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Breast Implant-Associated Anaplastic Large Cell Lymphoma Presenting as Left Shoulder Mass: A Case Report

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Introduction/Background

Breast implant-associated anaplastic large cell lymphoma (BIA-ALCL) is a rare type of non-Hodgkin lymphoma that can develop in the tissue surrounding breast implants [1]. This is not a type of breast cancer, but a cancer of the immune system. Anaplastic large cell lymphomas (ALCL) are a subset of T-cell lymphomas and are characterized by large T-cells with anaplastic morphology and CD30 positive phenotypes [2]. As reported in nearly all documented cases, BIA-ALCL is known to occur in implants with a textured surface (shell), as opposed to a smooth one [3]. Clinically, BIA-ALCL most commonly presents with breast asymmetry due to delayed peri-implant seroma or periprosthetic effusion surrounding the implant. Less common clinical manifestations include skin changes, capsular contracture or scarring, palpable breast masses, and regional lymphadenopathy [4]. Although the exact pathogenesis remains uncertain, chronic inflammation induced by the textured implant surface, bacterial biofilm formation, persistent T-cell stimulation, and host genetic susceptibility are proposed contributing factors in lymphomagenesis [5]. Diagnosis relies on a combination of clinical suspicion, radiological findings, histopathological examination of the capsule, and cytological evaluation of peri-implant effusion fluid with CD30 immunohistochemistry [4,5]. Imaging modalities such as MRI and PET-CT are often utilized to assess extracapsular spread, regional lymph node involvement, and distant metastases [6]. Standard treatment primarily consists of complete surgical excision including implant removal and total capsulectomy, while advanced cases may additionally require chemotherapy, targeted therapy such as brentuximab vedotin, and/or radiotherapy depending on disease extent [6,7]. The aim of this report is to highlight the importance of considering BIA-ALCL in patients with a history of breast implants presenting

with unusual symptoms or masses, even in locations distant from the breast.

Patient Presentation

A 50-year-old female patient presented with progressive left shoulder pain and immobilization for seven months, accompanied by increasing swelling over the left shoulder for two months. The patient had a medical history of hypertension, managed with medication. Patient had a history of bilateral breast implants placed approximately 20 years prior to presentation. There was a positive family history for breast cancer in her elder sister at age 50.

Case Description

The patient's primary complaints were left shoulder pain, restricted mobility, and swelling. Physical examination revealed two distinct masses: a 4 × 3.5 cm swelling anterior to the left shoulder above the clavicle, and a 4 cm firm swelling on the upper back, 4 cm above the scapular tip. Overhead abduction of the left arm was restricted. Patient had a history of bilateral breast implants 20 yrs back.

Diagnostic Assessment

MRI of left shoulder was suggestive of neoplastic etiology, likely lymphoma more than metastasis. Further evaluation with tissue sampling and histopathological correlation was recommended for further evaluation. Microscopically serially sectioned core needle biopsy showed a high-grade malignant tumor composed of large anaplastic cells having a high N:C ratio, nuclear pleomorphism, atypia and brisk mitotic activity. Cells with bizarre nuclei were seen and necrosis was noted (Figure 1).

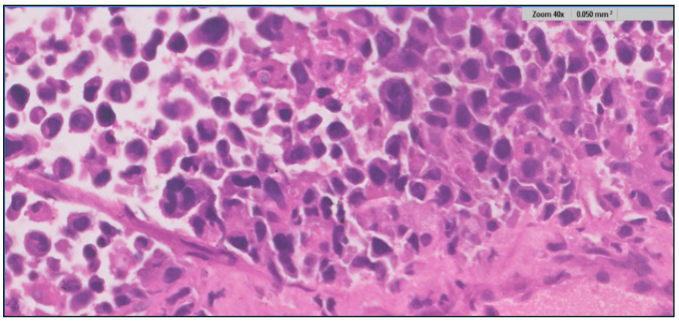

Figure 1: H & E Staining

Immunohistochemistry results were positive for Vimentin, LCA, CD3, BCL2, EMA, CD30, CD4 (variably) and negative for PAN CK, CD20, BCL6, CD8, ALK1, ALK (D5F3). Ki67 index was 80-90%. **(Figures: 2-7)**

