EXPERIMENTAL STUDY

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Use of a perichondrial autograft on the peritendinous adhesion: an experimental study in rabbits

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Objective: The aim of this experimental study was to evaluate the use of a clinically available perichondrium graft as an adjunct to surgical tendon repair.

Methods: Eight male New Zealand white rabbits of similar height and weight were used in this study. The left and right Achilles tendons were used as the experimental and control group, respectively. Perichondrium grafts were harvested from the right ears of the rabbits. Both Achilles tendons were clearly cut and repaired. After the repair, the perichondrium graft was wrapped around the tendon repair sites of the left Achilles tendons. Rabbits were sacrificed after six weeks and the tendons were examined macroscopically and histopathologically.

Results: Macroscopically and histopathologically, less adhesion occurred when the perichondrium graft was wrapped around the tendon repair site compared to the control group.

Conclusion: Perichondrium graft may isolate the repaired tendon and may reduce scar formation and adhesions during the healing period.

Key words: Perichondrium; rabbit; tendon adhesion.

Flexor tendon injuries are common injuries prone to many complications even after surgical repair. Despite the development in surgical methods and rehabilitation programs, adhesion between the tendon and its sheath is the most frequent complication following tendon repair. The initial injury, the resultant inflammatory response, the surgical trauma and the foreign body reaction contribute to the formation of scar tissue. Tendon healing is proceeded by a combination of extrinsic and intrinsic processes. Previously, it was thought that peritendinous adhesions contributed to the healing process, since the chemotaxis of precursor cells into the defect was believed to be an essential extrinsic process of tendon healing.[1,2] Currently, however, it is well known that tendon healing can, and ideally should, occur in the absence of peritendinous adhesions and through the intrinsic process: by the activity of tenocytes in the tendon sheath with sufficient supplies of cytokines and growth factors

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from the outside.[3,4] Despite a better understanding of tendon healing, the introduction of refined surgical techniques and the use of sophisticated rehabilitation programs, results within the tendon sheath following tendon repair remain highly unpredictable. Therefore, many modalities such as the use of pharmacologic modulators or mechanical barriers between the tendon and adjacent tissues have been investigated to prevent adhesion formation.[5-13] Mechanical barriers, including alumina sheaths, polyethylene membranes, cellophane, Sterispon wrapping, stainless steel or silicone sheeting, amniotic membranes, chitosan membranes, silicone rubber envelopes, polytetrafluoroethylene surgical membranes, Seprafilm, hydrogel sealant, chondroitin sulfate-coated polyhydroxyethyl methacrylate membrane or autogenous vein graft may be either biological or synthetic.[14-20] The ideal barrier would be easy to use, biocompatible, allow tendon movement, not add undue bulk, remain at the site of repair long enough to allow tendon healing (extrinsic healing), and cheap. While many of these barrier materials have shown initial promise, none have found their way to routine clinical use.

We have hypothesized that the perichondrium, which has a similar mesenchymal origin to the tendon sheath, may be used as a barrier to prevent fibrous ingrowth from surrounding tissues following flexor tendon repair. The purpose of this study was to evaluate the effect of perichondrial autograft in preventing adhesions following tendon injury.

Materials and methods

The protocol of the experiment was approved by the ethical committee of Izmir Ataturk Training and Research Hospital. Eight male white New Zealand rabbits, weighing 2.4 to 2.7 kg, were quarantined for a minimum of two days between purchase and testing. The rabbits were housed on a 12:12 light-dark cycle with food and water available ad libitum in the laboratory.

Anesthesia was induced with an intramuscular injection of 20 mg/kg ketamine hydrochloride and intracutaneous injection of 1% lidocaine. The operative site was shaved and stained with povidone-iodine. A preoperative dose of intramuscular cefazolin sodium (0.1 mg/kg) was administered for infection prophylaxis.

