"CLINICAL STUDY ON TUBERCULOUS CERVICAL LYMPHADENOPATHY"

Dissertation submitted

To

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI

With partial fulfilment of the regulations for the award of the degree of

M.S (General Surgery)

Branch-I

DEPARTMENT OF GENERAL SURGERY

Government Kilpauk Medical College

Chennai- 600010

April -2015

GOVT KILPAUK MEDICAL COLLEGE

CHENNAI

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled "**CLINICAL STUDY ON TUBERCULOUS CLINICAL LYMPHADENOPATHY**" is a bonafide and genuine research work carried out by me under the guidance of **Prof. P. N. SHANMUGASUNDARAM M.S.**, Department of General Surgery, Govt Kilpauk Medical College and Hospital, Chennai-10.

This dissertation is submitted to **THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY CHENNAI** in partial fulfilment of the degree of M.S. General Surgery examination to be held in **April 2015**.

DATE:

PLACE: **DR G. PRAMMARAJ @ SUBRAMANIAN**

CERTIFICATE BY THE GUIDE

This is to certify that this dissertation is the bonafide work of

DR G. PRAMMARAJ @ SUBRAMANIAN

On

"CLINICAL STUDY ON TUBERCULOUS CERVICAL LYMPHADENOPATHY"

During his course in M.S. General Surgery from August 2013 to August 2014 at Government Kilpauk Medical College and Hospital, Chennai-10.

DATE:

PLACE:

Prof. P. N. SHANMUGASUNDARAM M.S.

Professor and Head of the Department

Department of General Surgery

Govt Kilpauk medical college and hospital

Chennai - 600010

ENDORSEMENT BY THE HOD,

DEAN/ HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"CLINICAL STUDY ON CERVICAL TUBERCULOUS LYMPHADENOPATHY''** is a bonafide research work done by **Dr G. Prammaraj @ Subramanian**, Post Graduate in M.S. General Surgery, Kilpauk Medical College, Chennai-10 under my direct guidance and supervision in my satisfaction, in partial fulfilment of the requirements for the degree of **M.S. General Surgery**.

Prof P. N. SHANMUGASUNDARAM M.S.
PROFESSOR & H. O.D
DEPARTMENT OF GENERAL SURGERY
GOVT KILPAUK MEDICAL COLLEGE,
CHENNAI – 600010.

Prof. N. GUNASEKARAN M.D. D. T. C. D.
DEAN/ HEAD OF THE INSTITUTION
GOVT KILPAUK MEDICAL COLLEGE,
CHENNAI – 600010.

DATE:

PLACE:

DATE:

PLACE:

ACKNOWLEDGEMENT

I would like to begin by thanking the ALMIGHTY GOD for the things he has bestowed upon me.

I would like to thank my parents for making me who I am today and for their love, support and encouragement.

I take this opportunity to thank everyone who made this dissertation possible.

I express my sincere thanks to **Prof**.**N.GUNASEKARAN M.D, D.T.C.D** DEAN, Kilpauk Medical College and Hospital for giving me the opportunity to conduct this study in the Department of General Surgery, Government Kilpauk Medical College Hospital, Kilpauk Medical College, Chennai -10.

My deepest gratitude to my guide and mentor **Prof.P.N. SHANMUGASUNDARAM M.S.**, Department of General Surgery, Kilpauk Medical College, for his invaluable guidance during my training as a post graduate student and for guiding me with his professional expertise.

My special thanks to **Dr. VIJAYLAKSHMI M.S,Dr. K.SRIDEVI M.S. Dr. P. MATHUSOOTHANAN M.S.** for their valuable suggestions and guidance.

This study would have not been possible without the support of my fellow post graduates and interns who had been a valuable support.

My sincere thanks to the laboratory technicians for their help during the preparation of this dissertation. My sincere thanks to the Thoracic Medicine Centre in KMCH and National Tuberculosis Research Institute, Chetpet, Chennai.

The most important part of any medical research is patients. I owe great deal of gratitude to each and every one of them for their willingness and

co – operation during my study.

DATE:

PLACE: **Dr. G. PRAMMARAJ @ SUBRAMANIAN**

LIST OF ABBREVIATIONS USED

AFB : Acid Fast Bacilli

ATT : Anti-tubercular treatment

BCG : Bacille Calmette-Guerin

CG/NCG : Caseating granuloma/ Non caseating granuloma

DC : Differential count

DOTS : Directly observed treatment short course

ESR : Erythrocyte Sedimentation Rate

FA-ABS :Fluoroscent treponemal antibody absorption

FIG : Figure

FNAC : Fine Needle Aspiration Cytology

GMS : Gomori methenamine silver

INH : Isoniazid

MDR-TB: Multi-drug resistant tuberculosis

MOTT : Mycobacteria other than tubercle

SCL : Supraclavicular

SM : Submandibular

LDC : Lower Deep Cervical

RNTCP : Revised National Tuberculosis Control Programme

SES : Socio-economic status

UDC : Upper deep cervical

WBC : White Blood Cell

Z : Pyrazinamide

ZN : Ziehl Neelson

ABSTRACT

BACKGROUND

Tuberculosis because of its increasing prevalence continues to be the major burden in our country. Inspite of the advanced studies performed in the field of medicine, it continues to be a major burden. Pulmonary tuberculosis is most common. Among the extra-pulmonary infections, lymph node involvement is the most common. It is the most common cause of lymphadenopathy in the developing countries. Various modes of treatment are available which includes radiation, chemotherapy and antibiotics.

AIMS AND OBJECTIVES

- To study about the incidence of tuberculous cervical lymphadenopathy.
- To study about the clinical presentations (signs, symptoms) of tuberculous cervical lymphadenopathy.
- To correlate clinical diagnosis with the histopathological findings of tuberculous cervical lymphadenopathy and to interpret the results.
- To study about the clinical work-up and the various management options, their outcome and to follow up the clinical behaviour and improvement in the course of tuberculous cervical lymphadenopathy for a period of not more than 6 months.

MATERIALS AND METHODS

The prospective study was conducted on 100 cases presenting to the outpatient department of GOVT.KILPAUK MEDICAL COLLEGE AND HOSPITAL with the signs and symptoms suggestive of tuberculous cervical lymphadenitis out of which 70 cases proved to be positive for tuberculous cervical lymphadenopathy. This study was conducted on patients presenting to the OPD during the period of Aug 2013 to Aug 2014. This study was conducted by collecting data from individual patients in the form of pretested proforma. All the patients were exposed to FNAC/Excision Biopsy and were started on Anti – tuberculous treatment only after confirming the diagnosis based on the clinical findings and the histopathological reports.

RESULTS

In our present series, the incidence of tuberculous cervical lymphadenopathy was maximum in the age group of 21-30 years (35 cases-50%). The disease showed an increasing incidence in females as compared to the male patients, with the female to male ratio being 1.5:1. It is more common in the lower socio-economic status people (90%). The disease shows increased incidence among the rural population which accounts for about 70% of the cases. All the 70 cases presented with the complaint of swelling in the neck. 14 cases presented with fever, 7 cases each presented with pain, weight loss and cough respectively. About 70% of the cases presented within 3 months of the

start of the symptoms. The left side nodes were most commonly involved (57.1%). The lymph node involvement was discrete in 77.1% and 22.9% presented with matted lymph nodes according to the present study. The lymph nodes were firm in 70% of the cases and soft in the remaining 30%. The upper deep cervical nodes were involved in 60% of the cases, which was the most common lymph node group involved in tuberculous cervical lymphadenopathy in previous studies also.

57.1% % of the cases presented with raised ESR in the present study. 8.6% of the cases had features suggestive of pulmonary tuberculosis on chest x-ray. 82.9% of the cases showed caseating granuloma on FNAC. FNAC was inconclusive in 17.1% of the cases which were then subsequently subjected to Excision Biopsy which proved positive for tuberculous cervical lymphadenopathy. Tuberculin test was positive in 27.1% of the cases.

The patients were then categorised as Category I or Category II patients and were put on Anti-tubercular treatment based on the histopathological (FNAC/Excision Biopsy) reports and were followed up for a period of 6 months. 65 cases were symptom free at the end of the study.

INTERPRETATION AND CORRELATION

According to the present study, tuberculous cervical lymphadenopathy was more common among the female population with an increasing incidence in the

21-30 years of age. Swelling in the neck was the most common presenting complaint followed by fever, pain, weight loss and cough. Upper deep cervical group of nodes were the most commonly involved and most of the nodes were discrete and firm in consistency. All the patients were subjected to histopathological examination (FNAC/Excision Biopsy) and were started on ATT based on the reports and as per the RNTCP guidelines. All the patients had a 100% cure rate at the end of the study which included the follow up period of 6 months. Surgical treatments are only adjunctive to chemotherapy and not a replacement.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

LIST OF GRAPHS

INTRODUCTION

Tuberculosis remains to be the major health burden primarily affecting the lower socio-economic status people in our country.

In tuberculosis, the commonest type of infection is pulmonary tuberculosis. In extra pulmonary tuberculosis, lymph node involvement is the most common presentation. Cervical lymph nodes are affected frequently. Peripheral and mediastinal nodes are seen in immunocompromised population like HIV. [1]

The general hospitals witness a staggering 2-3% of patients suffering from glandular tuberculosis. It also leaves a permanent scar, often in parts of body that are exposed ,affecting the general confidence of the patients. The treatment modality for TB have changed from time to time , ranging from royal touch to chemotherapy, antibiotics and surgery.

My study focussed on 70 patients with cervical lymph node during a period of one year from August 2013 to August 2014.

AIMS AND OBJECTIVES

- To study about the incidence of tuberculous cervical lymphadenopathy.
- To study about the clinical presentations (signs, symptoms) of tuberculous cervical lymphadenopathy.
- To correlate clinical diagnosis with the histopathological findings of tuberculous cervical lymphadenopathy and to interpret the results.
- To study about the clinical work-up and the various management options, their outcome and to follow up the clinical behaviour and improvement in the course of tuberculous cervical lymphadenopathy for a period of not more than 6 months.

REVIEW OF LITERATURE

HISTORY

There are evidences showing that ancient Indians like Manu and Sushrutha have described tuberculous lymphadenopathy is a disease of the unclean.

Hippocrates also described tuberculosis as ‘phthisis’ meaning ‘I am wasting’. [2]

The old name for cervical tuberculous lymphadenitis is scrofula, meaning ‘glandular swelling’ in Latin. In French it is referred as ‘full necked sow’ because of its resemblance with pig’s neck.

In early days it was thought that the king’s touch can cure the disease popularly known as ‘royal touch’. There was also a saying dated back to 496 AD by a king ‘I touch thee, god heals thee’. [3]

The royal touch was finally abandoned after being practised for a period of 7 years by the English and 13 years by the French kings and queens. The most popular theory put forward before 1886 was by Dole boe. He proposed ‘THEORY OF OBSTRUCTION’, which states that it is made up of enlargement of numerous small glands made visible by obstruction. Large caseating glands are produced by the fusion of multiple small glands.

Sylvius coined the term ‘Tubercle’ following his serial observations in post mortem examinations to the typical nodular lesions. Laennec proved that tuberculosis can also occur in various parts of the body similarly by 2000 post mortem examinations.

The infectious nature of tuberculosis was recognized in early days, only to be proved experimentally by Villemin in 1865 by injecting the tubercle material into a rabbit. [(2)] Koch discovered the causative organism of tuberculosis as ‘tubercle bacilli’ in march 24 ,1882.

France became the first country to introduce the B.C.G. vaccination in 1921. In developed countries, better economy, health education and awareness reduced the incidence of tuberculosis.

The advent of chemotherapy revolutionised the treatment for tuberculosis.[(3)] The milestones in the development of chemotherapy [(2)] are.,

1.1943- Streptomycin discovered by WALKSMAN.

2.1946-PAS discovered by LEHMANN.

3.1952-Isoniazid discovered by JACK BERNSTEIN ET AL.

4.1966- the discovery of Rifampicin.

EMBRYOLOGY, ANATOMY AND PHYSIOLOGY OF LYMPH NODES

In embryo, at the end of 5th week the lymphatic system begins to develop. There are two theories describing the development of the lymphatic system. First theory was introduced by SABIN in 1902 which describes the lymphatic System development as a diverticulae of the endothelium of vein. Another

Theory was introduced by HUNTINGTON & MCCLURE in 1909 regarding the sprouting of endothelium in mesenchymal cleft which fuse and form the lymphatic system.

In embryo, at the end of 6th week, local dilatations of the lymphatic channels form 6 primary lymph sacs

1. Jugular lymph sacs are two in number which are present at the junction of the subclavian vein with the anterior cardinal vein.

2. Iliac lymph sacs are two in number and present at the junction of the iliac vein with the posterior cardinal vein.

3. Retroperitoneal lymph sac which is one in number present in the root of the mesentery on the posterior abdominal wall.

4. At the level of the adrenal glands and posterior to the retroperitoneal lymph sac [4] is situated the cistern chyli.

Lymph vessels to the head, neck and upper limbs develop from the jugular sacs

Lymph vessels to the lower limbs develop from the iliac sacs .

Lymph vessels to the gut develop from the retroperitoneal and cisternal sacs.

Right and left thoracic duct develop from the union of the cisterna chyli and the two jugular sacs. These ducts join the venous system near the angle of subclavian and internal jugular vein at base of the neck

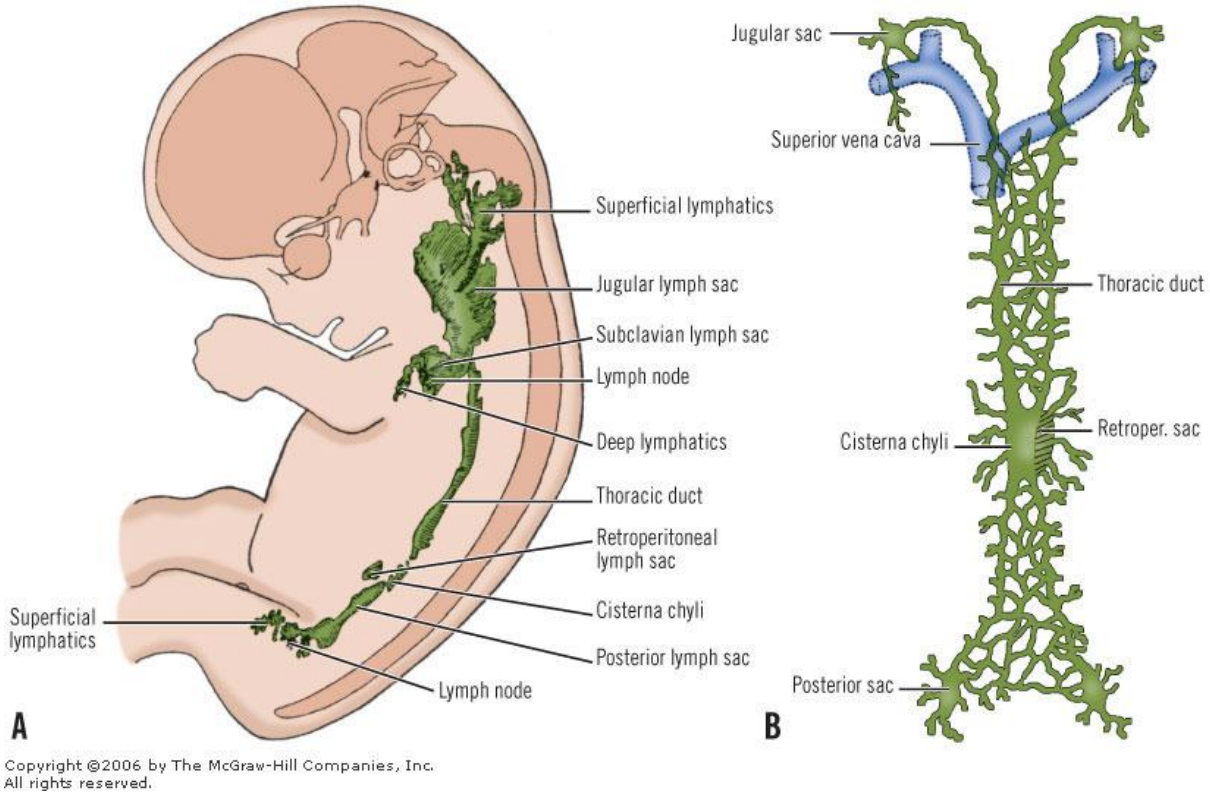


Fig 1 – Embryology of the lymph node

Lymph node develops from lymph sacs which are invaded by the mesenchymal cells to form the lymphatic sinuses or channels. Germinal lymphocyte production does not occur in the lymph node just before or after birth whereas the lymphocyte present in the node before birth develops from the thymus.

LYMPH NODE

It has two parts: the medulla and cortex. Medulla consists of network of minute lymphatic channels whereas the cortex contains lymphoid follicle and reticular fibres. In the medulla, afferent lymphatics are filtered and collected in the hilum by efferent lymphatics. Cortex mostly encircles the lymph node except in the hilum. The lymphnodes are many in number in the neck, axilla, mediastinum, mesentery, posterior abdominal wall and the pelvis.

It consists of the capsule, trabeculae and the reticular tissue with the cells embedded within it.

CAPSULE AND TRABECULAE

The capsule is made up of collagen fibres, elastin fibres and few fibroblasts. It encircles the node completely and extends into its contents dividing it into small compartments. Those extensions are termed the trabeculae and they resemble in structure with that of the capsule and form the supporting tissue of the node. [6]

The afferent lymphatics drain into the sub capsular lymphatic plexus. The reticulum consists of reticular cells and fibres.

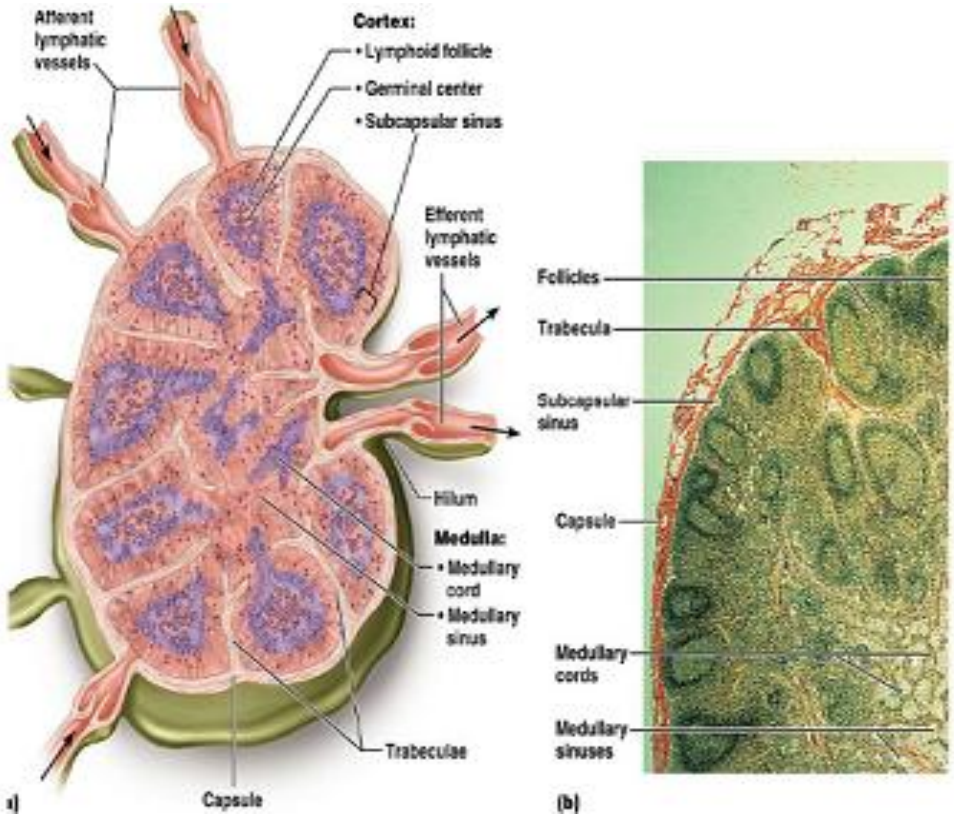


Fig 2 – Structure of the lymph node

RETICULAR FIBRES

The fibres produced by the primitive reticular cells are termed reticular fibres which consists of fine collagen. These are many in number and form a thick meshwork around the cortex, blood vessels and the lymphoid follicles.

CELLS OF LYMPH NODES

The lymph nodule consists mainly of the B-lymphocytes. The lymphoblasts occupy the paler germinal centre of the nodules which are stimulated by the antigens. These divide more repeatedly giving rise to many B-lymphocytes and they aggregate to form the dark rim around the germinal centre. They mature into plasma cells which are seen mainly in the medullary cords which form the antibodies which then ultimately reach the efferent vessel and the blood stream.

The medullary cords consist of the T-lymphocytes which form the intervening tissue between the nodules.

The reticular fibres consist of the fibroblasts which were previously termed the reticular cells. The lymphatic sinuses consist of numerous macrophages which phagocytose the antigens and ultimately present to the lymphocytes. [7]

AFFERENT VESSELS

The vessels enter at different parts from the periphery which after dividing form a dense plexus in the substance of the capsule and open into the sub capsular lymphatic plexus which is in continuity with the lymphatic sinuses of the cortex. The vessel while entering the node loses all its coverings except the endothelial covering.

EFFERENT VESSELS

The vessels originate from the lymphatic sinuses of the medulla and terminate at the hilum. The passage of the lymph though the sinus slows down gradually which arrests the morphological elements carried in the lymph.

PHYSIOLOGY OF THE LYMPH NODE

Lymph node plays an important role in the defence mechanism of the body. When there is an infection in the proximal part of the gland, the distal part gets inflamed due to the localisation of the bacteria and their toxins. They play an important role in the phagocytosis and destruction of the invading organisms. No organism or foreign body can enter the blood stream without getting filtered at the lymph node.

Bacteria enter the blood stream slowly through the lymphatics but the toxins and the viruses of large molecular weight(>20,000 LMV) do not enter the blood stream if the lymphatics are blocked. Immobilisation reduces the affected area and aids in the resolution. It also plays an important role in the production of the immunoglobulins. [8]

ANATOMY OF THE LYMPH NODES OF THE NECK

There are about 800 lymph nodes all over the body out of which around 300 lie in the neck. Lymph nodes of the neck are particularly targeted by tuberculosis.

Nodes of the neck are involved in infections of oral cavity, nasal cavity, ear and scalp. Neck nodes account for 90% of chronic lymphadenitis of the body. [9]

CLASSIFICATION OF LYMPH NODES OF NECK

- Circular group of nodes
- Vertical group of deep cervical nodes.

