Case Report:

'Changes in the Placenta of a Hypertensive and Diabetic Mother'

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ABSTRACT:

After delivery if the placenta is examined minutely, it provides much insight into the prenatal health of the baby and the mother. Placenta is the bridge between the mother and the foetus, formed from fetal membranes and endometrium. When Pregnancy is complicated by diabetes and or hypertension, both the maternal and foetal health is affected, along with structural and functional changes in the placenta of the mother.The placental changes of a hypertensive and diabetic mother have been described in this case report. The mother was a 31 years old primigravida. She came to the Gynaecology Out Patient department of NRS Medical College, Kolkata, in 39th week of her gestation. She had pregnancy induced hypertension and gestational diabetes. After admission she had undergone lower uterine segment Caesarean Section and gave birth to a male baby. Her placenta was collected from the operation theatre to observe the gross and histological features. The placental weight (455gm.) was less than normal and microscopic changes like calcification, fibrin deposition were found in the placenta.

Key Words: Placenta, pregnancy induced hypertension, gestational diabetes

INTRODUCTION

Placenta is the most accurate record of the infants' prenatal experience as stated by Benirschke (1981)[1]. Placenta is a vital organ for fetal development, derived from both foetal and maternal tissues, the maternal portion being the decidua basalis and the fetal portion is chorion frondosum[2]. It is basically meant for exchange of nutrients between maternal and foetal circulation to ensure an optimal environment for foetal growth and development[3,4]. Foetal membranes chorion and amnion cover the placenta[2]. Common pathologies of pregnancy like intrauterine growth retardation, preeclampsia (pregnancy induced hypertension), are associated with incomplete vascular remodeling in the placenta[5].

It is a medical problem, when pregnancy is complicated by diabetes and or hypertension which affect maternal health, architecture and functions of the placenta, may even jeopardize the fetal normalcy. The placenta being the bridge between maternal fetal activities, considered as a window through which maternal dysfunctions and their impacts on fetal well being can be understood[6]. Placental examination is of critical value not only in gathering knowledge about etiologies, outcome and management of the pathological processes affecting pregnancy, but also

in improving the management in subsequent gestations[3,4].

The morphology of placenta is vital for bringing oxygen and nutrients to foetus and removing carbon dioxide and waste products. Gestational and established diabetes and hypertension specifically cause more destruction to placental structure and alter its functions being the major contributors to insufficiency of placenta[2,7]. This case report was done to detect the changes in the placenta of a mother (a primigravida) with pregnancy induced hypertension (PIH) and gestational diabetes.

MATERIALS AND METHODS

The mother was a primigravida, both diabetic and hypertensive. She was thirty one years old, not having diabetes or hypertension before the onset of her pregnancy. But she had family history of diabetes mellitus; her parents are diabetic. She developed diabetes and hypertension during the gestational period and had gestational diabetes and pregnancy induced hypertension. She had undergone emergency lower segment Caesarean Section on 21st February, 2013, in NRS Medical College and gave birth to a male baby. Apgar Score[8] and birth weight of the baby were noted.

Her placenta was collected from the operation theatre. It was weighed with a portion of umbilical cord and membranes, gross features were observed and different measurements were taken. Weight was measured in a weighing machine; diameters, thickness etc. were measured with a scale, vernier calipers. Volume was measured by water replacement and surface area was measured with the help of a graph paper. Then the placenta was preserved in a jar with 10% formal saline.

For microscopic examinations small pieces of the placental tissue were taken from different sites including the centre, 12'clock, 6'clock, 3'clock margins, junction of the cord with placenta. Paraffin blocks were prepared later to make serial sections with a microtome. Some of the sections were fixed on glass slides and stained with haematoxylene and eosin stain for histo-pathological examinations. Photomicrographs were also taken .

