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Original Research Article

A cross-sectional study of correlation of climatic droplet keratopathy with serum proteins, serum calcium and lipid profile

K Vallabha[1], Vivea Naresh Nagdev[1]*, Keerti Gururaj Wali[1]

[1]Dept. of Ophthalmology, Shri B M Patil Medical College, BLDE (DU), Vijayapura, Karnataka, India

Abstract

Background: Climatic Droplet Keratopathy (CDK) is a progressive corneal degeneration linked to environmental exposure and possibly systemic factors. This study investigates associations between CDK and serum protein levels, calcium levels, and lipid profiles in a semi-arid Indian population.

Materials and Methods: A cross-sectional study involving 64 subjects (32 CDK patients and 32 controls) was conducted using detailed ophthalmic evaluations and serum biochemical testing. CDK grading was performed by slit-lamp biomicroscopy. Statistical analyses employed Chi-square and ANOVA tests.

Results: CDK patients were predominantly male (71.9%, p=0.006), with a high proportion engaged in farming (59.4%). Serum calcium levels negatively correlated with CDK severity (p=0.015). HDL levels were significantly reduced in cases (p<0.001), while VLDL showed significant variation across CDK grades (p=0.011). Total protein, albumin, and A/G ratio did not differ significantly.

Conclusion: CDK shows strong associations with hypocalcemia, and low HDL cholesterol. These findings support a multifactorial pathogenesis involving environmental stress and systemic metabolic factors. Early screening and public health interventions in vulnerable regions may reduce disease burden.

Keywords: Climatic droplet keratopathy, Serum protein, Serum calcium, Lipid profile.

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1. Introduction

Climatic Droplet Keratopathy (CDK) is a slowly progressive, degenerative disorder of the cornea characterized by subepithelial opacities and golden-yellow deposits of varying sizes. The deposits primarily involve the sub-epithelial layer, Bowman's layer and superficial stroma of the cornea.[1] These lesions progressively impair visual acuity and may eventually cause blindness if left untreated. CDK has been reported predominantly in populations residing in harsh climatic zones with elevated ultraviolet (UV) radiation, low humidity, and chronic exposure to dust and wind.[2-4] Although a number of histochemical studies have been conducted, the nature of the corneal deposits remains unclear.

The severity of the disease has been classified into three grades-[5]

Grade 1- Characterised by multiple small translucent subepithelial deposits near the temporal or nasal limbus. These are best seen with back-scattered slit illumination and under high magnification. The visual acuity remains uncompromised at this stage (**Figure 1** A)

Grade 2- Fuzzy appearance of cornea with the opacity extending over the lower two-thirds of cornea (**Figure 1** B).

Grade 3- Clusters of golden yellow deposits of varying sizes with involvement of central cornea. Vision is impaired in this stage (**Figure 3**).

*Corresponding author: Vivea Naresh Nagdev
Email: vivea11497@gmail.com

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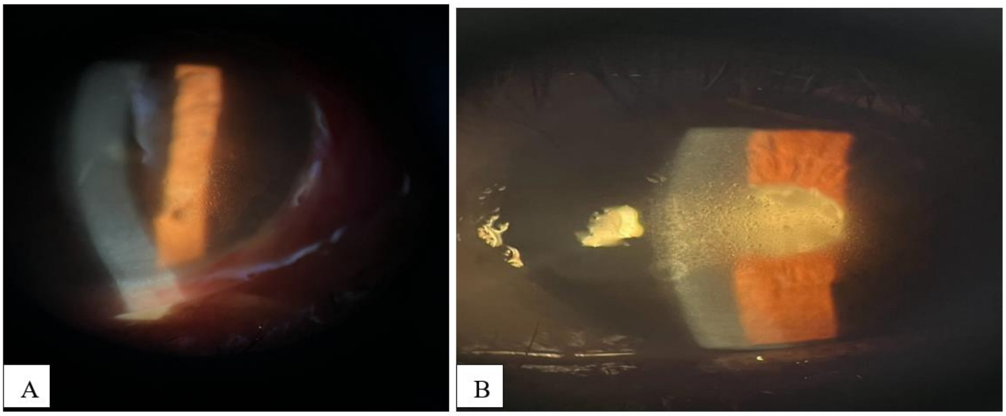

Figure 1: A): Grade 1 climatic droplet keratopathy; **B):** Grade 2 climatic droplet keratopathy

