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Case Report

Myopericytoma in the oral cavity: a rare case report and literature review

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Abstract: Myopericytoma (MPC) is a rare, benign, and cellular mesenchymal neoplasm. Histologically, it is characterized by concentric perivascular proliferation of spindle-shaped tumor cells. While MPCs predominantly occur in the subcutaneous or superficial soft tissues of distal extremities, oral MPCs are exceptionally rare. Herein, we present a case of MPC located in the sublingual region of a 74-year-old male. Additionally, we conducted a systematic review of 29 previously reported cases to delineate the clinicopathological and molecular features of MPC. This comprehensive analysis aims to improve diagnostic accuracy and enhance the cognition of this uncommon tumor entity's clinicopathological characteristics and biological behavior.

Keywords: Myopericytoma, mesenchymal tumor, oral cavity, mouth

Introduction

Myopericytoma (MPC), a rare slow-growing mesenchymal neoplasm, predominantly demonstrates benign behavior. These lesions typically manifest as well-demarcated subcutaneous nodules, with preferential localization to the subcutaneous and superficial soft tissue of distal extremities [1], followed by proximal extremities and internal organs such as the liver, spleen and kidney [2-5]. Their occurrence in the oral cavity constitutes an extraordinary rarity within diagnostic pathology.

Histopathologically, MPC displays hypercellular architecture featuring a morphological spectrum from oval to spindle cells, arranged in concentric patterns around vascular vessels-a hallmark of perivascular myoid differentiation. Due to its overlapping histological features with other mesenchymal spindle cell tumors, systematic immunohistochemical evaluation becomes imperative to exclude morphologic mimics.

While histomorphological analysis combined with immunophenotyping remains the diagnostic gold standard, the inherent diagnostic complexity underscores the necessity for multidisciplinary collaboration between clinicians and pathologists to achieve precise differentiation. In this context, we elucidate a novel case of sublingual MPC accompanied by a meta-analysis of 29 documented cases from 24 publications [6-29], thereby advancing the understanding of diagnostic challenges and management paradigms for this entity.

Case presentation

A 74-year-old male was referred to Shanghai Stomatological Hospital for evaluation of a painless, slow-growing sublingual mass in one-month duration. Physical examination identified a semi-firm mass in the mouth floor with well-circumscribed margins, mobile upon palpation without evidence of tissue adhesion. Magnetic resonance imaging (MRI) revealed a well-circumscribed spherical soft tissue mass occupying the sublingual space (**Figure 1**). The body of the tongue was pressed upward. The lesion demonstrated close anatomical proximity to the mandibular cortex, comprehensive radiographic evaluation showed preserved cortical integrity without evidence of osseous involvement or periosteal reaction.

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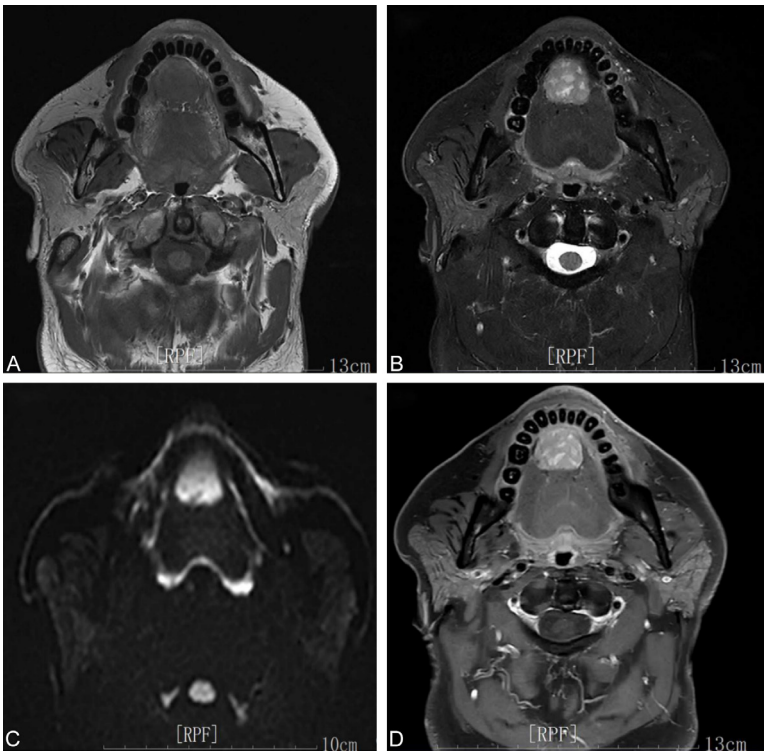

Figure 1. The soft tissue lesion by magnetic resonance imaging (MRI) in the sublingual region of the mouth. A. MRI displayed a well-defined, circular soft tissue lesion in the sublingual region of the mouth, which appeared isointense on the T1WI sequence. B. T2WI sequence showed the lesion with mixed isointensity and hyperintensity. C. The lesion presented as hyperintense on the DWI sequence. D. After contract enhancement, the lesion demonstrated heterogeneous enhancement, with regions varying from moderate to marked signal intensity elevation.

Local resection was performed, and the specimen was submitted for pathological analysis. A gross evaluation revealed a well-circumscribed soft tissue nodule with a rubbery texture. The lesion measured approximately 2.5 cm × 2.5 cm × 2.2 cm. The cut surface appeared yellow-brown with randomly distributed cystic spaces.

