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Bilateral Stromal Corneal Dystrophy Mimicking Central Visual Pathway Dysfunction: A Case Report

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ABSTRACT

Introduction: Progressive bilateral visual loss is a clinical red flag that frequently prompts evaluation for intracranial lesions involving the optic nerves, chiasm, or retrochiasmal pathways. However, anterior segment disorders such as corneal dystrophies may mimic central visual pathway pathology, especially when visual acuity loss occurs without overt ocular pain or inflammation.

Case Description: We report the case of a 31-year-old male presenting with progressive, symmetric visual impairment initially suspected to be neurologic in origin. Slit-lamp examination revealed bilateral mid-stromal crystalline opacities consistent with stromal corneal dystrophy.

Discussion: The absence of afferent pupillary defects, normal fundus findings, and lack of neurologic signs further supported a corneal etiology. This case illustrates the diagnostic challenge of distinguishing anterior segment disease from true neuro-ophthalmologic conditions. Failure to recognize corneal dystrophy may result in unnecessary neuroimaging and delayed treatment. Clinicians assessing bilateral vision loss, particularly in young adults, must include detailed anterior segment evaluation in their diagnostic algorithm to prevent mislocalization of pathology and ensure accurate management.

Conclusion:

Keyword: Cornea, Corneal Dystrophy, Bilateral Visual Loss, Neuro-Opthalmology, Keratoplasty.

1. Introduction

Progressive bilateral visual loss constitutes a major diagnostic concern in neurological and neurosurgical practice, as it frequently indicates pathology along the afferent visual pathways, including the optic nerves, optic chiasm, optic tracts, or retrochiasmal structures. Such presentations routinely prompt urgent evaluation for compressive intracranial lesions, inflammatory or demyelinating optic neuropathies, and vascular etiologies, given their potential for irreversible visual impairment if diagnosis is delayed. In this context, visual acuity decline, pupillary abnormalities, and visual field disturbances are commonly interpreted within a neuroanatomical localization framework to guide neuroimaging and surgical decision-making [1,2].

Despite this neurocentric approach, not all cases of bilateral visual loss originate from neural tissue. Disorders affecting the optical media may significantly attenuate visual signal transmission

before retinal or optic nerve processing occurs, thereby mimicking central afferent pathway dysfunction. Corneal opacification is a notable example, as bilateral corneal pathology can reduce visual acuity symmetrically and alter pupillary light reflex testing without true involvement of the optic nerve or intracranial visual pathways. Failure to recognize anterior segment disease at an early stage may therefore result in mislocalization of pathology, unnecessary neuroimaging, or inappropriate neurosurgical referral [3].

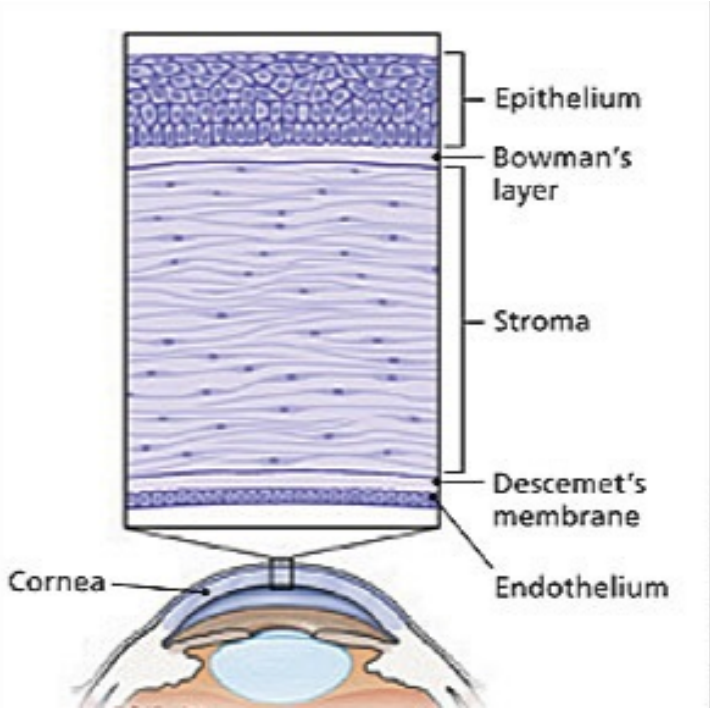

Figure 1. Histological structure of the cornea, illustrating its five principal layers: epithelium, Bowman's layer, stroma, Descemet's membrane, and endothelium

