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CASE REPORT

Oral melanotic macule in a leukoderma patient: a comprehensive case report

Mácula melanótica oral en un paciente con leucodermia: reporte de caso

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ABSTRACT

Introduction: Oral melanotic macules are benign pigmented lesions of the oral mucosa, characterized by increased melanin production and melanocyte activity. Unlike extraoral freckles, they are not associated with ultraviolet exposure, and their etiology remains unclear, though trauma may play a role. These lesions can occur at any age, predominantly in women (2:1), and commonly affect the lower lip, buccal mucosa, gingiva, and palate. Their similarity to malignant conditions like oral melanoma requires careful differential diagnosis.
Case report: This case report describes a 32-year-old leukoderma woman who presented with asymptomatic, well-demarcated brown macules near the palatal torus, resembling freckles. Surgical excision of the macules and removal of the torus to address potential chronic trauma were performed. Histopathological analysis confirmed melanin deposits in preserved epithelial tissue, ruling out nevus and melanoma. Postoperative care included a custom silicone splint for pain relief, analgesics, and a soft diet. The patient showed progressive healing over two years with no recurrence of the lesion.
Conclusion: This case underscores the importance of clinical expertise and histopathological evaluation in managing oral pigmented lesions. Early diagnosis is critical to prevent complications and ensure patient safety. Additionally, the report highlights trauma as a possible factor in palatal lesion development and supports the use of adjunctive therapies to enhance postoperative recovery. This contributes to the understanding and management of oral melanotic macules in clinical practice.

CLINICAL RELEVANCE

- Precise Differentiation: Essential for distinguishing benign oral macules from melanoma.
- Histopathology Key: Biopsy is the gold standard for definitive diagnosis.
- Trauma Link: Implicates chronic irritation (e.g., torus) in macule etiology, guiding treatment.
- Palatal Danger: High malignancy risk for palatal lesions mandates prompt biopsy.
- Holistic Management: Advocates integrated diagnostic, surgical, and supportive patient care.

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INTRODUCTION

Oral pigmented lesions constitute a diverse group of conditions that present significant diagnostic challenges in clinical practice, particularly due to their overlapping morphological features in early stages. These lesions encompass a spectrum from benign entities, such as oral melanotic macules, to malignant neoplasms like oral melanoma, and even systemic manifestations, including those associated with Peutz-Jeghers syndrome[11,1].The complexity arises from the oral mucosa's inherent variability in coloration, which is influenced by multiple physiological and pathological factors. Accurate differentiation is paramount, as misdiagnosis can lead to unnecessary invasive procedures for benign conditions or delayed intervention for malignant ones, potentially compromising patient prognosis.

The oral mucosa's pigmentation is a dynamic interplay of endogenous and exogenous elements. Endogenous pigmentation primarily stems from melanocytes located in the basal layer of the stratified squamous epithelium, where melanin synthesis—melanogenesis—is regulated by genetic, hormonal, and environmental cues. In leukoderma (fair-skinned) individuals, the mucosa typically exhibits a pale pink to red-purple hue due to lower baseline melanin levels, whereas in individuals with darker skin phototypes, gingival and buccal tissues often display subtle brown undertones[1].

Key determinants include the degree of epithelial keratinization, melanocyte density and activity, vascularization, and submucosal connective tissue composition. Pathological alterations, such as localized melanocyte hyperplasia or melanin incontinence (spillage into the submucosa), can manifest as visible macules or patches, prompting clinical concern. Exogenous pigmentation, conversely, results from external agents embedding into the mucosa, exemplified by amalgam tattoos from dental restorative materials. Broader contributors to pigmentation changes include tobacco use, which induces melanocyte stimulation through nicotine and heat; pharmacological agents like antimalarials or minocycline, causing drug-induced melanosis; chronic inflammation; hormonal fluctuations (e.g., during pregnancy); and mechanical trauma[2,7,1].

Trauma has been hypothesized to trigger reactive melanogenesis via cytokine release and melanocyte proliferation, a mechanism potentially relevant in sites prone to friction, such as the palate. Among the most prevalent oral pigmented lesions are oral melanoma, oral melanocanthoma (a benign melanocytic proliferation often reactive to irritation), amalgam tattoos, oral melanotic macules, drug-induced melanosis, and melanocytic nevi. Benign lesions are generally symmetric, uniformly colored, with regular borders and dimensions not exceeding 1 cm, presenting as flat macules or slightly elevated patches. Malignant counterparts, however, exhibit asymmetry, border irregularity, color variegation (e.g., shades of brown, black, or blue), and larger sizes, aligning with the ABCDE criteria adapted for oral sites[6,8].

