DIAGNOSTIC AND TREATMENT METHODS FOR ORAL SQUAMOUS CELL CARCINOMA OFFERED AT THE UNIVERSITY OF NAIROBI DENTAL TEACHING HOSPITAL-A TEN YEAR AUDIT [1997-2007]

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1.0.0 ABBREVIATIONS

BDS  Bachelor of Dental Surgery
KNH  Kenyatta National Hospital
MDS  Master of Dental Surgery
Mres  Master of Research
NRB  Nairobi
OSCC  Oral Squamous Cell Carcinoma
SPSS  Statistical package for social sciences
UONDH  University of Nairobi Dental Hospital
UON  University of Nairobi

2.0.0 ABSTRACT

Oral Squamous cell carcinoma remains the most common malignant neoplasm of the oral cavity accounting for over 90% of oral cancers. Its accurate diagnosis is therefore very important if appropriate treatment is to be offered to the patient. The objective of this study is to audit the diagnostic methods for OSCC and the treatment modalities offered or prescribed at University of Nairobi dental hospital between 1997 and 2007. The study will be a retrospective approach and it will involve getting data from patients' clinical records. The patients who will be included in the study will be those histologically diagnosed of OSSC and treated at the UONDH in the specified period. Descriptive longitudinal cohort study design will be used. Data collected will be analyzed and the results will be made available to UONDH to aid in the improvement of diagnostic methods for OSCC and patient management. The study will be conducted between September and October 2007.
INTRODUCTION

Oral Squamous cell carcinoma is a malignant neoplasm that affects the oral epithelium. It’s the most common neoplasm of the oral cavity accounting for over 90% of malignant oral cancers. Although it may occur at any intraoral site, certain sites are more frequently involved. Shafer, Hine and Levy 1974 found the tongue as the most affected site followed by the floor of the mouth, alveolar mucosa, palate and buccal mucosa.

AGE AND GENDER INCIDENCE

Oral cancer is an age related disease. According to Cawson and Odell 2002, 98% of patients in the United Kingdom are over 40 years old. In the general population the incidence is 1 in 20,000. In males above 75 years of age the incidence is 1 in 1100. The ratio of males to females is 3:2.

AETIOLOGY

Oral Squamous cell carcinoma has multifactorial etiology. Cawson and Odell 2002, have documented the following causative factors; Tobacco smoking is a major etiologic factor particularly in association with alcohol. It can be smoked in form of cigarettes, cigar, pipe or reverse smoking. Smokeless tobacco, which includes chewing and snuffing, is held in the lower buccal sulcus for prolonged periods. These habits cause extensive hyperkeratotic plaque and after decades of continuous use may lead to verrucous carcinoma as well as OSCC. Tobacco also contains numerous carcinogens like benzpyrines.

Alcohol dissolves carcinogens and enables them to pass through the bilipid layer of cells and therefore acts as a promoter. It may also predispose to carcinoma through its action in the liver and some studies have found a correlation between mouth cancer and cirrhosis. As with smoking, the total consumption is probably a critical factor.

Nutritional deficiencies such as lack of vitamins A, C and E, which scavenge for free radicals, may lead to development of cancers. This is because free radicals like superoxide and nitrides bind and damage the DNA. Indole compounds found in fruits and vegetables are known to bind and inactivate carcinogens and their lack may lead to development of neoplasm. Paterson-kelly syndrome in which iron deficiency is a feature is associated with a high incidence of mouth and esophageal cancers.

Immunossupression may also play a role because high cases of oral Squamous cell carcinoma are seen in young people who are HIV/AIDS positive.

Viral infections like Herpes viruses and human papilloma viruses [HPV] are implicated in Squamous cell carcinoma. DNA of Hpv-16 for instance has been found in oral cancers. It induces p53 gene mutation leading to increased frequency in oral cancer. Viral proteins E6 and E7 also bind to P53 and RB genes and inactivate them predisposing to development of cancers due to disruption of the cell cycle.

Chronic hyperplastic candidiosis which is a fungal infection can lead to formation of hyperkeratotic plaques or speckled leukoplakias with epithelial dysplasia. There have been isolated reports of malignant
change in such lesions, but overall, this accounts for a very small population of cases.

Syphilitic leukoplakia developing in late stage disease has a high malignant potential but it’s no longer a significant risk factor.

