RESULTS

The details of clinical data, blood glucose levels and fetal outcome in patients with preeclampsia is shown in appendix I, those for patients with eclampsia is shown in appendix II, those for patients with anaemia in appendix III and those in cardiac patients in appendix IV. Appendix V shows these data in patients with preeclampsia and anaemia. Appendix VI shows data in patients with cardiac disease and anaemia, Appendix VII shows data in patients with preeclampsia and cardiac disease, Appendix VIII shows data in patients with eclampsia and anaemia and appendix IX shows data in patients with preeclampsia, anaemia and cardiac disease.

The 102 patients included in the study gave birth to 110 infants (8 twins). The maternal risk factors for IUGR are shown in Table I.

Table I: Risk factors for IUGR (102 patients)

	Risk factor	No.of patients
I.	**Single risk factors**	
	Preeclampsia	50
	Eclampsia	6
	Anaemia	9
	Cardiac disease	6
	Total	(71)
II.	**Multiple risk factors**	
	Preeclampsia + anaemia	17
	Preeclampsia + Cardiac disease	5
	Preeclampsia + anaemia + cardiac disease	1
	Eclampsia + anaemia	5
	Cardiac disease + anaemia	3
	Total	(31)

Infants were divided into two groups according to the maternal fasting blood glucose level.

Group I: Includes infants of normoglycaemic mothers: These were infants where the maternal fasting blood glucose level was 60 mg/dl or more. This included 88 infants and it represents 80% of all infants.

Group II: Includes infants of hypoglycaemic mothers: These were infants where maternal fasting blood glucose level was less than 60 mg/dl. This included 22 infants and it represents 20% of all infants.

Table 2: Frequency of maternal hypoglycaemia in relation to risk factors.

RISK FACTORS		Total no. of cases	Hypoglycaemic cases	%
- All cases		102	21	20.6
* Single risk factors	(5 twins)	71	11	15.5
- Preeclampsia	(4 twins)	50	9	18.0
- Eclampsia	(1 twin)	6	-	10.0
- Anaemia		9	1	11.1
- Cardiac disease		6	1	16.7
* Multiple risk factors	(3 twins)	31	10	32.3
- Preeclampsia + anaemia	(2 twins)	17	4	23.5
- Preeclampsia + Cardiac disease		5	1	20.0
- Preeclampsia + anaemia + cardiac D.		1	1	100.0
- Eclampsia + anaemia	5	3	60.0	
- Cardiac disease + anaemia	(1 twin)	3	1	33.3

Table 2 shows that the number of hypoglycaemic mothers was 21 out of 102 cases, which represents 20.6% of all cases. In cases with single risk factors for IUGR, the number of hypoglycaemic cases was out of 71, which represents 15.5% of all cases and the number of hypoglycaemic cases in cases with multiple risk factors for IUGR was 10 out of 31 cases, which represents 32.3% of cases. There were no statistically significant difference in the number of hypoglycaemic mothers in cases with single and multiple risk factors for IUGR. ($P > 0.05$).

Table 3 shows the frequency of IUGR infants in relation to fasting blood glucose level (hypoglycaemic or normoglycaemic), with various maternal risk factors for IUGR. In the single risk factor group, patients with pre-eclampsia (54 patients) and eclampsia (7 patients) were put together in one group labelled pregnancy toxaemia group (61 patients). Also, in multiple risk factors groups, cases of preeclampsia and eclampsia associated with anaemia and or cardiac disease were put together in one group labelled pregnancy toxaemia group.

Table 3: The frequency of IUGR infants in relations to fasting blood glucose level with various maternal risk factors for IUGR.

Risk factor for IUGR	Total No.of infants	G_I(Normoglycaemic)			G_{II}(Hypoglycaemic)			P-value
		Total no.	IUGR no.	%	Total no.	IUGR no.	%	
All cases	110	88	13	14.8	22	14	63.6	< 0.001
Single risk factors	(76)	65	9	10.8	11	4	36.4	> 0.05
Pregnancy toxaemia	61	52	8	15.4	9	4	44.4	> 0.05
Anaemia	9	8	0	0	1	0	0	*
Cardiac	6	5	1	20.0	1	0	0	*
Multiple risk factors	(34)	23	4	17.4	11	10	90.9	< 0.001
Pregnancy toxaemia + cardiac and/or anaemia	30	21	4	19.1	9	8	88.9	< 0.0001
Cardiac + anaemia	4	2	0	0	2	2	100.0	*

This table shows that in the hypoglycaemic group, the number of infants was 22 and IUGR was present in 14. This represents 12.8% of all infants, 63.6% of infants from hypoglycaemic mothers and 51.9% of IUGR infants. In the normoglycaemic group, the number of infants was 88 and IUGR was present in 13. This represents 11.8% of all infants, 14.8% of infants from normoglycaemic mothers and 48.2% of IUGR infants. The number of IUGR infants was statistically higher in the hypoglycaemic group than in the normoglycaemic one ($P < 0.001$).

