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A Study of Evaluation of Serum Leptin and Serum Adiponectin as Biomarkers in Endometrial Carcinoma Endometrium 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 altered adipokine levels. Leptin and adiponectin may have potential roles as biomarkers of tumour progression and prognosis. This study evaluated serum leptin and adiponectin levels and their association with clinicopathological characteristics in Indian women with endometrial carcinoma.

Methods:

This cross-sectional observational study was conducted from March 2024 to September 2025 at MKCG Medical College and Hospital, Berhampur, Odisha. Eighty women aged 40–70 years with histopathologically confirmed endometrial carcinoma were enrolled. Pretreatment serum leptin and adiponectin levels were measured using enzyme-linked immunosorbent assay. Associations with tumour grade, FIGO stage, disease progression, metastatic status, and prognosis were assessed. Statistical significance was set at $p<0.05$.

Results:

The mean serum leptin was 9.30 ± 2.39 ng/mL and mean adiponectin was 12.14 ± 4.15 µg/mL. Serum leptin increased 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 decreased inversely. High disease progression was associated with higher leptin (28.3 ± 7.6 ng/mL) and lower adiponectin (8.1 ± 3.0 µg/mL; $p<0.01$ and $p=0.015$, respectively). The leptin/adiponectin ratio (LAR) was significantly higher in metastatic versus non-metastatic cases (3.91 ± 1.10 vs. 1.40 ± 0.35; $p<0.01$) and in poor versus good prognosis (4.34 ± 1.20 vs. 1.50 ± 0.45; $p<0.05$).

Conclusion:

Serum leptin, adiponectin, and particularly LAR were associated with disease severity, histopathological aggressiveness, metastatic potential, and prognosis.

Recommendation:

These adipokines may be considered as potential adjunctive biomarkers for risk stratification, but larger multicenter longitudinal studies are required to establish population-specific clinical cut-offs and validate their utility.

Keywords: *Endometrial carcinoma, Leptin, Adiponectin, Leptin/Adiponectin ratio, Biomarkers, FIGO stage, India*
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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 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, Maharaja Krushna Chandra Gajapati Medical College and Hospital (MKCG MCH), Berhampur, Odisha, India, over an 18-month period from **March 2024 to September 2025**. MKCG MCH is a tertiary-care teaching hospital providing comprehensive obstetric and gynaecological services, including the evaluation and management of gynaecological malignancies, histopathological diagnosis, surgical care, and laboratory investigations.

The study included women aged 40–70 years with histopathologically confirmed primary endometrial carcinoma. Diagnosis was established by endometrial biopsy and pathological examination. Patients with FIGO stages IA to IIIC were eligible. Women with concurrent non-endometrial malignancy or insufficient clinical documentation to establish the diagnosis were excluded. Participants were enrolled using convenience sampling.

Ethical Considerations

The study was approved by the **Institutional Ethics Committee of MKCG Medical College, Berhampur** (Approval No. **EC/NEW/INST/2022/2934**, dated **21 August 2024**). Written informed consent was obtained from all participants before enrolment. The study was conducted in accordance with the ethical principles applicable to research involving human participants.

Study Size

A total of **80 women** with histopathologically confirmed endometrial carcinoma were included in the study. Participants were recruited using **convenience sampling** during the study period from March 2024 to September 2025. All women who fulfilled the predefined eligibility criteria and provided informed consent were considered for enrolment, and a final sample of 80 participants was obtained for analysis.

Measures to Minimize Bias

Potential sources of bias were addressed by applying predefined inclusion and exclusion criteria and enrolling participants with **histopathologically confirmed primary endometrial carcinoma**. Clinical and demographic information was collected using predefined study variables, while tumour grade, histological subtype, FIGO stage, and depth of myometrial invasion were confirmed by pathological examination.

To minimize measurement bias, serum leptin and adiponectin concentrations were measured using standardized **enzyme-linked immunosorbent assay (ELISA)** procedures with a Thermo Scientific Varioskan LUX multimode microplate reader, following the manufacturer's instructions. Serum samples were obtained before treatment, and the leptin/adiponectin ratio (LAR) was calculated using the measured biomarker concentrations.

Because **convenience sampling** was used, some degree of selection bias cannot be completely excluded. This limitation has been acknowledged and should be considered when interpreting the generalizability of the findings.

Statistical Analysis

Descriptive statistics were used to summarize patient characteristics and biomarker concentrations. Continuous variables were expressed as mean ± standard deviation, and categorical variables were presented as frequencies and percentages. Pearson correlation analysis was used to assess the relationship between serum leptin and adiponectin. Between-group comparisons were performed according to

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histopathological parameters, disease progression, metastatic status, and prognostic categories. A *p*-value <0.05 was considered statistically significant.

