



ORIGINAL ARTICLE

Role of Autogenous Fat Grafting in Prevention of Tendon Adhesion after Repair of Tendoachilles in Male Albino Rats

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ABSTRACT

Background: Tendon injuries are common musculoskeletal disorders, often requiring surgical intervention. Postoperative adhesions are a major complication, hindering tendon function. Autogenous fat grafting has gained attention for its potential anti-inflammatory and regenerative effects in tendon repair. This study aimed to evaluate the role of autogenous fat grafting in preventing postoperative tendon adhesion and enhancing tendon healing following Achilles tendon repair in male albino rats. **Methods:** This prospective study included 26 male albino rats (52 Achilles tendons). The right tendon was repaired using the Modified Kessler technique with autogenous fat grafting (Group A), while the left tendon was repaired without grafting (Group B, control). Rats were followed for six weeks, and tendons were harvested on day 45 for histopathological and morphometric analysis. **Results:** Two rats died during anesthesia; 24 completed the study. Clinically, gait recovery and healing time showed no statistically significant differences between groups, although fat-grafted tendons demonstrated consistently better recovery trends. Histologically, Group A exhibited marked reparative processes with parallel collagen fiber orientation, reduced fibroplasia, and minimal adhesions. Morphometric analysis revealed a significant reduction in polymorphonuclear and macrophage counts in Group A compared to Group B ($p < 0.001$), while lymphocyte counts showed no significant difference. Collagen analysis demonstrated significantly higher type I collagen deposition in Group A ($50 \pm 7\%$) compared with Group B ($31 \pm 4\%$; $p < 0.001$). **Conclusion:** Autogenous fat grafting offers significant biological benefits in tendon repair. The reduction in inflammatory infiltration, particularly of neutrophils and macrophages, suggests that fat grafts may help modulate the immune response, potentially leading to a more favorable healing environment. **Keywords:** Autogenous fat grafting; Achilles tendon repair; Tendon adhesion; Collagen type I

INTRODUCTION

Tendons are dense connective tissues that extend from muscles, crossing joints to transmit force and produce motion. Despite their remarkable tensile strength, which allows them to tolerate the substantial forces generated by muscle contraction, tendons remain susceptible to injury, particularly through chronic overuse tendinopathies and traumatic ruptures[1].

Tendon injuries are among the most common musculoskeletal problems, representing a major cause of morbidity worldwide. They not only impose a significant financial burden on healthcare systems but also contribute to loss of productivity and long-term functional impairment [2]. Importantly, lacerated tendons lack the ability to undergo spontaneous healing, often necessitating surgical interventions.

Conventional repair strategies such as suturing, tendon grafting, or synthetic prosthesis replacement are widely employed. However, these techniques are frequently complicated by postoperative adhesion formation between the repaired tendon and surrounding tissues, resulting in limited range of motion and impaired biomechanical function[3].

Fat grafting has recently emerged as a promising adjunctive technique in tendon repair. Theoretically, autogenous fat grafts may enhance tenocyte regeneration and contribute to the organization of tissues resembling a healthy tendon complex[4]. Clinical evidence also supports this approach: reported successful treatment of dorsal foot tendon adhesions using tenolysis combined with fat grafting, with improved range of motion and pain relief sustained for two years.

The regenerative potential of fat tissue is partly attributed to its abundance of adipose-derived stem cells (ASCs), which possess the ability to differentiate into multiple cell types, including tenocytes and myocytes[5].

Additionally, fat tissue provides stromal vascular fractions (SVFs), known to support tissue repair and regeneration[6]. These properties help explain the potential role of fat grafting in accelerating tendon healing and limiting adhesion formation.

Given its reconstructive and regenerative properties, fat grafting has become an increasingly popular procedure in both esthetic and reconstructive surgery[7]. However, its role in tendon healing remains under investigation, particularly in experimental settings.

Aim of the work :

This study aimed to-evaluate the role of autogenous fat grafting in preventing postoperative tendon adhesion after Achilles tendon repair in male albino rats.

METHODS

This prospective experimental study was conducted at the Sobhi Hweidi Microsurgery Unit (SHMU), Plastic and Reconstructive Surgery Department, Faculty of Medicine, Zagazig University, Egypt. The study period extended from August 2023 to October 2024. A total of twenty-six healthy

young adult male albino rats, weighing between 200 and 350 grams, were included. All animals were obtained from a controlled microsurgery laboratory to ensure standardized conditions and minimize experimental variability. Female rats and those weighing outside the specified range were excluded. The study protocol was reviewed and approved by the Institutional Animal Care and Use Committee (ZU-IACUC) of the Faculty of Medicine, Zagazig University (Approval No: 3/F/99/2023). All procedures were conducted in compliance with institutional ethical standards, the principles of the Declaration of Helsinki, and the European Community guidelines for the care and use of laboratory animals.

The sample size was determined using Open Epi-Info software, based on previously reported data showing a mean collagen deposition of 0.9 ± 0.4 in the PRP group Rajabi et al. [8] with a power of 80% and a 95% confidence interval.

Operational design:

Each animal contributed both limbs to the study, yielding a total of fifty-two tendons. The right tendo-Achilles was assigned to the experimental group (Group A), in which the tendon was completely transected and subsequently repaired using the Modified Kessler suturing technique, followed by circumferential application of an autologous fat graft harvested from the inguinal region. The left tendo-Achilles served as the control group (Group B), in which the tendon was transected and repaired using the Modified Kessler suturing technique without the application of any adjunctive material.