In patients with a single risk factors for IUGR (pregnancy toxaemia, anaemia and cardiac disease), the number of infants was 76 infants, which represent 69.1% of all infants. In the hypoglycaemic group, the number of infants was 11 and IUGR was present in 4 infants. This represent 3.6% of all infants, 36.4% of infants from hypoglycaemic mothers and 14.8% of IUGR infants. In normoglycaemic group, the number of infants was 65 infants and IUGR infants was present in 9 infants. This represents 8.2% of all infants, 10.8% of infants from hypoglycaemic mothers and 33.3% of IUGR infants. There was no statistically significant difference in the number of IUGR infants in the hypoglycaemic and normoglycaemic groups ($P > 0.05$) in patients with a single risk factor for IUGR.

In patients with pregnancy toxaemia, the number of infants was 61, which represent 55.5% of all infants. In the hypoglycaemic group, the number of infants was 9 and IUGR was present in 4 infants. This represents 3.6% of all infant, 44.4% of infants from hypoglycaemic mothers and 14.8% of IUGR infants. In normoglycaemic

group, the number of infants was 52 and IUGR was present in 8 infants. This represent 7.3% of all infants, 15.4% of infants of normoglycaemic mothers and 29.6% of IUGR infants. There was no statistically significant difference in the number of IUGR infants in hypoglycaemic and normoglycaemic group ($P > 0.05$) in patients with pregnancy toxaemia.

In patients with anaemia, the number of infants was 9 which represents 7.3% of all infants. In the hypoglycaemic group, there was only one infant with no IUGR. In the normoglycaemic group, the number of infants was 8 and IUGR was not present in any of them. So, there was no IUGR infants in anaemic patients, so statistical evaluation was not done.

In patients with cardiac disease, the number of infants was 6 which represent 5.5% of all infants. In the hypoglycaemic group, there was one infant only with no IUGR. In the normoglycaemic group, the number of infants was 5 which represent 4.6% of all infants IUGR was present in one case only which represent 20% of infants of hypoglycaemic mothers and 3.7% of IUGR infants. Statistical evaluation could not be done because of the small number of cases and absence of IUGR infants in the hypoglycaemic group.

In patients with multiple risk factors for IUGR, the number of infants was 34 which represents 30.9% of the all infants. In the hypoglycaemic cases, the number of infants was 11 and IUGR was present in 10 infants. This represents 9.1% of all infants, 90.9%

of infants from hypoglycaemic mothers and 37.0% of IUGR infants. In the normoglycaemic cases, the number of infants was 23 and IUGR was present in 4 infants. This represents 3.6% of all infants, 17.4% of infants from normoglycaemic mothers and 14.8% of IUGR infants. The number of IUGR infants was statistically more in hypoglycaemic than in the normoglycaemic groups ($P < 0.001$).

In patients with pregnancy toxaemia and cardiac disease and/or anaemia the number of infants was 30. In hypoglycaemic cases, the number of infants was 9 and IUGR was present in 8 infants, which represent 7.3% of all infants, 88.9% of infants from hypoglycaemic mothers and 29.6% of IUGR infants. In the normoglycaemic cases, the number of infants was 21 and IUGR was present in 4 infants. This represents 3.6% of all infants, 19.1% of infants from hypoglycaemic mothers and 14.8% of IUGR infants. The number of IUGR infants was statistically more in hypoglycaemic than in the normoglycaemic group ($P < 0.0001$) in patients with multiple risk factors.

In patients with cardiac disease and anaemia, the number of infants was 4. In hypoglycaemic group, the number of infants was 2 with IUGR in both which represents 1.8% of all infants, 100% of infants from hypoglycaemic mothers and 7.4% of IUGR infants. In the normoglycaemic group, the number of infants was 2 with IUGR. Statistical evaluation could not be done also due to the small number of cases.

