



RESEARCH ARTICLE

NUCLEATED RED BLOOD CELLS (NRBCS) IN TERM NEONATAL UMBILICAL CORD BLOOD TO MOTHERS WITH GESTATIONAL DIABETES WHO ARE LARGE (LGA) AND AVERAGE FOR GESTATIONAL AGE (AGA) COMPARED TO (AGA) NEONATES TO HEALTHY MOTHERS

HelemaYasser .Al-Massawi

Al_Imamain AL-Kadhemain Medical City, Iraq-Baghdad Department of Obstetrics and Gynaecology

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A B S T R A C T

Background:-Nucleated red blood cells (NRBC) are fetal hematologic markers for placental dysfunction, hypoxemia, and asphyxia. NRBC count elevation at birth or persistence is linked statistically to adverse outcome, but clinical predictive value is variable, one of these adverse outcomes is gestational diabetes which is one of the most common disorders associated with pregnancy.

Objective: The aim of this study is to determine whether nucleated red blood cells count in cord blood of (LGA) and (AGA) infants of gestational diabetic mothers are elevated when compared to its level in (AGA) infants to normal mothers.

Design: Aprospective case comparative study. SETTING: Al-Imamain AL-Kadhemain Medical City.

Method(S): The study was conducted on 90 infants belonging to pregnant women with term pregnancy and single viable fetus delivered by elective caesarian section . The sample has been divided in to three groups, included thirty (LGA) infant of women with gestational diabetes mellitus, and another thirty (AGA) infants to women with G.D.M and a control group of thirty (AGA) infants to non-diabetic women. Umbilical cord blood samples collected during the first 12 hours of life and sent to the laboratory for differential count with N-RBC count done manually. Infants to mothers with hypertention, smoking, or drug abuse, abnormal FHR abnormalities, low apgar scores, haemolysis, blood loss and congenital anomalies has been excluded.

Result: There was a significant increase in the umbilical cord blood absolute N-RBC with p-value<0.001.

Conclusion: there is a positive correlation of absolute N- RBC level in umbilical cord blood with birth weight and maternal diabetes.

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INTRODUCTION

Gestational diabetes is defined by the World Health Organization (WHO) as carbohydrate intolerance resulting in hyperglycaemia of variable severity with onset or first recognition during pregnancy⁽¹⁾.

Approximately 7% of all pregnancies are complicated by GDM, resulting in more than 200,000 cases annually. The prevalence may range from 1 to 14% of all pregnancies, depending on the population studied and the diagnostic tests employed.⁽²⁾ Although most women with GDM revert to normal after delivery, impaired glucose tolerance and / or diabetes develop in about 50% within 10 years postpartum.⁽³⁾

The following have been shown to be independent risk factors for gestational diabetes and should be recognized as such by health care professionals : BMI more than 30 kg / m², Previous macrosomic baby weighing 4.5 kg or more, Previous gestational diabetes, Family history of diabetes (first-degree relative with diabetes). History of recurrent spontaneous abortions, History of unexplained intrauterine fetal death. Previous infants with major congenital anomalies. History of recurrent preeclampsia. Development of polyhydramnios and / or fetal macrosomia in present pregnancy. Middle Eastern (specifically women whose country of family origin is Saudi Arabia, United Arab Emirates, Iraq, Jordan, Syria, Oman, Qatar, Kuwait, Lebanon or Egypt).^(1,4)

*✉ **Corresponding author: HelemaYasser .Al-Massawi**

Al_Imamain AL-Kadhemain Medical City, Iraq-Baghdad Department of Obstetrics and Gynaecology

GDM is a multi factorial disease associated with both genetic and non-genetic/ environmental risk factors. Pregnancy was recognized to be potentially diabeto genic condition as early as 1824.(5) There is increase in insulin resistance and increased pancreatic insulin secretion as the pregnancy progresses.(6) It begins in mid pregnancy and continues until the end of gestation, it is due to placental secretion of hormones such as progesterone, cortisol, and placental lactogen acting in an insulin primed environment.(5) The underlying pathophysiology of GDM is in most instances similar to that of type 2 diabetes. The inability of the pancreatic beta cells to match increased insulin resistance to normalize systemic glucose translates in to maternal hyperglycemia.(5,6)

