Breast Augmentation: Procedure, Complications & Treatment. (REVIEW ARTICLE)

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Abstract:

Breast Augmentation is one of the most common aesthetic procedures performed with a very high satisfaction rate. After the description of first report of implant placement in 1964 by Cronin and Gerow, the popularity of the procedure is on the rise. The complications are few but require thorough understanding of each one of them for their prompt management and treatment. The procedure is generally performed by a prosthesis or implant. These implants are available in different sizes, shapes and profile and the surface of implant shell can be textured, micro textured or smooth with advantages to each type.

Procedure can be performed through submammary, intra-areolar, axillary or transumblical approach. These implants can be placed in partial submuscular, dual plane, muscle splitting biplane, subfascial and subglandular pockets with advantages to each.

In this chapter, over 1700 breast augmentations performed by the author were reviewed. All procedures were performed under general anaesthetic. Textured and smooth surface implants were used and using intra-areolar or submammary incisions, the devices were placed in partial submuscular, subglandular and muscle splitting biplane. All patients had at least single intravenous antibiotic and most of the procedures were performed as day cases.

Complications following breast augmentation are few and may present early or late. Early common complications are haematoma, seroma, infection and Mondors' Disease. Late complications include capsular contracture, implant rupture, malplacement of implant, dynamic breasts, implant flipping and rippling of the implant etc.

The management and treatment of these complications were assessed and out come evaluated.

Synmastia, bottoming down, periprosthetic infection and dynamic deformities are revisited and their treatment plans redefined. Mondors disease incidence, asymptomatic as well as symptomatic, was researched and its various presentations associated with breast augmentation described. Implant rupture, its association with quality and handling was reviewed. Implant rupture, its presentation and treatment plan redefined. Relationship of infection to length of antibiotic is analysed and various treatment modalities of periprosthetic infection and their outcome was assessed and suggestions and strategies for their management outlined.

Breast augmentation is a procedure with a very high satisfaction rate however complications arising following augmentations needs to be carefully evaluated and require a proactive and appropriate action plan. An informed consent should ideally include information outlining benefits of breast augmentation, possible complications along with the management plan of each one of them.

Introduction:

Historically women's breasts have always been considered as an attractive part of their body with an important anatomical, physiological and above all psychological role. A confident image of a woman is essential for her self-esteem and to carry out a competitive and constructive role in present day society. Almost every woman requesting augmentation mammoplasty expresses lack of confidence which in turn may affect her work, relationship with her partner, social life, choice of clothing, holidays etc. The patients requesting augmentation mammoplasty may feel inadequate due to the small size of the breast, their shape or associated asymmetry. They feel confident postoperatively with the attainment of a proportionate body that boosts their role in an interacting environment.

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It is interesting that, with the similar anatomical base, every breast depends in a way that makes it appear different in almost every woman.[1] The breast is composed of parenchyma, fat and skin and all three components are dynamic in nature and changes are seen in a woman right from the onset of puberty. These changes are noticed during normal cyclical changes of the month, due to body weight or fat changes, during pregnancy and lactation and are also seen with the passage of time.[2,3] Request to regain the shape and enhance the size of a breast is quite natural and implants quality, safety and choice have played an equally important role in the continued popularity of the procedure. Since the first report of implant placement in 1964 by Cronin and Gerow,[4] a large number of women have benefited from the procedure, that has almost certainly improved their quality of life. This is the reason that breast augmentation using breast implants, is one of the most common aesthetic procedures performed by plastic surgeons today. The current chapter is dedicated to the techniques, complications and management of complica-

tions related to augmentation mammoplasty using breast implants.

Surgical anatomy and aesthetic considerations:

The breast in a female is highly variable but the size of the base of the breast is fairly constant and extends from second to sixth rib in midclavicular line. From this circular base the breast protrudes and depends to a degree that varies in almost every individual female.[1] The unique anatomical shape of breast always appears different when viewed from different angle. An aesthetic breast has four anatomical boundaries but only three of them are visually distinct. In a dependent position, upper pole gradually merges with the bidimensional upper chest, medial, lower and lateral limits forms the other three anatomically distinct boundaries. Inframammary crease, extends from 5th and 6th rib medially curves down and extends to 7th or 8th rib laterally to the anterior axillary line, mid point usually lies just behind the areola at a level of 6th rib in midclavicular line.[1] This crease is a defining structure of a developed breast and its robust anatomical presence has led to the basis of the classification of ptosis.[5] Anatomically, the crease is a microscopic structure due to condensation of superficial Camper's fascia and the deeper Scarpa's fascia.[6] Medial boundary of breast or fold has its origin from the lateral border of sternum and together with the contra lateral breast, forms the cleavage of the breast. Although an axillary tail of the breast gland extends into the medial wall of the axilla, the lateral extent of the breast is defined by the lateral breast fold, which in turn, should aesthetically and ideally be limited by a line drop from anterior axillary line. These three visible boundaries, in a natural looking and dependent breast are the parameters within which an aesthetic surgeon has to plan the surgery. These parameters also dictate the shape and size of the implant and the pocket in which the implant needs to be placed.

Arterial anatomy. Blood supply to the breast is through multiple sources and includes thoracoacromial axis, internal mammary, lateral thoracic and intercostals arteries and their perforators.[7] This rich arcade of blood supply make devascularistaion of breast envelope almost impossible even after extensive pocket dissection. However, the blood supply of the breast envelope is better when a plane is dissected in a submuscular plane as the pectoral perforator system, arising from internal mammary artery and thoracoacromial axis, remains undisturbed.[7]

Nerve supply. Nerve supply to the breast envelope is mainly from the 2nd to the 6th anterior and lateral cutaneous branches of the intercostals nerves. The nerve supply to the nipple areolar complex is from the anterior and lateral coetaneous branches of the 3rd

4th and 5th intercostals nerves.[8] The sensory changes to the breast and nipple areolar complex, therefore, varies with the approach used for the surgery and pocket selected for implant placement.

Examination:

Preoperative examination and plan of surgery is the most important aspect of augmentation mammoplasty. A thorough assessment, bilateral exchange of information, views and limitation of the procedure should take priority and proceedings documented at the same time. A pro forma detailing all aspect of examination, finding and plan of surgery is a useful adjunct. Envelope charecterstics, history of breast cup size changes in the past, asymmetries of breast, ribs, costal margin and sternum should be documented. Implant selection is the most important aspect of the consultation and a very well executed surgical technique can be futile if the base, profile and size of the implant is not corresponding and proportionate to the breast width, size and compliance of the available skin envelope. When there is an inadequate tight skin envelope, a disproportionate size of implant may be difficult to place and an inflatable implant can be reasonable option in these cases. On the other hand, a breast with small envelope with a history of change of breast size can allow and accommodate a larger implant. In authors experience 125cc to 150cc on a band size of 32 to 34, is sufficient to top up the breast cup size by one.