Each rabbit provided two test sites on the left and right crural Achilles tendon and a perichondrial autograft was harvested from the right ear of each rabbit. The animals were placed in the dorsal recumbent position with the anklebone inflected. A posterior longitudinal skin incision of 2 cm was made in both legs sequentially. After dissection of the skin and subcutaneous tissues, the Achilles tendon was exposed and full thickness transverse tenotomy was made using a Number 11 blade at a point 0.5 cm proximal to the distal insertion of the tendon. Transected tendons were repaired with the modified Kessler technique using 5/0 monofilament polypropylene (Prolene; Ethicon Inc., Somerville, NJ, USA) sutures and a continuous circumferential adaptation suture with 6-0 monofilament polypropylene. Tendon sheaths were not repaired. The experimental group (Group 1) consisted of the 8 right legs where the repair site was circumferentially wrapped with perichondrial autograft which was harvested from the right ear of the rabbit (Figs. 1-3). The control group (Group 2) consisted of the 8 left legs, where the same procedures were applied without the perichondrial autograft wrapping. The skin was closed in an interrupted fashion with 5/0 silk sutures and no wound dressing was applied. All rabbits were immobilized with a plaster cast for 2 weeks.

Postoperatively, the animals were inspected for signs of wound infection or dehiscence. All animals survived and no tendon rupture was observed. Rabbits were able to walk in their cages without any difficulty. No wound dehiscence, wound infection or exposure of repaired tendons occurred. Six weeks later after surgery the animals were sacrificed with ether anesthesia and knee disarticulation was performed in both hind limbs.

The Achilles tendons in both hind limbs were excised en bloc with origin and insertion. The specimens were transferred to the pathology department in a solution of 10% formalin.

The criteria described by Tang et al.[21] were used for the macroscopic quantitative evaluation of peritendinous adhesions. Adhesion length, density and motion capacity of the repaired tendons were evaluated. Eight leg-tendon complexes for each group were evaluated according to these criteria (Table 1) and were used for macroscopic evaluation.

Specimens were fixed in neutral buffered formalin for a minimum of 48 hours and embedded in paraffin. Permanent sections of 5 to 7 μm were stained with hematoxylin and eosin. The histological sections (stained with hematoxylin and eosin) were evaluated microscopically (Olympus BX-50; Olympus Optical Co., Tokyo, Japan) (Figs. 4-8).

The degree of fibrosis, hypervascularity, inflammatory cell infiltration, and adhesions between the tendon and its sheath were rated between from (-) to (+++). The results were compared between the groups.

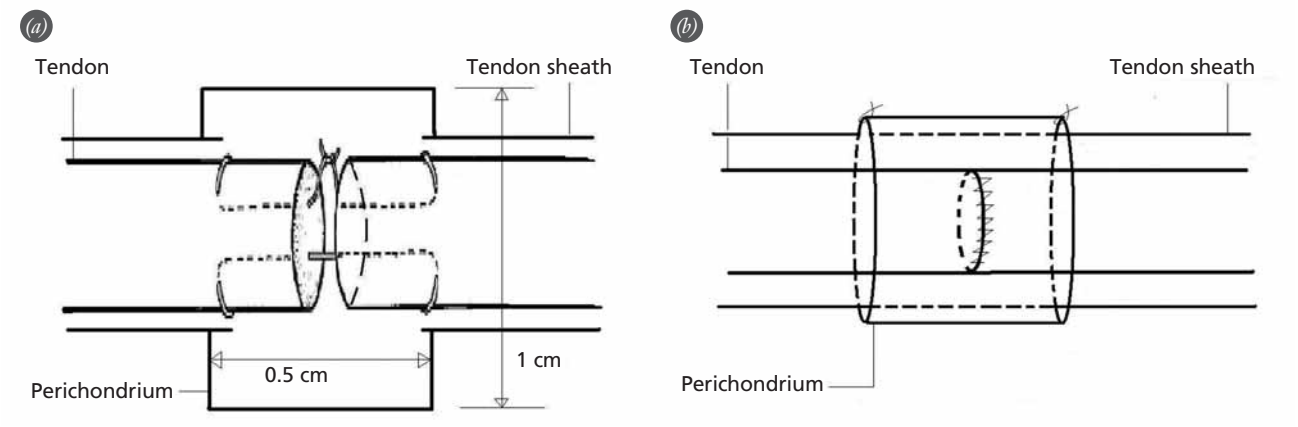

Fig. 1. **(a, b)** The repair zone is encircled with the perichondrial autograft as demonstrated in the above schemes.

SigmaStat® v3.11 (Statcon, Witzenhausen, Germany) software was used for statistical analyses. The Mann-Whitney U test was used for the comparison between the groups (Figs. 6-8). A p value of less than 0.01 was considered statistically significant.

