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EARLY TREATMENT OF SOFT TISSUE INJURIES

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TRENDS IN MODERN MEDICINE – EARLY MANAGEMENT OF ACUTE SOFT TISSUE INJURIES IN SPORT

Wayne Derman

MBChB, BSc (Med) (Hons) Exercise Science, PhD, FACSM, FFIMS

Professor of Sports and Exercise Medicine
UCT/MRC Research Unit for Exercise Science and Sports Medicine
Department of Human Biology
Faculty of Health Sciences
University of Cape Town
Sports Science Institute of South Africa
Boundary Road
Newlands
Cape Town 7700
South Africa
Email: wayne.derman@uct.ac.za

Contact details:

Tel: +27 21 650 4560

Fax: +27 21 686 7530

Brief biography

Prof. Derman is a Sports Physician at the University of Cape Town, based at the Sports Science Institute of South Africa. He is a partner in a Sports and Exercise Medicine Practice, and is a member of the Scientific Commission of the International Federation of Sports Medicine (FIMS). He serves on the High Performance Commission of the South African Sports and Olympic Committee and has served as Chief Medical Officer to the South African Olympic Team to Sydney 2000 & Athens 2004 Olympic Games and as Medical Officer to the South African Paralympic Team to Beijing 2008. He is on the editorial board of the British Journal of Sports Medicine and has published extensively in the field of Clinical Sports and Exercise Medicine.

INTRODUCTION

Acute musculoskeletal injuries are common in rugby.[1] The most common region to be injured in rugby union is the lower limb (in particular the knee, thigh and ankle); in fact, ligament (knee and ankle) sprain injuries and ruptures as well as skeletal muscle (hamstring, calf, quadriceps and groin) strain injuries and ruptures are the most common types of injuries sustained in rugby players.[2-4] The "soft tissue" injuries are the most common injuries in rugby players and occur at a rate of between 69 and 218 injuries per 1000 playing hours.[1-4]

It is the duty of the field-side medical or paramedical response team to ensure that ideal early treatment is provided to the injured rugby player as this early treatment will affect the healing response and subsequent efficacy of rehabilitation and return to sport.

THE PHYSIOLOGICAL RESPONSE TO ACUTE SOFT TISSUE INJURY

During acute soft tissue injury, the biological tissues are subjected to abnormal or excessive forces characterised by heavy compressive forces such as a direct blow to the skeletal muscle or overstraining/ shearing forces to the soft tissue such as those experienced during sprinting or jumping. The healing of injured soft tissue has recently been reviewed,[5;6] and follows a constant and continuous pattern irrespective of the underlying mechanism. This consists of 3 overlapping and interwoven phases[5]:

Phase 1: This phase is split into two and characterised by the immediate reaction to trauma or tissue destruction phase, which lasts from 0-60 minutes, and the acute inflammatory phase, which lasts from 0-72 hours.

Phase 2: Repair and regeneration phase, which lasts between 2 and 42 days. This phase is characterised by phagocytosis of the necrotized tissue and regeneration of tissue fibres as well as the production of a connective tissue scar with capillary in-growth into the injured area.

Phase 3: The tissue remodelling phase, which lasts between 21 days and one year, and is characterised by contraction and reorganisation of the scar tissue and subsequent recovery of the functional capacity of the soft tissue.

The aim of this article is to review current management strategies in Phase 1 of injury (first 48 hours) from an evidence-based perspective, to evaluate the physiological rationale and mechanisms of action of these interventions, and to provide clinical guidelines for management based on the best available evidence, whilst optimal management techniques in the later phases of recovery will be covered in other articles published by BokSmart.