Histopathological examination with hematoxylin and eosin staining demonstrated a well-circumscribed, hypercellular proliferative lesion containing a prominent vascular network (**Figure 2A**). Tumor cells were oval to short spindle-shaped with eosinophilic cytoplasm and uniform chromatin. Notably absent were significant nuclear atypia, increased mitotic activity, or necrosis. Histologically, the lesion exhibited characteristic fascicular and sheet-like growth patterns concentrically arranged around prominent squeezed, thin-walled vessels interspersed throughout (**Figure 2B**). In some areas, tumor cells demonstrated a distinctive morphology of concentric perivascular proliferation (**Figure 2C**). The tumor stroma demonstrated prominent myxoid degeneration with secondary cystic change, accompanied by numerous dilated thin-walled vessels (**Figure 2D**, **2E**). The unique pseudo-cystic stromal changes with angiomatous proliferation mimicked cavernous hemangioma histologically. Focal whirling myoid nodules protruding into vascular lumens were observed as well (**Figure 2F**). Tumor cells were notably positive for αlpha-smooth muscle actin (α-SMA) and calponin (**Figure 3A**) but negative for desmin, s100 calcium-binding protein (S-100), SRY-box transcription factor 10 (SOX10), signal transducer and activator of transcription (STAT6), β-catenin, and cytokeratin pan (CKpan) by immunohistochemical staining (**Figure 3B**). Cluster of differentiation 34 (CD34) and ETS-related gene (ERG) highlighted the vascular endothelial network rather than tumor cells (**Figure 3C**). The tumor cells had a low Ki-67 proliferation index, less than 5% (**Figure 3D**). Histopathological examination and immunohistochemical evaluation confirmed definitive myopericyte differentiation in the proliferating spindle cells. Therefore, the lesion was diagnosed as MPC in the mouth floor.

At the 5-month postoperative follow-up, the surgical site demonstrated satisfactory healing. Regular clinical examinations and MRI surveillance were recommended to evaluate for residual disease or recurrence.

Discussion

MPC is a rare benign mesenchymal neoplasm characterized by spindle-shaped perivascular myoid cells, which display apparent differentiation towards perivascular myoid cells named myopericytes [30]. Myopericytes are sited around capillaries and small vessels, and are

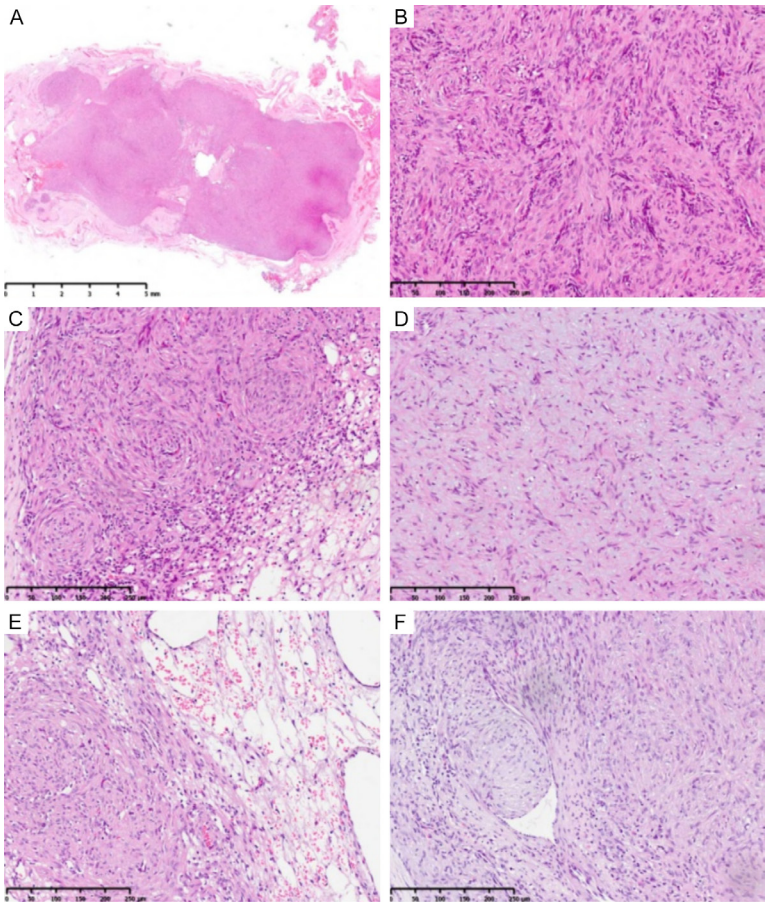

Figure 2. Histological features by hematoxylin and eosin (H&E) staining of myopericytoma (MPC). (A) Clear fibrovascular envelope of MPC. (B) The tumor demonstrated hypercellular fascicular or swirling growth patterns with squeezed, thin-walled vessels interspersed throughout. (C) The distinctive pattern of concentric perivascular growth around the blood vessels. (D) Mucous degeneration in the mesenchyme with sparse tumor cells. (E) The mimicking pattern of cavernous hemangioma by obvious mucous degeneration or microcystic change along with numerous gaping and dilated thin-walled vessels embedded inside. (F) Whirling myoid nodules protruding into the vascular lumen. ×5 (A), ×100 (B-F). Bar = 5 mm (A), 250 μm (B-F).

considered a transitional cell type between pericytes and vascular smooth muscle cell lines [31]. The contractile function of myopericytes could regulate blood vessel diameter and capillary permeability [7]. Besides vascular functions of angiogenesis and blood pressure regulation, myopericytes are involved in tumor formation, growth, vascular invasion, and metastasis. Additionally, myopericytes are regarded as a type of perivascular stem cells capable of differentiating into multiple mesenchymal lineages [32].

