Progressive Keratoconus in a Patient with Poorly Controlled Type 2 Diabetes Mellitus: A Three-Month Follow-Up Using Corneal Tomography

Introduction

Keratoconus (KC) is a progressive, non-inflammatory ectatic disorder of the cornea characterized by stromal thinning and irregular astigmatism. While diabetes mellitus (DM) is classically thought to alter corneal biomechanics, its role in keratoconus progression remains controversial. We present a rare case of bilateral keratoconus in a patient with poorly controlled type 2 DM, demonstrating notable topographic changes over a 3-month period.

Case Presentation

Patient: 39-year-old male
Medical History: Type 2 Diabetes Mellitus, poor glycemic control (HbA1c: 9.5% $\rightarrow$ 7.5% over 3 months), hyperuricemia.
Medications: Metformin 500 mg, Diamicron 120 mg, Atorvastatin 20 mg.
Ophthalmic Findings:
- Initial VA (22/11/2024): 20/60 (OD), 20/50 (OS) uncorrected.
- Refraction: OD -1.75/-1.50×65, OS -1.50/-1.75×50.
- Follow-up VA (13/2/2025): 20/60 (OD), 20/60 (OS) uncorrected.
- Corneal Topography: Progressive steepening and thinning noted in both eyes.
Tomographic Data:
- OD: Kmax increased, thinnest pachymetry ~498$\rightarrow$490 µm.
- OS: Kmax increased, thinnest pachymetry ~501$\rightarrow$489 µm.
Consistent with keratoconus-compatible patterns.

Retinal Findings

Retinal OCT: Normal macular thickness in both eyes with no signs of diabetic macular edema or retinopathy, supporting the cornea as the primary site of pathology.

Discussion

Although diabetes is associated with increased corneal cross-linking via non-enzymatic glycation of collagen, leading some to hypothesize a protective effect against KC progression, this case challenges that notion. The patient exhibited bilateral progression despite poor glycemic control and hyperglycemia-related biochemical changes. Potential explanations include chronic hyperglycemia causing microstructural corneal changes that weaken biomechanical stability, systemic metabolic stress contributing to extracellular matrix remodeling, and possible interaction between oxidative stress and advanced glycation end products (AGEs) in corneal stroma. This observation supports the importance of monitoring KC progression in diabetic patients, especially when glycemic control

is poor.

Conclusion

"This case highlights that poor glycemic control in type 2 diabetes mellitus does not preclude keratoconus progression. In fact, it may accelerate structural instability, warranting closer topographic monitoring and early intervention, even in patients traditionally considered at lower ectatic risk."

Key Takeaways

- Poor glycemic control does not guarantee protection from keratoconus progression.
- Serial tomography is essential in diabetic patients with KC.
- Further research is needed to elucidate the biomechanical interplay between hyperglycemia and corneal ectasia.