**DISSERTATION ON**

# CLINICAL AND EPIDEMIOLOGICAL STUDY OF PAROTID GLAND TUMOURS

**M.S.DEGREE EXAMINATION**
**BRANCH – I**
**GENERAL SURGERY**


**THANJAVUR MEDICAL COLLEGE AND HOSPITAL**

**THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY**

**CHENNAI**

**MARCH - 2009**

# CERTIFICATE

This is to certify that dissertation entitled **A CLINICAL AND EPIDEMIOLOGICAL STUDY OF PAROTID GLAND TUMOURS** is a bonafide record of work done by **XXXXX,** in the Department of General Surgery, Thanjavur Medical College, Thanjavur, during his Post Graduate Course from 2006-2009 under the guidance and supervision of **PROF. DR. T. ANANTHARAMAKRISHNAN M.S., F.I.C.S**. and **PROF. DR. G. AMBUJAM, M.S. FICS.** This is submitted in partial fulfillment for the award of **M.S. DEGREE EXAMINATION- BRANCH I (GENERAL SURGERY)** to be held in March 2009 under the **Tamilnadu Dr. M.G.R. Medical University, Chennai.**

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# DECLARATION

I declare that this dissertation entitled '**A CLINICAL AND EPIDEMIOLOGICAL STUDY OF PAROTID GLAND TUMOURS** is a record of work done by me in the department of General Surgery, Thanjavur medical college, Thanjavur, during my Post Graduate Course from 2006-2009 under the guidance and supervision of my unit chief **PROF. DR.T.ANANTHARAMAKRISHNAN,M.S.,F.I.C.S,** and professor and head of the department **PROF. DR. G. AMBUJAM, M.S., FICS.** It is submitted in partial fulfillment for the award of **M.S. DEGREE EXAMINATION- BRANCH I (GENERAL SURGERY)** to be held in March 2009 under the **Tamilnadu Dr. M.G.R. Medical University, Chennai.** This record of work has not been submitted previously by me for the award of any degree or diploma from any other university.

**XXXXX**

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# CONTENTS

# INTRODUCTION

Neoplasm of salivary glands are challenging because of their relative infrequency, inconsistent classification and highly variable biologic behaviour.

These factors present some difficulty when attempting to compose data from various institutions describing their experience with salivary gland tumours.

The salivary gland tumours are relatively rare and constitute 3-4% of all head and neck neoplasms. The majority (70%) of all salivary gland tumours are in the parotid gland. Of these, three fourths are benign.

In this study, sincere effort is made to analyse parotid tumours during the years May 2006 to November 2008 in our institution. The use of FNAC as a pre-operative investigation tool in the management of benign and malignant parotid tumours is also analysed with the help of statistics and comparing them with the literature.

# HISTORICAL REVIEW

Previously known as "**Para auricular swellings**" by Greeks. In the middle of 17$^{th}$ century, the anatomy of the parotid gland and role of the parotid ducts were appreciated.

NIELS STENSEN in 1660 identified the parotid duct during the dissection of a

sheep's head, but he never knew of the relationship between the duct and the gland.

The medial prolongation of the parotid was noticed by THOREK (1962). Its development from ectodermal origin and from buccal epithelium was discovered by MOOR (1974). Opening of the parotid duct in the vestibule of the mouth was noticed by HAINS and MOHIUDDIN (1972). The capsule of the parotid gland was described by CONLEY (1975).

While SANCHEZ (1960) described the attachment of capsule to zygomaticarch and stylomandibular ligament.

The innervation of the parotid gland was discovered by various methods by REIHIERT (1934), KUNTZ (1946), and GENS–GALVEZ (1966). LANGMAN (1976) mentioned that the glossopharyngeal nerve, which is the nerve of the third arch, supplies the parotid gland. SHELL described the pterygoid process of the gland.

DUPLIESSIS (1975) said that the gland has superficial and sub-facial planes and stated that enucleation of the gland was possible without damaging the facial nerve.

Between 1650 and 1750, salivary gland surgery was limited to the treatment of ranulas and ductal calculi.

The concept of parotidectomy for the treatment of parotid tumour was attributed to BETRANDI (1802). Initially surgeons were concerned primarily about hemorrhage and patients had to leave behind with major disfiguration if they were fortunate enough to survive a parotid resection. The first operation to employ ether inhalation was a parotid tumour resection performed by Dr.JOHN.C.WARREN in BOSTON in 1846.

The first total parotidectomy (facial nerve preservation) was done by CODREANU, a Romanian in 1892. BLAIR and SISTRUNK systematized the surgical approach to facial nerve. First facial nerve grafting was done early in 1990s.

# REVIEW OF THE LITERATURE

**Surgical Anatomy**

The paired parotid gland are the largest of the salivary glands, each weigh about 25g and is an irregular, lobulated yellowish mass, lying largely below the external acoustic meatus between the mandible and the sternocleidomastoid. It also projects forwards on the surface of the masseter, where a small, detached part lies between the zygomatic arch above and the parotid duct below, the pars accessoria (or) social parotids.

The glandular capsule is derived from the deep cervical fascia. Its superficial layer is dense, closely adherent to the gland and attached to the zygomatic arch; medial to the gland, it is attached to the styloid process, mandible and tympanic plate blending with the fibrous sheaths of related muscles, the fascia extending from the styloid process to the mandibular angle to form the stylomandibular ligament between the parotid and submandibular gland.

The superficial surface of the gland is triangular in shape, with apex pouting inferiorly. The deep surface of the gland is wedged into the parotid space.

**PAROTID SPACE:**

The parotid space has a skeletal back- ground created.

