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Evaluation of immunoglobulin M and A level in individuals with HIV-TB co-infection in NAUTH, Nnewi

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ABSTRACT

This study evaluated the serum concentrations of Immunoglobulin M and Immunoglobulin A in individuals with TB, HIV and HIV-TB co-infection in Nnamdi Azikiwe University Teaching Hospital (NAUTH), Nnewi South East, Nigeria. A total of 80 participants (males=44; females=36), with a mean age of 35.83±7.3 were randomly recruited by convenient sampling technique from patients attending TB-DOTS and HIV Clinics in NAUTH and grouped as: TB positive individuals (Group I), HIV-TB co-infection (Group II), HIV positives (Group III), and control (Group IV). 5mls of venous blood samples were collected from each participants using plain containers for estimation of immunoglobulin A and M classes by Immuno-turbidimetric Method. The results obtained showed that mean serum levels of IgM (78.14±42.41 and IgA (374.45±15.69) were significantly increased in participants with HIV-TB co-infection when compared with TB (49.21±3.67) and (315.41±52.79), HIV (41.91±25.97) and (342.23±92.67 and control groups (55.82±35.36) and (322.66±71.34) at $p<0.05$ respectively. There were significant increases in the mean serum IgM ($p=0.048$), IgA ($p=0.000$) and age ($p=0.001$) when group I participants were compared with group II individuals respectively, while IgM showed significant increase ($p=0.013$) in group II than in group III only. Furthermore, participants in group II showed significantly increased mean serum IgA levels compared to control ($p=0.005$), whereas significant negative correlations were seen in age and gender in various groups studied ($p<0.05$). Thus, evaluation of these antibodies may be used alongside other systemic markers and parameters to aid in predicting disease severity and progression and also monitoring response to treatment. This approach could further complement the current TB management especially in HIV-TB co-infection.

1. Introduction

Human immune deficiency virus (HIV) causes Acquired Immunodeficiency Syndrome (AIDS), that affects the cells of the immune system leading to immunodeficiency in affected individuals due to continuous depletion of CD4 T-cells.[1–4] This leaves the affected persons vulnerable to all forms of opportunistic pathogens. It is transmitted via sexual intercourse, shared intravenous drug paraphernalia, and mother-to-child transmission (MTCT), which can occur during the birth process or during breastfeeding by means of contact with infected body fluids such as blood, breast

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milk, semen and vaginal secretions.[5] HIV continues to be a major global public health issue with an estimated 37.7 million [30.2–45.1 million] people living with HIV and 680 000 [480 000–1.0 million] deaths from HIV-related causes in the 2020.[6]

Tuberculosis (TB) is an infectious disease caused by Mycobacterium tuberculosis (Mtb).[7] The spectrum of tuberculosis (TB) in humans is most widely characterized by two clinical states: active TB and latent TB. Individuals with active TB exhibit symptoms such as hemoptysis, fever and weight loss with detectable bacteria, while individuals with latent TB are not overtly clinically ill, have no detectable *Mycobacterium tuberculosis (Mtb),* and therefore no transmission risk.[8] TB infection occurs following the inhalation of droplets produced when an individual with active TB disease coughs or sneezes.[9] A total of 1.4 million people died from TB in 2019 (including 208 000 people with HIV). Worldwide, TB is one of the top 10 causes of death and the leading cause from a single infectious agent (above HIV/AIDS).[10] Indeed, tuberculosis (TB) is one of the deadliest infectious diseases that became a significant public health problem worldwide.[11] Notably Nigeria ranks among the eight countries listed as being responsible for two thirds of the total global TB burden[10] with high prevalence reported in various parts of Nigeria.[12] Increased TB risk has been associated with a low CD4+-cell count (≤200 cells/μL) and high viral load (>200 copies/mL).[13]

