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*Original Article*

# Evaluation of Fibrinogen, Haemoglobin and Platelet Indices in Patients with Alzheimer's Disease in Imo State

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**ABSTRACT:** *Finding reliable, less intrusive biomarkers for the early diagnosis and monitoring of Alzheimer's disease (AD) is still a big scientific problem. This study assessed fibrinogen, haemoglobin, and platelet indices in patients with Alzheimer's disease at Imo Specialist Hospital, Owerri, Nigeria. A total of 60 volunteers were included, consisting of 30 clinically confirmed Alzheimer's disease patients and 30 age-matched ostensibly healthy controls. We took 7 mL of blood from each person: 2 mL went into EDTA bottles for blood tests and 5 mL went into plain bottles for serum separation. The samples were spun in a centrifuge at 3,000 rpm for 5 minutes, and the sera were kept at −20°C until they were ready to be looked at. We used the clot-based approach to find fibrinogen, the cyanmethemoglobin method to get haemoglobin, and an automated haematology analyser to find platelet indices. We used SPSS version 27 to look at the data. We found the mean values, standard deviations, Student's t-test, Pearson correlation, and p-values.Mean haemoglobin (10.33 ± 1.39 g/dL), platelet count (178.77 ± 47.46 ×10⁹/L), PDW (16.00 ± 3.22%), and PCT (0.17 ± 0.05%) were significantly lower in Alzheimer's patients compared with controls (12.79 ± 0.94 g/dL, 307.63 ± 87.76 ×10⁹/L, 18.75 ± 4.41%, and 0.24 ± 0.07%, respectively; $p < 0.05$). On the other hand, MPV (9.25 ± 1.06 fL), P-LCR (20.57 ± 6.79%), and fibrinogen (472.60 ± 91.59 mg/dL) were much greater in AD patients than in controls ($p < 0.05$). There were no significant differences in any parameter according to age or gender. Haemoglobin had substantial positive relationships with platelet count and PCT, and a significant negative link with fibrinogen. Alzheimer's disease is linked to anaemia, platelet irregularities, and increased fibrinogen levels, indicating that hypoxia, platelet activation, and inflammation are significant factors in the illness's aetiology. These metrics may function as accessible biomarkers for the surveillance of Alzheimer's disease.*

**KEYWORDS:** *Fibrinogen, Haemoglobin, Platelet indices, Alzheimer's disease.*

## 1. INTRODUCTION

Alzheimer's disease (AD) is a progressive, irreversible neurological disorder and the most prevalent form of dementia globally, currently impacting over 35 million individuals. As life expectancy around the world goes up, the burden of the disease is growing. This makes AD one of the biggest public health problems of the twenty-first century. Clinically, Alzheimer's disease is marked by a progressive and insidious deterioration in memory, cognition, language, and executive skills, ultimately resulting in significant functional impairment and the loss of independence. These deficiencies result from gradual neuronal degeneration, synaptic impairment, and extensive cortical and subcortical brain injury. The initial and most significant clinical signs usually encompass episodic memory deficits and spatial disorientation, indicating early engagement of the hippocampus and related medial temporal lobe structures [2].

As Alzheimer's disease becomes worse, people have more and more trouble with language, math, learning, and making decisions. Eventually, they can't even do simple things like getting dressed or bathing. Together with decreasing cognitive function, there are behavioural and psychological symptoms including depression, anxiety, agitation, and apathy. These things all make life a lot more difficult for carers and reduce the person's quality of life. There is high prevalence of nutritional abnormalities in Alzheimer's Disease with potentially significant morbidity and mortality. Agnosia and apraxia prevent the patient from recognising food and coordinating the movements that are needed to eat, resulting in low calorie intake and progressive weight loss [3]. Furthermore, the dysphagia and anorexia secondary to mesial temporal cortex degeneration aggravate eating behavior [4]. Although weight loss is most frequent in patients, a subgroup of cases present hyperphagia and weight gain showing an abnormality of hypothalamic and limbic pathways controlling food and energy homeostasis [5]. These metabolic dysfunctions highlight the multisystem nature of AD.