Breast Asymmetries:

Asymmetries of breasts can adversely affect the out come and involves breast volume difference, nipple level difference in vertical axis[9] or its placement in horizontal axis.[10] The disparities may also exist in nipple to inframammary crease distance, nipple areolar complex size or inframammary crease level. Associated ribs, chondrocostal or sternal deformities can also affect the outcome of augmentation mammoplasty. These asymmetries are common and an incidence of up to 87.8% have been reported.[9] In a prospective study performed by author,[11] a breast size difference was present in almost half of the patients, of these left side breast was larger twice as many times than on the right, nipple areolar level was different in one third of patients and left nipple areolar complex was twice as commonly lower than right. Similar observations were made when inframammary crease measurements were recorded and were twice as common on the left as on the right. Different degrees of chest asymmetries were present and again they were significantly commoner on the left side. However only minority of these patients needed a different size implant and even fewer needed unilateral mastopexy for nipple level correction in vertical plane. When nipple placement is asymmetrical in horizontal plane, it was more common on the right and author lateralises the pocket on the affected side to offset its appearance.[10]

Choice of incisions:

There are four incisions available to approach and dissect an implant pocket. The choice of incision is basically determined by the choice of the patient or surgeons experience. Most commonly used incisions are periareolar and submammary, both giving equally good access to all available pockets with good scar healing in vast majority of the patients. Submammary and areolar incisions can allow an adequate visual and tactile feed of a dissected pocket and inframammary crease repositioning in a hy-

poplastic breasts can be achieved with a low risk of implant malplacements. Axillary approach can be used in patients who are wishing to have their scars away from the breast in a well-concealed area and most, but the muscle splitting biplane pocket, can be accessed through this technique. However the risk of implant malplacement can be high and may require additional corrective procedure.[12] Endoscopic axillary pocket dissection and implant placement has made it possible to reduce the malplacement associated with blind dissection[13] which has a limited visual and tactile feed back.

Umblical approach has a single hidden incision for saline implant placement in subglandular plane. Rippling of the saline implant in subglandular pocket and lack of reported subpectoral pocket through this incision are the main reason that this approach is not widely used.[13]

Choice of available pockets:

Selection of implant pocket is an important aspect of the surgery. Anatomically breast is anterior to the muscle and ideally an implant should be placed in front of the muscle. This is the reason that first implant was placed in subglandular plane,[4] however an unacceptable rate of capsular contracture[14] led to the placement of implant completely behind the muscles.[15] This pocket did reduce the rate of capsular contracture but flat muscles is not able to give a natural three-dimensional result and also had a high surgical morbidity. The reduction of capsular contracture in submuscular pocket was recognised and less extensive muscle cover in the form of partial submuscular pocket was introduced.[16] This was a good marriage between subglandular and complete submuscular pockets that allowed the breast lower pole to expand naturally in a hypoplastic breast. The technique was adopted by many plastic surgeons, especially after the introduction of saline implant, where muscle cover is essential for more natural look and feel. Although a lot of patients did benefit from this new combination but patients with lower pole skin excess are unable to get adequate and satisfactory results. Inadequate communication between submuscular and subglandular pocket does not allow the implant to fill out the lower pole. To fill out the relative skin envelope excess, dual plane technique[17] is introduced in which varying degree of muscle is released anteriorly from the breast and posteriorly from the intercostals margin, depending on the skin envelope excess. This intentional release allows the muscle to shift superiorly allowing the implant to fill the lower pole adequately. However, when a muscle is released from its fixed bony margin and acquires its new attachment to the breast, a voluntary or involuntary movement of muscle pull the soft tissue of the breast resulting in dynamic breast deformity. These deformities are seen in vast majority of the patient to a varying degree.[18] Subfascial pocket[19] is another choice in which dissection under anterior pectoral fascia gives an additional layer to the breast envelope. The procedure has the advantage of excluding surgical morbidity associated with muscle release including dynamic deformities. A new submuscular pocket has been described in which upper part of the prosthesis lies in submuscular plane while lower part of the implant lies in subglandular plane.[3] This allows the implant to enhance or expand the lower pole of the breast uninhibited by the flat pectoralis. The pectoralis in this plane has a unique relationship to the implant and lies in front and behind the prosthesis at the same time. Muscle is split obliquely along its fleshy fibres, up and laterally, to the anterior axillary fold instead of cutting across its tendinous origin along the costal margin. The split along the fleshy fibres make it less painful procedure and patients have a quicker recovery. Muscle efficiency remains undisturbed due to lack of muscle release from the costal margin and its origin remains attached, preventing dynamic breast deformity.[3] Author has used the technique consecutively in over 800 bilateral primary augmentation mammoplasties alone, in last 3 years. The technique can be used to convert a partial submuscular pocket in to muscle splitting pocket, correcting dynamic muscle deformity.[20]

Antibiotic prophylaxis:

Despite the lack of scientific evidence and lack of controlled, prospective and randomised trial,[21] the use of prophylactic antibiotic has more than doubled in the last 40 years with out a significant reduction in infection.[22,23] Augmentation mammoplasty is considered as a clean procedure [24] but the use of a prosthesis[25] and presence of staphylococcus in majority of the nipple secretion,[26] antibiotic prophylaxis is considered a good clinical option in augmentation mammoplasty.[25]

An acceptably low infection rate with out antibiotic has been reported.[26] In another study, a comparative analysis between two groups, one having antibiotic prophylaxis and other not, showed no significant difference between the two groups.[27] In authors personal experience of 1628 primary augmentation mammoplasties, duration of antibiotic and infection rate was analysed. Three groups were identified, one group had a single intravenous dose, second had a single intravenous and 24 hour oral dose and third group had a single intravenous and a 5 days oral course of antibiotics. Infection rate was least in the group having single intravenous dose of antibiotic. This clearly shows that antibiotics does not only increase the cost of surgery to patient but prolonged use of prophylactic antibiotic does not give any added protection against infection and may result in complications including colitis, thrush, rash, gastrointestinal disturbances and may result in the emergence of resistant organism. In authors opinion a single dose of antibiotic is adequate for infection prophylaxis.

Complications:

Complications seen after augmentation mammoplasty are few and may present early or late.

1) Common early Complications.

Haematoma after augmentation mammoplasty is a known but uncommon complication. A large Danish study reported an incidence of 1.3% in 875 patients over a period of 20 years.[28] All had their implant placed in

submuscular plane. Author's retrospective analysis of ten years data, involving 1628 augmentation mammoplasties showed over all incidence of 1.7% haematoma. When incidence was looked into type of pocket used for augmentation mammoplasty, partial submuscular augmentation (107) had 1.8%, subglandular augmentation (774) had 3.3% and muscle splitting submuscular augmentation (747) had 0.1% incidence of haematoma respectively. All haematomas except one were treated with evacuation and almost all but two presented with in 24 hours after surgery. Evacuation of haematoma is recommended as the management of choice as organisation of blood can potentially contribute to capsular contractures. Implant can be reused after checking its integrity and washing with povidine iodine. In a late presentation of a nonexpanding haematoma, combination of compression bandages and ice packs can a useful alternative in selected cases.

Infection and its Management:

Periprosthetic infection after mammoplasty is not uncommon [26,28,29] despite the increase in the use of antibiotics.[22,23] These infections almost always present with classical symptoms of erythema, swelling, discharge and pain.[26] Early management is usually conservative,[26,30] but often requires a surgical intervention which can vary from removal of implant and replacement later[31] to capsulectomy, debridement, pulse lavage and implant placement as a single stage procedure.[30]

Minor wound healing problems may also present as superficial wound breakdown and includes stitch extrusion, small-localised stitch abscess or delayed healing. These superficial infections or wound healing problems can be twice as common as periprosthetic infection. These superficial wound needs to be distinguished from periprosthetic infections and conservative treatment of superficial infections almost always lead to a full recovery with out leading to capsular contracture, an often complication of periprosthetic infection. Author's management of infection usually commence with a wound swab for culture and sensitivity and commencement of antibiotic and daily dressing as an outpatient. Courtiss et al has reported 45% success rate using conservative treatment but nearly 1/3 developed capsular contracture later.[26] Usual organism is Staph aureus and antibiotics can be changed if other organism are isolated on microbial cultures. If the infection and discharge responds to antibiotic therapy and repeat culture shows eradication of the causative organism, an option of single stage implant replacement under general anaesthetic is considered. Old implants are removed, swab taken for culture and sensitivity, cavity thoroughly washed with povidine iodine and saline and new implants placed. A suction drain is used and oral course of antibiotic is continued postoperatively. No pulse lavage or debridement was performed before implant replacement. Infections resulting from atypical Mycobacteria may have all the hallmark of infection with negative routine bacteriological results and these infections are resistant to routine antibiotic treatment, Acid Fast Bacilli staining must be considered for an early antituberculous therapy.[32,33] Sterile Pus has been reported secondary to silicone implant rupture with out classical hallmark of infection or bacterial growth on microbiological cultures.[34,35] The sterile pus in these cases is a result of non-infectious inflammatory response to leaked silicone and present as an autoinflation of breast. This acute but progressive swelling act as a marker of implants rupture and lack of bacterial growth allows it to be managed as a single stage procedure.[34,35]

