

## HISTOPATHOLOGY OF UTEROPLACENTAL BED IN NORMAL PREGNANCY AND IN PREGNANCY INDUCED HYPERTENSION AND ITS CORRELATION WITH FOETAL OUTCOME.

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### SUMMARY

Placental Central Bed Biopsy was taken from 50 patients at the time of Caesarean section. Out of 50 patients, 25 were normotensive and 25 had Pregnancy Induced Hypertension (PIH). Histologically the biopsy was suitable for study in 60% (30 out of 50 cases). Spiral vessel segment was seen in 80% (24 out of 30 cases). Vascular changes in the form of medial hypertrophy and subintimal oedema were observed in 30.76% (4 out of 13) pregnancy induced hypertension.

### INTRODUCTION

Human pregnancy requires the most intimate form of hemochorial placentation where maternal blood is brought into direct contact with the trophoblast of the fetal placenta by an arterial sinusoidal venous system in continuity.

Until, relatively recently morphological studies of human placentation in normal and abnormal pregnancy were based on the delivered placenta and on hysterectomy specimens or postmortem. The maternal blood supply to intervillous space is markedly reduced in hypertension due to any cause (Brown and Veall 1953).

The maternal blood reaches the intervillous space of placenta through

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100-150 spiral arteries (Wilkin 1965, Brosens and Dixon 1966, Boyd et al 1970) and these vessels are of prime concern in determining pathology of pregnancy induced hypertension.

Histological studies of human hemochorial placentation, must include material deep in the placental site, as it is there that vascular changes may occur.

Dixon and Robertson (1958) introduced the term "Uteroplacental bed biopsy" which included basal decidua as well as myometrium at the centre of implantations of placenta because vascular changes are maximum at the centre, being the site of implantation.

#### **MATERIAL AND METHODS**

Fifty women admitted in Obst. & Gynaecological department of Dayanand Medical College & Hospital, Ludhiana (Punjab) who had caesarean section under spinal or general anaesthesia were studied. Twenty five women were controls who were normotensive and 25 had PIH.

A detailed history, general physical examination and systemic examination was done. Routine haemoglobin, urine examination, blood grouping & Rh. factor, RFT and fundus examination were done in all cases. Ultrasound examination was done as & when required.

**SPECIAL INVESTIGATIONS :**  
Placental central bed biopsy.

**PLACENTAL CENTRAL BED BIOPSY :**

Placental central bed biopsy was taken in all patients at the time of

caesarean section after extraction of placenta under direct visualisation. After delivery of baby, the true centre of placenta, not the insertion of cord, was digitally marked by the assistant and placenta peeled away from its bed by the operator. Placental site was some what depressed, focally disrupted and friable area with recognition of sheared uteroplacental vessel including dark red thrombi. Then placental bed biopsy from centre of placental bed was taken with the help of cervical punch biopsy forceps, measuring approximately 1.5 cm. in length, 1 cm. in breadth and 0.5 cm. in depth. A strip from the periphery of placental bed was also taken just to compare the findings with those at the centre.

The tissues were examined histopathologically for

1. Presence of giant cells
2. Musculoelastic tissue in vessel wall
3. Fibrinoid material in vessel wall.
4. Foam cells i.e. acute atherosclerosis.
5. Hyperplastic changes in vessel wall

#### **OBSERVATIONS**

Of the 50 biopsies taken at the time of caesarean section, 30(60%) biopsies were considered suitable for inclusion in the study from the central area of placental bed which showed the presence of spiral arteries in the decidua and in the myometrium and the presence of interstitial giant cells in adequate number. Out of 30 biopsies obtained 17 (56.66%) belonged to normal pregnancies delivered at term by caesarean section and

13(43.33%) to pregnancies complicated by pregnancy induced hypertension (Table I).

