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■ **FOOT AND ANKLE**

The Achilles tendon and the retrocalcaneal bursa

AN ANATOMICAL AND RADIOLOGICAL STUDY

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Objectives

Inflammation of the retrocalcaneal bursa (RB) is a common clinical problem, particularly in professional athletes. RB inflammation is often treated with corticosteroid injections however a number of reports suggest an increased risk of Achilles tendon (AT) rupture. The aim of this cadaveric study was to describe the anatomical connections of the RB and to investigate whether it is possible for fluid to move from the RB into AT tissue.

Methods

A total of 20 fresh-frozen AT specimens were used. In ten specimens, ink was injected into the RB. The remaining ten specimens were split into two groups to be injected with radiological contrast medium into the RB either with or without ultrasonography guidance (USG).

Results

In specimens injected with ink, diffusion outside the RB was observed with staining of the anterior portion of the AT. In eight contrast-injected specimens (five USG, three non-USG), a similar localised diffusion pattern was observed, with the contrast identified superiorly and anteriorly. In two contrast-injected specimens (non-USG), the diffusion pattern was more extensive.

Conclusion

This study confirmed the existence of connections between the RB and the AT, especially rich in the anteroinferior portion of the tendon, which should be considered a weak zone for substances injected into the RB. We hypothesise that this part of the AT might be most vulnerable to rupture after corticosteroid injections.

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Article focus

■ This study aims to assess whether fluid injected into the retrocalcaneal bursa can spread outside and infiltrate the Achilles tendon.

■ In addition, this study also aims to describe anatomical connections between the retrocalcaneal bursa and the Achilles tendon.

Key messages

■ This study confirms that fluid may spread from the retrocalcaneal bursa to the Achilles tendon.

■ This study describes the anatomical weak zones through which fluid spread can occur.

■ The results of this study will allow for better analysis of the possible spread of corticosteroids from the retrocalcaneal bursa into Achilles tendon tissue.

Strengths and limitations

■ This anatomical study accurately describes how fluid injected into the retrocalcaneal bursa may diffuse outside of it and into the Achilles tendon.

■ The study provides data on the diffusion of fluids in real time.

■ Although the chemical properties of the contrast medium and corticosteroids are different, both are small molecular, water soluble agents and their spread should be similar.

Introduction

The retrocalcaneal bursa (RB) is a fluid-filled space located between the anteroinferior wall of the Achilles tendon (AT) and the posterosuperior surface of the calcaneum. The anterior margin of the RB is composed of fibrocartilage, the posterior margin is the paratenon of the Achilles tendon and the superior margin is made up of adipose tissue.[1] The RB lies in the region of the AT insertion into the calcaneum, and its function is to reduce friction associated with the tendon's movement in surrounding tissues.[2]

Inflammation of the retrocalcaneal bursa' is a common clinical problem and results in localised pain, tenderness, and swelling.[3] Disorders of the RB are a heterogeneous group of pathologies related to the region of the AT's attachment to the calcaneus, and are classified as either retrocalcaneal bursitis or superficial calcaneal bursitis.[4]

Treatment of bursitis may include the use of non-steroidal anti-inflammatory drugs, cryotherapy and shoe modification. In more severe cases, localised corticosteroid injections may be used to reduce inflammation.[5-8] However, steroid treatment has been reported to increase the risk of AT rupture.[6,7]

There have been few detailed anatomical investigations of the RB, despite the fact that such knowledge is clinically relevant due to its association with the AT.[9] A recent cadaveric study by Turmo-Garuz et al[8] described a connection between the RB and the AT in three specimens. The presence of such a connection could explain the impact of RB corticosteroid injections on the AT, however, the study was limited by a small sample size and was performed using India ink injections. Therefore, we decided to conduct a larger cadaveric investigation using both traditional (India ink injections) and radiologial methods to reveal any potential link between the RB and the adjacent AT.

Iopromide, in contrast to India ink, is a water-soluble compound and should better replicate tissue distribution of water soluble corticosteroids used in the treatment of retrocalcaneal bursitis.[10,11] Our hypothesis is that iopromide contrast medium injected into the RB will penetrate the AT in a similar manner as corticosteroids and by taking time interval radiographs we would be able to assess its spread into the surrounding tissues. In addition, we wanted to describe, if present, the pattern of fluid movement and the weak zones around the RB that allow for fluid diffusion.

