



A Clinico - Pathological Correlation & Study of Cervical Lymphnodes

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ABSTRACT

Introduction

Neck swellings are common in clinical practice and is of various types - congenital, acquired, inflammatory, neoplastic and miscellaneous. The workup of neck mass is different in children and in adults, due to differing etiologies. Neck masses are common in children and most often are due to inflammatory processes or congenital abnormalities. Only 2-15% are malignant. In adults, neck masses more often represents malignancy. Persistent masses larger than 2cm represents cancer in 80% of the cases.

The study is conducted in Sri Krishna Medical College Muzaffarpur, during the study period of September 2020 – September 2022 in which all the patients with swelling in neck region were included, excluding thyroid and its associated swellings as thyroid and its associated swellings itself are vast topics, needs separate study and so these conditions

are excluded from the present study.

The commonest cause of neck swelling is tubercular cervical lymphadenitis. It is known that 1.5% of India’s population is affected with tuberculosis. Other commonly encountered swellings are secondaries in the neck, acute lymphadenitis, chronic non specific lymphadenitis and lymphomas. Swellings like cystic hygroma, branchial cyst are seen less frequently.

In this present dissertation, it has been tried to review the literature and to know the disease occurrence and its distribution and to find out the possible etiological factors of the neck swellings with fine needle aspiration cytology and histopathological correlation and also to summarize the results of different forms of treatment for neck swellings especially tuberculosis.

AIMS AND OBJECTIVES

1. To study the various etiological factors of neck

swellings.

2. To study the clinical presentation of neck swellings.
3. To correlate the clinical diagnosis with FNAC and Histopathological report.

REVIEW OF LITERATURE

Historical Perspective

Earliest description of tuberculosis date back to 500 B. C. Hippocrates was aware of the condition and his account describes the anatomical pattern of lymph nodes and also origin of “scrofula”. Laennec (1781-1820) a French clinician was the first to discover and describe an “anatomic tubercle”.

In 1882 Robert Koch discovered tubercle bacilli. Stains for acid fast bacilli were introduced by Ziehl and Neilsen (1882).

Tuberculosis “captain of all men of death” as referred by John Bunyan in the 18th century, is still the biggest health challenge of the world. It is known that 1.5% of India’s population is affected with tuberculosis.

Cervical tuberculos lymphadenopathy or “scrofula” which was treated in medieval times in England by the “kings touch” and issuance of “gold coins”, is still the most common cause of persistent cervical lymph node enlargement in the developing countries.

The concepts of staging of lymphomas developed during the 20th century and were formalized in the United states by the establishment of the American Joint Commision on cancer in 1959. The working formulation adopted some aspects of the Rappaport, Lukes-Collins and Kiel Classification and was the primary system used in publication in much of the 1980s and 1990s. In the 1990s a group of hematopathologists proposed the revised European-American Lymphoma classification.

In 1836 Wilhelm Fredrick Von Ludwig described a disease process of neck infection which was almost always fatal.

The first branchial arch anomalies are classified by work as type I and II in 1977.

The terms lymph and lymphatics were first made in 1651 by Thomas Bartholin (1616-1680) and Finger and Rudbeck (1660-1702) who were anatomists of Sweden.

The term lymph Node was adopted in nomica anatomica at Paris only in 1955. The tech nique of lymphography by Kinmonth in 1955 proved the way to understand the connections of the lymphatic system.

Lushka first described the carotid body tumour in 1862. Scudder in 1903 successfully removed carotid body tumour without nerve injury and sparing the carotid vessels. Adenoid cystic carcinoma is an uncommon tumour of the salivary gland first described as “Cylindroma” by Billroth in 1859.

FNAC examination is simple and relatively painless and less cumbersome procedure introduced first by Martin, can be adopted instead of biopsy.

First description of radical neck dissection by George Crile in 1906, this procedure remained the cornerstone of surgical management of metastatic neck nodes for most of the past century.

EMBRYOLOGY1

Neck is formed mainly by the pharyngeal arches and the part of the region between the stomatodaeum and pericardium.

The pharyngeal arches are six in number, they are mesodermal thickenings, namely First- Mandibular arch, 2nd- hyoid arch, 3rd, 4th, 6th arches have got no special names and the 5th arch disappears.

The first arch gives rise to sphenomandibular ligament, mylohyoid and anterior belly of digastric. The second arch gives rise to stylohyoid ligament, part of the hyoid bone, posterior belly of digastric, platysma. The third arch gives rise to part of hyoid bone, stylopharyngeus. The fourth and sixth arches gives rise to muscles of pharynx, soft palate and larynx.

The ectodermal clefts similar to pharyngeal arches are

six in number. The second arch overgrows the third, fourth, sixth arches and hence the neck becomes a smooth surface. If the lower edge of the second arch does not fuse lower down, anomalies of the branchial cleft results. The third ectodermal pouch gives rise to the thymus and the inferior parathyroid and thyroid gland. The fourth pouch gives rise to superior parathyroid gland.

Fifth pouch forms the ultimobranchial body.

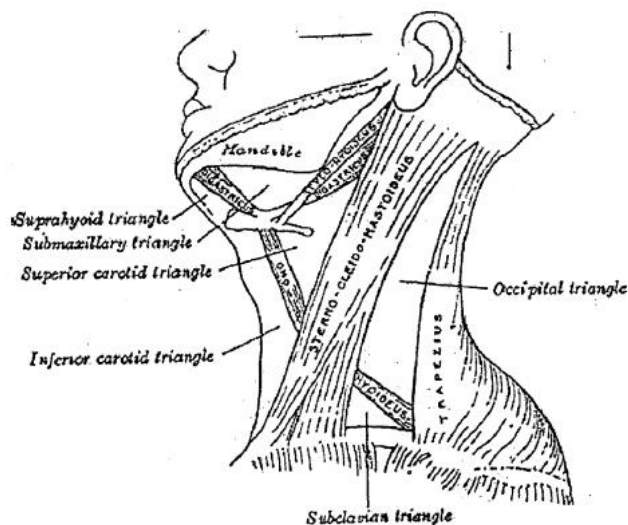


Fig. 1: Triangles of the neck

SURGICAL ANATOMY OF THE NECK

The prominent landmarks in the neck are hyoid bone, the thyroid cartilage, trachea, sternocleidomastoid muscles.

The Sternocleidomastoid muscle divide the neck on each side into the anterior and posterior triangles.

Anterior triangle is further divided into Submental, Submandibular, Carotid and Muscular triangles.

Boundaries of Different Triangles

Submental Triangle

Inferoposteriorly – by hyoid bone

Laterally on either side – by anterior belly of digastric muscle

Apex is formed by anterior median mandible.

Contents: Submental lymph nodes and small submental veins that joins to form the anterior jugular vein.

Submandibular or Digastric Triangle

Posteroinferiorly – Posterior belly of digastric and stylohyoid muscle

Superiorly (base) - base of the mandible and a line joining the angle of the mandible to the mastoid process.

Contents of the Triangle

Submandibular salivary gland and lymph nodes, facial

vein and artery. Deep structures are styloglossus muscle, stylopharyngeus muscle and hypoglossal, glossopharyngeal and pharyngeal branch of vagus nerve.

Carotid Triangle

Superiorly – Posterior belly of digastric muscle and stylohyoid muscle Antero inferiorly – Superior belly of omohyoid.

Posteriorly – Anterior border of sternomastoid muscle.

Contents: Common carotid, internal carotid, external carotid arteries and veins. Nerves – Vagus, hypoglossal, laryngeal.

Lymph nodes – deep cervical group.

The Muscular Triangle;

Superiorly – Hyoid bone

Laterally – Superior belly of omohyoid and anterior border of sternomastoid.

Anteriorly – The midline.

Contents – Sternohyoid, sternothyroid, thyrohyoid, omohyoid muscles.

Posterior Triangle

Anteriorly – Posterior border of sternomastoid muscle.

Posteriorly – Anterior border of trapezius.

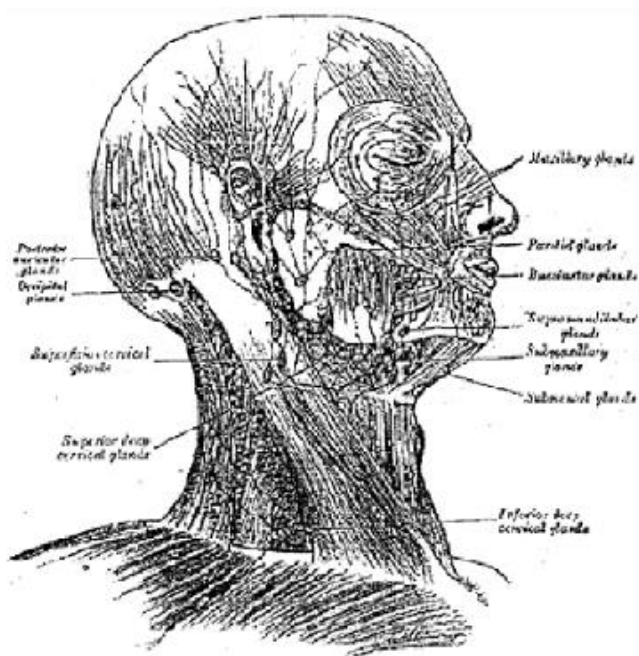
Inferiorly – Middle one third of the clavicle.

Contents – Spinal accessory nerve, cervical plexus, transverse cervical artery and occipital artery and vein

Facial layers of the neck

1. Superficial cervical fascia
2. Deep cervical fascia
 - a) Superficial layer or investing layer of deep cervical fascia (sternocleidomastoid, Strap muscles, trapezius).
 - b) Middle layer or the visceral layer, also known as prevertebral fascia that encloses Thyroid, Trachea, and Oesophagus.

Fig. 2: Lymphatics of Head and Neck



3. Deep layer or prevertebral fascia – Vertebral muscle, Phrenic nerve.

Head and Neck Lymphatics

The lymphatic drainage of the head and neck is divided into three systems.

1. Waldeyer's internal ring: adenoids, tubal and lingual tonsil, the palatine tonsil and aggregates of lymphoid tissue on the posterior pharyngeal wall.
2. Superficial lymph node system (Waldeyer's external ring) includes nodes situated around the skull base known as the occipital postauricular, preauricular, parotid and then buccal or facial nodes. They are in continuity with the superficial nodes in the upper neck situated along the external jugular vein and the anterior jugular veins.
3. Deep system (cervical lymph nodes proper) consists of the junctional nodes, the upper, middle and lower cervical nodal groups which are situated along the internal jugular vein, the spinal accessory group which accompanies the spinal accessory nerve, the nuchal nodes, the visceral nodes in the midline of the neck and nodes in the upper mediastinum.

A more surgically based classification which is simple and reproducible by means of describing the location of the nodes was described by Lindberg in 1972 as follows

Level I (Submental and submandibular groups) Level

Level II (Upper jugular group)

Level III (Middle jugular group)

Level IV (Lower jugular group) Level V (Posterior triangular group)

Level VI (Pretracheal, paratracheal and prelaryngeal group) Level VII (Upper mediastinal group)

In 1997 sub zones were given by Suen and Goepfert as 1A - Submental group of lymph nodes, 1B - Submandibular group of lymph nodes. 2A - Lymph nodes located anterior to spinal accessory nerve. 2B- Lymph nodes located posterior to spinal accessory nerve.

A. Classification of Neck Masses

I. Congenital

Lymphangiomas, dermoids, thyroglossal duct cysts, branchial cysts, branchial fistulae, thymic cysts, haemangiomas.

II. Acquired

Ranulas, Laryngoceles, Pharyngeal pouches.

III. Infective

Bacterial, Viral, Tuberculous

IV. Tumours of the Parapharyngeal Space

- Nodal secondaries: eg lymphoma, nasopharyngeal carcinoma.
- Salivary gland tumours (45%)
- Neurogenic (25%) Schwannoma, neurofibroma, neuroblastoma.
- Paragangliomas (chemodectomas) (15%) (Glomus vagale, Carotid body tumour, Glomus jugulare)
- Miscellaneous : lipoma, liposarcoma.

B. Classification Depending Upon the Location

I. Midline Neck Masses

Thyroglossal duct cysts, dermoid tumours, delphian nodes, thyroid masses, lipomas and sebaceous cysts.

II. Lateral Neck Masses

Enlarged benign or malignant lymphnodes.

Neuromas or neuro fibromas, carotid body tumours, branchial cleft cysts, lipomas, sebaceous cysts, parathyroid cysts or a primary soft tissue tumour.

Diagonostic Approach to Neck Masses

Neck swellings may be solitary, multiple, caused by medical condition and also due to a local or systemic disease.

History

Age of the patient is important – younger patients present with masses which are usually inflammatory or congenital. In patient over 40 years of age and particularly in those with history of cigarette smoking and alcohol ingestion, the primary concern must be malignancy until proven otherwise.

Location of the mass gives an idea as to from what structure it is arising such as thyroid gland mass is located along the anterior border of sternomastoid, the group of lymph nodes involved which can be indicative of the primary location of the tumour.

Symptoms of sore throat, unilateral hearing loss, earache and dysphagia,

Hoarseness should be diligently sought, however no symptom or combination of symptoms is strongly correlated with early head and neck cancer for any subsite except glottis.

Physical Examination

It is the single most important diagnostic test in the evaluation of a cervical mass. The primary tumour can be detected in 50% of patients by clinical examination alone and in a further 10-15% by pan endoscopy of the upper aerodigestive tract.

Skin over face and scalp should be inspected for ulcerations, nodule, pigmented lesions or other suspicious lesions. Nasal airway must be examined for the presence of a mass or epithelial discontinuity.

Mirror examination of the entire oral cavity with lips, nasopharynx, hypopharynx and larynx should be done. Vocal cords must be examined for mobility. Oral cavity should be palpated bimanually. Any

lesion- ulcerative or nodular should be subjected to biopsy.

Palpation of the neck mass to evaluate for the size, shape, surface, consistency and extension of the mass is to be done carefully.

Lymph node examination : site, size, number, consistency, mobility must be checked and also generalized involvement of lymphatic system and hepatosplenomegaly is to be looked for.

