ORIGINAL ARTICLE

Primary Peritoneal Serous Carcinoma Presenting as Inflammatory Breast Cancer

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■ **Abstract:** Metastasis to the breast from extramammary malignancies is rare. Nevertheless, its recognition is important because the prognosis and treatment differ from that of primary breast cancer. We report a unique case of primary peritoneal serous carcinoma that initially presented as inflammatory breast cancer. The patient received neoadjuvant chemotherapy for breast cancer and subsequently underwent bilateral total mastectomy and bilateral sentinel lymph node biopsy. She was found to have extensive intralymphatic carcinoma in both breasts, with only focal minimal breast parenchymal involvement, and residual metastatic carcinoma in bilateral sentinel lymph nodes. Further work-up revealed pelvic ascites and omental nodularities. The patient underwent laparoscopic bilateral salpingo-oophorectomy, which revealed high-grade serous carcinoma involving both ovaries and fallopian tubes. Molecular testing of tumor from the ovary and axillary lymph node showed an identical pattern of allelic loss, confirming a common origin for both tumors. To our knowledge, this is the first reported case of an extramammary primary malignancy that not only presented as inflammatory breast cancer but also was diagnosed and initially treated as such. ■

Key Words: breast cancer, inflammatory carcinoma, primary peritoneal, serous carcinoma

Ovarian or peritoneal serous carcinoma metastasizing to the breast and/or axillary lymph nodes is uncommon. Fewer than 50 cases have been reported previously, of which fewer than five were primary peritoneal carcinomas (1–12). Most of these have occurred in patients with a known history of primary ovarian or peritoneal carcinoma. It is rare for primary ovarian or peritoneal carcinoma to present clinically as a primary breast tumor, and it is rare for metastatic carcinoma from any extramammary site to present with signs of inflammatory breast cancer (6,7,9, 11,13–16). Although several cases of metastatic carcinoma from various sites have produced clinical signs mimicking inflammatory breast cancer, to our knowledge there is no previous report of metastatic carcinoma that not only presented as inflammatory breast carcinoma but also was diagnosed and initially treated as such.

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We report a unique case of primary peritoneal serous carcinoma that presented clinically as inflammatory breast cancer. The patient was treated with neoadjuvant chemotherapy for breast cancer, and the primary peritoneal tumor was not detected until 16 months after the initial breast manifestation.

CASE REPORT

A 56-year-old Caucasian woman with no family history of breast cancer and negative annual mammograms for seven consecutive years presented with slight redness around the periareolar area of the right breast. She was prescribed antibiotics, but the redness did not improve. A few weeks later, redness involved the left breast as well. Bilateral mammograms and MRIs were obtained, and these were negative. She had punch biopsies performed on both breasts, and each of these showed carcinoma in the dermal lymphatics. In a survey for metastatic disease, an MRI of the brain was negative. PET and CT scans from the base of the brain to the proximal thighs showed the

presence of a right pleural effusion, with abnormal hypermetabolic activity within the fluid highly suspicious for malignancy. Possible metastatic involvement of the right pleura and sternum was also noted. No other clear-cut evidence for metastatic disease was noted in the neck, chest, abdomen, or pelvis. Thoracentesis confirmed the presence of malignant cells within the pleural fluid.

The patient underwent induction chemotherapy consisting of four cycles of doxorubicin and cyclophosphamide for presumed inflammatory breast cancer, followed by four cycles of paclitaxel. A repeat PET scan no longer showed evidence of a right pleural or sternal lesion. A small amount of pelvic fluid was noted, which was thought likely to be physiologic. The patient then underwent bilateral simple mastectomy with bilateral sentinel node biopsies. Intralymphatic carcinoma was identified in both breast specimens, with additional minimal parenchymal involvement in the right breast. One sentinel node on the right and two on the left were positive for metastatic carcinoma. The tumor was strongly and diffusely positive for Wilms' tumor-1 antigen (WT-1).

