Histopathological Changes in Placentas of Gestational Hypertension Women

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ABSTRACT

Background: Gestational hypertensive is a frequent pregnancy condition that contributes considerably to motherly prenatal morbidity then mortality.

Material and Methods: The current study included fifty placentas, twenty-five were collected from uncomplicated normotensive pregnant women, and the rest (25 placentae) were obtained from females with pregnancies complicated by pregnancy induce hypertension at third trimester. Histological sections were prepared using routine hematoxyline and eosin staining.

Results: The morphometric parameters in hypertensive group revealed that there is a decrease in the mean placental weights, mean placenta diameter, mean placenta thickness and mean fetal weight in comparison with normal women. The histological study of placenta with gestational hypertension showed a significant increase in syncytial knots and calcification and hyalinization area also fibrinoid necrosis are observed.

Conclusion: The gestational hypertension well reflected on mother placenta.

Keywords: Gestational hypertensive, Placentae, Uncomplicated normotensive pregnant.

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INTRODUCTION

The placenta is a specialized structure of gestation that promotes the foetus’ typical progress and growth. It is meticulously controlled and synchronized to guarantee that materials and waste products are exchanged efficiently between the motherly and foetal cardiovascular systems.1

Nevertheless, pregnant women with hypertension and diabetes may have changes within macroscope in addition to microscope morphological aspect to the placenta and its free chorionic villi, indicating a risk to the foetus, women, and baby.2

Pregnancy induce hypertension is a frequent difficulty of gestation that can be identified if the mother’s blood pressure is higher than 140/90-millimeter hemoglobin during the second part of the gestation (subsequently twenty weeks of pregnancy) with no existing of protein urea.3,4

Gestational hypertension is a condition that affects the placenta in a variety of methods, together macroscopically as well as microscopically, and is frequently coupled with placental inadequacy, subsequent in foetal development retardation.5 The current study was showed to study the visible and histological variations of placental tissue in pregnancy induce hypertension.

MATERIAL AND METHODS

Fresh samples were collected from Department of Obstetrics and Gynecology inside Al-Mosil hospitals during the period between December 2020 and May 2021. Two studying groups was analyzed: First group, women with uncomplicated, healthy with singleton pregnancies, the group was considered as control group. Second collection comprised females with pregnancies difficult via gestational hypertension at third trimester. According to the Iraq Ministry of Health’s permission, the mother’s informed consent was obtained. All patients of women were delivered by Caesarean section and normal vaginal delivery. Fresh sample of placenta were placed in a labelled clean plastic container contain 10% normal buffer formaline (NBF) solution.6 Fixation, Dehydration, Clearing.

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Infiltration, Embedding, Blocking, Cutting, and Staining were the stages of automated processing. The slides were discolored with H&E stain section examined by the light microscope at magnification 10X, 40X, 100X and examinations focused on histological compound number of syncytial knots, terminal villi, zone of hyalinization, cytotrophoblast cell proliferation in terminal villi, calcification, fibrinoid necrosis. Altogether of the lesions described above were used as typical controls to compare with hypertensive cases. The data was examined using the Statistical Package for the Social Sciences (SPSS) programme form 23, and statistical analysis was performed using independent T-tests with p-value (p ≤ 0.05).

RESULT
The placenta morphometric study (Table 1) revealed that the mean placental weight was 603.76 ± 46.846 in the normal pregnant group and it was 488.68 ± 15.916 in the hypertensive group. The mean placental diameter 18.184 ± 0.2764 in normal and 17.02 ± 0.2828 in hypertensive mothers. The mean placental central thickness 2.68 ± 0.1555 in normal and 2.424 ± 0.2107 in hypertensive mothers.

The insertion of umbilical cord was central in 16 women and eccentric in 8 women and marginal insertion was recoded 1 case in control group. In 18 women, the umbilical cord insertion was eccentric, and in 7 women, it was marginal, as shown in the (Table 1). The mean birth weights for babies were 3077.52 ± 176.109 in the first group and 2691.28 ± 132.75 in hypertensive group. The mean of gestational age was 38.84 ± 0.68 in the control group and it was 36.64 ± 0.56 in the hypertensive cases.

Histological features of placentae in control group (Table 2 and Figure 1) revealed decrease number of terminal villi were 1(4.00%), calcification was 0(0.00%), cytotrophoblast cell proliferation was 1(4.00%), fibrinoid necrosis was 2(8.00%), hyalinization was 0(0.00%), syncytial knots formation was 2(8.00%).

Histological features of placentae in gestational hypertension group (Table 2), (Figures 1 and 2) showed decrease number of terminal villi were obvious 14(56.00%) with significant difference p≤0.05, also calcification was 4(16.00%), moreover, increased cytotrophoblast cell proliferation was 17(67.00%) and statically significant revealed increase in fibrinoid necrosis 19(76.00%), hyalinization was increased 5(20.00%) with p≤0.05. Finally, the number of syncytial knots was increased 14(56.00%) with significant difference p≤0.05.

