

Spontaneous Corneal Rupture of an Eye with Pellucid Marginal Degeneration, PMD

—A Case Report

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Abstract

Pellucid marginal degeneration (PMD) is the second-most common noninflammatory corneal degeneration after keratoconus. We report a case of a 42-year-old Saudi male who presented with spontaneous corneal perforation at the Pellucid Marginal Degeneration crescent in the left eye that healed with medical treatment. The perforation cornea was treated topically with cyanoacrylate glue, Bandage contact lens, moxifloxacin eye drops, and intra-ocular pressure lowering eye drops. The affected eye was followed daily until complete healing was observed without surgical intervention during this acute presentation. The PMD in the right eye was monitored for any progression.

Keywords

Pellucid Marginal Degeneration, Corneal Glue, Keratoplasty, Hydrops

1. Introduction

Pellucid marginal degeneration (PMD) is a noninflammatory ectatic corneal degenerative condition characterized by crescent-shaped thinning of the inferior cornea. It is the second most common cause of corneal ectasia after keratoconus [1]. PMD typically affects males aged 40 to 55 years [2]. Despite its significance, PMD is rarely reported in certain populations, such as Arabs, even though keratoconus—a related pathology—is prevalent in younger populations [3]. PMD is distinguished by a normal central corneal thickness and a crescent of thinning

located 1 - 2 mm from the limbus, sparing a peripheral rim of normal cornea [1].

Clinical Manifestations

Patients with PMD often present with a progressive decline in vision that is unresponsive to correction with spectacles [1]. Key clinical signs include:

- Inferior corneal thinning [1].
- Significantly reduced uncorrected visual acuity, which is not improved with spherocylindrical lenses but responds well to pinhole testing [1].
- Refraction and keratometry findings of irregular astigmatism that deviate from normal patterns [2].

Histologically, the condition is characterized by stromal thinning with intact epithelium, endothelium, and Descemet's membrane. Bowman's membrane is typically broken or absent at the crescent of thinning [4].

Complications

Although rare, PMD can lead to complications such as vertical stress lines, acute hydrops, and spontaneous corneal perforation, particularly following trivial trauma [1] [2]. Other possible findings include vascularization, scarring, Descemet's folds, and posterior stromal scarring, although the cornea typically remains clear with minimal neovascularization or lipid accumulation [1]. Notably, undergoing corneal refractive surgery in the presence of PMD increases the risk of ectasia and perforation, making its preoperative identification crucial [5].

Demographics and Specifics

PMD predominantly affects East Asian males in their fourth or fifth decade of life and is more frequently unilateral, as noted in 25% of Japanese cases [6]. In contrast to keratoconus and keratoglobus, PMD is not familial, and its genetic etiology remains unidentified [1]. In Arab populations, there is limited documentation of spontaneous corneal perforation and its management, despite the high prevalence of keratoconus-related perforations among Arab adolescents [3] [7].

Management

While surgical interventions are available, non-surgical approaches have shown promise in managing PMD and improving long-term visual outcomes.

1.1. Non-Surgical Management Approaches

1) Contact Lenses:

- *Rigid Gas Permeable (RGP) Lenses:* These lenses provide a smooth optical surface, effectively compensating for corneal irregularities. Studies have demonstrated that RGP lenses can significantly improve visual acuity in PMD patients. A study noted that "success has been shown with the use of rigid gas permeable contact lenses combined with over-refraction" [8].
- *Scleral Lenses:* A subset of RGP lenses, scleral lenses vault over the cornea, resting on the sclera. Their larger diameter provides enhanced comfort and stability. Recent findings suggest that scleral lenses may offer superior visual outcomes for PMD patients, with reports indicating "vision with these types of lenses is exceptional when fit correctly" [9].

2) Corneal Collagen Cross-Linking (CXL): CXL is a minimally invasive procedure that strengthens corneal collagen fibers, halting disease progression and improving visual acuity (in some cases). Long-term studies have shown its efficacy in PMD management. A retrospective study involving forty eyes of forty patients who underwent CXL for PMD found that “corneal collagen crosslinking in progressive PMD is a safe and effective long-term treatment for avoiding ectasia progression and also improves visual acuity” [10].

3) Customized Corneal Cross-Linking: Tailoring the CXL procedure to the individual’s corneal topography has yielded positive outcomes. A study reported that “corneal tomography showed stability and visual acuity improvement after customized crosslinking in all but one patient with a 1-year follow-up” [11].

1.2. Long-Term Outcomes

Non-surgical interventions, particularly specialized contact lenses and corneal cross-linking, have demonstrated favorable long-term outcomes for PMD patients. These treatments aim to stabilize the cornea, improve visual acuity, and enhance quality of life. However, individual responses can vary, and ongoing monitoring is essential to assess treatment efficacy and make necessary adjustments.

