

THEORETICAL MODELS IN THE SPORTSMAN PATELLA TENDINOPATHY OR JUMPER ´S KNEE PAIN (SÁNCHEZ, JM; 2003)

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Introduction

"Concepts are not supported by definitions. Precisely, concepts cannot stand the inalterability of definitions. They are not theoretically fixed, and later they are forced to work in clinic. Concepts correspond to a articulation of theory, to the method and the technique. This means that it is no succession, but an articulation..."

During the last years, the proportion of cases where patella tendinopathy appears has grown. This is probably due to the fact that sportsmen in general train and do competition for longer, and due to the higher knowledge of this pathology both for sports practitioners and for sports therapists.

Patella tendinopathy is mostly observed in sports where jumping is common: for example, basketball, volleyball, height jump. During this last decade, an exponential rise of this injury has been seen in european football (soccer). The number of studies on the etiopathogenesis, histopathology, clinics and biomechanics of this pathological entity are more frequent, but there is no unanimous meeting point about what the therapeutic orientation should be. In many cases, many sportsmen or athletes have to leave their sports activity during some months, or they may be even forced to abandon their sports life. Maurizio (1963) was the first one to observe the relationship between the patella tendinopathy in voleibal player, but it was Blazina et al (1972) the ones to name this pathological entity as "jumper ´s knee", after observing a high incidence in the athletes knee extensor apparatus whose sports involved repetitive, sudden and boosting movements of the knee.

The patella and the quadricipital tendon may be involved in the injury. The causes that make the tendinopathies arise are different. They are probably the result of repetitive and extended mechanical tensions which force the tendon to its elasticity limit (4 – 8 %) and cause cumulative micro-injuries, repetitive microtraumas by traction forces, in the specific case of patella tendon.

This type of tendinopathies due to overuse are more and more common in our dially practical clinic. Many researchers worldwide have remarked that the essential pathology in this conditions is tendinosis or collagen degeneration (Jozsa L et al, 1997; Khan, K et al , 1999, Puddu, G et al 1976). If we admit that overuse tendinopathies are due to a tendinosis, not a tendinitis, we should modify the therapeutic approach to our patients. If this statement is correct, then the traditional approach of tendinopathies as "inflammatory tendinitis" may also be wrong.

"Jumper´s knee" is considered as a typical functional overload tendinopathy which is located in a 65% of it in the insertion of the patella tendon in the inferior patella pole, in a 25% in the quadriceps insertion in the patella base, and in a 10% in the insertion of the patella tendon in the tuberosity of the tibia (Ferreti, 1986). These data give us an example how the most vulnerable region to repetitive microtraumas is the osteo-tendinous joint, more precisely, the more complex histological region and where more sensitive receptors gather.

Theoretical model of pain in the patella tendinopathy

The question we must ask ourselves as therapists is: which is the origin of pain in the patella tendinopathies? Even if the most common answer would be "in the inflammatory cells", it would probably be wrong (Khan et al 2000). Scientific discoveries demonstrate that the overuse patella tendon injury (from now on, patella tendinopathy) is no inflammatory process in a primary state. If it is not an inflammatory process, the question we must place is: what are the non inflammatory mechanisms that may produce pain in the patella tendon? There may be several proposals to explain pain in tendinopathies: from the most traditional model on inflammatory tendinitis, to the mechanical model, the biochemical model, and the neural model.

Traditional model on "inflammatory tendinitis"

Traditionally, it has been accepted that the tendon overuse produced inflammation and, therefore, pain. The clinical name "patella tendinitis" implies that there is inflammation, and the studies with ecography and MRN showed the existance of "inflammatory fluid" around the symptomatic patella tendons. This fact reenforces this model (Mc Loughlin et al 1995). But a study of Peddu et al 1976, demonstrated that in the Achilles tendinitis there was collagen separation and fragmentation. He named this stage "tendinosis", being a term that had already been used by the Germans in the forties. From then on, several authors have demonstrated that this

anatomopathological process is the most frequent discovery in the patella tendinopathies (Khan et al 1999). Macroscopically, they observed that patients with patella tendinopathy had a soft-consistence tendon and disorganized collagen fibers which had a brownish yellow colour in its deep posterior region adjacent to the patella's inferior pole (Karlson et al 1992; Karlson et al 1991; Raatikainen et al 1976). This macroscopic look is described as mucoid or mixoid degeneration. Through the microscope, it is seen how the collagen fibers are disorganized and separated by an increase in the ground substance. Consequently, collagen degeneration, a variable fibrosis and a neovascularization were the constant discoveries in the previously mentioned studies.

