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ORIGINAL ARTICLE

REVIEW

CASE REPORT

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Original Article

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Comparison of the effects of dry needling technique and combination of different manual therapy techniques on electrophysiological and psychological status in patients with myofascial pain syndrome - randomized, controlled, single blind, a pilot study

Sercan Ateş[1], Seyit Çitaker[2], Ayşegül Demirci Çoban[3], Arif Tolga Sönmez[4], Yasemin Öztürk[5]

[1]Vocational School of Health Services Physiotherapy Program, Atılım University, Ankara, Turkey
[2]Department of Physiotherapy and Rehabilitation, Faculty of Health Sciences, Gazi University, Ankara
[3]Physical Medicine and Rehabilitation, Vocational School of Health Services Physiotherapy Program, Atılım University, Ankara, Turkey
[4]Faculty of Medicine, Atılım University, Ankara, Turkey
[5]General Healt Insurance General Directorate, Social Security Institution of Turkey, Ankara, Turkey

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Corresponding Author: Sercan Ateş, sercanftr@gmail.com

ABSTRACT

Aims: Our aim was to compare the electrophysiological and psychometric properties of the effects of the dry needling technique, graston technique, and muscle energy technique in treating myofascial pain syndrome.

Methods: The study included 21 patients diagnosed with myofascial pain syndrome (MPS) and an active trigger point in the upper trapezius muscle. The patients were randomly divided into three groups of 7 each. The first group received only exercise and dry needling technique (DNT), the second group received graston technique (GT), muscle energy technique (MET) and exercise, and the third group received only exercise. Evaluations were conducted before and after treatment. The trigger point detected with an algometer in the upper trapezius muscle was evaluated with needle electromyography (EMG). The study used the Beck Depression Inventory (BDI) to determine its effects on psychological state.

Results: The change in EMG variable over time was significant in each treatment group ($p<0.05$). The improvement in EMG change was more pronounced in the DNT group compared to the other groups. There was also a significant decrease in BDI scores before and after treatment ($p<0.05$). Pairwise comparison analysis revealed an improvement in the BDI scores of all groups, except the control (exercise) group, after treatment, when compared to pre-treatment.

Conclusion: During the trigger point needle EMG examination of all subjects in the study, we detected spontaneous muscle activity (SMA) in addition to entry activity. After treatment, we observed improvement in SMA. These results suggest that the applied techniques are effective in treating trigger points.The effectiveness of the applied techniques can be evaluated in terms of creating alternatives.

Keywords: Dry needling, manual therapy, graston technique, muscle energy technique, myofascial pain syndrome

INTRODUCTION

Myofascial pain syndrome (MPS) is a non-inflammatory syndrome that affects a specific area of the body, characterized by taut bands on skeletal muscle fibers and fascia. It is very common among musculoskeletal system problems. Pain and muscle spasm develop due to myofascial trigger points (MTP) on taut bands. Movement restriction may occur in the joints related to MTP and regional autonomic symptoms may be seen.[1,2] MTPs are palpable and may produce tense, aching, tender, and referred pain on palpation. Passive MTPs do not produce referred pain. They can become active through physical activity or coercion.[3] MTPs can be found in many different muscle groups.It is frequently seen in the postural muscles and upper trapezius muscle and the muscles surrounding the pelvis.[4,5]

Although methods such as sonoelastography, thermography, doppler imaging, magnetic resonance elastography have been used in diagnostic imaging in recent years, there is no technique that specifically defines MTPs radiologically.[6-9]

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Electrophysiologically, changes such as spontaneous increase in electrical activity and delayed relaxation have been shown in MTPs.[10,11] Methods such as biochemical laboratory tests are not imaging, pressure algometer value and EMG continue to be investigated to obtain information about MTPs.[6,10,11] Many invasive and non-invasive treatment methods are used for trigger points in MPS. Dry needling technique (DNT) is an invasive and effective method.It is applied directly on tension bands and MTPs with acupuncture needles of various sizes.[12] Graston technique (GT) and muscle energy technique (MET) are noninvasive manual therapy techniques.[13,14] It is applied with tools made of stainless steel in various sizes designed according to the areas where GT is applied.The purpose of GT is to support healing with microtrauma in soft tissue and to increase tissue elasticity.[13] MET was developed by osteopaths.[14] The practice includes movements that require the active participation of the patient.[15] These movements primarily target the soft tissue and increase the range of motion of the joint. Also known as active muscle relaxation technique.[14,16]

The aim of this study is to compare the electrophysiological and psychometric properties of the effects of dry needling technique and graston and muscle energy techniques in the treatment of myofascial pain syndrome.

METHODS

Ethical Process

The study was carried out with the permission of Ankara Medicana International Hospital Ethics Committee (Date: 29.03.2021, Decision No: 12). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Partcipants

The study included 21 patients who applied to Medicana International Ankara Hospital Physical Therapy and Rehabilitation Polyclinic between August 2021 and June 2022, diagnosed and signed the consent form. The diagnosis of MPS was made based on the diagnostic criteria described by Travell and Simons.[17] Patients with neck and/or shoulder and/or back pain who had at least one active trigger point detected by algometry and palpation in the upper trapezius muscle were included in the study. Those with cervical radiculopathy, cancer, fracture, inflammatory disease diagnosis and surgery history in the last 6 months, and those who were under pregnancy follow-up were not included in the study.

Study Desing and Randomization

Participants were randomly(By cart selection) divided into 3 groups and the patients in the first group were given exercise+DNT, the patients in the second group exercise+MET+GT, and the patients in the third group only exercised. The treatments were applied as 3 sessions in two weeks. The exercises were given as a home program (Three times for every day). Depression level before and after the treatment was evaluated with Beck Depression Inventory (BDI) and electrophysiological activity of detected MTPs with needle electromyography (EMG). BDI is a questionnaire that expresses the level of depression, consists of 21 questions in total and has 4 items in each question (scored between 0 and 3 points). High scores indicate the severity of depression.[18] The patients filled the scale, which was organized in the form of a questionnaire, under the supervision of a physiotherapist by choosing the expression they felt closest to them. Needle EMG evaluations of MTPs were made by a blinded neurologist. The motor unit potential (MUP) amplitude value in the needle EMG examination we performed was recorded as a unit value in the report. In the upper trapezius muscle trigger point examination, SMA was observed except the entry activity. Measurements from the same trigger point before and after treatment were compared.

Statistical Analysis

Statistical analyzes were performed using the SPSS (IBM SPSS Statistics 23) package program. Descriptive statistics such as mean and standard deviation were given for numerical variables. Percentage values and frequency tables were given for categorical variables.Whether the numerical measurements to be taken from the patients changed over time were similar between the 3 groups, was analyzed by analysis of variance in repeated measurements. In order to determine the effects of the intervention on the measured variables, a mixed design ANOVA was applied with time (pre-treatment and post-treatment) as an within-subject factor (pre-treatment and after-treatment) and between-groups (exercise+DNT, exercise+MET+GT, exercise) as an inter-subject (group) factor. The Bonferroni test was used for pairwise (post-hoc) comparisons in case of significant factors or interactions. Statistical significance level was accepted as $p < 0.05$. In this study, the efficacy of 3 different treatment methods (including the control group) in patients diagnosed with MPS was investigated under exercise control. It was examined whether the EMG test results of the patients and the results to be obtained from BDI differ according to time and treatment type. As the primary outcome, the EMG value, which determines the pain intensity of the patients at the trigger points, was discussed.

RESULTS

The study recorded age, gender, height, weight, body mass index, smoking status, sleep problems, and average daily sleep time data of the included patients. Table 1, 2 presents demographic characteristics of patients with MPS.

Table 1. Demographic Characteristics of the Patients Participating in the Study -1

Variable		Number	Rate
Gender	Female	16	76.2%
	Male	5	23.8%
Smoking Status	No	13	61.9%
	Yes	8	38.1%
BMI*	18.5 and under Underweight	4	19.0%
	18.5 - 24.9 Normal Weight	14	66.7%
	25-29.9 Overweight	3	14.3%
Sleeping Problem	No	15	71.4%
	Yes	6	28.6%

*Body Mass Index (BMI)

Table2. Demographic Characteristics of the Patients Participating in the Study -2

Variable	Minimum	Maximum	Average	Standard deviation
Age	21	50	31.8	10.1
Weight (kg)	52	85	66.4	8.5
Height (cm)	157	184	170.1	7.3
BMI (kg/m2)*	23	17	28.0	2.6
Sleeping time (hours)	5	10	6.9	1.2

*Body Mass Index (BMI)

The average age of the patients between the ages of 21-50 who participated in the study was 31.8 years.The sleep duration of the patients varies between 5-10 hours per day, with an average sleep duration of 6.9 hours (**Table 2**). The mean EMG MUP amplitude values in all treatment groups were 5.3 units before treatment and 1.9 units after treatment. In each treatment group, a decrease was observed in the mean EMG value after treatment compared to pretreatment (**Figure 1**).

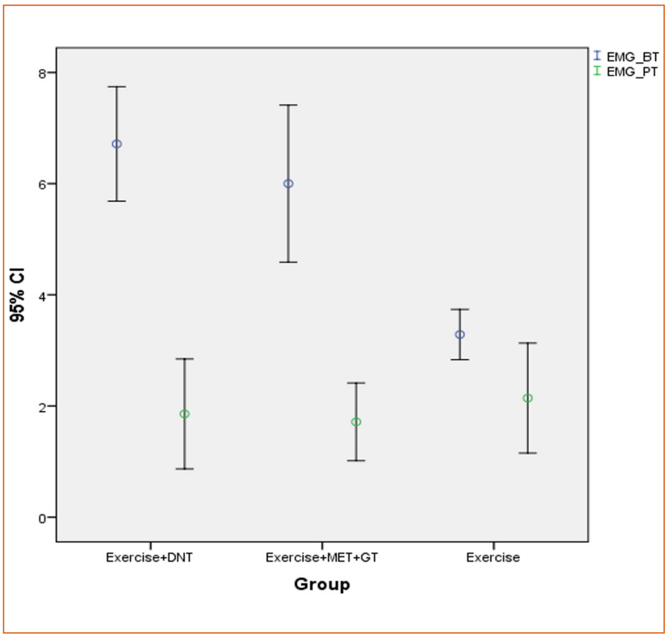

Figure 1. Exercise+Dry Needling, Exercise+Muscle Energy+Graston, and Exercise Effect on EMG (error bar graph with 95% Confidence Interval)

Electromyography Before Treatment (EMG BT)
Electromyography Post-Treatment (EMG PT)

According to the results of the ANOVA analysis in repeated measurements, the interaction effect between the groups was found to be significant for the EMG variable on a time basis ($p<0.05$). In other words, the change in EMG values over time differs according to the treatment methods (**Table 3**).

Table 3. Average EMG Values Before and After Treatment According to Treatment Groups

Group	Before Treatment Average±Standard Deviation	After Treatment Average±Standard Deviation	p
Exercise+DNT (n=7)	6.7±1.1	1.9±1.1	<0.05
Exercise+DNT+GT (n=7)	6.0±1.5	1.7±0.8	
Exercise (n=7)	3.3±0.5	1.9±0.9	

It was determined that a curative effect occurred before and after treatment in each treatment group evaluated with EMG values. According to the pairwise comparison analysis, improvement was detected in each group after treatment when compared with pre-treatment (**Table 4**). The difference between before and after treatment in all groups was statistically significant ($p<0.05$). When the pairwise comparison results are examined, it is seen that the greatest improvement in terms of EMG values is in the group treated with exercise and DNT.

While the mean BDI values were 14.1 units before the treatment in all treatment groups, it was 9.9 units after the treatment. In each treatment group, a decrease was observed in the mean BDI value compared to before (**Figure 2**).

Table 4. Paired Comparisons of Before and Post-Treatment EMG Values According to Treatment Groups

Group			Average Difference	Standard error	p	Confidence Interval Lower Limit	Confidence Interval Upper Limit
Exercise +DNT	BT	PT	4.8	0.46	0.00	3.88	5.83
	PT	BT	-4.8	0.46	0.00	-5.83	-3.88
Exercise +MET+GT	BT	PT	4.2	0.46	0.00	3.31	5.26
	PT	BT	-4.2	0.46	0.00	-5.26	-3.31
Exercise	BT	PT	1.1	0.46	0.02	0.17	2.12
	PT	BT	-1.1	0.46	0.02	-2.12	-0.17

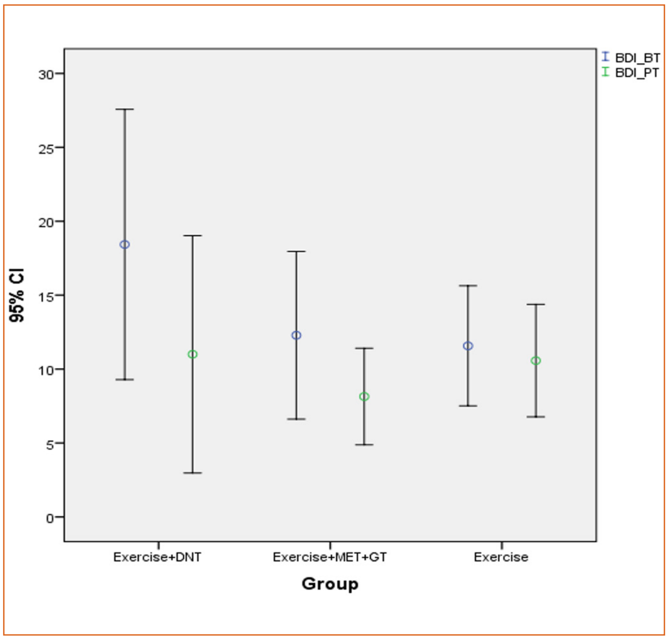

Figure 2. Exercise+Dry Needling, Exercise+Muscle Energy+Graston, and Exercise Effect on BDI (error bar graph with 95% Confidence Interval)

Beck Depression Inventory Before Treatment (BDI_BT)
Beck Depression Inventory Post Treatment (BDI_PT)

According to the results of the ANOVA analysis in repeated measurements, the interaction effect between the groups was found to be significant for the BDI variable on a time basis ($p<0.05$). In other words, the change in BDI values over time differs according to treatment methods (**Table 5**).

Table 5. Average Beck Depression Inventory Values Before and After Treatment

Group	Before Treatment Average±Standard Deviation	After Treatment Average±Standard Deviation	p
Exercise+DNT (n=7)	18.4±9.9	11±8.7	<0.05
Exercise+DNT+GT (n=7)	12.3±6.1	8.1±3.5	
Exercise (n=7)	11.6±4.4	10.6±4.1	

It was determined that there was a curative effect before and after the treatment in the exercise+DNT applied treatment group evaluated with BDI values and in the exercise+MET+GT treatment group.

The difference between before and after treatment in these groups is statistically significant ($p<0.05$). According to the pairwise comparison analysis, improvement was determined after the treatment in the exercise+DNT applied group and the exercise+MET+GT group compared with the pre-treatment. It is observed that the greatest improvement in BDI values was in the group treated with exercise and DNT (**Table 6**).

Table 6. Paired Comparisons of Beck Depression Inventory Values Before and Post-Treatment According to Treatment Groups

Group			Average Difference	Standard Error	p	Confidence Interval	
						Lower Limit	Upper Limit
Exercise +DNT	BT	PT	7.4	1.630	0.00	4.0	10.9
	PT	BT	-7.4	1.630	0.00	-10.9	-4.0
Exercise +MET+GT	BT	PT	4.1	1.630	0.02	0.7	7.6
	PT	BT	-4.1	1.630	0.02	-7.6	-0.7
Exercise	BT	PT	1.0	1.630	0.55	-2.4	4.4
	PT	BT	1.0	1.630	0.55	-4.4	2.4

DISCUSSION

In our study, we aimed to compare the effects of invasive and non-invasive treatments. Our results showed that DNT, MET and GT applications were more effective than exercise application.

In MPS, pain is sometimes reflected in areas that are trigger points, usually in areas far from trigger points. It can cause motor dysfunction, fatigue and autonomic disorders.[1,3]

It is one of the most common causes of pain encountered in the clinic. It is among the most common diseases among the musculoskeletal system disorders involving the neckand waist region.[19] Treatment in MPS is usually directed towards pain management and MTP. Inactivation of trigger points and loosening of taut bands is the first step in coping with pain.[2] Many treatment approaches, including invasive and non-invasive, are used in the treatment of MPS.[20] Among the invasive methods, dry needling, acupuncture, steroid, local anesthetic, botulinum toxin and prp injections; pharmacological drug therapy, physical therapy agents, exercise applications, postural and ergonomic supports, kinesiology taping and manual therapy techniques are preferred among noninvasive methods. There are many studies on these methods in the literature.[21-25] Exercise is a frequently preferred method in the treatment of MPS.In MPS, especially light stretching and posture exercises are preferred to resistance strengthening exercises. Because, in the presence of an active trigger point, mechanical stress on the muscle may increase symptoms.[21] Although there are many different studies showing the efficacy of DNT in MPS, recent systematic reviews have low and moderate levels of evidence. There is a need for more randomized controlled studies with specific evaluations for MTPs.[26,27] MET is an effective method for the treatment of chronic and acute neck pain. Used to reduce pain, increase flexibility and range of motion.[28] MET has been found to be effective in comparative studies on MTPs in the upper trapezius muscle.It produces muscle relaxation with postisometric relaxation and respiratory inhibition. Since it works with a mild active contraction during the application, it reduces the pain and allows the stretched band to relax.[29-31.] The number of studies in the literature on GT has been increasing in recent years. There are studies showing its effectiveness on pain, muscle strength and range of motion.[32,33] Studies comparing DNT with MET and GT and studies combining DNT with other applications have been published.[27,30,34] A combination of MET in practice has been recommended in some systematic reviews.[28] In recent years, GT and MET have been used together in clinical applications. There is no study in the literature comparing the combination of DNT with GT and MET. Many different imaging methods are used in the evaluation of MTP.[1,6-11] Although subjective evaluation methods were mostly preferred in comparative studies of DNT with different treatment approaches in the literature, objective studies evaluating its direct effectiveness were also conducted.[12,35,36] In our study, we preferred specific needle EMG evaluation over MTP.[10,37] We planned to compare DNT with the combination of MET and GT. We found that both DNT and GT and MET combination groups were effective on MTP EMG activation and depression symptoms compared to the control group. We think that studies with a larger sample size will be more effective in demonstrating the superiority between groups.

