

Case Report

A Rare Case of Amelanotic Melanoma with an Unknown Primary in the Buccinator Muscle

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Abstract

Malignant melanoma demonstrates strikingly varied clinical presentations, on account of which it is often challenging to diagnose. We describe herein a rare case of amelanotic malignant melanoma (AMM) in the buccinator muscle presenting as a solitary mass. A middle-aged woman presented with a non-pigmented swelling under the right buccal mucosa. Magnetic resonance imaging (MRI) revealed a well-circumscribed lesion without local invasion. A needle biopsy did not establish the diagnosis and was instead interpreted as suggestive of a salivary gland tumor, reinforcing an initial impression of myoepithelioma. Histopathological analysis of an excised specimen revealed poorly defined, pleomorphic tumor cells and a scattering of melanin-containing cells. The diagnosis of AMM was established by integrating these histopathological features with the clinico-radiological findings and the immunohistochemical results, which were positive for HMB-45, S-100, and SOX10 and negative for cytokeratin. Whole-body positron-emission tomography-computed tomography found no primary lesion. Because tumor nests suggesting possible residual disease were observed near the resection margin, a wider, secondary excision including the affected portion of the buccal mucosa and overlying skin was performed together with neck dissection. Histopathological examination of the re-resection specimen showed no residual tumor, indicating that no malignant cells were identified in the adjacent oral mucosa or facial skin. The patient has remained disease-free for three years.

Keywords: amelanotic malignant melanoma (AMM); melanoma of unknown primary (MUP); head and neck cancer

Academic Editor: Thomas Vogl

Received: 1 July 2026

Revised: 23 July 2026

Accepted: 31 July 2026

Published: 4 August 2026

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1. Introduction

Amelanotic malignant melanoma (AMM) accounts for only 2% of all melanomas and is characterized by minimal or absent pigmentation [1–3]. Its lack of melanin and its non-specific clinical features often mimic benign lesions, delaying diagnosis and treatment [1,4]. Melanoma of an unknown primary (MUP), which is thought to result from regression of the primary lesion, further complicates the diagnostic picture [5]. Each of these entities is uncommon in isolation. Amelanotic melanoma of the oral cavity is exceptionally rare; in a clinicopathologic series of oral amelanotic melanomas, the absence of pigment led to a wide range of clinical misdiagnoses, most often as benign or reactive

mucosal lesions, with consequent diagnostic delay [6]. Melanoma presenting within the skeletal muscle of the head and neck is likewise rare, and reports of intramuscular MUP are limited to isolated cases, almost all involving the temporalis muscle and almost all pigmented [7,8]. To the best of our knowledge, however, the convergence of all three features—an amelanotic phenotype, an intramuscular location within the buccinator muscle, and the absence of an identifiable primary lesion—has not previously been described. This combination is of clinical importance because each feature independently removes one of the diagnostic cues on which the recognition of melanoma ordinarily depends, and their coincidence can direct the clinician toward an entirely non-melanocytic differential diagnosis, as occurred in the present case. Herein we report a rare case of AMM in the buccinator muscle with features pointing to MUP. The present case illustrates pitfalls in diagnosing this type of cancer, emphasizes the value of immunohistochemistry as part of an integrated diagnostic assessment, and underscores the value of multidisciplinary management.

2. Case Report

A middle-aged woman with no significant medical history presented with a gradually enlarging, right buccal mass of several years' duration. Shortly before the current presentation, the lesion had begun increasing in size, prompting medical evaluation. An examination found a solitary, mobile, non-tender swelling under the right buccal mucosa, intact overlying mucosa and skin, and no fistulae (Figure 1A,B). The patient had no relevant medical or surgical history and no family history of melanoma or any other cutaneous malignancy. She had never undergone removal of a skin tag, nevus, or any other cutaneous lesion, and no previous dermatopathological analysis was therefore available. This is of relevance because the excision and discarding of an unexamined cutaneous lesion is one of the mechanisms by which a primary melanoma may go unrecorded; its absence here means that no such explanation for an occult primary could be invoked.

Magnetic resonance imaging (MRI) revealed a well-demarcated, encapsulated mass within the buccinator muscle but no evidence of local invasion (Figure 1C). A needle biopsy was performed, but it did not yield a definitive diagnosis. Rather than being simply non-contributory, the biopsy was actively misleading: the findings were interpreted as suggestive of a salivary gland tumor, and it was on this basis that myoepithelioma became the leading preoperative impression, with the differential diagnosis including other salivary gland tumors, schwannoma, and lymphoma. Two features of the lesion account for this. First, because the tumor was amelanotic, the sampled cells contained no pigment, and the single most recognizable cue to a melanocytic lesion was therefore absent from the biopsy material. Second, the epithelioid cytomorphology of the tumor cells, sampled in a small volume of tissue and divorced from the architectural context of the lesion as a whole, is compatible with a myoepithelial proliferation, and in a mass lying deep to an intact buccal mucosa, a salivary gland origin was the more probable interpretation on prior grounds. The biopsy therefore did not merely fail to establish the diagnosis; it reinforced an incorrect one. Given the diagnostic uncertainty, surgical excision was performed (Figure 2A,B).

