CEOG Clinical and Experimental Obstetrics & Gynecology

Does increase in body mass index effect primary dysmenorrhea?

M. Temur[1,3], U. Gök Balci[2], Y. A. Güçlü[2], B. Korkmaz[3], P.Ö. Özbay[4], N. Soysal[2], Ö. Yilmaz[1], T. T. Yilmazer[2], T. Çift[3], K. Öngel[2]

[1] Department of Obstetrics and Gynecology, Manisa Merkezefendi State Hospital, Manisa
[2] Department of Family Medicine, Tepecik Research and Training Hospital, İzmir
[3]Department of Obstetrics and Gynecology, Bursa Yüksek İhtisas Research and Training Hospital, Bursa
[4]Department of Obstetrics and Gynecology, Aydın Obstetrics and Pediatrics Hospital, Aydın (Turkey)

Summary

Purpose: In the present study, the aim was to evaluate the relationship between obesity and dysmenorrhea and the effects of sociodemographic features on it. *Materials and Methods:* A total of 303 women were included in the study .Grading of severity of dysmenorrhea was made based on Verbal Multidimensional Scoring System (VMSS). *Results:* When correlations between severity of dysmenorrheic symptoms and patients were assessed, there was a statistically significant difference between the rates of chronic disease in the dysmenorrhea groups and the rates of dysmenorrhea history in the family ($p = 0.037$ and $p = 0.008$, respectively). There was a statistically significant difference in the mean body mass index (BMI) in the dysmenorrhea grades ($p < 0.001$). The mean BMI for those without dysmenorrhea was higher than those with mild or moderate dysmenorrhea. Those with severe dysmenorrhoea had a significantly higher mean BMI than those with mild dysmenorrhea ($p < 0.001$, $p = 0.002$, and $p = 0.009$, respectively). There was a statistically significant difference in dysmenorrheal grades and BMI groups ($p = 0.002$). The severity of dysmenorrhoea in those with a BMI of 30 and above was greater than those of mild and moderate ones. *Conclusion:* The main underlying cause of dysmenorrhea may not be obesity, but it may be one of the correctible predisposing factors. Balanced diet and trying to decrease one's BMI within normal limits may lower the incidence of dysmenorrhea.

Key words: Dysmenorrhea; Obesity; Body mass index.

Introduction

Dysmenorrhea is a cyclic pain felt especially on suprapubic and pelvic region during menstrual period. Dysmenorrhea includes two types: primary and secondary dysmenorrhea. Primary dysmenorrhea is a cyclic pain stemming from intrinsic uterine factors. Though etiology of primary dysmenorrhea is not fully understood, prostaglandins and especially PGF alpha has been held responsible from the development of dysmenorrheic pain. PGF2 alpha induces myometrial contractions and ischemia leading to emergence of dysmenorrheic pain. Intrinsic as well as emotional factors, as depression and anxiety, induce primary dysmenorrhea. However, secondary dysmenorrhea can occur as a result of intrapelvic pathologies as endometriosis, adenomyosis, and ovarian cyst and application of an intrauterine device [1, 2].

Primary dysmenorrhea is a relatively prevalent problem in women of their reproductive ages. Dysmenorrhea adversely effects daily work and routine activities with resultant million dollars of material damage [3]. Worldwide studies on dysmenorrhea report its prevalence as ranging between 28 and 65.7 percent [4, 5]. However in Turkey, its incidence has been reported to vary between 58% and 89% [6-8].

Obesity is defined as an excessive accumulation of fat in the body. Obesity is an important risk factor for chronic diseases as diabetes, cardiovascular diseases, and cancer. Especially in recent years, it is an important health problem with increasing importance. Nowadays, nearly 300 million obese women have been detected worldwide. According to 2008, data nearly 1.4 billion adults are overweight and approximately 300 million of them are obese [9].