CONCLUSION

In the treatment of MPS, DNT, GT and MET showed a healing effect in upper trapezius muscle active MTPs. Clinicians may not always prefer invasive methods in practice when there are some obstacles such as patient fears. For such reasons, comparative studies are important in terms of creating an alternative. This study may play a role in clinicians' choice of invasive and non-invasive treatment modalities in patient management.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of Ankara Medicana International Hospital Ethics Committee (Date: 29.03.2021, Decision No: 12).

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Fernández-de-Las-Peñas C, Dommerholt J. International Consensus on Diagnostic Criteria and Clinical Considerations of Myofascial Trigger Points: A Delphi Study. *Pain Med.* 2018;19(1):142-150. doi:10.1093/pm/pnx207
2. Barbero M, Schneebeli A, Koetsier E, Maino P. Myofascial pain syndrome and trigger points: evaluation and treatment in patients with musculoskeletal pain. *Curr Opin Support Palliat Care.* 2019;13(3):270-276. doi:10.1097/SPC.0000000000000445
3. Bron C, Franssen J, Wensing M, Oostendorp RA. Interrater reliability of palpation of myofascial trigger points in three shoulder muscles. *J Man Manip Ther.* 2007;15(4):203-215. doi:10.1179/106698107790819477
4. Simons DG. Understanding effective treatments of myofascial trigger points. *J Bodyw Mov Ther.* 2002;6:81–88. doi: 10.1054/jbmt.2002.0271.
5. Meleger AL, Krivickas LS. Neck and back pain: musculoskeletal disorders. *Neurol Clin.* 2007;25(2):419-438. doi:10.1016/j.ncl.2007.01.006
6. Ariji Y, Kimura Y, Gotoh M, Sakuma S, Zhao YP, Ariji E. Blood flow in and around the masseter muscle: normal and pathologic features demonstrated by color Doppler sonography. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2001;91(4):472-482. doi:10.1067/moe.2001.111760
7. Cojocaru MC, Cojocaru IM, Voiculescu VM, Cojan-Carlea NA, Dumitru VL, Berteanu M. Trigger points--ultrasound and thermal findings. *J Med Life.* 2015;8(3):315-318.
8. Crema MD, Yamada AF, Guermazi A, Roemer FW, Skaf AY. Imaging techniques for muscle injury in sports medicine and clinical relevance. *Curr Rev Musculoskelet Med.* 2015;8(2):154-161. doi:10.1007/s12178-015-9260-4

9. Evans V, Behr M, Gangwar A, Noseworthy MD, Kumbhare D. Potential Role of MRI Imaging for Myofascial Pain: A Scoping Review for the Clinicians and Theoretical Considerations. *J Pain Res.* 2021;14:1505-1514. Published 2021 May 27. doi:10.2147/JPR.S302683
10. Hubbard DR, Berkoff GM. Myofascial trigger points show spontaneous needle EMG activity. *Spine (Phila Pa 1976).* 1993;18(13):1803-1807. doi:10.1097/00007632-199310000-00015
11. Simons DG, Hong CZ, Simons LS. Prevalence of spontaneous electrical activity at trigger points and at control sites in rabbit skeletal muscle. *J Musculoskel Pain.* 1995;3(1):35-48.
12. De Meulemeester KE, Castelein B, Coppieters I, Barbe T, Cools A, Cagnie B. Comparing Trigger Point Dry Needling and Manual Pressure Technique for the Management of Myofascial Neck/Shoulder Pain: A Randomized Clinical Trial. *J Manipulative Physiol Ther.* 2017;40(1):11-20. doi:10.1016/j.jmpt.2016.10.008
13. Baker RT, Nasypany A, Seegmiller JG, et al. Instrumentassisted soft tissue mobilization treatment for tissue extensibility dysfunction. *Int J Athl Ther Training.* 2013;18(5):16-21. doi:10.1123/ijatt.18.5.16
14. Ballantyne F, Fryer G, McLaunghlin P. The effect of muscle energy technique on hamstring extensibility: the mechanism of altered flexibility. *J Osteopathic Med.* 2003; 6 (2), 59-63.
15. Chila A, Foundation of Osteopathic Medicine. *Medicine;* 2010: 682-695.
16. Chaitlow L, Liebenson C (Ed) In: Muscle Energy Technicques. 2001: 2nd Edition, Donald R Murphy, London. 95-106.
17. Simons DG, Travell JG, Simons LS. Myofascial pain and dysfunction:the trigger point manual. 2nd ed. Baltimore: Williams & Wilkins;1999.
18. Hıslı N. The reliability and validity study of the beck depression inventory in a Turkish sample. *Turk J Psychiatry.* 1998;6:118-22
19. Chiarotto A, Clijsen R, Fernandez-de-Las-Penas C, Barbero M. Prevalence of Myofascial Trigger Points in Spinal Disorders: A Systematic Review and Meta-Analysis. *Arch Phys Med Rehabil.* 2016;97(2):316-337. doi:10.1016/j.apmr.2015.09.021
20. Gül K, Onal SA. Miyofasiyal ağri sendromlu hastalarin tedavisinde non-invazif ve invazif tekniklerin karşilaştirilmasi [Comparison of non-invasive and invasive techniques in the treatment of patients with myofascial pain syndrome]. *Agri.* 2009;21(3):104-112.
21. Mata Diz JB, de Souza JR, Leopoldino AA, Oliveira VC. Exercise, especially combined stretching and strengthening exercise, reduces myofascial pain: a systematic review. *J Physiother.* 2017;63(1):17-22. doi:10.1016/j.jphys.2016.11.008
22. Hou CR, Tsai LC, Cheng KF, Chung KC, Hong CZ. Immediate effects of various physical therapeutic modalities on cervical myofascial pain and trigger-point sensitivity. *Arch Phys Med Rehabil.* 2002;83(10):1406-1414. doi:10.1053/apmr.2002.34834
23. Galasso A, Urits I, An D, et al. A Comprehensive Review of the Treatment and Management of Myofascial Pain Syndrome. *Curr Pain Headache Rep.* 2020;24(8):43. doi:10.1007/s11916-020-00877-5
24. Urits I, Charipova K, Gress K, et al. Treatment and management of myofascial pain syndrome. *Best Pract Res Clin Anaesthesiol.* 2020;34(3):427-448. doi:10.1016/j.bpa.2020.08.003
25. Ay S, Konak HE, Evcik D, Kibar S. The effectiveness of Kinesio Taping on pain and disability in cervical myofascial pain syndrome. *Rev Bras Reumatol Engl Ed.* 2017;57(2):93-99. doi:10.1016/j.rbre.2016.03.012
26. Navarro-Santana MJ, Sanchez-Infante J, Fernández-de-Las-Peñas C, Cleland JA, Martín-Casas P, Plaza-Manzano G. Effectiveness of Dry Needling for Myofascial Trigger Points Associated with Neck Pain Symptoms: An Updated Systematic Review and Meta-Analysis. *J Clin Med.* 2020;9(10):3300. Published 2020 Oct 14. doi:10.3390/jcm9103300
27. Lew J, Kim J, Nair P. Comparison of dry needling and trigger point manual therapy in patients with neck and upper back myofascial pain syndrome: a systematic review and meta-analysis. *J Man Manip Ther.* 2021;29(3):136-146. doi:10.1080/10669817.2020.1822618
28. Sbardella S, La Russa C, Bernetti A, et al. Muscle Energy Technique in the Rehabilitative Treatment for Acute and Chronic Non-Specific Neck Pain: A Systematic Review. *Healthcare (Basel).* 2021;9(6):746. Published 2021 Jun 17. doi:10.3390/healthcare9060746
29. Kashyap R, Iqbal A, Alghadir AH. Controlled intervention to compare the efficacies of manual pressure release and the muscle energy technique for treating mechanical neck pain due to upper trapezius trigger points. *J Pain Res.* 2018;11:3151-3160. doi:10.2147/JPR.S172711
30. Basak T, Pal TK, Sasi MM, Agarwal S. A Comparative study on the efficacy of ischaemic compression and dry needling with muscle energy technique in patients with upper trapezius myofascial trigger points. *Int J Health Sci Res.* 2018;8:74–81.
31. Shah NA, Shah N. Comparison of two treatment techniques: Muscle energy technique and Ischemic compression on upper trapezius trigger point in subjects with non- specific neck pain. *Int J Ther Rehabil.* 2015;4:260–264. doi: 10.5455/ijtrr.000000100.
32. Aggarwal A, Saxena K, Palekar TJ, Rathi M. Instrument assisted soft tissue mobilization in adhesive capsulitis: A randomized clinical trial. *J Bodyw Mov Ther.* 2021;26:435-442. doi:10.1016/j.jbmt.2020.12.039
33. Lee J, Young A, Erb NJ, Herzog VW. Acute and Residual Effects of IASTM and Roller Massage Stick on Hamstring Range of Motion. *J Allied Health.* 2020;49(1):e51-e55.
34. Ahmadpour Emshi Z, Okhovatian F, Mohammadi Kojidi M, Akbarzadeh Baghban A, Azimi H. Comparison of the effects of instrument assisted soft tissue mobilization and dry needling on active myofascial trigger points of upper trapezius muscle. *Med J Islam Repub Iran.* 2021;35:59. Published 2021 May 8. doi:10.47176/mjiri.35.59
35. Turo D, Otto P, Hossain M, et al. Novel Use of Ultrasound Elastography to Quantify Muscle Tissue Changes After Dry Needling of Myofascial Trigger Points in Patients With Chronic Myofascial Pain. *J Ultrasound Med.* 2015;34(12):2149-2161. doi:10.7863/ultra.14.08033
36. Hakimeh A, Azadeh S, Ebrahimi E, Asghar R, Naderi F. B mode, Doppler and ultrasound elastography imaging on active trigger point in women with myofascial pain syndrome treated by dry needling. *Muscle Ligaments and Tendons J.* 2019;09:417. Doi:10.32098/mltj.03.2019.16.
37. Couppé C, Midttun A, Hilden J, Jorgensen U, Oxholm P, Fuglsang-Frederiksen A. Spontaneous needle electromyographic activity in myofascial trigger points in the infraspinatus muscle: a blinded assessment. *J Musculoskeletal Pain.* 2001;9:7-16. doi: 10.1300/J094v09n03_02.

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Original Article

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Comparison of spasticity measurements with surface electromyography in the affected and unaffected side of children with hemiparetic cerebral palsy

Tolga Duman[1], Seyit Çitaker[2]

[1]Department of Physiotheraphy and Rehabilitation, Health Science Institute, Gazi University, Ankara, Turkey
[2]Department of Physiotheraphy and Rehabilitation, Faculty of Health Science, Gazi University, Ankara, Turkey

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Corresponding Author: Tolga Duman, tolga.duman@gazi.edu.tr

ABSTRACT

Aims: Various methods are used in the evaluation of spasticity, which is common in Cerebral Palsy (CP). However, studies measuring upper extremity spasticity with Surface Electromyography (sEMG) were found to be insufficient in children with CP. In this study, the comparison of Modified Ashworth Scale (MAS) and sEMG measurements for bilateral upper extremity spasticity in children with Hemiparetic CP was aimed.

Methods: 33 (11.03 ± 3.82 years, 11F-2M) patients with hemiparetic CP and 32 healthy controls (10.31± 2.88 years, 16F-16M) with a similar mean age were included in the study.

Results: Upper extremity spasticities of both sides of the cases were evaluated with MAS and SEMG. Affected side MAS results were found to be higher than the unaffected side ($p<0.05$). The mean sEMG value of the wrist flexors was lower on the affected side during voluntary contraction and higher during voluntary relaxation ($p<0.05$). In addition, the maximum percentage of voluntary contraction was measured higher on the affected side ($p<0.05$).

Conclusion: Spasticity, as assessed by sEMG and MAS, was increased on the affected side. MAS and sEMG give consistent results. These results show us that the YEMG method can be used for spasticity measurements. It is considered that MAS would be a preferable method in evaluating spasticity owing to the fact that it is easy to use and it does not constitute extra costs.

Keywords: Cerebral palsy, muscle spasticity, surface electromyography

INTRODUCTION

Cerebral Palsy (CP) is a non-progressive developmental disorder that occurs as a result of brain damage during the prenatal, perinatal, or post-natal period and it is the most striking reason for disability in children. The most common type is the Spastic type, characterized by an increase in the muscle tonus.[1] As well as the motor functions of the patients, the sensory, cognitive, behavioral, perceptive, and communication skills of the patients are also affected severely.[2] Amongst the common problems observed along with Spasticity could be muscle shortness, involuntary contractions, and negligence of the affected side.[3,4]

When one half of the body is more affected than the other half, it is called hemiparetic CP. Although it may seem that only one side is affected in hemiparetic CP, both halves of the body are affected at different levels. This is defined by the transverse advancement of 90% of the corticospinal pathways whereas the rest advance ipsilateral.[5,7] It has been determined that there is a more distinctive impaction on the upper extremity, hands in particular, on 50% of the children with hemiplegic CP.[8] Observed in most of the patients, the hemiplegic hand, which is a spastic pattern where the forearm is in pronation, the elbow-wrist and fingers are in flexion and the thumb is in the palm, is an indicator of such impaction.[9-11]

Spasticity clinical manifestation, recognized as tonus increase, is characterized by decreased voluntary motor movement, increased reflex responses, and agonist-antagonist tone regulation disorder.[6,7] Since it is a multifaceted finding, its clinical evaluation is difficult. No measurement method can evaluate spasticity in all its aspects (speed dependence, frequency, severity, involuntary muscle contractions, phasic and tonic components, etc.).[12] The most frequent clinical evaluation of Spasticity is implemented using the Modified Ashworth Scale (MAS), in which resistance to passive movement is graded, as well as the Modified Tardieu, Fugyl Meyer, and Tonus Evaluation Scales.[13,14] Recent studies reveal that Surface Electromyography (sEMG) applications are also used in spasticity measurements.[15] Contrary to the aforementioned scales, the sEMG is a method that provides objective data and measures the electrical activity of skeletal muscles. sEMG, which is a non-invasive method, indicates the action potential values of the muscles during contraction and relaxation.[16] In the literature, studies measuring upper extremity spasticity using sEMG were found to be

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insufficient in children, unlike adult hemiplegia patients.[17-19] In this study, it was aimed to evaluate upper extremity spasticity in children with hemiparetic CP using sEMG and MAS and to compare the results of affected and unaffected extremities with each other and with healthy controls.

METHODS

Design

This was a cross-sectional study. In this study, children who were rehabilitated in Special Education and Rehabilitation Centres and diagnosed with Hemiparetic CP were evaluated.

Before initiating the study, ethical approval was obtained from the Hatay Mustafa Kemal University Tayfur Ata Sökmen Medical Faculty Clinical Researches Ethics Committee (Date: 08.04.2021, Decision No: 2021/35). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki

After giving detailed information about the study to the patients and their relatives, patients who voluntarily agreed to participate in the study and signed the consent form were recruited. Patients 12 years of age and older gave their consent for the study. Parental consent was obtained for patients younger than 12 years of age.

The inclusion criteria:

- Patients diagnosed with Spastic Hemiparetic CP
- Clinically stable patients
- Patients in the age group of 5-17 years.

The exclusion criteria are:

- Patients with severe cognitive impairment
- Those with pulmonary, neurological, and orthopedic diseases that will affect functionality.
- Patients who have undergone Botox procedures in the last 6 months.
- Patients with a history of surgery in the upper extremity.
- Patients hospitalized due to acute infection or exacerbation.

Evaluation Methods

Patients' information such as age, height, weight, gender, disability, background, and family history were recorded via data recording and evaluation form.

In this study, MAS and sEMG were used for spasticity assessment. MAS is the most widely used clinical scale to evaluate spasticity. Despite its widespread clinical use, the reliability of the scale is questioned in some studies.[15,16]

During the MAS evaluation, the individual lies on the bed in a relaxed supine position and the upper extremity to be evaluated is manually moved by the physiotherapist quickly and repetitively. During this passive movement, an appropriate score from 0 to 4 (0,1,1+,2,3,4) is attributed to the spasticity level as per the resistance of the muscle to be evaluated.[17] The increase in the score indicates an increase in spasticity.

sEMG is a method in which electronic devices are used to measure the electrical activity of active skeletal muscles. This electrical energy is converted into a form that humans can perceive and is transferred to the computer screen as a graphic or sound. Electrodes are used non-invasively in sEMG. It is a painless, harmless, and objective method. It also provides the opportunity to document muscle activity. The higher the sEMG activation, the greater the force produced by the muscle. The EMG signal is the electrical appearance of the neuromuscular activation of the contracted muscle and is one of the most easily measurable signals.[18]

In this study, a four-channel Neurotrac Myoplus Pro 4 EMG Biofeedback device was used for sEMG Biofeedback measurements. The program of the sEMG Biofeedback device was installed on the laptop computer and the device was connected to the computer via Bluetooth. The sEMG measurements of the wrist extensor and flexor muscles of the patients were performed as per the action potentials during "work=voluntary contraction" and "rest=voluntary relaxation" (Figure 1).