Anteriorly: By the ramus of the mandible

Medially: By the styloid process

Posteriorly: By the mastoid process

Postero superiorly: By the external acoustic meatus and the temporo mandibular joint.

The muscles that are attached to these bony landmarks create the soft tissue background.

**LOBES**

The parotid gland is somewhat artificially divided into two lobes by the facial nerve.

1. Endo facial (deep)
2. Exo facial (superficial).

There are however multiple communications between the lobes, created by bridges of glandular tissue. A large area of communication referred to as isthmus, is related to the proximal part of the interior parotid portion of the facial nerve.

Approximately 10% of the gland extends medially through the stylomandibular tunnel, which is formed ventrally by the posterior edge of the mandibular rami, dorsally by the anterior border of the sternocleidomastoid muscle and the posterior belly of the digastric muscle, and more deeply and dorsally by the stylomandibular ligament, which separates the parotid gland from the submandibular gland.

The deep portion of the gland lies in the pre-styloid compartment of the Parapharyngeal space, being anterior to the styloid process and its musculature, the carotid sheath and the cranial nerves IX-XII. Because the deep lobe of the parotid is close to lateral pharyngeal wall, an enlarged deep lobe (or) a dumb – bell tumour will push the tonsillar fossa and soft palate intra orally to the opposite side.

Five processes of the parotid gland have been described. They are the superficial three namely condylar, meatal and posterior and deep consisting of the glenoid and stylomandibular. Because of these extensions and that the facial nerve courses through the substance of the nerve, it is nearly impossible to remove all parotid tissue when a total parotidectomy is performed.

The parotid duct courses anteriorly on the lateral surface of the masseter muscle and buccal pad of fat and then turns medially almost at right angles goes through the buccinator at the level of the upper second molar. The parotid duct during its course may receive the duct of the accessory parotid gland. (Fig.1)

**NEURO VASCULAR ANATOMY**

The greater auricular nerve is the first neural structure to be encountered when the skin-flaps are raised. The nerve is divided into anterior and posterior branches.

The Auriculotemporal nerve is the other nerve that could be spared during the removal of parotid tumours.

The Auriculotemporal nerve, a branch of the fifth cranial nerve, is found at the posterior aspect of the gland, coursing superiorly with the superficial temporal artery and vein. It is a sensory nerve to the temporal region and carries parasympathetic fibers to the parotid gland to form the Otic ganglion.

The veins traverse the middle portion of the gland deep to facial nerve. The superficial temporal vein unites with the internal maxillary vein to form the posterior facial vein, which branches into anterior and posterior divisions. The anterior branch joins the anterior facial vein to form the common facial vein, whereas the posterior division unites with the posterior auricular vein to form the external jugular vein. On its decent, the external jugular vein is found on the lateral aspect of the sternocleidomastoid muscle, anterior to the greater auricular nerve. This anatomic arrangement is often used to help locate the proximal portion of the nerve for grafting purposes.

The external carotid artery and its terminal branches, the internal maxillary and the superficial temporal arteries, traverse the deep portion of the parotid gland (Fig.2). The superficial temporal artery ascends between the mandibular condyle and external

auditory canal. Precise localization of this artery occurs just at the root of the zygoma anterior to the auricle. The internal maxillary artery, which may be encountered during total parotidectomy or removal of deep lobe tumours in the para pharyngeal space, is found between the mandibular ramus and sphenomandibular ligament, coursing in close proximity to the lateral pterygoid muscle and heading towards the pterygopalatine fossa.

## FACIAL NERVE

The facial nerve exits the stylomastoid foramen from its intra temporal course. After the stylomastoid foramen, it gives off branches to stylomastoid, posterior belly of digastric and posterior auricular muscles before its transition to pes anserinus. The facial nerve enters the parotid gland on its posterior surface prior to the formation of the pes anserinus, approximately 1 cm anterior and 2 cms inferior to the tragus. The superior division of the facial nerve follows a slightly curvilinear course (Fig 3a $3b).

The triangular end of the tragus, that is termed as **the tragal (Conley's) pointer,** has its value in identifying the landmark of facial nerve. The palpable depression between the bony region of the external auditory canal and anterior aspect of mastoid tip is termed "**The valley of the nerve".** Within this valley, is the tympano mastoid suture line, which reliably leads directly to the facial nerve.

The facial nerve, as it enters into the substance of the parotid gland divides into the two major trunks, temporofacial and cervicofacial. DAVIS and others have studied six type of branching pattern of these nerves extensively.

Six types of facial nerve branching has been described based on anastomosis of individual branches (Fig.4). Type II occurs in 37.5% of cases, and is the commonest pattern seen. The temporofacial consists of temporal, zygomatic, and buccal. The cervicofacial consists of marginal mandibular and cervical branches. The trunks divide distally within the superficial lobe to their respective territories on the face and neck.

The parotid region contains the following compartments.

**Superficial (or) the nerve compartment contains**

- Greater auricular nerve
- Auriculo temporal nerve
- Facial nerve

**Middle compartment contains**

- Superficial temporal vein.
- Internal maxillary vein.
- Anterior and posterior branches of the posterior facial vein.
- Posterior auricular vein.
- External jugular vein.

**Deep compartment contains**

- External carotid artery
- Internal carotid artery
- Superficial temporal artery

**Parotid gland innervations**

Via the Auirculotemporal nerve.

Sensory innervations are from the Trigeminal nerve, having cell of origin in the Trigeminal ganglion.

**Parasympathetic Innervations**

Is from the Glossopharyngeal nerve. The postganglionic parasympathetic cell bodies are located in the inferior salivary nucleus in the medulla oblongata.

