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Polycystic Ovary Syndrome and the Risk of Eclampsia and Maternal Cognitive Impairment: A Population-Based Cohort Study

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ABSTRACT

Introduction: Polycystic ovary syndrome (PCOS) is a common endocrine-metabolic disorder associated with insulin resistance, chronic inflammation, and endothelial dysfunction in women. These abnormalities may increase the risk of hypertensive disorders during pregnancy and subsequent neurological complications. This study evaluated the association between PCOS, eclampsia, and cognitive impairment in women.
Methods: We conducted a narrative review and integrative analysis of studies retrieved from PubMed, Scopus, and Web of Science. Observational studies, clinical investigations, and mechanistic research examining polycystic ovary syndrome, hypertensive disorders of pregnancy, neurological complications, and cognitive outcomes were qualitatively synthesized in this review.
Results: Women with PCOS exhibited a higher risk of gestational hypertension, preeclampsia, and eclampsia than those without PCOS. Insulin resistance, obesity, chronic inflammation, oxidative stress, and endothelial dysfunction are major contributors to vascular and placental abnormalities. Severe hypertensive disorders during pregnancy are also associated with impaired memory, executive dysfunction, reduced attention, and cerebrovascular injury. Shared inflammatory and vascular pathways may explain the relationship between PCOS, eclampsia, and cognitive impairments.
Conclusion: PCOS may be an important risk factor for eclampsia and subsequent cognitive impairment through interconnected metabolic, inflammatory, and endothelial pathways. Early identification and multidisciplinary management may reduce maternal complications and improve long-term neurological outcomes.

Keywords

Polycystic Ovary Syndrome, Eclampsia, Cognitive Impairment, Endothelial Dysfunction, Neuroinflammation

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is one of the most prevalent endocrine and metabolic disorders among women of reproductive age, affecting approximately 6–20% of women worldwide, depending on the diagnostic criteria used [1,2]. The syndrome is characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, and is frequently accompanied by insulin resistance, obesity, dyslipidemia, and chronic low-grade inflammation [3]. Although traditionally considered a reproductive disorder, PCOS is

now recognized as a complex multisystem condition associated with significant metabolic, cardiovascular, and vascular complications that may persist throughout life [4].

Growing evidence indicates that women with PCOS experience a higher incidence of adverse pregnancy outcomes compared to the general obstetric population [5]. Among these complications, hypertensive disorders of pregnancy, including gestational hypertension, preeclampsia, and eclampsia, represent major causes of maternal and perinatal morbidity and mortality worldwide [6]. Eclampsia, defined as the occurrence of new-onset generalized seizures in women with preeclampsia, remains a severe obstetric emergency associated with substantial maternal neurological injury and adverse fetal outcomes [7]. The pathogenesis of eclampsia involves abnormal placentation, endothelial dysfunction, oxidative stress, systemic inflammation, impaired cerebrovascular autoregulation, and disruption of the blood–brain barrier [8]. Notably, many of these pathological mechanisms overlap with the metabolic and vascular abnormalities observed in PCOS. Chronic inflammation, insulin resistance, endothelial injury, and altered vascular reactivity may predispose women with PCOS to an increased susceptibility to hypertensive complications during pregnancy and their neurological consequences [9]. Recent studies have further demonstrated that women who experience severe hypertensive disorders of pregnancy face an elevated risk of long-term cognitive decline, executive dysfunction, memory impairment, and cerebrovascular disease later in life [10].

Despite increasing recognition of these associations, the potential mechanistic link between PCOS, eclampsia, and subsequent cognitive impairment remains insufficiently explored. Understanding this relationship is essential for improving risk stratification, optimizing preventive strategies, and establishing long-term neurological surveillance in women with PCOS. Therefore, this review aimed to examine the biological and clinical evidence linking PCOS with eclampsia and cognitive impairment, with particular emphasis on the shared metabolic, inflammatory, vascular, and neuroendothelial pathways that may contribute to these interconnected conditions.

METHOD

This narrative review was conducted to evaluate the potential association between polycystic ovary syndrome (PCOS), eclampsia, and cognitive impairment, with particular emphasis on shared metabolic, inflammatory, vascular, and neurological mechanisms. A comprehensive literature search was performed using the PubMed, Scopus, and Web of Science databases to identify relevant studies published up to March 2023. The search strategy incorporated combinations of Medical Subject Headings (MeSH) terms and free-text keywords, including “polycystic ovary syndrome, PCOS, eclampsia, preeclampsia, hypertensive disorders of pregnancy, cognitive impairment, cognitive dysfunction, neuroinflammation, endothelial dysfunction, vascular injury, and cerebrovascular disease. Boolean operators (AND, OR) were applied to optimize search sensitivity and specificity.

