Section: Pathology

Case Series

HISTOPATHOLOGICAL SPECTRUM OF SYNCHRONOUS ENDOMETRIAL AND OVARIAN CANCERS: A CASE SERIES

Srishti Lamba[1], Anuja Sharma[2], Sumaiya Farukh Rashid Zehgir[3], Bawana Raina[4], Divyanshi Mahotra[5]

[1]PG Resident, ASCOMS Jammu, India.
[2]Professor, Department of Pathology, ASCOMS Jammu, India.
[3]PG Resident, ASCOMS Jammu, India.
[4]Professor, Department of Pathology, ASCOMS Jammu, India.
[5]PG Resident, ASCOMS Jammu, India.

Received : 18/04/2026
Received in revised form : 10/06/2026
Accepted : 25/06/2026

Keywords:
Synchronous malignancy; Endometrial carcinoma; Ovarian carcinoma; Endometrioid histology; Case series.

Corresponding Author:
Dr. Srishti Lamba,
Email: Srishtilamba10@yahoo.com

DOI: 10.47009/jamp.2026.8.4.41

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2026; 8 (4); 218-224

ABSTRACT

Synchronous endometrial and ovarian cancer (SEOC) is an infrequent presentation of gynecological malignancy in which primary tumors arise concurrently in the uterus and ovary. Differentiating synchronous primaries from metastatic disease poses a diagnostic challenge but is of major clinical importance, as treatment planning and prognostic outcomes vary considerably between the two entities. SEOC is often identified at an early stage and commonly exhibits endometrioid histology. This case series describes patients diagnosed with synchronous endometrial and ovarian cancer over a defined period. The patients presented with varying clinical features but shared the diagnosis of synchronous primary endometrial and ovarian malignancies with varying histopathological subtypes. All cases underwent comprehensive surgical management with appropriate staging procedures. Histopathological assessment favored the diagnosis of synchronous primary malignancies rather than metastatic spread. **Conclusion:** Synchronous endometrial and ovarian cancer represents a distinct clinicopathological entity with a generally favorable prognosis when diagnosed early. Careful integration of clinical findings, histopathological criteria, and molecular studies, when available, is essential for accurate diagnosis. Reporting institutional case series enhances understanding of disease behavior and aids in refining diagnostic and therapeutic approaches.

INTRODUCTION

Synchronous malignancies of the female genital tract are rare clinical entities, defined by the simultaneous occurrence of two or more primary tumors at different anatomical sites.[12] Among these, synchronous endometrial and ovarian cancer (SEOC) represents the most frequently encountered combination.[6] SEOC accounts for a small proportion of gynaecological cancers, yet it holds significant clinical importance due to diagnostic, therapeutic, and prognostic implications.[5]

SEOC is characterized by the concurrent presence of primary endometrial carcinoma and primary ovarian carcinoma diagnosed at the same time. The reported incidence varies, with synchronous tumors identified in a minority of patients diagnosed with either endometrial or ovarian cancer.[6] Tumors at both sites may be of the same histologic type or different.[6]

Patients. Other histologic types such as serous, mucinous, clear cell, or mixed carcinomas have also been reported. Patients with SEOC tend to be younger and may present with symptoms such as abnormal uterine bleeding, pelvic pain, or an adnexal mass, although some cases are detected incidentally during evaluation for one primary tumor.[6]

A major diagnostic challenge in SEOC lies in distinguishing true synchronous primary tumors from metastatic disease involving the endometrium and ovary.[12] This distinction is crucial, as metastatic disease is associated with advanced staging, need for aggressive adjuvant therapy, and poorer prognosis, whereas synchronous primary tumors generally demonstrate favourable outcomes.[6] Traditional diagnostic approaches rely on clinicopathological criteria, including tumor grade, depth of myometrial invasion, laterality and extent of ovarian involvement, and the presence or absence of lymphovascular invasion.[5]

Despite established histopathological criteria, overlap between synchronous and metastatic tumors may occur, particularly when both lesions share similar histological features. Advances in immunohistochemistry and molecular profiling have provided additional tools to support accurate classification in challenging cases. Alterations in