Risk assessment for GDM should be undertaken at the first prenatal visit. Women with high risk to develop GDM should undergo glucose testing as soon as feasible. If they are found not to have GDM at that initial screening, they should be retested between 24 and 28 weeks of gestation. Women of average risk should have testing undertaken at 24–28 weeks of gestation.(7)

A fasting plasma glucose level >(7.0mmol/l) or a random plasma glucose >(11.1 mmol/l) meets the threshold for diagnosis of G.D.M. In the absence of this degree of hyperglycemia, evaluation for GDM in women with average or high-risk characteristics should follow one of two approaches

One-step approach: Perform a diagnostic oral glucose tolerance test (OGTT) without prior plasma or serum glucose screening.

Two-step approach: Perform an initial screening by measuring the plasma or serum glucose concentration 1h after a 50-g oral glucose load (glucose challenge test [GCT]) and perform a diagnostic OGTT on that subset of women exceeding the glucose threshold value on the GCT(8). When the two-step approach is employed, a glucose threshold value >140mg/dl (7.8mmol/l) identifies approximately 80% of women with GDM, and the yield is further increased to 90% by using a cut off of >130mg/dl(7.2 mmol/l).(7)

With either approach, the diagnosis of GDM is based on an OGTT. There are at least six different versions of the OGTT which involves ingestion of 75 gram glucose load, Table-1.(9) Table-1-World Health Organization criteria for the 2-hour 75 g oral glucose tolerance test.(9)

	Whole blood venous (mmol/L)	Whole blood capillary (mmol/L)	Plasma venous (mmol/L)	Plasma capillary (mmol/L)
Fasting	>=6.1	>=6.1	>=7.0	>=7.0
2 hours	>=6.7	>=7.8	>=7.8	>=8.9

Effect of Maternal Gestational Diabetes on Pregnancy

Fetal Complications: Congenital malformation : it is widely held that an increase in congenital anomalies can be expected with G.D.M. (11) These are(2-5) fold increased risk of cardiac anomalies, 200-400 fold risk of caudal regression (sacral agenesis), and neural tube defects.(8,9)

Miscarriages and still birth, in a recent study the incidence of fetal death was 5.4, 3.6, and 1.8 per 1000 in untreated, treated, and non diabetic mothers, respectively.(10,11)

Fetal macrosomia: occurs in 17-29% of pregnancies with gestational diabetes, it is associated with brachial plexus damage and clavicular fractures.(11). Long-term complications to the offspring include an increased risk of glucose intolerance, diabetes, and obesity.(1)

Neonatal Complications: Hypoglycemia: Approximately 15%-25% of neonates delivered from women with diabetes develop hypoglycaemia during the immediate new born period. This results from maternal hyperglycemia causing fetal hyperinsulinemia.(12)

Hypocalcaemia: Up to 50% of infants of diabetic mothers have low level of serum calcium that is less than 7mg/dl. These changes appear to be due to a functional hypoparathyroidism) Polycythemia: Central venous hemoglobin concentration of greater than 20gm/dl or hematocrit more than 65%. Hyperglycemia is a powerful stimulus to fetal erythropoietin mediated by decreased fetal O2 tension.

Hyperbilirubinemia: It occurs in approximately 25% of infants of diabetic mothers. The causes of hyperbilirubinemia are multiple but predominantly due to prematurity and polycythemia. Respiratory distress syndrome: The non-diabetic fetus achieves pulmonary maturity at a mean gestational age of 34-35 weeks. However in a diabetic pregnancy the risk of RDS may pass until after 38.5 weeks of gestation completed.(13)

Maternal complications

Polyhydramnios: occurs frequently in G.D.M. particularly when the fetus is macrosomic. It is due to fetal hyperglycemia and fetal hyperinsulinemia . hyperglycemia, in part through an osmotic action, result in polyurea; this leads to the development of polyhydramnios.(11,13)Preeclampsia: It is more frequent among women with diabetes, occurring approximately twice in diabetics as in non-diabetics population(5). Operative delivery: Maternal diabetes is a risk factor for caesarian delivery especially when there is fetal macrosomia as there is risk for 3rd and 4th degree tears with vaginal delivery,

