



T.C.
YEDITEPE UNIVERSITY
INSTITUTE OF HEALTH SCIENCES
DEPARTMENT OF PROSTHODONTICS

**PREVALENCE OF TEMPOROMANDIBULAR
DISORDERS AND ITS RELATIONSHIP WITH
PSYCHOLOGICAL STATES, SLEEP QUALITY, AND
ORAL HEALTH-RELATED LIFE QUALITY IN
FIBROMYALGIA PATIENTS**

DOCTOR OF PHILOSOPHY THESIS

GOZDE SARAK, DDS

ISTANBUL 2021



T.C.
YEDITEPE UNIVERSITY
INSTITUTE OF HEALTH SCIENCES
DEPARTMENT OF PROSTHODONTICS

PREVALENCE OF TEMPOROMANDIBULAR
DISORDERS AND ITS RELATIONSHIP WITH
PSYCHOLOGICAL STATES, SLEEP QUALITY, AND
ORAL HEALTH-RELATED LIFE QUALITY IN
FIBROMYALGIA PATIENTS

DOCTOR OF PHILOSOPHY THESIS

GOZDE SARAk, DDS

SUPERVISOR
Assist. Prof. BURCU BAL

ISTANBUL 2021

THESIS APPROVAL FORM

Institute : Yeditepe University Institute of Health Sciences
Programme : Doctoral Program in Prosthetic Dentistry
Title of the Thesis :Prevalence of Temporomandibular Disorders and Its
Relationship with Psychological States, Sleep Quality, and Oral Health-Related Life
Quality in Fibromyalgia Patients
Owner of the Thesis : Gözde Sarak
Examination Date : 08.07.2021

This study has approved as a Doctorate Thesis in regard to content and quality by the Jury.

	Title, Name-Surname (Institution)
Chair of the Jury:	Prof. Dr. Ender Kazazoğlu Yeditepe University
Supervisor:	Assist. Prof. Burcu Bal Yeditepe University
Member/Examiner:	Prof. Dr. Nurhan Güler Yeditepe University
Member/Examiner:	Prof. Dr. Hakan Akın Sakarya University
Member/Examiner:	Prof. Dr. Haneft Kurt Istanbul Medipol University

APPROVAL

This thesis has been deemed by the jury in accordance with the relevant articles of Yeditepe University Graduate Education and Examinations Regulation and has been approved by Administrative Board of Institute with decision dated and numbered

Prof. Dr. Bayram YILMAZ
Director of Institute of Health Sciences

DECLARATION

I hereby declare that this thesis is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which has been accepted for the award of any other degree except where due acknowledgment has been made in the text.

08.07.2021
Gözde Sarak



ACKNOWLEDGEMENT

I would like to express my deep gratitude and respect to Assist. Prof. Burcu Bal, my supervisor, for her generous help, valuable advice during my research period. I wish to thank her for her patience and precious time.

I am grateful to Prof. Dr. Ender KAZAZOĞLU, the chair of the Department of Prosthodontics, Yeditepe Dental School for offering me his remarkable expertise and criticism in prosthetic dentistry.

I grateful to Prof. Dr. Koray ORAL, our esteemed professor, who never spared his knowledge and experience.

I would like to thank Assist. Prof. Ayşe Yalnız Erzincanlı who has patiently conveyed her clinical knowledge and experience to me, who has trusted me and made me feel this, and who has supported me not only professionally and academically but also in all stages of my life.

I grateful to the faculty members of the Department of Prosthodontics, who have always helped me with their clinical and theoretical knowledge, never spared me their close interest, and contributed greatly to my clinical education.

I would like to thank my dear friends, Dt. Sena Salkim Alibrahim, Dt. Gülce Ecem Doğancı, Dr. Vahide Hazal Abat and Dt. Cihangir Alibrahim, who were my companions and confidants, as well as my colleagues, during my undergraduate and doctoral education.

In addition, I would like to express my gratitude to my beloved husband Dt. Ali Cem Sarak for all of his sacrifice and love throughout this thesis, as well as to my daughter Defne for her endless love.

And finally, I would like to express my deepest and most sincere appreciation to my parents Dr. İskender Akşen and Prof. Dr. Feyzan Akşen for their constant and endless support during my time of academic education.

TABLE OF CONTENTS

THESIS APPROVAL FORM	ii
DECLARATION	iii
TABLE OF CONTENTS	v
LIST OF TABLES	viii
LIST OF FIGURES	x
LIST OF ABBREVIATIONS	xii
ABSTRACT	xiv
ÖZET	xv
1. INTRODUCTION AND PURPOSE	1
2. GENERAL INFORMATION	3
2.1. Temporomandibular Joint	3
2.1.1. Anatomy	3
2.1.2. Etiology Of Temporomandibular Disorders	7
2.1.3. History Of The Temporomandibular Disorder	9
2.1.4. Classification Of Temporomandibular Disorders	10
2.1.5. Diagnostic Criteria For Temporomandibular Disorders	15
2.2. Fibromyalgia	15
2.2.1. History Of Fibromyalgia	16
2.2.2. Epidemiology	16
2.2.3. Etiology	17
2.2.4. Signs And Symptoms	17
2.2.5. Diagnostic Criteria For Fibromyalgia Syndrome	18
2.3. Temporomandibular Disorders And Fibromyalgia	20
3. MATERIALS AND METHODS	22
3.1. Ethical Approval	22
3.2. Sample Size	22

3.3. Informed Consent.....	22
3.4. Participants	22
3.5. Questionnaires And Clinical Examination	23
3.5.1. Diagnostic Criteria For Temporomandibular Disorder Axis I—Physical Assessment	24
3.5.2. Diagnostic Criteria For Temporomandibular Disorder Axis Ii	26
3.5.3. Bruxism32	
3.5.4. Revised Symptom Checklist-90.....	32
3.5.5. Oral Health Impact Profile.....	32
3.5.6. Pittsburgh Sleep Quality Index	35
3.6. Statistical Analysis.....	37
4. RESULTS.....	38
5. DISCUSSION AND CONCLUSION.....	69
5.1. Study Population.....	69
5.2. Study Setting.....	69
5.3. Fibromyalgia And Temporomandibular Disorder	71
5.4. Fibromyalgia.....	72
5.4.1. Sleep Quality.....	72
5.4.2. Oral Health-Related Quality Of Life.....	73
5.4.3. Psychological Status.....	73
5.5. Temporomandibular Disorders.....	74
5.5.1. Bruxism	74
5.5.2. Trauma	75
5.5.3. Sleep Quality.....	76
5.5.4. Oral Health-Related Quality Of Life.....	77
5.5.5. Psychological Status.....	78
5.5.6. Functional Limitation Of The Masticatory System	79
5.6. Bruxism	79
5.6.1. Sleep Quality.....	79
5.6.2. Oral Health-Related Quality Of Life.....	80

5.6.3. Psychological Status.....	81
6. REFERENCES	83
7. APPENDIX	103



LIST OF TABLES

Table 1 Classification of TMD, Okeson.....	10
Table 2 Taxonomy of TMD, DC/TMD	11
Table 3 The JFLS-20.....	28
Table 4 Patient Health Questionnaire-4.....	29
Table 5 Patient Health Questionnaire-9.....	30
Table 6 GAD-7.....	31
Table 7 OHIP-14.....	34
Table 8 PSQI.....	36
Table 9 Demographic Characteristics of the Sample.....	38
Table 10 Comparison of DC/TMD Diagnoses Between the Case Group and Control Group.....	40
Table 11 Comparison of FM and Bruxism Diagnoses and Trauma History Between Patients With and Without TMD.....	42
Table 12 Comparison of the Mean Age Between Cases and Controls.....	44
Table 13 Correlation Between Age and PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Scores.....	45
Table 14 Comparison of Marital Status Between the Case and Control Groups.....	46
Table 15 Comparison of PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Values Between Different Marital Statuses.....	47
Table 16 Comparison of the PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Values Between the Case and Control Groups.....	49
Table 17 Comparison of the PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Values Between Patients With and Without TMD.....	52
Table 18 Comparison of the PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Values Between Patients With and Without DD.....	55
Table 19 Comparison of the PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Values Between Patients With and Without Myalgia.....	58
Table 20 Comparison of the PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Values Between Patients With and Without Arthralgia.....	61
Table 21 Comparison of the PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Values Between Patients With Bruxism and the Control Groups.....	64

Table 22 Correlation Between the PSQI, OHIP, and SCL-90-R Symptom Dimensions
and DC/TMD Axis II Scores. 68



LIST OF FIGURES

Figure 1 Tender Points of FM.....	19
Figure 2 DC/TMD Diagnostic Decision Tree 1.....	25
Figure 3 DC/TMD Diagnostic Decision Tree 2.....	26
Figure 4 Marital Status in the Case and Control Groups.....	39
Figure 5 Prevalence of DC/TMD Diagnosis in the Case and Control Groups.....	41
Figure 6 Prevalence of FM, Bruxism, and Trauma History in Patients with and without TMD.	43
Figure 7 Prevalence of Good and Poor Sleepers in the Case and Control Groups.....	50
Figure 8 Comparison of Different DEP Levels Between the Groups.....	50
Figure 9 Comparison of Different ANX Levels Between the Groups.....	51
Figure 10 The Incidence of Psychiatric Symptoms in the Case and Control Groups....	51
Figure 11 Prevalence of Good and Poor Sleepers Among Patients with and without TMD.....	53
Figure 12 Comparison of Different DEP Levels Between Patients with and without TMD.....	53
Figure 13 Comparison of Different ANX Levels Between Patients with and without TMD.....	54
Figure 14 The Incidence of Psychiatric Symptoms in Patients with TMD.....	54
Figure 15 Prevalence of Good and Poor Sleepers Among Patients with DD.....	56
Figure 16 Comparison of Different DEP Levels Between Patients with DD.....	56
Figure 17 Comparison of Different ANX Levels Between Patients with DD.....	57
Figure 18 The Incidence of Psychiatric Symptoms in Patients with DD.....	57
Figure 19 Prevalence of Good and Poor Sleepers Among Patients with and without Myalgia.....	59
Figure 20 Comparison of Different Dep Levels Between Patients with and without Myalgia.....	59
Figure 21 Comparison of Different Anx Levels Between Patients with and without Myalgia.....	60
Figure 22 The Incidence of Psychiatric Symptoms in Patients with Myalgia.....	60
Figure 23 Prevalence of Good and Poor Sleepers Among Patients with and without Arthralgia.....	62
Figure 24 Comparison of Different DEP Levels Between Patients with and without Arthralgia.....	62

Figure 25 Comparison of Different ANX Levels Between Patients with and without Arthralgia.....	63
Figure 26 The Incidence of Psychiatric Symptoms in Patients with and without Arthralgia.....	63
Figure 27 Prevalence of Good and Poor Sleepers Among Patients with and without Bruxism.....	65
Figure 28 Comparison of Different DEP Levels Between Patients with and without Bruxism.....	65
Figure 29 Comparison of Different ANX Levels Between Patients with and without Bruxism.....	66
Figure 30 The Incidence of Psychiatric Symptoms in Patients with Bruxism.....	66



LIST OF ABBREVIATIONS

AAOP	American Academy of Orofacial Pain
ACR	American College of Rheumatology
ADD.S	Additional SCL-90-R subscale
ANX	Anxiety
DC/TMD	Diagnostic Criteria for Temporomandibular Disorder
DD	Disc Displacement
DEP	Depression
FM	Fibromyalgia
GAD-7	Generalized Anxiety Disorder-7
GCPS	Graded Chronic Pain Scale
GSi	Global Severity Index
HOS	Hostility
I-S	Interpersonal sensitivity
IHA	International Headache Association
JFLS	Jaw Functional Limitation Scale
O-C	Obsessive Compulsion
OHIP-14	Oral Health Impact Profile
OHRQoL	Oral Health-Related Quality of Life
PAR	Paranoid Ideation
PHOB	Phobic Anxiety
PHQ-15	Patient Health Questionnaires-15
PHQ-4	Patient Health Questionnaires-4
PHQ-9	Patient Health Questionnaires-9
PSQI	Pittsburgh Sleep Quality Index
PSY	Psychoticism

RDC/TMD	Research Diagnostic Criteria for Temporomandibular Disorder
SCL-90-R	Revised Symptom Checklist-90
SF-36	The 36-Item Short Form Health Survey Questionnaire
SOM	Somatization
TM	Temporomandibular
TMD	Temporomandibular Disorder
TMJ	Temporomandibular Joint
WHOQOL	The World Health Organization Quality of Life



ABSTRACT

Sarak, G. (2021). Prevalence of Temporomandibular Disorders and Its Relationship with Psychological States, Sleep Quality, and Oral Health-Related Life Quality in Fibromyalgia Patients. Yeditepe University, Institute of Health Science, Department of Prosthodontics, PhD thesis, Istanbul

The purpose of this study is to determine the prevalence of temporomandibular disorders (TMDs) in patients with fibromyalgia (FM) and its association with sleep quality, oral health-related life quality, and psychological states. This case-control study included 41 women who were diagnosed with FM according to the American College of Rheumatology (ACR) criteria and 41 healthy women who were not diagnosed with FM. Participants were examined by a rheumatologist to diagnose FM and a dentist to diagnose TMD and bruxism. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) were used for the diagnosis of TMD. Sleep quality was assessed with the Pittsburgh Sleep Quality Index (PSQI), oral health-related quality of life was assessed with the Oral Health Impact Profile (OHIP-14), and psychological status was assessed with the Revised Symptom Checklist (SCL-90-R). TMD and bruxism were found more commonly in the case group (75.6% and 68.3%, respectively) than in the control group (46.3% and 29.3%, respectively). The rate of poor sleepers in the case group (43.9%) was significantly higher than the rate of poor sleepers in the control group (7.3%). In the case group, the patients showed significantly higher values in the symptom dimensions of somatization, anxiety, obsessive-compulsion, depression, psychoticism, paranoid ideation, hostility, and general severity index. Similar to the case group, the rate of poor sleepers in patients with TMD (32.0%) was significantly higher than the rate of poor sleepers in patients without TMD (15.6%). In addition, participants with TMD had significantly higher symptoms of depression than participants without TMD. In conclusion, while TMD and bruxism were found to be more common in individuals with FM in our study, FM and TMD had negative impacts on sleep quality and oral health-related quality of life. It was determined that there was a negative relationship between these disorders and the general psychopathological state.

Keywords: Fibromyalgia, Temporomandibular Disorder, Psychological States, Sleep Quality, Oral Health-Related Quality of Life

ÖZET

Sarak, G. (2021). Fibromiyalji Hastalarında Temporomandibular Bozuklukların Yaygınlığı ve Psikolojik Durumlar, Uyku Kalitesi ve Ağız Sağlığına İlişkin Yaşam Kalitesi ile İlişkisi. Yeditepe Üniversitesi, Sağlık Bilimleri Enstitüsü, Protetik Diş Tedavisi Anabilim Dalı, Doktora Tezi, İstanbul

Bu çalışmanın amacı, fibromiyalji (FM) hastalarında temporomandibular rahatsızlıkların (TMR) görülme sıklığını belirlemek ve bu rahatsızlıkların uyku kalitesi, ağız sağlığı ile ilişkili yaşam kalitesi ve psikolojik durumlar ile ilişkisini belirlemektir. Bu çalışmaya Amerikan Romatoloji Derneği (ACR) kriterlerine göre FM tanısı konmuş 41 kadın ve FM tanısı konmayan 41 sağlıklı kadın dahil edildi. Katılımcılar FM tanısı için bir romatolog ve TMR ve bruksizm tanısı için bir diş hekimi tarafından muayene edildi. TMR tanısı için Temporomandibular Rahatsızlıklar için Tanı Kriterleri (TMR/TK) kullanıldı. Uyku kalitesi Pittsburgh Uyku Kalitesi İndeksi (PUKİ) ile, ağız sağlığı ile ilişkili yaşam kalitesi Ağız Sağlığı Etki Ölçeği (OHIP-14) ile ve psikolojik durum Psikolojik Belirti Tarama Testi (SCL-90-R) ile değerlendirildi. TMR ve bruksizmin, vaka grubunda (%75.6 ve %68.3 sırasıyla) kontrol grubundan (%46.3 ve %29.3 sırasıyla) daha sık görüldüğü tespit edildi. Vaka grubunda kötü uyku kalitesi görülme oranı (%43.9) kontrol grubunda kötü uyku kalitesi görülme oranından (%7.3) istatistiksel olarak anlamlı düzeyde yüksektir. Vaka grubunda kontrol grubuna göre somatizasyon, anksiyete, obsesif kompülsiyon, depresyon, psikotizm, paranoid düşünce, düşmanlık belirtileri istatistiksel olarak anlamlı düzeyde yüksektir. Vaka grubuna benzer şekilde, TMR'li hastalarda kötü uyku kalitesi görülme oranı (%32.0) TMR görülmeyen hastalarda kötü uyku kalitesi görülme oranından (%15.6) istatistiksel olarak anlamlı düzeyde yüksektir. Buna ek olarak, TMR görülen katılımcılarda depresyon belirtisi TMR görülmeyen katılımcılardan istatistiksel olarak anlamlı düzeyde yüksektir. Sonuç olarak, çalışmamızda FM görülen bireylerde daha sık TMR ve bruksizm görüldüğü tespit edilirken, FM ve TMR görülen bireylerde uyku kalitesi ve ağız sağlığı ile ilişkili yaşam kalitesi kötü olarak tespit edildi. Bu rahatsızlıklar ve genel psikopatolojik durum arasında da negatif bir ilişki olduğu tespit edildi.

Anahtar Kelimeler: Fibromiyalji, Temporomandibular Rahatsızlık, Psikolojik durum, Uyku Kalitesi, Ağız Sağlığı ile İlişkili Yaşam Kalitesi

1. INTRODUCTION AND PURPOSE

Temporomandibular disorder (TMD) is a group of clinical signs and symptoms that affect the masticatory muscles, the temporomandibular joint (TMJ), and other related structures. It is estimated that approximately a third of the population suffers from TMD, which is the most common chronic orofacial pain condition. [1, 2] TMD mostly occurs in adults between the ages of 20 to 40 and in women.

TMD is mainly characterized by pain in the muscles or joints, limitations or deviations in mandibular motion, and clicking or crepitation sounds in the TMJ, accompanied by additional symptoms such as tension headaches, dizziness, and tinnitus. [3] Sleep disorders and psychological disturbances such as ANX and DEP can also be present in TMD patients. [4]

According to the ACR, FM is a long-term condition in which one experiences pain and tender points in the upper and lower body, involving 11 of 18 musculoskeletal system parts, for at least three months. [5]

Patients with FM frequently complain of morning stiffness, fatigue, poor sleep quality, paresthesia, and psychological difficulties. They also mention migraines, chronic headaches, irritable bowel syndrome, atypical chest pain, and irritable bladder syndrome as nonspecific pain problems. [6]

Some authors have indicated that some oral and stomatognathic problems are associated with FM. These patients had a variety of oral health problems, including TMJ sounds and limited mouth opening, pain when moving their jaws or palpating TMJ, and xerostomia, glossodynia, and oral ulceration, among other oral conditions. [7-9]

Some symptoms that have been reported in FM patients, such as morning stiffness, fatigue, sleep disturbance, ANX, headaches, mood disorders, and stress, are also frequently diagnosed as part of TMDs. [8, 10] Numerous previous studies have affirmed that a high frequency of TMD symptoms has been seen among FM patients. [9, 11-14] According to some studies, FM and TMD share same clinical features, such as chronic progression, poorly understood pathophysiology, and serious physical and psychological consequences. [15]

Studies have shown that patients with FM have a high prevalence of TMD signs and symptoms in the range of 71% to 97%, and patients with TMD similarly have a high prevalence of widespread pain in other segments of the body in the range of 10% to 18.4%. [7, 13-15]

While studies indicate a link between TMD and FM, their relationship remains unclear, as several studies were retrospective or observational. [2, 14, 16] The ACR classification for FM and the criterion standard tool to evaluate TMD were only used in a few studies. [15, 17]

Aims of the study: This study aims to determine the prevalence of TMDs in patients with FM and to assess their association with sleep quality, oral health-related life quality, and psychological states.

The hypotheses are as follows:

1. TMDs are more common in FM patients than in healthy individuals.
2. Psychological disorders are more common in FM patients compared to healthy individuals.
3. Psychological disorders are more common in TMD patients compared to healthy individuals.
4. Poor sleep quality is more common in FM patients than in healthy individuals.
5. Poor sleep quality is more common in TMD patients than in healthy individuals.
6. Oral health-related poor life quality is more common in FM patients than in healthy individuals.
7. Oral health-related poor life quality is more common in TMD patients than in healthy individuals.

2. GENERAL INFORMATION

2.1. Temporomandibular Joint

Temporomandibular joint, which connects the mandible to the skull and regulates mandibular movements, is a diarthrodial joint formed by the mandibular condyle and glenoid fossa of the temporal bone and is located in the front of the outer ear canal. The TMJ allows hinging movement in one plane and, at the same time, also allows gliding movements, which classify it as a ginglymoarthrodial joint. Morphologically, the TMJ can vary from person to person, and the right and left joints can even vary in the same person. The TMJ is different from the other joints of the body due to it being a morphologically and functionally complex joint. Histologically, unlike other articular surfaces of the body, it is covered with fibrocartilage instead of hyaline cartilage.

2.1.1. Anatomy

The TMJ consists of the glenoid fossa, articular eminence, and condyle, which are defined as hard tissue, and a joint disc, retrodiscal tissues, a joint capsule, and joint ligaments, which are defined as soft tissue. [18]

2.1.1.1. Bony Structures of the Temporomandibular Joint

The bony structure comprises the articular fossa; the articular eminence, which is an anterior protuberance continuous with the fossa; and the condylar process of the mandible that rests within the fossa. The movable round upper end of the lower jaw is called the condyle, and the socket is called the articular fossa. The concave area, which is the squamous part of the temporal bone, is located in front of the fissura petrotympanica, also known as the articular or glenoid fossa. The mandibular condyle fits into the glenoid fossa. The articular eminence or tuberculum articulare is the convex part in front of the glenoid fossa. The articular eminence is supported by thick fibrous cartilage, which increases its resistance. The posterior part of the mandibular fossa is thin and not resistant to excessive loads. [18, 19]

2.1.1.2. Articular Disc

The articular disc separates the mandibular condyle and glenoid fossa from direct articulation. It is made up of dense fibrous connective tissue. Due to its viscoelastic properties, the disc acts as a buffer between the articular surfaces and absorbs the forces. It is biconcave and divides the articular joint into two compartments: a larger superior compartment and a smaller inferior compartment. [19, 20]

Hinging movements take place in the lower compartment and gliding movements take place in the upper compartment. [19] Functionally, the articular disc serves as a non-

ossified bone that permits the complex movements of the joint. The condyle-disc complex is responsible for the rotational movement of the TMJ. Due to the disc's loose attachment to the articular fossa, free sliding movement between these surfaces in the superior cavity is possible.

The disc has three parts in the sagittal section. The central area is called the intermediate zone and is the thinnest part. The anterior part is slightly thinner than the posterior part. The articular surface of the condyle is located on the intermediate part.

The intermediate part of the disc is the part that is the most exposed to pressure while functioning and acts as a stress relief area. This part is devoid of any blood vessels and nerve fibers. [21] Nevertheless, few blood vessels and nerve fibers are found in the anterior and posterior parts. The posterior part adheres to the retrodiscal area (bilaminar zone), which consists of dense neural and vascular structures.

2.1.1.3. Retrodiscal Tissue

The retrodiscal tissue, which is also called the retrodiscal lamina, bilaminar zone, or retrodiscal pad, is highly vascularized and innervated. A loose connective tissue makes up this part of the TMJ.

In the anterior region, the retrodiscal lamina adheres to the articular disc, and in the posterior region, it adheres to the posterior wall of the joint capsule. The retrodiscal area consists of two fibrous laminae: the superior retrodiscal lamina and the inferior retrodiscal lamina. The superior retrodiscal lamina contains many elastic fibers, while the inferior retrodiscal lamina is composed of collagenous fibers. [18]

A vascular pad containing many vessels, nerves, fat cells, and loose fibroelastic connective tissue exists between the upper and lower lamina. [22]

The joint capsule and retrodiscal area are rich in nerves and veins and are sensitive. It has an inflammatory capacity since it contains many proprioceptive and nociceptive receptors. [23]

2.1.1.4. Ligaments

Ligaments consist of collagenous connective tissue fibers that have determined lengths. They do not stretch, and they protect the structures. However, if extensive force is applied to a ligament over a prolonged period, the ligament can be elongated.

Although ligaments do not play an active role in joint functions, they have three main functions. They stabilize the joint and limit and restrict border movements. [18]

1. The collateral ligament
2. The capsular ligament

3. The TM ligament
4. The accessory ligaments of TMJ
 - a. The stylomandibular ligament
 - b. The sphenomandibular ligament

Other than these ligaments, the discomalleolar ligament (Pinto) and Tanaka ligament may also be found. [24]

Collateral ligaments connect the articular disc's medial and lateral borders to the condyle's poles. There are two types of discal ligaments: medial and lateral discal ligaments. The medial discal ligament connects the articular disc's medial edge to the condyle's medial pole, while the lateral discal ligament connects the articular disc's lateral edge to the condyle's lateral pole.

The superior and inferior joint cavities are separated by these ligaments, which divide the joint mediolaterally. They prevent the disc from moving away from the condyle. As the condyle slides anteriorly and posteriorly, they ensure passive movement of the disc with the condyle.

Vascular supply and innervation are present in the collateral ligaments. This innervation transmits data about joint position and movement. As a result, the strain on these ligaments causes discomfort.

The entire TMJ is surrounded by the capsular ligament, comprising fibrous and thin connective tissue. The collagenous fibers of the capsular ligament are superiorly attached to the articular surface of the temporal bone and inferiorly attached to the neck of the condyle. The articular disc is attached to the capsular ligament medially and laterally as well as anteriorly and posteriorly. This divides the joint into upper (superior) and lower (inferior) cavities. The anterior wall of the capsule, which comprises a loose connective tissue, cannot withstand excessive forces like the other parts of the capsule. [25] Laterally, the joint capsule is stringent and stabilizes the joint during jaw movements.

Any forces that tend to separate or dislocate the articular surfaces are resisted by the capsular ligament. Another function of the capsular ligament is to surround the joint, which helps to keep the synovial fluid within the joint space. The synovial membrane that covers the inner surface of the capsule procures synovial fluid. The synovial fluid meets the metabolic requirements of non-vascular joint surfaces and lubricates joint surfaces to reduce friction during function. [26] The joint surfaces are lubricated with the movement of synovial fluid from one region to another while the jaw moves. Further, the cartilage has the ability to store a limited amount of synovial fluid. [18]

Like the collateral ligament, the capsular ligament is innervated. This provides proprioceptive feedback.

The lateral ligament refers to the strong tight fibers that reinforce the weak lateral part of the capsular ligament. The TM ligament, which controls the movements of the condyle-disc complex, consists of two parts: an outer oblique portion and an inner horizontal portion. [18] The oblique portion of the lateral ligament limits the extent to which the mouth can open and affects the normal opening movement of the mandible. The TM ligament's inner horizontal portion restricts the condyle's and disc's posterior movement, protecting the retrodiscal tissues from trauma and the lateral pterygoid muscle from overextension.

The TMJ has two accessory ligaments: the sphenomandibular ligament, which is responsible for limiting mouth opening and the anterior and posterior movements of the lower jaw, and the stylomandibular ligament. Similar to the other accessory ligament, stylomandibular ligaments limit mouth opening and the anterior and posterior movement of the lower jaw. In terms of mandibular movements, neither accessory ligament has a significant limiting effect.

2.1.1.5. Muscles of Mastication

The masseter, temporalis, medial pterygoid, and lateral pterygoid are the muscles of mastication. The digastric muscle also plays an important role in mandibular function.

The masseter originates from the zygomatic arch and extends downward to the lateral aspect of the lower border of the ramus of the mandible. It is made up of two portions: the superficial portion and the deep portion. It is a powerful muscle that provides the force necessary to chew efficiently. When the masseter muscle contracts, it elevates the mandible, and the teeth are brought into contact. The superficial portion of the masseter aids in protruding the mandible.

The temporal muscle originates from the temporal fossa and the lateral surface of the skull. It is subdivided into three distinct areas based on the direction of the fibers and their function. The anterior portion consists of vertically oriented fibers, the middle part consists of oblique fibers, and the posterior portion contains horizontally directed fibers. As the fibers of the temporalis contract, the mandible is elevated. The contraction of the middle and posterior portions also retrude the mandible.

The internal pterygoid is active in elevating and protruding the mandible. As the fibers of the medial pterygoid contract, the mandible is elevated. Its unilateral contraction causes the mediotrusive movement of the mandible.

The external pterygoid is identified as two different muscles. The functions of these two muscles are almost the opposite. These muscles are called the inferior lateral pterygoid and the superior lateral pterygoid. The bilateral contraction of the inferior lateral pterygoid protrudes the mandible. The unilateral contraction causes a mediotrusive movement of the condyle and creates a lateral movement of the mandible to the opposite side. The inferior pterygoid is also active while opening. The superior lateral pterygoid, which is smaller than the inferior lateral pterygoid, is especially active during the closure of the mandible against resistance, as in while chewing or clenching the teeth. Unlike the inferior lateral pterygoid, the superior lateral pterygoid is inactive while opening.

The digastric, which is not considered a mastication muscle, has an important role in the function of the mandible. Similar to the anterior belly and posterior belly, it is divided into two portions. When the hyoid bone is stabilized and the digastric contracts bilaterally, the mandible is pulled backward and depressed. When the mandible is fixed, the digastric muscles elevate the hyoid bone with the suprahyoid and infrahyoid muscles. This is essential for swallowing. [18]

2.1.2. Etiology of Temporomandibular Disorders

Biological, environmental, social, emotional, and cognitive factors all play a role in the development of TMDs, as does their etiology. [27] There is no single etiologic factor that explains all the signs and symptoms. Various factors can contribute to TMDs. These factors consist of initiating, predisposing, and perpetuating factors. In some examples, a single element may cause and increase the TMD. The management of TMD depends on the identification and control of these factors. [18]

Factors contributing to the development of TMJ include occlusal abnormalities; orthodontic treatment; parafunctions such as bruxism, trauma and joint laxity; and psychological situations such as stress, ANX, and DEP.

2.1.2.1. Occlusal Factors

One of the etiological factors thought to initiate TMD is occlusal disorders. [28] Some authors concluded that malocclusion could act as a cofactor in the etiology of TMD, and some occlusal features might be consequences of the disease rather than its initiating factors. [29]

Most occlusal disorders do not cause pathology due to neuromuscular adaptation, but if the neuromuscular adaptation capacity decreases due to stress, emotional problems, and trauma, a disruption occurs in the load distribution in the joints and muscles.

Existing data, according to the literature, cannot define the precise function of occlusal variables in TMJ problems. As well as anterior open bite and five or more missing teeth TMDs can also be caused by posterior cross-bite, overjet or overbite greater than 5 millimeters, edge-to-edge bite and anterior open bite. [30]

2.1.2.2. Psychological Factors

Psychological studies have shown that patients with TMDs or other musculoskeletal pain disorders have similar psychological profiles. Although understanding the psychosocial and psychological aspects is important for understanding TMD, insufficient evidence exists to suggest that they are etiologic. [31, 32]

It has been suggested that TMJ pain may occur as a result of emotional stress. [33-35] Stress, ANX, and other psychological factors induce muscle hyperactivity and muscle fatigue. Additionally, TMD symptoms, particularly pain, play a role in the development of DEP and psychic diseases. [36]

2.1.2.3. Hormonal Factors

Epidemiologic studies have shown that the frequency and severity of TMDs are higher in women than in men. [37-39] Women are four times more likely than men to experience the signs and symptoms of TMD. [30]

In women, the incidence of chronic myofascial pain (MP) has been reported to be three times higher than in men. [37] Although the reason for the low prevalence of TMD in men has not yet been fully elucidated, a reasonable explanation could be the presence of higher testosterone levels.

There is a hypothesis that the presence of estrogen receptors in the TMJ of women alters metabolic functions and increases ligament laxity. In addition, studies have shown that patients undergoing estrogen replacement therapy for menopause experience a 30% increase in painful symptoms, while women on oral contraceptives experience a 20% increase. There is still no direct evidence that links female reproductive hormones to TMD. [30]

2.1.2.4. Trauma

Trauma is a predisposing and initiating factor for TMD. Abundant evidence supports that trauma to the facial structure can lead to TMD. It has a greater effect on intracapsular disorders than muscular disorders. Trauma is categorized into two general types: macrotrauma, which is any sudden force that can result in structural alterations, and microtrauma, which is any weak force that is repeatedly applied, such as clenching.

Whiplash-type injuries to the head or neck are commonly thought to be important risk factors for TMD. Whiplash trauma exposes one in every three people to the risk of developing delayed TMJ symptoms. [18]

2.1.2.5. Parafunction

Parafunctions such as clenching, grinding, and excessive gum chewing, can lead to TMD. Bruxism was discovered in 87.5 percent of patients with disc displacement (DD) and joint pain, according to research. Patients with muscle dysfunction are more likely to experience bruxism, whereas patients with joint dysfunction are less likely to experience it. This parafunction may result in condylar bone remodeling and articular cartilage degradation, as well as TMJ osteoarthritis. [18, 30]

2.1.2.6. Orthodontic Treatment

Previous studies have concluded that orthodontic treatment does not increase or cure TMDs. [40, 41] Even though most of the studies do not support the correlation between orthodontic treatment and TMDs, a study that investigated the genetic influence of TMD showed that the subjects who reported a history of orthodontic treatment had a significantly higher risk of developing TMD when they had a variant of the gene encoding catechol-O-methyltransferase. [42-44]

2.1.3. History of the temporomandibular disorder

In 1918, Prentiss, an anatomist, suggested that the etiology of TMD is the pressure that the condyle exerts on the auriculotemporal vein, nerve, and TMJ because of posterior tooth loss. He also suggested that this pressure causes orofacial pain.

