



## A CLINICAL SURVEY ON CORRELATION STUDY BETWEEN TUBERCULOSIS AND ITS PREDISPOSING FACTORS IN MATHURA REGION

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### ABSTRACT

The aim of the study was to correlate between tuberculosis and its predisposing factors and find out influence of Age, Sex, Occupation, Income, Dietary habits, Addiction (Smoking, Tobacco and Alcohol), Multiple drug therapy, Concomitant disease, Past history of Koch's on the patients under the supervision of Dr. Mayank maheshwari (Physician, Maheshwari Hospital, Mathura). In the present study "Correlation study between Tuberculosis and its pre disposing factors" total 91 patients were included. In case of age, the subjects below 25 years were 18.68% between 25-50 years were 54.94% and above 50 years were 26.37%. In case of sex, 71.42% were males and 28.57% were females. While in case of occupation, 10.98% were students, 7.69% retired, 19.78% house wife and 61.53% employed. And in case of income, the subjects below 5,000 Rs were 89.01%, between 5,000- 10,000 Rs were 7.69% and above 10,000 Rs were 1.09%. In case of dietary habit, 53.84% were vegetarians and 46.15% non vegetarians. In case of addiction, 14.28% were tobacco chewers, 4.39% alcoholics and 18.68% both (tobacco chewer and Alcoholics). In case of smoking habit 27.47% were smokers and 72.52% non smokers. In case of past history of Koch's 29.97% were new cases and 78.97% relapses ceases.

**Keywords:** Tuberculosis, Vegetarians, Tobacco, Addiction.

### INTRODUCTION

An increasing morbidity and mortality from tuberculosis (TB) in the near future is forecast for the world at large, with the number of newly occurring cases predicted to increase from 7.5 million a year in 1990 to 8.8, 10.2 and 11.9 million in the years 1995, 2002 and 2005 respectively; an increase amounting to 58.6 per cent over a 15-yr period<sup>2</sup>. The estimates were subsequently found to be appropriate for the year 2000 using a new method<sup>3</sup>. The proportion of tuberculosis cases co-infected with human immune deficiency virus (HIV) was also found to be rising, being 2-10 times greater for the 1997 estimates, than for 1992. The association with HIV and increasing multi drug resistant tuberculosis (MDRTB) appears to be a serious issue, especially for the developing nations. This infection is caused by one of two forms of mycobacterium *Mycobacterium tuberculosis*. Humans are the main host. The microbes cause pulmonary tuberculosis and are spread either by droplet infection from an individual with active tuberculosis, or in dust contaminated by infected sputum. *Mycobacterium bow's*. Animals are the main host. The microbes are usually spread to humans by untreated milk from infected cows, causing infection of the alimentary tract. In Britain the incidence has been greatly reduced by the elimination of bovine tuberculosis and the pasteurization of milk. It is still a significant infection in many countries<sup>7</sup>.

### PATHOPHYSIOLOGY OF TUBERCULOSIS

Once inhaled, the infectious droplets settle throughout the airways. The majority of the bacilli are trapped in the

upper parts of the airways where the mucus-secreting goblet cells exist. The mucus produced catches foreign substances, and the cilia on the surface of the cells constantly beat the mucus and its entrapped particles upward for removal<sup>3</sup>. This system provides the body with an initial physical defense that prevents infection in most persons exposed to tuberculosis<sup>9</sup>. Bacteria in droplets that bypass the mucociliary system and reach the alveoli are quickly surrounded and engulfed by alveolar macrophage<sup>4,9</sup>, the most abundant immune effector cells present in alveolar spaces<sup>10</sup>. These macrophages, the next line of host defense, are part of the innate immune system and provide an opportunity for the body to destroy the invading mycobacteria and prevent infection<sup>15</sup>. Macrophages are readily available phagocytic cells that combat many pathogens without requiring previous exposure to the pathogens. Several mechanisms and macrophage receptors are involved in uptake of the mycobacteria<sup>15</sup>. The mycobacterial lipo arabinomannan is a key ligand for a macrophage receptor<sup>13</sup>. The complement system also plays a role in the phagocytosis of the bacteria<sup>12</sup>. The complement protein C3 binds to the cell wall and enhances recognition of the mycobacteria by macrophages. Opsonization by C3 is rapid, even in the air spaces of a host with no previous exposure to *M tuberculosis*<sup>5</sup>. The subsequent phagocytosis by macrophages initiates a cascade of events that results in either successful control of the infection, followed by latent tuberculosis, or progression to active disease, called primary progressive tuberculosis<sup>4</sup>. The outcome is essentially determined by the quality of the host defenses and the balance that occurs between host defenses and