All procedures were performed under strict aseptic conditions using a 2.5× magnifying loupe. Anesthesia was induced with intraperitoneal injection of ketamine (25 mg) and xylazine (10 mg) per ml at a dose of 0.1 ml/100g body weight, with supplementation as required. Both hind limbs and inguinal regions were shaved and disinfected with 10% povidone-iodine solution prior to surgery. The animals were placed supine on a rodent operating board to facilitate fat harvesting from the inguinal region Figure 1A.

Surgical Technique

Following induction of anesthesia and preparation of the operative field, an inguinal incision was made and the inguinal fat pad was carefully dissected (Fig. 1A). The harvested fat was immediately placed in normal saline to maintain viability (Fig. 1B). The donor wound was then closed using vertical mattress sutures with 5/0 Prolene on a cutting needle (Fig. 1C).

Subsequently, the rats were positioned prone on a rodent operating board to allow exposure of both tendo-Achilles tendons. In Group A (right side), a longitudinal incision was made over the tendo-Achilles tendon (Fig. 1D), which was dissected and transected 5 mm proximal to its calcaneal insertion using a No. 15 scalpel blade (Fig. 1E). The tendon was repaired under 2.5× magnification using the Modified Kessler four-strand technique with 6/0 Prolene on a round needle (Fig. 1F). The harvested inguinal fat was then minced into small fragments using a No. 11 scalpel blade (Fig. 1G), and the diced fat was circumferentially applied around the repaired tendon prior to skin closure (Fig. 1H). In Group B (left side), the tendo-Achilles tendon was transected and repaired with the same technique but without the application of autologous fat grafting. Finally, skin closure of the hind limb wounds was achieved using simple interrupted sutures with 6/0 Prolene on a cutting needle, followed by splinting and dressing (Fig. 2). However, the splints were removed by the animals themselves shortly after regaining consciousness.

Clinical Follow-up:

After recovery from anesthesia, each rat was housed in a separate cage and monitored daily under veterinary supervision throughout the follow-up period of six weeks. Animals were carefully observed for feeding behavior, grooming activity, wound healing, and daily dressing changes. Once complete skin healing was achieved, the rats were allowed to live in groups until the end of the study period. Of the twenty-six operated rats, twenty-four survived, while two animals died during the follow-up period. During this period, gait patterns and the active range of motion of the ankle joint were assessed through

observation. Most rats demonstrated restoration of normal locomotion with full active range of motion.

At 45 days postoperatively, the surviving rats were humanely euthanized by pentobarbital overdose. Passive range of motion of the ankle joint was then examined, followed by dissection of both repaired tendo-Achilles tendons along with the overlying skin and surrounding soft tissues (Figure 3A, B). Notably, the fat-grafted right tendons (Group A) exhibited minimal adhesion formation compared to the control left tendons (Group B). In addition, remnants of the grafted fat tissue were clearly identified surrounding the repaired tendon (Fig. C).

Histological Evaluation

At the end of the follow-up period, the Achilles tendons from both limbs, along with the overlying skin, were carefully dissected, marked, and prepared for histopathological evaluation. The specimens were preserved in 10% buffered formol-saline and subsequently processed using an automated tissue processor. The processing protocol included a two-step fixation and dehydration procedure. Fixation was performed by immersing tissues in 10% buffered formalin for 48 hours, followed by removal of the fixative with distilled water for 30 minutes.

Dehydration was carried out by passing the tissues through a graded series of alcohol concentrations, starting with 70% alcohol for 120 minutes, then 90% alcohol for 90 minutes, and finally two cycles of absolute alcohol, each lasting one hour. Following dehydration, tissue clearing was performed in several changes of xylene: first in a mixture of 50% alcohol and 50% xylene for one hour, followed by immersion in pure xylene for 90 minutes. The samples were then impregnated with molten paraffin wax, embedded, and blocked.

Paraffin sections of 4–5µm thickness were prepared and stained using hematoxylin and eosin (H&E), Masson's trichrome, and toluidine blue stains according to previously published protocols. Stained sections were examined microscopically to assess degenerative changes, necrosis, apoptosis, metaplasia, fibroplasia, inflammatory cell

infiltration, granuloma formation, and regenerative processes.

Statistical analysis

All collected data were coded, revised, and entered into the Statistical Package for the Social Sciences (RStudio, version 2.3.2) for analysis. Qualitative variables were expressed as numbers and percentages, whereas quantitative variables with parametric distribution were presented as mean, standard deviation (SD), and range. For quantitative variables with non-parametric distribution, data were expressed as median and interquartile range (IQR). The Shapiro–Wilk test was applied to assess the normality of distribution for quantitative variables. For inferential statistics, the dependent (paired) t-test was employed to compare two matched groups with quantitative data of parametric distribution, while the Wilcoxon signed-rank test was used for comparisons involving non-parametric quantitative data. The confidence interval was set at 95%, with an accepted margin of error of 5%. Accordingly, a p-value < 0.05 was considered statistically significant, and a p-value < 0.01 was regarded as highly significant.

RESULTS

A total of 26 rats were included in this study. Two animals died during anesthesia, while the remaining 24 completed the experimental period. During the follow-up period, there was no statistically significant difference in the time of healing, recovery, or gait restoration between the fat-grafted right Achilles tendons (Group A) and the control left tendons (Group B). Gait assessment using the walking track showed restoration of normal locomotion and good recovery of both hind limbs in all surviving rats. During specimen collection, dissection of the fat-grafted tendons was easier, with less fibrosis and adhesion to surrounding tissues.

