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Comorbidity of lipedema and fibromyalgia; effects on disease severity, pain and health-related quality of life

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ABSTRACT

Aims: Both Fibromyalgia (FMS) and lipedema are characterized by pain in the soft tissue, and they have clinically similar aspects. The aims of this study were to determine how many of the patients with lipedema met the diagnostic criteria for FMS, the effect of the comorbidity of lipedema and FMS on pain and quality of life, and their relationship with extremity volumes, ultrasonographically measured soft tissue thickness and lipedema disease severity.

Methods: 53 women with lipedema and 32 patients with FMS without lipedema were included in the study. Symptom severity scale, widespread pain index, and FMS severity scale were calculated for the diagnosis of FMS. Pain intensity was determined by visual analog scale (VAS). The frequency of fibromyalgia was determined in the lipedema group. Lower extremity volumes of both groups were calculated by circumferential measurements and thigh and pretibial soft tissue thicknesses were measured ultrasonographically. Short form-36 quality of life scale was applied to both groups.

Results: The mean age of the 53 females with lipedema was 52±11.8 years, and for the 32 females with FMS it was 51.9±10.1 years ($p>0.05$). The extremity volumes and soft tissue thicknesses were higher in lipedema group than FMS group $p<0.001$).In lipedema group, 21(39.6%) patients have fulfilled the FMS criteria. FMS severity scores of Comorbid Lipedema and FMS group were similar with FMS patients ($p=0.199$). Bodily pain and VAS were more severe in Comorbid Lipedema and FMS group than lipedema group without FMS and FMS group ($p<0.001$). Generally, Short form-36 components were better in lipedema without FMS group than Comorbid FMS and FMS group ($p<0.05$)

Conclusion: The comorbidity of these two diseases in patients negatively affect their physical and mental functions. Investigation and treatment of comorbid FMS in lipedema patients may contribute to their quality of life and pain.

Keywords: Lipedema, fibromyalgia, soft tissue thickness, ultrasonography

INTRODUCTION

Lipedema is a disease that affects almost exclusively women and is characterized by a disproportionate distribution of abnormal adipose tissue between the extremities and trunk. Edema aggravated by orthostasis, easy bruising by minor trauma, increased sensitivity to pressure, and spontaneous pain are present in most patients. Its onset is usually during periods of hormonal changes such as puberty, pregnancy or menopause. There are no large epidemiological studies to determine the prevalence, but it is estimated to be about 0.1–9.7%.[1]

Fibromyalgia (FMS) is a syndrome that greatly affects quality of life and is characterized by chronic widespread pain, sleep disturbance, fatigue and cognitive impairment. Its estimated prevalence is 2.7%. It is the most important differential diagnosis of chronic soft tissue pain in clinical practice.[2]

FMS and lipedema have many demographic and clinical similarities including widespread pain and obesity.[2-4] Lipedema and FMS both have specific diagnostic criteria and although the diagnosis of lipoedema in stages 2 and 3 is not difficult, it may not be possible to distinguish stage 1 lipedema from FMS, because in stage 1 lipoedema, the skin surface is smooth and the subcutaneous fat tissue thickness is less.[4] These two chronic soft tissue pain syndromes may therefore coexist and be difficult to distinguish. However a few data are available on the frequency of FMS in lipedema patients. Angst et al.[4] showed that %34 lipedema patients have fulfilled American College of Rheumatology (ACR) 2016 FMS criteria.[5]

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Pain is the major complaint in lipoedema, impairing quality of life and correlated with depression. Reducing pain in lipoedema is one of the most important goals of treatment.[6] It is important to distinguish lipedema from FMS and from other chronic pain syndromes. Conservative treatment of lipedema pain is highly controversial, as there is no proven conservative treatment with long-term effectiveness.[6,7] There is no established curative gold standard treatment for lipoedema. Compression garments have some effect on mobility, but the effect on disease progression and pain has not been proven. Lipedema is more resistant to diet and exercise than obesity. Bariatric surgery and liposuction are useful in selected cases.[3] The effect of these treatments on lipedema pain is also unclear. It has been reported that the only effective treatment for lipoedema pain is microcannular tumescent liposuction.[6]