Use of a quid of areca nut, lime, tobacco and spices wrapped in betel leaf is a widespread habit in Indian sub-continent. The quid is held in the sulcus. Areca nut releases arecolin, which is carcinogenic, while the tobacco has numerous carcinogens. White patches may develop where the quid is held and these have a high risk of malignant change. Betel or areca nut chewing can also cause oral sub mucous fibrosis, which also appears to be premalignant.

Research by mehrota and Yadav, 2006 has found that growth regulators and TSGs [Tumor Suppressor Genes] act as transducers of negative growth signals. Genetic alterations involving the tumor suppressor genes p16 and p53 are frequently observed in head and neck tumors. Genetic abnormalities inactivating the p16 gene might confer cell growth defects, contributing to the tumorigenic process. These genes are involved in cell cycle regulation including cell cycle arrest and apoptosis. Alteration in both alleles of a gene is required for the loss of function. The TSG p53 is called as 'Guardian of the Genome', having a role in maintaining genomic stability, cell cycle progression, cellular differentiation, DNA repair and apoptosis. A number of findings indicate that p53 plays an important role in cell-cycle control (both G1/S and G2/M checkpoints) and in the induction of apoptosis. The gene can be inactivated by several mechanisms, including point mutations, deletions and binding with cellular and viral proteins. p53 gene-inactivation via the above mentioned factors, has been demonstrated in squamous cell carcinoma. Due to its high catabolic rate, it is not usually possible to demonstrate p53 protein in normal tissues using immunohistochemical procedures, whereas mutated p53 exhibits a much lower catabolic rate and accumulates in the cells. There are a few genetic disorders, notably dyskeratosis congenital of which oral cancer is a frequent feature.

3.3.0 CLINICAL FEATURES

The clinical features of OSCC vary and may include the following, white patches in the mouth which do not rub off [leukoplakia], red patches [erythroplakia] or speckled patches which are red and white [leukoerythroplakia]. It may present as an indolent ulcer, which doesn’t heal. The ulcer has rolled up borders. Paraesthesia and numbness, Loosening of teeth, Nonspecific unexplained pain but it’s painless unless secondarily infected, Involvement of regional lymph nodes due to metastasis. The lymph nodes are fixed and painless. The organ involved is usually fixed.

3.4.0 MODES OF DISTANT METASTASIS

Oral squamous cell carcinoma may spread to other parts of the body via either of the following ways;Lymphatics, Intravascular, Bone infiltration especially inferior alveolar bone, soft tissue infiltration or Perineural spread.

3.5.0 DIAGNOSIS
Diagnosis is achieved by combining findings from dental and medical history, clinical examination of the lesion and histology. Investigations done include incisional biopsy of the ulcer, biopsy of nodes and radiography especially OPG to check for alveolar bone infiltration.

Due to distant metastasis especially to lungs, liver and bones the following systemic investigations are necessary; chest x-ray, full blood screen, blood grouping and cross matching, urea and electrolytes, Liver function tests, electrocardiography [ECG] and blood gas analysis to check lung function.

3.6.0 TREATMENT

The treatment modalities available for OSCC include; surgery, radiotherapy, chemotherapy, photodynamic therapy and palliative treatment. A single or a combination approach is used.

4.0.0 LITERATURE REVIEW

4.1.0 DIAGNOSIS OF ORAL SQUAMOUS CELL CARCINOMA

Diagnosis is achieved by combining findings from clinical history and examination of the lesion, radiography, imaging and histological appearance.

4.1.1 CLINICAL FEATURES

Early features include; painless, red, speckled or white patches with only a few which are ulcerated. Late features are raised nodule which may become ulcerated to form an indurated ulcer with the typical rolled border, fixed lymph nodes which are painless unless secondarily infected, paraesthesia and numbness, loosening of teeth and bleeding either spontaneously or due to mild trauma.

4.1.2 RADIOGRAPHY AND IMAGING

An orthopantomogram is used to check for bone infiltration. A chest x-ray is taken to check for metastasis to the chest. Imaging techniques like CT scan, ultrasound of the nodes and MRI are also taken.

