

Preeclampsia: A Complicated Pregnancy Condition-An Overview, Diagnosis, Management, and Nursing Interventions

Fatimah Mohammed Saad¹, Abdulrahman Hamad Al Rutaiq², Aisha Ali Y Mohammed³, Samirah Mahammad Saad⁴, Ayesha Aayedh Hamdan Aljohani⁵, Abdullah Hamad Alyousef⁶, Asma Yousef Khada Baksh⁷, Mona Hassan Alablan⁸, AFRAH NAIF MARZOQ ALMOTAIRI⁹, Reem Nasser Hassan Abuamria¹⁰

Abstract

Preeclampsia is a hypertensive disorder in pregnancy that affects 2–8% of pregnancies globally, contributing significantly to maternal and neonatal morbidity and mortality. It typically arises after 20 weeks of gestation, with varying severity and associated complications such as eclampsia and HELLP syndrome. Despite advances in diagnosis and management, its etiology remains incompletely understood. This article provides an overview of preeclampsia, focusing on its pathophysiology, epidemiology, clinical diagnosis, management strategies, and nursing interventions. A comprehensive review of the literature was conducted to outline diagnostic criteria, clinical features, and management strategies for preeclampsia. Key interventions, including antihypertensive therapy and seizure prophylaxis, were detailed alongside evidence-based nursing practices. Preeclampsia manifests in two subtypes—early-onset and late-onset—with differing pathophysiological mechanisms. Diagnostic criteria include sustained hypertension, proteinuria, and organ dysfunction. Effective management involves timely diagnosis, blood pressure control using agents like labetalol and nifedipine, and seizure prophylaxis with magnesium sulfate. Nursing care focuses on monitoring maternal-fetal health, educating patients, and providing emotional support. Early detection and timely intervention are critical in managing preeclampsia to prevent severe complications. A multidisciplinary approach, incorporating medical and nursing care, is essential to improve maternal and neonatal outcomes. Further research is required to elucidate the disease's pathogenesis and optimize treatment strategies.

Keywords: *Preeclampsia, Hypertension In Pregnancy, Maternal Morbidity, Nursing Interventions, Magnesium Sulfate, Pregnancy Complications.*

Introduction

Hypertensive disorders of pregnancy are among the leading causes of maternal and perinatal mortality globally. Preeclampsia, with or without severe features, represents a pregnancy-related condition characterized by the onset of hypertension, often accompanied by proteinuria, typically occurring after 20 weeks of gestation and frequently near term. This condition exists on a spectrum of hypertensive disorders in pregnancy, commencing with gestational hypertension and potentially progressing to severe complications, including eclampsia and HELLP syndrome [1]. Preeclampsia accounts for approximately 2% to 8% of pregnancy-related complications worldwide, contributing to over 50,000 maternal deaths and more than 500,000 fetal deaths annually [2]. Early diagnosis and timely intervention are crucial in preventing maternal and neonatal complications, primarily through symptomatic management and the strategic planning of delivery. The diagnostic parameters for pregnancy-induced hypertension are well-defined. Mild hypertension is identified as a systolic blood pressure (SBP) of 140 mm Hg or more, or a diastolic blood

¹ KSA, Ministry Of Health, King Fahad Central Hospital

² KSA, Ministry Of Health, Jizan Mental Hospital

³ KSA, Ministry Of Health.

⁴ KSA, Ministry Of Health, Khamis Mushait General Hospital.

⁵ KSA, Ministry Of Health, Alharth General Hospital

⁶ KSA, Ministry Of Health

⁷ KSA, Ministry Of Health, Dawadmi West Center Of Primary Health Care Dawadmi Hospital

⁸ KSA, Ministry Of Health, Tumair General Hospital

⁹ KSA, Ministry Of Health, Tumair General Hospital

¹⁰ KSA, Ministry Of Health, Khamis Mushayt General Hospital

pressure (DBP) of 90 mm Hg or more, measured on at least two occasions four hours apart. Severe hypertension, defined as an SBP of 160 mm Hg or more or a DBP of 110 mm Hg or more, may be confirmed within shorter intervals. Hypertension occurring prior to 20 weeks of gestation is classified as pre-existing essential or chronic hypertension [3]. The initial manifestations of preeclampsia generally emerge near term, with progression often beginning with gestational hypertension and advancing to severe forms meeting specific laboratory and clinical criteria [4][5][6]. The evolving understanding of pregnancy-induced hypertension has refined its diagnostic criteria, transitioning from the classical triad of hypertension, edema, and proteinuria to a focus on hypertension and organ dysfunction, which may involve renal, hepatic, neurological, hematological, or uteroplacental systems. Nevertheless, the current definitions, largely endorsed by authorities like the American College of Obstetrics and Gynecology (ACOG), are based predominantly on expert consensus rather than primary research [7][5].

Etiology

Despite advancements in diagnosing and managing preeclampsia, its underlying etiology remains poorly understood. The principal pathogenic mechanism is thought to involve uteroplacental ischemia. This hypothesis stems from the frequent observation of placental infarctions in patients with eclampsia and experimental studies wherein autolyzed human placental extracts induced symptoms in animal models, such as convulsions and organ lesions, resembling those seen in fatal eclampsia cases [4][8][9]. Preeclampsia pathophysiology may thus be likened to the release of a toxic substance causing widespread vasculopathy, which, if unchecked, leads to severe complications, including eclampsia-associated seizures [8]. Evidence supporting the connection between placental ischemia and preeclampsia includes experimentally induced ischemia in animal models resulting in hypertension and proteinuria [10][11][12], reduced uterine blood flow in preeclamptic patients compared to unaffected pregnancies [13][14], and consistent placental histopathologic findings indicative of ischemia in preeclampsia and eclampsia [15]. Additionally, typical features of preeclampsia include failure of the physiological transformation of spiral arteries and atherosclerosis [16], as well as a heightened uterine artery pulsatility index in preeclamptic pregnancies [17]. While these findings form a widely accepted framework for understanding the disease, ongoing research seeks to further elucidate the mechanisms underpinning uteroplacental ischemia.

