ORIGINAL ARTICLE

Adjuvant Breast Disease: An Evaluation of 100 Symptomatic Women with Breast Implants or Silicone Fluid Injections

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(Received for publication on December 7, 1993)

Abstract. We evaluated 100 referred women with breast implants ($n = 97$) or silicone fluid injections ($n = 3$) into breasts who developed various symptoms. All reported symptoms occurred at a median latency period of 6 years (range 0–24 years) after implantation or injection of silicone. Commonest symptoms were weakness (95%), fatigability (95%), myalgia (90%), morning stiffness (89%), arthralgia (81%), memory loss (81%), sensory loss (77%), headache (73%) and dry eyes and dry mouth (72%). Laboratory results revealed abnormal levels of serum immunoglobulins or complement in 57% and autoantibodies in 78%. Sural nerve biopsy was abnormal in 80% with the major finding of loss of myelinated fibers in 79%. Biceps muscle biopsy was abnormal in 58% with the major finding of neurogenic atrophy in 27%. Ninety-six patients underwent implant removal; 60% of the patients were found to have one or both implants ruptured with silicone spilled into tissue. At time of removal, a pectoralis major muscle biopsy was taken which was abnormal in 89% with the major finding of neurogenic atrophy in 55%. Biopsy of implant capsule was abnormal in 94% showing foreign body giant cells containing refractile material consistent with silicone in 69% whether or not the elastomer shell was ruptured. Silicone can cause a systemic autoimmune disease with a variety of symptoms probably due to a global activation of the immune system. Since our patients had objective laboratory and histologic findings together with a high rate of mechanical implant failure, further investigations are necessary. (Keio J Med 43 (2): 79–87, June 1994)

Key words: implants, disease, autoimmunity, biopsy

Introduction

Health professionals, federal officials and patients have raised numerous questions about the safety and efficacy of breast implants because they have been associated with a number of local and systemic complications such as capsular contracture, implant rupture and gel migration, gel bleeding, granuloma formation, lymphadenopathy, infection, interference with tumor detection and rheumatologic and systemic autoimmune diseases.^{1–25} Dow Corning Corporation, who has been the biggest implant manufacturer until it quit the implant business in 1992, announced on March 19, 1993, that their silicone-gel had been found to be a strong irritant of the immune system.²⁶ Since approximately one million women in the United States have already received silicone breast implants,²⁷ the topic is of concern to the medical community as well as to the public.

We have now (December 1993) evaluated around 1500 women who developed an autoimmune disease

associated with breast implants. The purpose of this study was to find out whether women with breast implants or silicone fluid injections and symptoms have objective laboratory and histologic findings.

Patients and Methods

The patients are the first 100 consecutively presenting women with silicone breast implants or silicone fluid injections who were referred to our service for evaluation of their symptoms between 1985 and July, 1992. All patients developed the symptoms reported here after receiving silicone breast implants or silicone fluid injection.

Each patient received an evaluation consisting of history and physical examination including breast examination and neurological examination. We sent a questionnaire inquiring about the patient's implant history and the patient's symptoms to each patient. Every patient was seen in consultation by an outside plastic surgeon or

by a plastic surgeon at Baylor College of Medicine. All patients underwent quantitative measurements of immunoglobulin IgG, IgM, IgA and complement C3 and C4 by immune precipitation using rate-nephelometry, but results were only available in 76 patients. Ninety-three patients were tested for antinuclear antibodies (ANA) by indirect immunofluorescence, 92 patients for antimyelin associated glycoprotein antibodies (anti-MAG) by ELISA, 90 patients for rheumatoid factor (RF) by Latex agglutination, 88 for antiganglioside M1 antibodies (anti-GM1) by ELISA²⁸ and 79 patients for antisulfatide antibodies by ELISA.

Eighty-four patients received an MRI of the brain, 28 patients underwent a spinal tap with analysis of cells, protein, glucose and electrophoresis, 93 patients underwent EMG and studies of nerve conduction velocity, 20 patients had visual evoked responses measured, 32 patients had a xeromammogram of the breast, 40 patients had an ultrasound examination of the breast and 45 patients had an MRI of the breast.