Following surgery, the patient underwent chest wall radiation and was treated with anastrozole. CT of the chest subsequently revealed a recurrent pleural effusion. Thoracentesis was performed, followed by pleurodesis, and cytologic evaluation was again positive for metastatic carcinoma. A CT scan and MRI of the pelvis and abdomen revealed nodularities throughout the omentum and a small amount of ascites. The uterus and adnexa appeared to be of normal size. A PET scan showed that the omental nodules and ascites were not (^{18}F)-fluorodeoxyglucose avid. The serum CA125 level was significantly elevated at 274.1 U/mL (reference range 0–35 U/mL), and the CA27.29 level was increased to 175.7 U/mL (reference range 0–38 U/mL).

Eleven months after her initial presentation, the patient underwent a laparoscopic bilateral salpingo-oophorectomy and endometrial curettage. The right and left ovary were of normal size, but both ovaries and fallopian tubes were found microscopically to be involved by high-grade serous carcinoma. The endometrial curettage was negative. The histologic appearance and the extent and pattern of involvement fulfilled Gynecologic Oncology Group (GOG) criteria for primary peritoneal carcinoma (17). Subsequent DNA polymorphism analysis showed an identical pattern of allelic loss in 8 of 8 microsatellite foci, confirming that tumor above and below the diaphragm was of common origin. CT scans following laparoscopic surgery revealed progression of the intraabdominal disease. Omental caking greater than 8 cm was observed, and a second mesenteric nodule greater than 5 cm was noted. There was additional involvement of the liver and gastrocolic ligament with associated ascites.

Because of active abdominal and extraabdominal disease, the patient subsequently underwent additional chemotherapy for a peritoneal primary with docetaxel and carboplatin rather than debulking. At last follow-up, 16 months after her initial presentation, the intraperitoneal tumor volume had decreased dramatically. The omental involvement had decreased to approximately 3 cm and stabilized, and the CA125 and CA25.29 levels had decreased to 29.3 and 57.7, respectively.

PATHOLOGIC FINDINGS

Punch biopsies of both breasts prior to neoadjuvant chemotherapy showed dermal lymphatic carcinoma, which consisted of small clusters of high-grade carcinoma admixed with individual tumor cells within the lymphatic channels (Fig. 1). The tumor cells had a high nuclear to cytoplasmic ratio with either hyperchromatic or vesicular nuclei with prominent nucleoli. The tumor cells were negative for both estrogen and progesterone receptors, and FISH for evaluation of HER2 gene copy level was negative for amplification (the HER2:CEP17 ratio was 1.1). The Ki-67 labeling index was 25%.

Following neoadjuvant chemotherapy, no mass was identified in either of the mastectomy specimens, but microscopically, there was extensive involvement of lymphatic channels, including the dermal lymphatics, by high-grade carcinoma. On the right, there was also focal minimal breast parenchymal extension. One sentinel node on the right and two on the left contained clusters of high-grade metastatic carcinoma with a papillary morphology and associated psammomatous calcifications (Fig. 1). The metastatic tumor was positive for estrogen receptor, negative for progesterone receptor, and negative for HER2 amplification (the HER2:CEP17 ratio by FISH was 1.2). The Ki-67 labeling index was 47%. Immunohistochemical staining showed the tumor cells in the lymph node to be strongly and diffusely positive for WT-1 and negative for gross cystic disease fluid protein-15 (GCDFP-15), favoring metastatic serous carcinoma (Fig. 2).