Body mass index (BMI) is a simple measurement tool used worldwide for the classification of obesity. BMI is easily calculated based on body weight and height. BMI is estimated by dividing body weight by the square of body height. BMI is categorized according to this classification as follows: BMI < 18.5 kg/m^2, underweight; BMI: 18.50-24.99 kg/m^2, normal range; BMI: 25.00-29.99 kg/m^2, overweight; BMI: 30.00-34.99 kg/m^2, obese class I; BMI: 35.00-39.99 kg/m^2, obese class II; BMI ≥ 40.00 kg/m^2, obese class III [9].

In the present study, the authors aimed to evaluate the relationship between obesity and dysmenorrhea and the effects of sociodemographic features on dysmenorrhea.

Revised manuscript accepted for publication March 13, 2017

Table 1. — *General features of the patients.*

Age (years) Ort. ± SD (min-max)		32.3±9.0 (13-57)
Education level, n (%)	No	5 (1.7)
	Primary	83 (27.4)
	Middle	36 (11.9)
	High	93 (30.7)
	University	86 (28.4)
Economical level, n (%)	Poor	23 (7.6)
	High	59 (19.5)
	Moderate	221 (72.9)
Smoking history, n (%)		88 (29.0)
Chronic disease, n (%)		90 (29.7)
Parity Ort. ± SD (min-max)		1.0-1.2 (0-8)
Parity, n (%)	Nulliparity	136 (44.9)
	Primiparity	57 (18.8)
	Multiparity	110 (36.3)
Type of birth, n (%)	Vaginal birth	4 (1.3)
	Cesarean birth	163 (53.8)
	No	136 (44.9)
BMI (kg/m^2), Ort. ± SD (min-max)		28.2±8.7 (14.3-64.3)
BMI (kg/m^2), n (%)	< 18.5	14 (4.6)
	18.5-24.9	128 (42.2)
	25-29.9	53 (17.5)
	30≤	108 (35.6)
Dysmenorrhea history in family, n (%)		65 (21.5)
Dysmenorrhea grade, n (%)	No	112 (37.0)
	Mild	80 (26.4)
	Moderate	67 (22.1)
	Severe	44 (14.5)

Materials and Methods

A total of 303 women (age range, 15-44 years) in their reproductive age with regular menstruation periods who consulted to Obesity Clinic of Izmir Tepecik Training and Research Hospital between February 2013 and June 2013 were included in the study. Using a questionnaire form prepared by the investigators, sociodemographic characteristics of the female patients were recorded. In the outpatient clinic, body weight and height were measured and recorded in the questionnaire file. BMIs were calculated by dividing body weight in kg by square of height in meters and obesity was classified based on this formula. Grading of severity of dysmenorrhea was made based on Verbal Multidimensional Scoring System (VMSS) [10].

(A) Mild dysmenorrhea is defined as painful menstruation with no limitation of normal activity, with infrequent requirement of analgesics and no systemic complaints. (B) Moderate dysmenorrhea is defined as menstrual pain affecting daily activities, with requirement of analgesics for pain relief and few systemic complaints. (C) Severe dysmenorrhea is defined as menstrual pain with severe limitation of daily activities, poor response to analgesics, and apparent systemic complaints like vomiting, fainting, etc.

SPSS 15.0 was used for statistical analysis. Descriptive statistics, number, and percentage for categorical variables, mean, standard deviation, minimum, and maximum were given for numerical variables. Because the numerical variables did not satisfy the normal distribution condition, multiple group comparisons were made with the Kruskal Wallis test independently. Subgroup analyzes were done by Mann Whitney U test and interpreted by Bonferroni correction. Comparisons of ratios in groups were made with Chi Square Analysis. Monte Carlo simulation was applied when conditions were not provided. Statistical significance level of alpha was accepted as $p < 0.05$.

