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Correlation Between 25-Hydroxy-Vitamin D [25(Oh) D] Levels And Polycystic Ovary Syndrome In Obese Women: A Case-Control Study

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) occurs around 38-88% in overweight or obese women. Obese women with PCOS tend to have lower serum vitamin D levels compared to lean women with PCOS.

Objective: This study aimed to analyze the relationship between 25-hydroxy-vitamin D [25(OH)D] levels in obese women with PCOS.

Methods: This case-control study was conducted on 80 obese women aged between 26-35 yrs old from January to December 2024 at Hasanuddin University hospital and Wahidin Sudirohusodo hospital, Makassar, Indonesia. All participants were divided into 2 groups: women with PCOS (n=40) and women without PCOS (n=40).

All women were measured for body mass index (BMI) and Serum 25(OH)D levels were measured using enzyme-linked immunosorbent assay ELISA.

Results: This study found that obese women with PCOS had 25(OH)D levels that were significantly lower than obese women without PCOS (p value=0.004). However, women with PCOS tend to have a higher BMI than women without PCOS (p value=0.052). Low 25(OH)D levels are weak correlation with increased BMI (r=-0.265, p value=0.017).

Conclusion: Vitamin D levels were low in obese women with PCOS, therefore vitamin D supplementation in PCOS may be recommended.

Keywords: *Vitamin D, obesity, polycystic ovary syndrome*

1. INTRODUCTION

Polycystic ovary syndrome (PCOS) is a condition characterized by irregular menstruation, increased androgens, and/or the presence of small cysts on the ovaries [1]. The prevalence of PCOS occurs in 6-20% of women of reproductive age [2]. PCOS is influenced by genetic conditions that are worsened by obesity. Women with PCOS occur overweight or obese about 38-88% (Barber, 2022). Obesity is associated with low levels of vitamin D. High body fat content in obese women acts as a reservoir of fat-soluble vitamin D and increases vitamin D absorption, resulting in low vitamin D bioavailability [3].

Vitamin D deficiency leads to impaired fertility and PCOS [4]. Vitamin D deficiency in women with PCOS is reported to be around 67-85%, with serum 25(OH)D concentrations <20 ng/ml [5]. The association between vitamin D deficiency, obesity, and PCOS may be explained by the fact that in obese women, circulating 25(OH)D levels are lower due to increased absorption of fat-soluble vitamins in adipose tissue, and the tendency of obese women to avoid sun exposure. Vitamin D plays a regulatory role in androgen homeostasis, potentially through its interactions with sex hormone-binding globulin (SHBG) and parathyroid hormone. Deficiency in vitamin D has been associated with reduced SHBG concentrations, elevated levels of circulating free androgens, and an aggravation of hyperandrogenic features in women with PCOS [5].

Previous studies have also reported an association between vitamin D deficiency, obesity, and PCOS. Ibrahim and colleagues conducted a study in Egypt with the result that there is a relationship between vitamin D deficiency in obese women with

PCOS. Serum 25(OH) D was significantly lower in the PCOS group compared to the control group and it was found that serum 25(OH)D was significantly lower in obese PCOS women compared to non-obese PCOS women [6]. Similar results were reported that vitamin D levels were lower in women with PCOS compared to controls. In the PCOS group, vitamin D levels were lower in obese women [7]. However, different results were reported in India that there was no significant difference in vitamin D levels in the PCOS and control groups and obese and non-obese groups [8].

Previous studies have reported different results regarding the relationship between vitamin D levels and obesity and PCOS. Previous studies have compared vitamin D levels in obese and non-obese PCOS women, but have not compared obese women with and without PCOS. This study attempts to examine the relationship between vitamin D levels in obese women with and without PCOS. This study is expected to provide information on the role of vitamin D in predicting the occurrence of PCOS in obese women. Therefore, this study is interested in analyzing the relationship between vitamin D levels in obese women with PCOS.