Investigations

The complete blood count will provide useful data for the diagnosis of Acute or Chronic Leukemias, EBV, CMV, Mononucleosis, Lymphoma with a Leukemia component as well as pyogenic infections.

Serological studies may demonstrate antibodies specific to components of **HIV, EBV, CMV** and PPD test (purified protein derivative) detects exposure to mycobacterial infection.

Imaging Studies

Plain radiographs of neck and cervical spine may show soft tissue abnormalities. Chest x-ray may reveal pulmonary infiltration, tuberculosis and malignancy.

Computerized tomography and magnetic resonance imaging of the head and neck with contrast media may be obtained for tumour evaluation and occult lymphadenopathy detection.

CT scanning is best at evaluating bony destruction, whereas MRI scan is for soft tissue involvement.

Angiography or Digital Subtraction Vascular Imaging

These may be indicated if a vascular lesion such as carotid tumour is suspected. Angiography may have a beneficial therapeutic role to play by facilitating embolization of the lesion.

USG differentiates the solid and cystic lesion.

Positron Emission Tomography

PET scanning is more sensitive than CT in identifying the primary lesion but is not able to detect unknown primary tumour with more than 50% sensitivity.

FNAC

Fine needle aspiration cytology has been found to be a simple, safe, least traumatizing and an acceptable procedure to the patient, and the results are available within a short time. However, the procedure may fail when the diseased part of the gland is too small or fibrotic and in such cases, excisional biopsy is necessary.

Biopsy

Cytological diagnosis of nonspecific reactive lymphadenopathy should be confirmed by a period of careful observation and either repeat fine needle aspiration cytology or excisional biopsy.

I. Congenital Neck Masses

Cystic hygromas are congenital vasculolymphatic malformations that are frequently present at birth in about 50% and in remaining present by the age of 2 years. They have no prediction for sex or race and no malignant potential.

Clinical Features

Usually present in the posterior triangle of the neck, usually occupies the lower 1/3rd of the neck, it also present in the axilla groin, pelvis, mediastinum, retroperitoneal tissue and rarely seen in scrotum, mesentery and kidney.

Typical cystic hygromas cause no symptoms unless they enlarge in size or surround or invade adjacent normal anatomic structures. In this situation, cystic hygroma may cause symptoms such as feeding problems or breathing difficulties.

Swelling is soft cystic and brilliantly transilluminant, compressible and it increases in size when the child coughs.

Pathology

Cystic hygromas are multilobulated with thin wall, lymph containing sac where the fluid within the sac is usually clear or amber coloured, although occasionally it could be turbid or haemorrhagic.

Imaging Studies

After clinical evaluation, radiological assessment by using CT, Sonography and MR imaging is useful to confirm the diagnosis and define the extension and their relationship to adjacent structure.

Treatment

Excision is the treatment of choice and is recommended as soon as the diagnosis is established, because the incidence of infection, hemorrhage and growth increases with time.

Other methods of treatment such as aspiration, incision and drainage, irradiation and chemical sclerosis have not given any acceptable results.

Branchial Cleft Cysts

Branchial anomalies account for up to 17% of all pediatric cervical masses.

The branchial cleft apparatus that persists after birth may give rise to a number of neck masses. First branchial cleft cysts present in the preauricular or submandibular areas in association with external auditory canal or parotid gland and may require dissection of the facial nerve during excision.

Most of the branchial anomalies arises from the second branchial apparatus. A number of theories exist to explain the development of abnormalities within the cleft. The most widely held belief is that

incomplete obliteration of the cervical sinus plays an important role in this processes.

These cysts are more common in submandibular space, because of anatomic relationship of the second branchial apparatus and the cervical sinus. However, they can occur anywhere along a line from the oropharyngeal tonsillar fossa to the supraclavicular region of the neck.

Second Branchial Cleft Cysts Types

Type I : Most superficial and lies along the anterior surface of the sternocleidomastoid muscle, just deep to the platysma muscle.

Type II: This is most common which lies along the anterior surface of the sternocleido muscle, posterior to the submandibular gland.

Type III: Cyst extends medially between the bifurcation of the internal and external carotid arteries to the lateral pharyngeal wall.

Type IV: Cyst lies in the pharyngeal mucosal space and is lined by columnar epithelium.

Clinical features

The most common symptoms are sore throat, Dysphagia, dysarthria, dyspnea, abscess formation which adds pain, fever and cervical stiffness. Rarely paralysis of cranial nerves also occur.

On Examination

Painless fluctuant mass lies between the angle of the mandible and the anterior border of the sternomastoid muscle approximately measures about 2 x 3 cm.

Pathology

Cyst is usually filled with a turbid yellowish fluid and may contain cholesterol crystals. The walls are thin and lined by stratified squamous epithelium overlying lymphoid tissue.

USG sonographic appearance of a branchial cleft cyst is described as an echoic, well circumscribed cyst.

Treatment

Conservative Methods

Repeated aspiration of the cystic fluid, Marsupialization and injection of sclerosing agents but these methods often provide only temporary resolution of the symptoms. Definitive treatment:

Total excision of the cyst

Branchial Fistula and Sinus

Nearly always sinus is congenital and commences to discharge soon after birth and may present unilaterally or bilaterally. They are prone for attacks of inflammation repeatedly. They are derived from second branchial cleft which is persistent. They present in the first decade of life. A fistula can be acquired by the excision of a branchial cyst.

Clinical Features

Anomalies of first branchial cleft are seen in the submandibular and preauricular regions. Fistula of the 2nd branchial cleft are encountered along the anterior border of sternomastoid muscle in the lower 3rd, the tract extrudes mucus from the opening. It may be incomplete and ends blindly in the region of the lateral pharyngeal wall (sinus). A complete fistula may have an internal opening just behind the tonsil.

Treatment

Complete excision of fistula and sinus is the treatment of choice.

Transverse elliptical incision is made around the fistulous opening and a blunt metal probe can be inserted to facilitate accurate dissection and complete excision. Injection of methylene blue also facilitates dissection.

Dermoid Cyst

It is usually a midline mass present at birth derived from remnants of epithelial cells and may contain skin appendage, hilar and desquamated epithelium. It may be above or below the myelohyoid, therefore it requires bimanual palpation.

Clinical Features

Dermoid cyst is soft painless mass. They do not follow the movements of the tongue and are not transilluminant.

Treatment

Surgical removal is the treatment of choice.

Haemangioma

Haemangiomas are the most common benign tumours of infancy about 14 to 21% and the head and neck is affected most often within the masseter and trapezius muscles.

They typically appear shortly after birth, proliferative for 6-12 months and upto 50% may involute spontaneously. They are three times more common in females than in males.

Treatment

Steroids, Radiation Therapy, Cryotherapy, Laser and Surgical excision.

II. Acquired Neck Masses Ranula

Ranula is a cystic mass in the floor of the mouth or tongue which arises as a result of obstruction of minor salivary glands or sublingual gland.

They are caused by trauma or ductal abnormality.

Simple Ranula is confined to sublingual space, where as a plunging ranula presents as painless, non mobile neck swelling.

Complete excision is the treatment of choice.

Laryngocoele: Its a unilateral, occasionally bilateral narrow necked air containing diverticulum. It is due to

herniation of mucus membrane through the thyroid membrane, pierced by superior laryngeal vessels. It forms a visible swelling in the neck which appears when the patient blows his nose.

Sonographical Classification

1. Internal (simple) laryngocoeles are echo-free well defined structures inside the thyroid cartilage.
2. External (mixed) laryngocoeles have an additional cystic mass outside the laryngeal skeleton, which is connected through the thyroid membrane to the intralaryngeal component. When infected, the echoes within the laryngocoele are mixed and the walls may appear thickened.

Treatment

The sac should be excised, neck of the sac crushed, ligated and divided and stitched as in hernia repair.

Pharyngeal Pouch

Aetiology- It is protrusion through Killian's dehiscence which is a weak area of the posterior pharyngeal wall between thyropharyngeus and cricopharyngeus. Continued imperfect relaxation of the cricopharyngeus on swallowing repeatedly and high pressure in the oesophagus initiate the pouch.

As the pouch enlarges the resistance of the vertebral column behind causes it to turn usually towards the left.

Clinical features

Patients usually are elderly, males affected more than females. There are 3 stages in the development of symptoms.

Stage I: The diverticulum is small directed towards vertebral column, an incidental finding on a barium swallow, usually symptomless. Occasionally produces symptoms identical to foreign body in the throat.

Stage II: The diverticulum has larger mouth still in the vertical plane. Regurgitation of indigested food occurs unpredictably sometime after the meal, particularly after turning in the bed and the person may be awakened from bed by a feeling of suffocation followed by violent fit of coughing.

Stage III: The pouch has become large, mouth looks horizontally upwards, the pouch becomes dependent, so when the pouch is full it compresses the oesophagus. The symptoms of stage 2 persist in addition. There are gurgling noises in the neck when the patient swallows. In 1/3rd of the cases, the pouch is large enough to be visible in the neck and the pouch can enlarge when the patient drinks.

There is increasing dysphagia, with progressive loss of weight due to semistarvation and cachexia.

Investigations

Barium swallow a very thin emulsion must be used, the fundus of the sac may be seen invading the superior mediastium. A postero anterior view as well as a semilateral radiograph must be taken when the overflow of barium emulsion may be seen.

Chest x ray: reveals aspiration pneumonitis. Occasionally an associated hiatus hernia may be present which should be treated.

Oesophagoscopy: is unnecessary for diagnosis and dangerous because it may perforate the pouch and cause mediastinitis.

Treatment: When the pouch is of considerable size, surgery is strongly advised. when emaciation is extreme a temporary feeding gastrostomy or jejunostomy may be required.

Antibiotics are given prior to and after surgery to prevent mediastinitis.

Surgery: Pouch is inspected endoscopically and packed with ribbon gauze. Ryles tube is passed which will help to detect the position of the sac during dissection.

Approach: A transverse incision at the level of the cricoid cartilage or oblique incision following the anterior border of sternomastoid.

After treatment: Patient is fed through an indwelling transnasal gastric tube for 3 days after which gradually liquid diet is introduced.

Complications: 1. infection 2. pharyngeal fistula 3. stenosis 4. vocal cord paralysis

III. Inflammatory conditions of the neck:

Ludwig angina: Ludwig angina is defined as a potentially lethal, rapidly spreading cellulitis, involving the sublingual and submandibular spaces accompanied with tender swelling in the floor of the mouth with elevation and posterior displacement of the tongue, suprahyoid induration.

As this infection, cellulitis spreads in continuity along the fascial and muscle planes, rapid involvement of interconnecting tissue spaces such as lateral pharyngeal space may occur.

Clinical features

Presence of increasing swelling in the neck can lead to compression of airway, trismus, elevation of tongue, edema of the glottis.

Treatment:

Early aggressive intravenous antibiotic therapy and surgical decompression combined with alternative methods of airway management.

The immediate role of tracheostomy in these cases is questionable.

Parapharyngeal Space Infection and Abscess

Surgical anatomy of the PPS Parapharyngeal space

has pyramidal shape with a superior base. At the level of the palatine tonsil, its lateral limits are the deep lobe of the parotid gland in the posterior part and the ramus of the mandible covered by the medial pterygoid muscle in the anterior part. Its medial limits at this level are the retropharyngeal space in the posterior part and superior constrictor muscle covering the pharynx in the anterior part. Its posterior limit is the vertebrae and prevertebral muscles, and its anterior limit is the pterygomandibular raphe in which the buccal mucosa adheres to the anterior part of the ramus of the mandible.

Classically, the PPS is divided into 2 compartments separated by styloid muscles. The anterior or prestyloid compartment contains essentially the parapharyngeal fat. The posterior or retrostyloid compartment contains the internal jugular vein, the internal carotid artery, X, XI, XII cranial nerves and lymph nodes. The PPS communicates medially with the retropharyngeal space, laterally with the parotid space and inferiorly with the submandibular space.

Infection of this space usually originates from tonsil or after tonsillectomy. Abscess may burst spontaneously between the cartilaginous plates of

external auditory canal.

Clinically: Trismus is present with swelling over the lower part of the parotid gland. Swelling appears over the angle of the mandible when the apex is involved.

Complications: Thrombophlebitis of internal jugular vein, erosion of internal carotid artery, mediastinitis, pericarditis, meningitis and septic shock, airway obstruction, death.

Treatment: Incision below and behind the angle of the mandible on a line towards the hyoid bone. A finger is passed medial to the mandible and the space is entered and a large soft wick drain is kept.

Cervical Lymphadenitis

Inflammation of the lymph nodes in the neck.

Types: acute lymphadenitis, chronic lymphadenitis, pyogenic lymphadenitis and granulomatosis or tuberculous lymphadenitis.

Acute lymphadenitis: infection occurs from oral, nasal cavities, ear scalp and face. Lymphnodes are enlarged tender, pyrexia may be present.

Treatment: Antibiotics, if abscess formation occurs, incision and drainage. Chronically inflamed lymphnodes not resolving in the space of 3-4 weeks are nearly always tuberculosis.

Tubercular Lymphadenitis



INTRODUCTION

Mycobacterium tuberculosis is the most common cause of mycobacterial lymphadenitis and lymphadenitis due to non tuberculosis mycobacteria is being increasingly encountered. Peripheral lymphnode involvement is the commonest form of extrapulmonary tuberculosis and cervical region is the most frequently affected site.

EPIDEMIOLOGY

In the developing and underdeveloped countries, tuberculous lymphadenitis continues to be the most common lymphadenitis and lymphadenitis due to NTM is seldom seen.

In India mycobacterium tuberculosis has been the most common pathogen isolated from patient with mycobacterial lymphadenitis accounting for almost all the cases. Non tuberculous mycobacterium are the most frequently isolated pathogens from the lymphadenitis specimens in several parts from developed world.

PATHOGENESIS

Mycobacterium tuberculosis generally enters the body via the respiratory tract and undergoes haematogenous and lymphatic dissemination. Hilar and mediastinal lymph nodes are the first lymph nodes to be involved. Tonsil is also an important portal of entry. The infection may spread via the lymphatics to the nearest cervical lymph nodes.