THE PATHO-BIOLOGY OF PHASE 1 OF SOFT TISSUE INJURY

When a contusion injury to soft tissue occurs (e.g. to muscle tissue), tissue rupture occurs at or adjacent to the site of impact. The excessive mechanical force extends across the individual myofibres, tearing the sarcoplasm of the muscle stumps and leaving a tissue gap. A contraction band rapidly occurs like a fire-door to limit the extent of the injury.[7] Thus, the propagation of the tissue necrosis (or tissue death) is halted. Simultaneously with the damage to the muscle fibres, the blood vessels supplying the muscle fibres are torn, leading to haemorrhage and ischaemia of the distal tissues. Through the haemorrhage, blood-borne inflammatory cells gain access to the site of injury and the inflammatory reaction ensues.[8] The inflammatory process is then amplified by the satellite cells and necrotized myofibres release various chemotaxic factors which enhance the extravasation of inflammatory cells into the area. The activated macrophages and fibroblasts produce additional growth factors, cytokines and chemokines for the circulation inflammatory cells which start to direct the repair process.[8;9] Initially, polymorph leukocytes are the most common cell found at the injury site but after 24 hours they are replaced by monocytes.[7;10] The monocytes are eventually transformed into macrophages that then actively engage in the phagocytosis and proteolysis of the necrotic material by the release of enzymes.[7;11] The phagocytosis of the necrotic tissue leaves an intact scaffold of cylinders of basal laminas inside which the satellite cells begin the formation of new myofibres.[5;7]

CLINICAL MANAGEMENT OF PHASE 1 OF SOFT TISSUE INJURY

The most important period of management is perhaps the initial 24-hour period after acute injury as bleeding and secondary tissue hypoxia should be minimised. The "RICE-D" principles of management of soft tissue injuries is widely proposed and clinically practiced on a global scale. The individual components of this management are:

R – Rest (immobilisation); I – Ice (cryotherapy); C – Compression; E – Elevation; and D – Drugs, usually referring to use of the anti-inflammatory agents. In other papers, within the BokSmart material, the "D" has been referred to "Diagnosis", which is also crucially important, but for the purpose of this paper, we will refer to the "D" under the heading "Drugs", as this part is generally poorly understood by players and coaches. The overall justification for the R,I,C & E components of this practice seem extremely practical due to the fact that all these components aim to minimise both the extent of the injury and bleeding. However, following an extensive review of the literature, it should be stressed that there is not one randomised controlled clinical trial to study the efficacy of the "RICE" principle in the treatment of acute soft tissue injury.[12;13] Yet there are studies which have reported efficacy of the individual components of the "RICE" intervention and these studies will be reported.

REST

Physiology and potential mechanisms for increasing healing of soft tissues

By "rest", what is meant may vary from removal of the player from the field to splinting of the injury to prevent further injury and reduce subsequent injury extension and further haemorrhage. Whilst there are no direct clinical studies on this component of management, the logic behind protecting the athlete and the injury in the acute phase seems sound. Not only is the bleeding minimised but the athlete is made more comfortable and pain is reduced.

Scientific evidence that rest improves soft tissue healing

There is some scientific evidence for rest, which comes from studies of the effects of immobilisation on skeletal muscle healing.[5;14] In a muscle injury model, by placing the injured extremity to rest immediately after the trauma, far retraction of the ruptured muscle stumps is prevented resulting in: reduction of a large gap within the muscle; reduced size of haematoma and reduction of the size of the connective tissue scar.[5;14]

Current clinical guidelines for "rest" in the treatment of soft tissue injuries in sport

It is therefore prudent that following acute injury, the injured athlete is removed from the danger of sustaining further injury; and that the injured tissue is immobilised and protected in an attempt to reduce bleeding. The use of crutches, a sling (for upper-limb injuries) and strapping/bracing are examples of this application. However, the duration of the immobilisation and rest should be limited to only until the scar has reached sufficient strength to bear the muscle contraction forces without the risk of re-rupture. Although not based on scientific study, most practical expert opinion suggests that the period of immobilisation should last from 1-3 days until the inflammatory phase is complete. Gradual mobilisation and graded rehabilitation should follow.[15]

ICE (CRYOTHERAPY)

This modality of management of acute soft tissue injury is perhaps the oldest and apparently the most simple to use in the initial phases of injury; however, most of the recommendations have been based on expert opinion following practical experience or from the results of animal studies or studies on healthy volunteers, rather than through rigorous scientific study in clinical populations. Furthermore, there is wide variation in the recommendations for cryotherapy.