Clinical Recovery

Recovery degrees at 2, 4, and 6 weeks are summarized in Table 1. At two weeks, partial recovery was more frequent in Group A compared to Group B (8 vs. 2), although this difference was not statistically significant ($p = 0.722$). By four weeks, total recovery was observed in four rats in Group A versus two in Group B ($p = 0.560$). At six weeks,

total recovery was higher in Group A (20 vs. 16), whereas partial recovery was lower (4 vs. 8). However, statistical analysis showed no significant differences between the two groups at any time point ($p = 0.318$).

Histopathological Findings

Group A (*Fat-grafted Achilles tendons*)

Histological examination of fat-autografted tendons revealed marked reparative processes. The musculoskeletal and synovial attachments appeared intact and free of fibroplasia or inflammatory reactions. Tenocytes were remodeled, regularly aligned in parallel, and in some areas demonstrated focal cartilaginous transformation potential. At the peri- and para-tendinous regions where the adipose graft was placed, metaplastic stem cell changes with focal fibroblastic transformation were identified (Fig. 4). Morphometric analysis demonstrated progressive healing and tissue remodeling with increased deposition of collagen type I interspersed with lower amounts of collagen type III. The inflammatory and vascular changes were mild, and degenerative or destructive reactions were minimal. Image analysis revealed a decline in inflammatory cells, averaging 47 per 5 HPFs, including neutrophils (6), lymphocytes (26), and macrophages (15) (Fig. 5). Using Masson's trichrome staining, collagen fibers showed parallel orientation with prominent deposition of collagen type I (thick, dark-blue fibrils) compared to type III (thin, faint-blue fibers) (Fig. S1). Quantitative morphometric analysis confirmed significantly higher percentages of type I collagen deposition in fat-grafted tendons compared with controls.

Group B (*Non-fat-grafted Achilles tendons*)

Sections from the control left Achilles tendons demonstrated moderate tendinopathy with peri- and epitenon fibroblastic reactions, partial adhesions to surrounding gastrocnemius muscle fibers, and marked degenerative and lytic changes in tenocytes. Multifocal infiltration of lymphocytes and plasma cells was observed in the interstitial tissue (Fig. S2). Additional findings included inflammatory edema, fibroplasia at adhesion sites, interstitial edema, vascular dilation, and inflammatory infiltrates.

Masson's trichrome staining of control tendons showed unorganized collagen fiber orientation with lower collagen type I and higher collagen type III deposition (Fig. S3). Morphometric image analysis revealed a significantly higher inflammatory cell infiltrate in control tendons (average 90/5 HPFs) compared with fat-grafted tendons (47/5 HPFs). Differential cell counts demonstrated increased polymorphonuclear cells (41 vs. 13), lymphocytes (30 vs. 24), and macrophages (19 vs. 11) in the control group.

Statistical Comparisons

Quantitative morphometric analysis demonstrated significant differences between the fat-grafted (right) and control (left) Achilles tendons. Polymorphonuclear cells were markedly reduced in the right TA (range 7–19, mean±SD = 13 ± 4) compared to the left TA (range 28–41, mean±SD = 34±4; $p < 0.001$).

Table 1. Comparison of tendon recovery degree between Group A (fat-grafted) and Group B (control) at 2, 4, and 6 weeks post-repair

Timepoint	Recovery Degree	Group A (Fat Graft)	Group B (Control)	P-value (Fisher Exact Test)
2 weeks	No recovery	16	22	0.722
	Partial recovery	8	2	
	Total recovery	0	0	
4 weeks	No recovery	10	14	0.560
	Partial recovery	10	8	
	Total recovery	4	2	
6 weeks	No recovery	0	0	0.318
	Partial recovery	4	8	
	Total recovery	20	16	

0.001). Similarly, macrophage counts were lower in the right TA (6–19, mean±SD = 11±3) than in the left TA (8–22, mean±SD = 17 ± 4; $p < 0.001$). By contrast, lymphocyte counts showed no statistically significant difference between groups (right TA: 13–35, mean±SD = 24±6; left TA: 21–33, mean ± SD = 27 ± 3; $p = 0.116$). The total differential inflammatory cell count was also significantly lower in the right TA (38–57, mean ± SD = 47 ± 6) than in the left TA (65–91, mean ± SD = 78 ± 7; $p < 0.001$). In terms of extracellular matrix composition, type I collagen deposition was significantly higher in the fat-grafted group (36–64%, mean ± SD = 50 ± 7%) compared to the control group (25–39%, mean ± SD = 31 ± 4%; $p < 0.001$). These findings confirm a more favorable healing response in tendons treated with autogenous fat grafting (Table 2).

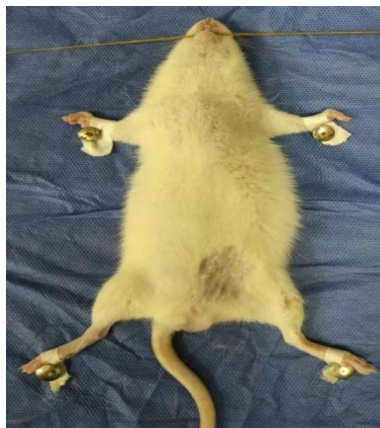
Table 2. Morphometric Analysis of Inflammatory Cells and Collagen Deposition

	Left TA (N = 24)	Right TA (N = 24)	p-value
Polymorph			<0.001
Range	28 - 41	7 - 19	
Mean ± SD	34 ± 4	13 ± 4	
Median (IQR)	34 (31, 37)	14 (10, 16)	<0.001
Macrophage			
Range	8 - 22	6 - 19	
Mean ± SD	17 ± 4	11 ± 3	0.116
Median (IQR)	18 (15, 20)	11 (8, 13)	
Lymphocyte			
Range	21 - 33	13 - 35	
Mean ± SD	27 ± 3	24 ± 6	
Median (IQR)	27 (24, 29)	(25 20, 29)	

