Public Health Journal
ISSN: 2997-6790 |
Volume 12 Issue 4, October-December, 2024
Journal Homepage: https://ethanpublication.com/articles/index.php/E11,
Official Journal of Ethan Publication

BLOOD AND COAGULATION PARAMETERS IN UTERINE FIBROID PATIENTS: EVIDENCE FROM FEDERAL MEDICAL CENTRE, YENAGOA

[1]Ebiowei Pere Emmanuel and [2]Okeke Chinedu Augustine

[1]Department of Medical Laboratory Science, Faculty of Basic Medical Sciences, College of Health Sciences, Niger Delta University, Wilberforce Island, Bayelsa State, Nigeria.

[2]Department of Medical Laboratory Science, Faculty of Science, Delta State University, Abraka, Delta State, Nigeria.

DOI: https://doi.org/10.5281/zenodo.17379430

Abstract

Uterine leiomyomas (fibroids) are benign tumors that can have effects on haematological and coagulation parameters, particularly in those with menorrhagia. Evaluation of such change has bearings on clinical care and preoperative preparation. This study assessed selected haematological and coagulation profiles of uterine leiomyoma patients attending Federal Medical Centre, Yenagoa, Bayelsa State. A case-control design comprising 20 patients with uterine leiomyoma and 10 age-matched controls selected through a convenient sampling technique. Five millilitres of blood samples were drawn from each participant. Full blood count was analysed using a SYSMEX XP 300 haematology analyser, and activated partial thromboplastin time (APTT) and prothrombin time (PT) were assayed using a semi-automated coagulometer. Data analysis was carried out using SPSS version 10, and statistical significance was established at $p<0.05$. The finding was that uterine leiomyoma patients had significantly higher mean packed cell volume (PCV: 35.40 ± 2.81), haemoglobin (Hb: 12.14 ± 1.41), red blood cell count (RBC: 4.68 ± 0.82), lymphocytes (51.15 ± 10.67), mean corpuscular volume (MCV: 81.15 ± 8.20), mean corpuscular haemoglobin (MCH: 27.40 ± 4.20), and mean corpuscular haemoglobin concentration (MCHC: 31.81 ± 1.90) compared to the control (PCV: 31.45 ± 2.70, Hb: 10.69 ± 1.48, RBC: 3.78 ± 0.58, lymphocytes: 41.35 ± 5.06, MCV: 79.25 ± 9.01, MCH: 24.30 ± 3.18, MCHC: 26.20 ± 2.94). APTT of patients with fibroids was also significantly raised (32.33 ± 6.80 vs 28.73 ± 4.93), whereas PT (11.82 ± 1.98 vs 11.07 ± 1.24) and platelet count (269.35 ± 68.62 vs 258.75 ± 65.33) were mildly elevated but not significant. These changes indicate that uterine leiomyoma is associated with a change in haematological and coagulation profile, and these may be indicative of adaptive mechanisms to bleeding, inflammation, or endocrine effect. Laboratory evaluation of these parameters as a routine is recommended for adequate diagnosis, risk estimation, and management in uterine fibroid patients.

Keywords; *Uterine leiomyoma, fibroid, Prothrombin time (PT), Activated partial thromboplastin (APTT) Hematological parameters, Coagulation profile.*

Introduction

Uterine leiomyomas (ULs), or fibroids or myomas, are the most commonly diagnosed benign tumours of the female reproductive tract, developing from the smooth muscle cells of the myometrium [5,6]. Worldwide, an estimated 70–80% of women are expected to develop fibroids by the time they are 50 years old, with about 25–30% becoming symptomatic[1]. The tumours are especially common and more aggressive in women of African ancestry, such as Nigerian women, who also develop fibroids at earlier ages and in larger sizes and greater numbers than women of other racial groups[2]. Despite their benign nature, ULs impose a serious gynaecological

burden by virtue of their ability to cause a plethora of complications ranging from menorrhagia[40,] pelvic pain, dysmenorrhea, and subfertility to urinary retention and recurrent pregnancy loss[3,4]. As such, fibroids are the most common indication for hysterectomy globally, with more than 200,000 hysterectomies and 300,000 myomectomies undertaken yearly in the United States alone[5]. In Nigeria, where healthcare infrastructure remains largely underdeveloped, this burden is worsened by poor access to specialized care, weak diagnostic facilities, and sparse health record systems[32,33,34].

Uterine leiomyomas are commonly classified based on their anatomical location into three broad categories: subserosal, intramural, and submucosal[6]. Subserosal fibroids grow on the exterior surface of the uterus and may compress surrounding organs; intramural fibroids grow within the muscular uterine wall and are the most prevalent; while submucosal fibroids project into the endometrial cavity and are strongly associated with abnormal uterine bleeding and infertility even at a small size[7]. Fibroids may also be classified based on size as small (<2 cm), medium (2–6 cm), or large (>6 cm) with clinical presentation and surgical planning implications[8,9]. These variations explain the wide range of symptoms and complicate treatment planning[10,11].

