J. Oral Diagn 2026;11:e412
https://doi.org/10.5327/2525-5711.412

CASE REPORT

Mandibular sarcomatoid carcinoma: a rare neoplasia with high aggressivity and diagnostic complexity

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Abstract:

Sarcomatoid carcinoma of the mandible is a rare and aggressive variant of squamous cell carcinoma characterized by biphasic epithelial and mesenchymal differentiation. The clinical and radiographic similarity to chronic inflammatory or infectious processes, such as peri-implantitis and osteomyelitis, can delay diagnosis. We report on the case of a 64-year-old patient with a painful mandibular lesion initially treated as peri-implantitis. Imaging studies revealed extensive bone destruction and soft tissue infiltration. Histopathological and immunohistochemical analysis confirmed the diagnosis of sarcomatoid carcinoma, with positivity for cytokeratins, p63, vimentin, and high Ki-67 index. Despite surgical resection with reconstruction, the patient experienced early recurrence and distant metastases, evolving to death. This case highlights the importance of accurate differential diagnosis, the contribution of imaging studies and immunohistochemical profile, and the poor prognosis associated with this neoplasm.

Keywords: Oral Squamous Cell Carcinoma; Spindle Cell Carcinoma; Immunohistochemistry.

INTRODUCTION

Sarcomatoid carcinoma (SC), also known as spindle cell squamous cell carcinoma, is understood as a rare and aggressive variant of squamous cell carcinoma (SCC), characterized by its biphasic morphology, with involvement of both epithelial and mesenchymal tissues[1,2]. This neoplastic subtype presents extensively invasive clinical behavior, being frequently associated with high rates of local recurrence and metastatic spread, contributing to a guarded prognosis. The prominent mesenchymal component often leads to diagnostic confusion with true sarcomas, highlighting its distinct and challenging nature compared to conventional SCC[2].

Although CS is most commonly documented in the urinary and upper respiratory tracts, it can also be found, extremely rarely, in the oral cavity, particularly in the mandibular region[1]. Due to its low incidence, there are few reports in the literature, making the investigation of its clinical, pathological, and radiological findings a challenge, especially concerning the differential diagnosis of other malignant lesions, such as osteosarcomas and soft tissue sarcomas[3].

Statement of Clinical Significance
This case underscores the need to consider rare malignancies in persistent peri-implant lesions, highlighting imaging and histopathology's role in early diagnosis and the importance of multidisciplinary evaluation to improve outcomes in aggressive oral cancers like sarcomatoid carcinoma.

The diagnosis of SC has been based on histopathological and immunohistochemical examinations, with the detection of epithelial markers, such as cytokeratins (AE1/AE3, OSCAR) and p63, as well as the expression of mesenchymal markers, such as vimentin, being essential[1]. These findings aid in the detection of definitive patterns for the differentiation of this neoplasm from other spindle cell neoplastic entities, such as true sarcomas and undifferentiated tumors[3].

The clinical management of SC generally involves wide surgical resection, with adjuvant radiotherapy and chemotherapy, depending on the stage and response to treatment[2]. However, even with advances, the patient survival rate remains lower than that of conventional SCC, reinforcing the need for early diagnosis and follow-up[1].

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Received on August 30, 2025. Accepted on December 10, 2025.
https://doi.org/10.5327/2525-5711.412

Given the rarity of sarcomatoid carcinoma of the jaw, this case report aims to describe the clinical presentation of this neoplasm, addressing the clinical, radiological and histopathological findings, in addition to the diagnostic and therapeutic difficulties involved.

CASE REPORT

A 64-year-old male patient with no previous comorbidities sought dental care due to an increase in volume in the left posterior region of the mandible, accompanied by pain and local ulceration. The patient reported no history of smoking or chronic alcoholism. Furthermore, the patient had been wearing a mandibular implant-supported fixed prosthesis (protocol) in the affected region for approximately one month. No family history of oral cancer or other relevant hereditary conditions was reported. He reported progressive evolution of symptoms in recent months, with difficulty in eating and episodes of local bleeding. During the first consultation with a professional in dental diagnosis, the clinical examination revealed an ulcerated exophytic lesion measuring approximately 4 cm, with irregular edges and induration. Palpation revealed a hardened consistency and the presence of palpable cervical lymph nodes at the left IB level.

The panoramic radiograph (Figure 1A) was the first examination analyzed, still showing the presence of implants in the affected region. Initially, the implantologist interpreted the condition as peri-implantitis associated with osteomyelitis, leading to the partial sectioning of the protocol prosthesis and removal of the implant in the affected area. This initial management occurred before the diagnostic confirmation of the neoplasia. After the lesion persisted, the implantologist referred to the patient for evaluation by a specialist in stomatology.