Corneal dystrophies are rare, genetically inherited, non-inflammatory disorders characterized by progressive deposition of abnormal material within specific corneal layers, leading to bilateral and often symmetric visual impairment. While endothelial dystrophies such as Fuchs endothelial corneal dystrophy are the most prevalent subtype, with reported prevalence up to 7.33% in adults over 30 years of age, stromal dystrophies including granular and lattice types are considerably less common, with prevalence estimates ranging from 0.09% to 0.13% in population-based studies. In young adults presenting with bilateral visual loss and unremarkable central nervous system evaluation, failure to consider corneal dystrophy may confound lesion localization and clinical decision-making. Consequently, systematic exclusion of anterior segment pathology remains a critical step in the neurosurgical assessment of suspected central visual dysfunction [4-6].

2. Case Series

A 22-year-old male The patient came with a complaint of weakness of all four limbs which was found since 1 day before the admission due to the patient jumping into the swimming pool with the back head hitting the bottom of the pool with a height of 1 meter. Weakness of all four limbs was felt simultaneously. Loss of consciousness was found after the incident with a duration of <5 minutes. Urination and defecation disorders were found since 1 day before the admission. Motoric score was 11111/22222 ; 11111/11111. MRI revealed cervical injury ASIA A at level C4-C5.

A 31-year-old male presented with progressively worsening bilateral visual acuity over the past four months. The symptoms were painless and symmetrical, raising concern for central visual pathway involvement such as compressive optic neuropathy, chiasmal lesion, or retrochiasmal dysfunction. The patient also reported photophobia, dry eyes, and a persistent foreign body sensation. There was no history of ocular trauma, systemic illness, or neurologic complaints, and no similar symptoms were reported in family members.

Neurological examination revealed intact extraocular movements, no relative afferent pupillary defect, and normal fundoscopy without optic disc edema or pallor. Visual acuity was reduced to 6/36 in both eyes. Given the bilateral nature and absence of clear retinal pathology, brain and orbital MRI with contrast was obtained. The imaging demonstrated no evidence of optic nerve enhancement, chiasmal compression, demyelination, or intracranial mass, effectively ruling out central causes of visual loss.

With central pathology excluded, anterior segment evaluation revealed bilateral, diffuse mid-stromal haze with multiple discrete, circular white opacities on slit-lamp examination. These lesions were confined to the stromal layer, with no epithelial or endothelial abnormalities (Figure 2 and 3). The findings were consistent with stromal corneal dystrophy, likely of the granular or lattice subtype. Due to local resource limitations, genetic testing was not pursued.

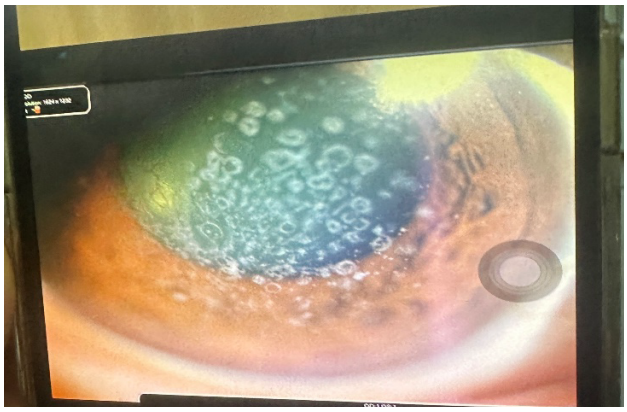

Figure 2. Slit-lamp photograph of the right eye showing mid-stromal opacities with ring-like crystalline deposits.

