Summary

Relative to the incidence of all cancers, oral and oropharyngeal squamous cell carcinomas represent about 3% of the total. The treatment of intraoral squamous cell carcinoma is guided by the clinical stage of the disease and consists of wide surgical excision, radiation therapy, or a combination of surgery and radiation therapy. The prognosis for patients with oral squamous cell carcinoma depends on both histological subtype and clinical extent of the tumor. The overall 5-year survival rate for oral squamous cell carcinoma is around 45 to 50%. A 45-year-old male patient was referred to our clinic with a complaint of nonhealing and progressively enlarging ulcer on the right mandibular alveolar ridge. After the lesion had been diagnosed as squamous cell carcinoma with biopsy, it was treated with both surgery and radiation. There was no second primary lesion or a new, separate lesion in the five follow-up years.

Introduction

Squamous cell carcinoma (SCC) of the oral cavity accounts for 4% of malignancies in men and 2% of malignancies in women, and is responsible for 3% of all cancer deaths [1]. The men/women ratio is about 2 to 1, and is associated with tobacco and alcohol use [2, 3, 4, 5, 6]. The cause of oral squamous carcinoma is multifactorial, both extrinsic and intrinsic factors may be responsible. Extrinsic factors include such external agents as tobacco smoke, alcohol, syphilis, and sunlight. Intrinsic factors include systemic or generalized states, such as general malnutrition or iron-deficiency anemia. All forms of tobacco smoking have been strongly linked to the cause of oral cancer [7, 8, 9].

The most common site of intraoral carcinoma is the tongue and lip vermillion. Other sites of involvement, in descending order of frequency are the soft palate, gingiva, buccal mucosa, labial mucosa, and hard palate. The lesions of the buccal mucosa and gingiva each account for approximately 10% of oral squamous cell carcinomas [8, 9]. Gingival and alveolar ridge carcinomas usually are painless and most frequently arise from keratinized mucosa on a posterior mandibular site [3]. The clinical appearance varies from a white patch to a nonhealing ulcer or an exophytic lesion [9]. Mucosal changes, such as leukoplakia and erythroplasia, are frequent clinical findings [2, 4, 6]. The tumor has a special propensity to mimic the benign inflammatory changes of the gingiva [3, 9]. If tumor is adjacent to a tooth, it mimics periodontal disease and pyogenic granuloma, and causes tooth mobility. The cancer may become clinically evident after tooth extraction [3, 4]. When oral squamous cell carcinomas present their typical clinical form of chronic nonhealing ulcers, other ulcerative conditions such as tuberculosis, syphilis, and deep fungal infections should be considered. History of the patient is particularly important, and biopsy confirms the diagnosis [9].

Case report

A 45-year-old male patient was referred to our clinic with a complaint of nonhealing ulcer on the right alveolar ridge of the mandible. The patient stated that the nonhealing ulcer had been present for 7 months and enlarged progressively. He reported that his dental practitioner extracted
four mandibular teeth from the ulcerative mandibular region, due to mobility. The patient stated that the enlargement of the ulcerative lesion was more rapid after the extraction of the teeth. The lesion had been noticed about 7 months previously as a small gingival mass, 2 x 3 mm in diameter. In the patient’s medical history, a significant alcohol and smoking habits were present.

Intraoral examination revealed slightly elevated ulcerative gingival mass (15 x 7 cm), extending from the right second molar region to the left lateral incisor region (Figure 1). The adjacent left lateral incisor was mobile and the lesion region was edentulous. The ulcerative gingiva was prone to bleeding when palpated. The panoramic radiograph revealed a destruction of the alveolar bone (Figure 2). CT scans showed that bilateral cervical lymph nodes were enlarged (Figure 3) and there was no distant metastasis. The result of incisional biopsy revealed that the lesion was squamous cell carcinoma.

The surgical treatment included resection of the mandible from right angle of the mandible to the left second premolar region. Radical neck dissection on the ipsilateral side, and elective neck dissection on the contralateral side were performed. After resection, the mandible was reconstructed with the vascularized free fibula flap (Figure 4).

The multiple histopathological sections revealed that a mass containing atypically spinal layer cells and concentric keratin lamellae at the center was present (Figure 5). The dysplastic epithelial layer cells formed masses between fibrines and erythrocytes.