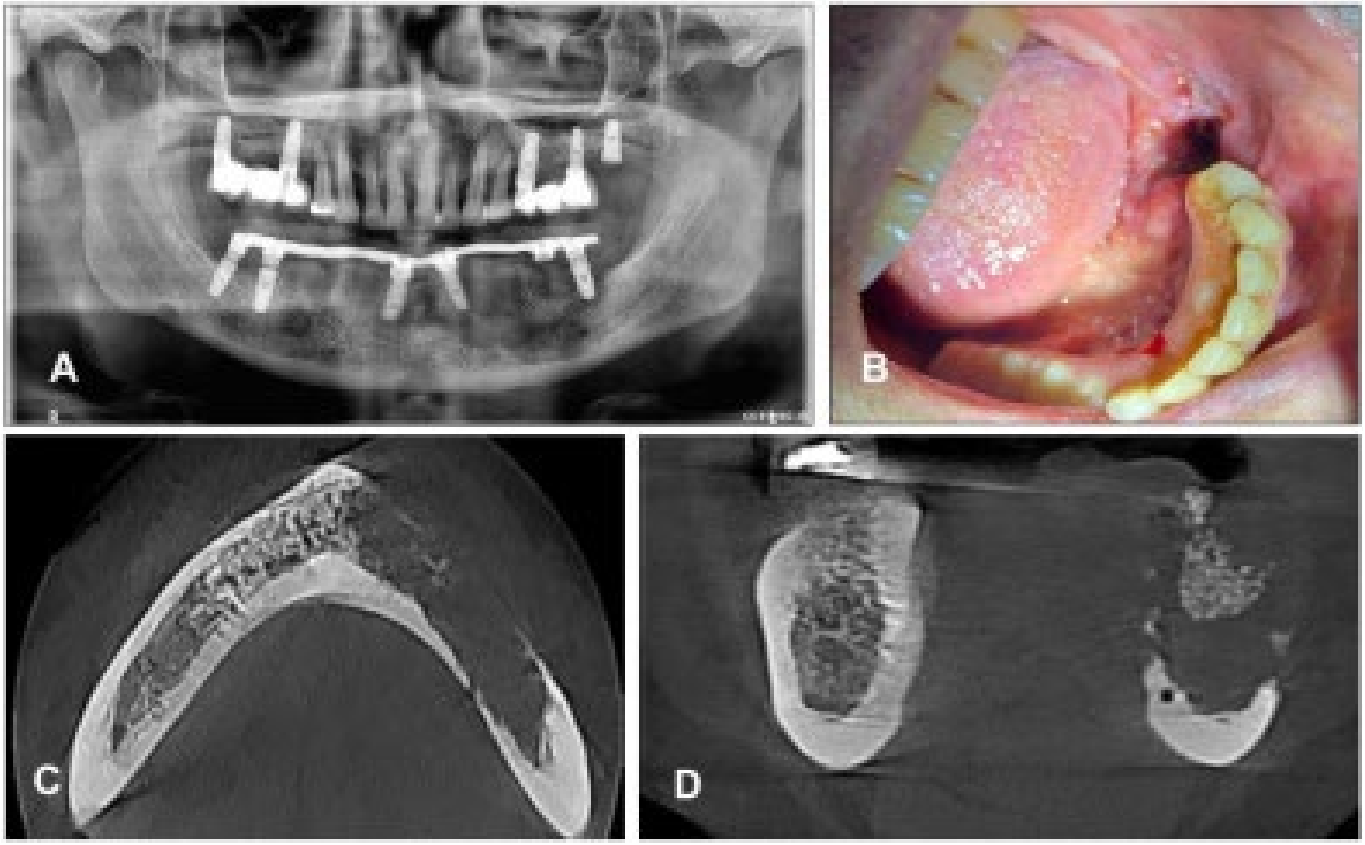

Figure 1. (A) Panoramic radiograph showing the presence of integrated bone implants in the mandibular region, with areas of bone rarefaction and bone resorption in the left posterior region. A radiolucent image with imprecise limits is observed, suggesting an osteolytic process. Initially, the condition was interpreted as peri-implantitis associated with osteomyelitis, leading to removal of the implant and conservative treatment. **(B)** Intraoral clinical view showing a lesion in the left posterior region of the mandible. **(C)** Axial computed tomography scan demonstrating significant bone destruction, with infiltration of the cortical plates (lingual and vestibular) and loss of medullary bone. **(D)** Coronal computed tomography scan illustrating severe vertical bone destruction, proximity to the inferior alveolar nerve canal, and poorly defined lesion margins.

During the initial dental consultation, a clinical examination revealed an exophytic ulcerated lesion, approximately 4 cm in size, with irregular borders and induration (Figure 1B). Palpation revealed firm consistency and the presence of palpable cervical lymph nodes at the left IB level. A cone beam computed tomography scan (Figure 1C, D) was performed, showing an extensive osteolytic lesion in the left posterior mandibular region, with vestibular and lingual cortical bone destruction and mandibular canal involvement. The radiographic report suggested an aggressive chronic inflammatory process or malignant neoplasm.

