

# Breast and Axillary Metastases from High-grade Serous Ovarian Carcinoma: A Rare Case Report Highlighting Diagnostic Challenges and Pitfalls

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## ABSTRACT

High-grade Serous Ovarian Carcinoma (HGSOC) is an aggressive malignancy that commonly metastasizes to peritoneum, omentum, pelvic and para-aortic lymph nodes, serosal surfaces of bowel and mesentery. Metastases to the breast and axillary lymph nodes is extremely rare and can closely mimic primary breast malignancy, making diagnosis extremely difficult, especially if it occurs after a prolonged disease-free interval. A 35-year-old nulligravid woman presented with a painless right breast lump of two months' duration along with bilateral axillary lymphadenopathy. She had already been diagnosed with HGSOC six years prior, for which she received neoadjuvant chemotherapy, optimum cytoreductive surgery, and several lines of chemotherapy. On radiological assessment, a suspicious right breast lesion with bilateral axillary lymph node enlargement and pleural effusion was discovered during the current presentation. Core biopsies from the right breast lesion and axillary lymph nodes showed a high-grade malignant epithelial tumour arranged in papillary pattern. Metastatic HGSOC was confirmed by immunohistochemistry, which showed positive expression for PAX8, WT1, CK7 with aberrant p53 and negative for breast-specific immunohistochemistry markers ER, PR, HER2, and GATA3. Given the chemoresistant disease and symptomatic breast lesion, the patient was treated with palliative local radiation, after which she experienced symptomatic relief and stable disease at short-term follow-up. The case highlights the importance of careful histopathological examination supported by an appropriate immunohistochemistry panel in making the correct diagnoses at unusual metastatic sites with long latency periods which can cause diagnostic confusion with primary breast carcinoma and prompted this case presentation.

**Keywords:** Axillary lymph node, Breast lump, Computed Tomography, Chemotherapy, Recurrence

## CASE REPORT

A 35-year-old nulligravid female presented with painless right breast lump of two-month duration associated with progressively enlarging bilateral axillary swellings. No fever, weight loss, skin changes, or nipple discharge were present.

She had a history of HGSOC, diagnosed six-years earlier, when she initially presented with insidious-onset right-sided lower quadrant pain, constipation, and bloating. She denied alcohol abuse, smoking or use of illicit drugs at the time of her initial diagnosis, and she had no notable family or medical history. Radiological assessment was reviewed from available electronic medical records. The original imaging files could not be retrieved due to archival limitations. The Contrast-Enhanced Computer Tomography (CECT) findings were suggestive of a large, multiloculated, cystic and solid right adnexal mass of size 15x12 cm with heterogeneous enhancement in the solid components. The surgical biopsy procured during exploratory laparotomy confirmed the diagnosis of HGSOC. She subsequently underwent four cycles of neoadjuvant chemotherapy with carboplatin and paclitaxel.

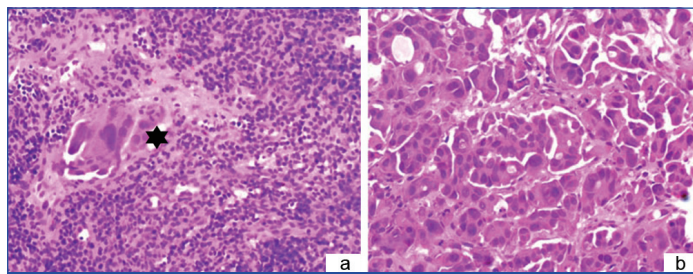
After a disease-free period of three months, surveillance CECT (as per the available written reports) revealed metastatic recurrence to multiple sub-centimetric mesenteric and para-aortic lymph nodes, which showed signs of enlargement and thus likely metastasis. The bowel loops were largely unremarkable, with no signs of pathological change. Additionally, a multiloculated solid-cystic left adnexal mass of size 5.5x2.6 cm, medial to the left iliac vessels, demonstrating an enhancing wall with internal solid components, raising suspicion for malignancy. A small enhancing nodule of size 1.0x0.9 cm was identified adjacent to the uterine fundus, extending towards the parietal peritoneum. Moderate ascites was

also noted which was treated with symptomatic measures such as salt restriction and diuretics along with close clinical monitoring. She subsequently underwent an optimal interval cytoreductive surgery in the form of total abdominal hysterectomy with bilateral salpingo-oophorectomy and omentectomy. Targeted therapies and several lines of chemotherapy were administered after surgery. This patient did not have BRCA1/2 (breast cancer gene 1/2) or Homologous Recombination Deficiency (HRD) testing due to financial constraints. At the current presentation her physical assessment revealed a 1.5 cm painless tumour lesion in the upper inner quadrant of the right breast' along with several hard, irregular lymph nodes in the bilateral axilla. The tumour marker assay revealed high levels of serum carbohydrate antigen-125 (CA-125: 862.9 U/mL; normal: <35 U/mL). All other biochemical tests, however, yielded normal results.

