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Case Report

Dentistry Section

Extensive Intramedullary Spread of Oral Squamous Cell Carcinoma with Minimal Intraoral Manifestation: A Case Report

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ABSTRACT

Globally, Oral Squamous Cell Carcinoma (OSCC) is a significant public health problem, especially in developing countries, where delayed diagnosis often leads to advanced-stage presentation and poor outcomes. OSCC typically presents with visible intraoral changes (e.g., ulceration, swelling, mucosal alterations), but uncommon cases with extensive intramedullary spread and minimal intraoral manifestations pose specific diagnostic challenges. Hereby, the authors present a case report of a 31-year-old female with a 10-year history of tobacco chewing who presented with severe mandibular pain radiating to the right ear and mild discomfort during clenching activities. Clinical examination revealed a firm swelling on the right lower jaw with an intraoral ulcerative lesion over the gingivobuccal region. Imaging demonstrated extensive intramedullary spread, erosion of the mandible crossing the midline, pathological fracture, and perineural invasion. Histopathology confirmed OSCC. Despite minimal intraoral features, radiological studies highlighted the aggressive nature of the tumour, with neurovascular involvement and metastatic spread to the lungs. The tumour was deemed unresectable due to extensive involvement of vital structures, and the patient was started on primary chemotherapy with curative intent. The present case illustrates the misleading presentation of OSCC with only minor intraoral manifestations, underscoring the importance of advanced imaging for timely diagnosis, accurate staging, and treatment planning. It further emphasises the need for a high index of suspicion in patients with known risk factors and the integration of radiological assessment in atypical cases to improve diagnostic accuracy.

Keywords: Mandibular neoplasms, Neoplasm invasiveness, Neoplasm staging, Smokeless, Tobacco, Tumour

CASE REPORT

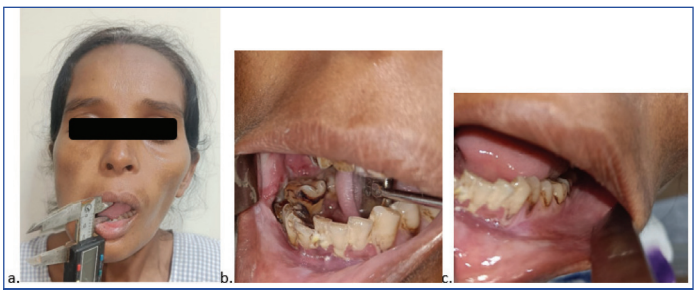
A 31-year-old female presented with mandibular pain radiating to the right ear for the past 45 days. She was a chronic chewer of kharra, a form of smokeless tobacco containing a mixture of tobacco, areca nut, and slaked lime, which she habitually placed in the right lower vestibule. She reported no other habits such as smoking or alcohol consumption. There were no associated systemic symptoms and no significant medical history. Family history of malignancy was negative. The patient noted a reduction in mouth opening, mild discomfort while chewing, and halitosis for approximately 15 days. Her past dental history was insignificant except for mild sensitivity in the lower right molar region, and she had not received any prior treatment.

On extraoral examination, a firm, diffuse swelling measuring approximately 3×2.5 cm was present on the right side of the lower jaw. The swelling, initially pea-sized, had gradually increased to the present size over one month. It extended superoinferiorly from the corner of the mouth to the inferior border of the mandible and anteroposteriorly from the corner of the mouth to 2 cm short of the angle of the mandible on the right side, with mild erythema and no fluctuation. A history of mandibular deviation towards the right side was also noted [Table/Fig-1a-c].

[Table/Fig-1]: Extraoral swelling: (a) Frontal profile; (b) Birds view profile; c) Right mid lateral profile.

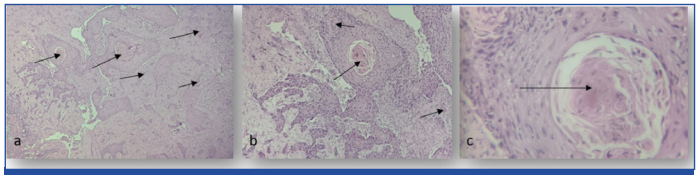
The patient had reduced mouth opening of approximately 20 mm interincisal distance [Table/Fig-2a], compared to the normal adult range of 35-55 mm, consistent with trismus. The restricted opening was likely due to involvement of the masticator space, temporalis, and masseter muscles by the tumour, as demonstrated on imaging. The overlying skin was intact without ulceration, and surface temperature over the swelling was not significantly raised. Marked tenderness was elicited over the right mandibular angle region.

