DISSERTATION ON

THE ROLE OF TRANS HIATAL ESOPHAGECTOMY IN THE CARCINOMA OF ESOPHAGUS

M.S.DEGREE EXAMINATION

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CERTIFICATE

This is to certify that this dissertation entitled " **THE ROLE OF TRANS HIATAL ESOPHAGECTOMY"** is a bonafide record work done by Dr. D.R. SHANKAR, Submitted as partial fulfillment for the requirements of M.S. Degree Examinations Branch I, General Surgery, MARCH 2007.

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INTRODUCTION

Carcinoma Oesophagus is the 6th most common cancer worldwide which represents 4% of all newly diagnosed cancers. It is generally considered as an extremely aggressive tumor with poor prognosis with an overall survival rate of 5%. Despite great advances in surgery, critical care, radiotherapy, chemotherapy, esophageal cancer afflicts a large number of patients every year and almost matching that number is the expected cause of death.

They remain as a major therapeutic problem confronting the surgeon. They are likely to result in early mortality owing to the likelihood of advanced disease at the time of diagnosis and the challenging nature of treatment.

Traditionally, esophageal cancer has been squamous type in patients with usual risk factors for other aerodigestive tract carcinomas, specifically, smoking (5 fold) and alcohol (5 fold) abuse. Heavy smoking and heavy drinking combine to increase the risk 25 to 100 fold. Remarkably within North America and Europe, the incidence of adenocarcinoma rose 100% in the 1990s.

Although the origin of this shift remains unknown, carcinoma of the esophagus now appears to affect younger, healthier patients. Nutritional factors and potential carcinogens have been incriminated, and familial disease Tylosis, which is an inherited autosomal dominant trait,also predisposes to esophageal

carcinoma. Some esophageal lesions are premalignant, including Barretts esophagus, Plummer Vinson syndrome, Achalasia, etc.,

In recent years, with an improved standard of surgical technique and perioperative & postoperative care, substantial reduction of operative morbidity & mortality has been achieved.

The study of esophageal cancer is interesting because of its biologic behaviour; it infiltrates locally,involves adjacent lymph nodes, and metastasizes widely by hematogenous spread.

The prognosis form patients with invasive squamous cell carcinoma is poor; the overall 5-year survival rate for patients with treated tumors is 5 to 12%

HISTORICAL REVIEW

An esophageal growth causing swallowing difficulty was first described in Western literature by Galen in 2nd century, which was documented as a cause for dysphagia by Avicenna in 10th century. Surgery for Carcinoma esophagus began in 1877, when Czerny carried out the first successful resection of cervical anastomosis. However, not until 1913 was the first successful resection of a thoracic Carcinoma esophagus performed.

The first successful one stage resection of a thoracic Carcinoma esophagus & reconstruction, using the whole stomach was described by Obsawa in 1933. Transabdominal or Trans thoracic esophagectomy with immediate esophago gastrostomy was done in 1946. In 1978 Orringer, revived the technique of Trans hiatal esophagectomy without thoracotomy. Multi-modal therapeutic approach such as adjuvant and neo-adjuvant chemoradiation for the treatment of esophageal cancers was introduced in the year 1984.

Newer techniques & treatment strategies are being planned since then and an effective tumoricidal regimen is quite a possibility in the near future.

AIM OF THE STUDY

The objectives of this prospective clinical study are:

1) To know the incidence of Carcinoma esophagus with regard to age, socioeconomic status in our region.

2) To know the aetiological & epidemiological factors associated with these Carcinoma.

3) To study the common histological types of Carcinoma esophagus.

4) To know the clinical features & possible investigations to aid the diagnosis & resectability of these tumors.

5) To evaluate the role Trans Hiatal oesophagectomy in Carcinoma oesophagus & its outcome.

REVIEW OF LITERATURE

ANATOMY OF THE ESOPHAGUS

EMBROLOGY:

Oesophagus starts to develop in the 4th week of embryonic development, from the Foregut, immediately caudal to the primordial pharynx & extends to the fusiform dilatation in the foregut. It elongates rapidly due to growth & descent of heart & lungs reaching its final length by 7th week.

By 5th week of development, the oesophagial epithelium is columnar & 2 cells thick and cilia develop by 10th week. The epithelial lining proliferates & partly or completely obliterates the lumen by 8th week, which is replaced with stratified squamous epithelium by 4th month. The inner circular layer is recognizable by 5th week & outer longitudinal layer takes shape by 8th week. Oesophagus attains its final length at 7th week of gestation, whose length at birth is 8 – 10 cm.

TOPOGRAPHY:

Oesophagus is a hollow muscular tube of about 25cms long which connects pharynx to the stomach. It commences in the neck, level with the lower border of cricoid cartilage (C6 vertebra) and descends mainly anterior to the vertebral

column traversing the diaphragm at the level of T10, ending in the abdomen at the cardia orifice of stomach at the level of T11.

Topographically, the esophagus is generally vertical, but has two shallow curves. Immediately below the pharynx, it is a midline structure, but inclines to the left as far as the root of neck, gradually returning to the midline near T5 vertebra & deviates to the left again at T7. It also curves in a coronal plane to follow the cervical & thoracic curvatures of vertebral column. The surgical relevance of this is that the cervical esophagus is best approached from the left side of neck & thoracic portion through right side of thorax, except the lower third. It lies anterior to vertebral column & longus colli muscles, posterior to trachea & adjacent to descending aorta.

There are a number of naturally occurring anatomical narrowings in the oesophagus. The cervical constriction occurs at the level of cricopharynx at approximately 15 cm from the incisors. The next constriction is at where it is crossed by aortic arch at 22 cm & by left main bronchus at 27 cm. The another constriction is at the level where it passes through diaphragmatic hiatus at the cardio-oesophageal junction at 37 – 40 cm from the incisors.

The esophagus is arbitrarily divided into 3 segments:

The cervical portion of esophagus is approximately 5 cm long & descends between vertebral column, from level of C 6 to the level of interspace between 1^{st} and 2^{nd} thoracic vertebrae posteriorly. The recurrent laryngeal lies in the right &

left grooves between trachea and esophagus. The left recurrent laryngeal nerve lies close to esophagus than the right.

The Thoracic esophagus is 20 cm long and 2.5 cm diameter, which runs in the superior mediastinum between trachea & vertebral column, passing behind and to the right of aortic arch to descend in the posterior mediastinum along the right side of the descending thoracic aorta. It then deviates further to the left & anteriorly entering the diaphragmatic hiatus at the level of T 10. On either side it is bounded by parietal pleurae.

Clinically, it is divided into 3 parts. The **upper thoracic** esophagus extends from cricopharyngeus to the carina. The **middle thoracic** esophagus extends from carina to halfway between carina & the OG junction. The **lower thoracic** one exists from halfway between carina & OG junction to include lower 3rd esophagus.

Oncologically, it is divided into **Supra carinal** (Upper esophagus) & **Infra carinal** (middle & lower oesophagus).

Abdominal oesophagus (2cm) emerges from the right diaphragmatic crus slightly to left of midline at the level of T10 & it is surrounded by Phreno oesophageal membrane.It is covered by peritoneum on its front and left side. Behind it is the left crus of diaphragm. It includes a portion of Lower esophageal sphincter (LES), which is the portion of esophagus subjected to positive pressure environment of esophagus.

HISTOLOGICAL STRUCTURE:

The esophagus is mucosal lined muscular tube lacking the serosa. It consists of **four layers**.

1. The **fibrous adventitial layer**, which is irregular and consists of loose areolar connective tissue containing elastin fibres, which permit considerable movement during swallowing.

2. **Muscular layer**, composed of outer thicker longitudinal & inner circular layer. The longitudinal fibres originate from a cricoesophageal tendon arising from the dorsal upper edge of anteriorly located cricoid cartilage.

   The longitudinal fibres surround the whole length of esophagus with a continous coat except posterosuperiorly where the longitudinal fibres separate and sweep around to the anterior aspect of esophagus before their insertion into posterior aspect of cricoid. This configuration allows a "V" shaped area in the posterior wall covered by cricopharynx above & circular fibres below.

   The circular layer is thicker than outer longitudinal layer. The geometry of circular muscle is helical and makes the peristalsis to assume a worm like drive.

3. the **submucosal layer,** which is very loose in order to permit the dilatation of esophagus during swallowing. It loosely connects mucosal & muscular layers & contains large blood vessels, nerves, mucous glands.

4. The **mucosal layer**, made of non-keratinising squamous epithelium which is arranged in longitudinal folds especially at lower end. The mucosal layer consists of lining epithelium, connective tissue with papilli (lamina propria) and nonstriated muscularis mucosa.

VASCULATURE

ARTERIAL SUPPLY

The esophagus is nourished by numerous segmental arteries all of which contribute to the extensive capillary network.

The **cervical esophagus** receives blood from Superior Thyroid artery as well as Inferior Thyroid Artery of Thyrocervical trunk with both sides communicationg through collateral vessels.

The **Thoracic esophagus** receives blood supply from Bronchial arteries, 4 to 6 aortic oesophageal arteries, supplemented by collateral vessels from Inferior Thyroid Artery, Intercostal Arteries, Inferior phrenic arteries and left gastric artery.

The **abdominal oesophagus** receives its supply from the ascending branch of left gastric artery and Inferior phrenic artery. On entering the wall of oesophagus, the arteries assume a “T”- shaped division to form a longitudinal plexus of capillary network in the submucosa.

VENOUS DRAINAGE

Blood from capillaries of oesophagus flows into a submucosal venous plexus & then into a peri oesophageal venous plexus from which the oesohageal vein originate.

In the cervical region, the oesophageal veins empty into Inferior Thyroid Vein, in the thoracic region empty into bronchial, azygos, hemi azygos veins and in the abdominal region empty into coronary veins.

LYMPHATIC DRAINAGE

The lymphatics form extensive mucosal, submucosal, muscularis and adventitial plexuses which communicate freely. These are grouped into three main tiers:

Ist tier : It composed of lymph nodes along the side of oesophagus (para esophageal)

IInd tier: It composed of the intermediate group, which contains mainly the mediastinal lymph nodes.

IIIrd tier: It composed of deep cervical, supraclavicular, tracheobronchial and celiac nodes from above downwards.

In general, lymph drainage from the upper 2\3 rds of oesophagus proceeds in cephalic direction and in lower 1\3 rds proceed in caudal direction to subdiahragmatic and celiac nodes.

Lymph drainage of Gastro esophageal junction mainly follows the arteries supplying it.

