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Unlocking the Secrets of Electrolyte Physiology in Pre-Eclampsia: A Vital Insight into Maternal and Fetal Health and its Management

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Abstract: ***Pre - eclampsia is a multisystemic illness of pregnancy in people who are predisposed genetically. It more frequently happens in first pregnancies, and while it raises blood pressure, it mostly affects the mother's renal, cerebral, hepatic, and coagulation capabilities. The fetus shows detrimental effects because of placental insufficiency brought on by improper "placentation" or the inadequate penetration of the maternal vasculature by trophoblasts, and sometimes maybe because of abnormal autocoid production. The placenta is the causative behind pre - eclampsia, and the only known treatment is delivery of the placenta. Its symptoms are thought to be secondary to organ hypoperfusion, which results from vasoconstriction, intravascular coagulation, and a decreased amount of maternal blood. According to most recent theories, pre - eclampsia is caused by excessive damage to maternal endothelium, possibly due to a cytotoxic substance secreted by the placenta. Although, this theory is widely accepted, there is little scientific support for it. It has proven to be challenging to define the aberrant balance of vasoactive factors in pre - eclampsia. Despite lower plasma concentrations of angiotensin II (A - II), Renin, and Aldosterone, injected Angiotensin - II had increased pressor activity. The balance of thromboxane/prostacyclin favors vasoconstriction and platelet aggregation, and prostacyclin production appears to be diminished. Neither decreased nitric oxide production nor increased endothelin production are suggested by strong evidence. In spite of the contraction in plasma volume, Atrial Natriuretic Peptide (ANP) concentrations in plasma are strangely increased. For e. g., why one patient experiences hepatic ischemia, and another has a comparable level of hypertension but involves a different organ system, such as renal insufficiency but adequate liver function, is a fascinating discovery that has not yet been fully explained. Volume homeostasis is disturbed with re - distribution of intravascular volume of fluids to the interstitial fluid area because of raised capillary permeability and also in some instances decreased plasma oncotic pressure. This re - distribution is not always clinically apparent as peripheral edema. Whether this change in volume of fluids is compensated for by veno - constriction and maintenance of ample cardiac output is undetermined. Improved understanding of the pathology of pre - eclampsia is absolutely necessary to allow better clinical assessment of this consequential disorder.***

Keywords: multisystemic illness, organ hypoperfusion, cytotoxic, Atrial Natriuretic Peptide, Capillary Permeability, Consequential Disorder

1. Introduction

Hypertension is a huge universal issue and it complicates nearly 10% of all pregnancies (1). Preeclampsia is operationally defined as a Triad of: -

1) Hypertension,
2) Proteinuria and
3) Edema

occurring after 20 weeks gestation in previously normotensive patients. It is very specific to the human pregnancy (2). It is a transient but potentially dangerous and heavy loss causing complication of pregnancy.

Preeclampsia is one of the major leading causes of maternal and fetal mortality and morbidity (2). It is a well - known fact that electrolytes have an important role in etio - pathogenesis of Hypertension in almost every other patient (1). Electrolytes viz. **Calcium (Ca^{2+}), Magnesium (Mg^{2+}) Sodium (Na^{+}) and Potassium (K+)** play a crucial role in preeclampsia as they contribute significantly in the functioning of the smooth vascular muscles (1). **Ca^{2+}** plays a critical and a crucial role in the function of smooth vascular muscles (1).

Changes in plasma **Ca^{2+}** concentration will lead to raised blood pressure (1, 2, 3). **Mg^{2+}** acts as co - factor for numerous enzymes (for e. g., Sodium Potassium ATPase) and is involved in peripheral vasodilatation (1, 2, 3). Some other studies show that blood **Ca^{2+}** and **Mg^{2+}** cause a relaxant effect on the blood vessels of pregnant women (2, 4). Hence this present study probes into the alteration of above - mentioned serum electrolytes in a normal woman who is pregnant but pre - eclamptic.

Epidemiology:

Pre - eclampsia is a multisystemic disorder that usually complicates nearly **3%–8%** of pregnancies in all of Western countries and **6.9 %** in the Indian population and constitutes a leading cause for morbidity and mortality worldwide. (5, 6) Approximately, **10%–15%** of maternal deaths are directly associated with both pre - eclampsia and eclampsia. (6) Some of the epidemiological values support the hypothesis for a genetic and/or immunological cause. The risk of pre - eclampsia is nearly **2 times to 5 times** higher in pregnant women with a maternal history of the related disorder.

