Review Article

Conjuntivochalasis: A Systemic Review

ABSTRACT

Conjunctivochalasis is defined as a redundant, nonedematous conjunctiva that causes a wide variety of symptoms ranging from completely asymptomatic, to worsening of an unstable tear film, and when severe, a real mechanical disruption of tear flow.

Due to the fact that conjunctivochalasis can be asymptomatic, it is often overlooked by the physician and it is classified as a normal variant of the eye due to the aging process. However, it must be kept in mind as a possibility for the origin of ocular irritation and tearing; and when severe enough it may be a cause a blurred vision, mucous discharge, dryness, ocular fatigue, subconjunctival hemorrhage, and eye stiffness upon awakening. This redundant conjunctiva can often be seen and frequently overlooked after tightening of lateral canthal tendon.

The symptoms are nonspecific and the onset may be insidious and uncertain, often confused with dry eye ones. Initial symptoms include foreign body sensation, burning, dry eyes and discomfort. Disorder of tear meniscus, impaired tear distribution and punctal occlusion play a role in the onset of symptoms. Slit lamp biomiocroscopy shows a prolapse of the conjunctiva over the lower lid margin in temporal, medial or nasal regions. This prolapsed conjunctiva causes a disruption of tear movement and outflow through the inferior lacrimal punctum, resulting in epiphora.

Although the etiopathogenesis of the disease has not yet been clearly understood, several theories have been proposed.According to the mechanical theory, age-related mechanical changes in the conjunctiva lead to a chronic obstruction of the lymphatic flow and lymphatic dilatation after this chronic obstruction leads to conjunctivochalasis. According to the inflammatory theory, the degradation of the extracellular matrix destruction in the tear increases as a result of disturbance in tear distribution. and this increase causes inflammatory changes resulting in conjunctival laxity.

While there is no need for treatment in the non-symptomatic group in the management of conjunctivosalasis, artificial tears and topical steroid treatment are the medical treatment options in symptomatic cases. [6] Surgical treatment is required in patients who do not respond to medical treatment.

Keywords:conjuntivochalasis,dry eye,epiphora cauter,argon laser, reduntant conjuntiva

INTRODUCTION

Conjunctivochalasis is defined as a redundant, nonedematous conjunctiva that causes a wide variety of symptoms ranging from completely asymptomatic, to worsening of an unstable tear film, and when severe, a real mechanical disruption of tear flow. **[1]**

Due to the fact that conjunctivochalasis (CCh) can be asymptomatic, it is often overlooked by the physician and it is classified as a normal variant of the eye due to the aging process. However, it must be kept in mind as a possibility for the origin of ocular irritation and tearing; and when severe enough it may be a cause a blurred vision, mucous discharge, dryness, ocular fatigue, subconjunctival

hemorrhage, and eye stiffness upon awakening. This redundant conjunctiva can often be seen and frequently overlooked after tightening of lateral canthal tendon**. [2]**

Conjunctivochalasis is usually bilateral, involving most commonly the inferotemporal conjunctiva, that may cause disruption of the inferior tear meniscus. It may present as loose superior conjunctiva, although it is less common.

The symptoms are nonspecific and the onset may be insidious and uncertain, often confused with dry eye ones. Initial symptoms include foreign body sensation, burning, dry eyes and discomfort. Disorder of tear meniscus, impaired tear distribution and punctal occlusion play a role in the onset of symptoms. **[3]** Slit lamp biomiocroscopy shows a prolapse of the conjunctiva over the lower lid margin in temporal, medial or nasal regions. **[Figüre 1,2]** This prolapsed conjunctiva causes a disruption of tear movement and outflow through the inferior lacrimal punctum, resulting in epiphora. **[Figüre 3]** Confusion arises in the management of this entity because there are other diseases and conditions that produce tearing and ocular irritation and must be ruled out as a causative agent of the tear flow disruption and tear instability (Dry Eye Syndrome, Keratoconjunctivitis Sicca, ocular allergies, thyroid eye disease, among others).