Together, HIV and Mycobacterium tuberculosis are the two leading causes of death among people living with HIV, accounting for one in four HIV-related deaths,[14,15] and continue to pose serious significant public health challenge in developing countries.[15,16] At least one-third of the people living with HIV worldwide are infected with latent *Mycobacterium tuberculosis.*[14] Varying prevalence of HIV-TB co-infection has been documented previous in both male and female individuals.[17,18] HIV and TB form a lethal combination, each speeding the other's progress. In 2019, about 208 000 people died of HIV-associated TB.[10] Some studies have shown elevated levels of IgA in the sera of HIV/TB co-infected individuals as well as in HIV positive persons but not in individuals with pulmonary tuberculosis (PTB) with significantly higher levels of IgM in PTB, HIV, and HIV/TB co-infection.[19] Other similar studies also documented variable results in the light of the above,[20,21] thus suggesting that the evaluation of serum immunoglobulins such as IgM and IgA may be beneficial in the prediction or monitoring of TB progression in HIV/TB co-infected individuals. Given, the increasing trends in HIV/TB co-infection in Nigerian population which poses significant treat to medical care of HIV/AIDS patients, there is need for timely and proper diagnosis and disease monitoring through the use of serological markers of immune activation associated with HIV and tuberculosis infections. Thus, we evaluated the levels of immunoglobulin M and A in participants with HIV-TB co-infection in NAUTH, Nnewi.

2. Materials and Methods

2.1. Study design

This study is case-controlled study designed to evaluate immunoglobulin M and A levels in participants with HIV-TB co-infection at the Directly Observed Treatment Short Course Centre for tuberculosis (TB-DOTs) and HIV Clinic in Nnamdi Azikiwe University Teaching Hospital, Nnewi, Anambra State, Nigeria. A total of 80 participants were randomly recruited for this study and comprised of 20 participants with TB infection only (group I), 20 participants with HIV-TB co-infection (group II), 20 participants with HIV infection only (group III) and 20 control participants (HIV and TB negative; group IV).

2.2. Inclusion criteria

1. Individuals who were HIV sero-negative and tested positive for the presence of mycobacterium tuberculosis and are actively on tuberculosis treatment.
2. Individuals who were HIV sero-positive and tested negative for the presence of mycobacterium tuberculosis and are actively on antiretroviral drugs.
3. Participants who tested positive to tuberculosis and also HIV sero-positive and are on antiretroviral therapy.
4. Participants aged between 18 and 60 years, who gave informed consent was eligible for inclusion.
5. Apparently healthy blood donors without any clinical symptoms were recruited as control participants.

2.3. Exclusion criteria

Individuals excluded from this study were children (0-17 years), adults above 60 years and pregnant women.

2.4. Sample collection

5ml venous blood sample was collected from participants using sterile disposable syringes into sterile plain tubes. The samples stood for 30minutes to clot, then centrifuged for 5minutes at 3500rpm. The serum was transferred into metal free plain tube and stored frozen at -20^{o}C until analysed.

2.5. Laboratory analysis

Control participants were screened for HIV status using HIV rapid test kits according to the National serial algorithm, using 3 types of commercial kits: Determine; Uni-Gold and Stat-Pak (tie-breaker) and testing was conducted according to manufacturer's manual. The quantification of serum immunoglobulin IgA, and IgM levels were performed using Total Human IgA, and IgM

Table 1: Mean± SD of IgM, IgA, age, duration of infection and treatment in participants studied.

Variables	IgM(mg/dl)	IgA(mg/dl)	Age (years)	Duration of infection (month)	Duration of treatment (month
TB infection (n=20)	49.21±31.67	315.41±52.79	32.10±8.08	7.20±2.88	6.37±2.50
HIV-TB co-infection (n=20)	78.13±42.40	374.45±15.69	45.53±11.17	6.69±2.50	6.69±2.50
HIV infection (n=20)	41.91±25.97	342.23±92.67	42.45±7.42		
Control (n=20)	55.82±35.36	322.66±71.34	23.25±2.51		
f-value	3.28	2.330	32.302	0.21	0.130
p-value	0.026*	0.042*	0.000*	0.606	0.721

*Statistically significant at p<0.05.

Table 2: Mean± SD of IgM, IgA, and age of TB and HIV-TB co-infection in participants studied.

Variables	IgM(mg/dl)	IgA(mg/dl)	Age (years)
TB infection (n=20)	49.21±31.67	315.41±52.79	32.10±8.08
HIV-TB co-infection (n=20)	78.13±42.40	374.45±15.69	45.53±11.17
t-value	2.513	8.620	2.120
p-value	0.048*	0.000*	0.001*

*Statistically significant at p<0.05.

Table 3: Mean± SD of IgM, IgA, and age of TB and HIV positive participants studied.

Variables	IgM(mg/dl)	IgA(mg/dl)	Age (years)
TB infection (n=20)	49.21±31.67	315.41±52.79	32.10±8.08
HIV (n=20)	41.91±25.97	342.23±92.67	42.45±7.42
t-value	0.113	1.431	0.136
p-value	0.430	0.270	0.000*

*Statistically significant at p<0.05.