Mondors disease:

Mondors'disease or thrombophlebitis of thoracoabdominal axial veins is due to an interruption of venous blood flow. [36-38] Retrograde flow through collateral vein is prevented due to unidirectional valves in these axial veins. Organised blood in these vessels appears as painful fibrous cord usually referred as Bow String sign.[39]An incidence of 0.95% has been reported in oncologic breast surgery.[40] On the other hand, in augmentation mammoplasty carried out through inframammary incision, an incidence of 1.07% and 4.55% has been reported in symptomatic and asymptomatic cases respectively.[39,41] Mondors' disease following augmentation mammoplasty, using inframammary approach, affects the thoracoabdominal system of veins, however, the process may involve axillary veins and has been reported after axillary nodes dissection in a patient with silicone lymphadenitis.[41] Mondors' disease following augmentation mammoplasty using areolar, axillary or transumblical approach has not been reported. Regardless of the approach or distribution of the area of involvement, the process is self -limiting and usually disappears after 6-8 weeks without any adverse affects.

2) Common late complications:

Asymmetries
Dynamic Breasts
Implant rupture
Capsular contractures
Rippling
Flipping or malorientation of implants
Implant malplacements:

Capsular contracture and asymmetries due to implant malplacement are frequently seen complications following augmentation mammoplasty. Incidence of capsular contracture is far less common due to subpectoral position of the implant,[14] better sized implant pockets and better quality implants.[42,43] Revision surgery secondary to implant malplacement is the second most common cause.[12]

These implant malplacement may present as superior (high riding breast) medial (synmastia) lateral (telemastia) and inferior (Bottoming down) and can be unilateral, bilateral, asymmetrical, combination of two or can be secondary to capsular contractures.Bottoming down With continued rise in the absolute number of breast augmentation performed today, bottoming down is seen more commonly than before. It is considered the

commonest presentation of malplaced implant. Bottoming down or implant ptosis is viewed as progressive lengthening of distance between nipple and breast fold and is the most common form of the implant malplacement. This deformity is almost always due to the result of implant descent or its malplacement. In subglandular pocket, downward transgression of implant usually accompanies inframammary crease (IMC). Whereas bottoming down in submuscular pocket may manifest independent of IMC descent, presenting as double bubble deformity. The process is quite often due to oversize pocket dissection in the lower pole area of the breast.

Bottoming down can be unilateral, bilateral, asymmetrical and may present in combination with synmastia (medial malplacement) and telemastia (lateral malplacement). The complication can equally be seen with augmentation mastopexies or breast reconstructive surgeries. The process of bottoming down can be due to multiple reason can be the result of gravity, size of the implant, pocket used for its placement,[17,44] stretching of the thin envelope[45] stretching of scar in vertical scar mastopexy with mammoplasty, early and heavy pectoral exercises,[46] local steroids etc. Bottoming down can be a direct result of oversize pocket and is more often seen when inframammary crease is approached from a distant place through transaxillary approach [12] or can be due to disruption of the IMC in transumblical breast augmentation.[45] Distant approach for pocket dissection may produce a lack of tactile or visual feed back and probably is the reason for more common breast asymmetries or malplacements seen in later approaches. The complication can also be seen when over recruitment of skin is performed when planning a pocket, using an inframammary crease incision, in a hypoplastic breast.

To prevent malplacement, pocket markings and dissection should be planned and executed precisely in an augmentation mammoplasty regardless of the incision used.[44]

The surgical anatomy of the local region [6,47,48] can explain the complication when it manifest differently in the same pocket dissected through different approaches. Bottoming down and downward displacement of IMC and implant, following subglandular augmentation, is usually independent of nipple areolar complex and results in unusually high nipples. Corrective surgery is aimed at relocation the IMC at a higher place and treatment can be conservative or invasive and depends on the timing of diagnosis after mammoplasty.[12] Combined downward transgression of IMC and NAC require combined IMC relocation and mastopexy to maintain normal relationship between these two important anatomical landmarks. Higher relocation of IMC will result in induction of ptosis and mastopexy alone will result in worsening of bottoming down.

Bottoming down in submuscular pocket, however, can manifest with out lowering of the IMC and can be a direct result of over dissection in the lower pole, when performed through axillary approach.[12,45] Double bubble is the direct manifestation of the undisturbed infra mammary crease, where implant is covered by pectoralis and its extended fascia. [6,47-48] Treatment of bottoming down include conservative or minimally invasive,[12] multilayer capsuloraphy,[42] repair may require autologous local capsular flaps [45.49] or allogenic dermal grafts for skin support.[50] Author routinely change the pocket into muscle splitting biplane[2] along with multilayer capsuloraphy, if the bottoming down is seen following mammoplasty in subglandular pocket. The change of pocket helps to improve the upper pole aesthetic, conceal the pre-existing rippling, if present and addition of muscle layer helps to enhance the longevity to results.[3]

Synmastia:

Synmastia is a relatively uncommon form of implant malplacement seen following augmentation mammoplasty and is seen in subglandular as well as submuscular plane. The deformity is due to the confluence of medial part of the breast. This is generally due to over dissection in the medial aspect of the breast or can be the inappropriately larger size of the implant. The tenting of the medial aspect of the breast envelope over the sternum can be an early sign of the deformity.

The true incidence of synmastia is not known and quite often the patient is not aware of up the complication. These deformities may present individually, may present in a combination, can be unilateral or bilateral and are also seen when an implant pocket dissection is performed either for mastopexy or breast reconstruction.[42,51-54] The condition is quite often described along with other implant malplacement[42,52] and only handful articles are written on this complication.[51,53,54] Treatment of choice is capsuloraphy,[42,55] and Alloderm grafts can be added to reinforce capsuloraphy. [52,54]

Inflatable implants is another option to allow the repair to consolidate before the delayed expansion of the prosthesis.[53] Change of pocket into muscle splitting biplane[2] is an option[51] to correct synmastia following subglandular augmentation. Procedure allows the deeper unused subpectoral anatomy to recreate medial boundary of the pocket.[51] The procedure does not require capsuloraphy[51] and improves the breast aesthetic and longevity of the results at the same time. [2,3]

Synmastia has been classified into developmental, acquired or secondary capsular contractures.[51]

Telemastia:

Telemastia is the mirror image malplacment of synmastia and is more commonly seen in clinical practice than synmastia. Telemastia generally can be caused by over dissection of breast pocket in its lateral quadrant, inadequate muscle release of sternocostal pectoralis in partial submuscular plane or can be secondary to capsular contracture. Treatment or correction can be achieved with lateral capsuloraphy and medial mirror image Capsulotomy.[42,56]

Dynamic Breasts:

Dynamic breast or animation deformity is a common complication frequently seen after partial submuscular

and dual plane augmentation mammoplasty. The incidence is high and presents with varying degree.[18] The deformity is not seen when muscle is placed in subglandular[4] or subfascial [19] plane. Muscle splitting biplane augmentation is a submuscular choice with extremely low risk of dynamic deformities.[2] The pathophysiology of the process is due to the release of pectoralis from its fixed attachment on sternocostal margin. Muscle release results in loss of its specialised covering, epimysium and ability to glide under the skin with out pulling the overlying skin. When this part of the denuded muscle gains its new attachment to skin and breast parenchyma, it acquires the ability to pull or displace the breast, a process seen in domestic animals and is due to panniculus carnosus.[1,20] The resulting animation deformity may displace breast alone with or with out implant displacement. In an inadequate release of muscle, strong and long fibres of pectoralis are capable of displacing the implant[20,56] and widening of cleavage is seen without breast displacement. On the other hand, muscle release and its displacement from sterno costal margin to breast parenchyma and skin[20] or its attachment to the capsule of the implant[57] may results in glandular as well as implant displacement. The complication and process of animation deformity can be largely prevented by avoiding the muscle release as in submuscular muscle splitting augmentation or by placing the implant in prepectoral position, either in subfascial or subglandular plane. In an established deformity, changing implant from dual plane to subfascial plane[57] or changing partial submuscular to muscle splitting plane[20] has been documented to eliminate dynamic breast deformity.

