Assessment Of Fetal Oxygenation In Grade III Placental Calcification

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Summary:

Background: *To determine whether advanced placental calcification is associated with impaired placental function by assessment of different grades of placental calcification in relation to cord blood NRBCs count, a marker for chronic hypoxia.* ***Study Design*** *Nucleated Red Blood Cells (NRBCs) from cord blood of 200 singleton term non -asphyxiated neonates were compared with cord blood NRBCs of 51 term singletons proved to have 2ed and 3ed Grade placental calcification by ultrasound examination Fetal weight and Apgar score were also assessed in different groups.*

Results *Grade III placental calcification (n=30) was not associated with a significant difference in NRBC counts compared to group one (n=200): mean 2.45.S.D.2.41 and mean 2.40,SD 2.12 respectively (P> 0.8).*

Conclusions *These result illustrated that advanced grades of placental calcification dose not Jeopardize fetal oxygenation, as evidenced by the non significant finding of NRBC counts in different groups, and the non Significant difference in fetal out come by comparing fetal weigh & Apgar scores for different groups.* ***Keywords;*** *Neonatal NRBC, placental maturation, fetal hypoxia.*

Introduction:

The placenta is an organ that is absolutely essential to the survival of the fetus of mammals, it undergoes continuously a change in weight, structure, shape and function in order to support prenatal life (l).

Recant advances in ultrasound have provided the clinician with the ability to visualize the placenta in situ, and have allowed investigators to describe progressive sonographic changes in the placenta .

Grannum et al (1979) describe a four graded system (0-3) for morphological characterization of the placenta with real time ultrasound, and stated that grade III placcntal calcification is the most mature placenta .

Ihe subject of placenta] maturity grading and its Significant have attracted an interest regarding its relation to fetal outcome, and have been studied extensively in recent years .

Fetal hypoxia has an important influence on survival and quality of life and requires careful follow up of the infant . However it is still difficult to determine the presence, duration and extent of fetal hypoxia. NRBCs in fetal circulation (cord blood) can provide information about fetal hypoxia, these cells (NRBCs) are released to the circulation when there is excessive erythroid production under the influences of fetal Erythropoitin (Ep) hormone which is produced in response to hypoxia in animal and human fetuses <>6).

The purpose of this study was to determine the relationship of utrasonogarphicaly observed placental calcification (maturation) to postnatal cord blood NRBCs count, a marker for chronic fetal hypoxia.

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Patients and method:

This is a prospective study extending through period of 16 month from June 1998 to OCT 1999. The obstetric ultrasound lab. of Baghdad teaching hospital examined all referred patients by means of Seimens sonoline verspro with 3.5 MHz transducer. Each examination included measurement of bipareital diameter and femur length and assessment of placental location and grade, according to the criteria of Grannum and associates .

We evaluated 251 consecutive ranged pregnant women, who delivered a life born infants, and fulfilled the following criteria: their age ranging from 20-35 years, weight between 45-90 kgs, height > 145 parities < 5, Rh + ve, no evidence of maternal illness, no complications or medications during pregnancy and they denied smoking or alcohol.

All cases were of Singleton, term pregnancy (gestational age > 37and < 42 weeks) by last menstrual period and /or early U/S. Patients were divided into three groups according to placental

maturation, according to Grannum. Group I (control group) are those with grade (0-1) while the second groups comprise those with grade II placental calcification and third group involving those with grade III placental calcification.

Umbilical vein blood was collected immediately after delivery by an ethylinediaminetetraactic acid tube (EDTA), Samples were labeled (NRBC study) and sent to the hospital lab. In the laboratory a slide was prepared with Leishman stain, and NRBCs were manually counted, the total white blood cell count were initially determined. The number of NRBCS was determined by examination of the blood smear from the differential white blood cell count. For the purpose of this study, the number of NRBC$ per 100 white blood cell is expressed as the NRBCs count.

Variables also considered include maternal age, parity, birth weight, 5 minute Apgar score.

Statistical analysis was performed by ANOVA test with LSD procedure and X" test.

Results:

A total number of 251 live born, terms, Singleton neonates who were delivered at our institution, were studied.

Table I compared the clinical characteristics of patients with Grade II placental maturation to those of control patients. The two groups were not significantly different with respect to maternal age, parity, birth weight or 5 minutes Apgar score.

