1. Introduction

1.1. Introduction

Tuberculosis (TB) has greater impact on morbidity and mortality in human immunodeficiency virus (HIV)-infected individuals than all other opportunistic infections. In fact, the rising incidence of TB in many regions of the world is closely related to the HIV epidemic. Approximately 1/3 of the 40 million people infected with HIV worldwide are co-infected with bacillus of *Mycobacterium tuberculosis* (MTB) complex (*M. tuberculosis*, *M. africanum*, *M. bovis*, *M. canetti* or *M. microti*). The prevalence of HIV in TB patients in Africa has been reported to be about 40% and the incidence of TB is more than 8 times higher in HIV-positive than in HIV negative people (Corbett, *et al*., 2006). Co-infection with HIV leads to challenges in both the diagnosis and treatment of tuberculosis. Further, there has been an increase in rates of drug resistant tuberculosis, including multi-drug (MDR-TB) and extensively drug resistant TB (XDRTB), which are difficult to treat and contribute to increased mortality. Because of the poor performance of sputum smear microscopy in HIV-infected patients, (Padmapriyadarsini, *et al*., 2011), HIV and MTB infections have synergic influence on the host immunoregulation. Indeed, HIV infection impairs cell-mediated immunity largely through depletion of CD4+ lymphocytes. The impaired immunity leads to increased number of cases of primary TB and reactivation TB in HIV-infected people. In turn, TB may accelerate the progression of HIV infection by increasing viral replication and intensifying immunodepression effect of HIV. It is likely that TB enhances immunodeficiency related to HIV infection. The incidence of primary TB and reactivation TB is increased in HIV-infected patients in comparison with HIV seronegative individuals the factors that lead to TB reactivation in HIV infection have not been determined in detail and the pathogenesis of TB is not always dependent on the stage of immunodeficiency (Hoffmann, *et al*., 2007).

In the individual host the two pathogens, *M. tuberculosis* and HIV, potentiate one another, accelerating the deterioration of immunological functions and resulting in premature death if untreated. Some 14 million individuals worldwide are estimated to be dually infected. TB is the largest single cause of death in the setting of AIDS, accounting for about 26% of AIDS-related deaths, 99% of which occur in developing countries

(Pawlowski, *et al*,. 2012). In some areas of sub-Saharan Africa, the rates of co-infection even exceed 1,000 per 100,000 populations. Approximately one-third of the 38.6 million HIV positive individuals in the world are infected with TB and are at increased risk of developing tuberculosis too. All persons who were diagnosed with HIV infection should be tested for TB and people living with HIV and at risk for TB exposure should also be tested annually for latent TB infection. Tuberculosis is the most common cause of mortality among patients with AIDS in the world (Alavi-Naini and Parsi., 2012).

At least one-third of the 34 million people living with HIV worldwide are infected with latent TB. Persons co-infected with TB and HIV are 21-34 times more likely to develop active TB disease than persons without HIV (WHO 2013). The estimates of the global burden of disease caused by TB in 2009 were as follows: 9.4 million incident cases (range 8.9-9.9 million), 1.3 million deaths among HIV-negative TB patients (range 1.2-1.5 million) and 0.38 million deaths among HIV-positive TB patients (range 0.32-0.45 million). Most TB cases were in the South-East Asia, African and Western Pacific regions (35, 30 and 20%, respectively). An estimated 11-13 per cent of incident cases were HIV-positive. TB may occur at any stage of HIV disease and is frequently the first recognized presentation of underlying HIV infection (Padmapriyadarsini, 2011).

Globally, 30% of HIV-infected persons are estimated to have concomitant (usually latent) infection with *M. tuberculosis*; this percentage varies from 14% in Europe to 46% in Southeast Asia (G etahun., *et al*, 2010), HIV prevalence among TB cases was estimated 13% and 12% in the world, eastern Mediterranean region respectively (Alavi-Naini and Parsi ., 2012).

1.2 Objectives:

1.2.1 General Objective:

- To evaluate the incidence of HIV in Tuberculosis patients.

1.2.2 Specific objectives:

- To detect HIV 1+2 antibodies using sandwich ELISA
- To promote early knowledge of their HIV status.
- To help in HIV prevention to reduce their risk of transmitting or acquiring HIV.
- To determine the correlation between HIV and TB infection.

1.3. Rationale:

HIV screening is very important for TB patients; TB is the largest single cause of death in the setting of AIDS, co-infection with HIV leads to challenges in both the diagnosis and treatment of tuberculosis. Further, there has been an increase in rates of drug resistant tuberculosis including multi-drug (MDR-TB).