Depending specifically on ethnicity, the incidence rate of pre - eclampsia has a range from **3% - 7%** in healthy nulliparas

and **1% - 3%** in multiparas. Moreover, nulliparity and a new partner have shown as important risk factors.

Other occurring risk factors identified are, previously known medical history of Chronic Hypertension, any sort of Chronic or Acute Kidney Disease, Diabetes, Obesity, born in Africa, age greater than or equal to 35 years, and pregnancy characteristics, such as twins or molar type of pregnancy, previous pre - eclampsia, or fetal congenital abnormality. (7, 8) High altitude has shown to increase the incidence of pre - eclampsia, and is attributed to greater placental hypoxia, smaller uterine artery diameter, and lower uterine artery blood flow. (9)

Pre - eclampsia may be many times life - threatening for both the mother and child, increasing feto - maternal morbidity and mortality. (10) In the mother, pre - eclampsia sometimes causes premature cardiovascular and pulmonary disease, such as Chronic Hypertension patients, ischemic heart diseases, and possibly Stroke in later life, (11) while in children being born after pre - eclamptic pregnancies and also who are relatively smaller at birth, have an inflated risk of stroke, coronary heart disease, and Metabolic Syndrome in adults. (12 - 14)

The only one curative treatment being delivery, management must continuously balance the risk–benefit ratio of induced preterm delivery and maternal–fetal complications. Screening of women at higher risks and preventing the recurrences are also vital issues in the treatment and management of pre - eclampsia.

Pathophysiology and Electrolyte imbalance: -

During normal pregnancy period, the villous cytotrophoblasts invade into the inner **1/3rd** of the myometrium layer, and spiral arteries tend to lose their endothelium layer and most of the muscle fibers lining them. These modifications which are structural in type are associated with alterations in function, such that the spiral arteries become very low resistance vessels, and thus are less sensitive, or even sometimes insensitive, to substances that are vasoconstrictive.

Pre - eclampsia has a very complex patho - physiology, the primary cause for it is abnormal placentation. Pre - eclampsia is characterized by cytotrophoblast cells' defective invasion of the spiral arteries. More recent studies have shown us that cytotrophoblast cell invasion into the uterus is actually a very unique pathway for differentiation in which the fetal cells adopt certain attributes of the maternal endothelium they normally replace.

This differentiation pathway is disrupted in pre - eclampsia. (15) Vascular tone is mostly controlled by the nitric oxide pathway, which may be connected to the anomalies. Furthermore, nitric oxide production in the mother is inhibited, which hinders embryo implantation. (16) Higher susceptibility to vasoconstriction and consequently to oxidative stress and persistent placental ischemia is caused by increased uterine vascular resistance. Fetal problems brought on by this persistent placental ischemia include intrauterine growth retardation and intrauterine mortality. Simultaneously, oxidative stress causes substances like cytokines, oxidized lipids, free radicals, and serum soluble vascular endothelial growth factor 1 to be released into the mother's bloodstream.

Endothelial dysfunction (17), vascular hyperpermeability, thrombophilia, and hypertension are caused by these anomalies, which are meant to make up for the decreased flow in the uterine arteries caused by peripheral vasoconstriction.

The mother's clinical symptoms, which include impairment of the hepatic endothelium that contributes to the onset of the **HELLP** (Hemolysis, Elevated Liver enzymes and Low Platelet count) syndrome, impairment of the cerebral endothelium that causes refractory neurological disorders, and even eclampsia, are caused by endothelial dysfunction. Vascular endothelial growth factor deficiency in the podocytes increases the ability of endotheliosis to obstruct the basement membrane's slit diaphragms, contributing to reduced glomerular filtration and proteinuria. In conclusion, endothelial dysfunction fosters microangiopathic hemolytic anemia, and edema, especially in the lower limbs or lungs, is brought on by vascular hyperpermeability linked to low serum albumin.

