Trigger Point Lidocaine Injection Versus Acupuncture in Chronic Low Back Pain

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Master Degree in Anaesthesiology and Intensive Care

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Abstract

Aim To compare the efficacy of acupuncture needling and 0.5% lidocaine injection of trigger points in chronic low back pain.

Methods Sixty participants with chronic low back pain were randomised to treatment with either acupuncture needling (n=30) at specific points or 0.5% lidocaine injection (n=30) at 2-3 trigger points twice weekly for 4 weeks. Pain scores, pressure pain intensity and depression were measured up to eight weeks from the first treatment.

Results Both groups improved, but there was no significant difference in reduction of pain in the two groups at any time point . Changes in depression showed only trends.

Conclusion There was improvement in both group in low back pain, and no significant difference between acupuncture needling and 0.5% lidocaine injection of trigger points for treating chronic low back pain.

Keywords

Acupuncture, lidocaine injection, trigger points, chronic low back pain.

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Dr. Mona Samir Allam

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LIST OF ABBREVIATIONS

ACTH	Adrenocorticotropic Hormone
AMA	American Medical Association
ASIPP	American Society of Interventional Pain Physicians
BMI	Body Mass Index
BTX-A	Botulinum Toxin type A
cLBP	Chronic Low Back Pain
Dipl.Ac	Diplomate of Acupuncture
DNIC	Diffuse Inhibitory Noxious Control
EBM	Evidence Based Medicine
EMG	Electromyography
FDA	Food and Drug Adminstration
FMRI	Functional Magnetic Resonance Imaging
GDS	Geriatric Depression Scale
Lac's	Licensed Acupuncturists
LBP	Low Back Pain

MPS	Myofacial Pain Syndrome
MRI	Magnetic Resonance Imaging
NCCAM	National Center for Complementary and Alternative Medicine
NIH	National Institutes of Health
NSAIDs	Non Steroidal Anti-Inflammatory Drugs
PET	Positron Emission Tomography
PPI	Pain Pressure Intensity
PPT	Pain Pressure Threshold
ROM	Range of Movement
QOL	Quality of Life
SD	Standard Deviation
SSEP	Somatosensory Evoked Potential
TCM	Traditional Chinese Medicine
TIH	Tension Headache
TrPs	Trigger Points

US	United States
VAS	Visual Analogue Scale

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INTRODUCTION

Low back pain is the fifth most common reason for all physician visits in the United States (**Hart et al., 1995 , Deyo et al., 2002**).

Approximately one quarter of U.S. adults reported having low back pain (LBP) lasting at least 1 whole day in the past 3 months (**Deyo et al., 2002**), and 7.6% reported at least 1 episode of severe acute low back pain within a 1-year period (**Carey et al., 1996**). Low back pain is also very costly, total incremental direct health care costs attributable to low back pain in the U.S. were estimated at $26.3 billion in 1998 (**Luo et al., 2004**). In addition, indirect costs related to days lost from work are substantial, with approximately 2% of the U.S. work force compensated for back injuries each year (**Andersson, 1999**).

Many patients have self-limited episodes of acute low back pain and do not seek medical care (**Carey et al., 1996**). Among those who do seek medical care, pain, disability, and return to work typically improve rapidly in the first month (**Pengel et al., 2003**). However, up to one third of patients report persistent back pain of at least moderate intensity 1 year after an acute episode, and 1 in 5 report substantial limitations in activity (**Von Kroff et al., 1996**). Approximately 5% of the people with back pain disability account for 75% of the costs associated with low back pain (**Frymoyer et al., 1991**).

Many options are available for evaluation and management of low back pain. However, there has been little consensus, either within or between specialties, on appropriate clinical evaluation (**Cherkin et al. a, 1994**) and management (**Cherkin et al., 1995**) of low back pain. Numerous studies show unexplained, large variations in use of diagnostic tests and treatments (**Cherkin et al. b, 1994 , Volinn et al., 1992**). Despite wide variations in practice, patients seem to experience broadly similar outcomes, although costs of care can differ substantially among and within specialties (**Carey et al., 1995 , Shekelle et al., 1995**).

Complementary modalities as acupuncture and trigger point injection are considered among the options of management .

