EXTENDED REPORT

Clinical characteristics of conjunctivochalasis with or without aqueous tear deficiency

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Aim: To show characteristic ocular surface findings caused by conjunctivochalasis (CCh) in dry eye patients with or without aqueous tear deficiency (ATD).
Design: Comparative non-interventional cases.
Patients and methods: Clinical data of five ATD patients without CCh (group A), eight CCh patients with ATD (group B), and eight CCh patients without ATD (group C) were retrospectively reviewed. Presence or absence of CCh was determined by fluorescein staining to outline tear meniscus and conjunctival folds with an enhancing filter. Dry eye symptoms, history of subconjunctival haemorrhage, meibum expression, tear break up time, fluorescein and rose bengal staining, and fluorescein clearance test, and other abnormal ocular surface findings were measured.
Results: CCh patients were significantly older ($p = 0.001$). In pure ATD, the principal symptom of dryness became worse as the day progressed. In contrast, blurry vision, burning sensation, and dryness became worse during reading in all CCh patients ($p = 0.0008$) or worse in the morning upon awakening in the majority patients with CCh only ($p = 0.02$). Besides the interpalpebral exposure, which was noted in ATD, positive fluorescein or rose bengal staining was noted in the redundant conjunctival folds and the non-exposure zone in CCh ($p = 0.0008$). Redundant conjunctival folds were present in both lower and upper bulbar conjunctiva, obliterating both lower and upper tear meniscuses, and spatially correlated with anterior migration of the mucocutaneous junction in CCh. Delayed tear clearance was significantly more prevalent in CCh than ATD ($p = 0.0008$). Vigorous blinking worsened in CCh but not in ATD ($p = 0.0008$). Lacrimal puncta were swollen in groups B and C, but not in group A ($p = 0.04$).
Conclusions: CCh is not restricted to the lower bulbar conjunctiva, and contributes to pathogenesis of dry eye by obliterating both lower and upper tear meniscus, causing unstable tear film and by creating delayed tear clearance. Dry eye symptoms were worsened by downgaze during reading and by vigorous blinking. Other characteristic signs including subconjunctival haemorrhage, swollen puncta, anterior migration of the mucocutaneous junction, and patterns of dye staining also help distinguish dry eye associated with CCh from that caused by ATD alone.

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Conjunctivochalasis (CCh) defined as ''a redundant conjunctiva typically located between the eyeball and the lower eyelid'' is not uncommon but often overlooked as a normal ageing variation. CCh tends to be bilateral and can be localised in the nasal, central, or temporal part of the lower eyelid margin. Patients with CCh may be asymptomatic, but may manifest dry eye by aggravating a pre-existing unstable tear film caused by aqueous tear deficiency (ATD) at a mild stage, cause episodic tearing by impeding the tear clearance at a moderate stage, and induce subconjunctival haemorrhage and exposure at a severe stage.[1] The association of CCh with keratoconjunctivitis sicca (KCS) was first described by Rieger in 1990[2] and Grene in 1991,[3] but more thoroughly explored by Höh *et al* in 1995,[4] who noted the risk of developing KCS increases with severity of CCh. It remains unclear whether KCS caused by ATD alone can be distinguished from KCS caused by CCh, and whether CCh induced dry eye presents unique clinical features even when it is associated with ATD.

Although the exact pathogenic mechanism of developing CCh remains unclear, we postulated that conjunctival looseness might result from excessive degradation of the extracellular matrix.[1] Our hypothesis was supported by our recent study showing that CCh fibroblasts produce more matrix metalloproteinase type 1 (MMP-1) and type 3 (MMP-3) than normal conjunctival fibroblasts in culture.[5] Because such overexpression of MMP-1 and MMP-3 is further upregulated by inflammatory cytokines such as interleukin 1 (IL-1) and tumour necrosis factor α (TNFα),[6] we speculated that CCh patients should carry signs of ocular surface inflammation. Because we have reported that the tear clearance is delayed in eyes with ocular surface inflammation,[7] and others have reported that delayed tear clearance is associated with elevated tear levels of gelatinase and IL-1 in rosacea patients,[8] we wondered whether delayed tear clearance may help distinguish dry eye with CCh from dry eye without CCh. Liu proposed that inferior loose conjunctiva interferes with tear clearance resulting in epiphora.[9] Nevertheless, it remains unclear whether CCh also disrupts or obliterates the upper tear meniscus. For all these reasons, we have undertaken a retrospective review of clinical data gathered from CCh patients with or without ATD and compared them with those obtained from ATD patients without CCh.