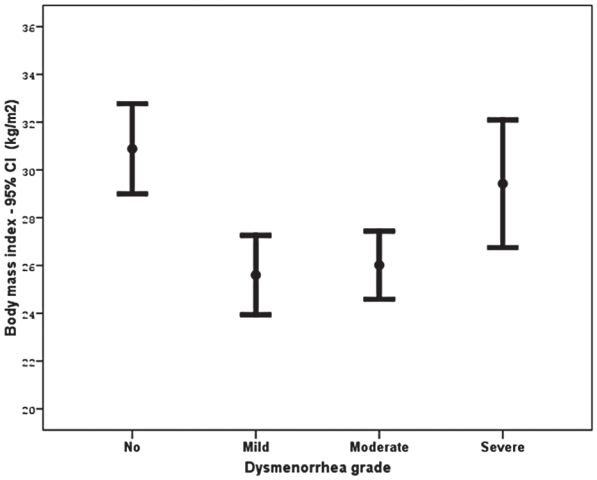

Figure 1. — The relationship between dysmenorrhea grade and body mass index.

Results

Three hundred three women with a mean age of 32.3 ± 9.0 years were included in the study. The general characteristics of the pregnants are summarized in Table 1.

Of the women 26.4% had mild, 22.1% had moderate, and 14.5% had severe dysmenorrhea; 37% of the women had no dysmenorrhea (Table 1). There was no statistically significant difference in the mean age, education and economic levels, smoking rates, and parity types of the dysmenorrhea groups ($p = 0.607$, $p = 0.164$, p = 0.600, $p = 0.172$, and $p = 0.888$, respectively).

There was a statistically significant difference between the rates of chronic disease in the dysmenorrhea groups and the rates of dysmenorrhea history in the family ($p = 0.037$ and $p = 0.008$, respectively). Those with severe dysmenorrhea had a higher rate of chronic illness (33.9% mild, 22.5% moderate 22.4%, and severe 43.2%). Those with moderate dysmenorrhea had a higher rate of dysmenorrhea history in the family (history of dysmenorrhea in the family 14.3%, mild 21%, moderate 35.8%, and severe 18.2%) (Table 2).

There was a statistically significant difference in the mean BMI in the dysmenorrhea grades ($p < 0.001$). The mean BMI for those without dysmenorrhea was higher than those with mild and moderate dysmenorrhea. Those with severe dysmenorrhea had a significantly higher mean BMI than those with mild dysmenorrhea ($p < 0.001$, $p = 0.002$, and $p = 0.009$, respectively). There was a statistically significant difference in dysmenorrheal grades and BMI groups ($p = 0.002$). The severity of dysmenorrhea in those with a BMI of 30 and above was greater than those with mild and moderate ones (Table 3, Figure 1).

Table 2. — *The relationship between dysmenorrhea severity and studied parameters.*

		Dysmenorrhea grade								
		No		Mild		Moderate		Severe		
		Ort. ± SD		Ort. ± SD		Ort. ± SD		Ort. ± SD		
Age (years)		32.8 ± 9.1		31.8 ± 8.7		32.9 ± 9.3		31.0 ± 9.0		0.607
		n	%	n	%	n	%	n	%	*p*
Education level	No	2	1.8	1	1.3	2	3	0	0	0.164
	Primary	33	29.5	20	25	19	28.4	11	25	
	Middle	19	17	5	6.3	6	9	6	13.6	
	High	26	23.2	24	30	27	40.3	16	36.4	
	University	32	28.6	30	37.5	13	19.4	11	25	
Economical level	Poor	6	5.4	5	6.3	9	13.4	3	6.8	0.600
	High	25	22.3	14	17.5	12	17.9	8	18.2	
	Moderate	81	72.3	61	76.3	46	68.7	33	75	
Smoking history		28	25	20	25	22	32.8	18	40.9	0.172
Chronic disease		38	33.9	18	22.5	15	22.4	19	43.2	0.037
Parity	Nulliparity	49	43.8	39	48.8	26	38.8	22	50	0.888
	Primiparity	20	17.9	15	18.8	14	20.9	8	18.2	
	Multiparity	43	38.4	26	32.5	27	40.3	14	31.8	
Dysmenorrhea history in family		16	14.3	17	21.3	24	35.8	8	18.2	0.008