2. MATERIALS AND METHODS

This case-control study was conducted on 80 obese women from January to December 2024 at the Hasanuddin University hospital and Wahidin Sudirohusodo hospital, Makassar, Indonesia. After PCOS diagnosis, participants were divided into 2 groups: women with PCOS (n=40) and women without PCOS (n=40). Demographic and clinical characteristics of the subjects including age, body mass index (BMI), residence, marital status, parity, and comorbidities were collected through medical records. The diagnosis of PCOS was established based on participants' medical history, physical examination, and the Rotterdam criteria. Based on the Rotterdam criteria, a diagnosis of PCOS requires at least two of the following three features: (1) oligomenorrhea or amenorrhea, defined as fewer than eight menstrual cycles per year or menstrual intervals exceeding 35 days (based on questionnaire) ; (2) Clinical signs of hyperandrogenism. Hyperandrogenism was assessed through physical examination, and hirsutism was clinically evaluated using the Ferriman-Gallwey scoring system, which examines nine body areas: the upper lip, chin, chest, upper back, lower back, upper arms, upper abdomen, lower abdomen, and thighs. (3) polycystic ovarian morphology on ultrasound, characterized by at least one ovary containing 12 or more peripheral follicles measuring 2–9 mm in diameter and/or an ovarian volume of ≥10 mL. Transvaginal ultrasound is preferred for imaging when the individual is sexually active and consents to the procedure. BMI was assessed using Asia-Pacific guidelines, individuals with a BMI of less than 18.5 kg/m² are categorized as underweight, 18.5–22.9 kg/m² as normal, 23.0–24.9 kg/m² as overweight (at risk), 25.0–29.9 kg/m² as obese class I, and ≥30.0 kg/m² as obese class II Serum 25(OH) D levels were measured using enzyme-linked immunosorbent assay ELISA method.

A structured questionnaire was utilized to gather socio-demographic information from the study participants. Obstetric history and clinical manifestations of hyperandrogenism, such as acne and hirsutism, were also documented by a designated nursing officer using a standardized format. Anthropometric measurements including height (in centimeters), weight (in kilograms), body mass index (BMI), were obtained using standardized procedures and calibrated instruments. BMI was calculated by dividing body weight in kilograms by the square of height in meters (kg/m²). Real-time ultrasonography was performed using a three-dimensional ultrasound machine equipped with a high-resolution 6 MHz probe. Transvaginal ultrasonography was the preferred method for women who were sexually active. Transabdominal USG was preffered for unmarried patient. Ovarian assessment included polycystic ovaries (≥ 12 follicles measuring 2-9 mm in diameter and/or an ovarian volume > 10 mL in at least one ovary).

Serum vitamin D levels were measured using the enzyme-linked immunosorbent assay (ELISA) method. Blood samples were collected under aseptic conditions, and serum was separated by centrifugation. The ELISA procedure was performed according to the manufacturer's instructions using a commercially available 25-hydroxyvitamin D ELISA kit. The assay quantitatively determined the concentration of 25(OH)D in serum samples, which is considered the most reliable indicator of vitamin D status. Absorbance was read at the specified wavelength using a microplate reader, and vitamin D levels were interpreted based on standard reference ranges provided by the kit.

Inclusion criteria were obesity with Body Mass Index (BMI) ≥ 25 kg/m2 and age between 18-35 yrs. Women with a history of consuming drugs such as antiseizure, rifampicin, and antiretroviral in the last 3 months, having comorbidities such as liver disease, kidney disease, and endocrine disorders such as diabetes mellitus and cancer, a history of consuming vitamin D supplements in the last 3 months, a history of consuming hormonal drugs in the last 3 months, a history of using sunscreen in the last 3 months, and a history of doing physical activity or intensive sports (> 2 hours/day) in the last 3 months were excluded from the study.

3. RESULTS

This study obtained 80 obese women consisting of 40 women with PCOS and 40 women without PCOS. Both groups did not differ based on age, education, occupation, type of obesity, and sun exposure, where most of them were 26-35 years old, educated > 12 years, working, married, obese 1, and not exposed to the sun. Both groups differed based on parity where most women with PCOS were nulliparous while those without PCOS were multiparous (Table I).

