Int. J. Morphol.,
44(1):99-107, 2026.

Tendinitis of the Temporalis Muscle Tendon: Morphological and Pathophysiological Implications in the Management with Platelet Concentrates – PRP

Tendinitis del Tendón del Músculo Temporal: Implicancias Morfológicas y Fisiopatológicas en el Manejo con Concentrados Plaquetarios - PRP

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HERNÁNDEZ, A.; ITURRIAGA, V.; VELASQUEZ, N.; MUÑOZ, J.; LANDAETA, H. & BORNHARDT, T. Tendinitis of the temporalis muscle tendon: Morphological and pathophysiological implications in the management with platelet concentrates - PRP. *Int. J. Morphol., 44(1)*:99-107, 2026.

SUMMARY: Tendinopathies are a common cause of musculoskeletal pain and functional limitation. Platelet concentrates, such as platelet-rich plasma (PRP), have emerged as therapeutic tools for tendon tissue regeneration. However, their specific application in the temporal muscle tendon (TMT) has received little attention. The aim of this article is to review the literature on the use of platelet concentrates in tendinopathies and to contextualize their potential applicability to the TMT based on its morphology, physiology, pathophysiology, and the mechanisms of action of platelet concentrates. Two literature searches were conducted: the first, in PubMed, Scopus, and EMBASE, focused on PRP use in tendinopathies in general; the second, specific to the TMT, was carried out using both open and manual searches to identify as many relevant publications as possible in the same databases and academic books, without year restrictions. A total of 266 articles were identified, of which 13 met the inclusion criteria for PRP and tendinopathies, and 15 addressed the TMT, mainly in relation to its anatomy, morphological variability, and clinical correlations. The available evidence suggests that PRP may be beneficial for TMT injuries, owing to its capacity to promote tissue repair and reduce pain. However, no clinical studies have directly assessed its efficacy in TMT tendinopathies, limiting the ability to establish definitive recommendations. The anatomical features of the TMT, including its depth, bone relationships, and variability of insertion, should be considered when applying therapies such as PRP. Well-designed clinical studies integrating these variables are needed to define effective and safe protocols for the management of TMT tendinopathies.

KEY WORDS: Temporal muscle; Tendinopathy; Facial pain; Platelet-rich plasma; Platelet-rich fibrin.

INTRODUCTION

Tendinopathies are a common cause of musculoskeletal pain and dysfunction (Sebbagh *et al*., 2023). These conditions, which mainly include tendinitis and tendinosis, most frequently affect tendons subjected to repetitive stress, such as the calcaneal tendon, patellar tendon, supraspinatus tendon, and the common extensor tendon at the lateral epicondyle (Unlu *et al*., 2017; Sebbagh *et al*., 2023). The progression of these injuries often involves persistent functional limitations, pain, and, in many cases, an insufficient response to conventional treatments (Devadas, 2024).

The temporalis muscle is a broad, flat, fan-shaped muscle that originates from the inferior temporal line of the skull and the deep layer of the temporal fascia. Its fibers converge to form the largest tendon in the craniofacial region. The temporalis muscle tendon (TMT) is broad and fibrous at its muscular origin, but as it descends, it becomes thicker and more cylindrical, narrowing to pass deep to the zygomatic arch and superficial to the cranial wall (Fig. 1). At its insertion, it expands again to attach to the medial, lateral, and superior aspects of the mandibular coronoid process (Ernest 3rd *et al*., 1991; Iturriaga *et al*., 2016;

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FUNDING: This research was supported by the Temporomandibular Disorders and Orofacial Pain Program, the Sleep & Pain Research Group and the Project DI20-0019 from the Universidad de La Frontera. The funders did not participate in the development, design and execution of the project that was carried out independently.

Received: 2025-08-20 Accepted: 2025-11-20

Bressler *et al*., 2017; Okeson, 2019; Kim *et al*., 2024). This anatomical arrangement enables the temporalis muscle and TMT to elevate and retract the mandible (Okeson, 2019).

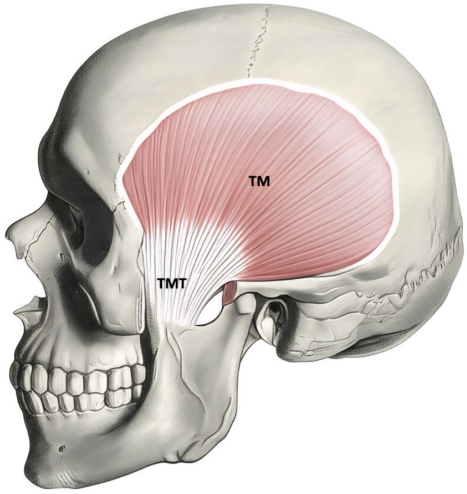

Fig. 1. Lateral view of the skull showing the temporal muscle (TM) and the temporal muscle tendon (TMT), which descends through the infratemporal fossa (zygomatic process sectioned) and inserts into the coronoid process of the mandible.