The **Thoracic duct** forms from the continuation of Cisterna chyli at a level between T12 and L2 and to the right side of abdominal aorta. It enters the

posterior mediastinum through aortic hiatus at the level of T10 to T12. It continues cephalad on the anterior surface of vertebral column between aorta and Azygos vein behind oesophagus.

At T4 level, the duct crosses to the left, passes under aortic arch and continues along the left side of oesophagus, to ascend into the neck posterior to left subclavian artery.

In the neck, the duct lies anterior to the vertebral vessels, Thyrocervical trunk, Phrenic Nerve and it enters the venous system at the junction of left subclavian and left Internal Jugular Vein.

The rich mucosal and submucosal networks allow tumor to extend intramurally. In addition, there is shared lymphatic drainage of the trachea and esophagus, thus making en bloc esophagectomy impossible for lesions above the tracheal bifurcation.

NERVE SUPPLY

Two plexuses of nerves in oesophageal wall (Meissner's & Auerbach's plexuses) form networks of multipolar ganglion, the processes of which are in

contact with one another. Postganglionic fibres of these plexuses innervate the smooth muscle cells.

The esophagus has both sympathetic and parasympathetic innervation. The recurrent laryngeal nerve branches of the vagus provide parasympathetic innervation to the cervical esophagus as well as innervation to the upper esophageal sphincter.

Sympathetic innervation consists of fibers to the cervical esophagus from the superior and inferior cervical sympathetic ganglia, to the thoracic esophagus from the upper thoracic and splanchnic nerves, and to the intra-abdominal esophagus from the celiac ganglion.

PHYSIOLOGY

Swallowing is a complex, rapid series of events that has been divided radiologically into six phases. Three types of contractions are seen in the esophageal body. **Primary peristalsis** is progressive and is triggered by voluntary swallowing. **Secondary peristalsis** is also progressive, but it is generated by distension or irritation, not by voluntary swallowing.

Tertiaty contractions are non-progressive simultaneous contractions that may occur either after voluntary swallowing or spontaneously between swallows.

EPIDEMIOLOGY

Carcinoma esophagus has the greatest variation in geographic distribution. The incidence is western countries is low. In U.S. the yearly incidence is 6.0 \ 1,00,000 population for males & 1.6 \ 1,00,000 in females.

In the West, it is predominantly a disease of elderly males with an overall incidence of 10 – 20 per hundred thousand population per annum. The highest incidence is in France followed by Scotland where the frequency of disease has more than doubled in the past 30 years. It is 20 – 30 times more common in China, Iran, and the Transki region of South Africa than West.

Through out the world, the incidence is increasing in both sexes with increasing trend towards females. There is a declining trend in Finland.

The incidence of Squamous cell carcinoma is increasing in Blacks than in whites. Adenocarcinoma, once an unusual carcinoma is diagnosed with increasing frequency & now it accounts for more than 50% in western countries. It is more common in whites than blacks.

ETIOLOGY

DIETARY

Pickled vegetables, preserved meat, salted dry fish (rich in Nitrosamines)

Micro nutrient deficiency (Vitamin A, B12, C, E, Beta – Carotene)

Trace element deficiency (Co, Cu, Mo, Zi)

ACQUIRED

Tobacco chewing

Smoking

Alcoholism

Chronic oesophagitis

Achalasia Cardia

Barrett's Oesophagus

Corrosive strictures

Plummer Vinson Syndrome

Other Aero - digestive malignancy

HEREDITARY

Tylosis

DIETARY FACTORS

World wide, nutritional deficiencies have been implicated in the pathogenesis. Because nitrates and nitrites can be converted within the body to carcinogenic N – Nitrosamines, they can cause malignancy.

The apparent risk reduction brought about by citrus fruits may be due to inhibition of endogenous nitrosamination by Vitamin C. The high prevalence of esophageal cancer in Gassim region in Saudi Arabia, has been linked to contamination of water by impurities such as petroleum oils.

ACQUIRED FACTORS

The risks associated with tobacco use appear to increase with the number of cigarettes smoked per day, duration of smoking and tar content. Alcoholism act both as an carcinogen & a promoter.

Endoscopic surveys in Iran & China showed that chronic esophagitis is directly to esophageal cancer in 65% to 80%.

In patients with Achalasia, the prevalence of malignancy is 3 to 6%. It has been estimated that the incidence of esophageal cancer among patients with a history of caustic ingestion is 1000 fold greater.

HEREDITARY FACTORS

Tylosis, an autosomal dominant disorder, characterized by hyperkeratosis of skin of palms & soles. They are more prone to develop Squamous cell carcinomas, more common in 6^{th} to 7^{th} decade. `

PATHOLOGY

SQUAMOUS CELL CARCINOMA

Squamous cell carcinoma accounts for about 90% if esophageal carcinoma. It arises from esophageal mucosa and histologically it is characterized by invasive sheets of cells that run together and are polygonal, oval or spindle shaped with a distinct or ragged stromal epithelial interface.

They are mainly located in Thoracic oesophagus, approximately 60% of these tumors are found in the middle 3rd and about 30% in distal 3rd.

The four major gross pathologic presentations are:

1) **Fungating type.**
2) **Ulcerative variety.**
3) **Infiltrative variety.**
4) **Polypoidal growths.**

ADENOCARCINOMA

It usually originates from the Barett's oesophagus following longstanding GastroEsophageal Reflux and it is the most common type in U.S. It arises from superficial & deep glands of esophagus mainly in lower 3 rd oesophagus, especially near OG junction.

It may have one of three origins;

1) Oesophageal submucosal glands.

2) Heterotopic islands of columnar epithelium.

3) Malignant degeneration of metaplastic epithelium (Barrett's oesophagus)

Unlike the mucin secreting cells of origin, Adenocarcinoma has a reduced cytoplasmic nuclear ratio.

CLASSIFICATION OF OG JUNCTON TUMORS

TYPE I : Adenocarcinoma of distal oesophagus,arising from metaplastic epithelium, infiltrating the OG junction.

TYPE II : Carcinoma of true cardia , arising from cardia epithelium or short segments with intestinal metaplasia at OG Junction.

TYPE III: Subcardial carcinomas infiltrating the OG Junction.

The incidence of adenocarcinoma has been rising steadily. Prospective evaluation has suggested that incidence of adenocarcinoma in patients with Barrett's esophagus is 40 to 125 times that expected in the general population.

In addition to classic Barrett's esophagus, there is a concern that patients with short segment Barrett's may also be at risk. In combined data from the Cleveland clinic and the University of Arizona, it was suggested that the risk for adenocarcinoma was the same.

OTHER MALIGNANT TUMORS

ADENOID CYSTIC CARCINOMA

The prevalence of this tumor is approximately 1 in 10,000 esophageal tumors. It occurs most commonly in elderly men and its presentation and site preference are similar to those of squamous cell carcinoma. Distant metastasis dominate the clinical course and the prognosis is poor.

SARCOMAS

Sarcomas of esophagus accounts for 0.8% of all oesophageal tumors. Epidermoid carcinoma with spindle cell features is the more common variety and includes **carcinosarcoma** and **pseudosarcoma**. Both the carcinomatous and sarcomatous elements of carcinosarcoma may metastasize, whereas usually only the carcinomatous elements of pseudosarcomas metastasize.

Another type of esophageal sarcoma is true sarcoma arising from mesenchymal tissue. This group includes leiomyosarcomas, rhabdomyosarcomas and fibrosarcomas.

Kaposi's sarcoma of the esophagus is increasingly seen with the emergence of the acquired immunodeficiency syndrome.

LYMPHOMA

Despite its rarity, accurate diagnosis of primary esophageal lymphoma is important because it is potentially curable. In most patients, esophageal involvement by lymphoma is secondary. About ten cases of isolated primary esophageal lymphoma has been published. The advent of AIDS has brought about an increase in the frequency of primary esophageal lymphoma. The lesions may be seen as a mass or as an ulcer which can be indistinguishable from an infectious ulcer.

ENDOCRINE CELL TUMORS (APUD OMAS):

Apudomas are rare in the esophagus (0.8% to 2.4% of malignant esophageal neoplasm). Primary small cell carcinoma is the most common variety.

PRIMARY MALIGNANT MELANOMA

Primary malignant melanoma, an extremely rare tumor, occurs primarily in older persons of either sex, accounting for only 0.1% of primary esophageal cancer. Tumor behavior is similar to melanomas elsewhere in the body; most patients die from distant metastases and the 5 year survival rate is about 4%.

ADENOSQUAMOUS CARCINOMA

It is a rare tumor that exhibits infiltrating elements of both adenocarcinoma and squamous carcinoma; however, these elements are not intimately mixed. When mature squamous epithelium is present, it is termed as Adenoacanthoma.

Other uncommon esophageal malignancies include mucoepidermoid carcinoma, primary esophageal carcinoid tumor and metastatic esophageal tumors from the breast, lungs and malignant melanoma from else where.

MODE OF SPREAD

Carcinoma esophagus is notorious for its aggressive biological behaviour. It infiltrates locally, involves adjacent lymph nodes and metastasizes widely by hematogenous spread. Lack of an esophageal serosal layer favours local tumor extension.

Tumors of upper & middle 3rd, infiltrate the tracheobronchial tree, aorta, Left Recurrent Laryngeal nerve, whereas lower 3rd tumor may invade diaphragm, pericardium, stomach. Cervical esophagus tend to drain to deep cervical, para oesophageal, posterior mediastinal nodes.

CLINICAL FEATURES

In more than 85% of patients, the presenting symptom is **dysphagia** which, initially is for solids and later progresses to liquids as the obstruction becomes complete.

The second most common presenting complaint is recent **weight loss** in about 40% of patients, owing to the combined catabolic & obstructive effects of tumor.

Chest pain is frequently reported as it arises from oesophageal spasms above a partially obstructing tumor, from irritation of esophagus by malignant ulceration or from direct invasion of mediastinal structures including spinal column.

Extension of tumor into tracheobronchial tree can cause **stridor** & if a tracheoEsophageal fistula develops, **coughing, choking,** and **Aspiration pneumonia** results. Rarely **severe bleeding** from erosion into the aorta or pulmonary vessels occur.

Vocal cord paralysis is caused by invasion of left Recurrent laryngeal nerve by primary tumor or by lymphatic secondaries. Systemic organ secondaries manifests as **jaundice** or **bone pain. Haemetemesis, regurgitation, Anorexia** is the other symptoms that can suggest the disease.

