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A Forgotten Entity following Breast Implant Contracture: Does Baker Need a Change?

Andrea Pagani, MD[1] Matthias M. Aitzetmüller, MD PhD[2,3] Lorenz Larcher, MD[4]

[1] Clinic and Policlinic of Plastic, Reconstructive and Hand Surgery, Klinikum Rechts der Isar–Technische Universität München, München, Germany
[2] Section of Plastic Surgery, Department for Traumatology and Hand Surgery, University Hospital Münster, Münster, Germany
[3] Plastic, Reconstructive, and Aesthetic Surgery, Hand Surgery, Fachklinik Hornheide, Münster, Germany
[4] Plastic and Reconstructive Surgery Unit, SABES (South Tyrolean Health Care System), Bressanone, Italy

Address for correspondence Lorenz Larcher, MD, Unit of Plastic Surgery, SABES (South Tyrolean Health Care System), Bressanone, Italy (e-mail: dr.lorenz.larcher@gmail.com).

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Abstract

Although capsular contracture represents one of the most important complications after breast augmentation, local inflammation and fibrosis can lead, to capsular calcification, an often-forgotten radiological sign of capsular contracture. In this article, the authors present a clinical case of breast implant calcification in an 81-year-old patient. Although this complication has been rarely described, the literature was reviewed to clarify the role of the local microenvironment in capsular contracture and calcification. At present, capsular contracture patients are classified using the conventional Baker score and the histological Wilflingseder classification. As it was not possible to consider capsular calcification when classifying our patient using the traditional scores, the authors propose an updated version of the current scale.

Keywords
- breast implants
- capsular calcification
- Baker score
- breast surgery
- implant surface

Breast augmentation represents one of the most common operative procedures in the field of plastic and reconstructive surgery. However, it is associated with both minor and major complications such as hematoma, seroma, implant rupture, dislocation, or deformation, double capsule, and capsular contracture (CC), which affect 2.8 to 20.4% of breast implant patients.[1] Although CC represents the most important complication after breast augmentation, the most severe postoperative risk is the rare condition of breast implant-associated anaplastic large cell lymphoma (ALCL), a cancer of the immune system that can lead to implant loss. Local inflammation and fibrosis around the implant are followed by loss of cellular activity, shrinkage, and necrosis.[2,3] In rare cases, CC is the ultimate step of this process.[4]

Herein, we present the case of a female patient with asymptomatic capsular calcification, a recognized but sometimes forgotten occurrence after breast augmentation. The patient had received smooth, round implants over 40 years ago. Due to the increased frequency of additive mastoplasty (more than 300,000 operations, with a 3% increase in 2017) and global population aging, there might follow a significant increase in patients reporting capsular calcification within the near future. The still controversial and unsolved debate about textured breast implants and their hypothesized correlation with ALCL will further increase implantations of smooth prostheses within the next years. Classical smooth prostheses could cause a long-term increase in the incidence of CC, hardening, and pain. As a result, an increase in

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periprosthetic calcifications can be expected. Valid laboratory and clinical data are urgently needed.

The Etiopathogenesis of Capsular Contracture

Capsular contracture is initially characterized by the proliferation and deposition of monocytes, macrophages, T-CD4$^+$ lymphocytes, collagen fibers, and fibroblasts in the "contact zone" between the implant surface and surrounding tissues. This leads to the formation of a capsule and over time to painful capsule thickening.[4] Local collagen fibers and fibroblasts act as the main protagonists of this process[5,6]: While the collagen fibers form a characteristic helical orientation, the fibroblast-to-myofibroblast conversion leads to the proliferative phase of contracture progression.[7]

In addition to the cellular composition, the molecular, immunological, and cytokine (e.g., interleukins [ILs], renin, histamine, tumor necrosis factors [TNFs], and transforming growth factor-β 1) composition of the implant capsule impacts CC. Whereas TNFα and collagen type IIIa (COL3A1) are associated with inflammation,[5] local mast cell degranulation activates neighboring cells and leads to proliferation and migration.[8] The matrix metalloproteinases (MMP-1 and -2) and their endogenous tissue inhibitors (TIMP-1 and -2) limit the fibrotic process by degrading the extracellular matrix.[3] Other immunological factors such as IL-4, IL-6, IL-10, IL-13, and IL-21 support cell recruitment, inflammation, and fibrosis.[9] Recent papers[2] have shown that breast implant texture and the adjacent cellular microenvironment affect each other.