Eligible publications included observational studies, cohort studies, case-control studies, clinical investigations, systematic reviews, meta-analyses, and experimental studies published in English. Studies were considered eligible if they examined the relationship between PCOS and hypertensive disorders of pregnancy, neurological outcomes associated with eclampsia or preeclampsia, or the biological mechanisms linking these conditions. Conference abstracts, editorials, commentaries, duplicate publications, and studies lacking sufficient methodological detail were excluded. Relevant articles were screened based on title, abstract, and full-text review. Data regarding study characteristics, clinical outcomes, pathophysiological mechanisms, and neurological findings were extracted and synthesized qualitatively. The evidence was subsequently organized into thematic domains, including metabolic dysregulation, inflammation, endothelial dysfunction, placental pathology, cerebrovascular injury, and cognitive impairment, to identify potential mechanistic links between PCOS, eclampsia, and long-term neurological outcomes.

RESULTS

The literature consistently demonstrates a significant association between polycystic ovary syndrome (PCOS), hypertensive disorders of pregnancy, and adverse neurological outcomes. Evidence from observational studies and clinical investigations indicates that women with PCOS have an increased risk of developing gestational hypertension, preeclampsia, and eclampsia compared to women without PCOS. This elevated risk appears to be mediated by insulin resistance, obesity, chronic low-grade inflammation, oxidative stress, and endothelial dysfunction, all of which contribute to impaired placental perfusion and vascular injury. Several studies have

identified overlapping pathophysiological mechanisms between PCOS and eclampsia. Hyperandrogenism and metabolic dysregulation are associated with increased production of pro-inflammatory cytokines and reduced endothelial nitric oxide bioavailability, promoting vascular dysfunction. These alterations may facilitate abnormal placentation and increase susceptibility to severe hypertensive complications during pregnancy.

In addition to obstetric complications, accumulating evidence suggests that severe hypertensive disorders of pregnancy are associated with long-term neurological sequelae. Women with a history of eclampsia have demonstrated a higher prevalence of cognitive impairment, including deficits in memory, executive function, attention, and processing speed. Cerebral endothelial injury, blood–brain barrier disruption, neuroinflammation, and cerebrovascular dysfunction have repeatedly been identified as potential contributors to these neurological outcomes. Collectively, the reviewed evidence supports a biological continuum linking polycystic ovary syndrome (PCOS), eclampsia, and cognitive impairment through interconnected metabolic, inflammatory, vascular, and neuroendothelial pathways. The principal findings derived from the literature are summarized in Table 1.

Table 1. Summary of evidence linking PCOS, eclampsia, and cognitive impairment

Domain	Findings	Potential Clinical Implications
PCOS and metabolic dysfunction	PCOS is associated with insulin resistance, obesity, hyperandrogenism, and chronic inflammation	Increased cardiovascular and obstetric risk
PCOS and vascular dysfunction	Endothelial dysfunction and oxidative stress are frequently observed in women with PCOS	Predisposition to hypertensive disorders during pregnancy
PCOS and eclampsia	Women with PCOS exhibit higher rates of gestational hypertension, preeclampsia, and eclampsia	Increased maternal morbidity and pregnancy complications
Eclampsia and neurological injury	Severe hypertensive disorders may cause cerebral endothelial injury and blood–brain barrier disruption	Acute and chronic neurological complications
Eclampsia and cognitive impairment	Cognitive deficits involving memory, attention, and executive function have been reported after eclampsia	Need for long-term neurological follow-up
Shared biological pathways	Inflammation, oxidative stress, endothelial dysfunction, and vascular injury are common to all three conditions	Potential targets for prevention and risk reduction

DISCUSSION

This review highlights the plausible biological and clinical associations between polycystic ovary syndrome (PCOS), eclampsia, and subsequent cognitive impairment. Although these conditions have traditionally been investigated separately, accumulating evidence suggests that they may represent interconnected manifestations of a common pathophysiological continuum characterized by metabolic dysregulation, chronic inflammation, endothelial dysfunction, oxidative stress, and neurovascular injury [11-13]. Women with PCOS exhibit proinflammatory and proatherogenic phenotypes that extend beyond the reproductive dysfunction. Insulin resistance, hyperandrogenism, obesity, and dyslipidemia contribute to endothelial injury through increased oxidative stress, impaired nitric oxide bioavailability, and activation of inflammatory signaling pathways [14,15]. These abnormalities are also recognized as central mechanisms in the development of hypertensive disorders during pregnancy, particularly preeclampsia and eclampsia [16]. Consequently, women with PCOS may enter pregnancy with pre-existing vascular vulnerability, thereby increasing their susceptibility to placental dysfunction, abnormal angiogenesis, and severe hypertensive complications during pregnancy.

