Corneal epithelial hyperplasia masquerading as ocular surface squamous neoplasia

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Key words: Keratitis, ocular surface squamous neoplasia, OSSN

Corneal ocular surface squamous neoplasia (OSSN) commonly present as grayish, avascular superficial corneal opacity with feathery margins and minimal limbal involvement.[1] We describe an unusual presentation of corneal epithelial hyperplasia masquerading clinically as an OSSN. A 45-year-old male presented to ocular oncology services with blurred vision in left eye of 5 months duration. His best-corrected visual acuity (BCVA) was 6/6, right eye and 6/18, left eye. At presentation, slit lamp microscopy showed [Fig.1a and b] grayish avascular growth involving inferior and central cornea with typical feathery edges. No epithelial discontinuity was noted on fluorescein staining. Anterior segment optical coherence tomography (ASOCT) showed the involved epithelium to have increased reflectivity, and an abrupt transition from the normal uninvolved epithelium [Fig. 1c]. The epithelial thickness was increased by 10 microns only, inconclusive of OSSN.[2] He was started on topical IFNa2b 1MIU/ml and minimal change was noted after 3 months of immunotherapy. With clinical diagnoses of pseudo-epitheliomatous hyperplasia (PEH) or OSSN, alcohol keratoepithelectomy was performed. Histopathology revealed squamous metaplastic corneal epithelium with parakeratosis. The epithelium showed reactive changes, mild nuclear enlargement, with patchy presence of peri nuclear halo [Fig. 1e and f]. Occasional superficial cell (arrow marked) showed mild nuclear hyperchromasia, with definite and distinct peri nuclear cytoplasmic clearing. There was no dysplasia. The lesion resolved completely following surgery and showed no recurrence [Fig. 1d]. No viral DNA of herpes simplex or human papilloma virus could be detected with polymerase chain reaction.

Discussion

Corneal epithelial hyperplasia has many etiologies and includes response to toxic irritants applied topically, microbiological infections, nutritional deficiencies, heritable defects, and exophthalmos due to space-occupying orbital masses. These are usually associated with stromal inflammation and neovascularization. The current case had neither

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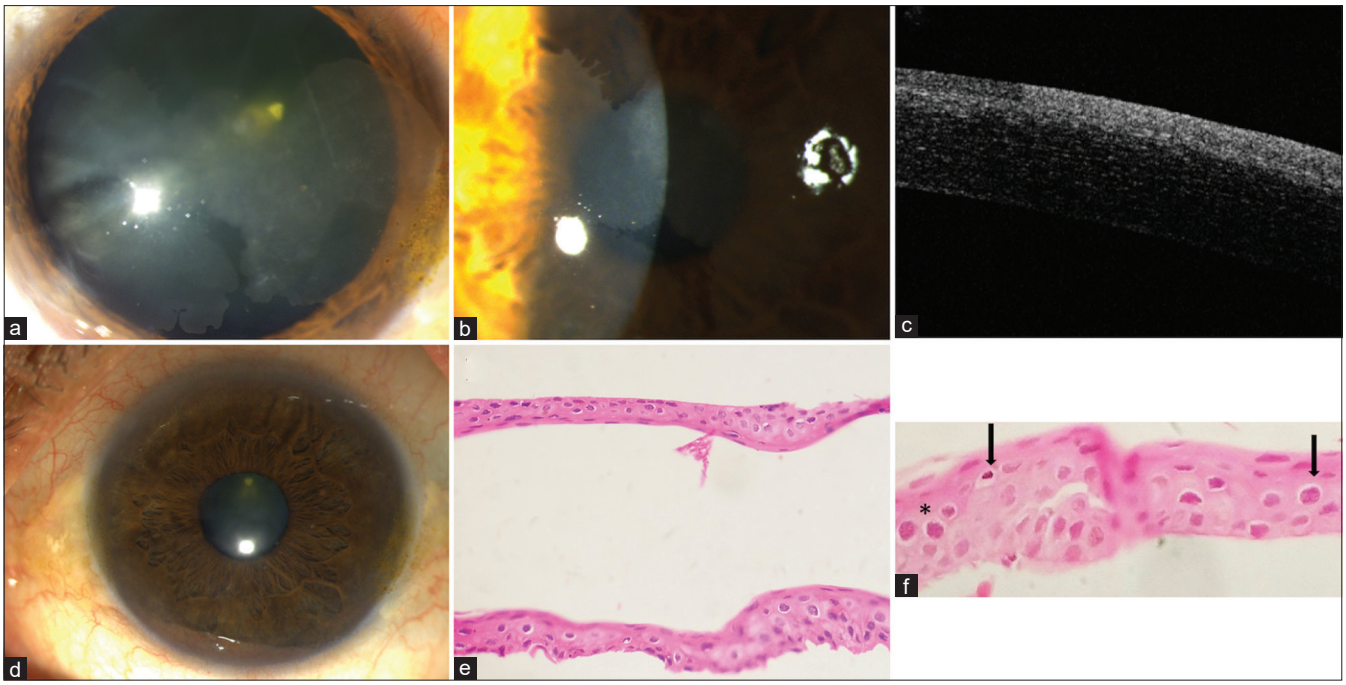

Figure 1: (a and b), Slit lamp photograph shows grayish avascular lesion involving inferior paracentral cornea with fimbriated borders. (c), ASOCT displays hyper-reflective epithelium with abrupt transition from the uninvolved epithelium. (d), Slit lamp image of clear cornea following keratoepithelectomy. (e and f), Photomicrograph (×20 objective) shows squamous metaplastic epithelium with dispersed cells displaying perinuclear cytoplasmic clearing (asterix marked; ×40 objective) and nuclear hyperchromasia and perinuclear halo (marked with arrow). There was absence of cytological atypia

vascularization and nor inflammation, which made us think of OSSN as our first differential diagnosis. The exact etiology in the current case remains unknown.

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Conflicts of interest

There are no conflicts of interest.

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