DEB/LP/VORI=SCC/KA

Med Derm Society Annual Meeting
Pearls of Wisdom: Case Presentation

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Objectives

- Pitfall: hypertrophic LP diagnosed as SCC
- Triggers of KA-like proliferations
- ?Speculative pathogenic mechanism
- Treatment of “SCC/KA” medically
Unobjectives

- Solve the KA/SCC debate
History

- 67-year-old woman with multiple keratotic nodules on the legs
- Biopsies read as “squamous cell carcinomas”
KA-Like Lesion in Hypertrophic LP

- Caucasian women > 60 years with extensive sun damage on legs
- Lesions may be of sudden onset
- Only KA/SCC-like lesions are seen initially
- Lesions of LP may not be evident
- Drug induced—HCTZ, pravastatin, beta blockers.
Hypertrophic Lichen Planus–Like Reactions Combined With Infundibulocystic Hyperplasia

Pathway to Neoplasia

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Figure 1. Patient 1. A, Violaceous plaque with keratotic rim on the left leg.
B, Skin biopsy specimen demonstrating infundibulocystic hyperplasia with lichenoid inflammation and irregular lobules of keratinocytes penetrating into the mid dermis (hematoxylin-eosin, original magnification ×40).
C, Violaceous lichen planus–like reaction on the right leg at sites of cryotherapy. D, Appearance of the left leg 1 year after commencing treatment.
KA in Hypertrophic LP
Medical Treatment

- Stop triggering medication
- Topical Steroids
- Topical Steroids with Occlusion
- Intralesional Steroids
- Antimalarials
- Intralesional 5FU to refractory lesions
- Oral Retinoids
Triggers of squamoproliferative lesions—KA/SCC

- LP/DLE
- Voriconazole-associated photosensitivity
- Chronic wounds
- Dystrophic EB
- Trauma
- Prurigo nodularis
Dermis and SCC/KA

- The dermis is important in development and behavior of squamoproliferative lesions.
- It’s not just the seed—it’s also the soil it grows in.
Mediated in part by Insulin-like growth factor (IGF) secretion by fibroblasts and IGF receptor expression on keratinocytes

IGF-R binding of IGF by keratinocytes is pro-apoptotic (pathway to get rid of DNA-damaged keratinocytes)

Fibroblasts are deficient in severely sun-damaged skin

Geriatric fibroblasts have reduced IGF production

Bottom line: If your dermis/BMZ is not good, you can grow epidermal neoplasms
AND

- Retinoids increase expression of IGF-R on keratinocytes
- Stopping inflammation along the BMZ may allow the IGF--IGF-R binding to work
- Making new fibroblasts may control squamoproliferative lesions (that patient who had the horrible reaction to 5FU and never got an AK/SCC in that area)
- Fractionated laser/TCA/Dermabrasion for areas of multiple SCC’s
What I learned in a Decade
And you in 15 minutes

- KA’s appear with hypertrophic LP and the treatment is to treat the LP
- SCC/KA occurs in conditions that damage the BMZ/upper dermis
- The Dermis is important in determining the generation and behavior of squamoproliferative lesions (think of your garden)
References


Thank you!
KA and Hypertrophic LP

*Br J Dermatol* 2003, 148:592

- Are these KA’s reactive? part of the LP? or SCC’s with limited progression potential
- Impossible to know
Figure 2. Patient 2. A, Ulcerated nasal columella with infiltrated violaceous plaque extending to the upper lip. B, Skin biopsy specimen of plaque revealed multiple dilated follicles outlined by prominent lymphocytic infiltrate resembling lichen planopilaris. C, Skin biopsy specimen from right nasal ala showing marked infundibulocystic hyperplasia, irregular cords, and lobules of keratinocytes penetrating through the full depth of the biopsy (hematoxylin-eosin, original magnification ×40 [B and C]). D, Appearance after 4 months of taking acitretin.

Figure 3. Patient 3. A, Hypertrophic verrucous nodules covering a skin graft on the left leg. B, Biopsy specimen from the left leg demonstrating marked infundibulocystic hyperplasia, with irregular buds of epithelium extending to base of the specimen with lymphocytic reaction. C, Biopsy specimen from nodule in graft on the right leg with a central cavity and series of irregular lobules of keratinocytes penetrating the mid dermis with lymphocytic inflammation (hematoxylin-eosin, original magnification ×40 [B and C]). D, Appearance of the left graft site 10 months after commencing treatment with acitretin.

Acitretin was continued for 20 months, and the area re-