



## GENERAL DENTISTRY

# Gingival squamous cell carcinoma: an unexpected clinical presentation

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Squamous cell carcinoma (SCC) is an aggressive tumor and represents the most common oral malignancy found by dental health care providers. Timely detection is paramount to reduce patient comorbidities of regional and distant metastases and

improve survival rates. To augment recognition of early stage of gingival SCC (GSCC), this article features the somewhat innocuous clinical findings in a 60-year-old female.

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**Key words:** diagnosis, early detection, gingiva, squamous cell carcinoma

In 2012, the GLOBOCAN project estimated that at least 300,000 new cases of oral cancer (predominately squamous cell carcinoma [SCC]) arise annually, representing 2.1% of the worldwide cancers.<sup>1</sup> Common locations of oral SCC include the tongue (mostly lateral border), gingiva, floor of mouth, lips, and hard palate.<sup>2</sup> Use of tobacco products, alcohol consumption, and in certain geographic regions, betel nut chewing, are the chief oncogenic mediators in premalignant and oral SCC.<sup>3</sup> Infection with the human papillomavirus (HPV) has not been strongly implicated with the pathogenesis of oral SCC,<sup>4</sup> which is in contrast to the increased frequency of oropharyngeal carcinoma in HPV-positive younger-aged nonsmokers.<sup>5</sup> Oral SCC is associated with a 1.6:1 male predilection and the mean age of patients at diagnosis is 63 years old.<sup>6</sup>

Historically, SCC has been recognized for an aggressive pathobiology and diagnosed at a more advanced stage of disease. With the heightened advocacy for clinical surveillance, goals to achieve a more timely detection of oral SCC, and advances in therapeutic management options, the overall prognosis over the last 40 years has continued to improve. Specifically, the 3-year survival rate for early-staged disease has increased from 78.0% to 92.2%, and late-stage from 51.9% to 70.3%.<sup>7</sup> Late-staged oral SCC usually presents with more obvi-

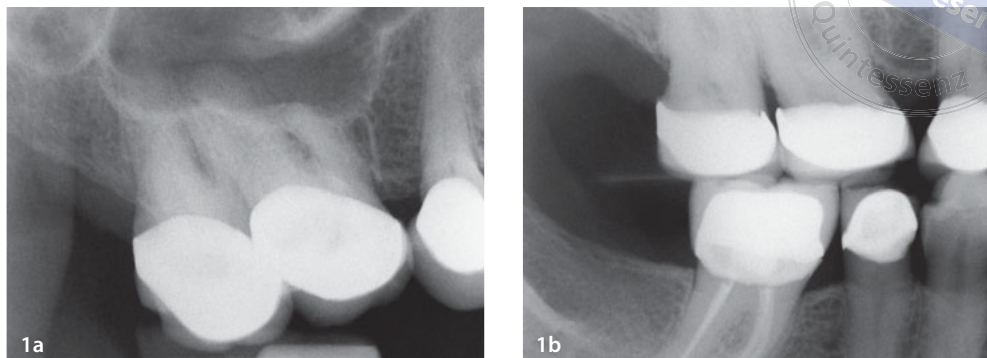
ous soft tissue alterations, such as a painful, often rapidly enlarging, indurated, exophytic mass and persistent deepened ulcerations with raised borders.<sup>8</sup> Avoidance of carcinogens and earlier detection of SCC offers the best chances to reduce patient mortality and a myriad of comorbidities.

The initial manifestations of gingival SCC (GSCC) may masquerade as plaque-induced periodontal disease or a traumatic lesion, prompting therapy that may ultimately prove ineffective, risking delay of the correct diagnosis, and institution of appropriate care. To increase awareness of the subtleties of the presentation of GSCC in its primordial stage of development, a clinical report is presented of a 60-year-old female who sought dental care for an abrupt episode of gingival bleeding.

### Case report

A 60-year-old female suddenly noticed “a lot of blood” that was “out of character” while flossing her teeth, localized to the maxillary right posterior region. Up to this point, the patient had flossed her teeth daily without evidence of bleeding. Within a week, the patient began experiencing soreness in this region and sought an oral evaluation. The attending dentist (SAS) described the gingiva as “irritated” and instructed the patient to

**Figs 1a and 1b** Absence of tumor invasion as seen on intraoral radiographs at initial presentation. (a) Periapical view of maxillary right posterior region. (b) Right bitewing.



swab the affected area two times per day with 0.12% chlorhexidine gluconate. Periapical and bitewing intraoral radiographs of the region were noncontributory (Fig 1).

