RESULTS

I- General Description of the sample

A- Age and Sex : table (1) to (3)

Describing age and sex distribution of the studied cases and controls.

Table (1): Shows that the mean ages for studied cases and control groups were (43.6 ±13.7) and (43 ±12.5) respectively with no statistically significant difference between both groups (P>0.05).

Table (2): Shows that the mean age of female studied cases (44 ± 0.2) is higher than that of males (39 ±21.2). This difference is not statistically significant (P>0.05).

Table (3): Describing number and percent distribution of the studied cases according to sex. This table shows that the higher percentages of studied male and female cases were over 50 years of age

B- Levels of education and occupation : table (4) to (7)

Tables (4): Percent distribution of studied cases and controls according to their level of education. This table shows that the highest percentage of studied cases and controls are illiterate (59% and 60% respectively). High education represents the lowest percentage among both groups (8% and 6% respectively). This difference in not statistically significant.

Table (5): Percent distribution of male and female studied cases according to levels of education. This table show that the highest percentage (38.4%) of male studied cases was basic education on the other hand, most of female studied cases are illiterate representing 55 cases (63.2%) out of 87 studied females.

Table (6) and table (7), describing percent distribution of male and female studied cases and controls according to their classes of occupation.

Table (6): Shows that 81 cases (93.1%) out of 87 studied female cases are non workers.

Table (7): Shows that the highest percentage (53.8%) of male studied cases were unskilled workers.

Conclusively :

- No difference in the mean age of studied cases and controls.
- Mean age of female cases in higher that of males, but the difference is not statistically significant.
- Most of the male and female studied cases are illiterate
- Most of the female cases are non workers and most of male cases are unskilled workers.

Table (1): Mean ($\bar{X}$) and standard deviation (SD) of age of the studied cases and control group.

Age	Cases	Controls
	n = 100	n = 100
$\bar{X}$	43.6	43.8
SD ±	13.7	12.5

t = 0.141 P>0.05

Table (2): Mean ($\bar{X}$) and standard deviation (SD) of male and female studied cases.

Age	Males	Females
	n = 13	n = 87
$\bar{X}$	39	44.2
SD ±	21.2	12.2

t = 1.29 P>0.05

Table (3): Number and percent distribution of age of the studied cases according to sex.

Age groups	Male n = 13		Females n = 87		Total n = 100	
	No.	%	No.	%	No.	%
20-	1	7.7	10	11.5	11	11
30-	4	30.8	20	23	24	24
40-	1	7.7	28	32.2	29	29
50+	7	53.8	29	33.3	36	36
Total	13	100	87	100	100	100

Table (4): Percent distribution of the studied cases and control groups according to levels of education.

Age groups	Cases n = 100		Controls n = 100		Total n = 200	
	No.	%	No.	%	No.	%
Illiterate	59	59	60	60	119	59.5
Basic education	33	33	34	34	67	33.5
High education	8	8	6	6	14	7
Total	100	100	100	100	200	100

$X^2 = 1.27$ P>0.05

NB. :

Basic education = primary + preparatory.

High education = Secondary and university.

El Gendy (1986).

Tables (5): Percent distribution of male and female studied cases according to levels of education.

Levels of education	Male n = 13		Females n = 87		Total n = 100	
	No.	%	No.	%	No.	%
Illiterate	4	30.3	55	63.2	59	59
Basic education	5	38.4	28	32.2	33	33
High education	4	30.3	4	46	8	8
Total	13	100	87	100	100	100

Table (6): Percent distribution of female studied cases and control groups according to occupation.

Occupation	Cases n = 87		Females n = 87		Total n = 174	
	No.	%	No.	%	No.	%
Workers	6	6.9	4	4.6	10	58
Non workers	81	93.1	83	95.4	164	94.2
Total	87	100	87	100	174	100

Table (7): Distribution of male studied group and control group according to occupations.

Occupations	Cases n = 13		Control n = 13		Total n = 26	
	No.	%	No.	%	No.	%
Leading profession and skilled workers	6	46.2	4	30.8	10	38.4
Unskilled workers	7	53.8	9	69.2	16	61.6
Total	13	100	13	100	26	100

*** NB: Barker (1982)**

Table (1.4): Describing percent distribution of male and female studies cases according to family history. This tables shows a highest percentage (15.4%) of family history among male studied cases than females (13.8%), though, this difference is not statistically significant.

Conclusively: A positive family history was found among cases than controls and among female studied cases than males.

Table (1.1): Percent distribution of studied cases and control groups according to family history of gall stone disease.

Family history	Cases n = 100		Controls n = 100		Total n = 200	
	No.	%	No.	%	No.	%
Positive history	14	14	5	5	19	9.5
Negative history	86	86	95	95	181	90.5
Total	100	100	100	100	200	100

X2 = 4.711 P <0.05

Table (1.2): Percent distribution of female studied cases and controls according to family history of gall stone disease.

Family history	Cases n = 87		Controls n = 87		Total n = 174	
	No.	%	No.	%	No.	%
Positive history	12	13.8	5	5.7	17	9.8
Negative history	75	86.2	82	94.3	157	90.2
Total	87	100	87	100	174	100

$X^2 = 3.194$ P >0.05

Table (1.3): Percent distribution of male studied cases and control groups according to family history of gall stone disease.