In the initial stages, the nodes may be discrete clinically. Periadenitis results in the matting and fixity of the lymph nodes, the lymph nodes coalesce and breakdown to form caseous pus. This may perforate the deep fascia and present as a fluctuant swelling on the surface (collar stud abscess). Over lying skin

becomes indurated, breaks down and leads to the formation of a sinus. Healing may occur with calcification or scar.

In NTM lymphadenitis, the pathogens usually enter the lymph nodes directly via oropharyngeal mucosa, salivary glands and tonsils.

CLINICAL FEATURES

Tuberculous cervical lymphadenitis tends to occur more often in females and presents in young adults.

Is usually presents as slowly enlarging lymph nodes and may be asymptomatic. Some patients manifest systemic symptoms. This includes fever, weight loss and fatigue, occasionally night sweats. Cough may be a prominent symptom in patient with mediastinal lymphadenopathy.

Jones and Chambel had classified peripheral tuberculous lymphadenopathy into five stages.

Stage I: Enlarged, firm, mobile, discrete nodes showing nonspecific reactive hyperplasia.

Stage II: Larger rubbery nodes fixed to surrounding tissue owing to periadenitis.

Stage III: matted nodes

Stage IV: Central softening due to abscess formation.

Stage V: Collar stud abscess formation.

Stage VI: Sinus tract formation.

DIAGNOSIS

Tuberculin Test

Tuberculin test is positive in about 75% patients with lymph node tuberculosis while it is often non reactive in patient with NTM lymphadenitis. However, a negative tuberculin test does not rule out the possibility of tuberculosis.

Mantoux Test

This is a single puncture tuberculin test, and most widely used procedure for administration of tuberculin.

Strengths

1. ITU- first strength PPD
2. 5TU- intermediate strength
3. 250 TU-Second strength

The above doses can be used for tuberculin skin testing. Interpretation

Tuberculin test begins positive at five to six hours, maximal at 48-72 hours By 24 hours:

- Induration of 10 mm or above is considered +ve
- Induration of less than 5 mm is considered –ve
- Induration between 6-10 mm is doubtful.
- >20 mm is considered to be strong reactive, increased chance of developing active tuberculosis.

Sputum AFB: Early morning sputum sample for consecutive 3 days. Imaging study

1. Chest radiograph to know the paratracheal, hilar and mediastinal lymphadenopathy and pleural effusion.
2. Ultra sound and CT abdomen may be required to assess the status of retroperitoneal, mesenteric nodes.

FNAC is a relatively non invasive, pain free, outpatient procedure for the diagnosis of peripheral lymphadenopathy.

Lymph node biopsy may be required when FNAC result is doubtful. Excisional biopsy remains the gold standard for diagnosis of TB lymph node.

Histopathology

The definitive diagnosis of TB lymph node is established by visualizing mycobacteria on histopathological sections or on smears stained for acid fast bacilli or by mycobacterial culture.

Treatment

Most of the patients can be managed with short course

6 month chemotherapy regimen. Surgery is rarely needed.

Initially started with 4 drugs for 2 months INH 5-10 mg/ kg usually 300 mg Rifampicin – 10 mg/kg up to 600 mg

Pyrazinamide – 20-40 mg/kg up to 1500-2000 mg Ethambutol – 15-25 mg/kg up to 1 gm

Followed by 2 drugs for 4 months INH and Rifampicin.

IV. Tumours of the Parapharyngeal Space:

Lymphomas

Lymphomas are primary malignancy of lymphoid cells. They are broadly classified as Hodgkin's lymphoma and Non-Hodgkin's lymphoma.

Hodgkin's Lymphoma Epidemiology

This disease demonstrates a classical bimodal incidence, the incidence reaches a peak in the late teens and falls during the middle ages but increases again with advancing age. It shows a male predominance and it is more common in higher social classes.

Etiology

The etiology of Hodgkin's lymphoma has not been determined but there is increasing evidence of infectious etiology. HIV is a risk factor for Hodgkin's lymphoma. EBV infection is also associated with increased risk.

Pathology: It is B cell origin. Malignant cells form only a small part of the tumor. The characteristic cells are the mononuclear Hodgkins cell, the definitive binucleate or multinucleate Reed-Steinberg cells and its variants like lacunar cells and lymphocytic histiocytic cells. RS cells are giant cells with abundant eosinophilic cytoplasm, multiple large deep blue staining nucleoli and characteristic two or more

nuclei, the so called owl eye appearance.

Classification

Hodgkins lymphoma is classified by Rye into the four following types.

1. Lymphocyte predominant Hodgkins disease: Here the predominant cells are lymphocytes and histiocytes, RS cells are rare.
2. Nodular sclerosing type (40-60%) It is characterized by thickening of lymph node capsule with interconnecting fibrous bands that divide the node into nodules.
Lacunar cells, a variant of RS cells are usually seen.
3. Mixed cellularity (15-30%) Lymph nodes are affected with the pleomorphic cellular infiltrate comprising a mixture of normal histiocytes, neutrophils and plasma cells. This type lacks sclerosis. RS cells are seen in abundance.
4. Lymphocyte depletion (5%) It shows general cellular depletion, numerous RS cells are seen

Clinical Features

It commonly presents as large painless progressive, discrete rubber lymphadenopathy usually in the neck and less commonly in the axilla. It spreads through lymphatic channels in an orderly fashion with successive involvement of contiguous lymph node regions. A third of patients complain of systemic symptoms like weight loss, fever and drenching night sweats. Pruritis is common it is severe and debilitating. Patients sometimes, complain of pain in the involved sites following alcohol ingestion. Extra nodal presentation rare (<5%) mostly seen in bone marrow, liver lung, pericardium pleura splenic enlargements and hepatomegaly is seen in 75% and 50% of cases respectively. It can present as superior

venacaval obstruction. It can sometimes present as symptomless mediastinal mass on chest X-ray. Occasionally Hodgkins lymphoma can present as pyrexia of unknown origin, the fever persisting for days to weeks followed by an afebrile interval and then recurrence. This pattern is called Pel Ebstein fever. It can also present with cutaneous disorders such as erythema nodosum, ichthyosis, paraneoplasitic syndromes like GB syndrome, Ophelia syndrome, hypocalcaemia and nephritic syndrome are also possibilities.

Ann Arbor Staging

Stage Definition

- I. Involvement of a single lymph node region or lymphoid structure (e.g., spleen, thymus, waldeyer's ring) or a single extra lymphatic organ or site (IE)
- II. Involvement of two or more lymph node regions on the same side of the diaphragm (the mediastinum is a single site: hilar lymph nodes should be consider later and when involved on both sides, constitute stage II disease).
Or localized involvement of an extralymphatic organ or site(IIIE)
- III. Involvement of lymph node regions or lymphoid structures on both sides of the diaphragm
Or localized involvement of an extralymphatic organ or site(IIIE) or spleen(IIIS) or both (IIISE)
- IV. Diffuse or disseminated involvement of one or more extralymphatic organs with or without associated lymph node involvement. Bone marrow and liver involvement are always stage IV.
Identification of the presence or absence of symptoms should be noted with each stage designation:

A: asymptomatic

B: fever , sweats, unexplained weight loss greater than

10% of body weight in a period of 6 months before staging investigation is done.

INVESTIGATIONS

Complete blood counts, ESR, liver function tests, renal function tests, lymph node biopsy and biopsy of extra lymphatic site. Bone marrow trephine biopsy is preferred, it is useful in assessing involvement of bone marrow. Bone marrow trephine biopsy is preferred, it is useful in assessing involvement of bone marrow.

Chest Radiography to look for mediastinal lymphadenopathy. CT scan for the chest, abdomen and pelvis. It has the ability to image enlarged lymph nodes not seen on lymphography. Radiographic skeletal survey. Bone involvement occurs in 5% of cases. Lesions may be sclerotic, lytic or mixed in appearance.

RADIO ISOTOPE SCANNING

PET scan has shown encouraging results in pretreatment staging and remission assessment.

STAGING LAPAROTOMY

It is the most sensitive investigation for assessment of intra abdominal Hodgkins disease. It is a solely diagnostic procedure with no direct therapeutic

benefit. Laparotomy should be performed by an experienced surgeon. But advances in imaging

techniques with wide spread availability of dynamic helical CT scan, lymphangiography, PET, improved non operative staging has lead to a dramatic decrease in the number of patients requiring staging laparotomy.

TREATMENT

Stage 1A and stage 2A- (supra diaphragmatic) wide field irradiation to the mediastinal, axillary, cervical and supra clavicular area (mantle field radiation

Stage 1A and stage 2A- (sub diaphragmatic) inverted Y radiotherapy to include iliac and para aortic nodes , splenic irradiation.

Stage 1B and stage 2B- chemotherapy as initial treatment (MOPP/ABVD) Stage 3A-chemotherapy

Stage 3B and stage 4-chemotherapy as initial treatment.

Relapse following Radiotherapy: salvage chemotherapy (MOP or MOP like regime)

Relapse following chemotherapy -high dose chemotherapy followed by bone marrow transplantation.

The Commonly Used Regimen

Name	Daily dose (mg/m ²)	Administration route	Administration on day	freq
Mechlorethamine	6	I.V	1,8	
Vincristine O	1.4	I.V	1,8	every4wks
Procarbazine P	100	P.O	1-14	for6-8
Prednisone p	40	P.O	1-14	cycles
Adriamycin A	25	I.V.	1-15	
Bleomycin B	10	I.V	1-15	
Vinblastine V	6	I.V	1-15	
Dacarbazine D	375	I.V	1-1	

Non-Hodgkins Lymphoma

Non-Hodgkins lymphoma are a group of lymphoid malignancy resulting from the clonal expansion of B or T lymphocytes.

Etiology

The cause in most of the cases is unknown

The factors associated with development of lymphoma are Immunodeficiency: Severe combined immunodeficiency, Hypogammaglobulinaemia, Wiskott Aldrich syndrome.

Infection: EBV, HTLV-1, HIV, HCV, Helicobacter Pylori. Herbicides : 2, 4-Dichlorophenoxy acetic acid Auto immune disorders: Rheumatoid Arthritis, Psoriasis, Celiac sprue. Post solid organ transplantation

History and Physical Examination

Clinically most patients present with lymphnode enlargement with or without hepatosplenomegaly.

Extra lymphatic manifestations are common in the region of head and neck, gastro intestinal tract, skin and rarely in the bone, testis, brain, liver and lungs.

The presence of specific symptoms like fever, night sweats and unexplained weight loss are known to have an adverse prognosis in patients.

A careful physical examination must include all lymph node bearing areas, the presence of hepatosplenomegaly and other organ systems.

Investigations: same as Hodgkin’s disease.

Classification of Non Hodgkin’s lymphoma

Kiel classification	Working formulation	Revised European-American Classification
Low-grade malignancy	Low grade	b-cell lymphomas
Lymphocytic, CLL	a.malignant lymphoma, small lymphocytic leukemia	low grade B-CLL
Lymphopoyetic, other	b.malignant lymphoma, follicular, predominantly small cleaved cell diffuse areas	lymphoplasmacytoid lympho follicle center lymphomas
Lymphoplasmacytoid	sclerosis	marginal zone lymphomas
Centrocytic	c.malignant lymphoma, follicular mixed, small cleaved and large cell diffuse areas	(MALT)
Centroblasticcentrocytic	sclerosis	Mantle cell lymphoma
Follicular without	immediate grade	Aggressive
Sclerosis	d.malignant lymphoma, follicular predominantly large cell diffuse areas sclerosis	diffuse large b-cell lymphoma
Follicular with sclerosis	e.malignant lymphoma, diffuse small cleaved cell	Primary mediastinal large b-cell lymphoma
Follicular with sclerosis	f.malignant lymphoma, diffuse mixed, small and large cell sclerosis epithelioid cell component	Burkitts lymphoma
Follicular and diffuse, with Sclerosis	g.malignant lymphoma, diffuse large cell	Precursor b-cell lymphoid
Diffuse	large cell cleaved cell	Lymphoma/leukemia
Low-grade malignant	non-cleaved cell	t-cell lymphomas
Lymphoma, unclassified	sclerosis	low grade T.CLL
High-grade malignancy	high grade	mycosis fungoides/sezary syndrome
Centroblastic	h.malignant lymphoma large cell	aggressive peripheral-cell lymphoma, unspecified
Lymphoblastic, burkitt's Type	Immunoblastic	angioimmunoblastic t-cell lymphoma
Lymphoblastic, convoluted Cell type	Plasmacytoid	lymphoma
Lymphoblastic, other (unclassified)	Clear cell	angiocentric lymphoma
Immunoblastic	Polymorphous	intestinal t-cell lymphoma
High-grade malignant	Epithelioid cell component	lymphoma
Lymphoma, unclassified	i.malignant lymphoma lymphoblastic convoluted cell	anaplastic large cell lymphoma
Malignant	non convoluted cell	
Lymphoma, unclassified (unable to specify high Grade or low grade)	non convoluted cell	
Composite lymphoma	j.malignant lymphoma small	

Treatment Radiation Therapy

It was the first treatment used for treatment of lymphoma Mega voltage may be used in stage 1 disease.

Superficial beam therapy is used in skin lymphomas

Total body irradiation is used as a part of myeloablative therapy. Involved field radiation is used for localized disease. It is an essential component of palliation

Chemotherapy:

It is the predominant mode of treatment in most patients.

Low grade NHL also called indolent lymphoma more than 80% present as stage IV disease with involvement of the bone marrow.

Chemotherapy produces complete response in most patients. Generally used regimens are CHOP – Cyclophosphamide, Doxorubicin, Vincristine, Prednisolone. COPP-Cyclophosphamide, Vincristine, Prednisolone, Procarbazine every month for 4 to 6 cycles.

Intermediate and high grade: CHOP or COPP. complete response in 45-55% of patients and cure approximately 30-35%, with addition of methotrexate, bleomycin, leucovorin, cytosine arabinoside drugs suggest increased rate of complete remission (85-85%),

Biologic Therapy

Interferon alpha, Monoclonal antibodies, Radio labeled monoclonal antibodies have been tried



In 90% of cases the cause of a neck lump will be in the head and neck. A vigilant search above the clavicle will provide the primary tumour site in approximately 50% of cases.

Most of carcinomas of central head and neck metastasize to the lymph nodes in deep cervical chain. A lymph node in the posterior triangle, particularly in young people may represent a metastasis from the post nasal space.

