RESULTS

STATISTICAL ANALYSIS (Hill, 1977).

Statistical analysis was carried out by the following measurements:

1} Standard deviation (S.D.) and arithmetic mean "X":

$$\bar{X} = \frac{\Sigma X}{n}$$

Where X = variable

Σx = sum of x.

n= number of variables.

$$S.D. = \sqrt{\frac{(X-\bar{X})^2}{n-1}}$$

X = variable

$\bar{X}$ = mean of variables.

n = number of variables.

2} Student's "t" test:

$$t = \frac{\bar{X}_1 - \bar{X}_2}{\sqrt{\frac{SD_1^2}{n_1} + \frac{SD^2}{n_2}}}$$

From "t" tables, the probability "p" can be estimated taking 99.75% as the level of significance. The results can be tabulated as highly significant if P<0.0125, < 0.01, < 0.005, significant if P<0.05 <0.025; insignificant if P > 0.05.

3. Person's correlation coefficient "r":

$$r = \frac{Xy - \frac{(X)(y)}{n}}{\sqrt{X^2 - \frac{(X^2)}{n} \quad X \; Y^2 - \frac{(-y)^2}{n}}}$$

Where X and Y are two variable of the same group.

In order to find out, whether the "r" is significant or not"t" for "r" is calculated from the formula.

$$t = \frac{r\sqrt{n-2},}{\sqrt{1-r^2}}$$

P_1 : Statistical significance between diabetic patients and controls.

P_2: Statistical significance between diabetic uremic patients on regular hemodialysis and controls.

P_3: Statistical significance between diabetic uremic patients on conservative treatment and controls.

P_4: Statistical significance between uremic patients on regular hemodialysis and controls.

ANALYSIS OF RESULTS

Table (1) and (2) show age and sex distribution among all the studied groups.

Table (3) shows the hemoglobin level among the studied groups. In the control group (V) it ranges from 11.6-14.7 with a mean of 13.4 ± 1.06 g/dl (mean ± S.D).

In the group (I) it ranges from 10.9 - 14.7 with a mean of 12.9 ± 1.16 g/dl ($P > 0.05$)

In the group (II) it ranges from 9-15 with a mean of 10.9 ± 2.04 g/dl which is significantly lower than the corresponding mean of the control group ($P < 0.05$).

In group (III) it ranges from 6.5 - 13.3 with a mean of 10.2 ± 2.28 g/dl which is significantly lower than the corresponding mean of the control group ($P < 0.05$).

In group (IV) it ranges from 6.5 - 10.9 with a mean of 9.2 ± 1.26 g/dl which is significantly lower than the corresponding mean of the control group ($P< 0.05$).

Table (4) shows the fasting serum glucose level (mmol/l) among the studied groups.

In group (V) it ranges from 3.850 - 5.225 with a mean of 4.4 ± 0.37 mmol/l.

In group (I) it ranges from 7.15 - 17.75 with a mean of 11.4 ± 3.72 mmol/l which is significantly higher than the corresponding mean of the control group ($P < 0.05$).

In group (II) it ranges from 8.25 - 16.995 with a mean of 12.3 ± 3.22 mmol/l which is significantly higher than the corresponding mean of the control group ($P < 0.01$).

In group III it ranges from 9.35 - 20.13 with a mean of 16.1 ± 3.26 mmol/l which is significantly higher than the corresponding mean of the control group ($P < 0.01$).

In group (IV). it ranges from 3.465 - 4.895 with a mean of 4.2 ± 0.44 mmol/l ($P > 0.05$).

Table (5) shows the post prandial serum glucose level (mmol/l) among the studied groups.

In group (V) it ranges from 5.50 - 6.93 with a mean of 6.1 ± 0.47 mmol/l.

In group (I) it ranges from 11.66 - 19.525 with a mean of 14.6 ± 2.86 mmol/l which is significantly higher than the corresponding mean of the control group ($P < 0.01$)

In group (II) it ranges from 12.925 - 21.395 with a mean of 16.3 ± 2.93 mmol/l which is significantly higher than the corresponding mean of the control group ($P < 0.01$).

In group (III) it ranges from 13.75 - 22.00 with a mean of 19.4 ± 2.83 mmol/l which is significantly higher than the corresponding mean of the controls ($P < 0.01$).

In group (IV) it ranges from 5.555 - 7.315 with a mean of 6.4 ± 0.54 mmol/l (P > 0.05).

Table (6) shows serum total protein level (g/l) among the studied groups.

In group (V) it ranges from 61-75 with a mean of 65 ± 4.48 g/l.

In group(I) it ranges from 58-72 with a mean of 65.4 ± 5.44 g/l (P > 0.05).

In group (II) it ranges from 55-70 with a mean of 62.8 ± 4.80 g/l (P > 0.05).

In group (III) it ranges from 51 - 72 with a mean of 63 ± 6.94 g/l (P > 0.05).

In group (IV) it ranges from 57 - 76 with a mean of 63.2 ± 5.86 g/l (p> 0.05).

Table (7): Shows serum creatinine level (mmol/l) among the studied groups.

In group (V) it ranges from 0.0796 - 0.1238 with a mean of 0.10 ± 0.016 mmol/l.

In group (I) it ranges from 0.0796 - 0.1414 with a mean of 0.11 ± 0.020 mmol/l (P > 0.05).

In group (II) it ranges from 0.8221 - 1.5028 with a mean of 1.17 ± 0.2 mmol/l which is significantlly higher than the corresponding mean of the control group (P < 0.01).

In group (III) it ranges from 0.3359 - 0.8840 with a mean of 0.64 ± 0.235 mmol/l which is significantly higher than the corresponding mean of the controls ($P < 0.01$).

In group (IV) it ranges from 1.3260 - 2.21 with a mean of 1.82 ± 0.366 mmol/l which is significantly higher than the corresponding mean of the controls ($P < 0.01$).

Table (8) shows glycosylated hemoglobin level (%) among the studied groups.

In group (V) it ranges from 6.7 - 9.5 with a mean of 7.72 ± 0.783%.

In group (I) it ranges from 10.6 - 12.6 with a mean of 11.5 ± 0.604% which is significantly higher than the corresponding mean of the control group ($P < 0.01$).