The medical history was significant for right-sided stage II breast cancer with one positive lymph node, about 19 years ago, managed with lumpectomy, adriamycin/cytosin chemotherapeutics, radiation, and a 5-year regimen with the estrogen receptor modulator tamoxifen. She has continued to receive a yearly physical examination and mammogram, without disease recurrence. Other comorbidities were Hashimoto thyroiditis, arthritis, osteopenia, gastritis, cholecystectomy, ocular migraine, temporomandibular disorder; and allergies to cephalixin, amoxicillin, and mold. The remainder of the review of systems was negative. The patient denied use of tobacco products but admitted to drinking one glass of wine daily for the past 40 years. Current medications were levothyroxine, fish oil, glucosamine sulfate, cyanocobalamin, and probiotic and microcrystalline hydroxyapatite supplements. Upon review of the familial medical history, the patient disclosed that her mother had been diagnosed with estrogen-positive breast cancer at age 82 years.

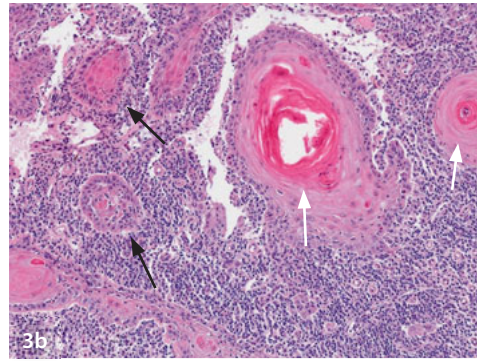
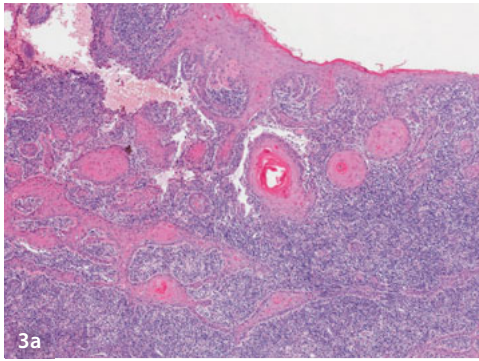
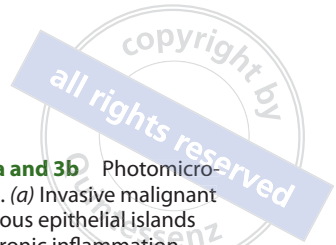
One week later, the pain increased to the extent that the patient avoided chewing on the right side and returned to the same practitioner, who commented that the gingiva had appeared “more irritated and red.” She was then referred to a periodontist (JWK) for further review. Clinical examination by the coauthor revealed moderately erythematous and ulcerated free-gingival margins, with somewhat loosened gingival cuffs, extending along the distopalatal aspect of the maxillary right second molar to the mesio-palatal aspect of the maxillary right first molar (Fig 2). Periodontal probing depths in the area were 2 to 3 mm, with the exception of a 4 mm pocket along the mesio-palatal of the maxillary right first molar, and the involved teeth were nonmobile. The remainder of the periodontal examination

was within normal limits. Paresthesia, swelling, and lymphadenopathy were not apparent. The tentative diagnosis was traumatic lesion and the patient was advised to rinse with “Magic Mouthwash” (Giant Pharmacy; compounded formulation, 2% viscous lidocaine, magnesium hydroxide, aluminum hydroxide, and diphenhydramine) as needed for discomfort and to return in 2 weeks for repeat assessment. With no clinical improvement at this follow-up, the patient requested an immediate biopsy, worried that she had recurrent breast cancer metastasis.

Following local anesthesia administration, an excisional biopsy of the palatal gingiva was performed, employing a 2-mm semi-lunar submarginal incision followed by a sulcular incision within the same line angles of the ulcerated lesion and extending to alveolar bone. Postoperatively, the patient was instructed to apply AO ProVantage gel (Periosciences) in the surgical site five times per day for the first week and three times a day for the second week.

**Fig 2** Clinical view. Palatal marginal gingival ulceration evident.





**Figs 3a and 3b** Photomicrographs. (a) Invasive malignant squamous epithelial islands and chronic inflammation (hematoxylin and eosin stain, original magnification  $\times 4$ ). (b) Islands of malignant epithelial cells (black arrows) and keratin pearl formations (white arrows) (hematoxylin and eosin stain, original magnification  $\times 10$ ).

