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Breast implant-associated anaplastic large-cell lymphoma: A rare diagnosis requiring multidisciplinary care

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Abstract

Breast Implant-Associated Anaplastic Large-Cell Lymphoma (BIA-ALCL) is a rare T-cell lymphoma associated with textured breast implants. Although often indolent, its nonspecific presentation can delay diagnosis and worsen prognosis. Due to the rarity of the disease, particularly in advanced presentations, treatment decisions are made on an individualized basis tailored to patient-specific disease characteristics. Here, we present the case of a 59-year-old female with a history of bilateral breast augmentation who developed a right peri-implant seroma and breast mass. Multiple imaging modalities and two rounds of ultrasound-guided biopsy were all suggestive of a benign abscess.

Definitive diagnosis of BIA-ALCL was confirmed after complete excision of the implants and capsules and pathologic evaluation. A staging Positron Emission Tomography (PET) scan demonstrated locally invasive stage IIE BIA-ALCL. The patient underwent induction treatment with three cycles of Brentuximab Vedotin, Cyclophosphamide, doxorubicin, and Prednisone (BV-CHP), followed by consolidative reduced-intensity radiation therapy with concurrent brentuximab vedotin, achieving a complete response. This case highlights the diagnostic challenges of BIA-ALCL, a multidisciplinary approach, and describes our strategy for managing locally advanced disease.

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Introduction

Breast Implant-Associated Anaplastic Large-Cell Lymphoma (BIA-ALCL) is a rare T-cell non-Hodgkin lymphoma that arises in the setting of textured breast implants [1]. It is a distinct subtype of Anaplastic Lymphoma Kinase (ALK)-negative anaplastic large cell lymphoma, a malignancy characterized by the proliferation of large, pleomorphic lymphoid cells that uniformly express CD30 [1,2]. BIA-ALCL most commonly presents with a peri-prosthetic fluid collection or seroma, typically developing around a decade after implant placement [1]. BIA-ALCL can be difficult to diagnose due to the rarity of the disease, as well as its nonspecific clinical presentation and immunophenotypic profile [3,4]. Here, we present a patient case that demonstrates these diagnostic challenges and outlines the management of locally advanced disease.

Case presentation

A 59-year-old female with a past medical history of hyperlipidemia presented to the emergency department with a 10-day history of right breast pain and redness, with pain radiating to the sternum. Past surgical history was also pertinent for a bilateral breast augmentation with submuscular 350 cc silicone

implants placed eight years prior. Her physical exam noted erythema and warmth of the right medial breast, as well as a firm, palpable mass in that area. Lab workup showed no elevation in white blood cells, no acute electrolyte abnormalities, and a normal lactate dehydrogenase. A chest Computed Tomography (CT) revealed a fluid collection medial to the right breast implant involving the right chest wall and abutting the sternum, with associated erosive changes. She was discharged on doxycycline with orders for further imaging due to concern for abscess versus malignancy.

Diagnostic mammography showed right medial skin thickening and a suspicious right breast mass, however it was located too far medially and posteriorly to fully evaluate radiographically. An ultrasound-guided biopsy of the right breast mass revealed acute and chronic inflammation with focal necrosis and suppurative inflammation consistent with an abscess. Sternal osteomyelitis was also suspected. Pancytokeratin Immunohistochemistry (IHC) stain was negative, supporting this diagnosis. The patient was referred to plastic surgery for discussion of implant removal and sternal debridement.

For surgical planning, a chest MRI obtained for further evaluation of the abscess revealed an infiltrative, anterolateral chest wall mass measuring at least 9.2×9.1×4.3 cm consistent with an infectious or inflammatory abscess and suspected sternal osteomyelitis. Internal mammary and retropectoral lymphadenopathy were also noted. Due to renewed concern for malignancy versus infection, a second ultrasound-guided soft tissue biopsy was obtained by interventional radiology, which again showed necrosis and mixed acute and chronic inflammation consistent with an abscess. No evidence of malignancy was found, with negative IHC for pancytokeratin and CAM5.2. With the biopsy suggesting infection, the patient underwent a bilateral capsulectomy, implant removal, and sternal debridement. The capsule and mass were sent for lymphoma workup. Blood cultures obtained were negative.