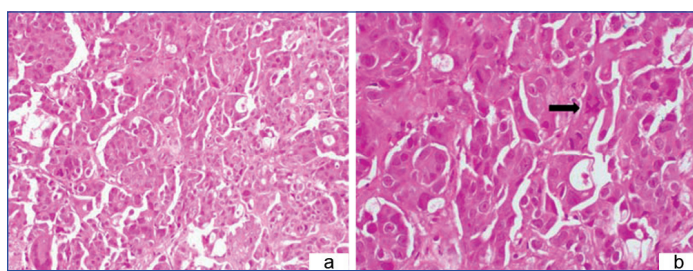
A whole-body CECT scan demonstrated bilateral pleural effusion, multiple enlarged axillary lymph nodes, and a nonspecific nodular lesion of the right breast, suspicious for metastasis. At the time of manuscript preparation, the original Digital Imaging and Communications in Medicine (DICOM) images were not retrievable from the institutional Picture Archiving and Communication System (PACS) due to routine archival and data-retention policies, and the investigators had to rely on details available from the electronic medical records.

The patient underwent therapeutic pleural tapping for symptomatic relief and the pleural fluid sample sent for cytopathologic evaluation revealed clusters and papillaroid fragments of atypical cells. Ultrasound-guided biopsies of the right breast lesion and the right axillary lymph nodes were performed, which showed infiltration by a tumour disposed in papillae and nests, as well as singly scattered [Table/Fig-1 a,b]. The biopsy procured during exploratory laparotomy

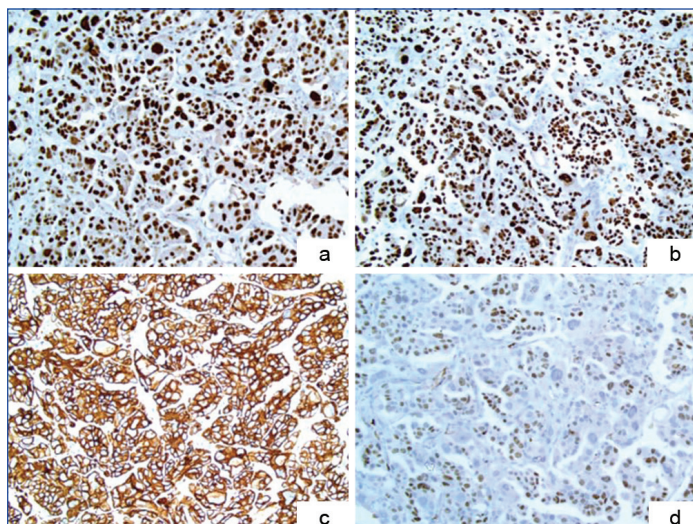
showed morphologically concordant tumour as shown in the [Table/Fig-2a,b]. On applying immunohistochemistry in the right breast lesion biopsy, the tumour cells showed positive expression for PAX8, p53, CK7, and WT1 [Table/Fig-3a-d], while negative expression for ER, PR, HER2 and GATA3. Thus, a diagnosis of metastatic HGSOc was established.



**[Table/Fig-1]:** a) Ovarian metastases to right axillary lymph node tissue (haematoxylin & eosin, 400x). Metastatic tumour deposit has been shown by a black asterisk; b) Ovarian metastases to right breast tissue (haematoxylin & eosin, 400x). The tumour cells show marked pleomorphism, round to oval nuclei, prominent nucleoli, and a moderate amount of cytoplasm.



**[Table/Fig-2]:** a) Section from the primary ovarian tumour biopsy procured during exploratory laparotomy, showing a tumour disposed as papillary architecture and display high-grade cytological atypia, morphologically overlapping with the metastatic deposits in the breast and axillary lymph nodes (H&E, x200). Ovarian stromal tissue is not represented in the available section; b) The tumour cells show marked pleomorphism, round to oval nuclei, prominent nucleoli, and a moderate amount of cytoplasm. The black arrow shows atypical mitosis. (H&E, x400).



