

MANAGEMENT OF CARDIO-METABOLIC FEATURES IN WOMEN DIAGNOSED WITH PREMENSTRUAL SYNDROME

Eugenia PETROVA^{1,2}, Florica ȘANDRU^{2,3},
Mara CARȘOTE^{1,2}, Adina GHEMIGIAN^{1,2},
Ana VALEA^{4,5}

¹“C.I. Parhon” National Institute of Endocrinology,
Bucharest, Romania

²“Carol Davila” University of Medicine and Pharmacy,
Bucharest, Romania

³Elias Emergency Hospital, Bucharest, Romania

⁴Clinical County Hospital, Cluj-Napoca, Romania

⁵“Iuliu Hatieganu” University of Medicine and
Pharmacy, Cluj-Napoca, Romania

Correspondence to:

Mara Carsote: carsote_m@hotmail.com

INTRODUCTION

Premenstrual syndrome (PM), and its severe variant, namely premenstrual dysphoric disorder (PMDD), associates a cluster of somatic (breast tenderness, fatigue, non-specific pain, bloating, extremities swelling, etc.) and psychologic (mood swings, sleep anomalies, irritability, food cravings, anger, etc.) elements with a menstrual cycle (MC) – related pattern (onset during luteal phase and resuming after menstruation) [1,2,3,4].

PM management starts from establishing the diagnostic that embraces a combination of self - reported accuses with cyclic appearance in addition to exclusion of other causes like thyroid and psychiatric (anxiety, depression) conditions which may actually overlap in some individuals, thus a clear multi-disciplinary distinction is necessary to improve the quality of life, to achieve optimal professional capacity in everyday life, and to provide an adequate outcome [1,2,5].

The increasing prevalence of PM in modern society underlines a multi-factorial panel that is more or less understood until this moment, but an important proportion of cases may still go underdiagnosed, thus untreated [1,2]. Also, the criteria of diagnostic among different societies and over time are heterogeneous, recently a new terminology was introduced, namely menstrual cycle-associated syndrome (MCAS) [2,6,7,8]. PM is found in reproductive female population with a variation from 10% to 90% (or even 100%) depending on the study and criteria of definition (approximately two thirds of females), while PMDD is reported in 2-8% of all women [1,9]. For instance, one recent study on 194 women with mean age of 30 years found 37% with PM, respective 15% with PMDD based on DSM-based PSST (premenstrual symptoms screening tool) [10].

Multiple mechanisms are involved as hormonal pathways concerning ovarian-associated sex steroids, neuroendocrine elements and central neurotransmitters;

PM (premenstrual syndrome) management starts from establishing the diagnostic that embraces a combination of self - reported accuses with cyclic menstrual appearance in addition to exclusion of other causes like thyroid and psychiatric conditions which may actually overlap in some individuals, thus a clear multi-disciplinary distinction is mandatory. Multiple mechanisms are involved in PM as hormonal pathways concerning ovarian-associated sex steroids, neuroendocrine elements, central neurotransmitters, anomalies of uterine-chemokine-brain-axis; genetic and epigenetic studies on PM still have conflicting results. New data suggested that PM is associated with cardio-metabolic conditions like high blood pressure (HBP) or obesity which also independently of PM affect a great number of people in certain populations including at young age. Our purpose is to focus on cardio-metabolic features in PM. This is a narrative review. Recent data suggests that approximately one in ten PM females associates HBP. Despite baseline normal values of arterial pressure, the menstrual cycle-related symptoms correlates with higher BP (mean, systolic, diastolic) during luteal phase versus follicular phase. Monthly variability of BP is correlated with PM symptoms via arterial stiffness and estrogens effects on renin-angiotensin system. In PM positive females diagnosed with refractory HBP, BP improves if SSRI are added, SSRIs representing the first line therapy in traditional PM management. One study found PM to be associated with a risk of developing HBP (hazard ratio of 1.4, CI 95%: 1.2-1.6), independent of age, smoking status and body mass index. In terms of PM and glucose profile, there is limited amount of published data. A few small sample size studies revealed glucose and insulin differences among PM females during menstrual cycle phases. Food behavior changes are part of PM. PM seems more frequent among obese individuals, but not all studies agree. Whether PM females are prone to cardio-metabolic comorbidities or they are incidental due to high prevalence in general reproductive female population of both conditions is still on open subject. But understating the cardio-metabolic features in PM is essential nowadays as modern society is oriented to a better management of conditions that impair the quality of life.

