Chapter 14
Recent Advances in Conjunctivochalasis

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Ocular surface health is maintained by a stable tear film, which requires both compositional and hydrodynamic factors [1]. The former comprises adequate production of mucins and aqueous tears. The latter includes the eyelids, with blinking to allow tear spread and clearance, whereas the eyelids close to avoid exposure. The stability and spread of the tear film requires coordinating effects between both compositional and hydrodynamic elements in the ocular defense system [1]. The compositional elements are comprised of the lacrimal glands, the meibomian glands, and the ocular surface epithelium to provide aqueous, lipid, and mucins in the tear fluid, respectively, while the hydrodynamic elements include tear spread, drainage, and evaporation, which are controlled by eyelid blinking and closure [1]. With each blink, tears spread from the conjunctival cul-de-sac in the fornix (third compartment) to the tear meniscus (second compartment) and finally to the preocular tear film (first compartment) [2, 3]. Also through blinking, the tear fluid is cleared into the nasal lacrimal drainage system.

Conjunctivochalasis (CCh) is defined as a disease process leading to the formation of a loose and redundant conjunctival fold interspersed between the globe and eyelids [4]. Advances have been made in the pathogenesis of CCh as a disease that is characterized by overexpression of matrix metalloproteinase (MMP) by CCh fibroblasts [5, 6]. The redundant conjunctival folds of CCh Conjunctivochalasis (CCh) redundant conjunctival fold interfere the tear flow by blocking the tear drainage puncta [7], disrupting continuity of the tear meniscus [4, 7, 8], and extending

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A.R. Djalilian (ed.), *Ocular Surface Disease*,
https://doi.org/10.1007/978-3-319-15823-5_14

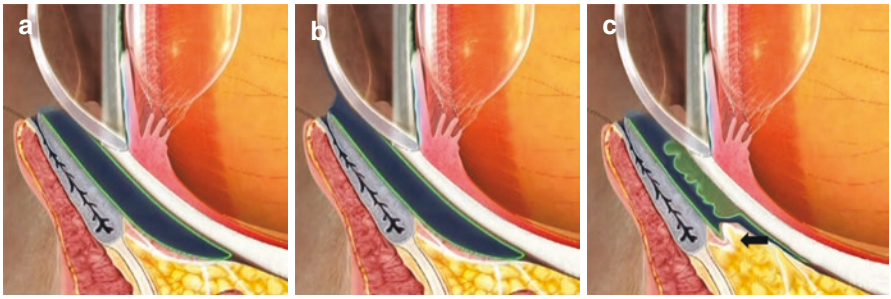

Fig. 14.1 Schematic demonstration of tear flow obliterated by CCh. Under the normal circumstance, the fornix tear reservoir depicted in *dark blue* is responsible for delivering tear fluids to the tear meniscus (**a**) and preocular surface (**b**) by blinking. In CCh, tear spread and fornix tear reservoir are obliterated by loose and wrinkled conjunctiva (**c**, *green*) and prolapse fat (**c**, *arrow*) due to degenerated Tenon's capsule (Taken with permission from Cheng et al. [9])

from the lid margin/meniscus locale to the fornix [9] to obliterate the tear flow from the fornix reservoir to the tear meniscus [10] (Fig. 14.1). Because CCh is highly prevalent [11] and age-dependent [11, 12], it can be a common but overlooked cause of ocular discomfort and may associate with tear instability that clinically mimics dry eye [reviewed in [4]]. Because both CCh and aqueous tear-deficient (ATD) dry eye are common in older populations, their coexistence makes it difficult to discern genuine dry eye from dry eye secondary to CCh. Furthermore, the aforementioned pathogenesis of CCh in perturbing the tear spread strongly suggests that the severity of dry eye can be aggravated by CCh. Herein, we illustrate three cases to highlight our proposed rationale in laying down a practical and logical algorithm for managing ocular surface diseases that manifest CCh.

Case #1

A 49-year-old female presented with ocular irritation, blurry vision, dryness, and tearing in both eyes for 6 years. She had been unsuccessfully treated with various topical concomitant medications including artificial tears, lubricants, conventional steroid, and cyclosporine. Her nasolacrimal drainage system was patent as confirmed by multiple times of probing and irrigation. Upon first office visit, slit lamp examination showed redundant conjunctival tissue interposed between the lid margin and the eye globe and an uneven but high tear meniscus (Fig. 14.2a). Her eyelids showed intact closure and blinking. All four puncta were swollen by inflammation and collapsed into a slit-like open appearance (Fig. 14.2b). Her tarsal and conjunctiva were diffusely injected (Fig. 14.2c, d).