In group (II) it ranges from 5.3 - 11.2 with a mean of 9.08 ± 2.167% which is significantly higher than the corresponding mean of the controls ($P < 0.05$).

In group (III) it ranges from 7 - 10.6 with a mean of 9.05 ± 1.166% which is significantly higher than the corresponding mean of controls ($P < 0.05$).

In group (IV) it ranges from 4.6 - 7.5 with a mean of 6.62 ± 0.94% which is significantly lower than the corresponding mean of the control group ($P < 0.05$).

Table (9) shows serum fructosamine level (mmol/l) among the studied groups.

In group (V) it ranges from 1.29 - 1.69 with a mean of 1.43 ± 0.145 mmol/l.

In group (I) it ranges from 2.4 - 3.06 with a mean of 2.75 ± 0.286 mmol/l which is significantly higher than the corresponding mean of the control group ($P < 0.01$).

In group (II) it ranges from 1.5 - 2.4 with a mean of 2.03 ± 0.258 mmol/l which is significantly higher than the corresponding mean of the controls ($P < 0.01$).

In group (III) it ranges from 1.48 - 2.5 with a mean of 2.04 ± 0.354 mmol/l which is significantly higher than the corresponding mean of the control group ($P < 0.01$).

In group (IV) it ranges from 1.35 - 1.8 with a mean of 1.52 ± 0.151 mmol/l ($P > 0.05$).

Table (10) shows correlation studies of GHb and its related parameters (F.B.S, P_p. B.S, Hb). There are positive correlations between GHb and both fasting and postprandial blood sugar which are significant only in group I,II,III ($P < 0.05$). Also there is a positive significant correlation between GHb and Hb among all the studied groups ($P < 0.05$).

Table (11) shows correlation studies between GHb and serum creatinine among all the studied groups. There is a negative insignificant correlation between GHb and serum creatinine among all the studied groups ($P > 0.05$).

Table (12) shows correlation studies of serum fructosamine and its related parameters (F.B.S, P_p. B.S., T.P) There are positive significant correlations between serum fructosamine and both fasting and postprandial blood sugar among all the studied groups ($P < 0.05$). Also there is a positive significant correlation between serum fructosamine and T.P among all the studied groups ($P < 0.05$).

Table (13) shows correlation studies between serum fructosamine and serum creatinine among all the studied groups, There is a negative insignificant correlation between serum fructosamine and serum creatinine in group I, II, III and IV but insignificant positive correlation in group V ($P > 0.05$).

Table (14) shows the correlation studies between serum fructosamine and GHb among all the studied groups. There is positive correlation between serum fructosamine and GHb among all the studied groups but it significant only in group I and III ($P < 0.05$).

Table (1): Age distribution of the studied groups.

Groups	< 45 y		45 +		Total	
	No.	%	No.	%	No.	%
I Diabetic group	6	60	4	40	10	100.0
II Diabetic uremic on regular hemodialysis	4	40	6	60	10	100.0
III Diabetic uremic on conservative treatment	2	20	8	80	10	100.0
IV Uremic group on regular hemodialysis	7	70	3	30	10	100.0
V Control group	10	100.0	0	0	0	0
Total	29		21			

Table (2): Sex distribution of the studied groups

Groups	Males		Females		Total	
	No.	%	No.	%	No.	%
I	4	40	6	60	10	100.0
II	7	70	3	30	10	100.0
III	6	60	4	40	10	100.0
IV	8	80	2	20	10	100.0
V	5	50	5	50	10	100.0
Total	30		20			

Table (3): Hemoglobin level (g/dl) among the studied groups

Group	I	II	III	IV	V
1	11.9	9.1	10.5	9.1	14.2
2	12.3	15.0	11.6	9.7	11.6
3	14.3	10.1	13.3	10.5	12.9
4	10.9	10.0	6.5	10.9	12.3
5	12.2	13.8	11.6	9.1	14.7
6	14.7	9.4	6.9	10.2	12.6
7	13.1	9.8	9.4	8.4	14.7
8	12.6	9.0	11.2	9.1	13.6
9	13.1	10.9	8.7	8.5	14.3
10	13.8	11.5	12.4	6.5	13.4
Mean $\bar{X}$	12.9	10.9	10.2	9.2	13.4
± S.D	1.16	2.04	2.28	1.26	1.06
P_1	>0.05	--	--	--	--
P_2	--	<0.05*	--	--	--
P_3	--	--	<0.05*	--	--
P_4	--	--	--	<0.05*	--

* Denotes statisticaly significant difference at the 0.05 level

Table (4): Fasting serum glucose level (mmol/l) among the studied groups.

Group	I	II	III	IV	V
1	14.520	11.825	16.50	4.345	4.675
2	10.175	16.995	15.40	4.565	4.125
3	17.215	12.595	17.60	3.74	4.400
4	8.910	9.350	9.350	4.015	5.225
5	9.350	15.900	17.60	4.29	4.510
6	17.750	12.650	11.66	4.565	4.125
7	7.150	9.350	17.325	3.850	4.235
8	10.065	8.250	17.050	4.895	3.850
9	8.800	9.900	18.700	3.905	4.455
10	9.900	16.555	20.130	3.465	4.510
$\bar{X}$	11.4	12.3	16.1	4.2	4.4
±S.D	3.72	3.22	3.26	0.44	0.37
P1	<0.05*	--	--	--	--
P2	--	<0.01*	--	--	--
P3	--	--	<0.01*	--	--
P4	--	--	--	>0.05	--

Table (5): Post prandial serum glucose level (mmol/l) among the studied groups.