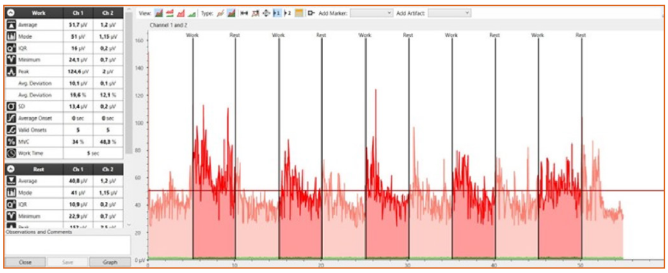

Figure 1. Affected Side sEMG result sample

When the individual to be tested was given "flex" and "relax" commands during flexor muscle activity, s/he contracted and relaxed the forearm flexor muscles. During the measurements which lasted 50 seconds, the measurement data were automatically recorded while the patient made voluntary muscle contractions and voluntary muscle relaxations five times. Mean contraction and mean relaxation activities, maximum contraction and relaxation activity percentages, mean deviation of relaxation, peak and minimum values, mean onset and release times were recorded with the device (Figure 1).

All evaluation measurements of the patients were conducted by the same physiotherapist.

Statistical Analysis of the Data

IBM SPSS 20.0 statistical program was used for statistical analysis. Descriptive statistics of the variables were calculated. In the comparison of affected and unaffected parties, the t-test was used in independent groups, and in the cases where the data did not comply with the normal distribution, the Mann Whitney-U test was used. The Spearman correlation analysis was used to evaluate the relationships.

In the interpretation of the correlation coefficient (r); Between 0.00-0.25 for a very weak correlation, 0.26-0.49 for a weak relationship, 0.50 and 0.69 for a moderate relationship, 0.70-0.89 for a high correlation, and between 0.90-1.0 for high correlation.[20] The probability of error in statistical analysis was determined as $p<0.05$ unless stated otherwise.

According to the G-Power (3.1.9.7) post hoc power analysis performed after the study, the effect size was calculated as=0.74, and the power of the study has been calculated as $(1-\beta)=0.91$ while $\alpha=0.05$.

RESULTS

33 patients and 32 healthy controls were included in the study (Figure 2). The sEMG values of the dominant sides of 32 healthy children [10 (8-13) years] who were in the same age range as the patients [12 (8-14) years] were used as the control group ($p=0.341$).

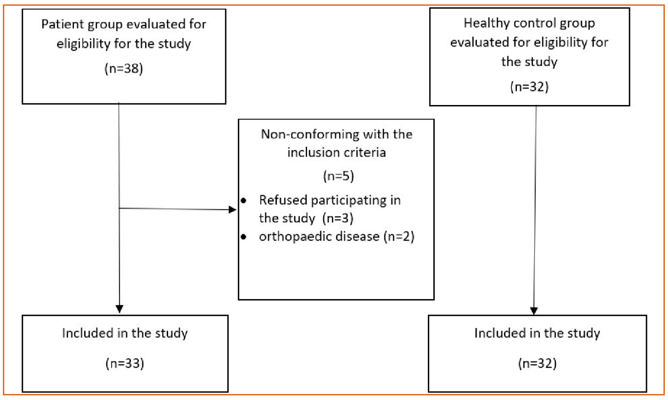

Figure 2. Workflow diagram

Age, height, weight, body mass index, gender, and demographic characteristics of the affected side of patients with CP and healthy controls are shown in **Table 1**. In Table 2, MAS measurement values for the pronator, wrist flexor, and elbow flexor muscles in children with CP are given for the affected and unaffected side and the MAS results on the affected side were found to be statistically and significantly higher than the unaffected side (p<0.05) (**Table 2**).

Table 1. Demographic characteristics of patients and healthy controls

Variables	CP X±CD M (IQR)	Control X±CD M (IQR)	p
Age (year)	12 (8-14)	10 (8-13)	0.314
Height (cm)	139.80±22.10	140.18±17.79	0.939
Weight (kg)	37 (23-50.50)	36.50 (26.25-55)	0.713
BMI (kg/m2)	17.63 (15.67-21.55)	19.26 (15.47-21.23)	0.865
	n/%		
Gender			0.213
Female	11 (33.3)	16 (50)	
Male	22 (66.7)	16 (50)	
Affected side			
Right	20 (61)		
Left	13 (39)		

X: mean, SD: Standard Deviation, M: Median, BMI: Body Mass Index.

Table 2. Comparison of sEMG and MAS measurement values on the affected and unaffected side in children with CP

Variables	AS X±SD M (IQR)	UAS X±SD M (IQR)	p
WFM Work			
AV (µV)	63.10 (45.05-77.45)	80.80 (56.50-111.90)	0.033*
MIN (µV)	4.7 (3.05-8.20)	3 (1.75-4.80)	0.022*
MVC (%)	30.89±14.10	26.14±9.38	0.112
WFM Rest			
AV (µV)	12.90 (7.20-17.95)	8.90 (4.55-13.95)	0.027*
MIN (µV)	2.60(1.10-5.50)	1.80(1.00-3.30)	0.172
MVC (%)	5.10(2.60-10.70)	2.70(1.20-4.25)	0.001*
WEM Work			
AV (µV)	67.90 (45-125.35)	95.10 (66.95-156.55)	0.081
MIN (µV)	5.60(2.85-9.85)	2.60(1.00-4.80)	0.005*
MVC (%)	34.76±11.09	31.29±10.42	0.195
WEM Rest			
AV (µV)	15.30 (10.60-23.25)	9.20 (5.95-14.15)	0.001*
MIN (µV)	4.20 (2.45-6.90)	1.80(1.00-3.30)	<0.001*
MVC (%)	7.60(4.30-11.25)	2.60 (1.55-4.60)	<0.001*
PS (MAS)	3 (2-3)	0(0-0.50)	<0.001*
WFS (MAS)	2 (2-3)	0 (0-0)	<0.001*
EFS (MAS)	2 (1-2)	0(0-0)	<0.001*

*p<0,05, X: mean, SD: Standard Deviation, M: Median, µV: Microvolt, AS: Affected Side, UAS: Unaffected Side, MVC: Maximum Voluntary Contraction Percentage, WFM: Wrist Flexor Muscles, WEM: Wrist Extensor Muscles, AV: Average EMG value, MIN: Minimum EMG value, MAS: Modified Ashworth Scale, PS: Pronator Spasticity, WFS: Wrist Flexor Spasticity, EFS: Elbow Flexor Spasticity.

The mean sEMG value during voluntary muscle contraction of the wrist flexor muscles on the affected side was statistically and significantly lower than the unaffected side, and the minimum sEMG value was higher (p<0.05). The mean sEMG and voluntary contraction percent MVC (%) values of the wrist flexor muscles during voluntary relaxation were also higher on the affected side (p<0.05). Maximum voluntary contraction percent MVC (%) values of wrist flexor muscles during voluntary muscle contraction were found to be similar on both sides (p>0.05) (**Table 2**).

The minimum sEMG value during voluntary muscle contraction of the wrist extensor muscles and the minimum sEMG and voluntary contraction percent MVC values during voluntary relaxation were higher on the affected side (p<0.05) (**Table 2**).

When the sEMG values of the unaffected side of the patients were compared with the healthy group; AV and MVC values of wrist extensors during voluntary contraction were lower on the unaffected side (p<0.05, **Table 3**). While there was no significant difference in the sEMG findings of the wrist flexors, it was found that the unaffected side was higher when the MAS data were examined (p=0.012).

Table 3. Comparison of sEMG values of the unaffected side and healthy controls in children with CP

Variables	UAS X±SD M (IQR)	CONTROL X±SD M (IQR)	p
WFM Work			
AV (µV)	80.80 (56.50-111.90)	98.40 (60.30-140.12)	0.325
MIN (µV)	3 (1.75-4.80)	3.30 (1.67-4.30)	0.979
MVC (%)	26.14±9.38	27.16±6.46	0.615
WFM Rest			
AV (µV)	8.90 (4.55-13.95)	7.45 (6.65-9.47)	0.783
MIN (µV)	1.80 (1.00-3.30)	2.40 (1.65-3.02)	0.318
MVC (%)	2.70 (1.20-4.25)	2.30 (1.50-3.27)	0.581
WEM Work			
AV (µV)	95.10 (66.95-156.55)	154.35 (108.85-195.35)	0.005
MIN (µV)	2.60 (1.00-4.80)	2.70 (1.45-4.37)	0.773
MVC (%)	31.29±10.42	38.18±5.41	0.001
WEM Rest			
AV (µV)	9.20 (5.95-14.15)	11.55 (5.67-13.02)	0.763
MIN (µV)	1.80 (1.00-3.30)	1.95 (1.17-3.80)	0.152
MVC (%)	2.60 (1.55-4.60)	2.80 (1.72-3.65)	0.665

*p<0,05, X: mean, SD: Standard Deviation, M: Median, µV: Microvolt, UAS: Unaffected Side, MVC: Maximum Voluntary Contraction Percentage, WFM: Wrist Flexor Muscles, WEM: Wrist Extensor Muscles, AV: Average EMG value, MIN: Minimum EMG value.

There was a weak positive correlation between the MAS on the affected side and the minimum sEMG (p=0,016 r=0,416) and MVC (p=0,021, r=0,401) values of the wrist flexor muscles during voluntary relaxation and a weak negative correlation between the mean sEMG value (p=0,030, r=-0,379) during voluntary contraction.

DISCUSSION

In this study, which aims to measure and compare spasticity values in children with hemiparetic SP using MAS and sEMG methods; [1] mean sEMG values of wrist flexor muscles during voluntary contraction are lower on the affected side; [2] mean FEMG values of wrist flexor muscles during voluntary relaxation are higher on the affected side; [3] sEMG values of the unaffected wrist extensors during voluntary contraction were lower compared to the healthy group; [4] There is no significant difference in sEMG findings of wrist flexors with healthy controls. When the MAS data is considered, it has been determined that spasticity on the unaffected side was higher; [5] it was ascertained that there is a positive correlation between the MAS of the wrist flexor muscles on the affected side and the minimum sEMG values during voluntary relaxation, and a negative one between the mean sEMG values during voluntary contraction.

Spasticity; It is an important complication that often accompanies CP and limits the mobility of the person.[21] Although it is easy to detect the presence of spasticity, it is not easy to quantify and grade. The degree of spasticity can vary from mild muscle stiffness to severe and sometimes painful muscle spasms.[22]

In the evaluation of spasticity, scales such as Ashworth Scale (AS), Modified Ashworth Scale (MAS), Modified Tardieu Scale (MTS), Fugyl Meyer Scale, Tonus Evaluation Scale are used in the clinic.[14,15,23]

MAS is frequently preferred in the clinic because it does not require any measuring device, it is easy and fast to apply.[24] However, the objectivity of MAS is questioned in current studies and it is emphasized that more studies are needed on this subject.[25-29]

Tederko et al.[30] evaluated the relationship between MAS and clinical examination results in 30 patients with spinal cord injury in a study they conducted and stated that MAS was insufficient to evaluate the tone of individual muscles, but it could indicate the increase in the muscle tonus in total. In addition, it was pointed out that it is difficult to repeat in young patients and that it is not a suitable method for evaluation in case of contracture. In a double-blind study of spinal cord injuries, the reliability of MAS was investigated among different evaluators. They reported that the MAS results were of poor reliability, both in the individual measurements of the evaluators and when compared with each other. In the same study, it was emphasized that the MAS was not psychometrically reliable and insufficient to measure rehabilitation effectiveness and it was argued that its use should be terminated.[31]

Alibioglu et al.[32] compared the MAS and neuromechanical measurement results of 34 stroke patients on both paretic and nonparetic lower and upper extremities. As a result, they found that there was no significant correlation between quantitative measurements of neural and muscle components of joint dynamic stiffness and MAS scores for neither the upper extremity nor the lower extremity. With these findings, they stated that Modified Ashworth scores were quite inconsistent with more objective spasticity measurements.

In their study comparing MAS and MTS measurements, Numanoğlu and Günel evaluated the lower and upper extremity muscles of 37 children with Spastic CP using both methods. As a result, it was determined that the reliability of MTS was better than MAS in each tested muscle. Although MAS is more advantageous than MTS in terms of applicability and time, it has been stated that it does not meet the requirements for clinical use because it depends on subjective decisions.[23] In our study, it was found that MAS can be used to detect the presence of spasticity and it is significantly higher on the affected side. Since the rehabilitation program was not applied to our patients and did not include follow-up results, the effect of determining the change in spasticity cannot be explained by the results of this study.

In addition to manual tests which are used to determine the severity of spasticity in the clinic, there are also biomechanical and electrophysiological methods that provide objective data.[24]

Electromyography and surface electromyography are among the evaluation methods that measure the electrical activity of active skeletal muscles with electronic devices.[18]

In a study conducted on spastic diplegic patients using EMG, it was shown that the mean EMG frequencies of the muscles in CP were higher than in the control group. In addition, it was also emphasized in the same study that dysfunction in the distal extremity muscles was more pronounced than in the proximal muscles.[33] In a study by Feltham et al.[24] on children with spastic hemiparetic CP, it was reported that sEMG values were higher in all muscles of the affected upper extremity and in the wrist and elbow flexor muscles of the unaffected side than in healthy controls. It was emphasized that this situation could be explained by the presence of systematic atrophy of type I muscle fibers in the heavily damaged (affected) arm.[24,34] Similarly, in our study, mean sEMG values of wrist flexor muscles during voluntary contraction were lower on the affected side; whereas the mean sEMG values of the wrist flexor muscles during voluntary relaxation were higher on the affected side. In the literature, it is suggested that the high-frequency components of the EMG spectrum are a reflection of the content of high-frequency action potentials produced by fast type II muscle fibers, while it is emphasized that slow type I muscle fibers produce low-frequency action potentials.[35,36]

When a muscle action potential is measured with sEMG in healthy individuals, it is expected that the muscle action potential will be high during maximum voluntary contraction and the muscle action potential will be at the lowest level during maximum voluntary relaxation.[37,38] Our results show that the muscle action potential value during maximum voluntary contraction may be lower than expected, and the muscle action potential value during maximum voluntary relaxation may be higher than expected in individuals with spasticity.

Unilateral brain injury causes changes in motor control not only of the contralateral body side but also of the ipsilateral side.[39,40] Staudt et al.[41] stated that in unilateral brain damage, there may be motor changes not only on the contralateral side but also on the ipsilateral side.

In our study, data from least affected parties were compared with healthy controls. Similar to the literature, sEMG values of the wrist extensors of the unaffected side during voluntary contraction were found to be lower compared to the healthy group.[24] While there was no significant difference between the FEMG findings of the wrist flexors and the healthy controls, it was observed that spasticity was higher on the unaffected side when the MAS data were examined.

In our study, the fact that MAS and sEMG revealed similar results when compared to both affected and unaffected extremities, as well as healthy controls, indicates that both measurement methods can be used in the evaluation of spasticity. When the correlation between MAS results and FEMG was examined, it was determined that there was a positive correlation between the MAS of the wrist flexor muscles on the affected side and the minimum sEMG values during voluntary relaxation, whereas there was a negative correlation between the mean sEMG values during voluntary contraction.

Validity and reliability studies for MAS will further clarify this study.

Limitations of the study

Because the study was conducted during the pandemic period, the high rate of absence of patients attending Special Education and Rehabilitation Canters as a precaution caused longer measurement times and problems in transportation to patients.

The results of future studies with more patients may differ and bring more clarity to the issue. The results can be generalized by conducting studies not only in a single city but also in other cities to be selected from different regions of Türkiye.

CONCLUSION

As a result, although sEMG is not as widely used as MAS in spasticity measurements in children with hemiparetic CP, the fact that the data obtained from sEMG have results based on numerical computerized measurement data may show that it can provide more objective data. In addition, our results show that MAS, which is widely used in clinic, gives results compatible with sEMG. These results show us that the YEMG method can be used for spasticity measurements. It is considered that MAS would be a preferable method in evaluating spasticity since it is easy to use and does not constitute extra costs. It is suggested that there is a need for validity and reliability studies in which the sEMG measurement method used in the evaluation of spasticity is investigated in detail.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of the Hatay Mustafa Kemal University Tayfur Ata Sökmen Medical Faculty Clinical Researches Ethics Committee (Date: 08.04.2021, Decision No: 2021/35).