Infection: Maternal infections are more common in diabetics.(9)

Good glycaemic control prepregnancy and during the 1st 8 weeks reduces the risk of congenital abnormalities. (HBA1c) level of 6.1% or less indicates good glycaemic control in the preceding 4-12 weeks. Self blood glucose monitoring (SBGM) (capillary) has now become the standard of care for monitoring of pregnant women .(9)

Diettherapy: The goals of dietary interventions in pregnancies complicated by diabetes are the optimization of glycaemic control while avoiding ketoacidosis and minimizing the risk of hypoglycemia in women taking insulin.(1)

The recommended daily caloric intake is 30 kcal/kg/day based on their present pregnant weight. In women with GDM who are overweight ($BMI > 30 \text{ kg/m}^2$), a 33% calorie restriction of their estimated energy needs is recommended (~25 kcal/kg/day).⁽⁸⁾

Exercise: Regular daily exercises (preferably after meals), can decrease insulin requirements by as much as 50%.^(8,9)

Insulin therapy is needed when good control of blood glucose cannot be achieved by diet alone. The duration of dietary treatment prior to initiation of insulin will depend on gestational age at diagnosis and the level of glycaemic control.⁽³⁾ For GDM refractory to dietary therapy and exercise, alone,⁽¹⁴⁾ or if ultrasound scans suggest fetal macrosomia,⁽¹⁾ insulin is generally recommended, but recent studies have evaluated oral hypoglycemic agents. Glyburide is a second-generation oral sulfonylurea, and metformin is an oral agent in the biguanide class that increases insulin sensitivity.⁽¹⁴⁾ Sulphonylurea and biguanides are usually avoided in pregnancy because they cross the placenta, potentially resulting in fetal hypoglycemia. However, they have been used in all trimesters and reported as safe.⁽⁹⁾ Failed dietary treatment (after 2 weeks of dietary method). Short acting insulin (regular) (actrapid), intermediate or long acting insulin may be used in a combination schedules to affect maternal euglycemia.⁽¹⁾

Fetal Surveillance: Ultrasonography at 4-6 week intervals, Maternal assessment of fetal activity, daily after 28 weeks of gestation Non stress test (NST). Contraction stress test or biophysical profile if NST non reactive; L/S, lung profile at 37-38 weeks.⁽¹⁰⁾

Decision regarding the timing and mode of delivery must balance with the risk of late intrauterine death and macrosomia. ⁽⁹⁾

The optimal time for delivery is typically at 38- 39th week. If the fetus is not macrosomic and the results of biophysical testing are reassuring the obstetrician can wait spontaneous labor.⁽⁶⁾ Women with suspected fetal macrosomia should have risks and benefits of all modes of delivery discussed with them.⁽¹²⁾ The American college of obstetricians and gynecologists recommends offering an elective C/S for diabetics with estimated fetal weight $> 4500 \text{ gm}$ or more.⁽¹³⁾

Maternal blood glucose during labor and delivery should be maintained approximately 4-7 mmol/l (72-144mg/dl), to avoid hypoglycemia with its adverse effects on them other and the baby.⁽⁹⁾

Once delivery is completed insulin infusion rate should be decreased in to the half, and once the mother starts to eat orally then we can commence the subcutaneous insulin regimen.

