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Comparison of Maternal Characteristics and Fetal Changes in Pregnant Women with Mild and Severe Pregnancy-Induced Hypertension (PIH) and Pregnant Women with Normotension

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Abstract:
The way people live their lives has changed dramatically in recent decades.The contemporary society places a high value on education and employment, which has boosted people's standard of living and length of life expectancy. On the other hand, it causes people to delay getting married and having children. Inactivity and poor diet are major contributors to the epidemics of obesity, diabetes, polycystic ovary syndrome, and high blood pressure. Certain issues related to pregnancies may be avoided if the bride and groom wait until the right time to start a family. Women with moderate and severe cases of pregnancy-induced hypertension (pih) are compared to women with normotension in terms of maternal features and foetal alterations.

1. Introduction

In underdeveloped countries, Pregnancy Induced Hypertension (PIH) is a leading cause of maternal and foetal morbidity and mortality. Pregnancy-related hypertension is a leading cause of mother and infant death in low-income nations. High blood pressure is the primary cause of preeclampsia, which commonly manifests after 20 weeks of pregnancy. Preeclampsia is a complex condition that threatens the health of both mother and unborn child. Multiple variables, including gestational age (GA) at beginning, illness severity, and co morbid diseases such diabetes mellitus, renal disease, thrombophilia, and preexisting hypertension, might influence the prognosis and complications of pregnancy in hypertensive disorders.[1]

These issues might be classified as either immediate or delayed. Foetal and maternal difficulties may arise in the short term, though the former are more common than the latter. Seizures (eclampsia), a decrease in glomerular filtration rate (which can cause minimal proteinuria or reversible asymptomatic nephrotic syndrome), a reduction in glomerular filtration rate (which can cause haemorrhagic and ischemic strokes that leave residual neurological deficits like hemiplegia), visual disturbances like cortical blindness, retinal detachment, pulmonary oedema, acute Complications include an increased risk of premature birth, caesarean section, abruptio placentae, and induction of labour.[2]

Complications during pregnancy include intrauterine foetal mortality, intrauterine growth restriction, oligohydramnios, preterm, antepartum and

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intrapartum asphyxia, and an increased need for neonatal intensive care unit admissions. Cardiovascular disease mortality in old age. Primigravida women with severe and early onset illness have a significant chance of recurrence in future pregnancies (25-65%), although this risk increases with each pregnancy. Women with a long history of hypertension are at far higher risk for developing the chronic forms of the condition as well as for complications such heart disease, stroke, diabetes, and thromboembolism.[3]

Multipara, early onset preeclampsia, recurring preeclampsia, severe preeclampsia, prenatal hypertension, and preeclampsia all increase the risk of cardiovascular problems during pregnancy. Women who have had preeclampsia are more likely to get kidney failure later in life. Repeated experiences of preeclampsia in two or more pregnancies raises the likelihood of this complication. Other maternal indications include preterm birth after 34–37 weeks of pregnancy, severe polyhydramnios for no apparent reason, severe maternal trauma, and persistent membrane rupture.[4]

The hypertensive diseases of pregnancy have been categorised in various ways. This categorization was proposed by the National High Blood Pressure Education Programme Working Group in 2000. Gestational hypertension (GH) and preeclampsia (PE) are two forms of pregnancy-related hypertension. Hypertension of pregnancy, preeclampsia, and eclampsia all in one.For the first time after 20 weeks of pregnancy and without proteinuria, GH is defined as blood pressure (BP) 140/90 mm of Hg, with BP returning to normal within 12 weeks postpartum.Proteinuria of 300 milligrammes per 24 hours or more on a dipstick after 20 weeks of pregnancy indicates preeclampsia. When a woman has seizures for no apparent reason, it is called eclampsia. Combination of preeclampsia with long-term hypertension. Women with hypertension may have proteinuria over 300 mg/24 hours after 20 weeks of pregnancy, but not before that time. Rapid elevation of proteinuria, hypertension, or platelet count 1,00,000/mm before 20 weeks' gestation in women with hypertension and proteinuria Diagnosis A pre-pregnancy blood pressure reading of 140/90 mm of Hg indicates chronic hypertension. Diagnosis of hypertension at 20 weeks of pregnancy with continued high blood pressure after 12 weeks of breastfeeding.[5]