Due to the involvement of soft tissues and suspicion of malignancy, a magnetic resonance imaging examination with spin-echo and turbo multiplanar techniques was requested, before and after the injection of paramagnetic contrast, demonstrating an area of heterogeneity in the anterior contour of the left mandibular hemibody, where heterogeneous tissue involving the alveolar ridge was observed, with heterogeneous enhancement by the paramagnetic contrast medium, with an ulcerated, infiltrative appearance, which may represent a primary neoplastic lesion originating in the alveolar ridge. Anteriorly, it extends to the region of the lower left lateral incisor, and posteriorly, it extends to the topography of the lower molars on this side, and no adjacent dental elements are observed. Laterally, it causes bulging over the oral vestibule and buccal mucosa, and medially, it presents infiltration of the sublingual space and the floor of the mouth on the left, promoting compression over the belly of the genioglossus muscle. Inferiorly, it also presents extension to the left sublingual space with extensive areas of bone erosion. The lesion involves the left inferior alveolar nerve canal, which may be associated with perineural spread. It measures approximately 5.1 x 3.5 cm in the largest axes in the axial plane. There are associated increased lymph nodes in levels I B, but without characterization of well-defined lymph node enlargement. The left submandibular gland presents a reduction in its dimensions and a slightly heterogeneous signal, which may represent a certain degree of chronic process with involvement of the gland duct due to the presence of the lesion (Figure 2).

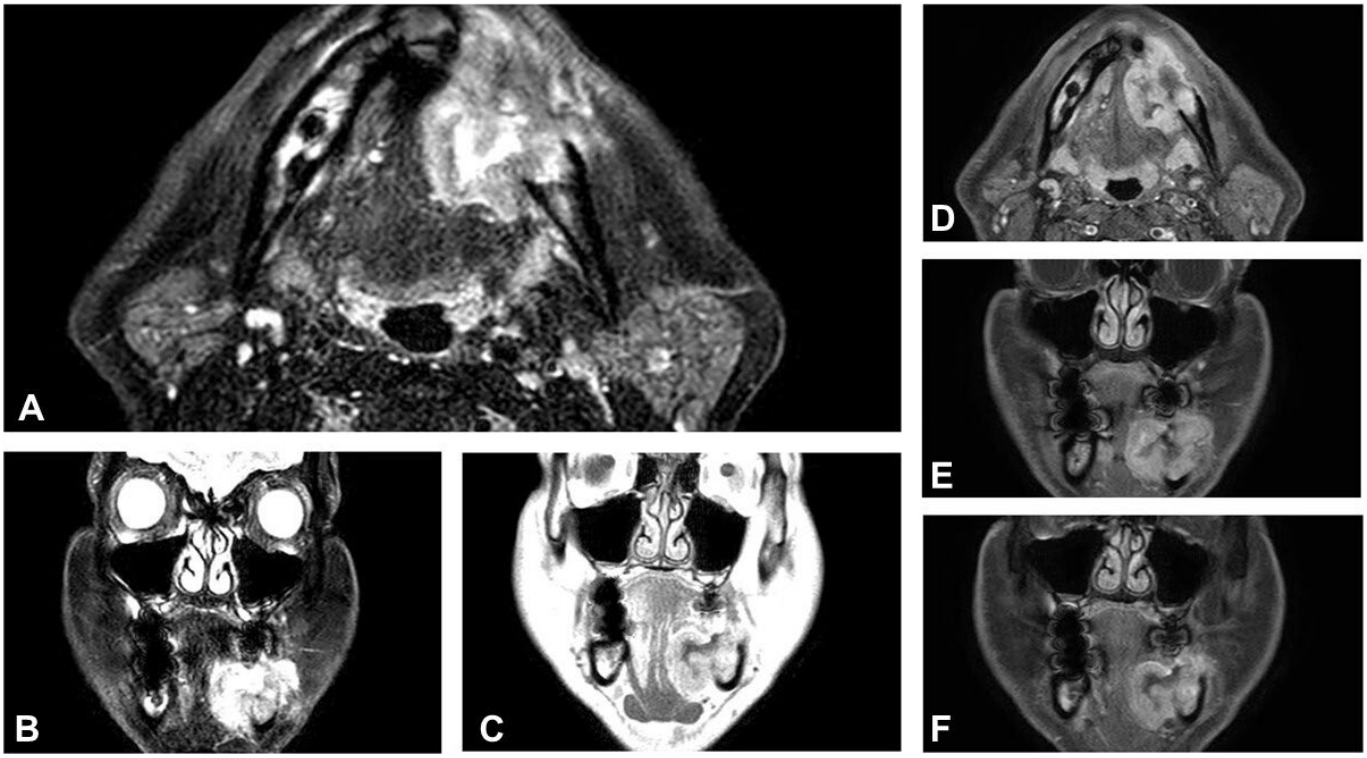

Figure 2. Magnetic resonance imaging demonstrating: **(A)** Infiltrative lesion in the left mandibular hemibody, with heterogeneous contrast enhancement and bone erosion. **(B)** Asymmetric involvement of the left side. **(C)** Heterogeneous enhancement of the left mandibular lesion and involvement of the inferior alveolar nerve canal, suggesting perineural spread. **(D)** Evident bone destruction on the left side and enhancement that reinforces the infiltrative character. **(E)** Deep infiltration of the sublingual space and adjacent tissues and asymmetry between the cervical compartments. **(F)** Infiltration of the sublingual space and enlarged lymph nodes at level I B are observed.

Given the clinical suspicion and complementary exams, an incisional biopsy of the lesion was performed. Histopathological examination revealed a fragment covered by squamous mucosa with atypia and subepithelial fibroconjunctival thickening. Fusiform cell proliferation with discrete atypia and a reactive appearance was observed (Figure 3).

Due to the need for diagnostic confirmation, an immunohistochemical study was performed (Table 1), which revealed positivity for cytokeratins (Oscar and AE1/AE3, focally positive) and p63, confirming the diagnosis of squamous cell carcinoma, spindle cell variant (sarcomatoid carcinoma).

