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A Discussion on the Need of Early Detection for Oral Cancer

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Abstract: *Oral cancer (OC) is an uncommon illness in Western countries, but it is one of the most common in certain high-risk areas of the world. It is a cancer that is often preventable since the majority of the major risk factors drinking, smoking, and eating betel nut—are behaviors that increase the likelihood of the sickness. Early diagnosis is essential due to the high fatality rate linked to this illness. The initial stages in both preventive and diagnostic prediction are the discovery of locally stimulating chronic inflammation and potentially malignant lesions of the oral mucosa. It is essential to locate and treat each lesion as soon as possible because of this. Oral mucosal lesions may be clinically assessed to identify up to 99% of mouth cancers and premalignancies.[1] The World Health Organization recommends a biopsy for any problematic lesion that remains after it has been detected and the local causes of irritation have been eliminated. Surgical biopsy remains the gold standard for diagnosing oral cancer. To help physicians with diagnosis, other technologies have been developed and studied. Two examples of these are toluidine blue vital staining and autofluorescence imaging. The fatality rate from oral cancer may soon decline as a result of other strategies, such as the identification of salivary indications of advancement.*

Keywords: Oral Cancer, Precancer, Early Diagnosis, Relevance

I. INTRODUCTION

Over 90% of oral cancer cases are melanomas, lymphomas, and OSCCs. Many degrees of differentiation in OSCC lead to nodal metastases. T stage, invasion depth, tumor thickness, and neck lymphatic migration are closely associated.

Oral and oropharyngeal cancers are the sixth most prevalent globally. Two-thirds of the 400,000 new oral cancer cases expected worldwide are in Asia, including Bangladesh, Pakistan, India, Indonesia, and Sri Lanka. Oral cancer is the most frequent cancer in high-risk nations, accounting for 25% of new cases. Oral cancer is most common in those over 60, however it is becoming more common in those under 40.[2]

Oral cancer's 40% 5-year survival rate is concerning. If the sickness is caught early (stages I and II), the survival rate may exceed 80%. Most oral cancer patients are asymptomatic and do not seek medical attention until they have symptoms like pain, bleeding, or a mass in the mouth or neck if lymphatic dissemination is present. Up to 50% of oral cancers are found at stage III and IV. A month-long diagnostic delay increases the risk of advanced mouth cancer. Poor medical care may delay oral cancer diagnosis and treatment.[3] A high proportion of patient errors delay diagnosis. Prognoses increase as the illness advances and the tumor becomes tougher to reach. Prognosis depends mostly on clinical and pathological findings. Due to the high death rate, early identification and prediction improve prognoses, survival rates, and treatment-related morbidity.

Heavy tobacco use, especially smokeless tobacco, alcohol, betel nut chewing, and chronic inflammation are oral cancer risk factors. In recent years, HPV-related oral and oropharyngeal cancers, especially type 16, have risen in younger individuals. Clinical and scientific investigations have examined the carcinogenic potential of oral microbiota, mucosal inflammation, and dental and prosthetic device-induced oral mucosal damage.[4] Actinic UV, especially UV-B, affects lip cancer. DNA repair defects increase the risk of hereditary diseases such ataxia-telangiectasia, Fanconi anemia, and xeroderma pigmentosum.

With most risk factors under control, mouth cancer is usually prevented. Even non-risky people may acquire it. Thus, oral cancer primary prevention involves reducing behavioral risk factors, opposing smoking and addiction, and

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restricting alcohol use. HPV vaccine may prevent cervical and anogenital cancer, even though its efficacy is uncertain. After the carcinogenic process begins, early diagnosis and treatment of oral premalignancies and early-stage cancers provide the basis of secondary prevention. Despite 40 years of mouth cancer awareness, the rate of advanced illness patients seeking treatment has not changed.

Unlike colorectal and cervical cancer, a population-based systematic screening program cannot suggest or fund oral cancer. A randomized controlled study in India found that screening programs may benefit high-risk populations or individuals who have had cancer beyond the head and neck. In nations with frequent dental visits, opportunistic screening for oral mucosal lesions may speed diagnosis.

