Management of soft tissue injury

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Abstract

Soft tissue injury is an acute connective tissue injury that may involve muscle, ligament, tendon, capsular structures and/or cartilaginous structures. Knowing relevant pathophysiology of a healing process is important in developing an appropriate treatment. In general, soft tissue injuries can be categorized into three degrees, depending on the severity of injury. The first degree is a mild stretch or minor tear of soft tissue with mild hemorrhage. The second degree is a moderate tear with some restrictions of movement. The most severe damage, the third degree, is an excessive stretch of soft tissue, causing a complete tear of the injured structure. After injury, the healing process is classified into three continuous, overlapping phases. They are inflammation, proliferation, and remodeling phases. Treatment is aimed to aid recovery and get the patients back to their normal activities as soon as possible without risks of re-rupture and chronic musculoskeletal impairments. The inflammatory phase comprises of extremely complex vascular and cellular responses that take three to five days to complete. The aims of treatment for this phase are to minimize inflammation and to provide optimal healing conditions. These aims can be achieved by applying “PRICE”: protection, rest, ice, compression, and elevation, immediately after injury. Once inflammation has ceased the development and growth of new blood vessels and granulation tissue occur in the proliferation phase. Generally, this phase lasts two to four weeks. The aim of treatment for this phase is to provide optimal environment for a new tissue growth and orientation. Early mobilization is recommended to provide mechanical load on regenerating tissue. Mechanical load causes cellular adaptation to external stress; thus, stimulates new fiber regeneration and orientation. In remodeling phase, the weak type III collagen is replaced by the strong type I collagen. The aim of treatment for this phase is directed towards quick and complete return to normal activities. Stretching and strengthening exercises within a limited degree of pain, are encouraged.

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With proper management for each stage of soft tissue healing, the risks of re-injury, time offf work, and costs of treatment will be reduced.

Keywords : soft tissue injury, RICE, treatment, therapeutic exercise, healing process

