Evaluation of the Biomet JuggerKnot Soft Anchor - 1.4 mm in a Rabbit Knee

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Suture anchors perform many roles in soft tissue repair. Many anchors are indicated for use in the shoulder, hand, knee, hip, and foot. While anchors tend to perform the same general function of securing tissue to bone, there are differences between them in terms of size, means of fixation, and materials of construction[1].

PEEK (Polyether Ether Ketone), a non-resorbable plastic, has been a common material of construction because it is radiolucent and is relatively strong. Composite suture anchors, a combination of a resorbable material such as PLLA (Poly-L-Lactic Acid) and a ceramic such as TCP (Tricalcium Phosphate), have seen increased use because they are resorbed and presumably replaced with bone.

Biomet Sports Medicine has developed a new style of suture anchor comprised of braided polyester, a material that has been used in the body for decades[2].

The primary objective of this study was to evaluate bone remodeling of the implanted Biomet JuggerKnot Soft Anchor - 1.4 mm under load, with no load, and a 3 mm empty defect in the knee of the rabbit.

Histological examination also provided a qualitative assessment on tissue response and cellular growth relative to the implant/defect sites.

Methods

Specimens

Three New Zealand White (NZW) rabbits were operated bilaterally in this study. The NZW rabbit has been utilized extensively for evaluating the biocompatibility and efficacy of bone substitute materials and tendon fixation strategies. Similar studies have historically been conducted in the rabbit femur to investigate defect healing[3,4,5].

Surgical Procedure

Three time periods of 2, 4 and 6 weeks were evaluated. One rabbit was assigned to each time period. Each rabbit was operated bilaterally and received one JuggerKnot Soft Anchor - 1.4 mm implanted into each distal medial femoral condyle to repair a transected MCL so that it would carry a load, one JuggerKnot Soft Anchor - 1.4 mm implanted into the same distal medial condyle and clipped short so that it would carry no load, and one empty 3 mm defect was created to serve as a control in the medial femoral condyle as shown in Figure 1.

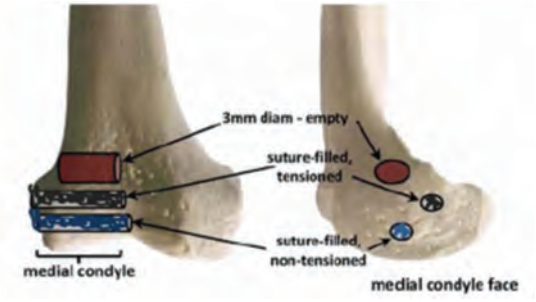

Fig. 1
Implantation site

Under general anesthesia, a 4-6 cm medial skin incision was made directly over the medial condyle in the distal femur and MCL. The origin of the MCL was sharply transected away from the femoral condyle. A 1.4 mm diameter hole was drilled into the medial femoral condyle at the MCL origin "footprint". A JuggerKnot Soft Anchor - 1.4 mm suture anchor was inserted into the condylar drill hole. The transected MCL end was sutured in a 2-strand Krackow (interlocking) pattern with non-absorbable suture Maxbraid #3-0 suture, and then tied to the JuggerKnot Soft Anchor - 1.4 mm suture as a loaded repair.

A second JuggerKnot Soft Anchor - 1.4 mm was inserted into the same femur approximately 5 mm away from the MCL repair. The sutures were cut to just exit the bone so that neither the suture nor the anchor would carry a load.

Lastly, a 3 mm diameter empty defect was created by drilling through the medial condylar cortex and cancellous bone, to the opposite cortex and spaced at least 5 mm away from the closest edge of the previously placed anchor as shown in Figure 2.

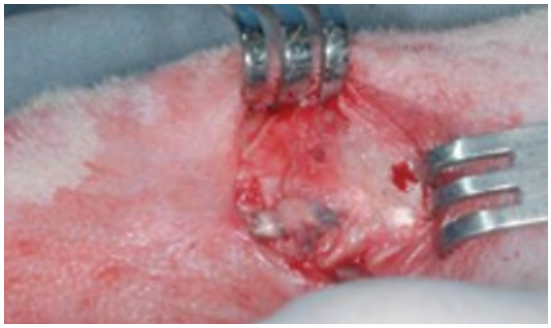

Fig. 2
Surgical implantation site of JuggerKnot Soft Anchor - 1.4 mm and empty defect

Unloaded JuggerKnot Soft Anchors and 3 mm defects were routinely alternated in condylar position between right and left femurs.

Results

Histology Examination

A total of 18 defects received treatment in this study. Three time periods of 2, 4, and 6 weeks were investigated. Specimens were imbedded in plastic to facilitate creation of cross sectional slides as shown in Figure 3.