Of the 17 physiological normal pregnancies, 15(88.23%) showed normal physiological changes in decidual and myometrical segments of spiral vessels, i.e. replacement of part of musculoelastic tissue of vessel wall by fibrinoid material and 2(11.76%) showed absence of fibrinoid material,

thus making physiological changes inadequate, though, spiral vessel segment was seen in 12 cases and only vessel wall in 5 patients (Table II).

In the study group, out of 13 patients with pregnancy induced hypertension, 12(92.3%) had no or inadequate physiological changes in the myometrial segments of spiral

**TABLE I**  
**HISTOPATHOLOGY OF UTEROPLACENTAL BED**

Histopathology of placental bed.	No. of cases.	PIH				Control				
		Absent		Present		Absent		Present		
		No.	%	No.	%	No.	%	No.	%	
Giant cells.	25	12	48%	13	52%	25	08	32%	17	68%
Fibrinoid material.	13	12	92.3%	01	7.69%	17	02	11.76%	15	88.23%
Spiral vessel segment.	13	01	7.69%	12	92.3%	17	05	29.4%	12	70.58%

**TABLE II**  
**CLASSIFICATION OF PHYSIOLOGICAL CHANGES IN PLACENTAL BED VESSELS**

Group	Total No. of cases.	Normal physiological changes.		Inadequate physiological changes.	
		No.	%	No.	%
PIH	13	01	7.69%	12	92.30%
Control.	17	15	88.23%	02	11.76%

TABLE III  
SPIRAL VESSEL SEGMENT STUDY

Sr. No.	Spiral vessel segment.	PIH		Normal	
		No.	%	No.	%
1.	Lumen.	12	66.66%	12	
	a) Normal.	8	66.66%	4	33.33%
	b) Wide. - -	8	66.		66%
	c) Narrow.	4	33.33%	-	-
2.	Medial hypertrophy.	02	16.66%	-	-
3.	Subintimal odema.	02	16.66%	-	-
4.	Acute atherosis	-	-	-	-

arteries and one case showed only vessel wall with fibrinoid material. 2(15.38%) patients showed medial hypertrophy of vessel wall towards occlusion of vessels and 2(15.38%) showed subintimal oedema. None of 13 patients showed acute atherosis (Table III).

#### DISCUSSION

The success rate was 60% (30 out of 50) in the present study. Brosens et al (1967) reported success rate of 70% while McFadyen et al (1986) had success rate of 43.11% (47 out of 109) cases and Frusca et al (1989) of 41.44% (46 out of 111 cases).

#### HISTOPATHOLOGY OF PLACENTAL BED BIOPSY.

##### Spiral vessel segment.

In the present study, spiral vessel

segment was seen in 80% cases (24 out 30). In remaining 6 cases, only vessel wall was visualised. However, Brosens (1964) and McFadyen et al (1986) visualised spiral vessel segment in 100% cases included in their studies while Gerretson et al (1981) observed spiral vessel segment only in 73 out of 175 cases (41.17%)

##### Physiological changes in spiral vessels.

In the study group, 12 patients (92.3%) out of 13 showed inadequate physiological changes limited to myometrial segments of spiral arteries and one (7.69%) showed no physiological changes. In the control group 15 out of 17 (88.23%) showed normal physiological changes in decidual and myometrial segments of spiral vessels and 2 (11.76%) showed inadequate physiological

**TABLE V**  
**COMPARISON OF VASCULAR CHANGES IN SPIRAL VESSELS**

Author	Year	No. of cases	Medical Hypertrophy & subintimal oedema				
			Present		Absent		
			No.	%	No.	%	
Dixon H.G. & Robertson W.P.	1958						
Study group.		2	2	100	-	-	
Control group.		15	-	-	15	100	
Present study	1990-1991						
Study group.		13	4	30.76	9	69.23	
Control group		17	-	-	17	100	

change. Frusca et al (1989) showed inadequate physiological changes in 100% whereas Khong et al (1986) had them in 10% (Table IV).