The most important thing to realize is that abnormal placentation is the main cause of pre - eclampsia. Two common theories appear to be interlinked, i. e., a genetic theory (18, 19) and an immunological theory. (20, 21) Several susceptibility genes may exist for pre - eclampsia. (19, 22) These genes probably interact in the hemostatic and cardiovascular systems, as well as in the inflammatory response. A few have been found and in candidate gene studies they have shown evidence of association with multiple genes, such as eNOS on 7q36 and angiotensinogen on 1 - q42–43; other significant loci are 2p12, 2p25, 9p13, and 10q22.1.19)

One way to think of pre - eclampsia is as a weakness in the mother's immune system that keeps it from identifying the fetoplacental unit. Excessive production of immune cells causes secretion of tumor necrosis factor alpha which induces apoptosis of the extra villous cytotrophoblast. (23)

Because women with pre - eclampsia have lower levels of HLA - G and HLA - E, the human leukocyte antigen (HLA) system may also be involved in the aberrant invasion of the spiral arteries. (24) During normal pregnancies, the interaction between these cells and the trophoblast is due to secretion of vascular endothelial growth factor and placental growth factor by natural killer cells. High levels of soluble FMS - like tyrosine kinase 1 (**sFlt - 1**), an antagonist of vascular endothelial growth factor and placental growth factor, have been found in women with pre - eclampsia. (23, 24)

Predicting pre - eclampsia may therefore be aided by assays for sFLT - 1, placental growth factor, endoglin, and vascular endothelial growth factor, all of which increase 4 - 6 weeks prior to the onset of the disease. The protective role of heme oxygenase 1 and its metabolite, carbon monoxide, during pregnancy has been demonstrated by recent data, pointing to this as a possible target for pre - eclampsia treatment. (25)

Clinical Presentation and Work up findings: -

The goal of clinical and laboratory testing is to identify pre - eclampsia and assess its severity. Brain edema is associated with headaches, tinnitus, phosphene signals, visual disorders, brisk tendon reflexes, and vigilance disorders; acute renal failure is associated with oliguria; placental abruption is associated with uterine contraction and vaginal bleeding; **HELLP** syndrome is associated with vomiting; subcapsular hepatic hematoma is associated with band - like epigastric pain; and cardiac failure is associated with dyspnea. Eclampsia, the primary neurological consequence of pre - eclampsia, is characterized by a convulsive episode or any other altered consciousness manifestation that appears in the context of pre - eclampsia and cannot be linked to a neurological condition that was present earlier.

Using an appropriate cuff, take the patient's resting blood pressure and screen for weight gain, edema (including signs of acute cerebral and pulmonary edema), cardiomyopathy, and acute renal failure. The best method for evaluating the fetus is electrocardiogram. A complete blood count that includes platelets, haptoglobin, and lactate dehydrogenase; a blood smear to check for schistocytes; an assessment of electrolytes, urea, and creatinine to check for acute renal failure or uremia; a 24 - hour proteinuria; prothrombin, activated thrombin time, and fibrinogen (microangiopathic hemolytic anemia); blood group; and irregular antibody screening are among the laboratory tests that are performed. Fetal ultrasonography with Doppler velocimetry of the umbilical, cerebral, and uterine arteries, fetal weight estimation, Manning score assessment of fetal well - being, and placenta examination are among the other tests performed. (26)

While there are differences in the definition of severe pre - eclampsia, the following elements are commonly agreed upon:

- Maternal neurological disorders such as eclampsia, acute pulmonary edema, proteinuria greater than **5 g/day**, oliguria less than **500 cc/day**, creatinine greater than **120 µmol/L**, **HELLP syndrome**, thrombocytopenia less than **100, 000/mm3** and,
- Fetal criteria, especially intrauterine growth retardation, oligohydramnios, or fetal death in utero, are among the conditions that must be met. Diastolic blood pressure greater than **90 mmHg**, measured twice at least **6 hours apart**, along with proteinuria (two or more protein spots on the dipstick, more than **300 mg** of total protein in a **24 - hour** urine collection, or a higher protein creatinine ratio than **30 mg/mmol**) are considered to be signs of mild pre - eclampsia.

