Abstract. Background: Capsular contracture is the most common complication and the main cause of dissatisfaction after augmentation mammoplasty, for both the patient and the plastic surgeon. The formation of fibrous tissue around the prosthesis alters the form or the consistency of the implant, thus modifying the breast shape, its contour and its softness. The initial satisfaction with the achieved aesthetic result is then transformed into great dissatisfaction, due to the presence of a shapeless and undesired mass. Patients and Methods: The following study considered data collected between 1998 and 2007. Sixty-seven female patients (aged between 35 and 53 years) who suffered from mammary hypotrophy and had undergone submuscular augmentation mammoplasty were enrolled. All the implanted prostheses were round and texturized, with a volume of 250 cm$^3$ to 450 cm$^3$. The patients underwent pre-, intra- and postoperative antibiotic therapy in order to prevent clinical and subclinical infection of the implants. Results: The follow-up ranged from a period of two to nine years. All patients were examined during the first antibiotic administration and again subsequently, after 1, 3, 6 and 12 months, to evaluate the results in terms of capsular contracture. Of all patients, 90% presented a degree I Baker’s classification, the remaining 10% a degree II. Not one of the patients treated showed grade III or IV capsular contracture nor was there any need to remove the prosthesis during the examination period. Conclusion: It is clear that a main role in capsular contracture is played by the infectious process, with the activation of specific inflammatory cells. Interfering with the infectious process can prevent fibrotic reaction evolving into capsular contracture. Although the process causing capsular contracture is multifactorial, our study showed a favourable response can be achieved when using antibiotic therapy associated with the transaxillary approach.

Capsular contracture represents the most common, long-term complication and main cause of dissatisfaction after augmentation mammoplasty, for both the patient and the plastic surgeon. The fibrous tissue generated around the prosthesis alters either the form or the consistency of the implant, thus modifying the breast shape, its contour and its softness (1). The initial gratification for the achieved aesthetic result may change into dissatisfaction, due to the presence of an undesired mass.

Capsular contracture may have various results. The augmented breast may be normal or may show various degrees of asymmetry, firmness and uneasiness. The fibrotic capsular contracture brings firmness to the prosthesis, causing the shape to change and as a consequence, the breast may become painful. In the literature, the incidence of capsular contracture ranges between 0.5 and 30% (2). This wide difference is also enhanced by the subjective measurement of Baker’s classification, depending on the experience of the plastic surgeon.

However, accurate studies have failed to identify a single cause of capsular contracture and several factors, both intra- and postoperative, have been held responsible for its development (3). There are still no accurate data available that identify the origin of the fibrosis. Many factors have been advocated to justify the onset of capsular contracture, including the type of implant used, its coating, the pocket position and some pre- and/or postoperative precautions followed by the surgeon (3-12). In order to avoid such complications, some measures of prevention have been proposed either intraoperatively (meticulous haemostasis, the use of catheters for the aspiration, intraluminal insertion of steroids and antibiotic, the use of texturized implants, positioning of a subpectoral implant and perioperative antibiotics), or postoperatively (massage to the breast, movement exercises on the implant, protracted pressure and...
topical or oral use of vitamin E). Despite this, it was ascertained that such treatments were not sufficient to prevent capsular contracture (3-5, 10, 12).

Among the different hypotheses, capsular contracture can also be attributed to an infectious process caused by *Staphylococcus epidermidis* present in all galactophores and, as a consequence, in the mammary milk, nipple secretion and breast parenchyma (7, 8). It is also possible that *S. epidermidis* could contaminate the breast implant pocket thus causing the onset of capsular contracture (9). Other microbes may also be responsible for capsular contracture.

At present, the treatment of capsular contracture is essentially surgical and consists of a capsulectomy or capsulotomy, which creates a new pocket with fresh tissues for the implant; however, capsular contracture may still occur (13). Considering the infective hypothesis as the main cause of capsular contracture, we report our retrospective study of patients treated with antibiotic therapy alone for augmentation mammoplasty, through a transaxillary approach.

**Patients and Methods**

The following study considered data collected between 1998 and 2007. Sixty-seven female patients aged between 35 and 53 years, average weight 62 kg, who suffered from breast hypotrophy having undergone augmentation mammoplasty for aesthetic purposes were enrolled in this study.

