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EDITORIAL

Polycystic Ovarian Syndrome (PCOS) in Adolescent Girls in Indonesia: A New Burden in Women's Reproductive Health

Sindroma Ovarium Polikistik (PCOS) Pada Remaja Wanita di Indonesia: Tantangan Baru dalam Kesehatan Reproduksi Wanita

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Background

Adolescence plays a crucial role in an individual's growth and development. During this period, young women experience significant physical and hormonal changes.[1,2] Adolescent health is crucial because it forms the foundation of health throughout adulthood and into old age.[3,4] In young adulthood, young women reach their peak reproductive potential, and maintaining general and reproductive health is crucial during this phase. 5 A healthy lifestyle during adolescence encompasses all aspects, including nutritional intake, physical activity, and stress management.[3–5] Within a few years of menarche, young women generally experience irregular menstrual cycles.[6] This is a physiological process, but it is important to note that it can be associated with an increased risk of polycystic ovary syndrome and other ovarian dysfunctions. A normal menstrual cycle lasts between 21 and 35 days and can vary from person to person.[1,6] Various factors can impact the menstrual cycle, including nutrition, ethnicity, age at menarche, physical activity, body mass index, and hormonal status. Menstrual cycle irregularities can include oligomenorrhea, polymenorrhea, or amenorrhea. These menstrual irregularities can increase with pregnancy, infection, malignancy, trauma, hormonal disorders, emotional stress, excessive physical activity or exercise, and poor dietary intake.[6]

The manifestation of menstrual cycle disorders in adolescents is most often caused by polycystic ovary syndrome, or better known as PCOS (Polycystic Ovarian Syndrome).[7,8] In 2021, the prevalence of PCOS cases was around 10,490,358.0 (95% UI: 7,423,407.5 – 14,808,757.1) in East Asia and 10,520,027.7 (95% UI: 7,378,813.9 – 14,809,823.5) in Southeast Asia. The prevalence rate of PCOS, based on age-standardized prevalence rates (ASPR), in East Asia and Southeast Asia is 1548.4 per 100,000 (95% UI: 1085.5 – 2170.7) and 28242.7 per 100,000 (95% UI: 1993.2 – 3997.5), respectively.[9] Based on the ASPR picture, it shows that the PCOS trend tends to increase in the period 1990 to 2021. The burden of PCOS varies across countries, but the trend will continue to increase in 2031.[9] The prevalence of PCOS in adolescents is estimated at around 6-10%, but this figure continues to increase along with changes in lifestyle, especially sedentary lifestyles and the

increasing prevalence of obesity in adolescents.[10,11] High-calorie diets, excessive intake of simple sugars, lack of physical activity, and psychological stress are factors that can contribute to the emergence and development of PCOS in adolescents.[10–12]

In recent years, various hypotheses have been proposed regarding the pathophysiology of PCOS.[13,14] This syndrome reflects the interaction of various proteins and genes influenced by epigenetic and environmental factors, which develops in early puberty and is characterized by excessive secretion of ovarian and/or adrenal androgens.[13] Intrinsic ovarian factors, such as impaired steroidogenesis, and external ovarian factors, such as hyperinsulinemia, contribute to the excessive production of ovarian androgens.[13] Distorted interactions among endocrine, paracrine, and autocrine factors responsible for ovarian follicle maturation also contribute to the ovarian dysregulation that causes PCOS.[13,14]

