



Original Research Article

Hypertension lead to Histo pathological changes in Placenta of PIH

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Abstract

Background: - Placenta is a vital organ for foetal development, derived from both foetal and maternal tissues and it reflect the status of maternal hypertension.

Aim: The aim of this original research article is to compare the microscopic anatomy of placenta from hypertension associated pregnancies and normal pregnancies.

Method: This study was done in Rama Medical College Kanpur. Placentas from 102 cases of PIH and 100 cases of normotensive pregnant women collected and histological sections studied.

Result: Microscopic study revealed that compare to normotensive women's histological pattern of placenta in PIH women's have higher fibrin deposition, calcification, infarction and chorioamnionitis.

Conclusion: Hypertension during the pregnancy lead to pathological changes in placenta.

Keywords: PIH, Preeclampsia, Eclampsia, Placenta, Pregnancy, Hypertension, fibrin deposition, calcification, infarction and chorioamnionitis.

Introduction

Hypertension, complicating 7% to 15% of all pregnancies, is a leading cause of maternal and foetal morbidity, particularly when elevated blood pressure (BP) is due to preeclampsia, either alone (pure) or "superimposed" on chronic vascular disease [1,2].

The placenta is a remarkable organ. In normal pregnancy its specialized cells (termed cytotrophoblasts) differentiate into various specialized

subpopulations that play pivotal roles in governing fetal growth and development. One cytotrophoblast subset acquires tumor-like properties that allow the cells to invade the decidua and myometrium, a process that attaches the placenta to the uterus. The same subset also adopts a vascular phenotype that allows these fetal cells to breach and subsequently line uterine blood vessels, a process that channels maternal blood to the rest of the placenta. In the pregnancy

complication preeclampsia, which is characterized by the sudden onset of maternal hypertension, proteinuria and edema, cytotrophoblast invasion is shallow and vascular transformation incomplete [3].

Foetal outcome is adversely influenced by pathological changes observed in placenta due to toxic factors released by the placenta and are believed to cause maternal endothelial dysfunction.

Aim and Objective

The aim of the study is to understand the micro anatomy of placenta in hypertension associated pregnancies and to compare the histological findings with normotensive women's placenta.

Material Method

This study was done in Department of Anatomy, Rama Medical College, Kanpur. Two Hundred two placentas collected from the labour room of Rama Hospital, Rama Medical College, Kanpur. One Hundred two placentas of this were from the women diagnosed for PIH and remaining one hundred from normotensive uncomplicated pregnancy.

Inclusion Criteria

- Gestational hypertension Without proteinuria or pathological oedema
- Pre-eclampsia-Hypertensio and proteinuria with or without pathological oedema.
- Eclampsia – Pre-eclampsia complicated with convulsions and / or coma.
- Pre-eclampsia or eclampsia superimposed on chronic hypertension

Exclusion Criteria

- Chronic hypertension
- Essential hypertension
- Chronic renal disease (reno vascular)
- Coarctation of aorta
- Pheochromocytoma
- Thyrotoxicosis
- Connective tissue disease-systemic lupus erythematosus

- Pre-existing Diabetes mellitus(IDDM-Type 1)
- Pre-existing Diabetes mellitus(NIDDM-Type 2)
- Gestational Diabetes Mellitus (GDM)
- Twins Pregnancy

The placenta collected from the labour room of Rama Hospital, Rama Medical College Kanpur, soon after the delivery, the placenta washed in running tap water till it loose the blood to its maximum then this placenta fixed with 10% formalin for one week.

This fixed placenta carried to Department of Anatomy, Rama Medical College for grossing and section cutting.

- Two cm wedge of tissue required for histopathology from
 - a) Maternal & Fetal surface 2 cm from periphery
 - b) Maternal & Fetal surface from centre
- Tissue packed in disposable cassettes with proper labelling.