Although its precise cause is uncertain, preeclampsia has been linked to a variety of potential contributors. In the first stage, placental hypoxia results from improper endovascular trophoblastic remodelling of the uterine arteries. Clinical symptoms of preeclampsia develop in stage 2 due to oxidative stress, which triggers the release of placental factors into the maternal circulation, which in turn triggers a systemic inflammatory response & endothelial cell activation. Cardiovascular or renal illness, obesity, diabetes, immunological problems, and heredity are all examples of preexisting maternal conditions that might affect the second stage. In normal pregnancy, placental implantation is accompanied by the proliferation of endovascular trophoblasts that invade the decidua or extend into the walls of the spiral arterioles, where they replace the endothelium and muscular wall to produce a dilated, low-resistance flow. [6]

This phenomenon is known as abnormal trophoblastic invasion. In preeclampsia, however, the spiral arteriolar wall is only partially invaded by endovascular trophoblasts, leading to the formation of a small-calibre artery that has a significant resistance to flow. The average diameter of these blood vessels will be less than that of regular placental blood vessels. It indicates that the severity of hypertension is proportional to the degree of faulty trophoblastic invasion. There is evidence that immunological mechanisms play a role in the development of preeclampsia, namely the loss or dysregulation of maternal immune tolerance to paternally generated placental and foetal antigens.[7]

Preeclampsia is more common in first pregnancies, molar pregnancies, and situations where the paternal antigenic load is large; this may be due to histological alterations at the maternal placental interface that are similar to those seen in acute graft rejection. Placental dysfunction in endothelial cells is thought to cause maternal endothelial dysfunction. Vasoconstriction and end-organ ischemia are among the clinical symptoms of preeclampsia, reflecting extensive endothelial dysfunction. Changes in the production and secretion of endothelial cell products may be an indicator of endothelial dysfunction. Endothelial damage, leading to the clinical condition of preeclampsia, is hypothesised to be triggered by antiangiogenic and metabolic factors as well as other inflammatory mediators. Extreme leukocyte activation

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in the maternal circulation has been linked to endothelial cell dysfunction. [8]

Cytokines including tumour necrosis factor-alpha (TNF-alpha) and the interleukins (IL) contribute to oxidative stress. Lipid peroxides, which may spread by themselves, are formed when reactive oxygen species are released. This throws off the body's natural prostaglandin equilibrium and nitric oxide synthesis. This then causes proteinuria and oedema by activating microvascular coagulation and increasing capillary permeability. Pre-eclamptic women had higher plasma levels of endothelin I (ET-I) compared to pregnant women who do not develop this condition. A powerful vasoconstrictor, this peptide consists of 21 amino acids. Systemic endothelial cell activation results in elevated endothelin levels.[9]

Pregnancy-related hypertension continues to be a major contributor to mother and infant mortality, particularly in low-income regions. They account for 8-9 percentage points of maternal mortality in India. they affect 5-10% of pregnancies.The placenta is essential for the baby to survive within the uterus. The placenta plays a crucial role during pregnancy, ensuring that the baby grows and develops normally. It's the best evidence we have of what the baby went through while in the womb. It's the anatomical record of the pregnancy, including what happened in the uterus and at delivery. Macro and microscopically, the placenta reflects the presence of pregnancy complications such as hypertension. Abnormal placental gross morphology and histology may compromise placental function, which may contribute to a number of problems in these pregnancies. Reduced utero-placental blood flow and constriction of the foetal stem arteries due to maternal vasospasm causes a number of macroscopic and microscopic alterations in the placenta. [10]

