

Conjunctivochalasis: 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 biomicroscopy 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 conjunctivochalasis, 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: conjunctivochalasis, dry eye, epiphora cauter, argon laser, redundant conjunctiva

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]

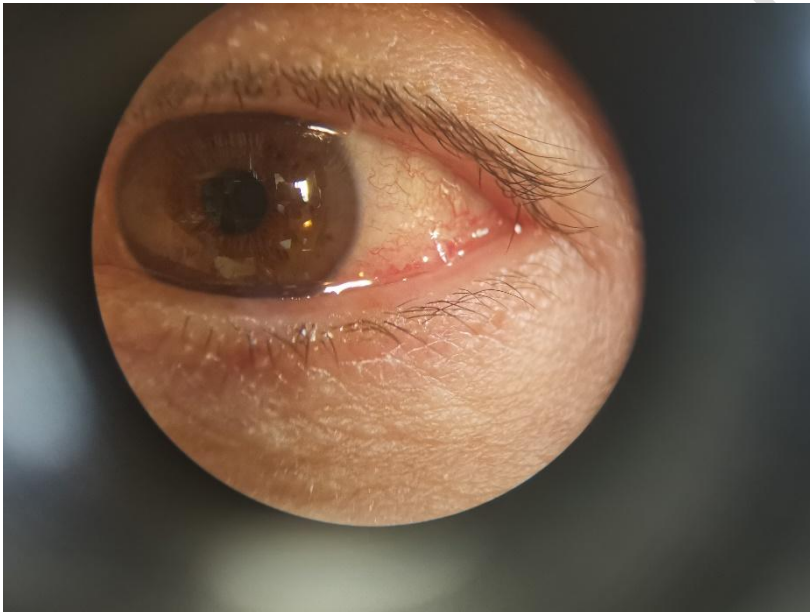
Due to the fact that conjunctivochalasis (CCh) can be asymptomatic, it is often overlooked by the physician and it is classified as a normal variant of the eye due to the aging process. However, it must be kept in mind as a possibility for the origin of ocular irritation and tearing; and when severe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

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

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

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

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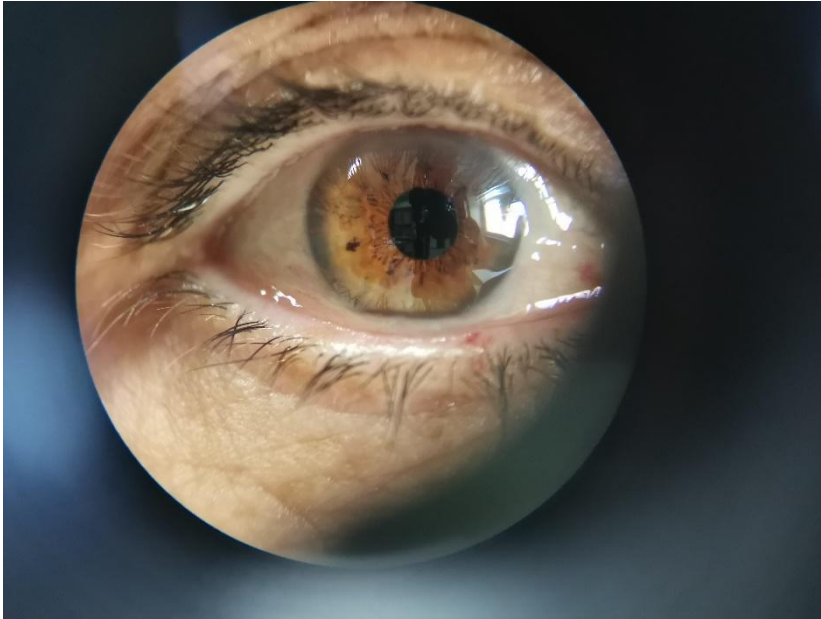


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36 **FIGURE 1:** Prolapse of the conjunctiva over the lower lid margin in temporal region

