

# Incidence and distribution of blood vessels in punch biopsies of Palmer 1A disc lesions in the wrist

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## Abstract

**Introduction** Although the partial excision of triangular fibrocartilage complex (TFCC) tears appears to be a clinically effective technique, little is known about the ability of the central disc tears to heal. Unlike peripheral tears, central tears do not have immediate access to blood supply. The purpose of this study was to examine the incidence and distribution of blood vessels in punch biopsies of symptomatic central TFCC tissue. In addition, the study investigated if arthroscopic debridement can reach vascularized tissue to enable a reparative response of the tear.

**Materials and methods** Thirty-two patients with symptomatic central traumatic tears in the TFCC (Palmer 1A) were included in this study. The cartilage was debrided arthroscopically using a biopsy punch. The debrided tissue was then examined histologically. To visualize blood vessels, the histological sections were stained with CD 31 antibodies. The presence/absence of blood vessels was recorded on a qualitative level.

**Results** In six patients, five or more blood vessels (CD31 positive endothelial cells) could be detected. In eight patients, fewer than five vessels could be found in the periphery of the slides. In 18 patients no vessels could be

identified. The incidence of vessels in the outer region was higher than in the inner region of the peripheral area. There was no correlation found between the time of trauma and incidence of blood vessels ( $P > 0.05$ ).

**Conclusion** Only in 43% of arthroscopic debridements of Palmer 1A tears vascularized tissue could be reached.

**Keywords** TFCC · Traumatic lesion · Wrist arthroscopy · Immunohistochemistry · Specific endothelial marker

## Introduction

The triangular fibrocartilage complex (TFCC) of the wrist acts as an important stabilizer of the distal radioulnar joint (DRUJ) and the ulnar carpus [15, 16]. Wrist injuries are common [1, 4–6, 8] and central traumatic injuries of the TFCC are frequent sources of ulnar wrist pain [2]. The normal articular disc is bi-concave, with its greater concavity on the ulnar surface [9]. Furthermore, the disc is the central, thinnest portion of the horizontal part of the TFCC. Most tears affect this particular part [15]. Palmer divided TFCC injuries into two basic categories: traumatic (type I) and degenerative (type II) [15]. Traumatic lesions are classified depending on the location of the tear within the TFCC (Table 1). It is generally agreed that the TFCC consists of a fibrocartilaginous disc and surrounding fibrous structures [3, 10–12, 17, 19–21]. The peripheral 10–40% of the articular disc are well vascularized, whereas the inner (horizontal) portion is avascular. Bednar et al. suggest that tears in the periphery of the TFCC may have sufficient blood supply to enable a reparative response and, in theory, have some healing potential. Tears that occur in the centre of the TFCC do not have immediate access to blood supply and are therefore not likely to heal [2].

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**Table 1** Palmer 1A classification

1A	Central, isolated tear
1B	Peripheral tear of the TFCC from its insertion of the distal ulna
1C	Disruption of the TFCC from the volar ulnocarpal extrinsic ligaments
1D	Avulsions of the TFCC from its radial attachment on the sigmoid notch

Although the partial excision of TFCC tears appears to be a clinically effective technique [7, 14], little is known about the ability of the TFCC to heal after traumatic injuries.

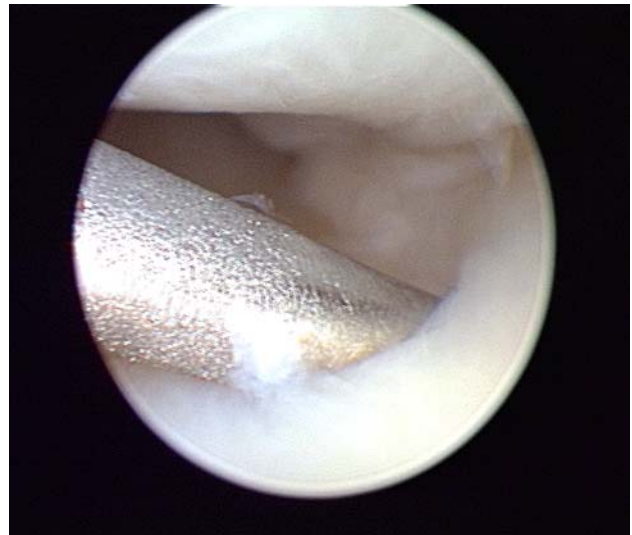
However, debridement represents a challenge to the surgeon who has to decide to what extent the area of the TFCC needs to be debrided. Unfortunately, no guidelines have yet been established to help the surgeon make this decision. In a clinical setting, it remains unknown, whether or not the surgeon ever reaches the vascularized area of the disc when debriding. This study investigated the incidence and distribution of blood vessels in punch biopsies of symptomatic Palmer 1A TFCC tears tissue [15]. The biopsy tissue was obtained during arthroscopic debridement.

