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Intramucosal nevus on the posterior hard palate

KEYWORDS
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Histopathological features

Acquired melanocytic nevus is a very common tumor on the skin, but it is rarely found on the oral mucosa.[1–3] This article presented a case of oral intramucosal nevus on the left posterior hard palate of a 32-year-old female patient.

This 32-year-old female patient was referred to our oral mucosal disease clinic by a local dentist for treatment of a black lesion on the left posterior hard palate for an unknown duration. The black lesion measuring 0.6 × 0.3 cm in area was soft, slightly elevated, and relatively well-demarcated (Fig. 1A). The clinical differential diagnoses included an oral mucosal nevus, melanotic macule, and common blue nevus. Because biopsy of all unexplained pigmented oral lesions was generally advised, after discussing with the patient and obtaining the signed informed consent, the black lesion was totally excised under local anesthesia. The removed soft tissue specimen was sent for histopathological examination. Microscopically, the surface stratified squamous epithelium was slightly hyperplastic and parakeratotic. In the lamina propria and submucosa, there was an accumulation of three types of nevus cells (Fig. 1B–F). The superficial several layers of nevus cells were epithelioid in shape (so-called type A nevus cells) and were full of melanin pigments in the cytoplasm. The nevus cells in the middle portion were lymphocyte-like (so-called type B nevus cells). Only a very small portion of these lymphocyte-like nevus cells possessed melanin pigments in the cytoplasm, and the majority of them had none of the melanin pigments in the cytoplasm (Fig. 1B–F). Moreover, the nevus cells in the deep portion of the lesion were composed of few number of the spindle-shaped fibroblast-like cells (so-called type C nevus cells) that had none of the melanin pigments in the cytoplasm (Fig. 1B–F). The aforementioned characteristic microscopic findings of the black lesion finally confirmed the histopathological diagnosis of an oral intramucosal nevus. The oral intramucosal nevus did not recur one year after the total surgical excision.

Oral nevi include intramucosal, junctional, compound, common blue, cellular blue, combined, and dysplastic nevi histologically.[1–3] Ferreira et al.[2] studied the clinicopathologic features of 100 oral melanotic nevi. Of these 100 nevi, 61 are intramucosal, 23 are common blue, 7 are compound, 3 are junctional, and other three types (cellular, combined, and dysplastic) each has 2 nevi.[2] Our previous study analyzed 29 oral nevi; of them, 21 are intramucosal, 6 are common blue, and 2 are compound nevi.[3] The findings of the above-mentioned two studies indicate that the most common histologic type of oral nevi is the intramucosal nevus, followed by the common blue nevus and compound nevus.

Oral nevi are discovered more commonly in female than in male patients with a female-to-male ratio ranging from 1.2: 1 to 3.6: 1 in the previously reported cases.[1–3] Our previous study of 29 oral nevi excised from 27 patients (19 female and 8 male patients) showed a female-to-male ratio of 2.4: 1.[3] Moreover, Ferreira et al.[2] reported 100 oral nevi