CIRCULAR CHAIN OF NODES:

It is composed of

A) OCCIPITAL NODES:

These are one or two nodes located between mastoid process and external occipital protuberance. They are enlarged in infection of back of the scalp.

B) POST-AURICULAR NODES:

Present over the mastoid process behind the pinna. They are involved in infections of temporal region of scalp, back of the pinna and external auditory meatus.

C) PRE-AURICULAR NODES:

Located exactly in front of tragus, superficial to parotid fascia. Outer surface of the pinna and side of the scalp drains into pre-auricular nodes.

D) PAROTID NODES:

They are located both in the substance of the parotid and deep to it., between it and lateral wall of pharynx.

The deeper nodes drain

1.nasopharynx

2.back of the nose.

Superficial group drain

1.eye lids.

2.front of the scalp.

3.external auditory meatus.

4.tympanic cavity..

E) FACIAL NODES:

Composed of deep and superficial nodes.

Superficial group consists of

a) Infra-orbital :located just below orbit.
b) Buccinator: over the buccinator muscle , lateral to the angle of the mouth.

c) Supra-mandibular: over the mandible, in front of the masseter around the facial artery.

They drain the conjunctiva, eye lids, nose and cheek.

DEEP GROUP:

They are situated around the maxillary vessels and in correspondence to the external pterygoid muscle. They are involved in infections of a) temporal fossa b)infra-temporal fossa c) back of the nose d) pharynx.

F) SUB-MANDIBULAR NODES:

These are located in relation to the sub-mandibular salivary gland in the sub-mandibular triangle. The lymph nodes are directly in contact with salivary gland situated under the deep fascia.

They drain

a) Side of the nose.
b) Inner angle of eye.
c) The cheek.
d) Angle of mouth.
e) Upper lip
f) Outer aspect of lower lip.
g) Gums
h) Side of the tongue.

G) SUB-MENTAL NODES:

Situated in the sub-mental triangle. They are involved in infections of central part of lower lip and floor of the mouth. They also drain the apex of the tongue..

H) SUPERFICIAL CERVICAL NODES:

Situated on the outer border of sternomastoid around the external jugular vein. They are involved in infections of the parotid gland and lower ear.

I) ANTERIOR CERVICAL NODES:

Located in the midline of neck, in front of trachea and larynx. Consists of deep and superficial group of nodes.

Superficial group: they are located around the anterior jugular vein and receive lymph from skin of the neck. They are insignificant.

1) Deep group:

a) Infra-hyoid nodes: present over the thyrohyoid membrane and receive lymph from front of the larynx
b) Pre-laryngeal nodes: located over the cricothyroid ligament.

They drain larynx. The afferent vessels pass through the foramen in the middle of cricothyroid ligament. They also assist in receiving lymph from the thyroid.

c) Pre-tracheal nodes: they are located in correspondence to the inferior thyroid veins in front of the trachea and receive lymph from the thyroid and trachea.

EFFERENT VESSELS OF CIRCULAR CHAIN:

The efferent vessels directly receive lymph from the nodes mentioned above to the deep cervical nodes except from facial and submental nodes. The efferents pass from facial and submental nodes pass to submandibular nodes first.

VERTICAL GROUP OF DEEP CERVICAL NODES:

These are situated in relationship to carotid sheath. A small group of nodes are located behind the pharynx and they are called retropharyngeal nodes. They receive lymph from pharynx, auditory tube and back of nose.

The vertical group of nodes lie along the pharynx, trachea and oesophagus,from the base of the skull to the root of the neck. They are divided conventionally into superior deep cervical and inferior deep cervical by omohyoid. Some of the nodes project beyond the posterior margin of

sternomastoid into posterior triangle of neck. A small number of nodes are situated in the groove between trachea and oesophagus along recurrent laryngeal nerve. They are known as paratracheal nodes and they drain the thyroid.

Two of the deep cervical nodes are specified:

1.Jugulo-digastric nodes: the node for tonsil.

2.Jugulo-omohyoid node: a node located on the common carotid above the point just above where the anterior belly of omohyoid crosses the vessel. It plays a vital role in the drainage of tongue.[10]

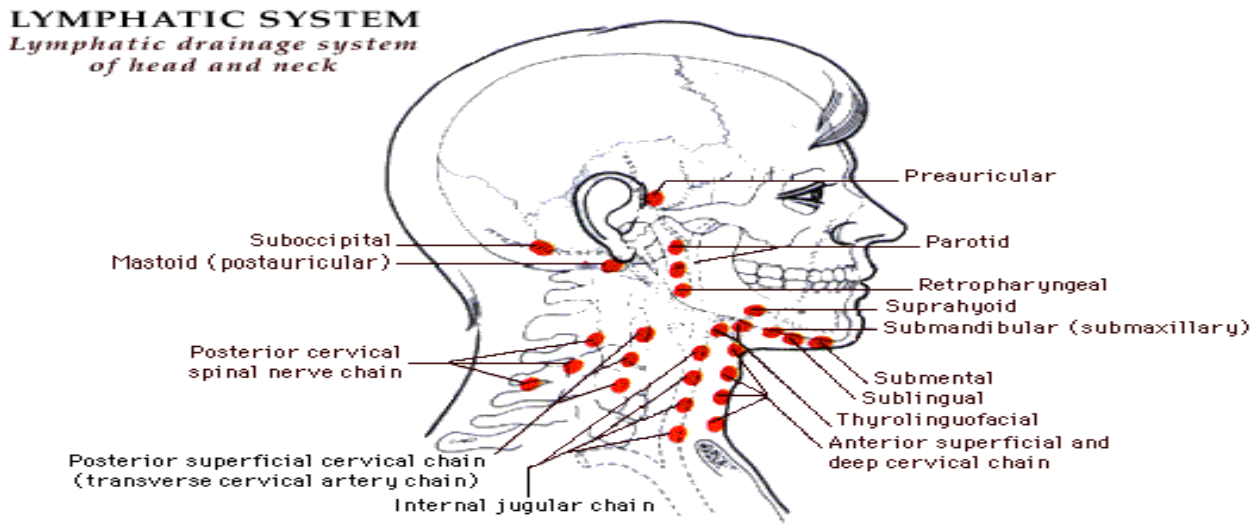
EFFERENTS OF VERTICAL CHAIN:

The lymph from the deep cervical trunk enters one trunk –jugular lymph trunk, that leaves the inferior deep cervical nodes. On the right side it enters the junction of subclavian vein and internal jugular vein. On the left side , it joins the thoracic duct.

AFFERENTS OF THE VERTICAL CHAIN:

The deep cervical nodes drain the entire head and neck, directly or indirectly from the circular chain. [10]

Fig 3 – Anatomy of the lymph nodes draining the head and neck



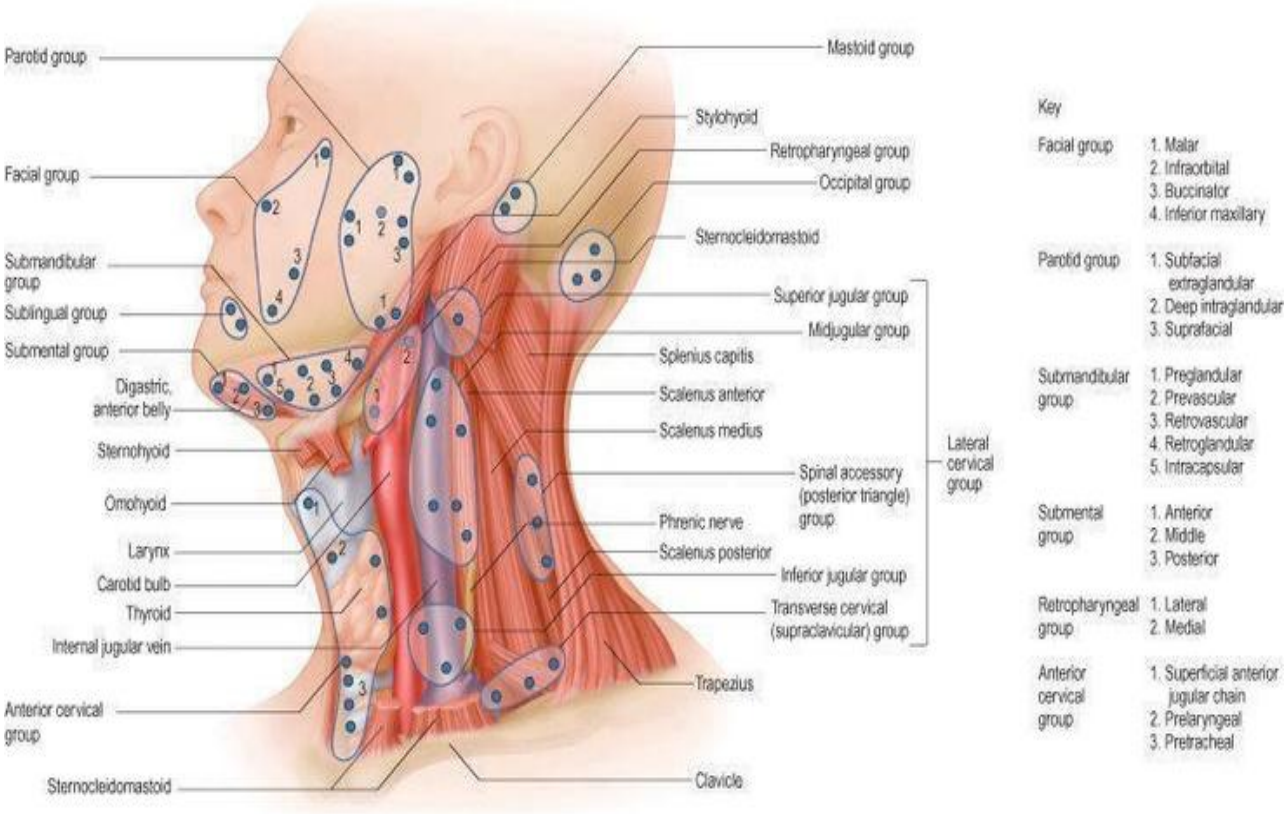


Fig 4 – Anatomy of the lymph nodes of the head and neck

BACTERIOLOGY

A) TUBURCLE BACILLI

1. Human mycobacterium tuberculosis
2. Bovine mycobacterium bovis
3. Murine mycobacterium microti
4. Avian –mycobacterium avium
5. Cold blooded – mycobacterium marinum

B) LEPRA BACILLI

1. Human mycobacterium leprae
2. Rat –mycobacterium lepral murium

C) MYCOBACTEERIA CAUSING SKIN ULCERS

1. Mycobacterium ulcerans
2. Mycobacterium balnei

D) ATYPICAL MYCOBACTERIA

1. Photo chromogens
2. Scoto chromogens
3. Non Photo chromogens
4. Rapid growers

E) JOHNE'S BACILLUS

1. Mycobacterium paratuberculosis

F) SAPROPHYTIC MYCOBACTERIA

1. Mycobacterium butyricum
2. Mycobacterium phlei
3. Mycobacterium stercoris
4. Mycobacterium smegmatis
5. Others

The term ‘mycobacteria’ means fungus like bacteria which are nothing but the slender rods showing branching filaments which resemble with that of the fungus mycelium. Mycobacteria are aerobic, non motile, non capsulated, non sporing. They are called the ‘Acid Fast Bacilli’ because they do not stain immediately but once stained, resist decolourisation with dilute mineral acids. Growth is slow. The genus includes obligate parasites, opportunistic pathogens and saprophytes.

Robert Koch announced his greatest discovery, the causative organism of tuberculosis, to the Physiological society in Berlin on 24th March 1882. Tuberculosis in human beings is caused by either the human type or the bovine type of bacillus. Subsequently the other types were also described. The generation time of the bacilli in vitro is 14-15 hours. Colonies are formed in 2weeks but can be delayed upto 6-8weeks. The growth is very slow and does not occur below 25°c or above 40º. Optimum temperature is 37ºC. Optimum pH is 6.4-7.0. Mycobacterium bovis is usually microaerophilic when it is primarily isolated but becomes aerobic on subculture. Mycobacterium bovis grows

sparsely whereas Mycobacterium tuberculosis grows luxuriantly but the growth can be stimulated by 5-10%CO2 or by adding glycerol but does not grow in media containing 500mg of p-nitrobenzoic acid. But the addition of these growth stimulants has either no effect or inhibits the bovine strains.

Koch grew the tubercle bacilli on heat coagulated bovine serum. Tubercle bacilli are highly susceptible to fatty acids in culture media but is neutralised by serum, charcoal or albumin. Solid media contains egg(Lowenstein Jensen, Petragini or Dorset), blood(Tarshis medium), serum(Loeffler's serum slope) or potato(Pawlowsky). Most widely used solid media is the Lowenstein medium without starch. This was recommended by IUAT. Lowenstein Jensen medium consists of mineral salt solution, coagulated hen's egg, asparaginase and malachite green. Malachite green is a selective agent inhibiting other bacteria. Another cheap alternative to the LJ medium is the media composed of eggs, coconut water and malachite green. Mycobacterium tuberculosis on solid medium produces dry, rough, raised, irregular colonies with a wrinkled surface which are tenacious, not easily emulsified, creamy white initially which subsequently changes to yellow/ buff colour. Mycobacterium bovis on solid medium forms flat, smooth, moist, white colonies which breaks up readily on touching. Liquid medium includes the Dubo's, Middlebrook's, Proskauer, Beck's, Sulo's and Sauton's media. Liquid media aids in sensitivity testing, chemical analysis, preparation of antigens and vaccines. In Dubo's medium,

there is a diffuse growth of the bacilli and contains Tween 80 (Sorbitan mono oleate).

The growth of the bacilli begins at the bottom, creeps up by the sides and forms a pellicle on the surface which may extend along the sides above the medium in liquid medium without dispersing agents. Cord factor which is present in both the pathogenic and some non-pathogenic species is not fully responsible for the virulence.

In liquid media, virulent strains tend to form long serpentine cords while the avirulent strains tend to grow in some dispersed fashion. Morphology of the colonies can be modified by the presence of bacteriophage in the strain.

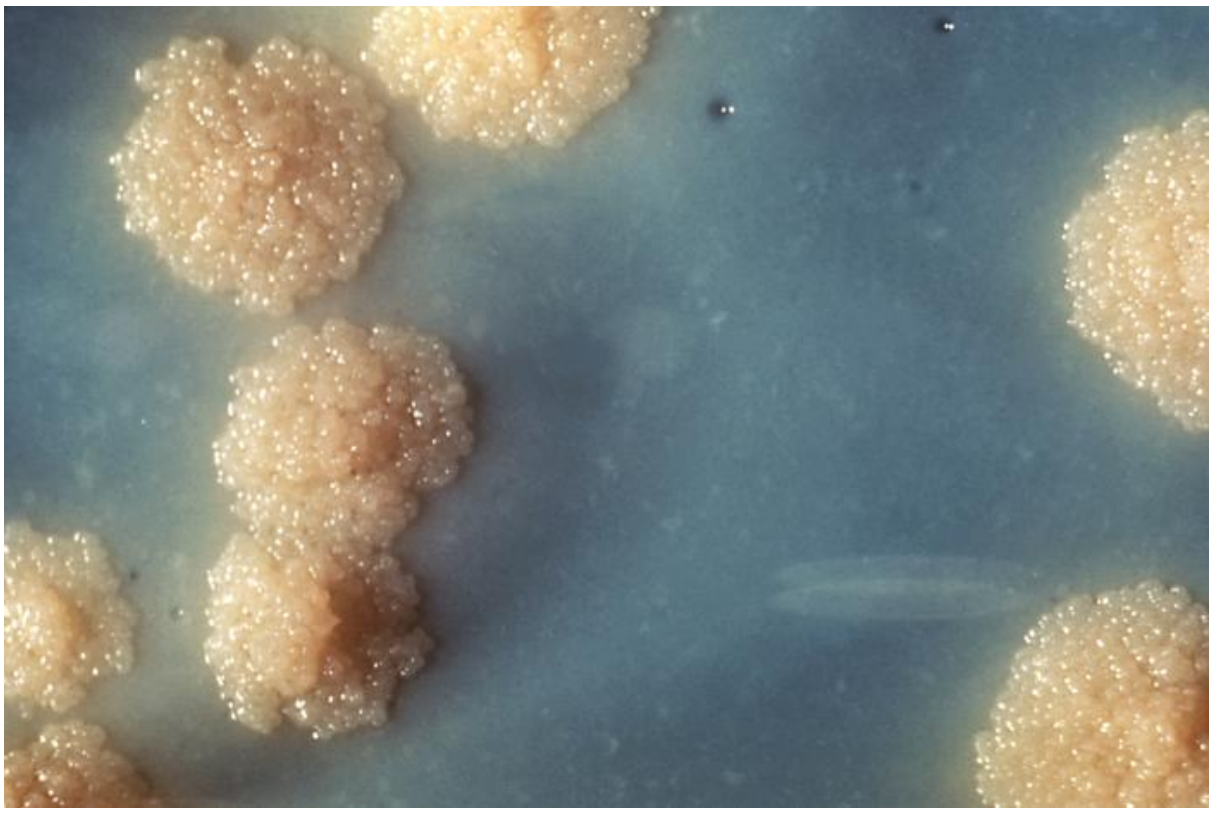


Fig 5 – Mycobacterium tuberculosis colonies

RESISTANCE:

Mycobacterial survival depends on the material in which the bacteria are present. The bacilli are killed at 60°C in 15-20mins but the bacilli in the sputum remains alive for 20-30hours and that present in the droplet remains viable for 8-10days. Cultures are killed by direct sunlight exposure for 2hours but remain viable for 6-8months at room temperature and 2years in the deep freeze cabinet at -20ºC.

Mycobacteria survives on exposure to 5%phenol, 15% sulphuric acid, 3% nitric acid, 5% oxalic acid and 4% sodium hydroxide but destroyed by the contact of the culture with tincture of iodine for 5minutes, and that of 80% ethanol in 2-10minutes. Hence, 80% ethanol is used as a skin disinfectant and a disinfectant for rubber gloves, clinical thermometer. Mycobacteria are sensitive to formaldehyde and glutaraldehyde.

BIOCHEMICAL REACTIONS:

Mycobacterial species can be identified by biochemical tests which includes :

1. **NIACIN TEST:**

   Addition of 10%cyanogen bromide and 4% aniline in 96% ethanol to a suspension of culture containing human tubercle bacilli shows a canary yellow colour (positive reaction) while the bovine type produces a

negative reaction. Tubercle bacilli produce niacin when grown on a medium containing egg.

2. ARYL SULPHATASE TEST:

The atypical mycobacteria are grown in culture containing 0.001M tripotassium phosphathalein disulfate and forms aryl sulphatase. If sodium hydroxide is added to the medium drop by drop, a pink colour is produced which indicates a positive reaction.

3. NEUTRAL RED TEST:

Virulent strain binds neutral red in alkaline buffer solution while the avirulent strains do not.

4. CATALASE-PEROXIDASE TEST:

Aids in knowing the sensitivity of the strain to INH (Isotonic Acid Hydrazine).Helps in distinguishing the tubercle bacilli which are weakly catalase positive and peroxidise positive from atypical mycobacteria which are strongly catalase positive. Tubercle bacilli resistant to INH lose their catalase and peroxidise activity.

To 5ml of the test culture, equal volume of 30 vol of H_2O_2 and 0.2% catechol in distilled water is added and allowed to settle for a few minutes. Catalase

production in indicated by effervescence and peroxidise activity by browning of the colonies.

5. AMIDASE TEST:

5 amides namely the acetamide, benzamide, carbamide, nicosanamide and pyracinamide are used which differentiates mycobacteria based on the ability to split amides. The bacilli in the solution is incubated with 0.00165M solution of the amide at 37°C and 0.1ml of MnSo4, 4H20, 1.0ml of phenol solution and 0.5ml of hypochlorite solution are added and placed in boiling water for 20minutes. A positive test is indicated by the presence of blue colour.

6. NITRATE REDUCTION TEST:

This is positive with Mycobacterium tuberculosis only. [11]

ANTIGENIC PROPERTIES:

Specificity of the group is due to polysaccharide and type specificity is due to the protein antigens. Delayed hypersensitivity is developed to the protein of the bacillus(tuberculin) following infection by the tubercle bacilli.

Tuberculin from Mycobacterium tuberculosis, Mycobacterium bovis and Mycobacterium microti appears to be indistinguishable. There is some antigenic relationship between the protein antigens of tubercle bacilli and atypical

mycobacteria shown by weak cross reactions in skin testing. A ribonucleoprotein from Mycobacterium tuberculosis reacts with serum from patients with lepromatous leprosy.

Mycobacterium tuberculosis has shown to be antigenically homogenous to Mycobacterium bovis and Mycobacterium microti by agglutination, agglutinin absorption, gel precipitation and passive hemagglutination.

BACTERIOPHAGE:

4 phage types of tubercle bacillus:

A-Commonest type worldwide

B-Occurs in Europe and North America

C-Seen rarely

D-Common in India and neighbouring countries. [11]

Phage 33D from a lysogenic environmental mycobacterium lyses all the types of Mycobacterium tuberculosis but not BCG.

MOLECULAR TYPING:

Tubercle bacilli are genetically homogenous where certain strains contain a chromosomal insertion sequence IS6110 which acts as a transposon. DNA fingerprinting of these insertion sequences are employed for the diagnosis of the infection, epidemiological studies for tracing the source of outbreaks.

HOST RANGE:

Natural infection in human beings, other primates, dogs and other animals who have close contact with human beings is caused by Mycobacterium tuberculosis which is highly infectious for guinea pigs and hamsters but non-pathogenic for rabbits, cats, goats, bovines and fowls. Mice are moderately susceptible and develop progressive infection following intra peritoneal, intravenous or intracerebral inoculation.

Mycobacterium bovis produces tuberculosis in cattles, human beings, primate carnivores which includes dogs, cats and badgers, swine and parrots. BCG, the tuberculin vaccine is the attenuated strain of Mycobacterium bovis. It is highly pathogenic for rabbits, guinea pigs and calves, moderately pathogenic for dogs, cats, horses and rats and non-pathogenic for fowls.

Mammalian tubercle bacilli are considered as variants of single species of Mycobacterium tuberculosis. Heterogenous group of tubercle bacilli isolated in Africa which shows properties intermediate between the human and bovine types are termed the African type. Human type of bacilli isolated in South India which has low virulence for guinea pigs are termed the Asian type. Mycobacterium avium causes tuberculosis in birds, ‘Yersin type ’ tuberculosis in rabbits and mice-proliferating without producing macroscopic tubercles. It is non-pathogenic for guinea pigs and rats.