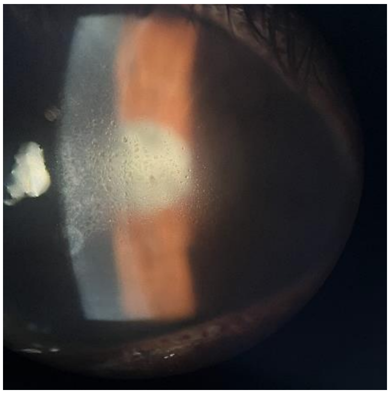

Figure 2: High magnification picture of grade 2 climatic droplet keratopathy

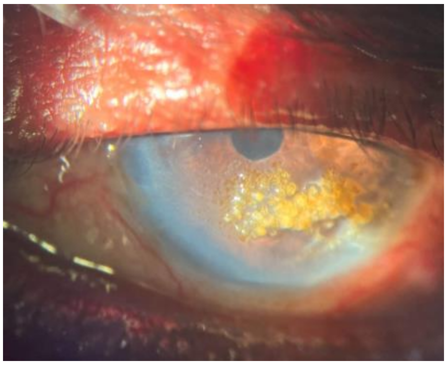

Figure 3: Grade 3 climatic droplet keratopathy

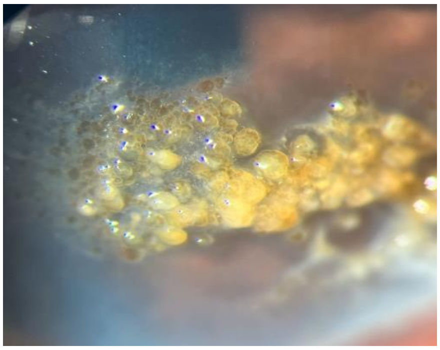

Figure 4: High magnification picture of Grade 3 climatic droplet keratopathy

Despite being globally distributed, CDK remains underreported in India, especially in regions like Karnataka where climatic and occupational exposure is conducive to its development.[4] Further, while previous research has focused primarily on environmental factors, little is known about the role of systemic biochemical parameters-serum protein levels, calcium homeostasis, and lipid profiles-in the pathophysiology of CDK.

Analysis of staining reaction in Climatic Droplet Keratopathy reported that within the spheroidal droplets there is protein with a considerable amount of amino-acids tryptophan, tyrosine, cysteine and cystine which are not normally present in large amounts.[6] Proteomic studies on CDK lesions have identified misfolded proteins and oxidative markers, indicating that the disease may share molecular similarities with amyloid-like degenerative conditions.[7,8] Research by Kaji et al. and Serra et al. has highlighted the presence of denatured proteins and ECM components within the stromal deposits of CDK-affected corneas.[8] These Studies have also suggested that chronic UV exposure leads to oxidative stress and protein denaturation in the corneal layers.

In addition to ocular surface stress, systemic nutritional factors may influence CDK progression. Serum proteins and albumin/globulin (A/G) ratios are markers of nutritional and inflammatory status. Likewise, serum calcium plays a vital role in maintaining epithelial barrier function and nerve signalling.[9] Tabara reported occasional positive staining of the deposits with Von kossa stain indicating the presence of calcium.[2]

Lipid peroxidation, a process in which reactive oxygen species (ROS) degrade polyunsaturated fatty acids in cell membranes, plays a key role in the pathogenesis of CDK. UV radiation promotes oxidative stress and lipid peroxidation, leading to cellular damage and the formation of abnormal protein-lipid aggregates in the cornea, contributing to the progression of CDK.

The objective of this study was to evaluate the correlation of CDK with, serum protein levels, serum calcium levels, and lipid profile, thereby identifying systemic and environmental factors contributing to its pathogenesis.

2. Materials and Methods

This cross-sectional observational study was conducted at a tertiary care hospital of northern Karnataka over 18 months. The region is semi-arid, with high UV index (7-8), intense summer temperatures (>44°C), and frequent dusty winds. Using G*Power software, a sample of 64 participants was calculated based on hyperlipidemia (14)(power = 0.80, α = 0.05). Participants included 32 CDK patients and 32 age- and sex-matched controls without CDK or clinical tear dysfunction. Adults aged more than 40 years with clinically confirmed CDK were included in the study.

2.1. Exclusion criteria

1. History of ocular trauma, intraocular surgery, autoimmune disorders, or uveitis.
2. Patients who refused to give consent.

All patients underwent a thorough screening and examination which included

1. Ophthalmic evaluation**:** All patients received a thorough screening that included slit-lamp examination and grading of CDK severity was done from 0-3.
2. Biochemical tests**:** To evaluate potential systemic associations with CDK, venous blood samples were collected and analyzed in the VITROS 4600 chemistry system for the following biochemical parameters: 1. Serum proteins 2. Serum Albumin 3. Serum calcium 4. Lipid profile

Data was entered into Microsoft Excel (Windows 7; Version 2007) and analyses were done using the Statistical Package for Social Sciences (SPSS) for Windows software (version 22.0; SPSS Inc, Chicago). Descriptive statistics such as mean and standard deviation (SD) for continuous variables, frequencies and percentages were calculated for categorical Variables were determined. Association between Variables was analyzed by using Chi-Square test for categorical Variables. Unpaired t Test was used to compare mean of quantitative variables between Cases and Controls. Level of significance was set at 0.05.

