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Ottawa 6/18/2010

Ms. Inez P. Petersen

[redacted]

Our File No: 103961

By Mail

Dear Ms. Petersen

I refer to your fax communication of June 10, 2010.

As requested, a copy of the Modified ID Report I prepared, dated July 24, 2007, is provided. In keeping with your request, copies of all correspondence between us are also provided. This includes letters, briefings, specimen transmittal information, continuity of custody documentation, etc.

The health problems you are experiencing may not be unusual amongst long term saline implant users because of the propensity of these devices to undergo elastomer degradation. Nearly all users of such implants show the phenomenon to a certain degree. The attached briefing may give you some insight.

An invoice is attached to cover photocopying and postage expenses.

Yours truly,

P. Blais, Ph.D., F.C.I.C.

Attachments

FIBROSIS AND CHRONIC TISSUE INJURIES FROM DEGRADING SALINE IMPLANTS

Implants Cause Fibrosis:

Injuries from breast implants are not strictly a matter of systemic toxic insult, sometimes termed as 'silicone poisoning'. Adverse effects are more often consequences of a cascade of events initiated by an implant 'being there' and not being well tolerated by its surroundings. A key source of implant problems is the response of the surrounding tissue to the surface of the implant. This takes the form of a fibrous membrane composed largely of long collagen fibers. Depending on implant composition, shape, configuration as well as amount and type of material originating from the implant site, membranes can evolve at different rates and can acquire a range of properties that impact systemically on the user. With time, the newly-formed fibrotic tissue matures and its properties change. As it forms, debris and soluble material released from the implant become incorporated within the membrane, sometimes accumulating within small pockets that become surrounded by secondary fibrotic membranes.

Through mechanical action such as tension, erosion, compression, patient movement, microbiological action or osmotic pressure buildup, the membrane remodels continuously with new layers being added on the external surface. Conversely, the inside layer nearest to the implant undergoes compaction, attrition and occlusion with deposited substances. The capsule loses cellular activity as viable cells diminish in number and eventually disappear. Dehydration begins, followed by tissue shrinkage and finally necrosis. Calcification is the last step where highly organized crystalline plaques resembling bony material form on the inner capsular surface ultimately lining the interface between the implant and the capsule.

Within the first few hours following insertion of an implant, a process leading to the formation of the connective tissue capsule begins. Initially very thin and rarely more than a tenth of a millimeter and highly fluid-permeable, the capsule may stabilize in that condition. This situation is generally believed to be normal. However, it may also continue to evolve into a more complex structure under less favorable conditions. High quality orthopedic implants habitually form very thin elastic capsules that allow unimpeded flow of fluids. Poorly engineered implants, in particular implants based on commercial silicone technology, do not have the same progression of events. Impurities within elastomers, which include inflammatory substances such as silica, metal oxide fillers and adulterated oils, elicit a continuous response which causes the capsule to thicken, sometimes reaching several millimeters in thickness. Even without frank failure of the implant, thick capsules are the norm for long term implant users.

The intracapsular space, protected by a thick capsule composed of dense collagen fibers, is not permeable to the same degree as other biological tissue. Thus, the intracapsular space for breast implants is rarely well irrigated by extracellular fluids. It collects debris of synthetic and natural origin that would otherwise be entrained and ultimately excreted through natural fluid transport processes. For improperly fabricated implants with large quantities of effluents, as is the case for most commercial breast implants, it is an area which becomes debris-laden.

The space between the implant and the tissue gradually fills with stagnant body fluids and soluble implant impurities. These ferment with time, producing other chemical substances which can impart chronic adverse health effects of their own. The problem is similar to having a foreign body-contaminated abscess or hematoma for a long time. If rupture or gross leakage of semi-fluids takes place from a breast implant site, this fluid floods into the intracapsular space forming a dispersion or emulsion with aqueous capsular fluids already present.

