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Prognostic and Predictive Factors for Oral Squamous Cell Carcinoma: A Review

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The extraordinary occurrence of oral squamous cell carcinoma and its unfavourable prognosis encourage further study of variables that can affect the course of the disease. In this review paper, the authors discuss the variables that may have an impact on the prognosis and ultimately help choose which patients will receive more aggressive treatments. Three primary groups of factors associated to patients, tumours, and treatments were formed using collected, chosen, and grouped published scientific data. Aspects that are well known as well as others that are uncommon or have only hypothetical utility are examined. The prognosis is greatly influenced by the disease stage, extracapsular spread, the presence of a disease-free resection margin, and tumour thickness. The research of tumour molecular variables has drawn increased attention recently, and some of these factors have been closely connected with the outcome, indicating prospective avenues for the creation of future prognostic systems and anticancer medicines.

1. INTRODUCTION

At least 90% of all oral cancers are squamous cell carcinomas (SCC, Fig. 1). 1,2 Oral cancer is the third most prevalent malignancy in south-central Asia, and it ranks eighth in terms of cancer incidence globally, with epidemiological variations by area. 3 The World Health Organization predicts that over the coming decades, the incidence of oral squamous cell carcinomas (OSCC) will increase globally.In the US, OSCC accounts for 2%–4% of all diagnosed malignancies each year and causes 8,000 fatalities annually.[4,5] In the US, 36% of patients had localised illness at the time of diagnosis, 43% have regionally distributed disease, and 9% have distant metastases.

The prevalence of OSCC has been on the rise in certain nations in western Europe, including Scotland, Greece, Portugal, Denmark, Belgium, and Denmark. In Eastern Europe, where OSCC is a serious public health concern, rising mortality rates have been seen for at least 20 years. 6 OSCC implies fairly high death and morbidity rates1,2,4,5, and despite the extensive research and surgical and oncological advancements made, the mortality rates have not changed.

2. METHODS AND MATERIALS

MEDLINE was used to launch a web-based search for all types of published articles using the keywords "oral," "cancer," and "prognosis." Then, the search was focused. Additionally, the websites of specialised scientific journals in oncology, oral and maxillofacial surgery, and

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were used. All authors participated in the selection of the information to be used in order to produce a text that was succinct and instructive, first on an individual basis and then by group decision.

We have subsequently developed a broad range of factors, some of which are well established and may have an impact on the course of this disease, while others are more recent and may merely have conjectural value. There hasn't been any extra statistical investigation. The difficulties were divided into three categories: factors relating to the patient, tumour, and treatment.

Numerous genetic anomalies have been found in OSCC, with chromosomes 3,9,11 as showing the highest frequency. Along with other factors, the inactivation of tumor-suppressor genes like p16 (9p21) and p53 (17p), the overexpression of oncogenes like PRAD-1 (11q), and changes to genes involved in carcinogen metabolism or DNA repair appear to contribute to the development of OSCC. Additionally, the majority of oral carcinomas exhibit telomerase activity. 19.Studying these changes is crucial for understanding how cancer cells behave and has implications for identifying individual and familial risk, noninvasive early diagnosis, tumour staging, treatment, and prognosis. 51 The following is a summary of some of the genetic changes implicated with OSCC's prognosis that have been the subject of the most in-depth research.

People who smoke and consume alcohol appear to be more likely than nonsmokers and abstainers to acquire second primary oral cancer, which carries with it more severe consequences.[14-17] This is also true for people who continue to use tobacco and drink alcohol after being diagnosed with a primary tumour.[15-18] Therefore, clinicians should make every effort to urge all patients—including those who have already received treatment for OSCC—to give up these harmful routines.

Socioeconomic circumstances. It appears that individuals with lower socioeconomic position and education had somewhat worse outcomes, most likely as a result of poorer oral hygiene and more challenging access to medical care.[10] Delays in diagnosis. The likelihood of increased tumour growth and dissemination is increased by diagnostic delays, which is highly likely to worsen the prognosis.