3. Results

From May 2023 to December 2024, comprehensive ophthalmological evaluations identified 49 cases of Climatic Droplet Keratopathy (CDK). Following a thorough screening process, 17 cases were excluded based on exclusion criteria that included a history of prior intraocular surgeries, ocular trauma, uveitis, or systemic syndromes associated with dry eye disease. The present study was thus conducted on 32 confirmed cases of Climatic Droplet Keratopathy, alongside a age matched control group, to evaluate relevant clinical, demographic, and environmental factors. The findings derived from this cohort are detailed in the subsequent sections.

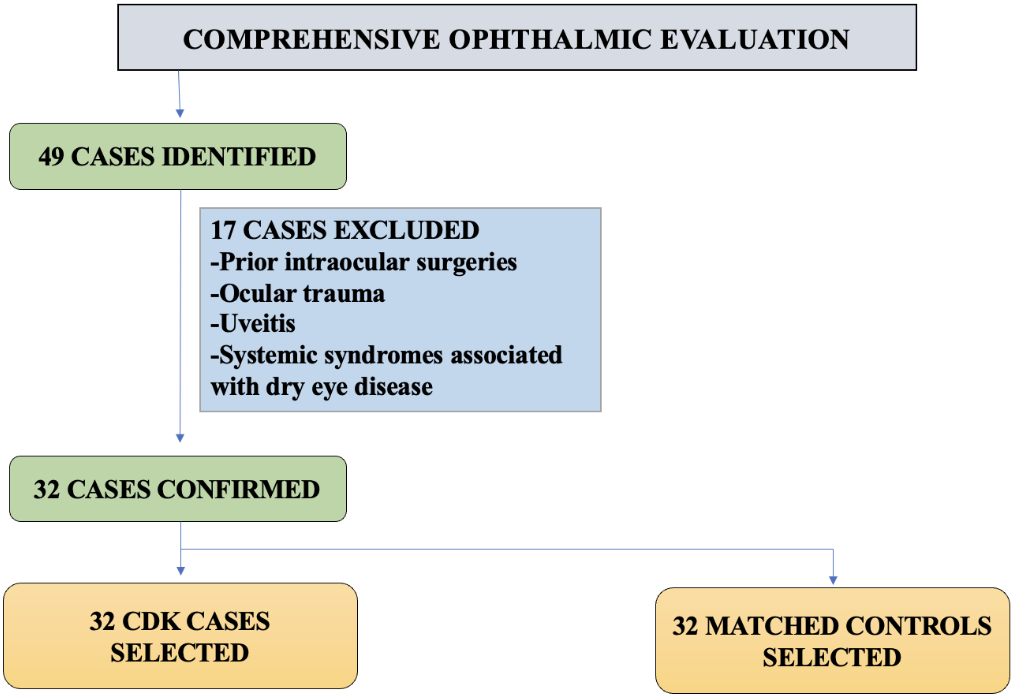

Figure 5: Selection of cases and controls

3.1. Demographics

The mean age of CDK cases was 69.41 years (SD = 9.66), compared to 62.56 years (SD = 10.89) in controls. While the age difference was not statistically significant (p = 0.178), older individuals tended to have more advanced disease (**Table 1**).

Gender distribution showed a statistically significant difference between cases and controls. Among the cases, a majority were male (71.9%), while females accounted for only 28.1%. In contrast, the control group had a predominantly female composition (62.5%), with males comprising 37.5%. The disparity in gender proportions was tested using the Chi-square test, yielding a P value of 0.006, indicating a statistically significant association between gender and case/control status. Farmers constituted 59.4% of CDK cases. Bilateral disease was observed in 81.3% of cases, suggesting a systemic-environmental aetiology.

3.2. Comorbidities

The distribution of comorbidities, specifically diabetes mellitus (DM) and hypertension (HTN), was compared between the two groups. Both cases and controls had an equal proportion (15.6%) of individuals with DM, indicating no difference in diabetes prevalence.

For hypertension, 25.0% of cases had HTN compared to 15.6% of controls. However, this difference was not statistically significant (P = 0.351).

