REVIEW

Breast augmentation: Part II – adverse capsular contracture

M.G. Berry*, V. Cucchiara, D.M. Davies

Institute of Cosmetic and Reconstructive Surgery, 1 Parkside, London W6 OUU, UK

Received 17 November 2009; accepted 11 April 2010

KEYWORDS
Breast augmentation; Adverse capsular contracture; ACC; Mammary prosthesis

Summary Although adverse capsular contracture (ACC) following breast augmentation remains an enigmatic phenomenon, significant progress has been made in diminishing its occurrence during the previous surgical generation. Given the rising global frequency of breast augmentation, however, ACC is likely to be with us for the foreseeable future and an understanding of its nature, and particularly prevention, will continue to be of foremost importance as breast augmentation undergoes a paradigm shift from anti-contracture to aesthetic result as the key outcome measure. Whilst clinical research has hitherto been the mainstay of investigation, providing both understanding and practical guidance, further improvements may derive from new developments in the fields of immunology and molecular biology: convergence of these complementary avenues may eventually yield a non-surgical treatment for ACC. This review presents a summary of our extant knowledge, providing evidence where it exists and a consensus view where it does not. It aims at providing a sound comprehension of the underlying aetiopathology that has provoked the measures seen to date and guides selection of the appropriate therapeutic strategy, which will be expanded in a future review.

The 1950s saw both the first report of potential clinical uses for silicone in Plastic Surgery1 and direct experience of implanted ‘pervious silicone’ sponges in canine models.2 In the latter, neither infection nor extrusion were observed, all implants retained their consistency and became encapsulated. This was the first observation of encapsulation, a characteristic of silicone thought to distinguish it from other contemporaneous biomaterials.

We now know that, despite being relatively inert, breast prostheses are no different from any foreign material implanted into the human body in their provocation of a protective immune reaction from the host. This ‘foreign body response’ (FBR) is universal and ideally removes, or failing that circumscribes, the ‘irritant’ material with fibrous tissue to prevent unwanted immune sequelae.3 A capsule is therefore the ‘necessary and universal
Ordinarily beneficial, problems ensue when such capsules become pathologically active and undergo a ‘constrictive fibrosis’ that deforms their contents and impairs the aesthetic outcome (Figure 1).

Although the capsule was initially described as a bilayer, with an inner cellular layer adjacent to the implant and an outer collagenous layer, a tri-laminar structure is now accepted with an intervening lamina comprising loose connective tissue and a rich cellular presence (Figure 2). Despite detailed clarification of the cellular and molecular components of the capsule, a clear correlation between either capsule thickness (Table 1) or the presence and composition of an inflammatory infiltrate and degree of contracture has not been universally demonstrated although there are numerous studies supporting this association. There are, however, histopathological differences between implant types that explain certain characteristics. In general, smooth implants have uniform capsules, thick collagen fibres and few cells whereas textured and polyurethane (PU)-coated implants show a more marked cellular component, often associated with giant cell granulomata. Synovial-type metaplasia (STM) is seen predominantly with textured prostheses and this phenomenon may explain the increased mobility, and therefore reduced ACC in such devices. The mechanism may be through proteoglycans, which have both been implicated in the genesis of STM and inhibit collagen lattice contracture.

Silicone itself has been identified in three different forms in peri-prosthetic tissue:
- irregular, translucent, amorphous droplets
- intracellular droplets
- elastomeric fragments within giant cell granulomata

While the first two forms occur with all silicone gel filled implants, the third is relatively specific to textured surfaces, be they saline- or silicone-filled. It is assumed that granulomata result from attempted phagocytosis of particles shed from the elastomer and are extremely rare with smooth implants. The impact of free silicone as a pathological entity is still debated, but there is little doubt that low-bleed gel implants have contributed to the decreased incidence of ACC.