In 2009, after evaluating different implant shells (e.g., Mentor Siltex, Allergan Biocell, Allergan Smooth, Cereplas Cereform, and Polytech MicroPolyurethane), Barr et al[2] reported mechanisms through which local fibroblasts interact with the nanostructure of breast implants to play a pivotal role in CC. Current smooth-surface implants are made by inserting a mandrel in liquid silicone. The surface then undergoes a drying and hardening process in a laminar flow oven. According to Sitbon,[10] as the implant begins to dry, the gradual creep of silicone down the side of the mandrel produces particular ripples on the implant surface. This procedure avoids the introduction of weaknesses in the implant shell, producing a safe medical device.

Until the best manufacturing technology is discovered and as breast augmentation becomes more common, it is crucial to obtain objective knowledge about the intrinsic characteristics of currently available breast implants to improve the safety of devices and patient satisfaction.

Case Presentation and Methods

We present the clinical investigation of a Caucasian female patient who presented to our plastic surgery unit reporting symptomatic breast deformation. The requirements of the Declaration of Helsinki as well as the principles of good clinical practice were taken into consideration. The patient gave full consent to the use of her personal data. A smooth, rounded, silicone breast implant was placed bilaterally under the mammary gland more than 40 years ago for breast augmentation after pregnancy.[7]

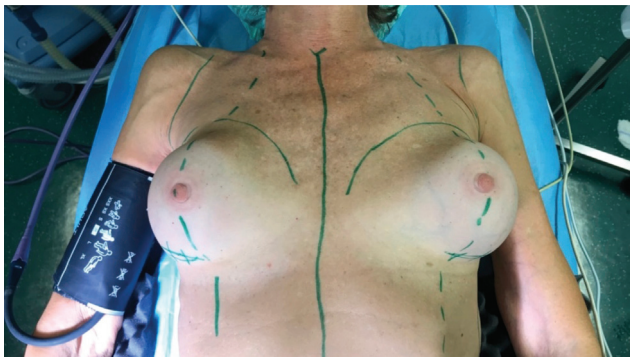

Fig. 1 The patients' preoperative clinical presentation. The patient reported firm, painful, and mineralized breast implants with deformation, consistent with the clinical diagnosis of capsular contracture.

Imaging Investigations and Histology

The first clinical examination showed significant deformation of the projection of the breast and a concomitant displacement of the implant, suggesting dislocation and rupture of the breast implant (►**Fig. 1**).

Ultrasonography (Toshiba Aplio XG) using a linear ultrasound probe (PLT-805AT) was performed subsequently and revealed signs of intracapsular rupture, deformity, an increased number of radial folds, and high thickness of the fibrotic capsule.[11] Additionally, magnetic resonance imaging (MRI) (Philips Intera 1.5T) showed the presence of a bulge of the prosthesis in the right cranial side in comparison with other MRI exams performed before 2017. No definite prosthesis rupture was detectable and no periprosthetic fluid accumulation or enhanced contrast agent uptake was reported. However, in the area of the medial border of the right breast gland, a slightly irregular tissue compaction (7×3 mm) was detected. No suspicious lesions and no pathologically enlarged lymph nodes were observed.

A core needle biopsy was performed. We harvested the mineralized samples, showing multilamellar crystal fibrous calcium deposits with true bone formation and osteocyte lacunae. On chest radiography, the breast implants presented with heterogeneous hyperechogenicity on the surface of the prosthesis, suggesting the occurrence of capsular calcification (►**Fig. 2**).

