

Platelet Indices and Coagulation Times in Women with Cervical Cancer in Owerri Nigeria

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Abstract

Background: Cervical cancer remains a major cause of illness and death among women in low- and middle-income countries. Malignancy can disturb haemostasis through tumour-associated inflammation, platelet activation, endothelial injury, treatment effects and activation of coagulation. Platelet indices are generated during routine automated blood counts and may provide useful information about platelet size and heterogeneity, while prothrombin time and activated partial thromboplastin time assess selected components of plasma coagulation. **Objective:** To compare mean platelet volume, platelet distribution width, platelet-large cell ratio, prothrombin time and activated partial thromboplastin time between women with cervical cancer and apparently healthy women in Owerri, Nigeria, and to examine the relationship between prothrombin time and activated partial thromboplastin time among the patients. **Methods:** This hospital-based comparative cross-sectional study enrolled 50 adult women, comprising 25 patients with histologically confirmed cervical cancer and 25 age-matched apparently healthy controls. Venous blood was collected into dipotassium ethylenediaminetetraacetic acid tubes for platelet indices and 3.2% trisodium citrate tubes for coagulation testing. Mean platelet volume, platelet distribution width and platelet-large cell ratio were obtained using an automated haematology analyser. Prothrombin time and activated partial thromboplastin time were determined from platelet-poor plasma by clot-based methods. Group means were compared with the independent-samples t test, and the relationship between prothrombin time and activated partial thromboplastin time was assessed using Pearson correlation. Statistical significance was set at $p < 0.05$. **Results:** Mean platelet volume was lower in the cervical cancer group than in controls (8.61 ± 1.06 versus 10.06 ± 0.91 fL; $p < 0.001$). Platelet distribution width was also lower ($11.72 \pm 1.90\%$ versus $14.18 \pm 1.50\%$; $p < 0.001$), as was platelet-large cell ratio ($17.88 \pm 2.80\%$ versus $21.12 \pm 4.36\%$; $p = 0.003$). Prothrombin time did not differ significantly between patients and controls (14.70 ± 1.91 versus 14.31 ± 1.29 seconds; $p = 0.400$), and activated partial thromboplastin time was likewise similar (34.68 ± 1.81

versus 33.81 ± 2.10 seconds; $p = 0.124$). Prothrombin time showed a very weak, non-significant positive correlation with activated partial thromboplastin time among patients ($r = 0.061$; $p = 0.772$). Conclusion: Women with cervical cancer in this study had lower platelet size and heterogeneity indices, whereas routine clotting times were not significantly altered. The results support further investigation of platelet indices as inexpensive adjunctive markers, but they do not justify using these indices alone to infer platelet activation, thrombosis risk or disease severity. Larger studies that account for tumour stage, treatment exposure, platelet count, inflammation and nutritional status are required.

Keywords: *Activated partial thromboplastin time, cervical cancer, mean platelet volume, platelet distribution width, platelet-large cell ratio, prothrombin time*

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1 Introduction

Cervical cancer arises from the cervix and is caused in nearly all cases by persistent infection with oncogenic human papillomavirus (HPV). Infection is common, but most infections clear spontaneously; malignant transformation usually follows persistent high-risk infection, precancerous change and the accumulation of host and viral alterations over many years. HPV types 16 and 18 account for the largest proportion of invasive disease. The long premalignant phase makes cervical cancer unusually amenable to prevention through HPV vaccination and screening, yet its burden remains concentrated in settings where access to these services and to timely treatment is limited.^{1–4}

The global burden is substantial. GLOBOCAN 2022 estimated about 660,000 new cases and 350,000 deaths worldwide, placing cervical cancer among the leading cancers affecting women. The disparity between countries is marked: incidence and mortality are highest in many countries in sub-Saharan Africa, where organized screening, pathology services, radiotherapy and follow-up are often insufficient. Nigeria contributes a large

absolute number of cases and deaths within Africa.

