African Vision and Eye Health
ISSN: (Online) 2410-1516, (Print) 2413-3183

Dry eye disease: Clinical evidence for a public sector intervention strategy

Authors:
Phindile P. Mdlalose[1]
Vanessa R. Moodley[1,2]
Naimah Ebrahim Khan[1]

Affiliations:
[1]Discipline of Optometry, College of Health Sciences, University of KwaZulu-Natal, Durban, South Africa

[2]Keratoconus Foundation South Africa, Durban, South Africa

Corresponding author:
Phindile Mdlalose,
200103298@stu.ukzn.ac.za

Dates:
Received: 14 June 2025
Accepted: 01 Dec. 2025
Published: 19 May 2026

How to cite this article:
Mdlalose PP, Moodley VR, Ebrahim Khan N. Dry eye disease: Clinical evidence for a public sector intervention strategy. Afr Vision Eye Health. 2026;85(1), a1085. https://doi.org/10.4102/aveh.v85i1.1085

Background: Dry eye disease (DED) is a common, yet largely underdiagnosed, disorder of the eye encountered in healthcare facilities. It disrupts the tear film, causing ocular discomfort symptoms such as itching, tearing and irritation.

Aim: This study aimed to determine the clinical profile of DED patients at a tertiary eye hospital to inform a public sector intervention strategy.

Setting: The study was conducted at McCord Provincial Eye Hospital (MPEH).

Methods: A quantitative, descriptive study design was undertaken and included 602 patients. The standardised patient evaluation of eye dryness (SPEED) questionnaire was administered, after which the tear break-up time (TBUT), Schirmer 2, blink rate, tear meniscus height (TMH) and meibography clinical tests were done. Data were managed using Statistical Package for Social Sciences (SPSS) version 28.0.

Results: The prevalence of DED was 83.2%, with the majority being in the age group 41 years – 55 years (mean = 48.54 ± 18.76) and female (84.1%). The disease increased significantly with age ($p = 0.02$) and the prevalence of aqueous deficient, evaporative and mixed DED was 3.2%, 62.7% and 34.1%, respectively. Irrespective of the associated risk factors, the majority had either moderate (45.3%) or severe (30.9%) DED, with those with a history of glaucoma, hypertension and post-cataract surgery mostly having severe DED ($p < 0.001$).

Conclusion: Noting the chronic discomfort and other complications of DED, the high prevalence found among public sector hospital patients warrants a strategic intervention. Interventions could include a DED management protocol with clinical guidelines for interventions from primary to tertiary levels of care. Further, as most older patients presented with systemic diseases such as diabetes and hypertension, DED clinical guidelines should be extended to multidisciplinary teams managing systemic diseases.

Contribution: In addition to contributing to the scholarship of DED, the study provides empirical data to assist the provincial hospital and its eleven catchment district facilities in developing a comprehensive DED management strategy. Although conducted in the eThekwini District, the guideline has generic features which may be applied to health districts throughout the province of KwaZulu-Natal.

Keywords: dry eye disease; prevalence of DED; clinical DED diagnosis; evaporative dry eye; meibograph.

Introduction

Dry eye disease (DED) is a:

> [*M*]ultifactorial disease of the ocular surface characterised by the loss of homeostasis of the tear film, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles.[1]

The disease disrupts the tear film, causing symptoms such as ocular discomfort, itching, tearing and irritation.[2] The tear film, consisting of the lipid and muco-aqueous layers, protects the eye from infections and foreign particles, creating a moist and healthy surface. Dysfunction of each layer can result in a specific form of DED. Untreated DED may lead to progressive ocular surface diseases, which may lead to corneal scarring; making accurate, timely diagnosis and appropriate management important.[3]

Dry eye disease is considered a significantly growing public health problem worldwide, with global surveys estimating prevalence, in various age groups, ranging between 5% and >80%

across different countries.[4] Although there is limited scholarly evidence, the prevalence across the province of KwaZulu-Natal in South Africa has been anecdotally reported as being high, by both private and public sector practitioners.

According to the Tear Film and Ocular Surface Society Dry Eye Workshop (TFOS-DEWS), DED is classified into three categories, which are aqueous deficient dry eye (ADDE), evaporative dry eye (EDE) and mixed, when there is an overlap between the two types.[1] Clinical assessment enables DED to be classified as mild, moderate or severe.[5] Practitioners generally do the Schirmer and tear break-up time (TBUT) tests or rely on DED symptom questionnaires to diagnose DED.[6] Siong et al.[6] advise that while most clinicians still practise the traditional clinical dry eye tests, such as Schirmer and TBUT, there needs to be a shift from traditional clinical testing methods to non-invasive diagnostics, such as utilisation of the Oculus Keratograph® 5M (Oculus Optikgeräte GmbH, Wetzovir, Germany), a corneal topographer and dry eye analyser.

Despite DED being identified as a common disorder, it remains largely underdiagnosed in many healthcare facilities, even among patients presenting with other ocular disorders. A review of the scholarly literature revealed very few studies having been undertaken in South Africa on dry eye prevalence, of which, three were facility based and revealing a high DED prevalence (40% – 92%).[7,8,9,10] No information is reported on the DED clinical profile of patients within the public health sector in the province of KwaZulu-Natal (KZN).

McCord Provincial Eye Hospital (MPEH) is the only public specialist eye facility in KZN and serves as the referral centre for eleven surrounding districts. Hospitals and clinics refer patients to MPEH as the majority do not have ophthalmic or other eye care services. Approximately 1250 patients with a diverse range of ocular conditions present at the outpatient department each week. The researcher's clinical observation reveals that most of the glaucoma, keratoconus and vernal keratoconjunctivitis (VKC) patients who present for treatment at MPEH also have DED. To date, there is no policy, defined protocol or clinical guideline on DED management, which may be because of a lack of empirical evidence of DED prevalence and profile to inform planning. This study aimed to determine the clinical profile of DED patients at a tertiary hospital to inform a public sector intervention strategy.