ISSN (O): 2687-5365; ISSN (P): 2753-6556

genes such as PTEN, PIK3CA, CTNNB1, and others involved in endometrial and ovarian carcinogenesis have been increasingly studied to aid in understanding tumor origin and biological behaviour.[6]

Given the rarity of SEOC and the limited data available from large cohorts, case series play an important role in expanding current knowledge regarding its clinical presentation, pathological characteristics, molecular features, and outcomes.The present case series aims to describe the clinicopathological profile of patients diagnosed with synchronous endometrial and ovarian cancer and to highlight the importance of accurate diagnosis for appropriate management and prognostication.[4]

Case Series

A. Chief Complaints (CC)

Case 1: Abnormal uterine bleeding associated with intermittent lower abdominal discomfort.

Case 2: Abdominal distension and vague pelvic discomfort.

Case 3: Postmenopausal bleeding with progressive abdominal fullness.

B. History of Present Illness

Case 1: A 39-year-old woman presented with abnormal uterine bleeding of several months' duration, associated with mild lower abdominal pain. There was no history of weight loss or bowel or bladder symptoms.

Case 2: A 73-year-old postmenopausal woman presented with gradually increasing abdominal distension and pelvic discomfort. There was no history of vaginal bleeding.

Case 3: A 58-year-old postmenopausal woman presented with postmenopausal bleeding and abdominal distension. She had received neoadjuvant chemotherapy prior to surgery for a diagnosed pelvic malignancy.

C. Relevant Past History

Case 1: No significant medical comorbidities. No prior gynecological malignancy.

Case 2: Known case of hypertension, on regular medication. No prior history of malignancy.

Case 3: No significant medical comorbidities. History significant for neoadjuvant chemotherapy for suspected advanced gynecological malignancy.

D. Radiological Findings

Case 1: Imaging revealed endometrial thickening with bilateral adnexal masses, suspicious for ovarian neoplasm.

Case 2: Radiological evaluation demonstrated bilateral adnexal masses with features suggestive of ovarian malignancy along with endometrial thickening.

Case 3: Imaging showed bilateral and endometrial involvement was also suspected. Post-chemotherapy imaging showed partial radiological response.

E. Surgical Procedure Performed

Case 1: Total abdominal hysterectomy with bilateral salpingo-oophorectomy, pelvic lymph node dissection, and omentectomy.

Case 2: Total abdominal hysterectomy with bilateral salpingo-oophorectomy, bilateral pelvic and para-aortic lymph node dissection, and omentectomy.

Case 3: Radical hysterectomy with bilateral salpingo-oophorectomy, bilateral pelvic lymph node dissection, para-aortic sampling, and omentectomy following neoadjuvant chemotherapy.

F. Gross Pathological Findings

Case 1: Uterus showed an ill-defined endometrial growth. Both ovaries were enlarged, with the left ovary showing surface nodularity.

Case 2: Uterus revealed an endometrial lesion. Both ovaries were enlarged with solid and papillary areas on cut surface. Omentum appeared grossly unremarkable.

Case 3: Uterus showed a small endometrial lesion. Both ovaries were enlarged and showed a solid, whitish cut surface.

G. Microscopic (Histopathological) Findings

G1. Endometrium

Case 1: Endometrioid carcinoma, FIGO grade 2, with less than 50% myometrial invasion. Lower uterine segment involvement was present. No lymphovascular invasion was identified. Serosa free of tumor

Case 2: High-grade serous carcinoma with less than 50% myometrial invasion. Lower uterine segment was involved. Cervix and serosa were free of tumor.

Case 3: High-grade serous carcinoma confined to the endometrium, with no myometrial invasion. Lower uterine segment involvement was noted. Chemotherapy response score (CRS) was 3.

G2. Ovary / Fallopian Tube

Case 1: Bilateral ovarian involvement by low-grade serous carcinoma. Ovarian capsules were intact.

Case 2: Bilateral ovaries showed endometrioid carcinoma. Ovarian capsules were intact, with no surface involvement. Tumor was present within the lumina of both fallopian tubes..

Case 3: Bilateral ovarian involvement by high-grade serous carcinoma. Capsule was intact. Left fallopian tube was involved by tumor. CRS for ovarian tumor was 1.