STAGING

Staging of the tumor is the critical step in determining which therapeutic option is appropriate. The stage of a tumor is classified most frequently by the staging

system devised by the American Joint Committee on Cancer. This system is a TNM based system. The 'T' (tumor) indicates the progressive degree (1 to 4) of invasion of the tumor into the esophageal wall. 'N' stands for nodal involvement and 'M' for distant metastasis.

Prognosis and outcomes are determined by stage. Five-year survivals for esophageal cancer are as follows: Stage I, 50 to 55%; Stage II, 15 to 30%; Stage III, 6 to 17%; and Stage IV less than 5%.

LYMPH NODES (N STAGE)

Lymph node involvement may be assessed by Endoscopic Ultrasound, CT, Positron Emission Tomography (PET), or Video Assisted Thoracoscopy and Laparoscopy.

T – PRIMARY TUMOR

TO No evidence of a primary tumor

Tis Carcinoma in situ (High grade dysplasia)

T1 Tumor invading the lamina propria, muscularis mucosae, or submucosa but not breaching the boundary between submucosa and muscularis propria.

T2 Tumor invading muscularis propria but not breaching the boundary between muscularis propria and periesophageal tissue.

T3 Tumor invading periesophageal tissue but not adjacent structures.

T4 Tumor invading adjacent structures.

N – REGIONAL LYMPH NODES

N0 No regional lymph node metastasis

N1 Regional lymph node metastasis

M – DISTANT METASTASIS

M0 No distant metastasis.

M1 Presence of distant metastasis

STAGE GROUPING OF ESOPHAGEAL CANCER

Stage 0		T0 N0 M0
		Tis N0 M0
Stage I		T1 N0 M0
Stage II	**II A**	T2 N0 M0
		T3 N0 M0
	II B	T1 N1 M0
		T2 N1 M0
Stage III		T3 N1 M0
		T4 Any N M0
Stage IV		Any T Any N M1

DISTANT METASTASIS (M STAGE)

Endoscopic Ultrasound is especially suited to visualize lymph nodes around the celiac axis and the left liver lobe (both considered distant metastases). CT is specific for liver, lung, and pleural metastases larger than 2cm in diameter, but evaluation with Fine Needle Aspiration or transbronchial biopsy is necessary for the determination of malignancy. Bronchoscopy is required for patients with tumors of the upper &middle third of the esophagus to view the pharynx, larynx and tracheobronchial tree for synchronous and metachronous malignancies.

CURRENT STAGING CLASSIFICATIONS

Based upon survival analysis indicating tumor penetration and lymph node metastases as the major prognostic factors, the WNM (wall penetration, node and distant metastases) system for staging was developed by skinner et al. Tumors limited to above the muscularis mucosa would be equivalent to W0 designation, T1 and T2 tumors would equate to W1 classification, and T3 and T4 tumors to the W2 classification. Ellis et al, compared the 1988 staging criteria with their modified Skinner WNM staging system and showed evidence that a modified staging system was more useful from a prognostic standpoint.

Stage 0	W0 N0 M0
Stage I	W0 N1 M0
	W2 N0 M0
Stage II	W1 N1 M0
	W2 N0 M0
Stage III	W2 N1 M0

W1 N2 M0

W0 N2 M0

Stage IV Any W Any N M1

INVESTIGATIONS

1. X-RAY CHEST

It is abnormal in only 50% of patients with findings such as an air fluid level in obstructed esophagus, a dilated esophagus, abnormal mediastinal soft tissue representing adenopathy or tracheal deviation. The evidence of lung secondaries and pleural effusion can be noted.

2. BARIUM SWALLOW

A double-contrast, full column barium swallow is very useful; the sensitivity in detecting an esophageal carcinoma is between 74% and 97%. Reports indicate that it is less efficient in screening for patients with Adenocarcinoma developing from Barrett's esophagus.

Fluoroscopically guided films taken at different angles are required in order to detect early lesions. Usually, anteroposterior, lateral, left and right anterior oblique projections are necessary. It can determine the location & length of tumor, and may reveal the presence of submucosal secondaries. The relationship of the tumor to the whole thoracic cavity and the tracheal bifurcation is useful in deciding the operative approach. Deformity of the

esophageal axis, such as tortuosity, angulation, deviation are signs indicating an advanced tumor with fixation and retraction from infiltration of the adjacent organs.

ESOPHAGOSCOPY

It is usually performed on every patient who is being evaluated for the presence of an esophageal carcinoma. The typical tumor is friable and exophytic,causing obstruction, or ulcerated with irregular raised borders. More subtle abnormalities include loss of esophageal wall motility caused by longitudinal submucosal infilltrlation. Close inspection for second synchronous primary tumors should be performed, particularly in patients with Squamous cell carcinoma.

Measurement of the distance of the abnormalities from the incisors is valuable in planning therapy. Important reference landmarks include cricopharyngeus, the aortic arch, the left mainstem bronchus, and the diaphragmatic hiatus. When the endoscope can be passed through the tumor, measurement of the overall length of involvement is also useful.

Histologic documentation of carcinoma is obtained routinely with cup forceps biopsies. Even when performed carefully, however, such biopsies are nondiagnostic in more than 7% of cases, in which flexible fiberoptic endoscope is used. The use of esophageal brush cytology can lead to a

diagnosis of malignancy in some cases in which all biopsies are negative and serves as a valuable adjunct to the endoscopic techniques.

3. ENDOSCOPIC ULTRASONOGRAM

The anatomy of esophageal wall and the surrounding lymph nodes is assessed using this EUS. Five distinct wall layers are identified in the normal esophagus, corresponding to the mucosa, submucosa, lamina propria, muscularis propria, and adventitia. Esophageal cancer appears as an irregularly delineated hypoechoic mass on EUS. The accuracy is greatest in patients with transmural tumors, particularly those involving adjacent structures. The overall accuracy is 70% to 85%.

It is very much useful in detecting mediastinal lymph nodes for metastatic involvement. Lymph nodes as small as 3 to 5 mm in diameter can be recognized. However, qualitative criteria are more important in recognizing involved nodes, which are better circumscribed and have a more irregular hypoechoic internal pattern than that found in normal lymph nodes. EUS guided FNAC can assess Lymph nodes in perioesophageal, periaortic, subcarinal, Coeliac axis regions.

4. BRONCHOSCOPY

Bronchoscopic examination is mandatory in patients with esophageal carcinoma involving regions adjacent to the trachea or mainstem bronchi.

The airway problems are frequently detected, indicating possible transmural spread of tumor. A large tumor mass can cause anterior displacement of the membranous portion of the trachea, although this does not necessarily imply invasion of the tracheobronchial tree.

Early findings that do indicate airway invasion include edema and elevation of the mucosa with contact bleeding. Blunting of the carina is normally caused by metastatic involvement of the carina by the primary tumor mass. Cytologic sampling of the subcarinal lymph nodes is possible by means of transcarinal Needle Aspiration.

5. CT SCAN OF THORAX & ABDOMEN

CT is currently the gold standard radiologic tool for evaluating esophageal carcinoma. The cross sectional imaging technique of CT makes it suitable for evaluating the esophageal wall & perioesophageal structures. It also provides useful information about lymph node enlargement and concomitantly evaluates the liver, lungs, and adrenal glands for metastatic spread.

It allows one to evaluate the esophageal wall thickness and tumor length. Maximum normal esophageal wall thickness is 5 mm, and asymmetric thickening is found in more than two thirds of patients with esophageal cancer. Invasion into

mediastinum, adjacent structures can be found out. One criterion of invasion is loss of fat plane between the tumor & an adjacent structure. Tracheobronchial invasion is predicted with an overall accuracy of more than 85%, the aortic wall invasion is predicted with an accuracy of 80%.

Mediastinal lymph nodes are considered abnormally large, when they are greater than 1cm in maximum diameter. CT is particularly good for detecting liver, adrenal, pulmonary and distant nodal metastases.

6. MAGNETIC RESONANCE IMAGING

Measurement of esophageal wall thickness in the absence of intraluminal air or other contrast agents is difficult. Visualization of the middle third oesophagus is complicated by artifacts resulting respiratory & cardiac motion. It can detect T4 disease and secondaries. At the same time, it tends to overstage Lymph node involvement and T disease.

7. POSITRON EMISSION TOMOGRAPHY (PET)

It is a promising investigational approach, which can evaluate the areas for increased focal uptake after injection of 18 F- fluorodeoxyglucose. However, PET facilitates selection of patients for operation by detecting distant metastases that is not identified by routine conventional radiologic techniques.

The sensitivity in detecting secondaries is 80 to 85% and it's specificity is 92 to 95%. Its disadvantages are the nonspecificity of areas of increased cellular FDG uptake, inability to determine the 'T' stage.

8. SCINTIGRAPHIC TEST FOR METASTASES

There is disagreement about the need for scintigraphic evaluationof metastases in patients with esophageal cancer. Bone scans using 99m Tc-labelled methylene diphosphonate commonly are used in staging patients with esophageal cancer and can be used to detect bone secondaries in asymptomatic patients in the absence of elevated serum calcium or alkaline phosphatase levels. However, the false positivity is found in almost 30% patients.

9. MINIMALLY INVASIVE TECHNIQUES

Laparoscopy with or without laparoscopic ultlrasonography has been routinely in some centres. More recently, thoracoscopic staging has been added to the staging armamentarium. Laparoscopic staging permits detection of metastatic disease precluding resection in 10 to 20% of patients and is more accurate than EUS in abdominal staging. Thoracoscopic staging is potentially valuable in permitting accurate stage assessment prior to any therapy. Laparoscopy, particularly as an initial step before formal resection, is likely to become a widely accepted technique for esophageal cancer.

10. OTHER STUDIES

Mediastinoscopy is used by some as a staging procedure for carcinoma of upper and middle thoracic esophagus to assess the mediastinal nodal status.

Percutaneous needle biopsy of suspected extranodal metastases is highly accurate and may eliminate the need for open biosy procedure.

In some cases, "mini-laparotomy" is of value in documenting metastatic spread to subdiaphragmatic sites when suspicious findings are noted on CT scan. Kraska & colleagues reported the sensitivity of 80% & specificity of 100% and the accuracy of 93% in detecting Thoracic lymph node by Video-Assisted Thoracoscopy (VATS)

TREATMENT

Carcinoma esophagus still remains as a lethal disease with poor 5year survival rate. The treatment of patients depends upon the stage of the disease & the general condition of the patient. Most of the cases are at a advanced stage,when it becomes symptomatic,precluding curative surgery.