Like other tendons, the TMT can be affected by tendinopathies, which are classified as temporomandibular disorders (TMD). This is reflected in section 2.2.1, "Myofascial orofacial pain attributed to tendonitis," of the International Classification of Orofacial Pain, 1st edition (ICOP) (2020); in category 2.B, "Tendonitis – Masticatory Muscle Disorders," of Expanding the Taxonomy of the Diagnostic Criteria for Temporomandibular Disorders (Peck *et al*., 2014); and in classification 4.a, "Temporal tendinitis – Inflammatory disorders of associated structures," within the diagnostic framework for TMD described in Okeson's textbook (Okeson, 2019). These tendinopathies represent a specific subtype of musculoskeletal pathology characterized by inflammatory and/or degenerative changes in the TMT, which result in localized pain and functional limitation, requiring a specialized diagnostic and therapeutic approach within the context of TMD.

On the other hand, platelet concentrates, such as platelet-rich plasma (PRP), are autologous platelet concentrates suspended in a small amount of plasma that release various growth factors and cytokines that promote tissue repair. It has been suggested that these growth factors stimulate cell proliferation, collagen synthesis, and angiogenesis (Devadas, 2024). This therapeutic option has established itself as a tool for modulating inflammation, stimulating tissue regeneration, and, primarily, restoring the functional architecture of tendons, leading to pain reduction (Kaux & Emonds-Alt, 2018; Tischer *et al*., 2020; Devadas, 2024). Its use has increased considerably in recent decades, and there is clinical literature supporting its application in tendinopathies. However, to date, its use in TMT tendinopathies has not been reported.

Furthermore, it is noted that the evidence is mixed due to variations in PRP preparation techniques, injection protocols, lesion location, and patient characteristics. Therefore, there is a critical need for studies evaluating the long-term clinical outcomes of PRP in patients with chronic tendinopathies, specifically in the TMT. Thus, the aim of this article is to review the literature on the use of platelet concentrates in tendinopathies and to contextualize their potential applicability to the TMT based on its morphology, physiology, pathophysiology, and the mechanisms of action of platelet concentrates.

MATERIAL AND METHOD

Two systematic literature searches were conducted. The first addressed the evidence on the use of platelet concentrates in tendinopathies in general, and the second focused specifically on TMT tendinopathies. Both strategies are detailed below.

I. PRP and its application in tendinopathies: A search was conducted in the PubMed, Scopus, and EMBASE databases in April 2025. The following terms were used: "Platelet-rich plasma", "PRP", "tendinopathy", "tendinitis", "tendinosis", and "tendon", combined using the Boolean operators AND and OR. The search was limited to studies in humans, published in English or Spanish in the last 10 years. Different levels of scientific evidence were included: systematic reviews, meta-analyses, controlled clinical trials, observational studies, comparative studies, and case reports. The search strategy was adapted to each database.

II. PRP and its application in TMT tendinopathies: An open, manual search was conducted to identify relevant articles in the same databases mentioned above, academic books, and scientific articles of interest. Studies in English and Spanish, conducted in humans, with no restriction on year of publication or design, were included. The strategy was adapted to each type of source.

In both cases, articles were selected based on a review of the title, abstract, and full text. References were managed with Zotero® software to eliminate duplicates and organize the documents. The evaluation and selection of articles were performed by the author AH.

RESULTS AND DISCUSSION

A total of 266 articles related to PRP and tendinopathies were identified, of which 13 met the selection criteria. Fifteen relevant articles were selected concerning TMT tendinopathies. Next, based on the selected and analyzed articles, the main topics of relevance describing the morphological and pathophysiological implications of platelet concentrates in tendinopathies were developed, contextualizing their specific applicability in TMT tendinopathies.

I. Prevalence of tendinopathies

Tendinopathies most commonly affect tendons subjected to functional overload or mechanical trauma (Sebbagh *et al*., 2023). It is reported that between 40 % and 50 % of athletes suffer tendon injuries, approximately 25 % of work-related injuries to the hand and wrist are tendinous, and in adults, 15 % of individuals between the ages of 50 and 59 have tendinous injuries. Similarly, these pathologies have a prevalence of 51 % in adults over the age of 80 (Wang & Nirmala, 2016).

Concerning tendon injuries of the TMT, tendinitis is a frequently underdiagnosed entity within the spectrum of TMDs, despite its significant clinical impact. In a retrospective study conducted by Dupont Jr. & Brown (2012), the clinical records of 449 patients diagnosed with TMD were evaluated, and it was observed that 353 of them (78.62 %) had concomitant TMT tendinitis. Furthermore, the condition was bilateral in 83 % of cases with a positive diagnosis, unilateral on the left in 10.48 % and unilateral on the right in 14.45 %. This same study revealed that 77.95 % of affected patients were women, which is consistent with the literature reporting a higher prevalence of orofacial pain and TMD in females (Dupont Jr. & Brown, 2012). On the other hand, other authors mention that TMT tendinosis is also often underdiagnosed and confused with other causes of orofacial pain, which may contribute to an underestimated clinical prevalence, despite its symptomatic relevance (Bressler *et al*., 2020).

