

Tendon injections

Does it matter what you use?

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Tendinopathy is one of the most fascinating and controversial areas of sports medicine and certainly provides a steady inflow of patients. Just as dermatology is meant to be a great area of medicine because the patients “never die and never get cured”, it seems that most patients with tendinopathy have long-term symptoms that don’t finish them off but rarely settle completely. As a general rule, surgeons in Australia have very little interest in tendinopathy, which may actually be a good thing. From the surgeons’ point of view, when the supply and demand curve for services is so far in your favour, why would you develop an interest in tendinopathy, when arthroscopy is quicker, pays more, and leads to higher patient satisfaction? From the patients’ point of view, why would you undergo an operation that has a 70% success rate, when all of the non-operative management also has a 70% success rate?

Tendon injections have always been very popular, but in the past this statement has been in reference to cortisone injections. The old management paradigm for tendinitis (which is what it was called in the old days) was simple - physiotherapy, followed by NSAIDs, followed by cortisone injections, followed by surgery. At every stage, if you waited long enough, there seemed to be the ubiquitous 70% success rate, which only varied for a surgical case series, where it could reach higher rates of success if the follow-up rate for failures was bad enough.

The old tendinitis paradigm was smashed by the announcement that inflammatory changes to tendons are the exception rather than the rule. Under the new tendinopathy paradigm, it is obvious why anti-inflammatory

cortisone injections didn’t work in 30% of cases, because inflammation was never the problem. The new mystery is why they ever worked in 70% of cases, in which inflammation was never the problem.

I participated in a tendon injection seminar in Melbourne a couple of months ago, in which the following types of injections were reviewed: cortisone, calcium gluconate, auto-injection of patients own blood, heparin, dextrose, aprotinin, sclerosants, dry needling and saline with local anaesthetic. There may have been another one or two suggestions that I have forgotten. Every doctor using these injections had a similar tale to tell – there was a very limited evidence base, but the clinical results were quite impressive (hardly anyone got worse with treatment, and a good proportion of patients got better – somewhere near 70%). Upon hearing these results, one is tempted to form the opinion that all of these injection substances are working through the same method, acting simply as an irritant which leads to a healing response. This form of treatment is sometimes referred to as *prolotherapy* (meaning a treatment that causes tissue proliferation) by the optimistic proponents of a new tendon injection technique. The cynics might call it a placebo response, in which an injected placebo is better than a non-injected one.

There may be more than a grain of truth in both the prolotherapy and placebo arguments, but for those doctors using tendon injections and those practitioners referring for tendon injections, there needs to be further argument about which injections are best to use. The first question is when to use cortisone injections? The biggest advantage of cortisone injections over

the other techniques is that they are the established bronze standard for tendon injections (if they were the gold standard we probably wouldn’t be looking at so many alternatives). For every aprotinin, autologous blood or calcium gluconate ever given, there have probably been 1000 cortisone injections given over the course of medical history, so cortisone is, at worst, the devil that we know. My approach to cortisone is to use it when there is evidence of secondary impingement, and to avoid it when there is concern about tendon rupture. Secondary impingement, which occurs particularly in areas such as the subacromial space, is a genuine theoretical indication for use of cortisone, as cortisone is known to shrink excessive scar tissue which can cause impingement (see Table). However, the risk of tendon rupture is a concern for prime movers without agonists, as we know that cortisone is a catabolic rather than anabolic substance.

Some tendinopathies with impingement (cortisone indicated)

Rotator cuff

Posterior ankle tendons

Gluteal insertions at greater trochanter

Carpal tunnel syndrome

Some tendinopathies where rupture is of concern (cortisone contraindicated)

Achilles

Patellar

Tibialis posterior insertion

When it comes to choosing other substances to use for tendon injections, it is important to pick an injection type and technique that is going to fit in well with adjunct management. The most important adjunct management is usually a combination of *eccentric exercises* and moderate tendon loading.

Although we don't fully understand the reasons why, a combination of controlled scientific studies and clinical experience tells us that eccentric (strengthening exercises with load only when the tendon is lengthening) exercises are more beneficial than concentric, isometric or an eccentric-concentric combination. Moderate tendon loading is important because of similar evidence which tells us that complete rest is bad for tendons. Overload or overstrain is generally the original cause of a tendon injury, but paradoxically, complete rest is generally counterproductive, as it is believed to lead to tendon atrophy. The preferred paradigm is moving towards a more active approach. It used to be thought that the correct amount of load for a damaged tendon was that load that caused no pain, but the work of Alfredson in particular has led to a slightly more aggressive approach. Load which causes mild pain at the time, but which is not leading to a deterioration in the overall condition, is now believed to be beneficial.

The definition of moderate load is the maximum load which is not causing a deterioration in tendon function.

There is a degree of finesse to this advice. The advice of how much loading is best will be different for every patient, and also different for each patient at any given time of their rehab. A good analogy is the game of Blackjack (Twenty-one). If you get too high a score (too much loading) you bust and lose, but if you are below the threshold (bust limit of '21') then the higher the better.

The combination of moderate tendon loading and eccentric exercises – now widely accepted as the mainstay of conservative treatment – is in conflict with the instructions traditionally given to patients after a cortisone injection. Usually the instruction post-cortisone is to rest the injected tendon for a time period ranging from 72 hours to 4 weeks. The basis of this is to avoid the risk of further tendon damage,

given the potential tendon-weakening and pain-masking effects of cortisone. Although it is logical to advise against further tendon damage, it is illogical to interrupt the other management that is considered to be critical to developing a tendon healing response (eccentric exercise and moderate loading).