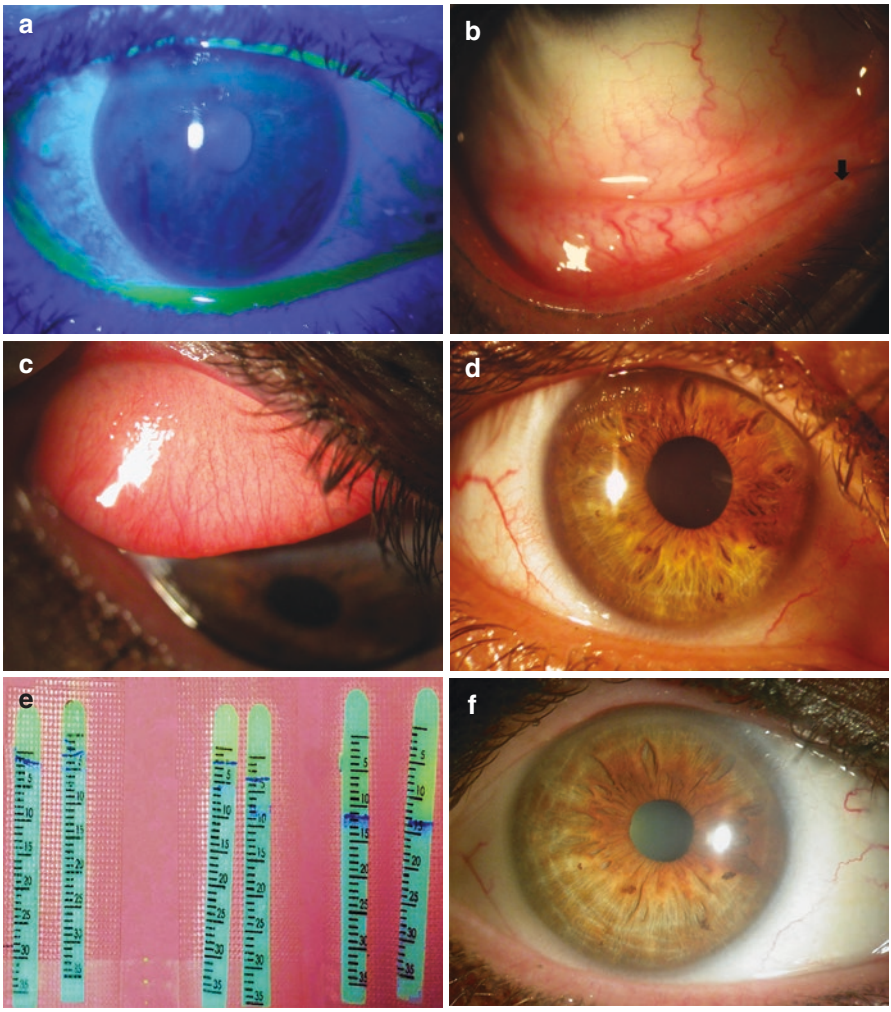

Fig. 14.2 Case #1. Although CCh is highlighted by conjunctival folds clinically resulting in a discontinuous tear meniscus (**a**), it can also be accompanied by swollen puncta (**b**, *arrow*), tarsal papillary reaction (**c**), and conjunctival injection most notable at above the lid margin (**d**), suggesting chronic ocular surface inflammation. FCT showed delayed tear clearance (**e**). After surgery, this eye recovered smooth, non-inflamed conjunctival surface (**f**), continuous tear meniscus (**g**), and non-swollen puncta (**h**, *arrow*). Repeat FCT revealed improved tear clearance but same low basal wetting length, i.e., 2 and 3 mm for OD and OS, respectively, for the first two pairs of strips (**i**). To resolve the remaining symptoms which were due to ATD dry eye, punctal occlusion by plug was performed (**j**)

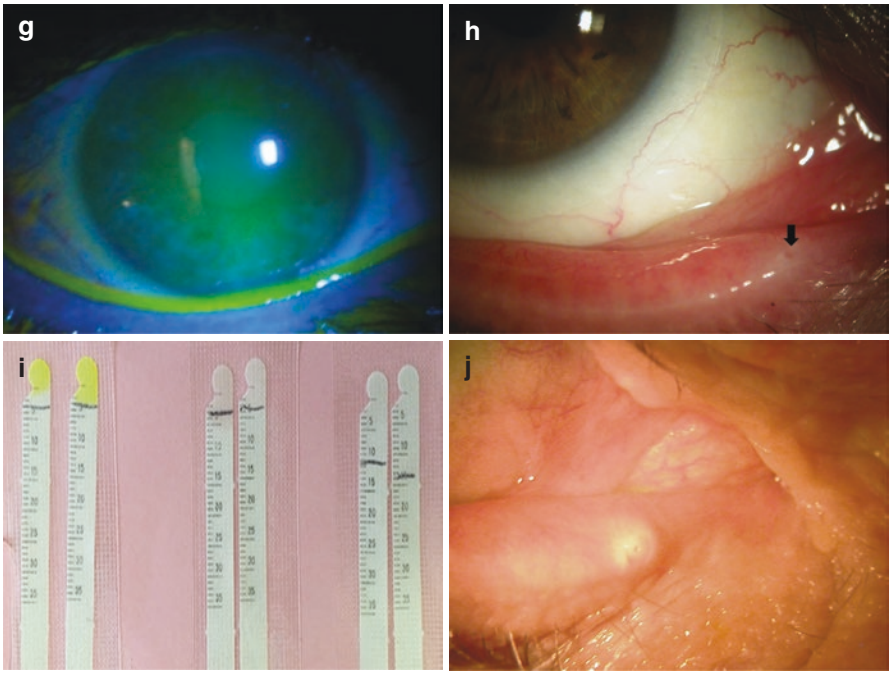

Fig. 14.2 (continued)

Why Did This CCh Patient Complain of Tearing While There was Patent Nasolacrimal Drainage?

The complaint of tearing is most likely resulted from delay tear clearance (DTC). Given that the aqueous tear fluid is cleared through punctum by the eyelid blinking pumping force and stable tear flow, DTC can arise from punctal occlusion, interference with the tear flow, or deficiency of any element in the hydrodynamic reflex. Nevertheless, DTC can also develop in cases of severe ATD to allow the tear meniscus to drop below the level discontiguous to the punctum [13]. Taken together, tear clearance is the convergent point of both hydrodynamic and compositional factors [1]. DTC also occurs when mucosal inflammation and swelling of the ocular surface impose a functional block of tear clearance [14]. One common cause of such mucosal swelling is chronic ocular surface inflammation inflicted by CCh. Conjunctival folds in CCh potentially can also block the punctum [7] besides interrupting the continuity of the tear meniscus [4, 7, 8], interfering with the tear flow from the fornix to the tear meniscus [10] and depleting the fornix tear reservoir [9]. DTC also aggravates ocular surface inflammation, which can then aggravate CCh because DTC also increased inflammatory cytokines in tear levels to upregulate expression of MMPs by CCh fibroblasts [15, 16]. Because DTC is worse during sleep due to the lack of blinking, inflammatory symptoms caused by CCh tend to be worse in the morning upon awakening and are frequently associated with swollen puncta in CCh [8, 14].