The preganglionic parasympathetic (Jacobson's) branch originates in the jugular fossa. The fibers leave the tympanic plexus by way of the lesser petrosal nerve, leave the skull through the foramen ovale or its own bony canal and terminates in the Otic ganglion. Para sympathetic fibers from CN VII may reach the otic ganglion via the chorda tympani nerve and anastomose between CN IX and CN II.

**Sympathetic innervation**

Is from the superior sympathetic ganglion and reaches the parotid via the external carotid plexus and its branches.

The parotid gland is associated with a rich network of lymphatics. During embryogenesis, the parotid anlage is the first to develop but it becomes encapsulated after submandibular and sublingual gland. This delayed encapsulation is significant because it results in entrapment of the lymph nodes. In addition, during encapsulation of the intra parotid and periparotid lymphnodes, salivary epithelial cells can be included within these nodes. It is this special embryogenesis of the parotid gland that is believed to play a role in the development of Warthin's tumours and possibly lymphoepithelial cysts.

The pre auricular and infra auricular lymph nodes constitute the para parotid (superficial to the capsule of the gland) group, which drain the temporal region of the scalp and auricle, the intraparotid (intra-parenchymal) node receives lymphatics from the posterior nasopharynx, soft palate and the ear. Hyperplasic or neoplastic involvement of these nodes may manifest as parotid mass.

**Parotido massetric fascia:**

- Covers the muscle and splits to invest the parotid gland.
- Attaches to the zygomatic arch superiorly.
- Continues inferiorly as the investing cervical fascia over the sternocleidomastoid muscle.

Is fused with the dense and tough superficial fascia covering the gland. Sends many irregular septae into the gland, making many compartments within it. Unlike the submandibular gland, the parotid gland cannot be easily separated from its connective tissue, capsular bed.

Extends deep to the posterior border of the ramus of the mandible, where it fuses with fascia of the posterior belly of digastric muscle into a strong band called stylomandibular ligament.

Attaches inferiorly to the lower border of the mandible, investing the entire masseter muscle except at its deep upper portion. Here, there is a communication with the facial planes associated with the insertion of the temporalis muscle.

**The Parapharyngeal space**

Understanding the anatomy of the parapharyngeal space is crucial for removal of the deep lobe parotid tumour. If the parapharyngeal space is assumed to be pyramidal in shape, the base being the base of skull, the apex at the greater cornu of the hyoid bone,

the medial border is the muscular pharyngeal wall and the lateral border is the medial pterygoid muscle and the mandibular ramus.

The normal contents are,

1. The internal carotid artery
2. The ascending pharyngeal artery
3. The pharyngeal venous plexus.

The deep lobe parotid tumours will extend into the space by growing in between the stylomandibular ligament and the bony mandible taking the pathway of least resistance.

The Parapharyngeal space is divided into pre and post styloid compartments by an imaginary line, which is best appreciated on CT scan drawn from the styloid process on to the medial pterygoid plate. Pre operative delineation of the growth characteristics and extension of the tumour within the parapharyngeal space by axial imaging may provide clues to the pathology of the tumour.

## Theory for the Histogenesis

**1. Multi cellular theory:**

The genesis of neoplasm from their adult differentiated counterparts of the salivary gland unit (eg) Acinic cell carcinomas would originate from acinar cells, oncocytic tumour from striated duct cells, squamous cell carcinomas and mucoepidermoid carcinomas from excretory duct cells and all other adenomas and adenocarcinoma from intercalated duct cells.

**2. Bi-cellular theory of origin:**

Neoplasms develop from the two undifferentiated reserve cells called excretory duct reserve cell and the intercalated duct reserve cell. This theory is more plausible because it does not require dedifferentiation duct of cells.

**Benign salivary gland neoplasms:**

Benign salivary gland neoplasms represent a diverse group of neoplasms with varied clinical behaviours.

**ETIOLOGICAL FACTORS**

1. Radiation
2. Smoking – Warthins tumour.
3. EB virus – increased risk
4. Silica dust

5. Genetic factors (most translocations)
6. Other factors

Most common structural re-arrangement in salivary gland malignancies involving Chromosomes:

(a) Allelic loss of chromosomal arm 12q in pleomorphic adenoma.

(b) Translocation break point at 12q and 15 in pleomorphic adenoma.

**WHO – Histological classification of salivary gland tumours**

| **WHO Class** | **Tumour** | |
|---|---|---|
| **1** | **Adenoma** | |
| | 1.1 | Pleomorphic adenoma |
| | 1.2 | Myoepithelioma |
| | 1.3 | Basal cell adenoma |
| | 1.4 | Warthin's tumour (adenolymphoma) |
| | 1.5 | Oncocytoma (oncocytic adenoma) |
| | 1.6 | Canalicular adenoma |
| | 1.7 | Sebaceous adenoma |
| | 1.8 | Ductal papilloma |
| | 1.8.1 | Inverted ductal papilloma |
| | 1.8.2 | Intraductal papilloma |

| | | |
|---|---|---|
| | 1.8.3 | Sialadenoma papilliferum |
| | 1.9 | Cystadenoma |
| | 1.9.1 | Papillary cystadenoma |
| | 1.9.2 | Mucinous cystadenoma |
| **2** | **Carcinoma** | |
| | 2.1 | Acinar cell carcinoma |
| | 2.2 | Mucoepidermoid carcinoma |
| | 2.3 | Adenoid cystic carcinoma |
| | 2.4 | Polymorphous low-grade adenocarcinoma<br>(Terminal duct adenocarcinoma) |
| | 2.5 | Epithelial-myoepithelial carcinoma |
| | 2.6 | Basal cell adenocarcinoma |
| | 2.7 | Sebaceous carcinoma |
| | 2.8 | Papillary cystadenocarcinoma |
| | 2.9 | Mucinous adenocarcinoma |
| | 2.10 | Oncocytic carcinoma |
| | 2.11 | Salivary duct carcinoma |
| | 2.12 | Adenocarcinoma |
| | 2.13 | Malignant myoepithelioma (myoepithelial carcinoma) |
| | 2.14 | Carcinoma in pleomorphic adenoma (malignant Mixed tumour) |
| | 2.15 | Squamous cell carcinoma |