Abbreviations: ATD, aqueous tear deficiency; CCh, conjunctivochalasis; FCT, fluorescein clearance test; KCS, keratoconjunctivitis sicca; LTD, lipid tear deficiency; MGD, meibomian gland dysfunction; MMP, matrix metalloproteinase; BUT, tear break up time

MATERIALS AND METHODS

This study was approved by the Institutional Review Board of the Baptist Hospital of Miami and South Miami Hospital to retrospectively review the clinical data of five ATD patients without CCh (group A), eight CCh patients with ATD (group B), and eight CCh patients without ATD (group C). The diagnosis of CCh was based on the criteria previously summarised by Meller and Tseng.[1] The diagnosis of ATD was based on the wetting length of the Shirmer test of less than 3 mm for 1 min according to the fluorescein clearance test (FCT).[7] The diagnosis of meibomian gland dysfunction (MGD) associated with lipid tear deficiency (LTD) was based on meibomian expression as previously reported.[10] None of these patients had any evidence of ocular infection or abnormal blinking.

From complete ophthalmic histories and examination, we extracted the following data for analysis. We quantified dry eye complaints as being worse in the morning upon awakening (AM), as the day progressed (PM), or during reading. From the past ocular history, we asked whether there was spontaneous subconjunctival haemorrhage in the past 6 months. The tear break up time (BUT) was measured in a conventional manner.[7 10] The pattern of fluorescein staining and subsequent 1% rose bengal staining was recorded according to whether it was located at the interpalpebral exposure zone or the non-exposure zone as previously reported.[10] We also noted whether these two dyes stained the exposed redundant conjunctiva and the adjacent mucocutaneous junction. To document obliteration or disruption of the tear meniscus, we photographed fluorescein staining with and without a Kodak Wratten yellow No 12 filter (Kodak, Rochester, NY, USA). Furthermore, we documented whether redundant conjunctival folds extended to the 6 o'clock position after vigorous blinking for a minimum three times. In addition, we documented the presence of pinguecula, floppy eye, and swollen lacrimal puncta. All photographs were analysed by two independent masked observers (ES and TK).

Statistical analysis

The comparison of age and BUT between group A patients and those of groups B and C was made by independent *t* test; the rest of the factors were compared using Fisher's exact test using Graph Pad InStat Version 3.05 (GraphPad Software Inc, San Diego, CA, USA). A p value of <0.05 was considered as statistically significant.

RESULTS

Relevant clinical data of each patient are summarised in tables 1 and 2.

Comparison between group A and groups B and C: CCh associated differences

Group A patients were significantly younger than groups B and C ($p<0.001$). All patients in these three groups were symptomatic and complained of dryness, but patients in groups B and C also complained of blurry vision and burning sensation. Symptoms tended to be worse as the day progressed in group A, but became worse during reading in groups B and C ($p<0.001$). History of subconjunctival haemorrhage was not noted in group A, but found in 11 of 16 patients in groups B and C ($p = 0.01$). In group A, two patients (cases 1 and 4) showed positive fluorescein staining and three patients (cases 3, 4, and 5) showed rose bengal staining at the interpalpebral exposure zone. Nevertheless, fluorescein and rose bengal staining were noted in the non-exposure zone, CCh areas, and the adjacent lid margin in all patients in groups B and C ($p<0.0001$, fig 3C and D). MGD with LTD was absent in group A, but present in all patients in groups B and C ($p<0.0001$). The tear meniscus was continuous in group A even if it was low in height (fig 1A), but was interrupted or obliterated by CCh in all patients in groups B and C (fig 1C to F). The altered tear meniscus was spatially correlated with anterior migration of the mucocutaneous junction (fig 2E to H). Puncta appeared normal in group A (fig 3A), but were swollen with elongation and subcutaneous oedema, yielding a configuration resembling ''volcano'' in 13 patients in groups B and C (fig 3B) ($p = 0.002$). Floppy eyelids were found in 12 patients ($p = 0.006$), and pinguecula was observed in three patients from groups B and C ($p = 0.5$, fig 3C and D).