Family history	Cases n = 13		Controls n = 13		Total n = 26	
	No.	%	No.	%	No.	%
Positive history	2	15.4	0	0	2	7.7
Negative history	11	84.6	13	100	24	92.3
Total	13	100	13	100	26	100

Table (2.1): Percent distribution of female studied cases and control groups according to number of pregnancies.

Number of pregnancies	Cases n = 87		Controls n = 87		Total n = 174	
	No.	%	No.	%	No.	%
2-	11	12.7	9	10.3	20	11.5
4-	28	32.2	26	29.9	54	31
6+	44	50.5	47	54	91	52.3
Total	83	95.4	100	94.2	165	94.9

$X^2 = 37$ P >0.05

Table (2.2): Percent distribution of female studied cases and controls according to contraceptive pills use.

Coutraceptive pills use	Cases n = 87		Controls n = 87		Total n = 174	
	No.	%	No.	%	No.	%
Users	42	48.3	35	59.8	77	44.3
Non users	45	51.7	52	40.2	97	55.7
Total	87	100	87	100	174	100

$X^2 = 1.15$ P >0.05

Table (2.3): Mean and standard deviation of different variables contributing to a hormonal risk factors for gall stone disease.

Variables	Cases			Controls			t	P
	No.	Mean	+SD	No.	Mean	±SD		
Age at marriage	*87	18.3	5.03	*87	17.3	4.03	1.6	>0.05
Onset of menarche in years	*87	13.1	1.5	*86	13.5	1.49	1.9	>0.05
Age at menopauuse in years	**35	44.6	8.8	**26	46.4	6.5	0.9	>0.05
Duration of menstrual life in years	**35	32.1	7.3	**26	33.2	6.3	0.7	>0.05
Age at first pregnancy in years	***83	17.6	3.8	*** 82	17.6	2.6	0.2	>0.05
Number of pregnancies	*** 83	6.8	3.4	*** 82	6.7	3.3	0.02	>0.05
Duration of pills use in years	**** 42	3.8	3.7	**** 35	3.6	3.1	0.1	>0.05

NB :

* Total number of female studied cases

** Number of females reached menopause

*** Number of females getting pregnancy.

**** Number of females using contraceptive pills.

Table (2.4): Distribution of female studied cases according to age at first pregnancy and age at discovery of gall stone disease.

Age at first pregnancy	Number of cases	Mean age at discovery	SD
15-	20	52.9	12.3
20-	44	43.9	11.6
25+	23	42.1	11.5

F = 3.68 P <0.05

3- Diet: Table (3.1) to (3.3)

Describing percent distribution of studied cases and control groups according to the consumption of different types of food.

Table (3.1): shows that 87 cases (87%) out of 100 studied cases, and (71%) of control group were animal fat consumers with a highly statistically significant difference between both groups ($P<0.01$). The same statistically significant difference in fat consuming is shown between female studied cases (89.6%) and controls (67.8%) in **table (3.2)** ($P<0.01$).

On the other hand, male studied cases consuming animal fat represent a lower percentage (69.2%) than control group (92.3%). This difference is not statistically significant ($P>0.05$) as shown in **table (3.3)**.

Animal protein consumers represent a higher percentage among all studied cases than controls, they represent 97 cases (97%) out of 100 studied cases and (94%) of control group as shown in table (3.1), (96.5%) of female studied cases and (95.4%) of control group as shown in table (3.2) and (100%) of male studied cases and (84.6%) of control groups shown in table (3.3). These differences are not statistically significant ($P>0.05$).

Vegetable consumers represent a lower percentage among studied cases than controls, they represent (95%) of

all studied cases and (97%) in control groups shown in table (3.1) and (84.6%) among male studied cases and (100%) of control group as shown in table (3.3). These differences are not statistically significant (P>0.05)

Table (3.4): Describing percent distribution of male and female studied cases according to consumption of different types of food.

This table shows that animal protein consumers show a higher percentage among male studied cases (100%) than females (69.5%) while animal fat consumers represent a higher percentage (89.6%) among female studied cases than male (69.6%) Difference in fat consuming is statistically significant.

Also, male studied cases show the lowest percentage (84.6%) of vegetable protein consumers than females (96.6%). This difference is not statistically significant (P>0.05).

Conclusively :

Fat consumers represent a high percentages of studied gall stone cases than controls. The highest percentage is shown among female studied cases (89.6%) with a highly statistical significant difference than controls.

Animal protein consumers represent a high percentage among all studied cases than controls being of highest percentage (100%) among male studied cases.

Vegetable consumers represent a lower percentage among studied cases than controls being of lower percentage among male studied cases.

Table (3.1): Percent distribution of studied cases and control groups according to the consumption of different types of food.

Food stuffs	Cases n = 100		Controls n = 100		Total n = 200	
	No.	%	No.	%	No.	%
Animal fat	87	87	71	71	158	79
Animal proteins	97	97	94	94	191	95.5
Vegetable proteins	95	95	97	97	192	96

X^2 for animal fat consumers versus controls = 6.7

P<0.01

X2 for animal protein consumers versus controls = 0.13

P>0.05

X2 for vegetable protein consumers versus control = 2.5

P>0.05

N.B : Comparison were done according to score (1) consumers described by Attili and GREPCD group (1984):

Score 0: consumption once or twice/month

Score 1: consumption 1-4 times/week

Score 2: consumption 7 times/week and more than once/days.

