International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Kumar D et al. Int J Reprod Contracept Obstet Gynecol. 2026 Sep;15(9):3449-3456
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20263025

Original Research Article

Spectrum of uterine malignancies and their oncological outcomes: a single centre experience

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Received: 14 May 2026
Revised: 16 August 2026
Accepted: 17 August 2026

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ABSTRACT

Background: Endometrial carcinoma is a significant contributor to the global female cancer burden. Its incidence in India is rising because of increasing life expectancy and metabolic risk factors, although Indian outcome data remain limited. This study aimed to evaluate the spectrum of uterine malignancies and assess their oncological outcomes in an Indian cohort.
Methods: This prospective observational study was conducted at a tertiary care centre over a five-year period (2020–2025). Seventy (70) women were included following histopathological confirmation of uterine malignancy. Data on clinical presentation, histological subtype, staging, treatment, and outcomes were recorded and analysed using descriptive statistics.
Results: The mean age was in the sixth decade, with 88.6% of patients being postmenopausal. Endometrioid adenocarcinoma was the most common histological subtype (75.7%), followed by serous carcinoma (7.1%), carcinosarcoma (4.3%), and clear cell carcinoma (4.3%). Most patients presented at FIGO Stage I (61.5%) and underwent primary surgical management. Adjuvant therapy was administered in 32.9% of cases based on stage and risk factors. At a median follow-up, 72.9% of patients were alive and recurrence was observed in only 1.4%. Binary logistic regression showed that none of the individual pathological factor's tumour grade, histology, LVSI, myometrial invasion, or cervical stromal involvement significantly predicted lymph node involvement.
Conclusion: Endometrioid carcinoma remains the predominant uterine malignancy in Indian women, typically presenting in postmenopausal women at an early stage with favourable outcomes. Given the inability of conventional pathological risk factors to reliably predict nodal involvement, comprehensive surgical staging using sentinel lymph node mapping or systematic lymphadenectomy should be considered in all cases, irrespective of initial stage.

Keywords: Uterine malignancy, Endometrial carcinoma, Oncological outcomes, Histopathology, Postmenopausal bleeding

INTRODUCTION

Uterine malignancies, particularly endometrial cancer, pose a significant global health challenge.[1] The spectrum also includes rare but aggressive uterine sarcomas.[2,3] As a group, uterine malignancies are heterogeneous, with distinct histological, molecular, and clinical characteristics. Endometrial cancer is broadly classified into Type 1 and Type 2 disease.[4] Molecular classification for instance, POLE-mutated or p53-abnormal tumours further refines prognosis and guides treatment.[5,6] The reported incidence of endometrial cancer in India is lower than the global median, although population-based registry coverage remains uneven.[7] Concurrently, the rising prevalence of obesity and diabetes among Indian women suggests a future shift towards lifestyle-associated endometrial cancers.[8] Understanding oncological outcomes is vital for prognostication and treatment

planning: for endometrial cancer, five-year survival for localized disease is high but falls sharply for advanced (Stage IV) disease.[9,10] Despite this burden, comprehensive clinical data and robust public health infrastructure for uterine malignancies in India remain underdeveloped.[11] This study aims to comprehensively characterize the spectrum of uterine malignancies in an Indian cohort, analyse their oncological outcomes, and identify factors influencing those outcomes within the Indian healthcare context.

METHODS

This was a hospital-based, prospective, single-centre observational study designed to evaluate the clinicopathological spectrum and oncological outcomes of uterine malignancies, with a specific focus on endometrial carcinoma. It was conducted at the Department of Obstetrics and Gynecology, All India Institute of Medical Sciences, Jodhpur, Rajasthan, India, over a period of five years commencing in 2020, following approval from the Institutional Ethics Committee.

All consecutive female patients presenting to the outpatient or emergency services with symptoms suggestive of uterine malignancy particularly abnormal uterine bleeding or postmenopausal bleeding were screened for eligibility. Women subsequently diagnosed with endometrial carcinoma on histopathological examination of endometrial biopsy specimens were enrolled after obtaining informed written consent. Patients were excluded if they were diagnosed with other types of uterine malignancy, such as sarcomas or non-epithelial tumours.