Table 1. Characteristics of Research Subjects

Characteristics	PCOS (n = 40)	Non-PCOS (n = 40)	P-value
	n (%)	n (%)	
Age (yr)			
18-25 yrs	10 (25.0)	5 (12.5)	0.152[a]
26-35 yrs	30 (75.0)	35 (87.5)	
Education			
≤ 12 yrs	5 (12.5)	11 (27.5)	0.094[a]
> 12 yrs	36 (87.5)	29 (72.5)	
Employment			
Employeed	32 (80.0)	34 (85.0)	0.556[a]
Unemployeed	8 (20.0)	6 (15.0)	
Marital status			
Married	37 (92.5)	40 (100.0)	N.A
Unmarried	3 (7.5)	0 ().0)	
Parity			
Nulliparous	33 (82.5)	7 (17.5)	
Primiparous	3 (7.5)	16 (40.0)	< 0.001*[b]
Multiparous	4 (10.0)	17 (42.5)	
Obesity			
Obesity 1	22 (55.0)	30 (75.0)	0.061[a]
Obesity 2	18 (45.0)	10 (25.0)	
Sun Exposure			
Yes	12 (30.0)	17 (42.5)	0.245[a]
No	28 (70.0)	23 (57.5)	

[a]Chi square, [b]Fisher exact test, *p < 0.05.

N.A= Not Available, PCOS

The results showed that there was a significant difference in 25(OH)D levels between women with PCOS and without PCOS with a p-value <0.05. Obese women with PCOS had significantly lower 25(OH)D levels than obese women without PCOS. Based on body mass index, there was a tendency that women with PCOS had a higher BMI than those without PCOS but it was not statistically significant with a p-value > 0.05 (Table II).

Table 2. Comparison of 25(OH)D levels and body mass index (BMI) between women with and without PCOS

	PCOS (n = 40)	Non-PCOS (n = 40)	P-value
	mean ± SD	mean ± SD	
25(OH)D (ng/mL)	9.19 ± 3.63 ng/mL	12.99 ± 7.120 ng/mL	0.004*
BMI (kg/m^2)	29.68 ± 3.47 kg/m^2	28.32 ± 2.60 kg/m^2	0.052

Data presented as Mean ± SD, *Independent sample t test, *p < 0.05. Polycystic ovary syndrome =PCOS, Body Mass Index

=BMI, 25(OH) = Vitamin D

The results of the correlation test showed that BMI was significantly related to 25(OH)D levels with a p-value <0.05. The correlation value of -0.265, a negative sign indicates a negative relationship (inversely proportional) between BMI and 25(OH)D levels. The higher the BMI, the lower the 25(OH)D levels. The correlation value of 0.265 indicates a weak correlation between BMI and 25(OH)D levels (Figure 1).

