RESULTS

CLINICAL DATA OF DIABETIC PATIENTS

No	Age	Sex	Weight (kg) percentile %	Height (cm) percentile %	Frequency of diabetes	Duration of diabetes	Control
1	10	F	25%	10-25	1	5	Fair
2	15	F	50-70%	<3	-	7	Fair
3	4	M	25	3-10	-	1	Good
4	8	F	50-70	10-25	-	3	Good
5	6	M	50	<3	2	3	Fair
6	10	M	3-10	< 3	-	3	Good
7	14	M	3-10	<3	3	8	Poor
8	11	F	3	3-10	2	7	Poor
9	10	F	75	25-30	1	4	Good
10	5	M	75-90	3-10	1	1	Good
11	10	M	90-97	3-10	3	5	Poor
12	13	F	3-10	3	1	7	Poor
13	5	F	75	25-50	-	3	Fair
14	15	M	< 3	< 3	-	3	Good
15	10	M	10	10-25	1	3	Good
16	12	M	25	3-10	3	6	Poor
17	8	F	25	10	1	3	Good
18	11	F	25-50	10-25	1	5	Poor
19	9	F	3	< 3	3	2	Good
20	4	M	25	10-25	2	2	Good
21	5	F	3-10	< 3	3	2	Poor
22	8	F	<10	3	1	3	Poor
23	7	m	3-10	< 3	-	2	Good
24	14	F	< 10	25	-	3	Good
25	13	F	10-25	25	-	3	Good
26	6	F	50	50-75	3	1	Good
27	14	M	25-50	25	1	4	Good
28	12	M	10-25	3	1	3	Good
29	12	M	20-25	3-10	-	2	Good
30	13	M	25	3-10	-	3	Good
31	15	M	3-10	3-10	-	3	Good

No	Age	Sex	Weight (kg) percentile %	Height (cm) percentile %	Frequency of diabetes	Duration of diabetes	Control
32	15	M	3	< 3	-	4	Fair
33	13	M	10-25	< 3	1	4	Good
34	15	M	3-10	< 3	-	2	Good
35	10	F	25-50	10-25	1	4	Good
36	7	M	10	10-25	-	4	Fair
37	12	F	10-25	10-25	-	2	Good
38	6	F	10-25	30-10	-	1	Good
39	6	M	< 3	< 3	-	6	Fair
40	13	M	3-10	< 3	-	1	Good
41	7	M	< 3	< 3	-	2	Good
42	9	M	25-50	> 97	1	2	Good
43	11	M	75-90	90-97	-	1	Good
44	14	M	10-25	3-10	-	1	Good
45	13	F	25	< 3	1	1	Good
46	11	F	10	< 3	1	2	Good
47	11	M	3	3-10	1	3	Fair
48	7	F	10-25	25-50	2	4	Poor
49	6	M	75-90	0-25	3	3	Poor
50	3	F	3-10	< 3	3	1	Poor

Table I

CLINICAL DATA OF CONTROLS

No	Age	Sex	Weight (percentile %)	Height (percentile %)
1	7	F	10-25	25-50
2	5	M	50-75	50-75
3	13	M	10-25	10-25
4	9	F	25	25
5	15	F	25	25
6	11	M	10-25	25
7	4	M	25	25
8	8	M	50	25-50
9	11	F	25	10-25
10	10	F	50-75	25-50
11	12	M	50	25-50
12	12	M	10-25	10-25
13	7	F	25-50	25
14	13	F	10-25	10-25
15	9	F	25-50	50
16	9	F	75	90-97
17	8	F	75	50
18	4	F	90	75
19	4	F	25-50	50-75
20	6	M	25-50	50-75
21	14	F	25-50	50
22	5	F	25-50	75
23	10	F	50-75	25-50
24	10	F	< 3	25-50
25	4	M	< 3	75

Table II

Laboratory data of diabetic patinets:

No	C. peptide ng/ml	Insulin µ. IU/ml	Glusose mg/dl	HBA_1C %	Amylase colour unit/100ml	Lipase IU/L
1	0.043	390.73	188 µg/dl	9.302	51.111	99.615
2	0.082	357.089	200 µg/dl	9.800	45.222	68.417
3	0.025	138.280	110 µg/dl	7.346	52.222	93.296
4	0.016	142.900	124 µg/d	18.171	121.111	74.736
5	0.043	100.082	263	9.856	55.555	80.587
6	0.086	110.739	94	7.510	56.666	68.417
7	0.094	390.645	300	10.039	73.333	93.581
8	0.026	147.66	230	10.184	100.000	75.975
9	0.037	109.23	190	8.459	87.778	93.851
10	0.021	82.043	400	8.748	105.556	80.444
11	0.004	330.266	247	10.908	60.000	67.037
12	0.002	240.562	290	11.523	121.111	98.320
13	0.080	80.678	280	9.145	45.556	102.321
14	0.004	89.432	255	8.339	67.778	97.872
15	0.013	65.382	240	8.953	64.444	104.565
16	0.024	140.520	368	11.198	92.222	106.770
17	0.086	209.75	249	0.791	119.693	104.266
18	0.064	245.32	340	13.188	110.486	88.266
19	0.075	144.95	282	8.897	85.933	49.123
20	0.006	308.01	250	8.275	128.900	76.195
21	0.009	241.70	397	12.832	110.486	92336
22	0.018	160.42	290	10.861	54.878	91.786
23	0.052	259.66	205	8.734	64.634	81.587
24	0.043	142.90	180	8.991	92.683	86.786
25	0.062	340.326	138	8.334	108.537	71.389
26	0.004	109.23	290	11.156	43.902	56.091
27	0.028	87.387	250	8.696	69.512	66.290
28	0.036	173.19	186	7.905	135.366	81.587
29	0.045	213.39	192	7.333	128.049	56.091
30	0.018	164.210	93.00	7.619	109.756	81.587
31	0.017	162.200	144.00	7.066	53.902	112183

No	C. peptide ng/ml	Insulin µ. IU/ml	Glusose mg/dl	HBA_1C %	Amylase colour unit/100ml	Lipase IU/L
32	0.025	240.500	280.00	7.943	54.659	61.190
33	0.036	162.200	240.00	8.763	132.927	91.786
34	0.024	180.302	118	7.845	72.245	27.342
35	0.018	200.811	220	8.407	85.321	41.013
36	0.064	390.400	181	7.469	87.11	72.261
37	0.084	138.362	117	6.786	68.288	60.543
38	0.053	147.510	130	7.342	78.802	81.718
39	0.064	109.710	189	7.013	70.037	77.320
40	0.082	209.637	199	7.261	50.872	106.250
41	0.074	245.851	210	8.543	80.321	80.226
42	0.095	30.975	150	7.246	82.038	56.290
43	0.081	259.441	171	7.280	74.417	71.091
44	0.006	142.908	220	8.851	66.851	56.389
45	0.002	340.145	235	8.290	95.637	80.183
46	0.007	109.339	187	8.090	105.516	61.199
47	0.078	173.188	180	9.389	92.183	28.310
48	0.064	213.791	350	11.183	84.543	106.260
49	0.032	209.241	290	12.199	80.246	36.440
50	0.046	308.897	244	12.310	93.290	28.230

Table III

Laboratory data of controls:

No	C. peptide ng/ml	Insulin µ. IU/ml	Glusose mg/dl	HBA_1C %	Amylase colour unit/100ml	Lipase IU/L
1	8.000	16.577	74	6.260	78.091	36.440
2	12.000	10.851	62	7.310	75.451	28.490
3	6.000	6.878	80	7.756	50.555	99.119
4	14.000	30.950	78	7.241	84.111	124.245
5	2.000	16.470	95	6.845	48.778	27.342
6	7.000	18.302	60	6.407	67.209	41.013
7	8.000	20.811	67	7.469	80.217	72.261
8	0.210	12.364	77	6.454	84.210	60.543
9	0.310	6.171	71	6.221	86.721	11.718
10	0.420	28.856	108	7.040	72.629	10.710
11	0.810	22.510	98	7.902	52.111	104.245
12	12.280	28.039	84	6.659	46.778	102.321
13	5.450	30.184	76	6.927	87.782	88.268
14	11.110	18.549	65	6.713	105.556	102.111
15	17.850	11.748	59	7.209	45.440	108.281
16	9.180	28.908	111	6.217	92.222	96.802
17	12.171	24.523	118	6.210	136.486	99.037
18	10.181	32.145	88	7.721	85.933	102.872
19	0.910	30.339	74	6.543	128.486	106.321
20	0.850	10.198	99	7.261	92.683	22.038
21	14.192	8.791	114	7.342	118.534	8.444
22	13.155	22.897	130	7.261	69.602	16.417
23	0.846	6.275	102	6.210	128.366	90.851
24	9.721	23.188	66	6.721	90.180	84.633
25	6.210	22.188	54	7.245	98.118	66.516

Table IV

NORMAL VALUES

Insulin	0-30 μ IU/ml	
C.peptide	0.1-20 ng/ml	
Glucose	80-120 mg/dl	
Amylase	45-200 color unit /100ml.	
	75-340 international units / 100 ml.	
Lipase	0-110 I.U. / L	(at 37 °C)
	0-105 cherry crandall unit.	
Glycohemoglobin	7.5-8.9%	Good control
	9-10.0%	Fair control
	↑ 10.0%	Poor control.