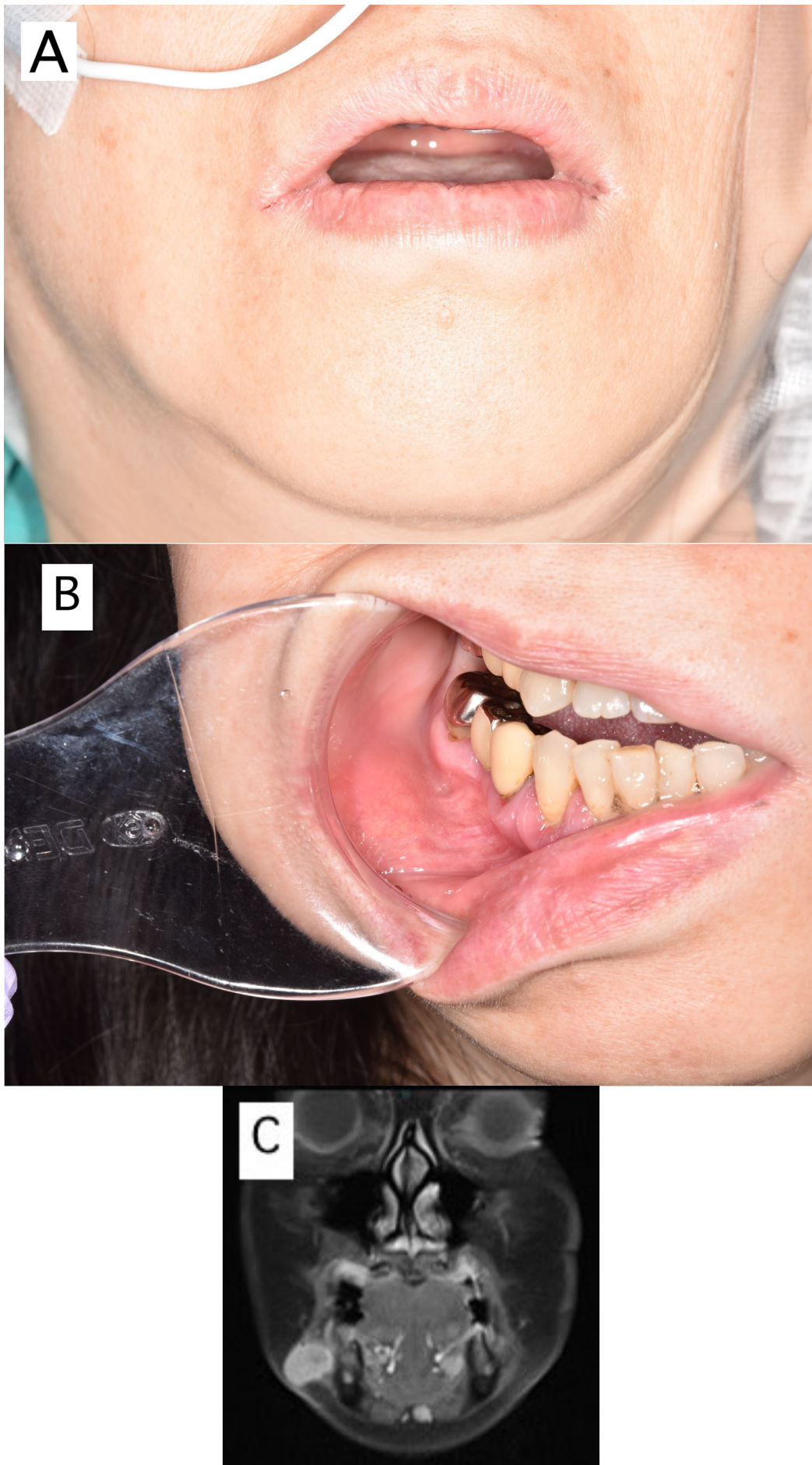


Figure 1. A solitary, mobile, non-tender swelling was palpable in both the external cheek skin (A) and internal buccal mucosa (B). Magnetic resonance imaging demonstrated a well-circumscribed, encapsulated lesion within the buccinator muscle (C).