"Hemangiopericytoma" was first named to describe a class of tumors characterized by round to oval cells and a presumed pericytic origin in 1942. The terminology "myopericytoma" was adopted to refer to a subset of pericytic tumors featuring concentric perivascular proliferation by myoid spindle-shaped cells around blood vessels in 1998. The World Health Organization (WHO) endorsed the terminology "myopericytoma" in 2002 to distinguish this lesion from "hemangiocytoma" previously described. Due to the significant overlap in morphology and histogenesis, myopericytoma, myofibroma, angioleiomyoma, and glomus tumor were classified under the broad category of pericytic neoplasms by the WHO classification of soft tissue tumors and bone in 2013.

The pathogenesis of MPCs remains poorly understood. Current evidence suggests an association between MPCs and prior trauma or chronic scarring, as documented in the literature [33]. MPC of our case was sited in the base of the oral cavity, where friction might trigger tumor formation. Epstein-Barr virus (EBV) has been etiologically implicated in rare cases of MPC, particularly among immunocompromised patients with acquired immunodeficiency syndrome (AIDS) [34, 35]. However, the mechanism underlying EBV-associated MPC remains unclear.

To improve the recognition of MPC and clarify its clinicopathological differences between oral and extraoral manifestations, we conducted a systematic review of 29 patients from 24 publications along with our original case, providing a comprehensive characterization of oral MPCs (**Tables 1**, **2**). MPCs have been reported across a broad age range, from newborns to elderly patients up to 84 years old [27, 36]. Combining literature reports and our MPC case data, we found that the age of onset for oral MPC ranges from 6 to 74 years (median: 42.17 ± 3.57), with a slight female predominance (male-to-female

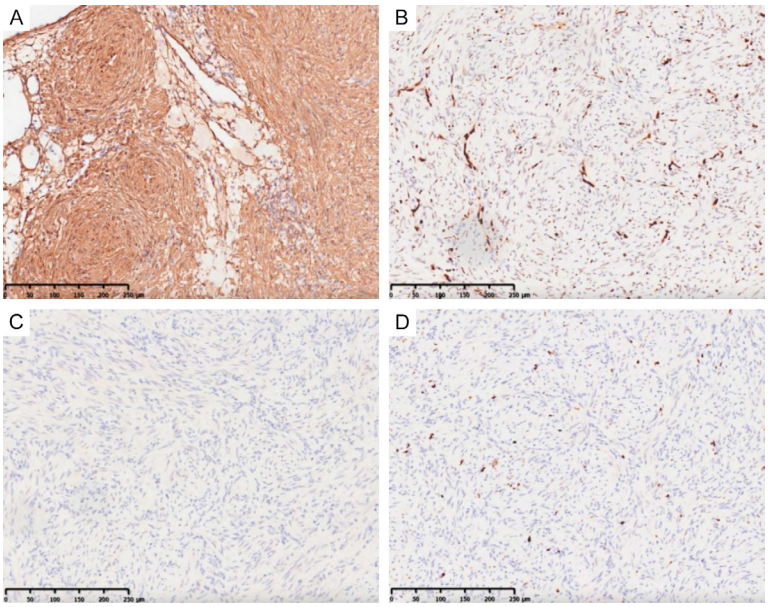

Figure 3. Immunohistochemical staining for MPC by EnVision, ×100. Bar = 250 μm. A. Tumor cells were positive for immunohistochemical staining of α-smooth muscle actin (α-SMA). B. Immunohistochemical staining of ETS-related gene (EGR) highlighted the vascular endothelial network instead of tumor cells. C. Tumor cells were negative for SRY-box transcription factor 10 (SOX10). D. Tumor cells displayed a low ki67 proliferation index.

ratio: 1:1.14). MPC typically arises in subcutaneous or superficial soft tissues of distal extremities. In contrast, oral MPC shows no site predilection. In our analysis, oral MPCs occurred at diverse sites: tongue (6 cases), lip (6), parotid gland (8), gingiva (5), buccal mucosa (3), base of oral cavity (1), and parapharynx (1). MPCs usually present as slow-growing, painless nodules that could last for several years and lack specific symptoms [37]. However, intravascular MPC could cause spontaneous deep vein thrombosis, leading to sudden-onset pain and swelling in the upper arm [38, 39]. MPC of the axillary artery can even cause strokes that manifest as a transient speech disorder [40]. Compared to its variable manifestations at other anatomical sites, the most common clinical presentation of MPCs in the oral cavity was painless, slow-growing masses. In our cohort, uncommon manifestations included pain (4 cases), bleeding (2), and ulceration (1).

MPCs generally measure ≤ 2 cm in diameter [41]. However, larger tumors may develop in deep-seated or visceral locations, as demonstrated by this hepatic case measuring up to 5.5 cm [42]. The tumor diameter of MPCs in the oral cavity ranged from 0.5 to 6.0 cm (mean: 2.33 ± 0.29 cm) (**Table 1**). Single lesions are relatively more common than multiple lesions and frequently occur in specific anatomic sites, particularly the foot or head and neck region. EBV-associated MPCs in AIDS patients often manifest as multiple lesions in unique sites, including intracranial, spinal canal, tongue, vocal cords, bronchus, periampullary regions, or other specialized parts [35]. MPCs in the oral cavity predominantly presented as solitary tumors, with exceptions including one case of multiple parotid gland masses and three cases of multifocal lesions. Compared to superficial nodules, multinodular or deep-seated tumors often display more aggressive biological behavior and higher recurrence rates. Long-term patient survival, even after incomplete resection, suggests that multi-site involvement may represent multicentric growth rather than metastatic spread [9].