II. Cell physiology of the temporal muscle tendon

TMT is composed of a variety of specialized fibroblast populations, called tenocytes, together with stem cells and tendon progenitor cells, arranged in a collagen matrix, surrounded by the epitendon. Its composition is characterized by a high concentration of water (55 % of its dry weight), the presence of proteoglycans in minimal amounts (<1 %), cells and type I collagen (85 %), as well as small amounts of other collagens (types III, V, XII, and XIV), elastin, and fibronectin. Type I collagen is characterized by an organization into parallel fascicles, which confers high resistance to tensile forces (Ernest 3rd *et al*., 1991; Fuentes & Ottone, 2021).

The osteotendinous junction of TMT in the coronoid process is subject to significant mechanical stress. In this anatomical region, a highly specialized transition zone is observed, characterized by a gradient in tissue composition, including tendon, fibrocartilage, and bone. This structure facilitates the transmission of forces and contributes to minimizing the risk of injury (Ernest 3rd *et al*., 1991). In this insertion, microscopic studies have demonstrated signs of tendon degeneration in symptomatic patients, including foci of necrosis, fibrillar disorganization, and hyaline changes (Okeson, 2019).

A relevant physiological characteristic of the TMT is its limited vascularization, which hinders its ability to regenerate from injuries or overload, making it susceptible to chronic alterations such as tendinosis in individuals exposed to trauma or repetitive microtrauma (Ernest 3rd *et al*., 1991; Okeson, 2019). The most significant blood supply is obtained from the vascular perimysium, which penetrates the paratendon, epitendon, and endotendon, forming an intratendon vascular network.

III. Pathophysiology of tendinopathies

Tendinopathies are divided into tendinitis and tendinosis (Dupont Jr. & Brown, 2012; Iturriaga *et al*., 2016; Okeson, 2019; Bressler *et al*., 2020). Tendinitis refers to an acute inflammatory process in which inflammatory cell infiltrates such as neutrophils and macrophages, interstitial edema, and increased vascularization are observed. It usually occurs in the early stages of injury, associated with a recent traumatic mechanical stimulus. On the other hand, tendinosis corresponds to a chronic and degenerative phase of the tendon, characterized by disorganization of collagen fibers, proliferation of fibroblasts, increased extracellular matrix, abnormal neovascularization, and absence of a classic inflammatory infiltrate. At this stage, structural alterations without apparent inflammation predominate, which hinders spontaneous recovery of the tendon (Zhou & Wang, 2016; Sebbagh *et al*., 2023; Devadas, 2024).

Histopathological studies have shown that tendinous tissues with tendinosis have a high proportion of type III collagen, which has lower mechanical strength, instead of the type I collagen characteristic of healthy tendons (Liu *et al*., 2022). This results in a less organized matrix that is prone to micro-tears, perpetuating the injury and pain. In the case of TMT, the acute process can be caused by mandibular

movements that exceed physiological limits. In contrast, the chronic process is associated with mechanical stress produced by the progressive degeneration of collagen fibers perforating from the periosteum to the bone.

In general, when faced with tendinopathy, the tendon has three phases of repair: 1) Inflammatory phase, 2) Proliferative phase, and 3) Remodeling phase. The inflammatory phase occurs approximately 48 hours after the injury and lasts up to 7 days, with a hematoma forming at the injured site. Neutrophils, macrophages, and other inflammatory cells migrate to the site of the injury to remove damaged tissue. At the same time, platelets degranulate and secrete a considerable amount of growth factors. Cytokines and other inflammatory mediators are released, amplifying the inflammatory response and preparing the tissue for repair. These include interleukin 6 (IL-6), tumor necrosis factor alpha (TNF-α), platelet-derived growth factor (PDGF), and insulin-like growth factor 1 (IGF-1). These activate fibroblast cells, which positively regulate the production of extracellular matrix elements. Then, in the proliferative phase, extrinsic cells from the peritendinous soft tissue, such as the tendon sheath, fascia, periosteum, and subcutaneous tissue, and intrinsic cells from the epitendon and endotendon migrate and proliferate around the tendon injury. New vascular tissue forms to supply oxygen and nutrients to the cells involved in repair. Fibroblasts proliferate and synthesize collagen and other components of the extracellular matrix. These cellular activities are the basis for forming granulation tissue, which is a young and fragile tissue that fills the defect in the tendon. Starting on the fifth day of tendon healing, this immature tissue mainly synthesizes type III collagen. Finally, the remodeling phase begins 6 to 8 weeks after injury and lasts about 1 to 2 years, depending on the age and condition of the patient. The components of the extracellular matrix are assembled into random tissue deposits that are reorganized longitudinally. In this phase, the onset of physiological loading promotes the recovery of the tendon's biomechanical strength. Under the action of an appropriate load, collagen fibers reorganize and align parallel to the lines of tension, restoring tendon function. In addition, type III collagen produced during the proliferative phase is replaced by type I collagen, which is mechanically stronger, and cellularity and vascularization decrease. Throughout this phase, prolonged exposure of tenocytes to inflammatory signals could lead to overproduction of matrix components and excessive scarring/fibrosis, resulting in tendon adhesions (Molloy *et al*., 2003).

