Treatment of Tendinopathies with Platelet-rich Plasma

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KEYWORDS

• Tendinopathy • Platelet-rich plasma (PRP) • Biologics • Tendons

KEY POINTS

- Pain and dysfunction related to tendinopathy are often refractory to traditionally available treatments and offer a unique challenge to physicians as there is no current gold standard treatment.
- Injectable biologics including platelet-rich plasma (PRP) may represent a new modality in conjunction with a multifaceted treatment approach.
- PRP injections are not associated with the systemic or tendon degradation risks of corticosteroids or the inherent risks of surgery.
- Basic science studies are promising but have not been replicated with high-powered evidence at the clinical level.
- Given this promise and the lack of a definitive treatment, further evidence to expand understanding of the role of PRP in the treatment of tendinopathy is needed.

CAUSES OF TENDINOPATHY

Tendons serve as the interface between muscles and the skeletal structures on which energy is transferred, ultimately leading to motion. They typically function at 30% to 40% of their maximum tensile strength and are injured when exposed to supramaximal loading.[1] A low metabolic rate allows tendons to function under prolonged stress but can also lead to delayed healing when injury occurs.[2,3] Common terms used in the past to describe tendon disorders include tendinitis, tendinopathy, and tendinosis. Tendinitis, which implies an inflammatory cause, has largely been abandoned as a term because of multiple studies showing a degenerative rather than inflammatory milieu in affected tendons.[4–6] Tendinopathy is an often-used term that implies a painful subacute functional loss that may or may not involve an inflammatory component. Tendinosis refers to the degenerative structural changes that occur in tendons that

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fail to heal after injury. Once thought to represent a persistent inflammatory process, tendinosis is now understood to be characterized by a scarcity of clinical or histologic signs of inflammation and instead shows fibrotic replacement of collagen, poor organization of remaining collagen fibers, increased neovascularity, and a lack of inflammatory mediators.[7–12]

Although the end result is more clear, the pathophysiology of tendinosis is still being debated, because multiple factors likely contribute to the hallmark pain and dysfunction of incomplete tendon healing.[11,12] Structures that are commonly involved include the rotator cuff tendons of the shoulder, the wrist flexor and extensor tendons, and the patellar and Achilles tendons of the knee and ankle, respectively. Like many other types of musculoskeletal disorder, both intrinsic and extrinsic risk factors are associated with the tendinopathy. Common intrinsic factors identified include altered biomechanics, decreased strength, both hypoflexibility and hyperflexibility, increasing age, female gender, certain medication use, and diabetes.[5,13–16] Extrinsic factors including load amount, duration, frequency, and direction of movement also contribute to the risk of developing tendinopathy.[17,18] In addition to pain and dysfunction, studies have shown a profound increase in risk for tendon rupture in degenerated tendons, highlighting the importance of developing effective treatment strategies not only for pain relief but also for tendon healing.[19]

HISTORY OF TENDINOPATHY MANAGEMENT

The classic tendinopathy treatment algorithm focused initially on palliation via arrest of a presumed inflammatory process. Nonsteroidal antiinflammatory drugs (NSAIDs), relative immobilization, topical modalities, compression, and corticosteroid injections are all common interventions used to quell pain and allow participation in a rehabilitation program focusing on stretching and eccentric strengthening. Although pain relief is an important goal in treating the patient with tendinopathy, modalities that are intended to decrease pain in the short term do not address the underlying tissue disorder. The inflammatory cascade that was previously targeted as a means of treating tendinopathy is now understood to be integral in the wound healing process, and blunting its effects may lead to delayed healing and slower functional recovery.[20–23]

It has been theorized that resetting the healing process via the introduction of concentrated growth factors, proteins, and many other bioactive substances, all of which are plentiful in platelet-rich plasma (PRP), may be an effective means of treating recalcitrant tendinopathies. PRP preparations have been used with proven efficacy for years to augment tissue healing in surgical wound closure as well as fat, skin, and bone grafting, and more recently have been used with success in treating tendinopathy as well.[24–28]

What is PRP?

PRP has been defined as an autologous concentration of platelets obtained after gentle centrifugation of whole blood.[29,30] The resultant supernatant contains a high concentration of platelets and the 7 fundamental protein growth factors secreted by the alpha granules of platelets to promote wound healing, including platelet-derived growth factors (PDGF) alpha, beta, and alpha/beta, transforming growth factors (TGF) beta 1 and beta 2, vascular endothelial growth factor (VEGF), and epithelial growth factor (EGF).[31,32] A summary of the functions of these and other bioactive components of PRP is presented in **Table 1**.

The plasma component of the centrifuged supernatant also contains 3 important proteins for tissue regeneration: fibrin, fibronectin, and vitronectin. Fibrin polymerizes