In 1932, Goodfriend [45] defined subarthrosis (subluxation), which was considered to be a partial dislocation, and dysarthrosis, which was a complete dislocation of the normal condylar glenoid relationship.

In 1934, Costen [46] described symptoms such as tenderness to palpation of the mandibular joint, deafness, dizzy spells, headaches when eyes and sinuses are found to be negative, and trismus as a sign of Costen's Syndrome and reported that these symptoms can be cured by changing the occlusion. The modern history of TMD started with these publications by Costen. [45]

In 1955, Schwartz [47] stated that spasms in the masticatory muscles and psychological factors play an important role in TMDs and named this disease "temporomandibular joint pain dysfunction syndrome." After Schwartz, in 1959, Shore and Lupton defined it as "temporomandibular joint dysfunction syndrome," and in 1965, Derksen and Bosman named it "pain dysfunction syndrome." [45]

In 1969, Laskin [48] used the term "myofascial pain dysfunction" and reported that muscle spasms were caused by chronic oral habits brought about by emotional stress. In 1989, the term "temporomandibular diseases" was coined by Bell and includes the pathologies of the TMJ and related structures and all diseases of the masticatory muscles. This term is also accepted by the American Dental Association.

2.1.4. Classification of Temporomandibular Disorders

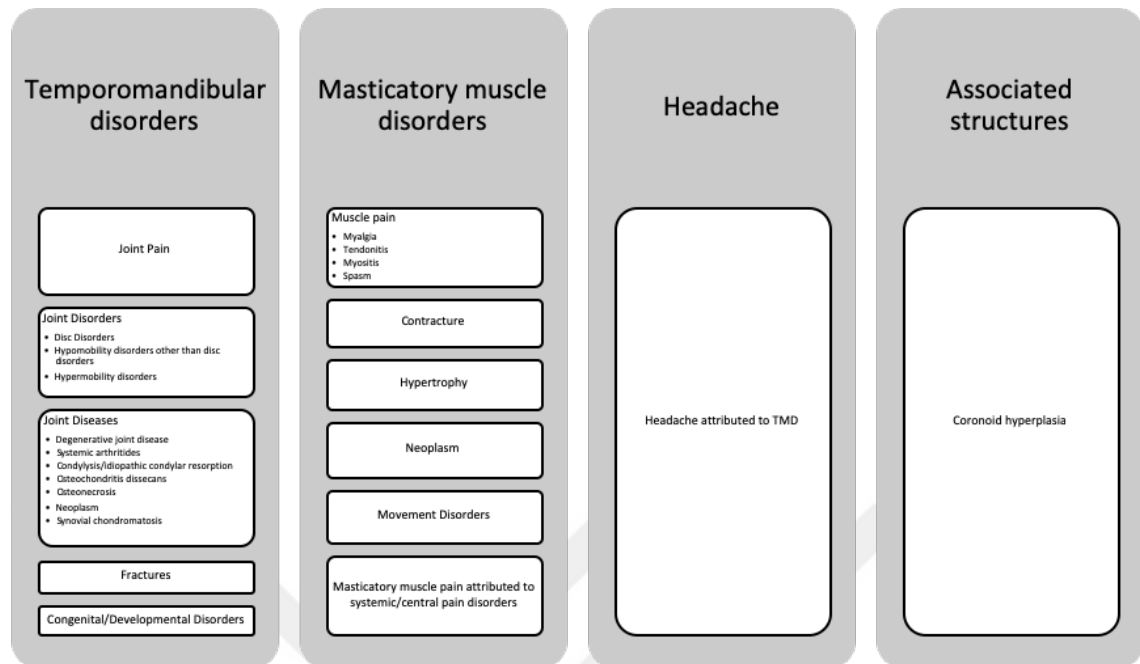
TMDs were included in the classification prepared by the American Academy of Orofacial Pain (AAOP) and the International Headache Association (IHA) in 1987. [49] This classification has been modified by Okeson, and according to this new classification, TMDs are classified into four groups as masticatory muscle disorders, TMJ disorders, chronic mandibular hypomobility, and growth disorders, as seen in Table 1. [18]

Table 1 Classification of TMD, Okeson [18]

Masticatory Muscle Disorders	Temporomandibular Joint Disorders	Chronic Mandibular Hypomobility	Growth Disorders
Protective Co-Contraction	Derangements of the Condyle-Disc Complex • Disc Displacement with Reduction • Disc Displacement without Reduction	Ankylosis	Congenital/Developmental Bone Disorders • Agenesia • Hypoplasia • Hyperplasia • Neoplasia
Local Muscle Soreness	Structural Incompatibilities • Adhesions/Adherences • Deviation in Form • Subluxation • Spontaneous Dislocation	Muscle Contracture	
Myofascial Pain	Inflammatory Disorders • Synovitis • Capsulitis • Retrodisclitis • Arthritides	Coronoid Impedance	Congenital/Developmental Muscle Disorders
Myospasm			
Chronic Centrally Mediated Myalgia			

According to the DC/TMD used in our study, TMDs were divided into four groups as TMDs, masticatory muscle disorders, headache, and associated structures, as seen in Table 2. [50]

Table 2 Taxonomy of TMD, DC/TMD [50]



2.1.4.1. Masticatory Muscle Disorders

2.1.4.1.A. Myofascial Pain

Myofascial pain is pain that arises from trigger points in a muscle. [51] Trigger points are small, sensitive areas in a muscle that cause pain in a distant region. Trigger points can be active or latent. Active trigger points cause pain or referred pain. [52, 53] In contrast to trigger points, tender spots cause pain locally.

When a person experiences myofascial pain, their muscles are in spasm with increased tension and decreased flexibility. [54] Clenching, bruxism, and other parafunctional habits are thought to cause myofascial pain and dysfunction. The result is a strain on the masticatory musculature, spasms, pain, and functional limitations in the mouth. [55]

2.1.4.2. Derangements of the Condyle-Disc Complex

The derangement of the condyle-disc complex is defined as an abnormal relationship between the disc and condyle in their positional relationship. [56] The etiologic factors of the derangements of the condyle-disc complex are anatomical factors of the TM joint, biomechanical factors (overloading of the TM joint due to oral habits), joint hypermobility, changes in joint lubrication, and trauma. [57] Disc derangements

result from the elongation of the capsular ligament and discal ligament. Symptoms such as preauricular pain, clicking, headache, earache, tinnitus, and neck pain are observed.

2.1.4.2.A. Disc Displacement with Reduction

DD is a condition whereby the articular disc is displaced from its normal functional relationship with the condyle. The articular disc is displaced in relation to the condyle when the mouth is closed; when the mouth is opened, the disc reduces and returns to the intermediate area between the condyle and the articular tubercle. This is referred to as DD with reduction. It is the most common type of disc dysfunction.

The articular disc can be displaced in any direction; however, anterior displacement appears to be the most common. During condylar translation, the disc reduces and the range of motion is not limited. [58] This alteration in normal disc movement can occur because of the elongation of the discal collateral ligaments and inferior retrodiscal lamina. Heavy compressive forces and traction of the lateral pterygoid muscle can also change the normal disc movements.

A clicking or popping sound may be produced by the movement of the disc. TMJ clicking is the most common complaint of patients. [59] The patient may report a catching sensation during mouth opening. It is generally a painless condition, causing little or no discomfort to the patient. [60] During mouth opening and closing, the movement of the articular disc can be felt by palpating the joints. Deviations are common clinical findings of DD with reduction. [18]

It has been suggested that DD with reduction could be the first stage of DD, progressing from DD with reduction to DD without reduction. [61]

2.1.4.2.B. Disc Displacement without Reduction and Intermittent Locking

The disc is displaced in a closed-mouth position and intermittently locks in the open-mouth position.

2.1.4.2.C. Disc Displacement without Reduction with Limited Opening

The disc is displaced in the closed-mouth position, and in the open-mouth position, it does not reduce to the normal position. It is also called "closed locking." The superior retrodiscal lamina is the only structure that can retract the disc posteriorly. As the elasticity of the superior retrodiscal ligament decreases and its length increases, the retraction of the disc becomes difficult and the disc cannot be reduced during the translation of the condyle. [18]

Disk displacement without reduction, in contrast to disk displacement with reduction, produces no sound. However, patients may report that there was a click before

the limited opening, and prior to the dislocation, there may have been a history of a catching sensation. The maximum opening between the upper and lower jaws is reduced. During the maximal opening, there is a deflection to the ipsilateral side. Normal eccentric movement to the ipsilateral side and restricted eccentric movement to the contralateral side are the other symptoms.

2.1.4.2.D. Disc Displacement without Reduction and Limited Opening

This condition is identical to the DD without reduction with the exception that mandibular movement is not limited. Nevertheless, limitation of the mandible has occurred in the past. [62]

2.1.4.3. Inflammatory Joint Disorders

Inflammatory joint disorders are a group of disorders in which intra-articular and peripheral tissues become inflamed by cause of trauma or DD. [18, 63] Inflammatory disorders are characterized by a constant, aching pain that is emphasized by joint movements. The inflammatory disorders are retrodiscitis, synovitis, capsulitis, and arthritis. Because of similar clinical symptoms, it is generally difficult to clinically differentiate the inflammatory disorders from each other. [18]

Synovitis, which is caused by trauma or infection, is defined as the inflammation of the synovial tissues surrounding the TMJ. It is characterized by a constant intracapsular pain that is increased by joint movements. [18, 64] Swelling may occur due to synovitis, and it can cause acute malocclusion. Disengagement of the ipsilateral posterior teeth and heavy contact with the contralateral side is observed.

Capsulitis is an inflammation of the capsule caused by a sprain, contusion, or tear of the ligaments as a result of trauma. [64] Even in the static joint position, capsulitis produces pain. Mandibular movements generally increase the pain. [18] The most common etiological factor of capsulitis is macrotrauma, especially an open-mouth injury.

Retrodiscitis is defined as the inflammation of the retrodiscal tissues. Because of their vascularized and innervated structure, retrodiscal tissues are incapable of tolerating more loading force. [65]

Like the other inflammatory disorders, retrodiscitis is characterized by constant aching pain, especially in the preauricular area, which is accentuated with jaw movements. Retrodiscitis may lead to swelling as in synovitis. Due to the swelling, acute malocclusion is seen as the disengagement of the posterior teeth on the affected side. When this occurs, the patient complains of an inability to bite. If force is applied, the pain

increases. Trauma is the most common etiological factor with retrodiscitis as with capsulitis.

The inflammation of the articular surfaces of the joint is called arthritis. Destructive bone changes are seen in arthritis. Several types of arthritis, such as rheumatoid arthritis, traumatic arthritis, and infectious arthritis hyperuricemia, can affect TMJ, but the most common type is osteoarthritis. [18]

Osteoarthritis is a degenerative joint disease characterized by the degeneration of articular surfaces and reparative adaptive osteogenesis. Excessive mechanical stress and increased loading of a joint play a major role in osteoarthritic changes. [18, 66] With continued loading forces, the articular surface softens and the bone resorbs.

It is generally associated with disc dislocation or perforation. When the condyle articulates directly with the fossa, the destructive process begins. In time, the dense fibrous articular surfaces are destroyed and bone resorption occurs. [18]

Primary and secondary noninflammatory osteoarthritis of the TMJ are the two types of osteoarthritis. The term "primary osteoarthritis" refers to a degenerative, non-inflammatory joint condition. Structural changes to the joint surfaces is a characteristic of this condition. Primary osteoarthritis, which primarily affects the elderly, is asymptomatic or has mild symptoms. Secondary osteoarthritis involves the same articular breakdown and remodeling process as primary osteoarthritis, but it can be linked to a previous event or disease that overloaded the remodeling mechanism. [64]

Osteoarthritis is usually painful and increases during jaw movements. Further, lateral palpation of the condyle increases the pain, and mandibular opening is limited due to joint pain. If osteoarthritis is not associated with DD, there is a soft end feel. Crepitation is one of its clinical findings. In the early stages of osteoarthritis, radiographs may appear normal.

Rheumatoid arthritis is a systemic autoimmune disease characterized by inflammatory arthritis and extra-articular involvement. The etiology of rheumatoid arthritis remains unknown and, therefore, cannot be prevented. [18]

Before the TMJ, other joints, especially small joints in the hands are affected by it. About 50% of patients with rheumatoid arthritis present with TMJ complaints. [67] TMJ involvement is more common in women.

Unlike osteoarthritis, both TMJs are affected. [68, 69] Resorption of the articular bone and anterior open bite may be seen in patients with rheumatoid arthritis. [70, 71]

2.1.5. Diagnostic Criteria for Temporomandibular Disorders

The use of different diagnostic criteria obstructs the accuracy and standardization of the research. The variability of the results reported in previous reviews could be due to differences in the clinical protocols used to diagnose TMDs.

In 1992, Dworkin et al. [72] published an article on the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD). RDC/TMD increased the level of uniformity between studies by means of the use of standardized diagnostic criteria.

The RDC/TMD provides a criterion for a dual-axis diagnosis. The Axis I questionnaires assess the patients' physical diagnosis, and the Axis II questionnaires assess pain-related disability and psychologic status. [72]

The RDC/TMD has been used in many studies since its publication in 1992. Nevertheless, the developers of the RDC/TMD indicated that there was a need to evaluate the Axis I physical diagnosis's criterion validity. The diagnostic criteria were compared to a standard reference for criterion validity, and it was determined that they needed to be improved. [73, 74] The Axis II questionnaires were found to be reliable and valid. [75] In 2012, a new diagnostic criterion called DC/TMD was reviewed and finalized.

Like the RDC/TMD, the DC/TMD consists of a dual-axis assessment. Accompanied by Axis I, Axis II was also improved. New instruments to assess pain behavior, psychological status, and psychosocial functioning were added to the new DC/TMD Axis II protocol. [50]

The DC/TMD provides clinicians and researchers with a standardized system that can be used for examining, diagnosing, and classifying TMD. When clinicians and researchers use the same criteria and taxonomy, it will allow an easier transfer of clinical questions and experiences into relevant research issues and research findings, which will make them more accessible to clinicians so that they can better diagnose and manage their patients. [76]

2.2. Fibromyalgia

FM syndrome, which has also been called fibrositis, fibromyositis, and myofibrositis, was named by Hench in 1976. Because it is not inflammatory but involves pain and tenderness, as illustrated by tender points, it was renamed as FM to better describe the condition. [77]

FM is defined as a chronic syndrome that results in widespread pain, fatigue, sleep disturbance, impaired cognitive function, and ANX. [78] Genetic, neurological,

psychological, sleep-related, and immunological factors are probable causes of FM. [79, 80]

In the literature, widespread body pain, fatigue, morning stiffness, headache, paresthesia, sleep disturbance, dry mouth, loss of libido, irritable bowel, joint hypermobility, reticular color changes in the skin, and TMJ dysfunction are complaints or symptoms reported by more than 25% of patients with FM. [81]

2.2.1. History of Fibromyalgia

FM was first described by Froriep [82] in 1843 as a rheumatic condition accompanied by tender points in the muscle. In 1904, Gowers [83] suggested that tender points in the muscle and rheumatisms were caused by inflammation in fibrous tissue, and thus, the term "fibrositis" was used for the first time.

In 1972, Smythe [84] described FM as widespread pain and tender points. In 1976, Hench [85] used the term FM for the first time, stating that the main problem is not inflammatory, and he suggested that FM would be a more appropriate term instead of fibrositis. [77] Yunus et al. [86] published the first controlled clinical study in 1989. Tender points and symptoms were confirmed in this study.

In 1990, the ACR defined the diagnostic criteria for FM. [5] Although these criteria were originally developed for research purposes only, they have been widely used as a diagnostic criteria in clinical practice, especially by rheumatologists. [87] Since the existing criteria were not sufficient, in 2010, a new criterion focusing on symptoms rather than the tender points was established by the ACR. [88] In 2013, due to the need for a more comprehensive diagnostic criteria, Bennett et al. [89] modified the existing diagnostic criteria. The 2010 modified ACR criteria was revised in 2016 following the feedback that it caused incorrect classifications. [90]

2.2.2. Epidemiology

The second most common "rheumatologic" disorder following osteoarthritis is FM. Most studies state that FM is prevalent in 2-4% of the population. [91-93] The symptoms are most commonly seen in the age range of 30-55 years. [94] Although prevalence increases with age, FM can also appear in children. [95, 96] Women are more commonly diagnosed than men with a 9:1 ratio. [91]

FM can appear in any ethnic group, age, and gender. This syndrome mostly affects women in the 30-55 age group and 85-90% of patients are women. [97]

2.2.3. Etiology

FM is a complex syndrome with a variety of symptoms and functional changes in many systems. [98] Patients with FM do not have rational structural or functional abnormalities in muscle tissue, but in the central nervous system, the pain processing mechanisms are dysfunctional.

It is not known exactly what causes FM, and its etiology is multifactorial. [99] Various factors are thought to play a role in its etiology, such as genetic, immunological, neurohormonal, environmental, and psychological factors as well as sleep disorders. [98]

Like other chronic pain syndromes, genetic factors play a role in the development of FM. [100] The risk of FM is 8 times higher in the first-degree relatives of patients with FM. [101] In addition, family members of patients with FM have a significantly higher number of tender points than controls and an increased risk of irritable bowel syndrome, TMD, headaches, and other chronic pain syndromes. [102] At the same time, twin studies showed that around 50% of the risk of developing FM and related conditions is genetic and 50% is environmental. [103]

The environmental factors most likely to trigger FM include stressors and some types of infections. Some psychological stressors may be childhood trauma, abuse, and daily life difficulties. [104, 105] Psychological stress, trauma, and deployment to war may also trigger FM. [106] Some types of infections that trigger FM are the Epstein-Barr virus, Lyme disease, Q fever, viral hepatitis. [103]

FM may also occur in patients who suffer from other chronic conditions such as osteoarthritis, rheumatoid arthritis, and lupus. About 10% to 30% of patients with these rheumatic disorders are affected by FM as well. [107]

2.2.4. Signs and Symptoms

Patients with FM mostly complain of chronic generalized pain, even though many patients suffer from accompanying symptoms, such as tiredness, morning stiffness, and sleepiness. [5]

The most specific symptom, pain, is characterized as a deep ache in the muscles with a more generalized burning, throbbing, tingling, intense, and persistent pain all over the body. [95, 108] Hyperalgesia and allodynia, which means pain due to an act that does not normally cause pain, such as wearing glasses, are common in FM patients. Furthermore, some patients experience paresthesia. [109]

Morning stiffness, which does not cause functional disability, is another common symptom. Bennett et al. stated that morning stiffness is among the four most important

symptoms among patients. In the same study, they also stated that morning stiffness decreases the quality of life. [89]

Moreover, fatigue, impaired cognition, and sleep difficulties were commonly observed. [108, 110] Patients complained about fatigue and weakness, especially in the morning, triggered by heavy exercise. 90% of FM patients reported non-restorative, non-refreshing sleep and, as a result, increased discomfort, fatigue, and a decrease in the pain threshold were observed. [111, 112]

Observations on abnormal parameters during sleep in FM patients were first published by Moldofsky et al. [6] Electroencephalography findings show alpha intrusion and a delta/alpha ratio decrease in patients with FM. Alpha in slow-wave sleep has been associated with myalgia and hyperalgesia; however, this is not specific to FM. It is possible to see this in psychological disorders and other chronic pain syndromes.

FM patients have impaired cognition, which is also called "fibro fog." [95, 113] They have poorer cognitive performance and decreased working memory and verbal fluency compared to similar-aged controls. [114] Several studies stated that the distraction of chronic pain and fatigue increases the impairment of cognition. [113]

Mood disorders, for example, DEP and ANX; stiffness; leg cramps; chronic headache; poor balance; dizziness; and hypersensitivity to noise, light, and chemicals are the other symptoms. Restless leg syndrome, irritable bowel and bladder, and TMJ disorders are syndromes that frequently occur with FM. [95, 115-117] Patients may also experience non-cardiac chest pains, Raynaud's phenomenon, muscle cramps, and dry mouth and eyes. [118]

2.2.5. Diagnostic Criteria for Fibromyalgia Syndrome

In 1990, the ACR criteria for FM were defined. These criteria include 3 or more months of wide-spread pain above and below the waist, on the right and left side of the body, and along the midline. Moreover, patients should notice pain from 4 kg of pressure in at least 11 of 18 specific tender points. (Figure 1)

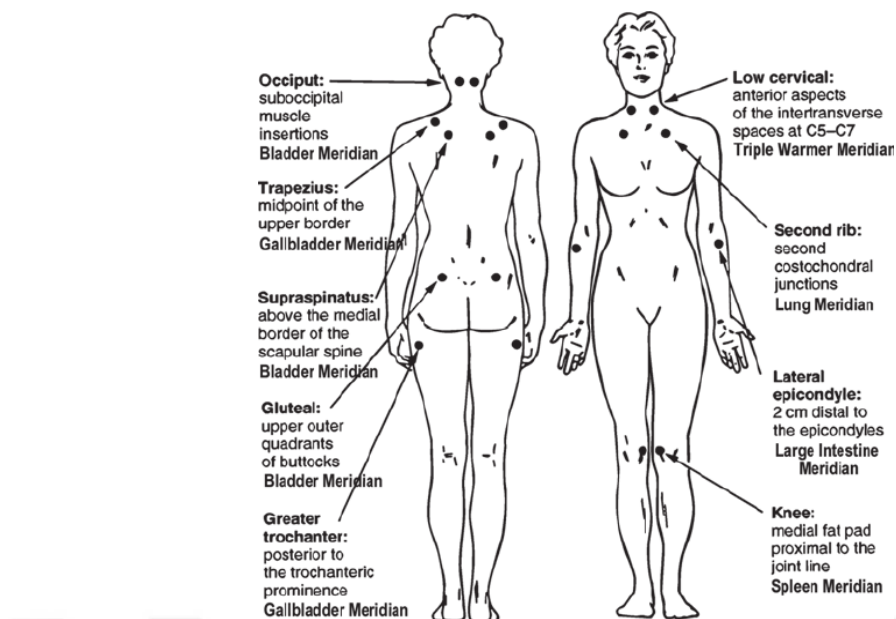


Figure 1 Tender Points of FM [119]

According to the ACR 1990 criteria, the presence of these two symptoms is adequate to diagnose FM. [5] However, many patients with FM do not have pain throughout their body or may not have at least 11 tender points. [120] In addition, the ACR 1990 diagnostic criteria helped to define FM, but it only focused on pain and not the other symptoms, for example, fatigue and morning stiffness. [95]

In brief, the ACR 1990 criteria is inadequate since it misses approximately 25% of the patients with FM and it does not clearly state how to categorize or assess these patients and because of the difficulty of the tender point examination.

In 2010, ACR published a new diagnostic criterion. In this new criterion, the ACR removed the tender points examination and added the assessment of fatigue, cognition, and other somatic symptoms. [88] The 2010 criterion was not conceived to replace the 1990 criteria but to assist in making a diagnosis in a general practice setting where a tender point examination is not routinely conducted. Because the tender point examination was no longer required, FM could be studied in surveys and clinical studies. [87]

These three criteria need to be met to diagnose FM:

- 1) Widespread Pain Index (WPI) ≥ 7 and Symptom Severity Score (SS) ≥ 5 , or WPI is 3-6 and SS ≥ 9 ;
- 2) the symptoms have been present at a similar level for at least 3 months;
- 3) the patient does not have a disorder that would otherwise explain the pain.

In 2019, Gur et al. [121] compared the ACR 1990 and 2010 classification criteria in the diagnosis of patients with FM, and the ACR 2010 classification criteria were found to be more sensitive than the ACR 1990 classification criteria. They thought that these criteria may be more favorable for clinical diagnosis and for monitoring the diagnosed disease. In 2011, Wolfe et al. [87] described the development and performance of a modified ACR 2010 criteria and a new FM symptom scale formed by the combination of the WPI and SS scales. They examined how the ACR 2010 SS scale could be best modified for survey research, and they applied the modified ACR 2010 criteria to patients surveyed in a longitudinal database. They also examined the performance and distribution of the FS scale across different rheumatic disorders. According to this diagnostic criterion, the newly created FS scale varies between 0 and 35, and if $(FS) \geq 13$, it is sufficient for the diagnosis of FM. [97]

Following the feedback that the 2010/2010 modified ACR caused misclassifications due to the removal of the tender point examination, "widespread pain, which is present in at least 4 or 5 regions," was added to the criteria as a revision in 2016. [90]

2.3. Temporomandibular Disorders and Fibromyalgia

Numerous studies have reported a link between FM and TMD in the past. [9, 11-13] Eriksson et al. [122] were the first to mention the link between FM and TMD; according to this study, FM patients also had TMD signs and symptoms. Other studies have also shown that patients with FM have a high prevalence of TMD signs and symptoms as well as significant orofacial involvement. [9-17, 123-129]

In comparison with patients with other TMD-related conditions, e.g. migraine and trigeminal neuralgia, several studies have further found a higher prevalence of TMDs in FM patients. [14] Ayouni et al. [125] explained this by stating that FM is defined by a polyalgic syndrome affecting multiple body regions and is not limited to a well-defined region.

Studies have shown that patients with FM have a high prevalence of TMD signs and symptoms in the range of 71% to 97%, and similarly, patients with TMD have a high prevalence of widespread pain in the range of 10% to 18.4%. According to these studies, it is obvious that patients with FM are more affected by TMD than the reverse. [7, 13-15, 17, 126-129] Furthermore, FM seems to have features that are both predisposing and triggering factors for TMD. [15, 17, 130]

Sensitivity in the palpation of TMJ and masticatory muscles, orofacial pain, muscular pain during opening and closing movements, constricted mouth opening, orofacial fatigue, masticatory problems, and increased trigger points and scores were considerably higher in patients with FM compared to healthy controls. [17, 125, 129, 130] A large percentage of FM patients reported facial pain and localized discomfort in the temporalis muscles, TMJ, jaws, neck, and ear. [9, 10, 14, 16] Muscle tenderness on palpation and masticatory muscle and TMJ pain were the most common symptoms of TMDs among patients with FM. The masseter and the temporalis muscles were reported as the most painful palpation areas. [123, 124]

FM and TMD have some clinical resemblances. Chronic progression, an unknown etiology, severe physical and psychological impacts, and common predisposing factors are among them. [125] Plenty of clinical research have affirmed the frequency of FM symptoms such as chronic headaches, aches, and stiffness in the morning, and ANX, DEP, stress, and exhaustion are frequently found among TMD patients. [15, 88]

On the other hand, FM and TMD patients have both reported a variety of general medical conditions, such as bowel complaints, DEP, muscle soreness, headaches, concentration difficulties, and ANX, which are commonly co-occurring in these disorders. [7, 10, 130, 131]

3. MATERIALS AND METHODS

3.1.Ethical Approval

The study protocol was approved by the Istanbul Medeniyet University Human Research Ethics Committee (#2020/0024) and carried out in accordance with the principles of the Declaration of Helsinki. (Appendix 1)

3.2.Sample Size

The sample size was determined with a power analysis based on previous studies. [13-15] As a result of a power analysis using G * Power program, effect size d: 0.683, SD: 8.2, Power: 0.80, and p: 0.05, the number of participants required was found to be a minimum of $n = 35$ for each group.

3.3.Informed Consent

All the participants were required to sign an informed consent, which briefly explained the purpose and the process of the thesis. This informed consent included the potential risks, benefits, and procedures of the study. It informed that the participant could leave the study at any time. According to this informed consent, the participant agreed that their data could be used for research purposes. (Appendix 2)

3.4.Participants

This case-control study was conducted from January 2020 to August 2020, and 82 patients were included. The patients who attended the rheumatology clinic in the Istanbul Medeniyet University Goztepe Research Hospital between January 2020 and August 2020 were consecutively examined by a rheumatologist (YY). FM was diagnosed in 212 out of the 640 patients who were examined. One hundred and forty-eight of these patients were excluded from the study because they had previously been diagnosed with FM and treated for it. Three were ruled out due to TMD treatment, and five were ruled out due to uncontrolled systemic diseases. Twelve of them refused to participate in the study, and 44 patients were eligible for this study. Three male patients who had agreed to participate in the study and were diagnosed with FM were not included in this study because the size of the male sample would be insufficient for comparison in terms of gender. Consequently, 41 female patients were included in the case group.

The participants in the control group were chosen from the dental hospital to avoid any other rheumatologic disease. The patients who attended the Yeditepe University Dental Hospital the Oral Diagnosis and Radiology Clinics between June 2020 and August 2020 were examined consecutively. Among 220 female and 142 male patients, 41 female patients were included in the control group. Due to the absence of male participants in

the case group, male patients were excluded from the control group as well. Due to uncontrolled systemic diseases and the medications they were taking, 96 of the 220 women were ruled out of the study. Only 52 of the remaining 124 patients agreed to take part in the research. A rheumatologist examined 52 patients, and three were excluded because FM was diagnosed using the ACR criteria. Eight of them desired to leave the study later. As a result, the control group consisted of 41 female patients who had agreed to participate in the study and were without uncontrolled systemic disease and FM.

As a consequence, this study included 41 women who were diagnosed with FM according to ACR criteria and 41 women who were not diagnosed with FM.

3.4.1. Inclusion criteria

The patients who agreed to participate in the study were included. In the case group, the patients who were diagnosed with FM for the first time by a rheumatologist at the Istanbul Medeniyet University Goztepe Research Hospital were included.

In the control group, patients with no known TMD and FM who applied to the Yeditepe University Dentistry Faculty were included.

3.4.2. Exclusion criteria

In both the groups, the following types of patients were excluded:

- patients with primary headaches
- patients with neurological disorders
- patients with ear infections
- patients with uncontrolled systemic diseases
- patients with previously treated and diagnosed TMD
- patients who use medications that might influence ANX or response to pain such as muscle relaxants, non-steroidal anti-inflammatory drugs, anticonvulsants, antidepressants, hypnotics, and anxiolytics

In the control group, the following types of individuals were excluded:

- individuals with musculoskeletal pain
- individuals who were previously diagnosed with FM according to the ACR criteria

3.5. Questionnaires and Clinical Examination

The study was based on demographic data (age, gender, and marital status), DC/TMD Axis I and Axis II, SCL-90-R, OHIP-14, PSQI, and clinical examination. Personal data also included medical history, daily medications taken, previous surgeries, and the presence of toothaches or headaches.

Before the examination, the participants were asked to complete all the questionnaires without any time restriction. In the second part of the study, all the individuals were clinically examined by a dentist (G.S.) to identify TMD and by a rheumatologist (Y.Y.) to identify FM.

In the first part of the study, all the patients completed 4 questionnaires: DC/TMD Axis II, SCL-90-R, OHIP-14, and PSQI. Validated full Turkish versions of the questionnaires were used. [132-134]

The clinical examination comprised intra-oral examinations for bruxism signs and TMD examinations. For TMD diagnosis, a systematically translated Turkish version of the DC/TMD was used. The DC/TMD is a dual-axis diagnostic system.

3.5.1. Diagnostic Criteria for Temporomandibular Disorder Axis I—physical assessment

Axis I is the physical assessment part, and it is used for clinical TMD diagnosis. It includes occlusal assessment, palpation of the masticatory muscles and articulation, examination of abnormal oral behavior and morning signs and symptoms. The validity of the Axis I diagnostic criteria was not improved by occlusal findings. Occlusal status can change with treatment, so occlusal evaluation is suggested.

Odontogenic disease and other pain disorders that can occur in the masticatory system must be ruled out prior to using Axis I diagnoses. When numbness, swelling, and redness are present, the clinician must rule out clinically significant disease, such as infections, neoplasms, and systemic conditions. Also, the doctor must take into consideration uncommon TMDs.

For the routine screening of pain related TMD the TMD Pain Screener is recommended. It helps healthcare providers identify patients who have pain related TMD, and who may require treatment. It additionally recognizes patients who might be at increased risk of worsening their pain during dental procedures.

Because of the inadequacy of criterion validity, Schiffman et al. [135] stated that the diagnostic criteria for TMJ intra-articular disorders except DD without reduction with limited opening, can only be used for screening. TMJ MRI for DD and TMJ CT for DJD are required for definitive diagnosis.