Table 4: Mean± SD ofIgM, IgA, and age of TB and control in participants studied.

Variables	IgM(mg/dl)	IgA(mg/dl)	Age (years)
TB infection (n=20)	49.21±31.67	315.41±52.79	32.10±8.08
Control (n=20)	55.82±35.36	322.66±71.34	23.25±2.51
t-value	1.038	1.824	16.930
p-value	0.537	0.717	0.000*

*Statistically significant at p<0.05.

Table 5: Mean± SD of IgM, IgA, and age of HIV-TB co-infection and HIV participants studied.

Variables	IgM(mg/dl)	IgA(mg/dl)	Age (years)
TB infection (n=20)	78.13±42.40	374.45±15.69	45.53±11.17
HIV-TB co-infection (n=20)	41.91±25.97	342.23±92.67	42.45±7.42
t-value	5.187	6.170	3.293
p-value	0.013*	0.143	0.390

*Statistically significant at p<0.05.

Table 6: Mean± SD ofIgM, IgA, and age of HIV-TB co-infection and control group studied.

Variables	IgM(mg/dl)	IgA(mg/dl)	Age (years)
HIV-TB co-infection (n=20)	78.13±42.40	374.45±15.69	45.53±11.17
Control (n=20)	55.82±35.36	322.66±71.34	23.25±2.51
t-value	0.657	13.575	27.464
p-value	0.129	0.005*	0.000*

*Statistically significant at p<0.05.

Table 7: Mean± SD ofIgM, IgA, and age of HIV and control group studied.

Variables	IgM(mg/dl)	IgA(mg/dl)	Age (years)
HIV positives (n=20)	41.91±25.97	342.23±92.67	42.45±7.42
Control (n=20)	55.82±35.36	322.66±71.34	23.25±2.51
t-value	2.631	0.057	15.496
p-value	0.165	0.459	0.000*

*Statistically significant at p<0.05.

Table 8: Mean± SD ofIgM, IgA, age, duration of infection and treatment in males and females participants studied.

Variables	IgM(mg/dl)	IgA(mg/dl)	Age (years)	Duration of infection (months)	Duration of treatment (months)
Male (n=44)	52.32±36.34	336.97±63.75	35.68±12.78	7.22±2.73	6.39±2.57
Female (n=36)	56.41±33.74	333.18±76.84	33.97±9.62	6.73±2.74	6.64±2.41
t-value	0.224	0.076	5.789	0.009	0.073
p-value	0.620	0.821	0.518	0.613	0.776

*Statistically significant at p<0.05.

Table 9: Levels of association between parameters studied in TB Participants (Group 1)

Parameters	Pearson r correlation	Participants (n)	f-value	p-value
IgM Vs IgA	0.453	20	0.045	>0.05
IgM Vs Gender	-0.050	20	0.833	<0.05
IgM Vs Age	-0.265	20	0.259	<0.05
IgM Vs Duration of infection	0.510	20	0.022	>0.05
IgM Vs Duration of treatment	0.358	20	0.122	>0.05

Table 10: Levels of association between parameters studied in TBVs HIV-TB Co-infection (Group 1 Vs 2)

Parameters	Pearson r correlation	Participants (n)	f-value	p-value
IgM Vs IgM2	-0.041	20	0.895	<0.05
IgM Vs IgA2	0.270	20	0.373	>0.05
IgM Vs Gender2	-0.100	20	0.746	<0.05
IgM Vs Age2	-0.192	20	0.529	<0.05
IgM Vs Duration of infection2	0.263	20	0.386	>0.05
IgM Vs Duration of treatment2	0.263	20	0.386	>0.05

Table 11: Levels of association between parameters studied in TBVs HIV Participants (Group 1 Vs 3)

Parameters	Pearson r correlation	Participants (n)	f-value	p-value
IgM Vs IgM3	0.194	20	0.414	>0.05
IgM Vs IgA3	0.285	20	0.223	>0.05
IgM Vs Gender3	-0.469	20	0.037	<0.05
IgM Vs Age3	0.027	20	0.912	<0.05

Table 12: Levels of association between parameters studied in TBVs HIV-TB Co-infection (Group 2)

