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Research Article

Assessment of Lipid Profile on Patients with Pulmonary Tuberculosis

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Abstract

Tuberculosis is one of the first most common causes of deaths in the world alongside HIV/AIDS, causing more than 9.6 million new cases and 1.5 million deaths globally in 2014 alone. This infection also accompanied by lipid profile alterations. The lipid alteration related to the disease is not yet well determined and has variation in different studies. The present study was conducted with the objective to find out lipid profile in newly diagnosed tuberculosis patients. This study aimed at evaluating the lipid profiles in pulmonary tuberculosis patients. A total of eighty (80) subjects were recruited for this study which consists of forty (40) patients with pulmonary tuberculosis out of which twenty (20) were new cases, ten (10) two months patients with pulmonary tuberculosis therapy and ten (10) six months patients with pulmonary tuberculosis therapy and forty (40) apparently healthy individuals which served as control. Collected bloods were tested using Mindray chemistry analyzer lipid profiles. The mean serum levels of triglyceride, total cholesterol, high density lipoprotein and low-density lipoprotein were significantly lower than their respective control groups ($p = 0.001$). Pulmonary Tuberculosis patients in this study had lipid profile abnormalities. Total Cholesterol, HDL, LDL and TG concentrations were significantly reduced as compared with control groups. The factors associated with Lipid profiles also due attention to prevent further complication. This study concludes that parameters of lipid profile were deranged in our tuberculous cases. To achieve maximum response of TB treatment we must consider normalizing lipid profile along with standard treatment for better recovery.

Keywords: Pulmonary; Tuberculosis; *Mycobacterium tuberculosis;* Total Cholesterol; HDL; LDL and TG

Introduction

Pulmonary tuberculosis (TB) is an infection of the lung and occasionally surrounding structures caused by the bacterium *Mycobacterium tuberculosis. Mycobacterium tuberculosis* complex (MTB) is a potentially fatal, contagious disease which still remains a major global health care issue [1]. Pulmonary tuberculosis is one of the oldest diseases, afflicting the human race since ancient times. It is caused by microorganism known as *Mycobacterium tuberculosis* (MTB). *Mycobacterium tuberculosis* which is also known as tubercle bacillus is a pathogenic bacteria species of the family Mycobacteriaceae. It forms a localized infection in the lungs after inhalation [2]. Infection with *Mycobacterium tuberculosis* does not necessarily lead to active disease as the immune response of most individuals can successfully contain it, but not eliminate the infection. It is mainly a lung disease where it forms localized infection after inhalation. Tuberculosis is a contagious disease that progresses from a systemic infection. Most commonly, *M. tuberculosis* is spread from person to person mainly by airborne transmission of droplet nuclei. TB can however affect any part of the body. Despite a relatively low transmission rate compared with other contagious diseases and the existence of effective chemotherapy for six decades, tuberculosis remains a major global public health problem with approximately one-third of the world's population is infected with tuberculosis with the majority of these individuals living in less developed countries [3].

Lipids, commonly known as fats are heterogeneous group of organic solvent but insoluble in water [4]. Lipid profile is a panel of blood tests that serves as an initial broad medical screening tool for abnormalities in lipids, such as cholesterol and triglycerides. The results of this test can identify certain genetic diseases and can determine approximate risks for cardiovascular disease, certain forms of pancreatitis, and other diseases [5]. Cholesterol regulates hormones in the body and basic cellular metabolism. LDL and HDL transport cholesterol back and forth between tissues and liver [6]. Cholesterol is used to make fluidic of cell membrane structure, participating in the activity of enzymes and in the functions of phagocytosis and cell growth [7,8]. Accumulation of cholesterol in the blood vessels narrows the blood tube and causes atherosclerosis [9]. Lipoproteins (LP), i.e. protein-lipid complexes, serve as carriers for lipid compounds in the body and are highly diversified with respect to the size, composition and density [10]. The largest lipoproteins are chylomicrons, generated in the intestinal epithelium after meals and discharged into the bloodstream [11].

