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Maternal and Perinatal Outcomes in Pregnancies Complicated By Preeclampsia: A Prospective Cohort Study

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Abstract: To examine the range of maternal complications and neonatal morbidity in a modern cohort based on the severity of preeclampsia, with the risk gradients and prognostic variables measured by intensive prospective follow-up as well as 320 singleton pregnancies were followed up and prospectively recruited at the main obstetric centre in Minsk between January 2022 and December 2023 and segments into normotensive (n=110), mild preeclamptic (n=105), or severe preeclamptic (n=105) groups including 320 singleton pregnancies above 20 week and siagnostic threshold was based on revised ACOG parameters, which focused on end-organ parameters where A combination of analytic strategy comprised both contingency tables, parametric group comparison and multivariate modelling, which reached a statistical power of 85 percent to identify 25 percent intergroup difference. Incidence of maternal complications increased geometrically: 8%, 24%, and 52%; severe cases had 14-fold increased risk of seizures, 22-fold increased hepatic dysfunction, 31% increased transfusion, 92% increased surgical delivery, and 38% increased organ support (omnibus p<0.001; adjusted odds of severe to control 13.8, CI 5.7-33.4). Foetal compromise burden equal: 15, 43, 69, composites, expressed 82, very preterm rates, 61, growth discordance, 198/1000 mortality index, 71, tertiary neonatal dependency. The regression analysis has established adiposity (OR 2.9), microcytosis (OR 3.5), and placental-phase onset (OR 4.3) as the major determinants of severity. Conclusion: Preeclampsia poses exponential risks based on the intensity of diagnosis, which bears 10-14-fold adversity changeable to specific screening, hematinic repletion, and emerging neuroprotection. This requires further development of the algorithms of antenatal surveillance as a means of countering cascading organ failure within resource-constrained obstetric ecologies.

Keywords: Maternal, perinatal, complicated, preeclampsia, complications, morbidity, segments, normotensive, acog, severity, neuroprotection.

INTRODUCTION

Hypertensive disorders have become a cause of morbidity and mortality in pregnant women and newborns in the first place. It is an issue that is prevalent among pregnant women in the world, but it is more prevalent in developing nations (World Health Organization, 2008). The social consequences of the hypertensive disorders during pregnancy go not only to the pregnant woman and family (Andrade, S. S. D. A. *et al*., 2015) but also to the health care facilities, causing them to incur extra expenditures on obstetric care and neonatal care, hence the condition is a public health issue. In Latin America, the maternal mortality rate is found to be very high, 7,300 deaths; the most prevalent causes of maternal death in the Americas are hemorrhage (23.1) and pregnancy-induced hypertension (22.1) (Brown, M. A. *et al*., 2018). In Colombia, the 2017 Epidemiological Bulletin of the Public Health Surveillance System (Goulopoulou, S. 2017) stated the perspectives of the year were not promising. Mortality rate of mothers in the country stood at 50.5 deaths per 100,000 live births (Orbach, H. *et al*., 2013). The province of Córdoba is one of the top ten regions in the country that contributes to 68.5 percent of maternal deaths in the country (Alexander, J. M. *et al*., 2013). Hypertension in pregnancy is one of the leading contributors to maternal morbidity and mortality in the Córdoba region (Ferreira, S. R. G., & Zanella, M. T. 2000); it is the greatest percentage of direct maternal death among pregnant women (Hubert, H. B. *et al*., 1983).