Both FMS and lipedema are diseases characterized by soft tissue pain seen in middle-aged women. Both diseases are chronic diseases with no curative treatment. Both diseases are seen in overweight people and together with depression. There are no abnormal imaging and laboratory findings that can be used in the differential diagnosis of these two conditions.[1-5] As far as we know, in the only study comparing these two chronic soft tissue pain conditions, it was reported that the clinical characteristics of FMS and lipedema were similar, and the perception of disease and comorbidities were more common in FMS patients than in lipedema patients.[4]

The aims of this study were to determine how many of the patients with lipedema meet the diagnostic criteria for FMS, the effect of the comorbidity of lipedema and FMS on pain and quality of life, and their relationship with extremity volumes, USG measured soft tissue thickness and lipedema disease severity. To the best of our knowledge, our study is the first in the literature to evaluate the pain and quality of life of patients with FMS and lipedema, and to investigate the relationship between dermal and subdermal ultrasonographic (USG) measurements and lipedema disease stage.

METHODS

We carried out a prospective cohort study. The protocol of our study approved by Ankara Training and Research Hospital Clinical Researches Ethics Committee (Date: 08.02.2023, Decision No: 1200/2023). The study have been conducted in accordance with the Helsinki Declaration of Principles. All patients included in the study signed the informed consent form.

The inclusion criteria for the study were a confirmed diagnosis of FMS according to ACR 2016 FMS criteria which consist of two anamnestic self-administered scores; The Widespread Pain Index (WPI) and the Symptom Severity Score (SSS) together subsumed in the Fibromyalgia Survey Questionnaire (FSQ). The WPI counts the number of painful body parts from 0 to 19. The SSS ranges from 0 to 12 and the sum of 3 dimensional items scaled 0=absent, 1=mild, 2=moderate, 3=severe/always and referring to: Daily fatigue, waking unrefreshed, cognitive symptoms plus 3 binary yes/no items regarding the presence (=1)/absence (=0) of headache, pain and cramps in the lower abdomen and depression. The diagnosis of FMS requires chronic pain (≥3 months) in 4 of 5 body regions (the 4 quadrants and the spine, assessed by the WPI) together with either (WPI ≥7 and SSS ≥5) or WPI 4-6 and SSS ≥9).[5] We included FMS patients with USG Thigh soft tissue thickness (sum of dermal and subdermal thickness) measurements less than 17.9 and pretibial soft tissue thickness measurements less than 11.7 mm.[8]

Lipedema stage 1- 3 were diagnosed according to S1 guidelines of the German Society of Phlebology. Summary of diagnostic criteria in lipedema; Onset during puberty, pregnancy, or menopause, disproportional proliferation of adipose tissue (extremities, trunk), cuffing around the joints, hands and feet are not affected, feelings of heaviness and tightness in the extremities affected, tenderness to palpation or spontaneous pain – increaising over the course of the day, Edema – increasing over the course of the day, easy bruising, Stemmer's sign negative.[1]

We included FMS patients with USG Thigh soft tissue thickness measurements greater than 17.9 and pretibial soft tissue thickness measurements greater than 11.7 mm.[8]

Exclusion criteria were other type of edema such as lymphedema, phleboedema, renal or hearth insufficiency, using any medication that could affect the body fluid and electrolyte balance, BMI>50, does not know Turkish well enough, low psycho-intellectual abilities, serious somatic disease.

Fifty three lipedema and 32 FMS patients were included in the study. The number of patients who completed the FMS diagnostic criteria in the lipedema group was determined. Demographic features of the patients were recorded. Classification of lipedema by morphological characteristics in arms and legs determined according to lipedema S1 guidelines. Stage 1: smooth skin; homogenous increase in subcotaneous tissue, Stage 2: irregular skin surface (indentations), nodular changes of the subcutaneous tissue, Stage 3: pronounced increase in circumference with loose skin/tissue ('dewlap') (**Figure 1**).[1]