Parameters	Pearson r correlation	Participants (n)	f-value	p-value
IgM2 Vs IgA2	0.059	20	0.849	>0.05
IgM2 Vs Gender2	0.513	20	0.073	>0.05
IgM2 Vs Age2	-0.405	20	0.170	<0.05
IgM2 Vs Duration of infection2	-0.525	20	0.065	<0.05
IgM2 Vs Duration of treatment2	-0.525	20	0.065	<0.05

Table 13: Levels of association between parameters studied in HIV-TB Co-infectionVs HIV (Group 2 Vs 3)

Parameters	Pearson r correlation	Participants (n)	f-value	p-value
IgM2 Vs IgM3	0.434	20	0.138	>0.05
IgM2 Vs IgA3	-0.187	20	0.541	<0.05
IgM2 Vs Gender3	-0.220	20	0.469	<0.05
IgM2 Vs Age3	0.160	20	0.602	>0.05

Table 14: Levels of association between parameters studied in HIV (Group 3)

Parameters	Pearson r correlation	Participants (n)	f-value	p-value
IgM3 Vs IgA3	0.269	20	0.251	>0.05
IgM3 Vs Gender3	-0.161	20	0.498	<0.05
IgM3 Vs Age3	0.102	20	0.668	>0.05

test kits, respectively (Chemelex, S.A., Barcelona) which employed a highly sensitive two-site Enzyme Linked Immunoassay (ELISA) technique. Optical Density of tests were read at wave length of 450nm using a Biobase hormone assay reader.

2.6. Statistical analysis

Statistical Package for the Social Sciences (SPSS) version 23.0 was used for the analysis of the results. Data obtained was presented as mean ± standard deviation (SD) and analyzed statistically using one way analysis of variance (ANOVA), posthoc t-test and Pearson correlation. The level of significance was set at P< 0.05.

3. Results

The mean serum levels of IgM and IgA as well as the mean age of the participants were significantly different compared amongst the groups studied (p<0.05) respectively. See Table 1.

The mean serum levels of IgA and IgM were significantly higher in HIV-TB co-infected participants than in TB infected participants only (p=0.048; 0.000) respectively. Also, the mean age of the HIV-TB co-infected participants were significantly higher than in TB infected participants (p=0.001). See Table 2.

There were no significant differences in the mean serum levels of IgA and IgM in TB infected participants when compared with HIV positive participants only (p>0.05) respectively although the mean age of the HIV infected participants were significantly higher than in TB infected participants (p=0.001). See Table 3.

Also, the mean serum IgA and IgM levels did not differ significantly in TB infected participants when compared between control participants (p=0.537; 0.717) respectively. See Table 4.

However, the mean serum IgM level was significantly higher in TB infected participants when compared to HIV-TB co-infected participants (p=0.013), while the mean serum IgA level and age of the participants did not differ significantly when compared between both groups (p=0.143; 0.390) respectively. See Table 5.

Further, the mean serum IgA levels as well as the mean age of participants were significantly higher in HIV-TB co-infected participants when compared to control participants (p=0.005; 0.000) respectively, whereas the mean serum IgM level were similar in both groups (p=0.129). See Table 6.

There were no significant differences in the mean serum levels of both IgA and IgM in HIV positive participants when compared to the control respectively (p=0.459; 0.165). See Table 7.

Also, there were no significant differences in the mean serum levels of IgM and IgA, as well as the age of participants, duration of infection and treatment in the males participants than in females participants studied (p>0.05). See Table 8.

Table 9 Shows the levels of association between parameters studied in TB (Group 1).this table shows there was no significant correlation between serumIgM Vs IgA (r=0.453, p>0.05), IgM Vs duration of infection (r=0.510, p>0.05), and IgM Vs duration of treatment (r=0.358, p>0.05). However, there was significant negative correlation between serum IgM Vs age (r= -0.265, p<0.05) and IgM Vs gender (r= -0.050, p<0.05).

Table 10 Shows the levels of association between parameters studied in TBVs HIV-TB co-infection (Group 1 Vs 2). There was no significant correlation between serum IgM Vs IgA2 (r=0.270, p>0.05), IgM Vs duration of infection2 (r=0.263, p>0.05), IgM Vs duration of treatment2 (r=0.263, p>0.05). However, there was significant negative correlation between serum IgM Vs IgM2 (r= -0.041, p<0.05), IgM Vs age (r= -0.192, p<0.05) and IgM Vs gender (r= -0.100, p<0.05).