Intraoral examination revealed a 1.5×2 cm ulcerative lesion with indurated margins in the vestibular depth extending from the first premolar to the second molar region of the right mandible. The surrounding mucosa appeared erythematous. Teeth in the affected region were mobile, suggestive of underlying bone involvement [Table/Fig-2b,c].



[Table/Fig-2]: (a) Reduced mouth opening of 20 mm; (b) Intraoral lesion on right side of lower gingivobuccal sulcus; (c) Left gingivobuccal sulcus showing no lesions.

An incisional biopsy was performed from the lower right gingivobuccal sulcus. Histopathological examination with Haematoxylin and Eosin (H&E) stain revealed well-differentiated squamous cell carcinoma characterised by nests of malignant epithelial cells with prominent keratin pearl formation and intercellular bridges [Table/Fig-3a-c]. Tumour cells demonstrated infiltration into the underlying bone with features of perineural invasion, indicating aggressive behaviour.



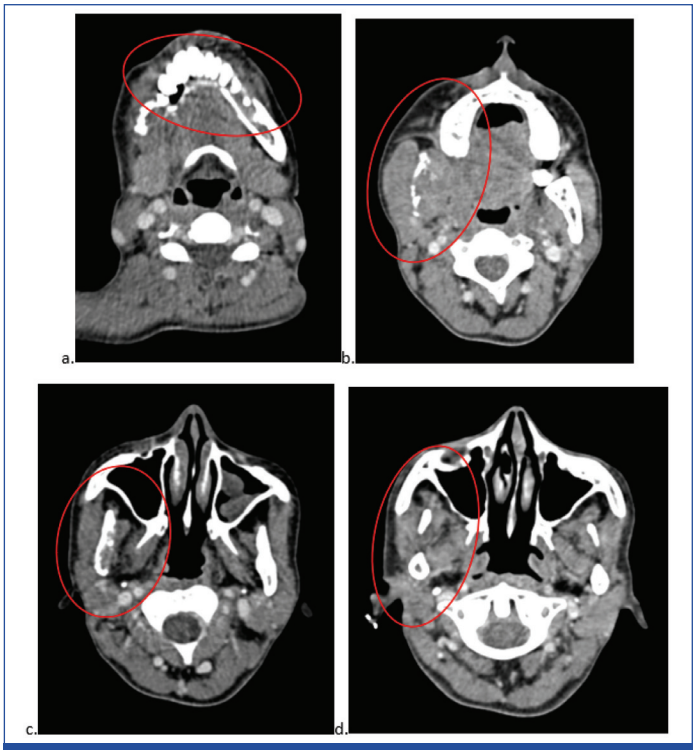
[Table/Fig-3]: Histopathological section of the intraoral lesion (Haematoxylin and Eosin stain: (a) 4x magnification; (b)10x magnification; (c) 40x magnification) showing invasive nests of squamous epithelial cells with keratin pearl formation (black arrow).

Based on the clinical presentation of mandibular swelling, pain, and intraoral ulceration, the differential diagnoses considered included chronic osteomyelitis, odontogenic infection, metastatic bone lesion, and intraosseous carcinoma. However, the history of kharra chewing and its habitual placement in the right lower vestibule, the progressive nature of the swelling, associated paraesthesia, mobility of teeth, and radiological evidence of cortical erosion and intramedullary spread favored a malignant process. A definitive diagnosis was established by incisional biopsy, which revealed well-differentiated squamous cell carcinoma.