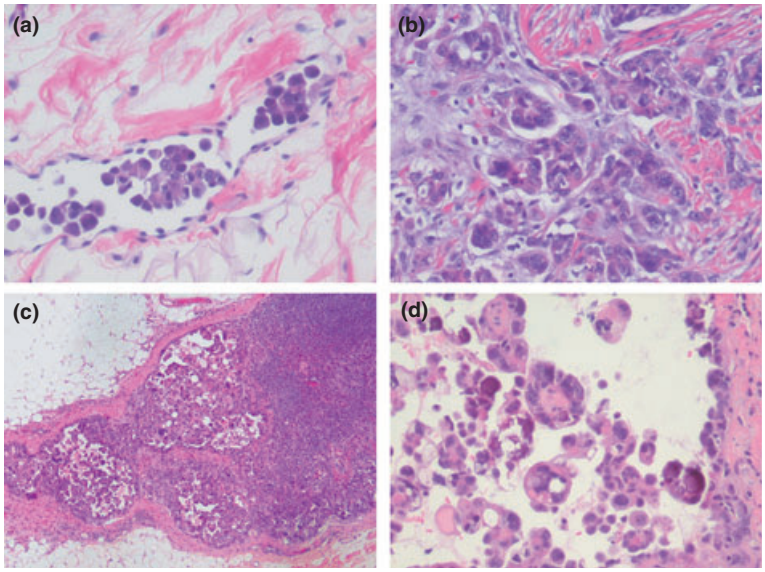

Figure 1. (a) Carcinoma cells within a dermal lymphatic channel of the breast. (b) Focal breast parenchymal involvement by invasive carcinoma. (c) Metastatic carcinoma in an axillary sentinel lymph node. (d) Psammomatous calcifications associated with the metastatic carcinoma.

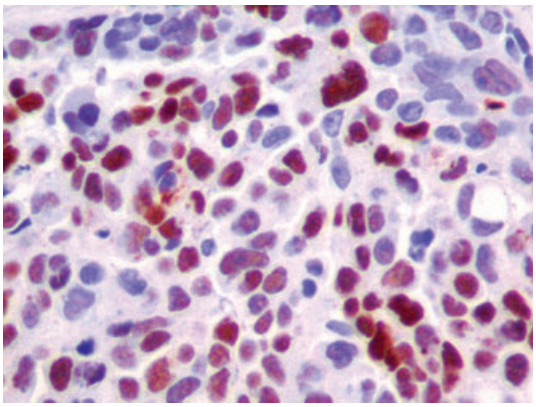

Figure 2. The tumor cells are diffusely and strongly positive for WT-1.

In the subsequent bilateral salpingo-oophorectomy specimen, high-grade papillary serous carcinoma involved both ovaries and fallopian tubes. The tumor in the ovaries involved the ovarian surface and underlying cortical stroma, with no parenchymal nodule larger than 5×5 mm (Fig. 3). Molecular testing was performed on tissue from the ovary and on metastatic tumor in axillary sentinel lymph nodes to confirm a common origin. Tissue was microdissected from three histologically distinct sites in the ovary (normal ovary, papillary serous carcinoma on the surface of the ovary, and nonadjacent infiltrating carcinoma) and on metastatic deposits of carcinoma in each of two involved axillary sentinel lymph nodes, and the pattern of random allelic loss of polymorphic microsatellite loci in the tumor from each site was compared. The pattern of allelic loss was found to be identical in 8 of 8 microsatellite loci. LOH mutations at both tumor sites were observed in 3p (5 foci), 17p, 17q, and 22q.

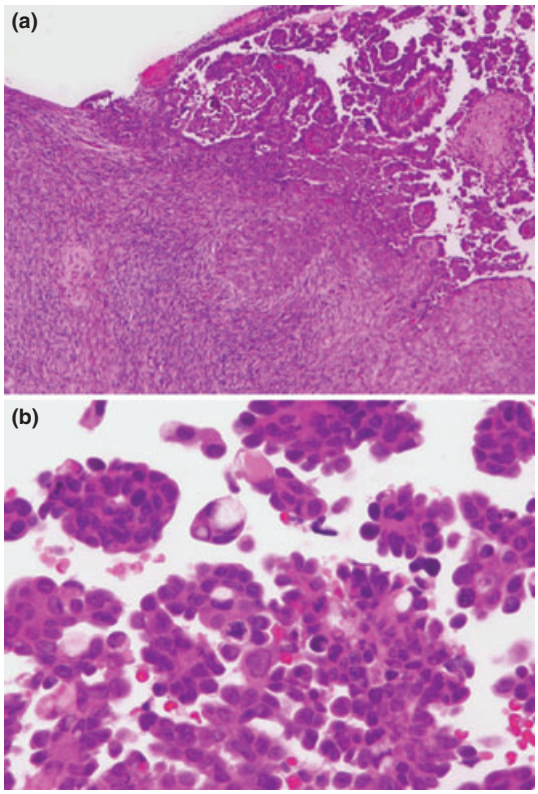

Figure 3. (a) Ovarian involvement by tumor was confined to the ovarian surface and underlying cortical stroma. (b) The tumor involving the ovary displays the typical morphology of papillary serous carcinoma and is morphologically similar to the tumor above the diaphragm.