G3. Lymph Nodes / Omentum / Other Tissues

Case 1: All sampled pelvic lymph nodes were negative for metastasis. Omentum showed no tumor deposits.

Case 2: All pelvic and para-aortic lymph nodes were negative for metastasis. Omentum was free of tumor.

Case 3: Pelvic lymph nodes showed metastatic carcinoma (right: 2/7 nodes; left: 1/8 nodes). Para-aortic nodes were not identified. Omentum and appendices epiploicae were free of tumor.

H. .Peritoneal / Ascitic Fluid Cytology

- Case 1: Negative for malignant cells.
- Case 2: Negative for malignant cells.
- Case 3: Negative for malignant cells.

I. Final Histopathological Diagnosis

Case 1: Synchronous endometrioid carcinoma of the endometrium and low-grade serous carcinoma of bilateral ovaries.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Case 2: Synchronous endometrioid carcinoma involving the endometrium and bilateral ovaries.
Case 3: Synchronous high-grade serous carcinoma of the endometrium and bilateral ovaries following neoadjuvant chemotherapy.

J. Pathological Staging (FIGO)

Case 1:

- Endometrium: FIGO stage IA
- Ovary: FIGO stage IB

Case 2:

- Endometrium: FIGO stage IA
- Ovary: FIGO stage IB

Case 3:

- Endometrium: FIGO stage IA (ypT1a)
- Ovary: FIGO stage IIIC (ypT1c pN1)

K. Ancillary Studies / IHC / Molecular Testing

Case 1: Immunohistochemistry and molecular studies were advised but not performed.
Case 2: Immunohistochemistry was advised; however, no ancillary studies were available for review.
Case 3: Immunohistochemistry was advised.

L. Follow-up and Outcome

Case 1: Patient received adjuvant therapy and is currently under follow-up.
Case 2: Patient was advised adjuvant chemotherapy and is on regular follow-up.
Case 3: Patient is receiving adjuvant chemotherapy and remains under close clinical follow-up.

RESULTS

Table 1: Case summaries.

Parameter	Case 1	Case 2	Case 3
Age (years)	39	73	58
Menopausal status	Premenopausal	Postmenopausal	Postmenopausal
Chief complaint	Abnormal uterine bleeding	Abdominal distension, pelvic discomfort	Postmenopausal bleeding, abdominal distension
Neoadjuvant chemotherapy	Not given	Not given	Given
Type of surgery	TAH + BSO + PLND + omentectomy	TAH + BSO + PLND + PALND + omentectomy	Radical hysterectomy + BSO + PLND + PALN sampling + omentectomy
Endometrial tumor histology	Endometrioid carcinoma	Endometrioid carcinoma	High-grade serous carcinoma
Endometrial tumor grade	FIGO grade 2	High grade	High grade
Myometrial invasion	<50%	<50%	Absent
Lower uterine segment involvement	Present	Present	Present
LVSI (endometrium)	Absent	Absent	Absent
Ovarian tumor histology	Low-grade serous carcinoma	Endometrioid carcinoma	High-grade serous carcinoma
Ovarian laterality	Bilateral	Bilateral	Bilateral
Capsule status	Intact	Intact	Intact
Ovarian surface involvement	Absent	Absent	Absent
Fallopian tube involvement	Absent	Present (bilateral luminal)	Present (left tube)
Lymph node status	Negative	Negative	Positive (3/15 pelvic nodes)
Peritoneal/ascitic fluid cytology	Negative	Negative	Negative
Omental involvement	Absent	Absent	Absent
Chemotherapy Response Score (CRS)	Not applicable	Not applicable	CRS 3 (endometrium), CRS 1 (ovary)
FIGO stage – Endometrium	IA	IA	IA (ypT1a)
FIGO stage – Ovary	IB	IB	IIIC (ypT1c pN1)
Final diagnosis	Synchronous endometrioid EC and low-grade serous OC	Synchronous endometrioid EC and endometrioid OC	Synchronous high-grade serous EC and OC post-NACT
Follow-up status	On follow-up	On follow-up	On adjuvant chemotherapy and follow-up

