

## ULTRASONOGRAPHIC AND HISTOLOGICAL STUDY OF PLACENTA IN ABNORMAL PREGNANCY CASES

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### SUMMARY

The present study was a correlative study of sonography and Histopathology of placenta on 49 cases of abnormal (pre-eclampsia, I.U.G.R., prolonged pregnancy, diabetic pregnancy & Rh. incompatibility) & 20 normal pregnancy cases. The mean gestational age for appearance of sonographic grade I, II & III was significantly earlier in IUGR and Grade I & II in pre-eclampsia while it is delayed in diabetes. The mean placental thickness significantly decreased in all the grades of pre-eclampsia and I.U.G.R. while it increased in diabetes and Rh incompatibility. The mean biparietal diameter & femur length is significantly less in all the grades in I.U.G.R. & pre-eclampsia while these parameters increased in diabetes in grade I. Histopathological changes showed increased incidence of syncytial knots, cytotrophoblast cell proliferation, fibrinoid necrosis, stromal fibrosis in grade II and significantly increased in grade III placenta of pre-eclampsia & I.U.G.R. while obliterative endarteritis and hypovascularity was more marked in pre-eclampsia and I.U.G.R. resulting in accelerated maturation and placental thinning. Placenta of diabetics and Rh incompatibility patients showed villour oedema, immaturity and hofbauer cells which cause increase in thickness and delay in maturational grades.

### INTRODUCTION

With the advent of ultrasonography antenatal evaluation of placenta have become

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Accepted for Publication on Nov' 96.*

essential in high risk pregnancy as fetal problems and neonatal outcome depend upon status, growth and abnormalities of placenta. The present work comprises of correlative study of ultrasonography of placenta as well as fetal growth (biparietal diameter (BPD) & femur length (FL) measurement) and placental histopathology in case of preeclampsia, intrauterine growth retardation (IUGR), prolonged pregnancy, diabetes and Rh incompatibility.

#### **MATERIAL AND METHOD**

The placental study was carried on 20 normal and 49 high risk pregnancy cases which include 20 pre-eclampsia, 10 IUGR, 10 prolonged pregnancy, 3 diabetic pregnancy and 6 cases of Rh incompatibility. Serial placental sonography (measurement of thickness, maturational grades according to Grannum classification) as well as fetal BPD and F.L. measurements were done from 28 wks onwards till term by Siemens imager Model 2380 having transducer of 3.5 MHz frequency.

The placenta was subjected to histopathological examination. Haemotoxylin and eosin staining was done and 200 villi studied in relation to each parameter (Syncytial knots, cytotrophoblastic cells, vasculo-syncytial membrane, fibrinoid necrosis, basement membrane, stromal villous vessels, foetal stem artery and villous immaturity, calcification and obliterative endarteritis).

#### **OBSERVATION AND DISCUSSION**

Placental maturational grade changes were studied in relation to gestational age, placental thickness, BPD & FL at different periods of gestation in normal and abnor-

mal pregnancy cases and correlated with histological findings of placenta.

#### **Placental Grade and Gestational Age (G.A.)**

The mean G.A. for appearance of placental grade I,II,III was significantly earlier in IUGR cases as compared to normal pregnancy while it was only significantly earlier in grade I and II in pre-eclampsia (Table I, Fig. 1). 80% cases of IUGR and 70% of pre-ecl. had grade III changes prior to delivery as compared to 40% cases of normal pregnancy, Grannum et al (1979), Kazzi et al (1983) reported grade III placental changes in 63% cases of small for gestational age births. Accelerated placental maturation in pre-eclampsia has also been reported by Carrol - (1980) Grannum and Hobbins (1983), Gast and Ott (1983), Quinlan & Cruz (1982). However in cases of prolonged pregnancy, gestational age for appearance of different placental grades was similar to that of normal pregnancy while 3 cases of diabetes showed delay in appearance of grade I changes only and none had grade III changes even at 38 weeks of gestation. Similar findings have been reported by Grannum & Hobbins (1983). In Rh incompatibility appearance of different grade changes were almost similar to normal pregnancy (in 3 out of 6 cases babies were Rh negative and there was no sensitization).