บทคัดย่อ
การบาดเจ็บของเนื้อเยื่อหมายถึงการบาดเจ็บของกล้ามเนื้อ เอ็นยึดกล้ามเนื้อ เอ็นยึดข้อ กระดูกอ่อน รอบข้อ หรือกระดูกอ่อน การเข้าใจสภาพของการซ่อมแซมเนื้อเยื่อจะนำไปสู่การวางแผนการรักษาที่ถูกต้อง การบาดเจ็บของกล้ามเนื้อสามารถแบ่งความรุนแรงออกได้ 3 ระดับคือ การบาดเจ็บเล็กน้อย มีเนื้อเยื่อฉีกขาดและเลือดไหลเล็กน้อย การบาดเจ็บระดับปานกลาง มีการฉีกขาดของกล้ามเนื้อมากกว่ากลุ่มแรก แต่การฉีกขาดไม่สมบูรณ์ ทำให้มีการจำกัดการเคลื่อนไหวเป็นบางส่วน การบาดเจ็บของเนื้อเยื่อข้อรุนแรง จะมีการฉีกขาดของเนื้อเยื่ออย่างสมบูรณ์ ทำให้ไม่สามารถเคลื่อนไหวได้ที่เข้าล่างจากการบาดเจ็บ เนื้อเยื่อมีการซ่อมแซมตัวเองได้อย่างต่อเนื่อง สามารถแบ่งออกได้เป็น 3 ระยะคือ ระยะอักเสบ เป็นระยะเวลากี่ที่มีการตอบสนองของระบบหลอดเลือดและระดับเซลล์ เพื่อการจัดเนื้อเยื่อที่ตายแล้ว และเตรียมความพร้อมในการสร้างเนื้อเยื่อใหม่ ระยะอักเสบนี้ กินเวลาประมาณ 3-5 วัน การรักษาในระยะนี้ มีวัตถุประสงค์เพื่อให้เกิดการอักเสบเนื้อเยื่อที่สุด และเกิดสภาพแวดล้อมที่เหมาะสมในการซ่อมแซมตนเอง โดยการใช้หลัก “PRICE” คือการพยุงส่วนที่ได้รับบาดเจ็บ (protection) พัก (rest) ใช้ความเย็นประชัน (ice) การพันผ่ายืด (compression) และการยกส่วนที่ได้รับบาดเจ็บให้สูงกว่าหัวใจ (elevation) หลังจากการอักเสบลดลง จะมีการสร้างเซลล์ใหม่เพื่อทดแทนเนื้อเยื่อที่ได้รับบาดเจ็บ ในช่วงนี้ เรียกว่าช่วงการเพิ่มจำนวนเซลล์ใหม่ๆยาวราวัครี อย่างไรก็ตาม เซลล์ใหม่มีความอ่อนแอ และยังไม่สามารถจัดเรียงตัวเป็นระเบียบได้ ระยะนี้ กินเวลา 2-4 สัปดาห์ ระยะนี้แสดงถึงการรักษาในระยะนี้คือ สร้างเซลล์ใหม่และเซลล์เสื่อมซึ่งมีการจัดเรียงตัวที่ดีขึ้น โดยการให้ผู้ป่วยเคลื่อนไหวส่วนที่ได้รับบาดเจ็บให้เร็วที่สุด หลังจากการพักโดยไม่มีการเคลื่อนไหวระยะแรกนี้ การเคลื่อนไหวในแนวเชิงเป็นการเพิ่มความแข็งแรงให้กับเนื้อเยื่อ ซึ่งจะส่งผลในการกระตุ้นการสร้างเซลล์ใหม่และช่วยให้การจัดเรียงตัวของเซลล์ใหม่ขึ้น ในระยะสุดท้ายของการซ่อมแซมตนเอง คือระยะการจัดเรียงตัวตามแบบของเนื้อเยื่อเดิม การรักษาในระยะนี้มีวัตถุประสงค์เพื่อให้เนื้อเยื่อที่ได้รับบาดเจ็บสามารถกลับไปทำหน้าที่ได้อย่างสมบูรณ์ ผู้ป่วยในระยะนี้สามารถออกกำลังกายเพื่อมิติกล้ามเนื้อและเพิ่มความแข็งแรงของกล้ามเนื้อให้ในช่วงที่ไม่มีอาการเจ็บปวด ทำให้ผู้ป่วยได้รับการรักษาที่เหมาะสมกับอายุ สภาพในแต่ละช่วงของการบาดเจ็บ จะทำให้เนื้อเยื่อมีการฟื้นตัวอย่างสมบูรณ์ ลดอัตราเสี่ยงต่อการเกิดการบาดเจ็บข้า ลดระยะเวลาการขาดงาน รวมถึงลดค่าใช้จ่ายในการรักษาพยาบาล

คำสำคัญ : การบาดเจ็บที่เนื้อเยื่อ การรักษาเนื้อผิว หลักการ PRICE การออกกำลังกายเพื่อการรักษากระบวนการซ่อมแซมเนื้อเยื่อ
1. Introduction

A soft tissue injury is a damage of the soft tissue of the body that may involve muscle, ligament, tendon, capsular structures and/or cartilaginous structures. Soft tissue injuries may be classified as direct trauma, which results from direct excessive force such as contacted sports or a fall; other injuries are indirect trauma which results from overloading or chronic overuse such as repetitive movements during work. It is recognized that many soft tissue injuries may result in some degrees of permanent impairment and leave their host with some permanent pains, restrictions, and loss of function (Liu and Nguyen 1999; Thelin, Holmberg et al. 2006).

The aims of treatment for soft tissue injury are to aid recovery and get the patient back to work or their sporting activity as soon as possible (Sexton. 2002). As a physical therapist, I will add the aim to “prevent re-injury and chronic musculoskeletal impairments”. Knowing about healing process is important in developing a safe and effective therapeutic exercise program. To ascertain what constitutes “the best treatment”, an understanding of the relevant pathophysiology is required.