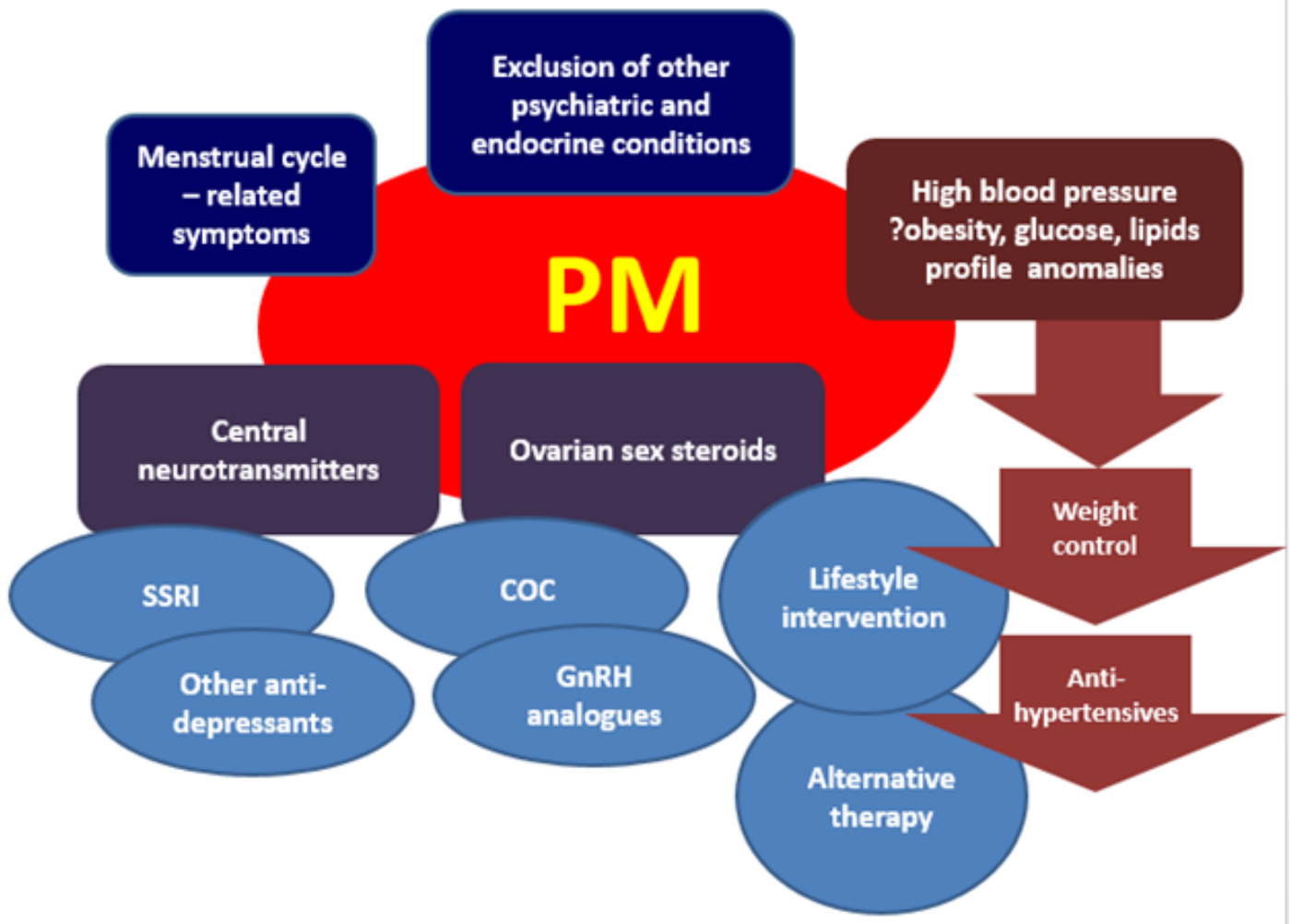
Keywords: premenstrual syndrome, diabetes mellitus, high blood pressure, obesity, estrogens, menstrual cycle, premenstrual dysphoric disorder, ovary, cholesterol, glucose, body mass index