In this study, all the patients were examined by a single dentist (G.S.). All the individuals were requested to complete the validated symptom questionnaire and the TMD pain screener and were subjected to a clinical examination in accordance with the DC/TMD Axis I guidelines. To collect data for specific DC/TMD Axis I diagnoses, the

DC/TMD Symptom Questionnaire was recommended for history taking. The TMD pain diagnosis (Figure 2) and intra-articular diagnoses (Figure 3) have clearly defined clinical examination specifications and decision trees. [135]

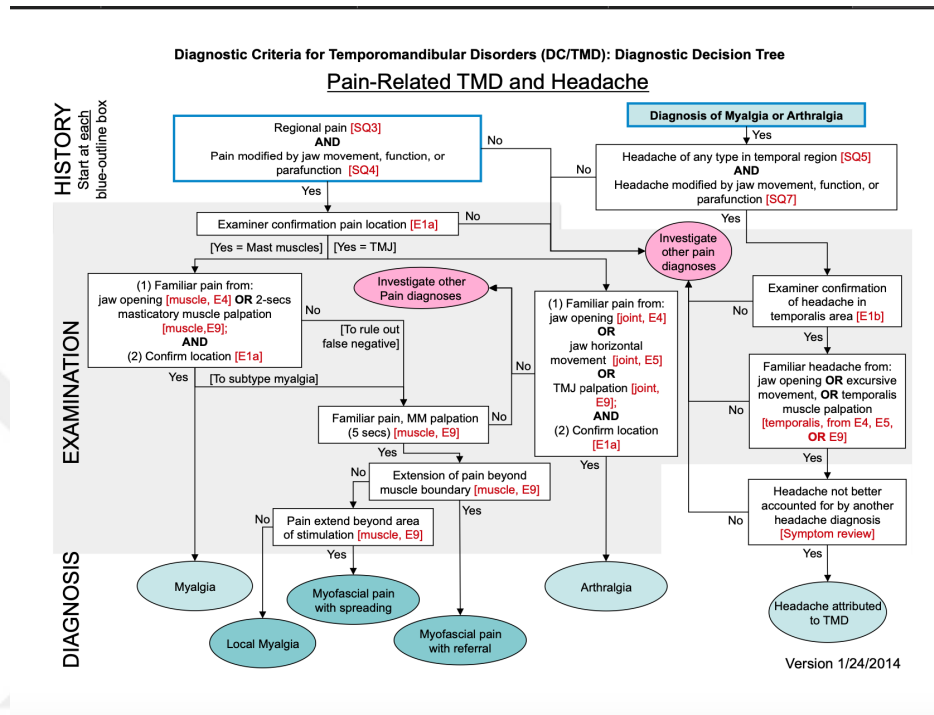


Figure 2 DC/TMD Diagnostic Decision Tree 1

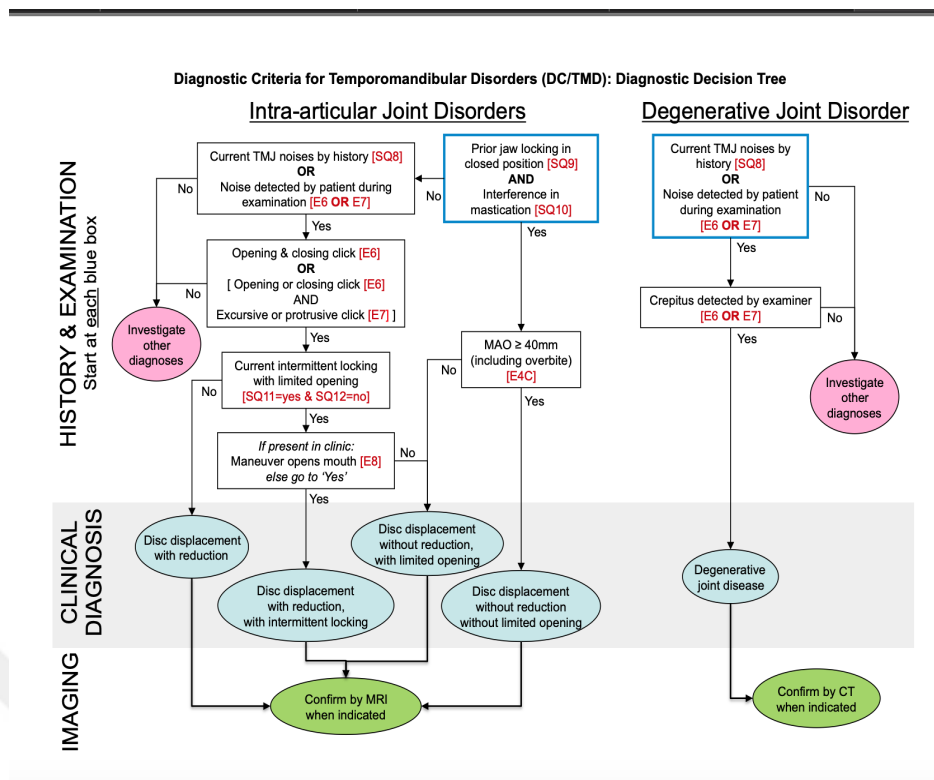


Figure 3 DC/TMD Diagnostic Decision Tree 2

3.5.2. Diagnostic Criteria for Temporomandibular Disorder Axis II

Axis II includes self-reported psychosocial and behavioral screening questionnaires. Five Axis II questionnaires were recommended for evaluating the psychosocial and behavioral factors that can affect a patient's response to treatment. These questionnaires were the Graded Chronic Pain Scale (GCPS), pain drawing, the JFLS, the Patient Health Questionnaire (PHQ-4), and the Oral Behaviors Checklist. In addition to these questionnaires, Axis II also includes PHQ-9, PHQ-15, and GAD-7.

The GCPS assesses the severity of pain and pain-related disability. The JFLS assesses functional limitation and complements the GCPS findings while also assessing how jaw mobility affects mastication and verbal and emotional expression.

The JFLS-20 (Table 3) was used to measure the degree of limitation depending on the success of 20 activities over the previous month on an 11-point numerical rating scale with 0 (no limitation) and 10 (severe limitation). The JFLS identifies the stomatognathic system's entire functional limitation in three dimensions: mastication, vertical mobility, and communication. It also quantifies the general limitation of jaw function, which is calculated as the average of the three dimensions. The global functional

limitation score was also calculated using a short form scale with eight activities (JFLS-8).

The PHQ-4 identifies psychological distress, such as ANX and DEP, which may affect the treatment process, according to the recommended Axis II questionnaires. When a patient's PHQ-4 (Table 4) score is higher than a certain cutoff point, additional psychological tests are recommended. Instead of using the PHQ-4, a more detailed approach to assessing distress includes using the PHQ-9 (Table 5) for depressive symptoms and the GAD-7 (Table 6) for ANX symptoms.

PHQ-9 is a nine-item self-rating scale used to determine the occurrence and severity of depressive symptoms. PHQ-9 provides a total score that ranges from zero to 27 for determining the severity of DEP. Mild DEP is indicated by a score of five to nine, moderate DEP by a score of 10–14, moderately severe DEP by a score of 15–19, and severe DEP by a score above 20. [136]

GAD-7 is a seven-item self-rating scale screening to measure ANX. The GAD-7 total score can be interpreted as the cut-off scores of 5, 10, and 15, with a maximum possible score of 21, representing mild, moderate, and severe ANX levels, respectively. [50, 135, 137, 138]

Table 3 The JFLS-20

Çenenin Fonksiyon Kısıtlanma Skalası – 20

Son bir ay boyunca, aşağıdaki her bir madde için kısıtlanma düzeyini belirtiniz. Eğer çok zor olduğu için aktiviteden tamamen kaçınılıyorsa 10'u daire içine alınız. Eğer aktivite ağrı ya da zorluktan başka bir sebepten dolayı yapılamadıysa boş bırakınız.

		Kısıtlama yok										Ciddi kısıtlanma
1.	Sert besin çiğnemek	0	1	2	3	4	5	6	7	8	9	10
2.	Sert ekmek çiğnemek	0	1	2	3	4	5	6	7	8	9	10
3.	Tavuk çiğnemek (ör. fırında hazırlanmış)	0	1	2	3	4	5	6	7	8	9	10
4.	Kraker çiğnemek	0	1	2	3	4	5	6	7	8	9	10
5.	Yumuşak besin çiğnemek (ör. makarna, konserve veya yumuşak meyveler, pişmiş sebzeler, balık)	0	1	2	3	4	5	6	7	8	9	10
6.	Çiğneme gerektirmeyen yumuşak besin yemek (ör. patates püresi, elma sosu, puding, püre yapılmış besin)	0	1	2	3	4	5	6	7	8	9	10
7.	Bütün bir elmayı ısırabilecek kadar geniş açmak	0	1	2	3	4	5	6	7	8	9	10
8.	Bir sandviçi ısırabilecek kadar geniş açmak	0	1	2	3	4	5	6	7	8	9	10
9.	Konuşacak kadar geniş açmak	0	1	2	3	4	5	6	7	8	9	10
10.	Bir fincandan içebilecek kadar geniş açmak	0	1	2	3	4	5	6	7	8	9	10
11.	Yutkunmak	0	1	2	3	4	5	6	7	8	9	10
12.	Esnemek	0	1	2	3	4	5	6	7	8	9	10
13.	Konuşmak	0	1	2	3	4	5	6	7	8	9	10
14.	Şarkı söylemek	0	1	2	3	4	5	6	7	8	9	10
15.	Mutlu bir yüz ifadesi takınmak	0	1	2	3	4	5	6	7	8	9	10
16.	Sinirli bir yüz ifadesi takınmak	0	1	2	3	4	5	6	7	8	9	10
17.	Somurtmak	0	1	2	3	4	5	6	7	8	9	10
18.	Öpüşmek	0	1	2	3	4	5	6	7	8	9	10
19.	Gülümsemek	0	1	2	3	4	5	6	7	8	9	10
20.	Kahkaha atmak	0	1	2	3	4	5	6	7	8	9	10

Telif hakkı Ohrbach R.'e aittir. Çeviri; Polat S, Polat NT, Çetinoğlu A tarafından yapılmıştır. <http://www.rdc-tmdinternational.org> da mevcuttur. Sürüm 12 Mayıs 2013. Çoğaltmak, tercüme etmek, görüntülemek veya dağıtmak için gerekli değildir.

Table 4 Patient Health Questionnaire-4

Hasta Sağlık Anketi - 4

Son 2 hafta boyunca, aşağıdaki problemlerden dolayı ne sıklıkla sorun yaşadınız? Lütfen cevabınızı belirten kutuya bir onay işareti koyunuz.

	Hiç 0	Birkaç gün 1	Günlerin yarısından fazla 2	Neredeyse her gün 3
1. Endişeli, huzursuz ya da tedirgin hissetmek	☐	☐	☐	☐
2. Tasalanmayı durduramamak ya da kontrol edememek	☐	☐	☐	☐
3. Bir şeyler yapmaya az ilgi duymak ya da yapmaktan az zevk almak	☐	☐	☐	☐
4. Moralsiz, depresif veya umutsuz hissetmek	☐	☐	☐	☐

TOPLAM PUAN =

Eğer herhangi bir problem işaretlediyseniz, bu problemler işinizi yapmanızı, evde bir şeylerle ilgilenmenizi veya diğer insanlarla geçinmenizi ne kadar zorlaştırdı?

Hiç zorlaştırmadı	Biraz zorlaştırdı	Çok zorlaştırdı	Aşırı zorlaştırdı
☐	☐	☐	☐

Telif hakkı Pfizer Inc.'e aittir. Çeviri: Polat S, Polat NT, Çelinoğlu A tarafından yapılmıştır. Çöğürüne çevirmeye, görüntülenmek veya dağıtmak için izin gerekli değildir. Kayna: <http://www.abnsgreeners.com/> da mevcuttur. Konsorsiyum sürümünü 12 Mayıs 2013, <http://www.rdc.tinduterra.ionol.org/> da mevcuttur

Table 5 Patient Health Questionnaire-9

Hasta Sağlık Anketi - 9

Son 2 hafta boyunca, aşağıdaki problemler sizi ne sıklıkla rahatsız etti? Lütfen cevabınızı gösteren kutuya bir onay işareti koyunuz.

	Hiç 0	Birkaç gün 1	Günlerin yarısından fazla 2	Neredeyse her gün 3
1. Bir şeyler yapmaya az ilgi duymak ya da yapmaktan az zevk almak	☐	☐	☐	☐
2. Moralsiz, depresif veya umutsuz hissetmek	☐	☐	☐	☐
3. Uykuya dalmada veya uyku halini sürdürmede zorlanmak ya da gereğinden fazla uyumak	☐	☐	☐	☐
4. Yorgun hissetmek veya enerjinizin az olması	☐	☐	☐	☐
5. İştahsızlık ya da aşırı yeme	☐	☐	☐	☐
6. Kendinizi kötü veya başarısız hissetmek ya da kendinizi veya ailenizi hayal kırıklığına uğrattığınızı düşünmek.	☐	☐	☐	☐
7. Gazete okumak ya da televizyon seyretmek gibi faaliyetlerde dikkatinizi toplamakta güçlük çekmek	☐	☐	☐	☐
8. Diğer insanların fark edeceği kadar yavaş hareket etmek ya da konuşmak. Ya da tam tersi - normalde olduğunuzdan daha fazla hareket ederek, kıpır kıpır olmak ve yerinde duramamak.	☐	☐	☐	☐
9. Ölmüş olsanız daha iyi olacağınızı düşünmek ya da kendinize bir şekilde zarar vermek istediğinizi düşünmek	☐	☐	☐	☐

TOPLAM PUAN =

Eğer herhangi bir problem işaretlediyseniz; bu problemler, işinizi yapmanızı, evde bir şeylerle ilgilenmenizi veya diğer insanlarla geçinmenizi ne kadar zorlaştırdı?			
Hiç zorlaştırmadı	Biraz zorlaştırdı	Çok zorlaştırdı	Aşırı zorlaştırdı
☐	☐	☐	☐

Telif hakkı Pfizer Inc.'e aittir. Çeviri; Polat S, Polat NT, Çetinoğlu A tarafından yapılmıştır. Çoğaltmak, çevirmek, görüntülemek veya dağıtmak için izin gerekli değildir. Kaynak atıfı için: <http://www.phqscreeners.com/> da mevcuttur. Konsorsiyum sürümü 12 Mayıs 2013. <http://www.edc.tmdinternational.org/> da mevcuttur.

Table 6 GAD-7

GAD - 7

Son 2 hafta boyunca, aşağıdaki problemlerden dolayı ne sıklıkla sorun yaşadınız? Lütfen cevabınızı belirten kutuya bir onay işareti koyunuz.

	Hiç 0	Bazı günler 1	Günlerin yarısından fazla 2	Neredeyse her gün 3
1. Endişeli, huzursuz ya da tedirgin hissetmek	☐	☐	☐	☐
2. Tasalanmayı durduramamak ya da kontrol edememek	☐	☐	☐	☐
3. Farklı şeyler hakkında çok fazla endişe duymak	☐	☐	☐	☐
4. Rahatlamada zorluk çekmek	☐	☐	☐	☐
5. Yöründe durmakta zorlanacak kadar huzursuz olmak	☐	☐	☐	☐
6. Kolayca rahatsız olmak veya asabileşmek	☐	☐	☐	☐
7. Korkunç bir şey olacaktı gibi korku hissetmek	☐	☐	☐	☐
TOPLAM PUAN =				

Eğer herhangi bir problem işaretlediyseniz, bu problemler işinizi yapmanızı, evde bir şeylerle ilgilenmenizi veya diğer insanlarla geçinmenizi ne kadar zorlaştırdı?

Hiç zorlaştırmadı	Biraz zorlaştırdı	Çok zorlaştırdı	Aşırı zorlaştırdı
☐	☐	☐	☐

3.5.3. Bruxism

The presence of noises associated with nocturnal teeth grinding, as reported by a relative, or clenching the teeth during the day, frequent awakenings at night while grinding or clenching, or a feeling of muscular tension and/or stifling, the presence of wear facets on the teeth, tongue scallop, and linea alba are the signs and symptoms for bruxism according to previous literature. [139-143] The patients were classified as having bruxism if at least one clinical sign and one symptom were positive.

3.5.4. Revised Symptom Checklist-90

In 1977, Derogatis et al. [144] introduced the SCL-90-R. It is a brief self-report psychometric questionnaire. SCL-90-R, developed using the Hopkins Symptom Checklist, is an instrument that measures the level of psychological symptoms in individuals and the dimensions in which these symptoms are related.

Internal consistency ranges from 0.77 to 0.90, and test-retest reliabilities range from 0.78 to 0.90. SCL-90-R was translated into Turkish, and validity and reliability studies were conducted. In 1991, Crohnbach's Alpha was found to be 0.97 by Dag. [133]

A patient's psychological status was determined by administering the SCL-90-R to them. The SCL-90-R is a 90-item self-report instrument that consists of nine primary symptom dimensions, including SOM, O-C, interpersonal sensitivity (I-S), DEP, ANX, HOS, phobic anxiety (PHOB), PAR, and PSY. In addition to these nine dimensions, there is an additional subscale relating to appetite and sleep disorders. It also uses the Global Severity Index (GSI) for general psychological distress, which is the mean score of all the responses.

The responses to SCL-90-R were scored with a five-point Likert scale. Each item was rated from 0 (none) to 4 (extreme). In the questionnaires, the individuals were instructed to relate their experiences from the last seven days including the day when they answered the questionnaire. The mean for each scale was calculated and considered to be positive if the mean was one or greater. [144]

3.5.5. Oral Health Impact Profile

Oral health-related quality of life (OHRQoL), which is measured by oral health impact profile (OHIP), is a model that focuses on the patient's perception of oral health. Slade et al. [145] developed the OHIP, which is the most commonly used OHRQoL instrument. Seven conceptually formulated dimensions are captured by the 49 questions in OHIP which based on Locker's [146] conceptual model of oral health. While the OHIP-49 is designed to provide detailed information on perceptions of impact on well-being,

use of the instrument's 49 questions is not allowed in some research settings. To give one specific example, health services researchers often prefer a shorter instrument OHIP-14, the shortened form of OHIP-49, is a valid and reliable instrument. It is appropriate to assess aspects of disability in patients suffering from orofacial pain and TMD. [147, 148]

The OHIP translation and validation study has been done in various languages. The validated Turkish version of OHIP-14 (Table 7) was used to assess the OHRQoL. [134] It contains seven OHRQoL domains: functional limitation, physical pain, psychological discomfort, physical disability, psychological disability, social disability, and handicap. It consists of 14 questions translated from English. For each question in the OHIP-14, people were asked how often they experienced the impact over the past six months. The responses were graded on a five-point ordinal scale as follows: zero (never), one (hardly ever), two (occasionally), three (fairly often), and four (very often). The total score varies from zero, as the minimum, to 56, as the maximum, and was calculated by summing the scores of the 14 items. A higher global score of OHIP-14 indicates a lower quality of life related to oral health. [145]

Table 7 OHIP-14

Ağızındaki, protezinizdeki veya dişlerinizdeki problemlerden dolayı;	Hiçbir zaman	Nadiren	Bazen	Sıklıkla	Her zaman
Kelimeleri telaffuz etmede zorluk çekiyor musunuz?					
Tat alma hissiniz kötüleşti mi?					
Ağızınızda size rahatsızlık veren ağrınız oluyor mu?					
Yemek yemede zorluk çekiyor musunuz?					
Dişleriniz veya ağızınızın farkında olarak mı yaşıyorsunuz?					
Kendinizi gergin hissediyor musunuz?					
Beslenmeniz sizi tatmin etmiyor mu?					
Yemeğe ara vermek zorunda kalıyor musunuz?					
Sakinleşmede/rahatlamada zorluk çekiyor musunuz?					
Biraz utanıyor musunuz?					
Diğer insanların yanında rahatsızlık duyuyor musunuz?					
Günlük işlerinizi yapmada zorluk çekiyor musunuz?					
Yaşantınızın daha az tatmin edici olduğunu mu hissediyorsunuz?					
İşlerinizi/görevlerinizi tamamen yapamaz durumda mısınız?					

3.5.6. Pittsburgh Sleep Quality Index

The PSQI was developed in 1989 by Buysse et al. [149] The PSQI was designed for clinical use as a simple and valid assessment of both sleep quality and disturbances that might affect sleep quality. The validity and reliability study of the Turkish version (Table 8) was conducted by Ağargün et al. in 1996, and Crohnbach's alpha was found to be 0.804. [132]

Nine out of the 10 questions included in the PSQI are self-report questions that measure the extent to which the respondent experienced a particular symptom during the last 30 days. The last question was answered by the spouse or roommate and was not included in the scoring.

By means of objective answers, seven components were evaluated: sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, use of sleeping medications, and daytime dysfunction. Each component was evaluated at a point range of zero to three. All the points were added together to obtain the final score. The final score was from zero to 21 points, and a higher score indicated poor sleep quality. Patients with a total PSQI score greater than 5 were considered poor sleepers. [149]

Table 8 PSQI

Pittsburgh Uyku Kalite İndeksi (PUKi)

Pittsburgh Sleep Quality Index (PSQI)

Hastanın Adı Soyadı: _____ Tarih: ____/____/____

Aşağıdaki sorulara vereceğiniz cevaplar için son bir ayı göz önünde bulundurun.
Lütfen tüm soruları cevaplandırın.

- 1 Geçen ay geceleri genellikle ne zaman yattınız? _____
- 2 Geçen ay geceleri uykuya dalmanız genellikle ne kadar zaman (dakika) aldı? _____ dakika
- 3 Geçen ay sabahları genellikle ne zaman kalktınız? _____
- 4 Geçen ay geceleri kaç saat uyudunuz (bu süre yatakta geçirdiğiniz süreden farklı olabilir) _____ saat
- 5 Geçen ay aşağıdaki durumlarda belirtilen uyku problemlerini ne sıklıkla yaşadınız?

	Haftada	Hiç	1'den az	1 - 2 kez	3'ten çok
a 30 dakika içinde uykuya dalamadınız	☐	☐	☐	☐	☐
b Gece yarısı veya sabah erkenden uyanınız	☐	☐	☐	☐	☐
c Tuvalete gittiniz	☐	☐	☐	☐	☐
d Rahat bir şekilde nefes alıp veremediniz	☐	☐	☐	☐	☐
e Aşırı derecede üşüdünüz	☐	☐	☐	☐	☐
f Aşırı derecede sıcaklık hissettiniz	☐	☐	☐	☐	☐
g Kötü rüyalar gördünüz	☐	☐	☐	☐	☐
h Ağrı duydunuz	☐	☐	☐	☐	☐
i Diğer nedenler	☐	☐	☐	☐	☐
j Öksürdünüz veya gürültülü bir şekilde horladınız	☐	☐	☐	☐	☐
- 6 Geçen ay uyku kalitenizi bütünü ile nasıl değerlendirirsiniz.

☐ Çok iyi	☐ Oldukça iyi	☐ Oldukça kötü	☐ Çok kötü
----------------------------------	--------------------------------------	---------------------------------------	-----------------------------------
- 7 Geçen ay uyumanıza yardımcı olması için ne sıklıkta (reçeteli veya reçetesiz) uyku ilacı aldınız?

☐ Hiç	☐ Haftada 1'den az	☐ Haftada 1 - 2 kez	☐ Haftada 3'ten çok
------------------------------	---	--	--
- 8 Geçen ay araba sürerken, yemek yerken veya sosyal bir aktivite esnasında ne kadar sıklıkla uyanık kalmak için zorlandınız?

☐ Hiç	☐ Haftada 1'den az	☐ Haftada 1 - 2 kez	☐ Haftada 3'ten çok
------------------------------	---	--	--
- 9 Geçen ay bu durum işlerinizi yeteri kadar istekle yapmanızda ne derecede problem oluşturdu?

☐ Hiç problem oluşturmadi	☐ Bir dereceye kadar problem oluşturdu
☐ Yalnızca çok az bir problem oluşturdu	☐ Çok büyük bir problem oluşturdu
- 10 Bir yatak partneriniz veya oda arkadaşınız var mı?

☐ Bir yatak partneri veya oda arkadaşı yok	☐ Partneri aynı odada fakat aynı yatakta değil
☐ Diğer odada bir partneri veya oda arkadaşı var	☐ Partner aynı yatakta
- 11 Eğer bir oda arkadaşı veya yatak partneriniz varsa son bir ayda ona aşağıdaki durumları ne sıklıkta yaşadığınızı sorun.

	Haftada →	Hiç	1'den az	1 - 2 kez	3'ten çok
a Gürültülü horlama	☐	☐	☐	☐	☐
b Uykuda nefes alıp verme arasında uzun aralıklar	☐	☐	☐	☐	☐
c Uyurken bacaklarda seğirme veya sıçrama	☐	☐	☐	☐	☐
d Uyku esnasında uyumsuzluk veya şaşkınlık	☐	☐	☐	☐	☐
e Diğer huzursuzluklarınız	☐	☐	☐	☐	☐

Boyse DJ, Reynolds CF 3rd, Monk TH (1989) Psychiatry Res. 1989 May;28(2):193-213



www.ftronline.com

Skorlama yönergesine
ftronline.com 'dan
ulaşabilirsiniz.

Tasarım ve düzenleme: Dr. Ender Salbaş 2019

3.6. Statistical Analysis

Statistical analysis was performed with SPSS version 17.0 software (IBM SPSS, Turkey). The descriptive data were expressed as mean, standard deviation, median, and minimum-maximum values. The variables were tested in relation to a normal distribution using histogram graphics and the Kolmogorov-Smirnov test. Categorical variables were compared using the Pearson Chi-Square Test. The Mann-Whitney U Test was used to evaluate the nonparametric variables between two groups, and the Kruskal Wallis Test was used to evaluate variables between more than two groups. The Spearman Correlation Test was used to measure the association between quantitative variables. The significance level was set at $p < 0.05$.



4. RESULTS

Eighty-two women between the ages of 32 and 65 participated in this study. The case group included 41 FM-positive women based on the ACR criteria. The average age of the case group was 49.53 (± 8.22), and the average age of the 41 healthy individuals in the control group was 47.34 (± 7.55). 11 (13.4%) of the participants were never married, 62 (75.6%) were married, and 9 (11%) were divorced. (Table 9)

Table 9 Demographic Characteristics of the Sample

		Case Group	Control Group
Gender	Female	41 (100%)	41 (100%)
	Male	0	0
Marital Status	Never married	5 (12.2%)	6 (14.6%)
	Married	31 (75.6%)	31 (75.6%)
	Divorced	5 (12.2%)	4 (9.8%)
Age	Mean $\pm$ SD	49.53 $\pm$ 8.22	47.34 $\pm$ 7.55
	Minimum	32	35
	Maximum	65	62

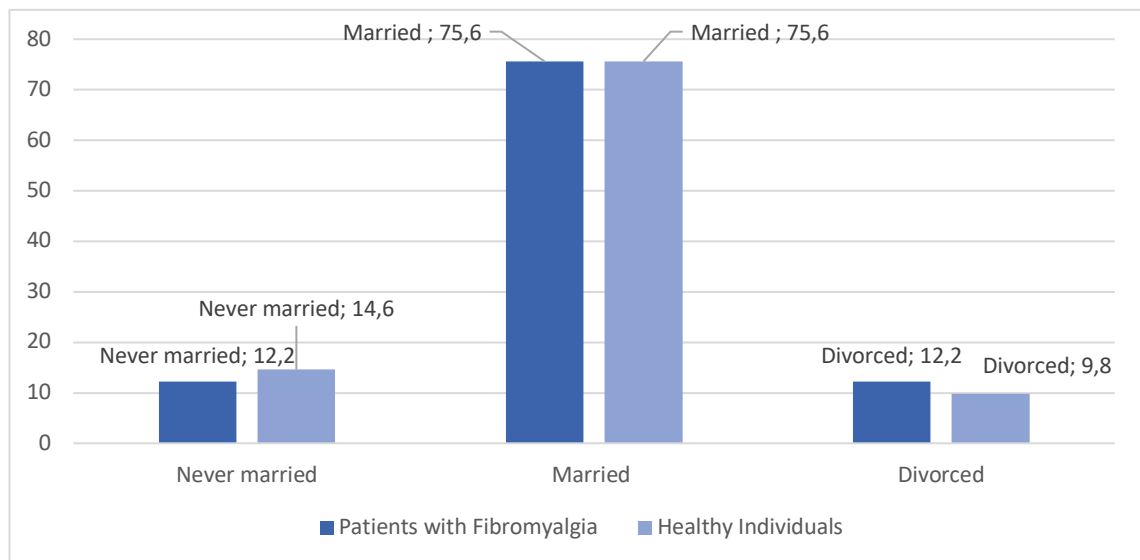


Figure 4 Marital Status in the Case and Control Groups.

Sixty-one percent of the individuals met the criteria of at least one DC/TMD diagnosis. TMD was found in 75.6 % of the case group and 46.3 % of the control group. ($p = 0.007$). DD was present in 53.7% of cases and 26.8% of controls, consecutively. This result indicated significant prevalence ($p = 0.013$). The results showed that 40.2% of participants had myalgia. In the case group, myalgia was identified in 48.8% of the patients compared with 31.7% of the control group. The results were not statistically significant ($p = 0.115$). Arthralgia was observed in 18.3% of the participants; meanwhile, this rate was 22% in the case group and 14.5% in the control group. There was no significant difference ($p = 0.391$). Bruxism signs were observed in 48.8% of the individuals: 68.3% of the case group and 29.3% of the control group. The prevalence of bruxism in the case group was significantly higher than in the control group ($p < 0.001$) (Table 10).

Table 10 Comparison of DC/TMD Diagnoses Between the Case Group and Control Group.

Chi-Square Test, $p < 0.05$

		FM				p
		Absent		Present		
		n	%	n	%	
TMD	Absent	22	53.66	10	24.39	0.007
	Present	19	46.34	31	75.61	
DD	Absent	30	73.17	19	46.34	0.013
	Present	11	26.83	22	53.66	
Degenerative Joint Disease	Absent	41	100	40	97.6	0.314
	Present	0	0	1	2.4	
Myalgia	Absent	28	68.29	21	51.22	0.115
	Present	13	31.71	20	48.78	
Arthralgia	Absent	35	85.4	32	78	0.391
	Present	6	14.6	9	22	
Bruxism	Absent	29	70.73	13	31.71	< 0.001
	Present	12	29.27	28	68.29	

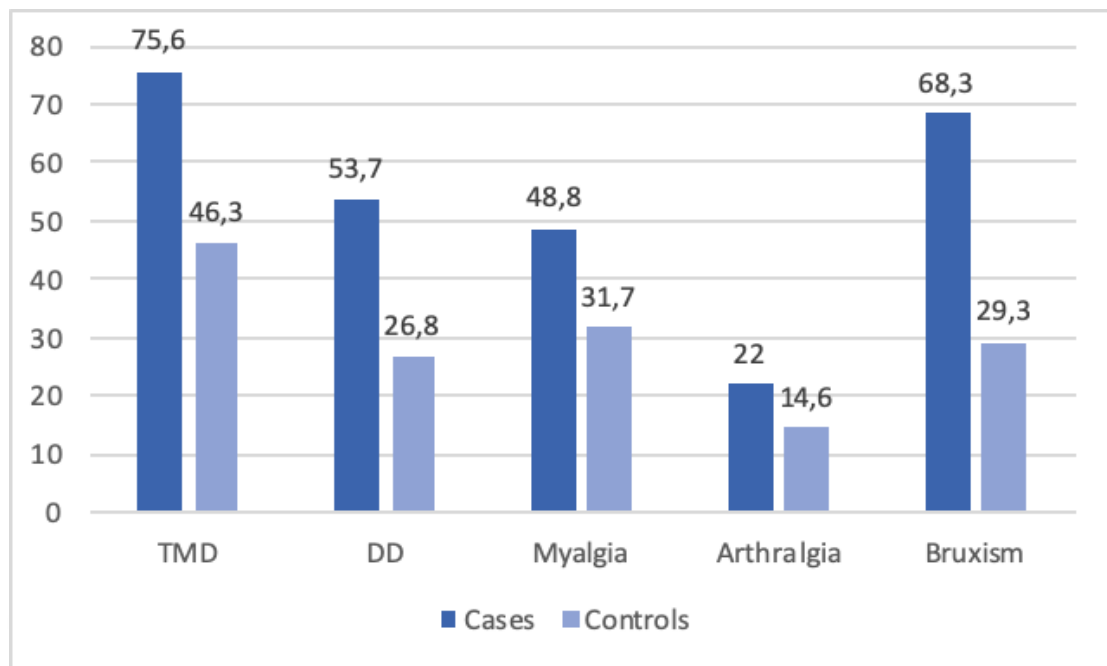


Figure 5 Prevalence of DC/TMD Diagnosis in the Case and Control Groups.

The results indicated a significant prevalence of FM, bruxism signs, and trauma history in the individuals with TMD compared to TMD-negative participants: 62% of TMD subjects and 38% of non-TMD participants had FM ($p=0.007$). Bruxism signs were present in 62% of the TMD patients as opposed to 28.1% of the individuals without TMD ($p=0.003$). 40% of TMD patients reported trauma history as compared to 9.4% of TMD-negative participants ($p = 0.003$) (Table 11).

Table 11 Comparison of FM and Bruxism Diagnoses and Trauma History Between Patients with and without TMD.

Chi-Square Test, $p < 0.05$

		TMD				p
		Absent		Present		
		n	%	n	%	
FM	Absent	22	68.75	19	38.00	0.007
	Present	10	31.25	31	62.00	
Bruxism	Absent	23	71.88	19	38.00	0.003
	Present	9	28.13	31	62.00	
Trauma	Absent	29	90.63	30	60.00	0.003
	Present	3	9.38	20	40.00	

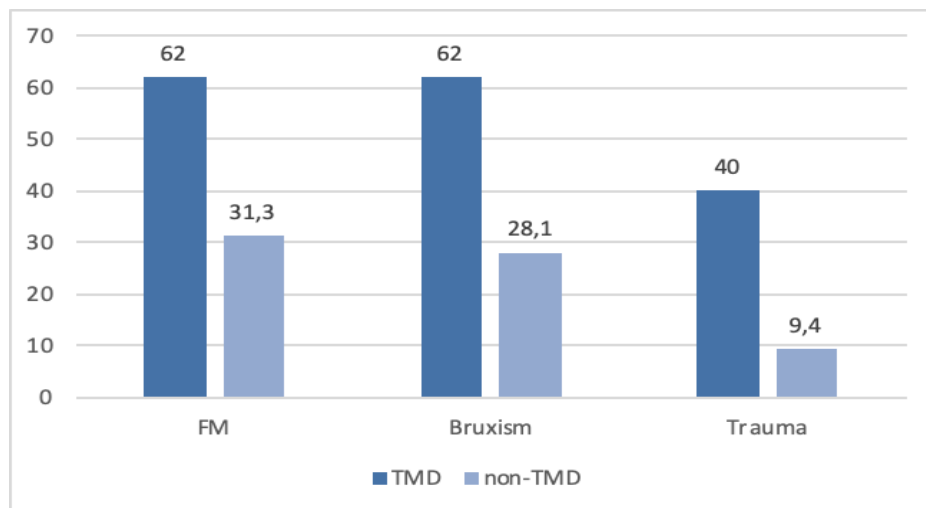


Figure 6 Prevalence of FM, Bruxism, and Trauma History in Patients With and Without TMD.

There was no difference between the groups according to age (Table 12). For the total study sample, there was no correlation between individuals' age and the questionnaires (Table 13).