In light of these findings, we present a case of a 45-year-old male who presented with pain and left eye decreased vision. Comprehensive history and examination collectively confirmed a diagnosis of spontaneous corneal perforation of hydrops at the crescent of PMD and successfully managed medically without the need for acute surgical interventions. This report strictly adheres to the ethical principles delineated in the Helsinki Declaration.

2. Case Report

A Saudi male aged 45 presented in the emergency department of a military hospital with complaints of pain and dimness of vision in the left eye for a 1-day duration. The patient did not have any medical history of systemic comorbidity. A detailed history did not reveal any recent trauma, frequent rubbing, use of contact lenses, or use of topical medications. Five months before this emergency, he visited a private eye clinic and was informed that he had corneal disease in his weak left cornea, making it protrude. No medical or surgical treatment was given at that time.

The vision was assessed using Snellen’s distance vision chart held 6 meters from the patient. His uncorrected distance vision was 20/100 in the right eye and 20/400 in the left eye. The intraocular pressure was measured by an applanation tonometer mounted on a slit-lamp biomicroscope (Topcon, USA) and was 14 mmHg in both eyes. The right cornea showed significant inferior corneal thinning almost from limbus to limbus from 4 to 8 o’clock, with an area of corneal protrusion superior to the thinning. The left eye had mild circumcorneal congestion and acute hydrops with Descemet’s tear from the central to the periphery at 4 o’clock,

with corneal edema and area of descemetocele inferiorly extending to 5 o'clock with a leak and inferior corneal thinning with an area of protrusion superior to it. Occasional pigmented cells were found in the anterior chamber, and both eyes had a posterior subcapsular cataract. The pupil was normal size and quickly reacted to light. The posterior segment was evaluated using an indirect ophthalmoscope which showed normal results in both eyes.. Corneal topography as long with pachymetry were obtained with a provisional diagnosis of PMD in both eyes with left corneal spontaneous perforation at the PMD crescent with hydrops was made. The corneal topography findings of the right eye were as follow: K1: 33.7, K2: 43.1, K-max: 50.1, Thinnest location displaced inferiorly with thickness of 488 um, with a crab claw curvature map. The patient was admitted for observation for 6 hours. A bandaged contact lens (BCL) was applied to the left eye. The eye was treated with moxifloxacin eye drops four times a day, Pred Forte eye drops four times a day, brimonidine eye drops twice a day, and sodium chloride 5% eye drops four times a day.

We reassessed the patient the next day, and despite compliance with all the medications, there was no improvement in vision or pain. The anterior segment evaluation revealed acute hydrops with Descemet's tear from the central to the periphery at 4 o'clock with corneal edema and an area of descemetocele inferiorly extending to 5 o'clock with a leak and inferior corneal thinning with an area of protrusion superior to it. The anterior chamber had moderate depth.

The patient was moved to the operating theater. The BCL was removed, and corneal glue (N-butyl cyanoacrylate glue) was applied without any patch at the PMD crescent after the cornea was anesthetized. The medications were continued for 1 week, and the patient was reassessed daily. The glue at the perforation area in the inferior corneal crescent with PMD was stable without any infiltration or haziness around it. The anterior chamber was deep and well-formed. The patient also reported an improvement in pain and vision. His uncorrected distance vision was 20/100 in the right eye and 20/200 in the left eye compared to the initial visual acuity at presentation.

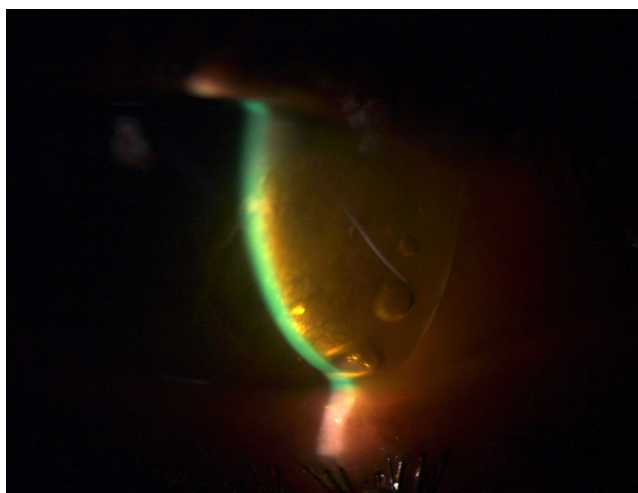
On evaluation 4 weeks after the presentation and management, the glue had dislodged, and the corneal hydrop was reduced in size and scarred. The anterior chamber was deep, and the fluorescein test showed no leaking.