Clinical and Operative Inspection

Considered the high degree of capsular calcification, we surgically removed the breast implants using the previous incision. Upon removing the breast implants, we evaluated contracture and the implant surface. The capsule was characterized by massive calcification that had led to destruction of the sample (►**Figs. 3** and **4**). However, gel was leaking in several locations. A thick and stick capsule adhered to the anterior surface of the breast implant. Hydroxyapatite crystals had accumulated in the area of close contact between the implant and the surrounding tissue. After removing the implant, we performed capsulectomy to remove foreign

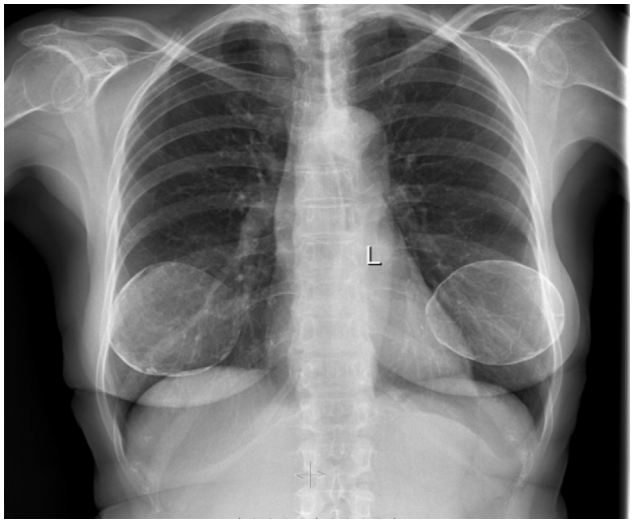

Fig. 2 Radiography showing implant calcification. A radiological image reveals mineralization of both breast implants.

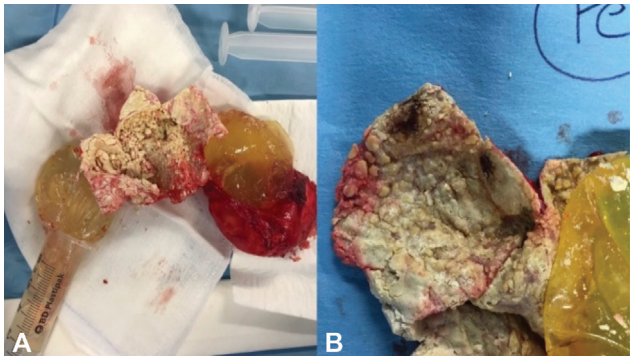

Fig. 3 (**A**, **B**) Calcification of the implants. After removing the breast implants, authors evaluated the rupture and the calcification of the implants.

particles in situ. The implant and capsulectomy material were evaluated macroscopically to analyze the mechanical properties of mineralization and the structural shell of the implant surface.

Discussion

CC is classified using the conventional Baker score and the histological Wilflingseder classification. This clinical history presents one of the many other cases in which an asymptomatic patient presented with a high-grade Baker score of CC. In the present case and in many other literature-reported cases, there was marked heterogeneity between the objective contracture severity and the patient's subjective signs and symptoms. It is not infrequent to find symptomatic women with mild Baker grade I contracture or asymptomatic patients, like our patient, with severe CC. Therefore, a reliable CC classification using both the Baker and Wilflingseder scores was not possible.

In 1977, Redfern et al[12] initially described breast implant calcification and the mechanism of cellular deposition as a common reaction affecting from 16 to 25% of removed devices. Within the following years, several journals published articles about this topic, leading to an attempted Food and Drug Administration (FDA) ban of first- and second-generation smooth saline-filled breast implants with Dacron or polyurethane particles in the United States. Ultimately, despite various pressures, the FDA failed to ban implants with polyurethane particles.