The objective of our study was to find out whether or not arthroscopic debridement reaches vascularized tissue in the periphery of the disc and, as a result, enables a reparative response in traumatic Palmer type 1A tears.

## Materials and methods

### Patients

Thirty-two patients (19 men, 13 women) with a Palmer 1A lesion were included in the study (Fig. 1). All patients had suffered an accident, which involved a fall to the ground with the wrist being in a pronated-hyperextended position. All patients had a history of trauma to the wrist and complained of ulnar-sided wrist pain. All patients showed, both, a positive sign during the ulnocarpal stress test, and tenderness at the distal end of the ulna. Before arthroscopy, all patients were first treated conservatively. A brace was worn for a time of three to four weeks and antiphlogistic medication was taken. Following this conservative treatment, patients showed no signs of improvement. In our study we only included patients who knew when exactly the trauma to the wrist had occurred. Patients who were unable to determine the time of trauma were excluded from the study. Further exclusion criteria were Palmer lesions other than type 1A, patient age exceeding 40 years, a positive ulnar variance, DRUJ instability, carpal instability, and acute, carpal bone, radius, or ulna fracture. The average age at the time of operation was  $32 \pm 5.7$  years. Duration of symptoms before surgical intervention averaged  $8.8 \pm 9.7$  months.



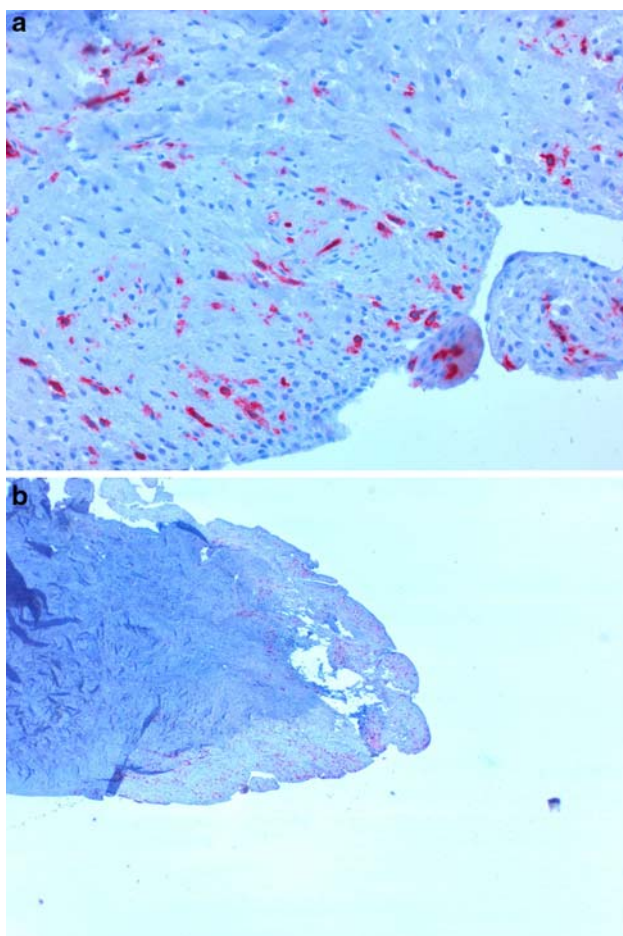
**Fig. 1** Palmer 1A tear in the triangular fibrocartilage disc with inserted probe

### Operative technique

Wrist distraction was provided by a wrist traction tower, which maintained approximately 10 lbs of distraction throughout the procedure via finger traps placed on the index, middle, and ring fingers. The wrist was distended with 5–10 mL of sterile saline solution, and the arthroscope (2.4 mm) was introduced through the 3–4 portal in accordance with standard technique [22]. In order to examine the biopsy histologically, sufficient material of the TFC central disc needed to be excised. The biopsy punch was 3 mm in diameter (Arthrex, Munich, Germany). All loose parts of the disc were routinely resected without violating the palmar or dorsal radioulnar ligament or the TFCC insertion at the base of the ulnar styloid. After completion of the procedure, the skin portals were closed with 4-0 nonabsorbable sutures. A compressive dressing and forearm-based wrist splint were applied for a duration of 2 weeks.