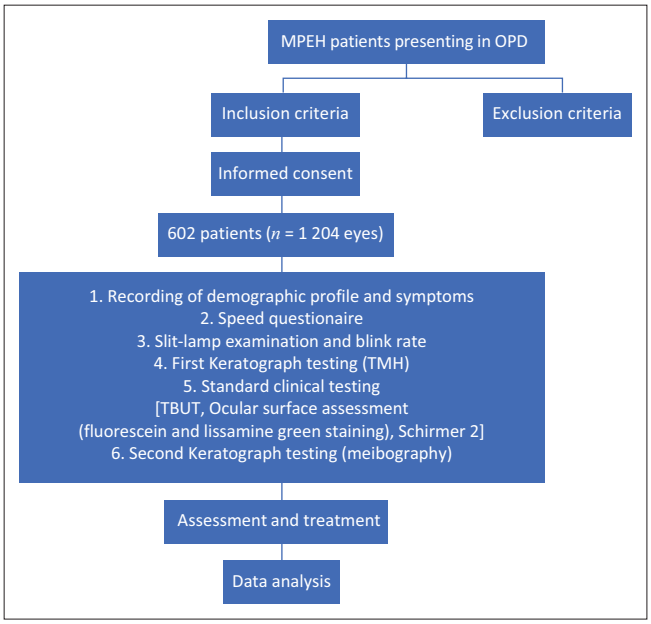
Research methods and design

A quantitative, single-centre, cross-sectional, descriptive study design was applied. Six hundred and two MPEH (tertiary eye hospital) patients, seated in the waiting area of the hospital were recruited to participate in the study between July 2023 and August 2023. Males and females aged 7 years and older were included, and patients with any ocular surface pathology (e.g. lid abnormality, corneal scarring, active ocular disease), recent intraocular surgery (< 3 months) or intellectual impairment were excluded. The study complied with the principles of the Helsinki Declaration for human participants, and informed consent and assent (for children) were obtained from study participants.

Study process

Demographic data, clinical symptoms and ocular and systemic medication history were recorded for eligible participants. The principal investigator administered the SPEED (standardised patient evaluation of eye dryness) questionnaire in English or isiZulu to each participant. Clinical tests were performed by the principal investigator in the following order to minimise the inter-test impact on results: blink rate, tear meniscus height (TMH), tear break-up time (TBUT), ocular surface assessment (corneal and conjunctival staining [fluorescein and lissamine green]), Schirmer 2 test, meibomian gland expression and meibography (Figure 1). Dry eye disease was diagnosed based on the TFOS DEWS II criteria and methodology report.[5,11]

The slit-lamp examination included eyelids being observed for any lid abnormalities that interfere with the normal spread of the tear film, followed by an examination of the lacrimal apparatus, cornea, sclera and conjunctiva. Thereafter, the blink rate was assessed subjectively while the participant sat at the slit-lamp, with < 15 blinks/min recorded as reduced. Meibomian gland digital expression for meibomian gland dysfunction (MGD) was performed with eyelids being everted and pressure applied to the lids using the cotton tip applicator to express the glands and squeeze out the oil. Meibomian orifices were examined for foam, secretion or blockage.



Source: Siong RL, Claudio KM, Dualan IJ, Sosuan GM. Clinical profile of dry eye disease at the Philippine General Hospital. Philippine J Ophthalmol. 2022;47(1):23–30

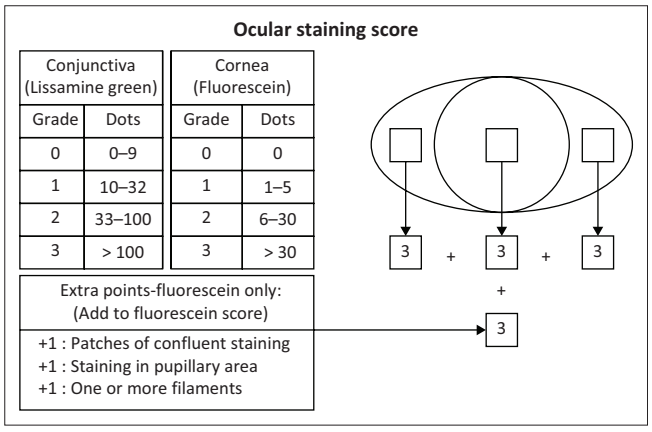
SPEED, standardised patient evaluation of eye dryness; TMH, tear meniscus height; TBUT, tear break-up time; OPD, outpatient department; MPEH, McCord Provincial Eye Hospital.

FIGURE 1: Study protocol flow chart.

The objective tests which were performed by the principal researcher, started with the TMH being measured using the Oculus Keratograph® 5M which measures the tear film pooling at the centre of the lower lid margin. The TMH picture was captured for each eye, and a TMH measurement was taken with a reading of < 0.2 mm considered abnormal. After imaging with the Oculus Keratograph®, TBUT was performed. A small amount of fluorescein was instilled into the eye, then the patient was asked to blink several times to distribute dye evenly. Using the stopwatch and cobalt filter on the slit lamp, a score was determined by counting the time in seconds between the last blink and the appearance of the first dry spot observed. This was repeated three times, and an average of three readings was taken. A reading of > 10 s was normal, and a TBUT of < 10 s indicated DED and tear film instability.[5] Immediately after the TBUT, ocular surface staining of the cornea and conjunctiva was performed in each eye.

A lissamine green strip was moistened and applied to the lower palpebral conjunctiva. After 1 min of staining the ocular surface, the conjunctiva and cornea were examined using the red-free filter on the slit lamp. The ocular surface was divided into three zones: nasal bulbar conjunctiva, corneal and temporal bulbar conjunctiva. Each zone was evaluated and graded from 0–3: 0 = no staining, 1 = mild staining, 2 = moderate staining and 3 = extensive staining. Corneal and conjunctival staining was graded using the SICCA ocular staining score, as shown in Figure 2.[12]

The Schirmer 2 test, also known as the basic secretion test, was performed after the TBUT. Each eye was anaesthetised with 0.5% tetracaine hydrochloride 0.5% minims, and a Schirmer strip was placed in the lower lid. A ruler was used to measure the wet area of the sterile Schirmer strip placed at the lower lid for 5 min with the patient's eyes closed. A reading of > 15 mm/5 min was normal, and less than 15 mm/5 min was diagnostic of dry eye and graded 9 mm – 14 mm/5 min: mild, 4 mm – 8 mm/5 min: moderate, and < 4 mm/5 min: severe.[5]



Source: Whitcher JP, Shiboski CH, Shiboski SC, et al. A simplified quantitative method for assessing keratoconjunctivitis sicca from the Sjögren's syndrome international registry. Am J Ophthalmol. 2010;149(3):405–415. https://doi.org/10.1016/j.ajo.2009.09.013 [12]

FIGURE 2: Sjögren's International Collaborative Clinical Alliance ocular staining score.

Lastly, the participant returned for meibography (infrared imaging of the eyelids to detect meibomian gland dropout) to be conducted. Each eyelid was everted using a sterile cotton bud, and a picture was taken of the meibomian glands. Dropout was graded as follows: grade 0: no dropout, grade 1: < 35% dropout (mild), grade 2: 33% – 67% dropout (moderate) and grade 3: > 67% dropout (severe) as shown in Figure 3. Both eyes were examined for all objective dry eye tests, and data were recorded for each eye.

