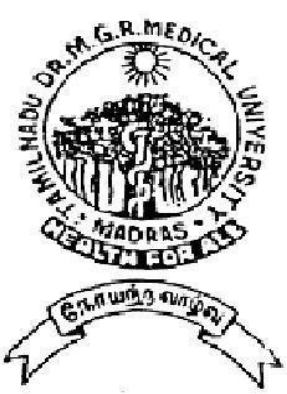
A CLINICOPATHOLOGICAL STUDY

ON LARYNGEAL CANCER

DISSERTATION SUBMITTED FOR

MASTER OF SURGERY Branch – IV

(OTORHINOLARYNGOLOGY)



THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

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This is to certify that the dissertation entitled **" A CLINICOPATHOLOGICAL STUDY ON LARYNGEAL CANCER "** is the bonafide work of **DR. SUMAN A. P.** in partial fulfilment of the university regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai, for **MASTER OF SURGERY BRANCH IV (OTORHINOLARYNGOLOGY)** examination to be held in **April 2015**.

NAME & DESIGNATION OF THE GUIDES:

1. **PROF. Dr.N.DHINAKARAN M.S.,**
   HOD, Department of otorhinolaryngology,
   Madurai Medical College

2. **Dr. P.S.K. THANGARAJ M.S., DLO.,**
   ASSISTANT PROFESSOR,
   Department of otorhinolaryngology,
   Madurai Medical College

CO-ORDINATOR

1. **PROF Dr. N.DHINAKARAN M.S.,**
   HOD, Department of otorhinolaryngology,
   Madurai Medical College

CERTIFICATE FROM HOD

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PROF. Dr.N.DHINAKARAN M.S.,
HOD, Department of otorhinolaryngology,
Madurai Medical College

CERTIFICATE FROM DEAN

This is to certify that the dissertation entitled **"A CLINICOPATHOLOGICAL STUDY ON LARYNGEAL CANCER"** is the bonafide work of **DR. SUMAN A.P.,** in partial fulfilment of university regulation of the Tamil Nadu Dr. M.G.R. Medical University, Chennai, **MASTER OF SURGERY BRANCH IV (OTORHINOLARYNGOLOGY**) examination to be held in **April 2015.**

CAPTAIN.DR.B.SANTHAKUMAR
M.Sc.,M.D(F.M).,PGDMLE.,Dip.N.B(F.M)
DEAN
Government Rajaji Hospital,
Madurai Medical College
Madurai

DECLARATION

I, **DR. SUMAN A. P.,** solemnly declare that, this dissertation **"*A CLINICOPATHOLOGICAL STUDY ON LARYNGEAL CANCER*"** is a bonafide record of work done by me at the Department of ENT, Govt. Rajaji Hospital, Madurai, under the guidance of **Dr. N. DHINAKARAN M.S.,** Professor and HOD, Department of ENT, Madurai Medical college, Madurai.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulations for the award of **MASTER OF SURGERY BRANCH IV (OTORHINOLARYNGOLOGY)** examination to be held in April 2015.

Place : Madurai

Date : **DR. SUMAN A. P.**

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CONTENTS

COMPARISION WITH PREVIOUS STUDIES :

VARIABLES	**OUR STUDY**	**SHELLY CHADHA ET AL(DELHI)**	**VILLANUEVA ET AL (PUERTO RICO)**
AGE	6 TH DECADE	5 TH DECADE	6^{TH} DECADE
SEX	M>F	M>F	M>F
SES	LOW	LOW	LOW
RISK FACTORS	TOBACCO ALCOHOL	TOBACCO, ALCOHOL	TOBACCO ALCOHOL
HPE	SCC	SCC	SCC
STAGE	ADVANCED	ADVANCED	LOCALISED
TREATMENT	CONCURRENT CHEMORT	SURGERY/RT	SURGERY/RT

SES-SOCIOECONOMIC STATUS

SCC-SQUAMOUS CELL CARCINOMA

CHEMORT-CHEMORADIOTHERAPY

RT-RADIOTHERAPY

INTRODUCTION

Laryngeal cancer is primarily a disease of middle aged men. It has peak incidence in the seventh decade. It is relatively uncommon in women. Worldwide it is the common head and neck cancer. Squamous cell carcinoma is the most common type of tumor, comprising 90 percent or more of all laryngeal malignancies. Laryngeal cancer is more common in areas with higher levels of social poverty.

In the world wide 70 large case control studies covering most of the world populations have examined the most common cause of squamous cell laryngeal cancer to be tobacco and alcohol. Occupational hazards are associated with the exposure to asbestos, strong inorganic acid, cement dust, silica. Diet rich in salted meat, high total fat content has increased risk of laryngeal malignancy. Intake of plant food, fruits, legumes are protective against laryngeal cancer. The international agency for research on cancer (IARC) has evaluated that around 89,000 deaths due to this malignancy in males and 12,000 among females worldwide in 2005. Survival rates are lower in under developed countries.

In a study in India the five year relative survival rates were 58.3% for glottis cancer and 31.4% for supraglottic cancers.

In France two case series have confirmed that gastroesophageal reflux disease is present in patients with laryngeal cancer. In a case control study conducted in Brazil, showed an increased risk of developing squamous cell cancer of head and neck whose first degree relative had some type of malignancy.

The cardinal symptoms of laryngeal cancer are voice change i.e., hoarseness of voice, dysphagia, odynophagia, otalgia, airway obstruction, neck mass, weight loss, haemoptysis.

Many factors must be considered in determination of optimal treatment for a patient.

1. Age and Sex
2. General health
3. Personal preferences
4. Social circumstances of the patient
5. Treatment facilities available
6. Stage of the tumour

The treatment modalities may be surgery, radiotherapy or chemotherapy or combination.

Cancer larynx is largely a preventable disease. The risk of cancer is decreased by cessation of smoking and alcohol, as well as reduction in carcinogen exposure in occupational settings. The risk diminishes dramatically only after 6 years of cessation of smoking and approaches

that of non smoker after 15 years. Early diagnosis is the key to survival and cure rate.

This is a hospital based prospective study to find out the common clinical presentation of patients with laryngeal carcinoma with reference to the age profile, the symptomatology, disease stage, etiological factors, occupational history, histological profile and to determine the treatment modalities offered to the laryngeal cancer patients attending ENT OPD at GRH., Madurai.

OBJECTIVES

1. To estimate the incidence of laryngeal cancer among the patients who attend the ENT OPD.
2. To know the age and sex ratio.
3. To assess the socio-demographic profile of laryngeal cancer.
4. To analyze the association of risk factors causing laryngeal cancer.
5. To know the treatment received by the laryngeal cancer patients at Government Rajaji Hospital., Madurai.

REVIEW OF LITERATURE

ANATOMY OF LARYNX

EMBRYOLOGY OF LARYNX

- The cranial end of respiratory diverticulum develops into the larynx.
- Epiglottis develops from caudal end of hypobranchial eminence.
- Fourth, fifth and sixth pharyngeal arches give rise to the thyroid, cricoid and the arytenoid cartilages.
- Epiglottis is the last to develop cartilagenous tissue. Branchial mesoderm gives rise to muscles of the larynx and have the same nerve supply.
- Hyoid bone is derived from the second and the third branchial arches.
- Epithelial and mesodermal mass between the vestibule and upper trachea forms the analogue of vocal cord by 8^{th} week.
- By 10^{th} week this mass splits sagitaly giving rise to a pair of vocal cords.

HISTOLOGY

Mucous membrane of larynx is lined by pseudo stratified ciliated columnar epithelium. But in parts where it comes in contact with swallowed food it is stratified squamous epithelium. These comprises anterior and upper part of posterior surface of epiglottis and upper parts of aryepiglottic fold. Though vocal cords do not come in contact with swallowed food they are exposed to some sort of stress during vibration of folds and are lined by stratified squamous epithelium.

Most of the laryngeal cartilages are hyaline and only epiglottis, corniculate and cuneiform and apical part of arytenoid are elastic cartilages. Increasing age may cause calcification in hyaline cartilage but not in elastic cartilages.

Larynx acts as a conduit for air, a sphincter and an organ of speech. Extending from the tongue to the trachea projects ventrally in between the major vessels of the neck. It is covered anteriorly by skin, fasciae and muscles which act as depressors of the hyoid bone. It moves with deglutition. In adult males it measures 44mm in length with saggital diameter 36mm and transverse diameter 43mm. In females it measures 41mm. It lies opposite to C3-C6 in adult male.

SKELETON OF LARYNX

Larynx is made up of laryngeal cartilages interconnected by ligaments and fibrous membranes and moved by a set of muscles. The hyoid bone is attached to the larynx. Cricoid, thyroid and epiglottis are unpaired cartilages. Arytenoid, cuneiform and corniculate and tritiate are paired cartilages. Surface landmarks in relation to the laryngeal framework are

C3 -Level of body of hyoid and greater cornua

C3 – C4 junction – Upper border of thyroid cartilage and bifurcation of common carotid artery

C4-C5 junction- Level of thyroid cartilage

C6- Level of cricoid cartilage

EPIGLOTTIS STRUCTURE

It is a thin leaf like structure containing of elastic fibro cartilage and projects downwards obliquely behind tongue and the body of the hyoid bone and present in front of the laryngeal inlet. It has a broad and round free end which is occasionally notched in the midline and directed upwards. It has a long and narrow stalk which is attached by the thyro epiglottic ligament to back of the laryngeal prominence of thyroid cartilage which is below the thyroid notch. Aryepiglottic folds attach the sides of epiglottis to the arytenoid cartilages. Reflections of the upper free anterior surface over the pharnyngeal aspect of the tongue and lateral

pharyngeal walls form the median and two lateral glossoepiglottic folds respectively. On either side of the median glossoepiglottic fold depressions called vallecula are present. Hyoepiglottic ligament connects the lower part of the anterior surface of epiglottis to the upper border of the hyoid bone. Lower part is separated from the thyrohyoid membrane by adipose tissue which encloses the clinically important pre epiglottic space. The posterior or the laryngeal surface of epiglottis is pitted by small mucous glands and is perforated by branches of internal laryngeal nerve through which it communicates with the pre epiglottic space.

THYROID CARTILAGE

It is the largest hyaline cartilage in the larynx. It has two laminae, quadrilateral in shape which fuse together in the midline. Due to the shallow angle the laryngeal prominence becomes more prominent and the vocal cords are long resulting in a more deeper pitched voice. The point of junction of upper portion of the laminas forms a V shaped notch called the thyroid notch . Their posterior borders are slender and long and form the superior and inferior cornua.

On the external surface of each lamina there is an oblique line extending from the superior thyroid tubercle to the inferior thyroid tubercle. This line gives attachment to the sternothyroid, thyrohyoid and thyropharyngeus. Fibers of stylopharyngeous and palatopharyngeus are attached to the thick posterior border.

The superior cornua is curved upwards and is long and narrow and gives attachment to the lateral thyrohyoid ligament. The inferior cornua is short and thick and has a facet for articulation with cricoid on the medial aspect of its lower end. The angle between the two lamina on the inner surface gives attachment to the vestibular folds, vocal ligaments, thyroarytenoid, thyroepiglottic and vocalis muscles.

CRCOID CARTILAGE

It is the only laryngeal cartilage which forms a complete ring around the airway. It is thicker and stronger and has an anterior arch which is narrow and a posterior broad flat lamina. The arch of the cricoid cartilage is narrow anteriorly with vertical height of 5-7mm and expands posteriorly to the lamina. On its outer aspect are attached the cricothyroid and cricopharyngeus muscles. The arch is separated from the laryngeal prominence by the cricovocal membrane.

The inferior border of cricoid is attached to the first tracheal ring by the cricotracheal ligament. The cricothyroid membrane is attached to the medial and lateral part of the superior border of cricoid cartilage. Inner surface of the lamina has a median notch on either side of which is a facet for articulation with the arytenoids.

ARYTENOIDS

They are paired hyaline cartilages. They articulate with superior border of cricoid lamina on its lateral aspect to form the cricoarytenoid

joint. Each arytenoid has an apex, two processes and three surfaces and a base. It has a rough and uneven anterolateral surface . There is an elevation near the apex from which a crest curves backwards then to the vocal processes. The upper part of crest gives attachment to the vocal ligament. The lower part gives attachment to the vocalis and the lateral cricoarytenoid. It has a smooth, triangular and concave posterior surface covered by transverse arytenoid. The medial surface of arytenoids in its lower edge forms the lateral boundary of the inter cartilagenous part of the rimaglottidis. Base articulates with lateral part of upper border of cricoid lamina. The muscular process of arytenoids projects backwards and laterally. In front is attached the lateral cricoarytenoid and behind is the posterior cricoarytenoid. The vocal ligament is attached to the pointed anterior angle. Apex is directed backwards and articulates with corniculate cartilage.

CORNICULATE CARTILAGE/ CARTILAGE OF SANTORINI

They are elastic fibro cartilages articulating with apex of arytenoid cartilage extending posteromedially. They are two conical nodules lying within the posterior part of aryepiglottic folds. Sometimes they are found fused with the arytenoid cartilages.

CUNEIFORM CARTILAGE/ CARTILAGE OF WRISBURG

They are small rod like with clubbed ends. They are situated in each aryepiglottic fold anteriorly and superiorly to corniculate cartilage and appear as whitish mucosal elevations.

JOINTS OF LARYNX

CRICOTHYROID JOINT

It is a synovial joint formed by articulation of the inferior cornua of thyroid and the sides of the cricoid cartilage. It has a capsule strengthened by fibrous bands. Along a transverse axis passing through the cricothyroid joints, cricoid moves on the inferior cornua and also glides on the thyroid cornua in different directions.

CRICOARYTENOID JOINT

It is a synovial joint formed by articulation of the base of arytenoid with the facets on the lateral parts of the upper border of the cricoid lamina. There is a joint capsule strengthened by the posterior cricoarytenoid ligament which is medial in position. The arytenoid rotate in an oblique axis swinging the vocal process laterally and medially there by causing an increase or decrease in the width of the rima glottidis. Complex gliding and tipping motion occurs moving the arytenoids laterally and downwards. The forward movement of the arytenoid cartilages on the cricoid cartilage are limited by the posterior

cricoarytenoid ligaments. The position of the crico arytenoid ligament at rest determines the position of denervated vocal cord.

ARYTENOCORNICULATE JOINTS

They are synovial cartilaginous joints linking the arytenoid and corniculate cartilages.

LIGAMENTS AND MEMBRANES OF LARYNX

EXTRINSIC LIGAMENTS AND MEMBRANES

THYROHYOID MEMBRANE

It is a broad, fibroelastic membrane. Inferiorly it is attached to the superior border of the thyroid cartilage and the front of the greater cornua. Superiorly attached to the body and the greater cornua of the hyoid bone. It moves up behind the body of hyoid forming a bursa in between, facilitating the ascent of larynx during swallowing. It is pierced by branches of the superior laryngeal vessels and the internal laryngeal nerve on the lateral part. The posterior border of the thyrohyoid membrane is formed by the cord like elastic lateral thyrohyoid ligaments. Its relations on the inner aspect are the epiglottis and pyriform fossa.

HYO- AND THYROEPIGLOTTIC LIGAMENTS

They attach epiglottis to the thyroid cartilage and hyoid bone.

CRICOTRACHEAL LIGAMENT

Lower border of cricoid cartilage is connected to the first tracheal ring by the cricotracheal ligament and it continues with the perichondrium of the trachea.

INTRINSIC LIGAMENTS AND MEMBRANES

QUADRANGULAR MEMBRANE

It extends from lateral border of epiglottis to the arytenoid cartilage on each side. It has free upper and lower borders. They are poorly defined. The upper border slips back forming the aryepiglottic ligament which forms the central component of the aryepiglottic fold. It traverses the fascial plane of the suspensory ligament of oesophagus posterior forming the median corniculo pharyngeal ligament. This ligament exerts vertical traction. Its lower border forms the vestibular fold.

CRICOTHYROID LIGAMENT AND CRICOVOCAL MEMBRANE

Cricothyroid membrane is mainly elastic and has anterior and lateral parts. Anterior part the median cricothyroid ligament and lateral part, the cricovocal membrane.

Cricothyroid membrane extends from upper border of the cricoid cartilage to the lower border of the thyroid cartilage. It is thickened in front forming the anterior cricothyroid ligament.

Cricovocal membrane/ Conuselasticus/ Lateral cricothyroid ligament – it is thinner arising from the lower margin of inner surface of cricoid cartilage. It ascends up under the lower border of thyroid cartilage and anteriorly attaches to the inner surface of angle of thyroid cartilage. It has a free upper end thickened to form the vocal ligament. Its inner surface is lined by mucosa and outer surface by the lateral cricoarytenoid and thyroarytenoid.

MUSCLES OF LARYNX

Larynx has two types of muscles the intrinsic and the extrinsic group. The extrinsic group move larynx as a whole during swallowing and phonation. These are the sternohyoid, sternothyroid and the thyrohyoid and inferior constrictor of the pharynx. These muscles can influence the tone and pitch of voice by raising or lowering the larynx. During deglutition geniohyoid moves larynx upwards and anteriorly.

The cricothyroid, lateral and posterior cricoarytenoid, transverse and oblique arytenoids thyroarytenoid vocalis have attachments within the larynx expect for transverse arytenoid which is paired. They lie within the thyroid cartilage and only cricothyroid is on the outer aspect.

The transverse arytenoid has the maximum amount of neuromuscular spindles. The muscle mass for adduction is more than that for abduction and also even in normal individuals histopathological examination reveals degenerative changes in the posterior cricoarytenoid

muscle which is the single abductor. Based on the actions they are classified as the Intrinsic muscles

- Muscles which change size and shape of laryngeal inlet - aryepiglottic and oblique arytenoids.
- Adductors - lateral cricoarytenoid, thyroarytenoid, interarytenoid, cricothyroid.
- Tensors - cricothyroid and thyroarytenoid

  Extrinsic muscles
- Elevators – mylohyoid, stylohyoid, thyrohyoid, digastric.
- Depressors – sternothyroid, sternohyoid, omohyoid

INTERIOR OF LARYNX

Larynx extends from the laryngeal inlet to the lower border of cricoid, then continues as trachea. It has two sets of folds projecting into the cavity. The vestibular folds /false cords which cover the median aperture rima vestibule. The true vocal cords are present below this and the guard the glottis opening or the rimaglottides. The part of larynx lying above the glottis is called the supraglottis. It includes the laryngeal ventricle, false cords, laryngeal surface of epiglottis, arytenoids and laryngeal aspects of the aryepiglottic folds.

LARYNGEAL INLET

It is the point of entry between the pharynx and the larynx. Anteriorly it is bound by the upper free edge of the epiglottis and the

interarytenoid mucosal fold posteriorly. Either sides are present the aryepiglottic folds extending from the side of the epiglottis to apex of the arytenoids.

ARYEPIGLOTTIC FOLD

It constitutes fibers from the upper border of quadrangular membrane and the oblique arytenoids. Two oval swellings are present in the posterior part of aryepiglottic folds representing the underlying cuneiform and corniculate cartilages.

LARYNGEAL INTROITUS

It is the space between the laryngeal inlet and the false cords. In the upper part it is wide and narrows down below and is high up in the anterior part. Posterior surface of epiglottis forms the anterior wall, lateral walls by the aryepiglottic folds. Mucosal folds between the arytenoids on the posterior part forms the posterior wall.

MIDDLE PART

The larynx is smallest in the middle extending from the rima vestibule to the rima glottis. Vestibular folds on either side, the ventricle and saccule of larynx are present.

VESTIBULAR FOLDS AND LIGAMENTS

The lower border of quadrangular membrane is thickened to form the vestibular ligament. Anteriorly it is attached to the thyroid angle and

posteriorly to the arytenoids above the vocal process in the antero lateral surface. Vestibular ligaments along with its covering mucosa are called the vestibular folds.

VENTRICLE/SINUS OF LARYNX

On both sides of the larynx, aperture between the false and the true cords leads to the ventricle of the larynx. It is fusiform in shape. Above it reaches till the laryngeal wall lateral to the false cords.

SACCULE OF LARYNX

Saccule extends forwards form the laryngeal ventricle and lies between the vestibular folds and the thyroid cartilage. It is a cone shaped cavity which is directed backwards. It has lot of submucosal mucous glands opening into the saccule. Thyroepiglottic muscle seperates the saccule from the thyroid cartilage. The secretions from these mucosal glands lubricate the vocal cords and prevent them from infection and desiccation.

VOCAL CORDS

They are two in number and are the true cords. They extend backward from the middle of thyroid angle to the vocal processes of the arytenoids. The mucosa covering vocal cord is thinned out lying directly over the vocal ligaments giving it a pearly white colour. Anteriorly the vocal cords meet at the anterior commissure. Broyle's ligament is formed by the fibres of the vocal ligament blending with the perichondrium of the

thyroid cartilage. The glottis consists of the vocal cords, the space which lies in a horizontal plane 1 cm inferior to the level of the upper surface of the vocal cord, the anterior commissure and the posterior commissure.

RIMA GLOTTIDIS

It is an aperture bound anteriorly by the vocal cords and posteriorly by the arytenoid cartilages. Vocal ligaments form its anterior inter membranous part and vocal process of arytenoids forms the inter cartilagenous part. It is the narrowest part of the laryngeal cavity.

SUBGLOTTIS

It lies between the vocal cords above and the lower border of the cricoid cartilage below. The shape of the subglottis is elliptical in the upper part and circular in the lower part. Subglottis continues with the trachea. This is a rare site for origin of cancers but may be involved in the glottis cancers. Subglottic malignancy has higher incidence of extra laryngeal spread. Due to proximity of cricothyroid membrane and postcricoid lymphatics.

SPACES OF LARYNX

REINKES SPACE

The mucosa over the vocal ligaments is loosely attached to the ligaments themselves. There is a submucosal space along most of the length of the free edge of the vocal cords extending from the superior

arcuate line to inferior arcuate line. Blood vessels and lymphatics are almost absent in this space preventing early spread of cancers.

SUBGLOTTIC SPACE

This area extends from inferior margin of true cords to the lower border of cricoid. It is a potential space filled with fibroelasticsubmucosal tissues between mucosa and conuselasticus. The subglottic area does not include the vocalis muscle and is limited superiorly at anterior commissure by the anterior commissure tendon.

CRICOID AREA

This potential space contains the areolar tissue medial to the internal perichondrium of the cricoid. The compartment is situated between the subglottic area and the trachea.

PREEPIGLOTTIC SPACE/SPACE OF BOYER

It is bound anteriorly by the thyrohyoid membrane and the thyroid cartilage above the thyroepiglottic ligament. Its superior relations are the hyoepiglottic ligament and the mucosa of the vallecula. It is posteriorly bound by the infrahyoid epiglottis and the thyro epiglottic ligament. This space is laterally continuous with the paraglottic space deep to the quadrangular membrane and superior to the ventricle. Cancer on the infrahyoid epiglottis spreads readily into the pre epiglottic space.

PARAGLOTTIC SPACE

This space is situated lateral to the ventricle and the glottis. Involvement of this space by malignancy causes fixation of the vocal cord and is considered to be the advanced stage of the cancer. It is bound anterolaterally by the thyroid cartilage and the cricothyroid membrane, superomedially by the quadrangular membrane, inferomedially by the conus elasticus and posteriorly by the reflection of the pyriform sinus mucosa. It blends with the preepiglottic space anterosuperiorly.