**[Table/Fig-3]:** a) Immunohistochemistry for PAX8 shows strong and diffuse nuclear positivity in the tumour cells (400x); b) Immunohistochemistry for p53 shows strong and diffuse nuclear positivity in the tumour cells (400x); c) Immunohistochemistry for CK7 shows strong and diffuse cytoplasmic positivity in the tumour cells (400x); d) Immunohistochemistry for WT1 shows strong and diffuse nuclear positivity in the tumour cells (400x).

Given the patient's chemoresistant disease and symptomatic breast lesion, palliative local radiation was chosen. The patient tolerated the treatment well and remains alive to date (>3 months) with a stable disease. The current case report emphasises diagnostic challenges at metastatic site especially when there is a late presentation.

## DISCUSSION

The HGSOc accounts for the most common and also the most lethal subtype of Epithelial Ovarian Carcinomas (EOC), as most of the cases are diagnosed at a late stage, by which time when

achieving a cure is very difficult [1]. It was previously speculated that the risk reduction is noted in women undergoing bilateral salpingectomy mostly indicates a diminished incidence of HGSOc. Studies support the fact that the majority HGSOcs arise from the distal fallopian tube [2,3], but they can also arise from the ovary. Genomic instability is a hallmark feature of HGSOc, with the earliest and defining feature being the nearly universal TP53 mutation. Additional alterations frequently involve homologous recombination repair deficiency, including BRCA1/2 dysfunction, or amplification of cell-cycle regulatory pathways such as CCNE1; these alterations are largely mutually exclusive. The emergence of late and unusual metastatic patterns, chemoresistance and aggressive clinical behaviour are thought to be caused by genomic instability, which also causes significant intratumoural heterogeneity. The prolonged disease course and unusual metastases in the present case may be explained by clonal evolution and selection of therapy resistant tumour subpopulations over time.

The common sites where HGSOc metastasizes are peritoneum, omentum, pelvic and para-aortic lymph nodes, serosal surfaces of bowel and mesentery [4]. The uncommon metastatic sites in HGSOc are breast, axillary lymph nodes, bone, lung, brain and skin [5]. Metastases of serous ovarian cancer to the breast and axillary lymph nodes are rare with an estimated incidence of 0.07% [6], diagnosis of which can be challenging as it shares many clinicopathological characteristics with the primary tumours that arise at these locations. Metastatic lesions in the breast often present as a discrete mass with accompanying axillary lymphadenopathy, thus leading to initial diagnostic bias as primary breast carcinoma. Consequently, histopathological examination remains the mainstay of diagnosis in situations where clinical and radiographic findings are non-specific or misleading. The current case emphasises the significance of a diagnostic approach that gives priority to identifying morphological characteristics that favour metastatic carcinoma, especially when there is a history of extramammary malignancy and the histology does not match typical primary breast cancer. In small biopsy samples, where in-situ component is absent or has not been sampled, the papillary and glandular architecture of HGSOc can morphologically overlap with the primary breast carcinoma or other papillary neoplasms, thereby raising the diagnostic difficulty to the microscopic level, especially in cases when the clinical history is remote or lacking.

A major contributor to misdiagnosis in such cases is the assumption that the presence of in-situ-like architecture or apparent myoepithelial rimming excludes metastatic disease. As demonstrated in this case and supported by prior reports [7,8], metastatic ovarian carcinoma may show pseudo-in-situ patterns due to lymphovascular spread, ductal colonisation, or reactive stromal responses, underscoring the need for caution in overinterpreting these features. Accordingly, in-situ-appearing structures and basement membrane-like stromal encasement may indicate metastatic tumour colonisation rather than true carcinoma in-situ. Therefore, unless concordant histologic evidence and immunohistochemical confirmation are present, the existence of in-situ-like morphology should not be considered definitive proof of a primary breast origin. The diagnosis can be further refined by correlation with the morphology of the primary tumour and other metastatic deposits. To prevent diagnostic pitfalls, immunohistochemical evaluation should be carefully tailored based on tumour morphology and the patient's known history of non-breast cancer, using a broad panel that includes both breast-specific and site-specific markers. The [Table/Fig-4] illustrates differential diagnosis of papillary lesions in the breast [5,9,10].