Emergency and its Management: -

The only effective treatment for pre - eclampsia is delivery. An obstetrician, an anesthetist, and a pediatrician are involved in the multidisciplinary management process. Subspecialists in nephrology, hypertension, and maternal fetal medicine may need to be consulted in certain situations. The risks to the mother of continuing the pregnancy must be weighed against the fetal risks of an induced preterm birth. The criteria for delivery are determined by two frequently linked factors: the severity of pre - eclampsia and the gestational age at diagnosis (estimated fetal weight). Treatment for severe pre - eclampsia should have two goals: it should stop the negative effects of high maternal blood pressure and stop eclampsia from happening. The first step in treating severe pre - eclampsia is to transport the mother to a maternity ward that can adequately care for her and her child in an ambulance or helicopter that is outfitted with all the necessary equipment. Clinical, cardiotocographic, laboratory, and ultrasonography testing are necessary to assess the severity of pre - eclampsia and adjust the course of treatment at admission and every day after that. When pre - eclampsia is detected after 36–37 weeks of pregnancy, there is no benefit to extending the pregnancy, regardless of the severity of pre - eclampsia. (27–29) Given the high risk of complications for the mother and the poor prognosis for the newborn, expectant management is also not justified for severe pre - eclampsia before **24 weeks**. The parents must then be informed about the possibility of a medical pregnancy termination by the obstetric team. In cases of mild pre - eclampsia, extending the pregnancy can be discussed and reevaluated on a regular basis. The degree of pre - eclampsia determines how the patient is managed at **34–37 weeks**. For moderate pre - eclampsia, expectant management can reduce the risk of an induced preterm delivery; however, because of the increased risk of complications for both the mother and the fetus, delivery is still the rule for severe pre - eclampsia.

Likewise, at **24–34 weeks**, the degree of pre - eclampsia determines how the patient is managed. Acute pulmonary edema, abruptio placentae, subcapsular hepatic hematoma, eclampsia, uncontrolled severe hypertension (not responsive to dual therapy), or thrombocytopenia, **50, 000/mm3**, are among the signs that indicate the need for an emergency delivery. If any of the following criteria are met, delivery following corticosteroid therapy for pulmonary maturation is required: de novo creatinine greater than **120 micro - mol/L**; oliguria below **20 mL/hour**; progressive **HELLP syndrome**; prolonged or severe variable decelerations with short - term variability less than **3 milliseconds**; persistent epigastric pain; signs of imminent eclampsia (headaches or persistent visual disorders); progressive **HELLP syndrome**. Cervical ripening can induce labor when an emergency delivery is not needed. (30).

Because the only known benefit of antihypertensive treatment is to reduce the risk of maternal complications (cerebral hemorrhage, eclampsia, or acute pulmonary edema), it is only helpful in cases of severe pre - eclampsia. (31) When it comes to antihypertensive therapy for pre - eclampsia, there is no worldwide agreement. The four medications that are approved to treat hypertension in cases of severe pre - eclampsia are Dihydralazine, clonidine, labetalol, and nicorandil. (31)

The fetus is harmed by an overly aggressive blood pressure reduction, and there is no optimal target blood pressure value. (32) When appropriate, combination therapy should be administered after therapy with a single agent as the first line of treatment. Gestational age must be taken into consideration when using corticosteroids to induce pulmonary maturation. When administered in **2 doses of 12 mg separated by 24 hours,** betamethasone is still the gold standard because it lowers the risk of intraventricular hemorrhage, hyaline membrane disease, and neonatal mortality. (33)

For severe pre - eclampsia, magnesium sulfate (**MgSO4**) might be included in the therapeutic arsenal. It can be used to treat eclamptic convulsions and prevent eclampsia in the future, taking the place of medications like diazepam, phenytoin, or the combination of promethazine, pethidine, and chlorpromazine. (34) It is well known that **MgSO4** is effective in lowering the complications of eclampsia in mothers and newborns. It is injected intravenously. The loading dose is **4 g over 15–20 minutes.** If convulsions recur, the dose can be repeated at **2 g**. After that, it is given at a maintenance dose of **1 g/hour for 24 hours. MgSO4** treatment must be thoroughly kept in check in the intensive care unit because organ failure may occur. This monitoring is based on repeated checking for a **Glasgow score of 15**, tendon reflexes, respiratory frequency more than **12 per minute**, and diuresis greater than **30 mL/hour**. Any manifestation of overdose requires stopping the infusion, considering injection of calcium gluconate, and measuring blood magnesium levels. Eclampsia is typically regarded as a sign that a cesarean section is necessary in an emergency. Even so, it is uncommon to decide to postpone a cesarean section if the mother's condition is stable and comforting following treatment and can be justified by the fetal status. (35)