2. Literature review

2.1. Human Immunodeficiency Virus (HIV)

The first cases of AIDS were seen by physicians in Los Angeles, San Francisco, and New York City. They observed clusters of young male patients with one or more of a complex of symptoms: severe pneumonia caused by *Pneumocystis* (carinii) *jiroveci* (ordinarily a harmless fungus); a rare vascular cancer called Kaposi sarcoma; sudden weight loss; swollen lymph nodes; and general loss of immune function. Eventually, virologists at the Pasteur Institute in France isolated a novel retrovirus, later named the human immunodeficiency virus (HIV) (Fig 1). The cluster of symptoms resulting from the degradation of the immune system by HIV was termed acquired immunodeficiency syndrome, or AIDS, by the medical community (Talaro, 2012). Human immunodeficiency virus type 1 (HIV-1) was first isolated in 1983. Several further HIV-1 isolates were reported in 1984 from the United States which, together with serological studies of prevalence, made a convincing case for HIV being the cause of AIDS. HIV-2 was first isolated in 1986. Retrospective serological surveys indicated that HIV-1 had begun to spread among American gay men from 1977 onwards, showing a considerable incubation period before the manifestation of AIDS. The earliest known positive blood sample was collected in 1959 in Congo. However, molecular clock analyses of diversity suggest a common origin for HIV-1 (Group M) dating from around 1931 and for HIV-2 from around 1940; HIV-1 and HIV-2 represent two separate viruses with distinct origins. Both viruses belong to the Lentivirus genus of retroviruses and have a similar genome organization, but some of their accessory genes differ (Zuckerman, *et al.*, 2009).

2.2. Transmission routes:

HIV transmission occurs mainly through two forms of contact: sexual intercourse and transfer of blood or blood products. Babies can also be infected before or during birth, as well as through breast feeding (Talaro, 2012). Infection through artificial insemination, skin grafts and organ transplants is also possible); sharing unsterilized injection equipment that has been previously used by someone who is infected; maternofetal transmission (during pregnancy, at birth, and through breastfeeding) (Hoffmann, *et al*., 2007).

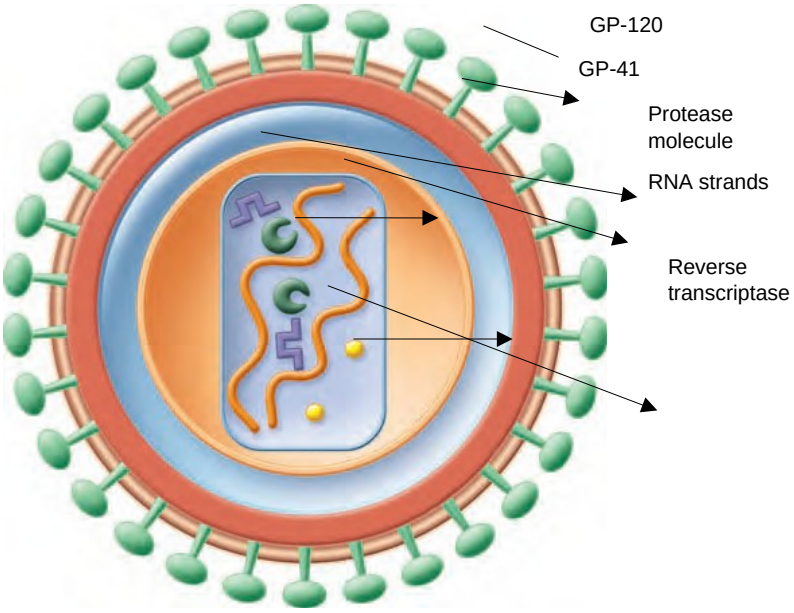

Fig 1: The general structure of HIV (Talaro, 2012)

2.3. Signs and symptoms

After an incubation period of a few days to a few weeks from exposure to HIV, most infected individuals present with an acute flu-like illness. Acute HIV infection is a very heterogeneous syndrome and individuals presenting with more severe symptoms during acute infection and a longer duration of the acute infection syndrome tend to progress more rapidly to AIDS (Breen, *et al.*, 2006). The clinical symptoms of acute HIV infection were described as an illness resembling infectious mononucleosis. The most common symptoms are fever, maculopapular rash, oral ulcers, lymphadenopathy, arthralgia, pharyngitis, malaise, weight loss, aseptic meningitis and myalgia (Hoffmann, *et al*., 2007). This phase is marked by high levels of free virus in the blood, followed by a rapid drop. Observe that the antibody levels rise at the same time that the virus load is dropping. The antibodies are responsible for neutralizing the free viruses in circulation during this stage. One feature of the ongoing HIV infection is a period of mostly asymptomatic disease (sometimes called latency) that varies in length from 2 to 15 years,

with the average being about 10 years. As the number of T helper cells declines, so too does the efficiency of the macrophage response and the ability to produce antibodies. Once the CD4 cell levels fall below 200 cells/mm 3 (μl) of blood, symptoms of AIDS appear (Talaro , 2012).