Acupuncture has demonstrated its potential as a promising treatment for chronic LBP (**Brinkhaus et al., 2006 , Molsberger et al., 2008**). While a number of theories of how acupuncture may treat LBP are available, no accepted mechanism has emerged (**Molsberger et al.,**

2008 , Koes et al., 1996). Similar to descending inhibitory and/or diffuse noxious inhibitory controls in the central nervous system, acupuncture may stimulate the small-diameter afferent fibres, which then reduce the transmission of pain signals thereby inhibiting pain discrimination and perception (**Kawakita et al., 1996**). Low back muscle spasm and muscle blood flow decrease are the main underlying causes of chronic LBP (**Johnson et al., 1989**). Acupuncture alleviates tension and improves blood flow in the treated muscles (**Kawakita , 1993**). Thus, acupuncture treatment may improve lumbar function and reduce pain via increasing the blood flow to the affected region (**Leibing et al., 2002**).

Trigger point injection can help to release tension in the muscle band and help the body to heal , also it alleviates tension and improves blood flow in the treated muscle (**Kawakita et al., 1993**).

It is well established that painful stimulation inhibits pain, and diffuse noxious inhibitory control (DNIC) has been proposed as a physiological basis of acupuncture and trigger point analgesia (**Bing et al., 1990**).

Aim of The Work

1-To determine the efficacy of acupuncture as well as trigger point injection in the management of chronic LBP .

2-To compare between both modalities in the management of chronic LBP .

Chronic pain

Introduction:

Chronic pain has several different meanings in medicine. Traditionally, the distinction between acute and chronic pain has relied upon an arbitrary interval of time from onset; the two most commonly used markers being 3 months and 6 months since the initiation of pain (**Turk and Okifuji , 2001**), though some theorists and researchers have placed the transition from acute to chronic pain at 12 months (**Main and Spanswick , 2001**). Others apply acute to pain that lasts less than 30 days, chronic to pain of more than six months duration, and subacute to pain that lasts from one to six months (**Thienhaus and Cole, 2002**). A popular alternative definition of chronic pain, involving no arbitrarily fixed durations is "pain that extends beyond the expected period of healing (**Turk and Okifuji, 2001**) .

Classification:

Chronic pain may be divided into "nociceptive" (caused by activation of nociceptors), and "neuropathic" (caused by damage to or malfunction of the nervous system) (**Keay et al., 2000**).

Nociceptive pain may be divided into "superficial somatic" and "deep", and deep pain into "deep somatic" and "visceral". Superficial somatic pain is initiated by activation of nociceptors in the skin or superficial tissues. Deep somatic pain is initiated by stimulation of nociceptors in ligaments, tendons, bones, blood vessels, fasciae and muscles, and is dull, aching, poorly-localized pain. Visceral pain originates in the viscera (organs). Visceral pain may be well-localized, but often it is extremely difficult to locate, and several visceral regions

produce "referred" pain when injured, where the sensation is located in an area distant from the site of pathology or injury (**Coda and Bonica, 2001**).

Neuropathic pain is divided into "peripheral" (originating in the peripheral nervous system) and "central" (originating in the brain or spinal cord) (**Bogduk and Merskey, 1994**). Peripheral neuropathic pain is often described as “burning,” “tingling,” “electrical,” “stabbing,” or “pins and needles.” (**Paice , 2003**) .

Pathophysiology:

Under persistent activation nociceptive transmission to the dorsal horn may induce a wind up phenomenon. This induces pathological changes that lower the threshold for pain signals to be transmitted. In addition it may generate non nociceptive nerve fibers to respond to pain signals. Non nociceptive nerve fibers may also be able to generate and transmit pain signals. In chronic pain this process is difficult to reverse or eradicate once established (**Vadivelu and Sinatra, 2005**).

Chronic pain of different etiologies has been characterized as a disease affecting brain structure and function. Magnetic Resonance Imaging studies have shown abnormal anatomical (**Geha et al., 2008**) and functional connectivity (**Baliki et al., 2008**) involving areas related to the processing of pain. Also, persistent pain has been shown to cause grey matter loss, reversible once the pain has resolved (**May , 2009**).

Management:

Complete and sustained remission of many neuropathies and most idiopathic chronic pain (pain that extends beyond the expected period of healing, or chronic pain that has no known underlying pathology) is