This argument is critical when assessing the usefulness of the 'other' types of tendon injections. Generally, the advocates of the more inert substances used for tendon injections, such as dextrose, calcium gluconate, autologous blood and most definitely dry needling, advocate a degree of tendon trauma as part of the injection technique. This is because it is believed that these substances, if injected only around the tendon, would be quickly washed away from the scene and have minimal effect. For this reason, some advocate injection under ultrasound vision, where the injection can be placed as close to the undersurface of the tendon as possible, and the tendon deliberately irritated, penetrated or traumatised. The instructions post-injection are usually the same as for cortisone injections – because of the fear of further tendon damage post-injection trauma, a rest period is advised, which often contradicts the other advice the practitioner is giving regarding tendon loading.

My preferred non-cortisone tendon injection substance is aprotinin, which is a high molecular weight substance that is both a protease-inhibitor (and hence perhaps a collagen promoter) and pro-coagulant. The major side effect for this injection is potential allergy – most patients get an itch and slightly less than 1 in 100 get a more severe allergic reaction, although this is usually non-systemic for a small peritendinous injection. Even if this substance is not a true collagen promoter and therefore the injection effect is due to prolotherapy (irritation) alone, this can be achieved with a peritendinous injection rather than through tendon traumatising. Most importantly, two randomised control trials showing effective results for aprotinin have been published for the

patella and Achilles tendons, which are those tendons where cortisone is most contraindicated. In these trials, the patients were allowed to exercise as much as they wished the day after the tendon injections, which is what I permit in a clinical setting. I am confident in this instruction because, on the basis of the trials and clinical results, I am quite sure that aprotinin does not cause tendon damage or atrophy, and I am equally sure that my injection technique does not cause tendon trauma. My instructions are more specific to the patient that the injections should not interfere with the mainstay of treatment, which is eccentric exercises and moderate tendon loading. In other words, the patients are not only permitted to moderately load the tendon the day after the injection, they are actually encouraged to.

Obviously it would be preferable to know more about the exact action of aprotinin and to have more results of randomised control trials comparing aprotinin injections to other substances. However, this criticism is valid for every injectable substance on the earlier list I cited. Although I have been facetiously quoting the 70% success rate for every type of treatment, eventually we will be able to prove which substances have the 75% success rates for injection and which ones have the 65% success rates. It is interesting that some recent animal trials regarding heparin, which being an anti-coagulant is an aprotinin antagonist, show that heparin injections cause tendon weakening. It doesn't necessarily follow that aprotinin will therefore cause tendon strengthening, but it does suggest that heparin should be one of the injection substances crossed off the list of preferred options. In general, I try very hard to refrain from claiming superiority of my preferred injection substance and technique to that of other doctors, when there is no hard evidence to support such claims. It is important though for everyone treating tendon disorders to have a holistic approach. It is not a matter of giving a patient a two minute consultation

which consists of an injection consent and an injection. Although we have much to learn, there is much that is known about tendinopathy, and patients should be educated as part of a successful management program.

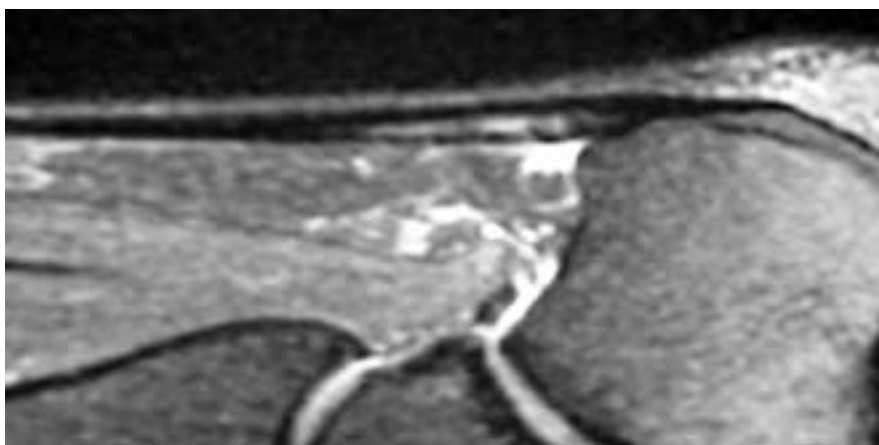
For more information on tendon disorders (including references), visit www.injuryupdate.com.au and to register interest in a RCT for aprotinin in Achilles tendinopathy, visit www.johnorchard.com. Please appreciate that these websites contain a great deal of information representing most of what I can pass on to fellow practitioners about these injections. I do not generally have time to pass on this information to multiple people over the phone. For the viewpoint of Nic Maffulli, who first advised me about Aprotinin and who conducted some of the RCTs, visit: www.physioroom.com/experts/expertupdate/interview_nmaffulli_20030701.php

Finally, please note that I have not covered other non-injectable pharmacological treatments such as Nitrate patches, which recent research suggest may have a role.

Figure 1.
Aprotinin as an injectable is available in Australia under the brand name Trasylol.



Figure 2.
MRI scan of Achilles tendinopathy (white degenerative region in black tendon).



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