Elevated cholesterol has a beneficial effect on tuberculosis resistance through the immune system. They cite the well-known fact that cholesterol constitutes about one-third of the cell membrane lipids content and participates in the fluidity of this structure, in the activity of membrane-bound enzymes and in membrane functions, such as phagocytosis and cell growth. The aim of this study was to assess the lipid profile of patients with pulmonary tuberculosis.

Materials and Methods

This study was carried out in in Central Hospital Benin City, Edo State, Nigeria. The study was conducted in Edo South Senatorial District which is one of the three senatorial districts in Edo State, Nigeria. Geographically, Edo South is located approximately between latitude 05°44N and 06° 87'N and longitudes 05° 00'E and 06°' 43E of the equator [12]. Politically, Edo South is divided into seven local government areas namely: Oredo, Orhionwon, Egor, Ovia North-East, Ikpoba-Okha, Ovia South-West and Uhunmwode respectively. Edo South Senatorial District constitutes 57.1 percent of the population in Edo State and virtually, all the groups traced their origin to Benin City; hence the dialects of the groups vary with their distance from Benin City. It is bounded in the North by Ondo State, North-East by three local government areas (Owan West, Esan West and Igueben) and the South by Delta State. Benin City which is the capital of Edo State encompasses three of these Local Government Areas. The total land area of the senatorial district is 10,835.37 km [13]. The inhabitants of Edo South have two major occupations (agricultural production and large/medium scale trading). They are mostly traders, civil servants, farmers and business men/women. The district has 553 health facilities which is made up of both public and private hospitals and further grouped into primary, secondary and tertiary health facilities. Oredo LGA has the highest number of health facilities with 31%, Ikpoba-Okha has 22% while Egor equally shares 19% of health facilities. Coincidentally, these local government areas also have the highest rate of maternal mortality in Edo South [14]. The following areas

Orhionmwo, Ovia North-East, Ovia South-West and Uhunmwode has a total share of 28% of health facilities [15,16]. However, the major target area for this study is Egor Local Government Area. The samples were examined in the Research Diagnostic Laboratory, of the department of Medical Laboratory Science, College of Medicine, Ambrose Alli University, Ekpoma.

Study population

A total of eighty (80) subjects were recruited for this study which consists of forty (40) patients with pulmonary tuberculosis out of which twenty (20) were old cases and twenty (20) were new cases and forty (40) apparently healthy individuals which served as control.

Research design

This research was carried out within three months and its specifically designed to assess the lipid profile of patients with pulmonary tuberculosis and make comparison with apparently healthy individuals. Blood samples were collected from patients with pulmonary tuberculosis in Central Hospital Benin City, Edo State, Nigeria, Edo State. The blood samples were centrifuged and serum was immediately separated from the cells into plain containers with label corresponding to initial blood sample bottle. The serum samples were stored frozen until the time for analysis. The serum obtained was analyzed using standard procedures described by the manufacturer.

Inclusion and exclusion criteria

Diagnosed tuberculosis patients who are the test subjects and apparently healthy individuals who serve as the control groups. Subjects and control whose ages are between 18-65 years old were included after their consents approved. Subjects on lipid lowering or rising medications like contraceptive, Diabetes mellitus and Hypertension were excluded from the study.

Ethical approval

Ethical permission was obtained from the Ambrose Alli University ethical committee and informed consent was obtained from subjects used for this research project after thorough explanation of the aim and benefits of the study.

Sample collection

BBlood samples (5mls) were collected by vene-puncture into an accurately labelled plain container for both tuberculosis patients and control group (apparently healthy individuals with no obvious sign or symptoms of illness). The blood samples were centrifuged with a laboratory centrifuge at 4000rpm for 10minutes at room temperature within two hours of collection and the serum separated into a clean plain container which are labelled corresponding to the initial blood samples containers. Analysis was carried out for triglyceride, total cholesterol, high density lipoprotein (HDL), low density lipoprotein (LDL) and very low-density lipoprotein (VLDL).