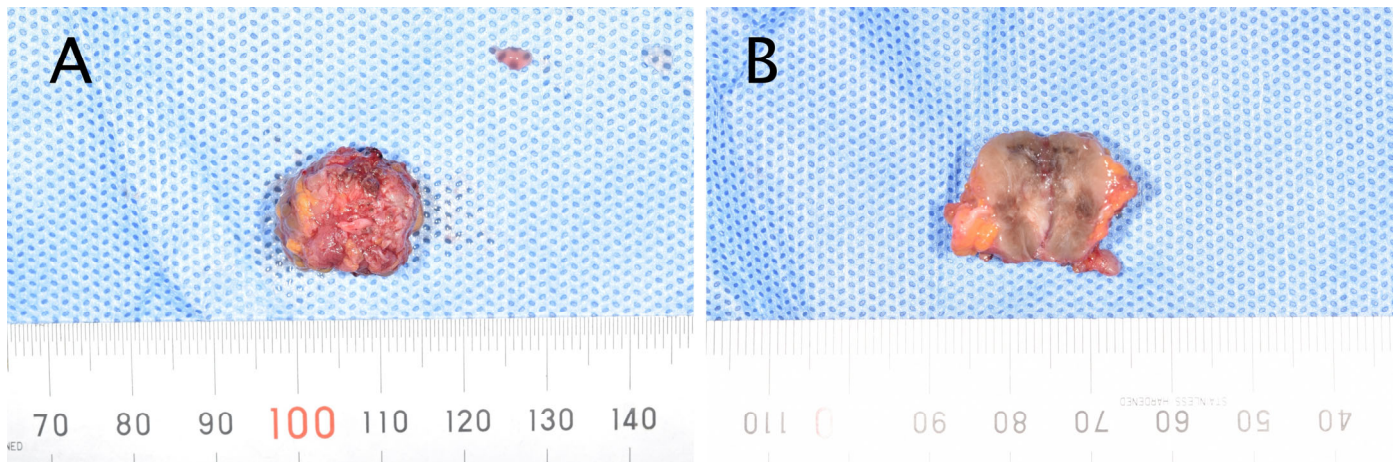
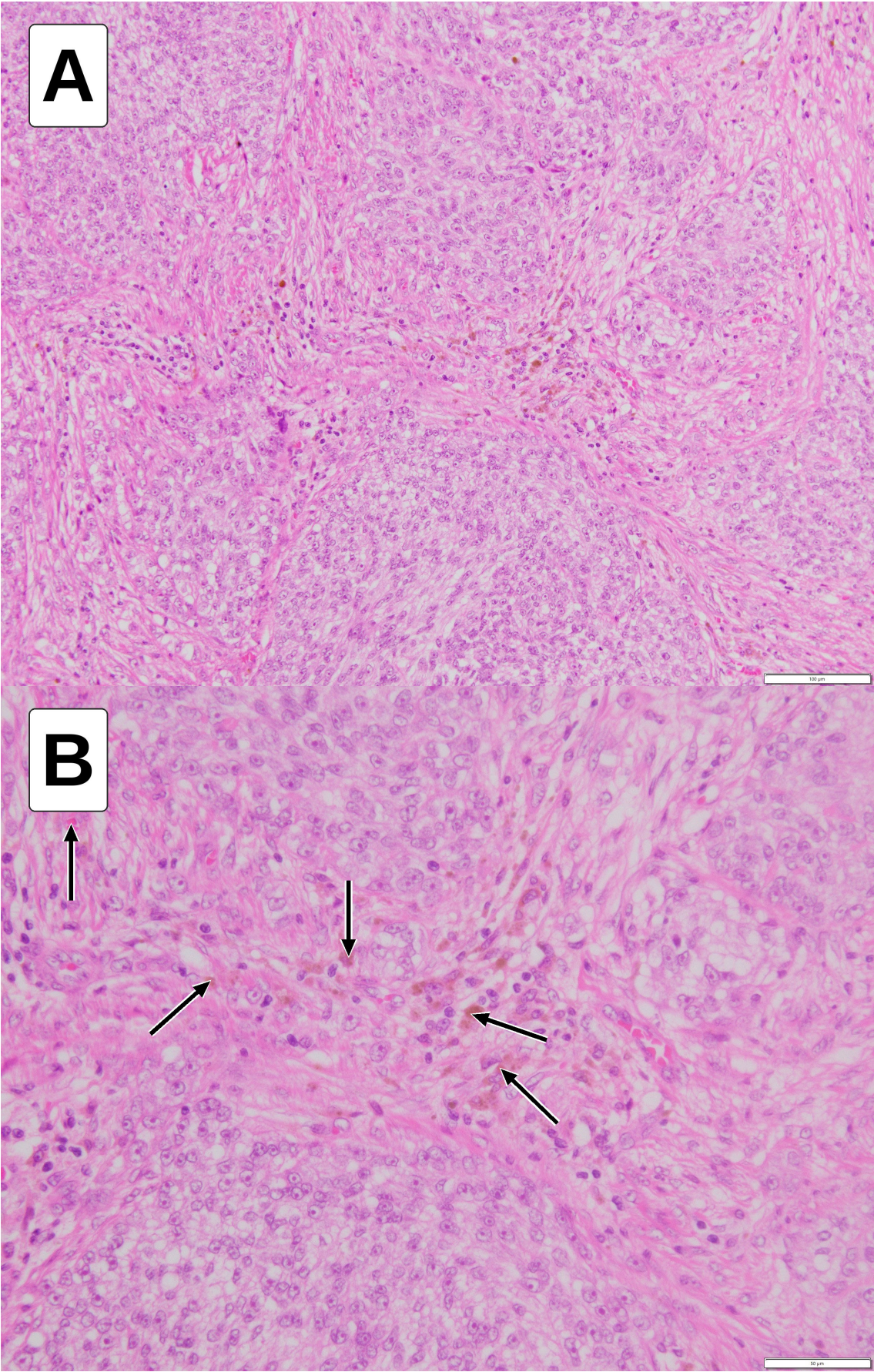