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Cans C. Surveillance of cerebral palsy in Europe: a collaboration of cerebral palsy surveys and registers. Surveillance of Cerebral Palsy in Europe (SCPE). *Dev Med Child Neurol.* 2000;42(12):816-824. doi:10.1017/s0012162200001511
2. Beckung E, Hagberg G. Neuroimpairments, activity limitations, and participation restrictions in children with cerebral palsy. *Dev Med Child Neurol.* 2002;44(5):309-316. doi:10.1017/s0012162201002134
3. Sanger TD. Movement Disorders in Cerebral Palsy. *J Pediatr Neurol.* 2015; 13(04):198-207.
4. Pascual JM, Koenigsberger MR. Parálisis cerebral: factores de riesgo prenatales [Cerebral palsy: prenatal risk factors]. *Rev Neurol.* 2003;37(3):275-280.
5. Erol E, Elbasan B, Erol BÖ, et al. Pediatrik fizyoterapi rehabilitasyon ünitesine başvuran hastaların demografik özellikleri. *Gazi Sağlık Bilim Derg.* 2016; 1(1): 49-59.
6. Kutlay S. Spastik El, Türkiye Klinikleri. *J PM&R Special Topics.* 2008; 1:84-97.
7. Rosales RL, Chua-Yap AS. Evidence-based systematic review on the efficacy and safety of botulinum toxin-A therapy in post-stroke spasticity. *J Neural Transm (Vienna).* 2008;115(4):617-623. doi:10.1007/s00702-007-0869-3
8. McCall JV, Ludovice MC, Blaylock JA, Kamper DG. A Platform for Rehabilitation of Finger Individuation in Children with Hemiplegic Cerebral Palsy. *IEEE Int Conf Rehabil Robot.* 2019;2019:343-348. doi:10.1109/ICORR.2019.8779537
9. Shaw L, Rodgers H, Price C, et al. BoTULS: a multicentre randomised controlled trial to evaluate the clinical effectiveness and cost-effectiveness of treating upper limb spasticity due to stroke with botulinum toxin type A. *Health Technol Assess.* 2010;14(26):1-iv. doi:10.3310/hta14260
10. Unlu E, Çevikol A, Çakcı A. Spastik Elde Değerlendirme ve Tedavi Yontemleri. *FTR Bil Der.* 2011; 24(7):24-27.
11. Keklicek H, Uygur F, Yakut Y. Effects of taping the hand in children with cerebral palsy. *J Hand Ther.* 2015;28(1):27-33. doi:10.1016/j.jht.2014.09.007
12. Surkar SM, Hoffman RM, Willett S, Flegle J, Harbourne R, Kurz MJ. Hand-arm bimanual intensive therapy improves prefrontal cortex activation in children with hemiplegic cerebral palsy. *Pediatr Phys Ther.* 2018;30(2):93-100. doi:10.1097/PEP.0000000000000486
13. Rasool G, Afsharipour B, Suresh NL, Rymer WZ. Spatial Analysis of Multichannel Surface EMG in Hemiplegic Stroke. *IEEE Trans Neural Syst Rehabil Eng.* 2017;25(10):1802-1811. doi:10.1109/TNSRE.2017.2682298
14. Li Y, Zhang X, Gong Y, Cheng Y, Gao X, Chen X. Motor function evaluation of hemiplegic upper-extremities using data fusion from wearable inertial and surface EMG sensors. *Sensors (Basel).* 2017;17(3):582. doi:10.3390/s17030582
15. Alhusaini AA, Dean CM, Crosbie J, Shepherd RB, Lewis J. Evaluation of spasticity in children with cerebral palsy using Ashworth and Tardieu Scales compared with laboratory measures. *J Child Neurol.* 2010;25(10):1242-1247. doi:10.1177/0883073810362266
16. Alibiglou L, Rymer WZ, Harvey RL, Mirbagheri MM. The relation between Ashworth scores and neuromechanical measurements of spasticity following stroke. *J Neuroeng Rehabil.* 2008;5:18. doi:10.1186/1743-0003-5-18
17. Başarır M, Özek MM. Spastisite ve tedavisi. *Türk Nöroşir Derg.* 2013; 23:158-173.
18. Cerrah AO, Ertan H, Soylu AR. Spor bilimlerinde elektromiyografi kullanımı. *Spormetre Beden Eğitimi ve Spor Bilimleri Derg.* 2010; 8(2): 43-49.
19. Lei Wang, Xin Guo, Peng Fang, et al. A new EMG-based index towards the assessment of elbow spasticity for post-stroke patients. *Annu Int Conf IEEE Eng Med Biol Soc.* 2017;2017:3640-3643. doi:10.1109/EMBC.2017.8037646
20. Hayran M, Hayran M. Sağlık araştırmaları için temel istatistik, 1. baskı, Omega araştırma, Ankara, 2011.
21. Kaya Kara Ö, Türker D, Özal C, et al. Serebral Palsi'de güncel fizyoterapi ve rehabilitasyon yaklaşımları. *Ufkun Ötesi Bilim Derg.* 2010; 10(1-2): 5-22
22. Nielsen JB, Crone C, Hultborn H. The spinal pathophysiology of spasticity--from a basic science point of view. *Acta Physiol (Oxf).* 2007;189(2):171-180. doi:10.1111/j.1748-1716.2006.01652.x
23. Numanoğlu A, Günel MK. Intraobserver reliability of modified Ashworth scale and modified Tardieu scale in the assessment of spasticity in children with cerebral palsy. *Acta Orthop Traumatol Turc.* 2012;46(3):196-200. doi:10.3944/aott.2012.2697
24. Biering-Sørensen F, Nielsen JB, Klinge K. Spasticity-assessment: a review. *Spinal Cord.* 2006;44(12):708-722. doi:10.1038/sj.sc.3101928
25. Pandyan AD, Price CI, Barnes MP, Johnson GR. A biomechanical investigation into the validity of the modified Ashworth Scale as a measure of elbow spasticity. *Clin Rehabil.* 2003;17(3):290-293. doi:10.1191/0269215503cr610oa
26. Ansari NN, Naghdi S, Hasson S, Azarsa MH, Azarnia S. The Modified Tardieu Scale for the measurement of elbow flexor spasticity in adult patients with hemiplegia. *Brain Inj.* 2008;22(13-14):1007-1012. doi:10.1080/02699050802530557
27. Yam WK, Leung MS. Interrater reliability of Modified Ashworth Scale and Modified Tardieu Scale in children with spastic cerebral palsy. *J Child Neurol.* 2006;21(12):1031-1035. doi:10.1177/7010.2006.00222
28. Malhotra S, Pandyan AD, Day CR, Jones PW, Hermens H. Spasticity, an impairment that is poorly defined and poorly measured. *Clin Rehabil.* 2009;23(7):651-658. doi:10.1177/0269215508101747
29. Fleuren JF, Voerman GE, Erren-Wolters CV, et al. Stop using the Ashworth Scale for the assessment of spasticity. *J Neurol Neurosurg Psychiatry.* 2010;81(1):46-52. doi:10.1136/jnnp.2009.177071
30. Tederko P, Krasuski M, Czech J, Dargiel A, Garwacka-Jodzis I, Wojciechowska A. Reliability of clinical spasticity measurements in patients with cervical spinal cord injury. *Ortop Traumatol Rehabil.* 2007;9(5):467-483.

31. Craven BC, Morris AR. Modified Ashworth scale reliability for measurement of lower extremity spasticity among patients with SCI. *Spinal Cord.* 2010;48(3):207-213. doi:10.1038/sc.2009.107
32. Alibiglou L, Rymer WZ, Harvey RL, Mirbagheri MM. The relation between Ashworth scores and neuromechanical measurements of spasticity following stroke. *J Neuroeng Rehabil.* 2008;5:18. doi:10.1186/1743-0003-5-18.
33. Wakeling J, Delaney R, Dudkiewicz I. A method for quantifying dynamic muscle dysfunction in children and young adults with cerebral palsy. *Gait Posture.* 2007;25(4):580-589. doi:10.1016/j.gaitpost.2006.06.009
34. Lieber RL, Steinman S, Barash IA, Chambers H. Structural and functional changes in spastic skeletal muscle. *Muscle Nerve.* 2004;29(5):615-627. doi:10.1002/mus.20059
35. Kupa EJ, Roy SH, Kandarian SC, De Luca CJ. Effects of muscle fiber type and size on EMG median frequency and conduction velocity. *J Appl Physiol (1985).* 1995;79(1):23-32. doi:10.1152/jappl.1995.79.1.23
36. Wakeling JM, Rozitis AI. Spectral properties of myoelectric signals from different motor units in the leg extensor muscles. *J Exp Biol.* 2004;207(Pt 14):2519-2528. doi:10.1242/jeb.01042
37. Garciaa MAC, Vieiria TMM. Surface electromyography: Why, when and how to use it. *Rev Andal Med Deporte.* 2011; 4(1):17-28.
38. Cerrah AO, Ertan H, Soylu AR. Elektromiyografi ile kuvvetin değerlendirilmesi. *Türkiye Klinikleri J Neur.* 2010; 5(3):160-166.
39. Yarosh CA, Hoffman DS, Strick PL. Deficits in movements of the wrist ipsilateral to a stroke in hemiparetic subjects. *J Neurophysiol.* 2004;92(6):3276-3285. doi:10.1152/jn.00549.2004
40. Wiley ME, Damiano DL. Lower-extremity strength profiles in spastic cerebral palsy. *Dev Med Child Neurol.* 1998;40(2):100-107. doi:10.1111/j.1469-8749.1998.tb15369.x
41. Staudt M, Grodd W, Gerloff C, Erb M, Stitz J, Krägeloh-Mann I. Two types of ipsilateral reorganization in congenital hemiparesis: a TMS and fMRI study. *Brain.* 2002;125(Pt 10):2222-2237. doi:10.1093/brain/awf227

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Pain, kinesiophobia, functional status, and physical activity among patients with knee osteoarthritis

Harun Durna[1], Özge Vergili[2], Seda Fidancı[3]

[1]Gebze Fatih State Hospital, Kocaeli, Turkey
[2]Department of Physical Therapy and Rehabilitation, Kırıkkale University, Kırıkkale, Turkey
[3]Department of Physical Medicine and Rehabilitation, Gebze Fatih State Hospital, Kocaeli, Turkey

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Corresponding Author: Harun Durna, hrndrn37@gmail.com

ABSTRACT

Aims: The present study attempted to investigate the relationship between pain, kinesiophobia, functionality, and physical activity among patients with knee osteoarthritis (OA).

Methods: We recruited 88 patients aged 40-70 years with OA, 21 males and 67 females, who applied to Gebze Fatih State Hospital, Department of Physical Therapy and Orthopedic Clinic between February 2022 and January 2023. We evaluated the patients' pain status with the Visual Pain Scale (VAS), their functional status with the WOMAC Osteoarthritis Index (WOMAC), their kinesiophobia with the TAMPA Scale for Kinesiophobia (TKS), and their physical activity levels with the International Physical Activity Questionnaire-Short Form (IPAQ-SF). We performed all statistical analyses using SPSS 25.0 (SPSS Inc., Chicago, IL, ABD) and p-value < 0.05 was accepted as statistically significant.

Results: The majority of the participants were females (76.1%, n=67), and the mean body mass index (BMI) was found to be 30.33. The findings revealed low positive correlations between the patients' BMI and their VAS (p=0.029) and WOMAC scores (p=0.017). While we found a moderate positive relationship between the patients' VAS and WOMAC scores (p=0.001), it was low between their TAMPA and WOMAC scores (p=0.001).

Conclusion: As a result of the study, the relationship between pain, kinesiophobia, functional status and physical activity levels of individuals with knee OA was revealed; The necessity of planning applications to increase the physical activity level of these individuals and to reduce their body mass index has been underlined.

Keywords: Osteoarthritis, physical activity, pain, functional status, kinesiophobia

This study was presented as a thesis at Kırıkkale University.

INTRODUCTION

Osteoarthritis (OA) is a long-term persisting chronic condition characterized by the deterioration of the cartilage in the joints, resulting in the bones rubbing against each other and causing stiffness, pain, and mobility impairment. This condition often affects the joints in the knees, hands, feet, and spine and is relatively prevalent in the shoulders and hip joints. Despite being primarily related to aging, OA is also associated with a variety of both modifiable and non-modifiable risk factors, including obesity, sedentary lifestyle, genetic predisposition, bone density, occupational injury, trauma, and sex.[1] In obese individuals, pain emerges most commonly in the lower extremities and weight-bearing joints, including the lower back, but may also occur in the upper extremity joints, hands and fingers, thoracic spine, and neck. Studies in cadavers have revealed that obesity is associated with higher knee OA severity compared to normal-weight individuals. In addition, the progression of OA is faster in obese individuals than in normal-weight individuals. Physical disability due to pain leads to obesity, followed by gait abnormalities and muscle weakness. In this regard, pain becomes an important factor by leading obesity-related deterioration in physical functionality and health-related quality of life.[2] Another important less-studied issue in OA is fear of movement (FOM), or kinesiophobia. It is defined as the fear of physical movement or activity resulting from the belief that it would cause pain, injury, or re-injury. The relevant research to date showed that FOM is associated with increased pain, poorer physical functionality, and greater physical and psychological disability, all of which are important paramaters for individuals with musculoskeletal disorders. In addition, FOM is associated with physical inactivity, which is particularly important for individuals with OA as activity is an essential component of their OA management.[3] Kinesiophobia due to pain is also linked with loss of functioning and physical performance in patients with knee OA. Thus, regular physical activity

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in light to moderate intensity is strictly recommended for its contribution to cardiovascular fitness and reducing the risk of diabetes and obesity. However, it is noticeable that previous research on individuals with OA rarely assessed their physical activity levels.[4]

When the literature is investigated, it is possible to come across various studies evaluating pain, functionality and balance in people with knee OA. However, there are few studies in which kinesiophobia and physical activity levels are evaluated in detail. Therefore, there is a need for studies evaluating the relationship between pain, kinesiophobia and physical activity levels.

The aim of this study is to investigate whether there is a relationship between pain, functionality, kinesiophobia and physical activity levels in individuals with knee OA.

METHODS

We carried out this study in Gebze Fatih State Hospital to explore the relationships between pain, kinesiophobia, functional status, and physical activity levels in individuals with knee OA. The Non-interventional Clinical Researches Ethics Committee of Kırıkkale University granted ethical approval to our study (Decision No: 2021.12.03, Date: 01.12.2022). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. In this regard, we recruited 88 patients aged 40-70 years with osteoarthritis, 21 males and 67 females, who applied to the Department of Physical Therapy and Orthopedics Clinic between February 2022 and January 2023. İndividuals with knee pain lasting more than 6 months, diagnosed with stage 2 or stage 3 knee osteoarthritis according to Kellgren Lawrence radiological staging in the radiological evaluation, and volunteered to participate in the study were included in this research. Nevertheless, we excluded patients having a hip or knee prosthesis, suffering any neurological disorders, having a different pathology other than gonarthrosis that could cause knee pain, having had an intraarticular knee injection for any reason in the last six months and having any cognitive and communication problems.

The G* Power program (version 3.0.10 Universität Düsseldorf, Düsseldorf, Germany) was used for the prior power analysis.[5] The effect size obtained in the reference study was found to be moderate (r=0.332). Considering that a lower level of effect size can also be obtained (r=0.3), as a result of the power analysis; It has been calculated that 80% power can be obtained at 95% confidence level when at least 82 people are included in the study. 88 people were included in our study. The effect size of the relationship between TAMPA questionnaire values and WOMAC levels obtained from these individuals was found to be moderate (r=0.355). For this effect size, it was calculated that our study reached 94% power at 95% confidence level.[6]

We administered a questionnaire to inquire about the participants' physical characteristics and demographic data. A Visual Analogue Scale (VAS) was used to assess pain. It is a ten cm-line scale ranging from 0 (no pain) to 10 (unbearable pain). In pain assessment, one is asked to mark a point on the scale according to the severity of the pain they feel. Then, the number denoted by that line is identified with the help of a ruler and noted down.[7] Besides, we utilized the TAMPA Kinesiophobia Scale (TKS) to measure the patients' fear of movement/re-injury. Published by Vlaeyen et al.[8] in 1995, it is a 17-item tool to measure fear of movement/re-injury and includes injury/re-injury and fear-avoidance parameters in work-related activities. Finally, we evaluated the patients' functional status with the WOMAC Osteoarthritis Index (WOMAC) and their physical activity levels with the International Physical Activity Questionnaire-Short Form (IPAQ-SF).

While continuous variables are presented as means, standard deviations, minimums, and maximums, categorical variables are demonstrated as numbers and percentages. Spearman's rank correlation coefficient (ρ) was calculated to reveal the associations between the variables. A ρ-value between 0.05-0.30 was accepted as a very weak or negligible correlation, between 0.30-0.40 as a low-to-moderate correlation, and between 0.40-0.60 as a moderate correlation, and between 0.60-0.70 as a robust correlation, and 0.70-1.00 as a perfect correlation.[9] We performed all statistical analyses using the Statistical Package for Social Sciences (SPSS) 22.0 (SPSS Inc., Chicago, IL, USA) program and considered a p-value < 0.05 to be statistically significant.

RESULTS

In the study, there were 88 patients, 21 males (23.9%) and 67 females (76.1%), aged between 40-70 years. While the youngest participant was 42 years old, the oldest one was 70 years old (M=57.69±7.95 years). Body mass index values were as follows: 48.9% (n=43) was found to be overweight, 27.3% (n=24) to be obese, 10.2% (n=9) to be severely obese, 4.5% (n=4) to be morbidly obese, 5.7% (n=5) to be normal, and 3.4% (n=3) to be underweight. The majority of the patients (67%, n=59) were housewives, 19.2% (n=17) were retired, 8% (n=7) were workers, and 5.7% (n=5) had a desk job.

Table 1. Patients' Demographic and Physical Characteristics

Variables	n=88	%	M±SD	Min.-Max.
Age			57.69±7.95	42-70
Employment				
Desk job	5	5.7		
Worker	7	8		
Retired	17	19.3		
Housewife	59	67		
BMI			30.33±5.2	17.58±46.88
Underweight	3	3.4		
Normal	5	5.7		
Overweight	43	48.9		
Obese	24	27.3		
Severely obese	9	10.2		
Morbidly obese	4	4.5		

SD: standard deviation, BMI: Body Mass Index, Min.: minimum, Max.: maximum, n: number of patients, %: percentage

The mean VAS score of the participants was found to be 6.25 (SD=1.4, min.=4, max.=9). The patients' BMI values varied between 17.58 and 46.88 (M=30.33, SD=5.2), while they scored between 29 and 51 on the TSK (M=43.35, SD=3.62). Their mean WOMAC score was calculated to be 40.06 (SD=16.41; min.=3, max.=75). Regarding IPAQ-SF, the lowest and highest metabolic equivalent of task (MET) were calculated to be 66 and 7413, respectively (M=836.09, SD=1127.64 (Table 2).

