

Management of Conjunctivochalasis

Keywords: argon laser, conjunctivochalasis, conjunctivoplasty, epiphora, dry eye, redundant conjunctiva

ABSTRACT

Conjunctivochalasis is defined as a redundant, nonedematous conjunctiva that causes a wide variety of symptoms. Excess conjunctival tissue may not cause any symptoms and may cause some symptoms like subconjunctival hemorrhage, epiphora, dry eye findings and corneal ulceration. Disturbance of tear meniscus, impaired tear distribution and punctal occlusion play a role in the onset of symptoms.

Although the etiopathogenesis of the disease is not yet 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 inflammatory theory, collagenolytic activity increases as a result of inflammation on the ocular surface, causing degeneration of elastic fibers. As a result, degeneration of elastic fibers lead to alterations in the extracellular components of the conjunctival tissue. This inflammatory changes resulting in conjunctival laxity.

Although conjunctivosalasis (CCh) is a clinical diagnosis, it is often overlooked by clinicians. CCh patients are can be symptomatic or asymptomatic. Medical and / or surgical treatment is generally needed in symptomatic patients, whereas treatment is not necessary in asymptomatic patients. Medical treatment is the first choice in the treatment of conjunctivochalasis. Artificial tear preparations are widely used in the treatment of CCh due to the deterioration of the tear film layer and dry eye symptoms. In clinical practice, topical anti-inflammatory eye drops are often preferred to reduce ocular surface inflammation. In cases where medical treatment is not sufficient, surgical treatment should be performed.

Today, there are many studies showing that surgical treatment is effective in reducing ocular symptoms and ocular surface damage in patients with CCh and in cases with and without dry eye. The surgical treatment plan should include the loose conjunctival tissue located in the lower part, as well as the excess conjunctival tissue located in the nasal and temporal regions and aim to correct the tear meniscus along the entire lower lid margin. The most preferred surgical method is crescent excision of CCh tissue and primary suture of the conjunctiva. Other surgical approaches include fibrin glue and amniotic membrane transplantation and direct scleral suture of CCh tissue. Another surgical method is electrocauterization of the conjunctival tissue. It is applied 5 mm away from limbus and there is no harm to fornixes.

INTRODUCTION

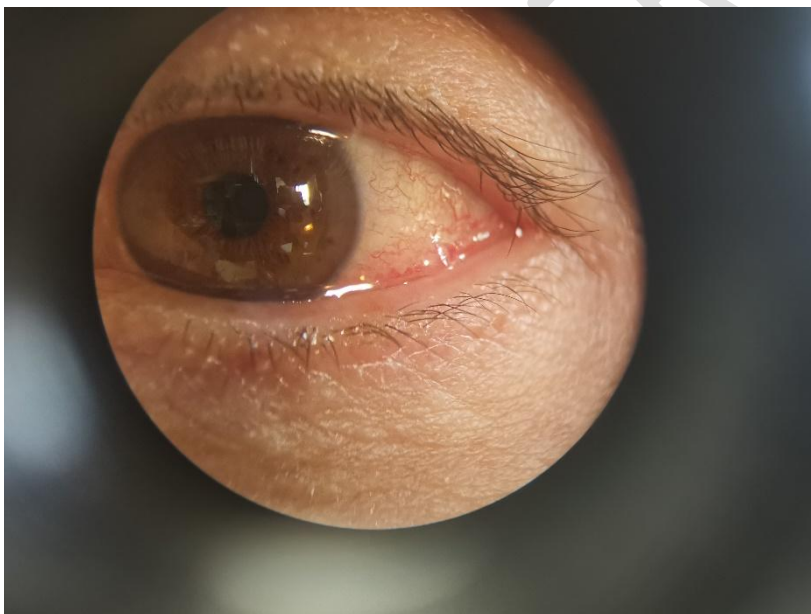
The conjunctiva is a mucous membrane that covers the inner part of the eyelids and the anterior part of the eyeball outside the cornea. The palpebral conjunctiva passes through the eyelids onto the bulbus and makes two recesses called fornix in the upper and lower parts. The conjunctiva on the tars is firmly attached to the underlying tissues and does not move. [1] Conjunctivochalasis is defined as a redundant, nonedematous conjunctiva that causes a wide variety of symptoms. Excess conjunctival tissue may not cause any symptoms and may cause some symptoms like subconjunctival hemorrhage, epiphora, dry eye findings and corneal ulceration. [2,3] Disturbance of tear meniscus, impaired tear distribution and punctal occlusion play a role in the onset of

20 symptoms.[2] Although the etiopathogenesis of the disease is not yet clearly understood, several
21 theories have been proposed.

