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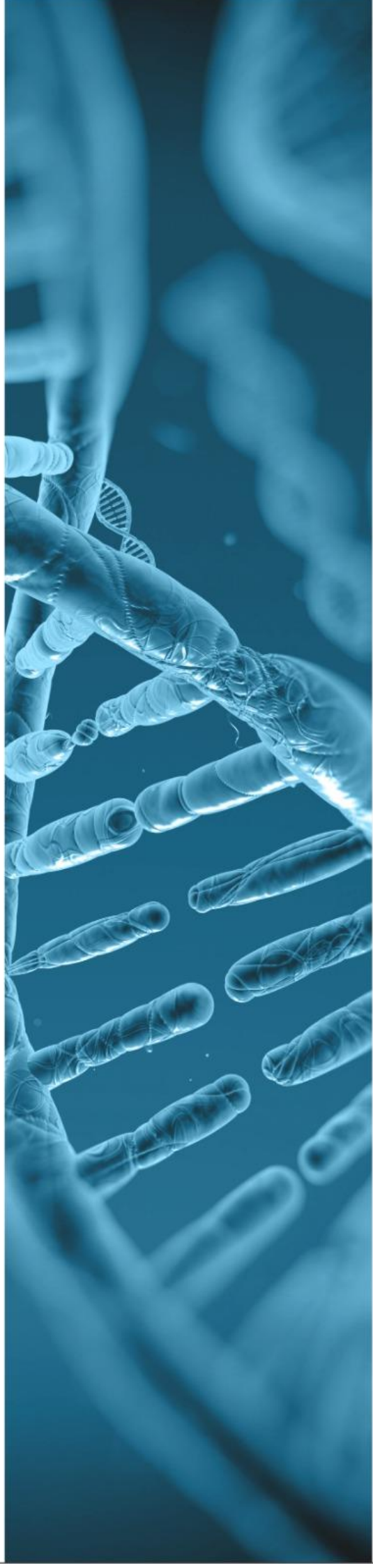

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Petunjuk Penulisan

Format penyusunan manuskrip
Manuskrip diketik pada kertas berukuran A4 (210 x 297 mm) dengan batas tepi 254 mm (*margin Normal)*, huruf diketik dengan tipe huruf (*font) Times New Roman*, besar huruf (*font size*) 12 point dengan menggunakan spasi rangkap 2 (*double space*). Setiap bagian dari manuskrip dimulai pada halaman baru dengan urutan sebagai berikut: halaman judul, abstrak dan kata kunci (*keywords*), teks keseluruhan, ucapan terima kasih, daftar pustaka, tabel dan gambar (setiap tabel dan gambar pada halaman terpisah). Nomor halaman dicantumkan secara berurutan dimulai dari halaman judul pada sudut sebelah kanan bawah. Manuskrip sebaiknya ditulis maksimal 16 halaman.

Halaman judul
Halaman judul mencakup: a) judul manuskrip yang dibuat sesingkat mungkin, spesifik informatif dan ringkasan judul tidak lebih dari 40 karakter (hitung huruf dan spasi) yang dicantumkan dibawah judul, b) nama penulis disusun berurutan dengan nama mahasiswa sebagai pengarang pertama, diikuti oleh Pembimbing sebagai pengarang kedua. Nama penulis ditulis lengkap tanpa gelar dan dicantumkan seperti aslinya, tidak dibalik seperti pada daftar pustaka dan sitasi, c) alamat setiap penulis, nama departemen dan lembaga afiliasi penulis, d) nama dan alamat penulis untuk korespondensi serta nomor telepon, nomor faksimili, alamat email. Judul penelitian dibuat jelas, singkat, spesifik, informatif, dan sesuai dengan topik manuskrip. Jumlah kata tidak lebih dari 12 kata agar mudah dan cepat dipahami pembaca.

Abstrak dan kata kunci
Abstrak berjumlah 200-250 kata ditulis dalam bahasa Indonesia dan Inggris. Abstrak berisikan latar belakang termasuk tujuan penelitian, metode, hasil, dan kesimpulan. Kata kunci dicantumkan di bawah abstrak pada halaman yang sama sebanyak 4-6 kata. Bagian abstrak merupakan ringkasan dari isi makalah yang dibuat secara singkat, informatif, dengan menekankan pada aspek baru dan penting dari penelitian.

Teks
Teks makalah manuskrip dibagi dalam beberapa bagian dengan judul sebagai berikut: ***Pendahuluan, Metode, Hasil, Pembahasan, Kesimpulan dan saran.***

Pendahuluan
a. Latar belakang merupakan bagian yang menjelaskan alasan mengapa masalah ini penting untuk diteliti. Bagian ini memuat penjelasan mengapa masalah itu dipandang menarik, penting, dan perlu diteliti untuk mencari pemecahannya. Penjelasan dapat diperoleh dari penelusuran pustaka yang berkaitan erat dengan masalah yang diteliti.
b. Keaslian penelitian dikemukakan dengan menunjukkan bahwa masalah yang dihadapi belum pernah dipecahkan oleh peneliti terdahulu atau dinyatakan dengan tegas perbedaan penelitian ini dengan penelitian terdahulu.
c. Tujuan penelitian yang menjelaskan hasil yang akan dicapai.