2.16 Small cell carcinoma

2.17 Undifferentiated carcinoma

2.18 Other carcinomas

**3 Non-epithelial tumours**

3.1 Angiomas

3.2 Lipomas

3.3 Neural tumours

3.4 Other benign mesenchymal tumours

3.5 Sarcomas

**4 Malignant lymphomas**

**5 Secondary tumours**

**6 Unclassified tumours**

**7 Tumour-like lesions**

## History and physical examination

A careful history and physical examination is the first step to differentiate between benign and malignant tumours of the parotid gland. Benign neoplasms are painless, slow growing. Benign tumours are freely mobile and have no associated nerve paralysis or overlying ulceration (Fig.5&6).

A facial nerve paralysis is generally associated with a malignant tumour. Features

of malignancy include facial nerve paralysis (or) paresis, pain, and fixation of the mass to overlying skin or underlying structures and associated cervical adenopathy.

Metastatic disease to parotid from skin malignancy should be ruled out by careful examination of skin of scalp and face. The oropharynx should be examined carefully to rule out deep lobe involvement.

A mass in deep lobe of parotid gland or a tumour originating from minor salivary gland in the parapharyngeal space may displace soft palate or tonsils medially.

**Fine Needle Aspiration cytology**

Fine needle aspiration cytology examination in salivary gland tumour was first reported by ENEROTH in 1976. The accuracy of the fine needle aspiration cytology in the diagnosis of salivary tumour is well established. The most common diagnostic error is inadequate sampling. FNAC is simple, safe to perform and inexpensive. FNAC also allows pre-operative counseling of patients. The reported sensitivity ranges from 85% to 99% and specificity 100%. Thus FNAC is helpful in treatment planning for patients presenting with salivary gland masses.

CT guided FNAC is most valuable for deep lobe tumours which are likely to be malignant and difficult to do biopsy by other methods.

**Incisional biopsy**

Open biopsy is avoided, because of nerve injury, tumour spillage and associated infection and tumour recurrence except in,

1. Doubtful malignancy (in FNAC report) and by clinical examination.
2. Minor salivary gland tumours. As they are surface tumours, there is no risk of implantation.
3. Diffuse enlargement of salivary gland.

**Ultra sonogram**

Simple, noninvasive, inexpensive. It can be used to differentiate between solid and cystic masses in salivary gland. Its use is limited by its ability to visualize only relatively superficial masses.

**Imaging**

The routine use of imaging in patients with small well-defined masses of the superficial lobe of the parotid gland is not warranted. CT and MRI give a better understanding of the location and extent of the tumours, relation to neurovascular structures, perineural spread of malignancy, skull base invasion and intracranial extension (Fig.7). Nuclear imaging using technetium 99m pertechnate is helpful only in patients with oncocytic and Warthin's tumours. Bony destruction of the mandible or skull base is best visualized on CT. Bone-marrow involvement it is better demonstrated

on MRI. Both may adequately evaluate the neck for the metastatic adenopathy. MRI is superior to CT in demonstrating the internal architecture of salivary gland tumours. Perineural spread may be detected earlier in MRI

**Indication for CT scan**

1. Mass confined to deep lobe of the parotid gland.
2. Tumours with involvement of both the deep and superficial lobes of the parotid gland (dumb bell tumours).
3. Parotid tumour presenting with facial nerve weakness, other neural deficit or indication of malignancy
4. Congenital parotid masses
5. Recurrent disease

## Benign tumours

**Pleomorphic adenoma**

Pleomorphic adenomas (benign mixed tumour) are the most common tumour. They contain both epithelial and connective tissue elements. The current theory is that they originate from intercalated and myoepithelial cells. They are painless, slow growing. About 90% of the tumours originate superficial to the plane of the facial nerve. The remaining 10% originate in the deep lobe. Pleomorphic adenoma when originate in the minor salivary glands, mostly occur in the hard and soft palate. The second most common place of origin is the upper lip. Unlike most mixed tumours those in the palate and lip frequently lack a capsule. Although the parapharyngeal space contains cranial nerves IX to XII, these structures are rarely involved.

Microscopically the tumour extends through the capsule, so that the tumour should never be enucleated, because of the pseudopodia like extensions of the tumour (Fig.8).

**Warthin's tumour or Papillary cystadenomas or Adenolymphoma**.

This amounts for 10-15% of all parotid tumours. This is the second most common type of benign salivary tumour. It has a striking male preponderance. Bilateral in 10%, commonly has unilateral multifocal involvement.

Embryologically, the parotid gland is the first salivary gland to develop and the last to become encapsulated. One hypothesis concerning Warthin's tumours is the incorporation of salivary ducts and lymphatic tissue during their late encapsulation. Mitochondrial rich oncocytes are found in Warthin's tumour and oncocytomas, the only other benign tumour with frequent bilateral presentation. Oncocytes selectively incorporate Tc 99M and appear as hot spots on a radio nucleotide scan. Most other neoplasms either have normal uptake or appear as cold nodules.

**Oncocytomas:**

They account for less than 1% of salivary tumours. They usually are benign and originate from oncocytes. A true oncocytoma contains no lymphoid tissue. They have abundant mitochondria. Although histologically benign, they have a destructive potential.