Under normal conditions, only one follicle will mature and be ovulated during the interaction process of follicle growth.[15] At birth, there are approximately 2-3 million ovarian primordial follicles, lower than the approximately 6-7 million follicles in the mid-gestation period.[13] The regulation of the rate and period at which new primordial follicles are added to the reserve pool is crucial for maintaining fertility and the ovarian reserve since follicles are constantly withdrawn from this reserve pool.[13] The status of active and dormant follicles is in a dynamic equilibrium. Anti-Mullerian Hormone (AMH), Follicle-Stimulating Hormone (FSH), and androgens are in an imbalance in PCOS, causing follicular arrest.[13–16] Theca cells produce androgens when luteinizing hormone (LH) levels are high, but when FSH levels are low and androgens cannot be converted to estradiol, no dominant follicle is selected, resulting in a long period of anovulation.[13,16] This equilibrium is tightly regulated by the hormone AMH, produced by granulosa cells, which prevents primordial follicles from maturing into primary follicles.[13,15] From this perspective, PCOS is characterized by an increase in the size of small follicles, followed by growth inhibition, resulting in a unique polycystic appearance.[13,15,16] AMH is essentially a transforming growth factor type Beta (TGF-β).[16–18] Because AMH regulates follicle growth, it is considered a marker of ovarian reserve. [17] The higher the plasma concentration of AMH, the more likely it is to induce ovarian reserve. indicates the severity of PCOS.[17,18]

Hyperandrogenism is a multifactorial pathology in PCOS influenced by a combination of environmental and hereditary elements.[18] This condition results from an imbalance in the Hypothalamic-Pituitary-Ovarian axis signaling process that leads to excessive secretion of luteinizing hormone and insulin. When clinical signs of hirsutism are unclear or absent, biochemical assessment of hyperandrogenism is essential for diagnosing PCOS.[18,19] Insulin plays a major role in glucose homeostasis and lipogenesis.[18] Insulin resistance plays a significant role in the development and progression of PCOS. Insulin resistance is caused by defects in the insulin receptor, resulting from excessive serine phosphorylation and decreased tyrosine phosphorylation, leading to decreased insulin activation of the phosphatidylinositol-3-kinase signaling pathway that regulates glucose transport and, consequently, increases glucose levels.[18,20,21] Increased insulin secretion directly triggers the pituitary gland to release luteinizing hormone, which in turn triggers androgen secretion and influences the growth and development of ovarian follicles. Both increased insulin and androgen levels will inhibit the secretion of sex hormone-binding globulin (SHBG), which leads to an increase in free bioactive androgens.[18,21]

Establishing a diagnosis of PCOS is not an easy task for clinicians. The consensus that is still the reference for establishing a diagnosis of PCOS is the Rotterdam Consensus of 2003.[14–17] The diagnosis of PCOS is established if 2 of the following 3 criteria are found: (1) oligomenorrhea and/or anovulation (OA); (2) hyperandrogenism (HA), which is defined as hirsutism (Ferriman-Gallwey/FG Index score >5); (3) identification of polycystic ovary (PCOM) morphology using ultrasonography where there are at least 12 follicles with a diameter of 2-9mm per ovary and/or increased ovarian volume (minimum 10mm3).[14,16–18] Based on these criteria, patients with PCOS can be classified into four phenotypic groups: phenotype-1 (OA+HA+PCOM), phenotype-2 (OA+HA), phenotype-3 (PCOM+HA), and phenotype-4 (OA+PCOM).[14,16,17,19] Based on the International Evidence-Based Guideline for the Assessment and Management of PCOS, the diagnosis of PCOS in adolescents must be done carefully because some physiological characteristics of puberty resemble PCOS. There are 2 criteria that can be used, the first is clinical or biochemical hyperandrogenism (eg, acne, hirsutism, or increased testosterone levels). The second criterion is persistent ovulatory disorders (cycles >90 days in the first year after menarche or <8 cycles per year in the three years after menarche). Polycystic ovarian morphology on ultrasonography is not recommended as a diagnostic criterion, although it can be found in adolescents with PCOS.[22–24]

Approximately 23% of women with PCOS have metabolic syndrome, primarily phenotype 1, followed by phenotypes 4, 2, and 3. The highest AMH levels were found in women with PCOS phenotype 1 (13.92 ng/ml), and this level is the highest compared to other phenotypes. Women with PCOS are associated with dyslipidemia, as high androgen levels increase the risk of atherosclerosis. AMH levels also correlate with metabolic syndrome markers such as HDL and triglyceride levels.[17]