Potentially Malignant Lesions of the Oral Mucosa

Before OSCC develops, potentially malignant oral epithelial lesions (PMOELs) may appear. Clinically problematic oral mucosal lesions include leukoplakia, erythroplakia, submucosal fibrosis, and lichen planus. Most PMOELs do not become cancerous. [5]

Oral leukoplakia was initially characterized by the WHO in 1978 as an uncontrolled white patch of oral mucosa without clinical or pathological features. Leukoplakia is a clinical word without a histology, hence it does not suggest an epithelial dysplasia stage. Any alternative diagnosis would categorize the lesion into a separate category, making it exclusionary. Therefore, it's important to rule out oral mucosal white patches with a recognized cause. like lichen planus, candidiasis, and trauma-induced sores. About 1-3% of persons suffer leukoplakia, which usually affects men and peaks between five and seven. These lesions initially affect the alveolar mucosa, then the buccal, palate, tongue, and oral floor. Patients frequently have several lesions.

As with OSCC, smoking increases the incidence of leukoplakia, although nonsmokers are equally at risk. Idiopathic leukoplakia. If smoking causes leukoplakia, quitting may remove it.

They have a 2–3% yearly leukoplakia transformation rate. In addition to the lesion's initial site, adjacent oral cavity and upper aerodigestive tract areas might develop malignancy. Oral mucosal cancer risk rises with leukoplakia.

Homogeneous and non-homogeneous oral leukoplakias exist clinically. Homogeneous lesukoplakias with minimal surface cracks are more common. They're usually harmless. Non-homogeneous leukoplakias might have nodules, exophytic, papillary/verrucous, white and red (erythroleukoplakia), flat and speckled, etc. Non-homogeneous leukoplakias are more likely to become cancerous, hence distinguishing them is vital. Some publications have related erythroplakia to non-homogeneous lesion change. In most instances, leukoplakias are benign keratosis, hyperkeratosis, or hyperplasia; dysplasia or malignancy are rare. The sample's dysplasia detection rate increases with lesion thickness.

The rare, high-risk multifocal leukoplakia proliferative verrucous leukoplakia (PVL) typically affects women in their 60s who have never smoked or drank. PVL usually affects the tongue, buccal, gingiva, and alveolar mucosa. PVL begins as small, uneven white plaques that grow into verucous and exophytic lesions. After surgical excision, recurrence and malignant transformation are 60%–100%. It must be diagnosed as soon as possible using accurate diagnostic criteria like those below: It is likely to develop cancer, thus it must be diagnosed as soon as possible.

two or more leukoplasic lesions in the mouth, commonly on the gingiva, palate, and alveolar processes; lesions that have become bigger or more scattered as the disease has progressed; deep OSCC with a biopsy.

Erythroplakia

Erythroplakia often manifests as a richly crimson, velvety-feeling lesion. Rarely, the lesion may also include reddish-white tissue patches; this is a disease known as speckled erythroplakia, erythroleukoplakia, or leukoerythroplakia. In addition to erythroplakia, the differential diagnosis also includes mucositis, systemic lupus erythematosus, candidiasis, and lichen planus. When erythroleukoplakia initially develops, people may feel pain or burning, even though erythroplakic lesions are often asymptomatic. Most often, this lesion affects the tonsillar pillars, ventral tongue, floor of the oral cavity, and soft palate. Most patients only have one lesion.[6]

In terms of frequency, leukoplakia is more common than erythroplakia. The frequency in the general population is thought to be between 0.2% and 0.02%. Alcohol use and smoking are two more risk factors for oral cancer. Some authors claim that every red, velvety lesion of the oral mucosa, whether or not it contains white components, should be diagnosed as cancer, or at the at least, as carcinoma in situ. This is due to the fact that up to 85% of erythroplakias

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exhibit histological signs of malignancy at the time of biopsy, including carcinoma in situ and invasive carcinoma. Treating erythroplakia is important because it poses a serious danger. Treatment should start quickly. Prior to the complete excision of lesions showing considerable dysplasia as identified by histopathologic evaluation, incisional or excisional biopsies should be carried out. After excision, recurrence is possible, especially in larger lesions.

Oral Lichen Planus

Lichen planus is a chronic inflammatory disorder with an immune base that mostly affects women between the ages of 30 and 60. It is most common in middle-aged persons. While oral lichen planus (OLP) mostly affects the skin, it may also cause damage to the oral mucosa. The oral form of lichen planus often lasts longer than the cutaneous form, which may disappear in six to twelve months. The three most common subtypes of OLP are reticular, which is characterized by hyperkeratotic plaques or papules and Wickham striae, bullous, which has bullae that can rupture and cause ulceration, and papular, plaque, atrophic, erosive with areas of ulceration associated with keratotic white striae. Since the oral lichen planus plaque subtype might resemble leukoplakia, a biopsy is strongly advised. The buccal mucosa, gingiva, and tongue are the most frequently affected areas in OLP; most patients have multiple, bilateral lesions.[7] Atrophic and decreasing OLP may make speaking and swallowing difficult and painful. There is much debate on the probability that oral lichen planus might progress to oral squamous cell carcinoma, and the rates of transition differ between 12.5% and 0%: Based only on histology or clinical symptoms, a malignant change in the future cannot be predicted. In this case, erosive OLP has the greatest likelihood of becoming cancer, whereas reticular OLP and atrophic OLP have the lowest likelihoods. Cancers connected to leukoplakia are not usually connected to OLP lesions and may arise in other areas of the oral cavity.

