Temporomandibular Joint Iontophoresis: A Double-Blind Randomized Clinical Trial

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This double-blind study evaluated the short-term effect of iontophoretic delivery of dexamethasone on the signs and symptoms of temporomandibular disorders in patients who had concurrent temporomandibular joint disc displacement without reduction and capsulitis. Twenty-seven patients with this clinical diagnosis were randomized to one of three groups: treatment group (dexamethasone sodium phosphate and lidocaine hydrochloride); control group (lidocaine hydrochloride); and placebo group (pH-buffered saline). Pretreatment and posttreatment data included items to calculate Helkimo's Anamnestic Dysfunction index, Helkimo's Clinical Dysfunction index, the Symptom Severity Index, and the Craniomandibular Index (CMI). The CMI is composed of the Dysfunction index (DI) and Muscle index. Analysis of variance showed no baseline differences on these measures between the three groups. Pretreatment and posttreatment values were compared with the paired t *tests. Posttreatment, the treatment group had an increased mean maximal active mandibular opening of 6 mm (*P = *.02), increased mean lateral excursion of 1.2 mm to the noninvolved side (*P = *.05), and reduced mean DI scores of 0.51 to 0.39 (*P = *.01); no statistically significant decrease in pain symptoms was reported. Analysis of variance showed a significant difference in the DI scores (*P = *.04) between groups from pretreatment to posttreatment, with the treatment group showing the greatest improvement in the DI scores relative to the other two groups. No other questionnaire items, exam items, or resultant indexes showed changes in any of the groups at* $P \leq .05$. *These results suggest that iontophoretic delivery of dexamethasone and lidocaine was effective in improving mandibular function, but not in reducing pain, in temporomandibular disorders patients who had concurrent temporomandibular joint capsulitis and disc displacement without reduction.*

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Temporomandibular disorders (TMD) in general includes both muscle and joint disorders. The most common temporomandibular joint (TMJ) disorders are capsulitis, disc displacement, and degenerative joint disease. These articular disorders may exist alone or in combinations and may also coexist with muscle disorders. Diagnostic criteria have been proposed to differentiate these disorders using historic and clinical parameters.[1–3] Validation of these criteria is an active area of TMD research.

Management goals for patients with symptomatic TMD are similar to those for patients with other orthopedic or rheumatologic disorders and include decreased pain, decreased adverse loading, restored function, and restored daily activity.[3] Conservative treatments such as medications, orthopedic appliances, physical medicine interventions (exercises and modalities), and cognitive behavioral interventions are endorsed for the initial treatment of nearly all patients with TMD.[3] However, there are few appropriately designed randomized clinical trials (RCTs) to evaluate physical medicine interventions for patients who have TMD.

Physical medicine interventions are standard therapies for most musculoskeletal disorders and, as such, seem appropriate for treatment of patients with TMD. Intra-articular injections of steroids are commonly used to treat clinically diagnosed inflammatory joint diseases. Iontophoresis is a battery-powered drug delivery system used to deliver water soluble ionizing drugs such as dexamethasone sodium phosphate and lidocaine hydrochloride through the skin. An animal study that used radiolabeled hydrocortisone sodium succinate reported that local tissue concentrations when iontophoresis was used were higher than those that would be obtained with systemic therapy and lower than those obtained by local injection.[4] The tissue drug level recorded in that study appeared to be clinically adequate, especially if the targeted anatomic structure was relatively superficial.[4] Similarly, dexamethasone may be transferred iontophoretically into tissues below the electrode. Since the local tissue steroid concentration is lower with iontophoresis than that found with injections, it seems logical that multiple treatments with iontophoresis of corticosteroids are needed to be comparable to a single injection. However, a systematic comparison of the ability of iontophoresis and joint injection to deliver steroids into the TMJ has not been achieved. Thus, it is not known whether these two treatment modalities are comparable.

