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Case Report

Tarlatamab Treatment for Metastatic Small Cell Neuroendocrine Carcinoma of the Breast: A Case Report

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Abstract

Background: Since their first inception, neuroendocrine neoplasms of the breast have undergone many iterations of classification and definition. Taken together with their extremely low incidence, the ever-evolving landscape has made diagnosis, treatment, and research exceedingly challenging.

Case presentation: Our patient is a 45-year-old female who had a breast mass with clinical workup favoring a breast primary. A diagnosis of small cell carcinoma was established after an extensive clinical and pathologic workup. The mass did not respond to traditional neoadjuvant platinum-based chemotherapy, and she underwent a total mastectomy. Shortly thereafter, imaging revealed widely metastatic disease involving the brain, chest, abdomen, and pelvis. She received whole-brain radiation and, given the previous lack of response to chemotherapy, was recommended to try off-label tarlatamab. To date, with a short-term follow-up of 5 months, after whole-brain radiotherapy administered concurrently with the first cycle of tarlatamab, subsequent imaging showed near-complete radiographic resolution of extracranial metastatic disease, while the brain lesions also showed radiographic resolution on follow-up MRI.

Conclusions: We report the novel and preliminary use of tarlatamab in a patient with small cell neuroendocrine carcinoma of the breast, with short-term follow-up. We also discuss the most critical challenges associated with this rare pathologic diagnosis and the important elements to consider to ensure that metastatic disease is excluded.

Introduction

Primary neuroendocrine neoplasms (NENs) of the breast are rare, representing <1% of breast neoplasms,[1] and are the rarest of all NENs (<1%).[2,3] Estimates of incidence range from <0.1% to as high as 20%,[1] incidences that have changed over time as our definitions of the entity have shifted. Initially defined in the 3rd edition of the World Health Organization (WHO) Classification of Tumors in 2003, the defining criteria have changed with every edition, including the recently released 6th edition. Their rarity, compounded by inconsistencies and variability in diagnostic criteria, has made studies regarding tumor biology, treatments, and outcomes very difficult.

As it stands in the most recent 6th edition of the WHO Classification of Breast Tumours, the unified NEN classification used at other anatomical sites is not directly applicable to the breast. Primary breast neuroendocrine tumors are exceptionally rare, whereas small cell neuroendocrine carcinoma (SCNEC) is recognized as a specific cytomorphological pattern of invasive breast carcinoma with neuroendocrine morphology. Importantly, an extramammary primary site must be excluded before diagnosing primary breast SCNEC. SCNEC of the breast accounts for approximately 0.1% of all breast cancers,[2,4,5] while representing approximately 4–10% of all extrapulmonary SCNECs.[6-8] The histomorphology of breast SCNEC is identical to that of SCNECs arising in other organ sites. Furthermore, distinguishing primary breast SCNEC from metastatic SCNEC is difficult, as immunohistochemical staining may not be very helpful due to substantial overlap in immunophenotypic profiles. As such, clinical and radiologic correlation becomes essential. We herein report a case of SCNEC of the breast to highlight the diagnostic and treatment challenges associated with this rare malignancy, as well as the preliminary therapeutic effect of an off-label treatment.