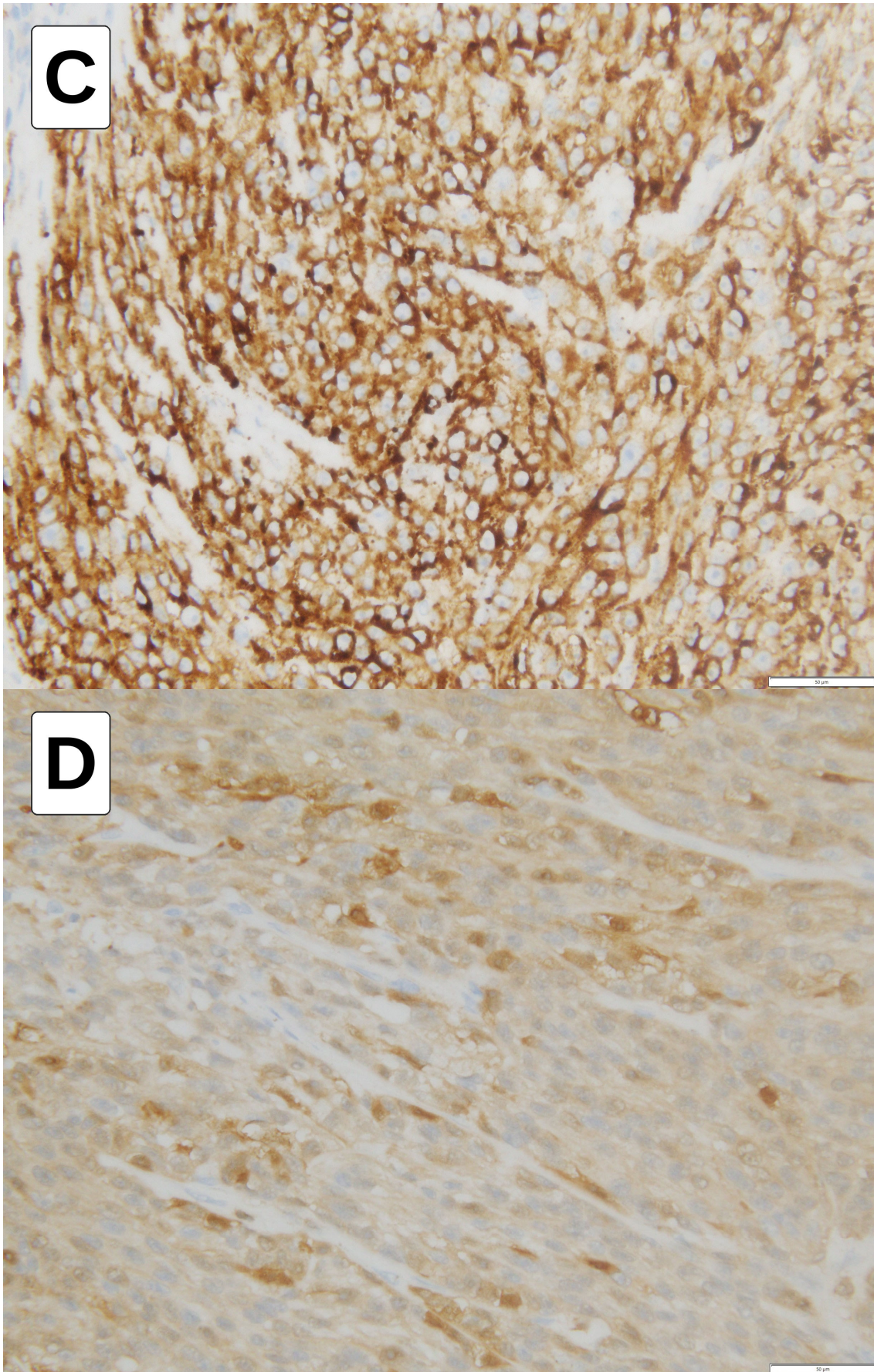
Figure 2. The surgical specimen (A). The cut surface of the tumor (B).