Aetiopathology

Upon implantation of any foreign material, opsonins immediately adsorb onto its surface allowing host immune cells to face a recognisable protein layer. Even if too large for digestion some elements of the phagocytotic sequence do occur. Orchestrated by multiple immune cell-derived cytokines, the net result of fibroblast proliferation and collagen production is, as always, scar deposition and encapsulation. Should a state of chronic inflammation, perhaps through persistent inflammatory stimuli, motion or infection, supervene the fibrotic response becomes magnified. Additionally, inherent biomaterial properties, including geometry and surface chemistry, are capable of altering tissue response and healing reactions. This facet has been successfully harnessed to combat ACC: physical modification and over-coating in the form of textured and PU-coated devices respectively. Pressure exerted on and by an implant also affects capsule formation, the latter principle being exploited by the latest generation, form-stable prostheses that provide resistance to the constrictive forces of an active capsule.
Historically, there have been two predominant, and somewhat artificially demarcated, investigative streams: the ‘endogenous’ and ‘exogenous’ hypotheses. Limitations of adopting one at the expense of the other have been cogently argued by Burkhardt, himself a declared proponent of the exogenous theory, when he demonstrated the observed incidence of ACC to be less than expected with an intrinsic factor alone, but more than if an exogenous factor was solely responsible. ACC is, of course, multi-factorial, the two philosophies complimentary and an ‘unified’ hypothesis combining features of both logical.

**Endogenous hypothesis**

Derangement of the normal healing reaction triggered by some unfavourable aspect of the host-implant interaction provides the basis for the endogenous hypothesis. Supporters believe this interface to be chiefly responsible for ACC based on the observation that it is not seen universally either between subjects or between individual breasts. As the majority never develop ACC, individual factors are clearly important if variable in their expression. Furthermore, were excessive contracture to be the result of prolonged or otherwise abnormal wound healing specific to a single individual, one would expect its occurrence bilaterally. No individual factor can explain the apparent randomness of the contracture, which is more commonly unilateral, but seven elements, predominantly characteristics of the implant, have received attention:

I. healing response modification
II. elastomer composition
III. implant content
IV. elastomer surface
V. polyurethane overcoating
VI. anatomical plane of implantation
VII. surgical technique

**Healing response modification**

Early on little was known about any means of influencing the healing response in general, and ACC in particular, other than with corticosteroids. Peri-implant triamcinolone was first used in the 1970s, however, adverse effects including parenchymal thinning, implant extrusion and ptosis were alarmingly frequent. Intra-implant methylprednisolone was substituted in order to obviate lower pocket pooling and Ellenberg et al.’s reduction from 67.6 to 9.9% prompted widespread use due to dismal ACC rates. Unfortunately, although the prevalence of ACC decreased, results were unpredictable and steroid-related complications sufficiently frequent to cause its abandonment.

**Elastomer composition**

With ACC having long been associated with both vacuolar and filler silicone, and with silicone droplets being absent with saline-filled implants, the theory of silicone ‘gel bleed’ was born. In an elegantly simple experiment, intact implants placed on filter paper evidenced trans-elastomer silicone permeation. Although silicone particles were later seen with saline-filled prostheses due to direct elastomer shedding, gel bleed is held responsible for the particularly high ACC rates of first- and second-generation implants. The fluoro-silicone barriered, third-generation silicone and saline implants have certainly contributed to reductions in the incidence of ACC (Table 2). Recently introduced highly cohesive gels, have allowed further improvements, probably through a combination of further limiting trans-elastomer silicone permeation and a simple physical resistance to contraction.

**Implant content**

With the United States having banned silicone prostheses for augmentation in the 1990s, it has a wealth of experience with saline-filled devices and Rohrich’s recent editorial was rather fulsome in its praise of them. Although good evidence for a protective effect against ACC exists in both prospective and retrospective, multicentre studies, significant aesthetic downsides and high device failure rates have limited their acceptance elsewhere in the world. Interestingly, a swift rebuttal to the biased editorial swiftly followed and with recent studies reporting that more women chose silicone-filled prostheses upon saline explantation coupled with reports of elevated systemic side effects and a poorer overall function in reconstruction with saline, the tide appears to be on the turn across the Atlantic.