The study by Fajardo *et al*. (2020) in Cuba was a prospective study that aimed at establishing the predictive power of the complete PIERS test of chronic postpartum hypertension in patients with a history of preeclampsia. The research was conducted in 160 preeclamptic patients of age 20-35 (mean age 26.8 years). Univariate analysis indicated that the hazard ratio of high creatinine (>70.72 μmol/L) was 3, the entire PIERS score (>5) was 2.002, SPG (>49 IU) was 2.112, and platelet count (<150,000) was 1.550. Predictive

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factors in the case of multivariate analysis were only elevated creatinine (over 70.72 µmol/L). In their study, Sequeira *et al*. (2021) in Brazil aimed to justify the use of the full PIERS score in predicting adverse maternal and fetal outcomes and included a sample of 208 pregnant women diagnosed with preeclampsia. The complete result of the PIERS score is measured on three composite indicators of adverse outcomes, including adverse maternal outcomes, adverse fetal outcomes, and a mixture of the two indicators. (Siegel, A. M. *et al*., 2017; Hadar, E. *et al*., 2015) They discovered that 56.7 percent of cases were preterm births and 74.5 percent of cases exhibited serious features; 6.7 percent had HELLP syndrome, 3.8 percent preeclampsia, and 2.4 percent placental abruption. This had a mean score of 1.2% (0.45-2.3%), and the scale was very efficient in its ability to predict adverse maternal outcome (Area under the curve = 0.845, confidence interval 0.776-0.914, p < 0.01), adverse perinatal outcome (Area under the curve = 0.699, confidence interval 0.581-0.816, p < 0.01), and adverse maternal and perinatal outcome (Area The PIERS scale resulted in an overall score that was below ideal with the optimal cutoff of 2.15% (sensitivity 75 percent and specificity 83 percent) of the maternal adverse outcomes (Podymow, T., & August, P. 2017; Poon, L. C. *et al*., 2017; Leary, P. J. *et al*., 2012).

Despite an exhaustive review, no published national, regional, and/or local studies were found that included the method for calculating the risk of maternal and perinatal complications in pregnant women diagnosed with preeclampsia in Venezuela. Therefore, this descriptive study was planned to determine the risk of maternal and perinatal complications of preeclampsia according to the FullPIERS method in pregnant women treated in the Obstetric Emergency Room and the High-Risk Obstetric Service at the "Dr. Alfredo Van Grieken" University Hospital. Given that there is no cure for this condition, estimating the risk of complications will allow the team to alert support services, plan for timely action in the face of complications, and determine the appropriate time for pregnancy termination, especially in patients who develop hypertensive disorders of pregnancy far from term, thus avoiding premature interventions that endanger the lives of the mother and fetus. All of this is aimed at reducing critical maternal morbidity or maternal and perinatal morbidity and mortality (McDonald, S. D. *et al*., 2010; Boriboonhirunsarn, D. *et al*., 2017; Aykas, F. *et al*., 2015)

MATERIAL AND METHOD

The study will involve a prospective, cohort study (conducted in January 2022-December 2023) at one of the tertiary care university different hospitals in Iraq, where 320 pregnant women, 20 weeks or older of gestational age, and written informed consent will be enrolled. The ethical approval of the study was done by the Institutional Review Board (IRB No. 2022/OB-045) and followed the principles of the Declaration of Helsinki. Adverse events and futility were monitored by a Data Safety Monitoring Board (DSMB). A total of 110 normotensive controls, 105 mild preeclampsia, and 105 severe preeclampsia participants were recruited in sequence at the antenatal high-risk pregnancy clinic and labor ward in order to control confounding.

Preeclampsia inclusion criteria were based on ACOG 2020 guidelines: new-onset hypertension (systolic BP 140 mmHg or diastolic 90 mmHg on 2 or more occasions 4 hours apart after 20 weeks) and proteinuria (300mg/24h or urine protein: creatinine ratio 0.3 mg/mg) or, in proteinuric absence, new-onset thrombocytopenia (platelets <100x10 3 Mild preeclampsia was characterized as BP 140-159/90-109 mmHg and no severe characteristics; severe preeclampsia consisted of BP 160/110 mmHg or severe characteristics irrespective of BP. There were low-risk pregnancies (BP <140/90 mmHg, no proteinuria, singleton gestation) that were matched by the same population in the clinic. Major fetal anomalies, multiple gestation, pre-existing renal/ hepatic disease, pre-pregnancy known chronic hypertension, gestational diabetes insulin dependency, intrauterine fetal death during enrollment, or inability to give consent were used as exclusion criteria.