Those ecography and MRN images which were interpreted as existence of "inflammatory fluid", correspond to areas with collagen degeneration, mucoid degeneration, and increase in the ground substance.

Some authors suggest a transition process from a normal tendon to tendinosis, through a previous stage of "inflammatory tendinitis". There is no proof which demonstrates that there is a provisional "itis" stage in the overuse tendinopathy. There are studies which demonstrate that there was no presence of inflammatory cells in a tendinopathy, what suggests that if there really is a provisional tendinitis stage, it must be very short (Kannus et al 1991; Khan et al 1997). Enweneka 1989, studied how long the tendon inflammatory process lasted in a surgical tenotomy model, transversally sectionning the Achilles tendon in rats. This intervention produced an intense inflammatory response that rised to its maximum point within five days, and disappeared withing eighteen days. The tenotomy, just like the percutaneous electrolysis technique, produced more inflammation than an overuse injury model. Inflammatory cells disappeared in three weeks after the surgical agression (Enweneka 1989). These data suggest that the tendinous repairing process is not long. Following the same statements, the Transtendinous Percutaneous Technique (TPET) produces several fenestrations by the multiple application of needles, and generates a beneficial bleeding inside the new channels created through a mucoid degeneration tissue. This collagen fiber rupture produced by electrolysis can transform a low scarring response in an extrinsic therapeutical response (Sánchez, JM; 2003).

Scientific data suggest that symptomes that persist more than a week, must have their origin in a non-inflammatory mechanism, even though it may exist an inflammatory period that could last for a few days.

Mechanical Model

Mechanical models suggest that pain is produced under two conditions: the former is due to a collagen fiber injury, and the latter is due to those who associate pain to a "tissue impingement". Those who affirm that pain is the consequence of the collagen fibers injury refer to the fact that the collagen fibers aren't the pain origin whenever they are intact, but they are when they break. There are many situations where the tendon is not completely intact and there is no pain nevertheless. A variable in the mechanical model states that it is not the collagen rupture the one that produces pain itself, but it is the intact persistent collagen which remains contiguous to the injured tissue the one that originates pain. This is thought because it would be subjected to a larger load as a result of the injury in the contiguous collagen.

It is thought that pain is generated when the proportion of damaged collagen tissue rises up to a critical threshold and the remaning collagen is submitted to a stress level which overcomes its normal load capacity. Nevertheless, the data from numerous studies which have used image techniques contradict this model (Lian et al 1996; Cook et al 2000; Khan et al 1997), because patients with patella tendon pain may have a normal MRN. On the other hand, clinical experience provides many exceptions to the theory exposed: a patient may have a very small morphological anomaly, or do not have any at all, and show meaningful symptomes. It has been demonstrated by the studies that a large number of asymptomatic sportsmen presented hypoecic regions in the ecographic studies, even in subjects without jumper's knee antecedents (Lian et al 1996; Cook et al 1998; Cook et al 2000). These discoveries demonstrate that tendon pain is due to something else besides the collagen continuity loss.

The mechanical model of tissue impingement accepts that both the patellar tendon and the infrapatellar fat are located in a location where they could suffer a compression by the patella and the proximal tibial extreme. This could actually be the origin of pain in the patella tendon (Jonson 1996). This same author explains that the patella tendon defeat against tension stress would affect more the superficial rather than the deep fibers. He would suggest that the origin of pain in the patella tendon is the consequence of the patella's inferior pole impingement against the tendon during the flexion of the knee. There are three clinical observations which are against this proposal: pain starts in the first stage by the time the sportsman touches ground after jumping with the knee in extension; in long evolution patients, pain starts even with the knee in discharge and extension, and pain is more symptomatic and pathognomonic to palpation in the deep fibers proximal area. Supposedly, impingement syndrome patients should obtain a symptomatic improvement when the knee is in extension. On the other hand, biomechanical studies show that the superficial fiber anchorage is stronger than the deeper fibers (Evans et al 1990; Evans et al 1991). It is therefore probably that the failure by tension stress affects more the deep fibers rather than the superficial ones.