OBSERVATIONS

A male child was born with Apgar score 9. Apgar scoring system is an aid for assessing the state of the newborn[8]. **Weight of the newborn baby was 2.5 kg.**

Macroscopic features of the placenta:

1. Weight: 455 gram.
2. Volume: 420cc.
3. Shape: ovoid.
4. Diameters: Longitudinal - 21cm., transverse - 13cm.
5. Surface Area: 215.53sq.cm.
6. Circumference: 48cm.
7. Thickness: 2.9cm (at the centre),
   1.9 cm, 1.8cm, 1.2cm. (at different points of periphery).
8. Cord insertion: eccentric in position to some extent.
9. Number of cotyledons - 16.
10. Length of the cord attached to the placenta: 14.2cm.
11. Foeto – placental weight ratio =
    Birth weight of the baby / Weight of the placenta= 5.61
12. Points of calcification and infarction were present.

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Histological Features (of the placental tissue):

1) Chorionic villi with increased capillaries (> 10 per villi) – Chorangiosis.
2) Infarcted villi, fibrin depositions and intervillous haemorrhage were present.
3) Focal calcification and thickened vessel walls are present.

Impression: Chorangiosis with infarction, suggestive of placental insufficiency are consistent with hypertensive disease.

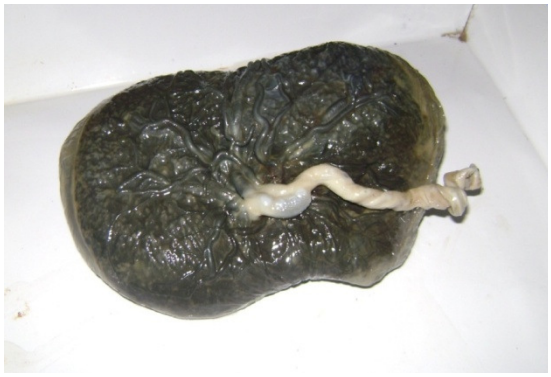

Figure –1

Placenta concerned; foetal surface is visible with the umbilical cord inserted to a side (eccentric insertion).

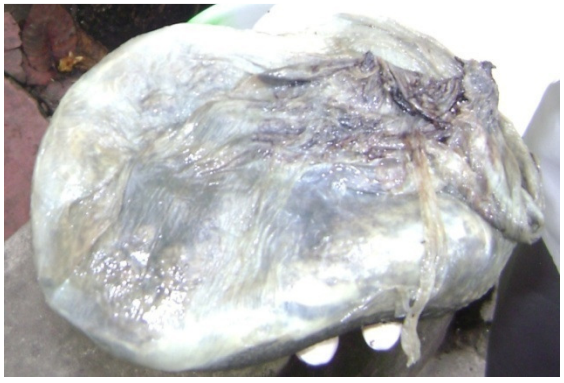

Figure – 2

Maternal surface of the placenta covered with membranes.

Figure – 3

An infracted area was pointed with a forceps in the maternal surface.

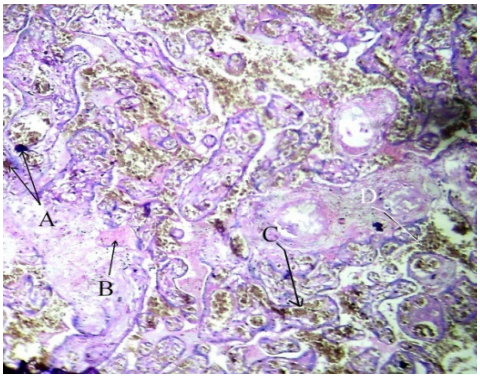

Figure – 4

Histological features of the placenta concerned.

Index: A – Calcification, B – Fibrin deposition, C – Chorangiosis, D –intervillous haemorrhage.

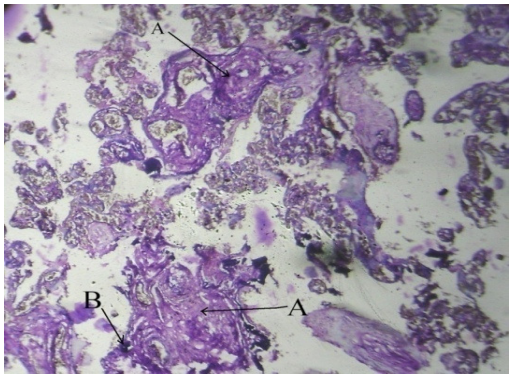

Figure – 5

Histological features of the placenta with villous crowding.

