

# Acquired Compound Melanocytic Nevus on the Palate of a Child: Report of a Case

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## ABSTRACT

**Background:** Oral melanocytic nevi are relatively rare in comparison to their cutaneous counterparts. The aim of this manuscript is to present a case of acquired compound oral melanocytic nevi on the hard palatal mucosa of a child.

**Methods:** A 5-year-old female girl was referred for evaluation of a pigmented lesion on the hard palate. The lesion was asymptomatic and present for approximately 2 months. Oral clinical examination revealed a well-circumscribed brownish macule on the hard palatal mucosa, adjacent to the left first primary upper molar. Considering the recent onset of the lesion, biopsy was recommended, but the patient returned 3 years later, when increase in size with slight asymmetry and colour variation were noticed. An excisional biopsy was performed.

**Results:** Microscopic examination revealed nevus cells randomly distributed along the basal cell layer and organized into nests along the junctional area and within the papillary layer of lamina propria, while immunohistochemical evaluation showed positivity of nevus cells for SOX-10 and Melan-A. A final diagnosis of compound melanocytic nevi was rendered, and the patient was advised to attend regular follow-up appointments.

**Conclusions:** Although oral melanocytic nevi are rare in childhood, their potential development should not be overlooked. Acquired oral melanocytic nevi need to be differentiated from several other common (e.g. amalgam tattoo) and uncommon (e.g. melanoma) oral pigmented lesions, as well as from the more rare congenital oral melanocytic nevi. Oral melanocytic nevi with junctional activity (i.e. junctional, compound subtypes) appear to be more common in children, possibly reflecting an earlier developmental stage.

**Keywords:** benign neoplasms; child; hard palate; melanin; melanocytic nevus; oral pathology.

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## INTRODUCTION

Melanocytic nevi (MN) are uncommon benign neoplasms of neural crest origin, characterized by proliferation of nevus cells and typically associated with endogenous melanin pigmentation [1,2]. Although MN are commonly found on the skin, they are rare in the oral cavity with a prevalence of just 0.1% in the general population [1,2].

Clinically, oral melanocytic nevi (OMN) present as macules, slightly elevated papules or nodules, which are well-circumscribed, round or ovoid in shape and of variable size, usually less than 1 cm diameter in maximum diameter. The hard palate is the most common location of OMN (accounting for 32 to 41% of all cases), followed by the buccal mucosa and mucobuccal fold, vermilion border of the lip, and gingiva [1-7].

Microscopically, OMN are further divided in distinct subtypes, mainly intramucosal, compound, junctional, and blue; intramucosal appears to be the most common OMN subtype, followed by common blue nevus [1-7]. Other MN subtypes, e.g. spitz, dysplastic, combined and cellular blue nevus, are rare on the oral mucosa [1,2].

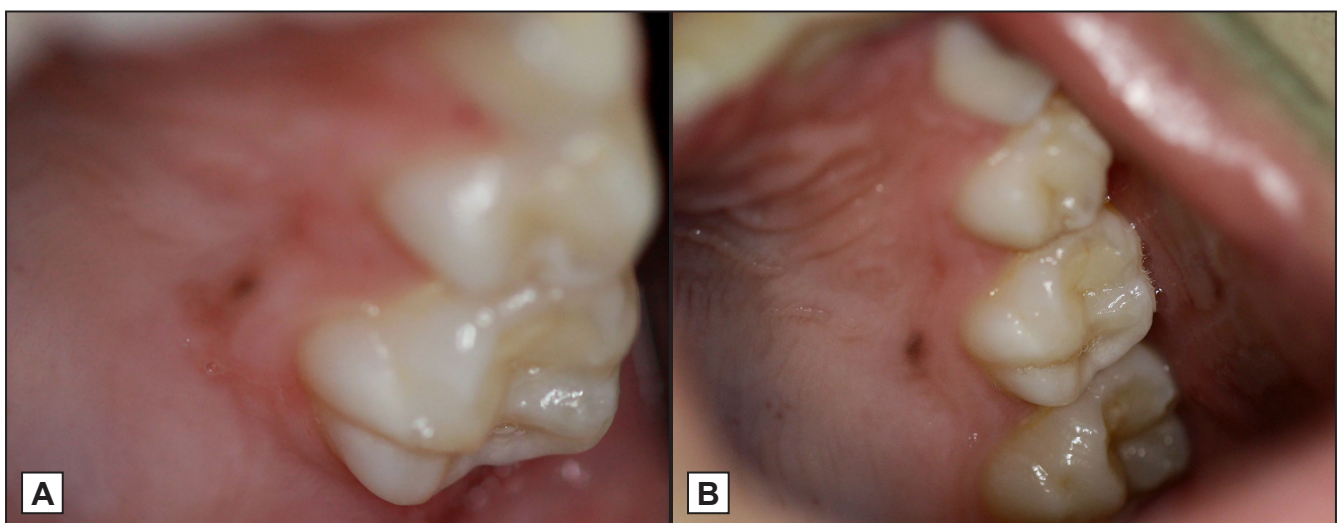
OMN, similar to their cutaneous counterparts, are classified either as congenital or acquired; the former is rare, usually present at birth or during the first year of life and only few cases have been reported in the literature [8-10]. On the other hand, acquired OMN are more often, frequently observed later in life with a mean age at diagnosis ranging from approximately 30 to 45 years in several large series; development in childhood is uncommon [2-7].