Physiology and potential mechanisms for increasing healing of soft tissues

The proposed basis of cryotherapy application is that reduced tissue temperature should:[16]

- reduce bleeding in the immediate phase of management
- provide an analgesic or pain-relieving effect
- decrease tissue metabolism
- decrease muscle spasm
- minimise the inflammatory process
- decrease secondary hypoxic injury
- reduce cell debris
- reduce edema or swelling

Scientific evidence that ice therapy (cryotherapy) improves soft tissue healing

Although there is sound scientific evidence to suggest that ice/cryotherapy is effective in reducing the deep tissue temperature in both animal and human studies[17-19] following review of the clinical effects of cryotherapy,[12;20;21] it is clear that there is wide variation in the following parameters:

- the nature of injury (surgically "induced" injury vs trauma vs healthy volunteers)
- method of ice application (crushed ice in moist cloth or towel; commercial ice bags; instant ice packs; injury immersion; reusable frozen gel packs; cryo-cuff device; cold water or cooling sprays)
- duration of application (application time ranged from 0.3 hours to continuous application; total application time ranged from 0.3 to 336 hours)[12]
- number of treatment sessions per day (ranged from 1 to continuous treatment)[12]
- time of cryotherapy initiation (ranged from immediate post-surgical intervention to 3 days following injury)
- initial temperature of the ice
- depth of subcutaneous fat

Following detailed review of the effects of ice/cryotherapy in clinical practice, it was concluded that only 14 studies in the literature were of sufficient quality to be included in a scientific review.[12;20;21] The following were the main findings of the review:

- Ice/cryotherapy therapy alone is more effective than no form of cryotherapy following knee surgery[22] and soft tissue injury.[23]

- Intermittent 10-minute ice treatments are most effective at cooling injured animal tissue and healthy human tissue[20]. Furthermore, in a randomised controlled study, intermittent 10-minute ice applications (10 minutes of ice with a 10-minute room temperature period followed by a further 10-minute ice application; applied every two hours; for a total period of 72 hours post-injury) showed enhanced therapeutic effect (pain relief) compared to a standard 20-minute protocol.[24]

- However, continuous ice therapy has been shown to be superior to intermittent 20-minute applications over the first 3 days following surgery in humans.[25]

- There is evidence that ice therapy is more effective than heat or contrast therapy (alternating cold/heat) in reducing swelling after ankle sprain.[26]

- There is evidence that there is no further benefit in the addition of electrical stimulation to ice therapy after ankle sprain.[27]

- There is conflicting evidence with respect to studies of the addition of compression to the cryotherapy technique. Whilst the addition of compression in combination with ice has been shown to be superior compared to ice alone,[28] there is little difference in the effectiveness (using many recovery variables) of ice and compression compared to compression alone.[29-32]

Current clinical guidelines for ice therapy (cryotherapy) in the treatment of soft tissue injuries in sport

Whilst no individual study has rigorously compared the different modes, durations and frequencies of ice treatment or cryotherapy, various opinion (Level 4 evidence) has culminated in the present clinical guidelines for ice/cryotherapy application:

- Ice /cryotherapy should be used following soft tissue injury

- There are many different forms of ice/cryotherapy techniques and most seem effective. It is desirable to reduce the deep tissue temperature to between 10-15 degrees Celsius.

- It appears from the literature that intermittent 10-minute minimum – 20-minute maximum applications (10-20 minutes ice followed by 10 minutes at room temperature, followed by a further 10-20 minutes of ice application); applied every 2-4 hours initially and then gradually reducing the frequency over the next 24-48 hours, seems an effective and practical protocol. The longer ice/cryotherapy period should be used in patients with a greater amount of subcutaneous fat.

- Ice/cryotherapy should begin as soon after injury as possible

- There may be some benefit in combining compression together with ice application; however, further studies are required to address this issue.

- It is important to note that a number of adverse effects of ice/cryotherapy have been reported in the literature. These include skin burns, nerve damage including palsy, altered prorioception and reduced muscle power output.[33-35]

- Ice/cryotherapy is contraindicated in patients with Raynaud's disease, cold allergy or peripheral vascular disease.