22 Although conjunctivochalasis (CCh) is a clinical diagnosis, it is often overlooked by clinicians.[4]
23 CCh patients are can be symptomatic or asymptomatic. Medical and / or surgical treatment is
24 generally needed in symptomatic patients, whereas treatment is not necessary in asymptomatic
25 patients. Medical treatment is the first choice in the treatment of conjunctivochalasis.[5] Artificial
26 tear preparations are widely used in the treatment of CCh due to the deterioration of the tear film
27 layer and dry eye symptoms. In clinical practice, topical anti-inflammatory eye drops are often
28 preferred to reduce ocular surface inflammation. In cases where medical treatment is not sufficient,
29 surgical treatment should be performed. The most preferred surgical method is crescent excision of
30 CCh tissue and primary suture of the conjunctiva. [6] Other surgical approaches include fibrin glue
31 and amniotic membrane transplantation and direct scleral suture of CCh tissue.[7,8] Another
32 surgical method is electrocauterization of the conjunctival tissue. It is applied 5 mm away from
33 limbus and there is no harm to fornixes. [9,10]

34 The symptoms of CCh are nonspecific and the onset may be insidious and frequently confused
35 with other ocular surface diseases. Generally initial symptoms include itching, foreign body sensation,
36 burning, dry eyes and discomfort. Disorder of tear meniscus, impaired tear distribution and punctal
37 occlusion play a role in the onset of symptoms. [6] On the examination, biomicroscopy shows a
38 prolapse of the conjunctiva over the lower lid margin in temporal, central or nasal regions. [Figure
39 1,2] This loose conjunctiva obstructs the lower lacrimal punctum and causes epiphora. [Figure 3]

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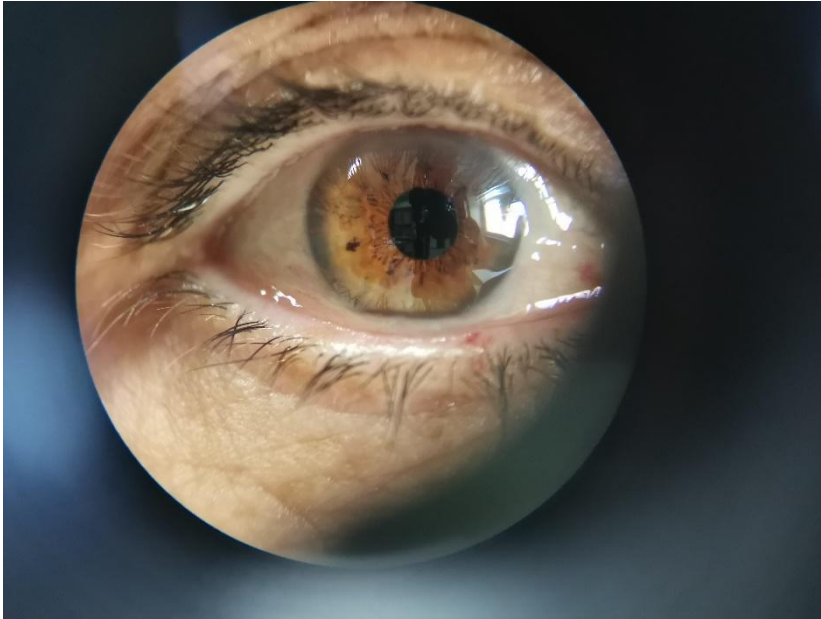


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42 **FIGURE 1:** Prolapse of the conjunctiva over the lower lid margin in temporal region

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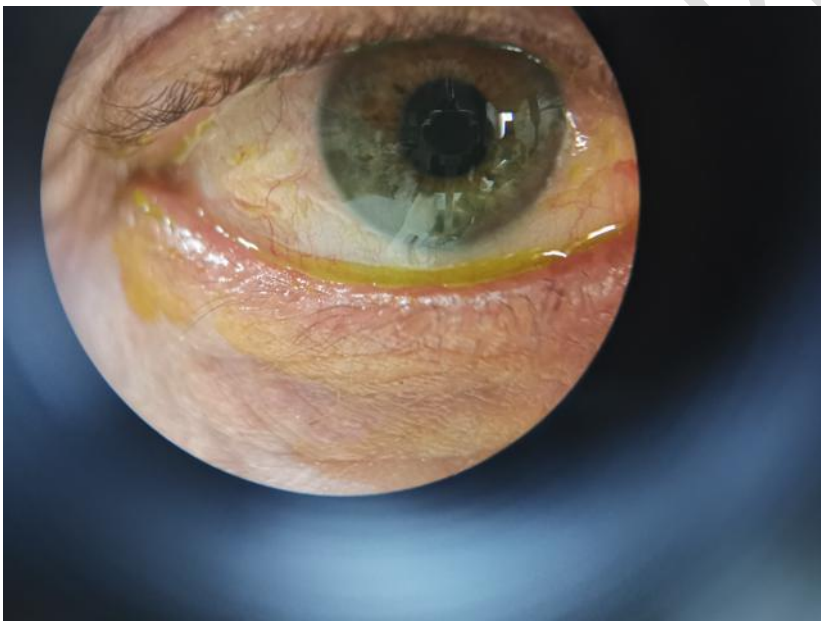
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46 **FIGURE 2:** Prolapse of the conjunctiva over the lower lid margin in temporal ,central and nasal region

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49 **FIGURE 3:** This loose conjunctiva obstructs the lower lacrimal punctum and causes epiphora.