Case reports, case series, and a clinical trial have suggested that iontophoretic delivery of anti-inflammatory medications is associated with a decrease in the signs and symptoms of clinically diagnosed capsulitis or tendonitis.[5–8] However, one RCT concluded that iontophoretically applied dexamethasone is no more effective than a saline placebo in providing pain relief or improvement in mandibular range of motion in patients with TMJ pain.[9] The purpose of this double-blind placebo controlled RCT was to determine the effect of iontophoretic delivery of dexamethasone sodium phosphate and/or lidocaine hydrochloride on the signs and symptoms of TMD in a more narrowly defined group of patients with concurrent TMJ capsulitis and disc displacement (DD) without reduction.

Materials and Methods

Study Sample

The study was reviewed and approved by the Committee on the Use of Human Subjects in Research, University of Minnesota, Minneapolis, MN. Twenty-seven consecutive consenting TMD patients presenting to the TMJ and Craniofacial Pain Clinic, University of Minnesota, for evaluation and treatment of *(1)* an abrupt inability to open their mouths to their normal distance and *(2)* concurrent TMJ/jaw pain were included in this trial. This included 24 females and three males with an mean age of 29 years (range 16 to 81 years), all with the clinical diagnosis of TMJ capsulitis and TMJ DD without reduction. Diagnostic criteria used in this study for TMJ DD without reduction were based on an adaptation of two sources: the recommendations, which have not been validated, of the American Academy of Craniomandibular Disorders[3]; as well as previously validated criteria by Schiffman et al.[2] The diagnostic criteria for TMJ capsulitis was based on the recommendations of the American Academy of Craniomandibular Disorders.[3] The adapted criteria for TMJ DD without reduction were

1. Pain precipitated by function (according to patient report)
2. Limited (active) mandibular opening (less than 40 mm, measured incisor to incisor plus vertical overlap of the incisors)
3. Deviation to the affected side on opening (greater than 2 mm)
4. Limited contralateral movements (less than 7 mm)
5. No joint noise with palpation or auscultation

The adapted criteria for capsulitis were

1. Point tenderness on palpation of the TMJ (2 lb total per scale calibration)
2. Pain at rest and exacerbated by function (according to patient report)
3. Range of motion limited by pain (less than 40 mm)

For a clinical diagnosis of TMJ DD without reduction, the first three criteria items and one of the two last items were required to be present. A

diagnosis of capsulitis required all three items to be met. In this study, patients presumably had limited active range of motion as a result of both pain and mechanical obstruction. At pretreatment, all patients reported pain in the area of the TMJ at rest, which was aggravated by eating. Clinically, at pretreatment, all patients reported increased pain with opening and with contralateral movements. All patients had various levels of concurrent muscle pain to palpation.

Exclusion criteria included presence of primary craniofacial pain disorders other than TMJ DD without reduction with capsulitis; taking prescription antidepressants, steroids, or narcotics; prior TMJ surgery; and pregnancy.

Study Design

The 27 patients were randomized to one of three groups: group 1, the treatment group (0.5 mL of 0.4% dexamethasone sodium phosphate and 1 mL of 4% lidocaine hydrochloride); group 2, the control group (1.5 mL of 4% lidocaine hydrochloride only); or group 3, the placebo group (1.5 mL of pH-buffered saline). The examiner, the physical therapist, and the patient were blind to the group assignment. All patients were asked to discontinue using any over-the-counter analgesics, prescription anti-inflammatories/analgesics, and muscle relaxants for the duration of the study. Use of intraoral appliances, jaw exercises, and/or self-administered heat or ice treatments were also discontinued in all groups. Although patients were instructed to discontinue treatments prior to study participation, no definitive time period was established during which potential carry-over effects of medications, splints, and other treatments were allowed to "wash out." No advice for identification or control of oral habits was suggested. Patients were instructed to eat foods that did not increase their symptoms. A clinical examination and a self-administered questionnaire were completed. Iontophoretic treatments were delivered using a Phoresor Model PM700 and Trans Q iontophoretic electrodes (IOMED, Salt Lake City, UT). The treatments were administered every other day for a total of three treatments according to the manufacturer's recommendations. The treatment electrode was placed unilaterally over the TMJ that had been diagnosed with TMJ disc displacement without reduction and with capsulitis; the ground electrode was placed over the ipsilateral trapezius muscle. For all three groups, the modal treatment time was 20 minutes. Dexamethasone sodium phosphate, and/or lidocaine hydrochloride or saline, was administered for a total of 40 mA-minutes per treatment; in each case, the current was adjusted to the patient's tolerance. One week after the last treatment was administered, patients were re-examined, and a self-administered questionnaire was again completed.