Epidemiology

Preeclampsia and eclampsia remain significant contributors to maternal mortality, accounting for over 50,000 deaths annually worldwide. The incidence of these conditions is closely linked to ethnicity and race, being particularly prevalent among Black and Hispanic populations in the United States, where they constitute approximately 26% of maternal deaths in these groups [4][18][2][19]. On a global scale, preeclampsia affects 2% to 8% of pregnancies, with notable geographic and ethnic variability [20]. For example, higher morbidity rates are observed in developing regions such as Africa and Latin America, while lower incidences are reported among populations in Asia, including China, New Zealand, and Asian Americans, when compared to Native Americans, Black Americans, and Europeans [21][22][23][24]. The risk factors associated with preeclampsia are diverse and multifactorial, encompassing conditions such as chronic hypertension, diabetes mellitus, renal disease, and obesity. Additional factors include short stature, nutritional deficiencies, previous occurrences of gestational hypertension, genetic predispositions, and autoimmune disorders such as systemic lupus erythematosus and antiphospholipid antibody syndrome. Other notable contributors include hydatidiform mole, multiple pregnancies, fetal macrosomia, nulliparity, advanced maternal age, high body mass index (BMI), and the use of assisted reproductive technologies [4][5][25][26].

Pathophysiology

Preeclampsia can be categorized into two primary subtypes: early-onset (placental) and late-onset (maternal) preeclampsia, each with distinct etiologies and clinical phenotypes [27]. Early-onset preeclampsia is characterized by defective placentation, as evidenced by numerous placental infarcts and arterial sclerosis, coupled with placental hypoperfusion resulting from impaired trophoblast invasion and subsequent ischemia [28]. In contrast, late-onset or maternal preeclampsia arises due to interactions between a healthy

placenta and maternal factors, such as endothelial dysfunction, leading to microvascular damage. This subtype typically manifests later in pregnancy and can often be managed expectantly until 37 weeks of gestation. Unlike early-onset preeclampsia, late-onset preeclampsia causes minimal alterations in arterial transformation, thereby preserving placental perfusion [29].

Histopathology

The histopathological characteristics of preeclampsia represent a complex interplay between maternal and placental factors. Examination of placental tissue in cases of preeclampsia often reveals significant maternal vascular malperfusion (MVM) lesions. These lesions include fibrinoid necrosis, atherosclerosis, and an increased number of syncytial knots, indicating abnormalities in maternal blood supply to the placenta. Additionally, trophoblastic abnormalities such as increased syncytial knot formation and reduced trophoblast invasion into maternal spiral arteries are frequently observed. Inflammatory responses, including immune cell infiltration and changes in placental vasculature, are also evident. Emerging studies indicate a higher prevalence of MVM lesions and low placental weight (below the 10th percentile) during the initial onset of preeclampsia compared to subsequent episodes. Furthermore, the first occurrence is associated with a higher incidence of small-for-gestational-age neonates and adverse neonatal outcomes [30]. Sustained diffuse vasculopathy can lead to severe complications, such as seizures in eclampsia. Delivery remains the definitive treatment to prevent further progression by eliminating the defective placenta.