The results of this work are presented in tables.

Table (1) Comparison between cases and controls as regards mean fasting serum C.peptied (value ng/ml):

	Cases	Controls
No	50	25
Mean	0.04	7.31
± S.D.	0.02	5.35

t 6.79

p < 0.001 Highly significants

Table (2) Comparison between cases and controls as regards serum insulin (μ-IU/ml):

	Cases	Controls
No	50	25
Mean	199.40	19.54
± S.D.	90.48	8.56

t 13.93

p < 0.001 Highly significant

Table (3) Comparison between cases and controls as regards mean blood glucse (mg/dl):

	Cases	Controls
No	50	25
Mean	221.75	84.40
± S.D.	74.87	20.86

t 12.07

p < 0.001 Highly significant

Table (4) Comparison between cases and controls as regards glycocylated hemoglobin (%):

	Cases	Controls
No	50	25
Mean	9.007	6.925
± S.D.	1.628	0.528

t 8.22

p < 0.001 Highly significant

Table (5) Comparison between cases and controls as regards serum amylase enzyme (column unit /100ml):

	Cases	Controls
No	50	25
Mean	83.433	84.262
± S.D.	25.638	25.613

t 0.13

p > 0.05 Not significant

Table (6) Comparison between cases and controls as regards seum lipase enzyme (IU/L):

	Cases	Controls
No	50	25
Mean	76.433	68.441
± S.D.	21.777	38.121

t 0.97

p > 0.05 Not significant

Table (7): Comparison between metabolic control of D.M and serum C.Peptide among cases (mg/ml):

	Poor control	Fair control	Good control
No	12	5	33
Mean	0.032	0.65	0.041
± S.D.	0.029	0.020	0.029

ANOVA = 2.36

P> 0.05 Not significant

Only significant difference of Poor V.S Good P < 0.05

Table (8): Comparison between metabolic control of D.M and serum insulin among cases (μ IU/ml):

	Poor control	Fair control	Good control
No	12	5	33
Mean	228.187	220.353	185.759
± S.D.	83.902	144.848	83.212

ANOVA = 1.12

P> 0.05 Not significant

No significant difference between any two groups.

Table (9): Comparison between metabolic control of D.M and blood glucse among cases:

	Poor control	Fair control	Good control
No	12	5	33
Mean	303.000	202.200	195.121
± S.D.	51.854	34.960	65.197

ANOVR = 14.43

P< 0.001 highly significant

Poor VS good is highly significant.

Poor V.S fair is highly significant. P < 0.001

Fair V.S good is highly significant

Table (10): Comparison between metabolic control of D.M and glycoselated hemoglobin among cases:

	Poor control	Fair control	Good control
No	12	5	33
Mean	11.465	9.500	8.039
± S.D.	0.985	0.316	0.666

ANOVA = 97.16

P< 0.001 highly significant

Poor V.S fair.

Poor V.S good Highly significant

Fair V.S good P< 0.01.

Table (11): Comparison between metabolic control of D.M and serum amylase enzyme amnog cases:

	Poor control	**Fair control**	**Good control**
No	12	5	33
Mean	85.347	57.925	86.592
± S.D.	23.984	19.621	25.442

ANOVA = 2.98 P > 0.05 Insignificant

Poor metabolic control = 10.0%

Fair metabolic control = 9-10.0

Good metabolic control = 7.5-8.9.

Table (12): Comparison between metabolic control of D.M and serum lipase enzyme among cases:

	Poor control	**Fair control**	**Good control**
No	12	5	33
Mean	78.196	75.850	75.880
± S.D.	26.413	30.013	19.293

ANOVA =0.05

P> 0.05 Not significant

Table (13): Correlation coefficient between serum C- peptide, and serum insulin, HBA1C, serum glucose, serum lipase and amylase among cases:

		R test	P	
C-peptide	Insulin	0.24	< 0.10	Border low significant
	HbA1C	-0.2	> 0.05	Not significant
	Glucose	-0.24	< 0.10	Border low significant
	Amylase	-0.16	> 0.05	Not significant
	Lipase	-0.03	> 0.05	Not significant

Table (14): Correlation coefficient between serum C- peptide, and serum insulin, HBA1C, serum glucose, serum lipase and amylase among controls:

		R test	P	
C-peptide	Insulin	0.16	> 0.05	Not significant
	HbA1C	-0.04	> 0.05	Not significant
	Glucose	0.17	> 0.05	Not significant
	Amylase	-0.08	> 0.05	Not significant
	Lipase	0.24	> 0.05	Not significant

Table (15): Correlation coefficient between duration and serum C. peptide, insulin, glucose, glycosylated hemoglobin, lipase and amylase:

	Results	r	P
Duration	C.peptide	0.076	< 0.05 significant
	Insulin	0.231	< 0.05 significant
	Glucose	0.248	> 0.05 Not significant
	Glycosylated Hb	0.363	< 0.05 significant
	Amylase	0.040	> 0.05 Not significant
	Lipase	0.229	> 0.01 Not significant

There is a positive correlation between duration and glycosylated hemoglobin

$r = 0.36$ $P < 0.01$ significant.

Table (16) Comparison between males and females as regards serum amylase among controls:

	Males	Females
No	10	15
Mean	79.557	87.398
±S.D.	21.317	28.392

$t = 0.79$

$P > 0.05$ · Not significant

Table (17) Comparison between males and females as regards serum lipase enzyme among controls:

	Males	**Females**
No of cases	10	15
Mean	57.869	34.336
±S.D.	75.489	40.002

t = 1.18

P > 0.05 Not significant

Table (18) Comparison between males and females as regards serum insulin among controls:

	Males	**Females**
No of cases	10	15
Mean	16.449	21.608
±S.D.	8.703	8.095

t = 1.49

P > 0.05 Not significant

Table (19) Comparison between males and females as regards blood glucose among controls:

	Males	Females
No of cases	10	15
Mean	74.500	91.000
±S.D.	16.814	21.163

t = 2.06

P < 0.05 significant

Table (20) Comparison between males and females as regards glycosylated hemoglobin among controls:

	Males	Females
No of cases	10	15
Mean	6.779	6.890
±S.D.	0.521	0.574

t = 0.4

P > 0.05 Not significant

Table (21) Comparison between males and females as regards serum C.peptid among controls:

	Males	**Females**
No of cases	10	15
Mean	7.165	7.413
±S.D.	4.717	5.906

t = 0.11

P > 0.05 Not significant

Table (22) Comparison between males and females as regards blood glucose among cases:

	Males	**Females**
No of cases	28	22
Mean	215.535	225.590
± S.D	74.566	76.268

t = 0.65

P > 0.05 Not significant

Table (27) Comparison between males and females as regards serum insulin among cases:

	Males	**Females**
No of cases	28	22
Mean	194.563	205.559
± S.D.	90.899	91.713

t = 0.42

P > 0.05 Not significant

Table (28): Comparsion between height and metabolic control detected by glycosylated hemoglobin (HbA_1C) among cases:

Metabolic control	**Height (percentile)**						**Total**
	< 25		**25-75**		**> 75**		
	No	**%**	**No**	**%**	**No**	**%**	
Poor control	10	83.3	2	16.7	-	-	12
Fair control	3	60.0	2	40.0	-	-	5
Good control	27	81.8	4	12.1	2	6.1	33

X^2 Chi -square = 3.42

P > 0.05 Not significant

Table (29): Comparison between weight and metabolic control detected by glycoselated hemoglobin among cases:

Metabolic control	Wight (percentile)						Total
	< 25		25-75		> 75		
	No	%	No	%	No	%	
Poor control	6	50.0	3	25.0	3	25.0	12
Fair control	3	60.0	2	40.0	-	-	5
Good control	18	54.5	12	36.4	3	9.1	33

X^2 (Chi -square) = 2.98

P > 0.05 Not significant

Comparison between cases and controls as regard C-peptide (value ng/ml)

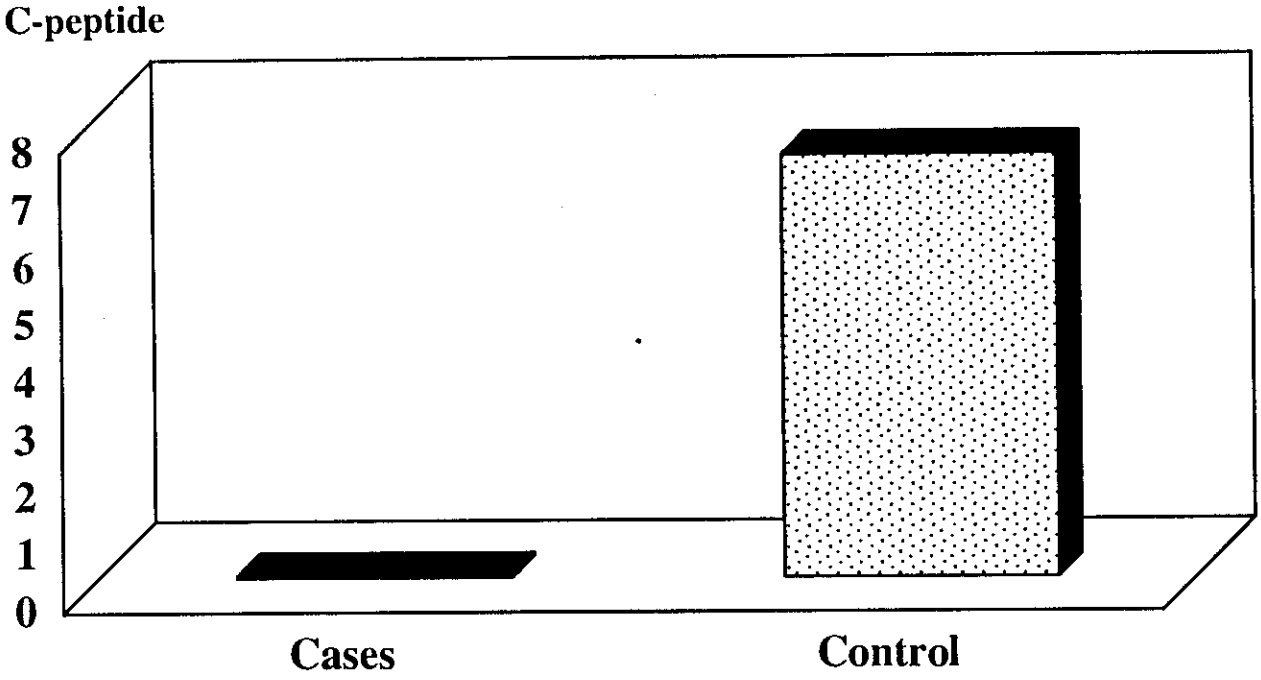

Fig (9)

Comparison between cases and controls as regard serum insulin (μ–IU/ml)

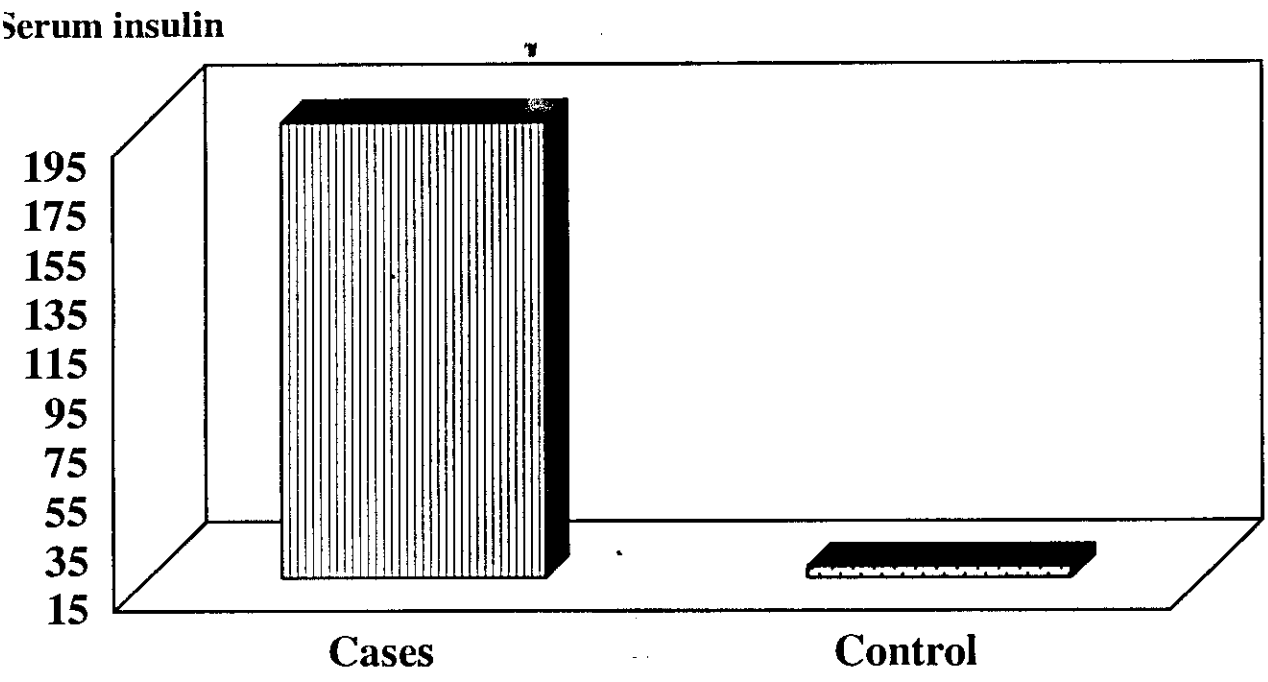

Fig (10)

Comparison between cases and controls as regards blood glucose (mg/dl)

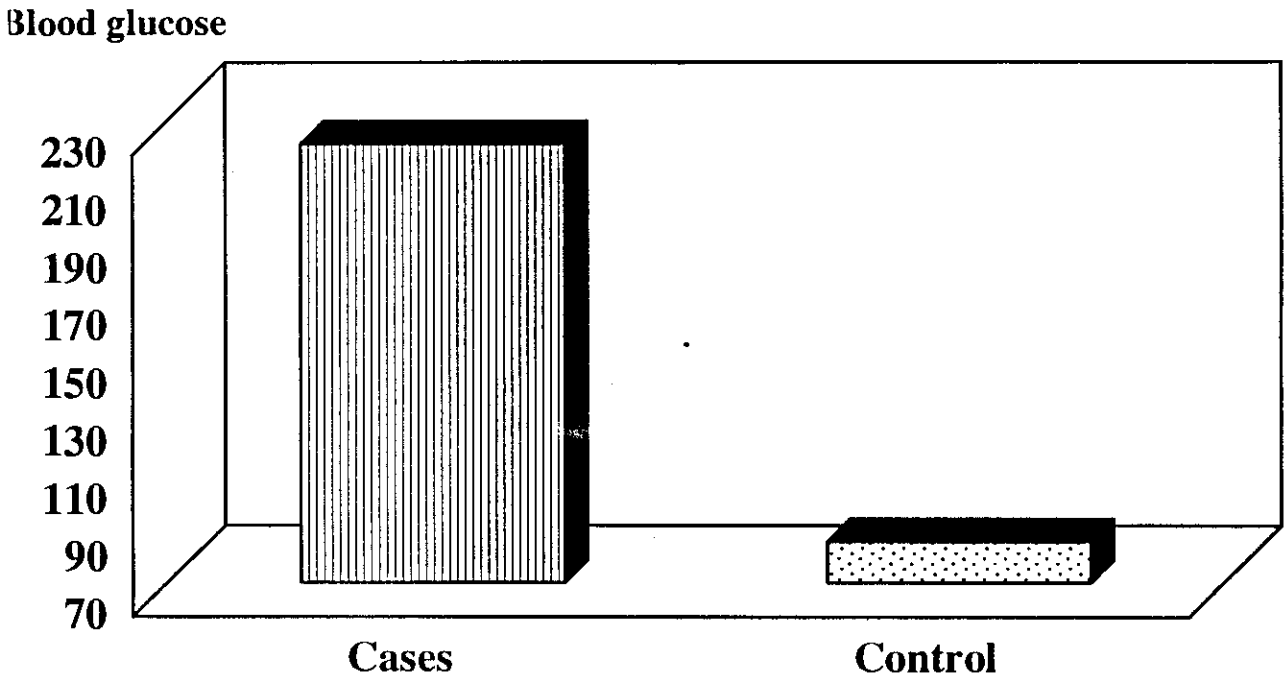

Fig (11)