---

**Table 1 Summary of documented capsule and clinical correlations**

<table>
<thead>
<tr>
<th>Author</th>
<th>ACC/thickness</th>
<th>ACC/inflammation</th>
<th>ACC/capsular silicone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wilflingseder (1974)</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Wagner (1977)</td>
<td>N/A</td>
<td>+/-</td>
<td>N/A</td>
</tr>
<tr>
<td>Domanskis (1976)</td>
<td>N/A</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Vistnes (1977)</td>
<td>+</td>
<td>+/-</td>
<td>+</td>
</tr>
<tr>
<td>Rudolph (1978)</td>
<td>+/-</td>
<td>N/A</td>
<td>-</td>
</tr>
<tr>
<td>Gayou (1979)</td>
<td>-</td>
<td>-</td>
<td>+/-</td>
</tr>
<tr>
<td>Thomsen (1990)</td>
<td>N/A</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Lossing (1993)</td>
<td>+</td>
<td>+</td>
<td>N/A</td>
</tr>
<tr>
<td>Van Diest (1998)</td>
<td>N/A</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Siggelkow (2003)</td>
<td>+</td>
<td>+/-</td>
<td>-</td>
</tr>
<tr>
<td>Prantl (2007)</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Author</td>
<td>Year</td>
<td>Generation</td>
<td>Number</td>
</tr>
<tr>
<td>----------</td>
<td>------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>p – patients</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>i – implants</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gylbert</td>
<td>1989</td>
<td>1st, 2nd, 3rd</td>
<td>15–21</td>
</tr>
<tr>
<td>Vinnik</td>
<td>1976</td>
<td>2nd</td>
<td>289 p</td>
</tr>
<tr>
<td>Hips</td>
<td>1978</td>
<td>2nd</td>
<td>453 p</td>
</tr>
<tr>
<td>Asplund</td>
<td>1984</td>
<td>3rd</td>
<td>65 p</td>
</tr>
<tr>
<td>McKinney</td>
<td>1983</td>
<td>2nd, 3rd</td>
<td>169 p</td>
</tr>
<tr>
<td>Gutowsky</td>
<td>1997</td>
<td>3rd</td>
<td>504 p</td>
</tr>
<tr>
<td>Scully</td>
<td>1981</td>
<td>2nd</td>
<td>100 p</td>
</tr>
<tr>
<td>Puckett</td>
<td>1987</td>
<td>3rd</td>
<td>3</td>
</tr>
<tr>
<td>Vazquez</td>
<td>1987</td>
<td>3rd</td>
<td>585 i</td>
</tr>
<tr>
<td>Coleman</td>
<td>1991</td>
<td>3rd</td>
<td>53 p</td>
</tr>
<tr>
<td>Ersek</td>
<td>1991</td>
<td>2nd, 3rd</td>
<td>228 p</td>
</tr>
<tr>
<td>Pollock</td>
<td>1993</td>
<td>3rd</td>
<td>197 p</td>
</tr>
<tr>
<td>Malata</td>
<td>1997</td>
<td>3rd</td>
<td>53 p</td>
</tr>
<tr>
<td>Hakelius</td>
<td>1997</td>
<td>3rd</td>
<td>25 p</td>
</tr>
<tr>
<td>Tarpila</td>
<td>1997</td>
<td>4th</td>
<td>21 p</td>
</tr>
<tr>
<td>Collis</td>
<td>2000</td>
<td>3rd</td>
<td>53 p</td>
</tr>
<tr>
<td>Fagrell</td>
<td>2001</td>
<td>4th</td>
<td>20 p</td>
</tr>
<tr>
<td>Inamed-1</td>
<td>2007</td>
<td>5th</td>
<td>455</td>
</tr>
<tr>
<td>Inamed-2</td>
<td>2007</td>
<td>5th</td>
<td>147</td>
</tr>
<tr>
<td>Mentor-1</td>
<td>2007</td>
<td>5th</td>
<td>551</td>
</tr>
<tr>
<td>Mentor-2</td>
<td>2007</td>
<td>5th</td>
<td>146</td>
</tr>
<tr>
<td>Handel-1</td>
<td>2006</td>
<td>5th</td>
<td>825 p</td>
</tr>
<tr>
<td>Handel-2</td>
<td>2006</td>
<td>5th</td>
<td>695 p</td>
</tr>
<tr>
<td>Stevens</td>
<td>2008</td>
<td>5th</td>
<td>1012 i</td>
</tr>
<tr>
<td>Heden</td>
<td>2009</td>
<td>5th</td>
<td>163 p</td>
</tr>
</tbody>
</table>
Elastomer surface

