The Progression of Muscle Healing By Using Voltaren Gel (NSAID) in Cases of Delayed Onset Muscle Soreness (DOMS)

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Abstract: The aim of this study is to identify the progression of muscle healing by using NSAID (Voltaren gel) in cases of delayed onset of muscle soreness (DOMS). Twenty injured players were engaged in the study, they were divided to two equal groups, ten injured players each. They were affected with DOMS, the diagnosis is based on clinical findings by specialists. The voltaren gel is applied locally to the skin 3 times daily and rubbed in gently in case of experimental group where as the control received placebo and the general approach after injuries: Protection, Rest, Ice, Compression, Elevation and Support. Biochemical variables estimated: B-endorphin, creatine kinase, ACTH before and after treatment, together with performance tests. Visual pain score and healing time.Results indicated a decreased concentration of B-endorphin, and creatine kinase in experimental group compared to control. Performance tests revealed a positive results also in experimental group which exhibit also better pain score and reduced healing time. **Conclusion:** Using NSAID in cases of DOMS induced rapid healing process and lower pain perception in favor of experimental group.

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1. Introduction

Delayed Onset Muscle Soreness (DOMS) is a very common complaint and another form of muscle injury. Following unaccustomed muscular exertion, athletes develop DOMS and experience a sensation of discomfort and pain in their muscles. For example, after prolonged eccentric muscle activity, athletes may experience pain in all the extensors and flexor muscles groups around the hip, thigh and leg. Structural abnormalities in the Z band and a rise in the serum creatine kinase have been described ***(Eger, 2006)***.

Temporary loss of strength, up to 50% can be present ***(Fomby and Mellion, 1997; Melone et al., 1996).*** The diminished performance may be due to reduced voluntary effort due to pain as well as reduced capacity of the muscle to produce force. The pain is believed to be related to reversible structural damage at the cellular level ***(Reid, 1992).***

There is no associated long term or residual damage or reduced function in the muscles. The syndrome of increasing pain, made worse by stretching the affected muscle groups and eventually sensory changes should alert the physician. The compartment is usually tense and tender to palpation ***(Walsh et al., 2006).***

Non steroidal anti-inflammatory drugs (NSAID), are commonly used in treating musculoskeletal sports injuries. Their use is based on physician's empiric results rather than objective scientific studies. Myorelaxants and analgesics can be prescribed if there is severe pain and spasm and corticosteroid injection to injured area is not indicated due to risk of scar tissue formation ***(Christer et al., 2006).***

Chattergea and Shinde (2006) reported that endorphins are a group of polypeptides which influence the transmission of nerve impulses. They are known as opioides because they bind to those receptors which bind opiates like morphine and plays a role in pain perception. They also added that endorphins are of three types A, B and Y also endorphins bind to CNS receptors like morphine and all of them play a role in the endogenous control of pain perception. Endorphins have a higher analgesic potency than morphine, that is endorphins affect pain suppression than morphine.

2. Subjects and Methods

The experimental method was used for its suitability.

Subjects:

This study was carried out on 20 cases of delayed onset Muscle soreness (DOMS), which is a very common complaint in muscle injury. They were divided into two groups experimental group and control group (10 male each). All of them were assigned to rehabilitation at El Agoza army rehabilitation center. Experimental group were continuous in normal program plus NSAID gel voltaren 3 times daily (100 mg of voltaren emulgel contains 1.16 gr of the active substance diclofenac diethylamine which corresponds to 1 gr diclofenac

sodium. The base of voltaren Emulgel is an oily imulsion in an aqueous gel an is applied to the skin of the injured muscles. The diagnosis is mainly based on clinical findings. Treatment of DOMS is generally symptomatic. Prices is the general approach, Protection, Rest, Ice, Compression, Elevation and Support for 24-36 hours.

For control group, the placebo was the ointment base of voltaren gel i.e. solve without active constituents and without therapeutic effects plasma levels of ACTH was measured by immunoassay techniques. Basal ACTH secretion shows a circadian rhythm, with lower levels in the early evening than in the morning. ACTH is secreted in pulsatile manner, leading to rapid fluctuations superimposed on this circadian rhythm. β–endorphin was measured by ELISA techniques.

Creatine phosphokinase was measured by spectrophotometer.

All biochemical variable were determined using specific kits therapeutic exercises were performed for the control group as for the experimental group, they performed therapeutic exercises plus NSAID voltaren gel until healing.