The submucosa of the ventricle is continuous with the paraglottic space which is bound by the conus elasticus inferomedially, quadrangular membrane superomedially and the thyroid ala laterally. Inferolaterally the paraglottic space is continuous with the cartilage defect between the thyroid and the cricoid cartilage. Tumor involving the ventricle invades the paraglottic space and then spreads transglottically. Vocal cord tumours which extend deep into the thyroarytenoid muscle invade the paraglottic space which later extends to the subglottic space and then spreads extra laryngeally. Lateral supraglottic tumours can travel lateral to ventricle along the inner surface of the thyroid ala and thus spread subglottically. The close proximity of the mucosa of the pyriform sinus to the posterior paraglottic space makes this a potential route for spread of pyriform sinus malignancy into the endolarynx resulting often in fixation of the hemilarynx.

SURGICAL ANATOMY

The larynx is divided into three sites and each of these sites is divided into sub-sites. This is done according to the International Union Against cancer.

1. SUPRAGLOTTIS

A. Suprahyoid Epiglottis
(including tip, lingual & laryngeal surfaces) } Epilarynx
B. Aryepiglottic fold laryngeal aspect } (Including Marginal Zone)
C. Arytenoid.

D. Infrahyoid Epiglottis } Supraglottis
E.Ventricular bands (False cords) } (Excluding Epilarynx)

2. GLOTTIS

A. Vocal cords

B. Anterior commissure

C. Posterior commissure

3. SUBGLOTTIS

BLOOD SUPPLY AND LYMPHATIC DRAINAGE

Most of the parts of the larynx are supplied by the superior laryngeal arteries except for the region around the cricothyroid which is supplied by the inferior laryngeal artery. Region around the posterior cricoarytenoids is supplied by the posterior branch of the inferior laryngeal artery.

Superior laryngeal branch of the superior thyroid artery supplies the supraglottis. Inferior laryngeal branch of the inferior thyroid artery supplies the subglottis and undersurface of the vocal cords. This accompanies the recurrent laryngeal nerve. Some blood supply comes from the cricoid branch of the superior thyroid artery.

Supraglottis – the lymph vessels pierce the thyrohyoid membrane and drain into the level II and level III jugulodigastric and juguloomohyoid nodes. Ventricular lymphatics also pass through the cricothyroid membrane and ipsilateral thyroid gland to the Level III and Level IV lymph nodes.

Subglottis- the lymphatics form three main trunks. One superficial trunk pierces the cricothyroid membrane and drains into the delphian nodes which inturn drains into the pre and paratracheal and the supraclavicular nodes. The two posterolateral trunks penetrate the cricotracheal membrane and terminate in the para tracheal node and the

superior mediastinum. These nodes drain into the lower deep cervical nodes.

Glottis- This is the watershed area with poor lymphatics. Anterior commissure drains into the prelaryngeal nodes.

Venous drainage is by the superior and inferior laryngeal veins which are the tributaries of the superior and inferior thyroid veins. Superior thyroid vein drains into the internal jugular vein. Inferior thyroid vein drains into the left brachiocephalic vein.

NERVE SUPPLY

Autonomic -Mucous glands throughout the larynx receive parasympathetic secretomotor fibres via the superior and recuurent laryngeal nerves. Postganglionic sympathetic fibres are from the superior and middle cervical ganglia.

Sensory – supraglottis and upper surface of vocal cords are supplied by the internal branch of superior laryngeal nerve. Subglotis and lower surface of vocal cords are supplied by the reccurent laryngeal nerve.

Motor – all intrinsic muscles except cricothyroid are supplied by the recurrent laryngeal nerve. Cricothyroid is supplied by the external branch of the superior laryngeal nerve. Extrinsic muscles are supplied by the ansacervicalis.

ANATOMY OF NECK

LAYERS IN THE ANTERIOR NECK

- Skin
- Superficial fascia with platysma, anterior jugular vein, submental lymph node, anterior cutaneous nerve.
- Deep fascia-Above the hyoid bone is single layer in medial plane splits to enclose submandibular salivary gland. Between hyoid and cricoid- single layer extending below right and left sternocleidomastoid muscle.
- Below cricoid- splits to enclose the suprasternal space.

STRUCTURES LYING ABOVE THE HYOID BONE

Mylohyoid muscle overlapped by

- Anterior belly of digastric
- Superficial part of submandibular salivary gland
- Mylohyoid nerve and vessels
- Submental branch of facial artery

ANTEROINFERIORLY

Hyoglossus muscle with intermediate tendon of digastric and bifurcated tendon of stylohyoid.

STRUCTURES LYING BELOW HYOID BONE

INFRAHYOID MUSCLES - sternohyoid, sternothyroid, thyrohyoid, superior belly of omohyoid.

PRE TRACHEAL FASCIA

Forms the false capsule of the thyroid gland and suspensory ligaments of berry which attach the thyroid gland to the cricoid cartilage. Deep to pretracheal fascia lies the thyrohyoid membrane pierced by the internal laryngeal nerve and the superior laryngeal vessels

ANTERIOR TRIANGLES OF THE NECK

Bounded medially by the anterior median plane of the neck, laterally sternocleidomastoid muscle, superiorly line joining the angle of mandible to the mastoid. It is divided into the submental, digastric, carotid, muscular triangle by the digastric and the superior belly of the omohyoid.

SUBMENTAL TRIANGLE

Bound on each side by the anterior belly of the digastric, base by the body of the hyoidbone, floor by the right and the left mylohyoid muscle and apex by the chin.

Two to four submental lymph nodes and the small submental veins join to form the anterior jugular vein.

DIGASTRIC TRIANGLE

Bound anteroinferiorly by the anterior belly of the digastric, postero inferiorly by the posterior belly of digastric and stylohyoid., superiorly by the base of the mandible and the line joining the mandible to the mastoid process. Floor is formed by the mylohyoid muscle.

Contents – anterior part of the triangle. Structures superficial to mylohyoid - superficial part of submandibular salivary gland, submental artery and the mylohyoid nerves and vessels. Superficial to hyoglossus- submandibular salivary gland, intermediate tendon of the digastric and the stylohyoid and the hypoglossal nerve.

POSTERIOR PART OF THE TRIANGLE

Superficial structures- lower part of parotid and the external carotid artery Deep structures between the internal and the external carotid artery- styloglossus, stylopharyngeous, styloid process, glossopharyngeal nerve, pharyngeal branch of vagus.

Deepest lies the internal carotid artery, internal jugular vein and the vagus nerve and the submandibular lymphnodes.

CAROTID TRIANGLE

Anterosuperiorly posterior belly of digastric and the stylohyoid, anteroinferoirly superior belly of omohyoid, posteriorly anterior border of sternocleidomastoid, roof by the skin, superficial fascia and the deep

fascia, Floor by the middle constrictor of the pharynx, inferior constrictor of the phaynx and thyrohyoid membrane.

CONTENTS

Arteries – common carotid artery with carotid sinus and the carotid body, internal carotid artery, external carotid artery with superior thyroid, lingual , facial, ascending pharyngeal and the occipital artery.

Veins – internal jugular vein with tributaries phrenic vein, lingual vein, common facial vein.

Nerves - vagus with superior laryngeal branches, spinal accessory nerve, hypoglossal nerve and sympathetic chain.

MUSCULAR TRIANGLE

Bound anteriorly by the anterior median line of the neck from hyoid to the sternum, posterosuperiorly superior belly of omohyoid posteroinferiorly, anterior border of sternocleidomastoid. The content is the infrahyoid muscles.

POSTERIOR TRIANGLE

It lies behind the sternocleidomastoid.

Bounded anteriorly by the posterior border of the sternocleidomastoid, posteriorly by the anterior border of the trapezius, inferiorly middle one third of the clavicle and apex on the supra nuchal line where the trapezius and the sternocleidomastoid meet. Roof is formed by the platysma, external jugular vein and the posterior jugular

vein, supraclavicular, transeverse cutaneous and the greater auricular nerves, branches of occipital, transverse cervical and suprascapular arteries.

Floor is formed by the prevertebral layer of the deep cervical fascia with the splenius capitis, levator scapulae and scalenus medius. It is divided by the inferior belly of the omohyoid into the large upper occipital triangle and the small lower supraclavicular or also called subclavian triangle.

OCCIPITAL TRIANGLE

Nerves-Spinal accessory nerve.

Four cutaneous branches of cervical plexus-they are lesser occipital, greater auricular, anterior cutaneous branch of the neck, supraclavicular nerve. Muscular branches of the levator scapulae, trapezius and the rhomboids.

Vessels- transverse cervical artery and the vein and occipital artery.

Nodes – supraclavicular and the occipital nodes.

SUBCLAVIAN TRIANGLE

- Lower trunk of the brachial plexus
- Nerve to serratus anterior
- Nerve innervating subclavius
- Suprascapular nerve
- Suprascapular artery and vein

- Lower part of the external jugular vein
- Transverse cervical artery
- Supraclavicular nodes

SUBOCCIPITAL TRIANGLE

Bound superomedially by the rectus capitis posterior major and the minor, superolaterally by the superior oblique muscle, inferiorly by the inferior oblique muscle,. Roof is mainly formed medially by the dense fibrous tissue covered by the semispinalis capitis and laterally by the longissmus capitis and the splenius capitis. Floor is formed by posterior arch of the atlas.

It contains third part of the vertebral artery, suboccipital nerve, suboccipital plexus of veins and the suboccipital muscles.

LYMPHATICS OF NECK

LEVEL Ia submental – bound superiorly by symphysis menti, inferiorly the hyoid bone and anterior belly of digastric.

LEVEL Ib submandibular- bound superiorly by the body of the mandible, inferiorly posterior belly of the digastric, medially anterior belly of digastric, laterally stylohyoid muscle.

LEVEL IIa upper jugular- bound superiorly by the lower level of bony margin of the jugular fossa, inferiorly level of the lower part of body of hyoid bone, medially by the stylohyoid muscle and laterally by the vertical plane defined by the accessory nerve.

LEVEL IIb upper jugular-bound superiorly by the lower level of jugular fossa, inferiorly by the lower level of hyoid bone, medially by the vertical plane defined by the accessory nerve and laterally by the posterior border of sternocleidomastoid.

LEVEL III middle jugular - bound superiorly by the lower level of hyoid bone, inferiorly by the horizontal plane along the inferior border of the anterior cricoid arch, medially by the lateral border of sternohyoid muscle and laterally by the posterior border of the sternocleidomastoid.

LEVEL IV lower jugular - bound superiorly by the horizontal plane along the inferior border of the cricoid arch, inferiorly by the clavicle, medially by the posterior border of the sternohyoid and laterally by the posterior border of the sternocleidomastoid.

LEVEL Va posterior triangle – bound superiorly by the sternocleidomastoid and the trapezius, inferiorly by the horizontal plane along the inferior border of anterior cricoid arch, medially by the posterior border of sternocleidomastoid and laterally by the anterior border of trapezius.

LEVEL Vb posterior triangle (supraclavicular) – bound superiorly by the horizontal pane along the inferior border of the anterior cricoid arch, inferiorly by the clavicle, medially by the posterior border of sternocleidomastoid and laterally by the anterior border of the trapezius.

LEVEL VI anterior compartment - bound superiorly by the hyoid bone, inferiorly by the sternal notch, medially by the right common carotid artery and laterally by the left common carotid artery.

LEVEL VII superior mediastinal – bound superiorly by the sternal notch, inferiorly by the innominate artery and both medially and laterally by the common carotid artery

MALIGNANT LARYNGEAL TUMOURS

Laryngeal carcinoma is the eleventh-most common form of cancer among men worldwide and is the second-most common malignancy of the head and neck. The primary functions of the larynx involve phonation, respiration, and deglutition but it also contributes to taste and smell by allowing the movement of air over the special sense organs. Thus, loss of laryngeal function affects speech and swallowing and some of the senses that allow us to enjoy the world.

Prevention and early diagnosis of laryngeal carcinoma is the most effective means for increasing cure rates and preserving function. Fortunately, glottic laryngeal carcinoma, tends to be detected at an earlier stage than tumors located at other subsites of the head and neck. Nevertheless, the symptoms of laryngeal carcinoma can also be nonspecific and may result in a delay of diagnosis. In general, hoarseness lasting longer than 3 weeks or odynophagia or dysphagia lasting longer than 6 weeks should warrant a referral to an otolaryngologist especially for patients older than 40 years with an extensive smoking or drinking history. Associated otalgia, stridor and weight loss should also be red flags to completely rule out a malignant process.

The preoperative evaluation of patients with laryngeal carcinoma begins with a thorough history and physical examination in the office with subsequent endoscopy and surgical biopsy for tissue diagnosis. Once a diagnosis is made, the physician and patient must together formulate an individualized plan based on the tumor characteristics as well as the patient's wishes.

Laryngeal cancer is more common in men with an incidence of 6.2 per 100000 compared to women with an incidence of 1.3 per 100000 females. It is more common after the age of 40 years. Some areas of the world has high incidence (>10/100000) such as Brazil, Hongkong, USA, India. Low incidence areas include(<2/100000) Japan, Norway, Sweden.

Within the larynx the tumour may arise from the glottis, supraglottis or subglottis. Study conducted amongst the western population shows increased incidence of glottic tumours, whereas the asian population has supraglottic tumours in common. The risk factors includes geography, lower socioeconomic strata, smoking, alcohol, radiation, occupational exposure of asbestosis, mustard gas, formaldehyde.

The initial assessment of patients with laryngeal cancer begins with a thorough history. Symptoms include hoarseness, dysphagia, chronic sore throat, neck mass and referred otalgia. The most common presentations are the gradual onset of chronic hoarseness (for glottic

tumors), sore throat, dysphagia, or otalgia (for supraglottic tumors). Subglottic tumors may have minimal symptoms.

Patients with reflux laryngitis, heavy smokers, and alcohol consumers often have an edematous, irritated larynx and may not appreciate a voice change from their usual chronic hoarseness. Supraglottic tumors have a more subtle symptoms. Dysphagia and odynophagia are more common symptoms and suggest extension to the tongue base or to the hypopharynx. Shortness of breath can be due to vocal cord fixation or ball-valve effect from a large supraglottic lesion. Supraglottic tumors present in association with evidence of cervical metastasis.The risk of occult or evident regional tumor spread from supraglottic carcinoma with T1, T2, T3, and T4 tumors is 20%, 40%, 60%, and 80%, respectively.

Glottic tumors present early with voice changes. Dysphagia and weight loss occur later.The lack of lymphatic drainage within the lamina propria of the vocal folds lymph node spread is uncommon. A mass in the neck represents direct extralaryngeal spread through the laryngeal cartilage or cervical metastasis and in either case suggests advanced disease. Difficulty in breathing (dyspnea or stridor) can occur later with glottic tumors and signifies bulky disease, vocal cord fixation, or subglottic extension with airway narrowing. Hemoptysis can occur if pulmonary involvement is present, but this may be a dangerous sign if the

primary laryngeal tumor is ulcerated. Genetics plays a role in the development of laryngeal cancer, and this may best be understood as an increased susceptibility to the environmental carcinogens. Finally, the presence of a squamous cell cancer of the head and neck places an individual at risk of approximately a 15% to 20% chance of developing a second primary within 5 years of initial diagnosis.

The patient's general condition, social and economic situation, plays a role in making treatment decisions. A general medical assessment, consists of complete haemogram, blood sugars, hypertension, renal parameters, pulmonary status, coronary artery disease. If previous myocardial infarction or carotid artery disease further work-up needed before definitive therapy for laryngeal Cancer.

Assessment of a patient's motor ability predicts success with a trachea - esophageal Voice prosthesis. Physical examination is a must due to high incidence of synchronous (5% to 8%) and metachronous primary lesions, a thorough head and neck examination is carried. Visual inspection should be combined with digital palpation of the tongue base, particularly if superior extension of a supraglottic tumor is suspected. If no evidence of a second primary is found elsewhere in the oral cavity, oropharynx, or hypopharynx, direct examination of the larynx can be done.

The neck is examined for evidence of metastatic disease or direct extralaryngeal extension. Bimanual palpation of all regions above the clavicles, including the central compartment of the neck, is to be done. Loss of the normal laryngeal crepitus during side-to-side manipulation of laryngeal cartilages can signify postcricoid involvement of the primary tumor and may be associated with complaints of dysphagia by the patient. Careful assessment of the thyrohyoid membrane, thyroid cartilage and cricothryoid membrane, overlying soft tissue of the laryngeal complex may provide a more clinically useful assessment of the tumor than any advanced imaging analysis. Laryngeal carcinoma most frequently metastasizes to nodal levels II, III, and IV in the neck. The term ‘‘transglottic’’ is given to a tumor from one laryngeal region that crosses the glottis in continuity with another region. Such a lesion must have invaded the paraglottic space and, for this reason, is associated with a high incidence of neck disease and cartilage invasion.

Initial laryngeal evaluation is made by indirect mirror laryngoscopy. Mirror laryngoscopy was first performed by the singing teacher Manuel Garcia in 1894 with a dental mirror and sunlight. This technique affords a clear, two-dimensional image of the larynx. It is limited by certain factors There is a small subset of patients who do not tolerate complete mirror laryngoscopy. In addition, because the protruding tongue is secured with a gloved finger, the larynx may be seen

in a non physiologic state. Flexible fiber-optic laryngoscopy, combined with stroboscopy, gives a closer look at individual areas and allows for video and photographic documentation of any visible pathology in a physiologic setting. Patients can visualize the lesion on the monitor and it may facilitate their understanding of the disease process. The vocal fold movement and movement of the cricoarytenoid joint can be appreciate. Making the patient to cough lightly, may help to elucidate arytenoids mobility.

Rigid 70^0 or 90^0 endoscopes provide a similar view as with indirect mirror laryngoscopy but with added magnification.

Combined with a stroboscopic light source allows for visualization of the mucosal wave and aid in detecting early glottis lesions by changes in mucosal wave dynamics. Extrusion of the tongue required for rigid endoscopy may place the larynx in a slight nonphysiologic position. The strong gag reflex can be solved with practical experience.

Imaging

Imaging is a critical adjunct to the physical examination, and not a substitute. It can be useful for staging. Imaging can provide valuable assessment of anatomic spaces difficult to objectively assess on physical examination or by endoscopy (pre-epiglottic space, paraglottic space, thyroid cartilage invasion). Imaging may also supplement clinical examination information as to the tumor volume and extralaryngeal

extension. Furthermore, imaging can assist with surgical planning and in the determination of whether or not the primary tumor is amenable to resection.

CT and MRI

Computed tomography (CT) or magnetic resonance imaging (MRI) of the head and neck from the skull base to clavicles are both appropriate initial imaging choices. T2-weighted MRI allows for excellent visualization of submucosal spread into the pre-epiglottic and paraglottic space. Paraglottic space involvement increases the risk of neck disease and cartilage invasion. The minor cartilaginous invasion (staged as T3) is now being treated with chemoradiation protocols, through-and-through cartilage invasion is an indication for surgical resection of the larynx because of the high incidence of chondronecrosis after radiation.

The role of fused modality positron emission tomography / computed tomography (PET/CT) in diagnosing and staging patients with primary metastasis and recurrent head and neck cancer is a new concept. PET/CT combines the detailed anatomic information of the CT with PET's ability to detect subtle lesions by alterations in metabolic activity. By detection of synchronous or metastatic disease, information provided by PET/CT led to changes in planned procedures or sparing of previously planned procedure and also helped guide biopsy to metabolically active areas in the larynx.

Metastatic work-up

In the laryngeal carcinoma ,distant spread of tumor is rare .Routine work-up for metastatic disease in laryngeal carcinoma is institution dependent. Lung, followed by liver, are the most frequent sites of distant metastasis and as such chest radiographs and laboratory investigation of liver function tests (LFTs) with possible liver ultrasonography has been the minimal standard at several institutions. Patients with an abnormal chest x-ray (CXR) and those with advanced disease or high clinical suspicion may warrant CT of the chest and abdomen.

Recently, the practice of obtaining a pretreatment PET/CT on patients with advanced stage laryngeal carcinomas has become more frequent.

Direct laryngoscopy and biopsy

Initial assessment of the laryngeal structures is done using the anterior commissure, or Holinger, rigid laryngoscopy. This instrument is useful for navigating through and around tumor growth. Once this endoscope has been used to fully visualize the relevant structures and tumor extent, the larger Dedo laryngoscope may be used. It is must to concentrate on the laryngeal extent of the lesion after full assessment for synchronous or metachronous lesions is completed. The site, size and extent of the tumour, vocal cord mobility is determined. Full panendoscopy should include the hypopharynx (including both piriform

sinuses and the postcricoid area) and complete esophagoscopy. The larynx is brought into complete view, and a photograph or drawing is made of the extent of the lesion. A suspension apparatus is useful at this point, particularly if two instruments—one to provide exposure and one to perform a biopsy—are needed. The rigid telescopes may be helpful in operative laryngoscopy evaluation of laryngeal tumors, but this technique is greatly aided by securing the laryngoscope with a suspension bar. The rigid 0^0, 30^0, 70^0 telescopes can be used during staging. These telescopes provide superior visualization of the subglottis, the anterior commissure, and the ventricle, which are critical areas that may be difficult to assess adequately.

HISTOLOGICAL CONFIRMATION OF LARYNGEAL CANCER

Currently it is normal to get the specimen for histopathological examination by direct laryngoscopy and carried under general anaesthesia, which also allows careful direct examination of the tumour. Biopsy material should include an adequate amount of the tissue from ulcerated areas and elsewhere if practicable. Biopsy should be deep and reach necrotic tumor, viable tumor and stroma or muscle to prove the presence of an invasive squamous cell carcinoma.

Biopsies should be obtained from the obvious tumor and any additional suspicious areas, particularly on contralateral vocal cord,anterior commissure and interarytenoid area. This is needed especially if a conservation laryngeal procedure is planned.Squamous cell carcinoma accounts for 95% of primary laryngeal cancers. Large lesions should be adequately sampled with the biopsy forceps to measure invasion below the basement membrane. The biopsy material is important on three grounds. First, it helps in the definitive diagnosis of malignancy. It is difficult to exclude or diagnose malignancy just by inspection. Sometimes even benign looking lesions may have malignancy underneath

them. Secondly, identification of the type of the tumour. Thirdly it helps to know the degree of differentiation. Sometimes difficulties in diagnosis of a malignancy do arise if there is negative biopsy. In such cases the biopsy to be repeated. Keratotic lesions yields a nonmalignant histopathological diagnosis and often difficult to decide whether such lesions will go for malignancy. Miscellaneous condition such as chronic laryngitis, tuberculosis, fungal laryngitis, wegeners granulomatosis, syphilis gives rise to diagnostic confusion. The histopathological classification can be done according to Broders Classification.

The endolaryngeal lesions are well or moderately differentiated. Histologic aggressiveness is suggested by a poorly differentiated lesion. This is not usually found to be predictive of biologic tumor behavior. Better predictors are deep and extensive invasion (T stage), microvascular invasion, and extracapsular spread from involved lymph nodes.

Verrucous carcinoma represents an unusual presentation of a well differentiated squamous cell carcinoma. This tumor, first described in larynx in 1966 by Krause and Perez-Mesa, is known for its locally aggressive behavior and is slow growing. Its gross presentation is that of a warty, keratotic tumor, most often in the oral cavity. Its second most common site is the larynx, where it is always endolaryngeal (glottic or supraglottic). This tumor accounts for 1% to 2% of laryngeal carcinomas. The lesion is usually exophytic, fungating, and broad based.

Whereas the gross appearance is that of neoplastic disease, typically the tumor contains hyperkeratotic squamous epithelium with minimal cytologic atypia. The tumor margins seem to be pushing rather than infiltrating.