Regard in oral hypoglycemic drugs they can be commenced for breast feeding mothers slowly with special care of infant

hypoglycemia.⁽⁹⁾ Nucleated red blood cells (nRBCs) are rarely found circulating in older children; they are commonly seen in the blood of new borns. They are primarily produced in the fetal bone marrow in response to erythropoietin and are stored in the marrow as precursors to reticulocytes and mature erythrocytes. NRBCs are present in the placental vessels through the first half of pregnancy, but are uncommon later in pregnancy and are usually absent or present only in small numbers at term. The finding of numerous nRBCs in the term placenta is non-specific and may indicate acute or chronic fetal hypoxia, maternal diabetes, fetal anaemia, or congenital TORCH infections (toxoplasma, other viruses, rubella, cytomegalovirus, and herpes).⁽¹⁶⁾

In 1924, Lippman reported NRBCs in the blood of 41 of 42 new borns in the first day of life. These cells constituted about 500 nRBCs / mm^3 or 0.1% of the new borns circulating red blood cells. Since then, many investigators have reported similar values at and shortly after birth. It is reasonable to conclude that the mean value of nRBCs in the first few hours of life in Healthy term newborns is about 500 nRBCs/mm, and that a value above 1000 nRBCs / mm^3 can be considered elevated. Expressed differently, 0-10 nRBCs/100WBCs are typical, and Values above 10-20 nRBCs/ WBC are elevated, although these values are highly dependent on the total leucocyte count.⁽¹⁶⁾

In the normal neonate, nRBCs are rapidly cleared from the blood stream after birth. By 12 hours of age, the counts fall by about 50%, and by 48 hours only 20-30 nRBCs/ mm^3 are found. In healthy term newborns, virtually nonRBCs are found after the third or fourth day of life, although they may persist in small numbers upto 1 week in preterm newborns.^(15,16,17) Causes of increase in NRBCs in fetuses & newborns:⁽¹⁶⁾

1. Physiological : Physiological causes of increase in NRBC are labour and vaginal delivery, postterm newborns, preterm newborns. 2. Increase erythropoiesis: Chronic hypoxia due to increase level of erythropoietin that stimulates erythropoiesis,^(18,19) growth restriction: due to chronic hypoxia, ⁽¹⁵⁾maternal preeclampsia, and hypertension, maternal smoking, maternal diabetes due to increase erythropoietin levels and direct haemopoietic effect of hyperinsulinaemia, anemia (due to blood loss or haemolysis due to ABO or Rh isoimmunization) and other causes (leukaemia, Down syndrome, TORCH infection). 3. Acute stress release: NRBCs released in response to acute or subacute hypoxia: depends on severity and duration of asphyxia and in Chorioamnionitis due to acute increase in erythropoietin level.^(18,19)

Aim of the Study

The aim of this study is to determine whether nucleated red blood cells count in cord blood of (LGA) and (AGA) infants of gestational diabetic mothers are elevated when compared to its level in (AGA) infants to normal mothers

Patients and Method

This prospective case comparative study was conducted at the department of gynaecology and obstetrics with the nursery care unit at Al-Imamain AL-Kadhemain Medical City during the period (July 2011–October 2011). Mothers consent was verbally taken before samples collection. This study included 90 infants belonging to pregnant women, who were admitted to the delivery ward, selected according to clinical signs, symptoms, and investigations those with gestational diabetes has been diagnosed by O.G.T.T and divided into 3 groups: Group 1: Included 30 (LGA) infants to gestational diabetic mothers. Group 2: Included 30 (AGA) infant to gestational diabetic mothers. Group 3: Included 30 (AGA) infants to normal mothers. For all groups gestational ages were calculated depending on their last menstrual cycles if regular, or according to their early ultrasound, with pediatric assessment of the new borns. Mode of delivery was by caesarean delivery, among nulliparae and multiparae for infants of diabetic mothers and control group. In an attempt to control various variables known to affect NRBC counts, any woman with one of the following criteria was excluded from the study; Hypertension. Preeclampsia. Chorioamnionitis. Smoking during pregnancy. Antepartum haemorrhage. Infants with one of the following criteria were excluded: Cyanotic heart diseases. Signs of fetal distress. Chromosomal anomalies. Perinatal blood loss, haemolysis (blood group incompatibility)

Immediately after delivery, the umbilical cord was double clamped; 2ml of umbilical cord blood was collected using a syringe into a test tube containing anti coagulant material ethylenediamine tetraacetic acid (EDTA).

A complete blood count was performed, blood smear stained with Leishman's stain was prepared and number of NRBC per 100 white blood cells was determined.