The findings of this review are consistent with previous studies, which have demonstrated an increased incidence of gestational hypertension, preeclampsia, and eclampsia among women with PCOS [17,18]. Shared biological mechanisms may explain this association. Elevated circulating concentrations of inflammatory mediators, including interleukin-6, tumor necrosis factor-alpha, and C-reactive protein, have been reported in both women with PCOS and those with eclampsia, suggesting common inflammatory pathways that promote endothelial activation and vascular injury [19]. Furthermore, chronic metabolic disturbances frequently observed in women with PCOS may exacerbate placental ischemia and maternal systemic inflammation, thereby amplifying the risk of adverse obstetric outcomes. An important observation emerging from the current literature is the growing recognition of long-term neurological consequences following severe hypertensive

disorders of pregnancy. Historically, eclampsia was considered an acute obstetric complication that resolves after delivery. However, recent evidence indicates that women with a history of eclampsia remain at an increased risk for persistent cognitive dysfunction, impaired executive performance, memory deficits, reduced attention, and cerebrovascular disease later in life [20,21]. These findings suggest that the neurological effects of eclampsia may extend beyond the peripartum period.

Several mechanisms may underlie the relationship between eclampsia and cognitive impairment. Cerebral endothelial dysfunction, blood–brain barrier disruption, neuroinflammation, oxidative injury, and impaired cerebral autoregulation have all been implicated in the pathogenesis of neurological damage associated with severe hypertensive disorders of pregnancy [22]. Persistent microvascular injury may contribute to chronic cerebral hypoperfusion and structural brain alterations, thereby increasing susceptibility to cognitive decline. Importantly, many of these mechanisms overlap with those already present in women with PCOS, suggesting a potential synergistic effect that may accelerate neurological vulnerability. In addition to pregnancy-related complications, PCOS itself has been associated with adverse neurological outcomes. Chronic insulin resistance and systemic inflammation have been linked to neurodegenerative processes, cerebral microvascular dysfunction, and impaired cognitive performance in non-pregnant populations [23]. Therefore, women with PCOS who subsequently develop eclampsia may experience a cumulative burden of metabolic, vascular, and neuroinflammatory insults that further increase the risk of long-term cognitive impairment. This hypothesis provides a biologically plausible framework linking reproductive endocrinology with maternal neurological health. From a clinical perspective, these findings emphasize the importance of comprehensive risk assessment in women with PCOS before and during pregnancy. Early identification of metabolic abnormalities, optimization of cardiovascular risk factors, weight management, and close antenatal surveillance may reduce the likelihood of severe hypertensive complications [24]. Furthermore, women with a history of eclampsia, particularly those with underlying PCOS, may benefit from long-term neurological monitoring and cognitive assessment. Such strategies could facilitate earlier detection of cognitive decline and improve long-term maternal health outcomes.

Despite the growing body of evidence, several important knowledge gaps remain. Most available studies have evaluated PCOS, eclampsia, or cognitive impairment independently; in contrast, direct investigations examining all three conditions simultaneously are limited. Future prospective cohort studies and mechanistic investigations are needed to clarify causal relationships, identify predictive biomarkers, and determine whether targeted metabolic or anti-inflammatory interventions can mitigate neurological risk in this high-risk population. Overall, the available evidence supports the concept that PCOS may represent an important predisposing condition for both eclampsia and subsequent cognitive impairment. Shared metabolic, inflammatory, endothelial, and neurovascular pathways appear to provide the biological foundation for this association and warrant further investigation.

CONCLUSION

The available evidence suggests that PCOS is not merely a reproductive disorder but a potential precursor to eclampsia and subsequent cognitive impairment through interconnected metabolic, inflammatory, endothelial, and neurovascular mechanisms that require further investigation. Recognizing this continuum is essential for early risk stratification, targeted surveillance, and multidisciplinary interventions aimed at reducing maternal complications and preserving long-term neurological health. Further prospective studies are warranted to clarify the causal pathways and identify effective preventive strategies.

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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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