Materials and Methods

Materials. A total of 20 fresh-frozen AT specimens (all male: ten left, ten right; mean age 49.7 years, SD 11.2) were obtained up to 24 hours after autopsy from the local Department of Forensic Medicine, Jagiellonian University Medical College. The obtained specimens consisted of the posterior half of the calcaneum with the AT attached and part of the triceps surae complex. Specimens with macroscopically visible injuries to the AT; congenital abnormalities of the lower limb; or a history of surgery to ankle or heel regions were excluded. The research protocol was approved by the Ethics Committee of Jagiellonian University Medical College. The study has been performed in accordance with the ethical standards established in the 1964 Declaration of Helsinki and its later amendments.

Dissection. All specimens were thawed for eight hours at room temperature (25°C) prior to investigation. The connective tissues surrounding the triceps surae complex were carefully removed, the triceps surae was elevated and the space between the calcaneum and AT was exposed. During the dissection, particular attention was paid to avoid damage to the walls of the RB and adjacent tissues. For all injections, the specimens were positioned to mimic a patient in the prone position - the calcaneum was directed upwards with a slight upward inclination of the AT.

India ink injection. Ten specimens (five left, five right) were injected with India ink as described by Turmo-Garuz et al.[8] After the superior margin of the RB had been identified, the RB was injected with 2 ml of India ink under ultrasonography (US) guidance. After one hour, the specimen was dissected and particular attention was paid to changes in the colour of tissues due to dispersal of the India ink. The area where the ink was visible was measured using an electronic caliper (Mitutoyo Corp., Kawasaki, Japan) with the superior border of the calcaneum used as a reference point.[6]

Iopromide injection. In a further ten specimens (five left, five right) iopromide was injected to radiologically assess spread of the contrast medium when injected into the RB. The iopromide injections were randomly allocated into two groups – either with (five specimens) or without US guidance (five specimens).

In the US guidance group, an 18 MHz linear probe (MyLab 25 Gold; eSaote, Florence, Italy) was used. The probe was positioned over the RB and in line with the Achilles tendon at the level of the calcaneal tuberosity (supplementary figures and video). A needle inserted with the iopromide syringe attached through an extension line to avoid displacing the needle during the injection; 2 mL of iopromide was then carefully injected into the RB. In the second group without US guidance, the needle was placed according to anatomical landmarks,[11,12] and the iopromide injected as described.

Radiological investigation. Prior to contrast injection, radiographs of all specimens were obtained (Fig. 1) in order to compare with post-injection images. After contrast injection, an x-ray image intensifier (Shimadzu Corp., Kyoto, Japan) was used to visualise fluid migration within the specimens on a lateral plane. The radiographs
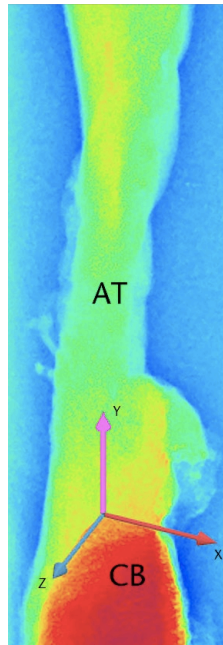
Fig. 1

Radiograph of an Achilles tendon specimen before contrast injection (CB, calcaneus; AT, Achilles tendon; x arrow, anterior direction; y arrow, superior direction; z arrow, depth).

Fig. 2

Anterior view of an Achilles tendon specimen with trace of India ink diffusion.

were taken at the following time points: prior to injection; immediately after injection (time 0); five minutes; 30 minutes; and 60 minutes post-injection. All images were analysed using Ashvins CD Viewer (Ashvins, Heidelberg, Germany) and ImageJ software (National Institute of Health, Bethesda, Maryland).[12]

Results

India ink injection. In the ten specimens that were injected with India ink, a similar pattern of ink spread was observed. Immediately after injection, the RB filled with ink. After one hour, the ink was observed to have penetrated outside of the RB, staining the anterior portion of the AT and the adipose tissue in Kager's triangle. Ink was observed to have spread up to 1.5 cm (mean 1.28 cm, 1.1 to 1.5) superiorly from the superior border of the calcaneum, and forward from the anterior surface of the AT into the surrounding connective tissue (Fig. 2).

Contrast injection. In eight specimens (all five from the USG group, and three from the non-USG group), a similar spread of the iopromide was observed. The radiographs obtained immediately after contrast injection showed the RB was filled, the borders were well defined, and no leakage of contrast outside the RB was seen (Fig. 3a). After the first five minutes, the contrast started to diffuse around the RB, mostly in the anterior and superior directions (weak zone), particularly in the region adjacent to the calcaneum, creating a strawberry-like pattern with the RB in the centre (Fig. 3b). At 30 minutes post-injection, the contrast had spread and infiltrated into further portions of

the AT, including adjacent connective tissue. The spread was mostly in the superior (mean 1.65 cm, 1.3 to 1.7, area 1.1 cm^2) and anterior directions, reaching the anterior surface of the AT, at the level of the superior border of the calcaneum (Fig. 3c). At one hour post-injection, the iopromide had infiltrated the AT up to 1.9 cm (mean 1.79 cm, 1.4 to 1.9, area 1.9 cm^2) above the superior border of the calcaneus. The diffusion in the most anterior portion (anatomical position) of the AT extended up to 1.2 cm (mean 1.04 cm, 0.8 to 1.2) superior to the superior border of the calcaneus (Fig. 3d).