MCAS mostly is considered an anomaly of uterine-chemokine-brain-axis; genetic and epigenetic studies on PM/PMDD still have conflicting results. [1,6,7,11,12]. The role of vitamins and minerals status as co-factors is controversial [1,13]. New data suggested that PM is associated with cardio-metabolic conditions like high blood pressure (HBP) or obesity which also affect a great number of people in certain populations including at young age, independently of PM [14] (**Figure 1**).

Understanding the pathogenic traits of the syndrome is mandatory for an adequate management [1,13]. Thus, the suppression of ovarian hormones (sex steroids) is typically done through estro-progestives (combined oral contraceptives; PM controlling is part of the non-contraceptive benefits of contraceptives), while severe situations require central suppression which is done by GnRH (gonadotropin-releasing hormone) analogues [1,15]. Central neurotransmitters anomalies are re-directed to a higher level of serotonin *via* using SSRIs (selective serotonin re-uptake inhibitors) which is first line therapy, and, in cases with major depressive elements, anti-depressants drugs like tricyclic antidepressants might be used [1,13,14]. Lifestyle intervention, alternative medicine like herbal plants, acupuncture, supplementation of calcium and vitamin D, →

Figure 1. Management of premenstrual syndrome.

Blue boxes represent the diagnostic approach; purple boxes represent the pathogenic elements; light blue boxes include traditional therapy; brown boxes represents the elements that are brought by cardio-metabolic features (if present) in terms of recognition and management. Identification of cardio-metabolic features in females with premenstrual syndrome requires a specific intervention in terms of life style habits and drug regimens like anti-hypertensive medication (Abbreviations: PM = premenstrual syndrome, SSRI = selective serotonin re-uptake inhibitors, COC = combined oral contraceptives, GnRH = Gonadotropin Releasing Hormone)



cognitive behavior therapy might be useful, in addition to controlling the comorbidities [12,16,17,18].

AIM
The purpose is to focus on PM in terms of cardio-metabolic elements that may be diagnosed in PM positive females.

METHODS
This is a narrative review. The inclusion criteria are: full length paper, English published articles, PubMed research using the key words: “premenstrual syndrome” or the combination of “premenstrual syndrome” and “high blood pressure” (or “arterial hypertension”), alternative with “diabetes” (or “glucose”), “obesity” (or “body mass index”), respective “dyslipidemia” (or “hyperlipemia” or “cholesterol”). We also included relevant data on PMDD.

The article is organized in several subsections following the specific cardio-metabolic features we searched. We cited papers with different levels of statistical significance, from original studies to reviews, meta-analysis and experts’ opinions. We excluded articles of case report or case series type. We included 6 studies on HBP subsection, 3 studies on glucose profile and PM, 5 studies on body mass index among PM females, and two studies on lipid profile in PM.

GENERAL DATA
PM and high blood pressure

Arterial hypertension in PM women may be found either as a chronic disease, either as a pregnancy-related event, either a combination of both; moreover, variations of the daily blood pressure values is recognized at the moment when PM symptoms are manifested on otherwise normotensive females [14,19-23]. Whether the

prevalence of cardio-metabolic conditions is higher in PM females *versus* persons without PM is still an open issue [19-23].

New data suggests that approximately one in ten PM females associates HBP [14]. A longitudinal study from 2021 on 7729 females (an extract from Australian Longitudinal Study on Women's Health) identified 9.8% of them with chronic high blood pressure and 10.8% with pregnancy-related hypertension among the subgroup with a prior pregnancy; also, chronic hypertension associates a statistically significant risk of MC irregularities (relative risk of 1.42, 1.17-1.72) [14].