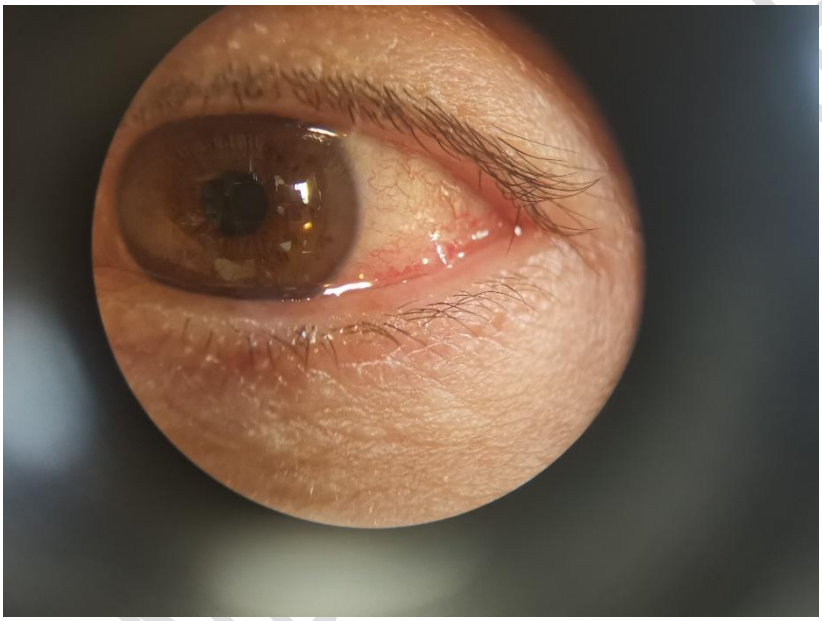

FIGURE 1: Prolapse of the conjunctiva over the lower lid margin in temporal region

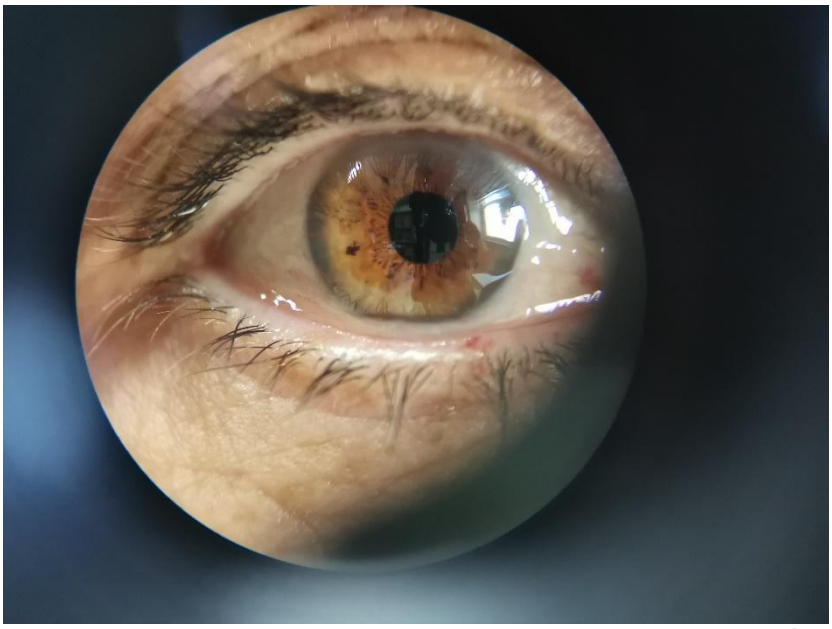

FIGURE 2: Prolapse of the conjunctiva over the lower lid margin in medial and temporal region

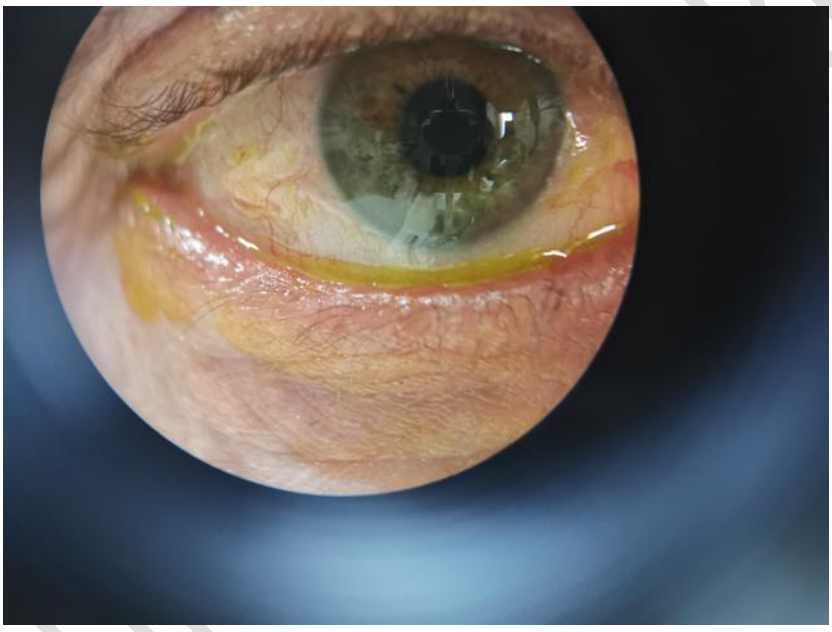

FIGURE 3: Prolapsed conjunctiva causes a disruption of tear movement and outflow through the inferior lacrimal punctum, resulting in epiphora.

