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ASSESSMENT OF RENAL AND HEPATIC DYSFUNCTION AMONG TYPHOID FEVER PATIENTS IN BINGHAM UNIVERSITY TEACHING HOSPITAL, JOS, NIGERIA: A RETROSPECTIVE STUDY

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ABSTRACT

Typhoid fever-induced organ dysfunction contributes greatly to high morbidity and mortality associated with the disease. It is crucial to monitor all affected organs during treatment to improve patients' survival. This retrospective study was conducted to evaluate the impact of typhoid fever on hepatic and renal functions among patients who attended Bingham University Teaching Hospital between 2019 and 2020. Records of 300 patients comprising 150 confirmed typhoid cases through widal test and blood culture, and 150 healthy controls were analysed. Results showed that typhoid fever resulted in severe anaemia, elevated serum liver enzymes and thrombocytopenia, however, significant alterations were more severe in male patients compared to females. Significant reduction in serum glucose levels, total white blood cell and differential white blood counts except lymphocyte counts were observed in typhoid fever patients. Although there was non-significant decrease in serum albumin and total protein of the patients, gender-specific analysis revealed a significant increase in serum albumin among female patients in contrast to male patients that exhibited a significant decrease in serum albumin. There were no significant differences in renal function parameters between the patient and the control except serum urea. In conclusion, hepatic dysfunction, severe anaemia and thrombocytopenia are strongly associated with typhoid fever in this population with the severity more pronounced among male patients compared to females, while renal impairment is relatively uncommon. It is advisable that patients in this area undergo liver and kidney function assessment in addition to their treatment to facilitate early detection of typhoid-induced hepatic and renal damage.

Keywords: Typhoid fever, Salmonella typhi, Liver dysfunction, Kidney dysfunction, Sanitary infrastructure

INTRODUCTION

Typhoid fever is a preventable deadly disease that predominantly affects people in low-income nations where people live in congested and unhygienic environments lacking essential amenities such as clean water, proper sanitation, safe food sources, and effective waste disposal systems (Cruz Espinoza *et al.*, 2019; Erhuotor *et al.* 2023; Garrett *et al*., 2022). Typhoid is transmitted through consumption of food and water contaminated with urine or faeces of individuals infected with *Salmonella typhi* or asymptomatic carriers of the pathogen (Ahmad *et al*., 2023; Garrett *et al*., 2022). Transmission can also occur through the consumption of raw fruits, vegetables cultivated with human faeces and contaminated dairy products (Abdulkarim *et al*., 2023; Ochiai *et al.,* 2008). Outbreaks often occur in communities relying on contaminated water as their main source of drinking water (Cruz Espinoza *et al.,* 2019). In addition, the persistence and spread of typhoid are also influenced by the emergence of antibiotic-resistant *Salmonella typhi* strains, poor hygienic practices and lack of affordable quality healthcare (Butt *et al*., 2021; Khanam *et al.,* 2022).

There are estimated 11-12 million incidences of typhoid fever annually which result in approximately 128,000 -161,000 death worldwide (Osu et al., 2024). In Nigeria, typhoid fever is endemic, with an estimated incidence rate of 100-200 cases per 100,000 population (Osu et al., 2024). Typhoid burden varies widely across Nigeria with documented prevalence as low as 0.071% in Oyo State and as high as 47.1% in Osun State (Magaji et al, 2025). Data from the Department of Public Health of the Federal Ministry of Health shows that typhoid is a public health concern due to its frequent diagnosis within healthcare facilities in Nigeria (Faruku et al., 2024). Evidence from Northern Nigeria revealed an alarming prevalence rate of typhoid, including a 15.2% occurrence among patients presenting with fever in some of the health facilities in the region (Musa et al., 2022). Comparable trends of typhoid incidence across the region underscore the strong association of typhoid fever with socioeconomic deprivation, limited availability of safe drinking water and inadequate sanitation (Magaji and Mahmud, 2025).

