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# Clinical Management of Acute and Chronic Soft Tissue Injuries: Integrated Orthopedic Techniques and Rehabilitation Protocols

**Zhe Yuan[1], Zequn Wang[1], Zhou Zhou[1], Jichao Yin[2,*]**

[1]Shaanxi University of Chinese Medicine, Xianyang 712046, Shaanxi, China
[2]Xi'an Hospital of Traditional Chinese Medicine, Xi'an 710021, Shaanxi, China
*Correspondence Author*

**Abstract:** ***Soft tissue injury is a common clinical problem, and its repair involves multi-dimensional biological mechanisms. This article aims to explore the pathophysiological characteristics of acute and chronic soft tissue injuries, as well as the latest repair strategies and research progress by searching the literature related to acute and chronic soft tissue injuries in recent years. This article discusses in detail the biological mechanisms of soft tissue repair, including the role of cells, extracellular matrix and growth factors. This paper summarizes the current traditional methods and emerging therapies for the treatment of soft tissue injuries. The latest clinical research and experimental findings were discussed, and current challenges and future research directions were pointed out. This study aims to provide a comprehensive perspective for understanding soft tissue injury and repair, and is of great significance to clinical practice and basic research.***

**Keywords:** Acute and chronic soft tissue injury, Soft tissue injury, Pathophysiology, Integrated traditional Chinese and western medicine treatment, Biomaterials, Inflammation-fibrosis axis, Repair**.**

## 1. Introduction

Soft tissue injuries are a common health problem in daily life and involve muscles, tendons, ligaments and other related structures. These injuries can be caused by sports, accidents, or other accidents that not only affect the individual's daily activities, but can also lead to long-term disability and pain. Its high incidence and complex repair mechanisms make it a focus issue in orthopedics and rehabilitation medicine. Soft tissue injuries can be divided into acute injuries and chronic injuries, and there are significant differences between the two in pathophysiological processes, clinical manifestations and treatment strategies [1]. Acute injuries are centered on the inflammatory waterfall reaction, while chronic injuries are delayed due to fibrosis and microcirculation disorders. Traditional Chinese medicine theory emphasizes that "if qi and blood are blocked, it will cause pain", which is highly consistent with the inflammation-fibrosis axis revealed by modern molecular biology. Although a large amount of research has been devoted to understanding the mechanisms of these injuries and finding effective treatments, the repair of acute and chronic soft tissue injuries remains a complex and challenging area. In recent years, with the advancement of molecular biology, biomaterials and tissue engineering technologies, our understanding of soft tissue damage and repair has improved significantly. New treatment strategies and the use of biomaterials provide new possibilities for the treatment of soft tissue injuries. This review aims to comprehensively analyze the pathophysiological characteristics of acute and chronic soft tissue injuries, explore the latest repair strategies and research progress, and point out future research directions.

## 2. Definition and Classification of Soft Tissue Injury

### 2.1 Acute Soft Tissue Injury

Acute soft tissue injuries are usually tearing injuries to muscles, tendons, ligaments or other local soft tissue to varying degrees caused by high-energy external forces (such as sports collisions, falls). This injury is often accompanied by local bleeding, pain, swelling, blood stasis and dysfunction. Common causes include acute lumbar sprain, limb joint sprain, etc. For example, "The Wings of the Golden Chamber" says: "If you have congestion and low back pain, you will get it from flash and strong Aram [2]. "The incidence rate has gradually increased in recent years [2] [3].

### 2.2 Chronic Soft Tissue Injury

Chronic soft tissue injuries are often formed under circumstances that are not easily detectable by the human body. They are soft tissue injuries caused by long-term, repeated stress or micro-trauma accumulation, and there is no obvious history of trauma. Its pathological essence is a vicious cycle of "fibrotic microenvironment". This kind of injury may be caused by overuse, improper posture or occupational activities. It can be more common in workers who have been engaged in a single position for a long time, causing some muscles, ligaments, fascia, etc. related to the human body to be in constant tension for a long time. During static tension, local blood flow, poor circulation, ischemia and hypoxia in these affected tissues will be reduced, resulting in gradual changes in fibrous tissue, which will persist for a long time, and gradually aggravate to form chronic cumulative damage. Also known as "strain" [4] [5]. Such as long-term stoop labor appears lumbar muscle strain, long-term bow work appears neck and shoulder soreness, etc. Chronic injuries can also evolve from acute injuries. The pathophysiological processes of chronic injury are more complex, involving chronic inflammation, fibrosis, tissue remodeling and scar formation. These processes can lead to persistent pain and dysfunction that are more difficult to treat.

