A CLINICAL STUDY OF SUPRAGLOTTIC TUMOUR

Dissertation submitted to

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

In partial fulfilment for the award of the degree of

MASTER OF SURGERY

OTORHINOLARYNGOLOGY

BRANCH-IV

DEPARTMENT OF OTORHINOLARYNGOLOGY,

THANJAVUR MEDICAL COLLEGE,

THANJAVUR – 613004.

MAY 2018

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INTRODUCTION

Cancer Larynx constitutes 2.63% of all body cancer.It is ten times more common in males than females.Both tobacco and alcohol are well established risk factors in Laryngeal Cancer and their effects are synergistic.Smoking is the main risk factor for glottis cancers whereas alcohol appears to be the bigger risk factor for supraglottic tumours . Supraglottic cancer is less frequent than glottis cancer, majority of the lesions are seen on the Epiglottis, and False cords followed by Aryepiglottic folds in that order.Supraglottic growths are often silent. Hoarseness is a late symptom. Throat pain, Dysphagia, and Referred pain in the ear or mass of Lymph nodes in the neck may be the presenting features.Nodal metastasis occurs early. Bilateral metastasis may be seen in cases of epiglottic cancer[1,2].

AIMS AND OBJECTIVES

1 . To study the tumours of various subsites of supraglottic region.

2 . To study the causal relationship between tumours of various subsites of supraglottic region and various etiopathogenesis.

3 . To study the type of malignancy in the supraglottic region.

4 .To study the symptomatology of tumours of various subsites of supraglottic region.

METHODOLOGY

Study Design: Discriptive study

Settings and Subjects: This study includes100 patients having history of persistence symptoms of supraglottic tumour for atleast six weeks inspite of symptomatic management in Thanjavur Medical College,Thanjavur from the period of june 2016 to October 2017 after getting ethical committee clearance.

Sample size:All the eligible patients according to the inclusion and exclusion criteria within the study period will form the study population.It is expected to be around 100.

Incusion criteria: 1.History of persistence symptoms of supraglottic tumour for atleast six weeks inspite of symptomatic management

2.Provision of informed consent

Exclusion criteria:1.Recurrence

2.Bleeding Disorder

3.Craniovertebral anomaly

4.Severe Trismus

5.Age below 35years

Data collection: Out patient and In patients of Department of OtoRhinoLaryngology, of Thanjavur Medical College Hospital, Thanjavur

Outcome meatures: Prevention of supraglottic tumour is the aim.The measures are:

1 .To Ascertain the causative factors

2. To Avoid the usage of risk factors

3 To Create the awareness of symptomatology which help for ealy diagnosis and complete cure.

4. To Reduce the incidence of supraglottic tumour in the community.

Data entry and analysis

The Data collected will be entered in a Excel Spreadsheet and will be analyzed using statistical software SPSS version 16. Mean,SD will be calculated: 't' test, Chisquare test and other appropriate statistical tests will be applied.

Descriptive study was designed to conduct in our Hospital after the protocol was approved by the ethical committee.

REVIEW OF LITERATURE

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I .EMBRYOLOGY

II.ANATOMY

III.PHYSIOLOGY

IV.AETIOLOGY

V. DIAGNOSIS AND STAGING

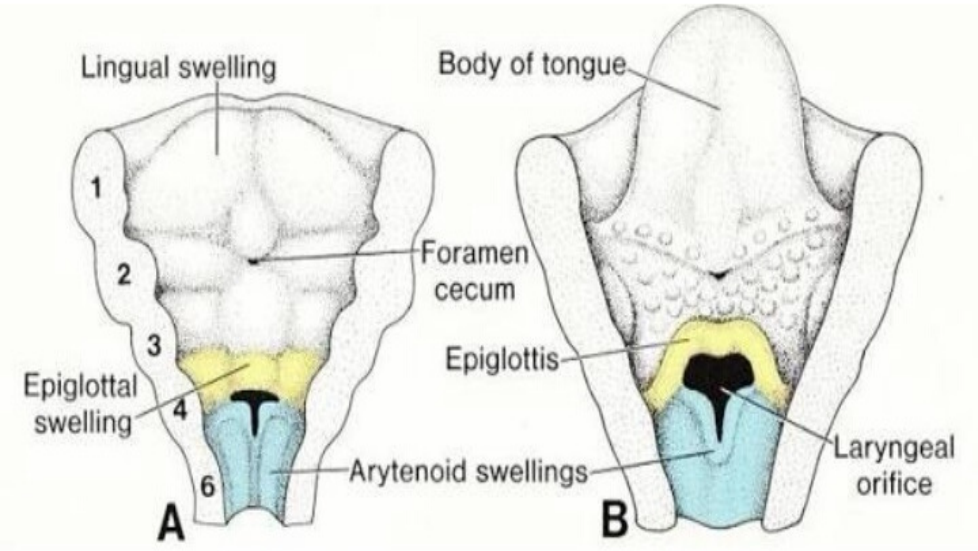
VI.TREATMENT DECISIONS IN LARYNGEAL CANCER

VII.VARIOUS SURGICAL TREATMENT OPTIONS

VIII.NECK DISSECTIONS

EMBRYOLOGY

This figure shows embryology of larynx.



The hypobranchial eminence is formed in the floor of the primitive pharynx between the ventral end of the third,fourth and second arch.The ventral foregut groove becomes demarcated as the laryngotracheal sulcus at the end of third week of fetal life.

Two distinct components are identified at this stage namely rostral pharyngeal groove and caudal laryngotracheal sulcus. The entire pulmonary system develops with development of the laryngeal portion which occurs initially and the lungs being formed last.

The esophagorespiratory separation occurs by a laryngotracheal septum . The structure derived caudal to the third pharyngeal arch forms the mesodermal elements of the larynx and will be innervated by superior laryngeal nerve.The trachea-branchial groove appears caudal to the hypobranchial eminence.

The larynx ,trachea and lungs develop from this groove which arises as a diverticulum posterior to hypo branchial eminence from foregut at above 4 weeks of embryonic life. A mesenchymal condensation from behind surrounds the eosophagus and respiratory tube while angiogenesis is beginning in the mesenchyme. The mesenchyme is denser in the glottis than the periphery and is localized in two planes ,i.e.,the internal constrictor which is derived from the fifth and sixth arch and the external constrictor which is derived from the fourth arch.

The external constrictor is the analogy of the inferior constrictor and the cricothyroid muscle.The inner constrictor is analogus to all the instric muscles of the larynx.The primitive respiratory groove which transforms into a laryngotracheal groove that communicates with the pharynx with laryngeal fissure.

Two swellings develope lateral to the laryngeal fissure which form the arytenoids and the primitive aryepiglottic folds. The hypobranchial eminence in the midline is responsible for the formation of prominence of primitive epiglottis.The hypobranchial eminence is notched rostrally and caudally denoting fusion of anterior extension of fourth arch and suggested a paired origin of epiglottis. The future laryngeal inlet is the representation of the paired arytenoid swellings and the the midline eminence in the floor of the pharynx.The triangular laryngeal inlet has a midline epiglottic fold and arytenoid swelling, Gradually the laryngeal lumen takes a 'T'shape and the laminar epithelium grows ventrally to obliterate the lumen by a solid cellular plug.

The cellular condensation then followed by central necrosis to form two canals, anterior vestibulotracheal canal and the posterior pharyngotracheal canal allowing the trachea to be separated from the esophagus and the hypopharynx at about 8 week the analage of the vocal cord is formed by an epithelial and mesodermal mass between the vestibule and the upper trachea.

By the 10 th week ,this mass splits sagittally giving rise to both pairs of vocal cords .Any incomplete fusion/split at this stage can lead to laryngeal web or atresia respectively.The laryngeal ventricle develops as

a medial and lateral fissure around the arytenoid eminence,form the 4 th branchial arch and cleft.

The lateral part forms the saccule of the ventricle and as it develops the true and false cords separate. The epiglottis is the last cartilaginous tissue to develop at the 18 mm stage. The thyroid cartilage is delineated at the 20 mm stage, developing from the ventral part of the 4 th branchial arch and fuses anterior to the pharyngotracheal canal.

The development is complete by the 10 th week by the formation of the Cricothyroid Joint. and at 55 mm stage the Cricoid cartilage develops.The hyaline cartilages of the larynx-Thyroid,Cricoid & Arytenoids-- develop from the 4^{th} branchial arch mesoderm,while the elastic cartilages-Epiglottis,Cuneiform & Corniculate - develop from the mesoderm of the floor of the pharynx. The hyoid bone is derived from the 2^{nd} and 3^{rd} branchial arches. Cricoid cartilage is derived from the 6^{th} arch derivative from the two mesodermal masses that fuse anterior to the pharyngotracheal canal. Laryngeal musculature develops as intrinsic mesenchymal condensation within the larynx.The laryngeal cavity has its adult form in 90 mm stage.

II. ANATOMY OF LARYNX

SITUATION AND EXTENT.

The larynx is the organ for production of voice.It is also an air passage and acts as a sphincter at the inlet of the lower respiratory passages.

The larynx lies in the anterior midline of the neck,extending from the root of the tongue to the trachea.In the adult male it lies in front of the 3^{rd},4^{th},5^{th}, and 6^{th} cervical vertebrae, But in children and in the adult female it lies at a higher level.

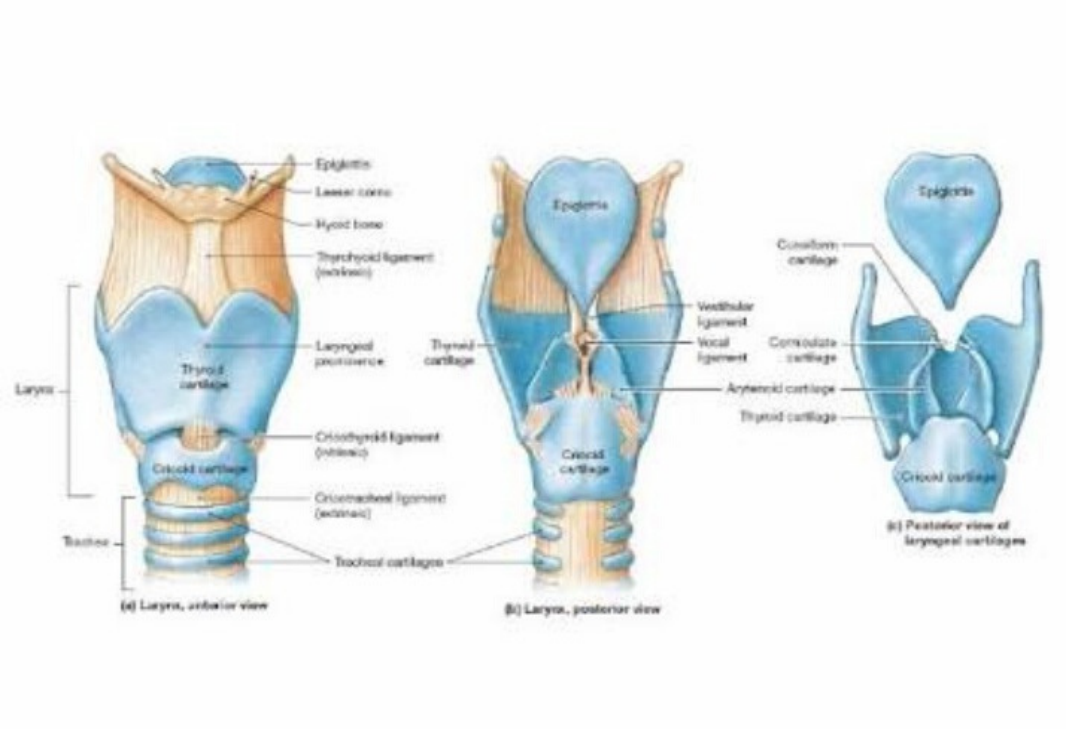
SIZE

The length of the larynx is 44mm in males and 36 mm in females.Until puberty the size is more or less same in the two sexes, But at puberty the male larynx grows rapidly and becomes larger than the female larynx.The pubertal growth of the female larynx is negligible.

CONSTITUTION OF LARYNX

The larynx is made up of a skeletal framework of cartilages.The cartilages are connected by joints,ligaments and membranes; and are

moved by a number of muscles.The cavity of the larynx is lined by mucous membrane.



This figure shows anatomy of larynx.

CARTILAGES OF LARYNX.;

The Larynx consists of three cartilages of which three are paired and three are unpaired.

UNPAIRED CARTILAGES: Thyroid, cricod,epiglottic cartilage

PAIRED CARTILAGES:

Arytenod,Corniculate,Cuneiform cartilage

THYROID CARTILAGE:

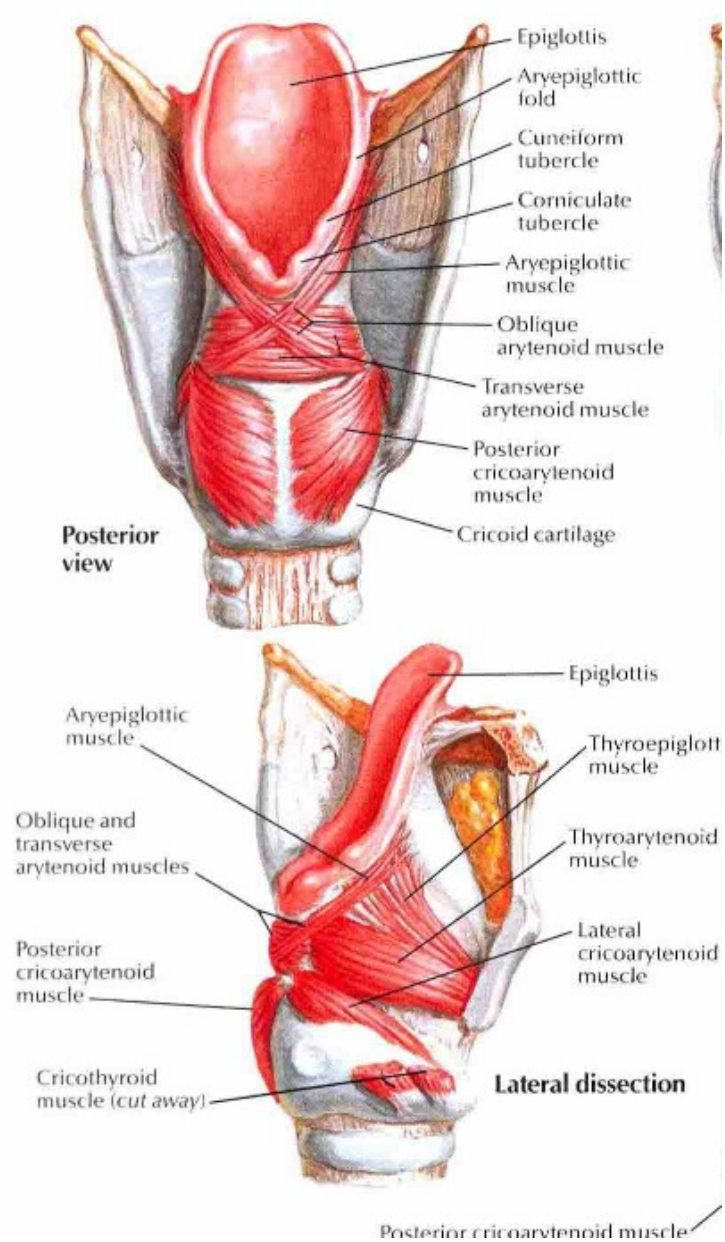
It is a V-shaped cartilage with a right and a left lamina, each lamina being quadrilateral in shape..Their posterior borders are far apart, but anterior borders approach each other at an angle, - about 90 degrees in males and 120 degrees in females..

The lower parts of anterior borders of right and the left laminae fuse and form median projection called laryngeal prominence .The upper parts do not meet .They are separated by a thyroid notch.The free posterior borders are prolonged upwards and downwards as superior and inferior cornua or horns.The superior cornua is connected with greater cornua of the hyoid bone by lateral thyrohyoid ligament.

