http://dx.doi.org/10.17159/2519-0105/2019/v75no8a7

What's new for the clinician?
- Excerpts from and summaries of recently published papers

SADJ September 2020, Vol. 75 No. 8 p454 - p458

Compiled and edited by V Yengopal

1. Conservative therapies to treat pain and anxiety associated with temporomandibular disorders - a randomized clinical trial

RA Melo, CM de Resende, CR Rêgo, et al. Conservative therapies to treat pain and anxiety associated with temporomandibular disorders: a randomized clinical trial. International Dental Journal 2020; 70: 245-53.

INTRODUCTION

Temporomandibular joint dysfunction (TMD) is a term used for disorders affecting the muscles of mastication and the temporomandibular joints (TMJ), including the mandibular condyles, fossa of the temporal bone, TMJ capsule and the articular disc.[1] TMD is characterized by pain in the TMJ and periauricular region, and/or the muscles of mastication.

Signs and symptoms include\limited mouth opening, restricted/asymmetric mandibular movement, and TMJ noise.[1] TMD can cause a clicking/popping noise emanating from the TMJ when mandibular movement occurs and the condyle crosses the rear margin of the articular disc. It is estimated that the prevalence of TMD in the world population is between 5% and 12%, although only about 2% require some intervention or treatment.

The aetiology of TMDs is complex and multifactorial.[1] Among the factors that increase the risk of the disease, starting or even accentuating the progression of the pain are: physical factors such as trauma, sources of deep pain, parafunctional habits, occlusal condition, postural characteristics, muscular hyperactivity; neuromuscular factors; and psychosocial factors, such as socioeconomic conditions, sleep disturbances, anxiety and depression. Patients with temporomandibular disorders, especially those with chronic pain, may present secondary psychiatric disorders such as anxiety, depression, social phobia, reduced capacity to work, as well as isolation, and suffering from loss of concentration and self-confidence.[1]

Veerasamy Yengopal: *BChD, BScHons, MChD, PhD*, Community Dentistry Department, School of Oral Health Sciences, University of Witwatersrand, Medical School, no. 7 York Road, Parktown 2193, South Africa.
ORCID Number: 0000-0003-4284-3367
Email: veerasamy.yengopal@wits.ac.za

Due to the multiplicity of factors associated with TMD, many treatment options have been proposed but it is widely agreed that initial treatment should be conservative and reversible, aimed primarily at pain relief, restoration of normal function, and the patient's physical and mental well-being.[1]

Conservative treatment options include using an occlusal splint (OS) which has been widely used to restore neuromuscular balance through the return of balanced occlusal contacts, repositioning of the condyle and muscle relaxation. This consists of a removable device made of thermo-polymerisable acrylic resin that can be used during the day or at night depending on the clinical situation. Manual therapy (MT) has also been used to restore normal range of motion, reduce local ischaemia, stimulate proprioception, break fibrous adhesions, stimulate synovial fluid production, and reduce pain.[1] Individualised counselling (CS) is another option that has been shown to significantly improve signs and symptoms of TMD.

Melo and colleagues (2020)[1] reported on a trial that sought to evaluate the effectiveness of treatments with occlusal splint (OS), Manual therapy (MT), Individualised counselling (CS), and the association of OS with CS (OSCS) within the pain and anxiety variables in TMD patients after 1 month of treatment. The expected hypotheses were that, irrespective of the treated group, there would be a reduction in pain and anxiety with 1 month of treatment, and that patients treated with CS associated with OS would present less pain and anxiety when compared with patients who received single therapies.

MATERIALS AND METHODS

A blinded randomised clinical trial was conducted in which the evaluating investigator was not aware of the therapy to which the patient was submitted. Initially, 300

patients were screened, but 188 patients were excluded because they did not meet the inclusion criteria and 23 patients withdrew. Thus, the convenience sample consisted of 89 patients diagnosed with TMD. Thus, after some sample losses and non-completion of the questionnaires by all patients, 89 patients were evaluated using TMJ diagnostic criteria (RDC), 85 patients by VAS, 83 patients by Hospital Anxiety and Depression Scale (HADS), 88 patients by Beck Anxiety Inventory (BAI), and 87 and 89 by STAI-trait and state, respectively.

