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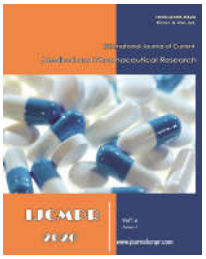

Case Report

INTRAORAL COMPOUND NEVUS: A RARE CASE REPORT

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ABSTRACT

Nevus is a congenital or acquired benign neoplasm of skin or mucosa, characterized by the presence of melanin- producing, neuroectodermally derived nevi cells. Although the acquired melanocytic nevus is probably the most common of all human "tumours" with an average of 10 to 40 cutaneous nevi per person (white adults), but intraoral lesions are uncommon. Intraoral nevus is located usually on the palate and less commonly on the buccal mucosa, gingiva and lips. This article presents a case report of a compound nevus involving the buccal mucosa in a 11 year old male child.

INTRODUCTION

The generic term *nevus* refers to malformations of the skin (and mucosa) that are congenital or developmental in nature.[1] The nevi are benign proliferations of nevus cells in either epithelium or connective tissue. Nevus cells have been postulated to originate from cells that migrate from the neural crest to the epithelium and dermis (submucosa), or to result from altered resident melanocytes.[2] Nevi are commonly seen on the skin, while intraorally they are rare and most commonly observed on the hard palate.[3]

The first documented case of an oral nevus was reported by Ackermann and Field in 1943.[4] Comerford *et al*. were the first to propose the term intralamina propria nevus. King *et al*., in1967, reported pigmented nevi as a rare entity in the oral cavity as they were found only in 0.1% of the patients in a large scale survey; they coined the specific term intramucosal nevus.[5]

The most common intraoral nevi are intramucosal (63% to 80.6%), and blue nevi (8.3% to 32%), while compound (5.9% to 16.5%) and junctional (5%) nevi are relatively rare.[6, 7, 8] In the present paper, we report a case of compound nevus of buccal mucosa(after taking consent from the grand parents of the patient) which is rare with regard to age, gender, site of presentation and appearance.

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Case Report

A 11 year old male patient reported to the Department of Oral Pathology and Microbiology with a chief complaint of a growth present on the left side of buccal mucosa. Probing into the past history his grandparents disclosed the presence of the lesion which they observed 8-9 years back and said that it was static in nature. The patient did not complain of any pain or other symptoms associated with the growth. Medical history, dental history and family history were not contributory.

On extraoral examination no abnormality was detected. On intraoral examination a well-defined, soft, painless, oval-shaped, varying shades of brown coloured, pedunculated growth was observed on the left side of the buccal mucosa opposite to the 26 tooth (Fig 1). The lesion was measuring approximately 1cm in diameter. There was no other pigmented lesion present anywhere on the oral mucosa.

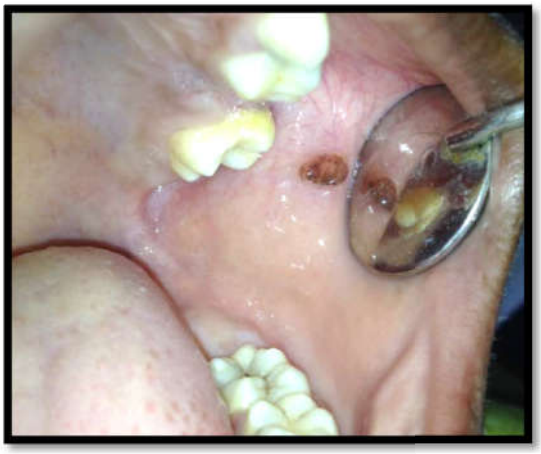
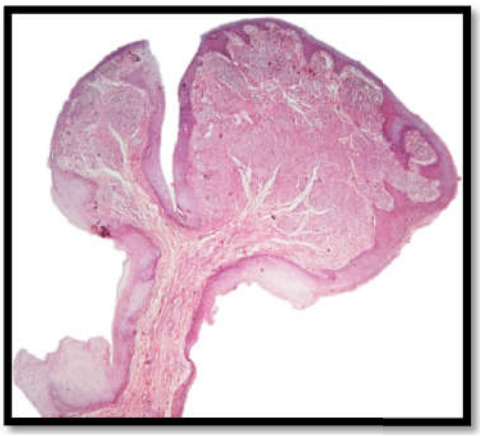
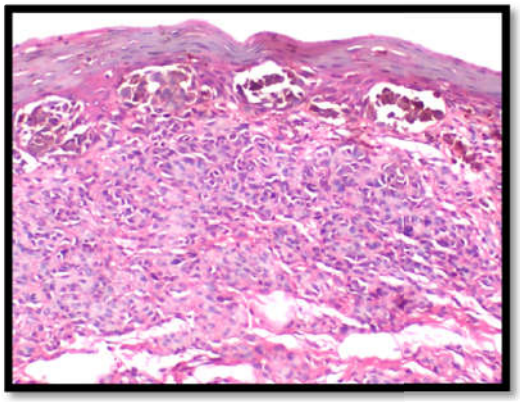