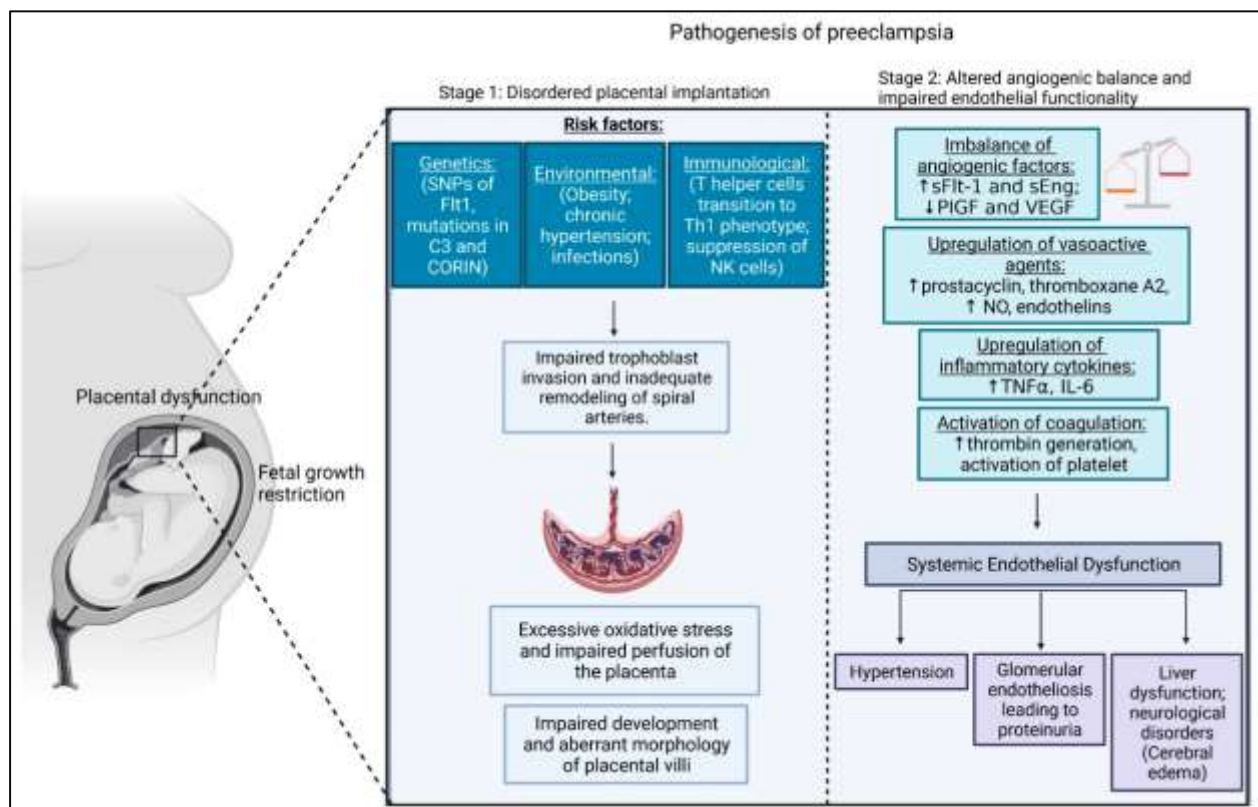


Figure 1. Pathogenesis of Preeclampsia

History and Physical

Preeclampsia often presents with a distinct clinical history and hallmark physical symptoms, although atypical presentations are possible. Common complaints include a new-onset headache resistant to conventional medication, which cannot be attributed to previous conditions such as migraines. This headache may occur alongside visual disturbances, upper right quadrant or epigastric pain, nausea, vomiting, shortness of breath, and increased swelling that exceeds typical pregnancy-related symptoms. Patients

presenting with any of these symptoms should undergo a comprehensive physical examination. Blood pressure measurements are crucial, with systolic blood pressure (SBP) ≥ 140 mmHg or diastolic blood pressure (DBP) ≥ 90 mmHg raising suspicion. For gestational ages of 20 weeks or later, readings from two separate measurements at least four hours apart necessitate further investigation. Recently, diagnostic criteria have expanded to include immediate intervention for sustained severe hypertension (SBP ≥ 160 mmHg or DBP ≥ 110 mmHg) after repeated measurements within minutes. For patients with prior gestational hypertension, the presence of severe-range blood pressure alone qualifies for a diagnosis of preeclampsia with severe features, even if other criteria are absent. Symptoms such as shortness of breath warrant lung auscultation and percussion, while right upper quadrant and epigastric regions should be palpated for tenderness. Edema assessment should target dependent areas like the lower extremities and independent regions such as the face or hands [4].

Evaluation

Following a detailed clinical history and physical examination, patients exhibiting signs of preeclampsia should undergo prompt diagnostic evaluation. This involves laboratory testing tailored to pregnancy-induced hypertension, including urinalysis to assess proteinuria. Diagnostic thresholds include a urine dipstick reading of $\geq 2+$ when alternative methods are unavailable, 24-hour urine protein ≥ 300 mg, or a urine protein-to-creatinine ratio of 0.3 or higher. Additionally, a complete blood count is performed to identify thrombocytopenia (platelet count $< 100,000/\text{mm}^3$), and a complete metabolic panel is used to detect liver dysfunction (liver enzyme levels exceeding twice the upper normal limit) and renal insufficiency (serum creatinine ≥ 1.1 mg/dL or twice the baseline level). Abnormal findings must exclude preexisting conditions or secondary causes to confirm a diagnosis. Although proteinuria accompanied by elevated blood pressure is traditionally central to diagnosing preeclampsia, it is not always present. In cases without proteinuria, alternative diagnostic criteria include thrombocytopenia, renal insufficiency, pulmonary edema, impaired liver function, or persistent new-onset headaches with or without visual disturbances. These findings collectively define preeclampsia without severe features. Furthermore, severe-range blood pressures (SBP ≥ 160 mmHg or DBP ≥ 110 mmHg) measured twice at least four hours apart, even without other abnormalities, can confirm the diagnosis [4][31].

Treatment and Management

The management of preeclampsia prioritizes early detection and appropriate intervention, with a focus on controlling blood pressure and preventing seizures.

Antihypertensive Management

Effective blood pressure control encompasses both immediate management for severe hypertension (defined as systolic ≥ 160 mm Hg and/or diastolic ≥ 110 mm Hg) and maintenance therapy during the antepartum or postpartum periods, depending on the clinical presentation. Antihypertensive agents that are both effective and fetal-safe include beta-blockers such as labetalol, calcium channel blockers like nifedipine, alpha-2 agonists such as clonidine, and vasodilators including hydralazine. For severe hypertension, intravenous (IV) labetalol, IV hydralazine, or orally (PO) administered immediate-release nifedipine are commonly employed. The selection of these medications often depends on IV access availability, with PO nifedipine being preferred when IV access is not feasible [32]. Maintenance therapy typically involves PO labetalol, extended-release nifedipine, or extended-release clonidine, with no significant difference in efficacy among these options. Extended-release medications, such as daily dosed nifedipine or weekly clonidine patches, provide advantages in patients with compliance challenges [33].