Despite baseline normal values of arterial tension, the MC-related symptoms correlate with higher values of blood pressure [19]. A prospective study on 370 females who experienced at least one symptom of PM during MC revealed a statistically significant higher blood pressure (mean, systolic, diastolic values) during luteal phase *versus* follicular phase [19]. It was suggested that BP fluctuations are due to arterial stiffness and hemodynamics changes that are induced by ovarian steroids pattern in PM. A study on 21 persons with PM *versus* 15 controls showed statistically significant higher arterial stiffness (based on pulse-wave velocity assessment) during luteal phases with synchronous changes of BP, thus BP monthly variability might be due to the presence of PM [20].

A blind randomized study on 40 females (mean age of 43 years) with PM and refractory HBP treated half of them with SSRI (sertraline 50 mg/day) and the other subgroup (control) without SSRI for 5 weeks; SSRI group had a statistically significant lower diastolic, not systolic BP, a results that makes an important point in management of severe BP in women of reproductive age with PM [21].

A transversal study on 408 individuals of mean age 21 years, enrolled between 2006 and 2014, identified 19% of them with PM; diastolic (not systolic) BP was statistically significant higher than control ($p = 0.02$) [22].

A prospective study included 1257 females (a sub-population from Nurses' Health Study II) with PM *versus* 2463 controls (the study was conducted between 1991 and 2005); HBP was identified in 342, respective 541 women; PM is found to be associated with a risk of developing HBP (hazard ratio of 1.4, CI 95%: 1.2-1.6), independent of age, smoking status and body mass index [23].

PM and glucose profile

In terms of PM and glucose profile, there is a limited amount of published data. No specific data/studies (with high quality statistics) pointing out the prevalence of type 2 diabetes mellitus in PM population are reported yet. Generally, we know that among lifestyle interventions, regular physical exercise might help, with potential improvement of glycaemia and insulin secretion and sensibility. [24].

We mention a comparative, cross-sectional study on glucose profile in PM women in relationship to MC phases that included 30 individuals and found that glycaemia and insulin resistance as measured by HOMA-IR (model assessment of insulin resistance) are lower during luteal and follicular phase *versus* control [25].

Anomalies of food behavior during luteal phase represent an element of PM/PMDD that might complicate the glucose profile and body mass index, underlying leptin and insulin release, but the data we have so far are reduced [26]. A study on 20 women with PMDD *versus* 18 controls showed significant changes of insulin, leptin and HOMA-IR between MC phases [26]. On the other hand, the use of GnRH in selected cases might impair the metabolic profile due to central hypogonadism [27].

PM and obesity

It is controversial if cyclic food cravings are direct contributors to body mass index variations [28-33]. However, the prevalence of PM elements among obese females of reproductive age is higher when compare to females with normal body mass index, according to majority of studies, but not all [28-33].

A cross-sectional study on 217 females of average age 28.8 +/- 7.9 years showed that, if PM elements are present, there is a higher calorie and carbohydrate intake, while the frequency of PM symptoms is higher in obesity and overweight group *versus* normal weight [29].

A prospective study published in 2020 on 63 females with PMDD *versus* 53 controls found that BMI is higher in PMDD group in addition to higher values of leptin during early and late luteal phases, while leptin increased from early to late phase with no changes of ghrelin levels [30]. In PMDD, body mass index correlates with variations of leptin levels between early and late luteal phases, with the presence of depression symptoms during late luteal phase and also with the identification of emotional eating behavior [30].

A transversal study on 1702 persons aged between 8 and 23 years identified a 19.3% prevalence of PM, respective of 4.6% for PMDD based on PSST-A (premenstrual symptoms screening tool for adolescents); among these, multivariate regression showed that younger age at menarche in addition to junk food intake are correlated with PMDD [31].

A longitudinal study on 1057 people with PM, aged between 27 and 44 years *versus* 1959 controls showed a correlation between body mass index and PM, at every 1 kg/sqm increase of body mass index there was a 3% increase of PM risk [32]. The risk of PM is higher in individuals with body mass index above or equal to 27.5 kg/sqm *versus* less than 20 kg/sqm at baseline [32]. Cyclic body mass index variations according to MC phases were not confirmed [32]. Increased body mass index correlates with the presence of abdominal cramps, back pain and swelling of legs [32].