Pathologically, Alzheimer's disease is defined by the extracellular deposition of Aβ plaques and intracellular aggregation of neurofibrillary tangles (NFTs) composed of hyperphosphorylated tau protein leading to neuronal transmission impairment and early stage initiation of neurodegeneration [6]. These characteristic lesions lead to synaptic disarray, death of neurons and ultimately reduced brain size. However, beyond these classical neuropathological features, an emerging body of evidence

indicates that systemic features including haematologic, inflammatory and vascular abnormalities are important players in illness pathogenesis and propagation. All of the above, including chronic inflammation, oxidative stress and endothelial dysfunction leading to impaired cerebral perfusion are known to drive the worsening of neuronal damage in Alzheimer's disease [7].

Haemoglobin is highly significant for transporting oxygen and maintaining the brain well-infused with an ample supply of oxygen. The brain is particularly sensitive to hypoxia because of its high metabolic demands and limited capacity for anaerobic metabolism. Decreased haemoglobin levels interfere with oxygen delivery to neurones, resulting in mitochondrial dysfunction, production of oxidative stress and subsequent apoptosis of nerve cells[8]. There have been several studies that have corroborated the association of Oxygen Transport Related Measures Reduced in dementia There is a significant association between decreased measures of haemoglobin leading to anaemia and cognition loss. Repetitive hypoperfusion of the brain-chronic cerebral hypoxia-(from anaemia) may promote amyloid-β deposition, tau phosphorylation and neuroinflammation leading to exacerbate disease progression. Second, erythropoietin - which regulates production of red blood cells- has been shown to have neuroprotective properties suggesting that disturbances in haemoglobin regulation might directly affect neuronal survival.

Platelets have also been found to play a major role in the pathophysiology of AD. They are enriched for amyloid precursor protein (APP) and the enzymes required for its amyloidogenic processing, and serve as a major provider of amyloid-β [10]. Activated platelets release pro-inflammatory and pro-thrombotic compounds known to be injurious to the endothelium, capable of inducing microvascular thrombosis, which may impede blood flow in the brain. The mean platelet volume (MPV), platelet distribution width (PDW), [the] plateletcrit (PCT) and platelet-large cell ratio (P-LCR) are among the most important tests in estimating activated level of platelets, reactivity[11]. The abnormalities are these markers reflect enhanced platelet and inflammation activity, known to be closely related with the vascular and inflammatory component of AD.

Fibrinogen, a major liver-produced coagulation protein, is also incredibly relevant in AD.The blood–brain barrier usually keeps fibrinogen out of the brain. But with AD, problems with blood vessels and inflammation make the barrier more permeable, letting fibrinogen into the central nervous system. Fibrinogen can attach to amyloid-β in the brain to make fibrin clots that are hard to break up. These clots make cerebral microcirculation worse and cause persistent neuroinflammation[12]. These unusual clots make it much harder for neurones to get oxygen and nutrients, which leads to a cycle of hypoxia, inflammation, and neurodegeneration. Although there is more and more evidence from throughout the world that links haemoglobin, platelet indices, and fibrinogen to Alzheimer's disease, there is still not much data from Nigerian communities. Genetic, environmental, dietary, and healthcare disparities may affect the expression and clinical significance of these biomarkers. Consequently, assessing these measures in Alzheimer's patients in Imo State is crucial for enhancing the comprehension of disease mechanisms within this population and for investigating their possible diagnostic and prognostic significance.

## 2. MATERIALS AND METHODS

### *2.1. STUDY AREA*

This study was conducted at Imo Specialist Hospital, Owerri, Imo State, Nigeria. The hospital is located along Port Harcourt Road, Umuguma, Owerri. Owerri has a land area of approximately 184 km² and a population of 195,652 according to the 2006 national census. The region has a tropical climate with two major seasons: the rainy season (March–September) and the dry season (October–March). The average annual temperature ranges from 22.5°C to 35.5°C, with relative humidity of approximately 74.3% and average annual rainfall of 240.6 mm. The major occupations of residents include trading, farming, and civil service.

### *2.2. STUDY DESIGN*

A hospital-based cross-sectional study was conducted between February and August 2024. The study population consisted of 30 clinically diagnosed Alzheimer’s disease patients and 30 apparently healthy age-matched non-Alzheimer individuals who served as controls. All laboratory analyses were performed at De-Rhema Diagnostics, Owerri. Data were analyzed using SPSS version 27.