COMPRESSION

Compression can be achieved through the use of firm bandages, elasticised bandages or a combination device, e.g. the cryocuff which provides both compression and cryotherapy. The hypothesis behind the use of compression is that the increased pressure induced by the bandage or device would reduce bleeding and thus swelling and inflammation would be minimised. There are however few studies which have studied compression in a sports injury model. Evidence exists with respect to studies of the addition of compression to the cryotherapy technique. Whilst the addition of compression in combination with ice has been shown to be superior compared to ice alone[28], there is little difference in the effectiveness (using many recovery variables) of ice and compression compared to compression alone.[29-32] Therefore compression seems to be valuable in the management of soft tissue injury.

However, no scientific-based guidelines exist with respect to the duration, intensity and frequency of the compression that should be applied. Therefore only practical guidelines exist.

- There appears to be benefit to using compression with respect to management of soft tissue injury
- The width of the compression area will vary according to the injured area
- If compression bandage is used, it should start distal to the area of injury with each layer overlapping the underlying layer and should extend to above the margin of the soft tissue injury
- The compression should be applied firmly but not so tightly as to cause pain or ischaemia

ELEVATION

Although scientific studies are lacking, elevation of the limb following acute injury is widely practiced. The hypothesis behind this practice includes a reduction in hydrostatic pressure and therefore a decrease in the accumulation of extracellular fluid. Thus the injured limb should be above the level of the heart (in the case of the upper limb) and the pelvis (in the case of the lower limb) for as long a time period as is practical during the initial 48 hours after injury.

DRUGS (PHARMACOLOGICAL AGENTS) – PAIN MANAGEMENT

Pharmacological agents are often used by clinicians, or in fact demanded by injured athletes, in an attempt to reduce pain, swelling and inflammation. Indeed, use of analgesics and anti-inflammatory agents is widespread following acute soft tissue injury in competitive contact sport[36-39]. It is probable that rates of use of these agents might be even higher in recreational athletes, especially as some of these agents are available for over-the-counter purchase. It is difficult to believe that the alarmingly high frequencies of use of these agents (in all sports) are for therapeutic reasons and therefore both athletes and medical staff must believe that use of these agents might extend some prophylactic benefit to the competing athlete. Furthermore, long-term use of these agents is also a concern. As these medications are not without significant side-effects, improved dissemination of knowledge regarding the use of the agents as well as guidelines for their judicious use in sports medicine should be directed at team physicians, general practitioners prescribing for recreational athletes, and athletes themselves.

Physiology and potential mechanisms for pain reduction

Pain is an extremely complex phenomenon. It is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage. Furthermore, pain is an individual, multifactorial experience influenced by culture, previous pain events, mood and ability to cope. Acute pain is defined as "pain of recent onset and probable limited duration. It usually has an identifiable temporal and causal relationship to injury"[40].

Through tissue injury, phospholipids are released from the cell membrane and are converted into arachidonic acid by the enzyme phospholipase A2. Arachidonic acid in turn is a substrate for the enzyme cyclo-oxygenase (COX), resulting in the production of various prostaglandins (PGs). This pathway and the substances that are produced are responsible for the pain and inflammation seen in sports injury. With respect to the COX enzyme, two isoforms have been established with different functions. These are COX-1 (constitutively present) and COX-2 (induced) (Table 1). The older NSAIDs (Non-Steroidal Anti-Inflammatory Drugs) inhibit both isoforms of the COX enzymes, whereas COX-2 specific inhibitors inhibit only the COX-2 isoform (COXIBs), with the possibility that selective inhibition of particularly the COX-2 isoform could reduce the side-effects of NSAIDs but still maintain the efficacy of these agents.