Table 4. Shows the effects of maternal risk factors (Preeclampsia, eclampsia, anaemia and cardiac disease) on fetal outcome in relation to fasting blood glucose level.

Table 4: The effect of maternal risk factor on fetal outcome in relation to fasting blood glucose level.

Risk factors	Total no.of cases	Normoglycaemia			Hypoglycaemia			P-value
		Total	IUGR	%	Total	IUGR	%	
Pregnancy toxaemia*	91	73	12	16.4	18	12	66.7	< 0.001
Anaemia**	38	27	4	14.8	11	9	81.8	< 0.001
Cardiac disease***	16	11	1	9.1	5	4	80.0	$= 0.01$

* Present alone or with cardiac disease and/or anaemia.

** Present alone or with cardiac disease and/or pregnancy toxaemia.

*** Present alone or with pregnancy toxaemia and/or anaemia.

Table 4 shows that in all patients with pregnancy toxaemia, there were 91 infants. In the hypoglycaemic group (18 infants), IUGR was present in 12 infants. This represents 10.9% of all infants, 66.7 % of infants from hypoglycaemic mothers and 44.4 % of IUGR infants. In normoglycaemic group (73) infants, IUGR was present in 12 infants. This represents 10.9% of all infants, 16.4 % of infants from normoglycaemic mothers and 44.4 % of IUGR infants. The number of IUGR infants was statistically more in hypoglycaemic group than in normoglycaemic group ($P < 0.001$).

In all anaemic cases, there were 38 infants. In the hypoglycaemic group (11 infants), IUGR was present in 9 infants. This represents 23.7% of all infants, 81.8% of infants from hypoglycaemic mothers and 69.2% of IUGR infants. In the normoglycaemic cases (27 infants), IUGR was present in 4 cases. This represents 10.5% of all infants, 14.8% of infants from normoglycaemic cases and 30.8% of IUGR infants. The number of IUGR infants was statistically more in hypoglycaemic group ($P < 0.001$).

In all cardiac cases, there were 16 infants. In the hypoglycaemic group (5 infants), IUGR was present in 4 infants. This represents 25% of all infants, 80% of infants from hypoglycaemic mothers and 80% of IUGR infants. In normoglycaemic group (11 infants) IUGR was present in one case which represents 6.3% of all infants, 9.1% of infants from normoglycaemic mothers and 20% of IUGR infants. The number of IUGR infants was statistically more in the hypoglycaemic group ($P = 0.01$).

Table 5 shows the mean maternal fasting blood glucose ($\pm$ S.D.), with and without IUGR, in patients with pregnancy toxaemia, single risk factors for IUGR and multiple risk factors for IUGR.

Table 5. Mean maternal fasting blood glucose (± SD) in patients with pregnancy toxaemia, single and multiple risk fators for IUGR.

Patients	Mean fasting blood glucose(mg/dl)		P-value
	IUGR	No IUGR	
All cases with pregnancy toxaemia* (n=91)	59.7±7.42	76.79±15.39	= 0.001
Cases with single risk factors for IUGR (n=76)	65.53±14.63	78.13±12.07	< 0.05
Cases with multiple risk factors for IUGR (n=34).	68.33±16.87	82.67±13.74	< 0.001

* Alone are with anaemia and or cardiac disease.

This table shows that in pregnancy toxaemia the mean fasting blood, glucose was statistically lower in cases with IUGR infants (P=0.001) In cases with single risk factors for IUGR, the mean fasting blood glucose was also statistially lower in cases with IUGR infants ($P < 0.05$). Moreover, in cases with multiple-risk factors for IUGR, the mean fasting blood glucose level was also statistically lower in cases with IUGR infants ($P < 0.001$).

Table 6. Shows the range and mean (± SD) maternal blood glucose levels (fasting and 10 and 60 minutes after IV glucose load) in infants, with and without IUGR. Results are also demonstrated in Figure. I

Table 6: Range and mean (± SD) maternal blood glucose level in infants with and without IUGR.

Blood glucose(mg/dl)	IUGR		no IUGR		P-value
	Range	Mean(±SD)	Range	Mean(±SD)	
Fasting	54-109	66.9259 ± 15.5636	54-109	80.3976 ± 14.3747	< 0.0001
10 minutes after IV glucose.	98-160	119.7778 ± 20.9658	98-160	134.1687 ± 16.7460	< 0.001
60 minutes after IV glucose	70-120	80.0741 ± 13.8117	70-120	93.0964 ± 14.0978	< 0.0001

This table shows that the maternal fasting blood glucose level ranged between 54-109 mg/dl in all the study patients. The mean maternal fasting blood glucose in cases without IUGR was 80.4 ± 14.4 mg/dl, compared to a mean of 66.9 ± 15.6 mg/dl in cases with IUGR. This difference was statistically significant. ($P < 0.0001$).