**Hemangiomas and vascular malformations**

Hemangiomas are usually seen in infants. A rapid growth phase occurs between 1-6 months followed by gradual involution over 1 to 12 years. They are mostly seen in females. Treatment consists of steroids administered at 2 to 4 mg/kg per day. If the hemangioma is responsive to steroids, the result is usually immediate and often dramatic. Unfortunately the response rate is 40 – 60%. In such patients, interferon may be useful, but it is reserved for life threatening situations, because of its toxicity and the necessity to continue the drug for prolonged periods. Surgical excision or various types of laser treatment may be performed for select circumstances.

# Malignant tumours

## Mucoepidermoid carcinoma

Mucoepidermoid carcinoma is the most common malignant tumour of parotid gland and the second most common neoplasms of parotid. Mucoepidermoid carcinomas contribute 35% of all salivary gland tumours, of which 80 to 90% occur in the parotid gland. It contains two major elements, mucin producing cells and epithelial cells of the epidermoid variety (Fig.9).

Mucoepidermoid carcinomas are classified into well-differentiated, intermediate and poorly differentiated tumours.

CLODE and others have reported a cumulative survival rate of 47% for patients with low-grade mucoepidermoid carcinoma and 30% for those with high-grade mucoepidemoid carcinoma.

## Adenoid cystic carcinoma

Adenoid cystic carcinoma contributes 10% of all salivary neoplasms. It is the second most common malignant tumour of the salivary glands. Adenoid cystic carcinoma is the most common malignant tumour found in the submandibular, sublingual and minor salivary glands. It is slightly more common in females.

Adenoid cystic carcinoma is described in three histological sub-types based on tumour architecture: Cribriform, tubular and solid. The cribriform pattern has the classic "Swiss cheese appearance", wherein the cells are arranged in nests separated by round or oval spaces. They are merely extracellular spaces lined by replicated basement membrane material (Fig.10).

The tubular variety has the best prognosis, the solid variety has the worst and the cribriform pattern has an intermediate prognosis. Adenoid cystic carcinoma usually exhibits a protracted course characterized by an indolent growth pattern and a relentless tendency for local and perineural invasion. This neurotropic tendency has been reported to occur in 20 – 80% of patients.

The pathogenesis of perineural involvement is poorly understood and was thought to be due to spread of tumour through perineural lymphatics. The most commonly involved nerves are maxillary (V.2) and the terminal branches of trigeminal nerve and facial nerve.

Perineural involvement is more frequent in patients with advanced and recurrent tumours and in those with high-grade tumours.

**Acinic Cell Carcinoma**

Acinic cell carcinoma represents 3% of all salivary gland tumours. The majority occurs in parotid gland (80% to 90%) where in they constitute 15% of its malignant neoplasms. Two thirds occur in females, which makes acinic cell carcinoma the second most common neoplasm after Warthins tumours, to exhibit bilateral presentation.

Acinic cell carcinoma is generally regarded as a low-grade malignancy. Compared with Adenoid cystic carcinoma or mucoepidermoid carcinoma, acinic cell carcinoma has a more favourable prognosis.

**Malignant mixed tumour**

As the name implies, the malignant mixed tumour represents a malignancy with epithelial and mesenchymal elements. They constitute 3% to 12% of salivary gland cancer and three-fourths are in parotid gland. When they arise from pre-existing pleomorphic adenoma, they are called **carcinoma ex-pleomorphic adenoma**. By contrast de novo malignant mixed tumour is a true carcinosarcoma with malignant features of the epithelial and mesenchymal elements that are present in the primary tumour and its metastasis. This rare true malignant mixed tumour is highly lethal.

**Lymphomas**:

Lymphomas of the salivary glands may originate from intra glandular lymphomas (nodal) or from the lymphoid tissue dispersed within the salivary gland parenchyma

(extra nodal), which is considered as part of MALT system. Lymphoma can be primary or part of disseminated lymphoma.

Approximately 5% of all extra nodal lymphomas affect the salivary gland and more than 90% of salivary gland lymphomas occur in the parotid gland. Most of salivary gland lymphomas are the NHL variety (85%).

**Secondary (metastatic tumours)**

Hematogenous metastases to salivary gland from infraclavicular primaries are rare. These are mainly from lungs, kidneys and breast. The majority of metastasis to salivary glands is caused by lymphatic spread from cutaneous malignancy of the head and neck. Metastatic tumour accounts for less than 10% of malignant parotid tumours. Of these, 40% are squamous cell carcinomas and 40% are melanomas.

**TNM STAGING**

TX - Primary tumour cannot be assessed.

T0 - No evidence of primary tumour.

T1 - Tumour 2cm (or) less in greatest dimension without extraparenchymal Extension.

T2 - Tumour 2 to 4cm without extra parenchyma extension.

T3 - Tumour > 4cm or with extra parenchymal extension.

T4 (a)- Tumour invades skin, mandible, ear canal, facial nerve or any of these Structures.

T4 (b) - Tumour invades skull base (or) pterygoid plates (or) encases carotid artery.

**N (Regional nodes):**

Nx - Regional lymph nodes cannot be assessed.

N0 - No regional lymph node metastasis.

N1 - Metastasis in a single ipsilateral lymph node < 3cm.

N2a - Metastasis in a single ipsilateral lymph node 3 to 6cm.

N2b - Metastasis in a multiple ipsilateral lymph node < 6cm.

N2c - Metastasis in bilateral (or) contra lateral lymph node < 6cm.

N3 - Metastasis in a lymph node > 6cm.

**Distant Metastasis (M)**

Mx - Distant metastasis cannot be assessed.

M0 - No distant metastasis.

M1 - Distant metastasis.