IV. Tendinopathies of the temporal muscle tendon

The main etiological factors of TMT tendinopathies can be grouped into intrinsic and extrinsic factors. Intrinsic factors include age, sex, anatomical variants, systemic diseases, and polymorphisms in collagen synthesis. Extrinsic factors include deleterious oral habits, such as nail biting or cheilophagia; macrotrauma or microtrauma, such as muscle strains and sustained muscle contraction; increases in vertical dimension due to interocclusal devices, restorations, or dental prostheses; and prolonged mouth opening (Iturriaga *et al*., 2016).

Clinically, TMT tendinitis manifests as pain on intraoral and extraoral palpation, localized or with referral patterns to other anatomical areas such as the upper and lower molars, temporal region, retroocular region, or preauricular area, which can lead to misdiagnosis and even unnecessary dental treatment (Dupont Jr. & Brown, 2012; Iturriaga *et al*., 2016; Okeson, 2019; Liu *et al*., 2022; Renner *et al*., 2024). This pain is usually sharp and/or dull, which can be exacerbated by jaw movement, potentially limiting mouth opening, either due to pain or functional restriction (Okeson, 2019; Bressler *et al*., 2020; Duffin *et al*., 2020; Stimmer *et al*., 2022).

Tendinosis, on the other hand, is a chronic degenerative condition of the tendon, characterized by disorganization of collagen fibers, hypocellularity, neovascularization, and foci of necrosis, with no evidence of active inflammation. Histopathological studies have confirmed these findings in surgical samples from the TMT, supporting its non-inflammatory nature (Ernest 3rd *et al*., 1991). Clinically, patients with tendinosis usually have persistent low-intensity pain, aggravated by masticatory function or sustained local pressure, and its diagnosis may go unnoticed for a long time (Bressler *et al*., 2020).

Regarding the influence of TMT tendinopathies on orofacial pain, a significant contribution to the understanding of the pathophysiology has been the experimental study by Yang *et al*. (2022), who demonstrated that injection of nerve growth factor (NGF) into the TMT increases mechanical sensitivity, both locally and in neighboring muscles such as the masseter. In addition, they observed an increase in the frequency of headaches during the following week, suggesting an active role of the TMT tendinopathy in the genesis of orofacial pain and secondary headache. These findings support the theory of peripheral sensitization with central repercussions, in which the TMT may act as a primary generator or perpetuator of pain in the craniofacial region (Yang *et al*., 2022).

V. Mechanism of action of Platelet Concentrates in tendons

Platelet concentrates are a biological therapy consisting of the local administration of autologous concentrate rich in platelets and/or leukocytes. There are

multiple types and classifications based on their cellular composition, degree of activation, and platelet concentration. Among the best known are the Mishra classification, the PAW classification (Platelet count, Activation, White cells), and the DEPA classification (Dose, Efficiency, Purity, Activation). These classifications allow us to distinguish between pure PRP (P-PRP), characterized by high platelet concentration, low leukocyte count, and low activation; and leukocyte-rich PRP (L-PRP), which includes the high platelet concentration fraction, with a higher proportion of leukocytes and proinflammatory cytokines (Tischer *et al*., 2020).

The choice between P-PRP and L-PRP should consider the stage of tendinopathy. In acute stages, L-PRP may be beneficial due to its immunomodulatory effect, while in chronic or degenerative injuries, P-PRP appears to induce less inflammation and promote tissue regeneration. However, further validated studies are still needed.

Regarding fibrin-rich plasma (PRF), it forms a three-dimensional matrix rich in fibrin that encapsulates platelets and leukocytes. Unlike PRP, PRF does not require anticoagulants or external activators, allowing for a progressive and sustained release of growth factors. This dense matrix facilitates the retention of the material at the application site and can act as a biological scaffold (Wang & Nirmala, 2016; Wesner *et al*., 2016).