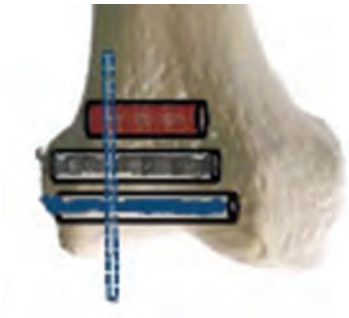

Fig. 3
Cross section of slides

Slides were then stained using hematoxylin and eosin, and were reviewed for histology. A qualified pathologist, who was unblinded to implant treatments, provided a qualitative assessment of new bone formation, and any unusual or unexpected cellular responses relative to implant/defect sites.

The cellular and tissue response for each specimen was scored. The cellular response histopathology evaluation scores were summed for each treatment group at each time point. Tables 1-4 display histological observation results/scores.

Table 1. Histiological Evaluation System - Cellular Response

Cellular response	Score				
	0	1	2	3	4
Polymorphonuclear cells	0	Rare, 1-5/phf*	6-10/phf	heavy infiltrate	packed
Lymphocytes	0	Rare, 1-5/phf	6-10/phf	heavy infiltrate	packed
Plasma cells	0	Rare, 1-5/phf	6-10/phf	heavy infiltrate	packed
Macrophages	0	Rare, 1-5/phf	6-10/phf	heavy infiltrate	packed
Giant cells	0	Rare, 1-2/phf	3-5/phf	heavy infiltrate	packed

*phf=per high powered (400x) field

Table 2. Histiological Evaluation System - Tissue Response					
Tissue response	Score				
	0	1	2	3	4
Neovasularization	0	Minimal capillary proliferation, focal, 1-3 buds	Groups of 4-7 capillaries with suporting fibroblastic structures	Broad band of capillaries with supporting structures	Extensive band of capillaries with supporting fibroblastic structures
Fibrosis	0	Narrow band	Moderately thick band	Thick band	Extensive band
Fatty infiltrate	0	Minimal amount of fat associated with fibrosis	Several layers of fat and fibrosis	Elongated and broad accumulation of fat cells about implant size	Extensive fat completely surrounding the implant
Chondroplasia	0	Few chondroblasts/ chondrocytes associated with fibrosis	Mild fibrocartilagenous transformation associated with fibrosis	Moderate fibrocartilagenous transformation associated with fibrosis	Extensive fibrocartilagenous replacement of fibrosis
Ossification	0	Few islets of osteoid producing osteoblasts associated with fibrosis or chondroplasia	Moderate ossification of fibrocartilagenous regions	Extensive ossification of fibrocartilagenous regions	Complete osseous transformation of fibrocartilagenous regions, continguous with mature bone
Necrosis	0	Minimal	Mild	Moderate	Extensive

Cellular response		Score		
		2 weeks	4 weeks	6 weeks
Polymorphonuclear cells	Loaded	0.0	0.5	0.5
	Unloaded	0.0	0.0	0.0
	Empty	0.0	0.0	–
Lymphocytes	Loaded	1.0	1.0	1.0
	Unloaded	1.0	1.0	1.0
	Empty	0.0	0.0	–
Plasma Cells	Loaded	0.5	0.0	0.0
	Unloaded	0.0	0.0	0.0
	Empty	0.0	0.0	–
Macrophages	Loaded	1.5	1.0	1.0
	Unloaded	1.5	1.0	0.5
	Empty	0.0	0.0	–
Giant cells	Loaded	0.5	0.5	0.0
	Unloaded	1.0	0.0	0.0
	Empty	0.0	0.0	–

Tissue response		Score		
		2 weeks	4 weeks	6 weeks
Neovascularization	Loaded	1.0	1.5	1.5
	Unloaded	1.5	1.5	2.0
	Empty	0.0	0.0	–
Fibrosis	Loaded	1.0	2.0	2.0
	Unloaded	2.0	2.5	2.0
	Empty	0.0	0.0	–
Fatty Infiltration	Loaded	0.0	0.0	0.0
	Unloaded	0.0	0.0	0.0
	Empty	0.0	0.0	–
Chondroplasia	Loaded	1.5	1.0	1.0
	Unloaded	0.0	0.5	0.0
	Empty	2.0	2.0	–
Ossification	Loaded	0.5	1.0	1.5
	Unloaded	0.5	0.5	0.0
	Empty	2.5	2.0	–
Necrosis	Loaded	0.0	0.0	0.0
	Unloaded	0.0	0.0	0.0
	Empty	0.0	0.0	–

Tables 3-4
Average time point ranking for each cellular and tissue response for holes containing loaded and unloaded JuggerKnot Soft Anchor's as well as the empty defects.

Histological Results

Disorganized cellular infiltrate occurred deep within the implant due to the porous nature of the braided construct of the anchor. In some instances, several types of tissue, including loose connective tissue, cartilage, and bone, were noted between the suture filaments. Results shown in Figures 4-6. More extensive ossification was seen in empty defects as shown in Figure 7.