4.1.3 BIOPSY

An Incisional biopsy of the ulcer is taken. It includes a portion of the ulcer, its border and a margin of normal tissue. Biopsy of the lymph nodes is done to check for metastasis. Brush biopsy- a round stiff bristle brush is used to collect cells from the surface and sub-surface layers of a lesion by vigorous abrasion until bleeding starts. The cells collected are transferred to a microscope slide and the smear is scanned in an image analyzer to identify abnormal cells, which are examined for diagnosis. Kosicki DM et al, 2007 of university of Zurich Germany reported the oral CDX brush biopsy as a proved valuable new minimally invasive method of early detection and surveillance of OSCC of innocuous appearance.

4.1.4 SALIVA ANALYSIS
A comprehensive salivary analysis for oral cancers diagnosis by Shpitzer et al of Tel Aviv Israel, 2006 revealed an overall altered salivary composition in OSCC indicating a compromised oral environment in these patients and suggesting salivary analysis as a new diagnostic tool for oral cancers. The salivary parameters analyzed included Na, K, Ca, Pi, total protein, albumin, LDH, IgG, IgA, igf-1, Metalloproteinases mmp-2,

4.1.5 LAB-ON-A-CHIP TECHNOLOGY

According to Yang Li et al, this technology can be used in screening and diagnosing oral cancers. The design of a microfluidic lab-on-a-chip system for point-of-care cancer screening and diagnosis of OSCC is presented. The chip is based on determining an approximately 30-gene transcription profile in cancer cells isolated from oral fluid samples. Microfluidic cell sorting using magnetic beads functionalized with antibody against cancer specific cell surface antigens e.g. epithelial cell adhesion molecule [EpCAM] is described. A comprehensive cancer diagnostic chip will integrate microfluidic components for cell lysis. Nucleic acid extraction and detection of a panel of mRNA isolated from a sub-population of cancer cells contained in a clinical specimen. It can also be used to detect serum transcriptome, which is successfully diagnostic of OSCC.

4.1.6 Tololinium chloride (Toluidine blue) rinsing,

This is a dye, which binds to nucleic acid and can be used as an oral rinse to stain carcinoma and dysplastic lesions blue. A rinse protocol is developed that establishes the diagnosis of early asymptomatic carcinoma.Studies by Imtiaz et al 2003 point to the favorable use of toluidine blue rinse as an effective adjunct to clinical diagnosis. This protocol improves patient outcome, and positively influences risk management. Toluidine blue is a useful diagnostic adjunct which can be used as a screening rinse in high-risk patients to encompass the entire oral mucosa after a clinical examination and as a guide to improve biopsy yields. Oral cancer examination and Toluidine blue screening are recommended for every patient with high risk factors for oral cancer. This should be scheduled at least once a year or more frequently according to the judgment of patient’s dentist or Ear, Nose and Throat surgeon.

4.2.0 HISTOLOGIC APPEARANCE

Individual cells show the features of malignancy i.e. large and irregularly shaped nuclei, darkly stained nuclei (Hyperchromatism), frequent and sometimes abnormal mitoses, loss of well ordered architecture of epithelium, nuclear pleomorphism and altered nuclear cytoplasmic ratio, drop shaped rete ridges, loss of polarity of cells, loss of intercellular adherence, deep cell keratinization and a lymphoplasmacytic infiltrate of highly variable intensity is usually present.

4.3.0 GRADING OF ORAL SQUAMOUS CELL CARCINOMA

Grading is done according to the degree of differentiation.

GRADE 1-well differentiated
-Cells have cytoplasm which stains palely with eosin or may form concentric layers of keratin.

GRADE 2- Moderately differentiated.

-Cells are more irregular and darkly staining and show little evidence of a squamous pattern.

GRADE 3-Poorly differentiated.

-Cells have little cytoplasms and may not be recognized as epithelial cells by routine microscopy.

-Poorly differentiated carcinomas tend to infiltrate more widely at an early stage and are more likely to metastasize and carry a poorer prognosis.

4.4.0 STAGE INFORMATION

The staging systems are all clinical staging and are based on the best possible estimate of the extent of disease before treatment. The assessment of the primary tumor is based on inspection and palpation when possible and by both indirect mirror examination and direct endoscopy when necessary. The tumor must be confirmed histologically, and any other pathologic data obtained on biopsy may be included. The appropriate nodal drainage areas are examined by careful palpation. Information from diagnostic imaging studies may be used in staging. Magnetic resonance imaging offers an advantage over computed tomographic scans in the detection and localization of head and neck tumors and in the distinction of lymph nodes from blood vessels. If a patient relapses, complete restaging must be done to select the appropriate additional therapy.