The mechanism of action of PRP on tendons is based on its ability to promote tissue repair through the release of growth factors and cytokines following platelet activation. These factors include platelet-derived growth factor (PDGF), transforming growth factor beta (TGF-β), vascular endothelial growth factor (VEGF), epidermal growth factor (EGF) and IGF-1. These bioactive elements are released from the alpha granules of platelets once activated, initiating cellular cascades that promote tenocyte proliferation, type I collagen synthesis, and angiogenesis, thus facilitating tendon repair (Wang & Nirmala, 2016; Zhou & Wang, 2016; Kaux & Emonds-Alt, 2018; Bressler *et al*., 2020).

During the inflammatory phase, PRP acts by attracting inflammatory cells such as neutrophils and macrophages to the site of injury, facilitating the removal of necrotic tissue and improving the environment for regeneration (Zhou & Wang, 2016). At this stage, the use of L-PRP will modulate cytokines such as IL-1b and IL-6. Studies have shown that L-PRP stimulates receptors such as CCR1, CCR3, CCR4, and CXCR4, which are involved in cell migration and early inflammatory regulation, but it has also been associated with increased release of proinflammatory cytokines and more intense stimulation of chemotactic receptors (Kaux & Emonds-Alt, 2018; Liu *et al*., 2022).

In the proliferative phase, factors released by PRP stimulate the migration and proliferation of tenocytes and fibroblasts (Zhou & Wang, 2016; Liu *et al*., 2022). These cells synthesize an extracellular matrix rich in type III collagen, which is subsequently replaced by type I collagen. PRP also stimulates angiogenesis through VEGF, improving oxygenation and nutrition of the injured tissue (Wang & Nirmala, 2016; Zhou & Wang, 2016; Sebbagh *et al*., 2023).

During the remodeling phase, which usually lasts between 6 and 12 weeks depending on the type and severity of the injury, PRP promotes tissue maturation by increasing type I collagen synthesis and parallel fiber orientation (Zhou & Wang, 2016). In addition, PRP has been shown to decrease cell apoptosis and improve the fibrillar organization of the extracellular matrix. These structural improvements also demonstrate better scar tissue quality (Zhou & Wang, 2016; Kaux & Emonds-Alt, 2018; Liu *et al*., 2022). Figure 2 outlines the effect of PRP on the different stages of tendon repair.

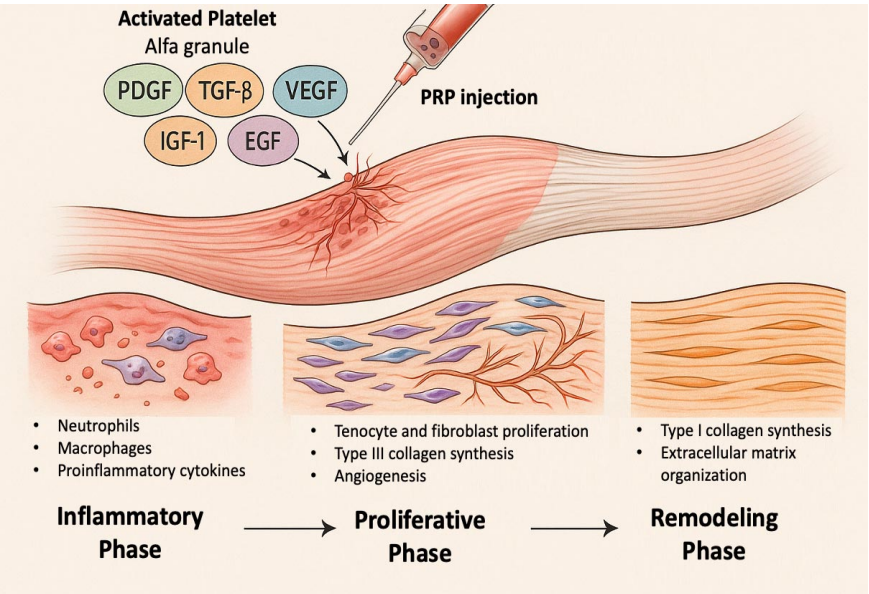

Fig. 2. Release of growth factors (Platelet-Derived Growth Factor (PDGF); Transforming Growth Factor Beta (TGF-b); Vascular Endothelial Growth Factor (VEGF); Insulin-like Growth Factor type 1 (IGF-1), Epidermal Growth Factor (EGF)) and effects of PRP administration in the different phases of tendon repair.

V.1 Effects of platelet concentrates on pain in tendinopathies

Several clinical studies and systematic reviews have evaluated the effect of PRP on pain reduction in tendinopathies, although results vary depending on the type of injury and the methodology used. The meta-analysis by Chen *et al*. (2018) shows that PRP generates a significant reduction in pain in lateral epicondylitis and rotator cuff tendinopathy, with a sustained effect in long-term follow-up. Abate *et al*. (2024) conducted a comparative clinical study between patients with and without type II diabetes mellitus (DM). In the control group, composed of 40 patients without a diagnosis of DM, the clinical response to a single PRP injection in the treatment of chronic tendinopathies of the calcaneal tendon or patellar tendon was evaluated, in which a reduction in pain was observed, as assessed by the visual analog scale (VAS), two months after the injection ($p<0.001$) and with a progressive trend of pain reduction up to six months. In most patients, pain reduction exceeded 50 % compared to baseline (Abate *et al*., 2024).