Management after delivery

The risk of complications lasts for a while after delivery, even though delivery is the only effective treatment for pre - eclampsia and even though clinical symptoms and laboratory abnormalities typically go away in the hours that follow. Pre - eclampsia is linked to mortality and long - term morbidity. In long - term follow - up, **20%** of women with pre - eclampsia experience hypertension or microalbuminuria. Additionally, compared to age - matched controls, the risk of developing subsequent cardiovascular and cerebrovascular disease is doubled in women with pre - eclampsia and gestational hypertension. According to a recent prospective epidemiological study, pre - eclampsia is associated with a higher risk of dying from cardiovascular disease, with a median follow - up period of **30 years.**

Patients with severe preeclampsia require postpartum hemodynamic, neurological, and laboratory monitoring. (36) Frequent blood pressure readings to enable antihypertensive medication adjustments are part of hemodynamic monitoring, as is frequent weight and diuresis monitoring based on intake (oliguria should prompt progressive fluid resuscitation and occasionally the use of diuretics). In neurological monitoring, symptoms such as headaches, phosphene signals, tinnitus, and fast tendon reflexes are monitored for indications of impending eclampsia. During the first week following delivery, when complications are thought to be most likely, clinical monitoring needs to be performed multiple times per day. An intensive care unit can be used for monitoring if necessary.

For the first **72 hours** following delivery, laboratory monitoring should be performed multiple times per day. After that, it should be adjusted based on the indices' advancement. A full blood count, tests for liver function, and an assessment of lactate dehydrogenase (**LDH**) must be part of it. If the patient is to continue receiving treatment for hypertension after discharge, regular monitoring by the patient's general practitioner will be required until all clinical and laboratory indices have returned to normal. Discharge from the hospital cannot be considered until this time.