Breast implants are frequently removed at this stage as a result of local or systemic manifestations that attract medical attention. Upon opening capsules, surgeons note that a paste-like substance occupies the intracapsular space. Examination of the paste reveals an elevated content of mineralized particles, in particular poorly-soluble calcium salts. The calcific material causes the pH to rise dramatically, frequently initiating alkaline hydrolysis of silicones used in breast implants. There is direct attack of silicone elastomers as evidenced by surface degradation and pitting in nearly all implants which have been in situ for more than about 10-12 years and where the implant site has sustained heavy mineralization.

The pasty, alkali-rich mixture is abrasive. With user movement, it easily erodes through implant shells causing comminution of the shell surface with release of surface debris. It also contributes to the surface attrition process of the tissue capsule, increasing the content of finely-divided denatured tissue within the capsular space. This mixture is ideally suited for many kinds of chemical reactions, all of which have potential for adverse effects on users. It is the nidus of alkali-based tissue and silicone denaturation. It frequently becomes colonized with micro-organisms which further diversify the chemical mixture.

Water-soluble, low molecular weight substances are formed in these mixtures and become potentially bioavailable. Perversely, the capsule which, at its peak maturity, was impermeable to fluids, becomes permeable as the degradation processes advance. If the user is subjected to procedures to control capsular contracture, such as compression capsulotomy, the integrity of the capsule is lost. As this takes place, the content of the intracapsular space is dispersed throughout the user's chest and must undergo disposal through conventional excretion routes, processes ill-suited for cleansing of poorly-soluble bioactive material. Large quantities of mineralized crystals are entrained in extracellular fluids, ultimately lodging into the lymphatic system and in organs responsible for excretion or metabolization of toxins. The most frequent demonstration of this process is palpable lymphadenopathy proximal to the implant site or markedly radiodense lymph nodes in the breast and axillary area.

This worsens the situation of stagnant body fluids by scattering the material in multiple sites thus enlarging the surface area of the reaction mixture. The situation occasionally progresses to loculated pockets surrounded by membranes of their own. With time, these sites multiply through break-up and fibrosis which produce porous, sponge-like composites of tissue and debris, termed 'granulomata'. Ongoing processes within granulomata, consisting of finely-divided pockets of heterogeneous fluids, accelerate the deterioration process and the diversity of components within these mixtures of decaying body fluid and alkali-laden masses. These processes have similarities to advanced situations of tuberculosis where mixtures of microbiological debris becomes intermingled in fibrous tissue to form large, solid masses.

For the fluid part of the prosthetic mixture which was originally dispersed as an emulsion and which was released slowly into the extracapsular area, there is no simple mechanism for excretion. The process of elimination is largely dependent on sequestration within fibrotic masses and transport to remote sites where other metabolic routes prevail to facilitate excretion. The dispersed globules, termed 'micelles', can be conveyed far from the implant site. Studies on microvasculature of users demonstrate extensive migration of elastomer particles which have the capacity to coat the lining of fine blood vessels. Some scientific circles perceive such globules as potentially antigenic or vaccine-like entities when in conjunction with endogenous substances or bacterial debris that occur naturally within living organisms. Such micelles are potential initiators and accelerators of abnormal immunity-related phenomena.

Implants Have Distal and Systemic Adverse Effects With Serologic Markers:
Distal adverse effects are nearly inevitable with time. Dysfunction may take place through frank occlusion of the circulatory and lymphatic systems by prosthetic debris or secondary fibrosis affecting fluid channels. Alternately, adverse effects may occur through weak antigens potentiated by denatured tissue proteins or immunoreactive polymer surfaces. Surface-active agents with a capacity to potentiate weak antigens, termed 'adjuvants', have been used in vaccine fabrication for more than half a century. Silica surfaces also initiate antigenic effects. Degraded silica-rich polymers of the kind used to make breast implant shells form such surfaces. Immunochemical aspects of prosthetic substances are reflected in many articles, for example: D. Radford Shanklin, D.L. Smalley; Pathogenetic and Diagnostic Aspects of Siliconosis; Reviews Environ. Health, 17, 1 (2002). get copy of this