As MPC is prone to misdiagnosis due to the absence of unique clinical and typical imaging features, excisional biopsy and histological examination are essential for definitive diagnosis. Applying strict morphologic criteria and selecting appropriate immunohistochemical markers can distinguish MPC from its analog. MPC usually presents as unencapsulated nodular proliferation with numerous thin-walled vessels. A distinctive feature of MPC is the concentric and perivascular arrangement of tumor cells around the blood vessels [41]. Eosinophilic and whirling myoid nodules may protrude into the vascular lumen. Occasionally, intravascular MPC presents as a lesion entirely within the venous lumen. However, none of the intravascular MPC represents a vascular invasion as no detectable tumor outside the vessel [38]. The MPC cells characteristically demonstrate oval to spindle-shaped morphology, featuring uniformly dispersed chromatin, inconspicuous nucleoli, and moderately abundant eosinophilic

Table 1. Clinical characteristics of 29 patients according to literature review

References	Sex	Age	Location	Symptoms	Size (cm)	Follow up (months)	Recurrence
[6]	F	45	Right buccal mucosa	Painless	2	108	no
[7]	F	36	Left lateral tongue	Painless	0.5	/	/
[8]	F	72	Alveolar gingiva	Painless	1	18	no
[9]	M	42	Tongue base, vocal cord, right frontal lobe, right cavernous sinus, left cavernous sinus	Painless	0.8	60	no
[10]	M	28	Lower lip	Painless, ulcer	1.5	36	no
[11]	F	41	Right parotid gland	Painless	5.6	9	no
[12]	M	65	Left parotid gland	Painless	6	24	no
[13]	F	43	Right parotid gland, right ear, inferotemporal concavity, parapharyngeal space, submandibular region, cheek	Painful	5.6	60	no
[14]	M	61	Right cheek mucosa	Painless	1	6	no
[15]	F	32	Left cheek, left facial area, parotid gland	Painless	2	96	no
[16]	F	61	Left mid-lateral tongue	Painless	2	18	no
[17]	M	14	Upper lip	Painless	2	6	no
[18]	M	48	Tongue base	Painless	3.2	36	no
[19]	F	42	left lower lip	Swelling, tenderness	3	6	yes
[20]	F	46	Parotid gland	Swelling	3.7	56	no
	M	10	Tongue	Swelling, bleeding	3.8	51	no
	F	41	Buccal mucosa	Painless	1.6	27	no
	M	61	Tongue	Painless	0.5	25	no
	F	62	Submandibular region	Swelling	3.5	8	no
[21]	M	46	Upper lip	Swelling	/	8	no
[22]	F	12	Gingiva	Painless	/	60	no
[23]	M	6	Right maxilla	Bleeding	2	96	no
[24]	M	62	Left parotid gland	Painless	3	17	no
	F	48	right parotid gland	Painless	2	60	no
[25]	F	42	anterior mandible	Painless	1	12	no
[26]	F	28	Right parotid gland	Painful	2.4	5	no
[27]	F	24	Lower lip	Painful	0.8	43	no
[28]	M	66	Parapharyngeal localization	Painful	1.3	/	no
[29]	M	7	Upper lip	Painless, swelling	1	3	no

Abbreviations: M, male; F, female; /, no detail information.

Table 2. Pathological features of 29 patients according to literature review

References	Morphology	SMA	Vimentin	H-Caldesmon	Calponin	Desmin	ki67
[6]	CPP, HLP, TPVL	+	/	+	/	-	/
[7]	CPP, HC	+	/	/	/	/	/
[8]	CPP, mitoses	+	/	+	/	Focal+	/
[9]	CPP, atypia	+	/	/	/	-	/
[10]	CPP, HC	+	+	/	/	-	/
[11]	CPP, HC	+	/	/	/	-	/
[12]	CPP	+	+	/	/	/	/
[13]	CPP, TPVL	+	/	/	/	-	/
[14]	CPP, atypia, mitoses	+	+	/	/	-	40%
[15]	CPP, HLP	+	/	/	/	/	/
[16]	CPP	+	/	/	/	-	5%
[17]	CPP, FB	+	/	/	/	/	/
[18]	HLP	/	/	+	/	/	< 5%
[19]	CPP	+	/	/	/	/	/
[20]	CPP, HLP, MD	+	+	/	/	-	< 5%
		Focal+	+	/	/	-	2%-3%
		Focal+	+	/	/	Focal+	1%
		+	+	/	/	-	< 5%
		+	+	/	/	-	1- 8%
[21]	HLP, HC	+	+	/	/	-	< 5%
[22]	CPP, HLP	Focal+	+	+	+	/	35%
[23]	CPP, FB	+	/	/	+	/	/
[24]	CPP	+	/	/	/	/	/
	CPP	+	/	/	/	-	/
[25]	HLP	+	/	/	/	/	/
[26]	CPP, HLP, FC, MD	+	/	+	+	Focal+	15%
[27]	CPP	+	/	+	/	Focal+	1%
[28]	CPP, HC	+	/	+	/	/	/
[29]	CPP, MD, FC	+	/	Positive	/	-	20%

Abbreviations: CPP, concentric perivascular pattern; HLP, hemangiopericytoma-like pattern; TPVL, tumor protruding into the vascular lumen; MD, mucinous degeneration; FC, fibrous capsule; FB, fibrocollagenous bundles; HC, hyalinized collagen; SMA, α-smooth muscle actin; +, positive expression; -, negative expression; /, no detail information.