Mycobacteria microti causes focal lesions in human beings, guinea pigs, rabbits and calves.[11] It causes tuberculosis in voles.

MORPHOLOGY:

Tubercle bacilli vary in length from 0.8-5.5µ and width from 0.2-0.6µ.They were examined under electron microscopy by Rosenblatt et al in 1942. Tubercle bacilli are rod shaped with rounded/ slightly flattened ends. Other forms includes curved, bean shaped and oval forms.

Human form is long, slender whereas the bovine form is short and stumpy.

Avian type is pleomorphic.

STAINING PROPERTY:

Tubercle bacilli are termed the acid fast bacilli due to the presence of lipoid fraction containing free hydroxyl groups known as mycolic acid. These are present in the capsule of the organisms (discovered by Anderson in 1938). Ziehl Neelson developed a staining technique with 5% carbol fuchsin in catechol for a few minutes at a higher temperature or for longer time at a lower temperature following which they resist decolourisation with potent hydrochloric acid. [12]

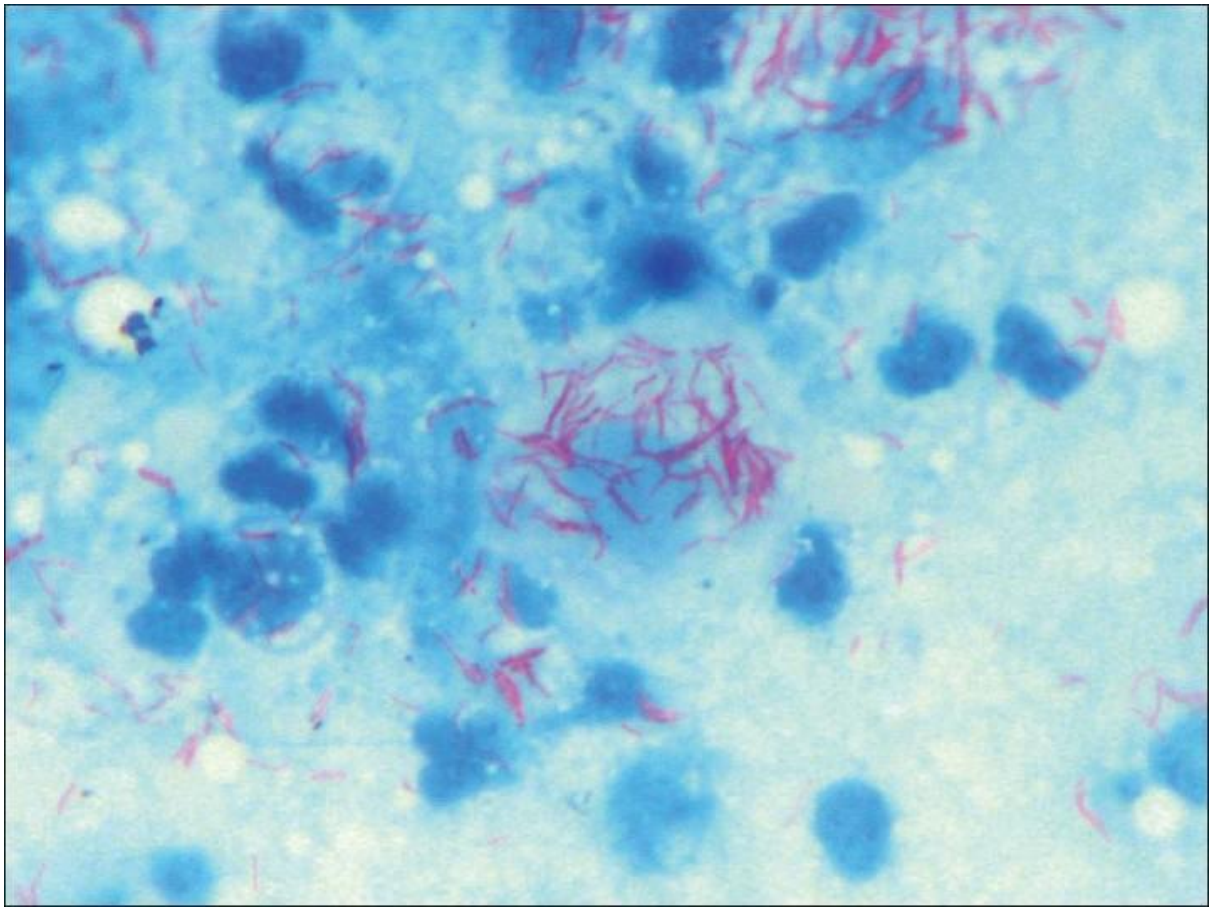


Fig 6 – Ziehl – Neelson staining

FLUOROSCEIN TECHNIQUE OF DETECTING BACILLI:

Tubercle bacilli absorb acromine fluorescent dye. When culture containing tubercle bacilli is exposed to UV light , the bacilli is seen as brilliantly illuminated structures against dark background even under the low power microscope.

CHEMISTRY OF THE BACILLI: [12]

Bacilli contain the following constituents:

PROTEINS:

Upon injection, protein bound to a wax fraction induces tuberculin sensitivity. Partially purified tuberculin contain a mixture of small proteins (10kDa).

CARBOHYDRATES:

Contains polysaccharide whose role in pathogenesis is uncertain.

LIPOID FRACTION:

Includes mycolipids (long chain fatty acids)[13], waxes and phospholipids. Muramyl dipeptides with mycolic acids cause granuloma formation. Isolated by Anderson in 1937.

- Responsible for resistance to chemical
- Adjuvant effect (waxD) enhancement of antigenic immunogenicity
- Intracellular persistence in non-activated macrophages by means of inhibition of the phagosome lysosome formation
- Complement resistance
- Virulence is due to the cord factor (trehalose 6,6-dimycolate) [14]

SOURCE OF INFECTION:

TONSILS:

In many cases, the tubercle bacilli enters through the tonsils of the corresponding side so that the lesions are either minimal/ has healed which

makes its identification impossible. Upper deep cervical group of lymph nodes are most commonly involved. The tuberculous process is usually restricted to the clinically affected group of lymph nodes in more than 80% of the patients but a primary focus in the lungs should always be suspected.[9] On examination, tonsils are not useful to aid in the presence or absence of infection due to chronic tonsillitis. [15]

TOOTH:

This portal of entry should be suspected only when the submandibular or submental group of lymph nodes are primarily involved. [16] If the portal of entry is the scalp, then the posterior triangle nodes are mainly involved which receives the lymphatic vessels from the adenoid also.

PATH OF INFECTION:

Main source of infection are patients who are open case of tuberculosis or carriers.

Contamination of the breast milk is extremely rare even when the mother is a tuberculous patient. Contamination of the cow milk is possible but because of the improvement in hygienic measures and the use of pasteurised milk, contamination of the cow milk is almost eradicated.

PORTAL OF ENTRY: [2]

The bacilli are carried by air borne infections which gets filtered at the nostrils and adenoids to produce lesions at the site of the draining lymph nodes.

DROPLET INFECTIONS:

TB carriers or patients possess thousands of bacilli in throat and nasal passages which spread during the process of coughing/ sneezing. Bacilli enters the host without losing much vitality even though some bacilli cling to the dust, clothes, screens, utensils for several weeks and start losing their vitality.

DUST INFECTION:

TB bacilli in the sputum when spit on open streets gets dried and can remain alive for several months which can enter the human body while inhaling but is secondary to droplet infection.

INTRA-UTERINE INFECTIONS:

Previous theories showed that the infection is usually postnatal and placenta is not a carrier of infection but recent theories have shown that the foetus can get infected even upto the extent of septicaemia without positive tuberculin test.

RETROGRADE LYMPHATIC SPREAD:

Lymphatic spread is common in tuberculous adenitis. The involvement of the lower deep cervical group of lymph nodes raises suspicion that it is involved secondary to mediastinal group of nodes.

Hematogenous spread is a rare phenomenon. Hematogenous involvement occurs in 50% instances of primary complex in younger age group and is associated with multiple lymph node involvement before the tuberculin test becomes positive. It is commonly associated with miliary tuberculosis or tuberculous meningitis.

In progressive pulmonary tuberculosis, extra- pulmonary tuberculosis is uncommon.

EPIDEMIOLOGY OF THE DISEASE:

TB, in the global prevalence, has been the most important of all the human Infections, with massive morbidity and mortality. Evidence of spinal tuberculosis has been identified in the Egyptian mummies. Prevalence increased rapidly following urbanisation and overcrowding, but now the incidence has come down with the improvement in standards of living. TB has been rightly described as the “parameter of social welfare”.

About 20 million open cases of tuberculosis are reported at any point of time all over the world, out of which 70-80% belong to the poorly developed nations.

About 4-5million new cases of open pulmonary tuberculosis arise each year out of which 3million people die each year because of TB. [17]

TB is the highest burden in India where 1.8million persons develop TB out of which 0.8million are smear positive. India accounts for 1/5th of the global burden of TB. In India, 2 out of every 5 are infected with TB bacilli and 2 persons die of TB every 3minutes. 0.4million die of TB every year. [18]

In developing countries, prevalence of both infection and active disease is high and mortality is increased in infants and children less than 5years of age. The infective rate is high by the age of 20 with a rate of 10-15%. [19]

Predisposing factors which favours the development of tuberculosis includes malnutrition, low socio-economic status, occupational exposure to silica, medical and paramedical workers who are in direct contact with the patients and infectious materials, population from the hilly regions who settle down in the plains.

Infections like measles, typhoid, sore throat and acute respiratory tract ailments may break down the host resistance and precipitate tuberculosis. Worldwide, the most important risk factor for the development of tuberculosis is HIV induced immunodeficiency. HIV and TB form a deadly combination. TB accounts for 25% of the deaths among people with HIV. [20]

In Western countries, because of the improvement in standards of living, attention to diet, housing, sanitation, preventive interventions and specific anti-tubercular drugs, the incidence of cervical lymphadenitis is so negligible which

is a successful attempt in eradicating tuberculosis. But in India, the problem still remains unresolved due to poverty, uncooperative patients, infections and irregular treatments.

ATYPICAL MYCOBACTERIA:

This group includes those bacilli other than the human or bovine tubercle bacilli which can also cause the human disease clinically resembling that of tuberculosis and has been termed the 'atypical', 'anonymous' or 'unclassified' mycobacteria.[21] 'Environmental or opportunistic mycobacteria' is the best suited term for these bacilli as their natural habitat is either soil or water and cause opportunistic infection in human beings. Over recent years, the term 'non- tuberculous mycobacteria ' has gained worldwide acceptance. They are also termed the 'paratubercle', 'tuberculoid' and MOTT (mycobacteria other than tubercle) bacilli. There is no proof of evidence of direct human to human transmission.

Saprophytic mycobacteria such as M.smegamtis and M.phlei are not capable of infecting human beings or animals.[21] At times the human infection with them is common while the disease is rare because, when injected into the guinea pigs, they do not cause progressive disease.

In developed countries, where tuberculosis is almost eradicated or under control, atypical mycobacteria is gaining importance. Some may produce pulmonary disease which becomes almost indistinguishable from tuberculosis

while others may cause lymphadenitis. In Western countries, the spread of HIV has increased the disease caused due to atypical mycobacteria. [22]

Atypical mycobacteria are almost resistant to the anti- tubercular drugs like Streptomycin and Isoniazid and sensitive to Rifampicin. Usually a combination of drugs is used but chemotherapy does not help in such cases and surgery becomes mandatory.

CLASSIFICATION:

RUNYON'S CLASSIFICATION OF ATYPICAL MYCOBACTERIA

on the basis of pigment production and the growth rate).

Identification of the species is by other classifications.

Group I – Photo chromogens

Group II – Scoto chromogens

Group III – Non photo chromogens

Group IV – Rapid growers

GROUP I – PHOTO CHROMOGENS:

They are slow growing but the growth is faster than that of the tubercle bacilli. They form colonies which produces no pigment in the dark but when exposed to light for 1hour and after reincubation, a yellow orange pigment appears.

Mycobacterium kansasi causing pulmonary disease which resembles that of tuberculosis.

Mycobacterium marinum causing swimming pool granuloma.

Mycobacterium simiae and Mycobacterium asiaticum causing pulmonary disease.[23]

GROUP II SCOTOCHROMOGENS:

Do not cause human disease except for Mycobacterium scrofulaceum. Strains produce yellow-orange-red pigmented colonies even in the dark.

M.scrofulaceum which causes cervical adenitis in children. [24]

M.gordonae which rarely produces human infection.

M.szulgai also belongs to this group.

GROUP III NON-PHOTO CHROMOGENS:

Colonies resembling that of tubercule bacilli do not form pigment even on exposure to light. [25]

M.intracellulare was first discovered as a human pathogen at the Battey State Hospital for TB in Georgia, USA. Hence, it is also known as the 'Battey Bacilli'. This produces the pulmonary and renal infection and lymphadenopathy.

M.avium rarely causes cervical adenitis in children and lung disease in the elderly.

M.xenopii occasionally causes chronic lung disease in human beings.

There are many controversies about whether M.avium and M.intracellulare should be considered as a variant of single species or are separate species and is still a debate. [26] As of now, they are considered in complex along with M.scrofulaceum and the entire complex is called the M.avium-intracellulare-scrofulaceum / MAIS complex.

GROUP IV RAPID GROWERS:

This group produces colonies within 7days of incubation at 37°C or 25°C.

The chromogenic rapid growers are saprophytes (e.g.: M.smegamtis, M.phlei).

M.fortuitum and M.chelonei which causes chronic abscess and do form any pigment.

M.fortuitum produces pulmonary lesions which is indistinguishable radiologically from typical tuberculosis.

SKIN PATHOGENS:

Skin lesions occur either as localised/ generalised in leprosy or tuberculosis.

Mycobacteria which causes skin pathogens are M.ulcerans and M.marinum.

They produce granulomatous lesion and chronic ulcers on the skin. [27] These mycobacteria multiply optimally at skin temperature and systemic invasion/ lymph node invasion does not occur.

PATHOGENESIS AND PATHOLOGY:

PATHOGENESIS:

Five factors aids in understanding the pathogenesis of tuberculosis which includes:

Virulence of the mycobacteria which is not related to any endotoxin.

Relationship of the hypersensitivity to the immunity against the infection

Pathogenesis of caseation necrosis

This ability of Mycobacterium tuberculosis to induce the disease in experimental animals is due to mycoside in the lipoid fraction of the bacterium.

Mycosides are covalently linked complex lipids and carbohydrates. Cord factor is responsible for the serpentine, cord like growth of the Mycobacterium tuberculosis in vitro and is toxic to mice. The cell wall of the bacilli contains wax D (a glycolipid), and muramyl dipeptide and they are powerful adjuvants and is important in the development of[28] delayed hypersensitivity, skin reactions and granulomatous inflammation at the sites of the infection.

In a previous unexposed immunocompetent individual, the pathogenesis of tuberculosis is based on the development of the cell mediated immunity that confers resistance to the organism and results in the development of tissue hypersensitivity to the tuberculous antigens. Within 2-3weeks,a positive skin reaction that is granulomatous occurs and the centre of the granulomas become caseous forming typical soft tubercles.

Sequence of events from inhalation of the Mycobacteria to the development of primary foci are:

Virulent strains enter the macrophage endosome and proliferate impairing the phagolysosome formation. Antigens from the bacilli reach the draining lymph nodes and is presented to the CD4+T cells which are sensitised and again recirculate back to the site of infection where they release cytokines (IFN ɣ) and

activate the macrophages forming granulomas which trap the residual micro-organisms. These granulomas get caseated at the [29]centre (soft tubercles), but many a times there is no caseation (hard tubercles). The granulomas are composed of epitheloid secretory cells and is responsible for necrosis.

Hypersensitivity is described by the caseating response to the tubercle bacillus, the acquisition of the immunity and the resistance to the organism. The

sensitised host produces a defensive reaction and produces enhanced necrosis. The protective or destructive response is determined based on the fact of whether the primary focus of infection is localised/ generalised.

TISSUE REACTIONS IN TUBERCULOSIS:

Tissue response is based on the dose, virulence and viability of the bacilli and whether the infection is primary or secondary (reinfection).

PRIMARY COMPLEX:

Bacilli usually gets lodged in the upper part of the lower lobe or the lower part of the upper lobe. These undergo consolidation (Ghon's focus) and this combined with the nodal involvement is termed the Ghon complex. In primary infection, the lesion heals with resorption whereas in reinfection, the lesion is progressive and reaction is accentuated due to the development of hypersensitivity to the tubercle protein and localised in the area of lesion. This lesion in glandular tuberculosis is due to primary infection in the lungs/ abdomen or the tonsils and the complementary lesion is in the lymph nodes.

TUBERCLE FORMATION:

In the initial 24 hours, leucocytes migrate to the area of infection to form a centre of small abscess. The local reticulo endothelial cells / monocytes of the blood produces mononuclear cells which migrate and covers the bacilli. In the subsequent 15-20 days, nodules/tubercles are produced by groups of epitheloid cells which produce the Langhan's giant cells encircled by the lymphocytes. Tubercles consists of central area of necrosis and is surrounded by epitheloid cells, giant cells and in the periphery by lymphocytes with surrounding fibroblastic proliferation. [30]

If the resistance of the individual is good, infection may resolve without any residual lesion. And the individual develops a positive reaction to tuberculin in 4-6weeks following infection.

The susceptible individuals who are not on chemotherapy develop the following changes:

CASEATION:

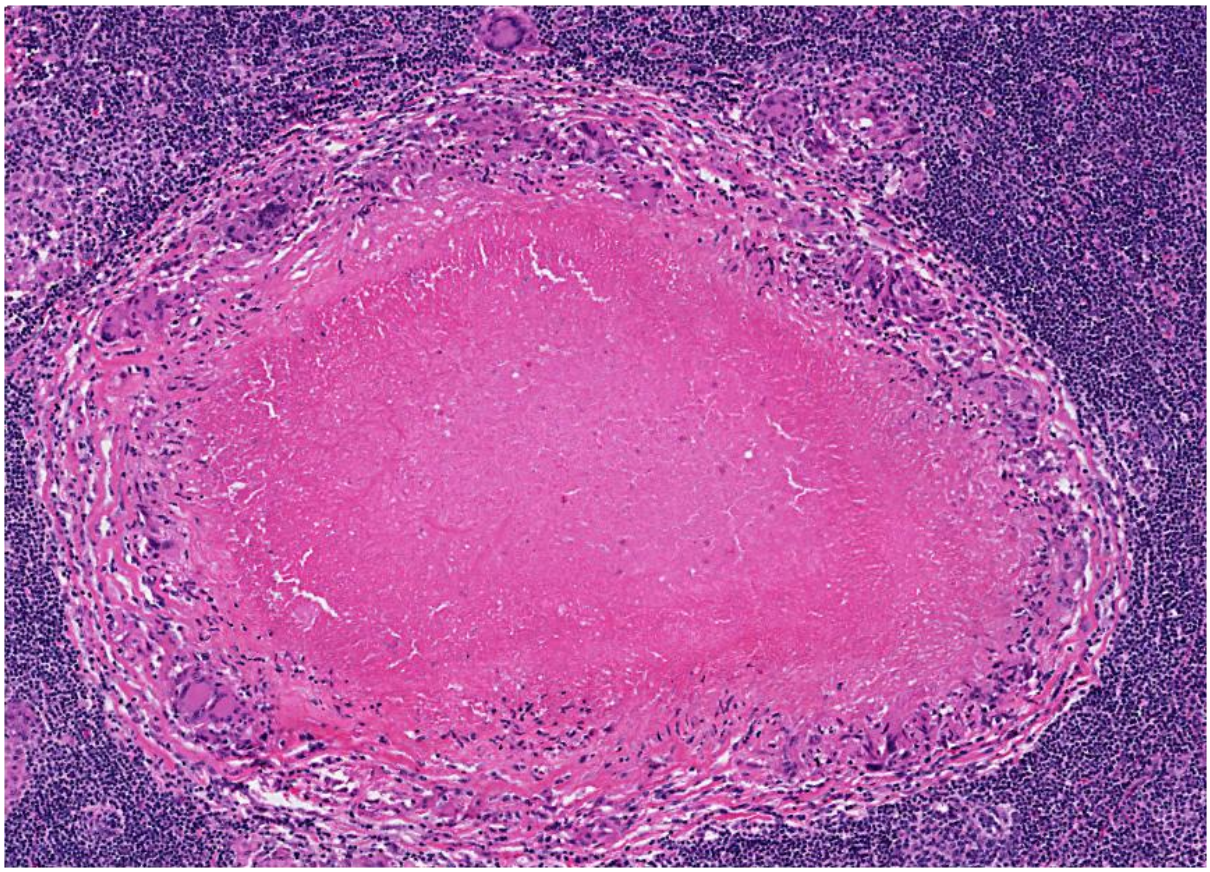


Fig 7 – Caseating granuloma

Due to the allergic hypersensitivity development following infection, the most important pathognomic factor of tuberculous degeneration and coagulative necrosis of cells with coagglutination of the tissues related to fibrin and hyaline fibrinoid substances is caseation. This has been ascribed to the glass like appearance and is therefore called the degenerated 'vitreous'.

LIQUEFACTION:

Caseous focus which has arrested growth of the bacilli is less dangerous compared to progressive lesion but once caseation occurs the healing is disrupted. The proliferation of the bacilli, migration of the leucocytes and monocytes cause the softening of caseous material forming a cold abscess which may either get absorbed or burst forming a sinus. [31]

FIBROUS CALCIFICATION AND OSSIFICATION:

The epitheloid cells and fibroblasts are involved in the formation of precollagen reticulin fibres and collagen and elastic fibres which aid in the conversion of the caseous focus to collagen and subsequently into the hyaline scar.

Calcification occurs most commonly in the primary lesion. The combination of Streptomycin, which hastens the fibroblastic activity and INH, which enhances the revascularisation in caseous area thereby helping in the regression of the swelling and healing of the fibrosis, should be used. Streptomycin alone should not be used.

CLASSIFICATION OF THE TUBERCULOUS NODE BASED ON THE PATHOLOGY:

1. **PROLIFERATIVE:**

Nodes are enlarged and hard. Capsule is intact.

No periadenitis and the lymph nodes are discrete.

2. **FIBROUS:**

Lymph nodes are enlarged, hard, mobile and shows calcification on x-ray.