Regarding the authors who defend the impingement mechanical theory of infrapatellar fat as the cause of

tendon pain, we must differ between two situations: the patella tendinopathy pain is referred to the infrapatellar fat, and the infrapatellar fat itself is considered as the cause of anterior knee pain. Infrapatellar fat is a very sensible structure (Dye et al 1998) with many nociceptors. This fat can take a specific shape of nociceptive peritendinous tissue which is sensitive to biochemical irritants. In other words, infrapatellar fat may play the same role in the patellar tendon than the paratendon in the Achilles tendinopathy. Mac Conell (1986) has been one of the first ones to highlight that infrapatellar fat was the cause for the pain and, therefore, that pain doesn't have to be exclusively tendinous.

Biochemical Model

The biochemical model is presented as a very attractive alternative to the previous models. Nirsch (1999) stated that the tendinosis pain cause is a chemical irritation due to regional anorexia and the lack of phagocytic cells to remove the cellular activity harmful products. The patella tendinopathy pain could be caused by biochemical factors which activate the peritendinous nociceptors (Kranshcar et al 1999).

The sulfate chondroitin which is released when the tendon damages may stimulate the nociceptors (Jozsa et al 1993; Brukner et al 2001). In the knee, the nociceptors are located in the medial and lateral patella sides, the infrapatellar fat, the synovial and the periosteum (Wintonski et al 1999) and all the structures may play an active role in the patella tendinopathy pain origin. In studies done with corpses who had suffered from patella tendinopathy, it has been observed a fine layer of fat stucked onto the back side of the patella tendon in its insertion at the inferior patella pole (Khan et al 1996). It is thought that the P substance and the neuropeptides related to it which are located next to the collagen fibers are involved in the tendon nociception (Marshall et al 1994).

Alfredson (2002) studied the lactate concentration in tendinosis with the microdialysis technique, comparing it to a normophysiological control group. It has been observed an increase in the lactate concentration in patients with tendinopathy, finding in the data meaningful statistic differences ($p < 0,005$). The high concentration of lactate in the pathological tendons show that there are anaerobic conditions in the tendinosis area. This may be a possible explanation for the pain.

Neural Model

Neural damage and hyperinnervation have been some of the theories which have been scarcely studied in the scientific bibliography (Fulkerson et al 1983), despite its attractive to explain the possible physiopathology pain mechanisms in patella tendinopathies. It seems that the positive nervous substances for P substance in the knee are located in the infrapatellar fat and in the internal patella aleron (Witonski et al 1999). Sanchis V. et al 1998, did a study where they demonstrated a correlation between patella tendon tendinopathy and hyperinnervation (Sanchis V. et al 1997; Sanchis V. et al 1999). This nervous fibers growth could belong to a anomalous repairing tissue process, preceded by repetitive microtraumas (Freemant et al 1997).

It seems that production of neural growth factor (NGF) and the consequently hyperinnervation may be induced by ischemia (Lee et al 1997). The neural theory correspond to the clinical discoveries which show mechanical alodinia in the inferior patella pole in patients suffering from patella tendinopathy. In our series, we have found a considerable number of cases which presented an hypersensitive region, very located in a small surface, on the superficial patella inferior pole region, which could be related to a region hyperinnervation. Through the application of one only session of Transtendinous Percutaneous Electrolysis (TPET), the symptoms disappeared definitively, reaching its maximum efficiency point at the third day of the application. On the other hand, the ischemical periodical crisis in the patella tendon may take place as a result of the force vectors in torsion and/or shear of the vessels of the proximal region in the inferior patella pole. These torsion crisis, related to the cyclical ischemia will produce the NGF release of axons and vessels, being responsible of the vascular and perivascular hyperinnervation (Sanchis et al 1998). Remembering that NGF stimulates the P substance release, nociceptive neurotransmitter (Lee et al 1996). Sanchis V. et al 1998, verified the existence of transitory repetitive ischemic episodes, finding vasculitis phenomena, fibroblasts with autophagic vacuoles, neoangiogenesis and neural germination. In these discoveries, we can observe the existing correlation with the discoveries found in the histopathological studies of tendinosis (Khan et al 1997).

As a result of the collagen fiber destruction and the increase in the volume of the mucoid substance, the extracellular environment acquires a standard of "homeostasis paralysis". Tenocytes use the glycolytic anaerobic metabolism for its survival in order to respond to its metabolic lack of oxygen and nutrients. If this toxic environment is not solved, the cell starts a journey towards its death, showing the histopathological study the existence of big lipolytic vacuoles which will end throwing cytotoxic substances to the extracellular space (glutamate, lactate, catabolites), substances which act as biochemical irritants of the afferent neural system.