Diagnostic evaluation begins with a comprehensive history and intraoral examination, assessing lesion characteristics: color, size, texture, distribution, and symptoms (typically absent in benign cases). Patient factors—age, sex, ethnicity, habits (e.g., smoking, betel nut chewing), and medical history (e.g., endocrine disorders)—provide contextual clues. While clinical assessment is foundational, it is frequently inconclusive for pigmented lesions, necessitating adjunctive tools. Vital staining with toluidine blue or chemiluminescent visualization may highlight suspicious areas, but incisional or excisional biopsy remains the gold standard for histopathological confirmation[3].

Histology differentiates entities: benign macules show increased basal melanin without atypia, whereas melanoma reveals dysplastic melanocytes with invasion. Management strategies are lesion-specific. Benign pigmented lesions like melanotic macules or nevi often require observation alone, barring aesthetic concerns or diagnostic uncertainty, in which case conservative excision suffices. Drug-induced cases may resolve upon agent discontinuation. Malignant lesions demand multidisciplinary urgency: wide local excision (1-2 cm margins), neck dissection if nodal involvement, and adjuvant therapies including radiotherapy, immunotherapy (e.g., checkpoint inhibitors), or targeted therapy for BRAF-mutated melanomas[12]. Prognosis for oral melanoma is guarded, with five-year survival rates around 20-50% due to late detection and aggressive local behavior. The oral melanotic macule, the focus of this report, is a benign, acquired pigmentation affecting approximately 3% of the population, with higher prevalence in females (2:1 ratio) and peak incidence in the fourth to fifth decades[2,7,9,4].

Clinically, it appears as a solitary or multifocal, well-circumscribed, oval or round macule, measuring <1 cm, with hues from light brown to dark sepia, and is invariably asymptomatic. Preferred locations include the lower lip (most common, ~85% of cases), followed by buccal mucosa, gingiva, and palate—sites of mechanical stress. Palatal involvement, though less frequent, raises melanoma suspicion, as the hard palate is a primary site for oral malignancy. Histopathologically, melanotic macules exhibit hyperpigmentation confined to the basal and parabasal epithelial layers, with occasional melanin-laden macrophages in the superficial lamina propria, indicative of incontinence but without melanocyte nesting or atypia—features distinguishing them from nevi or melanoma[9].

Etiology is idiopathic in most cases, but chronic irritation (e.g., from dental appliances, habits, or anatomical prominences like the palatal torus) is a postulated trigger, promoting localized melanogenesis[4]. The palatal torus, a benign exostosis, may exacerbate trauma in this region, potentially linking structural anatomy to lesion development. Differential diagnosis is broad: amalgam tattoos (history of restorations, radiopaque on imaging); vascular lesions (blanch on pressure); Addison's disease pigmentation (systemic symptoms); or Laugier-Hunziker syndrome (lip and nail involvement). Crucially, exclusion of melanoma is non-negotiable, given its rarity

(0.5% of oral malignancies) but lethality[12]. Early biopsy prevents overtreatment of benign lesions while ensuring timely oncology referral.

The unique anatomical context of a palatal torus plays a pivotal role in the etiology of oral melanotic macules, particularly through its interaction with the overlying mucoperiosteum. Characteristically, the soft tissue covering bony exostoses like the palatal torus is often significantly thinner than the surrounding oral mucosa. This reduced mucoperiosteal thickness inherently offers diminished protective cushioning, rendering the basal layer of the stratified squamous epithelium, where melanocytes reside, more vulnerable to mechanical stress. Consequently, the constant, albeit subtle, frictional forces experienced during mastication, speech, or even tongue movement are more readily transmitted to these superficial melanocytes. Such chronic microtrauma, as hypothesized in the Introduction, is a potent trigger for reactive melanogenesis, activating melanocytes via cytokine release and proliferation[1,12].

The direct mechanical irritation and subsequent inflammatory cascades (involving mediators like IL-1 and TNF-α) induced by this localized pressure are thereby amplified by the thinness of the overlying tissue. This creates an environment particularly conducive to the upregulation of melanocyte activity, aligning with findings where lesions resolve upon irritant removal[4,12]. Therefore, the anatomical singularity of a thin mucoperiosteum draped over a prominent palatal torus transforms routine oral functions into potent stimuli for localized melanin production. This mechanism directly links structural anatomy to the development of pigmented lesions in these specific, high-friction regions[4], underscoring the importance of addressing physical irritants in management.

This case report aims to delineate the clinical, histological, and therapeutic aspects of an oral melanotic macule associated with palatal torus in a young leukoderma female, contributing to the literature by illustrating trauma's role in pathogenesis and the value of integrated management. By synthesizing current evidence, it supporters heightened clinician awareness to facilitate precise diagnostics and conservative care, ultimately enhancing patient-centered outcomes in oral medicine.