Assessment Instruments

The pretreatment and posttreatment data collected included a self-administered questionnaire and a clinical examination. The questionnaire contained items to calculate Helkimo's Anamnestic Dysfunction Index (A_i) and the Symptom Severity Index (SSI).[10–14] The A_i is a symptom checklist that assesses the subject's current symptoms associated with the stomatognathic system. The A_i ranges from symptom free (A_i0) through severe symptoms (A_iII). The SSI is composed of a symptom checklist and five visual analog scales (VAS). The symptom checklist was used to evaluate the scope of general somatic symptoms. The five VAS were used to assess sensory intensity, affective intensity, duration, frequency, and tolerability relative to the worst symptom from the A_i. The lowest value for sensory and affective intensity and tolerability is 0. The lowest value for frequency and duration is 1. The symptom checklist and the VAS combined to form a summary index, the SSI. The SSI ranges from 0 to 1, with 0 being the lowest value.

The pretreatment and posttreatment clinical examination included measurement of mandibular range of motion and asking the patients whether there was pain with these movements. Measurements of opening were done from the medial incisal edge of the maxillary right central incisor to the incisal edge of the opposing mandibular incisor. Vertical overlap of these teeth was also measured. All opening measurements in the results section include vertical overlap of the teeth. Noise from the TMJ was evaluated with palpation and auscultation using a stethoscope. Pain from palpation of the TMJ, of the muscles of mastication, and of the superficial neck musculature was assessed using 2 lb of pressure from the distal phalanx of the index finger. The amount of pressure applied to these structures was standardized with a scale. The specific technique for the exam has been reported previously.[15] All exams were done by one blinded examiner (ELS), and his reliability was consistent with a prior report.[15] From these clinical features, the Craniomandibular Index (CMI) and Helkimo's Clinical Dysfunction index (D_i) were calculated.[10,11,14,15] The CMI is the mean of

two subindexes: the Dysfunction index (DI) and the Muscle index (MI). The DI measures the level of jaw dysfunction, and the MI measures the number of muscle sites tender to palpation. The CMI, the DI, and the MI scales vary between 0 and 1, with 0 being the lowest value. The D_i is composed of five subindexes, and this categorical index varies between clinically symptom free (D_i0) through severe symptoms (D_iIII).

Data Analysis

The SSI and the DI of the CMI were considered the primary outcome measures. For continuous variables or indexes, analysis of variance (ANOVA) was used to evaluate statistical differences between groups. Pretreatment and posttreatment values within groups were compared using the paired *t* test. Ordered categorical data were analyzed with nonparametric statistics. Pretreatment and posttreatment differences within groups were compared using Wilcoxon's signed rank test, and between group differences were evaluated with the Kruskal-Wallis and Mann-Whitney rank sum test. Changes and presence of pain were evaluated between groups with chi square analysis. All tests were considered statistically significant at a level of .05. Trends were defined as $.05 < P < .10$.

