RESULTS

RESULTS

Table (1): Sex distribution of the studied group.

died group	No. 24	60%	No.	60%	No.	60%
			12	60%	36	60%
		1				1
	16	40%	8	40%	24	40%
	40	100.0	20	100.0	60	100.0
		16		10 4070	16 40%	16 40% 8 4070

Table (1) shows:

DLC [n = 40 (males: 24 - females 16)]

Controls [n = 20 (males: 12 - females 8)].

Table (2): Age distribution of the studied group.

Studied g. Age (years)	roup 		DLC	Co	Controls Total		
< 45 years		No.	%	No.	%	No.	%
vio years		17	42.5	7	35.0	24	40.0
45 years		13	32.5	7	35.0	20	33.3
> 45 years		10	25.0	6	30.0	16	26.7
otai -		40	100.0	20	100.0	60	100.0

$$X^2 = 0.338$$

P > 0.05

Table (2) shows insignificant difference in age among DLC patients and controls.

Table (3): Comparison between the studied group regarding age.

		=	Ras	nge
Age (years) Studied	X (mean)	± SD	Minimum	Maximum
group DLC	47.2	± 13.4	23.0	68.0
Controls	50.9	± 11.9	32.0	70.0
t P	1.0 > 0			

Table (3) shows mean age distribution among the studied group.

DLC = 47.2 ± 13.4

Controls = 50.9 ± 11.9

Table (4): Blood picture of the studied group.

Studied group Blood	DLC	Controls		
picture	X±SD	X±SD	t	P
HB%	11.13 ± 2.11	13.15 ± 1.28	3.928	< 0.05 significant
ESR	20.8 ± 14.6	7.9±3.19	3.889	< 0.05 significant

Table (4) showing (mean Hb levels in DLC were 11.13 ± 2.11 and in controls 13.15 ± 1.28 gm%).

(ESR levels in DLC were 20.8 ± 14.6 and in controls 7.9 ± 3.19) and so it shows a significant difference between the two groups.

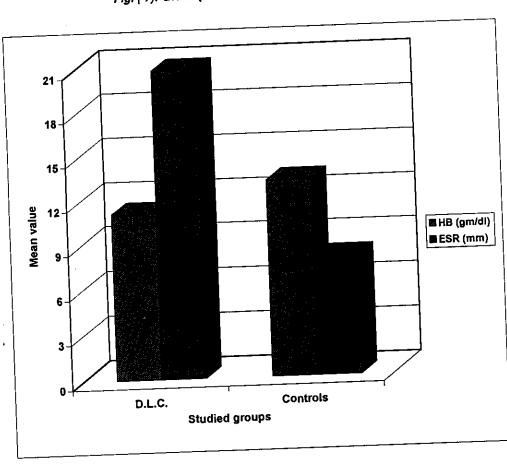


Fig. (1): Blood picture among the studied groups.

Table (5): Liver function test of the studied group.

Studied group	DLC	Controls		
function	X ± SD	X±SD	t	P
SGOT	36.77 ± 13.57	15.6 ± 2.99	6.867	< 0.01 (H.S.)
SGPT	46.2 ± 22.32	17.35±3.26	5.755	< 0.01 (H.S.)
S. Albumin	2.71 ± 0.49	3.92 ± 0.28	10.224	< 0.01 (H.S.)
rothrombin time	15.79 ± 2.14	12.84 ± 1.50	6.197	< 0.01 (H.S)

Table (5) shows a highly significant difference between DLC patients and control group.

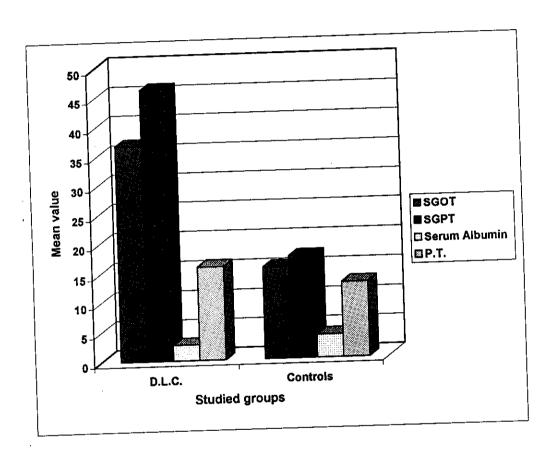


Fig. (2): Liver function tests among the studied groups.

