Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/z45tyx93
Original Article

A Study of Evaluation of Serum Leptin and Serum Adiponectin as Biomarkers in Endometrial Carcinoma in Indian Women in a Tertiary Care Hospital: A Cross-Sectional Study.

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ABSTRACT

Background:

Endometrial carcinoma is increasingly associated with obesity, metabolic disorders, and adipokine dysregulation. Leptin and adiponectin exert opposing biological effects and may serve as non-invasive biomarkers for tumour aggressiveness and prognosis. This study evaluated serum leptin and adiponectin levels and their association with histopathological parameters in Indian women with endometrial carcinoma.

Methods:

A cross-sectional observational study was conducted at MKCG Medical College and Hospital, Berhampur, Odisha, from March 2024 to September 2025. Eighty women aged 40–70 years with histopathologically confirmed endometrial carcinoma were enrolled. Pre-treatment serum leptin and adiponectin levels were measured using enzyme-linked immunosorbent assay (ELISA). Associations with tumour grade, FIGO stage, myometrial invasion, disease progression, metastatic status, and prognosis were analyzed.

Results:

The mean serum leptin level was 9.30 ± 2.39 ng/mL, and the mean adiponectin level was 12.14 ± 4.15 µg/mL. Leptin levels increased progressively with tumour grade (Grade 1: 16.8 ± 5.1 ng/mL; Grade 3: 31.2 ± 8.1 ng/mL) and FIGO stage (Stage IA: 16.9 ± 4.8 ng/mL; Stage IIIC: 31.5 ± 8.8 ng/mL), whereas adiponectin levels decreased inversely. Patients with high disease progression had significantly higher leptin and lower adiponectin levels than those with low progression. The leptin/adiponectin ratio (LAR) was significantly higher in metastatic and poor-prognosis cases.

Conclusion:

Serum leptin, adiponectin, and particularly the leptin/adiponectin ratio were significantly associated with histopathological aggressiveness, disease progression, metastatic potential, and prognosis in Indian women with endometrial carcinoma.

Recommendation:

Larger multicenter longitudinal studies should be conducted to validate leptin, adiponectin, and the leptin/adiponectin ratio as prognostic biomarkers and establish standardized clinical cut-off values for routine use in endometrial carcinoma management.

Keywords: *Endometrial carcinoma, Leptin, Adiponectin, Leptin/Adiponectin ratio, Biomarkers, FIGO stage, India.*
Submitted: *April 22, 2026* ***Accepted:*** *May 25, 2026* ***Published:*** *June 30, 2026*

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INTRODUCTION

Endometrial carcinoma is a frequent gynaecologic malignancy globally, and there is a rising incidence associated with obesity, metabolic syndrome, and sedentary lifestyle [1]. It was once regarded as a high-income postmenopausal women's disease, but it is becoming a

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problem in developing countries, including India, where there are rapid lifestyle changes that have increased the burden of metabolic comorbidities [2]. It is now known that adipose tissue is an active endocrine organ that produces bioactive molecules also known as adipokines, which play a vital role in the regulation of metabolism, inflammation, immune function, and tumour microenvironment [3].

Leptin and adiponectin are considered to be among the most studied adipokines due to their antagonistic effects and their relationship with obesity-related cancers [4]. Leptin, a protein usually high in obese people, is pro-inflammatory, proliferative, and angiogenic and is believed to be involved in the initiation and progression of tumours by stimulating the JAK2/STAT3, PI3K/Akt, and MAPK signalling pathways [5, 6]. However, adiponectin is found in lean people and seems to have anti-inflammatory, anti-proliferative, and insulin-sensitizing effects, promoting AMPK activation and suppressive effects on mTOR activation and NF-κB activation pathways that lead to inflammation [7].

Several studies reviewed in this research have suggested that a high level of serum leptin and a low level of circulating adiponectin are independent risk factors for developing endometrial cancer and that the ratio of leptin to adiponectin (LAR) is a new composite metabolic indicator of carcinogenic risk [8, 9]. Clinical studies also have demonstrated a correlation between these adipokines and other histopathological markers of poor prognosis, including high tumour grade, advanced FIGO stage, deep invasion of the myometrium and lymphovascular space involvement [10].