The sample was randomly divided into four groups: OS; MT; OSCS; and CS and patients were evaluated after one month. Individuals who abandoned the selected treatment or did not follow guidelines and recommendations, such as inadequate use of OS, absence of the MT sessions or even having taken any measurement that could influence therapy outcomes were excluded from the study. Patients who did not improve and were unable to remain in the initial group went through a waiting period of 3 months without receiving treatment, followed by a change to a new therapy.

The study included patients with a diagnosis of TMD who had not received any treatment for TMD in the last 3 months, had a report of pain in the orofacial region in the last 3 months, and who were between 18 and 65 years of age. Patients who were identified with some impairment of cognitive ability were excluded, as they were unable to understand the questions in the questionnaires; a history of head trauma that is related to the aetiology of orofacial pain; patients with intracranial disorders or headache; use of medications in the last 3 months that could interfere with the effect of tested therapies, such as muscle relaxants, anti-inflammatory medication, anticonvulsants, antidepressants and anxiolytics; use of medication to treat TMD or muscle pain during the research period; other causes of orofacial pain such as caries, periodontal diseases, or neuropathies and fibromyalgia.

The occlusal splints were made from thermo-polymerisable acrylic resin. At the appointment to deliver the splint, adjustments were made before patient was sent away with instructions on how long to use splints (either during the day or night or both). The first return occurred 15 days after the installation, for verification of the adaptation of the splint, adjustments and reinforcement of the advice, for the association group (OSCS). After 30 days of installation, the splints were readjusted, if necessary. At this evaluation, the diagnostic criteria for TMD and the questionnaires were administered.

The Manual Therapy (MT) applied in this study was based on the use of thermal agents (heat and cryotherapy) and therapeutic exercises that were performed clinically by a trained researcher. The therapeutic regimen consisted of 40-min sessions, performed twice a week for 4 weeks. Patients were also instructed to repeat at home, on a daily basis, all the procedures that were applied during the sessions, as noted below.

All treated patients, regardless of their diagnoses, were instructed to apply a gel packet at temperatures between 40°C and 50°C for 20 min, three times a day during the 4 weeks of treatment. The compresses were applied in the masseter, temporal and TMJ regions.

The therapeutic exercises used were masseter and temporal massage and stretching exercises for the jaw muscles. For counselling (CS), an investigation was made into habits and other factors that might be responsible for the aetiology of the patient's dysfunction, and then a series of orientated guidelines for each case were developed that individualize treatment according to personal needs.

In addition, general characteristics about the disease were clarified, so that patients understood their condition and felt able to manage it themselves. At the end of the consultation, the patient received a written booklet with dietary guidelines, physical exercises, deleterious habits, instructions on correct mandibular function, posture and sleep hygiene. After 15 days, a new appointment was arranged for reinforcement of the CS.

The instruments used to measure the variables were TMD diagnostic criteria (RDC/TMD), visual analogue pain scale (VAS), HADS, BAI and State-Trait Anxiety Inventory (STAI). These instruments were administered at baseline and after 1 month of treatment.

The VAS consisted of a graded visual scale from 0 to 10, where 0 means no pain at the moment and 10 is the worst pain imaginable. The HADS consisted of 14 questions about how the patient felt in the last week. Of these, seven include characteristics focused on anxiety symptoms and seven assess symptoms of depression. For each question there are four possible answers, which have a score from 0 to 3, totalling a maximum score of 21 points for each component of the questionnaire. Scoring classifies anxiety as normal (0-7) or mild to severe (8-21).