Antiseizure Management

Intravenous magnesium sulfate remains the first-line therapy for seizure prophylaxis in preeclampsia with severe features [4, 25, 31]. For patients in whom magnesium sulfate is contraindicated, levetiracetam is a viable alternative. Management of eclampsia, characterized by seizure activity, involves initial treatment with IV benzodiazepines [34]. For recurrent seizures unresponsive to magnesium sulfate or when

contraindicated, alternative agents include lorazepam (2–4 mg IV, repeatable once after 10–15 minutes), diazepam (5–10 mg IV every 5–10 minutes, maximum 30 mg), phenytoin (15–20 mg/kg IV, with an additional 10 mg/kg IV dose if necessary after 20 minutes), and levetiracetam (500 mg IV or orally, repeatable in 12 hours).

Antepartum Management and Delivery Timing

Fetal assessment encompasses ultrasonographic evaluation of the amniotic fluid index, estimated fetal weight, and antenatal tests such as non-stress tests and biophysical profiles. Decisions regarding delivery versus expectant management are often informed by fetal status. Delivery of the fetus is the definitive treatment for preeclampsia. In preterm cases, expectant management may be considered for patients with well-controlled gestational hypertension or preeclampsia without severe features, provided antepartum testing results are normal. Monitoring should include serial ultrasonography, weekly antenatal testing, and vigilant observation of maternal symptoms, blood pressure, and laboratory values. According to the American College of Obstetricians and Gynecologists (ACOG), delivery is recommended at 37 0/7 weeks for patients with gestational hypertension or preeclampsia without severe features. In cases of preeclampsia with severe features at or beyond 34 0/7 weeks, delivery is advised after maternal stabilization without delay for steroid administration. For gestations under 34 0/7 weeks, stabilization of maternal and fetal health should precede individualized management, often requiring inpatient or outpatient care. While this approach lacks robust evidence, it relies on expert consensus and tailored care plans [35, 36]. Expedient delivery is warranted for maternal or fetal deterioration. Fetal indications include abnormal antenatal testing and sustained reversed end-diastolic umbilical artery flow. Maternal indications encompass uncontrolled hypertension, persistent headaches or visual disturbances, epigastric or right upper quadrant pain unresponsive to medical therapy, myocardial infarction, stroke, pulmonary edema, HELLP syndrome, eclampsia, or suspected placental abruption. Antenatal steroids for fetal lung maturity should precede delivery before 34 0/7 weeks, although delivery should not be delayed. This recommendation also applies to late preterm periods (34 0/7 to 36 6/7 weeks).

Differential Diagnosis

The differential diagnosis of preeclampsia includes a broad spectrum of conditions that mimic its clinical presentation. These conditions encompass chronic hypertension, gestational hypertension, antiphospholipid antibody syndrome, thrombotic microangiopathies, lupus, epilepsy or seizure disorders, chronic renal disease, chronic liver disease, and other significant medical disorders such as pheochromocytoma or other endocrinopathies [4, 37]. Incorporating an accurate and comprehensive differential diagnosis is critical to distinguishing preeclampsia from other conditions and guiding optimal management strategies.

Prognosis

The prognosis of preeclampsia is markedly improved through early diagnosis, timely medical intervention, and consistent maternal and fetal surveillance. These measures significantly enhance outcomes for both the mother and the fetus. Despite advancements in maternal healthcare, preeclampsia remains a major contributor to maternal mortality, accounting for nearly a quarter of maternal deaths in some ethnic populations. The Caribbean and Latin American populations experience the highest rates, followed by Asian and Black populations. The condition underscores the critical need for timely medical care and ongoing monitoring to prevent its progression and associated complications. Healthcare providers must adopt evidence-based strategies to ensure routine surveillance, which includes blood pressure monitoring, laboratory testing, and fetal assessments. Timely intervention can lead to improved survival rates and reduced long-term health complications for both the mother and the neonate. Addressing social and economic disparities in healthcare access is equally crucial to mitigate risks among vulnerable populations. These efforts are particularly important for populations facing systemic barriers to receiving adequate prenatal care. Public health initiatives, community-based education programs, and enhanced healthcare infrastructure are essential components in reducing the burden of preeclampsia in at-risk populations. By prioritizing comprehensive and culturally sensitive care, healthcare providers can significantly decrease the

morbidity and mortality associated with preeclampsia. Emphasizing prevention, early detection, and appropriate treatment is critical in improving maternal health outcomes globally [4][25].

Complications

In preeclampsia, delaying the delivery of the fetus during the late preterm period often exacerbates maternal and fetal risks. Severe hypertension is a frequent outcome, leading to serious complications such as eclampsia, HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count), pulmonary edema, myocardial infarction, acute respiratory distress syndrome, stroke, renal impairment, and retinal injury. Fetal complications include restricted growth, placental abruption, or, in extreme cases, fetal and maternal mortality. These outcomes underscore the importance of precise decision-making in determining the timing of delivery [4][37]. Medical interventions for preeclampsia are not without risks. Antihypertensive agents such as labetalol, hydralazine, and nifedipine, while effective, can cause adverse effects, including tachycardia, hypotension, headaches, and fetal heart rate abnormalities. Furthermore, magnesium sulfate, employed for seizure prophylaxis, introduces the potential for respiratory depression and, in severe cases, cardiac arrest. Frequent laboratory testing of serum magnesium levels and physical assessments every 4 to 6 hours are imperative for patients receiving this therapy. The complexity of managing these complications calls for meticulous clinical judgment and a multidisciplinary approach to care. Clinicians must balance the benefits of prolonging pregnancy to allow for fetal development against the risks posed by the progression of preeclampsia. This delicate equilibrium requires continuous monitoring and prompt intervention to optimize outcomes for both the mother and the fetus. Enhanced education and training for healthcare teams are vital to effectively manage these risks and ensure safety [4].