Comparison between group A and group B: how CCh altered ATD dry eye

The mean age of group A was less than group B (32.4 (SD 2.6) years *v* 66 (SD 9.1) years, $p<0.0001$). Dry eye complaints tended to be worse when reading ($p = 0.0008$) and on awakening ($p = 0.02$) in group B. The mean BUT in group B patients was significantly less than that of group A (1.2 (SD 1.1) seconds *v* 2.8 (SD 0.8) seconds, $p = 0.02$). Positive staining with fluorescein and rose bengal at the non-exposure zone and in the CCh area was only noted in group B ($p = 0.0008$), as was MGD with LTD ($p = 0.0008$). Delayed tear clearance was noted in all patients in group B, but only two patients in group A ($p = 0.03$).

All eight patients except for one (case 8) showed multiple conjunctival folds distributed in temporal, nasal, and central (6 o'clock) bulbar aspects of both lower and upper lids (fig 1B to D). The extent of CCh was asymmetrical between the two eyes, and tended to be worse in the temporal bulbar conjunctiva than the nasal bulbar conjunctiva (fig 1C and D). Except for one patient (case 8), CCh in all patients in group B became worse at the 6 o'clock position after vigorous blinking (similar to what is shown in fig 2A *v* B). Although obliteration of the tear meniscus was in general worse in the lower lid, it was clearly observed in the upper lid of all group B patients (fig 1C and D), especially when a special filter was used to visualise the distribution of fluorescein in the tear fluid (see also examples shown in fig 2G and H). CCh was always associated with anterior migration of the mucocutaneous junction in the adjacent area (see also fig 2G and H). As well as the interpalpebral exposure, which was noted in group A, positive fluorescein or rose bengal staining was noted in the redundant conjunctival folds and the non-exposure zone in group B ($p = 0.0008$). Swollen puncta were noted in seven of eight patients in group B (similar to fig 3B), but none in group A ($p = 0.04$) (fig 3A)

Comparison between group B and group C: differences associated with ATD in CCh

There was no difference in mean age between group B and group C (64.2 (SD 9.1) years *v* 66 (SD 9.1) years, $p = 0.6$). There were more complaints of blurry vision in group B (6 of 8 patients) than group C ($p = 0.007$), while complaints of pain were higher in group C (6 of 8 patients) than group B ($p = 0.04$). The mean FCT wetting length in group C was 8 (SD 3.2) mm, which was significantly longer than 2.2 (SD 0.7) mm in group B ($p = 0.0003$). Severe obliteration of the lower tear meniscus and conjunctival folds led to an increase of the meniscus height and epiphora (compare fig 2E and F). With fluorescein staining conjunctival folds were more visible, but became more obvious when an enhancing filter was used (fig 2G and H). Bilateral pingueculae were noted in three patients in group C but in none of group B ($p = 0.02$) (fig 3C and D)

