HISTOLOGICAL CHANGES IN PLACENTA OF PATIENTS WITH PRE ECLAMPSIA

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Abstract

Background: Preeclampsia is a major disease of human reproduction, with 10% of human births being affected, mainly a systemic endothelial disease causing activation of platelets and diffuse ischemic disorders.

Objectives: The study aims to demonstrate the histological changes in placenta of women suffering from hypertensive disease.

Methods: Placental samples were obtained from 15 healthy uncomplicated pregnancies and 35 pregnancies complicated by intrauterine growth restriction due to sever preeclampsia. Samples were prepared and examined by light microscope.

Results: Variable changes have been observed in different patients, including degeneration of most of endothelial cells, degeneration of major trophoblast cells, hyalinization and fibrotic trophoblast cells in some patient sections and fatty infiltration within trophoblast cells in other patient sections.

Conclusion: The primary cause of preeclampsia is a disturbed growth of trophoblast cells with the degeneration of major endothelial cells.

Key words: Preeclampsia, Placenta, histological changes.

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Introduction

The hypertensive disorders of pregnancy complicate about 7-10% of all pregnancies[1]. Pregnancy induced hypertension, which includes preeclampsia, and eclampsia is responsible for 70%, where as chronic hypertension represents 30% of hypertensive disorders in pregnancy. Gestational hypertension (Preeclampsia) is a major disease of human reproduction, with 10% of human births being affected[2].

Preeclampsia is mainly a systemic endothelial disease causing activation of platelets and diffuse ischemic disorders[3,4]. It is characterized by generalized activation of maternal endothelial cells. Oxidative stress of the placenta is considered a key intermediary step, precipitating deportation of apoptotic fragments into the maternal circulation[5,6].

Since studies over the past decade have provided a better understanding of the potential mechanisms responsible for the pathogenesis of preeclampsia, the initiating events in preeclampsia has been postulated to be reduced uteroplacental perfusion as a result of abnormal cytotrophoblast invasion of spiral arterioles. Placental ischemia is thought to lead to widespread activation dysfunction of the maternal vascular endothelium that results in enhanced formation of endothelium and thromboxane[7,8].

The present paper was designed to study the histological changes in placenta of women suffering from hypertensive disease.

Materials and Methods

This work was carried out in the department of Obstetrics and Gynecology, at Al-Kadhiymia Teaching Hospital in Baghdad, for a period of ten months, from 1st August, 2000 to the 1st June 2001. 50 women were enrolled in the study, all were in early labor and sharing the following criteria:

1. All were singleton pregnancies.
2. Their parities were between 0- 4,

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3. All had full term pregnancies, confirmed by known last menstrual period and early ultrasound report.

They were divided into two groups. The first group (normal or control group) consisted of 15 women who had the following additional criteria:

1. No history of medical problems, smoking or drug intake.
2. No history of obstetrical problems.
3. Their newborns had birth weight within the 10th percentile of the individualized birth weight ratio.

The second group (patients group) consisted of 35 women with pregnancies complicated by intrauterine growth restriction (IUGR) and had the following additional criteria:

1. All had history of risk factors of complicated current pregnancy with pre-eclampsia or history chronic hypertension.
2. Examination of these women revealed inadequate fundal growth.
3. They had an ultrasonographic evidence of (IUGR), deviation from an appropriate growth percentile depending on biparietal diameter, head and abdominal circumferences measurements, with amniotic fluid index of less than 10cm.
4. Their newborns had birth weight less than the 10th percentile of the individualized birth rate ratio.

Tissues Preparation

Placental tissue samples were obtained from the outer area of the maternal surface of the placenta and cut into small pieces (2x2x2 mm) and prefixed in 2.5% Gluteraldehyde in phosphate buffer pH(7.2), the specimens were prepared for semi-thin sections of 0.5- 1*u*m, stained with 1% Methylene blue and special stain consists of Azar II and Basic fuchsin[9].

Results

In comparsion with normal women group (control group) (Figures 1a and b) shows variable changes in cells of placenta were observed including these following changes:

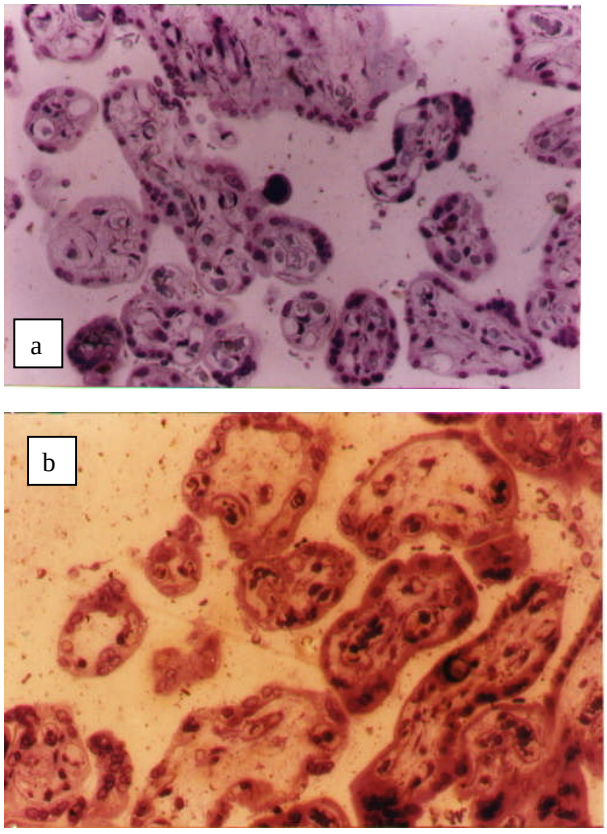

Figure 1a & b: Placental sections from normal women show normal appearance of placental cells.
a: Methylene blue (100X).
b: Basic fuchsin & Azur II (200X).