How Can One Measure Tear Clearance?

Measurement of tear clearance is indeed a useful clinical test to assess the status of ocular surface defense. The ocular tear volume is traditionally assessed by the Schirmer test, which is known to have a wide range of false positive and negative rates as well as lack of standardization [17–19]. Following application of 5 μL of Fluroress® (Akorn Inc., Abita Springs, LA, USA), which contains anesthetics, fluorescein clearance test (FCT) was performed by applying the Schirmer paper strip for 1 min every 10 min for a total of 30 min, at which time nasal stimulation is performed. Hence, FCT at one setting can determine basal tearing, reflex tearing, and tear clearance [14]. Because the paper strip is bent over the tear meniscus to reach the tear reservoir in the fornix, the measured tear volume conceivably includes both the tear meniscus (the second compartment) and fornix reservoir (the third compartment). The diagnosis of ATD is based on the wetting length of less than 3 mm for the first two sets (10th and 20th min) for measurement of the basal tear volume. The high variability of the Schirmer test [18] is minimized by using FCT, which avoids unquantified anesthetic drop size and reduces the contact of lashes from 5 min for Schirmer I test to 1 min. Tear fluorescence measurements by FCT using the naked eyes (Fig. 14.3) correlate well with fluorophotometry [20]. The intensity of fluorescein dye fades with time under the blue light. Tear clearance is regarded normal if fluorescein becomes invisible to the naked eye after 10 min (i.e., the first pair of strips). In contrast, DTC is defined as fluorescein present on the Schirmer strips at or beyond 20 min (i.e., the second and third pair of strips). FCT can help detect clinical and subclinical DTC which has been overlooked [14].

FCT revealed low basal wetting length for 10th and 20th min on both eyes. In addition, the tear clearance that was also delayed as evidenced by the dye could still be detected by the strip taken at the 20 min interval and later (Fig. 14.2e).

How Can One Treat CCh Patients with DTC?

As DTC aggravates ocular symptoms by precipitating accumulation and prolonging the contact of intrinsic irritative stimuli in the ocular surface, elimination of such intrinsic irritative stimuli/inflammation is the first step [13]. Medicamentosa with accumulation of intrinsic toxic topical medications that contain preservatives is a common overlooked etiology for the eyes with clinically apparent inflammation. It is likely that medicamentosa can precipitate DTC, which in turn can perpetrate medicamentosa in a vicious cycle. Application of topical preservative-free steroid such as methylprednisolone or dexamethasone has shown success in tear clearance improvement by breaking the aforementioned vicious cycle [14].

Under the impression of CCh and ATD with DTC, non-preservative dexamethasone 0.1% was prescribed to control inflammation. Two weeks after treatment, symptoms were partially relieved.

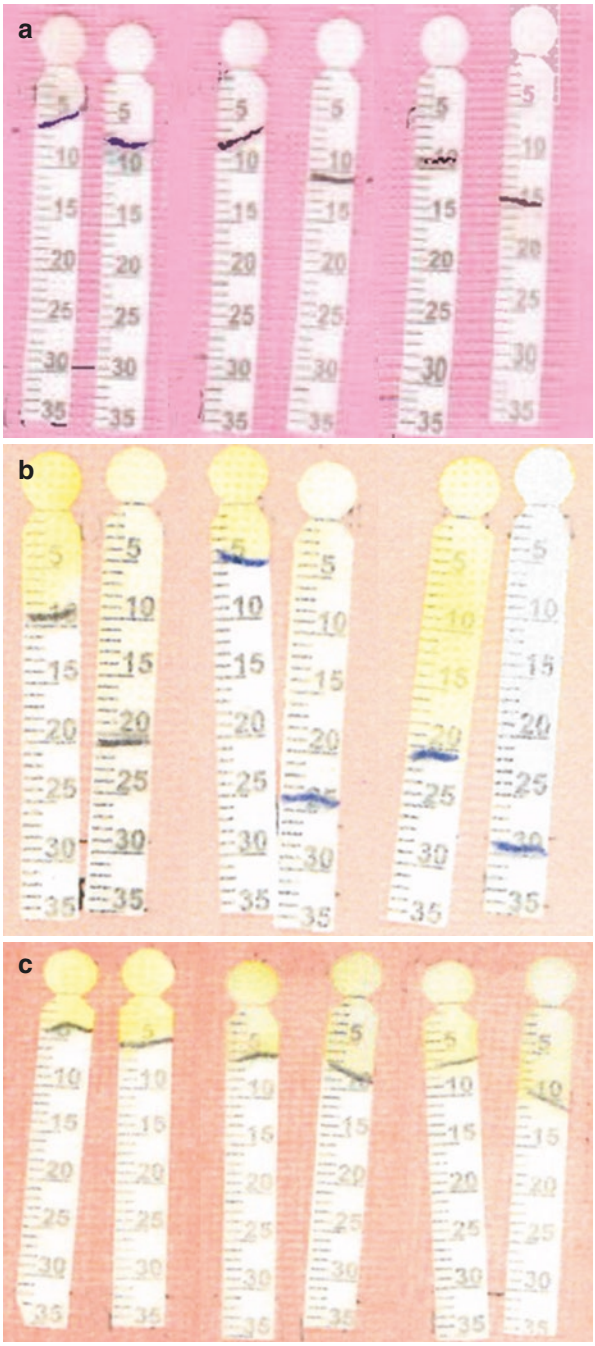

Fig. 14.3 Representative FCT Examples for different clinical diagnosis. (**a**) Normal basal tearing, clearance, and reflex tearing. Unilateral (**b**) and bilateral (**c**) delayed tear clearance as evidenced by fluorescein present in the right eye (**b**) or both eyes (**c**) beyond 20 min

What is the Pathogenesis of CCh?