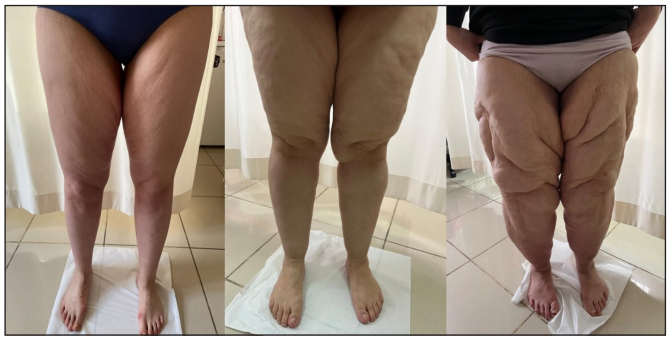

Figure 1. Lipedema stages

The truncated cone method was used to calculate the estimated volumes for the lower extremities in both groups. The right and left leg circumferences were measured at 4 cm. intervals starting from the ankle. The reliability and specificity of the calculated volume were previously reported.[9]

In addition to circumferential measurements and calculations, the dermal and subdermal thicknesses were measured by USG (7-12 MHz linear-array transducer, Logic P5, GE medical systems, Wisconsin, USA) at the same points on both limb by the same physician with more than 5 years' experience on musculoskeletal USG (BDÇ). USG gel was applied generously to the skin, and the probe was placed transversely on the leg. No pressure was applied during the USG measurements. Amato et al.[8] suggested a cut-off of 11.7 mm for more accurate pretibial soft tissue thickness measurements for the diagnosis of lipoedema, followed by a cut-off of 17.9 mm for the thigh. Anterior thigh measurements were made between the iliac crest and the lower patellar border. The pre-tibial measurements were made midpoint between anterior tibial tuberosity and medial malleolus.[8] Dermal and subdermal thicknesses were summed and recorded as a thigh and pre-tibial soft tissue thicknesses (**Figure 2**,**3**).

Health related quality of life was evaluated using the Short Form-36 (SF-36). SF-36 includes both physical and mental health parameters related to activities of daily living.[10]

Statistical Analysis

Statistical analysis was performed using SPSS version 15.0 for Windows (SPSS Inc., Chicago, IL, USA). The normality of variances was tested with the Shapiro–Wilk test. Descriptive analyses were used for the demographic data and Spearman's rank correlation coefficient to determine the relationship between the variables. The Mann Whitney U test and Student t test were conducted to evaluate the mean difference between groups when appropriate. The level of statistical significance was set at p<.05.

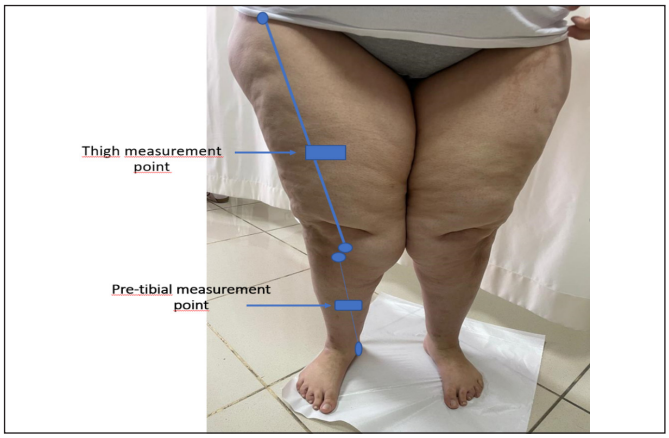

Figure 2. Thigh and pretibial measurement points where ultrasonographic soft tissue thicknesses are evaluated (Modified from Amato et al. 2021)

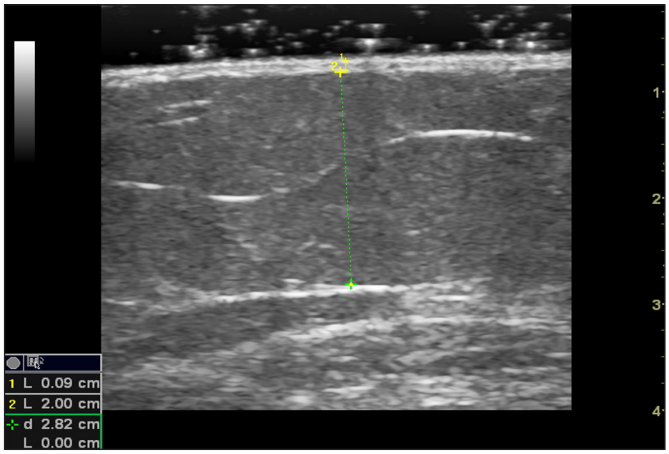

Figure 3. Ultrasonographic measurement of the dermal and subdermal thickness of the thigh