Deterrence and Patient Education

The prevention and management of preeclampsia heavily depend on early diagnosis, timely intervention, and comprehensive surveillance. Central to this effort is patient education, which equips individuals with the knowledge necessary to identify warning signs and seek timely care. Healthcare providers play a pivotal role in counseling patients about the definition of preeclampsia, associated symptoms such as persistent headaches, visual disturbances, abdominal pain, and the patient-specific factors that heighten their risk. By fostering an environment of informed decision-making, clinicians can significantly improve adherence to medical recommendations and outcomes. Maternal mortality associated with preeclampsia disproportionately affects women from low socioeconomic backgrounds and those with limited access to education. These disparities necessitate a nuanced approach to care, where clinicians tailor their communication to the specific needs and comprehension levels of their patients. Effective education strategies might include visual aids, community workshops, and culturally appropriate messaging that resonate with diverse populations. Healthcare systems must also address systemic barriers to care, such as inadequate healthcare infrastructure and financial constraints. Public health initiatives targeting at-risk groups can bridge gaps in knowledge and access, empowering women to advocate for their health. Overall, comprehensive education and support systems form the cornerstone of effective deterrence strategies, reducing both the incidence and severity of preeclampsia and improving outcomes for mothers and infants [4].

Enhancing Healthcare Team Outcomes

Managing preeclampsia presents unique challenges due to the dual focus on maternal and fetal care. The condition demands a collaborative, multidisciplinary approach involving physicians, advanced practitioners, nurses, pharmacists, and allied health professionals. Effective team coordination is essential to delivering high-quality, patient-centered care, which enhances maternal and neonatal outcomes. A cohesive healthcare team must prioritize patient education and support. Ensuring that patients feel informed and understood regarding their condition fosters trust and promotes adherence to treatment plans. Comprehensive communication among team members is equally crucial, enabling the seamless exchange of information about patient complaints, clinical signs, laboratory results, treatment regimens, and delivery strategies. The use of evidence-based protocols and guidelines, coupled with continuous professional development, ensures that all team members are equipped to manage the complexities of preeclampsia effectively.

Training programs focused on recognizing early warning signs, managing hypertensive crises, and administering magnesium sulfate can improve clinical outcomes. Furthermore, integrating technology such as electronic health records (EHRs) enhances information sharing and streamlines care coordination. By fostering collaboration and leveraging the expertise of diverse professionals, healthcare teams can address the multifaceted needs of patients with preeclampsia. This approach not only reduces morbidity and mortality rates but also promotes a culture of safety and excellence in maternal healthcare. Continuous evaluation of team performance and patient outcomes is vital to identifying areas for improvement and ensuring the delivery of optimal care [4].

Nursing Diagnosis and Intervention Plans

Effective nursing diagnosis and intervention plans are critical in the management of patients with preeclampsia, focusing on both maternal and fetal health outcomes. Nursing diagnoses in this context encompass a range of physical, psychological, and educational needs, which guide the development of tailored intervention strategies. These plans aim to address immediate medical concerns, prevent complications, and promote long-term health. One primary nursing diagnosis for preeclampsia is "risk for impaired maternal and fetal perfusion related to hypertension." This diagnosis highlights the need for immediate and continuous monitoring of blood pressure, fetal heart rate, and maternal symptoms such as persistent headaches or vision changes. Nursing interventions include frequent blood pressure assessments, administration of prescribed antihypertensive medications, and evaluation of fetal well-being through non-stress tests and ultrasound monitoring. Early detection and management of perfusion issues are essential to prevent severe complications such as placental abruption or fetal growth restriction. Another significant nursing diagnosis is "risk for injury related to seizure activity in eclampsia." Interventions focus on seizure prevention through the administration of magnesium sulfate as prescribed, monitoring for signs of magnesium toxicity, and ensuring safety measures such as padded side rails. Nurses must also educate patients and families on recognizing symptoms that warrant immediate medical attention, such as severe epigastric pain or changes in mental status. These measures aim to minimize the risks associated with eclamptic seizures, ensuring maternal and fetal safety.

A third relevant diagnosis is "anxiety related to potential complications of preeclampsia and lack of knowledge about the condition." The emotional and psychological impact of preeclampsia on patients can hinder compliance with medical recommendations and exacerbate health risks. Nursing interventions include providing clear, empathetic communication about the condition, its potential outcomes, and the steps being taken to ensure safety. Patient education should be individualized and culturally appropriate, utilizing tools such as visual aids or written materials to enhance understanding. Nurses also play a vital role in providing emotional support, encouraging patients to express their concerns, and referring them to mental health professionals when necessary. The diagnosis of "imbalanced nutrition: less than body requirements related to nausea, vomiting, or restricted diet" is also pertinent in managing preeclampsia. Adequate maternal nutrition is vital for fetal development and maternal well-being. Interventions include providing dietary counseling to ensure a balanced intake of essential nutrients, monitoring for signs of malnutrition, and collaborating with dietitians to develop personalized meal plans. Nurses must also educate patients on foods to avoid, such as those high in sodium, which can exacerbate hypertension.