The maternal blood glucose level 10 minutes after IV glucose load ranged between 98-160 mg/dl in all the study patients. The mean maternal blood glucose level in cases without IUGR was 134.2 ± 16.8mg/dl, compared to a mean of 119.8±21.0 in cases with IUGR. this difference was also statistically significant ($P < 0.001$).

The maternal blood glucose level 60 minutes after IV. glucose load ranged between 70-120 mg/dl in all study cases. The mean maternal blood glucose level in cases without IUGR was 93.6 ± 14.1 mg/dl, compared to a mean of 80.1 ± 13.8 in cases with IUGR. This difference was also statistically significant ($P < 0.0001$).

Fasting blood glucose level was catigorized into 3 groups:

G_A: Included patients with a fasting blood glucose less than 60 mg/dl (22 infants).

G_B Included patients with a fasting blood glucose between 60-79 mg/dl (44 infants).

G_C Included patients with a fasting blood glucose between 80-110 mg/dl (44 infants)

Table 7 shows the number of IUGR infants in each of the three groups.

Table 7: Number of IUGR infants in relation to fasting blood sugar:

Group	G_A	%	G_B	%	G_C	%	P-value
$\frac{\text{IUGR}}{\text{total}}$	$\frac{14}{22}$	63.64	$\frac{10}{44}$	22.73	$\frac{3}{44}$	6.82	< 0.0001
	$\frac{14}{22}$		$\frac{10}{44}$		-		$= 0.001$
	$\frac{14}{22}$		-		$\frac{3}{44}$		< 0.0001
			$\frac{10}{44}$		$\frac{3}{44}$		> 0.05

This table shows that the difference in the number of IUGR between the 3 groups was statistically significant ($P < 0000.1$). The number of IUGR was statistically more in group A compared to group B ($P = 0.001$). The number of IUGR infants was also statistically more in group A. compared to group C ($P < 0.00001$). However, the number of IUGR infants was not statistically more in group B compared to group C ($P > 0.05$).

Blood glucose level, 10 minutes after IV glucose load was also catigorized into 3 groups.

G_A: Included cases with blood glucose level less than 100 mg/dl (7 infants)

G_B: Included cases with blood glucose level between 100-129 mg/dl (41 infnts).

G_C: Included cases with blood glucose level between 130-160 mg/dl (62 infants):

Table 8 Shows the number of IUGR infants in each of the 3 groups

Table 8: Number of IUGR infants in relation to blood glucose level 10 minutes after I V. glucose load.

Groups	G_A	%	G_B	%	G_C	%	P-value
IUGR / Total	$\frac{6}{7}$	85.7%	$\frac{11}{41}$	26.83	$\frac{10}{62}$	16.1%	< 0.0001
	$\frac{6}{7}$		$\frac{11}{41}$		-		< 0.01
	$\frac{6}{7}$		-		$\frac{10}{62}$		< 0.001
	-		$\frac{11}{41}$		$\frac{10}{62}$		> 0.05

This table shows that the difference in the number of IUGR infants between the 3 groups was statistically significant ($P < 0.0001$). The number of IUGR infants was statistically more in G_A, compared to G_B ($P < 0.01$). The number of IUGR was also statistically more in G_A compared to G_C ($P < 0.001$). However, the number of IUGR was not statistically more in G_B compared to G_C ($P > 0.05$).

Blood glucose level 60 minutes after IV glucose load was catigorized into 3 groups:

G_A: Included cases with blood glucose level less than 80 mg/dl (35 infants)

G_B: Included cases with blood glucose level between 80-99 mg/dl (59 infants).

G_C: Included cases with blood glucose level between 100-120 mg/dl (16 infants).

Table 9 shows the number of IUGR infants in each of the 3 groups.

Table 9: The number of IUGR infants in relation to blood glucose level 60 minutes after IV-glucose load.