In untreated typhoid infection, mortality rate can be as high as 26% (Khanam *et al.*, 2022; Wain *et al.,* 2015). Intestinal perforation remains the most severe complication associated with the disease, occurring in roughly 7.6% of patients and leading to mortality rates approaching 80% of the affected patients (Kim *et al.,* 2022). Typhoid fever also impairs the functions of other organs in the body such as liver and kidney (Rajput et al., 2016). Chronic infection can affect the gallbladder resulting in gallstone formation (Crawford *et al*., 2010). This retrospective assessment conducted on confirmed typhoid patients who attended Bingham University Teaching Hospital, Jos, Plateau State, between 2019 and 2020 and tested positive to widal test and blood culture test for *Salmonella typhi* was aimed to evaluate the effects of typhoid fever on haematological, hepatic and renal function indices in both male and female patients and potential gender-based variations in these parameters to help in preventing organ-related complications associated with typhoid infection among patient

MATERIALS AND METHODS

Study Area and Population

Records of all patients who visited Bingham University Teaching Hospital, Jos, Plateau State, between 2019 and 2020 for typhoid fever treatment who had complete biochemical

data required for the study were included, their records were retrieved from the hospital records following ethical clearance. All personal identifiers, including patients' names and contact details, were excluded to ensure confidentiality.

Inclusion and Exclusion Criteria

The study involved patients who attended Bingham University Teaching Hospital for typhoid treatment from 2019 to 2020 who tested positive to widal test and blood cultures confirmed *Salmonella typhi* infection regardless of age and gender. The control group comprised healthy individuals whose blood samples tested negative for *S. typhi*. Patients with existing medical conditions that could influence the study such as patients with liver or kidney disorders, diabetes mellitus, haematological abnormalities and AIDS were excluded, even if they had a positive typhoid result.

The record of 300 patients comprising of 150 typhoid fever patients and 150 healthy control subjects were used.

Ethical Approval and Authorization

This retrospective study was carried out in accordance with the guidelines for the human study in retrospective research provided by Bingham University Karu, Nasarawa state. The protocol was approved by the University ethical Committee (BHU/FST/H20-00040) and authorization for the study was granted by the hospital management prior to data collection. To protect participants' privacy, identification codes were used, and all personal details were omitted.

Data Collection and Analysis

Patients' parameters were extracted from patient's record in the hospital laboratory and normalised. It was analysed with GraphPad Prism version 6. Comparisons between typhoid patients and the control group were done using the t-test ($p < 0.05$). Variations in measured parameters by sex were analysed by ANOVA (one-way) followed by Tukey's post hoc multiple comparison test.

RESULTS AND DISCUSSION

Evaluation of patients' data revealed that typhoid fever led to a significant ($P < 0.05$) decrease in haemoglobin, packed cell volume (PCV) and mean cell haemoglobin concentration (MCHC) compared to the healthy control group (Table 1), this signifies anaemia among the infected patients. The significant reduction ($P < 0.05$) in anaemia-related indices was more pronounced in male than in female patients (Table 2), indicating that *Salmonella typhi*-associated anaemia was more severe among the male patients. A significant ($P < 0.05$) decrease was also observed in total white blood cell count, eosinophils, neutrophils and platelets in infected individuals compared to controls (Table 1) whereas lymphocyte levels were significantly ($P < 0.05$) elevated in the typhoid patient (Table 1). However, there was no significant difference in these parameters between male and female typhoid patients (Table 2). In addition, typhoid infection resulted in significant ($p<0.05$) decrease in platelets count among the patients with the reduction in platelets counts more pronounced among male patients (Tables 2).

Table 1: Haematological Indices of Control and Typhoid Fever Group

Indices	Control	Typhoid Fever
PCV (%)	40.55 ± 3.81^{a}	29.51 ± 2.94^{b}
HAEMOGLOBIN(g/dl)	13.75 ± 1.24^{a}	8.74 ± 1.52^{b}
MCHC (g/dl)	34.12 ± 1.41^{a}	29.53 ± 1.45^{b}
TOTAL WBC ($X10^{9}/l$)	7.29 ± 1.68^{a}	1.75 ± 0.77^{b}
NEUTROPHIL (%)	58.53 ± 2.62^{a}	44.53 ± 3.04^{b}
LYMPHOCYTES (%)	19.77 ± 1.74^{b}	27.51 ± 1.56^{a}
MONOCYTES (%)	4.21 ± 2.73^{a}	0.03 ± 0.18^{b}
EOSINOPHILS (%)	1.58 ± 0.69^{a}	0.11 ± 0.31^{b}
BASOPHILS (%)	0.41 ± 0.49^{a}	0.01 ± 0.08^{b}
Platelet($x10^{3}/\mu l$)	200.55 ± 64.58^{b}	125.34 ± 45.55^{a}