### *2.3. METHOD OF RECRUITMENT*

A total of sixty participants (30 AD patients and 30 controls) were recruited. All participants were provided with detailed information about the study, and written informed consent was obtained. A structured questionnaire was administered to collect demographic and clinical information.

#### *2.3.1. SELECTION CRITERIA*

Inclusion Criteria

Clinically diagnosed Alzheimer’s disease patients aged 18 years and above.

Alzheimer’s patients without co-existing infections such as HIV, hepatitis B, hepatitis C, or syphilis

Participants who provided informed consent.
Age-matched apparently healthy non-Alzheimer individuals.

Exclusion Criteria
Alzheimer's patients below 18 years of age.
Individuals who declined or could not provide informed consent.
Participants with HIV, HBsAg, HCV, or syphilis infection.

### *2.4. SAMPLE COLLECTION*
Seven milliliters of venous blood were collected aseptically from the antecubital vein of each participant. Two milliliters were dispensed into an EDTA container for hematological analysis, while five milliliters were dispensed into a plain container for serum separation. Samples were properly labeled and stored at 4°C (EDTA) and −20°C (serum) prior to analysis.

#### *2.4.1. LABORATORY ANALYSIS*
Determination of Fibrinogen
Fibrinogen concentration was determined using the clot-based method described by Mosesson (2015). The principle is based on the conversion of fibrinogen to fibrin by thrombin, followed by colorimetric quantification. Absorbance was measured at 570 nm, and fibrinogen concentration was calculated using Beer's law.

Determination of Hemoglobin
Hemoglobin concentration was measured using the cyanmethemoglobin method. Whole blood was reacted with Drabkin's reagent to form cyanmethemoglobin, and absorbance was read at 540 nm using a spectrophotometer. Hemoglobin concentration was calculated relative to a standard.

Determination of Platelet Indices
Platelet count and platelet indices (MPV, PDW, PCT, and P-LCR) were determined using a Sysmex KX-21N automated hematology analyzer based on the electrical impedance principle.

Statistical Analysis
Data were analyzed using SPSS version 27. Results were expressed as mean ± standard deviation. Student's t-test was used to compare means between groups, while Pearson correlation analysis assessed relationships between variables. Statistical significance was set at $p < 0.05$.

## 3. RESULTS
### *3.1.MEAN VALUES OF HAEMOGLOBIN, PLATELET COUNT, PDW, MPV, PCT, PLCR AND FIBRINOGEN IN ALZHEIMER PATIENTS VERSUS CONTROLS*
The mean values of haemoglobin (10.33±1.39)g/dl, platelet (178.77±47.46) x $10^9$/L, PDW (16.00±3.22)%, and PCT (0.17±0.05)% were significantly reduced in Alzheimer patients when compared to the controls (12.79±0.94)g/dl, (307.63±87.76)x$10^9$/L,(18.75±4.41)%and(0.24±0.07)%.(t=8.02,p=0.000;t=7.07,p=0.000;t=2.76,p=0.000 and t=4.73,p=0.000).
The mean values of MPV (9.25±1.06)fl, PLCR (20.57±6.79)% and fibrinogen (472.60±91.59) mg/dl were significantly increased (p=0.037, p=0.052 and p=0.000) respectively in Alzheimer patients when compared to the control (8.72±0.84)fl, (0.24±0.07)% and (240.60±65.17)mg/dl.(t=2.14,p=0.037; t=1.98, p=0.052 and t=11.31,p=0.000)

**TABLE 1 Mean values of haemoglobin, platelet count, PDW, MPV, PCT, PLCR and fibrinogen in alzheimer patients versus control (Mean±S.D)**

| Parameters | Test n=30 | Control n=30 | t-value | p-value |
|---|---|---|---|---|
| Hb (g/dl) | 10.33±1.39 | 12.79±0.94 | 8.02 | 0.000* |
| Platelet x$10^9$/L | 178.77±47.46 | 307.63±87.76 | 7.07 | 0.000* |
| PDW (%) | 16.00±3.22 | 18.75±4.41 | 2.76 | 0.000* |
| MPV (fl) | 9.25±1.06 | 8.72±0.84 | 2.14 | 0.037 |
| PCT (%) | 0.17±0.05 | 0.24±0.07 | 4.73 | 0.000* |
| PLCR (%) | 20.57±6.79 | 17.34±5.76 | 1.98 | 0.052 |
| Fibrinogen (mg/dl) | 472.60±91.59 | 240.60±65.17 | 11.31 | 0.000* |