A cancer presenting with a node in the neck is four times more common in men especially those who have smoked than in women, with a mean age of 65 years in men and 55 years in women.

A secondary malignant node in the neck may also be due to a tumour below the clavicle; the lung, stomach,

breast, ovary, testis, these nodes are usually present in the left supraclavicular fossa and known as Virchow's nodes (Trosier's sign).

The histology of occult nodal carcinoma of unknown primary varies from case to case but between one third and one half of all such nodes are infiltrated by squamous carcinoma, with about one quarter reported as undifferentiated or anaplastic carcinoma and similar number of adenocarcinoma, if supraclavicular nodes are involved.

The primary sites in order of frequency are the nasopharynx and tonsil, along with the retromolar trigone, tongue base and pyriform fossa.

Papillary thyroid carcinoma tends to metastasis through the lymphatic system and cervical lymph node metastasis is found in 40-60% of patients at the time of diagnosis.

Advanced testicular germ cell tumour is established before the neck nodes is noted. In rare cases these

tumours have been found along with cervical lymphadenopathy in patients with a previously undiagnosed primary tumour.

Most common primary sites in patients presenting with neck masses. Diagnosis

Nodal level	Primary sites
I. (submandibular & submental)	Oral cavity
II. (Upper jugular)	Oro.nasopharynx & supraglottic larynx
III. (middle jugular)	Hypopharynx, larynx
IV. (lower jugular)	Thyroid, hypopharynx, larynx
Supraclavicular sites	
V. (posterior triangle)	Scalp, nasopharynx

HISTORY

Mass in the neck usually presents after several weeks with history of quick increase in size and is often painless. It is important to ask about other symptoms of disease within the head and neck particularly dysphagia, hoarseness, sore throat and nasal obstruction, and for pulmonary and gastric symptoms

such as cough, haemoptysis, indigestion, loss of weight and loss of appetite.

EXAMINATION

Full clinical examination of head and neck should be carried out. The mass should be examined for size, mobility and fixation to deep tissue & other lymphatic sites in the axilla, groin, and also palpate for the enlargement of the stomach, liver, spleen and testes.

Fine Needle Aspiration Cytology

Fine needle aspiration cytology offers an accurate, sensitive, inexpensive and rapid method for evaluation of a cervical adenopathy or mass. The diagnostic

reliability of aspiration biopsies of lymph nodes, a sensitivity of 85% and a specificity of 99% were achieved .Better results are always obtained if an experienced person aspirates the mass, slide preparation is critical for accurate diagnosis, and immediate inspection in a specialized cytopathology clinic allows additional material to be acquired of the aspirate if acellular or if further material is required for immunochemistry or culture. For patients with poorly defined or deep seated lesion image or ultrasound guidance can be used.

Role of computerized tomography, endoscopy and positron emission tomography:

Patients should undergo computed tomography of the head, neck and chest which should be followed by endoscopic examination of the upper aerodigestive tract under general anaesthesia. During endoscopy, biopsies should be taken of any suspicious lesions detected by computed tomography and also samples

taken from the base of tongue and nasopharynx, as neoplasms from both of these sites often present with an isolated neck metastasis.

PET : The role of positron emission tomography has yet to be determined, but occasionally it proves

helpful in finding either the primary tumour or unsuspected secondaries.

Management of Metastatic Neck Disease

Regional lymph node (N) staging for head and neck cancer

Stage	Description
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 >3cm but <6cm
N2b	Metastasis in a single ipsilateral lymph node none>6cm
N2c	Metastasis in bilateral or contralateral lymph node
N3	Metastasis in any lymph node>6cm

Staging of Head and Neck Cancer

Stage	Stage grouping
0	Tis, N0, M0
I	T1, N0, M0
II	T2, N0, M0
III	T3, N0, M0 & T1-T3, N0, M0
IVA	T4a, N0-1, M0 & T1-T4a, N0, M0
IVB	T4b, any N, M0 & any T, N, M0
IVC	Any T, any N, M.

TREATMENT

Surgery-Best treatment for patients with clinically positive neck nodes with upper aero digestive squamous cell carcinoma is radical or modified radical neck dissection followed by post operative radiotherapy.

Node negative neck management is based on patterns and distribution of lymph node metastasis hence selective neck dissection is the standard elective treatment for clinically negative neck nodes, with selective irradiation of specific nodal group.

Neck Dissection

Neck dissection is valuable surgical procedure for treating metastatic nodal disease in the neck. Since the first description of radical neck dissection by George Crile in 1906, this procedure remained the cornerstone of surgical management of metastatic neck node for most of the past century.

Following neck dissection can be performed. Classical radical neck dissection

Extended radical neck dissection Modified radical neck dissection (type-3)

Selective neck dissection (functional neck dissection)

Radical Neck Dissection

This operation removes the lymph node containing levels in the neck (I-V) and all three non lymphatic

structures (spinal accessory nerve, sternomastoid muscle and internal jugular vein)

Indications

Significant operable neck disease (N2a/ N2b/ N3) , prior to predicted flap reconstruction.

Contraindications

Untreatable primary tumour Patients unfit for major surgery Distant metastasis

Significant bilateral neck disease Inoperable neck disease

Extended radical neck dissection:

Removal of all structures resected in a radical neck dissection, along with one or more additional lymph node groups or non lymphatic structures or both.

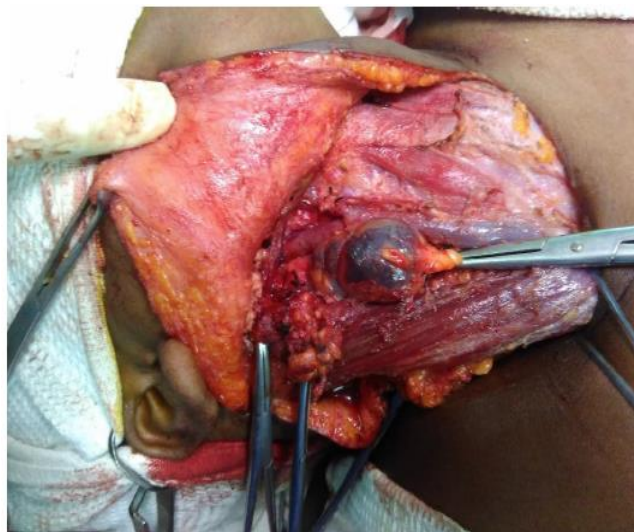
Additional lymph node groups include retropharyngeal, parotid or lymph nodes in level VI or VII.

Non lymphatic structures include mandible, parotid gland, prevertebral fascia, mastoid tip, diaphragmatic muscle, hypoglossal nerve and external carotid artery.

This is indicated when the tumour arises from above mentioned additional lymph node groups and non lymphatic structures.

Modified radical neck dissection

This operation consists of removal of lymph node groups (level I-V) with preservation one or more non lymphatic structures



Type I- Removal of all lymph node groups (level I-V) with preservation of spinal accessory nerve.

Type II- Removal of all lymph node groups (level I-V) with preservation of spinal accessory nerve and internal jugular vein.

Indication – when internal Jugular vein need not be resected. Type III – (Selective or functional neck dissection)

Removal of all lymph node groups (level I-V) with preservation of the spinal accessory nerve, internal jugular vein and sternomastoid muscle.

Treatment of N0 neck

Treatment of differentiated thyroid cancer, Skin tumours such as melanoma, squamous cells carcinoma

Selective neck dissection :

Type	Levels dissection	Main indication
Supraomohyoid	I-III	T1-T4 N0 Sq cell ca oral cavity
Extended supra Omohyoid (anteriolateral)	I-IV	Skin cancers (melanoma) anterior to the line of tragus
Lateral	II-IV	T2-T4; N0 Sq cell ca larynx, Oropharynx & hypopharynx
Posteriorlateral	II-V	Skin cancer (Sq cell ca & melanoma) post to the line of the tragus.
Anterior or central	VI	Differentiated thyroid carcinoma subglottic and hypopharyngeal SCC
Superior mediastinum	VII	Differentiated and medullary thyroid carcinoma, subglottic laryngeal and hypopharyngeal Sq cell ca.

Salivary Gland Disorders

The submandibular gland consists of a superficial and a deep part. The superficial part lies superficial to the mylohyoid muscle and hyoglossus, between the two bellies of the digastric muscle. The deep part lies below and lateral to submandibular gland and above and deep to mylohyoid muscle. The duct about 5cm long runs forwards from the deep part of the gland, opens on a papilla beside the frenulum of the tongue. The lingual nerve is attached to the superficial part, with the submandibular ganglion attached to its lower border. The facial artery enters the gland, towards the

posterior aspect of its deep surface or deeply grooves the gland and it lies behind the duct and is at risk when incision is made into the upper pole of the gland. Cause of benign enlargements of the salivary gland is inflammation.

Inflammatory Conditions of Submandibular Gland

Acute sialadenitis can be caused by Staphylococcus aureus and Mumps, Coxsackie virus.

Chronic sialadenitis is caused by granulomatous inflammations like Sarcodosis, Actinomycosis, Tuberculosis, Catscratch disease., Auto immune diseases like Sjogren Syndrome and post operative radiotherapy.

Clinical Features

Patient presents with a painful brawny swelling which corresponds to the shape of the gland, associated with fever and pus can be expressed from the duct.

Treatment

Antibiotics, meticulous oral hygiene and gland can be massaged gently at regular intervals. If not subsided by these treatment, incision and drainage should be considered.

Submandibular Gland Duct Obstruction

Causes: salivary calculi, strictures of duct wall, oedema or fibrosis of the papilla, pressure on the duct due to an adjacent mass, invasion of the duct by a malignant neoplasm.

Clinical Features

recurrent painful swellings at meal times. Proximal dilatation leads to repeated obstruction, inflammation and infection.

Investigations: X –ray of the submandibular gland region.

Sialography : a radiopaque dye is used as hypaque or lipiodol is introduced into the duct of the gland and a radiograph taken.

This enables detection of radiolucent obstructions, dilatation and narrowing of the duct and Salivary gland mass.

Treatment

Papillary stenosis- requires a papillotomy. Calculi within the duct - the duct wall is incised in the floor of the mouth and the stone extracted. Calculi within the gland- excision of the gland is performed.

Neoplasm of submandibular salivary gland

Tumours of salivary glands comprises those in the major glands (parotid, submandibular, sublingual) or in minor glands (oral mucosa, palate, uvula, floor of

the mouth, posterior tongue, peritonsillar area, pharynx, larynx and paranasal sinuses.

The parotid gland is the most common site of major salivary gland tumour and the palate is the most common site of minor salivary gland tumours. Approximately 20-25% of parotid tumours, 55-60% of submandibular tumours, 50% of palate tumours and 95-100% of sublingual gland tumours are malignant.

Diagnostic approach to submandibular salivary gland tumours :

History: Benign tumours grow slowly over a period of years but malignant usually grow rapidly from the onset and **the cardinal sign** of malignancy is pain.

In inflammatory or calculus disease there is often marked fluctuation in the size of the gland, together with pain and tenderness. In these conditions, eating always causes an increase in pain and swelling.

Examination

In inflammatory disease, one must see whether any pus can be expressed from the duct. In malignancy one must look for skin changes, tenderness over the swelling and also look for nerve involvement and consistency of the swelling. Submandibular gland should be palpated bimanually to know the deep lobe enlargement.

Imaging

Plain x-ray: Parotid tumours are almost always radiolucent, whereas submandibular stones are nearly always radio opaque. Chest x-ray is useful in malignant lesions such as adenoid cystic carcinoma. **Sialography:** This is the most useful radiological investigation in non-neoplastic salivary gland diseases.

USG : This is useful in assessing the cystic nature of tumours.

FNAC: a key diagnostic test, which has 95% sensitivity in salivary gland neoplasms.

CT or MRI: Magnetic resonance imaging (MRI) offers an advantage over computed tomographic scanning in the detection and localization of head and neck tumours and the distinction of lymph nodes from blood vessels. Bone invasion is better visualized by CT, MRI may give additional information on soft tissue extension especially of the deep lobe.

International Classification Of Salivary Gland Tumours

I. Epithelial Tumours

- Adenoma Pleomorphic adenoma Monomorphic adenoma (Adenolymphoma, oxyphilic adenoma)
- Muco epidermoid tumours
- Acinic cell tumours
- Carcinomas -Adenoid cystic carcinoma, Adeno carcinoma, epidermoid carcinoma, carcinoma in ex pleomorphic adenoma

II. Non Epithelial Tumours

Haemangioma, lymphangioma, lipoma, sarcoma.

Histological Classification

Malignant salivary gland tumours are divided histologically into low grade and high grade malignancies

Low Grade Malignancies: Acinic cell tumours, mucoepidemoid carcinoma (grade I or II)

High Grade Malignancies

Mucoepidermoid carcinoma (Grade III)

Adenocarcinoma poorly differentiated carcinoma: Anaplastic Carcinoma. Squamous cell carcinoma Malginant mixed tumours , Adenoid cystic carcinoma Mucorpidermoid and adenocarcinoma are the most common with adenoid cystic carcinoma next.

TNM Definitions

Primary Tumour (T)

TX: Minimum requirements to assess the primary tumour cannot be met To : No evidence of primary tumour.

T1: Tumor 2.0 cm or less in greatest diameter

T2: Tumor more than 2.0 cm but not more than 4.0 cm in greatest diameter T3 : Tumor more than 4.0 cm but not more than 6.0 cm in greatest diameter T4 : Tumor over 6.0 cm in greatest diameter.

Regional lymph nodes

NX: Nodal metastasis cannot be assessed N0: No regional lymph node metastasis

N1: Metastasis in a single ipsilateral lymph node, 3.0 cm or less in greatest dimension

N2: Metastasis in a single ipsilateral lymph node more than 3.0 cm but not more than 6.0 cm in greatest dimension, or in multiple ipsilateral lymph node, none more than 6.0 cm in greatest dimension, or in bilateral or contralateral lymph nodes, none more than 6.0 cm in greatest dimension.