The data are presented as the mean ± standard deviation for 150 samples

Values with different superscript within the same row differ significantly ($p<0.05$)

Table 2: Analysis of Gender Variation of Haematological Indices of Control and Typhoid Fever Group

Parameters	Control		Patients	
	Female	Male	Female	Male
PCV (%)	38.79 ± 2.33^{b}	42.79 ± 4.17^{c}	30.05 ± 2.83^{a}	28.75 ± 2.97^{a}
Haemoglobin(g/dl)	13.18 ± 0.94^{c}	14.50 ± 1.18^{d}	8.76 ± 1.40^{b}	6.71 ± 1.69^{a}
MCHC(g/dl)	34.32 ± 1.42^{a}	33.86 ± 1.37^{a}	29.59 ± 1.31^{b}	26.45 ± 1.69^{c}
White blood cells ($10^{9}/\mu l$)	7.28 ± 1.62^{b}	7.30 ± 1.75^{b}	1.80 ± 0.79^{a}	1.69 ± 0.74^{a}
Neutrophil (%)	58.00 ± 2.60^{b}	59.00 ± 2.60^{b}	45.00 ± 2.90^{a}	44.00 ± 3.20^{a}
Lymphocyte (%)	19.8 ± 1.64^{b}	19.8 ± 1.89^{b}	27.6 ± 1.54^{a}	27.4 ± 1.59^{a}
Monocyte (%)	3.93 ± 2.18^{c}	4.58 ± 3.30^{d}	0.02 ± 0.15^{a}	0.05 ± 0.22^{b}
Eosinophil (%)	1.59 ± 0.73^{b}	1.57 ± 0.64^{b}	0.10 ± 0.31^{a}	0.11 ± 0.32^{a}
Basophil (%)	0.47 ± 0.50^{d}	0.34 ± 0.48^{c}	0.01 ± 0.11^{b}	0.00 ± 0.00^{a}
Platelet($x10^{3}/\mu L$)	193.00 ± 50.56^{c}	210.00 ± 75.56^{d}	140.93 ± 40.35^{b}	115.93 ± 55.35^{a}

The data are presented as the mean ± standard deviation. Control female=85, control male=65; Patient female=88; Patient male=62

Values with different superscript within the same row differ significantly ($p<0.05$)

The results on hepatic function parameters demonstrated a direct association between hepatic dysfunction and typhoid infection (Table 3). Serum concentrations of liver function indicators were elevated significantly ($P < 0.05$) in typhoid patients compare to the control group (Table 3). However, gender-based association of the parameters showed that the

increase is more significant among the male patients compare to the female patients (Table 4). Additionally, serum glucose levels were significantly reduced in individuals with typhoid fever relative to healthy controls, although no significant variation was observed between male and female patients (Table 4). Although serum albumin and total protein levels showed a non-significant decrease overall, gender-specific analysis revealed a significant increase in serum albumin among female patients in contrast to male patients that exhibited a significant decrease in serum albumin.

Table 3: Liver Function Parameters of Typhoid Fever Patients and Control

Parameters	Control	Typhoid Fever
AST (U/I)	22.95 ± 5.63^{a}	66.34 ± 23.46^{b}
ALT (U/I)	28.23 ± 5.78^{a}	60.23 ± 14.03^{b}
ALP (U/I)	91.66 ± 21.85^{a}	122.00 ± 35.50^{b}
Total protein (g/dl)	7.19 ± 0.73^{a}	6.91 ± 2.27^{a}
Albumin (g/dl)	4.35 ± 1.03^{a}	4.31 ± 0.75^{a}
Total bilirubin (mg/dl)	1.00 ± 0.23^{a}	2.59 ± 1.05^{b}
Direct bilirubin (mg/dl)	0.13 ± 0.05^{a}	1.36 ± 0.67^{b}
GLUCOSE (mmol/l)	5.32 ± 0.57^{b}	2.76 ± 0.98^{a}