**Key:**
Hb = Haemoglobin
PDW = Platelet Distribution Width
MPV = Mean Platelet Volume
PCT = Plateletcret

P-LCR = Platelet-Large Cell Ratio
*: Significant p-value

### *3.2. COMPARISON OF THE MEAN VALUES OF HAEMOGLOBIN, PLATELET COUNT, PDW, MPV, PCT, PLCR and FIBRINOGEN IN ALZHEIMER PATIENTS BASED ON GENDER*

There was no significant difference in the mean values of haemoglobin, platelet, PDW, MPV, PCT, PLCR and fibrinogen in male Alzheimer patients (11.23±1.27)g/dl, (178.09±50.41) x$10^9$/L, (15.42±1.91)%, (9.26±1.34)fl, (0.16±0.06)%, (18.53±6.85)% and (432.72±100.32) mg/dl when compared to female Alzheimer patients (10.81±1.19)g/dl, (179.16±47.09) x$10^9$/L, (16.34±3.79)%, (9.24±0.89)fl, (0.17±0.04)%, (21.75±6.64)% and (495.68±79.93)mg/dl. (t=3.07,p=0.105; t=0.06 p=0.954;t=0.75,t=0.75, p=0.461; t=0.05,p=0.958; t=0.89, p=0.377; t=1.27, p=0.215 and t=1.89,p=0.069)

**TABLE 2 Comparison of the mean values of haemoglobin, platelet count, PDW, MPV, PCT, PLCR and fibrinogen in alzheimer patients based on gender**

| Parameters | Male n=11 | Female n=19 | t-value | p-value |
|---|---|---|---|---|
| Hb (g/dl) | 11.23±1.27 | 10.81±1.19 | 3.07 | 0.105 |
| Platelet x$10^9$/L | 178.09±50.41 | 179.16±47.09 | 0.06 | 0.954 |
| PDW (%) | 15.42±1.91 | 16.34±3.79 | 0.75 | 0.461 |
| MPV (fl) | 9.26±1.34 | 9.24±0.89 | 0.05 | 0.958 |
| PCT (%) | 0.16±0.06 | 0.17±0.04 | 0.89 | 0.377 |
| PLCR (%) | 18.53±6.85 | 21.75±6.64 | 1.27 | 0.215 |
| Fibrinogen (mg/dl) | 432.72±100.32 | 495.68±79.93 | 1.89 | 0.069 |

**Key:**
*: Significant p values
Hb = Haemoglobin
PDW = Platelet Distribution Width
MPV = Mean Platelet Volume
PCT = Plateletcret
P-LCR = Platelet-Large Cell Ratio

### *3.3. COMPARISON OF THE MEAN VALUES OF HAEMOGLOBIN, PLATELET COUNT, PDW, MPV, PCT, PLCR AND FIBRINOGEN IN ALZHEIMER PATIENTS BASED ON AGE*

There was no significant difference in the mean values of haemoglobin, platelet, PDW, MPV, PCT, PLCR and fibrinogen in Alzheimer patients of age (30-50) years (10.25±1.36)g/dl, (177.65±42.27)x$10^9$/L, (15.78±3.55)%, (8.96±0.83)fl, (0.18±0.04)%, (19.72±5.85)% and (463.65±87.00) mg/dl when compared to Alzheimer patients of age (51-70) years (10.51±1.51)g/dl, (181.00±58.98) x$10^9$/L, (16.44±2.55)%, (9.84±1.25)fl, (0.15±0.04)%, (22.28±8.44)% and (490.50±102.54)mg/dl. (p=0.631, p=0.859, p=0.606, p=0.228, p=0.110, p=0.338 and p=0.459)

