HISTOLOGICAL ASPECTS IN LIP CANCER

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Summary

Major risk factor for lip cancer is smoking and alcohol consumption. The aim of the study was to reveal histopathological particularity in lip cancer correlated with smoking. Were studied medical records of patients admitted in Maxillo-Facial Clinic, Timisoara, between years 2003-2008, and were noted total number of patients and the number of patients with oral tumors, respectively with lip tumor. From the histological results were noted some aspects regarding keratinisation, differentiation degree, ulceration, the type of cellular infiltrate, tumor invasion, lymph node infiltration and tumor stadialization. Were studied 1013 patients, 744 males (73.44%) and 269 females (26.56%), aged 24 and 85 years, with oral tumor surgically removed and histological diagnosed. Were selected 213 (30.86%) patients smokers and nonsmokers, with diagnostic squamous-cellular carcinoma (SCC), localized on lip. Keratinisation frequency in lip SCC was 85% in smokers and non smokers. From investigated cases the most frequent differentiation degree was G2, independent of smoking, which reveals that smoking doesn’t influence histological differentiation. Tumor invasion was present in ¼ of patients. Cellular inflammatory infiltrate was present in ⅓ of cases, with small differences depending of smoking. Most patients with lip tumor report to physician in stage I and II. Lip cancer incidence was increasing and was correlated with risk factor smoking. Were found increased frequency of keratinisation, cellular inflammatory infiltrate and invasion tendency in nearby tissues. Smoking cessation may decrease the risk of lip tumor formation.

Key words: lip cancer, squamouscellular carcinoma, histology, smoking

Introduction

The major risk factor for lip cancer is smoking and alcohol consumption.

Because smoking and alcohol consumption are correlated is difficult to quantify the combined effect of those factors (American Cancer Society, 2007). Based on a large population study was reported that smoking and drinking patients have 38 times greater risk for cancer development then abstinent (Ide et al., 2008; Kawado et al., 2005). Because of that it seems that both factors combined have a synergetic effect in lip cancer etiology.

The aim of our study was to correlate lip cancer with age, histopathological particularity, and risk factors.

Materials and method

Were studied medical records of patients admitted in Maxillo-Facial Clinic, Timisoara, Romania between years 2003-2008, and were noted total number of patients and the number of patients with oral tumors, respectively with lip tumor.

From the histological results were noted some aspects regarding keratinisation, differentiation degree, ulceration, the type of cellular infiltrate, tumor invasion, lymph node infiltration and tumor stadialisation.

Were calculated the frequency of each aspects reported to total cases. The results are shown in tables and charts.

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Results

Were studied 1013 patients, 744 males (73.44%) and 269 females (26.56%), aged 24 and 85 years, with oral tumor surgically removed and histological diagnosed.

Were selected 213 (30.86%) patients smokers over 20 cigarettes per day, and nonsmokers, with diagnostic squamous-cellular carcinoma (SCC), localized on lip, 1/3 of the selected patients recognize alcohol consumption.

Was noted the increased frequency of keratinisation in non smoking patients (92.95%) then in smoking patients (78.94%) from all patients with lip tumor.

Differentiation degree of patients with lip tumor is approximately G2 in 60% of cases, similar in non smokers (61.54%) and smokers (68.41%). Was noted increased frequency of G1 and G3 degree (21.43%) (Table I).

Ulceration was present in 42.86% of non smoking patients and in 42.10% of smoking patients.

Table I. Differentiation degree

<table>
<thead>
<tr>
<th>SCC - Differentiation</th>
<th>Non smokers</th>
<th>Smokers</th>
</tr>
</thead>
<tbody>
<tr>
<td>G0</td>
<td>0%</td>
<td>10.53%</td>
</tr>
<tr>
<td>G1</td>
<td>21.43%</td>
<td>10.53%</td>
</tr>
<tr>
<td>G2</td>
<td>61.54%</td>
<td>68.41%</td>
</tr>
<tr>
<td>G3</td>
<td>21.43%</td>
<td>10.53%</td>
</tr>
</tbody>
</table>

Inflammatory infiltrate was found in 28.57% in non smoking patients, and in 42.10% of smoking patients. Smoking can increase the inflammation in tumor area.