the invading mycobacteri<sup>6,15</sup>. After being ingested by macrophages, the mycobacteria continue to multiply slowly<sup>4</sup>, with bacterial cell division occurring every 25 to 32 hours<sup>1,14</sup>. Regardless of whether the infection becomes controlled or progresses, initial development involves production of proteolytic enzymes and cytokines by macrophages in an attempt to degrade the bacteria<sup>13,15</sup>. Released cytokines attract T lymphocytes to the site, the cells that constitute cell-mediated immunity. Macrophages then present mycobacterial antigens on their surface to the T cells<sup>15</sup>. This initial immune process continues for 2 to 12 weeks; the microorganisms continue to grow until they reach sufficient numbers to fully elicit the cell-mediated immune response, which can be detected by a skin test<sup>4,14,15</sup>.

### AIM OF THE STUDY

The aim of the study was to correlate between tuberculosis and its predisposing factors and find out influence of Age, Sex, Occupation, Income, Dietary habits, Addiction (Smoking, Tobacco and Alcohol), multiple drug therapy, Concomitant disease, Past history of koch's on the patients.

### SUBJECT AND METHOD

The study was carried out the medicine department hospital Mathura (U.P.). Total 91 patients satisfying the inclusion criteria of the study were enrolled. Potential study subjects were thoroughly interrogated for history in local dialect and questioned for detailed information pertaining to the disease. A through clinical examination was done for both pulmonary and extra pulmonary tuberculosis by medical specialist. After provisional diagnosis, the subjects had to undergo following laboratory investigation for confirmation of diagnosis as inclusion criteria for study.

All subjects received standard antibiotic for a week during investigation phase of minimize the chance of diagnostic error before confirming for tuberculosis. The patients were followed upon a weekly basis during the period of treatment.

#### Inclusion criteria

- Patients of either sex
- Patients diagnosed with tuberculosis.
- Patients should be more then 12 years.
- Patients admitted to the ward or visiting medicine OPD of Maheshwari at least once a day.

#### Exclusion criteria

- Patients less than 12 years.
- Pregnant / lactating females.

### STUDY DESIGN

This was an open, non comparative study to correlate between tuberculosis and predisposing factors in both

outpatients and inpatients coming for treatment at Maheshwari Hospital Mathura (U.P.)

### STUDY SCHEDULE AND PLAN

The patients were enrolled after oral informed consent as per the inclusion and exclusion criteria.

### ASSESSMENT

Age	Dietary habit
Sex	Concomitant disease
Income	Occupation
Addiction	Past History of Koch's
Smoking	

### RESULTS AND DISCUSSION

PARAMETERS	RESULT
Sex	Tuberculosis is prevalent in male > female.
Past history of koch's	71 were new cases and 20 were already suffered with kock's.
Smoking habit	Tuberculosis is prevalent in patients with non smoker> smoker.
Age	Tuberculosis was prevalent in patients with middle age (25-50 years).
Dietary habit	Tuberculosis prevalent in patients with Vegetarian> Non-vegetarian.
Occupation	Tuberculosis is prevalent in patients with Employed > House wife> Student > Retired.
Income	Tuberculosis is prevalent in low income group patients > middle income group > high income group.
Addiction	Tuberculosis is prevalent in patients with both (alcoholics + tobacco) > tobacco > alcoholic.

In the present study we correlated the tuberculosis and it's the predisposing factors in different categories of patients.

- With age group, the members of patients were found more between 25-50 years. This might be because of more interaction among this age group.
- With in sex group, the number of male patients was more than females. This might be because of frequent reporting of males.
- Within occupation and income group, tuberculosis was more prevalent in low income employed patients. This might be because of low income hence malnutrition status.
- Within dietary habit, tuberculosis was more prevalent in vegetarian than non vegetarian. Through the difference was not statistically significant as early reported.