3.3. Biochemical analysis- serum protein

Protein levels were categorized as low, normal, or elevated among both cases and controls. The majority of participants in both groups had normal protein levels: 84.4% in both cases and controls. Low protein levels were observed in 12.5% of cases and 15.6% of controls, indicating a slight but statistically insignificant variation. Elevated protein levels were reported in only one case (3.1%), and none among controls (**Figure 6**).

Table 1: Comparison of age between cases and controls (N=64)

	Group	
	Cases (n=32) n (%)	**Controls (n=32) n (%)**
Age (in Years)		
41-50	2 (6.3)	7 (21.9)
51-60	3 (9.4)	4 (12.5)
61-70	13 (40.6)	15 (46.9)
71-80	10 (31.3)	4 (12.5)
>80	4 (12.5)	2 (6.3)
Mean (SD)	69.41 (9.66)	62.56 (10.89)
Chi-square test, p value = 0.178, Not significant		
Gender		
Female	9 (28.1)	20 (62.5)
Male	23 (71.9)	12 (37.5)
Chi-square test, p value = 0.006, Significant		
Occupation		
Businessman		1 (3.1)
Driver	1 (3.1)	1 (3.1)
Farmer	19 (59.4)	13 (40.6)
Housewife	4 (12.5)	8 (25.0)
Shopkeeper	2 (6.3)	
Tailor		1 (3.1)
Unemployed	5 (15.6)	8 (25.0)
Vendor	1 (3.1)	
Chi-square test, p value = 0.319, Not significant		

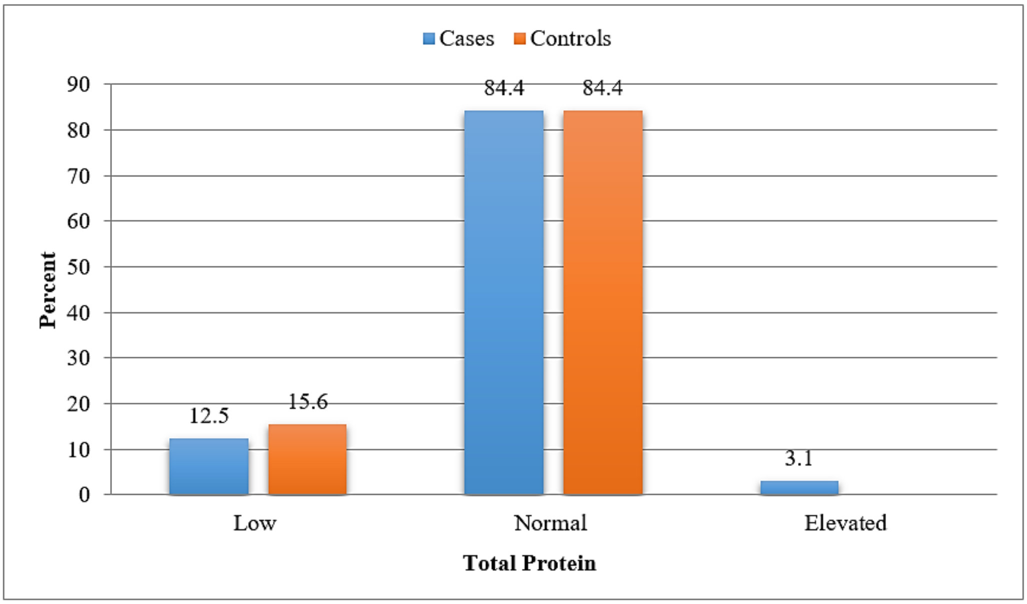

Figure 6: Comparison of serum total protein

Normal albumin levels were more common among controls (81.3%) compared to cases (59.4%). Low albumin was observed in a higher proportion of cases (37.5%) than controls (18.8%), suggesting a trend toward hypoalbuminemia in the case group. Elevated albumin was rare, seen in only 1 case (3.1%), and not observed in any control participants.

3.4. Biochemical analysis- serum calcium

Normal calcium levels were seen in the majority of participants in both groups—78.1% of cases and 84.4% of controls. Low calcium levels were more frequent in cases (21.9%) compared to controls (12.5%), indicating a modest difference. Elevated calcium was observed in 1 control participant (3.1%), but not in any of the cases (**Figure 7**).

3.5. Biochemical analysis- lipid parameters

Normal cholesterol levels were observed in majority of the participants—81.3% of cases and 84.4% of controls. Elevated cholesterol was slightly more frequent among cases (18.8%) than controls (15.6%). Normal TG levels were seen in the vast majority of participants: 87.5% of cases and 90.6% of controls. Elevated TG levels were reported in 4 cases (12.5%) and 3 controls (9.4%). A markedly higher proportion of cases (68.8%) had low HDL levels, compared to only 25.0% of controls. Conversely, normal HDL levels were found in 31.3% of cases and a significantly greater 75.0% of controls (**Figure 8**). No statistical difference was noted in the levels of LDL and VLDL among cases and controls.