The histopathologic diagnosis was moderately to well-differentiated SCC and the depth of the tumor invasion was at least 3 mm, extending to the margins of the surgical specimen. Low-power view showed stratified squamous epithelium with dysplastic changes, invasive malignant squamous epithelial islands, and marked chronic inflammation within the connective tissue stroma (Fig 3a). Higher-power view revealed pleomorphic hyperchromatic epithelial cells with an increased nuclear-cytoplasmic ratio, abnormal mitotic figures, keratin pearl formation (concentrically layered keratinized cells), and

admixed plasma cells and lymphocytes (Fig 3b). The patient was referred to an oral and maxillofacial surgeon (JEL) for consultation and additional surgery.

The subsequent oral assessment was remarkable for persistent linear ulcerations along the maxillary right posterior palatal gingiva. At this point, it was not clear whether the gingival alteration was due to residual carcinoma or a poor healing response after the biopsy. The remainder of the head and neck structures were within normal limits and cranial nerves II to XII were intact. The patient denied numbness, change in taste,



**Figs 4a to 4c** Cone beam computed tomography views shortly after excisional biopsy. (a) Axial view. Mildly diffuse reactive bone within the right molar region (arrow). Lack of tumor invasion in bone. (b) Sagittal view. (c) Volumetric.

**Fig 5** Obturator in place.

otalgia, dysphagia, or difficulty speaking. Cone beam computed tomography with contrast demonstrated a somewhat granular appearance of the cancellous bone, consistent with a reactive process, but without bony invasion or nodal involvement. Further imaging included computed tomography (CT) of the neck and facial bones with contrast and was negative for any suspicious cervical lymphadenopathy or bone invasion (Fig 4). She was classified as stage I SCC and designated T1N0Mx (T1, tumor  $\leq 2$  cm,  $\leq 5$  mm depth of invasion; N0, no regional lymph node metastasis; Mx, distant metastasis cannot be assessed).<sup>9</sup>

Treatment options discussed with the patient included surgical resection of the primary maxillary gingival cancer with immediate reconstruction versus definitive radiotherapy. Given the disease location within the posterior maxilla, preference was for a right partial maxillectomy. Elective lymph node neck dissection was not indicated based on tumor location, tumor size, and the negative findings on the CT scan.

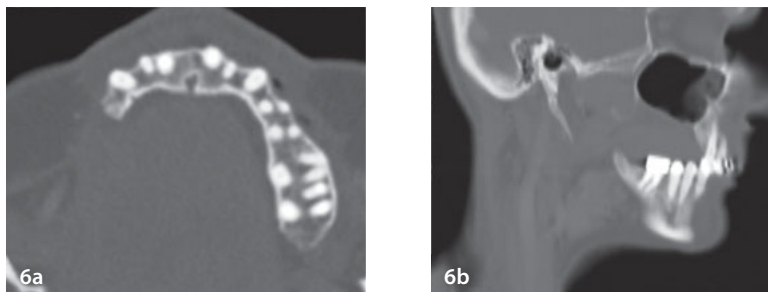
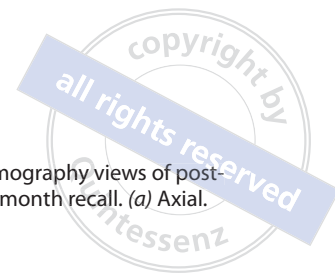
Perioperative laboratory studies were obtained and were only indicative for mild hypercholesterolemia. The patient underwent a right partial alveolar maxillectomy from the tuberosity to the distal aspect of the canine in an en bloc fashion, including the maxillary right second premolar and maxillary first and second molars, and incorporating a 1-cm oncologic soft tissue (buccal and palatal tissue) margin. Intraoperative frozen section margin analysis returned as negative for carcinoma or dysplasia. Primary closure was facilitated by a buccal fat pad graft and overlying buccal mucosal advancement flaps. No evidence of residual tumor was seen in the final surgical specimen. The patient was presented at the multidisciplinary head and neck tumor board with recommendation for close observation without adjuvant therapy. An interim obturator was fabricated to

mitigate the physiologic effects of the resultant surgical defect (Fig 5) and subsequently a conventional dental prosthesis was fabricated by a maxillofacial prosthodontist. At 4 months, the patient remains pain-free and without evidence of tumor as seen on clinical exam and CT imaging (Fig 6). The patient will continue to undergo routine tumor surveillance.