Hematoxylin and Eosin (H&E) sections of the right chest wall mass showed a nodular growth of large tumor cells with extensive central necrosis and surrounding fibrosis (Figure 1A). The tumor cells had mono- and occasional bi-lobed nuclei with prominent eosinophilic nucleoli and abundant cytoplasm, and were admixed with an inflammatory infiltrate composed of small lymphocytes, histiocytes, neutrophils, and occasional eosinophils (Figure 1B). The lymphoma cells were positive for CD30 (Figure 1C), CD15 (Figure 1D, focal/weak), MUM 1, CD4 (Figure 1H, focal), and EMA (focal/weak). They were negative for PAX5 (Figure 1F), CD20, CD79a, OCT2, BOB1, CD45 (Figure 1G), CD2, CD3 (Figure 1E), CD5, CD7, CD8, TIA 1, and ALK1. In situ hybridization study for EBV was negative. Flow cytometry study was performed; however, the specimen was insufficient for immunophenotypic analysis. The overall findings supported a diagnosis of CD30-positive large cell lymphoma consistent with BIA-ALCL. T-cell receptor gene rearrangement assay showed an equivocal result with a monoclonal T-cell population amidst polyclonal T-cells, findings that were suspicious for, but not definitive of, monoclonal proliferation. The sternum specimen showed changes consistent with fracture and negative for lymphoma. The capsule demonstrated synovial metaplasia without evidence of malignancy. These findings underscore the diagnostic challenges posed by such cases, particularly when immunophenotypic overlap with classic Hodgkin lymphoma (cHL) is present.

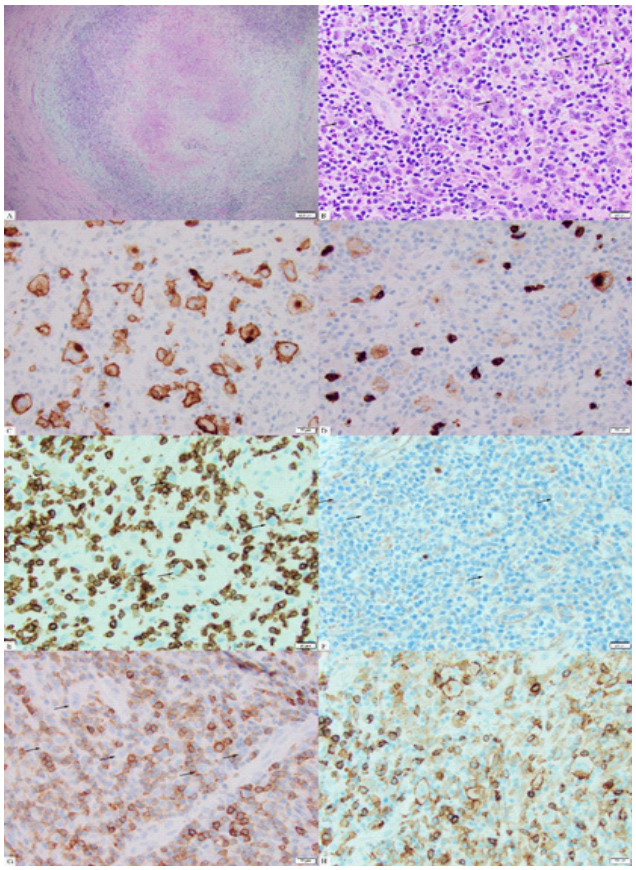

Figure 1: H&E of Breast implant-associated anaplastic large-cell lymphoma (A, ×40; B, ×400). CD30 (C, ×400). CD15 (D, ×400). CD3 (E, ×400), PAX5 (F, ×400), CD45 (G, ×400) and CD4 (H, ×400).

A Positron Emission Tomography (PET) scan demonstrated multiple foci of uptake along the anterior chest wall mass with involvement of the sternal body with a maximum Standardized Uptake Value (SUV) of 10.8. She was referred to hematology and diagnosed with Stage IIE BIA-ALCL due to extracapsular advancement without lymph node involvement. The presence of bone involvement rendered complete surgical excision impractical. She was therefore treated with three cycles of Brentuximab Vedotin, Cyclophosphamide, Doxorubicin, and Prednisolone (BV-CHP). An end-of-treatment PET scan showed significant improvement of the previously hypermetabolic anterior chest wall mass and sternal involvement, with no new hypermetabolic lesions, consistent with a complete response. She then underwent consolidation with reduced-intensity radiation therapy, where she was given 3600 cGy in 18 fractions, along with three concurrent cycles of brentuximab vedotin. Overall, treatment was well tolerated with no dose-limiting toxicities. A repeat PET imaging is planned in three months for restaging, and breast reconstruction may follow pending those results.

Discussion

BIA-ALCL is a rare subtype of ALCL associated with the placement of textured implants in the context of both post-mastectomy reconstruction and cosmetic breast augmentation [5]. While textured breast implants were previously utilized for their decreased risk of capsular contracture and rotation, their use in the US has declined to nearly zero since their association with BIA-ALCL was recognized by the World Health Organization in 2016 [6,7]. While these steps may reduce future instances of BIA-ALCL, as of 2023, an estimated 400,000 individuals in the US and over 10 million people worldwide still have textured implants, putting them at risk for this disease [1].