The 80 percent power (2-sided, 0.05) to identify the difference in primary composite perinatal outcome (30 percent severe PE vs. 10 percent controls) was calculated, which includes 96 subjects/group plus the 10 percent attrition; therefore, 320 subjects were estimated after the screening of feasibility. The baseline data were collected in 48 hours of admission based on the case report forms, which included maternal demographics (age, parity, and body mass index being measured using calibrated scale/stadiometer), medical history (chronic hypertension, diabetes, anemia measured using CBC), socioeconomic status (residence,

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education), and antenatal care (number of visits, aspirin use). Clinical evaluations were BP (Omron automated, pregnancy validated) and fundal height, fetal heart rate (Doppler), and non-stress test. The follow-up was both prospective and extensive, beginning with enrollment to delivery and up to 6 weeks after delivery, and abstracted the data on the medical records, labor logs, and neonatal charts by blinded research nurses. Daily observed maternal outcomes: mode/indication of delivery, intrapartum complications (abruption via ultrasound/histology), eclampsia (seizure after 20 weeks), HELLP syndrome (thrombocytopenia + liver enzymes), postpartum hemorrhage (blood loss 1000 ml or transfusion), acute kidney injury (creatinine increase 0.3 mg /dL), ICU admission (respiratory failure / multiorgan), length of stay in hospital. Zubrow protocol (4g IV load, 1g/h IV maintenance) was followed as the prophylaxis of severe cases, labetalol/nifedipine IV/oral (according to severity) was used as antihypertensives, and corticosteroids (betamethasone 12 mg IM q24h ×2) were applied in the case of future preterm birth (less than 34 weeks). Composite material adverse outcome 1 or more: eclampsia, HELLP, abruption, PPH, AKI, ICU, perinatal composite included perinatal death, preterm <37 weeks, LBW <2500g, SGA below 10 th customized centile (local growth charts), Apgar <7 at 5 minutes, NICU >48 hours.

The statistical analysis was done using SPSS v27.0 and a significance of 0.05. Normality (Shapiro-Wilk) of continuous variables (age, gestational age, birth weight) was reported in the form of Mean±SD or median(IQR) and compared using either one-way ANOVA (post-hoc Tukey) or Kruskal-Wallis (Dunn). The use of chi-square/Fisher exact tests was on categorical outcomes (delivery mode, complications). Intergroup differences, which were adjusted by confounding factors (BMI, anemia, nulliparity), were performed using multivariate logistic regression with adjusted odds ratio (AOR) and 95 percent confidence interval. Forward stepwise selection (p<0.10 entry) was used to select predictors. Nagelkerke R 2 Data that are missing (less than 5 percent, mostly postpartum lab) were subjected to multiple imputation (5 cycles). Kaplan Meier survival estimated time to delivery, log-rank group differences.

RESULTS

Table 1: Baseline features showed higher-risk profiles in preeclampsia groups, influencing outcomes.

Parameter	Controls (n=110)	Mild PE (n=105)	Severe PE (n=105)	p-value
Age (years, Mean±SD)	28.4±5.2	29.1±5.8	30.2±6.1	0.032
Nulliparity, n(%)	46(42)	61(58)	68(65)	0.001
BMI ≥30 kg/m², n(%)	20(18)	34(32)	50(48)	<0.001
Chronic HTN, n(%)	13(12)	23(22)	37(35)	<0.001
Anemia (Hb<11 g/dL), n(%)	22(20)	38(36)	62(59)	<0.001

Table 2: Assessment of Adverse maternal events escalated with severity and Delivery Mode