Implant rupture:

Rupture of the implant can be due to biochemical degradation of the silicon, injury to the implantation during implantation, fold flaw failures, or as a result of mechanical injury including mammography and closed capsulotomies.[58] Estimates of breast implant rupture prevalence varies widely in different studies and ranges from 0.3% to 77%. This wide difference is due to the difference methods used.[59-61]

Institute of Medicine of America, in 1999, defined silicone breast implant rupture as a breach of any size in the implant shell and reported that all silicone gel implants were susceptible to silicon bleed through the implant shell.[62] Unlike saline implants, presence of the shell rupture does not allow the silicone prosthesis failure to be picked up easily. In majority of the cases, the rupture is intracapsular, where the silicone is retained with the fibrous capsule and no volumetric changes are seen. Silicone gel rupture is more likely to be symptomatic when the rupture is extracapsular where it has extended beyond the fibrous capsule. However silicon gel leak can only be confirmed after explantation of the prosthesis, a procedure only carried out in symptomatic patients. Quantitative data is lacking on the prevalence of silicone implant rupture. Several studies have been carried out since Institute of Medicine meeting in 1999 but to date only one study has been considered reliable prosthesis rupture prevalence.[60] Holmich et al, in their study, concluded that approximately2% and 15% of third-generation implants that are intact after 3 years can be expected to develop definite ruptures by 5 and 10 years, respectively.[60]

Marotta et al in their meta analysis have confirmed that there was a general reduction in tensile strength, tear strength, and elongation for all types of explanted elastomer shells, representing a marked loss in the strength and durability of the shells. These changes in the physical properties of the shell make it more vulnerable to the physical stresses experienced by the implant in vivo. This factor alone is responsible for marked increased in rupture rates of prosthesis with time due to weakened by silicone fluid-swollen silicon swollen shells,[61] a view not shared by Brandon et al.[63,64] Diagnosis of silicon gel implants rupture is not easy and clinical history and examination alone is not reliable. Marques et al in their study found that there were patients with less than five years implantation with tight capsular contracture unrelated to gross silicon leakage. There were also patients without significant contracture and leakage or ruptured implants.[65] It is quite common to have an asymptomatic silent ruptured implant and quite often there is no history of significant chest trauma.[66] Prevalence of rupture of an implant can be difficult to assess because same implant used by different surgeons gives different results while same surgeon has different results when using different implants. An ideal implant should be able to withstand an average force used by surgeons during its insertion. An adequate skin incision is mandatory for a cohesive gel implant placement, small incisions require more force to insert an implant and may lead to a higher incidence premature implant failure.[35]

MRI is the most commonly employed investigations to diagnose ruptured silicone gel implants. In surgically validated meta-analysis, MRI diagnoses of implant rupture, a sensitivity of 89% and a specificity of 97% with a positive predictive value of 99% has been reported.[59] MRI scanning has the ability to delineate water droplets, air bubbles or other trapped material mixing with the silicon gel with in the implant called Salad Oil Sign.[67] MRI in this patient would have been helpful to pick up intraprosthetic migration of sterile pus. Mammography also is frequently done to screen implant integrity, it is inexpensive, easily available. Although it can delineate extracapsular ruptures, procedure can be difficult to perform in painful breast with capsular contractures[67] and on its own can result in implant damage .[58,59] Ultrasonography is commonly performed to screen implant integrity but has little value however it can be complementary to the mammography. Stepladder sign seen in ultrasonography is considered a diagnostic sign of implant rupture and free or leaked silicon in breast tissue is visualised as Snow Storm appearance, similar low level echogenic appearances seen in axillary lymph node is diagnostic of silicon

lymphadenitis, a sign which can pick a silicon gel bleed in the absence of loss of implant shell integrity.[67] However the procedure is operator dependent and is unable to visualise the damage to the posterior aspect of the implant or posterior surface pectoralis silicon extravasation.[68]

Autoinflation:

Autoinflation of breast is an uncommon but important complication. Autoinflation can be intraprosthetic or extraprosthetic. Intraprosthetic migration resulting in autoinflation is commonly due to saline. Spontaneous autoinflation, in these implants is due to the large macromolecules crossing the implant shell possibly due to the osmotic difference of solutes across the implant shell.[69] Autoinflation of implant with sterile pus has been documented as well and can be associated with autoinflation of the breast with sterile pus.[34] The sterile pus is an inflammatory response to leaked silicone and can be a marker of silicone implant rupture.[35] Late autoinflation of breast with intracapsular seroma formation has been reported in silicon gel implants.[70]

Presence of intracapsular fluid is a common observation and has been reported to be present in 21% of patients in symptomatic augmentation mammoplasties. Symptoms were either local, presenting as breast pain or capsular contracture or general as fatigue, arthralgia and paraesthesia. Presence of fluid was not limited to any particular type of implant and was equally seen in textured silicone, textured polyurethane or smooth silicone implants with almost equal distribution. The amount of fluid aspirated in these patients was small and varied in its consistency from clear, turbid to xanthochromic.[71,72]

Rippling:

Rippling of an implant is quite a known complication seen after augmentation mammoplasty. Rippling has normally two components, visible and palpable. Visible component can be present in upright position and made worse on leaning forward or only present on leaning forward otherwise known as traction rippling. Palpable rippling can be present with or with out visible rippling. Both, visible and palpable rippling and their distribution on the breast, depends on the type of the implant, thickness of the available pocket, pocket used for the implant positioning and relative dimensions of the pocket and implant diameter. Physical features of the saline implants have made it more prone to rippling and submuscular positioning has allowed concealing this physical characteristic in the upper part of the breast. However, the rippling can still be present, with or with out traction, regardless of the type of implant, in the lower and outer pole of the breast. Thicker the envelope less is the rippling. Pinch test has been used to determine the positioning of a silicone gel implant and when there is an inch to pinch, subglandular positioning of these implants is considered adequate.[17] However, breast tissue, with all its three components of Skin, fat and parenchyma, is dynamic structure and all these vary from time to time in female. The common factors affecting these three components are, body weight changes, body fat changes, pregnancy and breast feeding, ageing. Implant placement itself stretches the envelope and continuous pressure on compressible breast envelope by a non-compressible implant thins out the breast envelope. Longer the implant in a breast or younger the age of the patient at the time of implantation, more breast envelope changes are expected. A combination of muscle and breast envelope makes it more resilient to the changes seen in a breast and submuscular positioning give longevity to the results.[2,3]

Capsular Contracture:

Capsular contracture following breast augmentation is still considered one of the commonest complications requiring medical and surgical attention. [14] Causes of capsular are multiple and quite perplexing and the overall reported incidence varies between 4 to 17% of the women.[73-75] With continued changes in implant characteristics including texturing of the implant and subpectoral placement, the contracture rate has been less frequently seen although up to 70% has been reported in subglandular plane alone.[76] Most of the studies are retrospective, the only prospective study where the capsular contracture was objectively assessed, an overall incidence of 4% of capsular contracture was reported with a follow-up ranging between 3 months to 4 years. Only one third of the patients were graded as Baker III and IV. Although the rate of capsular contracture has changed from First-generation to the current Fourth-generation implants, Henriksen et al studied the capsular contracture on the basis of the charecterstics of implants and characteristics of patients.[73] In general retropectoral implants placement has reduced the capsular contracture rate or perhaps intervening muscle may be acting as a buffer to conceal the degree of contracture or its external manifestation. Adequate size pocket also helps to reduce the clinical manifestation of capsular contracture. Early concentric forces exerted by capsular activity can be accommodated by a little larger pocket. As not all the capsular contractures proceed to Baker Class III or IV, an adequate or slightly larger pocket may allow the initial compressible forces exerted by the capsule to be absorbed by an incompressible implant. Haematoma is another known cause of capsular formation prevention of this surgical morbidity is absolutely essential. In an untreated haematoma or in a subclinical haematoma, blood gets organised and can contribute towards capsular formation. Insertion of a drain with an inadequate haemostasis will not prevent a haematoma or blood may continue to build once drains are removed and can lead to the process of capsular formation. The infection is another known factor leading to capsular formation, Staphylococcus aureus is the commonly isolated organism in periprosthetic infections.[26] However in an established capsular contracture, it is the staphylococcus epidermidis which has been found in the up to 87% of the culture of capsules, either alone or in combination.[77,78] The timing and mechanism of Staphylococcus epidermidis replacing Staphylococcus

Aureus and leading an infection to a capsular contracture, is not known. Intraluminal antibiotics have been used to prevent local infection and capsular contracture with a significant reduction of grade III and IV capsular contracture. However the sample was small with an average follow-up of 12 months and had a 37% class III and IV contracture in the control group.[78]

Treatment of choice of capsular contracture is an open procedure including capsulotomy or capsulectomy. Closed capsulotomy may result in an implant rupture and calcium deposition, when present, may assist this damage. High recurrence rate is also seen with closed capsulotomy and FDA does not recommend the procedure. High recurrence rates also are seen with capsulotomy and capsulectomy. Change of pocket from subglandular to submuscular muscle splitting biplane is routinely done by the author and conversion from submuscular to dual plane is another option.[79] Changing pocket provides the prosthesis new and fresh start, in a new environment and potentially reduces recurrence rate. Soft and thinner capsules can be left behind with reduced surgical morbidity.[80]

Implant stability:

Implants surface can be textured, micro texture or smooth. Implant texturing has been known to reduce capsular contracture. Polyurethane foam texturing enjoyed popularity in the 1970s due to their resistance to early capsular formation.[81] Even after their removal from the market the idea of texturing the implants continued.[82] Implant texturing can be in the form of depression or nodules and their physical characteristics depend on the diameter of the texture, pore depth, width and density. The presence of these physical charecterstics allows the fibrous capsule to grow into the pores of the implant surface and larger the diameter, more is the in growth of capsular tissue. In reality the pore sizes and texturing appears as a mirror image on interface of the capsule when seen under electron microscope.[83] This hand in glove fitting disallows micro movement of the implant with the capsular cavity. Smooth or micro textured surfaced implants do not adhere to capsule and continued micro movement results in synovial metaplasia.[84,85] The resultant metaplasia further discourages tissue adherence or capsular in growth into a micro textured or smooth surface of an implant. A smooth or micro textured surface implant, especially with lower gel fill ratio can flip or turn back to front once the skin envelope is stretched and relaxed postoperatively. Similar movement is seen in an over dissected pocket. Patients present with a disc shape base of the implant facing outward and round shape of the implant facing inward. The deformity requires an outpatient manipulation and in recurrent cases, pocket dimension needs to be reduced using multilayer capsuloraphy.

Autoimmunity, connective tissue disorders and carcinogenesis.

There is an abundance of literature about the association of silicone and systemic and localized autoimmunity and connective tissue disorders.[86] The case reports available in literature envisage links between silicone and a number disorders including, sceleroderma, rheumatoid arthritis, systemic lupus erythromatosus, depigmentation, Sjogren's syndrome, polymyositis etc. These disorders and their association with augmentation mammoplasty has been studied and there was any significant difference when compared with large population based control samples.[87-90] Our bodies are exposed to silicone as it is used in both personal and domestic products. Silicone from these sources is primarily absorbed from the skin and the gastrointestinal tract. As a trace element, silicone makes up part of the building blocks of skin and bone connective tissues, notably collagen,[91] and currently is commercially available as a nutritional supplement.[92] Several studies were conducted in the past to evaluate the potential risks and claims regarding the adverse effects of silicones on women receiving breast implants. These studies focused particularly on connective tissue diseases, and the results showed that none of these claims and risks were linked or associated with silicone breast implants. The Independent Review Group in England has reported that after gel bleed, small molecules of silicone are handled by the body in exactly the same way as silicone molecules from other sources. This report includes extensive research on all the relevant aspects of silicones related to the health of patients including its inflammatory and immunologic role.[93] Goldblum et al reported antisilicone antibodies to ventriculoperitoneal shunts, [94] but further studies by independent workers did not confirm the observation of antisilicone antibodies.[95] Wolf et al [96] showed that sera from patients with ruptured silicone breast implants had more circulating immunoglobulin G (IgG) than sera from patients with intact implants, diabetic women on insulin exposed to silicone in syringe lubricants, and healthy women with no silicone breast implants. The American Council of Science and Health later challenged these results in 1997.[97] It was concluded that current evidence does not support the claims that silicones themselves provoke antibody responses in vivo.[93] However, in animals, self-proteins absorbed to silicone polymers can induce an antibody response, and silicones may sometimes have a modest adjuvant effect on antibody production.[98] Nevertheless, there is no evidence that the response can cause tissue damage.[93] The Independent Review Group considered that more research was needed to verify the proliferation of lymphocytes against collagen types I and III, fibronectin, and fibrin in women with silicone gel breast implants. [99] The other available work done with adults and children demonstrating a proliferation of T lymphocytes [100,101] was poorly controlled, and the findings were reported in a way that made them difficult to interpret. [93] A Danish epidemiologic survey studied the effects of silicone gel implants on esophageal disorders and found a threefold excess in children born to mothers with breast implants. However, this excess was present in the chil-

dren
born before the implant surgery performed with this particular group of women.[102]

Large epidemiologic studies have studied the risk of cancer after breast augmentation and have shown that there was a smaller risk of incidence of breast cancer in patient with breast implant [103,104] and that blood from patient with augmentation mammoplasty has the ability to kill breast cancer cells in tissue cultures.[104] Similarly there was no difference in diagnosis and 5-year and 10-year survival rate in augmented and nonaugmented patients.[105] These studies do not imply that an augmentation mammoplasty can give some protection against breast cancer in women but clearly establishes that there is no association between silicone implants and breast cancer.

Minimally invasive and non-invasive options and future of breast augmentation surgery.

Autologous fat transfer of fat for breast enhancement has been described by Lexer in the literature going back to early twentieth century[106] and was revisited by Mel Bircoll[107] in 1985 but was soon deplored by American Society of Plastic and Reconstructive Surgeons in 1987.[108] The committee decision put a halt on the procedure and the option was tabooed to be reviewed for a long time. It was not until Coleman revisited the fat grafting to the breast in 2007.[109] Improvement in techniques involving harvest, preparation and injection of fat gave a new direction to the use autologous fat transfers to breasts. Adipose-derived mesenchymal stem cell as a regenerating agent and its clinical application in breast surgery grafts has given a huge impetus to its application and broadening our understanding of this fascinating concept.[110] However the results are quite operator dependent and complications are not uncommon.[111] Lack of availability of the donor sites and limited improvement in breast cup size in a single session has not given it a wider acceptance as an alternative to breast implants.