Results

Analysis of pretreatment interventions including over-the-counter analgesics, prescription anti-inflammatories/analgesics, intraoral appliances, jaw exercises, and/or physical therapy treatments showed no statistically significant difference between groups for these interventions. Specifically, 11 subjects were using a splint (three subjects in group 1, three subjects in group 2, and five subjects in group 3); eight subjects were using anti-inflammatory medications on an as-needed basis, (two subjects in group 1, four subjects in group 2, and two subjects in group 3); five subjects were using physical therapy home interventions (one subject in group 1, one subject in group 2, and three subjects in group 3); one subject in group 2 was in counseling; and two subjects had tooth adjustments (one subject in group 2 and one subject in group 3). All interventions were implemented elsewhere prior to being seen by the authors and prior to the occurrence of the patient's limited mandibular range of motion (ROM).

Questionnaire

There were no statistically significant baseline differences in Helkimo's A_i or the SSI between groups. No significant pretreatment to posttreat-

Table 1 Change in Symptom Severity Index (SSI) and Subgroup Values From Pretreatment to Posttreatment*

	Group 1 (n = 9)			Group 2 (n = 9)			Group 3 (n = 9)		
	Pre mean (SD)	Post mean (SD)	*P*	Pre mean (SD)	Post mean (SD)	*P*	Pre mean (SD)	Post mean (SD)	*P*
Scope of symptoms	0.22 (0.07)	0.16 (0.07)	.06	0.18 (0.06)	0.16 (0.08)	.6	0.18 (0.08)	0.20 (0.1)	.3
Intensity	0.56 (0.14)	0.47 (0.20)	.2	0.51 (0.16)	0.43 (0.19)	.3	0.46 (0.16)	0.45 (0.17)	.8
Affective intensity	0.61 (0.17)	0.46 (0.22)	.09	0.44 (0.22)	0.44 (0.18)	.9	0.50 (0.22)	0.45 (0.20)	.3
Difficulty to endure	0.51 (0.26)	0.45 (0.27)	.3	0.48 (0.16)	0.41 (0.19)	.3	0.39 (0.25)	0.39 (0.20)	1.0
Frequency	0.19 (0.19)	0.29 (0.31)	.4	0.23 (0.15)	0.32 (0.14)	.1	0.13 (0.10)	0.14 (0.12)	.7
Duration	0.30 (0.33)	0.43 (0.37)	.1	0.52 (0.44)	0.72 (0.31)	.1	0.31 (0.40)	0.32 (0.39)	1.0
Total SSI	0.57 (0.1)	0.47 (0.2)	.09	0.48 (0.1)	0.40 (0.1)	.07	0.52 (0.2)	0.50 (0.2)	.8

*Group 1 = dexamethasone sodium phosphate and lidocaine hydrochloride; Group 2 = lidocaine hydrochloride; Group 3 = pH-buffered saline; Pre = pretreatment; Post = postreatment.

ment differences within groups were found with these indexes. All subjects had symptoms classifiable as A_1II (severe symptoms) at baseline; 26 subjects had A_1II at posttreatment. Trends suggesting pain reduction were found in affective intensity ($P \leq .06$) and scope of symptoms ($P \leq .09$) for group 1, and a trend was found for decreased SSI for groups 1 ($P \leq .09$) and 2 ($P \leq .07$) (Table 1).

Examination

The only baseline difference found between groups was less palpable muscle pain in group 2 compared to the other two groups when D_1 was used. Range of motion measures are summarized in Table 2. Statistically significant improvements from pretreatment to posttreatment in active opening and contralateral movements were found only in group 1 (treatment). Relative to the CMI, a statistically significant decrease (improvement) posttreatment was noted in the DI for group 1 (Table 3). The ANOVA showed a statistically significant difference between groups from pretreatment to posttreatment in the DI scores ($P = .04$) with group 1 showing a greater improvement in the DI relative to the other two groups. Prior to treatment, all subjects had pain with active opening and with contralateral movements. Table 4 shows the direction of change of mobility and presence of pain with maximum active opening and with contralateral movements at posttreatment. Although not statistically significant, a greater percentage of subjects in group 1 (treatment) had increased and/or pain-free mandibular movements relative to groups 2 (control) and 3 (placebo). No other individual exam items, including joint pain from palpation and joint noise, showed a statistically significant change from pretreatment to posttreatment.