The patient selection criteria were based on the insertion site of the breast prosthesis and the site of implant namely transaxillary. The patients underwent pre-, intra- and postoperative antibiotic therapy in order to prevent infection of the implants. All 67 patients underwent a breast augmentation with a round, textured breast prosthesis. The incision was performed posteriorly to the anterior axillary line and the subpectoral pocket was created using a Dingman paddle trans-dissector with swan-neck as shown in Figure 1. Drains were used in all cases, with their exit points positioned 2 cm inferiorly to incision points. All implants were textured with a volume of 250 cm³ to 450 cm³. The patients enrolled in this study received an antibiotic treatment (Table I) in order to prevent infection. On the operating day, patients received the following intravenous antibiotics: during the induction of anaesthesia, 600 mg rifampicin and 3 g ampicillin+ sulbactam; followed by 3 g intravenous ampicillin+ sulbactam in the afternoon, repeated in the evening. The postoperative antibiotic treatment of patients was administered as follows: day 1 to 4, 600 mg of rifampicin once a day and 500 mg ciprofloxacin twice a day; day 5 to 7, 500 mg ciprofloxacin, twice a day. The period of follow-up ranged from 1 to 10 years. All patients were examined using Baker’s classification (12-13) at 1, 3, 6 and 12 months, and every year in order to evaluate the onset of capsular contracture.

**Results**

We observed that 90% (60 patients) of the patients presented a degree I Baker’s classification, the remaining 10% (7 p.) a degree II. Not one of the patients treated showed grade III or IV capsular contracture. There was no need to remove the prosthesis during the examination period.

The antibiotic therapy showed favourable results in the prevention of the capsular contracture.

No complications were observed during or after the antibiotic treatment. Furthermore, all patients underwent blood test examinations before, during and after the therapy, and no alterations of the tests were observed.
Figure 2. Preoperative and long term postoperative augmentation mammoplasty in three cases.
Figure 2 shows the natural results of three cases of augmentation mammoplasty (preoperative and long-term postoperative). Baker class I, with transaxillary approach, accompanied by an antibiotic therapy as described in Table I.

**Discussion**

Around 12 million women worldwide have breast implants, either for aesthetical or reconstructive reasons (2, 5). Numerous studies indicate that these implants do not pose a health risk, are not linked to illnesses of the connective tissue and, furthermore, do not increase the risk of breast cancer (14-15). It has been demonstrated that silicone is not carcinogenic to humans nor is it associated with the increased incidence of breast cancer (12, 15). Furthermore, breast prostheses do not interfere with imaging during breast screening (16). Many patients worry about the effects of surgery on the breast and the nipple sense (17). In cases of diminished sensibility, this usually ceases in a few weeks or months, accompanied with paresthesia and dysesthesia (17). The decrease in sensibility is directly proportional to the diameter and size of the breast implant. The potential for breastfeeding is not compromised, neither when incisions inside the breast parenchyma are avoided, nor when implants are positioned behind the mammary gland or behind the pectoral muscle (18-19).

However, the exact mechanism of the response to breast implants is still unknown. It is known that an infectious process is triggered, generating healing around the prosthesis. The response can produce an alteration in the shape of the implant or can be responsible for painful healing (8, 20). Capsular contracture is the most common, long-term complication of augmentation mammoplasty and is still unresolved (14). Since the first studies on capsular contracture, numerous methods have been reported in order to prevent this process (21). Almost all studies carried out on capsular contracture used Baker’s classification. According to Baker’s classification, capsular contracture is classified by using values ranging from 1 to 4, on the basis of parameters based on inspection and palpation (Table II) (1, 10, 22). Unfortunately, this classification is strongly dependent on the subjective evaluation by the surgeon.

Many factors seem to influence the onset of capsular contracture: the type of implant, the material coating the implant, the breast pocket and the pre- and/or postoperative precautions followed (23). Textured implants were created to reduce the chance of capsular contracture in comparison with smooth breast implants (9). Breast implants coated with polyurethane foam also cause the lowest incidence of capsular contracture (9-25).

Experimental study has shown that as a consequence of using a silicone implant, a foreign body reaction results and the host tissues surround or encapsulate the implant. Studies on myofibroblasts have evidenced the presence of these cells in samples of both the contracted mammary capsule and the non-contracted one. The population of myofibroblasts appears during the initial inflammatory phase and the repair phase, and disappears during the remodelling phase of the cicatrisation process (25-26). The presence of myofibroblasts in contracted and non-contracted capsules suggests that all capsules contract to a certain degree. Within this biological environment, factors such as infection or haematoma could worsen the foreign body reaction in capsular contracture.