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51 Although the etiopathogenesis of the disease has not yet been clearly understood, several
52 theories have been proposed. According to the mechanical theory, age-related mechanical changes in
53 the conjunctiva lead to a chronic obstruction of the lymphatic flow and lymphatic dilatation after this
54 chronic obstruction leads to conjunctivochalasis. [11]

55 According to inflammatory theory, collagenolytic activity increases as a result of inflammation on
56 the ocular surface, causing degeneration of elastic fibers. As a result, degeneration of elastic fibers
57 lead to alterations in the extracellular components of the conjunctival tissue. [8] This inflammatory
58 changes resulting in conjunctival laxity. [12]

59 Previous studies in which histopathology of CCh was evaluated by light microscopy, stromal
60 lymphangiectasis, stromal edema, disintegration of elastic fibers, elastosis and chronic
61 nongranulomatous inflammation were reported. Electron microscopy revealed that intercellular
62 conjunctival stenosis and concomitant stromal elastin were observed. [8] While there is no need for
63 treatment in the non-symptomatic group in the management of conjunctivosalasis, artificial tears
64 and topical steroid treatment are the medical treatment options in symptomatic cases. [13] Surgical
65 treatment is required in patients who do not respond to medical treatment.

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67 **CONJUNCTIVOCHALASIS**

68 **Definition**

69 Conjunctivochalasis was first described by Braunschweig in 1921. [7] After Duke - Elder evaluated
70 it as a conjunctival hyperplasia requiring surgical excision or electrocoagulation, Liu et al. in 1986,
71 described this disease as loose, non-edematous excess bulbar conjunctival tissue protruding from the
72 eyelid by entering the globe between the lower eyelid. [14]

73

74 **Symptoms**

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

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

82 Increased symptoms such as pain and blurred vision may be associated with increased conjunctival
83 folds on the lower eyelid edge during activities that require prolonged stay in the downward
84 position, such as reading. [15] In a study comparing dry eye with CCh cases without aqueous tear
85 deficiency and CCh cases in terms of ocular surface symptoms, it was found that the symptoms
86 increased in the later hours of the day in patients with dry eye, [16] while the other group patients
87 stated that they woke up and had more dryness during the reading. Increased inflammatory cytokines
88 in tears due to decreased tear clearance during sleep have been thought to be the cause of
89 symptoms that become evident when awakened. [17]

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98 **Etiopathogenesis and Histopathological Changes**

99 Today, the etiology of CCh has not been fully elucidated. It is thought to be due to the aging of
100 elastic structures and supporting tissues in the conjunctiva. Mechanical friction of the lids to the
101 conjunctiva, conjunctival trauma and abnormal eyelids are accused. [14] Some investigators have
102 suggested that the addition of some inflammatory processes to age-related changes in the structure
103 of conjunctival tissue plays a role in the pathogenesis of the disease. [14, 18] While the mechanical
104 effect of the eyelids caused by the clipping movement contributes to these structural changes, local
105 trauma, ultraviolet radiation and delayed tear clearance can accelerate the inflammatory process.
106 [19,20,21]

107 Almeida et al. in their cohort study, investigated the relationship between autoimmune thyroid
108 diseases and CCh, and concluded that autoimmune thyroid disease was a predisposing risk factor for
109 CCh. [22]

110 Although most of the patients with chronic disease suffer from irritations, there are also
111 researchers who suggest that dry eye plays an important role in the etiology. [23] It has been argued
112 that dryness of the ocular surface increases the mechanical effect of the eyelids on the conjunctiva
113 and the amount of inflammation mediators released. [9]

114 Hoh et al. concluded that CCh contributes to the development of dry eye, the clinical and
115 physiopathological role of conjunctivochalasis in dry eye disease due to aqueous tear insufficiency is
116 not clearly known. [10 ,24, 25]

117 Francis et al. showed that 7 of 29 specimens had pathological changes, as 4 specimens showed
118 inflammatory infiltrate and 3 specimen showed elastosis.They think etiology of conjunctivochalasis is
119 multifactorial like UV radiation and trauma.[7]

120 Harbiyeli II et al. showed mechanical and inflammatory factors induce development of CCh, and
121 signs associated with these factors can be detected with light and electron microscopy of
122 conjunctival tissue. [26]

123 Kalin et al. in 7 patients with whom they could not find any other reason, CCh was named as
124 "chronic localized conjunctival chemosis". [27]

125 Kheirkhah et al. in their study in the upper location CCh cases, upper bulbar conjunctival slack in
126 the conjunctival tissue is not due to the excess of the conjunctival tissue, depending on insufficient
127 function of the conjunctiva and sclera caused by the loss of adhesion suggested that this situation.
128 [28]

129 There are two main theories about the mechanism that causes conjunctival tissue changes in the
130 development of CCh. [9] One of them is the mechanical theory which argues that chronic
131 obstruction caused by age-related mechanical changes in the conjunctival tissue plays the most
132 important role in the pathophysiology of the disease, while the other is the extracellular matrix
133 destruction mediated by tear cytokines mediated by increased tear cytokines due to delayed tear
134 clearance.[29]