Some patients with resectable lesions are unfit for surgery by virtue of significant comorbid disease. The choice of treatment is dependent on its morbidity & mortality risk, quality of palliation, risk of recurrence,the availability and the experience of particular discipline at the institution where the treatment is offered.

The therapeutic options include surgery, Radiotherapy, chemotherapy, Stenting, Palliative surgeries, or a combination of these techniques. Current trials have focused on radiation and chemotherapy with or without resection.

OPERATIVE TREATMENT

RESECTION

Cervical esophagus

- Pharyngolaryngoesophagectomy
- Free jejunal transfer

Superior Mediastinal

- Split sternum esophagectomy
- Three-phase esophagectomy

Middle and Lower third

- Lewis-Tanner operation

- Transhiatal esophagectomy
- Three-phase esophagectomy
- Esophagectomy (left thoracotomy approach)

Cardia

- Transhiatal esophagectomy
- Esophagogastrectomy (left thoracoabdominal approach)
- Esophagogastrectomy (abdominal right chest approach)
- Abdominal gastrectomy

BYPASS

- Kirschner gastric bypass
- Colon bypass
- Jejunum bypass

CURATIVE TREATMENT

Factors such as general disability, malnutrition, cardiac risk, multisystem dysfunction, liver failure, infection, invasion of a vital structure etc., limit the patient's health and chances of tolerating a curative surgical procedure. At best, only 50% of patients are eligible for a curative resection at the time of presentation.

Since the lymphatic drainage of esophagus is extensive, both within esophageal wall and in the surrounding mediastinal tissues, the longitudinal extension of esophageal carcinoma is extensive and of multicentric in origin.

Orringer, had proposed four goals of oesophagectomy;

1. To relieve dysphagia.
2. To achieve an operative mortality of less than 10%.
3. To require hospitalization of less than 14 days.
4. To minimize the late complications and morbidity.

If an esophagectomy is indicated, three major technical approaches are available.

1. **Transthoracic esophagectomy.**
2. **Transhiatal esophagectomy.**
3. **En-bloc Radical esophagectomy.**

Although no consensus has been formed on the preferred technique, Transthoracic esophagectomy is preferred by most thoracic surgeons. Since 1970, the reported 5-year survival rates for patients undergoing esophagectomy have risen from an average of 10 to 15% to a high 35% secondary to refinements in surgical

techniques, improved anaesthesia and critical care management, and an emphasis on nutrition by enteral and parenteral routes.

Despite these improvements in surgical outcome, the overall survival rate for carcinoma of esophagus has changed little, ***Katlic and colleagues*** noted only an 11% 5-year survival rate in patients with locally advanced N1 disease (Stages IIB or III) who were treated in 1990s.

Regardless of technique, Surgeons, generally agree on the desirability of a so-called R0 resection (i.e., a complete macroscopic and microscopic removal of tumor as the basic requirement in surgery with curative intent for carcinoma of esophagus and Gastroesophageal junction).Great controversy remains on the extent of the resection and the type of surgical access (i.e., Transthoracic, left or right sided, or Transhiatal resection). Reports from Japanese groups focus on the value of extended lymphadenectomy both in the mediastinum and in the superior abdominal compartment (two-field lymphadenectomy).

Many surgeons think that adding bilateral cervical lymphadenectomy (three-field lymphadenectomy) is essential, especially in patients with supracarinal tumors. As expected, these extensive resections and reconstructions may cause surgical morbidity and mortality. However, even in patients with advanced Stage III disease, 5-year survival rates of around 20% can be obtained after an R0 resection.

TRANSTHORACIC ESOPHAGECTOMY

1. The traditional surgical approach to distal esophageal carcinoma has been a left-sided thoraco-abdominal incision.
2. The distal esophagus, proximal stomach and adjacent lymph node bearing tissues are resected, and an intrathoracic esophagogastric anastamosis is performed.
3. A gastric drainage procedure (Pyloromyotomy or Pyloroplasty) is recommended to prevent subsequent PostVagotomy Gastric Outlet Obstruction from Pylorospasm.

DISADVANTAGES

1. Combined thoracic and abdominal operation in a debilitated patient may lead to respiratory insufficiency, resulting from postoperative incisional pain and an inability to breath deeply, that requires prolonged mechanical ventilatory assistance and often causes death.
2. Disruption of an intrathoracic esophageal anastamosis results in mediastinitis and sepsis, fatal in 50% of the patients.
3. Intrathoracic esophageal anastamosis is inadequate in providing long-term relief of dysphagia either because of anastamotic suture-line tumor recurrence or because of the development of reflux esophagitis above the anastamosis.

4. Intrathoracic esophagogastric anastamoses are almost invariably associated with the development of reflux oesophagitis, which follow disruption of the LES mechanism.
5. The operative mortality varies significantly, ranging from as high as 14% to as low as 2.2%.

EN BLOC ESOPHAGECTOMY

Because many patients present with metastases to regional lymph nodes as well as to the surrounding tissue and organs, a more radical resection, the en bloc esophagectomy, has been advocated by a few thoracic surgeons. An envelope of normal tissue is removed along with the spleen,celiac nodes, posterior mediastinum, azygos vein, thoracic duct, and adjacent diaphragm.

With this aggressive surgery, operative mortality ranges from 5.1 to 11%, not significantly different from other approaches. The two major complications are similar to Transhiatal and Transthoracic esophagectomies; anastomotic leak and respiratory complications. With the en bloc technique, 5-year survival rate is 40 to 55% for patients with Stage I adenocarcinoma confined to the esophageal wall.

TRANSHIATAL ESOPHAGECTOMY

In performing a transhiatal esophagectomy, the surgeon removes accessible cervical, intrathoracic, and intra-abdominal lymph nodes for staging, but a complete en bloc resection of adjacent lymphnode bearing tissue is not accomplished.

It is performed through an **upper midline abdominal and cervical incision** without thoracotomy, therefore, the thoracic esophagus is resected through the widened diaphragmatic hiatus and the neck. The stomach is mobilized by dividing the left gastric and left gastroepiploic vessels and the right gastric and the right gastroepiploic arcades are preserved. **Pylomyotomy and feeding jejunostomy** are performed routinely.

The entire thoracic oesophagus **from the level of the clavicles to the cardia** is resected, while one carefully monitors intra-arterial blood pressure to avoid prolonged hypotension resulting from cardiac displacement. The surgical stapler is used to fashion a gastric tube from the greater curvature of the stomach, while preserving the entire length. The stomach is mobilized through the posterior mediastinum in the original esophageal bed and is anastamosed to the cervical oesophagus.

For **distal-third esophageal tumors** localized to the cardia, the high lesser curvature of the stomach is resected 4 to 6cm beyond the gross tumor, while preserving the point on the high greater curvature that reaches cephalad for the cervical esophagogastric anastamosis. Critics of THE object to the limited exposure afforded by the hiatus to the intrathoracic oesophagus. The limited exposure potentially increases the risk of uncontrollable **haemorrhage**, however,

the peroperative blood loss is significantly **lower than that of Tranthoracic oesophagectomy.**

Contraindications to this procedure includes evidence of tumor invasion of the pericardium, aorta, tracheobronchial tree.

THORACOSCOPIC ESOPHAGECTOMY

Several authors have reported the use of **Video-Assisted Thoracoscopy** or **Laparoscopy** in performing esophagectomy. Techniques described include a standard laparotomy with Thoracoscopic mobilization of the esophagus used to complete the operation, a totally laparoscopic Transhiatal technique, laparoscopic gastric mobilization with a right mini-thoracotomy, and the combined laparoscopic and thoracoscopic technique with thoracoscopic mobilization of the esophagus, followed by laparoscopic gastric mobilization.

Advantages of Thoracoscopic esophageal mobilization over Thoracotomy or laparotomy with Transhiatal dissection have not been clearly demonstrated in these studies.

RECONSTRUCTION AFTER ESOPHAGECTOMY

After portion of the oesophagus is removed, or after complete esophagectomy, a conduit must be established for alimentary tract continuity. The **stomach, colon, and jejunum** have all been successfully used as esophageal

substitutes, but the stomach appears to be the conduit of choice because of it's ease in mobilization and ample vascular supply.

A higher incidence of mortality is noted with the use of colon because of the necessity for three anastomoses (**coloesophagostosmy, colojejunostomy, and colocolostomy)**. Jejunal loops can also be used, but their limited vascular supply restrict mobility & reach.

RADIATION THERAPY

Patients who undergo External-beam radiataion therapy, used alone in the treatment of esophageal carcinoma, have only a 5 to 10% 5-yeat survival, so this therapy is not considered curative. Radiation therapy has low morbidity and can relieve esophageal obstruction in most patients in 4 to 7 days. The relief of dysphagia is short lived, and recurrence is seen usually within 6 months. The goal of radiation therapy is to destroy the tumor, its microscopic extensions & other local sites of metastases without crossing the radiation threshold of normal adjacent cell.

The target includes a 5 cm margin on either side of the tumor and adjacent lymph node stations. The supraclavicular or celiac lymph nodes are targets if the tumor is in the upper or lower oesophagus, respectively. In the chest, the critical structures are the lung, spinal cord, bone marrow. Typically, custom-moulded casts or cradles are used to achieve immobilization and reproducibility.

Treatment can be given by hyperfractionation, accelerated fractionation, or conventionally. The range is from 5000 cGy in 20 treatments over 4 weeks to 6600 cGy in 33 treatments over 7 weeks. Some of the complications seen are pneumonitis, pericarditis, myocarditis, stricture, fistula formation and spinal cord damage. Radiation therpy is contraindicated in the presence of a TEF. Radiation necrosis of tumor promotes fistula formation when the tumor has penetrated the trachea or bronchus.

CHEMOTHERAPY

Chemotherapy as a single modality in the treatment of esophageal cancer is the least effective strategy. Although radiographic improvement can be seen in up to one-half of patients, two or three cycles (6 to 12 weeks) of chemotherapy are required , relief of dysphagia is slow and/or incomplete, and survival is anecdotal. Unfortunately, no reliable method exists to identify ”responders” before therapy is begun. Chemotherapy is used pre-operatively alone or in combination with radiation therapy to treat micrometastases and to reduced the size of the tumor to improve resectability rate.