3. CASEATION:

Caused by either blood or lymph borne infections. Nodes are matted. Cut surface shows pale yellow cream caseating areas. The lymph nodes are usually situated deep to the deep fascia of the neck and grows on to the skin forming a track which ultimately leads to cold abscess. The healing of the sinus is indicated with puckered scar. [31] The healing of one sinus subsequently leads to the formation of the other sinus such that multiple sinuses opens on the neck. The scrofulodermic scar are prone for keloid formation.

CLINICAL FEATURES

Tuberculous lymphadenitis is more common in females compared to males . Most commonly involves anterior triangle of neck .In the anterior triangle , upper deep cervical group is most common . It occurs in 2nd decacade of life . In most of the patients onset is insidious . It takes chronic course , it starts as a localized infection . complication occurs when secondary infection takes place . In 80% of the cases the disease is limited to the lymph node group that is affected . In some cases there may be a primary lung pathology and it has to be investigated .

Most common symptoms are evening rise of temperature , malaise and appetite loss. Duration may vary from months to years.

On examination , the consistency of lymph node may be hard or fluctuant .Inflammatory changes are minimal compared to other bacterial lymphadenitis . The swelling is non tender and freely mobile. skin changes are minimal .

The most common group affected is cervical(63%) , next group that is involved is mediastinal(27%) and then axillary(8%). Mostly it is unilateral and it involves deep cervical group of lymph nodes.

Multiple group of lymph nodes is involved if the origin is through hematogeneus route. There may be splenomegaly .Toxic symptoms are present

There are four clinical types:

ACUTE TYPE: common in infants and children , resembles acute Lymphadenitis [32]

CASEATING TYPE : common in young adults ,neighbouring lymph nodes are also involved . Typical matted nodes with caseation . cold abscess and sinuses are common .

HYPERPLASTIC TYPE: lymphoid hyperplasia is predominant. Mostly single group of lymph node is involved , usually it is firm and discrete .

ATROPHIC TYPE: common in elderly where there is natural involution. The nodes are usually small .

COURSE OF THE DISEASE:

There are 5 pathological stages (Jones and Campbell) . Patient may present with any of these stages . [33]

STAGE 1 :

Lymph nodes are enlarged . They are firm , discrete and freely mobile. There is solid inflammation and non specific hyperplasia.

STAGE 2:

Lymph nodes are rubbery and enlarged .They are matted together due to periadenitis .

STAGE 3:

Lymph nodes break down and liquefy to form abscess due to caseation. Pus collects beneath the fascia. There may be varying consistency . May be fluctuant without inflammatory skin changes (cold abscess).

STAGE 4:

The deep cervical fascia may get eroded and the pus may get collected beneath the superficial fascia which is laborious to form collar stud abscess .

STAGE 5:

The superficial abscess enlarges and then eventually burst through the skin when it gets ulcerated resulting in continuously discharging sinus until the necrotic material beneath the deep cervical fascia gets cleared . Healing may occur only when the conditions favour.

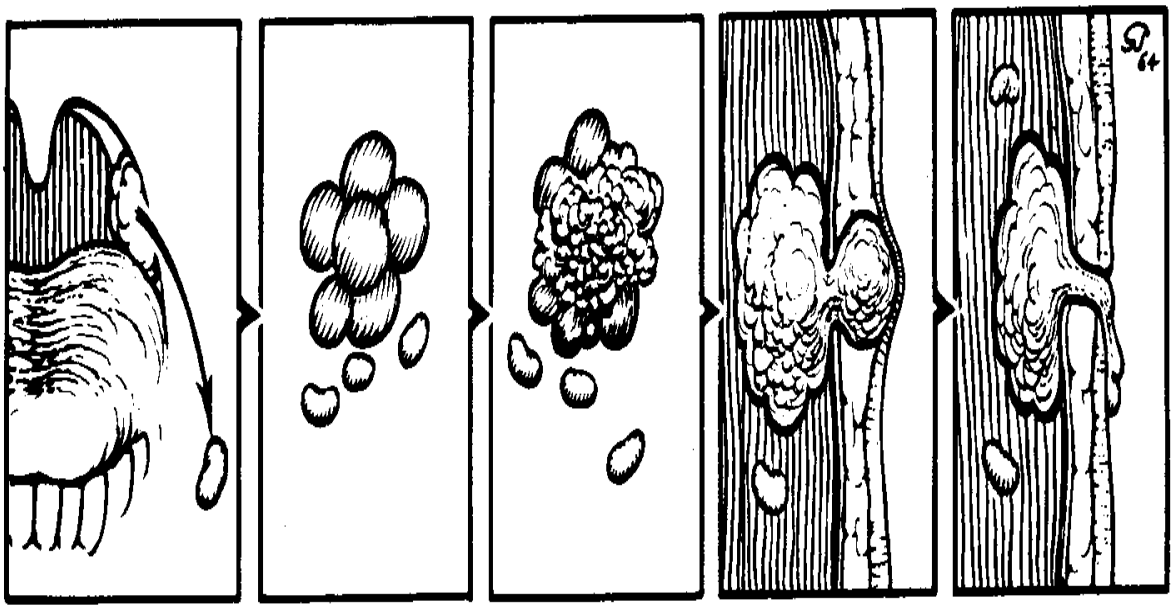


FIG. 186.—A summary of the natural history of tuberculous lymphadenitis.

Fig 8 – Stages of tuberculous cervical lymphadenitis

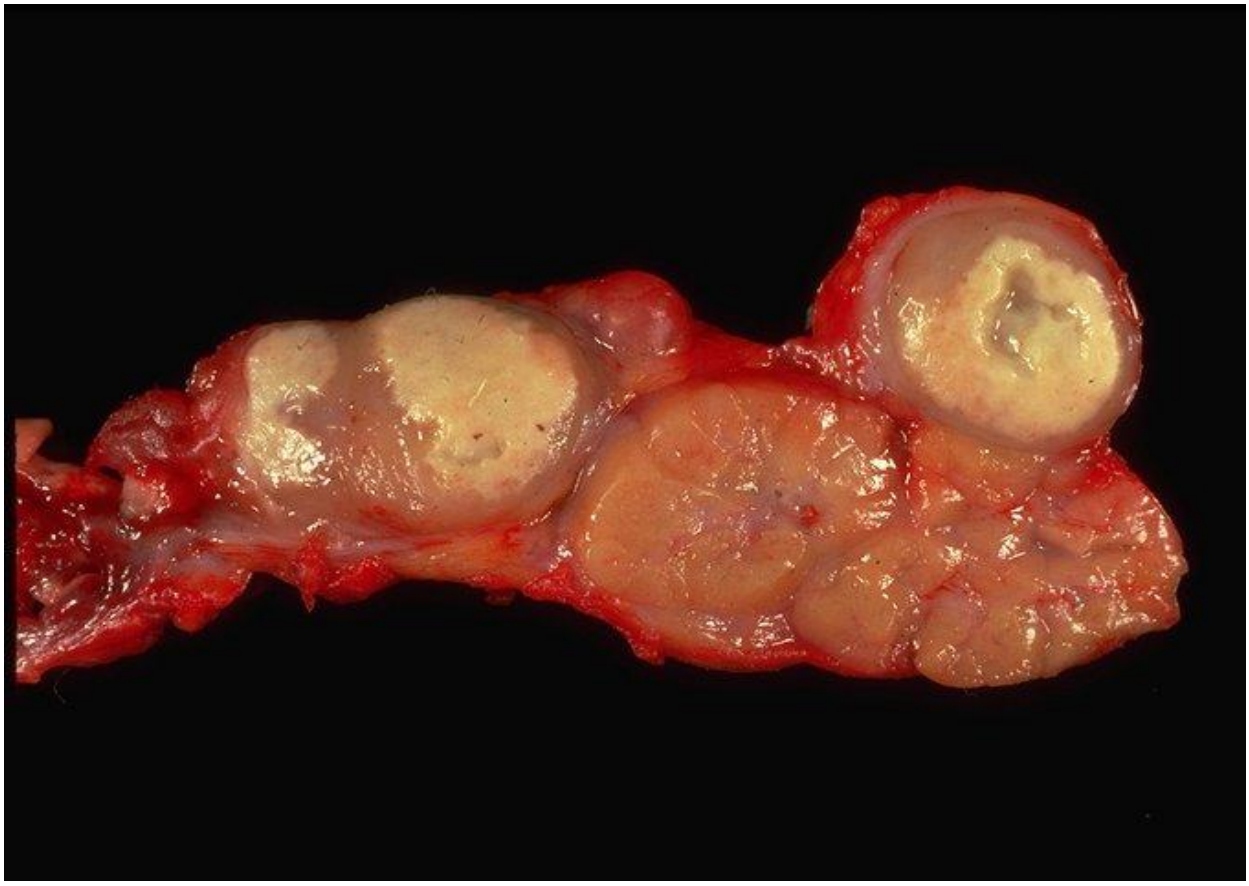


Fig 9 – Gross picture of a tuberculous lymph node

OTHER PRESENTATIONS

- In 10% individuals there may be long stem for the collar stud abscesses. The feeding infected node may be located in the other triangle of the neck with abscess presenting in some other triangle .In such cases it is necessary to palpate the full length of the stem.
- In some other cases the abscess is situated over the affected node and only small group of nodes are involved.

LABORATORY INVESTIGATIONS

- **RBC count**
- **Hb% -** may be microcytic hypochromic anemia
- **Differential count –** lymphocytosis [34]
- **WBC count -** not altered
- **ESR**- raised . It is a prognostic factor
- **Mantoux test** – positive test has no diagnostic value

  Negative test excludes tuberculosis

  PROCEDURE:

  0.1 ml of PPD (purified protein derivative) is injected in the volar aspect of the forearm .(0.1ml =5Tu) After 48 to 72 hours look for induration at the injected site. Induration is measured transversely and the erythema is not taken into account. Positive test is considered if the induration exceeds 10mm , Negative if it is < 5mm and doubtful if it is 6-9mm.

  POSITIVE TEST: suggestive of recent or past infection of the tubercle bacilli or BCG vaccination. It is due to hypersensitivity to

tubercular protein. It becomes positive only after 4 to 6 weeks after infection or vaccination.

NEGATIVE TEST: excludes tuberculous infection.

But it does not always rule out tuberculosis .

FALSE NEGATIVE: occurs in

- immunosuppression
- convalescence from viral infections
- severe malnutrition
- lymphoma , sarcoidosis
- improper injection techniques
- atypical Mycobacterial infection

Tubercular testing also used in the diagnosis of :

- active infection in infants and young children
- measuring TB prevalence in community

RADIOLOGICAL INVESTIGATIONS:

X - RAY NECK: look for calcification [34]

X – RAY CHEST : to rule out pulmonary tuberculosis

FNAC OF LYMPH NODE:

Smear is stained by Ziehl nelson technique, at least 10000 cells are required for positive result .Histopathology shows : Tubercle (granuloma) comprising area of caseation surrounded by epitheloid histiocytes ,giant cell, lymphocytes and plasma cells.

BIOPSY OF THE LYMPH NODE:

It is a superior and reliable investigation for confirmation of diagnosis .

MACROSCOPIC FEATURES:

EARLY STAGE: Translucent and grey patches

ADVANCED STAGE: opaque and yellow due to caseation

MICROSCOPIC FEATURES:[35]

At early stage ,it shows epitheloid cells and giant cells with peripheral nuclei. At the end of first week lymphocytes with dark nuclei and scanty cytoplasm .

at the end of third week caseation occurs at the centre with surrounding epitheloid and giant cells , around which are lymphocytes and plasma cells .

CULTURE:

Lowenstein Jensen media is used for culture . It takes 6 weeks for positive culture. Serenity medium takes 5 days . Middle brook 7H12 and 7H13 (BACTEC 12B AND 13B) are most sensitive culture media .BACTEC is an automated method which provides result in 4 days , radioactive C14 labelled broth culture is used. mycobacteria metabolises the culture to liberate radioactive co2.

SEROLOGY:

Passive hemagglutination may be positive in advanced disease only and negative in early stage , but no serological investigation is diagnostic of pulmonary tuberculosis . [36]

SPUTUM FOR AFB :

Three samples are taken . First and third day sample are spot samples .Second day sample is a early morning sample .Smear stained by Ziehl nelson method . Atleast 2 smear positivity is required to declare positive result. Smear is positive only when there is > 1-9 bacilli / 100 field.

MOLECULAR TESTING :

PCR testing is a very fast method . It is used to demonstrate Mycobacterial DNA fragments .It is a very useful test in a suspected case of Mycobacterial cervical lymphadenitis . 10 microorganisms are sufficient for positive PCR . PCR testing can also be done on the FNAC or biopsy smear and hence the need for open biopsy is reduced. Sensitivity of the test is 43 to 84% . specificity is 75 to 100%. PCR is done in the smear and culture negative cases.

IMMUNOPROPHYLAXIS

BACILLE CALMETTE GUERIN – (BCG) was identified by Calmette and Guerin (1921). It is a live attenuated vaccine ,[37] available in a freeze dried form which is stable compared to liquid .It is given intradermally at birth in infants . In developing countries ,it is given for children under 15 years.

It contains Mycobacterium bovis strain . It is attenuated by 239 serial subculture in glycerine bile potato medium for a period of 13 years. It induces delayed hypersensitivity reaction, hence shows positive tuberculin reaction. It provides immunity for 10 to 15 years. Natural TB infection also provides immunity for the same duration but there is progression of disease when there is reactivation but this does not occur in immunization.

EFFICACY:

It varies between 80% to no immune response .It depends on various factors .

- prevalence and virulence of infection
- type and potency of the vaccine used.

- Age and nutritional status of the individual.

COMPLICATIONS:[38]

LOCAL: abscess , indolent ulcer , keloid , tuberculids – it is satellite nodule developed adjacent to vaccinated site,lupus vulgaris ,lupoid reaction .

REGIONAL: axillary lymphadenitis or abscess of the lymph node.

SYSTEMIC: fever ,mediastinal lymphadenitis , erythema nodosum , otitis media , fatal meningitis in rare cases.

Vaccination confers immunity to skeletal , meningeal and military tuberculosis. It does not provide immunity for pulmonary TB especially in infants and young children , but the disease takes milder course.

DIFFERENTIAL DIAGNOSIS:

Cervical lymphadenitis can be due to:

- Viral
- Bacterial- tubercular, atypical tuberculosis, syphilis, brucellosis, non-Specific

- Fungal-fungal granuloma
- Organic-drug reaction, reactive follicular hyperplasia
- Malignancy-which includes primary (Hodgkin's lymphoma, lymphosarcoma) and secondary (with primaries in the neck, thorax and abdomen)
- Others

These can produce both acute or chronic lymphadenitis similar to tuberculosis.

SARCOIDOSIS:

Granulomatous lesions are commonly seen in the lungs, lymph nodes, skin, eye, liver, spleen, salivary glands, heart, skeleton and nervous system. The lymph node involvement is restricted usually to the superficial group, about 2-3cm in size, non-tender and no periadenitis. The lymph node involvement in sarcoidosis is differentiated from tuberculous lymph node by the absence of caseation. Mantoux test is negative and diagnosis is confirmed by biopsy and Kveim test.

CAT- SCRATCH DISEASE:

Caused by B.henselae where initially there is enlargement of either the axillary or the cervical lymph nodes. Primary skin lesion is indicated by a red papule appearing between 7-12 days following contact. Diagnosis is confirmed by skin

testing and biopsy of lymph nodes which reveals lesion of histiocytic proliferation and follicular hyperplasia.

INFECTIOUS MONONUCLEOSIS: [39]

Otherwise termed the GLANDULAR FEVER.

Rickettsia group of organisms causes this disease which is characterised by fever, cervical lymph node enlargement (posterior triangle group of lymph nodes are equally involved) which is painful and tender, sore throat, splenomegaly, hepatomegaly and skin rashes. Diagnosis is confirmed by blood investigations which include Paul-Bunnel test where there is an increase of agglutinins for sheep RBCs during the acute phase of the disease and atypical lymphocytosis. Epstein-Barr virus antibodies are detected by immunofluorescence and ELISA.

SYPHILITIC LYMPHADENITIS:

PRIMARY STAGE:

Genital chancres and groin nodal enlargement.

SECONDARY STAGE:

Generalised involvement of the lymph nodes especially the epitrochlear and occipital group of lymph nodes which are painless, discrete, firm and shotty.

TERTIARY STAGE:

Lymph nodes are rarely involved.

Presence of Treponema pallidum in dark ground illumination from the primary

lesion and positive W.R and Kahn tests confirms the diagnosis.

Specific tests includes FTA-ABS, MHA-TP. [40]

BRUCELLOSIS:

Otherwise termed the UNDULANT FEVER.

Brucella are small, gram negative, non-motile coccobacilli which presents with fever, malaise, headache, myalgia and GI disturbances with enlargement and tenderness of the spleen, liver and lymph nodes. Blood investigations which shows hypochromic anaemia,leukopaenia, agglutination and culture of the tissue confirms the diagnosis. Lymph node biopsy reveals features of either tuberculosis or Hodgkin's disease. ELISA is used to detect either the IgG or the IgM antibodies.

TULAREMIA:

It is highly infectious disease usually occurring in the farmers, hunters, butchers, people who handle the contaminated skin or the internal organs of the infected rabbits. It presents with symptoms like headache, feeling of cold and pyrexial episode and resembles a plaque with ulcer at the site of infection, enlargement of the regional lymph nodes where it becomes indistinguishable from tuberculosis showing caseation, necrosis, epitheloid and giant cell formation. The disease is caused by Pastuerella tularaemia. [41] Diagnosis is confirmed by the isolation of the organisms from the guinea pigs and rising titre of the agglutinins against tularaemia in the serum.

TOXOPLASMOSIS:

It is caused by an intra cellular parasite Toxoplasma gondii. It exists in 4 forms which includes cerebrospinal, lymphatic, exanthematous and the latent form out

of which lymphatic form is important. It presents with the enlargement of one or more group of lymph nodes with fever of several weeks of duration and marked constitutional symptoms. Diagnosis is confirmed by complement fixation tests, neutralising antibody test and toxoplasmosis skin test.

FUNGAL:

A chronic granulomatous process with/ without caseation necrosis is noted. Organisms like coccidiodomyces and blastomyces are identified with Pas Grithley and Gomori methenamine silver.

HODGKIN's DISEASE:

Lymph nodes are markedly enlarged with hard rubbery consistency where more than one group is involved in different regions of the body with no features of matting and periadenitis. Splenomegaly is present. Nodal enlargement in the axilla and mediastinum will occur in the course of several weeks or months. As in tuberculosis, non-specific inflammation or in secondary deposits, the nodes do not break or suppurate but in this case the nodes break and suppurate. Biopsy gives conclusive evidence.

Severe pain at the site of the disease after ingestion of alcohol, pyrexia, leucocytosis, eosinophilia are the common features.

SECONDARY CARCINOMA:

Due to the lymphatic metastasis, there is enlargement of the cervical group of lymph nodes in the elderly. The primary carcinoma is most often in the

mouth(tongue), lips, pharynx etc.,Malignant melanoma commonly leads to the secondary involvement of the lymph nodes.

TREATMENT: [42]

CHEMOTHERAPY:

Treatment of tuberculous lymphadenitis includes either medical or surgery but with the advancements of short course chemotherapy where the duration of therapy has reduced from 18months to 6months and a cure rate of 95%, the role of surgery is diminishing. Treatment is based on the WHO guidelines, where the National Tuberculosis Programme worldwide follows DOTS approach which provides intermittent chemotherapy for patients with tuberculous lymphadenitis. Cat I treatment is for those who are smear positive tuberculous lymphadenitis with pulmonary involvement or are severely ill.

Anti-tuberculous treatment was introduced in the early 1950s. The tuberculous research centre in Chennai in 1990 reported results of a supervised short course intermittent chemotherapy (6moths) consisting of streptomycin, rifampicin, isoniazid, pyrazinamide 3times a week for 2 months followed by streptomycin and isoniazid twice a week for 4 months on OP basis in children with lymph node tuberculosis and a favourable clinical response was noted at the end of the treatment. Hence in children, short course chemotherapy of 6 months is effective in treating tuberculous lymphadenitis.

FACTORS WHICH MAKES TUBERCULOSIS DIFFICULT TO TREAT:

- Caseation, an unique feature of tuberculosis, keeps the disease dormant where the organisms multiply preventing the contact of the environment of the body with the chemotherapeutic drugs.

- Fibrosis reducing the blood supply.
- The poor penetrating effect of the chemotherapeutic agents which allows the tubercle bacilli to remain viable and multiply even after being ingested by the macrophages.
- Quick development of the resistance when administered improperly.
- Prolonged duration of the treatment.
- Expensive treatment.

ANTI-TUBERCULOUS DRUGS:

BACTERICIDAL DRUGS:

ISONIAZID (1952):

ISONICOTINIC ACID HYDRAZIDE

The drug acting on both the extra cellular and intra cellular bacilli kills the fast multiplying organisms while the quiescent ones are only inhibited. It is considered the most effective drug against Mycobacterium tuberculosis.

MECHANISM OF ACTION:

Exact mechanism not known but probably due to the inhibition of a unique fatty acid component of a bacterial cell wall, MYCOLIC ACID. [43]

ABSORPTION AND RATE:

The drug penetrates through the caseous material with a peak plasma concentration of 3-5mg/ml developing 1-2hours following an oral ingestion. Being given orally/ parenterally, the drug is readily absorbed maintaining the blood level and therapeutic concentration in the CSF. The drug is metabolised by the liver by acetylation 75-95% of the dose is excreted in the urine in 24hours.

DOSAGE:

Daily dose of the drug is 5mg/kg with a maximum of 300mg and in children <4years , dose is 10mg/kg/day.

Tablet: 50mg, 100mg, 300mg.

Syrup: 10mg/ml.

Injection: 10mg/ml.

Pyridoxine added in a dose of 10-50 mg/day to prevent peripheral neuritis caused due to ISONIAZID.

Side effects includes hepatitis, rash, psychosis, acne and fever.

RIFAMPICIN:

It is a semi synthetic derivative of rifamycin-B.

This complex macrocyclic antibiotic is derived from Streptomyces mediterranei.

Affects both the extra-cellular and intra-cellular bacilli and acts on

slowly/intermittently dividing organisms.

MECHANISM OF ACTION:

Inhibits DNA dependant RNA polymerase suppressing the initiation of chain formation in RNA synthesis.