Table 3. — *The relationship between dysmenorrhea grade and body mass index.*

		Dysmenorrhea grade								
		No		Mild		Moderate		Severe		
		Ort. ± SD		Ort. ± SD		Ort. ± SD		Ort. ± SD		
BMI (kg/m^2)		30.9 ± 10.0		25.6 ± 7.5		26.0 ± 5.8		29.4 ± 8.8		<0.001
		n	%	n	%	n	%	n	%	*p*
BMI (kg/m^2)	< 18.5	5	4.5	2	2.5	5	7.5	2	4.5	0.002
	18.5-24.9	33	29.5	47	58.8	32	47.8	16	36.4	
	25-29.9	18	16.1	14	17.5	13	19.4	8	18.2	
	≥ 30	56	50.0	17	21.3	17	25.4	18	40.9	

Kruskal-Wallis (Mann-Whitney U test).

Discussion

In the pathophysiology of dysmenorrhea, together with withdrawal of progesterone, release of prostaglandins and leukotrienes from arachidonic acid is very important. Inflammatory response mediated by these prostaglandins (mainly PGF2) and leukotrienes induces vasoconstriction and myometrial contractions leading to development of ischemia and pain [11].

Dysmenorrhea is one of the most frequent gynaecological causes of presentation to hospital. In the literature its incidence ranges between 43 and 90 percent [12, 13]. In a study performed on a group of young girls in Italy, the incidence of primary dysmenorrhea was detected as 85% and in another study in Mexico its incidence was reported as 48.4% [14, 15]. The incidence of dysmenorrhea in Turkey changes between 63 and 70 percent [8, 16]. In this study the rate of dysmenorrhea was 63%, which is comparable with studies performed in this country. These outcomes demonstrate that dysmenorrhea still continues to be one of the prevalent gynaecologic problems among female population. Differences in the incidence of dysmenorrhea among countries might stem from differences in geographic, genetic, nutritional factors, and varying BMIs.

In studies performed, a correlation between family history and dysmenorrhea has been detected. In studies performed in compliance with many literature studies, family history can significantly affect prevalence of dysmenorrhea [1, 7]. In the present study, dysmenorrhea was more common in those who had dysmenorrhea history in the family.

In recent studies, cytokine gene expressions in peripheral blood of dysmenorrheic patients were analysed and dysmenorrhea has been assertedly related to alterations in the balance between proinflammatory cytokines and TGF beta superfamily. Increases in menstrual phase proinflammatory cytokines (IL1B, TNF, IL6, and IL8) and decreases in some TGF-β superfamily members (BMP4, BMP6, GDF5, GDF11, LEFTY2, NODAL, and MSTN) were detected

[17].

Adipose tissue is not only a source of energy, but it also functions as an active endocrine organ, with an ability to secrete various cytokines, peptides originating from adipose tissue. As observed in many studies, adipose tissue secretes increased amounts of various cytokines (TNF alpha, IL 6, and IL 8), prostaglandins (PGI2 and PGF2), and proinflammatory cytokines [18]. In some literature studies, a correlation was found between BMI and dysmenorrhea. However, in some other studies, though debatable, obesity has been indicated as one of the predisposing factors for dysmenorrhea [19]. In a study by Harlow *et al.*, and Ju *et al*. a correlation was found between higher BMI and dysmenorrhea [20, 21]. Origin of this finding may be related to the role played by cytokines in the pathophysiology of primary dysmenorrhea and increased release of cytokines from excess adipose tissue in patients with higher BMI concurrently with primary dysmenorrhea. Moreover, in some studies a correlation was found between decreased BMI and dysmenorrhea. However in these study populations obese patients were limited in number and these studies yielded scarce data for the evaluation of the impact of obesity on dysmenorrhea [22, 23], which investigated the association between obesity and dysmenorrhea in adolescent girls, the study group with BMI above 25 kg/m^2 consisted of 25.26% of the study population. In this study, although, a statistically significant difference between obese and non-obese patients as for dysmenorrhea could not be demonstrated, the obese group experienced dysmenorrheic symptoms more frequently [24]. In a study by Haidari *et al.*, the authors indicated the presence of a correlation between dysmenorrhea and some anthropometric parameters including waist-hip ratio and waist circumference [25]. In a study by Neal *et al.*, the authors detected that diets with low-fat content decreased concentrations of sex hormone-binding globulin and body weights without resultant alleviation of dysmenorrheic symptoms [26]. However, in their study the percentage of patients with BMI over 25 and 30 kg/m^2 was higher than those found in many other studies. Dysmenorrhea was statistically significantly less frequent in the group of patients with BMIs above 30 kg/m^2 ($p < 0.05$). This condition is strongly related to the fact that primary dysmenorrhea is associated with ovulatory cycles and also it may be correlated with non-observance of ovulatory cycles in women with BMI over 30 kg/m^2 [15]. In the present study, there was more severe dysmenorrhoea with a BMI of 30 and over. This may be due to increased secretion of dysmenorrhoea from fat cells. Interestingly, there was no dysmenorrhoea in those with a BMI of 30 or more. This can be explained by the presence of more ovulation defects in the excess of adipose tissue. Indeed when compared with the group with BMI lower than 30 mg/kg^2, in the group with higher BMI, a statistically significantly increased incidence of dysmenorrhea was detected ($p < 0.05$) This finding demonstrates potential effect of cytokines produced in adipose tissue on dysmenorrhea.