Keywords: Breast cancer; Neuroendocrine carcinoma; Small cell carcinoma; Metastasis; Immunohistochemistry; Tarlatamab.
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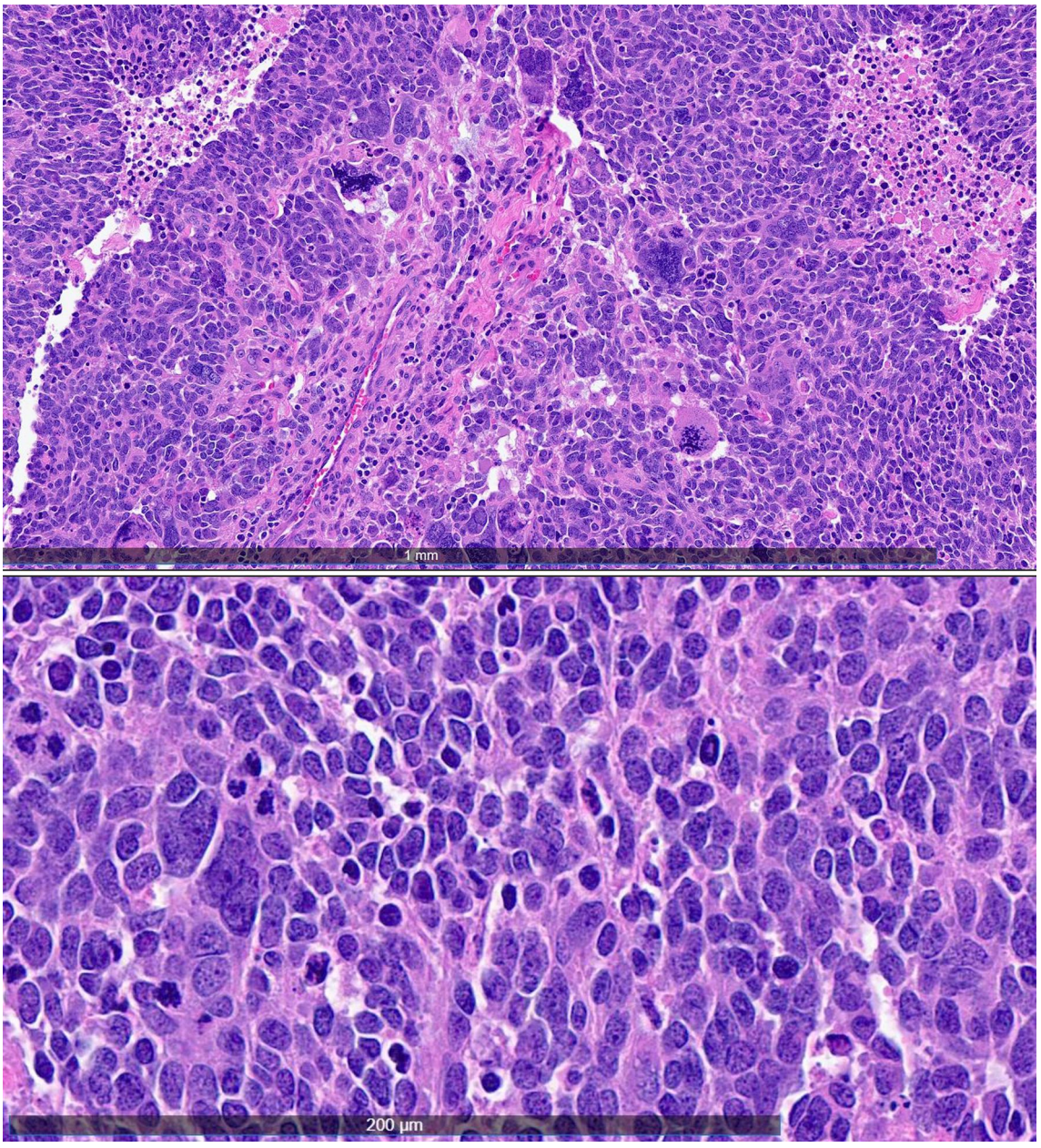


Fig. 1. H&E-stained sections (40× and 200× magnification, respectively) demonstrate solid sheets of tumor cells characterized by a high nuclear-to-cytoplasmic ratio, marked nuclear pleomorphism, nuclear molding, frequent mitotic figures, and focal necrosis. H&E, hematoxylin and eosin.

Case presentation

Our patient was a 45-year-old female and a former smoker who initially presented with a self-palpated 4 × 3 cm mobile mass in her left breast. She had no other symptoms or complaints, no relevant family history, and no prior history of breast screening. A lobulated mass was noted on mammography without any suspicious calcifications and was later confirmed by ultrasound, measuring 7.5 × 5.9 × 4.9 cm. A suspicious axillary lymph node was also noted. The contralateral breast revealed no abnormalities. Clinical metastatic workup, including computed tomography of the chest, abdomen, and pelvis and brain magnetic resonance imaging, did not reveal any evidence of disease elsewhere, and most notably, no suspicious lung or mediastinal lesions were seen. Initial biopsy results demonstrated solid sheets of tumor cells with high nuclear-to-cytoplasmic ratios, abundant nuclear pleomorphism, nuclear molding, marked mitotic activity, and areas of necrosis (Fig. 1). No *in situ* disease was present. Immunohistochemical staining showed that the tumor cells stained positively for CK7 and CK20 (both with perinuclear dot positivity), CD56, chromogranin, synaptophysin, and INSM1. The immunoprofile