Thereafter, DED was classified using the TFOS DEWS II recommended diagnostic criteria, including a symptom dry eye questionnaire plus one of the homeostasis marker tests (Schirmer 2, TBUT, TMH, ocular surface assessment).[1] Meibomian gland expression for MGD, meibography and TMH were considered for subclassification. The non-DED group, classified as the normal or control group, included participants without any or with very mild symptoms represented by a <4 SPEED score and no clinical signs (TBUT >10 s, Schirmer >15 mm/5 min, TMH >0.2 mm, no MGD and <grade 2 meibography score).

Participants with DED were further categorised into ADDE, EDE and mixed using TFOS DEWS II.[1] Participants classified as EDE had abnormal TBUT, abnormal meibography findings, MGD with normal Schirmer and TMH findings. Those with normal TBUT, no MGD and abnormal Schirmer and TMH were classified as ADDE, whilst those classified as mixed had both TBUT and Schirmer findings being abnormal.[11]

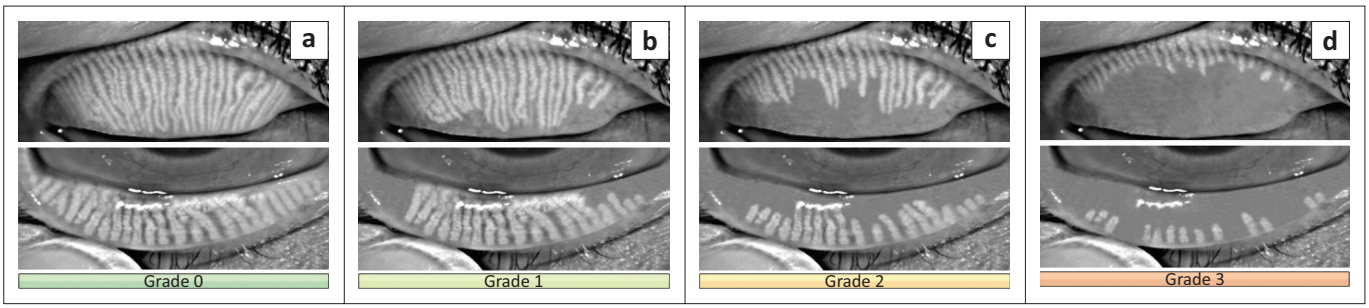
DED was further categorised by severity grades (Table 1) using TFOS DEWS II, which are mild, moderate, and severe.[1,13] Participants who required treatment for DED were referred for management and further review.

Data analysis

Data, including demographic information, SPEED questionnaire responses and objective test results, were collected using a standardised clinical data form and subsequently de-identified to maintain participant confidentiality. All data were entered into a password-protected Microsoft Excel spreadsheet and analysed using IBM SPSS Statistics software (Version 28.0).

Descriptive statistics such as means and frequencies were calculated to summarise the data. Inferential analyses included Chi-square tests and Fisher's exact tests to examine associations between categorical variables. For comparisons between two groups involving continuous parametric data, independent samples *t* tests were applied, while analysis of variance (ANOVA) was used for comparisons across more than two groups. In cases where data did not meet parametric assumptions, non-parametric tests were employed: Spearman's rank-order correlation was used for assessing relationships between two variables; the Wilcoxon signed-rank test and Kruskal–Wallis test were utilised for comparisons involving more than two groups.

To identify factors independently associated with the outcome, a binary logistic regression analysis was performed.



Note: Image from OCULUS Optikgeräte GmbH for grading the meibomian gland drop-out.

FIGURE 3: The structure and appearance of the meibomian gland drop out: (a) grade 0, (b) grade 1, (c) grade 2 and (d) grade 3.

TABLE 1: Dry eye severity classification.

Tear test	Mild	Moderate	Severe
SPEED	0–4	5–7	> 8
TBUT (s)	9–10	5–8	< 5
Schirmer test 2 (mm)	10–15	5–10	< 5
Corneal staining (SICCA score)	1–5	6–30	> 30

Source: Siong RL, Claudio KM, Dualan IJ, Sosuan GM. Clinical profile of dry eye disease at the Philippine General Hospital. Philippine J Ophthalmol. 2022;47(1):23–30

SPEED, standardised patient evaluation of eye dryness; TBUT, tear break-up time; SICCA, Sjögren's International Collaborative Clinical Alliance; s, seconds; mm, millimetre.

Variables showing a *p*-value of less than 0.25 in bivariate analyses were included in the multivariable regression model. The results of the regression are reported as odds ratios (OR) with 95% confidence intervals (CI). Statistical significance was set at $p < 0.05$.

Ethical considerations

Ethical clearance to conduct this study was obtained from the University of KwaZulu-Natal Biomedical Research Ethics Committee (BREC) on 24 July 2023. The ethics approval number is BREC/00003439/2021.

Results

A total of 602 patients, aged 7 years – 88 years, were included in the study. The mean age was 48.56 years (s.d. = 18.74), and females were the majority (78.6%). 'Most participants were of African ethnicity (83.2%; $n = 501$), with the remaining ethnic groups (white people [1%], indian people [13.5%] and mixed-race people [2.3%]) combined, only comprising 16.8% ($n = 101$)'.[10] The majority of participants ($n = 501$; 83.2%) were diagnosed with DED. The demographic profile of patients with and without DED is shown in Table 2. Binary logistic regression was performed to identify factors associated with DED. Age, occupation, diseases and medication were significantly associated with DED with the older population being more likely to experience DED.

Data analysis identified several significant risk factors for DED, including systemic diseases, medication, blink rate, contact lens wear, sleep apnea and previous cataract surgery. Although more participants presented with hypertension than other systemic diseases, the association with DED was found to be highest ($p = 0.01$) in patients with diabetes and glaucoma (Table 3).

TABLE 2: Demographic profile of the patients with and without dry eye disease.

Variable	Normal		DED		*p*-value
	n	%	*n*	%	
Age in years					0.0200*
7–18	18	29.5	43	70.4	-
19–40	39	24.5	120	75.4	-
41–55	19	11.9	140	88.1	-
56–65	11	10.7	92	89.3	-
> 65	14	11.7	106	83.3	-
Gender					0.2500
Females	75	15.9	398	84.1	-
Males	26	20.2	103	79.8	-
Occupation					0.0300*
Scholars	20	26.3	56	73.7	-
Employed	21	17.8	97	82.2	-
Unemployed	39	17.6	166	88.8	-
Pensioners	21	11.2	182	82.4	-
Systemic diseases	39	9.46	373	90.5	0.006*

DED, dry eye disease.

*, Denotes statistical significance.