When there is an injury in the tendon by degeneration, the damaged cells (tenocytes) together with the blood vessels release toxic chemical substances which impact against their neighbour sane cells. One of these substances is the glutamate aminoacid of negative charge, which produces a process known as excitotoxicity. When this tendon degeneration is produced, cells release huge quantities of this neurotransmitter, overexciting the neighbour cells and allowing the big ions entrance, causing destructive tissues processes. Alfredson (1999) did a study with the microdialysis technique to value the concentration of glutamate and prostaglandins E2 in

the pain of Achilles tendinopathy.

Microdialysis allows to study the substance concentrations in the tendons. It was observed an increase of the located glutamate concentrations in the group of tendinopathies related to the control group, the differences were statistically meaningful ($p < 0,005$). But on the other side, there weren't observed statistically differences on the prostaglandins E2 concentrations of the tendinopathy group related to the control ($p > 0,05$). Microdialysis seems to be a correct method to certainly study the metabolic tendon events. Larger concentrations of NT of excitatory glutamate in Achilles tendinopathies with painful nodules may be involved in the pain mechanism. Any inflammatory sign was observed in the tendons, since there were normal levels of prostaglandins E2.

In other study through the microdialysis technique and immunohistochemical analysis of the tendinous tissue in patients with patella tendinopathy, it was demonstrated a high concentration of glutamate and NMDAR1 receptors, but with inflammation signs (Alfredson et al 2001). Meaningful differences greater than the free glutamate are observed, but not of prostaglandins E2 (PGE2) in patella tendinopathies. In the biopsies, there was no presence of inflammatory cell infiltration, but it was observed a immunoreaction by the glutamate NMDAR1 reception in association with nervous tendons structures. These discoveries show that glutamate may be involved in the patella tendinopathy pain and emphasizes that there is no chemical inflammation, since the PGE2 levels are normal in these chronic conditions.

Glutamate plays an important role in the synaptic excitatory transmission, process through which neurones communicate ones another. An electrical impulse (action potential) in one of these cells produces a calcium input with the consequently release of this neurotransmitter. The neurotransmitter spreads through the synaptic cleft and hooks onto the next cell's receptors. These receptors are by themselves ionic channels that open up when the neurotransmitter hooks, allowing the Ca^{++} and Na^{+} ions to enter through them. This ion flow produces the depolarization of the plasmatic membrane with the generation of an electrical current that spreads onto the following cell. Glutamate is one of the principle excitatory neurotransmitters of the nervous system and acts through the ionotropic and metabotropic receptors. The activation of these receptors is the responsible for the excitatory synaptic transmission. Glutamate receptors, specially the ones from the NMDA family, are involved in cytotoxic disorders. The glutamate concentration in the synaptic cleft depends on the quantity of glutamate released, of the speed at which it is released, and at the speed at which it is removed from the synaptic cleft. The synaptic accumulation of high quantities of glutamate and its extended action over the postsynaptic glutamate receptors could be caused by a tissue degeneration or to an alteration in the receiving mechanisms due to a failure in the glutamate transporting proteins. The tendon degenerative events produce a cell lysis with the consequent release of intracellular glutamate. The oxidative stress can be a reason for cellular lysis, and stimulates the generation of free radicals which reduce the efficiency of the glutamate transporters. Therefore, the extracellular levels of glutamate rise. The NMDA receptor conditions the structural changes in the cell and makes that it stays activated for longer (fig.1).