Sample Analysis/Methodology

Estimation of total cholesterol

Enzymatic endpoint (CHOD-PAP) method was used to estimate the total cholesterol as described by Richmond, [17].

Procedure

Ten microliters of distilled water, standard and sample were dispensed into test tubes labelled blank, standard, and sample respectively. One milliliter of cholesterol reagent was added into the respective tubes, and the contents mixed and incubated at 37^0C for 5minutes. The absorbance of standard and samples were measured against blank at the wavelength of 500nm using spectrophotometer.

$$\text{TC conc. (mmol/dl)} = \frac{\text{Abs of test}}{\text{Abs of Std.}} \times \frac{\text{conc. of Std. (5.25mmol/l)}}{1}$$

Estimation of triglycerides

Colorimetric method described by Trinder, [18] was employed here.

Procedure

Ten microliters of distilled water, standard and sample were dispensed into test tubes labelled Blank, Standard and Samples respectively. One milliliter of triglyceride reagent was added into the respective test tubes; the contents were mixed and incubated in the water bath at 37^0C for 5minutes. The absorbance of standard and samples were measured against the blank at a wavelength of 500nm using spectrophotometer.

$$\text{TG conc. (mmol/dl)} = \frac{\text{Abs of test}}{\text{Abs of Std}} \times \frac{\text{conc. of Std. (2.21 mmol/l)}}{1}$$

Estimation of high-density lipoprotein - cholesterol

Precipitation method described by Lopes-Virella., *et al.* [19] was employed here.

Procedure

- **Stage 1:** Two hundred microliters of standard/sample was dispensed into test tube labelled standard/samples. Five hundred microliters of precipitant was added into the test tubes and mixed well, the content was allowed to stand for 10minutes at room temperature and then centrifuged for 10 minutes at 4000rpm, the supernatant was separated and the cholesterol content was estimated using CHOD - PAP method.
- **Stage 2:** One hundred microliters of distilled water, standard, supernatant and sample supernatant were added into test tubes labeled Blank, Standard and Samples respectively. One milliliter of cholesterol reagent was added into the respective test tubes; the contents was mixed and incubated at 37^0C for 5minutes. The absorbances of the standard and the samples were measured against the Blank at the wavelength of 500nm using spectrophotometer.

$$\text{HDL-C conc. (mmol/dl)} = \frac{\text{Abs of Test}}{\text{Abs of Std.}} \times \text{conc. of Std. (5.25 mmol/l)}$$

Estimation of low-density lipoprotein-cholesterol (LDL)

Friedewald formula was employed here as described by Friedewald, Levy, and Fredrickson, [20].

LDL- cholesterol (mmol/l) = Total cholesterol - (TG/ 2.2+ HDL-cholesterol).

Estimation of very low-density lipoprotein-cholesterol (VLDL)

The formula below was employed as described by Burtis and Ashwood, [21].

VLDL-C = Triglyceride / 5

Statistical analysis

The result was presented using tables. Data was analyzed using the SPSS software. The percentage prevalence was calculated in each case. Comparative analysis of the result was done using Chi-square statistical software and $p<0.05$ was considered statistically significant. Differences in means were analyzed using Student's *t* test. The relationship between outcome measures were estimated using Pearson's correlation coefficient.

Results

Table 1 shows the differences in the total cholesterol levels among pulmonary tuberculosis patient (154.80 ± 24.32) and control group (97.17 ± 8.61) indicated a significance difference among groups, P< .000. Differences in HDL levels among pulmonary tuberculosis patient (65.17 ± 12.99) and control group (40.37 ± 8.43) indicated a significance difference among group, P< .000. Differences in LDL among pulmonary tuberculosis patient (66.27 ± 18.45) and control group (39.13 ± 5.67) indicated a significance difference among group; P< .000. Also, differences in triglyceride among pulmonary tuberculosis patient (123.73 ± 18.84) and control group (91.30 ± 7.01) indicated a significance difference among group, P< .000.