However, a recent review by Masiello *et al*. (2022), concluded that although PRP reduces pain compared to baseline, it was not superior to other treatments such as corticosteroids or saline solution in most tendinopathies, except in carpal tunnel syndrome. In addition, Cruciani *et al*. (2019), identified moderate pain reduction in some systematic reviews, but the effects are limited, and the methodological quality of the studies was low.

V.2 PRP protocols in tendinopathies

As for administration protocols, there is still no unified consensus. Most studies suggest 1 to 3 injections, with intervals ranging from 1 to 3 weeks, applications that were defined by clinical criteria and response to treatment (Wesner *et al*., 2016; Sebbagh *et al*., 2023). In the study by Tischer *et al*. (2020), a consensus statement was developed to guide the clinical use of PRP in cartilage, tendon, and muscle injuries, recommending 2 to 4 injections for chronic tendinopathies (Tischer *et al*., 2020). The importance of choosing the type of PRP and the number of injections according to the phase of the pathology (acute or chronic) is emphasized. On the other hand, the volume of PRP injected depends on the anatomical site (Kaux & Emonds-Alt, 2018; Sebbagh *et al*., 2023), and PRP activation can be performed using agents such as calcium chloride or thrombin, although some authors suggest that endogenous activation (upon contact with tissues) may be sufficient (Kaux & Emonds-Alt, 2018). In any case, various clinical studies highlight the lack of consensus and standardization of methods for obtaining, activating, and applying PRP (Chen *et al*., 2018).

V.3 Current treatments for tendinopathies of the Temporal Muscle Tendon

The treatment of TMT tendinopathies should be individualized depending on whether it is an acute inflammatory condition (tendinitis) or chronic degeneration (tendinosis).

The literature currently describes initial treatment measures to which TMT tendinitis regularly responds, such as patient education, elimination of the causative factor, jaw rest, iontophoresis, phonophoresis, analgesics and anti-inflammatories, eccentric exercises, muscle stretching, thermotherapy, or cryotherapy (Iturriaga *et al*., 2016; Okeson, 2019; Liu *et al*., 2022). Otherwise, second-line measures such as corticosteroid injections, such as betamethasone, or local anesthetic injections may be effective in controlling pain and limiting inflammation (Iturriaga *et al*., 2016). However, the use of platelet concentrates has not been described among these measures.

On the other hand, specifically in the diagnosis of TMT tendinosis, treatment has been addressed by Bressler *et al*. (2020), who point out that, despite the growing identification of tendinosis as a cause of chronic orofacial pain, there are still no standardized therapeutic protocols (Bressler *et al*., 2020). The use of conservative therapies focused on patient education, modification of oral habits, functional rest of the temporal muscle, and muscle relaxation techniques is highlighted. Local anesthetic and corticosteroid injections are also described as an effective measure to reduce pain in patients with a confirmed diagnosis (Dupont Jr. & Brown, 2012; Chen *et al*., 2018). Ernest 3rd *et al*. (1991), provide histological evidence of tendon degeneration at the TMT insertion and propose a progressive treatment approach. In cases that are most refractory to conservative and infiltrative therapies, surgical intervention is described, including tendon release or partial resection of the coronoid process. However, these procedures should be reserved for cases in which less invasive options have failed or within a therapeutic plan (Ernest 3rd *et al*., 1991).

Addressing the use of PRP, numerous studies cited above have shown that PRP can accelerate regenerative processes in other chronic tendinopathies. However, this implies that although there is theoretical justification and its use has been suggested, a series of cases or controlled trials formally evaluating the use of PRP in TMT tendinopathies has not yet been reported in the clinical literature (Bressler *et al*., 2020).

VI. Morphological considerations in the therapeutic approach to tendinopathies of the Temporal Muscle Tendon

From an anatomical point of view, the TMT has morphological characteristics that must be considered in its clinical and therapeutic evaluation. Although its infiltration is regarded as a safe procedure with no significant risks, it is essential to be familiar with its anatomy and topographical relationships.

The insertion of the TMT into the coronoid process of the mandible, crossing the infratemporal space below the zygomatic arch, places it in an anatomically complex region, close to vascular and nerve structures such as the deep temporal artery and the buccal nerve (Fuentes & Ottone, 2021). This anatomical relationship poses challenges for the differential diagnosis of TMD and minimally invasive treatments such as PRP injections.