The pathogenesis of uterine leiomyomas is multicausal, with interaction of genetic, hormonal, inflammatory, and environmental mechanisms. Somatic mutations of the *MED12* gene occur in approximately 70–85% of fibroids, while *HMGA2* rearrangements or overexpression define a large subset, typically in tumors lacking *MED12* mutations[18], implicating two main and often distinct molecular pathways for leiomyoma development[12,18]. Other chromosomal rearrangements— trisomy 12, deletions in 7q, and translocations between chromosomes 12 and 14 have also been implicated in UL pathogenesis, with racial difference in mutational patterns described between populations[13]. Hormonal imbalance, and in particular estrogen and progesterone imbalance, is central to fibroid development and growth. Estradiol upregulates the expression of progesterone receptors, while progesterone itself promotes cell proliferation and extracellular matrix (ECM) deposition, both of which drive fibroid growth[14]. In addition to genetic and hormonal influences, inflammation is involved in fibroid biology. Research has described the occurrence of *CD68*positive macrophages[15] and increased cytokine expression in fibroid tissues, with more pronounced cellular leiomyomas at early stages. Such observations imply that chronic inflammation and wound-healing reactions are involved in ECM remodelling and tumor maintenance[16].

The clinical treatment of uterine leiomyoma is based on symptom severity, fibroid size and location, age, and the desire for fertility preservation. The treatment may vary from conservative management with observation and hormonal therapy (e.g., GnRH agonists) to interventional procedures like uterine artery embolization, myomectomy, or hysterectomy in extreme cases[17,18,19,20]. These treatment strategies are, however, constrained in low-resource communities where diagnosis and care delays are prevalent. Notwithstanding intensive research, one of the significant challenges is the heterogeneity of uterine fibroids with regard to size, number, growth pattern, and symptomatology. Some women have large fibroids and are asymptomatic, whereas others are severely symptomatic even with small submucosal fibroids[21]. Such clinical variability makes diagnosis and management challenging, particularly in low-resource environments[20]. One of the common manifestations of symptomatic fibroids is menorrhagia, which may result in iron-deficiency anemia and consequent changes in haematological indices like packed cell volume (PCV), hemoglobin (Hb), and red blood cell (RBC) counts[22]. On the other hand, in exceptional instances, fibroids may cause erythrocytosis through ectopic secretion of erythropoietin, leading to

increases in RBC counts and hemoglobin levels[23,24]. Such presentations require critical laboratory assessment for informed clinical management. Besides haematological alterations, uterine leiomyomas could affect haemostatic functions. Coagulation times like activated partial thromboplastin time (APTT) and prothrombin time (PT) are vital markers of the integrity of intrinsic and extrinsic coagulation pathways, respectively[25, 26]. Changes in these may result from chronic blood loss, endocrine influences, or inflammation and impact surgical planning, risk of bleeding, and transfusion requirements. The clinical utility of prolonged PT and APTT is their ability to unmask coagulopathies or subclinical bleeding tendencies, especially in fibroid patients facing invasive interventions[26].
The complete blood count (CBC) is still an essential investigative tool in the evaluation of the hematologic status of women presenting with uterine fibroids. The MCV, MCH, MCHC, WBC, and platelet count are especially useful for the detection of anemia, inflammation, and bleeding complications in these patients. Meanwhile, coagulation screening tests like APTT and PT detect potential risks of bleeding or hypercoagulable conditions, which are particularly relevant in fibroid patients who are surgical candidates[27].
Despite the global prevalence and clinical significance of uterine leiomyomas[36], localized studies evaluating the haematological and coagulation profiles of fibroid patients are lacking in Nigeria. Most existing studies are high-income country-based and might not capture the unique genetic, nutritional, and environmental exposures of Nigerian women[32,33,34]. With the rising burden of UL in low- and middle-income countries, there is an urgent need to develop context-specific data that can inform evidence-based clinical practice[32,33]. This study therefore attempts to evaluate some of the haematological and coagulation parameters in uterine leiomyoma patients presenting at the Federal Medical Center, Yenagoa, Bayelsa State. By comparing with age-matched controls, the study will identify the significant haematologic and haemostatic abnormalities associated with fibroids and provide a scientific basis for improved diagnosis, prognosis, and therapeutic care of affected females.

Materials and Methods Study Area

The study was carried out at Federal Medical Centre (FMC) Yenagoa, in Yenagoa Metropolis, Bayelsa State. Federal Medical Centre, Yenagoa is a tertiary health institution and serves as a referral centre within and outside the state. It is also the largest and most equipped medical center that serves as a training centre for both Medical and Allied Medical Science students.