On the contrary, some data showed that PM is more frequent among normal weight subjects [33]. For instance, we mention a study on 476 individuals (233 with PM and 243 without PM) showing that PM is two times more frequent in normal weight women *versus* those with BMI higher or equal with 25 kg/sqm (68% *versus* 31%), while PM is less frequent in females with higher fat mass as assessed through bioelectrical impedance [33].

To a larger extent, some data reported the association between PM or PMDD and eating disorders like bulimia →

nervosa or binge-eating (independent of food cravings in PM) [34-40]. Moreover, the presence of PM represents a marker of ovarian sensitivity in patients with eating disorders [34-40].

PM and lipid profile

Lipids profile may be correlated with hormonal levels and their variations among MC. Ovary-related estrogens are key players in different conditions like endometriosis, endometrial hyperplasia, excessive uterine bleeding, but also in aggravating HBP or hypercholesterolemia in females under oral contraceptives due to estrogen-associated effects on hepatic production of liver proteins, including the substrate of renin-angiotensin-aldosterone system [41,42,43]. Thus managing PM with combined oral contraceptives needs a careful cardio-metabolic co-morbidities evaluation due to potential side effects [41,42,43].

A community-based transversal study on 354 females with PM versus 302 healthy persons representing the control group identified statistically significant higher levels of triglycerides and lower values of HDL (high density lipoprotein); also, at every unit increase of PM score, the probability of metabolic syndrome increases by 12% after adjustment for age and body mass index [44]. Another study on 34 subjects with PMDD and 20 controls found that premenstrual cholesterol is lower in PM free group while the prevalence of premenstrual high levels of cholesterol above 200 mg/dL is 23% in PM group and zero in PM negative individuals [45].

DISCUSSIONS

Overall, the cardio-metabolic features in PM females represent a challenging topic and literature data we have so far is not clear in many areas. Checking PM elements in females with refractory high blood pressure seems a logical step in multidisciplinary management. Another topic with insufficient data concerns PMDD and associated metabolic features, there is not enough information to speculate a more severe metabolic profile if the elements of PMDD diagnostic are more severe than typical PM.

CONCLUSIONS

Whether PM females are prone to cardio-metabolic comorbidities or they are incidental due to high prevalence in general reproductive female population of both conditions is still on open subject. Understanding the cardio-metabolic features in PM is essential nowadays as modern society is oriented to a better management of conditions that impair the quality of life, noting that PM has a high epidemiological impact due to increased prevalence in general reproductive female population.