1. All placental sections of the diseased women have shown an acute type of inflammatory reaction of moderate degree expressed as inflammatory cells infiltration (Figure 2).

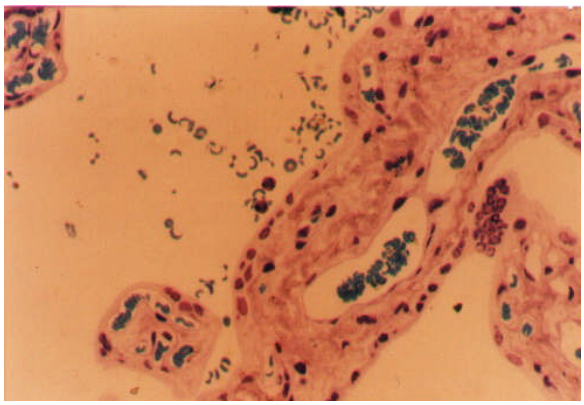

Figure 2: Placental section of hypertensive women shows inflammatory cells.
Basic fucshin – Azur II (100X).

2. All placental sections of preecalmptic women have shown a degeneration of some endothelial cells that reduced the thickness of vessel wall lead to occurrence of hemorrhage (Figure 3).

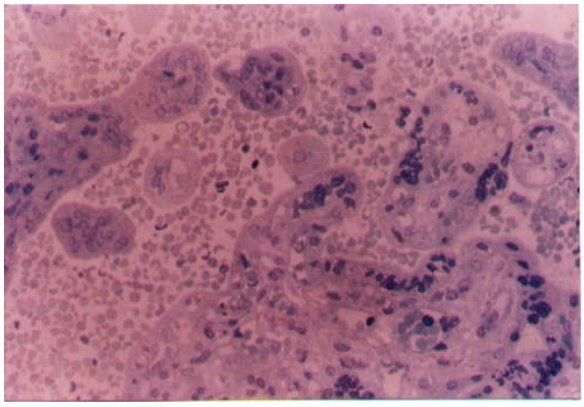

Figure 3: Placental section of hypertensive women shows haemorrage.
Methelene blue (100X)

3. In 70% of patients, fatty infiltration within trophoblast cells was observed (Figure 4).

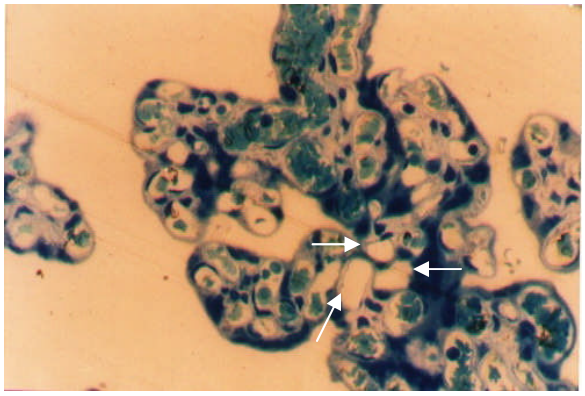

Figure 4: Placental sections of diseased women showing fatty infiltration within trophoblasic cells.
Methylen blue (200X).

4. Placental sections from other women with pre eclampsia, showed a hyalinization and fibrotic trophoblast cells with the degeneration of major trophoblast cells (Figure 5).

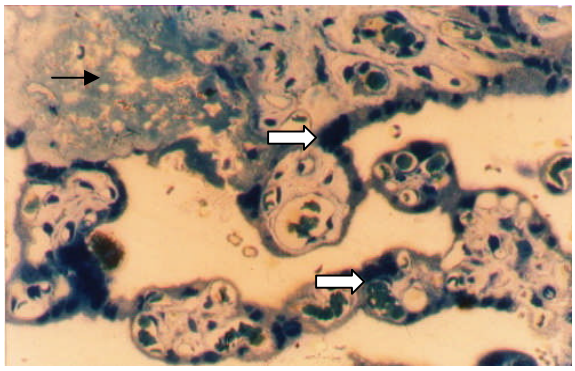

Figure 5: Placental section of hypertensive women showing hyalinization (→), and degeneration of trophoblastic cells (⇨)Methelyn blue (200X)

Discussion

Normal pregnancy is associated with reductions in total vascular resistance and arterial pressure possibly due to enhanced endothelium dependent vascular relaxation and decreased vascular reactivity to vasoconstrictor agonists. These beneficial haemo-dynamic and vascular changes do not occur in women who develop preeclampsia; instead, severe increases in vascular resistance and arterial pressure are observed[10].

Preeclampsia is triggered by various factors which can be immunological, vascular, or abnormalities of haemostasis. These defective placental results in a systemic endothelial disease with vasoconstriction[8].

In our study degeneration in major endothelial cells were observed, which reveled the explanation of abnormalities of haemostasis. These changes was accompanied with the degeneration of trophoblast which considered as the primary cause of preeclampsia[11]. This state is accompanied with the hemorrhage which reflects an absolute or relative placental ischemia due to vascular disease or hypertrophic placenta[12].

Moreover, in addition to these changes a fatty infiltration in the placental sections of some patients while the histological examination of other patients reflects a state of hyalinization, these

changes could be referred to the changes in the decidual cells, since the change in cell composition results in certain disturbance of physiological equilibrium of biological active substances produced by the decidual cells[13].

From this study, we can conclude that the primary cause of preeclampsia is a disturbed growth of trophoblast cells with the degeneration of or endothelial cells.

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