2.4. Pathogenesis and Virulence Factors of HIV

HIV enters a mucous membrane or the skin and is phagocytosed by a dendritic cell. In the dendritic cell, the virus grows and is shed from the cell without killing it. New viruses are taken up and amplified by macrophages in the skin, lymph organs, bone marrow, and blood. One of the great ironies of HIV is that it infects and destroys many of the very cells needed to combat it especially the helper (CD4 or T4) class of lymphocytes. It also infects monocytes, dendritic cells, and macrophages. Once the virus is inside a target cell, its reverse transcriptase converts it's RNA into DNA. Although initially many viruses produce a lytic infection, in some cells the DNA becomes inactive in the nucleus of the host cell and its DNA becomes integrated into host DNA, this event accounts for the lengthy course of the disease (Talaro, 2012). Because different host cells are in different stages of infection, some host cells are releasing new viruses and being lysed, and new T cells are constantly being infected. In the absence of treatment, the host cells ultimately lose this race for survival. The primary effects due directly to HIV infection are extreme leukopenia, with lowered levels of lymphocytes in particular. Both T cells and monocytes undergo extensive die-offs through programmed cell suicide (apoptosis). The CD4 memory clones and stem cells are among the prime targets. The viruses also cause formation of giant T cells and other syncytia, which allow the spread of viruses directly from cell to cell, followed by mass destruction of the syncytia. The central nervous system is affected when infected macrophages cross the blood-brain barrier and spread viruses into brain cells. Studies have indicated that some of the viral envelope proteins can have a direct toxic effect on the brain's glial cells and other cells. Other research has shown that some peripheral nerves become demyelinated and the brain becomes inflamed (Talaro , 2012).

2.5. Diagnosis of HIV Infection

Most viral testing is based on detection of antibodies specific to the virus in serum or other fluids, which allows for the rapid, inexpensive screening of large numbers of samples. Testing usually proceeds at two levels. The initial screening tests include the older ELISA (Enzyme Linked Immuno Sorbent Assay) and newer latex agglutination and rapid antibody tests (Talaro, *et al.,* 2012). This latter approach is by no means inferior to confirmation by Western blot Assays , is a methodology for which HIV is propagated in cell cultures, harvested, purified and denatured (i.e. split into its constituents). The resulting viral proteins are separated according to their molecular weight by electrophoresis and blotted onto a nitrocellulose membrane which is then cut into strips. To perform the test, the membrane is incubated with patient serum. If this contains antibodies against the various viral proteins, they will bind to the areas on the strip onto which the respective antigens have been blotted. This antigen-antibody reaction is revealed using an enzyme-labeled secondary antibody and matching substrate, whereupon the so-called "bands" appear on the test strip. (Hoffmann, *et al.*, 2007), quantitative detection of virus has become very important; the concentration of viral RNA in plasma, the so-called "viral load", has become an indispensable tool for guiding antiretroviral therapy (Ferrara et *al.*, 2006).

2.6. Treating HIV Infection and AIDS

It must be forcefully stated: There is no cure for HIV infection or AIDS. None of the therapies does more than slow the progress of the disease or diminish symptoms (Talaro, 2012).

2.7. Tuberculosis

Tuberculosis (TB) is a contagious disease caused by *Mycobacterium tuberculosis*. In fact, over 33% of the world’s population currently is infected with the tubercle bacillus and TB is killing more people every

year; in 2009 someone was dying of TB every 15 seconds (Pommerville and Jeffrey 2011).

Tuberculosis (TB) is an ancient disease. Scientists analyzing spinal column fragments from Egyptian mummies more than 4,400 years old have found pathological signs of tuberculosis. Hippocrates, more than 2,000 years ago described a widespread illness that he called “phthisis,” which was probably TB. Over the centuries, TB has continued to be a “slate wiper” in the human population. During the first half of the 20th century, TB was called “consumption” or “white plague” and it continued to be the world’s leading cause of death from all causes, accounting for one fatality in every seven cases. Tuberculosis is caused by Mycobacterium tuberculosis, the “tubercle” bacillus first isolated by Robert Koch in 1882. It is a small, aerobic, nonmotile rod whose cell wall forms a waxy cell surface that greatly enhances resistance to drying, chemical disinfectants, and many antibiotics (Pommerville and Jeffrey 2011).

2.8. Pathogenicity of Mycobacteria

There are marked differences in the ability of different mycobacteria to cause lesions in various host species. Humans and guinea pigs are highly susceptible to M tuberculosis infection, whereas fowl and cattle are resistant. M tuberculosis and Mycobacterium bovis are equally pathogenic for humans. The route of infection (respiratory versus intestinal) determines the pattern of lesions. In developed countries, M bovis has become very rare. Some "atypical" mycobacteria, now designated as nontuberculous (eg, Mycobacterium kansasii) produce human disease indistinguishable from tuberculosis; others (eg, Mycobacterium fortuitum) cause only surface lesions or act as opportunists (Brooks, *et al*,. 2010)

2.9 Primary Infection & Reactivation Types of Tuberculosis

When a host has first contact with tubercle bacilli, the following features are usually observed: (1) An acute exudative lesion develops and rapidly spreads to the lymphatics and regional lymph nodes. The exudative lesion in tissue often heals rapidly. (2) The lymph node undergoes massive caseation, which usually calcifies (Ghon lesion). (3) The tuberculin test becomes positive. This primary infection type occurred in the past, usually in childhood, but now frequently in adults who have remained free from infection and therefore tuberculin-negative in early life. In primary infections, the involvement may be in any part of the lung but is most often at the base. The reactivation type is usually caused by tubercle bacilli that have survived in the primary lesion. Reactivation tuberculosis is characterized by chronic tissue lesions, the formation of tubercles, caseation, and fibrosis. Regional lymph nodes are only slightly involved, and they do not caseate. The reactivation type almost always begins at the apex of the lung, where the oxygen tension (PO2) is highest (Brooks, et al,. 2010).