The only side effects from treatment were erythema on the skin under the electrode and transient report of dizziness during treatment. The dizziness resolved when the power source was turned off; the erythema resolved within 8 hours.

Table 2 Change in Mobility (mm) From Pretreatment to Posttreatment*

	Group 1 (n = 9)			Group 2 (n = 9)			Group 3 (n = 9)		
	Pre mean (SD)	Post mean (SD)	P	Pre mean (SD)	Post mean (SD)	P	Pre mean (SD)	Post mean (SD)	P
Active†	32.2 (6.5)	38.2 (10.2)	.02	36.3 (9.8)	38.3 (5.7)	NS	34.0 (7.8)	36.3 (5.6)	NS
Passive†	37.7 (7.4)	39.8 (9.4)	NS	41.9 (6.4)	43.6 (5.1)	NS	39.9 (7.3)	42 (7.0)	NS
Contralateral	6.2 (3.1)	7.4 (3.5)	.05	6.8 (2.6)	7.4 (1.9)	NS	8.7 (2.1)	9.4 (2.2)	NS

*Group 1 = dexamethasone sodium phosphate and lidocaine hydrochloride; Group 2 = lidocaine hydrochloride; Group 3 = pH-buffered saline; Pre = pretreatment; Post = postreatment.
†Incisor-to-incisor opening plus vertical overlap of incisors.

Table 3 Change in CMI, DI, and MI Values From Pretreatment to Posttreatment†

	Group 1 (n = 9)		Group 2 (n = 9)		Group 3 (n = 9)	
	Pre mean (SD)	Post mean (SD)	Pre mean (SD)	Post mean (SD)P	Pre mean (SD)	Post mean (SD)
DI	0.51 (.01)	0.39 (0.1)*	0.47 (0.1)	0.40 (0.1)	0.44 (0.1)	0.47 (0.1)
MI	0.43 (0.3)	0.40 (0.3)	0.28 (0.2)	0.22 (0.1)	0.44 (0.3)	0.38 (0.2)
CMI	0.47 (0.2)	0.39 (0.2)	0.38 (0.1)	0.31 (0.1)	0.44 (0.1)	0.43 (0.1)

*$P < .01$.
†*Group 1 = dexamethasone sodium phosphate and lidocaine hydrochloride; Group 2 = lidocaine hydrochloride; Group 3 = pH-buffered saline; Pre = pretreatment; Post = postreatment.

Table 4 Change and Presence of Pain With Maximum Active Opening (Open) and Contralateral Movements (CLM): Posttreatment Status

	Group 1 (n = 9)		Group 2 (n = 9)		Group 3 (n = 9)	
	Open	CLM	Open	CLM	Open	CLM
Increased movement	7	7	4	4	5	4
Decreased movement	1	1	3	2	4	2
No change	1	1	2	3	0	3
Painfree	3	6	1	4	0	2

Discussion

Case series have suggested positive clinical treatment effects with intra-articular injections of corticosteroids for TMD.[16,17] Two randomized comparative studies have shown that when betamethasone is injected into the TMJ, it is as efficacious as sodium hyaluronate in reducing the signs and symptoms of TMD, both short term and long term.[18,19] The lack of a control in these studies leaves unanswered whether either treatment is superior to no treatment or a placebo. There are no studies comparing injection versus iontophoretic delivery of corticosteroids, so it is unknown whether either delivery system is superior subjectively or objectively.