Group	I	II	III	IV	V
1	16.280	13.200	22.000	6.215	6.325
2	14.025	21.395	18.865	6.325	5.610
3	19.525	18.150	20.350	5.555	5.940
4	11.660	13.200	13.750	6.325	6.930
5	12.650	19.100	19.250	6.490	6.325
6	18.950	17.600	15.400	6.655	6.050
7	11.715	14.575	21.725	6.050	5.500
8	12.100	12.925	19.415	7.315	5.500
9	14.685	14.850	22.000	6.985	6.600
10	14.850	17.875	21.450	5.665	6.050
$\bar{X}$	14.6	16.3	19.4	6.4	6.1
±S.D	2.86	2.93	2.83	0.54	0.47
P_1	<0.01*	--	--	--	--
P_2	--	<0.01*	--	--	--
P_3	--	--	<0.01*	--	--
P_4	--	--	--	>0.05	--

Table (6): Serum total proteins level (g/L) among the studied groups

Group	I	II	III	IV	V
1	71	70	62	59	66
2	69	62	65	65	61
3	72	68	66	61	61
4	58	63	51	58	75
5	61	63	71	69	70
6	69	61	66	63	62
7	59	57	56	59	64
8	65	55	66	76	62
9	70	61	55	65	65
10	60	68	72	57	63
$\bar{X}$	65.4	62.8	63.0	63.2	65.0
±S.D	5.44	4.80	6.94	5.86	4.48
P_1	>0.05	--	--	--	--
P_2	--	>0.05	--	--	--
P_3	--	--	>0.05	--	--
P_4	--	--	--	>0.05	--

Table (7): Serum creatinine level (mmol/l) among the studied groups

Group	I	II	III	IV	V
1	0.1238	1.4144	0.3890	2.2100	0.1149
2	0.1238	1.2376	0.7072	1.5028	0.1238
3	0.1238	1.0166	0.7624	2.0951	0.1238
4	0.1414	1.0166	0.8840	1.4851	0.1149
5	0.1061	1.1934	0.3978	1.5382	0.1061
6	0.0796	0.8221	0.8752	2.2012	0.0972
7	0.1238	1.1492	0.3359	2.1216	0.1061
8	0.0972	1.2376	0.3713	1.3260	0.0796
9	0.0884	1.5028	0.7956	1.5382	0.0972
10	0.0972	1.0608	0.8652	2.1746	0.0796
$\bar{X}$	0.11	1.17	0.64	1.82	0.10
±S.D	0.020	0.200	0.235	0.366	0.016
P_1	>0.05	--	--	--	--
P_2	--	<0.01*	--	--	--
P_3	--	--	<0.01*	--	--
P_4	--	--	--	<0.01*	--

Table (8): Glycosylated hemoglobin level (%) among the studied groups

Group	I	II	III	IV	V
1	11.7	7.1	9.5	5.5	7.8
2	11.7	11.2	8.5	6.9	6.7
3	12.0	9.7	10.2	7.3	7.5
4	10.6	6.2	7.0	7.3	7.6
5	10.7	11.2	9.6	6.4	9.5
6	12.6	10.8	7.4	7.5	6.9
7	11.3	9.9	8.9	6.7	8.1
8	11.5	5.3	9.9	7.5	7.2
9	11.8	8.7	8.9	6.5	8.1
10	11.1	10.7	10.6	4.6	7.8
$\bar{X}$	11.50	9.08	9.05	6.62	7.72
±S.D	0.604	2.167	1.166	0.940	0.783
P_1	<0.01*	--	--	--	--
P_2	--	<0.05*	--	--	--
P_3	--	--	<0.05*	--	--
P_4	--	--	--	<0.05*	--

Table (9): Serum fructosamine level (mmol/l) among the studied groups.

Group	I	II	III	IV	V
1	3.06	2.00	2.12	1.50	1.59
2	2.90	2.40	1.80	1.62	1.30
3	3.02	2.20	2.33	1.50	1.37
4	2.40	2.10	1.48	1.37	1.69
5	2.50	2.10	2.40	1.59	1.61
6	3.06	2.12	1.60	1.70	1.36
7	2.50	1.70	2.10	1.40	1.35
8	2.62	1.50	2.30	1.80	1.29
9	3.00	2.01	1.80	1.40	1.37
10	2.40	2.19	2.50	1.35	1.34
$\bar{X}$	2.75	2.03	2.04	1.52	1.43
±S.D	0.286	0.258	0.354	0.151	0.145
P_1	<0.01*	--	--	--	--
P_2	--	<0.01*	--	--	--
P_3	--	--	<0.01*	--	--
P_4	--	--	--	>0.05	--

Table (10): Correlation coefficient (r) and probability value (P) of GHB and its related parameters (F.B.S, P_p.B.S, Hb%)

Group	F.B.S.		P_p.B.S.		Hb	
	r	P	r	P	r	P
I	0.7331	<0.05*	0.7848	<0.05*	0.6973	<0.05*
II	0.7731	<0.05*	0.8694	<0.05*	0.6336	<0.05*
III	0.8987	<0.05*	0.7775	<0.05*	0.8907	<0.05*
IV	0.5093	>0.05	0.4256	>0.05	0.8049	<0.05*
V	0.3325	>0.05	0.3482	>0.05	0.7790	<0.05*

Table (11): Correlation coefficient (r) and probability value (P) of GHb and creatinine

Group	r	P
I	- 0.4718	> 0.05
II	- 0.2866	> 0.05
III	- 0.3606	> 0.05
IV	- 0.4075	> 0.05
V	- 0.0595	> 0.05

Table (12); Correlation coefficient (r) and probability value (P) of fructosamine and its related parameters (F.B.S, P_p. B.S,T.P)

Group	F.B.S.		P_p.B.S.		T.P	
	r	P	r	P	r	P
I	0.7113	<0.05*	0.7629	<0.05*	0.9675	<0.05*
II	0.7661	<0.05*	0.7388	<0.05*	0.6411	<0.05*
III	0.8144	<0.05*	0.6590	<0.05*	0.6791	<0.05*
IV	0.8961	<0.05*	0.5812	<0.05*	0.7988	<0.05*
V	0.8407	<0.05*	0.7676	<0.05*	0.9088	<0.05*

Table (13): Correlation coefficient (r) and probability value (P) of fructosamine and creatinine.

Group	r	P
I	- 0.2392	> 0.05
II	- 0.2177	> 0.05
III	- 0.4679	> 0.05
IV	- 0.2960	> 0.05
V	0.3615	> 0.05

Table (14): Correlation coefficient (r) and probability value (P) between GHb and fructosamine.

Group	r	P
I	0.8670	$< 0.05^{*}$
II	0.6057	> 0.05
III	0.9531	$< 0.05^{*}$
IV	0.5233	> 0.05
V	0.5093	> 0.05

Fig. (2): Glycosylated hemoglobin level (%) among the studied groups.