Although the etiopathogenesis of the disease has not yet been clearly understood, several theories have been proposed.According to the mechanical theory, age-related mechanical changes in the conjunctiva lead to a chronic obstruction of the lymphatic flow and lymphatic dilatation after this chronic obstruction leads to conjunctivochalasis. **[4]**

According to the inflammatory theory, the degradation of the extracellular matrix destruction in the tear increases as a result of disturbance in tear distribution. and this increase causes inflammatory changes resulting in conjunctival laxity.

Previous studies in which histopathology of conjunctivalchalasis was evaluated by light microscopy, stromal lymphangiectasis, stromal edema, disintegration of elastic fibers, elastosis and chronic nongranulamatous inflammation were reported.Electron microscopy revealed that intercellular conjunctival stenosis and concomitant stromal elastin were observed**. [5]** While there is no need for treatment in the non-symptomatic group in the management of conjunctivosalasis, artificial tears and topical steroid treatment are the medical treatment options in symptomatic cases. **[6]** Surgical treatment is required in patients who do not respond to medical treatment.

CONJUNTIVOCHALASIS

Definition

Conjunctivochalasis was first described by Braunschweig in 1921. **[7]** After Duke - Elder evaluated it as a conjunctival hyperplasia requiring surgical excision or electrocoagulation, Liu et al. In 1986, they described this disease as loose, non-edematous excess bulbar conjunctival tissue protruding from the eyelid by entering the globe between the lower eyelid. **[8]**

Symptoms

In conjunctivochalasis patients, loose conjunctival tissue may not cause any symptoms, but excess conjunctival tissue may cause some symptoms by causing friction on conjunctival surface or between conjunctiva and cornea**. [2]** Blurred vision, watering, pain, stinging, burning, foreign body sensation, such as complaints of loose conjunctival tissue contributes to disrupt the tear meniscus. **[9]**

In these patients, where the tear distribution on the ocular surface is disrupted, although Schirmer test and tear break time measurements show that there is no lack of quality and quantity of tears, dry eye-like symptoms may occur.

Increased symptoms such as pain and blurred vision may be associated with increased conjunctival folds on the lower eyelid edge during activities that require prolonged stay in the downward position, such as reading. **[10]** In a study comparing dry eye with CCh cases without aqueous tear deficiency and CCh cases in terms of ocular surface symptoms, it was found that the symptoms increased in the later hours of the day in patients with dry eye, while the other group patients stated that they woke up and had more dryness during the reading.Increased inflammatory cytokines in tears due to decreased tear clearance during sleep have been thought to be the cause of symptoms that become evident when awakened**. [11]**

Etiopathogenesis and Histopathological Changes

No true aetiology is known for conjunctivochalasis. Senile changes involving the subcutaneous, elastic or supporting tissue in the conjunctiva was suggested as the cause. Eye rubbing, mechanical

irritation or trauma to conjunctiva, and abnormal eyelid position were all implicated. **[12]** Some investigators have suggested that the addition of some inflammatory processes to age-related changes in the structure of conjunctival tissue plays a role in the pathogenesis of the disease. **[5]** While the mechanical effect of the eyelids caused by the clipping movement contributes to these structural changes, local trauma, ultraviolet radiation and delayed tear clearance can accelerate the inflammatory process. **[13,14]**

Almeida et al. In their cohort study, they investigated the relationship between autoimmune thyroid diseases and CCh, and concluded that autoimmune thyroid disease was a predisposing risk factor for CCh. **[15]**

Although most of the patients with chronic disease suffer from irrigations, there are also researchers who suggest that dry eye plays an important role in the etiology. It has been argued that dryness of the ocular surface increases the mechanical effect of the eyelids on the conjunctiva and the amount of inflammation mediators released. **[9]**

Hoh et al. While conjunctivochalasis contributes to the development of dry eye, the clinical and physiopathological role of conjunctivochalasis in dry eye disease due to aqueous tear insufficiency is not clearly known**. [10,16]**

Francis et al., in a prospective clinical and histopathological study of 29 patients, 22 showed normal conjunctival histology, while 4 specimens showed inflammatory changes, and 3 specimens showed elastosis. They concluded and hypothesized that the etiology is multifactorial, including local trauma, UV radiation and delayed tear clearance as triggers of the disease. **[17]**

Harbiyeli II et al. showed mechanical and inflammatory factors induce development of CCh, and signs associated with these factors can be detected with light and electron microscopy of conjunctival tissue. **[18]**