ISSN (O): 2687-5365; ISSN (P): 2753-6556

CASE 1

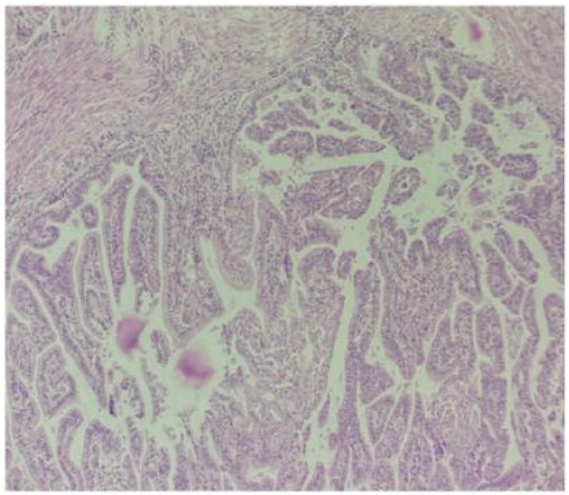

Figure 1a: Complex papillary and glandular architecture lined by atypical epithelial cells with fibrovascular cores, consistent with serous carcinoma (H&E stain, ×100)

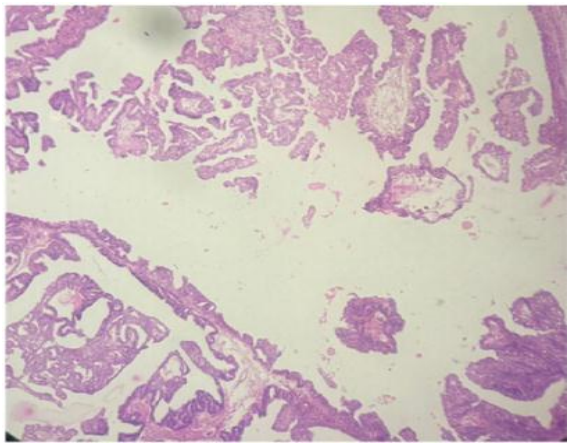

Figure 1b: Multiple papillary tumor fragments with hierarchical branching architecture, consistent with ovarian serous carcinoma (H&E stain, ×40)

CASE 2

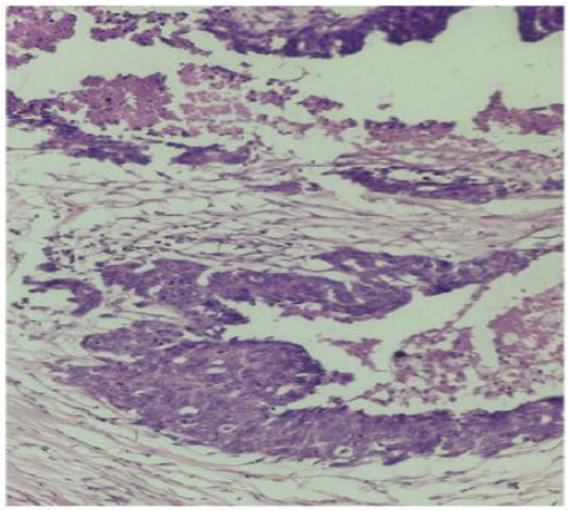

Figure 2a: Infiltrating malignant epithelial cells arranged in papillary architecture with marked nuclear atypia. (H&E stain, ×100)

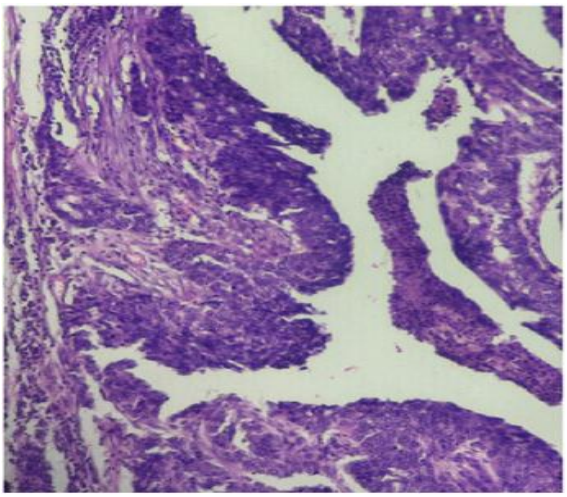

Figure 2b: Complex branching papillae lined by pleomorphic atypical epithelial cells with hyperchromatic nuclei (H&E stain, ×200)