There is a correlation between the incidence of PCOS in adolescents and a family history of PCOS. The pathophysiology of PCOS, which is related to its genetic component, accounts for approximately 10% of all cases. A family history of obesity is also correlated with the incidence of PCOS.25 Obesity carries a risk of irregular menstrual cycles and inhibited ovarian follicle maturation. Obesity also triggers insulin resistance, which often leads to PCOS. Approximately 50-70% of women with PCOS and 95% of obese women with PCOS have insulin resistance. Obesity and insulin resistance increase the release of pro-inflammatory cytokines, including high-sensitivity CRP (hs-CRP), IL-6, IL-18, and tumor necrosis factor alpha (TNF-α), in women with PCOS. Increased pro-inflammatory cytokines also reduce the sensitivity of pancreatic beta cells to elevated blood glucose levels.[25,26]

The pathophysiology of PCOS in adolescent girls needs to be addressed not only by clinical colleagues but also by adolescents and their parents to raise awareness. It is hoped that by creating awareness and understanding, preventive efforts for PCOS among adolescent girls can be more optimal.

PCOS in adolescents in Indonesia is a reproductive health challenge that requires a holistic, lifestyle-based approach. Education on nutrition, physical activity, and mental health should be part of the national adolescent health strategy. Early lifestyle-based interventions not only improve PCOS symptoms but also prevent long-term complications such as infertility and metabolic syndrome.

REFERENCES

1. Breehl L, Caban OC. Physiology,Puberty. Statpearls [Internet]. 2025;1–9. Available from: https://www.ncbi.nlm.nih.gov/books/NBK534827/
2. A life-course approach to women's health. Nat Med. 2024;30(1):1. Available from: https://www.nature.com/articles/s41591-023-02777-8
3. Feng C, Lai Y, Li R, et al. Reproductive health in Southeast Asian women: current situation and the influence factors. Glob Heal J. 2018;2(1):32–41. DOI: https://doi.org/10.1016/S2414-6447(19)30116-2
4. Paladine HL, Ekanadham H, Diaz DC. Health maintenance for women of reproductive age. Am Fam Physician. 2021;103(4):209–17.
5. Hanawi A, Saat M, Zulkafly M, et al. Impact of a Healthy Lifestyle on the Psychological Well-being of University Students. Int J Pharm Res Allied Sci. 2020;9(2–2020):1–7.
6. Aryani I. Menstrual cycle patterns of Indonesian adolescents. Paediatr Indones. 2018;58(3):101–5. DOI: http://dx.doi.org/10.14238/pi58.3.2018.101-5
7. Singh A, Vijaya K, Sai Laxmi K. Prevalence of polycystic ovarian syndrome among adolescent girls: a prospective study. Int J Reprod Contraception, Obstet Gynecol. 2018;7(11):4375. DOI: http://dx.doi.org/10.18203/2320-1770.ijrcog20184230
8. Ibáñez L, de Zegher F. Polycystic ovary syndrome in adolescent girls. Pediatr Obes. 2020;15(2):1–4. DOI: https://doi.org/10.1111/ijpo.12586
9. Chen Q, Zhao J, Wu X, et al. Temporal Trends of Polycystic Ovary Syndrome Burden in the East and Southeast Asia from 1990 to 2021: An Analysis from the Global Burden of Disease Study 2021. Int J Womens Health. 2025;17:2097–108. DOI: https://doi.org/10.2147/IJWH.S523350
10. Mizgier M, Jarzabek-Bielecka G, Formanowicz D, et al. Dietary and physical activity habits in adolescent girls with polycystic ovary syndrome (Pcos)-hastudy. J Clin Med. 2021;10(16):1–17. DOI: https://doi.org/10.3390/jcm10163469
11. Calcaterra V, Magenes VC, Massini G, et al. High Fat Diet and Polycystic Ovary Syndrome (PCOS) in Adolescence: An Overview of Nutritional Strategies. Nutrients. 2024;16(7):1–21. DOI: https://doi.org/10.3390/nu16070938
12. Said H, Fedre RW, Hernandez S, et al. The Prevalence and Risk Factors for Polycystic Ovary Syndrome (PCOS) among Adolescents in Indonesia: Implications for Early Intervention. Sriwij J Obstet Gynecol. 2023;1(2):84–97. DOI: https://doi.org/10.59345/sjog.v1i2.83
13. 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: https://doi.org/10.1210/js.2019-00078
14. Di Lorenzo M, Cacciapuoti N, Lonardo MS, et al. Pathophysiology and Nutritional Approaches in Polycystic Ovary Syndrome (PCOS): A Comprehensive Review. Curr Nutr Rep [Internet]. 2023;12(3):527–44. DOI: https://doi.org/10.1007/s13668-023-00479-8
15. Waghmare SV, Shanoo A. Polycystic Ovary Syndrome: A Literature Review With a Focus on Diagnosis, Pathophysiology, and Management. Cureus. 2023;15(10):1–9. DOI: 10.7759/cureus.47408
16. Liao B, Qiao J, Pang Y. Central Regulation of PCOS: Abnormal Neuronal-Reproductive-Metabolic Circuits in PCOS Pathophysiology. Front Endocrinol (Lausanne). 2021;12(May):1–11. DOI: https://doi.org/10.3389/fendo.2021.667422
17. Wiweko B, Handayani LK, Harzif AK, et al. Correlation of anti-müllerian hormone levels with metabolic syndrome events in polycystic ovary syndrome: A cross-sectional study. Int J Reprod Biomed. 2020;18(3):187–92. DOI: https://doi.org/10.18502/ijrm.v18i3.6716
18. Fahs D, Salloum D, Nasrallah M, et al. Polycystic Ovary Syndrome: Pathophysiology and Controversies in Diagnosis. Diagnostics. 2023;13(9):1–13. DOI: https://doi.org/10.3390/diagnostics13091559