Tumour invasion in adjacent tissues was 28.57% in nonsmokers, and 26.31% in smokers (table II). The invasion was more frequent in muscular tissue near tumor.

Table II. Inflammatory infiltrate and tumor invasion frequency in patients with lip SCC

<table>
<thead>
<tr>
<th>SCC – inflammatory infiltrate</th>
<th>Non smokers</th>
<th>Smokers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lip SCC</td>
<td>28.57%</td>
<td>42.10%</td>
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<table>
<thead>
<tr>
<th>SCC – tumor invasion</th>
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The patients with SCC of lip report to doctor in proportion of 37.71% of cases in nonsmokers, respectively in 31.58% of smokers in stage I and II of the disease (fig. 1). Probably, the presence of lip tumor in a visible place, with esthetic inconvenience, determined the patients to report to hospital in early stages of tumor development.

![Fig.1. Stage of lip SCC](image-url)
Case report

Patient RM, male, 61 years old.

Clinical Diagnostic: Upper lip tumor.

Macrosopically examination:
Skin fragment with subcuatanat tissue about 2.4/2 cm, with hair follicles. On para central epidermis surface presents a round lesion, well delimited 1.2 cm, elevated, with irregular margins, and with ulcerate center. On section surface presents a well delimited white lesion.

Histopathological diagnostic:
Scuamocellular carcinoma with vague keratinisation tendencies, poor differentiated, G3, ulcerates and inflamed. Extensive tumoral necrotic areas, carcinomatous invasion in deep muscular tissue (fig.2, 3). Malignant cells presented frequent atypical mitosis (fig. 4). Presents surgical safety excision limits in surface and deepness.

Fig. 2. Lip scuamocellular carcinoma poor differentiated G3 with central necrosis areas.

Fig. 3. Infiltrate tumor lobes formed with poor differentiated scuamos cells. Tumor necrosis areas.
Discussions

Until these date, most of the studies about the impact of smoking and alcohol consumption on lip cancer were based on a case control structure. As expected, smoking was correlated with cancer without the age factor, but there are not relevant as risk associated with the number of cigarettes smoked. According with Castellsague study, our results proved a higher incidence of lip SCC associated with smoking over 20 cigarettes per day and alcohol consumption.

Studies were made about the association of smoking and alcohol consumption and oral cancer in many areas around the world (Castellsague et al., 2004). Relative risk resulted from cohort studies on smoking men variants form 2.9 in Japan to 13 in England. Relative risk of cancer in former smokers seems to be decreased, suggesting the fact that after smoking cessation the risk decreases. It is considered that the patient is ex smoker after minimum one year after smoking cessation. IARC confirmed that the risk increase with the number of cigarette smoked (IARC, 2002).

From our results, 1/3 of patients admitted alcohol consumption. Alcohol is not carcinogenic at lab rats, and the mechanisms of carcinogenesis are unclear. Anyway, regarding histological and cytological studies on oral mucosa, alcohol may influence cell proliferation by intracellular and extra cellular pathways. A meta analyses showed that exists a clear risk gradient between the quantity of alcohol and cancer proliferation (relative risk of 1.75, 2.85, 6.01 for 25g, 50g, and 100g alcohol dose per day) (Bagnardi et al., 2001).

Based on estimated risk of alcohol consumption and smoking, was calculated that 75% of malignant lesions of lip could be prevented (Walker et al., 2003).

Conclusions

Lip cancer incidence was increasing and was correlated with risk factor smoking.

In order to quantify some histological and morphological aspects of scuamo- cellular carcinoma of lip, were calculated keratinisation, differentiation degree, ulceration, type of cellular
infiltrate, tumor invasion in tissues, lymph node affection, tumor stage.

There were found increased frequency of keratinisation (85%), cellular inflammatory infiltrate and invasion tendency in nearby tissues. From investigated cases was mostly found G2 differentiation degree independent of smoking. These means that smoking doesn’t influence the histological degree of differentiation.

Tumor invasion in nearby tissues was found in ¼ of patients. Cellular infiltrate was present in 1/3 of patients with lip SCC, with small differences dependent of smoking. Most of the patients with lip tumor report to hospital in stages I and II of the disease.

Smoking cessation may decrease the risk of lip tumor formation.

References