Breast implants are associated with serologically-discernible lipid abnormalities. Implant patients preferentially show large quantities of dispersed semi-solid, fatty substances in the bloodstream, a condition termed 'chylomicronaemia' and suggestive of disturbances in fat metabolism and lipid transport, as described in W. Wall, L. Martin, M.J. Fritzler, S. Edworthy; Non-Fasting Chylomicronaemia In Breast Implant Patients; Lancet, 345, 1380 (1995). get copy

Target organs most vulnerable to these phenomena include the liver, brain, kidney and other highly irrigated organs subject to occlusion and cell destruction. The lymphatic system is also targeted as it is interspersed with nodes designed to capture solid particles to facilitate metabolization in a form better suited for excretion. All of these phenomena are encountered in long term breast implant users. Confirmation of these effects through pathological studies on animals are published. To expect a user to remain free of such adverse consequences is inconsistent with basic anatomic, physiologic and clinical knowledge.

38 years

Upper Respiratory Problems from Fibrosis:
Lung problems in long term implant users are frequently encountered by individuals who had implants that released debris. The phenomena appears partly related to implants which cause or accelerate fibrosis and formation of tissue. Calcific deposits within capsules are another marker for such problems. Calcification of capsules appears to play a role either as an initiator or as a symptom of underlying problems. Implants with fabric fixation patches, popular in the sixties and seventies, caused proliferative fibrosis (hyperplasia) with extracapsular adhesions, frequently extending several centimeters beyond the capsules. They also accelerated calcification. Calcification causes continuous trauma to the area by damaging small blood vessels with release of blood products.

Fibrosis and adhesions involving the lining of the lung and sometimes the lung itself are encountered, both in implant users and in individuals who have had chest surgery and exposure to substances that cause fibrosis. Examples include workers involved with asbestos, quartz, coal, paint pigments as well as individuals who had fibrosis-inducing drugs. This subpopulation includes victims of war injuries, adventitious projectiles and firearms.

Analogies With Naturally-Occurring Phenomena:
The analogy between long term implant-related problems and consequences of natural diseases that cause scarring and traumatic injuries of the chest are well known to the medical community. Implant-related problems have been treated continuously from the introduction of breast implant technology in the fifties. Large upper chest implants, such as breast implants, violate basic anatomic considerations. It is inevitable that users will encounter major problems over time and that these problems resemble those of trauma patients.

From review of patient histories and on considerations based on the study of tissue and the interaction between tissue and prosthetic material, there is no basis to believe in early resolution of deeply-rooted problems such as inflammatory processes and fibrosis-related lung complications. The belief that such problems have slow and gradual resolution is a logical expectation on the basis of expertise acquired from the treatment of military and occupational injuries. The experience from the treatment of tuberculosis preceding and following WWII is also significant. Lung injuries from release of fibrosis-inducing substances, such as material released from breast implants or created by interaction of breast implant surfaces and surrounding tissue, would be similar to what occurs following tuberculosis and lung infections from other classes of bacterial and fungal pathogens. Tuberculosis is no longer a common disease in Europe and North America. Much of the medical treatment expertise in dealing with its long term consequences (fibrosis) developed as a result of the resurgence of cases following the end of World War II.

Problems associated with upper chest infections, long term impact of large abscesses and other diseases associated with prolonged dwell time of encapsulated hematomas, fluid pockets and reactive foreign objects, were of interest to military physicians during World War II. Experiments to improve field injury management were conducted by the Allies, the Soviets and the Fascists. This information is now largely forgotten, some of it embedded in war archives and in testimony in post-war trials on unethical treatment of prisoners.

Valuable information can be derived from pre World War II anecdotal studies and from records of wartime medical investigations on consequences of implanting foreign objects and performing procedures that led to large abscess-like structures. This data supports the belief that there can be long term systemic health consequences from abscesses and that sequelae can extend beyond resolution of local problems they engender. Such knowledge has not been utilized significantly in connection with treatment of breast implant complications. Rapidly escalating usage of implants of all types with rise in complications requires review and reconsideration of this knowledge to understand the limitations of implant technologies and their long term risks.

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