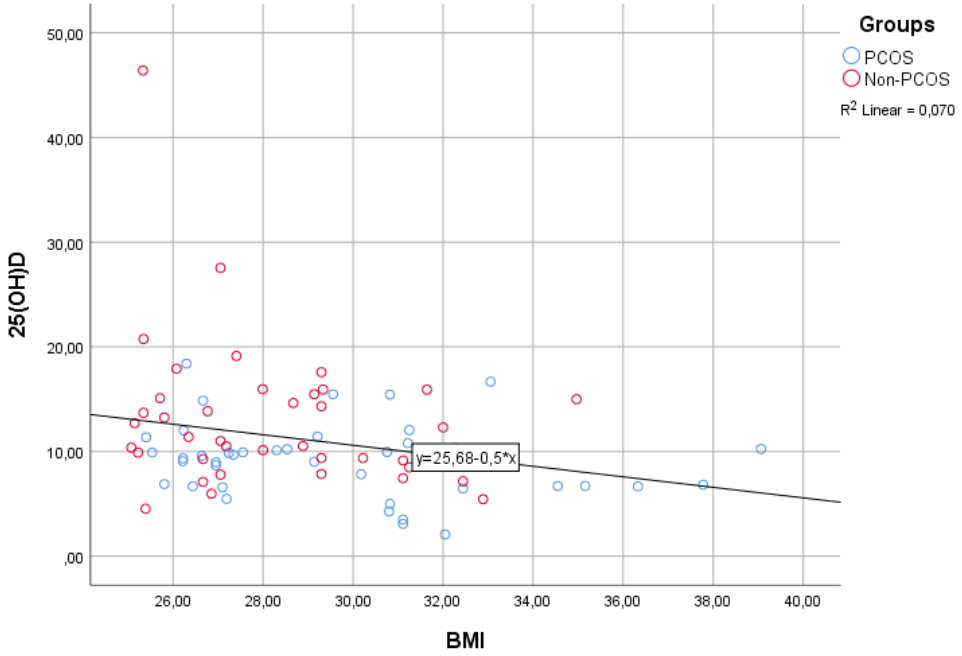

Figure 1. Correlation between BMI and 25(OH) D levels (r= -0.265, p value=0.017)

4. DISCUSSION

This This study was conducted on obese women who were 75% population aged 26-35 years, educated > 12 years, working, and married in both the PCOS and non-PCOS groups. Women with PCOS and without PCOS in this study had homogeneous characteristics based on age, education, occupation, and sun exposure. Thus, both groups have similar characteristics so they are worthy of comparison.

This study identified a significant difference in parity between the PCOS and non-PCOS groups. The majority of obese women with PCOS were nulliparous, whereas most obese women without PCOS were multiparous. These findings are consistent with previous research, which also demonstrated a disparity in parity between women with and without PCOS, where 66.3% of women with PCOS were nulliparous compared to 52.2% in the non-PCOS group [9]. Similarly, Talmo and colleagues reported that 54.9% of women with PCOS were nulliparous, while the majority of women without PCOS were either primiparous or multiparous (52.5%) [10]The difference in parity between the PCOS and non-PCOS groups can be explained by the risk of infertility in PCOS. The risk of PCOS makes women with PCOS tend to have lower parity than women without PCOS. This result is as reported that PCOS is the main cause of infertility and gynecological-endocrine disorders in 7 to 15% of women of reproductive age [11]. The mechanism explaining the cause of infertility in PCOS is the occurrence of insulin resistance and hyperinsulinemia due to the cyclical results of excess androgens that affect androgen secretion by the ovaries and adrenal glands. The cyclical pathogenetic interaction between hyperinsulinemia, hyperandrogenism and insulin resistance combined with hypothalamic-pituitary dysfunction, causes further ovarian dysfunction that can result in anovulation and infertility [12]. Hormonal dysfunction and inflammation of adipose tissue cause insulin resistance in PCOS. Abnormalities in lipid and glucose metabolism cause low-grade inflammation in the endothelium. Meanwhile, chronic inflammation causes the development of insulin resistance through adipokines released from adipose tissue. Insulin resistance disrupts ovulation due to excessive androgen secretion in the absence or reduction of progesterone concentrations. The disturbances caused by insulin resistance are maintained and exacerbated by hormonal and metabolic dysfunctions that cause immune dysregulation. The result is impaired oocyte maturation, and dysfunction of endometrial receptivity and abnormal growth of ovarian follicles that affect infertility [13].

The results of this study indicate that there is a significant difference in BMI between the PCOS and non-PCOS groups. BMI is greater in the PCOS group than without PCOS. This result is in line with the previous study that there is a relationship

between increased BMI and the risk of PCOS [14]. The severity of PCOS is associated with increased BMI. Similar results in the study by Jalilian, Haghnazari, and Rasolinia that the average BMI of PCOS was significantly higher in women with PCOS compared to those without PCOS [15].