Study Population

The study was carried out among uterine leiomyoma patients attending FMC, Yenagoa, Bayelsa State.

Sample Size

The sample size was 20 uterine leiomyoma patients and 10 non-uterine leiomyoma patients making a total of 30 participants.

Ethical Approval and Informed Consent

Ethical approval was obtained from the Research Ethics Committee of Federal Medical Centre, Yenagoa, Bayelsa State. All participants gave informed consent after receiving full explanations of the study's purpose and procedures. Data confidentiality was ensured throughout the study.

Public Health Journal
ISSN: 2997-6790 |
Volume 12 Issue 4, October-December, 2024
Journal Homepage: https://ethanpublication.com/articles/index.php/E11,
Official Journal of Ethan Publication

Inclusion and Exclusion Criteria

Only patients clinically and/or radiologically confirmed to have uterine leiomyoma and currently receiving care were included. Non-leiomyoma patients and those who declined consent were excluded.

Sampling Technique

Participants were selected using convenience sampling based on willingness to participate and availability during the study period.

Sample Collection and Processing

Five (5ml) millilitres of venous blood was collected aseptically from each participant into EDTA tubes for haematological analysis and citrated tubes for coagulation studies. Haematological parameters were analyzed using a Sysmex KX-300 automated hematology analyzer, while coagulation parameters were evaluated using a semi-automated coagulometer in the clinical laboratory.

Laboratory Analysis Full Blood Count (FBC):

The Sysmex KX-300 analyzer was used to analyze the EDTA-anticoagulated blood samples. Key haematological indices measured included hemoglobin (Hb), packed cell volume (PCV), red blood cell (RBC) count, white blood cell (WBC) count, platelet count, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC).

Prothrombin Time (PT):

PT was measured using thromboplastin reagents in the presence of calcium ions to assess the integrity of the extrinsic and common coagulation pathways. The time (in seconds) for clot formation was recorded. Reference range: 10–15 seconds.

Activated Partial Thromboplastin Time (APTT):

APTT was determined using cephaloplastin and calcium reagents to evaluate the intrinsic and common coagulation pathways. Clotting time was measured manually or using a semi-automated coagulation system. Reference range: 21–38 seconds.

Statistical Analysis

Data were analyzed using SPSS version 10.0. Continuous variables were expressed as mean ± standard deviation. Independent sample t-tests were used for comparison between fibroid and control groups for normally distributed data; Mann–Whitney U test was applied for non-parametric data. Fisher's exact test was used to assess associations between categorical variables. A *p-value* of < 0.05 was considered statistically significant.

Results

The results are presented as follows;

Table 4.1: Comparison of Mean Values of Haematological Parameters of Uterine leiomyomas and Control Groups.

Haematological (X±SD) n=20	Control Group (X±SD) n=20	Uterine Fibroid patients value	t-value	P- Parameters
PCV (%)	31.45 ± 2.70	35.40 ±2.81	4.627	0.000*

Public Health Journal
ISSN: 2997-6790 |
Volume 12 Issue 4, October-December, 2024
Journal Homepage: https://ethanpublication.com/articles/index.php/E11,
Official Journal of Ethan Publication

Hb (g/dl)	10.69 ± 1.48	12.14 ± 1.41	4.954	0.000*
RBC (x 10^9/L)	3.78 ± 0.58	4.68 ± 0.82	3.457	0.003*
TWBC (x 10^9/L)	5.34 ± 1.14	5.38 ± 1.44	0.332	0.743
NEUT (%)	35.55 ± 7.28	35.65 ± 9.74	0.093	0.927
LYMP (%)	41.35 ± 5.06	51.15 ± 10.67	-5.695	0.000*
MONO (%)	9.05 ± 1.87	9.51 ± 1.52	-0.819	0.385
EOSIN (%)	4.25 ± 1.11	4.30 ± 1.52	2.926	0.101
MCV (fl)	79.25 ± 9.01	81.15 ± 8.20	1.942	0.047*
MCH (pg)	24.30 ± 3.18	27.40 ± 4.20	1.886	0.010*
MCHC (g/dl)	26.20 ± 2.94	31.81 ± 1.90	7.380	0.000*

Key: Values with (*) superscript are statistically significant at p< 0.05. Results are expressed as Mean ± Standard Deviation. Student t- test was used to compare the two groups. p< 0.05 is considered significant; Hb=Haemoglobin; PCV= Packed Cell Volume; MCV= Mean Cell Volume; MCH= Mean Cell Haemoglobin; MCHC= Mean Cell Haemoglobin Concentration; TWBC=Total white cell; NEUT =Neutrophil; LYMP