Although there are conjunctival wrinkles, the underlying pathology of CCh does not reside in the conjunctiva but rather owes to dissolution of the Tenon capsule, which functions as a "carpet pad" to anchor the conjunctiva "carpet" to the underlying sclera "floor." In CCh, excessive degradation of the extracellular matrix MMPs under inflammatory cytokines results in Tenon's capsule degeneration [5, 16]. The lack of healthy Tenon's capsule hence leads to loose and wrinkled conjunctiva, prolapsed fat (Fig. 14.1c), and subconjunctival hemorrhage as the underlying blood vessels can be avulsed during conjunctival movement in the absence of the Tenon capsule.

What Will be the Next Step for Managing Symptomatic CCh After Inflammation Control?

Once ocular inflammations are controlled, restoration of fornix reservoir is the key to achieve effective tear spread for symptomatic CCh patients. Understanding of the underlying pathology of CCh helps formulate surgical procedure aimed at restoration of the fornix tear reservoir. This "reservoir restoration procedure" (Fig. 14.4) includes the following three key steps: (1) significant rearrangement of conjunctiva by recessing and anchoring it from the limbus to the fornix, (2) thorough removal of degenerated Tenon's capsule, and (3) replacement of the Tenon and the conjunctival tissue by two separate layers of cryopreserved amniotic membrane (AM) (BioTissue, Miami, FL) to help prevent recurrence and expedite patient's recovery.

Because of the residual symptoms persisted despite maximal medical treatments, the patient received fornix reconstruction surgery with conjunctival recession and AM transplantation. After surgery, the epithelial defects created by the denuded AM were rapidly epithelialized within 3 weeks. Her eyes recovered smooth, non-inflamed conjunctival surface (Fig. 14.2f) with restoration of fornix reservoir and tear meniscus (Fig. 14.2g) and non-swollen puncta (Fig. 14.2h). However, she noted partial but not complete relief of symptoms including blurry vision, dryness, tearing, and conjunctival redness. Repeat FCT revealed improved tear clearance but same wetting length (Fig. 14.2i).

Why Not Just Cut Off (Resect) or Cauterize the Redundant Conjunctiva?

As of now, a number of surgical procedures have been advocated to treat CCh such as scleral fixation suture [21], crescent bulbar conjunctival excision with direct closure [7], resection combined with inferior peritomy and radial relaxing

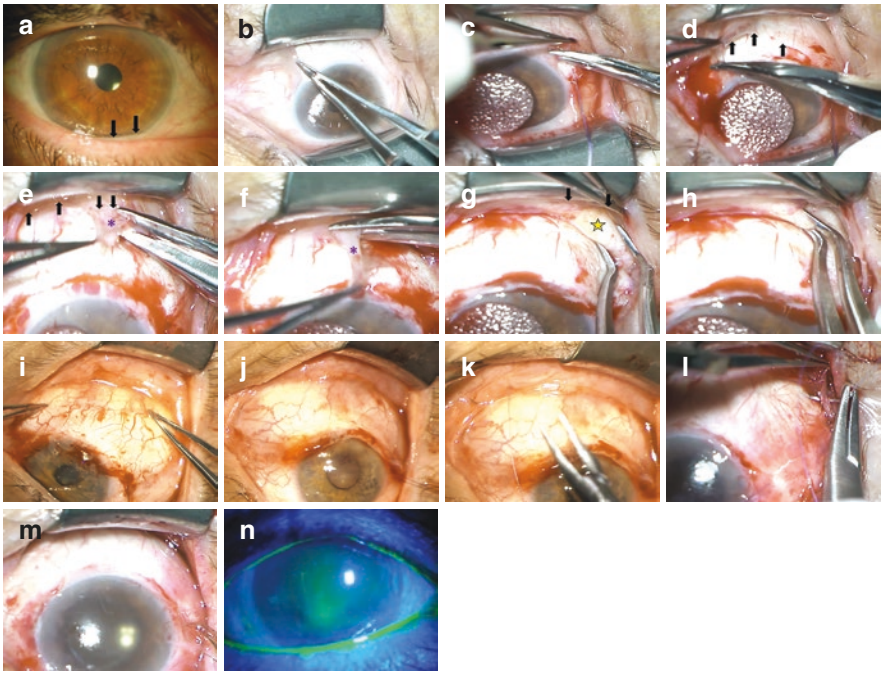

Fig. 14.4 Surgical steps of reservoir restoration procedure. Poor conjunctival adhesion to the sclera from dissolution of the Tenon capsule is noted as evidenced by easy separation of the conjunctiva from the sclera simply by forceps grabbing (**a**, *arrow*, **b**). After using several drops of epinephrine 1/1000 for hemostasis and 2% lidocaine gel for anesthesia, a traction suture made of 7-0 Vicryl is placed 2 mm posterior to the limbus at the 3 and 9 o'clock position and used to rotate the eye upward. An arc-like conjunctival peritomy is created 1–2 mm posterior to the limbus in the area of CCh (**c**) and extends to remove pinguecula, if present. Rearrangement of conjunctiva by recessing (**d**, *arrow*) from the limbus to the fornix. The abnormal Tenon's capsule (*asterisk*) that is distributed under the overlying recessed conjunctival epithelial tissue (*arrow*) and adherent over the bare sclera (**e**). The abnormal Tenon's capsule (*asterisk*) is grabbed and dissected off from the overlying conjunctival epithelial tissue and thoroughly removed by a sharp scissors (**f**). The recessed conjunctiva (*arrow*) is lifted up by a forceps to identify the prolapsed fat (*star*) that is distributed in the fornix (**g**) and cauterized to create a gap (**h**) for prevention of fat herniation through fornix. Two separate layers of cryopreserved AM are laid down to replace Tenon (**i**) and the conjunctival tissue (**j**) to help prevent recurrence and expedite patient's recovery. The conjunctival is recessed to anchor at the fornix with 8-0 Vicryl (**k**, **l**). Fornix deepening reconstruction with conjunctival recession and AM transplantation restores fornix tear reservoir (**m**) to help replenish tear meniscus and preocular surface tear film in symptomatic CCh patient (**n**)