Table 12 Comparison of the Mean Age Between Cases and Controls.

Independent-Samples T-Test, $p < 0.05$

		Age	p
		Mean $\pm$ SD	
FM	Absent	47.34 $\pm$ 7.55	0.212
	Present	49.54 $\pm$ 8.22	
TMD	Absent	48.69 $\pm$ 7.84	0.822
	Present	48.28 $\pm$ 8.05	
DD	Absent	48.63 $\pm$ 7.21	0.789
	Present	48.15 $\pm$ 8.99	
Myalgia	Absent	48.63 $\pm$ 8.23	0.786
	Present	48.15 $\pm$ 7.57	
Arthralgia	Absent	48.28 $\pm$ 7.58	0.710
	Present	49.13 $\pm$ 9.56	
Bruxism	Absent	46.86 $\pm$ 7.39	0.064
	Present	50.10 $\pm$ 8.21	

Table 13 Correlation Between Age and PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Scores.

Spearman's Correlation

	Age	
	r	p
PSQI	0.163	0.143
OHIP	-0.133	0.235
SOM	0.033	0.768
ANX	0.045	0.690
O-C	0.098	0.382
DEP	0.140	0.210
I-S	0.037	0.739
PSY	0.007	0.953
PAR	-0.023	0.837
HOS	0.099	0.374
PHOB	0.090	0.419
ADD.S	-0.118	0.290
GSI	0.052	0.642
JFLS-8	-0.015	0.894
JFLS-20	-0.013	0.905
PHQ-4	0.017	0.876
PHQ-9	-0.020	0.856
GAD-7	0.020	0.857

No significant differences were found between the groups according to marital status (Table 14). The marital status of the patients and the questionnaires were compared, and no significant difference was found (Table 15).

Table 14 Comparison of Marital Status Between the Case and Control Groups.

Chi-Square Test, $p < 0.05$

		Marital Status						p
		Never married		Married		Divorced		
		n	%	n	%	n	%	
FM	Absent	6	54.6	31	50	4	44.4	0.904
	Present	5	45.4	31	50	5	55.6	
TMD	Absent	5	45.4	23	37.1	4	44.4	0.819
	Present	6	54.6	39	62.9	5	55.6	
DD	Absent	7	63.6	37	59.7	5	55.6	0.935
	Present	4	36.4	25	40.3	4	44.4	
Degenerative	Absent	11	100	61	98.4	9	100	0.849
Joint Disease	Present	0	0	1	1.6	0	0	
Myalgia	Absent	8	72.7	34	54.8	7	77.8	0.271
	Present	3	27.3	28	45.2	2	22.2	
Arthralgia	Absent	9	81.8	52	83.9	6	66.7	0.459
	Present	2	18.2	10	16.1	3	33.3	
Bruxism	Absent	6	54.5	33	53.2	3	33.3	0.522
	Present	5	45.5	29	46.8	6	66.6	

Table 15 Comparison of PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Values Between Different Marital Statuses.
Kruskal-Wallis H Test, $p < 0.05$

	Marital Status						p
	Never married		Married		Divorced		
	Mean ± SD	Median (Min-Max)	Mean ± SD	Median (Min-Max)	Mean ± SD	Median (Min-Max)	
PSQI	4.36 ± 5.85	1 (0-8)	2.95 ± 2.99	2 (1-5)	2.33 ± 2.06	2 (1-2)	0.966
OHIP total	11.09 ± 7.08	11 (4-18)	11.53 ± 9.91	10 (4-17)	9.67 ± 6.93	10 (4-13)	0.923
SOM	0.75 ± 0.79	0.58 (0.17-1.17)	0.66 ± 0.52	0.5 (0.33-1)	0.71 ± 0.4	0.58 (0.42-1.17)	0.779
ANX	0.63 ± 0.88	0.3 (0.2-0.6)	0.61 ± 0.44	0.6 (0.2-0.9)	0.57 ± 0.35	0.6 (0.3-0.7)	0.562
O-C	1.22 ± 0.78	0.8 (0.7-1.5)	1.1 ± 0.68	0.9 (0.6-1.5)	1.07 ± 0.37	1.1 (0.7-1.4)	0.936
DEP	0.65 ± 0.74	0.31 (0.15-0.85)	0.91 ± 0.67	0.73 (0.38-1.31)	0.79 ± 0.36	0.69 (0.69-1)	0.277
I-S	0.67 ± 0.75	0.56 (0.11-0.89)	0.81 ± 0.64	0.72 (0.33-1.11)	0.67 ± 0.28	0.67 (0.44-0.89)	0.551
PSY	0.33 ± 0.35	0.2 (0.1-0.5)	0.52 ± 0.44	0.45 (0.2-0.8)	0.47 ± 0.39	0.4 (0.2-0.5)	0.344
PAR	0.73 ± 0.84	0.5 (0.17-1)	0.93 ± 0.6	0.83 (0.5-1.33)	0.8 ± 0.35	0.83 (0.67-0.83)	0.32
HOS	0.83 ± 0.84	0.67 (0.17-1.17)	0.67 ± 0.56	0.67 (0.17-1)	0.76 ± 0.44	0.67 (0.33-1)	0.676
PHOB	0.67 ± 1.14	0.29 (0.14-0.57)	0.42 ± 0.49	0.29 (0-0.57)	0.35 ± 0.34	0.29 (0-0.57)	0.824
ADD.S	0.62 ± 0.43	0.57 (0.29-1)	0.87 ± 0.55	0.86 (0.57-1.29)	0.89 ± 0.44	0.86 (0.57-1)	0.296
GSI	0.71 ± 0.68	0.52 (0.29-0.74)	0.76 ± 0.46	0.66 (0.37-1.02)	0.72 ± 0.24	0.74 (0.6-0.82)	0.507
JFLS-8	12.73 ± 11.37	13 (1-23)	13.39 ± 10.39	12 (4-21)	14.78 ± 14.3	9 (2-29)	0.932
JFLS-20	23 ± 18.22	21 (4-41)	26.53 ± 19.04	25 (9-41)	28.22 ± 24.43	19 (7-54)	0.818
PHQ-4	1.45 ± 2.5	0 (0-2)	1.95 ± 2.66	1 (0-3)	1.67 ± 2.24	0 (0-3)	0.703
PHQ-9	5.18 ± 6.29	4 (1-7)	5.73 ± 5.2	4.5 (2-8)	4.44 ± 4.72	3 (1-7)	0.672
GAD-7	4.18 ± 5.86	1 (1-5)	4.45 ± 4.1	3 (1-7)	4 ± 3.77	3 (1-5)	0.729

An analysis of the SCL-90-R data revealed statistically significant scores on all the subscales, except the PHOB subscale, for the case groups as compared with the controls. The SOM ($p = 0.019$), ANX, O-C, DEP, I-S ($p < 0.001$), PSY ($p = 0.001$), PAR ($p < 0.001$), HOS ($p = 0.004$), ADD.S (additional SCL-90-R subscale) ($p = 0.001$), and GSI ($p < 0.001$) subscale scores were found to be significantly higher in the case group. Although the difference was not statistically significant for the PHOB subscale, the case group had higher scores than the controls.

Sleep quality was measured using the PSQI. The PSQI scores were significantly higher in the case group ($p < 0.001$).

The OHIP-14 scores were higher in the case group; however, the difference was not statistically significant ($p = 0.166$).

There was no difference in the JFLS-20 scores between the case and control groups ($p = 0.054$). However, the differences in the JFLS-8 ($p = 0.005$), PHQ-4 ($p = 0.015$), PHQ-9 ($p = 0.009$), and GAD-7 ($p = 0.007$) scores were statistically significant (Table 16).

Table 16 Comparison of the PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II values Between the Case and Control groups.
Mann Whitney U Test, $p < 0.05$

	FM				p
	Absent		Present		
	Mean ± SD	Median (Min-Max)	Mean ± SD	Median (Min-Max)	
PSQI	1.24 ± 1.53	1 (0-2)	4.9 ± 3.81	4 (2-7)	< 0.001
OHIP total	10.02 ± 8.9	7 (3-16)	12.51 ± 9.49	11 (4-18)	0.166
SOM	0.52 ± 0.41	0.42 (0.25-0.67)	0.84 ± 0.62	0.67 (0.42-1.17)	0.019
ANX	0.42 ± 0.35	0.3 (0.2-0.6)	0.8 ± 0.56	0.7 (0.4-1)	< 0.001
O-C	0.8 ± 0.49	0.7 (0.5-1)	1.42 ± 0.67	1.4 (0.9-1.8)	< 0.001
DEP	0.55 ± 0.37	0.54 (0.31-0.69)	1.17 ± 0.72	1.15 (0.69-1.69)	< 0.001
I-S	0.5 ± 0.35	0.56 (0.22-0.78)	1.05 ± 0.71	1 (0.56-1.33)	< 0.001
PSY	0.34 ± 0.34	0.2 (0.1-0.5)	0.64 ± 0.46	0.6 (0.3-0.9)	0.001
PAR	0.64 ± 0.46	0.67 (0.33-0.83)	1.13 ± 0.66	1 (0.83-1.5)	< 0.001
HOS	0.5 ± 0.38	0.5 (0.17-0.67)	0.9 ± 0.69	0.83 (0.33-1.17)	0.004
PHOB	0.31 ± 0.35	0.14 (0-0.43)	0.59 ± 0.75	0.42 (0.14-0.86)	0.07
ADD.S	0.66 ± 0.46	0.57 (0.29-0.86)	1.02 ± 0.53	1 (0.57-1.29)	0.001
GSI	0.52 ± 0.31	0.5 (0.3-0.64)	0.97 ± 0.5	0.93 (0.6-1.23)	< 0.001
JFLS-8	10.12 ± 9.88	6 (2-19)	16.78 ± 10.86	17 (7-27)	0.005
JFLS-20	22.12 ± 18.55	15 (6-35)	30.37 ± 19.5	32 (15-45)	0.054
PHQ-4	1.12 ± 1.68	0 (0-2)	2.59 ± 3.08	2 (0-4)	0.015
PHQ-9	3.93 ± 4.05	3 (1-6)	7.1 ± 5.87	5 (3-12)	0.009
GAD-7	3.02 ± 3.22	2 (1-4)	5.71 ± 4.8	5 (2-9)	0.007

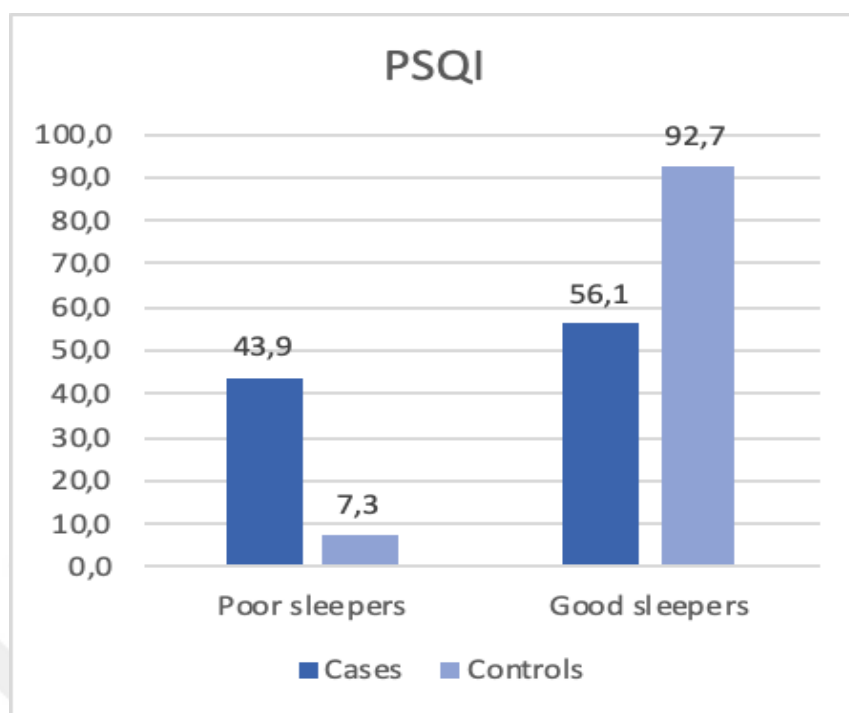


Figure 7 Prevalence of Good and Poor Sleepers in the Case and Control Groups.

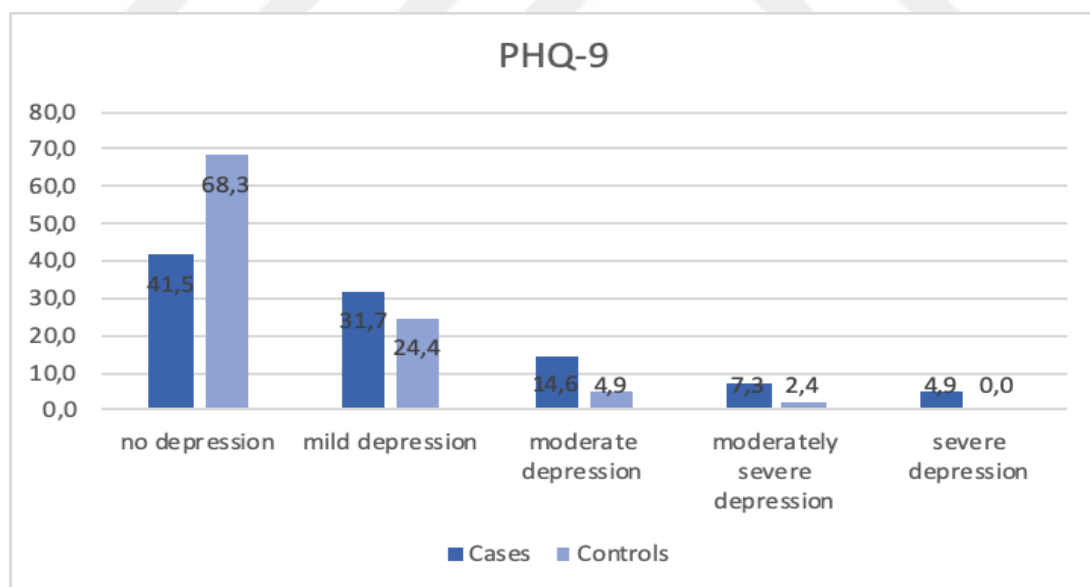


Figure 8 Comparison of Different DEP Levels Between the Case and Control Groups.

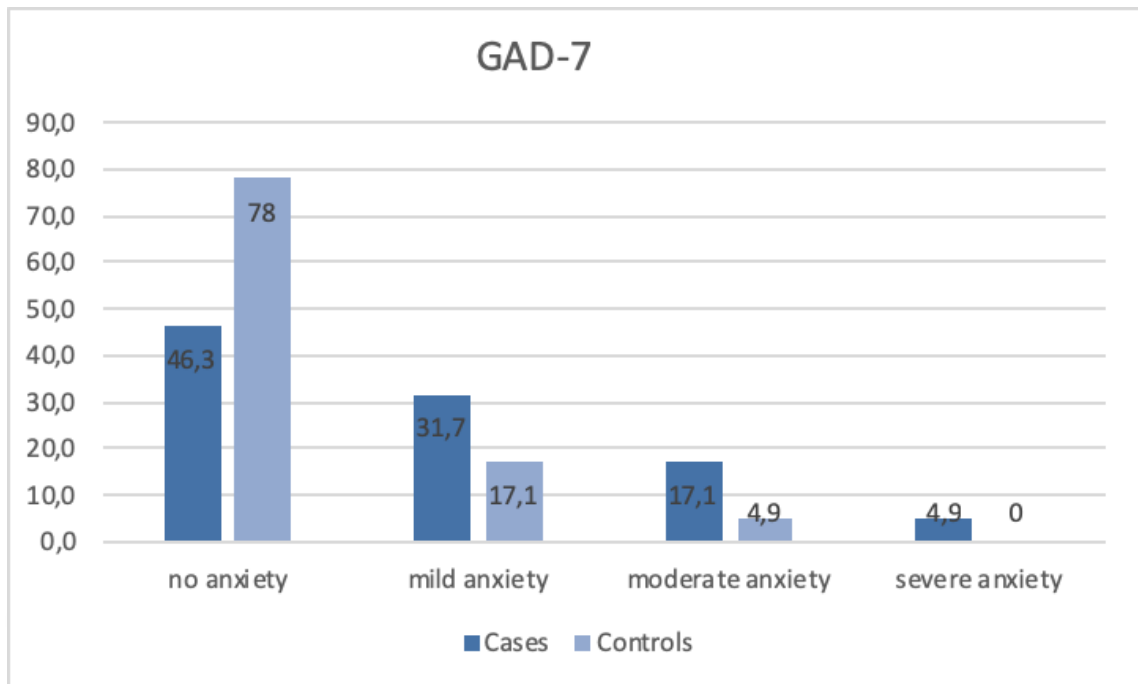


Figure 9 Comparison of Different ANX Levels Between the Case and Control Groups.

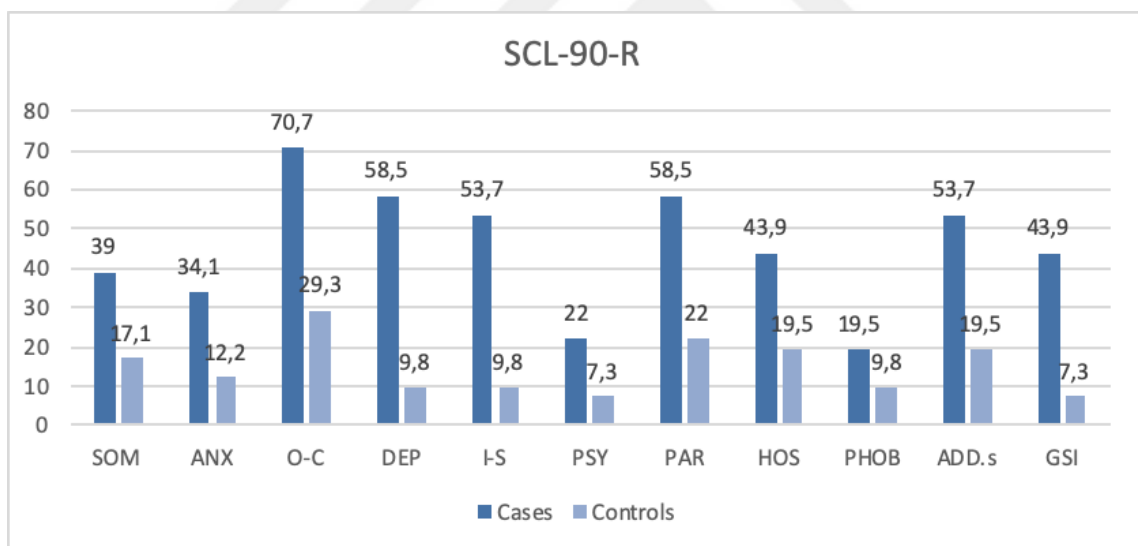


Figure 10 The Incidence of Psychiatric Symptoms in the Case and Control Groups.

DEP, one of the subscales of SCL-90-R, was significantly higher in the TMD patients than the patients without TMD ($p = 0.033$). The TMD patients had higher PSQI ($p = 0.043$) and OHIP-14 ($p < 0.001$) scores than the individuals without TMD. The differences were statistically significant. The JFLS-8 and JFLS-20 scores were also significantly higher in the TMD patients ($p < 0.001$) (Table 17).

Table 17 Comparison of the PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II values Between Patients with and without TMD.

Mann Whitney U Test, $p < 0.05$

	TMD				p
	Absent		Present		
	Mean ± SD	Median (Min-Max)	Mean ± SD	Median (Min-Max)	
PSQI	2.06 ± 2.29	1 (1-3)	3.72 ± 3.87	2 (1-5)	0.043
OHIP	5.81 ± 6.14	4 (2-7)	14.76 ± 9.23	14.5 (7-21)	0.001
SOM	0.59 ± 0.42	0.5 (0.29-0.87)	0.73 ± 0.61	0.58 (0.25-1.17)	0.577
ANX	0.55 ± 0.45	0.4 (0.2-0.75)	0.65 ± 0.54	0.6 (0.2-0.9)	0.425
O-C	0.91 ± 0.46	0.8 (0.6-1.35)	1.24 ± 0.73	1.1 (0.6-1.8)	0.07
DEP	0.68 ± 0.57	0.62 (0.31-0.81)	0.97 ± 0.68	0.85 (0.38-1.38)	0.033
I-S	0.64 ± 0.47	0.56 (0.28-0.89)	0.86 ± 0.7	0.72 (0.33-1.22)	0.195
PSY	0.45 ± 0.48	0.3 (0.1-0.65)	0.52 ± 0.39	0.5 (0.2-0.8)	0.211
PAR	0.73 ± 0.55	0.67 (0.17-0.92)	0.99 ± 0.64	0.92 (0.5-1.5)	0.07
HOS	0.7 ± 0.63	0.67 (0.33-0.83)	0.71 ± 0.57	0.67 (0.17-1)	0.738
PHOB	0.44 ± 0.5	0.29 (0.14-0.57)	0.45 ± 0.66	0.29 (0-0.57)	0.657
ADD.S	0.76 ± 0.48	0.57 (0.43-1)	0.89 ± 0.56	0.86 (0.57-1.29)	0.239
GSI	0.64 ± 0.4	0.57 (0.36-0.78)	0.81 ± 0.5	0.73 (0.47-1.08)	0.113
JFLS-8	2.56 ± 2.69	2 (0-4)	20.42 ± 7.94	21 (15-28)	0.001
JFLS-20	7.41 ± 6.64	5.5 (2-12)	38.3 ± 14.55	39.5 (28-51)	< 0.001
PHQ-4	1.91 ± 3.04	1 (0-2.5)	1.82 ± 2.26	1 (0-3)	0.7
PHQ-9	5.75 ± 5.72	3.5 (2-8)	5.36 ± 5	4 (1-8)	0.985
GAD-7	4.34 ± 4.44	3 (1-6)	4.38 ± 4.22	3.5 (1-7)	0.962

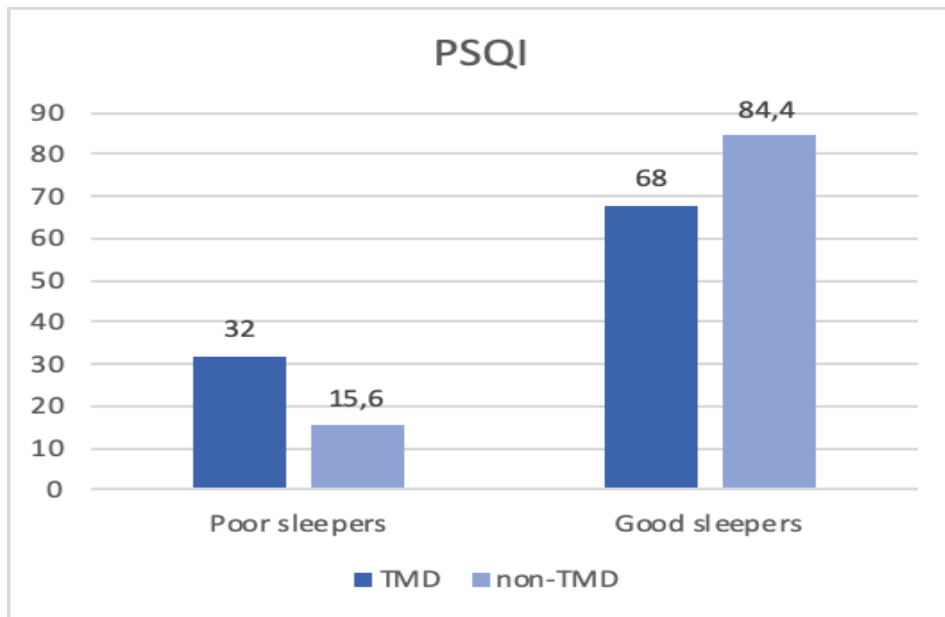


Figure 11 Prevalence of Good and Poor Sleepers Among Patients With and Without TMD.

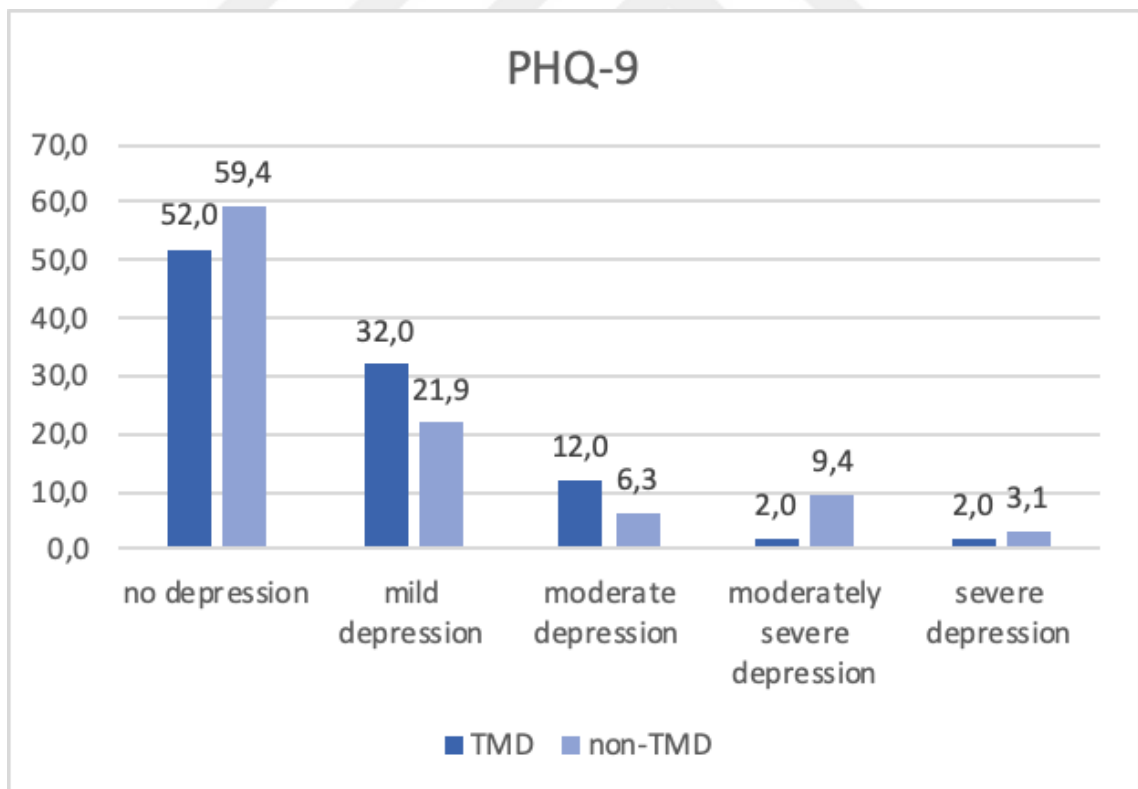


Figure 12 Comparison of Different DEP Levels Between Patients With and Without TMD.

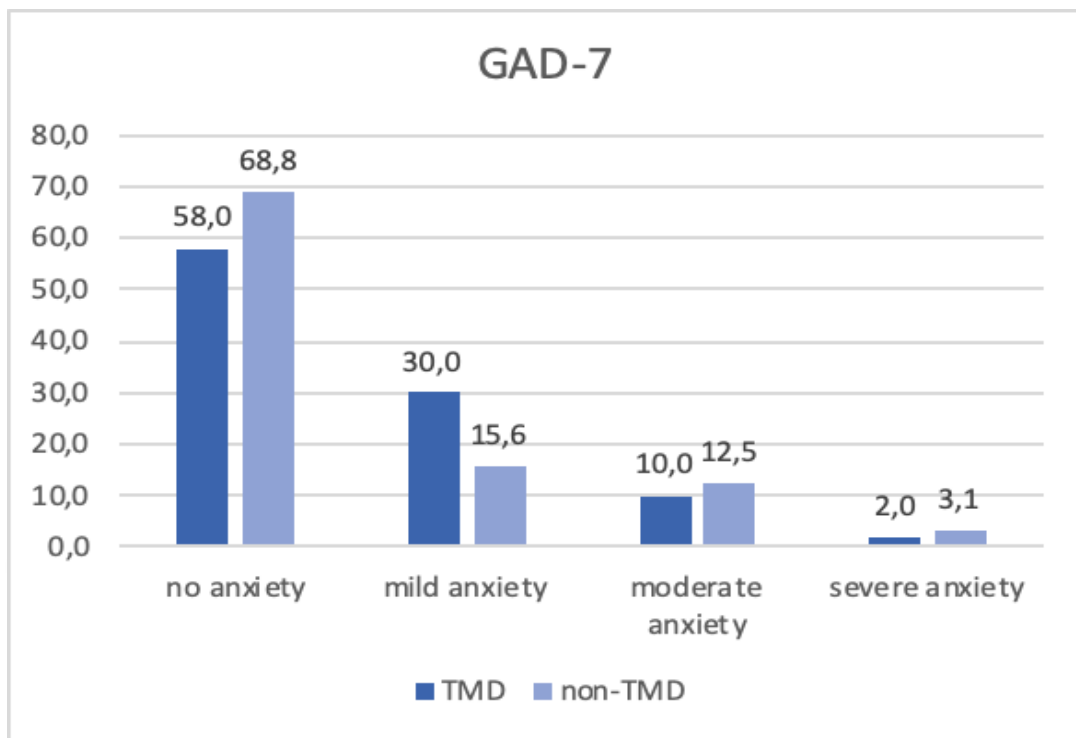


Figure 13 Comparison of Different ANX Levels Between Patients With and Without TMD.

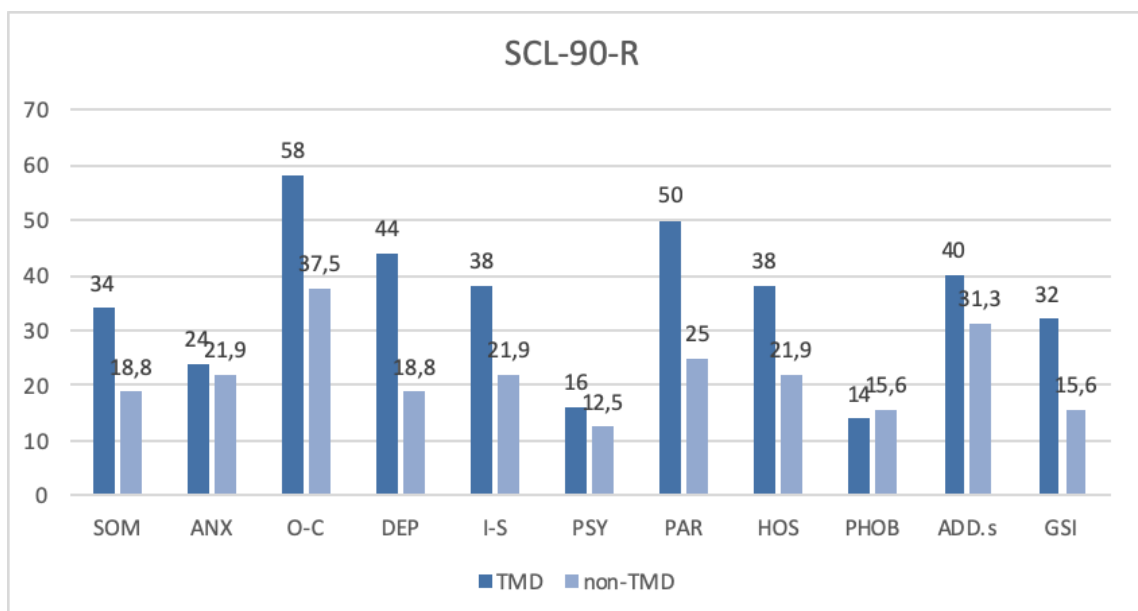


Figure 14 The Incidence of Psychiatric Symptoms in Patients With and Without TMD.

No significant difference was found in the subscales of SCL-90-R. The analyses showed statistically significant differences between the patients with DD compared to those without DD in the PSQI ($p = 0.015$), OHIP-14 (0.036), JFLS-8, and JFLS-20 ($p < 0.001$) (Table 18).

Table 18 Comparison of the PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Values Between Patients with and without DD.

Mann Whitney U Test, $p < 0.05$

	DD				p
	Absent		Present		
	Mean ± SD	Median (Min-Max)	Mean ± SD	Median (Min-Max)	
PSQI	2.22 ± 2.39	1 (1-3)	4.33 ± 4.28	3 (1-7)	0.015
OHIP	9.73 ± 8.83	6 (3-14)	13.55 ± 9.47	13 (5-18)	0.036
SOM	0.63 ± 0.48	0.5 (0.25-0.92)	0.75 ± 0.62	0.58 (0.33-1.16)	0.596
ANX	0.55 ± 0.44	0.4 (0.2-0.9)	0.7 ± 0.58	0.6 (0.3-1)	0.19
O-C	1.01 ± 0.54	0.8 (0.6-1.4)	1.26 ± 0.79	1.1 (0.6-1.8)	0.223
DEP	0.77 ± 0.58	0.69 (0.31-1)	0.99 ± 0.74	0.85 (0.31-1.38)	0.188
I-S	0.7 ± 0.55	0.56 (0.33-1)	0.89 ± 0.72	0.78 (0.33-1.33)	0.269
PSY	0.46 ± 0.46	0.3 (0.1-0.8)	0.53 ± 0.37	0.5 (0.2-0.8)	0.15
PAR	0.81 ± 0.57	0.83 (0.33-1.17)	1 ± 0.67	0.83 (0.67-1.33)	0.198
HOS	0.66 ± 0.59	0.67 (0.17-0.83)	0.78 ± 0.6	0.67 (0.33-1)	0.255
PHOB	0.42 ± 0.5	0.29 (0-0.57)	0.49 ± 0.73	0.29 (0.14-0.57)	0.578
ADD.S	0.76 ± 0.51	0.57 (0.43-1)	0.96 ± 0.54	0.86 (0.57-1.29)	0.056
GSI	0.68 ± 0.42	0.6 (0.37-0.94)	0.84 ± 0.53	0.74 (0.47-1.02)	0.15
JFLS-8	5.76 ± 5.48	4 (1-9)	24.88 ± 5.24	26 (21-29)	< 0.001
JFLS-20	13.43 ± 11.7	12 (4-19)	45.27 ± 10.9	46 (39-54)	< 0.001
PHQ-4	1.69 ± 2.6	1 (0-3)	2.09 ± 2.55	1 (0-3)	0.435
PHQ-9	5.27 ± 5.27	4 (1-8)	5.88 ± 5.3	5 (2-7)	0.481
GAD-7	4.16 ± 4.18	3 (1-6)	4.67 ± 4.48	4 (1-7)	0.591



Figure 15 Prevalence of Good and Poor Sleepers Among Patients With and Without DD.