The relationship between obesity and PCOS can be explained that obesity causes inflammation. The greater the BMI, the higher the inflammation that occurs. A higher the BMI also shows that the accumulation of adipocytes in visceral fat that is getting bigger causes increased hypoxia and necrosis, which causes increased production of inflammatory cytokines. Obesity also causes hyperinsulinemia and insulin resistance through increased levels of unesterified fatty acids (NEFA) in the blood. NEFA is used by skeletal muscles as an energy source and does not use glucose as an energy source so that hyperglycemia occurs resulting in hyperinsulinemia and rapid pancreatic reaction [16]. Because insulin directly inhibits SHBG production in the liver, it increases free androgen levels and hyperandrogenemia occurs in PCOS [17]. High adiposity also results in high leptin levels that inhibit aromatase mRNA expression in granulosa cells. This interferes with the conversion of androgens to estrogens, resulting in hyperandrogenemia in PCOS [16]. The level of obesity was not related to PCOS in this study because this study was conducted only on all obese women.

This study found that obese women with PCOS had lower levels of 25(OH)D than women without PCOS. The results of this study are in line with the research of Mogili and colleagues that vitamin D deficiency is associated with PCOS. Vitamin D deficiency occurs in 70.3% of women with PCOS [18]. Similar results in the study by Cahyo, Yuad, and Rahmah that there is a relationship between PCOS in adolescents accompanied by hyperandrogen with vitamin D levels and there was a relationship between PCOS in obese adolescents with vitamin D levels. In this study, the prevalence of vitamin D deficiency in PCOS patients was 58.8%. In this study, the prevalence of vitamin D deficiency occurred in 96.3% of women, which is greater than previous studies. This is because this study was conducted on obese women, while previous studies were conducted on women with various BMIs [19].

The high prevalence of vitamin D deficiency in women with PCOS and obesity occurs because vitamin D is fat-soluble [20]. In obesity, there is an increase in fat, so there is an increase in fat-soluble vitamin D. This causes serum vitamin D levels to decrease [21]. The mechanisms explaining the relationship between vitamin D deficiency and PCOS are related to insulin resistance, obesity, and increased androgen levels [22]. Vitamin D deficiency increases parathyroid hormone (PTH) production because PTH production is regulated by serum vitamin D levels. Increased PTH is associated with menstrual disorders in PCOS [20]. Vitamin D deficiency increases LH due to vitamin D receptor polymorphism and altered aromatase gene expression in vitamin D deficiency [23]. Increased LH in serum causes excess androgen production, leading to hyperandrogenism in PCOS [24].

The results of a review study reported that the prevalence of vitamin D deficiency in Southeast Asia was 22.0% [25]. A meta-analysis study in Indonesia reported that vitamin D deficiency in pregnant women aged 27.6–30.6 years was 63% [26]. Vitamin D deficiency in women of childbearing age (18-35 years) who are not pregnant in Bandung is 16.67% [27]. Research on women aged 16-44 years in Jambi reported a higher prevalence of vitamin D deficiency, namely 52% [28].

According to the Regulation of the Minister of Health of the Republic of Indonesia No. 28 of 2019 on Recommended Dietary Allowances, the recommended daily intake of vitamin D for women aged 19–49 years is 15 micrograms (mcg) per day. [29]. In a meta-analysis study it was reported that continuous administration of vitamin D at a dose of less than 4000 IU/day can improve insulin resistance and glucose metabolism, thereby preventing menstrual disorders and hyperandrogenism. Research on infertile women with PCOS, administration of vitamin D at a dose of 50,000 IU every two weeks for 8 weeks in women who will undergo in vitro fertilization (IVF) can reduce Anti-Mullerian Hormone (AMH) and insulin levels in the blood. Vitamin D supplementation is able to improve insulin metabolism and lipid profiles in infertile women with PCOS [30].

Regarding factors related to 25(OH)D levels, this study found that there was a relationship and inverse relationship between BMI and 25(OH)D levels. Obese women with higher BMI are associated with lower 25(OH)D levels. These results are in line with research by Suhartono, Astiarani, and Regina that there is a relationship between increased BMI and decreased serum vitamin D [21]. Similar results were reported by Alloubani and colleagues' study in Saudi Arabia, which stated that there was a strong relationship between increased BMI and decreased serum Vitamin D levels [31].