* Carbohydrate diet is excluded from comparison as both studied cases and controls share the same consumption score (score 2).

Table (3.2): Percent distribution of studied female cases and controls groups according to the consumption of different types of food.

Food stuffs	Cases n = 87		Controls n = 87		Total n = 174	
	No.	%	No.	%	No.	%
Animal fat	78	89.6	59	67.8	137	78.9
Animal proteins	84	96.5	83	95.4	167	95.9
Vegetable proteins	84	96.5	84	96.5	168	96.5

X^2 for fat consumers versus controls = 11.1 P<0.01

Table (3.3): Percent distribution of male studied cases and control according to the consumption of different type of foods.

Food stuffs	Cases n = 13		Controls n = 13		Total n = 26	
	No.	%	No.	%	No.	%
Animal fat	9	69.2	12	92.3	21	80.0
Animal proteins	13	100	11	84.6	24	92.3
Vegetable proteins	11	84.6	13	100	24	92.3

X^2 for fat consumers versus controls = 0.99
P>0.05

X^2 for animal protein consumers versus control = 0.46
P>0.05

X^2 for vegetable protein consumers versus controls = 0.14
P>0.05

Table (3.4): Percent distribution of male and female studied cases according to the consumption of different types of food.

Food stuffs	Males n = 13		Females n = 87		Total n = 100	
	No.	%	No.	%	No.	%
Animal fat	9	69.2	78	98.6	87	87
Animal proteins	13	100	84	96.5	97	97
Vegetable proteins	11	84.6	84	96.5	95	95

X^2 for male consuming fat versus females = 0.10 P <0.05

X^2 for male consuming animal proteins versus females = 0.84 P >0.05

X^2 for male consuming vegetable proteins versus females = 0.24 P >0.05.

4- Dietary habits : table (4.1) to (4.4)

Describing percent distribution of studied cases and controls according to dietary habits predisposing to gall stones.

Table (4.1): Shows that snack users represent 40 cases (40%) out of 100 studied cases and (15%) of control group. This also represents (42.5%) of female cases and (14.9%) of their control as shown in table (4.2). Both differences are statistically significant ($P<0.05$).

Table (4.1) and Table (4.2): Show that (8%) of studied cases and (9.2%) of female studied cases were practicising diet regimen the difference between both studied group and their control is not statistically significant ($P>0.05$).

Table (4.4): Shows that the highest percentage (42.5%) of snack users were among female studied cases while they represent only (23.1%) of male studied cases. The difference is not statistically significant ($P>0.05$).

Conclusively :

Dietary habits including using of snacks and dieting, show higher percentage among the whole studied cases and female studied cases than controls the difference is statistically significant. None of males were practicing diet regimen.

Table (4.1): Percent distribution of the studied cases and control groups according to dietary habits suggesting the development of gall stone disease.

Dietary habits	Cases n = 100		Controls n = 100		Total n = 200	
	No.	%	No.	%	No.	%
Shack users	40	40	15	15	55	27.9
Diet regimen practicisers	8	8	7	7	15	7.5

X^2 for shack users versus controls = 14.5
P<0.05.

X^2 for diet regimen practicisers versus controls = 0.8
P >0.05.

Table (4.2): Percent of distribution of female studied cases and control group according to dietary habits suggesting the development of gall stones.

Dietary habits	Cases m = 87		Females m = 87		Total m = 174	
	No.	%	No.	%	No.	%
Snack users	37	42.5	13	14.9	50	28.7
Diet regimen practicisers	8	9.2	7	8.1	15	8.6

X^2 for shack users versus controls = 14.9
P <0.05.

X^2 for diet regimen practicisers versus controls = 0.07
P >0.05.

Table (4.3): Percent distribution of male studied cases and control groups according to dietary habits suggesting the development of gall stone disease.

Snack	Cases n = 13		Controls n = 13		Total n = 26	
	No.	%	No.	%	No.	%
Users	3	23.1	2	15.4	5	19.2
None users	10	76.9	11	84.6	21	80.8
Total	13	100	13	100	26	100

Z = 0.50 P >0.05

* NB. : None of the studied male cases and controls were practicising diet regimen.

Table (4.4): Percent distribution of male and female studied cases according to dietary habits suggesting the development of gall stone disease.

Snack	Cases n = 13		Females n = 87		Total n = 100	
	No.	%	No.	%	No.	%
Users	3	23.1	37	42.5	40	40
None users	10	76.9	50	57.5	60	60
Total	13	100	87	100	100	100

Z = 1.61 P >0.05

5- Obesity: Table (5.1) to (5.3)

Describing mean and standard deviation of Body Mass Index and body fat % as indications of obesity.

Table (5.1): Shows that the mean body mass index (BMI) in kgm/m^2 of all studied cases and control group were (28.7 ±6.8) and (26.2 ±5.5) respectively. This difference is statistically significant ($P < 0.05$).

Table (5.2): Shows that the mean BMI of female studied cases and controls were (29.2 ±6.8) and (26.6 ±5.5) respectively. This difference is statistically significant.

Table (5.1) and (5.2): Show that the mean body fat % for all studied cases and controls is (37.9 ±7.1) and (36.1 ±6.2) respectively (table 5.1).

Also shows that the mean fat % for studied female cases and controls were (39.1 ±5.8) and (37.3 ±5.4) respectively (table 5.2). These differences were statistically significant ($P < 0.05$).

