Myofacial Pain and TMJ Disorders Botox Treatment with Oldness Affection

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Abstract:

Background:

TMJD is a disorder caused by masticatory myoarthropathy, which can be treated with botulinum toxin injections. This study aims to determine the effects of BTX-A injections on joint click, pain intensity, deviation on opening and maximum mouth opening.

Materials and Methods

Male and female patients between the ages of 16 and 42 were included in the human sample, all of whom were experiencing myofacial discomfort, trismus, and TMJ noises due to temporomandibular joint dysfunction. Individuals receiving 100 U of botulinum toxin type A (BTX-A) injected at 100 mL per temporalis, lateral pterygoid and masseter muscle were enrolled in the trial. Each individual was randomly allocated to one of four groups.: 7 individuals, both male and female, aged 16-22, made up Group I. Patients aged 23-29 made up Group II, which also included 7 men. 7 individuals between the ages of 30 and 36 made up Group III. Group IV had 7 patients (aged 37-43) of both sexes experiencing myofacial discomfort due to temporomandibular joint dysfunction.

The clinical evaluation

This study evaluated damage to the teeth and gums from clenching and grinding, also known as bruxism or parafunction. Four key TMD symptoms were reported both before and after a BTX-A injection: pain level, maximum mouth opening, deviation on opening and joint click. Clinical effectiveness and safety were assessed ten days, 30 days, and 3 months following injection. Results showed that older patients in Group IV (older age) reported the highest mean and SD values for pain intensity, maximum mouth opening, joint click, and deviation on opening. Group III finished in second place, followed by Groups II and I. (younger age). Maximal mouth opening, joint click, and opening deviation all showed improvement in HS between the first (pre-treatment visit) and subsequent visits following BTX-A injection.

Conclusions:

Oldness-related changes in neuromuscular junction physiology lead to muscle mass and strength loss, and the older group required larger doses of BTX-A than the younger group for treatment of temporomandibular joint problems and pain reduction.

Keywords:

Myofascial Discomfort, Arthritis, Oldness, Botulinum Toxin, TMJ Problems.

The term "temporomandibular disorder" (TMD) refers to arthrogenic and myofascial conditions that affect the TMJ, often in conjunction (1). Myofacial TMD is characterized by discomfort from overactive chewing muscles, resulting in inflammation and regional muscular hypoxia that eventually lead to chronic myositis. To the contrary, arthrogenic TMD is linked to intracapsular disease and joint-specific discomfort. Common symptoms include periauricular discomfort, headache, neck pain, jaw locking, noise at the joint with movement, and limited jaw excursion. Other symptoms may also be connected to pain around the joint and include any of the following: (2,3). Without proper care, periodontitis, which is brought on by poor dental hygiene, can eventually cause discomfort while chewing or the loss of teeth, as well as cause alterations to the normal structure of the jawbone and teeth. (4).

Botulinum toxin is a 150-kilodalton exotoxin produced by the bacteria Clostridium botulinum. It inhibits the acetylcholine (Ach) that is released at the neuromuscular junction. in addition to stopping muscular action, Clinical applications of BTX-A included pain alleviation for myofacial discomfort, reduction of TMJ soreness, and restoration of TMJ functioning (5,6).

In light of these considerations, we set out to examine the efficacy and duration of BTX-A injections into the temporalis, lateral pterygoid and masseter, muscles for the treatment of TMDs symptoms, specifically pain intensity, deviation from normal mouth opening, maximum mouth opening, and joint click in relation to patient age over the course of three months.

Material and Methods:

The 28 patients in the human sample—both men and women, aged 16 to 42—had myofacial discomfort, trismus, and TMJ noises associated with TMJ dysfunction. Patients who received treatment at the dental clinics of the specialised Swake centre in Babylon city between February 2016 and June 2019 were enrolled in the study. Each participant received three injections of 100 mL of botulinum toxin type A (BTX-A) into the

temporalis. masseter. and lateral ptervgoid muscles. Four groupings of topics were created: A. Group I, which included 7 men and 7 girls between the ages of 16 and 22, contained patients with myofacial discomfort symptoms and TMJ problems. B. Group II: The patients were 7 men and 7 females with ages ranging from 23 to 29 and symptoms of myofacial discomfort and TMJ problems .C.Group III: The patients, 7 males and 7 females with ages 30-36, experienced myofacial discomfort symptoms along with TMJ problems .D.Group IV: The patients, 7 men and 7 females with ages 37 to 43, all showed symptoms of myofacial discomfort and TMJ problems.

