Triceps Tendinopathy

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THE PATIENT

A 46-year-old, healthy, right-handed, active, cross-fit athlete had 2 months of increasing right posterior elbow pain with triceps exercises before presentation. On examination, he has pain with resisted elbow extension and tenderness at the triceps insertion with no defect. Radiographs of the elbow show a small olecranon spur but no osteoarthrosis. Magnetic resonance imaging (MRI) shows increased signal in the lateral and long heads of the triceps insertion.

THE QUESTION

What are the treatment options for triceps tendinopathy?

CURRENT OPINION

Triceps tendinosis is an enthesopathy akin to other enthesopathies of the upper extremity that are common during middle age (distal biceps, lateral epicondylitis, and rotator cuff). The signal changes on MRI are typically described as partial tears even when these are atraumatic changes resulting from enthesopathy.

Triceps enthesopathy occurs on a continuum from asymptomatic tendinosis to complete rupture. Macroscopic changes include tendon thickening and the pathophysiology is myxoid degeneration that may progress to collagen fibril rupture if there is an acute injury.[1] Degenerative changes generally precede a full-thickness tear (ie, healthy tendons do not usually tear).[2] Ruptures rarely have prior symptoms and symptomatic tendons typically do not rupture. Most tendinosis is unrelated to trauma. If there is an acute injury, there is usually a complete tear of at least 1 or 2 heads of the triceps insertion. Patients routinely describe pain at the triceps insertion and weakness with elbow extension.

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Most enthesopathies are self-limiting (eg, lateral epicondylitis, plantar fasciitis), but triceps enthesopathy is too uncommon to study well and the natural history is unknown. There is no known disease-modifying treatment for enthesopathy. Palliative treatments, reassurance, and time are the current best options. Complete ruptures are treated operatively but the role of surgery for ruptures of 1 or 2 of the heads is debated.

Strauch[3] made the point that the term "partial tear" can represent a wide spectrum of injury or tendinosis and is not well-defined. A 90% defect may have a different functional outcome compared with a defect of 10% when treated without surgery. He concluded that triceps defects should be reattached to the olecranon if the patient has decreased power of elbow extension and the defect is shown to be greater than 50% on MRI, but this depends on health and activity level.

Patient and surgeon frustration usually peak after a few months of symptoms; many patients are influenced by work absence, an insurance claim, and a sense that something needs to be done or they will never be able to depend on the arm (catastrophic thinking). These pressures may reinforce the familiar orthopedic cutoff of 6 months for nonsurgical treatment, but 6 months is probably too soon to give up on spontaneous resolution of symptoms from enthesopathies because most tend to be symptomatic for an average of 1 year and often much longer, and there are no effective disease-modifying treatments.

THE EVIDENCE

In one analysis of 1,014 tendon injuries before the advent of MRI, only 8 (0.8%) involved the triceps and 4 were lacerations.[4] Koplas et al[5] reviewed 801 consecutive MRI examinations of the elbow and upper extremity in 740 patients with elbow pain. Triceps pathology was identified in 79 studies (9.9%): complete tear, partial tear, tendinosis, or musculotendinous junction strain. Complete rupture was defined as a large fluid-filled gap between the olecranon and the tendon. The term "partial tear" was applied when there was discontinuity of some tendon fibers, but it is not clear how this is distinguished from tendinosis, which can also have defects or discontinuities. "Tendinosis" was used

to define a tendon that demonstrated intact fibers of increased signal intensity without focal disruption. Signal changes without defect were the most common finding and were present in 6% of triceps; they represent 60% of all triceps pathology identified on MRI in painful elbows. The prevalence of defects was 3.8% and 64% were incomplete. Only 28% of these changes occurred in athletes. The average age of those with defects was 47 years.

One report of bilateral ruptures of the lateral insertion of the triceps treated nonsurgically in a weight lifter showed good strength but some fatigue with lifting 55 weeks after injury.[6]

Mair et al[7] described 11 tears of the entire triceps tendon (90% to 100% of tendon involvement) and 10 tears of only 1 or 2 heads (30% to 75% of tendon involvement) in 19 professional football players over a 6-year period. Fifteen of them were lineman, which suggests an at-risk population. All had notable pain and weakness after an injury and were diagnosed based on examination and MRI. Seventeen described an eccentric contracture event. Sixteen had a palpable defect. Seven had reported pain before the injury, 5 of whom obtained steroid injections for presumed olecranon bursitis before the injury. Of the 10 incomplete tears, 8 were medial-sided as diagnosed by MRI. Six of the 10 had no residual pain or weakness at the end of the season with nonsurgical treatment; 3 showed no defect on follow-up MRI. Three were treated with delayed surgery after the end of the season. One player had complete rupture on return to play. All 10 patients with rupture of 1 or 2 heads of the triceps played at least 1 more year of professional football. The surgeons recommended return to play when symptoms resolved and the patients had near normal strength. Mair et al suggested nonsurgical treatment with defects up to 75% on MRI. Some patients with smaller defects requested surgery.

Bos et al[8] described 6 weeks of orthosis use in 30° flexion for an acute tear of the medial triceps in a 36-year-old hemodialysis patient after a fall. She had full motion and strength 6 months after injury.