19. Harada M. Pathophysiology of polycystic ovary syndrome revisited: Current understanding and perspectives regarding future research. Reprod Med Biol. 2022;21(1):1–13. DOI: https://doi.org/10.1002/rmb2.12487
20. Xu Y, Qiao J. Association of Insulin Resistance and Elevated Androgen Levels with Polycystic Ovarian Syndrome (PCOS): A Review of Literature. J Healthc Eng. 2022;2022. DOI: https://doi.org/10.1155/2022/9240569
21. Armanini D, Boscaro M, Bordin L, et al. Controversies in the Pathogenesis, Diagnosis and Treatment of PCOS: Focus on Insulin Resistance, Inflammation, and Hyperandrogenism. Int J Mol Sci. 2022;23(8). DOI: https://doi.org/10.3390/ijms23084110
22. Peña AS, Witchel SF, Hoeger KM, et al. 2- Adolescent polycystic ovary syndrome according to the international evidence-based guideline. BMC Medicine.2020;18(1):72. DOI: doi: 10.1186/s12916-020-01516-x
23. Witchel SF, Burghard AC, Tao RH, et al. The diagnosis and treatment of PCOS in adolescents: An update. Curr Opin Pediatr. 2019;31(4):562–9. DOI: doi: 10.1097/MOP.0000000000000778
24. Peña AS, Codner E, Witchel S. Criteria for Diagnosis of Polycystic Ovary Syndrome during Adolescence: Literature Review. Diagnostics. 2022;12(8). DOI: https://doi.org/10.3390/diagnostics12081931
25. Wahyuni A, Supriyatiningsih, Kusumawati W, et al. Family history of PCOS, obesity, low fiber diet, and low physical activity increase the risk of PCOS. J Kedokt dan Kesehat Indones. 2022;13(1):50-6. DOI: https://doi.org/10.20885/JKKI.Vol13.Iss1.art8
26. Calcaterra V, Verduci E, Cena H, et al. Polycystic ovary syndrome in insulin-resistant adolescents with obesity: The role of nutrition therapy and food supplements as a strategy to protect fertility. Nutrients. 2021;13(6):1–32. DOI: https://doi.org/10.3390/nu13061848.