Submucosal Fibrosis

A persistent fibrotic lesion of the oral mucosa is called submucosal fibrosis. The overhealing wound is most likely caused by the mucosal lining constantly being damaged by chemical or mechanical assaults. It is well accepted that there is an estimated 9% chance that this lesion is malignant. Palpable fibrous bands are a characteristic of submucosal fibrosis, which may impair tongue movements and restrict mouth opening since the afflicted tissue no longer has fibroelasticity. The buccal mucosa is the area most often affected, and most instances have been seen in men. The tongue, lip, palate, and gingiva come next in sequence. Previously believed to have an idiopathic cause, submucosal fibrosis now has a complex etiology including iron, zinc, and vitamin deficits as well as capsaicin, which is found in chilies. Chewing betel nuts has been proven to be the primary cause of arecoline-induced fibrosis in the lamina propria in India and other east Asian nations. Arecoline is a chemical present in areca nuts that activates fibroblasts. Fibroelasticity decreases when the illness worsens and the fibrosis spreads to the deeper submucosal tissues.

Chronic Inflammation

Chronic mucosal irritation has been identified as one of the etiological causes of oral cancer. Chronic inflammation that follows prompts the body to release mediators such as cytokines, which in turn trigger oxidative stress and subsequent damage to DNA inside cells, initiating the cascade that culminates in cancer.

Implants, prosthetics, ill-fitting dentures, and parafunctional habits (such as tongue thrusting and oral mucosa sucking) may all cause long-term stress on the oral mucosa. The oral mucosa is typically impacted when it comes into touch with teeth or dental implants, and the lateral border of the tongue is most frequently the location of cancers linked to chronic inflammation.[8]

Persistent mucosal trauma is known to both cause and accelerate oral cancer since it

Oral Bacteria and Cancer

It is well knowledge that people who have oral cancer generally practice very poor oral hygiene.

mouth bacteria, especially those present in periopathogenic biofilms, have been linked to the development of mouth cancer in several ways, according to recent study. Porphyromonas gingivalis and Fusobacterium nucleatum, two oral biofilm-forming bacteria, interact biologically with epithelial cells to produce this connection. Among the theories proposed to explain the possibility for cancer are

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Bacteria interfere with adhesin and E-cadherin synthesis and utilization, which leads to their disruption of immunodefensive mechanisms, effects on extracellular signal-regulated kinases, gingipains, and host matrix metalloproteinases, inhibition of apoptosis, and promotion of proliferation.

Other Lesions That May Be Premalignant Other likely premalignant disorders of the oral mucosa include dyskeratosis congenita, epidermolysis bullosa, discoid lupus erythematosus, recurrent Candida infections, and actinic keratosis (limited to lip cancer).[9]

Risk of Transformation

Most PMOELs do not progress to malignancy, and none of them are required as a prerequisite for OSCC. Differentiating PMOELs with a greater transformation risk from those with a lower risk may be challenging; dysplasia is thought to be the most helpful marker of potential malignant transformation. It's crucial to remember, however, that not all dysplastic lesions will eventually develop into cancer; some non-dysplastic lesions may also become malignancies.

It is not feasible to determine if dysplastic changes are present prior to taking a biopsy since the presence or absence of dysplasia is not directly connected with a particular clinical.

Dysplasia describes mucosal architectural changes. These modifications may make the illness mild, moderate, or severe.

DNA mutations in the mucosa influence multiple genes that regulate cell signaling, survival, proliferation, motility, angiogenesis, and cell cycle regulation. Changes cause cell growth variances. As a result, a malignant phenotype develops and may cause cancer. Histological indications of dysplasia include abnormal epithelial stratification, loss of basal cell polarity, drop-shaped ridges, increased mitotic figures, unusually shallow mitoses, dyskeratosis, and keratin pearls in rete pegs. In mild dysplasia, these changes only affect the bottom third of the epithelium; in moderate, they also affect the middle third; and in severe, they affect more than two-thirds.