Moreover, if surgery is not appropriate, chemotherapy is used with radiation therapy for palliation and possibly to improve survival. It is typically given in a combination of two or more drugs. **Cisplatin and 5-Fluorouracil** are the most frequently used agents. Other agents with activity in esophageal cancer include **Paclitaxel, Camptothecin, Irinitecan and Vinorelbine**. Combination therapy

has been promising, with response rates between 50 and 70% for Cisplatin based doublets. Adding a third agent, such as a **Vinca alkaloid , Bleomycin**, or **mitoguazone** has only fractionally improved response while almost universally worsening toxicity.

Most studies dealing with **neoadjuvant** therapy are based on combinations that contain Cisplatin, which seems to be well tolerated without increasing the post-operative mortality or morbidity rates. Response rates vary between 25 and 50%. ***Meluch and colleagues*** treated 49 patients with localized esophageal cancer with Paclitaxel, Carboplatin, and continuous low-dose Fluorouracil given with concurrent Radiotherapy. Forty six percent of the operated patients achieved a pathologic complete response, and an additional 30% were found to have only microscopic residual disease in the resected specimen.

PALLIATIVE TREATMENT

Palliation is appropriate when patients are too debilitated to undergo surgery or have a tumor that is unresectable because of extensive invasion of vital structures, recurrence of resected or irradiated tumor, and\or metastases. The goal of palliation is to use the most effective tumor and least invasive means possible to relieve dysphagia and discomfort, to support nutrition, and to limit hospitalization.

Depending on the perceived life expectancy, palliation includes **Dilatation, intubation, Photodynamic therapy, Radiotherapy with or without**

chemotherapy, surgery, and\or laser therapy. None of these methods have proven superior.

DILATATION

It is to palliate dysphagia and to allow endoscopic evaluation with a 2 to 3% risk of esophageal wall rupture or bleeding. Unfortunately, relief is measured only in weeks. Patients with high-grade malignant strictures more likely present with advanced disease.

STENTING

The purpose of a stent is to bridge the obstruction in the esophagus to allow luminal patency primarily to prevent pooling of saliva and secondarily for nutrition. Flexible, self-expanding stents are constructed of two layers of super alloy monofilament wire with a layer of silicon between them. The silicon sandwitched between the layers delays tumor in-growth through the holes in the wire mesh.

After administration of local or general anaesthesia, the stricture is dilated to 42 to 45 French, the lesion is identified, and the expandable covered stent is inserted under fluoroscopic or endoscopic control. Once the stent is inserted
and expanded, the ends flange out to anchor to the wall of the esophagus.

PHOTODYNAMIC THERAPY

For photodynamic therapy, a photosensitizer such as dihematoporphyrin ether, is given intravenously and after 2 to 3 days is retained in the tumor in a much higher concentration than in healthy tissue. Then, a low power Laser system that produces red light is delivered to the tumor by a flexible endoscope.

Two to three days after this therapy, esophagoscopy is repeated and the necrotic tissue is removed, often monthly. Complications include development of fistulas and aspiration. Edema of the hands & face, sensitivity to light are other complications.

MATERIALS AND METHODS

This is a prospective study of **160 patients** (out of **2260** cancer patients), of **Carcinoma Oesophagus,** who were admitted in the **Thanjavur Medical College Hospital,** from **June 2004 to September 2006.**

METHODS

The methods include, obtaining the important information from the patients through History, thorough clinical examination and doing the investigations, whatever is necessary to aid the diagnosis and resectability.

All the informations were entered in a Proforma, specially designed for this study.

METHODOLOGY

The following factors were taken into consideration while evaluating the patients.

- Age and sex incidence.
- Geographical factors.
- Socio-economic status.
- Personal habits.
- Symptoms and duration.
- Predisposing factors.

In all these patients, nourishment was noted. **Abdomen** was examined for any mass, hepatomegaly and ascites. **Rectal Examination** was done to find out Bloomer's shelf or deposit in the Pouch of Douglas. **Respiratory system** was examined to find out pleural effusion and signs of Aspiration pneumonia.

They were all subjected to **basic investigations** which included,

1. Urine Albumin\Sugar
2. Blood Hemoglobin.
3. Blood Sugar.
4. Blood Urea.
5. Serum Creatinine.
6. X-Ray chest.

Specific Investigations such as

1. Upper GI Endoscopy and Biopsy.
2. Barium Swallow.
3. Ultrasound Abdomen.
4. CT Thorax and Abdomen.

Upper GI Endoscopy was done in all the patients and the biopsy was taken from the growth. The presence, location, type of growth and it's distance from Incisors were studied.

All patients who had a positive endoscopy and biopsy were submitted for **Ultrasound** of the abdomen to rule out any metastases.

CT Thorax was done to study the location of tumor, esophageal wall thickness and the tumor length. The presence of mediastinal lymphadenopathy and the direct invasion of adjacent vital structures like Trachea, Aorta, heart etc., are noted and hence the resectability was noted.

CT Abdomen revealed the presence the hepatic, Adrenal secondaries.

Bronchoscopic examination was done in all the patients to rule out tracheobronchial involvement by the primary tumor.

After obtaining the essential informations from these studies, the surgical management in the form of **Transhiatal esophagectomy** was planned in selected 50 patients, excluding the remaining patients . In our Institution, Transthoracic esophagectomy is not practiced.

EXCLUSION CRITERIA

1. patients with carcinoma involving the cervical \ Upper 3rd esophagus.
2. patients with extensive disease.
3. patients with distant metastasis.
4. patients with infiltration of perioesophageal vital structures, precluding respectability.
5. patients with co-morbid conditions like Anaemia, CCF, CAHD etc.,
6. patients with poor general condition.
7. patients not willing for surgery.

The selected 50 patients were pre-operatively evaluated thoroughly and their general condition was improved. Out of 50 patients, per-operatively, we found that, in about 25 patients the tumor was found to be unresectable due to several causes like, extensive disease, invasion of adjacent vital structures, multiple peritoneal seedlings, small hepatic secondaries etc. Hence these (25) patients were submitted to Palliative Radiotherapy, Feeding Jejunostomy, Stenting.

With the facilities available, we adopted Transhiatal oesophagectomy in the remaining 25 patients as the curative surgical procedure with or without postoperative chemo\raditherapy.

Post operative complications were identified promptly and managed accordingly. Most of the patients who were operated were reviewed in our OP Department. Every attempt was made to evaluate the general condition, any evidence of recurrence, distant metastasis, while following up the patients.

OBSERVATIONS AND RESULTS

The incidence of carcinoma esophagus, by this study in our institution is 7.0% (160 \ 2260)

AGE INCIDENCE

AGE GROUP (YEARS)	NUMBER OF CASES	PERCENTAGE
< 30	00	0%
31 – 40	19	12%
41 – 50	51	32%
51 – 60	77	48%
61 – 70	13	08%
> 70	00	00%

Regarding the age of the patients, the **youngest** patient with carcinoma of esophagus in our institution was about 30 years, a female and the **oldest** was a 69 year old male.

Most of the patients affected were in the age group between 51 to 60 (48%) which correlates well with the ***Orringer et al.,*** studies.

SEX INCIDENCE

AGE GROUP (YEARS)	**MALES**		**FEMALES**	
	NUMBERS	**%**	**NUMBERS**	**%**
<30	00	0%	00	0%
31-40	09	9%	07	11%
41-50	30	32%	21	33%
51-60	48	50%	29	44%
61-70	09	9%	07	11%
>70	00	0%	00	0%
Total	96	100%	64	100%

In our study, **males** were more commonly affected, about 96 patients. The **male : Female** ratio is 1.5 :1.

It is well known that carcinoma of esophagus afflicts more number of patients in the **low socioeconomic** status. Regarding this, close to 100% of the patients were of the lowest socioeconomic status. This is mostly due to the fact that people seeking medical advice at Government based institutions are usually of the lower income group.

While evaluating the geographical distribution of the carcinoma of the esophagus, we found that as far as the cases referred to the TMCH, were concerned the maximum number of patients from the place **Trichy** (29). It accounts for about **18.12%** of the patients.

The relation of carcinoma of esophagus and personal habits has been well established in various studies.

HABITS		**FEMALES**	
		NUMBERS	**%**
CHEWING	**TOBACCO**	70	44%
	BETEL NUT	64	40%
	BOTH	80	50%
SMOKING		70	44%
ALCOHOL		62	39%

BOTH		86	54%
HOT SPICY FOOD		88	58%

The majority of patients had the habit of consuming **hot spicy foods**, corresponding to about 58%. About 54% of patients were both alcoholic and smokers. About 44% of patients were **smokers** and about 39% were **alcoholics.** . The alcoholism and smoking are considered as the important carcinogens.

About 44% patients were **tobacco** chewers and 40% had the habit of **Betel nut** chewing. These habits were found to be common in females.

FACTORS	**NUMBER OF CASES**	**PERCENTAGE**
STRICTURES	4	2%
BARRETTS	7	4%
IRRADIATION	0	0%
PV SYNDROME	4	2%
TYLOSIS	4	2%
PREVIOUS GASTRIC SURGERY	0	0%

Considering the other predisposing factors, very negligible numbers only could be documented. Only four patients had stricture, seven patients were previously

diagnosed to have **Barretts esophagus.** The **Plummer Vinson Syndrome** and **Tylosis** were present in four patients in each. None had a previous history of irradiation of any sort or a past gastric surgery.

The distribution according to the presenting complaints are given by the following symptomatology;

COMPLAINTS	NUMBER OF CASES	PERCENTAGE
DYSPHAGIA	140	88%
WEIGHT LOSS	115	72%
ANOREXIA	108	68%
VOMITING	51	32%
CHEST PAIN	45	28%
ODYNOPHAGIA	42	26%
COUGH	32	20%
ANOREXIA	108	68%
HOARSENESS	0	0%
DYSPNOEA	0	0%

Among the presenting complaints, the **commonest** and in many a times, the only complaint was **dysphagia** (88%). The second most complaint was the recent **weight loss**, which was present in about 72% of patients. About 68%, had

loss of appetite. Rarely, patients had the hematemesis (16%). No patient had hoarseness of voice or dyspnoea.

Distribution of cases according to the site of the tumor;

LOCATION	NUMBER OF CASES	PERCENTAGE
UPPER 1\3	30	19%
MIDDLE 1\3	48	30 %
LOWER 1\3	82	51%

There was a considerable variation in the statistics as far as the location of the tumor is considered. The commonest site of the carcinoma of esophagus was the lower 3 rd in our institution, about 82 which accounts for 51% and the middle 3rd growths corresponded to about 30% in 48 patients. About 19% had upper 3rd carcinomas, which were absolutely excluded from our study.