2. Degrees of severity of soft tissue injury

The word “injury” is often unclear whether the structural incompetence being treated on the basis overload from cumulative trauma, or of a single traumatic event during an unguarded movement (Mooney and Brigham. 2003). The rate of repair and the potential for spontaneous healing are often difficult to expect when a patient is initially evaluated. In general, soft tissue injuries can be categorized into three grades or degrees of severity of injury (Jarvinen, et al. 2000 ; Ardevol, et al. 2002 ; Hougulum and Perrin. 2005) based on the clinical impairment they bring about.

2.1 First degree (mild)

Mild stretch of ligaments or capsular structures, or over-stretch or direct blow to muscle causes minor tearing of ligament fibres (<20 percent) with mild hemorrhage (Sexton. 2002). There is minimal swelling and bruising, but mild pain is felt at the end of range of movement or on stretch or contraction of muscle (Kerr.et al. 2002). There is no joint instability, minimal muscle spasm and no loss of function.

2.2 Second degree (moderate)

The result of moderate stretch of ligament or capsular structures, or excessive stretch or direct blow to muscles causes partial tear of some fibres (20-80
percent) with moderate hemorrhage and reduced active motion (Sexton. 2002). There is moderate swelling and bruising, with moderate pain felt on any movement which interferes with the ability of the muscle to contract or lengthen. There may be some joint instability with ligament/capsular injuries. Moderate muscle spasm may result as a reflex response to both ligament/capsular injuries and muscle injuries (Kannus, et al. 2003). Due to the tearing of some fibres, there is a decrease in the tensile strength of ligament/capsule or a decrease in the contractile strength of muscle, both of which cause partial loss of function (Kannus, et al. 2003).

2.3 Third degree (severe)

The result of a severe overstretch of ligament, or excessive stretch or direct blow of muscle causes a complete tear of the injured structure (80-100 percent) (Sexton. 2002). There is significant swelling and bruising with severe pain even at rest which significantly interferes with function (Houglum and Perrin. 2005). Ligament injuries result in gross instability and significant decrease in tensile strength, with muscle injuries causing severe muscle spasm and ‘splinting’, while the injured muscle is incapable of producing force (Kannus, et al. 2003). Function is severely impaired.

3. Healing phases

Healing is a continuum of changing events. To understand and clarify this process, researchers and clinicians divide the events into three different phases (Kerr, et al. 2002; Kannus, et al. 2003; Houglum and Perrin. 2005). Keep in mind that the process is continuous, without clear-cut delineations. The body merely continues the process until the end is reached. The three phases designated by researchers and clinicians are inflammation, proliferation or fibroplastic, and remodeling or maturation phases.

3.1 The inflammatory phase

When an injury occurs, the body attempts to stabilize the injured site by rushing chemicals and cells into the area. These extremely complex processes take three to five days to complete (Kerr, et al. 2002; Kannus, et al. 2003; Houglum and Perrin. 2005). Inflammation often has negative connotation, but in this case, it is an important and necessary step in the healing process. Without inflammation, the body would be unable to complete the healing process. If inflammation did not occur, proliferation, maturation, and final resolution would not take place. The wound would remain unhealed. Inflammation becomes deleterious when it is prolonged, extending beyond the normal healing time. A simplified version of this process is summarized in Figure 1.
Figure 1 Immediate injury response.

Immediately after injury, local vasoconstriction occurs in order to stop bleeding, followed quickly by vasodilation. The vasodilation causes vascular and cellular responses. The vascular response causes tissue edema due to an increased in blood volume and exudate. The cellular response causes recruitment of phagocytes and macrophages to clear the debris from the injury site. Macrophages also help to provide growth factors and mediators that are necessary for the proliferation phase. In addition, the cellular response helps stop bleeding and provides tensile strength by attracting fibrin, fibronectin, and collagen.
When an injury occurs, blood and lymph vessel walls suffer damage. The immediate local vasoconstriction occurs in the small vessels in order to stop bleeding; then vasoconstriction is followed quickly by vasodilation (Kannus, et al. 2003).

