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Estimation of Plasma Dopamine Level after Intramuscular Trigger Point Corticosteroid Injection in Patient with Myofascial Pain Syndrome

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Abstract

Background: Myofascial pain dysfunction syndrome (MPDS) is a disorder comprising pain, jaw movement problems, and muscle spasm. Hyper-excitation of peripheral sensory neurons leads to spasms of the masticator muscles, causing an inductionresponse in the motor neuron.**Methods:**This study was conducted in private dental center during the period from 27/4/2019 to 3/11/2019. Thirty-three patients with myofascial pain syndrome and 25 control participants were included in this study, all patients were diagnosed by oral medicine specialist, and single dose steroid mixed with 0.5% lidocaine was injected in the tender muscles trigger point which includes masseter, temporalis and lateral pterygoid muscles. The goal of this study was to investigate plasma levels of dopamine in patients with myofascial pain syndrome (MFS) and healthy controls and assessment of plasma dopamine level, grinding tenderness scheme and visual analogue scale after trigger point steroid injection. **Results:**Dopamine in plasma differed significantly increased in between patients with Myofascial pain syndrome (1290.2 ± 314.4) and healthy controls (479.4 ± 148.4; $P < 0.01$) regarding to post injection group decreased significantly (1001.4 ± 232.9; $P < 0.01$). Patients stated significantly higher pain intensities VAS scale ($P < 0.001$) and had lower intensities ($P < 0.01$) compared with the post corticosteroid TrP injection.**Conclusions:** Trigger point injection is a valuable procedure for pain relief for patients in myofascial pain syndrome.

INTRODUCTION

Myofascial pain dysfunction syndrome (MPDS) is a disorder comprising pain, jaw movement problems, and muscle spasm. Hyper-excitation of peripheral sensory neurons leads to spasms of the masticator muscles, causing an induction response in the motor neuron. Long-term spasm results in muscular pain and irregular movements of the mandible. Pain is the most important trigger, so it should be the first thing to be dealt with in the management the muscle spasms [1].Myofascial pain dysfunction syndrome is an important cause of chronic Orofacial pain. It is more common among young unmarried females. Different etiologies include occlusalirregularities, intra-capsular problems, emotional stress, direct or indirect trauma,spine diseases, psychogenic causes like stress and bruxism. The presence of trigger point is an essential characteristic property of MPDS. Clinical characteristics include TMJ clicking sounds, defect in mandible movement, truisms, facial painpreauricular pain and jaw

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tenderness during function.Currently,treating the primary cause is the most widely accepted management strategy, in non-surgical or surgical approach [2].Dopamine is a neurotransmitter found in plasma and the central and peripheral nervous systems. In the CNS, dopamine plays an important role in motor regulation, cognition, and the reward system [3] but may involve also in pain perception [4] dopamine is synthesized in the CNS by dopaminergic neurons synthesize while in the peripheral nervous system by neuronal fibers, adrenal medulla, and neuroendocrine cells synthesize dopamine [5]. Rubi and Maechler reported that dopamine elevated in plasma in situations of psychological stress, hypovolemic, or muscle exercise. Bruxism, "a repetitive jaw-muscle action characterized by clenching or grinding of the teeth and/or by bracing or thrusting the mandible" has been related to abnormal levels in the central dopaminergic system [6].It has also been reported that changes in dopaminergic neurotransmission might be occurred in burning mouth syndrome [7]. Other conditions include fibromyalgia [8], and chronic Orofacial pain [9].The assumption is that the dopaminergic system may have a role in central pain modulation, but the role of dopamine at the peripheral level related to pain is uncertain, steroid act by blocking the body's inflammatory response to damage by many mechanisms, with adecrease in fluid transudation and edema. Intramuscular administration permits the use of repository (acetate) steroid drug forms, which allows slow absorption and long-term action [10]. When administered locally, steroids exert direct effect on eicosanoid and thereby prevent inflammatory processes. Additionally, locally applied Glucocorticoidsinhibit the signal transmission in nociceptive C-fibers and ectopic neuro mA discharge in damaged nerve [11].Myofascial trigger point (MTrP) is a highly localized painful or sensitive spot located in a palpable taut band of skeletal muscle fibers in patients with myofascial pain syndrome (MPS) [12]. Painsfrom MTrPs can presents spontaneously or in response to movement. A latent MTrP is a sensitive spot at which pain or discomfort occurs by compression only. The diagnosis of MPS, which consist of one or more active trigger point, usually is based on the patient's subjective symptoms and the presence of an active myofascial trigger point characterized by 1) tender spots in one or more palpable taut band, 2) a referred pain pattern, 3) a local twitch response (LTR), and 4) restricted range of motion (ROM) [13].Trigger point injection (TPI) with local anesthetic solution,steroids or saline, acupuncture, muscle massage, acupressure, ultrasonography, application of hot or cold, transcutaneous electrical nerve stimulation, and ethyl chloride Spray and Stretch technique [14].