2. Material And Methods

June 2020–December 2021 was the research period. In this research, we measured SBP, DBP, Alb, parity, and GA in the mothers, and foetal weight was measured in the fathers. Two numbers are used to determine a person's blood pressure. The first value is the pressure in the arteries during a heartbeat, and is known as the systolic blood pressure. The pressure in the arteries when the heart is at rest is the second value given. Most adults have normal blood pressure if their systolic SBPreading is less than 120 and their diastolic reading is less than 80. Mild hypertension is characterised by a sphygmomanometer reading of 140/90 mm Hg or above. When BP is consistently above 160/110 mm Hg, it is considered to be severe hypertension. The international organisation for the study of hypertension in pregnancy ISSHP provides the following definition of hypertension. DBP of 90 mm Hg, but tested twice at least 4 hours apart, reducing the impact of "white coat hypertension" or "hypertension in medical settings." Even a single measurement of diastolic pressure at 110 mm Hg is adequate for diagnosis.

The woman's blood pressure was taken as she sat with her arm at heart level. The right sized cuff was employed. Diastolic blood pressure was designated using the korotk off phase V notation. If one person's blood pressure readings are regularly higher than the other, the readings from the higher arm should be utilised going forward. Preeclampsia-validated automated blood pressure machines, mercury sphygmomanometers, and aneroid devices may all be used to take blood pressure readings. Comparison of readings with a mercury sphygmomanometer or a calibrated aneroid device is suggested since automated blood pressure equipment that have not been approved for use in women with preeclampsia may underestimate or overestimate blood pressure in such women.

The normal range for Alb is between 3.4 and 4.8 g/dl. Proteinuria may be diagnosed using a dipstick. The random pee dipstick test requires a collection of 50 ml of urine in a container for laboratory examination. The Uri plus 900 urinalysis strip is used for the dipstick analysis. Proteinuria severity levels: The normal range for blood alcohol content is as follows: traces (15–30 mg/dL), 1+ (30–100 mg/dL), 2+ (100–300 mg/dL), and 3+ (300–1000 mg/dL). Hormonal shifts during pregnancy may drastically alter a woman's physical appearance. An association between decreased blood albumin levels in the third trimester of pregnancy and higher mother and newborn mortality and morbidity has been suggested. Mother's parity represents the total number of live births from viable pregnancies. Primi-gravida refers to a first-time or former pregnant lady. A lady with many pregnancies is called a multi gravida.

gestational age the number of weeks along in a

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pregnancy. It is calculated by counting the number of weeks from the start day of the woman's most recent menstrual cycle. Pregnancies typically last between 38 and 42 weeks. Premature babies are those born before 37 weeks of gestation. Premature babies whose birth weight is less than 2,500 kg. Foetal birth weight ranges from around 2.5 to 4.5 kilogrammes. It was measured soon after birth. Premature babies whose birth weight is less than 2.5 kg. Births to mothers with high blood pressure are more likely to have infants with low birth weight.

3. Results

Maternal parameters

Table displays the average systolic and diastolic blood pressures, together with their respective standard deviations.Table details the alb, GA, and parity. Normal, moderate, and severe PIH all had SBP values of 116.0, 145.2, and,173.6mmHg, respectively. This result was statistically significant. DBP averages 75.0, 98.2, and 113.4 mm Hg in normal, moderate, and severe PIH, respectively. This result was statistically significant. Classifying proteinuria as 0+, 1+, 2+, or 3+. One hundred percent of women in the nil, 1+, moderate, and severe PIH categories had proteinuria. Protein was found in the urine of 36% of women with moderate PIH and 96% with severe PIH. There was a substantial difference here (P 0.001). Pregnancy at 35 weeks or more is considered GA. 8% of women with moderate PIH, 36% of women with severe PIH, and 100% of women with PIH at or after 35 weeks are considered normal. Statistically, this is a huge deal (P 0.001). In terms of parity, pregnancies may be either "primary" or "multigravida." All of the pregnancies in the normal, moderate, and severe PIH groups were primary pregnancies. Multiple pregnancies with moderate PIH (38%) and severe PIH (40%) are common. There was a substantial difference here (P 0.001). Statistically significant differences were detected in the current investigation between normal, moderate, and severe PIH in SBP, DBP, Alb, GA, and parity.

Table 1: Maternal parameters of pregnant women with moderate and severe pregnancy-induced hypertension (PIH) compared to pregnant women with normal blood pressure.