However, a thorough analysis of OSCC revealed that the data did not support this claim19, which was partially attributable to methodological shortcomings in the published research. Another hypothesis is that patients with more aggressive tumours have symptoms earlier and seek medical care sooner, but they still have to deal with more severe outcomes since these tumours exhibit more aggressive biologic behaviour.

Indicators for cell division. To measure cell proliferation, a variety of techniques have been employed. As a result of the heterogeneity of the series, the differences in anatomic locations, or other methodological variations, different markers have been considered relevant to prognosis in some papers while markers of proliferation failed to correlate with the prognosis in other papers, raising questions about their prognostic value. Molecules of intercellular adhesion. Intercellular adhesion molecules have a crucial role in the growth, invasiveness, and appearance of metastases in tumours. OSCC has been connected with some expression and/or functional changes, but E- and P-cadherins, catenins, and CD44 are the ones most frequently linked to OSCC prognosis. Numerous studies link a worse prognosis to original tumour changes in the CD44v9 phenotype, decreased CD44v3 expression, and particularly diminished E- and P-cadherin expression.

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Substances that aid in cell-to-cell adhesion. For a tumour to form, to be invasive, and to manifest metastases, intercellular adhesion molecules are crucial. It has been documented that OSCC patients have some expression and/or functional changes. E- and P-cadherins, catenins, and CD44 are the ones that are most frequently linked to OSCC prognosis. According to several articles, primary tumour alterations of the CD44v9 phenotype, decreased expression of CD44v3 and, most importantly, decreased expression of E- and P-cadherin are all associated with a worse prognosis.

The most often altered gene in many common human tumours is still p53, but even in those situations where there are no mutations, other mechanisms frequently affect the gene's function. Regarding HPV, this could happen as a result of an interaction between p53 and protein E6, which is encoded by the carcinogenic HPV types, primarily HPV-16 and HPV-18, and which causes p to undergo more ubiquitin-dependent proteolysis.Additionally, this strategy might help with staging and the selection of patients for adjuvant therapy. The selection of the type and extent of neck dissection, as well as the aggressiveness of surgical treatments, may benefit from sentinel node identification via lymphocyntigraphy or dye injection.The technique appears to be simple to use and quickly locates the sentinel node.

3. DISCUSSION

The mortality and morbidity rates for OSCC are still very high despite the advances made in diagnosis and treatment of the disease. This presents a challenge to the methods for determining prognosis and motivates researchers to look for new and improved markers, specifically molecular markers that relate thoroughly to known changes in tumour progression. The biological distinctiveness of each patient and the biologic distinctiveness of each malignancy must be taken into account when analysing the enormous diversity observed in clinical oncology.

Numerous molecular markers have been linked to the outcome of OSCC, illuminating the intricate processes involved in carcinogenesis and the spread of cancer.Additionally, some of the suggested markers are widely contested, and results occasionally appear to conflict. This condition may be explained by a number of variables, including the limited number of participants in each trial or the heterogeneity of the patients who were chosen, who regularly varied in many parameters, most notably tumour site.

Prognosis is influenced by a wide range of variables, and it's unlikely that any one marker can reliably forecast the outcome. Since tumour growth is a complex, multi-step process, it stands to reason that numerous marker analysis could be needed to determine the ultimate results.

Unfortunately, the general adoption of biologic markers into routine clinical practise has been delayed and often ineffectual, making it difficult to complete clinical investigations to determine their true value and promote their final adoption. The translation into the clinical context is further complicated by the dispersion of published data.

For the early detection and characterisation of diseases as well as the monitoring of health status, proteomic analysis of whole body fluid protein components has been established. Additionally, salivary biomarkers, which rely on a presumption that there is a connection between systemic disease and salivary proteins diseases, could be useful. Proteomic analysis of many indicators found in saliva, a benign and accessible procedure, may one day become a potent bedside technique.

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It is possible that a forthcoming comprehensive molecular and clinical staging system will make it easier to identify people who need more aggressive, targeted, or particular cancer treatment. We are sure that increased prognostic and therapeutic success, as well as subsequent adoption of the techniques described above, will significantly lower the morbidity and mortality rates related to OSCC.

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