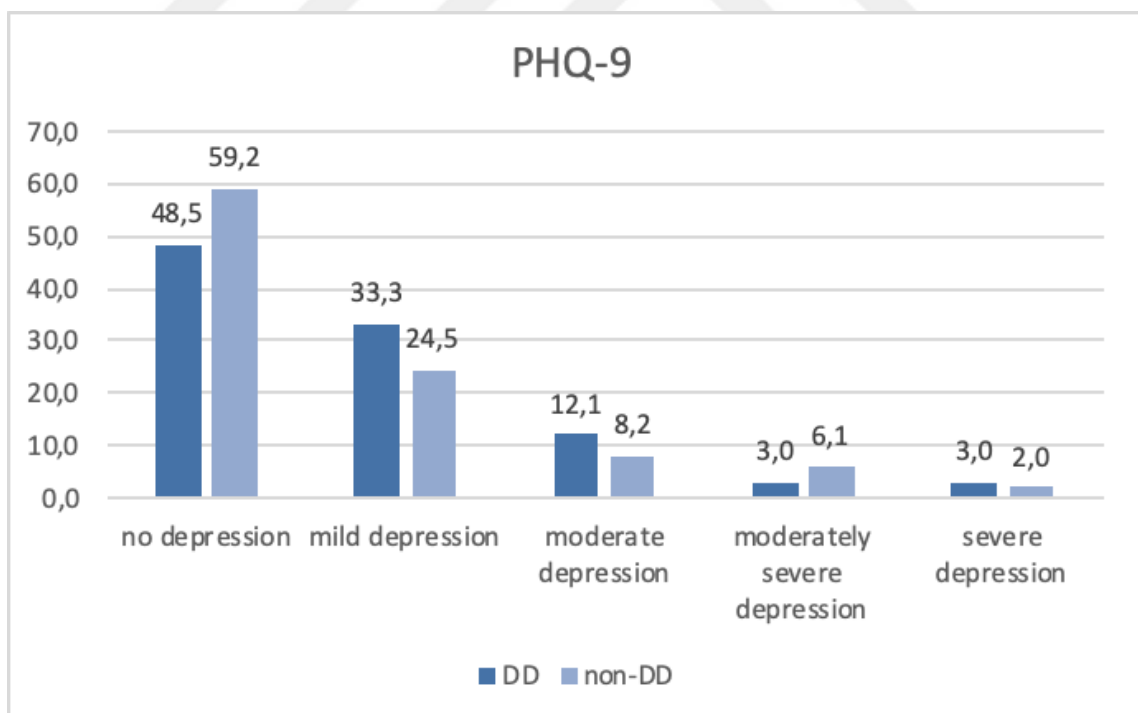


Figure 16 Comparison of Different DEP Levels Between Patients With and Without DD.

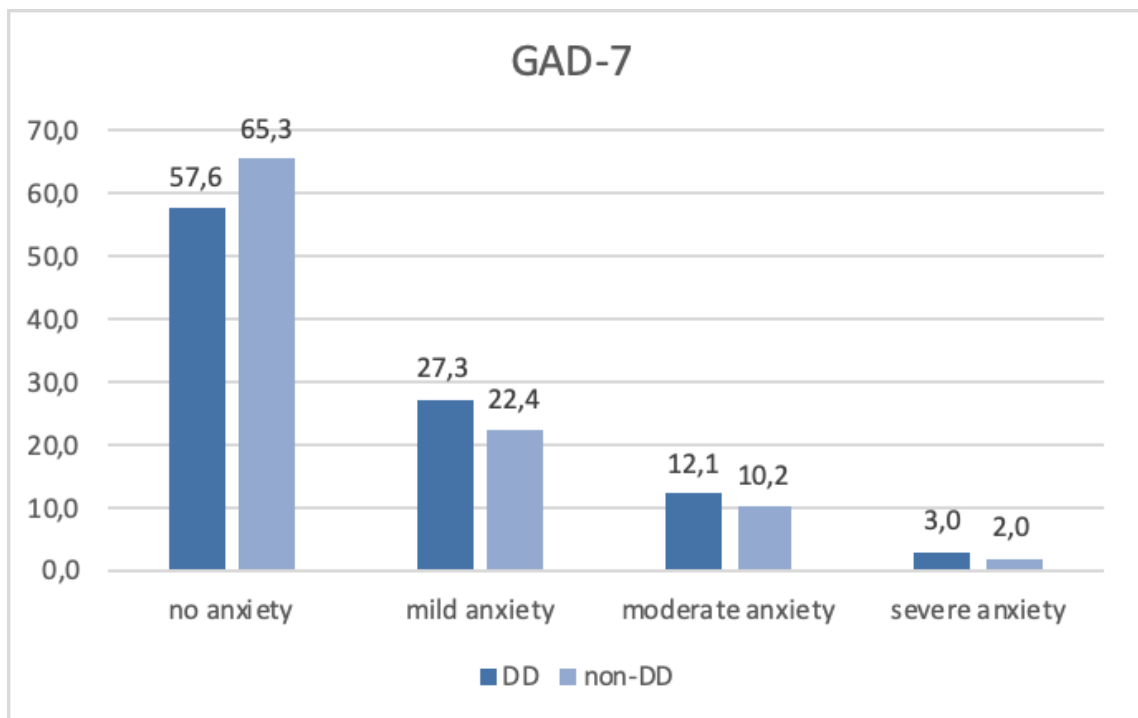


Figure 17 Comparison of Different ANX Levels Between Patients With and Without DD.

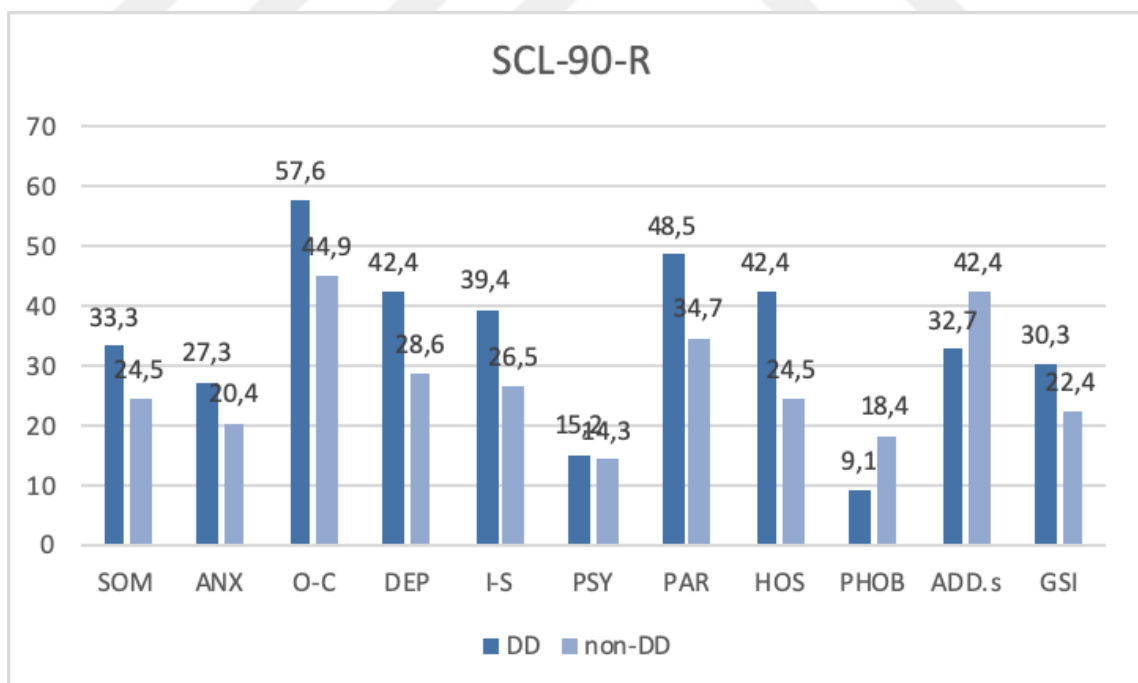


Figure 18 The Incidence of Psychiatric Symptoms in Patients with and without DD.

The DEP ($p = 0.019$), OHIP ($p < 0.001$), JFLS-8, and JFLS-20 ($p = 0.001$) values were significantly higher in patients with myalgia compared to individuals without myalgia (Table 19).

Table 19 Comparison of the PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Values Between Patients with and without Myalgia.

Mann Whitney U Test, $p < 0.05$

	Myalgia				p
	Absent		Present		
	Mean ± SD	Median (Min-Max)	Mean ± SD	Median (Min-Max)	
PSQI	2.98 ± 3.72	2 (1-3)	3.21 ± 2.97	2 (1-5)	0.433
OHIP	7.71 ± 7.71	4 (3-13)	16.55 ± 8.85	16 (11-21)	< 0.001
SOM	0.67 ± 0.56	0.5 (0.33-1)	0.69 ± 0.53	0.58 (0.25-1.16)	0.794
ANX	0.6 ± 0.55	0.4 (0.2-0.7)	0.63 ± 0.43	0.7 (0.2-0.9)	0.525
O-C	1.03 ± 0.66	0.8 (0.6-1.4)	1.24 ± 0.64	1.3 (0.8-1.8)	0.08
DEP	0.75 ± 0.67	0.69 (0.31-0.92)	1.02 ± 0.6	1 (0.54-1.46)	0.019
I-S	0.73 ± 0.6	0.67 (0.33-0.89)	0.84 ± 0.66	0.78 (0.33-1.22)	0.317
PSY	0.48 ± 0.46	0.4 (0.15-0.8)	0.5 ± 0.37	0.5 (0.2-0.8)	0.556
PAR	0.84 ± 0.63	0.83 (0.33-1)	0.96 ± 0.58	1 (0.5-1.5)	0.212
HOS	0.78 ± 0.66	0.67 (0.33-1)	0.59 ± 0.46	0.67 (0.17-1)	0.24
PHOB	0.51 ± 0.7	0.29 (0.14-0.57)	0.35 ± 0.42	0.14 (0-0.57)	0.198
ADD.S	0.85 ± 0.51	0.71 (0.57-1.14)	0.82 ± 0.56	0.71 (0.43-1.29)	0.812
GSI	0.72 ± 0.51	0.6 (0.37-0.82)	0.78 ± 0.42	0.74 (0.43-1.1)	0.281
JFLS-8	10.69 ± 11.83	4 (1-21)	17.55 ± 7.68	17 (12-23)	0.001
JFLS-20	21.24 ± 20.58	12 (4-41)	33.67 ± 14.77	34 (21-43)	0.001
PHQ-4	1.86 ± 2.81	1 (0-3)	1.85 ± 2.22	1 (0-3)	0.557
PHQ-9	5.61 ± 5.59	4 (2-8)	5.36 ± 4.8	4 (1-8)	0.958
GAD-7	4.31 ± 4.56	3 (1-6)	4.45 ± 3.91	4 (1-8)	0.721

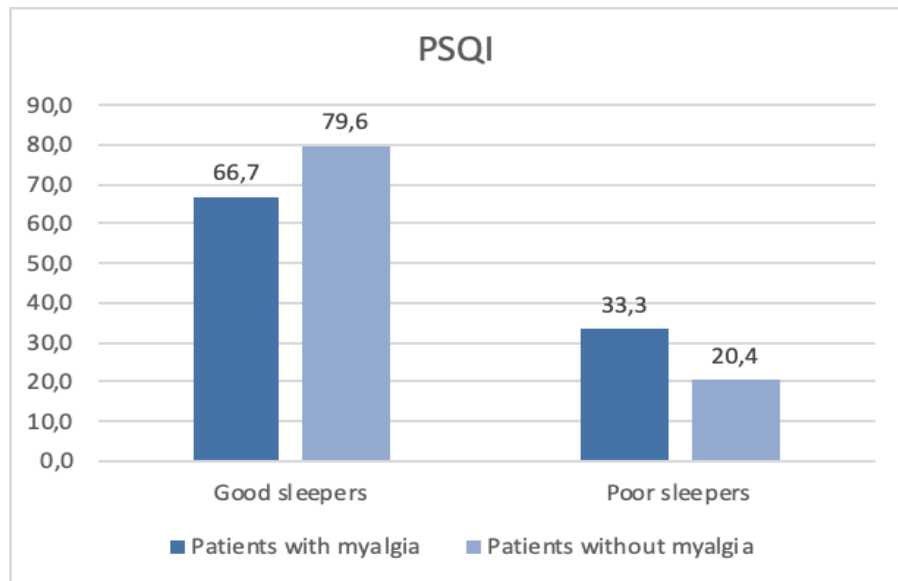


Figure 19 Prevalence of Good and Poor Sleepers Among Patients With and Without Myalgia.

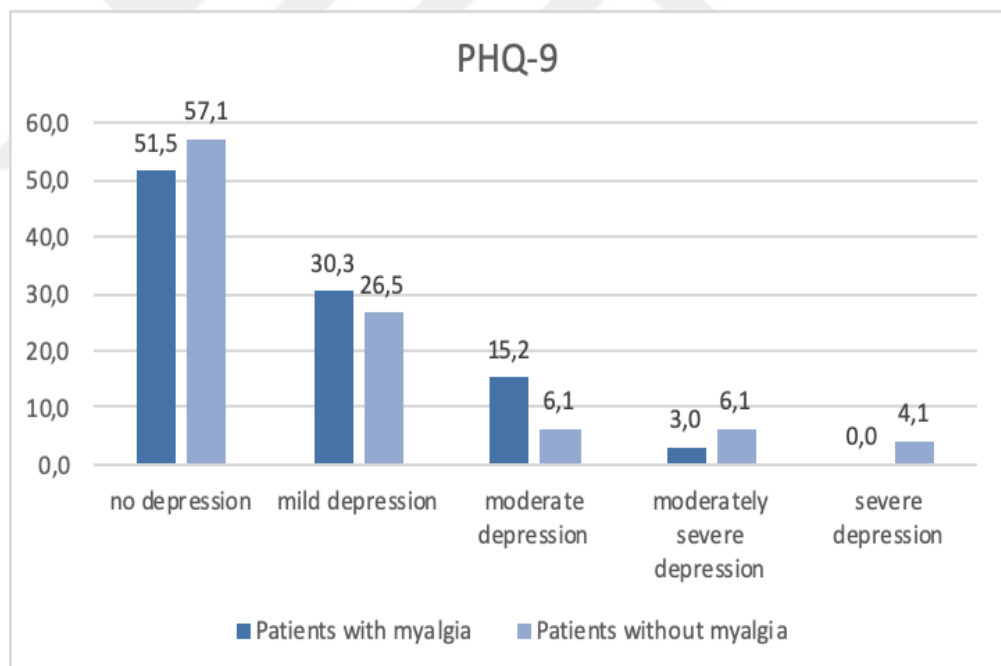


Figure 20 Comparison of Different DEP Levels Between Patients With and Without Myalgia.

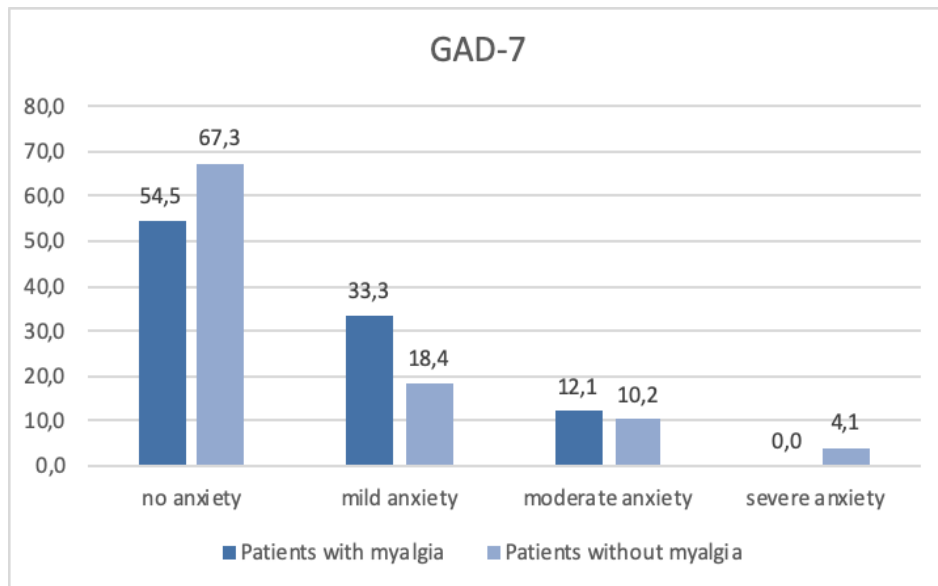


Figure 21 Comparison of Different ANX Levels Between Patients With and Without Myalgia.

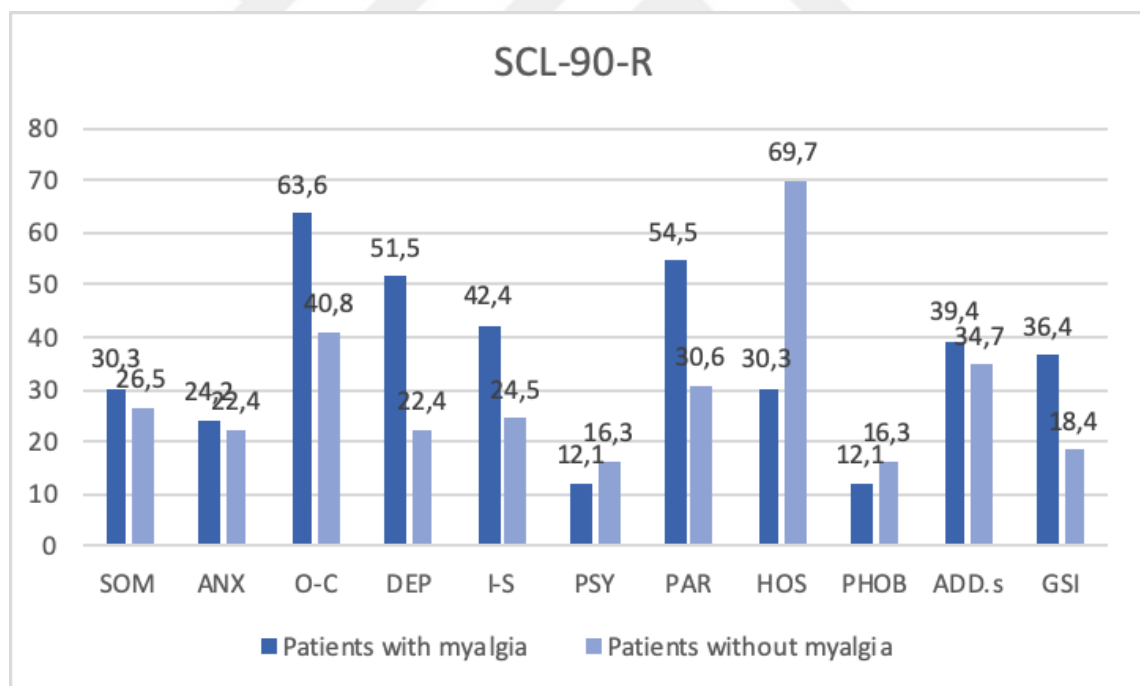


Figure 22 The Incidence of Psychiatric Symptoms in Patients With and Without Myalgia.

The JFLS-8 and JFLS-20 scores were significantly higher in patients with arthralgia ($p < 0.001$) (Table 20).

Table 20 Comparison of the PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Values Between Patients with and without Arthralgia.

Mann Whitney U Test, $p < 0.05$

	Arthralgia				p
	Absent		Present		
	Mean ± SD	Median (Min-Max)	Mean ± SD	Median (Min-Max)	
PSQI	2.82 ± 3	2 (0-16)	4.2 ± 4.84	2 (0-14)	0.49
OHIP	10.51 ± 8.84	7 (0-33)	14.67 ± 10.46	13 (0-37)	0.138
SOM	0.67 ± 0.54	0.5 (0-2.69)	0.74 ± 0.59	0.58 (0.08-2)	0.801
ANX	0.61 ± 0.51	0.5 (0-3)	0.6 ± 0.48	0.6 (0-1.7)	0.971
O-C	1.11 ± 0.63	0.9 (0.1-3.2)	1.11 ± 0.79	1 (0.1-2.9)	0.796
DEP	0.84 ± 0.64	0.69 (0-2.61)	0.95 ± 0.71	0.85 (0.15-2.46)	0.548
I-S	0.78 ± 0.6	0.67 (0-2.78)	0.75 ± 0.74	0.56 (0-2.44)	0.568
PSY	0.51 ± 0.44	0.4 (0-2.1)	0.39 ± 0.34	0.3 (0-1.1)	0.459
PAR	0.89 ± 0.6	0.83 (0-2.67)	0.86 ± 0.68	0.67 (0-2.5)	0.673
HOS	0.73 ± 0.62	0.67 (0-3.17)	0.61 ± 0.43	0.67 (0-1.33)	0.74
PHOB	0.47 ± 0.64	0.29 (0-4)	0.37 ± 0.38	0.29 (0-1.43)	0.951
ADD.S	0.85 ± 0.51	0.86 (0-2.43)	0.82 ± 0.64	0.57 (0.14-2.43)	0.591
GSI	0.75 ± 0.47	0.64 (0.13-2.54)	0.74 ± 0.51	0.74 (0.08-2)	0.919
JFLS-8	10.4 ± 9.42	7 (0-34)	27.07 ± 4.45	28 (17-33)	< 0.001
JFLS-20	20.9 ± 16.86	15 (0-61)	50.13 ± 8.98	52 (32-65)	< 0.001
PHQ-4	1.84 ± 2.61	1 (0-14)	1.93 ± 2.49	1 (0-8)	0.885
PHQ-9	5.55 ± 5.25	4 (0-21)	5.33 ± 5.5	4 (0-17)	0.767
GAD-7	4.42 ± 4.33	3 (0-19)	4.13 ± 4.19	4 (0-12)	0.749

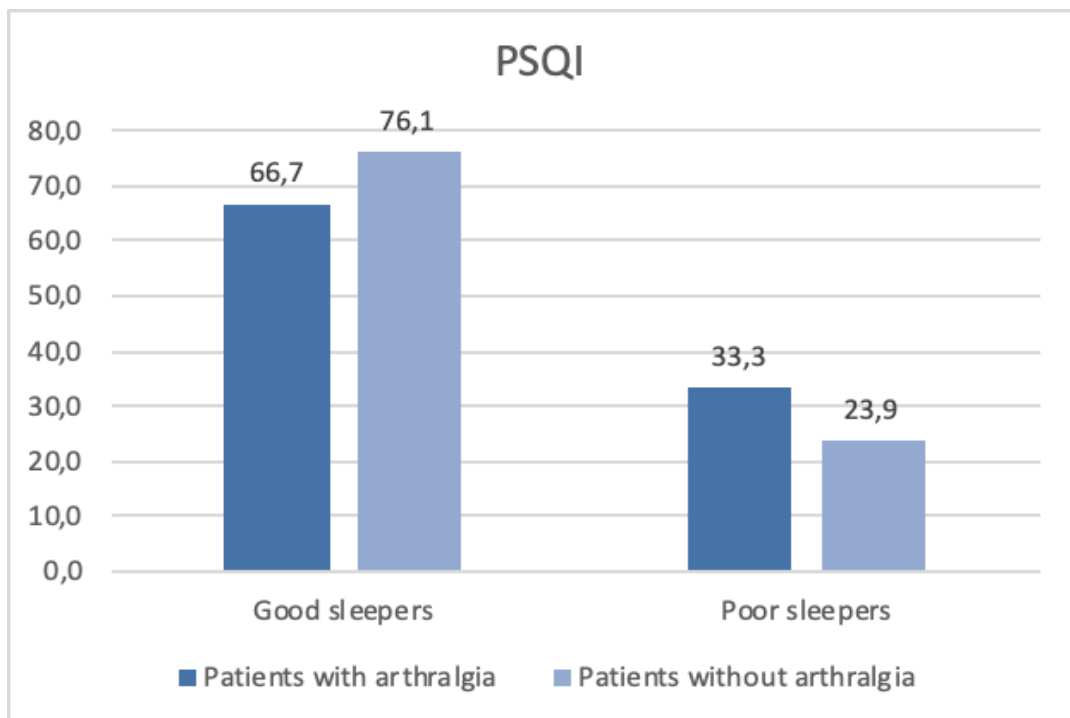


Figure 23 Prevalence of Good and Poor sleepers Among Patients With and Without Arthralgia.

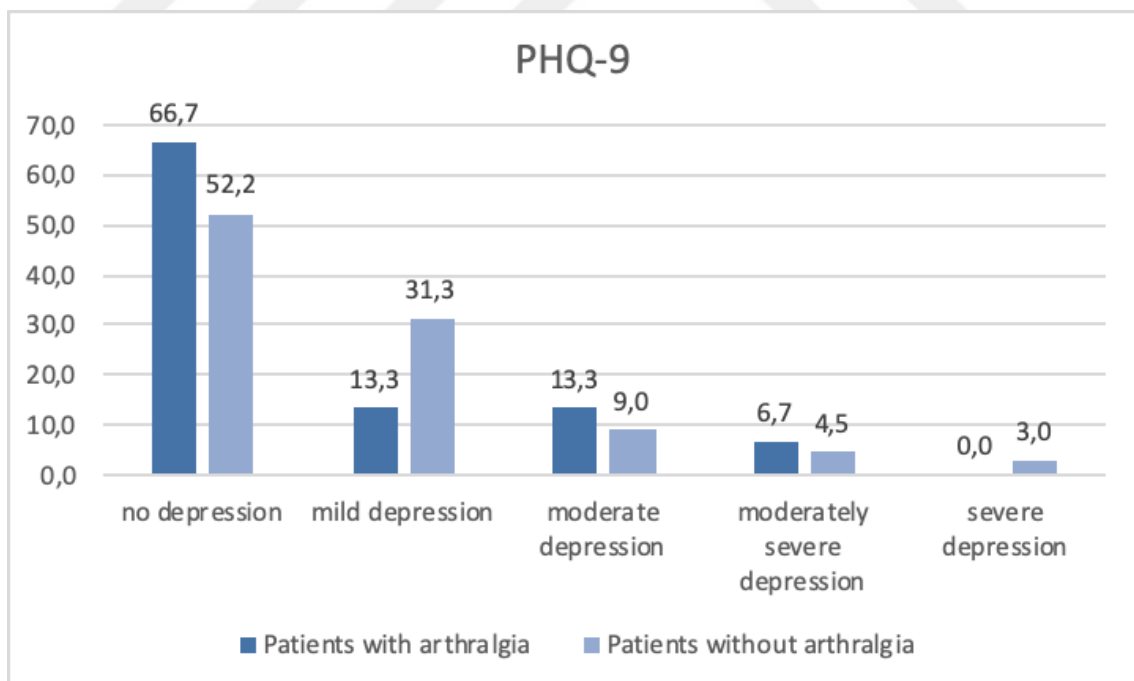


Figure 24 Comparison of Different DEP Levels Between Patients With and Without Arthralgia.

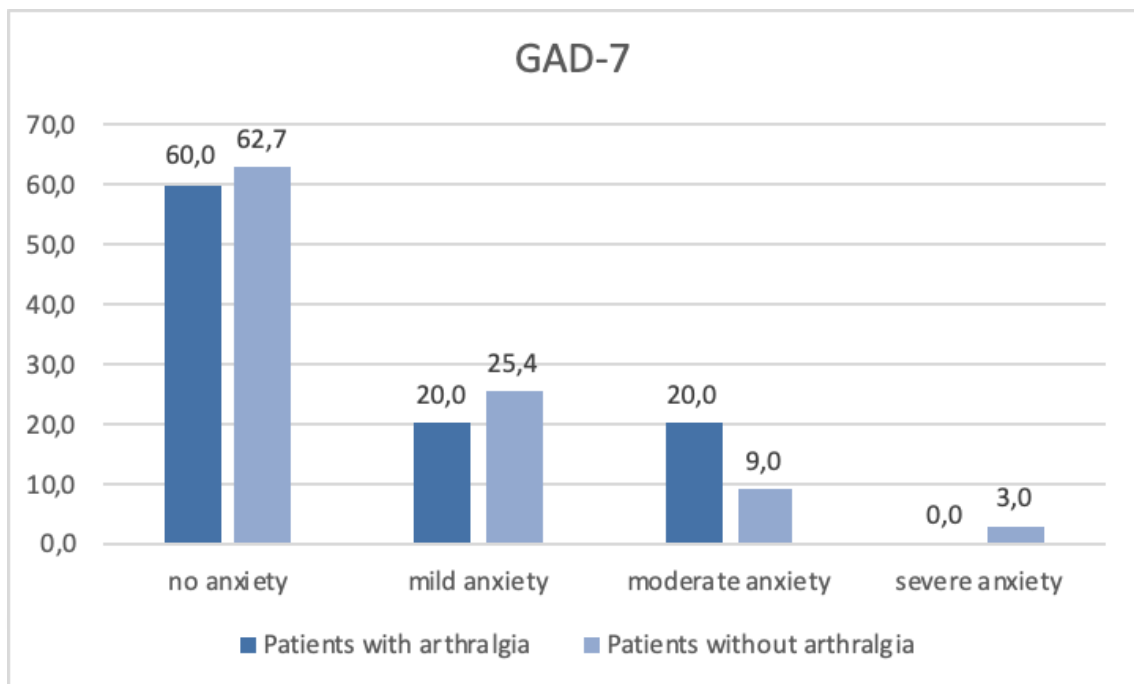


Figure 25 Comparison of Different ANX Levels Between Patients With and Without Arthralgia.

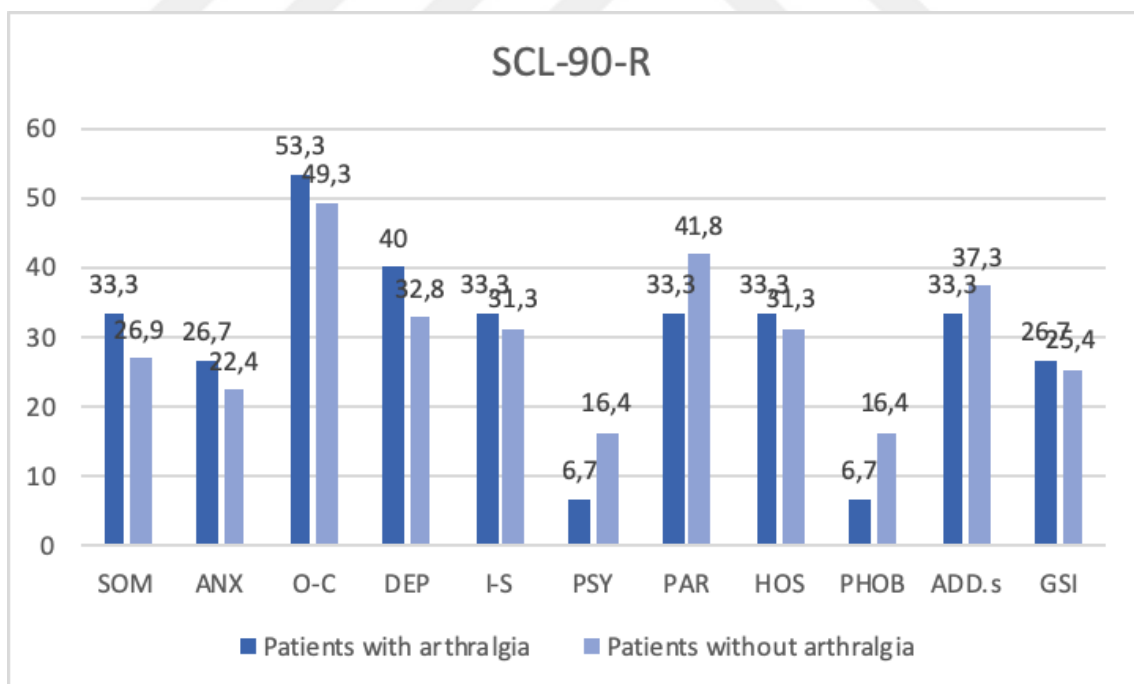


Figure 26 The Incidence of Psychiatric Symptoms in Patients with and without Arthralgia.

The O-C ($p = 0.038$), DEP ($p = 0.007$), PSQI ($p = 0.007$), OHIP-14 ($p = 0.004$), JFLS-8 ($p = 0.011$), and JFLS-20 ($p=0.016$) scores were significantly higher in patients with bruxism than those without bruxism (Table 21).

Table 21 Comparison of the PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Values Between Patients with Bruxism and the Control Groups.