ABSORPTION AND FATE:

Penetrates all the body fluids, absorbed orally with a peak concentration in 2-4hours and therapeutic levels in CSF with rapid elimination in bile by deacetylation.

DOSAGE:

>50kg – 450mg/day [(44)]

Children – 10mg/kg/day.

CAPSULE:

150, 300, 450 and 600mg.

ADVERSE REACTIONS:

Flu like symptoms (fever, chills, headache and malaise), GI disturbances (nausea, vomiting and abdominal cramps), flushing, pruritis, rash, hepatitis, orange pink discolouration of body secretions.

STREPTOMYCIN:

Till 1952,it was the only effective drug available to treat tuberculosis. It crosses the placenta and fetal serum levels are half of those in the maternal blood reaching a peak concentration of 1hour when administered i.m.

It is excreted by glomerular filtration and hence the dosage should be titrated in renal failure.

DOSAGE IN VARIOUS AGE GROUPS:

1gm - young individuals

0.75gm -40-60 years age group

0.5gm ->60years.

SIDE EFFECTS:

Ototoxicity (vestibular damage), anaphylaxis, renal tubular damage, blood dyscrasias.

PYRAZINAMIDE:

It is the synthetic pyrazine analogue of nicotinamide. It is lethal to the intra cellular bacilli exhibiting bactericidal activity only in acidic pH. Interacts with the fatty acid synthase and inhibits the mycolic acid synthesis. It is well absorbed from the GIT and widely distributed. Oral administration of 1gm produces plasma concentration of 45mg/ml at 2hours. Drug is excreted by glomerular filtration. The drug is hydrolysed and hydroxylated to 5-OH pyrazinoic acid which is the major excretory product available as 500mg/ 750mg tablets. Daily dose is 20-35mg/kg orally. Maximum dose is 3gm/day regardless of the weight. Side effects includes hepatotoxicity, gout, arthralgias, flushing, rashes and fever.

ETHAMBUTOL:

It is a bacteriostatic drug but in high concentrations it is bactericidal. The exact mechanism is not known. It inhibits the arabinosyl transferases involved in arabinogalactan synthesis and interferes with the mycolic acid incorporation into the Mycobacterial cell wall. About 75-80% of the oral drug is absorbed from the gastrointestinal tract and the peak plasma concentration is after

2-4hours.

The half life of the drug is 3-4hours. Two thirds is excreted in the urine and 15% is excreted in the form of the metabolites.

Tablet of 400mg and 800mg are available. Usual dose is 25mg/kg/day. Side effects includes ocular toxicity, diminished visual activity, hyperuricaemia, fever and rash.

DIFFERENT REGIMENS:

6 MONTH REGIME:

Initial phase of 2months – Ethambutol/Streptomycin + Isoniazid + Rifampicin + Pyrazinamide

Continuation phase of 4months – Isoniazid + Rifampicin

9 MONTH REGIME:

Initial phase of 2 months – Ethambutol/Streptomycin + Isoniazid + Rifampicin

Continuation phase of 7months – Isoniazid +Rifampicin

12 MONTH REGIME:

STM 1gm i.m for 3months with a total dose not exceeding 45gms.

It is given as follows:

Every day for the first 2weeks, every other day 1month, and every 3days for 1.5months.

RIF (600mg/day, p.o.) for 6months

INH (300mg/day p.o.) for 12 months or 18 months.

EMB (900mg/day p.o) for 12 months or 18 months.

PARA- AMINO SALICYLIC ACID:

This drug is rarely used with the advancement of short course chemotherapy. It

is not used because of the bulk and the unpleasant sensation.

THIOACETAZONE:

It is abandoned in developing countries but it is also used in some developing countries because it is cheap. It is available as 150 mg tablet in combination with Isoniazid. Usual dose is 150mg/day. Side effects are hypersensitivity reactions as Steven Johnson syndrome which includes the symptoms such as fever, rash, joint pain. This is a severe life threatening complication.

OTHERS:

Cycloserine, Kanamycin, Ethionamide, Capreomycin are the second line drugs.

NEWER ANTI-TUBERCULAR DRUGS:

The fluoroquinolone group of drugs are quite effective out of which ofloxacin is most effective which showed a therapeutic effect on mice. Other drugs like Sparfloxacin and Fleroxacin demonstrate anti tubercular activity. Rifabutin, a drug belonging to rifamycetins is an effective drug. These drugs have been effective in experimental animals and human cases but is not recommended for general use.

Multi- drug resistant tuberculosis MDR-TB is defined as tuberculosis resistant to at least Isoniazid and Rifampicin. When multi-drug resistance to the drugs involves at least one drug in each group then it is termed as the extensive drug resistant tuberculosis XDR-TB. MDR-TB can also involve the second line drugs which includes the fluoroquinolones and the injectable drugs. Treatment of MDR-TB must be done on the basis of sensitivity testing.

RISK FACTORS FOR MDR-TB INCLUDES:

- Failed TB treatment
- Failure to respond to TB treatment
- Relapse following standard TB treatment
- Previous incarceration
- HIV infection

If the treatment is for a suspected case of MDR-TB, the patient is started on SHREZ (Streptomycin+ Isonicotine hydrazine+ Rifampicin+ Ethambutol+ pyrazinamide) + MXF + Cycloserine until the report of the sensitivity testing is received.

ASSESSMENT OF PROGRESS:

Improvement of ESR is of prognostic value.

Periodic check-up has to be done. If the patient shows no improvement when under treatment then the doctor should find the cause and treat accordingly. Before stopping the treatment, it is best to investigate twice since staring the treatment all over again is difficult.

Treatment categories in DOTS chemotherapy in India:

Table 1 – Categories in DOTS chemotherapy

TREATMENT CATEGORY	TYPE OF THE PATIENT	TREATMENT REGIMEN
CATEGORY I	New sputum smear-positive	2 (HRZE)3 4 (HR)3
	Seriously ill sputum smear-negative Seriously ill extra-pulmonary	
	New sputum smear-negative, not seriously ill New extra- pulmonary, not seriously ill	
CATEGORY II	Sputum smear positive Relapse, failure and default	2 (HRZES)3 1 (HRZE)3 5 (HRE)3

The number before the letters in the treatment regimen indicates the number of months of treatment. The number after the letters indicates the number of doses per week.

H – Isoniazid (600mg)

R – Rifampicin (450mg)

Z – Pyrazinamide (1500mg)

E – Ethambutol (1200mg)

S – Streptomycin (750mg)

Patients more than 60kg receive an additional dose of rifampicin 150mg.

ROLE OF SURGERY

The role of surgery is decreasing with the advent of effective anti-tubercular therapy.

Surgical procedures are ;

1. Biopsy.
2. Excision
3. Aspiration

4. Excision of sinus and scar.
5. Curettage.
6. Excision of cold abscess.
7. Tonsillectomy.

BIOPSY:

It is usually done for superficial mobile lymph node. Biopsy is for confirmation of diagnosis before starting the short course chemotherapy.

EXCISION:

Excision of a mobile node with an intact capsule shortens the duration of treatment, lessens the chance of recurrence and disfigurements and is considered only when patient is not responding to chemotherapy. The lymph node should be removed in toto.

Excision is considered in case of matted nodes, cold abscess, discharging sinus and in patients who respond well to anti-tubercular drugs.

Contradiction to excision is the presence of secondary infection , multiple node involvement, tender nodes and the presence of active lesion elsewhere in the body.

Excision is not a contraindication in children but is better avoided in children.

ASPIRATION:

The presence of a big cold abscess, the chances of bursting open of the abscess, the presence of the disease in the acute phase are indications of aspiration from a non-dependent area with wide bore needle. Aspiration can also be repeated which makes excision easy.

INCISION AND DRAINAGE:

It facilitates the spread of infection.

CURETTAGE:

Curettage is considered when the lesion is close proximity to the nerve or in the presence of extensive skin necrosis. It has 70% cure rate.

OPERATION:

The presence of large, matted glands mandates removal but the removal is dangerous if the node is attached to the wall of IJV. The nodes are removed through a horizontal neck incision. If the bleeding of IJV is encountered, it is stopped either by finger pressure or by dissection of the vein above and below the matted glands, ligation and excision of the segment of the vein along with the glands.

Functional neck dissection involves fat removal , associated lymph nodes in the posterior and anterior triangles of the neck and is performed only when the glands and vein is involved extensively. The sternocleidomastoid muscle, accessory nerve and the IJV are to be preserved.

Removal of the gland should be followed with chemotherapy, otherwise sinuses form with persistent drainage and scars. Mostly the infection is from the ipsilateral tonsil , which mandates the removal of tonsil also.

MATERIALS AND METHODS.

The cases used for this study consists of elaborate study of 70 cervical tubercular lymphadenitis patients, diagnosed and treated at Government Kilpauk medical college , Chennai during the period August 2013 to August 2014. The patients were mainly from in and around Chetpet area.

The paper consists of seventy cases selected conveniently. For statistical evaluation the data from my study is compared with that of data from literature.

METHODS:

The case details were recorded as depicted in the proforma. The data are enclosed in the master chart for analysis.

INCLUSION CRITERIA:

1. Patients who were above 12 years of age and also confirmed to have cervical tubercular lymphadenopathy both clinically and pathologically.
2. Patients who gave consent for investigations and willing to undergo surgery if required.

EXCLUSION CRITERIA:

1. Patients less than 12 years of age.
2. Pregnant women with cervical lymphadenopathy.
3. Psychiatric patients.

4. Patients not willing to undergo investigations and surgery if required.

The patients are concluded to have cervical tubercular lymphadenopathy based on history, clinical examination and confirmed by either FNAC or histopathological examination.

Anti-tubercular chemotherapy was started only after confirmation of the diagnosis.

Investigations such as Hb%, total count and differential count , ESR and chest X-ray were done routinely. X-ray chest was done to rule out coexisting pulmonary lesion.

Haematinics were given to anaemic patients to improve the general status.

For the patients who underwent surgery , the parts were prepared one night before and kept on nil per oral for 12 hours . After getting the written consent from patients, they were operated under general anaesthesia. Antibiotics were given one hour before surgery prophylactically.

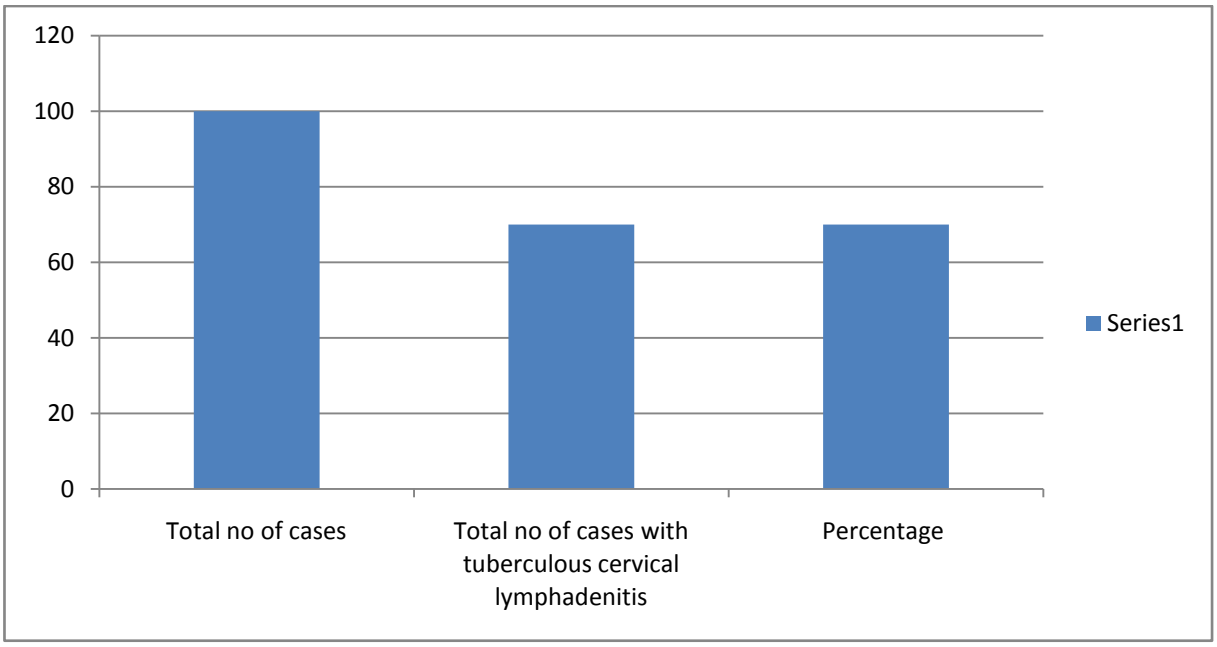
Follow of the patients was done from first post-operative day to the day of discharge. Once the diagnosis was confirmed the patients were advised to take short course anti-tubercular therapy for 6 months.

RESULTS

Table 2- Incidence of tuberculous cervical lymphadenitis

Total no of cases	Total no of cases with tuberculous cervical lymphadenitis	Percentage
100	70	70

Graph 1 – Showing incidence of tuberculous cervical lymphadenopathy

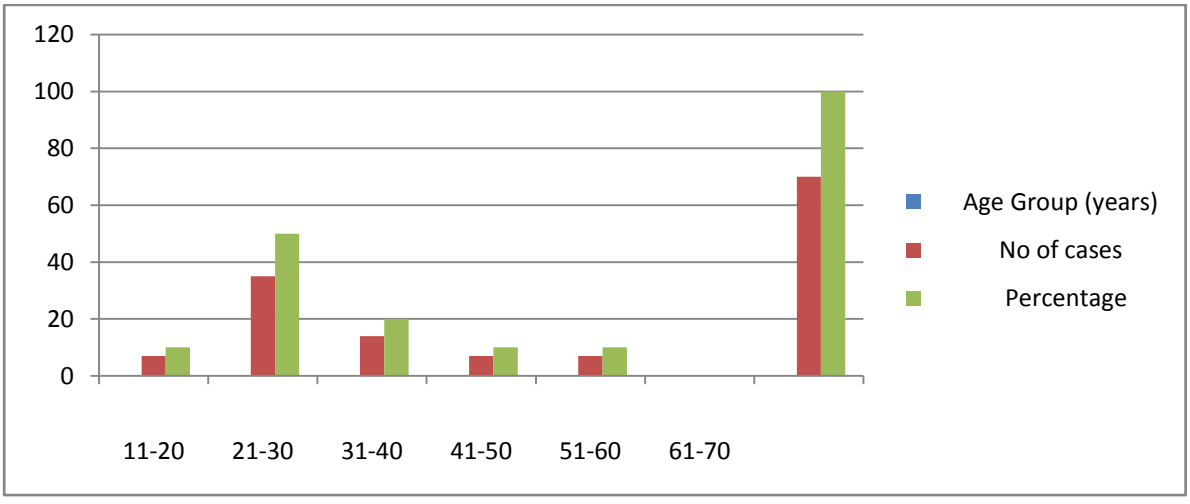


The most dreadful disease in India is tuberculosis which is the most common cause of cervical lymphadenopathy accounting for about 70 cases out of the 100 cases in the present study. Other causes of cervical lymphadenopathy were non-specific lymphadenitis (28cases), lymphoma (2cases).

Table 3 – Age Incidence

Age Group (years)	No of cases	Percentage
11-20	7	10
21-30	35	50
31-40	14	20
41-50	7	10
51-60	7	10
61-70	-	-
TOTAL	70	100

Graph 2 – Showing age incidence



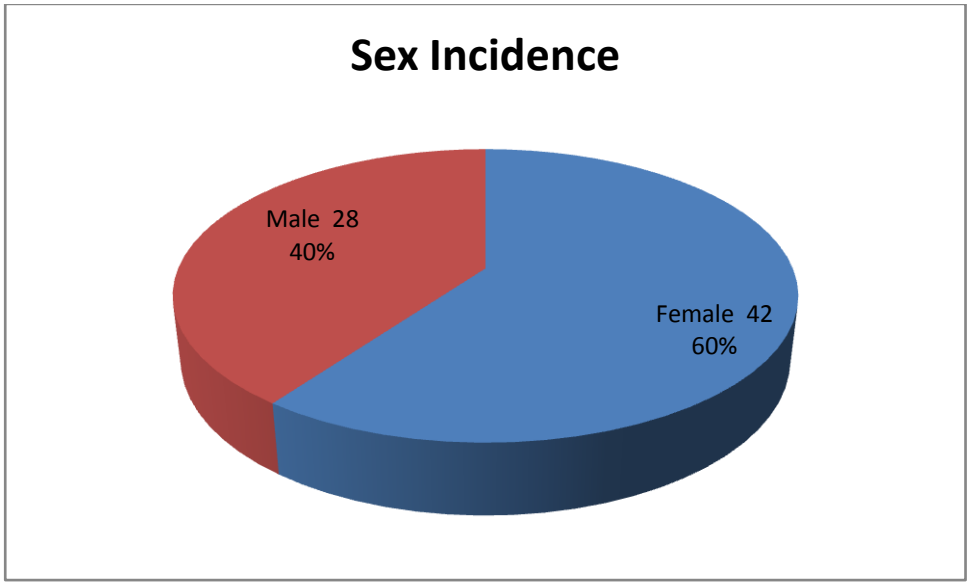
The disease occurs mostly in the age group of 21-30 years. No cases were

reported in the age group of 61-70 years in the present study. The youngest in the present series is 12 years old.

Table 4– Sex Incidence

Sex	No of cases	Percentage
Female	42	60
Male	28	40

Graph 3 – Showing sex incidence

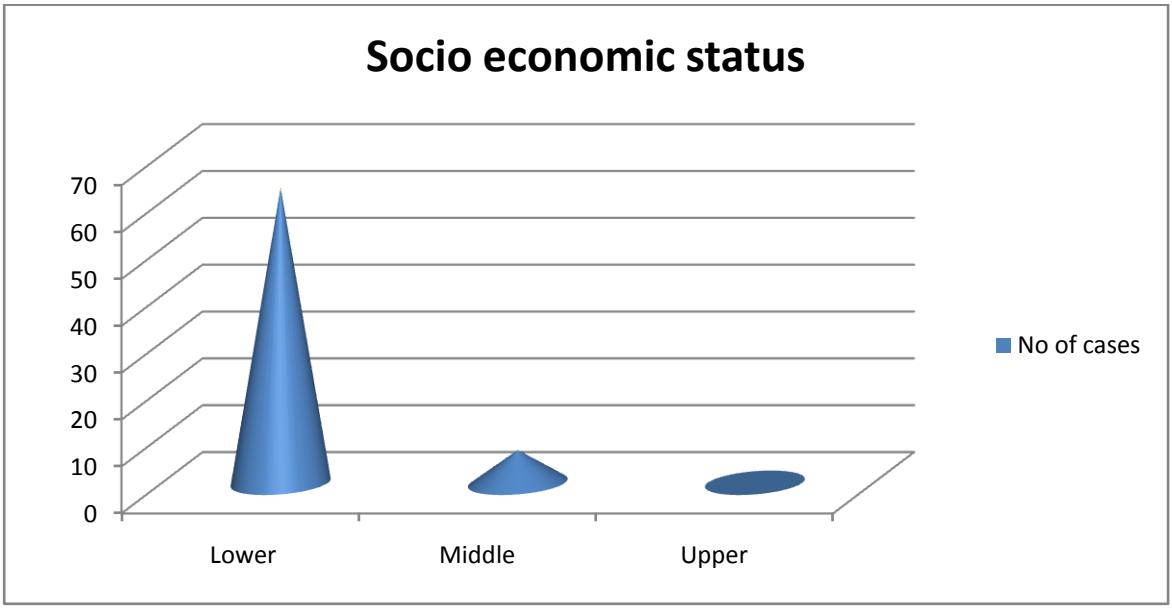


There is a higher incidence in females. There are 28 male cases and 42 female cases according to the present study with a female to male ratio of 1.5:1.

Table 5 – Socio Economic status

Socio economic status	No of cases	Percentage
Lower	63	90
Middle	7	10
Upper	-	-

Graph 4 – Showing socio – economic incidence

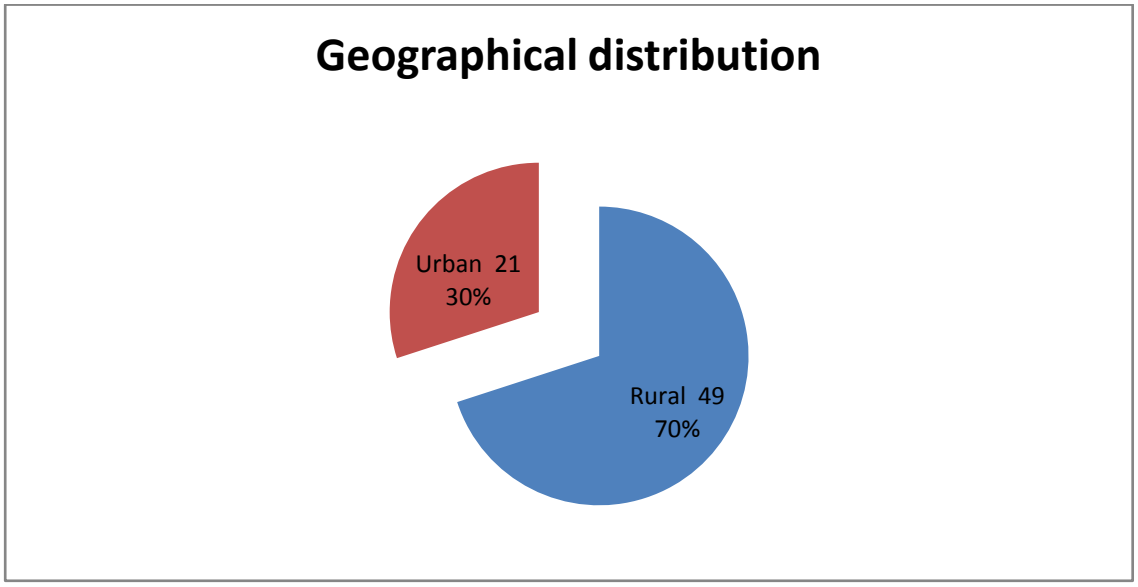


Based on Kuppusamy's socio – economic scale of classification, study cases were divided into lower, middle and upper socio-economic status. Tuberculosis mainly affected lower group of people comprising about 90% of cases.

