Vessel Cauterization as a Therapeutic Adjunct in Persistent Disciform Corneal Stromal Edema

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Abstract

This is a case report of a symptomatic non-clearing, vascularized, disciform, corneal stromal edema with a feeder vessel that has remained refractory to medical therapy of antiviral, steroid, and antibiotics, for a period of three weeks, but showed a rapid improvement in visual acuity of 0.1 Log mar within five days of feeder vessel cauterization, together with improvement of two psychometric scales in corneal cloudiness on a scale range of 0 to 3, clinical evidence of resolution of corneal edema, and subjective resolution of the patient's symptoms. Cauterization was done under magnification with a ball cautery point warmed in a spirit lamp following topical anesthesia. This intervention may become handy in difficult non-clearing corneal edema and prevent blinding consequences, in a low-resource facility, in selected applicable cases.

Keywords: Cauterization, corneal edema, disciform corneal edema, embolization, feeder vessel, pannus, viral keratitis

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Introduction

The corneal scar is responsible for 8% of the 1.3 million blind persons in Nigeria. Most of these are end-stage scars of persisting, non-clearing, vascularized corneal edema from various forms of keratitis, including, viruses and bacteria like Chlamydia, fungus, especially in rural uneducated farming population, and parasites like *Onchocerca volvulus* that cause onchocerciasis,[1] and physical as well as chemical agents. Beyond the physical disability, pain, discomfort, and economic burden; the disfiguring appearance of the eye causes a lot of psychological challenges, which in the traditional African setting may reduce the social values of the affected persons, especially when in the female gender. Keratitis of viral nature often tends to be recurrent and persistent because of the difficulty of total eradication of the viral gene from the host-infected cells. These recurrent inflammations break down the corneal anti-neovasculogenesis mechanism with a resulting in-growth of capillaries, and in some cases, larger arterioles and feeder vessels.

The normal cornea is an avascular, alymphatic, and immunologically privileged tissue in at least its very center. Vascular invasion bridges this immune privilege, allowing the diffusion of immune and allergic mediators, including cytokines, immunoglobulins, vasoactive proteins, and lipids, as well as, cellular infiltrates into the cornea. These mediators are capable of inducing increased vascular permeability in the normal vessels and inducing the growth of abnormal leaky vessels, with resulting edema in the surrounding corneal tissues.[2,3]

The resultant effect of all of these changes is loss of corneal clarity, pain and redness in the short run, as well as lipid and other forms of corneal degeneration, scarring, calcification, and finally functional visual loss in the long run.

Modalities that can rapidly stop the process of corneal edema will therefore be invaluable in preventing visual morbidity and blindness, with its consequent social and economic impact.

Case Report

A 26-year-old male, unemployed person, presented to the Ophthalmology Clinic on 14 August, 2012 with a three-week history of Blurring, Soreness, Lacrimation, Redness, and Pain in the left eye. He had three similar attacks in the last three months, for which he was hospitalized. His presenting visual acuity was 6/9 OD, 6/24 OS. He had lid edema, cilliary injection, a severely hazy central disciform corneal stromal edema on an old corneal scar, with lipid corneal degeneration, and multiple nummular keratitis at around the 1 o'clock position of the left cornea. The anterior chamber of the left eye did not show any cell or flare reaction. The pupil was small in size and reactive. The red reflex was poor due to the corneal haze, with no visible details of the fundus. The right eye of the patient was relatively normal.

A diagnosis of secondary Viral Disciform and Nummular Keratitis was made and patient was put on oral and topical ointment of Acyclovir together with a stat dose of cyclopentolate and antibiotics. He was to be followed up in a week by which time a steroid was to be included in his therapy.

The patient did not show up until six weeks later when his visual acuity had deteriorated to Counting Finger in the left eye. He complied with his last medical prescription for only the last two weeks to the date of his presentation. A topical steroid was added to his other medications and he was to be reviewed in another one week.

At the scheduled appointment, he was subjectively fine and comfortable, his vision had improved to 6/24 in the left eye and the conjunctiva injection had subsided; stromal edema had subsided in the superior cornea, and the nummular keratitis had disappeared. He had a prominent feeder vessel into an area of the thick corneal scar in the inferior aspect of the left cornea [Figure 1], with surrounding non-clearing, corneal edema and lipid keratopathy in the inferior-nasal quadrant bordering on the visual axis [Figure 2]. He had a deep anterior chamber, but the inferior iris and fundal details were not visible.