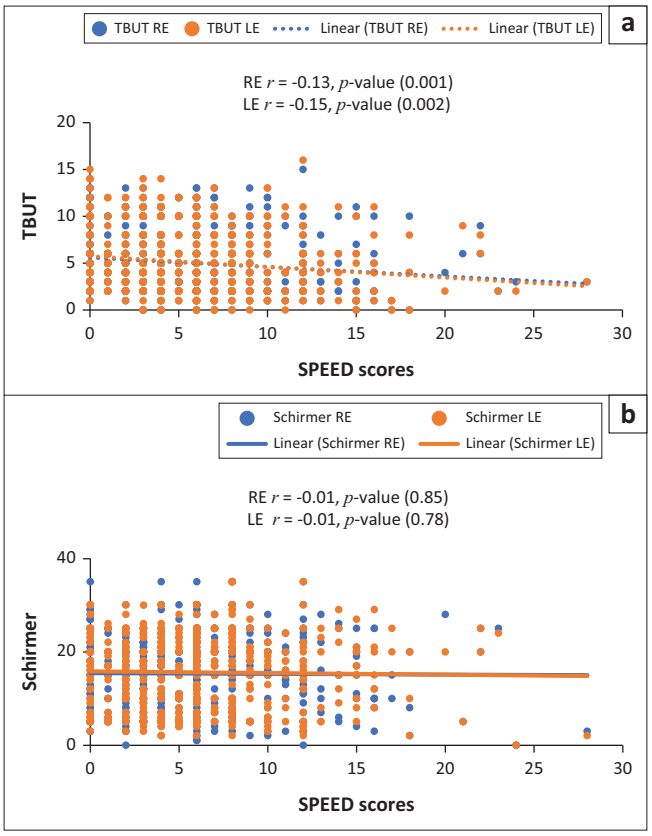
TABLE 3: Risk factors associated with dry eye disease.

Risk factor	Number of cases	DED cases		*p*-value
		n	%	
Systemic diseases	412	373	90.5	0.006*
Diabetes	99	91	92	0.010*
Hypertension	176	156	88.6	0.020*
HIV	78	72	92.3	0.020*
Glaucoma	46	41	89.1	0.010*
Arthritis	13	13	100	0.010*
Sleep apnea	102	87	85.2	0.540
Medication	327	-	-	0.010*
Topical	-	59	18	-
Systemic	-	268	82	-
Blink rate	502	466	92.8	0.002*
Contact lens wear	6	5	83.3	0.900
Previous cataract surgery	107	88	82.2	0.760

HIV, human immunodeficiency virus; DED, dry eye disease.

*, Denotes statistical significance.

Of the risk factors reported, systemic diseases ($p = 0.006$), medication ($p = 0.01$)[10] and blink rate ($p = 0.002$) were statistically significant. There were 327 (65.3%) participants on medication, of which 268 (82%) participants were on systemic (Beta-blockers, Antiretrovirals [ARVS] and non-steroidal anti-inflammatory drugs [NSAIDS]) and 59 (18%) participants on topical medication. The topical medication was mostly antiglaucoma topical drops (78%), with 22% participants using anti-allergic eye drops. Participants with DED had significantly lower blink rates than those without DED ($p = 0.002$).



RE, right eye; LE, left eye; TBUT, tear break-up time; SPEED, standardised patient evaluation of eye dryness.

FIGURE 4: Correlation between tear break-up time, Schirmer 2 and the standardised patient evaluation of eye dryness scores: (a) SPEED versus TBUT and (b) SPEED versus Schirmer.

TABLE 4a: Dry eye disease according to dry eye type.

p-value	Type of DED (*N* = 501)					
	ADDE		EDE		Mixed	
	n	%	*n*	%	*n*	%
< 0.001*	16	3.2	314	62.7	171	34.1

DED, dry eye disease; ADDE, aqueous deficient dry eye; EDE, evaporative dry eye.
*, Denotes significance.

TABLE 4b: Dry eye disease according to dry eye type and dry eye disease severity.

Variable	*p*-value	Severity of DED					
		Mild		Moderate		Severe	
		n	%	*n*	%	*n*	%
Total sample	-	119	23.8	227	45.3	155	30.9
ADDE	< 0.001*	12	75.0	4	25.0	0	0.0
EDE	< 0.001*	91	29.0	165	52.5	58	18.5
Mixed	< 0.001*	16	9.4	58	34.0	97	56.7

DED, dry eye disease; ADDE, aqueous deficient dry eye; EDE, evaporative dry eye.
*, Denotes significance.

The correlation between clinical tests (TBUT and Schirmer) for both eyes and SPEED scores is summarised in Figure 4. Spearman rank-order correlation was used to examine the relationships between subjective symptom scores (SPEED) and objective tear metrics (TBUT and Schirmer 2) for both eyes. There was a very weak negative correlation between SPEED and TBUT for the right (r = –0.13) and left eyes (r = –0.15; $p < 0.05$), suggesting that higher symptom scores were associated with slightly reduced tear stability. No significant correlation was found between SPEED and Schirmer 2 (r = –0.001; $p > 0.05$). Although statistically significant, the correlations were weak and unlikely to be clinically relevant.

The mean SPEED score of the DED group was 6.04 which reveals that, on average, there was mild (n = 209; 41.7%) to moderate (n = 137; 27.3%) DED (p = 0.03) (Table 4). Spearman correlation test analysis revealed significant differences in DED severity, with EDE (n = 165; 52.5%) and mixed (n = 58; 34.1%) being mostly moderate, while ADDE had significantly more participants with mild severity ($p < 0.001$).

The majority (n = 314; 62.7%) of participants with DED had EDE and were between the ages of 41 years – 55 years. There were very few (n = 16; 3.2%) with ADDE, with most of those affected (n = 6) being within the demographic of 19 years – 40 years old.

Meibomian gland dysfunction and meibomian gland dropout revealed a significant association with DED ($p < 0.001$) and both variables also increased with age. Fisher's exact test showed no statistically significant association between dry eye type (EDE, ADDE or mixed) and gender (p = 0.54). Corneal and conjunctival staining was only seen in the DED group (p = 0.007). There was a significant association between meibography and corneal staining (p = 0.002). Further, patients with moderate or severe meibography were almost twice as likely to have moderate or severe corneal staining (OR = 1.95; 95% CI: 129–196). There was a significant association ($p < 0.001$) between TBUT and meibography findings; as the TBUT values decreased, the meibomian gland dropout increased in severity.

Females were at higher risk of having DED, as 79 out of every 100 female participants, aged between 19 years and 65 years, presenting at the hospital had DED (Table 5). The majority (76.24%) of all participants with DED, irrespective of the associated risk factors, had moderate or severe dry eye, with those with glaucoma, hypertension and post-cataract surgery being the most severe ($p < 0.001$).