Farrar and Lippert[9] described a successful outcome after conservative management of an incomplete rupture diagnosed on examination. The determining factor in their treatment algorithm was the ability to extend the elbow against gravity. The patient was treated in a orthosis with the elbow held in 30° flexion for 4 weeks and was reported to have full range of motion and strength 9 months after injury.

One case report[10] describes a 47-year-old man with pain and weakness at the triceps insertion after a lifting injury. He had an MRI with a diagnosis of an incomplete tear (1 or 2 heads) or triceps tendinopathy with defect (unclear in the report). Five weeks after onset of symptoms, he was offered a platelet-rich plasma injection for unclear reasons. The patient returned to usual activities 4 months later and the authors seemed to credit the injection.

Athwal and colleagues[11] described an arthroscopic technique for triceps repair. They described 2 patients with isolated, deep, medial head tears. These were repaired with 2 double-loaded suture anchors, with good outcomes. The authors also looked at the insertional anatomy of the lateral, long, and medial heads of the triceps tendon. In 15 cadavers, 8 had an isolated medial head attachment whereas 7 had all 3 blended together for a surface area of 134 mm^2. In another study of 27 cadavers,[12] the footprint was measured as an average of 466 mm^2.

Heikenfeld and colleagues[13] described triceps tears or defects involving the long and lateral heads in 14 patients, including 2 weightlifters who denied anabolic steroid use. Twelve patients denied substantial trauma. They noted a strong correlation with chronic olecranon bursitis, which was seen in 10 patients. These were repaired with suture anchors and a bursectomy. They had noteworthy clinical and functional improvements at 1 year.

van Riet et al[14] described the results of repair of 23 ruptures in 22 patients, average age 47 years. Magnetic resonance imaging was performed in only 5 patients. Operative findings revealed complete tendon rupture in 8 elbows (7 patients). Fifteen patients had tears involving only 1 or 2 of the heads of the triceps. Fourteen triceps ruptures in 13 patients were repaired directly using transosseous tunnels, and 9 using a tendon graft (6 of 9 with incomplete tears). There were good results, especially when done within 3 weeks after the injury. However, 3 patients had a total of 4 re-ruptures after primary repair. Three had a second direct repair and 1 was reconstructed using a tendon graft after a delayed presentation, all with no subsequent sequelae. The authors did not describe patients treated nonsurgically.

SHORTCOMINGS OF THE EVIDENCE

The terms “tendinosis,” “tendinitis,” “partial tear,” and “tear” are used variably and without definition. It is often unclear whether triceps lesions are acute and the result of trauma or longstanding and the result of enthesopathy. Signal changes on MRI are often referred to as “tears,” which further confuses these issues.

Triceps tendinopathy is uncommon and there are limited data to guide treatment. For instance, there are

few or no data regarding the natural history of triceps tendinopathy. There are no known disease-modifying treatments. Studies are limited to small case reports, advanced imaging studies, and anatomical studies. Surgical treatment seems to be offered arbitrarily.

DIRECTIONS FOR FUTURE RESEARCH

To determine the natural history of triceps tendinopathy, the current authors could obtain MRIs of a large group of adults (100,000 or more) of all ages with healthy elbows and then evaluate their symptoms, triceps function, and MRI for 30 years. Such a study would determine the prevalence of asymptomatic MRI findings, the rate at which those findings become symptomatic, and the factors associated with tendinopathy, defects, and acute tears of one or more heads of the triceps.

To identify disease-modifying treatments and compare the effectiveness of various palliative, potentially disease-modifying, and salvage strategies (eg, injections, ultrasound-guided tenotomy, surgery), randomized controlled trials would be needed, including adequate sham injection, tenotomy, or sham surgery controls and involving multiple centers. It is possible that the acceptable outcomes from surgery result, at least in part, from the natural history of this enthesopathy, regression to the mean, or a placebo effect.

OUR CURRENT CONCEPTS FOR THIS PATIENT

In our opinion, the ideal study of the natural history of triceps tendinosis would likely show a high percentage (90%) of improvement without intervention over about a year. We also think that there are currently no disease-modifying treatments.

For the specific patient under consideration, we would offer reassurance and nonspecific palliative treatment. He can continue in his active lifestyle.

For an acute tear of 1 or 2 of the 3 heads of the triceps, we offer nonsurgical treatment with return to full activities when comfort and strength allow. The unusual patient who continues to return to the office after 12 months or more of symptoms and expresses frustration is the one we feel least prepared to help. More from desperation than science, we sometimes make a shared decision to try unproved treatments that are intended to be disease-modifying (eg, dry needling, platelet-rich plasma, ultrasound tenotomies).

The role of operative intervention and its timing are both debatable. We find the concept of tendon debridement and repair in the setting of tendinosis with defect to be appealing, although we acknowledge that the size of the defect that benefits and improvements over the natural history of untreated disease both remain unresolved. If there is no improvement after 18 months of symptoms, we offer surgical intervention, open or arthroscopic. If there is a defect and weakness and the patient is an athlete or laborer, we favor a debridement with a knotless anchor anatomic repair, as described by Paci et al.[15]

For an acute tear of the entire triceps in a nonsedentary individual, we offer a primary repair using the same knotless anatomic repair. If the patient comes to our office more than 3 weeks after injury, we would be prepared to do a triceps tendon reconstruction using semi-tendinosis or Achilles allograft.

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