Comparison between cases and controls as regards glycosylated hemoglobin (%)

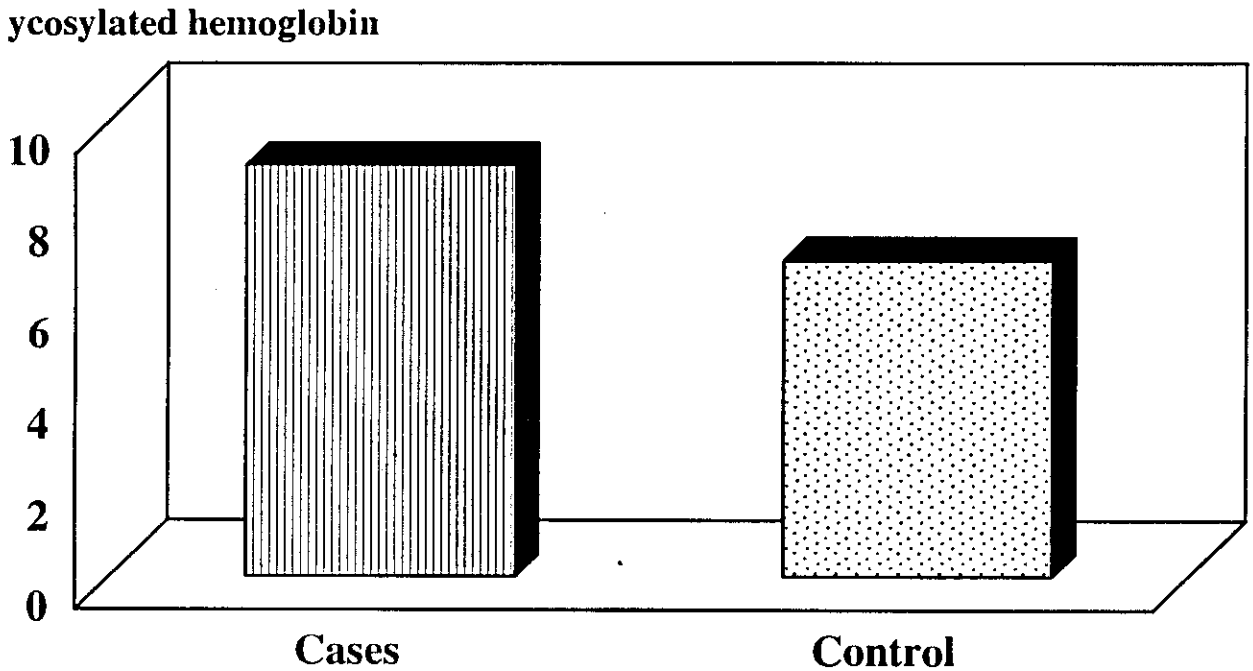

Fig (12)

Comparison between cases and controls as regards serum amylase enzyme (colour unit/ 100ml)

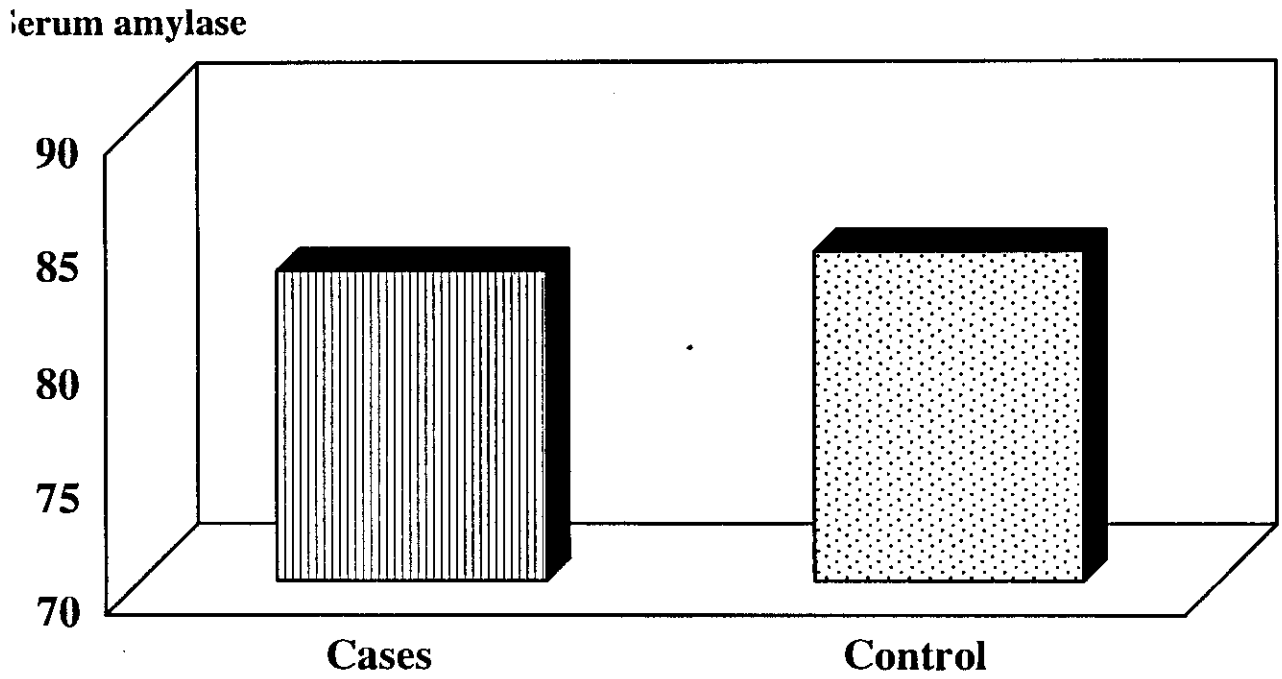

Fig (13)

Comparison between cases and controls as regards serum lipase enzyme (IU/L)

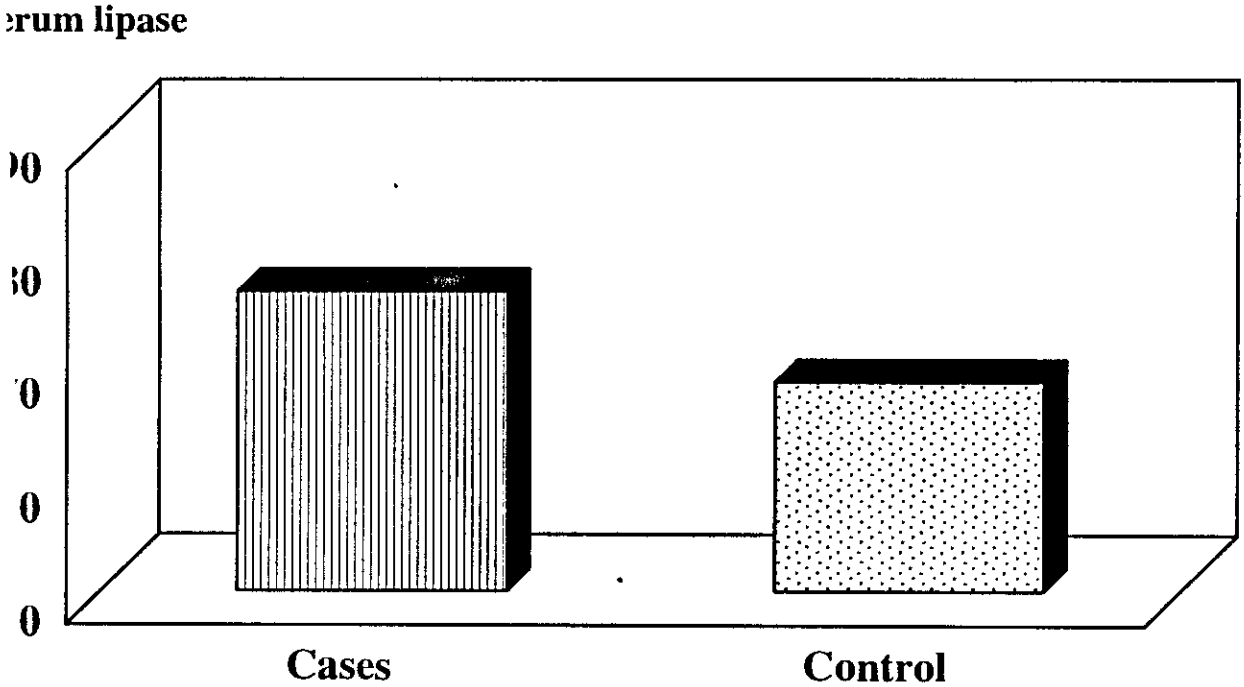

Fig (14)