Results

There was no significant difference between the groups in terms of the degrees of inflammatory cell infiltration in tendons and tendon sheaths, and the vascularization of the tendon and tendon sheaths (p>0.1) (Tables 2-5). However, there was significantly more fibrosis in the tendon sheaths (p=0.003) (Table 6) and in the tendons (p=0.001) (Table 7) of the control group than the experimental group. In Group 1, there was no adhesion in 5 of the 8 leg-tendon complexes and only mild adhesions in the other 3. In Group 2, there were

Table 1. Criteria described by Tang et al.[21] and macroscopic evaluation of adhesions.

Points	Features of adhesion
Length of adhesions	
0	No apparent adhesion
1	Localized, approximately 10 mm longitudinal adhesion
2	Between 10-15 mm
3	Dense, more than 15 mm
Quality	
0	No apparent adhesion
1	Loose, elastic and very moving
2	Moderately intense and moving
3	Dense, not filamentous and stationary
Grading of adhesions	
0	No apparent adhesion
1, 2	Slight
3, 4	Moderate
5, 6	Severe

Fig. 2. Preparation of the perichondrial autograft. [Color figure can be viewed in the online issue, which is available at www.aott.org.tr]

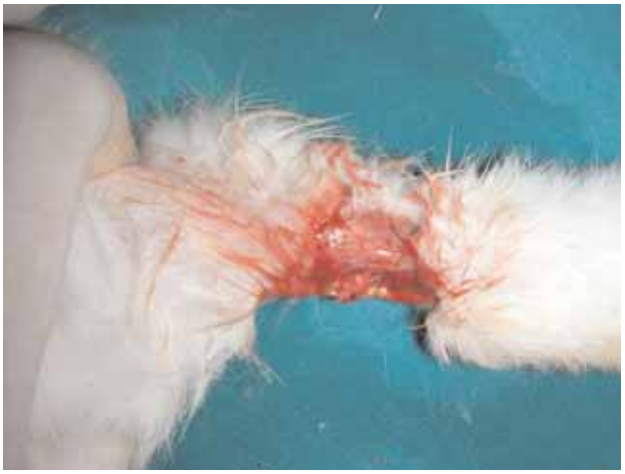

Fig. 3. Perichondrial autograft is wrapped around the repair site. [Color figure can be viewed in the online issue, which is available at www.aott.org.tr]

mild, moderate or severe adhesions in all cases. There was a significant difference between the grades adhesion in the experimental and control groups (p=0.001) (Table 8). The results of the macroscopic evaluation for fibrosis are presented in Table 9.

According to the histopathologic findings in Group 1, the synovial space was protected but in Group 2 there was a marked narrowing. In Group 2, there was also significant peritendinous fibrosis while there was minimal fibrosis in Group 1. There was no considerable difference in vascularity and cell infiltration (Figs. 4-8).

Discussion

The hand is the most commonly injured part of the body.[15] Injured tendons are capable of intrinsic healing without surgery provided that the injured tissue has access to the necessary nutrients. In such cases, it is vital that the formation of adhesions in the healing area be avoided to the greatest possible extent; toward this end, methods of separating the healing tendon from its surrounding tissues have been supported.[7]

Though primary repair of flexor tendons with accompanying preservation or repair of the tendon sheath has been accepted as the treatment of choice, complications are possible. The distance of the gliding tunnel may be restricted by the surgical sheath closure. In two studies, repaired tendons with a widened sheath tunnel had greater tendon movement than those within a narrowed sheath.[21] However, surgical repair of the sheath may not possible in cases in which the sheath is crushed or severely damaged and must be resected following injury.[19] In such cases, attempts are made to

Table 2. Histologic evaluation results for inflammatory cell infiltration on the tendon sheath.

	-	+	++	+++
Group 1 (n=8)	4	3	1	0
Group 2 (n=8)	5	2	1	0

Group 1: experimental group; Group 2: control group. p>0.1; no significant difference. - : none; +: mild; ++: moderate: +++: severe

Table 3. Histologic evaluation results for inflammatory cell infiltration on the tendon.