Various studies have documented a major shift in the histological pattern of the cancer esophagus, with a double fold rise from a traditional squamous cell carcinoma to adenocarcinoma over the last few decades. But, observations made in our study results do not correlate well with the changing trend as far as the

histological pattern is concerned. The commonest histological pattern is still Squamous cell carcinoma in our institution.

OGD SCOPY FINDINGS

TYPEOF GROWTH	NUMBER OF CASES	PERCENTAGE
FUNGATING	0	0%
ULCERATIVE	83	52%
INFILTRATIVE	64	40%
POLYPOID	13	08%

Most of the patients were found to have ulcerative growths. Infiltrating type of growths were the second common type of growth. Rarely, about 8% had polypoid growth.

HISTOLOGICAL VARIETIES

TYPE	NUMBER OF CASES	PERCENTAGE
SQUAMOUS CELL	115	72%
ADENOCARCINOMA	39	24%
NOT SPECIFIED	06	04%

Some lesions when biopsied were reported to as a specific type. some were given as lesions **highly suspicious of malignancy.** These have been included under the group as not specified.

Of 160 patients, the 50 patients were selected by adopting the exclusion criterias.

FACTORS	NUMBER OF CASES	%
UPPER 1/3 rd GROWTH	30	18.75%
EXTENSIVE DISEASE	48	30.00%
DISTANT METASTASES	11	06.87%
CO-MORBID ILLNESS	08	05.00%
POOR GENERAL CONDITION	10	06.25%
NOT WILLING FOR SURGERY	03	01.87%

Since the aim of this study is to highlight the role of Transhiatal esophagectomy (THE), the carcinomas involving the cervical esophagus were primarily excluded. About 48% of patients had advanced disease at the time of their initial presentation, which may be explained by the biological behaviour of the disease. Distant metastases were found in 11 patients (06.87%).

About 8 patients (5%) had **comorbid illnesses** like Ischemic Heart disease (3patients), Atrial fibrillation (2 patients), Complete heart Block (2 patients), COPD (1 patient) and hence they were excluded. Nearly 10 patients had very **poor general condition**, which made them unfit for surgery. After explaining the

risks of surgery, 3 patients (1.87%) were not willing for surgery. All the excluded patients were referred to the Radiotherapy Unit for further management.

Out of 50 selected patients, only 25 patients were found to have resectable growths. The following were the factors which precluded from doing THE preoperatively.

FACTOR	NUMBEROF CASES	PERCENTAGE
EXTENSIVE DISEASE	08	16%
PERIOESOPHAGEAL INVASION	07	14%
HEPATIC SECONDARIES	06	12%
PERITONEAL SEEDLINGS	04	08%

Inspite of thorough preoperative evaluation with Barium Swallow,

OGD Endoscopy, CT Thorax and USG Abdomen, we found extensive disease in about 16 patients (16%), invasion \ infiltration into trachea , Azygos vein in 7 patients (14%), small hepatic secondaries in 6 patients (12%) and multiple

Omental and peritoneal seedlings in about 4 patients (8.0%).

These patients were further managed as following;

TREATMENT	NUMBER OF CASES	PERCENTAGE
T H E	25	50%
RADIOTHERAPY	15	30%
STENTING	04	08%
JEJUNOSTOMY	06	12%

The preoperatively found unresectable patients were managed with palliative Radiotherapy (15 patients), Feeding Jejunostomy (6 patients),

Referred to higher centres for Palliative Stenting (4 patients).

Complications of the surgical procedure are usually less serious and could be managed conservatively and none required a repeat surgery.

COMPLICATIONS	NUMBER	PERCENTAGE
WOUND INFECTION	03	12%
ANASTOMOTIC LEAK	04	16%
STRICTURE	03	12%
WOUND DEHISCENCE	00	0%
CHYLOTHORAX	00	0%
HEMOTHORAX	03	12%
VOCALCORD PALSY	01	04%
PNEUMONITIS	02	08%
INTESTINAL OBSTRUCTION	00	0%
DEATHS	02	08%

The cervical anastamotic leak is present in 4 patients (16%) which had resolved with conservative management. About 3 patients (12%) had stricture, who were submitted to repeated Endoscopic dilatations. Three patients (12%) had hemothorax and two (8.0%) had pneumonitis, which were managed

conservatively. One (4.0%) had vocal cord palsy, which could be due to accidental injury of Recurrent Laryngeal Nerve during blunt thoracic dissection.

The post-operative mortality was 8.0% (2 patients), out of which one had **Myocardial Infarction** and another had **Atrial Fibrillation.** However, 7 patients (24%) had an uneventful postoperative period.

Follow up periods of the patients were variable.

PERIOD	NUMBER	PERCENTAGE
<1 MONTH	08	32%
1-3 MONTHS	10	40%
4-6 MONTHS	15	60%
7-9 MONTHS	12	48%
10-12 MONTHS	08	32%
1-2 YEARS	04	16%
> 2 YEARS	03	12%
LOST FOR FOLLOW-UP	06	12%

Patients who underwent incomplete treatment are also included in the group for lost follow-up. The mean period of follow-up was 7.6 months.

Recurrence of the disease in the form of adenopathy, visceral secondaries or recurrence at the previous site were also registered.

RECURRENT DISEASE	NUMBER	%
AT THE SITE	06	12%
LYMPH NODE	02	04%
VISCERAL SECONDARIES	03	06%

Restenosis is being considered as recurrent disease at the site. These patients were managed with Radiotherapy.

DISCUSSION

EPIDEMIOLOGY, SEX AND AGE INCIDENCE

The epidemiological characteristics of esophageal carcinoma are unusual, since its incidence in different geographic areas is extremely variable, with the greatest differences recorded for all tumors. The incidence of esophageal carcinoma varies from 8.1% recorded at the Chennai registry to 4.6 at Delhi. The incidence as per the surveillance made by the **National Cancer Registry Project (NCRP)** quotes an incidence of 8.6% at Bangalore and 6.8% at Mumbai. The incidence of carcinoma esophagus in our institution is 0.07%, which is relatively lower.

As per the study, the rise in esophageal cancer commences in the thirties and peaks in the 5^{th} decade. Studies conducted both in India and abroad, show peak incidence in the 7^{th} and 8^{th} decades. The exact cause of this difference in this regard is not known.

ETIOLOGY AND RISK FACTORS

All the patients in our study, who presented with esophageal carcinoma were of the lower socioeconomic group. ***Day and Munoz,*** **1982** and ***Schottenfeld*** **1984**, and several other series have shown an association between esophageal cancers and low socioeconomic status.

Low levels of retinol, riboflavin, Ascorbic acid, and alpha-tocopherol are prevalent in the population of Linxian, China, where esophageal cancer is epidemic. In Japan, poor food variety has been identified as a risk-enhancing factor and combinations of fruits, vegetables and fresh meat appear to be risk-reducing factors.

De Carli et al 1989 had stated that low intake of fruits, particularly citrus fruits and accordingly, reduced Vitamin C intake has been repeatedly associated with an increased risk of esophageal cancer. Deficiencies in various mineral elements such as selenium, Zinc, Molybdenum also have been cited as possible etiologic factors.

Francheschi et al 1990 discovered that the deficiencies are believed to make one more susceptible to the carcinogenic effects of exogenous factors.

From the data given in our study there is a strong association between the use of tobacco in both of its forms of usage viz., chewing and smoking and the development of esophageal cancers.

The most important risk factors for cancer of esophagus in developed countries are cigarette smoking ***(IARC 1986)*** alcohol consumption ***(IARC 1988).***The associations between cigarette smoking, alcohol consumption and esophageal

cancer are difficult to separate, largely because of the correlation in the two exposures and their mutual associations with the risk of cancer esophagus.

The risk of esophageal cancer has also been shown to be increased among non-tobacco smokers who consume alcohol and among non-drinkers of alcohol who smoke tobacco ***(La Vecchia and Negri 1989).***

The role of alcohol consumption was not clearly demonstrated in the French Department of Ille-et-Villaine where the risk rose steadily with the amount of alcohol consumed ***(Tuyns et al 1077)***

The risks associated with tobacco use appear to increase with the number of cigarettes smoked per day, duration of smoking, and tar content. ***(Tuyns et 1979 ; Rossi et al 1982 ; Yu et al 1988).***

A synergistic effect for the combined habit of alcohol drinking and tobacco smoking or chewing has also been reported.

MORPHOLOGICAL TYPE AND LOCATION

The predominant histomorphology our study was the squamous cell carcinoma which accounts for about 72% of the toal oesophageal cancers at the TMCH. In Europe, the incidence of adenocarcinoma rose to double fold in the 1990s.

Steiger et al 1987, stated that Primary Adenocarcinoma represent 3 to 8% of the esophageal cancer. Observations made in our study also show a rise in the incidence of adenocarcinoma. It accounts for about 24% of the esophageal cancers.

Esophageal cancer is usually located in the middle third in about 50% of the cases; the lower thoracic and upper thoracic esophagus are involved in a similar percentage of cases and the cervical esophagus is involved less frequently. ***(Guili and Gignoux 1980)***

In our study, we found that the most commonest site of the tumor is the lower third which corresponded to about (51%), while middle third was the commonest site in about 30% of patients. A few patients had growth involving the upperthird (9%)

SURGICAL THERAPY

In the past, esophageal surgery was burdened by high operative morbidity and mortality rates, the highest known in surgery ***(Lauois et al 1983).*** There has been a remarkable reduction in these rates in the last 10 years.

The pre-operative care of a patient who has to undergo esophagectomy should include a prophylaxis for any post-operative complications, particularly regarding respiratory problems. Smoking should be stopped at least 10 days before surgery

and in the presence of pulmonary problems, physiotherapy is advisable together with pre-operative bronchodilatation treatment. If the patient presents with malnutrition, hypercaloric parenteral nutrition or tube enteral feeding, at 2500 to 3500 calories\day, is advisable for a period of 7 to 10 days ***(Moghissi et al 1977)***

The selection of patients for Transhiatal esophagectomy is very important in computing to the outcome. Most of the time it depends upon the general condition (if operable) of the patient and the tumor stage (if resectable).

Usually the lower third esophageal growths and the lower half of the middle third (subcarinal) growths are the suitable growths for Transhiatal esophagectomy.