Sixty-six patients underwent a biopsy of the left sural nerve and 93 patients underwent a biopsy of the left biceps muscle. Ninety-six patients underwent removal of the implants and the surrounding implant capsule (open capsulectomy). At time of implant removal, 90 patients had a biopsy of the implant capsule and 71 patients had a biopsy of the pectoralis major muscle taken. Frozen sections of muscle tissue were processed for staining using NADH-tetrazolium reductase, myofibrillar ATPase at pH 9.4 and modified Gomori's trichrome. Frozen sections of nerve biopsy tissue were stained with H&E, modified Gomori's trichrome and crystal violet. Formalin sections of the implant capsule were stained with H&E.

A Kaplan-Meier²⁹ estimated survival curve of discovered time to implant rupture was plotted without regard to type of implant to characterize the time to implant failure.

Results

Patients demographics

The median age of the patients at the time of first implantation of silicone breast implants or silicone fluid injections was 32 years (range 19–52 years). The median age of onset of clinical symptoms was 38 years (range 23–57 years). The median latency period between insertion of silicone implants or injection of silicone fluid and development of clinical symptoms was 6 years (range 0–24 years).

Sixty-eight patients had received silicone breast implants ($n = 65$) or silicone fluid injections ($n = 3$) for cosmetic purpose. Thirty-two patients had received silicone breast implants for reconstruction of the breast

after mastectomy for either fibrocystic disease ($n = 28$) or breast cancer ($n = 4$).

Fifty-six patients had received silicone-gel filled breast implants, 20 patients had silicone-gel filled breast implants coated with polyurethane, 17 patients had double-lumen silicone breast implants and 4 patients had saline filled silicone breast implants. Three patients had silicone fluid injections into breast. Two of these later received silicone-gel breast implants. Forty-one patients had received more than one pair of breast implants due to local complications of the initial breast implants.

Local problems with the implants

Seventy-six patients experienced local problems with their breasts such as capsular contracture, tenderness, soreness or pain of the breasts, hot and swollen breasts, infections, numbness of the nipples or discharge from the nipples. The commonest local problem was capsular contracture, which was present in 67 patients and belonged to Baker classification III or IV.

Clinical symptoms

Initial symptoms were morning stiffness, skin rash, myalgia, arthralgia and easy fatigability followed by progressive weakness. Initially, these nonspecific symptoms were generally attributed to the effects of stress or a psychological disturbance. All patients presented with at least 20 to 30 symptoms. Some patients developed severe myalgia, weakness and fatigue which rendered them bedridden. The commonest rash was a livedo reticularis involving the lower more than the upper extremities and tended to become confluent over the knees and elbows and a rash in the "V" shape of the neck. Other rashes included erythematous reactions in face, trunk and extremities (Fig 1). Punch biopsies of the skin showed usually nonspecific inflammatory changes consistent with collagen vascular disease which could not be further classified. Fifty-eight patients developed Raynaud's phenomena with one patient's finger progressing into gangrene (Fig 2). The little finger in this patient had to be amputated. The patients' symptoms upon presentation to our service are summarized in Table 1. The patients' findings on neurological examination are summarized in Table 2.

Based on the history and the neurological examination, 83 patients were found to have a polyneuropathy syndrome, 10 patients a multiple sclerosis-like syndrome which was accompanied by a polyneuropathy in 8, 5 patients a motor neuron disease syndrome and 2 patients myasthenia gravis. Patients with multiple sclerosis-like syndrome or motor neuron disease syndrome had criteria for the diagnosis of multiple sclerosis or motor neuron

Table 1 Commonest Symptoms

Number of Patients	100
Weakness	95
Fatigue	95
Muscle Aches and Pain	91
Morning Stiffness	89
Joint Pain	81
Memory Problems	81
Numbness	77
Headache	74
Dry Eyes, Dry Mouth, Dry Vagina (Sicca Complex)	72
Vision Problems	72
Tingling	72
Sweats	68
Gel-phenomena	63
Loss of Balance	63
Shortness of Breath	63
Rash	62
Joint Swelling	58
Enlarged Lymph Nodes	58
Raynaud's Phenomena	58
Allergies	52
Dizziness	52
Swallowing Problems	52
Low Grade Temperature	52
Nausea	48