Table 6- Geographical Distribution

Area	No of cases	Percentage
Rural	49	70
Urban	21	30

Graph 5 – Showing geographical distribution

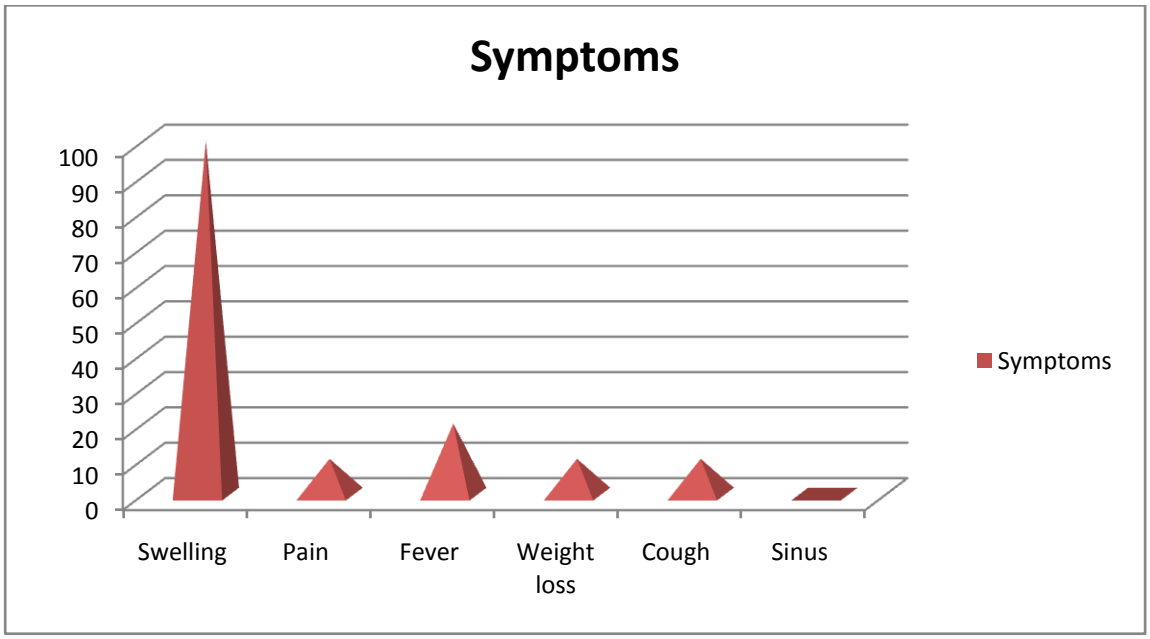


Tuberculous cervical lymphadenitis is more common in people living in the rural population comprising about 60% of the population.

Table 7 – Clinical Presentation

Symptoms	No of cases	Percentage
Swelling	70	100
Pain	7	10
Fever	14	20
Weight loss	7	10
Cough	7	10
Sinus	-	-

Graph 6 – Showing clinical presentation



All the 70 cases (100%) presented with swelling in the neck. 7 cases (10%) presented with pain, 7 cases (10%) presented with weight loss and 7cases presented with cough. About 14cases (20%) presented with fever.

Table 8– Duration of the disease

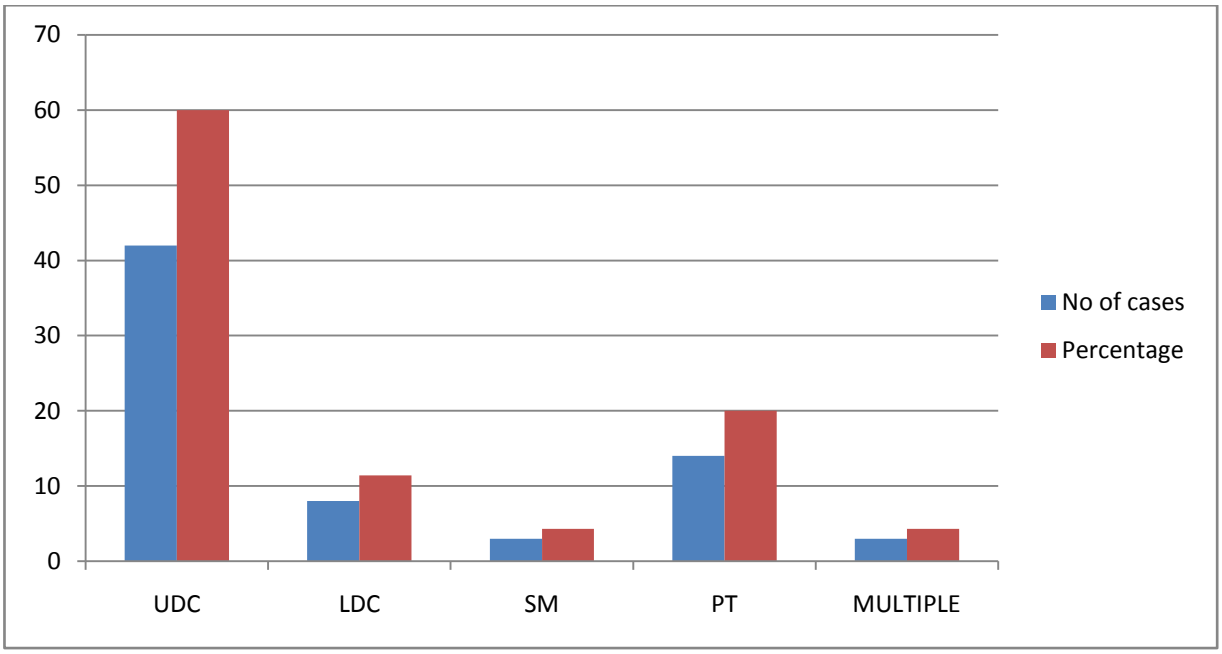
Duration	No of cases	Percentage
< 3months	49	70
>3 months	21	30

49 cases (70%) presented within the duration of 3 months.

Table 9– group of lymph nodes involved

Lymph node group	No of cases	Percentage
UDC	42	60
LDC	8	11.4
SM	3	4.3
PT	14	20
MULTIPLE	3	4.3

Graph 7 – Showing group of lymph nodes involved



Most commonly affected is the upper deep cervical group of lymph nodes involved in about 42cases (60%). Posterior triangle group of nodes were involved in about 14cases (20%). 8cases (11.4%) presented with the lower deep cervical involvement. Submandibular and multiple nodal involvement were the least constituting about 3%.

Table 10– Side of the lymph nodes involved

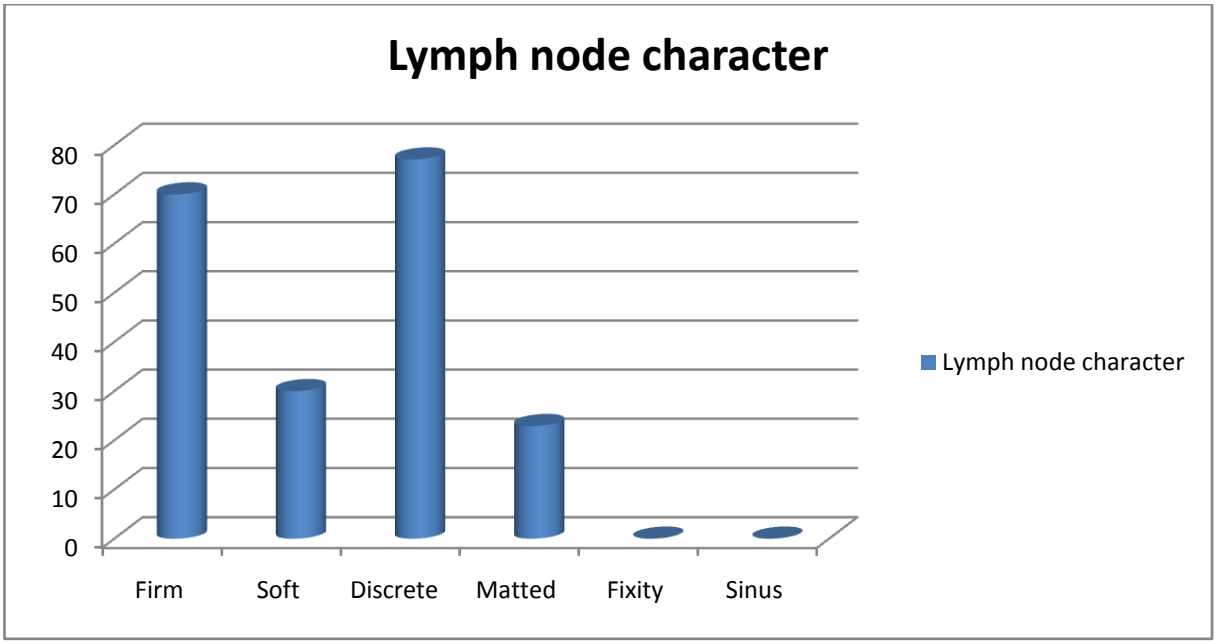
Side involved	No of cases	Percentage
Right	24	34.3
Left	40	57.1
Bilateral	6	8.6

Left side nodes (40cases) are most commonly affected in tuberculosis than the right side (24 cases).

Table 11– Lymph node character

Lymph node character	No of cases	Percentage
Firm	49	70
Soft	21	30
Discrete	54	77.1
Matted	16	22.9
Fixity	-	-
Sinus	-	-

Graph 8 – Showing lymph node character

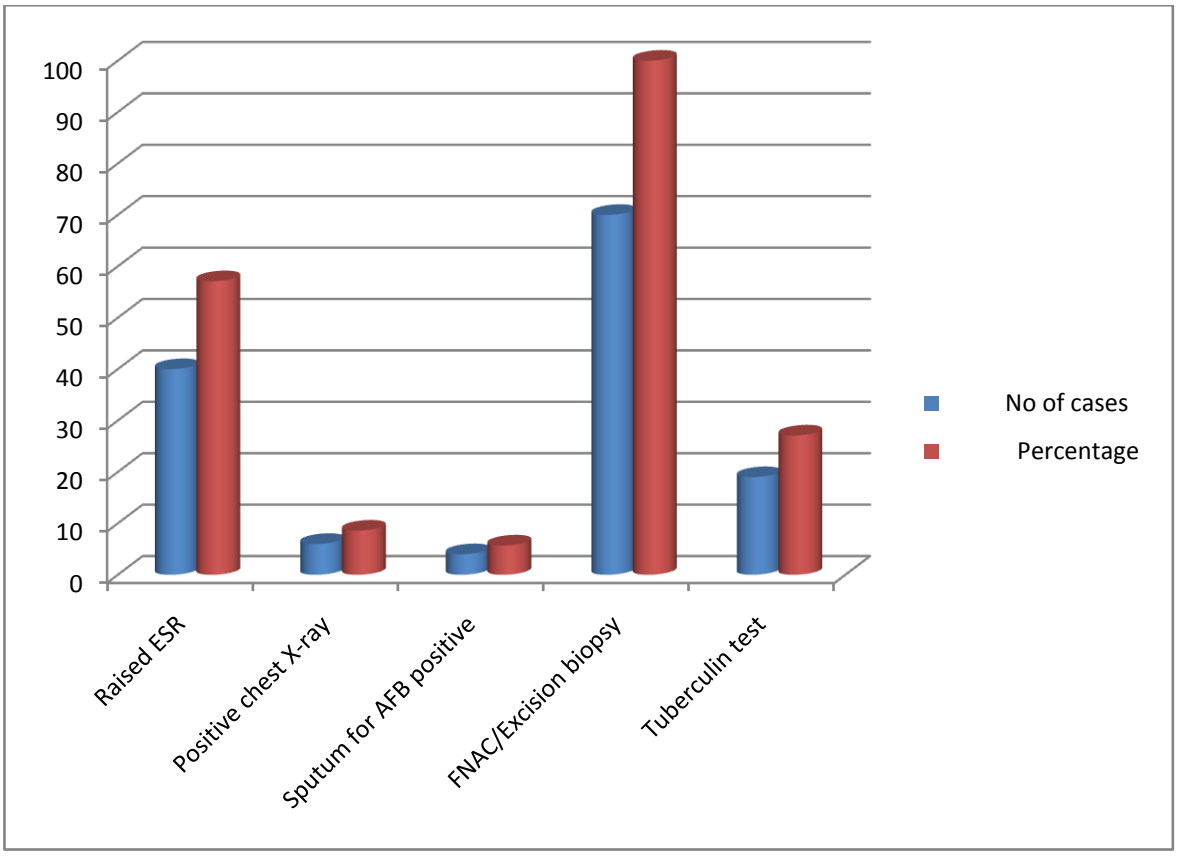


Firm lymph nodes were noted in about 49 cases (70%). 77.1% of the cases presented with discrete nodes. Matted nodes were noted in 16 cases (22.9%). None of them presented with fixed nodes or sinus according to the present study.

Table 12 - Investigations

Investigations	No of cases	Percentage
Raised ESR	40	57.1
Positive chest X-ray	6	8.6
Sputum for AFB positive	4	5.7
FNAC/Excision biopsy	70	100
Tuberculin test	19	27.1

Graph 9 – Showing Investigations



40 cases presented with raised ESR (57.1%, >30mm at the end of 1hour). All cases were confirmed by FNAC/Excision biopsy (100%). Features of pulmonary tuberculosis on chest X-ray were positive in 6cases and sputum AFB was positive in 4 cases. Tuberculin test was positive in 19 cases (27.1%).

Table 13– Results of FNAC and Excision Biopsy

Investigations	Caseating granuloma		Inconclusive	
	No of cases	Percentage	No of cases	Percentage
FNAC	58	82.9	12	17.1
Excision Biopsy	12	100	-	-

Fig 9 – Presentation of a tuberculous cervical lymph node

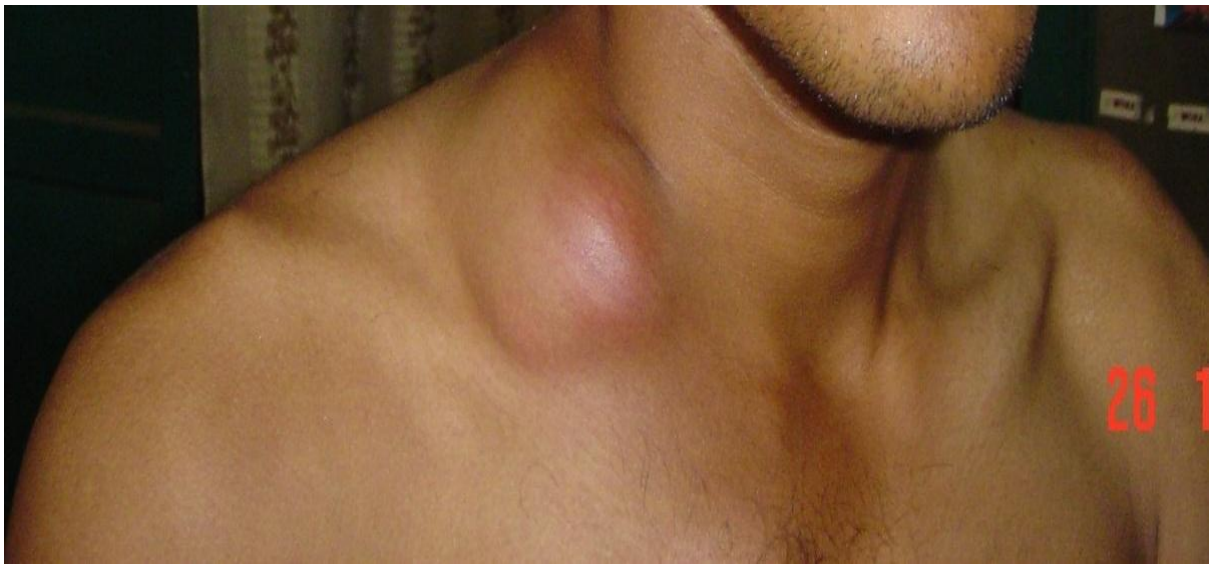


Fig 10 – Cold aspirate

All the 70 cases studied in the present series were put on short course chemotherapy after thorough confirmation by FNAC or histopathological examination of the excised lymph node. 58 cases were confirmed by FNAC which revealed caseating granuloma. 12 cases (17.1%) were inconclusive and these 12 cases were subjected to excision biopsy which showed caseating granuloma. None of them presented with cold abscess.

DISCUSSION

70 cases of tuberculous cervical lymphadenitis in the present series is compared with the other studies in the literature.

Table 14– Comparison of Age incidence with the other studies

Series	Age in years					
	11-20	21-30	31-40	41-50	51-60	61-70
Present series	7	35	14	7	7	-
Kumar Biswas series (45)	10	9	3	3	-	-
Muhammed MM series(46)	44	31	11	8	2	-

From the above table, it is clear that the most commonly affected population are in the age group of 21-30 years. It is compared with the other studies in the same table.

In Kumar Biswas study of 30 cases, 9 cases are in the age group between 21-30 years. In Muhammed Mudassar Majeed study of 96 cases, 31 cases are in the age group of 21-30 years. In the present series studied, 35 cases are in the age group of 21-30 years which is almost equal to the studies in the literature.

In this age group, the lymphatics play an important role with the lymph nodes acting as a powerful second line of defense against the infection. Poor nourishment with malnutrition acts as a predisposing factor in this age group.

None of them were in the age group of 61-70 years in the present study. The youngest in the present series is 11 years and the oldest is years.

Table 15 – Comparison of sex incidence with the other studies

Series	Male		Female	
	No of cases	Percentage	No of cases	Percentage
Present series	28	40	42	60
Kumar Biswas series	11	36.6	19	63.4
Muhammad MM series	38	38	62	62
Salman M et al study(47)	21	42	29	58

In the present study, the female to male ratio is 1.5:1 which is compared with the other series available in the literature. The ratio according to the Kumar Biswas study is 1.73:1 whereas in Muhammad Mudassar Majeed series it is 1.63:1. The present study is quite similar to the Salman M et al study where the ratio is 1.4:1.

The reason behind the female preponderance of the disease is due to the under nutrition, repeated and early pregnancies etc.,

Table 16 – Comparison of socio – economic status in various studies

Series	Low SES		Middle SES		High SES	
	No of cases	Percentage	No of cases	Percentage	No of cases	Percentage
Present series	63	90	7	10	-	-
Kumar Biswas series	18	60	9	30	3	10

The incidence of tuberculous cervical lymphadenitis is more common in the low socio- economic status which accounts for about 90% as compared to 60% in the Kumar Biswas series.

Overcrowding, poor ventilation, poverty, malnutrition and unhygienic environment are the predisposing factors for the increased incidence in the low socio-economic status group.

Table 17 – Comparison of clinical presentation in various studies

Symptoms	Present series		Jha BC et al study	
	No of cases	Percentage	No of cases	Percentage
Swelling	70	100	53	94.6
Pain	7	10	4	7.3
Fever	14	20	6	10.7
Weight loss	7	10	8	14.3
Cough	7	10	6	10.7
Sinus	-	-	3	5.3
Others	-	-	6	10.7

The most common symptom with which the patient presents to the hospital is neck swelling which is seen in almost all the cases i.e. about 100%. 20% cases presented with fever, 10% cases presented with pain, weight loss and cough each. These results were compared with the Jha BC et al study which are almost similar to the present study which accounts for about 94.6% cases presenting with swelling, 7 cases presented with pain, weight loss and cough each.14 cases presented with fever.

Cough is due to the upper respiratory tract involvement and is not a specific feature of tuberculous adenitis.

Pain is due to the stretching of the capsule or the deep fascia of the neck by the enlarging lymph nodes. Pain is more if superadded secondary infection is present.

General symptoms like fever, loss of weight etc., are not specific for tubercular adenitis.

Table 18 – Comparison of Lymph node characters in various studies

Lymph node character	Present series		Salman M et al study		Indian Council of Medical Research Study (48)	
	No of cases	%	No of cases	%	No of cases	%
Firm consistency	49	70	33	66	116	65.5
Soft consistency	21	30	6	12	-	-
Discrete	54	77.1	9	28	79	54.1
Matted	16	22.9	36	72	81	85.3
Fixity	-	-	-	-	34	79.1
Sinus	-	-	3	6	-	-

Lymph node involvement is discrete in 54 cases (77.1%) and matted in 16 cases (22.9%). Lymph nodes are firm in consistency in 49 cases (70%) and soft in consistency in 21 cases (30%). The results of the present series are compared with those in the Salman M et al study and ICMR study as quoted in the table. ICMR study included more number of cases and hence the results were higher in it. In Salman M et al study, 66% of cases had nodes which were firm in consistency and 12% of cases presented with nodes which were soft in consistency. 72% (36 cases) presented with matted nodes and 28% (9 cases) presented with solitary nodes. 6% had sinus. None of the cases presented with fixed nodes or sinus in the present study.

Table 19 – Comparison of groups of lymph nodes involved in various studies

Group of lymph nodes involved	Present series in %	Kumar Biswas study in %	Maharjan M et al study in % (49)
Upper deep Cervical	60	33.3	16
Lower deep cervical	11.4	10	6
Submandibular	4.3	13.3	15
Supraclavicular	-	3.4	9
Posterior triangle	20	20	42
Multiple	4.3	20	-

The upper deep cervical group of lymph nodes are most commonly affected in the present series accounting for 60% which is more when compared to the Kumar Biswas study and Maharjan M et al study. The submandibular and the multiple nodal involvement are the least accounting for 4.3% each. There is no supraclavicular involvement in the present series. In Maharjan M et al study, posterior triangle group of nodes are more commonly affected followed by the upper deep cervical group of nodes.

The tubercle bacilli are filtered at the nose, oral cavity, pharynx and tonsils and ultimately drain into the upper deep cervical group of lymph nodes and hence the lesions are produced here. The other portal of entry is the adenoids which drain into the lower deep cervical group of lymph nodes which accounts for about (11,4% cases) in the present study. Multiple nodal involvement is present in about 4.3% cases according to the present study.

Table 20 – Comparison of associated pulmonary tuberculosis in various studies

	Present series	Maharjan M et al study	Jha BC et al study(1)
Total no of cases studied	70	83	60
Associated pulmonary tuberculosis in percentage	5	14	16

In the present series, about 5% cases had associated pulmonary tuberculosis which is about 14% in the Maharjan M et al study and 16% in the Jha BC et al study.

Table 21 – Comparison of investigations in various studies

Series	Raised ESR in percentage	Chest X-Ray in percentage	FNAC/Excision biopsy in percentage
Present series	57.1	8.6	100
Jha BC et al study	100	16	100
Maharjan M et al Study	79	14	100

Erythrocyte sedimentation rate was raised in 57.1% of the cases in the present series which is 100% and 79% respectively in the Jha BC et al study and Maharjan M et al study. Chest x-ray showed features of tuberculosis in 8.6% of the cases which is 16% and 14% respectively in the Jha BC et al study and Maharjan M et al study.