These figures represent more than an epidemiological problem because women frequently present with symptomatic or advanced disease, when anaemia, infection, malnutrition, bleeding, thrombosis and treatment-related complications may occur together.^{1,2,5} Cervical carcinogenesis begins in epithelial tissue, but invasive disease can produce systemic effects. Tumour cells release procoagulant and inflammatory mediators, activate endothelial cells and interact with circulating blood cells. Platelets can adhere to tumour cells, release growth factors and cytokines, and support angiogenesis, immune evasion and metastatic dissemination. Cancer also increases venous thromboembolism risk through immobility, tissue-factor expression, vascular compression, surgery, chemotherapy and central venous catheters. These processes do not necessarily produce a uniform laboratory pattern. A patient may have clinically important thrombosis while routine prothrombin time (PT) and activated partial thromboplastin time (APTT) remain within the laboratory reference interval.^{6–9}

Automated haematology analysers report several platelet indices alongside the platelet count. Mean platelet volume (MPV) describes the average platelet size. Platelet distribution width (PDW) reflects variability in platelet size, while the platelet-large cell ratio (P-LCR) estimates the proportion of larger platelets. Larger platelets are often metabolically and enzymatically more active, but the interpretation of these indices is not simple. Platelet production, peripheral consumption, inflammation, marrow reserve, iron status, sample storage time, anticoagulant-related swelling and the measurement principle of the analyser can all affect results. An isolated high or low value therefore cannot be equated automatically with thrombosis, platelet activation or prognosis.^{10–12} PT primarily evaluates the tissue-factor-dependent and common pathways, whereas APTT assesses the contact-activation-dependent and common pathways under laboratory conditions. These tests are indispensable for investigating selected factor deficiencies and monitoring particular anticoagulants, but they were not designed as global screening tests for the hypercoagulable state of cancer. Their results are influenced by factor concentrations, inhibitors, anticoagulant contamination, specimen filling, citrate concentration, haematocrit, processing delay and reagent sensitivity. A normal PT or APTT does not exclude increased thrombin generation, platelet activation or venous thromboembolism.^{13–15}

Studies of platelet indices in gynaecological malignancies have produced inconsistent findings. Some investigators have reported higher platelet counts or altered MPV, PDW and plateletcrit in patients with cervical or other gynaecological

cancers, sometimes with associations with tumour stage or outcome. Other studies have observed lower MPV or no independent association after adjustment for clinical factors. Differences in disease stage, treatment status, ethnicity, inflammatory burden, analyser technology and pre-analytical handling may explain part of this heterogeneity.^{16–18}

In resource-constrained settings, indices already available from a complete blood count are attractive because they add no separate reagent cost. Their clinical value, however, depends on locally generated evidence and careful interpretation. Data from Owerri on the combined pattern of MPV, PDW, P-LCR, PT and APTT in cervical cancer are limited. This study compared these measures in women with histologically confirmed cervical cancer and apparently healthy controls. It also examined whether PT and APTT moved together among the patients.

2 Materials and Methods

2.1 Study design and setting

A hospital-based comparative cross-sectional study was conducted at the Federal Teaching Hospital, Owerri, Imo State, south-eastern Nigeria. The hospital serves patients from Owerri and surrounding communities and provides specialist gynaecology, oncology and laboratory services. The design permitted measurement of the selected haemostatic variables in patients and controls at a single period. Because exposure and outcome were assessed concurrently, the study was designed to identify group differences rather than determine temporal or causal relationships.¹⁹

2.2 Study population

The study comprised 50 adult women. The patient group included 25 women aged 18 years or older with histologically confirmed cervical carcinoma. The comparison group included 25 age-matched apparently healthy women without a history of malignancy. Controls were recruited from the community or among hospital attendees undergoing routine assessment for minor, non-inflammatory conditions.