	-	+	++	+++
Group 1 (n=8)	4	2	2	0
Group 2 (n=8)	3	2	2	1

Group 1: experimental group; Group 2: control group. p>0.1; no significant difference. - : none; +: mild; ++: moderate: +++: severe

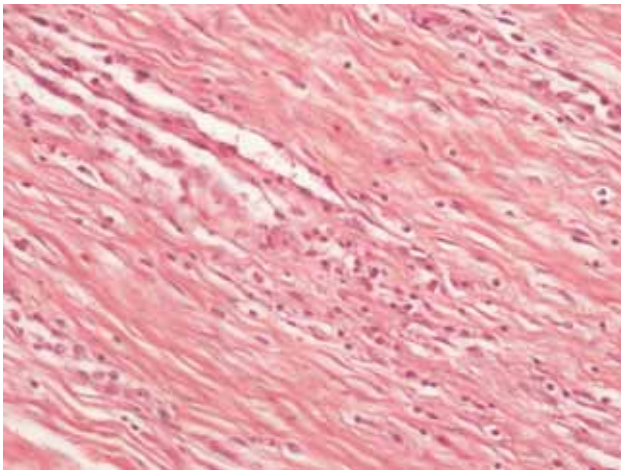

Fig. 4. Vascularization and inflammatory cell infiltration in Group 1 (H-E x10). [Color figure can be viewed in the online issue, which is available at www.aott.org.tr]

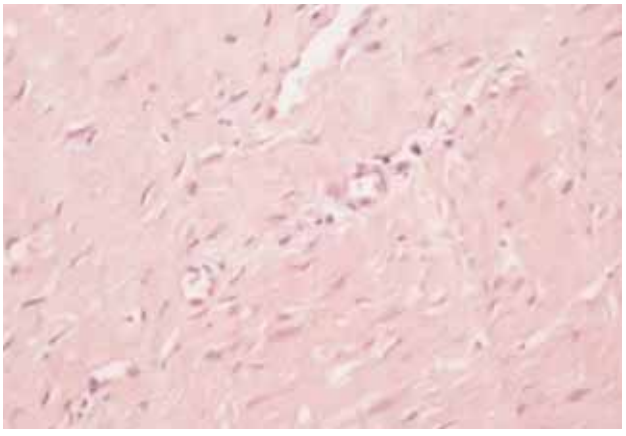

Fig. 5. Vascularization and inflammatory cell infiltration in Group 2 (H-E x10). [Color figure can be viewed in the online issue, which is available at www.aott.org.tr]

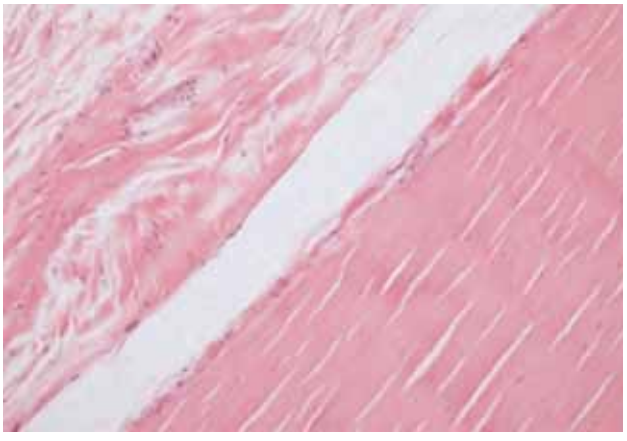

Fig. 6. Synovial space is well-preserved in Group 2 (H-E x10). [Color figure can be viewed in the online issue, which is available at www.aott.org.tr]

Table 4. Histologic evaluation results for vascularization on the tendon sheath.

	-	+	++	+++
Group 1 (n=8)	2	3	3	0
Group 2 (n=8)	1	4	2	1

Group 1: experimental group; Group 2: control group. p>0.1; no significant difference. - : none; +: mild; ++: moderate: +++: severe

Table 5. Histologic evaluation results for vascularization on the tendon.

	-	+	++	+++
Group 1 (n=8)	2	3	3	0
Group 2 (n=8)	1	4	2	1

Group 1: experimental group; Group 2: control group. p>0.1; no significant difference. - : none; +: mild; ++: moderate: +++: severe

Table 6. Histologic evaluation results for fibrosis on the tendon sheath.

	-	+	++	+++
Group 1 (n=8)	5	2	1	0
Group 2 (n=8)	1	1	3	3

Group 1: experimental group; Group 2: control group. p=0.003; significant difference. -: none; +: mild; ++: moderate: +++: severe

Table 7. Histologic evaluation results for fibrosis on the tendon.