The patient regularly followed up every month for the next 6 months, and recovery was uneventful. The patient underwent final surgical treatment with a large-sized corneal graft as a form of Penetrating Keratoplasty. The right eye then was monitored to ensure no progression of the thinned area. The Best corrected Visual acuity achieved in the left eye was 20/50. Patient expressed happiness, in which his life style and quality of his life have changed intensively.

3. Discussion

This is a case of bilateral PMD with spontaneous perforation and acute hydrops in the left eye. It was initially managed medically and then operated on for kera-

toplasty in the affected eye eye. The right eye thinning was closely monitored for any progression (See **Figure 1** and **Figure 2**).



Note. BCL is on top of the glued corneal hydrops post spontaneous perforation.

Figure 1. Photograph of left eye cornea under slit lamp biomicroscopic slit illumination and fluorescein stain.



Note. The thinning of the inferior peripheral cornea with clear cornea centrally.

Figure 2. Photograph of right eye cornea under slit lamp biomicroscopic slit illumination.

The PMD with perforation was clinically diagnosed. Several investigations are proposed to study further the extent of corneal damage and the presence of other ocular comorbidities. They include corneal topography, anterior segment optical coherence tomography, tear film evaluation, and limbal cell biology [12]. Such detailed case workup also helps differentiate PMD from conditions that cause corneal ectasia, like keratoconus, keratoglobus, and Terrien marginal degeneration.

In addition, they help surgeons decide the mode of management.

A detailed comparison between PMD and keratoconus reveals significant differences in clinical presentation and progression, despite both being corneal ectatic disorders. PMD predominantly affects the inferior peripheral cornea, resulting in crescent-shaped thinning 1 - 2 mm from the limbus, while the central cornea often remains unaffected in early stages. In contrast, keratoconus typically involves central or paracentral thinning, presenting as a conical protrusion of the cornea [13] [14]. Corneal topography further highlights these distinctions: PMD demonstrates a characteristic “crab-claw” or “butterfly” pattern, whereas keratoconus presents with an inferior steepening or irregular “bow-tie” pattern centered around the cone [14] [15].

Histopathologically, PMD is characterized by localized stromal thinning with no significant inflammatory response, whereas keratoconus exhibits disruptions in Bowman’s membrane, stromal thinning, and epithelial hyperplasia. Notable clinical signs in keratoconus, such as Fleischer rings (iron deposits) and Vogt’s striae (stress lines), are absent in PMD, underscoring the importance of meticulous examination [15].

The progression of PMD is generally slower than that of keratoconus; however, the risk of spontaneous perforation is higher in PMD due to peripheral corneal thinning. In advanced keratoconus, complications such as apical scarring and acute corneal hydrops are more common [14] [16]. Tear film abnormalities may exacerbate symptoms in both conditions, but keratoconus demonstrates a stronger association with atopic disorders and ocular allergies, highlighting potential differences in underlying pathophysiology [17].

Management strategies also differ. PMD is often managed conservatively with rigid gas-permeable (RGP) or scleral lenses for vision correction. Surgical interventions, such as crescentic lamellar keratoplasty or tectonic grafting, are reserved for severe cases with perforation. In contrast, keratoconus management includes spectacles or contact lenses in early stages, with collagen cross-linking being a cornerstone for halting progression. Advanced cases may require deep anterior lamellar keratoplasty (DALK) or penetrating keratoplasty (PK) [18] [19].

Imaging modalities like AS-OCT and pachymetry further aid differentiation by delineating the thinning patterns. PMD demonstrates inferior thinning with a relatively uniform central cornea, while keratoconus reveals localized thinning at the cone apex. These findings are critical for accurate diagnosis and surgical planning [19].

In conclusion, a detailed clinical and investigative workup is crucial for distinguishing PMD from keratoconus and other ectatic disorders. Understanding the nuances between these conditions not only enhances diagnostic accuracy but also informs individualized treatment approaches, ultimately improving patient outcomes.

In our case, we used synthetic glue to seal the corneal perforation without a patch. It helped prevent exogenous material, including organisms, from entering

the eyeball and supported the healed tissue on the hydrops under the BCL. It also provides rapid sealing of the perforation to prevent further damage, reduce the risk of endophthalmitis, and maintain structural integrity. The glue came off without any intervention after 6 weeks, and care was taken when removing the BCL so that the force did not detach this adhesion from surrounding corneal tissue. Both synthetic and natural tissue adhesives are used in different corneal conditions to seal perforations with varying degrees of success and side effects [20].