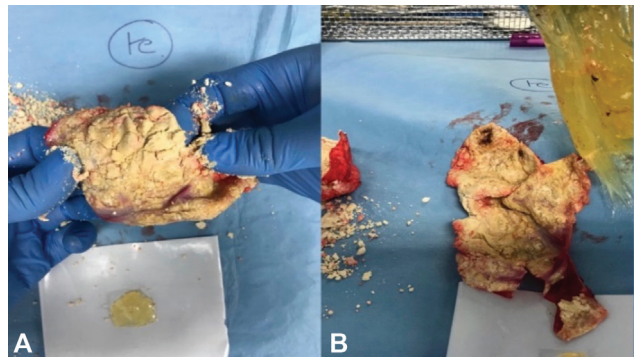

Fig. 4 (**A**, **B**) Porosity and calcification of the implants. During the surgical procedure, the porosity and consistency of the massive calcifications did not allow the authors to remove the implants without damaging the sample.

The strong disagreement and the heated debates among medical specialists in regards to the FDA decision allowed the following introduction of third-generation (1982), fourth-generation (1987), and fifth-generation implants with cohesive gels in 1992.[13] Their triple-layer shell with highly cross-linked silicone made capsular calcification a rare finding. The calcification of silicone-gel breast implant capsules occurs as small calcified plaques at the interface between the inner fibrous capsule and implant surface.[4] Capsular calcification has also been encountered with breast implants in patients with silicone envelopes filled with saline.[14] Calcification could interfere with effective tumor detection and diagnosis, which could potentially delay treatment, particularly in patients who have breast implants following reconstructive surgery for breast cancer. Peters and Smith histologically demonstrated a calcification rate of 16% in patients with CC.[15] Legrand et al investigated 18 breast implants that were explanted after more than 20 years and demonstrated calcification in all samples.[16]

The difference between the rate of postoperative complications of saline and silicone gel implants is broadly known. The latest-generation silicone devices are not comparable with the older traditional devices; the qualitative improvement of the surface, the better shell, and the innovative filling materials have transformed breast implants into safe medical devices. The modifications have made it possible to improve patients' quality of life and simultaneously reduce postoperative complications. However, CC is still reported in the current literature and could also continue to occur in the next years with new-generation prostheses.

In recent years, breast implant surface technology has emerged as one of the main topics in the field of tissue engineering. Nanotechnology is a focus of development,[17] with the aim of reaching the highest level of manipulation of individual atoms and molecules to create the finest-quality

medical devices. Regarding breast surgery, the adjacent soft tissue response is regulated by the cellular response to the texture of implants' surfaces. Textured products exhibit larger surface area, allowing the ingrowth of tissues in the proximity of the breast capsule. Research is focusing on the effects of interactions between different nanosurfaces and surrounding tissues on cell adhesion, proliferation, and synthesis.[18]

According to Fradinho et al,[19] Raso et al,[20] and Legrand et al,[16] the body responds to breast implants by producing inflammatory cells, fibroblasts, and collagen fibers. The conversion of local fibroblasts in active contractile myofibroblasts generates traction forces leading to contracture. The persistence of inflammation and fibrosis is followed by a loss of cellular activity, shrinkage, necrosis, and multilamellar deposition of hydroxyapatite crystals around the implants. Other cells, such as histocytes, macrophages, and foreign body giant cells, support the processes leading to chondral metaplasia and hyalinization after a median time of 11.7 years and dystrophic calcification after 11 to 22 years. Legrand et al conducted a thorough analysis of 18 explanted breast prostheses after 20 or more years of implantation and concluded that the continuous remodeling of bone-like hydroxyapatite minerals and their local proliferation depends upon the size and shape of implants.

After explanting saline-filled breast implants that had been inserted in the subglandular plane to three young patients (32, 34, and 44 years old), Peters et al[4] microscopically observed ivory-colored deposits on the anterior surfaces of the implants 7 to 23 years after initial surgery. Ultrastructural analyses showed large, electron-dense spherulitic aggregates of needle-shaped crystals ($40 \times 10 \times 10$ nm) and metaplastic bone areas. Some of the implants showed a massive aggregation of globular particles of calcium, while other implants presented defined bone formation. The exponential growth of young women who are undergoing additive mastoplasty and the significant increase in the average age of the population in the coming years, will expose an increasing number of people to this complication. As with many other clinical conditions, this age-related epidemiological trend is highly dependent on the individual genetic predisposition to inflammation and degenerative processes.