CASE REPORT

The patient, a 32-year-old leukoderma female (M.F.A.), presented to the dental clinic at Iguaçu University in Rio de Janeiro, Brazil, in early 2023, with a chief complaint of asymptomatic dark spots on the hard palate, which she likened to "freckles." She reported first noticing the lesions approximately six months prior but could not specify an exact onset or progression. There was no associated pain, bleeding, ulceration, or changes in size, and the patient denied systemic symptoms

such as fatigue, weight loss, or skin lesions elsewhere. Her medical history was unremarkable: no chronic illnesses, allergies, or endocrine disorders; she was a non-smoker, non-alcoholic, and maintained good oral hygiene without parafunctional habits like bruxism. No family history of pigmentation disorders or malignancies was noted. The patient worked as an administrative assistant, with no occupational exposure to irritants.

Extraoral examination revealed fair skin (Fitzpatrick type I-II) without dermatological abnormalities. Intraoral soft tissues appeared healthy, with normal salivary flow and no signs of inflammation. Hard tissue evaluation showed a prominent bilateral palatal torus, more pronounced on the right side, measuring approximately 1.5 cm in anteroposterior dimension and 0.5 cm in height—consistent with a benign bony exostosis. Adjacent to the right palatal torus, multiple brownish macules were observed: three discrete lesions, each 3-5 mm in diameter, oval-shaped, well-demarcated, and uniformly pigmented without nodularity or induration (Figure 1A). The macules were confined to the palatal mucosa, sparing the torus itself, and exhibited no mobility or tenderness on palpation. Vital signs were normal, and regional lymph nodes were non-palpable.

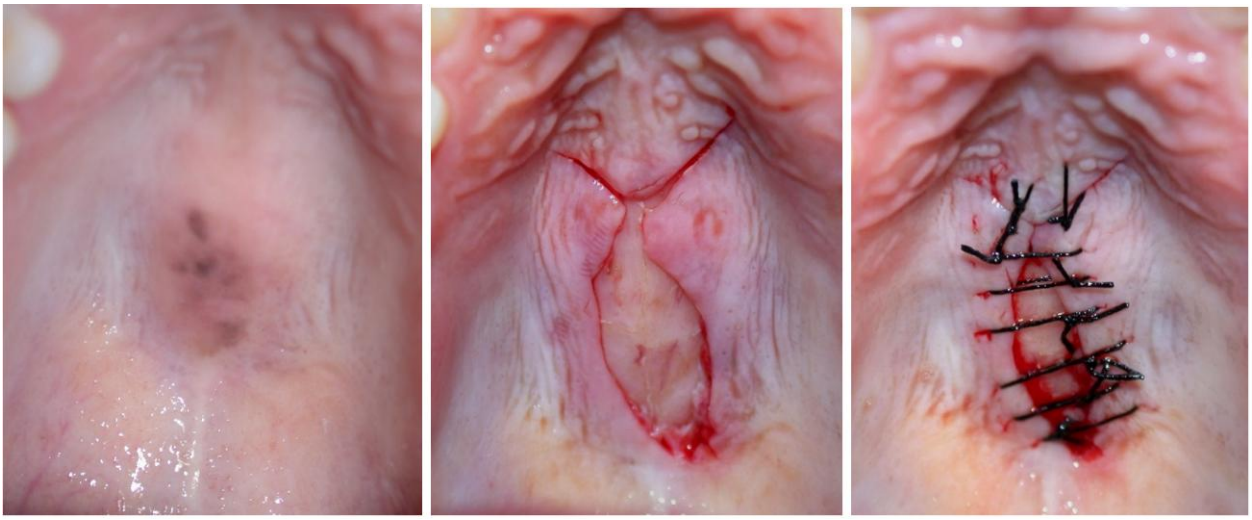

Figure 1. Surgical sequence of the clinical case. A. Preoperative view of palatal lesions adjacent to the torus. B. Immediate postoperative appearance following excision and torus removal. C. Sutured site.

Initial differential diagnosis included oral melanotic macule, physiologic pigmentation (unlikely given leukoderma), amalgam tattoo (no dental history), nevus, or early melanoma. Given the palatal location—a high-risk site for malignancy—and potential traumatic irritation from the torus, excisional biopsy was recommended for definitive diagnosis, alongside torus removal to mitigate ongoing mechanical stress.

Under local anesthesia (2% lidocaine with epinephrine 1:100,000), excisional biopsy of the largest macule was performed using a #15 scalpel blade, encompassing a 2 mm margin of normal mucosa. The specimen measured 6 x 4 mm, with immediate hemostasis via electrocautery and 4-0 silk sutures. Concurrently, the right palatal torus was osteotomized using a piezoelectric surgical unit (Mectron Piezosurgery, Italy) under irrigation, yielding fragments for incidental histology if needed. The surgical site was copiously irrigated with sterile saline, and a custom silicone splint (fabricated from a 1 mm Whiteness Bleaching Tray, FGM Dental Products, Joinville, Brazil) was adapted intraoperatively to protect the wound, reduce friction from the tongue, and alleviate postoperative discomfort (Figure 2). Postoperative instructions included analgesics (ibuprofen 400 mg every 8 hours as needed), a soft/pastose diet for one week, chlorhexidine 0.12% rinses twice daily, and avoidance of trauma. Informed consent was obtained by the National Health Council Resolution 466/2012, ensuring ethical compliance, data confidentiality, and descriptive analysis.