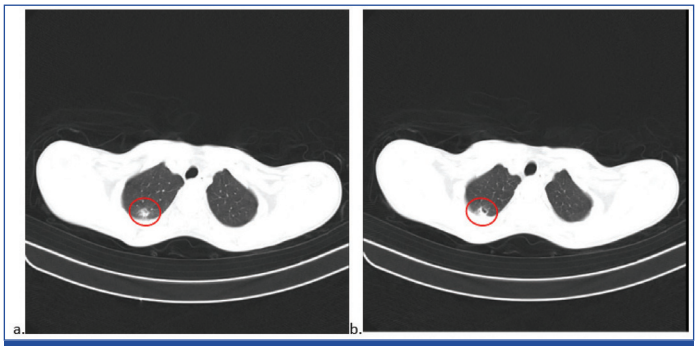
Contrast-enhanced Computed Tomography (CECT) of the buccal cavity and thorax was performed. CECT of the buccal cavity revealed a heterogeneously enhancing soft-tissue lesion measuring approximately 5.9×3×4.6 cm involving the right buccal mucosa and extending into the right buccinator muscle complex and inferior gingivobuccal sulcus. Anteriorly, the lesion reached the corner of the mouth, while posteriorly it involved the right retromolar trigone with erosion of the ramus of the right hemimandible. The mass also infiltrated the right masseter muscle and the inferior portion of the right temporalis muscle and was abutting the right medial pterygoid muscle with indistinct fat planes. There was loss of fat planes around the lesion and enlargement of the right submandibular gland. Notably, the superior gingivobuccal sulcus was spared. The lesion demonstrated erosion of the right hemimandible crossing the midline to the left, with soft-tissue extension along the mandibular canal indicating neurovascular involvement and a pathological fracture [Table/Fig-4a-d]. An enlarged right level II lymph node measuring 23×8 mm was identified, along with several subcentimeter lymph nodes at level I on the right and levels II and III on the left.

CECT of the thorax [Table/Fig-5a,b] showed multiple ground-glass nodules in the right upper lobe and in the superior basal and lateral segments of the left lower lobe. Additionally, two small soft-tissue density nodules were noted in the lateral segment of the right middle lobe and the anterior segment of the right lower lobe. A fibro-cavitatory lesion with adjacent consolidation patches was observed in the apical segment of the right upper lobe, measuring 10×10 mm, suggestive of metastatic disease. No additional investigations such as Positron Emission Tomography (PET-CT) were performed due to resource constraints. Laboratory investigations, including complete blood count and biochemical profile, were within normal limits.

The patient was deemed unresectable due to extensive mandibular involvement, the presence of perineural invasion, and radiological evidence of distant metastases. Based on these findings, Neoadjuvant Chemotherapy (NACT) with curative intent was advised. However, the requisite oncology services were not available at study Institution. Consequently, the patient was referred to a higher oncology centre for definitive management.



[Table/Fig-4]: CECT scan of buccal cavity: (a) Spread of lesion to the contralateral side; (b) Intramedullary spread of lesion with loss of medial cortical bone of right ramus of hemimandible at the retromolar trigone region; (c) Intramedullary spread of lesion with loss of medial cortical bone of right ramus of hemimandible involving the medial pterygoid muscle at the level of pterygoid plate; (d) Spread of lesion to the level of infratemporal fossa.



[Table/Fig-5]: CECT scan (a) and (b) showing fibrocavitatory lesion was seen with adjacent patches of consolidation noted in apical segment of right upper lobe measuring 10×10 mm which was suggestive of metastatic disease.

Unfortunately, despite appropriate referral, the patient was lost to follow-up.

The present case underscores the diagnostic dilemma posed by lesions with minimal intraoral presentation but extensive underlying spread. While the clinical appearance suggested a potentially resectable lesion, comprehensive diagnostic evaluation combining clinical examination, radiological imaging, and histopathological findings revealed the lesion to be aggressive and inoperable. This highlights the importance of thorough staging investigations, even in cases with deceptively mild clinical signs.

DISCUSSION

Oral cancer is more prevalent in developing countries due to risk factors such as tobacco use and limited access to early diagnostic tools, leading to delayed detection [1]. Globally, it accounts for 2% of all cancer cases and 1.9% of cancer-related deaths, with India alone contributing to one-third of the global oral cancer burden [2,3]. In India, oral cancer constitutes 30% of all cancer cases, with 80% diagnosed at advanced stages and higher mortality rates in men [1,4]. According to the Cancer Stat Facts data by the Surveillance, Epidemiology, and End Results Program (SEER) for cases between 2015 and 2021, the 5-year relative survival rate for all oral cavity cancers is 69.5% [5].

The OSCC has well-recognised clinical features; however, cases with minimal intraoral manifestations but extensive intramedullary spread, as observed in the patient, are rare and diagnostically challenging. This highlights the diverse and deceptive clinical spectrum of OSCC, particularly in individuals with known risk factors such as chewing tobacco, betel nut, kharra, smoking, and alcohol consumption [6]. The mechanism underlying extensive intramedullary spread in OSCC remains unclear but is thought to involve perineural invasion, hematogenous dissemination, or direct extension through vascular channels in the bone marrow. Perineural invasion, in particular, is recognised as a hallmark of aggressive OSCC and may explain early neural symptoms, such as paraesthesia and dysaesthesia, experienced by this patient. This pathophysiological pattern reflects the aggressive histological behaviour of OSCC, even in the absence of overt clinical findings [7].