Table 4.1 shows the comparison of mean values of haematological parameters of uterine leiomyomas and control groups. The result revealed that the mean values of PCV (35.40 ±2.81), Hb (12.14 ± 1.41), RBC (4.68 ± 0.82), Lymphocytes (51.15 ± 10.67), MCV (81.15 ± 8.20), MCH (27.40 ± 4.20) and MCHC (31.81 ± 1.90) in the uterine leiomyomas patients were significantly (p<0.05) higher when compared with the control group (31.45 ± 2.70, 10.69 ± 1.48, 3.78 ± 0.58, 41.35 ± 5.06, 79.25 ± 9.01, 24.30 ± 3.18 and 26.20 ± 2.94) respectively. The levels of total WBC (5.38 ± 1.44), Neutrophil (35.65 ± 9.74), monocytes (9.51 ± 1.52), and eosinophils (4.30 ± 1.52) in the uterine leiomyomas patients were insignificantly (p>0.05) higher than the control group (5.34 ± 1.14; 35.55 ± 7.28; 9.05 ± 1.87 and 4.25 ± 1.11) respectively.

Table 4.2: Comparison of Mean Values of Coagulation Parameters of Uterine leiomyomas Patients and Control Groups.

Haematological (X±SD) n=20	Control Group (X±SD) n=20	Uterine Fibroid patients value	t-value	P- Parameters
PLT (%)	258.75 ± 65.33	269.35 ± 68.62	1.613	0.123
PT (g/dl)	11.07 ± 1.24	11.82 ± 1.98	1.407	0.159
APTT (x 10^9/L)	28.73 ± 4.93	32.33 ± 6.80	2.086	0.041*

Key: Values with (*) superscript are statistically significant at p< 0.05. Results are expressed as Mean ± Standard Deviation. Student t- test was used to compare the two groups. p< 0.05 is considered significant; PLT= Platelets; PT= Prothrombin Time; APTT= Activate Partial Thromboplastic Time.

Public Health Journal
ISSN: 2997-6790 |
Volume 12 Issue 4, October-December, 2024
Journal Homepage: https://ethanpublication.com/articles/index.php/E11,
Official Journal of Ethan Publication

Table 4.2 shows the comparison of mean values of coagulation parameters of uterine leiomyomas patients and control groups**.** The result revealed that the levels of APTT (32.33 ± 6.80) in the uterine fibroid patients was significantly ($p<0.05$) higher the control group **(**28.73 ± 4.93). While, the mean values of platelet (269.35 ± 68.62) and prothrombin time (11.82 ± 1.98) in the uterine leiomyomas patients was slightly higher than the control group (258.75 ± 65.33 and 11.07 ± 1.24) respectively, but not statistically significant ($p>0.05$).

Table 4.3: Comparison of Mean Values of Haematological Parameters of the Studied Group by Age.

Haematological (X±SD) n=11	**20-30yrs (X±SD) n=05**	**31-40yrs (X±SD) n=04**	**41yrs and above**	**F- value**	**P-value Parameters**
PCV (%)	36.18 ± 2.48	36.20 ± 2.58	32.25 ± 2.06	1.980	0.176
Hb (g/dl)	12.35 ± 1.17	12.16 ± 1.67	11.27 ± 1.77	0.852	0.368
RBC (x 10^9/L)	4.83 ± 0.90	4.40 ± 0.55	4.57 ± 0.97	0.819	0.377
TWBC (x 10^9/L)	5.95 ± 1.52	4.70 ± 0.96	4.67 ± 1.29	4.305	0.053
NEUT (%)	34.72 ± 10.04	33.20 ± 10.52	41.25 ± 7.88	0.210	0.652
LYMP (%)	50.09 ± 11.12	54.60 ± 13.27	49.75 ± 6.91	0.231	0.637
MONO (%)	10.54 ± 2.50	10.40 ± 1.51	6.75 ± 1.25	2.620	0.123 EOSIN (%)
4.63 ± 1.36	5.20 ± 0.83	3.25. ± 0.50	1.201	0.287	
MCV (fl)	80.62 ± 9.50	82.40 ± 7.40	81.00 ± 7.78	0.089	0.769
MCH (pg)	26.72 ± 5.19	25.60 ± 2.88	25.50 ± 2.88	0.372	0.549
MCHC (g/dl)	31.81 ± 2.52	31.80 ± 1.09	31.75 ± 0.50	0.002	0.964
PLT (%)	302.54 ± 54.48	248.80 ± 69.41	203.75 ± 55.12	7.747	0.012*
PT (g/dl)	12.07 ± 2.27	12.02 ± 1.89	10.87 ± 1.21	0.384	0.543 APTT (x 10^9/L)
32.05 ± 6.79	32.88 ± 6.96	32.42 ± 8.55	0.039	0.845	

Key: Values with (*) superscript are statistically significant at $p< 0.05$. Results are expressed as Mean ± Standard Deviation. Student t-test was used to compare the two groups. $p< 0.05$ is considered significant. Hb=Haemoglobin; PCV= Packed Cell Volume; MCV= Mean Cell Volume; MCH= Mean Cell Haemoglobin; MCHC= Mean Cell Haemoglobin Concentration; TWBC=Total white cell; NEUT =Neutrophil; LYMP =Lymphocyte; MONO =Monocyte; EOSIN =Eosinophil; PLT= Platelet.