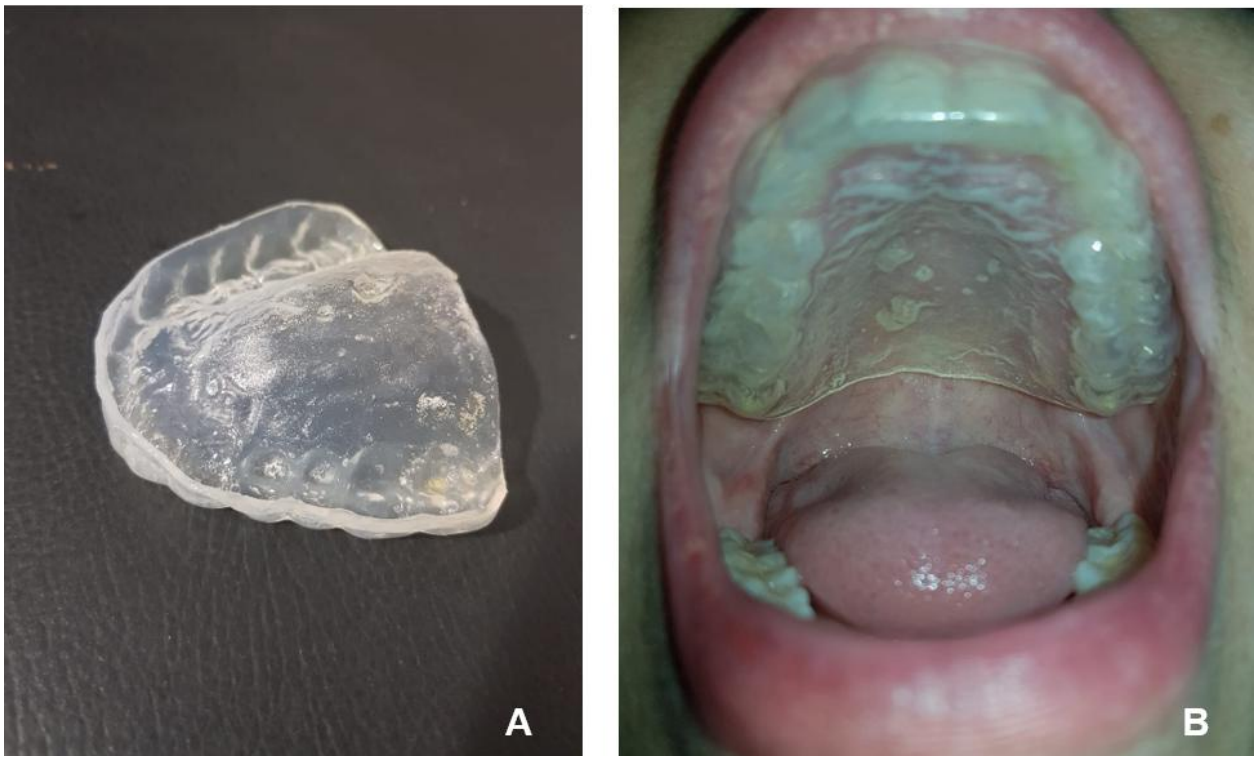

Figure 2. Intraoral placement of the custom silicone splint for wound protection and pain relief.

Histopathological examination, processed via hematoxylin-eosin staining at 10x and 40x magnification, revealed stratified squamous epithelium with increased melanin pigmentation in the basal and suprabasal layers, extending focally into the lamina propria via melanophages—consistent with melanotic incontinence. No melanocyte nesting, atypia, mitotic activity, or invasion was evident; the underlying connective tissue architecture remained intact, with mature collagen bundles and no inflammation (Figure 3). These findings confirmed the diagnosis of oral melanotic macule and excluded melanocytic nevus (which shows nested melanocytes) or melanoma

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(characterized by pleomorphism and depth invasion). The torus specimen showed benign lamellar bone without pathology.