S.No	Parameter	Groups	Mean	SE
1	Systolic bloodpressure(SBP)(mmHg)	Control	116.0	0.7
		PIH-mild	145.2	0.7
		PIH-severe	173.6	1.6
2	Diastolic bloodpressure(DBP)(mm Hg)	Control	75.0	0.7
		PIH-mild	98.2	0.5
		PIH-severe	113.4	1.0

Table2: Maternal parameters of pregnant women with moderate and severe pregnancy-induced hypertension (PIH) compared to pregnant women with normal blood pressure

S. No.	Variable	Category	Control (normal)	Mild PIH	Severe PIH	Statistics
1	Albumin	Niland1+	50	32	2	2=95.455
		2+and3+	0	18	48	P<0.001

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2	Gestationalage	< 35weeks	0	4	18	2=28.551 P<0.001
		> 35weeks	50	46	32	
3	Gravida	Primary	50	31	30	2=26.403 P<0.001
		Multi	0	19	20	
n=50each						

Foetal parameters: Normal, moderate, and severe foetal birth weights, together with their associated standard errors, are listed in Table. This was a significant finding statistically.

Table3: Foetal parameters in women with moderate and severe pregnancy-induced hypertension (PIH) compared with those in women with normal blood pressure throughout pregnancy.

S. No	Parameter	Groups	Mean	SE
1	Foetal-weight(g)	Control	3200.5	51
		PIH-mild	2703.0	82
		PIH-severe	2237.0	112
n=50each				

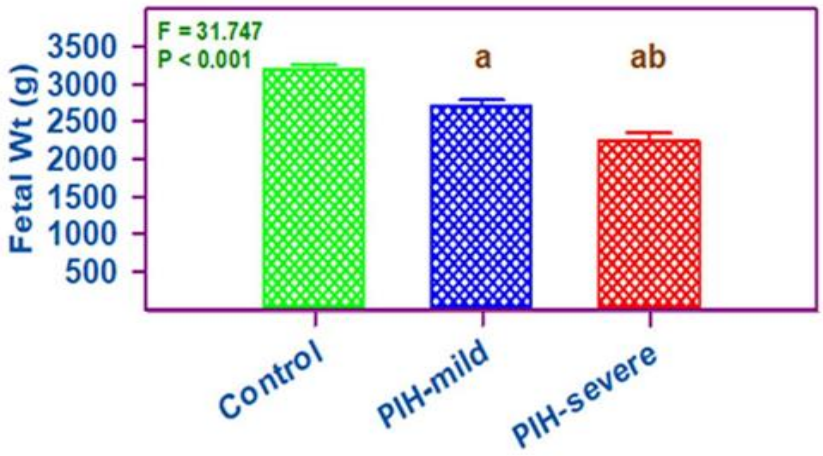

Figure1: Foetal weight in preeclamptic hypertension (mild vs. severe) vs controls. Sum of the means + standard error (n = 50 for each) One-way ANOVA followed by Bonferroni's correction yields the "F" and "P" values. „t" test. a PIH considerably different to the normative sample. statistically distinguishable from the PIH mild group.

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4. Discussion

As a leading cause of maternal and infant morbidity and death, hypertensive disorders during pregnancy have been recognised as a global health crisis. Researchers have found that pregnant women with hypertensive disorders are more likely to experience acute or chronic utero-placental insufficiency, which can affect perinatal and neonatal outcomes by causing anoxia before, during, or after delivery. Several studies have shown that hypertensive complications during pregnancy are associated with a higher risk of having a baby that is premature, has a low birth weight, or is born extremely prematurely. Maternal or foetal morbidity and death are mostly attributable to PIH, despite the fact that it is a very uncommon condition among pregnant women. Preterm birth, low birth weight, and very low birth weight babies were more common in hypertensive pregnancies compared to healthy pregnancies. Prematurity and birth asphyxia were the leading causes of perinatal death in this research.