Table (6): Kidney function test of the studied group.

Studied group Kidney function	DLC X ± SD	Controls X ± SD	t	P
Serum creatinine	1.50 ± 0.73	0.97 ± 0.28	3.099	< 0.05 (S)
Blood urea	26.47 ± 14.09	18.50 ± 3.33	2.486	< 0.05 (S)

Table (6) shows a significant disturbance in kidney function test in DLC patients in comparison to controls.

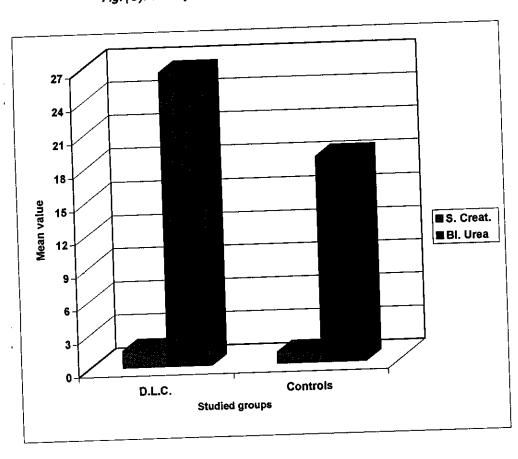


Fig. (3): Kideny function tests among the studied groups.

Table (7): Rapid urease test for detection of Helicobacter pylori in the studied group.

DUT	Studied group		DLC	Cor	Controls		
R.U.T.		No.	%	No.	%	No.	%
Positive		28	70.0	7	35	35	58.3
Negative		12	30.0	13	65.0	25	41.7
Total		40	100.0	20	100.0	60	100.0

$$X^2 = 6.720$$

P < 0.05

Table (7) shows that DLC patients had a significant positive results in comparison to controls (28 +ve tests).

• Table (8): Results of rapid urease test for detection of Helicobacter pylori infection in DLC with and without H.E.

Encep	nalopathy	Present	Absent	Total	
RUT					
+ve	No.	22	6	28	
	%	78.57	50	70	
-ve	No.	6	6	12	
	%	21.43	50	30	
Total		100.0	100.0	100.0	

$$X^2 = 5.215$$

P < 0.05

Table (8) shows that DLC patients with H.E. had a significant positive results (22 +ve tests).

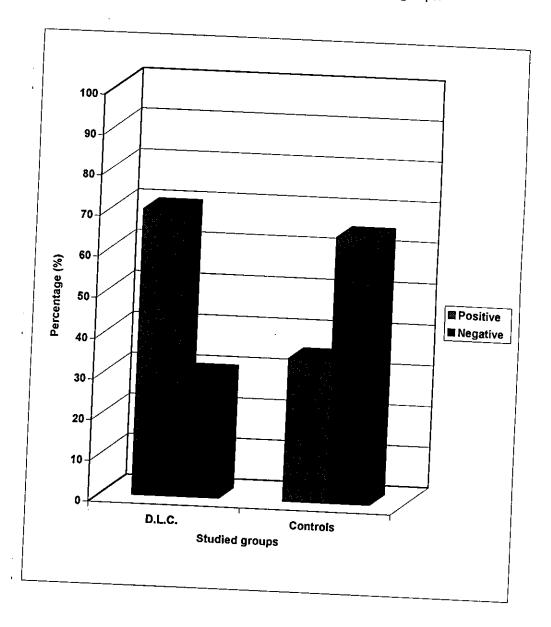


Fig. (4): Results of R.U. test among the studied groups.

