A STUDY OF PREGNANCY OUTCOMES AND PLACENTAL MORPHOLOGY ASSOCIATED WITH PREGNANCY-INDUCED HYPERTENSION

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ABSTRACT

BACKGROUND

Pregnancy-Induced Hypertension (PIH) contributes significantly to maternal and perinatal morbidity and mortality. Women with PIH are at a greater risk of abruptio placentae, cerebrovascular events, organ failure and disseminated intravascular coagulation. Foetuses of these mothers are at greater risk of intrauterine growth retardation, prematurity and intrauterine death. PIH brings about histomorphological changes in the placenta thereby resulting in placental dysfunction. Present study aims to understand the morphological changes in the placenta along with maternal and foetal outcomes in pregnancies complicated by PIH.

MATERIALS AND METHODS

A descriptive observational case-control study was conducted from March 2013 to April 2016 in a tertiary care hospital. Hundred women diagnosed with PIH and hundred women with normal gestation were enrolled in the study. Foetal and maternal outcome at term was evaluated in comparison to normal gestation. Placental specimens from term gestations (38-42 weeks) diagnosed with PIH and placenta from normal full-term gestations were studied to assess the morphological parameters. Statistical analysis was done using descriptive statistical measures.

RESULTS

A total of 100 cases with PIH and 100 control normotensive gestations were included in the present study. Gestational hypertension accounted for 66 cases, preeclampsia 23 and eclampsia 11 cases. Liver infarcts were the commonest maternal complication (11%) among the cases followed by renal failure and postpartum haemorrhage (5% each), however, no maternal mortality was documented in our study. Total number of preterm deliveries was 35 and foetal demise was documented in 9 cases. Low birth weight was the commonest foetal complication seen in 37% of the cases. Mean placental weight of PIH cases was found to be 406.5 g, which was significantly lesser than the mean weight of the control group placenta. Multifocal infarcts, retroplacental clots, fibrinoid necrosis and areas of calcification were significantly associated with placental specimens from PIH cases.

CONCLUSION

The study describes the various maternal, foetal and placental outcomes in pregnancies complicated by PIH. The presence of preeclampsia and eclampsia remain the major contributors to maternal and foetal outcome. Regular antenatal checkups, multidisciplinary treatment, optimum timing of delivery and prompt perinatal management reduces fetomaternal complications and mortality.

KEYWORDS

Pregnancy-Induced Hypertension, Placenta, Maternal-Foetal Exchange.

HOW TO CITE THIS ARTICLE: Rao JVN, Chandini J. A study of pregnancy outcomes and placental morphology associated with pregnancy-induced hypertension. J. Evid. Based Med. Healthc. 2017; 4(44), 2673-2676. DOI: 10.18410/jebmh/2017/531

BACKGROUND

Pregnancy-Induced Hypertension (PIH) remains the leading cause of maternal and perinatal morbidity and mortality.[1,2] PIH complicates about 6-10% of pregnancies.[3] Hypertensive Disorders of Pregnancy (HDP) can be classified as pre-existing hypertension, gestational hypertension, preeclampsia and other hypertensive effects.[4]

PIH is defined as Systolic Blood Pressure (SBP) >140 mmHg and Diastolic Blood Pressure (DBP) >90 mmHg that develops for the first time at ≥20 weeks' gestation with or without proteinuria.[4] Based on the clinical presentation, PIH is classified as mild (SBP 140-149 and DBP 90-99 mmHg), moderate (SBP 150-159 and DBP 100-109 mmHg) and severe (SBP ≥160 and DBP ≥110 mmHg).[5]

PIH stimulates initiation of systemic inflammatory response and maternal endothelial cell dysfunction thereby resulting in poor placentation and expression of proinflammatory substances resulting in adverse maternal and foetal manifestations.[6] Majority of the adverse reactions is accredited to severe PIH and the development of clinical

Financial or Other, Competing Interest: None.
Submission 16-04-2017, Peer Review 28-04-2017,
Acceptance 12-05-2017, Published 30-05-2017.
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DOI: 10.18410/jebmh/2017/531

manifestations between 20-34 weeks of gestation. Pregnancy-Induced Hypertension (PIH) associated haemolysis, elevated liver enzymes and low platelet count (HELLP syndrome) attributes to 83.3% of the mortality among mothers with PIH.[7] Haemorrhagic stroke and pulmonary oedema have been responsible for 60% of maternal deaths in eclampsia cases.[8] Other maternal short-term complications associated with PIH include central nervous system dysfunction, hepatocellular injury, thrombocytopenia, acute Disseminated Intravascular Coagulation (DIC), oliguria, pulmonary oedema and cerebrovascular events.[9] Foetal complications include intrauterine growth retardation, prematurity and intrauterine death.[10,11]

The placenta is a membranous vascular complex organ with a short lifespan that develops in female eutherian mammals during pregnancy. It facilitates nutrient uptake, waste elimination and gaseous exchange between the maternal and foetal circulations.[12] PIH produces variety of placental abnormalities such as decidual arteriopathy, multifocal infarcts, low placental weights, syncytial knots, maternal vessel thrombosis, fibrinoid necrosis, calcification and abruptio placentae.[13]

This study intends to understand the maternal, foetal outcomes and the possible gross macroscopic changes in the structure of placenta in pregnancies complicated by PIH. Additionally, the study compares the outcomes of gestations complicated by PIH with normal full-term gestations.