CASE 3

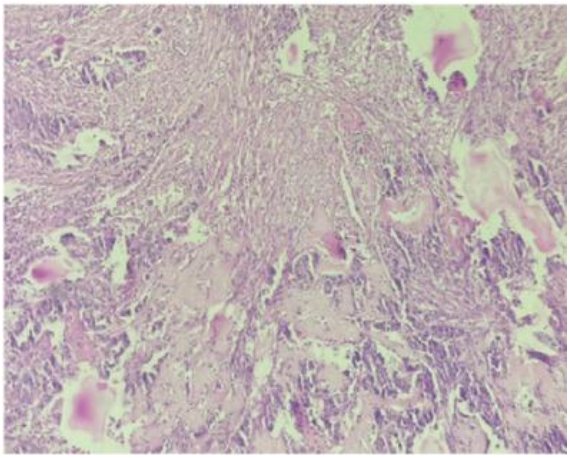

Figure 3a: Infiltrative malignant glands and papillary structures embedded within desmoplastic stroma, consistent with high-grade serous carcinoma (H&E stain, ×100)

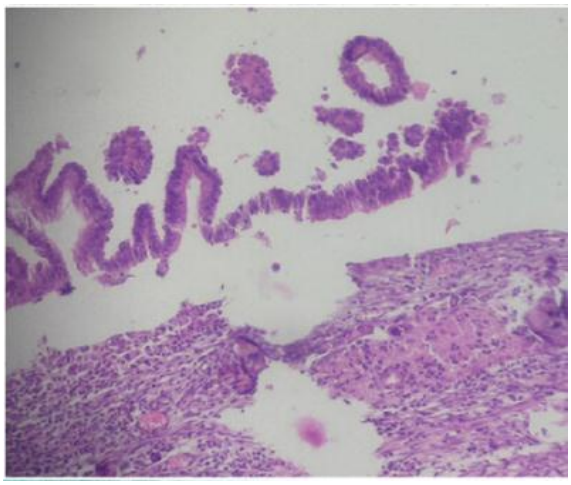

Figure 3b: Papillary clusters lined by atypical stratified epithelial cells with marked nuclear pleomorphism, consistent with serous carcinoma (H&E stain, ×200)

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Figure 3c: Gross specimen showing enlarged bilateral ovaries with solid-cystic cut surface and associated hysterectomy specimen from synchronous endometrial and ovarian carcinoma

DISCUSSION

Synchronous endometrial and ovarian carcinoma (SEOC) represents the most common form of synchronous gynaecologic malignancy, accounting for nearly 50%–70% of all synchronous tumors of the female genital tract.[1,2] Although uncommon overall, SEOC has major clinical significance because its prognosis, therapeutic approach, and long-term outcomes differ considerably from metastatic endometrial or ovarian carcinoma.[3,4] The reported incidence ranges from 3%–10% among women with endometrial carcinoma and approximately 10% among women with ovarian carcinoma.[5,6] Most patients are diagnosed at an early stage and demonstrate favourable clinicopathological characteristics.

The majority of women diagnosed with SEOC are younger than those with isolated endometrial or ovarian malignancies, with most cases occurring between 41 and 54 years of age.[2,7] Many patients are premenopausal, nulliparous, or overweight.[7,8]

Case 1 involved a comparatively younger patient who presented with features similar to those reported in previous SEOC series, supporting the observation that synchronous tumors may occur in younger women than isolated gynecological malignancies..[2,8] Previous studies have consistently shown that abnormal uterine bleeding is the most common presenting symptom, followed by pelvic pain, abdominal discomfort, or adnexal mass.[7,9] Similar findings were observed in our patient, Abnormal uterine bleeding occurred in Cases 1 and 3, not Case 2. Histologically, the endometrioid subtype is the most frequently encountered pattern in SEOC and is reported in nearly 50%–70% of cases.[1,7] Tumors are usually low-grade and diagnosed at an early FIGO stage, contributing to the relatively favourable prognosis associated with these neoplasms.[4,10] Case 2 demonstrated endometrioid histology in both the endometrium and ovary, comparable to previously published reports.[1,9]