### 2.3 Differences and Connections Between Acute and

**Chronic Soft Tissue Injuries**

There are significant differences between acute soft tissue injury and chronic soft tissue injury in etiology, pathophysiological process and clinical manifestations. Acute injuries usually have a clear starting point, while chronic injuries may develop gradually. However, there is also a connection between the two. For example, if acute injury is not properly treated and rested, an imbalance in matrix metalloproteinase-9 (MMP-9)-mediated extracellular matrix (ECM) degradation can lead to delayed repair and transformation into chronic injury. In clinical practice, chronic cumulative injuries are often more frequent than acute injuries.

## 3. Pathophysiology of Acute Soft Tissue Injury

The pathophysiological processes of acute soft tissue injury involve complex biological reactions that are the body's natural response to injury, designed to limit injury, clear damaged tissue, and initiate repair processes, including inflammatory responses, blood coagulation and fibrinolysis, as well as cell death and survival mechanisms. In the process of pathological reactions, it is mainly the secretion and release of various inflammatory mediators and various pro-inflammatory cytokines triggered by sterile inflammatory reactions [6] [7].

### 3.1 Inflammatory Response Immediately After Injury

After acute soft tissue injury occurs, the inflammatory response is the body's first line of defense against the injury, and the inflammatory response will occur quickly at the injured site. This process includes vascular dilation, increased vascular permeability, and leukocyte infiltration. Vascularization and increased permeability cause blood components and fluids to exude into the injured tissue, forming local edema and redness. This process is mainly mediated by inflammatory mediators such as histamine, prostaglandin and leukotrienes. Histamine is released by mast cells and can quickly cause blood vessel dilation and increased permeability; prostaglandins further aggravate the inflammatory response by promoting blood vessel dilation and increasing pain sensitivity. Leukocytes, especially neutrophils, are the main effector cells in the early stages of inflammation, migrating to the site of injury through chemotactic action to clear bacteria and damaged tissue [8]. Polymorphonuclear neutrophils (PMNs) and macrophages play key roles in the inflammation phase. The former is mainly responsible for clearing pathogens, while the latter cleans necrotic tissue through phagocytosis and secretes various cytokines and growth factors to promote subsequent repair process [9]. Reactive oxygen species (ROS) and proteases (such as elastase) released by neutrophils can also cause damage to surrounding healthy tissue while clearing pathogens. Subsequently, monocytes and macrophages gradually replaced neutrophils and became the main cell type in the later stages of inflammation. Macrophages not only clean up necrotic tissue through phagocytosis, but also promote tissue repair and regeneration by secreting a variety of cytokines such as tumor necrosis factor (TNF-α), interleukin (IL-1β), interleukin (IL-6) and growth factors such as transforming growth factor (TGF-β), platelet-derived growth factor (PDGF) [10].

### 3.2 Imbalance of Coagulation-fibrinolytic System

Blood clotting is another important process that occurs immediately after acute injury. Acute soft tissue injury may lead to exposure of vascular endothelial cells. Blood clotting activates platelets and coagulation factors to form a fibrin clot, which stops bleeding. Platelets not only play a role in hemostasis during coagulation, but also initiate the repair process by releasing various growth factors (such as PDGF, TGF-β). At the same time, the fibrinolytic system is gradually activated after coagulation, and plasminogen is converted into plasmin through plasminogen activators (such as tPA). Plasmin degrades fibrin clots, removes blood clots and tissue debris, and creates conditions for tissue repair and regeneration [11].

The balance of the fibrinolytic system is crucial for tissue repair. Excessive activation may lead to bleeding, while insufficient activation may lead to excessive fibrin deposition, affecting tissue repair. Studies have shown that overexpression of plasminogen activator inhibitor-1 (PAI-1) leads to abnormal deposition of fibrin and hinders regeneration [12]. Clinical data show that local application of hirudin can inhibit PAI-1 and improve repair efficiency ($P<0.05$) [13].

### 3.3 Mechanisms of Cell Death and Survival

Acute injury may lead to local cell death, mainly including necrosis and apoptosis. Necrosis is a passive death process caused by severe damage to cells or hypoxia. It is usually accompanied by the rupture of cell membranes and the release of intracellular substances, triggering an inflammatory response. Apoptosis is an active programmed death process of cells that usually does not trigger an inflammatory response. At the damaged site, surviving cells and stem cells begin to secrete a variety of growth factors and cytokines, promoting the repair process. These factors include vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF), platelet-derived growth factor (PDGF), etc. They promote tissue repair and regeneration by stimulating angiogenesis, cell proliferation and matrix deposition. In addition, stem cells also play an important role in injury repair. They can differentiate into multiple cell types and participate in tissue reconstruction.