Table 1: Differences between the isoforms of the cyclo-oxygenase enzymes (COX-1 and COX-2)

COX-1 ISOFORM	*COX-2 ISOFORM*
Constitutive	Inducible
Mainly physiological effects	Release of inflammatory mediators
Sites: stomach, intestine, kidney, platelets	Sites: macrophages, synoviocytes, fibroblasts

As clinicians, we are taught to actively treat and minimise the patient's pain and to return them to pre-injury level of functioning as quickly as possible, without compromising tissue healing. Indeed, many athletically-inclined patients place significant pressure on their treating physician to get them "back up and running" as soon as is possible. In this endeavour, we use pharmacological agents to treat the patient's pain so that detraining may be minimised and rehabilitation can be initiated. These agents might include analgesics, topical analgesics, non-steroidal anti-inflammatory agents, topical anti-inflammatory agents and corticosteroids. These agents will be discussed in the setting of the different forms of injury and biological tissue that is injured.

Agents used to treat pain - Scientific evidence of efficacy and other effects of pharmacological agents used to treat pain

Analgesics

Analgesics are commonly used in the first line management of acute sports injury to reduce pain. Further use of the analgesics will depend on the intensity and duration of pain. Agents in this group include acetylsalicylic acid, paracetamol, codeine and tramadol, used either as single agents or in combination.

Acetylsalicylic acid

Acetylsalicylic acid at low doses (up to 300 mg) has both an analgesic and antipyretic effect but has an anti-inflammatory effect at higher doses. However, at the higher doses, there is increased incidence of gastrointestinal side-effects. As this agent inhibits platelet aggregation and may increase bleeding, it does not have a role in the management of acute sports injuries.

Paracetamol

Paracetamol has both analgesic and antipyretic effects, but does not inhibit the inflammatory response or clotting process. It is thus safe for use in acute sports injuries at up to 3-4 g/day. The incidence of adverse effects is comparable to placebo.[41]

Codeine

Codeine is a more potent analgesic from the narcotic group. It is usually used in combination with either acetylsalicylic acid or paracetamol.[42] Its use is reserved for more severe pain.

Tramadol

Tramadol is also an effective analgesic from the narcotic group. It is also an effective agent in neuropathic pain.[43] However, a recent study has suggested that tramadol has lower efficacy and a greater incidence of adverse events compared to a COXIB in the management of chronic lower-back pain.[44] Its use in sports medicine is reserved for more severe injury, when additional analgesia is required. Ongoing need for use of this agent requires reassessment of the injury.

Topical analgesics

The majority of these agents are skin counterirritants and contain a combination of substances including methyl salicylate, eucalyptus, menthol, capsicum and camphor. The active ingredients cause erythrema and blood vessel dilatation and stimulate the pain and temperature receptors. These agents can be used in addition to a warm-up and can be of some benefit for minor sprains and strains.[45] However, they should not be used in the acute phase of injury (first 24-48 hrs) or on broken skin as they can cause further irritation, blistering and contact dermatitis.

NSAIDS

Perhaps the most common agents used in the practice of sports medicine today are the non-steroidal anti-inflammatory agents. Indeed, for many clinicians these agents are the first line of use to decrease pain, swelling and the inflammatory response when treating a soft tissue injury.

One of the main functions of non-steroidal anti-inflammatory drugs (NSAIDs) is to reduce the production of the substances that cause the inflammatory response, and therefore decrease pain, swelling, and loss of function following an acute sports injury. However, there have always been significant side-effects associated with the use of NSAIDs, in particular upper gastro-intestinal side-effects and renal side-effects.[46] NSAIDs can possibly be linked to water retention and hyponatremia in marathoners, but further research on this area of sports medicine is warranted.

Traditional NSAIDs carry the potential for greater adverse GIT side-effects and their effects on healing of tissues remain relatively unknown. However, they are not associated with cardiovascular side-effects and are effective analgesic agents.

