

# Salzmann nodular degeneration after photorefractive keratectomy for hyperopia

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## Abstract

**Purpose:** To report a case of bilateral central Salzmann nodular degeneration (SND) after photorefractive keratectomy (PRK) for hyperopia.

**Methods:** Case report.

**Results:** A 47-year-old male presented with complaints of glare and difficulty with night driving. He had undergone PRK for hyperopia (+3.5+1.5x180 in the right eye and +4.5+1.0x180 in the left eye) in both eyes about 15 years back. The early postoperative period was uneventful; however, he started having these symptoms after two to three months. At presentation, uncorrected visual acuity was 20/100 and best corrected visual acuity was 20/30 with +3.5+1.0x180 in both eyes. Slit-lamp examination showed bilateral 1-1.5 mm diameter, bluish white nodular lesions involving the central corneas. There were also prominent paracentral brown pigment ring deposits consistent with pseudo-Fleischer rings in both eyes. Scheimpflug images showed dense hyper-reflective nodules that were associated with elevated anterior corneal surface and measured 290  $\mu\text{m}$  in the right eye and 230  $\mu\text{m}$  in the left eye, along with presence of significant astigmatism (K1- 45.1D, K2- 47.8D right eye and K1- 45.4D, K2- 49.2D left eye). Central pachymetry was 485  $\mu\text{m}$  in the right eye and 464  $\mu\text{m}$  in the left eye.

**Conclusions:** SND is a rare complication of PRK for hyperopia that can lead to suboptimal visual outcome. PRK should be included in the list of etiologies leading to SND.

**Key words:** Salzmann, degeneration, photorefractive keratectomy

## Introduction

Salzmann nodular degeneration (SND) is a rare, non-inflammatory, degenerative condition of the cornea that is characterized by formation of usually bilateral, symmetrical, bluish-white elevated nodules on the anterior surface of cornea.<sup>1-3</sup> The etiopathogenesis of this condition is still not clear, however, it is usually associated with chronic, recurrent corneal disorders like meibomian gland dysfunction, trachoma, phlyctenulosis, keratoconus, interstitial keratitis, vernal keratoconjunctivitis, measles, scarlet fever and other viral diseases.<sup>3-5</sup> SND has also been reported to develop after laser in situ keratomileusis (LASIK) surgery, pterygium surgery, cataract extraction, hard contact lens use and ocular trauma.<sup>4-7</sup> Reported here, is a case that developed SND after photorefractive keratectomy (PRK) for hyperopia, a complication, which has not been previously reported in literature.

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## Case report

A 45-year-old-male presented with complaints of glare and difficulty with night driving. He had undergone PRK for hyperopia in both eyes 15 years back elsewhere. Pre-op refraction was +3.5+1.5x180 in his right eye and +4.5+1.0x180 in his left eye. The details of the surgical procedure were not available. The immediate postoperative period was uneventful; however, he started having these symptoms after two to three months along with regression of his refractive error. The patient did not have any history of atopic disease or tendency for keloid formation and did not have any systemic illness. At presentation, uncorrected visual acuity was 20/100 and best corrected visual acuity was 20/30 with +3.5+1.0x180 in both eyes. Slit lamp examination showed 1-1.5 mm bluish-white nodules, one in each cornea, involving the central cornea with mild elevation on narrow slit illumination (Fig. 1). There was associated paracentral brown pigmented deposits, consistent with pseudo-Fleischer rings, in the corneal epithelium along with faint haze in the superficial paracentral stroma in both eyes. There was no sign of previous keratitis or associated blepharitis. Ocular surface staining test with fluorescein was negative. The remaining ocular and systemic examination was unremarkable. Scheimpflug images with Pentacam (Oculus, Wetzlar, Germany) showed dense, hyper-reflective nodules that were associated with elevated anterior corneal surface and measured 290  $\mu\text{m}$  in the right eye and 230  $\mu\text{m}$  in the left eye (Fig. 2). Sagittal sections showed irregular steepening of central corneas in both eyes with significant astigmatism (K1- 45.1D, K2- 47.8D right eye and K1- 45.4D, K2- 49.2D left eye). The corneal thickness at the center of the pupil was 485  $\mu\text{m}$  in the right eye and 464  $\mu\text{m}$  in the left eye. The patient was counselled about surgical options including superficial keratectomy with application of mitomycin C and phototherapeutic keratectomy; however, he refused any surgical intervention.

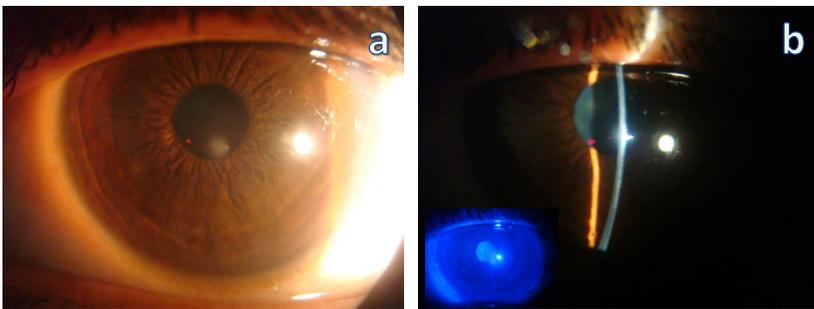


Fig. 1. Slit-lamp picture of left eye shows bluish-white nodular opacity in the papillary area (a), with anterior surface elevation on narrow slit beam illumination (b). Inset (b, bottom left) shows paracentral pseudo-Fleischer ring under cobalt blue filter illumination.