Springer et al in 2003 used the American Joint Committee on Cancer (AJCC) TNM classification to stage oral cancers as follows;

4.4.1 TNM definitions

*Primary tumor (T)*

- TX: Primary tumor cannot be assessed.
- T0: No evidence of primary tumor.
- Tis: Carcinoma in situ.
- T1: Tumor ≤ 2 cm in greatest dimension.
- T2: Tumor > 2 cm but ≤ 4 cm in greatest dimension.
- T3: Tumor > 4 cm in greatest dimension.
- T4: (lip) Tumor invades through cortical bone, inferior alveolar nerve, floor of mouth, or skin of face, i.e. chin or nose.
- T4a: (oral cavity) Tumor invades adjacent structures (e.g. through cortical bone, into deep [extrinsic])
muscle of tongue [genioglossus, hyoglossus, palatoglossus, and styloglossus], maxillary sinus, and skin of face).

- T4b: Tumor invades masticator space, pterygoid plates, or skull base and/or encases internal carotid artery.

  Superficial erosion alone of bone/tooth socket by gingival primary is not sufficient to classify a tumor as T4.

Regional lymph nodes (N)

- NX: Regional lymph nodes cannot be assessed.
- N0: No regional lymph node metastasis.
- N1: Metastasis in a single ipsilateral lymph node, ≤3 cm in greatest dimension.
- N2: Metastasis in a single ipsilateral lymph node >3 cm but ≤6 cm in greatest dimension; or in multiple ipsilateral lymph nodes, ≤6 cm in greatest dimension; or in bilateral or contralateral lymph nodes, ≤6 cm in greatest dimension.
- N2a: Metastasis in a single ipsilateral lymph node >3 cm but ≤6 cm in dimension.
- N2b: Metastasis in multiple ipsilateral lymph nodes, ≤6 cm in greatest dimension.
- N2c: Metastasis in bilateral or contralateral lymph nodes, ≤6 cm in greatest dimension.
- N3: Metastasis in a lymph node >6 cm in greatest dimension.

In clinical evaluation, the actual size of the nodal mass should be measured and allowance should be made for intervening soft tissues. Most masses >3 cm in diameter are not single nodes but are confluent nodes or tumors in soft tissues of the neck. The 3 stages of clinically positive nodes are: N1, N2, and N3. The use of subgroups a, b, and c is not required but is recommended. Midline nodes are considered homolateral nodes.

Distant metastasis (M)

- MX: Distant metastasis cannot be assessed.
- M0: No distant metastasis.
- M1: Distant metastasis.

4.4.2 AJCC STAGE GROUPINGS

Stage 0

- Tis, N0, M0
Stage I
- T1, N0, M0

Stage II
- T2, N0, M0

Stage III
- T3, N0, M0
- T1, N1, M0
- T2, N1, M0
- T3, N1, M0

Stage IVA
- T4a, N0, M0
- T4a, N1, M0
- T1, N2, M0
- T2, N2, M0
- T3, N2, M0
- T4a, N2, M0

Stage IVB

4.5.0 TREATMENT OPTION OVERVIEW

Depending on the site and extent of the primary tumor and the status of the lymph nodes, the treatment of lip and oral cavity cancer may be by surgery alone, radiation therapy alone, or a combination of these. Some general considerations are as follows; for lesions of the oral cavity, surgery must adequately encompass all of the gross as well as the presumed microscopic extent of the disease. If regional nodes are positive, cervical node dissection is usually done in continuity. With modern approaches, the surgeon can successfully ablate large posterior oral cavity tumors and with reconstructive methods can achieve satisfactory functional results. Prosthodontic rehabilitation is important, particularly in early-stage cancers to assure the best quality of life.

Radiation therapy for lip and oral cavity cancers can be by external-beam radiation therapy or interstitial implantation alone, but for many sites the use of both modalities produces better control and functional results. Small superficial cancers can be very successfully treated by local implantation using any one of several radioactive sources by intraoral cone radiation therapy or by electrons. Larger lesions are frequently managed using external-beam radiation therapy to include the primary site and regional lymph nodes even if they are not clinically involved. Supplementation with interstitial radiation sources may be necessary to achieve adequate doses to large primary tumors and/or bulky nodal metastases. A review of
published clinical results of radical radiation therapy for head and neck cancer suggests a significant loss of local control when the administration of radiation therapy was prolonged; therefore, lengthening of standard treatment schedules should be avoided whenever possible.