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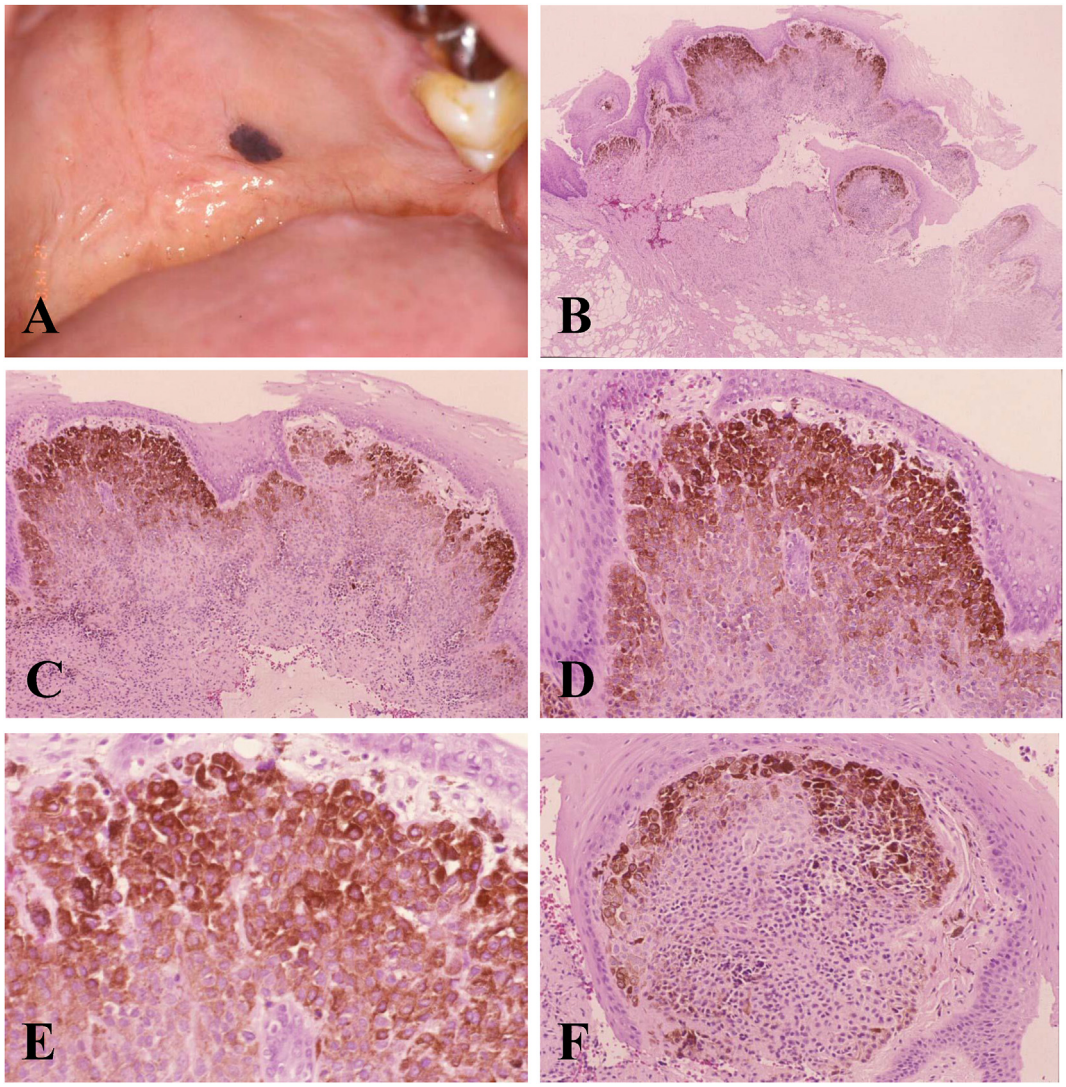

Figure 1 Clinical photograph and histopathological photomicrographs of our case of oral intramucosal nevus in a 32-year-old female patient. (A) A slightly elevated and relatively well-demarcated black lesion measuring 0.6 × 0.3 cm in area was found on the left posterior hard palate. (B, C, D, E, and F) Low-, medium-, and high-power photomicrographs showing an accumulation of three types of nevus cells in the lamina propria and submucosa of the hard palate. The superficial several layers of nevus cells were epithelioid in shape and were full of melanin pigments in the cytoplasm. The nevus cells in the middle portion were lymphocyte-like. Only a very small portion of these lymphocyte-like nevus cells possessed melanin pigments in the cytoplasm, and the majority of them had none of the melanin pigments in the cytoplasm. Moreover, the nevus cells in the deep portion of the lesion were composed of few number of the spindle-shaped fibroblast-like cells that had none of the melanin pigments in the cytoplasm. (Hematoxylin and eosin stain; original magnification; B, 4×; C, 10×; D and F, 20×; E, 40×).

taken from 100 patients (59 females, 38 males, and the gender was unavailable in three patients) with a female-to-male ratio of 1.6: 1. These results confirmed the female predilection for the occurrence of oral nevi.[1–3]

The mean age of the patients with the oral nevi are between 30 and 38 years. For our 29 oral nevi excised from 27 patients in our previous study, the mean age of the 27 patients is 33 years.[3] In addition, the mean age of 95 patients with oral nevi (the age is unknown in 5 patients) in the study of Ferreira et al.[2] is 36.6 years.

The most-frequent site for occurrence of oral nevi is the hard palate, followed by the buccal mucosa or vermilion border.[1–3] For our 29 oral nevi, the most-common site is the hard palate (12 cases), followed by the vermilion border (9 cases), buccal mucosa (4 cases), soft palate (2 cases), and retromolar pad (2 cases).[3] Regarding the location of 100 oral nevi reported by Ferreira et al.,[2] the most common location is the hard palate (33 cases), followed by the lip vermilion (18 cases), buccal mucosa (18 cases), gingiva (16 cases), soft palate (5 cases), retromolar pad (5 cases), and labial mucosa (5 cases). In addition, more than 80% of the oral nevi are smaller than 1 cm in the greatest diameter.[2,3]

Resende et al.[4] assessed the BRAFV600E mutation in 14 oral melanocytic nevi and 7 oral mucosal melanomas. They found that 5 (35.71%) of 14 oral melanocytic nevi and 3 (42.86%) of the 7 oral mucosal melanomas showed the

BRAFV600E mutation. This finding indicates that although the oral melanocytic nevi are benign lesions, the BRAFV600E mutation can be detected in approximately one-third of the oral melanocytic nevi.[4]

Declaration of competing interest

The authors have no conflicts of interest relevant to this article.

Acknowledgments

None.

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