### 3.4 Pain and Dysfunction

Acute soft tissue injuries are often accompanied by pain and dysfunction. Pain is due to the release of inflammatory mediators and increased sensitivity of the central nervous system. Dysfunction may be due to pain, swelling and local structural damage.

## 4. Pathophysiology of Chronic Soft Tissue Injury

The pathophysiological mechanism of chronic soft tissue injury is more complex than that of acute injury, involving multiple links such as chronic inflammation, fibrosis, microcirculation disturbance and tissue remodeling.

### 4.1 Chronic Inflammation and Fibrotic Processes

The inflammatory response of chronic injury is dominated by infiltration of monocytes and lymphocytes, with the continuous release of pro-inflammatory factors (such as IL-1β, TNF-α) and pro-fibrotic factors (such as TGF-β1), resulting in excessive activation of fibroblasts and secretion of a large number of collagen fibers, forming pathological scar tissue. TGF-β1 is a key factor in the fibrosis process. It promotes fibroblast proliferation and collagen synthesis through the Smad signaling pathway, while inhibiting collagenase activity, resulting in excessive collagen deposition [14]. Traditional Chinese medicine theory believes that chronic injuries are mostly caused by "chronic illness entering collaterals" and "blood stasis resisting resistance", poor local circulation of qi and blood, and mutual accumulation of phlegm and blood stasis, ultimately leading to "tendon knots" and "tendon arthralgia" [15]. Studies have shown that in long-term ischemic and hypoxic environments, mitochondrial dysfunction and oxidative stress intensify collagen deposition and further aggravate fibrosis. Oxidative stress causes cell membrane lipid peroxidation, protein denaturation and DNA damage by producing large amounts of reactive oxygen species (ROS), which in turn triggers apoptosis and tissue fibrosis [16].

### 4.2 Tissue Remodeling and Scar Formation

During the repair of chronic injuries, the ratio of type III collagen to type I collagen is unbalanced (normally 1:4, and scar tissue can reach 1:10), resulting in reduced tissue elasticity and limited function. Type III collagen is mainly found in new tissues and has good elasticity, while type I collagen is found in mature tissues and has high strength. In chronic injuries, excessive deposition of type I collagen leads to the formation of scar tissue, affecting the normal function of the tissue [17]. Studies have shown that the active ingredients in pangolin can inhibit the expression of TGF-β1, reduce collagen synthesis, and thereby reduce fibrosis. Through the "soft and hard masses" method (such as using pangolin, turtle shell and other drugs), the TGF-β/S mad signaling pathway can be inhibited and excessive collagen deposition can be reduced [18].

### 4.3 Microcirculation Disorders and Hypoxia

Chronic injury has insufficient local vascular regeneration, dysfunction of endothelial cells, and reduced blood perfusion, forming a vicious cycle of "blood stasis-hypoxia-fibrosis". Vascular endothelial growth factor (VEGF) is a key factor in angiogenesis. It promotes the formation of new blood vessels by promoting the proliferation and migration of endothelial cells. However, in chronic injury, VEGF expression is often suppressed, leading to insufficient angiogenesis and hypoxia in local tissues. Studies have shown that tanshinone in salvia miltiorrhiza can promote the expression of VEGF by activating the PI3K/Akt signaling pathway, thereby improving local blood perfusion and reducing fibrosis [20]. In chronic injury, glycolysis of fibroblasts is enhanced (Warburg effect), and lactic acid accumulation further activates hypoxia-inducible factor (HIF-1α). Ligustrazine reverses metabolic abnormalities by inhibiting lactate dehydrogenase (LDHA) activity and improves tissue oxygenation (pO2 increased by 28%) [21].

## 5. Biological Mechanisms of Soft Tissue Repair

### 5.1 Processes of Regeneration and Repair

Dynamic balance of fibroblasts and ECM: During the proliferative phase, fibroblasts proliferate in large numbers and synthesize ECM components, such as collagen and proteoglycans. It regulates the repair process by secreting a variety of growth factors (such as TGF-β and PDGF) and cytokines (such as IL-6 and IL-10). This period is characterized by the formation of granulation tissue, accompanied by the growth of new blood vessels (angiogenesis), which provide the necessary nutrients and oxygen [22] for tissue repair. Collagen remodeling: Over time, type III collagen is gradually replaced by type I collagen, and the strength and structure of the tissue is gradually restored. TGF-β plays an important role in this process, which regulates collagen synthesis and deposition [14] through the Smad signaling pathway.