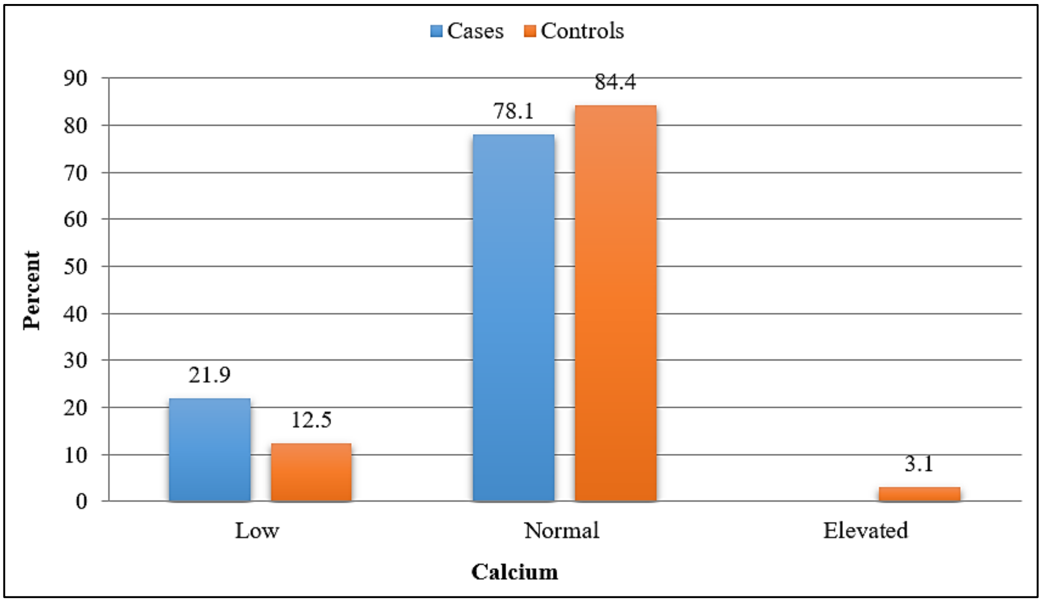

Figure 7: Comparison of serum calcium

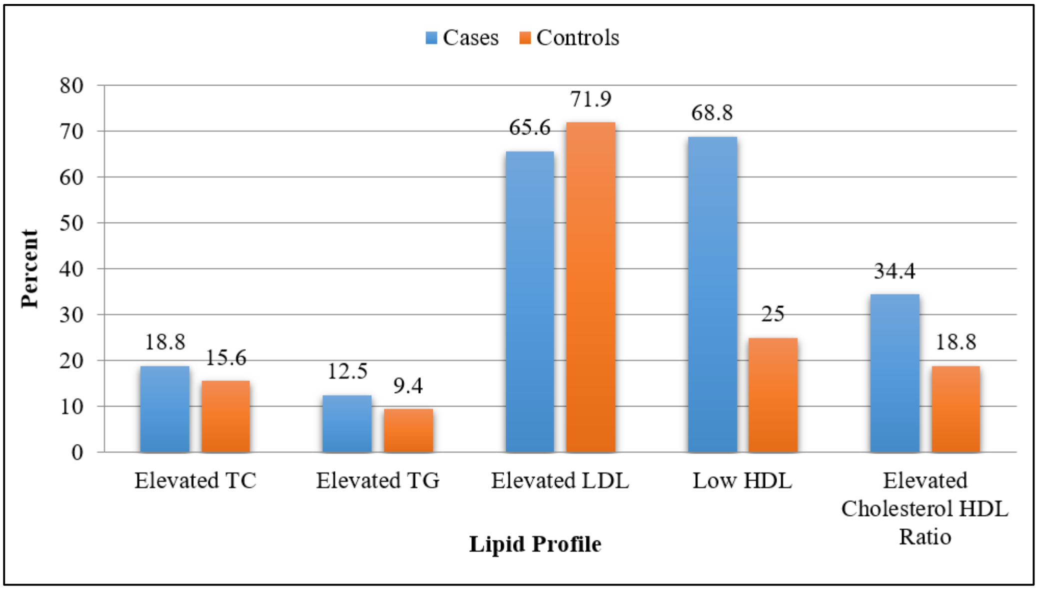

Figure 8: Comparison of Lipid parameters between cases and controls

Biochemical parameters of serum proteins (Total protein, albumin and globulin) were evaluated across different grades of Climatic Droplet Keratopathy (CDK) to assess potential biochemical associations with disease severity but showed no statistical significance. Serum calcium levels, however, showed a statistically significant difference across CDK grades (P = 0.015). The highest mean calcium was observed in Grade 1 (9.082 mg/dL), 8.838 in Grade 2 and the lowest was in Grade 3 (8.675 mg/dL**)**, indicating a potential inverse relationship between calcium levels and disease severity with p value of 0.015(statistically significant).