cytoplasm. Immunohistochemical evaluation reveals that the MPC cells exhibit diffuse strong positivity for alpha-smooth muscle actin (α-SMA), muscle-specific actin (MSA), calponin, and h-caldesmon [3]. In contrast, desmin immunoreactivity is either completely absent or only focally and weakly expressed. All the MPC tumor cells we analyzed were uniformly positive for SMA and mostly negative for desmin (weakly positive in rare cases) (**Table 2**). In addition, the tumor cells consistently show negative staining for S-100 protein, SOX10, and STAT6. Notably, immunohistochemical staining for CD34 selectively highlights the vascular network within the stroma, with a complete absence of staining in tumor cells. Ki-67 proliferation index of MPCs was generally low, mostly < 5%. While with low-grade malignant transformation, tumor cells could display the feature of cellular atypia, scattered mitotic figures, and elevated Ki-67 proliferation index (**Table 2**).

Malignant MPCs display concentric growth around vascular channels similar to the pattern of benign MPCs, but mixed with ill-defined cell borders, marked nuclear atypia, abundant abnormal mitotic figures, zones of necrosis, increased proliferative activity, and deeply infiltrative growth [43].

Histopathological examination revealed that the MPC of our case demonstrated characteristic alternating hypercellular and hypocellular

areas, accompanied by microcystic degeneration resulting from stromal mucin accumulation in the hypocellular regions. These morphological characteristics bore a striking resemblance to those of schwannoma, prompting its inclusion as our primary differential diagnosis. Schwannomas typically demonstrate alternating Antoni A (cellular) and Antoni B (hypocellular) areas. The Antoni A regions characteristically show tumor cells arranged in palisading patterns while lacking the perivascular concentric growth pattern that is typical for MPCs. The Antoni B areas feature sparse tumor cells within a loose myxoid stroma that frequently undergoes cystic degeneration, forming variably sized cysts. The prominent stromal myxoid degeneration posed diagnostic challenges by simulating Antoni B areas histologically. Notably, the presence of large, irregular, thick-walled hyalinized vessels in these hypocellular areas was diagnostic, being pathognomonic for schwannomas and excluding MPC. Furthermore, the immunohistochemical analysis provided definitive diagnostic clarification: schwannoma cells showed diffuse strong positivity for neural markers S-100 and SOX10 while negative for myopericytes markers-α-SMA and calponin [44]. The MPC cells exhibit the inverse immunophenotype: negative for neural markers but positive for markers of myopericytes.

All reviewed cases of oral MPC in our literature analysis (**Table 2**) demonstrated varying proportions of a concentric perivascular growth pattern. Additional variable histological features included tumor cells surrounded by prominent gaping, branching, thin-walled vessels-mimicking hemangiopericytoma, as well as multinodular growth patterns, collagen-rich or hyalinized stroma, mucinous degeneration, calcifications, fibrous capsules, and occasional nerve involvement in parotid gland cases. Tumor cells were uniformly positive for SMA and mostly negative for desmin (weakly positive in rare cases). As part of the perivascular tumor spectrum, MPCs share overlapping morphological characteristics with other myogenic tumors, such as solitary fibrous tumors (SFT), myofibromas, and glomus tumors. The SFT is characterized by the proliferation of ovoid to spindle-shaped cells accompanied by numerous thin-walled, branching blood vessels that form a distinctive “staghorn” pattern, creating a hemangiopericytoma-like appearance. Variants of SFT with tumor cells in perivascular distribution may mimic MPC. Immunohistochemical staining displays that SFT tumor cells are positive for CD34, CD99, and STAT6 and negative for SMA [45]. All tumor cells of 30 MPC cases evaluated (29 from the literature plus our current case) showed consistent negative immunoreactivity for CD34, CD99, and STAT6. This characteristic immunophenotype provides a reliable diagnostic criterion for distinguishing MPC from SFT. MPCs share an overlapping histologic pattern spectrum with myofibromas, particularly the morphology that tumor cells may extend into the vascular lumen in some cases. Tumor cells in concentric perivascular arrangement could be detected in MPC more or less. In contrast, myofibromas exhibit a nodular or multinodular growth pattern, with well-defined cell-rich and cell-sparse regions and biphasic morphology, which are absent in MPC [46].

Although sarcoma is not usually taken as the first consideration generally in the diagnosis of MPC, some sarcomas manifest similarities to MPCs in both pathologic morphology and micro-RNA expression profile [47]. Medical professionals and pathologists must pay high attention when distinguishing MPCs from other types of sarcomas. MPCs can be distinguished from malignant sarcomas by the typical pattern of concentric perivascular whorls and tumor cells with no cellular atypia, no necrosis, lower ki67 index, as well as clear tumor boundaries.