The BAI consists of 21 items and has questions that can be answered on a scale of 0 to 3 (absolutely not; lightly; moderately and severely). The score is given by the sum of the items and classifies anxiety into the following: minimum anxiety (0-7); mild to severe anxiety (8-63).

The STAI consists of two self-administered questionnaires that separately evaluate trait anxiety, which is considered a personality trait of the individual; and state anxiety, which occurs momentarily in the face of some specific stimulus. The results of the questionnaire responses are classified as mild anxiety (20-0), moderate anxiety (31-49), and severe anxiety (50-80).

RESULTS

Of the 89 participants, 5.61% had muscular TMD, while 6.73% patients had joint TMD, and 87.62% had mixed TMD.

In relation to the diagnosis of TMD at the 30-day evaluation, three patients from the OS group, three from MT and three from OSCS were diagnosed without TMD, whereas only one patient from the CS group

reached this result. In addition, from the 43 patients diagnosed with the worst prognosis (mixed TMD - both muscular and joint), only 18 remained with this condition.

There was a significant reduction in the pain variable, measured by the VAS, for all groups after 1 month of treatment. When comparing the different groups there was no significant difference between treatments in regard to reduction of pain. Thus, no group was better than another group in improving pain ($p = 0.260$).

The evaluation of anxiety using the Hospital Anxiety and Depression Scale (HADS) questionnaire showed that there was a reduction in anxiety symptoms for all groups, but no statistical difference was observed between them ($p = 0.260$). However, over time, all treatments resulted in a significant reduction of anxiety ($p < 0.001$).

The BAI questionnaire assessing anxiety showed that all four treatment groups achieved a significant reduction in anxiety symptoms over time ($p < 0.001$), comparing baseline time with 1 month of treatment. In addition, all groups presented similar therapeutic results in BAI-measured anxiety; therefore, there was no significant statistical difference between the four therapies groups (p=0.532). Thus, no group was better than another group in improving anxiety.

Similarly, in the evaluation of the state-trait anxiety measured by the STAI, there was a significant reduction in scores for all treatment groups; however, no significant statistical difference was found between the different groups ($p = 0.546$).

CONCLUSION

All of the conservative therapies used were effective in reducing pain and anxiety in patients diagnosed with TMD. However, no treatment was superior to the other in reducing the studied variables.

Implications for practice

TMD treatment and management has a number of conservative non-surgical options that appear to be effective. Clinicians should consider these treatment options before more radical approaches are considered for the management of TMD.

Reference

1. Melo RA, de Resende CM, Rêgo CR, Bispo AD, Barbosa GA, de Almeida EO. Conservative therapies to treat pain and anxiety associated with temporomandibular disorders: a randomized clinical trial. International Dental Journal. 2020; 70: 245-53.

2. The effects of vaping electronic cigarettes on periodontitis

F Karaaslan, A Dikilitaş, U Yiğit. The effects of vaping electronic cigarettes on periodontitis. Australian Dental Journal. 2020; 65: 143-9.

INTRODUCTION

Periodontitis is a group of inflammatory diseases that affect the connective tissue attachment and supporting bone around the teeth.[1] It is widely accepted that the initiation and the progression of periodontitis are dependent on the presence of virulent microorganisms capable of causing disease. Although the bacteria are initiating agents in periodontitis, the host response to the pathogenic infection is critical to disease progression.

Cytokines are defined as low molecular weight proteins produced by one cell acting on another cell within the same perimeter.[1] Cytokines underpin the immune cells and periodontal tissue cells for orchestrating periodontitis and propagating the inflammatory process after bacterial invasion.[1] Interleukin-8 (IL-8) and tumour necrosis factor-α (TNF-α) are important cytokines reported to have higher gingival crevicular fluid (GCF) levels in periodontitis patients.[1]