### Immunohistochemistry

The specimens were fixed in 4% formalin for one night and embedded in paraffin for light microscopy. Specimens were sectioned axially into slices of 3–4  $\mu\text{m}$ . After deparaffinization, the sections were stained immunohistochemically with antibodies for endothelial cells. Briefly, the appropriate sections were pre-treated with 0.2% pepsin (Sigma Chem., Deisenhofen, Germany) and subsequently incubated with the specific monoclonal primary antibody directed against CD 31 (DAKO, Hamburg, Germany). After several washings with buffer solution, the antigen-antibody complex was visualized by a biotinylated secondary antibody and a



**Fig. 2** 43% of punch biopsies contain blood vessels (staining with CD31) in the periphery. Original magnification:  $\times 200$  (a) and  $\times 25$  (b)

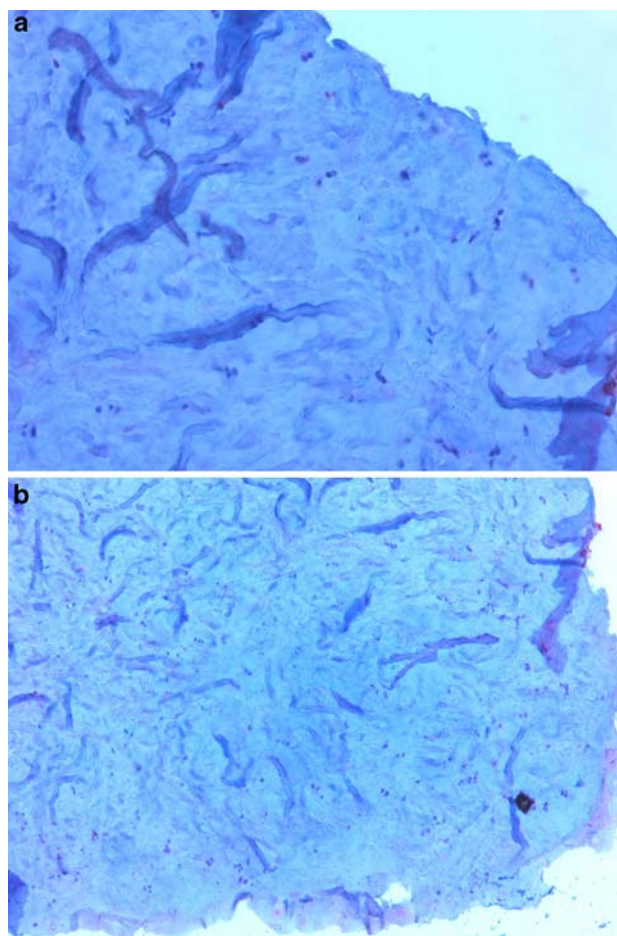
streptavidine-enzyme conjugate (LSAB-technique) with diaminobenzidine (Biogenex/DCS, Heidelberg, Germany) [13]. All sections were then analyzed under light microscopy.

#### Analysis

The presence of CD31 positive endothelial cells outlining blood vessels of different sizes was analyzed with special attention to the histo-anatomic location within the disc. Subsequently, the presence or absence of blood vessels was recorded on a qualitative level.

#### Statistical analysis

Due to the small number of patients, the Fisher exact test was used to analyze the correlation between the incidence of blood vessels and the amount of time passed since trauma. For all tests, a  $P$  value  $\leq 0.05$  was considered to be significant. Data analysis was performed using SPSS for Windows (SPSS inc. Chicago, IL, USA).



**Fig. 3** 47% of punch biopsies are free of blood vessels (staining with CD31) in the periphery. Original magnification:  $\times 200$  (a) and  $\times 100$  (b)

#### Results

In six patients, five or more vessels (CD31 positive endothelial cells) could be detected (Fig. 2). Eight patients had fewer than five vessels in the periphery of the slides. No vessels at all could be identified in 18 patients (Fig. 3). In cases where blood vessels were present, they could only be found in the outer region of the peripheral area of the biopsy. Therefore, the incidence of blood vessels increases, as one moves from the inner towards the outer region of the periphery of the disc. The incidence of blood vessels and the time passed since trauma did not correlate ( $P > 0.05$ ).

#### Discussion

Bednar et al. [2] stated that tears of the TFCC, which occur in the center of the disc, do not have immediate access to blood supply, and are not likely to heal. As a consequence, the surgeon is advised to debride the disc until vascularized



tissue in the periphery is reached. This, however, represents a challenge to the surgeon, as the exact area to be debrided is unknown in a clinical setting. To date, there are no guidelines, which could direct the surgeon. In addition, a problem could occur, if the debridement of the tear reaches too far into the periphery. Then, the dorsal or palmar radioulnar ligament could be violated and the DRUJ could become instable. However, if not enough material is debrided, parts of the tear tissue could clamp and prevent healing. As a result, the patient could experience persistent pain. So far, no study has been able to show, if debridement of Palmer 1A disc lesions can reach vascularized tissue, and provide the potential to heal. However, only one study could show that the disc has repair potential after ulna-shortening [18]. In this study, arthroscopic debridement was able to reach vascularized tissue in only 43 percent of patients with Palmer 1A tears. Fifty-seven percent of the punch biopsies contained no vessels in the periphery. None of the histological sections showed tissue of ligaments.

Further studies are needed to determine the correlation between the extent of the debrided area and the clinical outcome of Palmer 1A tears. In addition, future studies should explore whether or not the surgeon must reach vascularized tissue when debriding Palmer 1A tears.

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