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# Suture anchor reinsertions of distal biceps rupture: a histologic analysis of a torn tendon and clinical results at short- and long-term follow-up



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**Background:** Distal biceps brachii tendon (DBBT) rupture is a relatively rare injury. Nonsurgical treatment determines 30%-40% power loss of elbow flexion and up to 50% of forearm supination. Therefore, refixation of the DBBT is recommended. The DBBT is exposed to tension and compression loading. It is known that the tendon under compression might develop fibrocartilaginous metaplasia that improves the resistance to compression but reduces the resistance to tension. To test this hypothesis, the present study evaluated the presence of cartilage in DBBT samples. Furthermore, the present study evaluated the clinical and functional outcomes of anatomic reinsertion through suture anchors in a cohort of patients after 1, 3, and 5 years of follow-up.

**Methods:** Between 2011 and 2014, 21 patients with DBBT tear underwent a suture anchor reattachment. Histochemical and immunohistochemical analysis of torn samples of DBBT collected at the time of surgical repair were performed to test the presence of cartilage. During the follow-up examination, mobility, elbow radiographs, Mayo Elbow Performance Score, and isokinetic analysis were prospectively evaluated.

**Results:** Fibrocartilage was detected in all tendon samples collected. Two cases of transient paresthesia in the lateral antebrachial cutaneous nerve occurred, but they resolved in 6 weeks. There were no vascular deficits, re-ruptures, radioulnar synostoses, or infective complications at follow-up. Three patients reported loss of supination. Mayo Elbow Performance Score showed good and excellent clinical and functional results. No significant differences about strength and fatigue in flexion-supination were recorded between the surgical and contralateral side at 3 and 5 years of follow-up. Arm dominance influenced supination but not flexion.

**Conclusion:** On the basis of our results, we find that the presence of cartilage metaplasia might make the DBBT at higher risk of rupture assuming the compression loading and the hypovascular zone of the tendon. However, concerning the lack of histologic analysis of the healthy DBBT, its role in tendon pathology remains to be clearly defined. The technique of suture anchor reinsertion by a single incision was shown to be safe, with few complications and good functional results at 5 years of follow-up. No significant differences were reported between the injured and noninjured side in terms of flexion and supination isokinetic analysis, whereas arm dominance had a positive effect on supination.

The protocol, participant education and recruitment materials, and other requested documents—and any subsequent modifications—were reviewed and approved by the Department of Clinical and Molecular Science board according to the Policy of Clinical Orthopaedics, Università Politecnica delle Marche, Ancona, Italy. In this research, we evaluated the clinical and functional outcomes of patients who underwent anatomic reinsertion by a suture anchor due to the acute lesion of the distal biceps brachii. Even though we prospectively collected the data during years, these data were

retrospectively evaluated at the moment of writing the manuscript. The authors followed the principles of "good clinical practice" in accordance with Declaration of Helsinki.

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Distal biceps brachii tendon (DBBT) rupture is a relatively rare injury. It commonly occurs in the dominant arm of middle-aged men during an excessive eccentric tension.<sup>8</sup> Its incidence increases in athletes involved in competitive strength training and contact sports. Clinically, patients usually complain of a sudden, sharp, and painful tearing sensation in the antecubital region, with a palpable defect. Magnetic resonance imaging or ultrasound is helpful to make diagnosis and distinguish between partial and complete tears. Patients who underwent conservative treatment showed a remaining deficit, clinically evident in several activities.<sup>16</sup> Therefore, early anatomic reattachment is the treatment goal. Two potential mechanisms seem to be involved in the pathogenesis of DBBT rupture: the vascular supply and the mechanical impingement. It was hypothesized that the hypovascular zones of the tendon may predispose to its rupture. Furthermore, cadaveric studies showed a mechanical impingement of the distal tendon at the proximal radioulnar joint moving from full supination to full pronation.<sup>18</sup> The authors hypothesized that repeated stimuli due compression to the mechanical impingement through hypoxia could lead to the formation of a fibrocartilaginous structure characterized by rounded cells and a matrix containing type I and II collagen, chondroitin-4-sulfate and chondroitin-6-sulfate.<sup>5</sup> This could result in a tendon more resistant to compressive stress but, in the long term, less resistant to traction, predisposing it to rupture in case of excessive eccentric tension trauma.