Topical NSAIDs

A number of NSAIDs are available in different formulations including creams, ointments, sprays, gels and patches. Agents delivered in these forms include, amongst others, diclofenac, flubiprofen, ketoprofen and indomethacin. A recent systematic review of randomised controlled trials concluded that topical NSAIDs are effective in relieving the pain associated with soft tissue injuries without causing serious adverse effects.[47-51] However, NSAID patches are not effective in prophylactic use to prevent delayed onset muscle soreness (DOMS) in trained athletes.[52]

THE CYCLO-OXYGENASE-2 INHIBITORS (COXIBS)

These newer agents were developed to reduce the adverse GIT effects of the traditional NSAIDs. Most studies on the efficacy of these drugs show that they are effective in decreasing pain, swelling and loss of function.[53] However, the majority of these studies have been conducted in the osteoarthritis or rheumatoid arthritis models.[53] There are few studies on acute sports injuries. However, in some studies it has been demonstrated that the COXIBs are as effective as the older, non-selective NSAIDs in the management of ankle sprains and shoulder injuries. Studies show these agents generally are effective at decreasing pain and allow a quicker return to activity and rehabilitation.[44;54-59]. However, effects of these agents on joint stability and on joint injuries other than the ankle and shoulder are unknown.

In general, the COXIBs are associated with fewer gastrointestinal and other side-effects compared to the non-selective NSAIDs, and there appears to be a sparing effect on the kidney, provided patients are not sodium depleted.[47] These are advantages over the "older" drugs. There has however been some recent concern that the COXIBs may be associated with increased risk of thrombosis (by inhibiting prostacyclin), and an increased blood pressure following administration. However, the higher cardiovascular risk of the COXIBs (leading to the withdrawal of rofecoxib) compared to the non-selective NSAIDs have been seen in relation to some COXIBs but not others.[53] The FDA however commented that the short-term use of NSAIDs and COXIBs does not appear to increase cardiovascular risk. There may also be drug interactions, such as with warfarin (rofecoxib), cytochrome p-450 inhibitors (celecoxib), and sulphur drugs (rofecoxib). These have to be taken into consideration when these drugs are prescribed.

The effects of the (non-selective) NSAIDs and COXIBs on the healing process of various musculoskeletal tissues including bone, skeletal muscle, ligament and tendon have to be considered:

Bone injury

Although the focus of this article is predominantly soft tissue injury, it is prudent to discuss bony injury at this point as the findings are relevant to the overall discussion. Whilst the efficacy of the NSAIDs in attenuation of the formation of myositis ossificans and ectopic bone formation has been shown, the effects of these agents on the healing process and on loosening of prostheses require further studies.[60] NSAIDs have been widely used in the management of fracture pain, and their inhibitory effects on the bone healing process have raised concerns.[60-62]Studies evaluating fracture healing in mice treated with NSAIDs or in mice lacking the COX-2 gene demonstrate that deficiency of COX-2 impairs bone healing.[63-65] Limited clinical data also support the notion that COX-2 agents delay bone healing.[66]However, this finding has not been replicated in all studies.[66]It is apparent that both the older NSAIDs and newer COXIBs negatively affect bone healing to some extent.[66;67]

Skeletal muscle injury

Using different skeletal muscle injury models in animal studies, the COXIBs have shown that they impair healing and regeneration, with reduced myofibroblast proliferation, and in some instances increased fibrosis.[68-70] There have been limited studies on the effect of the non-selective NSAIDs and COXIBs on healing in human athletes. One study, investigating the effects of diclofenac patches following blunt trauma, showed that the intervention was safe and effective in reducing pain.[49] However, most of the available studies have examined the effects of these agents on delayed onset muscle soreness (DOMS) following eccentric loading exercise. The majority of these studies have failed to show any benefit of the administration of the anti-inflammatory medication with respect to induced muscle pain.[52;71-75]

Indeed, preliminary work in our laboratory has indicated that early administration of meloxicam prior to induced eccentric load damage results in higher concentrations of creatine kinase compared to administration of the agent 48hrs after muscle damage is induced. These findings suggest that early treatment with meloxicam might increase the extent of muscle damage induced by eccentric load exercise.[75]

Ligament injury

Animal studies have demonstrated that ligaments from celecoxib treated rats could resist less force and were less stiff compared to ligaments from control animals.[76;77] To date there are no studies on humans.