Table (5.3): Shows that the mean BMI (25.5 ±6.6) and body fat % (29.5 ± 8.7) of the studied male cases are higher than those of controls (23.8 ±4.5) and (28.8 ±4.9) respectively. These differences are not statistically significant ($P > 0.05$).

Table (5.4): Shows that the mean BMI (29.1 ±6.8) and mean body fat % (39.1 ±5.8) of female studied cases were higher than males (25.5 ±6.5) and (29.5 ±8.7) respectively. This difference is statistically significant ($P < 0.05$).

Conclusively :

All gall stones studied cases show a higher BMI and body fat % than their controls the difference is highly significant ($P < 0.01$) among female studied cases than controls and among female studied cases than males.

Table (5.1): Percent distribution of studied cases and control groups according to anthropometric measurements.

Anthropometric measurements	Cases n = 100		Controls n = 100		t	P
	Mean ± SD		Mean ± SD			
Weight in Kg	72.1	17.2	67.03	13.7	2.3	<0.05
Height in cm	159.3	9.3	58.5	7.6	.63	>0.05
BMI (Kg/cm^2)	28.7	6.8	26.2	5.5	2.8	<0.05
Body fat %	37.9	7.1	36.1	6.2	1.79	>0.05

Table (5.2): Percent distribution of female studied cases and controls according to their anthropometric measurements.

Anthropometric measurements measurements	Cases n = 87		Controls n = 87		t	P
	Mean ± SD		Mean ± SD			
Weight in Kg	72.5	17.4	67.2	13.9	2.19	<0.05
Height in cm	158.3	9.3	157.6	7.6	0.57	>0.05
BMI (Kg/cm^2)	29.2	6.8	26.6	5.5	2.77	<0.05
Body fat %	39.1	5.8	37.3	5.4	2.11	<0.05

Table (5.3): Percent distribution of male studied cases and control groups according to anthropometric measurements.

Anthropometric measurements	Cases n = 13		Controls n = 13		t	P
	Mean ± SD		Mean ± SD			
Weight in Kg	29.5	8.7	28.8	4.9	0.27	>0.05
Height in cm	166	6.2	165.8	3.1	0.64	>0.05
BMI (Kg/cm^2)	25.5	6.6	23.8	4.5	0.43	>0.05
Body fat %	29.5	8.7	28.8	4.9	0.27	>0.05

6- Diseases : table (6.1) to (6.4)

Describing percent distribution of studied cases and controls according to diseases predisposing to gall stones.

Table (6.1): Shows a higher percentage (40%) of bilharziasis among all studied cases than controls (32%), cirrhosis represents (10% of studied cases and (2%) of controls. Also diabetics represent (3%) and (1%) of studied cases and controls respectively. The difference is only statistically significant in case of cirrhosis ($P < 0.05$).

Table (6.2): Show a higher percentage of Bilharziasis among female studied cases (32.2%) and controls (25.3%).

Also, bilharziasis show a higher percentage among male studied cases (92.3%) than controls (69.2%) as shown in table (6.3) differences were not statistically significant ($P > 0.05$).

A higher percentage of both cirrhotics and diabetics were found among female studied cases (8.1% and 2.3% respectively) than controls (2.3% and 1.2% respectively) as shown in table (6.2). No statistically difference was found between both groups ($P > 0.05$).

Table (6.3): Shows a higher percentage of bilharziasis cirrhosis and diabetes among studied cases (92.3%, 23.1% and 7.7% respectively) than controls (69.2%, 0%, 0% respectively).

Table (6.4): Describing percent distribution of male and female studied cases according disease predisposing to gall stones.

This table shows a higher percentage (92.3%) of bilharziasis among male cases than females (32.2%) the difference is statistically significant. Liver cirrhosis and diabetes have a higher percentage among male studied cases (23.1%) and (7.7%) than female (8.1%) and (2.3%) respectively. These differences were not statistically significant.

Conclusively :

All diseases contributing to the risk factors of gall stones have higher percentages among male studied cases than females. However bilharziasis has the highest percentage among all of then.

Table (6.1): Percent distribution of studied cases and control groups according to diseases suggesting predisposition to gall stones.

Diseases	Cases n = 100		Controls n = 100		Total n = 200	
	No.	%	No.	%	No.	%
Bilharziasis	40	40	32	32	72	36
Liver cirrhosis	10	10	2	2	12	6
Diabetes	3	3	1	1	4	2

X^2 for bilharzial cases versus controls = 1.39
P >0.05

Z test for cirrhotic cases versus control = 2.41
P >0.05

Z test for diabetic cases versus controls = 1.01
P >0.05

Table (6.2): Percent distribution of female studied cases and control group according to diseases suggesting predisposition to gall stones.

Diseases	Cases n = 87		Controls n = 87		Total n = 174	
	No.	%	No.	%	No.	%
Bilharziasis	28	32.2	22	25.3	50	28.8
Liver cirrhosis	7	8.1	2	2.3	9	5.2
Diabetes	2	2.3	1	1.2	3	1.8

X^2 for bilharzial cases versus controls = 1.01
P >0.05

Z test for cirrhotic cases versus control = 1.73
P <0.05

Z test for diabetic cases versus controls = 0.58
P >0.05

Table (6.3): Percent distribution of male studied cases and control groups according to diseases suggesting predisposition to gall stone.