Statistical analysis

Data were analyzed using SPSS (version 16) and Microsoft Office Excel 2007. Numeric data were presented as mean ± SD except for RBC and Apgar score which were represented by (median and range), while nominal data were presented as number and percent. ANOVA test was used to study the correlation among numeric data while chi-square test was used to study association among nominal data. P-value less than 0.05 was considered significant.

RESULT

Table 1 Demographic Characteristics

Characteristics	Gestational diabetes		Non-diabetic	P
	LGA	AGA	controls	
Birth weight (grams)	4280.0 ± 212.78	3258.3 ± 361.05	3219.3 ± 290.84	<0.001
Gestational age (week)	267.17 ± 7.37	264.40 ± 5.44	266.07 ± 5.80	0.233
Maternal age (years)	28.10 ± 7.33	31.00 ± 7.34	27.80 ± 7.42	0.184
Parity	2.97 ± 1.51	2.93 ± 1.98	2.80 ± 2.00	0.934
Gravida	5.10 ± 2.33	4.63 ± 2.34	4.17 ± 2.53	0.328
1-min Apgar score	9 (8-10)	8 (8-10)	8.5 (8-10)	0.008
5-min Apgar score	10 (9-10)	9 (8-10)	9 (8-10)	0.008
Gender (M:F)	14:16	14:16	17:13	0.670

Table (1) shows that there is a statistically significant difference in birth weight between the three groups of comparison in the study, evident by a significant p-value < 0.001. It shows no statistically significant difference between them in gestational age, parity, gravidity, male: female ratio, Apgar score at 1 & 5 minutes.

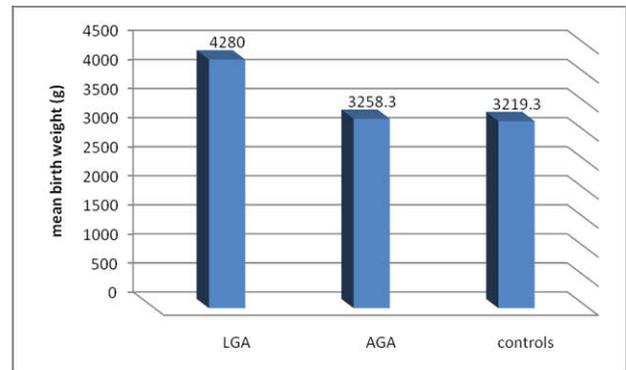


Figure 1 Mean birth weight per gram in the three groups.

The figure (1) shows the mean birth weight in grams in the three studied groups. It is 4280 (gm) for (LGA), 3258.3 (gm) for (AGA) infants to diabetic mothers and 3219.3 for control group.

Table 2 Hematological findings

Characteristics	Gestational diabetes		Non-diabetic	P
	LGA	AGA	Controls	
HCT	60.20 ± 6.47	55.13 ± 5.71	46.05 ± 7.20	<0.001
RBC (x10 ⁹ /L)	5.34 ± 0.68	5.14 ± 0.51	4.53 ± 0.77	<0.001
WBC	12.97 ± 3.72	9.71 ± 1.89	14.76 ± 3.50	<0.001
Platelet (x10 ⁹ /L)	252.87 ± 69.19	257.53 ± 78.01	240.27 ± 60.60	0.612
Absolute lymphocyte (x10 ⁹ /L)	6.47 ± 1.35	4.87 ± 1.44	5.35 ± 0.91	<0.001
Nucleated RBC/100 Corrected	31.56 ± 15.82	6.76 ± 6.42	2.06 ± 2.16	<0.001
WBC (x10 ⁹ /L) Absolute	29.007 ± 3.19	21.073 ± 6.37	18.627 ± 3.48	<0.001
nRBC (x10 ⁹ /L)	0.79 (0.07-0.91)	0.15 (0-0.47)	0.005 (0-0.080)	<0.001

Table (2) shows the haematological findings in the three groups. It shows significant differences in all variables (p-value < 0.001) except in platelet count, which was not statistically significant (p-value = 0.612).