Many surgical techniques have been described for the treatment of DBBT rupture, but there is still considerable controversy about the management of choice.<sup>9</sup> It has been well established that anatomic surgical treatment represents the gold standard technique. However, the best fixation techniques such as suture anchors, bone tunnels, interference screws, and cortical buttons remain controversial.<sup>15</sup> Clinical studies have demonstrated the advantages of a single incision, with excellent results in repair using suture anchors.<sup>13</sup> Indeed, single-incision repair showed a lower rate of posterior interosseous nerve palsy, heterotopic bone formation, and reoperation compared with double-incision.4

The aim of the present study was to evaluate the pathologic aspects of the injured tendons and in particular the presence of cartilaginous metaplasia of the tissue. Moreover, the secondary aim was to assess the clinical and functional outcomes of anatomic reinsertion of the DBBT through a suture anchor at short- and long-term of follow-up.

# Material and methods

The protocol of the present study, site-specific informed consent forms (local language and English versions), participant education and recruitment materials, and other requested documents—and any subsequent modifications—were reviewed and approved by the Department of Clinical and Molecular Science board in accordance with the Policy of Clinical Orthopaedics, Università Politecnica delle Marche, Ancona, Italy. All procedures were conducted in accordance with "good clinical practice" and Declaration of Helsinki 1964. Inclusion and exclusion criteria of patients are summarized in Table I.

### **Histologic study**

Between 2011 and 2014, tendinous samples of DBBT that were not of any medical use were harvested from 21 patients during surgery. Tissue samples of DBBT were collected at least 1 cm from the insertion. All the tendon specimens appeared macroscopically normal. The samples were obtained from the same patients included in the clinical study reported below.

#### Histochemistry

For light microscopy, specimens were fixed by immersion in 10% neutral buffered formalin solution for 24 hours and then embedded in paraffin for histologic analysis. Sections of 3-5 m thickness were obtained and stained with hematoxylin-eosin, Alcian blue pH 1.0, and Safranin O (all Bio-Optica, Milano, Italy).

#### Immunohistochemistry

Cut samples were dewaxed and rehydrated in a graded ethanol series. Intrinsic peroxidase activity was blocked by immersion in distilled water containing 3% hydrogen peroxide for 10 minutes. The following antibodies and dilutions were used: polyclonal anti-S100 protein (1:1500; Dako, Carpinteria, CA, USA), a marker of chondrocyte phenotype; polyclonal anti-type I and II collagen (1:150 and 1:100, respectively; Monosan, Uden, the Netherlands). Slides were incubated for 1 hour at room temperature and washed in Tris-buffered saline. Dako LSAB + kit was used to highlight the antigen-antibody binding, following the manufacturer's recommendations. Sections were observed with a Leica microscope (Leica Microsystems Cambridge Ltd., Cambridge, UK). For the negative control, the primary antibody was replaced with nonimmune serum.

# **Clinical study**

The present study evaluated the medical records of patients who underwent surgical treatment of DBBT injury during the period

Table I Inclusion and exclusion criteria

| Inclusion criteria                           | Exclusion criteria         |
|--|----------------------------|
| Distal, isolated, and closed biceps lesion   | Chronic injuries           |
| Less than 10 d between injury<br>and surgery | Rheumatic diseases         |
| Same surgical treatment (suture anchors)     | Diabetes                   |
| Minimum postoperative follow-<br>up of 5 yr  | Previous elbow pathologies |

between January 2011 and November 2014. The mean age of the patients at the time of trauma was 45 years (range, 32-54 years). All patients reported an eccentric muscle contraction against a heavy load in a semiflexed position as a cause of lesions. Lesions were diagnosed by physical examination (hook test), ultrasound, and magnetic resonance imaging, confirming a complete rupture of the DBBT.