Early cancers (stage I and stage II) of the lip, floor of mouth, and retromolar trigone are highly curable by surgery or radiation therapy. The choice of treatment is dictated by the anticipated functional and cosmetic results and by the availability of the particular expertise required of the surgeon or radiation oncologist for the individual patient. Advanced cancers (stage III and stage IV) of the lip, floor of mouth, and retromolar trigone represent a wide spectrum of challenges for the surgeon and radiation oncologists. Except for patients with small T3 lesions and no regional lymph node and no distant metastases or who have no lymph nodes >2 cm, for whom treatment by radiation therapy alone or surgery alone might be appropriate, most patients with stage III or stage IV tumors are candidates for treatment by a combination of surgery and radiation therapy. Furthermore, because local recurrence and/or distant metastases are common in this group of patients, they should be considered for clinical trials evaluating the following: the potential role of radiation modifiers to improve local control or decrease morbidity; or, the role of combinations of chemotherapy with surgery and/or radiation therapy both to improve local control and to decrease the frequency of distant metastases.

Early cancers of the buccal mucosa are equally curable by radiation therapy or by adequate excision. Patient factors and local expertise influence the choice of treatment. Larger cancers require composite resection with reconstruction of the defect by pedicle flaps.

Early lesions (T1 and T2) of the anterior tongue may be managed by surgery or by radiation therapy alone. Both modalities produce 70% to 85% cure rates in early lesions. Moderate excisions of tongue, even hemiglossectomy, can often result in surprisingly little speech disability provided the wound closure is such that the tongue is not bound down. If, however, the resection is more extensive, problems may include aspiration of liquids and solids and difficulty in swallowing in addition to speech difficulties. Occasionally, patients with tumor of the tongue require almost total glossectomy. Large lesions generally require combined surgical and radiation treatment. The control rates for larger lesions are about 30% to 40%. According to clinical and radiological evidence of involvement, cancers of the lower gingiva that are exophytic and amenable to adequate local excision may be excised to include portions of bone. More advanced lesions require segmental bone resection, hemimandibulectomy, or maxillectomy, depending on the extent of the lesion and its location.

Early lesions of the upper gingiva or hard palate without bone involvement can be treated with equal effectiveness by surgery or by radiation therapy alone. Advanced infiltrative and ulcerating lesions should be treated by a combination of radiation therapy and surgery. Most primary cancers of the hard palate are of minor salivary gland origin. Primary squamous cell carcinoma of the hard palate is uncommon, and these tumors generally represent invasion of squamous cell carcinoma arising on the upper gingiva, which is much more common. Thus, management of squamous cell carcinoma of the upper gingiva and hard palate are usually considered together. Surgical treatment of cancer of the hard palate usually requires excision of underlying bone producing an opening into the antrum. This defect can be filled and covered with a dental prosthesis, a maneuver that restores satisfactory swallowing and speech.

Patients who smoke while on radiation therapy appear to have lower response rates and shorter survival durations than those who do not therefore, patients should be counseled to stop smoking before beginning radiation therapy. Dental status evaluation should be performed prior to therapy to prevent late sequela.
Management of oral squamous cell carcinoma is complex and depends on the age, medical condition of the patient, exact site, degree of spread to regional lymph nodes [stage] and histological type. Treatment may be by surgery or radiotherapy and may be for cure or palliation. Most intraoral carcinomas are treated by surgery combined with radiotherapy [multimodality therapy].

4.5.1 SURGERY

Surgery alone is preferred for small carcinomas of the tongue, which may be excised for those involving bone because of the risk of later radionecrosis. Surgery can also be done when there has been a poor response to or recurrence after irradiation. The carcinoma is excised with as wide a margin as possible ideally 1cm or more. Modern surgical methods allow excision, reconstruction; grafting or bypassing of almost any structure in the oral regions. Reconstruction surgery is normally performed at the same operation as excision to provide better cosmetic and result. Kademani D and Dierks E Feb. 2006 of Mayo clinic of medicine USA have reported successful cases of surgically excised oral and mucosal dysplasias. Successful Laser excision of oral and mucosal dysplasias has been reported by Meltzer C of Santa Rosa medical centre USA. Okayama University in Japan 2006 reported a successful case of lower gingival squamous cell carcinoma with pulmonary metastasis by adjuvant chemotherapy including paclitaxel cisplatin and 5-fluorouracil following a surgical procedure.