Cases with incomplete medical records, those lost to follow-up prior to initiation of treatment, and those unwilling to participate were excluded from the final analysis. Following histopathological confirmation, patients underwent metastatic work-up and were staged according to the international federation of gynecology and obstetrics (FIGO) staging system. Chemotherapy regimens followed institutional protocols and generally comprised platinum-based combinations.

Oncological outcomes such as recurrence and survival were recorded and analysed. Descriptive statistics were used to summarize demographic and clinical variables. Binary logistic regression was performed to identify factors associated with nodal positivity, with a p value<0.05 considered statistically significant.

RESULTS

The study included a total of 70 participants diagnosed with uterine malignancies. Table 1 summarizes the demographic and clinical profile. The most common age group was 50–59 years (31.4%), followed by 60–69 years (30.0%) and 70–79 years (21.4%). A majority of patients (88.6%) were postmenopausal, with only 11.4% being premenopausal. The predominant presenting complaint was postmenopausal bleeding (65.7%), followed by heavy menstrual bleeding (18.6%), discharge per vaginum (5.7%), and abdominal pain (5.7%). One patient each presented with abnormal uterine bleeding, haematuria, and post-hysterectomy endometrial stromal sarcoma. The most frequently observed comorbidities were hypertension (50.0%) and diabetes mellitus (31.4%). Obesity was noted in 14.3% of cases, while 37.1% of patients had no identifiable risk factor. The mean duration of symptoms was 11.2 months (SD 16.2), and the mean uterine size was 10.5 cm (SD 4.1).

Table 2 presents the histopathological and radiological characteristics. Endometrioid adenocarcinoma was the most common biopsy finding, reported in 75.7% of patients. Other histologies included pre-malignant or miscellaneous lesions (8.6%), serous carcinoma (5.7%), carcinosarcoma or malignant mixed Müllerian tumour (4.3%), clear cell carcinoma (2.9%), and endometrial stromal sarcoma (2.9%). Myometrial invasion greater than 50% was noted in 52.9% of patients, 35.7% had less than 50% invasion, and 11.4% had no invasion. Lymph node involvement was absent in 75.7% of patients. Among the 17 patients with nodal involvement, the most common site was bilateral pelvic nodes (11.4%), followed by unilateral pelvic (5.7%), bilateral inguinal (2.9%), and paraaortic with pelvic or paratracheal nodes (2.9% and 1.4% respectively). Ascitic fluid cytology was positive in 4.3% of cases. Lymphovascular space invasion (LVSI) was noted in 17.1% of patients, absent in 80.0%, and not assessed in 2.9%.

Staging and surgical details are presented in Table 3. On pre-operative imaging, the most common stage was FIGO Stage IA (41.4%), followed by IB (28.6%) and Stage II (11.4%). Post-operative staging showed a modest redistribution, with 32.9% in Stage IA, 28.6% in Stage IB, and 17.1% in Stage II. Advanced stages (IIIA to IVB) were relatively infrequent, with only three patients in Stage IVB and the remainder scattered across intermediate stages.

Table 4 displays immunohistochemistry and final histopathological reports. Immunohistochemistry (IHC) was performed in 37.1% of cases, of which tumour suppressor, mesenchymal, or Müllerian markers were most frequently used (25.7%). Hormonal markers or carcinoembryonic antigen (CEA) were used in 8.6%, and sarcoma- or MMMT-specific markers in 2.8%. IHC was not done or not available in 62.9% of cases. Final histopathology confirmed endometrioid carcinoma in 75.7% of patients, while sarcomas and MMMTs accounted for 8.6%, serous carcinoma for 7.1%, clear cell carcinoma for 4.3%, and other aggressive subtypes for 4.3%. Histological grading was evenly distributed between Grade I and Grade II (40% each), with 20% of tumours classified as Grade III.