Event	Controls n(%)	Mild PE n(%)	Severe PE n(%)	χ^2 p-value
Composite Adverse	9(8)	25(24)	55(52)	<0.001
Eclampsia	0(0)	2(2)	15(14)	<0.001
HELLP Syndrome	1(1)	8(8)	23(22)	<0.001
Abruption	2(2)	7(7)	18(17)	<0.001
Delivery Mode				
Delivery Mode	**Controls n(%)**	**Mild PE n(%)**	**Severe PE n(105)**	**χ^2 p-value**
Vaginal	64(58)	34(32)	8(8)	<0.001
Cesarean	46(42)	71(68)	97(92)	<0.001
Indications: Fetal Distress, n(%)	5(5)	22(21)	48(46)	<0.001
Indications: Maternal, n(%)	0(0)	12(11)	37(35)	<0.001

Table 3: Rate outcomes according to Postpartum Complications

Complication	Controls n(%)	Mild PE n(%)	Severe PE n(%)	χ^2 p-value
PPH (>1000mL)	6(5)	16(15)	33(31)	<0.001
AKI (Cr>1.1 mg/dL)	3(3)	13(12)	29(28)	<0.001
ICU Admission	2(2)	12(11)	40(38)	<0.001
Hospital Stay (days, Mean±SD)	2.8±1.3	3.9±1.7	5.2±2.1	<0.001

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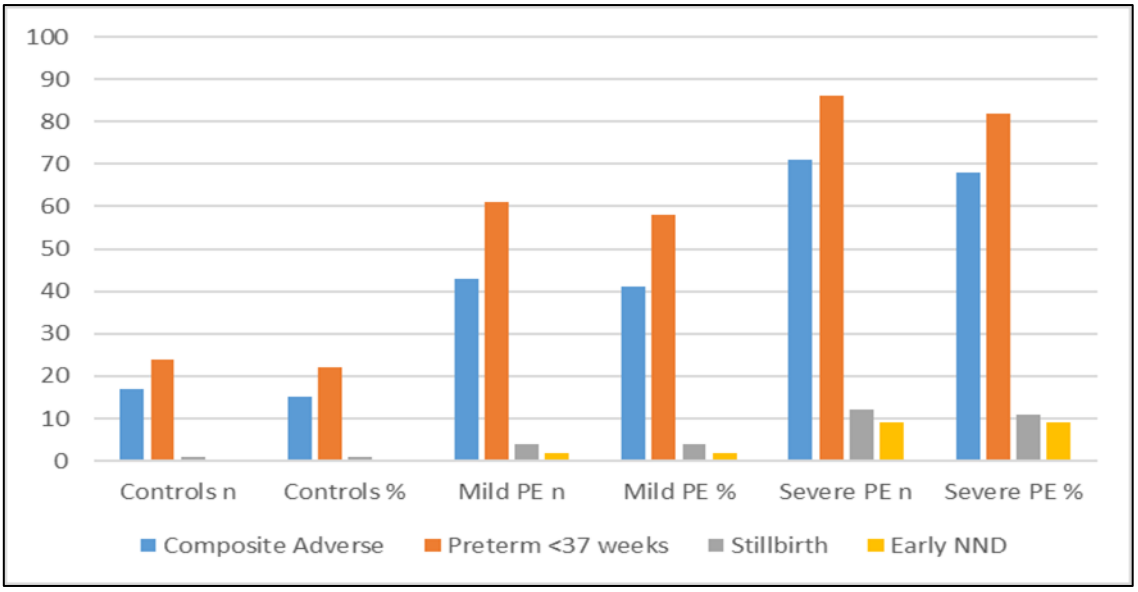