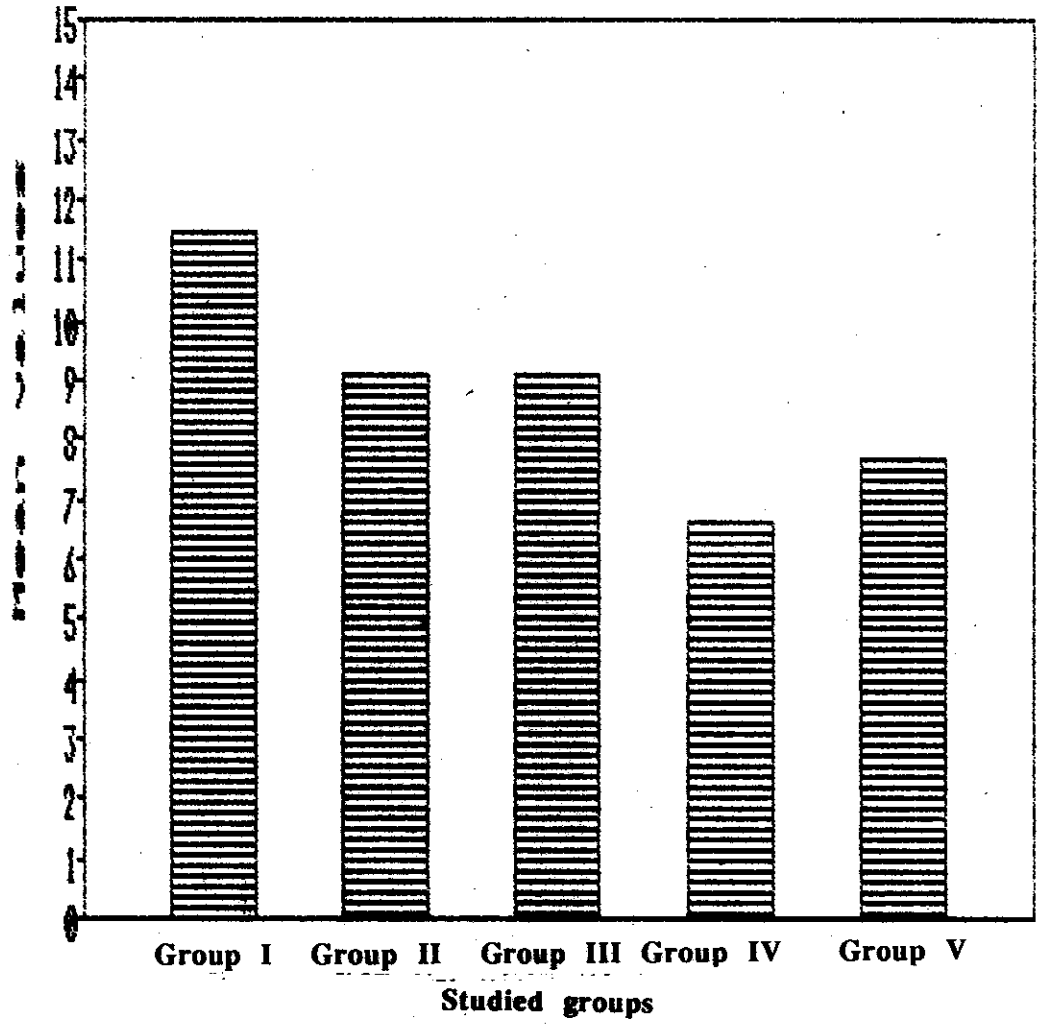


Fig. (3): Relationship between glycosylated hemoglobin level (%) and hemoglobin among group III.

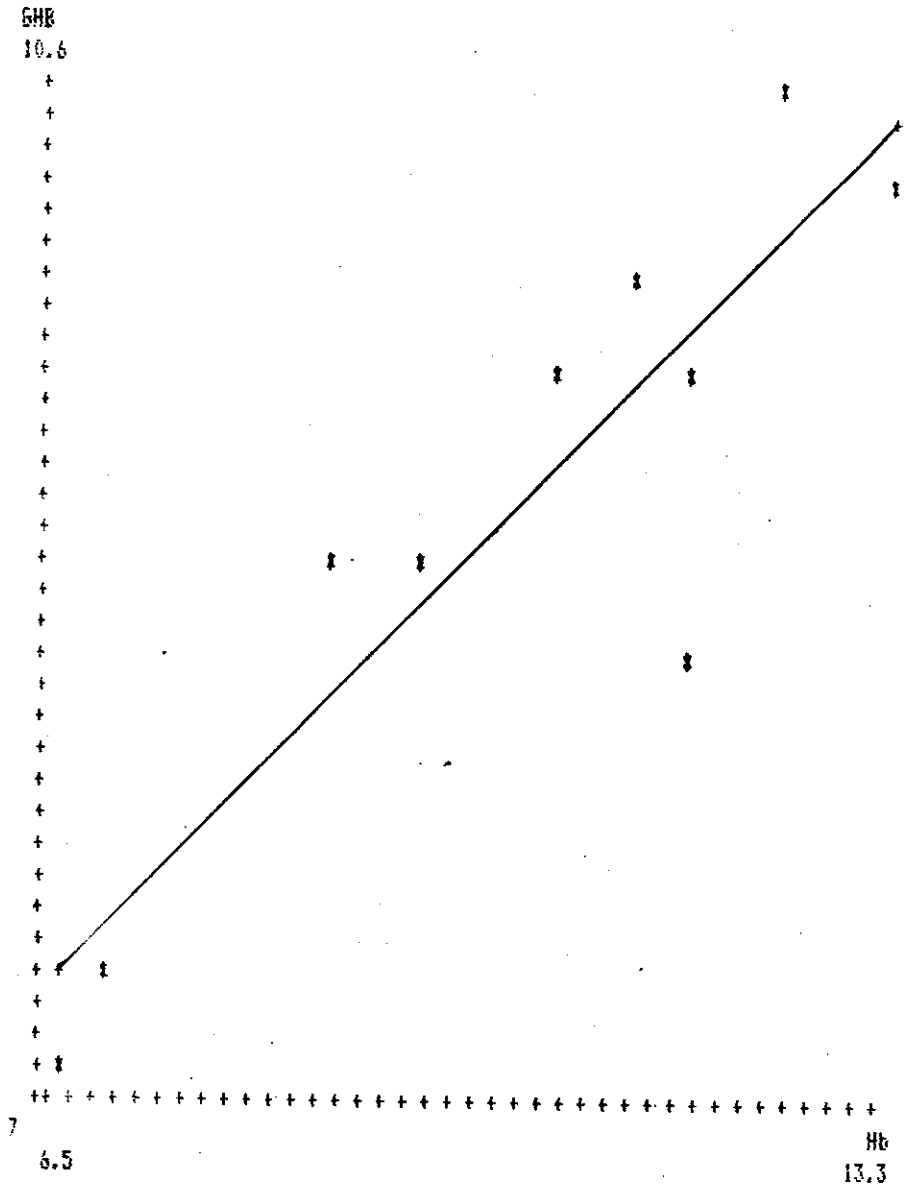


Fig. (4): Serum fructosamine level (m.mol./L) among the studied groups.