Table (1): Statistical analysis of the two groups in some basic characteristics:

Variables	Control Mean ± SD	Experimental Mean ± SD	Sig.
Age (yrs)	18.5 ± 3.1	17.9 ± 9.6	NS
Height (cm)	173.1 ± 6.6	172.2 ± 7.4	NS
Weight (Kg)	68.9 ± 6.4	69.8 ± 5.2	NS

$P < 0.05$

Visual pain score was performed together with healing time isotonic leg muscle strength (k) was evaluated on the injured leg using dynamometer together with running 30 mx5, max pressure on sole surface of foot, isotonic leg strength CK, B-endorphin, ACTH were measured before and after treatment, using spectrophotometer, ELISA technique. The participants were instructed to fast before blood sampling. At 09.00 h. to prevent diurnial variation of the hormones. The blood was drawn (5 ml, from cubital vein in tubes containing (EDTA).

Statistical methods

Statistical methods were included using student's "t" test, mean, standard deviation.

The level of significance at the 5% value ($P < 0.05$) was accepted for all statistical tests performed.

3. Results

Table (1) revealed statistical analysis of the control and experimental groups in some basic characteristics (age, height, weight). There was non significant changes ($P < 0.05$).

Table (2, 3) revealed Mean ± SD of the plasma B-endorphin, ACTH and CPK in the two groups before and after therapeutic therapy alone or with voltaren gel treatment, significant changes was noticed for the sake of the experimental group.

Table (4)Mean ± SD of some physical performance parameters of control and experimental groups after the treatments (healing) (Vertical jump, running 30 m x 5 times, max. pressure on sole surface of foot, isotonic leg muscle strength) for the sake of the experimental group.

Table (5)Mean ± SD of the healing time and visual pain score at the end of the therapy for the sake of the experimental group.

Table (2): Mean ± SD of the plasma NB-endorphin, ACTH and creatine phosphokinase in control and experimental groups before and after voltaren gel treatment.

Parameters	Control		Experiment	
	Before	After	Before	After
B-endorphin pg/ml	76 ± 12	44 ± 11*	80 ± 15	31± 9*
ACTH Pg/ml	162 ± 27	81 ± 19*	156 ± 23	62 ± 17*
Creatine kinase IU/L.	562 ± 50	492 ± 48*	554 ± 32	414 ± 35*

$P < 0.05$.

Table (3): Mean ± SD of the plasma B-endorphin, ACTH and creatine kinase in control and experimental groups after treatment:

Parameters	Control	Experiment
B-endorphin pg/ml	44 ± 11	31± 9*
ACTH Pg/ml	81 ± 19	62 ± 17*
Creatine kinase IU/L.	492 ± 48	414 ± 35*

P < 0.05.

Table (4): Mean ± SD of some physical performance parameters of control and experimental groups after healing:

Parameters	Control Mean ± SD	Experimental Mean ± SD	Sig.
Vertical jump (cm)	38.7 ± 4.1	40.5 ± 3.1	Sig*
Running 30m x 5 times	33.2 ± 4.3	28.2 ± 4.5	Sig*
Max. Pres. on sole surface of foot	50.22 ± 2.63	61.1 ± 2.4	Sig*
Isotonic leg muscle strength (k)	78.5 ± 4.2	87.4 ± 5.1	Sig*

P < 0.05.

Table (5): Mean ± SD of the healing time and visual pain score at the end of therapy in control and experimental groups:

Parameters	Control Mean ± SD	Experimental Mean ± SD	Sig.
Healing time (days)	8.7 ± 1.1	5.4 ± 0.9	Sig*
Visual pain score	4.2 ± 0.9	1.9 ± 0.4	Sig*

P < 0.05

4. Discussion

B-endorphin and ACTH (Table 2, 3) indicated concentration in injured muscles (DOMS) and the levels of the two hormone were suppressed after complete healing. The increased levels of β-endorphins and ACTH were also reported due to increased pain by ***Delitale et el., (1991); De Meirleir et al., (1986); Donovan and Andrew, (1987); Edgar et al., (1993) and Goldfarb et al., (1990).***

Guyton and Hall (2006) reported that the analgesia system consists of three major components:

1) The periaqueductal gray and periventricular areas of the mesencephalon and upper pons.
2) The raphe magnus nucleus in pons and medulla oblongata.
3) A pain inhibitory complex located in the corsol horns of the spinal cord. At this point the analgesia signals can block the pain before it is relayed to the brain.

Stocco (2001) and Larsen et al., (2003) stated that when ACTH is secreted by the anterior pituitary gland, several other hormones are secreted simultaneously.