Until now, the genetic landscape of MPC remains poorly characterized. Pericytoma with t(7;12)(p22;q13) translocation, which harbors an *ACTB-GLI* 1 fusion mutation, is considered a distinct subset of MPC. These tumors demonstrate indolent behavior and lack aggressive biological features. However, emerging evidence suggests that even these cytologically bland, nested mesenchymal neoplasms with *ACTB-GLI* 1 fusions may exhibit metastatic potential [48]. We propose the diagnostic term “*GLI* 1-altered mesenchymal tumor with malignant potential” for this entity. However, the pathogenetic and clinicopathological significance of t(7;12)(p22;q13) in MPCs requires further investigation. The optimal classification of these tumors as malignant pericytoma variants or novel sarcomas of uncertain origin remains undetermined. *BRAF V600E* mutations have been detected in 15% of MPCs, primarily located in the skin and thyroid. These mutations are associated with aggressive behavior. It was report-

ed that *BRAF* mutations represent a novel genetic aberration in MPC pathogenesis and could serve as a biomarker [49]. However, a subsequent study concluded that immunohistochemistry for *BRAF V600E*, which was highly concordant with *BRAF* mutation status, was negative in all tested MPCs. Therefore, it may not serve as a useful diagnostic immunohistochemistry marker for MPC [50]. Further studies with expanded cohorts and clearer molecular profiling are required to elucidate the role of *BRAF V600E* mutations in MPC pathogenesis. *PDGFRB* alterations are present in MPC, as well as in infantile myofibromatosis and other perivascular myoid neoplasms. These findings suggest that tyrosine kinase inhibition may be a therapeutic strategy for myopericytic neoplasms, particularly in the small minority of cases that fail conservative surgical management [51]. *NOTCH3* mutations occur in MPCs but lack specificity for tumor subclassification, as they also broadly distribute across the pericytic tumor spectrum [52]. Some MPCs with recurrent *SRF-RELA* gene fusions show dense cellularity and variable mitotic activity, but none exhibit nuclear pleomorphism or necrosis [53]. Ongoing molecular studies are necessary to identify the signaling pathways that are mutated or activated to provide targeted chemotherapy options in the future.

MPC primarily shows benign behavior with extremely rare malignant forms [47]. As well, all the oral MPCs we cited share a good prognosis (**Table 1**). A total of 28 patients followed postoperatively, ranging from 3 months to 108 months (mean duration: 34.18 ± 5.79 months). 24 MPCs showed no recurrence, and four patients had recurrence: one due to incomplete resection and three presenting as multifocal lesions (including one patient with HIV infection). The primary treatment for MPC is complete surgical excision, which has been found to have favorable clinical outcomes. Conservative treatment involving complete local surgical resection is recommended for MPCs in the maxillofacial region [23]. Superficial parotidectomy with facial nerve preservation is particularly recommended for MPC in the parotid gland [24]. MPCs commonly have benign biological behavior and can be surgically cured by complete tumor resection. However, a rare recurrence of MPC has been reported. The recurrence occurs mainly due to poor lesion boundaries or difficulty completely separating the tumor from adjacent tissues [19]. Patients with multiple lesions, abnormal sites, or a history of rapid growth should arouse high attention to the potential for malignant behavior. Consider ing the possibility of local recurrence or rare metastasis, strict long-term follow-up is essential. It may include further imaging, such as X-rays, to detect potential recurrence even after surgical removal.

In this study, we present a detailed analysis of MPC occurring at the base of the oral cavity. It exhibits a benign clinical course. The distinctive feature of blood vessels surrounded by oval to spindle-shaped tumor cells in the concentric perivascular arrangement is an important diagnostic criterion for MPC. Applying strict morphological criteria and appropriately selecting immunohistochemical markers can help distinguish oral MPC from its analogs. In conclusion, recognizing specific histological features combined with the characteristic immunophenotype of MPC can significantly enhance diagnostic accuracy, facilitating timely and effective treatment.

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Disclosure of conflict of interest

None.

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References

[1] Alqassab AT, Alsadah FZ, Elsharkawy T, Alhamad M and Alsayed H. Ankle myopericytoma: a rare case report and cytogenetic study. Cureus 2022; 14: e21307.

[2] Nigro MC, Pirini MG, Garelli E, Marchi M, Musto A, Pantaleo MA, Solli P, Ardizzoni A and Nannini M. Thoracic myopericytoma in an older adult, rare but possible: a case report. Thorac Cancer 2022; 13: 2532-2535.