We used silicone hydrogel lenses as BCLs for this patient. The use of BCLs is recommended along with several medications to treat noninfectious corneal injuries and the underlying corneal pathologies. The BCL acts as a physical barrier, protecting the fragile glued area from blinking and external contaminants. It also helps to spread the tear film evenly over the cornea, promoting a conducive environment for epithelial healing [21]. We used antibiotics to prevent secondary infection and anti-inflammatory medications to lower intraocular pressure and prevent dryness of the corneal surface under the BCL.

A key rationale for using synthetic glue and BCL is their role as a temporary but effective bridge therapy. While definitive surgical interventions, such as lamellar or penetrating keratoplasty, may ultimately be required, these procedures often necessitate a delay to allow for stabilization of the cornea, resolution of inflammation, or optimization of the recipient bed. The use of synthetic glue and BCL provides a period of stability, preserving the ocular anatomy and function until the eye is ready for more invasive treatment [22].

Challenges and Considerations

While synthetic glue and BCL offer numerous benefits, certain limitations must be considered:

- **Glue Dislodgement:** Particularly in cases with high lid tension or improper application, repeated procedures may be required.
- **Long-Term Complications:** Potential issues include delayed epithelial healing, corneal neovascularization, and secondary glaucoma if the glue is misapplied or left in place for extended periods [23].
- **Case Selection:** This approach is most suitable for small perforations (≤ 3 mm). Larger defects may necessitate immediate surgical intervention.

In the recent case, crescentic lamellar keratoplasty (CLK) was performed with both visual and anatomic success after 6 months. Surgical management of PMD described in the literature includes intrastromal corneal rings, collagen cross-linking in eyes without limbal stem cell deficiency, deep anterior lamellar keratoplasty, CLK, lamellar thermokeratoplasty, epikeratoplasty, crescentic lamellar wedge resection, tuck-in lamellar keratoplasty, penetrating keratoplasty, and full thickness wedge resection [24]. When selecting a surgical option, one should consider the status of the corneal stroma around the PMD crescent, the experience of the surgeon, and other ocular comorbidities in the affected eye. In the present case, there was a subcapsular cataract, so we planned a subsequent cataract surgery with the insertion of a toric intraocular lens.

4. Comparison of Keratoconus and PMD Perforations

Pellucid marginal degeneration seems to be an uncommon occurrence. In a review of 1113 individuals with corneal ectasias, Tummanapalli *et al.* discovered that 97% of the patients had keratoconus and just 3% had PMD. While keratoconus appears to be more common in males, PMD does not appear to differ in frequency across genders; in fact, it appears to be roughly equal. Patients with keratoconus are often discovered in their early twenties or adolescents (mean \pm 12 years), but those with PMD appear to be considerably more elusive, with a mean age of discovery of 37 years (mean \pm 14 years) [25]. In the present case, PMD was a bilateral ailment. In a large series in India, three-fourths of 559 patients with PMD had bilateral affection [26]. Although perforation is not an eventual catastrophe in all PMD cases, lifelong close monitoring is recommended for both eyes.

The prevalence of keratoconus in the young Saudi population is higher than in other populations [27]. PMD is a condition with unknown etiology but similar pathology. It is worth noting that older patients visiting the ophthalmic clinic during the corneal examination of cases. Some geographical cues might help reduce PMD misdiagnosis:

- 1) Keratoconus is more likely to cause mixed or hyperopic astigmatism.
- 2) The spherical equivalent is often more negative in PMD.
- 3) The extent of asphericity. The “Q” value (negative in normal) has greater sensitivity and specificity than the standard “e” value. Positive “Q” values suggest an oblate profile [28]. Thinning of the cornea in the inferior quadrant and having a healthy corneal margin could be highlighted in optometrist training in countries where they provide primary eye care. The residents and cornea subspecialists also should be trained to handle emergency ocular perforations and undertake detailed assessments to rule out PMD.

This study is based on a single-case report, which limits the generalizability of the findings. The conclusion drawn from one patient cannot be extrapolated to the broader population of individuals with PMD. Moreover, differences in treatment methods and clinical practices among patients can affect the outcomes. In addition, conducting the study at a single center may limit the generalizability of the results. Multi-center studies are necessary to ensure that the findings are applicable to a broader population. Standardized treatment protocols would help compare and analyze results more effectively.

5. Conclusion

Spontaneous corneal perforation inferior to the center of the cornea without a history of trauma and signs of inflammation should raise an alarm, and an ophthalmologist in the emergency department should look for PMD in both eyes. Once the diagnosis is confirmed, a medical line of treatment with glue and a BCL should be quickly initiated, followed by surgical treatment and lifelong follow-up.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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