Several theories explain the relationship between BMI and Vitamin D levels. The sequestration theory states that vitamin D is a fat-soluble vitamin. When there is an increase in fat in an increase in BMI, there is an increase in vitamin D in fat. This causes serum vitamin D levels to decrease. In this study, obesity 2 with a BMI greater than obesity 1 had a higher fat content so fat-soluble vitamin D was greater so serum vitamin D decreased. In addition, in individuals with a larger BMI, vitamin D is distributed in a larger volume, causing serum concentrations to appear lower. Genetic factors also play a role in the mechanism between vitamin D and obesity. Decreased expression of the cytochrome P450 2J2 gene which encodes the enzyme 25-hydroxylase and decreased expression of the cytochrome P450 27B1 gene which encodes the enzyme 1-α hydroxylase results in inhibition of the Vitamin D synthesis process. In addition, sedentary lifestyles carried out by obesity sufferers can also affect the process of vitamin D synthesis and lifestyles that tend to avoid exposure to sunlight (little outdoor activity) so that vitamin D synthesis is inhibited which further reduces vitamin D levels [21].

Research related to the relationship between vitamin D levels and PCOS has been widely conducted, but none have been specifically studied in obese women. This is the advantage of this study. This study only analyzed the relationship between vitamin D levels and PCOS. Various mechanisms and markers are involved in the relationship that were not studied in this study, such as luteinizing hormone (LH), parathyroid hormone (PTH), inflammatory cytokines, insulin resistance, and others.

5. LIMITATIONS

This study has several limitations that should be considered when interpreting the results. First, the sample size was relatively small (n=80), which may limit the generalizability of the findings to broader populations. Future studies with larger cohorts are needed to confirm these results. Second, the cross-sectional design of this case-control study does not allow for the establishment of a causal relationship between low 25(OH)D levels and the development of PCOS in obese women. Longitudinal studies would be more appropriate to determine causality. Third, although we attempted to control for potential confounders, such as age and BMI, other factors that could influence vitamin D levels such as dietary intake, physical activity, and genetic polymorphisms were not assessed in this study. These unmeasured variables may have influenced the observed associations. Additionally, the diagnosis of PCOS was based on clinical and ultrasound criteria, without including hormonal profiles such as serum androgen levels, which could provide a more comprehensive characterization of the condition.

6. CONCLUSION

Obese women with PCOS have lower vitamin D levels than obese women without PCOS. Low vitamin D levels are associated with increased body mass index. Higher BMI can be a risk factor for vitamin D deficiency and vitamin D testing can be useful as a predictor of PCOS. Therefore, vitamin D supplementation in PCOS may be recommended. Similar studies can be conducted using other markers for PCOS prediction to further clarify the mechanism of the relationship between vitamin D deficiency and PCOS.

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8. CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

[1] Ndefo UA, Eaton A, Green MR. Polycystic ovary syndrome: A review of treatment options with a focus on pharmacological approaches. P T. 2013;38(6):336–55.

[2] Witchel SF, Oberfield SE, Peña AS. Polycystic Ovary Syndrome: Pathophysiology, Presentation, and Treatment with Emphasis on Adolescent Girls. J Endocr Soc. 2019;3(8):1545–73. doi:10.1210/js.2019-00078.

[3] Aly YE, Abdou AS, Rashad MM, Nassef MM. Effect of exercise on serum vitamin D and tissue vitamin D receptors in experimentally induced type 2 Diabetes Mellitus. J Adv Res. 2016;7(5):671–9. doi:10.1016/j.jare.2016.07.001.

[4] Grundmann M, von Versen-Höynck F. Vitamin D - roles in women's reproductive health? Reprod Biol Endocrinol. 2011;9(1):146. doi:10.1186/1477-7827-9-146.