Table 4.3 shows the comparison of mean values of haematological parameters of the studied group by age. The result revealed that there was no significant ($p>0.05$) difference observed in all the analyzed haematological parameters in all age groups under study except platelets. However, the mean values of PCV (36.18 ± 2.48), Hb (12.35 ± 1.17), RBC (4.83 ± 0.90), WBC (5.95 ± 1.52), MCH (26.72 ± 5.19), MCHC (31.81 ± 2.52), and platelets (302.54 ± 54.48) in the uterine fibroid patients of 20-30 years of age were slightly ($p>0.05$) higher when compared with the other age groups, but not statistically significant. Lymphocyte (54.60 ± 13.27), eosinophils (5.20 ± 0.83), MCV (82.40 ± 7.40) and APTT (32.88 ± 6.96) in the patients within 30-40 years age bracket was slightly higher

(p>0.05) than other age groups. Neutrophil levels (41.25 ± 7.88) in the patient aged 40 years and above was slightly higher than other age groups, but not statistically significant (p>0.05).

Discussion

This study evaluated significant haematological and coagulation parameters among uterine leiomyoma patients compared with a non-fibroid control group in a Nigerian tertiary hospital. The results demonstrate statistically significant elevations in packed cell volume (PCV), haemoglobin concentration (Hb), red blood cell count (RBC), lymphocyte count, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), and mean corpuscular haemoglobin concentration (MCHC) among the fibroid group, while white blood cell indices, platelet count, and prothrombin time (PT) showed modest but non-significant differences. While uterine fibroids are classically associated with iron-deficiency anemia due to heavy menstrual blood loss, this observation highlights a less commonly reported phenomenon where erythropoiesis may be sustained or even increased in a subset of patients. One possible explanation is the ectopic production of erythropoietin by the fibroid tissue, a syndrome known as myomatous erythrocytosis syndrome. This finding does not align with that of [28] which reported reduction in red blood cell indices; however, it is in tandem with the case reports by[23,24] where patients presented with elevated haemoglobin and RBC concentrations that normalized after myomectomy or hysterectomy, implicating fibroid-derived erythropoietin in the erythrocytosis. These accounts add weight to the hypothesis that leiomyomas are not merely inert growths but endocrinologically and metabolically active tissues capable of systemic physiologic effects[39].

The slight rise in lymphocyte and eosinophil levels in fibroid patients aligns with the chronic inflammatory state often seen in fibroid tissue. The inflammatory microenvironment of leiomyomas has been previously characterized, with *CD68*-positive macrophage infiltration, mast cell presence, and overexpression of cytokines such as interleukin-6 (*IL-6*), tumor necrosis factoralpha (TNF-α), and monocyte chemoattractant protein-1 (MCP-1)[15,16]. Not only are these proinflammatory markers involved in tumor maintenance and extracellular matrix remodeling, but they also suggest systemic immune stimulation, which may be responsible for the mild increases in white cell subsets. Moreover, estrogen—a central growth factor for uterine fibroids, is itself involved in modulating the activity of immune cells and releasing cytokines, which can potentially augment these immune hematologic findings[29].

Coagulation studies revealed that activated partial thromboplastin time (APTT) was significantly prolonged in the leiomyoma group compared with controls, while prothrombin time (PT) remained statistically unchanged.

On analysis based on age groups, there were no statistically significant differences in red cell or white cell indices except for platelet count. However, younger women (20–30 years) had slightly higher values for PCV, Hb, RBC, and platelet parameters. This may be a reflection of a more competent compensatory hematopoietic response in the younger age group, in whom bone marrow activity and hormonal balance are likely to be better[28]. However, the neutrophil count exhibited a slight peak in the ≥40 age group, which may either be an expression of a cumulative inflammatory response or enhanced physiological stress as a result of long-standing fibroid disease [25]. Coagulation studies revealed that activated partial thromboplastin time (APTT) was significantly prolonged in the leiomyoma group compared with controls, while prothrombin time (PT) remained statistically unchanged. APTT is a sensitive indicator of the intrinsic and common pathways of coagulation, and prolongation may be due

to reduced levels of specific clotting factors, consumptive coagulopathy secondary to chronic uterine bleeding, or hormonal modulation of coagulation proteins. Estrogen has been shown to influence the synthesis of clotting factors by increasing levels of factors II, VII, IX, and X and inhibiting natural anticoagulants such as antithrombin III and protein S, with a consequent effect on haemostatic balance[25,26,38]. Despite a normal PT, the isolation prolongation of APTT points to a potential disturbance in the intrinsic pathway and underscores the need for comprehensive coagulation screening in fibroid patients, especially those planned for surgery. Platelet count in leiomyoma patients was moderately higher compared to the control group, although not statistically significant. This finding can be a manifestation of a physiological reaction to chronic blood loss with heightened production of thrombopoietin to maintain hemostasis. Furthermore, the involvement of estrogen in the proliferation of megakaryocytes and platelet activation has been described, indicating a hormonally mediated increase in platelet turnover even in the absence of clinical thrombocytosis[28]. While platelet counts themselves can still be within reference values, their function can be altered with relevance to the assessment of bleeding risk after surgical intervention.

