Infection Rate of Percutaneous Implants with Porous Metal Dermal Barriers

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Introduction: One common mode of percutaneous implant failure is epithelial downgrowth, where the epithelial layer of the skin migrates down the implant in an attempt to recreate a contiguous barrier against infections[1,3]. Texturing the implant surface and/or incorporating a subcutaneous flange to the implant are thought to stabilize the implant decreasing epithelial downgrowth and infection[3-7]. However, previous studies have been limited due to a lack of direct measurement of infection relating to these implant stabilization strategies. The goal of this research is to utilize dermal barriers, specifically a subcutaneous flange, to inhibit infection of percutaneous implants by investigating the infection rate in relation to porous and smooth metal surface textures. We hypothesize that an increase in porous surface area of the implant will result in a lower infection rate compared to an increase in smooth surface area of the implant.

Methods: The percutaneous implants fabricated from surgical grade titanium consisted of two elements: a subcutaneous disk (30mm x 10mm) and a percutaneous post (10mm x 15mm). Implants had a smooth polished surface texture or a porous surface texture (Thortex®, Portland, OR), with ~500 µm pore size and ~60% porosity. Four combinations of implant surface textures were investigated (a) smooth surface percutaneous post and subcutaneous disk (b) porous surface percutaneous post and subcutaneous disk, (c) smooth surface percutaneous post and porous surface subcutaneous disk (b) porous surface percutaneous post and smooth surface subcutaneous disk. The 4 device combinations were implanted on the dorsum of New Zealand White rabbits. Two midline incisions (cranial, caudal) were made and 4 subcutaneous pockets were created into which the subcutaneous disks were placed. A small, circular incision was created over the subcutaneous disk in which the percutaneous posts were seated (Fig 1).

Figure 1. Four percutaneous implants on rabbit dorsum.

After 8 weeks, animals were challenged with weekly applications of a 10^8 CFU *Staphylococcus aureus* (S. *aureus*) solution. At sign of grade II clinical infection[8], animals were sacrificed. Surface swabs and tissue biopsies were taken from the skin/implant interface to confirm infection. Specimens were fixed in 10% formalin, embedded in PMMA, stained with Giemsa, and analyzed with light microscopy.

Results: Preliminary results suggest that implants with a completely or partially smooth surface become infected twice as fast as a completely porous surface. Two animals were sacrificed with infection of smooth post/smooth disk implants after 4-5 weeks of bacterial application. One animal was sacrificed with infection of smooth post/porous disk implant after 9 weeks of bacterial application. Infections were confirmed with positive *S. aureus* tissue biopsies and surface swabs.

We observed epithelial downgrowth with completely smooth surface implants and a thicker fibrous capsule along smooth surface implants when compared to porous surface implants (Fig. 2). There was cellular ingrowth and vascularization within the pores of the porous implants (Figs. 3A, 3B, 3C).

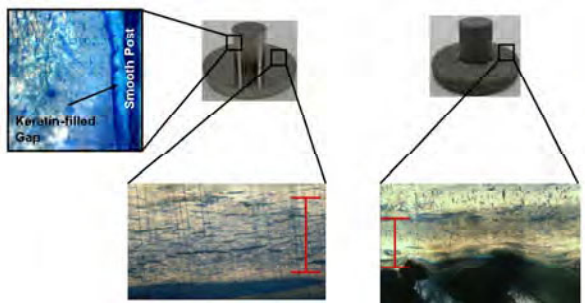

Figure 2. Epithelial downgrowth adjacent to smooth post with keratin-filled gap and fibrous capsule adjacent to smooth disk (10x). Fibrous capsule adjacent to porous disk (10x).

Figure 3. A. Fibroblasts and collagen fibers directed into pore (red arrow) (10x). B. Adipocytes and fibroblasts seen within pores (10x). C. Vasculature seen within pores (red arrows)(10x).

Conclusions: Our results suggest that when challenged with bacteria, porous surface implants have a decreased infection rate when compared to smooth surface implants. An interesting trend is that all three implants that became infected had smooth surface posts suggesting that the surface texture of the post contributes more to the vulnerability of infection. Though more results are pending for this study, we are continuing our research efforts with additional studies aimed at fortifying and accelerating the host tissue and implant integration.

References: (1)von Recum AF. JBMR. 1984;18:323-336 (2)Gross-Siestrup C. JBMR. 1984;18:357-82 (3)Pendegrass CJ. Biomaterials. 2006;27:4183-91 (4)Jansen JA. JBMR. 1990;24:295-307 (5)Jansen JA. J Mater Sci Mater Med. 2001;12:1033-7 (6)Gerristen M. JBMR. 2000;52:135-41 (7)Chehroudi B. Biomaterials. 2002;23:229-37 (8)Checketts RG. Orthofix Ext Fix in Trau and Ortho. 2000; 97-103.