Distinguishing true synchronous primary tumors from metastatic disease remains one of the greatest diagnostic challenges in gynaecologic oncology. Historically, the criteria proposed by Ulbright and Roth and later refined by Scully have been widely utilized for differentiating synchronous primaries from metastatic carcinoma.[12,13] These criteria incorporate factors such as histologic similarity, tumor grade, depth of myometrial invasion, laterality of ovarian involvement, vascular space invasion, and presence of precursor lesions.[12,13] Features favouring SEOC include low-grade tumors, superficial myometrial invasion (<50%), absence of lymphovascular invasion, and absence of distant metastatic disease.[11,13] Case 1 demonstrated superficial myometrial invasion, absence of lymphovascular invasion, and the overall clinicopathological findings supported the diagnosis of synchronous primary malignancies rather than metastatic disease. Radiological imaging also plays an important role in evaluation and staging. Ultrasonography, computed tomography, and magnetic resonance imaging commonly demonstrate unilateral complex solid-cystic ovarian masses associated with endometrial abnormalities.[14] Previous studies have reported that metastatic ovarian involvement from endometrial carcinoma is more often bilateral and solid, whereas synchronous tumors tend to be unilateral and confined.[14] Similar radiologic findings were noted in our cases, where imaging revealed bilateral adnexal masses associated with endometrial pathology, raising suspicion for synchronous malignancy.

Recent molecular and immunohistochemical advances have significantly improved understanding of SEOC biology. Traditional histopathological assessment may occasionally be insufficient, especially in tumors with concordant endometrioid histology.[15] Immunohistochemical markers such as vimentin, estrogen receptor (ER), progesterone receptor (PR), PAX8, β-catenin, and mismatch repair (MMR) proteins have been investigated to improve diagnostic accuracy.[15,16] Vimentin expression has been reported to show high sensitivity and specificity in differentiating primary ovarian from metastatic endometrial tumors.[16] Similarly, abnormalities in β-catenin expression and CTNNB1 mutations have been associated more strongly with synchronous primary tumors than metastatic disease.[17]

Molecular profiling studies have further demonstrated that many tumors previously classified as independent primaries may actually represent clonally related metastatic disease.[18,19] Next-generation sequencing studies evaluating mutations in PTEN, CTNNB1, PIK3CA, KRAS, ARID1A, TP53, and mismatch repair genes have provided important insights into tumor origin and pathogenesis.[18-20] Several studies have identified frequent PTEN and CTNNB1 mutations in SEOC, suggesting a distinct molecular signature compared to metastatic disease.[20,21] One reviewed study identified mutations involving CTNNB1, PIK3CA,

ISSN (O): 2687-5365; ISSN (P): 2753-6556

and PTEN in ovarian tissue, supporting activation of the PI3K/AKT and WNT signaling pathways in SEOC development.[9] Although molecular testing was not available in our patients, the clinicopathological findings correlated strongly with those reported in molecularly confirmed synchronous primary tumors.

The pathogenesis of SEOC remains incompletely understood. Several theories have been proposed, including the "secondary Müllerian system" hypothesis, which suggests that tissues derived from Müllerian epithelium respond similarly to hormonal and carcinogenic influences.[22] Shared estrogen receptor expression, endometriosis-associated malignant transformation, and common molecular pathways may contribute to synchronous tumorigenesis.[22,23] Case 2 demonstrated endometrioid histology in both the endometrium and ovary, supporting the concept of a common hormonal or molecular predisposition. Management of SEOC primarily involves comprehensive surgical staging. Standard treatment generally includes total abdominal hysterectomy with bilateral salpingo-oophorectomy, pelvic and para-aortic lymph node dissection, omentectomy, peritoneal biopsies, and cytological washings.[1,24] Complete surgical staging remains essential not only for diagnosis but also for prognostic stratification and treatment planning.[24] Adjuvant therapy is individualized according to stage, grade, histology, and risk factors. Patients with stage IA low-grade tumors may not require chemotherapy or radiotherapy, whereas advanced-stage disease generally warrants systemic treatment.[11,24] Similar to previously published cases, all three patients underwent complete surgical staging, and further treatment decisions were guided by the final histopathological findings.