Patients were operated by a single surgeon; he used a single anterior incision via the Henry approach (L-shaped incision at cubital flexion crease) to reattach the DBBT to the radial tuberosity through a single titanium suture anchor (5.5 mm; Stryker Italia, Rome, Italy) loaded with 2 high-resistance wires (not resorbable Force Fiber Stryker, Mahwah, NJ, USA). Postop immobilization was for 4 weeks in an above-elbow plaster cast with 90° of elbow flexion with the forearm in slight supination. This treatment was followed by a specific rehabilitation program under the supervision of a physical therapist team specialized in upper limb surgery. Patients started a controlled active elbow mobilization and muscle strength exercises 8 weeks after surgery. Return to normal daily activities was achieved 12-16 weeks after surgery.

We assessed prospectively the clinical and functional outcomes of the patients at 1-3 and 5 years of follow-up. At followup, patients were clinically evaluated through elbow radiographs, goniometer (measuring the degrees of flexion/extension and pronation/supination), and Mayo Elbow Performance Score (MEPS). Concentric strength (as measured by peak torque) and endurance (as measured by total work) of the elbow flexors and supinators were tested using an isokinetic dynamometer. Data analysis was carried out through a software system Biodex System 3 (Biodex Medical System; Brookhaven R&D, Shirley, NY, USA). Measures were compared between the injured and noninjured side. The noninjured arm was analyzed first. Peak torque (N/m) and total work (J) were respectively evaluated through a set of 3 reps at 90°/s of angular speed and a set of 30 reps at 240°/s of angular speed. Patients were divided into subgroups following the dominant and nondominant side of injury. Subsequently, we evaluated the deficit in terms of peak torque and total work between the operated and nonoperated arm in relation to dominance.

# Statistical analysis

Data were collected and organized using Excel (Microsoft, Redmond, WA, USA). Categorical variabilities were expressed in numbers and percentages. Continuous variabilities were expressed by mean and standard deviation. Data from groups were compared using the Mann-Whitney test, *t*-test, and Fisher exact test when appropriate. Statistical analyses were performed using SPSS (version 21.0; IBM, Armonk, NY, USA). A P value <.05 was considered as significant.

# Results

#### **Histologic results**

All DBBT specimens collected from surgery showed matrix disorganization, fatty degeneration, and numerous areas of cartilage, consisting of rounded cells, surrounded by collagen fibers. Chondrocyte-like cells were clustered in groups of 3 to 10 or randomly dispersed in the matrix. Safranin-O staining showed evident metachromasia of the cartilaginous matrix (Fig. 1). In all the samples, several areas of the extracellular matrix stained strongly for type I and type II collagen immunolabeling (Fig. 1).

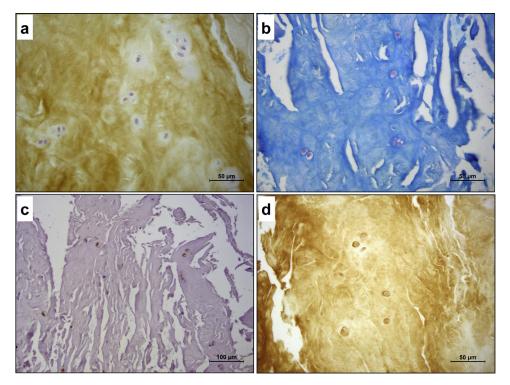
#### **Clinical results**

Twenty-one patients underwent surgery for acute rupture of the DBBT. Patients were divided into subgroups following the dominant (15 patients) and nondominant side of injury (6 patients). All patients reported an eccentric muscle contraction against a heavy load in a semiflexed position as a cause of lesions. There were no recorded intraoperative complications. Return to work with the full use of the operated limb was achieved in approximately 12 weeks (range, 10-14 weeks), and return to sports was achieved in approximately 16 weeks (range, 13-19 weeks). Two cases of transient paresthesia in the lateral antebrachial cutaneous nerve occurred, but they resolved in 6 weeks. There were no vascular deficits, re-ruptures, heterotopic ossifications, radioulnar synostoses, or infective complications at followup.

