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Polycystic Ovary Syndrome (PCOS) as a Risk Factor for Preeclampsia and Cognitive Dysfunction

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ABSTRACT

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder in women of reproductive age. PCOS is associated with various metabolic disturbances, such as insulin resistance, obesity, dyslipidemia, and chronic inflammation, which may increase the risk of pregnancy complications, such as preeclampsia. In addition to increasing the risk of preeclampsia, PCOS has also been linked to a decline in cognitive function resulting from the effects of hyperandrogenism, insulin resistance, and systemic inflammation on neuronal function. Therefore, PCOS not only increases the risk of developing preeclampsia but may also play an indirect role in cognitive dysfunction. This narrative review discusses the role of PCOS as a risk factor for preeclampsia and cognitive impairment and the interconnected pathophysiological mechanisms linking these conditions.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is an endocrine disorder that frequently occurs in women of reproductive age. According to the World Health Organization (2024), PCOS affects 6–13% of reproductive-aged women globally, with approximately 70% of cases remaining undiagnosed. The prevalence of PCOS in Indonesia has been reported to reach 45.7%, with cases predominantly occurring in the 26–30-year age group. Based on data from Statistics Indonesia (Badan Pusat Statistik, BPS), 12% of couples of reproductive age experience infertility, which is partly attributable to PCOS. Data from the Basic Health Research (Riskesdas) survey also recorded that 16% of adolescent girls who are overweight or obese contribute to the risk of PCOS [1].

The diagnosis of PCOS is most commonly established using the Rotterdam criteria, which require the presence of two of the following: oligo- or anovulation, clinical and/or biochemical signs of hyperandrogenism, or polycystic ovaries, together with the exclusion of other etiologies, such as congenital adrenal hyperplasia, androgen-secreting tumors, growth hormone–secreting pituitary tumors, and Cushing syndrome. PCOS presents with a wide variety of clinical phenotypes that may differ across ethnic groups [2]. PCOS is a heterogeneous endocrine disorder with diverse clinical manifestations and metabolic risks. According to the phenotypic classification proposed by the National Institutes of Health (NIH) in 2012, based

on the presence of hyperandrogenism (HA), ovulatory dysfunction (OD), and polycystic ovarian morphology (PCOM), PCOS is divided into four phenotypes (A–D). Phenotypes A and B are both characterized by hyperandrogenism and chronic oligo-anovulation (OA). Phenotype A, often referred to as classic PCOS, is additionally accompanied by polycystic ovarian morphology (PCOM); it is the most common phenotype, accounting for 50% of cases. Phenotype B is also known as hyperandrogenic anovulation. Phenotype C (ovulatory PCOS), although ovulation occurs, demonstrates metabolic abnormalities due to androgen excess. In contrast, phenotype D, which lacks hyperandrogenism, generally exhibits the mildest hormonal and metabolic profile, consisting of ovulatory dysfunction and polycystic ovarian morphology (PCOM) (non-hyperandrogenic PCOS) [3].

Clinically, PCOS presents a broad spectrum of symptoms, including menstrual irregularity, anovulation, subfertility or infertility, hyperandrogenism, and metabolic disturbances, such as insulin resistance (IR), dysglycemia, obesity, and dyslipidemia. These metabolic derangements not only contribute to the direct reproductive and endocrine manifestations of the syndrome but also predispose affected individuals to long-term risks, such as type 2 diabetes, metabolic syndrome, and cardiovascular disease. Phenotypes characterized by hyperandrogenism and OD (particularly phenotype A) tend to display more severe metabolic abnormalities and cardiovascular risk profiles, whereas non-hyperandrogenic phenotypes (e.g., phenotype D) may have a milder metabolic presentation [3]. In the neuropsychiatric domain, PCOS also exerts a considerable impact, such as cognitive dysfunction, anxiety, and depression [4]. Pregnant women with PCOS have an increased risk of pregnancy-related disorders, such as gestational diabetes mellitus (GDM), gestational hypertension, and preeclampsia in the second and third trimesters. In addition, there is an increased risk of preterm birth and low birth weight infants [5]. PCOS also causes further ovarian dysfunction that may result in anovulation and infertility [6]. The impact of PCOS may lead to long-term health problems, affecting not only reproductive health but also overall well-being, which can result in a reduced quality of life [4].