Table 2 result on the gender and total cholesterol among control revealed that there was no significant difference between male (154.76 ± 23.86) and female (154.81 ± 25.50); P>.005, among patients with pulmonary tuberculosis there was no significant difference between male (94.14 ± 6.93) and female (99.81 ± 9.26); P>.005. Gender and triglyceride among control group revealed that there was no difference between male (125.50 ± 24.28) and female (122.19 ± 13.03); P>.005. Among patients with pulmonary tuberculosis there was no difference between male (95.36 ± 6.61) and female (87.75 ± 5.33) on triglyceride; P>.005. Furthermore, Gender and HDL among control group revealed that there was no significant difference between male (60.00 ± 12.03) and female (69.68 ± 12.43); P>.005. Among patients with pulmonary tuberculosis there was no difference between male (34.18 ± 4.41) and female (47.42 ± 5.94) on triglyceride; P>.005. Finally, gender and LDL among control groups revealed that there was no significant difference between male (73.78 ± 18.49) and female (59.68 ± 16.20); P>.005. while gender and LDL among patients with pulmonary tuberculosis revealed that there was no significant

difference between male (42.00 ± 5.57) and female (35.85 ± 3.79), P<.05.

Result in table 3 in the new cases of pulmonary tuberculosis revealed that there was a significant difference in the total cholesterol among control group (154.80 ± 24.32), new cases (97.17 ± 8.16), 2 months (114.93 ± 8.47) and 6 months (129.79 ± 10.68); P< .005. There was also a significant difference in the triglyceride among control group (123.73 ± 18.84), new cases (91.30 ± 7.01), 2 months (101.93 ± 13.13) and 6 months (106.83 ± 8.68); P< .005. Also, there was a significant difference in HDL among control group (65.17 ± 12.99), new cases (40.37 ± 8.43), 2 months (45.16 ± 10.50) and 6 months (48.89 ± 7.27); P< .005. Finally, there

Serum Levels	Group	X	t-value`	p
TCHOL	PTP	154.80 ± 24.32	12.795	.000
	Control	97.17 ± 8.61		
HDL	PTP	65.17 ± 12.99	10.030	.000
	Control	40.37 ± 8.43		
LDL	PTP	66.27 ± 18.45	4.563	.000
	Control	39.13 ± 5.67		
TG	PTP	123.73 ± 18.84	9.047	.000
	Control	91.30 ± 7.01		

Table 1: Significant differences in the serum levels of triglyceride, total cholesterol, HDL, LDL and VLDL of patients with pulmonary tuberculosis and the control group.

Key: PTB - Pulmonary Tuberculosis Patient, TCHOL - Total Cholesterol, HDL - High Density Lipoprotein, LDL - Low Density Lipoprotein, TG – Triglyceride. N – Sample size, PTB (N = 30), TCHOL (N = 30), HDL (N = 30), LDL (N = 30), TG (N = 30)

*Values are significant at p<0.05.

Serum Levels	Group	Gender	X	Df	t-value`	P
	Control (n = 40)	Male	154.76 ± 23.86	29	-.003	.653
TCHOL		Female	154.81 ± 25.50			
	PTP (n = 40)	Male	94.14 ± 6.93	29	-1.876	.140
		Female	99.81 ± 9.26			
	Control (n = 40)	Male	125.50 ± 24.28	29	.474	.203
TG		Female	122.19 ± 13.03			
	PTP (n = 40)	Male	95.36 ± 6.61	29	3.488	.045
		Female	87.75 ± 5.33			
	Control (n = 40)	Male	60.00 ± 12.03	29	-2.162	.803
		Female	69.68 ± 12.43			
HDL	PTP (n = 40)	Male	34.18 ± 4.41	29	-6.982	.889
		Female	47.42 ± 5.94			
	Control (n = 40)	Male	73.78 ± 18.49	29	1.146	.293
LDL		Female	59.68 ± 16.20			
	PTP (n = 40)	Male	42.00 ± 5.57	29	3.314	.050
		Female	35.85 ± 3.79			

Table 2: Relationship between sex and serum levels of triglyceride, total cholesterol, HDL, LDL and VLDL of patients with pulmonary tuberculosis when compared with the control group.