"Risk for ineffective coping related to the stress of a high-risk pregnancy" is another critical nursing diagnosis. High-risk pregnancies often lead to significant psychological strain, affecting both the patient and their support network. Nursing interventions in this domain focus on fostering resilience and coping mechanisms. Techniques such as mindfulness training, stress-reduction exercises, and peer support groups can empower patients to navigate the challenges of preeclampsia. Nurses can also facilitate open communication within families, promoting shared decision-making and reducing the emotional burden on the patient. The diagnosis of "impaired fluid balance related to proteinuria and hypertension" necessitates vigilant monitoring of fluid intake and output, along with assessment of edema and laboratory values such as serum albumin levels. Nursing interventions include ensuring adequate hydration, managing diuretic therapy as prescribed, and educating patients on recognizing symptoms of fluid overload or dehydration. Collaborative care involving physicians and dietitians can optimize fluid balance and mitigate the risks associated with preeclampsia.

Finally, the “risk for impaired maternal-fetal bonding related to hospitalization and the stress of preeclampsia” underscores the importance of promoting early and meaningful interactions between mother and baby. Nurses can encourage bonding through strategies such as involving the patient in fetal monitoring sessions, providing educational materials about fetal development, and facilitating family-centered care during hospital stays. These measures help maintain the psychological connection between the mother and her unborn child, even in the face of medical challenges. In conclusion, nursing diagnosis and intervention plans for patients with preeclampsia are comprehensive and multifaceted, addressing both physical and psychosocial needs. Effective implementation of these plans requires a patient-centered approach, clear communication, and interdisciplinary collaboration. By tailoring care to the unique circumstances of each patient, nurses play a pivotal role in improving maternal and fetal outcomes while promoting overall well-being. Continuous education and support are vital components of these efforts, ensuring that patients feel empowered and informed throughout their care journey.

Conclusion

Preeclampsia remains a formidable challenge in maternal healthcare, significantly impacting both maternal and fetal outcomes. Despite advances in medical understanding, its pathogenesis remains poorly understood, underscoring the need for ongoing research. The condition’s dual subtypes—early-onset and late-onset—highlight the multifaceted nature of its etiology, which combines placental dysfunction and maternal factors. These subtypes demand tailored clinical approaches to ensure effective management. Early and accurate diagnosis is critical for optimizing outcomes. Current diagnostic protocols prioritize blood pressure monitoring and laboratory assessments, including proteinuria evaluation and organ function tests. Management strategies focus on controlling hypertension and preventing seizures, with agents like labetalol and magnesium sulfate forming the cornerstone of treatment. In cases of severe preeclampsia, prompt delivery remains the definitive intervention to mitigate maternal and fetal risks. Nursing care plays an integral role in the holistic management of preeclampsia. Nurses are pivotal in monitoring maternal and fetal well-being, identifying early warning signs, and ensuring adherence to therapeutic regimens. They also provide essential patient education, addressing concerns related to disease progression and postnatal recovery. Furthermore, emotional support from nursing professionals significantly alleviates the psychological burden often associated with this condition. While significant strides have been made in managing preeclampsia, challenges persist, particularly in low-resource settings where access to diagnostic and therapeutic resources is limited. Strategies to improve global outcomes must prioritize equitable access to maternal healthcare, education, and early screening programs. Multidisciplinary collaboration between obstetricians, nurses, and researchers is paramount in advancing the understanding and management of this complex condition. In conclusion, the fight against preeclampsia necessitates a comprehensive and collaborative approach, integrating evidence-based medical and nursing practices. Continued research into its etiology and pathophysiology is essential to develop targeted interventions and improve outcomes for affected mothers and their infants.

References

- Erez O, Romero R, Jung E, Chaemsaithong P, Bosco M, Suksai M, Gallo DM, Gotsch F. Preeclampsia and eclampsia: the conceptual evolution of a syndrome. *Am J Obstet Gynecol.* 2022 Feb;226(2S):S786-S803.
- Macedo TCC, Montagna E, Trevisan CM, Zaia V, de Oliveira R, Barbosa CP, Laganà AS, Bianco B. Prevalence of preeclampsia and eclampsia in adolescent pregnancy: A systematic review and meta-analysis of 291,247 adolescents worldwide since 1969. *Eur J Obstet Gynecol Reprod Biol.* 2020 May;248:177-186.
- Battarbee AN, Sinkey RG, Harper LM, Oparil S, Tita ATN. Chronic hypertension in pregnancy. *Am J Obstet Gynecol.* 2020 Jun;222(6):532-541.
- Gestational Hypertension and Preeclampsia: ACOG Practice Bulletin, Number 222. *Obstet Gynecol.* 2020 Jun;135(6):e237-e260.
- Homer CS, Brown MA, Mangos G, Davis GK. Non-proteinuric pre-eclampsia: a novel risk indicator in women with gestational hypertension. *J Hypertens.* 2008 Feb;26(2):295-302.
- Tanner MS, Davey MA, Mol BW, Rolnik DL. The evolution of the diagnostic criteria of preeclampsia-eclampsia. *Am J Obstet Gynecol.* 2022 Feb;226(2S):S835-S843.