METHOD

This narrative review was prepared through a literature search of scientific publications relevant to polycystic ovary syndrome (PCOS), preeclampsia, and cognitive function. Electronic databases, including PubMed, Scopus, ScienceDirect, and Google Scholar, were used for the literature searches. The keywords used, applied individually and in combination with the Boolean operators “AND” and “OR,” included “polycystic ovary syndrome,” “PCOS,” “preeclampsia,” “cognitive function,” “cognitive impairment,” “insulin resistance,” “hyperandrogenism,” and “pregnancy outcomes.” The literature reviewed comprised original research articles, review articles, and cohort studies published predominantly between 2015 and 2026 in English and Indonesian. Articles were selected based on their relevance to the topic, namely, the relationship between PCOS, the risk of preeclampsia, and cognitive function, with priority given to the most recent and pertinent publications. Information obtained from the selected literature was subsequently analyzed narratively and synthesized to describe the interconnected pathophysiological mechanisms linking PCOS, preeclampsia, and cognitive dysfunction.

PCOS as a Risk Factor for Preeclampsia

Preeclampsia (PE) is a multisystem hypertensive disorder of pregnancy characterized by new-onset hypertension (blood pressure ≥140/90 mmHg) after 20 weeks of gestation, accompanied by signs of maternal organ dysfunction (e.g., renal, hepatic, hematologic, or neurological involvement) and/or fetal growth restriction. Proteinuria (≥300 mg/24 hours) is a common but not essential criterion. PE affects approximately 2–8% of pregnancies worldwide, making it a leading cause of maternal and perinatal morbidity and mortality. The incidence of PE varies by region, with higher rates in low- and middle-income countries owing to limited access to prenatal care [7]. Currently, the most widely accepted theory regarding the development of PE is the two-stage theory. In this model, the first stage of the disease involves impaired placentation and reduced placental perfusion, whereas the second stage encompasses generalized maternal endothelial damage and dysfunction. This theory applies particularly to early-onset PE, which is characterized by placental

insufficiency and fetal growth restriction (FGR). However, late-onset PE, which is rarely associated with significant placental dysfunction, may have a somewhat different pathophysiological basis [8].

The second theory regarding the development of PE is cardiogenic theory. According to this model, the primary cause of the disease is preexisting cardiac dysfunction that is triggered or aggravated by pregnancy. This cardiac dysfunction subsequently leads to reduced placental perfusion and the characteristic clinical consequences observed in PE [8]. Many studies have shown that women with PCOS have an increased risk of developing preeclampsia. For example, in a prospective cohort study of patients with PCOS, a high body mass index (BMI) was found to be the strongest predictor of preeclampsia [9]. Furthermore, compared with pregnant women without PCOS, pregnant women with PCOS are more likely to be obese and nulliparous and to have received fertility treatment, all of which have been independently associated with the risk of preeclampsia [2]. Women with PCOS have an increased risk of pregnancy complications, including implantation failure, miscarriage, gestational diabetes, fetal growth restriction, preterm labor, and preeclampsia (PE). This may be attributable to specific vulnerabilities associated with PCOS before and during pregnancy, such as chronic systemic inflammation, insulin resistance, and hyperandrogenism, all of which have been linked to an increased risk of pregnancy complications. Giannakou et al. identified the strongest risk factors associated with PE, which include chronic kidney disease, PCOS, oocyte donation, obesity, and primiparity [10]. In addition to an increased risk of developing preeclampsia and various metabolic disorders, women with PCOS are also at risk of hypertension, leading to endothelial dysfunction that represents the early stage of the atherosclerotic process [7].