MATERIAL AND METHODS

This study was conducted in private dental center during the period from 27/4/2019 to 3/11/2019. Thirty-three patients with myofascial pain syndrome and 25 control participants were included in this study, all patients were diagnosed by oral medicine specialist, and single dose steroid mixed with 0.5% lidocainewas injected in the tender muscles trigger point which includes masseter, temporalis and lateral pterygoid muscles.Each subject was informed about the objectives of the study and consent was obtained from patient according to special forma depending on College of Dentistry University of Kufa. Case sheet of information about name, gender, age, medical and family history, pain history depending on visual analogue scale (VAS scale) grinding tenderness scheme and body mas' index was recorded.A total of 3 ml Venous blood was collected from all the participants in the study and control groups and

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another blood sample was collected from only the study group after 20 days from the injection days and all samples were stored in deep freeze until the end of the treatment.Dopamine hormone analysis was performed in private laboratory usingElabsciencekit, plasma was collected using EDTA as an anticoagulant,centrifuge samples for 15 min at 1000×g at 2-8℃ within 30 min of collection. The supernatant wascollected to carry out the test. 100 µL standard or sample was adding to each well and Incubated for 90 min at 37°C,after that removed the liquid andadding 100 µLBiotinylated Detection Ab to Incubate for 1 hour at 37°C procedure continuous after Aspirate and wash 3 times,add 100 µL HRP Conjugate. Incubate for 30 min at 37°C. Aspiration and washing 5 times 90 µL Substrate Reagent Add for each well, incubated for 15 min at 37°C. Finally50 µL Stop Solution add to finish procedure and reading at 450 nm immediately to calculate results. Body mas indexed was recorded for all participants, visual analogue scale for pain and grinding tenderness scheme was recorded for the study group to indicate the severity of pain in 0-10 scale and I-III regarding to tenderness scheme.

Excluded criteria

- Patient with symptoms and signs meeting the 1990 ACR (American College of Rheumatology) criteria for fibromyalgia;
- Patient with sign and symptoms of systemic diseases;
- Allergy history for drugs or injections,
- Having evidence of a cognitive deficit or difficulty with communication; exhibiting inadequate co-operation.

RESULT

Age and BMI descriptive statistics and differences showed that no significant differences P-value 0.74 and 0.61 respectively in between patients and control, mean, standard deviation and independent t-test in the table (1)

Table (1): Descriptive statistics and differences in age and BMI between patients and control

	Patients	Control	df	T - test	P value
Age(Years)	33.19 ± 6.55	33.19 ± 6.55	52	0.33	0.74
BMI(Kg/m^2)	28.2 ± 4.27	29.22 ± 4.54	52	0.5	0.61

Dopamine plasma level highly significantly increased in between patient and control and level decreased significantly after MTrP corticosteroid injection with P-value 0.000 using F-test as in table (2).

Table (2): Differences in dopamine Plasma level of Pre-injection patients, Post-injection patients and control groups

Dopamine Plasma Level	Mean ± SD	F-test	P value
Pre	1290.2 ± 314.4 A	59.2	0.000
Post	1001.4 ± 232.9 B		
Control	479.4 ± 148.4 C		

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Different letters refer to high significant difference at P value < 0.01; SD: Standard Deviation

Visual Analogue Scale (VAS scale) showed that highly significant reduction in pain intensity after MTrP corticosteroid injection using Z test with P-value 0.000

Table (3): Differences in Visual Analogue Scale level of patients before and after steroidMTrP corticosteroid injection

	Pre Patients(MS ±SD)	Post Patients(MS ±SD)	n	Z test	P value
Visual Analogue Scale	7.37 ± 1.14	3.03 ± 1.19	27	4.54	0.000 *