Basaloid squamous carcinoma is a variant of squamous cell carcinoma that is infrequent and unique. The most comon site is hypopharynx, base of tongue, and supraglottic larynx. This to be distinguished from small cell carcinoma, undifferentiated carcinoma, and adenoid cystic carcinoma. This type of laryngeal cancer seems to be more aggressive and has worse prognosis than squamous cell carcinoma. Preoperative metastatic analysis is necessary with this tumor because of its predilection to distant disease.

Non-squamous tumors of the larynx account for less than 5% of laryngeal tumors. They typically arise from salivary gland tissue or neuroendocrine cells within the larynx. Among salivary gland tumors, adenocarcinoma is the most common, followed by adenoid cystic carcinoma and mucoepidermoid carcinoma. The most common sarcoma of the larynx is the chondrosarcoma, and the common site is cricoid cartilage, particularly the posterior lamina. Tumors derived from neural cells include paragangliomas, carcinoid tumors, and oat cell tumors.

Staging

The staging of laryngeal cancer used in most of the world was published by the America Joint Committee on Cancer Staging (AJCC) . The AJCC was formed in 1959 and announced its recommendations in 1977. Since then they have periodically updated the staging system. In 1987 the AJCC merged with the European Union against Cancer, with its most recently updated staging system announced in May 2002 (6th edition). The changes in the most recent edition of the AJCC staging of laryngeal cancer are minimal. Tumors staged T4 are to be divided into resectable (T4a) and unresectable (T4b). N staging remains unchanged except for the addition of the descriptors U for nodal metastasis to the upper neck and L for nodal metastasis to the lower neck. Staging follows a similar pattern: Stage IVa disease is advanced resectable disease, stage IVb is defined as advanced unresectable disease, and stage IVc is defined as advanced distant metastatic disease.

An ideal staging system has three purposes. First, it provides the clinician with prognostic information for the patient (predictive power). Second, it allows for standardization of reporting methods to compare similar patients treated with different modalities. Third, it helps to guide the clinician toward a treatment plan.

Although the AJCC succeeds in fulfilling the first two purposes, it does not help stratify patients into treatment groups (ie, organ

preservation therapy versus total laryngectomy). For example, some patients with stage IV laryngeal (glottic) cancer are still candidates for organ-preservation surgery, whereas some patients with stage II laryngeal cancer (subglottic) are not. Current staging is dependant on office assessment, intra operative assessment, and radiologic evaluation.

TNM staging of laryngeal carcinoma

Primary tumor (T)

TX: Primary tumor cannot be assessed

T0: No evidence of primary tumor

Tis: Carcinoma in situ

Supraglottis

T1: Tumor limited to one subsite* of supraglottis with normal vocal cord mobility

T2: Tumor invades mucosa of more than one adjacent subsite* of supraglottis or glottis or region outside the supraglottis (eg, mucosa of base of tongue, vallecula, medial wall of pyriform sinus) without fixation of the larynx

T3: Tumor limited to larynx with vocal cord fixation or invading any of the following: postcricoid area, pre-epiglottic tissues

T4: Tumor invades through the thyroid cartilage or extends into soft tissues of the neck, thyroid, or esophagus

*Subsites include the following: ventricular bands (false cords), arytenoids, suprahyoid epiglottis, infrahyoid epiglottis, aryepiglottic folds (laryngeal aspect)

Glottis

T1 : Tumor limited to vocal cord(s) (may involve anterior or posteriorcommissure) with normal mobility

T1a : Tumor limited to one vocal cord

T1b : Tumor involves both vocal cords

T2 : Tumor extends to supraglottis or subglottis, or with impaired vocal cord mobility (T2b)

T3 : Tumor limited to the larynx with vocal cord fixation

T4 : Tumor invades through the thyroid cartilage or to other tissues beyond the larynx (eg, trachea, soft tissues of neck including thyroid and pharynx)

Subglottis

T1 : Tumor limited to the subglottis

T2 : Tumor extends to vocal cord(s) with normal or impaired mobility

T3 : Tumor limited to larynx with vocal cord fixation

T4 : Tumor invades through cricoid or thyroid cartilage and/or extends to other tissues beyond the larynx (eg, trachea, soft tissues of neck, including thyroid, esophagus)

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 or less in greatest Dimension.

N2 : Metastasis in a single ipsilateral lymph node, more than 3 cm but not more than 6 cm in greatest dimension, or in multiple ipsilateral lymph nodes, none more than 6 cm in greatest dimension, or in bilateral or contralateral lymph nodes, none more than 6 cm in greatest dimension

N2a : Metastasis in a single ipsilateral lymph node more than 3 cm but not more than 6 cm in greatest dimension

N2b : Metastasis in multiple ipsilateral lymph nodes, none more than 6 cm in greatest dimension

N2c : Metastasis in bilateral or contralateral lymph nodes, none more than 6 cm in greatest dimension

N3: Metastasis in a lymph node more than 6 cm in greatest dimension

In clinical examination, the size of the nodal mass should be measured, and allowance should be made for intervening soft tissues. Most masses larger than 3 cm in diameter are not single nodes but confluent nodes or tumors in soft tissues of the neck. There are three stages of clinically positive nodes: N1, N2, and N3.

The use of subgroups a, b, and c is not required but is recommended. Midline nodes are considered homolateral nodes

TREATMENT POLICY

Nearly 95% of laryngeal cancers are treatable. Main causes of untreatability is distant metastasis(less than 5%), bad general health, advanced tumour which involve all the subsites of larynx, fixed cord, with bilateral nodes and fixed on one side. In these patients five year survival rate is less than 5%.Hence any radical treatment to these patients is not justified.

Treatment plan may be classified into following groups:

1. CURATIVE INTENT:

Surgery

Radiotherapy (organ preservation with or without chemotherapy)

Surgery with post op radiotherapy or chemoradiotherapy

2. REHABILITATION

3. PALLIATION

General palliative care, nutrition support

Tracheostomy

Palliative surgery

Radiotherapy

Chemotherapy

Radiotherapy and chemotherapy

Treatment by curative intent depends on the age, performance status, treatment option opted by the patient, any previous treatment, patients distance from treatment facility, follow up reliability, imaging availability, skill of the surgeon and pathologist.

The following types of procedures are in use now a days,

1. VERTICAL PARTIAL RESECTION

a. Cordectomy

b. Frontal partial laryngectomy

c. Lateral partial laryngectomy

d. Frontolateral partial laryngectomy

e. Extended frontolateral partial laryngectomy

2. HORIZONTAL PARTIAL RESECTION

a. Epiglottectomy

b. Supraglottic partial laryngectomy

c. Extended supraglottic partial laryngectomy

3. TOTAL RESECTION

a. Total laryngectomy alone

b. Total laryngectomy with partial pharyngectomy or partial glossectomy

The operation of neck dissection can be combined with the above procedures.

TOTAL LARYNGECTOMY:

Billroth of Vienna first removed the larynx in 1873. In those days post op complications like septicemia, spreading cellulitis, mediastinitis, haemorhage, shock were severe and none of the patient survived. Later two stage procedures was introduced. First tracheostomy was done, after this larynx was removed after few weeks. Single stage surgery was proposed by Moure and Portmann in 1921, since then surgeons are following one stage surgery.

The surgery is done under general anaesthesia. The most commonly used incision is Gluck-Sorenson incision. Exposure of larynx is done by dissecting it out from sternomastoids and carotids, tying the vascular bundle. The strap muscle cut low in the neck and contralateral thyroid freed from the specimen. The trachea is divided below the tumour and tracheostome is fashoined. Removal of larynx from above downwards is easy, after cutting the constrictors muscle from the thyroid cartilage, preserving adequate mucosa. Closure of pharynx is in the shape of I or Y with connell suture. A posterior myotomy will avoid segments of spasm. Skin closed in layers over suction drains.

Feeding is done by nasogastric tube and regular tracheostomy care given. The complications include pharyngocutaneous fistula, which is more common in 4 to 10 days post operative days. Others include haemorrhage, wound infection, pulmonary and cerebral embolism,

tracheal crusting, stomal recurrence, thyroid and parathyroid insufficiency Swallowing after total laryngectomy is painful for first few days. Nasogastric tube can be removed on day 6.If complication like fistula occurs then tube left in situ for 10 days or till the fistula is healed well.

With the help of speech therapist functional rehabilitation of the voice to be considered. The patients who underwent total laryngectomy can satisfactorily develop oesophageal speech. This is produced by the subject charging the esophagus with air and using the vibrations at cricopharyngeal level or another level to phonate. Neoglottis can be created for voice production with the help of redundant mucosa from the post cricoid region. Bloom-Singer valve along with neoglottis improves voice production. Electrolarynx can be used for the same purpose.

The main disability after total laryngectomy is loss of normal voice. As there is no regular air passage through the nose, the subject cannot appreciate the smell and taste of food. The patient should avoid swimming. The subject to be careful while taking bath as water should not enter tracheostome. As the chest cannot be fixed due to lack of closure of larynx, strenuous work and lifting heavy weights not possible. Adaptation is good when the age is less than 60 yrs.

MANAGEMENT OF METASTATIC NECK DISEASE

The presence of metastatic neck disease is a bad sign. Careful and effective treatment will provide the cure. Clinical examination remains the first method. Great care should be taken in this analysis. False positive and false negative rates are around 20-30 percent. Some necks are difficult to examine. Retropharyngeal area lympnodes are unable to palpate, unless they are enlarged. Computed tomography have become an important tool in analysis of neck lymph nodes. It is more accurate than the clinical examination. It has a sensitivity of 84% and specificity of 83%.Physical examination has senstivity 74% and specificity of 81%.The detection of malignant lympnodes is based on the fact that cancer invades the lymph node, its size, shape and characteristics change, such that it enlarges. Hence centre goes for necrosis. There is rim of inflammation, which is seen as rim enhancement on CT. The width of non malignany cervical lymphadenopathy is 3 mm up to 3 cm, but nodes greater than 1 cm in size on CT may contain metastatic disease. Overall 80 percent accuracy achieved by considering the nodes greater than 1 cm has

malignancy in it except at level II and high level III where 1.5 cm size is the criteria.

MRI of neck, Ultrasound examination of neck and guided biopsy, radionuclide scan, Positron emission tomography, fine needle aspiration cytology can be used in evaluating the metastatic neck disease.

The treatment option for N0 neck can be elective surgery, elective radiotherapy, elective neck investigation(CT or MRI). Wait and see policy can be adopted. Elective neck treatment prevents cancer related deaths due vascular or lymphatic spread and produce distant metastasis. But there is no evidence to suggest that any decrease in survival by delaying neck treatment until nodes become palpable clinically palpable. For laryngeal malignancy N0 neck incidence is low. It is widely stated in the literature that external beam radiation of approximately 40-50 Gy to the clinically NO neck will control the occult metastasis in 90 % to 95 % cases. When the primary tumour is being treated by surgery then neck to be treated with elective surgery. If the primary is treated by radiotherapy then the same modality can be applied for neck also.

The NI neck can be treated surgically, because survival rate is good and less extra nodal spread. In this case conservation neck surgery is feasible. In palpable neck disease all five levels will be involved and should be dissected. The gold standard surgery is modified radical neck dissection. Morbidity of neck dissection arises due to level V dissection.

The role of radiotherapy is controversial and less preferred to surgery in NI neck.

The N2a and N2b neck represents advanced disease should be treated with radical surgery. Modified radical neck dissection can be performed. Bilateral and contralateral nodes that is N2c neck is less common. Over all occurrence is 5% of head and neck cancers. The prognosis depends on size, number of lymph nodes, presence or absence of extra capsular spread within the neck and not by the laterality.

The presence of massive nodes is less common occurrence. Only 5% patients present with N3 neck .Majority of the patients are incurable. May be treated with neck dissection or radiotherapy.

The contraindications for neck dissections are in whom primary tumour is untreatable. Patients who are unfit for surgery due to medical ailments, inoperable neck disease and distant metastasis. Locoregional control of malignancy is good with chemotherapy and radiotherapy used in different combination**.**

NECK DISSECTIONS

Neck dissection or cervical lymphadenectomy is the systematic removal of lymph nodes with their surrounding fibrofatty tissue from the various compartments of neck. In 1906 Crile described the radical neck dissection. Hayes Martin popularized it. This operation is the gold standard by which other operations are judged.

The types of neck dissections are:

1. Radical neck dissection
2. Modified radical neck dissection
3. Selective neck dissection
    a. Supraomohyoid
    b. Posterolateral
    c. Lateral
    d. Anterior compartment

when the alteration involves the preservation of one or more non-lymphatic structure relative to radical neck dissection it is called modified radical neck dissection. If alteration involves preservation of one or more lymphnode groups relative to radical neck dissection it is called selective neck dissection. If the modification involves additional lymph nodes or

non-lymphatic structure relative to radical neck dissection is called extended radical neck dissection (Eg. Carotid artery, paraspinal muscles, hypoglossal nerve, vagus nerve, superior mediastinal lymph nodes). En block neck dissection is removal of the primary in continuity with the neck dissection, as it avoids harbouring of tumour cells in the intervening tissues.

Neck incisions for unilateral dissections may be trifurcate or non-trifurcate. The trifurcate incisions are Hayes Martin, Slaughter, Kocher, Schobinger, Crile, Conley. The non –trifurcate incisions are Mac-Fee, J shaped, Hockey stick, Inverted hockey stick, Modified apron, Lahey, Modified slaughter.

For bilateral neck dissection incisions used are Bilateral hockey stick, Apron, Modified Apron, Bilateral inverted hockey stick, Modified Gluck Sorenson incisions.

RADICAL NECK DISSECTION

The boundaries of dissection involves superiorly inferior border of the mandible, Inferiorly clavicle, Posteriorly anterior border of trapezius, Anteriorly lateral border of sternohyoid muscle, hyoid bone, contralateral anterior belly of digastric muscle. The structures removed are Level I to V lymph nodes, spinal accessory nerve, Internal jugular vein and external jugular vein, Sternocleidomastoid muscle, omohyoid muscle, cervical plexus (sensory branches). Submandibular salivary gland and tail of the parotid gland. The indications are;

1. Clinicaly positive lymph nodes

(a) Previous radiotherapy given (b) Previous neck dissection done.

2. Fixed neck mass which becomes mobile with radiotherapy or chemotherapy.

3. Nodal involvement other than first echelon group.

The contraindications are;

1. Un controlled cancer of the primary site.

2. Evidence of distant metastasis

3. Unfit for major surgery.

4. Mass fixed to cervical spine or brachial plexus or trachea

5. Unresectable nodes unchanged by radiotherapy or chemotherapy

MODIFIED RADICAL NECK DISSECTION

The lymph nodes of the neck lie within the specific aponeurotic planes which can be carefully dissected with preservation of important structure (Spinal accessory nerve, sternocleidomastoid, internal jugular vein).Boundaries are similar to radical neck dissection.

The indications are

1. Elective neck dissection when there is >25 % risk of occult positive nodes and surgery is performed for the primary cancer.

2. NI neck when surgery is to be followed by radiotherapy.

3. For the lesser involved side when bilateral neck dissection is done.

Contraindications are similar to that of radical neck dissection.

The types of modified neck dissection are

Type I : The Spinal accessory nerve is preserved. Sternocleidomastoid and internal jugular vein sacrificed.

Type II : The Spinal accessory nerve and internal jugular vein preserved. Sternocleidomastoid sacrificed.

Type III : Boccas functional neck dissection (The Spinal accessory nerve, sternocleidomastoid, internal jugular vein preserved)

In all the three types, lympnodes level I to V removed.

SELECTIVE NECK DISSECTIONS

The suprahyoid neck disection done in N0 and NI neck located in first echelon of lymphatic drainage. Boundaries of dissection are superiorly inferior border of mandible , inferiorly junction of omohyoid with IJV. Anteriorly lateral border of omohyoid and anterior belly of opposite digastric. Posteriorly posterior border of sternocleidomastoid and cutaneous branches of cervical plexus. Structures removed are lymph nodes level I, II, III. In posterolateral neck dissection structures removed are suboccipital lymph nodes, retroauricular lymph nodes, lymph nodes level II, III, IV and V. In anterior compartment neck dissection boundaries are superiorly hyoid bone, inferiorly suprasternal notch and lateraly media border of carotid sheath. Structures removed are level VI lymph node group (Pretracheal, Paratracheal, Precricoid, Perithyroidal) and ipsilateral thyroid lobe. In lateral neck dissection boudaries are posterior triangle behind submandibular triangle and midline vertical structures anteriorly. Structures removed are levels II, III and IV.

COMPLICATIONS

The most common perioperative complications(within 24 hrs) are haemorhage which can be primary and secondary, shock, airway obstruction, pneumothorax, air embolism, carotid artery rupture, cervical sympathetic chain damage, subcutaneous emphysema and cervical spine injury.

The post operative complications (After 24 hrs) are air leak, secondary hemorrhage, wound infection, fistula formation, flap necrosis, wound scarring, peripheral motor nerve damage, internal jugular vein rupture, fluid and electrolyte imbalance, pulmonary complications like atelectasis, bronchopneumonia.

RADIOTHERAPY

The radiotherapy is defined as the application of radiation to gain the therapeutic success. Most commonly used is external beam radiotherapy, also called teletherapy. In this beam of radiation is directed to the patient from a machine placed outside the body of patient. In brachytherapy the radioactive material can be introduced into the tumour.

The linear accelerator is the standard megavoltage treatment unit used nowadays. The linear accelerator has, a stream of electrons produced by a filament in an electrically charged field is which accelerated through a series of wave guides in combination with a radiofrequency pulse. This electron beam can be used for treatment or can fall on a target to give a beam of maximum energy between 4 and 20 MV.

To have accuracy of delivery of the external beam radiotherapy the patient has to be immobilized perfectly. This can be achieved by moulded thermoplastic shells which covers head and neck region. These are placed on a fixed frame on treatment couch. Reference marks situated outside the shell. This allows treatment accuracy for 1-2mm.

The effects of radiation occur in the non-critical part of cell but, some can occur in the DNA. Ionising events results in single strand breakage that can be repairable, where as double strand break leads to worse repair. Hence cell death occurs within next cell divisions. Cell repair is less beneficial in tumour cells compared to that of normal cells. Nearly 95% of the sublethal damage repair to the cell is completed in 6 hours. Tumour cells which are more than 100 to 150 microns distance from the nearest blood vessel sufferes increasing hypoxia and necrosis. The anoxic cells are 2.5 to 3.0 times less sensitive to radiation therapy. Cellular hypoxia increases the radioresistant. Bioreductive nitroimidazole drugs such as misonidazole, etanidazole, nimorazole can mimic oxygen and grater tissue perfusion and can be used as hypoxic cell sensitizers.

RADIOTHERAPY FRACTIONATION

The correct total dose is a balance between the tumour cell kill and effect of early and late side effects. There are two types of fractionation one conventional and the other is unconventional which includes hypofractionation, hyperfractionation and accelerated fractionation .

The conventional fractionation defined as to the use of individual treatments(fractions) of 1.8 -2 gray which is given daily for five days a

week. Curative doasge is generaly of the range 66-70 gray given in 33-35 fractions over 6.5 to 7 weeks. In post operative radiotherapy the dosage is 10% lower in the range og 60 –64 gray given in 30 -32 fractions during 6-6.5 weeks.

The hypofractionation defined as fewer fractions and larger dose per fraction. This shorter course decreases the chance of tumour repopulation and decreased damaged DNA is repaired. This has property of late complications. The courses of treatment which uses larger fraction sizes than 2 Gy defined as hypofractionation. The hyperfractionation consists of use of smaller fractions(<1.8Gy). The use of smaller fractions lessens the risk of late adverse effects. Increase in the overall time tends to obviate the efficacy of the treatment. The regimens delivering 50-60 fractions throws a heavy burden on treatment.

In accelerated fractionation regimes is the overall treatment time is shortened. The usefulness is obtained by combining acceleration with hyperfractionation and treating two or three times per day and allowing for six hour gap between fractions. In CHART regime (Continous hyperfractionated accelerated radiotherapy) 1.5 Gy per fraction is utilised three times daily and continously for 12 days and a total of 54 Gy, without any weekend break.

The factors determining the effectiveness of radiotherapy on tumour control are the total dose which means increasing the dose from

66 to 70Gy improves local control by 5%.Concurrent platinum based chemotherapy increases overall benefit. The use of cetuximab helps for better tumour control. The cetuximab is a monoclonal antibody against the ligand domain of the EGF receptor. The taxanes, taxotere (docetaxel) and taxol increases the survival rate. Delay in the starting treatment, treatment interruptions, anaemia, smoking influence the outcome of the treatment regime.

IMAGE –MODULATED RADIOTHERAPY(IMRT)

In conventional radiotherapy we use only two or three fields which means a significant dose is absorbed by the tissues between skin and the target volume. If many fields are used relative contribution of each field lessens and absolute dose in front and behind the target is decreased. Relative intensity of individual part of each beam can be changed with the help of multileaf collimators. This constitutes IMRT .This helps in even coverage of tissue volume and adjacent normal tissues receive less dosage. Often seven or more multiple beams are used. Hence larger volume of normal tissue gets low-dose radiation. This increases the risk of second malignancy.

IMAGE-GUIDED RADIOTHERAPY(IGRT)

Effects to decrease the movement of the patient during treatment is very important to get the good result. This is greatly achieved by IGRT. This involves fitting the linear accelerator with CT capability so that patient position can be confirmed prior to the treatment. Further linear accelerator is programmed to carry the necessary shifts in the patient position during the treatment.

TREATMENT PLANNING FOR LARYNGEAL CANCER

The treatment for glottic tumours TI and TII, N0 is radical radiotherapy. Surgery can be kept as a salvage after radiotherapy. A lateral parallel opposed field which is used, with 5 cm(TI) or 6cm(TII) square fields which is centered on the vocal cord. This is one cm below the thyroid promontory and anterior to lower border of C5.For TI extends from the lower border of hyoid bone to lower border of cricoid cartilage. Usually 10 to 20 degree wedges can be used as missing tissue compensators.

For TII tumours extending to the supraglottic or subglottic field size can be extended. In cases of subglottic extension, paraesophageal paratracheal lymphnodes are also included in the field. If anterior commissure is involved radiation dose to neck to be increased.

Dose prescription, depends on field size,

1.<36 cm2, 50 Gy in 16 fractions should be treated on daily basis in five fractions a week.

2.36-42 cm2, 55 Gy in 20 fractions should be treated on daily basis in five fractions a week.

3.>42 cm2, 66-64Gy in 2 Gy per fraction, treated on daily basis, five fractions a week.

In the supraglottic growth radical radiotherapy is used in early tumours. This tumour has high incidence of occult lymphatic spread in levels I and II. Hence patients need the elective nodal irradiation to lymph nodes of these levels. This can be obtained by two phase technique. In phase I which includes the primary tumour, the entire larynx, pre epiglottic space and the cervical lymph nodes on both sides in levels Ib, II and III in front of the spinal cord. The phase II included are the primary tumour and larynx only. The parallel opposed wedged fields can be used in both of these cases. The total dose given is 66-70Gy in 2 Gy for each fraction, treating everyday, for five fractions a

week, to macroscopic disease and 44-50 Gy in 2 Gy for each fraction, treating daily for five fractions a week, to the microscopic pathology.

The subglottic growths usually present as locally advanced disease which requires surgery and followed by adjuvant radiotherapy. The enlarged paratracheal lymphnodes is observed in 50% of cases. The radiation port spreads from the thyroid cartilage to the level of mid trachea. The dose prescription is of 66 -70 Gy in 2 Gy for each fraction, treating every day , totally five fractions a week.