Diseases	Cases n = 13		Controls n = 13		Total n = 200	
	No.	%	No.	%	No.	%
Bilharziasis	12	92.3	9	69.2	21	80.8
Liver cirrhosis	3	23.1	0	0	3	11.6
Diabetes	1	7.7	0	0	1	3.9

X^2 for bilharzial cases versus controls = 0.12
P >0.05

Table (6.4): Percent distribution of male and female studied cases according to diseases suggesting predisposition to gall stones.

Diseases	Male n = 13		Females n = 87		Total n = 100	
	No.	%	No.	%	No.	%
Bilharziasis	12	92.3	28	32.2	40	40
Liver cirrhosis	3	23.1	7	8.1	10	10
Diabetes	1	7.7	2	2.3	3	3

X^2 for bilharzial cases versus controls = 17.1
P <0.05

Z test for cirrhotic cases versus control = 1.24
P >0.05

Z test for diabetic cases versus controls = 0.71
P >0.05

IV- Investigations

1- Blood group : table (7.1) and (7.3)

Table (7.1): Describing percent distribution of studied cases and control groups according to blood grouping.

This table shows that blood group (0) shows a higher percentage (38%) among all studied cases than control group (36%).

In the other hand, blood group (AB) shows a lowest percentage (2%) among studied cases than controls (9%) no statistical significant difference in blood grouping is found between case and controls (P >0.05).

Table (7.2): Describing percent distribution of male and female studied cases according blood grouping.

This table shows that blood group (A) and blood group (0) show the same higher percentage (38.5%) among studied male cases, although this percentage is lower than that of female cases in which blood group (A) represent (37.9%). No statistical significant difference was found between both of male and female studied cases in their blood groups (P >0.05).

Table (7.3): Describing laboratory finding including blood haemoglobin, fasting blood sugar and liver function tests among studied cases and control groups.

This table shows a significant higher mean fasting blood sugar (96 ±24.4) among studied cases than controls (86.7 ±22.3) ($P < 0.05$).

Conclusively :

The majority of gall stone studied cases were blood group (0) the higher percentage of male studied cases were blood group (A) and (0), while the higher percentage of female studied cases were blood group (0). A significant higher mean fasting blood sugar is found among studied cases than controls.

Table (7.1): Percent distribution of the studied cases and controls according to blood grouping.

Blood groups	Cases n = 100		Controls n = 100		Total n = 200	
	No.	%	No.	%	No.	%
A	34	34	36	36	70	35
B	26	26	24	24	50	25
AB	2	2	9	9	11	5.5
O	38	38	31	31	69	34.5
Total	100	100	100	100	200	100

$X^2 = 5.3$ P >0.05

Table (7.2): Percent distribution of male and female studied cases according to blood grouping.

Blood groups	Males n = 13		Females n = 87		Total n = 100	
	No.	%	No.	%	No.	%
A	5	38.5	29	33.3	34	34
B	2	15.4	24	27.6	26	26
AB	1	7.6	1	1.2	2	2
O	5	38.5	33	37.9	38	38
Total	13	100	87	100	100	100

$X^2 = 3.16$ P >0.05

Table (7.3): Mean (x) and standard deviation (SD) of laboratory findings of studied cases and control groups.

Laboratory investigations	Cases n = 100		Controls n = 100		t	P
	Mean ± SD		Mean ± SD			
Hb (gm%)	12.02	8.4	10.9	1.03	1.3	>0.05
Fasting blood sugar (mg%)	96.3	24.4	86.7	22.3	2.8	<0.05
Bilirubin (mg/100cc)	1.12	2.3	6.8	0.42	1.8	>0.05
AST (I11)	10.6	5.6	10.5	6.3	0.12	>0.05
ALA (I11)	10.4	4.9	11.2	5.7	1.08	>0.05
Serum albumin (gm/100cc)	3.99	2.6	4.1	4.3	0.28	>0.05

Table (8): Describing the correlation between age at discovery of gall stone and different variables contributing to disease occurrence.

This table shows a positive significant correlation between age at discovery of gall stone disease and duration of menstrual life ($P < 0.01$) and a negative significant correlation between age at discovery, body mass index and body fat % ($P < 0.05$). Also a negative significant correlation is found between age at marriage and age at discovery of gall stone disease.

Table (8): Correlation coefficient (r) between age at discovery and different variable contributing to the risk factors of gall stone diseases.

Variables	r	P
Age at marriage	-0214	<0.05
Duration of menestrual life	0.568	<0.01
Duration of contraceptive pills use	0.142	>0.05
Number of pregnancies	0.501	>0.05
Body mass index	-0.121	<0.05
Body fat %	-0.221	<0.05

Step-wise regression analysis of different variables in relation to the occurrence of gall stone disease.

Table (9): Shows that BMI, Animal fat diet, animal proteins and liver cirrhosis are the most important factors affecting the occurrence of gall stone disease.

Table (9): Step-wise regression analysis of different variables in relation to gall stone disease.

Variables	Partial F
BMI	9.21
Animal fat diet	10.9
Animal proteins	15.02
Liver cirrhosis	5.6

DISCUSSION

Gallstone disease is a major public health problem in developed countries. In the last 20 years there have been a marked increase in the frequency of cholecystectomy in many countries although the rate is markedly different even in parts of the one country (Sali, 1990).