disease, but there were also other findings in these patients such as myalgia, arthralgia, joint swelling, rashes, dry eyes and dry mouth (Sicca-Complex), Raynaud's phenomena, low grade fevers or lymphadenopathy. Eight of the patients with multiple sclerosis-like disease had additional findings of polyneuropathy. Five patients died during the study. Three patients with motor neuron disease syndrome died (one 7, two 8 years after the onset of symptoms) of respiratory failure. One of them had an autopsy and was found to have demyelination of the lateral columns and loss of anterior horn cells compatible with motor neuron disease. However, a biopsy performed when she initially presented revealed segmental demyelination in sural nerve; a sensory nerve. This finding was confirmed by the pathologist at time of autopsy. One patient died of renal failure and 1 of congestive heart failure. Both of the latter had evidence of systemic autoimmune disease with involvement of organs and polyneuropathy. The patient that died of congestive heart failure had a unique implant complication 2 years prior to her death. During a blood transfusion into her right subclavian vein, the needle went accidentally into her right silicone-gel breast implant. After inflation with a unit of blood, the implant ruptured, silicone-gel spilled in the abdominal tissues and the tissues of the thigh on the right side. She then developed sclerosis with progressive fibrosis of the skin in the distribution of the spilled gel. Over the ensuing 2 years, the sclerosis spread concentrically from the area of spill

Table 2 Findings on Neurological Examination

Number of Patients	100
Weakness	94
Loss of Vibration	78
Loss of Pin-prick	72
Trigrooved Tongue	20
Facial Weakness	19
Increased DTR	14
Increased Muscle Tone Lower Extremities	13
Babinski's Sign	12
Decreased DTR	11
Ptosis	11
Muscle Atrophy	4
Cerebellar Findings	4
Fasciculations	3

Table 3 Measurements of Immunoglobulins (IgG, IgA, IgM) and Complements (C3, C4) Levels in Serum

Number of Patients for Whom Results Were Available	76 (100%)
Patients with Abnormal Immunoglobulin or Complement Levels	43 (57%)
Decreased Immunoglobulins	24 (32%)
Increased Immunoglobulins	13 (17%)
Decreased Complements	14 (18%)
Increased Complements	9 (12%)

Table 4 Antibodies

Patients Who Had Autodirected Antibodies	78/100 (78%)
Anti-GM1 Antibodies	36/88 (41%)
Antinuclear Antibodies	33/93 (36%)
Anti-MAG Antibodies	28/92 (31%)
Antisulfatide Antibodies	22/79 (28%)
Rheumatoid Factor	10/90 (11%)

to encompass the circumference around her body like a cuirass. Dermatologists and internists considered her to have progressive systemic sclerosis. The gangrene of the breast required mastectomy on the right.

Laboratory results

Fifty-seven percent of the patients had abnormal levels of quantitative immunoglobulins and complement C3 and C4 (Table 3). Seventy-eight percent of the patients were found to have autodirected antibodies (Table 4). The cy/MAG and cy/GM1 ratios (ratio of IgM antibody titer/antibody titer to Histone H3)²⁸ were elevated in most patients.

Eighty-four patients underwent an MRI of the brain. Nineteen patients had multiple white matter lesions (9 of

Fig 1 Erythematous rash in a patient with double-lumen breast implants.

Fig 2 Gangrene of the right little finger in a patient with silicone-gel breast implants.

them had multiple sclerosis-like syndrome), 13 patients had multiple small ischemic lesions and the remaining 52 patients had a normal MRI of the brain. Twenty-eight patients underwent a lumbar puncture. Thirteen patients were found to have oligoclonal bands (10 of them had multiple sclerosis-like syndrome), 6 patients had increased gammaglobulin, 5 patients had increased total protein, 4 patients had an increased IgG synthesis rate, 1 had decreased protein and 2 patients had a normal CSF analysis. EMG with nerve conduction velocity (NCV) studies were done in 93 patients. Twenty-four had findings of small, short, low amplitude motor unit potentials on intramuscular recordings commonly referred to as myopathic potentials, but also seen in nerve terminal twig disease, 23 patients had evidence of denervation

Fig 3 Xeromammogram of the breast showing rupture of a silicone-gel breast implant (arrow 1), and free silicone in the milk ducts (arrow 2).

Fig 4 T2-weighted MRI of the chest showing a ruptured and fragmented silicone-gel breast implant with body fluid collection inside the implant (arrow). (1.5 Tesla, TR 900 msec, TE 24, slice thickness 6 mm, matrix 256 × 176)

Fig 5 Implant capsule biopsy: Large area of chronic inflammation with fibrous tissue (HE, 42 $\times$).

Fig 6 Implant capsule biopsy: Foreign body giant cells with phagocytized refractile foreign bodies consistent with silicone (HE, 105 $\times$).