After confirmation of the diagnosis, the patient was referred to a multidisciplinary team consisting of a head and neck surgeon, oncologist and radiation oncologist. The therapeutic approach established was left hemimandibulectomy with microsurgical reconstruction.

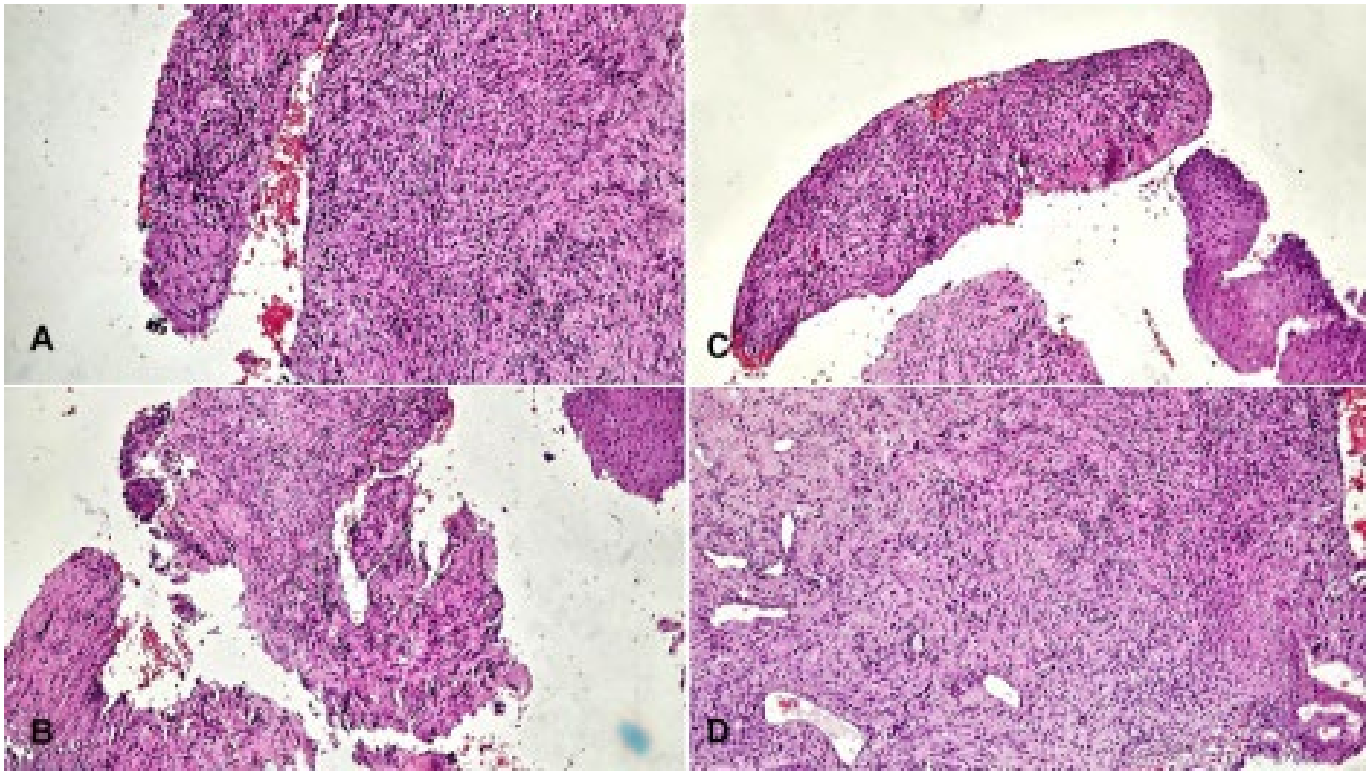

Figure 3. Microphotographs showing spindle cell/epithelioid proliferation with atypical cells with eosinophilic cytoplasm and pleomorphic nuclei, associated with atypical mitoses. An infiltrative growth pattern and architectural disarray of the epithelium are observed, with the presence of focal parakeratosis. **(A)** Hematoxylin and Eosin x10. **(B-C)** Hematoxylin and Eosin x4. **(D)** Hematoxylin and Eosin x40.

Table 1. Immunohistochemical profile of sarcomatoid carcinoma of the jaw. Positivity for cytokeratins and p63 confirms the epithelial component, while vimentin expression suggests mesenchymal differentiation. The high Ki-67 index indicates a high proliferative rate, characteristic of aggressive tumors.

Marker	Result	Interpretation
Cytokeratins (Oscar, AE1/AE3)	Positive (focal)	Confirms epithelial component of the tumor
p63	Positive	Indicates squamous differentiation
Vimentin	Positive	Suggests mesenchymal component, compatible with sarcomatoid
S-100	Negative	Excludes neoplasia of neural origin
Desmina	Negative	Excludes muscle differentiation
CD34	Negative	Absence of significant vascular component
Ki-67	High (>50%)	Indicates high proliferative rate, associated with tumor aggressiveness

The surgery was uneventful and the surgical specimen confirmed extensive tumor invasion. The patient evolved in the immediate postoperative period with good adaptation to the reconstructive flap and without initial surgical complications.