Mann Whitney U Test, $p < 0.05$

	Bruxism				p
	Absent		Present		
	Mean ± SD	Median (Min-Max)	Mean ± SD	Median (Min-Max)	
PSQI	2.14 ± 2.4	1 (1-3)	4.05 ± 4.04	2.5 (1-5.5)	0.008
OHIP	8.36 ± 7.67	4 (3-14)	14.32 ± 9.82	13 (6.5-20.5)	0.004
SOM	0.64 ± 0.46	0.46 (0.33-0.92)	0.72 ± 0.63	0.58 (0.25-1.16)	0.823
ANX	0.54 ± 0.43	0.4 (0.2-0.9)	0.69 ± 0.57	0.6 (0.3-0.9)	0.254
O-C	0.98 ± 0.66	0.8 (0.5-1.5)	1.25 ± 0.64	1.15 (0.75-1.5)	0.038
DEP	0.7 ± 0.62	0.58 (0.31-0.77)	1.03 ± 0.65	0.92 (0.65-1.35)	0.007
I-S	0.7 ± 0.65	0.56 (0.22-0.89)	0.86 ± 0.6	0.78 (0.5-1.17)	0.122
PSY	0.47 ± 0.47	0.35 (0.1-0.8)	0.51 ± 0.38	0.45 (0.2-0.8)	0.292
PAR	0.76 ± 0.6	0.67 (0.17-1.17)	1.02 ± 0.61	0.83 (0.58-1.5)	0.059
HOS	0.69 ± 0.49	0.67 (0.33-1)	0.72 ± 0.69	0.67 (0.17-1)	0.783
PHOB	0.39 ± 0.47	0.29 (0-0.57)	0.51 ± 0.72	0.29 (0-0.71)	0.575
ADD.S	0.75 ± 0.46	0.64 (0.43-1)	0.93 ± 0.58	0.86 (0.57-1.29)	0.149
GSI	0.66 ± 0.44	0.53 (0.36-0.94)	0.83 ± 0.49	0.76 (0.47-1.05)	0.063
JFLS-8	10.62 ± 10.49	5.5 (2-21)	16.43 ± 10.54	17 (7.5-26.5)	0.011
JFLS-20	21.38 ± 18.59	14.5 (4-35)	31.35 ± 19.06	34 (15-47)	0.016
PHQ-4	1.79 ± 2.28	1 (0-3)	1.93 ± 2.88	1 (0-3)	0.992
PHQ-9	5.21 ± 5.03	4 (1-8)	5.83 ± 5.53	4 (2-8.5)	0.641
GAD-7	4.14 ± 3.95	3 (1-6)	4.6 ± 4.64	3 (1-7)	0.794

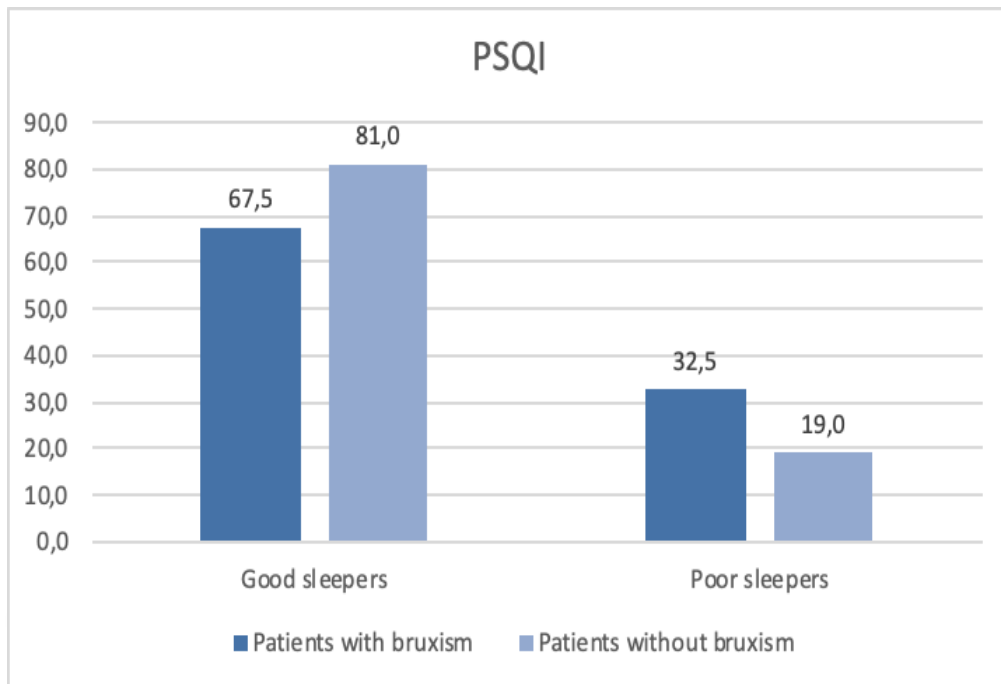


Figure 27 Prevalence of Good and Poor Sleepers Among Patients With and Without Bruxism.

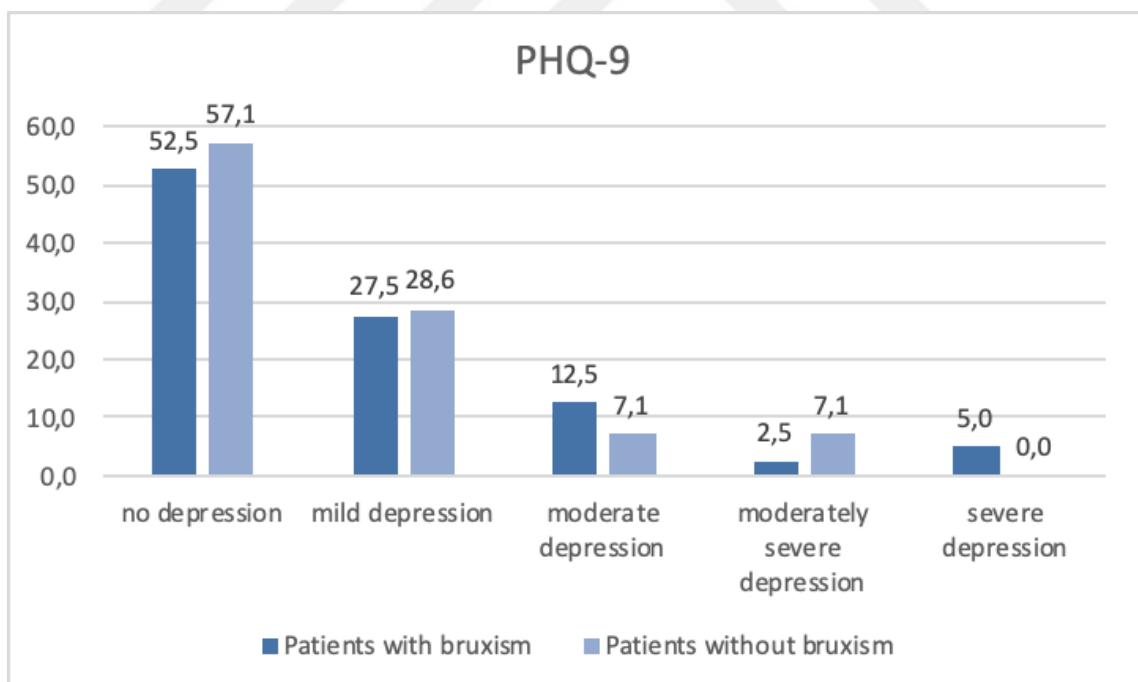


Figure 28 Comparison of Different DEP Levels Between Patients with and without Bruxism.

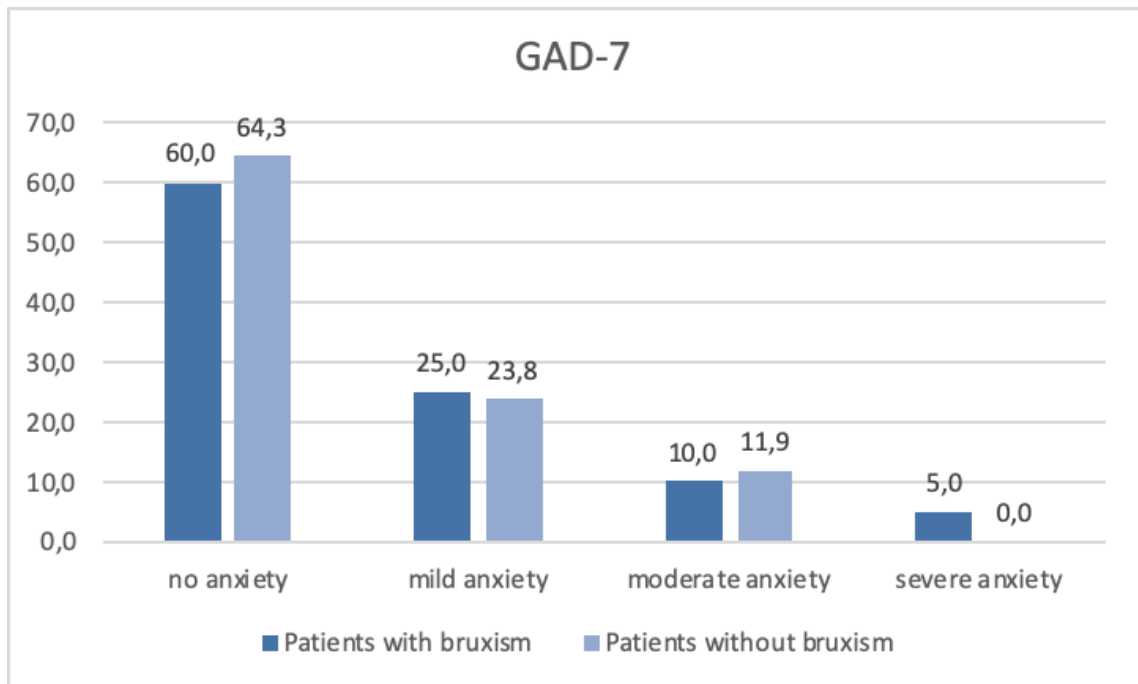


Figure 29 Comparison of Different ANX Levels Between Patients with and without Bruxism.

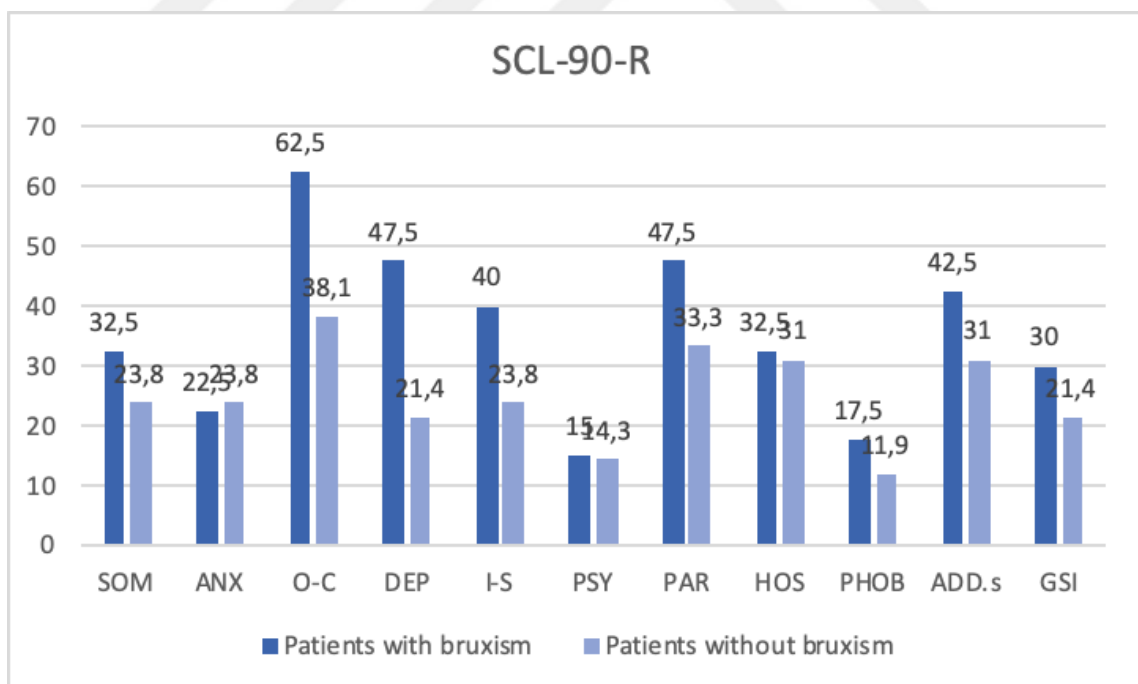


Figure 30 The Incidence of Psychiatric Symptoms in Patients with and without Bruxism.

According to Spearman's correlation for the total sample, the PHQ-9 and GAD-7 scores; PHQ-4, PHQ-9, and GAD-7 scores; JFLS-8 and JFLS-20 scores; all the SCL-90-R subscale scores; the SCL-90-R subscales and PHQ-4, PHQ-9, and GAD-7 scores; OHIP-14 and DEP, PSY, and GSI subscales of SCL-90-R, JFLS-8, and JFLS-20 scores; PSQI and SCL-90-R subscales; and JFLS-8, PHQ-4, PHQ-9, and GAD-7 scores were strongly correlated (Table 22).



Table 22 Correlation Between the PSQI, OHIP, and SCL-90-R Symptom Dimensions and DC/TMD Axis II Scores.

Spearman's Correlation

		OHIP total	SOM	ANX	O-C	DEP	I-S	PSY	PAR	HOS	PHOB	ADD.S	GSI	JFLS-8	JFLS- 20	PHQ-4	PHQ-9	GAD-7
PSQI	r	0.162	0.428	0.549	0.493	0.610	0.554	0.505	0.566	0.491	0.335	0.457	0.631	0.256	0.158	0.471	0.532	0.467
	p	0.145	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.002	0.000	0.000	0.020	0.155	0.000	0.000	0.000
OHIP total	r		0.214	0.096	0.193	0.288	0.161	0.236	0.105	0.022	0.072	0.101	0.234	0.329	0.326	0.041	0.009	-0.016
	p		0.054	0.392	0.083	0.009	0.148	0.033	0.350	0.846	0.520	0.366	0.035	0.003	0.003	0.714	0.934	0.890
SOM	r			0.696	0.673	0.662	0.615	0.531	0.473	0.363	0.470	0.490	0.780	0.095	0.051	0.555	0.575	0.582
	p			0.000	0.000	0.000	0.000	0.000	0.000	0.001	0.000	0.000	0.000	0.397	0.652	0.000	0.000	0.000
ANX	r				0.659	0.744	0.770	0.705	0.688	0.559	0.573	0.604	0.892	0.176	0.112	0.840	0.868	0.905
	p				0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.115	0.315	0.000	0.000	0.000
O-C	r					0.716	0.663	0.606	0.552	0.388	0.283	0.559	0.812	0.151	0.092	0.533	0.541	0.565
	p					0.000	0.000	0.000	0.000	0.000	0.010	0.000	0.000	0.175	0.413	0.000	0.000	0.000
DEP	r						0.755	0.689	0.760	0.463	0.462	0.622	0.898	0.210	0.156	0.669	0.626	0.617
	p						0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.059	0.161	0.000	0.000	0.000
I-S	r							0.719	0.758	0.565	0.369	0.557	0.860	0.181	0.103	0.635	0.618	0.629
	p							0.000	0.000	0.000	0.001	0.000	0.000	0.103	0.358	0.000	0.000	0.000
PSY	r								0.668	0.537	0.441	0.641	0.812	0.139	0.072	0.584	0.547	0.575
	p								0.000	0.000	0.000	0.000	0.000	0.213	0.521	0.000	0.000	0.000
PAR	r									0.575	0.400	0.611	0.803	0.226	0.167	0.589	0.564	0.580
	p									0.000	0.000	0.000	0.000	0.041	0.135	0.000	0.000	0.000
HOS	r										0.408	0.416	0.619	0.194	0.120	0.486	0.489	0.516
	p										0.000	0.000	0.000	0.080	0.284	0.000	0.000	0.000
PHOB	r											0.332	0.556	0.055	-0.047	0.507	0.532	0.471
	p											0.002	0.000	0.623	0.677	0.000	0.000	0.000
ADD.S	r												0.730	0.187	0.135	0.517	0.500	0.515
	p												0.000	0.092	0.227	0.000	0.000	0.000
GSI	r													0.204	0.129	0.752	0.743	0.755
	p													0.066	0.249	0.000	0.000	0.000
JFLS-8	r														0.963	0.075	0.066	0.084
	p														0.000	0.505	0.554	0.455
JFLS-20	r															0.017	0.013	0.052
	p															0.882	0.906	0.644
PHQ-4	r																0.878	0.864
	p																0.000	0.000
PHQ-9	r																	0.922
	p																	0.000

5. DISCUSSION AND CONCLUSION

5.1.Study population

A significant relationship was found between FM, TMD, and age in previous studies. Wolfe et al. [151] reported that the prevalence of FM increased with age. Meanwhile, according to White et al., [152] the prevalence of this syndrome is proportional to population age, reaching a peak at 50–60 years of age. Migliorini et al. [153] reported in a systematic review in 2021 that the effect of age on FM is debatable. Further, Ferreira et al. [154] stated that the prevalence of TMD increased with age. In general population studies, nevertheless, it appeared that children and adolescents had a higher prevalence of TMD symptoms than adults. [155, 156] In present study, participants were in the 30-65 age range. No significant relationship was found between age and TMD and FM.

While the rheumatology clinic patients were included in the case group, the control group included the patients who attended the dental hospital. This were because other painful rheumatology conditions could affect the results of the study in the patients who attended the clinic of rheumatology.

In the period that we conducted this study, 11 male patients who attended the Istanbul Medeniyet University Goztepe Hospital were diagnosed with FM. Only three of them agreed to participate in the study. Thus, no male participants were included in the study, as the sample size would be insufficient for gender comparison. As this study, in previous studies on the relationship between TMD and FM, all or most participants were female. This is due to the fact that both conditions are more common in women. [14, 15, 17, 129, 131]

Patients who were diagnosed with FM for the first time were included in the case group because FM treatment may affect the survey results. In addition, since medications used for FM treatment, such as ibuprofen, acetaminophen, or muscle relaxants, may affect the diagnosis of TMD, patients who had previously received treatment were not included in the study. The SCL-90-R, GAD-7, and PHQ-9 scores may also be affected by these medications. Additionally, sedatives, antidepressants, and muscle relaxants may affect the PSQI scores because they improve sleep. As a result, people who take these medications were excluded from the study. [14, 158, 159]

5.2.Study setting

Many studies have investigated the prevalence of TMD signs and symptoms, mood disorders, and sleep quality in FM patients. [2, 10, 11, 14, 15, 17, 129, 130, 150]

The studies used a variety of statistical methods, questionnaires, and diagnostic criteria. In many studies conducted to determine the prevalence of TMD in patients with FM, no diagnostic criterion was used, or diagnostic criterion was used only for one of these diseases. [2, 15, 129, 130] In this study, the ACR 2016 criterion, and DC/TMD were used for FM and TMD diagnosis as these are the most recent diagnostic methods.

The AAOP screening, the Craniomandibular index, RDC/TMD, and DC/TMD are methods for the diagnosis of TMD. Since its publication in 1992, the most used diagnostic criterion for the diagnosis of TMD is RDC/TMD. RDC/TMD has been modified over time, and a new evidence-based DC/TMD criteria has been published in 2014. [50] In the present study, DC/TMD, the most up-to-date evidence-based criterion, was preferred for the diagnosis of TMD.

ACR 1990 criteria was used to diagnose FM in most previous studies. [2, 14, 15, 17, 129, 130, 131] According to this criteria, the patient should feel pain in 11 of the 18 tender points to diagnose FM. However, as reported by Wolfe et al, [120] patients with FM do not always experience pain in 11 of 18 tender points. In the ACR 2010 and ACR modified 2010 criteria, the tender point examination in the ACR 1990 criteria was removed, and there was misclassification in the diagnosis. To prevent these misclassifications encountered in the diagnosis, the ACR 2016 criteria was published and "widespread pain, which is present in at least 4 or 5 regions" was added to the criteria. [90] ACR 2016 was preferred because it is an up-to-date version and the problems encountered in the diagnosis concerning the previous criteria were eliminated.

Numerous studies used OHIP-14 and OHIP-49, which are meaningful since they describe OHRQoL variables. Additionally, the World Health Organization Quality of Life (WHOQOL) and the 36-Item Short Form Health Survey questionnaire (SF-36) are validated questionnaires that assess general health-related quality of life variables, which is important for TMD as an interdisciplinary condition. [160, 161] Furthermore, the WHOQOL has a subdivision that includes the environment and individuals as a whole, whereas the SF-36 is primarily concerned with patients' mental and psychological health. [162, 163] OHIP, the most widely used OHRQoL instrument, was used in the present study. The shortened Turkish form, OHIP-14, which was validated by Mumcu et al., [134] was preferred. It is an excellent tool for assessing the aspect of disability in patients with TMD. [164-168] While SF-36 was used in studies evaluated the relation between TMD and quality of life, OHIP was preferred in most of them. We used the most widely used OHRQoL instrument, OHIP, in this study. [145, 147, 166, 167, 168 224]

The use of screening instruments may assist in identifying possible psychological disorders. Self-report inventories are simple to complete and administer and are readily available in clinical practice. Thus, numerous previous studies have used a variety of self-report inventories to assess the psychological status of patients with TMD and/or chronic pain, including the SCL-90-R, the Minnesota Multiphasic Personality Inventory, the Spielberger State-Trait Anxiety Inventory, the Beck Depression Inventory, and the Beck Anxiety Inventory. The SCL-90-R is a simplicity psychometric self-reporting instrument used extensively to evaluate psychological distress and multi-dimensional psychopathology. [169] It can be used as a screening tool by non-specialists. As a result, the SCL-90-R has been used in several clinical studies that analyze differences in personality features within TMD subgroups. [35, 169] We preferred to use the SCL-90-R because it is valid, reliable, and does not require a specialist. [170]

PSQI or the Medical Outcomes Study Sleep Scale were recommended for the diagnosis of sleep disturbances. Epworth Sleepiness Scale, the inappropriate sleep composite score or the Stanford sleepiness scale were recommended for the evaluation of sleepiness during the day. All these questionnaires should be used together with a detailed medical history focusing on common sleep disorders. If it is necessary, patients should be referred to sleep laboratories for the polysomnographic investigations. In this study, we preferred the PSQI which is a reliable, valid, and time-effective measure of sleep dysfunction in patients with chronic pain. [171-173]

5.3.Fibromyalgia and Temporomandibular Disorder

When our findings were compared with the findings reported by previous studies, they were found to be consistent. Our study demonstrated a higher prevalence of TMD in the case group than in the controls. [11, 14-17]

Except for the study by Plesh et al., [11] all the previous studies used the ACR criteria to diagnose FM. [2, 14-17, 129, 130] Plesh et al. [11] examined patients who had previously been diagnosed with FM, and they diagnosed TMD using RDC/TMD and determined the prevalence of TMD in FM patients.

As was the case with the study by Plesh et al., [11] some other studies also used RDC/TMD for the diagnosis of TMD, [14, 15, 17] and some studies used the AAOP criteria. [129, 130] By adopting a different approach for the diagnosis of TMD, Leblebici et al. [2] used a subjective symptoms questionnaire, an interview with detailed medical history, and magnetic resonance imaging (MRI). On the other hand, Korszun et al. [16]

questioned whether the patients who were diagnosed with FM using the ACR criteria had previously been diagnosed with TMD.

Although different criteria have been used for the diagnosis of TMD, the prevalence of TMD in FM patients was found to be high in the present study similar to how it was in the case of the previous studies. But contrary to the other studies, [2, 11, 15, 17] the intra-articular joint disease was found to be more common than MP. The number of participants is either close to or higher than in the other studies. More objective results about TMD subgroups can be obtained if studies are conducted with more participants.

There are studies that have evaluated bruxism in FM patients as is also the case with the present study. [15, 17] Fraga et al. [15] evaluated bruxism using the questionnaire developed by Conti et al. They reported that 25% of the participants had bruxism. Pimentel et al. [17] found self-reported bruxism in the case group, and its incidence was higher than in the case of the control, though the difference was not statistically significant. In the present study, contrary to other studies, bruxism was more common in the case group. The signs of bruxism were observed in 68.3% of the case group in the present study, which was significantly higher than was the case with the control group. ($p < 0.001$) Not all patients may be aware that they are bruxers. Self-reported bruxism evaluations could have missed bruxism being reported in those cases where the patients were unaware of having it. It is possible that this had an impact vis-à-vis the difference observed between the case and control groups.

5.4.Fibromyalgia

5.4.1. Sleep Quality

Sleep disturbances and non-refreshing sleep are common complaints among FM patients. [5, 14] A strong link has been found, in previous research studies, between sleep and chronic pain. [6, 14, 174, 175]

According to the PSQI global score, this study demonstrated high prevalence of poor sleepers in the case group. The PSQI global score was determined to be 4.9 ± 3.8 in the case group and 1.2 ± 1.5 in the control group. This difference is statistically significant ($p < 0.001$). Our findings are in line with those of the previous studies, which had also found high prevalence of poor sleepers. [14, 174, 176-182]

In previous studies, researchers mostly used PSQI to assess sleep quality, [14, 174, 176-178, 180-182] and poor sleep quality was reported in the FM patients in all of these studies. Landis et al. [179] used the sleep quality items from The Washington

Women's Health Diary to assess sleep quality. In their study too, which used a different method, poor sleep quality was reported in FM patients.

Wu et al. [183] reported in their meta-analysis that when sleep was measured using polysomnography, individuals with FM had longer periods of wakefulness during sleep, shorter sleep durations, and spent more time in light sleep than the healthy controls.

5.4.2. Oral Health-Related Quality of Life

To the best of our knowledge, in spite of health-related quality of life being evaluated in FM patients in the existing literature, OHRQoL has not been evaluated. FM patients have been reported to have a lot of oral symptoms. One of these oral symptoms is TMD. Considering that OHRQoL would be negatively affected due to these oral symptoms, we compared the OHIP-14 scores of the case and control groups.

According to the results of our study, the mean OHIP-14 global score was determined to be 12.51 in the case group, while it was determined to 10.02 in the control group. Although the case group showed better results, the difference was not significant. Possibly, a significant difference can be found in studies with more participants.

5.4.3. Psychological Status

FM is characterized by increased sensitivity to physical and psychological stress. [14, 184] Fatigue while suffering from chronic pain has been linked by previous research studies to symptoms of somatization and depression. [14, 185, 186] Notably, there have been studies that have found a link between FM and psychosocial disorders such as ANX, DEP, and SOM. [176, 187-189]

Prior studies on FM and ANX have found evidence of the two conditions being linked to each other. [190-194] While several studies used the State Trait Anxiety Inventory Scale, [190, 193, 194] one study used the Hospital Anxiety and Depression Scale, [191] and another used both the Hospital Anxiety and Depression Scale and the State Trait Anxiety Inventory Scale. [192] SCL-90-R and GAD-7 were used to assess anxiety in our study. Anxiety levels in the case group were higher than in the control group in this study, as they have been in previous studies. The difference between both SCL-ANX and GAD-7 scores between cases and controls were significant.

Gormsen et al. [188] also used GAD-7 and SCL-92 to conduct a psychological assessment. Although FM patients and neuropathic pain patients were compared, they reported that patients with FM had significantly higher scores on DEP, ANX, SOM, and O-C.

Many previous studies have found likenesses between FM and DEP. [188-195] Fatigue, sleep problems, muscle aches, concentration problems, and cognitive overload are all symptoms of FM and DEP. Antidepressants often produce a positive response in FM patients. However, it is still unknown whether DEP is one of the causes or a co-occurring disorder with FM.

While several studies used the Hospital Anxiety and Depression Scale, [191, 192, 194] one study has assessed depression using the Beck Depression Inventory. [193] In our study, we used SCL-90-R and PHQ-9, which is a DC/TMD axis II component. Our finding was that the SCL-DEP and PHQ-9 scores were higher in the case group than in the control group. This finding, which was supported previous studies, was statistically significant.

There are also studies that have used SCL-90-R to assess the psychological status of FM patients. [14, 188, 196] Although Gormsen et al. [188] compared FM patients with neuropathic pain patients, both reported significantly higher SOM subscale values. SOM was assessed in our study using the SCL-90-R SOM subscale, and as previously reported, the SCL-SOM score was significantly higher in the case group.

In one of these studies conducted by Balasubramaniam et al., [14] they compared FM and failed back syndrome, and they found that FM patients had higher SOM and O-C values. In our study, all SCL-90-R subscales except SCL-PHOB were found to be significantly higher than the control group. The difference between the findings of the present study and those of the study by Balasubramaniam et al. [14] could be due to the comparison in the latter with failed back syndrome, which is another chronic pain syndrome.

5.5.Temporomandibular Disorders

5.5.1. Bruxism

Owing to the complex etiology of both TMD and bruxism, the link between the two is debatable. [197, 198] Despite the fact that the subject is controversial, some studies have found an association between bruxism and TMD. [143, 197, 198] These studies used a variety of methods to assess bruxism, including laboratory-based polysomnography, self-reported questionnaires, and self-reported questionnaires along with clinical examination.

Bruxism was significantly higher in the TMD patients when compared to the non-TMD patients in the studies that assessed self-reported bruxism. [37, 199-202] While RDC/TMD was used in some of these studies to diagnose TMD, [37, 199, 200] in one of

the study, the researchers concerned preferred The Craniomandibular Disorders Protocol, [201] and in another study, DC/TMD was preferred. [202]

In the studies involving self-reported bruxism, TMD, MP, and TMJ pain were found to be significantly associated with bruxism. [37, 199-202] Nonetheless, the validity of self-reported bruxism was evaluated in a study conducted by Raphael et al. [203] in 2015, and they believed that self-reporting of bruxism is highly unlikely to be a valid indicator of true sleep bruxism as those affected by bruxism are not necessarily aware of it. Hence, in their study, the diagnosis of bruxism was based on clinical examination and detailed anamnesis. In another study published in 2003, Manfredini et al. [143] also used this method to diagnose bruxism, and TMD was diagnosed with the use of RDC/TMD. They concluded that among the TMD patients, bruxism cases were significantly high.

Some studies used a questionnaire in addition to tooth-wear evaluation. [204, 205] In one of these studies, DC/TMD was preferred for TMD diagnosis, [204] while in another one, RDC/TMD was used. [205] TMD and bruxism were also linked by those studies that included a clinical examination for bruxism's diagnosis. [143, 204, 205]

There have also been studies that have used a laboratory-based polysomnographic investigation to diagnose bruxism. In these studies, the TMD diagnosis was based on RDC/TMD. [206-208] In the polysomnographic investigation, MP was reported to be linked with bruxism. [207, 208] This link was also found in some other studies. TMD, on the other hand, has not been linked to bruxism by the polysomnographic studies. [206]

Due to the different diagnostic and statistical methods used in the different studies and the complicated etiology of both TMD and bruxism, the relationship between bruxism and TMD is controversial. However, as was the case with many other studies, the findings of this study revealed a link between TMD and bruxism.

5.5.2. Trauma

One of the etiological factors of TMD has been proposed to be whiplash trauma. However, the relationship between trauma and TMD is unclear. Though some studies concluded that there is no relation between TMD and trauma, some others found the topic to be contentious. Notably, there are also studies that show some relationship between TMD and trauma.

Patients who have suffered whiplash trauma have been examined in this regard in some studies. [209, 210] Jaw pain was found to be significantly common in the case groups by Haggman-Henrikson et al. [209] Pajedien et al. [210] reported that TMD symptoms are significantly more common in the case group.

In some of the studies, the patients with whiplash trauma were examined periodically, [211-213] and one of these studies reported that TMD was found to be more common in trauma patients during both acute and follow-up examinations. [212] According to the other studies on this subject, a whiplash injury is not a significant risk factor for TMD. [211, 213]

In their study published in 2003, Velly et al. [37] asked 83 participants diagnosed with MP based on RDC/TMD and 100 healthy participants about their trauma history. Drawing on the findings of their study, they reported no significant link between MP and trauma.

In the present study, self-reported trauma was compared between case and control groups as Velly et al. [37] did. Trauma was found in 40% of the TMD patients but only in 9.4% of the control group members. This difference is statistically significant.

Because of the lack of homogeneity in the study population and the lack of standardization in data collection, the relation between TMD and trauma is still controversial. More objective results can be obtained by increasing the number of prospective studies with whiplash patients and duly following-up with them.

5.5.3. Sleep Quality

TMDs are known to affect sleep quality as suggested by the findings of the research conducted on individual TMD patients and community samples. [214-216] Sleep quality has been shown to deteriorate as TMD's severity increases. Moreover, painful TMDs increase the likelihood of poor quality of sleep. [217]

The findings of our study support the data available in this regard in the existing literature. The PSQI global score was higher in TMD patients (3.72 ± 3.82) compared to the control group (2.06 ± 2.29). These results are statistically significant.

While RDC/TMD was used for TMD diagnosis in some TMD and sleep quality studies, [218-220] the DC/TMD method was used in others, [171, 177, 217] including the present study. Further, PSQI was used to assess sleep quality in all of these studies. All of these studies reported that the TMD group had significantly higher PSQI global scores than the control group. [171, 177, 214, 217-220] However, in TMD patients, the global PSQI scores in these studies are higher than was the case in our study. Racial and ethnic differences among the participants in the existing studies, on the one hand, and in our study, on the other, may also be responsible for the higher PSQI scores in the findings of the other studies as against our study's findings.

In one study, it was reported that the impairment of sleep quality was caused by the depressive symptoms frequently seen in TMD patients. [221] Our findings support this study's observation despite the fact that different methods were used by the two studies concerned. In our study, the PSQI global score and the PHQ-9 scores were highly correlated.

There are also studies comparing sleep quality in TMD subgroups. While Renner-Sitar et al. [218] found that only pain-related diagnoses were associated with a significant decrease in sleep quality, and Yap et al. [217] found that both painful and non-painful TMDs were associated with significantly poorer sleep quality. Benoliel et al. [171] compared sleep quality in TMD subgroups, but they found no differences. The present study also found that TMD has a negative impact on sleep quality, but we feel that more research is needed to compare TMD's subgroups in this regard.

5.5.4. Oral Health-Related Quality of Life

As expected, in the existing literature, the global OHIP-14 score was higher in TMD patients than in patients without TMD. In our study, the mean OHIP-14 total score was found to be 14.7 ± 9.23 in TMD patients and 5.8 ± 6.1 in patients without TMD.