Conflict of interest: none

Acknowledgement: none

References

1. Dilbaz B, Aksan A. Premenstrual syndrome, a common but underrated entity: review of the clinical literature. *J Turk Ger Gynecol Assoc.* 2021 May 28;22(2):139-148. doi: 10.4274/jtgga.galenos.2021.2020.0133.
2. Hall E, Steiner M. Psychiatric symptoms and disorders associated with reproductive cyclicality in women: advances in screening tools. *Womens Health (Lond).* 2015 Jun;11(3):397-415. doi: 10.2217/whe.15.1.
3. Romans S, Clarkson R, Einstein G, Petrovic M, Stewart D. Mood and the menstrual cycle: a review of prospective data studies. *Gend Med.* 2012 Oct;9(5):361-84. doi: 10.1016/j.genm.2012.07.003.
4. Hartlage SA, Freels S, Gotman N, Yonkers K. Criteria for premenstrual dysphoric disorder: secondary analyses of relevant data sets. *Arch Gen Psychiatry.* 2012 Mar;69(3):300-5. doi: 10.1001/archgenpsychiatry.2011.1368.
5. Kaye SL. An Overview of Premenstrual Voice Syndrome: Definition, Treatment, and Future Trajectories. *Med Probl Perform Art.* 2020 Mar;35(1):59-65. doi: 10.21091/mppa.2020.1008.
6. Roomruangwong C, Maes M. Biomarker Validation of a New Case Definition of Menstrual Cycle-Associated Syndrome (MCAS) Opinion Paper. *CNS Neurol Disord Drug Targets.* 2021;20(2):105-111. doi: 10.2174/1871527319666200930095149.
7. Roomruangwong C, Sirivichayakul S, Carvalho AF, Maes M. The uterine-chemokine-brain axis: menstrual cycle-associated symptoms (MCAS) are in part mediated by CCL2, CCL5, CCL11, CXCL8 and CXCL10. *J Affect Disord.* 2020 May 15;269:85-93. doi: 10.1016/j.jad.2020.03.033.
8. Kadian S, O'Brien S. Classification of premenstrual disorders as proposed by the International Society for Premenstrual Disorders. *Menopause Int.* 2012 Jun;18(2):43-7. doi: 10.1258/mi.2012.012017.
9. Dennerstein L, Lehert P, Heinemann K. Epidemiology of premenstrual symptoms and disorders. *Menopause Int.* 2012 Jun;18(2):48-51. doi: 10.1258/mi.2012.012013.
10. Mahfoud Z, Emam R, Anchassi D, Omran S, Alhaj N, Al-Abdulla S, El-Amin A, Shehata M, Aly S, Al Emadi N, Al-Meer F, Al-Amin H. Premenstrual dysphoric disorder in Arab women: Validation and cultural adaptation of the Arabic version of the premenstrual screening tool. *Women Health.* 2019 Jul;59(6):631-645. doi: 10.1080/03630242.2018.1539433.
11. McEvoy K, Osborne LM, Nanavati J, Payne JL. Reproductive Affective Disorders: a Review of the Genetic Evidence for Premenstrual Dysphoric Disorder and Postpartum Depression. *Curr Psychiatry Rep.* 2017 Oct 30;19(12):94. doi: 10.1007/s11920-017-0852-0.
12. Poiana C, Musat M, Carsote M, Chirita C. Premenstrual dysphoric disorder: neuroendocrine interferences, *Rev Med Chir Soc Med Nat Iasi.* 2009;113(4):996-1000.
13. Ismaili E, Walsh S, O'Brien PMS, Bäckström T, Brown C, Dennerstein L, Eriksson E, Freeman EW, Ismail KMK, Panay N, Pearlstein T, Rapkin A, Steiner M, Studd J, Sundström-Paromma I, Endicott J, Epperson CN, Halbreich U, Reid R, Rubinow D, Schmidt P, Yonkers K; Consensus Group of the International Society for Premenstrual Disorders. Fourth consensus of the International Society for Premenstrual Disorders (ISPMDD): auditable standards for diagnosis and management of premenstrual disorder. *Arch Womens Ment Health.* 2016 Dec;19(6):953-958. doi: 10.1007/s00737-016-0631-7.
14. Chung HF, Ferreira I, Mishra GD. The association between menstrual symptoms and hypertension among young women: A prospective longitudinal study. *Maturitas.* 2021 Jan;143:17-24. doi: 10.1016/j.maturitas.2020.08.006.