Table 11 Shows the levels of association between parameters studied in TBVs HIV infection (Group 1 Vs 3). There was no significant correlation between serum IgM Vs IgM3 (r=0.194, p>0.05), IgM Vs IgA3 (r=0.285, p>0.05). But, there was significant negative correlation between serum IgM Vs gender (r= -0.469, p<0.05), and significant positive correlation between IgM Vs age (r= 0.027, p<0.05).

Table 12 Shows the levels of association between parameters studied in HIV-TB co-infection (Group 2). There was no significant correlation between serum IgM2 Vs IgA2 (r=0.059, p>0.05), and IgM Vs gender2 (r=0.513, p>0.05). However, there was significant negative correlation between serum IgM2 Vs age (r= -0.405, p<0.05), IgM2 Vs duration of infection2 (r= -0.525, p<0.05), and IgM2 Vs duration of treatment2 (r=0.525, p<0.05).

Table 13 Shows the levels of association between parameters studied in HIV-TB co-infectionVs HIV (Group 2 Vs 3). There was no significant correlation between serum IgM2 Vs IgM3 (r=0.434, p>0.05), and IgM2 Vs age3 (r=0.160, p>0.05), while there was significant negative correlation between serum IgM2 Vs IgA3 (r= -0.187, p<0.05) and IgM2 Vs gender3 (r= -0.220, p<0.05).

Table 14 Shows the levels of association between parameters studied in HIV (Group 3). This table shows there was no significant correlation between serum IgM3Vs IgA3 (r=0.269, p>0.05), IgM3 Vs age3 (r= 0.102, p>0.05), while there was significant negative correlation between serum IgM Vs gender (r= -0.161, p<0.05).

4. Discussion

In this study, IgM and IgA levels were investigated in TB, HIV, and HIV-TB co-infection. Tuberculosis and HIV/AIDS (acquired immunodeficiency syndrome) constitute the main burden of infectious disease in resource-limited countries like Nigeria. In the individual host, the two pathogens, *Mycobacterium tuberculosis* and HIV, potentiate each other, accelerating the deterioration of immunological functions and resulting in premature death if untreated.[22] The determination of IgM and IgA levels in TB, HIV and HIV-TB co-infection could help predict or monitor TB disease progression especially in HIV-TB co-infection.

It was observed that age varied significantly among all the groups studied. Majority of those with TB, HIV-TB co-infection, HIV and control had mean ages of 32.10±8.08, 45.53±11.10, 42.45±7.42 and 23.25±2.51 years respectively. This agrees with previous reports indicating that both pathogens affect mostly individuals in their economically productive years.[5] Nigeria still ranks among the eight countries listed as being responsible for two-thirds of the total global TB burden[10] with high prevalence reported in various parts of Nigeria[12] and HIV prevalence among adults in Nigeria recorded at 3.2%.[23]

The mean total serum levels of IgA and IgMin TB, HIV-TB co-infection, HIV and control participants were significantly elevated variably. This finding is in keeping with some other previous studies in southeastern Nigeria as well as in other African countries.[20,21] Serum IgA was significantly elevated in HIV-TB co-infection when compared with TB group which is similar to the result of Moses and colleagues.[19] In line with the present result, Abdul and Andrew observed that the presence of low bacillary burden in less severe TB cases may lead to low IgA concentration unlike in more severe cases such as in the case of HIV-TB co-infection which is characterized by elevation in IgA levels.[24] Also, IgA was elevated in the HIV infected individuals compared to TB infected participants although the elevation was not statistically significant. There was no significant elevation when the serum levels of IgA were compared between TB infected and control individuals. Co-infected participants on the other hand showed significant elevation in IgA when compared with control and HIV positive participants. This partly agrees with the study of Moses et al. that recorded significantly higher IgA levels in HIV-TB co-infected participants compared with control but further noted no significant differences in the mean serum IgA level in HIV-TB co-infection than in HIV positive individuals.[19] The increase in IgA levels seen in HIV-TB co-infection in this study is in agreement with the fact that IgA is the most important immunoglobulin in mucosal immunity and this increase is seen in mucosal infections such as TB and HIV as TB affects the respiratory system and HIV mainly infects through the reproductive system. IgA is critical at protecting mucosal surfaces from toxins, viruses, and bacteria by direct neutralizations by preventing the binding to mucosal surfaces.[25] The level of IgA is dependent on the severity of the infection. As seen from the result, more severe infection such as HIV-TB co-infection showed elevated serum levels of IgA.