Histopathological analysis revealed an amelanotic malignant melanoma. Hematoxylin-eosin (H-E) staining demonstrated poorly defined, pleomorphic tumor cells containing eosinophilic cytoplasm and clear cell-like components (Figure 3B). The nuclei were enlarged and round to oval with prominent nucleoli. Mitotic activity was low, and scattered, melanin-containing cells were observed (Figure 3B, arrows). The mass was unencapsulated. Polygonal to spindle-shaped tumor cells with prominent eosinophilic nucleoli formed solid tumor nests that were irregularly compartmentalized by intervening stroma (Figure 3A). Growth was infiltrative rather than expansile, with tumor cells extending into the adjacent adipose tissue and skeletal muscle. Neither lymphovascular nor perineural invasion was identified, although scattered tumor cells extended close to the resection margin. Tumor necrosis was not identified. Immunohistochemistry showed diffuse cytoplasmic positivity for HMB-45 (Figure 3C), positivity for S-100 (Figure 3D), and diffuse strong nuclear positivity for SOX10 (Figure 3E), while cytokeratin was negative. The diagnosis of AMM rested principally on two findings: diffuse HMB-45 positivity and the identification, on H-E, of scattered melanin-containing cells within the lesion. The latter is easily overlooked in an otherwise amelanotic tumor but is diagnostically important, as it demonstrates melanin synthesis within the tumor cell population. The remaining markers served to exclude the principal alternatives. HMB-45 was the critical discriminator, because S-100 and SOX10, although sensitive melanocytic markers, are not by themselves specific: both are also expressed by schwannoma, and S-100 by myoepithelial neoplasms, and neither of these entities expresses HMB-45. Schwannoma was further excluded by the absence of an Antoni A/Antoni B architecture, of nuclear palisading and of Verocay bodies. Myoepithelioma and other salivary gland neoplasms—the entities favored by the needle biopsy—were excluded by the negativity for cytokeratin, by the absence of a ductal or myoepithelial architecture and of any chondromyxoid stroma, and by an immunoprofile that did not support epithelial or myoepithelial differentiation. Lymphoma was excluded by the pleomorphic, cohesive, epithelioid cytomorphology and by the absence of a lymphoid immunophenotype. The combination of HMB-45, S-100, and SOX10 positivity with cytokeratin (AE1/3), SMA, GFAP (Glial fibrillary acidic protein), and Desmin negativity, together with the melanin-containing cells, therefore established the melanocytic nature of the tumor. The immunohistochemical panel performed on this specimen comprised HMB-45, S-100, SOX10, cytokeratin (AE1/3), SMA, GFAP (Glial fibrillary acidic protein), and Desmin. Melan-A (MART-1) was not performed. Melanocytic differentiation had

already been established by diffuse HMB-45 positivity together with strong, diffuse nuclear SOX10 expression and S-100 positivity, corroborated by the melanin-containing cells identified on hematoxylin-eosin staining; a further melanocytic marker was therefore not considered necessary for diagnosis at the time.

Staging with positron-emission tomography-computed tomography (PET-CT) found no primary lesion. However, because tumor nests suggesting possible residual disease were detected near the initial resection margin, a wider, secondary excision including the buccal mucosa and overlying facial skin was performed together with neck dissection. Preoperative imaging demonstrated several enlarged lymph nodes in the submandibular region (level Ib). Because a melanoma with residual disease at the resection margin had been established and radiologically suspicious regional nodes were present, a comprehensive neck dissection clearing levels I to V was performed. A total of 29 lymph nodes were retrieved, and all were free of metastasis. The rationale for undertaking a comprehensive rather than a selective dissection was twofold. First, radiologically enlarged nodes were present and could not be assumed to be reactive in a patient with a confirmed melanoma; nodal clearance with definitive pathological staging was therefore indicated. Second, and reinforcing this, no primary lesion had been identified despite whole-body PET-CT and a full dermatological examination, so the possibility that the intramuscular deposit itself represented a regional metastasis from an occult primary could not be excluded. Under the AJCC framework, MUP presenting in subcutaneous tissue or lymph nodes is staged as stage III disease, for which regional nodal clearance is standard; and in the absence of a definable primary site, it was not possible to predict from a known index site which nodal levels were at greatest risk, which further favored comprehensive over selective clearance. Sentinel lymph node biopsy was not applicable in this setting, since the absence of a definable primary precludes the peritumoral tracer injection on which lymphatic mapping depends. That the retrieved nodes ultimately proved negative does not diminish the appropriateness of the decision, which rested on the radiological findings and the staging uncertainty present at the time. Histopathological analysis of the re-resection specimen, which incorporated the overlying buccal mucosa and facial skin, revealed no residual tumor; no malignant cells were identified in the sampled mucosa or skin, although this does not formally exclude microscopic involvement beyond the tissue examined. Importantly, no junctional (in situ) melanocytic component was identified within the mucosal epithelium. This argues against a primary mucosal melanoma of the buccal mucosa and supports interpretation of the intramuscular mass as a metastatic deposit. A clinical assessment by a dermatologist revealed no cutaneous lesions suggestive of a primary site, and no mucosal abnormalities were observed overlying the mass. Taken together, these findings suggested the possibility of melanoma of unknown primary (MUP), although a definitive diagnosis could not be established. As of this writing, the patient has remained disease-free for three years and has presented no clinical or radiological evidence of a recurrence or metastasis.





