Introduction
Tuberculosis (TB) is the most common life-threatening opportunistic infection (OI) in patients with HIV/AIDS. According to World Health Organization (WHO) about one third of the world’s population is infected with *Mycobacterium tuberculosis*. Increasing global burden of tuberculosis is linked to Human Immunodeficiency Virus (HIV) infection. In addition, HIV infection is one of the greatest risk factors for development of TB (Corbett *et al.*, 2003). The eventual destruction of immune system by HIV makes them particularly susceptible to the development of opportunistic infections, including a variety of fungal, viral, bacterial and protozoan diseases. It is estimated that 50 to 60% of HIV positive persons in India will develop TB in their lifetime and also more than 36 million people in the world are affected by HIV/AIDS (Swaminathan, *et al.*, 2000).

Infection with tubercle bacillus (most often *Mycobacterium tuberculosis*) is characterized by the formation of tubercles, hard nodules in the lungs that are the result of interaction between the bacteria and the host’s immune system. When *M. tuberculosis* enters the lungs, phagocytic cells of the host’s immune system, called macrophages, engulf the pathogen, but are unable to digest the bacteria due to its waxy mycolic acid cell wall. The Mycobacteria multiply within the macrophages, eventually killing these cells that are supposed to protect the host. The cycle continues as the bacteria released from the dead macrophages are then engulfed by other macrophages. While this immune system drama is being played out internally, the host normally shows a few external signs of infection, other than fever (Greenwell-Wild *et al.*, 2002).
The infected macrophages result in an inflammatory response (heat, swelling, dilated capillaries) which attracts more macrophages until the site of infection is completely surrounded by many of these compressed phagocytic cells. Inflammation triggers other cells within the host to essentially quarantine the area by depositing collagen fibers around the packed macrophages, forming an enclosed infection within the lung called a tubercle. The cells at the centre of the tubercle may eventually die, producing either an area of necrosis or an actual cavity (Converse et al., 1996). The majority of TB infections are restricted to the lungs, after inhalation of respiratory droplets expelled when an infected individual coughs, sneezes or speaks. Still, it is possible for the bacteria to disseminate and cause infection in other areas of the body.

Tuberculosis and HIV coinfection is recognized as a major setback to both tuberculosis and HIV co-infection control programme (Godfrey-Faussett et al., 2002). HIV is a strong risk factor for tuberculosis and contributes to the development of active tuberculosis from latent and exogenic reinfection. Tuberculosis and HIV coinfection has a negative impact on TB control programme by increasing case load due to excess incidence attributable to HIV infection (Corbett et al., 2003). Global incidence of TB is estimated at 8 million new cases yearly with 3 million deaths (De cock et al., 1996). An increase in TB incidence has been observed in the countries with a high prevalence of both tuberculosis and HIV infection (Chum et al., 1996). Case definition and diagnosis of TB is altered in case of TB with HIV coinfection. This can contribute to underdiagnosis or over diagnosis of smear negative disease. Delay of diagnosis could increase the case fertility ratio (CRF) (De Cock et al., 1996 & Mukadi et
Multidrug resistance and low treatment completion has been observed among TB/HIV coinfected patients (Weltman and Rose 1994) resulting in increased tuberculosis mortality and CFR (Mukadi et al., 2001). Tuberculosis and HIV coinfected cases have lower treatment compliance, more frequent drug adverse reactions, and intolerance to drug ingestion which further impairs the treatment outcome (Pozniak et al., 1999). The coinfected patients could be a source for HIV transmission to TB patients in health setup through inadequate sterilization of instrument for treatment procedure (World Health Organisation, 1989).

In addition to the burden HIV and tuberculosis, malaria is another major infectious disease responsible for causing more death in developing countries (Adjuik et al., 2006). It is estimated that over 5.6 million people are killed by HIV/AIDS, tuberculosis, malaria yearly (Tan et al., 2003). Malaria causes relatively higher mortality in HIV and tuberculosis infected patients (Adjuik et al., 2006). These three diseases constitute the most serious health challenges in sub-Saharan Africa (Breman, 2001).