Kalin et al. In 7 patients with whom they could not find any other reason, conjunctivalchalasis was named as “chronic localized conjunctival chemosis. **[19]**

Kheirkhah et al. in their study in the upper location CSA cases, upper bulbar conjunctival slack in the conjunctival tissue is not due to the excess of the conjunctival tissue, depending on insufficient function of the conjunctiva and sclera caused by the loss of adhesion suggested that this situation**. [20]**

There are two main theories about the mechanism that causes conjunctival tissue changes in the development of conjunctivosalasis. **[9]** One of them is the mechanical theory which argues that chronic obstruction caused by age-related mechanical changes in the conjunctival tissue plays the most important role in the pathophysiology of the disease, while the other is the extracellular matrix destruction mediated by tear cytokines mediated by increased tear cytokines due to delayed tear clearance.**[21]**

Inflammatory Theory

Inflammation of the ocular surface in conjunctivalchalasis is manifested by inflammatory cell infiltration in the conjunctival epithelium and stroma, and tears are also affected. Increased collagenolytic activity and degeneration of elastic fibers are changes in the extracellular components

of the conjunctival tissue**. [5]** Ocular surface inflammation has been suggested to play a role in the development of this degenerative process. It is thought that high inflammatory cytokines in the tears are released from the conjunctival epithelium or from the endothelium in the conjunctival vessels due to blinking and ocular movements and trauma to the loose conjunctiva.**[22]**

Clogging of the tear meniscus and punctum with excess conjunctival tissue causes delayed tear clearance. Delayed tear clearance increases the amount of cytokines in tears. **[10]**

Elevated levels of TNF-Alpha, IL-1, IL-6, IL-8 and IL-12 lead to increased levels of matrix metalloproteinase (MMP) involved in conjunctival epithelial and stromal connective tissue degradation and restructuring. The inflammatory response caused by elevated metalloproteinase levels, particularly MMP 3 and MMP 9, results in conjunctival elastosis.The role of increased oxidative stress due to insufficient function of antioxidant enzymes is known in age-related dry eye syndrome and skin aging. In a study on the role of oxidative stress in conjunctivochalasis, 8-hydroxy-2-deoxyguanosine (8-OHdG) showing oxidative stress-related DNA damage and N-hexanoyl-lysine (HEL) levels were evaluated as oxidative stress markers.In conjunctival samples, the number of conjunctival cells positively stained with N-hexanoyl-lysine and 8-hydroxy-2-deoxyguanosine and the HEL levels measured in tears were higher in the conjunctivosalasis group than in the control group, increased oxidative stress in etiopathogenesis. **[23]**

Acera A et al. find concentration of pro-MMP-9 was significantly higher in the conjunctivochalasis eyes than in the healthy controls in their study**. [24]**

Zhang XR et al. demonstrated the lamina propria of the bulbar conjunctiva mildly chronic inflammatory changes accompanied by a large number of lymphangiectasia who has conjunctivochalasis.They suggested bulbar conjunctival lymphangiectasia may be one of the reasons for the conjunctivochalasis. **[4]**

Jia YL et al. investigated the potential role of MAPK signaling pathways in conjunctivochalasis (CCH).They showed that the expression of p-ERK, p-JNK, and p-p38 in CCH conjunctiva was significantly higher than that in control group. The expression of p38 MAPK, JNK, and ERK proteins in CCH fibroblasts was significantly higher than that in control group. The total expression of MAPK mRNA in CCH fibroblasts was significantly higher than that in control group. The activated forms of p38 MAPK, JNK, and ERK proteins and mRNAs might up-regulate the expression of MMPs in CCH loose conjunctival tissue and fibroblasts, causing the degradation of collagen fibers and elastic fibers and promoting the occurrence of CCH. **[25]**

Li et al. showed overexpression of MMP-1 and MMP-3 mRNA in tissue cultured conjunctivochalasis fibroblasts, versus normal human conjunctival fibroblasts. Further studies showed that inflammatory mediators TNF-å and IL-1ß may increase the expression of MMP-1 and MMP-3 in conjunctivochalasis fibroblasts. Other inflammatory cytokines were also found in increased amounts in tear film of conjunctivochalasis patients (IL6 and IL8). After these observations, they concluded that the interaction of MMPs and their inhibitors, in a broken equilibrium may lead to a manifestation of a redundant conjunctiva with increased laxity. **[26]**