Group	G_A	%	G_B	%	G_C	%	P-value
IUGR / Total	21/35	60	4/59	6.68	2/16	2.5	< 0.0000001
	21/35		4/59		-		< 0.00001
	21/35		-		2/16		< 0.01
	-		4/59		2/16		> 0.05

This table shows that the difference between the 3 groups was statistically significant ($P < 0.0000001$). The number of IUGR infants was statistically more in group A compared G_B. ($P < 0.00001$). The number of IUGR infants was also statistically more in G_A compared to G_C ($P < 0.01$). However the number of IUGR infants was not statistically more in G_B compared to G_C ($P > 0.05$).

From table 7,8 and 9 the significant values for blood glucose levels in IUGR infants were:

- A fasting level less than 60 mg/dl.
- A 10 minutes after IV. glucose load level less than 100 mg/dl.
- A 60 minutes after IV. glucose load level less than 80 mg/dl.

To evaluate the accurracy of these significant values of blood glucose levels (Fasting, 10 minutes after IV glucose load and 60 minutes after IV. glucose load), further 5 statistical parameters were calculated (Stempel, 1982).

1) Sensitivity: This is the fraction of patients with an abnormal condition who have an abnormal test result $\frac{\text{True +ve}}{\text{True +ve + False +ve}}$

2. Specificity: This is the fraction of patients with a normal condition who have a normal test result $\frac{\text{True -ve}}{\text{True -ve + False -ve}}$

3) Predective value of an abnormal test (+ve result): This is the fraction of patients with an abnormal test result who have an abnormal condition $\frac{\text{true +ve}}{\text{True +ve + False -ve}}$

4) predective value of a normal test (-ve result): This the fraction of patients with a normal test result, who have a normal condition $\frac{\text{true -ve}}{\text{True -ve + False +ve}}$

5) Effeciency: This $\frac{\text{true +ve + true -ve}}{\text{True+ve +true -ve+False +ve+False-ve(total)}}$

Table 10 shows the comparative accuracy of glucose level in fasting, 10 minutes and 60 minutes after IV-glucose load blood samples to detect IUGR when IV. glucose tolerance was done.

Table 10: Comparative accuracy of various blood glucose levels to detect IUGR.

Blood glucose	Specificity	Sensitivity	Predective values		Effec-iency
			+ve result	-ve result	
1) Fasting	90.36%	51.85%	63.64%	85.23	88.91%
2) 10 minutes after IV.glucose load.	98.8%	22.22%	85.71%	79.61%	80:00%
3) 60 minutes after IV glucose load.	83.13%	77.78%	60.00%	82.00%	81.82%

This table shows that the specificity of the IV. glucose tolerance test was highest 10 minutes after IV-glucose load (98.8%), followed by fasting glucose level (90.36%) then by the level of 60 minutes after IV glucose load (83.13%).

Sensitivity of the test was highest 60 minutes after IV. glucose load (77.78%), followed by the fasting glucose (51.85%) and was least (22.22%) 10 minutes after IV. glucose load.

As regards the effeciency, fasting blood glucose had the highest effeciency 88.9%, followed by 60 minutes IV. glucose test (81.82%), then by 10 minutes after IV. glucose load 80% which had the lowest effeciency.

As regards the predective values, fasting blood glucose test had the highest predective value of -ve result (85.23%), with about 15% false -ve result. Therefore, fasting blood glucose test was the best -ve test that can predect healthy babies. Ten minutes glucose level after IV. glucose load