The contraindications for esophagectomy can be relative to the patient or to the tumor. At present, an elderly patient ***(Peracchia et al. 1988),*** the length of the tumor and the concomitance of Child-A risk liver cirrhosis ***(Fekete et al 1987)*** are not considered absolute contraindications for surgery.

SURGICAL APPROACH

Transhiatal esophagectomy without Thoracotomy ***(Orringer et al 1993)*** has been performed by an increasing number of authors in recent years. It is performed by isolating the mediastinal esophagus through a cervicotomy and laparotomy ***(Orringer et al 1984, 1987).*** We at the TMCH, have adopted this technique in the selected patients, since it reduces the time of the operation

compared with transthoracic esophagectomy and allows surgery to be performed on patients who would not tolerate a thoracotomy.

In our experience, this surgery seems to be properly indicated for cancers of lower third intrathoracic esophagus and for selected cardial cancers.

Akiyama et al 1978 stated that stomach is the viscus of choice to replace the esophagus resected for cancer. It is isolated and tubulized before transposition. We do agree with the statement, since stomach tubulization allows removal of the lymph nodes located the left gastric vessels, a possible metastasis station, improves the gastric vascularization and avoids mediastinal encumbrance which is possible when the whole stomach is transposed.

Interpositioning of a colonic segment and the transposition of a Roux-en-Y loop of jejunum was also done on two cases of lower esophageal cancer at TMCH. The results were not encouraging. This was partly due to fact that patients were not able to tolerate this long duration procedures.

Esophagogastric anastomosis is performed in our institution only by using hand sewn techniques.

Wong et al 1987 identified that the main post-operative complication is the anastomotic leakage. The anastomotic leak rate in our cases is well within acceptable range. It was about 16% (4\25)

RESULTS OF SURGICAL RESECTION

Contrasting data regarding the resectability rates and the long-term survival rates are reported in the literature. This is because there are very different therapeutic attitudes in the various centres ; that is, aggressive of conservative, varying criteria for the selection of the patients for the different types of treatment and a multiplicity of therapeutic protocols and schemes.

At present, the most experienced surgical teams report a postoperative mortality rate below 5 – 10% ***(Peracchia et al 1988; Muller et al. 1990)***

Observations made in our study report a mortality rate of about 8.0%.

POST-OPERATIVE MORTALITY

STUDIES	PERCENTAGE
KATARIYA et al	6.7%
MOON et al	7.3%
MICHIGAN UNIVERSITY	7.2%
GOLDMINC et al	6.4%
BOLTON et al	5.9%
SCHAKELFORDT et al	5.7%
ORRINGER et al	4.0%
OUR STUDY	8.0%

In our study, the cervical anastomotic leak was present in about 12% of patients which were managed conservatively.

STUDIES	PERCENTAGE	
	ANASTOMOTIC LEAK	STRICTURE
KATARIYA et al	15.1%	14.1%
GOLDMINC et al	14.0%	16.0%
MICHIGAN UNIVERSITY	12.0%	13.5%
SCHEKELFORDT et al	13.5%	14.6%
ORRINGER et al	07.9%	09.0%
OUR STUDY	16.0%	12.0%

About 12% of our patients had post-operative strictures, who were managed with enodoscopic dilatation. Incidence of both the anastomotic leaks and strictures in our study are well comparable with the international literature. These complications are relatively less in Transhiatal esophagectomy.

STUDIES	PERCENTAGE	
	VOCALCORD PALSY	CHYLOTHORAX
KATARIYA et al	11.3%	0.71%
GOLDMINC et al	10.5%	0.6 %
MICHIGAN UNIVERSITY	9 %	0.5%
SCHEKELFORDT et al	8.6 %	0.7 %
ORRINGER et al	19.2 %	0.2 %
OUR STUDY	4 %	0 %

We found vocal cord palsy (4%), post-operatively, which can be explained by the probability of accidental injury during blind thoracic dissection. No patient had Chylothorax, in our study.

STUDIES	PERCENTAGE	
	PULMONARY	CVS
KATARIYA et al	50 %	11.9 %
GOLDMINC et al	34 %	12 %
MICHIGAN UNIVERSITY	12 %	16.0 %
SCHEKELFORDT et al	36 %	09.8 %
ORRINGER et al	19.2 %	0.2 %
OUR STUDY	8 %	8 %

The pulmonary complications were in the form of pneumonitis, which were managed with higher antibiotics. Two died of Cardiovascular complications, one due to Myocardial Infarction, another due to Atrial Fibrillation.

About 3 of the 25 patients, who were offered surgical treatment are alive after 2 years which accounts for about 16%. This figure, actually, represents the actual number of patients cured of the disease. About 4 (16%) are alive after one year without any evidence of recurrent disease or other complications .

STUDIES	PERCENTAGE	
	ONE YEAR	TWO YEARS
KATARIYA et al	26.2 %	14.8 %
GOLDMINC et al	29.7 %	19.7 %
MICHIGAN UNIVERSITY	25.4 %	17.2 %
SCHEKELFORDT et al	31 %	18.5 %
ORRINGER et al	33.7 %	21.6 %

OUR STUDY	20.01 %	16 %

Since our study is of short duration (2 1\2 years) we are unable to calculate the 5 year survival rate. The actual 5 year survival rate after curative esohagectomy varies from 15% to 30% on the basis of tumor stage. Combining T H E with Thoracoscopic Lymphadenectomy may improve survival and needs evaluation by a trial.

CONCLUSION

1. Carcinoma esophagus is one of the most important leading sites of cancer in our country.

2. It is one among the cancers that have increased male : female ratio.

3. Carcinoma esophagus is more common in lower socioeconomic group.

4. The predominant histomorphology is Squamous cell carcinoma.

5. Carcinoma is more common in the lower 3rd esophagus and it is rare in the upper 3rd.

6. There is a significant rise in the incidence of Adenocarcinoma.

7. A strong association exists between carcinoma esophagus with smoking and alcoholism.

8. The Transhiatal esophagectomy scores significant role in the surgical management of lower 3rd and lower half of the middle 3rd growths.

9. The T H E avoids the risks of Thoracotomy and minimizes the hospital stay.

10. TransHiatal esophagectomy carries less postoperative complications, do not need mechanical ventilatory support.

11. Both Intra-operative and Post-operative mortality is less.

12. Signifcant 1 year and 2 year survival rates can be achieved with this surgical procedure.

PROFORMA

NAME **AGE** **SEX**

OCCUPATION **I.P NO.** **UNIT \ WARD**

ADDRESS

COMPLAINTS:

HISTORY OF PRESENTING ILLNESS

DYSPHAGIA **DURATION**

ODYNOPHAGIA **HOARSENESS**

CHEST PAIN **COUGH**

ANOREXIA **DYSPNOEA**

WEIGHT LOSS **HEMOPTYSIS**

REGURGITATION **HEMATEMESIS\MALEANA**

OTHERS

PAST HISTORY

IRRADIATION **DIVERTICULA**

CORROSIVE STRICTURES **TYLOSIS**

BARRETTS **OTHERS**

P V SYNDROME

PERSONAL HISTORY

MARITAL STATUS **DIET**

SMOKING **ALCOHOLISM**

OTHERS

FAMILY HISTORY

PHYSICAL EXAMINATION

PR **BP** **RR**

BUILT **NUTRITION** **ANEMIA**

JAUNDICE **LYMPHADENOATHY**

LOCAL EXAMINATION;

MOUTH AND PHARYND

NECK

CHEST

ABDOMEN

SPINE & CRANIUM

INVESTIGATIONS

URINE **HEMOGLOBIN**

BLOOD SUGAR **UREA**

CREATININE **ELECTROLYTES**

1 **ELECTROCARDIOGRAM** **CHEST X RAY**

ENT OPINION

BARIUM SWALLOW

ENDOSCOPY

BIOPSY

USG ABDOMEN

CT THORAX & ABDOMEN

FNAC OF LYMH NODES

TREATMENT

CURATIVE;

SURGERY

TRANS HIATAL ESOPHAGECTOMY

COMPLICATIONS

PALLIATIVE;

RADIOTHERAY

FEEDING JEJUNOSTOMY

STENTING

SURVIVAL RATE.

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ME	A/S	IP.NO	ADD	C/O	HABITS	SITE	BIOPSY	TREAT.	COMPLICAT
ASAMY	**55\M**	**804350**	**ORATHAND**	**D,W,V**	**B,S**	**L**	**S**	**T H E**	**S, I**
MPATH	45\M	813478	VALLAM	D,V,A	S,A	M	S	T H E	
PA	45\F	822942	POONDI	D,W,A	T	L	A	CT	
NIMEGALAI	69\F	825465	TIRUVARUR	D,V,A	B	M	S	STEN	
TONY	40\M	826515	ARIYALUR	W,V,A	B,S	L	S	T H E	
AKAMI	40\F	826854	MELATOR	D,A,H		M	A	FJ	
VINDAMAL	65\F	828768	PAVAKADU	D,V,O	T	M	S	RT	
INATHAN	56\M	828885	MANAMEDU	D,O,A	B,S	L	A	T H E	E
RADARAJAN	50\M	829292	TRICHY	W,V,H	T,S,A	M	S	FJ	
RAN	55\M	830220	KUPAMEDU	D,O,A	S	M	S	T H E	
RAMANIAN	47\M	835425	KUDAVASAL	D,W,A	B	M	S	RT	
NGAPPAN	50\M	837040	KUVALYOR	D,V,A	T	M	S	T H E	H
RAIRAJ	52\M	837530	TRICHY	D,W,A	S,A	L	A	T H E	
DAKRISHNAN	59\M	839354	TNJ	D,O,A	B	L	S	RT	
NJIUM	65\F	839601	PAPANASAM	A	T	L	A	T H E	L
NMUGAM	50\M	867513	TNJ	D,W,A,H	T,S	L	A	T H E	I
VINDARAJ	56\M	867942	KARUR	D,W,A	T,S,A	M	S	RT	
AMMAL	35\F	868370	IDAYATHI	D,V,O,H	B	M	S	RT	S
RUGAN	65\M	860094	TIRUVARUR	D,W,O,H	T,A	L	S	FJ	
MASAMY	65\M	883014	MANNAI	W,V,A,D	B,A	M	S	T H E	L,V
HOORAN	65\M	884448	TIRUKUVALA	D,V,H	T,S	M	S	RT	
RUGAIYAN	54\M	887445	POONDI	D,W,O,H	B,S,A	M	S	RT\CT	
GASAMY	60\M	889965	KUNNAM	D,V,A		L	A	FJ	
AGANATHAN	38\M	891895	MANALMEDU	W,V,H	T,S,A	M	S	T H E	I
THOSAM	56\M	899654	TRICHY	D,A	T,S	L	S	RT	
LAKAVATHI	40\F	841528	THIRUVAYAR	D,W,A	B	L	S	T H E	S
RAMANIAN	47\M	841664	NAGORE	D,W,	T	L	A	T H E	
NIYAMMAL	50\F	869037	PAPANASAM	D,A	B	M	A	CT	
RUPPAIAH	60\M	872518	TRICHY	D,W		L	S	T H E	L
MASAMY	65\M	873572	MANNAI	D,W,A	T	L	S	RT	
NESAN	69\M	874272	PATUKOTAI	W,A	T	L	S	T H E	I
LAR	36\F	875445	TNJ	A	B	L	S	RT	
THUSAMY	62\M	876278	DALMIAPET	D,W	T	M	A	STEN	
AJI	48\M	876702	ARANTHANGI	D,W	B	L	S	FJ	
VINDARAJ	65\M	877432	KOMBANCHERY	A	T	L	S	T H E	
AMELU	65\M	8789688	TV MARUDUR	D		M	S	FJ	
GILIMUTHU	60\M	8810288	TNJ	A	T	L	A	T H E	L,H
YAPURI	67\M	881719	KEEVALUR	D,W,Q	B,S,A	M	S	T H E	
THAMMAL	55\F	881849	KUDAVASAL	D,A	T	L	A	CT	
THALAXMI	45\F	883633	MUTHUPET	D,O,A	T	L	S	T H E	E
AKIRAMAN	55\M	841943	ORATHANAD	V,A	B	L	S	STE	
VICHANDRAN	38\M	841943	MANNAI	D,W,A	T,S,A	M	S	T H E	P
AGOPAL	62\M	843421	ALIVAIKAL	W,A,H	B,S	L	S	RT	
THALAXMI	54\F	846674	KUDANTHAI	D,W,O	B	L	A	T H E	
NGARAJ	50\M	847730	NK ROAD	V,A,H	T	M	S	T H E	
EKYALASHMI	45\F	854962	TRICHY	D,V,O,A		L	A	T H E	P,H
VARANI	30\F	8575421	PANKANNADU	D,W,O	B	M	A	T H E	