It is difficult to establish the factors involved in the genesis of capsular contracture, despite the numerous studies (9, 24, 25). Two main hypotheses were made to explain capsular contracture: the first is based on a non-infectious process, while the second is based on an infectious one (26).

The non-infectious hypothesis identifies silicone as both the cause and the site of origin of the capsular contracture. The possibility that the use of silicone could give rise in capsular contracture has not been supported by all studies (27-29). Other authors have discovered that subclinical infection is the principal cause of capsular contracture (30).

The infection hypothesis is based on the fact that all galactophores containing *S. epidermidis* grew either in the mammary milk or in the nipple secretion, and were also present in the breast parenchyma (7, 8). However, it is also possible that the *S. epidermidis* contaminating the breast implant pocket could be one of the causes of the onset of capsular contracture (9). It is also possible that other microbes could be responsible. Other authors have discovered that, in the presence of subclinical infection, the capsules generating around the silicone implant are bigger and show an abundant creation of collagen (9, 13). It was also apparent that even when the infection was clinically resolved, capsular contracture also increased (31-32). These authors have hypothesised that the infection is not entirely resolved, but becomes subclinical (8, 33-34).

Once capsular contracture has occurred, the recommendations for the treatment are: breast massage, leukotriene receptor antagonist therapy, oral therapy with

<table>
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<tr>
<th>DEGREE</th>
<th>Classification</th>
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<tr>
<td>I</td>
<td>Normal inspection and palpation: the breast consistency is identical to the non-operated one.</td>
</tr>
<tr>
<td>II</td>
<td>The inspection is normal but not the palpation; the implant is palpable but not visible and the breast consistency is augmented.</td>
</tr>
<tr>
<td>III</td>
<td>Either the inspection or the palpation result abnormal; the breast is hard and the prosthesis is palpable and visible.</td>
</tr>
<tr>
<td>IV</td>
<td>Patients refer mammary and mastodinia tension; deformation of the implant and the overlying skin is evident.</td>
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**Table II. Baker classification (1975).**
vitamin E, and surgical operation (capsulectomy or capsulotomy, with substitution of the implant or modification of its position) (3-5, 10, 12, 33-35). Nevertheless despite the use of these treatments, their success still remains highly variable (35-39). Not even surgical operation can guarantee a relapse-free result (33). The second surgical operation itself brings a number of potential complications, such as haematoma, infection, deflation, implant weakening and breast asymmetry, as well as the necessity of further revision (40-42). Most surgeons agree that the best treatment and the best means at their disposal in order to reduce the incidence of capsular contracture is prevention (23). In this study, antibiotic administration was continued postoperatively to avoid any contamination of the implant through the drains. The antibiotic approach with different antibiotics probably avoids the adhesion, biofilm production and multiplication of bacteria.

Rifampicin is a bactericidal antibiotic of the rifamycin group and is able to penetrate, in particular, Gram-positive bacteria, such as Staphylococci. Sulbactam associated with ampicillin is able to inhibit beta-lactamase and to penetrate both Gram-positive and Gram-negative bacteria, offering an immediate broad-spectrum treatment. Ciprofloxacin was administered orally after the day of operation until the healing of the breast was complete and the drains were removed. This is a fluoroquinolone antibiotic with a broad-spectrum action, inhibiting cell division, and is active against both Gram-positive and Gram-negative bacteria.

In our opinion, since the transaxillary access is far from the nipple and the ducts, there is a very low risk of contamination (4-5). Furthermore, transaxillary access allows more than enough healthy tissue between the implant and the surgical skin incision. Although the causes of capsular contracture after augmentation mammoplasty or breast reconstruction are still unknown, it is clear that a key role is played by a clinical or subclinical infectious process, with the activation of specific inflammatory cells. Interfering with the infectious process can prevent the fibrotic reaction from evolving into capsular contracture (43). Many authors agree with the infectious hypothesis of capsular contracture, but, at present, no common treatment of this complication exists (23, 44-46). Our study showed favourable results using antibiotic therapy and the transaxillary approach.

References

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