Texturisation of the silicone elastomer was prompted by the appreciably lower ACC rates seen with PU-coated implants\(^{39}\) and early studies were strongly supportive.\(^{40-43}\) Later studies provided a somewhat different view,\(^{44,45}\) and debate was for a time intense. Fortunately, some quality prospective studies are available to evidence the benefit of texturisation in the first year,\(^{41,46}\) when the majority of contractures occur:\(^{47}\) this benefit being maintained at 5\(^{47}\) and 10 years.\(^{48}\) Recently, a pair of meta-analyses calculated the overall benefit of textured implants to be an approximate five-fold reduction in ACC, maintained after three years\(^{49,50}\) (Table 3).

As to the texturisation itself, it appears that some element is more important than the precise surface detail as long as pore size exceeds 300 \(\mu\)m.\(^{49}\) From a clinical perspective, experience has shown a curious double-capsule effect with some textured implants, particularly McGhan: there is an apparent pseudo-encapsulation of the fibrous-coated implant appears to float within a second capsule, postulated to result from shearing forces separating a tightly adherent capsule.\(^{51}\)

Polyurethane overcoating

Polyurethane was originally applied as a coating for sponges found to be problematic in the 1950s. Initial reports were highly favourable (Table 4),\(^{32-35}\) and the capsules appeared distinctly different from those surrounding silicone. The much reduced ACC was ascribed to a disruption in the normally parallel capsular collagen fibres, which inhibited circumferential contraction. There was also an increased cellular content and marked adhesion to the implant that resulted not from the textured surface per se, but particles of polyurethane that had become detached.\(^{56}\)

As with textured silicone, long-term, homogenous data generated from PU-coated devices showed mixed results. Cooney’s single practice series showed ACC rates similar to silicone implants\(^{57}\) whereas Handel’s multi-surgeon study showed PU-coated implants to have very low rates, when compared with smooth or textured implants, but still rising to a level of 25% at 10 years.\(^{45}\) The latter suggested PU-coated implants delayed rather than prevented the onset of ACC because of the gradual loss of the polyurethane overcoat. Again, as with silicone, PU-coated devices have themselves survived scaremongering with respect to the potential carcinogenic risk of the breakdown-product 2,4-toluenediamine (TDA), which was later shown not to be clinically significant.\(^{58,59}\)

| Table 3 | Meta-analysis of prospective studies comparing textured with smooth implants and the incidence of ACC (See Wong et al\(^{\text{[66]}}\)) |
|---|---|---|---|---|
| Hakellius (1992) | 25 | 1 yr (100) | Textured better | 0 vs. 40 |
| Coleman (1991) | 53 | 1 yr (94) | Textured better | 8 vs. 58 |
| Collis (2000) | 33 | 3 yr (92) | Textured better | 11 vs. 59 |
| | | 10 yr (83) | | 11 vs. 65 |
| Tarpila (1997) | 21 | 1 yr (90) | No difference | 29 vs. 38 |
| Fagrell (2001) | 18 | 7.5 yr (90) | No difference | 22 vs. 33 |
| Burkhardt (1994) | 56 | 1 yr (87) | Textured better | 2 vs. 40 |

Anatomical plane of implantation

The subglandular plane was the initial standard, not least for its ‘anatomical’ nature, however, implantation beneath pectoralis major, introduced to ameliorate extrusion and visibility following subcutaneous mastectomy,\(^{60}\) was also found to greatly reduce the contracture rate.\(^{61}\) With respect to ACC specifically, evidence exists for a significant reduction with the submuscular location independent of surface texture,\(^{48}\) perhaps consequent upon the continuous massaging action of pectoralis major.\(^{61}\) The musculofascial plane also affords protection, indirectly via reduced infection, from potentially pathogenic breast flora\(^{62}\) and improves mammographic visualisation.\(^{63}\) Although one might expect a greater effect from combining texturisation with submuscular placement, the sole study to evaluate this failed to reach statistical significance.\(^{42}\)

Surgical technique

Although clearly open to influence by personal technique there are some key elements whereby surgical decision-making may influence ACC. The first is the choice of incision and non-inframammary fold placement was associated with an almost 6-fold increase in the relative risk (RR) of ACC in one large prospective study.\(^{64}\) Albeit retrospective, another study showed peri-areolar incisions to give a RR of 16, probably through violation of the mammary ductal system,\(^{65}\) and patients in whom concurrent areolar reduction is deemed beneficial should be counselled accordingly.