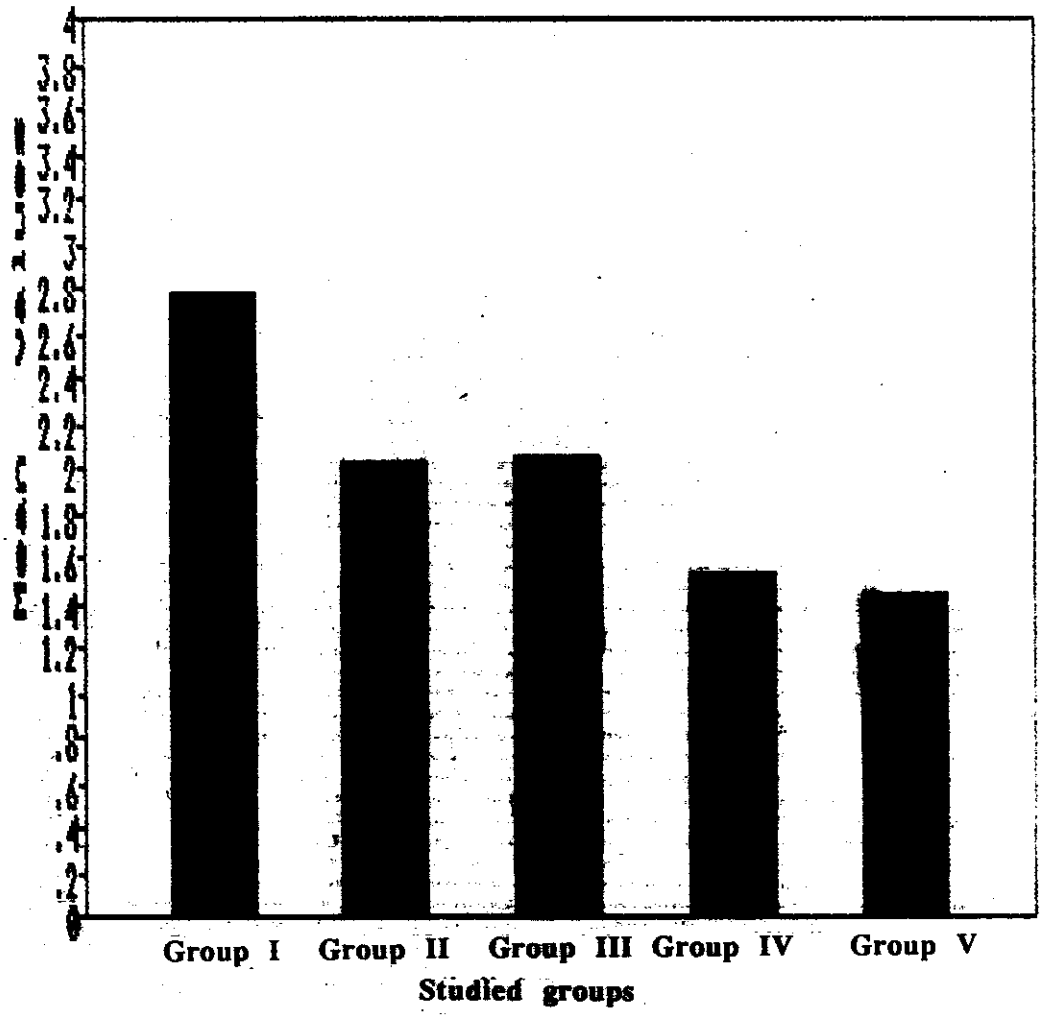


Fig. (5): Relationship between fructosamine and total protein among group I.