4.5.2 RADIOTHERAPY

Treatment by irradiation provides a more acceptable cosmetic and functional result than major surgery but involves considerable discomfort during a long course of treatment and has unwanted effects in the long term. These effects include;

**During treatment**

Severe xerostomia, Mucositis and ulceration, Acute candidiosis and Skin erythema.

**Long term**

These are; Xerostomia, Mucosal and skin atrophy, risk of osteomyelitis [osteoradionecrosis]. Scarring and fibrosis of tissue, cataract if eyes are irradiated e.g. antral carcinoma and risk of late radiation induced malignancy.

Radiotherapy is carried out by implantation of radioactive material into and around the neoplasm [brachytherapy] or by exposure to beams of x-rays or gamma rays from x-rays generators or radioactive isotopes such as Cobalt [teletherapy]. For teletherapy a mask is made to fit the patients head to allow reproducible beam angulations between visits. Treatment planning is critical because ionizing radiation damages both normal and neoplastic tissue. Imaging techniques accurately localizes the lesion and a dose, usually in the region of 60Gy for oral lesions is given. Damage to the surrounding tissues is limited by fractionating the dose over many visits [e.g. 30 daily fractions] and by applying external beams from many angles but avoiding radiosensitive tissue such as eye and bone.

Freier K et al in 2005 performed a retrospective analysis of 207 patients in Germany and he concluded that
neoadjuvant radiochemotherapy with 40Gy and concurrent low dose cisplatin monotherapy followed by selective surgery is a feasible and reliable therapy concept which results in encouraging overall and disease free survival rates for therapy responders and which reliably selects therapy non-responders by the histopathological assessment of the neck dissection preparation. Those therapy non-responders might benefit from intensified systemic therapy approaches.

4.5.3 CHEMOTHERAPY

A study conducted by Sheng J et al in 2005 in China shows that co-administration of cisplatin [4mg/ml] and SAHA [suberoylanilide hydroxamic acid] synergistically induces cytotoxicity and apoptosis in both Tca8113 and KB cell lines. Their results suggest that SAHA enhances tumor cell sensitivity to subtoxic doses of cisplatin.

The cox-2 selective inhibitor celecoxib suppresses proliferation and invasiveness in the human OSCC. An invitro study done by Kwak YE et al of Korea in 2006 indicated that the inhibition of proliferation and invasion/migration in OSCC line by celecoxib results in anticancerous effects via a variety of cellular and molecular mechanisms. The cox-2 inhibitor may be useful in the inhibition and/or prevention of metastasis.

4.5.4 PHOTODYNAMIC THERAPY

This is a recent approach whereby a light sensitive drug is administered and localizes to the tumor. Exposure of the tumor to light of an appropriate wavelength triggers a photochemical reaction, which should kill cancer cells.

4.5.5 PALLIATIVE TREATMENT

Palliative care is given to patients who have advanced tumors or when treatment fails. Radiotherapy is the most commonly used method for palliative care but surgery is occasionally used when a large tumor compromises the airway or becomes grossly necrotic. Chemotherapy is reserved for widespread metastases or salvage therapy. It causes serious complications and there is evidence that it reduces the chances of survival. Palliative care also includes non-intervention measures, which include tender loving care, counseling and pain control.

5.0.0 STATEMENT OF PROBLEM AND JUSTIFICATION OF THE STUDY

Oral squamous cell carcinoma is the most common malignant neoplasm of the oral cavity accounting for over 90% of all oral cancers. Its associated with a very poor prognosis especially when the patient presents late. Its incidence of 1:20,000 in the general population and 1:1100 in males aged 75 years and above is quite significant.

Diagnostic methods therefore need to be very specific, sensitive and accurate to avoid misdiagnosis of
OSCC. Treatment modalities also need to be up to date and as effective as the current advances allow.

UONDH being the largest Dental teaching hospital in Kenya needs to be highly equipped and well prepared for the management of the many cases of OSCC seen in this country. It is in this light that I recognize the need to audit the diagnostic methods and treatment modalities offered at the UON dental hospital.

