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Review Article

TUBERCULOSIS: A LITERATURE REVIEW

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ABSTRACT

From the beginning, tuberculosis is one of the serious health problem and one of the biggest reason of death worldwide. There is the crucial challenge to the control the active disease such as tuberculosis, multidrug-resistant tuberculosis, extremely drug resistant tuberculosis & human immunodeficiency virus. The commence of tuberculosis by inhalation of infected, a droplet of airborne particles which contain Mycobacterium tuberculosis organisms. Hereafter, bacterial reproduction and proliferation ensue, and immunological containment of the viable bacteria. Thus, resulting in the latent tuberculosis infection, it is state of obstinate bacterial viability, control of the immune system, and shows no confirmation of clinically active tuberculosis. Currently, there are no any methods available for directly diagnosticate of M. tuberculosis infection in humans. Hereafter, latent tuberculosis infection is mainly diagnosed by using a method tuberculin skin test (TST) or interferon-γ release assays (IGRAs) by the stimulation by Mycobacterium tuberculosis. This article will review the epidemiology, transmission of tuberculosis, pathogenesis, clinical Features, diagnosis, and treatment of latent tuberculosis infection along with current advancement.

Keywords: Tuberculosis, multidrug-resistant tuberculosis, extremely drug resistant tuberculosis, tuberculin skin test (TST), interferon-γ release assays (IGRAs).

INTRODUCTION

Epidemiology

Tuberculosis is one of the major health problems worldwide, according to the World Health Organisation (WHO); approximately death 5,000 people per day ubiquitously the world, the leading cause of death is Tuberculosis (TB) & it is a single treatable infectious disease. The World Health Organisation (WHO) reported that 1.4 million deaths in 2015 due to TB[1,2]. In the year 2011, 8.7 million people were infected with active tuberculosis all over the world, in which 13% of people were infected with the human immunodeficiency virus (HIV) and total deaths due to HIV is approximately 1.4 million, and patients infected with active tuberculosis and HIV the total death is 430,000. Whereupon 310,000 people occurrence matter of multidrug-resistant tuberculosis, caused by Mycobacterium tuberculosis get resistant to drug isoniazid and rifampin. More than 60% of people were infected with active tuberculosis they from the Russian Federation, South Africa, Pakistan, China, and India[3,4]. (figure 1: Percentage of new Tuberculosis cases with Multidrug-resistance tuberculosis[2]). Where 84 countries reported that patients infected with extensively drug-resistant tuberculosis, patients with multidrug-resistant tuberculosis and resistance to all three injectable (capreomycin, amikacin, & kanamycin) antituberculosis drugs with fluoroquinolones drugs[3,5]. Tuberculosis is an airborne disease which mainly carried some infectious nuclei droplet of Mycobacterium tuberculosis. For tuberculosis seven species are very closely related they are, (i) Mycobacterium Bovis, (ii) Mycobacterium Africanum, (iii) Mycobacterium Microti, (iv) Mycobacterium Caprae, (v) Mycobacterium Pinnipedii, (vi) Mycobacterium Canetti, (vii) Mycobacterium Mungi.

In the human body, M. tuberculosis takes a long time to show any clinical symptoms, this phase is known as the latent tuberculosis bacterial infection. Once our immune system becomes enervated, due to concurrent disease or age, the bacteria get a turn to the active phase. In the human body, easily reactivation of the tuberculosis bacteria because the human immunodeficiency virus (HIV) enervate the immune system. Co-infection with HIV and Tuberculosis enhance the progress of each other, this becomes a reason to death[6,7]. During the active stage, TB is highly contagious disease and it can be generating by inhaling the airborne particles of M. tuberculosis. After inhaling, these bacteria are mainly entered and capture the alveolar macrophages, but they abstain from the immune system and they stay for a long time in the dormant stage. and under the immune compromised conditions of the host reactivated to virulent. Treatment of active tuberculosis makes challenging due to slow as well as fast growing diseases[1,8].

DEFINITION OF MULTI-DRUG RESISTANCE TUBERCULOSIS[9,10]

Drug-resistant tuberculosis is defined as the M. tuberculosis get resistant to one or more anti- tuberculosis drugs. Drug-resistant tuberculosis is the M. tuberculosis resistance to first-line antituberculosis drug Rifampicin (R) and Isoniazid (H) with or without resistance to any other drugs.

TYPES OF DRUG RESISTANCE

Drug Resistance tuberculosis is generally two types, (i) Primary and (ii) Acquired drug resistance· Primary drug resistance is defined as a drug resistance, for a patient who does not receive any anti-tubercular therapy in the past. Acquired drug resistance is the when the patient received anti-tuberculosis drug treatment

in past. The primary or acquired drug resistance tuberculosis is due to unaware of treatment or the confidential history of previous treatment, it is well known as initial drug resistance. Hence, initial drug resistance is due to primary and some unedited acquired resistance tuberculosis. Where the combined resistance is the sums of primary & acquired drug resistance.

TRANSMISSION OF TB[10]

Tuberculosis mainly carried some airborne particles, which we knew as droplet nuclei, it is in diameter of 1-5 microns. The droplet of infectious nuclei is generally produced when persons have laryngeal or pulmonary TB disease by sneeze, cough, sing, or shout. The droplet of infectious nuclei of airborne particles in the environment remains suspended in the air for several hours. Tuberculosis is mainly transmitted by the air, not transmitted by the surface contact. Transmission of Mycobacterium tuberculosis occurs when a person inhales droplet of infectious nuclei which contain M. tuberculosis, and the droplet of infectious nuclei in the human body cross the mouth or nasal cavity, upper respiratory tract, & alveoli of the lungs through bronchial tubes.