DISCUSSION

The case we describe is, to our knowledge, the first report of an extramammary primary malignancy that not only presented as inflammatory breast cancer but also was diagnosed and treated as such. It is not surprising that the site of origin in this case was not detected until after the patient was treated for inflammatory breast cancer, as a punch biopsy with even scant intralymphatic carcinoma cells is generally regarded as confirmatory for inflammatory breast cancer in a patient without a history of an extramammary malignancy who presents with a swollen, erythematous breast. The primary peritoneal tumor in this patient was not detected until 11 months after the initial diagnosis of inflammatory breast cancer.

There have been rare reports of metastatic tumors to the breast mimicking inflammatory breast carcinoma. Six of these described metastases of ovarian origin, all of which were recognized as metastases following a previous diagnosis of primary ovarian carcinoma (6,7,9,11,18,19). Three patients with metastatic gastric carcinoma had clinical signs of inflammatory breast cancer, and each of these were similarly recognized as metastases following a previous diagnosis of signet ring cell carcinoma of the stomach (13–15). One series of 16 patients with metastases to the breast describes four patients who had ''diffuse skin thickening of one breast similar to inflammatory breast cancer.'' One of these was found to be metastatic squamous cell carcinoma of the tonsil, and another was metastatic squamous cell carcinoma of the lung. Both of these were in patients with known extramammary primaries. The breast lesion in a third case was the first manifestation of pancreatic adenocarcinoma, but it is not stated whether the lesion was initially diagnosed or treated as breast cancer. A fourth case might also have been a metastasis from an extramammary primary, although poorly differentiated adenocarcinoma of the breast was included in the differential diagnosis (16).

The breast is an uncommon site for metastasis. The reported incidence from autopsy series ranges from 1.7% to 6.6% (20,21), whereas the range is from 0.5% to 1.3% in clinical series (22). Most metastases to the breast appear as unilateral, well-circumscribed masses that generally lack microcalcifications. Of tumors that metastasize to the breast, the most common is carcinoma of the contralateral breast, followed by tumors of hematologic origin, melanoma, and bronchogenic carcinoma (5,23). Although metastases to the breast of ovarian origin are not the most common, they appear most likely to produce a clinical picture of inflammatory breast cancer, as described above. In all previous reports, however, this has occurred in the setting of a known extramammary primary.

Breast metastases of ovarian origin usually occur as single or multiple unilateral breast masses in patients with a history of advanced-stage ovarian carcinoma. A few patients have presented with bilateral breast masses. A majority of patients have also presented with synchronous axillary lymph node involvement (1,11). Metastases to the breast from ovarian primaries generally occur 2–3 years after the initial diagnosis (24). Breast metastases occurring before or concurrent with the initial presentation of an ovarian or peritoneal carcinoma are rare. Only 10 such cases are reported in the English literature (2,3,5,10–12,25,26), two of which presented initially as a breast mass before a primary tumor of the ovary was recognized (2,5). Metastases to the breast from primary peritoneal serous carcinomas are also rare (11). Two of the three cases of primary peritoneal carcinoma reported by Recine et al. that metastasized to the breast did so 11 and 16 months after the initial diagnosis of primary peritoneal carcinoma (11). The third case had a metastasis to the breast concurrent with the initial presentation of a primary peritoneal tumor.

The tumor in the breast and axillary lymph nodes in this report was not suspected to be metastatic carcinoma from an undiscovered ovarian or peritoneal primary until after the patient received neoadjuvant chemotherapy and the breast and axillary nodes were resected. Although the residual tumor in the breast was scant and predominantly intralymphatic with focal parenchymal extension, there was sufficient residual tumor in the lymph nodes to recognize the papillary architecture with associated psammomatous calcifications. The differential diagnosis of tumors in the breast with this morphology includes primary invasive micropapillary carcinoma of the breast and metastatic micropapillary carcinoma, most frequently of ovarian or peritoneal origin. Both appear as micropapillary clusters of tumor cells invading the stroma with prominent characteristic retraction artifact mimicking lymphovascular invasion (27).