Index: A –Villous crowding, B – Calcification.

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DISCUSSION

Complications of pregnancy like hypertension and gestational diabetes are reflected in the placenta in a significant way, both macroscopically and microscopically [9]. Gestational diabetes is much more common than pre-existing diabetes and it complicates 2% to 5% of pregnancies. When metabolic control is good, perinatal mortality should be no higher than in general population [10].

Pregnancy-induced hypertension (PIH) is a form of high blood pressure occurring in pregnancy. It occurs in about 7 to 10 percent of all pregnancies. It occurs most often in young women with a first pregnancy. Another type of high blood pressure is chronic hypertension - high blood pressure that is present before pregnancy begins. PIH is more common in twin pregnancies, and in women who had PIH in a previous pregnancy[11]. Usually, there are three primary characteristics of this condition, including the following i) high blood pressure (a blood pressure reading higher than 140/90 mm Hg or a significant increase in one or both pressures), ii)protein in the urine, iii) oedema[11].

Pregnancy-induced hypertension is associated with foetal growth retardation[12]. In a mother with hypertension, vasospasm leads to foetal hypoxia as the reduced maternal utero-placental blood flow contributes indirectly to constriction of foetal stem arteries[7,13,]. Rath (1994) stated that in hypertension arrangement of the intracotyledonous vasculature is altered; resulting in low birth weight of the babies[14].

The birth weight of the baby concerned was at the borderline of that of the normal and small for date babies, that is 2.5Kg[8].

The gross anatomic features of placenta e.g, weight, central thickness and diameter were significantly greater in diabetic group as compared to normal and hypertensive groups. Hypertensive group shows non significant decrease in weight of placentae while there was no change in central thickness and diameter in hypertensive than the normal group[10]. At term the placental diameter varies from 200 to 220mm, mean placental weight is 470 gram, mean thickness is 25 mm [5]. These values have similarity with the findings of the present case, though the weight was 455gm.

The values of other gross features also tally with the findings of the previous workers. In one such study gross anatomic features of placentae e.g., infarcted areas, calcified areas and marginal insertion of the umbilical cord in the hypertensive group show significant increase in value ($p>0.01$) when compared to that of the normal group[9]. Pretorius (1996) reported cases of marginal insertion of placenta in about 42% cases of pregnancy induced hypertension[15].

According to Shams et al (2012) there was significantly greater fibrinoid necrosis and hyalinization in placentae from mothers having diabetes and hypertension under light microscope[4]. Significant increase in the microscopic findings like fibrinoid necrosis and hyalinization in villi, proliferation of endothelial lining of capillaries, stromal fibrosis, calcification are seen in the placentae of hypertensive mothers, as described in another study in 2005[9].

Such microscopic findings depict the mosaicism of placenta, probably the aftermath of hypertension[7]. Again the mosaicism leads to placental insufficiency and ultimately to foetal growth retardation, thus creating a vicious cycle[16].In the present case also increased number of capillaries (chorangiosis) and thickened vessel walls due to endothelial

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proliferation, infracted villi, fibrin depositions were found in the placenta on histological observations. These features, suggestive of placental insufficiency, corroborate with the microscopic findings of different studies described above.

CONCLUSION

If maternal illness is a significant one, such as gestational diabetes and or hypertension, that can be harmful in successive pregnancies as well [17]. Placental examination is very important to know about these pathological processes with outcome and to improve the maternal and foetal management in subsequent gestations. The present case will contribute for the same objective.

ACKNOWLEDGEMENT:

We express our gratitude to Professor Sukumar Jana of the Department of Gynaecology and Obstetrics, Nilratan Sircar Medical College, Kolkata –14, West Bengal, India, under whom the mother concerned was admitted for Caesarean section. We are grateful to all the members of the Department of Anatomy and the Department of Pathology of the same institute for their cordial help to study the macroscopic and the microscopic features of the placenta.

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Date of submission: 18 September 2013 Date of Provisional acceptance: 22 September 2013
Date of Final acceptance: 20 October 2013 Date of Publication: 04 December 2013
Source of support: Nil; Conflict of Interest: Nil