In turn, the TMT can present morphological variations in its thickness, number of fascicles, and length, even with accessory insertions at the level of the mandibular ramus or extensions toward the maxilla. Furthermore, the length and insertion pattern of the TMT can vary significantly between individuals. In some cases, its insertion extends further inferiorly into the coronoid process, which could increase its exposure to functional stresses during wide mandibular openings, favoring inflammatory and/or degenerative processes. Anatomical and imaging studies have documented that the temporal tendon, with an average length of approximately 20 mm in the non-mobilized state, may have extensions that reach or exceed the inferior portion of the coronoid process and even extend toward the retromolar triangle region (Bénateau *et al*., 2001; Pinar *et al*., 2020; Yu *et al*., 2021). These morphological variations, observed in both magnetic resonance imaging and cadaver dissections, support the importance of considering the individual anatomy of the TMT in the diagnosis and treatment of its possible tendinopathies. It has also been observed that TMT can be divided into superficial and deep fascicles, with independent insertions (Nakagawa & Takahashi, 1997). These subdivisions may be clinically relevant as they can be differentially affected by microtears or degeneration and may require specific infiltration techniques.

Microscopically, TMT has a highly aligned organization of type I collagen fibers that allows it to resist the tensile forces generated during muscle contraction. However, the osteotendinous transition zone to the mandibular bone, composed of fibrous and fibrocartilaginous tissue, is an area of vulnerability to repetitive loads or excessive functional demands (Ernest 3rd *et al*., 1991). This region is also hypovascular, which limits its regenerative capacity in the face of chronic inflammatory or degenerative processes such as tendinosis. Although the blood supply to the TMT is limited, there is also variability between individuals, with some anastomoses originating from the deep temporal artery, the maxillary artery, and branches of the buccal artery (Hwang & Kim, 2006). In some cases, these anastomoses may be more developed, which influences the biological response to PRP and the distribution of the concentrate after infiltration.

From a clinical perspective, detailed knowledge of the morphology of TMT allows for the planning of more effective therapeutic approaches. The particularities of the tendon not only have diagnostic implications but are also decisive in optimizing therapeutic outcomes. Based on this, two anatomical sites are proposed for TMT puncture: one extraoral and one intraoral. The extraoral approach is located approximately 1 cm above the zygomatic arch, at the level of the myotendinous junction of the temporal muscle. The needle is angled 45° downward, with the puncture directed toward the superficial fascicle of the TMT (Fig. 3). This location, being in a superficial area, facilitates access and allows for effective peritendinous infiltration.

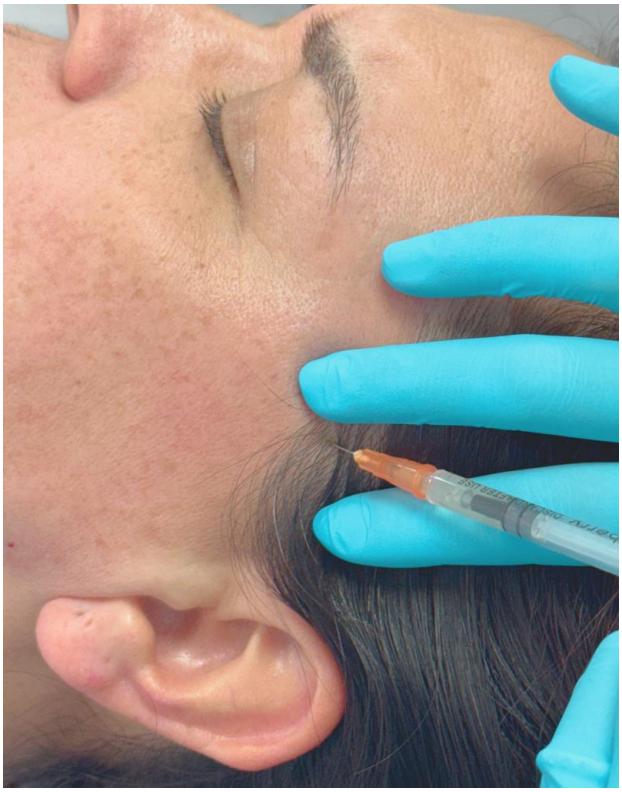

Fig. 3. Extraoral puncture technique for TMT.

On the other hand, the intraoral approach is more complex. The aim is to infiltrate PRP, or another substance, close to the medial aspect of the coronoid process, into the deep fascicle of the TMT. To perform this procedure, the patient must open their mouth as wide as possible, and the needle is positioned in the upper molar region with a vestibular orientation, at an angle of approximately 45° in a superolateral direction. The needle is advanced until it contacts the bone repair on the medial side of the coronoid process and then withdrawn slightly to deposit the liquid in the peritendinous space (Fig. 4). Both approaches can be performed during the same surgical procedure or independently, depending on the therapeutic objective.