Comparison between males and females as regardamylase enzyme among cases

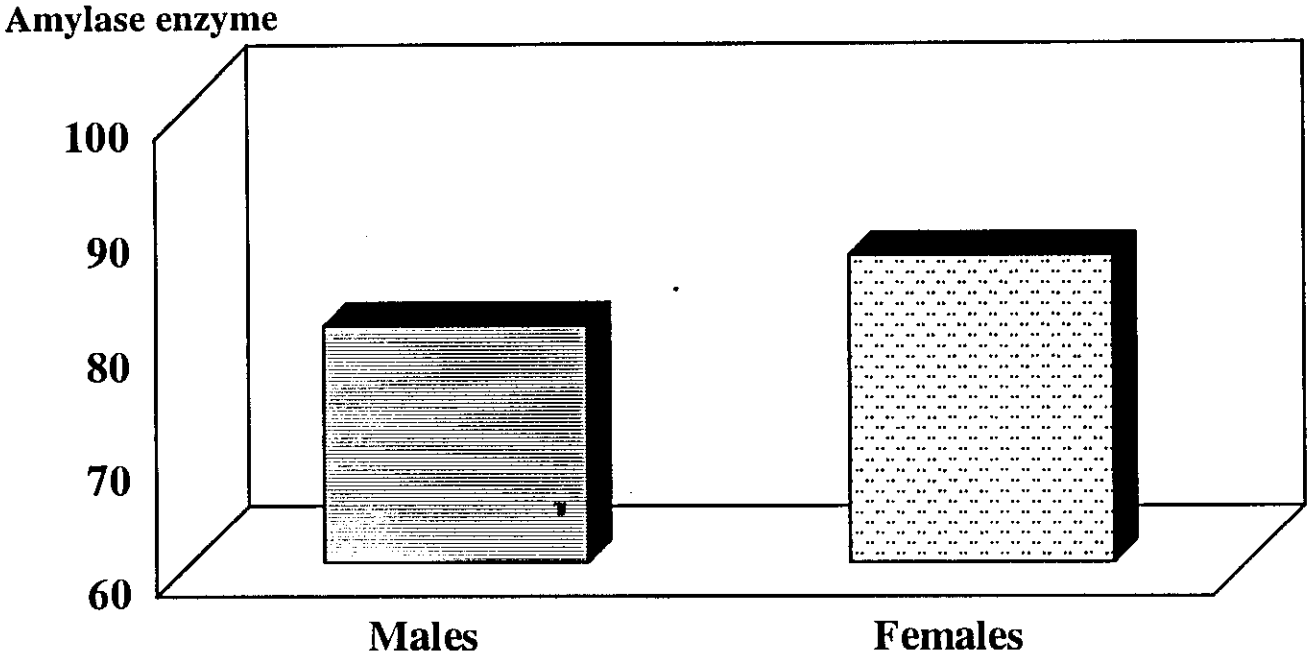

Fig (15)

Comparison between males and females as regard Lipase enzyme among cases

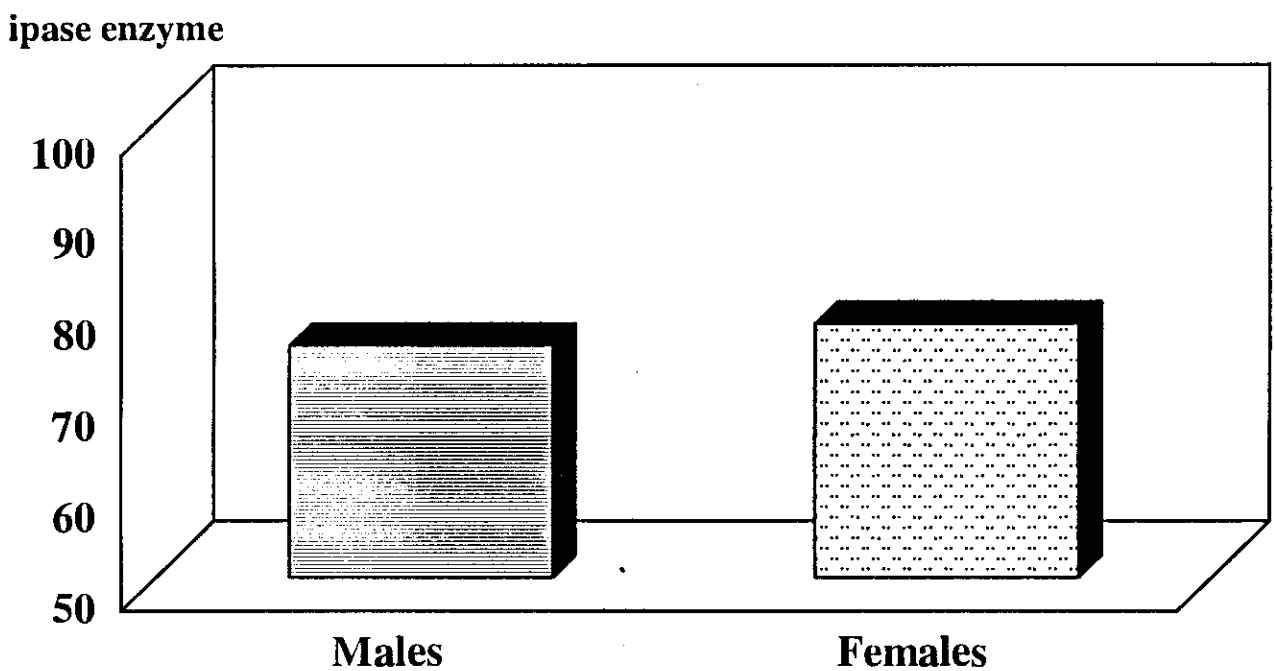

Fig (16)

Comparison between males and females as regard C-peptide among cases

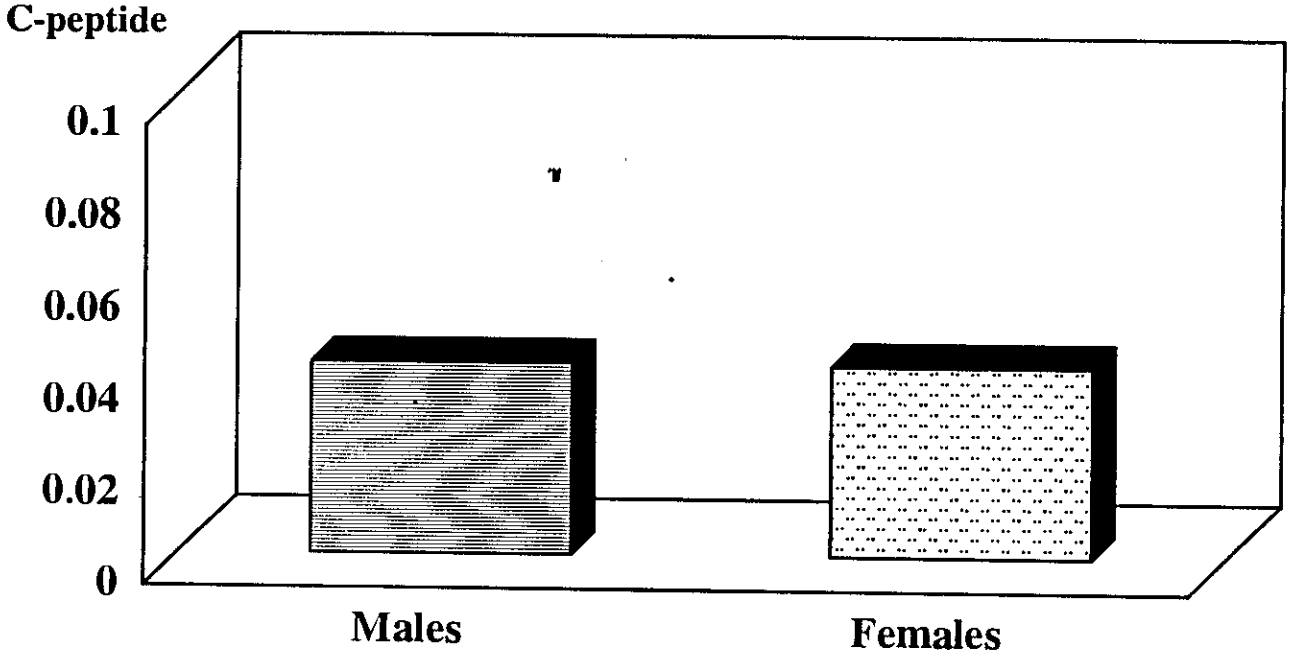

Fig (17)