MS: Mean of Scores; SD: Standard Deviation

* p <0.01: High significant difference by Wilcoxon Signed Rank Test

Regarding to granding tenderness scheme lateral pterygoid, masseter, temporalis muscle and TMJ joint capsule showed highly significant differences in reducing tenderness using Z-test with P-value 0.000 for each scheme as in tables (4-7)

Table (4): Differences in grandingtendernessschemelateral pterygoid muscle of patients before and after injection

	Pre Patients(MS ±SD)	Post Patients(MS ±SD)	N	Z test	P value
Lateral pterygoid m.	2.14 ± 0.81	0.48 ± 0.75	27	4.37	0.000 *

MS: Mean of Scores; SD: Standard Deviation

* p <0.01: High significant difference by Wilcoxon Signed Rank Test

Table (5): Differences in granding tenderness scheme masseter muscle of patients before and after injection

	Pre Patients(MS ±SD)	Post Patients(MS ±SD)	n	Z test	P value
Masseter m.	0.4 ± 0.5	0.07 ± 0.26	27	4.37	0.000 *

MS: Mean of Scores; SD: Standard Deviation

* p< 0.01: High significant difference by Wilcoxon Signed Rank Test

Table (6): Differences in granding tenderness scheme Temporalis muscle of patients before and after injection

	Pre Patients(MS ±SD)	Post Patients(MS ±SD)	n	Z test	P value
Temporalis m.	1 ± 0.62	0.14 ± 0.36	27	4.01	0.000 *

MS: Mean of Scores; SD: Standard Deviation

* p< 0.01: High significant difference by Wilcoxon Signed Rank Test

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Table (7): Differences in granding tenderness scheme Joint capsule of patients before and after injection

	Pre Patients(MS ±SD)	Post Patients(MS ±SD)	n	Z test	P value
Joint capsule	1.96 ± 0.58	0.25± 0.44	27	4.54	0.000 *

MS: Mean of Scores; SD: Standard Deviation
* p< 0.01: High significant difference by Wilcoxon Signed Rank Test

DISCUSSION

Dopamine plasma level highly significantly increased in between patient and control, with mean and standard deviation reach to 1290.2 ± 314.4 while control 479.4 ± 148.4 the plasma dopamine level significantly decreased after MTrP corticosteroid injection with P-value 0.000 and mean and standard deviation 1001.4 ± 232.9to discussthese results at beginning the role of dopamine in chronic pain is a fact and agree with previous studies stated that peripheral dopamine might be implicated in modifying peripheral pain. This point, together with reports in other studies, proposes that dopaminergic pathways could have a role in the pathophysiology of M-TMD, in addition to other chronic pain conditions [15].Previous studies have showed that dopaminergic neurotransmission is changed at a central level in group of patients with chronic pain conditions 8, 9. But the finding in this study that dopamine in plasma increased significantly with present pain intensity and granding tenderness scheme in the lateral pterygoid, masseter, temporalis muscle and joint capsule of TMJ with significantly decreased post corticosteroid TrP injection suggested that involvement of dopamine in pain modulation, at not only a central but a peripheral level as well [16].Chronic pain is hard to manage and sometimes harder to diagnose. In the brainstem, different pain modulators control the way the body perceives pain. Researchers are beginning to define the mechanics of chronic pain, to search how sensory neurons can become hypersensitive, inducing prolonged and exaggerated responses to stimuli.Findings from a study found one of the body's most important neurotransmitters was essential in the development of chronic pain—dopamine.Previous research has shown an alteration in dopamine receptors in fibromyalgia, burning mouth syndrome, and atypical facial pain. Dopamineis the focus of pharmaceutical researches in the setting of pain management. For over a century, many practitioners have used dopaminergic drugs to treat chronic pain. Usually classified as 'stimulants,' they potentiate opioid and minimize their use [17].In present study dopamine plasma level decreased significantly after TrP corticosteroid injection this disagree with previous studies that stated Glucocorticoids' enhancement of dopaminergic activity may explain the development of psychosis/ delusions in the context of the depressive episode [18]. Inother study in vitro stated that dopamine blocks steroid release [19], therefore this study suggested that dopamine level decreased after corticosteroid injection.

Research limitation: some patient fairing from intra-muscular injection of steroid especially after informed about complication of steroid even single dose, therefore some patient rejected treatment and also the group of patient accepted the injection dropping through follow up.

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