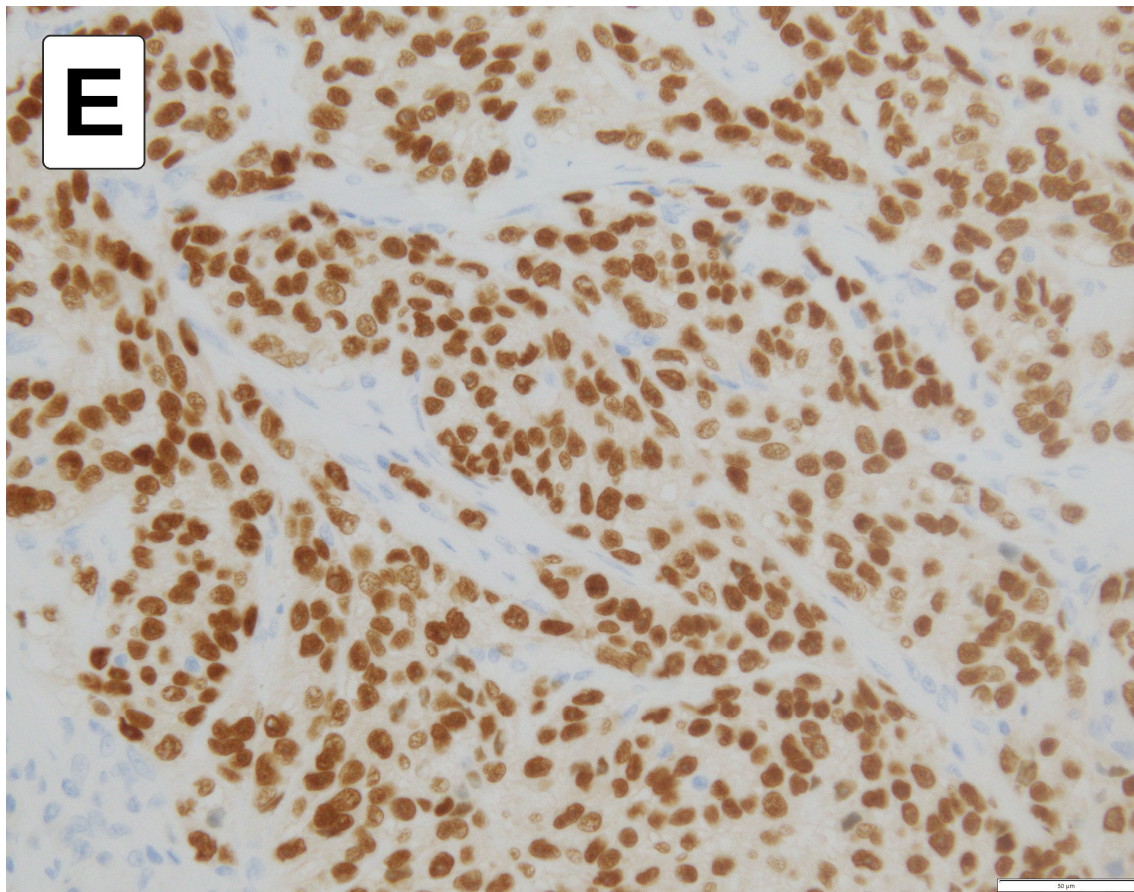


Figure 3. A low-power photomicrograph (A) shows the overall architecture of the unencapsulated lesion, in which solid tumor nests are irregularly compartmentalized by intervening stroma within skeletal muscle. At higher magnification (B), hematoxylin-eosin staining demonstrated poorly defined, pleomorphic tumor cells containing mixed, eosinophilic cytoplasm and clear cell-like components. The nuclei were enlarged, round to oval, and had prominent nucleoli. Scattered melanin-containing cells were observed (arrows, (B)). Diffuse cytoplasmic positivity for HMB-45 (C), positivity for S-100 (D), and diffuse strong nuclear positivity for SOX10 (E) were found. Scale bars: 100 μ m (A); 50 μ m (B–E).