The relative quietness of the non-vascularized superior portion of the left cornea contrasted with the active edematous cornea area bordering on the feeder vessel in the inferior portion of the left cornea. It was, therefore, hypothesized that this could be explained by vascular leakage and the inflammatory mediators from the supplying feeder vessel into the adjacent cornea area. Therefore, an intervention of cauterization of the supplying feeder vessel was planned.

After the patient gave informed consent, he was put under a slit lamp microscope. The upper eyelid was manually retracted by the examiners gloved fingers after topical anesthesia with amethocaine. The cautery ballpoint was heated in a spirit lamp and the tip applied to about a 4 mm length of the sclera portion of the feeder vessel, to cauterize and separate the feeder vessel from the scarred and edematous portion of the inferonasal left cornea. The procedure lasted for about four minutes without any complication and was well-tolerated by the patient. The procedure was imaged pre- and post-intervention and the patient was given a stat dose of oral Ibuprofen–paracetamol combination. The immediate postoperative image is shown in Figure 3.

Five days post intervention, the patient felt well with no more pain and his vision had improved. On examination, Visual Acuity OD remained 6/9 and OS had improved to 6/18, unaided. There were conjunctival petechial hemorrhages in the area of the cautery and the intra-cornea feeder vessel had regressed [Figure 4]. On slit lamp examination, the corneal edema had completely regressed. The corneal clearity had improved

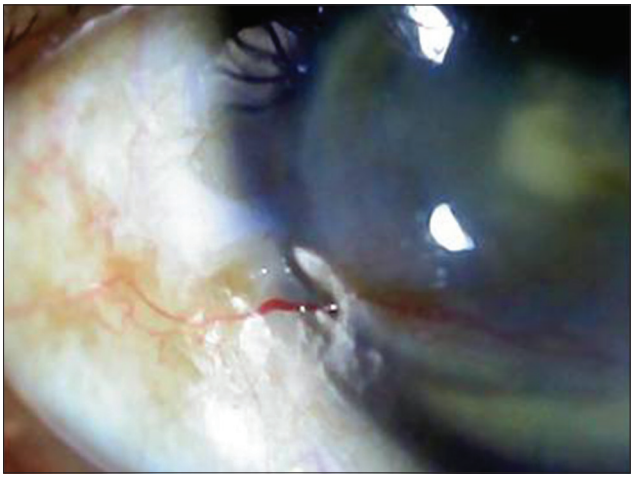

Figure 1: Image of edematous scarred vascularized left cornea

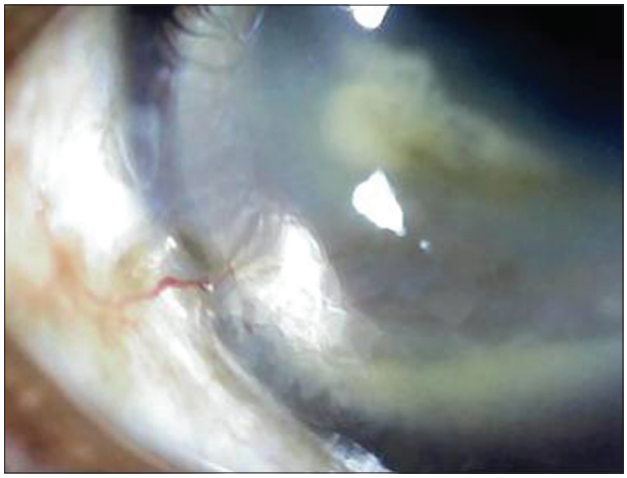

Figure 2: Image of edematous scarred left cornea

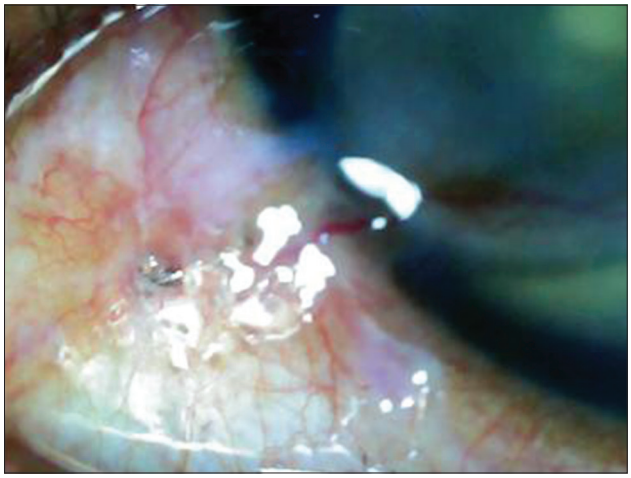

Figure 3: Image of the left eye, immediately post intervention

enough to allow easy visualization of the details of the inferior Iris and a normal fundus, with C:D = 0.3, a pink disc, normal macula, and a peripheral normal pink flat retina. These details were not visualizable prior to the cautery intervention five days earlier.