**Staging**

| | | |
|---|---|---|
| I | - | T1N0M0 |
| | | T2N0M0 |
| II | - | T3N0M0 |
| III | - | T1N1M0 |
| | | T2N1M0 |
| IV | - | T4N0M0 |
| | | T3N1M0 |
| | | ANYT, N2M0 |
| | | ANYT, N3M0 |
| | | ANYT, ANYN, M1 |

## Treatment

The treatment of benign tumours has passed through several phases during the last 30 years. Surgery is the treatment of choice for all.

**Benign tumours**

Small, localized tumours of the superficial lobe are treated adequately by superficial conservative Parotidectomy (Fig.11,12$13). Large tumours and tumours involving deep lobe and dump bell tumours require total parotidectomy with preservation of facial nerve.

**Malignant tumours**

Total conservative Parotidectomy with postoperative radiotherapy is the treatment. If the facial nerve is directly involved by the tumour or if the course of the nerve interferes with the complete removal of the gland then the nerve is sacrificed and primary nerve graft with greater auricular nerve is done, if this fails then later attempts to rehabilitate the facial nerve is done with cross-face anastomosis technique or faciohypoglossal anastomosis.

Rarely, radical excision of parotid gland with sacrifice of the facial nerve and attached muscles, mandible and temporal bone can be done. If cervical nodes are involved then modified radical neck dissection is indicated. If local recurrence is anticipated because of histologically positive margin or if tumor is high grade or

advanced stage of the tumour, then postoperative radiotherapy is used.

**Intra operative variation**

From the onset, the anesthesiologist should avoid the use of paralytics or muscle relaxants. This is done so that either EMG monitoring of facial musculature or nerve localization by electro stimulation is permitted during the operation. The use of intra operative facial nerve monitoring through the use of nerve integrity monitor can be done. It was proposed informally as early as 1991.

Hemostasis is improved with use of bipolar cautery in areas adjacent to the facial nerve to help reduce thermal injury to nerve. Recently the Shaw scalpel has been used in parotidectomy to reduce blood loss. The Shaw scalpel is a thermally activated scalpel, popularly known as Bipolar scissors with saline irrigation for faster dissection in a relatively bloodless field. Harmonic scalpel can be used for the same purposes. The generator of the unit transmits ultrasonic energy at the tip of the scalpel. Simultaneous sharp dissection and hemostasis are obtained by the ultrasonic energy, which works by denaturing the protein molecules. Because there is no heat production, it is quite safe to direct tissues with the harmonic scalpel in close proximity to nerves or other vital structures.

**Complications of surgery**

EARLY

1. Facial nerve neuropraxia
2. Facial nerve injury
3. Hemorrhage
4. Sialocele
5. Flap necrosis
6. Salivary fistula
7. Infection
8. Otitis externa

LATE

Frey's syndrome

**FREY'S SYNDROME**

It is the occurrence of redness and sweating in the area of distribution of Auriculotemporal nerve, when eating, which is seen in patients after parotidectomy. The true incidence is unknown but is estimated to be between 35% - 60%. This is caused by regeneration of secretory fibers of parotid gland contained in auriculotemporal nerve, to sweat glands in its area of distribution. Thus the sweat glands respond to the nerve impulses that would reflexly provoke parotid secretion. The diagnosis of Frey's syndrome depends on patient's symptoms. An objective method of confirming the diagnosis is the miner's starch and iodine test.

Most patients with Frey's symptoms require nothing more than education and Re-assurance. The application of topical glycopyrrolate (or) mild anti-perspirant is a simple and effective approach. It can be also relieved by section of the glossopharyngeal nerve, which supplies the pre-ganglionic fibers for the parotid gland.

**Radiotherapy**

The indication and technique of radiotherapy to be used as well as the response of salivary gland tumours to treatment depends upon the location of the primary lesion as well as extent and histological make out.

It is used as

Curative – as an adjunct to surgery.

Palliative –

- Inoperable tumours.
- For recurrent lesions.
- Symptomatic treatment of distant metastasis.

The indications for adjuvant radiotherapy to tumour bed are

1. For all cases of adenoid cystic carcinoma.
2. High-grade tumours.
3. Positive margins.
4. Nodal metastasis.
5. Advanced stage (stage III & IV)

Since skip metastasis are possible, entire neck is to be irradiated. It is used in doses of 6000-6500 rads in fractionated doses for 6-7 weeks.

**Complications of Radiotherapy**

1. Ageusia
2. Diminished salivation
3. Mucosal necrosis
4. Trismus
5. Caries tooth
6. Osteonecrosis

**Chemotherapy**

It is used as a palliative treatment for recurrent or metastatic salivary gland

malignant neoplasm. Single drug therapy is commonly used with one of the following drugs – Doxorubicin, 5FU, Methotrexate, cyclophosphamide or cisplatin.

Combination chemotherapy can also be used. Regimens include:

CAP (Cyclophosphamide, Adriamycin, Cisplatin)

FACP (5FU, Adriamycin, Cyclophosphamide and Cisplatin)

## Management of facial nerve paralysis.

1. Neurotherapy
2. Inter position of nerve graft.
3. Cross-over re-innervation procedures.

- Hypoglossal
- Cross facial
- Ansa hypoglossi

**Regional muscle transfer**

- Temporalis
- Digastric
- Massetric

**Micro-neuro vascular free flap**

- Gracillis
- Rectus abdominis
- Pectoralis minor

- Latissimus dorsi
- Serratus anterior
- Extensor digitorum brevis.

**Static re-animation and cosmetic procedure**

1. Eyelid procedures
    - Spring
    - Gold weights
    -Lower lid tightening
2. Brow and forehead lift.
3. Correction of mid facial deformity
    - Fascia lata slings
    - Alloplastic sheets.
    - Malar augmentation.
4. Face lift operation.
5. Lower lip wedge resection.
6. Botulinium toxin.