Inclusion criteria:

TMD was a condition that affected research participants for more than six months. After non-invasive therapy failed, BTX was utilized. The usage of patients' medical information was disclosed to them. The ethics committee granted the study's request for permission to proceed ^(1,7). **The exclusion criteria** included those who disclosed a history of any known trauma affecting a luxated TMJ. Subjects who had any extra-articular issues, such as face or neck abscesses or infections, that might have reduced mouth opening were excluded from the trial. Study participants with diseases including oral submucous fibrosis or those who had undergone any type of TMJ surgery were also disqualified ⁽⁷⁾.

Clinical evaluation:

To assess any periodontal, dental, or occlusion issues brought on by bruxism or parafunction, an intraoral examination was performed. It was also assessed whether dentoskeletal dysmorphosis existed. The initial (pre-treatment visit around one week before injection) and subsequent reports following BTX- Pain intensity, maximal mouth opening, deviation on opening and joint click were the four primary TMD symptoms discovered by an injection. Clinical evaluations of the injection's effectiveness and tolerance were carried out ten days, 30

days, and three months after that. Re-examination of the patients was done to measure the pain severity, maximal mouth opening, deviation on opening, joint click, symptom relief, and duration of the BTX effect.

Statistical analysis was performed with the help of the Social Science Statistical Software (SPSS Inc). The quantitative variables' estimates were derived using central tendency (mean) and dispersion statistics (standard deviation). As the data were normally distributed, we were able to compare the intervals between visits using the paired t-test. Two-tailed tests were performed on all statistical data at a.05 level of significance.

Results: Patients' ages varied from 29.4 to 7.879 on average (ranged from 16.2 to 42.0 years). The gender and

age distributions of (Table- 1). Group IV (older age) exhibited the greatest mean and SD (standard deviation) values for pain intensity, joint click, deviation on opening, and maximal mouth opening during the pre and post-treatment visit following the BTX-A injection. The third, second, and first groups (younger age) followed (Table-2). T-test and P-value comparisons between the first (pre-treatment) and second (post-treatment) visits after BTX-A injection showed Substantial changes in pain intensity, mouth opening maximum, mouth opening deviation, and joint click (Figures 1,2,3,and 4)

eviation, and joint click (Figures 1,2,3,and 4)

Table 1: Age and gender distribution

Oldness	S	ex	Total	
	Male	Female		
16-22	3	4	7	
23-29.	1	6	7	
30-36	2	5	7	
37-43.	3	4	7	
Total	9	19	28	

Table-2: descriptive data on the degree of discomfort, the widest possible mouth opening, the joint click, and the opening deviation

	Groups	N.	Pre	Pre	1st	1st	2nd	2nd SD	3rd	3rd
			Mean	SD	Mean	SD	Mean		Mean	SD
pain intensity	Group1	7	8.785	0.505	3.728	0.388	2.1	0.213	1.928	0.127
	Group2	7	9	0.338	4.328	0.249	2.914	0.253	2.457	0.271
	Group3	7	9.185	0.294	5.971	0.310	3.985	0.203	4.971	0.218
	Group4	7	9.171	0.310	6.528	0.271	4.585	0.304	5.557	0.282
maximum	Group1	7	8.542	0.206	4.514	0.229	4.057	0.184	4.471	0.224
mouth opening	Group2	7	8.542	0.244	5.585	0.269	5.014	0.247	5	0.239
	Group3	7	8.571	0.291	6.042	0.244	5.542	0.277	6.042	0.244
	Group4	7	8.571	0.291	6.571	0.265	6.071	0.265	7.071	0.265
joint click	Group1	7	9.057	0.219	3.542	0.206	2.557	0.219	2.042	0.206
	Group2	7	9.028	0.260	4.028	0.205	3.028	0.205	3	0.244
	Group3	7	9.028	0.260	5.042	0.244	4.042	0.244	5.042	0.238
	Group4	7	9.028	0.260	5.557	0.219	4.528	0.260	5.557	0.219
deviation on	Group1	7	7.071	0.265	3.071	0.265	2.1	0.250	1.557	0.219
opening .	Group2	7	7.071	0.291	4.014	0.247	3.557	0.219	4.1	0.287
	Group3	7	7.071	0.265	5.042	0.244	4.028	0.254	4.557	0.219
	Group4	7	7.071	0.265	6.071	0.231	5.042	0.244	5.571	0.244