Table 2. Findings of Research Variables

n=88	M±SD	Min.-Max.
VAS	6.25±1.4	4-9
BMI	30.33±5.2	17.58-46.88
TKS	43.35±3.62	29-51
WOMAC	40.06±16.41	3-75
IPAQ-SF / MET	836.09±1127.64	66-7413

n: number of patients, SD: standard deviation, Min.: minimum, Max.: maximum, VAS: Visual Analog Scale, BMI: body mass index, TSK: TAMPA Kinesiophobia Scale, WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index, IPAQ-SF: International Physical Activity Questionnaire - Short Form, MET: metabolic equivalent of task.

We discovered 60.2% of the patients (n=53) to be inactive, 34.1% (n=30) to be minimally active, and 5.7% (n=5) to be very (HEPA) active (Table 3).

Table 3. Patients' Physical Activity Levels

Level	n	%
Inactive	53	60.2
Minimally active	30	34.1
Very (HEPA) active	5	5.7
Total	88	100

%: percentage, n: number of patients

Table 4 presents the relationships between pain, BMI, kinesiophobia, functionality, and physical activity among the participating patients.

The findings revealed a low positive correlation between the patients' VAS scores and BMI (p=0.029) but no significant relationship between their VAS and TKS scores. The patients' VAS scores had a low positive relationship with the WOMAC stiffness (p=0.026) and moderate positive associations with their WOMAC pain (p=0.001), WOMAC physical function (p=0.001), and WOMAC total scores (p=0.001). Except for WOMAC stiffness, the patients' BMI showed low positive correlations with their WOMAC pain (p=0.019) and WOMAC physical function (p=0.018), and WOMAC total scores (p=0.017). We could not conclude a significant relationship between the patients' TKS and IPAQ-SF scores. However, we discovered low-to-moderate associations between the patients' TKS scores and their WOMAC pain (p=0.001), WOMAC stiffness (p=0.003), WOMAC physical function (p=0.002), and WOMAC total scores (p=0.001) (Table 4).

DISCUSSION

In this study, we investigated the relationships between pain, kinesiophobia, functional status, and physical activity among patients with knee OA. Our findings showed that the patients' pain status had a low relationship with their BMI and a moderate link with their functional status. However, we could not find significant associations between the patients' pain and their kinesiophobia and physical activity levels. It was also the case between their kinesiophobia and functionality and physical activity levels. Similarly, there was no relationship between their functionality and physical activity levels.

The female sex is known to be one of the primary reasons for knee OA.[10] Despite little knowledge of why it is prevalent among women, it was previously claimed to be due to some factors, such as menopausal changes, changes to cartilage structure, and weaknesses in muscle strength and postural alignment in the lower extremities.[11] The prevalence of knee OA was also shown to be higher in women than in men.[12] In the study conducted by Tas et al.[13] on individuals with OA, 66 individuals were female and 14 individuals were male. Our study consisted of 88 individuals, 21 male and 67 female, and is compatible with the literature.

The incidence of symptomatic knee osteoarthritis is expressed as 13% in people over 55 years of age. In our country, knee OA due to symptoms in individuals over the age of 50; it was found to be 22.5% in female patients, 8% in males, and 14.8% in total.[12] In our study, the mean age was found to be 57.69 years, which is consistent with the literature.

Obesity and OA are two related health problems affecting the majority of the adult population.[14] It is predicted that the number of those with knee OA will increase due to the aging of the world population and the prevalence of obesity.[15] In the study by Ozcakır et al.[16] the mean BMI value of the patients with knee OA was 31.9. Similarly, in our study, the BMI values of individuals with osteoarthritis were found to be high, with an average of 30.33. In addition to negatively affecting many body systems, it is apparent that BMI is a significant risk factor for OA and needs to be controlled.

Table 4. Relationships Between Research Variables

		VAS	BMI	TSK	WOMAC Pain	WOMAC Stiffness	WOMAC Physical Function	WOMAC Total Score
VAS								
BMI		r=0.232 *p=0.029						
TSK		0.128 0.235	0.187 0.082					
WOMAC	Pain	r=0.596 **p<0.001	r=0.250 *p=0.019	r=0.363 **p=0.001				
WOMAC	Stiffness	r=0.238 *p=0.026	0.175 p=0.103	r=0.313 *p=0.003	r=0.382 **p<0.001			
WOMAC	Physical Function	r=0.494 **p<0.001	r=0.252 *p=0.018	r=0.322 *p=0.002	r=0.818 **p<0.001	r=.612 **p<0.001		
WOMAC	Total Score	r=0.530 **p<0.001	r=0.253 *p=0.017	r=0.355 **p=0.001	r=0.875 **p<0.001	r=0.624 **p<0.001	r=0.991 **p<0.001	
IPAQ-SF/MET		0.001 0.996	0.083 0.44	0.073 0.499	-0.044 0.686	-0.168 0.118	-0.072 0.505	-0.075 0.489

Spearman's Rank-Order Correlation, *p < 0.05 **p < 0.001. SD: standard deviation. BMI: body mass index. WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index. IPAQ-SF: International Physical Activity Questionnaire - Short Form, MET: metabolic equivalent of task TSK: TAMPA Kinesiophobia Scale

In obese individuals, pain commonly appears in the lower extremities and weight-bearing joints, including the lower back. Studies in cadavers have revealed that obesity is associated with higher knee OA severity compared to normal-weight individuals. Obesity is also associated with a faster progression of OA than normal weight. Reyes et al.[14] found that the risk of developing knee OA increased by two times in the overweight and up to 4.7 times in level 2 obese individuals.

Physical disability due to pain leads to obesity, followed by gait abnormalities and muscle weakness. It is also considered a significant factor that leads to impairment in physical functioning and health-related quality of life.[2] In a study conducted by Atamaz et al.[17] regarding pain and disability in OA; obesity, advanced age, female gender, and advanced radiological stage have been shown to be important factors. In our study, we found a significant relationship between the participants' BMI values and VAS scores. In line with this result, when we consider the mechanical effects of obesity in terms of OA, we see that individuals with high BMI have to cope with more pain.

People who are afraid of pain tend to perform worse because they want to avoid pain while doing activities of daily living. In OA patients who are more inactive, reductions in muscle strength are also seen, resulting in more activity limitation.[18] Pain occurring in the case of an injury restricts movements; the person may be afraid to move even when the injured part is recovered. The higher level of pain perception, the more avoidance response is seen in the person. Kori et al.[19] defined kinesiophobia as the feeling of sensitivity and anxiety against active movement caused by a pain-resulting injury and re-injury. Many studies have shown the negative effect of fear during movement in individuals with OA.[20] In the study of Scopaz et al.[21] in which they investigated the relationship between fear, anxiety, and depression of patients with knee OA, and their physical functions, it was found that high fear avoidance was associated with a decrease in patients' functionality. Similarly, in our study, a statistically significant correlation was found with TAMPA values in all sub-parameters of WOMAC such as pain, stiffness and physical function, and in the total WOMAC score. It was observed that the functional status of individuals with high levels of fear was also negatively affected. For this reason, kinesiophobia was seen as the possible reason why individuals with knee OA approach with fear and experience functional difficulties while participating in daily life activities.

Pain is considered a leading physical and psychological barrier for individuals with OA.[22] About 80% of patients with OA have movement restrictions due to pain, 11% have difficulties in personal care, and 25% deal with problems in their activities of daily living.[23] In our study, we found a significant relationship between pain and all WOMAC parameters. Thus, it can be proposed that pain negatively affects physical functioning.

A study was conducted by Alaca examining the relationship between pain and fear of movement and clinical parameters in individuals with knee OA. As a result of this research, a moderate statistical relationship was found between pain and kinesiophobia, and it was emphasized that individuals' pain perception levels were also important in this process.[24] In another study conducted by Erden et al.[8] to investigate pain status, kinesiophobia, anxiety level and quality of life in patients with knee osteoarthritis, no relationship was found between kinesiophobia and pain. In our study, similar results were found with Erden et al. No statistically significant relationship was found between the pain states of the individuals and their fear of movement. As stated in the study by Alaca, we think that individuals' perception of pain is important in reaching this result.

In the study on the relationship between the psychological status and physical activity levels of individuals with knee OA, Uritani et al.[25] found physical activity status to be associated with kinesiophobia. In the study conducted by Güney-Deniz et al.[26] on 46 patients who had undergone knee replacement surgery, the effect of post-operative fear on the functional status of patients in the early period was examined. According to the results of the study, the range of motion, pain status and physical activities of individuals with low kinesiophobia were found to be statistically more significant than individuals with high kinesiophobia scores. In another study by Monticone et al.[27] in which 110 individuals with knee replacement surgery were included, it was shown that the kinesiophobia of individuals who regularly engaged in physical activity decreased over time. In this research, we could not find a significant relationship between the patients' kinesiophobia scores and physical activity levels, which can be attributed to the participants' sedentary lifestyles.

Rosemann et al.[28] evaluated patients with hip and knee OA with the IPAQ to determine the factors affecting physical activity. Their results showed that 55% of the cases were sedentary, 38% were minimally active, and 7% were active. Then, they stated that it is essential to increase physical activity in the conservative treatment of OA. Considering the physical activity levels of our participants, 60.2% (n=53) were inactive, 34.1% (n=30) were minimally active, and 5.7% (n=5) were rather active. Therefore, in parallel with the literature, the majority of OA patients were found to be inactive. This situation should be taken into consideration when evaluating individuals with OA, and individuals should be tried to be more active with activities which are prescribed specific to patient needs.

Voorrips questionnaire was applied by Thomas et al.[29] to examine the level of physical activity in 59 patients with knee osteoarthritis. When they compared the level of physical activity with healthy individuals, they found that it was low in all areas (such as housework, leisure time, exercise). While it was stated that this condition was associated with low functional performance, they said that it was not associated with pain. In the study conducted by Tonetelli et al.[30] on 208 individuals with knee OA, pain alone was not to be sufficient to determine the level of physical activity; and showed that although there were significant differences between the pain states of individuals, their physical activity states were to be possibly similar. In our study, no statistically significant relationship was found between physical activity scores and pain, in line with the results of Thomas et al. We think that the reason for this may be depending on many factors, especially psychological ones, that affect physical activity as well as pain. In this direction, it was seen that it was necessary to carry out studies to increase the physical activity levels of individuals.

CONCLUSION

The results of our study contributed to the literature in terms of demonstrating the relationship between pain, kinesiophobia, functionality and physical activity in individuals with knee osteoarthritis. Since studies evaluating physical activity are limited in the literature, an additional contribution has been made to the literature in this sense.

It has been observed that individuals with knee OA are overweight, and since it is known that this situation will overload the joint mechanically, we think that these individuals should be referred to dietitians for appropriate diet programs. At the same time, this process should definitely be supported with exercise programs and trainings to be organized in accordance with these individuals. Since it is seen that the pain conditions of individuals with OA negatively affect the individuals functionally, preventive measures should be taken and studies should be carried out for pain. Again, since these individuals are seen to be inactive people, it is necessary to provide trainings to increase the level of physical activity, to raise awareness of individuals and to create appropriate exercise programs.

The lack of a control group in our study and the fact that the mini mental test was not used to determine the cognitive levels of individuals more objectively were considered as limitations.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of Kırıkkale University Non-interventional Clinical Researches Ethics Committee (Date: 01.12.2022, Decision No: 2021.12.03).

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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REFERENCES

1. Haq I, Murphy E, Dacre J. Osteoarthritis. *Postgrad Med J.* 2003;79(933):377-383. doi:10.1136/pmj.79.933.377
2. Vincent HK, Heywood K, Connelly J, Hurley RW. Obesity and weight loss in the treatment and prevention of osteoarthritis. *PM R.* 2012;4(5 Suppl):S59-S67. doi:10.1016/j.pmrj.2012.01.005
3. Gunn AH, Schwartz TA, Arbeeva LS, et al. Fear of movement and associated factors among adults with symptomatic knee osteoarthritis. *Arthritis Care Res (Hoboken).* 2017;69(12):1826-1833. doi:10.1002/acr.23226
4. Kilinç H, Karahan S, Atilla B, Kinikli Gİ. Can fear of movement, depression and functional performance be a predictor of physical activity level in patients with knee osteoarthritis?. *Arch Rheumatol.* 2018;34(3):274-280. doi:10.5606/ArchRheumatol.2019.7160
5. Faul F, Erdfelder E, Lang AG, Buchner A. G*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behav Res Methods.* 2007;39(2):175-191. doi:10.3758/bf03193146
6. Korkmaz, N. Diz osteoartritli hastalarda kinezyofobi, ağrı, fonksiyonel durum ve öz-etkililik arasındaki ilişkinin incelenmesi. Yüksek lisans tezi. Hacettepe Üniversitesi Sağlık Bilimleri Enstitüsü, 2019.Ankara.
7. Erden A, Malkoç A, Kocabal A. Diz Osteoartritli bireylerde kinezyofobi, ağrı şiddeti,anksiyete-depresyon durumu ve yaşam kalitesinin i̇ncelenmesi. *Uluslararası Hakemli Ortopedi Travmatoloji ve Spor Hekimliği Derg.* 2016;7:1-17.
8. Vlaeyen JWS, Kole-Snijders AMJ, Boeren RGB, van Eek H. Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. *Pain.* 1995;62(3):363-372. doi:10.1016/0304-3959(94)00279-N
9. Hayran, M. Sağlık araştırmaları için temel istatistik: Omega Araştırma. 2011.
10. Elboim-Gabyzon M, Rozen N, Laufer Y. Gender differences in pain perception and functional ability in subjects with knee osteoarthritis. *ISRN Orthop.* 2012;2012:413105. doi:10.5402/2012/413105
11. Silva A, Serrão PR, Driusso P, Mattiello SM. The effects of therapeutic exercise on the balance of women with knee osteoarthritis: a systematic review. *Rev Bras Fisioter.* 2012;16(1):1-9.
12. Uysal G, Başaran S. Diz Osteoartriti. *Türk Fiz Tıp Rehab Derg.* 2009;55: 1-7.
13. Taş S, Güneri S, Baki A, Yıldırım T, Kaymak B, Erden Z. Diz osteoartriti şiddetinin yürüyüşün zaman mesafe parametreleri üzerine etkileri. *Acta Orthop Traumatol Turc.* 2014;48(6):635-41.
14. Reyes C, Leyland KM, Peat G, Cooper C, Arden NK, Prieto-Alhambra D. Association between overweight and obesity and risk of clinically diagnosed knee, hip, and hand osteoarthritis: a population-based cohort study. *Arthritis Rheumatol.* 2016;68(8):1869-1875. doi:10.1002/art.39707
15. Kiadaliri AA, Lamm CJ, de Verdier MG, et al. Association of knee pain and different definitions of knee osteoarthritis with health-related quality of life: a population-based cohort study in southern Sweden. *Health Qual Life Outcomes.* 2016;14(1):121. doi:10.1186/s12955-016-0525-4
16. Ozcakir S, Raif SL, Sivrioglu K, Kucukcakir N. Relationship between radiological severity and clinical and psychological factors in knee osteoarthritis. *Clin Rheumatol.* 2011;30(12):1521-1526. doi:10.1007/s10067-011-1768-4
17. Atamaz F, Hepgüler S, Öncü J. Diz osteoartritinde ağrı ve özürlülükle ilişkili faktörler. *Türk Fiz Tıp Rehab Derg.* 2006; 52:119-22.
18. Tanaka R, Hirohama K, Ozawa J. Can muscle weakness and disability influence the relationship between pain catastrophizing and pain worsening in patients with knee osteoarthritis? A cross-sectional study. *Braz J Phys Ther.* 2019;23(3):266-272. doi:10.1016/j.bjpt.2018.08.011
19. Yılmaz Ö, Yakut Y, Uygur F, Uluğ N. Tampa Kinezyofobi Ölçeğinin Türkçe Versiyonu ve Test-tekrar Test Güvenilirliği. *Fizyoterapi Rehabilitasyon.* 2011;22(1):44- 49.
20. Shelby RA, Somers TJ, Keefe FJ, et al. Brief Fear of Movement Scale for osteoarthritis. *Arthritis Care Res (Hoboken).* 2012;64(6):862-871. doi:10.1002/acr.21626
21. Scopaz KA, Piva SR, Wisniewski S, Fitzgerald GK. Relationships of fear, anxiety, and depression with physical function in patients with knee osteoarthritis. *Arch Phys Med Rehabil.* 2009;90(11):1866-1873. doi:10.1016/j.apmr.2009.06.012
22. Pells JJ, Shelby RA, Keefe FJ, et al. Arthritis self-efficacy and self-efficacy for resisting eating: relationships to pain, disability, and eating behavior in overweight and obese individuals with osteoarthritic knee pain. *Pain.* 2008;136(3):340-347. doi:10.1016/j.pain.2007.07.012
23. Deveza LA, Hunter DJ. Pain relief for an osteoarthritic knee in the elderly: a practical guide. *Drugs Aging.* 2016;33(1):11-20. doi:10.1007/s40266-015-0331-4
24. Alaca N. The relationships between pain beliefs and kinesiophobia and clinical parameters in Turkish patients with chronic knee osteoarthritis: a cross-sectional study. *J Pak Med Assoc.* 2019;69(6):823-827.
25. Uritani D, Kasza J, Campbell PK, Metcalf B, Egerton T. The association between psychological characteristics and physical activity levels in people with knee osteoarthritis: a cross-sectional analysis. *BMC Musculoskelet Disord.* 2020;21(1):269. doi:10.1186/s12891-020-03305-2
26. Güney-Deniz H, Irem Kınıklı G, Çağlar Ö, Atilla B, Yüksel İ. Does kinesiophobia affect the early functional outcomes following total knee arthroplasty?. *Physiother Theory Pract.* 2017;33(6):448-453. doi: 10.1080/09593985.2017.1318988
27. Monticone M, Ferrante S, Rocca B, et al. Home-based functional exercises aimed at managing kinesiophobia contribute to improving disability and quality of life of patients undergoing total knee arthroplasty: a randomized controlled trial. *Arch Phys Med Rehabil.* 2013;94(2):231-239. doi:10.1016/j.apmr.2012.10.003
28. Rosemann T, Kuehlein T, Laux G, Szecsenyi J. Factors associated with physical activity of patients with osteoarthritis of the lower limb. *J Eval Clin Pract.* 2008;14(2):288-293. doi:10.1111/j.1365-2753.2007.00852.x
29. Thomas SG, Pagura SM, Kennedy D. Physical activity and its relationship to physical performance in patients with end stage knee osteoarthritis. *J Orthop Sports Phys Ther.* 2003;33(12):745-754. doi:10.2519/jospt.2003.33.12.745
30. Tonelli SM, Rakel BA, Cooper NA, Angstom WL, Sluka KA. Women with knee osteoarthritis have more pain and poorer function than men, but similar physical activity prior to total knee replacement. *Biol Sex Differ.* 2011;2:12. doi:10.1186/2042-6410-2-12