N2a: Metastasis in a single ipsilateral lymph node, none more than 6.0 cm in greatest dimension

N2b: Metastasis in multiple ipsilateral lymph nodes, none more than 6.0 cm in greatest dimension

N2c: Metastasis in bilateral or contralateral lymph nodes, none more than 6.0 cm in greatest dimension.

N3: Metastasis in a lymph node more than 6.0 cm in greatest dimension.

Distant Metastasis (M)

MX: Minimum requirements to assess the presence of distant metastasis cannot be met

M0:No (known) distant metastasis M1: distant metastasis present **Stage I**

Stage I salivary gland cancer is defined as one of the following TNM groupings T1a, N0, M0

T2a, N0, M0

Stage II

Stage II salivary gland cancer is defined as the following TNM grouping T1b, N0, M0

T3b, N0, M0 T3a, N0, M0

Stage III

Stage III salivary gland cancer is defined as one of the following TNM grouping T3b, N0, M0

T4a, N0, M0

Stage IV

Stage IV salivary gland cancer is defined as any of the following TNM grouping T4b, any N, M0

Any T, N2 or N3, M0

Any T, any N, M1

Treatment

Early stage low grade salivary gland tumours are usually curable by adequate surgical resection alone.

Large bulky tumours or high grade tumours carry a poor prognosis and may best be treated by surgical resection combined with post operative irradiation.

Unresectable or recurrent tumours may respond to chemotherapy. Fast neutron beam radiation therapy or accelerated hyper fractionated photon beams have been shown to be effective in the treatment of inoperable, unresectable and recurrent tumours.

Neurogenic Tumors

Neuroblastoma is the most common extracranial solid tumour of childhood, accounting for 8-10% of all paediatric cancers.

The median age at diagnosis is 2 years in most cases. 85% are diagnosed by 5 years of age. Neuroblastoma is extremely rare in children over 10 years of age. Familial cases occur. Neuroblastoma has also been observed in patients with neuro fibromatosis,

Hirschsprungs disease, Beckwith Wiedemann syndrome and foetal hydantoin syndrome.

Neuroblastomas originate in neural crest cells of the sympathetic nervous system and secrete a variety of neurologically derived substances, including catecholamines, serotonin and ferritin.

Pathology

Pathologically neuroblastomas are classified into three histologic subgroups: neuroblastoma, ganglio neuroblastoma and ganglioneuroma. These consists of mature ganglion cells embedded in a bulky stroma composed of schwann cell sheets enveloping neuritic processes and perineural and endoneural elements.

Clinical Manifestations

The clinical presentation of neuroblastoma is varied. Common presentations include a hard, painless mass in neck, a localized intra thoracic mass found incidentally on chest radiographs or palpable abdominal mass.

A palpable abdominal mass may result from an enlarging primary adrenal or retroperitoneal tumour or from hepatomegaly secondary to tumour metastasis. Constitutional non specific symptoms such as fever, malaise and pain are frequent presenting features.

Laboratory and Radiological Evaluation

Laboratory studies include CBC, RFT, LFT, urine catecholamine. Chest and skeletal radiographs of skull and orbital views, bone scan and bone marrow aspiration.

Ultrasound abdomen and pelvis CT and MRI to know the accuracy of anatomic extent of the disease.

Treatment

The treatment of neuroblastoma is based on disease extent. Accurate staging of the tumour is essential.

International Staging System for Neuroblastoma

Stage Description

1. Localised tumour confined to the area of origin; complete gross excision, with or without microscopic residual disease; identifiable ipsilateral and contralateral lymphnodes microscopically negative.
- 2A. Unilateral tumour with incomplete gross excision; identifiable ipsilateral and contralateral lymphnodes, microscopically negative.
- 2A. Unilateral tumour with incomplete gross excision; positive ipsilateral lymph nodes; identifiable contralateral lymph nodes, microscopically negative.
3. Unresectable unilateral tumour or infiltrating across the midline with or without regional lymph node involvement or unilateral tumour with contralateral lymph node involvement or midline tumour with bilateral lymph node involvement.
4. Metastatic tumour involvement to distant lymph nodes, bone, bone marrow, liver and other organs.

Treatment

Stage 1: Surgical resection and observation only, no chemotherapy.

Stage 2A: Surgical resection and low dose of cyclophosphamide and doxorubicin for 5 cycles.

Stage 2B/3: Aggressive multiagent chemotherapy with radiation to regional disease.

Stage 4: Multiagent chemotherapy, autologous or allogenic BMT.

Neurofibromas and Schwannoma

Neurofibroma and schwannoma are derived from the nerve sheath Schwann cell is the parent cell of both tumours.

Neurofibroma also has an origin from the perineurium and is thus inseparable from the nerve of origin.

Malignant change is very unusual and may not occur at all. Surgical excision is the treatment.

Paraganglioma

Paragangliomas can occur in along the jugular venous system (glomus jugulare), vagus nerve and ganglia (glomus vagale), aortic arch (carotid body tumour), visceral autonomic system and adrenal medulla.

Paragangliomas are rare forming less than 0.5% of all tumours. Carotid body tumour is the commonest variety of paraganglioma.

Paragangliomas are derived from the mesenchymal elements of the third branchial arch and neural crest elements originating from the neural crest ectoderm.

As neural crest cells migrate in close association with autonomic ganglion cells, neoplasm arising from these migrating neural crest cells are referred to as "paragangliomas" or chemodectomas.

Clinical Features

Paragangliomas can be of the chromaffin or non chromaffin variety. The chromaffin variety presents with a pressor-amine syndrome characterized by dizziness, flushing, palpitations, Tachycardia, hypertension, arrhythmias, headache, diaphoresis and photophobia due to the production of epinephrine, norepinephrine and serotonin.

Histology

Tumours are highly vascular and between the many capillaries are clusters of cells called Zellballon or cellballs. Two types of cells make up the Zellballen, one the sustentacular or supporting cells and the other are epitheloid or chief cells, which have finely granular eosinophilic cytoplasm containing epinephrine, norepinephrine and serotonin.

Investigations

Angiography is the gold standard, in which characteristic vascular blush is seen. CT and MRI to know the extension of the tumour.

Blood catecholamines

Treatment : Surgical excision

Carotid body tumour

Carotid body tumour is the commonest variety of paraganglioma and the only pathology to affect the carotid body.

Etiology

1. Their etiology is unknown though hypertrophy of the carotid body and incidence of CBT are higher among those living at higher altitude as a result of relative hypoxia.
2. Recent biogenetic discoveries reveal that mutations in oxygen sensing genes are another etiology, accounting for approximately 35% of cases and that these two etiologies are probably additive.
3. Carotid body tumours are usually sporadic. Familial tumours accounts for 10% of all CBT, they have autosomal dominant mode of transmission and are characterized by higher incidence of bilateral tumours - 32% versus 5% bilateral tumours in the sporadic form.

Clinical features

CBT often present as asymptomatic mass. These masses progress steadily in size and can lead to hoarseness, dysphagia and horner's syndrome with about 20% having cranial nerve involvement .Only 5% of CBT are endocrinologically active.

Shamblin and colleagues described three anatomic groups of CBT

Group 1 Consists of relatively small tumours, which are minimally attached to the carotid vessels and surgical excision is not difficult.

Group II Tumours are larger with moderate attachment to the carotid vessels, these tumours are amenable to careful surgical excision.

Group III tumours are very large encasing the carotid vessels and often require arterial resection and grafting.

The reported rates of malignancy range from 2-50% in the literature. The malignant potential of these tumours is predicted only by the presence or absence of lymph node or distant metastasis and not by grade, size or other histological markers. These tumours rarely metastasize to kidney, thyroid, pancreas, brain and lungs.

Investigations

Catecholamine level estimation

Angiography would delineate neovascularity and help in exposure and hemostasis. Its the gold standard for the diagnosis of CBT. Splaying of the carotid bifurcation with a tumour blush known as the lyre sign is classical. Duplex scans, CT Angiograms and MRI are now becoming the accepted modalities of pre-operative imaging with the added advantage of being able to image the opposite side to rule out bilateral tumour.

Treatment

Alpha blockers like phentolamine or phenoxy benzamine are started two to three weeks prior to surgery to bring down the blood pressure and heart rate.

Preoperative embolization has been reported to be useful in decreasing vascularity and hence improve

the safety of surgical excision. However it carries the hazard of ICA thrombosis and cerebral embolization.

Surgery: Excision of CBT (subadventitial) is recommended at the time of initial diagnosis to avoid the difficulty of subsequent excision of an enlarging and highly vascular tumour with possible encasement of the carotid arteries and higher chances of post operative complications like cranial nerve palsies.

Radiotherapy: unsatisfactory, except for control of residual or recurrent disease.

Chemotherapy has no role.

MATERIALS AND METHODS

The present study involved 100 patients with swellings in the neck excluding thyroid who attended the Sri Krishna Medical College Muzaffarpur during the study period from September 2020 – September 2023

Inclusion Criteria

All patients presenting with neck swelling

Exclusion Criteria

Those patients with thyroid and its associated swellings. The data was collected in a proforma approved by the guide. After detailed history and clinical examination, fine needle aspiration cytology of involved lymph nodes were performed. Biopsy of the lymph nodes were performed when fine needle aspiration cytology was either negative or doubtful.

Routine tests like hemoglobin percentage, total count, differential count, erythrocyte sedimentation rate, chest radiographs were done in all patients. Sputum

examination for acid fast bacilli done for only suspected cases of TB lymphadenitis.

After conformation of diagnosis all TB patients were treated with short term chemotherapy and were followed up at monthly intervals for 6 months and progress was assessed by clinical examination as well as monthly ESR estimation. Cystic hygromas, branchial cysts and lipoma were excised. Metastatic work up was done for unknown primary with relevant investigations. The age and sex distribution, clinical presentation, diagnostic methods and treatment were evaluated and compared with standard published literature.

OBSERVATION AND RESULTS

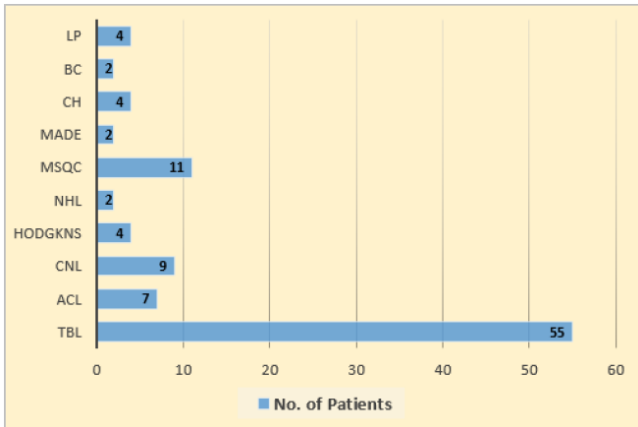
The data of 100 patients presenting with swelling in the neck (excluding thyroid) who were out patient and in patients in Sri Krishna Medical College Muzaffarpur. Tuberculosis was the commonest cause of lymphadenitis, observed in 55 out of 100 cases. Other cases of cervical lymphadenopathy were metastatic (13 patient) , chronic nonspecific lymphadenitis (9 patients), lymphomas (6 patients), acute lymphadenitis (7 patients). Other swellings of the neck included in this study were cystic hygroma (4 patients), branchial cyst (2 patients) & lipoma (4 patients).

Table 1: Total Distribution of Neck Swellings

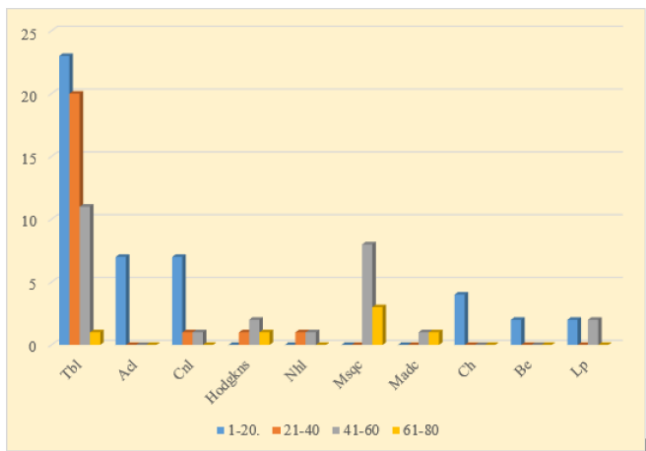
Disease Distribution	No. of Patients
Tbl	55
Acl	7
Cnl	9
Hodgkins	4
Nhl	2
Msqc	11
Made	2
CH	4
BC	2
Lipoma	4

The majority of cases among the study were tubercular lymphadenitis followed by secondaries in the neck, chronic non specific lymphadenitis.

Graph 1. Total distribution of neck swellings



Graph 2: Age distribution



- o Among the patients studied with a neck swelling, the age distribution was from 1 to 80 years.
- o Tuberculosis was observed in 55 out of 100 cases of cervical lymph node enlargement, the commonest age group affected was 5-20 years.
- o Secondaries in the neck accounted for 13 out of 100 cases of cervical lymph node enlargement, the age group affected was above 40 years.

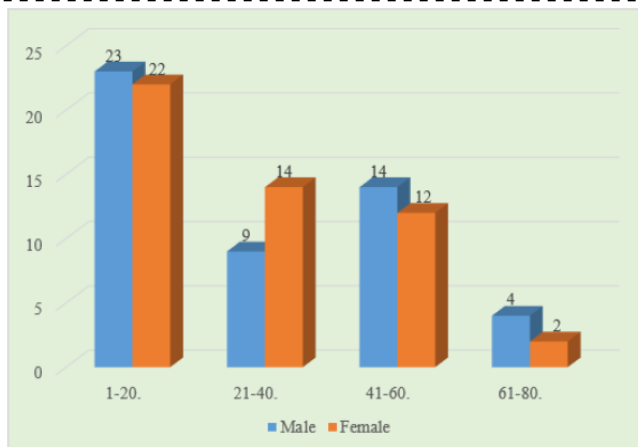
- o Lymphoma was observed in 6 out of 100 cases of cervical lymphadenopathy. The commonest age group affected was 41-60 yrs.
- o Cystic hygroma was diagnosed in 4 out of 100 cases. The age range being was 5-15 years.
- o Branchial cyst was diagnosed in 2 out of 100 cases.