The relationship between leiomyomas and systemic hematologic change is increasingly evident. Leiomyomas are not only local gynecologic tumors but exert endocrine and paracrine effects that impact distant systems such as the bone marrow, liver, and vascular endothelium[30]. In particular, the involvement of fibroids in erythropoietin production, cytokine signaling, and coagulation cascades provides a basis for the growing body of evidence that these tumors create a complex relationship between hormones, inflammation, and hematopoiesis. Numerous studies support this model of systemic fibroid pathophysiology, reporting significant alterations in hemoglobin count, platelet function, and coagulation times in women with symptomatic fibroids[27,21,37]. In resourcepoor environments such as Nigeria, where access to advanced diagnostic imaging and molecular assays may be limited, routine laboratory tests such as full blood count (FBC), activated partial thromboplastin time (APTT), and prothrombin time (PT) continue to play a central role in the evaluation and management of uterine fibroid patients. These simple, cost-effective investigations FBC, APTT, and PT remain pivotal in low-resource settings like Nigeria, where access to advanced diagnostics is limited. They offer critical insight into anemia, bleeding tendencies, and coagulation status factors essential for pre-surgical risk assessment and clinical monitoring[31]. In addition, these laboratory parameters often serve as the only available tools in many peripheral or underfunded facilities, and their findings may guide treatment decisions ranging from conservative hormonal therapy to invasive options like uterine artery embolization or hysterectomy. Despite the value of this study, a few limitations must be acknowledged. The relatively small sample size could affect the generalizability of the findings, and key variables such as serum erythropoietin and fibrinogen concentrations were not assessed due to logistic constraints. Future studies should consider these markers, along with fibroid-associated genetic mutations such as *MED12* and *HMGA2*, to gain deeper insight into the molecular basis of haematological alterations in leiomyoma patients.

Conclusion

The study observed that there was a significant increase in haematological and haemostatic parameters in uterine leiomyoma patients. Thus, these findings highlight the importance of routine haematological and coagulation parameters profiling in the diagnosis and management of uterine fibroids.

Acknowledgement

The authors hereby deeply acknowledge and appreciate the staff of obstetrics and gynaecology clinic, Federal Medical Centre, Yenagoa for their supportive role during this study.

Competing Interest

The authors declare that there is no competing interest.

Author's Contribution

All authors contributed reasonably to this work.

REFERENCES

Stewart, E. A., Cookson, C. L., Gandolfo, R. A., & Schulze-Rath, R. (2017). Epidemiology of uterine fibroids: A systematic review. *BJOG: An International Journal of Obstetrics & Gynaecology, 124*(10), 1501–1512. https://doi.org/10.1111/1471-0528.14640.

Baird, D. D., Hill, M. C., Schectman, J. M., & Hollis, B. W. (2013). Vitamin D and the risk of uterine fibroids. *Epidemiology, 24*(3), 447–453.

Vitale, S. G., Riemma, G., Ciebiera, M., & Cianci, S. (2020). Hysteroscopic treatment of submucosal fibroids in perimenopausal women: When, why and how? *Climacteric, 23*, 355–359.

Marsh, E. E., Al-Hendy, A., Kappus, D., Galitsky, A., Stewart, E. A., & Kerolous, M. (2018). Burden, prevalence, and treatment of uterine fibroids: A survey of US women. *Journal of Women's Health, 27*(11), 1359–1367. https://doi.org/10.1089/jwh.2018.707

Yang, Q., & Al-Hendy, A. (2023). Update on the role and regulatory mechanism of extracellular matrix in the pathogenesis of uterine fibroids. *International Journal of Molecular Sciences, 24*(6), 5778. https://doi.org/10.3390/ijms24065778

Abdelmtalab, M. A., Tahir, O., Hussein, K., & Badawi, K. (2020). Anatomical locations of uterine fibroids in Sudanese women. *Anatomy Journal of Africa*, 9(1), 1701-1706.

Don, E. E., Middelkoop, M., Hehenkamp, W. J. K.(2023). *Endometrial Angiogenesis of Abnormal Uterine Bleeding and Infertility in Patients with Uterine Fibroids: A Systematic Review*. International Journal of Molecular Sciences, 24(8), 7011. doi:10.3390/ijms24087011.