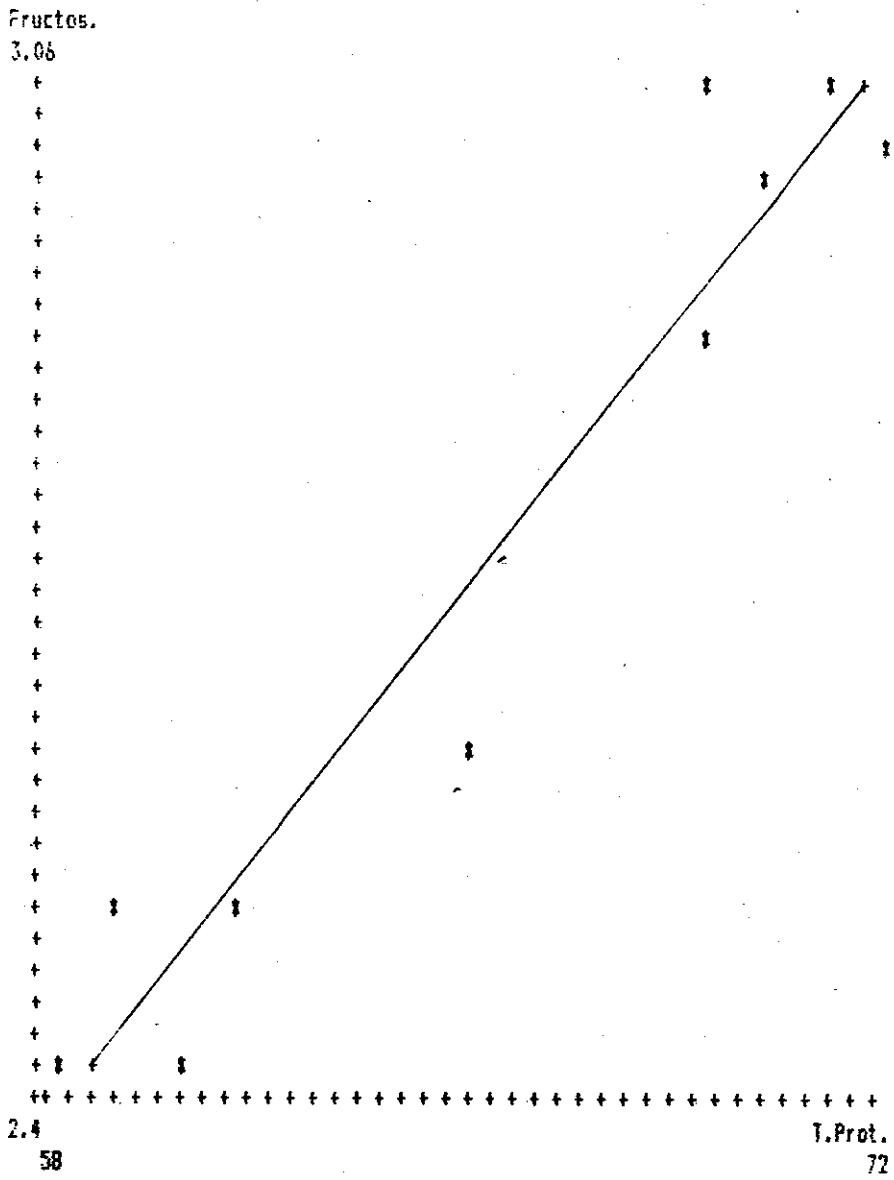


Fig. (6): Relationship between glycosylated hemoglobin level (%) and fructosamine among group I.

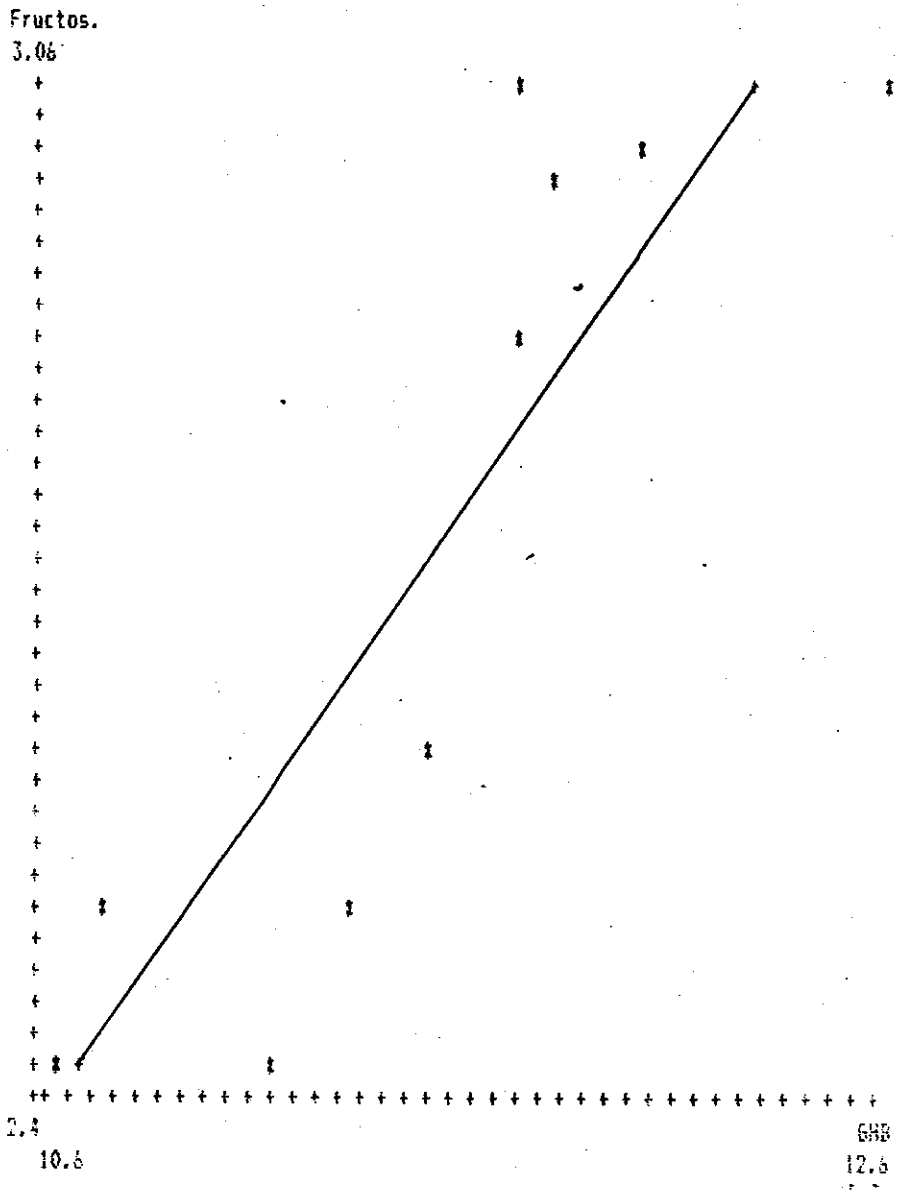
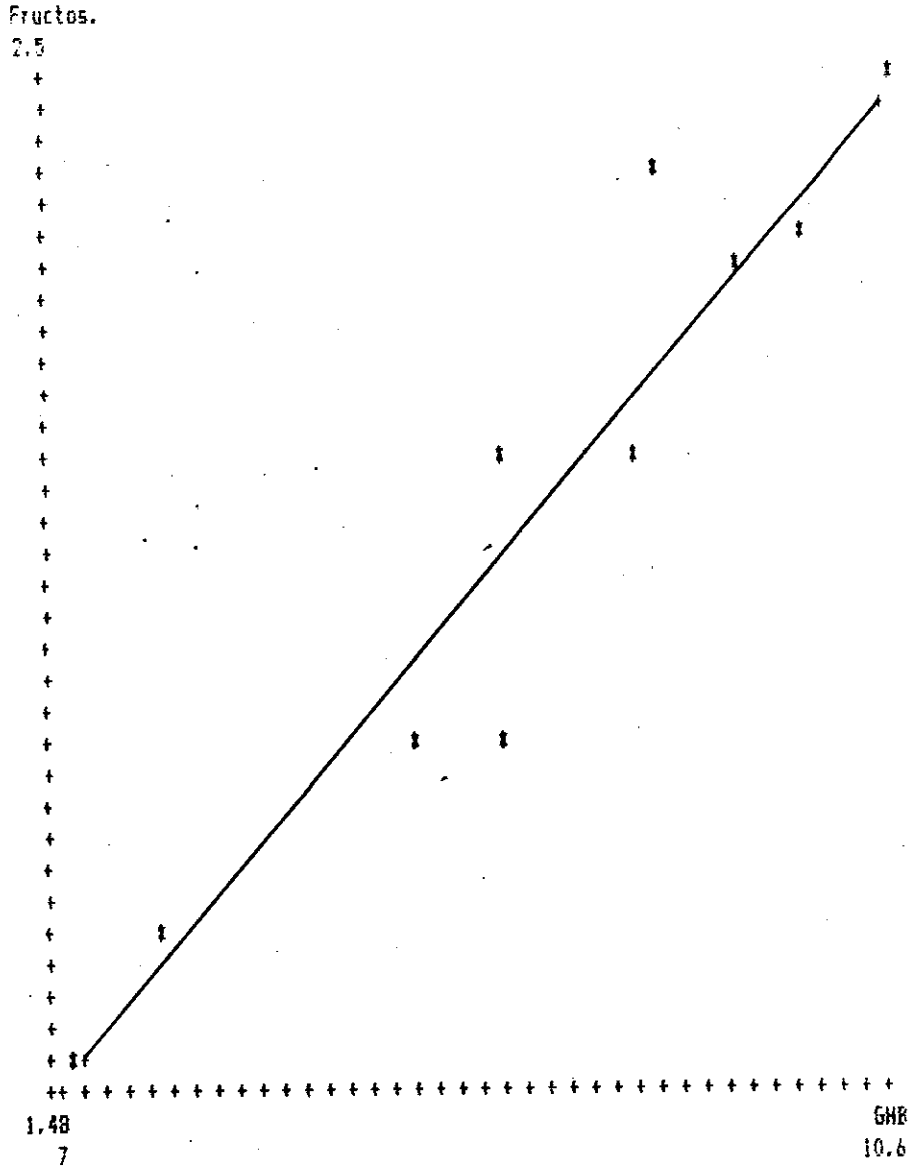