#### **Vascular changes in spiral vessels.**

In the present study, out of 4 patients with vascular changes in the form of medial hypertrophy and subintimal oedema, 3 (75%) had blood pressure of more than 160/100 mmHg. grade I hypertensive fundal (eye) changes and proteinuria (++) while all the 4(100) had pedal oedema and serum uric acid suggesting a significant correlation of blood pressure, pedal oedema, fundus

changes and proteinurea with vascular changes in spiral vessels (Table V).

#### **Acute Atherosclerosis**

None of the patients in the study group in present study showed lesion of acute atherosclerosis in the placental bed vessels.

#### **Fetal outcome**

In the present study, all patients in both the groups had live births. Fetal distress was observed in 56% cases in study group and 28% cases in control group. All the 4 cases with vascular changes had fetal distress indicating

TABLE IV  
COMPARISON OF PHYSIOLOGICAL CHANGES

Author	Year	Total		Pregnancy Induced Hypertension						Control						
		No. of cases.	Total cases.	Physiological changes		No Physiological changes		Inadequate physiological change.		Total cases.	Physiological changes		No Physiological change		Inadequate physiological changes	
				No.	%	No.	%	No.	%		No.	%	No.	%	No.	%
Gerretson et al	1981	53	30	1	3.33	29	96.66	-	-	23	22	95.65	1	4.34	-	-
Khong T.T. et al	1986	64	46	18	39.13	23	50.00	5	10.86	18	18	100.00	-	-	-	-
Frusca et al	1989	38	24	-	-	-	-	24	100.00	14	13	92.85	-	-	1	7.14
Present study	1990-91	30	13	1	7.69	-	-	12	92.30	17	15	88.23	2	11.76	-	-



that hypertensive changes in the vessels led to occlusion of vessels leading to fetal distress.

Mean birth weight was 3036 gms. in the group with normal physiological changes 2675 gms. in the group with inadequate physiological changes and 2312 gms. in the group with vascular changes. Only 2 babies in study group were small for gestational age, the remaining being appropriate for gestational age.

### CONCLUSION

In conclusion, in normal pregnancy, physiological changes were observed in spiral vessels in decidua as well as myometrium leading to dilatation of vessels in order to cope up with increased demands in pregnancy.

In pregnancy induced hypertension inadequate physiological changes limited to myometrial segments were observed. In 30.76% cases, medial hypertrophy and subintimal oedema in the vessels were observed leading to

occlusion of vessels and fetal distress. There were only 2 babies who were small for gestational age in study group, the remaining being appropriate for gestational age.

### REFERENCES

1. Boyd J.D. and Hamilton W.J. *The human placenta*, Cambridge, 1970
2. Brosens I : *J. Obst. Gynec. Br Commonwealth* 7: 222, 1964.
3. Brosens I and Dixon H.G. *J. Obst. Gynec. Br. Commonwealth.* 73: 357, 1966
4. Brosens I; Robertson W.B. and Dixon H.G. : *J. Path. Bact* 93: 569, 1967
5. Browne J.C.M and Veall N. *J. Obst. Gynec. Br. Commonwealth* 60: 141, 1953
6. Dixon H.C.; Robertson W.B. : *J. Obst. Gynec. Br. Commonwealth* 65: 803, 1958
7. Frusca T.; Morassi L.; Pecoralli S.; Grigolato P. and Gastaldi A. *Br. J. Obst. & Gynec* 96: 835, 1989
8. Gerretson G, Huisjes H.J. and Elema J.D. *Br. J. Obst. Gyn* 88: 876 1981
9. Khong T.Y.; De Wolf F. Robertson W.B., Brosens I. *Br. J. Obst. Gynec* 93: 1040, 1986.
10. Mc Fadyen I.R., Price A.B., and Garsson R.T.; *Br. J. Obst. & Gyn.* 93: 476, 1986
11. Wilkin P.: *Pathologic duplacentia Paris Marson*, P240, 1965