	Left TA (N = 24)	Right TA (N = 24)	p-value
Total differential cell			<0.001
Range	65 - 91	38 - 57	
Mean $\pm$ SD	78 $\pm$ 7	47 $\pm$ 6	
Median (IQR)	78 (74, 82)	47 (43, 53)	
T1 Collagen (%)			<0.001
Range	25% - 39%	36% - 64%	
Mean $\pm$ SD	31 $\pm$ 4	50 $\pm$ 7	
Median (IQR)	30 (28, 36)	49 (46, 54)	

*: Statistically significant difference

p-value: Wilcoxon rank test



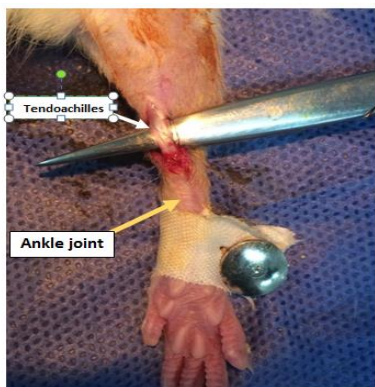
A



B



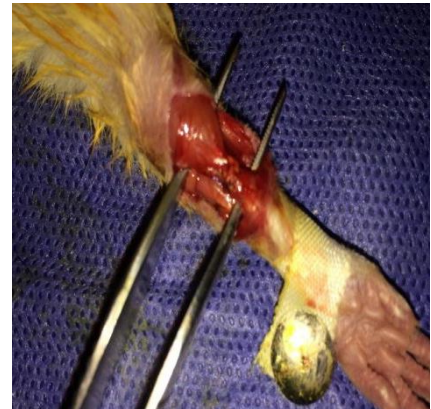
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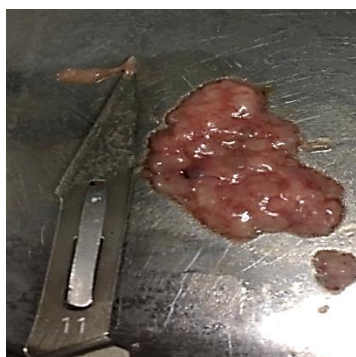
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E



F



G



H

Figure 1: 1A) Rat in supine position with shaved inguinal region. 1B) Dissected inguinal fat. 1C). Closure of inguinal incision. 1D) Dissected

tendoachilles. 1E) Transection of TA. 1F). Repaired tendoachilles. 1G). Diced inguinal fat. 1H). Diced fat placement around the repaired tendon

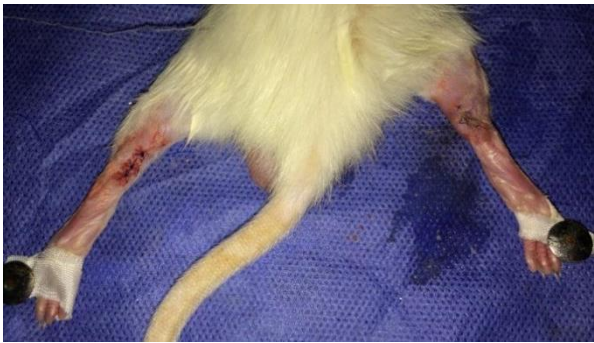


Figure 2: Closure of both hindlimbs with simple sutures



3A



3B



3C

Figure 3: (A) Marking of the skin to be excised with the underlying Achilles tendon. (B) Harvesting of the specimen during dissection. (C)

Arrow indicating the surviving grafted fat tissue surrounding the repaired tendon.

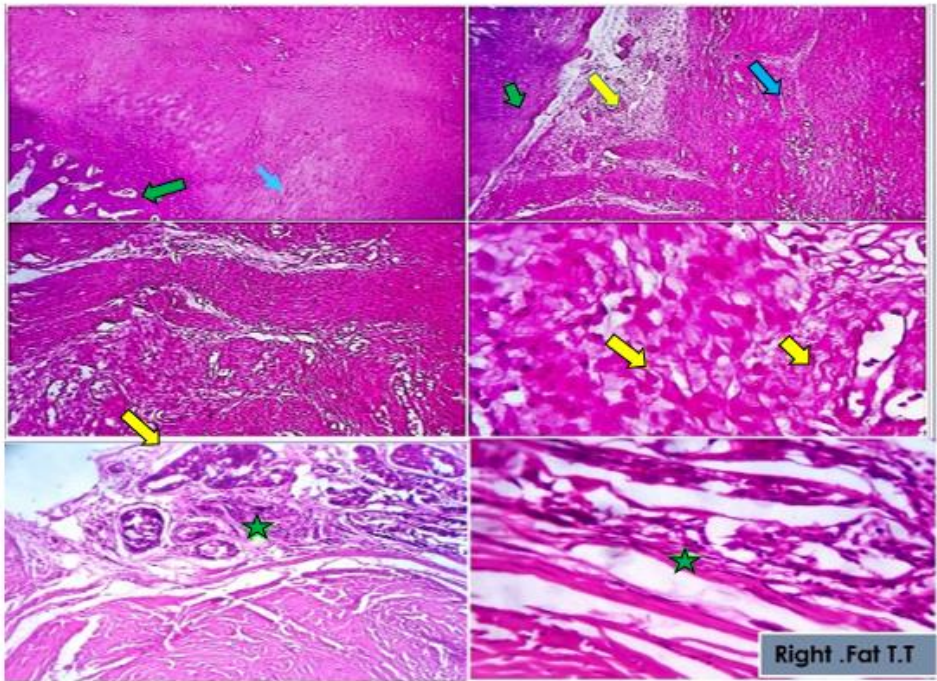


Figure 4. Photomicrographs from the right Tendochilles showing the descriptive lesional findings.