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By Raditya Wratsangka

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There is a correlation between the incidence of PCOS in adolescents and a family history of PCOS. The pathophysiology of PCOS, which is related to its genetic component, accounts for approximately 10% of all cases. A family history of obesity is also correlated with the incidence of PCOS.25 Obesity carries a risk of irregular menstrual cycles and inhibited ovarian follicle maturation. Obesity also triggers insulin resistance, which often leads to PCOS. Approximately 50-70% of women with PCOS and 95% of obese women with PCOS have insulin resistance. Obesity and insulin resistance increase the release of pro-inflammatory cytokines, including high-sensitivity CRP (hs-CRP), IL-6, IL-18, and tumor necrosis factor alpha (TNF-α), in women with PCOS. Increased pro-inflammatory cytokines also reduce the sensitivity of pancreatic beta cells to elevated blood glucose levels.[25,26]

The pathophysiology of PCOS in adolescent girls needs to be addressed not only by clinical colleagues but also by adolescents and their parents to raise awareness. It is hoped that by creating awareness and understanding, preventive efforts for PCOS among adolescent girls can be more optimal.

PCOS in adolescents in Indonesia is a reproductive health challenge that requires a holistic, lifestyle-based approach. Education on nutrition, physical activity, and mental health should be part of the national adolescent health strategy. Early lifestyle-based interventions not only improve PCOS symptoms but also prevent long-term complications such as infertility and metabolic syndrome.

REFERENCES

1. Breehl L, Caban OC. Physiology,Puberty. Statpearls [Internet]. 2025;1–9. Available from: https://www.ncbi.nlm.nih.gov/books/NBK534827/
2. A life-course approach to women's health. Nat Med. 2024;30(1):1. Available from: https://www.nature.com/articles/s41591-023-02777-8
3. Feng C, Lai Y, Li R, et al. Reproductive health in Southeast Asian women: current situation and the influence factors. Glob Heal J. 2018;2(1):32–41. DOI: https://doi.org/10.1016/S2414-6447(19)30116-2
4. Paladine HL, Ekanadham H, Diaz DC. Health maintenance for women of reproductive age. Am Fam Physician. 2021;103(4):209–17.
5. Hanawi A, Saat M, Zulkafly M, et al. Impact of a Healthy Lifestyle on the Psychological Well-being of University Students. Int J Pharm Res Allied Sci. 2020;9(2–2020):1–7.
6. Aryani I. Menstrual cycle patterns of Indonesian adolescents. Paediatr Indones. 2018;58(3):101–5. DOI: http://dx.doi.org/10.14238/pi58.3.2018.101-5
7. Singh A, Vijaya K, Sai Laxmi K. Prevalence of polycystic ovarian syndrome among adolescent girls: a prospective study. Int J Reprod Contraception, Obstet Gynecol. 2018;7(11):4375. DOI: http://dx.doi.org/10.18203/2320-1770.ijrcog20184230
8. Ibáñez L, de Zegher F. Polycystic ovary syndrome in adolescent girls. Pediatr Obes. 2020;15(2):1–4. DOI: https://doi.org/10.1111/ijpo.12586
9. Chen Q, Zhao J, Wu X, et al. Temporal Trends of Polycystic Ovary Syndrome Burden in the East and Southeast Asia from 1990 to 2021: An Analysis from the Global Burden of Disease Study 2021. Int J Womens Health. 2025;17:2097–108. DOI: https://doi.org/10.2147/IJWH.S523350
10. Mizgier M, Jarzabek-Bielecka G, Formanowicz D, et al. Dietary and physical activity habits in adolescent girls with polycystic ovary syndrome (Pcos)-hastudy. J Clin Med. 2021;10(16):1–17. DOI: https://doi.org/10.3390/jcm10163469
11. Calcaterra V, Magenes VC, Massini G, et al. High Fat Diet and Polycystic Ovary Syndrome (PCOS) in Adolescence: An Overview of Nutritional Strategies. Nutrients. 2024;16(7):1–21. DOI: https://doi.org/10.3390/nu16070938
12. Said H, Fedre RW, Hernandez S, et al. The Prevalence and Risk Factors for Polycystic Ovary Syndrome (PCOS) among Adolescents in Indonesia: Implications for Early Intervention. Sriwij J Obstet Gynecol. 2023;1(2):84–97. DOI: https://doi.org/10.59345/sjog.v1i2.83
13. 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: https://doi.org/10.1210/js.2019-00078
14. Di Lorenzo M, Cacciapuoti N, Lonardo MS, et al. Pathophysiology and Nutritional Approaches in Polycystic Ovary Syndrome (PCOS): A Comprehensive Review. Curr Nutr Rep [Internet]. 2023;12(3):527–44. DOI: https://doi.org/10.1007/s13668-023-00479-8
15. Waghmare SV, Shanoo A. Polycystic Ovary Syndrome: A Literature Review With a Focus on Diagnosis, Pathophysiology, and Management. Cureus. 2023;15(10):1–9. DOI: 10.7759/cureus.47408
16. Liao B, Qiao J, Pang Y. Central Regulation of PCOS: Abnormal Neuronal-Reproductive-Metabolic Circuits in PCOS Pathophysiology. Front Endocrinol (Lausanne). 2021;12(May):1–11. DOI: https://doi.org/10.3389/fendo.2021.667422
17. Wiweko B, Handayani LK, Harzif AK, et al. Correlation of anti-müllerian hormone levels with metabolic syndrome events in polycystic ovary syndrome: A cross-sectional study. Int J Reprod Biomed. 2020;18(3):187–92. DOI: https://doi.org/10.18502/ijrm.v18i3.6716
18. Fahs D, Salloum D, Nasrallah M, et al. Polycystic Ovary Syndrome: Pathophysiology and Controversies in Diagnosis. Diagnostics. 2023;13(9):1–13. DOI: https://doi.org/10.3390/diagnostics13091559