RESULTS

Comparison Between All Lipedema Patients and FMS Group

The mean age of the 53 females with lipedema was 52±11.8 years, and for the 32 females with FMS it was 51.9±10.1 years (p>0.05). There was statistically significant difference between the two groups with respect to body mass index (BMI) values (p<0.001). The extremity volumes and soft tissue thicknesses were higher in lipedema group than FMS group p<0.001). The FMS severity scores were higher in FMS group than Lipedema group (Table 1). The mean VAS score, general health, vitality, social functioning, role emotinal and mental health scores were better in lipedema patients than FMS patients (Table 2).

Comorbid Lipedema and FMS Group and FMS Group

In lipedema group, 21 (39.6%) patients have fulfilled the ACR 2016 FMS criteria. Comorbid Lipedema and FMS group FMS severity scores (SSS and WPI) were slightly lower than FMS patients. BMI and extremity volumes were not different between Lipedema without FMS and Comorbid lipedema and FMS group (Table 3).

Comparison between Lipedema without FMS Group, Bodily pain and VAS were more severe in Comorbid Lipedema and FMS group than the others. But all SF-36 components were better in lipedema without FMS group than Comorbid FMS and FMS group. Not surprisingly, mental health is better in lipedema without FMS group than the others (Table 4).

According to correlation analysis, there was no relationship between lipedema stage and FMS severity scores, VAS and SF-36 parameters (p>0.05).

Table 1. Demographic characteristics, extremity volumes and Fibromyalgia severity scores (SSS and WPI) of the all Lipedema and FMS patients

	Lipedema Group (n=53)	FMS Group (n=32)	p
Age (years) (mean±SD)	51.5±11.8	51.9±10.1	0.733*
BMI (kg/m²) (mean±SD)	39.4±5.2	29.3±3.1	<0.001*
Lipedema Stage n (%) Stage 1 Stage 2 Stage 3	 8 (15.9) 37 (69.8) 8 (15.9)		
SSS (median (interquartile))	6 (4-8)	9 (8-9)	<0.001[a]
WPI (median (interquartile))	10 (8-13)	15 (12-15)	0.002[a]
FSS (median (interquartile))	16 (9-21)	23 (21-24)	<0.001[a]
Extremity volume R (liter) (mean±SD)	12.9±2.1	7.9±2.6	<0.001*
Extremity volume L (liter) (mean±SD)	11.3±2.3	8.1±2.6	<0.001*
Fulfilled FMS Criteria (ACR 2016) n (%)	21 (39.6)	32 (100)	

SD: Standard deviation, BMI: Body mass index, SSS: Symptom severity score, WPI: Widespread pain index, FSS: Fibromyalgia severity scale, R: Right, L: Left, FMS: Fibromyalgia syndrome, ACR: American College of Rheumatology, *Student t test statistics, a Mann Whitney U Test statistics

Table 2. USG soft tissue thicknesses, VAS and SF-36 components of all lipedema and FMS patients

	Lipedema Group (n=53) (median (interquartile))	FMS Group (n=32) (median (interquartile))	p*
Thigh USG soft tissue thickness (mm)	38.5 (33.4-43.6)	14.39 (11.9-16.1)	<0.001
Pretibial USG soft tissue thickness (mm)	25 (18-30.9)	8.6 (7.6-9.8)	<0.001
VAS (0=best, 10=worst)	37 (12-53)	40 (25.5-45)	0.839
SF-36 Components (0=worst, 100=best)			
Physical components;			
Role physical	0 (0-75)	0 (0-37.5)	0.267
Physical functioning	40 (20-60)	30 (20-50)	0.244
Bodily pain	35 (10-57.5)	40 (20-38.7)	0.689
General health	35 (30-50)	30 (20-38.7)	0.014
Mental components;			
Vitality	50 (40-70)	30 (20-43.7))	<0.001
Social functioning	62.5 (50-75)	45.2 (25-63)	0.005
Role emotional	66.6 (0-100)	0 (0-33)	0.002
Mental health	64 (48-72)	40 (37-60)	<0.001

