
Relationship of Placental Infarcts with the Foetal Outcome in Pregnant Patients having Hypertensive Disorders

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Abstract

Objective: to determine the frequency of placental infarcts in pregnant patients having hypertensive disorders and to find out its relationship with the Foetal outcome in these patients

Study Design: cross-sectional study.

Place and Duration of study: department of Obstetrics and Gynaecology unit 111, Lady Willington Hospital, Lahore over a period of six months from 1-01-2012 to 30-6-2012.

Methodology: 100 cases of pregnancy induced hypertensions were included in the study. The placentas of the subjects were examined and weighed after umbilical cord, extra placental membranes and adherent blood were removed and then fixed in 10% formalin.

Results: mean age of the patients was 24.9±3.7. Out of one hundred patients, placental infarcts were found in 34(34.0%) patients. Distribution of placental infarcts according to their grades reveals 21(61.7%) patients belong to \leq 3cm while in remaining 13(38.3%) patients grade of placental infarcts was > 3cm. Out of 34 Placental infarcts, 12 (35.3%) were focal and 22 (64.7%) were multifocal. Stillbirth occurred in 4 (11.7%) patients, Intra uterine growth restriction (IUGR) in 19 patients (55.9%) and Apgar score of less than 7 in 11 (32.4%).

Conclusions: placental infarcts are seen in 34% placentae of women with hypertension. Foetus is adversely affected by the placental infarcts.

Keywords: pregnancy induced hypertension, placental infarcts, still birth.

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Introduction

The hypertensive disorders of pregnancy are found in around 12 to 22% of all pregnancies, which can cause significant maternal and perinatal mortality and morbidity, however, in the developed countries incidence of hypertension and severity of its complications has fallen markedly due to effective and widespread antenatal care.¹

Hypertension in pregnancy is of two kinds, either it is of gestational hypertension, which occurs solely in pregnancy or it includes pregnancy induced hypertension (PIH) and Preeclampsia or chronic hypertension.² The international society for the study of hypertension in pregnancy (ISSHP) uses the term gestational hypertension when the women have previously been normotensive.³ Gestational hypertension is responsible for 70 % cases of hypertension in pregnancy.⁴ There needs to be one measurement of diastolic BP of 110 mmHg or more or two consecutive measurements of diastolic BP of 90 mmHg or greater four hours or more apart at rest after 20 weeks of gestation to qualify for the definition⁵. Pre-eclampsia is said to be the disease in which placenta, mother and foetus are adversely affected. Placenta shows both macroscopic and microscopic changes due to hypertension.⁶ Acute atherosclerosis is a disorder seen in the placenta of the woman suffering from pre-eclampsia, which leads to placental infarcts and basal haematomata. Peripheral infarcts are more common and are usually of no clinical significance, on the other hand the infarcts

occupying more than 5% of placental mass or >3 cm of size are associated with perinatal mortality.⁷

The incidence of placental infarction ranges from about 33% in case of mild pre-eclampsia to approximately 60% in severe disease.⁸

Foetal effects of placental infarcts are placental insufficiency and foetal growth restriction, which ultimately leads to foetal hypoxia, foetal distress and foetal death. It is also responsible for Apgar score of <7 at 5 minutes in the newborn.⁹ The aim of our study was to correlate PIH and placental infarcts clinically and to see the effects of placental infarcts on foetal outcome, due to the importance of this topic, its clinical implications and absence of sufficient local data on the subject, present study was planned to be conducted at the local setup. PIH and placental infarcts has adverse effects on foetal outcome and the condition is recurrent as well, this study also helped us in planning and management of future pregnancy of the patients studied as well as all other hypertensive patients.

Methodology

This cross sectional study was carried out at the department of Obstetrics and Gynaecology, unit 111 Lady Willington Hospital, Lahore, from 1.1 2012 to 30.6.2012 i.e. a period of six months. One hundred cases were selected who fulfilled the inclusion criteria by non-probability consecutive sampling.

Inclusion criteria: patients of 15-45 years and of any parity with pregnancy induced hypertension

admitted after 28 weeks of gestation calculated by dating scan.

Exclusion criteria: pregnant women with other illness like diabetes (random blood sugar > 160 mg %), renal disease (serum creatinine > 1.2 mg/dl), non-compliant subject and patients having hypertension before 20 weeks of gestation were excluded from the study.

Permission was obtained from ethical committee of Lady Willington Hospital, Lahore. After taking written informed consent, the placentas of the selected patients were taken from the labour room. The placentas of the patients were examined and weighed after umbilical cord, extra placental membranes and adherent blood were removed and then fixed in 10% formalin, and then the samples were sent to Hospital laboratory. Gross examination of placenta was done and section of placenta was stained with haematoxylin eosin stain. Slides were reviewed by the pathologist for presence or absence of infarcts.