The undoubted increase in the frequency of gallstones may be due to an increase in the prevalence of gall bladder disease or to a change in the threshold accepted as an indication for surgical treatment. The threshold level is determined by the balance between demand for and the supply of health services. The demand reflects not only the incidence of the disease but also the awareness and tolerance of the symptoms and the accessibility and availability of medical services (Plant et al., 1973).

Gallstone disease has a significant impact on health care cost in terms of demands on medical personnel and utilization of health care facilities (Low-Beer, 1985). It is also costly in terms of time lost from work.

Despite the good therapeutic modalities for treatment of cholelithiasis, gallstones remain an important cause of morbidity and mortality (Grund and Mok, 1977). Gunn (1982) stated that there has been a 130% increase in the rate of

cholecystectomy over the last 15 years. He found an operative mortality rate of 0.427%. Moreover, **Ransohoff et al. (1983)** reported that the case fatality rate from gallstone disease was found to be 0.3% and the life expectancy was decreased by 7 days in a patient undergoing cholecystectomy at 30 years of age.

However it is difficult to establish the incidence of the disease because population studies using cholecystographic techniques are impractical. Moreover, operation rates are indirect measures of prevalence since those rates are affected by clinical policy and economic circumstances **(Godfray, 1984)**. Recently the use of echography provided a high sensitivity, safety and specificity in diagnosis of gallstones compared with traditional x-ray procedures.

For these reasons, the epidemiology of gallstone disease has attracted so much interest during the last few years so as to answer precise questions concerning aetiology, natural history and prevention of the disease especially after the availability of comprehensive and standardised procedures for the conducting of population field studies.

Finally the interrelationship between gallstone disease atherosclerosis and myocardial infarction is

another source of interest. In fact some common metabolic channels such as those involving lipid metabolism are apparently involved in such conditions. This stimulated many investigators dealing with cardiovascular and liver diseases to approach the epidemiology of gallstones **(Menotti, 1983).**

If risk factors of gallstone disease working through known pathogenic mechanisms can be identified and modified, it might found a foundation upon which to develop a program of gallstone prevention.

The present study was a case control study of a sample of male and female cases of gall stones in Benha city.

In this study the age of the patients ranged from 20 to 72 with a mean of 43.6. For females, it ranged from 20 to 72 with a mean of 44.24, and in males it ranged from 20 to 59 with a mean of 39. Non of our patient were below 20 years.

Agreeing with the present study is what was found by **Sali (1990)** who stated that gall stones are extremely rare before the age of 10 years. He also found a definite increase in gall stone disease in women from the time of puberty, this increase tends to rise sharply up to 50-60 years of age, then rises slowly.

Also, **Calabreses and Pearlman (1971)** found that only 5.3% of cholycystectomies in two New-York hospitals were below the age of 22 with only one patient was 10 years old.

Epidemiological studies have demonstrated that cholesterol stones have a predilection for women, a female to male preponderance have been observed by many authors **(Friedman et al., 1966, Thistle et al., 1971, Trotman and Soloway, 1975 and Bennion and Grundy, 1978).**

Agreeing with these findings, is what was found in the present study in which a female to male ratio of 6.7 to 1 has been found.

However **Sali (1990)** reported a ratio ranging between 2:1 to 4:1 only. The high preponderance of females in the present study may be explained by multiparity, obesity and other environmental factors including type of diet, dietary habits, genetic and familial factors that were observed in our patients.

LaMonte (1981) described the female preponderance to be due to endogenous estrogen and progesterone or both through an effect on bile saturation and smooth muscle function of the gall bladder and intestine during the phases of menstrual cycle and pregnancy.

In studying the factors suggesting hormonal role in the pathogenesis of gall stones, the present work has shown that there was no difference between the female cases studied and their controls as regards, the age at marriage, age of menarche, duration of menstrual life, age at 1st pregnancy, multiparity and duration of contraceptive pill use (table 23).

This finding might exclude the hormonal role in influencing the metabolism of cholesterol and the development of gall stones.

However in the literature, there is controversy concerning the effects of such factors on the pathogenesis of gall stones.

Strom and West (1985) stated that it has been suggested since the 19th century that pregnancy is associated with cholelithiasis.

Moreover, **Gollish et al. (1982)** found that the percentage of nulliparous females with gall stones was the same as the percentage of males with gallstones, but multiparous women had a greater incidence of cholelithiasis than nulliparous woman.

Also, **Honore (1980)** found a strong association between parity and cholelithiasis.

In contrast to the previous studies, and agreeing with the present study, is what was reported by **Sampliner et al. (1970)** that no association was found between risk factors contributing to hormonal effect on gall stone disease in their case control study.

On studying the correlation between the age of presentation of gall stone disease and the age at 1st pregnancy, in the present work, it was found that the older the age at first pregnancy, the younger the age of the presentation of gallstone disease. This may attract attention that patients getting their first pregnancy at an older age group are at a higher risk of developing gallstone disease. Agreeing with this **Layde et al. (1982)** found a significant trend for increased gallstone risk with older age at first pregnancy.

It has been proposed that the association between gall bladder disease and parity may be related to increased cholesterol saturation of bile during pregnancy. Another suggested explanation for the relation between gall bladder disease, age at first pregnancy and multiparity is gall bladder contractility. A woman having her first child at an older age may have less gall bladder

contractility after delivery than another woman who has had her first child at a younger age simply as a result of aging process.