USG: Ultrasonography, VAS: Visuel analogue scale, SF-36: Short Form 36, *Mann Whitney U test statistics

Table 3. Demographic characteristics, extremity volumes and Fibromyalgia severity scores (SSS and WPI) of the comorbid Lipedema and FMS group, lipedema without FMS group and FMS patients

	Comorbid Lipedema and FMS Group 1 (n=21)	p (1-2)	Lipedema without FMS Group 2 (n=32)	p (2-3)	FMS Group 3 (n=32)	p (1-3)
Age (years) (mean±SD)	50.9±13.2	0.524*	51.9±10.9	0.655*	51.9±10.1	0.935*
BMI (kg/m²) (mean±SD)	38.7±4.7	0.476*	40±5.6	<0.001*	31.3±3.1	<0.001*
SSS (median (interquartile))	8 (6-9)	0.001[a]	4 (1-4)	<0.001[a]	9 (8-9)	0.037[a]
WPI (median (interquartile))	13 (10-15)	0.026[a]	9 (0-11)	<0.001[a]	15 (12-15)	0.304[a]
FSS (median (interquartile))	21 (17-24)	0.001[a]	10 (6-15)	<0.001[a]	23 (21-24)	0.199[a]
Extremity volume R (liter) (mean±SD)	11.8±2.8	0.696*	11.3±1.9	<0.001*	7.9±2.6	<0.001*
Extremity volume L (liter) (mean±SD)	11.8±2.8	0.561*	11.2±1.9	<0.001*	8.1±2.6	<0.001*
Fulfilled FMS Criteria (ACR 2016) n (%)	100 (100)		0 (0)		32 (100)	

SD: Standard deviation, BMI: Body mass index, SSS: Symptom severity score, WPI: Widespread pain index, FSS: Fibromyalgia severity scale, R: Right, L: Left, FMS: Fibromyalgia syndrome, ACR: American College of Rheumatology, *Student t test statistics, a Mann Whitney U Test statistics

Table 4. USG soft tissue thicknesses, VAS and SF-36 components of the comorbid lipedema and FMS group, lipedema without FMS group and FMS patients

	Comorbid Lipedema and FMS Group 1(n=21)	p* (1-2)	Lipedema without FMS Group 2 (n=32)	p* (2-3)	FMS Group 3 (n=32)	p* (1-3)
Thigh USG soft tissue thickness (mm)	39.9 (33.7-45)	0.174	36.1 (31-42.1)	<0.001	14.39 (11.9-16.1)	<0.001
Pretibial USG soft tissue thickness (mm)	28.1 (21.7-31.6)	0.124	22.9 (16.6-29.2)	<0.001	8.6 (7.6-9.8)	<0.001
VAS (0=best, 10=worst)	60 (50-71.25)	<0.001	27 (0-37)	0.001	40 (25.5-45)	<0.001
SF-36 Components (0=worst, 100=best)						
Physical components;						
Role physical	0 (0-75)	<0.001	75 (0-100)	<0.001	0 (0-37.5)	0.970
Physical functioning	25 (12.5-40)	0.125	67.5 (55-80)	<0.001	30 (20-50)	0.424
Bodily pain	22.5 (0-40)	<0.001	62.5 (54.3-78.1)	<0.001	40 (20-38.7)	0.001
General health	35 (22.5-40)	<0.001	55 (42.5-70)	<0.001	30 (20-38.7)	0.451
Mental components;						
Vitality	45 (30-65)	0.062	55 (50-70)	<0.001	30 (20-43.7)	0.01
Social functioning	62.5 (50-62.5)	0.002	75 (62.5-87.5)	<0.001	45.2 (25-63)	0.135
Role emotional	33 (0-100)	0.317	83.3 (0-100)	0.001	0 (0-33)	0.025
Mental health	60 (42-72)	0.133	70 (58-76)	<0.001	40 (37-60)	0.01

USG: Ultrasonography, VAS: Visuel analogue scale, SF-36: Short Form 36, *Mann Whitney U test statistics