Stabilized hyaluronic acid of nonanimal origin (NASHA™ gel; Q-Med AB, Uppsala, Sweden) has been in used for facial filler and wrinkles treatments for sometime, Its wide popularity has made it possible to potentially use it as an option for breast enlargement. The available product is marketed as Macrolene™ (Q-Med AB) and was approved in Europe in 2006 and is available in two volume restoration factors, VRF20 and VRF30, and both have received CE mark. The product gives temporary enhancement and the reported bilateral mean injection, per patient, was 211 ml. Though it gives a good options to patients not keen on implant surgery, however, limited improvement in cup size, its temporary nature and high post injection inflammatory reactions are limiting factors.[112]

but the safety of the breast implants, nature of the procedure with persistent good results associated with a satisfied patient make it almost impossible that implant surgery will be replaced in the foreseeable near future.

The tissues ability to grow when subjected to sustained, low-level mechanical distraction has given a new dimension to breast augmentation surgery. The concept is applied to augment and enhance breast size. This nonsurgical non-pharmaceutical external device has been studied, clinically applied and is a fascinating development that gives an added choice to patients who are not keen on invasive or minimally invasive procedure available for breast enhancement.[113] However the limitation of enhancement is the limiting factor. To conclude, the safety and choices of implant shape and sizes and the techniques available, make the implant surgery the most sought after option for breast enhancement surgery in the foreseeable future.

References:

1. Last RJ (1978) Anatomy regional and applied. 6th ed. Churchill Livingstone: Edinburgh, pp. 65-66
2. Khan U D: Muscle Splitting Biplane Breast Augmentation. Aesth Plast Surg 31:353-358, 2007
3. Khan U D: Selection of Breast Pocket Using Pinch Test in Augmentation Mammoplasty: Can it be Relied in the Long Term? Aesth Plast Surg. Online First (In Press)
4. Cronin, T. D. and Gerow, R. M. Augmentation Mammoplasty: New "Natural Feel" Prosthesis. In translation of the third International Congress of the Plastic Surgery. Amsterdam: Excerpta Medica, 1964. pp. 41-49. Excerpta Medica International Congress Series, No. 66
5. Regnault P: Breast Ptosis: Definition and Treatment. Clin Plast Surg 91:657-62, 1976
6. Muntan CD, Sundine MJ, Rink RD, Acland RD: Inframammary Fold: A Histologic Reappraisal. Plast Reconstr Surg 105: 549-56, 2000
7. Cormack GC, Lamberty BGH (1994) The Arterial Anatomy of Skin Flaps 2nd ed. Churchill Livingstone: Edinburgh, pp 168
8. Schlenz I, Kuzbari R, Gruber H, Holle J. The Sensitivity of the Nipple-Areola Complex: An Anatomic Study. Plast Reconstr Surg. 105:905-909, 2000
9. Rohrich RJ, Hartley W, Brown S. Incidence of breast and chest wall asymmetry in breast augmentation: A retrospective analysis of 100 patients. Plast Reconstr Surg 115:1039-1050, 2005
10. Khan UD. Breast Augmentation in Asymmetrically Placed Nipple Areolar Complex in Horizontal Axis: Lateralisation of Implant Pocket to Offset lateralised Nipples. Aesth Plast Surg.33:591-596.2009
11. Khan UD. Incidence of Common Breast and Chest Asymmetries in Augmentation Mammoplasty. 7th Croatian Congress of Platic, Reconstructive and Aesthetic Surgery with International participation. Split, Croatia, October 01-05, 2008. BOA pp 54.

12. Troilius C: Correction of Implant Ptosis after a Transaxillary Subpectoral Breast Augmentation. Plast Reconstr Surg 98:889-95,1996Troilius C: Correction of Implant Ptosis after a Transaxillary Subpectoral Breast Augmentation. Plast Reconstr Surg 98:889-95, 1996

13. Grab and Smith's (1997) Plastic Surgery. Fifth Edition. Lippincott-Raven: Philadelphia, pp715

14. Biggs T M., Yarish R S: Augmentation Mammoplasty: A Comparative Analysis. Plast. Reconstr. Surg. 85:368, 1990

15. Dempsey W C, Latham WD. Subpectoral implants in Augmentation Mammoplasty: A preliminary report. Plast Reconstr Surg. 42:515, 1968.

16. Regnault, P., Partially submuscular breast augmentation. Plast. Reconstr. Surg. 59: 72, 1977

17. Tebbetts JB: Dual-plane breast augmentation: Optimizing implant soft tissue relationship in a wide range of breast types. Plast Reconstr Surg 107:1255, 2001

18. Spear LS, Schwartz J, Dayan JH, Clemens MW. Outcome Assessment of Breast Distortion Following Submuscular Breast Augmentation. Aesth Plast Surg. 33:44-48, 2009

19. Graf R M., Bernardes A, Rippel R, Araujo LRR. Damasio RCC,

Auersvald, A. Subfascial Breast Implant: A new Procedure. Plast. Reconstr. Surg. 111:904,2003.

20. Khan UD. Dynamic Breasts: A Common Complication Following Partial Submuscular Augmentation and its Correction Using Muscle Splitting Biplane Technique. Aesth Plast Surg. 33:353 -360.2009

21. Rohrich RJ, Rios JL. The role of prophylactic antibiotics in

plastic surgery: whom are we treating? Plast Reconstr Surg 86:617-618, 2003

22. Krizek TJ, Koss N, Robson MC. The current use of prophylactic

antibiotics in plastic and reconstructive surgery. Plast Reconstruct Surg 55:21-32, 1975

23. Lyle WG, Outlaw K, Krizek TJ, Koss N, Payne WG, Robson MC.

Prophylactic Antibiotics in Plastic Surgery: Trends of Use Over 25 Years of an Evolving Specialty. Aesth Surg J 23:177-183, 2003

24. Perrotti JA, Castor SA, Perez PC, Zine JE. Antibiotic use in aesthetic surgery: a national survey and literature review. Plast Reconstr Surg 109:1685-1693, 2001

25. Wolfe A, Podds S. Prophylactic Antibiotics in Aesthetic Surgery.

Aesth Surg J 26:93-94, 2006

26. Courtiss EH, Goldwyn RM, Anastasi GW. The fate of breast

implants with infection around them. Plast Reconstr Surg;63:812-816. 1979

27. Leroy J, Given KS. Wound infectionin breast augmentation: the role of prophylactic perioperative antibiotics. Aesth Plast Surg 15:303-305. 1991

28. Kjoller K et al. Epidemiological Investigation of Local Complications After Cosmetic Breast Implant Surgery in Denmark. Ann Plast Surg : 48;229-237. 2002

29. Cholnoky T. Augmentation mammoplasty: survey of complications in 10,941 patients by 265 surgeons. Plast Reconstr Surg 45:573. 1970.

