Review Article

Glaucoma in Iridocorneal Endothelial Syndrome: A Brief Review

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Abstract

The iridocorneal endothelial (ICE) syndrome is a sporadic corneal endothelial disease that causes corneal endothelial cell abnormality as epithelialization, resulting in intractable glaucoma and corneal decompensation, often needing glaucoma surgery and corneal transplantation without a good prognosis. The most effective way to diagnose early is in-vivo confocal microscopy. Early treatment, especially for resultant glaucoma, improves diagnosis.

Keywords: Glaucoma; Iridocorneal Endothelial Syndrome; Intraocular Pressure.

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Introduction

The iridocorneal endothelial (ICE) syndrome is typically unilateral [1] (in more than 90 % of cases), [2] non-familial, progressive spectrum of proliferative abnormal corneal endothelial cells present in the iris stroma and anterior chamber angle [1, 3-5].

This spectrum includes essential iris atrophy as predominant iris stromal atrophy causing corectopia, polycoria, and peripheral anterior synechiae, Chandler syndrome with severe corneal endothelial involvement causing a "hammered-silver" or "beaten-bronze" appearance [6, 7] and corneal edema, with the least amount of iris abnormality, and iris nevus / Cogan-Reese syndrome causing iris nevus or nodules [1, 4, 8-13]. Chandler syndrome is the most common variant in Whites, but the Cogan-Reese syndrome is more frequent in Asians [1, 7, 14].

It typically manifests in white young or middle-aged adults in the third to fifth decade of their lives [1, 15, 16], with a female predilection [10, 17- 22], but there are some reports of this abnormality in childhood age [23-27]. The major complications of ICE syndrome are glaucoma and corneal edema. Glaucoma arises from changed endothelial cells. Epithelialization of them leads to secondary open and angle closure glaucoma with proliferation and migration of endothelial cells over the angle, resulting in peripheral anterior synechiae. The corneal edema in ICE syndrome is secondary to elevated intraocular pressure (IOP) and subnormal pump function of diseased corneal endothelial cells [28].

Pathophysiology

The major pathology is in diseased corneal endothelial cells called ICE-cells with a tendency to divide, mitosis, and develop into multiple endothelial layers [29-32]; sometimes, there are called subtotal ICE, so diseased and normal endothelial cells are adjacent to each other in a transmission zone called a boundary zone with non-motile but high metabolic activity [31]. Similar to ex-vivo, in-situ characteristics are mentioned in multiple studies [16, 31, 33-35]. Proliferation, contraction, and migration of diseased endothelial cells across the angle and on the iris. This results in corneal decompensation, collapsed trabecular beams, decreased intertrabecular spaces, and synechiae formation resulting in secondary open angle or angle closure glaucoma [6, 7, 20].

Another change in the corneal endothelial cells is contraction causing iris atrophy, thinning of iris stroma, and full-thickness holes' formation are named stretch holes and ectropion uvea, polycoria, and corectopia [12, 22, 23, 29, 36].

The diseased endothelial cells acquire an epithelial-like pattern prominently on the apical surface, owning desmosomes, filopodia and microvilli, vimentin, blebs, and cytokeratin [37-39].

Its etiology is unclear [1, 3-7], but some evidence supports the viral origin, such as the existence of inflammatory cells on histological evaluation [40]. Herpes simplex virus or Epstein Barr virus is proposed as the primary etiology of this disease [11, 36, 41-43] by the integration of viral genetic material into the human genome [3, 37], resulting in necrotic events [33].

There are some reports of accompanying other ocular diseases such as microspherophakia [11] and keratoconus [37, 39, 44-47], macular edema [48-50], retinal neurosensory detachment [47], and also systemic diseases such as Downs syndrome [23].

Diagnosis

Patients typically perceive predominantly morning visual haze, leading to decreased visual acuity and halos due to corneal edema. Some patients prefer changes in the shape or

position of the pupils [51].

Subclinical corneal abnormalities are not uncommon in the fellow eye [18, 19] and bilateral clinical ICE or are reported [23, 52, 53]. Specular or in-vivo corneal confocal microscopy is useful for diagnosis confirmation [7]. Specular microscopy shows ICE-cells that are epithelial-like endothelial cells as large, rounded, and pleomorphic cells with polymegathism and also a typical "light-dark reverse" as a dark surface with light spots at the central area (hyperreflective nuclei) and also a light border between cells [6, 20, 34, 54].

In-vivo confocal microscopy is over specular microscopy because of higher resolution, image contrast, and the ability to image all corneal cellular layers. It is less affected by corneal pathologies such as edema or mild scarring [33].

Confocal microscopy shows two patterns of diseased endothelial changes in ICE syndrome. The first type is small cells with indistinct borders and bright, prominent, uniform nuclei. The second type is larger, epithelial-like cells with irregular borders and non-homogenous, diversely shaped nuclei, but increased reflectivity of the intercellular spaces is seen in nearly all cases [55].