Regarding the range of motion of the injured side, the flexion and extension resulted complete in all patients, as reported in Table II. However, 14% of patients (dominant side injured) lost supination from  $10^{\circ}$  to  $20^{\circ}$  in comparison with the contralateral side.

MEPS showed good/excellent clinical and functional results at 1-3 and 5 years of follow-up. No significant differences time-dependent were reported between groups. At 5 years of follow-up, the mean of MEPS was  $93.12 \pm 6.30$  points ( $94.63 \pm 5.09$  for the dominant upper limb and  $89.43 \pm 9.26$  for the nondominant side).

Isokinetic analysis of the surgical and healed side at different follow-ups is reported in Table III. All analyses are validated with 3 consecutive measurements. No statistically significant differences were observed between the "repaired" and "healthy" group at 3 and 5 years of follow-up. The strength and endurance of flexion and



**Figure 1** Distal tendon biceps brachii. (a) Fibrocartilaginous tissue shows spherical cells surrounded by collagen fibers (type II collagen, original magnification  $\times$ 40). (b) Cluster of chondrocytes (Alcian blue original magnification  $\times$ 40). (c) Chondrocytes staining for S-100 protein in the damaged tendon (original magnification  $\times$ 20). (d) Type I collagen expression in the damaged tendon (original magnification  $\times$ 40).

Table II Comparison of range of motion (ROM) between repaired/healthy and dominant/nondominant side

|            | Repaired    | Healthy     | P value | Dominant    | Nondominant | P value |
|------------|-------------|-------------|---------|-------------|-------------|---------|
| ROM        |             |             |         |             |             |         |
| Flexion    | 129.3 (8.2) | 129.5 (4.2) | .56     | 130.2 (7.5) | 128.1 (5.1) | .47     |
| Extension  | 1.2 (0.9)   | 0.4 (0.3)   | .11     | 0.7 (0.5)   | 0.9 (0.4)   | .32     |
| Pronation  | 85.2 (4.5)  | 86.3 (5.1)  | .48     | 85.9 (6.2)  | 85.4 (4.8)  | .51     |
| Supination | 82.6 (5.8)  | 85.4 (6.2)  | .28     | 80.0 (7.4)  | 84.2 (6.5)  | .12     |

Data are presented as mean (standard deviation).

supination were significantly different between groups at 1 year of follow-up. Moreover, the strength and endurance of flexion and supination were statistically different between 1 and 3 years of follow-up in the "repaired" group.

The deficit in terms of peak torque and total work between the operated and nonoperated arm in relation to dominance and time is reported in Table IV. No significant differences were reported in flexion between the dominant and nondominant groups during follow-up. On the contrary, peak torque and total work of supination showed a significant recovery of the dominant side with respect to the nondominant one at 1, 3, and 5 years of follow-up.

#### Discussion

The present study reported the presence of fibrous cartilage metaplasia in all of the DBBT samples collected. It has been well established that fibrocartilage has mechanical properties intermediate between tendons, subject primarily to tension and the hyaline cartilage, which is designed to submit to high levels of compression. As reported for the rotator cuff and long head of the biceps brachii, we hypothesize that fibrous cartilage represents a histologic arrangement of the DBBT to address the compression loading of the tendon, making it at higher risk of rupture.<sup>3,7</sup> Indeed, the mechanical impingement on the biceps tendon during forearm