When compared to control subjects, the TMD patients had higher global OHIP-14 scores. The mean global OHIP-14 score for TMD patients in the present study was similar to the TMD patients' scores in the previous studies, [164, 166, 167, 222] but lower than in some of the cases. [147, 168, 223] It is proposed that differences in race, ethnicity, and psychology could be partially responsible for the differences in the findings of the present study and the findings of other studies.

Pigozzi et al. [161] concluded in their systematic review and meta-analysis studies that included studies using both RDC/TMD and DC/TMD that TMD patients have a lower quality of life.

It is difficult to compare differences in the OHIP-14 scores between TMD patients and controls in other studies because most control groups were drawn from the general population. [145, 147, 166, 224] Furthermore, there were no control groups in some of the studies. [167, 168] Miettinen et al. [222] included dental students as a control group.

Given that there are studies that show a relationship between the OHIP-14 scores and ages of study participants, [225] similar age groups were included in our study from among patients with and without TMD. Almoznino et al. [164] and Balık et al. [223] reported, no significant relationship was found between age and OHRQoL. No significant relationship of TMD with marital status was found in our study. However, some studies

have reported a significant difference between OHRQoL and marital status or age. [167, 168, 222, 226]

5.5.5. Psychological Status

Prior studies on TMD have found a link between it and psychosocial disorders like ANX, DEP, and SOM. [37, 169, 196, 227-229] Except for the study by Simoen et al., [228] all of these studies used the SCL-90-R to assess psychological status. GAD-7 and PHQ-9 from the DC/TMD axis II were preferred by Simoen et al. [228] In the present research study, SCL-90-R, GAD-7, and PHQ-9 were used.

In their studies focusing on SCL-DEP and SCL-SOM, the patients with MP and TMJ pain were found to be at the most psychosocial risk by Yap et al. [35] Manfredini et al. [169] compared 3 groups, MP, TMJ pain, and both TMJ pain and MP. SOM and DEP values were found high in all these groups; however, no statistically significant difference was found. This result could be explained by the fact that all of the three groups suffered from painful TMD.

Filligim et al. [196] used SCL-90-R SOM and DEP subscales and the State Trait Anxiety Inventory Scale. While SOM and DEP were significantly higher in the TMD patients, there was no difference in the anxiety values between these two groups. Bertoli et al. [227] reported a high prevalence of DEP and ANX in different samples of TMD patients. Canales et al. [229] used SCL-90-R as well as RDC/TMD axis II. They reported that the patients with TMD frequently exhibit high to moderate levels of SOM and DEP. According to Simoen et al., [228] the patients suffering from TMD have higher PHQ-9 and GAD-7 scores than the general population.

In the present study, when TMD patients and non-TMD patients were compared, only the SCL-DEP scores were found to be significantly higher in the case group. Although they were found high in the SCL-ANX and SCL-DEP case groups, the differences were not significant. There was no difference between PHQ-9 and GAD-7 scores. This may be due to the fact that patients with FM, another chronic pain syndrome, were in the control group.

Two studies of Velly et al. [37, 230] that used SCL-90-R were published in 2002 and 2003. In their study published in 2002, they found a relationship between DD and ANX, but not between DD and DEP. In our study, no relationship was found between DD and SOM, ANX, or DEP. In the 2003 study of Velly et al., [37, 230] MP patients and the control group were compared. SOM and ANX were found to be associated with MP, but no relationship was found between DEP and MP. However, in the present study, the

SCL-DEP score in patients with myalgia is higher than those without myalgia. No relation was found between SOM and myalgia and ANX and myalgia.

5.5.6. Functional Limitation of the Masticatory System

Functional limitations of the jaw have been reported in a variety of conditions, including TMD, motor neuron degeneration, and atypical odontalgia. [231-234] Previous research has found that TMD and neurological conditions primarily affect mobility and mastication but not communication. [234-237] According to a study, it is difficult to correlate functional limitations with the diagnosis of various types of TMD. [232] Other studies have, nevertheless, shown that global limitations are mainly impacted by arthralgia than by combined joint and muscle pain, myalgia, and, least of all, internal derangement of TMJ without pain. This suggests that chewing may relate more to mechanical or structural jaw dysfunction, leading to more joint pain than muscle pain. [236, 238]

Self-reported jaw function limitation is affected mostly by orofacial pain than by intracapsular disorder, and it is largely caused by TMD-related headache. Further, according to Fetai et al., it is affected more by pain intensity than chronicity. [237]

In the present study, as in the previous literature, the JFLS-20 score in TMD patients was significantly higher than those without TMD.

5.6. Bruxism

The results reported by the patients participating in this study were compared for bruxers and non-bruxers. The most significant finding was that the quality of life and sleep for those with bruxism was poorer when compared to the control participants.

5.6.1. Sleep Quality

Several research teams have attempted to assess the relationship between sleep bruxism and sleep quality over the years. These studies used different diagnostic methods and assessed quality of sleep also by using various methods that are used for assessing sleep quality in bruxers. The American Academy of Sleep Medicine's diagnostic criteria for sleep bruxism were used to make a diagnosis of bruxism in many of these the studies. [239-241] Another research focused on self-reported bruxism. [242] The PSQI was used to assess sleep quality in all of these studies just as it was used in the present study.

In addition to the American Academy of Sleep Medicine's Diagnostic Criteria for Sleep Bruxism, two of the said studies used the Bruxoff portable electromyography and electrocardiography device for bruxism diagnosis, [239, 240] while another study opted for a laboratory-based polysomnographic investigation. [243]

The two studies which used the same methods to diagnose bruxism and assess sleep quality, yielded different results even with similar numbers of participants. Camara-Souza et al. [240] found that bruxers had poorer sleep quality than non-bruxers. Ohlmann et al. [239] reported no association between sleep bruxism and sleep quality. However, the individuals in Ohlmann et al.'s study [239] were not matched age-wise and gender-wise, and probable sleep bruxism co-effects, such as TMD, were not ruled out. Moreover, these individual study subjects were pre-selected as required by the original study design. This could be the reason for the differences in the results of these two studies.

Previous literature data show that patients with bruxism had significantly lower sleep quality. [241, 242] However, there is also a study that evaluated the severity of bruxism as it linked to sleep quality, and this study found that the severity of bruxism has no effect on sleep quality.[243]

In our present study, the sleep quality assessed with the help of PSQI was compared as far as the bruxism and non-bruxism patients were concerned. Our research led us to the conclusion that bruxers have poorer sleep quality than the controls. However, due to conflicting scientific reports on the impact of sleep bruxism on sleep quality, we suggest that more research is needed to consider in further detail the co-occurring disorders of sleep bruxism and poor sleep quality.

5.6.2. Oral Health-Related Quality of Life

A majority of the bruxism- and OHRQoL-related research has been conducted on children. It must be noted, though, that studies have also been conducted on adults. In the studies conducted on adults, Bruxism was found to have a negative impact on OHRQoL in adults, [240, 244, 245] though the studies on children indicating otherwise. [246, 247] Obviously, the disparity could be due to the different populations, but it is also possible that the factors influencing the children's responses to global ratings are unclear, and parents/caregivers may have underestimated the impact of oral problems on a child's quality of life. [248]

The studies that used the Diagnostic Criteria of The International Classification of Sleep Disorders for the diagnosis of bruxism, reported that bruxism had a negative impact on OHRQoL. [244, 245]

In their study published in 2019, Camara-Souza et al. [240] compared OHRQoL between groups, which they assessed with the help of OHIP-14 in addition to sleep quality. They concluded that bruxers had significantly higher total OHIP-14 scores than the controls.

As was the case with the said studies, in the adults that we studied, the average OHIP-14 total score in the case of the bruxers was significantly higher than in the case of the control group. In the bruxers, the mean OHIP-14 total score was 14.32 ± 9.82 , while in the control group, it was 8.36 ± 7.67 . The OHIP-14 scores, thus, revealed that bruxers had experienced a more negative impact on their OHRQoL compared to the controls.

5.6.3. Psychological Status

Some of the previous literatures have found an association between bruxism and ANX/DEP symptoms. [249-254] A study had also established a link between stress and bruxism by demonstrating that the level of catecholamine, which is typically associated with emotional stress, was high in patients with bruxism. [255]

According to Velly et al., [37] there is no relation between bruxism and the psychological status. They used SCL-90-R to determine the psychological status of the patients. Similarly, we also preferred using SCL-90-R. However, we also used the DC/TMD axis II elements PHQ-9 and GAD-7. Upon analyzing the results, we found no significant difference between PHQ-9 and GAD-7 scores. Of the SCL-90-R subscales, only DEP and O-C scores were significantly different between bruxers and non-bruxers.

Thus, contrary to what the previous literature suggests, the absence of a relationship between ANX and bruxism in the case of the results of our study may be due to the presence of TMD in individuals without bruxism. Nevertheless, the SCL-90-R scores did show a relationship between DEP and bruxism. This supports the findings of previous studies that there is no difference in the PHQ-9 score.

In conclusion,

- This study's findings confirmed our hypothesis that TMD is more common in FM patients than in healthy controls. Simultaneously, it was also proven that the prevalence of FM was higher in the TMD patients. Although studies evaluating FM prevalence in TMD subgroups have reported that individuals with FM have more MP, we found no significant difference in this regard between patients with and without muscle pain. We suggest that the TMD subgroups should be evaluated by including more study participants.
- This study found high levels of psychological distress in both FM and TMD patients. However, although some scores such as SCL-SOM, SCL-ANX, GAD-7, and PHQ-9 turned out to be high in the case of our TMD patients, which is contrary to the data in the existing literature, yet the difference is not significant.
- According to the findings, patients with TMD and patients with FM experience poorer quality of sleep. TMD has a significant negative impact on OHRQoL. Even though we predicted that FM would have a negative impact on OHRQoL due to the frequent oral symptoms in FM patients, the difference between the OHIP-14 scores of TMD and FM patients was not significant. If future studies are performed by increasing the number of study participants, it can be assessed whether FM has a negative effect on OHRQoL.
- In this study, FM and TMD were diagnosed with the most up-to-date criteria: ACR 2016 and DC/TMD. The study is performed with objective diagnostic methods.
- We suggest that TMJ should also be examined while diagnosing FM patients. TMD management is important to improve the quality of life of FM patients. A multidisciplinary approach needs to be adopted while managing both FM and TMD in the patients concerned. In such cases, the psychologist, rheumatologist, and dentist must work in coordination with each other to manage these syndromes effectively.

6. REFERENCES

1. Thilander, B., et al. Prevalence of temporomandibular dysfunction and its association with malocclusion in children and adolescents: an epidemiologic study related to specified stages of dental development. *Angle Orthod.* 2002; **72**(2): p. 146-54.
2. Leblebici, B., et al. Coexistence of fibromyalgia, temporomandibular disorder, and masticatory myofascial pain syndromes. *Rheumatol Int.* 2007. **27**(6): p. 541-4.
3. Headache Classification Subcommittee of the International Headache, S., The International Classification of Headache Disorders: 2nd edition. *Cephalalgia*, 2004. **24 Suppl 1**: p. 9-160.
4. Bonjardim, L.R., et al. Anxiety and depression in adolescents and their relationship with signs and symptoms of temporomandibular disorders. *Int J Prosthodont.* 2005; **18**(4): p. 347-52.
5. Wolfe, F., et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. Report of the Multicenter Criteria Committee. *Arthritis Rheum.* 1990; **33**(2): p. 160-72.
6. Moldofsky, H. Sleep and pain. *Sleep Med Rev.* 2001; **5**(5): p. 385-396.
7. Rhodus, N.L., et al. Oral symptoms associated with fibromyalgia syndrome. *J Rheumatol.* 2003; **30**(8): p. 1841-5.
8. Manfredini, D., et al. Comparison of masticatory dysfunction in temporomandibular disorders and fibromyalgia. *Minerva Stomatol.* 2004; **53**(11-12): p. 641-50.
9. Hedenberg-Magnusson, B., M. Ernberg, and S. Kopp. Presence of orofacial pain and temporomandibular disorder in fibromyalgia. A study by questionnaire. *Swed Dent J.* 1999; **23**(5-6): p. 185-92.
10. Dao, T.T., W.J. Reynolds, and H.C. Tenenbaum. Comorbidity between myofascial pain of the masticatory muscles and fibromyalgia. *J Orofac Pain.* 1997; **11**(3): p. 232-41.
11. Plesh, O., F. Wolfe, and N. Lane, The relationship between fibromyalgia and temporomandibular disorders: prevalence and symptom severity. *J Rheumatol.* 1996; **23**(11): p. 1948-52.

12. McCain, G.A. and R.A. Scudds. The concept of primary fibromyalgia (fibrositis): clinical value, relation and significance to other chronic musculoskeletal pain syndromes. *Pain*. 1988; **33**(3): p. 273-87.
13. Friction, J.R., The relationship of temporomandibular disorders and fibromyalgia: implications for diagnosis and treatment. *Curr Pain Headache Rep*. 2004; **8**(5): p. 355-63.
14. Balasubramaniam, R., et al. Prevalence of temporomandibular disorders in fibromyalgia and failed back syndrome patients: a blinded prospective comparison study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2007; **104**(2): p. 204-16.
15. Fraga, B.P., et al. Signs and symptoms of temporomandibular dysfunction in fibromyalgic patients. *J Craniofac Surg*. 2012; **23**(2): p. 615-8.
16. Korszun, A., et al. The relationship between temporomandibular disorders and stress-associated syndromes. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1998; **86**(4): p. 416-20.
17. Pimentel, M.J., et al. Features of temporomandibular disorders in fibromyalgia syndrome. *Cranio*. 2013; **31**(1): p. 40-5.
18. Okeson, J.P. *Management of Temporomandibular Disorders and Occlusion*. 7th ed. 2012.
19. Alomar, X., et al. Anatomy of the temporomandibular joint. *Semin Ultrasound CT MR*. 2007; **28**(3): p. 170-83.
20. Porto, G.G., et al. Comparison between human and rat TMJ: anatomic and histopathologic features. *Acta Cir Bras*. 2010; **25**(3): p. 290-3.
21. Wink, C.S., M. St Onge, and M.L. Zimny. Neural elements in the human temporomandibular articular disc. *J Oral Maxillofac Surg*. 1992; **50**(4): p. 334-7.
22. Sano, T. Recent developments in understanding temporomandibular joint disorders. Part 2: changes in the retrodiscal tissue. *Dentomaxillofac Radiol*. 2000; **29**(5): p. 260-3.
23. Langendoen, J., J. Muller, and G.A. Jull. Retrodiscal tissue of the temporomandibular joint: clinical anatomy and its role in diagnosis and treatment of arthropathies. *Man Ther*. 1997; **2**(4): p. 191-198.

24. Loughner, B.A., L.H. Larkin, and P.E. Mahan. Discomalleolar and anterior malleolar ligaments: possible causes of middle ear damage during temporomandibular joint surgery. *Oral Surg Oral Med Oral Pathol.* 1989; **68**(1): p. 14-22.
25. Johansson, A.S. and A. Isberg. The anterosuperior insertion of the temporomandibular joint capsule and condylar mobility in joints with and without internal derangement: a double-contrast arthrotomographic investigation. *J Oral Maxillofac Surg.* 1991; **49**(11): p. 1142-8.
26. Dijkgraaf, L.C., et al. Structure of the normal synovial membrane of the temporomandibular joint: a review of the literature. *J Oral Maxillofac Surg.* 1996; **54**(3): p. 332-8.
27. Scrivani, S.J., D.A. Keith, and L.B. Kaban. Temporomandibular disorders. *N Engl J Med.* 2008; **359**(25): p. 2693-705.
28. Racich, M.J. Orofacial pain and occlusion: is there a link? An overview of current concepts and the clinical implications. *J Prosthet Dent.* 2005; **93**(2): p. 189-96.
29. Cooper, B., et al. Trauma modifies strength and composition of retrodiscal tissues of the goat temporomandibular joint. *Oral Dis.* 1999; **5**(4): p. 329-36.
30. Chisnoiu, A.M., et al. Factors involved in the etiology of temporomandibular disorders - a literature review. *Clujul Med.* 2015; **88**(4): p. 473-8.
31. Suvinen, T.I. and P.C. Reade. Temporomandibular disorders: a critical review of the nature of pain and its assessment. *J Orofac Pain.* 1995; **9**(4): p. 317-39.
32. Dworkin, S.F. and D.L. Massoth. Temporomandibular disorders and chronic pain: disease or illness? *J Prosthet Dent.* 1994; **72**(1): p. 29-38.
33. Kight, M., R.J. Gatchel, and L. Wesley. Temporomandibular disorders: evidence for significant overlap with psychopathology. *Health Psychol.* 1999; **18**(2): p. 177-82.
34. Sipila, K., et al. Association between symptoms of temporomandibular disorders and depression: an epidemiological study of the Northern Finland 1966 Birth Cohort. *Cranio.* 2001; **19**(3): p. 183-7.
35. Yap, A.U., et al. Depression and somatization in patients with temporomandibular disorders. *J Prosthet Dent.* 2002; **88**(5): p. 479-84.
36. Ferrando, M., et al. Psychological variables and temporomandibular disorders: distress, coping, and personality. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2004; **98**(2): p. 153-60.

37. Velly, A.M., M. Gornitsky, and P. Philippe. Contributing factors to chronic myofascial pain: a case-control study. *Pain*. 2003; **104**(3): p. 491-499.
38. Johansson, A., et al. Gender difference in symptoms related to temporomandibular disorders in a population of 50-year-old subjects. *J Orofac Pain*. 2003; **17**(1): p. 29-35.
39. Phillips, J.M., et al. Clinical implications of sex in acute temporomandibular disorders. *J Am Dent Assoc*. 2001; **132**(1): p. 49-57.
40. Kim, M.R., T.M. Graber, and M.A. Viana. Orthodontics and temporomandibular disorder: a meta-analysis. *Am J Orthod Dentofacial Orthop*. 2002; **121**(5): p. 438-46.
41. Mohlin, B.O., et al. Malocclusion and temporomandibular disorder: a comparison of adolescents with moderate to severe dysfunction with those without signs and symptoms of temporomandibular disorder and their further development to 30 years of age. *Angle Orthod*. 2004; **74**(3): p. 319-27.
42. Diatchenko, L., et al. Genetic basis for individual variations in pain perception and the development of a chronic pain condition. *Hum Mol Genet*. 2005; **14**(1): p. 135-43.
43. Slade, G.D., et al. Orthodontic Treatment, Genetic Factors and Risk of Temporomandibular Disorder. *Semin Orthod*. 2008; **14**(2): p. 146-156.
44. Michelotti, A. and G. Iodice. The role of orthodontics in temporomandibular disorders. *J Oral Rehabil*. 2010; **37**(6): p. 411-29.
45. De Kanter, R.J., et al. Prevalence in the Dutch adult population and a meta-analysis of signs and symptoms of temporomandibular disorder. *J Dent Res*. 1993; **72**(11): p. 1509-18.
46. Costen, J.B. A syndrome of ear and sinus symptoms dependent upon disturbed function of the temporomandibular joint. 1934. *Ann Otol Rhinol Laryngol*. 1997; **106**(10 Pt 1): p. 805-19.
47. Schwartz, L.L. Pain associated with the temporomandibular joint. *J Am Dent Assoc*. 1955; **51**(4): p. 394-7.
48. Laskin, D.M. Etiology of the pain-dysfunction syndrome. *J Am Dent Assoc*. 1969; **79**(1): p. 147-53.
49. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. Headache Classification Committee of the International Headache Society. *Cephalalgia*, 1988. **8 Suppl 7**: p. 1-96.

50. Schiffman, E., et al. Diagnostic criteria for temporomandibular disorders (DC/TMD) for clinical and research applications: recommendations of the International RDC/TMD Consortium Network* and Orofacial Pain Special Interest Groupdagger. *J Oral Facial Pain Headache*. 2014; **28**(1): p. 6-27.
51. Simons, D.G., J.G. Travell, and L.S. Simons. Travell & Simons' Myofascial Pain and Dysfunction. The Trigger Point Manual : Upper Half of Body Vol. 1. Baltimore: Lippincott Williams & Wilkins; 1999.
52. Hong, C.Z., et al. Referred pain elicited by palpation and by needling of myofascial trigger points: a comparison. *Arch Phys Med Rehabil*. 1997; **78**(9): p. 957-60.
53. Fischer, A.A. Documentation of myofascial trigger points. *Arch Phys Med Rehabil*. 1988; **69**(4): p. 286-91.
54. Yap, E.C. Myofascial pain--an overview. *Ann Acad Med Singap*. 2007; **36**(1): p. 43-8.
55. Ghali GE, M.M., Waite PD, Peterson's principles of oral and maxillofacial surgery. 2012.
56. Murakami, S., et al. Magnetic resonance evaluation of the temporomandibular joint disc position and configuration. *Dentomaxillofac Radiol*. 1993; **22**(4): p. 205-7.
57. Naeije, M., et al. Disc displacement within the human temporomandibular joint: a systematic review of a 'noisy annoyance'. *J Oral Rehabil*. 2013; **40**(2): p. 139-58.
58. Tasaki, M.M., et al. Classification and prevalence of temporomandibular joint disk displacement in patients and symptom-free volunteers. *Am J Orthod Dentofacial Orthop*. 1996; **109**(3): p. 249-62.
59. Jussila, P., et al. Prevalence of Temporomandibular Disorders in the Northern Finland Birth Cohort 1966. *J Oral Facial Pain Headache*. 2017; **31**(2): p. 159-164.
60. de Leeuw R, L.G. American Academy of Orofacial Pain. Diagnosis and Management of TMDs. 2013.
61. Stegenga B, d.B.L. TMJ Disc Displacement. Quintessence Publishing Co, Inc.; 2006.

62. Young, A.L. Internal derangements of the temporomandibular joint: A review of the anatomy, diagnosis, and management. *J Indian Prosthodont Soc.* 2015; **15**(1): p. 2-7.
63. Martin, M.D., et al. Intubation risk factors for temporomandibular joint/facial pain. *Anesth Prog.* 2007; **54**(3): p. 109-14.
64. McNeill, C. Management of temporomandibular disorders: concepts and controversies. *J Prosthet Dent.* 1997; **77**(5): p. 510-22.
65. Curl, D.D. and G. Stanwood. Chiropractic management of capsulitis and synovitis of the temporomandibular joint. *J Orofac Pain.* 1993; **7**(3): p. 283-93.
66. Fujisawa, T., et al. A repetitive, steady mouth opening induced an osteoarthritis-like lesion in the rabbit temporomandibular joint. *J Dent Res.* 2003; **82**(9): p. 731-5.
67. Tabeling, H.J. and M.F. Dolwick. Rheumatoid arthritis: diagnosis and treatment. *Fla Dent J.* 1985; **56**(1): p. 16-8.
68. Celiker, R., Y. Gokce-Kutsal, and M. Eryilmaz. Temporomandibular joint involvement in rheumatoid arthritis. Relationship with disease activity. *Scand J Rheumatol.* 1995; **24**(1): p. 22-5.
69. Chenitz, J.E. Rheumatoid arthritis and its implications in temporomandibular disorders. *Cranio.* 1992; **10**(1): p. 59-69.
70. Nordahl, S., et al. Radiographic signs of bone destruction in the arthritic temporomandibular joint with special reference to markers of disease activity. A longitudinal study. *Rheumatology (Oxford).* 2001; **40**(6): p. 691-4.
71. Kobayashi, R., et al. Ankylosis of the temporomandibular joint caused by rheumatoid arthritis: a pathological study and review. *J Oral Sci.* 2001; **43**(2): p. 97-101.
72. Dworkin, S.F. and L. LeResche. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Craniomandib Disord.* 1992; **6**(4): p. 301-55.
73. Bossuyt, P.M., et al. Toward complete and accurate reporting of studies of diagnostic accuracy. The STARD initiative. *Am J Clin Pathol.* 2003; **119**(1): p. 18-22.
74. Truelove, E., et al. The research diagnostic criteria for temporomandibular disorders. III: validity of Axis I diagnoses. *J Orofac Pain.* 2010; **24**(1): p. 35-47.

75. Ohrbach, R., et al. The research diagnostic criteria for temporomandibular disorders. IV: evaluation of psychometric properties of the Axis II measures. *J Orofac Pain*. 2010; **24**(1): p. 48-62.
76. Manfredini, D., et al. Research diagnostic criteria for temporomandibular disorders: a systematic review of axis I epidemiologic findings. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2011; **112**(4): p. 453-62.
77. Williams, D.A. and D.J. Clauw. Understanding fibromyalgia: lessons from the broader pain research community. *J Pain*. 2009; **10**(8): p. 777-91.
78. Kia, S. and E. Choy. Update on treatment guideline in fibromyalgia syndrome with focus on pharmacology. *Biomedicines*. 2017; **5**(2).
79. Bazzichi, L., et al. One year in review 2016: fibromyalgia. *Clin Exp Rheumatol*. 2016; **34**(2 Suppl 96): p. S145-9.
80. Ata A.M., A.C. Fibromiyalji Tanımı, Epidemiyolojisi. *Türkiye Klinikleri* 2015.
81. Evcik, D., A. Ketenci, and D. Sindel. The Turkish Society of Physical Medicine and Rehabilitation (TSPMR) guideline recommendations for the management of fibromyalgia syndrome. *Turk J Phys Med Rehabil*. 2019; **65**(2): p. 111-123.
82. M Yunus, A.M. Fibromyalgia, restless legs syndrome, periodic limb movement disorder, and psychogenic pain., in *Arthritis and allied conditions: a textbook of rheumatology*. 1996.
83. Boissevain, M.D. and G.A. McCain. Toward an integrated understanding of fibromyalgia syndrome. I. Medical and pathophysiological aspects. *Pain*. 1991; **45**(3): p. 227-238.
84. Smith, J.A. and H.A. Smythe. Fibrositis syndrome. *Can Fam Physician*. 1977; **23**: p. 69-74.
85. Hench, P.K. and M.M. Mitler. Fibromyalgia. 1. Review of a common rheumatologic syndrome. *Postgrad Med*. 1986; **80**(7): p. 47-56.
86. Yunus, M.B., A.T. Masi, and J.C. Aldag. A controlled study of primary fibromyalgia syndrome: clinical features and association with other functional syndromes. *J Rheumatol Suppl*. 1989; **19**: p. 62-71.
87. Wolfe, F. and W. Hauser. Fibromyalgia diagnosis and diagnostic criteria. *Ann Med*. 2011; **43**(7): p. 495-502.
88. Wolfe, F., et al. The American College of Rheumatology preliminary diagnostic criteria for fibromyalgia and measurement of symptom severity. *Arthritis Care Res (Hoboken)*. 2010; **62**(5): p. 600-10.

89. Bennett, R.M., et al. Criteria for the diagnosis of fibromyalgia: validation of the modified 2010 preliminary American College of Rheumatology criteria and the development of alternative criteria. *Arthritis Care Res (Hoboken)*. 2014; **66**(9): p. 1364-73.
90. Wolfe, F., et al. 2016 Revisions to the 2010/2011 fibromyalgia diagnostic criteria. *Semin Arthritis Rheum*. 2016; **46**(3): p. 319-329.
91. Goldenberg, D.L., C. Burckhardt, and L. Crofford. Management of fibromyalgia syndrome. *JAMA*. 2004; **292**(19): p. 2388-95.
92. Harris, R.E. and D.J. Clauw. How do we know that the pain in fibromyalgia is "real"? *Curr Pain Headache Rep*. 2006; **10**(6): p. 403-7.
93. Vincent, A., et al. Prevalence of fibromyalgia: a population-based study in Olmsted County, Minnesota, utilizing the Rochester Epidemiology Project. *Arthritis Care Res (Hoboken)*. 2013; **65**(5): p. 786-92.
94. Goldenberg, D.L. Multidisciplinary modalities in the treatment of fibromyalgia. *J Clin Psychiatry*. 2008; **69 Suppl 2**: p. 30-4.
95. Jain, A.K., et al. Fibromyalgia syndrome: Canadian Clinical Working Case Definition, Diagnostic and Treatment Protocols—A Consensus Document. *Journal of Musculoskeletal Pain*. 2003; **11**(4): p. 3-107.
96. Buskila, D. Pediatric fibromyalgia. *Rheum Dis Clin North Am*. 2009; **35**(2): p. 253-61.
97. Wolfe, F., et al. Fibromyalgia criteria and severity scales for clinical and epidemiological studies: a modification of the ACR Preliminary Diagnostic Criteria for Fibromyalgia. *J Rheumatol*. 2011; **38**(6): p. 1113-22.
98. Ay, S. Fibromiyalji Sendromunun Patogenezi. *Türkiye Klinikleri*, 2015.
99. Ablin, J., L. Neumann, and D. Buskila. Pathogenesis of fibromyalgia - a review. *Joint Bone Spine*. 2008; **75**(3): p. 273-9.
100. Diatchenko, L., et al. The phenotypic and genetic signatures of common musculoskeletal pain conditions. *Nat Rev Rheumatol*. 2013; **9**(6): p. 340-50.
101. Arnold, L.M., et al. Family study of fibromyalgia. *Arthritis Rheum*. 2004; **50**(3): p. 944-52.

102. Buskila, D., et al. Increased rates of fibromyalgia following cervical spine injury. A controlled study of 161 cases of traumatic injury. *Arthritis Rheum.* 1997; **40**(3): p. 446-52.
103. Buskila, D., F. Atzeni, and P. Sarzi-Puttini. Etiology of fibromyalgia: the possible role of infection and vaccination. *Autoimmun Rev.* 2008; **8**(1): p. 41-3.
104. Hauser, W., et al. Emotional, physical, and sexual abuse in fibromyalgia syndrome: a systematic review with meta-analysis. *Arthritis Care Res (Hoboken).* 2011; **63**(6): p. 808-20.
105. Kivimaki, M., et al. Work stress and incidence of newly diagnosed fibromyalgia: prospective cohort study. *J Psychosom Res.* 2004; **57**(5): p. 417-22.
106. Lewis, J.D., et al. Central sensitization as a component of post-deployment syndrome. *NeuroRehabilitation.* 2012; **31**(4): p. 367-72.
107. Phillips, K. and D.J. Clauw. Central pain mechanisms in the rheumatic diseases: future directions. *Arthritis Rheum.* 2013; **65**(2): p. 291-302.
108. Staud, R. Chronic widespread pain and fibromyalgia: two sides of the same coin? *Curr Rheumatol Rep.* 2009; **11**(6): p. 433-6.
109. Offenbaecher, M. and M. Ackenheil. Current trends in neuropathic pain treatments with special reference to fibromyalgia. *CNS Spectr.* 2005; **10**(4): p. 285-97.
110. Mease, P., et al. Fibromyalgia syndrome module at OMERACT 9: domain construct. *J Rheumatol.* 2009; **36**(10): p. 2318-29.
111. Bigatti, S.M., et al. Sleep disturbances in fibromyalgia syndrome: relationship to pain and depression. *Arthritis Rheum.* 2008; **59**(7): p. 961-7.
112. Lentz, M.J., et al. Effects of selective slow wave sleep disruption on musculoskeletal pain and fatigue in middle aged women. *J Rheumatol.* 1999; **26**(7): p. 1586-92.
113. Glass, J.M. Fibromyalgia and cognition. *J Clin Psychiatry.* 2008; **69 Suppl 2**: p. 20-4.
114. Glass, J.M. and D.C. Park. Cognitive dysfunction in fibromyalgia. *Curr Rheumatol Rep.* 2001; **3**(2): p. 123-7.
115. Shuer, M.L. Fibromyalgia: symptom constellation and potential therapeutic options. *Endocrine.* 2003; **22**(1): p. 67-76.
116. Brand, K., et al. The fibromyalgia bladder index. *Clin Rheumatol.* 2007; **26**(12): p. 2097-2103.

117. Wood, P.B. Role of central dopamine in pain and analgesia. *Expert Rev Neurother.* 2008; **8**(5): p. 781-97.
118. Bennett, R.M. Clinical manifestations and diagnosis of fibromyalgia. *Rheum Dis Clin North Am.* 2009; **35**(2): p. 215-32.
119. Leskowitz, E. Energy-Based Therapies for Chronic Pain. 2008. p. 225-241.
120. Wolfe, F. The relation between tender points and fibromyalgia symptom variables: evidence that fibromyalgia is not a discrete disorder in the clinic. *Ann Rheum Dis.* 1997; **56**(4): p. 268-71.
121. Gur, M., A. Gulkesen, and G. Akgol. Comparison of ACR 1990 and ACR 2010 classification criteria in fibromyalgia syndrome. 2019.
122. Eriksson, P.O., et al. Symptoms and signs of mandibular dysfunction in primary fibromyalgia syndrome (PSF) patients. *Swed Dent J.* 1988; **12**(4): p. 141-9.
123. Karibe, H., et al. Comparison of patients with orofacial pain of different diagnostic categories. *Cranio.* 2011; **29**(2): p. 138-43.
124. Fujarra, F.J., et al. Temporomandibular disorders in fibromyalgia patients: are there different pain onset? *Arq Neuropsiquiatr.* 2016; **74**(3): p. 195-200.
125. Ayouni, I., et al. Comorbidity between fibromyalgia and temporomandibular disorders: a systematic review. *Oral Surg Oral Med Oral Pathol Oral Radiol.* 2019; **128**(1): p. 33-42.
126. Salvetti, G., et al. Clinical features of the stomatognathic involvement in fibromyalgia syndrome: a comparison with temporomandibular disorders patients. *Cranio.* 2007; **25**(2): p. 127-33.
127. Velly, A.M., et al. The effect of fibromyalgia and widespread pain on the clinically significant temporomandibular muscle and joint pain disorders--a prospective 18-month cohort study. *J Pain.* 2010; **11**(11): p. 1155-64.
128. Nobrega, J.C., et al. Differential diagnosis in atypical facial pain: a clinical study. *Arq Neuropsiquiatr.* 2007; **65**(2A): p. 256-61.
129. da Silva, L.A., et al. High prevalence of orofacial complaints in patients with fibromyalgia: a case-control study. *Oral Surg Oral Med Oral Pathol Oral Radiol.* 2012; **114**(5): p. e29-34.
130. Garcia-Moya, E.J., J.M. Montiel-Company, and J.M. Almerich-Silla. Case-control study of craniomandibular disorders in patients with fibromyalgia. *J Clin Exp Dent.* 2015; **7**(2): p. e293-8.