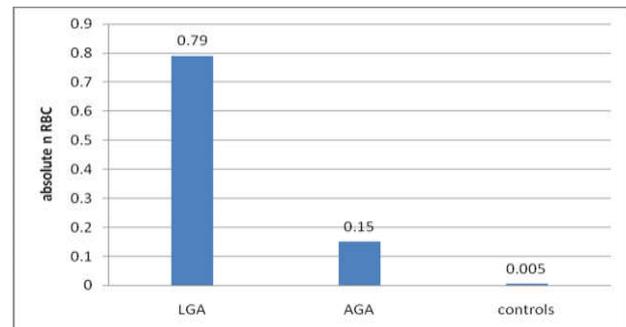


Figure 2 Shows absolute nucleated red blood cell count in the three studied groups.

The figure (2) shows the absolute nucleated red blood cell count in median (range)in the three studied groups. It is 0.79 for (LGA), 0.15 for (AGA) infants to diabetic mothers and 0.005 for control group.

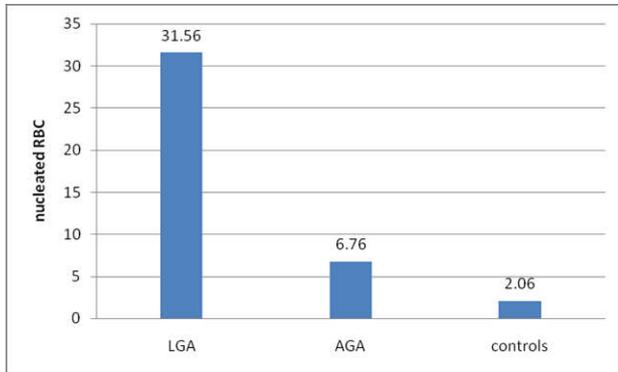


Figure3 Shows nucleatedredblood cell count/100WBC inthethree studied groups.

The figure (3) shows the nucleated red blood cell count /100WBC in (mean±SD) in the three studied groups. It is 31.56 for (LGA), 6, 76 for (AGA) infants to diabetic mothers and 2.06 for control group.

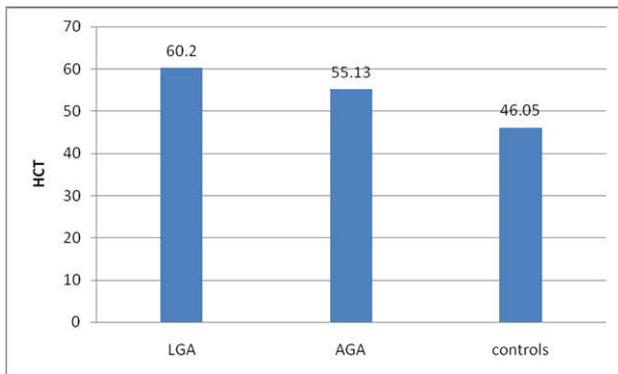


Figure 4 shows the haematocritlevelin the threestudiedgroups.

The figure(4) shows the haematocrit level in (mean±SD) in the three studied groups. It is 60.2 for (LGA),55.13 for (AGA) infants to diabetic mothers and 46.05 for control group.

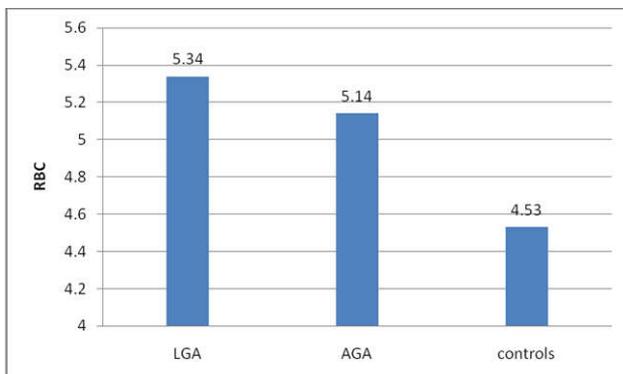


Figure5 mean RBC countinthe three studied groups.

The figure(5) shows the red blood cell countin (mean±SD) in the three studied groups. It is 5.34 for (LGA), 5.14 for (AGA) infants to diabetic mothers and 4.53 for control group.

