

Innovations in Diagnosis: Precancer to Skin Cancer, Pathology, and CPC Cases

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In the development of actinic keratoses (AKs) and squamous cell carcinomas (SCCs), keratinocytes are exposed to ultraviolet B rays and develop DNA lesions called cyclobutane thymine dimers, leading to mutations and neoplastic formation. The p53 and p16 genes are of prime importance with regards to this mechanism. Cyclobutane dimers in the skin can be stained and viewed under histopathology. Langerhans cells also decrease in number and ability to properly function. Background keratinocytes help keep lesions in check, but eventually the outer layer of the epidermis becomes thickened and scaly. Cells can become numerous and replace areas of epidermis and expand into the dermis and eventually spread into lymph or blood vessels throughout the body.

AKs and SCCs demonstrate the same type of genetic change – the cytology of both look the same and consist of thickened epidermis, large cells with pinkish neoplasm, atypical mitotic figures and necrotic cells. Lesions are focal and initially spare the follicle. A more diffuse AK with involvement of epidermis and buds impinging down to papillary dermis for full thickness is suggestive of a developing SCC. About 90% of SCCs have contiguous AKs, and it is estimated that 13-20% of patients with AK will develop an SCC over time. Metastatic SCC is the most common cause of death in transplant patients. There is a classical pathway and differentiated pathway that an AK may take in becoming an SCC; it is impossible to tell which pathway an AK may pursue. AKs are like miniature SCCs confined to epidermis, there is no precancer.