



Assessment of the Impact of Pulmonary Tuberculosis on Immunoglobulins in Pulmonary Tuberculosis Patients in Owerri, Imo State

*Ukamaka Edward ¹, Festus Chidi Emengaha ², and Eberechi Nwanguma ¹

¹Department of Medical Laboratory Science, Faculty of Health Science, Imo State University Owerri, Nigeria.

²Department of Medical Biochemistry Faculty of Basic Medical Science Imo State University Owerri, Nigeria.

DOI: [10.5281/zenodo.1817312](https://zenodo.1817312)

Submission Date: 28 Nov. 2025 | Published Date: 07 Jan. 2026

*Corresponding author: **Ukamaka Edward**

Department of Medical Laboratory Science, Faculty of Health Science, Imo State University Owerri, Nigeria

Abstract

Pulmonary tuberculosis (PTB), induced by *Mycobacterium tuberculosis*, is a persistent infectious illness that impacts the human immune system and may modify circulating immunoglobulin levels. This study examined serum levels of immunoglobulins (IgG, IgA, IgM, and IgE) in pulmonary tuberculosis patients at the Federal Teaching Hospital, Owerri, Imo State. A total of three hundred (300) volunteers aged 20–60 years were recruited, consisting of one hundred and fifty (150) verified PTB patients and one hundred and fifty (150) ostensibly healthy age- and sex-matched controls. The GeneXpert MTB/RIF assay was used to check for tuberculosis infection in sputum samples. After that, blood samples were taken from those who had been confirmed to have the disease. We used enzyme-linked immunosorbent assay (ELISA) kits to measure the levels of IgG, IgA, IgM, and IgE in serum. We used SPSS version 21.0 to analyse the data, and the findings were reported as mean \pm standard deviation, with $p < 0.05$ being statistically significant. The findings indicated that the mean serum concentrations of IgG, IgA, and IgE were markedly elevated in PTB patients (1815.17 ± 107.05 mg/dL, 395.93 ± 18.32 mg/dL, and 194.87 ± 26.64 IU/mL, respectively) in comparison to controls (1356.17 ± 40.91 mg/dL, 231.47 ± 11.66 mg/dL, and 83.33 ± 5.76 IU/mL, respectively; $p = 0.001$). On the other hand, PTB patients had much lower levels of IgM (119.67 ± 5.06 mg/dL) than controls (125.30 ± 5.33 mg/dL). Age and sex did not significantly affect immunoglobulin levels in PTB patients ($p > 0.05$). According to treatment status, newly diagnosed patients showed the greatest average levels of IgG, IgA, and IgE and the lowest levels of IgM. Patients on intensive treatment had the next highest levels, and patients on continuation therapy had the lowest levels of IgG, IgA, and IgE but the highest levels of IgM. The alterations were statistically substantial ($p = 0.001$), showing that therapy caused the immune system to change over time. Correlation analysis indicated a significant negative connection between IgG and IgM ($r = -0.718$, $p = 0.001$), alongside robust positive correlations between IgG and IgA ($r = 0.966$, $p = 0.001$) and IgE ($r = 0.982$, $p = 0.001$). In conclusion, pulmonary tuberculosis correlates with increased levels of IgG, IgA, and IgE, alongside diminished quantities of IgM, indicating prolonged antigenic stimulation and immunological dysregulation. Immunoglobulin profiling may thus function as a valuable supplementary tool in assessing disease progression and therapy efficacy in individuals with pulmonary tuberculosis.

Keywords: Pulmonary tuberculosis, immunoglobulin patients, Owerri.

INTRODUCTION

Pulmonary tuberculosis (PTB) is a long-lasting infectious disease of the lungs that is mostly caused by *Mycobacterium tuberculosis*. It is still one of the most persistent and deadly infectious illnesses in the world, infecting millions of people every year and causing a lot of illness and death around the world. *Mycobacterium TB* is an aerobic, non-motile, acid-fast bacillus that is part of the *Mycobacterium tuberculosis* complex. Other members of this complex are *M. bovis*, *M. africanum*, and *M. microti* [1]. The organism has a special cell wall that is rich in lipids and contains mycolic acids. This makes it resistant to drying out, disinfectants, and the immune system of the host, which makes it more likely to survive

and cause disease. Most of the time, transmission happens when infected people cough, sneeze, or talk and release aerosolised droplets into the air [2]. Airborne transmission is most effective in places that are busy and poorly ventilated. It depends on the bacillary load, how long someone is exposed, and how susceptible the host is. Tuberculosis is still one of the top causes of death from a single infectious agent in the world. The World Health Organization's Global Tuberculosis Report says that over 10.3 million people got TB in 2023. This led to an estimated 1.3 million fatalities among people who did not have HIV and an additional 167,000 deaths among persons who did have HIV. The disease burden is much larger in low- and middle-income nations, especially in Southeast Asia and sub-Saharan Africa. Almost a quarter of all TB cases in the world are in Africa. Nigeria is one of the eight nations that make up more than two-thirds of the world's TB burden [3]. Tuberculosis is still a big public health problem in Nigeria, with an expected incidence rate of 219 cases per 100,000 people in 2023. Poverty, overcrowding, malnutrition, poor access to healthcare, and HIV co-infection are still driving transmission. Tuberculosis is still common in Imo State, especially in Owerri, which is a significant city. This shows how important it is to keep doing epidemiological and immunological research [4].