#### **Placental Thickness and Gestational Age**

The placental thickness in pre-eclampsia & IUGR for the same gestational period decreases significantly

**Table I**  
**MEAN GESTATIONAL AGE & PLACENTA THICKNESS**  
**IN DIFFERENT MATURATIONAL GRADES**

Group	Grade I		Grade II		Grade III				
	nM. Gest. (wks)	AgeM. Pl. Thick (mm)	nM. Gest. (wks)	AgeM. Pl. Thick. (mm)	n M. Gest. (wks)	AgeM. Pl. Thick. (mm)			
Normal Pregnancy	19	31.2+1.9 <sup>†</sup>	49.7+5.4	19	35.6+1.6	40.0+3.4	08	37.5+1.2	34.2+3.6
Ab. Preg.									
Pre-Eclampsia	17	29.8+1.4 <sup>#</sup>	43.3+5.6 <sup>#</sup>	17	34.5+2.0 <sup>NS</sup>	36.5+5.0 <sup>#</sup>	14	36.9+1.2 <sup>NS</sup>	32.7+6.2 <sup>NS</sup>
I.U.G.R.	10	29.8+1.4 <sup>#</sup>	41.8+4.2 <sup>**</sup>	10	33.0+0.9 <sup>**</sup>	35.1+5.3 <sup>#</sup>	08	34.5+0.5 <sup>**</sup>	31.1+5.3 <sup>NS</sup>
Prolong Pregnancy	07	31.8+2. <sup>NS</sup>	47.0+4.4	07	35.8+2.2 <sup>NS</sup>	42.1+5.5 <sup>NS</sup>	07	38.0+1.0	33.1+3.1 <sup>NS</sup>
Pregnancy with Diab.	03	34.3	65.0	01	36.0	56.0	-	-	-
Rh Incomp	06	31.5+1.5	60.5+4.7 <sup>NS</sup>	06	35.8+1.3 <sup>NS</sup>	57.8+5.2	02	37.0+0.0	47.0

P (0.05<sup>#</sup> = Significant P 0.01<sup>\*\*</sup> = Highly Significant n=No. of cases)

as compared to normal pregnancy (Table II). The observations of Grannum & Hobbins (1983) were similar to ours who also reported that these two conditions have thin placenta. In cases of prolonged pregnancy the thickness variation is similar to normal pregnancy while there was an increase in thickness in cases of diabetes and significant increase was noted in cases of Rh incompatibility.

#### Placental Thickness in Different Placental Grades

The placental thickness in Grade I, II and III was found to be significantly less in pre-eclampsia and IUGR cases as compared to normal pregnancy while in IUGR cases decrease in Grade I was highly significant showing that placental thinning starts earlier, in one case placental thickness was 23 mm in grade III placenta of 35 wks

**Table II**  
**MEAN PLACENTAL THICKNESS IN DIFFERENT PERIODS OF GESTATION**

Gest. age	n	28-32 wks	n	32-36 wks	n	after 36 wks
		M.Pl.thickness (mm)		M.Pl. thickness (mm)		M.Pl.thickness (mm)
Normal Pregnancy	15	51.86±5.7	15	43.7±4.5	20	37.4±5.5
Ab.Preg.						
Pre-Eclampsia	16	44.25±5.2**	16	38.5±6.1*	20	32.7*±6.5
I.U.G.R.	09	41.3±9.1*	09	37.7±5.5	10	33.1±6.2
Prolong Pregnancy	07	49.6±3.9	07	34.5±4.6	10	34.7 <sup>NS</sup> ±3.7 <sup>NS</sup>
Pregnancy with Diab.	03	67.0	03	65.0	03	61.0
Rh Incomp	06	60.5±5.26*	06	59.3±5.67*	06	56.17±7.94*

n+ = Serial scan cases

n= Serial + Single Scan cases

P < 0.05\* = Significant

P < 0.01\*\* = Highly Significant

and this patient had IUD. (Fig. 2) However in cases of prolonged pregnancy (Fig. 3) thickness of different grades did not show any deviation from normal while there was increase in thickness in all the grades in cases of diabetes (Fig. 4) and Rh incompatibility (Fig. 5). Similar observation have been reported by Grannum et al (1979) and Gottesfeld (1984). The increase may be due to

villous oedema (Table I) which is present in all the cases of diabetes while 4 out of 6 cases of Rh incompatibility had villous oedema.

#### **Placental Grade and Biparietal Diameter & Femur Length**

The mean BPD & femur length is significantly less than normal in all the corresponding grades in IUGR while in grade I & II only in pre-eclampsia

cases. This may be due to accelerated placental maturation as reported by Fischer et al (1976), Grannum et al (1979) and Kazzi et al (1983) in I.U.G.R. & by Carrol (1980) and Grannum and Hobbin (1983) in preeclampsia. The values in cases of prolonged pregnancy and Rh incompatibility were almost similar to normal values. However, there is increase in these parameters in pregnancy with diabetes in grade I placenta only. The BPD values for normal

as well as complicated pregnancies given by Grannum et al (1979) was 76-96 mm for grade I, 81-98 for grade II and 87-95 mm for grade III placenta. They did not find any meaningful co-relation between placental grades.