Fine Needle Aspiration Cytology was done in all the cases and found to be positive in all the cases which is the same as compared to the Jha BC et al study and Maharjan M et al study.

Table 22 – Comparison of results of FNAC and Excision biopsy in various studies

Study series	FNAC in %		Excision Biopsy in %
	Caseating granuloma	inconclusive	
Present series	82.9	17.1	100
Muhammad Mudassar series	69	31	-
Salman M et al study	82	-	18

In the present series, FNAC revealed caseating granuloma in 82.9% of cases compared to 69% in Muhammad Mudassar series and 82% in Salman M et al study. FNAC showed inconclusive evidence in 17.1% of the cases in the present series. According to the Salman M et al study, excision biopsy revealed caseating granuloma in 18% of the cases.

Table 23 - Comparison of treatment given in various studies

Treatment given	Present series in %	Salman M et al study in %	Maharjan M et al study in %	Jha BC et al study in %
Excision biopsy	17.1	18	4.8	7.14
FNAC	82.9	82	95	92.8
ATT	100	100	100	100
Non-dependent drainage	-	-	-	3.57

12 cases (17.1%) were subjected to excision biopsy of the affected lymph nodes according to the present study. 58 cases (82.9%) were diagnosed by FNAC alone. This result is similar to that of the Salman M et al study in which 82% of the cases were diagnosed by FNAC alone and 18% of the cases were diagnosed by excision biopsy. In Jha BC et al study, FNAC proved effective in 92.8% of the cases and excision biopsy was conducted in 7.14% of the cases. 3.57% of the cases underwent non-dependent aspiration of the nodes according to the Jha BC et al study. In the present study, none of the cases were subjected to non-dependant aspiration.

After confirmation of diagnosis, patients were put on Anti-tubercular drugs and were followed up. The cure rate was 100%. This result is also quite similar to that of the Jha BC et al study and Maharjan M et al study.

Over the years, because of the advancement in medical science, chemotherapy is more effective and has almost limited the role of surgery to drainage of the cold abscess, excision of the residual lymph node mass or scars, excision biopsy of the affected lymph nodes.

FOLLOW UP:

The cases which were put on anti-tubercular treatment were followed up at an interval of about 1-2 months from the commencement of ATT to a period of 6 months. During this period, cases were assessed by clinical examination and ESR examination. The decrease in the size of the swelling and the generalised improvement in the health status of the patients were studied and majority of the cases showed improvement within 4weeks of the start of the treatment.

CONCLUSION

Historical aspects on tubercular cervical lymphadenitis has been reviewed.

A brief description on the anatomy, physiology of the lymph nodes draining the neck, along with the distribution of the lymph nodes, with the aetiology, pathology, clinical aspects and the type of treatment are discussed in detail.

100 cases were taken, out of which 70 cases of cervical tuberculous lymphadenitis were studied, analysed in detail and the following conclusions were drawn.

The incidence of tuberculous cervical lymphadenopathy was maximum in the age group of 21-30 years (35 cases-50%). The youngest in the study is 12years.

The disease showed an increasing incidence in females as compared to the male patients with the female to male ratio being 1.5:1.

It is more common in the lower socio-economic status people (90%).

The disease shows increased incidence among the rural population which accounts for about 70% of the cases.

All the 70 cases presented with the complaint of swelling in the neck. 14 cases presented with fever, 7 cases presented with pain, weight loss and cough respectively.

The lymph node involvement was discrete in 77.1% of the cases and matted in

22.9% of the cases. 70% patients had firm consistency of the lymph nodes. The upper deep cervical nodes were involved in 60% which was the most common lymph node involved in tuberculous cervical lymphadenopathy. 57.1% presented with raised ESR . 8.6% had features suggestive of pulmonary tuberculosis on chest x-ray. 82.9% showed caseating granuloma on FNAC. FNAC was inconclusive in 17.1% of the cases who were then subsequently subjected to Excision Biopsy which proved positive for tuberculous cervical lymphadenopathy. Tuberculin test was positive in 27.1% of the cases.

The patients were then categorised as Category I or Category II patients and were put on Anti-tubercular treatment based on the histopathological (FNAC/Excision Biopsy) reports and were followed up for a period of 6 months. 65 cases were symptom free at the end of the study.

SUMMARY

This is a study involving 100 cases out of which70 cases proved to be tuberculous cervical lymphadenitis. These cases were studied at Govt. Kilpauk Medical College and Hospital for a period of 1 year from Aug2013-Aug2014.

1. Tuberculosis is more common in the 21-30 years age group which is about 35 cases (50%) according to the present study. The youngest in the present series is 12 years of age.
2. The disease showed a preponderance in females as compared to the male patients with the female to male ratio being 1.5:1. It is quite similar to that of the Salman M et al study where the female to male ratio is 1.4:1.
3. It is more common in the lower socio-economic status people (90%).
4. The disease shows increased incidence among the rural population which accounts for about 70% .
5. All the 70 cases presented with the complaint of swelling in the neck. 14cases presented with fever, 7cases presented with pain, weight loss and cough respectively. The left side nodes are most commonly involved (57.1%). The lymph node involvement was discrete in 77.1% and firm in 70% . 22.9% presented with matted lymph nodes according to the present study. The upper deep cervical nodes were involved in 60% which was the most common lymph node involved in tuberculous cervical lymphadenopathy.

6. 57.1% presented with raised ESR according to the present study. 8.6% had features suggestive of pulmonary tuberculosis on chest x-ray. 82.9% showed caseating granuloma on FNAC.
7. FNAC was inconclusive in 17.1% which were then subsequently subjected to Excision Biopsy which proved positive for tuberculous cervical lymphadenopathy.
8. Tuberculin test was positive in 27.1% .
9. The patients were then categorised as Category I or Category II patients and were put on Anti-tubercular treatment based on the histopathological (FNAC/Excision Biopsy) reports. For the first 2 months , patients under category I were given INH, Rifampicin, Ethambutol and pyrazinamide and under category III were given INH, pyrazinamide, rifampicin. In the continuation phase only INH and rifampicin were given to the patients for a period of 4 months. They were followed up for a period of 6 months. 65 cases were symptom free at the end of the study.

BIBLIOGRAPHY

1. Jha BC, Dass A, Nagarkar NM et-al. Cervical tuberculous lymphadenopathy: Changing clinical patterns and concepts in management. Postgrad Med J 2001; 77 (905): 185-7.
2. Iseman MD. A clinician's guide to tuberculosis. India: Lippincott Williams and Willkins: 2000. Chapter 1. Tuberculosis down through the centuries; p.2,4,7,14-17,51-53.
3. Pesani Edward L. History of tuberculosis: In tuberculosis a clinical handbook. Larry.I.Lutwic, 1 edition, London: Chapman and Hall, 1995:1-5.
4. Sadler TW. Langman's Medical Embryology. 10th edition. New Delhi: Lippincott William and Wilkins; 2008. Chapter 12, Cardiovascular system: p.191-92.
5. Oliver G. Lymphatic vasculature development. Nat Rev Immunol. 2004;4:35-45.(Pub Med)
6. Wigley C, Cecil C. Blood, lymphoid tissue and haemopoiesis. In: Standring S, editor. Gray's Anatomy. 40th ed. Spain: Elsevier publishers; 2008. Chapter 4.p.72-73.
7. Singh IB. Textbook of Human Histology. 5th edition. New Delhi: Jaypee publishers; 2006. Chapter 11. Lymphatics and lymphoid tissue: p.184-189.

8. Ganong WF. Dynamics of Blood and Lymph flow. Review of Medical Physiology. 22nd edition; McGrawHill;2005:p.593.
9. David JH, Valerie JL. Pharynx, larynx and neck. In: Norman SW, Christopher JK, Bulstrode, O'Connell PR, editors. Bailey and Love's short practice of surgery. 25th ed, London : Edward Arnold Ltd; 2008. Chapter 45.p.731-732.
10. Decker GAG. Lee Mcgregor's synopsis of Surgical Anatomy. 12th ed. Bombay: Varghese Publication; 1999. Chapter 16. Lymph tissues of the head and neck, surgical anatomy; p.189-196.
11. Paniker CKJ. Textbook of microbiology. 7th ed. Chennai: Orient Longman publishers; 2005. Chapter 39, Mycobacterium tuberculosis; p.351-64.
12. Brooks GF, Karen CC. Mycobacteria. In: Jawetz, Melnick, Adelberg, editors. Medical microbiology. 24th ed. USA: McGraw Hill; 2007. Chapter 24, p.320-27.
13. Kayser FH, Bienz KA, Eckert J, Kayser Medical Microbiology. 10th ed. Germany: 2005. Chapter 4, Bacteria as human pathogens; p.264.
14. Bloch H, Sorkin E. A toxic lipid component of tubercle bacillus. II: Isolation from petroleum ether extracts of young bacterial cultures. Am Rev Tuberc 1953;67:629-643.
15. Meltezon HC, Spyridis P, Kafetzis DA. Extrapulmonary tuberculosis in children. Arch Dis Child 2000;83:342-6.

16. Butt T, Riffat AN, Syed KY, Raja AK, Mahmood A. An update on the diagnosis of tuberculosis. JCPSP 2003;13 (12): 728-34.
17. Tuberculosis incidence (Internet) 2012 (cited 2010 october). Available from:http://www.who.int/mediacentre/factsheets/fs104/en/index.html.
18. Park K. Park's textbook of preventive and social medicine. 20th ed. Jabalpur: Bhenot;2009. Chapter 5, Epidemiology of communicable disease; p.160-161, 163-73.
19. Wares F, Balasubramanian R. Mohan A, Sharma SK. Extrapulmonary tuberculosis: Management and Control. In: Agarwal SV, Chauhan LS, editors. Tuberculosis control in India. New Delhi: Directorate General of Health Services. Ministry of Health and Family Welfare; 2005. P. 95-114.
20. Robbins, Kumar. Pathology basis of disease. 8th ed. W.B. Saunders, Philadelphia;2008. Chapter 13. The lung and upper respiratory tract: p.517-19.
21. Dass A, Nagarkar NM et- al. Cervical tuberculous lymphadenopathy: Changing clinical pattern and concepts in management. Postgrad Med J. 2001; 77 (905):185-7.
22. Head and neck imaging. Ed. By Peter M. Som, Hugh D. Curtin. St Louis (Mo): Mosby-Year Book,2003.
23. Ioachim's lymph node pathology. Editors. Herry L. Ioachim, L. Jeffrey Mederios. Philadelphia, Pa ; Lippincott Williams and Wilkins, 2008.

24. Jones PG, Campbell PE. Tuberculous lymphadenitis in childhood: the significance of anonymous mycobacteria. Br J Surg 1962;50:302-14.
25. Gupta AK, Nayar M, Chandra M, et al. Critical appraisal of fine needle aspiration cytology in tuberculous lymphadenitis. Acta Cytol 1992; 36: 391-4.
26. Lakhey M, Bhatta CP, Mishra S. Diagnosis of tubercular lymphadenopathy by fine needle aspiration cytology, acid-fast staining and Mantoux test. JNMA J Nepal Med Assoc. 2009 Jul-Sep; 48 (175): 230-3.
27. Avery G. Mycobacterium tuberculosis and other non- tuberculous mycobacteria. In: Connie RM, Donal CL, George M, editors. Text book of diagnostic microbiology. 3rd ed. New Delhi. Elsevier publishers; 2007. Chapter 26, 693-699, 700-05.
28. Manitchotpisit B, Kunachek S, Kulapralithram B: Combined use of fine needle aspiration cytology and polymerase chain reaction in the diagnosis of cervical tubercular lymphadenitis. J Med Assoc Thai 1999; 82: 363-68.
29. Kwon KS, Oh CK, Jang HS, Lee CW: detection of mycobacterial DNA in cervical granulomatous lymphadenopathy from formalin-fixed, paraffin embedded tissue by PCR. J Dermatol 2000; 27: 355-360.
30. Mario CR, Richard JO. Tuberculosis. In : Fauci AS, Braunwald E, Kasper DL, Hauser SL, editors. Harrison's principle of internal medicine. 17th ed. McGraw Hill; 2008. Chapter 158, p.1006-1020.

31. Das S. A concise textbook of surgery. 6th ed. Kolkata; 2010. Chapter 17. Diseases of the lymphatic system; p.296-309.
32. Treatment of tuberculosis: American thoracic society/ Centre for disease control/ Infectious disease Society of America- Recommendations and Reports. MMWR Morb Mortal Wkly Rep. 2003;52: 1-77.
33. British Thoracic Society Research Committee. Six months versus nine months chemotherapy for tuberculosis of lymph nodes: Final results. Respir Med 1993; 87: 621-3.
34. Tripathi KD. Essentials of medical pharmacology 5th ed. New Delhi: Jaypee publishers; 2004. Chapter 53, Antitubercular drugs; p.698-708.
35. Joel GH, Lee EL, Gillman AG. Goodman and Gillman's the Pharmacological basis of therapeutics. 10th ed. USA: McGraw Hill; 2001. Chapter 48. Drugs used in chemotherapy of tuberculosis and leprosy; p.1274-86.
36. Van Loenhout-Rooyackers JH, Laheij RJ, Richter C, Werbeek AL: Shortening the duration of treatment for cervical tuberculous lymphadenitis. Eur Respir J 2000; 15: 192-95.
37. Dalton, T. Cegielski, P. Akksilp, S. Asencios, L. Caoili, J. C. Cho, S.N.Erokhin, V.V.Ershova, J.et al. (2012). "Prevalence and risk factors for resistance to second line drugs in people with multidrug-resistant tuberculosis in eight centuries: A prospective cohort study". The Lancet. Doi: 10.1016/S0140-6736(12)60734-X.

38. Goble M, Iseman MD, Madsen LA, Waite D, Ackerson L, Horsburgh CR Jr(1993)." Treatment of 171 patients with pulmonary tuberculosis resistant to isoniazid and rifampin". N Engl J Med 328(8): 527-532.

39. Farquharson M, Moran B. Farquharson's text book of operative general surgery. 9th ed. Great Britain: Holder Arnold; 2005. Chapter 9, Surgery of the neck; p.164-165.

40. Danielides V, Patrikakos G, Moerman M, Bonte K, Dhooge C, Vermeersch H: Diagnosis, management and surgical treatment of non tuberculous mycobacterial head and neck infection in children. ORL J Otorhinolaryngol Relat Spec 2002;64;284-289.

41. Alessi DP, Dudley JP: Atypical mycobacteria induced cervical adenitis. Treatment by needle aspiration. Arch Otolaryngol Head Neck Surg.1999;1109-1113.

42. Panesar J, Higgins K, Daya H, Forte V, Allen U; Nontubercuolous mycobacterial cervical adenitis: A ten-year retrospective review. Laryngoscope 2003;113:149-154.

43. Hussain Md.W, Haque Md.A, Banu SA, Ekram ARMS, Rahman MF. Extra pulmonary tuberculosis: Experience in Rajshahi chest disease clinic and chest disease hospital. The journal of Teachers Association-June 2004; Volume 17(1): 16-19.

44. Biswas PK, Begum SMK. Tubercular cervical lymphadenopathy Clinicopathological study of thirty cases. The journal of Teachers Association- June 2007; Volume 20(1): 36-38.
45. Muhammad MM, Bukhari MH. Evaluation for granulomatous inflammation on Fine Needle Aspiration Cytology using special stains. SAGE-Hindawi Access to Research Pathology. Research International – May 2011; Article ID 851524, 1-8.
46. Salman M, Zeba A, Mahira Y, Muhammad SM. Evaluation of tuberculosis cervical lymphadenopathy. Pakistan Journal of Surgery – 2009; Volume 25(3):176-79.
47. Pamra, Mathur. Tuberculosis in children. The Indian Journal of Tuberculosis- April 1987; Vol 34(2): 41-44. Table 4, character to glands related to type of lymphadenitis; p.44.
48. Maharajan M, Hirachan S, Kafla PK, Bista M, Shrestha S, Taran KC. Incidence of tuberculosis in enlarged neck nodes. Our experience. Kathmandu University Medical Journal-2009; Vol.7(25);53-54.
49. Lai KK, Stottmaier KD, Sherman IH, et al. Mycobacterial cervical adenopathy. JAMA 1984;251:1286-9.
50. Lincoln EM, Sewell EM, Tuberculosis in children. New York, NY: Mc Graw – Hill Book Company Inc, 1963:207-15.
51. Campbell IA. The treatment of superficial tuberculous lymphadenitis. Tubercle 1990;71:1-3.

52. Seth V, Kabra SK, Semwal OP, et al. Tubercular lymphadenitis: Clinical manifestations. Indian J Pediatr 1995;62:565-70.
53. Belin RP, Richardson JD, Richardson DL, et al. Diagnosis and management of scrofula in children. J Pediatr Surg 1974;9:103-7.

ANNEXURE-I

PROFORMA

1) PARTICULARS OF THE PATIENT:

Name:

Age:

Sex:

I.P/O.P no:

Religion:

Occupation:

Income:

2) CHIEF COMPLAINS:

3) HISTORY OF PRESENT ILLNES:

a) Duration of node enlargement/ swelling in the neck

b) Associated symptoms

- Fever and cough
- Loss of appetite
- Throat pain
- Oropharyngeal and scalp sepsis.

c) Pain

d) Sinus present or absent

If so, 1) duration

ii) How did it start

iii) Nature of discharge

4) PAST HISTORY

- Pulmonary or Extrapulmonary
- Lymphadenitis

5) FAMILY HISTORY

6) PERSONAL HISTORY

- Contact with tuberculosis
- Habit of taking unboiled milk
- Living conditions and diet

7) MENSTRUAL / OBSTETRIC HISTORY

8) GENERAL PHYSICAL EXAMINATION

- Build
- Febrile
- Anemia
- Jaundice
- Clubbing
- Lymphadenopathy
- Weight

- Pulse
- Blood pressure

SYSTEMIC EXAMINATION

- CVS
- Respiratory system
- Abdomen
    - Liver
    - Spleen
    - Lymph nodes
    - Others
- Local examination: swelling in the neck
    - Unilateral- single groups or multiple groups
    - Bilateral- single groups or multiple groups
    - Skin over the swelling
- Associated with any other group of lymph nodes
    - ✓ Axillary -pectoral

      Subscapular

      Lateral

      Central

      Apical
    - ✓ Inguinal- horizontal

Vertical

Mediastinal

- Nature of the nodes
  - Situation
  - Size
  - Shape
  - Number
  - Inflammation
  - Consistency
  - Discrete or matted
  - Fixity]
  - Skin involvement
  - Surface
- Sinus if present
  - Number
  - Situation
  - Search for feeding node
  - Discharge
  - Other physical character
- Examination of draining area
  - Oral cavity

