International Journal of Microbiology and Allied Sciences



REVIEW ARTICLE OPEN ACCESS

# Transmission of Tuberculosis and its Prevention

**Tauseef Ahmad*, Anees Fatima, Muhammad Farooq, Muhammad Ayub Jadoon, Akbar Hussain and Sikandar Khan Sherwani[2]**

[1]Department of Microbiology, Hazara University, Mansehra, Khyber Pakhtunkhwa, Pakistan

[2]Department of Microbiology, FUUAST Karachi, Pakistan

***Corresponding author:*** *Tauseef Ahmad; E-mail: hamdardmicrobiologist@gmail.com;*

*Cell No: +92-346-9403966*

## ABSTRACT

The Tuberculosis (TB) is virtually transmitted from one person to another person. For the control and eradication of TB the Directly Observed Treatment short-course (DOTS) program launched in 1994. The discovery of streptomycin changes the history of TB. Bacillus Calmette-Guerin (BCG) vaccine was used to protect the TB patients.

**Keywords:** Tuberculosis, Directly Observed Treatment short-course, Streptomycin, Bacillus Calmette-Guerin.

## TUBERCULOSIS TRANSMISSION

There are some major factors which increase the incidence of TB like socioeconomic condition, Human immunodeficiency virus (HIV) epidemic, poor living standard, demographic change and inadequate attention to TB in health policies. The transmission of disease from mother to child is a common phenomena because siblings in a joint family system 1. The Mycobacterium tuberculosis (MTB) virtually transmitted from person to person, usually by mucous droplets i.e. cough, sneezes, laugh, sings or even breath. The size of particles are an estimated 1–5 mm, for prolonged time periods normal air currents can keep them airborne when it is inhales by a susceptible person these droplet nuclei traverse the mouth or nasal passages reach to the alveoli of the lungs. The infections depend on duration of exposure to infectious droplet nuclei and its concentration 2. In health-care facilities the transmission of MTB is a recognized risk and associated with close contact with those persons who have infectious TB e.g., bronchoscopy 3. Aerosol treatments and sputum induction induce coughing may also increase the potential for transmission of MTB 4.

## PREVENTIVE THERAPY FOR TB INFECTION

Directly Observed Treatment short-course (DOTS) was launched in 1994. In 1998, the Stop TB Partnership was established, while in 2000, World Health Organization (WHO) set up "Green Light Committee" (GLC). In 2000, the WHO and its partner launched "Global Plan to Stop TB" which included global targets for TB control. In 2006 and 2010 the WHO released updated plans 5-6. The Global Fund was established in 2002, to provide significant financial support for global responses to TB 7. The main purposes of the DOTS program are; case detection, standard short-course therapy, regular drug supply, monitoring and evaluating the program. Globally the case detection rate is increased from 45% to 65% in 2003. While the treatment success rate of DOTS is 87% in 2009 8-9. In Pakistan, the treatment success rate of TB was 90% in 2010. In 2010, of the 22 High Burden Countries (HBCs), the 14 counties reached or exceeded a treatment success rate of 85% among all new cases. South Africa (53%), Russian Federation (66%), Uganda (68%), Brazil (72%), Zimbabwe (76%), Ethiopia (77%), Nigeria (81%) and Thailand (83%) are those countries which reported lower rates of treatment success of DOTS program in 2010 10.

## DRUG TREATMENT OF TB

The discovery of Streptomycin and other subsequent anti-tuberculosis agents changed the history of TB. For the controlling of TB drug treatment is fundamental. In 1944 anti-tuberculosis drug treatment was started. The drug resistant bacilli emerged after the introduction of anti-tuberculosis drugs. The combined drug treatment was established in 1950. After that a few years later, Isoniazid (INH) was introduced. In cases of infections the mutations associated with incomplete or insufficient treatment of TB infection, co-infection of HIV and TB as well as irrational use of anti-tuberculosis antibiotics has increased the drug resistance problem. TB control has another major problem, difficulty in diagnosis of TB cases 11-12.

Initial phase: Currently the initial phase of treatment of TB consists of 8 weeks with Isoniazid (INH), Rifampin (RIF), Pyrazinamide (PZA), and Ethambutol (EMB) and is administered daily for 2 weeks; then, therapy is switched to twice weekly for an additional 6 weeks.

Continuation phase: And the continuation phase consists of 18 weeks of INH and RIF administered twice weekly 13.

The controlling of TB infection the development of effective vaccines remains an important goal. For ultimately eradicating this disease vaccination is still the best hope to fight against MTB infection is a challenges in attempts to design and develop successful vaccines. In animal models the pathogenesis of TB infection is studied and there is no completely satisfactory animal model of human TB available 14. For the study of MTB in humans there is no perfect animal model, animal studies have the potential to provide useful information about MTB infection 15.

## VACCINES FOR TUBERCULOSIS

### Recombinant Bacillus Calmette-Guerin (BCG)

The mainly used of current TB vaccine to prevent severe disease (miliary TB) in children under 5 years of age. The BCG vaccine protects against severe childhood forms of the disease but the BCG vaccine fails to protect against adult pulmonary TB in those countries in which the TB is endemic 16. Each year with BCG Roughly 100 million infants (more than 80% of the annual cohort) are vaccinated 17. Nearly a century ago the recombinant BCG vaccine was designed and BCG originally developed from Mycobacterium bovis. Vaccinating against MTB infection BCG is considered to be a safe cost effective option 15. However, in different countries multiple BCG vaccine trials carried out. These vaccines have shown to be variable in its degree of protection against MTB infection. The effectiveness ranging of BCG possesses from 0-80% and its efficacy is believed to wane with time, remaining protective for only 10-20 years 18.