Fluctuation in levels of total cholesterol, Triglycerides, LDL and HDL did not show any statistical significance across grades of CDK. HDL cholesterol peaked in Grade 1 (42.50 mg/dL) and was lowest in Grade 0 (34.67 mg/dL). However, the differences were not statistically significant (P = 0.399). Very low-density lipoprotein (VLDL) levels exhibited a significant difference across CDK grades **(**P = 0.011). VLDL was lowest in Grade 1 (18.05 mg/dL), with a modest increase observed again in Grade 3 (19.90 mg/dL).

4. Discussion

The discussion on CDK aligns with established research, highlighting a higher prevalence in males and individuals with outdoor occupations. The study revealed a statistically significant male predominance among CDK patients (p = 0.006). This sex-based disparity may be attributable to occupational patterns — where men are more likely to engage in farming, outdoor labor, and long-term exposure to harsh climates, all of which heighten susceptibility. This is consistent with the well-documented role of long-term environmental exposure in the development of the condition.[11,12] The findings from Taylor et al.'s Australian study, which linked CDK in older Aboriginal populations to a lifetime of sunlight exposure without adequate eye protection, strongly support this environmental etiology.[13] Similarly, Mathur A et al.'s research in western Rajasthan further underscores the impact of harsh climates, characterized by high ultraviolet (UV) radiation, low humidity, and airborne dust.[14] The study was carried out in the semi-arid region of northern Karnataka, India, an area marked by intense solar radiation, low ambient humidity, and recurrent dust-laden winds that collectively heighten ocular surface stress. During the peak summer months, temperatures frequently reach 44°C under clear skies, accompanied by a high ultraviolet (UV) index, thereby amplifying the risk of phototoxic damage to the corneal epithelium. Conversely, the winter season is dominated by dry, gusty winds and minimal atmospheric moisture, conditions that aggravate tear film instability and evaporative dry eye. These extreme climatic factors provide a compelling ecological setting for the onset and progression of climatic droplet keratopathy (CDK).[15,16] This synergy of UV radiation and mechanical irritation from dust likely contributes to chronic ocular surface damage, a key step in CDK development.

The article delves into the microscopic and biochemical basis of CDK, particularly the role of proteins. Although the precise chemical composition of the disease-causing droplets is not fully known, evidence points to protein accumulation. Johnson and overall's study from Labrador demonstrated that the droplets accumulate in areas with the highest concentration of plasma proteins, suggesting a role for systemic factors. The finding that Bowman's membrane, a critical layer of the cornea, did not stain for albumin, while the droplets did, highlights the specific nature of this protein deposition.[17] Further insight comes from Menegay et al.'s proteomic analysis, which identified a complex mixture of proteins, including both extracellular matrix proteins and plasma proteins, within the droplets.[18] This suggests that the pathology is not just a simple deposition of external substances but a complex process involving both local tissue degradation and the influx of systemic components.

A key hypothesis presented by Kaji et al. proposes that Advanced Glycation End products (AGEs) are involved in the pathogenesis of CDK.[731] These are harmful compounds formed when proteins or lipids are glycated, often due to high blood sugar levels or oxidative stress. The aggregation of AGE-modified proteins could be a central mechanism in the formation of the characteristic corneal droplets, explaining the progressive nature of the disease. This suggests that the pathophysiology of CDK involves not just environmental insults but also a cascade of biochemical reactions that lead to irreversible protein damage and deposition. Although not statistically significant, albumin, total protein, and A/G ratio were marginally lower in CDK cases in our study. Albumin is the primary carrier of antioxidants like zinc and copper and plays a role in maintaining oncotic pressure and tissue repair. Hypoalbuminemia may signal chronic inflammation, protein loss, or malnutrition, all of which impair the eye's ability to respond to environmental insult.[19] Prior studies in ocular surface disorders like xerophthalmia and trachoma have linked low serum protein states to delayed corneal epithelial healing and increased susceptibility to keratinization.[20]

The discussion moves beyond local environmental damage to explore the influence of systemic health markers. While the study's findings on calcium were not statistically significant in a simple comparison, a deeper look reveals an inverse relationship between serum calcium levels and CDK grade. This is a crucial finding, as it suggests that lower calcium levels are associated with more severe disease. Calcium is a vital component for maintaining the integrity and renewal of the corneal epithelium. It plays a role in cellular signaling, tight junction formation, and wound healing.[21] Chronic hypocalcemia impairs the renewal of corneal epithelium and compromises stromal structure, potentially enhancing susceptibility to environmental injury.[22] Oxidative stress, a key driver of CDK, is exacerbated by calcium imbalance, which disrupts mitochondrial function and epithelial resistance to UV-induced apoptosis.[23] This finding provides a compelling link between systemic nutritional status and ocular surface resilience.