6.0.0 OBJECTIVES

6.1.0 General
To audit the diagnostic and treatment methods for OSCC offered at the UONDH within a span of 10 years.

6.2.0 Specific
1. To find out the diagnostic methods used for OSCC in the UON Dental hospital.
2. To find out the types of treatment for OSCC offered at the dental hospital.
3. To find out whether the diagnostic and treatment methods for OSCC at the UONDH have been improving over the past 10 years.

7.0.0 RESEARCH HYPOTHESIS
Oral squamous cell carcinoma is diagnosed by combining findings from patients history, clinical examination, investigations and histology. Its treatment is by a multimodality approach.

8.0.0 VARIABLES

8.1.0 Independent
I. Gender
II. Age

8.2.0 Dependent
i). Date of presentation
ii). Chief complaint
iii). Site of the lesion
iv). Medical condition of the patient
v). Histological diagnosis
vi). Treatment modality used
9.0.0 METHODOLOGY

9.1.0 Study Area

The study will be conducted at the UONDH. It is located in Nairobi the capital city of Kenya. Nairobi is a cosmopolitan city with people from diverse ethnic, cultural and racial backgrounds. UONDH is located along Argwing’s Kodhek road and about 3 km from the city center. It’s a referral hospital and a tertiary learning institution with ability of managing a wide range of Orofacial pathologies.

9.2.0 Study population

The study will include clinical records of patients who were diagnosed of OSCC and treated at the UONDH between 1997 and 2007.

9.3.0 Study design

Descriptive longitudinal [cohort] study.

9.4.0 Population size

All the clinical records of the patients who were diagnosed of OSCC and treated at UONDH between 1997 and 2007 are 53. All the 53 cases will be included in the study. A prevalence of 3% was used in the study.

9.5.0 Data collection

Data will be collected from clinical records in patients’ files.

9.6.0 Data to be collected

Data collected included: age, sex, date of presentation, main complaint of the patient, clinical type and site of the lesion, diagnosis made and type of treatment offered.

9.7.0 Data analysis and presentation

Data will be analyzed using SPSS and presented as graphs, pie charts and tables.

9.8.0 Inclusion criteria

Patients diagnosed of OSCC and treated wholly or partially at the UONDH between 1997 and 2007.

Patients diagnosed of OSCC elsewhere and treated at UONDH between 1997 and 2007.

9.9.0 Exclusion criteria
Patients diagnosed of OSCC at the UONDH but treated wholly elsewhere.

Patients diagnosed of OSCC and treated at the UONDH before 1997.

10.0 ETHICAL CONSIDERATIONS

Permission will be sought from UONDH administration to carry out the study. Research proposal will be submitted to the KNH ethics committee for approval. All information collected will be treated with confidentiality and will be used for research purposes.

11.0 PROBLEMS ANTICIPATED

Some patients who are diagnosed at the UONDH went to other hospitals for treatment.

Lack of important information due to improper record keeping i.e. incomplete clinical notes and misplacement of patients’ clinical files.

12.0 STUDY BENEFITS

The study will provide information about the reliability of methods used in coming up with a precise OSCC diagnosis. It will also provide information on the treatment modalities for OSCC in UONDH to see whether they are up-to-date with what is currently advocated worldwide. The study will show how equipped and prepared the UONDH is in the management of OSCC. The information will be made available to UONDH administration to aid in planning and improving diagnosis of OSCC and patient management.
13.0 PROPOSED BUDGET

**Preliminary phase**

- Internet browsing for 400 min. @50 cts.  
  200
- Rim of foolscaps  
  300
- 2 Bic pens @ 15  
  30
- Typing 30 pages @ 10  
  300
- Printing 30 pages @4  
  120
- Photocopying report of 30 pages @2  
  60
- Binding 3 reports @150  
  450
- 1 flash disk  
  1000

**Data collection phase**

- 2 Bic pens @15  
  30
- Typing 10 pages @10  
  100
- Printing 10 pages @4  
  40

**Report writing**

- Typing 20 pages @10  
  200
- Printing 20 pages @4  
  80
- Binding 3 reports @150  
  450
- Miscellaneous  
  500

**TOTAL COST**  
3850
14.0 REFERENCES


9) Shafer, Hine and Levy. A Textbook of oral pathology, 1974. 3rd ed. chapter 2; pg 106-121


DATA COLLECTION SHEET

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</table>

KEY;
C/O-major complaint
MED.COND-general medical condition of the patient
DX-histological diagnosis
RX-treatment offered