These differences between primary infection and reinfection or reactivation are attributed to resistance and hypersensitivity induced by the first infection. It is not clear to what extent each of these components participates in the modified response in reactivation tuberculosis (Brooks, *et al*,. 2010)

2.10. Clinical Findings

Since the tubercle bacillus can involve every organ system, its clinical manifestations are protean. Fatigue, weakness, weight loss, fever, and night sweats may be signs of tuberculous disease. Pulmonary involvement giving rise to chronic cough and spitting of blood usually

is associated with far-advanced lesions. Meningitis or urinary tract involvement can occur in the absence of other signs of tuberculosis. Bloodstream dissemination leads to miliary tuberculosis with lesions in many organs and a high mortality rate (Brooks, *et al*,. 2010).

2.11. Epidemiology and Transmission of Tuberculosis

Tuberculosis is primarily an airborne disease and, as such, the bacilli are transmitted from person to person in small, aerosolized droplets when a person with active pulmonary disease sneezes, coughs, spits, or even sings. The infectious dose is quite small and the inhalation of even a single *M. tuberculosis* cell can lead to a new infection. However, individuals with prolonged, frequent, or intense contact with a diseased individual are at most risk of becoming infected, with an estimated 30% infection rate. Thus, crowded conditions and poor ventilation often contribute to disease spread and people who live in overcrowded, urban ghettoes often contract TB. Malnutrition and a generally poor quality of life also contribute to the establishment of disease (Pommerville and Jeffrey 2011).

2.12. Diagnosis of Tuberculosis

Clinical diagnosis of tuberculosis traditionally includes some combination of these techniques: tuberculin or immunologic testing, roentgenography (X rays), direct identification of acid-fast bacilli (AFB) in sputum or some other specimen, and cultural isolation and identification. Final diagnosis of overt or latent TB cannot be made on a single test alone but requires an overall medical evaluation (Talaro , 2012).

2.13. Tuberculin Sensitivity and Testing

Because infection with the TB bacillus can lead to delayed hypersensitivity to tuberculoproteins, testing for hypersensitivity has been an important way to screen populations for tuberculosis infection.

The tuberculin test, called the Mantoux test, involves local injection of purified protein derivative (PPD), a standardized solution taken from culture fluids of *M. tuberculosis*. The injection is done intradermally into the forearm to produce an immediate small bleb. After 48 and 72 hours, the site is observed for a red wheal called an induration, which is measured and classified according to size The current practices for interpreting tuberculin tests are focused on selected groups known to have higher risk for tuberculosis. It is no longer a routine screening method among populations of children or adults who are not within the target groups. The reasoning behind this change is to allow more focused screening and to reduce expensive and unnecessary follow-up tests and treatments. Guidelines for test groups and methods of interpreting tests are listed in the following summary (Talaro , 2012).

2.14. Treatment

Tuberculosis is an extremely stubborn disease especially with the development of antibiotic resistance. TB has been traditionally treated with such first-line drugs as isoniazid and rifampin. Ethambutol, pyrazinamide, and streptomycin also are used to help delay the emergence of resistant strains. Unfortunately, the appearance of multidrug-resistant tuberculosis (MDR-TB) has occured and now accounts for 5% of new TB cases. This has necessitated a switch to a group of second-line drugs, including fluoroquinolones and kanamycin. If drug therapy is effective for pulmonary TB, patients usually become noninfectious within three weeks as determined by bacteriafree sputum samples. Still, for such individuals, antimicrobial drug therapy is intensive and must be extended over a period of six to nine months or more (Pommerville and Jeffrey 2011).

2.15. Prevention

Vaccination against TB cansometimes be rendered by intradermal injections of an attenuated strain of *Mycobacterium bovis,* a species

that causes tuberculosis in cows as well as humans. The weakened strain is called **Bacille Calmette-Guérin (BCG),** after Albert Calmette and Camille Guérin, the two French investigators who developed it in the 1920s. Though the vaccine is used in parts of the world where the disease causes significant mortality and morbidity, health officials in the United States generally do not recommend the BCG vaccine because it has limited effectiveness for preventing TB in adults and is only moderately effective in children for 10 years. New vaccines consisting of subunits, molecules of DNA, and attenuated strains of mycobacteria are currently being developed (Pommerville and Jeffrey 2011).