The tuberculin reactivity following BCG vaccination depends on the age of administration. During infancy the administration of the BCG vaccine has been shown to be highly effective in protecting against severe cases of MTB due to the rapid waning of tuberculin reactions. After the first year of life BCG vaccination of individuals, however, has been shown to result in persistent tuberculin reactions 19. It is argued that the increased incidence of TB infection may be the reason of loss of protection from childhood through young adult life. For the variation in the protective efficacy an overall failure of the BCG vaccine several reasons have been suggested. The variations in the effectiveness of the BCG vaccine are believed to be either directly or indirectly related to genetic differences in vaccinated populations, dose and vaccination protocols, differences in BCG strains, administration of the vaccine to already sensitized or infected individuals, over-attenuated 68 parent strains, and the interaction between BCG and environmental mycobacteria which can mask or block its protective effect. The 0-80% variable efficacy of BCG and the difficulty of improving its protection have lead to the need for more effective tuberculosis vaccines 18.

The United States and other industrialized countries with low incidences of tuberculosis infection have discontinued the use of the BCG vaccine. BCG vaccination has been shown to affect TST which makes it difficult to interpret the results. Because there is no reliable method to distinguish between an individual displaying a true-positive TST because of actual MTB infection from an individual with a false-positive TST resulting from prior vaccination with BCG, creating a more effective vaccine to aid in the prevention of MTB is one of extreme importance 20.

## CONCLUSION

From this study it was concluded that the TB is still a challenge for the world population especially for the developing countries. For prevention and elimination of TB, new case detection, proper treatment, preventive measure, awareness regarding the disease are necessary.

## ACKNOWLEDGEMENT

The authors are thankful to unknown reviewer for their valuable comments.

## REFERENCES

1. Khan IM and Yassin KM. Urging health system research: identifying gaps and fortifying tuberculosis control in Pakistan. Croat Med J. 2002; 43(4): 480-484.

2. Fine P, Floyd S, Stanford J, Nkhosa P, Kasunga A, Chaguluka S, Warndorff D, Jenkins P, Yates M and Ponnighausj. Environmental mycobacteria in northern Malawi: implication for the epidemiology of tuberculosis and leprosy. Epidemiol Infect. 2001; 126 (3): 379-387.

3. Lundgren R, Norrman E and Asberg I. Tuberculous infection transmitted at autopsy. Tubercle. 1987; 68:147–150.

4. Beck-Sague C, Dooley SW and Hutton MD. Outbreak of multidrug-resistant Mycobacterium tuberculosis infections in a hospital: transmission to patients with HIV infection and staff. JAMA. 1992; 268:1280–1286.

5. Stop TB Partnership, The Global Plan to Stop TB: 2011-2015. World Health Organization. 2010. (http://www.stoptb.org/assets/documents/global/plan/TB_GlobalPlanToStopTB2011-2015.pdf)

6. World Health Organization. Groups at Risk: WHO Report on the Tuberculosis Epidemic. World Health Organization, Geneva, Switzerland.1996.

7. The Global Fund to Fight AIDS, Tuberculosis, and Malaria. The Global Fund is Alive and Well, but Global Health Progress is in Peril. December, 2011. (http://www.theglobalfund.org/en/mediacenter/announcements/2011-12)

8. WHO. Global Tuberculosis Control. WHO/CDS/TB/.133. WHO, Geneva, Switzerland. 2008.

9. World Health Organization. Global Tuberculosis Control. WHO, Geneva, Switzerland. 2011. (http://www.who.int/tb/publications/global_report/2011/gtbr11_full.pdf)

10. World Health Organization. Global tuberculosis Report 2012. World Health Organization, Geneva, Switzerland. 2012.

11. Davis AL. A historical perspective on tuberculosis and its control. In LB. Reichman and ES. Hershfield (Eds.) Tuberculosis: A comprehensive international approach. New York: Marcel Dekker. 2000; 3–54.

12. Victor TC, Van-Helden PD and Warren R. Prediction of drug resistance in M. tuberculosis: molecular mechanisms, tools, and applications. IUBMB Life. 2002; 53: 231–237.

13 Iseman MD. A Clinician's Guide to Tuberculosis. Baltimore: Lippincott Williams and Wilkens. 2006.

14. Schluger NW. Recent advances in our understanding of human host responses to tuberculosis. Respir Res. 2001; 2(3):157-163.

15. Orme IM. Tuberculosis vaccines: Current progress. Drugs. 2005; 65(17): 2437-2444.

16. Andersen P and TM. Doherty. The success and failure of BCG – implications for a novel tuberculosis vaccine. Nat Rev Microbiol. 2005; 3(8): 656-62.

17. Bourdin TB, Fine P and Dye C. Effect of BCG vaccination on childhood tuberculous meningitis and miliary tuberculosis worldwide: a meta-analysis and assessment of cost-effectiveness. Lancet. 2006; 367: 1173–1180.

18. Sable SB, Kalra M, Verma I and Khuller GK. Tuberculosis subunit vaccine design: The conflict of antigenicity and immunogenicity. Clin Immunol. 2007; 122(3): 239-51.

19. Menzies D. What does tuberculin reactivity after bacille calmette-guerin vaccination tell us? Clin Infect Dis. 2000; 3: 71-74.

20. Cohn DL. The effect of BCG vaccination on tuberculin skin testing. does it matter? Am J Respir Crit Care Med. 2001; 164 (6): 915-916.