The aim of this study is to present a case of an acquired intraoral compound nevus in a 5 years old girl and to highlight the importance of careful clinicopathologic correlation.

## CASE DESCRIPTION AND RESULTS

A 5-year-old girl of Caucasian descent was referred in March 2018 to an Oral Medicine Clinic, in Athens, Greece, for evaluation of a pigmented lesion on the hard palate. The lesion was asymptomatic and, according to her mother, was first noticed 2 months ago. The medical history was unremarkable, and no medications were used. Oral clinical examination revealed a well-circumscribed brownish round to ovoid macule on the hard palatal mucosa, adjacent to the left first primary upper molar, measuring approximately 0.2 cm in maximum diameter (Figure 1A). There were no other similar lesions on the oral mucosa, the skin or anywhere else. The clinical differential diagnosis mainly included OMN, oral melanotic macule, and foreign body pigmentation (such as amalgam or graphite tattoo). However, amalgam tattoo was excluded from our differential, since no fillings had been performed, while the possibility of other source of foreign body pigmentation was low, as no related habits (e.g. pencil placement in the mouth) or trauma in the area were reported. Considering the recent onset and the site of the lesion, an excisional biopsy was discussed with the mother, who opted for observation only.

The patient returned 3 years later (at age 8) and, although the lesion remained asymptomatic, the mother reported an increase in size. Indeed,



**Figure 1.** Oral clinical examination. A = initial visit (at age 5). A well-circumscribed brownish round-to-oval macule on the hard palatal mucosa, adjacent to the left first primary upper molar of approximately 0.2 cm in maximum diameter. B = second visit (at age 8). An elongated ovoid brownish macule with slight asymmetry and colour variation; a small increase in size compared to the first appearance is seen.



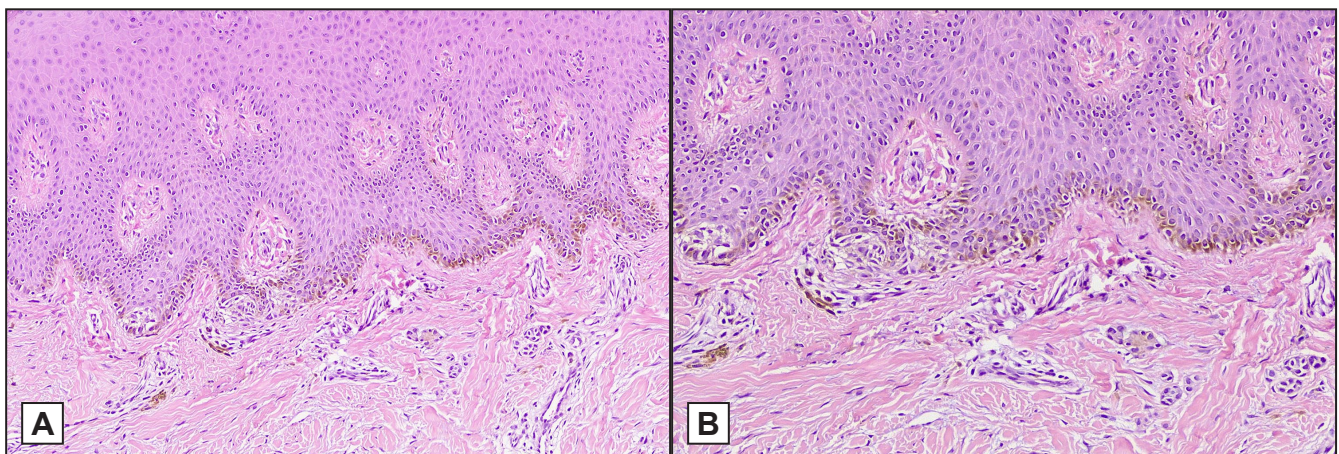
on clinical examination, the lesion appeared somewhat increased with a more ovoid and elongated shape (measuring approximately 0.3 x 0.2 cm); in addition, slight asymmetry and colour variation were noticed (Figure 1B). The lesion was excised, and microscopic examination revealed nevus cells randomly distributed along the basal cell layer, organized into nests along the junctional area and within the papillary layer of lamina propria (Figure 2A and 2B); mitoses were absent. Immunohistochemical evaluation showed positivity of nevus cells for SOX-10 (Figure 3A) and Melan-A (Figure 3B). Thus, a final diagnosis of acquired compound OMN was rendered. No evidence of recurrence was seen at 6 months postoperatively and regular follow-up was recommended.