Gayathre, S. P., & Sivakumar, T. (2021). Giant uterine fibroid: a rare differential diagnosis for an abdominopelvic mass. *International Surgery Journal*, 8(11), 3475-3478.

Mathew, R. P., Francis, S., Jayaram, V., & Anvarsadath, S. (2021). Uterine leiomyomas revisited with review of literature. *Abdominal Radiology,* 46(10), 4908-4926.

Tu, W., Yano, M., Schieda, N., Krishna, S., Chen, L., Gottumukkala, R. V., & Alencar, R. (2023). Smooth Muscle Tumors of the Uterus at MRI: Focus on Leiomyomas and FIGO Classification. RadioGraphics, 43(6), e220161.

De Bruyn, C., Ceusters, J., Vanden Brande, K., Timmerman, S., Froyman, W., Timmerman, D. & Van den Bosch, T. (2023). Ultrasound features using MUSA terms and definitions in uterine sarcoma and leiomyoma: retrospective cohort study. *Ultrasound in Obstetrics & Gynecology*.

Galindo, L. J., Hernández-Beeftink, T., Salas, A., Jung, Y., Reyes, R., de Oca, F. M., ... & Almeida, T. A. (2018). HMGA2 and MED12 alterations frequently co-occur in uterine leiomyomas. *Gynecologic Oncology, 150*(3), 562–568. https://doi.org/10.1016/j.ygyno.2018.07.007

Shtykalova, S. V., Egorova, A. A., Maretina, M. A., et al. (2021). Molecular genetic basis and prospects of gene therapy of uterine leiomyoma. *Russian Journal of Genetics, 57*, 1002–1016. https://doi.org/10.1134/S1022795421090118

Faustino, F., Martinho, M., Reis, J., & Águas, F. (2017). Update on medical treatment of uterine fibroids. *European Journal of Obstetrics & Gynecology and Reproductive Biology, 216*, 61–68. https://doi.org/10.1016/j.ejogrb.2017.06.047

Protic, O., Toti, P., Islam, M. S., Occhini, R., Giannubilo, S. R., Catherino, W. H., ... & Ciarmela, P. (2016). Possible involvement of inflammatory/reparative processes in the development of uterine fibroids. *Cell and Tissue Research, 364*(2), 415–427. https://doi.org/10.1007/s00441-015-2324-3

Zannotti, A., Greco, S., Pellegrino, P., Giantomassi, F., Delli Carpini, G., & Goteri, G. (2021). Macrophages and immune responses in uterine fibroids. *Cells, 10*(5), 982. https://doi.org/10.3390/cells10050982.

Jeng, C. J., Long, C. Y., & Chuang, L. T. (2020). Comparison of magnetic resonance-guided highintensity focused ultrasound with uterine artery embolization for the treatment of uterine myoma: A systematic literature review and meta-analysis. *Taiwanese Journal of Obstetrics and Gynecology*, 59(5), 691-697.

Yang, Q., Ciebiera, M., Bariani, M. V., Ali, M., Elkafas, H., Boyer, T. G., & Al-Hendy, A. (2022). Comprehensive review of uterine fibroids: Developmental origin, pathogenesis, and treatment. *Endocrine Reviews, 43*, 678–719. https://doi.org/10.1210/endrev/bnab039

Singh, S., Kumar, P., Rathore, S. S., Singh, Y., & Garg, N. (2023). Contemporary approaches in the management of uterine leiomyomas. *European Journal of Obstetrics & Gynecology and Reproductive Biology.*

Stanescu, A. D., Loghin, M. G., Ples, L., Balan, D. G., Paunica, I., & Balalau, O. D. (2021). Therapeutic approach of uterine leiomyoma; choosing the most appropriate surgical option. *Journal of Clinical and Investigative Surgery,* 6(1), 1-5.

Giuliani, E., As-Sanie, S., & Marsh, E. E. (2020). Epidemiology and management of uterine fibroids. *International Journal of Gynecology & Obstetrics, 149*(1), 3–9. https://doi.org/10.1002/ijgo.13102

Ali, M., Sara, A. R., & Al-Hendy, A. (2021). Elagolix in the treatment of heavy menstrual bleeding associated with uterine fibroids in premenopausal women. *Expert Review of Clinical Pharmacology, 14*(4), 427–437. https://doi.org/10.1080/17512433.2021.1900726.

Ono, Y., Hidaka, T., & Fukuta, K. (2014). A case of myomatous erythrocytosis syndrome associated with a large uterine leiomyoma. *Case Reports in Obstetrics and Gynecology, 2014*, 602139. https://doi.org/10.1155/2014/602139.

De Boer, J. P., Velders, G., Aliredjo, R., Scheenjes, E., & Flinsenberg, T. W. H. (2017). Polycythaemia in a patient with uterus myomatosus. *Nederlands Tijdschrift Voor Geneeskunde, 161*, D1161–D1161.