Normal placental development requires a complex network of bidirectional communication signals (cytokines, metabolites, hormones, and exosomes) between embryo-derived cells (trophoblasts and macrophages) and maternal-derived cells (endometrial glandular epithelium, stroma, macrophages, natural killer cells, dendritic cells, and T cells). Decades of epidemiological research have identified more than 70 maternal risk factors associated with the development of preeclampsia (PE). Pre-existing maternal pathophysiological factors play a role in the development of abnormal placentation and related complications, such as PE. Pre-existing maternal pathological features, such as chronic systemic inflammation, insulin resistance, and hyperandrogenemia, which occur in women with polycystic ovary syndrome (PCOS), can alter normal placental development, metabolism, and physiology at all stages of pregnancy [10]. Maternal angiogenic imbalance caused by placental antiangiogenic factors plays a central role in the systemic vascular dysfunction underlying PE. The severity of the maternal antiangiogenic state is closely correlated with maternal and perinatal outcomes. Assessment of angiogenic imbalance and several tests of vascular function have also emerged as means of detecting systemic vascular dysfunction during pregnancy [11]. Hyperandrogenism is associated with adverse pregnancy outcomes, such as PE, gestational diabetes, preterm birth, and small-for-gestational-age (SGA) infants. Women with PCOS who have high testosterone levels during pregnancy appear to be at a higher risk of PE. Early-onset preeclampsia and preeclampsia in women with small-for-gestational-age infants are considered subtypes of preeclampsia that have a more pronounced placental component than late-onset preeclampsia and preeclampsia in women without small-for-gestational-age infants. Placental studies have shown that the placentas of pregnant women with PCOS have altered histological structures, even in uncomplicated pregnancies. Placental lesions consist of microscopic changes in early trophoblast invasion and placentation [12].

PCOS and Cognitive Dysfunction

Cognitive function refers to a set of mental processes involved in learning, memory, attention, perception, language, intelligence, and reasoning. Cognitive function can be assessed in several ways. One way to assess cognitive function using a blood biomarker is through the measurement of brain-derived neurotrophic factor (BDNF) levels, an important protein that plays a significant role in enhancing the survival of synapses and neurons involved in memory and learning processes. BDNF is mainly expressed in the hippocampal and parahippocampal areas, which are crucial for cognitive tasks related to memory and learning [13,14]. In addition to BDNF levels, cognitive function can also be assessed using various questionnaire-based examinations. One frequently used questionnaire is the Montreal Cognitive Assessment (MoCA), which is generally used to screen for mild cognitive impairment (MCI). The domains assessed by the MoCA consist of attention and concentration, executive function, memory, language, visuoconstruction, conceptual thinking, calculation, and orientation skills [13]. Cognitive function may decline due to various independent predictor factors. One strategy to prevent cognitive impairment is to address modifiable risk factors. Factors that may drive a decline in cognitive function include brain injury, such as stroke, brain tumors, brain infections, and head injury. In addition to brain injury, aging, vascular disease (hypertension), metabolic disease (diabetes mellitus), and lifestyle factors such as physical inactivity, unhealthy diet, and smoking habits may also cause a decline in cognitive function [15]. One disease that carries cardiometabolic risk factors and also affects neuropsychiatric conditions is PCOS [16].

In PCOS, long-term complications may also occur in the form of metabolic disturbances and metabolic syndrome, which are commonly observed across various PCOS phenotypes. Metabolic syndrome may also affect cognitive performance. The role of insulin in the brains of healthy individuals includes central modulation of metabolism, enhancement or regulation of memory, and other cognitive and emotional functions. In PCOS, insulin resistance in the brain occurs, which can be defined as the failure of brain cells to respond to insulin, typically disrupting synaptic, metabolic, and immune responses. In addition to insulin resistance, a decline in cognitive function may also be caused by obesity, dyslipidemia, and disturbances in homocysteine metabolism, which may be the main factors in the development of cognitive impairment in women with PCOS, rather than androgens [4]. Obesity and being overweight are common problems among women with PCOS. Research has shown that obese women have substantially lower cognitive function and attention, indicating early impairment of cognitive function in obese women. The mechanism underlying the relationship between obesity and cognitive dysfunction remains unclear; however, it may be related to cerebral atrophy, cerebrovascular disturbances, and systemic and central inflammation. Some evidence supports the adverse impact of obesity on cognitive function in women; however, the longitudinal impact of obesity and weight gain in women with PCOS remains unclear [17].