In the locally later stages of the disease of larynx, stage III and IV of glottis, supraglottis and subglottis can be effectively treated by chemoradiation as an alternate to the surgery as a mode of therapy .The target volume involved in this are the larynx, pre-epiglottic space, involved metastatic lymphnodes and all the lympnodes in draining area which may not be enlarged. In phase I the primary tumour, the entire larynx, pre-epiglottic space and bilateral cervical lymph nodes levels Ib to V included in the radiation port. In phase II therapy, the level II and level V group of lymph nodes are included. The dose is of 66-70Gy in 2 Gy for each fraction, treating every day, for five fractions a week, to macroscopic pathology and 44-50 Gy in 2 Gy for each fraction, treating daily, five fractions a week, to microscopic pathology.

The post op radiotherapy depends on several pathological factors. Presence of extracapsular spread of the nodal disease is considered as

high risk disease. The factors which determine the post op radiotherapy are excision margins less than 5 mm, stage III and stage IV, perineural invasion, vascular invasion, poor differentiation, more than four nodes positive, soft tissue invasion dysplasia, poor differentiation or carcinoma in situ at the resection margin. Following laryngectomy in addition to the above factors, presence of transglottic disease is an additional risk factor.

The potential doubling time for squamous cell carcinoma can be as low as less than five days. Accelerated repopulation is observed after surgery due to increase in the inflammation and tumour growth factors. Hence post op radiotherapy should be initiated within six weeks of surgery once healing is completed. The patient who receive post op radiotherapy with extranodal disease should be of 63 Gy in 35 fractions.

ADVERSE EFFECTS OF RADIOTHERAPY

The adverse effects of radiotherapy can be divided into acute and late effects. Acute adverse effects defined as the changes in the tissues or any symptoms noted within 90 days of initiation of the radiotherapy. The commonly used grading system is RTOG acute radiation morbidity scoring system. In this classification acute effects classified from grade0

(No change) to grade 4 (severe change). The acute effects most commonly seen are

1. Mucositis (erythema, fibrinous exudates, membranous)

2. Skin changes (erythema, desquamation, hair loss, hyperpigmentation)

3. Altered taste

4. Xerostomia

5. Swallowing difficulty

6. weight loss

The orofacial tissues affected by radiation are salivary glands, mucus membrane, taste buds, teeth, mandible, skin.

The late adverse effects defined as the changes in the tissues or any symptoms which occur more than three months from the start of radiotherapy. The RTOG/EORTC late radiation morbidity score is used. Mostly late complications develop in first three years. In mucosa we can see non healing ulcers. In teeth and mandible osteoradionecrosis is seen due to decrease in the production of saliva and also change in chemical composition, which is highly acidic. In skin we can see teleangiectasia in an atrophic dermis under thinned epidermis. Fibrosis may follow later. Due to xerostomia there may be difficulty in swallowing, chewing .Cervical spine radiation exposure may lead to L'hermitte syndrome, foot drop, spasticity, weakness, hemiparesis, brown - sequard syndrome.

CHEMORADIATION THERAPY

The most commonly used chemoradiation therapy regimes in the treatment of squamous cell carcinoma is formulated emperically. The drugs which act against tumour cells was selected and combined with the radiotherapy and their administration schedule was selected for safety. Different chemotherapy schedules are

1. ADJUNCTIVE CHEMOTHERAPY: This is the use of cytotoxic agents with intent to improve survival before, during or after standard local treatment by surgery, radiotherapy or both.

2. INDUCTION CHEMOTHERAPY (Neoadjunctive, upfront, pre-emptive chemotherapy) : This is the use of cytotoxic agents before going to standard treatment with the intention of improving survival.

3. CONCURRENT CHEMOTHERAPY (Concomitant chemotherapy) : In this the use of cytotoxic agents with the intention of improving survival during a course of radiotherapy. Synchronous chemotherapy or radiotherapy may precede or follow surgery or can be regarded as definitive treatment.

4. ADJUVANT CHEMOTHERAPY(Maintainance chemotherapy) : In this use of cytotoxic agents following definitive local treatment either surgery or radiotherapy to improve survival

5. PALLIATIVE CHEMOTHERAPY : The use of cytotoxics usually single agent, in an attempt to relieve symptoms in an incurable disease. This is administered in patients with advanced unresectable tumours. The mechanism of action of combined chemotherapy and radiotherapy is, the chemotherapy enhances the biological response of cell to radiation by DNA damage and cell cycle effect. Helps to overcome the tumour resistance to radiotherapy like hypoxia, tumour repopulation.

CISPLATIN

It is potent radio sensitizer and used past many years in the treatment of squamous cell carcinoma of head and neck along with the radiotherapy. It is an inorganic heavy metal coordination complex. Following single IV administration, 90% of the drug is attached to the proteins. It gets concentrated mainly in liver, kidneys and intestines. The recommended dose is 40 mg/m2 weekly or 100mg/m2 in divided doses every three weeks as a single intravenous dose. Mechanism of action is inhibition of DNA synthesis, inhibition of transcription elongation by DNA, inhibition of repair of lethal damage, radio sensitization of hypoxic cells.

The toxicities of cisplatin are nephrotoxicity, ototoxicity, haematological toxicity, gastrointestinal toxicity and electrolyte disturbance. Less common are nuerotoxicity, occular toxicity, anaphylactic reactions, alopecia.

BIOLOGY OF LARYNGEAL CANCER

ENVIRONMENTAL PROMOTERS OF CARCINOGENESIS

1. TOBACCO :

Cigarette smoking acts independently and also synergisticaly with alcohol in causation of laryngeal cancer. Relative risk of developing laryngeal cancer is 2.4% for smokers who smoke <7 per day to 16.4% for smokers who smoke >25 per day. Cessation of smoking decreases risk by 70% after 10 yrs. The burning of tar gives off a methyl cholanthrene and benzathrene which are broken down by aryl hydrocarbon hydroxylase to carcinogenic epoxides which bind to DNA. Tobacco products and betal chronicaly irritate the squamous epithelium to produce metaplasia.

2. ALCOHOL :

Combined use of alcohol and tobacco increases the risk by 50% over the estimated risk, if these factors were considered additive. Dark liquors (whisky, rum) have greater organic compounds then light liquors (vodka, gin). The risk increases with dark alcohol intake.

3. VITAMINES AND MINERALS DEFICIENCY :

Several study associate high fruits and vegetables intake decreases the risk of laryngeal cancer. This is due to the intake of antioxidants or free radical scavenging Vit A,C,E.

4. OCCUPATIONAL FACTORS :

Nickel, mustard gas, asbestos, sulphuric and hydrochloric acid exposure is associated with laryngeal cancer. Car mechanics, battery plant workers have increased risk.

5. INFECTION :

Human papilloma virus may play a role in laryngeal cancer.

6. GASTROESOPHAGEAL REFLUX DISEASE :

7. RADIATION EXPOSURE :

8. IMMUNO SUPPRESSION :

Laryngeal cancers are more common in immuno suppresed due to HIV or organ transplantation.

MOLECULAR GENETICS OF LARYNGEAL CANCER

Laryngeal cancer is due to random accumulation of genetic alterations. This is critical for cancer cell survival, which are selected in a darwanion manner. Critical molecular alterations for cancer development activate genes that promote oncogenesis or inactivate genes that prevent oncogenesis. The glutathione S-transferases are responsible for detoxification of wide range of xenobiotics. P53 tumour suppressor gene mutation seen in cigarette smoking. IL-4 receptors are increased in smokers. Protooncogene bcl-2 acts by inhibiting apoptosis. Consistent amplification of 11q13 protooncogene is seen in cancer larynx. The ras oncogenes family mutation is seen in tobacco and betel nut users.

Over expression of growth factors such as epidermal growth factor, platelet derived growth factor, fibroblast growth factor ,vascular endothelial growth factor and transforming growth factor alfa is seen.

The E6 and E7 regions of high risk HPV types (6,8,33) inativates the tumour suppressor gene products p53 and prb potentiating neoplastic change and cell immortalization.

The extracellular matrix disruption is needed for cancer spread. Degradation of this matrix is by matrix metallo proteinases and tissue inhibitors both of which are produced by tumour and imbalance of which leads to proteolysis.

PATHOLOGY OF LARYNGEAL CANCER

Squamous cell carcinoma (SCC) is the most common malignancy of the larynx.

Macroscopy

Laryngeal SCC may present as a flat plaque with a well defined, raised edge, or exhibits a polypoid exophytic appearance, which may relate to prognosis. The surface of th

e tumour is sometimes ulcerated. Tumours, particularly in the supraglottic region, frequently show one or more sinuses, which extend deeply into the growth. Sometimes these are very narrow and slit-like.

Histological examination always shows a malignant squamous epithelial surface lining the whole sinus or slit, often with areas of necrosis and fibrin exudation. These deficiencies of the tumour may be related to ischaemia in parts furthest away from the blood supply. In a small proportion of cases the tumours may show one or more papillary elements. These are most common in the glottic and subglottic regions, but are occasionally found in the supraglottic portion of a laryngeal neoplasm.

MICROSCOPY

Squamous differentiation, is seen as keratinization with variable "pearl" formation, and the invasive growth are needed prerequisite features of SCC. Invasion is determined by disruption of the basement membrane, and extension into the underlying tissue, which is accompanied by the stromal reaction. Angiolymphatic and perineural invasion are extra additional signs of cancer. The tumours of larynx are traditionally divided into well-, moderately-, and poorly differentiated SCC. Well differentiated SCC looks very similar to normal squamous epithelium. Moderately differentiated SCC has distinct nuclear pleomorphism and mitotic activity, which includes abnormal mitoses; there is very less keratinization. In poorly differentiated SCC, immature cells are very high, with many typical and atypical mitoses, and less keratinization. Although keratinization is seen in well- or moderately-differentiated SCC, it should not be taken for consideration as an important histological criterion in classifying SCC. Most SCC are moderately differentiated, so grading by differentiation is of less prognostic value, as compared to pattern of invasion. Tumour growth at the invasive part can show an expansive pattern, an infiltrative pattern, or

both can be seen. Expansive growth pattern is characterised by large tumour islands and well-defined pushing margins and is associated with a better prognosis. Infiltrative growth pattern is characterized by scattered irregular cords or single tumour cells, with very less defined infiltrating margins and is associated with an aggressive course. Invasive SCC is almost always has stromal reaction that consists of desmoplasia with deposition of extracellular matrix and multiplication of myofibroblasts . Neo vascularization is commonly seen.

SUMMARY OF PUBLICATIONS

1. Assessment of Risk Factors in Laryngeal Cancer in India :

A Case – Control study

Umesh Kapil, Preeti Singh, Sudhir Bahadur, Sada Nand Dwivedi.

Asian Pacific Journal of Cancer Prevention Vol 6, 2005

Abstract and Result

In the univariate analysis it is observed a lower consumption of roots and tubers green leaf vegetable other vegetables and fruits, and increased consumption of milk, eggs, meat, tea, alcohol, smoking, consumption of betel leaf along with tobacco as well as a liking for spicy and fried foods emerged as significant positive variables. There was a great difference in the dietary consumption patterns of laryngeal cancer patients and controls, which indicates a role for nutritional factors in the causation of laryngeal cancer in the Indian population.

2. TOPOGRAPHICAL DISTRIBUTION OF LARYNGEAL CARCINOMA – A STUDY OF FIFTY CASES

R. Raychowdhury, M.A. Rashid, L. M. Ghosh

Indian Journal of otolaryngology and head and neck surgery vol.54 Oct-Dec. 2002.

Abstract and Result

The proportion of Head and Neck Carcinoma affecting the larynx also varies – 32.86% in Goa, Daman and Diu (Verma and Sequera, 1979), 21.54% at Chandigarh (Deyasi et al, 1977), 28% at Ajmer (Yadav et al, 1986), 7.33% at Patiala (Gupta et al, 1986). Most international studies describe the glottis as commonest site affected by laryngeal carcinoma, based upon study of several investigators (Lederman, 1952; Powell and Robin, 1983; Thawley, 1991; Sasakii and Carlson, 1993). The senior author (Chakravarty et al, 1992) found Supraglottic cancer more common, as did verma in 1990.

3. A study of patient factors and tumor characteristics in malignancy of larynx: A tertiary care center experience Shelly Chadha, Bulbul Gupta, Shraddha Jaiwani.

Journal of laryngology and voice July-Dec.2011 Vol.1

Abstract and Result

In this group study, the age range was 26-76 years in males and 35-68 years in ladies with an average of 52 and 53 years, respectively. Most males were affected in fifth and sixth decades and females in the sixth decade with male-female ratio of 10:2:1 (7:1 in the developed countries). They pressed more on that 25 percent were affected in the third and fourth decades of life.

4. Occupational causes of laryngeal cancer

JORN OLSEN AND SVEND SABROE

Journal of Epidemiology and Community Health, 1984, 38, 117–121.

Abstract and Result

Increased risk ratios for laryngeal cancer were found for semiskilled and unskilled workers, workers exposed to dust, out of doors workers, drivers, and the people working in the cement industries and port industry. The study hypothesis was that exposure to chromium or nickel is the etiology in the incidence of laryngeal cancer

5. **Laryngeal Cancer in Women: Tobacco, alcohol, Nutritional, and Hormonal Factors Silvano Gallus, Cristina Bosetti, Silvia Franceschi, et al.**

Cancer Epidemiology, Biomarkers Prev 2003: 12:514-517

Abstract and Result

Laryngeal cancer was strongly associated with cigarette smoking (OR = 435.7, 95% CI: 38.2 - 4964.4 for smokers of – 25 cigarettes/day) and alcohol drinking (OR = 4.3, 95% ?CI: 0.8-24.1 for 5 drinks/day). An indirect relation was observed for vegetables (OR = 0.3, 95% CI: 0.1-0.9 for the highest level of consumption), fruit (OR = 0.5, 95% CI: 0.2-1.3), and olive oil (OR = 0.3, 95% CI: 0.1.-0.9). Reproductive and hormonal changes were not consistently associated to laryngeal cancer. This

investigation, was based on a uniquely high number of laryngeal cancers in women, which provided definite evidence that cigarettes smoking is the very high risk factor for laryngeal cancer in women, accounting for 78% of cases in this population. Alcohol and other selected dietary aspects account for – 30% of cases, however menstrual and hormonal factors does not appear to have a consistent role in laryngeal carcinogenesis.

6. Fried foods: a risk factor for laryngeal cancer

C Bosetti, R. Talamini, F.Levi, E.Negri

British Journal of Cancer 2002, 87, 1230 - 1233

Abstract and Result

The role of fried foods on laryngeal cancer risk was studied in a case- control study from Italy and Switzerland on 527 cases and 1297 hospital controls. A significant rise in the risk was found for high consumption of fried meat, fish, eggs and potatoes, with odds ratios of 1.6, 3.1, 1.9 and 1.9. respectively.

7. **The epidemiology of laryngeal cancer in Brazil**

Victor Wunsch Filho

Sao Paulo Medical Journal Vol.122 no.5 2004

Abstract and Result

The city of Sao Paulo has one of the biggest incidences of laryngeal cancer in world and Brazil presents high occurrence, compared

with other Latin American countries. Around 8,000 new cases and 3,000 deaths by laryngeal cancer occur every year in the Brazilian population. Tobacco smoking and alcohol intake, occupational hazards have also been linked with the disease, such as asbestos, strong inorganic acids, cement dust and free crystalline silica. Added to this, salted meat and total fat intake have been linked to high risk of laryngeal cancer. Conversely, several other studies have confirmed the fruits, raw leaf vegetables and the legumes protect against the cancer. Some research is have said a possible association between laryngeal squamous cell carcinoma and human papilloma virus (HPV), but this is not universally accepted. Gastroesophageal reflux disease is weakly, but always correlated with laryngeal cancer. Familial cancer clusters, of head and neck tumors, seem to increase the risk of laryngeal cancer.

8. **Laryngeal Cancer and Gastroesophageal Reflux Disease: A Case-control study**

Michael F. Vaezi, MD, Ph.D., MSept, Mohammed A.

The American Journal of Medicine 2006 119, 768-776

Abstract and Result

A total of 96 cases were matched to 192 controls. On univariable analysis, significant risk factors were current smoking, odds ratio (OR) 5.46 (95% confidence interval ICII.2.59-11.50): alcohol, OR 1.97 (CI.1.19.3.26): and GERD, OR 1.79 (CI. 1.03-3.11). On multivariable

analysis, only smoking OR 2.11. (CI. I.16-3.85), respectively. Smoking and gastroesophageal reflux disease are significant high risk factors for laryngeal cancer and have an independent incremental risk for laryngeal carcinogenesis.

9. **Larynx Preserving Treatments in the Early and Advanced Laryngeal Cancers ; A Retrospective Analysis**

Kamian Shaghayeah, Aghili Mahdf

Journal of Cancer Science & Therapy Vol.2 Issue 1

Abstract and Result

In 147 cases, chemo radiotherapy was given for 61 patients. Twelve cases were excluded from the analysis because of the treatment discontinuity or death. Fifty eight cases were early-staged disease. In median time of follow up (9.9 months), mean overall survival of the patients were 51 months and 37 months in early lesions, and 30 months and 17 months in locally advanced tumors, respectively.

The local control rate was 60% in early-staged disease and 43.5% in locally advanced cancer. The mean total radiation dose given significantly affected the tumor control in chemo radiation group. It implies that the radiotherapy or chemo radiation can be used as an appropriate alternative to the total laryngectomy in the laryngeal cancer patients.

10. Cancer of the larynx in Puerto Rico

Albert Villanueva-Reyes, Ed.D. CCC.S1P

PRHS Vol 27N0.3 September 2008

Abstract and Result

The study showed that the average incidence of laryngeal cancer in Puerto Rico was 3.8 & 100,000 from 1997-1998 and 3.5 & 100.000 from 2001-2002 (-1.07 APC). Of all these cases (n=848) of laryngeal cancer reviewed, 88% were male. Females were more likely to be diagnosed before age 50 than males (p=0.02). In this study, women had twice the increased probability of being alive at the end of the study (OR = 1.97: CI:1:14-3.45). The two most frequent types of single treatments for laryngeal cancer were radiation therapy (39%) and surgery (33%). Cases of laryngeal cancer are decreasing in Puerto Rico. Significant differences by gender were observed, in the stage of the disease at the time of diagnosis. Future still studies on medical treatment modalities that can better preserve vocal function concurrently with voice therapy are recommended for laryngeal cancer patients

MATERIALS AND METHODOLOGY

The patients attending the ENT OPD of GRH., Madurai, falling into the inclusion criteria will be selected for the study. The study subjects will be first administered an informed consent form. After explaining the details of the study to the subjects in detail written consent will be obtained from those who agree to participate in the study. For every individual consenting to participate in the study, a case record form will be filled.

The patients with histopathologically proven laryngeal cancer in the inclusion criteria are considered. The common clinical presentation of patients, age profile, the symptomatology, disease stage, etiological factors, histological profile, family history, occupational history and the treatment modalities offered to them will be studied.

1. The procedures done to examine the larynx are indirect laryngoscopy, videolaryngoscopy, direct laryngoscopy.

2. Radiological investigations include chest X-ray, CT scan neck and chest.

3.Clinical investigations- Complete haemogram and biochemical profile.

4. Histopathological examination of biopsy taken from laryngeal growth.

Inclusion criteria:

1. Proven cases of laryngeal cancer by biopsy and histopathological examination.
2. Patients who have not undergone any treatment for laryngeal cancer
3. Patients who are in good mental health to give reliable answers for the Questionnaire
4. Patients who are willing for the study.

Exclusion criteria :

1. Patients who are on chemotherapy or radiotherapy
2. Patients with recurrent laryngeal cancer
3. Patients who have discontinued the treatment
4. Patients with malignancy in other sites along with laryngeal cancer.

RESULTS AND OBSERVATIONS

1. INCIDENCE :

Our hospital is the highest referral unit in the south Tamil Nadu. It covers a total population of twenty five million. Every year on an average we get an out patient census of about 25, 58, 614 at our Hospital. It is a 2518 bedded hospital. At ENT department the census is 83633 per year. All the newly diagnosed laryngeal cancer patients were included in the study between July 2013 to June 2014.It was found that the incidence was 0.135.The most common cancer at our ENT department was laryngeal cancer followed by hypopharyngeal cancer.

2. AGE AT DIAGNOSIS :

AGE (Yrs)	MALES	FEMALES	TOTAL
31-40	6	-	6
41-50	10	-	10
51-60	35	1	36
61-70	52	3	55
71-80	6	-	6

The peak incidence of laryngeal cancer occurred in the age group of sixth to seventh decade of life.48.67% were in the sixth decade,31.85% were in the fifth decade. The average age at diagnosing the laryngeal cancer was 64years.The males had a mean age of 64.30. and the females had a mean age of 65.75.The youngest patient in our study was a male of age 36 yrs male and the oldest patient was 80 years old male. Our analysis also revealed that men are diagnosed early compared to women. The patients with glottic cancers presented to hospital early compared to that of supraglottic cancers. Amongst the males and females supraglottic growth predominated.

3. SEX :

SEX	NO. OF PATIENTS
MALE	109
FEMALE	4
TOTAL	113

The laryngeal cancer is more common in males 96.46% compared to females 3.53%.Reccurrence was not seen in any of our patients. No deaths were seen amongst females in our study. The general condition was better in females compared to the males. The adverse effects of the chemoradiotherapy was also less prominent in females.

4. **FAMILY HISTORY:**

In our study conducted at our hospital none of them had a family history of laryngeal cancer.

5. **OCCUPATION:**

The common occupations in which the most of our patients involved were agriculture, manual laborers, daily wages laborers, factory workers, masons, carpenters, tailors. Most of the men at the time of diagnosing the laryngeal cancer were not involved in any work due to disease morbidity. All the females were house wives.

6. **SOCIO-ECONOMIC GROUP:**

As our hospital caters to a population which belongs to below poverty line, most of the patients are from low socioeconomic strata. The Kuppusamy classification was used for the same.

7. **DEMOGRAPHIC DISTRIBUTION:**

Most of the patients taking treatment at our hospital are from south tamil nadu which commonly includes the following districts, Madurai, Ramnad, Shivakasi, Theni, Trichy, kanyakumari, Coimbatore. The maximum number of patients are from Madurai district followed by Ramnad. Majority of the patients belonged to rural area.

8**. DIETARY HABITS:**

As diet is an important etiological factor for cancer larynx detailed history was obtained.92% of them were non vegetarians. Almost all

consumed hot, spicy food. The commonly consumed non vegetarian diet was meat followed by chicken, which was baked in reused oil. Amongst the vegetables the most commonly consumed was tubers, roots, less of green leafy vegetables and fruits. Consumption of dairy products was less, compared to hot tea.

9. **OTHER HABITS AND ADDICTIONS:**

HABITS	MALE	FEMALE
SMOKING	113	-
ALCOHOL	113	-
TOBACC0	72	4

(a) **SMOKING:**

In our study, chronic smoker is one who smokes more than one pack per day for the past ten years. All the male patients in our study were smokers. None of the female patients had smoking habit. Most of them smoked for more than 30 years, ranging from twenty to forty years. The average pack-year of smoking was 30.6 years. Beedi smoking was more common compared to cigarette smoking.

(b) **ALCOHOL:**

A patient was considered alcoholic when consumed more than 80 gm of alcohol per day for at least 5 years. All the male patients were alcoholic.

None of the female patients were alcoholic. Hence in our study all the male patients were both smokers and alcoholics.