2.16. Preventing TB among HIV-infected individuals

The World Health Organization WHO currently recommends that all HIV-infected persons should be screened for TB, and HIV-infected persons without active TB disease shoud be evaluated for treatment of latent TB infection. Two meta-analyses have shown that isoniazid (INH) taken daily for six months (6H) reduces the incidence of TB by over two-thirds among HIV-infected individuals. The most widely recommended regimen for TB preventive therapy is isoniazid 300 mg daily for 6 months. WHO guidelines (2010) strongly recommend the use of 6H regimen, with 36H (3 years of isoniazid) being a conditional recommendation for countries to adopt depending on local needs and resources. However, very few high-burden TB countries have routinely implemented isoniazid preventive therapy (IPT) for PLWH, because of concerns about how to exclude TB disease, fears about selection for INH-resistant *M. tuberculosis* (MTB) strains, and the absence of public health models for how to deliver this treatment (Padmapriyadarsini *et al*,.2011). Symptom screening can detect culture-confirmed TB disease with greater than 90 per cent sensitivity and 97 per cent negative

predictive value. Studies from India and South Africa found the 6-month isoniazid regimen to be effective, well tolerated with low rates of emergence of drug resistance. The South African cohort study, which used three new prophylactic regimens, did not find any superiority over the control regimen of 6 months of isoniazid. In contrast, a randomized double-blind, placebo-controlled trial in Botswana found that 36 months isoniazid prophylaxis was more effective for prevention of TB than was 6-month prophylaxis, chiefly benefitting those who were tuberculin skin test positive and those initiating ART. The National AIDS Control Organization (NACO) intends to test the effectiveness and feasibility of the WHO IPT guidelines in ART clinics as a precursor for adopting this recommendation (Padmapriyadarsini *et al*,.2011).

3. Materials and methods

3.1. Study design

Cross sectional study design, hospital clinic based study.

3.2 Study area and duration

This study was conducted in the Abo Anga Hospital from March to September 2014.

3.3. Study population

This study targeted tuberculosis patients.

3.4. Sample size and sampling technique

Ninety blood samples were collected as randomized sampling from TB patients.

3.5. Ethical considerations

Permission from patient was taken and the verbal informed consent was taken

3.6. Specimen collection

Blood collected by venipuncture from TB patients 3ml in plan container was allowed to clot naturally and completely – the serum was separated from the clot as early as possible to avoid haemolysis of the RBCs, care was taken to ensure that the serum specimens are clear and not contaminated by microorganism with work in safety cabinet level two and transportation occurred as soon as possible and storage of specimens in - 20º C.

3.7. Data collection

Data was collected through verbal questionnaire.

3.8. Data analysis

Data was analyzed by using SPSS 14 for windows.

3.9. Laboratory investigation

Enzyme Linked Immune Sorbent Assay (ELISA) was used to detect antibodies of HIV 1+2.

3.10. Principle

ELISA is rapid test used for detecting antibodies to HIV 1/2, is a two steps incubation antigen “sandwich” enzyme assay, which used polystyrene microwell stripe pre-coated with recombinant

HIV antigens expressed in *E. coli* (recombinant HIV-1gp41, gp120 and recombinant HIV-2 gp-36). Patient's serum sample is added, and during the first incubation step, the specific HIV1/2 antibody will be captured inside the wells if present. The microwells then washed to remove unbound serum proteins. A second set of recombinant antigens conjugated to the enzyme Horseredish Peroxidase (HRP-Conjugate) and expressing the same epitopes as the pre-coated antigens is added, and during the second incubation, they will bind to captured antibody. The microwells washed to remove unbound conjugate, and chromogen solutions are added into the wells. In the wells containing the antigen-antibody-antigen (HRP) "sandwich" immunocomplex, the colorless chromogens are hydrolyzed by the bound HRP conjugate to a blue colored product. The blue color turns yellow after the reaction is stopped with sulfuric acid. The amount of color intensity can be measured and it is proportional to the amount of captured in the wells, and to the sample respectively. Wells containing samples negative for anti- HIV 1/2 remain are colorless.

3.11. Procedure

Reagent preparation: reagent was allowed to reach room temperature (18-30°C). Wash buffer was checked to concentrate for presence of salt crystals.

Step1 preparation: three wells were marked as Negative control, two wells as positive control and one blank (neither sample nor HRP- Conjugate Should be added into the blank well).

Step2 Adding Sample: 100µl was added of positive control, negative control and specimen into their respective wells except the blank well.

Step3 (Incubating): Plate was covered with plate cover and incubated for 30 minutes at 37° C.

Step4 (Washing): At the end of incubation plate cover was removed. Each well was washed 5 by washing machine times

with diluted washing buffer. Each time allowed the microwells to soak 30-60 seconds. After the final washing cycle, plate was turned down onto blotting paper and taped to remove any remainders.

Step5 Adding HRP-Conjugate: (Horseradish Peroxidase) 100 µl were added into each well except the blank.

Step6 (Incubating): Plate was covered with plate cover and incubated for 30 minutes at 37° C

Step7 Washing: At the end of incubation plate cover was removed. Each well was washed 5 times with diluted washing buffer. Each time allowed the microwells to soak 30-60 seconds. After the final washing cycle, plate was turned down onto blotting paper and taped to remove any remainders.