The vasodilation causes the release of blood and blood products into the injured site, including blood platelets and serum proteins. Upon tissue damage and ruptured blood vessels, intrinsic cells (such as endothelin and epitenin cells) will trigger a coagulation cascade and form a clot around the injured area (Molloy, et al. 2003). The clots contain cells and platelets that will release a variety of molecules, most notable growth factors (such as platelet-derived growth factor (PDGF), transforming growth factor (TGF), and insulin-like growth factor (IGF-I and IGF-II), causing a local inflammation (Molloy, et al. 2003). As these products accumulate in the injury, chemicals are released, and other cells are attracted into the area. These substances are important in the subsequent healing process.

Within the first few hours of injury, the body attempts to remove debris from the site. The early process runs by neutrophils, or polymorphonuclear leukocytes (PMNs) and granular leukocyte family include eosinophils and basophils. The late phase, within 24-48 hours, monocytes and macrophages, then, replace the neutrophils. Both PMNs and the macrophages act as phagocytes to remove debris and dead tissue from the area (Molloy, et al. 2003).

Debridement (removal of debris) is necessary for healing to continue. Before the subsequent phases can occur, the injury site must be cleared of excess fluid and other waste materials that have accumulated. For this reason alone, macrophages are vital to the healing process. Macrophages also release growth factors and may trigger the termination of tissue growth when the healing process is complete (Molloy, et al. 2003).

3.1.1 Signs of inflammation

Many complex events go on during the inflammation phase. The injured area undergoes intense activity during this time. The cardinal signs of inflammation are localized redness, edema, pain, increased temperature, and loss of normal function.

3.1.1 Redness, increased local temperature, and edema are caused by the leakage of fluid, cells, and chemicals in the area because of the local vasodilation and increased vascular permeability. Inside the dilated capillaries, the rate of blood flow slows, and by four hours after injury, white blood cells are beginning to pass through the vessel walls. Histamine and other released hormones and vasodilation cause redness (Houglum and Perrin. 2005). The
increase in local cellular and chemical activity increases local temperature (Kerr, et al. 2002).

3.1.2 Edema is the result of increased substances in the area and blockage of lymph vessels whose normal responsibility is drainage (Kerr, et al. 2002). The presence of protein in the inflammatory exudates raises the osmotic pressure of the tissue fluid in the damaged area, and consequently, draws more fluid into the injured tissues. These inflammatory exudates are necessary for the healing process because they contain fibrinogen, which forms fibrin that is a necessary part of the body’s defense mechanism against infection. However, an excessive amount of inflammatory exudates results in extreme formation of fibrin that may become scar tissue (Houglum and Perrin 2005). Inflammatory swelling starts to develop approximately two hours after injury and may continue for up to four days (Kannus, et al. 2003).

3.1.3 The chemical substances that are released at the site, such as histamine, prostaglandins, and bradykinin make the local nerve endings hypersensitive and irritable, causing pain (Kerr, et al. 2002). As swelling begins to develop, pain will also result from increased tissue pressure (Kerr, et al. 2002).

3.1.4 Direct damage to tissues also prevents them from functioning normally. The goal of treatment in the inflammatory phase is to allow minimal inflammation and encourages healing process. Initial first aids including rest, ice, compression, and elevation are generally recommended immediately after injury.

3.2 Proliferation phase

There is an overlap of phases as the injury site heals. As stated earlier, there is no clear-cut delineation between one phase and another. Rather, as the body steadily accomplishes the tasks in one phase, the next phase evolves (Kannus, 2000; Kannus, et al. 2003; Houglum and Perrin 2005; Jarvinen, et al. 2005). The proliferation process is demonstrated in Figure 2.

Once debris has been moving from the damaged area, the development and growth of new blood vessels and granulation tissue occur. A significant increased in the number of fibroblasts and extra cellular matrix components (proteoglycans, matrix glycoprotein, and epithelial cell mitosis) along with a decrease to minimal or nonexistent levels of PMNs are the hallmarks of the proliferation phase (Kannus, et al. 2003; Houglum and Perrin 2005). These fibroblasts are responsible for synthesizing the new extracellular matrix, consisting largely of collagens and glycoaminoglycan (Molloy,
et al. 2003). These extracellular matrixes are required for ultimate scar tissue formation and maturation. Angiogenesis occurs at a rapid rate during this phase to support scar tissue formation and the subsequent healing process. The duration of the proliferation phase depends on factors such as the size of the injury and the tissue type involved. Generally, this phase is thought to last two to four weeks.

