

Silicone-induced granuloma of breast implant capsule mimicking anaplastic large cell lymphoma

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A 58-year-old female patient underwent an esthetic breast augmentation surgery in 2014 which 5 years later, led to edema, hyperemia and a volume increase of the left breast (Figure 1). Initially diagnosed

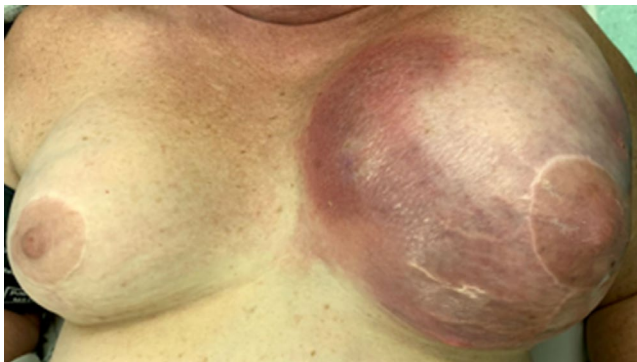


FIGURE 1 Left breast shows inflammatory signs with edema hyperemia and volumetric increase

with mastitis she was prescribed oral nonsteroidal anti-inflammatory drugs and antibiotics for a period of 10 days, and after no improvement, the patient was admitted in hospital for intravenous antibiotics treatment.

The admission ultrasound (US) uncovered a late seroma characterized by an intracapsular heterogeneous collection compressing the breast implant on the left breast (Figure 2).

A magnetic resonance imaging (MRI) was performed for further investigation. The scan showed a late seroma, with a hydro-aerial level and progressive enhancement of the fibrous capsules especially on the left breast, as well as intracapsular masses with late contrast enhancement, "black drop sign" and pericapsular edema. The implants were intact at MRI. No extra capsular collections were observed (Figure 3).

After being submitted to surgical intervention, in order to explantation and capsulectomy an intracapsular collection and a friable fibrous capsule with thickened areas were retrieved and sent to histologic analysis.

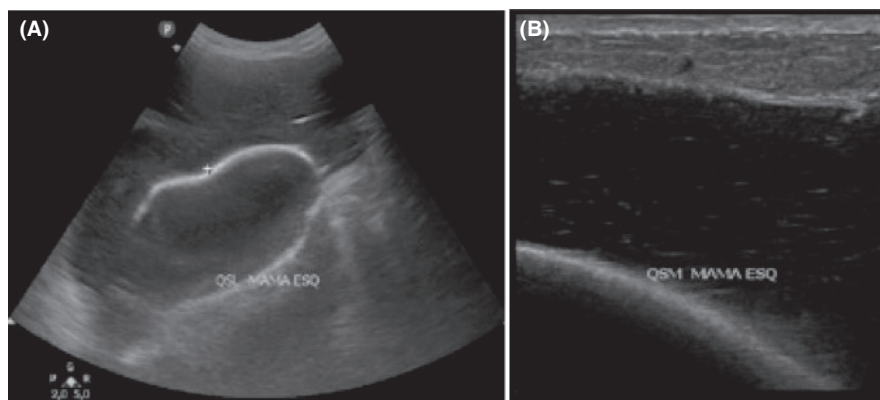


FIGURE 2 Ultrasound convex probe (A) and axial probe (B). Intracapsular heterogeneous collection compresses the breast implant on the left breast (late seroma)

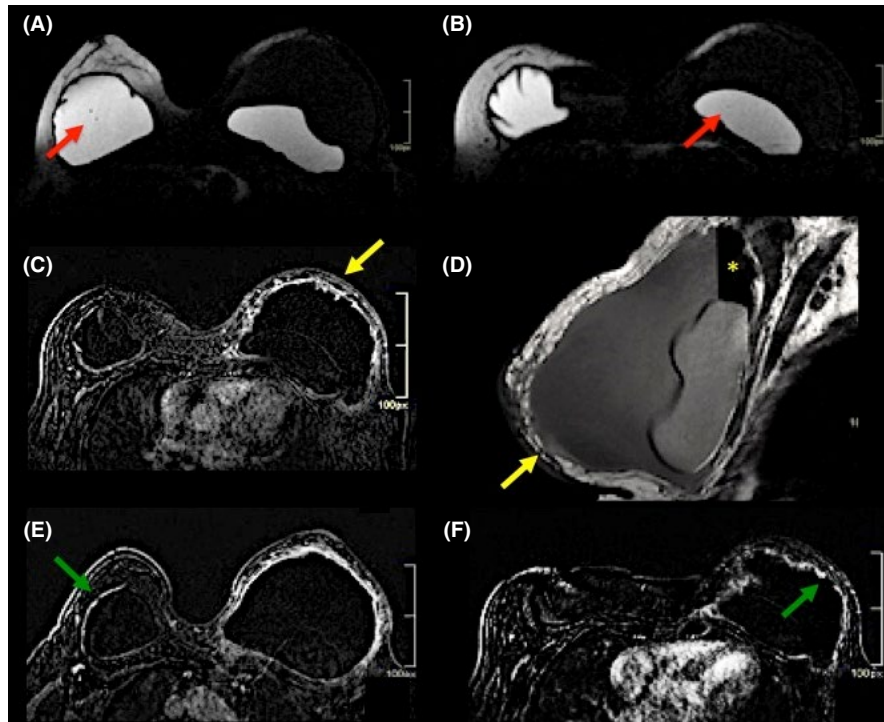


FIGURE 3 Breast magnetic resonance imaging. A and B, Axial T2-weighted faspin-echo water-suppressed: “water droplets” (red arrow) suggesting an alteration in implant permeability. C, Axial dynamic contrast enhancement: the thickening of the fibrous capsule and intracapsular mass both with delayed contrast enhancement, indicating an inflammatory capsular process (yellow arrow). D, Sagittal proton density-weighted: the thickening of the fibrous capsule (yellow arrow) and hydro-aerial level (yellow asterisk). E and F, Axial dynamic contrast enhancement: bilateral “black drop sign” (green arrow) radiologically representing the silicone gel bleeding into the fibrous capsule