[3] Kafle S, Karki S and Pant AD. Myopericytoma of the liver: a rare tumor - a case report. Int J Surg Case Rep 2025; 128: 111110.
[4] Schiavetti A, Pecorella I, Masselli G, Ceccanti S and Cozzi DA. Splenic myopericytoma/glomangiopericytoma in an adolescent male. Pediatr Blood Cancer 2022; 69: e29406.
[5] Taniguchi K, Yanai H, Honma R, Yabushita H, Yamasaki R and Ichimura K. A rare tumor of intravascular myopericytoma in the right renal vein. Int J Surg Pathol 2025; 33: 167-171.
[6] Ide F, Obara K, Yamada H, Mishima K and Saito I. Intravascular myopericytoma of the oral mucosa: a rare histologic variant in an uncommon location. Virchows Arch 2007; 450: 475-477.
[7] Datta V, Rawal YB, Mincer HH and Anderson MK. Myopericytoma of the oral cavity. Head Neck 2007; 29: 605-608.
[8] Laga AC, Tajirian AL, Islam MN, Bhattacharyya I, Cohen DM, Plamondon CJ and Robinson-Bostom L. Myopericytoma: report of two cases associated with trauma. J Cutan Pathol 2008; 35: 866-870.
[9] Lau PP, Wong OK, Lui PC, Cheung OY, Ho LC, Wong WC, To KF and Chan JK. Myopericytoma in patients with AIDS: a new class of Epstein-Barr virus-associated tumor. Am J Surg Pathol 2009; 33: 1666-1672.
[10] Sapelli S, Ribas M, Martins WD, de Noronha L and Gomes AP. Myopericytoma of the lip: report of case. Head Neck 2009; 31: 561-564.
[11] Chu ZG, Yu JQ, Yang ZG, Zhu ZY and Yuan HM. Myopericytoma involving the parotid gland as depicted on multidetector CT. Korean J Radiol 2009; 10: 398-401.
[12] Kuczkowski J, Rzepko R and Szurowska E. Myopericytoma of the parotid gland--a pathological conundrum. J Craniomaxillofac Surg 2010; 38: 595-596.
[13] Xia L, Chen Y, Geng N, Jiang J, Yang M and Zhang W. Multifocal myopericytoma in the maxillofacial region: a case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2010; 109: e59-62.
[14] Terada T. Myopericytoma of low grade malignancy in the oral cavity. Rare Tumors 2012; 4: e9.
[15] Jung YI, Chung YK and Chung S. Multiple myopericytoma of the face and parotid gland. Arch Plast Surg 2012; 39: 158-161.
[16] Akbulut S, Berk D, Demir MG and Kayahan S. Myopericytoma of the tongue: a case report. Acta Medica (Hradec Kralove) 2013; 56: 124-125.
[17] Vasenwala SM, Afroz N, Ansari HA, Khan AH, Basari R and Rehman S. Myopericytoma of lip: a rare lesion in an unusual location. Indian J Pathol Microbiol 2015; 58: 211-213.
[18] Rubino S, De Berardinis R, Colombo D and De Padova A. Myopericytoma of the tongue base: a case report. Acta Otorrinolaringol Esp (Engl Ed) 2018; 69: 304-305.
[19] Strayer E, Throndson R, Clement C and McDaniel K. Myopericytoma of the lip: a case report of this rare lesion. J Oral Maxillofac Surg 2018; 76: 1464-1467.
[20] Ju WT, Zhao TC, Liu Y, Dong MJ, Wang LZ, Li J, Tan YR and Zhong LP. Clinical and pathologic analysis of myopericytoma in the oral and maxillofacial region. Oral Surg Oral Med Oral Pathol Oral Radiol 2019; 128: 393-399.
[21] Ralli M, D'Aguanno V, De Vincentiis L, de Vincentiis M and Corsi A. Glomangiopericytoma-type glomus tumour/myopericytoma of the lip. Br J Oral Maxillofac Surg 2019; 57: 923-925.
[22] Almeida LKY, Jabur RO, Silveira HA, Polanco XBJ, da Silva RAB, Ribeiro-Silva A and León JE. Gingival ossifying myopericytoma in a pediatric patient: immunohistochemical analysis and literature review. Oral Oncol 2020; 107: 104826.
[23] Porat Ben Amy D, Yaffe V, Kawar R, Akrish S and Abu El-Naaj I. Oral myopericytoma: a rare pediatric case report and a review of the literature. BMC Oral Health 2021; 21: 176.
[24] Pan Y, Chen L, Shi D, Chen Y and Yu RS. Imaging features of myopericytoma arising from the parotid gland: report of 2 cases and literature review. Medicine (Baltimore) 2021; 100: e25471.
[25] Polat E, Atak İ, Özlü M and Paksoy CS. A rare tumor of the oral mucosa, myopericytoma: case report. European Annals of Dental Sciences 2022; 49: 92-95.
[26] Roig NJ, Wu M, Hernandez O, Liu CZ and Brandler TC. Myopericytoma of the parotid and molecular profiling: report of a rare case and review of the literature. Int J Surg Pathol 2022; 30: 574-580.
[27] Tan MWP, Wei Tay AY, Tang PY, Chew KY and Nicholas Yeo EM. Myopericytoma: a review of twenty-three cases over twelve years and a case report of a rare neoplasm. Am J Dermatopathol 2022; 44: 623-631.
[28] Dolcetti V, Ruggiero S, Pellini R, Mercurio A and Vidiri A. Myopericytoma: a rare case of parapharyngeal localization. J Radiol Case Rep 2023; 17: 8-12.
[29] Wei B, Liu G, Li K and Quan H. Pediatric upper lip myopericytoma: a case report and comprehensive review. BMC Oral Health 2024; 24: 478.
[30] Matsuzaki K, Mitsui H, Minami Y, Oishi N, Osada A, Oyama T, Kawamura T and Tsukamoto K. Case of cutaneous myopericytoma in a child and a mini-review of cases with children and adolescents. Int J Dermatol 2023; 62: e307-e309.