Gan JY et al. analysied of the literature, they found elastic fibers was a complex protein molecule from the structure and composition; the degradation of elastic fibers was one of the histopathological features of the disease; the vast majority of the factors related to the pathogenesis of CCh ultimately pointed to abnormal elastic fibers. By reasonably speculating, they considered that abnormal elastic fibers cause the conjunctival relaxation. In conclusion, they hypothesized that elastic fibers play an important role in the pathogenesis of CCh. Studies on the mechanism of

synthesis, degradation of elastic fibers are helpful to clarify the pathogenesis of the disease and to find effective treatment methods. **[27]**

Mechanical Theory

Another factor that plays a role in the development of conjunctivochalasis is age-related degenerative structural changes in the conjunctiva. **[28]** In addition to studies suggesting that inflammatory changes occurring in conjunctival epithelium and stroma lead to loose conjunctival tissue formation, there are also researchers who hypothesize that histopathological changes are only associated with conjunctival stroma and protect the structure of conjunctival epithelium**. [29]**

Watanabe et al. demonstrated subconjunctival microscopic telangiectasia in 4 of 39 specimens taken from patients with severe conjunctivochalasis, with no evidence of inflammation. They also noted fragmented elastic fibers and sparse assemblages of collagen fibers in 44 specimens. They concluded that mechanical forces between the lower eyelid and conjunctiva gradually impaired lymphatic flow, resulting in lymphatic dilation and clinically redundant conjunctiva. **[3]** Even though there is little to no histopathologic evidence to support inflammation as a major contributor to conjunctivochalasis, mechanistic evidence suggests a shift in the normal balance of the conjunctival matrix metalloproteinases (MMPs) and tissue inhibitors of metalloproteinases (TIMPs).

Poh S et al. showed that meibomian gland dysfunction and female gender were associated with lower tear meniscus area , while older age was associated with increased severity of conjunctivochalasis. **[30]**

Hashemian H et al. evaluated the histopathologic changes in the conjunctiva of patients with conjunctivochalasis (CCh) compared to age-matched controls.They showed that there were no significant differences between the two groups (27 patients with CCh and 16 control group) in terms of elastosis, fibrosis, lymphangiectasia, or infiltration of inflammatory cells in conjunctival stroma. Therefore,they suggested the primary pathology of CCh may not be within the conjunctiva itself. Instead, loose attachment of the conjunctiva to the underlying tissue may be the reason for the redundant folds in the bulbar conjunctiva. **[31]**

It has been suggested that these changes occur as a result of mechanical forces between the lower eyelid and the conjunctiva disrupting the lymphatic flow in the conjunctiva with advancing age. **[32]**

Clinical Findings

Conjunctivalchalasis is usually detected bilaterally by the presence of excess conjunctival tissue located nasally, centrally, or temporally around the lower eyelid**. [10]**

Depending on the amount of loose conjunctival tissue, conjunctival folds may be remarkable at first glance, and in some cases it may be necessary to push the conjunctiva over the eyelid to see the folds. On examination, decreased tear break time, corneal and conjunctival staining, delayed tear clearance, eyelid edge erosions, impaired tear meniscus, punctum blistering, subconjunctival haemorrhage are among the findings. **[33]** Pinguecula and loose eyelid syndrome are other ocular findings that can be seen in patients with conjunctivochalasis.

In the mild form of the disease, the tear film layer deteriorates due to the effect of the tear meniscus, while the tear clearance decreases in the next stage. In more serious forms of the disease,

conjunctival dryness may be seen due to external exposure of the dellen formation and the conjunctiva overhanging the eyelid edge due to tear dispersion**. [34]**

Loose conjunctival tissue accumulates at the edge of the lower eyelid, disrupting the tear meniscus and adversely affects tear dispersion. In addition, this excess in the conjunctival tissue causes inflammation at the eyelid edge due to its mechanical effect. Inflammation of the valve edge causes meibomian gland dysfunction and the lipid layer of the tear. **[10]** The reduced tear break time detected in these patients can be attributed to these changes in tear content and distribution. Corneal and conjunctival staining is another clinical finding related to tear content and distribution in the distribution. **[5,9]** Fluorescein and Rose-Bengal staining and corneal staining, as well as staining of conjunctival tissue folds can be shown.

In a study investigating the effect of tear inflammatory cytokines on ocular surface findings in conjunctivosalasis patients, the amount of inflammatory cytokine in tears was shown to be associated with corneal epithelial damage and corneal staining. **[5]**

Injury of the conjunctival epithelium, meibomian gland dysfunction as well as the friction effect caused by pinching between the eyeball and the lower eyelid.In addition to the deterioration of the tear meniscus, anatomical changes in the punctum also play a role in mechanically reducing the transition of the tear to the canalicular system. **[10]** Another reason for the tear overflow in these patients is that insufficient meibum does not show an effective lipid barrier function. Delayed tear clearance can be detected in these patients, where the complaint of watering is in the foreground.