There are case reports and case series that have suggested a positive treatment effect with iontophoresis of corticosteroids for TMD.[7,8] One randomized clinical trial in the physical therapy literature has shown iontophoresis of dexamethasone sodium phosphate and lidocaine hydrochloride to be superior to a placebo for treating shoulder tendonitis.[6] In the TMD literature, Reid et al[9] reported that iontophoretically applied dexamethasone with lidocaine was no more effective than a saline placebo in providing pain relief or improvement in range of motion in patients with TMJ pain, when the most symptomatic joint was treated. The patients in the Reid et al study had a variety of TMJ diagnoses; however, when subgroup analysis was done, there was a trend noted for pain reduction in patients with a diagnosis of osteoarthritis, but no trends for patients with a diagnosis of acute or chronic DD without reduction (15 subjects with DD without reduction of a total of 53 subjects). Approximately half of the patients with DD without reduction were diagnosed with either acute or chronic DD (Reid K, e-mail communication, 1995). Thus, the overall patient populations between that study and the present study are difficult to compare. Furthermore, the methodologic differences between these two studies are significant. The present study's inclusion criteria required patients to have increased TMJ pain with active opening, contralateral movements, and TMJ palpation; Reid et al required only increased pain with TMJ palpation and passive stretch (Reid K, personal communication, 1995). Reid et al also did not analyze change in contralateral movements from the symptomatic/treated TMJ, but rather analyzed change in lateral movements without regard to the treated side (Reid K, e-mail communication, 1995). Finally, Reid et al limited pain evaluations to changes in pain intensity on a 100-mm VAS. Nonetheless, limited comparisons between these two studies are warranted. Both studies showed no statistically significant decrease in pain intensity following treatment in any group, although both studies did find trends for reduction in pain intensity for the patients treated with dexamethasone and lidocaine. In addition, the present study found trends for reduction in scope of symptoms for group 1 and a trend for decreased SSI for groups 1 and 2. Relative to range of motion parameters, only the present study found in the treatment group a statistically significant increase in active opening and in contralateral movements relative to the treated joint. These two studies differ in that the present study used additional outcome measures including the DI of the CMI, and it was the DI that showed significant improvement for the treatment group. The DI does not specifically measure TMJ dysfunction but rather mandibular dysfunction related to any masticatory structure.[20] Since iontophoresis enables the steroid to go beyond the joint into adjacent tissues,[4] it potentially can affect any structure involved in mandibular function (including the TMJ and adjacent structures) as well as pain with movement of these structures. In short, iontophoresis has an apparent overall effect on mandibular function, and focus-

ing on limited outcome measures and ignoring the summary measures loses this overall effect.

Although the present study found a statistically significant increase in the subjects' active ROM and a significant decrease in the DI, is this improvement clinically significant? As Table 4 shows, only in the treatment group did a majority of the subjects show an improvement with opening, contralateral movements, and elimination of pain with contralateral movements, which strengthens the clinical significance of the statistically significant findings. These results in total suggest that iontophoretic delivery of dexamethasone sodium phosphate and lidocaine hydrochloride is effective in improving mandibular function and that these results are clinically significant.

The fact that the MI and thus the CMI did not significantly change with treatment suggests that these treatments do not have a significant effect on the number of masticatory muscles tender to palpation. Use of a pressure algometer would have been useful to evaluate if treatment affects the level of pressure needed to lead to a report of pain. Finally, if secondary muscle splinting existed throughout the masticatory muscles, this may have further influenced the final ROM.

In the present study, the presumed targeted tissue was the TMJ capsule and adjacent structures. Subjects had signs and symptoms that included both pain and mechanical factors. However, the presence of an inflammatory condition in the capsule and adjacent structures of all subjects in this study is speculative. Studies have shown the presence of inflammatory mediators including prostaglandin and leukotrienes in symptomatic TMJs[21,22]; however, the relevance of these mediators is dependent on showing that they are either absent or present at a significantly lower level in asymptomatic subjects relative to symptomatic subjects. To date, these comparisons have not been reported, and therefore, there is no objective method to detect a clinically significant intra-articular inflammatory process. Since the response of the patient to steroids is theoretically dependent on an inflammatory process being present, the results in this study may have varied relative to the presence or absence of inflammatory mediators in the subjects' TMJs.