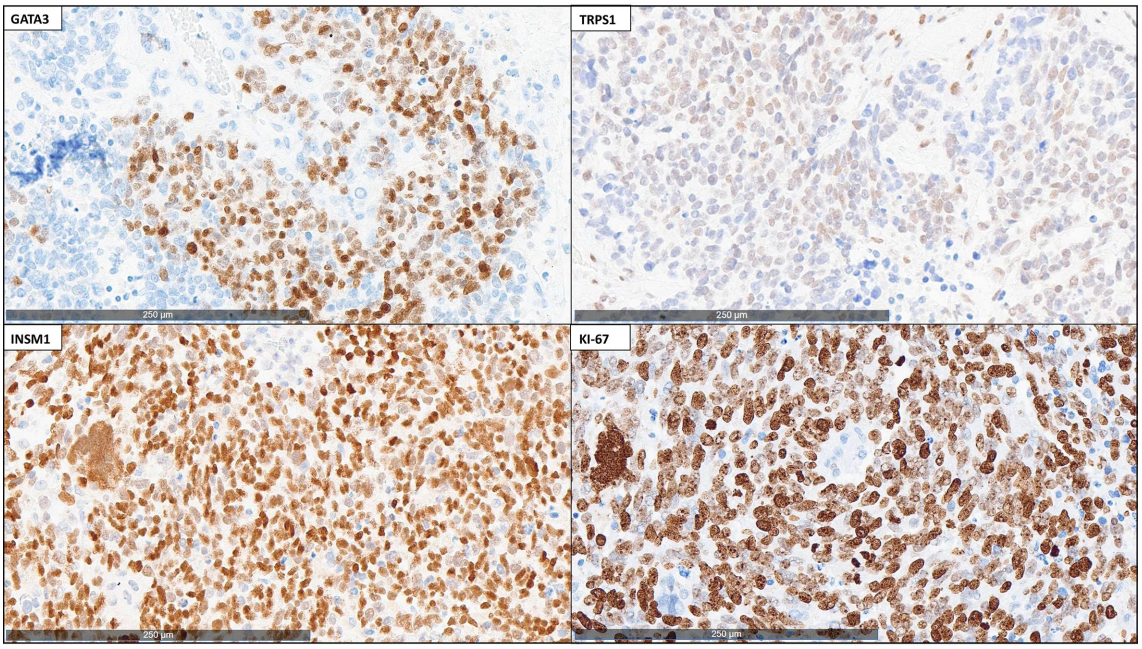


Fig. 2. The tumor cells from the resection specimen demonstrate focal expression of GATA3 and TRPS1 and diffuse expression of INSM1 (200× magnification). The Ki-67 proliferation index is approximately 100%. Scale bars represent 0.25 mm. TRPS1, trichorhinophalangeal syndrome type 1; INSM1, insulinoma-associated protein 1.

was most consistent with a high-grade NEC, morphologically consistent with small cell carcinoma. Additional stains performed on the biopsy were negative for GATA3, TRPS1, PAX8, SOX17, CDX2, estrogen receptor (ER), progesterone receptor (PR), human epidermal growth factor receptor 2 (HER2), TTF-1, and Merkel polyomavirus. Ki-67 staining showed a proliferative index approaching 100%. As no other masses were noted on radiologic examination, specifically no evidence of lung disease, and the patient had no history to suggest gastrointestinal or gynecologic sources, this was favored to represent a primary small cell carcinoma of the breast.

Given the rapid progression of disease, she was recommended to undergo neoadjuvant chemotherapy with cisplatin followed by etoposide, following a standard treatment regimen for small cell carcinoma of the lung. She began her treatment regimen approximately 3 weeks following the initial biopsy diagnosis. Each cycle consisted of etoposide 100 mg/m² given on days 1, 2, and 3 and cisplatin 75 mg/m² on day 2 of the treatment cycle. By cycle 3, there was concern for enlargement, and imaging findings were notable for an interval increase in tumor size, now measuring 9.8 × 7.8 × 6.8 cm, consistent with disease progression. An abnormal left axillary lymph node was also re-demonstrated. Given these findings, she was recommended to undergo interval surgical resection. An additional metastatic workup was otherwise negative. Two months following the start of chemotherapy, she underwent a left modified radical mastectomy and left axillary lymph node dissection. Pathologic findings were notable for a residual carcinoma measuring 10.5 cm and four of six lymph nodes positive for metastatic disease without significant treatment effect. There were multiple foci of lymphovascular invasion and dermal lymphovascular invasion. No *in situ* disease was noted. Repeat immunostains confirmed that the tumor was ER/PR/HER2 negative but was focally TRPS1 and GATA3 positive (Fig. 2). The excision specimen was likewise negative for TTF-1, mammaglobin, and GCDFP-15.

Due to the lack of response to neoadjuvant chemotherapy, she was recommended to proceed only with postmastectomy radiation to the chest wall and regional lymph nodes. However, prior to the start of radiation therapy, she experienced seizure activity, and imaging revealed findings concerning for metastatic disease in the brain. Further workup revealed marked interval progression of disease in the chest, abdomen, and pelvis, involving the lungs, mediastinum, kidneys, liver, gastrointestinal tract, and reproductive organs. She completed whole-brain radiation therapy with hippocampal avoidance (3,000 cGy over 10 fractions) over the course of 14 days and, given the rapid progression of disease, was simultaneously started on cycle 1 of tarlatamab (Imdelltra®) approximately 2 months following her surgical resection, and 5 months after initial diagnosis.

