Soft Textured Implants: Roughness, Friction, and the Complications

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A B S T R A C T

Friction and shear between soft medical implants and the contacting cells and tissues can lead to the production and secretion of pro-inflammatory proteins and small molecules: cytokines, chemokines, and damage-associated molecular patterns (DAMPs). Biotribological considerations should be included in biocompatibility studies and the design of soft medical implants. Clinical experience with textured breast implants has revealed a number of complications linked to chronic inflammation. Long-term exposure to elevated levels of pro-inflammatory cytokines, proteins, and DAMPs may lead to chronic inflammation, which can have numerous negative biological consequences: pain, swelling, seromas, DNA damage, and even cancer. Contact pressure, surface roughness, surface elastic modulus, and friction all contribute to the dynamics and shear stresses reacted across these interfaces during use; in many soft implant applications, controlled mobility (without migration) is desirable, and slip is essentially unavoidable. Depending on the magnitude of shear stress, cells will undergo a wide range of responses from proliferation and remodeling, to cytokine and chemokine production, and even cell death (apoptosis or necrosis). In soft medical implants, friction coefficients and shear stresses increase with increasing roughness. Models of breast implant contact pressure as a function of geometry and relative size reveal that the shear stresses of the aggressively textured implants may exceed 100 Pa, while the smooth implants are likely well below 100 Pa. At shear stresses of around 100 Pa, epithelial cells have been shown to produce pro-inflammatory cytokines and undergo programmed cell death (apoptosis). Increasing roughness and texturing of soft medical implants should be considered a risk factor for implant biocompatibility, particularly for larger, high-projection designs.

1. Introduction

Medical implants made from silicone have been the standard in soft elastic implants for over 3 decades. In addition to silicone coatings for implantable medical devices, there are also numerous soft elastic implants made from silicone: ports, pumps, drains, shunts, cochlear implants, tracheal prostheses, catheters, gastric bands, tubes, and a wide range of prostheses and cosmetic implants [1,2]. Today, the majority of all breast implants in the world are made of silicone. Silicone surfaces are largely considered biocompatible [3,4], biostable, and chemically inert; silicone also has a number of practical manufacturing benefits for medical devices including a long shelf life, high temperature stability, ease of sterilization, and generally good resistance to contamination (bacterial and organics). Surface texturing of silicone can be performed through a number of processing and molding routes, and some breast implant models have been intentionally and aggressively roughened (Fig. 1). Texturing has been widely used in manufacturing to mask seams and blend discontinuities [5], which may have been an under-lying motivation with larger implants. Of the various medical reasons for surface texturing, the most frequent arguments are around fixation and positioning. A stable implant is highly desirable as migration can lead to asymmetric errors and anatomical distortions. The hypothesis was that the textured implants would increase friction, help the implant “stick” to the body, and prevent unintended migration of the implant [6]. There was some anecdotal historical evidence that polyurethane textures on the surface of breast implants reduced complications and this may have encouraged the aggressive texturing of recent breast implant designs [7].

During the healing process after implantation, soft medical implants made from non-biologic materials often induce the development of a capsule that walls off the implant from the rest of the body. These capsules are comprised of fibrotic tissues, and under some conditions will contract or “squeeze” an implant, causing deformation, capsular contracture [9], and in extreme cases, even implant rupture. For breast

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implants, capsular contracture can be a serious and painful complication. Recent studies have shown that the aggressively roughened implants form thicker capsules than smooth implants [11]. An excessive fibrotic reaction to the roughened implant can lead to fibrosis and excessive production of collagen. The recruitment of fibroblasts and the formation of the capsule is part of a wound healing process and is regulated through a complex sequence of secreted soluble proteins including cytokines, chemokines, and growth factors. Recent work with epithelial cells has found that frictional shear stresses as low as 60 Pa are capable of cellular upregulation and production of pro-inflammatory cytokines [12]; implants with increased friction coefficients may produce and maintain a chronic inflammatory state. Chronic inflammation can be the basic driver for a number of complications illustrated in Fig. 2: swelling, seromas and hematoma, capsular contracture, and double capsules. There is growing evidence that aggressively textured silicone surfaces are involved in a number of complications that are specific to such implants, including: double encapsulation [13] (Fig. 2d), seromas (or effusions [14]) and hematoma [15] (Fig. 2b), and cancer (Breast Implant-Associated Large-Cell Lymphoma, BIA-ALCL) [16–19] (Fig. 2b).