Toward the end of the proliferation phase, the original fibrin clot becomes gradually replaced by a more permanent structure called granulation tissue (Kannus, et al. 2003). Granulation tissue is the combination of the matrix and newly formed capillary buds. Endothelial cells, the most important cell in the formation of these capillaries, contain a plasminogen activator. The plasminogen activator breaks down and removes the fibrin network that was formed during the inflammation phase so that lymphatic flow can be restored to remove local excess fluid (Houglum and Perrin, 2005). Then fibrous elements of the matrix (collagen, reticulin, and elastin) are formed. Collagen fibers provide a supporting framework of tissues and cells while elastin fibers help in distributing stress to maintain the resilience adapted to local tissue requirements (Ushiki. 2002).

In early 48 to 72 hours after injury, collagen type III is produced. Although collagen type III is relatively weak, it provides the wound’s primary tensile strength in the early stages of healing. It is replaced by type I collagen, a stronger and more durable collagen by day 10 (Jarvinen, et al. 2005). However, a relatively longer time is still needed until the tensile strength of the tissue is completely restored to preinjury level (Jarvinen, et al. 2005).
3.3 Remodeling phase

In the phase of remodeling, the proteoglycan-water, cell, and capillary content of the healing tissue gradually decreases (Kannus, et al. 2003). The maturation of the wound’s collagen structure and arrangement are the primary activities during the remodeling phase, hence its name. Collagen type III is replaced by collagen type I. Collagen type I starts to reorganize itself into parallel fashion and forms the greatest number of cross-links and arrangement are the primary activities during the remodeling phase, hence its name. Collagen type III is replaced by collagen type I. Collagen type I starts to reorganize itself into parallel fashion and forms the greatest number of cross-links and arrangement are the primary activities during the remodeling phase, hence its name. Collagen type III is replaced by collagen type I. Collagen type I starts to reorganize itself into parallel fashion and forms the greatest number of cross-links and arrangement are the primary activities during the remodeling phase, hence its name. Collagen type III is replaced by collagen type I. Collagen type I starts to reorganize itself into parallel fashion and forms the greatest number of cross-links and arrangement are the primary activities during the remodeling phase, hence its name. Collagen type III is replaced by collagen type I. Collagen type I starts to reorganize itself into parallel fashion and forms the greatest number of cross-links and
thereby possesses optimal strength. As collagen is converted to type I, it becomes more insoluble and less resistant to destruction. As fluid is reduced in the area, the collagen fibers can produce more cross-links with each other, further strengthening the scar’s structure (Houglum and Perrin. 2005). This collagen cross-linking becomes the primary source of the scar’s tensile strength.

About six to eight weeks after the injury, the new collagen fibers begin to withstand stress rather well (Kannus, Parkkari et al. 2003). This activity is generally thought to be about 12 months long, but may range from 6 months to 18 months (Kannus. 2000).

In remodeling phase, several visible changes can be observed. The scar’s red colour changes to white and eventually into more normal skin tones (Houglum and Perrin. 2005). Swelling is diminished and wound sensitivity is also reduced.

Figure 3 Remodeling phase.

The extracellular cells and capillary used for the proliferation phase have decreased. Collagen type I provides the main tensile strength for the injury site by rearrangement itself and cross-link formation.
4. Treatment of soft tissue injury

As stated earlier that the aim of the treatment for soft tissue injury are to enhance recovery and get the patient back to their normal activities as soon as possible (Sexton. 2002), physical therapists play an important role in this statement. Without proper diagnosis and treatment, these injuries may become chronic and may cause a serious risk for recurrence of soft tissue damage. Sports health professionals have put lots of interest and efforts in discovering ways for faster and better healing of the injury, and in this respect, the question is “What is the best management programme for each stage of healing?”