Green arrows and stars: marked reparative process as the tendon appeared healthy and less of fibroplasia or inflammatory reaction.**Light**

blue arrows: tenocytes appeared remodeled and parallel arranged with focal cartilaginous transdormation potential. **Yellow arrows:** a characteristic stem cells metaolastic changes with focal fibroblastic transformation can be seen

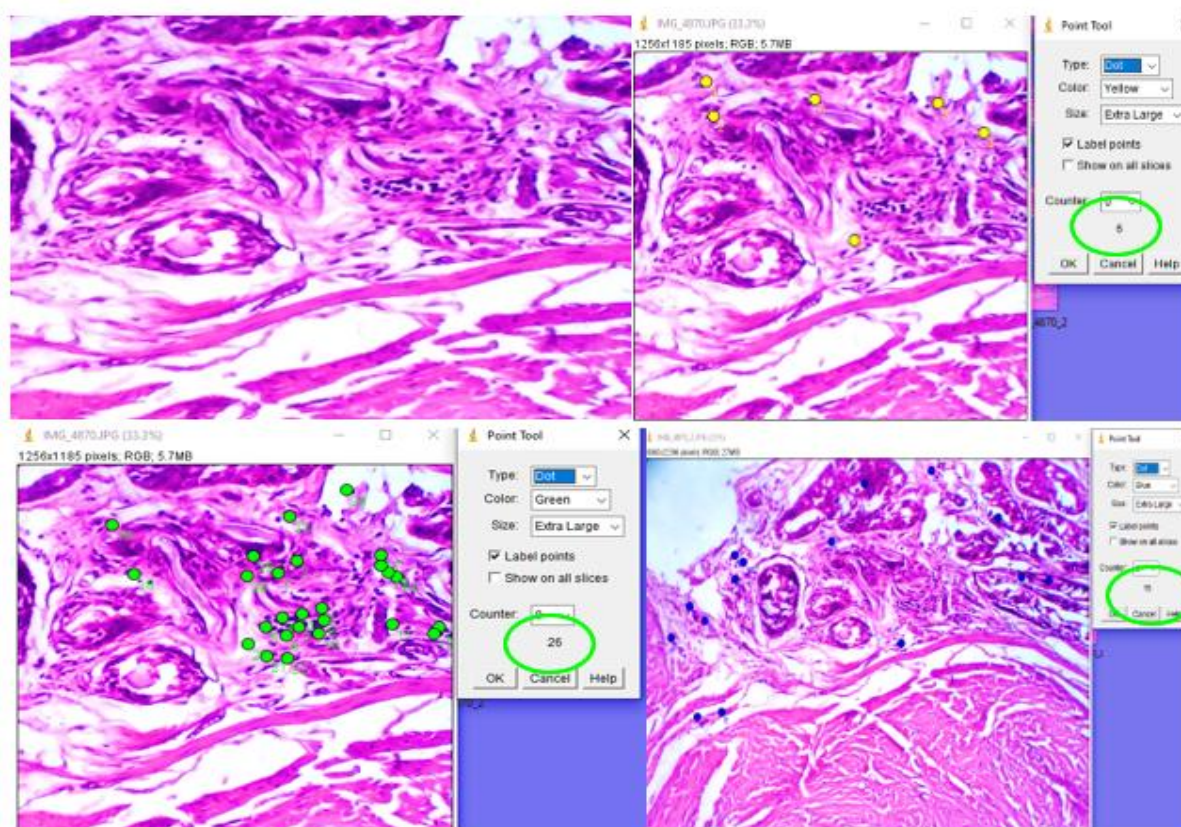


Figure 5. Micrographs showing the morphometrically estimated total and differential inflammatory cellular infiltrate in right tendon Achilles of fat autograft - received group. (**yellow dots** denotes neutrophils, **green dots** denotes lymphocytes and **blue dots** denote macrophages)

The results of the current study demonstrate that autogenous fat grafting significantly reduced polymorphonuclear and macrophage cell counts in the right TA (treated side) compared to the left TA (control side), suggesting a potential anti-inflammatory effect of fat grafting in tendon repair.

Reduction in Inflammatory Cells

The significant decrease in polymorphonuclear (PMN) and macrophage cell counts in the fat-grafted right TA compared to the control left TA (* $p < 0.001$) suggests that fat grafting may attenuate the acute inflammatory response following tendon repair.

This aligns with studies highlighting the anti-inflammatory properties of adipose-derived stem cells (ASCs). Smith et al. [9] demonstrated that ASCs modulate macrophage polarization toward an anti-inflammatory (M2) phenotype, reducing

DISCUSSION

fibrosis and scar formation.

Similarly, Anderson et al. [10] reported that stromal vascular fraction (SVF) from adipose tissue suppresses pro-inflammatory cytokines such as TNF- α and IL-6, promoting a regenerative microenvironment.

However, some studies caution that excessive suppression of inflammation may impair early tendon healing. Millar et al. [11] noted that certain inflammatory signals (e.g., TGF- β) are necessary for fibroblast activation and collagen synthesis. Thus, while fat grafting appears beneficial in reducing adhesions, further research is needed to determine the optimal balance between inflammation modulation and tissue repair.