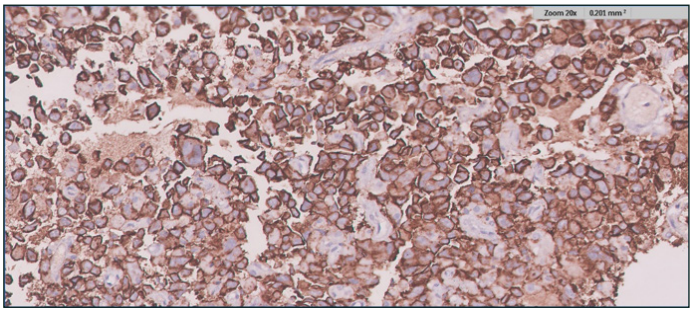

Figure 2: IHC marker for CD30

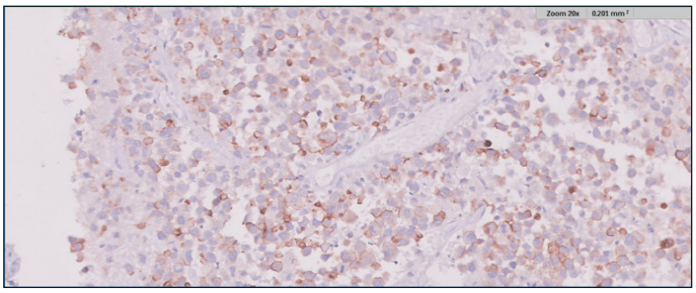

Figure 3: EMA IHC marker

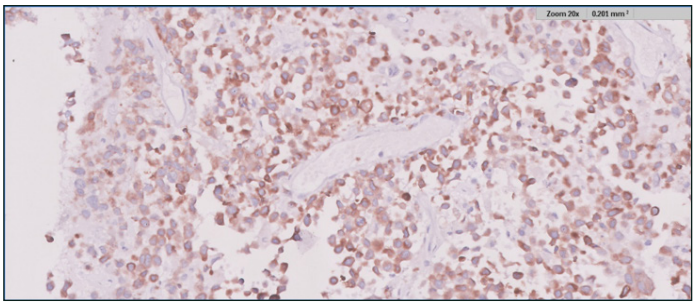

Figure 4: CD3a Marker

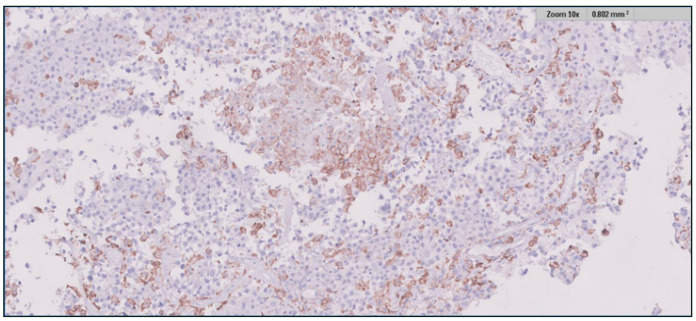

Figure 5: CD4 Marker

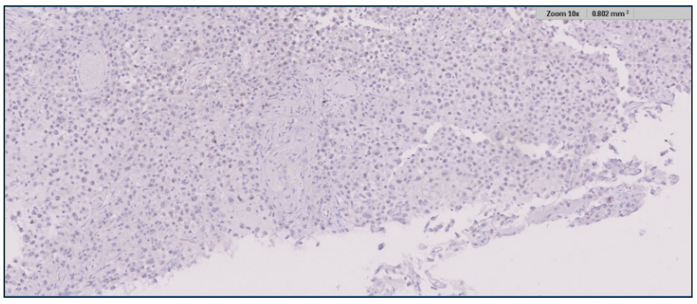

Figure 6: CD20 Marker

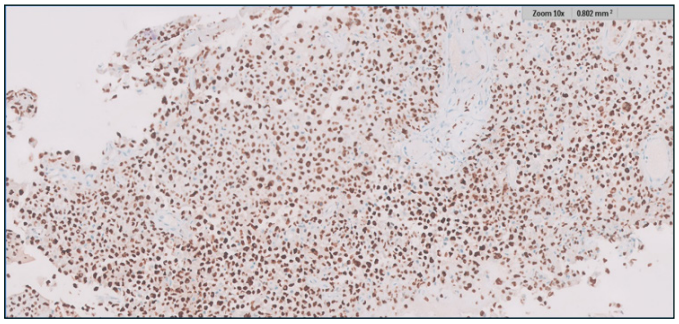

Figure 7: Ki67 Index

Final histopathological diagnosis revealed High-grade T-cell Non-Hodgkin Lymphoma, subtype - ALK-negative Anaplastic Large Cell Lymphoma (ALCL), i.e. Breast Implant-associated ALCL (BIA-ALCL)

Therapeutic Intervention

Standard treatment for BIA-ALCL typically involves surgical removal of the breast implants and surrounding scar tissue, systemic chemotherapy, often including drugs such as CHOP (cyclophosphamide, doxorubicin, vincristine, and prednisone) and targeted therapy with brentuximab vedotin, an anti-CD30 antibody-drug conjugate

Follow-Up and Clinical Outcomes

The patient was lost to follow up. Typically, patients with BIA-ALCL require close monitoring with regular imaging studies and clinical examinations to assess treatment response and detect any recurrence.

Discussion

BiALCL is a T-cell lymphoma in which malignant T-cells are characterized by large pleomorphic and anaplastic morphology and immunoreactivity for CD30, closely resembling primary cutaneous anaplastic large cell lymphomas (pcALCL). It typically arises in the fibrous capsule and peri-implant seroma surrounding breast implants and has a favorable clinical outcome like nonimplant-associated pcALCL [8].

The estimated incidence of BIA-ALCL varies across studies and geographical regions, with more recent literature suggesting a higher risk among patients with textured implants than previously recognized. The incidence as per literature is 1/3000 cases [9]. Clinically, BIA-ALCL most commonly presents as a delayed peri-implant seroma occurring several years after implantation, usually with breast swelling, pain, capsular contracture, or a peri-capsular mass. Around 20% cases may present with regional lymph nodes as well [10]. The median interval between implant placement and diagnosis is approximately 8–11 years, although longer latency periods have also been documented.