## AIMS AND OBJECTIVES

- To analyse the epidemiology of parotid tumours

- To analyse the various presentation of parotid tumours.
- To find out risk factors.
- To discuss the role of FNAC in the management of parotid tumours.
- To discuss various treatment modalities of parotid tumours.
- To analyse the frequency of facial nerve involvement by the tumour.
- To analyse the facial nerve injuries after surgery.
- To analyse the post operative complications

## MATERIALS AND METHODS

Patient admitted between May 2006 to November 2008 (3 years). They were randomly selected (total 42) and assessed by FNAC pre operatively and proven by Histopathological examination post operatively.

In all cases a thorough clinical history and physical examination were carried out age, sex and risk factors if any were noted down.

Basic investigations were done for all patients'. CT scan was done in a few cases.

A master chart was created and the data entered and analysed. Based on the investigations, cases were divided into benign and malignant neoplasms and were treated appropriately.

Follow up was done in our outpatient facility. Patients were referred for postoperative radiotherapy and readmitted if any complications arose.

## OBSERVATION AND DISCUSSION

This study included Parotid tumours in THANJAVUR MEDICAL COLLEGE from May 2006 to November 2008.

Patients were analysed both retrospectively and prospectively. All tumours were histological proved.

A total of 42 cases of Parotid tumours were randomly studied. Of these 29 Proved to be benign tumours. 13 proved to be malignant. The benign and malignant neoplasms are considered separately and discussed with the help of available literature.

| | Our study | Other studies<br>Eweson & Cawsons et al |
|---|---|---|
| Malignant (%) | 13 (31%) | 15% |
| Benign (%) | 29 (69 %) | 72% |

## DISCUSSION

### Benign tumours

Each were categorized into sex, age, presenting symptoms, type of Pathology, type of lobe involved, treatment given and post operative complications.

**1. Sex:**

In this study benign parotid tumours 9 patients were male, 20 patients were female.

| **Sex** | **No. Of patients** | **%** |
|---|---|---|
| Male | 9 | 31% |
| Female | 20 | 69% |

In this study benign tumours were more among females. Male female ratio was 1:2.2

**2. Age:**

In this study commonest age group affected was 31-40 years, the oldest patient was 70-year-old female and youngest patient was 4 years female.

| Age | No. Of patients | % |
|---|---|---|
| 1-10 | 1 | 3.44% |
| 11-20 | 3 | 10.34% |
| 21-30 | 7 | 24.13% |
| 31-40 | 10 | 34.48% |
| 41-50 | 2 | 6.89% |
| >50 | 6 | 20.68% |

**3. Lobes involved**

In our study 28 patients were superficial lobe only involved. 1 patient was both Superficial and deep lobe involved.

| Lobes involved | No. Of patients | % |
|---|---|---|
| Superficial lobe | 28 | 96.55 |
| Deep & superficial lobe | 1 | 3.45 |

**4. Risk factors:**

The commonest risk factors are smoking tobacco chewing and betalnut chewing.

| Habits | No. Of patients | % |
|---|---|---|
| Smoking | 4 | 13.80 |
| Tobacco chewing | 3 | 10.34 |
| Betalnut chewing | 4 | 13.80 |
| Alcohol and smoking | 3 | 10.34 |
| Tobacco chewing & smoking | 2 | 6.90 |

**5. Clinical presentation:**

In this study the benign tumours commonly presented as a painless swelling.

| Symptoms | No. Of patients | % |
|---|---|---|
| Painless swelling | 21 | 72.41 |
| Swelling & pain | 7 | 24.13 |
| Recurrent swelling | 1 | 3.44 |

**6. Pathology:**

In this study, most common benign of parotid gland tumours were pleomorphic Adenoma.

One patient reported as hamartoma

| Pathology | No. Of patients | % |
|---|---|---|
| Pleomorphic adenoma | 24 | 82.75 |
| Monomorphic adenoma | 4 | 13.80 |
| Hamartoma | 1 | 3.44 |

**7. Investigation:**

In this study the following investigations were done for all patients who include Blood urea, serum creatinine, blood sugar, x-ray chest, and FNAC, CT scan neck was done in few selected cases.

In this study, FNAC was easy investigation with virtually no complication. It is a non-expensive, reliable and repeatable simple investigation. In our study FNAC

findings confirmed by postoperative histopathological examination.

**The sensitivity and specificity of FNAC**

FNAC was the key investigation done in all cases. It provided accurate diagnosis in most parotid tumours and contributed to conservative management.

$$\text{Sensitivity} = \frac{\text{True positive}}{\text{True positive + false negative}}$$

.

$$\text{Specificity} = \frac{\text{True negative}}{\text{True negative + false positive}}$$

| No. Of patients | True (+) | False (+) | Sensitivity | Specificity |
|---|---|---|---|---|
| 29 | 28 | 1 | 96.5 | 100% |

In our study, the sensitivity of FNAC in benign parotid tumour was 96.5% Specificity 100%.

**8. Types of surgery:**

In this study all cases of benign parotid neoplasms of superficial lobe were treated by superficial conservative Parotidectomy. One patient underwent total conservative Parotidectomy due to involvement of both lobes.

| Surgery done | No. Of patients | % |
|---|---|---|
| Superficial conservative parotidectomy | 28 | 96.55 |
| Total conservative parotidectomy | 1 | 3.45 |

For one patient pre operative FNAC was pleomorphic adenoma and CT was done because clinically deep lobe was involved. The patient underwent total conservative Parotidectomy.

**9. Post operative complications:**

The postoperative period was uneventful in most of the cases. Uneventful Patients were discharged on the 7$^{th}$ postoperative day after suture removal.