The pathophysiology of pulmonary tuberculosis illustrates a complicated interplay between *M. tuberculosis* and the human immune system. After inhalation, the bacilli arrive in the alveoli, where alveolar macrophages devour them. Instead of being killed, *M. tuberculosis* can live inside cells by stopping the fusion of phagosomes and lysosomes and fighting off oxidative death mechanisms [5]. This causes immune cells to come to the area and develop granulomas, which are a sign of tuberculosis pathology, in order to stop the infection from spreading. The course of disease is contingent upon the equilibrium between bacterial virulence and host immunological competence. Immunocompetent persons can carry latent infections for years without exhibiting symptoms, whereas immunocompromised individuals frequently progress to active disease marked by significant lung inflammation, tissue necrosis, and cavitation. Cellular immunity, especially T-helper 1 (Th1) responses and macrophage activation, is crucial for controlling infection [5]. In addition to cellular immunity, humoral immune responses are increasingly acknowledged as significant factors in the immunopathogenesis of tuberculosis. B lymphocytes make immunoglobulins, which are glycoproteins that come in four types: IgG, IgA, IgM, and IgE. Each kind has its own immune role. IgG is the most common antibody in serum. It helps protect against infections for a long time by opsonisation and neutralisation. IgA is the main kind of antibody found in mucosal secretions. It protects against respiratory pathogens by stopping microbes from sticking to cells. IgM is the first antibody made during primary immune responses and is very good at activating complement. IgE, on the other hand, is involved in hypersensitive reactions and protecting against parasite infections [6].

In pulmonary tuberculosis, immunological homeostasis is frequently compromised by chronic antigenic stimulation, leading to modifications in both cellular and humoral immunity. Chronic infection is linked to hypergammaglobulinemia, especially increased serum IgG and IgA levels, but IgM concentrations may remain stable or decline, indicating chronic rather than acute immune activation [7]. These changes in immunoglobulins may be caused by imbalances in cytokines and changes in Th1/Th2 responses, which impact how B cells develop and how antibodies switch classes. Prolonged immunological activation may result in immune fatigue and dysregulation, hence facilitating disease persistence and tissue damage [8].

Numerous investigations have recorded modified immunoglobulin patterns in TB patients. Reports indicated markedly increased IgG and IgA levels in Nigerian tuberculosis patients relative to healthy controls, implying persistent humoral immune activation. It was noted that elevated IgA levels in active TB patients were associated with disease severity and the degree of pulmonary involvement. These results underscore the potential effectiveness of immunoglobulin profiling as an indicator of disease activity, immunological state, and therapy response [9]. However, variations within groups indicate that genetic background, dietary state, environmental factors, and co-morbid disorders may affect immunoglobulin responses, underscoring the necessity for localised investigations [10].

Even though more people are interested in humoral immunity in tuberculosis, there is still not enough localised data on immunoglobulin patterns among Nigerian TB patients, especially in southeastern Nigeria. The majority of research has concentrated on cellular immunological responses and cytokine profiles, with insufficient emphasis on modifications in specific immunoglobulin classes [11]. Due to Nigeria's genetic diversity and the impact of socioeconomic and environmental factors on immune responses, studies from other locations may not accurately represent local immunological trends. Consequently, assessing serum immunoglobulin levels in pulmonary tuberculosis patients in Owerri, Imo State, is crucial for comprehending local immune response dynamics [12].

This study is to assess blood concentrations of IgG, IgA, IgM, and IgE in pulmonary tuberculosis patients at the Federal Teaching Hospital, Owerri. This work aims to elucidate immunoglobulin variations and their correlation with treatment status, so enhancing the comprehension of tuberculosis immunopathogenesis, facilitating improved disease surveillance, and refining clinical management approaches in this endemic context. Ultimately, these insights may help lessen the burden of tuberculosis and improve patient outcomes in Nigeria.

MATERIALS AND METHODS

Study Area

This study was conducted at the Federal Teaching Hospital (FTH), Owerri, Imo State, Nigeria. Owerri, the capital of Imo State, is located in southeastern Nigeria within the Igbo ethnic region. It is the largest urban center in the state and comprises three Local Government Areas: Owerri Municipal, Owerri North, and Owerri West. The Federal Teaching Hospital is situated in Owerri Municipal. Geographically, Owerri lies at latitude 5.485°N and longitude 7.035°E, approximately 150 m above sea level, covering an estimated land area of about 100 km², with a population of approximately 1,401,873 as of 2016 (NPC, 2016). The city is bordered by the Otamiri River to the east and the Nworie River to the south. Owerri experiences a humid tropical climate with distinct wet (April–October) and dry (November–March) seasons, conditions that may influence the epidemiology of communicable diseases, including tuberculosis.