The current case presented as an atypical manifestation of BIA-ALCL, with the primary presentation being a left shoulder mass rather than the more common peri-implant effusion or capsular mass. Such atypical extranodal or soft tissue presentations are uncommon and may delay diagnosis if clinical suspicion is low. The patient's long-standing history of breast implants (approximately 20 years) aligns with the typical latency period observed in BIA-ALCL cases. Although the exact etiology of BIA-ALCL is unknown, chronic antigenic stimulation, biofilm-mediated inflammation, host genetic susceptibility, and dysregulated JAK/STAT signaling pathways have all been implicated in its pathogenesis [11,12].

Molecular studies have demonstrated recurrent activation of the JAK/STAT pathway, including STAT3, JAK1, and JAK3 mutations, supporting the role of persistent immune stimulation in lymphomagenesis [12]. In the current case, histomorphology demonstrating large pleomorphic atypical lymphoid cells along with the immunohistochemical profile, particularly the strong CD30 positivity and ALK negativity, supports the diagnosis of BIA-ALCL [2,4]. Current recommendations emphasize evaluation of all delayed peri-implant seromas with cytological examination and CD30 immunostaining [6]. Appropriate management of BIA-ALCL is the complete surgical removal of the implant and total capsulectomy [3,13].

Conclusion

This case emphasizes the importance of maintaining a high index of suspicion for BIA-ALCL in patients with a history of breast implants, even when presenting with symptoms or masses in atypical locations. It highlights the need for heightened awareness of BIA-ALCL among clinicians and pathologists, particularly in patients with textured breast implants presenting with atypical soft tissue masses or regional symptoms distant from the implant site. Early recognition and timely pathological evaluation are critical, as

complete surgical management in localized disease is associated with favorable outcomes and excellent long-term survival.

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