The data are presented as the mean ± standard deviation for 150 samples
Values with different superscript within the same row differ significantly ($p<0.05$)

Table 4: Effect of Gender Variation on Liver Function Indices of the Control and Typhoid Fever Group

PARAMETERS	CONTROL		PATIENTS	
	FEMALE	MALE	FEMALE	MALE
AST (IU/L)	22.96 ± 5.98^{a}	22.85 ± 5.38^{a}	55.50 ± 22.17^{b}	81.71 ± 15.29^{c}
ALT (IU/L)	28.59 ± 6.21^{a}	27.75 ± 5.17^{a}	57.44 ± 16.60^{b}	64.20 ± 7.79^{c}
ALP (IU/L)	87.94 ± 18.53^{a}	96.52 ± 24.87^{b}	115.60 ± 34.43^{c}	131.10 ± 35.28^{d}
Protein(g/dL)	7.22 ± 0.74^{b}	7.17 ± 0.72^{b}	6.80 ± 1.3^{a}	7.06 ± 1.17^{b}
Albumin(g/dL)	4.45 ± 0.68^{b}	4.13 ± 0.80^{a}	5.12 ± 0.93^{c}	3.82 ± 0.72^{a}
Total Bilirubin(mg/dl)	1.02 ± 0.24^{a}	0.96 ± 0.22^{a}	2.46 ± 1.21^{b}	2.78 ± 0.73^{c}
Direct Bilirubin(mg/dl)	0.13 ± 0.05^{a}	0.14 ± 0.05^{a}	1.47 ± 0.78^{c}	1.19 ± 0.42^{b}
Glucose (mmol/l)	5.30 ± 0.60^{a}	5.34 ± 0.53^{a}	2.73 ± 1.05^{b}	2.79 ± 0.92^{c}

The data are presented as the mean ± standard deviation. Control female=85, control male=65; patient female=88; patient male=62
Values with different superscript within the same row differ significantly ($p<0.05$)

No significant difference was observed in the kidney function indices of the patients and the control except the serum Urea (Tables 5&6). This probably indicated that typhoid fever infection does not have adverse effect on the renal function of the patients.

Table 5: Renal Function Indices of Typhoid Fever Patients and Control

Parameters	Typhoid Fever	Control
UREA (mmol/l)	4.56 ± 1.01^{a}	3.79 ± 1.20^{a}
CREATININE (mg/dl)	0.74 ± 0.14^{a}	0.74 ± 0.13^{a}
SODIUM (mmol/l)	139.59 ± 2.59^{a}	138.83 ± 2.5^{a}
POTASSIUM (mmol/l)	3.92 ± 0.81^{a}	3.98 ± 0.76^{a}
CHLORIDE (mmol/l)	100.78 ± 3.80^{a}	100.61 ± 3.85^{a}

The data are presented as the mean ± standard deviation for 150 samples
Values with different superscript within the same row differ significantly ($p<0.05$)

Table 6: Effect of Gender Variation on Renal Function Parameters of Typhoid Fever Patients and Control

PARAMETERS	CONTROL		PATIENTS	
	FEMALE	MALE	FEMALE	MALE
Urea (mmol/L)	4.53 ± 1.03^{b}	4.60 ± 0.98^{b}	3.88 ± 1.21^{a}	3.66 ± 1.17^{a}
Creatinine (mg/dl)	0.75 ± 0.13^{a}	0.73 ± 0.15^{a}	0.74 ± 0.1254^{a}	0.75 ± 0.14^{a}
Sodium (mmol/L)	138.70 ± 2.43^{a}	139.0 ± 2.51^{a}	139.6 ± 2.75^{a}	139.5 ± 2.37^{a}
Potassium (mmol/L)	4.09 ± 0.70^{a}	3.83 ± 0.80^{a}	3.97 ± 0.81^{a}	3.86 ± 0.82^{a}
Chloride (mmol/L)	100.60 ± 4.03^{a}	100.60 ± 3.65^{a}	101.00 ± 3.74^{a}	100.50 ± 3.90^{a}

The data are presented as the mean ± standard deviation. Control female=85, control male=65; patient female=88; patient male=62
Values with different superscript within the same row differ significantly ($p<0.05$)