	-	+	++	+++
Group 1 (n=8)	6	2	0	0
Group 2 (n=8)	0	3	3	2

Group 1: experimental group; Group 2: control group. p=0.001; significant difference. - : none; +: mild; ++: moderate: +++: severe

restore the integrity of the sheath or to use various biological or synthetic materials.[7] The idea of a mechanical barrier to adhesion formation is not new. Previously, many similar materials, both biological and synthetic, have been tried.[14-20] Biological barriers have met with variable success and add the complications of donor site morbidity and surgical complexity to the procedure. Some synthetic materials fail because they stimulate a severe inflammatory response or allow ingrowths of adhesions around the edges of the material. Other materials prevent nutrient diffusion to the healing tendon leading to tendon necrosis. In recent years, focus has turned to diffusible membranes and inhibited adhesions were found with the use of a hyaluronic acid membrane in a study using chicken models.[13] This has not found wide clinical application, however, because the material is difficult to prepare

Table 8. Histologic evaluation results for adhesion between the tendon and its sheath.

	-	+	++	+++
Group 1 (n=8)	5	3	0	0
Group 2 (n=8)	0	2	3	3

Group 1: experimental group; Group 2: control group. p=0.001; significant difference. - : none; +: mild; ++: moderate: +++: severe

Table 9. Macroscopic evaluation results for fibrosis on the tendon sheath.

	-	+	++	+++
Group 1 (n=8)	5	3	0	0
Group 2 (n=8)	0	1	4	3

Group 1: experimental group; Group 2: control group. p=0.003; significant difference. - : none; +: mild; ++: moderate: +++: severe

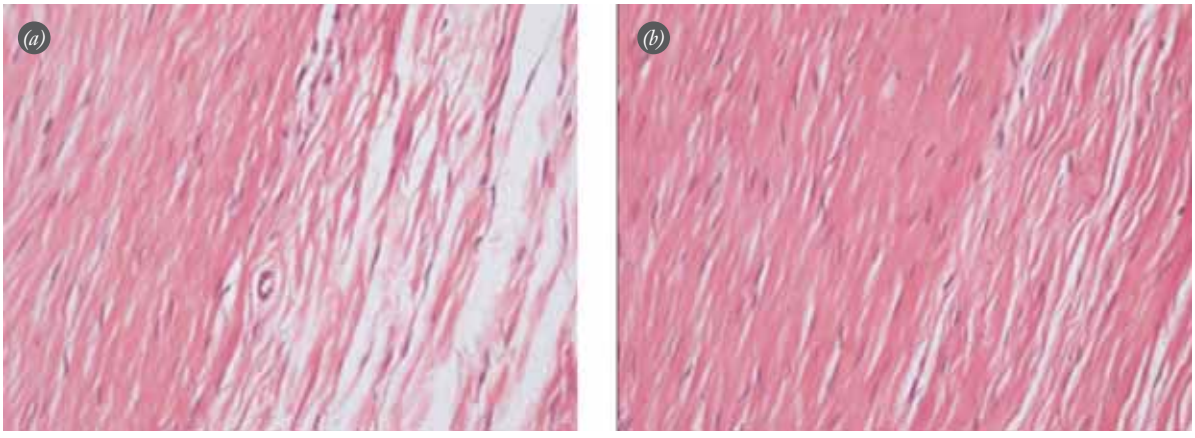

Fig. 7. **(a, b)** Two slides from Group 2 are seen with the same narrowed synovial space (H-E x10). [Color figure can be viewed in the online issue, which is available at www.aott.org.tr]