Tendon injury

The effects of the COXIBs have been investigated in animal trials using an Achilles tendon and a rotator cuff, and patellar tendon, injury and repair model. Whilst improved tendon repair was reported in one study,[78] other studies reported adverse effects of both COXIBs and non-selective NSAIDs on tendon healing.[37;38;79]

A review of the above studies certainly suggests that the anti-inflammatory agents and indeed both the non-selective NSAIDs and the COXIBs have a significant ***negative*** effect on musculoskeletal tissue healing and this finding remains a subject of much debate. [72;80-82]This is particularly evident with respect to animal study models. Whilst animal studies are important precursors in initial evaluation of drug safety and efficacy, care should be taken in extrapolating the results in many of these laboratory-based studies to the clinical setting. Further clinical trials with respect to use of these agents in the athletic population are urgently needed. In particular, the timing of administration of the agent and relative dosing (in comparison to animal models) requires further study.

Current clinical guidelines for the use of analgesics and NSAIDs/COXIBs in the treatment of soft tissue injuries in sport: Current practice in the Sports Medicine Practice at the Sports Science Institute of South Africa

- As is evident from the above discussion, the inflammatory process seems to be an important part of the healing process in the musculoskeletal tissue in humans. We therefore use only analgesics in the first 48 hours following injury to allow the first part of the physiological healing process to occur. Examples of agents that are used for pain management in this phase are paracetamol or paracetamol plus codeine.
- Rest, ice, compression and elevation are important elements of the patient management in the first 48 hours following injury, as detailed in the discussion above.
- After 48 hours post-injury, if repeat assessment of the injury reveals clinical signs and symptoms of excessive inflammation (swelling and pain) we use an NSAID or COXIB for up to a limited period (five days) as these agents have shown to reduce pain and promote function following injury.
- If the athlete has a history of gastro-intestinal side effects or other side effect following non-selective NSAID use, paracetamol should be continued or a COXIB or COXIB plus proton pump inhibitor should be considered.
- Physiotherapy, including therapeutic ultrasound, followed by rehabilitation (as outlined in other BokSmart publications) form an essential part of treatment from 24 hours after injury.
- Generally, if the use of an NSAID, COXIB or analgesic is required for longer than a five-day period, the patient should be reassessed and the diagnosis revisited.
- NSAIDs and COXIBs should not be used prophylactically to prevent muscle soreness after exercise or to prevent pain during sport.
- There is evidence of efficacy of use of the NSAIDs in the following injuries: ligament sprains of the ankle, knee and shoulder joints; conditions where the pathological disorder is tissue entrapment or impingement of nerves and other structures due to soft tissue swelling, for example in the following conditions: carpal tunnel syndrome, Morton's neuroma, intervertebral disc prolapse, thoracic outlet syndrome, bursitis in rotator cuff disease, trochanteric bursitis and iliotibial band friction syndrome.
- There is no role for NSAIDs in the management of the chronic degenerative tendon conditions including Achilles tendinosis, as the pathology has been shown not to be inflammatory in origin. Furthermore, there is no evidence to support use of NSAIDs for long-term pain from sports injury without impingement.
- Many athletes with sports injury do not take sufficient time off their training to allow for complete tissue healing. They might in fact ingest these agents to facilitate early return to sport, which can put them at risk of further injury. Adequate time for recovery, physiotherapy and rehabilitation should be allowed before returning to sport.

INTERVENTIONS WHICH MAY DELAY THE HEALING PROCESS FOLLOWING ACUTE SOFT TISSUE INJURY

It is unfortunate that many athletes and clinicians have "favourite treatments", which may incorporate traditional or historical beliefs and /or myths related to healing of injury. However, many of these interventions are not evidence-based and may in fact extend the injury or at least delay healing. These interventions should be avoided in the period of acute injury and include:

Heat therapy

Heat acts as a vasodilator and as such would encourage bleeding in the early stages of injury. Thus tissue metabolism and the inflammatory process would be accelerated. A previous study has shown that following ankle sprain, recovery takes 6 days following early cryotherapy, 11 days following late cryotherapy and 15 days following heat therapy.[83] Furthermore there seems to be no benefit with respect to injury healing when using alternating hot and cold therapies and thus this practice cannot be recommended.[84]

Alcohol

Alcohol, either in systemic use or in the format of topical use, is a vasodilator and as such would increase bleeding in the early stage of injury. There is no scientific evidence to support its use in this regard.