Because of the small size of the anchors and defects, only one 3 mm defect site was located at 4 weeks, only one unloaded site was located at 6 weeks and no 3 mm defect sites were located at 6 weeks.

Empty Defect Response Score

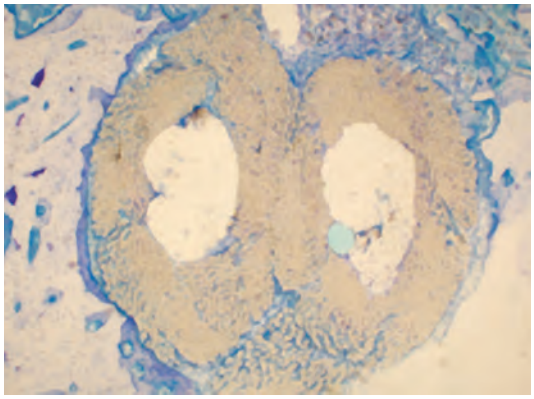

Fig. 4
Representative cross section of implanted JuggerKnot Soft Anchor - 1.4 mm at 2 weeks

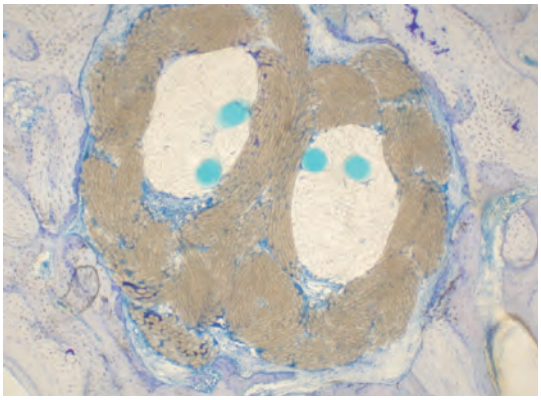

Fig. 6
Representative cross section of JuggerKnot Soft Anchor - 1.4 mm at 6 weeks.

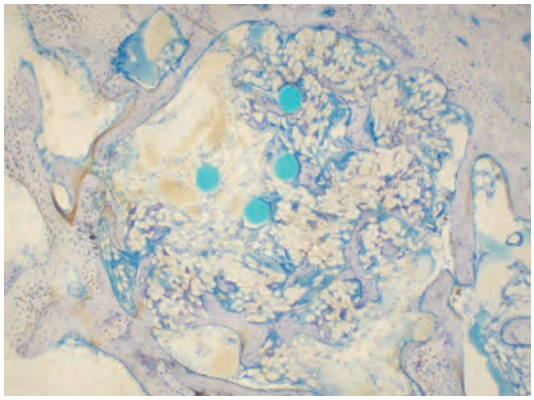

Fig. 5
Cross section superficial to the anchoring portion of the JuggerKnot Soft Anchor - 1.4 mm at 6 weeks of an unloaded anchor.

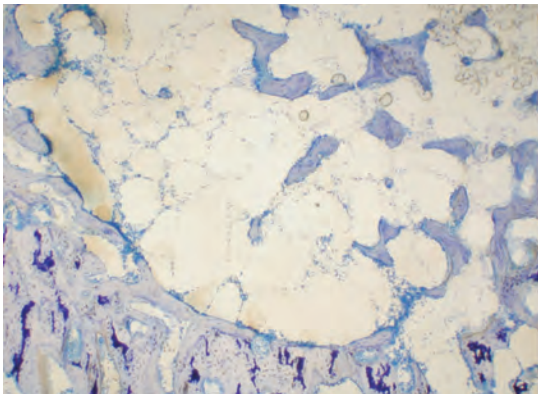

Fig. 7
Representative cross section of 3 mm void at 4 weeks.

Discussion

The tissue response of the JuggerKnot Soft Anchor - 1.4 mm suture anchors, both loaded and unloaded, were compared to the tissue response of a 3 mm empty defect, in this study. Significant amounts of cellular activity were noted in and around the anchor.

Limitations of the study were the difficulty in finding empty control defects to compare with the JuggerKnot Soft Anchor - 1.4 mm. The 3 mm empty defect is smaller than the 5 mm commonly referenced critical size defect. We believe this accounts for the difficulty in finding some of the empty defects. Due to the size and construct of the anchor, the pathologist was unblinded to implant treatments.

While rabbits have historically been a model used to study bone healing, there are limits to the conclusions that can be drawn with respect to clinical performance.

Conclusion

In this study where the implant was placed in bone, the surrounding bone generated osteoblasts which infiltrated the implant and formed islands of newly formed bone. Overall, the reactions observed within and around the implant were those that are expected in tissues implanted with foreign material. There were no unexpected cellular healing responses, negative biological bone responses or resorption noted in the hard or soft tissue of the defects. Normal and expected healing response (locally reparative) were observed at the implant sites throughout the course of the study.

References

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Notes

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