Fig. (7): Relationship between glycosylated hemoglobin level (%) and fructosamine among group III.



DISCUSSION

Most patients with chronic renal failure display glucose intolerance *(Kerr, 1979)*. The mechanisms underlying this glucose intolerance have not been agreed upon but both insulin resistance and impaired insulin secretion could play an important role in the uremic glucose intolerance *(Briggs et al., 1967)*.

Glycohemoglobin is formed progressively and irreversibly in the erythrocyte during its 120 day life. The red cell glycohemoglobin concentration is dependent on the average blood glucose concentration over a period of weeks and is stable for the life of the cell. Therefore, measurement of glycohemoglobin (HbA_1) as percent of total hemoglobin provides a valuable method for assessing the long term control of diabetics, since HbA_1 levels approach normal values as diabetics respond to treatment *(Trivelli et al., 1971, and Gabbay et al., 1977)*.

There are a number of pathophysiologic situations such as chronic renal failure, hemolytic anaemia, and pregnancy that shorten red cell survival and the concentration of HbA_1 *(Rosenthal et al., 1981)*.

Fructosamine test is a new colorimetric assay designed to measure serum glycosylated protein concentration *(Johnson et al., 1982)*.

Measurement of plasma fructosamine appears to be a reliable indicator of blood glucose conentrations over the previous 7-21 days *(Baker et al., 1984, and Lim and Staley, 1985).*

All the previous studies determined the validity of both HbA_1 and fructosamine as index for glycemic control in diabetics only, but our work aimed to compare between the two parameters in diabetic patients with chronic renal failure and to determine which of them is a more reliable index for glycemic control in these patients.

In our work we found that the level of HbA_1 was 7.7 ± 0.78% for the controls (Table 8) and this agreed with *Trivelli et al., (1971)* who reported that the mean HbA_1 in normal non diabetic subjects was 6.5 ± 1.5% of total Hb.

As regarding for the diabetic groups with or without uremia the level of HbA_1 was significantly higher when compared to the control group (Table 8). This was in consistent with *Huisman and Dozy, (1962), Rahbar et al., (1969), Trivelli et al., (1971) and Gabbay et al., (1977),* who demonstrated that HbA_1 fraction was increased in diabetic patients and its levels were generally two to three times than normal. *Sharma et al., (1989)* reported that HbA_{1c} level in diabetic subjects was found to be markedly elevated and was found to be highly significant. *Also Bruns et al., (1984)* denoted that HbA_1 was significantly higher in diabetic uremic than non diabetic uremic patients.

Hb level was significantly lower in uremic groups with or without diabetes when compared to the control group whereas no significant difference could be detected between diabetic non uremic group and the controls (Table 3). Also there was a positive significant correlation between HbA_1 and Hb levels among all groups (Table 10).

Red cell survival was variable but generally shortened by one-half by time in advanced renal failure *(Shaw, 1967).*

Eschabach et al., (1977) reported that shortening of red cell life span played an important role in the anaemia of renal disease.

Giovannetti et al., (1974) demonstrated that guandine derivatives were elevated in the serum of uremic patients and may be the cause of hemolysis seen in chronic renal failure (C.R.F)

Higgins et al., (1982) noted that the non enzymatic glycosylation of hemoglobin was determined by three major variables: mean plasma glucose concentration, red cell life span and red cell glucose permeability.

As regarding for uremic non diabetic group the HbA_1 level was significantly lower compared to the control group (Table 8). This result agreed with the result of *Dandona et al., (1979),* who reported that C.R.F. was associated with markedly lower HbA_1% without concomitant change in blood glucose concentrations. The diminished HbA_1% was consistent with shortened R.B.C. survival in these patients. Also *Freedman et al., (1982)* demonstrated that

HbA_1 in uremic patients was significantly lower than that in control subjects and also the concentration of HbA_1 in patients on hemodialysis, was not significantly different either form that in uremic patients prior to dialysis or that in patients on peritoneal dialysis suggesting that the main cause of shortened R.B.C. survival in these patients was uremia and not hemodialysis.