Details of adjuvant therapy and follow-up are summarized in Table 5. Adjuvant chemotherapy was administered to

17.1% of patients, while radiotherapy and vaginal brachytherapy were each given to 7.1%; only one patient received combined chemotherapy and radiotherapy. A majority (67.1%) received no adjuvant therapy. Follow-up data were available for 72.9% of patients, 21.4% were lost to follow-up, and 5.7% had died during the study period. Table 5 also outlines oncological outcomes. At last follow-up, 72.9% of patients were alive, 5.7% had died, and data were unavailable for 21.4% of the cohort. Recurrence was documented in only one patient (1.4%); the remaining 98.6% had no evidence of recurrence. Among the 70 patients, 44 (62.9%) underwent laparoscopic surgery and 26 (37.1%) underwent open surgery. Survival outcomes were significantly better in the laparoscopic group, with 100% (32/32) of followed-up patients surviving, compared with 82.6% (19/23) in the open-surgery group. All four reported deaths occurred in the open-surgery group, although none were related to the surgical procedure itself. The difference in survival between the two groups was statistically significant (Pearson's chi-square=6.002, p=0.014; Fisher's exact test p=0.026). In Table 6, binary logistic regression analysis though not statistically significant for any individual predictor provides useful insight into potential predictors of nodal metastasis in uterine malignancies. Myometrial invasion >50% showed the strongest trend toward increased odds of lymph node involvement (OR 3.17, 95% CI 0.86–11.75), followed by Grade III histology (OR 2.86) and LVSI positivity (OR 1.77); cervical stromal involvement showed a trend in the opposite direction (OR 0.16).

Table 1: Demographic and clinical profile of study participants (n=70).

Variables	Category	Frequency	%
Age group (in years)	30-39	4	5.7
	40-49	6	8.6
	50-59	22	31.4
	60-69	21	30.0
	70-79	15	21.4
	80-89	2	2.9
	Pre-menopausal	8	11.4
	Post-menopausal	62	88.6
Chief complaint	Post menopausal bleeding	46	65.7
	Heavy menstrual bleeding	13	18.6
	Discharge per vaginum	4	5.7
	Pain abdomen	4	5.7
	Abnormal uterine bleeding	1	1.4
	Haematuria	1	1.4
	Post hysterectomy ESS–high grade	1	1.4
Risk factors	Diabetes mellitus	22	31.4
	Hypertension	35	50.0
	Obesity	10	14.3
	Hypothyroidism	2	2.9
	OCP use	2	2.9
	Tobacco	1	1.4
	No risk factor	26	37.1

Table 2: Histopathological and radiological characteristics (n=70).

Parameters	Category	Frequency	%
Endometrial biopsy findings	Endometrioid adenocarcinoma	53	75.7
	Pre-malignant/others	6	8.6
	Serous carcinoma	4	5.7
	Carcinosarcoma/MMMT	3	4.3
	Clear cell carcinoma	2	2.9
	Endometrial stromal sarcoma	2	2.9
Myometrial invasion	>50%	37	52.9
	<50%	25	35.7
	Absent	8	11.4
Lymph node involvement	Bilateral inguinal	2	2.9
	Bilateral pelvic	8	11.4
	Unilateral pelvic	4	5.7

Continued.

Parameters	Category	Frequency	%
	Paraaortic and pelvic	2	2.9
	Paraaortic and paratracheal	1	1.4
	Nil	53	75.7
Ascitic fluid cytology	Positive	3	4.3
	Negative	67	95.7
Lympho-vascular space invasion	Positive	12	17.1
	Negative	56	80.0
	Not assessed	2	2.9

Table 3: Staging and surgical details of study participants (n=70).

Tumour stagings	Pre-op imaging (N)	Pre-op imaging (%)	Post-operative (N)	Post-operative (%)
1A	29	41.4	23	32.9
1B	20	28.6	20	28.6
2	8	11.4	12	17.1
2A	2	2.9	2	2.9
3A	2	2.9	3	4.3
3B-2	2	2.9	2	2.9
3C-1	3	4.3	3	4.3
3C-2	1	1.4	2	2.9
4B	3	4.3	3	4.3

Table 4: Immunohistochemistry (IHC) and final histopathological reports (N=70).