Although all patients were asked to discontinue using over-the-counter analgesics, prescription anti-inflammatories/analgesics, muscle relaxants, intraoral appliances, jaw exercises, and/or self-administered heat or ice treatments for the duration of the study, no specific wash-out period was used prior to this study for the potential therapeutic carry-over effect of any of these interventions. However, analysis of these pretreatment interventions showed no statistically significant difference between groups for these interventions. Furthermore, a randomized design was used in this study, so theoretically, all groups were equivalent for both known or unknown prognostic factors.

Additional factors that may have affected the outcome include whether three treatments is an appropriate number of treatments; if bilateral TMJ treatments would have had a different outcome, since the two joints are physically connected and are not independent; and whether a DD without reduction is an appropriate model. The latter factor is important, since this condition is characterized by mechanical limitations that presumably would not be affected by steroids. Thus, limitations with the ROM of the TMJs would only be improved relative to the control of the inflammatory component and its subsequent effect on pain during movement. Persistent mechanical limitations (ie, a nonreducing disc) could also perpetuate pain through mechanical stimulation of nociceptive receptors in the TMJ capsule. Perhaps a better model would have been TMJ capsulitis without concurrent mechanical limitations. Disc displacement without reduction with limited ROM was used in this study because it appeared to be associated with the most significant level of capsulitis clinically, and thus presumably would have the best potential to show a significant response of both signs and symptoms with treatment. However, in our pain clinic, patients who have DD without reduction with limited ROM and concurrent capsulitis are usually treated with a combination of iontophoresis and mobilization. Mobilization of the TMJ may be a very significant part of the intervention, since it directly addresses the mechanical component of DD without reduction. A study on other stages of TMJ DD with capsulitis with limited pain-free active openings using six iontophoresis treatments allowing either unilateral or bilateral treatments (depending on the patients' symptoms) is ongoing, and a study with DD without reduction using iontophoresis with and without mobilization is planned in the future so these factors can be evaluated.

A larger issue that future research needs to address is that, clinically, it is common for patients to be treated with a number of different physical, behavioral, and psychosocial interventions. It may be that by isolating individual treatment strategies, their significance is minimized, because multiple interventions work synergistically and the total becomes more than its parts.[23]

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Resumen

Iontoforesis de la Articulación Temporomandibular: Estudio Clínico al Azar y Doble-Ciego

Se ha reportado que la liberación iontoforética de corticosteroides es un tratamiento para reducir el dolor mandibular y mejorar la función mandibular. El propósito de este estudio fue el de evaluar el efecto a corto plazo de la liberación iontoforética de dexametasona sobre los signos y síntomas de desórdenes temporomandibulares en pacientes que sufrían de desplazamiento del disco de la articulación temporomandibular sin reducción y de capsulitis, al mismo tiempo. En este estudio al doble-ciego, participaron 27 pacientes con este diagnóstico clínico. Los pacientes fueron colocados (al azar) en uno de los siguientes grupos: *(1)* grupo de tratamiento (fosfato sódico de dexametasona e hidrocloruro de lidocaína; *(2)* grupo de control (hidrocloruro de lidocaína); y *(3)* grupo de placebo (solución salina tamponizada). Se administraron tres tratamientos iontoforéticos un día sí y otro no utilizando electrodos Phoresor Modelo PM 700 y Trans Q. Una semana después del último tratamiento, los pacientes fueron examinados nuevamente. La información antes y después del tratamiento incluyó datos para calcular el Indice Anamnéstico de Helkimo, el Indice de Disfunción Clínica de Helkimo, el Indice de Severidad Sintomática, y el Indice Craneomandibular (ICM). El ICM está compuesto del Indice de Disfunción (ID) y el Indice Muscular. El análisis de varianza no mostró diferencias en las medidas del examen inicial, entre los tres grupos. Los valores antes y después del tratamiento fueron comparados con los exámenes *t* apareados. Después de la terapia, el grupo de tratamiento mostró un aumento en la apertura mandibular activa máxima media, de 6 mm ($P = 0,02$), un aumento en las excursiones laterales medias de 1,2 mm en el lado no afectado ($P = 0,05$), y unos valores medios reducidos en el ID de 0,51 a 0,39 ($P = 0,01$); no se registró una reducción estadísticamente significativa en los síntomas de dolor. El análisis de varianza mostró una diferencia significativa en los valores del ID ($P = 0,04$) entre los grupos desde antes hasta después del tratamiento, siendo el grupo de tratamiento el que mostró la mejoría mas alta en relación a los valores del ID en comparación con los otros dos grupos. Ningún otro cuestionario, examen o índice mostró cambios en los otros grupos ($P \leq 0,05$). Estos resultados indican que la liberación iontoforética de dexametasona y lidocaína fue efectiva en la mejoría de la función mandibular, pero no en la reducción del dolor, en pacientes con desórdenes temporomandibulares que tenían capsulitis de la articulación temporomandibular y desplazamiento del disco sin reducción, al mismo tiempo.