Step8 Coloring: 50µl of chromogen A (Urea Peroxide) were added and 50µl of chromogen B (Tetramethyl Benzidine, TNB) solution into each well including the blank. Plate was incubated at 37°C for 15 minutes avoiding light. The enzymatic reaction between the Chromogen solutions and the HRP-Conjugate produced blue color in Positive control and anti-HIV ½ positive sample wells.

Step9 Stopping Reaction: with multichannel pipette was used to add 50µl of Stop solution was added into each well and mix gently. Intensive yellow color was developed immediately in positive control and anti-HIV ½ positive sample wells.

Step10 (Measuring the Absorbance): Plate were calibrated with the blank well and the absorbance was read at 450nm.

3.12. Calculation

Calculation the cut- off value (C.O,) =Nc+ 0.12(Nc = the mean absorbance value for three negative controls).

- The A value of the blank, which contains only chromogen and stop solution was < 0.080 at 450 nm.
- The A value of the positive control ≥ 0.800 at 450 nm after blanking.

- The A value of the Negative control < 0.100 at 450 nm after blanking.

3.13. Interpretations of the results

Negative Results (A/C.O. <1): specimens giving absorbance less than the cut-off value was negative for this assay, which indicates that, no anti-HIV 1/2 antibodies have been detected

Positive Results (A/C.O. ≥ 1): specimens giving an absorbance equal to or greater than the cut-off value are considered initially reactive, which indicates that anti-HIV 1/2 antibodies have probably been detected.

Borderline (A/C.O. = 0.9-1.1): specimens with an absorbance to cut-off value ratio between 0.9-1.1 were considered borderline

4. Results

Out of 90 Tuberculosis patients in this study 74 were male (82%) and 16 were female (18%) (Fig- 2)

Out of ninety TB samples tested 13(14%) were HIV (1+2) positive and 77(86%) were HIV negative (Fig - 3)

According to gender out of the 13 positive patients 8 were males (62) and 5 females (38), (Fig- 4)

According to TB and HIV (1+2) infections and the age group, the most age group effected with TB and HIV was 20 to 30 years (Figure 5)

According to HIV infection and the duration of TB drugs there was no significant effect (P>0.05) on distribution of HIV infection (Table 1)

4.