References continues on the
next page

References continues from the previous page

15. Schindler AE. Non-contraceptive benefits of oral hormonal contraceptives. *Int J Endocrinol Metab.* 2013 Winter;11(1):41-7. doi: 10.5812/ijem.4158.
16. Naheed B, Kuiper JH, Uthman OA, O'Mahony F, O'Brien PM. Non-contraceptive oestrogen-containing preparations for controlling symptoms of premenstrual syndrome. *Cochrane Database Syst Rev.* 2017 Mar 3;3(3):CD010503. doi: 10.1002/14651858.CD010503.pub2.
17. Hofmeister S, Bodden S. Premenstrual Syndrome and Premenstrual Dysphoric Disorder. *Am Fam Physician.* 2016 Aug 1;94(3):236-40.
18. Sharifi F, Simbar M, Mojab F, Majd HA. Comparison of the effects of *Matricaria chamomila* (Chamomile) extract and mefenamic acid on the intensity of premenstrual syndrome. *Complement Ther Clin Pract.* 2014 Feb;20(1):81-8. doi: 10.1016/j.ctcp.2013.09.002.
19. Danborn AM, Nwankwo M, Kure J, Eluwa C. Prevalence of Premenstrual Syndrome and Changes in Blood Pressure with Menstrual Cycle Among University Students. *Niger J Physiol Sci.* 2018 Dec 30;33(2):117-124.
20. Stamatielopoulos KS, Georgiopoulos G, Papaioannou T, Lambrioudaki I, Kouzoupis A, Vlachopoulos C, Georgiou SP, Manios E, Alevizaki M, Papamichael CM, Sfrikakis PP. Can premenstrual syndrome affect arterial stiffness or blood pressure? *Atherosclerosis.* 2012 Sep;224(1):170-6. doi: 10.1016/j.atherosclerosis.2012.05.037.
21. Ranjbar F, Akbarzadeh F, Asadlou M. The Effects of Sertraline in Controlling Refractory Hypertension in Women with Premenstrual Syndrome. *Iran J Psychiatry.* 2016 Oct;11(4):234-238.
22. Bertone-Johnson ER, Houghton SC, Whitcomb BW, Sievert LL, Zagarins SE, Ronnenberg AG. Association of Premenstrual Syndrome with Blood Pressure in Young Adult Women. *J Womens Health (Larchmt).* 2016 Nov;25(11):1122-1128.
23. Bertone-Johnson ER, Whitcomb BW, Rich-Edwards JW, Hankinson SE, Manson JE. Premenstrual Syndrome and Subsequent Risk of Hypertension in a Prospective Study. *Am J Epidemiol.* 2015 Dec 15;182(12):1000-9. doi: 10.1093/aje/kwv159.
24. Orio F, Muscogiuri G, Ascione A, Marciano F, Volpe A, La Sala G, Savastano S, Colao A, Palomba S. Effects of physical exercise on the female reproductive system. *Minerva Endocrinol.* 2013 Sep;38(3):305-19.
25. Zarei S, Mosalanejad L, Ghobadifar MA. Blood glucose levels, insulin concentrations, and insulin resistance in healthy women and women with premenstrual syndrome: a comparative study. *Clin Exp Reprod Med.* 2013 Jun;40(2):76-82. doi: 10.5653/ceerm.2013.40.2.76.
26. Akturk M, Toruner F, Aslan S, Altinova AE, Cakir N, Elbeg S, Arslan M. Circulating insulin and leptin in women with and without premenstrual dysphoric disorder in the menstrual cycle. *Gynecol Endocrinol.* 2013 May;29(5):465-9. doi: 10.3109/09513590.2013.769512.
27. Nguyen TV, Reuter JM, Gaikwad NW, Rotroff DM, Kucera HR, Motsinger-Reif A, Smith CP, Nieman LK, Rubinow DR, Kaddurah-Daouk R, Schmidt PJ. The steroid metabolome in women with premenstrual dysphoric disorder during GnRH agonist-induced ovarian suppression: effects of estradiol and progesterone addback. *Transl Psychiatry.* 2017 Aug 8;7(8):e1193. doi: 10.1038/tp.2017.146.
28. McNeil J, Doucet É. Possible factors for altered energy balance across the menstrual cycle: a closer look at the severity of PMS, reward driven behaviors and leptin variations. *Eur J Obstet Gynecol Reprod Biol.* 2012 Jul;163(1):5-10. doi: 10.1016/j.ejogrb.2012.03.008.
29. Elliott SA, Ng J, Leow MK, Henry CJ. The influence of the menstrual cycle on energy balance and taste preference in Asian Chinese women. *Eur J Nutr.* 2015 Dec;54(8):1323-32. doi: 10.1007/s00394-014-0812-y.