Compared with metastatic endometrial or ovarian carcinoma, SEOC is associated with significantly better outcomes. Reported five-year survival rates range from 65%–90%, particularly among patients with early-stage endometrioid tumors.[4,8,25] Favorable prognostic factors include younger age, early-stage disease, low-grade histology, absence of lymphovascular invasion, and complete surgical staging.[8,25] Soliman et al. demonstrated that patients with synchronous endometrioid tumors often achieve prolonged disease-free survival approaching 10 years.[5] Similarly, studies by Song et al. showed improved prognosis in younger women with low-grade synchronous tumors.[25] Case 1 had favourable postoperative recovery and remains disease-free on follow-up, comparable to outcomes reported in the literature.

Despite advances in molecular pathology and genomic profiling, several challenges remain in the diagnosis and classification of SEOC. Histopathological overlap with metastatic disease may still lead to diagnostic uncertainty, particularly in tumors with concordant histology.[18] Additionally, access to advanced molecular testing may be limited in resource-constrained settings, as was encountered in our cases. Therefore, integration of clinicopathological features with immunohistochemical and molecular findings remains essential for accurate diagnosis and individualized patient management.

In conclusion, synchronous endometrial and ovarian carcinoma represents a distinct clinicopathological entity with generally favorable prognosis when diagnosed at an early stage. Careful evaluation using established histopathological criteria, supported by imaging, immunohistochemistry, and molecular profiling when available, is essential to differentiate synchronous primary tumors from metastatic disease. Continued research into the molecular mechanisms and genetic signatures of SEOC may further improve diagnostic precision, prognostic stratification, and therapeutic decision-making in the future.

CONCLUSION

Synchronous endometrial and ovarian carcinomas constitute a distinct subset of gynecological malignancies with important diagnostic and prognostic implications. Accurate differentiation between synchronous primary tumors and metastatic disease is essential, as it directly influences staging, therapeutic decision-making, and patient outcomes. This case series highlights the morphological diversity of synchronous tumors, encompassing both endometrioid and serous histological subtypes, variable grades, and differing patterns of spread.

The present cases emphasize that synchronous tumors may occur across a wide age range and can present with both low-grade and high-grade histologies. Careful evaluation of tumor morphology, depth of myometrial invasion, ovarian laterality, surface involvement, lymph node status, and response to chemotherapy is critical in establishing the diagnosis. In selected cases, discordant histological features or differential chemotherapy response between tumor sites may further support independent tumor origin.

Thorough gross examination, extensive tissue sampling, and comprehensive histopathological reporting remain the cornerstone of diagnosis. While immunohistochemistry and molecular studies can provide valuable adjunctive information in diagnostically challenging cases, meticulous histopathological assessment continues to play a pivotal role, particularly in resource-limited settings.

In conclusion, synchronous endometrial and ovarian carcinomas represent a heterogeneous group of tumors with generally favorable outcomes when correctly identified and appropriately staged. Awareness of this entity and adherence to a systematic, pathology-driven approach are essential to avoid misclassification and to ensure optimal patient management.

Conflict of Interest: The authors declare no conflict of interest.

Funding: No funding was received for this study.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