Table I clinical characteristics of the placental Grade II population in comparison to control group

	Grade 11 population	control	P
No.	21	200	
Age (years ± SD)	22.3 ±6.1	22.3 ± 5.6	NS
Parity (± SD)	2.50 ± 1.1	2.50± 1.1	NS
Birth weight (gm ± SD)	3.209±515	3228 ±416	NS
5 min. Apgar score (± SD)	8.8 ±0.5	8.8 ±0.7	NS

Table 2 presents a similar comparison between grade 111 population and control group, the two groups were not significantly different for any characteristics.

Table 3 present the comparison of placental calcification (grade TI) and grade III population to the control group in relation to NRBCs/ 100 WBC. Neither grade TI (n=21) nor grade III (n=30) were associated with any Significant difference in NRBCs count compared to control group (n=200).

Table 2 clinical characteristics of the placental grade III population in comparison to control group.

	Grade 111 population	control	P
No.	30	200	
Age (years ± SD)	21.7 ±5.9	22.3 ±5.6	NS
Parity (± SD)	2.51 ±1.1	2.50± 1.1	NS
Birth weight (gm ± SD)	3.142 ±559	3228±416	NS
5 min. Apgar score (± SD)	8.7 ±0.4	8.8 ±0.7	NS

Table 3 comparison of placenta! calcifications grade II and III to control group in relation to NRBCs/IOOWBC.

	n	mean	SD	min.	max.
control group	200	2.40	2.124	0.00	10.00
grade TT	21	2.512*	2.401	0.00	10.00
grade IIT	30	2.546*	2.419	0.00	10.00

***P value >0.6**

Discussion:

Fujikura l4) studied in vitro placental calcification, and noted decreasing calcification with increasing parity. He could find no relation between fetal or placental weight and the severity of calcification.

Tindall and Scott(4>who studied placental calcification radiographically, showed increasing calcification with increasing maturity. They also demonstrated an inverse correlation between calcification and increasing maternal age and parity. The degree of calcification was unrelated to the placental weight; fetal weight ratio, fetal asphyxia or perinatal complication.

Grannum et al(found a correlation between maturational changes of the placenta as seen by U/S and fetal pulmonic maturity as indicated by L/S ratio.

Winsberg * ' and Fisher and associates (\ suggested a relationship between early placental maturation and declining placental function.

Patterson and associates f4), in their cross sectional study on the assessment of early placental calcification, they tried to establish a definition on early placental maturation, and evaluated the Significance of this finding as a predictor of perinatal out come. They found a trend toward lower mean birth weights in the group with early placental maturation as compared to controls;

however these differences did not reach to statistical Significance. They concluded that early placental calcification was an insensitive predictor of poor perinatal out come with respect to maternal hypertension, antepartum or intrapartum fetal distress, and perinatal asphyxia .

Burton (1995) demonstrated that the progression from immature to mature appearance is associated with an increase of approximately 40% in the diffusing capacity of the placenta and this most facilitate gaseous exchange.

In the present study we attempted to demonstrate the relationship between ultrasonographically assessed placental maturation, birth weight, perinatal asphyxia (defined as five minutes Apgar score < 6) and postnatal cord blood NRBC count as a predictor of chronic intrauterine hypoxia.

In approval with other studies we failed to demonstrate a statistically significant difference in mean birth weight of infants in the grade II and III population as compared to the control group.

With the use of placental maturation as predictor of perinatal out come, we found that it was an insensitive predictor of perinatal out come as evidenced by statistically non significant difference in 5 min. Apgar score of infants in the grade II and III population as compared to control group.

NRBCs count of cord blood present a relatively sensitive indicator of fetal oxygenation during intrauterine life .

In contrast to suggestion that extensive calcification might indicate an inability of the feto-placental unite to function properly (, the result in this study showed no significant elevation of NRBCs count in the grade II and III population as compared to control group, to our knowledge there have not been a research that studied the relation between placental maturation and NRBCs count. The major limitation of this study is small sample size , and the need to other predictor of fetal hypoxia such as cord blood Erythropoietin concentration which might provide additional substantiating evidence.

Conclusions:

These result illustrated that advanced grades of placental calcification dose not Jeopardize fetal oxygenation, as evidenced by the non significant finding of NRBC counts in different groups, and the non Significant difference in fetal out come by comparing fetal weigh & Apgar scores for different groups.

As observed from this study and other studies, ultrasonographically assessed placental maturation have less clinical significance in the absence of other obstetrical risk factors.

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