S.NO	NAME	A/S	IP.NO	ADD	C/O	HABITS	SITE	BIOPSY
51	PONAMBALAM	62\M	8100461	PONAMBALAM	D,W,L	S,	M	S
52	AYYAKANNU	50\M	811840	TRICHY	D,L	S,A	U	S
53	VEERAIAN	48\M	813088	PUDUKUDY	A	S,B	L	S
54	SENTHAMARAI	47\M	821127	VALLAM	D,V	S,T	M	A
55	SAROJA	50\F	822351	TNJ	D,A	S,A	L	S
56	MARUDAMUTHU	69\M	823149	JEYANKONDAM	L,A	B	M	A
57	PUNIYAMURTHY	45\M	823391	PATUKOTAI	C	T	U	S
58	MANIYAMMAL	60\F	823391	TV MARUDUR	C	B	L	A
59	MOORTHY	63\M	823698	VELANKANI	D,W,	T	L	S
60	KARUPPAIYA	69\M	826384	BAGORE	D,W,	A	M	A
61	SELVARAJ	40\M	826338	TIRUVARUR	A	S	L	S
62	NATARAJAN	57\M	8826649	TRICHY	C	A	L	S
63	THANGAVEL	69\M	827256	PUDUKOTAI	D,L	BN	L	S
64	NAGARAJAN	57\M	829375	KANDARVAKOT	D,L	B,T	L	S
65	DAKSHINA	60\M	830530	KALAKUDI	A,L	T	M	A
66	KANAKAN	56\M	830535	VETIKADU	A,D	B	L	A
67	JAGANATHAN	64\M	832092	PUDALUR	V	B	M	S
68	MUTHAMMAL	53\M	835621	PILLAYARPET	C	A	U	S
69	SHANTHI	48\F	836128	MUTHUPET	C	A,T	M	A
70	SIVAN	49\M	82746	TRICHY	H,L	S,B	L	A
71	SUGUNTHAN	39\M	871024	VELLAKADU	D	A	M	S
72	RAJAN	65\M	856432	KURUNKULAM	D,A	T	U	S
73	VADIVEL	45\F	836934	MANNAI	D.A	T	L	S
74	KALIYAPERUMAL	50\F	838141	PATIKAD	A	S	L	S
75	OGIR BEEVI	45\F	839656	KATAMPET	H	T B	M	S
76	RATHNAM	60\F	840093	PERAMBALOR	D,L	B	L	A
77	GANESAN	66\M	840216	ARIMANUR	L,A	S A	L	A
78	MAMURDY	50\M	840375	SETIKADU	L,W	S	U	S
79	SAMINATHAN	60\M	840985	MELATUR	D.H	S	M	S
80	SUBRAMANIAN	47\M	841132	TIRUVARUR	L,A	S	L	A
81	TAMILARASI	50\F	841398	ARIMANUR	D,A		L	A
82	SOMASUND	55\M	842403	SIRUKULAM	D,L	S	U	S
83	NATARAJAN	50\M	841910	KANDARVAKO	D,A,V	A	L	S
84	RAJAMANIKAM	45\M	843412	TIRUTHURAI	D,L		M	S
85	DURAIRAJ	48\M	844257	MANAMEDU	D,.V	A	L	S
86	MEENAKSHI	69\M	844864	MATIKADU	D,V	T	L	S
87	RAJGOPAL	62\M	846428	MUTHUPET	A,C	B	U	A
88	TAMILSELVI	30\F	846428	PAVADI	D,C	B	M	A
89	RAJ	67\M	883105	PERUMPALLAM	C,D	A	M	S
90	SUGUKUMAR	35\M	866174	KALIKAVAL	L	A	M	S
91	RAKAMAL	67\F	707105	TRICHY	D		U	NOS
92	AYYAVU	35\M	861301	MANNAI	W	C	U	S
93	GOMATHY	42\F	816305	NANDANAM		H	L	S
94	MEENAL	52\F	8588560	IIRATHANAD	W		L	A
95	MURUGAN	55\M	860094	PATALAM	D	L	U	S
96	MUTHAMMAL	69\F	860493	PPATI	D	A	L	S
97	ALAGU DURAI	58\M	860639	TNJ	D	A	U	S
98	KULANDAI	65\M	860798	TRICHY	A		U	NOS
99	KALIAMMAL	55\F	870712	PAPANASAM	A	L	U	S
100	SELVARAJ	58\M	870771	VILUKADU	W	L	S	A

S.NO	NAME	A/S	IP.NO	ADD	C/O	HABITS	SITE	BIOPSY
101	MALAR	49\F	870331	TNJ	D	T	U	S
102	NAGAMMAL	46\F	870445	TRICHY	D	T	U	S
103	SARAVANAN	59\F	875585	PULIKADU	A	A	L	S
104	BALAMMAL	56\F	875689	TRICHY	W		L	S
105	JEMILA	49\F	875703	VELLAPURAM	W	B	L	A
106	SAGUNTALA	47\F	879021	TNJ	D	A	M	S
107	RASU	54\M	897827	TRICHY	D	A	M	S
108	NAGAMMAL	59\F	88670	KANDARVA	A	B	L	A
109	JEETA BEEVI	48\F	89876	MELATOR	D	B	M	S
110	NAGAMMAL	60\F	889106	SIRUNGULAM	D,W,	T	U	S
111	FATHIMA	45\F	883603	TRICHY	A	T	L	S
112	MASILAMANI	55/F	88883437	PAPANASAM	D,L	B	L	S
113	NACHIYAR	5/F6	882632	AYYAMPET	D,L	B	L	S
114	RASU	58/M	880679	MARIA.KOVIL	D,A,		M	S
115	VAIRAPPU	59/F	872347	NEEDAMANG	D	B	L	A
116	VAIRAM	64/F	825463	TIRUKAVUR	A	B	M	S
117	MANIYAMMAL	69/F	836574	TIRUKAVUR	D	T	M	S
118	PATTAMBAL	58/F	884000	TRICHY	A	B	L	A
119	GANESAN	66/M	844140	TIRUCHITRAM	A,D	B	L	A
120	MEENAKSHI	64/F	846545	VALLAM	L		L	S
121	TAMILARASI	56/F	8842993	VURAIYUR	D,		U	S
122	SARADAMBAL	55/F	883612	TRICHY	A	B	U	S
123	MARUDAN	34/M	838231	TRICHY	S	S,A	L	S
124	CINNAN	55/M	876451	MANAMED	A	A	L	A
125	KALIYAN	67/M	889432	PATIKADU	D	S,A	L	S
126	MANIAMMAL	78/F	878652	KULATHALAI	A	T	A	A
127	BAGKAM	33/F	878643	MUSIRI		T	A	A
128	MURUGAN	66/M	887643	TNJ	L		L	S
129	RENGAMAL	65/F	911206	SIRUKAVORD,	D,	T		
130	DEIVANAI	50/F	909073	POONDI	A	T		
131	SAMI	62/F	909600	TRICHY	A			
132	GNANAMANI	55/F	908788	TNJ	A	B		
133	MASILAMANI	55/F	889281	NAGAI	D	B		
134	VASUDEV	50/M	889438	PUDUKUDY	D	S		
135	MEENAKSHI	69/F	844257	PUDUKOTAI				
136	TAMILSELVI	35/F	846545	TNJ	L	B		
137	MURUGESH	56/M	887435	TRICHY	L	A		
138	NAGESH	36/M	88225	MANNAI	A	S		
139	CHELLAMAL	46/F	908788	PAPANASAM	A	T		
140	CINDAMANI	58/F	909079	PUNALVASAL	D	T		
141	NAHOORAN	49/M	884477	KUDAVASAL	D			
142	MANOHARAN	68/M	894345	TNJ	A			
143	SIVAKUMAR	67/M	882343	TRICHY		S		
144	GEETHA	64/F	898453	NAGAI	H	A		
145	PREMA	69/F	909454	VELANKANNI	A			
146	LATHA	44/F	989091	NAGORE				
147	SUJATHA	66/F	897654	TNJ	D		L	S
148	MANJULA	68/F	88765	TRICHY	A		U	A
149	MANI	66/M	88765	MUSIRI			L	S
150	RAVI	56/M	88789	VALLAM			L	S