REFERENCES

1. Patru CL, Marinas MC, Drocas I, et al. Synchronous primary ovarian and endometrial carcinomas in a young patient: Case report and literature review. Rev Chim. 2020;70(12):4360-4365.
2. Liu Y, Li J, Jin H, Lu Y, Lu X. Clinicopathological characteristics of patients with synchronous primary endometrial and ovarian cancers: A review of 43 cases. Oncol Lett. 2013;5(1):267-270.
3. Wang T, Zhang X, Lu Z, Wang J, Hua K. Comparison and analysis of the clinicopathological features of SCEO and ECOM. J Ovarian Res. 2019;12(1):10.
4. Matsuo K, Machida H, Blake EA, et al. Trends and outcomes of women with synchronous endometrial and ovarian cancer. Oncotarget. 2018;9:28757-28771.
5. Soliman PT, Slomovitz BM, Broaddus RR, et al. Synchronous primary cancers of the endometrium and ovary: A single institution review of 84 cases. Gynecol Oncol. 2004;94(2):456-462.
6. Zaino R, Whitney C, Brady MF, et al. Simultaneously detected endometrial and ovarian carcinomas—a prospective clinicopathologic study of 74 cases. Gynecol Oncol. 2001;83:355-362.
7. Makris GM, Manousopoulou G, Battista MJ, et al. Synchronous endometrial and ovarian carcinoma: A case series.Case Rep Oncol. 2017;10(2):732-736.
8. Song T, Seong SJ, Bae DS, et al. Prognostic factors in women with synchronous endometrial and ovarian cancers.Int J Gynecol Cancer. 2014;24(3):520-527.
9. Žilovič D, Čiurlienė R, Šidlovska E, et al. Synchronous endometrial and ovarian cancer: A case report. World J Clin Cases. 2023;11(18):4341-4349.
10. Rijal Y, Shrestha S, Bogati L, et al. Synchronous primary endometrium and ovarian carcinoma: A case report. Clin Case Rep. 2022;10:e06432.
11. Dębska-Szmich S, Czernek U, Krakowska M, et al. Synchronous primary ovarian and endometrial cancers: A series of cases and a review of literature. Prz Menopauzalny. 2014;13(1):64-69.
12. Ulbright TM, Roth LM. Metastatic and independent cancers of the endometrium and ovary: A clinicopathologic study of 34 cases. Hum Pathol. 1985;16:28-34.
13. Scully RE, Young RH. Metastatic tumors of the ovary. In: Blaustein's Pathology of the Female Genital Tract. Springer; 2002:987-990.
14. Moro F, Leombroni M, Pasciuto T, et al. Synchronous primary cancers of endometrium and ovary vs endometrial cancer with ovarian metastasis: An observational study. Ultrasound Obstet Gynecol. 2019;53(6):827-835.
15. Singh N. Synchronous tumours of the female genital tract. Histopathology. 2010;56:277-285.
16. Desouki MM, Kallas SJ, Khabele D, et al. Differential vimentin expression in ovarian and uterine corpus endometrioid adenocarcinomas: Diagnostic utility in distinguishing double primaries from metastatic tumors. Int J Gynecol Pathol. 2014;33:274-281.
17. Irving JA, Catasús L, Gallardo A, et al. Alterations in the beta-catenin (CTNNB1) pathway are associated with independent primary tumors and favorable prognosis. Hum Pathol. 2005;36:605-619.
18. Anglesio MS, Wang YK, Maassen M, et al. Synchronous endometrial and ovarian carcinomas: Evidence of clonality. J Natl Cancer Inst. 2016;108:djv428.
19. Schultheis AM, Ng CK, De Filippo MR, et al. Massively parallel sequencing-based clonality analysis of synchronous endometrioid endometrial and ovarian carcinomas. J Natl Cancer Inst. 2016;108:djv427.
20. Reijnen C, Küsters-Vandevelde HVN, Ligtenberg MJL, et al. Molecular profiling identifies synchronous endometrial and ovarian cancers as metastatic endometrial cancer with favorable clinical outcome. Int J Cancer. 2020;147:478-489.
21. Chao A, Wu RC, Jung SM, et al. Implication of genomic characterization in synchronous endometrial and ovarian cancers of endometrioid histology. Gynecol Oncol. 2016;143:60-67.
22. Khan M, Amin SV, Srinivas SB, et al. Hydrosalpinx as a rare presentation of synchronous ovarian and endometrial carcinoma—a case report. J Clin Diagn Res. 2016;10(7):QD01-QD03.
23. Matias-Guiu X, Stewart CJR. Endometriosis-associated ovarian neoplasia. Pathology. 2018;50:190-204.
24. Shin W, Park SY, Kang S, et al. How to manage synchronous endometrial and ovarian cancer patients? BMC Cancer. 2021;21:489.
25. Khalid N, Ullah F, Zafar H, et al. Synchronous primary endometrial and ovarian cancers: Trends and outcomes of the rare disease at a South Asian tertiary care cancer center. Cureus. 2020;12(7):e9163.

ISSN (O): 2687-5365; ISSN (P): 2753-6556