Ottolina, J., Bartiromo, L., Dolci, C., Salmeri, N., Schimberni, M., Villanacci, R., .& Candiani, M. (2020). Assessment of coagulation parameters in women affected by endometriosis: validation study and systematic review of the literature. *Diagnostics*, *10*(8), 567.. https://doi.org/10.3390/diagnostics10080567.

Kobayashi, T., Hirayama, M., Nogami, M., Meguro, K., Iiduka, M., Foidart, J. M., ... & Harada, T. (2024). Impact of estetrol combined with drospirenone on blood coagulation and fibrinolysis in patients with endometriosis: a multicenter, randomized, open-label, activecontrolled, parallel-group study. *Clinical and Applied Thrombosis/Hemostasis*, *30*, 10760296241286514.https://doi.org/10.1177/10760296241286514

Keshta, M. S., Ghanem, M., Alsayed, Y., Zeidan, O., Khorma, Y., Jeddy, R., ... & Jeddy, R. (2024). A multicenter retrospective cohort study assessing the incidence of anemia in patients associated with uterine fibroids. *Cureus, 16*(9). https://doi.org/10.7759/cureus.69899.

Covali, R., Socolov, D., Pavaleanu, I., Caraăuleanu, A., & Socolov, R. (2020). Platelet indices in uterine leiomyoma compared with other major gynecological diseases. *Journal of Advances in Medicine and Medical Research, 32*(24), 164–176. https://doi.org/10.9734/JAMMR/2020/v32i2430764

Ciebiera, M., Esfandyari, S., Siblini, H., Prince, L., Elkafas, H., Wojtyła, C., ... & Ali, M. (2021). Nutrition in gynecological diseases: Current perspectives. *Nutrients, 13*, 1178. https://doi.org/10.3390/nu13041178

Guyton, A. C., & Hall, J. E. (2006). Hemostasis and blood coagulation. *Textbook of medical physiology*, *1*, 419-429.

Mahlangu, J., Diop, S., & Lavin, M. (2024). Diagnosis and treatment challenges in lower resource countries: State-of-the-art. *Haemophilia, 30*, 78–85. https://doi.org/10.1111/hae.14956

Ezeama, C. O., Ikechebelu, J. I., Obiechina, N. J., & Ezeama, N. N. (2012). Clinical Presentation of Uterine Fibroids in Nnewi, Nigeria: A 5. year Review. *Annals of medical and health sciences research*, *2*(2), 114-118

Akinola, L. A., Okohue, J., Ikechebelu, J., Alabi, C. O., & Olumodeji, A. M. (2021). Approach to Management of Submucous Fibroids by Gynaecological Endoscopy Surgeons/Trainees in Nigeria. *African Journal of Reproduction and Gynaecological Endoscopy*, *6*(1), 1-9.

Iwuoha EC, Uwaegbulam CE, Eni I, & Thomas J. (2022). Prevalence of Uterine Fibroids Among Women Presenting at Abia State University Teaching Hospital, Abia State: a Five-Year Review. *A Medical Journal Of Abia State University Medical Students' Association, 14(1)1-12*

Adegbesan-Omilabu, M. A., Okunade, K. S., & Gbadegesin, A. (2014). Knowledge of, perception of, and attitude towards uterine fibroids among women with fibroids in Lagos, Nigeria. *Scientifica*, *2014*(1), 809536.

Sefah, N., Ndebele, S., Prince, L., Korasare, E., Agbleke, M., Nkansah, A., ... & Agbleke, A. A. (2023). Uterine fibroids—Causes, impact, treatment, and lens to the African perspective. *Frontiers in Pharmacology*, *13*, 1045783.

Arip, M., Yap, V. L., Rajagopal, M., Selvaraja, M., Dharmendra, K., & Chinnapan, S. (2022). Evidence-based management of uterine fibroids with botanical drugs-a review. Frontiers in Pharmacology, 13, 878407.

Borahay, M. A., Asoglu, M. R., Mas, A., Adam, S., Kilic, G. S., & Al-Hendy, A. (2017). Estrogen receptors and signaling in fibroids: role in pathobiology and therapeutic implications. *Reproductive sciences*, *24*(9), 1235-1244.

Ali, M., Ciebiera, M., Vafaei, S., Alkhrait, S., Chen, H. Y., Chiang, Y. F., ... & Al-Hendy, A. (2023). Progesterone signaling and uterine fibroid pathogenesis; molecular mechanisms and potential therapeutics. *Cells*, *12*(8), 1117.

Uimari, O., Subramaniam, K. S., Vollenhoven, B., & Tapmeier, T. T. (2022). Uterine fibroids (Leiomyomata) and heavy menstrual bleeding. *Frontiers in Reproductive Health*, *4*, 818243.