A study conducted to investigate the effects of PCOS on brain activity and to explore the relationship between brain activity and sex hormone levels in women with PCOS showed that high luteinizing hormone (LH) levels may cause changes in the activity of brain regions responsible for visuospatial working memory, facial processing, and episodic memory [18]. In addition, higher free testosterone levels in women with PCOS have been associated with poorer cognitive function, particularly psychomotor speed and visuospatial learning [19]. Complications or diseases associated with metabolic syndrome, such as hypertension, may also be linked to cognitive impairment. High blood pressure is another complication of PCOS. Studies have shown that hypertension impairs the structure and function of cerebral blood vessels, leading to ischemic damage to the white matter involved in cognitive function, which may be related to Alzheimer pathology. There is strong evidence regarding the destructive effects of hypertension on cognitive function in middle-aged individuals [20,21]. The impact of PCOS on cognitive function has been examined by reviewing specific domains, finding evidence of mild but measurable deficits such as verbal fluency, working memory, visuospatial ability, and executive function compared with matched control groups. Several studies have reported reduced fluency associated with PCOS, whereas more studies have shown poorer memory findings in PCOS. Studies examining visuospatial cognition have shown mixed results, whereas executive function is impaired in PCOS. Some studies have specifically examined the impact of hyperandrogenism on cognition in PCOS and reported lower executive function associated with high androgen levels [16].

Existing research on cognition in women with PCOS has focused almost exclusively on women of reproductive age, typically ranging from late adolescence to the early 40s, and has not grouped or analyzed cognitive outcomes in perimenopausal subgroups. Estrogen is known to bind to neuroreceptors, with evidence of binding to extranuclear receptors within the hippocampus. In addition, estrogen signaling may have protective effects on central autonomic nuclei involved in cardiovascular regulation, an effect that is disrupted by the decline in estrogen during menopause. A recent study in women with PCOS, with a mean age of 54.7 years, showed lower performance in the cognitive domains of attention and cognitive control, verbal learning and memory, and category fluency compared to women without PCOS [16]. Women with PCOS exhibit distinct cognitive vulnerabilities, particularly in the executive, attention, language, and orientation domains. Cognitive dysfunction may be an underrecognized aspect of PCOS, warranting routine screening and future longitudinal studies. Recent attention has shifted toward the potential of PCOS to affect cognitive function. Emerging evidence suggests that women with PCOS may experience mild cognitive deficits across various domains, including memory, attention, executive function, and visuospatial skills. The etiology of these cognitive changes is not yet fully understood; however, hormonal, metabolic, and inflammatory factors are thought to play a role [22]. Another study showed that patients with PCOS had significantly poorer performance in the memory domain, as assessed by the Rey Auditory Verbal Learning Test (RAVLT), compared to the control group. According to the results of this test, immediate verbal memory was impaired in patients with PCOS. It has also been observed that recognition in the long-term delayed recall task was impaired in patients with PCOS [23].

Mechanism Linking PCOS – Preeclampsia – Cognitive Function

The relationship between PCOS, preeclampsia, and cognitive function can be explained through several interconnected pathophysiological mechanisms. PCOS is associated with insulin resistance, hypertension, dyslipidemia, infertility, and adverse pregnancy outcomes. Women with PCOS have an increased risk of miscarriage, hypertensive disorders during pregnancy, gestational diabetes, and preterm birth, and these risks vary depending on the PCOS phenotype. During pregnancy, normal placental development requires a complex network of bidirectional communication signals between

embryo- and maternal-derived cells. Pregnant women with PCOS experience chronic systemic inflammation, insulin resistance, and hyperandrogenemia. This chronic inflammation can disrupt trophoblast invasion and spiral artery remodeling, both of which play a role in the development of preeclampsia. In PCOS, long-term complications, such as metabolic syndrome, may occur, which can also affect cognitive performance. In pregnant women with PCOS, maternal angiogenic imbalance caused by placental antiangiogenic factors plays a central role in the systemic vascular dysfunction underlying preeclampsia. The release of antiangiogenic factors from the placenta can cause endothelial damage and impaired organ perfusion, including that of the brain. Damage to cerebral blood vessels resulting from endothelial dysfunction and chronic inflammation can affect neuronal function and lead to a decline in cognitive function.

CONCLUSION

Polycystic ovary syndrome (PCOS) is an endocrine disorder in reproductive-age women linked to metabolic complications, including type 2 diabetes mellitus, insulin resistance, cardiovascular disease, hypertension, dyslipidemia, infertility, and adverse pregnancy outcomes. Women with PCOS have increased preeclampsia risk, which can impair brain perfusion, damage neurons, and indirectly contribute to cognitive dysfunction.

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I.F.Y. conceived the study, conducted a literature review, and drafted the manuscript. I.R. supervised the study and revised the manuscript critically. Y.I., T.K., D.P.N., and E.Y. contributed to the data interpretation, manuscript review, and intellectual content. All the authors have read and approved the final manuscript.

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