2.3 Eligibility criteria

Patients were eligible if they were adult women with histologically confirmed cervical carcinoma, were able to provide written informed consent and had not used anticoagulant or antiplatelet medication during the preceding 14 days. Women with a known bleeding or clotting disorder, clinical evidence of acute or chronic inflammatory disease, pregnancy or another active malignancy were excluded. Additional exclusions applied to both groups included recent blood transfusion, severe hepatic or renal impairment, lactation, active systemic infection, rheumatoid arthritis and systemic lupus erythematosus. These criteria were intended to reduce non-cancer influences on platelet indices and clotting times.

2.4 Sample size

The source study calculated a minimum of approximately 25 participants per group using a two-group comparison of means, a two-sided significance level of 5%, statistical power of 80%, an assumed pooled standard deviation of 0.33 and an expected mean difference of 0.26. The calculation gave 25.24, which was rounded to 25 participants per group.

2.5 Ethical considerations

Ethical approval was obtained from the Health Research Ethics Committee of the Federal Teaching Hospital, Owerri. Permission was obtained from the relevant clinical departments before recruitment. The study purpose, procedures, possible risks and potential benefits were explained, as appropriate. Each participant gave written informed consent and was informed of the right to withdraw without affecting her care. Personal identifiers were replaced with study codes, and laboratory and clinical information was handled confidentially.

2.6 Specimen collection and processing

Participants were instructed to fast for 8-12 hours before venepuncture. After cleaning the selected site with 70% isopropyl alcohol, 5 mL of venous blood was collected from an antecubital vein with minimal tourniquet time. A 2.5 mL portion was transferred into a dipotassium ethylenediaminetetraacetic acid tube and mixed by gentle inversion for haematological analysis. The remaining 2.5 mL was placed in a 3.2% trisodium citrate tube filled to its intended volume and gently inverted. Correct filling was important because coagulation testing requires an approximate blood-to-anticoagulant ratio of 9:1.13–15

Specimens were transported promptly to the laboratory at ambient temperature. The EDTA samples were analysed soon after collection to limit time-dependent changes in platelet volume. Citrated specimens were centrifuged at approximately $1,500 \times g$ for 15 minutes to obtain platelet-poor plasma. Plasma was separated carefully without disturbing cellular material and used for PT and APTT measurement.13–15

2.7 Laboratory measurements

MPV, PDW and P-LCR were obtained using a Sysmex KX-21N automated haematology analyser. The instrument counts and sizes blood cells by electrical impedance; platelet-size distribution is used to derive the reported indices. Daily start-up procedures and internal quality-control checks were performed according to laboratory practice. Samples were mixed gently before aspiration to resuspend cells without vigorous agitation.

PT and APTT were measured in platelet-poor citrated plasma using clot-based procedures. For APTT, equal volumes of plasma and APTT reagent were incubated at 37°C before pre-warmed 0.025 mol/L calcium chloride was added, and the interval to fibrin-clot formation was recorded. PT was recorded in seconds after initiation of the extrinsic pathway with thromboplastin reagent.^{13–15}

2.8 Statistical analysis

Data were analysed with IBM SPSS Statistics. Continuous variables were summarised as mean ± standard deviation. Mean values in the cervical cancer and control groups were compared using the independent-samples t test. Pearson product-moment correlation was used to assess the relationship between PT and APTT among the 25 patients. All tests were two-sided, and p < 0.05 was considered statistically significant.

3 Results

Table 1: Platelet indices in women with cervical cancer and apparently healthy controls

All three platelet indices were significantly lower among women with cervical cancer. Mean MPV was 8.61 ± 1.06 fL in patients and 10.06 ± 0.91 fL in controls (t = 5.19; p < 0.001). Mean PDW was 11.72 ± 1.90% in patients compared with 14.18 ±

1.50% in controls (t = 5.09; p < 0.001). P-LCR was 17.88 ± 2.80% in patients and 21.12 ± 4.36% in controls (t = 3.12; p = 0.003). The direction of all three differences indicates a circulating platelet population with a lower average size, less size heterogeneity and a smaller proportion of large platelets in the patient group.