**Histological Abnormalities and Sonographic Placental Grading in Normal and Abnormal Pregnancies**

There is an increase in syncytial knot count, cytotrophoblastic cell proliferation,

**Table III**  
**BPD & F.L. INDIFFERENT PLACENTAL GRADES IN**  
**NORMAL & ABNORMAL PREGNANCY**

Group	Grade I			Grade II			Grade III		
	n	B.P.D. (mm)	F.L. (mm)	n	B.P.D. (mm)	F.L. (mm)	n	B.P.D. (mm)	F.L. (mm)
Normal pregnancy	19	80.6±5.8	61.0±4.9	20	89.5±4.7	70.0±2.9	08	92.2±1.2	73.3±3.1
Ab. preg.									
Pre-Ecl.	17	77.2±3.4 <sup>#</sup>	58.0±3.2 <sup>#</sup>	17	87.6±3.4 <sup>#</sup>	67.7±3.8 <sup>#</sup>	08	92.0±3.9 <sup>NS</sup>	72.3±3.1 <sup>NS</sup>
I.U.G.R.	10	77.6±3.1 <sup>#</sup>	57.4±3.3 <sup>##</sup>	10	84.5±1.8 <sup>##</sup>	65.0±2.3 <sup>##</sup>	08	87.7±0.9 <sup>##</sup>	68.8±0.9 <sup>##</sup>
Prolong pregnancy	07	81.9±4.8 <sup>NS</sup>	62.3±5.7 <sup>##</sup>	07	89.8±3.1 <sup>NS</sup>	70.2±3.6 <sup>NS</sup>	07	93.2±0.9 <sup>NS</sup>	73.2±0.8 <sup>NS</sup>
Pregnancy with Diab.	03	91.1	71.6	01	90.0	70.0	-	-	-
Rh Incomp	06	82.0±2.3	62.0±2.31	06	89.8±1.9	70.8±2.48	02	92.0	73.5

NS = Not Significant

P < 0.05# = Significant P<0.01## = Highly Significant

Table IV

## HISTOLOGICAL ABNORMALITIES &amp; SONOGRAPHIC PLACENTAL GRADING IN NORMAL PREGNANCY, PRE-ECLAMPSIA &amp; IUGR

Histo-path. Features	N. preg. n = 20		Pre-Eclamp. n = 20		I.U.G.R. n = 20	
	Gr. II	Gr. III	Gr. II	Gr. III	Gr. II	Gr. III
	n %	n %	n %	n %	n %	n %
	12	*8	6	14	2	8
Syn. Knots						
10-30%	11(91.7)	6(75)	3(50.0)	1(7.2)	-	2(67)
30-60%	1(8.3)	2(25)	3(50.0)	10(71.4)*	2(100)	6(75.9)*
above 60%	-	-	-	3(21.4)*	-	1(12.5)*
Cyt. Bl. CL						
0-20%	9(75.0)	5(62.5)	-	-	-	2(25)
20-40%	3(25.0)	3(37.5)	5(83.3)	6(42.8)	-	-
above 40%	-	-	1(16.7)	8(57.2)*	2(100)	6(75) **
Vas. Syn. Memb.						
0-5%	12(100)	6(75)	5(83.3)	13(92.8)	1(50.0)	6(75) **
above 5%	-	2(25)*	1(6.7)	1(7.1)	1(50.)	2(25)
Fib. Necro						
0-3%	12(100)	5(62.5)	3(50)	7(50)	-	1(12.5)
above 3%	-	3(37.5)*	-	4(28.6)*	1(50)	6(75)
B.M. Thick						
0-3%	12(100)	5(62.5)	2(50)	7(50)	-	1(12.5)
above 3%	-	-	-	4(28.6)*	1(50)	6(75)
ST. Fib.						
0-3%	12(100)	6(75)	6(67)	4(28.6)	-	1(12.5)
above 3%	-	2(25)*	2(33.3)	10(71.4)*	2(100)	7(87.5)*
Vill. Oed.	-	-	1	-	-	-
Hofbauer cells	1	1	-	1	-	-
Hypo-vascular	-	2(25)	-	2(14.2)	1(50)	5(62.5)
Obl. End	-	-	3(50.0)	7(50)	1(50)	4(50)
Immaturity	-	-	-	-	-	-
Calcification	5(41.7)	7(87.5)*	2(33.3)	10(71.4)*	1(50)	7(87.5)