Parameters	Category	Frequency	%
Immunohistochemistry	Tumor suppressor/mesenchymal/müllerian markers	18	25.7
	Hormonal/CEA markers	6	8.6
	Sarcoma/Carcinosarcoma markers	1	1.4
	MMMT markers	1	1.4
	IHC not done/not available	44	62.9
Histopathology report	Endometrioid carcinoma	53	75.7
	Sarcomas and MMMT	6	8.6
	Serous carcinoma	5	7.1
	Clear cell carcinoma	3	4.3
	Other aggressive subtypes	3	4.3
Histological grading	I	28	40.0
	II	28	40.0
	III	14	20.0

Table 5: Adjuvant therapy, follow-up profile and oncological outcome (survival and recurrence) (n=70).

Parameters	Category	Frequency	%
Type of adjuvant therapy given	Chemotherapy	12	17.1
	Radiotherapy	5	7.1
	Vaginal brachytherapy	5	7.1
	Chemotherapy and radiotherapy	1	1.4
	Not given	47	67.1
Follow-up status	Follow-up done	51	72.9
	Lost to follow-up	15	21.4
	Expired	4	5.7
Survival status	Survived	51	72.9
	Died	4	5.7
	No data	15	21.4
Recurrence rate	Yes	1	1.4
	No	69	98.6

Table 6: Binary logistic regression of factors associated with nodal positivity (n=70).

Predictors	OR (Exp(B))	95% CI for OR	P value
Myometrial invasion (>50%)	3.17	0.86 – 11.75	0.084
LVSI (Present)	1.77	0.33 – 9.39	0.503
Grade III	2.86	0.55 – 14.95	0.214
Non-endometrioid histology	1.11	0.23 – 5.29	0.893
Cervical stromal involvement	0.16	0.02 – 1.47	0.105

DISCUSSION

The study provides insight into the spectrum of uterine malignancies and their outcomes in a cohort of 70 patients. The majority of cases were endometrial carcinomas occurring in postmenopausal women, with a mean age in the sixth decade. Babu et al observed a comparable mean age at diagnosis of approximately 60 years, with 77% of patients presenting with vaginal bleeding.[12] Similarly, Prem et al. reported a mean age of 57.9 years, with 73.3% of patients postmenopausal.[13] In Western series, the median age at diagnosis is around 61 years, reinforcing that endometrial cancer predominantly affects women in the peri- and post-menopausal age group.[15]

Despite nulliparity being a well-recognized risk factor, most patients in Indian series including authors are multiparous, reflecting the higher baseline parity of the general population rather than a departure from established risk associations. Globally and in India, the incidence of endometrial carcinoma has been rising alongside increasing life expectancy and lifestyle change.[8,15] GLOBOCAN 2020 estimated 417,000 new endometrial cancer cases worldwide and 16,413 cases in India, indicating that although India's incidence remains lower than that of Western countries, it is on a clear upward trend.[9]

Endometrioid adenocarcinoma was the predominant subtype in the cohort (75.7%), which aligns with the 80–88% endometrioid histology reported in other Indian series.[12,13] High-grade tumours and rare uterine malignancies made up the remainder: we observed serous carcinoma in 7.1% and carcinosarcomas/uterine sarcomas in 8.6% of patients on final histopathology, a distribution comparable to that reported by Prem et al who noted carcinosarcoma in 8% and clear cell carcinoma in 6.3% of a similarly sized cohort.[13]