Anti - hypertensive treatment algorithm for Pre - eclampsia

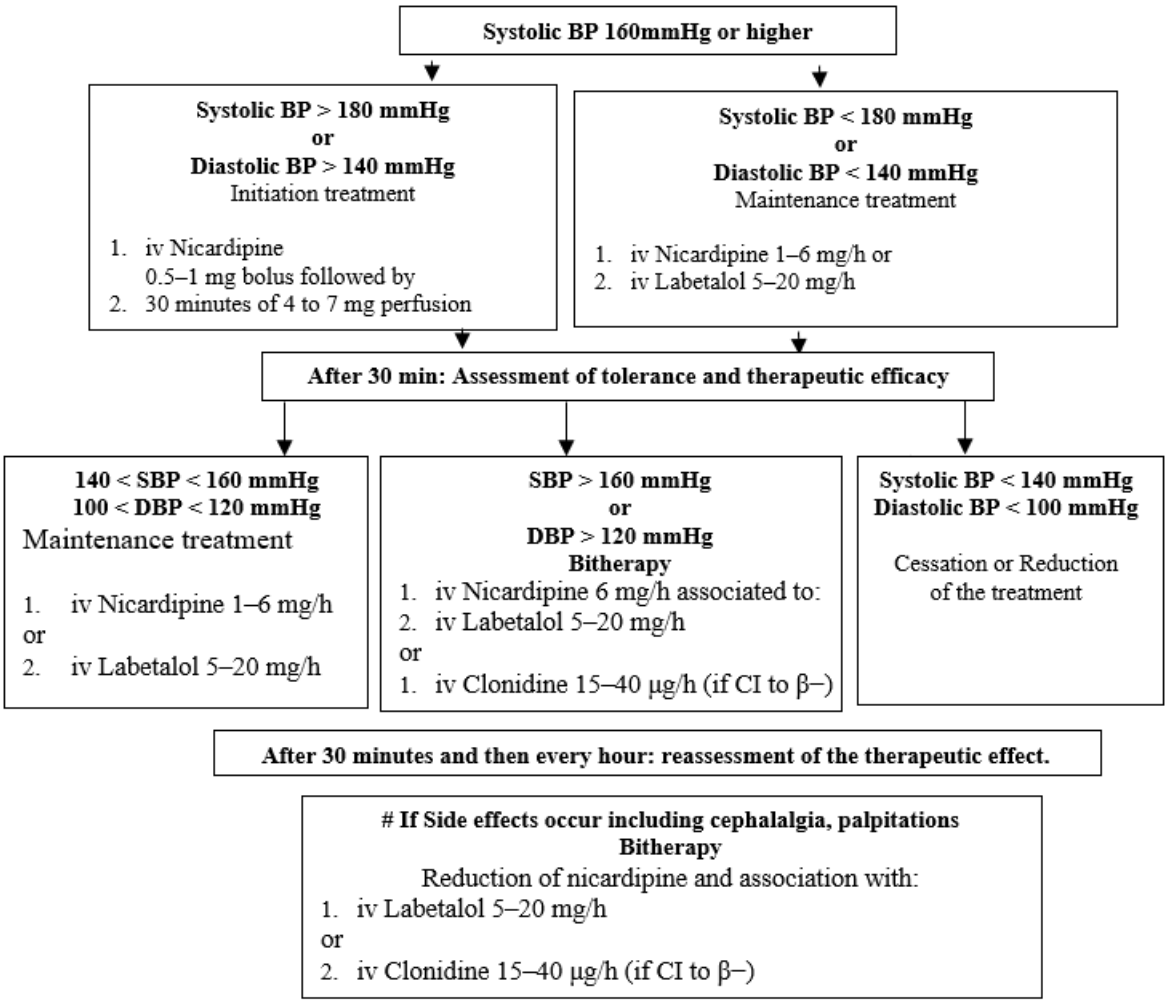

Figure 1: Anti - hypertensive treatment algorithm for Pre - eclampsia

It is necessary to take into account the possibility of pre - eclampsia reoccurring during a subsequent pregnancy. According to estimates, the risk is less than **10%** in all cases of pre - eclampsia (37) but increases if the condition is detected before **28 weeks**. When pre - eclampsia manifests at **20–33 weeks**, the relative risk is **15, at 33–36 weeks**, and at **37 weeks, it is 10**. (37)

The patient's primary care physician may request screening for underlying renal or hypertensive disease three months after delivery. This type of screening is meant to look for the return of normal blood pressure readings and the elimination of proteinuria; if abnormalities are still present, a nephrologist or hypertension specialist should be consulted to identify the cause. Pre - eclampsia may conceal systemic, renal, or thrombophilia diseases that were previously undetected, which is why this examination is crucial. A urine dipstick test, blood pressure monitoring, a clinical examination searching for indications of autoimmune diseases, and a predetermined set of questions should all be part of it. Antiphospholipid antibody testing is advised following severe pre - eclampsia.

In cases of early pre - eclampsia, pre - eclampsia with any intrauterine growth retardation, abruptio placentae, or in utero death, as well as antithrombin III, protein C and S assays, and a test for resistance to activated protein C, it is recommended to search for hereditary thrombophilia. (38) Only if kidney failure continues at three months after delivery, or if signs of a systemic underlying condition or proteinuria continue at six months, should a percutaneous needle biopsy of the kidney be carried out.

Individuals with cardiovascular risk factors who are not pregnant may have predispositions similar to those who have experienced severe pre - eclampsia. (39). Thus, following severe pre - eclampsia, long - term monitoring of cardiovascular, renal, and metabolic risk factors is advised.

2. Prevention

The identification of modifiable risk factors is the foundation for primary prevention of pre - eclampsia. There is a wealth of information in the literature about pre - eclampsia risk factors, but it should be interpreted cautiously. Women who have experienced severe pre - eclampsia in the past are considered high risk, whereas women who have never experienced pre - eclampsia but have at least one risk factor been considered low risk.

There are numerous risk factors, including genetic risk factors, family history of pre - eclampsia, immunologic factors, nulliparity, a new partner, and demographic factors such as a maternal age more than **35 years,** the woman's own gestational age and birth weight (with elevated risks for women born before 34 weeks or weighing less than **2500 gms at birth**), factors related to the pregnancy, such as multiple pregnancy, congenital or chromosomal anomalies, a

hydatidiform mole, or urinary infection, risk factors associated with maternal disease, including chronic hypertension, kidney disease, obesity, insulin resistance, and diabetes, as well as thrombophilia, and environmental factors such as living at a high altitude and stress.