In India overall prevalence of HIV infection is less than one percent and hence India continues to be in the category of low prevalence countries. Unfortunately, these are the parts of the world where TB has been flourishing unhindered since ages, forming a deadly synergy. Globally, 9% of all new TB cases (31% in Africa) in adults were attributable to HIV/AIDS, as were 12% of the 1.8 million deaths from TB, in the year 2000 (Sharma, 2005). As the result of HIV/AIDS, incidence rates of TB in certain countries have gone up to more than 6 percent per year crippling the already overburdened health care resources. TB accounts for about 13 percent of all
HIV-related deaths worldwide (Harries, 2004). Of the 5.1 million HIV infected people in India, about half of them are co-infected with *M. tuberculosis*. Respiratory infections continue to be common in HIV-1 infected individuals, even with the advent of the era of highly active antiretroviral therapy (Posner, 2002). The lifetime risk of developing TB is 50-70 percent persons dually infected with HIV and TB, as compared to a 10% risk in HIV negative individuals (Praveen Kumar *et al.*, 2002). Thus, TB is a leading cause of morbidity and mortality in patients with HIV/AIDS (Russell, 2004).

HIV and TB are also intricately linked to malnutrition, unemployment, alcoholism, drug abuse, poverty and homelessness (Sharma *et al.*, 2005). The direct and indirect costs of illness due to TB and HIV are enormous, estimated to be more than 30 percent of the annual household income in developing countries. It has a catastrophic impact on the economy in the developing world (Havlir and Barnes, 1999). HIV breaks down the immune system and makes patients highly susceptible to TB. Thus, co-infection with HIV and TB (HIV-TB) is not only a medical malady, but a social and an economic disaster and is aptly described as the “cursed duet”.

HIV infected persons who become newly infected by *M. tuberculosis* rapidly progress to active TB. HIV will worsen the TB epidemic. These patients can then spread TB to other people (Vaidyanathanl and Sanjay, 2003). TB is the most common serious opportunistic infection occurring among HIV-positive persons and is the first manifestation of AIDS in more than 50% of cases in developing countries. Perneger *et al.*, (1995) reported that, about 50-60% of HIV-positive patients in India will develop TB in their lifetime. The HIV epidemic has tripled TB cases in some countries (Kenneth, 1998). In a developing country like
India, the potential extra burden of new TB cases attributable to HIV could overwhelm budgets and support services, as has already happened in countries most heavily affected by the HIV epidemic. TB shortens the survival of patients with HIV infection. It is the cause of death for one out of every three people with AIDS worldwide. It may accelerate the progression of HIV to six- seven fold increase in HIV viral load (VL) in TB patients (Horsburgh et al., 1993).

The co-epidemics of tuberculosis and HIV require a concerted effort to tackle. This is of prime importance due to the unholy nexus between these two diseases. The two epidemics need a joint effort from both TB as well as HIV/AIDS control programmes. The strategies should be complementary even though they may be different in nature. The best approach to curb the HIV epidemic has so far been based on preventive interventions since a cure is not yet available. Unlike HIV, tuberculosis is curable in all, including the HIV infected. This concept of joint action for a synergistic impact has been in place from 2001 onwards in India, and presently covers six high HIV prevalence states and eight moderately prevalence states of India. The key part in the action plan is the coordination between the Designated Microscopy Centre (DMC) of the TB control programme and the Voluntary Counselling and Confidential Testing Centres (VCCTCs) of the HIV/AIDS control programme. These are present under the same roof in almost all the high HIV prevalence districts, to facilitate speedy cross-referrals of co-infected patients. To minimise the stigma associated with HIV, confidentiality is maintained at the VCCTC level and TB is treated irrespective of HIV status. The HIV status of the patient is not disclosed to the tuberculosis control programme while on anti-TB treatment.
Tuberculosis is one of the most frequent opportunistic infection in HIV infected individuals. The HIV epidemic has been recognized as a major factor contributing to the increase of tuberculosis in both the developing and developed countries. There are gaps in understanding the TB control measures in the era of both tuberculosis and HIV epidemics. There are only a few studies on the situation regarding TB and HIV coinfection in India. Earlier data were from all cases of TB under treatment while this study is the first on newly diagnosed cases. There were no earlier studies on the prevalence and pattern of the tuberculosis and HIV. So there is a lack of data on the impact of TB and HIV co-infection on the sputum smear results and health status. Hence, the aim of the present study was undertaken to analyse coexisting infections and their impact on health status of tuberculosis patients. Objectives of the present study were as follows,

I. To analyze the prevalence and pattern of various bacterial isolates of Lower Respiratory Tract Infection (LRTI) among subjects with and without HIV and to compare them with published reports.

II. To find out the prevalence and pattern of parasitic infestation among chronic diarrhoeal patients with and without HIV/AIDS in Madurai, south India.

III. To find out the prevalence of Pneumocystis carinii pneumonia among HIV infected patients.

IV. To understand the haematological status of pulmonary tuberculosis (PT) patients with and without HIV infection,

V. To study serum zinc and albumin status in adult pulmonary tuberculosis (PTB) with and without HIV co infection and compare with healthy control.