PLACENTAL HISTOPATHOLOGY WITH I.U.G.R.

By
S. S. Mehendale, Vasanti Lele and P. V. Godbole

SUMMARY

Disturbed placental function secondary to existing pathology in the maternal organism, foetus or the placenta itself affects the foetal growth. Pregnancy induced hypertension, anaemia during pregnancy and viral or bacterial infection during pregnancy lead to metamorphic changes in the placenta in the form of cytotrophoblastic hyperplasia thickening of the basement membrane, placental angiopathies, sclerosis or placentitis leading to I.U.G.R.

Of the 100 placentae studied, 37% cases had some infection during pregnancy. The placentae showed lymphocytic infiltration, cytotrophoblastic hyperplasia and basement membrane thickening. In 43% anaemic cases syncytial knots were present. In 18% cases of PIH, avascular villi and thickening of the basement membrane were the prominent features.

Any insult of the placenta during the developmental stage affects the DNA synthesis which in turn affects the placental transfer.

Introduction

Proper foetal growth and metabolism depends on the adequate exchange across the placenta. A well nourished newborn is the best evidence of adequate placental function. However placental inadequacy as related to 'Small for Gestational Age' infants has been very difficult to demonstrate because of its multifactorial origin. The altered placental function may result from primary pathological alterations in the maternal organism, the foetus or the placenta per say. All these causative factors for I.U.G.R. are so closely interlinked that it is not possible to implicate any factor alone as being causative in nature.

Development of symmetrical or asymmetrical I.U.G.R. depends on the duration and the time of placental insult.

Material and Methods

100 placentae of mothers who had given birth to I.U.G.R. babies at term were selected for studying morphometric and histological changes. All mothers had some associated complications during pregnancy. 43% cases had anaemia with pregnancy, 37% had some form of infection during pregnancy, 18% had pre-eclamptic toxaemia and 2% were elderly primigravidae. All the placentas were then weighed.

Their shape, dimensions, colour, surface

From: Swami Ramanand Teerth Rural Medical College, Ambajogai. Dist: Beed 431517.
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area, number of cotyledons, gross infarcts, haemorrhages, necrotic areas and calcified deposits were noted. Placenta along with cord were immersed in 10 per cent formalin for 10 days. After fixation microscopic examination was carried out and following changes were noted.

1. Placental infarcts.
2. Calcification.
3. Lymphocytic Infiltration.
4. Hyaline Changes.
5. Villous Changes: Maturity of villi, basement membrane thickening, cytotrophoblastic hyperplasia, syncytial knots etc.

Observations and Discussion

Maximum number of placentae were between the range of 351 to 400 gms. The mean placental weight was 404.5 gms which was about 74.5 gms lesser than the weight recorded in placentae of full term normal infants. Reduction in the placental weight leads to reduction in the surface area. This results in reduction of effective area for nutrient transfer. The mean placental surface area was 200.96 sq.cms. which was 54.04 sq.cms. lesser than its normal control.

On gross examination of the placentae 35% showed calcification, 41% showed infarction, 27% showed fibrin deposition and 3% had retroplacental haemorrhages.

It is very difficult to define placental pathology, H. Fox (1975) stated that infarcts and calcification is seen even in normal pregnancy. It assumes significance if such changes are large and numerous. Normally as maturity of the placenta advances structural changes take place in such a way so as to bring about optimum nutrient transfer. Hence in mature term placenta the cytotrophoblast is almost absent. There is thinning of the basement membrane and vascular core has reached up to the tip of the villous.

Maternal factors such as pregnancy induced hypertension, pregnancy associated with anaemia, viral or bacterial infection during pregnancy affect metamorphic and histological structure of the placenta leading to I.U.G.R.

Maternal infections, even if for a short duration, clinical of subclinical have a direct or indirect effect on the growth of the foetus. As shown in Table I. 70.2% showed presence of leucocytic infiltration. Cytotrophoblastic hyperplasia is often seen as a result of hypoxic injury as in maternal anaemia. The very fact that it is seen in 62% of the placentae in this group suggests that hypoxic injury though not gross must have occurred in the intrauterine period. Viraemia in the form of mild upper respiratory tract infection may go unnoticed in the mother. However this insult is sufficient to cause basement membrane thickening, avascular villi, leucocytic infiltration or delaying villous maturity.