Haematoma had fallen under suspicion previously\(^{66,67}\) and Handel recently confirmed its correlation with ACC,\(^{45}\) however, blunt dissection has, or should have, been entirely superseded by direct vision and meticulous haematostasis\(^{68}\) to render this issue of lesser importance today, although a role for occult haematoma probably remains. Mirroring this, drains were found to be beneficial initially,\(^{67}\) but the current low prevalence of ACC is associated with their avoidance.\(^{69-71}\)

Exogenous hypothesis

Proponents of the exogenous hypothesis cite infection as the leading cause for ACC\(^{72,73}\) and although explaining asymmetric occurrence, the theory falls short of definitive causality. Overt infection is a rare event in BA, perhaps surprisingly so given the magnitude of the foreign body challenge, and much effort has been expended in studies...
Involving bacteriostatic and bacteriocidal agents. The former, in the shape of povidone-iodine, has been clearly shown to reduce ACC: Burkhardt’s landmark trial, addressing two of Koch’s postulates, showed that ACC was both caused by infection and could be inhibited by local povidone-iodine. It also appears to possess a synergistic benefit when combined with antibiotics as a pocket irrigant, but has been banned by the FDA due to concerns with elevated device rupture following intra-luminal administration. This prompted the combination of multiple antibiotics, which have produced even lower ACC rates.

Beyond ‘infection’, in a non-specific sense, evidence has accumulated for the role of biofilms in mediating ACC. Surface bacteria secrete a hydrated polymeric matrix in which they aggregate as sessile communities — the ‘biofilm’ — which are much less conspicuous to the host immune system and provide an explanation for antibiotic-resistant and culture negative inflammation. Positive culture rates in severely contracted capsules have been quoted as high as 89.5% compared to 10.5% in grade I/II culture rates in severely contracted capsules have been.

A unified view of ACC and potential non-surgical treatment modalities

If encapsulation is the normal end-point, then ACC is an aberrant healing response comprising a more active role for the immune system. Fibroblasts and macrophages are the predominant capsular cell types in the inner layer, beneath which lie activated CD4+ and numerous antigen presenting cells. Overall, an important T-mediated immune response is postulated as the basis for encapsulation as serum samples from ACC patients have increased levels of cytotoxic activity, but a clear and precise causal relationship has yet to be established.

The cytokine TGF-ß is topical and appears to play a fundamental role in numerous conditions characterised by excessive fibrosis, including keloid. Both myofibroblasts and macrophages from ACC patients express ß1 and ß2 isoforms, indicating that the local capsular cytokine environment may be the long-sought missing trigger for abnormal inflammatory responses. TGF-ß1 and ß2 levels, and their downstream mediator, connective tissue growth factor, are higher in periprosthetic capsules. TGF-ß inhibition, both with antibody-receptor and antisense-mRNA blockade, reduces skin scarring and ACC in animal models and has been found to decrease capsule thickness.

There are other unexpected avenues for potential research, for example angiotensin II, which has a range of effects beyond that of haemodynamic regulation. It protects against renal fibrosis and ACE-inhibitors diminished inflammatory cell infiltrate, collagen III content, vessel density, TGF-ß immunostaining and capsule thickness in a recent rat study where, interestingly, textured performed uniformly better than smooth surfaced implants. Leukotrienes are potent inflammatory mediators and their antagonists, including Zafirlukast, have been trialled with some success in early and established ACC, however, these agents are presently ‘off label’ for ACC and, in their usual guise of asthma prophylactics, have been somewhat alarmingly linked with reports of hepatic failure and death. Finally, tissue remodelling matrix metalloproteinases (MMP) and their inhibitors (tissue inhibitor of metalloproteinase, TIMP), are altered in conditions associated with unbalanced ECM degradation. Concentrations of certain TIMPs was found to be elevated and the MMP:TIMP ratio reduced in ACC. Intriguingly, hyaluronan levels correlate with Baker grade so may offer the possibility of a serum marker for ACC.