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139 **Inflammatory Theory**

140 Inflammation of the ocular surface [30] in CCh is manifested by inflammatory cell infiltration in
141 the conjunctival epithelium and stroma. Increased collagenolytic activity and degeneration of elastic
142 fibers are changes in the extracellular components of the conjunctival tissue. [5] Ocular surface
143 inflammation has been suggested to play a role in the development of this degenerative process. It
144 is thought that high inflammatory cytokines in the tears are released from the conjunctival
145 epithelium or from the endothelium in the conjunctival vessels due to blinking and ocular
146 movements and trauma to the loose conjunctiva.[12]

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

149 Elevated levels of TNF-Alpha, IL-1, IL-6, IL-8 and IL-12 lead to increased levels of matrix
150 metalloproteinase (MMP) involved in conjunctival epithelial and stromal connective tissue
151 degradation and restructuring. The inflammatory response caused by elevated metalloproteinase
152 levels, particularly MMP 3 and MMP 9, results in conjunctival elastosis. The role of increased
153 oxidative stress due to insufficient function of antioxidant enzymes is known in age-related dry eye
154 syndrome and skin aging. In a study on the role of oxidative stress in conjunctivochalasis, 8-hydroxy-
155 2-deoxyguanosine (8-OHdG) showing oxidative stress-related DNA damage and N-hexanoyl-lysine
156 (HEL) levels were evaluated as oxidative stress markers. In conjunctival samples, the number of
157 conjunctival cells positively stained with N-hexanoyl-lysine and 8-hydroxy-2-deoxyguanosine and the
158 HEL levels measured in tears were higher in the conjunctivochalasis group than in the control group,
159 [31] increased oxidative stress in etiopathogenesis. [32]

160 Acera A et al. find concentration of pro-MMP-9 was significantly higher in the
161 conjunctivochalasis eyes than in the healthy controls in their study. [33]

162 Zhang XR et al. demonstrated the lamina propria of the bulbar conjunctiva mildly chronic
163 inflammatory changes accompanied by a large number of lymphangiectasia who has
164 conjunctivochalasis. They suggested bulbar conjunctival lymphangiectasia may be one of the reasons
165 for the conjunctivochalasis. [8]

166 Jia YL et al. investigated the potential role of MAPK signaling pathways in conjunctivochalasis
167 (CCH). They showed that the expression of p-ERK, p-JNK, and p-p38 in CCH conjunctiva was
168 significantly higher than that in control group. The expression of p38 MAPK, JNK, and ERK proteins in
169 CCH fibroblasts was significantly higher than that in control group. The total expression of MAPK
170 mRNA in CCH fibroblasts was significantly higher than that in control group. The activated forms of
171 p38 MAPK, JNK, and ERK proteins and mRNAs might up-regulate the expression of MMPs in CCH
172 loose conjunctival tissue and fibroblasts, causing the degradation of collagen fibers and elastic fibers
173 and promoting the occurrence of CCH. [34]

174 Li et al. showed that overexpression of MMP-1 and MMP-3 mRNA by conjunctivochalasis
175 fibroblasts is correlated with their increased protein levels and proteolytic activities. They claim that
176 increase of this mediator can explain how conjunctivochalasis manifests excessive degradation of
177 the conjunctival matrix and Tenon's capsule. [19, 21]

178 Gan JY et al. reported that the degradation of elastic fibers is one of the histopathological features
179 of the disease. Abnormal elastic fibers were found in the majority of the factors related to the
180 pathogenesis of CCh. They thought that abnormal elastic fibers caused conjunctival relaxation. As a
181 result, they assumed that elastic fibers play an important role in the pathogenesis of CCh. [35,36]

182 **Mechanical Theory**

183 Another factor that plays a role in the development of CCh is age-related degenerative structural
184 changes in the conjunctiva. [29] In addition to studies suggesting that inflammatory changes
185 occurring in conjunctival epithelium and stroma lead to loose conjunctival tissue formation, there are
186 also researchers who hypothesize that histopathological changes are only associated with
187 conjunctival stroma and protect the structure of conjunctival epithelium. [37]

188 Watanabe et al. 44 patients were examined histologically. They found 39 patients had microscopic
189 lymphectectasis. In all cases, elastic fiber disintegration and sparse collagen fibers were seen. They
190 claimed that mechanical forces between the lower lid and conjunctiva gradually interfered with
191 lymphatic flow. Chronic, prolonged mechanical obstruction of the lymphatic flow may cause
192 lymphatic dilatation and finally lead to clinical conjunctivochalasis. [12,21]

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 CCh. [38]

195 Hasemi et al. obtained biopsies from the bulbar conjunctiva of 27 patients with CCh and 16 healthy
196 subjects. They found no significant difference in histopathological features (infiltration of
197 inflammatory cells, fibrosis, lymphangiectasia) between two groups. They suggested the primary
198 pathology of CCh may not be within the conjunctiva itself. Instead, loose ligation of the conjunctiva
199 to the sclera may be the cause of excessive folds in the bulbar conjunctiva. [39,40]