Excelsior AS™ Tissue Processor

Processing

- Tissue processing done under automated tissue processing machine, the Thermo Scientific™, Excelsior AS™.
- Specimens are held in a specified reagent, usually a fixative, until

The start time is reached. Excelsior AS then process the specimens overnight so that they are ready for the next stage in tissue processing workflow, the following morning, at the

Specified end time. Approximately 13 hrs needed for processing.

Embedding

- Embedding done using automated Tissue-Tek TEC Machine. When the Embedding Module is turned on, either manually or automatically, 4 hours is required for paraffin to Liquefy and reach set temperature.
- Using forceps, place tissue(s) from cassette into base mold over hot plate under paraffin dispenser. If tissues do not sink to bottom of mold, use tampers or forceps to lightly push tissue(s) down (into proper position) on the bottom of base mold.
- Move the base mold to the cold spot. Bottom of the base mold rapidly cools and a thin layer of paraffin solidifies



Automated Tissue Embedding Machine

Freezing

- Paraffin block now store at 0°C 2hrs 30 min



Tissue Freezing Machine

Trimming

- Trimming is usually done at thicknesses between 10 and 30 μm .



Microtome for Section Cutting

Section Cutting

- Use the adjusting knob on the right side of the microtome front panel to select the 5 micron section thickness.
- Start sectioning by rotating the hand wheel evenly in clockwise direction.
- We get ribbon of paraffin with embedded tissue.



Hot water Oven

Hot Water Bath

- Picked the ribbon gently to hot water bath.
- Apply the albumin to glass slide.
- Insert the slide perpendicular in hot water bath and attach a single floating film of paraffin at midway of slide.
- Perform this action fast, otherwise albumin get wash



Slide warmer

Deparaffinization

- This is done by placing the slide over slide warming table at 60°C for 20 min.
- Immerse slide in xylene for 10 minutes. Repeat once in fresh xylene for 10 minutes.

- If needed, repeat a third time in fresh xylene for 10 minutes.



Automated Tissue Staining Machine

Staining

- This is done by automated tissue strainer Tissue-Tek DRS.
- Open the lower door only and place one or two baskets of slides in position S1 then close the lower door and press METHOD and choose the correct protocol using the UP

or DOWN arrow keys. Press SELECT then Press START.

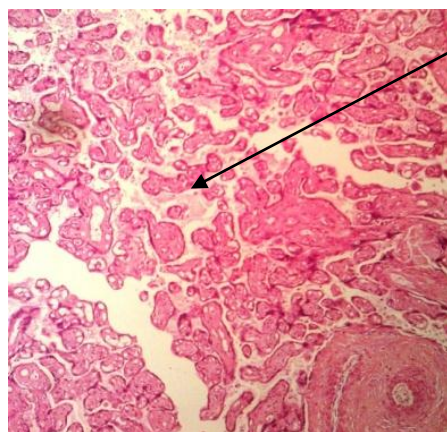
- Approximately 1 hr required for H&E stain.

Cover Slip

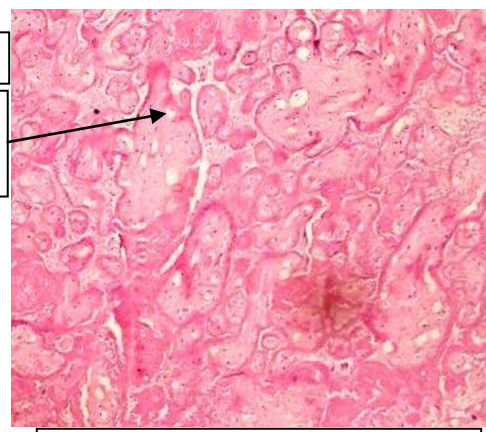
Place a drop of Permount(Xylene) on the slide using a glass rod taking care to leave no bubbles and angle the coverslip and let fall gently onto the slide, allow the Permount to spread beneath the coverslip, covering all the tissue and dry overnight in the hood.