30. Yen JY, Lin HC, Lin PC, Liu TL, Long CY, Ko CH. Leptin and ghrelin concentrations and eating behaviors during the early and late luteal phase in women with premenstrual dysphoric disorder. *Psychoneuroendocrinology.* 2020 Aug;118:104713. doi: 10.1016/j.psyneuen.2020.104713.
31. Kamat SV, Nimbalkar A, Phatak AG, Nimbalkar SM. Premenstrual syndrome in Anand District, Gujarat: A cross-sectional survey. *J Family Med Prim Care.* 2019 Feb;8(2):640-647. doi: 10.4103/jfmpc.jfmpc_302_18.
32. Bertone-Johnson ER, Hankinson SE, Willett WC, Johnson SR, Manson JE. Adiposity and the development of premenstrual syndrome. *J Womens Health (Larchmt).* 2010 Nov;19(11):1955-62. doi: 10.1089/jwh.2010.2128.
33. Mizgier M, Jarzabek-Bielecka G, Jakubek E, Kedzia W. The relationship between body mass index, body composition and premenstrual syndrome prevalence in girls. *Ginekol Pol.* 2019;90(5):256-261. doi: 10.5603/GP.2019.0048.
34. Nobles CJ, Thomas JJ, Valentine SE, Gerber MW, Vaewsorn AS, Marques L. Association of premenstrual syndrome and premenstrual dysphoric disorder with bulimia nervosa and binge-eating disorder in a nationally representative epidemiological sample. *Int J Eat Disord.* 2016 Jul;49(7):641-50. doi: 10.1002/eat.22539.
35. Czajkowska M, Drosdzol-Cop A, Gałazka I, Naworska B, Skrzypulec-Plinta V. Menstrual Cycle and the Prevalence of Premenstrual Syndrome/Premenstrual Dysphoric Disorder in Adolescent Athletes. *J Pediatr Adolesc Gynecol.* 2015 Dec;28(6):492-8. doi: 10.1016/j.jpag.2015.02.113.
36. Ryan S, Ussher JM, Hawkey A. Managing the premenstrual body: a body mapping study of women's negotiation of premenstrual food cravings and exercise. *J Eat Disord.* 2021 Oct 9;9(1):125. doi: 10.1186/s40337-021-00478-6.
37. Badrasawi MM, Zidan SJ, Natour N, Sharif I, Atrash S, Abueid G, Al-Jounde S. Binge eating symptoms are associated with the severity of premenstrual symptoms among university students, cross sectional study from Palestine. *J Eat Disord.* 2021 Jun 9;9(1):68. doi: 10.1186/s40337-021-00425-5.
38. Hildebrandt BA, Racine SE, Keel PK, Burt SA, Neale M, Boker S, Sisk CL, Klump KL. The effects of ovarian hormones and emotional eating on changes in weight preoccupation across the menstrual cycle. *Int J Eat Disord.* 2015 Jul;48(5):477-86. doi: 10.1002/eat.22326.
39. Çoban ÖG, Karakaya D, Önder A, İşleyen Z, Adanır AS. Association of Premenstrual Dysphoric Disorder and Eating Behaviors Among Nursing Students: A Cross-Sectional Study. *J Pediatr Adolesc Gynecol.* 2021 Apr;34(2):203-208. doi: 10.1016/j.jpag.2020.11.019.
40. Hardin SL, Thornton LM, Munn-Chernoff MA, Baker JH. Premenstrual symptoms as a marker of ovarian hormone sensitivity in eating disorders. *Int J Eat Disord.* 2020 Feb;53(2):296-301. doi: 10.1002/eat.23213.
41. Carsote M, Terzea DC, Valea A, Gheorghisan-Galateanu AA. Abdominal wall endometriosis (a narrative review). *International Journal of Medical Sciences (Int J Med Sci).* 2020;17(4):536-542. doi:10.7150/ijms.38679.
42. Sabbatini AR, Kararigas G. Estrogen-related mechanisms in sex differences of hypertension and target organ damage. *Biol Sex Differ.* 2020 Jun 1;11(1):31. doi: 10.1186/s13293-020-00306-7.
43. Ribeiro CCM, Shimo AKK, Lopes MHB, Lamas JLT. Effects of different hormonal contraceptives in women's blood pressure values. *Rev Bras Enferm.* 2018;71(suppl 3):1453-1459. doi: 10.1590/0034-7167-2017-0317.
44. Hashemi S, Ramezani Tehrani F, Mohammadi N, Rostami Dovom M, Torkestani F, Simbar M, Azizi F. Comparison of Metabolic and Hormonal Profiles of Women With and Without Premenstrual Syndrome: A Community Based Cross-Sectional Study. *Int J Endocrinol Metab.* 2016 Feb 14;14(2):e28422. doi: 10.5812/ijem.28422.
45. Hsiao MC, Liu CY, Hsu SC, Hsiao CC, Lin YH, Hsieh TT. Elevated serum cholesterol levels in women with premenstrual dysphoric disorder. *Int J Psychiatry Med.* 2011;42(1):85-92. doi: 10.2190/PM.42.1.f.