Parameters	Control	PTB New case (n = 20)	2 Months (n = 10)	6 Months (n = 10)	t-value	Sig
TCHOL	154.80 ± 24.32	97.17 ± 8.16	114.93 ± 8.47	129.79 ± 10.68	2.96	0.006
TG	123.73 ± 18.84	91.30 ± 7.01	101.93 ± 13.13	106.83 ± 8.68	4.63	0.000
HDL	65.17 ± 12.99	40.37 ± 8.43	45.16 ± 10.50	48.89 ± 7.27	10.74	0.000
LDL	66.26 ± 18.45	39.13 ± 5.67	49.50 ± 10.25	61.52 ± 9.48	-5.52	0.000

Table 3: Paired T-test showing differences in the serum levels of triglyceride, total cholesterol, HDL, LDL and VLDL of patients with pulmonary tuberculosis when compared with the according to the cases (New case, 2 months old and 6 months).

*Values are significant at p<0.05.

was significance in the LDL among control group (66.26 ± 18.45), new cases (39.13 ± 5.67), 2 months (49.50 ± 10.25) and 6 months (61.52 ± 9.48), P< .005.

Discussion

Mycobacterium tuberculosis complex is the century old organism responsible for tuberculosis. Tuberculosis is a worldwide bacterial infection affecting all ages, rich and poor [22]. Among communicable diseases, tuberculosis is the second leading cause of mortality worldwide, with two million deaths each year. Overcrowding, malnutrition, smoking, depressed immunity are the important risk factors for the disease to occur [23].

Lipids are important determinants of nutritional status of the body. Low level of lipids in the body, specifically cholesterol, has been found to increase susceptibility to various infections including TB. Around one third of worlds' population is infected with MTB, but only a small fraction of this develops active disease. Therefore, some specific condition must be there which predisposes these individuals to develop active TB disease [24]. Cholesterol constitute a major portion of the lipid content of cell mambranes. It is essential for maintaining membrane fluidity. Even the macrophages require cholesterol for their phagocytic activity like cell motility, exocytosis and endocytosis. In cholesterol deficiency, its phagocytic activity is deranged [25-29].

Total cholesterol, HDL and LDL levels were significantly higher in pulmonary tuberculosis patients as compared to the control group (apparently healthy individuals) (table 1) revealing a mean score of 154.80 with SD of 24.32 in total cholesterol, mean score of 123.73 with SD of 18.84 in Triglyceride, mean score of 65.17 with a SD of 12.99 in High density lipoprotein and mean score of 66.27 with a SD of 18.45 in Low density lipoprotein among pulmonary tuberculosis patients, while a mean score of 97.17 with a SD of 8.61 in total cholesterol, mean score of 91.30 with a SD of 7.01 in Triglyceride, mean score of 40.37 with a SD of 8.43 in High density lipoprotein and mean score of 39.13 with a SD of 5.67 in Low density lipoprotein among healthy individuals which serve as the control group. In a study conducted by Muhammad., *et al*. [30] they found that mean total cholesterol was 114.62 ± 29.81mg/dl, mean TG was 112.01 ± 13.47 mg/dl, the mean HDL was 38.07 ± 7.02 mg/dl and mean LDL in was 89.00 ± 23.49mg/dl the results are comparable to others in our study.