However, postoperative follow-up revealed worrying signs. The control MRI showed signs of surgical manipulation characterized by pelvic glossectomy and left hemimandibulectomy, with distortion of the local muscle-adipose planes, with an area of retraction and loss of substance, with a post-surgical appearance. In the depth of the surgical bed, in the transition with the remaining floor of the mouth and oral tongue, heterogeneous tissue was observed characterized by intermediate signal on T1 and hypersignal on T2, with heterogeneous uptake by the paramagnetic contrast medium and hypersignal on the diffusion-weighted sequence denoting high cellularity, with extension to the anterior segment of the transition between the root of the tongue and the remaining oral tongue, measuring approximately 4.1 x 4.3 cm in the largest axes in the axial plane, compatible with a persistent/recurrent lesion. A nodular image stands out in the left submandibular space, next to the submandibular gland, which may represent lymph node enlargement, measuring approximately 1.6 x 1.2 cm in the largest axes in the axial plane (Figure 4).

Based on these findings, a chest computed tomography scan was performed to screen for distant metastases. The examination revealed multiple irregular bilateral pulmonary nodules measuring up to 2.5 cm, highly suggestive of metastatic spread. A small left pleural effusion and thickening of the bronchial walls were also observed, suggesting concomitant inflammatory/infectious bronchopathy (Figure 5).

The patient underwent oncological palliative care due to the advanced stage of the disease. As the condition progressed, his respiratory condition worsened, requiring ventilatory support. In December 2024, he died due to respiratory complications associated with the neoplasm. The chronology of clinical events, initial management, diagnosis, and outcome is summarized in Figure 6.

This case illustrates the aggressiveness of sarcomatoid carcinoma of the oral cavity, its rapid progression, the high potential for recurrence and distant metastasis, and the diagnostic and therapeutic challenges associated with this rare entity. A multidisciplinary approach and early diagnosis are essential to improve the prognosis of patients with this condition.

DISCUSSION

Sarcomatoid carcinoma of the mandible is a rare and highly aggressive neoplastic condition characterized by its biphasic appearance, involving both epithelial and mesenchymal components, making it a major diagnostic and therapeutic challenge[1]. This subtype of squamous cell carcinoma has aggressive and highly invasive biological behavior, leading to a high recurrence rate and significant metastatic potential, especially to the lungs and cervical lymph nodes[2,3]. According to reports identified between 2014–2024 in PubMed (Table 2)[1-13] similar patterns of tumor aggressiveness are observed. In the present case, the patient presented a lesion initially diagnosed as peri-implantitis associated with osteomyelitis,

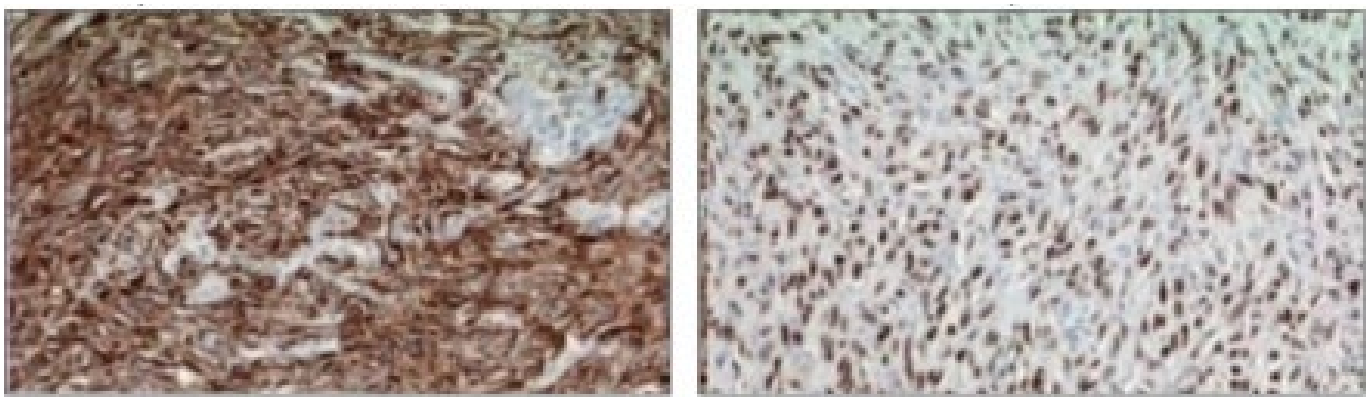

Figure 4. Postoperative magnetic resonance imaging showing persistent/recurrent lesion in the transition between the floor of the mouth and the remaining oral tongue (4.1 x 4.3 cm), with heterogeneous contrast uptake. Also noteworthy is a nodule in the left submandibular space (1.6 x 1.2 cm), suggestive of lymph node enlargement.