SBP, DBP, Alb, GA, normal parity, moderate PIH, and severe PIH were all shown to be statistically different from one another. The severe group had a higher average SBP (173.6) than the moderate group (145.2). The results of the statistical analysis of this parameter were positive. Findings showed that severe cases had higher mean DBP (113.4) than moderate cases (98.2). The results of the statistical analysis of this parameter were positive.Protein was found in the urine of 36% of women with moderate PIH and 96% with severe PIH. There was a substantial difference here (P 0.001). One study by VassilikiKrielessi and Nikos Papantoniou (2013) found that proteinuria of more than 300 mg per day was present in 80% and 65.45% of cases of severe and mild hypertension, respectively, while proteinuria of more than 2 gm per day was present in 38.18% and 12.72% of cases of severe and mild hypertension, respectively. Statistical analysis confirmed the validity of these findings. Urine albumin concentrations were formerly employed as a criterion for determining the severity of hypertensive disorders during pregnancy.

Women with severe hypertension were more likely to give birth prematurely than those with moderate hypertension, according to the current research. A greater proportion of premature births were seen in severely hypertension women compared to mildly hypertensive women, as measured by the gestational age at delivery. Women's parity is a risk factor for hypertensive disorders during pregnancy. There was a considerable rise in the number of first-time mothers, but there was no correlation between parity and hypertension severity. First-time mothers were more likely to have hypertension problems. They found that hypertension problems in women decreased as their fertility increased. Another research, conducted in 2007, by Sandhya et al., found similar outcomes. Higher proportion of first-time mothers with severe hypertension compared to those with moderate hypertension.[11]

When comparing the birth weight of foetuses from mothers with moderate and severe hypertension, it was shown that the mean birth weight of foetuses born to mothers with mild hypertension was substantially higher (2703 kg) than that of foetuses born to mothers with severe hypertension (2237 kg). There was a statistically significant difference between the two groups on this metric. Comparing the placental changes in mild and severe hypertensive disorders complicating pregnancy, Thesevere hypertensive group had a lower mean birth weight than the mild hypertensive group. The results of these research suggest that there is a correlation between hypertension severity and mean birth weight. similar findings; they found that low birth weight was substantially related with the severe hypertension group. Braunthal and Brateanu (2019) found similar outcomes in one of their studies.[12]

The results of the current investigation showed that the calcifications, TPC, and FD of moderate and severe hypertensive placentas differed significantly with maternal age, whereas the other histological characteristics did not.Analysed55 mildly hypertensive placentas and 55 severely hypertensive placentas. Although they found a higher prevalence of calcifications in the severe hypertension group, they did not find this difference to be statistically significant when compared to the moderate hypertension group (25.45%). The severity of hypertension was connected with the results. The degree of hypertension was also connected with the results of a research of 130 placentas.[13] Calcifications were reported to be more common in the severe hypertension group compared to the moderate hypertensive group. The current investigation found comparable outcomes. Studies have shown that severe

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hypertensive people are more likely to have calcifications in their placentas. In their study of 200 instances of preeclampsia, researchers Vijayalakshmi, Sunitha Kitteli, et al. found that there was a statistically significant increase in the frequency of Tenny parker alterations (syncytial knots) in the severe cases compared to the moderate ones. Syncytial knots were shown to occur more often in the severely hypertension subjects evaluated compared to the mildly hypertensive cases. Researchers found that severe hypertensive patients were more likely to have fibrin deposition than those with moderate hypertension. The frequency of fibrin deposition was higher in the hypertensive group than in the normal group, according to a research by Das, Dutta, and Chakraborty, although they did not distinguish between moderate and severe hypertension.[14]

When comparing women with mild and severe hypertensive disorders of pregnancy, the severe group's placental weight, FTP ratio was lower than the mild group's even after accounting for gestational age. Similar outcomes. Another research, finding that the mean foetal weight to placental weight ratio was lower in the severe hypertension group compared to the moderate group.[15]

5. Conclusion

According to the results of this study, researchers discovered no correlation between maternal age and placental thickness, diameter, or weight, however they did find a correlation between FTP ratio and the severity of PIH. According to research by Saurjya Ranjan Das, Pradeep Kar, et al., the number of patients with decreased placental thickness was greater in the severe hypertension group than in the moderate hypertensive group.

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