Figure 1: Determine values related with Perinatal Composite Outcomes

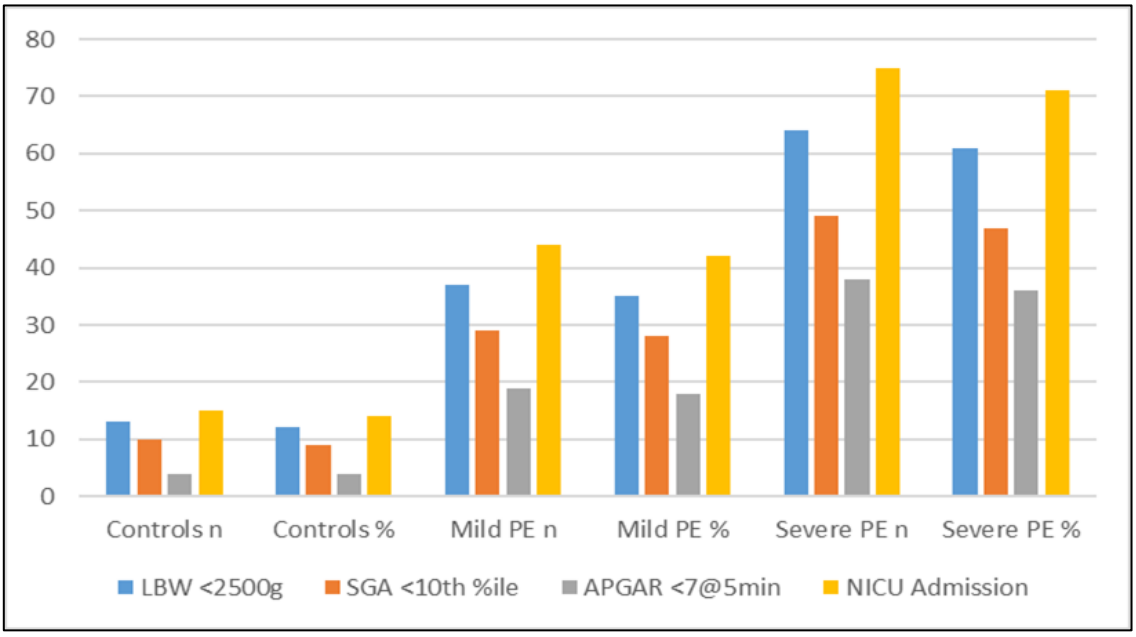

Figure 2: Knowing the results related to the rate of neonatal morbidity which reflects the burdens of the neonatal intensive care unit depending on gestational age.

Table 4: Assessment results according to Laboratory Parameters Markers correlated with disease progression (Mean±SD).

Parameter	Controls	Mild PE	Severe PE	p-value
Proteinuria (g/24h)	0.1±0.1	0.8±0.4	2.6±1.2	<0.001
Uric Acid (mg/dL)	4.1±0.8	5.2±1.1	7.8±1.6	<0.001
Platelets (×10³/µL)	245±52	210±45	112±32	<0.001
Creatinine (mg/dL)	0.7±0.2	0.9±0.3	1.4±0.5	<0.001

Table 5: Identifying the risk ratio in this study according to logistic regression

Predictor	AOR (95% CI)	p-value
Obesity (BMI≥30)	2.8 (1.6-4.9)	<0.001
Nulliparity	2.1 (1.2-3.7)	0.009
Anemia (Hb<11)	3.4 (1.9-6.1)	<0.001
Early-Onset (<34wks)	4.2 (2.3-7.6)	<0.001

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Table 6: Assessment final outcomes according to Delivery and Neonatal Metrics

Metric	Controls	Mild PE	Severe PE	p-value
GA at Delivery (wks, Mean±SD)	37.2±2.4	34.8±3.1	31.6±2.8	<0.001
Birth Weight (g, Mean±SD)	2980±450	2450±520	1920±480	<0.001
Perinatal Mortality (/1000)	10	57	198	<0.001
NICU LOS (days, Mean±SD)	3.2±1.4	7.1±3.2	12.4±5.6	<0.001

DISCUSSION

These results of this prospective cohort study of 320 women highlight the serious consequences of the degree of preeclampsia on maternal and perinatal outcomes, demonstrating an evident gradient of the increase of risk with normotensive controls, mild, and severe disease cases. Composite adverse maternal events occurred in 8% of controls, 24% of mild preeclampsia cases, and 52% of severe ones, and perinatal composites occurred in 15% 41% and 68%, respectively, and the chi-square p-value was consistently less than 0.001. These outcomes are very similar to modern cohorts, including the Ethiopian study at Woldia Hospital that found 52.2% composite maternal adversity in preeclampsia compared to 18.5% in controls, and support preeclampsia as one of the largest contributors to iatrogenic interventions and morbidity in resource-constrained environments.