100 90-80 70 Percentage (%) 60-■ Positive RUT 50 ■ Negative RUT 40 30-20-10 Absent Present Hepatic Encepahlopathy

Fig. (5): Results of R.U. test among patients with and without hepatic encephalopathy.

Table (9): Results of histopathological examination for detection of Helicobacter pylori infection of the studied group.

Studied Sex	group		DLC	Co	ntrols		Total
		No.	%	No.	1 %	No.	
-ve		9	22	10	50.0	19	33.3
+ve / -ve		4	10.0	1	5.0	5	6.7
+ve		16	40.0	5	25.0	21	35.0
†+ve		9	22.5	3	15.0	12	20.0
++ve		2	5.0	1	5.0	3	5.0
Total		10	100.0	20	100.0	60	100.0

Table (9) shows positive histopathological changes concomitent with *H. pylori* infection more common in DLC.

Table (10): Results of histopathology for detection of Helicobacter pylori in DLC patients with and without H.E.

	Present Absent		Abse	nt	Total	
H.E.		%	No.	%	No.	%
Histopathology	No.		6	50.0	9	22.50
-ve	3	10.71				
+/-	3	10.71	1	8.33	4	10.00
+ve	11	39.29	5	41.67	16	40.00
++ve	9	32.14	0	00	9	22.50
	2	7.14	0	00	2	5.0
+++ve Total	28	100.0	12	100.0	40	100.

Table (10) shows a histopathologic changes of Helicobacter pylori infection more common in DLC patients with H.E.

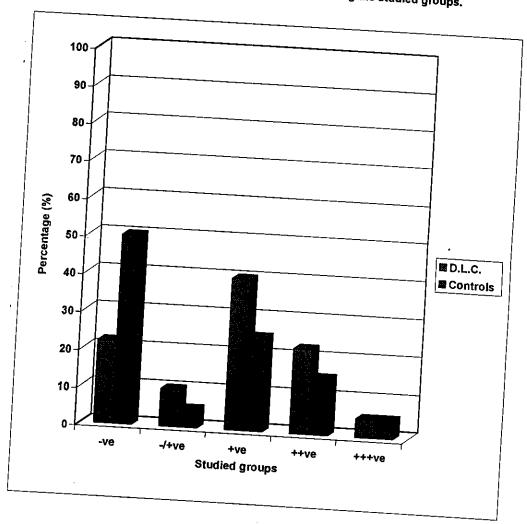


Fig. (6): Histopathological examination among the studied groups.

Fig. (7): Histpath. Examination among patients with and without hepatic Encephalopathy.

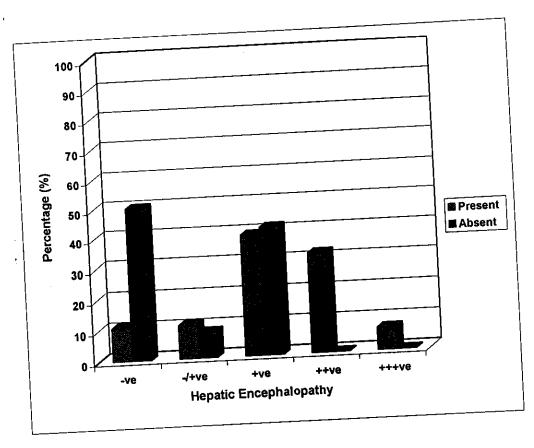


Table (11): Results of ELIZA in detection of Helicobacter pylori infection in the studied group.

Studied group ELIZA	 	DLC	C	ontrols		Total	-	
IgA	No.	%	No.	%	No.		X^2	P
+ve	36	90.0	13	65.0	49	81.7		
-ve Total	4	10.0	7	35.0	11	18.3	5.566	< 0.05
gG gG	40	100.0	20	100.0	60	100.0		(S)
+ve	33	82,5	11	55.0	44	73.3		
-ve	7	17.5	9	45.0	16	26.7	5.156	< 0.05
Total	40	100.0	20	100.0	60	100.0		(S)

Table (11) shows a significant higher immunoglobulin levels in DLC patients.