DISCUSSION
The current study reveals that the placental weights, placental diameter and placental thickness show lower value in hypertensive group (GH) than in normal group. As a result of obstructed utero-placental blood flow, changes in weight and dimension may affect in placental inadequacy. As a result, it has a negative impact on newborn natal weight. These finding agree with studies of other works. In this study it was also found that the birth weight of neonatal in hypertensive group was decreased compare with control group. Pathological alterations discovered in the placenta have a negative impact on foetal weight because the placenta is a mirror that reflects the foetus’ intrauterine state, as a result, examining the placenta throughout pregnancy and parturition can reveal a lot about the child’s and woman’s prenatal well-being. The occurrence of marginal, eccentric umbilical cord attachment in the patient group was greater as compare to control group which correlated with other researches. Different forms of organization of intracotyledonary arteries of placentae of complex gestation

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Controls N=25 (Mean ± SEM)</th>
<th>GH N=25 (Mean ± SEM)</th>
<th>p-value between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placental weight (gm)</td>
<td>603.76 ± 46.846</td>
<td>488.68 ± 15.916</td>
<td>0.003</td>
</tr>
<tr>
<td>Placental diameter</td>
<td>18.184 ± 0.2764</td>
<td>17.02 ± 0.2828</td>
<td>0.003</td>
</tr>
<tr>
<td>Central thickness (cm)</td>
<td>2.68 ± 0.1555</td>
<td>2.424 ± 0.2107</td>
<td>0.004</td>
</tr>
<tr>
<td>Insertion of Umbilical cord</td>
<td>n (%)</td>
<td>n (%)</td>
<td></td>
</tr>
<tr>
<td>Central</td>
<td>16 (64.00)</td>
<td>0(0.00)</td>
<td>0.004</td>
</tr>
<tr>
<td>Eccentric</td>
<td>8(32.00)</td>
<td>18(72.00)</td>
<td>0.003</td>
</tr>
<tr>
<td>Marginal</td>
<td>1(4.00)</td>
<td>7 (28.00)</td>
<td>0.005</td>
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<table>
<thead>
<tr>
<th>Histopathological features</th>
<th>Control N=25 n=%</th>
<th>GH N=25 n=%</th>
<th>p-value between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decrease number of terminal villi</td>
<td>1(4.00)</td>
<td>19(76.00)</td>
<td>0.005</td>
</tr>
<tr>
<td>Calcification</td>
<td>0(0.00)</td>
<td>4(16.00)</td>
<td>0.001</td>
</tr>
<tr>
<td>cytotrophoblast cells proliferation</td>
<td>1(4.00)</td>
<td>17(67.00)</td>
<td>0.003</td>
</tr>
<tr>
<td>Fibrinoid necrosis</td>
<td>2(8.00)</td>
<td>19(76.00)</td>
<td>0.002</td>
</tr>
<tr>
<td>hyalinization</td>
<td>0(0.00)</td>
<td>5(20.00)</td>
<td>0.001</td>
</tr>
<tr>
<td>Numerous syncytial knots</td>
<td>2(8.00)</td>
<td>14(56.00)</td>
<td>0.005</td>
</tr>
</tbody>
</table>
cause marginal cord insertion due to changed foetal blood transfer in the placenta. This vascular configuration preventing equivalent delivery of foetal blood stream within the placenta, putting the women and foetus at risk.\textsuperscript{15}

The mean of pregnancy age at birth of hypertension females was lower than normal women. These findings agree with other research.\textsuperscript{16,17} The aggregation of syncytiotrophoblast nuclei on the superficial of terminal villi is known as syncytial knots. The increased number of the syncytial knots in the placentae complicated by GH cause fetal growth restriction induced by hypoxia.\textsuperscript{18} The Syncytial Knots creation was elevated in this study. These results agree with other researchers.\textsuperscript{19,20}

The fibrinoid necrosis was increased significantly in gestational hypertensive group as compare with normal women.\textsuperscript{21} Fibrinoid necrotic has long been thought to be a sign of an immune response inside tissue of trophoblast. Although it was previously reported to have evolved from a degenerative alteration in villous cytotrophoblast, it remains an enigma because its pathogenesis and importance are unknown.\textsuperscript{22} In the current study, the cytotrophoblast cell proliferation was higher in gestational hypertension women as compare with normal as found in some previous researches.\textsuperscript{23,24} The calcification of the placenta occurs at the conclusion of gestation, nevertheless it may remain a symptom of early ageing in hypertensive women,

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure1.png}
\caption{Section of placenta showing A- Control group, present terminal villi (H&E) 100x, scale bar 200 µm. B- Hypertensive group, present calcification, (H&E) 10 x, Scal bar 200 µm.}
\end{figure}

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure2.png}
\caption{Section of placenta showing A- Hypertensive group, present cytotrophblast (Cy) in terminal villi (H&E) 10x, Scal bar 20 µm. B- Hypertensive group, present syncytial knots (Sy), fibrinoid necrosis (FN) (H&E) 10 x, scale bar 200 µm.}
\end{figure}
reducing the quantity of nourishment and oxygen delivered to the newborn and perhaps worsening the postpartum outcome.\textsuperscript{25} This study recorded increased the calcification significantly in GH group, whereas in the normal women, no apparent calcification processes. This observation is similar to other previous studies.\textsuperscript{14,25} The current results suggested that decreased number of terminal villi in placentae of GH group compared with control.\textsuperscript{26} The decrease number of terminal villi was possibly because the blood capillaries, which initiates villous sprouting in the placental core, had not been established.\textsuperscript{26} The hyalinization area was significant increase in GH women compared with normal women.\textsuperscript{27,28}

**CONCLUSION**

The mother’s placenta showed significant changes in the macroscope and microscope as a result of gestational hypertension. As a consequence of a compromised utero-placental blood stream, morphological aspects of the placenta can produce placental deficit, which can impact the fetus’s development.

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