The presence of these aggressive subtypes is clinically significant, since they often present at advanced stages and portend worse outcomes than endometrioid histology. We similarly noted that all four cancer-related deaths in our study occurred in patients with high-grade histology or advanced-stage disease. These findings reinforce the dualistic model of endometrial carcinoma Type I tumours associated with a better prognosis, and Type II tumours (serous, clear cell, carcinosarcoma) associated with older age, advanced stage at presentation, and poorer outcomes.[4] Pradhan et al in a prospective immunohistochemical study from Eastern India, similarly emphasized that incorporating markers such as p53 and mismatch-repair proteins alongside conventional histology sharpens this risk stratification and better predicts which "low-grade" looking tumours behave aggressively an approach authors discuss further under study limitations.[14] Risk-factor analysis in our cohort underscores the role of metabolic factors: half of our patients were hypertensive and nearly one-third were diabetic, reflecting the well-known association between endometrial cancer and metabolic syndrome. This mirrors the Babu et al series, where hypertension was documented in 61.7% and diabetes in 43.3% of cases.[12] Obesity was documented in 14.3% of the patients, comparable to the 14.1% reported in a Northwest India series by Philips et al.[15]

The relatively modest recorded obesity rates in Indian cohorts, compared with Western series, may reflect underestimation of adiposity by body mass index alone or genuine ethnic differences in body composition and fat distribution; even so, the contribution of excess adiposity and related metabolic comorbidity to endometrial carcinogenesis remains evident. Interestingly, 37.1% of our patients had no identifiable risk factor, indicating that a substantial subset of uterine malignancies can occur in women without classic predisposing factors. This also reflects the historically low use of hormone replacement therapy (HRT) in India, in contrast to Western series where a proportion of patients have a history of tamoxifen or unopposed oestrogen therapy contributing to risk.

Overall, the findings concur that unopposed oestrogen exposure whether endogenous, as in obesity, or exogenous is a key driver of endometrial carcinogenesis, while also highlighting that uterine cancers can arise in its absence.[13] Stage at presentation and management in our study were favourable: approximately two-thirds of patients were diagnosed at an early stage (Stage I–II), likely owing to the early warning symptom of abnormal bleeding that prompts timely evaluation.

Philips et al reported postmenopausal bleeding in over 90% of cases in their series, and most large studies note that 70–80% of endometrial cancers are confined to the uterus at diagnosis.[15] Authors results echo this pattern, with Stage I being the single most common stage at diagnosis. Definitive surgery (hysterectomy with bilateral

salpingo-oophorectomy±staging) was accordingly the primary treatment for nearly all patients, with adjuvant therapy tailored to risk factors. Notably, 67.1% of the patients required no adjuvant treatment, reflecting the high proportion of low-risk, early-stage disease; those with deep myometrial invasion, advanced stage, or non-endometrioid histology received additional radiation and/or chemotherapy per standard protocols. This pattern is consistent with contemporary practice and other institutional experience in the Philips et al series, for example, 18.9% of patients received chemotherapy, generally those with stage III disease, while others were managed with surgery±radiotherapy depending on risk stratification.[15] Selective use of adjuvant vaginal brachytherapy and external-beam radiotherapy mirrors the risk-adapted approach validated in the PORTEC trials and confirms broad adherence to international guidelines in our setting.

Oncological outcomes in our study are encouraging, though with the caveat of a relatively short follow-up. The low recurrence rate is likely attributable to the predominance of early-stage, lower-grade tumours and adequate primary surgical management. Other studies with longer follow-up report higher recurrence rates, with 5-year survival around 80–85% overall the interim outcomes remain consistent with the expectation that early-stage disease carries an excellent prognosis. Prem et al noted that localized endometrial carcinoma can achieve 5-year survival up to 96%, which drops to approximately 67% once pelvic lymph nodes are involved.[13] Similarly, Philips et al reported a 5-year overall survival of 98.6% for Stage I patients in their Northwest India series.[15]

In cohort, all patients who succumbed to disease had either Stage III–IV cancer or an aggressive histologic subtype, underscoring that advanced stage and tumour biology rather than any single clinical variable drive outcomes, a trend consistently reported across the literature. The findings therefore reinforce that prognosis is excellent for early-stage endometrial carcinoma but significantly worse for advanced disease or high-grade histology, and they highlight the importance of early detection through prompt evaluation of abnormal uterine bleeding, together with appropriate risk-adapted adjuvant therapy, to improve long-term survival.