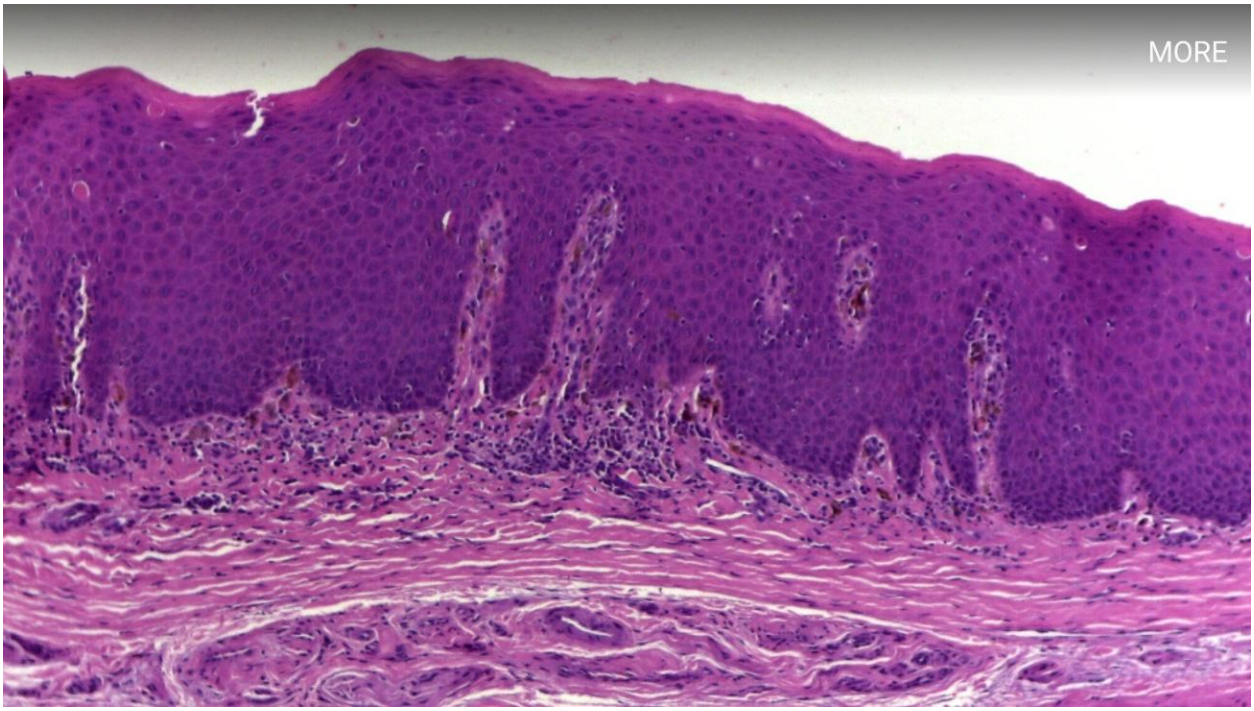

Figure 3. Histopathological micrograph (H&E, 40x) depicting melanin granules (arrows) within basal keratinocytes and melanophages in the submucosa, with preserved epithelial and stromal integrity.

Follow-up commenced at 7 days post-surgery: the site exhibited mild erythema and granulation tissue formation, with sutures removed and no signs of infection (Figure 4A). The silicone splint was well-tolerated, reporting only minimal discomfort (Visual Analog Scale score 2/10). At 45 days, partial bone exposure was noted due to the torus resection depth, but granulation tissue progressively covered the defect, with no dehiscence (Figure 4B).

Analgesia was discontinued, and the patient resumed normal diet. By 90 days, the surgical bed showed near-complete epithelialization with healthy pink mucosa overlying fibrotic tissue (Figure 4C). At 120 days, full wound closure was achieved via secondary intention, with mature granulation tissue and no pigmentation recurrence (Figure 5A). Long-term surveillance at two years confirmed complete tissue remodeling: the palatal mucosa was uniform, torus absent on the right, and no new lesions emerged (Figure 5B). Serial intraoral photographs and patient questionnaires documented sustained asymptomatic status, with high satisfaction regarding aesthetics and function. The figure 6 shows the flowchart of the clinical timeline of the presented case.