Beyond histopathological and immunohistochemical differentiation, understanding metastatic pathways of ovarian carcinoma is equally important in interpreting breast and axillary lymph node involvement.

Entity	Clues on histopathology	Key positive immunohistochemistry markers	Key diagnostic clues
Primary breast carcinoma (papillary/invasive ductal carcinoma with papillary features)	Papillary or solid nests; in-situ component ±	GATA3, Mammaglobin, GCDFFP-15, ER/PR (variable)	Presence of DCIS; negative expression for PAX8, WT1, TTF-1
Metastatic HGSOC	Papillary architecture, psammoma bodies, high-grade nuclei	PAX8, WT1, CK7, aberrant p53	Clinical history, expression of Müllerian markers; negative expression for GATA3, Mammaglobin, GCDFFP-15
Metastatic lung adenocarcinoma	Papillary/acinar pattern	TTF-1, Napsin A	Often ER/PR negative; negative expression for PAX8, GATA3
Metastatic renal cell carcinoma (clear cell type)	Clear cells, rich vascular network	PAX8, RCC marker, CD10	Clear cytoplasm, sinusoidal vasculature; negative expression for GATA3, Mammaglobin
Metastatic papillary thyroid carcinoma	Papillae with nuclear grooves, inclusions	TTF-1, Thyroglobulin (most specific), PAX8	Negative expression for GATA3, WT1
Metastatic gastrointestinal adenocarcinoma	Glandular or papillary pattern	CK20, CDX2	CK7/CK20 pattern useful; negative expression for GATA3, PAX8

**[Table/Fig-4]:** Papillary lesions in the breast [5,9,10].

It is a rare instance to find metastases to the axillary lymph node in a case of primary ovarian carcinoma. In ovarian cancer cases, axillary lymph node metastases are rarely detected without breast metastases and often coexist with them. Serous carcinoma is the most prevalent kind of ovarian cancer that spreads to the breast [11]. Given that the ovary's lymphatic drainage flows toward the paracaval and para-aortic lymph nodes, as documented by Kleppe et al., it would make sense to assume that cancerous cells could disseminate to supradiaphragmatic lymph nodes, encompassing the axillary lymph nodes, from the ovaries [12].

Given the rarity of this metastatic pattern and its association with advanced systemic disease, management is largely palliative and individualised.

Current treatment option includes palliative radiation, lumpectomy, mastectomy, and systemic chemotherapy. Nevertheless, for this condition, there is no standard dose or fractionation protocol for radiotherapy for ovarian cancer metastases to the breast and axilla, and it typically is administered with palliative intent. On reviewing the literature, the main treatment modality for these cases is systemic chemotherapy with local radiation therapy. The radiation dosages described in the literature vary greatly and have been personalised according to symptom burden, previous therapies, institutional practice, and overall disease status. In the present case, given the

patient's chemoresistant disease and symptomatic breast lesion, palliative local radiation was chosen. The dose of 3000 cGy in four fractions was selected based on institutional practice and published literature [13,14]. Despite local symptom may improve with radiotherapy, the presence of breast metastases typically indicates advanced systemic disease and is linked to poor overall prognosis.

Almost all cases of ovarian cancer that have spread to the breast and been reported in the literature have been indicative of advanced, multidrug-resistant disease. This feature is particularly evident in recurrence cases where patients underwent several rounds of sequential chemotherapy after developing recurrence of breast metastases. Patients of ovarian carcinoma who are affected with metastases to the breast are known to have advanced chemoresistance and carry a dismal prognosis.

In the literature, radiation is used as the primary treatment for breast metastases in only a tiny percentage of these case reports, especially in chemoresistant cases. Local radiation could quickly reduce the tumour and control symptoms [15]. Before beginning definitive or palliative therapy, a precise histopathological diagnosis is crucial due to the overlapping clinical and radiological characteristics and the management implications.