In the literature the relationship between BMI and dysmenorrhea is debatable. The common characteristic of the studies which asserted a correlation was that they detected higher incidence of dysmenorrhea in study groups with lower and higher BMIs, rather than those with normal BMI. The main underlying cause of dysmenorrhea may not be obesity, but it may be one of the correctible predisposing factors. Therefore, a balanced diet and trying to decrease one's BMI within normal limits may lower the incidence of dysmenorrhea.

Acknowledgement

The authors are grateful to Dr. Özgür Yılmaz for his valuable contributions to the statistical analysis. This study did not receive any specific grant from any funding agency in the public, commercial or not-for-profit sector. This study was supported by Muzaffer Temur (first author).

References

[1] Dorn L.D., Negriff S., Huang B., Pabst S., Hillman J., Braverman P., Susman E.J.: "Menstrual symptoms in adolescent girls: association with smoking, depressive symptoms, and anxiety". *J. Adolesc. Health,* 2009, *44*, 237.

[2] Wallace S., Keightley A., Gie C.: "Dysmenorrhoea". *Obstet. Gynaecol.,* 2010, *12*, 149.

[3] Coco AS.: "Primary dysmenorrhea". *Am. Fam. Physician,* 1999, *60*, 489.

[4] Burnett M.A., Antao V., Black A., Feldman K., Grenville A., Lea R.: *et al.*: "Prevalence of primary dysmenorrhea in Canada". *J. Obstet. Gynaecol. Can.,* 2005, *27*, 765.

[5] Rodrigues A.C., Gala S., Neves Â., Pinto C., Meirelles C., Frutuoso C.: *et al.*: "Dysmenorrhea in adolescents and young adults: prevalence, related factors and limitations in daily living". *Acta Med. Port.,* 2011, *24*, 383.

[6] Nur N., Sumer H.: "Prevalence of dysmenorrhea and related risk factors in adolescents". *Surekli. Tip. Egitimi. Dergisi.,* 2008, *7*, 27.

[7] Polat A., Celik H., Gurates B., Kaya D., Nalbant M., Kavak E., *et al.*: "Prevalence of primary dysmenorrhea in young adult female university students". *Arch. Gynecol. Obstet.,* 2009, *279*, 527.

[8] Unsal A., Ayranci U., Tozun M., Arslan G., Calik E.: "Prevalence of dysmenorrhea and its effect on quality of life among a group of female university students". *Ups. J. Med. Sci.,* 2010, *115*, 138.

[9] World Health Organisation: "Global database on body mass index". Available at: http://apps.who.int/bmi/index.jsp?introPage=intro_3.html

[10] Andersch B., Milsom I.: "An epidemiologic study of young women with dysmenorrhea". *Am. J. Obstet. Gynecol*., 1982, *144*, 655.