Ethical approval

The ethical clearance (FTH/OW/HREC/VOL1/157) was obtained from the Ethics Committee of Federal Teaching Hospital, Owerri. Informed consent was obtained from all study participants.

Study Design and Population

This was a hospital-based case–control study conducted between October and December 2024. The study population consisted of adults aged 20–60 years attending the Directly Observed Treatment, Short-course (DOTS) Clinic of the Federal Teaching Hospital, Owerri.

Two main groups were enrolled:

Cases: One hundred and fifty (150) confirmed pulmonary tuberculosis (PTB) patients.

Controls: One hundred and fifty (150) apparently healthy individuals, age- and sex-matched with the cases, with no clinical or laboratory evidence of tuberculosis or other chronic illnesses.

Pulmonary tuberculosis was confirmed using the GeneXpert MTB/RIF assay. The PTB patients were further stratified based on sex (male and female), age groups (20–29, 30–39, and 40–60 years), and treatment status (newly diagnosed prior to treatment initiation, intensive phase, and continuation phase of anti-tuberculosis therapy).

Sample Size Determination

The minimum sample size was calculated using the Cochran formula with a tuberculosis prevalence of 6.67% reported in Nigeria, a 95% confidence interval, and a margin of error of 0.05. The calculated minimum sample size was approximately 100 participants. To improve statistical power and account for non-response, a larger sample size of 300 participants (150 cases and 150 controls) was ultimately recruited.

Inclusion and Exclusion Criteria

Inclusion Criteria:

Participants were included if they:

Were aged between 20 and 60 years.

Had pulmonary tuberculosis confirmed by GeneXpert MTB/RIF assay (for cases).

Were classified according to treatment status (newly diagnosed, intensive phase, or continuation phase).

Provided written informed consent.

Were apparently healthy individuals without evidence of tuberculosis or chronic illness (controls).

Exclusion Criteria:

Individuals were excluded if they:

Were younger than 20 years or older than 60 years.

Had co-existing chronic illnesses such as HIV/AIDS, diabetes mellitus, autoimmune disorders, chronic liver or kidney disease, or malignancies.

Were on immunosuppressive or corticosteroid therapy.

Were pregnant or lactating women.

Declined consent.

Sample Collection and Processing

Sputum Collection and Analysis

Sputum samples were collected from suspected PTB patients under standard biosafety conditions. Participants were instructed to rinse their mouths with clean water before expectorating sputum into sterile, labeled containers in a well-ventilated area. Samples with a minimum volume of 1 mL were disinfected externally with 5% sodium hypochlorite and

transported to the laboratory within 48 hours. Only samples confirmed positive for *Mycobacterium tuberculosis* by GeneXpert MTB/RIF assay were included for further analysis.

Blood Collection and Serum Preparation

Five milliliters (5 mL) of venous blood were collected aseptically from each participant via venipuncture into plain tubes. Samples were allowed to clot and centrifuged at 3000 rpm for 5 minutes to separate serum. The sera were aliquoted into labeled containers and stored at -20°C until analysis.

Laboratory Analysis

Detection of *Mycobacterium tuberculosis*

The GeneXpert MTB/RIF assay (Cepheid, USA) was used for the qualitative detection of *Mycobacterium tuberculosis* complex DNA and rifampicin resistance. The assay is based on real-time polymerase chain reaction (PCR) and molecular beacon technology targeting the *rpoB* gene, providing automated results with minimal operator intervention.

Determination of Serum Immunoglobulins

IgG and IgM: Serum IgG and IgM concentrations were determined using immunoturbidimetric methods, using Biosino Biotechnology reagents. Turbidity was measured spectrophotometrically at 700 nm for IgG and 340 nm for IgM, with concentrations extrapolated from calibration curves.

IgA: Serum IgA was measured using a sandwich enzyme-linked immunosorbent assay (ELISA) kit (Elabscience, USA). Optical density was read at 450 nm, and concentrations were determined using a four-parameter logistic standard curve.

IgE: Serum IgE levels were measured using a sandwich ELISA method (Elabscience, USA). Absorbance was read at 450 nm, and concentrations were derived from standard curves generated using curve-fitting software.

All assays were performed in duplicate, strictly following manufacturers' instructions. Quality control procedures were observed throughout.

Statistical Analysis

Data were analyzed using the Statistical Package for Social Sciences (SPSS) version 21.0. Results were expressed as mean \pm standard deviation (SD). The independent Student's *t*-test was used to compare means between two groups, while one-way analysis of variance (ANOVA) was applied for comparisons among more than two groups. A *p*-value less than 0.05 was considered statistically significant.

