INTRODUCTION

Breast implant surgery, like any other medical intervention, carries potential risks and complications. One complication that has recently drawn significant attention from plastic surgeons is breast implant-associated anaplastic large cell lymphoma (BIA-ALCL). This rare form of T-cell non-Hodgkin lymphoma is specifically associated with breast implants, especially those with textured surfaces [1]. Typical manifestations of BIA-ALCL include late seroma formation, which can alter the size or shape of the breast, and palpable masses in the breast or axillary regions. The diagnosis of BIA-ALCL is confirmed through immunohistological staining for anaplastic lymphoma kinase (ALK) and the CD30 cell surface protein. Although rare, it is crucial to distinguish BIA-ALCL from other diseases that may arise after breast implant surgery, such as breast parenchymal cancer, squamous cell carcinoma [2], and cutaneous T-cell lymphoma [3]. We present a case of a late breast implant complication that clinically mimicked BIA-ALCL.

CASE REPORT

A 30-year-old woman of Korean ethnicity underwent breast augmentation in 2015 at a local clinic. Prior to the surgery, she had no underlying medical conditions. The procedure involved the use of POLYTECH silicone-type, anatomical, round, textured implants. Throughout the postoperative period, she reported no complications, including any trauma or accidents impacting the breast area. However, approximately 1 month before her clinical visit, she experienced sudden swelling in her left breast. Additionally, 2 weeks prior to this, she noticed changes in the color of the skin on her breast. Upon examination, breast asymmetry was observed, with swelling noted at the inferomedial and inferolateral poles of the left breast (Fig. 1). Additionally, skin abnormalities were present, particularly at the inferomedial pole of the left breast. The affected skin exhibited irregular scaly plaque lesions that were black in color and measured approximately 5 × 5 cm. No palpable axillary lymph nodes were detected. A brief ultrasound assessment conducted at the clinic revealed significant fluid collection. Consequently, a breast magnetic resonance imaging (MRI) was scheduled for further evaluation. MRI revealed a rim-enhancing fluid collection measuring approximately 7 × 5 × 7 cm, associated with focal skin involvement in the inferomedial region of the breast. Another rim-enhancing fluid collection, approximately 6 × 3 × 6 cm in size, was identified at the inferolateral pole (Fig. 2). Additionally, a diffuse
periprosthetic fluid collection was observed, predominantly affecting the base of the implant, but without a definite inner enhancing portion. No signs of intracapsular or extracapsular rupture were detected. After identifying nonspecific fluid collections in the patient's breast, the seroma was aspirated for analysis. Approximately 50 cc of fluid was collected and sent for cytological assessment. However, before the formal report of aspiration cytology was available, a surgical operation to explore and evacuate the fluid collection was decided upon due to the patient's overwhelming discomfort and the increasing size of the fluid collection.

Prior to surgery, the patient stated that she did not intend to consider implant re-insertion in the future. She underwent the removal of bilateral breast implants, and the affected skin was also excised and reconstructed using a rotational local flap. Due to the extensive presence of multiple seromas, en bloc excision was not feasible; instead, capsulectomy was performed separately and with caution. During the dissection, encapsulated seromas were discovered between the silicone implant capsule and the subcutaneous fat. The seroma was drained, and samples were collected for biopsy, cytology, immunohistochemistry, culture, and flow cytometry analysis. A gross examination of the excised capsule revealed pinhole fistulas on both the medial and lateral sides, with one hole on each side. Inside the capsule, dirty, nonspecific necrotic tissue was found. The breast implant was removed, and a total capsulectomy was performed using an electrosurgical device, a curette, and extensive irrigation (Fig. 3).

The right-side breast implant removal and partial capsulectomy were performed through the same incision used for the left-side breast implant removal. No specific findings were observed at the surgical site of the right breast implant removal. The skin defect on the left breast, created by the surgical elliptical incision, was reconstructed using a rotation flap elevated along the ipsilateral inframammary fold.

The excised left capsule was divided into four segments: the left lateral capsule, left superficial capsule, left base capsule, and seroma capsule. A gross examination of the specimen revealed no specific findings. The evaluation was conducted using hematoxylin and eosin staining. Histological sections from the left lateral capsule and seroma capsule displayed fibrous tissue characterized by chronic inflammation and granulation features. The left base capsule and left superficial capsule specimens exhibited focal fibrin depositions and inflammation associated with fibrin material (Figs. 4, 5).