Table 3. Sex Distribution

Age	Male	Female	Total
1-20	23	22	45
21-40	9	14	23
41-60	14	12	26
61-80	4	2	6
Total	50	50	100

CC-0.138 p value-0.587

Graph – 3 Sex Distributions



Sex distribution of the 100 cases of neck swelling : fifty were males and fifty were females (male : female ratio 1:1)

- 55 out of 100 cases were diagnosed to have tubercular lymphadenopathy. 21 were males and 34 were females with M:F ratio-being 1:1.5, showing female predominance.
- Secondaries in the neck were diagnosed in 13 out of 100 cases (squamous cell carcinoma, adenocarcinoma). Out of which 11 were males and 2 females, showing male predominance.
- Cystic hygroma was diagnosed in 4 out 100 cases in which 2 were male patients and 2 females, showing equal sexual distribution (1:1).

- Lymphoma (Hodgkins & NonHodgkins) was diagnosed in 6 out of 100 cases.

5 were males and 1 female, showing male predominance.

- Branchial cyst was diagnosed in 2 cases. Both patients were females.

Socio Economic Status:

Most of the patients were poor and daily wage workers and most of them came from over- crowded and rural areas. 79 patients out of 100 cases belonged

to lower socio economic class. In tuberculosis 48 patients (87.3%) belonged to the lower socioeconomic group.

Table 4: Socio Economic Status

	Low	High
Tbl	48	7
Ac1	6	1
Cnl	7	2
Hodgkins	4	0
Nhl	0	2
Msqc	7	4
Made	2	0
Ch	1	3
Be	1	1
Lp	3	1
TOTAL	79	21

CC-0.419, P Value-0.012

Graph 4: Socio Economic Status

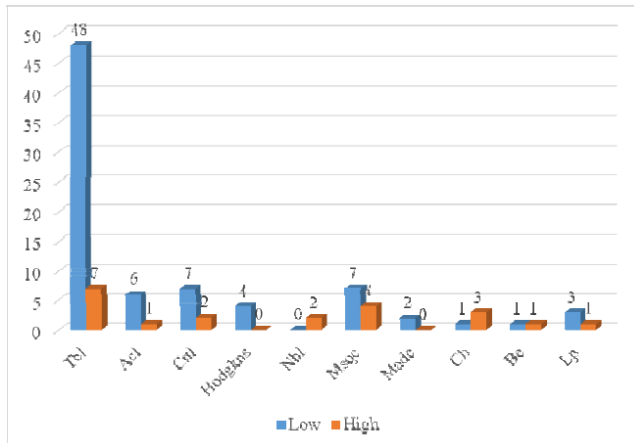


Table 5: Distribution of the swelling

Disease	Right	Left	Both	Front	Total
Tbl	33	15	7	0	55
Acl	4	3	0	0	7
Cnl	4	3	1	1	9
Hodgkins	1	0	3	0	4
Nhl	1	0	1	0	2
Msqc	4	7	0	0	11
Made	0	2	0	0	2
Ch	0	4	0	0	4
Bc	0	1	1	0	2
Lp	2	1	0	1	4
Total	49	36	13	02	100

CC-0.595 P Value-.001

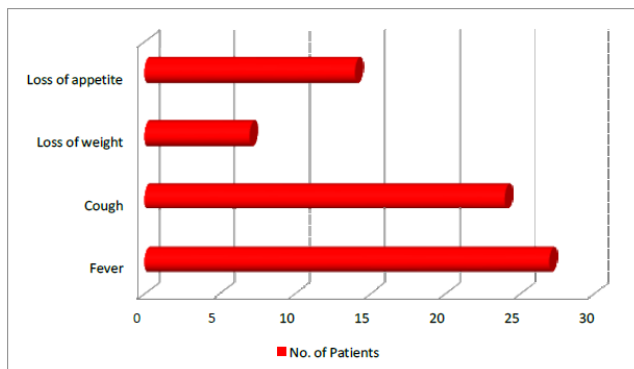
Out of 100, 49 (49%) patients were having swelling on right side of the neck, and 36 on left side, 13 presented with either side of the neck, 2 patients presented with

swelling in front of the neck. In tuberculosis 33 out of 55 (60%) presented with right sided cervical lymphadenopathy where as metastatic lymph node were found predominately affecting left side lymph nodes.

Table 6: Constitutional symptoms

Symptoms	No. of patients
Fever	27
Cough	24
Loss of weight	7
Loss of appetite	14

Graph 5: Constitutional Symptoms



Among the cases of tuberculosis studied only 27 out of 55 cases (49%) presented with fever. 24 cases presented with history of cough and 7 cases presented with a history of loss of weight and 14 cases presented with loss of appetite. Only 5 cases (9.1%) came with a background of positive family history of tuberculosis. 2 patients were having past history of tuberculosis with incomplete course of chemotherapy. Out of 6 diagnosed cases of lymphoma, 3 cases presented with fever, 4 cases presented with loss of weight and 3

cases presented with loss of appetite. Patients with secondaries in the neck, 8 cases presented with loss of weight, 5 cases presented with loss of appetite and 2 patients presented with dysphagia. All patients of tuberculosis were followed at monthly interval for six months during the course of chemotherapy and progress was assessed by clinical examination as well as ESR estimation.

Level of Lymph Node Involvement:

Table 7: Level of Lymph Node Involvement

Diagnosis	1	2	3	4	5	Total
Tbl	4	39	2	0	10	55
Acl	6	1	0	0	2	7
Cnl	1	4	2	0	3	9
Hodkins	0	1	0	0	3	9
Nhl	0	2	0	0	0	2
Msqcc	0	4	2	3	0	11
Madeno	0	0	0	2	0	2
Total	11	51	6	5	17	90

Tuberculosis: cervical lymph node alone were involved in 49 patients, cervical and axillary lymph nodes were involved in 6 patients. Examination of cervical lymph nodes revealed that multiple matted nodes in 16

cases, single discrete nodes were present in 30 cases, and multiple discrete nodes were seen in 9 cases. Most common group of lymph nodes involved were the upper deep jugular

followed by posterior trianglular group of lymph nodes, submandibular and submental groups.

Investigations

Hemoglobin percentage was near normal limit in the

majority of patients studied.

The patients admitted were also subjected for evaluation of total count and differential count, erythrocyte sedimentation rate.

Table 8: ESR

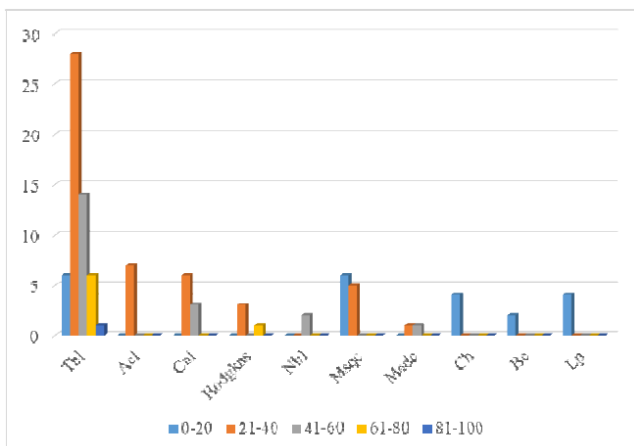
	0-20	21-40	41-60	61-80	81-100	Total
Tbl	6	28	14	6	1	55
Acl	0	7	0	0	0	7
Cnl	0	6	3	0	0	9
Hodgkins	0	3	0	1	0	4
Nhl	0	0	2	0	0	2
Msqc	6	5	0	0	0	11
Madc	0	1	1	0	0	2
Ch	4	0	0	0	0	4
Bc	2	0	0	0	0	2
Lp	4	0	0	0	0	4
Total	22	50	20	7	1	100

CC-0.652 P. Value-0.001

Erythrocyte sedimentation rate is said to be raised in chronic inflammatory disease, tuberculosis, lymphomas and secondary's in the neck.

Tuberculosis : 28 cases out of 55 patients showed rise in ESR between 21-40 mm/hour.

Graph 6: ESR



Diagnosis

AFB Sputum AFB was positive in only 14 patients out of 55 diagnosed cases of tubercular lymphadenitis.

Radiologically : 25 patients showed evidence of pulmonary Koch's, and 2 cases showed evidence of malignancy (cannon ball appearance).

Table 9: Chest X-ray

Diagnosis	Not done	TB	Malignancy	Normal	Total
Tbl	1	25	0	29	55
Acl	0	0	0	7	7
Cnl	0	0	0	9	9
Hodgkins	0	0	0	4	4
Nhl	0	0	0	2	2
Msqcc	0	0	2	9	11
Madeno	0	0	0	2	2
Ch	0	0	0	4	4
Be	0	0	0	2	2
Lp	0	0	0	4	4
Total	1	25	2	72	100

CC-0.553, p Value-0.020

FNAC: 75 out of 100 cases were subjected for FNAC. Among them 46 (TBL), 7 (acute lymphadenitis), 9 chronic nonspecific lymphadenitis, 11 metastatic squamous cell carcinoma, 2 metastatic adenocarcinoma..

In tuberculosis: 46(55) patients subjected for FNAC. Among them 42 were positive and 4 were negative, these negative patients underwent lymph node biopsy to confirm the diagnosis.

Acute lymphadentis: 7 patient underwent FNAC. All cases were diagnosed to have acute lymphadenitis. Out of 9 patients of chronic nonspecific lymphadenitis, 7 were diagnosed on FNAC, where as in 2 patients FNAC showed negative smear. These 2 patients were subjected for lymph node biopsy.

Secondaries in the neck (sq cell ca/adeno ca): 13 patient were subjected for FNAC. All patients were found to be having positive cytological diagnoses.

Lymph node biopsy : 21 patient out of 100 cases were subjected for lymph node biopsy and sent for HPE. Among them 13 were tuberculosis lymphadenitis, 2 were nonspecific lymphadenitis, 6 were lymphomas.

Medical Treatment

All diagnosed cases of tubercular lymphadenopathy were subjected for 6 months chemo- therapy.

Surgical Treatment

4 case of cystic hygroma, 2 case of branchial cysts, 4 cases of lipoma under went surgical excision. 4

patients underwent FND. Cases link lymphoma (6), secondaries in the neck (11) were referred to cancer center for chemo radiation. Complications and follow up. Complication were seen in only 6 patients having tuberculosis following lymph node biopsy. Abscess formation was observed in four cases. Wound infection was noticed in two cases.

All patients of tuberculosis were followed at monthly interval for six months during the course of chemotherapy and progress was assessed by clinical examination as well as ESR estimation.

DISCUSSION

In this study of 100 cases, tubercular lymphadenopathy predominated and accounted for 55%. Secondaries in the neck accounted for 13%, acute and non specific lymphadenitis 16%, lymphoma 6%, others 10%. Study conducted by A.K. Gupta et al in 1988, among 101 patients tubercular lymph node accounted for 50.49% (51), nonspecific for 20% (21) metastatic for 15.8% (16), lymphoma 7.1% (6), others 4.95% (5). Our study results almost matches with the results of study conducted by A.K. Gupta et al.

Tuberculosis

We encountered 55 patient, (55%) with cervical tubercular lymphadenopathy out of 100 patients. Commonest age group affected in our study was 5-20 years (41.8%) followed by 21-40 year (36.4%) was similar to the study done by Subramaniyam. The ratio of male to female in this study was 1:15, showing female predominance which is similar to that found by Dandapat et al (1:1.1)³¹ and Subramanyam .

Constitutional Symptoms

49.1% (27) presented with fever, 43.6% (24) cases presented with cough, 12.7% (7) presented with loss

of weight and 25.5% (4) cases came with loss of appetite. Patel Mehta observed weight loss in 77% and fever in 73% cases, similarly Dandapat et al ³¹ also noted weight loss in 85% and fever 40% of their patients, so in this respect our observation differ significantly from those of others. The most common group of lymph nodes affection in this study was that of upper deep jugular, this is similar to the finding of Dandapat et al.³¹ Associated lung involvement as detected by chest radiography was seen in (45.5%) (25 cases) the figure is similar to 40%-50% described in the text book "Clinical Tuberculosis". Fine needle aspiration cytology is a well established diagnostic tool in assessment of cervical masses. In our study we found it a very useful diagnostic tool to identify the patients of tuberculosis lymphadenopathy, accuracy rate was 91.0%. In our study, we successfully treated with short course chemotherapy with minimum six month period of follow up. No patient was found to have a recurrence of local or systemic disease.

Secondaries in the neck

In our study second most common cause for cervical lymph node enlargement is metastatic deposits from oropharyngeal carcinoma. It is well accepted that oropharyngeal SCC shows marked tendency for lymphatic spread, even at early stage. In our study 13 (13%) case were included 11 male patients and 2 female patient. Primary site of distribution for oropharyngeal squamous cell carcinoma in our study were tonsillar fossa 63%, post pharynx 27%, larynx 9.1%, which is almost similar to that found by Young Cheng et al in which primary sites were found to be tonsillar fossa 65%, base of the tongue 23%, post pharynx 20%, larynx 12%. In our study, no case was found to have any primary in the base of the tongue.

In our study most commonly involved lymph node level was II (upper deep jugular) and IV (posterior triangle group of lymph node). The study results were similar to that of Byers et al⁷⁴. Oropharyngeal SCC commonly spreads to levels II through IV rather than levels I through III. FNAC : 13 case of metastatic lymphnode (sq cell ca, met adeno 2) were subjected to FNAC and diagnostic accuracy was found to be 100%. This is in close agreement with 100 percent successful results obtained by Gupta SK et al.

Treatment: 4 patients underwent FND. Post operative period was uneventful.

Follow up: Out of 13 patients 11 patients were referred to cancer centre for chemoradiation. 2 patients who had underwent FND, were referred for further chemo radiation. 2 patients didn't turn for follow up.

Lymphoma: In our study of 6 cases of lymphomas, maximum distribution was observed in the age group of 41-60 that is 3 cases were noted. The usual presentation being painless lymphadenopathy, commonly in cervical region with typical rubbery feel in consistency.

Constitutional symptoms (commonly termed B symptoms) these include fever in 50% (3 cases), loss of weight in 66% (4 cases), loss of appetite 50% (3 cases). The is similar to study conducted by Lister et al. All patients underwent lymph node biopsy and diagnosis was confirmed by histo- pathological study and referred for further management.