RESULTS

Table 4.1 Mean Values of Serum IgG, IgA, IgM and IgE in Pulmonary TB Patients versus Controls (Mean \pm SD)

Parameter	Pulmonary TB Patients (n=150)	Control Subjects (n=150)	t-value	p-value
IgG (mg/dL)	1815.17 \pm 107.05	1356.17 \pm 40.91	21.94	0.001*
IgA (mg/dL)	395.93 \pm 18.32	231.47 \pm 11.66	41.49	0.001*
IgM (mg/dL)	119.67 \pm 5.06	125.30 \pm 5.33	-4.20	0.001*
IgE (IU/mL)	194.87 \pm 26.64	83.33 \pm 5.76	22.42	0.001*

KEY:

n: sample size

*: Statistically significant (*p* < 0.05)

Analysis

Table 4.1 indicates the mean \pm SD values of serum immunoglobulins (IgG, IgA, IgM, and IgE) in pulmonary tuberculosis (TB) patients compared with control subjects.

The mean \pm SD value of IgG was higher in pulmonary TB patients (1815.17 ± 107.05 mg/dL), which was statistically significant (*p* = 0.001) when compared to the mean \pm SD value of the control subjects (1356.17 ± 40.91 mg/dL).

The mean \pm SD value of IgA was higher in pulmonary TB patients (395.93 ± 18.32 mg/dL), which was statistically significant (*p* = 0.001) when compared to the mean \pm SD value of the control subjects (231.47 ± 11.66 mg/dL).

The mean \pm SD value of IgM was lower in pulmonary TB patients (119.67 ± 5.06 mg/dL), which was statistically significant (*p* = 0.001) when compared to the mean \pm SD value of the control subjects (125.30 ± 5.33 mg/dL).

The mean \pm SD value of IgE was higher in pulmonary TB patients (194.87 ± 26.64 IU/mL), which was statistically significant (*p* = 0.001) when compared to the mean \pm SD value of the control subjects (83.33 ± 5.76 IU/mL).

Table 4.2 Mean Values of Serum IgG, IgA, IgM and IgE in Male versus Female Pulmonary TB Patients (Mean \pm SD)

Parameter	Male Pulmonary TB Patients (n=75)	Female Pulmonary TB Patients (n=75)	t-value	p-value
IgG (mg/dL)	1841.00 \pm 120.83	1789.33 \pm 87.79	1.34	0.191
IgA (mg/dL)	400.67 \pm 20.25	391.20 \pm 15.40	1.44	0.161
IgM (mg/dL)	120.07 \pm 4.92	119.27 \pm 5.34	0.43	0.673
IgE (IU/mL)	200.33 \pm 29.49	189.40 \pm 23.15	1.13	0.268

KEY:**n:** sample size**p < 0.05** indicates statistically significant difference

Analysis

Table 4.2 indicates the mean \pm SD values of serum immunoglobulins (IgG, IgA, IgM, and IgE) in male and female pulmonary tuberculosis (TB) patients.

The mean \pm SD value of IgG was higher in male pulmonary TB patients (1841.00 \pm 120.83 mg/dL), which was not statistically significant (p = 0.191) when compared to the mean \pm SD value in females (1789.33 \pm 87.79 mg/dL).

The mean \pm SD value of IgA was higher in male pulmonary TB patients (400.67 \pm 20.25 mg/dL), which was not statistically significant (p = 0.161) when compared to females (391.20 \pm 15.40 mg/dL).

The mean \pm SD value of IgM was slightly higher in male pulmonary TB patients (120.07 \pm 4.92 mg/dL), which was not statistically significant (p = 0.673) when compared to females (119.27 \pm 5.34 mg/dL).

The mean \pm SD value of IgE was higher in male pulmonary TB patients (200.33 \pm 29.49 IU/mL), which was not statistically significant (p = 0.268) when compared to females (189.40 \pm 23.15 IU/mL).

Table 4.3: Comparison of Mean \pm SD Values of Serum IgG, IgA, IgM and IgE among Different Age Groups of Pulmonary Tuberculosis Patients (Mean \pm SD)

Parameters	PTB Patients 0–29 yrs (n=50)	PTB Patients 30–39 yrs (n=50)	PTB Patients 40–60 yrs (n=50)	f-value	p-value
IgG (mg/dL)	1857.50 \pm 115.40*	1837.50 \pm 93.52	1750.50 \pm 87.57	3.27	0.050
IgA (mg/dL)	404.50 \pm 15.85*	400.00 \pm 15.99	383.30 \pm 17.25	4.65	0.018
IgM (mg/dL)	120.20 \pm 5.49	118.40 \pm 5.60	120.40 \pm 4.27	0.46	0.639
IgE (IU/mL)	201.30 \pm 27.99	202.80 \pm 23.39	180.50 \pm 24.69	2.40	0.110

KEY:**n:** population size***:** Statistically significant (P < 0.05)**PTB:** Pulmonary Tuberculosis

Analysis

Table 4.3 indicates the mean \pm SD values of serum immunoglobulins (IgG, IgA, IgM, and IgE) among different age groups of pulmonary tuberculosis (PTB) patients.