Discussion

The observed significant ($P < 0.05$) reductions in PCV, MCHC, and haemoglobin (Hb) among the patients compared with controls suggest the presence of anaemia in individuals with typhoid fever. Typhoid infection usually results in the direct interaction between the *Salmonella typhi* or their toxins

with host haematopoietic tissues, such interactions can interfere with the function of blood-forming organs like the bone marrow, decrease haemoglobin synthesis, thereby impairing haematopoiesis (Muhammad *et al.,* 2021; Ndako *et al*., 2020). In addition, bacterial toxins and metabolites interact with erythrocyte membrane proteins, promoting further destruction of erythrocytes leading to an anaemic state of the patients (Muhammad *et al.,* 2021; Ndako *et al.,* 2020). Severe anaemia in typhoid fever patients might have also resulted from intestinal haemorrhage (Ndako *et al.,* 2020). Similar findings of anaemia among typhoid-infected individuals have been recorded by Kakaria *et al.,* (2014) and Ndako *et al.,* (2020).

A significant ($p < 0.05$) decline in total white blood cell count and differential white blood cells counts, except lymphocytes, was observed among the patients, consistent with the findings of Kakaria *et al*., (2014). However, no significant gender-based variation was detected in these typhoid-related induced changes in these parameters. These changes may be attributed to haemophagocytosis and suppression of bone marrow activity which are the major attacking mechanism of *Salmonella typhi* in typhoid fever patients (Ndako et al., 2020). Platelet counts were also significantly reduced in infected individuals, with the reduction being more pronounced ($p < 0.05$) in male patients. Bone marrow in typhoid fever patients often exhibits myeloid maturation arrest, a decrease in erythroblasts and megakaryocytes, and increased histiocytic phagocytic activity (Khosla *et al.,* 1995). *S. typhi* is known to impair bone marrow function and induce haemophagocytosis, both of which contribute to haematological abnormalities (Ringoringo *et al.,* 2022; Shrivastava *et al.,* 2015). The thrombocytopenia observed might have resulted from reduced platelet production due to bone marrow suppression during acute infection or their increased destruction by an enlarged spleen (Anusuya *et al.,* 2015). These haematological alterations are further supported by the finding of Lokhandwala *et al.,* (2012) and Qamar & Aijaz, (2013) who reported a positive correlation between bacteraemia and blood haematological parameter alteration among typhoid patients. Lymphocytosis accompanied by neutropenia is recognized as an indicator of typhoid complications (Ifeanyi, 2014). The elevated lymphocyte counts alongside significant decrease in neutrophil levels observed in this study may therefore reflect disease complications among the male patients examined.

Evaluation of clinical parameters in typhoid patients indicated a direct correlation between elevated serum liver function markers and typhoid infection. Hepatomegaly and impaired liver function are frequently reported among individuals with typhoid fever, with increased liver enzyme activity correlating significantly with disease severity (Khosla *et al.,* 1988; Morgenstern & Hayes, 1991; Ratnayake *et al.,* 2011). The increase in hepatic biomarkers reflects liver dysfunction (Khosla *et al.,* 1988). Such biochemical alterations could result from *Salmonella* invasion of hepatic tissue or the accumulation of bacterial endotoxins causing hepatocellular damage. In addition, during infection, *Salmonella typhi* migrate from the intestinal lumen to the liver (Küpffer cells) where it multiplies and spreads through the bloodstream into the other part of the body (de Andrade & de Andrade Júnior, 2003). The lysis of *Salmonella typhi* releases toxic lipopolysaccharides which activate macrophages in the liver's Küpffer cells to secret cytokines which induce the production of ROS that destroy Salmonella typhi but also destroy the hepatocytes (de Andrade & de Andrade Júnior, 2003; Natheua *et al.,*2020).The ROS produced during infection would be proportional to the toxic lipopolysaccharides released by *Salmonella typhi* during the lysis which will correlate positively with the elevation of hepatic biomarkers released by the liver into the serum as the duration of the disease progresses (Ahmed & Ahmed, 2010). In addition, bacterial proliferation within hepatocytes may trigger cytokine production and release, contributing to further hepatic injury (Morgenstern & Hayes, 1991). The significant increase in serum bilirubin simultaneously with significant elevation of alkaline phosphatase and transaminases indicated the disturbance in liver secretion of bilirubin in typhoid fever patients and less due to haemolysis (Ahmed & Ahmed, 2010). The elevated serum ALP and transaminases indicated the involvement of hepatobiliary system which further confirm typhoid induced hepatic dysfunction in the patients (Haleem *et al.,* 2017; Khosla *et al.,* 1988; Vaishnavi *et al.,* 2005). Serum albumin and total protein levels showed a non-significant decrease overall. Reduction in albumin may be as a result of typhoid induced hepatic damage since albumin biosynthesis occurs mainly in the hepatocytes. Destruction of hepatocyte leading to the release of contents will also leads to the loss of albumin-synthesizing enzymes. This might have resulted in decrease in albumin synthesis due to liver dysfunction. Significant decrease in serum albumin observed among male patients further confirmed that the typhoid fever has more adverse effect on the male hepatic function than the female patients. The decrease in total protein may be attributed it to enteric protein loss as a result of disruption in the integrity of the intestinal mucosa. Decrease in serum total protein concentration in patients is similar to the finding of (Ndukaku *et al.,* (2015) and Shamin *et al.,* (2012). Decrease in serum proteins may also be due to large vascular leakage of plasma proteins owing to raised capillary permeability, reduced protein intestinal absorption due to low ingestion, albumin clearance in urine, elevated protein catabolism, and diminished liver synthesis due to hepatic damage (Shamim et al., 2012).