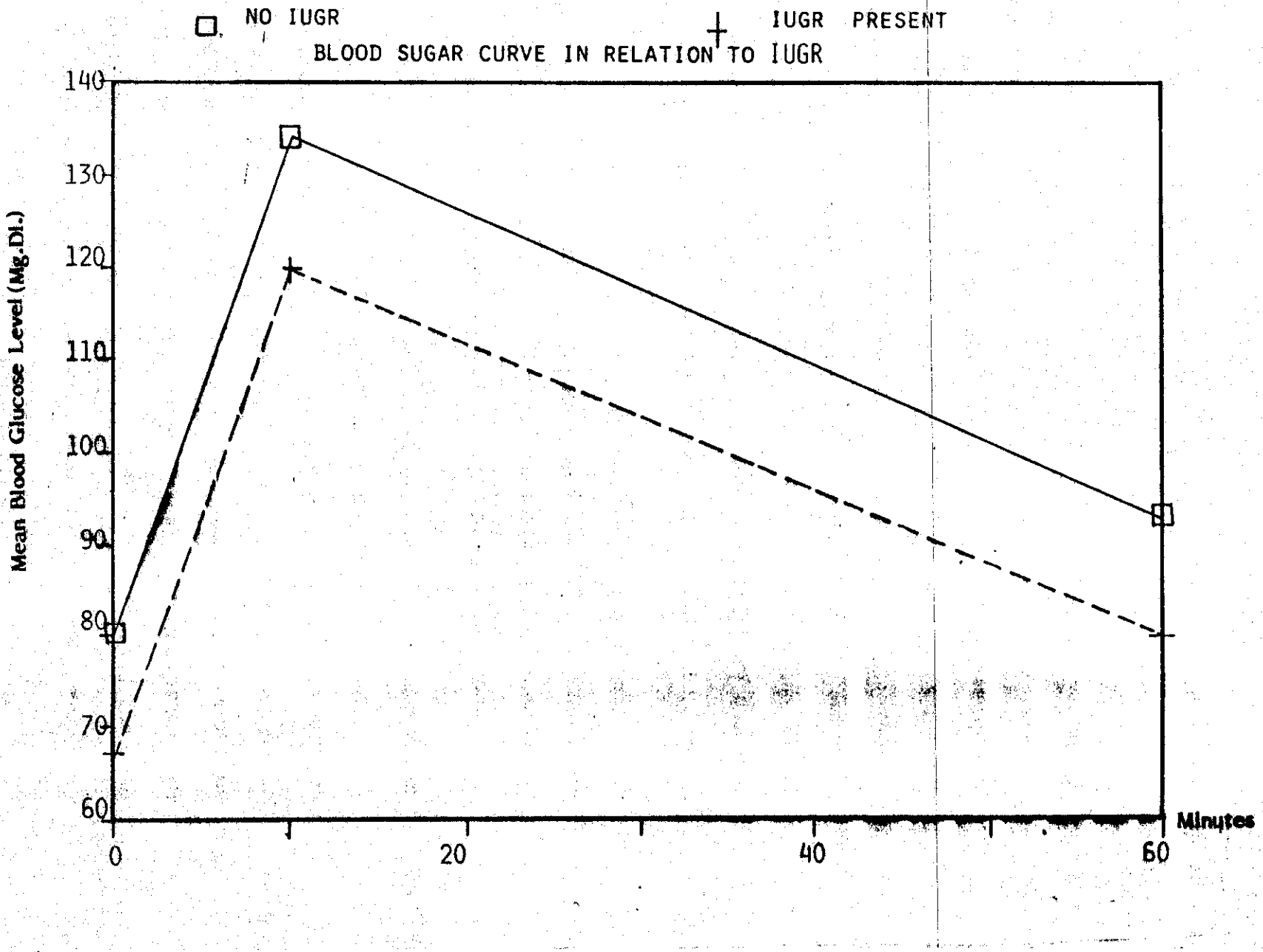
had the highest predective value of +ve result (85.71%), with a false +ve result around 14%. Therefore, the 10 minutes glucose level after IV. glucose load was the best +ve test that can predect IUGR infants.

Table 11: Shows the range of HB level, Mean (± SD) and P-value in patients with and without IUGR.

Table 11: Haemoglobin level range, mean ± (SD) and P-value.

Fetal outcome	Hb. range	Mean ± SD	P-value
IUGR	52 - 80%	61.0741 ± 6.0251	< 0.01
no IUGR	52 - 80%	65.0.602 ± 6.707	

In this table, the mean Hb% in patients with IUGR infants was 61.07 ± 6.03%, compared to 65.06 ± 6.71 in those with no IUGR. The mean Hb% was significantly lower in patients with IUGR infants (P < 0.01).


NO IUGR
IUGR PRESENT
BLOOD SUGAR CURVE IN RELATION TO IUGR
Mean Blood Glucose Level (Mg.Dl.)
140
130
120
110
100
90
80
70
60
0
20
40
60
Minutes

by restriction of basic substrate necessary for normal fetal growth and that simple intravenous dextrose thera y may be a simple method of treatment of IUGR before delivery and may improve pregnancy outcome. However, Abell (1979), found that hypertonic dextrose therapy was regarded as successful only if urinary oestriol excretion returned to, and remained at normal levels (41.9 percent) ($P < 0.05$). Hypertonic dextrose therapy administered to a patient with persistantly subnormal urinary oestriol excretion was less likely to cause a favourable outcome. This would suggest that the adverse effects of abnormal glucose tolerance were early, possibly on the placenta and not correctable in third trimester.