3. Discussion

The present case underscores the importance of considering AMM in the differential diagnosis of non-pigmented, solitary, oral masses. AMM, a subtype of cutaneous melanoma comprising approximately 2% of melanoma cases, presents little or no pigmentation macroscopically or histopathologically [2,3]. Owing to its heterogeneous morphology and the absence of definitive diagnostic criteria, this rare variant is frequently misdiagnosed or detected only when already advanced. Its nonspecific appearance may delay its diagnosis and treatment, thereby worsening the prognosis [4]. On the other hand, due to its higher immunogenicity as seen in immunologically mediated, primary site regression, MUP has better outcomes than stage-matched AMM [9]. This reported survival advantage derives, however, from cohorts with conventional nodal or subcutaneous presentations, and should not be assumed to apply to an amelanotic, intramuscular presentation such as the present one. AMM is an extremely rare form of melanoma, and the oral mucosa often lacks pigmentation [3]. It is instructive to compare the present case with previously reported oral and intramuscular melanomas. In a clinicopathologic series of eight oral amelanotic melanomas with a review of the literature, Soares et al. found that the absence of pigment led almost uniformly to an initial clinical impression of a benign or reactive

process, and that the diagnosis ultimately rested on immunohistochemistry performed after excision—a sequence that mirrors the present case, in which myoepithelioma was suspected preoperatively [6]. Importantly, that series comprised mucosal lesions arising at the epithelial surface, whereas, in our patient, the tumor lay entirely within the buccinator muscle beneath an intact mucosa, so that even the surface-derived clinical cues available in those cases were absent. The amelanotic phenotype is not merely a diagnostic inconvenience: in a matched analysis of head-and-neck melanomas, Guo et al. showed that amelanotic tumors carry a significantly worse prognosis than stage-matched pigmented tumors, a difference attributed at least in part to delayed recognition [10]. Reports of melanoma presenting within the skeletal muscle of the head and neck are correspondingly scarce and have almost exclusively involved the temporalis muscle: Grech et al. described a MUP of the temporalis muscle that had been clinically mistaken for a lipoma [7], and Dalle Carbonare et al. reported a comparable temporalis lesion [8]. In both of those reports, however, the tumor was heavily pigmented, so that melanocytic differentiation became evident on sectioning; the present case differs in that the amelanotic phenotype removed even this final morphological cue. Taken together, these comparisons suggest that the present case is distinguished not by any single feature but by the coincidence of an amelanotic phenotype, an intramuscular location and an unidentifiable primary. Accordingly, to the best of our knowledge, no previous report has described AMM presenting as a suspected MUP within the buccal region. A further feature of the present case deserves emphasis, because it compounded the diagnostic difficulty. The radiological and histopathological assessments of circumscription were discordant: MRI demonstrated a well-demarcated mass without evidence of local invasion, whereas histological examination showed an unencapsulated tumor infiltrating the adjacent adipose tissue and skeletal muscle. The apparent circumscription on imaging therefore reinforced the preoperative impression of a benign or low-grade lesion suggested by the needle biopsy, and the infiltrative nature of the tumor became apparent only on histological examination of the excised specimen. In a non-pigmented intramuscular mass, neither imaging nor needle biopsy should be relied upon to exclude malignancy. Data specifically addressing the proportion of AMM presenting as MUP are limited; in one case series of 24 patients with MUP, only one case (4.2%) was amelanotic [11].

MUP was originally defined in 1963 by Dasgupta et al. as melanoma in the subcutaneous tissue, lymph nodes, or visceral organs without the presence of cutaneous, ocular, or mucosal primary [12]. Sixty percent of MUP presents in the lymph nodes—most frequently axillary lymph nodes, followed by cervical, inguinal, and parotid lymph nodes [13]. Other sites of presentation include the subcutaneous tissue, lung, gastrointestinal tract, and brain [14]. The American Joint Committee on Cancer (AJCC) classifies MUP presenting in a lymph node or subcutaneous tissue as stage three and MUP presenting within a visceral organ as stage four [13]. The majority of studies diagnose MUP using a multidisciplinary approach that incorporates patient history, physical examination, pathology, and imaging [15]. As a result, substantial heterogeneity exists in the criteria used to define MUP [16–20]. Variations also arise in the extent of physical examination considered adequate and in whether patients with preexisting cutaneous lesions are excluded. In the present case, a full skin examination—including subungual and mucosal surfaces—performed by a dermatologist, as well as whole-body evaluation with PET-CT, revealed no lesions suggestive of a primary site. Therefore, although a definitive diagnosis could not be established, the possibility of MUP was considered.