Fig 1 Intraoral Photograph

Routine blood investigations showed Hg- 11gm%, Bleeding time- 1 minute 50 seconds, Clotting time- 5 minutes 10 seconds and were found to be within normal limits. The excisional biopsy of the lesion was performed under local anaesthesia and excised specimen was sent for histopathological examination.

The H & E stained paraffin embedded section on microscopic examination showed parakeratinized stratified squamous epithelium of variable thickness with round to ovoid shaped nevus cells containing melanin granules. The underlying connective tissue core contained numerous round to oval shaped nevus cells arranged in nests, sheets and lobulated pattern. Sub-epithelially scattered areas of melanin containing cells were also seen. In the stalk region, connective tissue was collagenous with few capillaries. The clinical and histopathological features were suggestive of compound nevus. (Fig 2) A 2 year follow-up of the patient showed, proper healing of the lesional area without any significant changes.





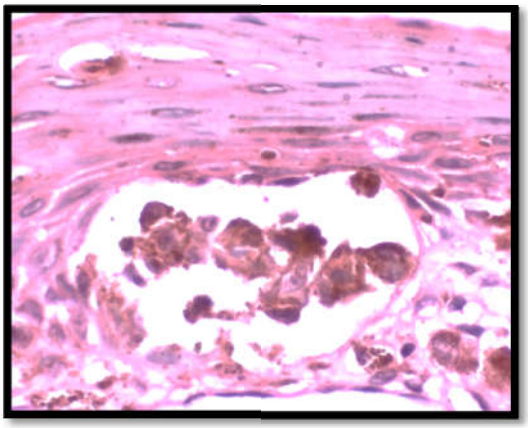


Fig 2 Histopathological examination (40X, 100X and 400X magnification respectively)

DISCUSSION

Oral nevi are benign proliferation of nevus cells. In 1927, Becker first identified melanocytes in the oral epithelium. During early stages of intrauterine life, precursors of melanocytes, i.e. melanoblasts differentiate into the dendritic cells and migrate to the epidermis from the neural crest. Nevus that are thought to originate from melanocytes have hydropic swollen nucleoli that occupy a large portion of nucleus.[5] Normally melanin is retained by the nevus cells and not transferred to the adjacent keratinocytes as they lack the dendritic processes.[9]

Intraoral melanocytic nevi are distinctly uncommon and usually present as asymptomatic, well‑circumscribed, round or oval macules or papules with a smooth surface that range in color from light brown to black depending on the amount of melanin produced and the depth of the pigment relative to the surface. They are typically small in size measuring 0.1- 0.6 cm in diameter.[10] They are slightly predominant in women rather than men and the average age of diagnosis is 35 years.[1] The oral compound nevus, affects mainly the hard palate (33.3% to 57.1%).Less common sites are the buccal mucosa (28.6% to 41.7%), labial mucosa, gingiva, alveolar ridge, and vermilion border of lip.[11, 12]

Ferreira et al. retrospectively studied 100 cases of intraoral nevus and reported intramucosal nevus as the most common type (61%), followed by common blue nevus (23%), compound nevus (7%), and junctional nevus (3%). The hard palate was the most commonly affected site (33%), followed by the buccal mucosa (18%), vermilion border of the lip (18%), and gingiva (15%). The mean age of patients in their study was 36.6 years(range 4-82 years), however, the average age varied according to histologic subtype. In their study, patients with junctional and compound nevi were younger (mean age 6 and 23 years, respectively) than patients with intramucosal and common blue nevi (mean age 35.1 and 48.7 years, respectively).[13]