Apgar score was noted with the help of specified chart, baby's birth weight was recorded by birth weight machine, whereas, the IUGR was assessed with Doppler ultrasound after 28 weeks antenatally. Still birth was determined after delivery. Data was recorded on specially designed Performa and was stratified for severity and grades of placental infarcts to control effects modifiers. Presence of placental infarcts was graded as less than 3cm or more than 3cm while severity of disease was as focal or multifocal.

The data was analyzed by using SPSS version 16.0. Mean and standard deviations were

determined for quantitative variables like age of the mother and gestational age. Frequency and percentages were determined for qualitative variables like placental infarcts (presence of absence) and foetal outcome (alive birth, low Apgar score, still birth, IUGR).

Results

Among the 100 cases, regarding the age distribution of patients, majority of the patients i.e. 53 (53.0%) were between 20-30 years of age while minimum 9 patients (9.0%) were more than 40 years of age. Mean age of the patients was 24.9 ± 3.7 .

Most of the patients, 48 (48.0%) presented with 37-40 weeks of gestation while minimum 7 patients (7.0%) were between 28-32 weeks gestation with mean gestational age of 38.1 ± 3.1 weeks.

Out of one hundred patients, placental infarcts were found to be present in 34 patients (34.0%) (Table I).

Table I. Distribution of cases by placental infarcts

Placental infarcts	Number	Percentage
Yes	34	34.0
No	66	66.0
Total	100	100.0

Distribution of placental infarcts according to their grades reveals 21 patients (61.7%) belong to ≤ 3 cm while in remaining 13 patients (38.3%) grade of placental infarcts was > 3 cm (Table II).

Table II. Placental infarcts according to grades (n= 34)

Grades	Number	Percentage
≤ 3 cm	21	61.7
> 3 cm	13	38.3
Total	34	100.0

Out of 34 placental infarcts, 12 (35.3%) were focal and 22 (64.7%) were multifocal (Table III).

Table III. Placental infarcts according to severity (n=34)

Severity	Number	Percentage
Focal	21	35.3
Multifocal	22	64.7
Total	34	100.0

Foetal outcome in these 34 patients of placental infarct shows, stillbirth in 4 patients (11.7%), IUGR in 19 patients (55.9%) and Apgar score of less than 7 in all patients (32.4%) (Table IV).

Table IV. Foetal outcome of cases with placental infarcts (n=34)

Foetal outcome	Number	Percentage
Stillbirth	4	11.7
IUGR	19	55.9
Apgar score of less than 7	11	32.4
Total	34	100.0

Discussion

Pregnancy is associated with many complications, among these pre-eclampsia is one of the serious complications in which there is an altered uteroplacental circulation. In pregnancies complicated by hypertension placental villi undergo many changes, which are enormous if the condition is severe, and maternal and perinatal outcomes are adversely affected by the grades of pregnancy induced hypertension.¹⁰

Placental infarcts represent focal utero placental ischemia and the extent of it in pregnancies complicated by pre-eclampsia. As the severity of the disease increases infarcts frequency also increases.¹¹ In placenta of the woman affected by hypertensive disorders of pregnancy there is an

increased prevalence of infarction, inflammation, ischemia and intervillous hemorrhage, resulting in foetal intrauterine growth restriction.¹² Placental infarctions are the second common type of placental pathology seen in low birth weight infants.¹³

Foetus and placenta are adversely affected by the hypertensive disorders of pregnancy as shown in a previous study conducted by Udainia et al.¹⁴ Our study also proved that the frequency of placental infarction is higher in hypertensive patients i.e., 34.0%. Foetal hypoxia was also observed when pregnancy reaches near term which may lead to foetal distress resulting in foetal death. If pre-eclampsia impairs the placental function risk is enormous.¹⁵ Babies of the mothers with uncontrolled hypertension are small for dates and have birth asphyxia. The current study showed a low Apgar score in newborn babies of the hypertensive patients having infarctions in their placentae, significant association between the placental infarcts and Apgar score of newborns was seen among hypertensive women. Another study has shown similar association.¹⁶

Zook et al carried out a study in the Department of pediatrics, section of Neonatology, Christiana Care health system, Newark, USA. They demonstrated placental infarction in 45% of hypertensive patients¹⁷. In our study this frequency was 32%. In another study carried out on 37 autopsied stillbirths with IUGR, they found placental infarction was present in 26 cases that is why it is considered to be a morphologic marker of uteroplacental insufficiency. Nine of the 26 cases

with both IUGR and placental infarction, where archival tissue was available, had grey matter ischemic lesions that were subsequently identified as "Pontosubicular necrosis". This lesion is now considered as a localized form of apoptosis. A further 8, third trimester stillbirth cases with both IUGR and placental infarction were ascertained prospectively.¹⁸ In this current study, strong association was found between IUGR and placental infarction in stillborns, suggesting that chronic foetal hypoxemia and cerebral apoptosis is caused by uteroplacental insufficiency.

Conclusion

Placental infarcts are seen in 34% placenta of women with hypertension. Foetus is adversely affected by the placental infarcts.

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