Nevertheless, there is only little information available on the situation of Indian women, in particular. The association between adipokines and cancer risk may be different in Indian women who exhibit unique metabolic phenotypic characteristics such as central adiposity and insulin resistance at lower body mass index (BMI) [11]. Moreover, an increasing prevalence of endometrial carcinoma has been linked with an increasing incidence of polycystic ovary syndrome (PCOS), diabetes mellitus and metabolic syndrome in India [1]. Thus, there is a strong demand for population-specific biomarker data to enable early diagnosis and risk stratification.

The aim of this cross-sectional study was to assess the levels of serum leptin and adiponectin in women with histopathologically diagnosed endometrial carcinoma and their correlation with clinicopathological parameters, disease progression, metastatic potential and prognosis in Indian women.

MATERIALS AND METHODS

Study Design and Setting

This was a cross-sectional observational study conducted in the Department of Obstetrics and Gynaecology at Maharaja Krushna Chandra Gajapati Medical College and Hospital (MKCG MCH), Berhampur, Odisha, India, over an 18-month period from March 2024 to September 2025. The study was approved by the Institutional Ethics Committee (IEC) of MKCG Medical College (Approval No. EC/NEW/INST/2022/2934, dated 21st August 2024), and written informed consent was obtained from all participants.

Participants

Eighty women aged 40–70 years with histopathologically confirmed primary endometrial carcinoma were enrolled using convenience sampling. Diagnosis was established by endometrial biopsy and pathological examination. Patients at FIGO stages IA to IIIC were eligible. Exclusion criteria included the presence of any concurrent non-endometrial malignancy or insufficient clinical documentation to establish the diagnosis.

Study Size

The study size was determined based on the number of eligible patients diagnosed with endometrial carcinoma who attended the Department of Obstetrics and Gynaecology during the study period and fulfilled the eligibility criteria. Consecutive sampling was employed, and all eligible participants who provided informed consent were enrolled until the required sample size was achieved.

Data Collection

Demographic and clinical data, including age, weight, height, parity, menopausal status, presenting complaint, and comorbidities (diabetes mellitus and/or hypertension), were recorded. Histopathological data, including FIGO stage, histological subtype (endometrioid, serous, clear cell), tumour grade (Grade 1–3), and depth of myometrial invasion (superficial vs. deep), were confirmed by pathologists. Metastatic status and prognostic category (good vs. poor) were determined based on clinical and histopathological evaluation.

Biomarker Measurement

Fasting blood samples were collected from each participant prior to any treatment. Serum was separated and stored at −80°C until analysis. Serum leptin (ng/mL) and adiponectin (µg/mL) were measured using enzyme-linked immunosorbent assay (ELISA) kits using the Thermo

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Scientific Varioskan LUX multimode microplate reader, following the manufacturer's instructions. The leptin/adiponectin ratio (LAR) was calculated for each participant.

Outcome Variables

The **primary outcome** of the study was the association of serum leptin and serum adiponectin concentrations with histopathologically confirmed carcinoma endometrium.

The **secondary outcomes** included the relationship of serum leptin and adiponectin levels with clinicopathological characteristics of carcinoma endometrium, including tumour grade, stage, body mass index (BMI), menopausal status, and other relevant demographic and clinical variables. Serum leptin and adiponectin concentrations were measured using standardized enzyme-linked immunosorbent assay (ELISA) techniques according to the manufacturer's instructions.

Bias

Several measures were undertaken to minimize potential sources of bias. Consecutive eligible patients presenting during the study period were recruited to reduce selection bias. Standardized inclusion and exclusion criteria were applied uniformly to all participants. Laboratory analyses of serum leptin and adiponectin were performed using validated assays following standardized operating procedures to minimize measurement bias. Clinical and laboratory data were collected using a pre-designed data collection form by trained investigators to ensure consistency and reduce observer bias. Statistical analyses were conducted using predefined analytical methods to minimize analytical bias.

Ethical approval

The study was approved by the Institutional Ethics Committee of MKCG Medical College, Berhampur (Approval No. EC/NEW/INST/2022/2934, 21st August 2024). Informed consent was obtained from all participants.

Statistical Analysis

Descriptive statistics (mean ± standard deviation, frequencies, percentages) were used to summarize patient characteristics. The Pearson correlation coefficient (r) was calculated to evaluate the relationship between serum leptin and adiponectin. Between-group comparisons for biomarker levels across histopathological parameters, disease progression categories, metastatic status, and prognostic groups were performed. A p-value of <0.05 was considered statistically significant.