Surprisingly, the mean serum levels of IgA did not differ significantly when compared between HIV infected participants and control group. Ndiokwere and coworkers as well as Ifeanyichukwu et al. found no significant differences in the mean serum IgA levels in HIV positive participants compared with control groups which are in consonance with the result of this study[26,27]

In this study, it was also observed that the mean serum levels of IgM in TB, HIV-TB co-infection, HIV and control groups was also significantly elevated variably. This is in line with the work of Amilo et al. who reported that the mean serum IgM levels in TB, HIV and HIV-TB co-infected patients were significantly elevated. This elevation was marked in HIV-TB co-infection. IgM is frequently associated with immune response to antigenically complex blood-borne infectious organisms; hence the significant increase in HIV-TB co-infection.[27] Serum IgM levels was also elevated in TB infected participants when compared with HIV positive individuals although, this elevation was not statistically significant and may be due to response to TB treatment in the TB infected participants. Co-infected participants showed significant elevation in serum IgM levels when compared with HIV infected individuals. This may be as a result of the fact that both HIV and TB potentiate the immune response to each other. The increase may be due to stimulation of immunoglobulin synthesis by opportunistic infections or direct stimulation by HIV itself.

Furthermore, HIV-TB co-infection showed elevated level of IgM when compared to control. This corroborates well with the previous report some similar studies.[19]

When serum IgM levels of HIV participants was compared with control, serum IgM levels were decreased in HIV seropositives than in control. This confirms the results of some previous similar studies,[28] although it was in contrast with the report of Ifeanyichukwu et al. which reported an increase in IgM concentration in HIV seropositive participants compared with HIV negative healthy individuals which may be attributed to challenge and activation of mature B cells by HIV antigens, which initially produce a relatively non-specific IgM.[29] Thus, it could be that HIV infection present different pattern of antibody response depending on the stage of the disease.

The present study showed that serum IgM levels were higher in females than in males, though this elevation was not significant. IgM is regarded as a potent agglutinin and a monomer of IgM is used as a B-cell receptor[30] and it mediates mucosal immunity by binding to poly-immunoglobulin receptors in a process that brings it to mucosal surfaces and secretions such as gut lumen and breast milk.[31]

In the TB participants, there was no significant correlation observed between IgM and IgA. This may be due to the difference in the location of these antibodies in the body, since IgA is present mostly on mucosal surfaces and secretions. But there was a significant negative correlation between IgM Vs age and IgM Vs gender. It is a well known notion that humoral immunity tends to be higher in females than in males[32,33] and also IgM decreases with age.[34]

Also, between TB participants and those with HIV-TB co-infection, there was a negative correlation between IgM in TB and IgM in co-infection. This may be due to exacerbation of immune response in HIV-TB co-infection. Negative correlations were also seen in gender while positive significant correlation was seen in age. However, there was no significant correlation between IgM in TB and IgA in co-infection which may be due to difference in the location of these antibodies in the body. There was also no correlation between IgM in TB IgM and IgA in HIV but negative significant correlation was seen in IgM Vs age as well as in IgM Vs gender. More so, between HIV-TB participants and HIV participants, IgA and gender showed negative correlation to IgM. Significant negative correlation was observed in HIV participants between IgM Vs age. The appreciable increase in levels of IgA and IgM in HIV-TB co-infection observed in this study may be a pointer to an evidence of polyclonal B cell activation in patients with co-infection. Other studies have shown that the HIV viral envelope proteins especially gp41 which induces polyclonal B cell activation usually results in excessive and abnormal serum levels of immunoglobulins.[21] Thus, evaluating IgM and IgA levels in individuals with co-infection may provide useful information on disease progression and severity and also aid in monitoring of treatment.

5. Conclusion

The levels of IgM and IgA were elevated in HIV-TB co-infection. Also, IgM level was higher in TB than in HIV infected individuals while IgA was elevated in HIV than in TB infected individuals. Significant negative correlations were recorded between antibody levels Vs age, and gender. Thus, evaluation of these antibodies may be used alongside other systemic markers and parameters to aid in predicting disease severity and progression and also monitoring response to treatment. This approach could further complement the current TB management especially in HIV-TB co-infection.

6. Ethical Approval

The ethical approval for this research was obtained from Nnamdi Azikiwe University Teaching Hospital Ethical Committee (Reference no: NAUTH/CS/66B/VOL.2/076).

7. Source of Funding

None.

8. Conflict of Interest

None.

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