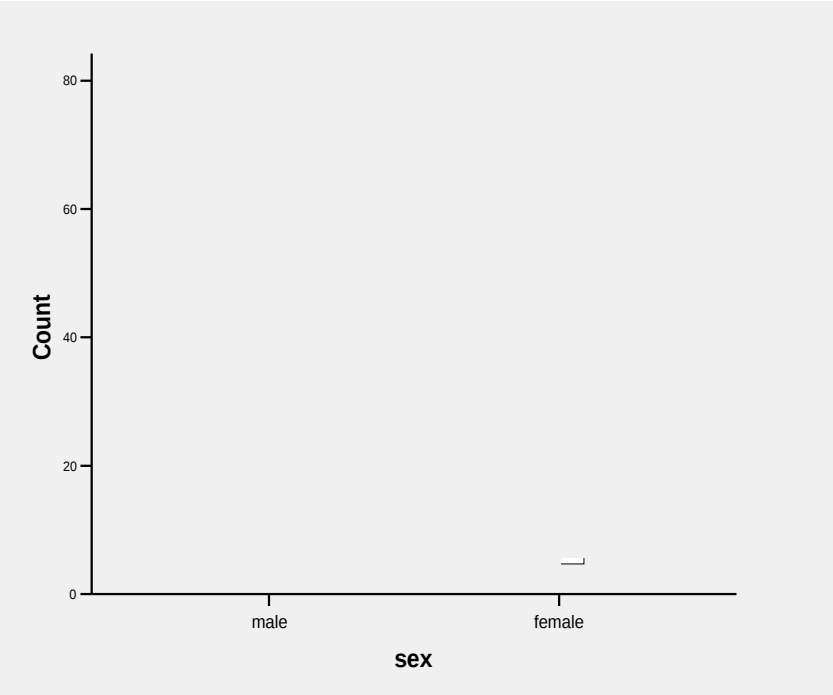

Fig 2: Distribution of Tuberculosis patients according to gender

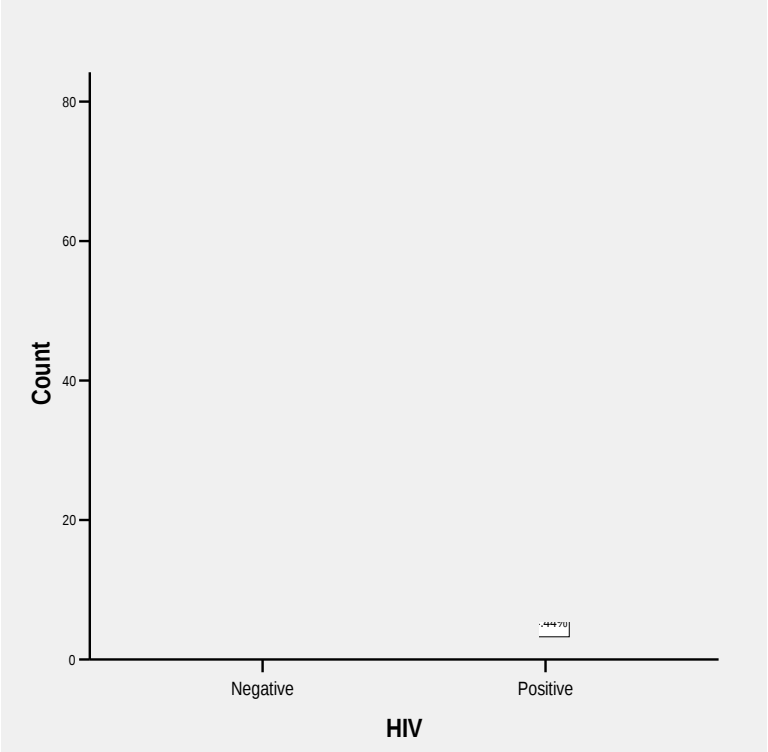

Fig 3: Distribution of HIV (1+2) in Tuberculosis patients

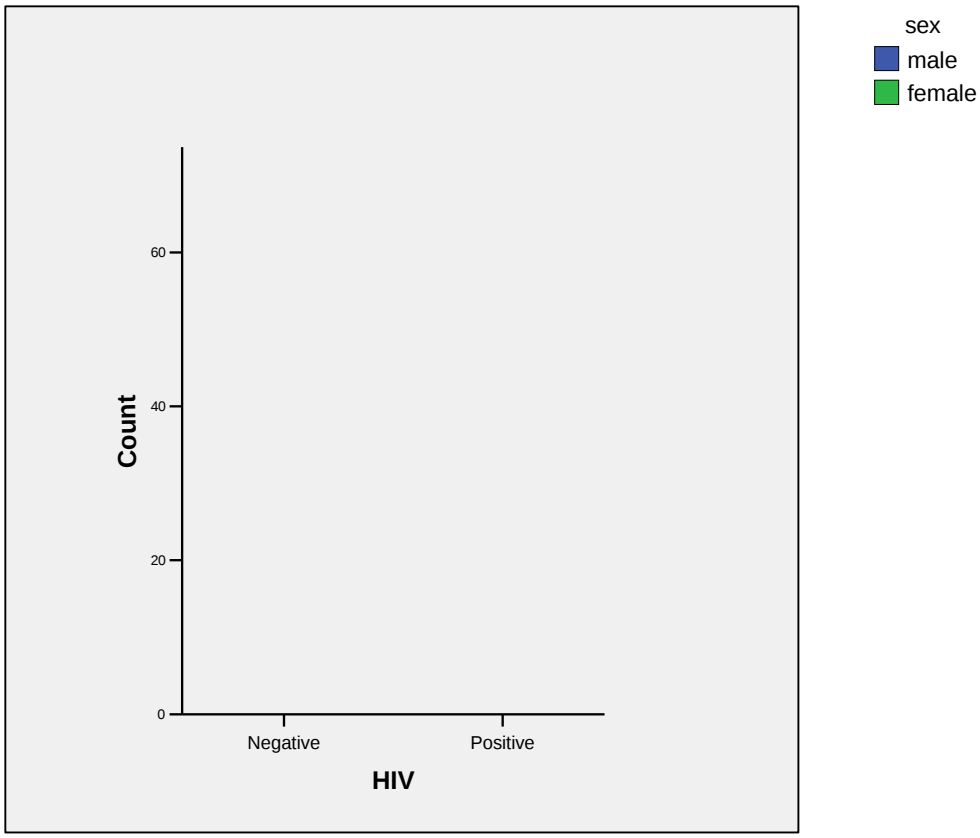

Fig 4: Distribution of HIV (1+2) infection according to gender

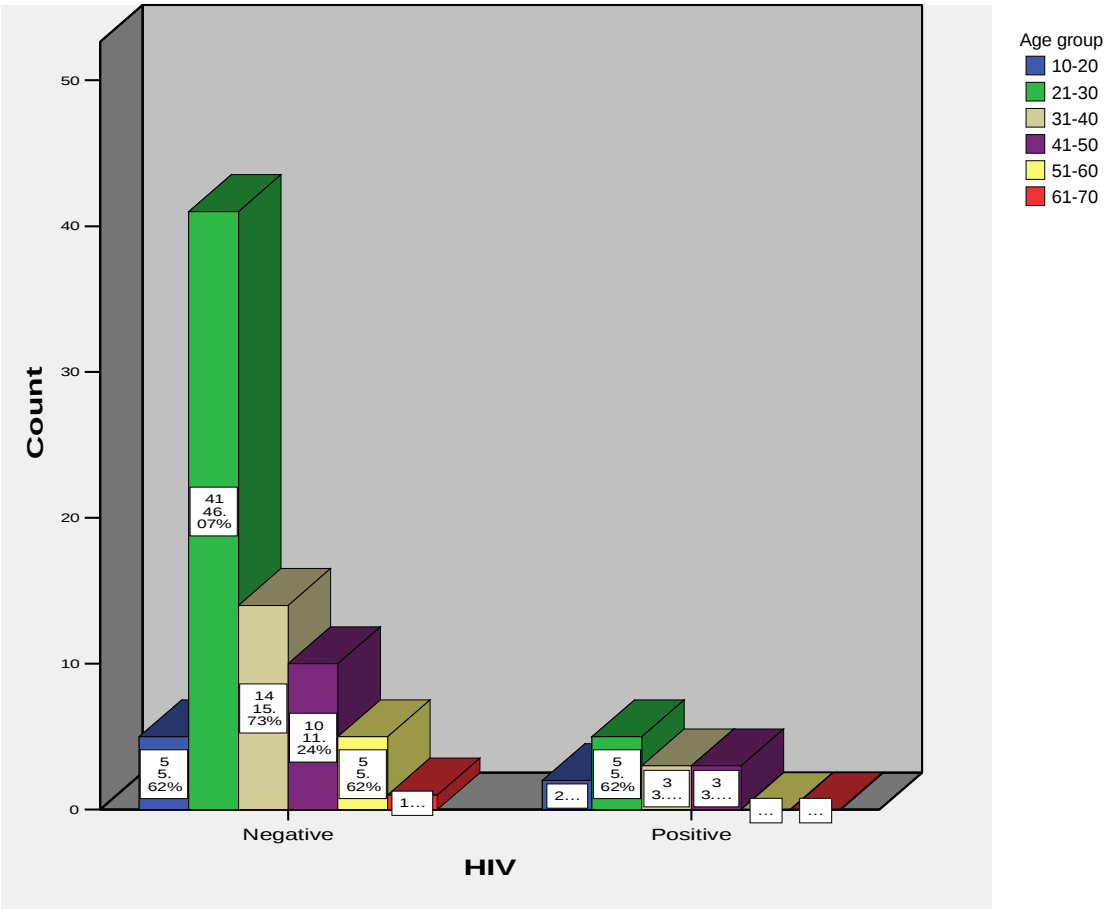

Fig 5: Distribution of HIV infection according to age group

Table 1: Distribution of HIV (1+2) infection according to duration of TB drugs

Duration of treatment/month * HIV (1+2) Cross tabulation

Count

		HIV(1+2)		Total
		Negative	Positive	
Duration of drug/month	.0	8	2	10
	1.0	10	0	10
	2.0	15	2	17
	3.0	5	4	9
	4.0	5	1	6
	5.0	11	0	11
	6.0	17	2	19
	7.0	2	1	3
	8.0	1	0	1
	9.0	1	0	1
	10.0	1	0	1
	12.0	1	0	1
	24.0	0	1	1
Total		77	13	90

P>0.05

5. Discussion

5.1. Discussion

TB and HIV co infection is well recognized as a major public health problem in worldwide and many investigations have been performed and published from western counties, The United Nations joint program on HIV/ADIS 2009 report estimated that 33.4 million people are living with HIV and one third of them are co infected with TB. Screening of HIV among TB population should be attached more importance in Sudan which would be much helpful for treatment of both diseases, 14% were HIV seropositive among TB patients.

In this study the result is greater than that obtained by Getahun, *et al* 2010 in Zambia and Harries *et al.,* 2012 in South African who reported only 4% and 10% respectively and Dye. *et al* 1999 in India 3% of the estimated cases of HIV related TB, and lower than that obtained by Howard and El-Sadr,.2010 in sub-Saharan Africa which represent 38% of HIV seropositive and lower than that obtained by Wells *et al.,* 