The mean \pm SD value of IgG was highest in PTB patients aged 0–29 years (1857.50 \pm 115.40 mg/dL), followed by those aged 30–39 years (1837.50 \pm 93.52 mg/dL) and lowest in those aged 40–60 years (1750.50 \pm 87.57 mg/dL), which was statistically significant (p = 0.050).

The mean \pm SD value of IgA was also highest in PTB patients aged 0–29 years (404.50 \pm 15.85 mg/dL), followed by those aged 30–39 years (400.00 \pm 15.99 mg/dL) and lowest in those aged 40–60 years (383.30 \pm 17.25 mg/dL), which was statistically significant (p = 0.018).

The mean \pm SD value of IgM was lowest in PTB patients aged 30–39 years (118.40 \pm 5.60 mg/dL), followed by those aged 0–29 years (120.20 \pm 5.49 mg/dL) and highest in those aged 40–60 years (120.40 \pm 4.27 mg/dL), which was not statistically significant (p = 0.639).

The mean \pm SD value of IgE was highest in PTB patients aged 30–39 years (202.80 \pm 23.39 IU/mL), followed by those aged 0–29 years (201.30 \pm 27.99 IU/mL) and lowest in those aged 40–60 years (180.50 \pm 24.69 IU/mL), which was not statistically significant (p = 0.110).

Table 4.4: Comparison of Mean \pm SD Values of Serum IgG, IgA, IgM and IgE among Pulmonary Tuberculosis Patients of Different Treatment Status (Mean \pm SD)

Parameters	Newly Diagnosed PTB Patients (n=50)	PTB Patients on Intensive Treatment (n=50)	PTB Patients on Continuation Treatment (n=50)	f-value	p-value
IgG (mg/dL)	1932.00 \pm 54.83*	1788.50 \pm 28.78	1725.00 \pm 90.00	28.27	0.001
IgA (mg/dL)	413.50 \pm 10.29*	394.70 \pm 7.93	379.60 \pm 16.49	19.64	0.001
IgM (mg/dL)	115.20 \pm 3.36*	120.20 \pm 1.32	123.60 \pm 5.46	12.50	0.001
IgE (IU/mL)	222.50 \pm 13.18*	188.10 \pm 7.56	174.00 \pm 25.92	20.68	0.001

KEY:**n:** population size***:** Statistically significant (P < 0.05)**PTB:** Pulmonary Tuberculosis**Analysis**

Table 4.4 indicates the mean \pm SD values of serum immunoglobulins (IgG, IgA, IgM, and IgE) among pulmonary tuberculosis (PTB) patients based on treatment status.

The mean \pm SD value of IgG was highest in newly diagnosed PTB patients (1932.00 \pm 54.83 mg/dL), followed by those on intensive treatment (1788.50 \pm 28.78 mg/dL) and lowest in those on continuation treatment (1725.00 \pm 90.00 mg/dL), which was statistically significant (p = 0.001).

The mean \pm SD value of IgA was highest in newly diagnosed PTB patients (413.50 \pm 10.29 mg/dL), followed by those on intensive treatment (394.70 \pm 7.93 mg/dL) and lowest in those on continuation treatment (379.60 \pm 16.49 mg/dL), which was statistically significant (p = 0.001).

The mean \pm SD value of IgM was lowest in newly diagnosed PTB patients (115.20 \pm 3.36 mg/dL), followed by those on intensive treatment (120.20 \pm 1.32 mg/dL) and highest in those on continuation treatment (123.60 \pm 5.46 mg/dL), which was statistically significant (p = 0.001).

The mean \pm SD value of IgE was highest in newly diagnosed PTB patients (222.50 \pm 13.18 IU/mL), followed by those on intensive treatment (188.10 \pm 7.56 IU/mL) and lowest in those on continuation treatment (174.00 \pm 25.92 IU/mL), which was statistically significant (p = 0.001).

Table 4.5 Pearson Correlation of IgG with IgM, IgA and IgE in Pulmonary TB Patients

Dependent Variable	n	r-value	p-value
IgM	150	-0.718	0.001*
IgA	150	0.966	0.001*
IgE	150	0.982	0.001*

Key:**r** = Pearson correlation coefficient**n** = sample size***:** Significant at the 0.01 level (two-tailed)**Analysis**

Table 4.5 shows the correlation of serum IgG with IgM, IgA, and IgE in pulmonary tuberculosis (PTB) patients.

There was a significant negative correlation of IgG with IgM (r = -0.718, p = 0.001) in pulmonary tuberculosis patients.

There was a significant positive correlation of IgG with IgA (r = 0.966, p = 0.001) and IgE (r = 0.982, p = 0.001) in pulmonary tuberculosis patients.