Discussion

Dry eye disease is a common ocular condition affecting a significant portion of the population, leading to discomfort, visual disturbances and diminished standard of living. To ensure optimal patient care and inform clinical eye health services planning, it is important to understand the prevalence and profile of DED among patients attending public health facilities. The study findings revealed that most patients presenting to MPEH, from various referral centres, were females in the 40 years to 70 years age group. Dry eye disease diagnosis was established in the majority (83.2%) of the participants, with a higher prevalence in females (84.1%), a finding concurring with other studies.[14,15] However, Idiculla and Zachariah[16] in a prospective study reported a slightly higher DED prevalence in males (n = 122, 51.9%). Possible reasons for the difference could be the variation in the study

TABLE 4c: Dry eye disease according to dry eye disease severity and screening test values.

Tear test	DED according to speed, Schirmer 2 and TBUT												*p*-value
	Mean values				DED severity								
	Normal		DED		Normal		Mild		Moderate		Severe		
	Mean	s.d.	Mean	s.d.	*n*	%	*n*	%	*n*	%	*n*	%	
Schirmer 2 (mm/5min)	19.56	± 5.52	14.69	± 7.67	219	43.7	132	26.3	124	24.8	26	5.2	< 0.001*
TBUT	10.21	± 1.66	4.02	± 2.39	17	3.5	44	8.9%	112	22.4	328	65.5	< 0.001*
Speed	4.54	± 3.47	6.04	± 4.39	-	-	209	41.7	137	27.3	155	31.0	0.030

DED, dry eye disease; TBUT, tear break-up time; SPEED, standardised patient evaluation of eye dryness; s, seconds; min, minutes; mm, millimetre.
*, Denotes significance.

TABLE 5: Prevalence and severity of dry eye disease according to risk factors.

Risk factor	DED prevalence		DED severity			
	DED cases per 100	*p*-value	Mild (%)	Moderate (%)	Severe (%)	*p*-value
Diabetes	18	0.01*	19.8	44.0	36.3	0.130
Glaucoma	8	0.28	7.3	46.3	46.3	0.001*
HIV	15	0.01*	22.2	43.1	34.7	0.510
Hypertension	31	0.03*	14.1	48.1	37.8	< 0.001*
Post-cataract surgery	18	0.72	15.9	40.9	43.2	< 0.001*
Sleep apnea	17	0.57	19.5	49.4	31.0	0.300
Age in years (19–65)	70	0.01*	24.6	43.7	31.7	0.790
Gender						
Females	79	0.22	22.4	45.5	32.1	0.280
Males	21	-	29.1	44.7	26.2	-

HIV, human immunodeficiency virus; DED, dry dye disease.
*, Denotes statistical significance.

selection criteria, as their study included 122 males and 118 females. Further, they only included healthy participants, excluding systemic diseases, contact lens users and post-ocular surgery participants.

As shown in Table 1, DED increased with age, a well-documented DED risk factor, resulting from a decreased lacrimal response which occurs with ageing.[17,18] Further, with increasing age, the chances of having systemic diseases and taking medication increase, conditions which may result in increasing aqueous tear deficiency.[19]

A significant association was found between DED and occupation (p = 0.03), even though the scholars and employed group comprised only 25.4% of the total sample. These groups had a much lower blink rate (< 10 blinks per minute) than pensioners and the unemployed. This could be because of increased usage of computers and mobile devices by the employed and scholars, as studies show an increase in DED with a reported decrease in blink rate by 40% – 60% and incomplete blinking.[20] Work and study tasks increasingly command the prolonged use of computers, cell phones or other visual display units (VDUs), leading to DED. Furthermore, air-conditioned rooms and low-relative-humidity office environments negatively impact the tear film, exacerbating DED. These negative influences can be alleviated by modifying the ergonomics of the work environment with changes such as reducing screen brightness and glare and using the appropriate screen location. Patients should be advised on blinking exercises, taking frequent breaks and modifiable factors like air-conditioners and low humidity.

Although only 1% of patients of the total sample were contact lens wearers, it is a concern that five had DED. Similarly, Uddin[3] found all six contact lens wearers in his study sample to have DED. Possible reasons could be a low blink rate and incomplete blinking in contact lens wearers. Furthermore, poor lens wettability may cause high evaporative loss during contact lens wear and contribute to tear film lipid composition changes.[21] Contact lenses with high Dk and low water content are recommended to maintain normal homeostasis of the tear film.[22] Jimenez et al.[23] cite blinking exercises and the use of preservative-free artificial tears supplements as important to contact lens wearers to reduce DED and improve patient comfort.

Similar to previous studies, such as the 'Beaver Dam Eye Study',[24] that found DED associated with systemic diseases, this study revealed a significant association with arthritis, hypertension, diabetes, thyroid disease, HIV and glaucoma.[9,10,25,26,27] The association with diabetes and glaucoma has been found to be the highest when compared to other conditions. Zhang et al.[28] assert that poor glycaemic control affects both anterior and posterior segments, increasing the prevalence of diabetes-associated DED. Furthermore, diabetics also have reduced corneal sensitivity, which contributes to DED, as corneal nerves are responsible for the blinking reflex, tear production and secretion.[28]

The profile of significant DED severity varied by systemic condition, with glaucoma and hypertension being more significantly associated with severe DED as compared to other systemic diseases. Noting that 31 out of every 100 hypertensive patients presenting at the hospital may likely have DED, an intervention by the facility is necessary. Yu et al.[26] stated that the association of DED with systemic

diseases warrants practitioners obtaining a detailed medical history and understanding which systemic diseases predispose to severe DED to improve overall patient management. Screening for the presence of DED in patients with risk factors helps with an early diagnosis and appropriate management. The study findings support a protocol whereby primary care clinicians, managing patients with related chronic systemic diseases, include referrals for a comprehensive tear function evaluation as part of their diagnostic protocols. Tear function assessments in patients with a history of any of the systemic diseases will help with early detection and treatment, ultimately improving patient comfort and preventing visual disturbances because of a disruptive tear film or ocular surface compromise.[29]

Ronaldo et al.[29] stated that the number of drugs administered systemically increases with age. The majority of study participants were on medication, many of which are known to have DED side effects, resulting in inhibition of the production of efficient tears. Only 18% were on topical medication (antiglaucoma and antihistamines), while 82% were on systemic medication, which included Beta-blockers, ARVS and NSAIDS. Some of these medications alter the tear volume and quality via the protein kinase pathway and may cause dry mouth.[30] Clinicians should be aware that if they cause a dry mouth, the chances of them causing dry eye are high.[31] Fraunfelder et al.[30] stated that long-term usage of topical ocular therapy may cause dry eye as well.