Comparison between males and females as regard insulin among cases

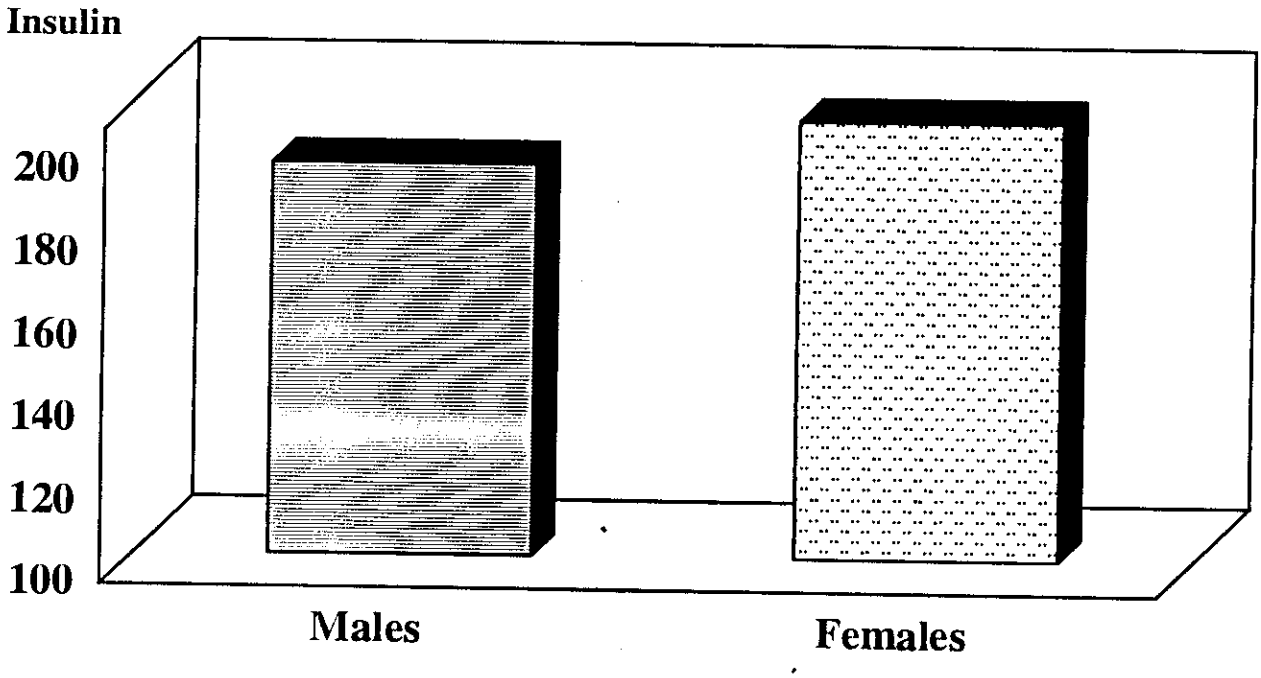

Fig (18)

Comparison between males and females as regard blood glucose among cases

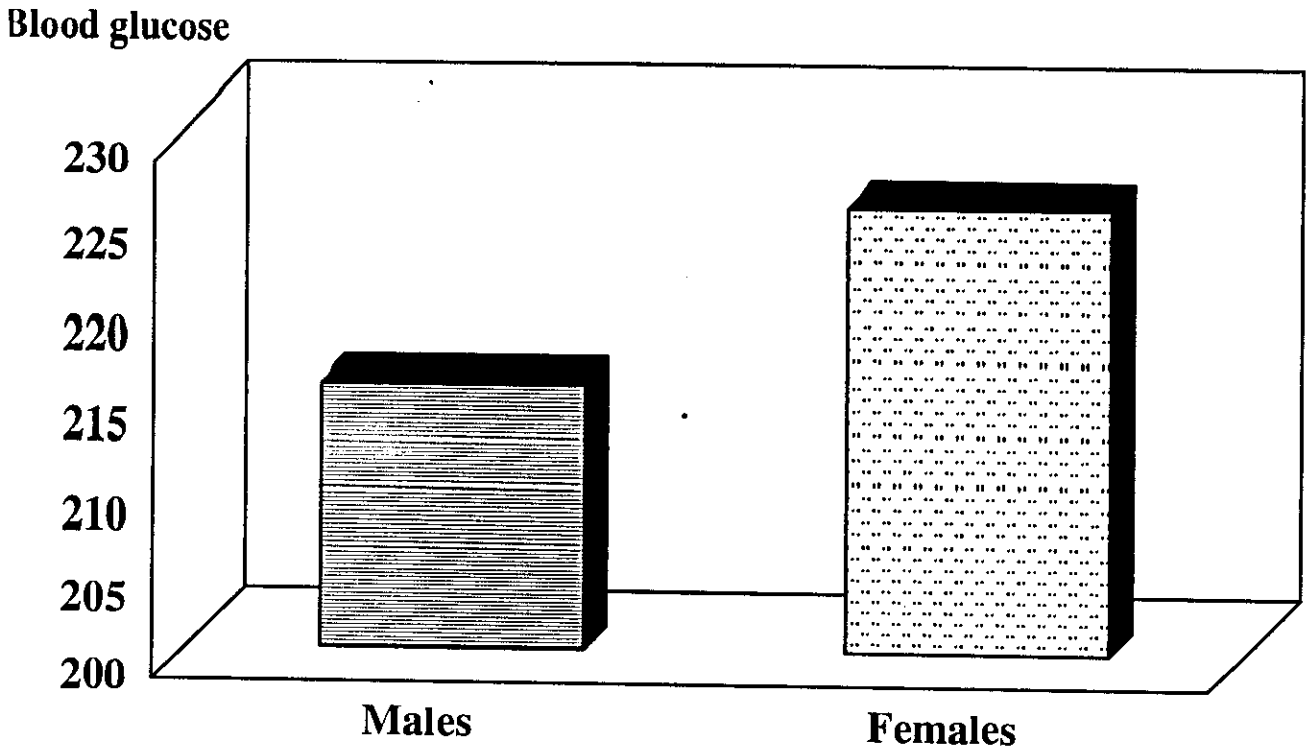

Fig (19)