Similarly, the article highlights the strong association between dyslipidemia and CDK. The study found significantly lower levels of High-Density Lipoprotein (HDL), often referred to as "good cholesterol," in CDK cases. HDL is a powerful antioxidant and anti-inflammatory agent.[24] Its protective role on the ocular surface is significant, as UV radiation is known to cause lipid peroxidation, a form of oxidative damage. Low HDL levels could leave the corneal epithelial membranes more susceptible to this oxidative stress, compromising their barrier function and making them prone to damage.[25] The observed correlation between VLDL levels and CDK grade further reinforces this link between systemic lipid profile and local ocular pathology. This aligns with findings from other studies, such as the one among Korean women that connected dyslipidemia to dry eye and inflammation. The dietary link observed among Kazakh adults, who have a high-fat, low-vegetable diet and a high prevalence of CDK, provides a powerful example of how systemic dietary habits and dyslipidemia can contribute to the development of this ocular disease.[10] The integration of these systemic factors with the UV induced stress and oxidative imbalance, as proposed by Serra and Holopainen, provides a comprehensive model for understanding the complex pathogenesis of CDK.[23]

5. Limitations and Future Directions

1. The cross-sectional design prevents causal inference.
2. Small sample size limits subgroup analysis by occupation or income.
3. Environmental data (UV, humidity) were not quantitatively recorded.
4. Nutritional history and micronutrient levels were not assessed.

6. Future Research

1. Longitudinal studies tracking biochemical markers and CDK progression.
2. Tear proteomics to identify early disease biomarkers.
3. AI-driven risk prediction tools combining clinical and systemic parameters.

7. Conclusion

Climatic Droplet Keratopathy is a multifactorial ocular surface disease influenced by chronic environmental stress and systemic metabolic imbalance. Significant associations with low HDL underscore the need for integrated diagnostic and preventive strategies. Early identification of at-risk individuals and public health interventions in vulnerable populations can mitigate long-term visual morbidity.

8. Source of Funding

None.

9. Conflict of Interest

None.

10. Ethical Approval

Ethical No.: BLDE (DU)/IEC/902/2023-24.

References

1. Matta CS, Tabbara KF, Cameron JA, Hidayat AA, al-Rajhi AA. Climatic droplet keratopathy with corneal amyloidosis. *Ophthalmology*. 1991;98(2):192–5. https://doi.org/10.1016/s0161-6420(91)32317-0
2. Tabbara KF. Climatic droplet keratopathy. *Int Ophthalmol Clin*. 1986;26(4):63–8. https://doi.org/10.1097/00004397-198602640-00006
3. Serra HM, Holopainen JM, Beuerman R, Kaarniranta K, Suárez MF, Urrets-Zavalía JA, et al. Climatic droplet keratopathy: an old disease in new clothes. *Acta Ophthalmol*. 2015;93(6):496–504. https://doi.org/10.1111/aos.12628
4. Lai K, Reidy J, Bert B, Milman T. Spheroidal degeneration in H626R TGFBI variant lattice dystrophy: a multimodality analysis. *Cornea*. 2014;33(7):726–32. https://doi.org/10.1097/ICO.0000000000000140
5. Suárez MF, Correa L, Crim N, Espósito E, Monti R, Urrets-Zavalía JA, et al. Climatic Droplet Keratopathy in Argentina: Involvement of Environmental Agents in Its Genesis Which Would Open the Prospect for New Therapeutic Interventions. *Biomed Res Int*. 2015;2015:527835. https://doi.org/10.1155/2015/527835
6. Johnson GJ, Overall M. Histology of spheroidal degeneration of the cornea in Labrador. *Br J Ophthalmol*. 1978;62(1):53–61. https://doi.org/10.1136/bjo.62.1.53
7. Kaji Y, Oshika T, Takazawa Y, Fukayama M, Fujii N. Immunohistochemical localisation of D-beta-aspartic acid-containing proteins in climatic droplet keratopathy. *Br J Ophthalmol*. 2009;93(7):977–9. https://doi.org/10.1136/bjo.2008.144212
8. Serra HM, Suarez MF. Environmental proteinaceous corneal degeneration: A rare disease. *Rare Disease, India*. 2017.
9. Ashok N, Khamar P, D'Souza S, Gijs M, Ghosh A, Sethu S, et al. Ion channels in dry eye disease. *Indian J Ophthalmol*. 2023;71(4):1215–26. https://doi.org/10.4103/IJO.IJO_3020_22
10. Hua Z, Han X, Li G, Lv L, He X, Gu L, et al. Prevalence and associated factors for climatic droplet keratopathy in Kazakhs adults: a cross-sectional study in Tacheng, Xinjiang, China. *BMC Ophthalmol*. 2021;21(1):316. https://doi.org/10.1186/s12886-021-02065-4
11. Singh P, Tripathy K. Keratopathy. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025.
12. Kaji Y, Nagai R, Amano S, Takazawa Y, Fukayama M, Oshika T. Advanced glycation end product deposits in climatic droplet keratopathy. *Br J Ophthalmol*. 2007;91(1):85–8. https://doi.org/10.1136/bjo.2006.099812
13. Taylor HR. Climatic droplet keratopathy and pterygium. *Aust J Ophthalmol*. 1981;9(3):199–206. https://doi.org/10.1111/j.1442-9071.1981.tb01013.x
14. Mathur A, Chauhan A. A study of risk factors for Climatic Droplet Keratopathy in Elderly Persons from Western Rajasthan. *Sch J App Med Sci*. 2016;4(6C):2032–4.
15. Urrets-Zavalía JA, Knoll EG, Maccio JP, Urrets-Zavalía EA, Saad JA, Serra HM. Climatic droplet keratopathy in the Argentine Patagonia. *Am J Ophthalmol*. 2006;141(4):744–6. https://doi.org/10.1016/j.ajo.2005.10.044