Oxidative stress is an inflammatory process defined as an imbalance between excessive reactive oxygen species production and antioxidant mechanisms.[1] Increased oxidative stress has been associated with the pathogenesis of periodontitis in a rapidly growing body of research. The most commonly-used stable product for evaluating oxidative DNA damage is 8-hydroxydeoxyguanosine (8-OHdG), and its relationship to periodontitis has been shown.[1] Glutathione peroxidase (GSH-Px) also has an important role in human defence against oxidative stress, which has been reported in the GCF of periodontitis patients.[1]

Smoking traditional cigarettes (T-cigs) is well-established as a major risk factor for periodontitis, increasing the risk two to fivefold. It is well-accepted that smoking changes the host's immune response through mechanisms that include the disruption of cytokine and inflammatory mediator production, impairment of gingival vascular function and creating a source of oxidative stress.[1]

In recent years, inhaling the vapours of electronic cigarettes (E-cigs) has been gaining popularity among individuals who want to reduce or stop tobacco smoking. Although the use of E-cigs is escalating, there is limited information available regarding the impact of vaping E-cigs on periodontal health. Karaaslan and colleagues reported on a clinical trial that sought to compare the effects of smoking T-cigs, vaping E-cigs and smoking

cessation on GCF levels of and tumour necrosis factor -α (TNF-α), Interleukin-8 (IL-8), 8-hydroxydeoxyguanosine (8-OHdG), Glutathione peroxidase (GSH-Px) and clinical periodontal parameters in patients with periodontitis. It was hypothesized that vaping E-cigs produces fewer harmful effects on clinical and biochemical parameters of periodontitis, compared with smoking tobacco.

MATERIALS AND METHODS

The study consisted of two parts: a clinical examination and gingival crevicular fluid (GCF) sampling of a total of 57 individuals aged between 29 and 39 years.

For enrolment, participants met the following inclusion criteria:

i). Individuals diagnosed with periodontitis.
ii). T-cig smokers: those who had smoked for at least 10 years and a minimum of 10 cigarettes per day.
iii). E-cig vapers: participants who were ex-smokers having smoked more than 10 T-cigs/day for at least 10 years, and had been vaping E-cigs for at least 12 months.
iv). Former smokers: those who had smoked more than 10 T-cigs/day for at least 10 years in their lifetime and who currently had not been smoking for at least 12 months.

Exclusion criteria included:

i). Dual-smoking patients (use of both T-cigs and E-cigs). Cigar, pipe and waterpipe smokers.
ii). Diabetics.
iii). Non-smokers.
iv). Patients with any disease that can affect periodontal health.
v). Patients who had received any periodontal treatment in the last 6 months.
vi). Alcohol consumers.
vii). Patients who had taken any drugs which can affect periodontal tissues, such as antibiotics and non-steroidal anti-inflammatory drugs, within the past 6 months.

Participants included according to the above criteria were divided into three groups:

Group I: T-cig smoker periodontitis group consisted of 19 patients.
Group II: E-cig vaping periodontitis group consisted of 19 vaper patients.
Group III: Former smoker periodontitis group consisted of 19 individuals who had quit smoking at least 12 months previously.

The clinical examination of patients assessed plaque index (PI), gingival index (GI), probing depth (PD) and clinical attachment loss (AL). All clinical parameters were measured with a Williams periodontal probe (Hu-Friedy).

Clinical periodontal measurements were obtained from six points around each tooth except third molars. The patients were diagnosed as having periodontitis under these criteria: their interdental AL was detectable at ≥2 non-adjacent teeth; their buccal or oral AL was ≥3 mm with pocketing >3 mm detectable at ≥2 teeth and the observed AL could not be attributed to non-periodontitis causes.

Gingival crevicular fluid (GCF) samples were collected one day after the periodontal clinical measurements. Samples were taken from the two deepest pockets in each quadrant, including four maxilla and four mandibular sites, using paper strips (Periopaper) at a similar time of day for each patient.