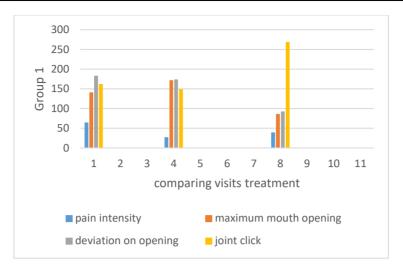


Figure-1: Intensity of discomfort, widest possible opening of the mouth, presence or absence of joint click, and angular deviation upon opening were compared between the first (pre-treatment) and second (post-treatment) visits for group1 by used T-test and p-value.

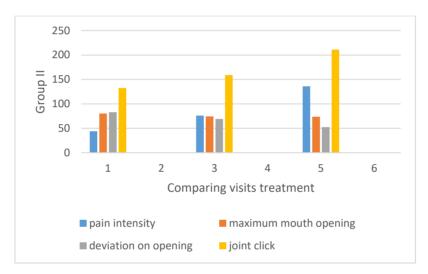


Figure-2: Intensity of discomfort, widest possible opening of the mouth, presence or absence of joint click, and angular deviation upon opening were compared between the first (pre-treatment) and second (post-treatment) visits for group II by used T-test and p-value.

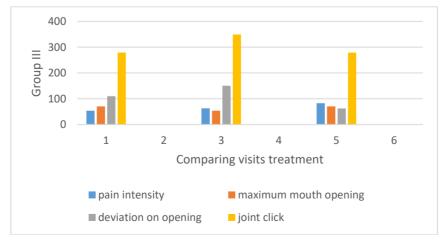
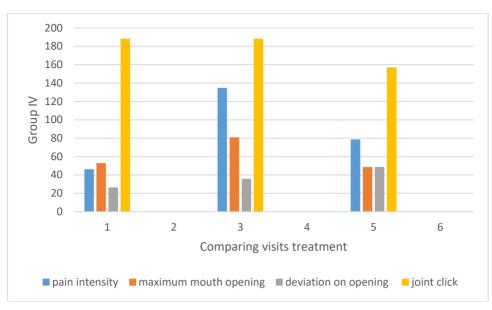


Figure-3: Intensity of discomfort, widest possible opening of the mouth, presence or absence of joint click, and angular deviation upon opening were compared between the first (pre-treatment) and second (post-



treatment) visits for group III by used T-test and p-value.

Figure-4: Intensity of discomfort, widest possible opening of the mouth, presence or absence of joint click, and angular deviation upon opening were compared between the first (pre-treatment) and second (post-treatment) visits for group IV by used T-test and p-value

Discussion:

Many medical disorders connected to muscle tightness or pain have been treated with BTX-A. Injecting botulinum toxin into the masseter, temporalis, and other facial and masticatory muscles related to TMD is a common method of treating orofacial symptoms⁽⁶⁾. By preventing acetylcholine from being released at the neuromuscular junction, the use of botulinum toxin causes a limited reduction in muscle activity, which results in flaccid paralysis of the muscle fibers. Six hours after receiving BTX, paralysis begins to set in, and clinical consequences are seen 24-72 hours later (8). Natural botulinum toxin serotype A (BTX-A) blocks acetylcholine (ACH) production at the neuromuscular junction of presynaptic motor nerve terminals, hence preventing muscle contractions. Immediately after injection, this neurotoxic can lower the local muscle's strength, a reduction that can continue up to three to six months before the muscle regenerates its innervation and resumes functioning normally (9). By modulating the release of numerous endogenous substances like substance P, calcitonin gene-related peptide, and glutamate, it is hypothesized that Tts' analgesic effect uses independent action on peripheral nociceptors (10). Additionally, BTX-A may increase the effectiveness of treatment for those with tension-type headaches by reducing parafunctional oral behaviors that use the masticatory muscles (11) (12) Injections of 100 U of botulinum BTXA has a therapeutic effect that decreases muscle activity by blocking the mechanisms that cause excessive muscle contractions, and this effect decreases with increasing patient age, decreases the masseter muscle, temporalis and lateral pterygoid muscle pain intensity, eliminates the joint click sound, increases the maximum mouth opening (interincisal distance that has been limited), and reduces deviation. The effectiveness and duration of BTXA injections treatment was shown in the current study to be greater in younger TMD patients than older TMD patients when comparing pre- and posttreatment results of each group regarding pain intensity, joint click, maximum mouth opening, and deviation on opening revealed. Presynaptic terminal ultrastructure is primarily defined by the presence of a significant number (usually) of synaptic vesicles and a range of other organelles, such as autophagosomes and endoplasmic mitochondria, and reticulum, endosomes. neurotransmitters to be released, all of these factors must work together, and it is possible that these factors will change as we age (14). In addition, the presynaptic membrane may be "bare" and directly apposed to the extracellular matrix in some locations due to the absence of the subsynaptic reticulum in the postsynaptic muscle

cell. Aging flies also have NMJs that are generally shorter and thinner than those of younger flies, have more variable vesicle sizes, and have a decrease in distant vesicle density (15). Old causes changes in muscle physiology. These old-related changes to muscular strength, function, and mass (16). Strength decline is inversely correlated with loss of muscle mass. Reduced muscle fiber and motor unit counts, as well as a drop in muscle fiber size, are the main contributors to muscle mass loss. Apoptosis starts if muscle fibers are larger than a crucial minimum size. Denervation and neuronal loss are other factors in apoptosis as we age. Additionally, motor unit strength capacities decline. Strength capacities are decreased, muscle metabolism is slowed, and the danger of muscular damage is increased with muscle fiber loss. With aging, the rate of muscle protein synthesis declines. Age also results in decreased muscle healing abilities (17). Old-related declines in overall muscular strength may be due to a decreasing contribution from the progressively fewer big tensionproducing type II fibers (16). Due to the loss of type II fibers, older people have smaller and weaker muscle mass (16). A katabolic effect on muscles and bones is caused by the disproportionally high rate of atrophy of type IIa fast twitch (FT) muscle fibers and their motor units, as well as the reduction of anabolic hormones. Reduced levels of dehydroepiandrosterone, growth hormone, testosterone, and insulin-like growth factor-I as well as physical inactivity and an uptick in catabolic factors including interleukin-1, tumor necrosis factor (TNF), and interleukin-6 contribute to a decline in muscle mass and strength⁽¹⁷⁾. As a result, the same dosage of BTX-A utilized in the younger group and the older group did not provide the same effectiveness and duration due to changes in the physiology of the neuromuscular junctions and muscle with advancing age. It is in accordance with the findings of the study by Jing L. et al., which found that the dosage of BTX-A utilized in the older group was 45 to 150 U and the younger group was 30 to 200 U. Two groups significantly reduced pain, despite the older group's mean BTX-A doses that are somewhat more than those utilised by the younger age group. This suggests that both modest and large doses may have clear therapeutic effects (18). This result is in line with what Zhang et alinvestigation .'s found (19).

Conclusions

The physiology of neuromuscular junctions' changes with progress years, and there is a clear reduction in muscle mass and strength. The older group required larger BTX-A dosages than those used by the younger group to treat TMJ issues and relieve pain since the older group had a deterioration in the efficacy and duration of BTX-A when taken at the same dosage as the younger group.

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