incisions [22], excision with AM transplantation [23, 24], cauterization/laser coagulation with or without excision [25, 26], and subconjunctival fibrin sealant followed by excision [27]. Most of these procedures focus on elimination of conjunctival folds close to the tear meniscus—but do not address fornix reconstruction. While these resection procedures may be effective in many cases,

reconstruction and deepening of the fornix is best done with conjunctival "recession" not "resection." In addition, complications such as scar formation and fat prolapse might be attributed to the aforementioned surgical techniques if not performed appropriately.

What is the Clinical Significance if Repeat FCT Shows No Change in the Basal Wetting Length After CCh Surgery?

Changes of basal tear volume after fornix reconstruction help discern genuine or concomitant ATD to clarify logical step in the clinical management algorithm for dry eye [9, 13]. Genuine ATD dry eye is identified by the unchanged basal wetting length despite restoration of fornix tear reservoir after CCh surgery. If there are still residual non-resolving dry eye manifestations, treatment should be directed to compositional deficiency dry eye [13]. In this regard, the conventional treatment for ATD dry eye can be managed more effectively by punctal occlusion, which remains the mainstay of managing ATD dry eye when the application of artificial tears reaches a maximum certain daily frequency. Because punctal occlusion invariably delays tear drainage/clearance, it can prolong the beneficial effect of topical artificial tears for ATD but potentially cause toxicity to the ocular surface as a result of prolonged retention of preservative-containing artificial tears. Hence, it is advised that non-preserved tear substitutes are preferred, because CCh can aggravate DTC, which is why surgical correction of CCh is performed before punctal occlusion is contemplated.

Plugs were placed in her inferior puncta (Fig. 14.2j). She noted significant complete resolution of all symptoms. Such improvement was accompanied by reduction of conjunctival hyperemia and improvement of the visual acuity of 1 Snellen line.

Case #2

A 54-year-old male noted irritation and some "collarets" in lash roots in both eyes for months. There were no associated symptoms such as photophobia, blurry vision, dryness, and tearing. Upon presentation, his vision was 20/20 with mild conjunctiva and eyelid injection (Fig. 14.5a, b). Slit lamp examination revealed cylindrical dandruffs located at the base of the lashes. Lashing sampling confirmed demodex blepharitis. There was redundant conjunctival tissue interposed between the lid margin and the eye globe. Obliterated tear meniscus was shown with fluorescein staining (Fig. 14.5c, d). FCT showed basal wetting length of 5 mm and normal tear clearance as evidenced by the disappearance of the dye in 10 min in both eyes (Fig. 14.5e).