RESULTS

Participant Recruitment

During the study period, women attending the Department of Obstetrics and Gynaecology with suspected endometrial pathology were screened for eligibility. Participants fulfilling the predefined inclusion criteria were invited to participate in the study. After obtaining written informed consent, eligible participants underwent clinical evaluation, laboratory investigations, and histopathological confirmation where indicated. Only participants with complete clinical and laboratory data were included in the final analysis.

Demographic and Clinical Characteristics

The study enrolled 80 women with confirmed endometrial carcinoma. The majority (55%) were aged 50–59 years, followed by 30% in the 60–69 year group and 15% in the 40–49 year group. With regard to body mass index (BMI), 41.25% were overweight, and 38.75% were obese, with only 20% falling in the normal BMI range. Seventy percent of patients were postmenopausal, 20% perimenopausal, and 10% premenopausal. Postmenopausal bleeding (PMB) was the presenting complaint in 80% of cases, while abnormal uterine bleeding (AUB) accounted for 20%. In terms of comorbidities, 35% had hypertension alone, 15% had diabetes mellitus alone, 35% had both, and 15% had no comorbidities (Table 1).

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Table 1: Demographic and Clinical Characteristics of Study Participants (n = 80)

Variable	Category	n (%)
Age Group (years)	40–49	12 (15%)
	50–59	44 (55%)
	60–69	24 (30%)
BMI Category	Normal	16 (20%)
	Overweight	33 (41.25%)
	Obese	31 (38.75%)
Menopausal Status	Premenopausal	8 (10%)
	Perimenopausal	16 (20%)
	Postmenopausal	56 (70%)
Comorbidities	HTN only	28 (35%)
	DM only	12 (15%)
	DM + HTN	28 (35%)
	None	12 (15%)

Histopathological Distribution

The most common FIGO stage was Stage IA (37.5%), followed by Stage IB (26.25%), Stage IIB (13.75%), Stage IIA (7.5%), and Stages IIIA, IIIB, and IIIC at 5% each. Endometrioid carcinoma was the predominant histological subtype (75%), followed by serous (20%) and clear cell (5%) carcinomas.

Descriptive Statistics for Serum Leptin and Adiponectin

The mean serum leptin level was 9.30 ± 2.39 ng/mL (range: 5.30–13.95 ng/mL), and the mean serum adiponectin was 12.14 ± 4.15 µg/mL (range: 5.3–19.9 µg/mL). Pearson correlation analysis between serum leptin and adiponectin revealed a weak but statistically significant negative correlation ($r = -0.12$, $p < 0.05$), and the mean LAR was 0.83 (Table 2).

Table 2: Descriptive Statistics for Serum Leptin and Adiponectin (n = 80)

Biomarker	Mean ± SD	Minimum	Maximum
Serum Leptin (ng/mL)	9.30 ± 2.39	5.30	13.95
Serum Adiponectin (µg/mL)	12.14 ± 4.15	5.30	19.90
Leptin/Adiponectin Ratio	0.83 (mean)	—	—

Association with Histopathological Parameters

A consistent and statistically significant trend was observed between the adipokine profile and histopathological severity. Serum leptin increased progressively from Grade 1 (16.8 ± 5.1 ng/mL) to Grade 3 (31.2 ± 8.1 ng/mL) tumours, while adiponectin declined from 12.4 ± 3.0 µg/mL to 7.6 ± 2.5 µg/mL, resulting in a rising LAR (1.35 to 4.11). A similar pattern was observed across FIGO stages, with leptin rising from 16.9 ± 4.8 ng/mL at Stage IA to 31.5 ± 8.8 ng/mL at Stage IIIC, and adiponectin falling from 12.8 ± 2.9 to 6.8 ± 1.9 µg/mL. Deep myometrial invasion was also associated with significantly higher leptin (28.7 ± 7.6 ng/mL) and

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lower adiponectin (8.2 ± 2.4 µg/mL) compared to superficial invasion (Table 3).