MATERIALS AND METHODS

This descriptive, observational, case-control study was carried out in the Department of Obstetrics and Gynaecology (OBG) of a tertiary care hospital from March 2013 to April 2016. Hundred pregnant women diagnosed with PIH (cases) and hundred normotensive pregnant women with no added complications (controls) were enrolled as study subjects. All pregnant women attending the OBG Outpatient Department with PIH diagnosed as per the guidelines provided by the Society of Obstetricians and Gynaecologists of Canada (SOGC) HDP guidelines were included in the study.[4] Patients with preexisting hypertension, renal diseases, twin gestation, hydatidiform mole, type I/II diabetes, epilepsy and chronic diseases or metabolic disorders were excluded from the study. A written informed consent was obtained from all the mothers included in our study. Personnel information such as age, parity, previous obstetric history, family history of diabetes and hypertension, any chronic illness in the past, present complications, medications, diet and blood group of all the subjects was noted. All subjects included in the present study belonged to the same race and ethnicity.

The PIH cases were managed as per the SOGC HDP guidelines.[4] Patients were followed up regularly at antenatal clinic and home blood pressure monitoring. They were informed to report immediately in case any complication (PIH, preterm labour, premature rupture of membranes or decrease foetal movement) should occur. Ultrasonography was done early in gestation for foetal anomalies and was repeated if indicated. At each antenatal visit, blood pressure, urine for protein, maternal and foetal well-being were assessed and if there was any complication, the patient was readmitted and managed accordingly. Doppler ultrasound and kick-count technique was used for foetal surveillance.

Expectant management at 24-33 weeks was employed to reduce neonatal respiratory distress syndrome, necrotising enterocolitis and NICU care despite poor foetal growth velocity during the time gained decision about time and mode of delivery was made at 34-37 weeks of gestation. Timing of delivery was individualised. The paediatrician assessed all newborns immediately after delivery and the data was entered in a structured proforma.

The placenta from the subjects was obtained were either by vaginal delivery or by caesarean section. After delivery of the placental tissue, the shape and dimensions were noted. The umbilical cord was observed for its position of insertion, number of vessels, knots or any other changes. The cord was cut 5 cms away from its site of insertion. The placental membrane was trimmed by rolling technique and the placental specimens were weighed on weighing machine graduated in grams (g) after rinsing with running tap water and drying with blotting paper. The specimen was then subjected to gross macroscopic examination.

Statistical Analysis- Chi-square test was applied to categorical data such as maternal and foetal outcomes. Numerical data, i.e. Foetal weight, diameter, vascular pattern of the placenta and site of attachment of the umbilical cord were presented as mean scores and student's t-test was applied to compare the mean between two groups (cases and control). Entire data was calculated on 95% CI. A p value <0.05 was considered significant.

RESULTS

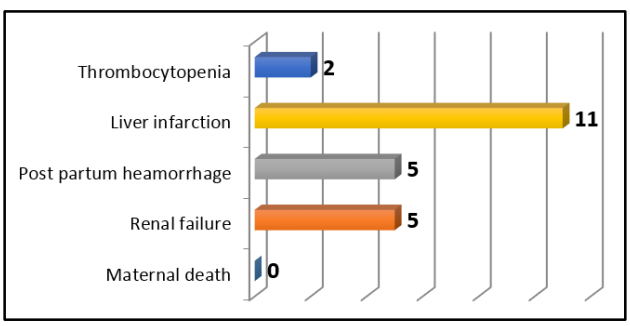
A total of 100 PIH cases and 100 normotensive controls were included in the present study. Out of the 100 PIH cases, gestational hypertension accounted for 66 cases, preeclampsia 23 and eclampsia 11 cases. Mean age of the study population was 27.3 ± 2.1 years with the eldest subject being 36 years and the youngest being 24 years of age.

Maternal complications observed in the present study are documented in figure/table 1. Postpartum haemorrhage and acute renal failure was noted in 5% of the cases. Two cases suffered from thrombocytopenia and 11% of them presented with liver infarction. All complications were promptly managed successfully treated by multidisciplinary approach and therefore no maternal mortality was documented in the present study.