There was a significant reduction in serum glucose of the typhoid fever patients compare to the healthy control. The reduction has been associated with direct consumption of the host glucose and modulation of host glycolysis in macrophages by *S. Typhi* as observed by other studies (Haleem *et al.,* 2017; Morgenstern & Hayes, 1991; Ratnayake et al., 2011). Wang *et al.,* (2021) have shown that S. typhi suppresses glycolysis and hexokinase gene expression in human macrophages while simultaneously increasing the glucose uptake by the human macrophages. This greatly reduced the amount of glucose undergoing glycolysis despite the increase in glucose uptake by the human macrophages. The accumulated glucose is use by the bacterial for systemic infection, *S. typhi* intracellular replication, inhibition of host immune responses in favour of the bacterial survival and activation of its virulence factors. This will result in low blood glucose level in typhoid fever patients.

Creatinine is a by-product of muscle metabolism excreted by the kidneys. Creatinine serum concentrations are regulated and maintained by continued production and urinary excretion, a simultaneous increase in concentrations of serum creatinine and urea indicates renal failure (Wankasi et al.,2022). Evaluation of the patients' results indicated no significant difference in renal function parameters between typhoid patients and the control group, except for serum urea levels. This finding is consistence with previous study that showed that simultaneous hepatic and renal dysfunction in typhoid fever is rare and occurs in only a small proportion of cases (Hayashi et al., 2005; Khan et al., 1998; Pancharoen *et al.,* 2001; Rajput *et al.,* 2016; Seratin *et al.,* 2022) but contrary to the findings of Janssen van Doorn et al., (2006) and

Pancharoen *et al.,* (2001) reported involvement of kidney. Renal dysfunction associated with typhoid infection is typically acute, transient, and reversible mostly linked to multiple mechanisms such as immune complex deposition, toxin-mediated injury, and, in some cases, direct bacterial invasion of renal tissue by *Salmonella typhi* (Hayashi *et al.,* 2005; Khan *et al.,* 2009; Leung *et al.,* 2012). Increase in serum urea without corresponding increase in creatinine may be due to reduced renal blood flow, secondary to decreased plasma volume during dehydration that impair glomerular filtration rate, leading to a transient accumulation of these nitrogenous waste products in the circulation and not actual typhoid induced renal damage (Atata *et al*., 2025).