(c) **TOBACCO CHEWING WITH BETEL LEAF AND BETEL NUT:**

All the female patients were tobacco chewers along with betel leaf and betel nut. Female patients mostly consumed the same for more than 25 years.63.71% of the male patients were tobacco chewers. Male patients consumed for more than thirty years.

10. **CLINICAL PRESENTATION:**

SYMPTOMS	MALES	FEMALES	TOTAL
DIFFICULTY IN BREATHING	38.99%	1.76%	40.76%
VOICE CHANGE	36.27%	0.88%	37.16%
DYSPHAGIA	9.72%	2.65%	12.38%
NECK SWELLING	7.97%	1.76%	9.73%

A. **Difficulty In Breathing :** The most common symptom with which patients presented to us was difficulty in breathing, stridor. 40.76% of the patient presented with stridor. Amongst them were 38.99% males and 1.76% were females.

B. **Change of voice :** Next common symptom of presentation was change of voice. This was seen in 37.16% of the patients. Most of them were males.0.88% females and 36.27% were males.

C. Dysphagia : 12.38% patients had dysphagia. This was mostly seen in supraglottic growth rather than the glottic growth

D. Swelling in the neck : Swelling in the neck was the main complaint for 9.73%of patients

E. Pain : Pain in the ear was noticed in minority of the patients.

F. Cough and throat irritation : Minority of the patients had cough and throat irritation.

G. Anorexia, Loss of weight : This was seen in all the patients.

11. CLINICAL EXAMINATION:

On general physical examination there was mild palor 26% of patients. Ten patients who had supraglottic growth complained of haemoptysis. Icterus was observed in two patients.

Almost 2/3 of the patients were cachectic. They had significant weight loss in recent past. The weight loss was also rapid.35% of the patients were bed ridden before coming to the hospital. There was no generalized lymphadenopathy or edema noted. Fever was present in 11 patients due to cough with expectoration.

12. SYSTEMIC EXAMINATION:

In all our patients ststemic examination was normal. No gross abnormality detected.35% of the patient had crepitations in their chest.

13. INVESTIGATIONS:

A. COMPLETE HAEMOGRAM :

All the patients admitted at our hospital were investigated with complete haemogram which included haemoglobin, total count, differential count, erythrocyte sedimentation, renal parameters, liver function tests, random blood sugar level. 26% of patients had haemoglobin less than 10 gm% .In all the patients total count and differential count was near normal. All the patients had liver and renal function tests within normal limit.

B. CHEST X-RAY :

Chest X-ray was obtained in all the patients. All the females had normal chest X-ray. Amongst the males 26% had emphysematous changes. No mediastinal widening noted. No other significant abnormality detected in chest x-ray.

C. COMPUTERISED TOMOGRAPHY OF NECK :

CT NECK was obtained in all our patients both axial and coronal cuts with and without contrast. Sagital reconstruction was done in some patients. This helps to know the tumour extent, involvement of the

lymphnodes, invasion of cartilage, invasion of oropharynx and hypopharynx.

D. **ABDOMINAL ULTRASONOGRAPHY :**

Abdominal USG done in all the cases to know for the distant metastasis.None of our patients had metastasis in the liver.

E. **VIDEOLARYNGOSCOPIC EXAMINATION :**

This is an office procedure done under local anaesthesia, the patient in sitting posture in barking dog position. A 30 degree and 70 degree Hopkins rod passed into the oral cavity, oropharynx and larynx. The three parts of the larynx examined. All the patients were subjected to this. The different types of growth seen are proliferative, ulcero proliferative and ulcerative type. Involvement of the different subtypes of the larynx studied.

1. GROSS TYPE OF GROWTH : Most of the tumours were proliferative in nature, later followed by ulceroproliferative type
2. TOPOGRAPHICAL REPRESENTATION: The most common sub site involved was supraglottis. Later followed by glottis. In our study we did not have any subglottic growth. In the supraglottic the most common subsite was aryepiglottic folds, followed by false vocal cords, arytenoids and epiglottis.
3. MOVEMENT OF THE TRUE VOCAL CORDS: Most of the patients had true vocal cords fixity

SITE	MALE	FEMALE	TOTAL
B/L SUPRAGLOTTIC	38	2	40
RIGHT SUPRAGLOTTIC	19	1	20
LEFT SUPRAGLOTTIC	22	-	22
B/L VOCAL CORDS	8	-	8
RIGHT VOCAL CORD	11	1	12
LEFT VOCAL CORD	11	-	11
SUB-GLOTTIS	-	-	-

F. DIRECT LARYNGOSCOPYC EXAMINATION AND BIOPSY

To obtain the tumour tissue for his to pathological examination biopsy obtained from the growth and sent for study. All the patients had squamous cell carcinoma as the histological diagnosis. Broders classification was used. Three types of seen.

They are well differentiated, moderately differentiated and poorly differentiated types.

HISTOPATHOLOGY	MALE	FEMALE	TOTAL
WELL.DIFF	43	1	44
MOD.DIFF	58	2	60
POORLY.DIFF	8	1	9

In our study most of our patients had moderately differentiated squamous cell carcinoma, constituting 53.09%.Well differentiated SCC was seen in 38.93% and poorly differentiated seen in 7.96%.Amongst the females two patients had mod diff SCC and one each patient had well differentiated and poorly differentiated SCC.

14. CLINICAL STAGING:

T STAGING:

T STAGING	MALE	FEMALE	TOTAL
T 1	1	-	1
T 2	21	-	21
T 3	85	4	89
T 4a	2	-	2
T 4b	-	-	-

N STAGING:

N STAGING	MALE	FEMALE	TOTAL
N0	55	-	55
N 1	33	2	35
N 2a	12	1	13
N 2b	5	-	5
N 2C	-	-	
N 3	4	1	5

TNM STAGING:

TNM	MALE	FEMALE	TOTAL
STAGE 0	-	-	-
STAGE I	1		1
STAGE II	14	-	14
STAGE III	71	2	73
STAGE IVa	18	1	19
STAGE IV b	5	1	6
STAGE IV C	-	-	-

For clinical staging of the laryngeal cancer AJCC used. The TNM staging was used, where T stands for tumour, N for lymph nodes metastasis and M for distant metastasis. Most of our patients were in T3 .Lymph nodes most commonly involved was N0.None of our patients had distant metastasis. The most common staging was stage III.

15. TREATMENT :

The different modalities of treatment offered to our patients at our hospital are surgery, radiotherapy and chemotherapy often in combinations. Nearly half of our patients came to us with stridor and emergency tracheostomy was performed. Few of our patients had difficulty in swallowing who were unable to take oral diet.In such patients feeding jejonostomy was performed. Patients initially evaluated

for operability and for chemo radiotherapy. First modality of treatment offered to our patients was chemoradiotherapy as we had the most of the patients from stage III and Stage IV. Surgery was not opted in any of our patients.

A) EMERGENCY TRACHEOSTOMY:

TRACHEOSTOMY	MALE	FEMALE	TOTAL
DONE	51	1	52
NOT DONE	58	3	61

Chemotherapy as a single modality of treatment was given in very few patients who were unfit for surgery and radiotherapy. Otherwise usually combined with the radiotherapy.

In significant minority of patients received only palliative chemoradiotherapy due to late stage of disease and moribond health status.

B) CHEMORADIOTHERAPY:

At our hospital for radiotherapy we use Cobalt-60 radiation unit. A Cobalt-60 machine uses a radioactive isotope that produces gamma photons as the source decays. The cobalt source has a half-life of 5.27 years and should be replaced about every five years. The focus to skin distance for a cobalt machine is usually 80cm. The patient's dose for

treatment using the Cobalt-60 machine is calculated using several factors. The daily dose rate available from the cobalt must be known.

The amount of centigray or Gray per fraction, the depth of the tumor, the size of the field and the daily dose rate must all be entered into the equation that will determine the time of the treatment. The only way we can regulate the amount of the dose from the cobalt machine is by varying the length of time the machine is on. The concurrent chemoradiotherapy was offered to most of our patients.

TREATMENT GIVEN :

TREATMENT	MALE	FEMALE	TOTAL
CONCURRENT CHEMORT	94	3	97
PALLIATIVE RT	10	-	10
CHEMOTHERAPY	5	1	6

The tumoricidal dose of radiotherapy for therapeutic purpose used was 6000Cgy for 6 weeks. Five days a week.200 Cgy per fraction. The palliative radiotherapy dose was 4800 Cgy for 16 fractions. Each fraction of 300 Cgy.

The commonly used chemotherapy at our hospital is cisplatin, methotrexate and 5-flurouracil.The recommended doses of cisplatin is 40mg/m2 weekly or 100mg /m2 in divided doses every 2 weeks as a single intravenous dose. The methotrexate is used in the dose of

500mg/m2 given on a weekly basis for 2 weeks. The 5-flurouracil is used in the dose 450-600 mg/sq.meter IV weekly, for 2 weeks.

16. ADVERSE EFFECTS OF CHEMORADIOTHERAPY:

The acute side effects pertaining to skin changes were seen in all the patients. The confluent moist desquamation and moderate edema was seen in 62 % of cases. Mucositis was yet another condition seen in all the patients .All most all of our patients had severe pain. They were prescribed narcotics to treat the pain. Majority of patients had thick saliva, altered taste and difficulty in swallowing. Mild to moderate hair loss was observed in most of our patients. Loss of sense of smell was also seen in minority of patients and was severe in tracheostomised patients.

17. FOLLOW-UP

In our study we had 113 patients. Four patients died during the study period. six patients did not complete the treatment as they absconded. For four patients radiotherapy was with held to improve haemoglobin status. Five patients lost the follow up. After completion of treatment follow up was done every month for first three months.

DISCUSSION

Cancer of larynx causes great misery to the patient due to development of difficulty in speech and swallowing which are the basic need in day today life of any individual.

The mean age of laryngeal cancer patients in our study was 65 years. These results were consistent with studies conducted by Shelly Chadha Et al at Delhi and study at Puerto Rico by Albert Villanueva. In present study laryngeal cancer predominantly present in the males. It has been reported in earlier studies that there is marked male preponderance.

As our study was conducted at a government hospital where patients from low socioeconomic strata are in major numbers. According to Kuppusamy classification they fall in class V. Due to their low socio-economic status, less education qualification, they have less purchasing power. Most of our patients come from south Tamil Nadu. Madurai followed by Ramnad districts patients were more amongst them. Our results similar to that of study conducted by Umesh Kapil Et Al.

Most of our patients were non-vegetarians taking meat, chicken with reused oil in most of the days of the week. They used less green leafy vegetables and protective fruits. Skipping of morning breakfast and

late lunch predisposed to gastroesophageal reflux disease. Irregular dietic habits seen in most of the patients.

All our male patients were smokers and alcoholics. The female patients were tobacco chewers along with betel nut and betel leaves. More than just half of our male patients were also tobacco chewers. Most of our patients smoked and consumed alcohol for more than 30 years. The same resluts were observed in India as well as western studies.

The most common presentation to us was with difficulty in breathing, stridor. Nearly half of our patient presented with stridor and underwent emergency tracheostomy. Next common presentation was change of voice. This is followed by dysphagia, swelling in the neck, pain, cough and throat irritation. Almost 2/3 of the patient had significant weight loss. Nearly ¼th of the patients had low haemoglobin level. Liver and renal function tests were normal in all our patients. In the study conducted by Shelly Chadha Et al at Delhi, hoarseness of voice was the most common symptom.

Except for few minor abnormalities, chest X-ray was normal. The CT scan was taken in all the patients.

The most common tumour staging was stage III followed by stage IVa. The most common site involved was supraglottis. The T (tumour) staging more of T3,next common was T4a.The nodal staging more common was N0 followed by N1.Our study results were similar Shelly

Chadha Et al results. Patients presented in advanced stage. They had N2 as the most common nodal stage, but in our study it was N0.

All our patients had squamous cell carcinoma as the histopathological diagnosis. The most common histopathological grading was moderately differentiated squamous cell carcinoma. This was followed by well differentiated squamous cell carcinoma. Both Indian studies and western studies had squamous cell carcinoma as the most common type of pathology.

At our hospital all patients received chemoradiotherapy as the major modality of the treatment. In four patients palliative radiotherapy was offered due to end stage of the disease. The cartilage involvement seen in two patients and chemotherapy was offered to them to prevent chondronecrosis due to radiotherapy. Most of our patients received therapeutic chemoradiotherapy. Kamian Et al supported the radiotherapy with or without chemotherapy to all the stages of laryngeal cancers.

SUMMARY

- In our study most of them were male patients. The most common age group Was 65 years.
- We had only 4 female patients in our study and all had supraglottic growth.
- All the patients belonged to low socioeconomic strata.
- The most common part of larynx involved is supraglottis.
- None of our patients had distant metastasis.
- The most common presentation was difficulty in breathing and stridor, later followed by change of voice and dysphagia.
- The common histopathological finding was squmous cell carcinoma. In that moderatelty differentiated was the most common type.
- The most common stage was stage III.
- The most common treatment offered at our hospital was concurrent chemoradiotherapy.

CONCLUSION

- Cancer larynx is highly preventable disease by avoiding the risk factors like tobacco and alcohol.
- Any patient complaining of change of voice for more than three weeks should be investigated thoroughly with videolaryngoscopic examination. Especially in an elderly male who is a chronic alcoholic and smoker.
- Curative rates are high in cancer larynx if diagnosed early and treated appropriately.
- Increase in the socioeconomic strata, education level can create awareness in the public and to approach near by health care centre for change of voice especially in elderly males.
- Unlike in the west, supraglottic cancer is common at our hospital.
- Chemoradiotherapy has made real impact in treatment stratergy. This is one of the treatment modality which helps in organ preservation and thus the voice production. Prognosis is poor in later stages of the disease.

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PROFORMA

Name:

Age:

Sex:

Address:

Occupation:

Socioeconomic status:

Hospital OP No:

Date of entry into study:

Informed consent obtained: Yes/ No

<u>Symptoms:</u>

Hoarseness of voice:

Difficulty in swallowing:

Painful swallowing:

Irritation/Sore throat:

Painful Vocalisation:

Noisy breathing:

Respiratory distress:

Heartburn/vomiting:

Haemoptysis:

Neck swelling

Earache:

Weight loss:

Cough:

Onset :Sudden/gradual. Duration:

<u>Past history</u>:

Diabetes mellitus, Hypertension ,Ischemic heart disease, Tuberculosis,syphilis, Thyroid disorders, Gastroesophageal reflux disease.

<u>Personal history</u>:

Diet, Appetite, Sleep, Bowel and Bladder habits

H/o smoking (no. of cigarettes/beedies – in pack years):

H/o Alcohol intake (amount and duration):

H/o of betel nut/ pan/ gutka chewing:

Socio-economic status:

Exposure to dusty atmosphere/chemical irritants/Fumes:

<u>Family history</u>:

Similar complaints in family members:

Malignancy:

<u>Clinical Examination</u>:

<u>General examination</u>:

Built:good/moderate/poor

Pallor/Icterus/clubbing/cyanosis/pedal oedema

Lymph node status:Site/Size/Consistency/mobility

Systemic Examination:

Cardiovascular system:

Respiratory system:

Per Abdomen:

Central nervous system:

ENT Examination:

Oral cavity:

Oropharynx:

Indirect Laryngoscopy:

Video Laryngoscopy:

Rigid laryngoscopy and Biopsy:

Examination of Neck:

Examination of Ears:

Examination of Nose and Paranasal sinuses:

Provisional diagnosis:

Investigations:

Blood: Hb/BT/CT/ESR/WBC/Liver function tests

Chest X-ray/X-ray Neck

Biopsy:

CT Scan from Skull base to upper mediastinum:

Final diagnosis:

Treatment:

SL.NO	NAME	AGE	SEX	SES	PLACE	IP NO	CHIEF COMPLAINTS	PAST HISTORY	FAMILY HISTORY	PERSONAL HISTORY
1	KULAINDAVEL	70	M	CLASS V	THIRUMANGALAM	**46258**	NECK SWELLING,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
2	JANAKI	63	F	CLASS V	MADURAI	46902	DIFF SWALLOWING,VOICE CHANGE	NS	NS	TOBACCO CHEWER
3	KUPPUSAMY	**60**	M	CLASS V	THIRUPATTUR	47916	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
4	SANTHARAM	**65**	M	CLASS V	DINDIGUL	49280	DIFF SWALLOWING,VOICE CHANGE	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
5	PALANI	70	M	CLASS V	MADURAI	49689	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
6	SUBRAMANI	55	M	CLASS V	SHIVAGANGAI	50343	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
7	YESUDAS	60	M	CLASS V	RAMNAD	50300	NECK SWELLING,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
8	BALAKRISHNAN	56	M	CLASS V	DINDIGUL	51574	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
9	PERUMAL	65	M	CLASS V	DINDIGUL	57532	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
10	KARUPPU	48	M	CLASS V	USLAMPATTI	51771	NECK SWELLING,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
11	RAJARAM	70	M	CLASS V	RAMANATHAPURAM	51943	NECK SWELLING,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
12	AYYASAMY	76	M	CLASS V	VIRUDHNAGAR	52375	DIFF SWALLOWING,VOICE CHANGE	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
13	SUBRAMANIAN	59	M	CLASS V	THIRIPURAGUNDRAM	52853	NECK SWELLING,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
14	VIKRAMADITYAN	70	M	CLASS V	NARAYANAPURAM	52613	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
15	BHASKARAN	47	M	CLASS V	MADURAI	53929	NECK SWELLING,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
16	KALIAPPAN	62	M	CLASS V	MADURAI	54887	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
17	SELVARAJ	58	M	CLASS V	THIRUMANGALAM	54628	DIFF SWALLOWING,VOICE CHANGE	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
18	RAMASAMY	70	M	CLASS V	TUTTUKUDI	55354	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
19	RAJAGOPAL	64	M	CLASS V	TUTTUKUDI	55620	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
20	SERMAKANNI	65	M	CLASS V	SHIVAKASI	55892	NECK SWELLING,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
21	MUTHU	65	M	CLASS V	KORAIKULAM	56922	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
22	MUTTAIAH	60	M	CLASS V	USLAMPATTI	57218	DIFF SWALL,VOICE CHANGE-6 MTHS	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
23	KRISHNAMOORTHY	65	M	CLASS V	MADURAI	57217	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
24	JAMBULINGAM	63	M	CLASS V	THENI	57222	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO
25	SHIVAGURUNATHAN	62	M	CLASS V	MADURAI	57799	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO
26	GANESHAN	55	M	CLASS V	DINDIGUL	58325	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
27	SHIVAGURUMOORTHI	62	M	CLASS V	THIRUMANGALAM	58874	NECK SWELLING,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
28	KANDASAMY	64	M	CLASS V	MADURAI	59403	change of voicedifficulty swallow	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
29	KAYAMBU	60	M	CLASS V	RAMNAD	59569	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
30	SYED ABUDHABI	54	M	CLASS V	PUDUKOTTAI	60389	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
31	CHINNASAMY	40	M	CLASS V	MADURAI	61047	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
32	KRISHNAN	63	M	CLASS V	PARAMAKUDI	62212	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
33	SELVARAJ	55	M	CLASS V	MADURAI	63891	DIFF SWALLOWING,VOICE CHANGE	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
34	RAJU	70	M	CLASS V	ARUPPUKOTTAI	64783	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
35	MUTTAIAH	60	M	CLASS V	MADURAI	64898	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
36	RAMAN	70	M	CLASS V	VIRUDHNAGAR	67146	VOICE CHANGE,DIFFI SWALLOWING	HTN,DM	NS	CHR.ALCO,CHR.SMO, TOB CHEW
37	DHANABHAGYAM	60	M	CLASS V	MADURAI	59645	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	DM,HTN,CVA	NS	CHR.ALCO,CHR.SMO
38	RAMAN	62	M	CLASS V	MADURAI	69018	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
39	DHANUSHKODI	51	M	CLASS V	VIRUDHNAGAR	68947	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	DM	NS	CHR.ALCO,CHR.SMO
40	MUTHU	55	M	CLASS V	DINDIGUL	71933	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
41	VANANGAMUDI	55	M	CLASS V	MADURAI	72334	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
42	PAALSAMY	60	M	CLASS V	DINDIGUL	74642	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
43	ARJUNAN	47	M	CLASS V	SHIVAGANGAI	76849	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
44	VELMURUGAN	38	M	CLASS V	MADURAI	78934	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
45	KARUPPADEVAR	59	M	CLASS V	DINDIGUL	80545	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
46	PONUCHAMY	80	M	CLASS V	MADURAI	82491	NECK SWELLING,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO

SL.NO	NAME	AGE	SEX	SES	PLACE	IP NO	CHIEF COMPLAINTS	PAST HISTORY	FAMILY HISTORY	PERSONAL HISTORY
47	KANNAN	60	M	CLASS V	RAMNAD	82735	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
48	VELU	62	M	CLASS V	RAMNAD	82736	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
49	MUTHU	65	M	CLASS V	MADURAI	74156	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
50	THANGAVELU	62	M	CLASS V	MADURAI	83435	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
51	SADAYANDI	65	M	CLASS V	MADURAI	87387	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
52	KRISHNAN	49	M	CLASS V	DINDIGUL	87168	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
53	RAJARAMAN	65	M	CLASS V	MADURAI	85934	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
54	KRISHNAN	56	M	CLASS V	MADURAI	90773	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
55	KASIPAMBU	62	M	CLASS V	MADURAI	90752	DIFF SWALL,VOICE CHANGE,NECK SWELLING	NS	NS	CHR.ALCO,CHR.SMO
56	SENTHIL	38	M	CLASS V	SHIVAGANGAI	93738	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO
57	ANGUSAMY	60	M	CLASS V	MADURAI	93711	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
58	JAFAR	67	M	CLASS V	MADURAI	94976	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
59	SETHUPATHI	55	M	CLASS V	RAMNAD	95396	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
60	GOPAL	63	M	CLASS V	KARUR	97477	DIFF SWALOW. CHANGE VOICE	NS	NS	CHR.ALCO,CHR.SMO
61	SAKARAI	47	M	CLASS V	DINDIGUL	97914	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
62	IBRAHIM	60	M	CLASS V	RAMNAD	93206	change of voicedifficulty swallow	NS	NS	CHR.ALCO,CHR.SMO
63	VILLAN	65	M	CLASS V	SHIVAGANGAI	92451	change of voicedifficulty swallow	NS	NS	CHR.ALCO,CHR.SMO
64	MARIAYAPPAN	65	M	CLASS V	SHIVAKASI	93957	NECK SWELLING,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
65	RAJAN	50	M	CLASS V	KULURNALAI	94975	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
66	IDUMBAN	65	M	CLASS V	RAMNAD	10983	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
67	LAKSHMANAN	70	M	CLASS V	MADURAI	11016	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
68	IRULANDI	60	M	CLASS V	MADURAI	11752	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
69	ADISINGAM	66	M	CLASS V	SHIVAGANGAI	11793	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
70	RAJAGOPAL	50	M	CLASS V	RAJAPALYAM	11808	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
71	THANGAPERUMAL	61	M	CLASS V	VIRUDHNAGAR	13336	change of voicedifficulty swallow	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
72	MUTTAIAH	60	M	CLASS V	MADURAI	14328	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
73	PERUMAL	50	M	CLASS V	THIRUMANGALAM	13350	change of voicedifficulty swallow	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
74	KUMAR	36	M	CLASS V	MADURAI	15219	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
75	SODALAI	65	M	CLASS V	MADURAI	16145	change of voicedifficulty swallow	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
76	KADALKARAI	40	M	CLASS V	MADURAI	17613	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
77	RAJENDRAN	74	M	CLASS V	MADURAI	17655	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
78	CHINNAIAH	47	M	CLASS V	MADURAI	17899	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
79	AYYANKALI	60	M	CLASS V	DINDIGUL	14835	change of voicedifficulty swallow	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
80	KATTAPULI	50	M	CLASS V	DINDIGUL	20876	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
81	RAMASAMY	52	M	CLASS V	MADURAI	21038	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
82	KARNAN	65	M	CLASS V	MADURAI	21336	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
83	KADARPACHA	74	M	CLASS V	TRICHY	21223	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
84	SAMUEL	40	M	CLASS V	MADURAI	21951	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
85	KUPPURAJA	70	M	CLASS V	DINDIGUL	21977	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
86	MUTHAYEE	70	F	CLASS V	MADURAI	23305	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	TOBACCO CHEWER
87	GANAPATHY	56	M	CLASS V	RAMNAD	23325	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
88	JAYAPAL	67	M	CLASS V	RAMNAD	23297	SWELLING IN NECK,VOICE CHANGE,DIFFI SWAL	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
89	KUMARAN	61	M	CLASS V	MADURAI	23287	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
90	SUBRAMANI	61	M	CLASS V	DINDIGUL	24007	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
91	MARIYAYEE	53	F	CLASS V	KARUR	25204	DIFF SWALL,VOICE CHANGE,NECK SWELLING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
92	PALANIVEL	60	M	CLASS V	KARUR	23953	COUGH,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW

SL.NO	NAME	AGE	SEX	SES	PLACE	IP NO	CHIEF COMPLAINTS	PAST HISTORY	FAMILY HISTORY	PERSONAL HISTORY
93	MARIAYAPPAN	60	M	CLASS V	MADURAI	24010	NECK SWELLING,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
94	SUNDARA MAHALINGAM	65	M	CLASS V	MADURAI	23733	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
95	APPUNATHAN	55	M	CLASS V	DINDIGUL	26585	SWELLING IN NECK,VOICE CHANGE,DIFFI SWAL	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
96	MOHAMMAD ALIYAR	55	M	CLASS V	DINDIGUL	26031	DIFF SWALL,VOICE CHANGE,NECK SWELLING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
97	KARPAGAM	70	F	CLASS V	SHIVAGANGAI	26789	SWELLING IN NECK,VOICE CHANGE,DIFFI SWAL	NS	NS	TOBACCO CHEWER
98	VELLAICHAMY	65	M	CLASS V	RAMNAD	26895	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
99	SADASIVAM	65	M	CLASS V	MADURAI	26911	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
100	PAULSAMY	63	M	CLASS V	MADURAI	26978	SWELLING IN NECK,VOICE CHANGE,DIFFI SWALL	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
101	veluchamy	68	M	CLASS V	DINDIGUL	29870	SWELLING IN NECK,VOICE CHANGE,DIFFI SWALL	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
102	KANDASAMY	59	M	CLASS V	KARUR	29910	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
103	JayaPALsamy	65	M	CLASS V	RAMNAD	30001	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
104	Thanga perumal	70	M	CLASS V	RAMNAD	30101	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO, TOB CHEW
105	RasuRaman	54	M	CLASS V	SHIVAGANGAI	30111	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
106	Irullappa pillai	62	M	CLASS V	TRICHY	30153	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
107	Veeraiah	73	M	CLASS V	MADURAI	30242	DIFF. SWALLOWING, VOICE CHANGE, NECK SWELLING	NS	NS	CHR.ALCO,CHR.SMO
108	Aavudiyappan	55	M	CLASS V	THIRUMANGALAM	30312	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
109	Muthu	71	M	CLASS V	MADURAI	30421	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
110	SundARAm	66	M	CLASS V	SHIVAGANGAI	30521	CHANGE OF VOICE, DIFFICULTY SWALLOW	NS	NS	CHR.ALCO,CHR.SMO
111	Chellaiah	62	M	CLASS V	RAMNAD	30614	VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
112	VELLAICHAMY	65	M	CLASS V	TRICHY	30745	STRIDOR,VOICE CHANGE,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO
113	Manian	62	M	CLASS V	MADURAI	30888	NECK SWELLING,DIFFI SWALLOWING	NS	NS	CHR.ALCO,CHR.SMO

SL.NO	NAME	AGE	SEX	GPE	SYSTEMIC EXAM	VLE	EAR	NOSE	EMER TRAC	DLS BIO	HPE	CHEST X-RAY	USG ABD
1	KULAINDAVEL	70	M	PALE	CREPTS	ULCEROPROL GROWTH	NORMAL	NORMAL	**YES**	YES	MOD.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
2	JANAKI	63	F	PALE	CREPTS	PROL GROWTH	NORMAL	NORMAL	**NO**	YES	WELL.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
3	KUPPUSAMY	**60**	M	PALE	CREPTS	**PROL GROWTH**	NORMAL	NORMAL	**YES**	YES	MOD.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
4	SANTHARAM	**65**	M	PALE	CREPTS	**PROL GROWTH**	NORMAL	NORMAL	**YES**	YES	WELL.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
5	PALANI	70	M	PALE	CREPTS	**PROL GROWTH**	NORMAL	NORMAL	**NO**	YES	WELL.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
6	SUBRAMANI	55	M	PALE	CREPTS	PROL GROWTH	NORMAL	NORMAL	**YES**	YES	MOD.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
7	YESUDAS	60	M	PALE	CREPTS	ULCEROPROL GROWTH	NORMAL	NORMAL	**YES**	YES	MOD.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
8	BALAKRISHNAN	56	M	PALE	CREPTS	ULCEROPROL GROWTH	NORMAL	NORMAL	**NO**	YES	WELL.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
9	PERUMAL	65	M	PALE	CREPTS	PROL GROWTH	NORMAL	NORMAL	**YES**	YES	MOD.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
10	KARUPPU	48	M	PALE	CREPTS	PROL GROWTH	NORMAL	NORMAL	**NO**	YES	MOD.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
11	RAJARAM	70	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
12	AYYASAMY	76	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	**NO**	YES	MOD.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
13	SUBRAMANIAN	59	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	**NO**	YES	WELL.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
14	VIKRAMADITYAN	70	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	**YES**	YES	MOD.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
15	BHASKARAN	47	M	NORMAL	CREPTS	ULCEROPROL GROWTH	NORMAL	NORMAL	**NO**	YES	WELL.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
16	KALIAPPAN	62	M	NORMAL	CREPTS	ULCEROPROL GROWTH	NORMAL	NORMAL	**NO**	YES	WELL.DIFF.SQCC	EMPHYSEMATOUS LUNGS	NORMAL
17	SELVARAJ	58	M	PALE	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
18	RAMASAMY	70	M	PALE	NORMAL	PROL GROWTH	NORMAL	NORMAL	**NO**	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
19	RAJAGOPAL	64	M	PALE	NORMAL	PROL GROWTH	NORMAL	NORMAL	**NO**	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
20	SERMAKANNI	65	M	PALE	NORMAL	PROL GROWTH	NORMAL	NORMAL	**NO**	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
21	MUTHU	65	M	PALE	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
22	MUTTAIAH	60	M	PALE	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	HYPER INFLA LUNGS	NORMAL
23	KRISHNAMOORTHY	65	M	PALE	CREPTS	ULCEROPROL GROWTH	NORMAL	NORMAL	**YES**	YES	MOD.DIFF.SQCC	HYPER INFLA LUNGS	NORMAL
24	JAMBULINGAM	63	M	PALE	CREPTS	ULCEROPROL GROWTH	NORMAL	NORMAL	**NO**	YES	MOD.DIFF.SQCC	HYPER INFLA LUNGS	NORMAL
25	SHIVAGURUNATHAN	62	M	PALE	CREPTS	ULCEROPROL GROWTH	NORMAL	NORMAL	**NO**	YES	MOD.DIFF.SQCC	HYPER INFLA LUNGS	NORMAL
26	GANESHAN	55	M	PALE	CREPTS	ULCEROPROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SCC	HYPER INFLA LUNGS	NORMAL
27	SHIVAGURUMOORTHI	62	M	NORMAL	NORMAL	ULCEROPROL GROWTH	NORMAL	NORMAL	**NO**	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
28	KANDASAMY	64	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	**NO**	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
29	KAYAMBU	60	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	**YES**	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
30	SYED ABUDHABI	54	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
31	CHINNASAMY	40	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	**NO**	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
32	KRISHNAN	63	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
33	SELVARAJ	55	M	PALE	NORMAL	PROL GROWTH	NORMAL	NORMAL	**NO**	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
34	RAJU	70	M	PALE	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
35	MUTTAIAH	60	M	PALE	NORMAL	PROL GROWTH	NORMAL	NORMAL	**YES**	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
36	RAMAN	70	M	PALE	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
37	DHANABHAGYAM	60	M	PALE	CREPTS	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	PATCHY PNEMONITIS	NORMAL
38	RAMAN	62	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	PATCHY PNEMONITIS	NORMAL
39	DHANUSHKODI	51	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SCC	PATCHY PNEMONITIS	NORMAL
40	MUTHU	55	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	PATCHY PNEMONITIS	NORMAL
41	VANANGAMUDI	55	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	PATCHY PNEMONITIS	NORMAL
42	PAALSAMY	60	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	PATCHY PNEMONITIS	NORMAL
43	ARJUNAN	47	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	PATCHY PNEMONITIS	NORMAL
44	VELMURUGAN	38	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	PATCHY PNEMONITIS	NORMAL
45	KARUPPADEVAR	59	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SQCC	PATCHY PNEMONITIS	NORMAL
46	PONUCHAMY	80	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	PATCHY PNEMONITIS	NORMAL

SL.NO	NAME	AGE	SEX	GPE	SYSTEMIC EXAM	VLE	EAR	NOSE	EMER TRAC	DLS BIO	HPE	CHEST X-RAY	USG ABD
47	KANNAN	60	M	PALE	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
48	VELU	62	M	PALE	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
49	MUTHU	65	M	PALE	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
50	THANGAVELU	62	M	PALE	NORMAL	ULCEROPROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
51	SADAYANDI	65	M	PALE	NORMAL	ULCEROPROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
52	KRISHNAN	49	M	NORMAL	NORMAL	ULCEROPROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
53	RAJARAMAN	65	M	NORMAL	NORMAL	ULCEROPROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
54	KRISHNAN	56	M	NORMAL	NORMAL	ULCEROPROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
55	KASIPAMBU	62	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	POORLY.DIFF.SQCC	NORMAL	NORMAL
56	SENTHIL	38	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
57	ANGUSAMY	60	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
58	JAFAR	67	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
59	SETHUPATHI	55	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
60	GOPAL	63	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	HYPER INFLA LUNGS	NORMAL
61	SAKARAI	47	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SCC	HYPER INFLA LUNGS	NORMAL
62	IBRAHIM	60	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	HYPER INFLA LUNGS	NORMAL
63	VILLAN	65	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	HYPER INFLA LUNGS	NORMAL
64	MARIAYAPPAN	65	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	HYPER INFLA LUNGS	NORMAL
65	RAJAN	50	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
66	IDUMBAN	65	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SCC	NORMAL	NORMAL
67	LAKSHMANAN	70	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	POORLY.DIFF.SQCC	NORMAL	NORMAL
68	IRULANDI	60	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
69	ADISINGAM	66	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
70	RAJAGOPAL	50	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SQCC	HYPER INFLA LUNGS	NORMAL
71	THANGAPERUMAL	61	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	HYPER INFLA LUNGS	NORMAL
72	MUTTAIAH	60	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	HYPER INFLA LUNGS	NORMAL
73	PERUMAL	50	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	HYPER INFLA LUNGS	NORMAL
74	KUMAR	36	M	NORMAL	CREPTS	PROL GROWTH	NORMAL	NORMAL	YES	YES	POORLY.DIFF.SQCC	HYPER INFLA LUNGS	NORMAL
75	SODALAI	65	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
76	KADALKARAI	40	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
77	RAJENDRAN	74	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
78	CHINNAIAH	47	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
79	AYYANKALI	60	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
80	KATTAPULI	50	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
81	RAMASAMY	52	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
82	KARNAN	65	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
83	KADARPACHA	74	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
84	SAMUEL	40	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
85	KUPPURAJA	70	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
86	MUTHAYEE	70	F	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
87	GANAPATHY	56	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
88	JAYAPAL	67	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
89	KUMARAN	61	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
90	SUBRAMANI	61	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
91	MARIYAYEE	53	F	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
92	PALANIVEL	60	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	NORMAL	NORMAL

SL.NO	NAME	AGE	SEX	GPE	SYSTEMIC EXAM	VLE	EAR	NOSE	EMER TRAC	DLS BIO	HPE	CHEST X-RAY	USG ABD
93	MARIAYAPPAN	60	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	POORLY.DIFF.SQCC	NORMAL	NORMAL
94	SUNDARA MAHALINGAM	65	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
95	APPUNATHAN	55	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	POORLY.DIFF.SQCC	NORMAL	NORMAL
96	MOHAMMAD ALIYAR	55	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
97	KARPAGAM	70	F	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	POORLY.DIFF.SQCC	NORMAL	NORMAL
98	VELLAICHAMY	65	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
99	SADASIVAM	65	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
100	PAULSAMY	63	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
101	veluchamy	68	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
102	KANDASAMY	59	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
103	JayaPALsamy	65	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	POORLY.DIFF.SQCC	NORMAL	NORMAL
104	Thanga perumal	70	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
105	RasuRaman	54	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
106	Irullappa pillai	62	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
107	Veeraiah	73	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
108	Aavudiyappan	55	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	POORLY.DIFF.SQCC	NORMAL	NORMAL
109	Muthu	71	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
110	SundARAm	66	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
111	Chellaiah	62	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	NORMAL	NORMAL
112	VELLAICHAMY	65	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	YES	YES	MOD.DIFF.SQCC	NORMAL	NORMAL
113	Manian	62	M	NORMAL	NORMAL	PROL GROWTH	NORMAL	NORMAL	NO	YES	WELL.DIFF.SQCC	NORMAL	NORMAL

SL.NO	NAME	AGE	SEX	CT NECK	TNM	STAGE	DIAGNOSIS	TREATMENT	FOLLOWUP	1MTH,VLE	2ND MTH,VLE	3RD MTH,VLE
1	KULAINDAVEL	70	M	Y	**t3n1mo**	**stage 3**	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
2	JANAKI	63	F	Y	**t3n2m0**	**stage 4a**	SUPRAGLOTTIC CA	CHEMOTHERAPY	Y	N	N	N
3	KUPPUSAMY	**60**	M	Y	**t3n0m0**	**stage 3**	GLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
4	SANTHARAM	**65**	M	Y	**t3n1m0**	**stage4a**	RT.SUPRAGLOTTIC CA	PALIATIVE RT	Y	N	N	N
5	PALANI	70	M	Y	**t2n0m0**	**stage 2**	RT.VOCAL CORD CA	CONCURRENT CHEMORT	Y	N	N	N
6	SUBRAMANI	55	M	Y	**t3n1m0**	**stage 3**	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	EXPIRED DURING RT			
7	YESUDAS	60	M	Y	**t3nom0**	**stage3**	B/L VOCAL CORD CA	CONCURRENT CHEMORT	WITH HELD			
8	BALAKRISHNAN	56	M	Y	**t2n1m0**	**stage 3**	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
9	PERUMAL	65	M	Y	**t3n0m0**	**stage 3**	GLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
10	KARUPPU	48	M	Y	**t2n1m0**	**stage3,**	RT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
11	RAJARAM	70	M	Y	T3N1M0	STAGE3	RT.SUPRAGLOTTC CA	CONCURRENT CHEMORT	OBSCUNDED			
12	AYYASAMY	76	M	Y	**t3n3m0**	**stage 4b**	LT.SUPRAGLOTTIC CA	PALIATIVE RT	Y	N	N	N
13	SUBRAMANIAN	59	M	Y	**t3n1m0**	**stage 3**	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
14	VIKRAMADITYAN	70	M	Y	**t3nn1m0**	**stage 3**	RT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
15	BHASKARAN	47	M	Y	**t2n2m0**	**stage 4a**	LT.SUPRAGLOTTIC CA	PALIATIVE RT	Y	N	N	N
16	KALIAPPAN	62	M	Y	**t1nomo**	**stage 1**	RT.VOCAL CORD CA	CONCURRENT CHEMORT	Y	N	N	N
17	SELVARAJ	58	M	Y	T3N0M0	STAGE3	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
18	RAMASAMY	70	M	Y	**t3n1m0**	**stage 3**	RT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
19	RAJAGOPAL	64	M	Y	**t2n0m0**	**stage2**	lt glottic ca	CONCURRENT CHEMORT	Y	N	N	N
20	SERMAKANNI	65	M	Y	**tt2n1m0**	**stage 3**	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
21	MUTHU	65	M	Y	**t2n1mo**	**stage3**	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
22	MUTTAIAH	60	M	Y	T2N0M0	STAGE2	RT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	OBSCUNDED			
23	KRISHNAMOORTHY	65	M	Y	**t3n1m0**	**stage3**	RT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
24	JAMBULINGAM	63	M	Y	**t3n0m0**	**stage3**	RT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	WITH HELD			
25	SHIVAGURUNATHAN	62	M	Y	T3NOMO	STAGE3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
26	GANESHAN	55	M	Y	T3N2M0	stage 4a	LT.SUPRAGLOTTIC CA	PALIATIVE RT	Y	N	N	N
27	SHIVAGURUMOORTHI	62	M	Y	t2n2m0	stage4a	SUPRAGLOTTIC CA	PALIATIVE RT	EXPIRED DURING RT			
28	KANDASAMY	64	M	Y	t2n0m0	stage2	RT.GLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
29	KAYAMBU	60	M	Y	t3nomo	stage 3	SUPRAGLOTTIC CA	OBSCUNDED	Y	N	N	N
30	SYED ABUDHABI	54	M	Y	tt2n1m0	stage 3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
31	CHINNASAMY	40	M	Y	t2nomo	stage 2	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
32	KRISHNAN	63	M	Y	t3n2m0	stage4a	LT.VOCAL CORD CA	PALIATIVE RT	Y	N	N	N
33	SELVARAJ	55	M	Y	t3n0m0	stage3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
34	RAJU	70	M	Y	t3n0m0	STAGE 3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
35	MUTTAIAH	60	M	Y	t3nomo	stage3	RT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
36	RAMAN	70	M	Y	T3N1M0	STAGE 3	LT.GLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
37	DHANABHAGYAM	60	M	Y	t3nomo	stage 3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
38	RAMAN	62	M	Y	t2nomo	stage2	LT.VOCAL CORD CA	CONCURRENT CHEMORT	Y	N	N	N
39	DHANUSHKODI	51	M	Y	T3N0MO	STAGE3	B/L VOCAL CORD CA	CONCURRENT CHEMORT	Y	N	N	N
40	MUTHU	55	M	Y	t3nomo	stage 3	RT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	OBSCUNDED			
41	VANANGAMUDI	55	M	Y	t3n0m0	stage3	VOCALCORD CA	CONCURRENT CHEMORT	Y	N	N	N
42	PAALSAMY	60	M	Y	tt3n1m0	STAGE 3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
43	ARJUNAN	47	M	Y	t3nomo	stage3	LT.VOCAL CORD CA	CONCURRENT CHEMORT	Y	N	N	N
44	VELMURUGAN	38	M	Y	t3n1m0	stage3	RT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
45	KARUPPADEVAR	59	M	Y	T3N0M0	STAGE3	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	LOST FOLLOWUP	
46	PONUCHAMY	80	M	Y	t3n2m0	STAGE 4A	LT.SUPRAGLOTTIC CA	PALIATIVE RT	EXPIRED DURING RT			