Observation

All H & E stained slide observed at 100X in binocular microscope and focused specially for fibrin deposition, calcification, infarction and chorioamnionitis in cases and controls.

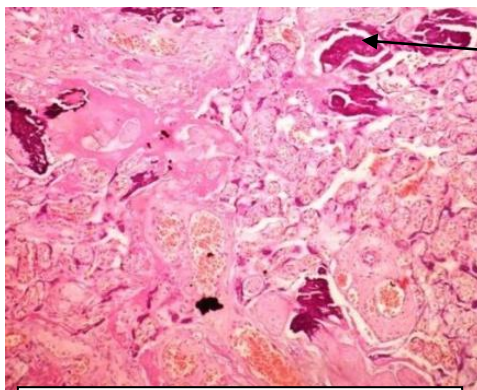


**Maternal side of Placenta at centre
H & E Stain at 100 X**

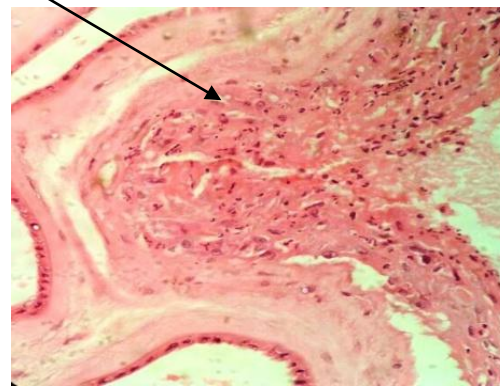


**Maternal side of Placenta at centre
H & E Stain at 100 X**

Fibrin
Coagulative
Necrosis



**Maternal side of Placenta at centre
H & E Stain at 100 X**



**Maternal side of Placenta at centre
H & E Stain at 100 X**

Chorioamnionitis

Calcificatio

Result

Histological observation in PIH

CASES	FIBRIN	CALCIFICATION	INFARCTION	CHORIOAMNIONITIS
102	74	55	39	22

Histological observation in Control

CASES	FIBRIN	CHORIOAMNIONITIS	CALCIFICATION	INFARCTION
100	41	32	24	4

Discussion

It has been found that in pregnancy associated with hypertension. Fibrin deposition is higher followed by calcification, infarction and chorioamnionitis and also in control; fibrin deposition is higher but followed by chorioamnionitis, calcification and infarction.

It has also found that the frequency of pathological changes is higher in pregnancy associated with hypertension.

Estellés Amparo et al found in the study over Abnormal Expression of Type 1 Plasminogen Activator Inhibitor and Tissue Factor in Severe Preeclampsia and said plasminogen activator inhibitor (PAI-1) levels are significantly increased in plasma and placenta from pregnant women with preeclampsia compared to normal pregnant women which result in higher fibrin deposition [4].

Kanfer A et al said Placental antifibrinolytic potential is increased in pregnancy-induced hypertension and preeclampsia. This change, and the association of the highest plasminogen activator inhibitor (PAI-2) placental concentrations with the lowest concentrations of thrombomodulin, may contribute to the prethrombotic state and to the excessive placental perivillous fibrin deposition observed in these situations [5].

It has also been observed in many studies over placental calcification that placental calcification is associated with pregnancy-induced hypertension [6,7,8].

Brosens I and Renaer M said, placental infarction is apparently a direct result of the occlusive hypertensive lesions in the spiral arteries, it is also to be considered as the ultimate evidence of failure of adequate placentation [9].

Martius, J. & Eschenbach, D.A found that the role of bacterial vaginosis as a cause of amniotic fluid infection, chorioamnionitis and prematurity [10].

Conclusion

PIH positively Correlated with fibrin deposition, calcification and infarction.

Early onset of Preeclampsia causes more fibrin deposition, calcification and infarction.

Frequency of fibrin deposition is higher in PIH, followed by calcification and infarction.

PIH do not Correlated with chorioamnionitis.

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