In our study 4 cases (4%) cystic hygroma, 2 cases (2%) of branchial cyst were reported. These patients underwent surgery. No complications occurred

CONCLUSION

1. TB was found to be the most commonest cause of

cervical lymphadenitis and so as the commonest cause for neck swelling.

2. Most of the cases studied were belonged to lower socioeconomic class. If standards of living are improved the incidence of tuberculosis may decrease.
3. All the patients with tuberculosis did not show much constitutional symptoms, but few patients presented with fever, cough, loss of weight, loss of appetite.
4. The sex ratio in TB lymphadenitis was M; F (1:1,5) showing that female predominance and upper deep cervical group of lymph nodes were commonly affected.
5. Radiologically, majority of the patients usually did not show the evidence of pulmonary Koch's.
6. All patients of TB lymphadenitis showed good response to 6 months of chemotherapy.
7. Mantoux test was not employed. Sputum AFB was positive in few cases, however negative sputum AFB did not rule out the TB.
8. A part from the other investigation FNAC & histopathological examination were the main tools in diagnosis of the neck swellings.
9. Patient with secondaries in the neck and lymphoma were referred to oncology centers for chemoradiation.
10. Surgery played a role in other benign neck swellings like branchial cyst, cystic hygroma and lipoma 4 patients of secondaries in the neck underwent FND.

SUMMARY

- Tuberculosis was the commonest cause of lymphadenitis, accounting for 55% of patients. Other cases of cervical lymphadenopathy were

- metastatic 13%, Chronic nonspecific lymphadenitis 9%, lymphomas 6%, acute lymphadenitis 7%. Other swellings of the neck included in this study were cystic hygroma 4% branchial cyst 2%, lipoma 4%.
- The distribution of neck swelling in our study was observed commonly between the age group of 1 to 20 yrs (45%). Where as the distribution for the age group 21 to 40 yrs, 41 to 60, 61 to 80 were found to be 23%, 26%, and 6% respectively.
 - There were 50 male and 50 females (M: F ratio was 1:1). 79% belonged to the lower socioeconomic group. In tuberculosis male to female ratio was observed to be 1:1.5 with 87.3% of patients belonging to lower socioeconomic class.
 - The time interval between onset of symptoms and time of presentation varied from 1 to 36 months. The main complaint at the time of presentation was swelling in the neck, majority of the patients were having cervical lymphadenitis due to tuberculosis (55%), secondaries in the neck (13%), lymphomas (6), acute lymphadenitis (7%) and chronic nonspecific lymphadenitis (9%). Rest of the patients presented with neck swellings (10%) Cervical lymph nodes alone were involved in 90 patients (90%), Cervical and axillary lymph nodes were involved in 6 patients (6%). Examination of cervical lymph nodes revealed multiple matted nodes in 16 cases (17.7%), multiple discrete nodes were seen in 9(10%) cases, single discrete nodes were in 46 cases (55.1%), firm shotty lymph nodes were found in 6 cases (6.6%) and consistency was hard in 13 cases (14.4%).
 - Most common group of lymph node involvement was the upper deep jugular group, observed in 61 cases (67.7%), followed by posterior triangle group of lymph nodes seen in 17 cases (18.8%).
 - ESR was raised in almost all the patients. In 50% of the cases it was found to be elevated between the range of 21 to 40 mm / hr. chest X-ray showed evidence of pulmonary Koch's in 25 cases (45.5%) and in 2 cases features of malignancy were detected.
 - Fine needle aspiration cytology revealed a positive diagnosis in 69 out of 75 cases (92%). In some cases lymph node biopsy was needed to confirm the diagnosis.
 - Sputum for acid fast bacilli was positive in only 14 cases (25.5%) out of 55 diagnosed cases of T B lymphadenitis.
 - In 100 cases, 21 cases were subjected for lymph node biopsy and sent for histo pathological examination to confirm the diagnosis.
 - Patients with tuberculosis lymphadenitis showed good response to short course chemotherapy but the follow up was poor after completion of chemotherapy. Complication were seen in only 3 patients having tuberculosis following lymph node biopsy. Abscess formation was observed in one case, wound infection was noticed in two cases. Cystic hygroma & branchial cyst were noted in pediatric age group and underwent surgical excision with uneventful post operative period.
 - Secondaries in the neck & lymphomas were referred to cancer center for chemoradiation but those patients were lost for follow up.

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PROFORMA

“A CLINICOPATHOLOGICAL STUDY OF NECK SWELLINGS EXCLUDING THYROID”

Name OPD No. Age
IP No. Sex Unit Occupation
Diagnosis Address
Complaints
Swelling in Neck-location-Anterior/Posterior/RT/LT side/Bothside
Duration
Number
1. Pain 2. Fever
3. Cough 4. Wt Loss
5. Loss of appetite 6. Ulcer/Sinus
7. Dyspagia
H/O presenting complaints
1.
Swelling in neck
1. Duration 2. Pain
3. Course 4. Secondary changes
5. Swellings else where
II. Pain
1. Onset-before or after appearance of swelling
2. Duration 3. Site
4. Type of pain
Severity – Mild/Moderate/Severe
Aggravating factors/Relieving factors
III. Fever
1. Duration
2. Degree
3. Onset-before or after appearance of swelling
4. Type of fever-remittent/intermittent/periodic

5. Diurnal variation if yes more in evening- Yes/No.

IV. Cough

1. Dry/Productive 2. Hemoptysis-Yes/No

V. Weight Loss – Yes/No

VI. Loss of Appetite – Yes/No

Loss of weight – Yes/No

VII. Ulcer/Sinus-Yes/No

Any discharge-yes/no

Nature-purulent/serious/Hemorrhagic/Caseous

VIII. Any other complaints

1. Fever – Yes/No

Whether evening raise of temperature-yes/no

Exposure with case of tuberculosis-yes/no

Family History

Case of tuberculosis in family/relatives

Personal History Appetite Bowels Bladder

Treatment history

Drug therapy for tuberculosis-yes/no complete

General Physical examination

1. Appearance 2. Pallor

3. Build 4. Icterus

5. Nourishment 6. Pedal edema Yes/No

Vital data

1.

Pulse

2.

Temperature 3. B. P. Systemic

examination

1. R.S.

2. C.V.S

3. P.A.

4. C.N.S. Local examination

I. Inspection

1. Position 2. Number

3. Shape 4. Extent

5. Redness 6. Ulcer/Sinus-Yes/No

7. Surface-Smooth/Nodular/Irregular 8. Dilted Veins-
Yes/No.

9. Fluctuation-Yes/No.

Vertical/Horizontal/Both

10. Mobility-

11.

Plans of swelling

12. Relation to surrounding structures. III.

Oral examination

1. Gums 2. Teeth

3. Tongue 4. Buccal mucosa

IV. ENT Examination

Provisional diagnoses

V. Investigation

Blood urine

- HB sugar

- ESR ALB

- TC/DC Micro

VI. Radiological Study

- Chest X ray

VIII. Lymph node Biopsy & HPE

XI. Upper GI Endoscopy & Laryngoscopy

SILNO	IPOPNO	AGE	SEX	SES	SWELLING	DURATION IN MONTHS	PAIN	FEVER	COUGH	LOSS OF WEIGHT	LOSS OF APPETITE	PHT	LEVEL OF LYMPHNODE	LC	HEMOGLOBIN(%)	ESR	TOTAL COUNT	DIFFERENTIAL COUNT	SPUTUM AFB	CHEST XRAY	PAAC	HPK	LYMPHOIDE BDDPSY	DIAGNOSIS	MEDICAL TREATMENT	SURGICAL TREATMENT	RH	COMPLICATIONS				
1	34314	37	M	H	B	6	A	P	A	A	A	A	2	-	-	3	-	15	25	†	L	E	ND	NORMAL	-	NHL	D	NHL	ANTI	EX.ND	YES	NIL
2	1834	48	F	L	R	4	A	A	P	P	P	A	2	5	-	2	3	12	29	N	L	-	P	TB	-	TBL	D	TBL	ATT	EX.ND	NO	NIL
3	10465	62	M	L	B	6	P	P	A	P	P	A	5	-	-	1	-	8	22	N	L	-	ND	NORMAL	-	HODG	D	HODG	ANTI	EX.ND	YES	NIL
4	22345	25	F	H	L	3	A	A	P	A	A	A	2	5	-	4	-	13	11	N	L	-	P	TB	-	TBL	D	TBL	ATT	EX	NO	ABSC
5	5689	12	M	L	L	3	A	A	P	P	P	A	2	-	-	2	3	12	20	N	NE	L	P	TB	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
6	25917	8	F	L	R	4	A	A	P	A	A	A	5	-	-	2	-	14	22	N	L	-	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
7	2855	26	F	L	R	4	A	A	A	A	P	A	2	-	-	2	-	13	40	N	L	-	ND	TB	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
8	23345	50	F	H	R	6	A	A	A	P	P	A	2	-	-	2	-	12	22	N	L	-	ND	NORMAL	-	NHL	D	NHL	ANTI	EX.ND	YES	NIL
9	23480	18	M	L	R	4	A	P	A	A	A	A	5	-	-	2	-	12	70	†	L	-	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
10	29877	67	F	L	B	2	A	P	A	A	A	A	3	5	-	2	-	9	9	N	N	-	A	NORMAL	MSQC	-	ND	MSQC	ANTI	FND	NO	NIL
11	23757	50	M	L	R	6	A	A	A	A	A	A	2	-	-	4	-	10	16	N	N	-	ND	NORMAL	-	-	ND	LP	ANTI	EX	NO	NIL
12	20508	54	M	L	L	6	A	A	A	P	P	A	4	-	-	2	-	13	20	N	N	-	ND	TB	TB	-	ND	TB	ATT	EX.ND	YES	NIL
13	28282	45	M	L	L	5	A	A	A	P	A	A	4	-	-	2	-	13	60	N	N	-	ND	NORMAL	ADCA	-	ND	MADE	ANTI	EX.ND	YES	NIL
14	1149	79	F	H	L	6	A	A	A	A	A	A	4	-	-	2	-	12	60	N	N	-	ND	NORMAL	CNL	-	ND	CNL	ANTI	EX.ND	YES	NIL
15	21963	72	M	H	R	6	A	A	A	P	A	A	5	-	-	5	-	16	32	M	NE	-	ND	NORMAL	CNL	-	ND	CNL	ANTI	EX.ND	YES	NIL
16	28543	48	M	H	L	5	A	A	P	A	A	A	3	-	-	5	-	12	10	N	N	-	ND	NORMAL	SQ.CC	-	ND	M.SQC	ANTI	EX.ND	YES	NIL
17	2576	7	M	H	R	2	A	P	P	A	A	A	5	-	-	4	-	16	30	N	L	-	A	TB	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
18	23715	46	F	L	B	5	A	P	P	A	P	A	2	3	5	2	3	8	90	†	L	-	P	TB	TBL	TBL	D	TBL	ANTI	EX	NO	NIL
19	23782	62	M	L	L	5	A	A	A	P	A	A	2	-	-	5	-	14	20	N	N	-	ND	NORMAL	CNL	-	ND	ACL	ANTI	EX.ND	YES	NIL
20	21965	11	M	H	R	3	A	P	A	A	A	A	1	2	-	2	-	14	15	†	NE	L	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL

21	1800	62	M	L	L	6	A	A	A	P	A	A	2	-	-	6	-	13	30	†	N	-	ND	NORMAL	SQ.CC	-	ND	M.SQC	ANTI	FND	YES	NIL
22	27860	54	M	L	R	4	A	A	P	A	A	A	2	-	-	2	-	10	28	N	NE	L	P	TB	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
23	29250	43	F	H	R	5	A	A	A	A	A	A	2	-	-	5	-	12	29	N	N	-	ND	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	YES	NIL
24	18566	31	M	L	R	5	A	A	A	A	P	A	2	-	-	5	-	11	22	†	N	-	ND	TB	TBL	-	ND	TBL	ATT	EX.ND	YES	NIL
25	28967	5	F	H	L	2	A	A	P	A	A	A	5	-	-	2	-	9	42	N	NE	L	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
26	2556	13	F	L	B	6	A	A	A	A	A	A	2	-	-	2	-	9	40	N	N	-	ND	NORMAL	-	BC	ND	CH	ANTI	EX	NO	NIL
27	30081	55	M	L	R	6	A	A	A	P	P	A	4	-	-	1	-	10	26	N	N	-	ND	NORMAL	CNL	-	ND	ACL	ANTI	EX.ND	YES	NIL
28	2646	45	M	L	L	2	A	A	A	A	P	A	2	-	-	4	-	12	22	N	L	-	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
29	25599	17	F	L	L	6	A	A	A	A	A	A	2	-	-	2	-	14	23	N	N	-	ND	NORMAL	CNL	-	ND	CNL	ANTI	EX.ND	NO	NIL
30	24008	49	M	L	B	2	A	P	A	A	A	A	3	5	-	2	-	14	28	N	N	-	A	TB	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
31	10737	12	F	L	L	5	A	A	A	A	A	A	2	-	-	2	-	16	25	N	N	-	A	NORMAL	CNL	-	ND	CNL	ANTI	EX.ND	NO	NIL
32	2776	34	F	L	R	3	A	P	P	A	P	P	2	5	-	2	3	12	31	N	N	-	P	TB	-	TBL	D	TBL	ATT	EX.ND	NO	NIL
33	28688	39	M	L	L	6	A	A	A	A	A	A	3	-	-	5	-	11	30	N	N	-	ND	NORMAL	SQ.CC	-	ND	M.SQC	ANTI	FND	YES	NIL
34	23805	16	F	L	R	4	A	A	A	A	A	A	2	-	-	2	3	12	30	N	N	-	A	NORMAL	CNL	-	ND	CNL	ANTI	EX.ND	NO	NIL
35	25244	47	F	L	R	4	A	A	P	A	P	A	2	5	-	2	-	13	23	†	L	-	P	TB	-	TBL	D	TBL	ATT	EX.ND	NO	NIL
36	212925	10	M	H	R	6	A	A	A	A	A	A	2	-	-	4	-	14	10	N	N	-	ND	NORMAL	CNL	-	ND	ACL	ANTI	EX.ND	NO	NIL
37	290960	53	M	L	L	6	A	A	A	P	P	A	4	-	-	2	3	15	60	N	N	-	ND	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	YES	NIL
38	2918	38	F	L	R	4	A	P	A	P	P	A	2	3	-	2	-	9	10	N	L	-	P	TB	-	TBL	D	TBL	ATT	EX	NO	NIL
39	1923	21	M	L	L	3	A	A	P	P	P	A	2	-	-	2	3	12	20	N	N	L	P	TB	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
40	1431	47	F	L	B	5	A	P	P	A	P	A	2	3	5	2	3	8	90	†	L	-	P	TB	TBL	TBL	D	TBL	ATT	EX	NO	NIL