When the differences were compared between pulmonary tuberculosis patients and control group, the parameters studied were statistically significant (p< .000) (table 2). Low levels of total serum cholesterol and HDL have been shown in tuberculous patients. It has been hypothesized that diet fortified with cholesterol might accelerate the sputum sterilization in these patients. A close relationship has been linked between acute phase reactant and HDL cholesterol, that may explain disease in nutritionally deficient patients [31]. Several studies found that lipid levels, especially of high-density lipoproteins, were low in pulmonary TB when these were compared to the normal healthy individuals. A study included 32 cases of TB reported mean total cholesterols in TB cases was 114.41 ± 15.5 mg/dl, triglyceride 98.6 ± 22.25 mg/dl, HDL 30.37 ± 2.0 mg/dl, LDL 64.31 ± 13.0 mg/dl and VLDL was 19.72 ± 4.5 mg/dl [32]. Another study reported similar results [33]. According to Perez Guzman., *et al*. [34] low serum cholesterol might be a factor

for the development of pulmonary TB. They also found that values of about 90 mg/dL were strongly associated with mortality in those patients with miliary disease. Although very scantily investigated, these associations have been already mentioned by others.

On the relationship between sex and serum levels of triglyceride, total cholesterol, HDL and LDL of patients with pulmonary tuberculosis when compared with the control group. It was revealed on assessment of the total cholesterol of the control group between male (154.76 ± 23.86) and female (154.81 ± 25.50) subjects that there was no significant differences; P>.005, and also among the pulmonary tuberculosis patients there was no significant differences between male (94.14 ± 6.93) and female (99.81 ± 9.26); P>.005 but the total cholesterol of the females were higher than that of the male subjects in both the control and tests subjects. Although total cholesterol of the male and female control subjects were higher than that of the test subjects male and female. On comparison between gender and triglyceride among control group revealed that there was no difference between male (125.50 ± 24.28) and female (122.19 ± 13.03); P>.005. Among patients with pulmonary tuberculosis there was no difference between male (95.36 ± 6.61) and female (87.75 ± 5.33) on triglyceride; P>.005, but it is worthy to note that TG was higher in male subjects for both control and test group than female subjects test and control groups studied. Although the control subjects (male and female) have higher TG than tests subjects (male and female). Furthermore, gender and HDL among control group revealed that there was no significant difference between male (60.00 ± 12.03) and female (69.68 ± 12.43); P>.005. Among pulmonary tuberculosis patients there was no difference between male (34.18 ± 4.41) and female (47.42 ± 5.94) on triglyceride; P>.005, but HDL was higher in female subjects for both control and test group than male test and control subjects studied. Although the test subjects (male and female) have lesser HDL than control subjects (male and female). Finally, on the gender and LDL among control groups revealed that there was no significant difference between male (73.78 ± 18.49) and female (59.68 ± 16.20); P>.005. while gender and LDL among pulmonary tuberculosis patients revealed that there was no significant difference between male (42.00 ± 5.57) and female (35.85 ± 3.79); P<.05, but LDL was higher in male subjects for both control and test group than female test and control subjects studied. Although the test subjects (male and female) have lesser LDL than control subjects (male and female). A study showed that hypo-cholesterolaemia was identified in 140 out of 323 (43.3%, 37.9 - 48.8) pulmonary tuberculous patients. There was high prevalence of lower cholesterol at diagnosis, 51 vs 91 (56.0%, 45.8 - 66.3). This hypo-cholesterolaemia prevalence improved at completion of treatment 19 vs 59 (32.2%, 20.9 - 44.3). Further analysis showed that male, diabetes, and tuberculous treatment duration were associated more with hypo-cholesterolaemia. Hence the study concluded that the overall prevalence of hypo-cholesterolaemia among participants was high especially in males with pulmonary tuberculosis [35].

Studies have indicated preferential utilization of fatty acids over carbohydrates by Mycobacterium tuberculosis. Hence, during chronic infection, cholesterol utilization by tubercle bacilli might decrease the host pool. Decreased rate of production and higher catabolism during chronic infection may also be the cause of low cholesterol level in TB patients [29].