Fig 7 Pectoralis major muscle biopsy: Myositis; large area of chronic inflammation (modified trichrome on a frozen section, 42 $\times$).

Fig 8 Pectoralis muscle biopsy: Foreign material consistent with free silicone and chronic inflammation (modified trichrome on a frozen section, 105 $\times$).

Fig 9 Intraoperative picture of the removal of a ruptured silicone-gel breast implant.

Fig 10 Fragments from the removal of a ruptured silicone-gel breast implant.

Fig 11 Severe degradation of polyurethane of a silicone-gel polyurethane covered breast implant.

with polyphasia, giant motor units, fibrillations, or decreased recruitment, 11 patients had findings of carpal tunnel syndrome, 4 patients had findings of an axonal neuropathy and the 2 patients with myasthenia gravis had a decrement of greater than 8% between the 1st and 4th response on repetitive stimulation. The remaining 44 patients had normal NCV and EMG. Twenty patients underwent measurements of visual evoked responses and 11 patients were found to have delayed responses (8 bilaterally, 2 on the right and 1 on the left, out of them 10 patients had multiple sclerosis-like syndrome).

Thirty-two patients had a xeromammogram of the breasts. Six patients showed findings of fibrocystic disease, 6 patients showed calcification of the implants and the implant capsule, 3 patients showed lymphadenopathy with silicone uptake, 2 patients showed evidence of implant rupture, 1 patient showed ruptured implants and

free silicone in the milk ducts (Fig 3) and 15 patients had a normal mammogram. Forty patients had an ultrasound of the breasts. Eight patients showed evidence of implant rupture, 6 patients showed free silicone in the surrounding tissue, 5 patients had lymphadenopathy, 3 patients showed fibrocystic disease and 22 patients had a normal ultrasound. Forty-five patients had an MRI of the breasts. Eleven patients showed evidence of implant rupture (Fig 4), 9 patients showed free silicone in the surrounding tissue, 1 patient with previous silicone injections showed free silicone fluid in tissue and 21 patients had a normal MRI.

Histological findings

Eighty percent of the patients had an abnormal sural nerve biopsy with the major finding of loss of myelinated fibers in 79%. Most patients had moderate loss of myelinated fibers (estimated loss of 35–45%). Fifty-eight percent of the patients had an abnormal biceps muscle biopsy with the major finding of neurogenic atrophy in 27%.

Ninety-four of the patients had an abnormal implant capsule biopsy with findings of foamy histiocytes, macrophages or lymphocytes in 81% and foreign body giant cells sometimes containing silicone in 69% (Table 5). Figure 5 shows an area of chronic inflammation in a capsule biopsy. Figure 6 shows foreign body cells with phagocytized silicone. Eighty-nine percent of the patients had an abnormal pectoralis major muscle biopsy with the major finding of neurogenic atrophy in 55%. Figure 7 shows the findings of chronic inflammation, rounding of muscle fibers and increased connective tissue as seen in myositis. Figure 8 shows free silicone that had spilled into the pectoralis major muscle on pectoralis major muscle biopsy.

Implant removal

Ninety-six patients underwent removal of the implants and the surrounding fibrous implant capsules. The median age of the patients at removal was 44 years (range 30–59 years). Fifty-seven patients (60%), of whom 41 had more than one pair of implants, were found to have

Fig 12 Survival curve based on a Kaplan-Meier estimated survival curve of discovered time to implant rupture. This survival curve presents the cumulative proportion of implants discovered to be intact versus time to removal.

ruptured implants with spilled silicone into tissue (Fig 9). Figure 10 shows the removed fragments of a ruptured silicone-gel implant. Figure 11 shows severe degradation of the polyurethane of a silicone-gel polyurethane covered breast implant. This patient who had previous augmentation of her breasts was found to have breast cancer at time of implant removal.

Implant survival analysis

The Kaplan-Meier estimated survival function shown in Fig 12 has a median of 14 years regardless of type of implant. The relationship between time to removal and the cumulative proportion of intact implants is almost linear. The minimum and maximum removal times for which implant failure was discovered were one third of a year and 28 years. These data clearly demonstrate that implants rupture as they age.