During the development of roughened implants, a guiding hypothesis was that the fibrotic capsule would integrate with the aggressive surface roughness and lead to an interlocking of the tissue to the implant. Here the roughness, described as aggressive, was intentionally an order-of-magnitude larger than the size of a cell, so the interlocking of the implant to the tissue was intended to be through a mechanical engagement at a collective- or tissue-level, not a biomolecular surface adhesion mechanism at a cellular or subcellular level. This macroscopic interlocking was thought to help eliminate movement, slumping, and positional changes of the implant over time, and thereby maintain shape, projection, and symmetry. However, a number of problems associated with these textured implants have emerged in recent years, primarily involving the peri-implant interfaces as illustrated in Fig. 2.

Textured implants have led to a number of complications that are more prevalent and/or unique in comparison to smooth implants. Seromas (Fig. 2b) are pockets of inflammatory fluid, blood plasma, and secreted proteins from injured, stressed, or dying cells. Seromas should be transudates, with low cell counts and low protein content, whereas peri-implant effusions of BIA-ALCL patients contain liquefied and necrotic lymphoma cells with a high protein content [19]. Hematomas (Fig. 2b) are primarily caused from blood leaking into a pocket due to injury. Chronic seromas, effusions, and hematomas have a profoundly higher incidence in textured implants and may be the result of persistent inflammation and ongoing injury, respectively. Anaplastic Large-Cell Lymphoma has been reported in the literature as a disease that is associated with textured breast implants (BIA-ALCL) (Fig. 2b) [16,18,20–22], as well as other prostheses [23–26]. Epidemiological studies of BIA-ALCL suggest that this cancer is rare and can be cured following implant removal, but all cases with adequate history have involved a textured breast implant [17,27]. Capsular contracture (Fig. 2c) was not eliminated by the textured implants [28], and the occurrence of a double capsule (Fig. 2d) is unique to textured implants; double capsule formation may be the result of a shear accommodation strategy during capsule formation that allows smooth sliding across a biological interface.

Epidemiological studies consistently correlate BIA-ALCL with aggressively textured implants [29]. BIA-ALCL is considered to be a new, distinct entity, with a multifactorial inflammatory cause [30], and the association with texturing has led to the assertion that the chronic inflammatory cause is both site- and material-specific. Numerous efforts are underway to quantitatively characterize texture by various metrics (surface area, peak heights, feature spacing and size, etc.) and look for correlations between incidence and roughness parameters. In this manuscript we suggest that frictional shear may be increased through the aggressive texturing, and that at elevated levels of shear stress, cells produce and release proinflammatory cytokines that generate a chronic inflammatory state in the tissue around the implant. The texture and the materials may be biocompatible in the absence of sliding, but recent work with epithelial cells has clearly demonstrated the role of shear stress in inflammation [12] and programmed cell death [31]. The rarity of BIA-ALCL has also led researchers to hypothesize that a possible genetic predisposition may also be involved.

2. Modeling and Discussion

An emerging hypothesis in the medical literature is that these implants are inflammatory through a mechanism of shear, which may or may not include physical motion and sliding of the implant [32]. Smooth breast implants are known to form a thin capsule around the implant, which provides natural motion within a constrained compartment. The capsule is a type of scar-like fibrotic tissue that forms a membrane-like pocket around the implant. For smooth implants, there is little to no physical interlocking between the implant and the capsule; the implant is essentially free to translate within the capsule as dynamics and activity dictate. In this case, the implant motion and changes in the capsule geometry may lead to the implant changing position or “slumping” within the breast over time. To a first-order approximation, the implant can be modeled as a distorted hemisphere, with an approximate contact pressure, \( P \), given by Eq. (1), which balances the body force of the implant (\( \rho \), implant density; \( g \), acceleration due to gravity; \( V \), implant volume) across the inferior implant surface (\( R_w \), implant width; \( r \), implant projection as illustrated in Fig. 3).
It is interesting that the simplified expression for contact pressure does not include the projection, \( r \). The contribution to increased volume with increased projection is cancelled by a corresponding increase of the inferior surface; both mass and area scale to the same proportion with increasing projection and the result is that contact pressure has no dependence on projection. Although there is a wide range of implant designs, the results are that most implants have contact pressures on the order of 300–500 Pa. Assuming a friction coefficient of \( \mu = 0.10 \), the smooth implants probably experience shear stresses, \( \tau \), on the order of 30–50 Pa (Eq. (2)).

\[
P = \frac{\rho g V}{2 R_o r} = \frac{\pi \rho g R_o}{4}
\]

(1)

\[
\tau = \mu \frac{\pi \rho g R_o}{4}
\]

(2)

Recently, in research with contact lenses and epithelial cells it has become clear that shear and sliding can produce pro-inflammatory cytokines under physiological conditions at frictional shear stresses even below 100 Pa [12]. Based on experience with epithelial cells, these shear stresses for smooth implants are within the physiologically-tolerated levels and are just below the transition point for friction-induced inflammation [12] and friction-induced apoptosis [31]. This simple analysis suggests that the smooth implants should be reasonably well-tolerated from a biotribology perspective, although capsular contracture has been found in both smooth and roughened implants [33].