Variable localization of loose conjunctival tissue on the sclera allows subconjunctival vessels to easily rupture by rubbing or blinking the eye. Another cause of subconjunctival hemorrhage is increased oxidative stress related vasculopathy. **[5]**

Francis et al. Emphasized that there were no ocular findings such as conjunctival staining, eyelid edge erosion, punctum swelling, subconjunctival hemorrhage in conjunctivochalasis patients with irrigated complaints, they suggested that these findings were related to drying on the ocular surface. **[35]**

Although CCh is frequently seen in the temporal conjunctival tissue of the lower eyelid, it can also be seen in the nasal and upper conjunctival tissues. Loose and excess conjunctival tissue in the upper bulbar conjunctiva may cause a clinical picture similar to superior limbic keratoconjunctivitis (SLK). Although the relationship between SLK and CCh has been emphasized in some studies, the pathological significance of this relationship is still unclear**. [36]** The loose upper bulbar conjunctiva, a characteristic clinical manifestation of SLK, has been proposed as evidence supporting the theory of mechanical friction in the pathogenesis of SLK, as in CCh**. [37]**

Cases with conjunctivalachalasis located nasally are characterized by different effects of loose conjunctival tissue on punctum and tear drainage mechanism.

Diagnosis

CCh is one of the most common misdiagnosed ocular surface disease. Non-specific symptoms along with intermittent findings of conjunctival edema often lead to misdiagnosis. Due to fluctuation in findings of disease process, several clinical exams may be required to confirm the diagnosis. Many

patients are first diagnosed when epiphora develops due to blocked puncta caused by redundant conjunctiva. Diagnosis of CCh is mainly clinical.

Slit lamp biomicroscopy shows prolapsed or folds of conjunctiva in temporal, nasal or central part of lower lid-margin. Presence of redundant conjunctiva over lower lid margin, reduced tear film break up time (BUT), anterior migration of muco-cutaneous junction caused by overspill of aqueous tears due to obliteration of tear meniscus suggests CCh. Prolapsed conjunctiva causes a disruption of tear movement and outflow through inferior lacrimal punctum resulting in epiphora.

Diagnosis of CCh is mainly clinical but also some medical device can be used.. Gumus K et al. showed that the AS-OCT is a useful and reproducible instrument to measure the cross-sectional area of conjunctiva prolapsing into the tear meniscus of patients with conjunctivochalasis. The method can monitor effectiveness of thermoreduction of conjunctivochalasis**. [38]**

The differential diagnosis can be difficult, as common signs and symptoms may be misleading. One tool that may help is tear film osmolarity. In symptomatic patients with a low osmolarity reading, conditions other than dry eye may be present.

Pain is another differentiator—patients with ocular surface disease often complain of chronic irritation; however, those with conjunctivochalasis complain of pain specifically in the affected area.Lastly, conjunctivochalasis may look similar to conjunctival chemosis, which often responds to antihistamines and anti-inflammatories. If you get a limited response from such therapies, consider conjunctivochalasis as the diagnosis. **[39]**

MANAGEMENT AND TREATMENT

Management must be individualize according to the severity and discomfort of each patient. No treatment is needed if the patient is asymptomatic. If a patient becomes mildly symptomatic, the clinical can start therapy with trials of lubrication and courses of topical corticosteroids. If patients continue to be in discomfort despite the medical management, surgical options can be taken into account.

Medical Treatment

If patients are asymptomatic then they can simply be observed. The finding of conjunctivochalasis alone does not warrant treatment.

When patients become symptomatic, the first line of treatment is medical. The goal is to reduce the effects of conjunctivochalasis, especially in regards to the disruption of the tear film. Lubricants tend to be the first-line medications, and while there have not been specific studies looking at different viscosities and their effects. However, more viscous drops are able to stay suspended on the surface of the conjunctiva and act in a fashion similar to a tear lake.

In severe cases in which the conjunctivochalasis causes the conjunctiva to be exposed even when the eyelids are closed, using artificial tear ointment and patching the eyes at night can be beneficial. **[40]** These patients may also need to use the ointment during the day if they have

significant desiccation of the exposed conjunctiva.

In addition to the medical approaches described, any other underlying inflammatory conjunctival conditions should be treated to assist in controlling the patient's symptoms. This includes treating allergic inflammation with topical antihistamines/mast cell stabilizers, and generally increased inflammation with topical steroids**.[40]** If patients remain symptomatic despite medical treatment, then surgery becomes a reasonable option.