SILNO	IPORNO	AGE	SEX	SES	SWELLING	DURATION IN MONTHS	PAIN	FEVER	COUGH	LOSS OF WEIGHT	LOSS OF APPETITE	FHT	LEVEL OF LYMPHOIDE	L.C	HEMOGLOBIN(%)	ESR	TOTAL COUNT	DIFFERENTIAL COUNT	SPUTUM AFB	CHEST XRAY	FNAC	HPE	LYMPHOIDE BIOPSY	DIAGNOSIS	MEDICAL TREATMENT	SURGICAL TREATMENT	RE	COMPLICATIONS
41	18578	20	F	L	R	4	A	P	P	P	P	P	2 5 -	2 3	14	20	↑	L -	P	TB	TBL	TBL	D	TBL	ATT	EX.ND	NO	ABSC
42	22567	63	M	L	B	6	A	P	A	P	P	A	5 - -	1 -	14	15	N	N -	ND	NORMAL	SQC	MSQC	D	MSQC	ANTI	FND	YES	NIL
43	31362	9	F	H	L	2	A	P	P	A	A	P	2 - -	2 -	13	5	N	N -	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
44	7637	10	M	L	R	3	A	A	P	A	A	A	5 - -	4 -	13	20	N	L -	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
45	11023	8	M	L	R	2	A	P	A	A	A	A	2 - -	2 -	12	10	N	L -	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
46	28177	19	M	H	L	4	A	A	A	A	A	A	2 - -	2 -	10	4	N	N -	ND	NORMAL	-	CH	ND	CH	ANTI	EX	NO	NIL
47	1504	17	F	L	R	2	A	P	P	A	A	P	1 2 -	2 -	11	16	↑	L -	A	NORMAL	LP	-	ND	LP	ANTI	EX.ND	NO	NIL
48	1018	28	M	L	R	4	A	P	P	A	P	A	2 - -	2 -	11	20	↑	L -	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
49	2978	40	M	L	R	5	P	A	A	A	A	A	5 - -	3 -	12	27	N	L E	ND	NORMAL	-	TBL	D	TBL	ATT	EX.ND	YES	NIL
50	28073	7	F	H	L	1	A	A	A	A	A	A	2 - -	1 -	10	10	N	N -	ND	NORMAL	-	BC	ND	CH	ANTI	EX.ND	YES	NIL
51	28314	38	M	H	B	6	A	P	A	A	A	A	2 - -	3 -	15	25	↑	L E	ND	NORMAL	-	NHL	D	NHL	ANTI	EX.ND	YES	NIL
52	18194	45	F	L	R	4	A	A	P	P	P	A	2 5 -	2 3	12	29	N	L -	P	NORMAL	LP	LP	D	LP	ANTI	EX.ND	NO	NIL
53	280453	69	M	L	B	6	P	P	A	P	P	A	5 - -	1 -	8	22	N	L -	ND	NORMAL	-	HODG	D	HODG	ANTI	EX.ND	YES	NIL
54	11857	23	F	H	L	3	A	A	P	A	A	A	2 5 -	4 -	13	11	N	L -	P	TB	-	TBL	D	TBL	ATT	EX	NO	ABSC
55	57733	13	M	L	L	3	A	A	P	P	P	A	2 - -	2 3	12	20	N	NE L	P	TB	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
56	17917	5	F	L	R	4	A	A	P	A	A	A	5 - -	2 -	14	22	N	L -	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
57	60155	29	F	L	R	4	A	A	A	A	P	A	2 - -	2 -	13	40	N	L -	ND	TB	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
58	10545	52	F	H	R	6	A	A	A	P	P	A	2 - -	2 -	12	22	N	L -	ND	NORMAL	-	NHL	D	NHL	ANTI	EX.ND	YES	NIL
59	2590	19	M	L	R	4	A	P	A	A	A	A	5 - -	2 -	12	70	↑	L -	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
60	298677	69	F	L	B	2	A	P	A	A	A	A	3 5 -	2 -	9	9	N	N -	A	TB	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL

SILNO	IPORNO	AGE	SEX	SES	SWELLING	DURATION IN MONTHS	PAIN	FEVER	COUGH	LOSS OF WEIGHT	LOSS OF APPETITE	FHT	LEVEL OF LYMPHOIDE	L.C	HEMOGLOBIN(%)	ESR	TOTAL COUNT	DIFFERENTIAL COUNT	SPUTUM AFB	CHEST XRAY	FNAC	HPE	LYMPHOIDE BIOPSY	DIAGNOSIS	MEDICAL TREATMENT	SURGICAL TREATMENT	RE	COMPLICATIONS	
61	137577	52	M	L	R	6	A	A	A	A	A	A	2 - -	4 -	10	16	N	N -	ND	NORMAL	-	-	ND	LP	ANTI	EX	NO	NIL	
62	312050	50	M	L	L	6	A	A	A	P	P	A	4 - -	2 -	13	20	N	N -	ND	TB	TB	-	ND	TB	ATT	EX.ND	YES	NIL	
63	21428	46	M	L	L	5	A	A	A	P	A	A	4 - -	2 -	13	60	N	N -	ND	NORMAL	ADCA	-	ND	MADE	ANTI	EX.ND	YES	NIL	
64	281402	80	F	H	L	6	A	A	A	A	A	A	4 - -	2 -	12	60	N	N -	ND	NORMAL	CNL	-	ND	ACL	ANTI	EX.ND	YES	NIL	
65	83406	70	M	H	R	6	A	A	A	P	A	A	5 - -	5 -	16	32	M	NE	-	ND	NORMAL	CNL	-	ND	CNL	ANTI	EX.ND	YES	NIL
66	281433	50	M	H	L	5	A	A	P	A	A	A	3 - -	5 -	12	10	N	N -	ND	NORMAL	SQ.CC	-	ND	MSQC	ANTI	EX.ND	YES	NIL	
67	11576	8	M	H	R	2	A	P	P	A	A	A	5 - -	4 -	16	30	N	L -	A	TB	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL	
68	49715	45	F	L	B	5	A	P	P	A	P	A	2 3 5	2 3	8	90	↑	L -	P	TB	TBL	TBL	D	TBL	ANTI	EX	NO	NIL	
69	23682	60	M	L	L	5	A	A	A	P	A	A	2 - -	5 -	14	20	N	N -	ND	NORMAL	CNL	-	ND	CNL	ANTI	EX.ND	YES	NIL	
70	300365	12	M	H	R	3	A	P	A	A	A	A	1 2 -	2 -	14	15	↑	NE L	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL	
71	487400	60	M	L	L	6	A	A	A	P	A	A	2 - -	6 -	13	30	↑	N -	ND	NORMAL	SQ.CC	-	ND	MSQC	ANTI	FND	YES	NIL	
72	307860	50	M	L	R	4	A	A	P	A	A	A	2 - -	2 -	10	28	N	NE L	P	TB	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL	
73	292950	40	F	H	R	5	A	A	A	A	A	A	2 - -	5 -	12	29	N	N -	ND	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	YES	NIL	
74	71566	30	M	L	R	5	A	A	A	A	P	A	2 - -	5 -	11	22	↑	N -	ND	NORMAL	MSQC	-	ND	MSQC	ANTI	EX.ND	YES	NIL	
75	28967	6	F	H	L	2	A	A	P	A	A	A	5 - -	2 -	9	42	N	NE L	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL	
76	24556	12	F	L	B	6	A	A	A	A	A	A	2 - -	2 -	9	40	N	N -	ND	NORMAL	-	BC	ND	BC	ANTI	EX	NO	NIL	
77	30061	50	M	L	R	6	A	A	A	P	P	A	4 - -	1 -	10	26	N	N -	ND	NORMAL	CNL	-	ND	ACL	ANTI	EX.ND	YES	NIL	
78	7646	40	M	L	L	2	A	A	A	A	P	A	2 - -	4 -	12	22	N	L -	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL	
79	29599	14	F	L	L	6	A	A	A	A	A	A	2 - -	2 -	14	23	N	N -	ND	NORMAL	CNL	-	ND	CNL	ANTI	EX.ND	NO	NIL	
80	294008	40	M	L	B	2	A	P	A	A	A	A	3 5 -	2 -	14	28	N	N -	A	TB	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL	

SILNO	IPVOPAO	AGE	SEX	SES	SWELLING	DURATION IN MONTHS	PAIN	FEVER	COUGH	LOSS OF WEIGHT	LOSS OF APPETITE	FHT	LEVEL OF LYMPHNODE		LC	HEMOGLOBIN(%)	ESR	TOTAL COUNT	DIFFERENTIAL COUNT	SPUTUM AFB	CHEST XRAY	FNAC	HPE	LYMPHNODE BIOPSY	DIAGNOSIS	MEDICAL TREATMENT	SURGICAL TREATMENT	RH	COMPLICATIONS	
81	10737	10	F	L	L	5	A	A	A	A	A	A	2	-	-	2	25	N	N	-	A	NORMAL	CNL	-	ND	CNL	ANTI	EX.ND	NO	NIL
82	298776	30	F	L	R	3	A	P	P	A	P	P	2	5	-	2	31	N	N	-	P	TB	-	TBL	D	TBL	ATT	EX.ND	NO	NIL
83	28688	30	M	L	L	6	A	A	A	A	A	A	3	-	-	5	30	N	N	-	ND	NORMAL	SQCC	-	ND	M.SQC	ANTI	EX.ND	YES	NIL
84	213805	14	F	L	R	4	A	A	A	A	A	A	2	-	-	2	30	N	N	-	A	NORMAL	CNL	-	ND	ACL	ANTI	EX.ND	NO	NIL
85	15244	40	F	L	R	4	A	A	P	A	P	A	2	5	-	2	23	↑	L	-	P	TB	-	TBL	D	TBL	ATT	EX.ND	NO	NIL
86	312925	16	M	H	R	6	A	A	A	A	A	A	2	-	-	4	10	N	N	-	ND	NORMAL	CNL	-	ND	CNL	ANTI	EX.ND	NO	NIL
87	290960	55	M	L	L	6	A	A	A	P	P	A	4	-	-	2	60	N	N	-	ND	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	YES	NIL
88	299118	30	F	L	R	4	A	P	A	P	P	A	2	3	-	2	10	N	L	-	P	TB	-	TBL	D	TBL	ATT	EX	NO	NIL
89	38923	19	M	L	L	3	A	A	P	P	P	A	2	-	-	2	20	N	N	L	P	TB	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
90	304231	45	F	L	B	5	A	P	P	A	P	A	2	3	5	2	20	N	L	-	P	NORMAL	MSQC	-	D	MSQC	ANTI	EX.ND	NO	NIL
91	28578	24	F	L	R	4	A	P	P	P	P	P	2	5	-	2	20	↑	L	-	P	TB	TBL	TBL	D	TBL	ATT	EX.ND	NO	ABSC
92	282567	60	M	L	B	6	A	P	A	P	P	A	5	-	-	1	15	N	N	-	ND	NORMAL	-	TBL	D	TBL	ATT	EX.ND	YES	NIL
93	311362	7	F	H	L	2	A	P	P	A	A	P	2	-	-	2	5	N	N	-	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
94	7637	12	M	L	R	3	A	A	P	A	A	A	5	-	-	4	20	N	L	-	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
95	78023	5	M	L	R	2	A	P	A	A	A	A	2	-	-	2	10	N	L	-	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
96	281177	15	M	H	L	4	A	A	A	A	A	A	2	-	-	2	4	N	N	-	ND	NORMAL	-	CH	ND	CH	ANTI	EX	NO	NIL
97	29504	12	F	L	R	2	A	P	P	A	A	P	1	2	-	2	16	↑	L	-	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
98	101	25	M	L	R	4	A	P	P	A	P	A	2	-	-	2	20	↑	L	-	A	NORMAL	TBL	-	ND	TBL	ATT	EX.ND	NO	NIL
99	283078	42	M	L	R	5	P	A	A	A	A	A	5	-	-	3	27	N	L	E	ND	NORMAL	-	TBL	D	TBL	ATT	EX.ND	YES	NIL
100	280453	8	F	H	L	1	A	A	A	A	A	A	2	-	-	1	10	N	N	-	ND	NORMAL	-	BC	ND	BC	ANTI	EX.ND	YES	NIL

KEY TO MASTER CHART

- M - Male
- F - Female
- SES - Socio economic status
- R - Right L -
- Left B - Both
- FR - Front
- P - Present
- A - Absent
- FHT - Family History of Tuberculosis
- D - Done
- ND - Not Done
- LC - Lymphnode consistency
- TB - Tuberculosis
- MALG - Malignancy
- ESR - Erythrocyte sedimentation rate
- - Increased
- N - Normal
- Ne - Neutrophilia
- L - Lymphocytosis
- E – Eosinophilia
- AFB - Acid fast bacilli
- FNAC - Fine Needle Aspiration cytology
- HPE - Histopathological examination
- TBL - Tubercular lymphadenitis
- ACL - Acute lymphadenitis
- CNL - Chronic lymphadenitis
- HODG - Hodgkins lymphoma
- NHL - Non hodgkins lymphoma
- M.SQCC - Metastatic Squamous cell carcinoma
- Made - Metastatic adeno carcinoma
- CH - Cystic hygroa
- BC - Branchial cyst

LP - Lipoma

ANTI - Antibiotics

ATT - Anti Tubercular Treatment

EX - Excision

EXND - Excision not done

FND - Functional Neck dissection

ABSC - Abscess

WI - Wound infection

RH - Referred to higher center