On the other hand, *Cohen and Lee, (1981), Lantz et al., (1981), and Oimomi et al, (1981),* reported that the concentration of HbA_1 was increased in uremic patients. However, *Fluckiger et al., (1981)* demonstrated that the increased levels of fast Hb in uremic patients might not due to glycosylation at all and were apparently related to interference in the assays from other altered hemoglobins in uremic blood, in particular, carbamylated hemoglobin which was a consequence of the spontaneous dissociation of urea into ammonia and cyanate. Carbamylated Hb had much the same net electrical charge as some HbA_1.

In correlation study we found positive significant correlation between HbA_1 and both fasting and post prandial blood sugar among the diabetic groups with or without uremia (Table 10). This agreed with *Rahbar et al., (1969), Trivelli et al., (1971), Gabbay et al., (1977), and Gonen et al., (1977),* who reported that HbA_{1c} or HbA_1 levels were elevated in diabetic patients and that the degree of elevation correlated with the degree of glucose tolerance, the amount of urinary glucose excretion and blood

glucose concentration. Also *Goldstein et al., (1982), Nathan et al., (1984), and Mc Cance et al., (1988),* demonstrated that there was a remarkebly good correlation between HbA_1 and blood glucose concentration. *Bruns et al., (1984)* showed that in diabetic uremic patients there was a significant correlation between HbA_1 and fasting plasma glucose.

There was positive insignificant correlation between HbA_1 and both fasting and post prandial blood sugar in the uremic non diabetic and the control groups (Table 10). This was in consistent with *John and Richardson (1986)* who determined that the non diabetic group showed only poor correlation between HbA_1 and fasting and 2 hours blood glucose concentration.

The cause of positive insignificant correlation in uremic group could possibly be explained by the shortening of life span of R.B.C. which was reflected by decrease of level of HbA_1 and in the control group the cause of this positive insignificant correlation between HbA_1 and both fasting and post prandial blood sugar as blood glucose is not so high when compared to D.M.

Also, we found a negative insignificant correlation between HbA_1 and creatinine levels among all groups (Table 11).While *Bruns et al., (1984)* reported that HbA_1 was negatively correlated with urea. Also *Rezk , (1981)* reported that no significant correlation between the levels of HbA_{1c} and the levels of blood

urea or plasma creatinine in control subjects and in patients with chronic renal failure.

In our study for fructosamine we found that the mean value of serum fructosamine was 1.43 ± 0.145 mmol/l for the controls (Table 9) and this result agreed with the result of *Baker et al., (1985)* who reported that the normal range of fructosamine concentration in healthy was 1.28 - 1.76 mmol/l.

Regarding the diabetic groups with or without uremia the mean value of serum fructosamine was significantly higher as compared to the controls (Table 9). This was demonstrated by *Negoro et al., (1988)* who reported that the levels of fructosamine were significantly higher in diabetic subjects compared with those in non diabetic and were also significantly different in diabetic subjects with well controlled and poorly controlled.

Regarding the uremic non diabetic group we found that the mean value of serum fructosamine was not significantly different from the corresponding value in the control group (Table 9). This was demonstrated by *Baker et al., (1983)* who reported that no significant difference in the level of serum fructosamine in the uremic patients when compared with that of the controls.

Also, we found both fasting and post prandial blood sugar insignificantly changed in uremic non diabetic group compared to the control group (Table 4,5), total protein (T.P) levels insignificantly changed among all groups compared to the

controls (Table 6) and that there was a positive significant correlation among all groups between T.P and serum fructosamine levels (Table 12). *Baker et al., (1983)* demonstrated that there was no linear relation between fructosamine and serum protein concentration in normal subjects and those with uremia. Serum protein concentrations became important only when there was coexisting severe hypoproteinemia. Also *Koskinen et al., (1987)* reported that physiological states altering the rate of synthesis or elimination of serum proteins should be considered in the interpretation of fructosamine levels.

The insignificant change of fructosamine level in uremic non diabetic group could be explained by the finding that both parameters which affect serum fructosamine level (blood sugar and T.P) showed no significant alterations in these patients.

In correlation study we found a significant positive correlation between the fructosamine and both fasting and post prandial blood sugar among all groups (Table 12). This was demonstrated by *Johnson et al ., (1982), Baker et al., (1983), , Roberts et al., (1983) and Baker et al.,(1985),* who stated that there was positive correlation between serum fructosamine levels and fasting plasma glucose levels. *Jermendy et al., (1988)* reported that there was a significant correlations between serum fructosamine values and the mean blood glucose as well as 24

hours urinary glucose excretion values measured 10-14 days previously in diabetics.

Also, there was a negative insignificant correlation between the fructosamine and the creatinine in diseased groups (Table 13), and this was in agreement with *Baker et al., (1983) and Dominiczak et al., (1989)* who reported that in patients with abnormal renal function there was no correlation between serum fructosamine and either urea or creatinine

There was a positive correlation between HbA_1 and serum fructosamine among all the studied groups. However, this relationship was significant only among the diabetic as well as the diabetic uremic on conservative treatment (Table 14). This was in consistent with *Baker et al., (1983) and Daubresse et al., (1987)* who determined that an excellent correlation between fructosamine and HbA_{1c} and between each of them and various parameters of metabolic control. Also *Allgrove and Cockrill, (1988)* reported that in diabetic children there was a highly significant correlation between HbA_{1c} and fructosamine, but this was lost when only concentrations within the established normal ranges were considered.

On conclusion, these results implies that fructosamine is a test of choice for judging glycemic control in uremia for the following reasons:

1) HbA_1 was affected by uremia due to shortening red cell life span which was reflected by decrease level of HbA_1 while fructosamine was not affected as T.P levels among all the studied groups insignificantly changed.

2) The automaticity, reporducibility and lower cost for fructosamine assay argue strongly in favour of this assay in comparison to those of other glycosylated proteins *(Baker et al., 1983, Negoro et al., 1988).*