The inferior cornua articulates with Cricoid cartilage to form cricothyroid joint.The superior border of thyroid cartilage is convex in front, and concave behind. In the median plane it is connected to the Cricoid cartilage by the conus elasticus.The outer surface of each lamina is marked by an oblique line,extending from superior

thyroid tubercle(in front of the superior cornua) to the inferior thyroid tubercle.

The Thyrohyoid, Sternothyroid, and Thyropharyngeus part of Inferior Constrictor muscle of pharynx are attached to the oblique line[3] of Thyroid cartilage. Lower border and inferior cornua give insertion to the triangular Cricothyroid muscle.



This figure shows muscles of larynx

Contents of the larynx:

1) Median thyroepiglottic ligament

2) Thyroepiglottic muscle on each side

3) Vestibular fold on each side

4) Vocal fold on each side

5) Thyroarytenoid

6) Vocalis muscle on each side

CRICOID CARTILAGE:

It is a complete ring encircling the larynx below the thyroid cartilage.The ring has a narrow anterior part called arch, and a broad posterior part called the lamina.The lamina projects upwards behind the thyroid cartilage, articulating superiorly with the arytenoid cartilages.

The inferior cornua of the thyroid cartilage articulates with the side of the cricoid cartilage at junction of arch and lamina.

ATTACHMENTS:

Anterior part of arch of cricoid gives rise to triangular cricothyroid muscle, a tensor of the vocal cord.

Anterolateral aspect of the arch gives origin to lateral cricoarytenoid muscle, an adductor of the vocal cord.

Lamina of cricoid cartilage gives origin to posterior cricoarytenoid muscle.

EPIGLOTTIS

It is a leaf shaped cartilage,of elastic type- attached to the anterior wall of interior of the upper part of larynx.It has a lingual surface,and a laryngeal surface.The lingual surface is innervated by glossopharyngeal nerve,the laryngeal surface is innervated by the vagus nerve

ATTACHMENTS

The lower end of the epiglottis is attached by the thyroepiglottic ligament to the upper part of the angle between the two laminae of the thyroid cartilage inferiorly.

The right and left margins of the Epiglottic cartilage provide attachment to the aryepiglottic folds. The anterior surface of the Epiglottis is connected :

1) To the tongue by median glossoepiglottic fold.

2) To the hyoid bone by the hyoepiglottic ligament.

The posterior surface is covered by the mucous memberane, presenting a tubercle in the lower part.

INTERIOR OF THE LARYNX;

The interior of the larynx is lined by respiratory epithelium except over vocal cords and parts of epiglottis which is lined by stratified squamous epithelium.

The laryngeal inlet is bounded by

1.Above and in front by the free margin of the epiglottis.

2.Laterally by the aryepiglottic fold

3.Posteriorly by the interarytenoid region.

DIVISIONS OF THE LARYNX

The laryngeal interior is divided by two folds into three parts.The two folds are the true vocal cord and the false vocal cord(ventricular band). The three parts are;

1.Supraglottis

-vestibule

-ventricle

2.Glottis

3.Subglottis

The division of the larynx is based on the developmental analage.The parts that are derived from the 3rd and the 4th arch is the supraglottic larynx whereas the parts that are derived from the 6th arch (RESPIRATORY ANALAGE) are the glottis and the subglottis.The 5th arch disappears during the development. The embryological subdivision also creates natural barriers as the lymphatic drainage,blood and nerve supply are well compartmentalized depending on the arch of origin.The malignancy of the larynx is also restricted to laryngeal compartments in the early stage due to the natural barriers before they infiltrate into other compartments.Thus conservation laryngectomy is possible in early stages.

SUPRAGLOTTIS

The wall is formed by quadrangular membrane which extends from vestibular fold ligaments to aryepiglottic folds.

The supraglottis comprises of the following structures:

1.Epiglottis

Suprahyoid part

Infrahyoid part

Laryngeal aspects of aryepiglottic folds

Arytenoids

Ventricular bands

Ventricles

THE INFERIOR LIMIT OF THE SUPRAGLOTTIS IS:

1.CLINICALLY –Imaginary horizontal plane passing through the apex of laryngeal ventricle.

2.ANATOMICALLY-Superior arcuate line where the squamous epithelium and respiratory epithelium meet.

Thus the roof of ventricle and saccule are included in supraglottis and floor belongs to glottis.

The marginal zone comprises of

1.Suprahyoid epiglottis

2.Aryepiglottic folds

It is clinically important, because of

Aggressive clinical behavior of cancer arising in this area.

There is lack of embryologic separation from the adjacent hypopharynx and it carries the worst prognosis among laryngeal cancers.

Mucous glands are abundant in saccule and periarytenoid area.

Early lymphatic spread of supraglottic cancer is because of rich vascularity and lymphatics associated with these glands.

GLOTTIS

This includes :

1.True vocal cords

2.Anterior commissure

3.Posterior commissure

The glottis extends from the lateral angle of ventricle to the upper border of the cricoid cartilage.Lower limit of glottis is controversial and the commonly accepted level is horizontal plane passing 1cm below the free margin of the vocal cords at the anterior commissure and 0.5cm below the posterior commissure.Some authors feel that the lower limit of the glottis is horizontal plane 20mm below the anterior commissure.

Lamina propria of the vocal cords consists of three layers.The superficial layer is composed of loose fibrous tissue that makes the Reinke's space.The intermediate and deep layers consist of elastic and collagenous fibers that make the vocal ligament.

Conus elasticus is a membrane which extends from superior border of cricoid cartilage to merge with inferomedial surface of vocal ligament.It resists the extralaryngeal spread of glottis and subglottic cancers.

SUBGLOTTIS

It extends inferior to glottis to lower border of cricoid cartilage.

This is a rare site of origin of cancers of larynx. But may be involved in glottic cancers.

Subglottic malignancy has higher incidence of extralaryngeal spread.

REASONS

Proximity of cricothyroid membrane.

Rich postcricoid lymphatics.

COMPARTMENTS OF LARYNX

Reinke's space

The mucosa over the vocal cord is attached loosely to the vocal cord ligaments themselves.There is a submucosal space along most of the length of free edge of the vocal cord extending from superior arcuate line to inferior arcuate line. Blood vessels and lymphatics are almost absent in Reinke's space preventing early spread of cancers.

Supraglottic area

It lies beneath the supraglottic mucosa superficial to quadrangular membrane.

Subglottic area

This area extends from inferior margins of true cord to inferior rim of cricoid. It is a potential space filled with fibroelastic submucosal tissue between mucosa and conus elasticus.It does not include the vocalis muscles and is limited superiorly at anterior commissure by the anterior commissure tendon.

Cricoid area

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

PRE-EPIGLOTTIC SPACE/SPACE OF BOYER

Boundaries: 1.Anterior

Thyrohyoid membrane

Thyroid cartilage above thyroepiglottic ligament

2,Superior

Hyoepiglottic ligament

Mucosa of the vallecula

3.Posterior

Infrahyoid epiglottis

Thyroepiglottic ligament

This space is continuous laterally with paraglottic space deep to the quadrangular membrane and superior to the ventricle.

Clinical importance

Cancer on laryngeal surface of the infrahyoid epiglottis spreads readily into the pre-epiglottic space.

Paraglottic space

This space is situated lateral to the ventricle and glottis.Involvement of this space by malignancy causes fixation of the vocal cord and is considered as advanced stage of cancer.

Boundaries:

Anterolaterally: thyroid cartilage and cricothyroid membrane

Superomedially: quadrangular membrane

Inferomedially: conus elasticus

Posteriorly: anterior reflection of pyriform sinus mucosa.

It blends with pre epiglottic space anterosuperiorly.

The submucosa of ventricle is continuous with para glottic space which is bounded by conus elasticus inferomedially, quadrangular membrane superomedially, thyroid ala laterally. The posterior limit of paraglottic space is the mucosa of the pyriform sinus. Inferolaterally the paraglottic space is continuous with the cartilaginous defect between thyroid and cricoid cartilage.

Paraglottic space has great significance in determining the spread of cancer within the larynx.Tumour involving the ventricle invades the paraglottic space and later spreads transglottically.

Vocal cord tumours which extend deeply into thyroarytenoid muscle which invade the paraglottic space,which later on extends to subglottis and extra laryngeal spread.

Lateral supraglottic tumours can travel lateral to ventricle along the inner surface of thyroid ala and thus spread subglottically.

The close proximity of pyriform sinus mucosa to the posterior paraglottic space makes this a potential route for spread of pyriform sinus carcinoma into the endolarynx resulting often in fixation of hemilarynx.

Nerve supply

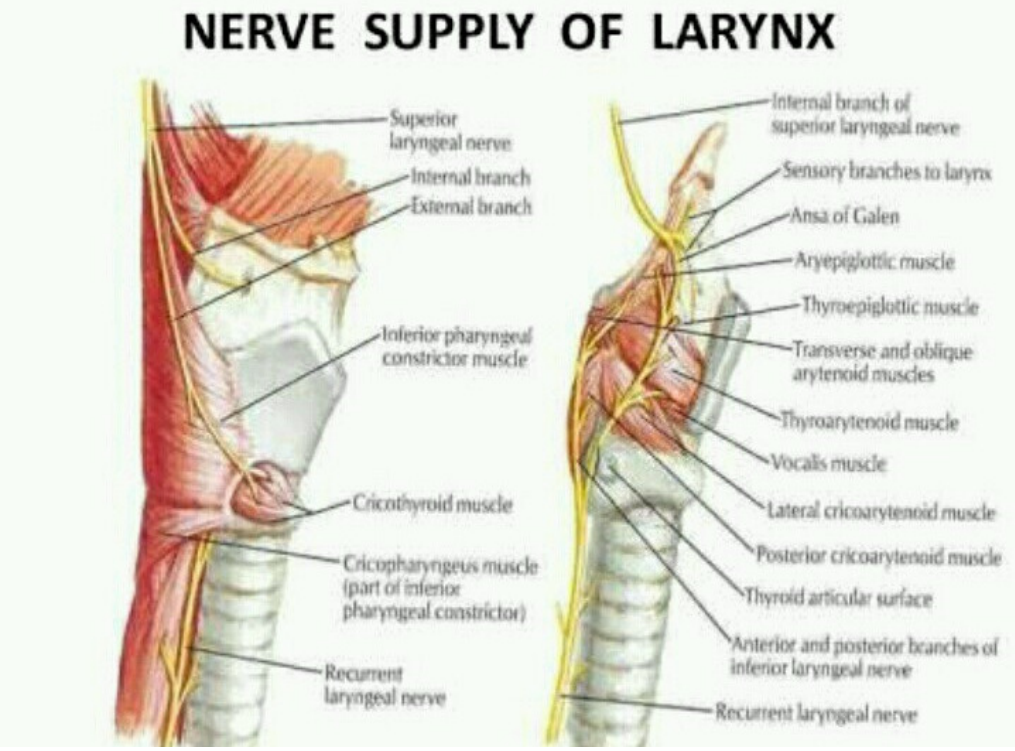
Sensory nerve supply:

Supraglottis and upper surface of the vocal cords: Internal branch of superior laryngeal nerve.

Subglottis and lower surface of vocal cords: Recurrent laryngeal nerves.

Motor nerve supply:

Intrinsic muscles:



This figure shows nerve supply of larynx.

All muscles except cricothyroid muscle: Recurrent laryngeal nerve

Cricothyroid Muscle: External branch of superior laryngeal nerve

Extrinsic muscles:

Ansa cervicalis: It is a branch of hypoglossal nerve.

BLOOD SUPPLY

Arterial and venous supply

Superior laryngeal branch of Superior thyroid artery and it supplies the paraglottis.

Inferior laryngeal branch of Inferior thyroid artery supplies the subglottis and the undersurface of vocal cord.This accompanies recurrent laryngeal nerve.Some blood supply comes through the cricothyroid branch of superior thyroid artery.

LYMPHATIC DRAINAGE

The Lymphatics of the two sides hardly communicate with each other. Thus restricting the tumour spread in laryngeal cancers.

Supraglottis: The lymph vessels pierce the Thyrohyoid membrane and drain into nodes level II and III (jugulodigastric and jugulo omohyoid).Ventricular lymphatics also pass through Cricothyroid membrane and ipsilateral thyroid gland to L3 and L4 nodes.

Subglottis: The lymphatics form three main trunks.One superficial trunk (anterior) pierces the cricothyroid membrane and drains to the Delphian (prelaryngeal) node which in turn drains into pre and paratracheal and supraclavicular nodes.

The two posterolateral trunks penetrate the cricotracheal membrane and terminate in paratracheal node and superior mediastinum.The nodes drain into deep cervical nodes.

Glottis:

This is the ''watershed area"- with poor lymphatics. Anterior commissure drains into the prelaryngeal (Delphian node).

III. PHYSIOLOGY

Larynx performs many functions and its primary function is to protect the lower respiratory tract. Phonation is also an important function of the larynx which facilitates communication. The functions are enumerated as follows;

Protection of tracheobronchial tree is facilitated by:

Three tier sphincter action

Laryngeal elevation – mylohyoid muscle

Laryngeal tilting –stylopharyngeous muscle

Cricopharyngeal sphincter relaxation occurred.

Cough reflex

Cessation of respiration

RESPIRATION: Reflex adjustments of the glottis apparatus plays a role in the mechanism of respiration which contributes to the regulation of acid base balance.

To increase intrathoracic pressure: this is done for fixation of the chest by glottic closure.This is essential for straining , climbing etc..

PROTECTION OF LOWER RESPIRATORY TRACT

Phylogenetically this is the most primitive function of larynx.This protective function is carried out by the closure of laryngeal inlet. That

occurs during swallowing by a three tier sphincter action.There is closure of laryngeal vestibule by contraction of the aryepiglottic and the interarytenoid muscles. The ventricular bands approximate which constitute the second tier.The third tier is by the adduction of the vocal cords by contraction of the adductors.The epiglottis plays a negligible role in closure of the laryngeal vestibule.

The cough reflex plays an important role in expelling foreign particles entering the tracheobronchial tree.

PHONATION;

The phonatory function of the larynx has developed with the evolution.It occurs at the time of inspiration when the vocal cords are approximated following adduction and the air escapes through causing vibration of the vocal cords.These vibratory tones are articulated by various structres in the oral cavity and resonated by the pharynx, nasal/oral cavity and paranasal sinuses to produce speech.

The pitch of the voice is determined by the number of vibrations of the vocal cord per second.The length and volume are determined by the capacity of the lungs.The quality of speech is dependent on the resonators as described. The articulators of voice include the lips, gums, teeth, tongue, palate and the jaws.

INTENSITY AND DURATION of voice depends on the respiratory bellows and subglottic pressure built up by adduction of the cords.

Pitch depends on the vibratory mass of vocal folds,laryngeal adductors and tensors.

Timbre depends on resonators and articulators[3,4].

IV.DIAGNOSIS AND STAGING

1. **History taking**

Detailed history of risk factors like Alcohol,Tobacco Chewing, Smoking, Genetic predisposition, Symptomatology like FB sensation, Dysphagia,Dyspnea,voice change etc…

2. **Clinical examination**

Pallor,metastatic neck nodes,Bocca's sign etc…

3. Direct laryngoscopy

Gold standard investigation, used to asses the three dimensional extension, and staging/ biopsy taking.

4. Biopsy under anesthesia

Punch biopsy,excision biopsy.

5. Imaging modalities like CT, MRI, X-RAY STNL,

CT scan used to asses the extension and staging especially laterally and anteroposteriorly. MRI is superior to CT.

6. Indirect laryngoscopic examination

IDL is used to visualize the hidden areas of larynx.Used to asses the site of lesion,extension and staging.

7. Video laryngoscopic examination

Used to assess the lesion by illumination and magnification.

8. Video stroboscopy

Mainly using for vibratory mucosal wave pattern vocal cord.

STAGING

Staging is used mainly for treatment and predict prognosis.