Some components of eye drop formulations can be toxic to the ocular surface with preservatives cited as the most common offenders, potentially causing epithelial cell damage and punctate epithelial keratitis, which interferes with ocular surface wetting.[2,32] Glaucoma treatment, which decreases or controls the intraocular pressure, may also disrupt the homeostasis of the tear film.[33] At MPEH, appropriately treating glaucoma patients presenting at the facility will be challenging as they are generally prescribed with antiglaucoma drops and tear supplements with preservatives, which may worsen the DED. This problem is inherent within the health system, as clinics and hospitals only prescribe medication contained in the essential drug list produced by the National Department of Health. Noting the availability of empirical data, it may be necessary for relevant stakeholders to engage to improve care of DED patients presenting at provincial facilities. Optimal clinical protocols should include consideration of disease and medication history, taking cognisance of contraindications when managing all patients presenting at the hospital. A further suggestion is to introduce appropriate DED management clinical guidelines at all facilities. Application of guidelines may warrant prescribing preservative-free tear supplements and alternative ocular or systemic medication to alleviate DED.

Poor correlation between signs and symptoms in DED has been a challenging issue highlighted in DED research. The study revealed that the prevalence of DED was 83.2% with clinical tests while with SPEED was only 56%. The study findings correlate with others which conclude that symptoms do not always correlate with clinical signs.[34,35] Although the screening questionnaires serve to identify potential dry eye patients, comprehensive diagnosis must include clinical assessments. Gierow and Kacz,[36] however, found a positive correlation between clinical tests and a Tear Evaluation and Research Tool for Contact Lens dry eye questionnaire (TERTC-DEQ), which may be considered for further studies.

Although the Schirmer test is the most used test to assess aqueous tear production, results revealed no correlation between the Schirmer 2 test and the SPEED score, which is also consistent with studies that used the OSDI questionnaire for comparison.[35,37] However, TBUT, a test commonly used to assess tear film stability, was found to have a significant correlation with SPEED scores ($p = 0.002$). Similarly, a correlation was revealed in Lee et al.[38] study between the ocular surface disease index (OSDI) and non-invasive keratograph break-up time (NIKBUT), supporting the notion that subjective symptoms are more likely to indicate a lipid deficiency as opposed to an aqueous deficiency. Variations in correlations between DED tests and symptoms revealed in this study highlight the multifactorial aetiology of DED and the fact that each diagnostic assessment for DED assesses a particular layer of the tear film, contributing to inconsistent symptoms experienced. Therefore, solely using a symptom questionnaire as a dry eye diagnostic tool may misdiagnose a significant portion of the DED population. Study findings highlight the importance of doing clinical tests together with a dry eye questionnaire to diagnose DED. This approach is also included in the latest TFOS DEWS III recommendation, which states that an abnormal score in the symptomatic dry eye questionnaire (OSDI) plus one of the tear film markers (NIKBUT, TBUT, osmolarity) and ocular surface staining (lid, corneal and conjunctival) is considered as a positive diagnosis of DED.[39] This recommendation, endorsed in this study, is further supported by Kyei et al.[35] who advise that dry eye diagnosis should focus on symptom assessment and clinical test results.

The study showed an association between TBUT and meibography ($p < 0.001$); as meibography increased in severity, so did TBUT. However, there was no correlation between Schirmer findings and TMH. This requires further investigation to strengthen the call for a complete shift from traditional clinical testing methods to non-invasive diagnostics, as recommended by Siong et al.[6] Further, studies are needed to support the inclusion of non-invasive tests in resource constrained public sector facilities to enhance DED diagnosis, classification and management as advised in other studies.[40]

Dry eye disease severity was graded according to the types of clinical tests done. Decreased TBUT indicates a tear film instability and is a vital indicator in diagnosing and treating DED. In this study, moderate dry eye was most prevalent and DED severity was (45.3%). Dry eye severity was significantly

associated with age ($p = 0.06$). This could be because of the beginning of menopause. However, these results are in discordance with Chalmers et al.[41] who underestimated dry eye severity by at least one grade compared to subject's self-assessment, especially among the elderly and females. Differences may be attributed to clinicians in their study mainly relying on the subject's global dry eye self-assessment as opposed to the use of clinical tests or both.

Successful management of DED depends on early diagnosis, accurate DED subtype classification and severity determination. Garcia et al.[13] advise that a patient's dry eye classification and severity status should be comprehensively diagnosed, as individualised treatment will ensure improvement of the patient's therapeutic outcome.

Corneal and conjunctival staining were only seen in the DED group and in those with severe DED ($p < 0.001$). Studies report that DED symptoms may occur before the evidence of ocular surface damage; thus, staining alone is not appropriate to diagnose DED.[42] However, corneal and conjunctival staining is important in determining dry eye severity and facilitates the measurement of ocular surface inflammation in DED.[43]

Increased tear evaporation, tear hyposecretion and/or mucin dysfunction are known aetiologies of DED.[44] In the current study, more than half of the DED group had reduced TBUT, with most having MGD and meibomian gland dropout, contributing to the diagnosis of EDE. As shown in Table 3, EDE was most prevalent, with only 3.2% of participants solely having ADDE. This is not unexpected as EDE, or reduced TBUT, was noted by the Asia Dry Eye Society as a predominant form of DED.[42] Other studies also reported that more than 80% of DED cases fall into EDE or mixed, with 10% being solely ADDE.[45,46] Findlay et al.[47] suggest that EDE management should focus more on underlying ophthalmic and systemic conditions and aggressive therapies to protect the ocular surface rather than mainly focusing on artificial tears and warm compressors. Public health sector facilities in KZN could note this treatment recommendation in the DED management resource planning as currently, many only provide artificial tear supplements to treat EDE.

Limitations

The limitation of the study is that it was a hospital-based study with a predominance of one ethnic group (Black African people), which excludes information on any genetic predisposition to DED. Although a single site was used, MPEH does receive referrals from eleven districts (referral centres), making it representative of the provincial profile of public sector patients.

Conclusion

The study revealed high DED prevalence (83.2%), which concludes that a significant number of DED patients are presenting at public sector hospitals in the province of KZN. Evaporative dry eye is the most prevalent type, and significant associations were revealed among DED, age, systemic diseases and medication. Notably, hypertension and glaucoma may lead to patients having a more severe form of DED. As many patients routinely visit their primary care practitioners to manage their systemic diseases and obtain chronic medication, dry eye assessments should be included as part of the comprehensive care of patients with diabetes, hypertension, glaucoma, HIV, arthritis and other related diseases. Given the high DED prevalence and clinical profile data contributed by this study, the authors recommend implementing strategies to achieve early diagnosis, prevention and appropriate treatment for DED patients. A multifaceted approach to manage this complex condition should be considered by appropriate public sector decision-makers and additionally, the optometry minimum competency standards be revised and updated accordingly by the regulator.

