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The effect of late infection and antibiotic treatment on capsular contracture in silicone breast implants: A rat model

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Summary *Background:* The effect of late infection on capsular contracture has yet to be established, leaving a gap in clinical guidelines for the treatment patients with breast implants. This trial is the first to assess if the treatment of these infections can reverse this effect in an in vivo rat model and whether late distant infections increase the incidence of capsular contracture.
Materials and Methods: Three groups of female Wistar rats ($n = 42$) received two silicone implants in separate dorsal, subcutaneous pockets. All groups except control underwent injection of a human strain of methicillin-sensitive *Staphylococcus aureus* (MSSA) at least 30 days after implantation, allowing for physiologic capsule formation. The infection group received a peritoneal injection, inducing a transient bacteremia, the treated group received a course of antibiotics following bacterial inoculation, and a final group received no intervention and served as control.
Results: Implants were removed 4 months after insertion, and capsules measured for thickness and sent for bacterial quantification. Compared to both the control and treated groups, capsule thickness in the infection group was statistically greater ($p < 0.05$), a difference not observed between treated and control groups. In addition, a statistically significant positive correlation was found between capsule thickness and bacterial count ($R = 0.614$, $p < 0.01$).
Conclusions: The difference in thickness between the control capsules and those from the infection group is an indication that bacterial contamination of a capsule from a remote late infection may increase the incidence of capsular contracture suggesting that treating late infections could in fact prevent capsular contracture.

Summary: This study presents a rat model aiming to demonstrate how late infections can affect the development of capsular contracture of silicone implants and how systemic

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antibiotic therapy may be useful to revert such situation.
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Introduction

In recent years, research has established a definitive link between infection and the development of capsular contracture around silicone breast implants.[1–5] During surgery, the implant may be contaminated with common skin flora such as *Staphylococcus epidermidis,* resulting in subclinical infection and biofilm formation. This method makes a low-virulence bacteria previously thought to be non-pathogenic capable of inducing the most significant complication of breast implantation, with no way of recognizing the threat until it is too late for the patient.[6,7] Preventative treatments for early infections of this kind have been shown to dramatically reduce the incidence of capsular contracture in animal models.[8–10] In addition, the development of silicone implants designed to disrupt biofilm formation and increase biocompatibility is also being investigated.[11]

While strides continue to be made to combat these types of early infections and the resulting complications, late infections are an entirely different story. In vivo models of capsular contracture and infection have focused on the capsular response to bacteria introduced at the time of implantation as described previously. However, capsular contracture continues to occur months or even years after implantation with no sign of biofilm formation and no other apparent cause. Various case reports have described late infections of silicone implants resulting from bacteria translocation from a distant foci of infection.[12–15] Up until this point though, no research has been carried out in an attempt to identify a link between a remote infection established well after implantation, the hematogenous spread of bacteria to a capsule and capsular contracture. A late infection originating in this fashion, like the early subclinical infections now known to significantly increase the risk of contracture, could also induce an accelerated or prolonged inflammatory process even if not acutely evident.

The aim of this study was to establish a rat model of late distant infection resulting in transient bacteremia, allowing us to investigate the potential for capsular seeding and contracture and the efficacy of antibiotic treatment in preventing such effects.

Materials and Methods

Animals and experimental conditions

All procedures were conducted on female Wistar rats weighing 200—250 g. They were allowed food ad lib, housed in open-air cages in groups of 3—5 and maintained under conditions of controlled temperature with a 12-h light/dark cycle. The Animal Ethics Committee of the University of Navarra approved this experimental protocol and animal treatment complied with the regulations of the European Commission (rule n 86/609/EEC). All aspects were considered to minimize suffering.

Study design

Two silicone blocks (1 cm^3) were implanted into each rat ($n = 42$) and the animals were divided into three experimental groups.

In order to simulate an infection with resulting transient bacteremia, Group A (infection group, $n = 11$) received an intraperitoneal injection of 4×10^7 CFU of Staphylococcus *aureus,* a quantity proven to be sufficient to produce a several-hour period of bacteremia in rats without secondary mortality.[16] A period of 1 month was allowed to pass before bacterial inoculation was carried out. This period was sufficient for the formation of a physiologic capsule around a silicone implant in a rat, which was important for simulating the conditions of late infection.[17]

Group B (treated group, $n = 14$) received an intraperitoneal injection in the same manner as Group A. Antibiotic treatment was administered after 1 week, allowing time for potential seeding and establishment of subclinical infection in the implants. A dose of 150—30 mg/kg of amoxicillin—clavulanic acid was administered subcutaneously every 12 h for a total of 7 days. Group C (control group, $n = 14$) underwent implantation without any additional procedures.

Surgical procedure

Before surgery, rats were anesthetized by an intraperitoneal injection (2 mL) of a mixture of ketamine, diazepam, and atropine. The dorsum of the rat was shaved and cleansed with povidone iodine, and all surgical procedures were carried out under sterile conditions. Two remote 1-cm skin incisions were made on the back of each animal at a distance sufficient to avoid merging of the pockets. Pockets were created by blunt dissection beneath the panniculus carnosus plane with dimensions barely sufficient to accommodate the implant, and one silicone block was placed in each. Incisions were then closed with stitches making sure the suture line was not directly over the implant as shown in Figure 1.

The silicone blocks were removed under sterile conditions together with their capsules 4 months after implantation. Animals were all sacrificed after removal of the implant. Three rats from the infection group were lost due to an overdose of anesthesia.

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