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Review

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Biomechanics, pathomechanics, diagnosis, treatment, and return to play criterias of lateral ankle sprains: an evidence-based clinical guideline

Fatih Özyurt[1], Caner Karartı[1], Hakkı Çağdaş Basat[2]

[1]Department of Physiotherapy and Rehabilitation, Kırşehir Ahi Evran University, Kırşehir, Turkey
[2]Department of Orthopedics and Traumatology, Kırşehir Ahi Evran University, Kırşehir, Turkey

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Corresponding Author: Caner Karartı, fzt.caner.92@gmail.com

ABSTRACT

Lateral ankle sprain (LAS), which is common in recreational and professional athletes, recurs and becomes chronic if left untreated. Since the number of mechanoreceptors it contains is high, LAS should not be considered only as a musculoskeletal disorder. A detailed clinical evaluation is recommended. Clinically, the patient presents with limitation of dorsiflexion range of motion (ROM), muscle atrophy and unequal strength between the muscles, deterioration in walking, running and jumping, and proprioceptive losses. The patient should be approached from a biopsychosocial perspective both in the evaluation and treatment phases. The treatment of the patient is carried out in 3 phases and the transition between phases should be based on mentioned criterias. In the acute phase of rehabilitation, emphasis should be placed on eliminating pain and edema, and preventing loss of ROM and muscle strength. In the subacute and chronic stages, emphasis should be placed on increasing the proprioceptive sense and muscle strength, improving the biomechanics of daily life and sports-specific movements such as walking, running, jumping. If the patient is an athlete, return to sports should be planned after the criterias met.

Keywords: Lateral ankle sprain, rehabilitation, foot, ankle

INTRODUCTION

Stabilization of the talocrural joint is provided by the passive subsystem, the active subsystem, and the neural subsystem. While the active substructures responsible for stabilization are intrinsic and extrinsic foot muscles, the neural subsystem is musculotendinous receptors, local and global ligamentous receptors, and plantar cutaneous receptors.[1,2] The passive subsystem consists of the bone structures that make up the arches, the plantar fascia and the ligaments.[1] Medial to the ankle joint is the deltoid ligament, while lateral to the ankle joint is the anteriortalofibular ligament (ATFL), calcaneofibular ligament (CFL) and posteriortalofibular ligament (PTFL). Injury to one or all of these ligaments is called a lateral ankle sprain (LAS). LAS is also known as inversion or over-supinated injuries.[2] LAS is not only a musculoskeletal injury but also a neurophysiological injury due to the high number of mechanoreceptors contained in the ligaments.[3,4] LAS is one of the most common musculoskeletal injuries among both recreational and professional athletes. LAS accounts for 15-20% of injuries in the musculoskeletal system. LAS is seen in 50% of sports involving movements such as jumping, running, and cutting. While ATFL is injured in isolation in 65-73% of these injuries, ATFL and CFL are injured together in 25% of these injuries.[5]

FUNCTIONAL ANATOMY AND BIOMECHANICS OF THE FOOT-ANKLE COMPLEX

Ankle joint is composed of three joints: the talocrural joint, the subtalar joint and the distal tibiofibular joint.[6] Talocrural joint; the dome of the talus is formed by the union of the medial malleolus, lateral malleolus, and the lower surface of the tibia.[6] Due to its shape during walking, the talocrural joint is responsible for transferring the load by external/internal rotation of the tibia and fibula and pronation/supination of the bare foot. The axis of rotation of the talocrural joint passes through the medial and lateral malleolus.[6] The axis of rotation passes slightly behind the fibula as it passes slightly in front of the tibia in the frontal plane. While the dorsiflexion/plantar flexion movement, which is the main movement of the talocrural joint, occurs in the sagittal plane, a small movement occurs in this joint in the transverse and frontal planes.[7] Passive stabilization of the talocrural joint is provided by the joint capsule, ATFL, CFL, PTFL, and deltoid ligament.[2]

The ATFL is located on the dorsolateral aspect of the foot. It courses from the lateral malleolus anteriorly and medially to the talus at an angle of approximately 45 degrees in the frontal plane. ATFL averages 7.2 mm wide and 24.8 mm long.[8] In vitro kinematic studies have shown that ATFL

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prevents anterior displacement of the talus from the tenon and excessive inversion and internal rotation of the talus on the tibia.[9] The tension in the ATFL increases as the ankle moves from dorsiflexion to plantar flexion. Studies on the ATFL, PTFL, CFL, and deltoid ligament have shown that the ATFL has the least tensile and maximum load-bearing capacity. This explains why the ATFL is most often injured.[10]

The CFL runs downward and posteriorly from the lateral malleolus and attaches to the lateral calcaneus at an angle of approximately 133 degrees from the long axis of the fibula.[8] CFL restricts excessive supination of both the talocrural and subtalar joints. In vitro experiments have shown that CFL restricts excessive inversion and internal rotation of the bare foot and is most tense when the ankle is in dorsiflexion.[9] The CFL is the second most frequently injured ligament within the lateral talocrural ligaments.[9]

The PTFL runs posteriorly from the lateral malleolus and terminates in the posterolateral portion of the talus. The PTFL has wide endings on both the talus and the fibula. It limits both inversion and internal rotation of the talocrural joint during walking. PTFL is the least injured of the lateral ankle ligaments.[10]

The distal tibiofibular joint is formed between the distal ends of the tibia and fibula. This joint, in the form of syndesmosis, has limited range of motion between the two bones.[13] However, the limited gliding movement in this joint is important for the normal mechanics of the entire ankle complex. In the LAS, the normal position of the distal tibiofibular joint is impaired and therefore the ankle dorsiflexion movement is restricted.[11,12] Therefore, posterior and superior mobilizations to the fibula bone in the early and middle period of rehabilitation allow patients to relieve their symptoms.[13]

By contracting the muscles, they create stiffness in the muscle-tendon units and thus contribute to the dynamic stabilization of the joints. Muscles are responsible for releasing movement by concentric contraction, and by eccentric contraction, they make an important contribution to dynamic stabilization.[14,15] The peroneus longus and brevis muscles contract eccentrically to support the lateral ankle ligaments during supination of the bare foot. In addition, the tibialis anterior, extensor digitorum longus, extensor digitorum brevis and peroneus tertius muscles on the front of the leg contract eccentrically during supination of the hind foot and support the lateral ligaments. These muscles provide support by slowing the ankle plantar flexion movement, especially when the bare foot is in supination.[14,15]

INJURY MECHANISM

LAS most commonly occur due to excessive supination of the bare foot about an externally rotated tibia and fibula soon after initial contact of the bare foot during gait or landing from a jump.[16] Excessive inversion and internal rotation of the bare foot, coupled with external rotation of tibia and fibula, results in strain to the lateral ankle ligaments. If the strain in any of the ligaments exceeds the tensile strength of the tissues, ligamentous damage occurs. Increased plantar flexion at initial contact appears to increase the likelihood of suffering a lateral ankle sprain.[16] The ATFL is the first ligament to be damaged during a lateral ankle sprain, followed most often by the CFL. PTFL injury appears to be least common.[16]

After LAS, damage to the ligaments and sensory disturbances in the central nervous system occur.[17] Subsequently, changes in ligament structure and inhibition of spinal reflexes appear. The result is altered joint loading and altered normal movement patterns. Thus, the risk of osteoarthritis in the ankle increases.[17]

CLASSIFICATION OF LATERAL ANKLE SPRAIN

LAS can be classified according to the severity and duration of the injury. Traditionally, LAS are diagnosed by a grading system. Grade I LAS are the most common type of sprains, and patients with grade I LAS typically present with mild pain and swelling over the ATFL with no joint instability.[18] Patients with grade II LAS present with greater disability, moderate pain, and swelling over both the ATFL and the CFL. Furthermore, grade II LAS typically involve complete rupture of the ATFL and stretching of the CFL fibers. The most severe sprains are classified as grade III, but these are relatively uncommon in athletes and result in extreme disability. This type of injury involves damage to the ATFL, CFL, and PTFL.18 According to the duration of the injury, it is called acute ankle instability for 0-6 weeks, subacute for 6-12 weeks, and chronic ankle instability for 12 weeks and above.[5]

CHRONIC ANKLE INSTABILITY

Chronic ankle instability (CAI) is defined as a continuous ankle sprain and feeling of emptiness 1 year after the ankle injury. CAI is divided into functional and mechanical.[19] If anatomical changes occur in the first ankle injury, mechanical instability develops in the ankle. Mechanical instability with untreated injury leads to other changes. These changes are pathological laxity, deterioration in arthrokinematic movements, and degenerative joint damage. These changes can occur in isolation or in combination with each other.[18]

Injury to the lateral ligaments of the ankle results in adverse changes to the neuromuscular system that provides dynamic support to the ankle. Freeman et al.[20] first described the concept of functional instability in 1965. They attributed impaired balance in individuals with lateral ankle sprains to damaged articular mechanoreceptors in the lateral ankle ligaments, which resulted in proprioceptive deficits. The contribution of impaired proprioception, while important, does not fully explain why ankle-ligament injury predisposes athletes to functional ankle instability. The pathoetiologic model is not com plete without including impaired neuromuscular control, thus resulting in inadequacies of the dynamic defense mechanism protecting against hypersupination of the bare foot.[4]

CLINICAL EVALUATION

Usually following 3 tests are used in physical examination:

- Anterior drawer test
- Anterolateral drawer test
- Reverse anterolateral drawer test

Anterior Drawer Test

Musculoskeletal care providers routinely examine talocrural joint integrity with the anterior drawer test (ADT) to identify the severity of anterior talocrural joint laxity in the acute setting, to advance clinical progression during rehabilitation, or to assign subjects to a research group on the basis of those joint examinations. The ADT presumably can be used to detect the presence of injuries to the ATFL. The clinician performs the test by manually applying an anteriorly directed force at the calcaneus or a backward push on the tibia and attempting to discern pathological talocrural joint laxity from normal physiological laxity.[21] van Dijk et al. evaluated 160 inversion ankle injuries and reported a sensitivity of 71% and specificity of 33% in the diagnosis of ankle ligament tears when the physical exam was based on a cluster of signs and symptoms, such as the formation of a hematoma, location of pain, palpation, and the results of the ADT, with arthrography used as the gold standard.[22] In the ADT, when the ATFL is injured, the medial deltoid ligament can remain intact, which can lead to false negative results.[23]

Anterolateral Drawer Test

The test is performed in a sitting position with the calf hanging over the edge of the examination bed. While the tester stabilizes the tibia with one hand and pulls the calcaneus forward with the other hand while the ankle is plantar flexed, he tries to feel the forward displacement of the talus with his thumb. More specifically, the index and middle fingers are pressed firmly against the posterior aspect of the heel to provide the force directed forward. The palm supports the sole of the foot to provide 10°–15° plantar flexion and tighten the lateral ligaments. The thumb is placed on the relatively flat plane of the lateral surface of the anterior talar dome and 1 cm proximal to the tip of the anterior surface of the lateral malleolus. Evaluation is done as in ADT.[24]

Reverse Anterelateral Drawer Test

The examination is perform, while the patient was lying in the bed with the knee flexed and the angle of the knee adjusted to facilitate plantar flexion. The heel is completely pressed on the bed by the examiner with one hand after adjusting the ankle to a 10°–15° degree plantar flexion and unconstrained internal rotation. The index and middle fingers is place along the relatively smooth plane of the lateral aspect of the anterior talar dome and the anterior aspect of the lateral malleolus 1 cm proximal to its tip. The other hand hold the distal tibia, and the base of the palm pushed against the tibia to induce a posteriorly oriented displacement of the tibia with a force parallel with that of the articular surface arch of the talus. This test is more sensitive than ADT and ADLT.[25]

CLINICAL SYMPTOMS

Inflammation symptoms such as pain, edema, increased temperature, redness and loss of function occur after acute ankle injury. In CAI, the symptoms are divided into 4 main headings: i) limitation of movement, ii) loss of strength, iii) loss of balance, and iv) functional limitation.[26]

Range of Motion

Loss of range of motion (ROM) in LAS may occur due to arthrokinematic and osteokinematic reasons. ROM is the both a cause and a consequence of the LAS. It has been reported that patients with ankle dorsiflexion limitation of motion are more likely to have LAS.[26] In daily life, 10 degrees of passive ankle dorsiflexion movement is required for walking and going down stairs, and 20-30 degrees of active ankle dorsiflexion movement is required for running and sprinting.[27] People with dorsiflexion limitation of motion change their biomechanics to compensate for the loss of ROM. Thus, they become more vulnerable to injury. Due to pain and inflammation after acute LAS, patients can only do 7-8° dorsiflexion movements.[28] After inversion injury, the ATFL applies an inferior, anterior directional traction force to the fibula. This leads to biomechanical changes in the proximal and distal tibiofibular joints, resulting in loss of dorsiflexion range of motion.[29] At the same time, in chronic LAS, ankle dorsiflexion ROM decreases due to limitations in posterior talus sliding movements.[30,31]

Strength

Both concentric and eccentric strength losses are seen in CAI.[32] The ability of the skeletal muscle surrounding the ankle to provide dynamic support during joint decelerations and perturbations is important for normal ankle function and injury prevention.[32] Following an acute LAS, isometric eversion strength is reduced when compared bilaterally for up to 3 weeks following injury.[33,34] However, inversion strength deficiencies have also been described in the first few weeks following a LAS, and therefore both inverter and evertor muscle strength may be equally important in the early stages of LAS rehabilitation.[35] During early recovery, strength deficiencies can be predicted to be associated with previous deficiencies present before the initial injury, pain-related muscle inhibition and swelling from the acute inflammatory process, or acute stretching of the surrounding musculature. However, the long-term consequences of a LAS, including decreased physical activity, decreased ankle ROM, and altered motion patterns, potentially result in recurrent strength deficits in a subset of developing patients.[36] For this reason, atrophy that may occur can be prevented by isometric muscle strengthening in the acute period of the injury. Concentric, eccentric and pilometric strengthening should be emphasized in the middle and late periods.[18]

Postural Control

Individuals with LAS injury have decreased neuromuscular control and proprioception sense compared to healthy controls.[18] As the reason for the decrease in postural control; Inhibition of spinal reflexes due to disruption of sensory pathways in the central nervous system can be demonstrated.[37] Although there are differences in the physiological definitions of neuromuscular control and proprioception, both can be improved with balance exercises. After LAS, the static balance of the patients can be evaluated by standing on one leg, and the dynamic balance can be evaluated with the Star Excursion Balance Test.[38] In the literature, it is stated that balance exercises reduce the incidence of ankle sprain.[39] In rehabilitation after LAS, balance exercises should have an important place in improving neuromuscular control and proprioception sense. Balance exercises should be progressed with different methods such as from static to dynamic, from simple to difficult, from single task to double task, from fixed floor to moving floor.[37-39]

Functional Activity

Loss of functional activity in CAI occurs during walking, running, jumping and cutting movements. In the acute and chronic period after LAS, an increasing inversion pattern is observed in the ankle before the first contact in walking.[40] In addition, patients experience a decrease in plantar flexion ROM and increase in knee flexion ROM. The reason for this is that they feel safe by putting their ankle in the closed position.[41] Thus, especially in the acute phase of the injury, patients walk with shorter stride length, shorter single leg support time and reduced maximum power.[42] In the literature, it has been shown that gait training given to patients after LAS injury improves the gait of patients.[43] There are changes in the biomechanics of jumping and landing in patients after LAS.[36] Acute LAS patients decrease the flexor moment on the injured side and increase the extensor moment on the healthy side while jumping. This creates an asymmetry between the two sides. The reason for this is that they slow down the body and exhibit a protective mechanism. Similarly, protective reactions continue in the descent position and patients use increased hip flexion to reduce ground reaction.[44] In chronic LAS, patients use increased inversion and knee flexion while descending extra.[45]

BIOPSYCHOSOCIAL MODEL OF LATERAL ANKLE SPRAIN

If the LAS injury is not treated, CAI develops. Detailed biopsychosocial model has been stated by Hertel et al.[46] Primary tissue injury occurs after LAS. There is an effect of biopsychosocial factors in both recovery and chronicity of LAS. The biopsychosocial model of LAS includes pathomechanical disorders, sensory-perception disorders, and motor-behavioral disorders, and there are personal and environmental factors that affect them. While personal factors affecting the course of injury after LAS are demographic information, past medical history, physical attitude and psychological state, environmental factors are physical activity, work-home demands, social support and access to health care.[46] Evaluation and treatment of patients after chronic LAS within the framework of a biopsychosocial model may allow patients to fully recover.[47] Table 1 shows the biopsychosocial model of LAS.