Table 1 Clinical data of patients with or without conjunctivochalasis

Case	Age	Sex	Symptoms	Worst time or condition	History of subconjunctival bleeding	BUT	FCT	DTC	Reflex tearing	FL	RB	MGD-LTD
Group A												
1	32	M	Dryness	PM	No	4	2	Absent	Present	Positive	Negative	Absent
2	32	M	Dryness	PM	No	2	1	Present	Present	Negative	Negative	Absent
3	36	M	Dryness	PM	No	2	2	Absent	Present	Negative	Positive	Absent
4	31	F	Dryness	PM	No	3	1	Present	Present	Positive	Positive	Absent
5	31	F	Dryness	PM	No	3	1	Absent	Present	Negative	Positive	Absent
Mean (SD)	32.4 (2.6)					2.8 (0.8)	1.5 (0.5)					
Group B												
6	61	F	Dryness	PM, reading	Yes	3	2	Present	Absent	Positive	Positive	Present
7	82	F	Blurry vision	AM, reading	Yes	1	3	Present	Present	Positive	Positive	Present
8	73	M	Blurry vision, burning sensation	AM, reading	No	2	3	Present	Present	Positive	Positive	Present
9	59	F	Blurry vision, burning sensation	AM, reading	No	0	1	Present	Absent	Positive	Positive	Present
10	65	M	Blurry vision	PM, reading	Yes	0	3	Present	Present	Positive	Positive	Present
11	53	F	Blurry vision	AM, reading	No	2	2	Present	Present	Positive	Positive	Present
12	64	M	Blurry vision	AM, reading	Yes	0	2	Present	Absent	Positive	Positive	Present
13	71	F	Pain, dryness	AM, reading	Yes	2	2	Present	Present	Positive	Positive	Present
Mean (SD)	66 (9.1)					1.2 (1.1)	2.2 (0.7)					
Group C												
14	59	M	Dryness	PM, reading	Yes	2	5	Present	Present	Positive	Positive	Present
15	52	F	Pain, light sensitivity	PM, light, wind/ reading	Yes	0	8	Present	Present	Positive	Positive	Present
16	67	F	Pain	PM, reading	Yes	2	6	Present	Present	Positive	Positive	Present
17	75	F	Pain	PM, reading	Yes	2	5	Present	Present	Positive	Positive	Present
18	63	F	Pain	PM, reading	No	2	10	Present	Present	Positive	Positive	Present
19	79	F	Pain, dryness	AM, reading	Yes	1	7	Present	Present	Positive	Positive	Present
20	57	M	Pain, dryness	AM, reading	No	2	8	Present	Present	Positive	Positive	Present
21	60	F	Pain, dryness	PM, reading	Yes	3	15	Present	Present	Positive	Positive	Present
Mean (SD)	64 (9.1)					1.7 (0.8)	8 (3.2)					

FCT = fluorescein clearance test measuring the average wetting length (mm) for 1 min with Schirmer strip (with anaesthetic) at 10 and 20 min time points. Reflex = reflex tearing determined by nasal stimulation performed at the 30 min time point (present: wetting length (mm) greater than the above baseline value; absent: wetting length equal to or no greater than the baseline value). DTC = delayed tear clearance measured by FCT (absent: no DTC and is found in normal subjects when dye cleared within 20 min; present: dye clearance was delayed more than 20 min). BUT = tear break up time (seconds). MGD = meibomian gland dysfunction determined by meibum expression (absent: meibum was readily expressed; present: meibum poorly or not expressed by digital compression). PM = as the day progresses; AM = upon awakening; F = female; m = male; FL = fluorescein; RB = rose bengal.

Table 2 Ocular surface changes in patients with or without conjunctivochalasis

Case	CCh location			↑ CCh by fast blink	Staining in CCh	Lid margin erosion	Obliterated tears meniscus			Swollen punctum	Pinguecula*	Floppy eyelids
	T	N	C				T	N	U/L			
Group A												
1	–	–	–	–	–	–	–	–	–	–	–	–
2	–	–	–	–	–	–	–	–	–	–	–	–
3	–	–	–	–	–	–	–	–	–	–	–	–
4	–	–	–	–	–	–	–	–	–	–	–	–
5	–	–	–	–	–	–	–	–	–	–	–	–
Group B												
6	+++	++	+	+	+	+	+++	++	+/++	–+	–	–
7	+++	++	+	+	+	+	+++	++	+/++	+	–	1+
8	+++	++	-	-	+	+	+++	++	+/++	+	–	1+
9	+++	++	+	+	+	+	+++	++	+/++	+	–	–
10	+++	++	+	+	+	+	+++	++	+/++	+	–	3+
11	+++	++	+	+	+	+	+++	++	+/++	+	–	–
12	+++	++	+	+	+	+	+++	++	+/++	+	–	1+
13	+++	++	+	+	+	+	+++	++	+/++	+	–	–
Group C						+						
14	+++	++	+	+	+	+	+++	++	+/++	+	+	1+
15	+++	++	+	+	+	+	+++	++	+/++	–	–	1+
16	+++	++	+	+	+	+	+++	++	+/++	+	+	1+
17	+++	++	+	+	+	+	+++	++	+/++	–	–	1+
18	+++	++	+	+	+	+	+++	++	+/++	+	+	1+
19	+++	++	+	+	+	+	+++	++	+/++	+	–	1+
20	+++	++	+	+	+	+	+++	++	+/++	+	–	1+
21	+++	++	+	+	+	+	+++	++	+/++	+	–	1+

T = temporal bulbar conjunctiva; N = nasal bulbar conjunctival; C = central (inferior) bulbar conjunctival; U/L = upper and lower tear meniscuses. Severity of CCh based on folds of loose conjunctiva: +++, severe; ++, moderate; +, mild; −absent. Staining in the CCh was determined by rose bengal staning. Lid margin erosion was determined by the presence of anterior migration of the mucocutaneous junction shown by fluorescein staining in or near CCh. Obliterated tear meniscus was determined by fluorescein staining. +; positive or present; −, negative or absent. Severity of floppy eyelids was graded as 1+ when less than 1/3 of the upper tarsus was visible after lid eversion, 2+ when up to ½ of the upper tarsus was visible, and 3+ when more than ½ of the upper tarsus was everted.
*Pingueculae were present in both nasal and temporal bulbar conjunctiva.

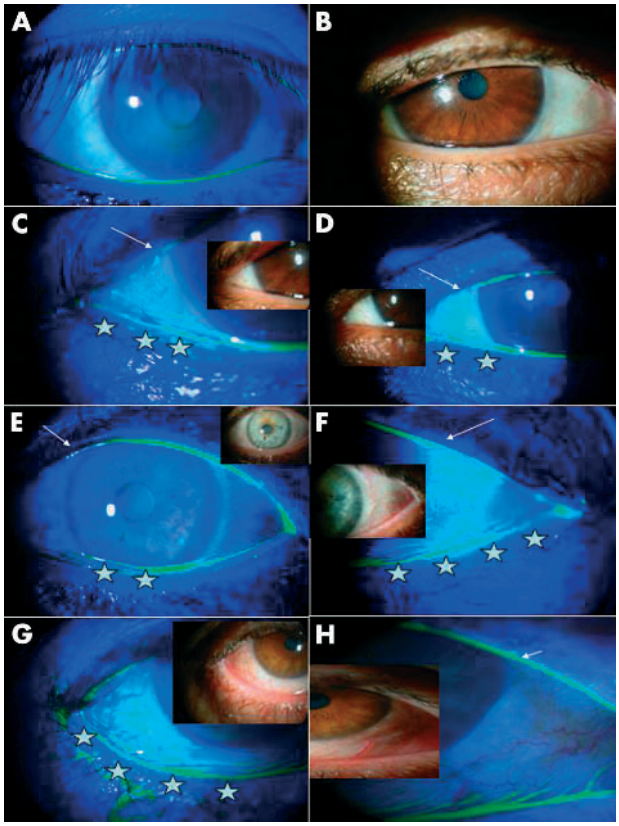

Figure 1 Fluorescein staining showing obliteration of tear meniscus of lower and upper lids by CCh. (A) In this representative ATD patient without CCh, there is continuous low tear meniscus of the lower and upper lids. (B to D) In this representative ATD with severe CCh, conjunctival folds were noted in the temporal bulbar conjunctiva (B). However, even if the meniscus was low, obliteration of the tear meniscus by temporal CCh becomes apparent when fluorescein is used, and is noted in both the lower and upper lids (C) and by nasal CCh (D) (stars and arrow respectively). (E and F) In this representative patient with severe CCh without ATD, obliteration of the tear meniscus of the lower lid (stars) and of the upper lid (arrows) by conjunctival folds is noted in both temporal and nasal bulbar conjunctiva, respectively. (G and H) Another representative patient with more severe CCh without ATD, obliteration of the tear meniscus of the lower lid (stars) and of the upper lid (arrows) by multiple conjunctival folds is noted in both temporal and nasal bulbar conjunctiva, respectively. Insets show the corresponding control without fluorescein.