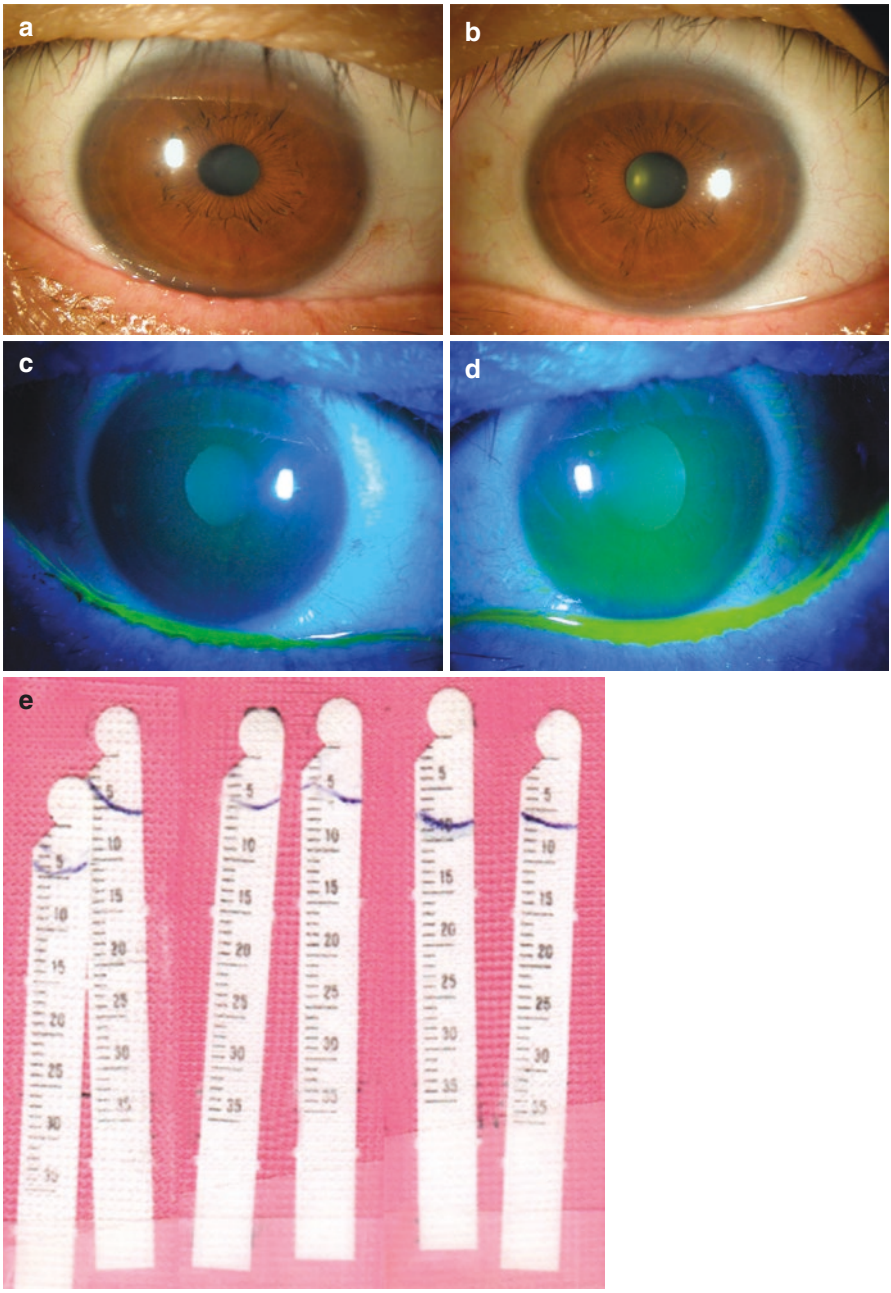

Fig. 14.5 Case #2. Bilateral mild conjunctival and eyelid injection of the right eye (**a**) and left eye (**b**) and CCh as evidenced by interference with the tear meniscus by redundant conjunctival tissue (**c**, **d**) in this asymptomatic patient with CCh. FCT shows the basal wetting length of 5 mm and normal tear clearance as evidenced by the disappearance of the dye in 10 min (**e**)

How Can One Discern Whether CCh is the Cause of the Said Eye Irritation?

As recently reported [10], we propose to measure the speed of recovery of the tear meniscus after depletion as a way to discern whether CCh is the cause of patient's ocular irritation. To do so, we first instill a 5 μL fluorescein drop into the inferior fornix via a pipette. [One can also do so by applying fluorescein drop by wetting a fluorescein strip with saline.] Under slit lamp examination with a blue light, the fluorescent inferior tear meniscus is then depleted by using a capillary tube. [One can also deplete it by touching with a dry Weck-Cel (Micrsponge™, Alcon®).] Immediately afterward, the time in sec is measured to see the recovery of the fluorescent tear meniscus (Fig. 14.6a–c). Using this technique, we have reported that the tear reservoir in the fornix rapidly replenishes the meniscus under normal circumstances but not in CCh patients. Without eyelid blinking, the original tear meniscus

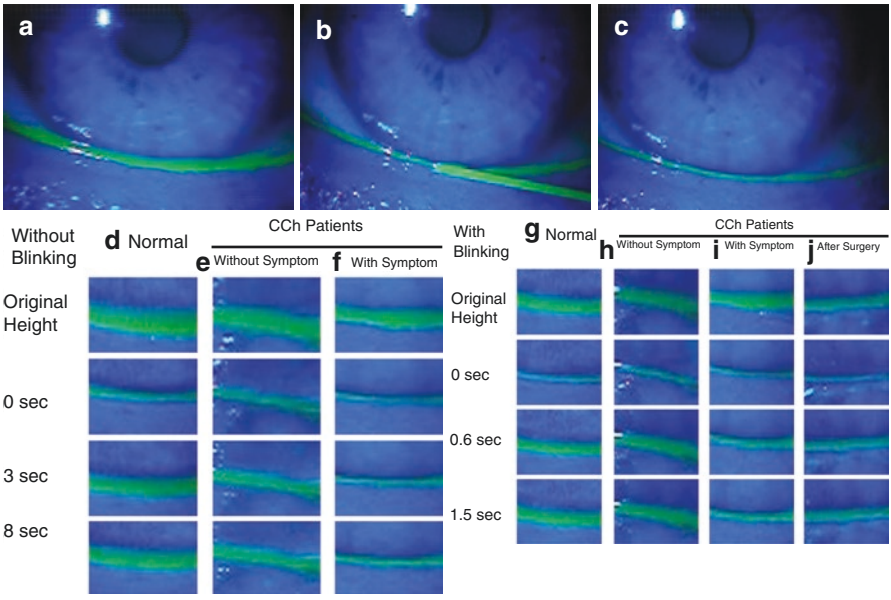

Fig. 14.6 Effect of blinking for fornix tear replenishment in asymptomatic CCh. Original tear meniscus height (**a**) is depleted by capillary tube (**b**, **c**). Fornix rapidly replenishes the meniscus back to normal height in normal people (**d**) but not in CCh patients regardless of whether symptoms present (**e**, **f**). However, one blink was sufficient to recover the tear meniscus back to normal height in asymptomatic CCh patients (**h**) as rapidly as normal subjects (**g**), whereas the tear meniscus height remained low in symptomatic CCh (**i**). CCh patients can be asymptomatic as blinking is an effective compensatory mechanism to facilitate the tear flow from the fornix to the meniscus and consequently the preocular tear film. Intriguingly, the meniscus height of symptomatic CCh after blinking could be facilitated back to normal level by restoration of fornix tear reservoir via reconstruction with conjunctival recession and AM transplantation (**j**) (Taken with permission from Huang et al. [10])

height can be recovered by fornix replenishment in <3 s in patients without CCh (Fig. 14.6d). In patients with CCh with symptoms, the meniscus recovery was significantly retarded, i.e., not recovered in 8 s (Fig. 14.6f). In patients with CCh but without symptoms, the recovery was intermediate (Fig. 14.6e). Importantly, eyelid blinking is the key compensatory mechanism to facilitate the tear flow from the fornix reservoir to the tear meniscus. Hence, upon one eyelid blinking, the meniscus recovery is significantly facilitated in patients with CCh without symptoms (asymptomatic) (back to normal tear meniscus height as in Fig. 14.6g, h) but not in CCh patients with symptoms (symptomatic) (Fig. 14.6i). Intriguingly, the meniscus height of symptomatic CCh after blinking could be facilitated back to normal level by restoration of fornix tear reservoir via reconstruction with conjunctival recession and AM transplantation (Fig. 14.6j). Collectively, blinking is an effective compensatory mechanism to distinguish CCh severity and identify asymptomatic CCh. Importantly, restoration of the tear reservoir by fornix reconstruction is an effective treatment for symptomatic CCh to ensure a continuous supply of tears from the reservoir to the tear meniscus and the preocular tear film.