In view of all these considerations, the type of filling (saline vs. silicone), implant generation (first, second, and third), duration of the implant in situ, implant integrity, and age of patients are the most significant factors related to CC and capsular calcification. Despite the great improvements in surface, shape, and material features, CC remains a significant clinical problem. The ultimate goal of tissue engineering and manufacturing in breast implant development is to produce a tailor-made device for every single patient to support local physical and biological processes and eventually improve patients' quality of life.

Our Proposed Upgrade of the Traditional Classification Systems

The two current classification systems of CC are the histological Wilflingseder classification and the Baker score. While the Wilflingseder stages (I–IV) are related to the thickness of the capsule and to the presence of fibrosis, giant cells, inflammatory cells, foreign body granulomas, neovascularization, and neuromas,[21] the Baker scale (I–IV) is widely recognized and used to evaluate clinically the degree of contracture[22] (►**Table 1**). A modification was developed by Spear and Baker that included IA and IB, dependent on the breast reconstruction method and on the physical examination.

Although the Baker system is widely used, there is marked heterogeneity between the objective contracture severity and patients' subjective symptoms. It is not infrequent to find symptomatic women with mild Baker grade I contracture or asymptomatic patients with severe CC. In addition, in some cases like the one presented herein, it is not possible to classify CC using the traditional Baker classification. Through a deeper analysis of the history and the etiopathogenesis of capsular calcification and considering the heterogeneity of patients' clinical signs and symptoms, the authors believe that the traditional classification system should be modified accordingly, taking into account further radiological aspects as well as patient-dependent factors. From our point of view, an additional descriptor of "(A)" (without symptoms) and "(B)" (with symptoms) should be added to all stages, both for classification purposes and to potentially match patients' objective data to clinical and histological parameters. The addition of a grade V within the classification will allow surgeons to report any neoformations present within the capsular microenvironment and better predict patients' outcomes (►**Table 2**).

To summarize, considering the increasing frequency of additive mastoplasty, population aging, and the increase in frequency of usage of smooth implants, capsular calcification will affect a small but significant number of patients in the future.

Table 1 The traditional Baker classification (I–IV)

Grade	Score
I	Breast and implant shell are soft and not palpable, breast appears natural in size and shape
II	Breast and implant shell are slightly firm, breast appears normal
III	Breast and implant shell are clearly firm, implant is visible, breast appears abnormal
IV	Breast and implant shell are firm, implant dislocation/deformation, breast is painful to the touch and appears abnormal

Note: In the first stage, the breasts appear natural in size and shape, while in stage IV, patients report a firm, dislocated, and painful breast.

Table 2 The upgraded Baker classification

	Score	Symptoms	
Grade		YES [A]	NO [B]
I	Breast and implant shell are soft and not palpable, breast appears natural in size and shape		
II	Breast and implant shell are slightly firm, breast appears normal		
III	Breast and implant shell are clearly firm, implant is visible, breast appears abnormal		
IV	Breast and implant shell are firm, implant dislocation/deformation, breast appears abnormal		
V	Breast and implant shell are firm, implant dislocation/deformation, breast appears abnormal, intra-operative detection of capsular calcification		

Note: The introduction of a "symptomatic" classification and the concomitant addition of stage V within the Baker score could allow surgeons to better report the clinical symptoms of breast implant patients.

Based on an analysis of the current histological and clinical classification systems for CC by Wilflingseder and Baker, we additionally provide a "symptomatic" modification of the traditional stages. The introduction of a symptomatic classification with the addition of grade V within the Baker classification will allow surgeons to better report the clinical presentation of patients with CC.

Altogether, we think this article sheds light on capsular calcification, an almost forgotten entity, and might drive future research to improve implants' safety and durability.

Author Contributions

All authors contributed to the study conception, design, and manuscript writing.

Patient Consent

The patients provided written informed consent for the publication and the use of their images.

Conflict of Interest

None declared.

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