In the present study, a significant negative correlation was found between the age at marriage and the age at discovery of gallstone disease. This may be explained by the fact that older patients at marriage have their first pregnancy at an older age, thus getting their symptoms earlier.

The onset of ovarian function in females has been found to be accompanied by a decrease in the total bile acids and chenodeoxycholic acid pools and a simultaneous increase in bile cholesterol saturation, increasing the possibility of gallstone formation. Cessation of ovarian function as a result of surgical castration is accompanied by expansion of the bile acid pool and a decrease in bile cholesterol saturation, suggesting that endogenous oestrogen and progesterone or both are responsible (Sali, 1990).

In contrast to this, no significant difference was observed in the present study in relation to the age of menarche, the age of menopause and the reproductive period (menstrual life) between the cases studied and their controls. This may suggest that these factors might not

have the leading role in determining the biochemical changes ending in gallstone formation (table 2.1).

Several studies reported an increased risk of gall stone disease among women using oral contraceptives. An increase in cholycystectomy rates paralleled an increase in oral contraceptive use have been reported by many authors. Oral contraceptive was found to slow gall bladder emptying, increase cholesterol secretion by 50% and decrease total bile acid secretion leading to increased bile lithogenicity (Boston Collaborative drug surveillance 1973).

In contrast to the previous studies, Layde et al. (1982) have failed to substantiate the theory that oral contraceptives users have more lithogenic bile and are at greater risk for gall stone disease. This agrees with the present study in which no statistically significant difference could be found between female studied cases and their controls in the duration of contraceptive pill use or in the percentage of contraceptive pill users.

A possible explanation for this lack of association is that contraceptive pill users are already at high risk for cholelithiasis due to other causes and that contraceptive pills accelerate the disease. Another explanation may be that cholelithiasis may be more

frequently diagnosed in women using oral contraceptives since women taking contraceptives are recognised to be at increased risk for a number of medical conditions so they tend to see their physicians more frequently and may be examined more thoroughly than non users (**Braverman et al., 1980, Layde et al., 1982** and the **Royal College of General Practitioner, 1982**).

Several studies have noted an association between the increasing incidence of gall bladder disease and excessive consumption of high purified carbohydrates and animal fats, with decreased intake of vegetables and fibres (**Strom and West., 1985**).

Moreover, **Sarles and Crotte (1971)** have found that patients with gall stones have a significantly higher intake of calories irrespective of their dietary composition. These results suggest that apart from its effect on causing obesity the ingestion of high calorie diets promotes gall stone formation through an increase in hepatic cholesterol secretion.

The questionnaire interview methods used for recording dietary habits in the present study does not permit calculation of daily intake of calories.

Moreover, the analysis of carbohydrate diet as a risk factor for gall stone is excluded from the study as both studied cases and control groups were equally sharing the same high score consumption of less purified carbohydrates. Nevertheless, patients of the present study are consuming more animal fat and animal proteins than their controls the difference being statistically significant in animal fat consumption.

On the whole, it was observed that the studied group of patients has a decreased intake of fibres and an increased intake of animal proteins and animal fat than controls. This agrees with what is reported in the literature by many authors who found that, it appears that the intake of excess animal proteins animal fat and refined carbohydrates with less intake of dietary fibres is associated with high risk of gall stone diseases **(Nagase et al., 1978, and Storm and West, 1985).**

This is explained by the fact that eating fats and proteins rather than full fibre foods inflates energy intake and depress fibre intake. In the short term, it makes bile more saturated with cholesterol at least in susceptible people though the mechanism is unknown. In the long term, if obesity results from this or any eating habits, the formation of gall stones will again be promoted **(Heaton, 1983).**

In contrast to the present study and with a common opinion that gall stone disease is associated with higher intake of animal protein and fats is what was reported by **Attilli and GREPCO (1984)** who found that women with gall stones consumed less proteins and less fatty meals and even less carbohydrates than women without gall stones. They explained this contrast by the fact that their patients were aware of their gall stones and were afraid of developing biliary colic thus modifying their diet and by the fact that their patients were older in age thus naturally modifying their food intake.

Increased biliary cholesterol saturation has been demonstrated during fasting while gall bladder stimulation has an opposite effect **Williams et al. (1977)**. Decreased meal frequency is regarded as a possible risk factor for the development of gall stone in man. Low frequency of snacks between meals and overnight feeding which could mean low gall bladder stimulation and contraction can be considered a risk factor for gallstone formation **(Attili and GREPCO group 1984)**.

In contrast to the previous study, the present study revealed that 40% of the studied patients with gall stones were used to have snacks between meals while only 15% of the controls used to do so.

The high frequency of snacks between meals observed in the present study that is considered by **(Attili and GREPCO Group 1984)** to be a protective factors against gall stone disease can still be considered a risk factors for the development of the disease in patients involved in the present study. A long term increase in energy intake through excessive consumption of food between meals almost certainly increases the risk of gall stones if it results in increased body fat **(Heaton, 1984)**.

Female studied patients were accustomed to have more snacks between meals (42%) than males (23%) this was highly statistically significant.

In the present study only 8% of patients practicised diet regimens for weight reduction for different periods of time compared to 7% of the controls which is not statistically significant.