Irregular diffusion pattern. In two tendon specimens (both from the non-USG group), just after injection, a trace of contrast infiltration could be seen as a rectangular area (3.3 cm x 0.8 cm, area 3.1 cm^2), of which the anterior wall was the anterior border of the AT. The posterior wall of this rectangle was a line running parallel to the fibres of the AT, located about 0.7 cm from the posterior border of the tendon. The inferior wall was made up of the calcaneum, and the superior wall was located 3.3 cm (mean 3.1 cm, 2.9 to 3.3) above the superior border of the calcaneum (Fig. 4a). At five minutes post-injection, spread of contrast had progressed 0.6 cm in the superior direction and in addition, the contrast started to spread in the direction of the posterior surface of the AT (Fig. 4b). At 30 minutes post-injection, the contrast had reached the posterior surface of the AT. The whole tendon was infiltrated by contrast that had spread from the RB extending up to 4.1 cm (mean 3.9 cm, 3.7 to 4.1; area 6.1 cm^2) from the superior border of the calcaneus (Fig. 4c). At 60 minutes post-injection, contrast had spread from the RB and extended up to 4.2 cm (mean
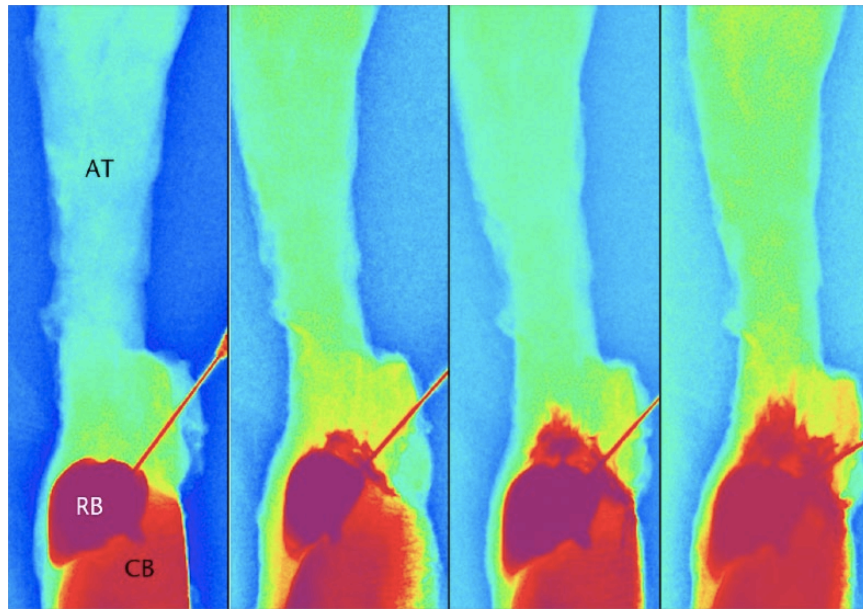
Fig. 3

Radiographs demonstrating a regular pattern of contrast migration in the following time periods after injection: a) 0; b) 5; c) 30; and d) 60 minutes (RB, retrocalcaneal bursa; CB, calcaneum).
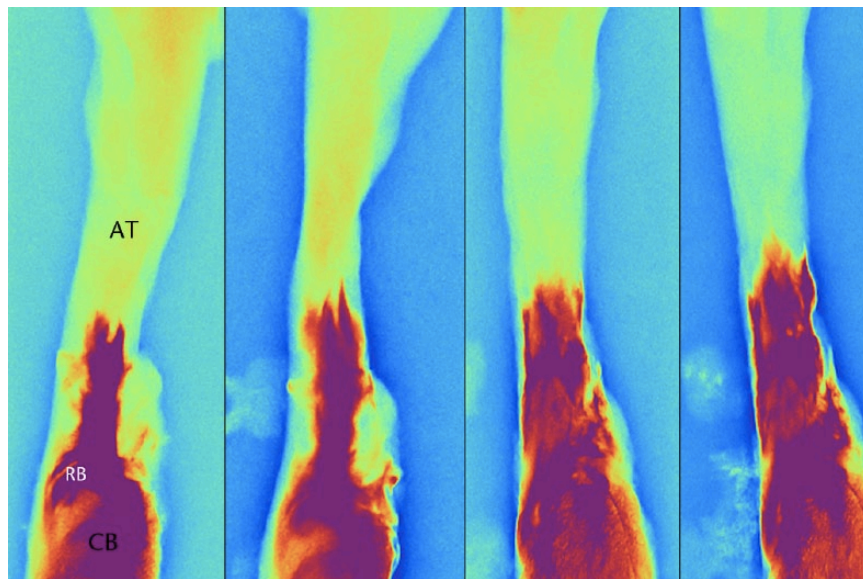