4.1 The inflammatory phase

The first phase is the acute inflammatory phase, from zero to seven days. During this phase there are ischemia, metabolic disturbance, and cell membrane damage causing inflammation, which is essential for healing. Treatment in this phase is aimed at minimizing hemorrhage, swelling, inflammation, cellular metabolism and pain, and to provide optimal healing conditions (Kerr, et al. 2002 ; Sexton. 2002). Prolonged inflammation may cause excessive scarring and lead to mobility and strength impairments.

Current practice as mentioned earliar, recommends protection, rest, ice, compression, and Elevation (PRICE) during the first 72 hours (Kerr, et al. 2002 ; Sexton. 2002 ; Kannus, et al. 2003 ; Jarvinen, et al. 2005). Protection is required to protect the injured tissues from undue stress which may disrupt and delay the healing process (Kerr, et al. 2002). Several authors stated that it is necessary to immobilize the injured tissue for a substantial period of time in order to increase the amount of intramuscular connective tissue and enhance type III collagen and fibronectin formation (Kannus, et al. 2003 ; Jarvinen, et al. 2005). Protection may be applied by taping, sprinting, or bandaging.

The benefits of RICE application immediately after injury has been extensively reviewed elsewhere (Jarvinen, et al. 2000 ; Kannus. 2000 ; Kerr. et al. 2002 ; Sexton. 2002 ; Kannus, et al. 2003 ; Jarvinen, et al. 2005). Briefly, rest is required to reduce the metabolic demands and blood flow of the injured area. The patient must avoid any activity involving the injured area that may compromise the healing process but need to continue general activity. Ice is used to reduce the temperature of the tissues at the site of injury and consequently reducing metabolic demand, inducing vasoconstriction and limiting the bleeding (Airaksinen, et al. 2003). Ice may reduce pain by increasing threshold levels of free
nerve endings and at synapse, and by increasing nerve conduction latency to promote analgesia (Kerr, et al. 2002). Shorter, intermittent ice applications are most effective in reducing tissue temperature and enhancing the analgesic effect (Bleakley, et al. 2006). Compression is applied to reduce the swelling and consequently, reduce pain and allow greater range of motion (Pollard and Cronin. 2005). Compression also helps to control the amount of fibrin into the injured area (and ultimately the production of scar tissue) and control the osmotic pressure of the tissue fluid in the injured area (Pollard and Cronin. 2005). Elevation of the injured part lowers the pressure in local blood vessels and helps to limit the bleeding. It will also increase drainage of the inflammatory exudate through the lymph vessels, thus reducing and limiting edema and its resultant complications (Kerr, et al. 2002).

4.2 Proliferation phase

The proliferation phase takes approximately three to 21 days (Sexton. 2002). In this phase, there is formation of pathologic, dense connective scar tissue. Type III collagen (weak collagen) is produced within two to three days after injury to provide the wound initial tensile strength. These weak collagens are replaced by type I collagen by day 10. At the beginning of proliferation phase, immobilization is recommended to promote fibroblast differentiation and invasion in the injured area and consequently, promote the production of collagen fibers (Jarvinen, et al. 2005). If there is no immobilization period, a larger connective tissue scar ensues, and the initial penetration of muscle fibers through the connective tissue scar appears to be impaired in comparison to immobilized tissue (Nash, et al. 2004). In addition, reruptures at the site of the original tissue trauma are common. Immobilization appears to provide the new granulation tissue with the needed tensile strength to withstand the forces created by muscle contraction.

Recent researchers have agreed that immobilization period should be short duration followed by controlled and progressive mobilization (Kannus, et al. 2003; Nash, et al. 2004; Jarvinen, et al. 2005). The duration of immobilization should be considered by the severity of pain and the extent of injury (Kerr, et al. 2002). A moderate (second degree) injury requires three to five days immobilization. Mild (first degree) injuries may require a shorter period and severe (third degree) injuries require longer time.