Most commonly used staging is TNM staging system.T- stage represent the extent of primary disease. N- stage represent the extent of regional lymph node metastasis.

M- stage represent the presence or absence of distant metastasis.

Histological grade, status of post surgical residual growth, lymphatic invasion, venous invasion are considered as important factor for prognosis.

DISTANT METASTASIS (M)

Laryngeal cancers normally metastasize to the lungs. Skeletal or hepatic metastasis occur less often. Mediastinal lymph node involvement is considered as distant metastasis[5].

For all sites	
'T' Stage	
T_x	Primary tumor cannot be assessed.
T_0	No evidence of primary tumor
T_{is}	Carcinoma in situ
T_1	Tumor limited to one subsite of supraglottic with normal vocal cord mobility.
T_2	Tumor invades mucosa of more than one adjacent subsite of supraglottis or Region outside the supraglottis (e.g mucosa of base of tongue, vallecula, Medial wall of pyriform sinus) without fixation of the larynx.
T_3	Tumor limited to larynx with vocal cord fixation and/or invades any of the following postcricoid area, or pre-epiglottic tissues, paraglottic space, and or minor thyroid cartilage erosion.
T_{4a}	Tumor invades through the thyroid cartilage and/or invades tissues beyond the larynx (e.g. trachea, soft tissues of neck including deep extrinsic muscle of the tongue, strap muscles, thyroid or esophagus.
T_{4b}	Tumor invades prevertebral space, encases carotid artery, or invades mediastinal structures.
Glottis	
T_1	Tumor limited to the vocal cord(s) (may involve anterior or posterior Commissure with normal mobility)
T_{1a}	Tumor limited to one vocal cord.
T_{1b}	Tumor involves both vocal cords
T_2	Tumor extends to supraglottis and or sub glottis, or with impaired vocal cord mobility.
T_3	Tumor limited to the larynx with vocal cord fixation, and/ or invades paraglottic space and minor thyroid cartilage erosion.

T_{4a}	Tumor invades through the thyroid cartilage and or invades tissues beyond the larynx, (e.g trachea, soft tissue of neck including deep extrinsic muscle of the tongue, strap muscles, thyroid, or esophagus.
T_{4b}	Tumor invades through the thyroid cartilage and/ or invades tissues beyond the larynx (e.g. trachea, soft tissues of neck including deep extrinsic muscle of tongue, Strap muscles, thyroid or esophagus.
T_{4b}	Tumor invades prevertebral space, encases carotid artery or invades mediastinal structures.
Subglottis	
T_1	Tumor limited to the sub glottis
T_2	Tumor extends to vocal cord(s) with normal or impaired mobility.
T_3	Tumour limited to larynx with vocal cord fixation
T_{4a}	Tumor invades cricoid or thyroid cartilage and or invades tissues beyond the larynx(e.g trachea, soft tissues of neck including deep extrinsic muscles of the tongue. and the thyroid or esophagus.)
T_{4b}	Tumor invades prevetebral space encases carotid artery or mediastinal structures.

'N' Stage	
Regional lymph nodes(N)	
N_x	Regional lymphnodes cannot be assessed.
N_0	No regional lymph node metastasis
N_1	Metastasis in a single ipsilateral lymph node, 3 cm or less in greatest dimension
N_2	Metastasis in a single ipsilateral lymph node, more than 3cm not more than 6 in greatest dimension, or in multiple ipsilateral lymphnodes none more than 6cm in greatest dimension or in bilateral or contralateral lymphnodes, none more than 6cm of greatest dimension.
N_{2a}	Metastasis in a single ipsilateral lymph node more than 3cm but not more than 6 cm in greatest dimension
N_{2b}	Metastasis in multiple ipsilateral lymph nodes, none more than 6 cm in greatest dimension

N_{2c}	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

'M' Stage	
Distant metastasis (M)	
M_S	Distant metastais cannot be assessed
M_0	No distant metastasis
M_1	Distant metastasis

The disease is staged as shown

Stage 0	T_{is}	N_0	M_0
Stage I	T_1	N_0	M_0
Stage II	T_2	N_0	M_0
Stage III	T_3	N_0	M_0
	T_1	N_1	M_0
	T_2	N_1	M_0
	T_3	N_1	M_0
Stage IVA	T_{4a}	N_0	M_0
	T_{4a}	N_1	M_0
	T_1	N_2	M_0
	T_2	N_2	M_0
	T_3	N_2	M_0
	T_{4a}	N_2	M_0
Stage IVB	T_{4b}	Any N	M_0
	Any T	N_3	M_0
Stage IVC	Any T	Any N	M_1

V. AETIOLOGY

1.Alcohol consumption

2.Tobacco chewing

3. Smoking

4.Radiation

5.Genetic

6.Virus and

7.Environmental Factor

VI.TREATMENT DECISIONS IN LARYNGEAL CANCER

Early stage including stage I /II :

1. Radiotherapy
2. Stripping
3. Open partial laryngectomy
4. Endoscopic CO_2 laser surgery
5. Conservative laryngeal surgery

Advanced stage including stage III /IV :

1. Near total laryngectomy
2. Total laryngectomy

3.Supracricoid laryngectomy

4.Supraglottic laryngectomy

Along with neck node clearance.

Non surgical treatment : 1.Radiotherapy

2.Chemotherapy

Treatment Aims :

1.Maximum cure rate

2.Voice preservation

3.Preserve nasal respiration

VII. VARIOUS SURGICAL TREATMENT OPTIONS

SUPRAGLOTTIC CANCER

T-stage, site within the supraglottis, 'N' status, age and pulmonary status are the important determinants of the choice of treatment.

A. T_1, T_2 Supraglottic cancer :

Treatment options include:

a) Endoscopic resection

b) Radiation therapy

c) Open partial laryngectomy

Transoral microlaryngoscopic resection with the CO_2 laser :

Indicated in T1/T2 stage of supraglottic tumour

a) Day care surgery

b) No need tracheostomy

c) It preserve voice,swallowing

d) It is easier to perform when the lesion is at free margin of epiglottis or along the aryepiglottic folds.

e) Pre-epiglottic space can be approached by this route

f) Cure rate is 85 -100%

g) Neck dissection is carried out same sitting or 4-5 days later

h) Alternative option is open partial laryngectomy[6,7,8,9]

b) Radiotherapy

Indicated in the following conditions:

1. T1 and small T2 Lesion

2 Lesion at the free border of epiglottis/along the aryepiglottic folds

3 . Larger tumour

4.Infrahyoid epiglottic tumour

5.Endoscopic resection is not possible in any reason.[10,11]

c) Open paritial laryngectomy is indicated in the following conditions

1. T2 lesions
2. Lesions of infrahyoid epiglottis
3. Larger metastatic neck nodes (N2a/N3)
4. Laser resection not possible for any reason

Types of open partial laryngectomy :

I.Horizontal supraglottic partial laryngectomy

It is adviced in the following conditions

1. T1 stage/ small T2 stage
2. Bulky T2 lesions
3. Glotto-supraglottic tumour –lesion extending to anterior commissure

II.The supracricoid partial laryngectomy with crico-hyoidopexy

B. T_3 SUPRAGLOTTIC CARCINOMA

Stage T3 includes

Invade into pre-epiglottic space But cords are mobile,

Lesions where cords are fixed But arytenoids are free

Supraglottic cancer with hemilarynx fixity

No cartilage erosion or Extra laryngeal soft tissue invasion

Treatment options include:

1. Chemo-radiotherapy / Radiotherapy
2. Voice conservation surgery
3. Total Laryngectomy

i. Chemo-radiotherapy/ Radiotherapy

Non surgical organ preservation therapy.

Concurrent chemoradiotherapy includes cisplatinum,5flurouracil.

Radiotherapy 66-70Gy in 6 ½-7 weeks

The chances of success with radiotherapy alone is in low volume T3 tumour <6ml[11,12].

2. Voice Conservation Surgery

The surgical organ preservation plan is particularly recommended in

i. Young patients when one prefers avoiding radiotherapy

ii. Those who either cannot tolerate chemotherapy or refuse chemo radiotherapy plan.

A number of voice conservation surgical procedures are possible in selected subsites of T_3 Supraglattic Cancers.

a. Horizontal partial laryngectomy is recommended when the tumour is T_3 by virtue of invasion of the pre-epiglottic space, But vocal cords are freely mobile and the patient is medically fit to withstand the problems of postoperative aspiration.

b. Supracricoid partial laryngectomy (SPCL) with cricohyoidopexy is indicated in :

T3 supraglottic cancer and extends to glottis

Cord fixity and arytenoid mobile

Subglottic extension minimal

The only problem is aspiration.

c.**Three-quarter laryngectomy** is recommended in

T3 supraglottic cancer extended to glottis and spilling over medial wall of PFS across the AE fold.

Subglottic extension minimal, Arytenoid mobile and free,

Apex of pyriform sinus is free of disease

Transoral endoscopic resection with the CO_2 laser is indicated in the following situations :

1.When vocal cords are freely mobile.

2.CT scan shows early invasion of pre-epiglottic space.Major advantanges like avoidance of tracheostomy,minimal physiological stress, minimal hospitalization, preserve functioning arytenoid, preventing aspiration,allowing good quality of speech.[13,14,15,16]

c. Near-total laryngectomy (NTL) is suitable in the following conditions

1. Lateralised lesions of the supraglottis with fixity of the hemilarynx.

2.Procedure preserves good,lung powered speech,aspiration is minimal.

3.Need permanent tracheostomy[17].

3.**Total laryngectomy.**

It is indicated in T_3 supraglottic cancers with fixity of the hemilarynx and involvement of the interarytenoid region or more than one-third of the contralateral vocal cord.

Postoperative radiation therapy is recommended in T_3 supraglottic cancer. Management of the neck in T_3 supraglottic cancer is very important. because of the high propensity to lymph node metastasis.

C. **T4 SUPRAGLOTTIC CANCER**

This is indicated in cartilage erosion or gross extra laryngeal soft tissue invasion.

Surgery followed by radiation therapy is the treatment of choice.

1. Cases with lateralised lesions are amenable to near-total laryngectomy.

2.Only in a selected cases with mobile arytenoids, minimal subglottic extension and minimal thyoid cartilage erosion will the procedure of supracricoid partial laryngectomy with CHP be feasible.

NATURAL BARRIERS OF SUPRAGLOTTIS

1.Overlying perichondrium of thyroid and cricoid cartilage

2.Ventricle

3.Conus elasticus

4.Quadrangular membrane

5.Thyrohyoid membrane

6.Hyoepiglottic ligament.

As the disease advances and breaks through the barriers of the anatomical compartments, wider resections become necessary. The range of partial laryngectomy procedures has been broadened over the years to deal with these more extensive lesions. Since such resections have to ensure that the remnant larynx still maintains its functions of phonation, nasal respiration and airway protection, a basic functioning unit (cricoarytenoid joint) has to be left behind. Here the importance of the cricoid, the arytenoid and the crico-arytenoid must be recognized vis-à-vis laryngeal functions. Equally important is the sensory innervation of the laryngeal remnant.

The cricoid is the rostrum on which the entire larynx rests. It is the only portion of the larynx with a complete signet ring of hyaline cartilage that helps maintain the airway. Resection of a major portion of this cartilage will result in larynx col1apse, blocking the airway. The arytenoid cartilage or rather, the crico-arytenoid segment of the cricoid cartilage , is essential, as the basic functioning unit of the larynx, for the production of speech and for prevention of aspiration.

ADVANTAGES OF LARYNGEAL REMNANT

1.To preserve the voice

2.To preserve the respiration

3.To prevent the aspiration

4.To protect the airway

In order that the larynx retains its sphincteric action, the laryngeal remnant must be innervated. Preservation of the recurrent laryngeal nerves on both the sides ensures sensory supply to the subglottic region and motor innervations of the glottis. For the sensory innervation of the supraglottic region, particularly over and arytenoids, it is important to preserve the main trunk and the posterior descending branch of the internal laryngeal division of the superior laryngeal nerve.

Preservation of the main trunk of the superior laryngeal nerve does not prevent complete resection of the pre epiglottic and paraglottic spaces. The posterior descending branch can be preserved during tumour resection by leaving it attached to the previously elevated, and preserved mucosa of the pyriform sinus. A branch of this nerve anastomoses with an ascending branch of the recurrent laryngeal nerve & a twig from this anastomosis penetrates to supply the posterior laryngeal mucosa

Following extensive resections, some form of construction is at times necessary to reconstitute a functioning glottic aperture. The aim is to ensure that the anteroposterior diameter of the larynx is maintained so that there is no stenosis and that the posterior glottic bulk, normally provided by the arytenoids, is reconstructed to prevent aspiration and produce good quality of voice. Hence, when the glottis is resected across the anterior commissure, a silicone keel is placed, temporarily separating the two sides to prevent a web formation with consequent stenosis. The resected arytenoids is generally replaced with wether a piece of throid cartilage or with the strap muscles to provide the posterior glottis bulk

The tumour extent may be found to be greater than that assessed preoperatively. So that an alternative, more extended procedure is utilized to tackle the lesion adequately. At times, findings at surgery may reveal that none of the partial laryngectomy procedures is safe and the surgeon may have to resort to total laryngectomy. It is mandatory, therefore, to have a written consent for total layngectomy prior to undertaking any conservative procedure of the larynx.

Partial laryngectomy as surgical salvage of radiation failure is not contraindicated, but certainly calls for a lot of caution to be exercised. Preoperative evaluation must confirm that the initial lesion prior to radiation therapy was suitable for a conservation procedure, That the recurrence is on the same site as before and that the recurrence fulfils all the eligibility criteria required for the particular conservation procedure to be performed[18,19,20,21].

CONVENTIONAL SUPRAGLOTTIC LARYNGECTOMY

Surgery is performed under general anaesthesia. If necessary, a preliminary tracheostomy is performed. A horizontal incision is placed at the level of the thyroid cartilage, from one sternomastoid to the other. The sternohyoid and sternothyroid muscles are transected along the superior border of the thyroid cartilage exposing the latter. The

peichondrium of the cartilage is incised along the upper border and reflected downwords over the upper half of the thyroid cartilage. It is important to save the perichondrium as it helps in the closure, on completion of the supraglottic laryngectomy. The inferior constrictor muscle is divided along the upper half of the postero lateral border of the thyroid cartilage on the dominant side of the in all these steps great care is taken not to damage the superior laryngeal nerve along the neurovascular pedicle. The perichondrium from the inner surface of the thyroid cartilage is elevated only postero-laterally to free the pyriform mucosa if tumour extension to this site.

CLEARANCE OF THE PRE-EPIGLOTTIC SPACE,

In early tumours with little or no infiltration of the pre epiglottic space, the entire hyoid can be preserved by subperiosteal dissection of the pre epiglottic space which is resected along with the tumour. Preserving the hyoid allows a more secure closure and early rehabilitation. With gross infiltration of space, at least the body of the hyoid or the entire hyoid is resected to show satisfactory clearance of the pre epiglottic space. It is very important to preserve the sensory supply, particularly over and around the order to avoid distressing and aspiration consequently a turbulent post-operative period. It is vital that the

superior laryngeal nerve and the posterior descending branch of its internal division are preserved on both side.

Preservation of this important nerve is compatible with an oncologically safe supraglottic laryngectomy[22].

RESECTION OF THE TUMOUR

1.Prelimnary tracheostomy performed.

2.Entry into the vallecula by transvallecular approach

3.Epiglottis is grasped with an allis forceps and retracted downwards

4.Pharyngostome is enlarged and excellent view of tumour and larynx was exposed.AE folds are divided well anterior to arytenoids on both sides.

5.Resection continued inferiorly through ventricles and preserve true cords.Remove both false cords completely under direct vision with adequate free margin.

6.Lateralized lesions, to preserve the uninvolved supraglottic tissue on the contralateral side.Resection based upon the anatomical line with an endeavor to preserve only the arytenoids.