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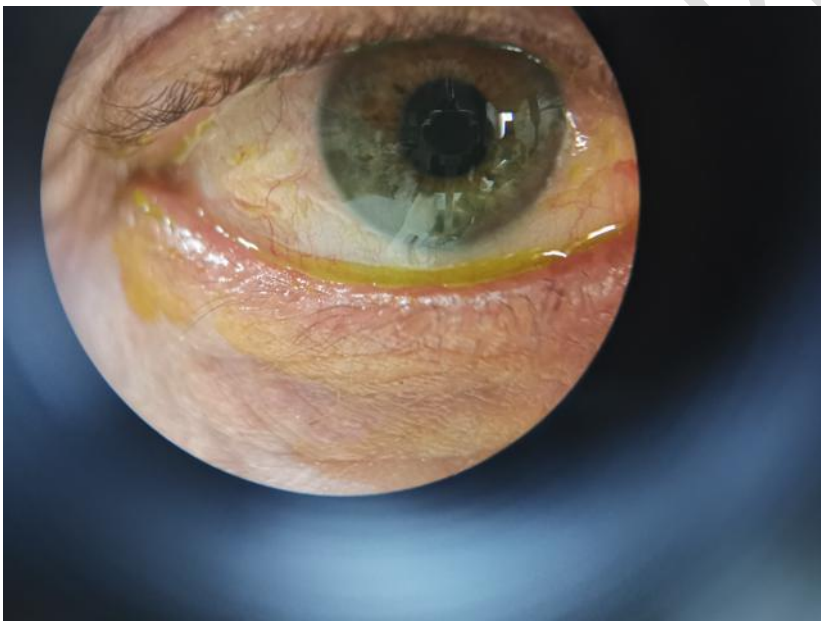
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40 **FIGURE 2:** Prolapse of the conjunctiva over the lower lid margin in medial and temporal region

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42

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

45

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

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

53 Previous studies in which histopathology of conjunctivalchalasis was evaluated by light
54 microscopy, stromal lymphangiectasis, stromal edema, disintegration of elastic fibers, elastosis and
55 chronic nongranulomatous inflammation were reported. Electron microscopy revealed that
56 intercellular conjunctival stenosis and concomitant stromal elastin were observed. [5] While there is
57 no need for treatment in the non-symptomatic group in the management of conjunctivosalasis,
58 artificial tears and topical steroid treatment are the medical treatment options in symptomatic cases.
59 [6] Surgical treatment is required in patients who do not respond to medical treatment.

60

61 **CONJUNTIVOCHALASIS**

62 **Definition**

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

67

68 **Symptoms**

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

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

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

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89 **Etiopathogenesis and Histopathological Changes**

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

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

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

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

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

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

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

115

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

118 Kheirkhah et al. in their study in the upper location CSA cases, upper bulbar conjunctival slack in the
119 conjunctival tissue is not due to the excess of the conjunctival tissue, depending on insufficient
120 function of the conjunctiva and sclera caused by the loss of adhesion suggested that this situation.
121 [20]

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

128

129 **Inflammatory Theory**

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

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

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

139 Elevated levels of TNF-Alpha, IL-1, IL-6, IL-8 and IL-12 lead to increased levels of matrix
140 metalloproteinase (MMP) involved in conjunctival epithelial and stromal connective tissue
141 degradation and restructuring. The inflammatory response caused by elevated metalloproteinase
142 levels, particularly MMP 3 and MMP 9, results in conjunctival elastosis. The role of increased
143 oxidative stress due to insufficient function of antioxidant enzymes is known in age-related dry eye
144 syndrome and skin aging. In a study on the role of oxidative stress in conjunctivochalasis, 8-hydroxy-
145 2-deoxyguanosine (8-OHdG) showing oxidative stress-related DNA damage and N-hexanoyl-lysine
146 (HEL) levels were evaluated as oxidative stress markers. In conjunctival samples, the number of
147 conjunctival cells positively stained with N-hexanoyl-lysine and 8-hydroxy-2-deoxyguanosine and the
148 HEL levels measured in tears were higher in the conjunctivochalasis group than in the control group,
149 increased oxidative stress in etiopathogenesis. [23]

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

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

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

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

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

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

179 **Mechanical Theory**

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

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

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

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

203

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

206

207 **Clinical Findings**

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

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

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

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

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

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

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

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

240 Francis et al. Emphasized that there were no ocular findings such as conjunctival staining, eyelid
241 edge erosion, punctum swelling, subconjunctival hemorrhage in conjunctivochalasis patients with
242 irrigated complaints, they suggested that these findings were related to drying on the ocular surface.
243 [35]

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

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

253

254

255 **Diagnosis**

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

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

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

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

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

273

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

279

280 **MANAGEMENT AND TREATMENT**

281

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

287

288 **Medical Treatment**

289

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

292

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

298

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

302 significant desiccation of the exposed conjunctiva.

303

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

309

310 **Surgical Treatment**

311

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

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

327

328 **Cautery approaches:**

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

336

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

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

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

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

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

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

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

363

364 **Argon laser:**

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

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

377

378 **Incisional/glue approaches:**

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

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

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

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

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

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

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

408 Wang S et al. used a different surgery method which Conjunctival semiperitomy combined with
409 gentle subconjunctival cauterization. They claimed that their procedure is a better therapy for
410 conjunctivochalasis because it both resects the redundant conjunctiva and reestablishes the
411 anatomic tight adhesion between the conjunctiva and the underlying sclera along the corneal limbus.
412 [64]

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

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

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

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442 **CONCLUSION**

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

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UNDER PEER REVIEW