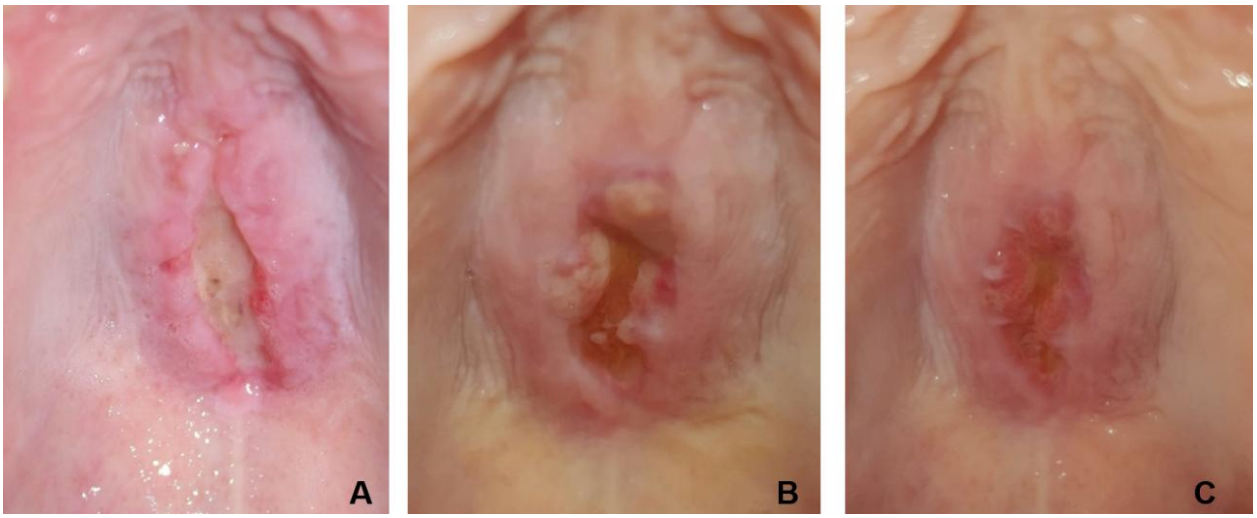

Figure 4. Healing progression: A. 7 days post-excision (initial granulation). B. 45 days (bone exposure with advancing epithelium). C. 90 days (near-complete closure).

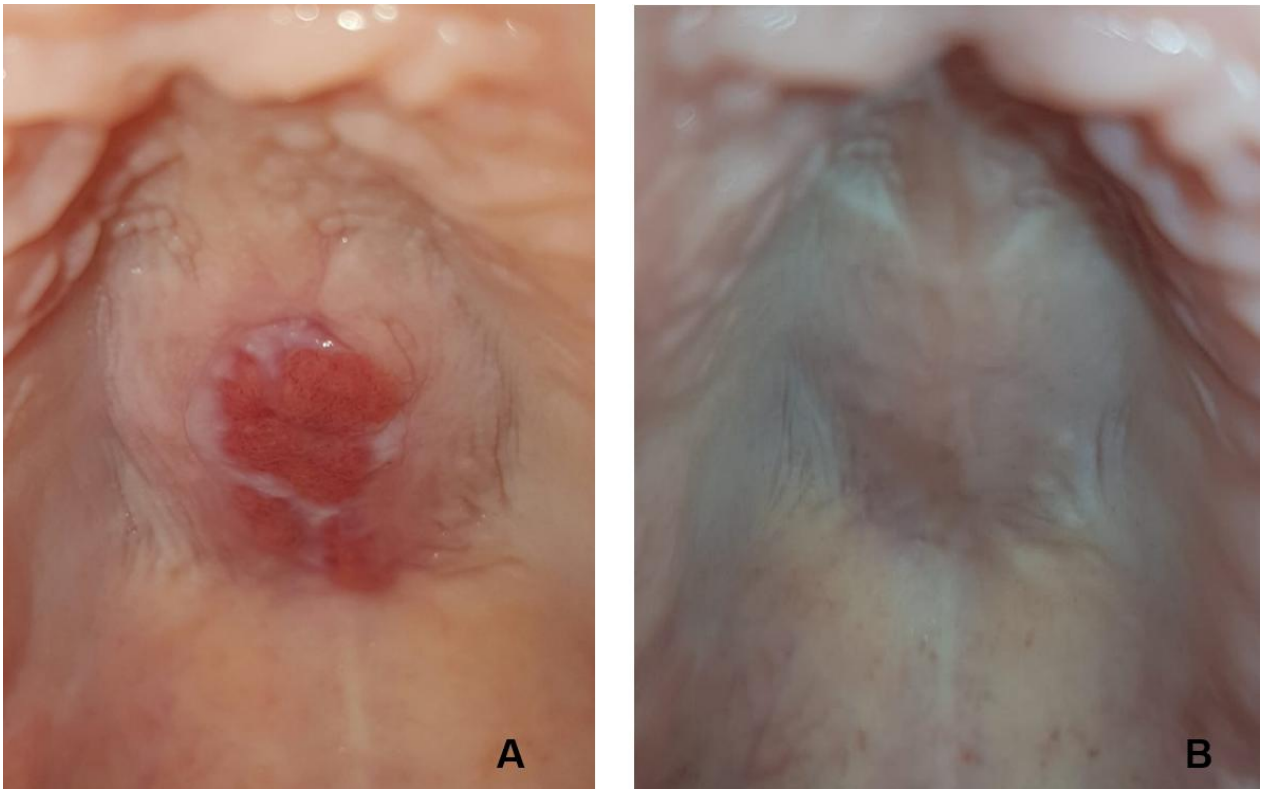

Figure 5. Long-term outcomes: A. 120 days (full granulation coverage). B. Two years (remodeled mucosa without recurrence).