16. Jauhonen HM. Effects of cis-urocanic acid on ocular surface. Publications of the University of Eastern Finland. Dissertations in Health Sciences, 436. 2017.
17. Lei Z, Beuerman RW, Chew AP, Koh SK, Cafaro TA, Urrets-Zavalia EA, et al. Quantitative analysis of N-linked glycoproteins in tear fluid of climatic droplet keratopathy by glycopeptide capture and iTRAQ. *J Proteome Res*. 2009;8(4):1992–2003. https://doi.org/10.1021/pr800962q
18. Menegay M, Lee D, Tabbara KF, Cafaro TA, Urrets-Zavalía JA, Serra HM, et al. Proteomic analysis of climatic keratopathy droplets. *Invest Ophthalmol Vis Sci*. 2008;49(7):2829–37. https://doi.org/10.1167/iovs.07-1438
19. Karnati R, Laurie DE, Laurie GW. Lacritin and the Tear Proteome as Natural Replacement Therapy for Dry Eye. *Exp Eye Res*. 2013;117:10.1016/j.exer.2013.05.020. https://doi.org/10.1016/j.exer.2013.05.020
20. World Health Organization. Global prevalence of vitamin A deficiency in populations at risk 1995–2005. Geneva: WHO Press; 2009.
21. Urrets-Zavalía JA, Maccio JP, Knoll EG, Cafaro T, Urrets-Zavalia EA, Serra HM. Surface alterations, corneal hypoesthesia, and iris atrophy in patients with climatic droplet keratopathy. *Cornea*. 2007;26(7):800–4. https://doi.org/10.1097/ICO.0b013e31806bef31
22. Pohjola S. Ocular manifestations of idiopathic hypoparathyroidism. *Acta Ophthalmol (Copenh)*. 1962;40:255–65. https://doi.org/10.1111/j.1755-3768.1962.tb02365.x
23. Holopainen JM, Serra HM, Sánchez MC, Sorsa T, Zalentein WN, Barcelona PF, et al. Altered expression of matrix metalloproteinases and their tissue inhibitors as possible contributors to corneal droplet formation in climatic droplet keratopathy. *Acta Ophthalmol*. 2011;89(6):569–74. https://doi.org/10.1111/j.1755-3768.2009.01764.x
24. Hagan S, Martin E, Enríquez-de-Salamanca A. Tear fluid biomarkers in ocular and systemic disease: potential use for predictive, preventive and personalised medicine. *EPMA J*. 2016;7(1):15. https://doi.org/10.1186/s13167-016-0065-3
25. Chun YH, Kim HR, Han K, Park YG, Song HJ, Na KS. Total cholesterol and lipoprotein composition are associated with dry eye disease in Korean women. *Lipids Health Dis*. 2013;12:84. https://doi.org/10.1186/1476-511X-12-84

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