2007, in Kenya 26%, Uganda 49%, in Ethiopia 50%, which it's high prevalence of HIV infection in TB patients and greater than that obtained by Sharma *et al.,* 2005 in India which found 20% HIV seropositive, and higher than study obtained by Corbett *et al.,* 2003 in South African was 5% .

This result agreed with the study obtained by Swaminathan *et al*., 2010 in India which found 14.8% HIV positive and by

Wells *et al.,* 2007 in Eritrea which found 15% HIV/ TB co-infection.

The distribution of TB patients according to gender in our study that showed 74 was males (82%) and 16 was females (18%) and the distribution of HIV infection according to gender 8 males (62%) and 5 females (38%) and the results is greatest than that obtained by Perriens *et al*., 1995 in Zaire that showed (40%) male infected with HIV.

Distribution of HIV infection according to duration of TB treatment showed no significant effect (P>0.05) on distribution of HIV and duration of TB and the treatment and the result disagree with study by Gandahi *et al.,* 2009 that showed 85% MDR TB patient in related HIV .

5.2. Conclusion

The study addressed the status of TB and HIV co-infection in Khartoum State, Sudan. Ninety tuberculosis patients were screened to HIV, the results that showed 14% HIV seropositive among TB patients. Youth of age group 20-30 years are most affected. No significant difference of TB drug on the distribution of HIV infection.

5.3. Recommendations

1. HIV screening test should be introduced as routine screening to all new TB cases
2. Use of large sample size from different hospitals and employing the confirmation techniques such as real-time PCR will reflect the real situation of HIV infected TB patients.

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