While looking for these risk factors is crucial, they might not be able to accurately predict pre - eclampsia on their own. Predicting pre - eclampsia accurately, however, would allow for early and best management of high - risk women. Currently being evaluated are a number of predictive tests. These include clinical tests, which are less sensitive and specific than others. Examples of these include blood pressure monitoring during the second trimester or **24 - hour ambulatory blood pressure monitoring.**

Assays for uric acid, urinary kallikrein, and fibronectin are among the laboratory tests for oxidative response that have been evaluated; however, thus far, no proof of their significance has been discovered. (40)

Alpha fetoprotein, beta human chorionic gonadotropin, and unconjugated estriol are the markers used to screen for **trisomy 21** during the second trimester. Among these markers, elevated alpha fetoprotein is linked to an increased risk of pre - eclampsia (unless there are neural tube abnormalities, as when beta human chorionic gonadotropin is elevated). Women with elevated levels should be monitored frequently; however, because of the low negative predictive value of these tests, they might not be used for screening. Activin, pregnancy - associated plasma protein A, inhibin A, corticotropin - releasing hormone, and other 40 serum markers for **trisomy 21** in the first trimester have been examined, but it appears that their likelihood ratios are not high enough.

Imaging tests have been evaluated, including uterine artery Doppler ultrasound. (41, 42) Intestinal artery Because the likelihood ratios in low - risk populations are too variable to predict more than one - third of pre - eclampsia cases, doppler ultrasound is not recommended in the first or second trimester in these populations. (43)

Uterine artery Doppler can be performed during the second trimester morphologic ultrasound examination in a high - risk population, the precise definition of which is often ill - defined. If abnormal results (**Resistance index more than 0.58 or 90–95th percentile, unilateral or bilateral notch)** are found, the examination can be repeated a month later. The risk of pre - eclampsia may be predicted as early as the first trimester by combining a uterine artery Doppler examination with a three - dimensional ultrasound to measure placental volume. (44)

In clinical practice, testing multiple markers is currently the trend because no single marker can accurately predict the risk of pre - eclampsia. During the first or second trimester, the most widely used set of markers evaluates sFlt - 1, placental growth factor, endoglin, and vascular endothelial growth factor. Pre - eclampsia risk is markedly elevated in the first trimester when elevated vascular endothelial growth factor and endoglin levels are paired with elevated sFlt - 1 and decreased placental growth factor.

Serum markers and Doppler indices have been shown to improve pre - eclampsia prediction. C - reactive protein, uterine artery mean resistance index, and maternal serum cystatin C in the second trimester were found to be independent predictors of pre - eclampsia in a recent nested case - control study. (45)

Antiplatelet aspirin therapy, the cornerstone of secondary prevention, lowers the risk of pre - eclampsia in women with at least one risk factor by **10%**. (46) As of right now, no study makes it possible to determine the precise dosage or the ideal time to start taking aspirin. Aspirin should, however, be started as soon as possible—that is, prior to the onset of the first phase of trophoblast invasion, which occurs at **12 to 14 weeks.** Aspirin's effectiveness has only been demonstrated in women who did not have thrombophilia and who had prior pre - eclampsia linked to intrauterine growth retardation. Only in cases of complicated thrombophilia (history of thromboembolic complications or pre - eclampsia) is low molecular weight heparin indicated. (47) Preventing pre - eclampsia in women whose daily calcium intake is **600 mg is advised to start 1.5 g/day of calcium supplementation at week 15 and continue it for the duration of the pregnancy. (**48)

Statins can reduce early - onset pre - eclampsia by increasing HO - 1 expression and preventing sFlt - 1 release. Other treatments such as oligo - elements, vitamin C and E antioxidant therapy, and nitric oxide have not been proven to work.

3. Conclusion

Pre - eclampsia is a rare pregnancy - related condition with an unpredictable course that can have serious consequences for both the mother and the fetus. Delivery is the simple form of therapy. Before having an induced preterm delivery, a comprehensive risk - benefit analysis of the mother and the fetus must be completed. Determining the delivery criteria is therefore essential to the best management in the event of pre - eclampsia. The primary goal of current research is to predict the onset of pre - eclampsia, including severe pre - eclampsia, in order to improve the morbidity and mortality associated with this disease and allow for early management. It is also necessary to develop specific secondary prevention tools for recurrent pre - eclampsia.

Disclosure:
The authors report no conflicts of interest in this work.

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