RECONSTRUCTION

1. Cricopharyngeal myotomy may be performed to facilitate post operative swallowing.
2. Closure of the defect is commenced by suturing the cut edges of the pyriform mucosa below, to the oropharyngeal mucosa above, starting laterally and progressing towards the center.
3. This is not necessary if a classical supraglottic laryngectomy is done with preservation of the pyriforms. As the region of the resected supraglottis is approached, primary mucosal apposition is no longer possible.
4. Closure is now obtained by approximating the upper end of the remaining thyroid cartilage to the base of the tongue. This is achieved by using three 1-0 sutures that are passed through the thyroid cartilage inferiorly and the base tongue musculature superiorly.
5. If hyoid is preserved during the pre epiglottic space clearance, the sutures pass around the hyoid superiorly to give a more secure closure.
6. The thyroid perichondrium which was preserved,is now sutured to the base tongue musculature as the second layer of closure.

7. EXTENDED SUPRAGLOTTIC LARYNGECTOMY

The horizontal supraglottic laryngectomy can be extended to include resection of the involved arytenoid, the pyriform, or the valleculae with the adjacent base of the tongue.

(a) Arytenoid Resection:

1. Arytenoid cartilage removed partially or completely, to avoid damage to RLN during disarticulation of cricoarytenoid joint.
2. Following resection,posterior glottic bulk is deficient which leads to aspiration,poor quality of speech.
3. To avoid this,remnant of vocal cord is medialised and suturing on the superior border of cricoid cartilage by non absorbable suture.
4. Adequacy is checked by stimulating the trachea with suction catheter thereby initiating cough reflex and glottic closure.
5. Raw area of posterior glottis is closed by pyriform sinus mucosa and extensive resection of endolaryngeal tissue is replaced by cartilage and muscle.

(b) Resection of the tongue base/valleculae:

It is indicated in the following conditions

1. Lesions invoving lingual surface of epiglottis, vallecula and adjacent area of tongue base along with supraglottic larynx.
2. Tumour primarily from the supraglottis to involve the oropharynx

3. Primary oropharyngeal tumour with secondary involvement of supraglottis.
4. At least one half of tongue base along with its blood supply must be preserved. Direct closure of the defect is difficult due to loss significant amount of soft tissue, a pectoralis major myocutaneous flap is used.

(c) Resection of the lateral wall of the Pyriform fossae:

Resection of the lateral wall of the pyriform fossa along with involved portions of the lateral and posterior pharyngeal wall is compatible with the extended supraglottic laryngectomy. Closure of the defect however requires a myocutaneous flap.

VIII. NECK DISSECTION

MANAGEMENT OF THE NECK IN LARYNGEAL CANCER

The status of the cervical lymph nodes is the single most important factor that influences survival in squamous cancers of the head and neck, and cancer of the larynx is no exception. In fact, a higher clinical 'N' stage significantly increases the risk of distant metastasis in squamous cell cancer of the larynx[23,24].

INCIDENCE & PATTERN OF CERVICAL NODE METASTASIS

The supraglottis has a rich lymphatic network and mitoses to the cervical nodes are frequent. Between 23-50% of all supraglottic cancers present with cervical lymph node involvement. 40% of patients may develop contra lateral or bilateral neck metastases[25]. However, this risk of contra lateral node metastasis is significant only when the ipsilateral nodes are involved. Treatment failure in supraglottic cancer is often a result of regional recurrence rather that local disease. Even early supraglottic tumors (T_1/T_2) are at significant risk for neck metastasis.[26,27,28] And demand serious consideration for treatment of the neck.[29,30]

The most common sites of cervical lymph node metastases from cancer of the larynx are the nodes along the internal jugular vein viz. levels, II, III and IV . In addition, lymph nodes at level VI, viz the pre-laryngeal, paratracheal and para tracheal cervical lymph nodes, are at risk in transglottic cancers and glotto subglottic cancers. Histological involvement of lymph nodes in the para tracheal region is documented in 20% of cases of advanced cancer of the larynx[31]. Metastases to lymph nodes at levels I and V, viz the supradigastric and the posterior triangle of neck, are very rare in laryngeal cancer. It is the pattern of lymph node involvement that is the basis for selectively targeting lymph nodes at levels, II, III. IV and VI, when dealing with a clinically 'N' neck in the presence of locally advanced laryngeal cancer[32,33,34].

As the larynx is a midline organ, it is importance to understand the risk of lymph node metastases to both sides of the neck, in lesions that are well lateralized. The risk to the contra lateral neck is very low if the ipsilateral neck is 'N' this is true even if the lesion has crossed the midline but the lymph nodes on the dominant side are negative for metastases. If however, the ipsilateral lymph nodes are positive for metastases, the risk to the contralaterl neck nodes is significant.

MANAGEMENT

Treatment of the neck in laryngeal cancers depends upon:

The site of the primary;

a) The T- stage of the primary;

b) The clinical 'N' stage

c) The choice of treatment modality for the primary.

The clinically 'N_0' neck in supraglottic Cancer

Management of the 'N_0' neck in supraglottic cancer is largely influenced by the choice of treatment for the primary. The following guidelines are suggested.

1. If the primary tumor is treated with radiation, both sides of the neck are included in the field of radiotherapy, regardless of the stage of the disease.
2. If an early primary is resected endoscopically with the CO_2 laser, the 'N_0' neck is not treated selectively. Instead, close follow-up is maintained clinically and with the color doppler for early detection and treatment of metastatic neck nodes.
3. When open partial laryngectomy is performed for an early supraglottic cancer, The neck is entered and therefore, Lymph node sampling and frozen section examination is carried out. This is done for lymph nodes at level II, III on the ipsilateral side for lateralized

lesions, and bilaterally for midline lesions. If on frozen section there is presence of metastasis a selective infradigastic (levels II,III,IV,V) neck dissection is performed. Very low incidence of metastasis at level I, justifies sparing of this region. Even though the incidence of metastatic at level V is also low, it is advisable to clear this region to enable a more satisfactory and comprehensive infradigastric clearance as a block dissection. It is not necessary to clear lymph nodes at level VI in early supraglottic cancer.

If the sampled nodes do not show metastasis on frozen section, the neck is observed. If however, metastasis is revealed on subsequent paraffin section, the neck is treated postoperatively with radiation therapy.

4. If a near – total or total larygectomy is performed for a locally advanced supraglottic cancer, the clinically 'N^0' neck is treated with bilateral selective neck dissection clearing levels II, III and IV in addition to this, on the ipsilateral side, level VI is also cleared. The incidence of para tracheal node metastasis (level VI) in advanced laryngeal cancer has been shown to be as high as 20% with majority of these nodes less than 1cm in diameter and appearing clinically negative. The neck will need to be treated postoperatively with

radiation therapy if the final histopathology shows multiple lymph node metastasis or perinodal infiltration.

The clinically N_+ neck is supraglottic cancer :

Neck dissection is indicated in the following conditions

1. Metastatic neck node is large (N2a/N3)
2. N status >3cm in size.

   N status <3cm in size, treatment of choice is radiotherapy or chemoradiotherapy alone.
3. N1 or N2a disease, selective infradigastric neck dissection sparing level I.
4. N2b or N3 disease, Radical neck dissection adviced,if paratracheal nodes are included in the block dissection ,total or near total laryngectomy indicated.
5. Contralateral neck dissection done for level II, III, IV, if contralateral neck node is positive.
6. In bilateral metastatic neck node, atleast one internal jugular vein preserved in minimally involved side.

1. Clinicopathological study of 50 cases of tumours of larynx.

Dinesh kumar sharma. Barjinder singh sohal.M.S.Bal., Sangeetha Aggarwal., 2011, studied prevalence of benign tumours was more amongst labourers (25%),followed by business community (20%) and housewives(20%). The business community consisted of shopkeepers or milkmen.

Malignant tumour were highest (36.6%) amongst labourers,followed by farmers(13.3%) and businessmen (10%).

SITE OF LARYNGEAL TUMOURS ;

Site of growth	Benign tumours	Malignant tumours
Supraglottic	25%	50%
Glottic	70%	20%
Subglottic	5%	0%
Transglottic	0%	30%

Some patients had extensive growths involving more than one region, e.g, supraglottic and pyriform fossae(20%), supraglottic and glottis(6.6%), and subglottic(3.3%)[35].

1. Thompson Ld, Wenig Bm, Heffner Dk, And Gneep Dr (1999), Exophytic and Papillary squamous cell carcinomas of the larynx; a Clinicopathologic series of 104 cases.Otolaryngology head and surgery 120; 718-724pp.

Thompsons et al.(18) studied 104 cases of Exophytic and Papillary SCCs of larynx out of which 30% were Supraglottic,46% Glottis,3% Subglottic and 21% Transglottic[36].

2. Bakshi et al.(3) in a study of 690 cases of Laryngeal malignancy found that 56% of tumours were supraglottic, 17% glottis,3.6% subglottic and 13% transglottic tumours[37].

The malignant tumours of the larynx occur more commonly in the supraglottis which is consistent with the studies of Bakshi et al. But not with that of Thompson et al (18).The present study is in line with both the above studies the subglottis is the least common site.

4.James A. Koufman MD, and Alan J.Burke, MBBS Studied the Etiology and pathogenesis of laryngeal carcinoma[38].

In the united states,Laryngeal cancer accounts for approximately 1% of all new cancer diagnosis but fewer than 1% of all cancer deaths[39]. Laryngeal cancer is usually diagnosed relatively early Because It alters the sensitive phonatory and airway functions of larynx; 60% of patients present with localized disease alone, 25% with local disease and regional nodal metastatic disease, and 15% with advanced disease or distant metastases or both.

Squamous cell carcinoma is the predominant type of Laryngeal cancer, Accounting for over 90% of cases, and laryngeal SCC accounts for 26% of all cases of head and neck SCC.Undifferentiated carcinoma and verrucous

carcinoma are major variants of SCC; Lymphoepithelial carcinoma and spindle cell carcinoma are less common[40].

Most patients who develop laryngeal SCC are men,and the overall male to female ratio for this disease is approximately 5;1 [41].However, since the 1950s,an increasing incidence of Laryngeal SCC has been observed in black male patients and in all female patients(both white and black).This increase in women from 0.5 to 1.5 per 100000 population[42] is particularly significant, and It may relate to the increased use of tobacco and ethanol by women during that period. By the mid -1980,the overall 5-year survival rates for all patients with Laryngeal SCC had improved to approximately 67%,But since then they have remained unchanged[43].

The rate of metachronous SCC disease of the upper aerodigestive tract, including the larynx is reported to be between 5% and 35% of cases[44,45,46,47].The esophagus is the most common second site, and consequently,It has been postulated that gastroesophageal reflux may be a carcinogenic co factor.Changing patterns of disease with time and exposure to comparable risk factors in different countries of the world have helped elucidate a group of common carcinogenic variables that are now being associated with a broader,more multifactorial hypothesis of Laryngeal carcinogenesis[48].

This multifactorial theory is supported by observations that mucosal carcinoma is often multicentric a phenomenon often called field cancerization. This phenomenon is usually attributed to the panmucosal carcinogenic influences of a variety of a environmental factors.

Although many of the presumed carcinogenic influences have been shown to alter the internal environment,not one has been proved to cause carcinoma.Virtually all of the available data is associative that is epidemiologic[49].This is even true for well established causes of head and neck carcinoma such as tobacco and ethanol.

6. Head And Neck; Laryngeal Tumours; An Overview studied in 2008-2012 , Glottis carcinomas represent the majority of Laryngeal cancers (50 -60%), followed by the supraglottic carcinomas (30-40%), while the subglottic carcinomas are uncommon (5% or less).

Prognosis; the overall 5- year survival following treatment is 80% for glottis and 50% for supraglottic tumours,mainly because the latter present an increased incidence of nodal metastases.Nearly two –thirds of patients with supraglottic cancers have neck metastases at the time of initial treatment.Survival decreases by more than one third when clinically positive lymph nodes are present.Five year disease free survival of patients with supraglottic cancer is 80% for stage 1-11, 70% for stage 111 and 40 % for stage 1V.Patients with glottis cancers have a better long-term prognosis. Five year disease free for stages 1-11 is 85%-90%, for stage 111 is 75% and for

stage 1V is 45%-50%.However, in cases with an extremely advanced local and regional disease. The overall 5-year survival is less than 5%[50].

7. Laryngeal biopsies with special reference to malignant tumours; A histopathological studied by K P varalakshmi,V., Shivashankar naik, R., Sujeeva swapna,p., sravani, M., neeraja.

A total of 95 patients were included in this study[51].

Results from that study ,Out of 95 Laryngeal biopsies, 25 biopsies from inflamed larynx,49 from neoplastic growths, 8 from laryngeal nodules,2 from laryngoceles,2 showed no significant lesions, 1 from the infected cyst ,And 8 were inadequate biopsies.Out of 41 malignant tumours ,39 were squamous cell carcinoma,basaloid squamous type and adenocarcinoma one each.A highest number of malignant tumours were seen between 41 and 60 years.

SUBSITE DISTRIBUTION OF MALIGNANT TUMOURS OF LARYNX;

SITE	NUMBER OF CASES (%)
Supraglottis	31.7%
Glottis	65.8%
Subglottis	2.4%
Transglottic	0%

AGE –WISE DISTRIBUTION OF MALIGNANT TUMOURS OF LARYNX;

AGE IN YEARS	NO OF MALIGNANT TUMOURS
0-20	Nil
21-40	21.9%
41-60	60.9%
61 and above	17.07%

GRADING OF SQUMOUS CELL CARCINOMA

GRADES	NO OF CASES %
I	25.6%
II	83.8%
III	7.6%
IV	0%

In this study, dysplasia of grade II was seen in only one biopsy.The relation between the chronic laryngitis and malignant transformation of such larynx is well known since 1923.[52,53,54]Proper follow-up provides greater information about the fate of such larynx. Most of the members (91.3%) in this study were smokers, And many studies were also noticed strong association of chronic non-specific laryngitis with tobacco smoking.[55,56]

INCIDENCE OF SCC IN VARIOUS STUDIES

STUDY	PERCENTAGE OF SCC %
Kaufman and Burke	90
Kumar et al	95
Domanowski	96
Jaiswal and Hoang	99
Wang et al	99
Bakshi et al	69
Non –keratinizing SCC	27
Keratinizing SCC	

3. Clinico Pathological Study of Laryngeal Masses Studied By Dr.kiran jayawant shinde, Dr.syed imdad Husain hashmi, Jan-2015.

Results from that study, total 100 cases of laryngeal masses were obtained, 86 malignant and 14 benign. Among malignant laryngeal masses supraglottic region was the commonest site and majority patients presented in late stageIII &IVA .Among benign laryngeal masses vocal fold polyps were the commonest. Clinical diagnosis was 100% correct in vocal fold nodule,intubation granuloma and epiglottic cyst whereas it was correct in only 66.66% cases of vocal fold polyp and 98.83% of malignant laryngeal masses.This study showed that proportion of laryngeal cancers was significantly high,They constituted a major burden

in our hospital with proportion higher than that reported in any other studies which requires prompt attention.We can be conservative with vocal fold nodules and cysts. But we should be aggressive in the management of vocal fold polyps. [57]

4. Epidemiological study of laryngeal carcinoma in western Nepal done by Krishna koirala.

This is a retrospective study carried out in the Department of ENT, Manipal Teaching Hospital, and Pokhara, Nepal from 1st August 2012 to 31st December 2014.

Results from that study,One hundred and one histopathologically confirmed new cases of laryngeal squamous cell carcinoma were included in our study. Age of the patients ranged from 40 years to 92 years with the mean age of 64.16 years. The mean age of females was 58.5 years And that of males was 65.5 years. Twenty-Eight of our patients belonged to less than 60 years while seventy-three patients belonged to age range of more than 60 years. Forty one percent of our patients belonged to the age range of 60-69 years. Males accounted for 81% and females accounted for 20% of total patients. Male to Female ratio was 4:1 . Fifteen of our patients had transglottic carcinoma due to difficulty to locate the exact site of origin of transglottic malignancies, It was not taken into account for the sub-site comparison groups. Final

comparison was made amongst 86 patients of supraglottic, glottic and subglottic subsites of the larynx.