Acknowledgements

Special thanks to Mr B. Kotze from OCULUS and Mr N. Sivalingum from GENOP for providing the Oculus Keratograph and Mr S. Govender, Department of Optometry University of KwaZulu-Natal for assistance in securing equipment for data collection. We would aslo like to thank Ms Catherine Connolly and Dr Zelalem Dessie for assistance with the statistical analysis.

Competing interests

The authors, Phindile P. Mdlalose, Vanessa R. Moodley and Naimah Ebrahim Khan, declare that they have no financial or personal relationships that may have inappropriately influenced them in writing this article.

CRediT authorship contribution

Phindile P. Mdlalose: Conceptualisation, Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Visualisation, Writing – original draft, Writing – review & editing. Vanessa R. Moodley: Conceptualisation, Supervision, Writing – review & editing. Naimah Ebrahim Khan: Supervision, Writing – review & editing. All authors reviewed the article, contributed to the discussion of results, approved the final version for submission and publication, and take responsibility for the integrity of its findings.

Funding information

The authors received no financial support for the research, authorship and/or publication of this article.

Data availability

The data that support the findings of this study are not openly available and are available from the corresponding author, Phindile P. Mdlalose, upon reasonable request.

Disclaimer

The views and opinions expressed in this article are those of the authors and are the product of professional research. They do not necessarily reflect the official policy or position of any affiliated institution, funder, agency or that of the publisher. The authors are responsible for this article's results, findings and content.