Fig. 4

Radiographs demonstrating an irregular pattern of contrast migration in the following time periods after injection: a) 0; b) 5; c) 30; and d) 60 minutes (RB, retrocalcaneal bursa; CB, calcaneum).

4.1 cm, 3.9 to 4.2). No significant progression of contrast was observed between 30 and 60 minutes (area 6.1 cm^2 *vs* 6.3 cm^2) (Fig. 4d).

Discussion

Steroid injections are often used for the treatment of severe retrocalcaneal bursitis.[11,13] However, there are reports in the literature of an increased risk of tendon rupture.[6,7,14,15] In the study by Turmo-Garuz et al,[8] the authors found some evidence of a clinically relevant link between the RB and the AT. However, the main limitation of their study was small sample size and a reliance on macroscopic anatomical findings might not be sufficient to determine the precise mechanism of fluid spreading from the RB to the adjacent AT. The aim of our study was to determine the extent and rate of fluid spread, using radiological techniques in addition to the standard India ink method.

Our findings after India ink injection suggested the existence of a link, which allowed the fluid injected into

the RB to spread to the adjacent tissues and into the AT. We investigated this possible link with use of a radiological contrast medium. Iopromide is highly soluble in water and physiological fluids and we believe that this an important mechanism for the compound to spread through the local soft tissues from the RB towards the AT, perhaps mimicking the spread of corticosteroids used in clinical practice. In contrast, India ink is a colloid of different carbon-based particles with differing size and shape bound with shellac and it is the concentration of shellac that plays a key role in ink properties and we believe this will influence the spread of the ink through the soft tissues.

Permeability of corticosteroids through tissues is related to the chemical properties and in particular the lipophilicity (logP).[16] Iopromide with different chemical properties is less permable in soft tissues[17] and this has been confirmed experimentally.[18] Intravenous injection of iopromide is distributed only in extracellular fluid. In contrast, intravenous corticosteroids are widely distributed in tissues.[19] Due to this superior permeability, we believe that corticosteroids spread from the RB to the AT will be more significant than iopromide, as observed in our study.

Our radiological results demonstrated that contrast injected into the RB will infiltrate adjacent tissues and the AT. However, the rate of fluid diffusion varied significantly in the tested specimens. Two distinct patterns of contrast spread were observed: localised – not exceeding 1.9 cm (mean 1.79 cm, 1.4 to 1.9, area 1.9 cm^2) from the RB wall, mainly in the superior and anterior direction (weak zones) in eight tendons; and diffuse – reaching up to 4.2 cm (mean 4.1 cm, 3.9 to 4.2, area 6.3 cm^2) from the calcaneum and mainly in a superior direction in the two specimens.

All injections were conducted by an experienced orthopaedic surgeon (KAT). In the five specimens injected without USG, in two samples an irregular pattern of contrast migration was noted. In these two specimens, there was significant spread of contrast along the anterior surface of the AT, perhaps due to inadvertent needle damage of the RB wall. This hypothesis is supported by the fact that in all specimens injected under USG, there were no cases of abnormal diffusion, and the pattern of contrast penetration was similar in all the specimens. If the RB wall was perhaps injured because USG was not used, the spread of contrast onto the AT was greater than in those specimens in which the RB was injected under USG. From a clinical perspective therefore, accurate RB injection with USG may help to prevent complications of corticosteroid injections such as AT rupture.

The extent of contrast spread did not significantly change between 30 and 60 minutes after injection. Therefore, we believe that after injection of the RB, spread into the local soft tissues and potentially the AT occurs in the first 30 minutes and perhaps steps should be taken by the clinician to limit the spread after injection. Local cooling of the soft tissues after injection might reduce the spread from the RB towards the AT. A reduction of local tissue temperature is associated with slower steroid diffusion[20] and this mighty be important in the 30 minutes after the injection. Further clinical studies are required to assess this hypothesis.