**TABLE 3 Comparison of the mean values of haemoglobin, platelet count, PDW, MPV, PCT, PLCR and fibrinogen in alzheimer patients based on age**

| Parameters | 30-50 n=20 | 51-70 n=10 | t-value | p-value |
|---|---|---|---|---|
| Hb (g/dl) | 10.25±1.36 | 10.51±1.51 | 0.48 | 0.631 |
| Platelet x$10^9$/L | 177.65±42.27 | 181.00±58.98 | 0.18 | 0.859 |
| PDW (%) | 15.78±3.55 | 16.44±2.55 | 0.52 | 0.606 |
| MPV (fl) | 8.96±0.83 | 9.84±1.25 | 2.32 | 0.228 |
| PCT (%) | 0.18±0.04 | 0.15±0.04 | 1.65 | 0.110 |
| PLCR (%) | 19.72±5.85 | 22.28±8.44 | 0.98 | 0.338 |
| Fibrinogen (mg/dl) | 463.65±87.00 | 490.50±102.54 | 0.75 | 0.459 |

**Key:**
Hb = Haemoglobin
PDW = Platelet Distribution Width
MPV = Mean Platelet Volume
PCT = Plateletcret
P-LCR = Platelet-Large Cell Ratio

### *3.4. CORRELATION OF HAEMOGLOBIN WITH PLATELET INDICES AND FIBRINOGEN IN ALZHEIMER PATIENTS*

There was a significant positive correlation of haemoglobin with platelet, PCT and fibrinogen in asthmatic patients (r=0.48, p=0.000; r=0.37, p=0.004 and r=0.62, p=0.000). There was a non-significant positive correlation (r=0.14, p=0.289; r=0.17, p=0.186 and r=0.24, p=0.067) of haemoglobin with PDW, MPV and PLCR in asthmatic patients(r=0.14, p=0.289; r=0.17, p=0.186 and r=0.24, p=0.067).

**TABLE 4 Correlation of haemoglobin with platelet indices and fibrinogen in alzheimer patients**

| Variables | N | r | p-value |
|---|---|---|---|
| Platelet x$10^9$/L | 30 | 0.48 | 0.000 |
| PDW (%) | 30 | 0.14 | 0.289 |
| MPV (fl) | 30 | 0.17 | 0.186 |
| PCT (%) | 30 | 0.37 | 0.004 |
| PLCR (%) | 30 | 0.24 | 0.067 |
| Fibrinogen (mg/dl) | 30 | 0.62 | 0.000 |

**Key:**
Hb = Haemoglobin
PDW = Platelet Distribution Width
MPV = Mean Platelet Volume
PCT = Plateletcret
P-LCR = Platelet-Large Cell Ratio

## 4. DISCUSSION

Alzheimer's disease (AD) is the primary cause of dementia among the elderly and is an increasing global public health challenge. Despite substantial research, the biochemical underpinnings of Alzheimer's disease remain inadequately elucidated. AD now affects more than 5.3 million people in the US. By 2050, that number is expected to climb to 13.5 million[13]. There is more and more evidence that systemic haematological, inflammatory, and vascular problems may play a role in the start and evolution of this condition.
In the current investigation, average haemoglobin levels were markedly decreased in Alzheimer's patients relative to age-matched controls. A lower haemoglobin content may indicate cerebral hypoxia, ischaemia, and oxidative stress, all of which are known to speed up neurodegeneration. Low haemoglobin has also been associated with dysregulation of hypoxia-inducible factor and erythropoietin, both of which are essential for neuronal survival and oxygen homeostasis[14]. This finding aligns with prior research indicating that anaemia in older persons correlates with accelerated advancement of white matter damage and cognitive decline. Chronic kidney disease, which is frequently accompanied by low haemoglobin levels, was also demonstrated to exacerbate hypoxia in the brain. In these circumstances, reduced erythropoietin levels threaten to exacerbate loss of neurones as there are receptors in the brain for this hormone, which protect neurons in the experimental situations of hypoxia and ischaemic stroke[15]. As a result, low levels of haemoglobin in Alzheimer's disease patients could represent a major factor in neuronal vulnerability and cognitive decline.