Angiogenesis and nerve regeneration: VEGF and ANG-1 cooperate to promote neovascularization, while BDNF and NGF support nerve fiber regeneration [23].

### 5.2 The Role of Cells and Extracellular Matrix

Mesenchymal stem cells (MSCs) regulate immune responses through paracrine effects and differentiate into tendon cells or fibroblasts. MSCs are able to secrete a variety of growth factors (such as VEGF, FGF, HGF) and cytokines (such as IL-10, TGF-β) to promote angiogenesis, cell proliferation, and matrix deposition, thereby accelerating repair [24]. The mechanical properties (such as stiffness) of the extracellular matrix (ECM) affect cell migration and differentiation through integrin signaling. The stiffness of ECM can promote the proliferation and collagen synthesis of fibroblasts by activating the RhoA/ROCK signaling pathway. However, excessive stiffness may lead to fibrosis and scar formation [25]. Among the growth factors and cell signaling pathways, TGF-β pathway dominates the fibrosis process, and inhibiting its excessive activation is the key [26] to anti-scar treatment. Wnt/β-catenin pathway can promote stem cell self-renewal, but persistent activation may lead to heterotopic ossification [27].

## 6. Therapeutic Approaches for Acute and Chronic Soft Tissue Injuries

### 6.1 Traditional and Emerging Therapies for Acute Injuries

6.1.1 Traditional therapies

Traditional treatment: The RICE principles (Protection, Ice, Compression, Elevation) are the gold standard for acute treatment. Ice application reduces prostaglandin E2 (PGE2) synthesis by inhibiting cyclooxygenase (COX) activity, thereby relieving pain and swelling. Compression bandaging reduces interstitial pressure and exudate accumulation.

External application of traditional Chinese medicine: such as

Qili powder and Yunnan Baiyao can quickly relieve swelling and pain. Panax notoginseng saponins (R1 and Rg1) in Yunnan Baiyao can reduce the levels of IL-6 and TNF-α by inhibiting the NF-κB pathway (animal experiments showed that inflammatory factors decreased by 40%). Dragon's blood extract (lonhemin B) in Qili Powder can promote platelet aggregation and accelerate blood coagulation (shortening the clotting time by 25%in vitro) [28].

6.1.2 Emerging therapies

Low-intensity pulsed ultrasound (LIPUS): Zhou S can stimulate cell membrane calcium channels through mechanical stress and up-regulate the expression of VEGF. Clinical trials show that LIPUS joint RICE in treatment of acute muscle strain, shorten the repair time to 7 days (the control group was 14 days) [29].

Kinesio taping: Kinesio taping is made of soft and breathable pure cotton cloth with water rippling acrylate low sensitivity glue. It does not contain latex and drug, has strong extensibility, and can reach 120%~140% of the original length, and exerts certain pressure [30, 31, 32] on the skin. It can enhance the contraction ability of the damaged muscle, reduce the pain caused by muscle overextension, reduce the occurrence of muscle fatigue and spasm; It can improve local blood and lymph

circulation, relieve local edema and inflammation; Increase the range of motion of joints; It can stimulate skin and muscle, correct the abnormal arrangement of

subcutaneous tissue, and has analgesic effect [30, 31, 33]. A large number of studies have confirmed that kinesio tape can increase pain threshold [34-36] and improve muscle strength.

**6.2 Treatment of Chronic Injuries**

6.2.1 Comprehensive treatment of TCM

Oral administration of Taohong Siwu Decoction promoted blood circulation and removed blood stasis. Taohong Siwu Decoction (peach kernel, safflower, Angelica sinensis, Chuangxiong) inhibited collagen deposition by down-regulating the TGF-β1/Smad3 pathway (Western blot showed that the phosphorylation level of Smad3 was reduced by 50%, $P<0.01$) [37]. Clinical studies showed that after 8 weeks of treatment, the patient's pain VAS score of periarthritis of shoulder decreased from 7.2 to 3.1 ($P<0.05$), and ultrasound showed a 20% [38] reduction in tendon thickness. Acupuncture (at ashi point and Weizhong point) improved local qi and blood. Electroacupuncture at Zusanli (ST 36) and ashi point can promote muscle fiber regeneration and inhibit fibrosis [39] in rabbits with acute blunt force injury. A multicenter study showed that acupuncture combined with tuina therapy reduced the recurrence rate of chronic low back pain to 15% at 6 months (compared with 45% in the control group) [40]. A transdermal topical preparation of Chinese medicine (containing frankincense and myrrh) reduced IL-6 and TNF-α levels [41].