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201 It has been suggested that these changes occur as a result of mechanical forces between the lower
202 eyelid and the conjunctiva [21] disrupting the lymphatic flow in the conjunctiva with advancing age.
203 [6]

204

205 **Clinical Findings**

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

208 Depending on the amount of loose conjunctival tissue, conjunctival folds may be remarkable at
209 first glance, and in some cases it may be necessary to push the conjunctiva over the eyelid to see the
210 folds. On examination, decreased tear break time, corneal and conjunctival staining, delayed tear
211 clearance, eyelid edge erosions, impaired tear meniscus, punctum blistering, subconjunctival
212 haemorrhage are among the findings. [7] Pinguecula and loose eyelid syndrome are other ocular
213 findings that can be seen in patients with conjunctivochalasis.

214 In the mild form of the disease, degradation of tear film layers by the effect of tear meniscus, [31]
215 while the tear clearance decreases in the next stage. In more serious forms of the disease,
216 conjunctival dryness may be seen due to external exposure of the dellen formation and the
217 conjunctiva overhanging the eyelid edge due to tear dispersion. [13]

218 Loose conjunctival tissue accumulates at the edge of the lower eyelid, disrupting the tear
219 meniscus and adversely affects tear dispersion. In addition, this excess in the conjunctival tissue
220 causes inflammation at the eyelid edge due to its mechanical effect. Inflammation of the eyelid
221 margin causes meibomian gland dysfunction and the lipid layer of the tear. [10] The reduced tear
222 break up time detected in these patients can be attributed to these changes in tear content and

223 distribution. Corneal and conjunctival staining is another clinical finding related to tear content and
224 distribution in the distribution. [8,9] Cornea as well as conjunctival tissue folds can be shown to be
225 stained by fluorescein and Rose-Bengal dyes.

226 In a study investigating the effect of tear inflammatory cytokines on ocular surface findings in
227 conjunctivochalasis patients, the amount of inflammatory cytokine in tears was shown to be
228 associated with corneal epithelial damage and corneal staining. [8]

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

235 Variable localization of loose conjunctival tissue on the sclera allows subconjunctival vessels to
236 easily rupture by rubbing or blinking the eye. Another cause of subconjunctival hemorrhage is
237 increased oxidative stress related vasculopathy. [8]

238 Francis et al. emphasized that there were no ocular findings such as conjunctival staining, eyelid
239 edge erosion, punctum swelling, subconjunctival hemorrhage in conjunctivochalasis patients with
240 irrigated complaints, they suggested that these findings were related to drying on the ocular surface.
241 [7]

242 Although CCh is frequently seen in the temporal conjunctival tissue of the lower eyelid, it can also
243 be seen in the nasal and upper conjunctival tissues. Loose and excess conjunctival tissue in the upper
244 bulbar conjunctiva may cause a clinical picture similar to superior limbic keratoconjunctivitis (SLK).
245 Although the relationship between SLK and CCh has been emphasized in some studies, the
246 pathological significance of this relationship is still unclear. [41] The loose upper bulbar conjunctiva, a
247 characteristic clinical manifestation of SLK, has been proposed as evidence supporting the theory of
248 mechanical friction in the pathogenesis of SLK, as in CCh. [42]

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

251

252

253 **Diagnosis**

254

255 Although CCh not a rare disease, it is one of the most commonly misdiagnosed ocular surface
256 diseases. The nonspecific symptoms of the disease often cause misdiagnosis. Because of the different
257 findings during the disease, several examinations may be necessary to make the correct
258 diagnosis. The most common and first complaint of the patients is epiphora due to occluded puncta.
259 CCh is diagnosed clinically and usually does not require testing. Biomicroscopy shows prolapse
260 conjunctival folds in the nasal, temporal or central part of the lower lid margin. The presence of a
261 redundant conjunctiva on the lower lid, shortening of the tear film break up time (BUT), epiphora
262 indicate CCh. Prolonged epiphora leads to deterioration of the inferior lacrimal punctum. [21]

263 Differential diagnosis can be difficult because there are common symptoms and signs with many
264 diseases. Patients with ocular surface disease often complain of chronic irritation; However, those
265 with CCh suffer from pain in the affected area. Conjunctival chemosis may often be confused with
266 CCh. Consider CCh as a diagnosis if patients with conjunctival chemosis do not have sufficient clinical
267 response to anti-inflammatory and antihistamines.

268

269 Diagnosis of CCh is mainly clinical but also some medical device can be used. Gumus K et al.
270 showed that the AS-OCT is a useful and reproducible instrument to measure the cross-sectional area
271 of conjunctiva prolapsing into the tear meniscus of patients with conjunctivochalasis. The method
272 can monitor effectiveness of thermoreduction of CCh. [43]

273

274

275 **Differential diagnosis**

276 Conjunctivochalasis and dry eye syndrome (DES) due to aqueous tear insufficiency can be
277 confused both due to the similarity of the symptoms and the common clinical findings. Symptoms in
278 DES worsen later in the day, while CCh in the morning becomes more pronounced or unchanged
279 during the day. In DES, subjective complaints increase in upward position, while CCh increases
280 symptoms in downward position.