In our study, there was no significant difference between the numbers of vaginal deliveries in comparison to the numbers of caesarean sections (Figure/Table 2). Out of the 100 deliveries, 56% were full term and 35% were preterm births. Weight of the baby was less than the expected weight for gestational age in 37% of the study population, (Figure/Table 3) 2 stillbirths and 7 abortions were recorded.

Placental weight was significantly lower in PIH group when compared to the control group. The mean weight of

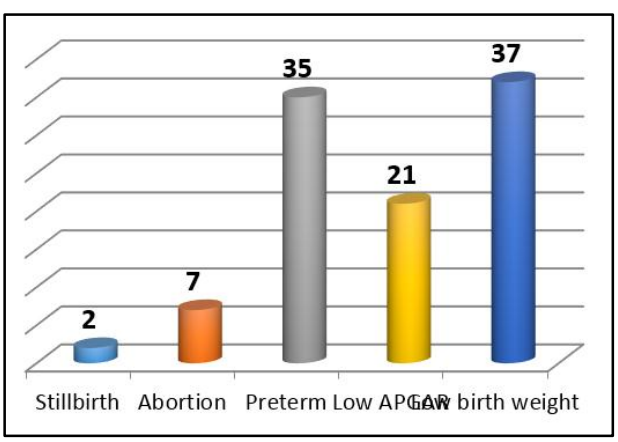
placenta in control group was 502 g and in PIH mothers it was 406.5 g, the difference between them was 95.5 g (p value <0.5).[14] Placental specimens from normotensive controls showed no significant complications in contrast large multifocal infarcts were the most common placental pathology noted in all 11 cases of eclampsia and 2 cases of preeclampsia. Presence of retroplacental clot was noted in 8% and placental calcification was noted in 7% of the study population (p value <0.05).[14]



Figure/Table 1. Unfavourable Maternal Outcome among Cases

Mode of Delivery	Cases (n=100)	Controls (n=100)
Vaginal delivery	42%	85%
Forceps assisted delivery	6%	3%
Vacuum delivery	1%	Nil
C-section	51%	12%

Figure/Table 2. Distribution of Cases and Controls According to the Mode of Delivery



Figure/Table 3. Unfavourable Foetal Outcome among Cases

DISCUSSION

PIH is a common condition responsible for majority of maternal and foetal morbidity and mortality. Incidence of preeclampsia in the present study was 23 and eclampsia was 11 out of 100 PIH cases over a period of three years. The decreasing trend of incidence maybe attributed to increased awareness, early diagnosis and prompt treatment of preeclampsia.

In the present study, the mean age of presentation was 27.3 ± 3.9 years. A study by Kolluru V et al[15] showed a mean age of presentation of 23.52 ± 4.33 years. A large population-based cohort study conducted in Nova Scotia, Canada[16] from 1988-2000 found the mean age of presentation to be 27.225 ± 5.85. Therefore, no definitive association of maternal age with PIH incidence could be appreciated.

Liver infarction was found to be the most common maternal complication associated with 11% of the study population followed by postpartum haemorrhage and acute renal failure in 5% of the study population. Studies by Seyom et al[17] showed 12% incidence of liver infarction followed by 7% incidence of renal failure. Studies by Kolluru et al[15] showed 1.28% incidence of acute renal failure and postpartum haemorrhage.

In this present study, number of vaginal deliveries (49%) were almost equal to the number of caesarean deliveries (51%), which is similar to studies by Uddin AW et al[18] and Kolluru V et al.[15] However, studies by Parmar et al[19] and Bangal et al[20] showed a higher incidence of vaginal deliveries among PIH cases. The higher rate of C-section in our study can be attributed to our hospital being a higher referral center to manage complicated cases.

Low birth weight was the most common complication totalling up to 37% followed by low Apgar that was found in 21%. Studies by Seyom et al[17] showed 31% incidence of low birth weight and 19% incidence of low Apgar comparable to the findings of the present study. Low foetal mortality rate in the present study is attributed to the multidisciplinary management, NICU facility and round the clock care received by the neonates.

This study documents a low mean placental weight among PIH cases with multifocal infarcts being the most common pathology followed by retroplacental clots and calcification. The findings of the present study correlated with the findings of studies by Jain U.A et al[21] and Majumdar et al.[22] PIH adversely influences the placental pathology thus histomorphological examination occupies an undisputed role in the analysis of placental dysfunction associated with PIH.

CONCLUSION

Present study documents PIH as an obscure aetiological cause of fetomaternal mortality and morbidity. The study also describes the various gross macroscopic changes seen in the placenta of mothers with PIH in comparison with normal gestation.

Early detection combined with prompt treatment proves to have a crucial and definite role in reducing the morbidity and mortality of both mother and foetus. Adverse placental pathology affects the growth and nutrition of foetus in utero. Therefore, the interruption of placental dysfunction can bequest to significant improvement in maternal and foetal well-being in subjects with PIH.

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