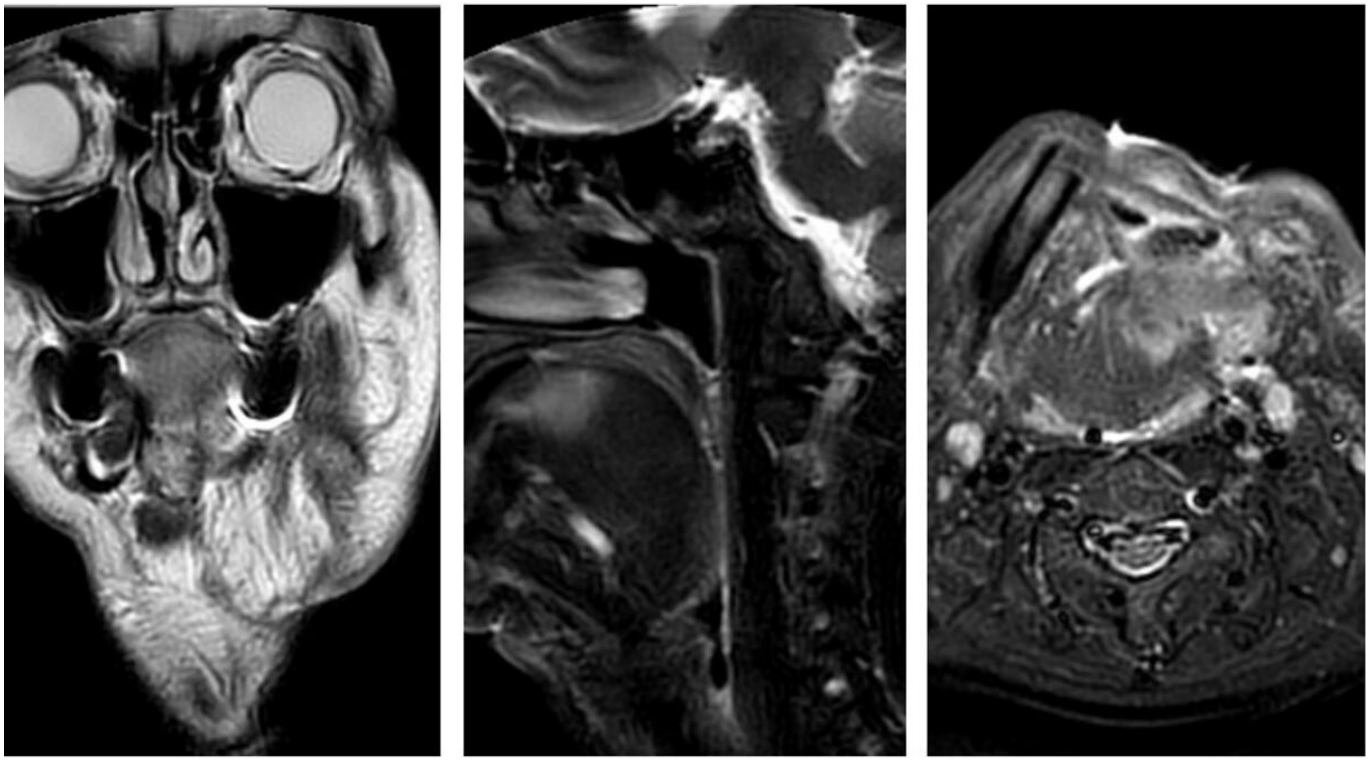

Figure 5. Chest CT scan showing: **(A)** Coronal reconstruction showing both lungs, with findings of multiple bilateral pulmonary nodules, with an irregular appearance, dispersed in both lung fields; Changes in the bronchial walls indicate inflammatory bronchopathy (thickening, irregularity). **(B)** Coronal section with density adjusted for contrast between soft and lung tissues, with findings of enhancement of the pleural silhouette and parenchyma with emphasis on nodular opacities; Possible pleural effusion on the left, subtle, more evident at this density. **(C)** Classic axial section of chest CT with lung window, with clear visualization of bilateral pulmonary nodules with metastatic characteristics (multiple, distributed bilaterally, without consolidation halo); Preservation of the parenchyma in some areas, with regions of bronchial thickening. **(D)** Three-dimensional reconstruction of the airways and lung fields, with visual reinforcement of the structural changes of the airways; Nodules distributed throughout both lungs, representing hematogenous dissemination typical of pulmonary metastases.