Zusammenfassung

Kiefergelenksiontophorese: Ein randomisierter klinischer Doppelblindversuch

Es ist schon verschiedentlich über die Applikation von Kortikosteroiden durch Iontophorese zur Behandlung von Myoarthropathien berichtet worden. Der Zweck dieser Studie war die Evaluation der Kurzzeiteffekte der Iontophorese mit Dexamethason auf die Zeichen und Symptome von Myoarthropathien bei Patienten mit Diskusluxation ohne Reduktion und gleichzeitiger Capsulitis. Bei dieser Doppelblindstudie wurden 27 Patienten mit obigen Diagnosen randomisiert zu einer der folgenden 3 Gruppen zugeteilt: *(1)* behandelte Gruppe (Dexamethason mit Lidocain), *(2)* Kontrollgruppe (Lidocain) und *(3)* Plazebogruppe (Kochsalzlösung). Es wurden 3 Iontophorese-Sitzungen unter Benutzung eines Phoresor Model MP700 und von Trans Q-Elektroden durchgeführt. Eine Woche nach der letzten Behandlung wurden die Patienten wieder untersucht. Die Daten vor und nach der Behandlung wurden zur Berechnung des Anamnese-Indexes nach Helkimo, des klinischen Dysfunktionsindexes nach Helkimo, des Symptom-Schweregrad-Indexes, und des "Craniomandibular Index (CMI)" verwendet. Der CMI setzt sich aus dem Dysfunktionsindex (DI) und dem Muskelindex zusammen. Die Varianzanalyse zeigte keine Grunddifferenzen dieser drei Messungen zwischen den 3 Gruppen. Die Werte vor und nach der Behandlung wurden mittels des *t*-Tests verglichen. Nach der Behandlung wies die Gruppe mit Dexamethason/Lidocain durchschnittlich eine Verbesserung der maximalen aktiven Mundöffnung von 6 mm ($P = 0.02$) auf, eine Verbesserung der Laterotrusionen um 1.2 mm im Vergleich zur nichtbetroffenen Seite ($P = 0.05$) und verringerte DI-Werte von 0.51 bis 0.39 ($P = 0.01$). Es wurde keine statistisch signifikante Verringerung von Schmerzsymptomen gefunden. Die Varianzanalyse zeigte eine signifikante Differenz der DI-Werte ($P = 0.04$) vor und nach Behandlung, wobei die grösste Verbesserung bei der Gruppe mit Dexamethason/Lidocain auftrat. Bei einem $P \leq 0.05$ zeigte keine der anderen Untersuchungsresultate eine Veränderung bei einer der 3 Gruppen. Diese Ergebnisse lassen vermuten, dass die iontophoretische Verabreichung von Dexamethason/Lidocain bei den oben geschilderten Patienten zwar effektiv bei der Verbesserung der Unterkieferbeweglichkeit half, aber keine Schmerzreduktion zur Folge hatte.