Table 1. Biopsychosocial model of LAS

Subgroups of the related impairments		
Pathomechanical	**Sensory-Perceptual**	**Motor-Behavioral**
Pathologic laxity	Pain	Altered reflexes
Arthrokinematic restrictions	Diminished somasosensation	Nöromuskuler inhibition
Osteokinematic restrictions	Perceived instability	Muscle weakness
Secondary tissue injury	Kinesiophobia	Balance deficit
Tissue adaptations	Lower self-reported funciton	Altered movements patterns
	Reduced quality of life	Reduced physical activity

REHABILITATION

LAS injuries are treated in 3 phases: acute, subacute and chronic.

Acute Phase

During this period, which covers the first 4 weeks, the patient has pain, edema, and limitation of movement. This period is important to prevent the disease from becoming chronic and to reduce functional losses. Our aim in this phase is to restore the ankle joint and soft tissue, to prevent muscle imbalance, to increase proprioceptive sensation and to provide normal ROM (Table 2).[18]

Therefore, POLICE (Protect, Optimal Loading, Ice, Compression, Elevation)[48] application should be started immediately after injury. Ice application can be applied to the area where the lateral ligaments are located for 12-15 minutes either as a fixed or as an ice massage every 2 hours. In Grade II and III injuries, brace, rigid taping and kinesiotaping can be used for immobilization if necessary.[18,49]

Due to the deterioration of the biomechanics of the distal tibiofibular joint and talocrural joint, limitations in ankle dorsiflexion movement may develop. Therefore, the anterior-posterior mobilization of the talocrural joint and the upward and posterior mobilization of the distal talofibular joint will increase the ankle dorsiflexion movement. This reason, mobilization exercises for ATFL injuries are important in the restoration of ROM. Green et al.[29] reported that the anterior and posterior mobilization of the talus increases painless dorsiflexion movement in the acute period ATFL sprain. In subacute and chronic period, it has been reported that there is a discernable treatment effect on ankle ROM and pain with early mobilization with movement.[13,50,51] When edema and pain disappear, Phase 2 is started.[52]

Table 2. Rehabilitation techniques in the acute period
POLICE
Active dorsiflexion-plantar flexion movements of the ankle
4-way isometric muscle strengthening to the muscles around the ankle
4-way isotonic strengthening of the muscles around the hip area
TENS, NMES
In-water proprioceptive applications
Posterior mobilization of the talocrural joint
Distal talofibular joint mobilization with movement
Cardiovascular training with stationary bike
Lumbopelvic region exercises

TENS: Transcutaneous electrical nerve stimulation, NMES: neuromuscular electrical stimulation

Subacute Phase

This period, when the patient has no or minimal pain and edema, covers 3-6 weeks. The subacute phase begins when the patient begins to tolerate weight bearing. Neuromuscular training and balance exercises are emphasized for the provision of impaired proprioceptive sense, muscle hypertrophy and proprioceptive sense. Mobilizations are continued to increase the ankle dorsiflexion ROM. Balance exercises should be given progression such as double-leg-single-leg, fixed surface-moving surface, eyes open-closed. Wobble board exercises and stretching exercises for the gastrosoleus muscle are performed on the incline board. Initially, the focus should be on dorsiflexion and plantar flexion. When the ligament healing is completed, first active movement and then resistant movements are started in inversion and eversion movements.

Elastic bandage is recommended for foot exercises. After single-planned movements, exercises should also be performed in the diagonal plane (inversion/eversion). During this period, clinicians should perform exercises aimed at sitting and correct walking biomechanics. When full ROM without pain is achieved, phase 3 is initiated when the muscle strength reaches 80-90% of the contralateral side (Table 3).[52]

Table 3. Rehabilitation techniques in the subacute period

Ankle resistant dorsiflexion-plantar flexion motion
Active eversion-inversion motion of the ankle
Resisted eversion-inversion movement of the ankle [If there is no pain in active movement, VAS<3 in resistance exercise (concentric first / then eccentric)]
4-way isotonic strengthening of the muscles around the hip area
NMES
Proprioceptive applications (Must be progressive)
Mobilization techniques for the talocrural and distal talofibular joint
Heel-raise
Cardiovascular training with stationary bike
Walking training
Balance exercises (must be progressed) Core stabilization exercises

NMES: Neuromuscular electrical stimulation

Chronic Phase

During this period when the patient's symptoms are not relieved, muscle strengthening, increasing proprioceptive, coordination, correction of walking and running biomechanics, and advanced exercises are included. It is continued until the 3rd month. After the 3rd month, sports-specific movements are started (Table 4).

Table 4. Rehabilitation techniques in the chronic period

Advanced proprioceptive exercises
Advanced balance exercises
Exercises for resistant (concentric/eccentric) lower extremity muscles
Plyometric exercises
Improvement of walking, running, jumping, cutting biomechanics
Aerobic exercises
Sport-specific movements
Core stabilization exercises

CURRENT CRITERIAS FOR RETURN TO PLAY AFTER LAS

Due to the high re-injury rate after rehabilitation, the time to return to sports and recreational activities should be well planned. There are no common criterias for returning to sports after LAS in the literature. Although there are different opinions for the criteria for returning to sports after LAS, the criteria in Table 4 as a result of the literature review can be used to decide on returning to sports. However, further studies are needed.

CONCLUSION

LAS, which is common in recreational and professional athletes, recurs and becomes chronic if left untreated. Since the number of mechanoreceptors it contains is high, LAS should not be considered only as a musculoskeletal disorder. A detailed clinical evaluation is recommended. Clinically, the patient presents with limitation of dorsiflexion ROM, muscle atrophy and unequal strength between the muscles, deterioration in walking, running and jumping, and proprioceptive losses. The patient should be approached from a biopsychosocial perspective both in the evaluation and treatment phases. The treatment of the patient is carried out in 3 phases and the transition between phases should be based on mentioned criterias. In the acute phase of rehabilitation, emphasis should be placed on eliminating pain and edema, and preventing loss of ROM and muscle strength. In the subacute and chronic stages, emphasis should be placed on increasing the proprioceptive sense and muscle strength, improving the biomechanics of daily life and sports-specific movements such as walking, running, jumping. If the patient is an athlete, return to sports should be planned after the criterias met.

ETHICAL DECLARATIONS

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Parameter	Evaluation criteria	Criterion
Pain/edema	VAS NPRS Foot and Ankle Index	Free of pain and swelling
ROM	Goniometric Weight bearing lunge test	Full ROM
Strength	Handheld dynamometry Standing jump	A return to normal strength 90% of uninjured limb strength
Static balance	Single-leg balance test Balance error system	Ability to balance without pain Maintain single-limb stance for 10 s
Dynamic balance	SEBT Y balance test	Compare with the uninjured limb 80% of uninjured limb reach Balance test with little to no pain
Running	Run program Running technique	Little to no pain while running Run at max speed without pain
Sports-specific movements	Lateral hopping Vertical hopping Shuttle run Sport-specific test Jumping and cutting Agility T test	80% of the uninjured leg amount Consistent T-test times between 8.9 and 13.5 s Speed and quality movement during sport-specific test
Patient-based reports	Foot and ankle ability measure Lower-extremity function scale Lower limb task questionnaire Injury-psychological readiness to return sport scale	Athlete feels confident The athlete is ready to return

VAS: Visual analog scale, NPRS: Numeric pain rating scale, SEBT: Star excursion balance test

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REFERENCES

1. McKeon PO, Hertel J, Bramble D, Davis I. The foot core system: a new paradigm for understanding intrinsic foot muscle function. *Br J Sports Med.* 2015;49(5):290. doi:10.1136/bjsports-2013-092690
2. Renström PA, Konradsen L. Ankle ligament injuries. *Br J Sports Med.* 1997;31(1):11-20. doi:10.1136/bjsm.31.1.11
3. Hupperets MD, Verhagen EA, van Mechelen W. Effect of sensorimotor training on morphological, neurophysiological and functional characteristics of the ankle: a critical review. *Sports Med.* 2009;39(7):591-605. doi:10.2165/00007256-200939070-00005
4. Hertel J. Sensorimotor deficits with ankle sprains and chronic ankle instability. *Clin Sports Med.* 2008;27(3):353-vii. doi:10.1016/j.csm.2008.03.006
5. Weerasekara RM, Tennakoon SU, Suraweera HJ. Contrast therapy and heat therapy in subacute stage of grade I and II lateral ankle sprains. *Foot Ankle Spec.* 2016;9(4):307-323. doi:10.1177/1938640016640885
6. Huson A. Joints and movements of the foot: terminology and concepts. *Acta Morphol Neerl Scand.* 1987;25(3):117-130.
7. Lundberg A, Goldie I, Kalin B, Selvik G. Kinematics of the ankle/foot complex: plantarflexion and dorsiflexion. *Foot Ankle.* 1989;9(4):194-200. doi:10.1177/107110078900900409
8. Burks RT, Morgan J. Anatomy of the lateral ankle ligaments. *Am J Sports Med.* 1994;22(1):72-77. doi:10.1177/036354659402200113
9. Cass JR, Settles H. Ankle instability: in vitro kinematics in response to axial load. *Foot Ankle Int.* 1994;15(3):134-140. doi:10.1177/107110079401500308
10. Hertel J. Functional Anatomy, Pathomechanics, and Pathophysiology of Lateral Ankle Instability. *J Athl Train.* 2002;37(4):364-375.
11. Kavanagh J. Is there a positional fault at the inferior tibiofibular joint in patients with acute or chronic ankle sprains compared to normals?. *Man Ther.* 1999;4(1):19-24. doi:10.1016/s1356-689x(99)80005-8
12. Denegar CR, Miller SJ 3rd. Can chronic ankle instability be prevented? rethinking management of lateral ankle sprains. *J Athl Train.* 2002;37(4):430-435.
13. Mulligan B. Manual therapy "NAGS","SNAGS","MWMS" etc. plane view services. Wellingt New Zeal. Published online 1995.
14. Ashton-Miller JA, Ottaviani RA, Hutchinson C, Wojtys EM. What best protects the inverted weightbearing ankle against further inversion? Evertor muscle strength compares favorably with shoe height, athletic tape, and three orthoses. *Am J Sports Med.* 1996;24(6):800-809. doi:10.1177/036354659602400616
15. Sinkjaer T, Toft E, Andreassen S, Hornemann BC. Muscle stiffness in human ankle dorsiflexors: intrinsic and reflex components. *J Neurophysiol.* 1988;60(3):1110-1121. doi:10.1152/jn.1988.60.3.1110
16. van den Bekerom MP, Kerkhoffs GM, McCollum GA, Calder JD, van Dijk CN. Management of acute lateral ankle ligament injury in the athlete. *Knee Surg Sports Traumatol Arthrosc.* 2013;21(6):1390-1395. doi:10.1007/s00167-012-2252-7
17. Wikstrom EA, Hubbard-Turner T, McKeon PO. Understanding and treating lateral ankle sprains and their consequences: a constraints-based approach. *Sports Med.* 2013;43(6):385-393. doi:10.1007/s40279-013-0043-z
18. Donovan L, Hertel J. A new paradigm for rehabilitation of patients with chronic ankle instability. *Phys Sportsmed.* 2012;40(4):41-51. doi:10.3810/psm.2012.11.1987
19. McCriskin BJ, Cameron KL, Orr JD, Waterman BR. Management and prevention of acute and chronic lateral ankle instability in athletic patient populations. *World J Orthop.* 2015;6(2):161-171. Published 2015 Mar 18. doi:10.5312/wjo.v6.i2.161
20. Freeman MA. Instability of the foot after injuries to the lateral ligament of the ankle. *J Bone Joint Surg Br.* 1965;47(4):669-677.
21. Croy T, Koppenhaver S, Saliba S, Hertel J. Anterior talocrural joint laxity: diagnostic accuracy of the anterior drawer test of the ankle. *J Orthop Sports Phys Ther.* 2013;43(12):911-919. doi:10.2519/jospt.2013.4679
22. van Dijk CN, Mol BW, Lim LS, Marti RK, Bossuyt PM. Diagnosis of ligament rupture of the ankle joint. Physical examination, arthrography, stress radiography and sonography compared in 160 patients after inversion trauma. *Acta Orthop Scand.* 1996;67(6):566-570. doi:10.3109/17453679608997757
23. Lähde S, Putkonen M, Puranen J, Raatikainen T. Examination of the sprained ankle: anterior drawer test or arthrography?. *Eur J Radiol.* 1988;8(4):255-257.
24. Phisitkul P, Chaichankul C, Sripongsai R, Prasitdamrong I, Tengtrakulcharoen P, Suarchawaratana S. Accuracy of anterolateral drawer test in lateral ankle instability: a cadaveric study. *Foot Ankle Int.* 2009;30(7):690-695. doi:10.3113/FAI.2009.0690
25. Li Q, Tu Y, Chen J, et al. Reverse anterolateral drawer test is more sensitive and accurate for diagnosing chronic anterior talofibular ligament injury. *Knee Surg Sports Traumatol Arthrosc.* 2020;28(1):55-62. doi:10.1007/s00167-019-05705-x
26. Gribble PA, Hertel J, Denegar CR, Buckley WE. The Effects of Fatigue and Chronic Ankle Instability on Dynamic Postural Control. *J Athl Train.* 2004;39(4):321-329.
27. Harris GF. Analysis of ankle and subtalar motion during human locomotion. *Inman's Joints of the Ankle.* 1991;2:75-84.
28. Youdas JW, McLean TJ, Krause DA, Hollman JH. Changes in active ankle dorsiflexion range of motion after acute inversion ankle sprain. *J Sport Rehabil.* 2009;18(3):358-374. doi:10.1123/jsr.18.3.358
29. Green T, Refshauge K, Crosbie J, Adams R. A randomized controlled trial of a passive accessory joint mobilization on acute ankle inversion sprains. *Phys Ther.* 2001;81(4):984-994.
30. Denegar CR, Hertel J, Fonseca J. The effect of lateral ankle sprain on dorsiflexion range of motion, posterior talar glide, and joint laxity. *J Orthop Sports Phys Ther.* 2002;32(4):166-173. doi:10.2519/jospt.2002.32.4.166
31. Vicenzino B, Branjerdporn M, Teys P, Jordan K. Initial changes in posterior talar glide and dorsiflexion of the ankle after mobilization with movement in individuals with recurrent ankle sprain. *J Orthop Sports Phys Ther.* 2006;36(7):464-471. doi:10.2519/jospt.2006.2265
32. Munn J, Beard DJ, Refshauge KM, Lee RY. Eccentric muscle strength in functional ankle instability. *Med Sci Sports Exerc.* 2003;35(2):245-250. doi:10.1249/01.MSS.0000048724.74659.9F
33. Konradsen L, Olesen S, Hansen HM. Ankle sensorimotor control and eversion strength after acute ankle inversion injuries. *Am J Sports Med.* 1998;26(1):72-77. doi:10.1177/03635465980260013001
34. Kobayashi T, Gamada K. Lateral ankle sprain and chronic ankle instability: a critical review. *Foot Ankle Spec.* 2014;7(4):298-326. doi:10.1177/1938640014539813
35. Wilkerson GB, Pinerola JJ, Caturano RW. Invertor vs. evertor peak torque and power deficiencies associated with lateral ankle ligament injury. *J Orthop Sports Phys Ther.* 1997;26(2):78-86. doi:10.2519/jospt.1997.26.2.78
36. Miklovic TM, Donovan L, Protzuk OA, Kang MS, Feger MA. Acute lateral ankle sprain to chronic ankle instability: a pathway of dysfunction. *Phys Sportsmed.* 2018;46(1):116-122. doi:10.1080/00913847.2018.1409604
37. McKeon PO, Hubbard TJ, Wikstrom EA. Consequences of Ankle Inversion Trauma: A Novel Recognition and Treatment Paradigm. INTECH Open Access Publisher; 2012.
38. Nakagawa L, Hoffman M. Performance in static, dynamic, and clinical tests of postural control in individuals with recurrent ankle sprains. *J Sport Rehabil.* 2004;13(3):255-268. doi:10.1123/jsr.13.3.255
39. de Vries JS, Krips R, Sierevelt IN, Blankevoort L, van Dijk CN. Interventions for treating chronic ankle instability. *Cochrane Database Syst Rev.* 2011;(8):CD004124. doi:10.1002/14651858.CD004124.pub3
40. Doherty C, Bleakley C, Hertel J, Caulfield B, Ryan J, Delahunt E. Lower extremity function during gait in participants with first time acute lateral ankle sprain compared to controls. *J Electromyogr Kinesiol.* 2015;25(1):182-192. doi:10.1016/j.jelekin.2014.09.004
41. Delahunt E, Monaghan K, Caulfield B. Altered neuromuscular control and ankle joint kinematics during walking in subjects with functional instability of the ankle joint. *Am J Sports Med.* 2006;34(12):1970-1976. doi:10.1177/0363546506290989
42. Punt I, Ziltener JL, Laidet M, Armand S, Allet L. Functional deficits in acute lateral ankle sprain patients. *Gait Posture.* 2014;(39):S19-S20. DOI:10.1016/j.gaitpost.2014.04.029
43. Feger MA, Hart JM, Saliba S, Abel MF, Hertel J. Gait training for chronic ankle instability improves neuromechanics during walking. *J Orthop Res.* 2018;36(1):515-524. doi:10.1002/jor.23639
44. Doherty C, Bleakley C, Hertel J, Caulfield B, Ryan J, Delahunt E. Single-leg drop landing motor control strategies following acute ankle sprain injury. *Scand J Med Sci Sports.* 2015;25(4):525-533. doi:10.1111/sms.12282
45. Caulfield BM, Garrett M. Functional instability of the ankle: differences in patterns of ankle and knee movement prior to and post landing in a single leg jump. *Int J Sports Med.* 2002;23(1):64-68. doi:10.1055/s-2002-19272
46. Hertel J, Corbett RO. An updated model of chronic ankle instability. *J Athl Train.* 2019;54(6):572-588. doi:10.4085/1062-6050-344-18
47. Engel GL. The need for a new medical model: a challenge for biomedicine. *Science.* 1977;196(4286):129-136. doi:10.1126/science.847460
48. Bleakley CM, Glasgow P, MacAuley DC. PRICE needs updating, should we call the POLICE?. *Br J Sports Med.* 2012;46(4):220-221. doi:10.1136/bjsports-2011-090297

49. Chen ET, Borg-Stein J, McInnis KC. Ankle Sprains: Evaluation, Rehabilitation, and Prevention [published correction appears in Curr Sports Med Rep. 2019 Aug;18(8):310]. *Curr Sports Med Rep.* 2019;18(6):217-223. doi:10.1249/JSR.0000000000000603
50. Hudson R, Baker RT, May J, Reordan D, Nasypany A. Novel treatment of lateral ankle sprains using the Mulligan concept: an exploratory case series analysis. *J Man Manip Ther.* 2017;25(5):251-259. doi:10.1080/10669817.2017.1332557
51. Weerasekara I, Deam H, Bamborough N, et al. Effect of Mobilisation with Movement (MWM) on clinical outcomes in lateral ankle sprains: A systematic review and meta-analysis. *Foot (Edinb).* 2020;43:101657. doi:10.1016/j.foot.2019.101657
52. Chinn L, Hertel J. Rehabilitation of ankle and foot injuries in athletes. *Clin Sports Med.* 2010;29(1):157-167. doi:10.1016/j.csm.2009.09.006
53. Alahmari KA, Silvian P, Ahmad I, et al. Effectiveness of low-frequency stimulation in proprioceptive neuromuscular facilitation techniques for post ankle sprain balance and proprioception in adults: a randomized controlled trial. *Biomed Res Int.* 2020;2020:9012930. doi:10.1155/2020/9012930
54. Dastmanesh S, Shojaedin SS, Eskandari E. The effects of core stabilization training on postural control of subjects with chronic ankle instability. *Ann Biol Res.* 2012;3(8):3926-3930.
55. Rivera MJ, Winkelmann ZK, Powden CJ, Games KE. Proprioceptive training for the prevention of ankle sprains: an evidence-based review. *J Athl Train.* 2017;52(11):1065-1067. doi:10.4085/1062-6050-52.11.16
56. Ismail MM, Ibrahim MM, Youssef EF, El Shorbagy KM. Plyometric training versus resistive exercises after acute lateral ankle sprain. *Foot Ankle Int.* 2010;31(6):523-530. doi:10.3113/FAI.2010.0523
57. Tassignon B, Verschueren J, Delahunt E, et al. Criteria-Based Return to Sport Decision-Making Following Lateral Ankle Sprain Injury: a Systematic Review and Narrative Synthesis. *Sports Med.* 2019;49(4):601-619. doi:10.1007/s40279-019-01071-3
58. Wikstrom EA, Mueller C, Cain MS. Lack of Consensus on Return-to-Sport Criteria Following Lateral Ankle Sprain: A Systematic Review of Expert Opinions. *J Sport Rehabil.* 2020;29(2):231-237. doi:10.1123/jsr.2019-0038
59. Smith MD, Vicenzino B, Bahr R, et al. Return to sport decisions after an acute lateral ankle sprain injury: introducing the PAASS framework-an international multidisciplinary consensus. *Br J Sports Med.* 2021;55(22):1270-1276. doi:10.1136/bjsports-2021-104087
60. Cross KM, Worrell TW, Leslie JE, Van Veld KR. The relationship between self-reported and clinical measures and the number of days to return to sport following acute lateral ankle sprains. *J Orthop Sports Phys Ther.* 2002;32(1):16-23. doi:10.2519/jospt.2002.32.1.16

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Case Report

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Bilateral capitellar fracture's: a case report

Levent Horoz, Mehmet Fevzi Çakmak

Department of Orthopedics and Traumatology, Faculty of Medicine, Ahi Evran University, Kırşehir, Turkey

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Corresponding Author: Levent Horoz, dr.leventhoroz@gmail.com

ABSTRACT

Bilateral capitellar fractures are very rare injuries. If the anatomy is not restored properly, limitation in elbow movements and permanent disability are inevitable. Fixation of the capittal fracture to allow early movement is important. In prolonged immobilization, limitation of movement is encountered. We present a case of a 52 years old women affected by a shear fracture of a capitellum after fall from chair. Open reduction and internal fixation were performed for both capitellum fractures. Early rehabilitation was initiated to prevent joint motion contracture. As a result of the long-term follow-up of the patient, no limitations were observed in the activities of daily living. We suggest early rehabilitation following open reduction and internal fixation in patients admitted to us with capitellum fracture.

Keywords: Capitellum fracture, open reduction, headless screw, early rehabilitation

INTRODUCTION

Capitellar fractures are rare injury's.[1] They account only %1 of all elbow fracture's (isolaterd fracture of capitellum). These fracture often can occur after fall from outstretched hand.[2] The mechanism of the fracture is axial loading force admitted radial head to capitellum humeri. The clasification system most commonly used for capitellar fracture is Byran-Morrey.[3] There are three type of capitellar fracture has been described by Byran and Morrey. Type 1 fractures are coronal shear fractures that include most of the capitellum fractures. As a importance of the capitellum anatomic reduction and proper fixation is necessary to allow early elbow range of motion and preserve elbow from arthritis (fracture of the capitellum) There is a few bilateral capitellar fracture has been published in the literature yet.[1,4-6]

CASE REPORT

We report case 52 years old female who felt from chain on his out stretched hands. First examination; lateral sided elbow pain at both elbow, swelling and dermabrasion. Pain was triggered with motion. Bilateral elbow had range of motion restriction. Patient didn't fully extend his elbow both pasifly and actively. There was no laxity on both elbow. No neurovascular injury was noted during the administration to the emergency room (ER). We confirmed bilateral Type 1 hahn- steinthal capitellum fracture both on radiographs and CT scan (**Figure 1**).

A posterolateral Kocher approach was applied for anatomic reduction and stable fixation. Stable fixation maintained with two headless screw in convergent configuration, though to Posterior anterior direction and additional one screw at different direction to improve fixation strength (**Figure 2**). Before the definitive fixation, temporary k wire used to facilitate reduction. No early postoperative complication was reported.

Figure 1. Preoperative Xray and CT scan views

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Figure 2. Postoperative right and left elbow X-rays

Both elbows were immobilized for two weeks, and early intensive rehabilitation started after cast removal to preserve joint from stiffness. On the 12 week both fracture's healed without any reduction loses or implant failure. Trabecular unification presented at 12 weeks on control radiographs (Figure 3).

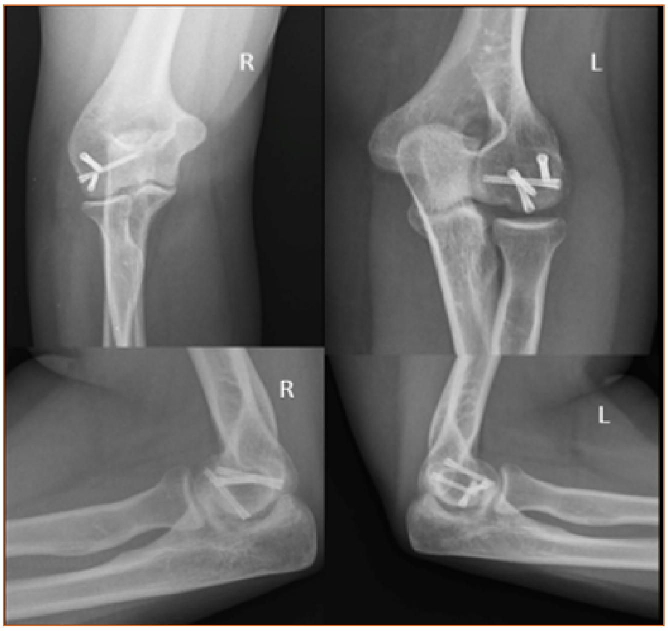

Figure 3. Postoperative 12. Week control X-ray

At final fallow up 6 moths postoperatively her both elbows had residual flexion contracture 5 degrees at both side, there was no flexion restriction on both side, there was no pain at elbows during the Daily activity she had satisfactory outcome from surgery (Figure 4).


Figure 4. Postoperative 8 month range of motion

DISCUSSION

Bilateral capitellum fractures are rarely encountered in daily orthopedic practice. Few cases of bilateral capitellar fractures have been reported in the literature. Capitellum fracture is relatively rare injury that can occur female patient more often due to superior carrying angle of elbow (hanh steinhanh fracture report of two cases)(fracture of the capitellum and trohlea) Type 1 hahn- steinthal fracture is the most common type that produced by axial loading on the outstretched hand.[7] If surgical treatment isn't performed precisely, displaced capitellum fracture block motion both flexion and extension.[7] Considering the cases treated for bilateral capitellar fractures, it was observed that the results were not satisfactory if anatomical reduction and early mobilization could not be achieved in cases followed up with closed reduction and plaster cast.[1] There are several treatment option for capitellar fracture such as; open reduction and internal fixation, fragment excision, closed reduction and cast immobilization, arthroplasty.[8,9] Few studies in literature have reported about fragment excision, according to this studies result of the fragment excision much similar to radial head. Short and mid-term results are acceptable. Fragment excision can be treatment option. If the fracture that are not amenable for open reduction and internal fixation.[10] It has been shown that poor results are obtained as a result of k-wire applications for capitellar shear fractures. The reason for this was that the k-wire was insufficient for fixation and prolonged immobilization was observed.[4]

Most of the studies advocate open reduction and stable fixation for large capitellar fracture. We prefer headless compression screw placement in posterior to anterior direction and additional screws at different direction to improve fixation strength. Better functional results with only one direction (posterior-anterior) screw placement has mentioned before at literature.[11] Mighell et al.[12] argued that when posterior- anterior direction prefer, it can disturb circulation of the capitellum and it can cause AVN. We didn't observe any AVN findings at our patient. Screw placement at anterior-posterior direction has been shown biomechanically superior to posterior-anterior direction.[13] In our patient we haven't seen any reduction lose or implant failure at fallow ups. We haven't seen any additional screw related complication. Ruchelsman et al.[14] recommends fixation with 2 screws in divergent direction in order to avoid iatrogenic fracture and to provide adequate fixation. In order to avoid iatrogenic fracture, attention should be paid to the distance between the two screws.[12] In our case, no iatrogenic fracture associated with the application of 3 screws was encountered. Additional plate fixation is recommended for more complex fracture patterns. In more complex fracture patterns, additional plate fixation is recommended in cases where the posterior cortex is not intact.[15] There are studies showing that the use of biodegradable screws for capitellar shear fractures gives good clinical results.[16] In addition, arthroscopic approaches for capitellum fracture fixation have been described and good results have been reported.[17]

Limitation of the elbow range of motion is the most common complication of the capitellar fracture 3. Other common complication of capetellum fracture is arthritis. Especially after joint incongruity after inadequate reduction.[18] Arthritis range from %11-51 after capitellum fractures.[18] We havent seen any arthrosis finding at our fallow-ups for 2 years. Early range of motion seem to be the key to successful outcome. Early mobilization of the elbow can prevent elbow from joint stiffness.[7,19] Anatomic reduction, stable fixation and early mobilization is the key success of the capitellar shear fractures.

CONCLUSION

In the surgical treatment of capitellum fractures, attention should be paid to anatomical reduction and stable fixation. Early movement and rehabilitation can prevent joint movement limitations. Fixation of capitellum fractures with headless screw allows early rehabilitation and joint movement.

ETHICAL DECLARATIONS

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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REFERENCES

1. Schindler OS. Bilateral capitellum humeri fracture: a case report and review of the literature. *J Orthop Surg (Hong Kong).* 2003;11(2):207-212. doi:10.1177/230949900301100218
2. Amis AA, Miller JH. The mechanisms of elbow fractures: an investigation using impact tests in vitro. *Injury.* 1995;26(3):163-168. doi:10.1016/0020-1383(95)93494-3
3. Rausch V, Königshausen M, Schildhauer TA, Gessmann J, Seybold D. Fractures of the capitellum humeri and their associated injuries. *Obere Extrem.* 2018;13(1):33-37. doi:10.1007/s11678-018-0441-9
4. Polat O, Arikan M, Güngör S, Karatas M. Bilateral capitellum humeri fracture; a case report. *Acta Chir Belg.* 2009;109(5):647-650. doi:10.1080/00015458.2009.11680508
5. Zaddoug O, Benchakroun M, Lazrak K. Fracture bilatérale du capitulum, à propos d'un cas avec revue de la littérature [Bilateral capitulum humeri fracture, a case report and review of the literature]. *Chir Main.* 2006;25(3-4):159-162. doi:10.1016/j.main.2006.04.004
6. Are A, Tornatore I, Theodorakis E. Operative management of a shear fracture of the bilateral capitellum: A case report and review of the literature. *Chin J Traumatol.* 2016;19(4):231-234. doi:10.1016/j.cjtee.2015.11.017
7. Dubberley JH, Faber KJ, Macdermid JC, Patterson SD, King GJ. Outcome after open reduction and internal fixation of capitellar and trochlear fractures. *J Bone Joint Surg Am.* 2006;88(1):46-54. doi:10.2106/JBJS.D.02954
8. Cutbush K, Andrews S, Siddiqui N, Brown LM, Ross M. Capitellar fractures-is open reduction and internal fixation necessary?. *J Orthop Trauma.* 2015;29(1):50-53. doi:10.1097/BOT.0000000000000148
9. Chang N, King GJW. Elbow Hemiarthroplasty for the Treatment of Distal Humerus Fractures. *Orthop Clin North Am.* 2020;51(2):265-277. doi:10.1016/j.ocl.2019.11.009
10. Garner MR, Schottel PC, Hotchkiss RN, Daluiski A, Lorich DG. Capitellum fracture fragment excision: a case series. *HSS J.* 2015;11(3):204-208. doi:10.1007/s11420-015-9452-x
11. Lopiz Y, Rodríguez-González A, García-Fernández C, Marco F. Open reduction and internal fixation of coronal fractures of the capitellum in patients older than 65 years. *J Shoulder Elbow Surg.* 2016;25(3):369-375. doi:10.1016/j.jse.2015.12.004
12. Mighell M, Virani NA, Shannon R, Echols EL Jr, Badman BL, Keating CJ. Large coronal shear fractures of the capitellum and trochlea treated with headless compression screws. *J Shoulder Elbow Surg.* 2010;19(1):38-45. doi:10.1016/j.jse.2009.05.012
13. Elkowitz SJ, Polatsch DB, Egol KA, Kummer FJ, Koval KJ. Capitellum fractures: a biomechanical evaluation of three fixation methods. *J Orthop Trauma.* 2002;16(7):503-506. doi:10.1097/00005131-200208000-00009
14. Ruchelsman DE, Tejwani NC, Kwon YW, Egol KA. Coronal plane partial articular fractures of the distal humerus: current concepts in management. *J Am Acad Orthop Surg.* 2008;16(12):716-728. doi:10.5435/00124635-200812000-00004
15. Wang P, Kandemir U, Zhang K, et al. Treatment of capitellar and trochlear fractures with posterior comminution: minimum 2-year follow-up. *J Shoulder Elbow Surg.* 2019;28(5):931-938. doi:10.1016/j.jse.2018.09.004
16. Kramer M, Pamnani A, Strauch RJ. Internal fixation of a capitellum fracture using a biodegradable screw. *Orthopedics.* 2002;25(7):765-766. doi:10.3928/0147-7447-20020701-20
17. Yari SS, Bowers NL, Craig MA, Reichel LM. Management of distal humeral coronal shear fractures. *World J Clin Cases.* 2015;3(5):405-417. doi:10.12998/wjcc.v3.i5.405
18. Guitton TG, Zurakowski D, van Dijk NC, Ring D. Incidence and risk factors for the development of radiographic arthrosis after traumatic elbow injuries. *J Hand Surg Am.* 2010;35(12):1976-1980. doi:10.1016/j.jhsa.2010.08.010
19. Heck S, Zilleken C, Pennig D, Koslowsky TC. Reconstruction of radial capitellar fractures using fine-threaded implants (FFS). *Injury.* 2012;43(2):164-168. doi:10.1016/j.injury.2011.04.009