PATHOGENESIS

Patients who infected with active pulmonary tuberculosis the causing agent of active tuberculosis is the Mycobacterium tuberculosis. The persons those infected with active tuberculosis is more than 90% and the pathogen is included as asymptomatic latent infection. Recent studies explained that they raise the possibility of active tuberculosis if the people those infected with mycobacterium tuberculosis and the persons those receive and eject acute infection of mycobacterium tuberculosis[11]. The time required for showing the symptom of initial active pulmonary tuberculosis 5% in the 18 months and 5% in a remaining lifetime[12]. An approximately 2 billion people infected with latent infection and they have the risk for reactivation[3]. The persons those infected with a latent infection they reduce the chance of reinfection on incessant exposure, whereas the persons infected with active tuberculosis they enhanced the chance of reinfection a second episode of tuberculosis on incessant exposure[12,13]. Drug-resistant tuberculosis becomes apparent from unplanned chromosomal mutations. (Figure 2: Risk of mycobacterium tuberculosis infection and disease[56]). Drug-resistant tuberculosis is mainly caused by wrong use of antituberculosis drugs, such as the addition of single drugs to imperfection regimens or monotherapy, and that leads to the emergence of acquired resistance (resistant mutants). Transmission of such infected mycobacterium tuberculosis to any other person may cause initial infection and after some time primary resistance. Most deadly drug-resistant infection have been documented, especially in those people spreading of HIV infection is high[14,15,16,17]. The drug resistance tuberculosis failure to detect due to increase in the mortality rate, treatment failure, inappropriate prescription regimens, and transmission of such infected drug-resistant diseases[18].

CLINICAL FEATURES

The clinical symptom of pulmonary tuberculosis that shows in patients having a long-term cough, production of phlegm, lack of appetite, weight loss, continuously fever, sweating in the night, and hemoptysis (lung cancer related). Whereas that 10 to 42% of patients infected with active extrapulmonary tuberculosis, and it depends on age, existence or absence of underlying cause of disease, and immune response of the human body[19]. Active extrapulmonary tuberculosis can influence any parts of the body, and it has different clinical manifestations, and therefore extrapulmonary tuberculosis requires a high clinical indication. Person those who infected with HIV, clinical management have to phase the special challenges in patients with active tuberculosis. The patients those infected with HIV, the opportunity of active tuberculosis increased[20] and the persons those are HIV-negative at this stage they have a chance of active pulmonary diseases. The T lymphocytes (CD4 cell count or T cell count) if any persons those have less than 200 per cubic millimeter T cell counts, they increased the chance of active pulmonary tuberculosis, and 50% of patients found with extrapulmonary tuberculosis. If the CD4 cells counts found less than 75 per cubic millimeter, pulmonary tuberculosis may be absent, and outstretched tuberculosis, incurable illness with extensive organ participation and mycobacteremia, with a high rate of mortality[21]. Asymptomatic tuberculosis with a negative result of a chest radiography and sputum smear and culture results found to be positive is a common symptom of HIV-associated tuberculosis and 10% of cases in which tuberculosis is spatial[22,23]. Whereas up to 25% of HIV patients have unrecognized active tuberculosis[3]. Therefore, tuberculosis screening is strongly recommended for all patients with HIV infection to identify whether patients have active tuberculosis. The persons which any one of four symptoms weight loss, night sweats a cough and fever is strongly recommended for tuberculosis screening. Tuberculosis proactive screening is highly recommended in areas where the disease is highly spatial, since tuberculosis patients with noncommunicable diseases or HIV infection (example; tobacco-related chronic lung disease and diabetes mellitus and) may be missed[24,25].

DIAGNOSIS

Conventional methods

From the beginning, Lowenstein-Jensen culture methods have been used for the diagnosticate of M. tuberculosis drug sensitivity. Generally, methods used for the diagnosis of tuberculosis they are (*i*) the absolute concentration method (*ii*) the resistance ratio method (*iii*) the proportions method[26,27]. The conventional method required 6-8-week time to know the result of drug resistance tuberculosis.

In this conventional absolute concentration method, the minimal inhibitory concentration (MIC) is determined by inoculating the control medium culture and drug containing medium culture with a carefully controlled environment inoculum of M. tuberculosis. The medium culture containing respectively two times dilutions with each drug. Drug resistance is determined by the lowest concentration of the drug required for inhibiting the growth of bacteria, at the end of four-week formation of 20 or > 20 colonies.

In resistance ratio method, this technique mainly developed to control for the variability in growing rate of bacteria conditions by adding a reference control strain. This resistance ratio method is similar to the absolute concentration method. In this method, 2 mg inoculum Lowenstein-Jensen culture medium used. Then 2 mg of inoculum is poured out in to 0.4 ml of distilled water. The resistance ratio of minimal concentration inhibiting the growth of (test strain) is divided by the minimal concentration inhibiting the growth of (control strain). If the resistance ratio is found as 2 or less than 2, then it is defined as sensitive, resistance ratio is found as 8 or more than 8 is resistant.

In this proportion method, the bacilli are cultured on two media, one with drug-containing medium another one without drug containing a medium. The number of colonies obtained during incubation is the proportion of resistant bacilli and incubation with drug or without drug-containing medium and that allowed to monitoring inoculum size and below a certain proportion, strain become sensitive then it is said to be resistant. This technique uses the same Lowenstein-Jensen growth medium. Namely[26,27].

Latent Infection

The patients infected with latent Mycobacterium tuberculosis their diagnosis and treatment, because the latent infection is highly disseminated diseases, patients those are HIV infected they have a high risk of reactivation of diseases[28,29]. A tuberculin skin test (TST) or an interferon-gamma release assay (IGRAs) is recommended for mainly diagnosticate latent M. tuberculosis infection. The National Institute for Health & Clinical Excellence (NICE), the European Centre for Disease Prevention & Control (ECDC), and the Centers for Disease Control & Prevention (CDC) has recommended the interferon-gamma release assay (IGRAs) or tuberculin skin test (TST) for diagnosticate the latent M. tuberculosis infection[30,31,32]. The interferon-gamma release assay (IGRAs) is expensive, sensitive but it is less specific, whereas the tuberculin skin test (TST) is less expensive[33]. (Figure 3: Alternative strategies for screening for LTBI[57]).

Active Tuberculosis

Active tuberculosis is diagnosticated by a standard method using sputum microscopy and culture in liquid medium accompanied by ensuing drug-susceptibility testing. The solid medium culture is used in the diagnosis of active tuberculosis more economical in the support poor countries. No, any method was developed for the diagnosis of active tuberculosis, latent tuberculosis infection is diagnosed by using tuberculin skin tests (TST) or Interferon-gamma release assays[30,34]. Active tuberculosis & HIV infection are the high proliferation diseases, approximately more than 90% patients infected with extensively drug-resistant tuberculosis (XDR-TB) & multidrug-resistant tuberculosis (MDR-TB) and 30% of patients infected with active tuberculosis[3,5]. A new tool to diagnosticate tuberculosis called as the Xpert MTB/RIF assay, it detects bacteria M. tuberculosis within 2 hours. The Xpert MTB/RIF assay is highly sensitivity and much faster than sputum microscopy[35]. Therefore, the rate of detection is increased by 45%, when compared with sputum microscopy, for the patients those are infected by HIV[36]. The Xpert MTB/RIF assay has the possibilities to improve the performance of national tuberculosis programs and currently being implemented in 67 countries[3].

Modern methods for Drug-Resistant Tuberculosis

The radiometric method is developed for the diagnosis of drug-susceptibility testing of M. tuberculosis[37,38]. In this radiometric method Becton-Dickinson (BACTEC-460), medium (7H12) which contain palmitic acid, labeled as 14C-palmitic acid (radioactive carbon). As soon as these mycobacteria metabolize fatty acids after this radioactive carbon dioxide (14CO2) is liberated which mainly used to measure the bacterial growth. This method is modified by including the Becton-Dickinson (BACTEC-460) technique in the place of conventional method Lowenstein-Jensen culture and this Becton-Dickinson test gives the sensitivity results within 10 days[37,39].

The mycobacteria growth indicator tube (MGIT) and drug susceptibility testing (DST) is a non-radiometric and fast method for the diagnosis of drug resistance M. tuberculosis. The mycobacteria growth indicator tube which contains the silicone plug and the bottom of the test tube and that become fluorescent when the bacteria consume oxygen throughout the evolution of mycobacteria. The mycobacteria growth indicator tube (MGIT) tubes incubated into a the MGIT instrument and they observed for enhancement of fluorescence every 60 minutes. The growth of bacteria enhances the fluorescence (emission of light). The growth of bacteria is visually observed in the presence of non-homogeneous light turbidity or appearance of small granular in the medium[38,40].

The rifampicin drug resistance tuberculosis is detected by the FASTPlaqueTB-RIF method in M. tuberculosis cultures, and it developed by Biotec Laboratories Ltd (Ipswich, UK), & it gives the result within 48 hours. The FASTPlaqueTB-RIF methods mainly make use of mycobacteriophage technology for the finding of rifampicin drug resistance. Viable target tuberculosis bacilli are infected with this phage. For destroys any phage remaining outside the bacilli, the sample is treated with a viricide. The addition of a non-pathogenic M. smegmatis, a new phase is amplified which help in the phase replication. The sample is mixed into an agar mixture and kept overnight for incubation. This test can be performed in any laboratory and basic microbiological equipment, and it does not require any sophisticated equipment[41,42].

TREATMENT
Latent Infection

The persons infected latent M. tuberculosis infection they have the chance of active tuberculosis they required a deterrent treatment. The persons infected with HIV, the preferred dosages regimen is isoniazid for 9 months treatment and they have a high risk of spreading of tuberculosis[43,44]. The directly observed weekly administration reported that isoniazid and rifapentine 12 weeks dosages regimen shows more effective than isoniazid alone without infected HIV. Isoniazid and rifapentine have some serious adverse effect than 9 months of isoniazid alone and the discontinuation of treatment shows more adverse effect[45]. World Health Organisation (WHO) recommended that patients infected with HIV, with the positive or negative result of the tuberculin skin test (TST) & without active tuberculosis they must have to receive preventive dosages regimen with isoniazid for at least 6 months. Active tuberculosis with HIV-infected persons three dosages regimens are recommended (i) Daily dose of isoniazid for 6-9 months, (ii) Daily dose of rifampin and isoniazid for 3 months, (iii) Daily dose of rifampin and isoniazid twice weekly for 3 months[43,44]. Rifampin dosages regimens contain higher rates of drug toxicity. (Table 1: Daily recommend regimen for tuberculosis therapy). The active tuberculosis patients with HIV-infected difficult to diagnosticate use of isoniazid dosages regimens is a protective treatment. Active tuberculosis patients and their test for tuberculin skin test (TST) result found to be positive, use of isoniazid have reduced rates of active tuberculosis and mortality rate. Few month isoniazid therapy wanes active tuberculosis and terminates diseases. Use of 36 months isoniazid therapy as compared with 6 months isoniazid therapy reduces the chance of active tuberculosis by 43%[46]. A daily dosages regimen of rifapentine and isoniazid 1-month therapy reduces the chance active tuberculosis. being studied. The persons with active tuberculosis & HIV- infected, use of isoniazid is recommended as protective therapy[47].

Drug-Sensitive Active Tuberculosis

For successful tuberculosis therapy requirement of early and accurate diagnosis, monitoring of drug resistance and HIV, under the guidance of doctor administration drug. The four first-line drugs used in the treatment of active tuberculosis (i) isoniazid, (ii) rifampin, (iii) pyrazinamide, (iv) ethambutol, in trail condition, the cure rates of this drug achieve more than 95%. Tuberculosis therapy done into two phases with 6 months dosages regimens, for intensive phase 2 months drug therapy required of all four drugs (isoniazid, rifampin, pyrazinamide, & ethambutol) and for continuation phase 4 months drug therapy required two drugs (isoniazid & rifampin). If deterioration of these risk factor does not happen they include, immunosuppression, vast disease, cavitation, and a sputum culture test result remains positive till 8

weeks then treatment may be extended up to 9 months. There are some challenges in the treatment of tuberculosis they are, lack of the drug quality, to the administration of a drug to a patient by directly observed therapy, the support provided to patients, toxic effects, the obstacle in therapy due to changes in dosage regimen & side effect, pharmacokinetic interactions with an antiretroviral drug[3,48].

Tuberculosis and HIV Coinfection

Tuberculosis resultant enhancement of HIV replication, boost the progress of HIV infection, and that leads to high mortality. Start-up of antiretroviral therapy that reduces the mortality rate. The patients infected with active tuberculosis & HIV and still, they do not receive antiretroviral therapy, and the T-cell count is very less, have the perilous imperil of death. After initiation of tuberculosis treatment started within the first 8 weeks and it is recommended by World Health Organisation (WHO) and within 2 weeks patients infected with tuberculosis & HIV antiretroviral treatment begins for the patients, their T cell count < 50 per cubic millimeter. Patients those infected with tuberculous meningitis, start-up of antiretroviral treatment has increased the chance of adverse reaction. During the antiretroviral therapy, 10% of HIV-infected patients endure the immune reconstitution inflammatory syndrome (IRIS) and it also called as unmasking IRIS and clinical depolarize during tuberculosis treatment started after antiretroviral therapy called paradoxical IRIS. Deteriorating respiratory system and enhancement of lymphadenopathy is the most common side effect of immune reconstitution inflammatory syndrome (IRIS). Persons with less number of T cell count immune reconstitution inflammatory syndrome (IRIS) is more common, during the course of tuberculosis beginning of antiretroviral therapy. From the beginning of tuberculosis, antiretroviral therapy should start within 4 weeks for the patients that have T cell count < 50 per cubic millimeter. Active tuberculosis with HIV-infected patients the preferred dosages regimens, efavirenz is the first drug of choice with non-nucleoside reverse transcriptase inhibitors. The use of drug rifampin decreased blood serum concentrations of protease inhibitors. The replacement of rifabutin for rifampin enhancement of doses regimens for the boost up of protease inhibitors to avert the lack of blood serum concentration[49,50,51]. Active tuberculosis with HIV-infected patients recommended dose for preventive treatment with the combination of sulfamethoxazole & trimethoprim.

Multidrug-Resistant Tuberculosis

For the effective treatment of multidrug-resistant tuberculosis (MDR-TB) require a combination of five hierarchical group of first & second line antituberculosis drugs. (Table 2: MDR-TB treatment drugs and related toxicities). Such combination used in multidrug-resistant tuberculosis (MDR-TB) shows the intolerable and serious toxic effects. Such dosages regimens selected on a systemic or experiential basis and after drug susceptibility testing result become available, then it switched to individual treatment.

Although, trustworthy drug-susceptibility testing is not available in the regions, where tuberculosis is spatial, especially for second-line drugs. World Health Organisation (WHO) recommended the therapy for multidrug-resistant tuberculosis (MDR-TB) for intensive phase dosages regimen should be at least 8 months. A fluoroquinolone and injectable drug should be administered with a combination with at least four second-line drugs and that will have same or similar effectiveness (example-pyrazinamide). For patients, they do not they receive the previous multidrug-resistant tuberculosis therapy drug administered for at least 20 months, and for the patients, they receive the previous multidrug-resistant tuberculosis therapy drug administered for at least 30 months. One of the experimental results showed that administration of a dosages regimen for a shorter period of time 9 to 12 months, admissible effectiveness and some adverse reactions and this experiment was done with patients who does not receive previously the second line drug. This trail is done for shortening the dosages regimen for patients with multidrug- resistant tuberculosis (MDR-TB).

STREAM is a modified Bangladesh regimen, and it is comparing with the current WHO recommended a regimen for MDR-TB. The dosages regimen was replaced of gatifloxacin with moxifloxacin. The extensively drug-resistant tuberculosis is enormous to diagnosticate and treat in countries where tuberculosis is the endemic diseases. The patient with MDR-TB and HIV infection mortality rate is more than 98%. Various new drugs in the trial stage show the effectiveness against multidrug-resistant tuberculosis and extensively drug-resistant tuberculosis[52-55].

Table 1: Daily recommend regimen for tuberculosis therapy

Type of Infection	Daily recommended dose	Comments
Active disease		
Newly diagnosed cases that are not multi-drug resistant	Daily dose of Isoniazid, rifampin, ethambutol & pyrazinamide for (2 month - intensive phase), follow by isoniazid and rifampin for (4 month - continuation phase)	For prevention of isoniazid-induced neuropathy, pyridoxine is recommended
Multidrug-resistant disease (second-line antituberculosis drugs)	Pyrazinamide, fluoroquinolone, ethionamide, prothionamide, and either para-aminosalicylic acid or cycloserine	Treatment based on the diagnosis of initial testing and after drug-susceptibility test result. switched to drug fluoroquinolones (e.g. moxifloxacin or levofloxacin) is recommended
Latent infection	Daily dose of isoniazid (300 - mg) for 6 months or either 9-month therapy preferred	9-month therapy for HIV-infected persons, daily dose for 6 months also preferred with lower efficacy and it also extended to 36 months for decreased the risk of HIV-positive patients
	Weekly dose of isoniazid (900 – mg) with rifapentine (900 – mg) for 3 months administration under directly observed therapy	Higher completion rate with HIV-uninfected persons & shows the equal efficacy, as compared to 9 months isoniazid therapy
	Daily dose of rifampin (600 – mg) for 4-month therapy	Effective therapy for persons infected with silicosis
	Daily dose of isoniazid (300 – mg) with rifampin (600 – mg) for 3 months therapy	Effective therapy for HIV-infected persons
	Twice weekly dose of isoniazid (900 – mg) with rifampin (600-mg) for 3-month therapy	Effective therapy for HIV-infected persons

Table 2: MDR-TB treatment drugs and related toxicities

Group	Drug	Abbreviation	Common toxicities
Group 1 First-line drug	Pyrazinamide	Z [Pza]	Hyperuricemia and gout, Hepatotoxicity, Anorexia, vomiting
	Ethambutol	E [Emb]	Retrobulbar neuritis (Central or peripheral) in is not usually available
Group 2 Injectable drug	Aminoglycosides		Irreversible ototoxicity and Nephrotoxicity
	Kanamycin	[Km]	
	Amikacin	[Amk]	
	Cyclic polypeptide		
	Capreomycin	[Cm]	
Group 3 Fluoroquinolones	Moxifloxacin	[Mfx]	GI disturbance (nausea, vomiting & diarrhea), Neurologic disturbance (insomnia & Restlessness)
	Levofloxacin	[Lfx]	
	Ofloxacin	[Ofx]	
Group 4 Bacteriostatic Second-line drug	Terizidone	[Trd]	CNS effects (paranoia, mood changes, vertigo, confusion, psychosis, aggression, tremor)
	Cycloserine	[Cs]	
	Prothionamide	[Pto]	Hepatotoxicity and Hypothyroidism, GI intolerance (anorexia, nausea, vomiting, metallic taste) Neurotoxicity, Peripheral and optic neuropathy
	Ethionamide	[Eto]	
	Para-aminosalicylic acid	[Pas]	GI intolerance (anorexia, nausea, vomiting, diarrhea) Hepatotoxicity approx. 0.5%, Hypothyroidism
Group 5 Drug with unclear role in Multidrug resistance tuberculosis Treatment	High dose isoniazid	H [Inh]	Rash (2%), Neurotoxicity, CNS (seizure, hallucinations, mental changes, Depression, Hepatoxicity
	Clofazimine	[Cfz]	Red to brown skin, GI disturbance (nausea, vomiting & cramps)
	Linezolid	[Lzd]	Nausea, diarrhea & headache
	Amoxicillin/Clavulanate	[Amx/Clv]	Rarely, (hypersensitivity/anaphylaxis)
	Imipenam/Cilastatin	[Imp/Cln]	
	Clarithromycin	[Clr]	GI disturbance common (nausea, cramping, diarrhea, abnormal taste)

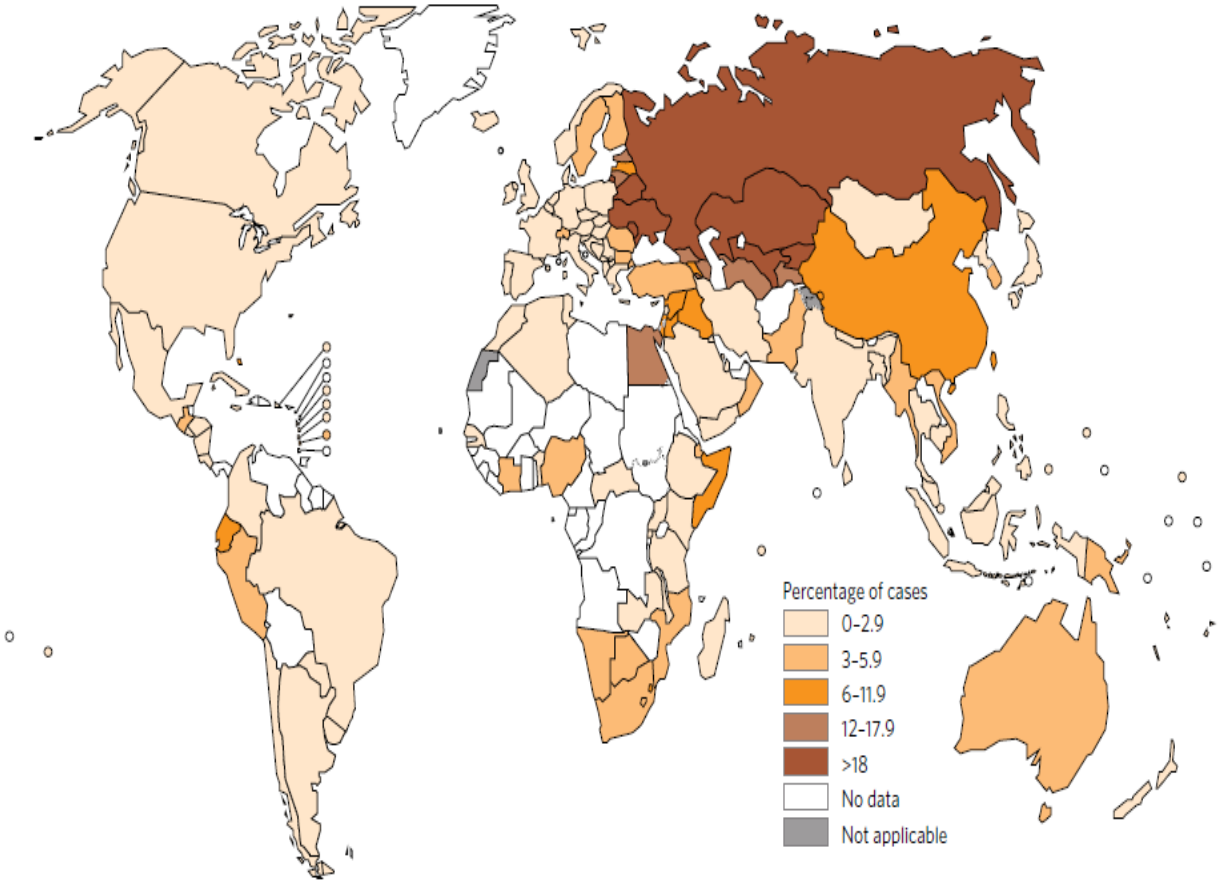

Figure 1: Percentage of new Tuberculosis cases with Multidrug-resistance tuberculosis[2]

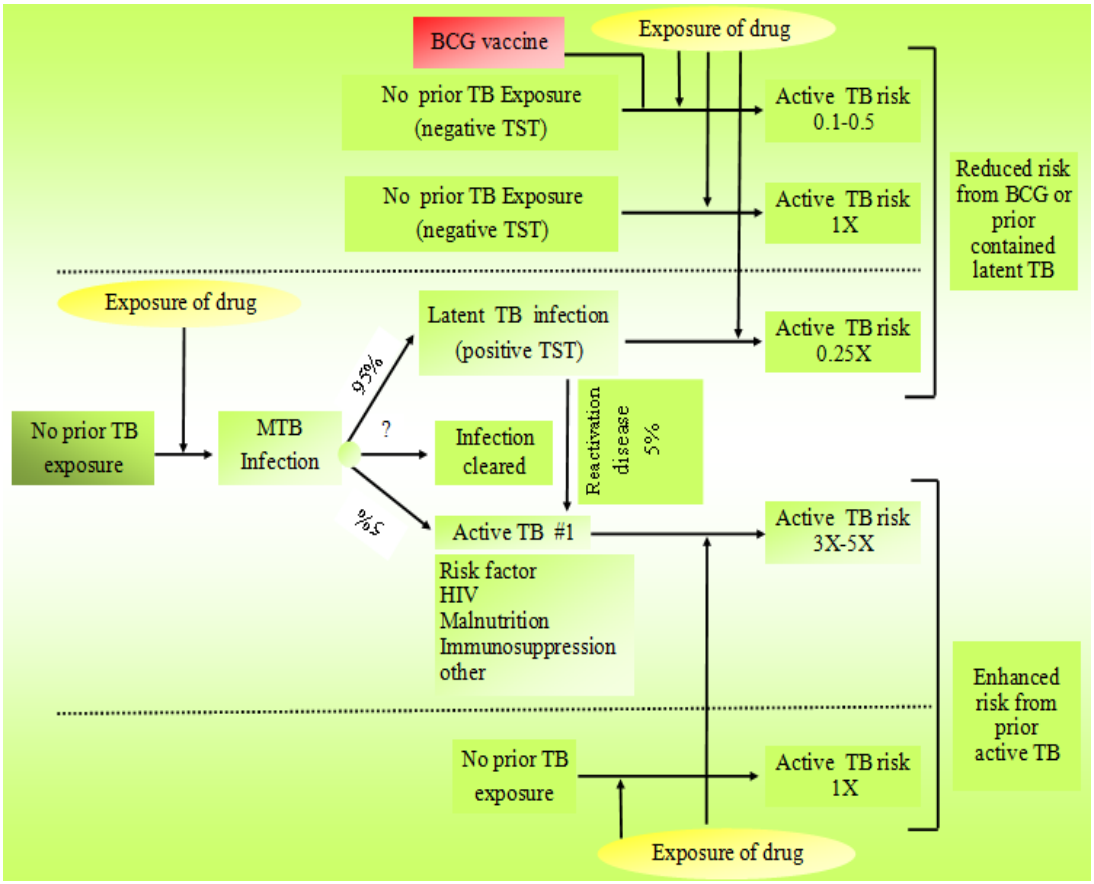

Figure 2: Risk of mycobacterium tuberculosis infection and disease[56]

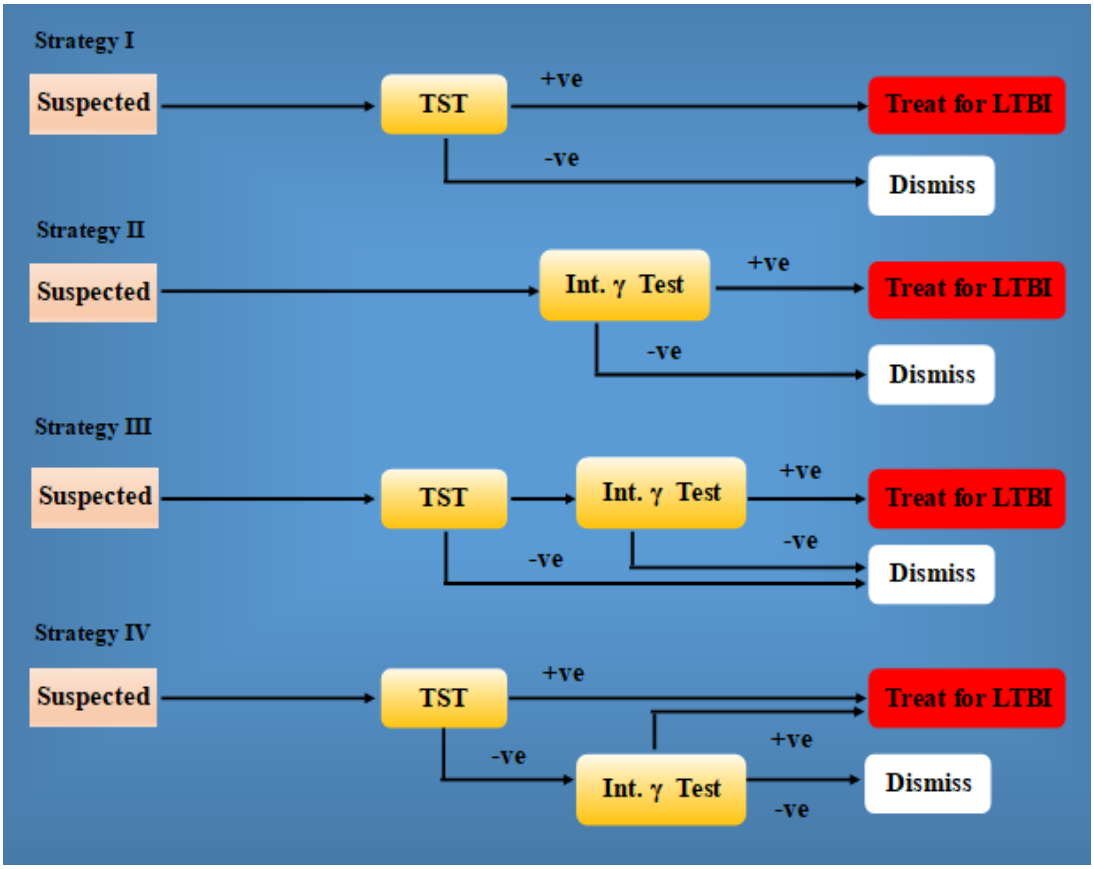

Figure 3: Alternative strategies for screening for latent tuberculosis bacterial infection (LTBI)[57]

CONCLUSION

The commence of tuberculosis by inhalation of infected, a droplet of airborne particles which contain Mycobacterium tuberculosis organisms. Hereafter, bacterial reproduction and proliferation ensue, and immunological containment of the viable bacteria. The enhancement and expansion of active tuberculosis and epidemic disease human immunodeficiency virus (HIV), are quite difficult to control and treatment of active tuberculosis. Modern diagnosis method and technology made easier to diagnosticate active tuberculosis disease. New antituberculosis drugs molecules have reduced the active tuberculosis treatment regimens for drug-sensitive tuberculosis and more effective treatment for latent bacterial infection tuberculosis & drug-resistant tuberculosis. Several new antituberculosis vaccines and drug molecules are in the clinical trials, the expectancy for control tuberculosis in future.

REFERENCES

1. Sarkar S, Mavanur R. Suresh. An overview of tuberculosis chemotherapy-a literature review. Journal of Pharmacy and Pharmaceutical Sciences 2011; 14:148-161.
2. Global tuberculosis report 2016 [Internet]. World Health Organization. 2017 [cited 20 May 2017]. Available from: http://www.who.int/tb/publications/global_report/en/
3. World Health Organization [Internet]. World Health Organization. 2017 [cited 20 May 2017]. Available from: http://www.who.int/en/
4. Zignol M, van Gemert W, Falzon D, Sismanidis C, Glaziou P, Floydet K, et al. Surveillance of anti-tuberculosis drug resistance in the world: an updated analysis, 2007-2010. Bulletin of the World Health Organization 2012; 90:111-119.
5. Tuberculosis MDR-TB and XDR-TB: 2011 progress report. Geneva: World Health Organization, [Internet]. 2017 [cited 20 May 2017]. Available from: http://www.who.int/tb /challenges/mdr/factsheet_mdr_progress_march2011.pdf
6. Goletti D, Weissman D, Jackson RW, Graham NM, Vlahov D, Klein RS, et al. Effect of Mycobacterium tuberculosis on HIV replication. Role of immune activation. The Journal of Immunology 1996; 157:1271-1278.
7. Mariani F, Goletti D, Ciaramella A, Martino A, Colizzi V, Fraziano M, et al. Macrophage response to Mycobacterium tuberculosis during HIV infection relationships between macrophage activation and apoptosis. Current Molecular Medicine 2001; 1:209-216.
8. Strohmeier GR, Fenton JM. Roles of lipoarabinomannan in the pathogenesis of tuberculosis. Microbes and Infection 1999; 1:709-717.
9. Prasad R. MDR TB: current status. Indian Journal of Tuberculosis 2005; 52:121-131.
10. Transmission and Pathogenesis of Tuberculosis - CDC, https://www.cdc.gov/tb/education/corecurr/pdf/chapter2.pdf.
11. Ewer K, Millington KA, Deeks JJ, Alvarez L, Bryant G, Lalvani A, et al. Dynamic antigen-specific T-cell responses after point-source exposure to Mycobacterium tuberculosis. American Journal of Respiratory and Critical Care Medicine 2006; 174:831-839.
12. Andrews JR, Noubary F, Walensky RP, Cerda R, Losina E, Horsburgh CR, et al. Risk of progression to active tuberculosis following reinfection with Mycobacterium tuberculosis. Clinical Infectious Diseases 2012; 54:784-91.
13. Lahey T, Mackenzie T, Arbeit RD, Bakari M, Mtei L, Mateeet M, et al. Recurrent tuberculosis risk among HIV infected adults in Tanzania with prior active tuberculosis. Clinical Infectious Diseases 2013; 56:151-158.
14. Frieden TR, Munsiff SS, Ahuja SD. Outcomes of multidrug-resistant tuberculosis treatment in HIV-positive patients in New York City, 1990-1997. International Journal of Tuberculosis and Lung Disease 2007; 11:116.
15. Dheda K, Shean K, Zumla A, Badri M, Streicher EM, Willcox P, et al. Early treatment outcomes and HIV status of patients with extensively drug-resistant tuberculosis in South Africa: a retrospective cohort study. Lancet Respiratory Medicine 2010; 375:1798-1807.
16. Udwadia ZF, Amale RA, Ajbani KK, Rodrigues C. Totally drug-resistant tuberculosis in India. Clinical Infectious Diseases 2012; 54:579-581.
17. Totally Drug-Resistant TB: a WHO consultation on the diagnostic definition and treatment options. Geneva: World Health Organization (http://www.who.int/tb/challenges/ xdr/Report_Meeting_totallydrugresistantTB_032012.pdf).
18. Nathanson E, Nunn P, Uplekar M, Floyd K, Jaramillo E, Lonnroth K, et al. MDR tuberculosis-critical steps for prevention and control. The New England Journal of Medicine 2010; 363:1050-1058.
19. Caws M, Thwaites G, Dunstan S, Hawn TR, Thi Ngoc Lan N, Thuong NTT, et al. The influence of host and bacterial genotype on the development of disseminated disease with Mycobacterium tuberculosis. PLOS Pathogens 2008; 4: e1000034.
20. Von Reyn CF. Optimal treatment of codisease due to HIV and tuberculosis. The Journal of Infectious Diseases 2011; 204:817-819.
21. Von Reyn CF, Kimambo S, Mtei L. Disseminated tuberculosis in human immunodeficiency virus infection: ineffective immunity, polyclonal disease and high mortality. The International Journal of Tuberculosis and Lung Disease 2011; 15:1087-1092.
22. Mtei L, Matee M, Herfort O, Bakari M, Horsburgh CR, Waddell R, et al. High rates of clinical and subclinical tuberculosis among HIV-infected ambulatory subjects in Tanzania. Clinical Infectious Diseases 2005; 40:1500-1507.
23. Cain KP, McCarthy KD, Heilig CM, Monkongdee P, Tasaneeyapan T, Kanara N, et al. An algorithm for tuberculosis screening and diagnosis in people with HIV. The New England Journal of Medicine 2010; 362:707-716.
24. Getahun H, Kittikraisak W, Heilig CM, Corbett EL, Ayles H, Cain KP, et al. Development of a standardized screening rule for tuberculosis in people living with HIV in resource-constrained settings: individual participant data metaanalysis of observational studies. PLOS Medicine 2011; 8: e1000391.
25. Bates M, O Grady J, Mwaba P, Chilukutu L, Mzyece J, Cheelo B, et al. Evaluation of the burden of unsuspected pulmonary tuberculosis and co-morbidity with non-communicable diseases in sputum producing adult inpatients. PLOS One 2012; 7: e40774.
26. Vareldzis BP, Grosset J, de Kantor I, Crofton J, Laszlo A, Felten M, et al. Drug-resistant tuberculosis: laboratory issues. World Health Organization recommendations. Tubercle and Lung Disease 1994; 75: 1-7.
27. Citron KM, Girling DJ. Tuberculosis. In: Weatherall DJ, Ledingham JGG, Warrel DA, editors. Crofton and Douglas respiratory diseases. Oxford: Oxford University Press/English Language Book Society;1987; 5.278-5.299.
28. Horsburgh CR Jr, Rubin EJ. Latent tuberculosis infection in the United States. The New England Journal of Medicine 2011; 364:1441-1448.
29. Ferrara G, Murray M, Winthrop K, Centis R, Sotgiu G, Migliori GB, et al. Risk factors associated with pulmonary tuberculosis: smoking, diabetes and anti-TNFα drugs. Current Opinion in Pulmonary Medicine 2012; 18:233-240.
30. Updated guidelines for using interferon gamma release assays to detect Mycobacterium tuberculosis infection-United States, 2010. The Morbidity and Mortality Weekly Report 2010; 59:1-25.

31. Tuberculosis: clinical diagnosis and management of tuberculosis, and measures for its prevention and control. London: National Institute for Health and Clinical Excellence, 2011 (http://www.nice.org.uk/nicemedia/live/13422/53642/53642.pdf).
32. Use of interferon-gamma release assays in support of TB diagnosis [Internet]. 2017 [cited 20 May 2017]. Available from: http://ecdc.europa.eu/en/publications/Publications/1103_GUI_IGRA.pdf
33. Mc Nerney R, Maeurer M, Abubakar I, Marais B, McHugh TD, Ford N, et al. Tuberculosis diagnostics and biomarkers: needs, challenges, recent advances, and opportunities. The Journal of Infectious Diseases 2012; 205:147-158.
34. Hopewell PC, Pai M, Maher D, Uplekar M, Raviglione MC. International standards for tuberculosis care. The Lancet Infectious Diseases 2006; 6:710-25.
35. Boehme CC, Nabeta P, Hillemann D, Nicol MP, Shenai S, Krapp F, et al. Rapid molecular detection of tuberculosis and rifampin resistance. The New England Journal of Medicine 2010; 363:1005-15.
36. Lawn SD, Kerkhoff AD, Vogt M, Ghebrekristos Y, Whitelaw A, Wood R, et al. Characteristics and early outcomes of patients with Xpert MTB/RIF-negative pulmonary tuberculosis diagnosed during screening before antiretroviral therapy. Clinical Infectious Diseases 2012; 54:1071-1079.
37. Roberts GD, Goodman NL, Heifets L, Larsh HW, Lindner TH, McClatchy JK, et al. Evaluation of the BACTEC radiometric method for recovery of mycobacteria and drug susceptibility testing of Mycobacterium tuberculosis from acid-fast smear-positive specimens. Journal of Clinical Microbiology 1983; 18:689-696.
38. Adjers-Koskela K, Katila ML. Susceptibility testing with the manual mycobacteria growth indicator tube (MGIT) and the MGIT 960 system provides rapid and reliable verification of multidrug-resistant tuberculosis. Journal of Clinical Microbiology 2003; 41:1235-1239.
39. Lee CN, Heifets LB. Determination of minimal inhibitory concentrations of antituberculosis drugs by radiometric and conventional methods. American review of respiratory disease 1987; 136:349-352.
40. Bemer P, Palicova F, Rusch-Gerdes S, Drugeon HB, Pfyffer GE. Multicenter evaluation of fully automated BACTEC Mycobacteria Growth Indicator Tube 960 system for susceptibility testing of Mycobacterium tuberculosis. Journal of Clinical Microbiology 2002; 40:150-154.
41. Takiff H, Heifets L. In search of rapid diagnosis and drug resistance detection tools: is the FASTPlaqueTB test the answer? International Journal of Tuberculosis and Lung Disease 2002; 6:560-561.
42. Albert H, Trollip AP, Mole RJ, Hatch SJ, Blumberg L. Rapid indication of multidrug-resistant tuberculosis from liquid cultures using FASTPlaqueTB-RIF, a manual phage-based test. International Journal of Tuberculosis and Lung Disease 2002; 6: 523-528.
43. Martinson NA, Barnes GL, Moulton LH, Msandiwa R, Hausler H, Ram M, et al. New regimens to prevent tuberculosis in adults with HIV infection. The New England Journal of Medicine 2011; 365:11-20.
44. Guidelines for intensified tuberculosis case-finding and isoniazid preventive therapy for people living with HIV in resource-constrained settings. Geneva: World Health Organization, 2011 (http://whqlibdoc.who.int/publications/2011/9789241500708_eng.pdf).
45. Sterling TR, Villarino ME, Borisov AS, Shang N, Gordin F, Bliven-Sizemore E, et al. Three months of rifapentine and isoniazid for latent tuberculosis infection. The New England Journal of Medicine 2011; 365:2155-2166.
46. Samandari T, Agizew TB, Nyirenda S, Tedla Z, Sibanda T, Shang N, et al. 6-Month versus 36-month isoniazid preventive treatment for tuberculosis in adults with HIV infection in Botswana: a randomised, double-blind, placebo-controlled trial. The Lancet 2011; 377:1588-1598.
47. Lawn SD, Wood R. Antiretroviral therapy for control of the HIV-associated MDR and XDR tuberculosis epidemic in South Africa. American Journal of Respiratory and Critical Care Medicine 2010; 182:1567.
48. Combs DL, O Brien RJ, Geiter LJ. USPHS Tuberculosis Short-Course Chemotherapy Trial 21: effectiveness, toxicity and acceptability: the report of the final results. Annals of Internal Medicine 1990; 112:397-406.
49. Abdool Karim SS, Naidoo K, Grobler A, Padayatchi N, Baxter C, Gray AL, et al. Integration of antiretroviral therapy with tuberculosis treatment. The New England Journal of Medicine 2011; 365:1492-501.
50. Blanc FX, Sok T, Laureillard D, Borand L, Rekacewicz C, Nerrienet E, et al. Earlier versus later start of antiretroviral therapy in HIV-infected adults with tuberculosis. The New England Journal of Medicine 2011; 365:1471-1481.
51. ClinicalTrials.gov. Rifampin-based tuberculosis treatment versus rifabutin based tuberculosis treatment in HIV (http://clinicaltrials.gov/ct2/show/NCT01601626).
52. Falzon D, Jaramillo E, Schunemann HJ, Arentz M, Bauer M, Bayona J, et al. WHO guidelines for the programmatic management of drug-resistant tuberculosis: 2011 update. European Respiratory Journal 2011; 38:516-528.
53. Van Deun A, Salim MA, Das AP, Bastian I, Portaels F. Results of a standardized regimen for multidrug-resistant tuberculosis in Bangladesh. International Journal of Tuberculosis and Lung Disease 2004; 8:560-567.
54. STREAM: the evaluation of a standardized treatment regimen of anti-tuberculosis drugs for patients with multi-drug resistant tuberculosis (MDR-TB). 2011 (http://www.controlled trials.com/isrctn/pf/78372190).
55. Jacobson KR, Tierney DB, Jeon CY, Mitnick CD, Murray MB. Treatment outcomes among patients with extensively drug-resistant tuberculosis: systematic review and meta-analysis. Clinical Infectious Diseases 2010; 51:6-14.
56. Zumla A, Raviglione M, Hafner R, Von Reyn C.F. Current Concepts Tuberculosis. The New England Journal of Medicine 2013; 368:745-55.
57. Dinnes J, Deeks J, Kunst H, Gibson A, Cummins E, Waugh N, et al. A systematic review of rapid diagnostic tests for the detection of tuberculosis infection. Health Technology Assessment 2007; 11:133-145.

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