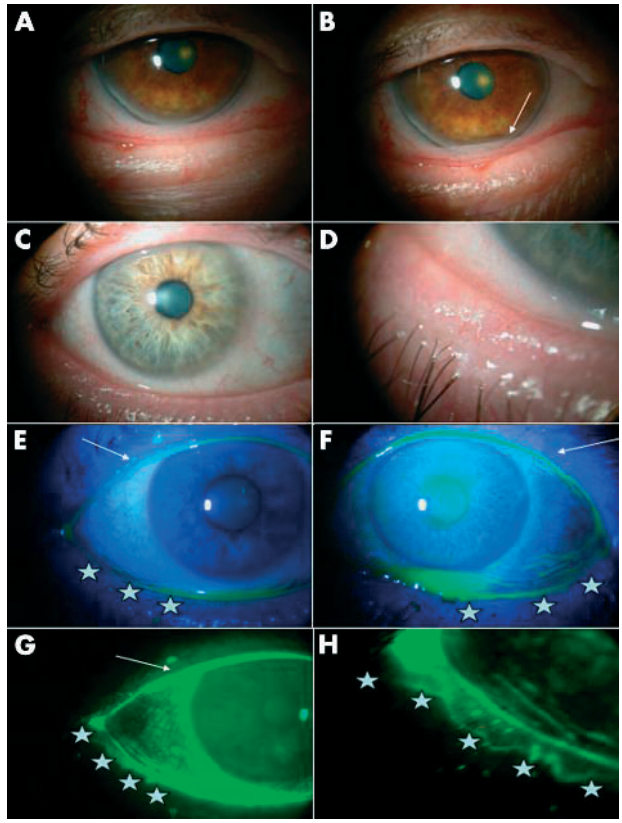

Figure 2 Worsening of CCh after vigorous blinking and anterior migration of the mucocutaneous junction and CCh shown by fluorescein staining. (A and B) Conjunctival folds noted in the 6 o'clock position (A) become much worse by covering more inferior cornea (B, arrow) after vigorous blinking. (C and D) In a representative CCh patient without ATD, signs of inflammation of the temporal lid margin are observed in the lower lid, especially under higher magnification. (E and F) In the same patient as C and D, obliteration of tear meniscus and conjunctival folds are noted in the lower (stars) and upper (arrow) lids of the right eye (E) and the left eye (F). The extent of CCh is worse in the left eye than the right eye and tear meniscus was pooled before conjunctival folds in the left eye. (G and H) In the same patient, when an enhancing filter is used, obliteration of tear meniscus (see arrow in G) and conjunctival folds become more obvious (compare with E). In addition, erosion leading to anterior migration of the mucocutaneous junction becomes more apparent (stars) immediately adjacent to CCh. Under higher magnification, such migration is noted passing orifices of meibomian glands.

DISCUSSION

CCh patients were significantly older than ATD patients without CCh, consistent with the notion that CCh is associated with ageing.[9 11–13] Dryness was worse as the day progressed in ATD due to progressive exposure and desiccation throughout the day. In contrast, CCh patients also complained of additional blurry vision and pain, both of which tended to be worse when reading. This is because CCh folds are increased by downgaze during reading. On the contrary, the interpalpebral exposure zone increases from downgaze to upgaze,[14] explaining why ATD dry eye is worse in upgaze, especially when working with a computer screen.[15] Therefore, differences in symptoms upon upgaze (computer work) or downgaze (reading) help differentiate pure ATD induced dry eye from CCh induced dry eye. Furthermore, frequent blinking exacerbated CCh, resulting in the spread of conjunctival folds to the 6 o'clock position, further aggravating blurring vision and dryness. In contrast, increasing blinking shortens the inter-blink interval, invariably stabilising the tear film and improving ATD dry eye.[16] Furthermore, if ATD patients also had CCh, symptoms were worse in the morning upon awakening because delayed tear clearance is worsened during sleep (also see below).

Traditionally, CCh is recognised only in the lower aspect.[1] The present study, however, revealed that CCh was also found in the upper aspect. This was best demonstrated by fluorescein staining to delineate the tear meniscus, especially with the aid of a special filter. Interruption or obliteration of the tear meniscus was noted in both lower and upper lids in CCh but not in any patient with pure ATD. Severe CCh is frequently associated with subconjunctival haemorrhage.[1] Because of poor affixation of CCh conjunctiva onto the sclera, subconjunctival vessels are prone to rupture by blinking or rubbing. Therefore, the clinical finding of subconjunctival haemorrhage should prompt one to consider CCh as a cause. Because subconjunctival haemorrhage also takes place in the upper bulbar conjunctiva (not shown), we believe that CCh also involves the upper conjunctiva, a notion also suggested by the finding that superior limbic keratoconjunctivitis develops in some CCh patients.[17]