Biomechanically, there is a significant mismatch between the surface elastic modulus of the implants and the surrounding breast tissue. This is not surprising, as one of the functions of breast implants is to increase the effective breast modulus and maintain projection through the addition of a higher modulus material. However, as the volume fraction of the implant increases, the mechanics and dynamics are biased increasingly towards the properties of the implant. Assuming complete integration of a capsule occurred across an implant (as promoted for the textured implants), shear stresses are dynamically redistributed during movement. One mechanism of support is across the posterior of the implant (Figs. 3, 4, and Eq. (3)). During posterior support, increasing projection, \( r \), is a driver for increasing the shear stress during activity. Eq. (3) is derived as a best-case scenario where the mass of the implant is carried in shear across the largest possible contact area.

\[
\tau = \frac{\rho g r^*}{2}
\]

(3)

Due to the non-spherical shapes of the breast, every radial dimension from the center of the implant has a unique fractional length

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Fig. 2. Illustrations of painful complications with textured implants that may necessitate surgical revisions: a) swelling and asymmetry; b) seromas, effusions, hematomas, and lymphoma (BIA-ALCL); c) capsular contracture; and d) double capsule formation.

Fig. 3. Nomenclature for the breast implant designs: \( R_o \), width (top left) or height (bottom left); \( r \), projection; \( r' \), curve. Depending on the implantation method, various contact pressures are expected: (1) \( P_m \), submuscular implantation placement will have contact pressures due to pectoralis major muscle contractions; (2) \( P_b \), posterior pressures of the posterior implant surface; and (3) \( P \), body force pressure supporting the gravitational load of the implant along the base of the implant.
between the implant and tissue as shown in Fig. 4. A dimensionless radial fraction $R^*$ is given by the ratio of length of the implant, $R$, to a characteristic length of the breast surface, $C$, at the same radial co-ordinate $(\theta, \phi)$. Given that significant displacements are often expected and desirable, it is naive to expect that such movement can maintain a no-slip condition at the tissue implant interface. In fact, relatively simplistic analysis reveals that the increase in shear stress, $\tau$, for any finite deformation is proportional to $1/(1-R^*)$.

Force balance reveals that as the implant occupies a greater fraction of the breast volume, it challenges the system tribologically in two significant ways: by (1) increasing contact pressure and (2) increasing slip at the interface. Friction coefficients have been estimated to be on the order of $\mu = 0.25$ for the aggressively textured implants [20], and this is likely due to large viscoelastic dissipations during sliding across tissues as opposed to the low friction frequently observed for smooth sliding against a surface comprised of soft aqueous extracellular matrix.

Our simple model (Fig. 5a) shows that shear stresses increase during sliding along the inferior surface with increasing friction coefficient; as such, friction coefficient is a risk factor for damage and inflammation. Regardless of the assumed load support mechanism, shear stresses at the implant-tissue interface are expected to exceed 100 Pa. For a stationary implant under posterior support (neglecting additional forces from muscle contractions) Fig. 5b shows that shear stresses span from 100 to 300 Pa over a range of projection, $r$, from 20 to 60 mm.

Given predicted shear stresses of hundreds of Pascals, the cellular responses may involve apoptosis, inflammation, and even cell necrosis [12,31] – cell death mechanisms which are generally pro-inflammatory and painful [34,35]. Elevated shear between soft medical implants and the surrounding cells and tissues can lead to the production and release of pro-inflammatory proteins and small molecules: cytokines, chemokines, and damage-associated molecular patterns (DAMPs). Under continuous production of pro-inflammatory cytokines, the local...
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The surface roughness and texture of medical devices can significantly impact patient outcomes. The present review aims to explore the biotribology of textured breast implants, with a focus on the mechanisms that lead to complications.

3. Closing Remarks

Biotribological analyses of contact pressure, shear stress, and motion between the surfaces of medical devices and the surrounding tissues reveal that shear stresses can be above 100 Pa for textured breast implants. Shear stresses above physiological levels may induce a chronic inflammatory response that can lead to complications such as capsule formation, fibrosis, and immune response.

Declarations of Competing Interest

The authors have no conflicts of interest regarding this manuscript on the biotribology of textured breast implants.

References