Out of 86 patients, 61 patients (70%) had supraglottic carcinoma, 24 (28%) had glottic carcinoma and one patient had subglottic carcinoma. Supraglottic larynx was the commonest subsite of larynx to develop the disease . All of our patients were smokers whereas 67 patients used to drink alcohol also. Out of 61 patients of supraglottic carcinoma, 46 used to smoke and drink and 15 used to smoke only. Out of 24 patients of glottic carcinoma, 11 patients used to smoke and drink whereas 13 patients used to smoke only. There was significant association between smokers and drinkers and smokers alone in supraglottic and glottic cancers (p=0.009) . This observation shows that supraglottis is the commonest subsite of larynx to harbor squamous cell carcinoma and squamous cell carcinoma of supraglottis is common in people who smoke and drink alcohol[58]

Number of Patients According to Age Range and Sex

Age Range	Males	Females	Total Number of patients
40-49 yrs	7	6	13
50-59 yrs	13	2	15
60-69 yrs	34	7	41
70-79 yrs	19	5	24
>80 yrs	8	0	8
Total	**81**	**20**	**101**

Subsite Distribution of Laryngeal Cancers

Subsite	No. of patients
Supraglottic	61
Glottic	24
Subglottic	1
Transglottic	15
Total	**101**

Comparision of Laryngeal Cancers amongst Smokers and Alcohol Consumers

Subsites	Smokers and alcohol users	Smokers	Total
Supraglottis	46 (75%)	15 (25%)	61
Glottis	11 (46%)	13 (54%)	24
Total	57	28	85

5. A study by Raquel Ferreira et al (2013) also supports the role of alcohol as a risk factor for developing cancer of the oral cavity,pharynx,esophagus,somach,larynx,colorectum,central nervous system,pancreas,breast and prostate[59].

6. Thapa et al.(2003) in a retrospective chart review of 8 month duration in Nepal medical college,Kathmandu,Nepal,found that Supraglottic carcinoma was commoner than glottis carcinoma.It is difficult to draw a conclusion from their study due to small number of patients[60].
7. In a study performed by koirala K et al.(2013),Supraglottic larynx was reported to be the commonest subsite of larynx (78.2%) to harbor laryngeal malignancy[61].
8. In a study done by jukka et al.(2000) in finland from 1974 to 1995, Glottic carcinoma was marginally in higher position than supraglottic carcinoma.This fact was documented to be due to increased use of alcohol as well as smoking in the study population[62].
9. Mohanty BK et al.(2002) have stated that Supraglottic cancer is more common than glottis in indian subcontinent. This variation in demographics might be due to the higher prevalence of chewing tobacco rather than smoking in indian subcontinent which results in tobacco rich saliva coming in contact with the supraglottis.In contrast, Smoke from tobacco smokking has more contact with the glottis in the way to lungs[63].

RESULTS

HISTOPATHOLOGY					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	SOUAMOUS CELL CA	6	6.0	6.0	6.0
	SQUAMOUS CELL CA	94	94.0	94.0	100.0
	Total	100	100.0	100.0	

Moderately Differentiated Squamous Cell Carcinoma

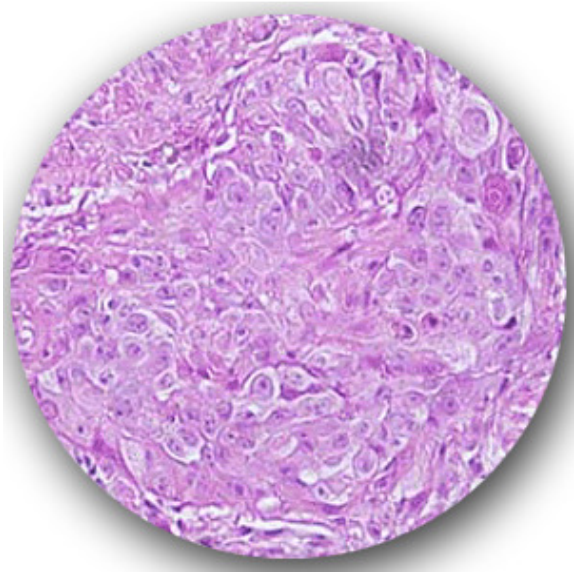


Figure – Alveolar Pattern

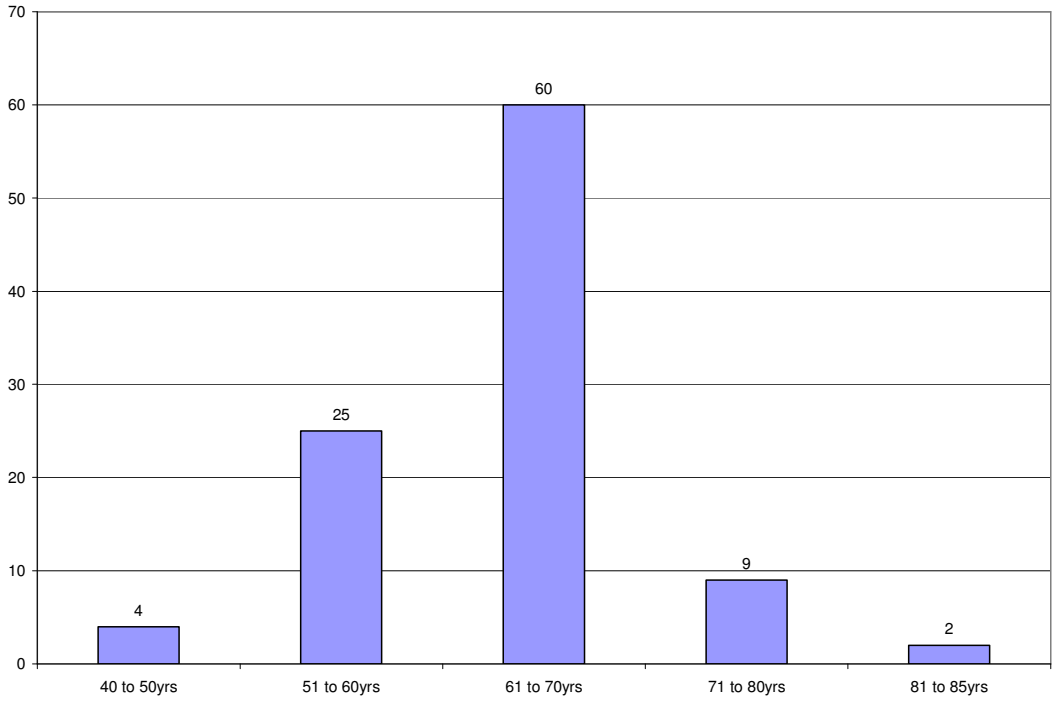
Descriptive Statistics

Variables	No	Min.	Max.	Mean	S.D
Age	100	40	85	63.61	6.828
Duration of exposure	100	18	40	29.45	5.496

AGE

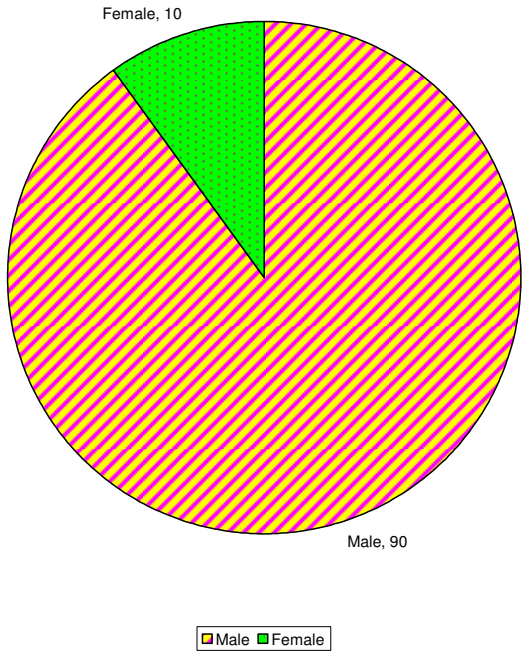
Particulars	Frequency	Percentage
40 to 50yrs	4	4.0
51 to 60yrs	25	25.0
61 to 70yrs	60	60.0
71 to 80yrs	9	9.0
81 to 85yrs	2	2.0
Total	100	100

In our study more than 50% cases occur in above 60 years.



SEX

Particulars	Frequency	Percentage
Male	90	90.0
Female	10	10.0
Total	100	100.0

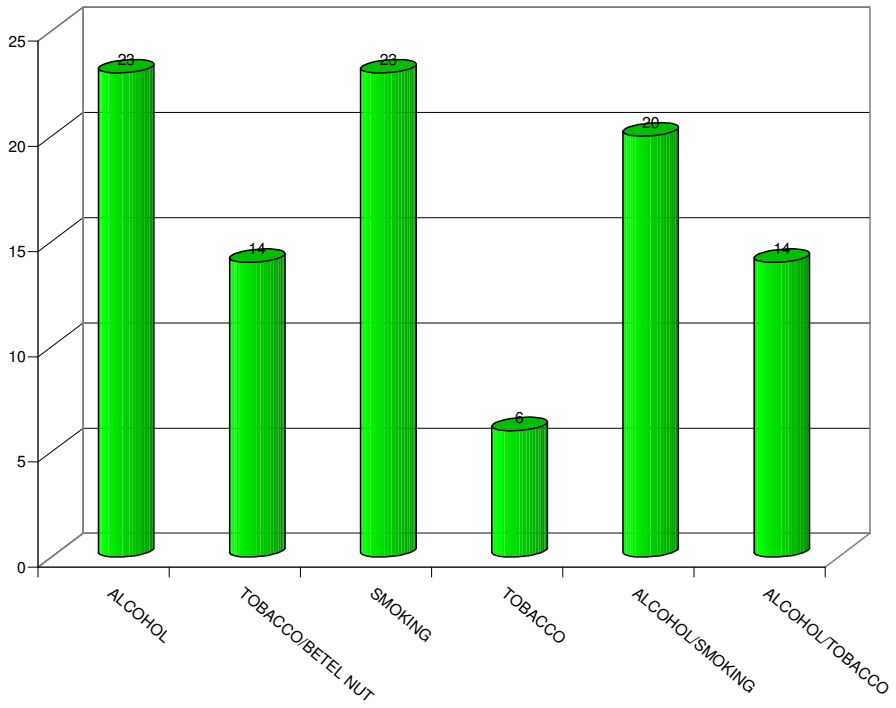


In our study out of 100 cases 90 Cases were males 10 Cases were female.

RISK FACTORS

Particulars	Frequency	Percentage
Alcohol	23	23.0
Tobacco / Betel Nut	14	14.0
Smoking	23	23.0
Tobacco	6	6.0
Alcohol / Smoking	20	20.0
Alcohol / Tobacco	14	14.0
Total	100	100.0

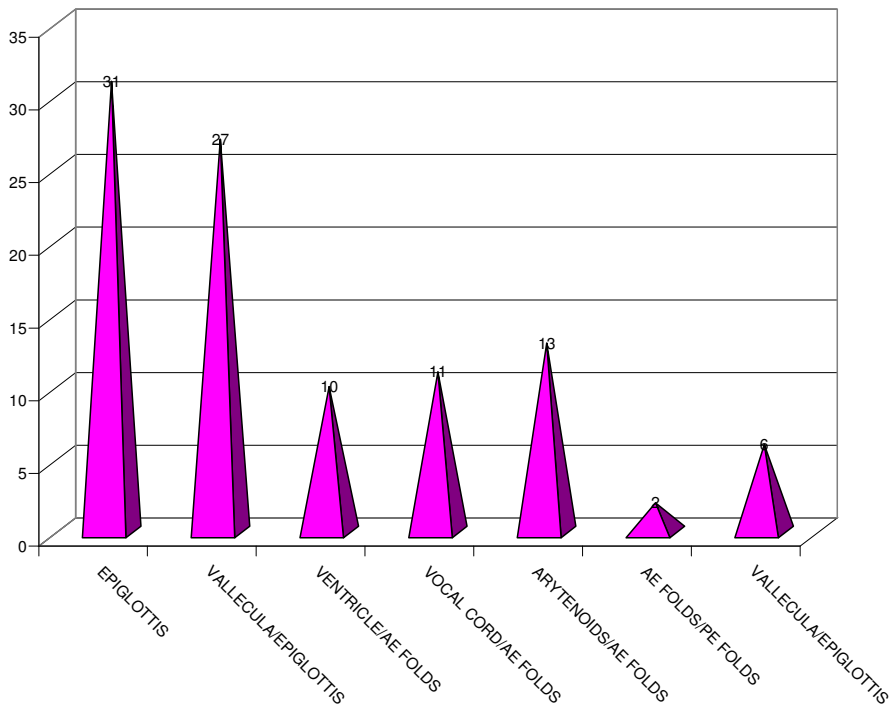
Out of 100 cases 46 Cases affected by alcohol and smoking equally,Least number of cases affected by tobacco alone.



SUB SITE

Particulars	Frequency	Percentage
Epiglottis	31	31.0
Vallecula / Epiglottis	27	27.0
Ventricle / AE Folds	10	10.0
Vocal Cord / AE Folds	11	11.0
Arytenoids / AE Folds	13	13.0
AE Folds / PE Folds	2	2.0
Vallecula / Epiglottis	6	6.0
Total	100	100

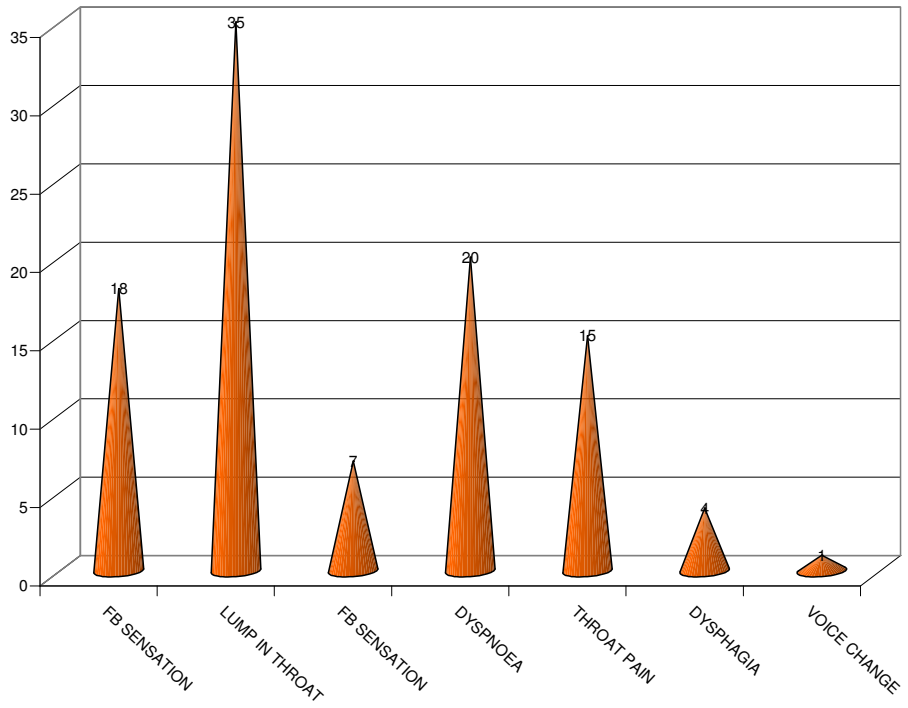
Most commonly affected subsite was Epiglottis (31%).