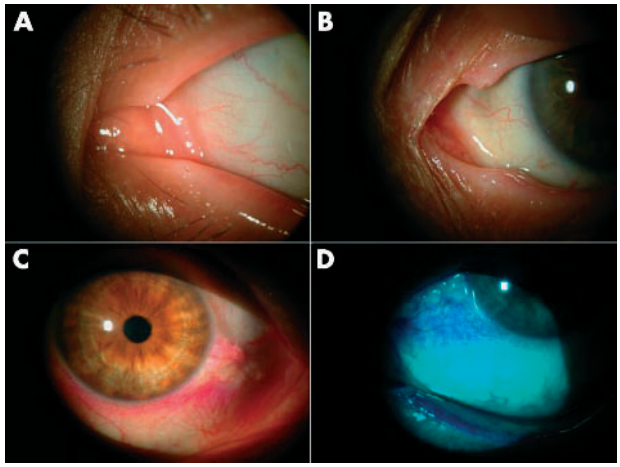

Figure 3 Swollen puncta and rose bengal staining. (A) Normal appearance of lower and upper puncta in an ATD patient. (B) Swollen puncta with elongation and subcutaneous oedema of lower and upper puncta in a CCh patient. (C and D) Under normal light and a green filter, respectively, rose bengal staining is detected at non-exposure zone (inferior limbal region), pingueculae (exposure zone), and the mucosal surface of the inferior lid margin (due to CCh in the region) in a CCh patient without ATD.

In pure ATD, an unstable tear film tends to develop at the interpalpebral exposure zone, which is characteristically stained by rose bengal. By interrupting or obliterating tear meniscuses, CCh destabilises the tear film immediately above the CCh area. That was why dye staining decorated the area above the redundant conjunctival fold in a linear pattern (fig 3C and D). That was also why rose bengal stained CCh areas, which upon pulling down the lower lid spread to the non-exposure zone (inferior to the exposure zone) as previously reported,[1] and the adjacent mucosal surface of the lid margin (fig 3D). If CCh progressed to the 6 o'clock position, the tear film would break up at the inferior corneal/limbal area, a pattern different from random tear break up of the cornea in pure ATD.

Another striking finding of CCh was its spatial correlation with anterior migration of the mucocutaneous junction. This pathology is presumably caused by overspill of aqueous tears due to the space occupying effect and obliteration of the tear meniscus by CCh (fig 2F). As a result, regional lid margin inflammation ensues and it is frequently mistaken for blepharitis. This pathology might also be aggravated by MGD and LTD because insufficient meibum in the lid margin leads to an ineffective lipid barrier, facilitating aqueous overspill to the skin. We do not know whether MGD is causatively linked with CCh or simply coexists with CCh due to old age. Because of its frequent association with MGD, CCh may worsen dry eye by contributing to additional LTD.

As stated earlier, ocular surface inflammation aggravates CCh. In CCh, inflammation might arise from lid margin or MGD. Inflammation, if pre-existing, could conceivably be worsened by delayed tear clearance, which was found in all CCh patients. We do not know whether delayed tear clearance is pre-existing or caused by CCh obliterating the tear meniscus. Because of delayed tear clearance, we anticipate that episodic tearing (epiphora) may occur in ATD patients with CCh. Previously, we have reported that delayed tear clearance aggravates ocular surface inflammation, leading to inflammatory symptoms that tend to be worse in the morning upon awakening, and is frequently associated with swollen puncta.[7] In the present study, we noted swollen puncta in 50% of CCh patients and delayed tear clearance in 100% of CCh patients. Because tear levels of inflammatory cytokines are increased in delayed tear clearance,[8] and inflammatory cytokines upregulate MMP-1 and MMP-3 of CCh fibroblasts,[5] we speculate that collectively they might contribute to the development and worsening of CCh.

As patients in group A were young, and all older patients with ATD would have some extent of CCh and MGD, it is important to determine if CCh contributes to the development of eye irritation by tear film instability (dry eye), delayed tear clearance, or inflammation. Clinical characteristics of CCh described herein help us look into such pathogenic mechanisms as interruption or obliteration of tear meniscus, ocular surface inflammation, delayed tear clearance, and anterior migration of the mucocutaneous junctions. Accordingly, we also believe treatments should include anti-inflammatory therapies. When medical treatments fail, the ultimate solution might be to resort to surgical correction of tear spread and clearance caused by CCh. Recognition of CCh in dry eye patients is thus of utmost important to devise effective treatment plans.

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