Discussion

The goal of this study was to find out whether patients with silicone breast implants or silicone injections and symptoms have objective laboratory findings. The result of this study is that the patients who presented to us with symptoms after receiving silicone breast implants or silicone fluid injections had objective findings on physical examination, laboratory testing and tissue pathology. Fifty-seven percent of the patients had abnormal levels of immunoglobulins or complements (Table 3), 78% of the patients had autodirected antibodies (Table 4), 80% of the patients had an abnormal sural nerve biopsy,

Table 5 Findings on Implant Capsule Biopsy

Number of Patients Who Had Biopsy Taken	90 (100%)
Abnormal Biopsy	85 (94%)
Foamy Histiocytes, Foamy Macrophages or Lymphocytes	73 (81%)
Foreign Body Giant Cells Sometimes Containing Silicone or Polyurethane	62 (69%)
Free Silicone or Polyurethane	59 (66%)

58% of the patients had an abnormal biceps muscle biopsy, 94% of the patients had an abnormal implant capsule biopsy (Table 5) and 89% of the patients had an abnormal pectoralis major muscle biopsy. Patients also presented with a high number of local problems of the breasts (76%) and were found to have an unusual high incidence of implant rupture (60%).

All of our patients presented with a variety of rheumatological and nonspecific symptoms as listed in Table 1 in addition to the neurological symptoms. Only 36% of our patients had positive ANA (most of them had a low titer) and only 11% had positive RF. When in addition other antibodies such as anti-GM1, antisulfatide or anti-MAG antibodies were measured, a total of 78% of the patients had autodirected antibodies. Almost all patients with idiopathic systemic lupus erythematosus have a positive ANA³⁰ and about 70% of patients with idiopathic rheumatoid arthritis have a positive RF.³⁰ Thus, routine laboratory tests for rheumatic diseases such as ANA or RF were often negative, but other laboratory tests and tissue biopsies showed abnormalities in a high number of patients. Therefore, our patients did have objective findings, but the disease characteristics and laboratory features differed from idiopathic rheumatological or neurological diseases. All of our patients reported at least 20 to 30 different symptoms (Table 1). The symptoms of patients with idiopathic diseases are usually more circumscribed. Hence, our patients developed a syndrome of neurological, rheumatological and non-specific signs and symptoms that was diagnosed with "Adjuvant Breast Disease" as first described by Miyoshi *et al* in 1964.³¹ An adjuvant is a substance that enhances or changes immune system responses.

Bridges *et al*²² reported 156 women with silicone breast implants who had developed atypical rheumatic disease. In their study, only 9% had tested positive for RF and only 22% had tested positive for ANA. They concluded that women with silicone breast implants may develop atypical rheumatic diseases which differ from idiopathic diseases. Love *et al*²³ reached the same conclusion. They investigated 13 patients who developed myositis after receiving silicone breast implants and found as well that their clinical and immunogenetic features differ from idiopathic myositis. Freundlich *et al*²⁴ reported 24 patients with silicone breast implants who developed atypical Sjogren's-like syndrome with dry eyes and dry mouth, adenopathy and glandular mononuclear cell infiltrates in the absence of serological findings.

Controlled epidemiologic studies about the occurrence of rheumatological and neurological symptoms in women with breast implants don't exist yet. It is not possible in our opinion to compare the incidence of the atypical disease (Adjuvant Breast Disease) patients with breast implants tend to develop with the known incidence of idiopathic rheumatological or neurological disease in the

general population, since patients with breast implants seem not to develop idiopathic diseases. We believe our patients are presenting with a new disease called "Adjuvant Breast Disease".

Because many women with breast implants and symptoms currently seek medical attention, we can not just wait starting to treat these women until the final proof of a cause and effect relationship between silicone breast implants or silicone fluid injections and disease is delivered. Regardless of cause, these women are ill and need medical attention. If we assume for a moment that there is not enough evidence of a cause and effect relationship between silicone breast implants and diseases, how shall the patient with breast implants and autoimmune disease be managed? One answer could be to disregard the implants and just to treat the disease with anti-inflammatory or immunosuppressive drugs. The other answer could be to remove first the foreign body and then, if necessary, treat the patient with additional medications because other foreign bodies and chemicals such as silica (up to 30% in the gel and the envelope of silicone breast implants) have been associated with the development of rheumatological and systemic autoimmune diseases in the past^{32,33} and there is no proof that silicone breast implants could not do the same. The implant manufacturer Dow Corning recommends in their current package insert³⁴ that if an immune response in a patient is suspected and the response persists, the prosthesis and the surrounding capsule tissue should be removed and such patients should not be reimplanted.