Weight reduction was associated with a 25% incidence of cholesterol gall stone development over 8 weeks period of dieting in a study done by **Liddle et al. (1989)**. It is likely that weight reduction causes lithogenic bile through a marked reduction in bile acid secretion as a result of decreased hepatic secretion rates of all biliary lipids during caloric restriction periods. Also the

greater lithogenicity of bile during fasting is related to sequestration of bile acids in the gall bladder (Bloch et al., 1980).

Obesity is recognised as an important risk factor for gall stone disease (Friedman et al., 1966), Trotman and Soloway, 1975, Honore, 1980 and Layde et al., 1982).

Friedman et al. (1966) stated that people weighing at least 20% more than the median weight for their sex and height have nearly twice the risk of gall bladder disease than did people weighing 10% less than the medium weight.

Layde et al. (1982) found an increased risk of gall bladder disease to be significantly associated with Quetlet Index (BMI) (Wt/h+2) and Body fat % this agrees with what was have found in the present study.

The mean body mass index was found to be statistically significantly higher in our patients with gall stone disease (28.7) than in controls (26.2). Also a higher mean values of BMI and Body fat % were found in female studied cases than males.

In contrast other studies have failed to confirm the association between the BMI and gall tones. Sampliner et al. (1920) found that the mean BMI did not differ

significantly between individuals with gall bladder diseases and individuals without the disease.

Two possible explanation may account for these discripancies. First the populations studied may differ. Second, different investigators used different definitions of obesity thereby making comparisons among studies difficult.

The role of obesity as a risk factor for gall stone diseases is explained by **Shaffer and Small (1977)** as that obese persons have been found to have gall bladder bile that is more highly saturated with cholesterol than that of non obese persons.

Total bile acid pool sizes appear to be normal in obese individuals, suggesting that their highly saturated bile is a direct result of increased cholesterol secretion.

Bennion and Grundy (1978) found that although total bile acid pool size is not related to weight, the chenodeoxycholic acid pool size is inversely related to weight. Obese persons would have small chenodeoxy cholic acid pool sizes.

An increased familial frequency of gallstone disease is reported by many authors. **Gilat et al. (1983)** found

that the frequency of gallstone was twice as high among first degree relatives as compared with controls. Agreeing with this a statistically significant higher incidence of a positive family history of gallstone disease was observed in the present study among the cases studied than their controls.

The cause of increased familial frequency is uncertain and may be explained by the genetic aetiology of gallstone disease. The epidemiological evidence of a rapidly rising frequency of gallstone disease supports the effect of environmental factors (diet?) as a cause of the increased familial frequency of gallstones (**Heaton, 1973, Kalos et al., 1977**). These may however act on genetically susceptible subjects. Also familial dietary habits could be a factor. However, in view of the negative findings in spouses as found by **VanderLinden and Westlin (1966)** and by **Friedman et al. (1966)**, this is very questionable. Decades of eating similar food by both husband and wife should show some effect unless the critical period is in childhood.

In studying the role of diseases as a risk factor predisposing to gall stone development, the present study revealed that (40%) of the whole studied cases, (32.2%) of female studied cases, and (92.3%) of male studied cases have a positive history of Bilharziasis. The higher

lithogenic with respective inspection could reveal any stone in the biliary tract in their 19 patients studied. This suggested that this could be due to the fact that these patients do not live long enough actually to develop gall stones.

El Hadary (1988) proved that presence of lithogenic bile in patients with bilharzial hepatic fibrosis but the stated that whether the liver of those bilharzial patients secrete a lithogenic bile or the bile is rendered lithogenic due to changes in the gall bladder cannot be determined from his study as hepatic bile was not investigated.

The association between diabetes mellitus and gall stone disease has been debated in the literature for several years where as autopsy studies have found diabetics to be at increased risk for disease, epidemiologic studies have not established these findings **(Strom and West, 1985).**

In the present study, inspite that the percentage of diabetes mellitus in cases and controls was not significant, yet a significant difference in the mean fasting blood sugar between cases and controls was observed. This agrees with several studies that recorded a

positive correlation between fasting blood sugar and cholesterol (Franzblan and Criqui, 1984, Barret-Connor et al., 1980).

Inspite of this controversy, diabetic patients are thought to have a two fold to three fold increased risk of cholesterol gall stones (Stone et al., 1988). This increased risk of gall stones in diabetics occurs regardless of whether they are categorised as type I (insulin dependent) or type 2 (insulin independent) diabetics (Stone and Thiel, 1985).

Diabetics tend to be obese and to have hyper triglyceridemia. Both of these disorders are associated with increased risk of gall stones and increased cholesterol saturation of bile (Shaffer et al., 1972 and Bennion and Grundy, 1975).

Impaired gall bladder emptying due to diabetic neurogenic bladder has been thought as well to be one of the important factors in the increased incidence of gall stones in diabetics (Stone et al., 1988).

On studying laboratory finding among studied cases and controls no specific relation can be proved in the present study between occurrence of gall stone, and levels of haemoglobin liver functions tests or blood groups.

In conclusion, it was found in the present study that females outnumbered greatly males and that their mean age was higher than that of males. It was also found that obesity that constituted a significant risk factor for gallstone formation in females was not a risk factor in males

The possible risk factor for gallstone disease formation observed in this study were the female sex, a positive family history bad dietary habits, obesity and liver cirrhosis however a link could not be established between hormonal factors in the female patients and gallstone disease. In the same time snacks between meals, diet regimen, history of systemic disease and laboratory tests showed no difference between cases and controls and were not considered risk factors for the formation of gallstones.