Comparison between males and females as regard HBA1C among cases

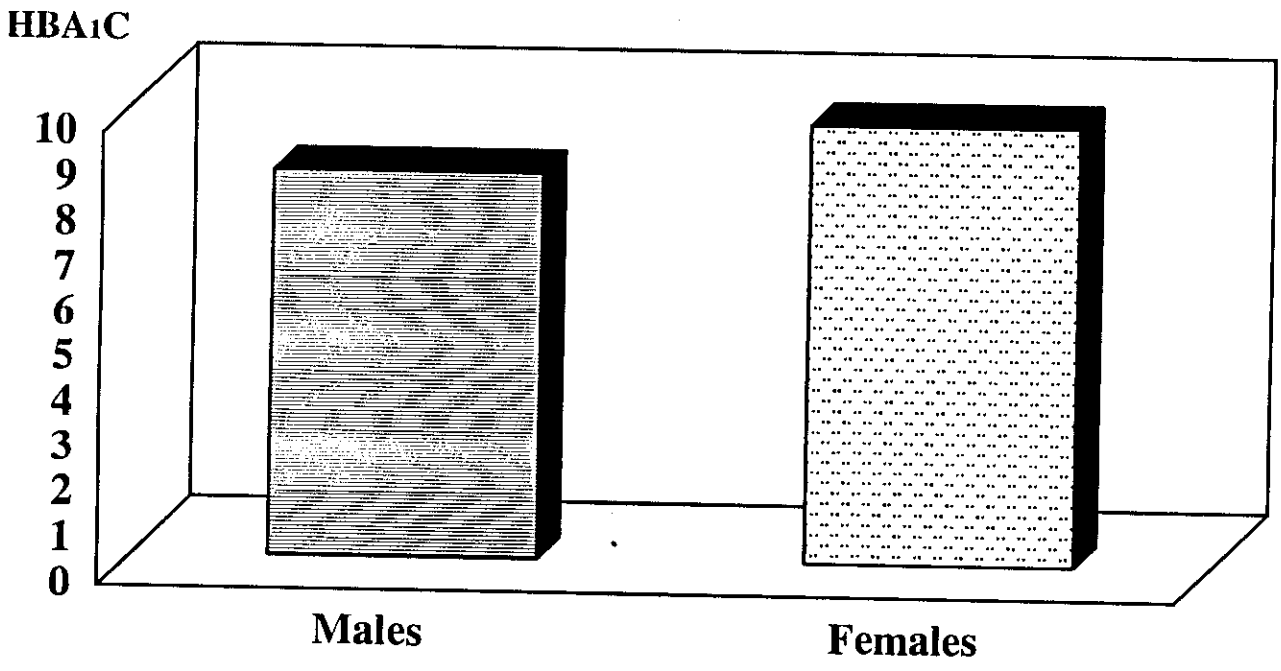

Fig (20)

DISCUSSION

DISCUSSION

Diabetes mellitus is a chronic metabolic disorder with many long term complications. Both duration of diabetes and its metabolic control have been identified as risk factors most strongly associated with the development of diabetic micro vascular complications (Bodansky et al., 1982). Long term glycemic control still remains as the cornerstone for the prevention of these complications, poor control of blood glucose is thought to be an important factor in the development of the diabetic complications (Mc Cance et al., 1989).

Several reports now support at the view that most patients with IDDM have residual B-cell function the onset of the disease and that the remaining capacity for insulin secretion might be clinically important for, at least, the short term prognosis (Ivarson et al., 1987). Other investigators suggested that a high residual B-cell function is correlated with a better glycemic control and lower requirements for exogenous insulin (Marner et al., 1985).

In the present study (Table 4), the mean glycosylated hemoglobin A1 (HbA1) of diabetic patients was significantly higher compared to the mean value of the control group.

Glycosylated hemoglobin A1 seems to reflect integrated blood glucose control over long periods of time (Anderson et al., 1989). That is why it was of interest to investigate the relationship between HbA1 and the mean blood glucose of the preceding three months calculated from routine blood glucose monitoring of patients to find which period of routine blood glucose monitoring is more closely correlated to HbA1 values.

In the present study (Table 9) Glycosylated HbA1 was correlated to mean blood glucose values of either first, second or third months preceding the analysis.A similar study was done by kennedy and his colleague (1984). Where they assessed HbA1 and post prandial blood glucose at sequential clinic visits over a period of 9-12 months in IDDM patients; they reported that the two parameters were only concordant in approximately 40% of observations.

The same as our finding Ghaly et al., (1990) and other workers (Svendsen et al., 1982; Nathan et al., 1984) have reported a correlation between the concentration of glycosylated hemoglobin and mean values of blood glucose. Instability in blood glucose control may reduce this correlation (Boden et al., 1980; Pecoraro et al., 1982).

Gabbay et al., (1977) reported that HbA1, is significantly correlated with the amount of glucose excreted in 24 hr urine obtained at 0-14 days, 1 months, 2 months and 3 months prior to blood sampling with the highest correlation with the glucose excretion in the second month collection. A contrast study was done by Schleicher et al., (1984) who found discrepancies between HbA1 and blood glucose level in 10 IDDM patients over a year period.

It is evident from the present work that regular follow up of diabetic patients improve their level of glycemic control, it assess compliance and modify insulin dose.

Travis et al., 1987 categorized their patients according to glycosylated HBA1 values into patients with very good glycemic control (HbA1 6 -8%), fair control (8-10%), bad control (10-13%) and terrible control (> 13%).

According to this classification, our patients fall either in the category of very good or fair glycemic control achieved by their regular attendance to diabetic clinic which existed before the study was initiated. Regular measurement of HbA1 minimizes the problem of multiple sampling errors attendant with blood glucose measurement and unreliability of urine glucose test. It also serves accuracy, reliability and compliance of the child.

Larsen et al., (1990) have found that regular monitoring of hemoglobin A1C measurements for one year led to a decrease in hemoglobin A1C in patients with IDDM. The decrease was mainly due to lowering of hemoglobin A1C values in patients with poor glycemic control and it was achieved by better identification of such patients. These patients were seen more often in the clinic, had more frequent changes in their insulin reginens, and the number of their short term hospital admissions decreased. The distribution of hemoglobin A1C values in the group monitored by larsen and his colleagues was very similar to that found by Goldstein et al., (1982), among 187 youths with diabetes in an open longitudinal study, 47 percent of patients reached hemoglobin A1C values below 9%. Larsen et al., (1990) has found that, although the decrease in the mean hemoglobin A1C value from 10.1 to 9.5 percent in the monitored group was significant, it represents a calculated decrease in mean blood glucose of only 1m mol per liter (20 mg per deciliter).

(Hinde and Johnston., 1986) have reported a considerable seasonal variation in HbA1 concentration with a nadir in spring and early summer. It's therefore important to measure and compare HbA1 concentrations from one year to the next one and care must be taken to eliminate the effect of seasonal variation (Allgrove., 1988).

It is well established now, that serum C-peptide is considered an accurate, though, indirect measure for the insulin secretory activity of pancreatic B-cell (Horwitez et al., 1975; Cauter et al., 1992). First c-peptide is secreted in equimolar amounts by islet of langerhans (drury, 1986). Second in contrast to insulin, c-peptide is removed from the circulation to a minor degree by the liver (Kuhl et al., 1978). Third, c-peptide has a constrat peripheral clearance under various physiological circumstances (Polonsky and Rubenstein, 1984).

In the present work (Table 1) the mean fasting serum c-peptide level in the diabetic group was lower compared to the mean fasting values of the control group.

This findings coincided with that of other researchers (Drury., 1986; Fukuda et al., 1988). who also reported a statistically significant lower c-peptide value in IDDM patients compared to the normal controls.

It is possible that the fasting c-peptide level in IDDM reflects only the basal function of B-cell, mean while in response to meals or glucose stimulation, these patients, may be unable to release additional insulin (Porte and Bagdade., 1970).

The DCCT research group in (1987), supported the view of stimulation tests when assessing residual B-cell function in IDDM patients, those patients with very low basal c-peptide values were able to increase their serum levels substantially when challenged. The use of a standardized mixed meal feeding may help to identify non responders (Shah et al., 1989). Stimulation with glucagon or standardized breakfast meal yield the same response (Madsbad et al., 1978).

The significance of residual insulin secretion, established by measurement of the plasma c-peptide immunoreactivity (CPR) level, in the degree of metabolic control of patients with IDDM has been subjected to much controversy. Some investigators reported that B-cell function contributed to the stability of blood glucose level and to the reduction of required insulin dosage (Shima et al., 1977; Fukuda et al., 1988).

Nakanishi et al., (1990) has reported that CPR responders who were defined based on the sensitive CPR assay showed low HbA1C and FBG values than those in CPR non responders. In contrast Lutterman et al., (1981) reported that residual insulin secretion is not a prerequisite for the stability of blood glucose levels in IDDM patients.

We tried to speculate the relationship between residual pancreatic B-cell reserve, estimated by c-peptide level and degree of metabolic control (determined by GHbA1, blood glucose, number of hospital admission for control and for diabetic ketoacidosis and also by insulin dose).

In the present work (Table 13) there was no correlation between basal c-peptide values and glycosylated hemoglobin and border low significance with blood glucose.

A similar study was accomplished by the DCCT research group in 1987 on a larger sample size (610 diabetic patients) and no difference in glycosylated hemoglobin was observed among these patients.