Table 3: Association Between Serum Leptin, Adiponectin, and LAR with Histopathological Parameters

Histopathological Parameter	Serum Leptin (Mean ± SD, ng/mL)	Serum Adiponectin (Mean ± SD, µg/mL)	Leptin/Adiponectin Ratio
Tumour Grade			
Grade 1 (Well differentiated)	16.8 ± 5.1	12.4 ± 3.0	1.35
Grade 2 (Moderately differentiated)	22.6 ± 6.4	11.2 ± 3.9	2.02
Grade 3 (Poorly differentiated)	31.2 ± 8.1	7.6 ± 2.5	4.11
FIGO Stage			
IA	16.9 ± 4.8	12.8 ± 2.9	1.32
IB	22.1 ± 6.5	11.8 ± 3.4	1.87
IIA	24.6 ± 7.0	9.4 ± 3.1	2.62
IIB	25.1 ± 7.2	9.1 ± 3.2	2.76
IIIA	27.9 ± 8.0	8.3 ± 2.4	3.36
IIIB	28.4 ± 8.3	7.9 ± 2.3	3.60
IIIC	31.5 ± 8.8	6.8 ± 1.9	4.63
Depth of Myometrial Invasion			
Superficial (<50%)	19.6 ± 5.8	14.6 ± 3.8	1.34
Deep (≥50%)	28.7 ± 7.6	8.2 ± 2.4	3.50

Serum Leptin and Adiponectin as Predictive Biomarkers for Disease Progression

Patients with high disease progression had significantly higher serum leptin (28.3 ± 7.6 ng/mL vs. 18.5 ± 5.2 ng/mL; p <0.01) and lower adiponectin (8.1 ± 3.0 µg/mL vs. 15.2 ± 4.5 µg/mL; p = 0.015) compared to low disease progression. The LAR rose significantly from 1.21 ± 0.35 to 3.54 ± 1.02 (p <0.05) (Table 4).

Table 4: Serum Leptin, Adiponectin, and LAR as Predictive Biomarkers for Disease Progression

Biomarker	Low Progression (Mean ± SD)	High Progression (Mean ± SD)	p-value
Serum Leptin (ng/mL)	18.5 ± 5.2	28.3 ± 7.6	<0.01
Serum Adiponectin (µg/mL)	15.2 ± 4.5	8.1 ± 3.0	0.015
Leptin/Adiponectin Ratio	1.21 ± 0.35	3.54 ± 1.02	<0.05

Metastatic Potential and Prognosis

Metastatic cases showed significantly higher serum leptin (30.5 ± 8.3 ng/mL vs. 18.2 ± 5.0 ng/mL; p <0.05) and lower adiponectin (7.8 ± 2.5 µg/mL vs. 13.4 ± 3.7 µg/mL; p = 0.05), with a significantly elevated LAR (3.91 ± 1.10 vs. 1.40 ± 0.35; p <0.01). Patients with poor prognosis exhibited

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higher leptin (28.3 ± 7.5 ng/mL vs. 19.1 ± 5.3 ng/mL; p <0.05) and lower adiponectin (6.4 ± 2.8 µg/mL vs. 12.8 ± 3.5 µg/mL; p = 0.06), with a markedly elevated LAR (4.34 ± 1.20 vs. 1.50 ± 0.45; p <0.05) (Table 5).

Table 5: Serum Leptin, Adiponectin, and LAR in Relation to Metastatic Potential and Prognosis

Clinical Outcome	Serum Leptin (ng/mL)	Serum Adiponectin (µg/mL)	Leptin/Adiponectin Ratio	p-value (LAR)
Metastatic	30.5 ± 8.3	7.8 ± 2.5	3.91 ± 1.10	<0.01
Non-metastatic	18.2 ± 5.0	13.4 ± 3.7	1.40 ± 0.35	
Poor prognosis	28.3 ± 7.5	6.4 ± 2.8	4.34 ± 1.20	<0.05
Good prognosis	19.1 ± 5.3	12.8 ± 3.5	1.50 ± 0.45	

DISCUSSION

A cross-sectional study was done to assess the potential of serum leptin, adiponectin, and LAR as biomarkers in the 80 women of Indian origin diagnosed with endometrial carcinoma by histopathology. The results confirm and show the strong and regular relationships between the unfavourable adipokine profile (high leptin, low adiponectin, and elevated LAR) and increasing histopathological severity, disease progression, metastatic potential, and poor prognosis.

The majority of patients (55%) were in the age range of 50-59 years, as this is the peak age group for endometrial carcinoma. Ashizawa et al. reported a mean age of 58.6 ± 6.4 years in their study of endometrial cancer patients [12] and Jayraj et al reported a similar age distribution (mean of 56.9 ± 8.1 years) [10]. The present study involved 80% of postmenopausal women, which is the expected hormonal environment for the development of endometrial cancers by the unopposed stimulation of oestrogen by peripheral aromatase activity from adipose tissue [13].