Immunohistochemistry staining revealed no evidence of CD-30 (Ki-1) or ALK. Aspiration and cell block cytology were negative for malignant cells, although they did show numerous leukocytes, lymphocytes, and some monocytes. Tissue cultures taken from the
left capsule exhibited no bacterial growth, and both acid-fast bacilli staining and nontuberculous mycobacterium polymerase chain reaction tests showed no evidence of mycobacteria. The right breast capsule specimen yielded no specific findings. After surgery, the patient received regular follow-up and showed no evidence of recurrence or disease for 6 months. Subjectively, she was satisfied with the contour of her breast postoperatively.

**DISCUSSION**

We report a unique case of fibrinoid accumulation characterized by multiple fluid collections situated between the subcutaneous fat and the capsule of a silicone implant. To our knowledge, this is the first documented instance of fibrinoid accumulation presenting with symptoms similar to those of BIA-ALCL.

Numerous late-stage complications can arise after breast implant surgery, such as capsular contracture, implant rupture, latent infection, and BIA-ALCL. These complications typically develop over time without any apparent external trauma [4]. Previous reports have documented cases of breast swelling that were initially confused with BIA-ALCL, including conditions such as silicone granuloma, breast implant rupture, late seromas, hematoma, infection, or chronic inflammation [5,6].

Our patient’s case closely resembled BIA-ALCL for several reasons. First, the timing of onset: BIA-ALCL usually manifests 8–10 years after breast implant surgery. Second, the presence of multiple fluid collections, both extracapsular and intracapsular, which are observed in 60% to 85% of BIA-ALCL cases [7].

The exact cause of the massive fibrin deposition in this case remains unknown and is difficult to conjecture. The most common cause of focal fibrin deposits is typically inflammation following an infection; however, both Gram staining and culture tests returned negative results. Additionally, the patient denied experiencing any symptoms of systemic infection or inflammation at the time, and her vital signs remained stable throughout the hospitalization period. Other potential causes of focal inflammation that might lead to fibrin deposits include neoplasm or trauma, but neither appeared applicable to this patient.

One possible theory involves the use of fibrin sealants during the initial breast augmentation surgery. Fibrin is crucial in wound healing and tissue repair. After surgical tissue damage, the coagulation cascade is triggered, transforming fibrinogen into fibrin. This fibrin forms a matrix at the surgical site, serving multiple essential functions: it ensures hemostasis, provides a scaffold for wound heal-
ing, and reduces the risk of infection. Biomedical engineers have capitalized on the advantageous properties of fibrin, developing fibrin sealants that have shown wide-ranging applicability [8].

While the normal healing process efficiently degrades the fibrin matrix, excessive accumulation of fibrin has been linked to complications such as fibrosis, thrombosis, tissue adhesions, wound dehiscence, and an increased risk of infection [9]. Our patient's case appears to fit this scenario; however, a thorough investigation is crucial to determine the exact cause of this abnormal fibrinoid accumulation.

Some non-BIA-ALCL periprosthetic fluid collections have been reported, including various malignancies that result in late seromas, such as B-cell malignancies, squamous cell carcinoma, breast parenchymal cancer, and mesenchymal tumors. Other potential causes include synovial metaplasia, capsular epithelialization, and late-stage hematomas. In many cases, a total capsulectomy is either recommended or mandated [10].

The current guideline for BIA-ALCL recommends that when BIA-ALCL is suspected, further assessment should preferably involve breast MRI or ultrasound [7]. If a mass or effusion is present, the physician should conduct a biopsy and/or aspiration cytology, along with immunohistochemistry and flow cytometry, to confirm the diagnosis of BIA-ALCL. Further management should be deferred until pathological confirmation is obtained. However, the patient experienced exceptionally severe discomfort and pain, which led to anxiety and depression. Consequently, a decision was made to expedite surgery unusually quickly. Since this decision deviated from standard guidelines, caution is advised when interpreting this case report. The decision was based on MRI findings that suggested the capsule was not involved, which allowed for a more proactive consideration of surgical intervention.

We present a rare case that could easily be mistaken for BIA-ALCL, which manifested 8 years after breast implant surgery. By documenting what we believe to be the first case of its kind, we aim to raise awareness among plastic surgeons about this potential late complication and emphasize the need for further research to understand the pathophysiology and characteristics of this unusual fibrin accumulation.

NOTES

Conflict of interest
No potential conflict of interest relevant to this article was reported.

Patient consent
The patient provided written informed consent for the publication and the use of images.

ORCID
Heewoong Yang https://orcid.org/0009-0002-6844-6183
Jun Ho Park https://orcid.org/0000-0003-0235-1261

REFERENCES