Why Are some CCh Patients Asymptomatic?

The aforementioned clinical measurement of how a depleted tear meniscus is recovered after one eyelid blinking clearly demonstrates that CCh patients can be asymptomatic if the fornix tear reservoir is not completely obliterated and if the blinking remains effective in facilitating the tear flow from the fornix to the meniscus and consequently the preocular tear film. In contrast, patients with CCh can become symptomatic if the severity of CCh obliterates the fornix tear reservoir. Under this scenario, blinking although shortens the interblink interval, it still fails to replenish the already depleted tear meniscus. Given that blinking can help restore obliterated tears in the tear meniscus in asymptomatic CCh, it is also clinically important to evaluate whether some CCh patients can become symptomatic if blinking is ineffective.

Case #3

A 52-year-old male presented with ocular irritation, dryness, burning, gritty sensation, blurred vision, and photophobia in both eyes for 5 years. His past history did not disclose any associated systemic disease such as Sjögren disease, rheumatoid arthritis, or systemic lupus erythematosus. He remained symptomatic despite conventional medical therapies including topical concomitant medications (artificial tears, lubricants, steroid, and cyclosporine), bandage contact lens, or punctal occlusion. Clinical examination disclosed with ATD and CCh. The diagnosis of ATD was based on the basal wetting length of less than 3 mm at the 10th and 20th min according to FCT.

Why Did This Patient Find No Relief from Conventional Eye Drops in Dry Eye Caused by CCh?

The loose conjunctiva in CCh patients occupies the space not only in the tear meniscus but also obliterates the fornix tear reservoir to interfere tear spread from the fornix to the tear meniscus (Fig. 14.1c) [4, 7, 8, 10]. The fornix tear reservoir is the primary compartment for an eye to hold tears, i.e., estimated to be responsible for at least 50% of the total tear volume [28]. The finding that the fornix tear reservoir is obliterated by CCh [9] explains why the eye failed to hold the artificial tears. Consequently, the tears in the meniscus and the preocular tear film cannot be replenished by the fornix tear reservoir through blinking. Hence, artificial tears alone are incapable of maintaining a stable tear film.

Upon presentation, his vision was 20/30 for both eyes. His eyelids had normal blinking and closure.

What Clinical Work Up Should You Perform to Discern Whether the Eye Suffered from ATD, CCh, or Both?

The close association between CCh and ATD has been recognized [29, 30]. It has been reported that the risk of developing ATD increases with severity of CCh, suggesting that CCh itself may be a cause leading to ATD dry eye [30]. However, because both CCh and ATD dry eye are prevalent in aged populations, it is also challenging to discern ATD that may exist as an independent disease. Clinically, dry eye caused by CCh can be differentiated from dry eye caused by ATD by history taking focused on inquiring the dry eye symptoms to see if they are worsened by any specific diurnal relationship, gaze, blinking, or punctal occlusion [8]. Ocular irritation is worsen as the day progresses due to progressive exposure and desiccation in genuine ATD but tends to remain the same throughout the day in dry eye caused by CCh. CCh patients usually complain of blurry vision and pain, which are aggravated by increased conjunctival folds in downgaze during reading. On the contrary, the interpalpebral exposure zone that increases from downgaze to upgaze explains why ATD dry eye is worse in upgaze, especially when working with a computer screen. Furthermore, frequent blinking results in accumulation of conjunctival folds to the 6 o'clock position which further aggravates dry eye symptoms in CCh patients. In contrast, increasing blinking shortens the interblink interval, invariably stabilizes the tear film, and improves ATD dry eye. An additional test which is sometime helpful is that in some patients with symptomatic CCh, manually retracting the lower lid can provide some relief of their symptoms (perhaps this maneuver temporarily increases the lower fornix tear reservoir). Punctal occlusion invariably delays tear drainage and prolongs the beneficial effect of topical artificial tears for ATD. However, it further retards tear clearance in the tear meniscus and fornix that is already compromised by CCh and leads to exacerbation of CCh mimetic dry eye

symptoms. The use of rose bengal may highlight ocular surface epithelial cells that exhibit a mucin-deficient state in the interpalpebral exposure zone for ATD but in the non-exposure zone for CCh. By obliterating tear meniscuses, CCh destabilizes the tear film immediately above the redundant conjunctival fold in a linear pattern of CCh area (inferior to the exposure zone).

His symptoms were worse in the morning upon awakening and remained the same throughout the day. They were aggravated with blinking and down-gaze during reading but not watching computer or television. His symptom severity was graded 25 (moderate) based on Ocular Surface Disease Index (OSDI) score. Prior maximal conventional medical treatments including artificial tears, lubricants, steroid and cyclosporine, and bandage contact lens on both eyes failed to relieve his symptoms. Symptoms persisted and were worsen with punctal occlusion. Slit lamp examination showed nasal and temporal redundant conjunctival tissue interposed between the lid margin and the eye globe impeded the formation of a proper and smooth tear meniscus (Fig. 14.7a). Fluorescein staining showed the obliterated tear meniscus but no superficial punctuate keratitis on corneal surface (Fig. 14.7b). Rose bengal staining could not be seen on the exposure zone of the central cornea. FCT revealed the basal wetting length of 2 mm (Fig. 14.7c) in both eyes.