## DISCUSSION

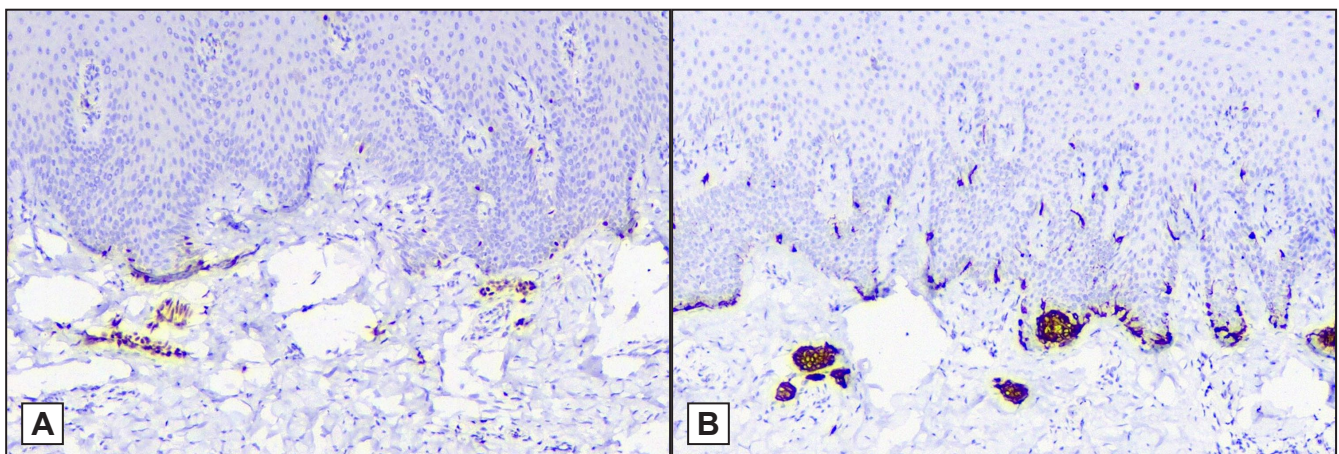
While cutaneous MN is very common, involvement

of the oral mucosa is very unusual affecting just 0.1% of the population [1-5]. In addition, OMN appear to represent a minority of oral pigmented lesions; for example, Buchner et al. [5] reported that, in a total of 89,430 oral biopsies, 773 (0.83%) were melanocytic lesions, among which only 91 were diagnosed as OMN, corresponding to 11.8% of melanocytic lesions and just 0.1% of all biopsies. These results are in accordance with a study by Ferreira et al. [2], who reported that OMN corresponded to 0.067% of the total number of oral biopsies. Similarly, Amérigo-Góngora et al. [3], in a retrospective study, reported that among all oral lesions (total 5,499 biopsies), OMN accounted for 0.18%, while out of 46 pigmented lesions, 10 (21.8%) were classified as OMN.

Especially in children, OMN are quite rare with a limited number of reported cases. Meleti et al. [7] found that, out of 119 OMN cases, the total number of lesions between ages 0 to 9 and 10 to 19 were 5 (4.2%) and 9 (7.5%), respectively.



**Figure 2.** Histopathologic examination of melanocytic nevus (hematoxylin and eosin stain, original magnification x200). A and B = nevus cells randomly distributed along the basal layer of the epithelium, forming small nests in the junctional area and within the papillary layer of the lamina propria.



**Figure 3.** Immunohistochemical examination of melanocytic nevus (original magnification x100). Positivity of nevus cells for: A = SOX-10; B = Melan-A.

In another study by Buchner et al. [5], the corresponding numbers were somewhat higher: out of 88 OMN, 7 (8%) and 17 (19.3%) affected patients in the 0 to 9 and 10 to 19 age groups, respectively. Moreover, Ferreira et al. [2], in a total of 100 OMN cases, found that 5.3% developed between 0 to 9 years old and 15.8% between 10 to 19 years old. Therefore, our patient, aged just 5 years old at the time of initial clinical diagnosis, constitutes an unusual example of OMN development in the first decade of life. However, it should be kept in mind that most relevant information on OMN in childhood is extracted from large studies including all ages, in which the time of excision does not necessarily coincide or even approach the time of onset; therefore, OMN may be present in childhood and persist into adulthood, before detection and final diagnosis.