These strips were inserted into the crevice, not more than 1-2 mm, for 30 s. Strips contaminated with blood were discarded. GCF volume was determined by a calibrated Periotron 8000, and readings were converted to an actual volume (μL) by reference to a standard curve. The samples were stored in a microcentrifuge tube for further analysis.

Samples were assessed in duplicate wells and concentrations were estimated. The mean concentration of each marker was calculated, adjusted to GCF volume and expressed as picograms per millilitre. All clinical and biochemical parameters were compared using one-way ANOVA test.

RESULTS

A total of 57 patients, 39 (68.4%) male and 18 (31.6%) female, were included in the study. The mean age of all participants was 35.19 ± 2.23 years ranging from 29 to 39.

In Group I, the mean number of T-cigs smoked per day was 13.68 ± 3.67 and the mean years of smoking T-cigs was 13.95 ± 3.01. In Group II, the mean years of vaping E-cigs was 2.32 ± 0.75, the mean years of smoking T-cigs before changing to E-cigs was 12.11 ± 1.52, and the mean number of T-cigs smoked per day before changing to vaping E-cigs was 12.89 ± 2.54. In Group III, the mean years of non-smoking was 2.41 ± 0.95, the mean years of smoking T-cigs before quitting was 12.11 ± 1.70, and the mean number of T-cigs smoked per day before quitting was 12.11 ± 2.54.

When comparing the mean number of T-cigs smoked per day between Group I, Group II (when smoking T-cigs before changing to E-cigs), and Group III (T-cigs smoked before quitting), no statistically significant difference was found ($p > 0.05$).

Although there was no statistically significant difference between Group II and Group III, in terms of mean years of smoking T-cigs before vaping and before quitting, respectively, the mean years of smoking T-cigs of Group I was statistically higher than Groups II and III ($p < 0.05$). In addition, there was no significant difference between mean years of vaping E-cigs (Group II) and mean years of non-smoking (Group III) ($p > 0.05$).

Although there were no significant differences among the groups for mean attachment loss (AL), probing depth (PD), and plaque index (PI) ($p > 0.05$), the mean (gingival index (GI) score of Group I (1.53 ± 0.29) was

significantly lower than in both Groups II (1.81 ± 0.30) and III (2.08 ± 0.35), and the mean GI score of Group II was significantly lower than Group III's.

There was a significant difference among the groups for mean GCF volume. Group I's (1.63 ± 0.04) was significantly lower than that of both Group II (1.82 ± 0.03) and Group III (1.95 ± 0.08). In addition, the mean GCF volume of Group II was significantly lower than Group III's.

There was a significant difference among the three groups for mean IL-8 levels. The mean IL-8 level of Group I (70.47 ± 2.76) was significantly lower than those of Group II (77.11 ± 2.38) and Group III (80.11 ± 3.41). The mean IL-8 level of Group II was also significantly lower than that of Group III.

There was a significant difference among the three groups for mean TNF-α level. The mean TNF-α level of Group I (4.20 ± 0.14) was significantly higher than those of Group II (3.41 ± 0.21) and Group III (2.98 ± 0.11), and Group II's level was significantly higher than Group III's.

Although there was no significant difference between Group I (6.41 ± 0.20) and Group II (6.45 ± 0.20) for mean Gash-Px level ($p > 0.05$), the mean level in Group III (6.67 ± 0.21) was significantly higher than those of both Groups I and II.

There was no significant difference among the three groups for mean 8-OHdG level ($p > 0.05$)

CONCLUSIONS

This study demonstrated that both T-cigs and E-cigs had unfavourable effects on markers of oxidative stress and inflammatory cytokines, and that smoking cessation appeared to have a beneficial effect.

Implications for practice

Patients need to be informed that the harmful effect of traditional smoking also applies to new modes of smoking such as Vaping.

Reference

1. Karaaslan F, Dikilitas A, Yigit U. The effects of vaping electronic cigarettes on periodontitis. Australian Dental Journal. 2020; 65: 143-9.