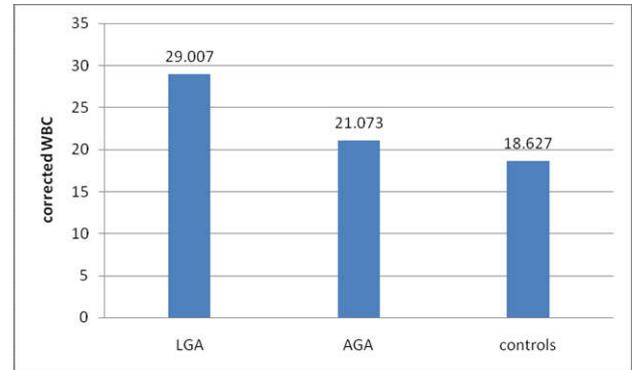


Figure 6 Shows corrected white blood cell count in the three studied groups.

The figure (6) shows the corrected white cell count in (meanS±D) in the three studied groups. It is 29.007 for (LGA), 21.073 for (AGA) infants to diabetic mothers and 18.627 for control group.

Table 3 Shows numbers of diabetic mothers according to the methods of treatment of diabetes.

Characteristics	Gestationaldiabetes		P
	LGA	AGA	
Mixed	10:30	5:30	0.136
Insulin	9:30	10:30	0.781
OHD	2:30	5:30	0.424
Diet	9:30	10:30	1.000

The table (3) shows that the difference between the numbers of mothers subjected to different methods of treatment was not significant statistically (P-valuewas<0.001) in all the studied groups.

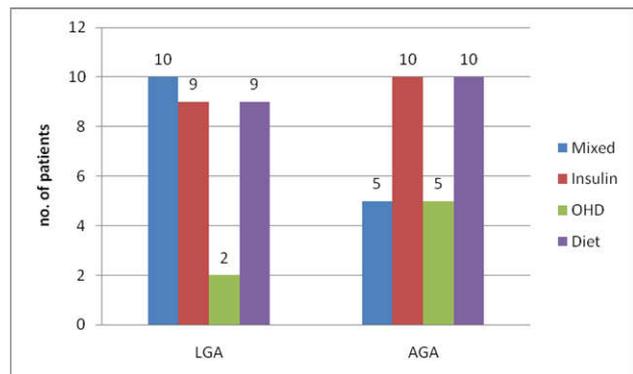


Figure-7 Shows the distribution of diabetic mothers according to different methods of treatment of diabetes.

The figure (7) shows that there is no difference in the number of mothers treated by insulin or oral hypoglycemic agents between the two groups of gestational diabetic mothers, but shows that the number of gestational diabetes mothers who give birth to (LGA) infants treated by mixed treatment=diet and insulin) are double that of mothers who give birth to (AGA) infants and received the same treatment, while the number of mothers who delivered (AGA) infants managed

by dietary control are more than double that of mothers who give birth to (LGA) infants managed by dietary control.

DISCUSSION

This study focused on the count of nRBCs in relation to birth weight, gestational age and gestational diabetes showed that the difference in birth weight between LGA infants to diabetic mothers and AGA infants to diabetic or non-diabetic mothers is (4280.0+₋212.78, 3258.3+₋361.05 and 3219.3+₋290.84) respectively is statistically significant (P-value less than 0.001). While the difference in other variables which are (gestational age, maternal age, parity, gravidity, 1-minute Apgar score, 5-minute Apgar score and gender) was not significant statistically P-value more than 0.05 (table 1, fig. 1).

It also showed that LGA infants to diabetic mothers had higher absolute nRBC count than AGA infants to non-diabetic mothers or AGA infants to mothers with gestational diabetes 0.79(0.07-0.91) for LGA infants to mothers with gestational diabetes, 0.15(0-0.47) for AGA to mothers with GDM, 0.005(0-0.080) AGA infants to healthy mothers), the difference between absolute nRBCs in LGA infants to mothers with GDM and the other two groups of AGA was statistically significant (p-value less than 0.001) (table 2, fig 2,3).