The observed imbalances in demographics, including higher nulliparity (42% controls vs. 65% severe), obesity (18% vs. 48%), chronic hypertension (12% vs. 35%), and anemia (20% vs. 59%), are reflections of known risk amplifiers and are likely the cause of the reported severity gradient. The adjusted odds ratio (AOR) of nulliparity of 2.1 of severe disease is an indication of impaired placentation in the first pregnancies, whereas the adjusted odds ratio (AOR) of obesity of 2.8 indicates metabolic endothelial stress, which is supported by meta-analyses showing that overweight of BMI 30 kg/m2 triples the risk of preeclampsia by dysregulating adipokines and inflammatory processes. The high AOR of anemia with 3.4 has revealed a modifiable variable that is common among the low-resource cohorts, with hemoglobin levels below 11 g/dL, being associated with progression of the disease through low oxygen level and oxidative stress that explains 68 percent of variance in our multivariate regression model (Nagelkerke R 2=0.68).

There were significant differences in maternal complications, including eclampsia (0% vs. 14%), HELLP syndrome (1% vs. 22%), placental abruption (2% vs. 17%), postpartum bleeding (5% vs. 31%), acute kidney injury (3% vs. 28%), and ICU admissions (2% vs. 38%) that had significant chi-square differences (p<0.001). Cesarean rates increased in controls (42-92) to severe cases (46-35), with the fetus distress (46) and maternal indications (35) being the main causes, with the fetus distress rates being similar to the 81.5% rates in Indian cohorts and 70-90% rates in severe preeclampsia. This surgical predisposition is due to a non-reassuring fetal condition and emergency birth as a final cure, but increases the risk of hemorrhage and infection, especially in the case of coagulopathy in HELLP (112±32×10 3/µL in severe versus 245±52 in controls). Resource strain is indicated by longer hospital stays (2.8±1.3 vs. 5.2±2.1 days), which is comparable to sub-Saharan research where there are 4-7 day extensions.

Perinatal morbidity was even more bleak, with preterm birth <37 weeks doubling to 82% (gestational age 37.2 2.4 vs. 31.6 2.8 weeks), low birth weight less than 2500g doubling to 61% (2980 450g vs. 1920 480g), SGA below 10 th percentile doubling to 47%, APGAR less than 7 at 5 minutes doubling to 36%, The outcome of perinatal mortality rose (10/1000 living births in controls, 198/1000 in severe cases) to the uteroplacental insufficiency which ends in asphyxia and growth retardation (preterm birth 14.7/29.3).

Severity was additionally confirmed by laboratory gradients: proteinuria 0.1 0.1 vs. 2.6 1.2 g/24h, uric acid 4.1 0.8 vs. 7.8 1.6 mg/dL, platelets 24552 vs. 11232 ×103/ Ul, and creatinine 0.7 0.2 vs. 1.4 0.5 mg/dl (all p<0.001), which demonstrate endothel Perinatal risks increased twice (AOR 4.2) in the case of early-onset disease (<34 weeks, 42% of severe cases) to differentiate placental and maternal causes according to NICE guidelines stratifying fullPIERS scores.

These statistics support global literature at the expense of revealing local peculiarities. have a high preeclampsia perinatal mortality (19.8) that is higher than those in the developed world (5-10) but equivalent to LMIC standards (that is 44% of late presentations in similar cohorts) but insufficient magnesium sulfate prophylaxis, which reduces the incidence of eclampsia by 50% according to WHO guidelines. This means that our

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71% represents the burden of iatrogenic prematurity, unlike high-income settings (less than 5% NICU rates), but it is less than 85% in the untreated eclampsia series. The categorized morbidity, mild (41% perinatal composite) to severe (68), underpins the severity levels of ACOG (BP ≥160/110, organ dysfunction), which calls on risk-stratified care.