6.2.2 Biomaterials and Tissue Engineering

Biomaterials Application: Professor Dai Fei Elmer Ker's team at the Chinese University of Hong Kong has developed a visible light-cross-linked polythiamine ester (PHT) hydrogel, which can be prepared by prepolymer resin in 30 minutes through a two-step reaction and realize 3D printing of personalized scaffolds. The material can be loaded with fibroblast growth factor 2 (FGF-2) and transforming growth factor β3 (TGF-β3) to achieve 1cm tendon regeneration in the rabbit rotator cuff defect model. The mechanical strength of the newly formed tendon recovered to 83% [42] of the healthy side at 8 weeks after operation. Collagen scaffold combined with MSCs transplantation promoted tendon regeneration, combined with electrical stimulation to enhance cell directional migration. Dodel M transplanted umbilical cord mesenchymal stem cells (UC-MSCs) loaded with type III collagen scaffold into the rabbit Achilles tendon injury model. After 12 weeks, collagen was orderly arranged, and tensile strength returned to 85% of normal level (control group: only 50%) [43].

WenMinShui gel delivery system: (carrying tanshinone WenMinShui gel can realize sustained release, targeted, inhibit fibrosis Zheng Juan by chitosan/beta - such as glycerol phosphate WenMinShui gel load tanshinone II A, slow-release can achieve 72 hours. The results showed that the IC50 of the drug was 12 μM, which was 60% [45] lower than that of the free drug. Zhang X et al. showed that the drug loading efficiency of 3D-printed PHT hydrogel was 85%, which has important potential [46] for the treatment of rotator cuff injury and other soft tissue injuries.

4D printed biomimetic scaffolds: Shape-memory polymer-based 4D printed scaffolds can respond to temperature or pH changes and dynamically simulate the mechanical properties of soft tissue. For example, polycaprolactone (PCL) scaffolds self-fold into helical structures at body temperature, promoting the orientation of cells. Animal experiments have shown that: In the rabbit Achilles tendon defect model, the tensile strength of the newly formed tendon reached 90% of that of the normal group (60% of the control group) at 8 weeks after operation, and the histological score showed that the collagen was arranged in an orderly manner (Sharir score 8.2 vs 5.4, $P<0.05$) [47].

Outside secrete targeted therapy: mesenchymal stem cells (MSCs) secreted by secrete carry miR - 29 b and miR - 21, by inhibiting the TGF - beta 1 and PTEN pathways synergy anti fibrosis. A clinical trial (NCT04519684) showed that local injection of exosomes in the treatment of chronic Achilles tendinitis reduced the pain score (VAS) by 60% [48].

Bioactive glass (BG) and its derivatives: BG the release of inorganic ions in a certain extent, have anti-inflammatory, antibacterial and hemostatic effect, helps to bone and soft tissue repair [49-53]. BBG hydrogel can enhance angiogenesis by releasing antibacterial ions $Cu^{2+}$, restoring HIF-1 α pathway, and regulating inflammation by reducing the expression of inflammation-related factors, increasing the secretion of anti-inflammatory cytokines, and promoting the rapid transformation of macrophages into M2 type, thereby promoting diabetic wound healing [54].

6.2.3 Physical and molecular targeted therapy

Extracorporeal shock wave (ESWT): Rompe JD induces the formation of microporesin the cell membrane by mechanical stress and promotes the release of growth factors. Meta-analysis showed that the total effective rate of ESWT in the treatment of chronic Achilles tendinitis was 82% (RR=1.53, 95%CI 1.28-1.83) [55].

TGF-β inhibitors: Davies M R et al. used the small molecule inhibitor SB-431542 to block TβR I kinase activity in preclinical studies showing a 55% [56] reduction in collagen area in a mouse model of tendon fibrosis.

## 7. Conclusions

The repair mechanism and treatment strategy for acute and chronic soft tissue injuries are shifting from single anti-inflammation to multi-target regulation. The collaborative innovation of integrated traditional Chinese and Western medicine and biomaterial technology provides new ideas for solving fibrosis and scar formation, and is expected to break through the bottleneck of fibrosis and scar formation. In the future, it is necessary to strengthen interdisciplinary cooperation and promote the transformation of basic research results into clinical practice. Finally achieve the ultimate goal of “precise repair” and “functional reconstruction”.

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