- Within addiction, tuberculosis was more prevalent in both (alcoholics and tobacco chewer). This might be due to fibrogenesis and collagen tissue formation in the liver which disturbs the power of detoxication of liver and causes hepatic injury also acetaldehyde produced during metabolism appears to damage hepatocyte.
  - Within smoking habit, tuberculosis was more prevalent in non smoker. This might be because of more smokers in society.
  - Within past history of koch's, there were relapses of tuberculosis. This might be because of irregular treatment or complete instruction behalf of treatment was not followed by the patients.
7. Goodman & Gilman's, The pharmacological basis of therapeutics, 11<sup>th</sup> ed., Mcgraw-hil Publisher, United States of America 2006, 587.
  8. Joe M, Bai Y, Nacario RC, Lowary TL. Synthesis of the docosanasaccharide arabinan domain of mycobacterial arabinogalactan and a proposed octadecasaccharide biosynthetic precursor. J Am Chem Soc. 2007;129(32):9885–9901.
  9. Jensen PA, Lambert LA, Iademarco MF, Ridzon R; Centers for Disease Control and Prevention. Guidelines for preventing the transmission of Mycobacterium tuberculosis in health-care settings, 2005. MMWR Recomm Rep. 2005;54(RR-17):1–141.
  10. Korf JE, Pynaert G, Tournoy K, et al. Macrophage reprogramming by mycolic acid promotes a tolerogenic response in experimental asthma. Am J Respir Crit Care Med. 2006;174(2):152–160.

### REFERENCES

1. American Thoracic Society and Centers for Disease Control and Prevention. Diagnostic standards and classification of tuberculosis in adults and children. Am J Respir Crit Care Med. 2000;161(4 pt 1):1376–1395
2. Dolin PJ, Raviglione MC, Kochi A. A review of current epidemiological data and estimation of future tuberculosis incidence and mortality. WHO/TB/93.173. Geneva: World Health Organization; 1993.
3. Dye C, Scheele S, Dolin P, Pathania V, Raviglione MC. Global burden of tuberculosis: estimated incidence, prevalence and mortality by country. WHO Global Surveillance and Monitoring Project. JAMA 1999; 282: 677-86.
4. Frieden TR, Sterling TR, Munsiff SS, Watt CJ, Dye C. Tuberculosis. Lancet. 2003;362: 887–899.
5. Ferguson JS, Weis JJ, Martin JL, Schlesinger LS. Complement protein C3 binding to Mycobacterium tuberculosis is initiated by the classical pathway in human bronchoalveolar lavage fluid. Infect Immun. 2004;72: 2564–2573.
6. Goyot-Revoll V, Innes JA, Hackforth S, Hinks T, Lalvani A. Regulatory T cells are expanded in blood and disease sites in patients with tuberculosis. Am J Resp Crit Care Med. 2006;173:80.
11. Lee RB, Li W, Chatterjee D, Lee RE. Rapid structural characterization of the arabinogalactan and lipoarabinomannan in live mycobacterial cells using 2D and 3D HR-MAS NMR: structural changes in the arabinan due to ethambutol treatment and gene mutation are observed. Glycobiology. 2005;15(2):139–151.
12. Li Y, Petrofsky M, Bermudez LE. Mycobacterium tuberculosis uptake by recipient host macrophages is influenced by environmental conditions in the granuloma of the infectious individual and is associated with impaired production of interleukin-12 and tumor necrosis factor alpha. Infect Immun. 2002;70:6223–6230
13. Nicod LP. Immunology of tuberculosis. Swiss Med Wkly. 2007;137(25–26):357–362.
14. Porth CM. Alterations in respiratory function: respiratory tract infections, neoplasms, and childhood disorders. In: Porth CM, Kunert MP. Pathophysiology: Concepts of Altered Health States. Philadelphia, PA: Lippincott Williams & Wilkins; 2002:615–619.
15. Van Crevel R, Ottenhoff THM, van der Meer JWM. Innate immunity to Mycobacterium tuberculosis. Clin Microbiol Rev. 2002;15: 294–309.

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