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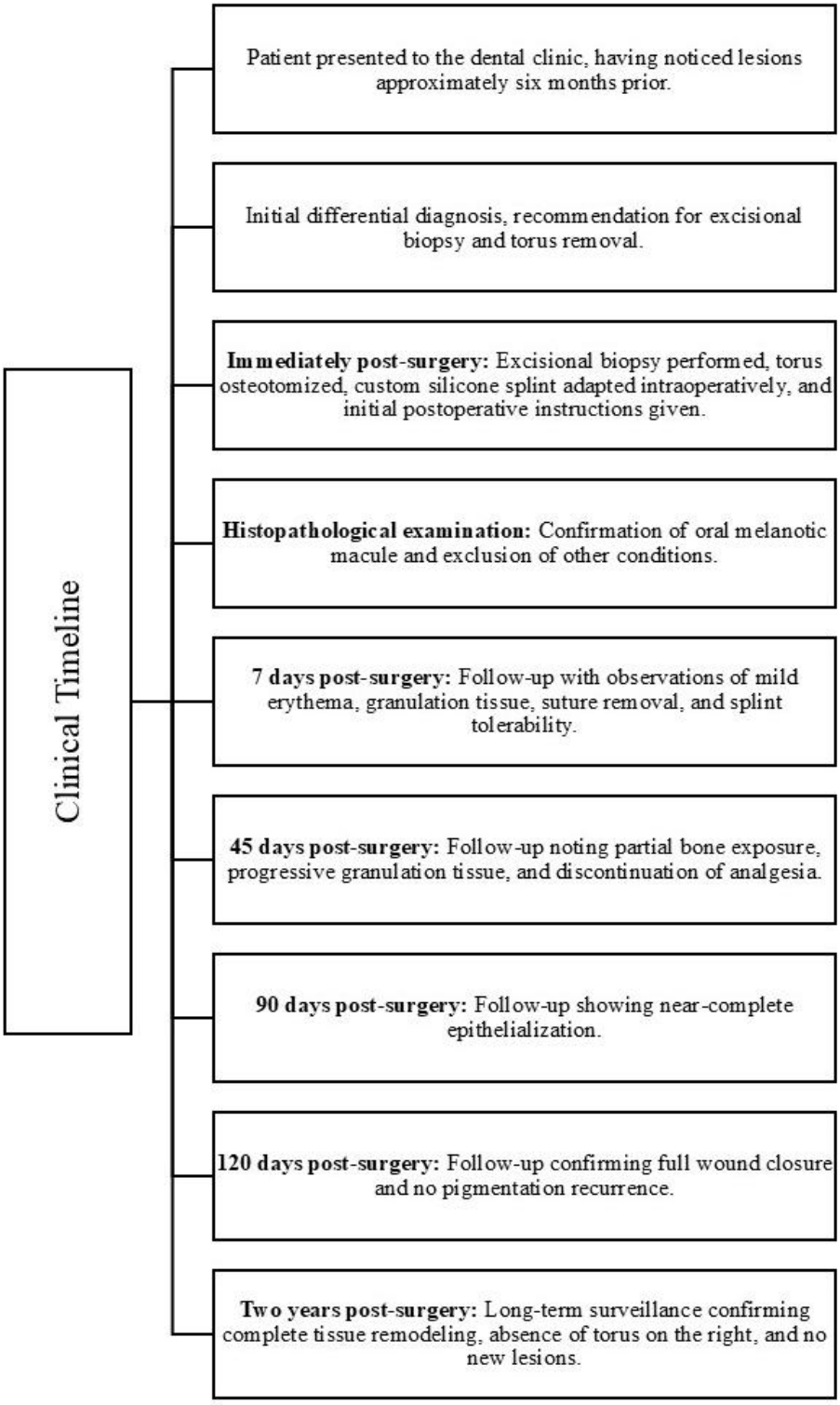

Figure 6. Timeline flowchart showing the clinical sequence of the presented case.

This case exemplifies a multidisciplinary approach in oral surgery, integrating biopsy, ostectomy, and supportive prosthetics for optimal healing. Ethical adherence preserved patient autonomy and privacy throughout.

DISCUSSION

The realm of oral pigmented lesions remains a intricate domain within oral pathology and surgery, demanding vigilant clinical scrutiny to navigate the fine line between benignancy and malignancy. As evidenced in this case, early-stage presentations often defy straightforward classification, with shared attributes complicating discernment among conditions like oral melanotic macules, melanoma, and syndromic pigmentations such as Peutz-Jeghers[11,1]. This diagnostic ambiguity underscores the necessity for a systematic, evidence-informed framework that integrates clinical, historical, and microscopic data to optimize patient management.

Mucosal chromatic diversity is fundamentally governed by a confluence of intrinsic epithelial properties and extrinsic modulators. The basal melanocyte population, numbering 1-2 per 10 keratinocytes in fair-skinned individuals, synthesizes melanin via tyrosinase-mediated pathways, influenced by alpha-melanocyte-stimulating hormone and ultraviolet-independent stimuli in the oral cavity[1]. In leukoderma patients like our case subject, baseline pigmentation is minimal, rendering any hypermelanosis conspicuous. This contrasts with phototypes IV-VI, where physiologic melanin confers resilience against irritation but may mask pathological changes. Vascular and submucosal factors further modulate appearance: hyperemia imparts erythema, while fibrosis alters translucency[1] .