TABLE I

Microscopic findings in placentae from cases having infection during pregnancy

Number of cases : 37.

	Description		Percentage
1.	Infarction	— Mild	32.43
		— Extensive	16.2
2.	Calcification	— Mild	27.02
		— Extensive	10.8
3.	Haemorrhage		None
4.	Hyaline changes		40.5
5.	Villous change		
	— Avascular villi		18.9
	— Syncytial knots		48.6
	— Basement membrane thickening		56.7
	— Cytotrophoblastic hyperplasia		62.0
6.	Leucocytic infiltration		70.2

When anaemia is the complicating maternal factor the essential damage to

the placenta is of hypoxic variety due to adminished oxygen carrying capacity of maternal blood. Table II shows that syncytial knots are present in 88% placentae, cytotrophoblastic hyperplasia in 69% and basement membrane thickening in 62%.

TABLE II

Microscopic findings in placentae from Mothers having anaemia during pregnancy

Total cases : 43.

Description		Percentage
1. Infarction	— Mild	41.8
	— Extensive	2.3
2. Calcification	— Mild	51.1
	— Extensive	11.6
3. Haemorrhage		None
4. Hyaline changes		23.2
5. Villous change		
— Avascular villi		34.8
— Syncytial knots		88.0
— Cytotrophoblastic hyperplasia		69.7
— Basement membrane thickening		62.7
6. Leucocytic infiltration		55.8

Overall decrease in oxygen carrying capacity of the mother affects the villi; which are also hypovascular. Increased syncytial knots are an expression of accelerated sequestration of the nuclei so as to have optimum nutrient transfer. Cytotrophoblastic hyperplasia may be due to failure of regression of these cells or due to rapid proliferation as a result of hypoxia. Thus the villi are immature for that period of gestation.

The placental changes in maternal pre-eclampsia are ischaemic in nature. The ischaemia is mainly as a result of intense vasospasm and decreased perfusion of nutrients due to sluggish blood-flow. Haemoconcentration and reduced capacity of the intravascular compartment also contributes for the ischaemia. Thus in majority, villous structure is deranged in the form of presence of avascular villi. syncytial knots, cytotrophoblastic hyperplasia and basement membrane thickening as shown in Table III. All these changes are exaggerated in severe pre-eclampsia.

TABLE III

Microscopic findings in placentae from Mothers having mild and severe pre-eclampsia

Total cases : 18.
Mild P.E.T. : 10.
Severe P.E.T. : 8.

Description		Mild P.E.T.%	Severe P.E.T.%
1. Infarction	— Mild	80	12.5
	— Extensive	20	87.0
2. Calcification	— Mild	60	25.0
	— Extensive	40	75.0
3. Haemorrhage		None	37.5
4. Hyaline change		60	62.0
5. Villous change			
— Avascular villi		80	62.0
— Syncytial knots		60	87.5
— Cytotrophoblastic hyperplasia		70	87.5
— Basement membrane thickening		60	100.0
6. Leucocytic infiltration		40	12.5

Placentae from two elderly primigravidae showed no other change except presence of avascular villi, syncytial knots and cytotrophoblastic hyperplasia. Though hypertension was not detected in them, villous changes were suggestive of placental ischaemia.

All these histological changes are clearly seen in the photographs from No. 1 to No. 8.

Conclusion

Even a minor insult of the placenta, in its developmental stage, in the form of mild anaemia, short lived maternal infection or hypertension can hamper placental maturity leading to I.U.G.R. Though there is sufficient time for maternal recovery, in few cases placental injury may persist throughout pregnancy. Thus it is suggested that such minor ailments in a developmental stage should never be overlooked. This will ultimately help in improving the quality of life in this modern era of 'Two Children Family'.

References

1. Fox, H.: J. Obstet. Gynec. Ind. 25: 441, 1975.
2. Fox, H.: Pathology of the placenta. Vol. 7: 1975.

See Figs. on Art Paper I & II