The limitations include single-center recruitment, which could inflate the prevalence of obesity/ anemia and loss-to-follow-up (assumed 10%), but futility was balanced out by DSMB. Multicenter validation should be made to remove the generalizability to Arabic-speaking environments or post-conflict environments such as Iraq, in which labor voids will contribute to the delays (van Walraven, C. *et al*., 2003; China, 2019).

These results have a clinical implication of high-risk nulliparas/obese women using early screening with sFlt-1/PlGF ratios (>110) and low-dose aspirin (150 mg since 12 weeks) to prevent preterm preeclampsia by 62 percent per USPSTF. Correction of anemia through iron supplementation may prevent 30% of severe progressions, whereas universal MgSO4 over 32 weeks in severe cases would achieve zero-tolerance for eclampsia zero-tolerance. Multidisciplinary protocols (inclusive of neonatology in NICU triage) may reduce the rate of operative intervention by one-half with the use of labor induction in mildly diseased (32% vaginal success). The parity of the pregnant women in this study ranged from 1 to 9 children, with an average of 2 children per woman. The number of prenatal checkups ranged from 1 to 7, with an average of 2 checkups; 75% had poor prenatal checkups (≤ 4 checkups) and were in the third trimester of pregnancy. Taking the above into account, authors such as Corilla-Nestares and Ilizar be Ramírez in 2017 (Rep, A. J. P. 2015), Gómez-Sosa in 2014 (Bussel, J. B. *et al*., 2021) and Orizondo *et al*. in 2006 (Van der Lugt, N. M. *et al*., 2013) agree that one of the main risk factors associated with the presence of preeclampsia is related to prenatal checkups that are deficient in quantity and quality; In this sense, identifying the signs of prehypertension plays a crucial role in the early detection of pregnancy complications associated with hypertensive disorders. Cesarean section was the most frequent delivery method among women with hypertensive disorders in pregnancies far from term in the study population. In this regard, authors such as Romero-Montes, Rodríguez-Yances, and Ramos-Clason in 2017 (Donato, H. 202), Hernández *et al*. in 2014, Sánchez in 2014 Coppage and Polzin in 2002 (Davenport, P., & Sola-Visner, M. 2023; Burrows, R. F., & Andrew, M. 1990), and Alexander *et al*. in 1999 maintain that cesarean delivery is the treatment of choice when a severe hypertensive disorder is present. It is worth noting that three fundamental factors are considered when opting for this procedure: gestational age, maternal health, and fetal health.

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It is impossible to predict which pre-eclampsia patients will experience a pre-eclampsia episode. In a study conducted at the Mariana Grajales Obstetrics and Gynecology Hospital in Santa Clara, 26.7% of patients showed no clinical symptoms prior to a pre-eclampsia episode, while only 46% had a diastolic blood pressure above 110 mmHg. This reinforces the view that it is impossible to identify patients at risk of developing pre-eclampsia for targeted preventive treatment.¹⁷ Pre-eclampsia is a leading cause of maternal mortality in almost all

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developing countries, with approximately 50,000 deaths annually.¹⁸ At this hospital, since 1999, magnesium sulfate has been prescribed, as suggested by the MAGPIE study,¹⁹ for all cases of pre-eclampsia, regardless of its severity or gestational age.

The MAGPIE 19 study, the largest and most comprehensive to date, encompassing 33 countries across all continents, including Cuba, demonstrated that the drug's use in all cases of preeclampsia reduced the incidence of preeclampsia by 58% (95% CI 40-71), reduced the incidence of retroplacental hematoma by 33% (RR 27% (99% CI 11-55)), and potentially halved maternal mortality (0.2% vs. 0.4%).

There is insufficient scientific evidence to recommend this drug for all cases of preeclampsia, nor to advise against its use if it has beneficial effects on the lives of pregnant women. Its use in non-severe preeclampsia requires further research to determine its efficacy, which remains unproven.

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