NS = Not Significant P &lt; 0.05\* = Significant

P &lt; 0.01\*\* = Highly Significant

**Table V**  
**HISTOLOGICAL ABNORMALITIES & SONOGRAPHIC**  
**PLACENTAL GRADING IN PROLONG PREGNANCY,**  
**DIABETES AND RH INCOMPATIBILITY**

Histopath Features	Prol. Preg. n=10		Diabetes n=3		Rh. Incomp. n=6	
	Gr. II	Gr. III	Gr. I I	Gr. III	Gr. II	Gr. III
	n % 3	n % 7	n % 2	n % 1	n % 4	n % 2
Syn.Knots						
10-30%	2 (67)	1 (14.3)	-	-	-	-
30-60%	1 (33.3)	4 (57.1)*	2 (100)	1 (100)	4 (100)	2 (100)
above 60%	-	2(28.6)*	-	-	-	-
Cyt. Bl. CL.						
0-20%	1 (33.3)	3 (42.9)	-	-	-	-
20-40%	2 (67.0)	4 (57.1)*	1 (50)	1 (100)	4 (100)*	2 (100)
above 40%	-	-	-	1 (100)	-	-
Vas. Syn. Memb.						
0-5%	3 (100)	6 (85.7)	2 (100)	-	1 (25)	-
above 5%	-	1 (14.3)	-	1 (100)	3 (75)	2 (100)
Fib. Necro						
0-3%	2 (67)	3 (42.9)	-	-	-	-
above 3%	1 (33.3)	4 (57.1)NS	2 (100)	1 (100)	4 (100)**	2 (100)
B.M. Thick.						
0-3%	3 (100)	6 (85.7)	1 (50)	-	2 (50)	-
above 3%	-	1(14.3)	1 (50)	1 (100)	2 (50)	2 (100)
ST. Fib.						
0-3%	2 (67)	1 (14.3)	1 (50)	1 (100)	2 (50)	1 (50)
above 3%	1 (33)	6 (85.7)*	-	-	2 (50)	1 (50)
Vill. Ocd.	-	-	2 (100)	1 (100)	4 (400)**	-
Hofbauer cells	-	-	1 (50)	1 (100)	3 (75)**	-
Hypo-vascular	-	4(57.1)	1 (50)	1 (100)	2 (50)	1 (50)
Obl. End.	-	1 ( )	-	-	-	-
Immaturity	-	-	1(50)	1(100)	2 (50)	1 (50)
Calcification	3(100)	5 (71.4)	2(100)	1 (100)	1 (25)	1 (50)

NS = Not Significant      P < 0.05\* = Significant

P < 0.01\*\* = Highly Significant

fibrinoid necrosis, stromal fibrosis, villous hypovascularity in grade II placenta of pre-eclampsia, IUGR and prolonged pregnancy as compared to grade II & grade III normal placenta while incidence of obliterative endarteritis & hypovascularity was more marked in cases of pre-eclampsia & IUGR only. The grade III placenta of these condition showed significant increase in syncytial knot count, cytotrophoblastic cell proliferation, and fibrosis, whereas incidence of fibrinoid necrosis was only significantly increased in IUGR cases. Basement membrane thickening was significantly increased in pre-eclampsia & highly significantly in IUGR. Obliterative endarteritis and hypovascularity was most marked in IUGR cases. Placenta in diabetes and Rh incompatibility showed excessive fibrinoid necrosis while villous oedema was present in all cases of diabetes resulting in increase in thickness. Hofbauer cells, hypovascularity, villous immaturity was seen more in cases of diabetes. Villous immaturity was feature with Rh positive babies only. Grannum et al (1979) reported delay in the maturational process of placenta due to oedema and hyperplasia while Spirt et al (1979) observed increase in thickness upto 50 mm due to villous oedema in moderate and severe type of Rh immunization.

Increased syncytial knot count shows ageing while increase in cytotrophoblastic cell count suggests uteroplacental ischaemia (Wigglesworth, 1962; Fox 1983) and syncytial damage (Fox 1983). Excessive fibrinoid necrosis in IUGR is believed to be due to an immunological reaction

secondary to ageing changes in villi (Fox 1983). Basement membrane thickening is believed to be a response to utero-placental ischaemia (Fox 1983) while stromal fibrosis is considered to be a morphological hall mark of a reduced foetal villous perfusion and hypovascularity and obliterative endarteritis of a further reduced vascular supply.

### CONCLUSION

The study concludes that sonographic placental changes in terms of thickness with advanced maturational grades correspond to histological features suggestive of villous ageing, uteroplacental ischaemia and hampered foetal villous perfusion thus adversely affecting growth of foetus.

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