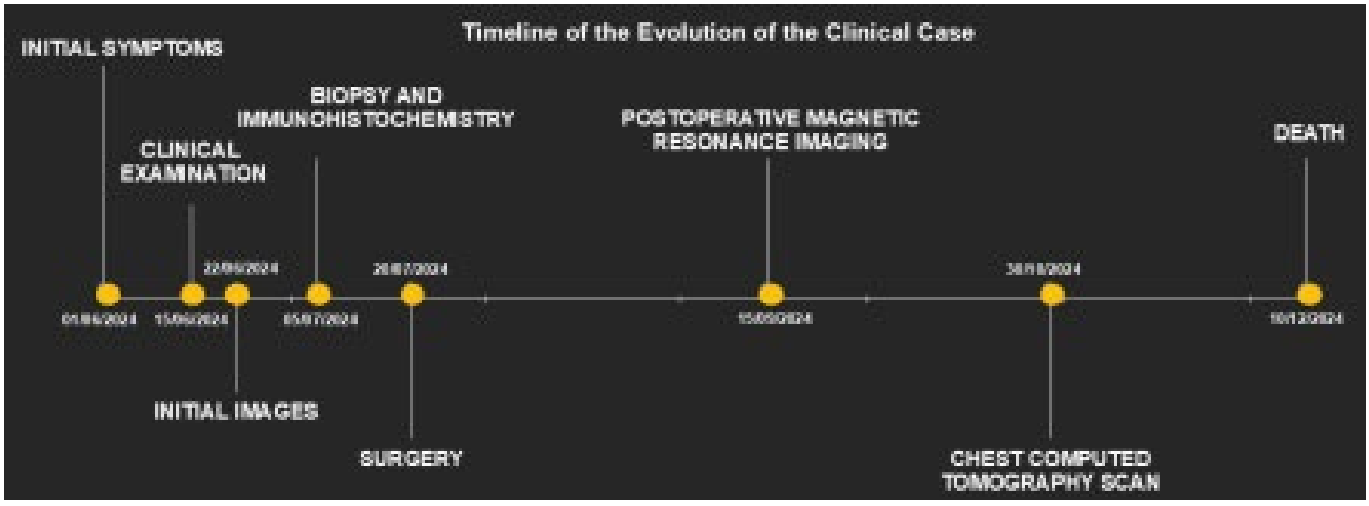

Figure 6. Timeline of the Clinical Case Evolution representing the main events in the diagnosis and treatment of sarcomatoid carcinoma of the mandible. It includes everything from the onset of symptoms to the final stage, highlighting examinations, biopsy, surgical treatment, and disease progression.

Table 2. Summary of clinical, immunohistochemical, therapeutic and outcome data for sarcomatoid carcinoma of the oral cavity, obtained from case reports and series published between 2014 and 2024. Survey carried out in the PubMed database.

Reference N (No. of cases)	Age/Sex	Lesion location	Immunohistochemistry markers	Treatment	Outcome
Shen et al.[1] Case report (N1)	72/M	Left mandible.	Vimentin, smooth muscle actin (SMA), S100, CK and p63.	Partial mandibulectomy.	Death within 8 months – extensive metastases.
Chaudhary et al.[3] Case report (N1)	60/M	Mandible.	Vimentin and CK.	No follow-up.	Unknown.
Reyes et al.[5] Case series (N2)	60/M	Tongue and left floor of mouth.	Cytokeratin AE1/AE3, vimentin and EMA.	Radical mandibular resection with lymphatic inclusion. Adjuvant treatment with radiotherapy and chemotherapy.	Metastasis after 5 months. Palliative radiotherapy was indicated.
	43/F	Left edge of the tongue and floor of the mouth.	Vimentin, cytokeratin AE1/AE3 and EMA.	Total glossectomy with radical lymphatic inclusion was performed. Postoperative chemotherapy.	Good condition after 2 months.
Mahajan et al.[9] Case report (N1)	51/M	Left mandible.	Pancytokeratin, vimentin and EMA.	Dissection of the supraomohyoid neck (levels IA, IB, IIA, IIB and III), excision of the submandibular gland and tail of the parotid gland.	Unknown.
Kim et al.[10] Case report (N1)	57/M	Left side of the floor of the mouth.	Pancytokeratin, EMA, vimentin and p63.	Resection of the floor of the mouth, excision of the submandibular gland and radical neck dissection. Adjuvant radiotherapy.	Metastasis after 6 months. Palliative chemotherapy.
Silva et al.[2] Case report (N1)	64/M	Left side of the tongue.	Vimentin, P53, alpha-smooth muscle actin (α-SMA), EMA and p63.	Patient refused proposed surgery/Treatment abandoned at an early stage.	Abandoned treatment at an early stage.
James et al.[8] Case report (N1)	55/M	Left side of the tongue.	Vimentin and cytokeratin.	Local radical excision with elective neck dissection.	Unknown.
Neha et al.[6] Case series N11	33 – 80 (Average age: 52)	The oral cavity was the most affected site (n=6), followed by the oropharynx (n=3) and larynx (n=2).	Vimentin (n=11/11), CK (n=5/11), EMA (n=1/11), p40 (n=3/11), p63 (n=4/11), SMA (n=3/11), S100 (n=5/11).	Retrospective survey.	1 patient alive (84 months). 4 patients – death (first year). 1 patient – death not from unknown cause (30 months). 1 patient – unrelated death (36 months). No follow-up information was available for the remaining four cases.
Seta et al.[11] Case report (N1)	83/F	Right mandible.	Cytokeratin (AE1/AE3) and vimentin.	Hemimandibular resection, including excision of adjacent muscles.	Good condition after 12 months.

Continue...

Table 2. Continuation.

Reference N (No. of cases)	Age/Sex	Lesion location	Immunohistochemistry markers	Treatment	Outcome
Franchi and Agaimy[7] Case series (N17)	57–80 (Average=69,1).	Larynx (n=12); Tongue (n=4);	p53 (n=13/17); p63 (n=8/17); AE1/AE3 (n=8/17); CK 5/6 (n=3/17); p40 (n=2/17).	Retrospective survey.	4 patients are alive, 4 had recurrences and the others have no record.
Ghosal et al.[12] Case report (N1)	62/M	Hypopharyngeal	p63 S-100, vimentin, PAN CK, p40 and Ki67.	Tumor excision with right segmental mandibulectomy and right supraomohyoid neck dissection with level I to IV lymph nodes.	Diffuse lymph node metastases. Patient in palliative care.
Mahapatra et al.[4] Case report (N1)	68/M	Right side of the jaw.	Vimentin, CD68, Ki67, CK AE1, AE3, CK 5 and CK6.	A localized excision with tumor-free margins of 1 cm was performed under general anesthesia.	There was no evidence of recurrence in the last 3 months.
Surolia et al.[13] Case report (N1)	15/F	Right side of the tongue.	Not described.	Patient received 4/20 cycles of curative neoadjuvant chemotherapy.	Diffuse lymph node metastases. Presence of extraoral lesion. Palliative care.

which delayed the definitive diagnosis of the neoplasm. This diagnostic delay is frequently described in the literature due to the clinical and radiological similarity of sarcomatoid carcinoma with other inflammatory and infectious bone pathologies[4,5].