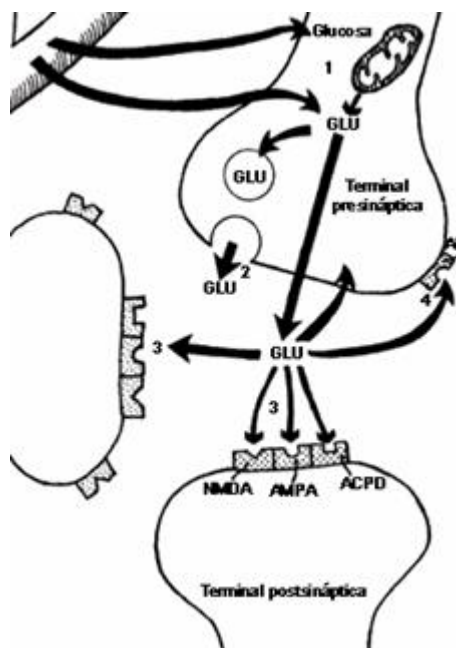


Fig. 1: The glutamatergic synapse. Glutamate (GLU), a prime excitatory amino acid, is taken directly by the blood and the extracellular space, or through glucose and the metabolic conversion in the presynaptic ending (1). It may be directly released both from this ending or from the vesicle storage (2). The GLU may occupy three types of postsynaptic neuronal receptors (3). These receptors are named according to the substance with which they interact: the NMDA receptors (N-metil-D-aspartate), the no NMDA (sensitive to AMPA) and the

metabotropic, sensitive to the transamino-ciclo pentano-dicarboxilic acid (ACPD). The aminoacid may also interact with autoreceptors (AR) (4).

Integration Model

Once studied and presented the different models of pain production in the patella tendinopathies, it is logical to expect that my opinion balances forwards to an integrative model in the tendinopathy's pain origin. From the mechanical posture, we consider that the cyclical stress excess in the tendon will modify the vascular standards, producing focused repetitive ischemia. This will bring as a consequence in a disorder in the tendon vascular repairing mechanisms. (Sanchis, V. et al 1998).

In activities which require exercises that limit the supraphysiological load threshold, a disorder in the tendon's basal metabolism recovery will be produced. Cyclical ruptures of collagen fibers stimulate the release of cytotoxic substances in the interstitial space, which will act as neuronal and metabolic biochemical irritators, stimulating the glycolytic anaerobic metabolism of tenocytes. These free aminoacids and proteins, released by the cells in their final necrosis stage, modify the interstitial pH, amplifying the glutamate neurotransmitter release and the neuroplasticity of the NMDAR1 receptors. These elements are the precursor neurological mechanisms of the maintenance of the nociceptive nervous fibers depolarization. The endurance of the nociceptive depolarization stimulates the reduction in the exciting threshold and the appearance of the mechanical allodynia.

This integrating paradigm of the mechanical and neurobiochemical models makes it easier to understand the possible etiopathogenesis of pain in the front area of the knee, located at the patella tendon. To know the different mechanisms of pain in the tendon will allow us to establish a correct approach in its therapy in order to achieve its healing. If we analyse this pathology on a integratory perspective basis, we will achieve more satisfactory results than if we only approach the disorder from only one paradigm (Table 1).

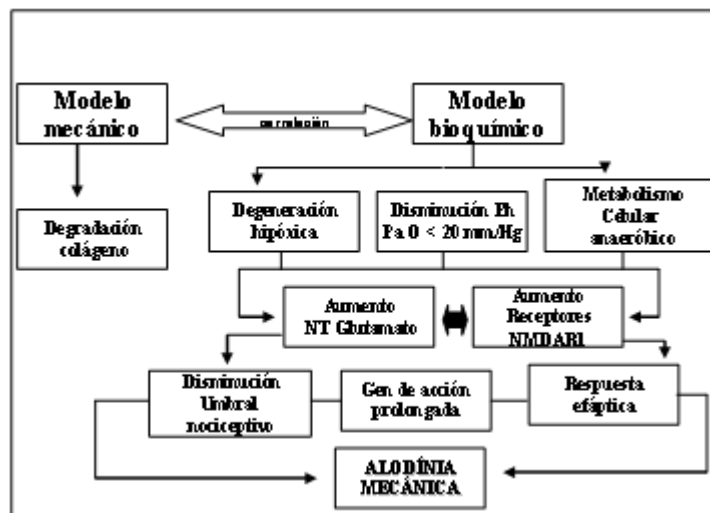


Table 1 : Integrating model of pain origin in patella tendinopathies. The supraphysiological cyclical charges produce the collagen fibers degradation. The collagen fibers rupture stimulate the release of irritating biochemical substances. The increase in volume of ground substance as a result of the mixoid degradation, modifies the interceluar space pH, producing the anaerobic glycolytic metabolism activation by the tenocytes. During the cytological necrosis stage, high concentrations of glutamate secretion are released, stimulating the extended nociceptive depolarization. The neural extended stimulus produces a NMDAR1 receptor neuroplasticity, generating finally the mechanical allodynia (Sánchez JM 2003).

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