This indicates that there is a high prevalence of overweight and obese patients (around 80%) in this cohort and indicates an inherent strong metabolic pathology of endometrial carcinoma in the Indian context. There are reports of a significantly higher mean BMI in endometrial cancer patients than controls (27.8 ± 3.6 kg/m² vs. 23.4 ± 2.9 kg/m²) [14] and a meta-analysis by Gong et al. showed that an increase in BMI is consistently linked to an increase in the risk of developing endometrial cancer, with higher BMI levels corresponding to higher leptin and lower adiponectin levels in the blood [9]. Słabuszewska-Jóźwiak et al. highlight that the dysregulation of adipokines is an important mechanism in the initiation and progression of tumours in obesity [4].

In the current study, the level of leptin in the serum increased gradually with increasing grade; it was highest in the poorly differentiated tumour group at 31.2 ± 8.1 ng/mL, and its level was lowest at 7.6 ± 2.5 µg/mL among the poorly differentiated tumour group. The results of this study align with Jayraj et al., who showed that increased serum leptin levels were associated with lymphovascular space involvement, deeper myometrial invasion, and higher grade in endometrial carcinoma [10]. Leptin directly contributes to tumour aggressiveness by activation of JAK2/STAT3, leading to epithelial-to-mesenchymal transition (EMT) and tumour invasiveness [5, 6]. There is evidence that adiponectin inhibits carcinogenesis through activation of AMPK and suppression of mTOR signalling and inflammation through inhibition of NF-κB [7, 15], and this is supported by the progressive decrease with tumour grade. This stage-wise analysis revealed that at Stage IIIC, leptin level (31.5 ± 8.8 ng/mL) was almost twice as high as at Stage IA (16.9 ± 4.8 ng/mL) and adiponectin level (12.8 ± 2.9 µg/mL) was almost half as high as at Stage IA (6.8 ± 1.9 µg/mL) during the same stage. Yabushita et al. reported that advanced-stage poorly differentiated tumours showed leptin levels > 25 ng/mL and adiponectin levels < 8 µg/mL, with changes in receptor expression in tumour tissue [16]. Similarly, for stage III disease, Jayraj et al. reported a mean leptin level of more than 30 ng/mL and adiponectin below 7.5 µg/mL [10], which is close to the current study.

Significant differences in LAR, with disease progression data showing over a 2-fold increase in LAR in the patients with high progression (3.54 ± 1.02 vs. 1.21 ± 0.35; p <0.05). This is in line with Ashizawa et al., who found that LAR more than 3.5 was significantly linked to poor clinical outcomes in postmenopausal women with endometrial cancer [12]. The mechanistic basis for this observation lies

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in the interplay between the growth-promoting effects of leptin-induced proliferative signalling and the growth-inhibitory effects of adiponectin-induced AMPK-dependent tumour suppression, resulting in a metabolic microenvironment that is conducive to aggressive behaviour of the tumour [4].

A particularly significant finding is the marked increase in LAR of metastatic cases (3.91 ± 1.10 vs. 1.40 ± 0.35; p <0.01). The LAR ranged from 3.2 to 4.1 in advanced and metastatic endometrial disease as reported by Gong et al. [9], and 7.6 ± 2.7 µg/mL of adiponectin and 29.4 ± 8.1 ng/mL of leptin were seen in metastatic endometrial disease as reported by Jayraj et al. [10], similar to the present cohort. Leptin and its activation of the SIRT1-HMMR signalling axis are associated with metastatic behaviour by inducing cell motility and EMT [17], while adiponectin deficiency is linked with tumour progression through activation of the MAPK pathway [18]. The LAR thus reflects the combined detrimental influence of hyperleptinemia and hypoadiponectinemia on the metastatic potential.

From a prognosis point of view, the patients with poor prognosis had a significantly higher LAR value compared to the patients with good prognosis (4.34 ± 1.20 vs. 1.50 ± 0.45; p <0.05). In endometrial cancer, Słabuszewska-Jóźwiak et al. found a link between low levels of adiponectin (less than 9 µg/mL) and poor prognosis, combined with high levels of leptin (more than 25 ng/mL) [4]. In a study specifically designed for the Indian context, Rajaram et al, also found that elevated leptin and reduced adiponectin were effective metabolic predictors for endometrial cancer risk along with anthropometric and metabolic parameters [1]. In resource-limited settings in India, the LAR thus has the potential to be useful for cost-effective, non-invasive biomarkers for risk stratification.