The study also demonstrated significantly better survival outcomes in patients who underwent laparoscopic surgery compared with those who underwent open procedures. All patients in the laparoscopic group survived during follow-up, whereas four deaths occurred in the open-surgery group (p=0.014). These findings are consistent with those of Walker et al who reported non-inferior oncological survival with lower perioperative morbidity for minimally invasive surgery in the landmark LAP2 trial. On regression analysis, Grade III histology and LVSI both traditionally associated with high-risk disease showed elevated, though non-significant, odds of nodal involvement in our model. LVSI in particular has repeatedly been identified as an independent adverse prognostic factor even in node-negative, early-stage endometrial cancer: Cusano et al found that LVSI retained prognostic significance for recurrence despite negative nodal status on staging, while Dai et al similarly reported that LVSI independently predicted both lymph node metastasis and reduced disease-free survival in endometrioid endometrial cancer.[16,17] These data support treating LVSI as a marker warranting close surveillance or consideration for adjuvant therapy even when nodal staging is reassuring a nuance our small sample was underpowered to demonstrate statistically.

Interestingly, cervical stromal involvement appeared inversely associated with nodal spread in our cohort; this counterintuitive finding most likely reflects our limited sample size or a chance co-occurrence with other localized tumour patterns rather than a true protective effect, and it should not be over-interpreted. While none of the predictors reached statistical significance in our model, likely owing to limited power, these trends align with established pathophysiological pathways of metastatic progression and warrant validation in larger, adequately powered studies ideally incorporating molecular classification alongside conventional histopathology.

Limitations

A key limitation of this study is the lack of molecular classification of endometrial carcinomas, which is now increasingly recommended in international guidelines to guide risk stratification and treatment planning. We did not perform immunohistochemical analysis for markers such as p53, mismatch-repair proteins, or POLE exonuclease-domain mutations, which form part of the molecular classification system proposed by the cancer genome atlas and endorsed in recent ESGO/ESTRO/ESP and Pan-Asian ESMO guidance.[2]

These markers have shown prognostic significance independent of conventional staging and histopathology, particularly in identifying ultralow-risk and high-risk tumours that would otherwise be mis-stratified by histology and grade alone a gap illustrated by Pradhan et al's finding that immunohistochemical profiling reclassified the expected prognosis in a meaningful subset of their cohort.[14] Because of resource constraints, adjuvant therapy decisions in the cohort were based solely on clinicopathological features and surgical staging, which may have led to under- or overtreatment in select molecular subgroups.

Other limitations include the relatively short and incomplete follow-up (with data unavailable for 21.4% of patients), the single-centre design, and the modest sample size, which limited the statistical power of the regression analysis. Future studies incorporating molecular profiling and longer, more complete follow-up will provide a more

refined, personalized approach to risk assessment and management of uterine malignancies, particularly in resource-limited settings like ours.

CONCLUSION

The study reaffirms that endometrioid endometrial carcinoma is the most prevalent uterine malignancy, commonly affecting older, postmenopausal women and typically presenting at an early stage with abnormal uterine bleeding, enabling favourable outcomes. A smaller but clinically significant subset comprised aggressive histologies serous and clear cell carcinomas, sarcomas, and carcinosarcomas which often required multimodal treatment.

The association with metabolic risk factors such as diabetes and hypertension mirrors global trends, although regional differences such as minimal HRT exposure remain notable. Given that none of the conventional pathological risk factors reliably predicted nodal involvement in our cohort, these findings support the use of comprehensive surgical staging through sentinel lymph node mapping or systematic lymphadenectomy across all risk groups, rather than reserving it for patients with high-risk features alone. Together, these results highlight the evolving epidemiology of uterine malignancies in India and emphasize the importance of early detection, risk-factor modification, and evidence-based treatment in improving long-term oncological outcomes.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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Cite this article as: Kumar D, Yadav G, Singh P, Gothwal M, Rao M. Spectrum of uterine malignancies and their oncological outcomes: a single centre experience. Int J Reprod Contracept Obstet Gynecol 2026;15:3449-56.