Parameter	Cervical cancer n = 25	Controls n = 25	t value	p value
MPV fL	8.61 ± 1.06	10.06 ± 0.91	5.19	<0.001*
PDW %	11.72 ± 1.90	14.18 ± 1.50	5.09	<0.001*
P-LCR %	17.88 ± 2.80	21.12 ± 4.36	3.12	0.003*

Values are mean ± standard deviation. *Statistically significant at p < 0.05. MPV, mean platelet volume; PDW, platelet distribution width; P-LCR, platelet-large cell ratio.

Table 2: Coagulation times in women with cervical cancer and apparently healthy controls

PT and APTT were slightly longer in patients, but neither difference reached statistical significance. Mean PT was 14.70 ± 1.91 seconds in patients and 14.31 ± 1.29 seconds in controls (t = 0.85; p = 0.400). Mean APTT was 34.68 ± 1.81 seconds in patients and 33.81 ± 2.10 seconds in controls (t = 1.57; p = 0.124). These results indicate that the study did not detect a large group-level alteration in the pathways measured by the two screening tests.

Parameter	Cervical cancer n = 25	Controls n = 25	t value	p value
PT seconds	14.70 ± 1.91	14.31 ± 1.29	0.85	0.400
APTT seconds	34.68 ± 1.81	33.81 ± 2.10	1.57	0.124

Values are mean ± standard deviation. APTT, activated partial thromboplastin time; PT, prothrombin time.

4 Discussion

4.1 Principal findings

This study found lower MPV, PDW and P-LCR in women with cervical cancer than in apparently healthy controls. By contrast, PT and APTT did not differ significantly between groups, and the two clotting times were not meaningfully correlated among patients. The findings point to a difference in the distribution of platelet size without evidence, from these tests, of a broad disturbance in the extrinsic, intrinsic or common coagulation pathways. The platelet findings were internally consistent because all three size-related indices changed in the same direction.

The magnitude of the MPV difference was about 1.45 fL, while PDW differed by about 2.46 percentage points and P-LCR by 3.24 percentage points. These group differences were statistically persuasive in this sample. Statistical significance, however, does not establish diagnostic usefulness.

4.2 Interpretation of the platelet indices

MPV is often described as a marker of platelet size and, indirectly, platelet reactivity. PDW reflects size variability, and P-LCR estimates the relative abundance of large platelets. A fall in all three measures may reflect a circulating population dominated by smaller and more uniform platelets. Several mechanisms are plausible. Larger or more reactive platelets may be preferentially recruited to inflamed endothelium or the tumour microenvironment. Chronic inflammation, nutritional deficiency, marrow suppression or treatment exposure may also alter thrombopoiesis. These explanations remain hypotheses because platelet production, platelet count, inflammatory markers, iron indices, treatment history and bone-

marrow function were not measured in the reported analysis.^{10–12}

The result does not fit a universal pattern of increased platelet size in malignancy. Investigations in cervical and other gynaecological cancers have reported both higher and lower platelet indices. Some studies have linked platelet-associated measures with stage, lymph-node involvement, treatment response or survival, but the specific indices and directions of association vary. A study of preoperative platelet-associated indicators in cervical cancer, for example, suggested prognostic information from composite platelet measures, while research across gynaecological tumours found potential diagnostic value in platelet count, plateletcrit, MPV and P-LCR.^{16–18} Such observations support continued research but do not make the indices interchangeable across populations or analysers.

Pre-analytical and analytical factors are especially important for MPV. EDTA can cause time-dependent platelet swelling, and the effect depends on the interval between collection and measurement. Impedance and optical analysers do not generate identical results, and manufacturers use different algorithms and reference intervals. The present study attempted prompt processing and used the same analyser for cases and controls, which reduces differential measurement error. Even so, journal publication should state the exact time to analysis, internal quality-control performance and analyser-specific reference interval so that readers can judge comparability.^{11,12}

It would be unsafe to conclude from the lower indices alone that the patients had chronic platelet

activation or consumption. Direct markers such as soluble P-selectin, platelet factor 4, beta-thromboglobulin, platelet-monocyte aggregates or flow-cytometric activation markers were not measured. Nor was thrombosis assessed clinically or by imaging. The more defensible conclusion is that platelet size distribution differed between groups. Future work can test whether that difference is related to activation, consumption, stage, treatment or outcome.