As tarlatamab is not approved for the treatment of small cell carcinoma of the breast, the patient was given the previously described regimen approved for lung small cell carcinoma. She was initiated on the first cycle of tarlatamab with the recommended "step-up" dosing schedule of 1 mg of tarlatamab on day 1, followed by 10 mg on days 8 and 15, with preemptive admission due to the risk of cytokine release syndrome (CRS). She did experience grade 2 CRS, requiring tocilizumab during this first cycle. Subsequently, she was able to receive cycles 2 through 5 in the outpatient setting, in 28-day cycles on days 1 and 15. She tolerated these cycles well, with constipation being her only remaining complaint.

At the time of writing of this manuscript, the patient is 10 months out from her initial diagnosis. Approximately 5 months

following the start of tarlatamab treatment and a 14-day course of whole brain radiation, the patient had completed five cycles of treatment. Interval computed tomography following cycle 3 (6 months after the initial diagnosis), demonstrated near-complete radiographic resolution of her extracranial disease in the chest, abdomen, reproductive organs, and lymph nodes, with no evidence of any new disease. Tumor response was not formally assessed using RECIST criteria. Magnetic resonance imaging of the brain obtained 1 month following whole-brain radiation therapy showed a decrease in the size of the brain lesions and imaging at 3 months showed complete radiographic resolution of the previously identified brain lesions. At the time of writing of this manuscript, no predetermined endpoint for tarlatamab therapy has been established, and the patient is currently scheduled to receive cycle 6 of therapy, followed by repeat interval imaging.

Discussion

Certainly, in the context of small cell carcinoma of the breast, excluding a lung primary is of the utmost importance. While metastasis to the breast from extramammary malignancies is extremely rare, with a reported incidence of 0.2–1.3%, in 11–30% of patients, metastasis to the breast is the first sign of malignancy.[9-11] However, it is particularly rare for a primary lung carcinoma to present as a breast metastasis,[12] a consideration that is important given that it is usually the most common differential diagnosis when encountering a NEC of the breast. NENs that do metastasize to the breast most commonly originate from the lung and gastrointestinal tract,[10,13-15] largely reflecting their overall frequency at these sites. Importantly, in the largest study to date of metastatic NENs to the breast, 87% of cases had extramammary metastases as well, with the most frequent other sites being the liver, lymph nodes, lungs, and bones.[15]

Immunohistochemistry is often deployed to help define the etiology of a lesion; however, the most important fact to remember when assessing for possible metastasis is that no immunohistochemical marker is 100% sensitive or specific. When it comes to smaller biopsies, focal positivity, necrosis, and processing factors need to be heavily considered. The case report described here is a perfect example of these pitfalls, as our biopsy was negative for definitive breast markers. NECs do appear to show less frequent ER/PR expression than conventional breast carcinomas (ranging from 12–50%) and typically demonstrate a triple-negative phenotype.[5,16-20] Conversely, it is important to note that while a large number of NENs of the breast will stain positively for ER/PR, that in and of itself should not exclude a possible metastasis, as primary NENs of the gastrointestinal tract and lung can also show positivity for these markers (in up to 61% of cases, with varying intensity).[21-23]

When assessing the utility of "organ-specific" markers, some pitfalls emerge as well. The largest study regarding TRPS1 in breast NENs had 56 total cases (39 NETs/17 NECs).[24] Within this cohort, nearly all NETs stained positively (97%), while only 23.5% of NECs stained positively for TRPS1. Similarly, GATA3 was negative in 94% of NECs and positive in 100% of NETs. While this was the largest cohort, it is important to note that it still included only 17 cases, a theme seen throughout most of the studies pertaining to this entity. Within that study, the authors also looked at cases from the lung and gastrointestinal system (a total of 30), with only one gastrointestinal NEC showing scattered positivity for TRPS1. These findings lead us to conclude that positive GATA3 or TRPS1 results are quite helpful, but we cannot entirely rely on a negative result. As lung SCNEC is typically one of the top differentials, TTF-1 immunohistochemistry is also universally used in the diagnostic workup. Approximately 80% of all primary small cell carcinomas (pulmonary and extrapulmonary) stained positively for TTF-1.[25] That study included only one case of breast small cell carcinoma, which stained positive. A follow-up study of 10 breast cases showed that 20% of cases had strong, diffuse nuclear staining for TTF-1,[19] while a more recent study showed that more than half of cases had positive TTF-1 expression.[5] Metastases from lung small cell carcinomas will stain positively for TTF-1 in 70–90% of cases.[26,27] These experiences with the varying specificity of immunohistochemical stains highlight the need not to rely entirely on the immunoprofile of a tumor and to take the whole clinical scenario into consideration.

