7.
14. Grütters G, Ritz-Timme S, Reichelt JA, Nölle B. Alcohol-induced morphologic and biochemical corneal changes. *Ophthalmologe* 2002;99:266-9.
15. Honey D, Caylor C, Luthi R, Kerrigan S. Comparative alcohol concentrations in blood and vitreous fluid with illustrative case studies. *J Anal Toxicol* 2005;29:365-9.
16. Reim M, Lax F, Lichte H, Turss R. Steady state levels of glucose in the different layers of the cornea, aqueous humor, blood and tears in vivo. *Ophthalmologica* 1967;154:39-50.
17. Serbecic N, Beutelspacher SC. Vitamins inhibit oxidant-induced apoptosis of corneal endothelial cells. *Jpn J Ophthalmol* 2005;49:355-62.
18. Chen Y, Mehta G, Vasiliou V. Antioxidant defenses in the ocular surface. *Ocul Surf* 2009;7:176-85.
19. Sharma N, Singhal D, Nair SP, Sahay P, Sreeshankar SS, Maharana PK. Corneal edema after phacoemulsification. *Indian J Ophthalmol* 2017;65:1381-9.
20. Claesson M, Armitage WJ, Stenevi U. Corneal oedema after cataract surgery: Predisposing factors and corneal graft outcome. *Acta Ophthalmol* 2009;87:154-9.

21. Hull DS, Csukas S, Green K. Chlorpromazine-induced corneal endothelial phototoxicity. *Invest Ophthalmol Vis Sci* 1982;22:502-8.
22. Jeng BH, Galor A, Lee MS, Meisler DM, Hollyfield JG, Schoenfield L, *et al.* Amantadine-associated corneal edema potentially irreversible even after cessation of the medication. *Ophthalmology* 2008;115:1540-4.
23. Raizman MB, Hamrah P, Holland EJ, Kim T, Mah FS, Rapuano CJ, *et al.* Drug-induced corneal epithelial changes. *Surv Ophthalmol* 2017;62:286-301.
24. Richa S, Yazbek JC. Ocular adverse effects of common psychotropic agents: A review. *CNS Drugs* 2010;24:501-26.
25. Tanaka E. Toxicological interactions involving psychiatric drugs and alcohol: An update. *J Clin Pharm Ther* 2003;28:81-95.

Supplementary Figure



Supplementary Figure 1: Corneal edema with striation improved in both eyes after treatment for 2½ months (a and b: Right eye; c and d: Left eye)



Supplementary Figure 2: Corneal edema with striation progressed again, especially in the Left eye, despite treatment (a and b: Right eye; c and d: Left eye)