OMN in children may also show a different predilection for the various microscopic subtypes, compared to adults. In the series by Buchner et al. [5], among 7 OMN developing in the first decade of life, a preference for compound and junctional nevi (4 and 2 cases, respectively) was noticed, while only one intramucosal case was recorded; among 17 cases affecting patients in the 10 to 19 age group, 8 intramucosal, 6 compound, 2 blue and 1 junctional nevus were detected. Interestingly, junctional OMN developed exclusively (3/3) at early ages (between 7 to 14 years), with a mean age of 9.3 years. Moreover, the majority of compound nevi (10 out of 14 cases) affected young patients in the first two decades of life. On the other hand, only 9 out of 56 cases of intramucosal nevi and 2 out of 15 cases of blue nevi appeared in children and adolescents [5]. Similar results are seen in other large series of OMN; for example, Ferreira et al. [2] also found that all 3 cases of junctional nevus (out of a total of 100 OMN) were in the 0 to 9 age group; also, almost half (3 out of 7) of compound nevi were diagnosed in the first two decades of life. In contrast, among 56 intramucosal nevi, only 10 were noticed in patients younger than 20, all of them appearing in the second decade of life. Overall, it appears that junctional and compound OMN, albeit infrequent in adults, are comparatively more common in children, possibly reflecting the stage of evolution and progression of nevus cell proliferation. At early stages, nevus cells forming nests are found along the basal cell layer of the epithelium, especially at the tips of the rete ridges, corresponding to the microscopic appearance of a junctional nevus. At a later stage, as the nevus cells continue to proliferate, some of them migrate to the underlying lamina propria. Concomitant presence of nevus cells, distributed both at the basal cell layer of

the epithelium and the upper parts of the connective tissue, renders a diagnosis of compound nevus. Intramucosal nevus, the most common subtype, represents a later stage of evolution, when nevus cells are limited into the connective tissue, typically in a more diffuse pattern [1,2]. This developmental progression model provides reasonable explanation for the predilection of the junctional and, to a lesser degree, the compound variants at younger ages. Although this hypothesis cannot be directly proved without properly designed longitudinal studies, it seems to support the different percentages of OMN subtypes at different ages. Similarly, our 5-year-old patient presented with an OMN of the palate, which had reportedly developed 2 months earlier, and, on biopsy performed at age 8, showed features of compound nevus; it could be hypothesized that a progressive maturation of the lesion may have occurred from a junctional to a compound stage during the 3 year period from the onset to the removal of the lesion, while a longer period could have theoretically resulted in further transition to an intramucosal stage.

The location of our patient's OMN on the hard palatal mucosa appears to be relatively common. In several sizeable series, the hard palate is reported as the site of predilection for OMN development. In a study of 100 OMN cases, Ferreira et al. [2] identified the palate as the most common location (33%), followed by the buccal mucosa, vermillion of the lip and gingiva (18%, 18% and 16%, respectively). Also, in the study by Meleti et al. [7], hard palate accounted for 34.5% of all lesions, while 26.4% and 22.7% of cases were located in the mucobuccal fold and gingiva, respectively. Quite similar results were obtained from Buchner et al. [5], who reported that the respective percentages of OMN in the palate, buccal mucosa, vermillion border and gingiva were 44%, 22%, 18% and 12%.

Differential diagnosis of OMN may be challenging, encompassing a variety of lesions and conditions presenting as focal, multifocal or diffuse pigmentations. These may be due to either endogenous processes (such as melanotic macule, melanoacanthoma, melanotic neuroectodermal tumour of infancy or malignant melanoma - MM) and exogenous causes (such as foreign body implantation, e.g. amalgam or graphite tattoo) [2-5,8-12]. Biopsy is the gold standard in order to rule out any other pigmented lesions, including those most commonly seen in children, such as graphite tattoo; the most frequent location of the latter entity is the hard palate, as children may habitually place a pencil in their mouth. Also, MM, despite its relative rarity



in the oral cavity and especially in children [13], should be always considered. Selecting which oral pigmented lesions need to be biopsied depend on several parameters, such patient's age, duration, and site and size of the lesion; a general rule is to remove all pigmented lesions of recent or unknown onset, as well as those exhibiting asymmetry, abnormal borders, large size, irregular pigmentation, or any changes (according to ABCDE clinical system applied to cutaneous nevi); further, it has been suggested that lesions located on palate or the maxillary alveolar mucosa and gingiva should be removed due to higher propensity of oral MM (OMM) development in this anatomic location [14,15]. Accordingly, our young patient showing a recent onset of a pigmented lesion on the palate qualified for a biopsy; upon her return, the reported and observed clinical changes further corroborated the decision for biopsy removal.