The present study showed a remarkable decrease in the platelet count, platelet distribution width (PDW) and plateletcrit (PCT) in Alzheimer's patients when compared to controls. The importance of platelets extends beyond coagulation or stopping bleeding to include inflammation, modifying the immune response and tissue healing [16]. Platelets are also the principal source of serotonin (5-hydroxytryptamine) in the body and contain the enzymes that can cause degradation of APP [17]. It is believed that APP metabolism and abnormal platelet activation occur early in AD, which may contribute to the manufacture of amyloid-β and a dysfunctional neurovascular system22. Lower Platelet Indices Reported in the Present Study Align with Previous Reports of Altered Platelet Fractions Found in AD and Their Association with Cognitive Decline.

On the other hand, Alzheimer's patients had much higher mean platelet volume (MPV) and platelet-large cell ratio (P-LCR). MPV shows the size and activity of platelets, and bigger platelets are considered to be more active in terms of metabolism and enzymes, which makes them more likely to cause blood clots and inflammation [19]. The elevated MPV and P-LCR identified in this study indicate heightened platelet activation in Alzheimer's disease, potentially leading to microvascular blockage, endothelial dysfunction, and neuroinflammation. These results are consistent with earlier research indicating heightened platelet reactivity in neurodegenerative and vascular diseases. Although the use of antidepressants can affect platelet function, it was challenging to exclude this confounding variable due to the significant frequency of depression and antidepressant treatment among Alzheimer's disease patients in both study groups.

Fibrinogen levels were markedly increased in Alzheimer's patients relative to controls. Fibrinogen is a critical component of inflammation and coagulation, Macritchie said. It may also bind to receptors on the immune cells that trigger messages,

leading to inflammation[20]. In AD, increased levels of fibrinogen can pass through a compromised BBRB and accumulate in the CNS, where it associates with Aβ and forms abnormal fibrin clots that hamper cerebral blood flow and promote neuroinflammation [21]. The present findings are consistent with the results of Amor et al. (2014) , who reported that fibrinogen contributes to the pathogenesis of AD through induction of inflammatory pathways. Moreover, it has been suggested that fibrinogen could also act as a biological marker and modulator of inflammation-mediated neurodegeneration [22]. So, raised fibrinogen over time in AD might accelerate damage to neurones and loss of cognitive function.

A significant difference in haemoglobin and platelet indices, and fibrinogen levels was not observed between Alzheimer disease patients classified by age or gender. This suggests that these haematological and coagulation derangements are likely related more to the disease process as opposed to demographic factors, similar to other studies [23]. Correlation analysis showed positive correlation between haemoglobin and both platelet count and PCT, indicating that reductions in haemoglobin are also associated with a reduction of platelet mass in Alzheimer's patients. This finding is consistent with that of [24], who suggested that common pathogenic mechanisms (such as chronic inflammation or bone marrow inhibition) could be involved in both anaemia and thrombocytopenia in patients with neurodegenerative diseases [25]. A significant inverse association was found between haemoglobin and fibrinogen, thereby implying that low haemoglobin is associated with elevated concentration of fibrinogen, and this observation strengthens the evidence linking anaemia, inflammation and AD. Haemoglobin showed a positive correlation with PDW, MPV and P-LCR, which was a non-statistically significant association, a finding consistent with those of previous studies [26, 27].

## 5. CONCLUSION

Alzheimer's disease (AD) is associated with significant haematological and inflammatory changes, including decreased haemoglobin (HB), platelet (PLT) count, platelet distribution width (PDW) and plateletcrit (PCT) levels and increased mean platelet volume (MPV), platelet-large cell ratio (P-LCR) and fibrinogen levels. These changes are driven by hypoxia, platelet activation and systemic inflammation, which may facilitate the disease initiation and progression. These measures are not affected in Alzheimer's disease by age and sex. And haemoglobin has a positive association with platelet count and PCT, while having a negative correlation with fibrinogen, which suggests the interdependence of anaemia, thrombocytopenia and inflammation in Alzheimer's disease. These findings suggest that haematological and coagulation indices are possible novel, easily accessible biomarkers in the evaluation and monitoring of Alzheimer's disease.

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