Classification

Several methods have been proposed for the assessment and quantification of ACC (Table 5). Baker’s original clinical classification underwent subtle modification to accommodate breast reconstruction and patient symptoms and, despite the lack of critical validation, remains widely accepted. The present ‘British standard’ has been presented by Stone. The Swedes favour the Breast Augmentation Classification (BAC), based on Baker’s, but excluding the opinion of the patient. Although subjective, grades I and II are considered acceptable and III and IV not, the latter two constituting adverse capsular contracture. Applanation tonometry is a more objective technique that measures breast displacement by a perspex disc of known weight and has been shown to be more sensitive in detecting early contracture than the BAC, but is not used routinely.

The magnitude of the problem: prevalence of ACC

Breast implants have generated debate since their very introduction and, whilst the 1990’s were devoted to safety concerns regarding autoimmune disease and cancer, the current focus has shifted to adverse local effects. While patient satisfaction with breast augmentation remains high, the most frequent local complication is ACC, which, if not posing actual health risks, may be symptomatic, compromises the aesthetic outcome and frequently requires surgical intervention. Furthermore, patients with ACC are more likely to have it recur after revision, fuelling a vicious circle of poor outcomes.

**Table 4 Incidence of ACC with PU-coated implants**

<table>
<thead>
<tr>
<th>Baker III/IV ACC (%)</th>
<th>Number</th>
<th>Follow up (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Capozzi (1991)</td>
<td>1.8</td>
<td>54</td>
</tr>
<tr>
<td>Pennisi (1990)</td>
<td>2 vs. 22</td>
<td>85 vs. 115</td>
</tr>
<tr>
<td>Melmed (1988)</td>
<td>0.9</td>
<td>320</td>
</tr>
<tr>
<td>Hester (1988)</td>
<td>0.2</td>
<td>690</td>
</tr>
<tr>
<td>Gasperoni (1992)</td>
<td>3.3</td>
<td>210</td>
</tr>
<tr>
<td>Vazquez (2007)</td>
<td>0.5</td>
<td>404</td>
</tr>
<tr>
<td>Colney (1991)</td>
<td>28</td>
<td>173</td>
</tr>
<tr>
<td></td>
<td>58</td>
<td></td>
</tr>
<tr>
<td></td>
<td>19</td>
<td></td>
</tr>
<tr>
<td>Handel (2006)</td>
<td>25</td>
<td>305</td>
</tr>
</tbody>
</table>

* The two values refer to delayed and immediate reconstruction after subcutaneous mastectomy.
Fortunately, ACC rates have improved dramatically over the years (Table 2) even if the numerous generations, fillers and other variables make data extrapolation by necessity vague. Whilst thought to occur primarily in the first two years, with some reporting a zero rate of delayed ACC, extended surveillance shows that there is, in fact, a long-term, cumulative increase in ACC. There are, however, recent studies that have addressed long-term follow up with homogeneous implants. Both Inamed (www.allergan.com) and Mentor (www.mentorcorp.com) have released premarket, core study data. Other single-institution or -surgeon, fourth-generation outcomes show encouraging results over 11 and 13 years respectively. These results are summarised in Table 2 and, as is apparent, the prevalence of ACC remains not inconsequential indicating that a significant number of patients may well require revision surgery in the future.