The high level of umbilical cord - nRBCs has been suggested to be considered as a sign of chronic fetal hypoxia (17,20,21,).

Studies have established that in term infants, this index may be affected by factors such as maternal diabetes mellitus, fetal growth restriction, rhesus isoimmunization, maternal tobacco use, and chorioamnionitis (22, 23, 24).

The assumption has been that erythropoietin production, secondary to hypoxia, causes the production of nRBC. It has been shown that in sheep, acute hypoxemia results in erythropoietin increases after at least 3 h, and the resulting reticulocytosis does not peak until 4 days. However, in rats, acute hypoxia resulted in elevated n-RBCs sometime after 4–12 h (25,26).

It has also been suggested that acute hypoxia in humans can be associated with elevated nucleated red blood cells. In humans, labor has been associated with elevated umbilical cord erythropoietin levels (27,28).

Increased nRBC have also been found in placentas in association with cases of acute hypoxia. Other previous studies have reported the correlation of neonatal asphyxia and n-RBC count (29,30).

The correlation is likely to be attributable to the mechanism in which asphyxia, upon delivery, transfers blood from the intrauterine placenta to a fetus (31).

The circulatory blood increases due to the increased blood influx into the placenta, leading to an increase in then RBC count due to the influx of premature red blood cells into the circulation along with the increased blood volume (32).

If hypoxia is prolonged, continued increased RBC production might lead to neonatal polycythemia (33).

Regarding the other haematological characteristics in our series we found that the hematocrit was significantly higher in LGA infants to mothers with GDM than in AGA infants to mothers with or without GDM (P-value less than 0.001. (table 2, fig 4).

This result is in agreement with that reported by Salvesen et al who mentioned that the erythropoietin, hemoglobin, and erythroblast counts were significantly higher than the appropriate normal mean for gestation. There were significant associations between (1) fetal erythropoietin and erythroblast count, (2) fetal erythroblast count and hemoglobin, (3) fetal haemoglobin and maternal glycosylated hemoglobin, and (4) maternal glucose fetal glucose, pH, and lactate (34).

It is also in agreement with Ferber et al who found a significant association between erythropoietin and nRBC counts in term single to n fetuses (35).

It is in line with Yeruchimovich Metal who mentioned that hematocrit and WBC count were higher in LGA infants of diabetic mothers compared with the other groups. The RBC count in our study was significantly higher in LGA infants compared to the control AGA group, this in contrast with Yeruchimovich Metal who found that there was no significant differences among the groups in his study (33).

In our study the WBC counts (total and corrected), were significantly higher in LGA infants to mothers with GDM than the other two groups (table 2, fig 6). The cause of leukocytosis in those infants was not known but might be related to leukocytosis secondary to increased cortisol secretion (36).

In concern to them methods of treatment for which mothers with gestational diabetes has been subjected, although the number of those mothers of (AGA) infants who are gestational diabetic treated by oral hypoglycaemic drugs is more than double the number of mothers of (LGA) infants with gestational diabetes treated by the same treatment. While the number of mothers of (LGA) infants to mothers with GDM treated by (diet and insulin) i.e mixed treatment is double that of mothers with AGA infants treated by mixed treatment in this state the difference was not significant statistically, (table 3, fig 7).

It also has been shown that the number of gestational diabetic mothers who give birth to (LGA) and (AGA) infants was almost the same in the two groups who were controlled by diet alone.

CONCLUSION

From this study we concluded that absolute n-RBC was elevated in cord blood of infants to diabetic mothers in both groups belonging to diabetic mothers whether they are LGA or AGA.

There were no significant differences in gestational age, gender, maternal gravidity or parity, male to female ratio and 1 and 5 minutes Apgar scores.

Recommendation

Suggestion for other studies

1. Biochemical assessment of lipid profile (cholesterol triglyceride, LDL HDL), in correlation with serum Insulin level in the pregnant diabetic patient.
2. Assessment of serum free radicals Malondialdehyde (MDA) to evaluate its level in pregnant women with GDM and its relation to nucleated red blood cell in the new born.

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