SYMPTOMS

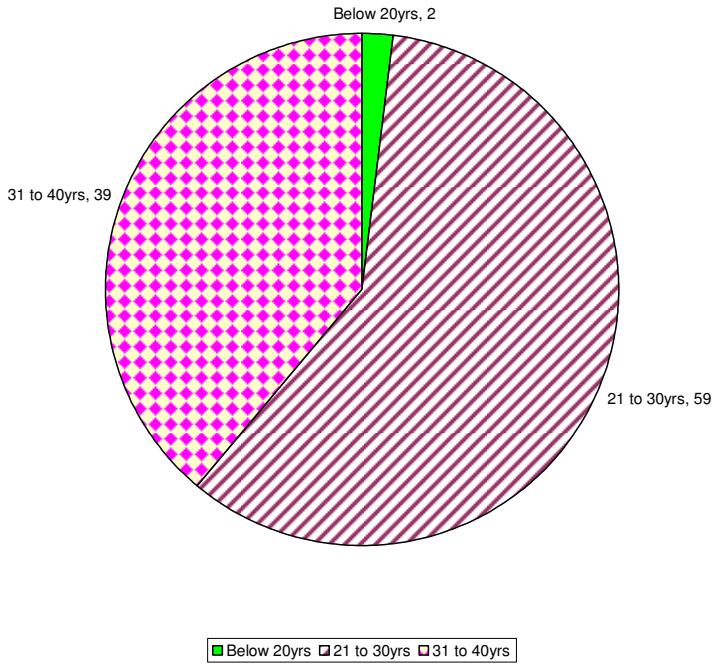
Particulars	Frequency	Percentage
FB Sensation	18	18.0
Lump in throat	35	35.0
FB Sensation	7	7.0
Dyspnoea	20	20.0
Throat Pain	15	15.0
Dysphagia	4	4.0
Voice Change	1	1.0
Total	100	100

Most common symptom of supraglottic tumour was lump in throat (35%).



DURATION OF EXPOSURE

Particulars	Frequency	Percentage
Below 20yrs	2	2.0
21 to 30yrs	59	59.0
31 to 40yrs	39	39.0
Total	100	100.0



Chi-square test

SUB SITE	Age												Statistical inference
	40 to 50yrs		51 to 60yrs		61 to 70yrs		71 to 80yrs		81 to 85yrs		Total		
	n	%	*N*	%	*n*	%	*n*	%	*n*	%	*n*	%	
Epiglottis	3	75.0 %	11	44.0%	17	28.3%	0	.0%	0	.0%	31	31.0%	X^2=25.406 Df=24 .384> 0.05 Not Significant
Vallecula / Epiglottis	0	.0%	6	24.0%	17	28.3%	3	33.3%	1	50.0%	27	27.0%	
Ventricle / AE Folds	1	25.0 %	2	8.0%	6	10.0%	1	11.1%	0	.0%	10	10.0%	
Vocal Cord / AE Folds	0	.0%	1	4.0%	9	15.0%	1	11.1%	0	.0%	11	11.0%	
Arytenoids / AE Folds	0	.0%	5	20.0%	4	6.7%	3	33.3%	1	50.0%	13	13.0%	
AE Folds / PE Folds	0	.0%	0	.0%	2	3.3%	0	.0%	0	.0%	2	2.0%	
Vallecula / Epiglottis	0	.0%	0	.0%	5	8.3%	1	11.1%	0	.0%	6	6.0%	
Total	**4**	**100%**	**25**	**100%**	**60**	**100%**	**9**	**100%**	**2**	**100.**	**100**	**100%**	

Chi-square test

SUB SITE	Male		Female		Total		Statistical inference
	N	**%**	***N***	**%**	***N***	**%**	
Epiglottis	29	32.2%	2	20.0%	31	31.0%	
Vallecula / Epiglottis	22	24.4%	5	50.0%	27	27.0%	
Ventricle / AE Folds	10	11.1%	0	.0%	10	10.0%	
Vocal Cord / AE Folds	11	12.2%	0	.0%	11	11.0%	X^2=17.277 Df=6 .008<0.05 Significant
Arytenoids / AE Folds	13	14.4%	0	.0%	13	13.0%	
AE Folds / PE Folds	2	2.2%	0	.0%	2	2.0%	
Vallecula / Epiglottis	3	3.3%	3	30.0%	6	6.0%	
Total	**90**	**100.0%**	**10**	**100.0%**	**100**	**100.0%**	

In our descriptive study,by using Chi square test , p- value is .008 which is less than 0.05 .So our study is significant.

Chi-square test

SUB SITE	Risk Factors														Statistical inference
	Alcohol		Tobacco /Betel Nut		Smoking		Tobacco		Alcohol/Smoking		Alcohol/Tobacco		Total		
	N	*%*	*n*	*%*	*N*	*%*	*n*	*%*	*N*	*%*	*N*	*%*	*n*	*%*	
Epiglottis	18	78.3%	2	14.3%	1	4.3%	6	100.0%	3	15.0%	1	7.1%	31	31.0%	X^2= 109.187 Df=30 .000< 0.05 Significant
Vallecula / Epiglottis	4	17.4%	7	50.0%	2	8.7%	0	.0%	7	35.0%	7	50.0%	27	27.0%	
Ventricle / AE Folds	0	.0%	0	.0%	6	26.1%	0	.0%	4	20.0%	0	.0%	10	10.0%	
Vocal Cord / AE Folds	0	.0%	0	.0%	7	30.4%	0	.0%	4	20.0%	0	.0%	11	11.0%	
Arytenoids / AE Folds	1	4.3%	1	7.1%	5	21.7%	0	.0%	1	5.0%	5	35.7%	13	13.0%	
AE Folds / PE Folds	0	.0%	0	.0%	2	8.7%	0	.0%	0	.0%	0	.0%	2	2.0%	
Vallecula / Epiglottis	0	.0%	4	28.6%	0	.0%	0	.0%	1	5.0%	1	7.1%	6	6.0%	
Total	23	100%	14	100%	23	100%	6	100%	20	100.0%	14	100%	100	100%	

Chi-square test

SUB SITE	Duration of exposure								Statistical inference
	Below 20yrs		21 to 30yrs		31 to 40yrs		Total		
	n	*%*	*N*	*%*	*N*	*%*	*N*	*%*	
Epiglottis	1	50.0%	27	45.8%	3	7.7%	31	31.0%	X^2=48.912 Df=12 .000<0.05 Significant
Vallecula / Epiglottis	0	.0%	13	22.0%	14	35.9%	27	27.0%	
Ventricle / AE Folds	0	.0%	6	10.2%	4	10.3%	10	10.0%	
Vocal Cord / AE Folds	0	.0%	8	13.6%	3	7.7%	11	11.0%	
Arytenoids / AE Folds	0	.0%	4	6.8%	9	23.1%	13	13.0%	
AE Folds / PE Folds	1	50.0%	0	.0%	1	2.6%	2	2.0%	
Vallecula / Epiglottis	0	.0%	1	1.7%	5	12.8%	6	6.0%	
Total	2	100.0%	59	100.0%	39	100.0%	100	100.0%	

In our stu dy Epiglottis was involved in 31% of cases,Next commonly involved subsite was vallecula and epiglottis.

DISCUSSION

Supraglottic tumour is the most common laryngeal malignant tumour next to glottic cancer ,In our study out of 100 cases 90 Cases were male and 10 Cases were female. Most commonly affected age group were 61to70 year(60%), 51 to 60 years(25%),71to 80 years(9%),40to 50 years(4%),81 to 85 years(2%), in that order of frequency. The most common age group was 61 to 70 years (60%), and least common age group was 81 t0 85 years(2%).

According to the gender, Males were more commonly affected .The ratio was 9:1 , 90% Cases were male, Remaining 10% of cases were female.

The most common risk factor for supraglottic cancer was alcohol.And Least one was tobacco alone. Alcohol (23%), smoking (23%),alcohol/smoking (20%) ,tobacco/betel nut(14%), alcohol/tobacco (14%), tobacco(6%).in that order of frequency.

Accoding to the subsites,Epiglottis alone most commonly involved(31%),Least commonly involved AE folds/PE folds(2%). Next Vallecula/Epiglottis(27%), Arytenoids/AE folds (13%),Vocal cord /AE folds(11%), Ventricle/AE folds (10%),Vallecula/Epiglottis(6%), in that order of frequency.

The most common symptom of supraglottic tumour was lump in throat (35%), and Least common common symptom was voice change(1%).Next dyspnoea(20%), throat pain(15%), foreign body sensation(7%), dysphagia (4%), in that order of frequency.

Based upon Thompson et al.(18) 30% were Supraglottic,(46%) glottis,(3%) subglottic, and transglottic(21%).

Bakshi et al.(3) ,56% were supraglottic,17% glottis,3.6 % subglottic, and 13% transglottic tumours.

Exposure of risk factor to subsites were below 20 years for 2%, 21 to 30 years for 59%, 31 to 40 years for 39%, in that order of frequency. Maximum risk of exposure was 20 to 30 years. And minimum below 20 years.

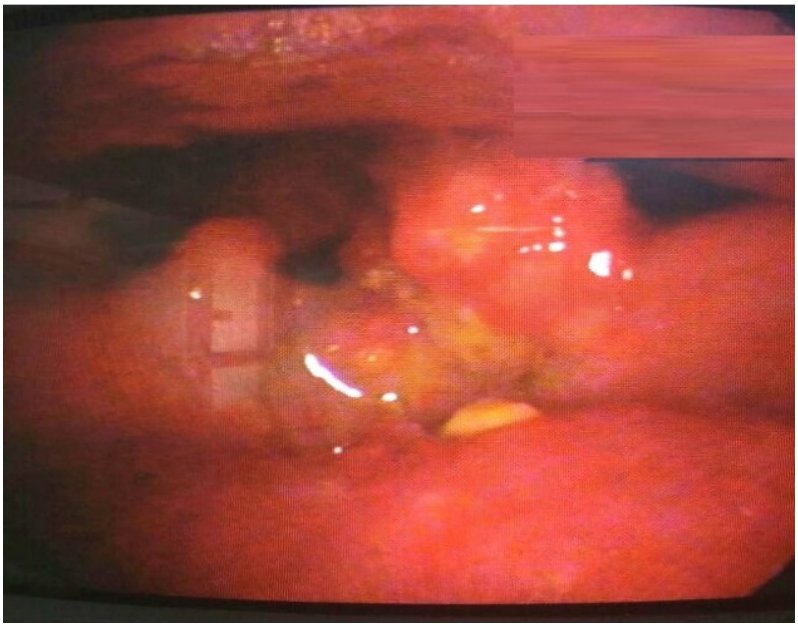
In our descriptive study ,Relationship between the subsite and age was X^2 =25.406, Df =24 So, The p- value was 0 .384>0.05 ,So This is not significant. P-value was > 0.05

The relationship between the subsite and gender was X^2=17.277 , Df =6 , 0 .008 < 0.05 ,So This study was significant. P-value was <0 .05

The relationship between causative risk factor and Subsite X^2 =109.18 ,Df =30 , 0.000<0.05 , So This study was significant.

P- value was <0.05.

The relationship between the subsites and duration of risk factor exposure was analysed, by using Chi square test and the results derived are as X^2 =48.912, Df =12 , 0.000< 0.05 This study was significant. P- value was <0.05



VLE-Supraglottic growth

SUMMARY

Supraglottic tumour is the most common malignant neoplasm next to glottic carcinoma in India.For which Treatment is surgery ,radiotherapy and chemotherapy.In advanced cases Palliative care is only advised.

This descriptive study was conducted examining 100 patients in the department of otorhinolaryngology, Thanjavur medical college hospital,Thanjavur from june 2016 to October 2017.

The disease was more common in elderly male age group 40-60 years.

The most common symptoms were lump in throat,followed by dyspnea,foreign body sensation,throat pain,and dysphagia. All patients were subjected to histopathological examination, radiological examination, videolaryngoscopic examination, and relevant clinical examination & history taking.,Every patient was subjected to HPE, videolaryngoscopic examination, -site of lesion,nature of lesion, and its extension were noted.In all cases primary and all associated symptoms were noted.All other relevant clinical data like age,sex and all type of risk factors were noted.

In our descriptive study ,relationship between the subsite and age was X^2 =25.406, Df =24 So, The p- value was 0 .384>0.05 ,So This is not significant. P-value was > 0.05

The relationship between the subsite and gender wasX^2=17.277 , Df =6 , 0.008 < 0.05 ,So This study was significant. P-value was < 0 .05

The relationship between causative risk factor and Subsite X^2 =109.18, Df =30 , 0.000<0.05 , So This study was significant. P- value was <0.05. The relationship between the subsites and duration of risk factor exposure was analysed as X^2 =48.912, Df =12 , 0.000< 0.05 This study was significant. P- value was <0.05

CONCLUSION

Most commonly affected age group was 61to70 year(60%),and 51 to 60 years(25%), 71to 80 years(9%) , 40to 50 years(4%), 81 to 85 years(2%) , in that order of frequency. The most common age group was 61 to 70 years (60%), And the least common age group was 81 t0 85 years(2%).

According to the gender, Males were more commonly affected .The ratio was 9:1 ,

The most common risk factor for supraglottic cancer was alcohol.And the least one was tobacco used alone. Alcohol (23%), smoking (23%), alcohol&smoking (20%) , tobacco&betel nut(14%), alcohol&tobacco(14%), tobacco used alone(6%).were found in that descending order of frequency.

Accoding to the subsites,Epiglottis alone was the most commonly involved(31%)site.,the least commonly involved site was AE folds/PE folds(2%). Next was Vallecula&Epiglottis(27%). Arytenoids&AE folds (13%),Vocal cord &AE folds(11%), Ventricle&AE folds (10%),Vallecula&Epiglottis(6%), were found in that descending order of frequency.

The most common symptom of supraglottic tumour was lump in throat (35%), And the least common common symptom was voice change(1%).Next dyspnoea(20%),throat pain(15%),foreign body sensation(7%),dysphagia(4%),were found in that descending order of frequency.

In our descriptive study ,by using Chisquare test, the relationship between the subsite and age was X^2 =25.406, Df =24 So, The p- value was 0 .384>0.05 , This is not significant. P-value was > 0.05

By using chisquare test,the relationship between the subsite and gender was X^2=17.277 , Df =6 , 0 .008 < 0.05 ,So This study was significant. P-value was <0 .05

By using chisquare test,the relationship between Causative risk factor and Subsite X^2 =109.18 ,Df =30 , 0.000<0.05 , So This study was significant. P- value was <0.05

By using chisuare test, the relationship between the subsites and duration of risk factor exposure was analysed as X^2 =48.912, Df =12 , 0.000< 0.05 this study was significant. P- value was <0.05

Supraglottic tumour is a common condition in our region.This variation in demographics might be due to the higher prevalence of chewing tobacco ,consumption of alcohol,and smoking which results in tobacco rich saliva coming in contact with the supraglottis.Supraglottic laryngeal carcinoma is in its increasing prevalence in developing countries because of the practice of consumption of alcohol and smoking.The poor overall five year survival rate was also due to supraglottic cancer and its presentation at late stage.Public awareness towards the avoidance of alcohol and smoking will reduce the risk of development of supraglottic laryngeal cancers and head and neck cancers overall in the developing countries.So our aim of - Prevention of supraglottic tumour-can be achieved by

1 .by educating the public about the causative factors,

2. urging the public to avoid the risk factors,

3. To creating the awareness of symptomatology which helps in early diagnosis and hence complete cure.

BIBLIOGRAPHY:

1.Dhingra PL ,And Shruti Dhingra., " Diseases of Ear,Nose and Throat and Head and Neck Surgery".6th Edn.,Elsevier.,2014:282-286pp.

2.John C Watkinson and Ralph W Gilbert,."Stell and Maran's Textbook of Head and Neck Surgery and Oncology ".5th Edn.,Hodder Arnold, : 645-655pp

3.Chaurasia BD.,"Human Anatomy Regional and Applied".Vol.3,3rd Edn.,CBS Publishers And Distributers.,1998:203-210pp.