MUP is thought to arise from an undetected, spontaneously regressed primary melanoma [5]. This hypothesis may explain the absence of a primary lesion in our patient. Preoperative fine needle aspiration (FNA) findings were inadequate to diagnose the present case. Indeed, melanoma is difficult to diagnose even on the basis of histopathological

findings because it is histomorphologically diverse and often masquerades as any of a number of non-melanocytic neoplasms, especially amelanotic types. As each tumor type expresses a characteristic marker pattern, immunohistochemical analysis is often essential for a definitive diagnosis [21]. S-100 and HMB-45 are well-known melanoma cell markers, and S-100 reportedly has a sensitivity of 97–100% for malignant melanoma [22]. However, parallel staining with more specific immunohistochemical markers is required because the specificity of S-100 is relatively low (75–87%) [22]. HMB-45 has a sensitivity of 69–93% (77–100% for primary melanoma and 56–83% for metastatic melanoma) [22], and staining with HMB-45 is extremely specific for malignant melanoma [22–24]. Contemporary diagnostic practice, however, no longer relies on S-100 and HMB-45 alone. SOX10 is now widely regarded as the optimal single melanocytic marker, offering sensitivity comparable to S-100 but greater specificity, and retaining reactivity in spindle cell and desmoplastic variants in which Melan-A (MART-1) and MiTF are frequently negative [25]. In a comparative series of melanoma metastases, S-100 and SOX10 each achieved 100% sensitivity, with Melan-A at 97% and HMB-45 at 95%, whereas the specificity of S-100 was the lowest of the panel at 85%, reflecting its reactivity in histiocytes [26]. PRAME (preferentially expressed antigen in melanoma) has more recently emerged as a highly specific adjunct that assists in distinguishing malignant from benign melanocytic proliferations, although its sensitivity is lower than that of the conventional markers, reported at approximately 85% in metastatic disease [26]. A panel-based approach combining a nuclear marker such as SOX10 with a cytoplasmic marker such as HMB-45 or Melan-A, supplemented where necessary by PRAME, is therefore preferable to any single stain, particularly in amelanotic tumors where morphology provides little support. In the present case, this principle is directly illustrated: the diagnosis was supported by a panel that paired the cytoplasmic marker HMB-45 with the nuclear marker SOX10, the latter showing diffuse strong positivity (Figure 3E), alongside S-100 positivity and cytokeratin negativity. The concordance of these markers, and in particular the addition of SOX10 to the more traditional S-100 and HMB-45, strengthened the immunohistochemical argument for a melanocytic tumor and helped exclude the epithelial and myoepithelial entities suggested by the needle biopsy. We nonetheless acknowledge as a limitation that a broader contemporary panel—for example, the further addition of Melan-A or PRAME—was not undertaken. Treatment for melanoma, including AMM, consists of surgical excision with wide margins; chemotherapy; to a lesser extent, immunotherapy and radiation; and finally, continuous monitoring of the nodal status [6,27]. Monitoring should also include regular skin and mucosal check-ups provided by a dermatologist. This is of particular importance when no primary lesion has been identified, since an occult primary may declare itself during follow-up, and since patients with a melanoma of any type remain at risk of a subsequent independent primary lesion. It should be emphasized that our patient has been followed for only three years. Although she remains free of disease, this interval is too short for the outcome to be regarded as established: melanoma is well recognized for late recurrence, and the natural history of an amelanotic melanoma arising within skeletal muscle with no identifiable primary is essentially unknown. Prolonged surveillance is therefore necessary, and the favorable course to date should be interpreted with corresponding caution.

4. Conclusions

The absence of melanin and slow growth of AMM can mimic a benign lesion, thereby delaying the diagnosis. If the biopsy findings of a head-and-neck tumor are insufficient for a definitive diagnosis, melanoma should be retained in the differential diagnosis and immunohistochemistry incorporated into the diagnostic work-up. Immunohistochemistry does not, however, establish the diagnosis by itself: it is an adjunct whose results must

be interpreted together with the histomorphological findings and the clinical and radiological context, and, where a MUP is suspected, alongside a thorough dermatological examination and systemic staging.

Author Contributions: Conceptualization, Y.M.; investigation, Y.W.; writing—original draft preparation, Y.M.; writing—review and editing, R.N.; supervision, Y.W. and A.W.; project administration, Y.M. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: No new data were created or analyzed in this study. All data supporting the findings of this case report are contained within the article. Further clinical details are not publicly available in order to protect the privacy of the patient and can be obtained from the corresponding author upon reasonable request.

Conflicts of Interest: The authors declare no conflicts of interest.

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