Severe dysplasic alterations in epithelium thickness and stratum imply intraepithelial cancer, or "carcinoma-in-situ."

First Diagnosis

As soon as possible, identify and treat PMOELs to increase survival and minimize death. The diagnostic approach begins with a clinical oral examination, which includes digital palpation and mouth cavity visual inspection. A complete dental checkup may identify 99% of oral malignancies.

The most common oral mucosal carcinoma symptoms are shown in Table 1. Even while the tumor may not produce symptoms early on, it will change the mucosal lining's surface and texture.

reoccurring mouth sores and/or pain-restricted oral mucosa material and color changes; a localized bleeding patch; a consistently white, red, or mixed white and red plaque; or a persistent lump or development.

Suspicious lesions need further assessment. Since histological examination is the gold standard for diagnosing OSCC, a recent evidence-based protocol from the World Health Organization, the National Institute of Dental and Craniofacial Research, and the American Dental Association recommends biopsying any mucosal lesion that lasts two weeks or longer after local irritants like broken teeth, ill-fitting appliances and prosthetics, dental plaque, etc.

The conventional cytologic study of an oral mucosa epithelial smear lacks the sensitivity and specificity to predict squamous cell carcinoma, although being less invasive. Brush-biopsy and microbiopsy are new oral cytology research methods. These methods are useful for monitoring precancerous lesions and may replace surgical biopsies.

Novel diagnostic auxiliary technologies may overcome oral clinical examination restrictions. While less successful than a biopsy, these methods may assist detect benign from malignant tumors.

Tests include toluidine blue staining, light-based detection, and salivary biomarker evaluation using point-of-care devices.

Biopsies are still the gold standard, but worry, tension, pain, tissue damage, infection risk, discomfort, temporary incapacity, and cosmetic concerns may deter patients.

Clinical objective assessment and surgical biopsy remain the most effective mouth cancer and pre-malignant lesion diagnosis methods.

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Toluidine Blue Staining

To detect oral cancer and pre-cancerous lesions, toluidine blue staining is straightforward, inexpensive, and non-invasive. Some dysplastic epithelium is royal blue-stained with toluidine blue.

The staining pattern is assessed after 30 seconds of 1% aqueous solution on the suspicious lesion, followed by 1% acetic acid to remove germs and salivary pellicle.

Many theories explain how toluidine blue attaches to high-risk and malignant cells, including its affinity for nucleic acids and capacity to bind sulfated mucopolysaccharides. However, this binding mechanism is unknown. Dependence on its operator is a major downside. Its estimated sensitivity and specificity of 72.5–84% and 61.4–70% are still debated. Combining toluidine blue staining findings with clinical examination data may increase sensitivity to 100%, particularly for malignant tumors. Pre-malignant lesions have lesser sensitivity. Toluidine blue sticks to all forms of mucosal ulcerations—traumatic, inflammatory, and pre-neoplastic—limiting its specificity. Toluidine blue positivity is associated with a poorer prognosis because positive lesions grow bigger and develop malignancy more often than negative lesions.

Autofluorescence Imaging

Autofluorescence imaging may help diagnose oral cancer and distinguish lesions that need biopsy, even if clinical examination with white light is still the main method. Many gadgets have been released in recent decades. They should supplement visual and tactile clinical examination, not replace it.[10]

Tissues may autofluoresce in reaction to particular light wavelengths because they contain endogenous fluorophores such NADH, collagen, elastin, keratin, and FAD. Autofluorescence imaging is supported by the idea that cancer and dysplasia alter mucosal fluorescence. Normal mucosa has light-green fluorescence; illness darkens it. Because of a disturbance in the distribution of autofluorescent chemicals in healthy tissues. This method's weak specificity (estimated at 58%) is due to the fact that benign illnesses such inflammatory diseases may resemble malignant and pre-malignant tissue autofluorescence alterations. Benefits include the method's non-invasiveness and 91% sensitivity.[11]

II. CONCLUSION

A patient's life may be saved by early detection of oral cancer, which also lessens the detrimental effects of invasive surgical intervention on quality of life. The availability of novel point-of-care tests using salivary biomarkers that identify the likelihood of malignant transformation, together with a number of screening diagnostic tools and visual aids, may enhance a clinician's ability to define any worrisome lesion. While surgical biopsies and histology are still regarded as the "gold standard," objective clinical assessment should also involve additional procedures. The scientific community is always improving screening protocols and preventative measures to identify oral cancer early on, with the goal of minimizing the diagnostic delay that, in the majority of instances, might save the patient's life.

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