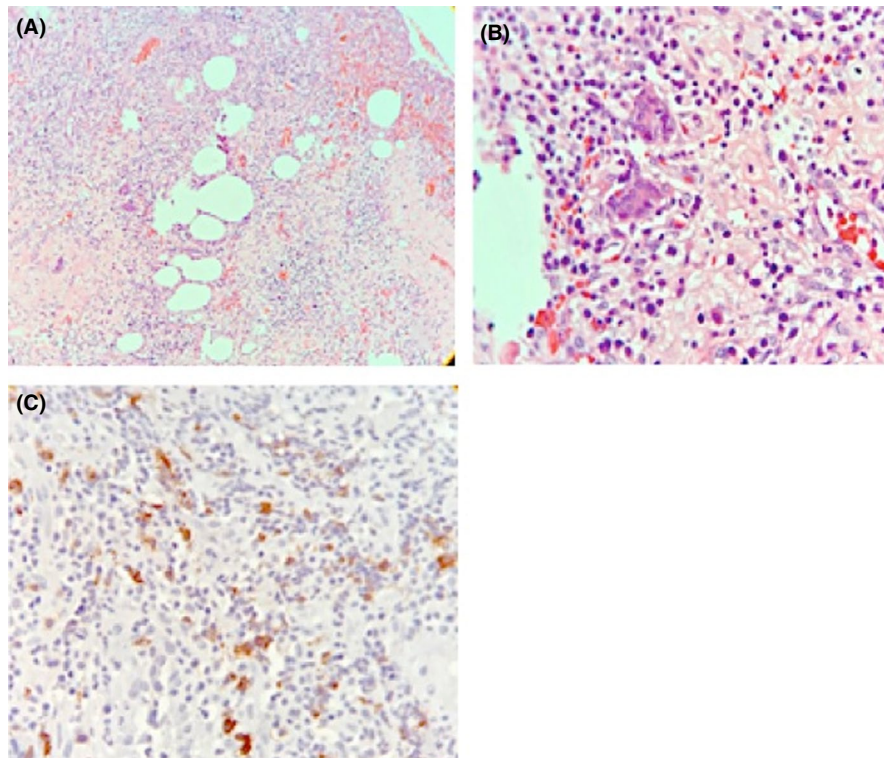


FIGURE 4 Microscopy. The germinal center of B and T lymphocytes is observed by optical microscopy with chronic foreign body inflammatory process type and rare xanthomatous histiocytes (smaller increase (A) and larger increase (B)). The vacuoles observed in A represent negative images of the silicone particles. (C) shows milder positivity for the expression of CD30

Histologic findings showed predominance of intracapsular silicone with an aggregate lymphocyte population in fibrous capsule focal thicket areas. It was associated with a chronic foreign body inflammatory process type and rare xanthomatous histiocytes. In immunohistochemistry, CD30 was mildly observed. No individualized atypical cells are observed (Figure 4).

The number of breast implant-associated anaplastic large cell lymphoma (BIA-ALCL) has risen over the last years. However, some studies have recently questioned the scope of this condition and especially its etiopathogenesis.

Some authors have proposed the silicone-induced granuloma of breast implant capsule (SIGBIC) as a differential diagnosis. This complication is defined by an immune-mediated response caused by leakage of silicone particles from an intact silicone implant, secondary to the elastomers' frailty (gel bleeding).

The leakage particle, when in contact with fibrous capsule, triggers a variable chronic inflammatory/immune response, which induces macrophage activation, cytokine production and T cell recruitment. This response, accounts for the similarities in clinical-radiologic presentation between SIGBIC and BIA-ALCL, suggesting that they could be different spectra of the same disease, or perhaps, distinct pathologies with similar trigger.

Clinically, both conditions usually present with an increase in breast volume and inflammatory syndrome, as well as a late seroma image with capsular enhancement on magnetic studies. From a histologic standpoint, BIA-ALCL exhibits an abnormal proliferation of T cells which are strongly positive for CD30, while

for SIGBIC, this positivity will depend on the magnitude of the immune response as well as the stage in which the diagnosis is made.

We believe that the underdiagnosis of BIA-ALCL in Brazil is mainly justified by the late diagnosis partly due to the differential diagnosis with other pathologies—in particular SIGBIC—and partly as a result of the use of antibiotics and nonsteroidal anti-inflammatory drugs which cool the inflammatory process and implicate in a milder positivity for the expression of CD30.

Therefore, although the number of breast implant-associated anaplastic large cell lymphoma has escalated over the last years, its incidence is still much inferior to the prevalence of the clinical-radiologic findings which suggests its occurrence. On the other hand we observed the presence of SIGBIC in about 30% of the patients submitted to the MRI.

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