Rubbing or massage

In the acute phase of injury immobilisation is required as discussed earlier. The use of friction massage, other forms of rubbing and massage, stretching or early exercise of the soft tissue is contraindicated as the tissues are fragile and this would facilitate haemorrhage and tissue necrosis. Similarly, there is no scientific evidence to support the practice of these techniques in the acute phase of injury.

SUMMARY

In summary, soft tissue injuries are very common in sport and particularly in rugby. It is important that soft tissue injuries are correctly managed, particularly in the initial stages after acute injury. In this review, the potential mechanisms, scientific evidence and clinical applications of the "RICE-D" principles of management were reviewed. Wherever possible, practical clinical guidelines based on the best available evidence at the time have been presented. However, further important clinical studies should be conducted, even with respect to some of the more widely practiced interventions, to enhance our understanding and ensure the best treatment of acute soft tissue injuries in rugby players.

PRACTICAL APPLICATION: AN EXAMPLE OF TWO PROTOCOLS OF TREATMENT OF ACUTE ANKLE SPRAIN IN RUGBY PLAYERS

Case study: 1.

A rugby player presents with an acute ankle sprain late during the second half of a rugby match. The player is assessed on-field. He is encouraged to play on as the match is crucial for the team and the score is even. Strapping from one of the other players is provided and the last ten minutes of the game are played, with the injured player experiencing significant pain. After the game, in the pub (where significant quantities of beer are consumed), his wife offers to wrap a hot towel around the injury and rub it in with deep heat to soothe the pain. Before going to sleep, the player ingests a double dose of over-the-counter anti-inflammatory agents in an attempt to get some sleep. The following day the pain and swelling is so severe that the player seeks the assistance of his GP.

Clinical examination of the ankle shows significant swelling, haemorrhage and reduced range of motion. There is marked tenderness on the lateral ligament complex and lateral malleolus; and the patient cannot weight-bear on that limb. An X Ray of the foot and ankle shows no abnormality. A final diagnosis of a Grade I-II ankle sprain is made. The patient is given crutches and an ankle guard and referred to a physiotherapist for further management and rehabilitation. The time to recovery so that rehab can take place for this ankle sprain is 15 days.

Case study: 2.

Another rugby player presents with an acute ankle sprain during a rugby match. The player is assessed on-field by his team physio and he is initially assessed as having a Grade I-II lateral ankle sprain. He is immediately assisted off the field and a cryocuff is immediately applied to the ankle injury for a 15-minute period. Following this period a 10-minute "rest period" is given followed by another 15-minute cryocuff period. Thus both cryotherapy and compression are provided. An ankle brace is immediately applied and crutches are used to get the player to a waiting car to take him home. Once home, he applies ice to his ankle using the 10-minute intermittent protocol every 4-6 hours for the first 24 hours. This is reduced to 3 times a day in the second 24-hour period. He phones his doctor and is advised to take two paracetamol tablets to manage his discomfort and to elevate his ankle on three pillows whilst he sleeps. He is advised to sleep with an elasticised ankle guard to provide some compression. The following evening the player goes to consult with his doctor.

Patient 2 still has difficulty weight-bearing. Clinical examination of the ankle shows some swelling, and reduced range of motion. There is marked tenderness on the lateral ligament complex and lateral malleolus. An X Ray of the foot and ankle shows no abnormality. A final diagnosis of a Grade I-II ankle sprain is made. As 48 hours have now elapsed the doctor prescribes an anti-inflammatory agent and the patient is referred for physiotherapy 1-2 days later. Recovery takes place in 9 days at which time the player is referred to a biokineticist for final-phase rehabilitation and a return to sport programme.

These two hypothetical cases illustrate how correct application of the "RICE-D" principles may lead to an optimal recovery time following acute soft tissue injury.

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