[11] Harel Z.: "Dysmenorrhea in adolescents and young adults: etiology and management". *J. Pediatr. Adolesc. Gynecol*., 2006, *19*, 363.

[12] Svanberg L., Ulmsten U.: "The incidence of primary dysmenorrhea in teenagers". *Arch Gynecol.,* 1981, *230*, 173.

[13] Jamieson D.J., Steege J.F.: "The prevalence of dysmenorrhea, dyspareunia, pelvic pain and irritable bowel syndrome in primary care practices". *Obstet. Gynecol*., 1996, *87*, 55.

[14] Balbi C., Musone R., Menditto A., Di Prisco L., Cassese E., D'Ajello M., *et al.*: "Influence of menstrual factors and dietary habits on menstrual pain in adolescence age". *Eur. J. Obstet. Gynecol. Reprod. Biol.,* 2000, *91*, 143.

[15] Ortiz M.I., Rangel-Flores E., Carrillo-Alarcón L.C., Veras-Godoy H.A.: "Prevalence and impact of primary dysmenorrhea among Mex-

ican high school students". *Int. J. Gynaecol. Obstet.,* 2009, *107*, 240.
[16] Aykut M., Gunay O., Gun I., Tuna R., Balcı E., Ozdemir M., et al.: "Biyolojik, sosyo-demografik ve nutrisyonel faktörlerin dismenore prevelansına etkisi". *Erciyes Tıp. Dergisi*., 2007, *29*, 393.
[17] Ma H., Hong M., Duan J., Liu P., Fan X., Shang E., et al.: "Altered cytokine gene expression in peripheral blood monocytes across the menstrual cycle in primary dysmenorrhea a case-control study". *PLoS One*, 2013, *8*, e55200.
[18] Wang Y.Y., Lin S.Y., Chuang Y.H., Chen C.J., Tung K.C., Sheu W.H.: "Adipose proinflammatory cytokine expression through sympathetic system is associated with hyperglycemia and insulin resistance in a rat ischemic stroke model". A*m. J. Physiol. Endocrinol. Metab.*, 2011, *300*, E155.
[19] Lefebvre G., Pinsonneault O., Antao V., Black A., Burnett M., Feldman K.: "Primary dysmenorrhea consensus guideline". *J. Obstet. Gynaecol. Can.,* 2005, *27*, 1117.
[20] Harlow S.D., Park M.: "A longitudinal study of risk factors for the occurrence, duration and severity of menstrual cramps in a cohort of college women". *Br. J. Obstet. Gynaecol.,* 1996, *103*, 1134.
[21] Chauhan M., Kala J.: "Relation between dysmenorrhea and body mass index in adolescents with rural versus urban variation". *J. Obstet. Gynaecol. India,* 2012, *62*, 442.
[22] Tangchai K., Titapant V., Boriboonhirunsarn D.: "Dysmenorrhea in Thai adolescents: prevalence, impact and knowledge of treatment". *J. Med. Assoc. Thai.,* 2004, *87*, 69.
[23] Kamacı M., Önder Y., Akman N.: "Adölesanlarda primer dismenorenin vücut kitle indeksiyle ilişkisi" *Van. Tıp. Dergisi.,* 1997, *4*, 154.
[24] Kaur K.: "Obesity and dysmenorrhea in young girls: is there any link". *Hum. Biol*., 2014, *3*, 214.
[25] Haidari F., Akrami A., Sarhadi M., Shahi M.M. : "Prevalence and severity of primary dysmenorrhea and its relation to anthropometric parameters". *J. HAYAT.,* 2011, *17*, 70.
[26] Barnard N.D., Scialli A.R., Hurlock D., Bertron P.: "Diet and sex-hormone binding globulin, dysmenorrhea, and premenstrual symptoms". *Obstet. Gynecol*., 2000, *95*, 245.

Corresponding Author:
M. TEMUR, M.D.
Manisa Merkezefendi Hospital
Department of Obstetrics and Gynecology
45020, Merkezefendi, Manisa (Turkey)
e-mail: drmuzaffer@yahoo.com