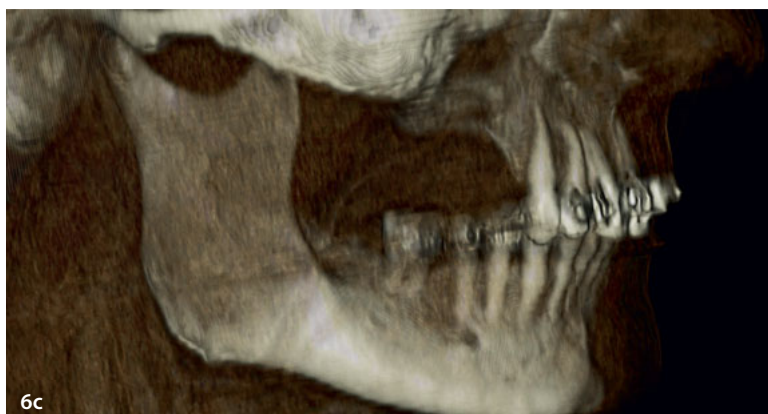
## Discussion

The stage of oral SCC during which the patient seeks preliminary medical attention varies geographically and culturally. An 11-year incidence study in the United States, involving 38,016 patients with oral cavity cancer, found 51.2% of cases were initially diagnosed with localized disease, 32.7% with regional spread, 6.8% were staged with distant disease, and 9.3% were unstaged.<sup>10</sup> Alternatively, in an 11-year retrospective investigation comprising 21,260 cohorts in Brazil with oral and oropharyngeal SCC, the percentages of affected patients at diagnosis progressively increased with each stage of disease, totaling 7.5% of patients at stage I, 14.2% at stage II, 22.2% at stage III, and 56.1% at stage IV.<sup>11</sup> Similarly, a 9-year study of 8,986 patients from Taiwan discerned 5.6% of patients presented with stage I disease, 11.8% at stage II, 19.0% at stage III, and 63.7% at stage IV.<sup>12</sup> The survival rates of SCC negatively correlate to the tumor-node-metastasis clinical staging system, with the percentage of patients reaching 5 years from initial diagnosis at stage I disease 72%, stage II 58%, stage III 45%, and stage IV 32%.<sup>13</sup>

The reasons for lateness in diagnosis of SCC are complex and not always definable. Often patients delay seeking medical attention because of fear (particularly cancer phobia); anxiety;



**Figs 6a to 6c** Computed tomography views of post-resection of right maxilla at 4-month recall. (a) Axial. (b) Sagittal. (c) Volumetric.



or lack of lesional awareness, clinical relevance, or symptomatology (pain, bleeding, swelling).<sup>14</sup> Although pain is typically a motivator to initiate assessment, 24% of GSCCs are painless, presumably leading some affected patients to defer care, and others, who simply may not be aware of the lesional presence, to not pursue evaluation altogether.<sup>15</sup> Moreover, financial constraints and cultural and religious beliefs may play a role in patient decisions to postpone therapy.<sup>16</sup> Once a patient has sought care, whether related to a chief complaint or for a routine appointment, the onus is on the attending dental clinician to perform a comprehensive oral cancer examination.

The incidence of gingival involvement of oral SCC has generally ranged from 2% to 27%.<sup>6,11</sup> Most series of cases have reported a preferential occurrence of mandibular GSCC,<sup>17,18</sup> although Lubek et al<sup>19</sup> found an almost equal proportion of gingival lesions in both arches. Another distinguishing characteristic of GSCC is its weak association with tobacco and alcohol consumption as compared to the heightened risk of these agents with SCC in the floor of the mouth and tongue.<sup>20</sup> Barasch et al<sup>21</sup> questioned whether GSCC should be reclassified as a separate lesion with an undefined pathoetiology. The featured patient's daily intake of one glass of wine was regarded as a negligible risk factor for the cancer.<sup>20</sup> Conflicting information has been avail-

able about GSCC and patient gender; a female predilection often has been recognized,<sup>18,19,21</sup> whereas some investigations occasionally have found a higher percentage of affected males.<sup>17</sup> The mean age of onset of GSCC is comparable to SCC of all oral sites, namely in the seventh decade of life. Nevertheless, clinicians should not be dismissive of the existence of GSCC in the pediatric population, as isolated cases have been documented in patients as young as 6 years old.<sup>22</sup>

Early stage GSCC may present as an insidious localized finding, appearing inflamed and associated with bleeding, pain or painless, and ulceration, easily mistaken for plaque-induced periodontal disease or a traumatic lesion.<sup>23,24</sup> Rarely, these lesions have been found synchronous with dental implants and mimic peri-implantitis.<sup>25</sup> Initial diagnostic procedures that should be undertaken include hard and soft tissue and lymph node examination, radiographic evaluation, and periodontal assessment, followed up with efforts to eliminate or mitigate preexisting conditions (local debridement, replacement of defective dental restorations, smoothing of any sharp-edged dental appliances). For palliation, antimicrobials (chlorhexidine gluconate, antibiotics), nonsteroidal anti-inflammatory agents, topical or systemic corticosteroids, and topical agents such as "Magic Mouthwash" should be considered.