Macroscopically, nevi may proliferate in two configurations, i.e. Unna's and Meischer's nevi. In Unna's nevi, nevus cells develop in a papillary or round pattern, giving an exophytic outlook, whereas in Meischer's nevi, there is diffuse insinuation of the cells into the subepithelial region giving an endophytic outlook.[5]

Melanocytic nevi frequently harbour oncogenic serine/ threonine-protein kinase B-Raf (BRAF) or, less commonly, neuroblastoma ras viral oncogene homolog (NRAS) mutations. Initially oncogenic mutations might cause hyperproliferation that results in the formation of the nevi and a subsequent oncogenic-induced cellular senescence may account for the cessation of further growth.[11]

Nevi are classified based on their histological features into three phases: proliferation of nevus melanocytes along the submucosal– mucosal junction (junctional nevus); migration of nevus cells to the underlying mesenchymal tissue (compound nevus); and loss of the junctional component of the nevi, so that all remaining nevus cells are located within the subepithelial connective tissue stroma (subepithelial or intramucosal nevus) (Fig 3).[11] Other than the three histologic phases (junctional, compound and intramucosal type), Spitz nevus and blue nevus are also variants of nevi. The differential diagnosis of compound nevus includes melanotic macule, amalgam tattoo and melanoma. In case of more purplish or bluish lesions, haemangioma, hematoma, Varix, Kaposi's sarcoma should also be considered in differential diagnosis.

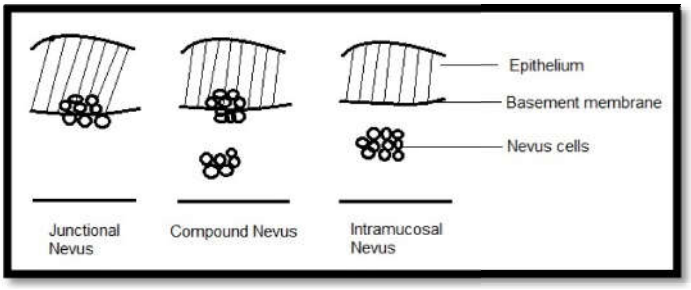


Fig 3 Melanocytic nevus subtypes.

Zones of different types of nevus cells are often appreciated during the development of the lesion.

Type A nevus cells (epithelioid): These are superficial cells which classically appear large epithelioid, with ample cytoplasm;

Type B nevus cells (lymphocyte-like): round-to-polygonal cells, which ensure less cytoplasm and are present in intermediate portion of the lesion;

Type C nevus cells (spindle-shaped): small and round with spindle-shaped nuclei, considerably like Schwann cells or fibroblasts, and are present in deeper portion of lesion.[5]

Histopathologically, nevi is characterized by benign, unencapsulated proliferation of nevus cells. Nevus cells are large ovoid, rounded, or spindle-shaped cells with pale cytoplasm, and may contain granules of melanin pigment in their cytoplasm. The nucleus is vesicular and lacks the dendritic processes typical of melanocytes. They tend to be grouped in sheets or cords which are called nest or thèque. Nevus cells also have the ability to migrate from the basal cell layer into the underlying connective tissue.

The primary concern regarding congenital nevi is malignant transformation and there is general agreement that the risk increases with the size.[14] Although there is lack of evidence about the malignant potential of oral melanocytic nevi but approximately one-third of oral melanomas are preceded by pigmented lesions for months or years.[6, 7] Meleti et al studied 119 patients with oral melanocytic nevus and none developed melanoma during the 8.6 years of follow-up.[6] Surgical excision of all intraoral pigmented nevi is recommended to exclude early stage melanoma and presumably, to prevent the risk of malignant transformation due to constant chronic irritation to the mucosa in nearly all intraoral sites occasioned by eating, tooth brushing etc.[11, 14]

CONCLUSION

Proper clinical evaluation, followed by conservative surgical excision and histopathologic examination should be done for all solitary pigmented oral lesions. Despite the lack of evidence for its real potential for malignancy, histopathological confirmation and follow-up is strongly recommended to exclude malignant melanoma at an early stage and to exclude dysplastic changes. The usually small size of the lesion also justify the recommendation for excisional biopsy.

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