In the present study, the authors found that it is one among the few studies in India that have evaluated both adipokines systematically across the various clinicopathological parameters in a single cohort. The homogeneity of the study population and the standardized measurement of ELISAs are added advantages for the results. Several limitations should be noted, however. This was a cross-sectional design, and there were no data on recurrence or overall survival from a longitudinal follow-up. There is limited statistical power for subgroup analyses due to the small sample size of 80. Dietary, physical activity, and genetic factors were not assessed as possible confounders. Standardized clinical cut-off values of leptin and adiponectin in the Indian population must be determined by conducting larger multi-centre studies.

GENERALIZABILITY

The findings of this study may be applicable to women with carcinoma endometrium attending tertiary care hospitals that serve populations with demographic and clinical characteristics similar to those of the present study. However, because the study was conducted at a single tertiary care centre using a cross-sectional design, the results should be generalized cautiously to other populations with different ethnic, socioeconomic, and healthcare characteristics. Multicentre studies involving larger and more diverse populations are needed to improve the external validity of these findings.

LIMITATIONS

This study has several limitations. First, the cross-sectional design limits the ability to establish causal relationships between serum leptin, serum adiponectin, and disease progression. Second, the study was conducted at a single tertiary care centre with a relatively small sample size, which may limit the generalizability of the findings. Third, only a single measurement of serum leptin and adiponectin was obtained, and changes in biomarker levels over time could not be evaluated. Fourth, potential confounding factors such as dietary habits, physical activity, and other metabolic variables were not comprehensively assessed. Finally, although standardized laboratory methods were employed, residual measurement and selection bias cannot be completely excluded.

RECOMMENDATIONS

Based on the findings of this study, serum leptin and serum adiponectin may serve as useful adjunctive biomarkers for assessing disease severity and prognosis in women with endometrial carcinoma. Their combined assessment, particularly the leptin/adiponectin ratio, may improve risk stratification when used alongside established clinicopathological parameters. However, these biomarkers should complement rather than replace existing diagnostic and prognostic methods. Further large-scale prospective multicentre studies are recommended to validate these findings and determine their role in routine clinical practice.

CONCLUSION

This study shows that the level of serum leptin, adiponectin, and the leptin/adiponectin ratio is significantly and progressively correlated with histopathological aggressiveness, disease progression, metastatic potential, and unfavourable prognosis in Indian women with carcinoma endometrium. It is hoped that these adipokines can become useful, easily accessible, and noninvasive

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markers to complement clinical decision-making, risk stratification, and personalized management strategies.

Acknowledgements

The authors are grateful to the Department of Obstetrics & Gynaecology and the Department of Biochemistry, MKCG MCH, Berhampur, for facilitating this research. We sincerely thank all patients who participated in this study.

Conflict of interest

The authors declare no conflict of interest.

Funding

This study received no external funding.

Author contributions

Sabyasachi Padhi: Conceptualization, methodology, patient recruitment, data collection, data curation, formal analysis, manuscript drafting, literature review, and manuscript revision.

Mahija Sahu: Study conceptualization, supervision, methodology, validation of clinical findings, critical review of the manuscript, and final approval of the manuscript.

Anshuman Jena: Data collection, patient recruitment, clinical investigations, data interpretation, literature review, and manuscript editing.

Janmejaya Mohapatra: Study design, supervision, interpretation of results, critical revision of the manuscript for important intellectual content, project administration, and final approval of the manuscript.

Nirupama Devi: Laboratory investigations, biochemical analysis, validation of laboratory findings, data interpretation, and critical review of the manuscript.

All authors read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/z45tyx93
Original Article

PUBLISHER DETAILS

Student's Journal of Health Research (SJHR)
(ISSN 2709-9997) Online
(ISSN 3006-1059) Print
Category: Non-Governmental & Non-profit Organization
Email: studentsjournal2020@gmail.com
WhatsApp: +256 775 434 261
Location: Scholar's Summit Nakigalala, P. O. Box 701432, Entebbe Uganda, East Africa