RESULTS

Participant Recruitment

Women attending the Department of Obstetrics and Gynaecology, MKCG Medical College and Hospital, Berhampur, Odisha, during the study period from **March 2024 to September 2025** were assessed for eligibility. Women aged 40–70 years with **histopathologically confirmed primary endometrial carcinoma** were considered eligible for participation. Patients with concurrent non-endometrial malignancy or insufficient clinical documentation to establish the diagnosis were excluded. Eligible women who provided written informed consent were enrolled using **convenience sampling**. A total of **80 women** fulfilled the study requirements and 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).

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%)

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

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).

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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)	—	—

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

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

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

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

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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).

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

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

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 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).

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	

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

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

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Original Article

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 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 histopathologically confirmed endometrial carcinoma receiving care at tertiary-care hospitals with access to gynaecological oncology services, histopathological evaluation, and biochemical testing. The study provides evidence of an association between serum leptin, adiponectin, and the leptin/adiponectin ratio and important clinicopathological characteristics of endometrial carcinoma in an Indian population. However, the external validity of the findings should be interpreted cautiously because the study was conducted at a single tertiary-care centre and included a relatively small sample of 80 participants recruited using convenience sampling. These characteristics may limit the applicability of the findings to women from other geographic regions, healthcare settings, or populations with different demographic and metabolic characteristics. Larger multicentre studies involving more diverse Indian populations are required to validate these findings and determine whether similar associations are observed across different settings.

Limitations

This study has several limitations. First, its **cross-sectional observational design** limits the ability to establish a temporal or causal relationship between serum adipokine levels and tumour progression or prognosis. Second, the relatively small sample size of 80 participants may have limited the statistical power of subgroup analyses. Third, the use of **convenience sampling** may have introduced selection bias and limits the generalizability of the findings. Fourth, potential confounding factors related to metabolic

status, lifestyle, dietary patterns, and other patient characteristics were not comprehensively evaluated. Finally, the study did not provide longitudinal assessment of recurrence, disease-free survival, or overall survival. Therefore, the findings should be interpreted as preliminary and require validation through larger, multicentre prospective studies.

Recommendation

Based on the findings of this study, serum leptin, adiponectin, and particularly the **leptin/adiponectin ratio (LAR)** may have potential as adjunctive biomarkers for assessing disease severity and identifying patients with more aggressive clinicopathological features. However, these biomarkers should not currently replace established histopathological and clinical prognostic parameters. Further large-scale, multicentre and prospective studies are recommended to validate these associations, establish clinically relevant cut-off values, and determine whether incorporating adipokine measurements into existing risk-stratification models improves clinical decision-making.

Conclusion

This study shows that the level of serum leptin, adiponectin, and, significantly, the leptin/adiponectin ratio is significantly and progressively correlated with histopathological aggressiveness, disease progression, metastatic potential, and unfavourable prognosis in Indian women with endometrial carcinoma. It is hoped that these adipokines can become useful, easily accessible, and noninvasive markers to complement clinical decision-making, risk stratification, and personalized management strategies. A large-scale longitudinal validation study to set up the validated cut-off values for routine clinical use in India is recommended.

List of Abbreviations

Abbreviation	Full form
AMPK	AMP-activated protein kinase
AUB	Abnormal uterine bleeding
BMI	Body mass index
ELISA	Enzyme-linked immunosorbent assay
FIGO	International Federation of Gynecology and Obstetrics
HTN	Hypertension
JAK2	Janus kinase 2
LAR	Leptin/adiponectin ratio
MAPK	Mitogen-activated protein kinase
mTOR	Mechanistic target of rapamycin
NF-κB	Nuclear factor kappa B
PCOS	Polycystic ovary syndrome
PI3K/Akt	Phosphoinositide 3-kinase/protein kinase B
PMB	Postmenopausal bleeding
SD	Standard deviation
STAT3	Signal transducer and activator of transcription 3

Author Contributions

Sabyasachi Padhi: Conceptualization, study design, participant recruitment, data collection, data analysis, literature review, manuscript drafting, and final approval of the manuscript.

Mahija Sahu: Study supervision, clinical guidance, interpretation of findings, critical revision of the manuscript, and final approval of the manuscript.

Anshuman Jena: Participant recruitment, data collection, literature review, interpretation of results, critical revision of the manuscript, and final approval.

Janmejaya Mohapatra: Study supervision, gynaecological oncology expertise, interpretation of clinical and histopathological findings, critical revision of the manuscript, and final approval.

Nirupama Devi: Biochemical analysis, laboratory supervision, interpretation of serum leptin and adiponectin results, critical revision of the manuscript, and final approval.

Important: These roles should be verified with the authors before submission because the source manuscript does not explicitly document each author's individual contribution.

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Before submission: Add each author's **actual qualifications** (e.g., MBBS, MD/MS, DNB, PhD) to these biographies after confirming them with the authors.

Data Availability

The datasets generated and/or analyzed during the current study are not publicly available because they contain participant-level clinical and laboratory information. The data may be made available from the corresponding author upon reasonable request, subject to institutional approval and applicable confidentiality requirements.

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Conflict of interest

The authors declare no conflict of interest.

Funding

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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.

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