30. Spear SL, Howard MA, Boehmler JH, Ducic I, Low M, Abbruzzesse MR. The Infected or Exposed Breast Implant: Management and Treatment Strategies. Plast Reconstr Surg 113:1634-1644. 2004

31. Cronin TD, Greenberg RL. Our experience with the silastic gel breast prosthesis. Plast Reconstr Surg 46:1-7. 1970

32. Macadam SA, Mehling BM, Fanning A, Dufton JA, Kowalewska- Grochowska KT, Lennox P, Anzarut A, Rodrigues M. Nontuberculous mycobacterial breast implant infections. Plast Reconstr Surg. 119:337-44.2007

33. Heistein JB, Mangino JE, Ruberg RL, Bergese JJ. A prosthetic breast implant infected with mycobacterium fortuitum. Ann Plast Surg. 44:330- 3.2000

34. Khan UD. Unilateral Breast Autoinflation and Intraprosthetic Collection of Sterile Pus. An Unusual Operative Finding of Silicon Gel Bleed With Silicon Lymphadenitis. Aesth Plast Surg. 32:684-687. 2008

35. Khan UD. Breast Autoinflation with Sterile Pus as a Marker of Implant Rupture: Single-Stage Treatment and Outcome for Five Consecutive Cases. Aesth Plast Surg. 33:58-65.2009

36. Fagge CH (quoted by Herman JB): Trombophlebitits of breast and contiguous thoracoabdominal wall (Mondors' disease). NY State J Med 66:3146-3152, Dec 15.1966

37. Mondor H: Tronculite sous-cutanee subaigue de la paroi thoracigue antero-laterale. Mem Acad Chir. 65: 1271-8. 1939

38. Weinstein EC: Mondors' disease, West J Med 123:56 -57. 1975

39. Khan UD: Incidence of Mondor Disease in Breast Augmentation: A retrospective Study of 2052 Breasts Using Inframammary Incision. Plast Reoconstr Surg 122:88-89e Viewpoints 2008

40. Hou MF, Huang CJ, Huang YS, Hsieh JS, Chan HM, Wang JY, Chuang CH, Chen FM, Huang TJ: Mon-

dors' disease in the breast. Kaoshing J Med Sci. 15 (11): 632-9. 1999

41. Khan UD. Mondors' Disease: A review of the literature and a case report. Aesth Surg Jour.29:209-212.2009

42. Spears SL, Little JW, III. Breast Capsulorraphy. Plast. Reconstr. Surg. 81: 274-79, 1988

43. Millan Mateo J, Vaquero Perez: Innovative New Concepts in Augmentative Breast Surgery. PartII: Systematic and Drawing. Aesth Plast Surg 25:436-442, 2001

44. Khan U. D: Lower Pole Enhancement in Breast Augmentation. 6th Croatian Congress of Plastic, Reconstructive and Aesthetic Surgery, Opatja-Rijeka, Croatia. 2006, BOA pp 63

45. Rowe NM, Freund R: The autologous Internal Breast Splint: A Novel Technique for the Treatment of Postaugmentation Ptosis: Aesth Surg 25:545-664,2005

46. Clarendon CCD: Implant Descent: A Complication of Augmentation Mammoplasty and Its Correction. Ann Plast Surg. 21:452-7. 1988

47. Barnett A: Transaxillary Subpectoral Augmentation in the Ptotic Breast: Augmentation by Disruption of the Extended Pectoral Fascia and Parenchymal Sweep. Plast Reconstr Surg 86:76-83, 1990

48. Tebbetts JB: Transaxillary Subpectoral Augmentation Mammoplasty: Long Term Follow-Up and Refinements. Plast Reconstr Surg 74:636-47,1984

49. Parsa FD: Breast Capsulopexy for Capsular Ptosis after Augmentation Mammoplasty. Plast Reconstr. Surg. 85:809-12, 1990

50. Baxter RA: Intracapsular Allogenic Dermal Grafts for Breast Implant-Related Problems. Plast Reconstr Surg. 112:1692-1696, 2003

51. Khan UD. Correction of Acquired Synmastia Following Subglandular Mammoplasty Using Submuscular Muscle Splitting Biplane Technique. Aesth Plast Surg. 33:605-610.2009

52. Baxter RA: Intracapsular Allogenic Dermal Grafts for Breast Implant-Related Problems. Plast Reconstr Surg . 112:1692-1696, 2003

53. Becker H, Shaw KE, Kara M. Correction of Synmastia using an adjustable implant. Plast Reconstruct Surg. 115:2124-2126. 2005

54. Spear SL, Bogue DP, Thomassen JM. Synmastia after breast augmentation. Plast. Reconstr. Surg. 118: 168S-171S, 2006

55. Chasen PE, Francis CS. Capsuloraphy for Revisionary Breast Surgery. Aesth Surg J; 28:63-69.2008

56. Ganong WF: review of Medical Physiology. 13th ed. Appleton and Lang, San Mateo, California, 1987

57. Baxter RA, Subfascial Breast Augmentation: Theme and Variation. Aesth Surg J; 25:447-453.2005

58. McLaughlin JK, Lipworth L, Murphy DK, Walker PS. The Safety of silicon Gel- Filled Breast Implants: A Review of the Epidemiologic Evidence. Ann Plast Surg 59:569-580. 2007

59. Holmich LB, Jon F, Kjoller K, Breiting VB, Jorgensen A, Krag C, McLaughlin JK. The Diagnosis of Silicon Breast-Implant Rupture: Clinical Findings Compare With Findings as Magnetic Resonance Imaging. Ann Plast Surg 54:583-589.2005

60. Holmich LB, Friis S, Fryzek JP, Vejborg IM, Conrad C, Sletting S, Kjoller K, McLaughlin JK, Olsen JH. Incidence of Silicon Breast Implant Rupture. Arch Surg.138:801-06.2003

61. Marotta J, Goldberg EP, Habel MB, Amery DP, Martin PJ, Urbaniak Dj, Widenhouse CW. Silicon Gel Breast Implant Failure: Evaluation of Properties of Shells and Gels for Explanted Prosthesis and Meta-analysis of Literature Rupture Data. Ann Plast Surg 49:227-247. 2002

62. Bomdurant S, Ernster V, Herdman R. Safety of Silicon Breast Implants: Report of the Committee on the safety of Silicon Breast Implants(IOM). Washington, DC: National Academy Press; 1999.

63. Brandon HJ, Young VL, Jerina KL, Wolf CJ. Scanning Electron Microscopy Characterization of Surgical Instrument Damage to Breast Implants. Plast Reconstr Surg 108:52-61.2001

64. Brandon HJ, Taylor ML, Powell TE, Walker PS. Microscopy Analysis of Breast Implant Rupture Caused by Surgical Instrument Damage: Aesthetic Surg J. 27:239-256.2007

65. Marques A, Brenda E, Amarante MTJ, Pereira D, Castro M, Haddad A. Findings in the removal of silicon gel breast implants: a correlation between time of implantation and rupture. Eur J Plast Surg 20:15-18.1997

66. Anderson B, Alani H, Kapetansky D, Hawtof D. The diagnosis of Ruptured Breast Implants. Plast Reconstr Surg 84:903-07 1989

67. Weekman WH, Van Sraalen WR, Hage JJ, Taets van Amerongen AHM, Mulder JW. Imaging Signs and Radiologists' Jargon of Ruptured Breast Implants. Plast Reconstr Surg 102:1281-89. 1998

68. Ahn CY, DeBruhl ND, Gorczyca DP, Shaw WW, Bassett LW. Comparative silicon breast implant evaluation using mammography, sonography, and magnetic resonance imaging: Experience with 59 implants. Plast Reconstr. Surg. 94:620.1994

69. Robinson jr OG, Benos DJ, Mazzochi C. Spontaneous Autoinfaltion of Saline Mammary Implants: Further Studies. Aesthetic Surg J. 25:582-586.2005

70. Tatla T, Ho-Asjoe M, Shibu M. Sudden onset breast swelling: an unusual presentation of ruptured prosthesis 10 years post-implantation. Eur J Plast Surg.27:391-393.2005

