Causes and Treatment of Benign Hyperkeratotic Lesions

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Description

Many pathologists refer to frictional keratoses and genuine leukoplakias without epithelial dysplasia as hyperkeratosis and acanthosis (benign epithelial hyperplasia). The common benign alveolar ridge keratoses on the retromolar pad and lesions of persistent frictional keratosis from parafunctional behaviors (cheek biting or chewing) all represent frictional keratoses and will also show hyperkeratosis and acanthosis. As a result, when a clinician receives a report of "hyperkeratosis, acanthosis, or epithelial hyperplasia" without any further explanation, it is considered as the lesion that may be a genuine leukoplakia with the potential to develop dysplasia or invasive cancer, or it may be a completely benign lesion brought on by friction. When such lesions are employed in a leukoplakia study instead of controls but rather as lesions of real leukoplakia, maybe early or mild dysplasia and the results will be contaminated. In fact, numerous publications have classified these frictional hyperkeratosis lesions with epithelial hyperplasia as "leukoplakia."

There are many things to think about. Firstly, there is only, utmost modest interexaminer agreement across pathologists when it comes to the diagnosis of dysplasia. The discordance is more pronounced in mild dysplasia cases than in moderate or severe dysplasia, as would be expected. The "reactive epithelial atypia," or epithelial alterations subsequent to reaction to injury or inflammation, may be responsible for the modest or focal epithelial changes seen in mild dysplasia. This can be seen in oral lichen planus biopsies or along the ulcer's margin. On the other hand, reactive epithelial atypia may be mistaken for dysplasia. For this reason, some pathologists now distinguish between low-grade and high-grade dysplasia, with high-grade dysplasia allegedly having a higher propensity to develop into invasive cancer. It is acknowledged that reactive atypia may be challenging to distinguish from low-grade lesions or mild dysplasia. The examination of dysplasia must also take into account of architectural defects such as verrucous configuration without signs of cytologic dysplasia. Furthermore, a single biopsy from a big or non-homogenous clinical lesion might not be typical. Underdiagnosis from a single biopsy against several biopsies was 29.5% and 11.9%, respectively, in the study by Lee et al. that looked at 200 cases. Invasive carcinoma was more common in the resection specimen (12.0% *vs.* 2.4% in single *vs*. multiple biopsy instances). Thirdly, it's possible that leukoplakic regions that histopathologically exhibit keratosis or hyperkeratosis but show minimal signs of cytologic abnormality may reflect the very first changes in carcinogenesis. The histopathologic changes are those of hyperkeratosis and verrucous epithelial architecture in two clinically recognized entities, verrucous leukoplakia and proliferative verrucous leukoplakia, with little to no evidence of epithelial dysplasia. Hansen et al. first identified the clinicopathologic condition known as proliferative verrucous leukoplakia in 1985.

Conclusion

Leukoplakia cases that were "slow-growing, chronic, irreversible, and frequently acquired erythematous components" were identified by the researchers. According to studies, invasive carcinoma will develop in 40%-70% of these lesions during the course of a long-term follow-up. It is extremely likely that all three of these elements contribute, in varying degrees, to the development of invasive carcinoma from so-called "benign hyperkeratosis." Clinically, leukoplakias and homogeneous leukoplakias in particular-are primarily sharply bordered plaques between the keratotic area and the surrounding normal mucosa, at least for a portion of the lesion. While not always present, this characteristic is typically seen in homogeneous leukoplakia and less frequently in erythroleukoplakia. Additionally, homogenous leukoplakia frequently exhibits superficial surface fissures. Numerous genetic alterations have been identified at the molecular level in investigations; however, none of them have consistently been linked to dysplasia. Mild atypia is seen in cases of dysplasia but however, it is uncertain whether this is caused by reactive or frictional keratoses in these cases.

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