It is also important to investigate the patients' implant condition because a ruptured implant alone, whether the woman has symptoms or not, is, and has always been, an indication for implant removal because of the known adverse effects of free silicone in tissue. We believe the surrounding implant capsule should be removed together en-bloc (as a single unit) with the breast implants because our findings and those of other clinicians^{5–7} show that silicone leaks in the surrounding tissue whether or not the implants were ruptured. Surgeons should make every effort to remove as much spilled silicone and surrounding capsule tissue as possible in order to increase the chance of improvement in women with symptoms and to diminish adverse effects of free silicone in tissue.

It has also been suggested that native proteins may be denatured by interaction with the silicone surface and the products of this interaction may be a target of an immune response.^{21,35} The immune response at this point would still be normal because damaged proteins present with different antigenic character and are physiologically recognized as foreign and therefore "cleaned out of the system". The immune system might get out of control down the line confusing damaged proteins with normal proteins and may start a cross-reaction towards the normal tissue resulting in a pathological autoimmune

reaction. This might be the case in our patients here, since 78% of our patients had autodirected antibodies. Thus, our patients might have developed an autodirected immune response triggered by the foreign material in their bodies. The symptoms and tissue abnormalities in our patients therefore might be due to indirect autoimmune mechanisms rather than direct effects of silicone. Since our patients had a long list of different symptoms, a rather diffuse activation of the immune system might have occurred. Also, the elevation of the cy/MAG and cy/GM1 ratios as seen in most patients indicating polyclonal antibody reactivity shows diffuse activation of the immune system.

Symptoms developed in our patients after a median latency period of 6 years between implantation or injection of silicone and development of first clinical symptoms. Sixty percent of the patients were found to have ruptured implants. To investigate the life expectancy of implants, we presented the status of the implants at time of removal in Fig 12 as a survival curve. As expected, the passage of time presages implant ruptures and the relationship between time to removal and implant status is almost linear. Hence, failure rate appears to be a constant function of time. The half life of an implant was estimated from this data to be 14.0 years. However, a 14 year half life is an overestimation, since time to failure is always less than removal time. The implant failure rate observed in our patients was 37.1%. This rate is similar to the one reported by Robinson *et al*.³⁶ Breast implants tend to degrade causing implant rupture. Patients therefore need to be aware that surgeries to remove ruptured implants and spills of free silicone into tissue are likely to be necessary.

DeCamara *et al*³⁷ had a similar observation. They found that the percentage of ruptured implants increased dramatically after 7 years and virtually all implants that were more than 10 years old were ruptured or leaking. Their data also suggests that silicone implants were likely to break as they age, regardless of whether a woman experienced trauma.³⁷ The House of Representatives Subcommittee on government operations analyzed 700 randomly selected letters of women who wrote to Chairman Weiss.²⁷ The women with problems tended to have had implants for 5–10 years or even longer. This is consistent with other experts' findings and our own findings that women with symptoms have implants that ruptured 7 years or more after implantation. We suggest that the time of onset of clinical symptoms might correlate with the degradation of the device in form of implant rupture or major silicone leak. However, we observed a broad range in the latency period of 0 to 24 years. The fact that our patients had received various kinds of implants from different manufacturers which exposed them to different chemicals and the interpersonal variability of the immune system might be responsible for the

broad range in the latency period.

In view of the accumulating evidence, we suggest silicone breast implants and silicone fluid injections can provoke an autoimmune disease (Adjuvant Breast Disease) with a variety of symptoms probably due to a global activation of the immune system. The disease characteristics and laboratory features differ from idiopathic diseases. Because of the presence of objective abnormalities and a high rate of implant failure in our series, symptoms in women with silicone breast implants or silicone injections should not be neglected nor underestimated even if routine laboratory tests are normal. Physicians should be aware of an atypical disease in women with silicone breast implants. It is also important to examine the patient's breast implants thoroughly because an implant rupture might be present in patients with silicone breast implants and symptoms.

Acknowledgements: The study was supported by Mr George Lindler, a retired Houston builder. We thank the Department of Pathology at the Methodist Hospital for processing and examining removed breast implants and recuts of the implant capsules, and Dr David Netscher and Dr Steven Hamilton, Department of Plastic Surgery, for giving us pictures of removed implants. None of the authors have any financial interest concerning breast implants.

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