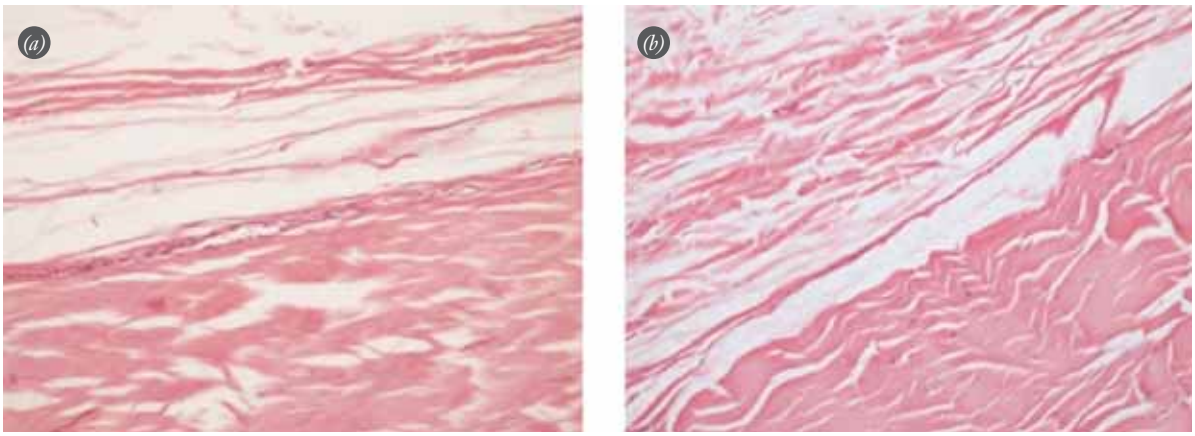

Fig. 8. Histological examination showed **(a)** minimal peritendinous fibrosis in Group 2 and **(b)** significant fibrosis in Group 1 (H-E x10). [Color figure can be viewed in the online issue, which is available at www.aott.org.tr]

and must be sewn around the tendon repair. Mentzel et al.[22] studied a bioresorbable gel composed of a carbohydrate polymer (ADCON-T/N; Gliatech, Inc., Cleveland, OH, USA). Although a decrease in tendon adhesion formation was demonstrated in an animal model, clinical trial results have been disappointing and there is some question as to whether the material migrates from the repair site.[13]

Due to a similar mesenchymal origin of the tendon sheath and perichondrium, the tendon repair site was wrapped with a perichondrial autograft to prevent fibrous ingrowth from surrounding tissues in this experimental study (Fig. 1). No experimental or clinical study concerning the possible effects of perichondrial autograft on the healing or adhesion formation in tendons has been made. In our study, we tested the hypothesis that the use of perichondrial auto graft serves as a safe barrier and significantly reduces the extent of peritendinous adhesion formation after tendon repair. In addition, we evaluated the ability of perichondrial auto graft to restore the integrity of the sheath. For this purpose, a bilateral Achilles tendon repair rabbit model was utilized. Our histologic and macroscopic results showed that the use of perichondrial graft around the flexor tendon repair decreased adhesion formation (Figs. 6-8). There was a statistically significant difference between the groups. To our knowledge our study is the first to evaluate the effect of perichondrial graft in flexor tendon adhesions.

A limitation of our study was the lack of a complementary biomechanic study. However, our technique is simple and can be used in all cases of primary and secondary repairs, especially in delayed flexor tendon repairs. Early postoperative mobilization and early exercise after tendon repair are encouraged.[23-26] Because of the disastrous effects that adhesion formation leaves on the gliding mechanism of flexor tendons, a number of techniques have been devised to permit early movement and to prevent adhesion formation.

In conclusion, both clinically and experimentally, it has been shown that early mobilization techniques after flexor tendon repair within the digital sheath improve tendon healing and the final result. As our technique reduces adhesion formation, improves tendon nourishment, allows early mobilization, decreases the need for intensive physiotherapy, and improves significantly the function of the operated hand when compared to other methods, it may substitute for the conventional tendon repair techniques and perhaps become a standard technique in the future. We believe that the perichondrial autograft technique has merit, and further prospective randomized controlled trials need to be performed to prove our hypothesis.

Conflicts of Interest: No conflicts declared.

References

1. James R, Kesturu G, Balian G, Chhabra AB. Tendon: biology, biomechanics, repair, growth factors, and evolving treatment options. J Hand Surg Am 2008;33:102-12.
2. Potenza AD. Tendon healing within the flexor digital sheath in the dog. J Bone Joint Surg Am 1962;44-A:49-64.
3. Gelberman RH, Manske PR, Akeson WH, Woo SL, Lundborg G, Amiel D. Flexor tendon repair. J Orthop Res 1986;4:119-28.
4. Ingraham JM, Hauck RM, Ehrlich HP. Is the tendon embryogenesis process resurrected during tendon healing? Plast Reconstr Surg 2003;112:844-54.
5. Khanna A, Friel M, Gougoulias N, Longo UG, Maffulli N. Prevention of adhesions in surgery of the flexor tendons of the hand: what is the evidence? Br Med Bull 2009;90:85-109.