In this study, the level of TC was significantly lower in patients with TB. Similar observations were reported by Deniz., *et al*. [36] and Perez-Guzman., *et al*. [34]. An adequate level of cholesterol is necessary for the proper functioning of the immune system against infection. Perez-Guzman., *et al*. [34] have shown that a cholesterol-rich diet accelerates bacteriologic sterilization in patients with TB. It was found in this study that total cholesterol, TG, HDL and LDL level was decreased significantly in new cases TB patients when compared with TB patients on 2 months and 6 months treatment. TB patients on treatment for 6 months have the highest levels of total cholesterol, TG, HDL and LDL level. Similar results were obtained by Taparia., *et al*. [32]. Similarly, another study was conducted to determine lipid-levels in newly diagnosed and re-treatment PTB patients, with objective of finding the association between lipid-levels with disease severity and inflammatory levels. Newly diagnosed (n = 32) and re-treatment (n = 26) cases of PTB were enrolled in the study. Patients enrolled were of both genders. The average age for men and women was 37.16 ± 1.2 years, 39.44 ± 1.5 years, respectively [37]. In their study, all the lipid parameters were significantly ($p < 0.05$) low in both newly diagnosed and relapse cases of Pulmonary Tuberculosis (PTB) than controls.

Our study is very much reflecting the same trend in our

population. LDL, serum cholesterol, and HDL levels can be utilized as a marker of tuberculosis severity assessment, the lower levels of which indicates advanced disease. Further research is needed on dyslipidemias in TB patients and policy improvements regarding assessment of these lipids and nutritional management [35]. This might implicate on tuberculosis control programs, especially in countries with high prevalence and burden of tuberculosis [37]. Few more study also address role of lipids in immunity against pulmonary tuberculosis and decrease the prevalence of tuberculosis. At the end of discussion, we have concluded that still further research and studies are needed to confirm or exclude the role of lipids in tuberculosis prevalence.

This study concludes few of the parameters of lipid profile were not normal indicating the derangement of these parameters in cases of tuberculosis. To achieve maximum response of TB treatment we must consider normalizing such parameters along with standard treatment to achieve better outcome. From our study we could summarize that there were significant statistical correlation between hypolipidemia and prevalence of pulmonary tuberculosis so further investigation and studies are needed to confirm any correction between pulmonary tuberculosis and hypolipidemia. In this study, we showed that the recovery from TB is accompanied by normalization of lipid parameters such as cholesterol and HDL-C. Despite the rise of lipid parameter levels in TB treated patients, atherogenic indices were somewhat normal.

Conclusion

In conclusion, according to our results, we found that patients with pulmonary tuberculosis have hypocholesterolemia that proved to be a consequence of the disease itself rather than a risk factor. This hypocholesterolemia proved to be correctable to normal levels with regular intake of anti TB treatment and normal diet. Further research is needed with larger number of patients and longer follow up periods in order to provide additional support to this assertion.

Based on the above research finding the following recommendation are forwarded;

- The mechanism of lipid metabolism in PTB disease should be further studied for better understanding patho-physiology of the disease and consequently to novel treatment approaches.
- Lipid profile abnormalities in pulmonary tuberculosis are common and may be used as valuable aids in patients clinical management.
- Patients should be monitored for their marked change of lipid profile, and other risk factors at the time of diagnosis, treatment and follow up.
- The mechanism of lipid metabolism in PTB disease should be further studied for better understanding patho-physiology of the disease and consequently to novel treatment approaches.
- Large observational studies are required to establish a possible role of PTB in lipid alteration and its effect on the rest of biochemical values by using appropriate sample size.
- Studies are needed on tuberculosis applying technology advancement in molecular biology and bacterial genome to identify the genes and enzymes involved in the disease progression and lipid alterations should be considered in further investigation.
- It would be interesting to study the effectiveness of cholesterol supplement alone or in combination with anti-tuberculosis therapy in TB patients care.
- Additional research is needed to more fully assess the link between TB treatment and levels of total cholesterol and its components in patients with pulmonary TB.

Conflict of Interest

The authors declare no conflicts of interest. The authors alone are responsible for the content and the writing of the paper.

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Authors' Contributions

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