Estimates of prevalence and risk vary by population and study design. In the United States, BIA-ALCL is estimated to occur in approximately 1 in 914 patients with textured implants, with a prevalence of 109.4 cases per 100,000 patients [8]. As seen in our case, the median latency of BIA-ALCL is 7.5-11 years after implant placement [9].

While the pathogenesis of BIA-ALCL is not completely understood, it is thought that the surface and mechanical friction of the textured implant may contribute to the formation of a bacterial biofilm, leading to chronic inflammation, T-cell activation and expansion, and ultimately malignant transformation [10].

BIA-ALCL most commonly presents with a peri-prosthetic fluid collection or seroma, a palpable breast mass, and pain [11]. Other less common presentations include capsular contracture, breast skin changes, and systemic symptoms, such as fever, night sweats, and weight loss [11]. Imaging modalities, such as MRI and ultrasound, can detect a peri-implant seroma or mass, and are useful in the initial evaluation of this disease. However, imaging cannot differentiate between benign and malignant etiologies of these findings, necessitating pathologic evaluation [3]. Upon pathologic examination, there are three criteria needed for a diagnosis of BIA-ALCL: (1) cytology showing large pleomorphic lymphocytes with irregular nuclei, prominent nucleoli, and abundant cytoplasm; (2) a monoclonal T cell population on flow cytometry; and (3) an IHC stain positive for CD30 [4,12].

While clear guidelines have been established for diagnosis, there are several challenges complicating this process. From a clinical perspective, BIA-ALCL's nonspecific presentation of late seroma can be difficult to distinguish from other benign causes, such as infection [3]. This along with the rarity of the disease can make clinical suspicion for BIA-ALCL low. From a pathologic perspective, there are multiple factors complicating diagnosis. First, recognition can be difficult if pathologists are unfamiliar with BIA-ALCL, especially if the tumor burden is low [3]. BIA-ALCL can also have incomplete expression of T-cell antigens, as seen in our case, complicating the identification of a monoclonal T-cell population [4]. Lastly, this disease exhibits overlapping morphologic and immunophenotypic features with cHL and ALK-negative ALCL, further contributing to its diagnostic complexity. Therefore, given these added challenges, broad awareness of BIA-ALCL and the careful integration of clinical, radiologic, and pathologic findings are essential for timely diagnosis.

The treatment and prognosis of BIA-ALCL are highly dependent on its stage, as classified through the Lugano (Ann Arbor) system [12]. Surgical excision of the lymphoma, implants, and fibrous capsule is the first-line treatment for all stages [13]. Most patients present with Stage I disease, localized to the capsule, which enjoys a high cure fraction with surgical resection alone. However, in patients who cannot undergo complete excision, or those with disease extending beyond the capsule, there is no standard of care. Case series and expert opinion suggest benefit from radiation (24-36 Gy) and/or systemic therapy [14].

While there are currently no standardized treatment guidelines for advanced (Lugano II-IV) BIA-ALCL, a combination anthracycline-based chemotherapy, such as CHOP (cyclophosphamide, doxorubicin, vincristine, and prednisone) has demonstrated efficacy against BIA-ALCL [12]. Brentuximab vedotin (BV), a chimeric anti-CD30 monoclonal antibody linked to a microtubule inhibitor, has shown promising activity for this disease as well [12]. In our case, we pursued a combined modality therapy approach of induction BV-CHP for three cycles followed by consolidative radiotherapy with concurrent BV for three cycles. This decision was guided by several lines of evidence including: (1) data demonstrating the curative potential of three cycles of R-CHOP followed by consolidative radiation therapy in early stage diffuse large B-cell lymphoma (DLBCL) [15]; (2) data supporting a role for consolidative radiotherapy in early-stage Peripheral T-Cell Lymphoma (PTCL) [16]; and (3) data suggesting a radiosensitizing effect of BV [17,18]. The patient achieved a complete response on end of treatment PET/CT without dose-limiting toxicities.

The prognosis of BIA-ALCL varies based on the extent of the disease and its clinical presentation. The estimated three- and five-year overall survival rates are 97% and 92%, respectively, with a median overall survival of 12 years [19]. However, patients presenting with disease confined to the capsule and without a mass generally have more favorable outcomes, whereas those with more advanced disease or with a mass have lower overall and event-free survival rates [19,20]. Thus, given these findings, it is essential that clinicians maintain a high level of suspicion to maximize early recognition.

Conclusion

Here, we aim to illustrate the diagnostic challenges of BIA-ALCL including its rarity, nonspecific clinical presentation, and inconclusive imaging and biopsy results, as well as demonstrate our approach to managing locally advanced disease by using a combination of targeted therapy, chemotherapy, and reduced-intensity radiation. While the use of textured implants in the US has considerably declined, a large population of patients with existing textured implants remains at risk for BIA-ALCL. This case highlights the need for clinical vigilance across the multidisciplinary team and the development of standardized treatment guidelines.

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