281 With the blink movement and the tear film covering the corneal surface, a feeling of relaxation is
282 seen in DES, while the mechanical effects and folds applied to the loose conjunctiva by blink increase
283 the symptoms in patients with CCh. While subconjunctival hemorrhage may be seen in CCh due to
284 the fragile conjunctival vessels in the loose conjunctival tissue, it is not an unusual finding in DES.
285 Fluorescein staining pattern was accompanied by a continuous shallow tear meniscus in DES, while
286 tear meniscus was interrupted or wiped by excess conjunctival tissue in CCh. Tear clearance was
287 found to be normal or delayed in DES, whereas it was often delayed in CCh. Punctum occlusion
288 increases the tear meniscus and tear ocular surface time and improves symptoms in DES, while the
289 increase in the amount of inflammatory cytokines in tears and exacerbation of epiphora causes an
290 adverse effect. Other diseases that may be considered in the differential diagnosis include
291 conjunctival tumors, trichiasis, entropion and ectropion. [42,44] Diagnosis of these diseases can be
292 made easily by careful examination including eyelids.

293

294

295 **MANAGEMENT AND TREATMENT**

296

297 Treatment should be specific to the patient's signs and symptoms. If the patient is asymptomatic
298 there is no need to treat. Patient can be followed up periodically before the treatment is started for
299 progression findings. Treatment options in symptomatic patients are divided into two as medical and
300 surgical treatment. Firstly, medical treatment options should be considered and surgical procedures
301 should be applied in cases with no response. [21]

302
303
304

Medical Treatment

305 Symptoms of CCh are caused by a significant deterioration of the tear meniscus and compression
306 of the loose conjunctival tissue between the globe and the lower eyelid. Artificial tear and lubricant
307 gel treatment can provide partial relief both by regenerating the deteriorating tear film layer and by
308 reducing the frictional complaints associated with lubricant effect. [3,29,46]

309 Increased inflammation on the ocular surface is blamed in the etiopathogenesis of the disease and
310 its reduction should be one of the goals of treatment in symptomatic patients. [19] Low-dose topical
311 steroids, topical anti-inflammatory agents, and cyclosporine A can be used to both prevent
312 progression and reduce existing complaints. [19,28] However, other anterior segment problems
313 associated with cch should be treated. (blepharitis, allergic conjunctivitis, dry eye, etc.) [47]

314

315

Surgical Treatment

317 Today, there are many studies showing that surgical treatment is effective in reducing ocular
318 symptoms and ocular surface damage in patients with conjunctivochalasis and in cases with and
319 without dry eye. [5,29,48]

320 Surgical treatment of CCh patients with tear film defects is positive. In the traditional approach,
321 punktal occlusion is considered as the first choice in patients with conjunctivochalasis who have dry
322 eye and medical treatment is inadequate. In some recent studies, it has been reported that CCh
323 surgery is the first-line treatment option. [48]

324 The surgical treatment plan should include the loose conjunctival tissue located in the lower part,
325 as well as the excess conjunctival tissue located in the nasal and temporal regions and aim to correct
326 the tear meniscus along the entire lower lid margin. Some studies have reported the inclusion of
327 pinguecula into the excised conjunctival tissue in the presence of inflamed pinguecula associated
328 with CCh. [13]

329 It has been shown in many studies that surgical treatment is effective in reducing ocular
330 symptoms and ocular surface damage in patients with non-responsive medical treatment. [5,29,48]

331 Nowadays, there are several different surgical techniques to treat CCh. Some of these surgical
332 techniques require operating room conditions, while others require outpatient clinic. Dr. Hughes
333 performed the first known conjunctivochalasis surgery. [49] Hughes closed the conjunctival incision
334 with continuous suture after cutting the conjunctiva. However, he described the fixation of the
335 conjunctiva to the sclera with a 6-0 Vicryl suture. [50] In addition to surgical excision of the
336 conjunctiva, the local conjunctiva is produced by electrocoagulation method and this method allows
337 the conjunctiva to be firmly attached to the Tenon capsule. [22] Surgical techniques mentioned in the
338 literature are listed below.