- Reddy M, Fenn S, Rolnik DL, Mol BW, da Silva Costa F, Wallace EM, Palmer KR. The impact of the definition of preeclampsia on disease diagnosis and outcomes: a retrospective cohort study. *Am J Obstet Gynecol.* 2021 Feb;224(2):217.e1-217.e11.
- Phipps EA, Thadhani R, Benzting T, Karumanchi SA. Pre-eclampsia: pathogenesis, novel diagnostics and therapies. *Nat Rev Nephrol.* 2019 May;15(5):275-289.
- Jung E, Romero R, Yeo L, Gomez-Lopez N, Chaemsaitong P, Jaovisidha A, Gotsch F, Erez O. The etiology of preeclampsia. *Am J Obstet Gynecol.* 2022 Feb;226(2S):S844-S866.
- Berger M, Cavanagh D. Toxemia Of Pregnancy. The Hypertensive Effect Of Acute Experimental Placental Ischemia. *Am J Obstet Gynecol.* 1963 Oct 01;87:293-305.
- Labarrere CA, DiCarlo HL, Bammerlin E, Hardin JW, Kim YM, Chaemsaitong P, Haas DM, Kassab GS, Romero R. Failure of physiologic transformation of spiral arteries, endothelial and trophoblast cell activation, and acute atherosclerosis in the basal plate of the placenta. *Am J Obstet Gynecol.* 2017 Mar;216(3):287.e1-287.e16.
- Staff AC, Johnsen GM, Dechend R, Redman CWG. Preeclampsia and uteroplacental acute atherosclerosis: immune and inflammatory factors. *J Reprod Immunol.* 2014 Mar;101-102:120-126.
- McMaster-Fay RA. Failure of physiologic transformation of the spiral arteries of the uteroplacental circulation in patients with preterm labor and intact membranes. *Am J Obstet Gynecol.* 2004 Nov;191(5):1837-8; author reply 1838-9.
- Mlambo ZP, Khaliq OP, Moodley J, Naicker T. Circulatory and Placental Expression of Soluble Fms-like Tyrosine Kinase-1 and Placental Growth Factor in HIV-infected Preeclampsia. *Curr Hypertens Rev.* 2023;19(1):27-33.
- Young J. The Etiology of Eclampsia and Albuminuria and Their Relation to Accidental Hæmorrhage. *Trans Edinb Obstet Soc.* 1914;39:153-202.
- De Wolf F, Robertson WB, Brosens I. The ultrastructure of acute atherosclerosis in hypertensive pregnancy. *Am J Obstet Gynecol.* 1975 Sep 15;123(2):164-74.
- Gallo DM, Poon LC, Akolekar R, Syngelaki A, Nicolaides KH. Prediction of preeclampsia by uterine artery Doppler at 20-24 weeks' gestation. *Fetal Diagn Ther.* 2013;34(4):241-7.
- Miller EC, Wilczek A, Bello NA, Tom S, Wapner R, Suh Y. Pregnancy, preeclampsia and maternal aging: From epidemiology to functional genomics. *Ageing Res Rev.* 2022 Jan;73:101535.
- Wheeler SM, Myers SO, Swamy GK, Myers ER. Estimated Prevalence of Risk Factors for Preeclampsia Among Individuals Giving Birth in the US in 2019. *JAMA Netw Open.* 2022 Jan 04;5(1):e2142343.
- Shi P, Zhao L, Yu S, Zhou J, Li J, Zhang N, Xing B, Cui X, Yang S. Differences in epidemiology of patients with preeclampsia between China and the US (Review). *Exp Ther Med.* 2021 Sep;22(3):1012.
- Osungbade KO, Ige OK. Public health perspectives of preeclampsia in developing countries: implication for health system strengthening. *J Pregnancy.* 2011;2011:481095.
- Fasanya HO, Hsiao CJ, Armstrong-Sylvester KR, Beal SG. A Critical Review on the Use of Race in Understanding Racial Disparities in Preeclampsia. *J Appl Lab Med.* 2021 Jan 12;6(1):247-256.
- Chu H, Ramola R, Jain S, Haas DM, Natarajan S, Radivojac P. Using Association Rules to Understand the Risk of Adverse Pregnancy Outcomes in a Diverse Population. *Pac Symp Biocomput.* 2023;28:209-220.
- Yang Y, Le Ray I, Zhu J, Zhang J, Hua J, Reilly M. Preeclampsia Prevalence, Risk Factors, and Pregnancy Outcomes in Sweden and China. *JAMA Netw Open.* 2021 May 03;4(5):e218401.
- Sibai BM, el-Nazer A, Gonzalez-Ruiz A. Severe preeclampsia-eclampsia in young primigravid women: subsequent pregnancy outcome and remote prognosis. *Am J Obstet Gynecol.* 1986 Nov;155(5):1011-6.
- Thoma ME, Boulet S, Martin JA, Kissin D. Births resulting from assisted reproductive technology: comparing birth certificate and National ART Surveillance System Data, 2011. *Natl Vital Stat Rep.* 2014 Dec 10;63(8):1-11.
- Nirupama R, Divyashree S, Janhavi P, Muthukumar SP, Ravindra PV. Preeclampsia: Pathophysiology and management. *J Gynecol Obstet Hum Reprod.* 2021 Feb;50(2):101975.
- Goswami D, Tannetta DS, Magee LA, Fuchisawa A, Redman CW, Sargent IL, von Dadelszen P. Excess syncytiotrophoblast microparticle shedding is a feature of early-onset pre-eclampsia, but not normotensive intrauterine growth restriction. *Placenta.* 2006 Jan;27(1):56-61.
- Varghese, B., Joy, C. A., Josyula, J. V. N., Jangili, S., Talukdar, R. K., Mutheni, S. R., & Adela, R. (2023). Machine learning-based protein signatures for differentiating hypertensive disorders of pregnancy. *Hypertension Research*, 46(11), 2513-2526.
- Dankó I, Kelemen E, Tankó A, Cserni G. Correlations of Placental Histopathology, Neonatal Outcome, and Cardiotocogram Baseline Variability and Acceleration Patterns in the Growth Restricted Preterm Population. *Pediatr Dev Pathol.* 2023 Sep-Oct;26(5):447-457.
- Kattah AG, Garovic VD. The management of hypertension in pregnancy. *Adv Chronic Kidney Dis.* 2013 May;20(3):229-39.
- Vigil-De Gracia P, Lasso M, Ruiz E, Vega-Malek JC, de Mena FT, López JC., or the HYLEA treatment study. Severe hypertension in pregnancy: hydralazine or labetalol. A randomized clinical trial. *Eur J Obstet Gynecol Reprod Biol.* 2006 Sep-Oct;128(1-2):157-62.
- Xu B, Charlton F, Makris A, Hennessy A. Antihypertensive drugs methyldopa, labetalol, hydralazine, and clonidine improve trophoblast interaction with endothelial cellular networks in vitro. *J Hypertens.* 2014 May;32(5):1075-83; discussion 1083.
- Dimitriadis, E., Rolnik, D. L., Zhou, W., Estrada-Gutierrez, G., Koga, K., Francisco, R. P., ... & Menkhorst, E. (2023). Preeclampsia. *Nature reviews Disease primers*, 9(1), 8.
- Fu J, Li C, Gou W, Lee A, Li X, Chen Q. Expectant or outpatient management of preeclampsia before 34 weeks: safe for mother but associated with increased stillbirth risk. *J Hum Hypertens.* 2019 Sep;33(9):664-670.
- Duvekot JJ, Duijnhoven RG, van Horen E, Bax CJ, Bloemenkamp KW, Brussé IA, Dijk PH, Franssen MT, Franx A, Oudijk MA, Porath MM, Scheepers HC, van Wassenaer-Leemhuis AG, van Drongelen J, Mol BW, Ganzevoort W., TOTEM study collaboration group. Temporizing management vs immediate delivery in early-onset severe