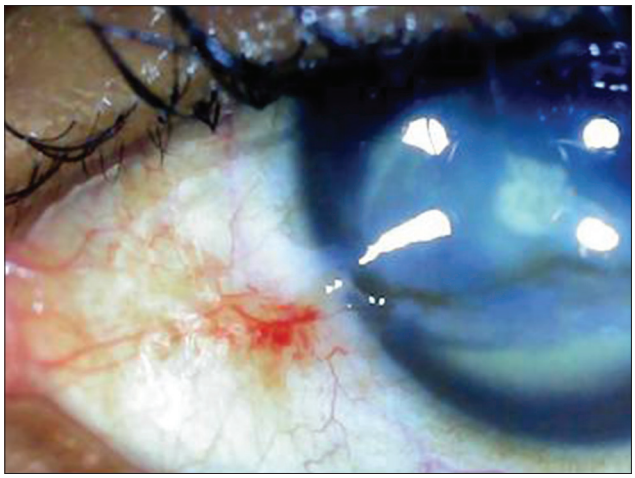

Figure 4: Image five days post intervention

This patient had achieved rapid corneal edema reversal with significant functional improvement and has remained so, via phone conversation, a month later.

Discussion

This patient had progressive worsening of visual acuity from 6/24 to CF in the left eye despite two weeks usage of antiviral ointment of Acyclovir, but had a reversal of visual deterioration from CF to 6/24 in the left eye upon one week's institution of steroid. This evidence supports the dependency of the pathology and its functional performance on immune mechanism, and therefore, the vessel that brings the mediators as a prospective target for immune modulation.

The concept of cauterization, embolization, and new vessel regression in control of abnormal blood flow,[3,4] infective keratitis, enhanced corneal graft tolerance, control of a lipid keratopathy,[5] and even metaplasia,[6] is not new, and numerous agents have been produced to achieve these purposes. Some of these include fine needle diathermy,[5] angiosclerotic agents, obliterative radiotherapy,[7] laser therapy,[8] thrombotic sponge, particles and microspheres, and in the eye, anti-vascular endothelial growth factor and steroids. These vessels are usually microvessels that supply inaccessible parts of the body as opposed to prominent macrovessels (pre-capillary arterioles) seen in an accessible part of the cornea in this patient. The size and location of the feeder vessel offered an opportunity for a cost-effective and feasible direct localized surgical cauterizing intervention, with wider diffuse corneal impact well away from the site of the burn.

Following medical intervention with antiviral, antibiotic, and steroid, this patient showed a differential regression of tissue pathology, edema and functional improvement in the vascularized and non-vascularized portion of the cornea, with the vascularized portion lagging behind in the regression of cornea edema and transparency. The rapid thinning out of the vascularized edematous cornea, assessed by the slit lamp beam, was well and beyond the change seen in the non-vascularized portion, coupled with a complete reversal of the relative thickness of the cornea in the vascularized and non-vascularized portions of the cornea following intervention gives a clue to the cause–effect relationship.

Although the patient had his first post intervention review in five days, the effect of edema regression was probably earlier, but was still adjudged rapid.

Macrovascular cauterization is affordable, requires no expensive technology, and is feasible even in remote economically poor-resourced regions, as only magnification, a spirit lamp, and a cautery point were required. It was an equally safe, short, and tolerable procedure in this patient.

Episcleral and limbal cauterization is a routine procedure in cataract surgery and has not been associated with any significant adverse effect, when correctly applied.

This method of cauterization will, however, be more difficult to apply where the panni are multiple, microvascular, and more diffuse.

Cornea vascularization with edema is an important theme in many important corneal diseases; therapy can, therefore, achieve a wider application.

Conclusion

This case study has demonstrated the accelerated regression of corneal edema and functional corneal performance following cauterization of a cornea feeder vessel. It is a safe, feasible, affordable, and accessible intervention to consider in difficult non-clearing corneal edema of selected patients. We encourage further research on this, to confirm the efficacy of this intervention, as well as researches to develop the applicable intervention of a similar nature for diffuse micropannus diseases of the cornea.

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