4.Hazarika P, Nayak DR, And Balakrishnan R., "Text book of Ear,Nose,Throat and Head and Neck surgery clinical and practical ".3rd Edn.,CBS Publishers and Ditributers.,2013:567-578

5.Pittsburg H.,Kirchner J.,"Clinical vs Histalogical staging in laryngeal cancer Arch Otolaryngol". 1979:105-157pp

6.Ambrosch P.,Kron M.,Sieiner W., 1998 "Carbon dioxide laser microsurgery for early supraglottic carcinoma Ann oto Rhinol 107:680-688pp.

7.Davis RK.,Kelly SM.,Hayes J.,1991,"Endoscopic co_2 laser excisional biopsyof early supraglotticcancer.Laryngoscope".113:132-138pp.

8.Davis RK.,Kriskovich MD.,Galloway EB.,Buntin CS., And Jepsen MC.,2004"Endoscopic supraglottic laryngectomy with postoperative radiation Ann otol Rhinol Laryngol". 113:132-138pp.

9.Eckel HF.,1998,"Endoscopic laser resection of supraglottic carcinoma Otolayngol Head Neck Surg".117:681-87pp.

10.Naktoor bM.,spiro IJ.,Wang CC.,Martins P.,Montgomery W., And Fabian R.,1998,"Results of accelerated radiotherapy for supraglottic carcinoma:A Massachusetts General Hospital and Massachusetts Eye and Ear Infirmary Experience.Head Neck".20(5):379-384pp.

11.Mendenhall WM.,Parsons JT.,Mancuse AA et al.,1996,"Radiotherapy For Squamous Cell Carcinoma Of The Supraglottic Larynx.An Alternative to surgery.Head Neck".18:24-35pp.

12.Freeman DE.,Mancuso AA.,Parsons JT et al.,1990,"Irradiation Alone For Supraglottic Larynx Carcinoma.Can CT Findings Predict Treatment Results? Int J Radiat Oncol Biol Phys.19:485-490pp.

13.Zeitels SM.,Vaughan CW.,1991,"Preepiglottic Space Invasion In Early Epiglottic Cancer.Ann Otol Rhinol Laryngol".100:789-792pp.

14.Iro H.,Waldfahrer F.,Steiner W et al.,1998,"Transoral Laser Surgery Of Supraglottic Cancer.Arch Otolaryngol Head Neck Surg". 124:1245-1250pp.

15.Steiner W.,Ambrosch P.,1996,"Laser Microsurgery For Cancer Of The Larynx.Mitat".5:159-164pp.

16.Steiner W.,Ambrosch P.,2000,"Endoscopic Laser Surgery of the Upper Aerodigestive tract.With Special Emphasis on Cancer Surgery.Stuttgart".New York:Georg Thieme.

17.Zeitels SM., Koufman JA.,Dvis RK.,Vaughan CW.,1994,"Endoscopic treatment of supraglottic and hypopharynx cancer.Laryngoscope".104:71-78pp.

18.Kirchner J.,Som ML.,1975,"The anterior commissure technique of partial laryngectomy:Clinical and laboratory observations.Laryngoscope".85:1308-1317pp.

19.Sessions DG.,Ogura JH.,Fried MP.,1975,"The anterior commissure in glottis carcinoma.Laryngoscope5:1624-1632pp.

20.Som ML.,Silver CE.,1968,"The anterior commissure technique of partial laryngectomy.Arch Otolaryngol".87:138-145pp.

21.McNaught RC.,1950,"The surgical correction of anterior web of the larynx.Laryngoscope".60:264-272pp.

22.Rassekh Christopher H et al.,1998,"Preservation of superior laryngeal nerve in supraglottic and supracricoid partial laryngectomy.The Laryngoscope". 108(3):445-447pp.

23.Matsuo JM et al.,2003,"clinical nodal stage is an independently significant predictor of distant failure in patients with squamous cell cancer of the larynx.Ann surg". 238(3):412-421pp,discussion 421-422pp.

24.Moe k., Wolf G T.,Fisher SG.,Hong WK.,1996,"Regional metastasis in patients with advanced laryngeal cancer.Arch Otolaryngeal Head Neck Surg".122(6):644-648pp.

25.Som M.,1970,"Conservation surgery for carcinoma of the supraglottis.J.Laryngol Otol". 84:655pp.

26.Desando LW.,Magrine C.,O'Fallon.,1990,"The second side of the neck in supraglottic cancer.Otol-HN Surgery". 102(4) 351-361pp.

27.Bocca E.,Pignataro O.,Oldini C.,1983,"Supraglottic laryngectomy 30 years of experience.An Otol Rhinol Laryngol". 92:14pp.

28.Lindberg R.,1972,"Distribution of cervical lymph nodes from squamous cell carcinoma of the upper respiratory and digestive tracts.cancer". 29:1446pp.

29.Shuller DE.,Bier-Laning CM.,1997,"Laryngeal carcinoma nodal metastasis and then management.Otolaryngol.Clin.North America bo (2):167-177pp.

30.Felito A., Silver CE.,Rhinaldo A.,Smith RV.,2000,"Surgical treatment of neck in cancer of the larynx. ORL J Otolaryngol.Relat.Spec." 62(4):217-225pp.

31.Timon CV., Toner M.,Conlon BJ.,2003,"Paratracheal lymph node involvement in advanced cancer of the larynx,hypopharynx and cervical oesophagus.Laryngoscope". 113(9):1595-9pp.

32.A dos Santos CR.,Goncalves FJ.,Magrin J.,Johnson LF.,Ferlito A.,Kowalshi LP.,2001,"Involvement of lower level I lymph nodes in advanced squamous cell carcinoma of the larynx.Ann Otol Rhinol Laryngol". 110(10):982-984pp.

33.Ferlito A.,Rinaldo.,1998," Level I dissection for laryngeal and hypopharyngeal cancer:is it indicated? J Laryngol Otol". 112(5):438-440pp.

34.Spriano G.,Piantanida R.,Pellini R.,Muscatello L.,2003,"Elective treatment of the neck in squamous cell carcinoma of the larynx:clinical experience.Head and Neck". 25(2):97-102X.

35.Dinesh K S., And Barjinder S S., 2013 "Clinico-Pathological Study Of 50 Cases Of Tumours Of Larynx",indian J otolaryngol head neck surgery 65:S29-S35.

36.Thompson L .D.,Wenig B.M., Heffner D.K.,Gnepp D.R., 1999,''Exophytic and papillary squamous carcinomas of the larynx a clinicopathologic series of 104 cases".Otolaryngol head neck surgery,120:718-724.

37.Bakshi J., Panda NK.,Gupta AK.,Mann SBS., 2004 ,"Survival patterns in treated cases of carcinoma larynx in north india: a 10 year follow up study".Ind J Otolaynngol Head Neck Sur 56(2):99-103

38.James A. Koufman., And Alan J.Burke.,1997,"The Etiology And Pathogenesis Of Laryngeal Carcinoma",otolaryngologic clinics of north America vol:30.

39.Rothman, KJ.,Cann, CI.,Flanders, W.,et al:"Epidemiology of laryngeal cancer".Epidemiol Rev2:195-209,1980.

40.Ferlito, A.,:"Histological classification of larynx and hypopharynx cancers and their clinical implications",Acta otolaryngol(Stockh)Suppl 342:8-38,1977.

41.SEER Cancer Statistics Review .,1973-1991(Gloeckler Ries LA.,Miller, BA.,Hankey, BF.,et al).surveillance program,Division of cancer Prevention and control,National Cancer Institute, Washington DC,1992.

42.Dorn, HF.,Cutler, SJ.,:" Mobidity can cancer in the united states. Part I. Variation in incidence by age, sex,marital status, and geographical region".Public health Monograph No.29,1995.

43.Cann, CI., Fried, MP.,Rothman, KJ.,:"Epidemiology of squamous cell cancer of the head and neck" .Otolaryngol Clin North AM 18:367-388,1985.

44.Healey, GB., Styrong, MS., Uchmakli, A., et al:"Carcinoma of the palatine arch:The rationale of therapeutic selection".Am J Surg 132:498,1978.

45.Mansel, RH.,Vermeersch, H.,:"Panendoscopy for second primaries in head and neck cancer".Am Laryngol soc Meeting,Vancouver,BC,May 1981.

46.McGuirt, WF., Matthew, B.,Koufman, JA.,:"Multiple simultaneous tumors in patients with head and neck cancer".Cancer(50):1195-1199,1982.

47. Shapshay ,SM., Hong, WK., Fried ,MP.,et al:"Simultaneous carcinomas of the esophagus ang upper aerodigestive tract".Otolaryngol Head and Neck Surg(88):373,1980.

48.Koufman JA:"The otolaryngologic manifestations of gastero esophageal efflux disease".Laryngoscope 101(suppl 53):1-78,1991.

49.Rothman, KJ., Causation and causal inference.In schottenfeld, D., Fraumeni, JFJ.,(reds):"Cancer Epidemiology and prevention" ,Philadelphiya WB Saunders,1982,pp 15-22.

50.Nikolaos S Mastronikolis et al.,2008-2012,"Head And Neck:Laryngeal Tumours: an overview"university of patras,Greece.

51.Varalakshmi KP., Sivashankar Naik., V.,Sujeeva Swapna R.,Sravani P., Neeraja,M.,2016,''Laryngeal Biopsies With Special Reference To Malignant tumours: A Histopathological Study''.International channel of scientific study,4:197-202.

52. Crissman, JD., "Laryngeal keratosis and subsequent carcinoma". Head Neck Surg 1979(1):386-91.

53. Gabriel, CE., Jones, DG.," Hyperkeratosis of the larynx". J Laryngol Otol 1962(76):947-57.

54. Norris, CM., Peale, AR., "Keratosis of the larynx". J Laryngol Otol 1963(77):635-47.

55. Auerbach, O., Hammond, EC., Garfinkel, L.," Histologic changes in the larynx in relation to smoking habit". Cancer 1970(25):92-104.

56. Lederer, FJ., Soboroff ,BJ.," Medical problems related to diseases of the larynx". Otolaryngol Clin North Am 1970(3):599-608

57. Shinde KJ and Hashmi SIH ., 2015 “Clinicopathological study of laryngeal masses’’.J of Dental and Medical sciences,14:61-79.

58.Krishna Koirala.,2015,”Epidemiological study of laryngeal carcinoma in Western Nepal”,Asian pacific Journal of Cancer Prevention, vol:16.

59.Raquel FM.,Anke B.,Luiz C,et al.,2013,”Alcohol consumption and risk of cancer:a systematic literature review.Asian Pac J Cancer Prev”.(14):4965-4972pp.

60.Thapa NM.,Jha A.K.,Rijal JP.,2003,”Study on head and neck tumors presented in ENT OPD of Nepal Medical College Teaching Hospital.NMC Journal,(5):79-81pp.

61.Koirala, K,. Sharma, V,. (2013).” Laryngeal malignancy: relation of tumor size and neck node metastasis”. Nepal J Medical Sciences,(2): 48-51

62.Jukka A V.,Pasi P H.,Eero J K,et al.,2000,”Is the subsite Distribution of laryngeal cancer related to smoking habits? Acta Oncologica,(39):77-79pp.

63.Mohanty BK.,Bahadur S.,Lal P.,2002,”Text book of Radiation Oncology-Principles and Practice,Elsevier,New Delhi :162-163pp.

ANNEXURE-I
PROFORMA

Case No: Hospital:

Name: IP/OP No.

Age: Ward:

Sex: Income:

Occupation:

Date of Admission:

Date of operation:

Date of discharge:

DIAGNOSIS

CHIEF COMPLAINTS

1.Foreign body sesation

2.Throat irritation

3.Dysphagia

4.Throat pain

5.Voice change

6.Lump in throat

7.Loss of weight

8.Loss of appetite

9.Neck swelling

HISTORY OF PRESENTING ILLNESS

1.H/o Dysphagia:

- Duration
- Onset Sudden/Insidious
- For solid/liquid
- Absolute/relative
- Aggravating/relieving factor

2.H/O VOICE CHANGE:

- Continuous/intermittent
- Onset Sudden/Insidious
- Hot potato/muffled voice
- Aggravating/relieving factor

3.H/o:Dyspnea

- Sudden /insidious onset
- Aggravating/Relieving factor factor
- Associated with vomiting,sweating,giddines

5.H/o Headache:

- Duration
- Onset-sudden/insidious
- Site- frontal/occipital/vertex/Bitemporal
- Unilateral/bilateral
- Nature of pain:Thhrobbing/aching/stabbing/tingling
- Relation to time
- Relation to posture
- Associated facial pain
- Associated nasal discharge
- Associated with watering of eye
- Associated with blurring of discharge
- Associated with vomiting /Nausea
- Aura:yes/no

- Photophobia:yes/no

6.H/o Epistaxis:

- Quantity
- Following steroid nasal spray:yes/no
- Spontaneous/traumatic
- Spontaneous/traumatic
- Unilateral/bilateral

7.Ear complaints:

- Deafness/ear block
- Discharge
- Referred pain

8.Throat complaints:

- Difficulty in swallowing/Pain while swallowing
- Foreign body sensation
- Frequent clearing of throat
- Throat pain
- Change of voice
- Itching in the throat

PAST HISTORY:

- Diabetes mellitus/Hypertension/seizure disorder/bronchial asthma/ prvious surgery/tuberculosis/ishemic heart disease.

PERSONAL HISTORY:

Mixed diet,

Bladder & bowel habbits

Alcohol consumption

Tobacco chewing

Smoking

FAMILY HISTORY:

Any other family member suffering from simillar illness

TREATMENT HISTORY:

Previous hospitalization

Drug allergy

GENERAL EXAMINATION:

General condition:

Conscious: Oriented:

Built: Nourishment

Pallor Jaundice:

Clubbing:Cyanosis:

Generalised pedal edema:

vital signs:

Pulse Rate: RR:

BP: Temperature:

SYSTEMIC EXAMINATION:

Cardiovascular system:

Central Nervous system:

Respiratory system:

GIT:

ENT EXAMINATION:

ORAL CAVITY:

- Mouth opening adequate
- Lips:Normal
- Gingivobuccal sulcus:
- Gums,teerh:Normal
- Orodental hygiene:Normal
- Floor of mouth:
- Tongue:

- Retromolar trigone:
- Palate:
- Anterior pillars:
- Uvula:
- Posterior pillars:
- Posterior pharyngeal wall:congested and granular
- Post nasal discharge:

INDIRECT LARYNGOSCOPIC EXAMINATION

Tongue base
Bilateral glossoepiglottic fold:
Bilateral valleculae
Epiglottis :deformed/not
Bilateral pharyngoepiglottic fold :
Bilateral pyriform fossae
Bilateral aryepiglottic fold : Ulceroproliferative growth
Bilateral arytenoids :mobile/fixed
Bilateral ventricular bands
Bilateral vocal cords : mobile/Fixed

HEAD AND NECK EXAMINATION

External laryngeal framework
Symmetrical/asymetrical
Trachea
Midline/deviated
Laryngeal crepitus
Present/absent
Thyroid cartilage prominence or not
Enlarged palpable neck node present
Or not if present, level
Size,site,number,side,matted or not,mobile/immobile,fixed with underlying/adjacent structures,consistency,ulcerated/fungated,tender, warmth.

NOSE AND PARANASAL SINUS EXAMINATION

External nasal framework

Collumella
Vestibule
Septum
Inferior turbinate and meatus
Middle turbinate and meatus
Floor of nasal cavity
Roof of nasal cavity

FUNCTIONAL TEST
Cotton wool test (movements) :
Cold spatula test(fogging) :

POST NASAL EXAMINATION
Adenoid :yes/no
Eustachian tube opening: normal/abnormal
Torus tubarious :normal/abnormal
Posterior end septum:
Posterior end of IT:
Posterior end of MT:
Lateral pharyngeal band:

PARANASAL SINUS EXAMINATION

Sinus tenderness :yes /no
Frontal sinus: unilateral/bilateral/absent
Maxillary sinuses:unilateral/bilateral/absent

EAR

	RT	LT
Preauricular area		
Pinna		
Post auricular area		
EAC		

Tympanic membrane
Mastoid

Facial nerve

TUNING FORK TEST

Rinne,s test :Negative/positive

Webers test :Lateralized/ not lateralized

Abc :Reduced/not reduced

ANNEXURE-II

CONSENT FORM

I Hosp.No……. in my full senses hereby give my complete consent for or any other procedure deemed fit which is a diagnostic procedure/biopsy/transfusion/operation to be performed on me /my son/my daughter/my ward aged under any anasesthesia deemed fit .The nature and risks involved in the procedure have been explained to me to my satisfaction.