DISCUSSION

Pulmonary tuberculosis (PTB) continues to be a significant global public health issue, especially in underdeveloped nations, and is induced by *Mycobacterium tuberculosis*, an intracellular infection that predominantly targets the lungs but can impact several organ systems [13]. The infection triggers a multifaceted immune response that encompasses both cellular and humoral elements, which together affect the course of the disease, tissue pathology, and clinical outcomes [14]. Immunoglobulins are a vital component of humoral immunity and elucidate the extent and dynamics of immune activation in chronic infections like PTB [15]. This study assessed serum immunoglobulin profiles in PTB patients in Owerri, Imo State, focussing on differences associated with sex, age, treatment status, and inter-immunoglobulin interactions.

The markedly increased blood IgG levels in PTB patients relative to healthy controls indicate persistent antigenic stimulation due to chronic *M. tuberculosis* infection. Continued exposure to mycobacterial antigens results in sustained B-cell activation and increased IgG production [16]. IgG is important for opsonising and neutralising mycobacterial components, and it also helps produce granulomas, which are a sign of tuberculosis pathology [17]. This observation aligns with reports by [18], which demonstrated elevated IgG concentrations in active TB patients. Nonetheless, inconsistencies with studies indicating stable IgG levels may signify variations in illness stage, immunological competency, or demographic variables [19].

In the same way, serum IgA levels were much greater in PTB patients than in controls. IgA is an important immunoglobulin that protects the mucous membranes and is the first line of defence against respiratory infections. Its increase is probably due to the mucosal immune system being activated by *M. tuberculosis* antigens in the respiratory tract [20]. Increased IgA synthesis may signify an adaptive strategy to limit pathogen adhesion and spread at mucosal surfaces. This conclusion corresponds with prior findings that connect elevated IgA levels with persistent lung infections and the activation of mucosa-associated lymphoid tissue [21].

In contrast, serum IgM levels were marginally but significantly lower in PTB patients compared to controls. IgM is usually made early in an illness and then goes down as the immune system gets stronger and switches to IgG and IgA [22]. The decrease in IgM indicates that tuberculosis is a long-term disease rather than a short-term infection. This finding supports earlier reports that chronic TB cases have lower IgM levels [23].

Serum IgE levels were also notably increased in PTB patients. Elevated IgE may indicate a transition to Th2-type immune responses during chronic infection, with cytokines including interleukin-4 and interleukin-13 facilitating IgE production [24]. Increased IgE has been associated with immunological dysregulation and hypersensitivity reactions in the context of tuberculosis infection (Gudmundsson et al., 2002). The current results align with previous research indicating elevated IgE levels in tuberculosis patients [25].

A sex-based analysis indicated somewhat elevated immunoglobulin levels in males compared to females, however these disparities were not statistically significant. This indicates that sex has a negligible impact on humoral immune responses in PTB within the study cohort. Similar results have been shown by [26], suggesting that immunological responses to *M. tuberculosis* are predominantly analogous between genders, notwithstanding possible hormonal effects noted in other studies [27].

An age-related investigation revealed markedly elevated IgG and IgA levels in younger individuals, accompanied by a decrease in older cohorts. This tendency may be due to immunosenescence, which is when the body makes fewer antibodies and the immune system becomes less sensitive as people get older [28]. Younger individuals typically demonstrate more robust humoral and mucosal immune responses, resulting in increased immunoglobulin synthesis during infection [29]. IgM levels exhibited no significant age-related variation, indicating the relative stability of early immunological responses across age groups, but IgE shown a non-significant reduction with advancing age, perhaps reflecting diminished Th2 activity in older individuals.

An analysis based on treatment showed that IgG, IgA, and IgE levels were highest in patients who had just been diagnosed and then slowly dropped during the intensive and continuation phases of therapy. This trend shows that anti-tuberculosis medication worked to lower the number of mycobacteria and increase the body's immune response [30]. On the other hand, IgM levels rose as treatment went on, which could mean that the immune system is getting better and antibody production is returning to normal as the illness goes away [31]. These results underscore the potential efficacy of immunoglobulin profiling in assessing therapy response.

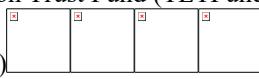
Correlation analysis revealed a substantial negative relationship between IgG and IgM, aligning with immunoglobulin class flipping in the context of persistent infection. Robust positive correlations between IgG and both IgA and IgE indicate synchronised humoral immune activation facilitated by continuous antigen exposure and common regulatory pathways [32]. These interrelationships highlight the intricacy of immune regulation in pulmonary tuberculosis.

CONCLUSION

This study showed that people with pulmonary tuberculosis had far higher levels of serum IgG, IgA, and IgE than healthy controls. IgM levels were only slightly lower. These results suggest increased humoral immune activation linked to persistent *Mycobacterium TB* infection. Changes in immunoglobulin levels between age groups and therapy phases support the idea that antibody profiles show how long an illness lasts and how well a treatment works. Including immunoglobulin profiling in regular clinical evaluations could offer significant insights into disease activity, immunological state, and treatment monitoring in pulmonary tuberculosis, especially in endemic regions like Owerri, Imo State.