[31] Díaz-Flores L, Gutiérrez R, García MP, Díaz-Flores L Jr, Valladares F and Madrid JF. Ultrastructure of myopericytoma: a continuum of transitional phenotypes of myopericytes. Ultrastruct Pathol 2012; 36: 189-194.
[32] Mravic M, Asatrian G, Soo C, Lugassy C, Barnhill RL, Dry SM, Peault B and James AW. From pericytes to perivascular tumours: correlation between pathology, stem cell biology, and tissue engineering. Int Orthop 2014; 38: 1819-1824.
[33] Kumar MK, Panchal S, Dabral L and Prabhakar A. Myopericytoma involving proximal phalanx of index finger: masquerading as tenosynovial giant cell tumor - a case report. J Orthop Case Rep 2021; 11: 88-91.
[34] Hong MAC and Sih IMY. Epstein-Barr virus-associated intracranial myopericytoma in a child: case report and review of literature. Childs Nerv Syst 2022; 38: 649-654.
[35] Szymanski L, See K-Y, Gokoffski K, Barger M, Hu J, Blodget E, Wagle N, Patel V, Gill K and Lerner A. Treatment of multifocal central nervous system AIDS-related Epstein Barr virus-associated malignant myopericytoma with bevacizumab. Radiology of Infectious Diseases 2017; 4: 121-127.
[36] Herrtwich A, Sciot R and Ernst C. Myopericytoma in an infant-imaging characteristics of a rare but benign entity. J Belg Soc Radiol 2024; 108: 3.
[37] Awosusi BL, Attia OM and Nwadiokwu J. Myopericytoma of the right middle finger: a case report of an uncommon perivascular neoplasm. Cureus 2024; 16: e61938.
[38] Jang LC and Yoo KC. Spontaneous deep vein thrombosis of the upper arm due to an intravascular myopericytoma: a case report and literature review. Medicine (Baltimore) 2023; 102: e36566.
[39] Sato C, Takahashi O, Ogata S, Fujisaku M, Edo H, Sato K, Susa M and Matsukuma S. A case of intravascular myopericytoma: a painful subcutaneous tumor with an intraosseous lesion. Cureus 2024; 16: e72695.
[40] Kasa K, Shukuzawa K, Ozawa H, Chono Y, Iwauchi A, Kawachi K and Ohki T. Myopericytoma of the axillary artery in a 4-year-old girl presenting with stroke and subsequent progression of micro-isolated lesion. J Vasc Surg Cases Innov Tech 2024; 10: 101493.
[41] Mentzel T, Dei Tos AP, Sapi Z and Kutzner H. Myopericytoma of skin and soft tissues: clinicopathologic and immunohistochemical study of 54 cases. Am J Surg Pathol 2006; 30: 104-113.
[42] Yuan J, Li J, Dong Z, Xu W and Wang Z. Primary hepatic myopericytoma coexisting with multiple cystic hepatic lesions: a case report. World J Surg Oncol 2023; 21: 15.
[43] Eubanks BN, Hall JR, Hall KH, Buck SP and Wohltmann WE. Malignant myopericytoma: case report and review of the literature. J Cutan Pathol 2023; 50: 39-42.
[44] Bi WL, Gupta S, Mei Y, Abdulmohsen SA, Giantini Larsen A, Unadkat P, Ramkissoon S, Abedalthagafi M and Dunn IF. Immunophenotype of vestibular schwannomas. Otol Neurotol 2020; 41: e1290-e1296.
[45] Li XL, Zhang H, Hu CC, Zhou JF, Zhuang MY, Fan XX, Hu LW, Chen YP, Huang Q, Zhang S and Wang XF. Evolution of grading for solitary fibrous tumors of the central nervous system: a clinical pathological and prognostic analysis. Zhonghua Zhong Liu Za Zhi 2025; 47: 275-282.
[46] Souza LL, Fonseca FP, Cáceres CV, Soares CD, Gurgel AD, Rebelo Pontes HA, Corrêa Pontes FS, Paradela CA, Correia-Neto IJ, Kalinin Y, Lopes MA, Santos-Silva AR, Almeida OP, Vargas PA, Robinson L and van-Heerden WF. Head and neck myofibroma: a case series of 16 cases and literature review. Med Oral Patol Oral Cir Bucal 2024; 29: e734-e741.
[47] Zhang HL, Zhang M, Guo JQ, Wu FN, Zhu JD, Tu CY, Lv XL and Zhang K. Malignant myopericytoma originating from the colon: a case report. World J Gastrointest Surg 2024; 16: 1918-1925.
[48] Kerr DA, Cloutier JM, Margolis M, Mata DA, Rodrigues Simoes NJ, Faquin WC, Dias-Santagata D, Chopra S, Charville GW, Wangsiricharoen S, Lazar AJ, Wang WL, Rosenberg AE and Tse JY. GLI1-altered mesenchymal tumors with ACTB or PTCH1 fusion: a molecular and clinicopathologic analysis. Mod Pathol 2024; 37: 100386.
[49] Sadow PM, Priolo C, Nanni S, Karreth FA, Duquette M, Martinelli R, Husain A, Clohessy J, Kutzner H, Mentzel T, Carman CV, Farsetti A, Henske EP, Palescandolo E, Macconaill LE, Chung S, Fadda G, Lombardi CP, De Angelis AM, Durante O, Parker JA, Pontecorvi A, Dvorak HF, Fletcher C, Pandolfi PP, Lawler J and Nucera C. Role of BRAFV600E in the first preclinical model of multifocal infiltrating myopericytoma development and microenvironment. J Natl Cancer Inst 2014; 106: dju182.
[50] Mito JK and Jo VY. BRAF V600E is not a consistent feature of myopericytoma. J Cutan Pathol 2016; 43: 1248-1249.
[51] Hung YP and Fletcher CDM. Myopericytomatosis: clinicopathologic analysis of 11 cases with molecular identification of recurrent PDGFRB alterations in myopericytomatosis and myopericytoma. Am J Surg Pathol 2017; 41: 1034-1044.

[52] Iwamura R, Komatsu K, Kusano M, Kubo C, Inaba Y, Shiba E, Nawata A, Tajiri R, Matsuyama A, Matoba H, Koga K, Takeda M, Itami H and Hisaoka M. PDGFRB and NOTCH3 mutations are detectable in a wider range of pericytic tumors, including myopericytomas, angioleiomyomas, glomus tumors, and their combined tumors. Mod Pathol 2023; 36: 100070.

[53] Antonescu CR, Sung YS, Zhang L, Agaram NP and Fletcher CD. Recurrent SRF-RELA fusions define a novel subset of cellular myofibroma/myopericytoma: a potential diagnostic pitfall with sarcomas with myogenic differentiation. Am J Surg Pathol 2017; 41: 677-684.