Deciding upon the right time to start active mobilization after soft tissue injury can be difficult for the physical thera-
pist. This decision can, however, be based on two simple and inexpensive measures: the ability to stretch the injured muscle as much as the healthy contralateral muscle, and the pain-free use of injured muscle in basic movements (Jarvinen, et al. 2000). When this critical point is reached, the patient should be encouraged to start active and progressive mobilization.

Jarvinen, et al (2005) had recommended that a short period of immobilization would be beneficial, but it should be limited only to the first three to seven days after injury. Controlled mobilization is then recommended to enhance collagen formation. Controlled mobilization can be performed by moving the less injured part of soft tissues earlier to induce some mechanical stress to the injured area. Changes in mechanical loads produce alteration in cellular biochemistry and, in turn, feedback to remodel cell and tissue structures so as to more effectively bear and respond to applied stress (Ingber. 2005). Early mobilization, therefore, induces more rapid and intensive capillaries ingrowth into the injured area, better regeneration of muscle fibers, and more parallel orientation of the regenerating myofibrils in comparison to immobilization (Jarvinen, et al. 2005). They also can prevent tissue atrophy as well as substantial production of intramuscular connective tissue caused by immobilization. Functionally, early mobilization was reported to be beneficial in earlier return to work (Crawford, et al. 2004), decreased pain, swelling, and stiffness, and less mobility impairments (Nash, et al. 2004).

Controlled muscle stretching and joint movement is claimed to improve the orientation of collagen fibers and to regain mechanical strength. Mechanical stretching can modulate proliferation of human tendon fibroblasts and increase the cellular production of collagen type I (Yang, et al. 2004). In recent animal research, passive stretching exercises showed a strong antifibrotic effects and improved muscle regeneration and strength when compared with the no-exercise group (Hwang, et al. 2006). The best time to begin stretching exercise was 14 days after soft tissue injury for antifibrosis and muscle regeneration. This finding is probably well applicable to soft tissue injuries of humans, although the time frame of healing can be different.

4.3 Remodeling phase

In remodeling phase, type III collagen is replaced by type I collagen. This activity lasts six to 12 months. However, about six to eight weeks after the injury, new collagen fibers begin to withstand the tensile stress well and there are no pathophysiologic reasons to continue protection
any longer. The rehabilitation is thus directed towards quick and complete return to exercise and sports. Physical therapy can promote this phase by improving local circulation and proprioception (Verhagen, van der Beek et al. 2004), inhibiting pain and strengthening muscle-tendon units (Myer, et al. 2004).

A carefully structured rehabilitation program accelerates recovery. All physical rehabilitation activities should start with warming-up of the injured muscle, which reduces muscle viscoelasticity and relaxes muscle (Bishop. 2003). Stimulated muscle absorbs more energy during physical exercise and, thus, the risk of re-rupture is reduced (Stewart, et al. 2006). Stretching is another important pre-exercise strategy which is aimed to distend the scar tissue while it is still elastic (Weerapong, et al. 2004). These pre-exercise activities would help minimize the risk of rerupture and chronic musculoskeletal impairments.

5. Conclusion

Soft tissue injury occurs not only in sports competition but in normal activities as well. Knowing relevant pathophysiology of the healing process is important in developing an appropriate treatment. After injury, the healing process is classified into three continuous, overlapping phases. They are inflammation, proliferation, and remodeling phases. In general, treatment is aimed to aid recovery and get the patients back to their normal activities as soon as possible without the risk of re-rupture and chronic musculoskeletal impairments. To be specific, in the inflammation phase, the aim of treatments are to minimize inflammation and to provide optimal healing conditions by applying “PRICE”; protection, rest, ice, compression, and elevation, immediately after injury. In the proliferation phase, the aim of treatment is to provide optimal environment for a new tissue growth and orientation. Currently, early mobilization is recommended to promote cellular adaptation to external stress, which stimulates new fiber regeneration and orientation. In the remodeling phase, the aim of treatment is directed towards quick and complete return to normal activities. Stretching and strengthening exercises, within a limited degree of pain, are encouraged. With proper management for each stage of soft tissue healing, the risks of re-injury, time off work, and costs of treatment will be reduced.
References