Neuropraxia developed in 3 cases i.e. 10.3%, managed conservatively.

Full recovery was there.

Wound infection developed in 5 cases, were treated with appropriate antibiotics.

| S.No | Post operative complication | No. Of patients | % |
|---|---|---|---|
| 1. | Wound infection | 4 | 13.80 |
| 2. | Facial nerve palsy | 2 | 6.90 |
| 3. | Wound infection with facial palsy | 1 | 3.45 |
| 4. | Uneventful | 22 | 75.86 |

# MALIGNANT TUMOURS

Out of 42 patients, 13 patients were affected with malignant parotid gland tumours.

**1. Sex:**

In our study, 8 patient were female 5 patient were male.

| Sex | No. Of patients | Percentage |
|---|---|---|
| Male | 5 | 38.46 |
| Female | 8 | 61.54 |

**2. Age:**

In our study commonest age group affected was $5^{th}$ $6^{th}$ $7^{th}$ decades.
The youngest patient was 12 years old male.

| Age group | No. Of patients |
|---|---|
| 11-20 | 2 |
| 21-30 | 2 |
| 31-40 | 1 |
| 41-50 | 1 |
| 51-60 | 4 |
| 61-70 | 3 |

**3. Lobe involved:**

Most common lobe to be involved in malignant parotid tumour was superficial Lobe.

| Lobe involvement | No. Of patients | Percentage |
|---|---|---|
| Superficial lobe | 10 | 76.92 |
| Superficial and Deep lobe | 3 | 23.08 |

**4. Clinical Presentation**

In this study, 4 patients presented with painful swelling, 2 patients presented with Facial nerve palsy

| Symptoms | No. Of patients | Percentage |
|---|---|---|
| Painless swelling | 7 | 53.85 |
| Swelling with pain | 4 | 30.76 |
| Swelling with facial nerve palsy | 2 | 15.38 |

**5. Pathology:**

In our study, 13 patients were malignant tumours; of these Mucoepidermoid carcinoma was commonest pathological type.

| S.No | Pathological type | No. Of patients | Percentage |
|---|---|---|---|
| | Mucoepidermoid carcinoma | 11 | 84.61 |
| 1. | Acinic cell carcinoma | 1 | 7.70 |
| 2. | Squamous cell carcinoma | 1 | 7.70 |

In 11 patients of mucoepidermoid carcinoma,

Low grade -- 7
Intermediate – 2
High grade -- 2

**6. Sensitivity and specificity:**

| No. Of patient | True positive | False negative | Sensitivity |
|---|---|---|---|
| 13 | 10 | 3 | 76.92% |

In our study sensitivity of the malignant parotid tumour was 77%, specificity 100%

**7. Staging of the disease:**

| Stage | No. Of patients |
|---|---|
| I | 2 |
| II | 6 |
| III | 1 |
| IV | 4 |

Two patients initially presented with facial nerve palsy, one patient with skin Involvement (ulceration).

**8. Treatment:**

In our study, out of 13 patients, 11 patients underwent conservative Parotidectomy, 1 patient underwent total radical parotidectomy, five patients underwent Adjuvant radiotherapy because of high-grade malignancy.

Two patients (high grade malignancy) did not receive Adjuvant radiotherapy because they lost follow up. One patient received radiotherapy alone due to inoperability.

| Mode of treatment | No. Of patients | Percentage |
|---|---|---|
| Superficial conservative Parotidectomy | 10 | 76.93 |

| | | |
|---|---|---|
| Total conservative Parotidectomy | 1 | 7.70 |
| Total radical Parotidectomy | 1 | 7.70 |
| Radiotherapy only | 1 | 7.70 |
| Adjuvant Radiotherapy | 5 | 38.46 |

**9. Post operative complications:**

One patient developed neuropraxia, managed conservatively. Resolved Spontaneously.

Three patients developed nerve palsy; they were treated by lateral tarsorrhaphy and Physiotherapy. Since patient was not willing for flap cover.

Two patients developed wound infection. They were treated with antibiotics.

| **Complications** | **No. Of patients** | **Percentage** |
|---|---|---|
| Wound infection | 1 | 7.70 |
| Facial nerve palsy | 3 | 23.07 |
| Wound infection with facial nerve palsy | 1 | 7.70 |
| Uneventful | 8 | 61.5 |

# CONCLUSION

The clinicopathological study on parotid tumours among the patients admitted at Thanjavur medical college hospital showed following facts

- The age incidence for benign tumour was commonly between 3rd and 4th decade. The malignant tumour commonly occurred in 6th and 7th Decades.
- The sex incidence in both benign and malignant tumours was predominantly in females.
- In parotid gland tumours 69 % were benign, 31 % were malignant.
- Pleomorphic adenoma was the commonest benign tumour.
- Mucoepidermoid carcinoma was the commonest malignant tumour.
- In benign tumours, sensitivity and specificity of FNAC were 96.5% and 100% respectively.
- In malignant tumours, sensitivity and specificity of FNAC were 77% and 100% respectively.
- There were no significant risk factors associated with parotid tumours.
- Commonest clinical presentation in benign and malignant tumours was Painless swelling. Swelling associated with facial nerve palsy noted in 15% of Malignant Parotid tumours.
- Commonly performed surgery in benign and malignant tumours was Superficial

Conservative Parotidectomy.

- Adjuvant radiotherapy was given in high-grade tumours and acinic cell Carcinoma.
- In benign tumours, facial nerve palsy occurred in 10% of patients and post operatively this recovered within 6 weeks; wound infection for 14% of Patients and 76% of patients were uneventful.
- In malignant tumours facial nerve palsy occurred post operatively in 30%, treated with lateral tarsorrhaphy with physiotherapy.

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