Sabata (1979), performed oral glucose tolerance test in women with small for dates fetuses and in controls with healthy fetuses in the 38th weeks of prelgnancy, 2 days and 6 weeks after delivery. Two hours after an oral loading dose of 50 gm glucose, the blood glucose level remained significantly higher in pregnant women with small for dates fetuses ($P < 0.05$). This could be explained by the facts that in small for date the passage of glucose across the placental membrane and its retention by the fetus is decreased.

Khouzami et al. (1981), also demonestrated that maternal hypo-glycaemia was significantly associated with IUGR and that 3-hours glucose tolerance test was an effective early screening test in detecting pregnancies at risk of IUGR. In order to assess the value of the GTT as a clinical tool in predecting the low birth weight and non low birth weight IUGR outcome, the sensitivity, the specificity and positive and negative predective values of hypoglycaemia were determined

for low birth weight IUGR and non-LBW IUGR groups. In absence of hypoglycaemia, there was 90% probability that the pregnancy will not result in either form of IUGR. However, in the presence of hypoglycaemia only 15% will result in LBW IUGR, but approximately 55% will result in non LBW IUGR. The sensitivity is around 15% for LBW IUGR and increases to 55% for non LBW. IUGR.

To eliminate the gastrointestinal factors, Sokol et al. (1982), measured blood glucose after intravenous glucose adminstration in the third trimester. Glucose 25 gm was injected into the antecubital vein. Blood samples were obtained before and every 10 minutes after the glucose was injected for a period of one hour. They found that infants birth weight, adjusted for gestational age, was significantly related to maternal glucose metabolism. Increased glucose utilization rate (determined from the slope of a graph of log of glucose concentration against time) and 10 minutes plasma glucose concentration and decreased plasma glucose concentration at fasting and 60 minutes were found to be associated with decreased infants birth weight adjusted for gestational age.

In our study, patients were classified into those with single risk factors for IUGR(71 patients gave birth to 76 infants including 5 twins)and those with multiple risk factors for IUGR(31 patients gave birth to 34 infants including 3 twins).The normoglycaemic state provided a control against which the effect of hypoglycaemia on fetal outcome could be evaluated. Infants birth weight, adjusted for gestational gestational age, was found to be significantly related to maternal glucose metabolism, as measured by third trimester intravenous glucose tolerance test.

In patients with a single risk factors for IUGR there was no statistically significant difference between the number of IUGR infants in those with hypoglycaemic compared to those with normoglycaemic mothers ($P < 0.05$) (Table 3). However, the mean fasting blood glucose level in patients with IUGR infants was statistically lower than in those with no IUGR in these group ($P < 0.05$) (Table 5).

In cases with multiple risk factors for IUGR, the number of IUGR infants was statistically higher in hypoglycaemic patients (P 0.0001) (Table 3). Moreover the mean fasting blood glucose level in patients with IUGR infants was statistically lower than in those with no IUGR in these group ($P < 0.001$) (Table 5).

We found that the mean fasting blood glucose and blood glucose level 10 and 60 minutes after intravenous glucose load was significantly lower in patients with IUGR ($P < 0.0001$, $P < 0.001$ and $P < 0.0001$ respectively) (Table 6 and Fig.1). We found that IUGR was present in 63.64% of infants from hypoglycaemic patients (14 out of 22 infants) compared to 14.77% of infants from normoglycaemic patients 13 out of 88 infants), meaning that the number of IUGR infants was statistically higher in the hypoglycaemic group than in the normoglycaemic group (P 0.0001) (Table 7).

Long et al. (1980), showed that, the prevel nce of fetal growth retardation was not significantly increased in patients with preeclampsia compared to his total hospital population. However, when analyzed according to the time of onset of preeclampsia. They found that there was a sharp increase in the occurrance of IUGR(18% in patients with early onset of preeclampsia) and a reduced occurrance (6%)

in those with late onset of preeclampsia. Also, in patients who subsequently developed early onset preeclampsia with fetal growth retardation, hypoglycaemia was found in 33% which was significantly higher than in all patients who subsequently developed preeclampsia 14% ($P < 0.001$).