**Role of histopathology and immunohistochemistry:** Histopathological examination has a crucial role in establishing the diagnosis of metastatic HGSOC at uncommon sites such as breast and axillary lymph nodes, where clinical and radiological findings frequently mimic a primary breast malignancy. Morphologically, metastatic HGSOC shows papillary, micropapillary or glandular architecture along with high-grade nuclear atypia. These features may show overlap with invasive ductal carcinoma of the breast, particularly in small core biopsy, especially in the absence of in-situ components. In such situations, immunohistochemistry plays an important role for accurate tumour categorisation. The expression of Mullerian markers such as PAX8 and WT1 shows ovarian origin, while the expression of aberrant p53 shows molecular hallmark of HGSOC. In the current case, the absence of breast-specific markers such as ER, PR, HER2 and GATA3 excludes a primary breast malignancy. Thus, despite a long disease-free interval and lack of primary tumour tissue, the characteristic histomorphology and supportive immunohistochemistry (PAX8+, WT1+, aberrant p53 and GATA3-) enabled a definitive diagnosis of metastatic HGSOC, highlighting the importance of histopathology together with supportive IHC in resolving this diagnostic pitfall.

Similar diagnostic challenges and clinicopathological patterns have been documented in previously published case reports and small case series [8,11,13].

Metastases from ovarian carcinoma to the breast and axillary lymph node are extremely uncommon that accounts for <1% of all breast malignancies [14]. Most reported cases have high-grade serous histology and manifest many years after the initial diagnosis. They are frequently misdiagnosed as primary breast malignancy until histopathology and IHC establish the Mullerian origin. The illustrates review of similar reported cases showing metastases to the breast [Table/Fig-5] [8,11,13-19].

Author (year)	Age (years)	Time to diagnose breast metastases	Initial treatment for breast metastasis	Recurrence; Time for recurrence	Treatment for recurrence	Survival time after diagnosis
Present Study (2025)	35	6 years	Local radiation (3000 cGy)	-	-	>3 months (ATP)*
Maeshima Y et al., 2021 [8]	69	9 years	Chemotherapy	-	-	24 months (ATP)*
Tempfer CB et al., 2016 [11]	51	1 year	Chemotherapy, Mastectomy	-	-	6 months (LTF)**
Dueño S et al. 2024 [13]	59	2 years	Local radiation (2300 cGy), Chemotherapy	Yes; 8 months	Chemotherapy	24 months (ATP)
Caruso G et al. 2020 [14]	53	2 years	Chemotherapy	Yes; 2 months	Local radiation (3500 Gy), Chemotherapy	36 months (death)
Wang J et al. 2020 [15]	40	6 years	Chemotherapy	-	-	4 months (LTF)**
Abu-Tineh M et al., 2020 [16]	54	14 months	Chemotherapy, Local radiation (3000 cGy)	-	-	24 months (ATP)*
Antuono L et al., 2018 [17]	54	3 years	Chemotherapy	-	-	5 months (ATP)*
Amichetti M et al., 1990 [18]	50	10 months	Excision, Local radiation	-	-	8 months (death)

Chiva I et al., 2022 [19]	79	7 years	Chemotherapy, Palliative radical mastectomy	-	-	2 months (death)
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**[Table/Fig-5]:** Previously reported cases of breast metastases from ovarian cancer [8,11,13-19].

\*ATP: Alive at the time of publication of this case report; \*\*LTF: Lost to follow-up; data summarized from previously published case reports

When taken as a whole, these previously documented cases show how uncommon breast metastases from ovarian cancer are across a broad age range and varying latency periods after first diagnosis. The lack of standard guidelines for management is shown in the table, which also emphasises the variety of treatment options, such as palliative radiation, surgery, and systemic therapy. Survival outcomes following diagnosis of breast metastasis are variable but generally limited, reinforcing the aggressive nature of the disease and the associated diagnostic and therapeutic challenges.

## CONCLUSION(S)

Metastasis of HGSOV to breast and axillary lymph nodes is a rare incidence and causes diagnostic challenges, especially when it occurs after a long disease-free interval. On clinical and radiological assessment, such presentations can mimic primary breast cancer, leading to potential misdiagnosis. The current case report highlights the importance of histopathologic examination, supported by appropriate immunohistochemistry panel, including Mullerian markers such as PAX-8 and WT-1, helping in establishing correct diagnosis. Crucially, in-situ-like architecture should be viewed cautiously since metastatic tumours may exhibit pseudo-in-situ patterns and should not be regarded as definitive proof of primary breast cancer in the absence of supporting histologic and immunohistochemical correlations. It is important to recognize this unusual metastatic pattern so as to avoid unnecessary surgical intervention and guide appropriate systemic and palliative management, while the limitations in molecular characterisation (BRCA1/2 and HRD testing), reflects practical difficulties frequently encountered during long-term follow-up of ovarian carcinoma.

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