For academic and scientific purpose the operation/procedure may be televised or photographed.

Date:

Signature/Thumb

Impression of patient/Guardian

Name:

Designation:

Guardian:

Relationship:

Full address:

S.NO	NAME	AGE	SEX	RISK FACTORS	SUB SITE	HISTOPATHOLOGY	SYMPTOMS	DURATION OF EXPOSURE
1	AROKIYASAMY	46	M	ALCOHOL	EPIGLOTTIS	SQUAMOUS CELL CA	FB SENSATION	21 YEARS
2	MUNIYASAMY	48	M	ALCOHOL	EPIGLOTTIS	SQUAMOUS CELL CA	FB SENSATION	22YEARS
3	MEENATCHI	74	F	TOBACCO/BETEL NUT	AE FOLDS/EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	25 YEARS
4	VEERA MUTHU	47	M	SMOKING	VENTRICLE/AE FOLDS	SQUAMOUS CELL CA	FB SENSATION	21 YEARS
5	VADIVEL	54	M	SMOKING	EPIGLOTTIS	SOUAMOUS CELL CA	FB SENSATION	21 YEARS
6	PITCHAI	56	M	TOBACCO	EPIGLOTTIS	SQUAMOUS CELL CA	FB SENSATION	25 YEARS
7	KARUNANIDHI	59	M	ALCOHOL	EPIGLOTTIS	SOUAMOUS CELL CA	LUMP IN THROAT	27 YEARS
8	VEERA SAMY	61	M	ALCOHOL/SMOKING	VENTRICLE/AE FOLDS	SQUAMOUS CELL CA	DYSPNOEA	28 YEARS
9	KALIAPERUMAL	59	M	SMOKING	AE FOLDS	SQUAMOUS CELL CA	DYSPHAGIA	29 YEARS
10	MUNIYANDI	62	M	ALCOHOL	EPIGLOTTIS	SQUAMOUS CELL CA	FB SENSATION	27 YEARS
11	DHANDAPANI	58	M	ALCOHOL	EPIGLOTTIS	SQUAMOUS CELL CA	FB SENSATION	26 YEARS
12	PALANIYANDI	52	M	SMOKING	VOCAL CORD/AE FOLDS	SOUAMOUS CELL CA	DYSPNOEA	24 YEARS
13	CHELLAMAL	67	F	TOBACCO/BETEL NUT	EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	21 YEARS
14	DURAI	68	M	ALCOHOL	EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	26 YEARS
15	RAMALINGAM	62	M	ALCOHOL	EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	27 YEARS
16	ANNAMALAI	55	M	SMOKING	VENTRICLE	SQUAMOUS CELL CA	FB SENSATION	26 YEARS
17	ULAGA NATHAN	54	M	TOBACCO	EPIGLOTTIS	SQUAMOUS CELL CA	FB SENSATION	21 YEARS
18	VENKATESAN	58	M	ALCOHOL	EPIGLOTTIS	SQUAMOUS CELL CA	FB SENSATION	29 YEARS
19	KANDASAMY	60	M	ALCOHOL	EPI GLOTTIS/AE FOLDS	SQUAMOUS CELL CA	FB SENSATION	30 YEARS
20	PALANI	55	M	SMOKING	VENTRICLE/FALSE CORD	SQUAMOUS CELL CA	FB SENSATION	23 YEARS
21	NEELAMEGAM	62	M	TOBACCO	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	LUMP IN THROAT	28 YEARS
22	MUTHUKANNU	63	M	TOBACCO	EPIGLOTTIS	SQUAMOUS CELL CA	FB SENSATION	27 YEARS
23	UTHIRAPATHY	62	M	SMOKING/ALCOHOL	VENTRICLE/FALSE CORD	SQUAMOUS CELL CA	DYSPNOEA	26 YEARS
24	RAMASAMY	60	M	ALCOHOL	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	FB SENSATION	27 YEARS
25	SELVI	69	F	TOBACCO/BETEL NUT	EPIGLOTTIS	SOUAMOUS CELL CA	LUMP IN THROAT	24 YEARS
26	SUNDARAM	71	M	SMOKING	VENTRICLE/FALSE CORD	SQUAMOUS CELL CA	DYSPNOEA	28 YEARS
27	PALANIVELU	56	M	ALCOHOL	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	LUMP IN THROAT	27 YEARS
28	VELUCHAMY	62	M	TOBACCO/ALCOHOL	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	LUMP IN THROAT	23 YEARS

S.NO	NAME	AGE	SEX	RISK FACTORS	SUB SITE	HISTOPATHOLOGY	SYMPTOMS	DURATION OF EXPOSURE
29	MANOHARAN	64	M	SMOKING	VENTRICLE/FALSE CORD	SQUAMOUS CELL CA	DYSPNOEA	24 YEARS
30	AYYACHAMY	55	M	ALCOHOL	EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	22 YEARS
31	NARAYANAN	60	M	ALCOHOL/SMOKING	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	LUMP IN THROAT	34 YEARS
32	CHINNATHAMBI	70	M	ALCOHOL/TOBACCO	EPIGLOTTIS	SQUAMOUS CELL CA	FB SENSATION	24 YEARS
33	THANGAVELU	63	M	SMOKING/ALCOHOL	VENTRICLE/FALSE CORD	SQUAMOUS CELL CA	DYSPNEA	24 YEARS
34	ELANGOVAN	68	M	ALCOHOL	EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	23 YEARS
35	GANESAN	62	M	ALCOHOL/SMOKING	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	LUMP IN THROAT	27 YEARS
36	ANJALAI	67	M	TOBACCO/BETEL NUT	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	LUMP IN THROAT	29 YEARS
37	MAHALINGAM	65	M	SMOKING	VENTRICLE/FALSE CORD	SQUAMOUS CELL CA	DYSPNOEA	32 YEARS
38	SETHURAMAN	66	M	ALCOHOL	VALLECULA/EPIGLOTTIS	SQUAMOUS CELL CA	FB SENSATION	34 YEARS
39	MATHIALAGAN	69	M	ALCOHOL/SMOKING	EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	21 YEARS
40	BAVUNAMBAL	72	F	BETEL NUT/TOBACCO	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	LUMP IN THROAT	36 YEARS
41	CHANDRAN	64	M	SMOKING	VENTRICLE/FALSE CORD	SQUAMOUS CELL CA	DYSPNOEA	26 YEARS
42	GOVINDAN	67	M	ALCOHOL/TOBACCO	EPIGLOTTIS/AE FOLDS	SOUAMOUS CELL CA	LUMP IN THROAT	28 YEARS
43	RENGASAMY	68	M	ALCOHOL/SMOKING	VENTRICLE/FALSE CORD	SQUAMOUS CELL CA	DYSPNEA	24 YEARS
44	CHELLAMUTHU	60	M	ALCOHOL/TOBACCO	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	FB SENSATION	30 YEARS
45	THANGAVELU	65	M	SMOKING	VENTRICLE/FALSE CORD	SQUAMOUS CELL CA	DYSPNEA	32 YEARS
46	THANGARASU	63	M	ALCOHOL	EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	28 YEARS
47	KARUPPIAH	61	M	ALCOHOL/TOBACCO	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	LUMP IN THROAT	27 YEARS
48	CHINNASAMY	68	M	ALCOHOL/SMOKING	VENTRICLE/FALSE CORD	SQUAMOUS CELL CA	FB SENSATION	25 YEARS
49	RATHINAM	64	M	ALCOHOL	EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	30 YEARS
50	RAMASAMY	85	M	TOBACCO/BETEL NUT	ARYTENOIDS/AE FOLDS	SQUAMOUS CELL CA	THROAT PAIN	38 YEARS
51	MAHALINGAM	58	M	ALCOHOL/TOBACCO	ARYTENOIDS/AE FOLDS	SQUAMOUS CELL CA	DYSPHAGIA	30 YEARS
52	SELVI	67	F	TOBACCO/BETEL NUT EPIGLOTTIS/PE FOLDS		SQUAMOUS CELL CA	LUMP IN THROAT	25 YEARS
53	MUTHU	62	M	BEEDI SMOKING	AE FOLDS/PE FOLDS	SQUAMOUS CELL CA	THROAT PAIN	35 YEARS
54	SEKAR	62	M	SMOKER	AE FOLDS/PE FOLDS	SQUAMOUS CELL CA	THROAT PAIN	20 YEARS
55	MURUGAIYAN	40	M	TOBACCO	EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	18 YEARS
56	JEYARAMAN	60	M	TOBACCO	EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	21 YEARS

S.NO	NAME	AGE	SEX	RISK FACTORS	SUB SITE	HISTOPATHOLOGY	SYMPTOMS	DURATION OF EXPOSURE
57	RAMACHANDRAN	65	M	ALCOHOL/SMOKING	VALLECULA/EPIGLOTTIS	SQUAMOUS CELL CA	THROAT PAIN	34 YEARS
58	SANDANAM	84	M	ALCOHOL/SMOKING	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	THROAT PAIN	35 YEARS
59	KANNAN	65	M	ALCOHOL/SMOKING	EPIGLOTTIS	SQUAMOUS CELL CA	THROAT PAIN	28 YEARS
60	KALIYAN	72	M	ALCOHOL/TOBACCO	AE FOLDS/ARYTENODS	SQUAMOUS CELL CA	DYSPHAGIA	37 YEARS
61	RAJANGAM	60	M	SMOKING	AE FOLDS/ARYTENODS	SQUAMOUS CELL CA	THROAT PAIN	35 YEARS
62	VALLIYAMMAI	67	F	TOBACCO/BETEL NUT	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	LUMP IN THROAT	36 YEARS
63	ANNADURAI	59	M	ALCOHOL/SMOKING	EPIGLOTTIS	SQUAMOUS CELL CA	THROAT PAIN	30 YEARS
64	NATARAJAN	61	M	ALCOHOLISM	AE FOLDS/ARYTENOIDS	SQUAMOUS CELL CA	DYSPNOEA	28 YEARS
65	JEGANATHAN	62	M	ALCOHOL/TOBACCO	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	THROAT PAIN	31 YEARS
66	THANGARASU	58	M	SMOKING	ARYTEOLDS/AE FODS	SQUAMOUS CELL CA	THROAT PAIN	30YEARS
67	RENGANATHAN	63	M	ALCOHOL/SMOKING	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	DYSPHAGIA	35 YEARS
68	GANESAN	64	M	ALCOHOL/TOBACCO	AE FOLDS/ARYTENODS	SQUAMOUS CELL CA	LUMP IN THROAT	36 YEARS
69	ELANGOVAN	60	M	ALCOHOL/SMOKING	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	THROAT PAIN	32 YEARS
70	MARIMUTHU	62	M	SMOKING	AE FOLDS/ARYTENOIDS	SQUAMOUS CELL CA	LUMP IN THROAT	34 YEARS
71	ULAGA NATHAN	65	M	ALCOHOL	EPIGLOTTIS	SQUAMOUS CELL CA	FB SENSATION	36 YEARS
72	AYYAPPAN	57	M	ALCOHOL/SMOKING	AE FOLDS/ARYTENOIDS	SQUAMOUS CELL CA	DYSPNOEA	30 YEARS
73	VEERAMUTHU	70	M	ALCOHOL/TOBACCO	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	THROAT PAIN	38 YEARS
74	MARIYAMMAL	64	F	TOBACCO/BETEL NUT	VALLECULA/EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	35 YEARS
75	LAKSHMANAN	68	M	ALCOHOLISM	EPIGLOTTIS	SQUAMOUS CELL CA	FB SENSATION	37 YEARS
76	KALIMUTHU	72	M	SMOKING	AE FOLDS/ARYTENOIDS	SQUAMOUS CELL CA	THROAT PAIN	39 YEARS
77	ALAGAPPAN	67	M	ALCOHOLIS/SMOKING	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	THROAT PAIN	36 YEARS
78	PALANIYAPPAN	72	M	ALCOHOL/TOBACCO	AE FOLDS/ARYTENOIDS	SQUAMOUS CELL CA	FB SENSATION	39 YEARS
79	NALLATHAMBI	69	M	ALCOHOL/TOBACCO	VALLECULA/EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	37 YEARS
80	SAMINATHAN	60	M	SMOKING	AE FOLDS/ARYTENOIDS	SOUAMOUS CELL CA	DYSPNOEA	32 YEARS
81	NALLAMUTHU	62	M	ALCOHOL/SMOKING	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	THROAT PAIN	34 YEARS
82	MARUTHAMALAI	65	M	ALCOHOL/TOBACCO	AE FOLDS/ARYTENOIDS	SQUAMOUS CELL CA	LUMP IN THROAT	31 YEARS
83	DHANDAPANI	68	M	ALCOHOL	EPIGLOTTIS	SQUAMOUS CELL CA	FB SENSATION	37 YEARS
84	MANIMEGALAI	62	F	TOBACCO/BETEL NUT	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	LUMP IN THROAT	28 YEARS

S.NO	NAME	AGE	SEX	RISK FACTORS	SUB SITE	HISTOPATHOLOGY	SYMPTOMS	DURATION OF EXPOSURE
85	KALIYAN	67	M	ALCOHOL/TOBACCO	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	FB SENSATION	36 YEARS
86	IYYAPPAN	70	M	SMOKING	AE FOLDS/FALSE CORD	SQUAMOUS CELL CA	DYSPNOEA	38 YEARS
87	PANDYYAN	69	M	ALCOHOL	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	LUMP IN THROAT	24 YEARS
88	ELAIYAPERUMAL	72	M	ALCOHOL/SMOKING	AE FOLDS/VENTRICLE	SQUAMOUS CELL CA	VOICE CHANGE	26 YEARS
89	LAKSHMI	64	F	TOBACCO/BETEL NUT	VALLECULA/EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	23 YEARS
90	SINGARAM	68	M	SMOKING	AE FOLDS/VENTRICLE	SQUAMOUS CELL CA	DYSPNOEA	30 YEARS
91	SOMU	71	M	TOBACCO/BETEL NUT	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	FB SENSATION	32 YEARS
92	VISWANATHAN	69	M	ALCOHOL	EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	30 YEARS
93	APPASAMY	70	M	ALCOHOL/SMOKING	AE FOLDS/VENTRICLE	SQUAMOUS CELL CA	DYSPNOEA	35 YEARS
94	MANIKKAM	67	M	SMOKING	VENTRICLE/VOCAL CORD	SQUAMOUS CELL CA	DYSPNOEA	37 YEARS
95	KATHIRVEL	71	M	TOBACCO/BETEL NUT	VALLECULA/EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	35 YEARS
96	VARATHAN	64	M	ALCOHOL	AE FOLDS/EPIGLOTTIS	SQUAMOUS CELL CA	FB SENSATION	35 YEARS
97	ARIYAMUTHU	68	M	SMOKING	VENTRICLE/VOCAL CORD	SQUAMOUS CELL CA	DYSPNOEA	40 YEARS
98	KAMATCHI	67	F	TOBACCO/BETEL NUT	VALLECULA/EPIGLOTTIS	SQUAMOUS CELL CA	LUMP IN THROAT	35 YEARS
99	NALLAMUTHU	70	M	ALCOHOL/SMOKING	AE FOLDS/VENTRICLE	SQUAMOUS CELL CA	DYSPNOEA	38 YEARS
100	BALAN	56	M	BEEDI SMOKING	EPIGLOTTIS/AE FOLDS	SQUAMOUS CELL CA	DYSPHAGIA	25 YEARS