19. Harada M. Pathophysiology of polycystic ovary syndrome revisited: Current understanding and perspectives regarding future research. Reprod Med Biol. 2022;21(1):1–13. DOI: https://doi.org/10.1002/rmb2.12487
20. Xu Y, Qiao J. Association of Insulin Resistance and Elevated Androgen Levels with Polycystic Ovarian Syndrome (PCOS): A Review of Literature. J Healthc Eng. 2022;2022. DOI: https://doi.org/10.1155/2022/9240569
21. Armanini D, Boscaro M, Bordin L, et al. Controversies in the Pathogenesis, Diagnosis and Treatment of PCOS: Focus on Insulin Resistance, Inflammation, and Hyperandrogenism. Int J Mol Sci. 2022;23(8). DOI: https://doi.org/10.3390/ijms23084110
22. Peña AS, Witchel SF, Hoeger KM, et al. 2- Adolescent polycystic ovary syndrome according to the international evidence-based guideline. BMC Medicine.2020;18(1):72. DOI: doi: 10.1186/s12916-020-01516-x
23. Witchel SF, Burghard AC, Tao RH, et al. The diagnosis and treatment of PCOS in adolescents: An update. Curr Opin Pediatr. 2019;31(4):562–9. DOI: doi: 10.1097/MOP.0000000000000778
24. Peña AS, Codner E, Witchel S. Criteria for Diagnosis of Polycystic Ovary Syndrome during Adolescence: Literature Review. Diagnostics. 2022;12(8). DOI: https://doi.org/10.3390/diagnostics12081931
25. Wahyuni A, Supriyatiningsih, Kusumawati W, et al. Family history of PCOS, obesity, low fiber diet, and low physical activity increase the risk of PCOS. J Kedokt dan Kesehat Indones. 2022;13(1):50-6. DOI: https://doi.org/10.20885/JKKI.Vol13.Iss1.art8
26. Calcaterra V, Verduci E, Cena H, et al. Polycystic ovary syndrome in insulin-resistant adolescents with obesity: The role of nutrition therapy and food supplements as a strategy to protect fertility. Nutrients. 2021;13(6):1–32. DOI: https://doi.org/10.3390/nu13061848.

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