 Table III
 Comparison between repaired and healthy side

|                   | Follow-up (yr) | Repaired   | Healthy    | <i>P</i> value |
|-------------------|----------------|------------|------------|----------------|
| Flexion           |                |            |            |                |
| Peak torque (N/m) | 1              | 38.6 (2.7) | 49.4 (4.4) | <.01           |
|                   | 3              | 44.2 (3.9) | 47.2 (4.9) | .06            |
|                   | 5              | 45.8 (4.1) | 48.3 (5.3) | .10            |
|                   | 1-3            | P < .01    | P = .12    |                |
|                   | 3-5            | P = .23    | P = .27    |                |
| Total work (J)    | 1              | 1300 (56)  | 1605 (101) | <.01           |
|                   | 3              | 1545 (92)  | 1580 (96)  | .11            |
|                   | 5              | 1556 (102) | 1594 (98)  | .12            |
|                   | 1-3            | P < .01    | P = .29    |                |
|                   | 3-5            | P = .33    | P = .31    |                |
| Supination        |                |            |            |                |
| Peak torque (N/m) | 1              | 8.1 (0.8)  | 10.1 (0.9) | <.01           |
|                   | 3              | 9.5 (0.7)  | 9.6 (1.6)  | .35            |
|                   | 5              | 9.2 (0.5)  | 9.9 (1.3)  | .06            |
|                   | 1-3            | P < .01    | P = .29    |                |
|                   | 3-5            | P = .39    | P = .37    |                |
| Total work (J)    | 1              | 320 (51)   | 450 (65)   | <.01           |
|                   | 3              | 415 (56)   | 423 (72)   | .34            |
|                   | 5              | 400 (67)   | 436 (77)   | .06            |
|                   | 1-3            | P < .01    | P = .24    |                |
|                   | 3-5            | P = .36    | P = .37    |                |

Data are presented as mean (standard deviation).

| Table IV | Deficit ( | D%) ( | of peak | torgue and | total work | < of | repaired | side | compared | with | healthy | / side | in relatio | n to | dominance |  |
|----------|-----------|-------|---------|------------|------------|------|----------|------|----------|------|---------|--------|------------|------|-----------|--|
|          |           |       |         |            |            |      |          |      |          |      |         |        |            |      |           |  |

|                | Follow-up (yr) | Dominant   | Nondominant | P value |
|----------------|----------------|------------|-------------|---------|
| Flexion        |                |            |             |         |
| D% peak torque | 1              | -6.8 (8.2) | -9.1 (9.7)  | .28     |
|                | 3              | -5.1 (9.1) | -7.4 (8.5)  | .30     |
|                | 5              | -4.8 (7.9) | -6.7 (11.2) | .31     |
| D% total work  | 1              | -2.9 (6.4) | -7.3 (6.2)  | .12     |
|                | 3              | -2.1 (4.2) | -5.1 (9.1)  | .13     |
|                | 5              | -1.5 (5.4) | -4.0 (8.7)  | .17     |
| Supination     |                |            |             |         |
| D% peak torque | 1              | -4.9 (6.1) | -18.8 (5.9) | <.01    |
|                | 3              | -4.2 (5.3) | -16.3 (6.2) | <.01    |
|                | 5              | -3.8 (4.8) | -15.8 (5.3) | <.01    |
| D% total work  | 1              | -6.0 (5.3) | -19.2 (7.2) | <.01    |
|                | 3              | -5.4 (6.7) | -17.2 (6.3) | <.01    |
|                | 5              | -4.9 (5.9) | -16.8 (8.3) | <.01    |

Data are presented as mean (standard deviation).

rotation and its hypovascularity may induce the development of cartilage.<sup>11</sup> Subsequently, the cartilage metaplasia could inhibit the neoangiogenic processes necessary for tendon healing and remodeling, leading to a vicious circle. This mechanism could constitute a predisposing biological factor involved in the pathogenesis of DBBT tear.

The technique of reinsertion of the DBBT through a suture anchor by single access appeared to be safe, fairly

inexpensive, and reliable.<sup>19</sup> It showed few complications and good functional results at short- and medium-term follow-up. Regarding complications, the single anterior approach should avoid the risk of proximal radioulnar synostosis by conserving the intraosseous membrane, but it could entail a non-negligible risk of neurologic complications.<sup>6</sup> Indeed, no cases of radioulnar synostosis have been observed in our series. On the other hand, we reported 2 cases of transient paresthesia in the lateral antebrachial cutaneous nerve that spontaneously resolved in approximately 6 weeks.