Primary invasive micropapillary carcinoma generally presents as a mass with indistinct borders, whereas metastatic micropapillary carcinoma is

usually observed radiographically as one or more round, sharply circumscribed masses (28). The presence of associated intraductal carcinoma (DCIS) favors a primary breast tumor (27). Although immunohistochemical staining for gross cystic disease fluid protein-15 (GCDFP-15) can be positive in up to 50% of breast cancers, it is an apocrine marker and is generally absent in the invasive micropapillary subtype of breast cancer (29). WT-1 expression generally favors an ovarian or peritoneal primary, as most papillary serous carcinomas are strongly positive, whereas only a minority of invasive micropapillary carcinomas of the breast express WT-1, and those that do have only focal expression (30–32). Levels of serum CA125 are not useful in this differential diagnosis, as they may be elevated with breast, ovarian, or peritoneal primaries (33,34).

The tumor in this report lacked an associated intraductal component and was diffusely positive for WT-1 by immunohistochemistry and negative for GCDFP-15. The patient was subsequently found to have ovarian involvement, with the bulk of tumor radiographically in the omentum, and the tumor in the axillary nodes was shown to have a pattern of allelic loss identical to that of the pelvic tumor.

To determine whether tumor at two different sites is derived from the same original neoplasm, DNA polymorphism analysis can be performed to analyze the pattern of random allelic loss of polymorphic microsatellite loci at each site. In essence, the tumor's ''genetic fingerprint'' at each site can be compared (35,36). In this case, an identical pattern of allelic loss was found in 8 of 8 microsatellite loci. Moreover, the specific LOH mutations observed included mutations at 17p and 17q. Although BRCA1 is located on chromosome 17, it is known to undergo mutation in only a small proportion of breast carcinomas, and this patient's tumor morphology was not typical of patients with BRCA mutations (37). LOH mutations in both 17p and 17q are commonly described in primary peritoneal serous carcinoma and are not common in breast cancer (38–40).

Because the pelvic tumor was recognized histologically to be high-grade papillary serous carcinoma and ovarian involvement was confined to the ovarian surface and underlying cortical stroma, with no tumor nodule in the ovary greater than 5×5 mm, the pelvic tumor fulfilled GOG criteria for primary peritoneal carcinoma (17). The demonstration of common origin by DNA polymorphism analysis confirmed that the tumor above the diaphragm was a metastasis from the peritoneal primary rather than a separate primary. Although the prognosis of stage IV primary peritoneal carcinoma is poor even with the most appropriate treatment (median survival is 1.3 years compared with 2.9 years for inflammatory breast cancer), the recommended chemotherapeutic regimens for primary peritoneal carcinoma and inflammatory breast cancer are different, and misdiagnosis will result in inappropriate treatment (41,42).

In summary, the clinical presentation of an erythematous, swollen breast has a broad differential diagnosis that includes conditions ranging from nonneoplastic disease (such as cellulitis, deep venous thrombosis, radiation-induced change, and panniculitis) to neoplasia, with inflammatory breast carcinoma being the most common neoplastic etiology. Our reported case highlights a rare but clinically crucial pitfall that can occur in diagnosing inflammatory breast cancer. A bilateral presentation should always raise the possibility of metastatic disease, as bilateral inflammatory breast cancer is exceedingly rare (43). In the case we describe, the patient presented with unilateral redness and was treated initially with antibiotics. A few weeks later, redness involved the left breast as well.

With a classic unilateral presentation in the absence of a known extramammary malignancy, routine staining of punch biopsy specimens for WT-1 is not warranted. However, the presence of a micropapillary architecture in tumor from a breast biopsy specimen prior to neoadjuvant chemotherapy or from the subsequent surgical resection specimen should alert the pathologist to the possibility of metastatic disease of ovarian or peritoneal origin. In this situation, a thorough clinical evaluation for a possible pelvic primary tumor should be performed, and if ovarian or pelvic involvement is detected, staining for WT-1 and DNA polymorphism analysis can be useful in helping to confirm a common mullerian origin for tumor above and below the diaphragm.

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