CHAPTER 3

MEIBOMIAN GLAND DYSFUNCTION AND DRY EYE DISEASE: MECHANISMS, DIAGNOSTICS, AND EVİDENCEBASED THERAPIES

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ABSTRACT

Dry eye disease (DED) is a multifactorial, symptomatic disease characterised by loss of tearfilm and ocular surface homeostasis, with tearfilm instability, hyperosmolarity, inflammation, tissue damage, and neurosensory abnormalities acting as central aetiologic factors. [1] Meibomian gland dysfunction (MGD) is the leading cause of evaporative DED, reflecting obstruction and/or qualitative changes in meibum that disrupt the lipid layer, accelerate evaporation, and destabilise the optical surface. [2,3] Population-based estimates suggest a global prevalence for DED between 5% and 50%, influenced by case definition, demographics, and environmental context. [4] A cause-based diagnostic approach integrating symptom indices with objective homeostasis markers and eyelid/blink related assessments improves precision in phenotyping and treatment selection. [1,5] Evidence supports tiered management from conservative care (warm compresses, lid hygiene, lipidenhancing drops) to device-based treatments (thermal pulsation, intense pulsed light) and selected pharmacologic/procedural options, with safety and durability profiles guiding shared decisionmaking. [2,6,7]

1. INTRODUCTION AND SCOPE

DED is recognised as a complex disorder sustained by a self-perpetuating cycle of instability and inflammation, often with neurosensory contributions that modify symptom perception. [1] Within this framework, MGD emerges as the prima-

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