339

Cautery treatment

341 One of the surgical methods is electrocoagulation. A type of thermally induced shrinkage or
342 excision that is avoid unnecessary conjunctival folds in the lower lid. It is easy to apply and can be
343 done in outpatient conditions also increases its popularity. With this method, local inflammation of

344 the conjunctiva is created. As a result the conjunctiva is firmly attached to the Tenon capsule.[4]

345

346 Subconjunctival electrocoagulation was performed using a fine needle electrode placed under the
347 loose conjunctival tissue removed by forceps in a study using high frequency radiod wave. In this
348 method, the power of the radiofrequency generator was adjusted to allow the conjunctival tissues to
349 contract without burning and 10-20 subconjunctival coagulations were performed in the horizontal
350 plane. In severe cases, vertical coagulation was added. During the application, care was taken not to
351 cauterize the vascular structures adjacent to the limbus and the procedure was terminated when the
352 surgeon decided that there was no loose conjunctival tissue. Advantages of the technique over other
353 methods; good surgical control, minimal scar formation, better wound healing and better cosmetic
354 outcome. Youm et al. said that there were no serious cicatricial complications after electrocautery
355 application and conjunctival inflammation in the membrane.[2]

356 Trivli A. et al. said that CCh treatment with radiofrequencies seems to be a safe, short-term and
357 useful surgical technique. The operation time is very short and can be performed in places like
358 outpatient clinics that do not require much equipment.[49]

359 Nakasato S et al. showed that thermocautery is a simple and effective treatment for symptomatic
360 inferior CCh. [51]

361 Chan TC et al. showed that superficial conjunctival cauterization is an effective technique for
362 management of conjunctivochalasis in the short term. An increase in tear film lipid layer thickness
363 along with a decrease in corneal thickness and volume were observed after surgical correction of
364 CCh. [52]

365 Kim KH et al. showed that electrocauterization for conjunctivoplasty can be advantageous in
366 terms of inflammation compared with simple suturing and excision. [53]

367 Jiang LH et al. showed that conjunctivochalasis line bipolar coagulation and removal of loose
368 conjunctiva crescent with considerable effect, bipolar coagulation was significantly shorter operative
369 time, a significant reduction in postoperative complications, surgical procedures easier. [54]

370 Haefliger IO et al. showed that gentle superficial cauterization of the inferior bulbar conjunctiva
371 can induced significant reduction of a moderate conjunctivochalasis. [55]

372

373 Argon laser treatment

374 Argon laser has been used for the treatment of many eye-related diseases as well as to shrink the
375 redundant conjunctiva. [56]

376 Choi S. et al. treated with argon green laser of 29 eyes of 18 patients with CCh. They argue that
377 the results of the study are clinically significant, and this method is a simple, effective and easily
378 applicable method, especially in mild and moderate CCh. [4]

379 Yang J et al. used near-infrared laser thermal energy for conjunctivoplasty .In their study a fold of
380 loose conjunctiva is grasped by a pair of forceps. The laser light is delivered through an optical fiber
381 and a laser line is aimed exactly on the conjunctival fold by a cylindrical lens. Ex vivo experiments
382 using porcine eye was performed to investigate the induced shrinkage of conjunctiva and decide the
383 optimal laser parameters. They found that up to 45% of conjunctiva shrinkage could be achieved.
384 [57]

385

386 **Incisional/glue approaches:**

387 ***Crescent Resection and Primary Suture***

388 This technique described by Braunschweig involves crescent excision of the lower bulbar
389 conjunctiva at a distance of 5 mm from the limbus and incorporation of the remaining conjunctival
390 tissues with suturing.[55]

391 Petris CK et al. supported that conjunctivoplasty using a simple medial conjunctival resection is an
392 effective treatment for patients with epiphora secondary to conjunctivochalasis. [56]

393 Wang X et al. evaluated the efficacy of two surgical methods (simple resection method, and
394 resection and fixation method) for CCh. They found the recurrence rate of conjunctivochalasis was
395 6/16 in the simple resection group and 1/17 in the resection and fixation group on month 6 after
396 operation, and there was significant difference ($P = 0.039$). They said that both conjunctival resection
397 and conjunctival resection with sclera fixation can effectively improve symptoms, but the latter
398 'resection and fixation method' has a lower recurrence rate. [58]

399 ***Combined Resection with Radial Relaxing Incision and Lower Peritomy***

400 Serrano and Mora modified this technique in order to prevent postoperative complications such
401 as scar tissue and lower conjunctival fornix retraction of the technique described by
402 Braunschweig.[59] This modification involves the excision of the loose conjunctiva with two relaxant
403 radial incisions.[13,59]

404 Wang et al. performed a corneal limbus-based conjunctival semiperitomy. Subsequently, they
405 applied cauterization to the subconjunctival area. They claimed that conjunctival semiperitomy
406 combined with subconjunctival cauterization is a clinically relevant surgical technique for CCh.
407 Because both the redundant conjunctiva and the corneal limbus re-establish the anatomical tight
408 adhesion between the underlying sclera.[60]

409 ***Conjunctival Excision, Episcleral Fixation and Amniotic Membrane Transplantation with Fibrin***
410 ***Adhesive***

411 It has been shown in many studies that the amnion membrane can be used effectively in the
412 repair of conjunctival surface after conjunctival tissue excision in CCh surgery.[13,28,61]

413 Fixation of the amniotic membrane to the scleral surface can be accomplished by suturing or the
414 use of tissue adhesives. The use of suture has many disadvantages, such as prolonged surgical time,
415 reduced patient comfort in the early postoperative period, and the incidence of suture related
416 complications (abscess, granuloma formation, development of giant papillary conjunctivitis).
417 Therefore, as in other ocular surface surgery, the use of fibrin-based tissue adhesives has become
418 widespread in CCh surgery.[28]

419 In a retrospective study, Tseng et al. demonstrated that surgical repair of conjunctivochalasis and
420 deepening of the lower fornix provided better clinical results than excision alone. Tseng describes the
421 normalization of the fornix during the removal of the conjunctiva by allowing the loose inferior
422 bulbar conjunctiva to perform a crescent excision beginning with a peritomy about 2 mm posterior to
423 the limbus. The bare scleral defect is then covered with frozen amniotic membrane and secured
424 using sutures [62] or fibrin glue. [63].