[5] Pal L. Polycystic ovarian syndrome. New York: Springer; 2013. doi:10.1007/978-1-4614-8394-6_19.

[6] Ibrahim ZM, Ahmed Kishk E, Mohamed Fahmy S, Tharwat Taha O. Serum 1,25-dihydroxy vitamin D- vitamin D3- among obese Egyptian women with polycystic ovary syndrome. Obstet Gynecol Reports. 2020;4(1):1–3. doi:10.15761/ogr.1000141.

[7] Sidabutar E, Halim B, Siregar MFG, Lutan D, Adenin I, Kaban Y. Vitamin D Levels in Women with Polycystic Ovary Syndrome. ASPIRE Conf Proc. 2016. doi:10.18502/kme.v1i1.547

[8] Lakshman L, Pillai B, Lakshman R, Kumar H, Sudha S, Jayakumar R. Comparison of vitamin D levels in obese and non-obese patients with polycystic ovarian syndrome in a South Indian population. Int J Reprod Contraception, Obstet Gynecol. 2013;2(3):336–43. doi:10.5455/2320-1770.ijrcog20130915.

[9] Ni Z, Mei S, You S, Lin Y, Cheng W, Zhou L, et al. Adverse Effects of Polycystic Ovarian Syndrome on Pregnancy Outcomes in Women With Frozen-Thawed Embryo Transfer: Propensity Score-Matched Study. Front Endocrinol (Lausanne). 2022;13:878853. doi:10.3389/fendo.2022.878853.

[10] Talmo MSA, Fløysand IS, Nilsen GØ, Løvvik TS, Ødegård R, Juliusson PB, et al. Growth Restriction in the Offspring of Mothers With Polycystic Ovary Syndrome. JAMA Netw Open. 2024;7(8):e2430543.

doi:10.1001/jamanetworkopen.2024.30543.

[11] Collée J, Mawet M, Tebache L, Nisolle M, Brichant G. Polycystic ovarian syndrome and infertility: overview and insights of the putative treatments. Gynecol Endocrinol. 2021;37(10):869–74. doi:10.1080/09513590.2021.1958310.

[12] Cunha A, Póvoa AM. Infertility management in women with polycystic ovary syndrome: a review. Porto Biomed J. 2021;6(1):e116. doi:10.1097/j.pbj.0000000000000116.

[13] Kicińska AM, Maksym RB, Zabielska-Kaczorowska MA, Stachowska A, Babińska A. Immunological and Metabolic Causes of Infertility in Polycystic Ovary Syndrome. Biomedicines. 2023;11(6):1567. doi:10.3390/biomedicines11061567.

[14] Fang Y, Liu L, Yang Y, Zhang B, Xie S. Causal Association Between BMI and Polycystic Ovarian Syndrome: Bidirectional 2-Sample Mendelian Randomization Study. J Clin Endocrinol Metab. 2024. doi:10.1210/clinem/dgae446

[15] Jalilian N, Haghnazari L, Rasolinia S. Leptin and body mass index in polycystic ovary syndrome. Indian J Endocrinol Metab. 2016;20(3):324–8. doi:10.4103/2230-8210.180005.

[16] Sadeghi HM, Adeli I, Calina D, Docea AO, Mousavi T, Daniali M, et al. Polycystic Ovary Syndrome: A Comprehensive Review of Pathogenesis, Management, and Drug Repurposing. Int J Mol Sci. 2022;23(2):583. doi:10.3390/ijms23020583.

[17] Pateguana NB, Janes A. The contribution of hyperinsulinemia to the hyperandrogenism of polycystic ovary syndrome. J Metab Health. 2019;4(1):50. doi:10.4102/jir.v4i1.50.