Acknowledgements:

The authors are grateful to the Tertiary Education Trust Fund (TETFund) for providing financial support for this research (TETF/DR&D/CE/UNI/IMO/IBR/2020/VOL1.)



Funding

This research was funded by the Tertiary Education Trust Fund (TETFund), Nigeria, through the Imo State University, Owerri, Institution-Based Research (IBR) Intervention (2024).

REFERENCES

1. Adeniyi, O. O., Olofinbiyi, B. A., Adewumi, O. A., Akinsipe, C. I., Abayomi, W., & Thomas, A. A. (2024). Statistical overview on tuberculosis in Nigeria: Epidemiological insights and public health implications. *International Journal of Advanced Community Medicine*, 7(1), 114–119.
2. Dabitao, D., & Bishai, W. R. (2023). Sex and gender differences in tuberculosis pathogenesis and treatment outcomes. *Current Topics in Microbiology and Immunology*, 441, 139–183.
3. Gupta, M., Srikrishna, G., Klein, S. L., & Bishai, W. R. (2022). Genetic and hormonal mechanisms underlying sex-specific immune responses in tuberculosis. *Trends in Immunology*, 43(8), 640–656.
4. Mwape, R. K., Barday, M. A., van der Zalm, M. M., & Verhagen, L. M. (2025). Overview of mucosal immunity and respiratory infections in children: A focus on Africa. *Current Opinion in Pediatrics*, 37(2), 137–144.
5. Ogunniyi, T. J., Abdulganiyu, M. O., Issa, J. B., Abdulhameed, I., & Batisani, K. (2024). Ending tuberculosis in Nigeria: A priority by 2030. *BMJ Global Health*, 9(12), e016820.
6. Riccardi, N., Canetti, D., Martini, M., Diaw, M. M., Di Biagio, A., Codecasa, L., Barberis, I., Bragazzi, N. L., & Besozzi, G. (2020). The evolution of a neglected disease: Tuberculosis discoveries in the centuries. *Journal of Preventive Medicine and Hygiene*, 61(1), E9–E12.
7. Scriba, T. J., Maseeme, M., Young, C., Taylor, L., & Leslie, A. J. (2024). Immunopathology in human tuberculosis. *Science Immunology*, 9(102), eado5951.
8. Gupta, R. K., Lucas, S. B., Fielding, K. L., & Lawn, S. D. (2015). Prevalence of tuberculosis in post-mortem studies of HIV-infected adults and children in resource-limited settings: A systematic review and meta-analysis. *AIDS*, 29(15), 1987–2002.
9. Herrera, M. T., Guzmán-Beltrán, S., Bobadilla, K., Santos-Mendoza, T., Flores-Valdez, M. A., Gutiérrez-González, L. H., & González, Y. (2022). Human pulmonary tuberculosis: Understanding the immune response in the bronchoalveolar system. *Biomolecules*, 12(8), 1148.
10. Okeke, C., Okonkwo, R., Ibeh, N., Chukwuma, O., & Okeke, C. (2023). Assessment of gender differences in some inflammatory cytokines of tuberculosis patients before and during treatment. *African Health Sciences*, 23(3), 336–342.
11. Islam, R. E., Zewdie, M., Mussa, D., Abebe, Y., Ottenhoff, T. H. M., Franken, K. L. M. C., Abebe, F., & Wassie, L. (2025). The role of IgA and IgG in *Mycobacterium tuberculosis* infection: A cross-sectional study in Ethiopia. *Clinical and Experimental Immunology*. Advance online publication.
12. Stewart, P., Patel, S., Comer, A., Muneer, S., Nawaz, U., Quann, V., Bansal, M., & Venketaraman, V. (2023). Role of B cells in *Mycobacterium tuberculosis* infection. *Vaccines*, 11(5), 955.
13. Villar-Hernández, R., Ghodousi, A., Konstantynovska, O., Duarte, R., Lange, C., & Raviglione, M. (2023). Tuberculosis: Current challenges and beyond. *Breathe*, 19(1), 220166.
14. Liu, Q., Que, S., Qiu, Y., Tang, M., Liu, S., Yang, G., Wang, Y., Deng, A., Hu, X., Lian, X., & Gao, Q. (2025). Host immune response to *Mycobacterium tuberculosis* infection: Implications for vaccine development. *Journal of Inflammation Research*, 18(2), 8429–8445.
15. Oladimeji, O., Oladimeji, K. E., Nanjoh, M., Banda, L., Adeleke, O. A., Apalata, T., Mbokazi, J., & Hyera, F. L. M. (2022). Contributory factors to successful tuberculosis treatment in Southwest Nigeria: A cross-sectional study. *Tropical Medicine and Infectious Disease*, 7(8), 194.
16. Ilyas, U., Mahmood, A., Pansuriya, A. M., Umar, Z., & Landry, I. (2022). Miliary tuberculosis: A case report highlighting the diagnostic challenges associated with the condition. *Cureus*, 14(9), e29339.
17. Fukunaga, R., Glaziou, P., Harris, J. B., Date, A., Floyd, K., & Kasaeva, T. (2021). Epidemiology of tuberculosis and progress toward meeting global targets—Worldwide, 2019. *Morbidity and Mortality Weekly Report*, 70(12), 427–430.
18. Gill, C. M., Dolan, L., Piggott, L. M., & McLaughlin, A. M. (2022). New developments in tuberculosis diagnosis and treatment. *Breathe*, 18(1), 210149.
19. Zellweger, J. P., Sotgiu, G., Corradi, M., & Durando, P. (2020). The diagnosis of latent tuberculosis infection (LTBI): Currently available tests, future developments, and perspectives to eliminate tuberculosis (TB). *La Medicina del Lavoro*, 111(3), 170–183.