No Significant Change in Lymphocyte Counts

The lack of a significant difference in lymphocyte counts between the fat-grafted and control tendons ($*p* = 0.116$) suggests that fat grafting may primarily influence innate immune responses rather than adaptive immunity.

This observation is consistent with Millar et al. [11], who found that ASCs predominantly regulate innate immune cells (e.g., macrophages) while having minimal effects on lymphocytes.

In contrast, Smith et al. [9] reported lymphocyte infiltration in some fat-grafted tissues, possibly due to graft resorption or immune reactions. The absence of such findings in this study may be attributed to differences in graft preparation (e.g., diced fat vs. whole tissue) or the timing of histological analysis.

Increased T1 Collagen Deposition

The significantly higher T1 collagen content in the fat-grafted right TA ($50 \pm 7\%$) compared to the control ($31 \pm 4\%$, $*p* < 0.001$) indicates improved tendon remodeling. Type I collagen is a key marker of mature tendon healing, providing tensile strength and structural integrity. These results are supported by Kim et al. [12], who observed enhanced collagen organization and mechanical strength in tendons treated with fat grafting and platelet-rich plasma. However, Martin et al. [13] cautioned that rapid collagen deposition does not always correlate with functional recovery, as disorganized fiber alignment can lead to stiffness. Biomechanical properties (e.g., load-to-failure testing) to confirm whether the increased T1 collagen translates to improved tendon function needs to be carefully assessed.

Clinical Implications

Although statistical significance was not achieved during follow-up, the fat-grafted group consistently demonstrated better recovery trends than the control group across all time points. The early improvement in partial recovery, followed by greater total recovery at later stages, suggests that autogenous fat grafting may enhance tendon healing by acting as a protective barrier and

supporting tissue regeneration, consistent with previous experimental findings [14]. The reduction in inflammatory cells and increased collagen deposition suggest that fat grafting could be a promising adjunct to tendon repair, particularly in minimizing adhesions. Smith et al. [9] reported improved range of motion and reduced pain in clinical cases of tendon adhesions treated with fat grafting, further supporting our findings. Nevertheless, challenges such as graft retention and variability in ASC potency must be addressed. Thompson et al. [15] emphasized the need for standardized fat processing techniques to ensure consistent therapeutic effects.

This aligns with previous research by James et al. [5], who highlighted the regenerative properties of adipose-derived stem cells (ASCs) in reducing inflammation and promoting tissue repair. Similarly, Bora and Majumdar [6] emphasized the role of stromal vascular fractions (SVFs) in adipose tissue, which contribute to tissue regeneration and reduced inflammatory responses. The significant decrease in total differential cell counts in the right TA further supports the hypothesis that fat grafting may minimize postoperative adhesions by modulating the inflammatory environment.

The significant decrease in polymorphonuclear cells and macrophages in the fat-grafted group ($*p* < 0.001$) suggests that fat grafting may help modulate the inflammatory microenvironment, thereby reducing fibrosis and adhesion formation. This is particularly relevant in tendon repair, where postoperative adhesions often lead to restricted mobility and functional impairment. Brown et al. [16] demonstrated in clinical cases that fat grafting following tenolysis in the foot and hand resulted in improved range of motion and reduced pain, with no recurrence of adhesions at 2-year follow-up. Similarly, Anderson et al. [17] highlighted the regenerative potential of adipose-derived stem cells (ASCs) in promoting organized tissue repair rather than scar formation. However, the variability in clinical outcomes must be acknowledged. Morris et al. [18] noted that while some patients benefit

from reduced adhesions, others may experience minimal improvement, possibly due to differences in graft quality or surgical technique. Future studies should investigate whether specific patient factors (e.g., age, comorbidities) influence the efficacy of fat grafting in adhesion prevention. One of the key challenges in translating these findings to clinical practice is ensuring consistent graft retention and survival. Mozzola et al. [19] emphasized that the therapeutic benefits of fat grafting depend heavily on the methods of harvest, processing, and delivery. For example: Standardizing these protocols will be crucial for reproducibility. Bora & Majumdar[6] proposed that SVF-enriched fat grafts, prepared under Good Manufacturing Practice (GMP) conditions, could improve consistency in regenerative applications. While this study focused on histological outcomes, the clinical success of fat grafting also depends on functional recovery. Smith et al.[9] reported that combining fat grafting in Achilles tendon repairs led to earlier return to activity and improved biomechanical strength in animal models. However, human trials are needed to confirm these benefits.

Limitations

Despite these promising findings, the study has certain limitations. The rat model used may not fully reflect the complex biomechanical and biological aspects of human tendon healing, particularly in weight-bearing scenarios. Furthermore, the relatively short follow-up period of eight weeks does not provide insight into long-term outcomes, such as graft resorption, chronic inflammatory changes, or late adhesion recurrence. Finally, variations in fat graft handling and surgical technique could influence the reproducibility of the results in clinical practice. Future research involving larger animal models, extended follow-up durations, and standardized operative protocols is required to validate these outcomes and to establish the clinical applicability of autogenous fat grafting in tendon repair.

CONCLUSION

We conclude that autogenous fat grafting exerts a beneficial role in Achilles tendon repair by modulating the local healing environment. Fat grafting significantly reduced inflammatory cell infiltration, particularly neutrophils and macrophages, and promoted collagen remodeling with organized type I collagen deposition, which supports stronger and more physiological tendon regeneration. In addition, the graft functioned as a biological barrier, limiting fibroblastic adhesions between the tendon and surrounding tissues. Although functional recovery did not reach statistical significance, a consistent trend toward faster and more complete restoration of gait was observed in the fat-grafted group, highlighting its potential as an adjunctive technique for tendon repair.