Diagnosis and management of OSCC rely on radiological examination, which is used to assess the extent of the primary tumour, involvement of surrounding structures including regional lymph nodes and/or distant sites. Imaging modalities such as CT and Magnetic Resonance Imaging (MRI) provide details of bone erosion, soft tissue invasion, and neurovascular involvement, which are essential for staging and treatment planning. These images may be further enhanced with PET. However, CT scans have been reported to show higher false negatives in detecting focal invasion [8]. MRI is more reliable for detecting soft tissue extent and bone involvement, though it has a higher false-positive rate [8]. CT is the standard tool for detecting primary tumours and local bone infiltration [9].

Most OSCC lesions are poorly differentiated, making them relatively easier to distinguish from surrounding tissues [10]. The sensitivity of CT in detecting tumours ranges from 41-82% and specificity from 82-100%, while for bone infiltration, sensitivity is 63-80% and specificity is 81-100% [9]. Prognostic factors influencing recurrence and survival include tumour size, nodal involvement, bone invasion, tumour grade, and positive surgical margins. Radiological evaluation is also valuable in detecting pathological fractures and monitoring disease progression or recurrence during follow-up. In this case, imaging was essential in diagnosis and treatment planning, revealing mandibular erosion, neurovascular involvement, and a pathological fracture.

Three-dimensional radiographic imaging further enhances tumour assessment. While CT is optimal for detecting bone involvement, MRI is superior for assessing perineural spread and soft tissue contrast. PET-CT is particularly useful in detecting occult metastases. Imaging also aids in treatment planning, response monitoring, and differentiating residual disease from post-treatment changes.

Once the medullary space is invaded, the tumour progresses toward the least resistant areas within cancellous bone [10]. Intramedullary spread is more common in gingivobuccal OSCC of the dentate region compared to lower-lip SCC. This is due to site specificity: the dentate region contains abundant cancellous bone encased by thick cortical bone, allowing tumour spread without significant biological barriers [10]. In OSCC of the gingivobuccal sulcus involving the mandible, the Inferior Alveolar Canal (IAC) ceases to act as a barrier to vertical spread. Tumour cells can infiltrate downward through the cancellous bone, as the medullary space lacks a solid bony barrier. The presence of SCC in this area strongly suggests spread beyond the IAC, requiring consideration of deeper infiltration during treatment planning [11].

A tumour is considered “non resectable” when it extends into anatomic areas where it cannot be removed with adequate safety margins [12]. In this case, notable features included involvement of the right masseter and inferior temporalis muscle with abutment of the medial pterygoid, erosion and invasion of the right hemimandible, and tumour spread along the mandibular canal, suggesting perineural invasion and poor prognosis. These findings led to the classification of the tumour as non resectable, with primary chemotherapy planned with curative intent as the treatment of choice.

Similar cases of OSCC with minimal mucosal involvement but extensive intramedullary and neurovascular spread have been sparsely reported in the literature. Hong SX et al., discussed mandibular invasion patterns in gingival carcinoma, emphasising aggressive spread in the posterior mandible due to rich cancellous bone and limited anatomic barriers [11]. Trivedi NP reviewed cases involving the masticator space and classified them as T4b lesions, which carry an extremely poor prognosis and are often deemed inoperable [12].

Histopathologically, Well-differentiated Squamous Cell Carcinoma (WDSCC) is characterised by keratin pearls, intercellular bridges, and minimal cellular atypia. Although WDSCC is generally considered less aggressive than poorly differentiated variants, the presence of perineural invasion and deep bony infiltration, as seen in this case, are negative prognostic indicators. Literature suggests that perineural invasion is strongly associated with local recurrence and reduced survival, necessitating aggressive treatment and close follow-up [7].

The prognosis for this patient with extensive intramedullary spread of OSCC is generally poor due to delayed detection and the aggressiveness of the disease. However, advancements in imaging techniques and molecular diagnostics hold promise for improving early detection in atypical cases.

CONCLUSION(S)

The aggressive presentation of this case, with extensive intramedullary spread and perineural invasion, precluded surgical resection. Diagnosis and staging were achieved with advanced imaging, and neoadjuvant chemotherapy was chosen with curative intent. Early detection, especially in high-risk individuals, is critical to improving prognosis, as underscored by present case. With further developments in imaging and molecular diagnostics, earlier diagnosis and improved treatment outcomes may be achieved for such atypical and aggressive OSCC cases.

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