- Tonsil

9) INVESTIGATIONS

a. Blood

   Hb%

   TC

   DC

   ESR

b. Sputum if present for AFB

c. Radiological examination

   X-ray neck

   X-ray chest

d. Biopsy

   Macroscopic picture

   Biopsy report

e. Tuberculin test

10) TREATMENT

i. Anti tubercular

ii. Supportive

iii. Surgical

11) FOLLOW UP

ANNEXURE-II

MASTER CHART

S. N	NAME	AGE/S	IP/OP NO	RUR/URB	SES	NECK SWELLI	ASSOCIATED	LYMPH NODE	RT/LT/BL	DIS/MATTED	FIRM/SOFT	Hb	TLC	DC	ESR	CHEST X-RAY	FNAC CG/inc	EXCISION BIOF	TUBERCULIN	ATT
1	RENUKA	22/F	26879	RUR	L	1 YEAR	PAIN-10 DAY:	UDC	RT	DIS	FIRM	11	4,300	P65L30E5	32	-	CG	-	T	ATT
2	GOPINATH	23/M	1421522	RUR	L	15DAYS	-	UDC	RT	DIS	SOFT	9.8	5,690	P82L13E5	12	-	RH	-	-	-
3	STEPHAN	12/M	27834	URB	L	3 WEEKS	-	UDC	BL	MATTED	SOFT	7	4,580	P67L33E0	23	-	CG	-	-	ATT
4	DEVI	18/F	28413	RUR	L	25DAYS	FEVER-5DAYS	UDC	RT	DIS	FIRM	7.8	5,670	P58L36E6	16	-	RH	-	-	-
5	RAMANI	28/F	1426786	RUR	L	1 MONTH	-	LDC	LT	DIS	FIRM	12	6,790	P78L20E2	34	-	CG	-	-	ATT
6	SHLOK	35/M	29320	RUR	L	20 DAYS	-	SM	LT	DIS	SOFT	9	7,860	P67L39E4	31	-	CG	-	-	ATT
7	MANEESHA	24/F	1421671	URB	M	9 MONTHS	FEVER-20DA\	UDC	LT	MATTED	FIRM	8	5,980	P44L50E6	18	-	Inc	+	-	ATT
8	PATRICK	36/M	1415915	RUR	L	1 YEAR	-	UDC	RT	DIS	SOFT	8.7	5680	P80L18E2	23	+	CG	-	T	ATT
9	DHANALAKSHMI	26/F	27113	URB	L	7 MONTHS	WEIGHT LOS:	PT	RT	MATTED	FIRM	7.9	4590	P67L30E3	33	-	CG	-	-	ATT
10	RAMYA	27/F	1432868	RUR	L	2 WEEKS	-	UDC	LT	DIS	FIRM	11.2	4580	P50L28E22	32	-	CG	-	-	ATT
11	SEETHA	34/F	27234	RUR	L	20DAYS	-	UDC	LT	DIS	SOFT	12.8	3780	P40L58E2	16	-	RH	-	-	-
12	DIVYA	39/F	1416768	RUR	L	25 DAYS	PAIN-5DAYS	LDC	RT	DIS	FIRM	13.8	6780	P78L20E2	18	-	Inc	+	-	ATT
13	GURMEET	45/M	28119	URB	M	1 YEAR	-	LDC	RT	DIS	FIRM	10.7	7890	P56L40E4	32	-	RH	-	-	-
14	JOSHUA	39/M	1421711	RUR	L	3 WEEKS	-	MUL	LT	DIS	SOFT	7.8	8970	P67L39E4	35	-	CG	-	-	ATT
15	MANJU	27/F	29324	URB	L	2 YEARS	COUGH	UDC	LT	MATTED	FIRM	9.3	6560	P74L22E4	34	-	CG	-	-	ATT
16	CHANDINI	24/F	1426868	URB	L	1 MONTH	-	LDC	RT	DIS	SOFT	10.4	7860	P44L50E6	26	-	Inc	+	-	ATT
17	THIRUPATHI	23/M	28116	RUR	L	30DAYS	-	UDC	LT	DIS	FIRM	8.6	5400	P85L10E5	18	-	RH	-	-	-
18	SAI PRANAV	43/M	1423899	RUR	L	2 MONTHS	WEIGHT LOS:	LDC	RT	DIS	FIRM	9.5	6700	P65L31E4	35	-	CG	-	T	ATT
19	SRINIVAS	34/M	29118	RUR	L	25DAYS	-	UDC	RT	MATTED	FIRM	11.2	7800	P67L32E1	28	-	RH	-	-	-
20	SWATHI	24/F	23143	RUR	L	1.5MONTHS	FEVER-3DAYS	SM	BL	DIS	FIRM	8.5	13800	P75L24E1	32	-	CG	-	-	ATT
21	RAJ	67/M	25345	RUR	L	1YEAR	-	UDC	LT	DIS	SOFT	7.8	9800	P87L10E3	16	-	L	-	-	-
22	ROJA	23/F	21356	URB	M	4 MONTHS	-	LDC	LT	DIS	SOFT	12.5	8900	P64L34E2	11	-	CG	-	-	ATT
23	RIYA	35/F	21675	RUR	L	2 WEEKS	-	LDC	RT	DIS	SOFT	9.5	11890	P50L28E22	27	-	CG	-	-	ATT
24	RAJA	12/M	1420987	RUR	L	2.5MNTHS	COUGH	UDC	LT	MATTED	FIRM	11.7	6700	P77L22E1	9	-	RH	-	-	-
25	ROSE	25/F	1425456	URB	L	15DAYS	-	LDC	LT	DIS	FIRM	9.8	8700	P44L50E6	15	-	RH	-	-	-
26	KIRAN	24/F	1421256	RUR	L	5MONTHS	WEIGHT LOS:	UDC	LT	DIS	SOFT	12	6780	P72L24E4	35	-	CG	-	-	-
27	SHAHEER	26/M	1425987	URB	L	15DAYS	-	LDC	RT	DIS	FIRM	14.7	4670	P70L27E3	34	-	CG	-	-	ATT
28	KAMINI	18/F	24546	RUR	L	20DAYS	-	LDC	LT	DIS	FIRM	8.9	6900	P65L30E5	24	-	RH	-	-	-
29	SRIKANTH	45/M	27565	RUR	L	1 YEAR	FEVER-4DAYS	MUL	LT	DIS	SOFT	9.4	8400	P64L34E2	11	+	INC	+	-	ATT
30	TAMILSELVI	56/F	28574	URB	L	25DAYS	-	UDC	BL	DIS	SOFT	12.8	9200	P76L20E4	27	-	RH	-	-	-
31	MUNUSAMY	39/M	1417654	RUR	L	6 WEEKS	PAIN-10DAYS	UDC	LT	DIS	FIRM	6.8	10200	P58L36E6	36	-	CG	-	-	ATT
32	KOLAPURI	55/F	1426575	URB	L	20 DAYS	COUGH	LDC	LT	DIS	SOFT	12.7	7800	P59L38E4	6	-	CG	-	T	ATT
33	SRINATH	23/M	27565	RUR	M	1MNTH	FEVER-5DAYS	UDC	BL	DIS	FIRM	8.9	8200	P44L55E2	21	-	RH	-	-	-
34	SADHANA	39/F	1426465	URB	L	4WEEKS	-	UDC	LT	DIS	FIRM	9.6	8400	P88L11E1	12	-	L	-	-	-
35	URVASI	12/F	1417574	RUR	L	20 DAYS	-	PT	RT	MATTED	FIRM	10.6	7900	P57L40E3	37	-	CG	-	-	ATT
36	JAYAKUMAR	38/M	27565	URB	M	4 MOTNHS	FEVER-10DA\	UDC	LT	DIS	SOFT	8.5	8100	P56L34E10	9	-	CG	-	-	ATT
37	JOTHIKA	43/F	27757	RUR	L	20 DAYS	-	PT	LT	DIS	FIRM	8.4	9400	P67L39E4	33	-	Inc	+	-	ATT
38	POORNIMA	45/F	1417555	RUR	L	5DAYS	-	UDC	LT	DIS	FIRM	10.7	5900	P68L20E2	11	-	RH	-	-	-
39	NISHANTH	23/M	23546	RUR	L	3 WEEKS	PAIN-10DAYS	PT	BL	DIS	FIRM	9.7	7300	P65L30E5	34	-	Inc	+	-	ATT
40	RAMASAMY	38/M	22765	RUR	L	1 MONTH	-	SM	BL	DIS	SOFT	10.4	5040	P74L22E4	16	-	CG	-	T	ATT
41	BARATI	25/F	27869	RUR	L	2 MONTHS	WEIGHT LOS:	PT	LT	DIS	FIRM	9.4	9300	P57L40E3	36	-	CG	-	-	ATT
42	MANIMOZHI	44/F	29876	RUR	L	3 WEEKS	-	PT	LT	DIS	FIRM	9.6	8600	P62L34E6	34	-	CG	-	-	ATT
43	RAMBA	34/F	1423555	URB	L	2 WEEKS	-	UDC	LT	DIS	SOFT	11.9	8900	P90L9E1	18	+	RH	-	-	-
44	PRITHVI	26/M	1417577	RUR	L	2 YEARS	FEVER-1MNT	PT	LT	DIS	FIRM	12.7	11000	P59L40E1	24	-	CG	-	-	ATT
45	BHUVI	26/F	1418676	RUR	L	2 MONTHS	-	UDC	BL	DIS	FIRM	7.8	12300	P68L30E2	37	-	CG	-	-	ATT
46	VIJAY	38/M	1426324	URB	L	1 WEEK	-	PT	LT	DIS	FIRM	11.7	8200	P67L33E0	35	-	Inc	+	-	ATT
47	DEEPA	25/F	1427897	RUR	L	6 MONTHS	COUGH	MUL	RT	MATTED	FIRM	9.6	9020	P65L24E11	36	-	CG	-	-	ATT
48	SALMAN	45/M	27685	RUR	L	3 WEEKS		UDC	LT	DIS	FIRM	13	7900	P85L10E5	18	-	RH	-	-	-
49	DAISY	37/F	26565	RUR	M	5 MONTHS	FEVER-15DA\	UDC	RT	MATTED	SOFT	7.6	8100	P64L34E2	32	-	CG	-	T	ATT
50	SURIYA	28/M	1427676	RUR	L	20 DAYS	-	UDC	RT	MATTED	FIRM	8.9	7300	P59L40E1	26	-	Inc	+	-	ATT

S. N	NAME	AGE/S	IP/OP NO	RUR/URB	SES	NECK SWELLI	ASSOCIATED	LYMPH NODE	RT/LT/BL	DIS/MATTED	FIRM/SOFT	Hb	TLC	DC	ESR	CHEST X-RAY	FNAC CG/inc	EXCISION BIOF	TUBERCULIN	ATT
51	ISHITA	23/F	1425768	RUR	L	1.5MNTHS	-	UDC	LT	DIS	FIRM	7.8	10300	P72L26E2	8	-	RH	-	-	-
52	RAMAN KUMAR	29/M	28674	RUR	L	2 WEEKS	COUGH	PT	RT	MATTED	FIRM	7.6	12000	P50L28E22	47	-	CG	-	-	ATT
53	ASHWINI	23/F	27578	RUR	L	15DAYS	PAIN-2DAYS	LDC	LT	DIS	FIRM	8	9500	P71L24E5	6	-	RH	-	-	-
54	DHARINI	22/F	28675	URB	L	2WEEKS	-	UDC	LT	DIS	SOFT	9.7	12400	P48L39E13	12	-	RH	-	-	-
55	DIVYA	34/F	1417675	URB	L	1MNTH	-	UDC	RT	DIS	FIRM	9	7,400	P76L20E4	11	-	RH	-	-	-
56	RAJNEESH	34/M	29785	RUR	L	1YEAR	-	UDC	LT	DIS	FIRM	8.5	5900	P67L30E3	21	-	RH	-	-	-
57	SAROJA	27/F	28675	URB	M	2 WEEKS	-	PT	RT	DIS	FIRM	11	6870	P68L29E3	36	-	INC	+	-	ATT
58	KANNAN	47/M	24654	RUR	L	25 DAYS	FEVER- 3DAY	UDC	LT	DIS	FIRM	8.4	6780	P50L28E22	37	-	CG	-	T	ATT
59	SUNDRAMMAL	56/F	1423765	URB	L	2WEEKS	-	UDC	LT	DIS	SOFT	8.9	5940	P70L27E3	9	-	RH	-	-	-
60	DILIP	24/M	1426543	RUR	M	1 WEEK	-	UDC	RT	DIS	FIRM	7.8	9280	P74L25E1	11	-	CG	-	T	ATT
61	JAMUNA	24/F	24657	URB	L	6 MNTHS	PAIN-20DAYS	UDC	BL	MATTED	FIRM	12	6090	P81L16E3	23	-	INC	+	-	ATT
62	LAKSHMAN	23/M	26764	RUR	L	12DAYS	-	UDC	RT	DIS	SOFT	11.9	8580	P45L50E5	27	-	RH	-	T	-
63	KUPPAMMA	55/F	27678	RUR	L	3 WEEKS	-	UDC	LT	DIS	FIRM	8.6	10350	P70L27E3	45	-	CG	-	-	ATT
64	MEENAKSHI	24/F	1427895	RUR	L	2 YEARS	FEVER-1WEE	PT	LT	DIS	SOFT	13	10200	P63L36E1	34	-	CG	-	-	ATT
65	SENDIYAMMAL	46/F	23786	RUR	L	2 WEEKS	-	UDC	LT	DIS	SOFT	7.8	11300	P63L34E3	8	-	CG	-	T	ATT
66	MAHESHWARI	17/F	24556	RUR	L	2 MOTNHS	COUGH	UDC	RT	DIS	FIRM	12.9	12030	P65L34E1	36	-	CG	-	T	ATT
67	PITCHANDI	56/M	24356	RUR	H	20DAYS	PAIN-4DAYS	UDC	LT	DIS	FIRM	9.5	9320	P89L10E1	16	+	RH	-	-	-
68	ANGEL	26/F	1412823	URB	L	1 YEAR	FEVER-20DA	UDC	RT	MATTED	FIRM	10.9	12900	P58L36E6	32	-	CG	-	-	ATT
69	ANJALI	15/F	26566	RUR	L	4 WEEKS	-	UDC	RT	DIS	FIRM	11.8	11,300	P65L30E5	8	-	CG	-	T	ATT
70	SURESH	46/M	27678	RUR	L	20 DAYS	-	UDC	LT	DIS	FIRM	12.1	12,200	P59L40E1	31	-	CG	-	-	ATT
71	PRAVEEN	23/M	1421980	URB	L	15 DAYS	-	UDC	LT	DIS	SOFT	12	8790	P45L52E3	25	-	CG	-	-	ATT
72	PREM	11/M	23455	RUR	L	1 MONTH	-	UDC	LT	DIS	FIRM	11.5	7200	P71L24E5	35	-	Inc	+	-	ATT
73	MUNIYAMMAL	58/F	23656	RUR	L	2 MONTHS	FEVER-7DAYS	UDC	RT	MATTED	FIRM	8.6	8900	P68L30E2	13	-	CG	-	-	ATT
74	RAASHI	18/F	25658	RUR	L	10 DAYS	-	LDC	LT	MATTED	FIRM	9.8	9300	P59L39E2	23	-	RH	-	-	-
75	YABESH	25/M	29796	URB	L	1 MONTH	-	UDC	LT	DIS	SOFT	9	6700	P55L44E1	36	-	CG	-	T	ATT
76	PRADEEP	35/M	1415677	RUR	L	3 WEEKS	-	UDC	LT	DIS	FIRM	11.8	7400	P59L40E1	10	-	CG	-	-	ATT
77	VIVEK	58/M	31373	RUR	L	1.5 MONTHS	FEVER-10DA	PT	LT	MATTED	FIRM	10.2	4570	P72L24E4	22	-	CG	-	-	ATT
78	CHELLAMA	34/F	1416676	RUR	L	15DAYS	-	UDC	LT	DIS	FIRM	9.2	8400	P80L18E2	14	-	RH	-	-	-
79	SASHI	25/F	1426787	RUR	L	2 MONTHS	COUGH	UDC	LT	DIS	FIRM	9	5680	P67L33E0	50	-	CG	-	-	ATT
80	MANJU	27/F	24243	URB	L	2 YEARS	PAIN-6MNTH	UDC	RT	DIS	FIRM	11	8200	P55L44E1	45	+	Inc	+	T	ATT
81	REEMA	34/F	1416788	RUR	M	1MNTH	-	LDC	LT	DIS	FIRM	10.4	8490	P72L26E2	23	-	RH	-	-	-
82	VIDYA	28/F	1417688	RUR	L	20DAYS	-	UDC	LT	DIS	FIRM	11.6	5800	P73L22E5	9	-	CG	-	-	ATT
83	CHANDRA	37/F	24466	URB	L	1 MONTH	WEIGHT LOS	UDC	LT	DIS	FIRM	9.6	7200	P74L21E5	52	-	CG	-	-	ATT
84	ROMA	23/F	24354	RUR	L	2YEARS	FEVER-25DA	UDC	LT	DIS	SOFT	12.6	5460	P70L27E3	8	-	CG	-	-	ATT
85	SHANTHANU	17/M	1413680	RUR	L	20 DAYS	-	UDC	LT	DIS	FIRM	9	6900	P55L44E1	44	-	CG	-	-	ATT
86	RAAGHAVI	21/F	25567	RUR	L	3 WEEKS	PAIN-4DAYS	UDC	LT	DIS	FIRM	11.7	5670	P59L40E1	13	-	RH	-	-	-
87	RANJITHA	23/F	1415778	RUR	L	1 MONTH	-	UDC	RT	MATTED	FIRM	8.6	8200	P45L52E3	21	-	CG	-	T	ATT
88	DEVENDRA	24/M	25346	RUR	L	6 MONTHS	FEVER-2DAYS	UDC	LT	DIS	SOFT	12	7940	P84L14E2	43	-	CG	-	-	ATT
89	YUVARANI	15/F	23355	URB	L	1MNTH	WEIGHT LOS	UDC	LT	DIS	SOFT	7.9	6750	P69L30E1	8	-	RH	-	-	-
90	RAHEEMA	25/F	1415678	URB	L	2 WEEKS	-	PT	LT	DIS	FIRM	13.2	8390	P68L31E1	20	-	CG	-	-	ATT
91	CHELLAPA	34/M	1412898	RUR	L	9 MONTHS	WEIGHT LOS	UDC	LT	DIS	FIRM	11	5430	P78L18E4	11	+	CG	-	T	ATT
92	SHAILAJA	26/F	26580	URB	L	10DAYS	-	UDC	RT	DIS	FIRM	9.8	7800	P69L30E1	36	-	CG	-	-	ATT
93	MANJUNATH	23/M	1417879	RUR	M	1 MONTH	-	UDC	RT	MATTED	FIRM	12	6940	P45L52E3	24	-	CG	-	T	ATT
94	MAMTHA	27/F	23456	URB	L	2 YEARS	FEVER-20DA	UDC	LT	DIS	SOFT	8.8	7200	P80L18E2	18	-	CG	-	-	ATT
95	SWARNA	56/F	24570	RUR	L	15DAYS	-	UDC	LT	DIS	FIRM	12	9320	P72L24E6	47	-	CG	-	T	ATT
96	BHEESHMA	23/M	26757	RUR	M	25DAYS	PAIN-10DAYS	LDC	LT	MATTED	FIRM	14.2	5970	P71L24E5	15	-	RH	-	-	-
97	FATHIMA BEEVI	54/F	1416789	URB	L	10 DAYS	-	UDC	LT	DIS	FIRM	9	4070	P68L30E2	11	-	CG	-	-	ATT
98	GIRIJA	35/F	1416768	RUR	L	2 MONTHS	COUGH	UDC	LT	MATTED	FIRM	8.7	8590	P48L39E13	53	-	CG	-	-	ATT
99	NAGESH	15/M	26879	RUR	L	3 WEEKS	WEIGHT LOS	PT	LT	DIS	SOFT	10	8370	P60L32 E8	26	-	CG	-	-	ATT
100	SUBADRA	52/F	29790	RUR	L	1 YEAR	PAIN-3MNTH	UDC	RT	DIS	FIRM	12	5790	P55L44E1	43	-	CG	-	T	ATT

KEY TO MASTER CHART

M : Male

F : Female

IP : Inpatient

OP : Out patient

SES : Socioeconomic status

RUR : Rural

URB : Urban

DIS : Discrete

RT : Right

LT : Left

UDC : Upper deep cervical node

LDC : Lower deep cervical node

PT : Posterior triangle node

SM : Submandibular node

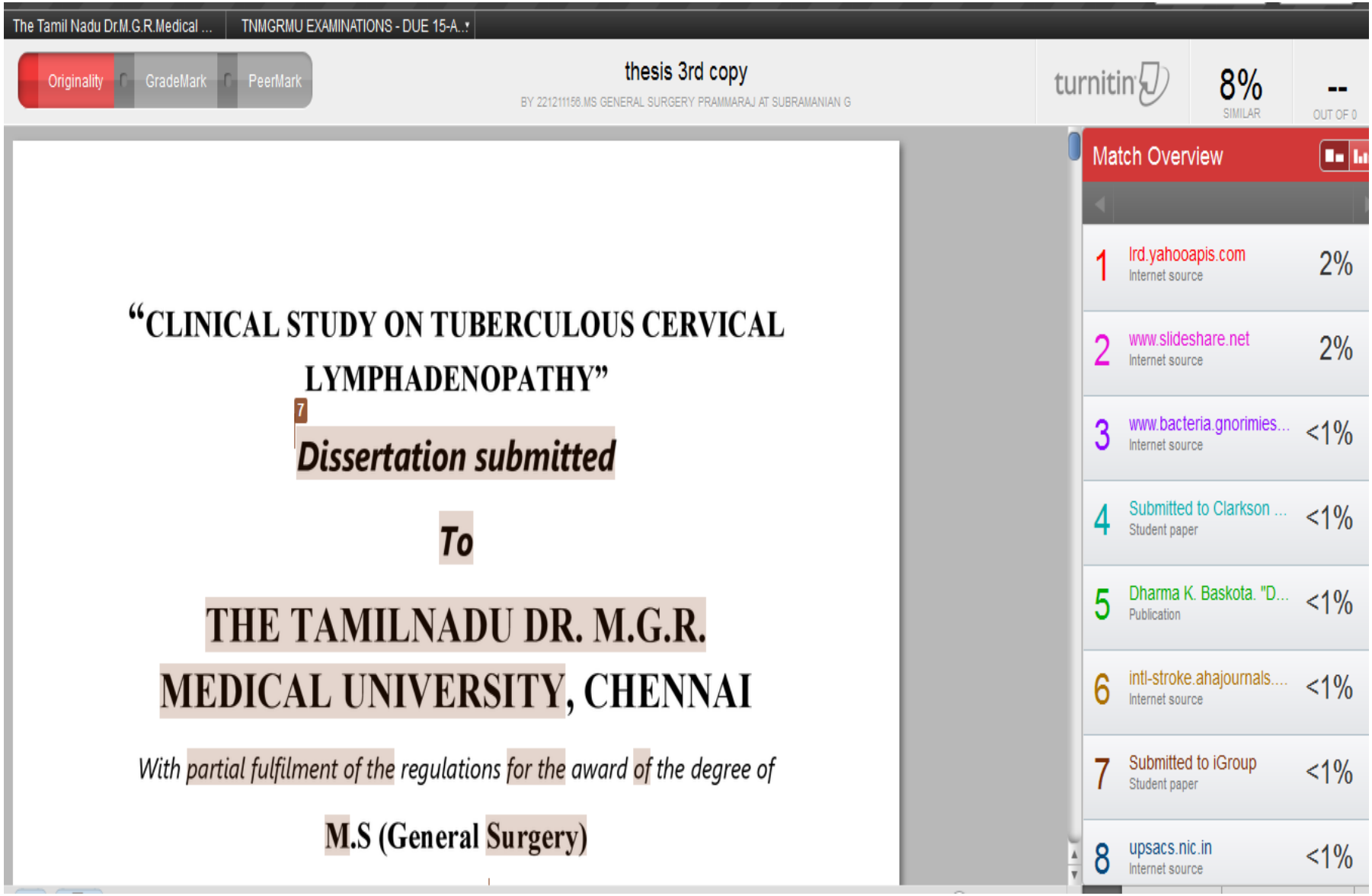
MUL : Multiple group

BL : Bilateral

L : Lower

M : Middle

H : Higher


The Tamil Nadu Dr.M.G.R.Medical ...
TNMGRMU EXAMINATIONS - DUE 15-A..
Originality
GradeMark
PeerMark
thesis 3rd copy
BY 221211156.MS GENERAL SURGERY PRAMMARAJ AT SUBRAMANIAN G
turnitin
8%
SIMILAR
--
OUT OF 0
"CLINICAL STUDY ON TUBERCULOUS CERVICAL LYMPHADENOPATHY"
Dissertation submitted
To
THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI
With partial fulfilment of the regulations for the award of the degree of
M.S (General Surgery)
Match Overview
1 lrd.yahooapis.com Internet source 2%
2 www.slideshare.net Internet source 2%
3 www.bacteria.gnorimies... Internet source <1%
4 Submitted to Clarkson ... Student paper <1%
5 Dharma K. Baskota. "D... Publication <1%
6 intl-stroke.ahajournals.... Internet source <1%
7 Submitted to iGroup Student paper <1%
8 upsacs.nic.in Internet source <1%

INSTITUTIONAL ETHICAL COMMITTEE
GOVT. KILPAUK MEDICAL COLLEGE
CHENNAI-10
REF.NO.18520/ME-I/Ethics/2013 Dt:05.12.2013
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai-10 reviewed and discussed the application for approval "A Clinical Study on cervical tubercular lymphadenopathy" – For Research work submitted by Dr.G.Prammaraj @ Subramanian, MS (General Surgery) PG Student, KMC, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occuring in the course of the study any change in the protocol and patient information / informed consent and asks to be provided a copy of the final report.

GOVT. KILPAUK MEDICAL COLLEGE
28 JAN 2014
Kilpauk, Chennai-10.

28/1/14
CHAIRMAN
Ethical Committee
Govt. Kilpauk Medical College, Chennai