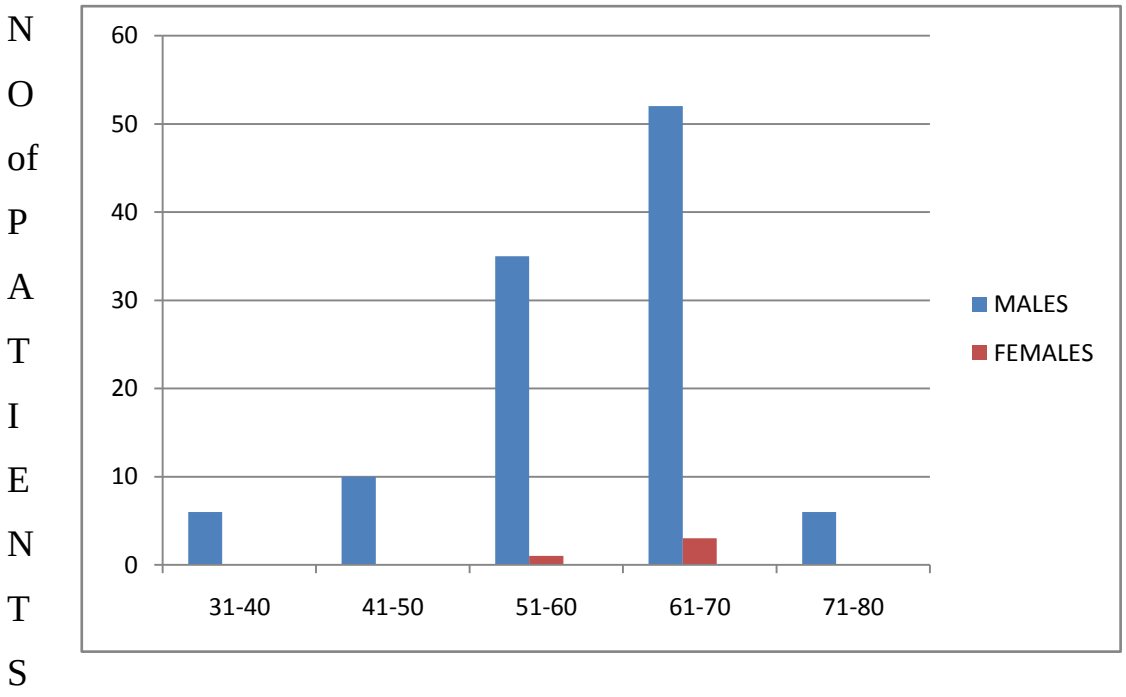
SL.NO	NAME	AGE	SEX	CT NECK	TNM	STAGE	DIAGNOSIS	TREATMENT	FOLLOWUP	1MTH,VLE	2ND MTH,VLE	3RD MTH,VLE
47	KANNAN	60	M	Y	t3n0m0	stage 3	RT.VOCAL CORD CA	CONCURRENT CHEMORT	WITH HELD			
48	VELU	62	M	Y	t3n0m0	stage 3	RT.VOCAL CORD CA	CONCURRENT CHEMORT	Y	N	N	N
49	MUTHU	65	M	Y	T2 N0M0	stage 2	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
50	THANGAVELU	62	M	Y	t3n0m0	stage3	LT.VOCAL CORD CA	CONCURRENT CHEMORT	Y	N	N	N
51	SADAYANDI	65	M	Y	t3n1m0	stage 3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	WITH HELD			
52	KRISHNAN	49	M	Y	t2n0m0	stage 2	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
53	RAJARAMAN	65	M	Y	T3N1M0	STAGE 3	VOCALCORD CA	CONCURRENT CHEMORT	Y	N	N	N
54	KRISHNAN	56	M	Y	t3nn2m0	stage 4a	SUPRAGLOTTIC CA	PALIATIVE RT	Y	N	N	N
55	KASIPAMBU	62	M	Y	T4 N2BM0	STAGE 4A	SUPRAGLOTTIC CA	PALIATIVE RT	EXPIRED DURING RT			
56	SENTHIL	38	M	Y	T3N1M0	STAGE 3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
57	ANGUSAMY	60	M	Y	t3n1m0	stge 3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
58	JAFAR	67	M	Y	t2n0m0	stage2	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
59	SETHUPATHI	55	M	Y	t3 n0m0	stage 3	rt glottic growth	CONCURRENT CHEMORT	Y	N	N	N
60	GOPAL	63	M	Y	t3 n0m0	stage3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
61	SAKARAI	47	M	Y	T3NOMO	STAGE3	LT.GLOTTIC CA	CONCURRENT CHEMORT	Y	LOST FOLLOWUP		
62	IBRAHIM	60	M	Y	t2n0m0	stage 2	LT.GLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
63	VILLAN	65	M	Y	t3n1mo	stage3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
64	MARIAYAPPAN	65	M	Y	t3n2m0	stage4a	LT.SUPRAGLOTTIC CA	PALIATIVE RT	Y	N	N	N
65	RAJAN	50	M	Y	t3n0m0	stage3	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
66	IDUMBAN	65	M	Y	T3N1M0	STAGE3	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	OBSCUNDED			
67	LAKSHMANAN	70	M	Y	T3N2BMX	STAGE 4A	SUPRAGLOTTIC CA	PALIATIVE RT	Y	N	N	N
68	IRULANDI	60	M	Y	t3n0m0	stage3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
69	ADISINGAM	66	M	Y	t3n1m0	stage3	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
70	RAJAGOPAL	50	M	Y	T3NOMO	STAGE3	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
71	THANGAPERUMAL	61	M	Y	t3n0m0	stage3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
72	MUTTAIAH	60	M	Y	T3N0M0	STAGE3	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
73	PERUMAL	50	M	Y	T2NOMO	STAGE2	rt sg growth	CONCURRENT CHEMORT	Y	N	N	N
74	KUMAR	36	M	Y	T3N2M0	STAGE 4A	SUPRAGLOTTIC CA	PALIATIVE RT	Y	N	N	N
75	SODALAI	65	M	Y	T3NOMO	STAGE 3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
76	KADALKARAI	40	M	Y	T3N1M0	STAGE3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
77	RAJENDRAN	74	M	Y	T3N1M0	STAGE 3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
78	CHINNAIAH	47	M	Y	T3N1MX	STAGE 3	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
79	AYYANKALI	60	M	Y	t3n0m0	stage 3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
80	KATTAPULI	50	M	Y	t3n0m0	stage3	RT.VOCAL CORD CA	CONCURRENT CHEMORT	Y	N	N	N
81	RAMASAMY	52	M	Y	t3n1m0	stage 3	RT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	LOST FOLLOWU
82	KARNAN	65	M	Y	t3n0m0	stage3	RT.VOCAL CORD CA	CONCURRENT CHEMORT	OBSCUNDED			
83	KADARPACHA	74	M	Y	T4AN2M0	STAGE4A	LT.VOCAL CORD CA	CHEMOTHERAPY	Y	N	N	N
84	SAMUEL	40	M	Y	t3n1m0	stage3	B/L SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
85	KUPPURAJA	70	M	Y	t3n1m0	stage 3	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
86	MUTHAYEE	70	F	Y	T3N1MX	STAGE 3	RT.VOCAL CORD CA	PALIATIVE RT	Y	N	N	N
87	GANAPATHY	56	M	Y	t3n1m0	stage 3	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
88	JAYAPAL	67	M	Y	T3N3MX	stage4b	LT.SUPRAGLOTTIC CA	CHEMOTHERAPY	Y	N	N	N
89	KUMARAN	61	M	Y	t3n1m0	stage3	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
90	SUBRAMANI	61	M	Y	t3n0m0	stage3	RT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
91	MARIYAYEE	53	F	Y	T3N1MX	STAGE 3	B/L SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
92	PALANIVEL	60	M	Y	t3n0m0	stage3	LT.VOCAL CORD CA	CONCURRENT CHEMORT	Y	N	N	N

SL.NO	NAME	AGE	SEX	CT NECK	TNM	STAGE	DIAGNOSIS	TREATMENT	FOLLOWUP	1MTH,VLE	2ND MTH,VLE	3RD MTH,VLE
93	MARIAYAPPAN	60	M	Y	T3N3MX	STAGE 4B	LT.SUPRAGLOTTIC CA	CHEMOTHERAPY	Y	N	N	N
94	SUNDARA MAHALINGAM	65	M	Y	t3n0m0	stage3	RT.VOCAL CORD CA	CONCURRENT CHEMORT	Y	N	N	N
95	APPUNATHAN	55	M	Y	T3N2BMX	STAGE 4A	RT.SUPRAGLOTTIC CA	CHEMOTHERAPY	Y	N	N	N
96	MOHAMMAD ALIYAR	55	M	Y	T3N1MX	STAGE 3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
97	KARPAGAM	70	F	Y	T3N3M0	stage4b	RT.SUPRAGLOTTIC CA	CHEMOTHERAPY	Y	N	LOST FOLLOWUP	
98	VELLAICHAMY	65	M	Y	T3NOMO	STAGE 3	SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
99	SADASIVAM	65	M	Y	t2n0m0	STAGE 2	LT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
100	PAULSAMY	63	M	Y	T3N2M0	stage 4a	RT.SUPRAGLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
101	veluchamy	68	M	Y	t3n2m0	stage 4a	bil supraglottic	CONCURRENT CHEMORT	Y	N	N	N
102	KANDASAMY	59	M	Y	T3N0M0	STAGE3	bil glottic	CONCURRENT CHEMORT	Y	N	N	N
103	JayaPALsamy	65	M	Y	T3N2BMX	STAGE 4A	SUPRAGLOTTIC GROWTH	CONCURRENT CHEMORT	Y	N	N	N
104	Thanga perumal	70	M	Y	T3N2BMX	STAGE 4A	bil sg growth	CONCURRENT CHEMORT	Y	N	N	N
105	RasuRaman	54	M	Y	t3n0m0	stage3	lt glottic ca	CONCURRENT CHEMORT	Y	N	N	N
106	Irullappa pillai	62	M	Y	t3n0m0	stage3	RT.GLOTTIC GROWTH	CONCURRENT CHEMORT	OBSCUNDED			
107	Veeraiah	73	M	Y	t2n0m0	stage2	LT.GLOTTIC CA	CONCURRENT CHEMORT	Y	N	N	N
108	Aavudiyappan	55	M	Y	T4AN1MX	STAGE 4A	SUPRAGLOTTIC GROWTH	CONCURRENT CHEMORT	Y	N	N	N
109	Muthu	71	M	Y	T3N0M0	STAGE3	SUPRAGLOTTIC GROWTH	CONCURRENT CHEMORT	Y	N	N	N
110	SundARAm	66	M	Y	T2NOMO	STAGE 2	RT.GLOTTIC GROWTH	CONCURRENT CHEMORT	Y	N	N	N
111	Chellaiah	62	M	Y	T3N3M0	stage4b	RT.SUPRAGLOTTIC	CONCURRENT CHEMORT	Y	LOST FOLLOWUP		
112	VELLAICHAMY	65	M	Y	T3N0M0	STAGE3	BIL.GLOTTIC GROWTH	CONCURRENT CHEMORT	Y	N	N	N
113	Manian	62	M	Y	t3n2m0	stage4a	RT.SUPRAGLOTTIC	CONCURRENT CHEMORT	Y	N	N	N

KEY TO MASTER CHART

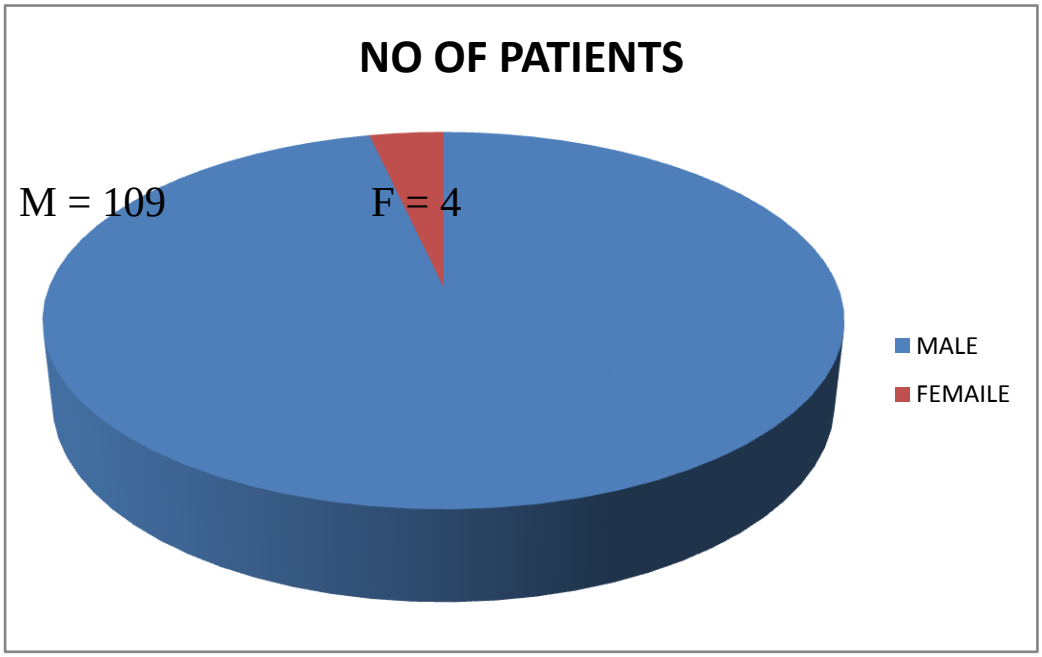
ALCO	=	ALCOHAL
CHR	=	CHRONIC
CHEMORT	=	CHEMORADIOTHERAPY
CHEW	=	CHEWING
DIFFI	=	DIFFICULTY
DIFF	=	DIFFERENTIATED
MOD	=	MODERATELY
PROL	=	PROLIFERATIVE
SCC	=	SQUAMOUS CELL CARCINOMA
SES	=	SOCIO ECONOMIC STATUS
SMO	=	SMOKING
SWALL	=	SWALLOWING
TOB	=	TOBACCO
Y	=	YES