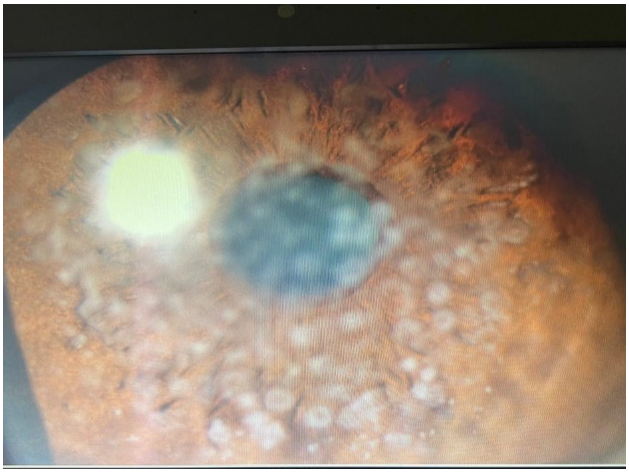

Figure 3. Slit-lamp photograph of the left eye demonstrating symmetrical mid-stromal lesions.

Management included preservative-free artificial tears for symptomatic relief and Ofloxacin 0.6% eye drops (1–2 drops, three times daily) to prevent secondary infection. The patient was counseled regarding disease progression and possible need for keratoplasty in the future. This case

illustrates the importance of considering anterior segment pathology in cases of bilateral visual loss and highlights the role of neuroimaging in differentiating corneal causes from central nervous system disease.

3. Discussion

Progressive bilateral visual loss in young adults often triggers a neurosurgical diagnostic workup aimed at excluding central visual pathway pathology, such as chiasmal compression, retrochiasmal lesions, or demyelinating optic neuropathy. These lesions frequently produce distinct patterns of visual field defects and may be associated with afferent pupillary defects or optic disc changes, prompting urgent neuroimaging. In this context, an accurate clinical localization strategy is paramount, because failure to correctly differentiate between central and peripheral causes may lead to unnecessary neurosurgical evaluation or delay in definitive care [7,8].

Nonetheless, not all bilateral visual loss reflects lesions within the central nervous system. Media opacities and anterior segment pathology, such as corneal dystrophies, may degrade the afferent visual signal before it reaches the retina, thereby reducing visual acuity in a way that *mimics* central visual pathway dysfunction. In this case, the absence of typical field defects and normal pupillary responses suggested that the neural visual pathways were likely intact, highlighting the importance of systematic exclusion of anterior segment causes early in the diagnostic algorithm. Electrodiagnostic testing, such as visual evoked potentials (VEP), can aid in distinguishing signal degradation due to optical media from true neural conduction delay, as VEP abnormalities with delayed latencies are characteristic of optic nerve dysfunction but not media opacity [9-11].

Corneal stromal dystrophies are hereditary, non-inflammatory disorders in which abnormal extracellular deposits impair corneal transparency and scatter light, leading to progressive bilateral visual loss without necessarily affecting visual field patterns or neural transmission. Stromal dystrophies such as granular, lattice, and macular dystrophy present with mid-stromal opacities that reduce image clarity but do not cause neurologic field defects. Because these anterior segment disorders can masquerade as neurologic visual loss in clinical presentation, integrating slit-lamp examination and anterior segment imaging into the early evaluation helps localize pathology and avoid misclassification as central nervous system disease [12,13]

Management of stromal corneal dystrophies differs fundamentally from that of central visual pathway disorders. While optic neuropathies or compressive lesions may require high-resolution imaging, corticosteroids, or surgical decompression, anterior segment pathology is managed through ocular surface lubrication, prophylactic antibiotics, or surgical procedures such as phototherapeutic keratectomy or keratoplasty in advanced cases. In clinical practice, a stepwise diagnostic framework that incorporates neurologic, neuro-ophthalmologic, and anterior segment evaluation can improve diagnostic precision, prevent unwarranted neurosurgical intervention, and ensure that patients receive timely and appropriate therapy [14,15].

4. Conclusion

In young adults presenting with bilateral visual loss, the reflexive consideration of central nervous system pathology such as chiasmal compression, optic neuropathy, or demyelinating disease is warranted but must be balanced against peripheral mimics like corneal stromal dystrophy. This case underscores the neurosurgical relevance of anterior segment disorders that degrade visual input

without altering the neural conduction pathways. Failure to identify such media opacities may lead to unnecessary neuroimaging, misinterpretation of visual evoked potential abnormalities, or delayed diagnosis. A structured neuro-ophthalmologic approach that integrates anterior segment examination is therefore critical to accurately localize the lesion, streamline diagnostics, and avoid inappropriate neurosurgical workup.

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