Congenital OMN appears to be significantly less common than acquired, with only six published cases [8-10]. The distinction between acquired and congenital OMN in children may be established through clinicopathological correlation. Besides age of onset (with congenital nevi being present at birth or shortly thereafter), congenital OMN are usually larger (> 1.5 cm) than acquired OMN (typically less than 0.6 cm) [8]. Further, congenital OMN are expected to grow proportionally with the overall growth of the child [10]. Microscopically, it has been suggested that, in the congenital type, nevus cells extend into deeper layers of the lamina propria in a more diffuse pattern between collagen bundles and around vessels, while, in the acquired type, nevus cells are characteristically organized in nests in the basal cell layer or in the superficial lamina propria [8-10]. In our case, the diagnosis of an acquired OMN was supported by the recent onset of the lesion (based on mother's account), the small size and the microscopic features of nests formation in superficial localizations.

Melanocytic nevi (MN) are benign neoplasms in which mutations in various genes, such as *BRAF*, *NRAS*, *HRAS*, *PTEN*, *CDKN2A*, *GNAQ*, *GNA11*, have been reported [1,16-20]. Some of these mutations are common in MM, and, at least on skin, MN are well-characterized premalignant lesions with a significant proportion of cutaneous MM deriving directly from a pre-existing MN [19,21]. For example, Pampena et al. [21], in a meta-analysis study, highlighted that, although most of the cutaneous MM arise *de novo*, 30% of them derive from a pre-existing nevus, more frequently associated with the acquired type (77.4% vs. 22.6% of congenital) and the intradermal (54%) histopathologic subtype. However, the association of OMN with OMM is less clear, without sufficient

evidence to support the premalignant nature of the former. Meleti et al. [7] evaluated 119 cases of OMN and none of them transformed to melanoma in a mean follow-up period of 8.6 years, suggesting that OMN is not a precursor lesion or a marker for an increased risk of developing melanoma. Nonetheless, considering that oral melanocytic tumours share similar clinical and microscopic features with their cutaneous counterparts, the possibility of OMN malignant transformation cannot be excluded [22]. Indeed, in a recent clinicopathologic study, Liu et al. [23] reported on 7 cases of so-called nevus-associated melanoma (NAM) of the oral mucosa, defined as OMM showing coexistence of malignant melanocytes with nevus cells, and compared them with 74 OMN cases; interestingly, features associated with NAM included older age, male gender, gingival location and junctional subtype; in multivariate analysis, the latter emerged as an independent factor associated with an increased risk for MM development [23]. Although the molecular landscape of oral melanocytic tumours, especially OMN, remains largely unknown, recent evidence suggest that similar to their cutaneous counterparts, OMN harbour the oncogenic BRAFV600E mutation in a significant percentage (42%) of cases, which is actually much higher compared to OMM (6.4%) [16,24]. This is the most common driver mutation in cutaneous acquired nevi and melanomas and shows a much higher prevalence in the former, pointing to its role in early melanocytic tumorigenesis; therefore, the same trend in oral melanocytic tumours possibly indicates similar oncogenic mechanisms for oral and cutaneous melanocytes, lending credence to the hypothesis that OMN may also constitute premalignant lesions, in the sense that additional mutations may lead to their transformation to OMM.

Regarding OMN management, surgical excision is the treatment of choice, since it provides a definitive diagnosis by means of histopathologic examination and eliminates the risk of lesions progression. Following OMN removal, close follow-up is advisable, especially if atypical microscopic features, such as a lentiginous growth pattern, are observed [17].

## CONCLUSIONS

Oral melanocytic nevi are very uncommon compared to cutaneous melanocytic nevi and, especially, their development in childhood is quite rare with a very limited number of reported cases. Nonetheless, they should be always included in the differential diagnosis

of oral mucosal pigmented lesions in children. Careful clinicopathologic correlation is necessary in order to distinguish between acquired and congenital oral melanocytic nevi. Since oral melanocytic nevi may clinically mimic early-stage melanoma, a definitive diagnosis is established only after histopathologic examination. Although the evidence suggesting that oral melanocytic nevi shows a predilection for malignant transformation, similar to their cutaneous counterpart, remains limited, conservative surgical removal and subsequent follow-up is recommended.

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