131. Pimentel, M.J., et al. Sleep quality and facial pain in fibromyalgia syndrome. *Cranio*. 2015; **33**(2): p. 122-8.
132. Ağargün, M., H. Kara, and O. Anlar. Pittsburgh uyku kalitesi indeksinin geçerliği ve güvenilirliği. *Türk Psikiyatri Dergisi*. 1996; **7**(2): p. 107-115.
133. Dağ, I. Belirti Tarama Listesi (Scl-90-R)'nin Üniversite Öğrencileri için güvenilirliği ve geçerliği. [Reliability and validity of the Symptom Check List (SCL-90-R) for university students.]. *Türk Psikiyatri Dergisi*. 1991; **2**(1): p. 5-12.
134. Mumcu, G., et al. Oral health related quality of life is affected by disease activity in Behcet's disease. *Oral Dis*. 2006; **12**(2): p. 145-51.
135. Schiffman, E. and R. Ohrbach. Executive summary of the Diagnostic Criteria for Temporomandibular Disorders for clinical and research applications. *J Am Dent Assoc*. 2016; **147**(6): p. 438-45.
136. Kroenke, K., R.L. Spitzer, and J.B. Williams. The PHQ-9: validity of a brief depression severity measure. *J Gen Intern Med*. 2001; **16**(9): p. 606-13.
137. Lowe, B., et al. Validation and standardization of the Generalized Anxiety Disorder Screener (GAD-7) in the general population. *Med Care*. 2008; **46**(3): p. 266-74.
138. Spitzer, R.L., et al. A brief measure for assessing generalized anxiety disorder: the GAD-7. *Arch Intern Med*. 2006; **166**(10): p. 1092-7.
139. Molina, O.F., et al. A clinical study of specific signs and symptoms of CMD in bruxers classified by the degree of severity. *Cranio*. 1999; **17**(4): p. 268-79.
140. Thorpy, M.J. Classification of sleep disorders. *J Clin Neurophysiol*. 1990; **7**(1): p. 67-81.
141. Lavigne, G.J., P.H. Rompre, and J.Y. Montplaisir. Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. *J Dent Res*. 1996; **75**(1): p. 546-52.
142. Chung, S.C., Y.K. Kim, and H.S. Kim. Prevalence and patterns of nocturnal bruxofacets on stabilization splints in temporomandibular disorder patients. *Cranio*. 2000; **18**(2): p. 92-7.
143. Manfredini, D., et al. Prevalence of bruxism in patients with different research diagnostic criteria for temporomandibular disorders (RDC/TMD) diagnoses. *Cranio*. 2003; **21**(4): p. 279-85.
144. Derogatis, L.R. SCL-90-R: Symptom Checklist-90-R: Administration, Scoring and Procedures manual. 1994.

145. Slade, G.D. and A.J. Spencer. Development and evaluation of the Oral Health Impact Profile. *Community Dent Health*. 1994; **11**(1): p. 3-11.
146. Locker, D. Measuring oral health: a conceptual framework. *Community Dent Health*. 1988; **5**(1): p. 3-18.
147. John, M.T., et al. Oral health-related quality of life in patients with temporomandibular disorders. *J Orofac Pain*. 2007; **21**(1): p. 46-54.
148. Dahlstrom, L. and G.E. Carlsson. Temporomandibular disorders and oral health-related quality of life. A systematic review. *Acta Odontol Scand*. 2010; **68**(2): p. 80-5.
149. Buysse, D.J., et al. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res*. 1989; **28**(2): p. 193-213.
150. Hedenberg-Magnusson, B., M. Ernberg, and S. Kopp. Symptoms and signs of temporomandibular disorders in patients with fibromyalgia and local myalgia of the temporomandibular system. A comparative study. *Acta Odontol Scand*. 1997; **55**(6): p. 344-9.
151. Wolfe, F., et al. The prevalence and characteristics of fibromyalgia in the general population. *Arthritis Rheum*. 1995; **38**(1): p. 19-28.
152. White, K.P., et al. The London Fibromyalgia Epidemiology Study: the prevalence of fibromyalgia syndrome in London, Ontario. *J Rheumatol*. 1999; **26**(7): p. 1570-6.
153. Migliorini, F., et al. BMI but not age and sex negatively impact on the outcome of pharmacotherapy in fibromyalgia: a systematic review. *Expert Rev Clin Pharmacol*. 2021; p. 1-10.
154. Ferreira, C.L., M.A. Silva, and C.M. Felicio. Signs and symptoms of temporomandibular disorders in women and men. *Codas*. 2016; **28**(1): p. 17-21.
155. Christidis, N., et al. Prevalence and treatment strategies regarding temporomandibular disorders in children and adolescents-a systematic review. *J Oral Rehabil*. 2019; **46**(3): p. 291-301.
156. Al-Jundi, M.A., et al. Meta-analysis of treatment need for temporomandibular disorders in adult nonpatients. *J Orofac Pain*. 2008; **22**(2): p. 97-107.
157. Mazzetto, M.O., et al. Severity of TMD related to age, sex and electromyographic analysis. *Braz Dent J*. 2014; **25**(1): p. 54-8.

158. Moldofsky, H., et al. The effect of zolpidem in patients with fibromyalgia: a dose ranging, double blind, placebo controlled, modified crossover study. *J Rheumatol.* 1996; **23**(3): p. 529-33.
159. Bennett, R.M., et al. A comparison of cyclobenzaprine and placebo in the management of fibrositis. A double-blind controlled study. *Arthritis Rheum.* 1988; **31**(12): p. 1535-42.
160. Su, N., et al. Associations of pain intensity and pain-related disability with psychological and socio-demographic factors in patients with temporomandibular disorders: a cross-sectional study at a specialised dental clinic. *J Oral Rehabil.* 2017; **44**(3): p. 187-196.
161. Pigozzi, L.B., et al. Quality of life in young and middle age adult temporomandibular disorders patients and asymptomatic subjects: a systematic review and meta-analysis. *Health Qual Life Outcomes.* 2021; **19**(1): p. 83.
162. Mello dos Santos, C., et al. Comparison of two assessment instruments of quality of life in older adults. *Rev Bras Epidemiol.* 2013; **16**(2): p. 328-37.
163. Ware, J.E., Jr. and B. Gandek. Overview of the SF-36 Health Survey and the International Quality of Life Assessment (IQOLA) Project. *J Clin Epidemiol.* 1998; **51**(11): p. 903-12.
164. Almoznino, G., et al. Oral health-related quality of life in patients with temporomandibular disorders. *J Oral Facial Pain Headache.* 2015; **29**(3): p. 231-41.
165. Zheng, J., M.C. Wong, and C.L. Lam. Key factors associated with oral health-related quality of life (OHRQOL) in Hong Kong Chinese adults with orofacial pain. *J Dent.* 2011; **39**(8): p. 564-71.
166. Schierz, O., et al. Comparison of perceived oral health in patients with temporomandibular disorders and dental anxiety using oral health-related quality of life profiles. *Qual Life Res.* 2008; **17**(6): p. 857-66.
167. Barros Vde, M., et al. The impact of orofacial pain on the quality of life of patients with temporomandibular disorder. *J Orofac Pain.* 2009; **23**(1): p. 28-37.
168. Blanco-Aguilera, A., et al. Application of an oral health-related quality of life questionnaire in primary care patients with orofacial pain and temporomandibular disorders. *Med Oral Patol Oral Cir Bucal.* 2014; **19**(2): p. e127-35.
169. Manfredini, D., et al. Psychosocial profiles of painful TMD patients. *J Oral Rehabil.* 2009; **36**(3): p. 193-8.

170. Kim, M.J., et al. Comparison between the SCL-90-R and MMPI in TMD patients with psychological problems. *Oral Dis.* 2012; **18**(2): p. 140-6.
171. Benoliel, R., et al. Subjective sleep quality in temporomandibular disorder patients and association with disease characteristics and oral health-related quality of life. *J Oral Facial Pain Headache.* 2017; **31**(4): p. 313-322.
172. Rener-Sitar, K., et al. Exploration of dimensionality and psychometric properties of the Pittsburgh Sleep Quality Index in cases with temporomandibular disorders. *Health Qual Life Outcomes.* 2014; **12**: p. 10.
173. Sommer, I., G. Lavigne, and D.A. Ettlin. Review of self-reported instruments that measure sleep dysfunction in patients suffering from temporomandibular disorders and/or orofacial pain. *Sleep Med.* 2015; **16**(1): p. 27-38.
174. Moldofsky, H.K. Disordered sleep in fibromyalgia and related myofascial facial pain conditions. *Dent Clin North Am.* 2001; **45**(4): p. 701-13.
175. Sayar, K., M. Arikian, and T. Yontem. Sleep quality in chronic pain patients. *Can J Psychiatry.* 2002; **47**(9): p. 844-8.
176. Consoli, G., et al. The impact of mood, anxiety, and sleep disorders on fibromyalgia. *Compr Psychiatry.* 2012; **53**(7): p. 962-7.
177. Sanders, A.E., et al. Associations of sleep disturbance, atopy, and other health measures with chronic overlapping pain conditions. *J Oral Facial Pain Headache.* 2020; **34**: p. s73-s84.
178. Segura-Jimenez, V., et al. Agreement between self-reported sleep patterns and actigraphy in fibromyalgia and healthy women. *Clin Exp Rheumatol.* 2015; **33**(1 Suppl 88): p. S58-67.
179. Landis, C.A., et al. Self-reported sleep quality and fatigue correlates with actigraphy in midlife women with fibromyalgia. *Nurs Res.* 2003; **52**(3): p. 140-7.
180. Bolukbas, Y. and B. Celik. Assessment of sleep quality in fibromyalgia syndrome and its effect on postural balance and functional status. *J Back Musculoskeletal Rehabil.* 2021; **34**(2): p. 235-242.
181. Akkaya, N., et al. Assessment of the relationship between postural stability and sleep quality in patients with fibromyalgia. *Clin Rheumatol.* 2013; **32**(3): p. 325-31.
182. Andrade, A., et al. The relationship between sleep quality and fibromyalgia symptoms. *J Health Psychol.* 2020; **25**(9): p. 1176-1186.

183. Wu, Y.L., et al. Sleep disturbances in fibromyalgia: A meta-analysis of case-control studies. *J Psychosom Res.* 2017; **96**: p. 89-97.
184. Aaron, L.A. and D. Buchwald. A review of the evidence for overlap among unexplained clinical conditions. *Ann Intern Med.* 2001; **134**(9 Pt 2): p. 868-81.
185. de Leeuw, R., J.L. Studts, and C.R. Carlson. Fatigue and fatigue-related symptoms in an orofacial pain population. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2005; **99**(2): p. 168-74.
186. Lavidor, M., A. Weller, and H. Babkoff. How sleep is related to fatigue. *Br J Health Psychol.* 2003; **8**(Pt 1): p. 95-105.
187. Pagano, T., et al. Assessment of anxiety and quality of life in fibromyalgia patients. *Sao Paulo Med J.* 2004; **122**(6): p. 252-8.
188. Gormsen, L., et al. Depression, anxiety, health-related quality of life and pain in patients with chronic fibromyalgia and neuropathic pain. *Eur J Pain.* 2010; **14**(2): p. 127 e1-8.
189. Ucar, M., et al. Health anxiety and depression in patients with fibromyalgia syndrome. *J Int Med Res.* 2015; **43**(5): p. 679-85.
190. Thieme, K., D.C. Turk, and H. Flor. Comorbid depression and anxiety in fibromyalgia syndrome: relationship to somatic and psychosocial variables. *Psychosom Med.* 2004; **66**(6): p. 837-44.
191. Arnold, L.M., et al. Relationships among pain and depressive and anxiety symptoms in clinical trials of pregabalin in fibromyalgia. *Psychosomatics.* 2010; **51**(6): p. 489-97.
192. dos Santos, E.B., et al. An evaluation of anxiety and depression symptoms in fibromyalgia. *Rev Esc Enferm USP.* 2012; **46**(3): p. 590-6.
193. Ramiro Fde, S., et al. Investigation of stress, anxiety and depression in women with fibromyalgia: a comparative study. *Rev Bras Reumatol.* 2014; **54**(1): p. 27-32.
194. Bartkowska, W., W. Samborski, and E. Mojs. Cognitive functions, emotions and personality in woman with fibromyalgia. *Anthropol Anz.* 2018; **75**(4): p. 271-277.
195. Isik-Ulusoy, S. Evaluation of affective temperament and anxiety-depression levels in fibromyalgia patients: a pilot study. *Braz J Psychiatry.* 2019; **41**(5): p. 428-432.
196. Fillingim, R.B., et al. Associations of psychologic factors with multiple chronic overlapping pain conditions. *J Oral Facial Pain Headache.* 2020; **34**: p. s85-s100.

197. Manfredini, D. and F. Lobbezoo. Relationship between bruxism and temporomandibular disorders: a systematic review of literature from 1998 to 2008. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2010; **109**(6): p. e26-50.
198. Jimenez-Silva, A., et al. Sleep and awake bruxism in adults and its relationship with temporomandibular disorders: a systematic review from 2003 to 2014. *Acta Odontol Scand.* 2017; **75**(1): p. 36-58.
199. Michelotti, A., et al. Oral parafunctions as risk factors for diagnostic TMD subgroups. *J Oral Rehabil.* 2010; **37**(3): p. 157-62.
200. Sierwald, I., et al. Association of temporomandibular disorder pain with awake and sleep bruxism in adults. *J Orofac Orthop.* 2015; **76**(4): p. 305-17.
201. Fujita, Y., et al. Oral habits of temporomandibular disorder patients with malocclusion. *Bull Tokyo Dent Coll.* 2003; **44**(4): p. 201-7.
202. Phuong, N.T.T., et al. Bruxism, related factors and oral health-related quality of life among Vietnamese medical students. *Int J Environ Res Public Health.* 2020; **17**(20).
203. Raphael, K.G., et al. Validity of self-reported sleep bruxism among myofascial temporomandibular disorder patients and controls. *J Oral Rehabil.* 2015; **42**(10): p. 751-8.
204. Khayat, N., et al. The prevalence of posterior crossbite, deep bite, and sleep or awake bruxism in temporomandibular disorder (TMD) patients compared to a non-TMD population: a retrospective study. *Cranio.* 2019; p. 1-7.
205. Storm, C. and A. Wanman. A two-year follow-up study of temporomandibular disorders in a female Sami population: validation of cases and controls as predicted by questionnaire. *Acta Odontol Scand.* 2007; **65**(6): p. 341-7.
206. Rossetti, L.M., et al. Association between sleep bruxism and temporomandibular disorders: a polysomnographic pilot study. *Cranio.* 2008; **26**(1): p. 16-24.
207. Rossetti, L.M., et al. Association between rhythmic masticatory muscle activity during sleep and masticatory myofascial pain: a polysomnographic study. *J Orofac Pain.* 2008; **22**(3): p. 190-200.
208. Raphael, K.G., et al. Sleep bruxism and myofascial temporomandibular disorders: a laboratory-based polysomnographic investigation. *J Am Dent Assoc.* 2012; **143**(11): p. 1223-31.

209. Haggman-Henrikson, B., et al. Pain and disability in the jaw and neck region following whiplash trauma. *J Dent Res.* 2016; **95**(10): p. 1155-60.
210. Pajediene, E., et al. Patterns of acute whiplash-associated disorder in the Lithuanian population after road traffic accidents. *J Rehabil Med.* 2015; **47**(1): p. 52-7.
211. Kasch, H., et al. Temporomandibular disorders after whiplash injury: a controlled, prospective study. *J Orofac Pain.* 2002; **16**(2): p. 118-28.
212. Lampa, E., et al. The course of orofacial pain and jaw disability after whiplash trauma: a 2-year prospective study. *Spine (Phila Pa 1976).* 2020; **45**(3): p. E140-E147.
213. Ferrari, R., H. Schrader, and D. Obelieniene. Prevalence of temporomandibular disorders associated with whiplash injury in Lithuania. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1999; **87**(6): p. 653-7.
214. Natu, V.P., et al. Temporomandibular disorder symptoms and their association with quality of life, emotional states and sleep quality in South-East Asian youths. *J Oral Rehabil.* 2018; **45**(10): p. 756-763.
215. Lei, J., et al. Temporomandibular disorders symptoms in Asian adolescents and their association with sleep quality and psychological distress. *Cranio.* 2016; **34**(4): p. 242-9.
216. Dreweck, F.D.S., et al. Association between painful temporomandibular disorders and sleep quality: a systematic review. *J Oral Rehabil.* 2020; **47**(8): p. 1041-1051.
217. Yap, A.U., et al. Temporomandibular disorder severity and diagnostic groups: their associations with sleep quality and impairments. *Sleep Med.* 2021; **80**: p. 218-225.
218. Rener-Sitar, K., et al. Sleep quality in temporomandibular disorder cases. *Sleep Med.* 2016; **25**: p. 105-112.
219. Schmitter, M., et al. Sleep-associated aspects of myofascial pain in the orofacial area among temporomandibular disorder patients and controls. *Sleep Med.* 2015; **16**(9): p. 1056-61.
220. Selaimen, C.M., et al. Sleep and depression as risk indicators for temporomandibular disorders in a cross-cultural perspective: a case-control study. *Int J Prosthodont.* 2006; **19**(2): p. 154-61.

221. Dubrovsky, B., et al. Depressive symptoms account for differences between self-reported versus polysomnographic assessment of sleep quality in women with myofascial TMD. *J Oral Rehabil.* 2017; **44**(12): p. 925-933.
222. Miettinen, O., S. Lahti, and K. Sipilä. Psychosocial aspects of temporomandibular disorders and oral health-related quality-of-life. *Acta Odontol Scand.* 2012; **70**(4): p. 331-6.
223. Balik, A., K. Peker, and M. Ozdemir-Karatas. Comparisons of measures that evaluate oral and general health quality of life in patients with temporomandibular disorder and chronic pain. *Cranio.* 2019; p. 1-11.
224. Reissmann, D.R., et al. Functional and psychosocial impact related to specific temporomandibular disorder diagnoses. *J Dent.* 2007; **35**(8): p. 643-50.
225. Ujin Yap, A., et al. Age-related differences in diagnostic categories, psychological states and oral health-related quality of life of adult temporomandibular disorder patients. *J Oral Rehabil.* 2021; **48**(4): p. 361-368.
226. Rener-Sitar, K., et al. Factors related to oral health related quality of life in TMD patients. *Coll Antropol.* 2013; **37**(2): p. 407-13.
227. Bertoli, E. and R. de Leeuw. Prevalence of suicidal ideation, depression, and anxiety in chronic temporomandibular disorder patients. *J Oral Facial Pain Headache.* 2016; **30**(4): p. 296-301.
228. Simoen, L., et al. Depression and anxiety levels in patients with temporomandibular disorders: comparison with the general population. *Clin Oral Investig.* 2020; **24**(11): p. 3939-3945.
229. Canales, G.T., et al. Distribution of depression, somatization and pain-related impairment in patients with chronic temporomandibular disorders. *J Appl Oral Sci.* 2019; **27**: p. e20180210.
230. Velly, A.M., M. Gornitsky, and P. Philippe. A case-control study of temporomandibular disorders: symptomatic disc displacement. *J Oral Rehabil.* 2002; **29**(5): p. 408-16.
231. Karacayli, U., et al. The effects of chronic pain on oral health related quality of life in patients with anterior disc displacement with reduction. *Community Dent Health.* 2011; **28**(3): p. 211-5.
232. Ohrbach, R., et al. Clinical findings and pain symptoms as potential risk factors for chronic TMD: descriptive data and empirically identified domains from the OPPERA case-control study. *J Pain.* 2011; **12**(11 Suppl): p. T27-45.

233. Baad-Hansen, L., et al. Comparison of clinical findings and psychosocial factors in patients with atypical odontalgia and temporomandibular disorders. *J Orofac Pain*. 2008; **22**(1): p. 7-14.
234. Riera-Punet, N., et al. Functional limitation of the masticatory system in patients with bulbar involvement in amyotrophic lateral sclerosis. *J Oral Rehabil*. 2018; **45**(3): p. 204-210.
235. Ohrbach, R., P. Larsson, and T. List. The jaw functional limitation scale: development, reliability, and validity of 8-item and 20-item versions. *J Orofac Pain*. 2008; **22**(3): p. 219-30.
236. Lee, Y.Y., et al. The risk of developing major depression among individuals with subthreshold depression: a systematic review and meta-analysis of longitudinal cohort studies. *Psychol Med*. 2019; **49**(1): p. 92-102.
237. Fetai, A., et al. To what extent are the characteristics of painful temporomandibular disorders predictors of self-reported limitations in jaw function? *Cranio*. 2020; p. 1-8.
238. Armijo-Olivo, S., et al. Head and cervical posture in patients with temporomandibular disorders. *J Orofac Pain*. 2011; **25**(3): p. 199-209.
239. Ohlmann, B., et al. Are there associations between sleep bruxism, chronic stress, and sleep quality? *J Dent*. 2018; **74**: p. 101-106.
240. Camara-Souza, M.B., O.M.C. de Figueredo, and R.C.M. Rodrigues Garcia. Association of sleep bruxism with oral health-related quality of life and sleep quality. *Clin Oral Investig*. 2019; **23**(1): p. 245-251.
241. Neu, D., et al. Effect of sleep bruxism duration on perceived sleep quality in middle-aged subjects. *Eur J Oral Sci*. 2018; **126**(5): p. 411-416.
242. Serra-Negra, J.M., et al. Sleep bruxism, awake bruxism and sleep quality among Brazilian dental students: a cross-sectional study. *Braz Dent J*. 2014; **25**(3): p. 241-7.
243. Smardz, J., et al. A polysomnographic study on the relationship between sleep bruxism intensity and sleep quality. *Cranio*. 2020; p. 1-6.
244. Yildirim, G., et al. Evaluation of the effects of bruxism on oral health-related quality of life in adults. *Cranio*. 2020; p. 1-8.
245. Tay, K.J., Y.A. Ujin, and P.F. Allen. Impact of sleep bruxism on oral health-related quality of life. *Int J Prosthodont*. 2020; **33**(3): p. 285-291.

246. Castelo, P.M., T.S. Barbosa, and M.B. Gaviao. Quality of life evaluation of children with sleep bruxism. *BMC Oral Health*. 2010; **10**: p. 16.
247. Antunes, L.A., et al. Childhood bruxism: related factors and impact on oral health-related quality of life. *Spec Care Dentist*. 2016; **36**(1): p. 7-12.
248. Jokovic, A., D. Locker, and G. Guyatt. What do children's global ratings of oral health and well-being measure? *Community Dent Oral Epidemiol*. 2005; **33**(3): p. 205-11.
249. Gungormus, Z. and K. Erciyas. Evaluation of the relationship between anxiety and depression and bruxism. *J Int Med Res*. 2009; **37**(2): p. 547-50.
250. Manfredini, D., et al. Psychic and occlusal factors in bruxers. *Aust Dent J*. 2004; **49**(2): p. 84-9.
251. Ohayon, M.M., K.K. Li, and C. Guilleminault. Risk factors for sleep bruxism in the general population. *Chest*. 2001; **119**(1): p. 53-61.
252. Kampe, T., et al. Personality traits in a group of subjects with long-standing bruxing behaviour. *J Oral Rehabil*. 1997; **24**(8): p. 588-93.
253. Kampe, T., et al. Reported symptoms and clinical findings in a group of subjects with longstanding bruxing behaviour. *J Oral Rehabil*. 1997; **24**(8): p. 581-7.
254. Goulart, A.C., et al. Anxiety, depression, and anger in bruxism: a cross-sectional study among adult attendees of a preventive center. *Psychiatry Res*. 2021; **299**: p. 113844.
255. Vanderas, A.P., et al. Urinary catecholamine levels and bruxism in children. *J Oral Rehabil*. 1999; **26**(2): p. 103-10.

7. APPENDIX

1. ETHICAL APPROVAL

**S.B. İSTANBUL MEDENİYET ÜNİVERSİTESİ GÖZTEPE EĞİTİM VE ARAŞTIRMA HASTANESİ
KLİNİK ARAŞTIRMALARI ETİK KURULU (2013-KAEK-64)
KARAR FORMU**

SAYI

Tarih: 21.01.2020

KONU: Etik Kurulu Kararı

ARAŞTIRMANIN AÇIK ADI	Fibromiyalji Hastalarında Temporomandibular Rahatsızlık Görülme Sıklığının Belirlenmesi ve Yaşam Kalitesi, Uyku Kalitesi ve Anksiyete ile İlişkinin Değerlendirilmesi
VARSA ARAŞTIRMANIN PROTOKOL KODU	

KLİNİK ARAŞTIRMALAR ETİK KURULU

ETİK KURULUN ÇALIŞMA ESASI	İlaç ve Biyolojik Ürünlerin Klinik Araştırmaları Hakkında Yönetmelik, İyi Klinik Uygulamaları Kılavuzu
BAŞKANIN UNVANI / ADI / SOYADI:	

Unvanı/Adı/Soyadı	Uzmanlık Alanı	Kurumu	Cinsiyet	Araştırma ile İlişki	Katılım *	İmza
Doç. Dr. Şükrü Sadık ÖNER	Tıbbi Farmakoloji	S.B. İstanbul Medeniyet Üniversitesi Göztepe Eğitim ve Araştırma Hastanesi	E ☒ K ☐	E ☐ H ☒	E ☒ H ☐	
Prof. Dr. Aytekin OĞUZ	İç Hastalıkları Anabilim Dalı	S.B. İstanbul Medeniyet Üniversitesi Göztepe Eğitim ve Araştırma Hastanesi	E ☒ K ☐	E ☐ H ☒	E ☐ H ☒	
Prof. Dr. Işıl MARAL	Halk Sağlığı Anabilim Dalı	S.B. İstanbul Medeniyet Üniversitesi Göztepe Eğitim ve Araştırma Hastanesi	E ☐ K ☒	E ☐ H ☒	E ☒ H ☐	
Prof. Dr. Asif Yıldırım	Üroloji	S.B. İstanbul Medeniyet Üniversitesi Göztepe Eğitim ve Araştırma Hastanesi	E ☒ K ☐	E ☐ H ☒	E ☒ H ☐	
Prof. Dr. Süleyman Daşdağ	Biyofizik	S.B. İstanbul Medeniyet Üniversitesi Göztepe Eğitim ve Araştırma Hastanesi	E ☒ K ☐	E ☐ H ☒	E ☐ H ☒	
Prof. Dr. Derya Büyükkayhan	Çocuk Sağlığı ve Hastalıkları Anabilim Dalı	T.C. Sağlık Bakanlığı Zeynep Kamil Kadın ve Çocuk Hastalıkları Eğitim ve Araştırma Hastanesi	E ☐ K ☒	E ☐ H ☒	E ☒ H ☐	
Doç. Dr. Asiye KANBAY	Göğüs Hastalıkları Anabilim Dalı	S.B. İstanbul Medeniyet Üniversitesi Göztepe Eğitim ve Araştırma Hastanesi	E ☐ K ☒	E ☐ H ☒	E ☒ H ☐	
Doç. Dr. Sıdika Şeyma ÖZKANLI	Tıbbi Patoloji	S.B. İstanbul Medeniyet Üniversitesi Göztepe Eğitim ve Araştırma Hastanesi	E ☐ K ☒	E ☐ H ☒	E ☒ H ☐	
Dr. Öğr. Üyesi Hacer Hicran Mutlu	Aile Hekimliği	S.B. İstanbul Medeniyet Üniversitesi Göztepe Eğitim ve Araştırma Hastanesi	E ☐ K ☒	E ☐ H ☒	E ☒ H ☐	
Uzm. Dr. Ergül Demirçivi Bôr	Kadın Hastalıkları ve Doğum	S.B. İstanbul Medeniyet Üniversitesi Göztepe Eğitim ve Araştırma Hastanesi	E ☐ K ☒	E ☐ H ☒	E ☐ H ☒	
Avukat Mahmut ÇELİK	Avukat	Çelik Hukuk Bürosu	E ☒ K ☐	E ☐ H ☒	E ☐ H ☒	
Saliha Şahin	İşçi		E ☐ K ☒	E ☐ H ☒	E ☒ H ☐	

*:Toplantıda Bulunma

Karar: ☒ Onaylandı ☐ Reddedildi

T.C. S.B. İSTANBUL MEDENİYET ÜNİVERSİTESİ
GÖZTEPE E.A.H.
Sakrî Sadık ÖNER
Soyadı, Dr. Şükrü Sadık ÖNER
Çocuk Hastalıkları Anabilim Dalı

2. INFORMED CONSENT

BİLGİLENDİRİLMİŞ GÖNÜLLÜ OLUR FORMU

“Fibromiyalji hastalarında temporomandibular rahatsızlık görülme sıklığının belirlenmesi ve yaşam kalitesi, uyku kalitesi ve anksiyete ile ilişkisinin değerlendirilmesi” adlı bu çalışma İstanbul Medeniyet Üniversitesi Göztepe Eğitim ve Araştırma Hastanesi ve Yeditepe Üniversitesi Dış Hekimliği Fakültesi tarafından yürütülmektedir. Araştırmanın amacı yaygın bir rahatsızlık olan temporomandibular eklem (TME) problemlerinin fibromiyalji rahatsızlığı olan bireylerde görülme sıklığını saptamak ve temporomandibular rahatsızlık bulgu ve belirtilerinin psikolojik etkenlerle ilişkisini incelemektir.

Çalışmaya katılım tamamen **gönüllülük** esasına dayanmaktadır. *Katılmak isteyip istemediğinize karar vermeden önce araştırmanın neden ve nasıl yapılacağını, bilgilerinizin nasıl kullanılacağını ve çalışmanın neleri içerdiğini anlamanız önemlidir. Lütfen aşağıdaki bilgileri dikkatlice okumak için zaman ayırınız. Eğer anlayamadığınız ve sizin için açık olmayan şeyler varsa ya da daha fazla bilgi almak isterseniz lütfen bize sorunuz. Bu formu okuyup onaylamanız, araştırmaya katılmayı kabul ettiğiniz anlamına gelecektir.* Ancak, çalışmaya **katılmama** veya katıldıktan sonra herhangi bir anda çalışmayı **bırakma** hakkına sahipsiniz.

Göztepe Eğitim ve Araştırma Hastanesi Fiziksel Tıp ve Rehabilitasyon bölümüne gelen fibromiyalji hastaları çene eklemi hastalıkları alanında uzmanlaşmış bir diş hekimi tarafından muayene edilecektir. Çalışma kriterlerine uyan bireylerin demografik bilgileri, Temporomandibular Eklem Rahatsızlık (TMR) bulgularını, ağrı semptomlarını, uyku kalitelerini ve yaşam kalitelerini (PSQI, SCL-90, OHIP) içeren dört anket doldurması istenecektir. Yaklaşık 20 dakikanızı ayırarak bu sorulara yanıt vermeniz çalışmanın devamlılığı ve doğru katılımcılarla ılerlenmesi açısından önemlidir. Anket sonuçlarına ve diş hekimi muayenesine göre temporomandibular rahatsızlık tanısı konacaktır. Çalışma yalnız **tanı amaçlı** yapılacak olup, **tedaviyi içermemektedir**. Muayenede vücuda zararlı herhangi bir işlem uygulanmayacaktır. Muayene için hastalardan herhangi bir ücret talep edilmeyecektir.

Araştırmada yapılan değerlendirmelerin sonuçları yalnızca araştırma kapsamındaki çalışmalarda kullanılacaktır. Araştırma bir kongrede ya da dergide yayın olarak kabul edilse bile gönüllülerin kimliğini ortaya çıkarabilecek kayıtlar gizli tutulacaktır. Bilgilendirilmiş gönüllü olur formunun imzalaması halinde gönüllü veya kanuni temsilcisi, araştırmayı yürüten, araştırma sürecini izleyen kişilerin ve etik kurul gibi kurumların bilgileri gizli tutmak

kayıtlı ile gönüllünün sağlık kayıtlarına doğrudan erişim sağlanmasına izin vermiş olacaktır. Araştırma sürecinde gönüllünün katılımcı olarak devam etme isteğini etkileyebilecek her yeni bilgi gönüllü ile paylaşılacaktır.

Koordinatör ve sorumlu araştırmacı: Doç. Dr. Yasemin Yumuşaklı

Yardımcı Araştırmacı: Dr. Öğretim Üyesi Burcu Bal Tel. No: 02163636044

Yardımcı Araştırmacı: Dt. Gözde Sarak Tel. No: 05309218138

Bilgilendirilmiş gönüllü olur formundaki tüm açıklamaları okudum. Bana yukarıda konusu ve amacı belirtilen araştırma ile ilgili yazılı ve sözlü açıklama aşağıda adı belirtilen hekim tarafından yapıldı. Araştırmaya gönüllü olarak katıldığımı, istediğim zaman gerçekçi veya gerekçesiz olarak araştırmadan ayrılabilceğimi biliyorum. **"Fibromiyalji hastalarında temporomandibular rahatsızlık görülme sıklığının belirlenmesi ve yaşam kalitesi, uyku kalitesi ve anksiyete ile ilişkisinin değerlendirilmesi"** adlı incelemeyi içeren bu çalışmaya hiçbir baskı ve zorlama olmaksızın kendi irademle katılmayı kabul ediyorum.

Gönüllünün Adı/ Soyadı / İmzası / Tarih:

Açıklamaları Yapan Kişinin Adı / Soyadı / İmzası / Tarih: