

Retrospective histological assessment of oral keratotic white lesions in Department of Oral Medicine

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Introduction

Diagnosis of oral white lesions could be quite challenging. These lesions represent a good spectrum of lesions with different etiology and various prognoses. The diagnosis of white lesions varies from benign reactive lesions to more serious dysplastic and carcinomatous lesions. While there are some classic features that help distinguish these lesions, similar features may produce to some complications in diagnosis. Efforts should be made to determine a particular diagnosis to stop time elapse in treatment of patients with more serious lesions. a choice tree may be a flowchart that organizes features of lesions so as to assist clinicians to succeed in a logical conclusion. To use the choice tree, one should begin from the left side of the tree, makes the primary decision, and proceeds to the far right of the tree where the definite diagnoses are listed. Oral lesions are often classified into four groups comprising of ulcerations, pigmentations, exophytic lesions, and red-white lesions [2]. Although white lesions constitute only 5% of oral pathoses, a number of these lesions like leukoplakia, lichen ruber planus, and proliferative verrucous leukoplakia have malignant potential as high as 0.5–100% [3]. Therefore, white lesions mandate an appropriate clinical diagnostic approach to exclude the likelihood of malignancy. The onset of oral white lesions are often acquired or congenital, with a history of long-lasting existence within the latter form. Oral white lesions are often caused by a thickened keratotic layer or an accumulation of non-keratotic material. Accordingly, when a clinician confronts a white area on the oral mucosa, the primary issue to be elucidated is whether or not it are often scraped off by means of a bit of gauze or not. If so, a superficial non-keratotic layer like pseudomembranes, most ordinarily caused by fungal infections or caustic chemicals, should be suspected. Otherwise, white lesions are often attributed to increased thickness of keratin layer, which could be induced by local frictional irritation, immunologic reactions, or more crucial processes like premalignant or malignant transformation. In the next step, any specific clinical pattern of white lesions like papular, annular, reticular or erosive-ulcerative patterns, or a mixture of them (characteristic for lichenoid lesions) should be inspected so as to differentiate white patterned lesions from non-patterned ones. Therefore, this narrative review paper focuses on three clinical steps to approach oral white lesions: the primary step is to work out whether the lesion is congenital or acquired; the second and third steps are to examine if it are often wiped off or not and if it's a special pattern or not.

This diagnostic process is presented as an updated clinical decision tree. a choice tree may be a flowchart used for organizing features of lesions or diseases that help clinicians make a constellation of rational decisions instead of haphazard ones to succeed in a conclusive diagnosis.

White sponge nevus (WSN), also called Cannon disease or familial white folded dysplasia, is an inherited autosomal dominant disorder that is defined as dyskeratotic hyperplasia of mucous membranes [2]. WSN is a rare condition with no sex predilection [8]. A prevalence of below one in 200,000 population has been reported [4]. The lesions generally present at birth or early in childhood, but sometimes the condition appears during adolescence. Mutations in keratin genes are responsible for coding of epithelial keratin types K4 and K13 results in lack of normal keratinization. Both intraoral and extra oral mucosal sites might be involved. Intraoral lesions are symmetrical, thickened, white, corrugated or velvety, diffuse, spongy plaques of variable sizes with an elevated, irregular, and fissural surface. Buccal mucosa is affected bilaterally in most patients. Other areas of the oral cavity such as the ventral surface of the tongue, labial mucosa, soft palate, alveolar mucosa, and floor of the mouth can also be affected, but the amount of involvement might vary from patient to patient. Extraoral sites include nasal, esophageal, laryngeal, and anogenital mucosa; however, their involvement is relatively unusual in the absence of oral manifestations. White sponge nevus can cause dysphagia when the esophagus is involved; otherwise, the lesions are asymptomatic. Due to the benign nature of the lesion, good prognosis, and infrequent recurrence rate no treatment is suggested for WSN.

Background: Oral keratotic white lesions are quite common in oral diagnostic section in Myanmar. Biopsy is a gold standard for early detection of oral cancer is important for patient's life. **Aim:** To detect histological variants of biopsy specimens that appear as oral keratotic white lesions. **Material & Methods:** Biopsy specimens which are written as keratotic white lesions in pathological requisition forms and sent to Department of Oral Medicine, University of Dental Medicine, Mandalay from 2014 to 2019 (n=65) are retrospectively studied by hematoxylin and eosin stain. Clinical data are obtained from pathology request forms.

Results: Age range is from 20 to 77 years. Common age is 51 to 60 years age range (23/65) (35%). Male to female ratio is 3.6:1. Sites of distributions are right buccal mucosa (42%), left buccal mucosa (28%) and tongue (14%). Of the 65 cases, 37 cases (57%) are diagnosed as oral leukoplakia of which 21(57%) are mild

oral epithelial dysplasia (OED), 13(35%) moderate epithelial dysplasia and 3(8%) severe epithelial dysplasia. Oral squamous cell carcinoma with early invasion and microinvasion is seen in 10 cases (15%) and verrucous carcinoma 4 cases (6%). PVL is only one case (2%) and epithelial hyperplasia and hyperkeratosis is 5 cases (8%). Other cases are oral lichen planus 6 cases (9%), and OSMF one case (2%).

keratotic white lesions is higher than other populations and early histopathological examination to these lesions plays an important one.

Biography

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Conclusions: Prevalence of OED and OSCC in oral

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