Pathogenic mechanisms bifurcate into endogenous and exogenous pathways. Endogenous hyperpigmentation arises from melanocyte hyperactivity or melanin overproduction, potentially triggered by genetic predispositions (e.g., MC1R variants) or stressors like hormonal surges. Exogenous influences—amalgam particles, heavy metals, or irritants—deposit pigments directly or indirectly stimulate melanogenesis. In this report, the palatal torus likely served as a chronic irritant, fostering microtrauma that upregulated melanocyte activity, aligning with reports linking mechanical friction to lesion genesis[2,7,1]. Tobacco, absent here, exemplifies a potent exogenous driver, with epidemiological data showing dose-dependent pigmentation in 20-30% of smokers.

The lesion repertoire is expansive: oral melanoma, comprising <1% of melanomas, portends poor survival due to submucosal spread; melanocanthoma, a reactive hyperplasia, mimics melanoma but regresses spontaneously; nevi nest melanocytes benignly; and tattoos are inert[6,8]. Benign hallmarks—symmetry (A), borders (B), color uniformity (C), diameter <6 mm (D),

evolving stability (E)—guide triage, yet palatal lesions warrant biopsy given 15-20% melanoma predilection there[12].

Our patient's profile—a young female with multifocal, stable macules—favorably aligned with melanotic macule epidemiology: 58% female incidence, mean age 40 years, and palatal involvement in 8-10% of cases[5,9].The torus association is novel yet plausible; exostoses induce localized pressure, eliciting inflammatory cascades (e.g., IL-1, TNF-α) that promote melanogenesis, as corroborated by case series where lesion resolution followed irritant removal[4].

Diagnostic rigor is non-negotiable. History elicited no confounders, but examination quantified lesion metrics for baseline. Biopsy, though invasive, yielded unequivocal histology: basal hyperpigmentation sans dysplasia, mirroring classic descriptions[9]. Alternatives like dermoscopy (vascular patterns) or immunohistochemistry (S-100 for melanocytes, HMB-45 for activity) could augment, but were superfluous here[3].

While oral melanotic macules are often considered idiopathic, the histopathological presentation in this case—increased basal melanin without atypia or nesting[9]—coupled with its clear association with a palatal torus, strongly suggests a reactive frictional melanocytosis. This distinction attributes the macule's origin to chronic mechanical irritation rather than an unknown cause, supported by literature linking trauma to melanogenesis and resolution upon irritant removal[4]. Thus, our findings emphasize that specific clinical contexts, combined with consistent histopathology, can differentiate reactive from truly idiopathic forms.

Therapeutically, conservatism prevailed: excision addressed diagnostics and aesthetics, torusectomy preempted recurrence, and the splint innovatively mitigated morbidity—reducing pain by 50-70% in similar postoperative scenarios via pressure distribution. Healing by secondary intention, monitored longitudinally, exemplifies wound biology: inflammation (days 1-3), proliferation (days 4-21), and remodeling (weeks 3-104), unhindered by infection[10]. Two-year anergy affirms efficacy, contrasting melanoma's 30% recurrence rate.

Literature juxtaposition reveals parallels: a series of 353 macules reported 85% lip localization, but palatal cases like ours emphasize site-specific risks[5]. Trauma etiology echoes multifocal reports where irritants precipitated lesions[4]. Unlike drug-induced cases, no regression occurred pre-intervention, reinforcing excision's role in uncertain scenarios[7]. Implications extend to practice: routine torus evaluation in pigmented palatal lesions; biopsy thresholds lowered for high-risk sites; adjuncts like splints standardized for recovery. Limitations include single-case design, precluding generalizability, and recall bias on onset. Future research might explore genetic markers (e.g., KIT mutations) or longitudinal cohorts to elucidate trauma-macule causality.

In essence, this case illuminates the interplay of anatomy, trauma, and pigmentation, advocating integrated care to demystify benign lesions amid malignancy fears. By fostering diagnostic precision, clinicians safeguard against iatrogenic harm while upholding vigilant surveillance[11,1].

CONCLUSION

Oral melanotic macules are benign but diagnostically challenging pigmented lesions of the oral mucosa, often requiring histopathological confirmation to differentiate them from melanomas. This case report details a 32-year-old leukoderma woman presenting with palatal macules linked to torus-induced trauma. Surgical excision, torus removal, and adjunctive silicone splint use enabled effective healing and a recurrence-free two-year follow-up. The findings highlight the importance of clinical expertise, conservative treatment, and careful assessment of trauma as a potential cause. This reinforces the need for precise differential diagnosis to prevent overtreatment of benign lesions while ensuring timely identification and management of malignant cases.

CONFLICT OF INTEREST

The authors have no conflicts of interest to disclose.

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The authors have no financial relationships relevant to this article to disclose.

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