The 6000 cGy radiation therapy was performed postoperatively to the floor of the mouth and to both right and left neck region. When the

![Figure 1. Intraoral examination revealed slightly elevated ulcerative gingival mass (15 x 7 cm), extending from the right second molar region to the left lateral incisor region](image1.png)

![Figure 2. The panoramic radiograph revealed a destruction of the alveolar bone](image2.png)

![Figure 3. CT scans showed bilateral cervical lymph nodes were enlarged](image3.png)

![Figure 4. The panoramic radiograph demonstrated reconstruction of the mandible with the vascularized free fibula flap](image4.png)
radiation therapy finished, a partial removable denture was made to provide masticatory function to the patient. The postoperative course was uneventful (Figure 6). There was no recurrence of the primary lesion or distant separate lesion in the five follow-up years.

Discussion

The metastatic spread of oral SCC is largely through the lymphatics to the ipsilateral cervical lymph nodes [8]. Occasionally, contralateral or bilateral metastatic deposits are seen, and approximately 2% of patients have distant metastases at diagnosis. Tumor stage, size, margin status, and metastatic spread of oral squamous cell carcinoma have been used to guide treatment and indicate the patient's prognosis. Quantifying of these clinical parameters is called "staging" of the disease. The most popular staging protocol is the tumor-node-metastasis (TNM) system: T is a measure of the primary tumor size. N represents local lymph node metastasis, M is a determination of distant metastasis. Use of this system allows more meaningful comparison of data from different institutions and helps guiding the therapeutic decisions [6, 8, 9].

SCC arises from dysplastic surface epithelium and is characterized histopathologically by invasive islands and cords of malignant epithelial cells, which show differentiation toward a squamous morphology. The grading of a lesion is the microscopic determination of the tumour cell differentiation. Lesions are graded on a three-point (grades I to III) or a four-point (grades I to IV) scale. The poorly differentiated tumors receive higher numerals. Well-differentiated lesions generally have a less aggressive biologic course than less-differentiated lesions. Most oral SCCs are moderately or well-differentiated lesions. Keratin pearls and individual cell keratinization are usually evident [8, 9].

Six percent of patients with SCC of the oral cavity present early primary tumour (T1 or T2) and a clinically negative neck [1, 3, 10]. Management of the neck in these patients is controversial. In general, treatment for stage I and II lesions has been either surgery or radiation therapy as a backup in the evidence of recurrence as a single modality. Larger lesions with palpable lymph node require surgical excision with postoperative radiation lective neck dissection for stage I and II cancers are controversial. Therapeutic neck dissection has been performed in patients with clinically palpable lymphadenopathy [1, 2].

In the present case, because the lesion exceeded T2 stage, combined surgery and radiation therapy as well as radical neck dissection on the ipsilateral side, and elective neck dissection on the contralateral side were performed. Factors that determine which treatment is to be used include lesion location, histological type, institution facilities and philosophy, referral patterns, and therapist skills [8, 9]. Oral SCC are generally resistant to chemotherapeutic measures. They are used as adjunctive therapy in advanced cases. Radiotherapy is more effective on less well-differentiated lesions. The radiation level needed to kill malignant cells ranges from 40 to 70 Gy (daily dose 2 Gy) [9].
A number of factors play a determining role in the prognosis of gingival carcinomas: size and site of the lesion, the nature of bony involvement, presence or absence of metastasis, and stage of the disease. The most important indicator of prognosis is the clinical stage of the disease [2, 3, 8-11], The 5-year disease free survival rate for intraoral carcinoma is 76% if metastasis has not occurred by the time of diagnosis (stage I and II) [3, 6], 41% when the cervical nodes are involved (stage III) and only 9% when metastasis below the clavicle is present (stage IV).

Current improvement of survival rates lies in early detection, an area in which dental practitioners must have a primary role. The oral cavity is readily accessible for examination and biopsy, making early diagnosis a realistic and achievable goal in oral cancer control [3, 8]. In the present case, due to the risk of recurrence, the patient was followed up periodically. There was no recurrence of the primary lesion or distant separate lesion in the five follow-up years. The patient is still under our control.

References


Correspondence to: Dr. Hatice Altundal, Yeditepe University, Faculty of Dentistry. Bagdat Caddesi, No. 238, 34728-Goztepe, Istanbul, Turkey. E-mail: haticealtundal@yahoo.com