DISCUSSION

In this study, we investigated the presence of FMS in lipedema patients and found that 39% of our lipedema patients met the ACR 2016 FMS diagnostic criteria. We found that the severity of FMS disease in comorbid lipedema and FMS patients was similar to FMS patients without lipedema. When we compared the quality of life of lipedema and FMS patients, we found that physical health of FMS patients was similar to lipedema patients, while their mental health was worse than lipedema patients. However, when we evaluated comorbid lipedema and FMS patients alone, we saw that the presence of lipedema and FMS in the same patient affects physical health more negatively than mental health. In the correlation analysis, we also did not find a significant relationship between the stage of lipedema and the parameters that determine the stage and severity of lipedema such as limb volumes and soft tissue thickness, and pain and quality of life. We also could not show a relationship between pain and disease severity in lipedema in this study.

What we saw in our clinical practice actually correlated with the results in our study. While there were no pain in some stage 3 patients, we also encountered very painful stage 1 patients. The latest lipoedema position document also agrees on this issue. According to the lipedema position document, the staging of lipedema is dependent on the subjective assessment of physicians and is based solely on morphological criteria. The actual symptoms of the patient are not taken into account. These stages do not reflect the clinical reality. Some patients have stage 3 lipoedema and have severe disproportionate adipose tissue in their limbs but have no or mild symptoms. Some patients have stage 1 lipoedema but complain of severe pain and restlessness in their legs.[3] However, Chakraborty et al.[11] reported that pain intensity and neuronal cell body distribution in the skin are stage dependent. They also identified neuropathic pain in lipoedema, evidence of neurogenic inflammation on skin biopsy, and increased cutaneous mechanical sensitivity. They also observed that neuronal density (Tuj-1+ dermal neuron) decreased in the abdomen across the lipedema stages; this suggests a systemic change associated with changes in lipedema tissue and neuronal density. Neurogenic features of lipoedema pain aside, neuropathic/nociplastic pain is also inherent in FMS disease. Serra et al.[12] reported that abnormal C nociceptor activity and increased mechanical sensitivity might contribute to the tenderness and pain suffered by FMS patients. In addition, small fiber neuropathy was detected in FMS patients.[13] These studies suggest that these two soft tissue pain syndromes can share common neuropathic/nociplastic features in the pathogenesis.

There were no pain in cardiogenic edema and lymphedema. If edema was the cause of pain, these patients should also have pain. In addition, contrary to its name, edema could not be detected in lipoedema. There is disproportionate accumulation of adipose tissue.[3] Some authors have suggested that the pain in lipedema is caused by tissue damage that is responsible for inflammatory and hypoxic processes. Adipose tissue increase causes local increase in proinflammatory hormones (adipokines).[3,14] Compared with normal subjects, the amount of sodium detected by magnetic resonance imaging was found to be increased in the skin and subcutaneous adipose tissues of people with lipedema. This is known as an indicator of inflammation. The authors stated that tissue sodium and adipose content might be an objective imaging-based biomarker that can be used in the differential diagnosis of lipoedema and obesity.[15] However, others speculated that the pain experienced by lipedema patients might be more related to the way the brain and nervous system interpret the stimulus, rather than tissue damage. The etiopathogenesis of FMS, as well as lipedema, depends on the biopsychosocial model of medicine and the complex mind-body relationship.[2,3]

For the last 20 years, researchers have defined FMS as nociplastic pain.[16] This type of pain is consistent with the definition of fibromyalgia as part of the group of central sensitivity syndromes.[17] Of course, the pathogenesis of FMS cannot be explained by a single etiological factor. It is known that genetic factors also play a role in the etiopathogenesis.[2] Peripheral mechanisms also play an important role in the pathogenesis of FMS. The high prevalence of FMS in patients with rheumatoid arthritis may be an evidence that joint pain, as a source of peripheral pain, can initiate the nociplastic process as a painful stimulus from the periphery.[18] Treatment of peripheral pain generators such as osteoarthritis leading to improvement in FMS symptoms suggest that the peripheral nervous system is involved in the pathogenesis. Peripheral nociceptive impulses are also thought to increase central sensitization. Centralized pain, also referred to as central sensitization or as nociplastic pain/FMS, is seen in patients with OA, inflammatory joint diseases as well as chronic low back and neck pain, complex regional pain syndrome, carpal tunnel syndrome, lateral epicondylitis, joint hypermobility syndrome. It also affects the patient's pain level, disease activity measures, and treatment selection and outcomes.[19] The comorbidity of lipoedema and FMS may activate the central sensitization of lipoedema as a peripheral pain generator, resulting in the onset or exacerbation of FMS symptoms. In addition, excessive load on the joints caused by obesity and excessive fat storage in lipedema patients may also contribute to the nociplastic process.