References

1. Craig JP, Nichols KK, Akpek EK, et al. TFOS DEWS II definition and classification report. Ocul Surf. 2017;15(3):276–283. https://doi.org/10.1016/j.jtos.2017.05.008
2. Behera S, Sahoo S, Hota G, Mahapatra B. Clinical profile of dry eye disease at a tertiary care centre in western Odisha. J Evid Based Med Healthc. 2017;4(72):4274–4277. https://doi.org/10.18410/jebmh/2017/851
3. Uddin MF. Clinical profile of patients of dry eye – A prospective observational study. Sch J Appl Med Sci. 2023;6:1212–1215. https://doi.org/10.36347/sjams.2023.v11i07.007
4. Stapleton F, Alves M, Bunya VY, et al. TFOS DEWS II epidemiology report. Ocul Surf. 2017;15(3):334–365. https://doi.org/10.1016/j.jtos.2017.05.003
5. Wolffsohn JS, Arita R, Chalmers R, et al. TFOS DEWS II diagnostic methodology report. Ocul Surf. 2017;15(3):539–574. https://doi.org/10.1016/j.jtos.2017.05.001
6. Siong RL, Claudio KM, Dualan IJ, Sosuan GM. Clinical profile of dry eye disease at the Philippine General Hospital. Philippine J Ophthalmol. 2022;47(1):23–30.
7. Castelyn B, Majola S, Motilal R, et al. Prevalence of dry eye amongst black and Indian university students aged 18–30 years. Afr Vis Eye Health. 2015;74(1):6. https://doi.org/10.4102/aveh.v74i1.14
8. Nonkula DM. The prevalence of dry eye syndrome among patients at the eye clinic in Nelson Mandela Academic Hospital [dissertation]. Bloemfontein: University of the Free State, 2019; p. 84.
9. Khan E. Prevalence of dry eye syndrome in a South African diabetic paediatric population. Cont Lens Anterior Eye. 2024;47(5):102175. https://doi.org/10.1016/j.clae.2024.102175
10. Mdlalose PP, Moodley VR. Dry eye: A hospital-based sociodemographic, risk and clinical classification profile. Health SA Gesondheid. 2025;30:2793. https://doi.org/10.4102/hsag.v30i0.2793
11. Vidal-Rohr M, Craig JP, Davies LN, Wolffsohn JS. The epidemiology of dry eye disease in the UK: The Aston dry eye study. Cont Lens Anterior Eye. 2023;46(3):101837. https://doi.org/10.1016/j.clae.2023.101837
12. Whitcher JP, Shiboski CH, Shiboski SC, et al. A simplified quantitative method for assessing keratoconjunctivitis sicca from the Sjögren's syndrome international registry. Am J Ophthalmol. 2010;149(3):405–415. https://doi.org/10.1016/j.ajo.2009.09.013
13. Rodriguez-Garcia A, Babayan-Sosa A, Ramirez-Miranda A, et al. A practical approach to severity classification and treatment of dry eye disease: A proposal from the Mexican dry eye disease expert panel. Clin Ophthalmol. 2022;2022:1331–1355. https://doi.org/10.2147/OPTH.S351898
14. Shah S, Jani H. Prevalence and associated factors of dry eye: Our experience in patients above 40 years of age at a tertiary care Center. Oman J Ophthalmol. 2015;8:151-156. https://doi.org/10.4103/0974-620X.169910
15. Borrelli M, Frings A, Geerling G, Finis D. Gender-specific differences in signs and symptoms of dry eye disease. Curr Eye Res. 2021;46(3):294–301. https://doi.org/10.1080/02713683.2020.1801758
16. Idiculla T, Zachariah G. A prospective study at a tertiary care center on the clinical profile of dry eye. Int J Acad Med Pharm. 2024;6(1):2080–2083.
17. Sharma A, Hindman HB. Aging: A predisposition to dry eyes. J Ophthalmol. 2014;2014(1):781683. https://doi.org/10.1155/2014/781683
18. Li X, Wang Z, Mu J, Puerkaiti H, et al. Prevalence and associated risk factors of dry eye disease in Hotan, Xinjiang: A cross-sectional study. BMC Ophthalmol. 2023;23(1):214. https://doi.org/10.1186/s12886-023-02955-9
19. Onua AA, Chukwuka IO. Prevalence of dry eye disease in a rural Niger Delta community, southern Nigeria. Open J Ophthalmol. 2017;7(2):95–102. https://doi.org/10.4236/ojoph.2017.72014
20. Jaiswal S, Asper L, Long J, et al. Ocular and visual discomfort associated with smartphones, tablets and computers: What we do and do not know. Clin Exp Optom. 2019;102(5):463–477. https://doi.org/10.1111/cxo.12851
21. Gupta N, Prasad I, Jain R, D'souza P. Estimating the prevalence of dry eye among Indian patients attending a tertiary ophthalmology clinic. Ann Trop Med Parasitol. 2010;104(3):247–255. https://doi.org/10.1179/136485910X12647085215859
22. Chaudhary S, Ghimire D, Basu S, Agrawal V, Jacobs DS, Shanbhag SS. Contact lenses in dry eye disease and associated ocular surface disorders. Indian J Ophthalmol. 2023;71(4):1142–1153. https://doi.org/10.4103/IJO.IJO_2778_22
23. Fernández-Jimenez E, Diz-Arias E, Peral A. Improving ocular surface comfort in contact lens wearers. Cont Lens Anterior Eye. 2022;45(3):101544. https://doi.org/10.1016/j.clae.2021.101544
24. Paulsen AJ, Cruickshanks KJ, Fischer ME, et al. Dry eye in the beaver dam offspring study: Prevalence, risk factors, and health-related quality of life. Am J Ophthalmol. 2014;157(4):799–806. https://doi.org/10.1016/j.ajo.2013.12.023
25. Alesaeidi S, Salehiabari I, Alvand S. The systematic review of comorbidities with dry eye syndromes. Int J Adv Biotech Res. 2017;8(3):2375–2393.
26. Yu K, Bunya V, Maguire M, Asbell P, Ying GS. Dry eye assessment and management study research group. Systemic conditions associated with severity of dry eye signs and symptoms in the dry eye assessment and management study. Ophthalmology. 2021;128(10):1384–1392. https://doi.org/10.1016/j.ophtha.2021.03.030
27. Moss SE, Klein R, Klein BE. Prevalence of and risk factors for dry eye syndrome. Arch Ophthalmol. 2000;118(9):1264–1268. https://doi.org/10.1001/archopht.118.9.1264
28. Zhang X, Zhao L, Deng S, Sun X, Wang N. Dry eye syndrome in patients with diabetes mellitus: Prevalence, aetiology, and clinical characteristics. J Ophthalmol. 2016;2016(1):8201053. https://doi.org/10.1155/2016/8201053
29. Rolando M, Cantera E, Mencucci R, Rubino P, Aragona P. The correct diagnosis and therapeutic management of tear dysfunction: Recommendations of the PICASSO board. Int Ophthalmol. 2018;38:875–895. https://doi.org/10.1007/s10792-017-0524-4
30. Fraunfelder FT, Sciubba JJ, Mathers W. The role of medications in causing dry eye. J Ophthalmol. 2012;2012(1):285851. https://doi.org/10.1155/2012/285851
31. Sheppard J, Shen Lee B, Periman LM. Dry eye disease: Identification and therapeutic strategies for primary care clinicians and clinical specialists. Ann Med. 2023;55(1):241–252. https://doi.org/10.1080/07853890.2022.2157477
32. Zhang X, Vadoothker S, Munir WM, Saeedi O. Ocular surface disease and glaucoma medications: A clinical approach. Eye Contact Lens. 2019;45(1):11–18. https://doi.org/10.1097/ICL.0000000000000544
33. Nijm LM, De Benito-Llopis L, Rossi GC, Vajaranant TS, Coroneo MT. Understanding the dual dilemma of dry eye and glaucoma: An international review. Asia-Pacific J Ophthalmol. 2020;9(6):481–490. https://doi.org/10.1097/APO.0000000000000327
34. Lemp MA, Crews LA, Bron AJ, Foulks GN, Sullivan BD. Distribution of aqueous-deficient and evaporative dry eye in a clinic-based patient cohort: A retrospective study. Cornea. 2012;31(5):472–478. https://doi.org/10.1097/ICO.0b013e318225415a
35. Kyei S, Dzasimatu SK, Asiedu K, Ayerakwah PA. Association between dry eye symptoms and signs. J Curr Ophthalmol. 2018;30(4):321–325. https://doi.org/10.1016/j.joco.2018.05.002
36. Gierow JP, Kacz L. Effect of age and gender on dry eye according to tests and symptoms. Investig Ophthalmol Vis Sci. 2018;59(9):3826.
37. Onwubiko SN, Eze BI, Udeh NN, Onwasigwe EN, Umeh RE. Dry eye disease: Concordance between the diagnostic tests in Africa. Eye Contact Lens. 2016;42(6):395e400. https://doi.org/10.1097/ICL.0000000000000218
38. Lee JH, Kim CH, Choe CM, Choi TH. Correlation analysis between ocular surface parameters with subjective symptom severity in dry eye disease. Korean J Ophthalmol. 2020;34(3):203–209. https://doi.org/10.3341/kjo.2019.0133
39. Wolffsohn JS, Benítez-Del-Castillo J, Loya-Garcia D, et al. TFOS DEWS III diagnostic methodology. Am J Ophthalmol. 2025;279:387–450. https://doi.org/10.1016/j.ajo.2025.05.033
40. Covita A, Chen MH, Leahy C. Correlation between meibomian gland appearance and tear breakup time using a slit scanning ophthalmoscope. Investig Ophthalmol Vis Sci. 2019;60(9):6793.
41. Chalmers RL, Begley CG, Edrington T, Caffery B, Nelson D, Snyder C, Simpson T. The agreement between self-assessment and clinician assessment of dry eye severity. Cornea 2005;24(7):804–810. https://doi.org/10.1097/01.ico.0000154410.99691.3c
42. Tsubota K, Yokoi N, Shimazaki J, et al. New perspectives on dry eye definition and diagnosis: A consensus report by the Asia Dry Eye Society. Ocul Surf. 2017;15(1):65–76. https://doi.org/10.2147/OPTH.S351898
43. Yang S, Lee HJ, Kim DY, Shin S, Barabino S, Chung SH. The use of conjunctival staining to measure ocular surface inflammation in patients with dry eye. Cornea. 2019;38(6):698–705. https://doi.org/10.1016/j.jtos.2016.09.003
44. Rao PD. Etiology of dry eye. In: Roshandel D, editor. Recent advances in dry eye disease. London: IntechOpen; 2023. Chapter 2. https://doi.org/10.5772/intechopen.110142
45. Messmer EM. The pathophysiology, diagnosis, and treatment of dry eye disease. Dtsch Ärztebl Int. 2015;112(5):71. https://doi.org/10.3238/arztebl.2015.0071
46. Vidal-Rohr M, Craig JP, Davies LN, Wolffsohn JS. Classification of dry eye disease subtypes. Cont Lens Anterior Eye. 2024;47(5):102257. https://doi.org/10.1016/j.clae.2024.102257
47. Findlay Q, Reid K. Dry eye disease: When to treat and when to refer. Aust Prescr. 2018;41(5):160. https://doi.org/10.18773/austprescr.2018.048