Significant alterations in anaemia-related indicators were more severe in male patients compared to females. In males, the cytokine response to intestinal infection is more pro-inflammatory compared to females (Al-Dahhan *et al.,* 2020). This stronger inflammatory reaction may lead to more extensive tissue damage in the Peyer's patches (lymphoid tissues in the intestines), which in turn can lead to more significant internal bleeding or occult blood loss in stools, contributing to greater anaemia (Al-Dahhan *et al.,* 2020). Also, men generally have higher iron stores (ferritin levels) than women. During an acute infection, the body sequesters iron to limit its availability to bacteria, often termed "nutritional immunity (Ganz,2009). Differences in the regulation of iron-regulating hormones like hepcidin between men and women could potentially play a role in how this iron sequestration impacts red blood cell production. During *S. Typhi* infection, hepcidin is strongly upregulated leading to hypoferremia and reduction in red blood cell production, hence the anaemia observed (Darton *et al.,* 2015).

A significant thrombocytopenia observed in male patients compared to female suggest that platelets' activation could be a major factor in typhoid infection in male when antibody level rises. Platelets have been reported to be activated by bacteria and soluble chemicals such as toxins (Ndako *et al.,* 2020). Once activated, it undergoes viscous metamorphosis that leads to intravascular thrombus formation which is a prelude to disseminated intravascular coagulation (Ndako *et al.,* 2020). The decrease in platelet count observed in male patients could also be due to decreased production of platelets by the bone marrow during acute infection or in part by their increased destruction by an enlarged spleen (Anusuya *et al.,* 2015).

Gender-specific analysis revealed a significant increase in serum albumin among female patients in contrast to male patients that exhibited a significant decrease in serum albumin. It is important to note that most studies actually report *decreased* or *normal* serum albumin levels in typhoid fever, making significant elevation a less common presentation.

The body does not produce excess albumin in response to physiologic stimuli; albumin synthesis is tightly regulated and does not increase beyond what is needed to maintain normal pools (Soeters et al., 2019). Serum albumin concentration was reported to be lower during infection (Shamim et al., 2012). Dehydration is the most common cause of increased serum albumin levels, resulting in haemoconcentration that artificially elevates albumin concentration in the blood. The unusual *elevation* in serum albumin in female typhoid patients may primarily be attributed to dehydration (haemoconcentration), which concentrates existing albumin in a reduced plasma volume. Typhoid fever is often associated with symptoms like prolonged fever, vomiting, and diarrhoea, leading to significant fluid and electrolyte loss. This loss of plasma water reduces the total blood volume, which in turn concentrates the existing albumin, causing a measured increase in its serum level. This is a relative, not absolute increase in albumin (Ohta et al., 2024). Patients with diabetes may have higher rates of hyperalbuminemia, possibly related to osmotic diuresis from hyperglycaemia (Ohta et al., 2024). Inflammation typically suppresses albumin synthesis (leading to low albumin), In the very acute phase of an infection, there might be a temporary or relative increase in albumin levels before the typical hypoalbuminemia response occurs which might be what happen to the female patients (Don et al.,2004). The magnitude of the increase in albumin among the female patients observed in our study likely reflects the degree of dehydration and not actual increase in response to the infection.

CONCLUSION

Analysis of the data of the typhoid patients treated at Bingham University Teaching Hospital, Jos, Plateau State, between 2019 and 2020, showed that typhoid fever in this cohort was strongly associated with significant haematological disturbances and hepatic dysfunction, with notable gender variations. Gender-based comparisons revealed that both hepatic and haematological alterations were more severe in male patients than the females. Conversely, renal function was largely preserved. It is advisable that individuals with recurrent typhoid fever infection especially, the male patients in this area undergo liver and kidney function assessments in addition to their treatment to facilitate early detection of typhoid-induced hepatic and renal damage.

Limitation of the Study

This study is a retrospective study that examine the record of the patients who attended Bingham University Teaching Hospital for typhoid treatment from 2019 to 2020 who tested positive to widal test and *Salmonella typhi* infection confirmed by blood cultures; with healthy individuals whose blood samples tested negative for *S. typhi* according to the laboratory records serving as control. The test for the confirmation of typhoid patients was performed by the laboratory staff using their laboratory procedure. Human error from the laboratory staff may not be completely rules out, also non-disclosure of the complete health status by some patients whose records were examined is also possible.

Abbreviations

AST: Aspartate aminotransferase; **ALT:** Alanine aminotransferase; **ALP:** Alkaline phosphatase; **PCV:** Packed cell volume; **MCHC:** Mean corpuscular haemoglobin concentration; **RBC:** Red blood cell; **WBC:** White blood cell;

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