Physical and mental stresses are known as factors that worsen pain.[20] Psychological disorders are common in FMS patients and affect the patient's life and even disease severity.[21] Lifetime prevalence of anxiety disorders is 60% and depression is 14-36% in FMS patients.[22] Antidepressants especially duloxetine and milnacipram are FDA-approved medicines in FMS. These medicines are effective for pain rather than depressive symptoms.[23] Psychological problems have also been investigated in patients with lipedema. In a study conducted at the Földi Clinic[24], 50% of 150 lipedema patients had a mental health disorder that started 12 months before the development of lipedema-related pain. 80% of women with lipedema had psychological symptoms prior to the onset of lipedema-related pain. There is no evidence that lipoedema causes mental health problems. However, psychological problems may contribute to the development of lipedema. Moreover, depression and posttraumatic stress disorders were associated with patients' pain severity.[24] Studies have found an increase in inflammatory markers in people with depression, social stress, or posttraumatic stress disorder without any association with the underlying somatic disease.[25,26] Considering the psychological vulnerability of patients with lipoedema, chronic stress and psychological symptoms can create a vicious circle by activating inflammatory mediators, increasing pain intensity and worsening mental stress.[3] When we compared patients with lipedema and FMS in our study, we found that both diseases were equally badly affected in the physical health components of SF-36. However, in the mental health component, we observed that FMS patients were affected more badly than lipedema patients. However, we would expect the physical functions and mobility of lipedema patients to deteriorate further. Only VAS scores of comorbid lipedema and FMS patients were higher than FMS patients. In addition, while the BMI of lipedema patients is higher, their physical functions are similar to those of FMS patients, which indicates that FMS is a disease that can cause serious disability.

Angst et al.[4] evaluated the frequency of FMS in lipedema and found it to be 34%. It is a rate similar to our rate. In this study, it was observed that the comorbidity of these two chronic soft tissue pain syndromes negatively affected the patient's quality of life. In addition, in our study, lipedema was evaluated systematically in FMS patients and FMS patients without lipedema were included. In Angst et al 's study, FMS patients were not evaluated for lipedema. In our study, lipedema was ruled out by evaluating the extremity volumes and soft tissue thickness of FMS patients. This is one of the strengths of our work. However, despite the diagnostic criteria of both diseases, stage 1 lipoedema patients cannot be distinguished from FMS.

These two soft tissue pain syndromes can share common neuropathic/nociplastic features in the pathogenesis. The comorbidity of lipoedema and FMS may activate the central sensitization of lipoedema as a peripheral pain generator, resulting in the onset or exacerbation of FMS symptoms. In addition, excessive load on the joints caused by obesity and excessive fat storage in lipedema patients may also contribute to the nociplastic process. despite the diagnostic criteria of both diseases, stage 1 lipoedema patients cannot be distinguished from FMS. With our current knowledge, we cannot distinguish which disease is the cause and which is the effect, but we can see that these two diseases have a lot in common in terms of clinical and pathogenetic aspects.

One of the limitation of our study is that we did not use disease specific severity and quality of life scales for lipedema and FMS. Although there are specific scales for FMS, specific scales for lipedema are not widely used. Another limitation of our study is the small number of patients .

CONCLUSION

FMS and lipedema are two common chronic soft tissue conditions. They have many common features such as their localization, pain characteristics, gender distribution, clinical course, comorbidities and non-curative treatment options. The comorbidity of these two diseases in a patient also negatively affects physical and mental functions. Therefore, investigation and treatment of comorbid FMS in lipedema patients may contribute to their quality of life and pain.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of Ankara Training and Research Hospital Clinical Researches Ethics Committee (Date: 08.02.2023, Decision No: 1200/2023).

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.

Financial Disclosure: The authors declared that this study has received no financial support.

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.

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