preeclampsia between 28 and 34 weeks of gestation (TOTEM study): An open-label randomized controlled trial. Acta Obstet Gynecol Scand. 2021 Jan;100(1):109-118.

Amaral LM, Wallace K, Owens M, LaMarca B. Pathophysiology and Current Clinical Management of Preeclampsia. Curr Hypertens Rep. 2017 Aug;19(8):61.

التمريضية والتدخلات، الإدارة، التشخيص، عامة لمحة - معقدة حمل حالة: الولادة قبل ما تسمم مقدمات

الملخص:

ويساهم، عالمياً الحمل حالات من 2-8% على يؤثر الحمل أثناء الدم ضغط ارتفاع اضطراب هي الولادة قبل ما تسمم مقدمات: الخلفية شدتها في اختلاف مع، الحمل من أسبوعاً 20 بعد عادةً تظهر. الولادة وحديثي الأمهات بين والوفيات الاعتلال معدلات في كبير بشكل مسيبتها تزال لا، والإدارة التشخيص في التقدم من الرغم على. هيلب ومتلازمة الولادة قبل ما تسمم مثل بها المرتبطة والمضاعفات بالكامل مفهومة غير

ص والتشخيص، الأوبئة وعلم، المرضية الفيزيولوجيا على التركيز مع الولادة قبل ما تسمم مقدمات عن شاملة نظرة المقال هذا يقدم: الهدف التمرضية والتدخلات، الإدارة واستراتيجيات، السريري

قبل ما تسمم مقدمات إدارة واستراتيجيات السريرية والميزات التشخيص معايير لتوضيح للأدبيات شاملة مراجعة إجراء تم: الطرق ضحية التمريض الممارسات جانب إلى، التشنجات من والوقاية للضغط الخافض العلاج ذلك في بما، الرئيسية التدخلات تفصيل تم. الولادة الأدلة إلى المستندة

بيررما تشمل. مختلفة مرضية فيزيولوجية آليات مع - والمتأخر المبكر - فرعيين بنوعين الولادة قبل ما تسمم مقدمات يظهر: النتائج، المناسب الوقت في التشخيص الفعالة الإدارة تتضمن الأعضاء في الوظيفي والخلل، البروتينية، المستمر الدم ضغط ارتفاع التشخيص الرعاية تركيز. المغنيسيوم كبريتات باستخدام التشنجات من والوقاية، والنيفيديبين اللابيتالول مثل أدوية باستخدام الدم ضغط في التحكم النفسي الدعم وتقديم، المرضى وتثقيف، والجنين الأم صحة مراقبة على التمرضية

مضاعفات حدوث لمنع الولادة قبل ما تسمم مقدمات إدارة في حاسماً أمراً المناسب الوقت في والتدخل المبكر الاكتشاف يعد: الخلاصة إلى اجتهد هناك. والتمريضية الطبية الرعاية يشمل التخصصات متعدد نهج اتباع الولادة وحديثي للأمهات النتائج تحسين يتطلب شديدة العلاج استراتيجيات وتحسين المرض مسببات لتوضيح الأبحاث من مزيد

كبريتات، التمرضية التدخلات، الأمهات اعتلال، الحمل أثناء الدم ضغط ارتفاع، الولادة قبل ما تسمم مقدمات: المفاتيح الكلمات الحمل مضاعفات، المغنيسيوم