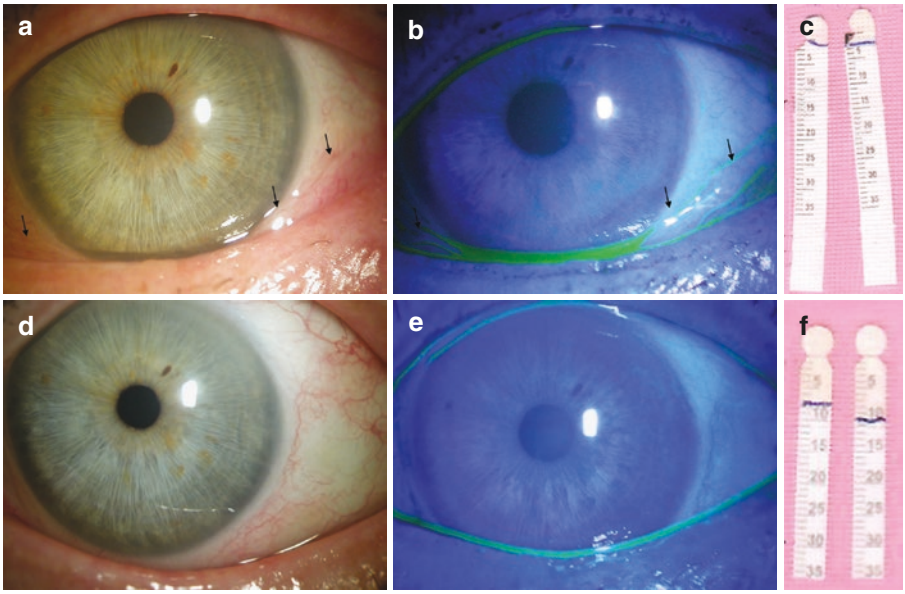

Fig. 14.7 Case #3. Redundant conjunctival folds (*arrows*) interposed between the lid margin and the eye globe obliterated the tear meniscus (**a**, **b**). FCT showed a low basal wetting length suggestive of dry eye before surgery (**c**). One month after reservoir restoration by AM transplantation, the eye regained a smooth, quiet, and non-inflamed bulbar conjunctiva (**d**) and a continuous tear meniscus (**e**). Repeat FCT showed normal basal wetting lengths (**f**), indicating that prior dry eye was secondary to CCh but not caused by ATD

Because of persistent symptoms despite the maximal medical treatments, the patient received CCh surgery with an attempt to alleviate the symptoms and restore the ocular surface health. The operative technique includes excision of mobile and degenerated Tenon's capsule, fornix deepening reconstruction with conjunctival recession, and cryopreserved AM transplantation (secured with the use of fibrin glue) under topical anesthesia (Fig. 14.4). After surgery, the patient received ofloxacin 0.3% eye drops three times daily and non-preservative dexamethasone 0.1% eye drops four times daily for 3 weeks, tapering off within 2 weeks. All epithelial defects created by the denuded AM were rapidly epithelialized within 3 weeks (Fig. 14.7d, e). Complications such as fat prolapse or scarring-induced entropion of the lower lid, retraction of the lower fornix, and restricted motility were not found. He experienced a total relief of symptoms. Furthermore, he also exhibited improved visual acuity of 1 Snellen line for both eyes. His eyes recovered smooth, wet, white, non-inflamed conjunctival surface being symptom-free in 1 month. Repeat FCT revealed increased basal wetting length from 2 to 7 and 10 mm for the right and left eye, respectively (Fig. 14.7f).

Why Did FCT Show the Improvement of Basal Wetting Length After AMT in CCh?

Under normal circumstance, the fornix tear reservoir is responsible for delivering tear fluids to the tear meniscus by blinking [10]. In CCh, the fornix tear reservoir is obliterated by loose and wrinkled conjunctiva and prolapsed fat due to degenerated Tenon's capsule [5, 9, 16]. The occupied and depleted tear reservoir conceivably results in a low basal wetting length, no different from ATD dry eye. Restoration of the fornix tear reservoir is achieved by fornix deepening reconstruction with conjunctival recession and AMT. This explains why the postsurgical basal wetting length can be improved in dry eye caused by CCh, i.e., resulting in the disappearance of ATD dry eye secondary to CCh. Using AMT to achieve reservoir restoration, we can differentiate dry eye caused primarily by ATD or secondarily by CCh [9]. Elimination of dry eye caused by CCh first by AMT also allows us to treat dry eye caused by ATD more effectively later on. That is also why we advocate AMT to restore tear reservoir prior to managing ATD dry eye as a more logical and practical management algorithm for dry eye [13].

Summary

In summary, the pathogenesis of CCh involves dissolution of the Tenon capsule that allows the conjunctiva to fold, which in turn block the tear flow into the nasolacrimal drainage system [7], interrupt the continuity of the tear meniscus [8], obliterate

the fornix tear reservoir [9], and interfere with the flow or tears from the fornix tear reservoir to the tear meniscus [10]. Elimination of intrinsic irritative stimuli/inflammation by topical preservative-free dexamethansone or methylprednisolone is the initial treatment for DTC-associated CCh. After differentiating dry eye caused by CCh from that caused by ATD and determining that CCh is the cause of ocular irritation, restoration of the fornix tear reservoir by fornix reconstruction with conjunctival recession and AM transplantation is an important surgical procedure to resolve symptoms and signs in CCh. Punctal occlusion and specific management of genuine ATD dry eye take place after elimination of CCh. In conclusion, fluorescein-based testing of the tear meniscus can help distinguish symptomatic CCh from genuine ATD (independent of CCh) and allow physicians to adopt an effective treatment algorithm to restore the integrity of the ocular surface.

Acknowledgements *Financial Disclosure*: Dr. Tseng has obtained a patent for the method of preparation and clinical uses of amniotic membrane and has licensed the rights to TissueTech, Inc., which procures and processes, and to Bio-Tissue, Inc., which is a subsidiary of TissueTech, Inc., to distribute cryopreserved amniotic membrane for clinical and research uses.

Funding/Support: This study was supported in part by an unrestricted grant from Ocular Surface Research Education Foundation, Miami, FL.

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