This case illustrates the importance of differential diagnosis. With the increase in dental implant rehabilitation, oral lesions are often initially attributed to inflammatory conditions such as peri-implantitis[4,5]. However, clinicians must be alert to red flags that differentiate aggressive malignancies from chronic inflammation[6]. Specifically, in this case, the rapid progression of pain and swelling, combined with the extensive, poorly defined osteolytic bone destruction observed on cone-beam computed tomography (CBCT), should raise strong suspicion of malignancy, even in the presence of an implant. Any lesion around an implant that does not respond promptly to conventional anti-inflammatory or surgical therapy should require immediate biopsy[1,4,5]. This early step is crucial to avoid the fatal delay experienced by this patient, reinforcing the importance of a low threshold for diagnostic biopsy in atypical peri-implant lesions.

Imaging tests were essential for assessing the extent of the lesion. The initial panoramic radiograph revealed an extensive osteolytic area in the mandible, a finding that led to the suspicion of a chronic inflammatory process. However, cone beam computed tomography showed significant bone destruction without well-defined sclerotic margins, a pattern frequently observed in sarcomatoid carcinoma[6]. Postoperative magnetic resonance imaging demonstrated a recurrent lesion, with infiltration of the sublingual space and compression of the genioglossus muscle, in addition to possible perineural spread through the inferior alveolar nerve canal. These findings are consistent with previous reports, which highlight the capacity for tumor infiltration and involvement of adjacent tissues as markers of worse prognosis[14,15].

The definitive diagnosis was established by histopathological examination and immunohistochemistry. Positivity for cytokeratins AE1/AE3 and p63 confirmed the epithelial origin of the tumor, while vimentin expression indicated mesenchymal differentiation, a typical pattern of sarcomatoid carcinoma[16]. The high proliferative index detected by Ki-67 (>50%) suggests highly aggressive behavior, which is in agreement with the literature, which associates this characteristic with tumors with rapid progression and a higher risk of metastatic spread[17,18].

The management of sarcomatoid carcinoma usually involves wide surgical resection with free margins, complemented by radiotherapy and/or chemotherapy in advanced cases[19]. The patient in the present study underwent hemimandibulectomy with microsurgical

reconstruction, but presented early tumor recurrence and pulmonary metastases, factors that limited therapeutic options. This pattern of local recurrence and systemic dissemination has been described in several studies, reinforcing the need for rigorous follow-up and more effective therapeutic approaches for this neoplasm[7,20].

The prognosis of sarcomatoid carcinoma is poor, with reduced survival rates compared to conventional squamous cell carcinoma[21]. Rapid tumor progression and the presence of distant metastases at the time of diagnosis are factors that directly impact patient survival. In the case presented, the patient died due to respiratory complications associated with lung metastases, an outcome frequently reported in patients with advanced disease[8].

Given the aggressive nature of this neoplasm, it is essential that oral and maxillofacial health professionals and oncologists be alert to persistent oral lesions, especially in patients with implant history or other relevant risk factors for oral cancer. Early identification, accurate diagnosis and multimodal treatment are essential to try to improve the clinical outcomes of this highly challenging condition[9].

CONCLUSION

This case highlights the aggressiveness of sarcomatoid carcinoma of the mandible, a rare neoplasm that is difficult to manage clinically. The rapid progression, early recurrence, and extensive metastatic involvement reinforce the invasive nature of this pathology. The delay in initial diagnosis, due to its similarity to inflammatory bone diseases, highlights the importance of judicious use of imaging tests and a multidisciplinary approach for differential diagnosis. Wide surgical resection remains the main treatment, although the high rate of recurrence and distant dissemination demonstrates the need for more effective complementary therapeutic strategies. The patient's outcome, with fatal pulmonary progression, corroborates reports in the literature about the poor prognosis of this type of tumor. Therefore, early recognition and aggressive treatment are essential to improve the chances of survival of patients affected by this rare and highly lethal condition.

AUTHORS' CONTRIBUTIONS

AGAJ: Conceptualization, Investigation, Methodology, Visualization, Writing – original draft; Writing – review & editing. GPO: Formal analysis, Investigation, Methodology, Visualization, Writing – review & editing. ASBP: Conceptualization, Formal analysis, Investigation, Methodology, Visualization, Writing – review & editing.

CONFLICT OF INTEREST STATEMENT

Funding: The authors declare that no funds, grants, or other support were received during the preparation of this manuscript.

Competing interests: The authors have no relevant financial or non-financial interests to disclose.

Ethics approval: Written informed consent was obtained from the patient.

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