Conflict of Interest: There are no conflicting interests, according to the authors.

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Availability of the data: Upon reasonable request, the associated author will make the datasets created and/or examined during the current work available.

Authors contribution: In addition to writing and getting the book ready for publication, the writers were in charge of gathering and analyzing the data. The final version was examined and approved by all authors.

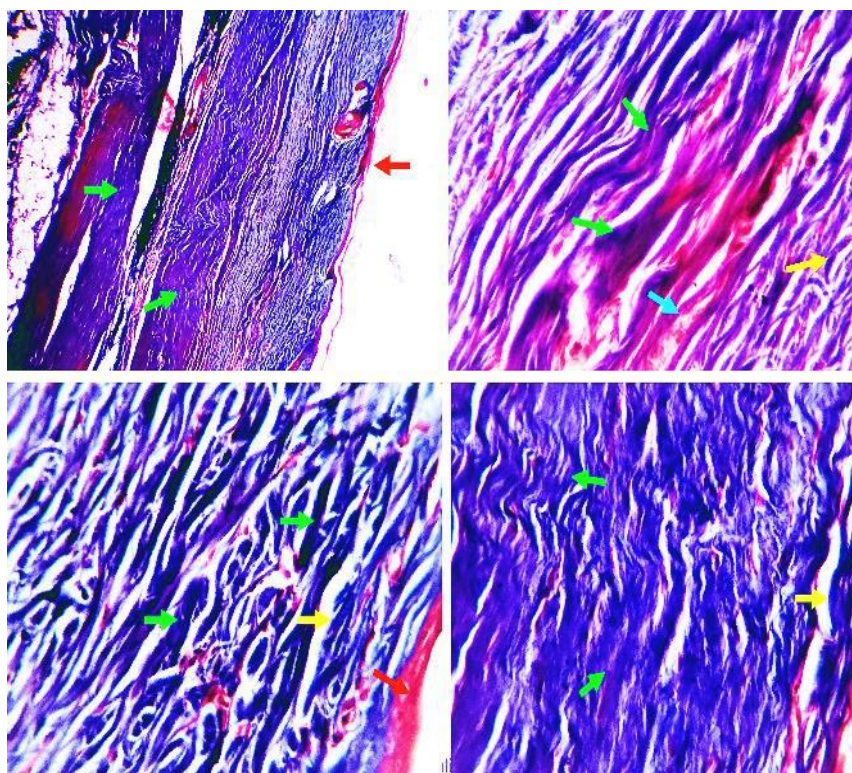
Supplementary file: figure S1,S2,S3

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SUPPLEMENTARY FILE:



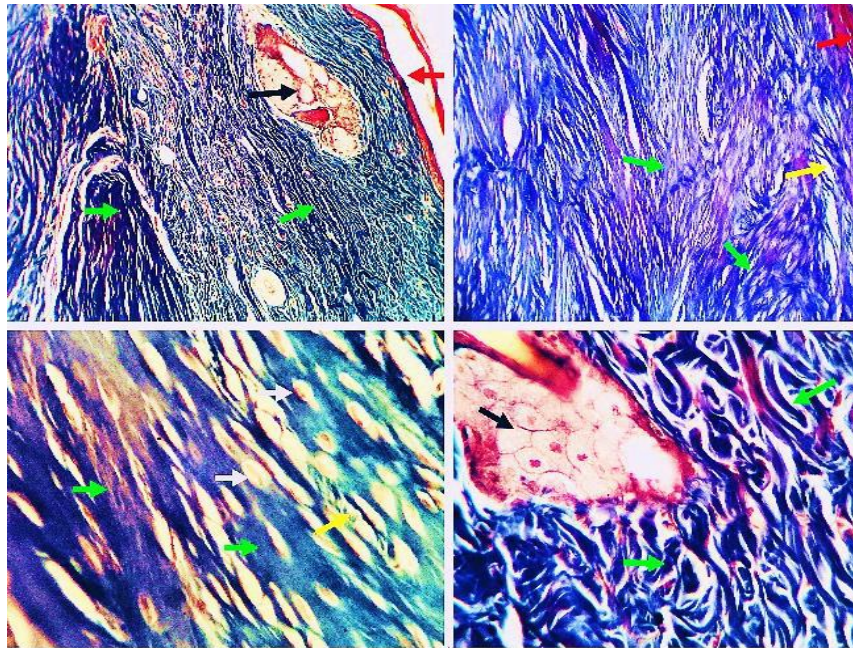


Figure S1 . Photomicrograph from rat's tendon Achilles of right fat- autograft treated groups showing: **green arrows** : parallel orientation of collagen fibers which appears with more collagen type

I (thick coarse spiraled dark blue fibrils) than type III. **yellow arrows:** collagen type I (thin faint blue fibers). **red and black arrows:** No adhesive changes are seen with normal skin surface and appendages