Surgical Treatment

There are a number of different techniques that have been described to correct conjunctivochalasis. Some of these are minimally invasive and can be done in the office, while others require an operating room. According to the literature, Hughes was the first to successfully treat this condition by removing a section of the conjunctiva under the lower eyelid and closing the incision with a continuous black silk suture **[41]** Furthermore, suture fixation of the conjunctiva to the sclera with 6-0 Vicryl sutures has been described **[42]**. Additionally, electrocoagulation represents another method, which allows local inflammation to occur and the conjunctiva to attach to the subconjunctival Tenon's capsule **[43]**

Trivli and at al. previously reported another approach where, after surgical removal of the excess conjunctiva, preserved human amniotic membrane was placed over and sutured with a 10-0 nylon continuous suture to the free conjunctival edges . **[44]** In the another study, similar to electrocoagulation,their surgical procedure using radiofrequencies achieves conjunctival shortening by burning excessive conjunctiva. The operation is performed in an outpatient clinic setting, is applicable to the conjunctival folds at any location, and patients experience minimal intraoperative and postoperative ocular irritation**. [45]**

Cautery approaches:

Some type of thermally-induced shrinkage or excision to get rid of the redundant folds of conjunctiva clinically visible and resting on the lower lid. One study described using thermocautery as a way to excise the excess conjunctiva at the slit lamp with results showing greater than 90-percent improvement in both subjective and objective findings. **[46]** Their technique involved grasping the redundant conjunctiva with smooth forceps and then excising the grasped portion with a handheld low-temp cautery. There was scarring noted postoperatively in 15 percent of their patients, but it didn't have any sequelae.

Diana Muñoz, MD, and her colleagues in Bogota, Columbia, describe using a bipolar electrocautery forceps to apply the treatment directly to the symptomatic fold itself, which they identify by positive lissamine green staining. The procedure uses a traction suture through the inferior limbus to rotate the eye superiorly, so this may best be done in a controlled environment. After anesthetizing the area, the stained portion of conjunctiva is elevated and its base is grasped using the bipolar forceps. Energy is applied to the area directly at a rate of 30mA until "complete shrinkage" is achieved. The benefit of this technique is that you target the exact area the symptoms are coming from for that individual patient, whether it is nasal, inferior or temporal. Dr. Muñoz's group noted that all of their patients had complete resolution of their symptoms without scar development. **[47]**

Nakasato S et al. showed that thermocautery is a simple and effective treatment for symptomatic inferior conjunctivochalasis**. [48]**

Trivli A. et al. supported that CCh treatment with radiofrequencies appears to be a safe, quick, and effective surgical technique. Operation time is less than 10 min and can be performed in an outpatient clinic. **[45]**

Chan TC et al. showed that superficial conjunctival cauterization is an effective technique for management of conjunctivochalasis in the short term. An increase in tear film lipid layer thickness along with a decrease in corneal thickness and volume were observed after surgical correction of conjunctivochalasis**. [49]**

Kim KH et al. showed that electrocauterization for conjunctivoplasty can be advantageous in terms of inflammation compared with simple suturing and excision**. [50]**

Jiang LH et al. showed that conjunctivochalasis line bipolar coagulation and removal of loose conjunctiva crescent with considerable effect, bipolar coagulation was significantly shorter operative time, a significant reduction in postoperative complications, surgical procedures easier**. [51]**

Haefliger IO et al. showed that gentle superficial cauterization of the inferior bulbar conjunctiva can induced significant reduction of a moderate conjunctivochalasis**. [52]**

Argon laser:

Argon laser has also been used to “shrink” the redundant conjunctiva. In South Korea, Sangkyung Choi, MD, and his group describe using argon green laser treat the inferior conjunctiva. They apply approximately 100 burns during the treatment using “proper shrinkage” as the endpoint for their procedure. Their results showed a statistically significant improvement in the Ocular Surface Disease Index and in tear breakup time, improving from 9.2 seconds to 10.2 seconds. The treatments were more successful in mild and moderate cases. **[53]**

Yang J et al. used near-infrared laser thermal energy for conjunctivoplasty .In their study a fold of loose conjunctiva is grasped by a pair of forceps. The NIR laser light is delivered through an optical fiber and a laser line is aimed exactly on the conjunctival fold by a cylindrical lens. Ex vivo experiments using porcine eye was performed to investigate the induced shrinkage of conjunctiva and decide the optimal laser parameters. They found that up to 45% of conjunctiva shrinkage could be achieved**. [54]**

Incisional/glue approaches:

In the operating room, the redundant conjunctiva can be removed in a number of ways previously described in the literature, including: simple excision with direct closure **[55]** injection of fibrin glue subconjunctivally, then pinching and excising; **[56]** as well as a technique in which a limbal peritomy is made with radial relaxing incisions, allowing the loose conjunctiva to be pulled anteriorly and excised with subsequent approximation of the cut edge of conjunctiva to the limbus.**[57]**