4.3 Interpretation of prothrombin time and activated partial thromboplastin time

The absence of significant PT and APTT differences is biologically credible. Cancer-associated hypercoagulability often involves tissue-factor-bearing microparticles, increased thrombin generation, impaired fibrinolysis, inflammatory activation and platelet-endothelial interactions. PT and APTT measure the time to clot formation after strong laboratory activation and are more sensitive to reduced coagulation-factor activity or inhibitors than to many prothrombotic changes. Consequently, normal or similar group means do not exclude cancer-associated thrombosis or a hypercoagulable state.^{6–9,13}

The small numerical increases in PT and APTT among patients may represent chance variation, modest factor changes or unmeasured differences in disease and nutrition. The study was not powered to detect very small differences, and confidence intervals were unavailable. Advanced cervical cancer can cause bleeding through local tumour invasion, while infection, liver dysfunction, disseminated intravascular coagulation, chemotherapy or massive transfusion can prolong

coagulation tests in selected patients. Those clinical states were either excluded or not characterised in this cohort, which may partly explain the relatively stable results.

4.4 Clinical and laboratory implications

The practical attraction of MPV, PDW and P-LCR is that they are produced during routine full blood count analysis. In a setting where specialised coagulation biomarkers are not readily available, trends in these indices may prompt closer review of the complete blood count, smear, clinical stage and treatment exposure. Their use should remain adjunctive. A single platelet index cannot determine whether a patient requires anticoagulant prophylaxis, predict bleeding or replace established clinical risk assessment. Anticoagulation decisions in cancer require consideration of tumour site, treatment, renal function, platelet count, active bleeding and patient-specific thrombotic risk.^{8,9}

Laboratories can improve the value of platelet indices by standardising collection tubes, fill volume, mixing, transport temperature and analysis time. They should establish or verify local reference intervals for the analyser in use and participate in internal and external quality assessment. For coagulation testing, the citrate concentration and blood-to-anticoagulant ratio must be controlled, platelet-poor plasma prepared appropriately, and reagent/instrument details documented. These measures are necessary before small between-group differences can be interpreted confidently.^{13–15}

5 Conclusion

Women with cervical cancer in Owerri had significantly lower MPV, PDW and P-LCR than apparently healthy women, while PT and APTT

were not significantly different. The evidence supports a difference in circulating platelet size distribution, but it does not establish the mechanism or clinical consequence of that difference. Platelet indices may be useful, inexpensive research variables and possible adjuncts to routine assessment, provided that pre-analytical conditions and analyser-specific interpretation are controlled. They should not be used alone to diagnose hypercoagulability, estimate thrombosis risk or guide anticoagulation.

6 Recommendations

Future studies should recruit larger, multicentre cohorts and report baseline clinical characteristics, tumour histology, FIGO stage and treatment status. Platelet count, plateletcrit, haemoglobin, leucocyte indices, C-reactive protein, D-dimer, fibrinogen and relevant liver and renal measures should be assessed alongside MPV, PDW and P-LCR. Prospective follow-up for venous thromboembolism, bleeding, treatment response and survival would show whether the indices have prognostic value beyond readily available clinical information.

Laboratory reports should document collection-to-

analysis time, analyser model, reagents, calibration and quality-control performance. Studies intended to develop clinical cut-offs should use an adequately powered validation cohort, report effect sizes and confidence intervals, and evaluate discrimination and calibration. Until such evidence is available, clinicians should interpret platelet indices as part of the complete clinical and laboratory picture.

Declarations

Ethics approval and consent to participate

Ethical approval was obtained from the Health Research Ethics Committee of the Federal Teaching Hospital, Owerri. Written informed consent was obtained from all participants.

Competing interests

The authors declare no competing interests.

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