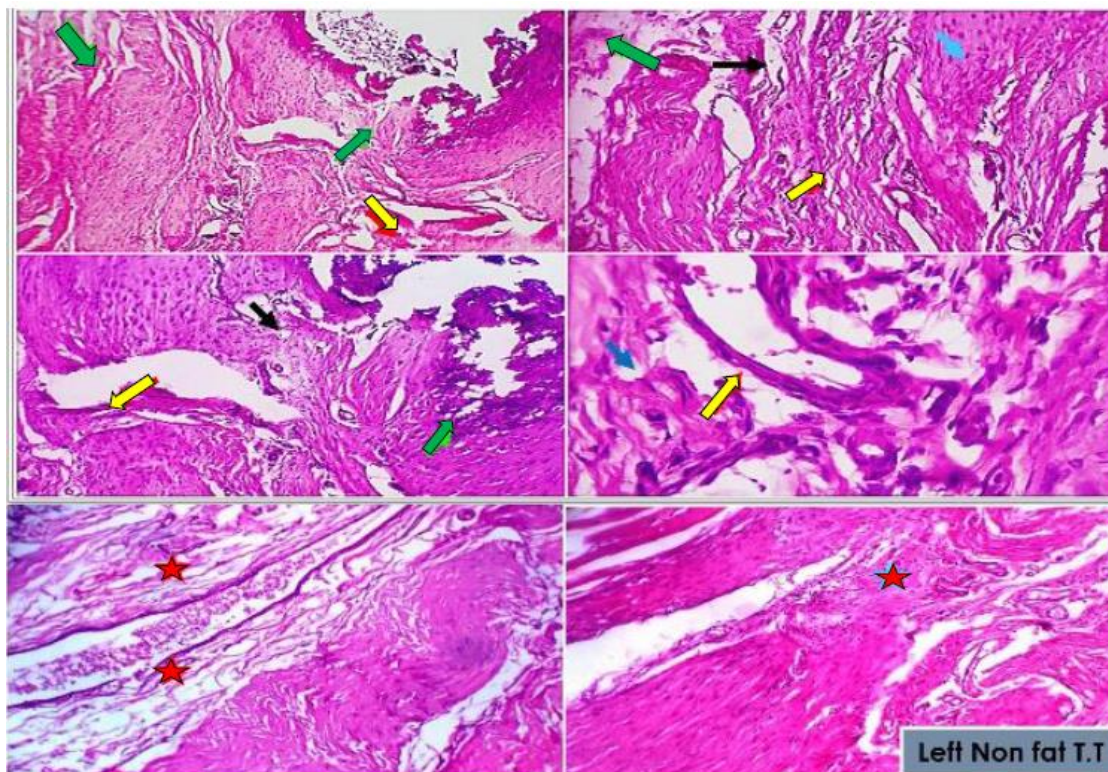


Figure S2. Photomicrographs from the left Tendon Achilles showing the descriptive lesional findings. **Green arrow:** moderate tendinopathy represented by peri and epitenon fibroblastic reactive changes with partial adhesions to the surrounding gastrocnemius muscles and inner tendon tissue. **Red stars:** inflammatory edema

and fibroplasia at the adhesion site are seen. **Yellow arrow:** marked tenocytes disruptive, degenerative and lytic changes. **Dark arrows:** mild to moderate interstitial edema, vascular dilation and infiltration of round cells (lymphocytes and plasma cells) are seen in multi focal areas of the interstitial tissue.

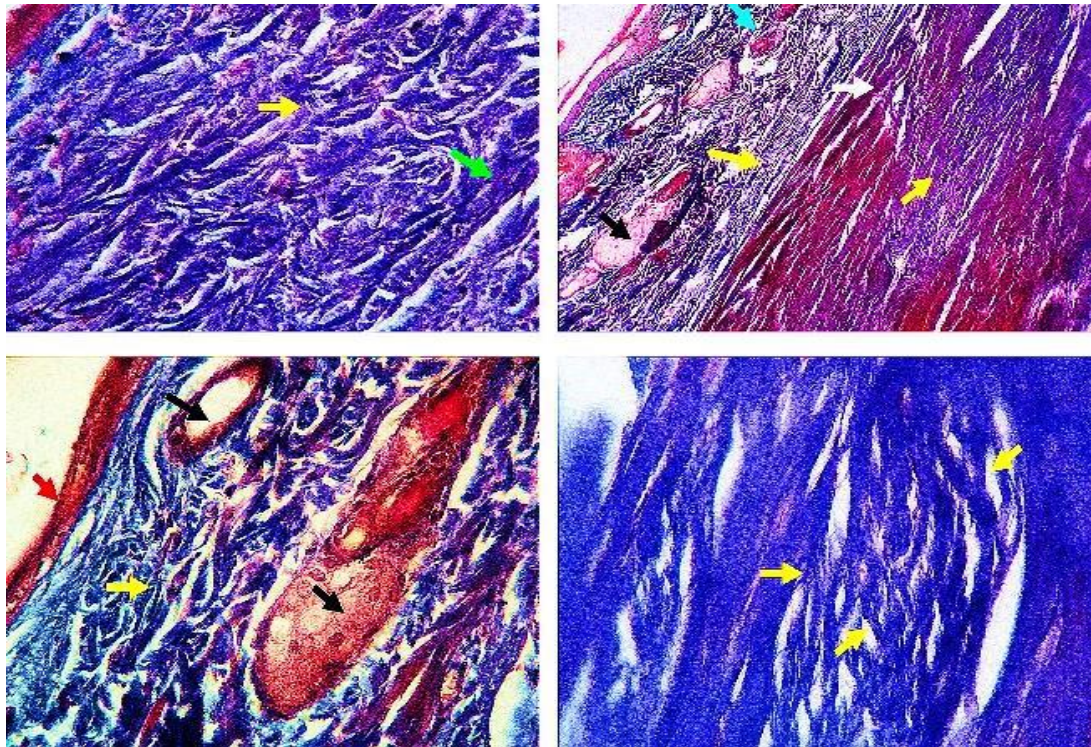


Figure S3. Photomicrograph from rat's tendon type III. **yellow circle:** subcutaneous adhesion. **green light blue arrows :** dilated capillaries. **red arrows:** denote unorganized repair of the injured tendons with less amount of the tense collagen I deposition and **yellow arrows:** more collagen are also seen.

Citation

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