However, the persistence of atypical alterations of gingival architecture in a patient with an otherwise healthy periodontium, without definable etiologies and coincident with lack of clinical improvement or resolution within 2 weeks following implementation of the aforementioned local measures, should raise suspicion of the possible manifestation of a systemic disorder and perhaps malignancy.<sup>26</sup> As such, these circumstances warrant a biopsy and submission of the excised tissue for histopathologic review. With disease progression, other clinical changes of GSCC may be discernible, including leukoplakia/erythroplakia/leukoerythroplakia, conspicuous exophytic growth, surface textural changes, nodularity, uncharacteristic tooth mobility or alveolar bone loss, regional lymph node spread, potential destruction of contiguous structures, and ultimately distant metastasis.<sup>27,28</sup>

The differential diagnosis of ulcerative gingival lesions that could mimic early stage SCC is diverse. The more common pathologies to consider are traumatic ulcer and attributed to toothbrush abuse (excessive force and frequency, hardness of bristles, use of worn toothbrush), poor-fitting dental prostheses, thermal burns, chemical burns (hydrogen peroxide, tooth whitening mouthwash, chemotherapeutic agents, topical application of aspirin and illicit drugs [cocaine, amphetamines], cinnamon-flavored foodstuffs and chewing gum, various dental products), iatrogenic procedures, factitial injury (picking, scratching), necrotizing ulcerative gingivostomatitis, herpetic gingivostomatitis, erosive lichen planus, pyogenic granuloma, and plasma cell gingivitis.<sup>29-31</sup> Rarer ulcerated gingival entities include chronic ulcerative stomatitis, crushed garlic burn, medication-induced (calcium channel-blockers, methotrexate, piroxicam), herpes zoster, systemic lupus erythematosus, epidermolysis bullosa, mucous membrane pemphigoid, pemphigus vulgaris, bullous pemphigoid, Crohn disease, sarcoidosis, tuberculosis, other malignant disorders, and possibly metastatic disease.<sup>32</sup>

Overall improved disease outcomes with oral SCC are fundamentally contingent on the timeliness of the clinical detection and diagnosis-to-treatment interval,<sup>33</sup> particularly with GSCC, as the typical thickness of attached gingiva is 2 to 3 mm and may facilitate rapid invasion to subjacent alveolar bone.<sup>34</sup> It is important to note that the degree of GSCC invasion in bone, as seen clinically and radiographically, is not always commensurate with the histopathologic findings, leading to disease overestimation in 22% of affected patients.<sup>19</sup> In contradistinction, Nomura et al<sup>35</sup> emphasized that “some” cases of mandibular GSCC micrometastasize to underlying bone without radiographic evidence of invasion.

Stage T1 GSCC and without lymph node spread, as seen in the featured report, is usually managed with surgical resection with wide margins, and it is advised that patients undergo post-operative evaluations every 2 months for the first 2 years, every 4 months for years 3 and 4, every 6 months for year 5, and annually thereafter. Elective cervical neck dissection and/or radiotherapy are usually reserved for stages T2 to T4 gingival tumors to reduce the propensity for regional recurrence,<sup>36</sup> particularly with cases displaying poorer degrees of cellular differentiation, perineural or vascular invasion, extracapsular spread, and diffuse infiltration.<sup>37</sup> There is emerging evidence that the combined use of CT, positron emission tomography, and single-photon emission CT as screening modalities for maxillary GSCC may improve the detection of occult lymph node spread.<sup>38</sup>

## Conclusion

Oral and maxillofacial SCC is regarded as an aggressive neoplasm. This article has reported an example of a rather nondescript appearance of GSCC and emphasizes the importance of both early discovery and the implementation of therapeutic modalities that may lead to improved clinical outcomes. The fortuitous and timely resection served to mitigate the chances of underlying bone invasion concomitant with regional and distant metastasis. Gingival alterations with atypical presentations, undefined etiologies, and those that fail to demonstrate improvement within 2 weeks following employment of local corrective measures should undergo biopsy and submission for histopathologic review to establish the lesional identity and rule out malignant disease. ■■

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