Kheirkhah A et al. showed that reinforcement of conjunctival adhesion onto the sclera by amniotic membrane with either fibrin glue or sutures and they suggested their procudere is effective in alleviating symptoms and signs in eyes with superior CCh. **[58]**

Wang X et al. evaluated the efficacy of two surgical methods (simple resection method, and resection and fixation method) for conjunctivochalasis.They found the recurrence rate of conjunctivochalasis was 6/16 in the simple resection group and 1/17 in the resection and fixation group on month 6 after operation, and there was significant difference (P = 0.039).They said that both conjunctival resection and conjunctival resection with sclera fixation can effectively improve symptoms, but the latter 'resection and fixation method' has a lower recurrence rate**. [59]**

Santiago E et al. evaluated the outcomes of paste-pinch-cut conjunctivoplasty and cautery conjunctivoplasty for the treatment of symptomatic conjunctivochalasis. They found paste-pinch-cut and thermal cautery conjunctivoplasty are both safe and effective surgical treatments for the repair of conjunctivochalasis, with patients reporting greater improvement in symptoms after the cautery technique. **[60]**

Doss LR et al. described a new surgical technique for the repair of conjunctivochalasis (CCh) using subconjunctival injection of fibrin sealant followed by conjunctival resection.They suggested paste-pinch-cut conjunctivoplasty as a simple and an effective surgical treatment for the repair of CCh in symptomatic patients**. [61]**

Petris CK et al. supported that conjunctivoplasty using a simple medial conjunctival resection is an effective treatment for patients with epiphora secondary to conjunctivochalasis**. [62]**

Otaka I et al. showed that they treated conjunctivochalasis with conjunctival fixation to sclera, which strongly suggests that conjuctival folds are caused by the folding and the elevating of loosely adherent bulbar conjunctiva of the lower eyelid**. [63]**

Wang S et al. used a different surgery method which Conjunctival semiperitomy combined with gentle subconjunctival cauterization.they claimed that their procedure is a better therapy for conjunctivochalasis because it both resects the redundant conjunctiva and reestablishes the anatomic tight adhesion between the conjunctiva and the underlying sclera along the corneal limbus**. [64]**

However, the most highly recommended surgical procedures not only excise the redundant folds of the conjunctiva or tighten it, but also reestablish the fornix. This is because if excision alone is performed, one of the possible complications is scarring leading to foreshortening of the fornix and the possible development of a cicatricial entropion. It's now believed that, when attempting to improve the function of the tear film, returning the depth of the fornix to its physiologic baseline is as important as removing the redundant conjunctiva. Scheffer Tseng, MD, and his group at Bascom Palmer showed that the tear reservoir in the inferior fornix will rapidly replenish the tear meniscus in normal patients, but that this process is blocked by the additional redundant conjunctiva in the fornix of conjunctivochalasis patients. **[65]** The group showed that repairing this surgically and deepening the inferior fornix reestablishes the normal function of the reservoir and thus provides better resolution of dry eye and ocular surface discomfort symptoms than excision alone.

Dr. Tseng describes being able to normalize the fornix during excision of the conjunctiva by making a crescentic excision of the loose inferior bulbar conjunctiva starting with a peritomy approximately 2 mm posterior to the limbus. He excises all of the loose and thin conjunctival tissue, allowing the

remaining conjunctiva to recess into the fornix. The bare scleral defect is then covered with cryopreserved amniotic membrane and anchored using either sutures **[66]** or fibrin glue. **[67]** Glue has become preferred due to less inflammation and better patient comfort. In a retrospective review by Dr. Tseng's group, there was significant improvement in dry-eye symptoms and clinical findings. An added benefit was that 56 percent of their patients who had a prior diagnosis of aqueous-deficient dry eye, which they termed aqueous tear deficiency, had normalized on their fluorescein clearance testing. **[68]** They hypothesize that the conjunctivochalasis caused so much disruption and blockage in the fornix that it had created an aqueous-deficient state.

CONCLUSION

Ocular surface discomfort is a common and often frustrating condition for both patients and physicians. It's important to look at the entire ocular surface to diagnose and appropriately treat a patient's symptoms. With targeted treatment, there is a much better chance for success. To that effect, don't overlook the amount that conjunctivochalasis contributes to these symptoms. After trying conservative management with topical medications, if a patient is still symptomatic, then it's beneficial to take them to the operating room. While all of the surgical procedures discussed here produce great outcomes, excision of the redundant and loose conjunctiva with the attempt to reestablish the normal contour of the inferior fornix is a good choices.

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