6. Stark HH, Boyes JH, Johnson L, Ashworth CR. The use of paratenon, polyethylene film, or silastic sheeting to prevent restricting adhesions to tendons in the hand. J Bone Joint Surg Am 1977;59:908-13.
7. Wheeldon T. The use of cellophane as a permanent tendon sheath. J Bone Joint Surg 1939;21:393-6.
8. Potenza AD. Critical evaluation of flexor-tendon healing and adhesion formation within artificial digital sheaths. J Bone Joint Surg Am 1963;45:1217-33.
9. Hunter JM, Salisbury RE. Flexor-tendon reconstruction in severely damaged hands. A two-stage procedure using a silicone-dacron reinforced gliding prosthesis prior to tendon grafting. J Bone Joint Surg Am 1971;53:829-58.
10. Peterson WW, Manske PR, Dunlap J, Horwitz DS, Kahn B. Effect of various methods of restoring flexor sheath integrity on the formation of adhesions after tendon injury. J Hand Surg Am 1990;15:48-56.
11. Kobayashi M, Toguchida J, Oka M. Development of polyvinyl alcohol-hydrogel (PVA-H) shields with a high water content for tendon injury repair. J Hand Surg Br 2001;26:436-40.
12. Hanff G, Hagberg L. Prevention of restrictive adhesions with expanded polytetrafluoroethylene diffusible membrane following flexor tendon repair: an experimental study in rabbits. J Hand Surg Am 1998;23:658-64.
13. Işik S, Oztürk S, Gürses S, Yetmez M, Güler MM, Selmanpakoğlu N, et al. Prevention of restrictive adhesions in primary tendon repair by HA-membrane: experimental research in chickens. Br J Plast Surg 1999;52:373-9.
14. Demirkan F, Colakoglu N, Herek O, Erkula G. The use of amniotic membrane in flexor tendon repair: an experimental model. Arch Orthop Trauma Surg 2002;122:396-9.
15. Ferguson RE, Rinker B. The use of a hydrogel sealant on flexor tendon repairs to prevent adhesion formation. Ann Plast Surg 2006;56:54-8.
16. Güdemez E, Ekşioğlu F, Korkusuz P, Aşan E, Gürsel I, Hasirci V Chondroitin sulfate-coated polyhydroxyethyl methacrylate membrane prevents adhesion in full-thickness tendon tears of rabbits. J Hand Surg Am 2002;27:293-306.
17. Menderes A, Mola F, Tayfur V, Vayvada H, Barutçu A. Prevention of peritendinous adhesions following flexor tendon injury with seprafilm. Ann Plast Surg 2004;53:560-4.
18. Ozgenel GY. The effects of a combination of hyaluronic and amniotic membrane on the formation of peritendinous adhesions after flexor tendon surgery in chickens. J Bone Joint Surg Br 2004;86:301-7.
19. Siddiqi NA, Hamada Y, Ide T, Akamatsu N Effects of hydroxyapatite and alumina sheaths on postoperative peritendinous adhesions in chickens. J Appl Biomater 1995;6:43-53.
20. Zhang H, Sheng ZJ, Hou CL. Effect of chitosan membrane on tendon adhesion and healing. [Article in Chinese] Zhongguo Xiu Fu Chong Jian Wai Ke Za Zhi 1999;13:382-5.
21. Tang JB, Shi D, Zhang QG. Biochemical and histologic evaluation of tendon sheath management. J Hand Surg Am 1996;21:900-8.
22. Mentzel M, Hoss H, Keppler P, Ebinger T, Kinzl L, Wachter NJ. The effectiveness of ADCON-T/N, a new anti-adhesion barrier gel, in fresh divisions of the flexor tendons in Zone II. J Hand Surg Br 2000;25:590-2.
23. Conway H, Smith JW, Elliott MP. Studies on the revascularization of tendons grafted by the silicone rod technique. Plast Reconstr Surg 1970;46:582-7.
24. Galanakis I, Aligizakis A, Katonis P, Vavouranakis H, Stergiopoulos K, Hadjipavlou A. Functional evaluation after primary flexor tendon repair in zone II. Acta Orthop Belg 2003;69:252-6.
25. Herndon JH. Treatment of tendon injuries in children. Orthop Clin North Am 1976;7:717-31.
26. Strickland JW, Glogovac SV. Digital function following flexor tendon repair in zone II: a comparison of immobilization and controlled passive motion techniques. J Hand Surg Am 1980;5:537-43.