In confocal microscopy, there are two types of ICE cells: ICE minus variant of endothelial cells (large cells with widely spaced centrally placed hyperreflective nuclei) or ICE plus variant of diseased endothelial cells (smaller cells with more tightly packed, eccentrically placed hyperreflective nuclei) without any effect of prognostic effect but more minus type in males [2]. Corneal confocal microscopy, with its high magnification, also detects ICE cells [2, 33, 55, 56], so confocal microscopy could provide good evidence for early diagnosis of ICE syndrome, especially for differentiation of ICE mimickers such as Fuchs endothelial dystrophy [56].

Ultrasound biomicroscopy (UBM) may be useful for detecting anterior chamber angle changes in corneal edema that make gonioscopy impossible [6, 51].

The two major vision-threatening sequelae of ICE syndrome are glaucoma and corneal decompensation. The severity of the disease and the predominant clinical variants differ among various ethnicities [1, 10, 57]. Chandran et al. reported secondary glaucoma in 73 % of the ICE cases. 50 % of them endured refractory glaucoma and were scheduled for glaucoma surgery. They also found corneal edema in 56 % of ICE syndromes, and 14 % of these cases needed corneal transplantation [9].

The differential diagnosis of the ICE syndrome is Fuchs endothelial dystrophy and Posterior polymorphous corneal dystrophy [15, 56].

Treatment

In recent years, there have been some advancements in the treatment of ICE syndrome, and new references have emerged. Currently, there is still no preventative method for the disease, and patients are typically monitored until they develop increased intraocular pressure or corneal decompensation [25]. The treatment focuses on managing the two major complications of the disease, corneal edema, and glaucomatous optic neuropathy.

Medical management remains the first choice of treatment for ICE syndrome, similar to other types of glaucoma. However, antiglaucomatous medications have limited effects on the disease due to its refractory nature, and medical therapy may have some limitations in these patients. For example, dorzolamide usage may lead to deterioration in the endothelial cell function [58-61], and latanoprost has been associated with herpes simplex recurrence, which may interfere with

the ICE syndrome [41]. Therefore, the rate of IOP control failure with topical antiglaucoma medications is high, ranging from 60-80 % [51]. Surgical intervention is often necessary for patients with ICE syndrome. Trabeculectomy has been used to treat the disease, but it results in a lower success rate than shunt procedures. The success rate of trabeculectomy ranges from 60-73 % at one year, approximately 40 % two years after surgery, and 21-29 % five years after surgery [5, 62-64]. Even after re-trabeculectomy, the success rate may still be low, with 20 % of cases requiring a second surgery [63]. The ingrowth of diseased endothelial cells in the filtration site may contribute to this failure [4]. In cases where trabeculectomy fails, glaucoma drainage devices may be required, with success rates ranging from 50-53 % after five years of follow-up [64, 65].

Cyclo-destructive procedures have been found to be more effective than other types of glaucoma treatments for controlling IOP in ICE syndrome, especially considering the age of patients. However, due to the ingrowth of diseased endothelial cells, deep sclerectomy is contraindicated in ICE syndrome [66].

Hypertonic solutions may provide temporary relief of morning visual blurring, but eventually, patients may need corneal transplantation, such as Descemet membrane endothelial keratoplasty or Descemet's stripping automated endothelial keratoplasty. However, DSAEK can be challenging in patients with ICE syndrome and peripheral anterior synechiae (PAS) due to the shallow anterior chamber and iris abnormalities that may make it harder for the donor tissue to unfold correctly [15, 67-73].

Recent studies have investigated new treatment options for ICE syndrome, including the use of selective laser trabeculoplasty (SLT) and gonioscopy-assisted transluminal trabeculotomy (GATT). SLT has shown promising results in reducing IOP in patients with ICE syndrome, with one study reporting a mean IOP reduction of 20.8 % after six months [74]. GATT, a minimally invasive surgical technique, has also shown promise in reducing IOP in patients with ICE syndrome, with one study reporting a mean IOP reduction of 52.8 % after six months [75]. Another study reported successful outcomes with the use of a microinvasive glaucoma surgery device called the Kahook Dual Blade in patients with ICE syndrome [76]. These new treatment options may provide additional options for managing IOP in patients with ICE syndrome and may help reduce the need for more invasive surgical interventions.

In summary, while there is no preventative method for ICE syndrome, medical management and surgical intervention can help manage the disease's complications. Despite its refractory nature, treatment options such as cyclo-destructive procedures and glaucoma drainage devices may be effective for controlling IOP. In cases where corneal transplantation is necessary, DSAEK can be challenging but remains a viable option for patients.

Conclusion: In conclusion, ICE syndrome is a rare disorder that can lead to significant vision loss and glaucomatous optic neuropathy. Despite the refractory nature of the disease, early diagnosis and careful management are crucial for a better prognosis. While medical management is the first choice of treatment, it may have limited effects due to the endothelial cell abnormalities present in ICE syndrome. Surgical options, including trabeculectomy, glaucoma drainage devices, and newer techniques such as selective laser trabeculoplasty and gonioscopy-assisted transluminal trabeculotomy, have shown

promising results in reducing IOP in patients with ICE syndrome. Additionally, careful consideration of the challenges associated with corneal transplantation techniques may be necessary. Close follow-up and monitoring are critical in the management of ICE syndrome to prevent catastrophic consequences and preserve vision.

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Footnotes and Financial Disclosures

Conflict of interest:

The authors have no conflict of interest with the subject matter of the present manuscript.