Treatment for breast NECs mirrors the chemotherapy used in the lung and consists of a platinum agent and etoposide,[26,28-32] since biologic markers of small cell carcinomas of the breast are similar to those of the lung.[4] Other regimens also use taxane-based chemotherapy, FEC (5-fluorouracil, epirubicin, cyclophosphamide), and FAC (5-fluorouracil, Adriamycin, cyclophosphamide).[29] Conversely, others have used similar chemotherapy regimens and indications to those used for conventional breast carcinomas.[31] In the neoadjuvant setting, the most recent study suggests its use in 13% of cases,[28] with several case reports reporting good effectiveness and even pathologic complete response.[33-35] In light of the paucity of data, one study suggested the following parameters for consideration of neoadjuvant therapy: large mass (>5 cm), desire for breast conservation, locally advanced disease, or inoperable cases.[30]

Tarlatamab is a bispecific T-cell engager immunotherapy targeting delta-like ligand 3 (DLL3) on tumor cells and CD3 on T cells. DLL3 is expressed on the surface of 85–96% of patients with small cell lung cancer (SCLC). Tarlatamab redirects CD3-positive T cells to DLL3-expressing tumor cells, leading to tumor-cell lysis without relying on major histocompatibility complex class I recognition. It is a new treatment that has been approved by the U.S. Food and Drug Administration for use in patients with extensive-stage SCLC who have experienced progression following platinum-based chemotherapy. It is administered intravenously at a dose of 10 mg every 2 weeks. Approval was based on the DeLLphi-304 phase 3 trial,[36] in which treatment with tarlatamab demonstrated significantly longer overall survival than second-line chemotherapy (13.6 vs. 8.3 months) in patients who had progressed during or after platinum-based therapy. Previous accelerated approval, based on the DeLLphi-301 phase 2 trial,[37] demonstrated an objective response rate of 40%, a median duration of response of 9.7 months, and median overall survival of 15.2 months among patients with SCLC who had been previously treated with two or more lines of therapy. Although DLL3 is expressed in the vast majority of SCLC tumors, DLL3 expression level was not required for trial entry in that study, and among those who did have levels tested, it did not appear to predict response. Of note, limited data are available on DLL3 expression in the breast, but one study of 20 breast NECs did not demonstrate DLL3 expression.[38] The first dose is administered using a "step-

up" dosing schedule in a monitored setting with mandatory observation given the risk of CRS and immune-mediated neurotoxicity. This represents a significant advance over prior post-platinum therapies such as topotecan, which yielded objective response rates under 20% and median overall survival under 9 months.[36,39] ClinicalTrials.gov currently lists an active trial enrolling patients with metastatic extrapulmonary small cell carcinoma.[40]

Limitations

Our report is certainly limited in that we do not have long-term follow-up data yet, but our case highlights promising preliminary results in a metastatic case with otherwise limited treatment options. Follow-up data from the actively enrolling clinical trial will be further contributory and demonstrate whether this will become a possible treatment option for other patients and become more widely used as a treatment for small cell carcinomas of the breast. As this medication is designed to be given continuously until disease progression or intolerance and is individualized to each patient's response and side-effect profile, we do not yet know what an optimal length of treatment will be for our patient. Additionally, we do not have DLL3 expression data for the tumor in our case. However, as noted previously, prior data have demonstrated that DLL3 expression levels may not predict response to tarlatamab. Whether this finding applies to breast SCNEC remains unknown.

Conclusions

To our knowledge, our case is one of the first published experiences of the use of tarlatamab in the setting of metastatic breast SCNEC. Additionally, in the workup of our case, we highlight the limitations of immunohistochemistry in limited biopsy specimens and the importance of clinical and radiologic correlation when dealing with neuroendocrine small cell carcinomas.

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Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that influenced the work reported in this paper.

Author contributions

Writing – original draft (DP), review & editing (DP, HZ), and conceptualization (DP, HZ). Both authors have approved the final version and publication of the manuscript.

Ethical statement

Per the policies of the University of Rochester Office for Human Subjects Protection Research Subjects Review Board, formal ethical review is not required for individual anonymized case report studies. The patient's informed consent was obtained for the publication of this case report. This anonymized case report was conducted in accordance with the Declaration of Helsinki.

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