425 In this surgical technique, conjunctival peritomy was performed in the lower conjunctiva at 1-2 mm
426 distance from the limbus and excess conjunctival tissue was excised. Then, the appropriate sized
427 amniotic membrane laying on the sclera with the stromal surface facing downwards was folded over
428 itself and fibrinogen solution was dropped onto the stromal surface of the membrane. Following
429 administration of thrombin solution to the scleral surface, the folded half of the amniotic membrane
430 was laid on the scleral surface. The same procedure was repeated for the other half of the amniotic
431 membrane and bare sclera, allowing the membrane to be laid smoothly on the scleral surface. [63]

432 In this technique, the fixation of conjunctival tissue adjacent to the fornix with 8-0 or 10-0
433 polyglactin sutures can be added to the sclera to prevent the recession of the conjunctiva remaining
434 distal to the excised conjunctival tissue.

435 ***Conjunctival Excision and Amniotic Membrane Transplantation with Suture***

436 In this technique, the bare scleral area was covered with amniotic membrane after crescent excision
437 from the lower conjunctiva at a distance of 2 mm to the limbus and fixed with 9-0 and 10-0 nylon
438 sutures passing through the episclera to the membrane conjunctival edges. During fixation of the
439 amniotic membrane on the scleral surface, care was taken to ensure that it was laid smoothly.[13]

440 ***Conjunctival Excision and Closure of Tissue Adhesive***

441 Looser conjunctival tissue was excised by two radial incisions to the medial and lateral cantus
442 following a peritomy of 180 degrees of the lower conjunctiva. Using tissue adhesive, conjunctival
443 wound lips were joined and conjunctival integrity was achieved.[64]

444 ***Strengthening of Conjunctival Adhesion on Sclera with Amniotic Membrane***

445 It is thought that the underlying pathology is not an excess of conjunctival tissue in upper
446 pathogenesis of CCh and loss of adhesion between conjunctiva and sclera causes a loose appearance
447 in conjunctiva.[28] For this reason, the surgical technique to be applied in cases with upper-located
448 CCh should be aimed at increasing the adhesion between the conjunctiva and sclera rather than
449 excising the conjunctiva.

450 Kheirkah et al. in the surgical technique applied to the upper-located CCh cases, they excised the
451 weak tenon tissue after conjunctival peritomy performed between 10 and 2 hours at a distance of 5
452 mm from the limbus, then covered the amniotic membrane with the sclera and stabilized with fibrin
453 glue or suture. The conjunctival flap covered on the amniotic membrane is also attached to the
454 amniotic membrane by tissue adhesive or suture. With this method, adhesion between the
455 conjunctiva and sclera was strengthened without excision of the conjunctival tissue, preventing the
456 loose upper bulbar conjunctival tissue from sagging on the cornea.[28] In the study, it was reported
457 that there was no significant difference between the patient group who underwent suturing and
458 tissue glue in terms of both symptoms and signs in the postoperative period. [28]

459 Santiago E et al. evaluated the outcomes of paste-pinch-cut conjunctivoplasty and cautery
460 conjunctivoplasty for the treatment of symptomatic CCh. They found paste-pinch-cut and thermal
461 cautery conjunctivoplasty are both safe and effective surgical treatments for the repair of
462 conjunctivochalasis, with patients reporting greater improvement in symptoms after the cautery
463 technique. [4,65]

464 Doss LR et al. using subconjunctival injection of fibrin sealant followed by conjunctival
465 resection. They received 139 eyes of 70 patients with conjunctivosalasis who did not respond to

466 medical treatment. They have achieved 90% clinical success with the paste, pinch and cut method
467 they have defined. [4,66]

468 Otaka I et al. showed that they treated conjunctivochalasis with conjunctival fixation to sclera,
469 which strongly suggests that conjunctival folds are caused by the folding and the elevating of loosely
470 adherent bulbar conjunctiva of the lower eyelid. [62,67]

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473 **CONCLUSION**

474 Conjunctivochalasis is a common disease especially in the elderly and may cause ocular surface
475 complaints especially in the advanced stages. Patients with ocular surface complaints should receive
476 a good history and a detailed ophthalmologic examination. Since there are many diseases that may
477 cause similar ocular surface complaints, conjunctivochalasis is one of the preliminary diagnoses that
478 comes to mind during the examination and it is very important for the diagnosis. When medical
479 treatment is not sufficient, surgical methods should be applied. Although most of the methods used
480 in CCh surgery are successful, the most appropriate surgery should be chosen for the patient.

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