In conclusion, this study confirmed the existence of anatomical connections between the RB and the AT, especially rich in the anteroinferior portion of the tendon, which should be considered weak zones. We believe it is important that the injected steroid remains contained within the RB and spread out of the RB should be limited. We strongly recommend that corticosteroid injections into the RB is performed under USG to avoid inadvertent damage to the RB which might allow increased spread of steroid out of the RB and towards the AT.

Supplementary material

Photos taken before and after the procedure and a video, both of ultrasound-guided injection of dye into the retrocalcaneal bursa, are available alongside the online version of this article at www.bjr.boneandjoint.org.uk

References

1. **Canoso JJ, Liu N, Traill MR, Runge VM.** Physiology of the retrocalcaneal bursa. *Ann Rheum Dis* 1988;47:910-912.
2. **Doral MN, Alam M, Bozkurt M, et al.** Functional anatomy of the Achilles tendon. *Knee Surg Sports Traumatol Arthrosc* 2010;18:638-643.
3. **Aronow MS.** Posterior heel pain (retrocalcaneal bursitis, insertional and noninsertional Achilles tendinopathy). *Clin Podiatr Med Surg* 2005;22:19-43.
4. **van Dijk CN, van Sterkenburg MN, Wiegerinck JI, Karlsson J, Maffulli N.** Terminology for Achilles tendon related disorders. *Knee Surg Sports Traumatol Arthrosc* 2011;19:835-841.
5. **Schepsis AA, Jones H, Haas AL.** Achilles tendon disorders in athletes. *Am J Sports Med* 2002;30:287–305.
6. **Vallone G, Vittorio T.** Complete Achilles tendon rupture after local infiltration of corticosteroids in the treatment of deep retrocalcaneal bursitis. *J Ultrasound* 2014;17:165–167.
7. **Nichols AW.** Complications associated with the use of corticosteroids in the treatment of athletic injuries. *Clin J Sport Med* 2005;15:370-375.
8. **Turmo-Garuz A, Rodas G, Balius R, et al.** Can local corticosteroid injection in the retrocalcaneal bursa lead to rupture of the Achilles tendon and the medial head of the gastrocnemius muscle? *Musculoskelet Surg* 2014;98:121-126.
9. **Kachlik D, Baca V, Cepelik M, et al.** Clinical anatomy of the retrocalcaneal bursa. *Surg Radiol Anat* 2008;30:347-353.
10. **Draghi F, Robotti G, Jacob D, Bianchi S.** Interventional musculoskeletal ultrasonography: precautions and contraindications. *J Ultrasound* 2010;13:126-133.
11. **Goldberg-Stein S, Berko N, Thornhill B, et al.** Fluoroscopically guided retrocalcaneal bursa steroid injection: description of the technique and pilot study of short-term patient outcomes. *Skeletal Radiol* 2016;45:1107-1112.
12. **Mizia E, Tomaszewski KA, Lis GJ, Goncerz G, Kurzydło W.** The use of computer-assisted image analysis in measuring the histological structure of the human median nerve. *Folia Morphol (Warsz)* 2012;71:82-85.
13. **Kearney RS, Parsons N, Metcalfe D, Costa ML.** Injection therapies for Achilles tendinopathy. *Cochrane Database Syst Rev* 2015;26:CD010960.

14. **Hamilton B, Remedios D, Loosemore M, Maffulli N.** Achilles tendon rupture in an elite athlete following multiple injection therapies. *J Sci Med Sport* 2008;11: 566-568.

15. **Metcalfe D, Achten J, Costa ML.** Glucocorticoid injections in lesions of the achilles tendon. *Foot Ankle Int* 2009;30:661-665.

16. **Faassen F, Kelder J, Lenders J, Onderwater R, Vromans H.** Physicochemical properties and transport of steroids across Caco-2 cells. *Pharm Res* 2003;20: 177-186.

17. **Benet LZ, Broccatelli F, Oprea TI.** BDDCS applied to over 900 drugs. *AAPS J* 2011;13:519-547.

18. **Krause W, Schuhmann-Giampieri G, Staks T, Kaufmann J.** Dose proportionality of iopromide pharmacokinetics and tolerability after i.v. injection in healthy volunteers. *Eur J Clin Pharmacol* 1994;46:339-343.

19. **Brady ME, Sartiano GP, Rosenblum SL, Zaglama NE, Bauguess CT.** The pharmacokinetics of single high doses of dexamethasone in cancer patients. *Eur J Clin Pharmacol* 1987;32:593-596.

20. **Oren I, Fleishman SJ, Kessel A, Ben-Tal N.** Free diffusion of steroid hormones across biomembranes: a simplex search with implicit solvent model calculations. *Biophys J* 2004;87:768-779.