We preferred to use basal c-peptide values for correlation and comparative statistics better than stimulated values for several reasons. Firstly fasting state is more reliable than stimulated state because of variation in size or composition of the mixed carlohdrate meal. Secondly, bacal c-peptide value, were strongly correlated to stimulated values. Thirdly no significance difference was observed between both.

Shah et al., 1989 reported the abscence of relationship between c-peptide values and degree of glycemic control.

Although long term glycemic control is markedly affected by the residual c-peptide secretion but also other factors such as good compliance of the patient to the diabetic clinic, diet control, emotional and physical stress and degree of physical activity may be of great importance for control of the disease and may well explain the discrepancy of the different parameters of the control with age or duration reported by authors (Kennedy and Lyons., 1989).

In the present work, (Table 2). The comparison between cases and controls as regards serum insulin was highly significant. Type I diabetes is

characterized by a progressive B-cell destruction leading to an absolute insulin deficiency.

Recent reports indicate that the B-cell destruction often starts long before the clinical onset of the disease (Gorsuch et al., 1981; Srikanta et al., 1985).

Even after the clinical onset most type I diabetics show residual B-cell function as indicated by measurable immunoreactive insulin (Ludvigsson and Heding, 1978; Heinze et al., 1979; Madsbad et al., 1980).

The duration of such residual B-cell function is however highly variable (Vetter et al., 1980; Knip et al., 1982). During the first 2 years of diabetes, most patients have detectable residual B-cell function, there after the prevalence declines to about 15% after 10 year (Madsbad et al., 1978). Gepts and Lecompte in (1981) found that most of the islets examined in post mortem specimens were composed only of A and D cells, the number of B-cells was reduced to 10% of the normals or less. The effect of duration of IDDM on serum c-peptide and serum insulin values was studied in the present work (Table 15), both basal c-peptide and insulin levels were significantly reduced in patients with 5 or more years duration compared to those with a duration less than 5 years.

It has been shown in several studies that the duration of diabetes influences the prevalence of residual B-cell function; (Block et al; 1971) found a group of patients who had diabetes before age 16 years, it was found that residual B-cell function was present in 75% of patients with diabetes of five years or less duration, only 20% of the patients with duration longer than

5 years had functioning beta cells. In another study (Faber et al., 1976) found 83 patients with age at onset of diabetes ranging from one to 50 years, 82 percent in the one to five years group had residual beta cell function compared to 16 percent in the group with duration longer than five years.

In the present work (Table 15), the correlation coefficient between duration and glycosylated hemoglobin was significant. This is not in accordance with the finding of Allen et al., (1992) who reported a considerable stabilization in HbA1 values during the 2nd year of study. On the other hand, Shah et al., (1989) have found that glycosylated HbA1 levels in all subjects were reduced to the non diabetic range during the first 3 months after diagnosis regardless of the initial therapeutic intervention. In the same study, levels in the conventionally treated group began to exceed the non diabetic range after six months and continued to increase for the remainder of the year, this increase coincided with the decline in the stimulated c-peptide response (Shah et al., 1989).

Although long term glycemic control is markedly affected by the residual c-peptide secretion; other factors such as good compliance of the patients to the diabetic clinic, adherence to the prescribed diet, emotional and physical activity may be of great importance for their glycemic control and may well explain the discrepancy of objective parameters of control with age or duration reported by various authors (Kennedy and Lyons., 1989). This may explain our finding that the mean values of blood glucose monitoring were high in diabetics older than 10 years at onset. Diabetic patients falling in this age group are all adolescent who are rebellious to instructions, on diet, insulin injection and monitoring, imposed on their life by their condition.

In the present work (table 23), the comparison between males and females as regard, glycosylated hemoglobin was significant

Stickland and his colleagues (1984) reported that glycosylated hemoglobin proved to differ between diabetic female and male subjects despite similar plasma glucose concentrations, suggesting a role for gender in metabolic control.

A larger clinical study on the prevalent cases of IDDM in Pittsburgh also reported obsence of an overall difference in HbA1c values between males and females (Daneman et al., 1981).

Allen et al (1992) have reported that male adolescents had a poorer metabolic control than other male age groups and female adolescents during their 2 nd year of IDDM. They have explained this by the observation that male patients were less compliant with diabetes self management, taking fewer insulin injections which may indicate that insulin coverage is less optimal in this group. This contrasts with a clinical study at Duke University (Zimet and Thompson, 1989) in which investigations identified female adolescents as less well controlled than their male counterparts.

The role of sex on early metabolic remission in IDDM patients. Dahlquist et al., (1988) have found that the early loss of B - cell function was unrelated to sex and was somewhat at variance with the findings of knip et al., (1982). Whoreported a higher frequency of clinical remission in boys compared to girls. However it must be emphasized that clinical remission is not equivalent to residual B- cell function but rather a combination of that and the peripheral insulin sensitivity (YKi - Jaryinen and Koivisto., 1984).

In the present work (Table 24) the comparison between males and females as regard serum amylase levels and serum lipase levels (Table 25) among cases showed no significant difference i.e. no effect of sex on exocrine function of pancreas. (Table 16) (Table 17) showed no significant difference between males and females as regards serum lipase and amylase enzyme among control.

As regards exocrine function of pancreas in our study (tables 5 and 6) Show a comparison between serm lipase and amylase levels among controls and cases; there is no significant difference between cases and controls.

i.e. No effect of diabetes on exocrine function of pancreas.

No reference is available about the relation ship between diabetes and serum lipase and serum amylase enzyme.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

The relation between residual pancreatic B- cell capacity and glycemic control has been emphasized by many researchers. Recently the development of an accurate assay to measure C-peptide concentration released in equimolar amount with insulin provide an accurate method of assessing beta cell function.

The main objective of the work is to evaluate the glycemic control in a group of the IDDM patients and relate it to the residual insulin secretion indirectly measured by C-peptide and to assess the exocrine function of pancreas measured by serum lipase and amylase enzymes.

The study included 50 IDDM children (28 males and 22 females) and 25 controls (10 males and 15 females).

- Routine fasting blood glucose was done.
- Glycosylated hemoglobin was analysed.
- Fasting serum insulin and fasting serum C-peptide was done.
- Serum lipase and serum amylase enzyme was done.

Analysis Of The Data Revealed The Following Results:

- Patients had a higher glycosylated HbA1 than controls (9.0 ± 1.6 % Vs 6.9 ± 0.5 % $P < 0.001$)
- Patients had low fasting serum C-peptide than controls (0.04 ± 0.02 Vs. 7.31 ± 5.35 $P < 0.001$)

- Patients had high fasting serum insulin than controls (199.4 ± 90.48 VS 19.54 ± 8.56 P< 0.001) (That is due to exogenous insulin as treatment of diabetes).

- Patients had no significant difference in serum amylase than controls.

  (83.43 ± 25.63 Vs 84.26 ± 25.61 P>0.05).

- Patients had no significant difference in serum lipase than controls.

  (76.4 ± 21.7 Vs 68.4 ± 38.1 P>0.05)

- Basal C-peptide values were significantly reduced, the longer the duration of diabetes, the younger the age of onset and more in males.

In Conclusion:

The presence of correlation between HbA1 and mean value of glucose indicate that regular follow up in a clinic specialized in diabetes has a positive influence on long term glycemic control assessed by HbA1.

Also our results indicate that there is no effect of diabetes on exocrine function of pancreas, it provides an evidence of pancreatic B-cell destruction in type I diabetics which is significantly shortened by younger age of onset, longer duration of diabetes and male gender. This may be helpful in selection of patients who would not likely benefit from the future therapeutic modalities such as immunosuppresion or other therapies. Future information derived from analysis of the recovery phase may be helpful in identifying the optimal time for intervention strategies.

RECOMMENDATIONS

RECOMMENDATION

From the work presented we recommend the following:

1- Every effort should be paid to optimize glycemic control in IDDM patients. Routine blood glucose monitoring should be available and its accuracy should be continuously assessed. Also determination of glycosylated hemoglobin A1 is a complementary test for routine blood glucose monitoring, both will optimize glycemic control in IDDM patients.

2- Regular follow up of diabetic patients in a specialized diabetic unit has an influenced role on the degree of glycemic control of patients and continuous diabetes education program should be always emphasized by medical personnal dealing with diabetes.

3- Further effort should be directed to study c-peptide in different diabetic stages (acute, remission and established phases) to determine its course and also to study some of the individual cases, both may put more insight on variuos factors which may have an impact on residual pancreatic B-cell among diabetic population.

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