Metode
Metode penelitian berisi uraian terpadu dan sistematis mengenai bagaimana penelitian akan dilaksanakan. Metode terdiri dari :
a. Desain
b. Populasi / sampel (subjek) penelitian
Diuraikan kriteria inklusi dan eksklusi subjek penelitian, cara pemilihan sampel (subjek penelitian) secara random atau non-random, serta besar sampel yang akan di pilih. Teknik pemilihan sampel harus dijelaskan secara rinci. Bila perlu dibuat alur pemilihan sampel.
c. Bahan dan alat serta pengukuran
Bahan dan alat yang harus disajikan pada laporan terbatas pada bahan (materi) dan alat utama yang diperlukan untuk penelitian dan harus disebutkan spesifikasinya. Prosedur pengukuran perlu dijelaskan sesuai dengan tahapan yang dilakukan.
d. Alur kerja penelitian
Jalannya penelitian perlu dijelaskan mengenai jenis pendekatan yang dipakai untuk
mendapatkan data, melalui pendekatan laboratorium, klinik, komunitas, observasi, dll.
e. Analisis data
Perlu dijelaskan jenis teknik statistik yang digunakan untuk menjawab masalah dan mencapat tujuan penelitian. Data yang diperoleh dapat dianalisis menggunakan teknik statistik secara parametrik dan non-parametrik.

Hasil
Suatu hasil penelitian hendaknya disajikan dengan jelas, logis, runut, sehingga mudah untuk dimengerti. Hasil penelitian sebaiknya ditampilkan selain dalam bentuk narasi dapat pula berupa gambar, tabel, foto, dan grafik sehingga memudahkan untuk dipahami. Hasil dan interpretasi analisis statistik dituliskan secara jelas dalam uraian hasil penelitian.
Pada tahap awal disajikan distribusi karakteristik subjek penelitian, yang biasanya dibuat pada sebuah tabel. Kemudian disajikan temuan penting yang diperoleh, kalau cukup banyak sebaiknya pada sebuah tabel. Bila terbatas misalkan hanya satu atau dua temuan cukup dalam bentuk narasi/teks.
Tabel, bagan/gambar, grafik dibuat dengan jelas, diberi nomor urut serta keterangan yang jelas. Keterangan

tabel diletakan di atas tabel dan keterangan gambar diletakkan di bawah gambar. Maksimal tabel dan gambar 5. Semua tabel, grafik dan gambar diberi nomor dan keterangan yang jelas. Setiap tabel dianalisis dan diinterpretasi secara sistematik, dan hasilnya ditulis di bawah tabel tersebut. Perhitungan statistik detail tidak perlu ditulis dalam bagian hasil ini. Bila perhitungan statistik dianggap perlu ditulis, maka sebaiknya diletakkan dalam lampiran saja.

Pembahasan

Langkah awal harus diuraikan temuan penting yang diperoleh dari penelitian sesuai dengan tujuan penelitian. Kemudian bandingkan hasil penelitian yang diperoleh dengan hasil-hasil penelitian sebelumnya. Perlu dijelaskan kesesuaian dan ketidaksesuaian hasil penelitian yang didapat terhadap kerangka teori atau hasil penelitian lain yang telah dilakukan sebelumnya. Selanjutnya menggunakan teori-teori yang ada uraikan mekanisme terjadinya hasil penelitian tersebut. Bagian pembahasan juga menjelaskan mengenai kelemahan dan kelebihan penelitian yang telah dilakukan. Uraikan implikasi dari hasil penelitian yang diperoleh.

Kesimpulan

Kesimpulan hendaknya dibuat dalam bentuk narasi dan menguraikan secara singkat, jelas, padat menurut urutan yang sistematis. Bagian ini memuat tentang hasil penelitian yang telah diperoleh untuk menjawab tujuan penelitian. Saran menguraikan perlunya dilakukan penelitian lebih lanjut untuk memperbaiki kelemahan/ keterbatasan dari penelitian yang telah dilakukan.

Ucapan terima kasih

Ditujukan kepada pihak-pihak yang memberikan bantuan dana dan dukungan antara lain dukungan dari bagian dan lembaga, para professional yang memberikan kontribusi dalam penyusunan makalah, dan untuk penguji I maupun penguji II. Pembimbing tidak perlu dicantumkan pada Ucapan Terima Kasih karena sudah dicantumkan sebagai penulis.

Daftar Referensi

Daftar referensi/rujukan hanya mencatumkan rujukan yang telah digunakan dan ditulis menurut sistem Vancouver.

Online Submission

Penulis dapat mengirim naskah manuskrip melalui *online submission* di *website* Jurnal Biomedika dan Kesehatan.

Langkah online submission:

1. Pastikan naskah yang akan diunggah sudah mengikuti semua petunjuk penulisan
2. Lakukan pendaftaran author di : https://jbiomedkes.org/index.php/jbk/user/register
3. Setelah terdaftar silakan unggah naskah manuskrip dan isi form yang terdapat di dalam website, dan ikuti langkah selanjutnya.

Daftar Cek Pengiriman Naskah Manuskrip

□ Naskah manuskrip belum pernah dipublikasikan sebelumnya, juga tidak dalam pengajuan ke jurnal lain.
□ File manuskrip harus berformat OpenOffice, Ms. Word atau RTF dokumen, *font* 12, *Times New Roman, double spacing.*
□ Halaman judul harus memuat jelas judul, nama lengkap penulis tanpa gelar, departemen penulis, universitas, alamat lengkap, nomor telepon dan email.
□ Pelaporan data manuskrip dari penelitian yang melibatkan manusia dan hewan memerlukan persetujuan formal (kaji etik) oleh dewan peninjau atau komisi etik institusi yang bersangkutan.
□ Daftar rujukan memuat semua rujukan yang terdapat di dalam manuskrip dan ditulis sesuai urutan pengutipannya menggunakan sistem Vancouver.

Daftar Isi

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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.

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

by Aditya Khrisna Murthi

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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.

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