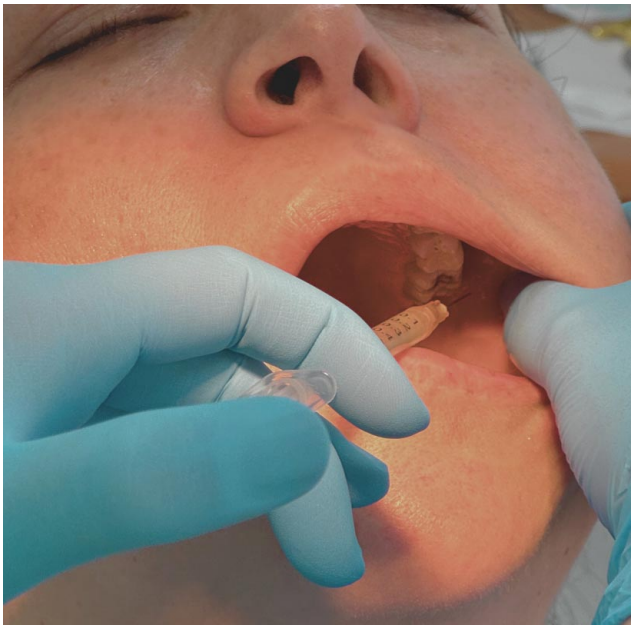

Fig. 4. Intraoral puncture technique for TMT.

LIMITATIONS OF THE EVIDENCE REVIEWED

Although the literature reviewed provides relevant theoretical and clinical evidence on the use of PRP in tendinopathies, some limitations must be considered, such as the diversity of PRP types, variations in administration protocols, and anatomical differences between the tendons studied, which make direct comparison between studies difficult. Furthermore, no studies were found that specifically focused on the administration of PRP in TMT tendinopathies, so the conclusions drawn are subject to extrapolation of the results.

CONCLUSIONS

Morphological analysis of the TMT shows that its dimensions, thickness, and insertion pattern present significant anatomical variability, which directly influences the pathophysiology of tendinopathies and the choice of therapeutic strategies. The deep location of the TMT and its proximity to bone structures such as the zygomatic arch and coronoid process condition both the clinical presentation and the planning of minimally invasive procedures. In this context, the use of PRP is emerging as a treatment alternative with great potential in acute and chronic degenerative tendinopathies with a low inflammatory component; however, the studies reviewed show differences depending on the type of PRP, the administration form, and the outcome variables, making it difficult to draw definitive conclusions. Incorporating these morphological and pathophysiological considerations into clinical practice allows for personalized intervention, minimizes risks, and promotes more predictable and satisfactory results in the management of this pathology.

HERNANDEZ, A.; ITURRIAGA, V.; VELASQUEZ, N.; MUÑOZ, J.; LANDAETA, H.; BORNHARDT, T. Tendinitis del tendón del músculo temporal: Implicancias morfológicas y fisiopatológicas en el manejo con concentrados plaquetarios - PRP. *Int. J. Morphol., 44(1)*:99-107, 2026.

RESUMEN: Las tendinopatías son una causa frecuente de dolor musculoesquelético y limitación funcional. Los concentrados plaquetarios, como el plasma rico en plaquetas (PRP), han surgido como una herramienta terapéutica para la regeneración de tejidos tendíneos, pero su uso específico en el tendón del músculo temporal (TMT) ha sido escasamente abordado. El objetivo de este artículo es realizar una revisión de la literatura sobre el uso de concentrados plaquetarios en tendinopatías, contextualizando su aplicabilidad en el TMT con base en su morfología, fisiología, fisiopatología y mecanismos de acción plaquetaria. Se realizaron dos búsquedas bibliográficas: la primera, sobre el uso de PRP en tendinopatías generales, en PubMed, Scopus y EMBASE; y la segunda, específica para el TMT, abierta y manual, orientada a identificar la mayor cantidad posible de artículos relevantes en las mismas bases de datos y libros académicos, sin restricción de año. Se identificaron 266 artículos, de los cuales 13 cumplían los criterios de inclusión sobre PRP y tendinopatías, y 15 artículos, para el TMT, centrados principalmente en su anatomía, variabilidad morfológica y correlación clínica. La información disponible sugiere que el PRP podría ser beneficioso en lesiones del TMT, gracias a su capacidad para promover la reparación tisular y reducir el dolor. Sin embargo, no existen estudios clínicos que evalúen directamente su eficacia en tendinopatías del TMT, lo que limita la posibilidad de establecer recomendaciones definitivas. Las particularidades anatómicas del TMT, incluyendo su profundidad, relaciones óseas y variabilidad de inserción, constituyen factores a considerar en la aplicación de terapias como el PRP. Se requieren investigaciones clínicas bien diseñadas que integren estas variables para definir protocolos efectivos y seguros para el manejo de tendonopatías del TMT.

PALABRAS CLAVE: Músculo temporal; Tendinopatías; Dolor facial; Plasma rico en plaquetas; Plasma rico en fibrina.

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