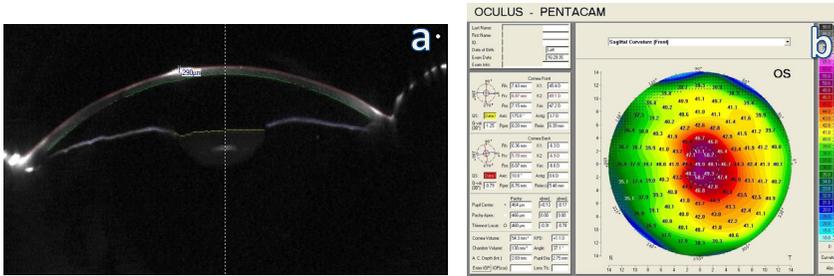


Fig. 2. Scheimpflug image of left eye shows dense hyper reflective nodular lesion measuring 290  $\mu\text{m}$  (a). Sagittal section shows irregular steepening in the central part of cornea corresponding with the site of the nodule (b).

## Discussion

PRK has been found to be a safe and predictable procedure for the correction of mild to moderate hyperopia and involves steepening of the central curvature by flattening the peripheral curvature by laser correction.<sup>8-9</sup> Common complications of this procedure include loss of corneal transparency due to haze formation, refractive error regression, and formation of surface irregularities.<sup>10</sup> Formation of central corneal haze, a well-known complication after PRK for myopia, is not seen in hyperopes. O'Brart *et al.* did not see loss of central corneal transparency in any of the 40 eyes with 7.5-years follow-up.<sup>9</sup> However, they reported peripheral ring of haze and sub-epithelial iron ring deposition, which was present in our case too. Although we could not find any previous report of SND after PRK, we believe it might be a case of using different nomenclature to describe this condition. Stakheev *et al.* reported one case of bilateral apical nodular sub-epithelial scar leading to irregular astigmatism in their study of 98 eyes of 52 patients who underwent PRK for hyperopia.<sup>8</sup> Although the clinical picture of our patient is similar to this case, a definite comparison could not be made as photograph of that case has not been published. Sener *et al.* reported 12 eyes of six patients with apical sub-epithelial nodular scars in eyes that had hyperopic PRK retreatment.<sup>11</sup> Comparison of their published photographs shows similarity with the current case. It is interesting that these nodular opacities were not labelled as SND. The reason for this could be lack of definite distinguishing features between these two entities in ophthalmic literature.

SND is usually considered an idiopathic condition associated with chronic inflammatory ocular surface diseases. However, it has also been reported after refractive surgeries like LASIK.<sup>4-6</sup> The pathogenesis of SND is not clear; however, previous authors have speculated that these nodules develop from chronic ocular surface irritation, uneven surfaces, corneal exposure and secondary to tear film disturbances.<sup>5-6</sup> In their cases of SND after LASIK, the patients showed clinical features of dry eye in the postoperative period. Although our patient did not show any evidence of dry eyes at presentation, its presence in early postoperative period cannot be ruled out. We speculate that SND in the current case could be the result

of aberrant wound healing response of the cornea to the surgical trauma. Interestingly, the central corneal thickness of our case was towards the lower side, but its role in SND formation could not be established. It can be argued that the current case could be labelled as a hypertrophic scar and not SND. However, ophthalmic literature is not very clear on distinction between these two entities. We believe these entities have strong analogy to dermatological conditions of keloid and hypertrophic scar. However, in dermatology literature, keloids and hypertrophic scars have been shown to be having different characteristics on histology and immunochemistry.<sup>12</sup> A similar study on human corneas can provide further insight into this pathology. At present, the diagnosis of SND is clinical due to wide variation in the pathology of these nodules.<sup>13-14</sup> Various imaging modalities like optical coherence tomography (OCT) have proved to be helpful in the *in-vivo* analysis of the morphological characteristics of this pathology.<sup>6-7</sup> Scheimpflug images of the current case revealed nodular elevation of the anterior surface, significant astigmatism and allowed measurement of the nodules, which shows that it can be a useful imaging modality in analysis of this pathology. Although we could not perform histopathology on the current case, previous studies have shown a strong correlation with OCT findings.<sup>7</sup>

The conservative medical management of SND includes topical lubricants, warm compresses ocular hygiene, topical non-steroidal anti-inflammatory therapy, topical steroids and oral doxycycline.<sup>1</sup> However, these topical and systemic measures are unsatisfactory in achieving cure of the nodules and surgical treatment may be indicated in symptomatic cases. Various surgical modalities described in literature for treatment of SND include manual removal or nodulectomy with or without topical mitomycin C application, superficial keratectomy with or without amniotic membrane transplantation, nodulectomy followed by excimer laser phototherapeutic keratectomy to smoothen the corneal surface, cryotherapy and lamellar keratoplasty or penetrating keraoplasty.<sup>1,3,15</sup> Although our patient was counselled about these options, he did not want any surgical intervention at the time.

## Conclusion

SND is a rare complication of PRK for hyperopia that can lead to suboptimal visual outcome. PRK should be included in the list of etiologies leading to SND.

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