In our series of patients, the range of motion in terms of the flexion and extension resulted complete in all patients, whereas 14% of patients lost supination from  $10^{\circ}$  to  $20^{\circ}$  in comparison with the contralateral side, as reported by other studies.<sup>1,10</sup> Gallinet et al<sup>6</sup> reviewed retrospectively 28 patients with DBBT ruptures repaired anatomically with the suture anchor technique after at least 6 months of followup; mobility testing of the injured limbs revealed a loss of supination in 29% of patients. A possible explanation of this deficit is that the DBBT functions as a supinator and secondarily as a flexor of the forearm along with the brachialis. Therefore, the rupture and reinsertion of the DBBT predominately affect supination compared with flexion.<sup>6</sup> However, in our prospective series of patients, the deficit of supination was acceptable and unnoticed for daily activities, as reported by MEPS results.

Regarding functional evaluation, isokinetic analysis was performed because it is more sensitive than manual muscle testing and provides a more comprehensive quantitative assessment of muscle performance than isotonic or isometric measures.<sup>12</sup> Balabaud et al<sup>2</sup> and Sarris et al<sup>17</sup> reported, respectively, 6% loss in flexion and none in supination, and 10% loss in flexion and 5% in supination by isokinetic analysis. The prospective series of patients in the present study showed an excellent recovery of strength and endurance in flexion and 5 years of follow-up between the surgical and contralateral side (Table III). The recovery of force and fatigue appears to be generally satisfactory for flexion and supination especially from 1 year to 3 years of follow-up.

It has been well established that isokinetic analysis is influenced by arm dominance.<sup>1</sup> Indeed, Askew et al<sup>1</sup> found that in healthy individuals, the dominant arm is 3% stronger in flexion and 8% stronger in supination compared with the nondominant one by isokinetic analysis. For these reasons, we reported the deficit in terms of peak torque and total work in flexion and supination between the operated and nonoperated arm in relation to dominance (Table IV). On the basis of our results, we found that time did not affect the recovery of flexion and supination in terms of strength and endurance. The present study shows that the deficit of flexion appears not to be significantly different in relation to limb dominance. On the other hand, looking at supination, the dominant side showed significantly better results in every test. It appears clear that arm dominance influenced supination strength and endurance, as reported by other studies.<sup>15</sup> It has been demonstrated that supination is more a fine motor skill compared with flexion. Therefore, the preferential use of the dominant arm for activities of daily livings could promote the coordination and an easier recovery of supination.<sup>14</sup> This mechanism could justify isokinetic results of the present study about significant differences in terms of deficit of supination between the

dominant and nondominant arm. We hypothesized that rehabilitation could benefit especially in case of the nondominant side to counteract this difference.

Many questions remain to be addressed. First of all, as supposed above, if the fibrous cartilage might be responsible for the rupture. Considering the normal macroscopic aspect of the torn tendon, we could postulate that cartilage metaplasia might only be a contributing cause of the rupture with the trauma. In addition, it would be interesting to know if aging could predispose to more metaplasia within the tendon and if different work-related activities reflect different content of cartilage.

# Conclusion

On the basis of our results, we find that the presence of cartilage metaplasia might make the DBBT at higher risk of rupture due to compression loading and the hypovascular zone of the tendon. However, the small cohort of patients and the lack of histologic analysis of the healthy DBBT mean that the role of cartilage metaplasia in tendon pathology remains to be defined. The technique of suture anchor reinsertion by single access was shown to be safe, with few complications and good functional results at 5 years of follow-up. No significant differences were reported between the injured and noninjured side in terms of flexion and supination isokinetic analysis, whereas arm dominance had a positive effect on supination.

### Disclaimer

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