Discussion

As shown by the research currently available to us, ACC has a multifactorial and incompletely characterised aetiology. Surgical trauma initiates wound healing in the normal fashion, but this may be modified by inherent implant characteristics or the local milieu to produce an abnormal pathological outcome. In a fashion not dissimilar to oncogenesis, there may in some cases be a single initiator that is sufficiently potent to produce the effect alone, for example, frank infection. In others the initiator, perhaps the biomaterial-host interface, may require potentiation by promoter(s), such as occult contamination or haematoma. Whatever the precise situation, such factors precipitate a chronic inflammatory reaction that stimulates a cascade resulting in propagation of the fibrogenic stimulus (Figure 3). In conclusion, a solid evidence base exists for avoiding direct contact between gel silicone and host tissues, by both the fluorinated and multilaminated elastomer and the high cohesivity of the gel itself. Texturisation or polyurethane coating are also proven reducers of ACC as is something antibacterial, previously povidone-iodine, today synergistic antibiotic combinations. The submuscular plane also appears clear-cut from an evidentiary view, but the additional depth and cover might simply mask ACC rather than reducing its prevalence per se. Surgeon-influenced aspects include operative precision, meticulous haemostasis and avoidance of haematoma with the need for drainage.

Whilst aetiologically not yet completely unravelled, the incidence of ACC has been brought to manageable levels and may soon become an endangered species helping to spare our patients further, and less predictable, surgical intervention some of the strategies of which will be explored in a future review. To borrow from Burkhardt ‘hard breasts, soft data’ we can expect much softer breasts and are beginning to firm up on the aetiology of the sometimes capricious entity that is ACC. Paradoxically, as its occurrence diminishes fewer index cases to research may shift us further from the precise answer. In this respect, continued homogeneous, long-term studies yield

<table>
<thead>
<tr>
<th>Table 5</th>
<th>Comparison of subjective assessment methods of capsular contracture</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I</td>
<td>Absolutely natural; no one could tell breast augmented</td>
</tr>
<tr>
<td>Class II</td>
<td>Minimal contracture: I can tell surgery was performed, but patient has no complaint</td>
</tr>
<tr>
<td>Class III</td>
<td>Moderate contracture; patient feels some firmness</td>
</tr>
<tr>
<td>Class IV</td>
<td>Severe contracture; obvious from observation</td>
</tr>
<tr>
<td>Grade I</td>
<td>Soft No deformation</td>
</tr>
<tr>
<td>Grade II</td>
<td>Slightly thickened consistency None-to-slight deformation</td>
</tr>
<tr>
<td>Grade III</td>
<td>Firm to hard None-to-slight deformation</td>
</tr>
<tr>
<td>Grade IV</td>
<td>Hard Severe deformation</td>
</tr>
</tbody>
</table>
Breast augmentation — adverse capsular contracture

strong objective data on which to base discussion with the patient about possible outcomes of a procedure that remains one of the most satisfactory in cosmetic surgery.

Conflict of interest

None.

Funding

None.