In our study, when we analyzed the risk factors either single or multiple in relation to hypogycaemia in the mothers and IUGR in infants we found that:

- In patients with pregnancy toxaemia only (56 patients), there were 61 infants (including 5 twins) and IUGR was present in 12 infants (19.67%). From hypoglycaemic patients IUGR was present in 4 out of 9 infants (44.44%) compared to 8 out of 52 infants from normoglycaemic mothers (15.38%), which was not statistically significant difference ($P > 0.05$). Therefore, hypoglycaemia was not a factor associated with the occurrance of IUGR in patients with pregnancy toxaemia only (Table 3).

- In patients with pregnancy toxaemia associated with cardiac disease and/or anaemia(28 patients),there were 30 infants(including 2 twins)and IUGR was present in 12 infants (40%). From hypoglycaemic patients , IUGR was present in 8 out of 9 infants (90.9%) compared to 4 out of 21 infants from normoglycaemic mothers(19.05%),which was high statistically a significant difference ($P < 0.0001$) (Table 3). Therefore, hypoglycaemia was a factor associated with the occurrance of IUGR in patients with pregnancy toxaemia when associated with other risk factors.

- In all patients with pregnancy toxaemia,either only or associated with other risk factors(84 patients), there were 91 infants (including 7 twins) & IUGR was present in 24 infants (26.37%). From hypoglycaemic patients IUGR was present in 12 out of 18(66.67%) infants compared to 12 out of 73 infants from normoglycaemic mothers (16.44) which statistically a significant difference ($P<0.001$) (Table 4).

A low maternal haemoglobin concentration has been implicated in the genesis of fetal growth retardation. IUGR is commonly seen in fetuses of women with sickle cell disease and with anaemia associated with serious maternal disease (Prichard and MacDonald, 1985).

In our study, the mean haemoglobin concentration was significantly lower in cases with IUGR ($P < 0.01$)(table. 11). all anaemic cases either alone or associated with other risk factors(35 patients), there were 38 infants(including 3 twins)& IUGR was present in 13 cases.In hypoglycaemic cases, IUGR was present in 9 out of 11 infants (81.82%), compared to 4 out of 27 infants in the normoglycaemic group(14.81%), which was a statistically significant difference ($P < 0.001$)(Table 4).

In the 9 patients with anaemia only, there was no IUGR in both hypoglycaemic (one patient) and normoglycaemic (8 patients). Therefore, anaemia appears to represent a significant cause in IUGR, if associated with other maternal disease such as, preeclampsia or cardiac disease.

Any disease complicated by severe maternal hypoxia are much more likely to lead to delivery of small for date fetuses e.g.

fetuses of women with cyanotic heart disease of pulmonary insufficiency (Prichard and Mac Donald, 1985). However, fetal outcome in rheumatic heart disease in pregnancy is usually very good. (Michael de Swiet, 1984).

In our sltudy, in caridac patients (15) there were 16 infants (including one twin)(all are rheumatic heart disease). IUGR was present in 4 out of 5 infants in hypoglycaemic cases(80 percent)compare to 9.1 percent(1/11 in infants)from normoglycaemic mothers.The number of IUGR was statistically more in the hypoglycaemic group (P = 0.01), most probably due to association with other maternal diseases.

From our study, we agree with most workers that hypoglycaemia may be a causative factor in IUGR in patients with a high risk pregnancy, and IVGTT is a good tool in predecting IUGR in these patients. To evaluate a test which can be used as a screening test for IUGR in high risk pregnancy, specificity, sensitivity, effeciency and predective values of +ve and -ve results were calculated for the blood glucose level in the fasting, 10 and 60 minutes after 25 gm IV. glucose load.

- Fasting blood glucose level had the highest effeciency (88.91%) and the highest predictive value of -ve result (85.23%) So it is a good negative test that can detect 85.23% of healthy infants. It had a false -ve result around 15%.

- Ten minutes after IV. glucose injection blood glucose had the highest specificity (98.8%) and the highest predective value

of +ve result (85.71%). Therefore, it is a good +ve test that can detect 85.23 percent of IUGR. It had a false +ve result around 14%.

- Sixty minutes after IV. glucose injection blood glucose, had the highest sensitivity (77.78) and its predective value of negative result was 82% (i.e. between the other two tests). However, it had the lowest specificity, predective values of +ve result and effeciency.

Therefore, fasting blood glucose is a good -ve test as it had a false -ve result around 15%. However, if it is +ve (less than 60 mg/dl) it has a false negative result of a 37%. Therefore, it needs to be backed by the 10 minutes blood glucose level which has a false +ve result around 14%, and so it increases the accurray of fasting blood glucose test by 23%.