AGE AT DIAGNOSIS

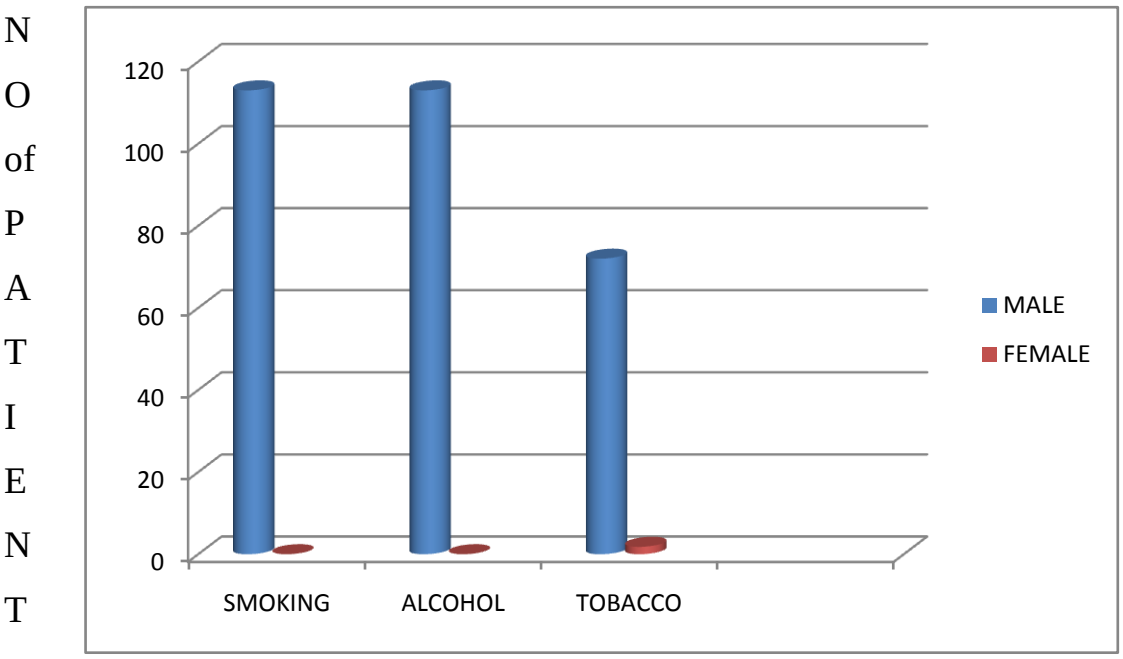


AGE IN YEAR

SEX INCIDENCE

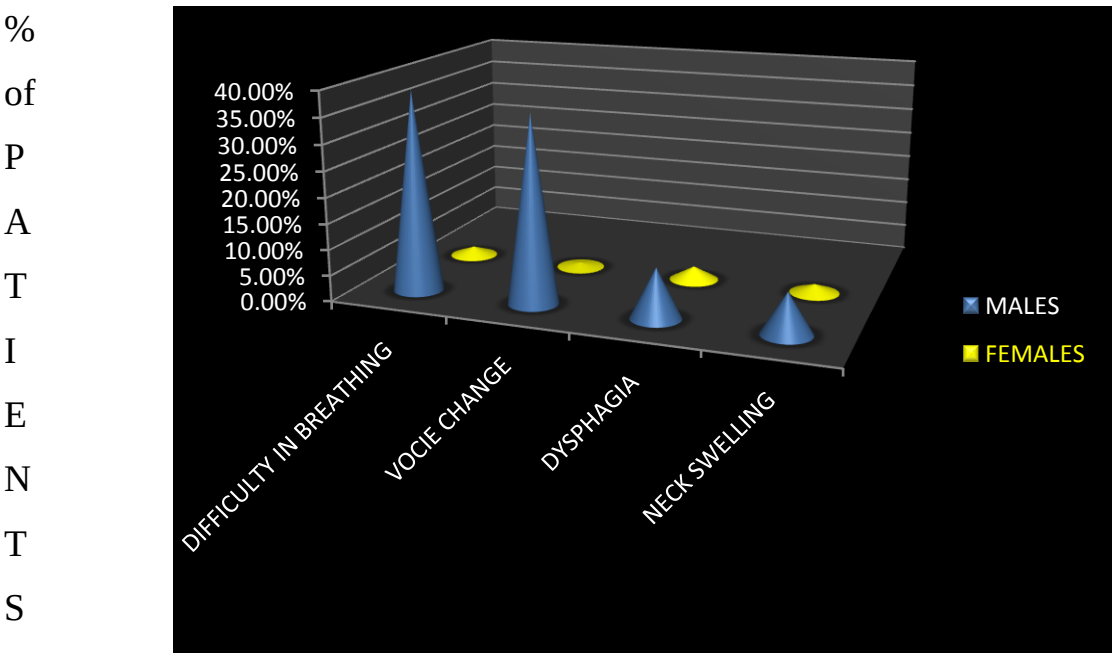


HABITS



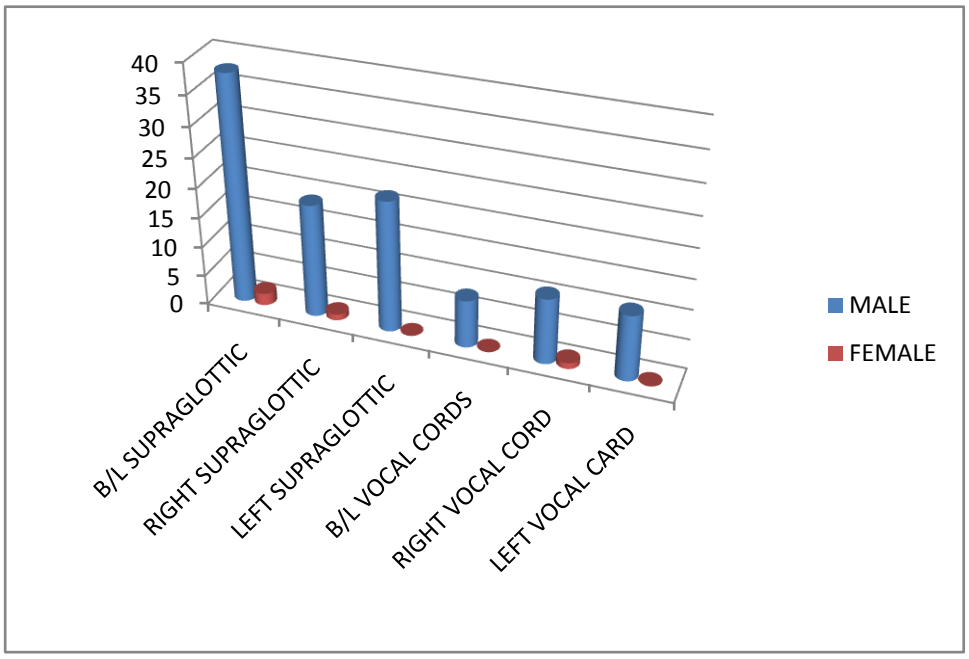
HABITS

SYMPTOMS

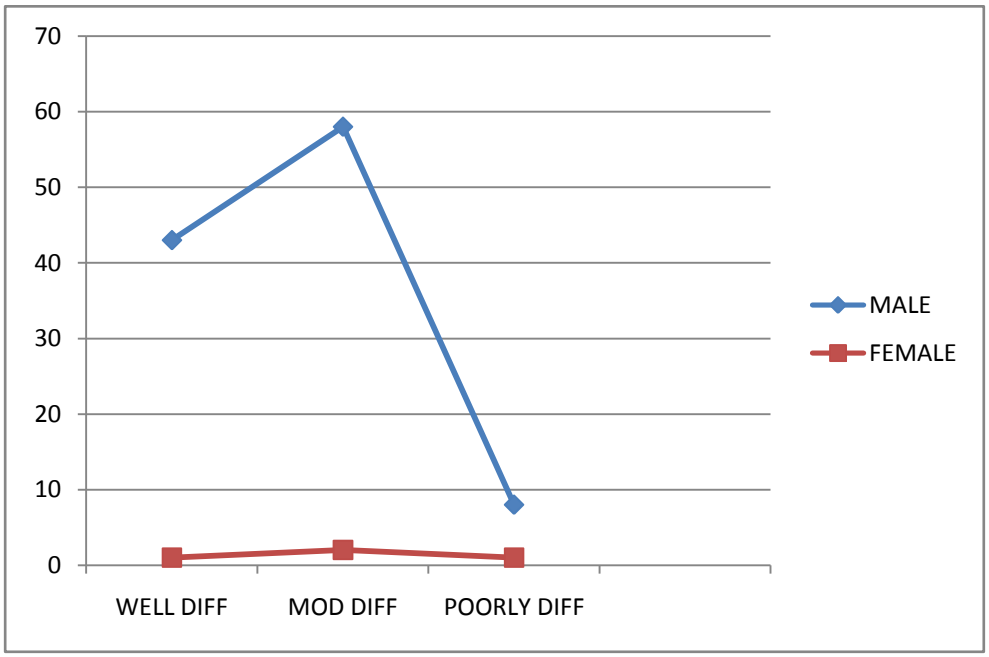


SYMPTOMS

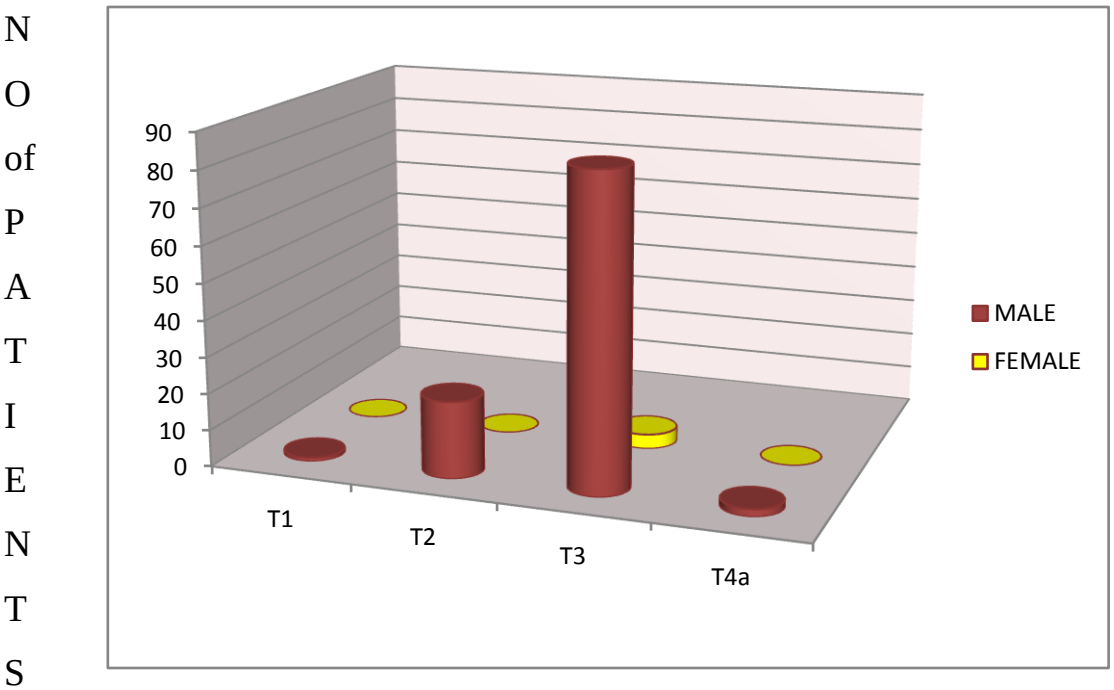
TOPOGRAPHICAL REPRESENTATION



HISTOPATHOLOGY

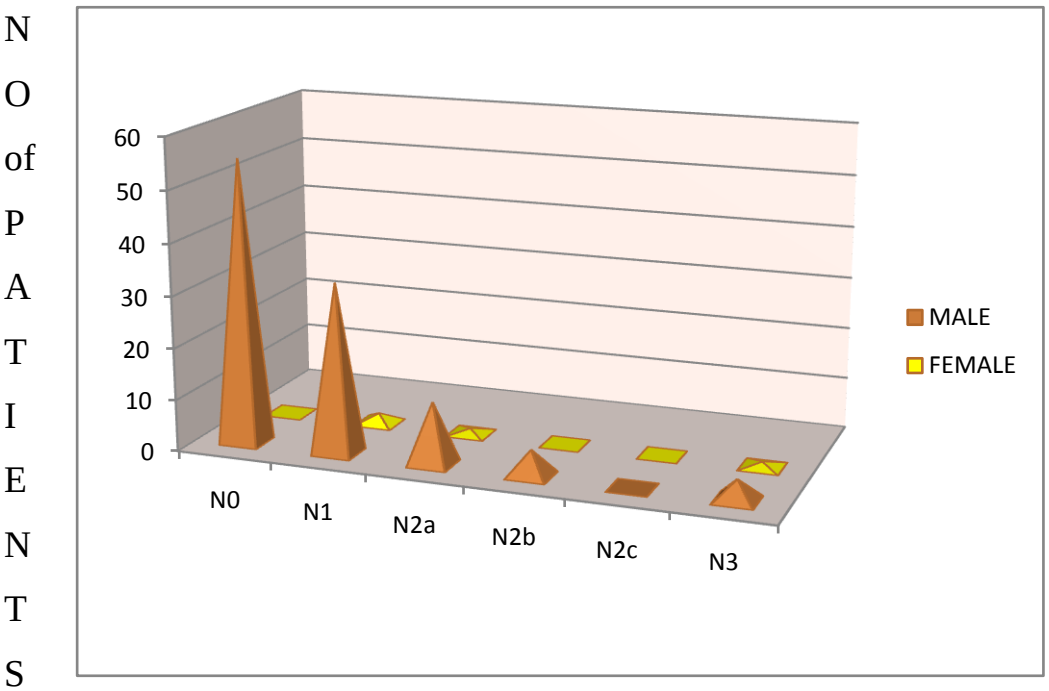


T STAGING



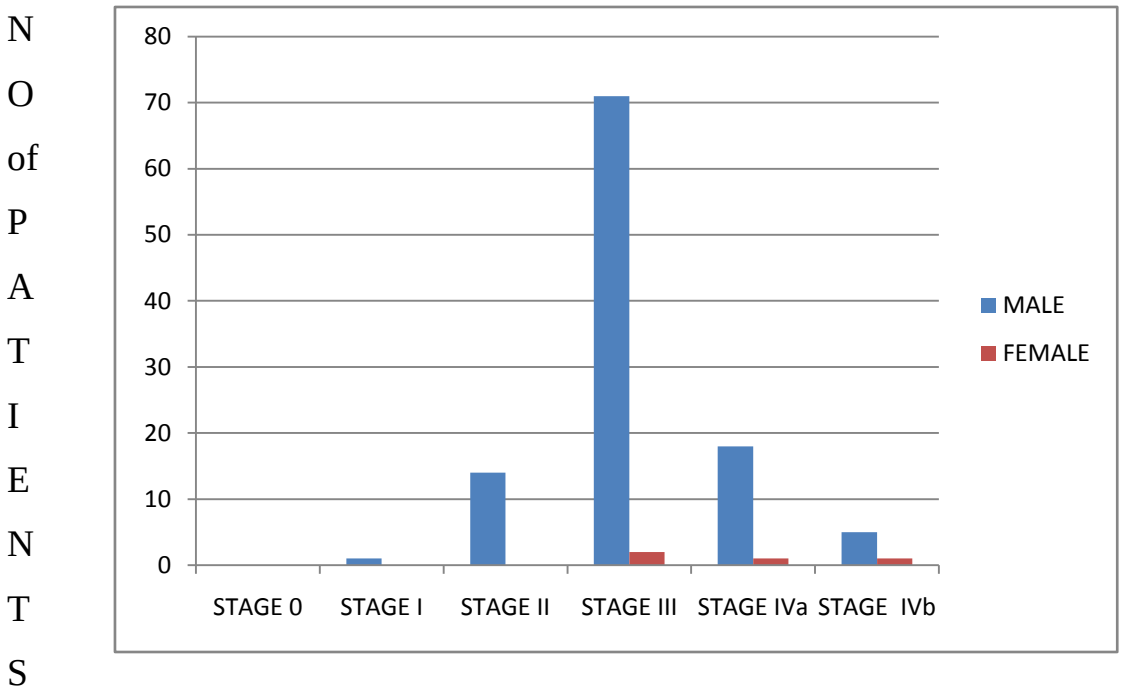
T STAGING

N – STAGING



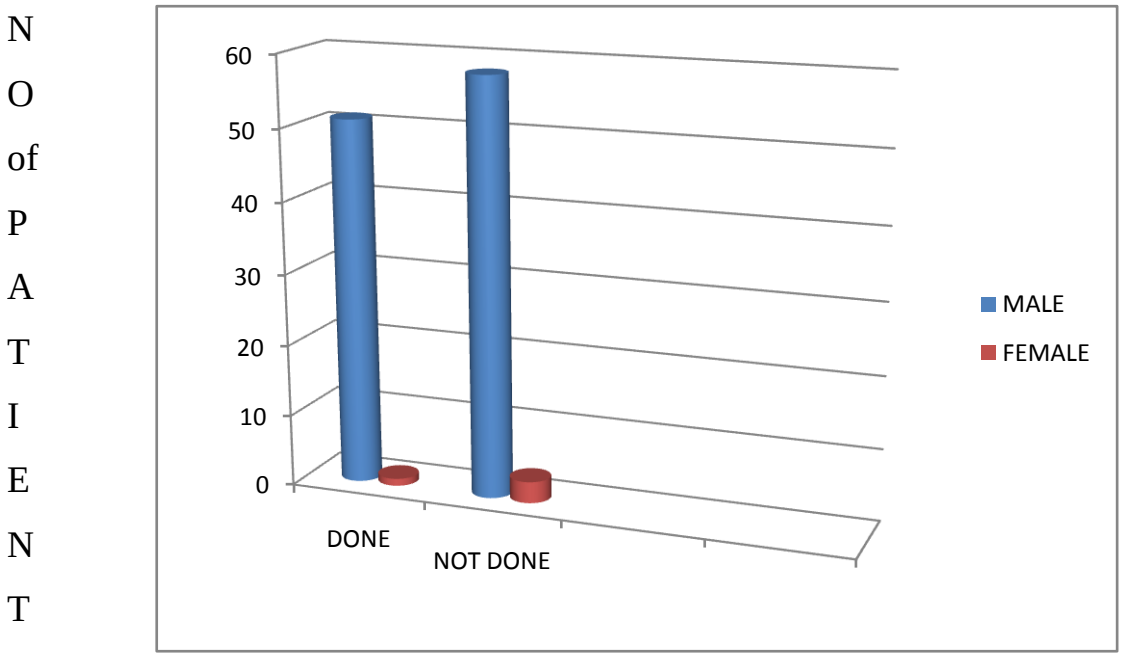
N – STAGING

TNM STAGING



TNM STAGING

TRACHEOSTOMY



TRACHEOSTOMY

TREATMENT GIVEN

T
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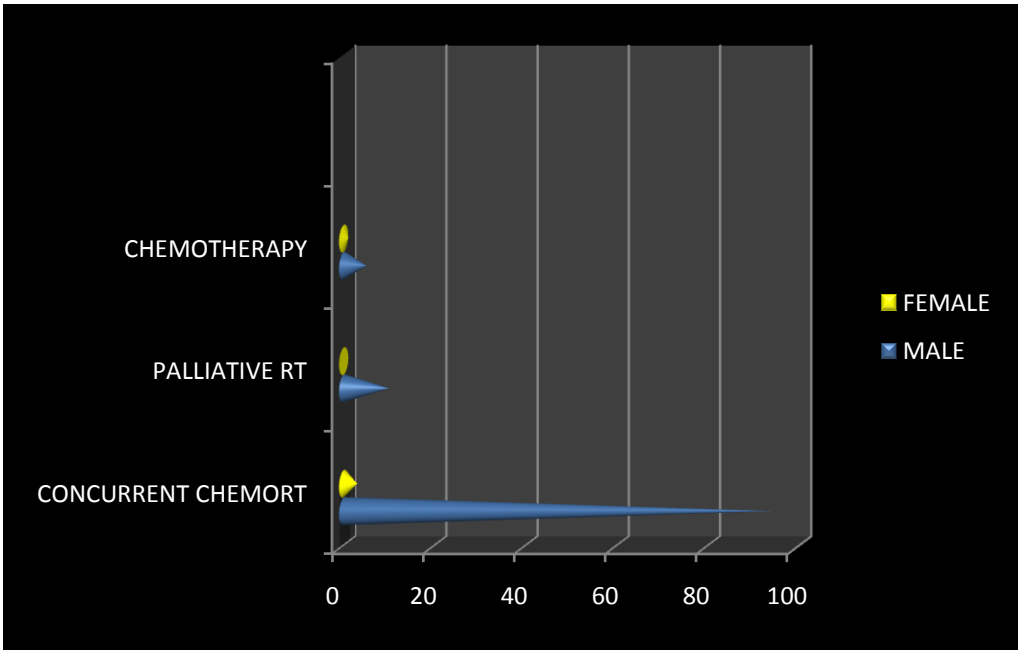
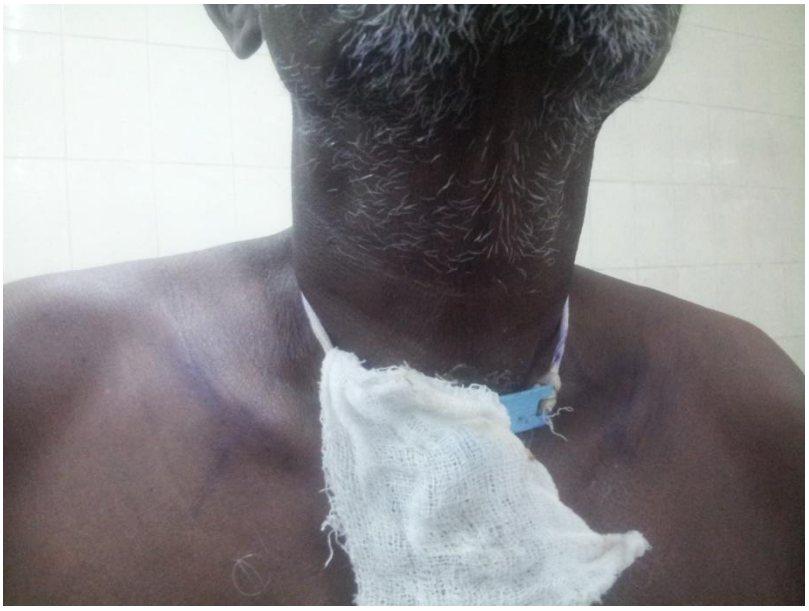
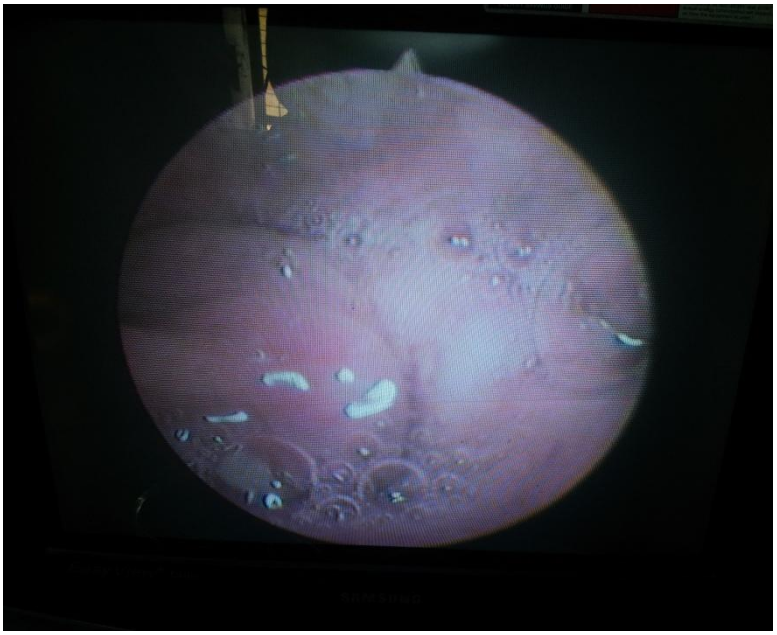


NO of PATIENTS

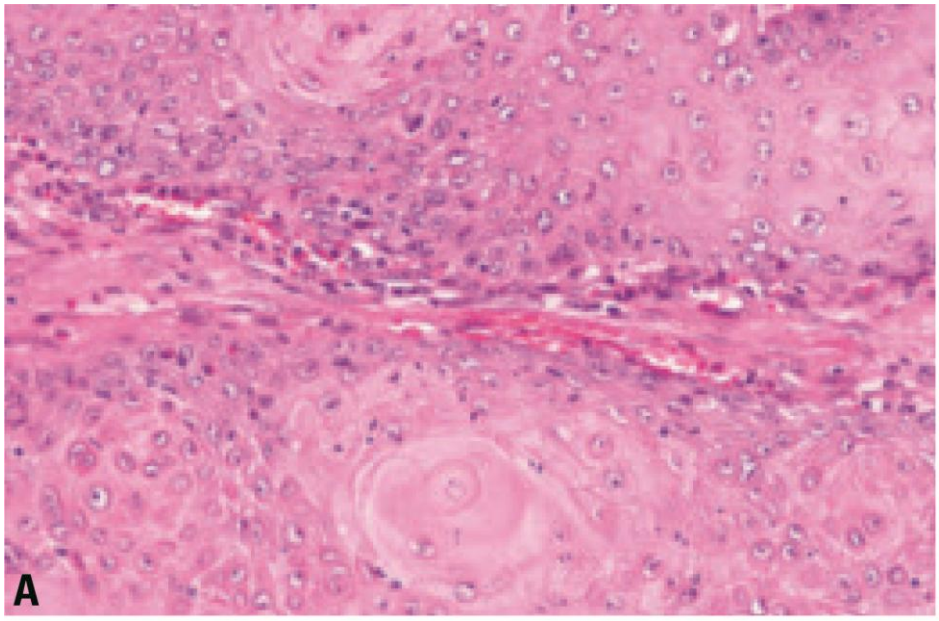




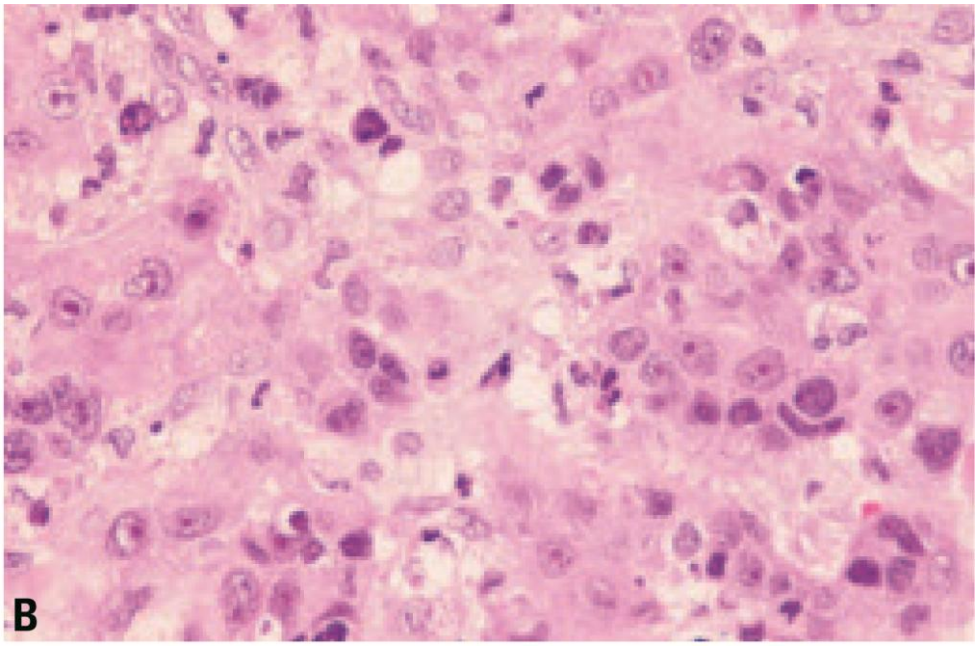
SUPRAGLOTTIC SCC T3N1M0

STAGE III

COBALT 60

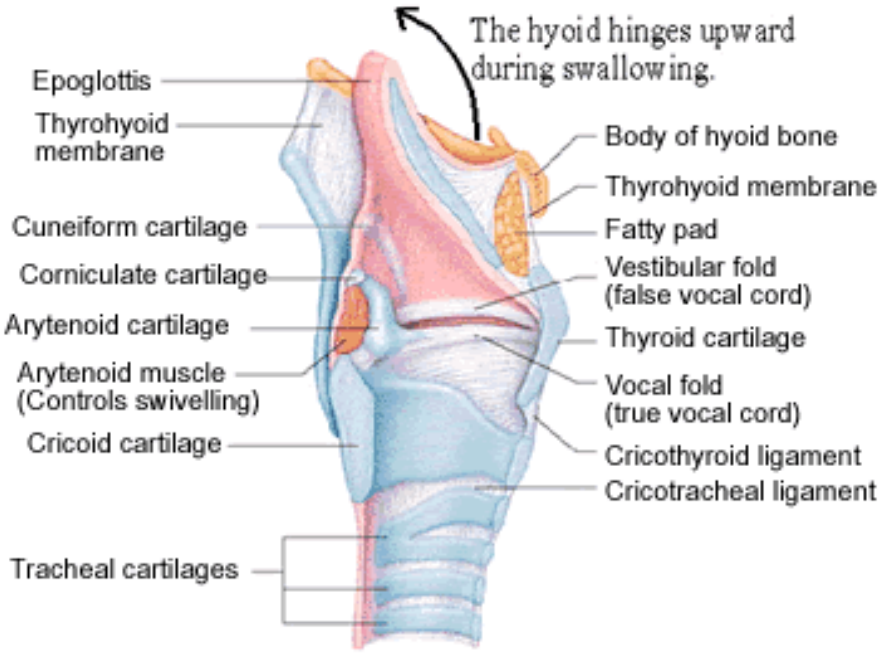


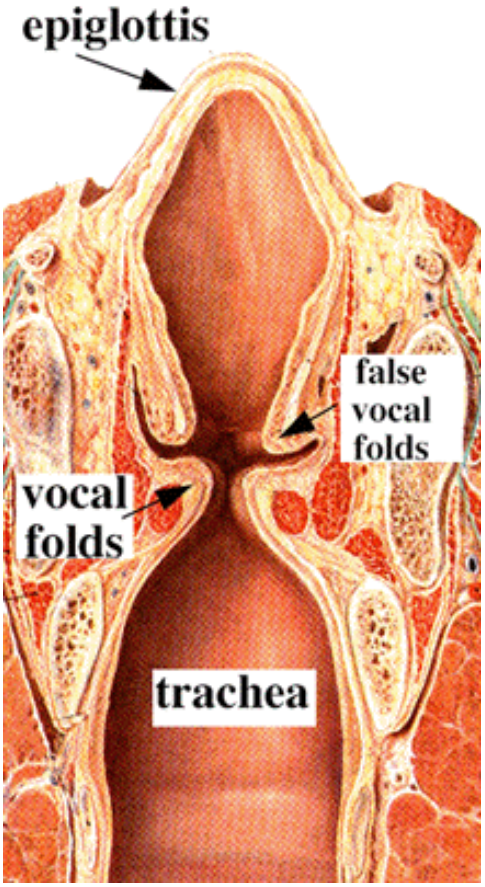
WELL DIFFERENTIATED SCC



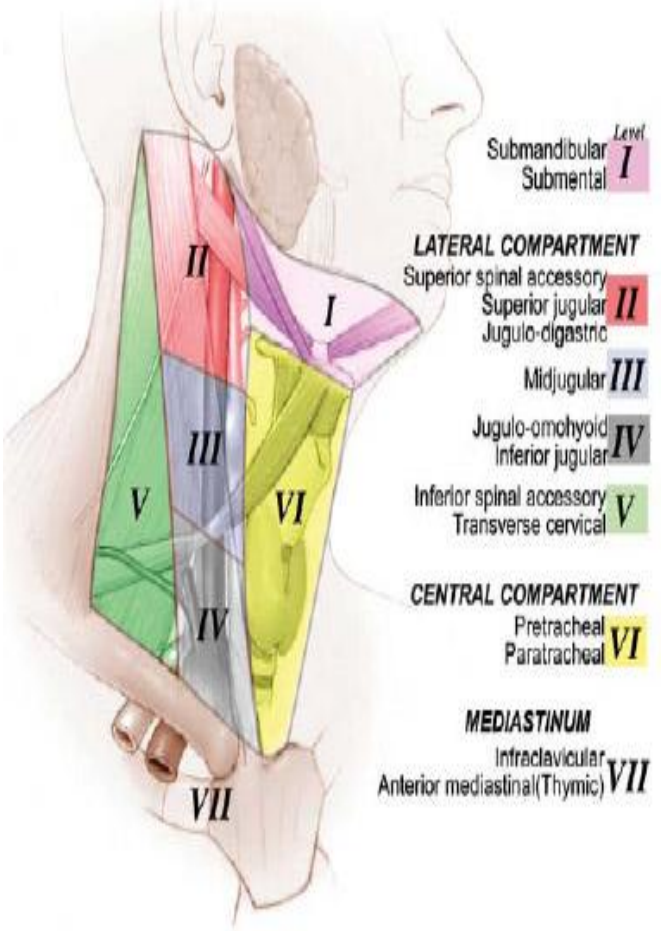
POORLY DIFFERENTIATED SCC

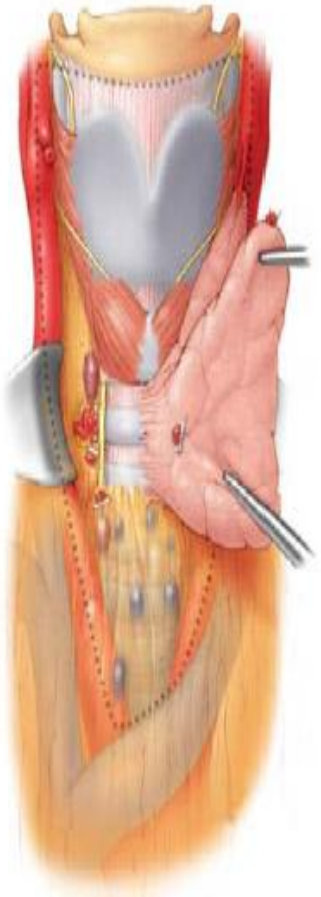
THE LARYNX


The hyoid hinges upward during swallowing.
Epoglottis
Thyrohyoid membrane
Cuneiform cartilage
Corniculate cartilage
Arytenoid cartilage
Arytenoid muscle (Controls swivelling)
Cricoid cartilage
Tracheal cartilages
Body of hyoid bone
Thyrohyoid membrane
Fatty pad
Vestibular fold (false vocal cord)
Thyroid cartilage
Vocal fold (true vocal cord)
Cricothyroid ligament
Cricotracheal ligament


epiglottis
false vocal folds
vocal folds
trachea

CERVICAL LYMPHNODES





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DISSERTATION SUBMITTED FOR

MASTER OF SURGERY Branch – IV

(OTORHINOLARYNGOLOGY)

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

APRIL 2015

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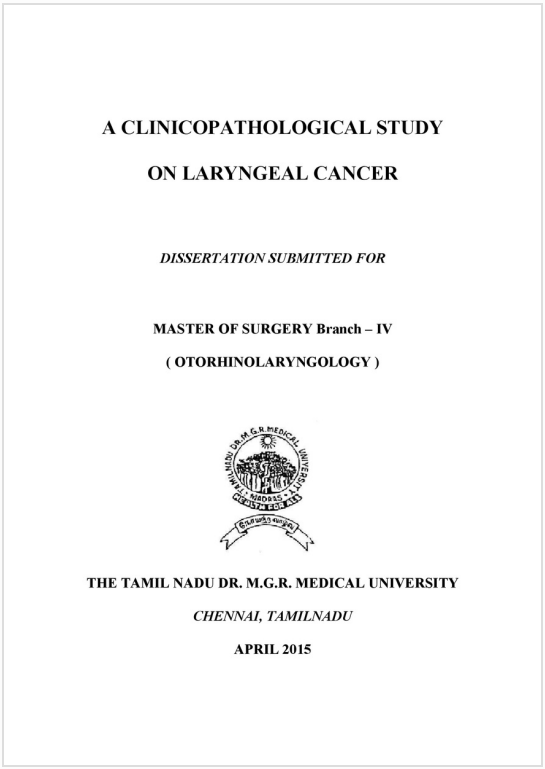

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