20. Sharma, S. K., Mohan, A., & Kohli, M. (2021). Extrapulmonary tuberculosis. *Expert Review of Respiratory Medicine*, 15(7), 931–948.
21. Grace, P. S., Dolatshahi, S., Lu, L. L., Cain, A., Palmieri, F., Petrone, L., Fortune, S. M., Ottenhoff, T. H. M., Lauffenburger, D. A., Goletti, D., Joosten, S. A., & Alter, G. (2021). Antibody subclass and glycosylation shift following effective TB treatment. *Frontiers in Immunology*, 12, 679973.
22. Greene, D., Moore Fried, J., & Wang, J. (2025). IgE in allergic diseases. *Immunological Reviews*, 334(1), e70057.
23. Shamji, M. H., Valenta, R., Jardetzky, T., Verhasselt, V., Durham, S. R., Würtzen, P. A., & van Neerven, R. J. J. (2021). The role of allergen specific IgE, IgG, and IgA in allergic disease. *Allergy*, 76(12), 3627–3641.
24. Yang, Q., Han, J., Shen, J., Peng, X., Zhou, L., & Yin, X. (2022). Diagnosis and treatment of tuberculosis in adults with HIV. *Medicine*, 101(35), e30405.
25. Wang, Q., Nag, D., Baldwin, S. L., Coler, R. N., & McNamara, R. P. (2024). Antibodies as key mediators of protection against *Mycobacterium tuberculosis*. *Frontiers in Immunology*, 15(1), 1430955.
26. Lourens, R., Singh, G., Arendse, T., Thwaites, G., & Rohlwink, U. (2025). Tuberculous meningitis across the lifespan. *The Journal of Infectious Diseases*, 231(5), 1101–1111.
27. Mahapatra, A., Thiruvengadam, K., Nair, D., Padmapriyadarsini, C., Thomas, B., Pati, S., Bulliyya, G., Das, D., Chowdhury, J., Bang, A., & Swaminathan, S. (2024). Effectiveness of food supplement on treatment outcomes and quality of life in pulmonary tuberculosis: Phased implementation approach. *PLOS ONE*, 19(7), e0305855.
28. Fu, J., Li, J., Liu, Z., Zheng, S., Li, X., Ning, X., Wang, J., Gao, W., & Li, G. (2022). Sex-specific differences in the clinical profile among patients with tracheobronchial tuberculosis: A hospital-based cross-sectional study in Shenzhen, China. *International Journal of General Medicine*, 15(3), 5741–5750.
29. Zhao, E. J., Cheng, C. V., Mattman, A., & Chen, L. Y. C. (2021). Polyclonal hypergammaglobulinaemia: Assessment, clinical interpretation, and management. *The Lancet Haematology*, 8(5), e365–e375.
30. Zhuang, L., Yang, L., Li, L., Ye, Z., & Gong, W. (2024). *Mycobacterium tuberculosis*: Immune response, biomarkers, and therapeutic intervention. *MedComm*, 5(1), e419.
31. Consonni, F., Chiti, N., Ricci, S., Venturini, E., Canessa, C., Bianchi, L., Lippi, F., Montagnani, C., Giovannini, M., Chiappini, E., Galli, L., Azzari, C., & Lodi, L. (2022). Unbalanced serum immunoglobulins in clinical subtypes of pediatric tuberculosis disease. *Frontiers in Pediatrics*, 10(3), 908963.

CITATION

Edward, U., Emengaha, F. C., & Nwanguma, E. (2026). Assessment of the Impact of Pulmonary Tuberculosis on Immunoglobulins in Pulmonary Tuberculosis Patients in Owerri, Imo State. In Global Journal of Research in Medical Sciences (Vol. 6, Number 1, pp. 6–14). <https://doi.org/10.5281/zenodo.18173128>