References

29. Barker DE, Retsky MI, Shultz S. "Bleeding" of silicone from bag- 
gel breast implants and its clinical relation to fibrous capsule 
implant core study. results at 6 years. Plast Reconstr Surg  
2007;120:85–185.
31. Rohrich RJ, Reece EM. Breast augmentation today: saline 
versus silicone — what are the facts? Plast Reconstr Surg 2008; 
121:669–72.
32. Asplund O. Capsular contracture in silicone gel and saline-filled 
breast implants after reconstruction. Plast Reconstr Surg 1984; 
73:270–5.
33. Gylbert L, Asplund O, Jurell G. Capsular contracture after breast 
reconstruction with silicone gel and saline-filled implants; a 6- 
34. McKinney P, Tresley G. Long-term comparison of patients with 
gel and saline mammary implants. Plast Reconstr Surg 1983; 
35. Gutowski KA, Mesna GT, Cunningham BL. Saline-filled implants; 
a plastic surgery educational foundation multicenter outcome 
36. Haecke P, Galsberg SB, Roth AZ, et al. The saline versus sili-
cone breast implant debate: separating fact from fiction. Plast 
37. Stevens WG, Pacella SJ, Hirsch E, et al. Patient retention and 
replacement trends after saline breast implants: are deflations 
38. Macadam SA, Ho AL, Cook EF Jr., et al. Patient satisfaction and 
health-related quality of life following breast reconstruction: a 
comparison of patient-reported outcomes amongst saline and 
1097/PRS.0b013e3181cb5c18.
contracture: the conventional breast implant and the Pitts-
40. Pollock H. Breast capsular contracture: a retrospective study of 
textured versus smooth silicone implants. Plast Reconstr 
41. Coleman DJ, Foo ITH, Sharpe DT. Textured or smooth implants 
for breast augmentation? A prospective controlled trial. Br J 
42. Asplund O, Gylbert L, Jurrell G, et al. Textured or smooth 
implants for submuscular breast augmentation: a controlled 
43. Burkhardt BR, Demas CP. The effect of Siltex texturing and 
povidone-iodine irrigation on capsular contracture around 
saline inflatable breast implants. Plast Reconstr Surg 1994;93: 
129–30.
44. Fagrell D, Berggren A, Tarpila E. Capsular contracture around 
saline-filled fine textured and smooth mammary implants: a 
prospective 7.5-year follow-up. Plast Reconstr Surg 2001; 
108:2108–12.
45. Handel N, Cordray T, Gutierrez J, et al. A long-term study of 
outcomes, complications, and patient satisfaction with breast 
46. Hakelius L, Ohlsen L. A clinical comparison of the tendency to 
capsular contracture between smooth and textured gel-filled 
silicone mammary implants. Plast Reconstr Surg 1992;90: 
247–54.
47. Hakelius L, Ohlsen L. Tendency to capsular contracture around 
smooth and textured gel-filled silicone mammary implants: a 
48. Collins N, Coleman DJ, Foo IT, et al. Ten-year review of a 
prospective randomised controlled trial of textured versus 
textured subglandular silicone gel breast implants. Plast 
49. Wong CH, Samuel M, Tan BK, et al. Capsular contracture in 
subglandular breast augmentation with textured versus smooth 
breast implants; a systematic review. Plast Reconstr Surg 2006; 
118:1224–36.
50. Barnsley GP, Sigurdson LJ, Barnsley SE. 2006 PRS 117 2182 
Textured surface breast implants in the prevention of capsular 
contracture among breast augmentation patients: a meta- 
analysis of randomized controlled trials. Plast Reconstr Surg 
2006;117:2182–90.
51. Pandya AN, Dickson MG. Capsule within a capsule: an unusual 
52. Capozzi A. Long-term complications of polyurethane-covered 
53. Pennisi VR. Long-term use of polyurethane breast prostheses; 
54. Melmed EP. Polyurethane implants: a 6-year review of 416 
with the polyurethane-covered mammary prostheses for 
treatment of capsular contracture, primary augmentation and 
56. Barone FE, Perry RN, Maxwell P, et al. The biomechanical and 
histopathologic effects of surface texturing with silicone and 
polyurethane in tissue implantation and expansion. Plast 
57. Cooney BC, Cooney TB, Hearne VA. Nineteen years experience 
with polyurethane foam-covered mammary prosthesis: a 
2.4–toluenediamine in urine and serum samples from women with 
59. Hester Jr TR, Tebbetts JB, Maxwell GP. The polyurethane- 
covered mammary prosthesis: fact and fiction (II). Clin Plast 
60. Dempsey WC, Latham WD. Subpectoral implants in augmenta-
61. Scully SJ. Augmentation mammoplasty without contracture. 
infection in significant breast implant capsules. Plast Reconstr 
mammographic visualization of the breast after augmentation 
64. Henriksen TF, Fryzek JP, Holmich LR, et al. Surgical interven-
tion and capsular contracture after breast augmentation: a 
65. Wiener TC. Relationship of incision choice to capsular 
66. Williams C, Aston S, Rees TD. The effect of hematoma on the 
thickness of pseudosheaths around silicone implants. Plast 
67. Hippis CJ, Raju DR, Strelth RF. Influence of some operative 
and postoperative factors on capsular contracture around breast 
68. Tebbetts JB. Achieving a predictable 24-hour return to normal 
activities after breast augmentation: part I. Refining practices 
by using motion and time study principles. Plast Reconstr 
69. Heden P, Jernbeck J, Hoher M. Breast augmentation with 
70. Adams WP, Rios JL, Smith SJ. Enhancing patient outcomes in 
aesthetic and reconstructive breast surgery using triple anti-
biotic breast irrigation: a six-year prospective clinical study. 
71. Cunningham B. The mentor core study on silicone MemoryGel 
discussion 305–325.
Breast augmentation — adverse capsular contracture