The reason for this is that ACTH synthesis initially causes the formation of proopiomelanocortin (POMC), which is the precursor of ACTH as well as β-endorphins. Thus both hormones are secreted simultaneously when an athletes is subjected to pain or high level of intensity exercises. They also added that several transmitter substances are involved in analgesia system, especially enkephalin and serotonin. Thus the analgesia system can block pain signals at the initial entry point to the spinal cord, leading to decreased sensation of pain.

Braunwald et al., (2001) stated that ACTH and a number of peptides included β–endorphon are made in anterior pituitary gland. They varies during the day as a result of their pulsatile secretion. Stress causes their release and activation of sympathetic nervous system. ACTH increased glucocorticoids having anti-inflammatory and modulate the immune response via the so-called immune-adrenal axis. This loop is one mechanism by which stress increase adrenal hormone secretion leading to decrease inflammation induced by muscle injury. This anti-inflammatory action may lead to the rapid healing reported in the experimental group (Table 2, 3) due to the action of β–endorphin which suppress pain, ACTH as anti-inflammatory action and reducing muscle damage as showed by reduced creatine kinase.

Melchionda et al., (1984) suggested that the increase in B-endorphin may serve as a form of pain modulation, also ***Gombert et al., (1981)*** suggested that the function of beta endorphin in the blood may perform a function in the modulation of the exercise induced pain or stress. Stress induced increased ACTH

which in turn increased adrenal cortex hormones leading to metabolic changes in most cells that help to resist stress ***(Farrell et al., 1983).***

Muscle damage is associated with increases in creatine kinase concentration ***(Kuipers, 1994)*** and its routine biochemical evaluation in the diagnosis of muscle disease has been proposed ***(Clarkson et al., 1992).*** Table (2, 3) indicated that muscle injury in case of the control and experimental group denoted an elevated concentration of CK after muscle injury and followed by a reduction after healing in both group. The decreased concentration of CK was lower after NSAID (voltaren) compared to the control group. Since inflammation appears to play an important role in the pathogenesis of muscular damage induced by continuous exercise, many studies attempted to evaluate the ability of (NSAID) in reducing the biochemical signs of muscular injury, and appear to significantly reduce creatine kinase ***(Bourgeois et al., (1999) and Cordova et al., (2004).***

Muscle soreness and weakness accompany intense or prolonged physical activity. Voltaren gel message help formulate an understanding of its effect and role in exercise related muscle pain, due to its anti-inflammatory and antiedema effect of voltaren gel, hence reducing pain ***(Best et al., 2013).*** It was also noted that mechanical stimulation accompanying voltaren gel message might increase the ability to regenerate muscle fibres, which is very important in the process of muscle healing which occur after muscle degeneration, this may account for the rapid healing denoted in the experimental group compared to control group (Table 4). It is also noted that dystrophin positive cells were counted following with mechamical stimulation ***(Choi et al., 2012; Eom et al., 2011; Pecanha et al., 2012).***

Physical performance and recovery from healing and exercise are enhanced by optimal nutrition. During time of high physical activity and recovery of healing energy and nutrient must be met in order to replenish glycogen stores and providing adequate protein for building and repair of tissues ***(American Dietetic Association ADA, 2005).***

As for physical performance table (4) it was noticed that performance increased in case of experimental group compared with the control group. This result may be due to the method of voltaren treatment which reduced inflammation and pain, accordingly increased the strength of the muscle.

Previous studies evaluating the efficacy of anti-inflammatory drugs in muscular damage and muscular healing have suggested that only steroids may prevent this phenomenon, but the use of these drugs is associated with severe side effects ***(Hasson et al., 1993, Jacobs et al., 1996).***

In contrast, voltaren (NSAID) treatment is associated with no side effects ***(Prieto et al., 2001).*** Also the effect of treatment on muscle strength and vertical jump and performance was also reported by ***Byrne and Eston, (2002).*** Who reported that the performance effect may be due to neuroendocrine effects or due the anti inflammatory effects which is the underlying mechanism in delayed onset muscle soreness ***(Smith, 1991).***

From the above discussions the hypothesis that NSAID gel might lead to the progression of healing in cases of delayed onset muscle soreness has been realized.

Conclusion

It may be concluded that NSAID (voltaren gel) may affect the interrelated phases of muscle healing by decreasing inflammation and pain through neuroendocrine stimulation of B-endorphin and ACTH and by reducing muscle damage marker creatine phosphokinase in cases of delayed onset muscle soreness (DOMS).

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