71. CY Ann, CY Ko, Wager EA, RS Wong, Shah WW. Clinical significance of intracapsular fluid in patients' breast implants. Ann Plast Surg. 35:455-7.1995

72. CY Ann, CY Ko, Wager EA, RS Wong, Shah WW. Microbial evaluation: 139 implants removed from symptomatic patients. Plast. Reconstr. Surg. 98:1225-9.1996

73. Henriksen TF, Fryzek JP, Holmich LR, McLaughlin JK, KjollerK, et al. Surgical Intervention and Capsular Contracture After Breast Augmentation: A Prospective Study of Risk Factors. Ann Plast Surg.54:343-351. 2005

74. Cunnigham BL,Lokeb A, Gutowski KA. Slaine-filled breast implant safety and efficacy: a multicentre retrospective review. Plast Reconstr Surg. 105:2142-2149. 2000

75. Kjoller K, Holmich LR,Jacobson PH, et al. Capsular contracture after cosmetic breast implant surgery in Denmark. Ann Plast Surg. 47:357-366. 2001

76. Sculy SJ. Augmentation mammoplasty without contracture. Ann Plast Surg. 6:262. 1981

77. Burkhardt BR, Dempsey PD, Schnur PL et al. Capsular contracture: A prospective study of the effect of local antibacterial agent. Plast Reconstr Surg. 77:919. 1986

78. Burkhardt BR, Fried M, SchnurPL, Tofield JJ. Capsules , Infection, and Intraluminal Antibiotics. Plast. Reconstr. Surg.68:43. 1981

79) Spear SL, Carter ME, Ganz JC. The Correction of Capsular Contracture by Conversion to "Dual-Plane" Positioning: Technique and Outcomes. Plast Reconstr Surg. 112:456-466. 2003

80. Chiat N Baran et al. A Different Strategy in the Surgical Treatment of Capsular Contracture: Leave Capsule Intact. Aesth Plast Surg. 25:427-431. 2001

81. Ashley FL. A new type of breast prosthesis: Preliminary report. Plast Reconstr Surg 45:421,1970

82. Hester TR, Jr., Nahi F, Bostwick J, Cukic J. A 5-year experience with polyurethane covered mammary prosthesis for treatment of capsular contracture, primary augmentation mammoplasty and breast reconstruction. Clin Plast Surg 15:569, 1988

83. Danino et al. Comparison of the Capsular Response to the Biocell RTV and Mentor 1600 Siltex Breast Implant Surface Texturing: A Scanning ElectronMicroscopy Study. Plast Reconstr Surg 108:2047-2052, 2001

84. Raso DS, Grene WB, Metcalf JS. Synovial metaplasia of periprosthetic breast capsule. Arch Pathol Lab Med. 118:249,1994

85. Del Rosario AD, Bui HX, Petrocine S et al. True synovial metaplasia of breast implant capsules: A light and electron microscopic study. Ultrastruct Pathol. 19:83, 1995

86. Khan UD. Bilateral Areolar Depigmentation After AugmentationMammoplasty: A Case Report and Literature Search. Aesth Plast Surg 32:143–146. 2008

87. Brody GS, Conway DP, Deapan DM et al. Consencus statement on the relationship of breast implants to connective tissue disorders. Plast Reconstr Surg. 9:1102. 1992

88. Gabriel SE, O'Fallen WM, Kurland LT et al. Risk of connective tissue disease and other disorders after breast implantation. N Eng J Med. 326:1697. 1994

89. Sanchez-Guerro J, Schur PH, Sergent JS, Liang MH. Silicone breast implants and rheumatic disease; Clinical, immunologic and epidemiologic studies. Arthritis Rheum. 37:158. 1994

90. Sanchez Guerrero J, Colditz GA, Karlson EW et al. Silicone breast implants and the risk of connective tissue diseases and symptoms. N. Engl J Med. 332:1666. 1995

91. Schwarz K. A bound form of silicon in glycosaminoglycans and polyuronides. Proc Natl Acad Sci USA 70:1608–1612. 1973

92. Clouatre D, Connolly KM: BioSil: Collagen and the health of the skin and bones. Total health: Special report, April 2006

93. The Independent Review Group, Rogers J, et al: Silicone gel breast implants: The Report of the Independent Review Group. Retrieved at http://www.silicone-review.gov.uk/index.htm. 1998

94. Goldblum RM, Pyron D, Shenoy M Modulation of IgG binding to silicones by human albumin. FASEB J 9:A1029,(Abstract 5967). 1995

95. Rohrich RJ, Hollier LH, Robinson JB. Determining the safety of the silicone envelope: In search of a silicone antibody. Plast Reconstr Surg 98:455–458.1996

96. Wolf LE, Lappe M, Peterson RD, Ezraison EG. Human immune response to polydimethylsiloxane (silicone): Screening studies in a breast implant population. FASEB J 7:1265–1268. 1993

97. American Council on Science and Health. Silicone breast implants: Why has science been ignored? ACSH, New York. 1996

98) Schaefer CJ, Whalen JD, Knapp T, Wooley PH . The influence of silicone implantation in type II collagen induced arthritis in mice. Arthritis Rheum 40:1064-1072. 1997

99. Ellis TM, Hardt NS, Campbell L, Piacentini DA, Atkinson M. Cellular immune reactivities in women with

silicone breast implants: A preliminary investigation. Ann Allergy Asthma Immunol 79:151–154. 1997.

100. Smalley DL, Shanklin DR, Hall MF, Stevens MV, Hanissian AImmunologic stimulation of T lymphocytes by silica after use of silicone mammary implants. FASEB J 9:424–427. 1995
101. Smalley DL, Levine JJ, Shnklin DR, Hall MF, Stevens MV: Lymphocytes response to silica among offspring of silicon breast implant recipients. Immunobiology 196:567–574, 1996/1997
102. Kjoller K, Friis S, Signorello LB, McLaughlin JK, Blot WJ, Lipworth L, Mellemkjaer L, Winther JF, Olsen JH. Health outcome in offspring of Danish mother with cosmetic breast implants. Ann Plast Surg 48:2.2002
103. Berkel H, Birdsell DC, Jenkins M. Breast augmentation: A risk factor for breast cancer? N Engl J Med.326:1649,1992
104. Brody G, Silicone implants and the inhibition of cancer (discussion) Plast Reconstr Surg .96@519,1995
105. Birdsell DC, Jenkins H, Berkel H. Breast Cancer Diagnosis and survival in women with and with out breast implants. Plast Reconstr Surg 92:795,1993
106. Hinderer UT, Del Rio JL. Erich Lexer's mammoplasty. Aesth. Plast. Surg. 16:101, 1992
107. Bircoll M. Cosmetic breast augmentation utilizing autologous fat and liposuction techniques. Plast. Reconstr. Surg. 18:327, 1987
108. ASPRS Ad-Hoc Committee on New Procedures. Report on autologous fat transplantation, September 30,1987.
109. Coleman RC, Saboeiro AP. Fat Grafting to the Breast Revisited: Safety and Efficacy. Plast. Reconstr. Surg.119:775-785, 2007.
110. Rigotti G, Marchi A, Sharbati A. Adipose-Derived Mesenchymal Stem Cells: Past, Present, and Future. Aesth. Plast. Surg. 33:271-273.2009.
111. Hyakusoku H et-al. Complications after Autologous Fat injection to the Breast. Plast. Reconstr. Surg. 123:360-370.2009.
112. Heden P et-al. Body Shapping and Volume Restoration: The Role of Hyaluronic Acid. Aesth. Plast. Surg.33:274-282,2009.
113. Khouri R, Schlenz I, Murphy BJ, Baker TJ. Nonsurgical Breast Enlargement Using an External Soft-Tissue Expansion System. Plast. Reconstr. Surg. 105:2500-2512,2000.