[18] Mogili KD, Karuppusami R, Thomas S, Chandy A, Kamath MS, TK A. Prevalence of vitamin D deficiency in infertile women with polycystic ovarian syndrome and its association with metabolic syndrome – A prospective observational study. Eur J Obstet Gynecol Reprod Biol. 2018;229:15–9. doi:10.1016/j.ejogrb.2018.08.001

[19] Cahyo H, Yuad H, Rahmah BI. The relationship of vitamin D to polycystic ovary syndrome (PCOS) teenagers at Koto Tangah Padang District High School in 2022. Andalas Obstet Gynecol J. 2018;4(4):527–34. doi:10.25077/aoj.8.1.615-631.2024

[20] Thomson RL, Spedding S, Buckley JD. Vitamin D in the aetiology and management of polycystic ovary syndrome. Clin Endocrinol (Oxf). 2012;77(3):343–50. doi:10.1111/j.1365-2265.2012.04434.x.

[21] Suhartono J, Astiarani Y, Regina. Association between body mass index and vitamin D serum in Asian population: a systematic review. Public Health Prev Med Arch. 2022;10(1):69–79. doi:10.53638/phpma.2022.v10.i1.p08.

[22] Gokosmanoglu F, Onmez A, Ergenç H. The relationship between vitamin D deficiency and polycystic ovary syndrome. Afr Health Sci. 2020;20(4):1880–6. doi:10.4314/ahs.v20i4.45.

[23] Anjum S, Saleem A, Noor S, Mahmood A, Khalid S, Sarwa A. Relationship of vitamin D deficiency with obesity, metabolic abnormalities and insulin resistance in patients with PCOS. Pak J Med Health Sci. 2022;16(7):115–7. doi:10.53350/pjmhs22167115.

[24] Fattah A, Al-Kader DA, Jones Amaowei EE, Amini H, Hewadmal H, Rasuli SF, et al. A comparative study of luteinizing hormone levels in polycystic ovarian syndrome with hyperandrogenism: Metformin versus oral contraceptive pills. Cureus. 2022;14(9):e29487. doi:10.7759/cureus.29487.

[25] Cui A, Zhang T, Xiao P, Fan Z, Wang H, Zhuang Y. Global and regional prevalence of vitamin D deficiency in population-based studies from 2000 to 2022: A pooled analysis of 7.9 million participants. Front Nutr. 2023; doi:10.3389/fnut.2023.1070808.

[26] Octavius GS, Daleni VA, Angeline G, Virliani C. A systematic review and meta-analysis of prevalence of vitamin D deficiency among Indonesian pregnant women: A public health emergency. AJOG Glob Rep. 2023;3(2):100189. doi:10.1016/j.xagr.2023.100189.

[27] Sukarsa RA, Budi RS, Purwara BH, Syam HH, Sadikin H. Differences of vitamin D level in non-pregnant reproductive age women and first trimester pregnant women. Indones J Obstet Gynecol. 2019;7(4):2–7. doi:10.32771/inajog.v7i4.998

[28] Herlambang, Safira NNA, Fitri AD, Puspasari A. Gambaran kadar vitamin D pada wanita usia subur: model kebijakan kesehatan reproduksi. Medic. 2021;4(1):269–73. doi:10.22437/medicaldedication.v4i2.15597

[29] Kementerian Kesehatan Republik Indonesia. Peraturan Menteri Kesehatan Republik Indonesia Nomor 28 Tahun 2019 tentang Angka Kecukupan Gizi yang Dianjurkan untuk Masyarakat Indonesia. Sustain. 2019;11(1):1–14. https://peraturan.bpk.go.id/Details/138621/permenkes-no-28-tahun-2019

[30] Mohan A, Haider R, Fakhor H, Hina F, Kumar V, Jawed A, et al. Vitamin D and polycystic ovary syndrome

(PCOS): a review. Ann Med Surg. 2023;85:3506–11. doi:10.1097/ms9.0000000000000879

[31] Alloubani A, Akhu-Zaheya L, Samara R, Abdulhafiz I, Saleh A, Altowijri A. Relationship between vitamin D deficiency, diabetes, and obesity. Diabetes Metab Syndr Clin Res Rev. 2019;13(2):1457–61. doi:10.1016/j.dsx.2019.02.021.