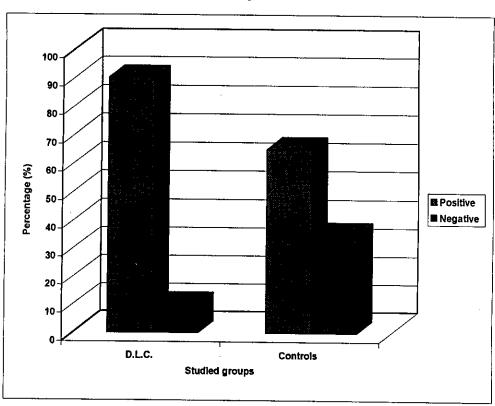
Table (12): Results of ELIZA in detection of Helicobacter pylori in DLC patients with and without H.E.

H.E.	Pre	sent	Abs	ent	To	tal	X²	P
ELIZA	No.	%	No.	%	No.	%	A	
IgA								
+ve	28	100. 0	8	66.67	36	90.0	10.37	< 0.01
-ve	0	00	4	33,33	4	10.0		(S)
Total	28	70.00	12	30.0	40	100.0		
IgG								
+ve	25	89.29	7	58.33	32	80.0	5.030	< 0.05
-ve	3	10.71	5	41.67	8	20.00		(S)
Total	28	70.00	12	30.00	40	100.0		

Table (12) shows that immunoglobulin levels were significantly higher in DLC patients with H.E.

Fig. (8): Results of ELIZA IgA and IgG among the studied groups.





lgG

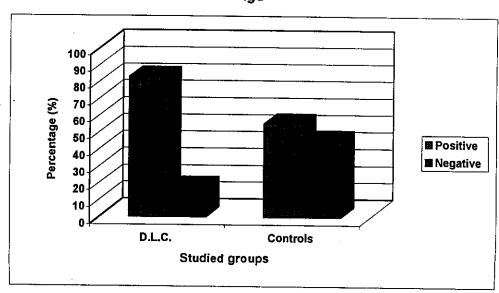
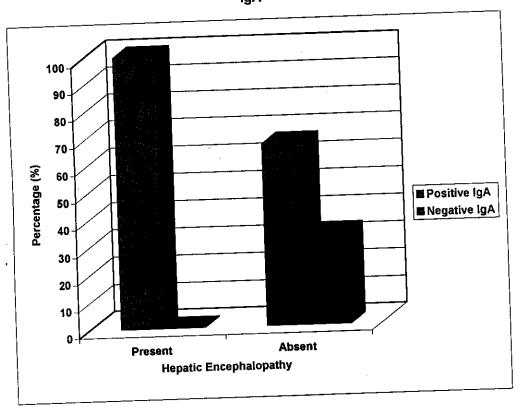


Fig. (9): Results of ELIZA among patients with and without Hepatic Encephalopathy.





lgG

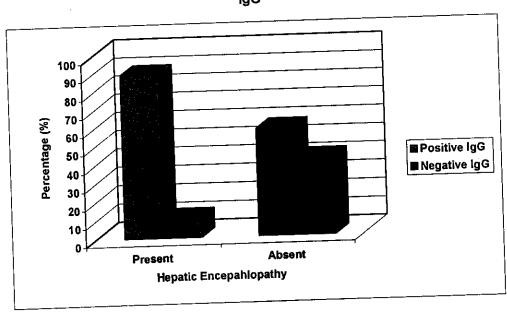


Table (13): Blood ammonia level of the studied group.

Blood ammonia level Studied groups	X (mean)	± SD	t	P
DLC	84.86	±34.61		
			7.403	< 0.01
Controls	26.05	± 10.48		

Table (13) shows significant high blood ammonia level in DLC patients (84.86 \pm 34.61).

Table (14): Blood ammonia level in patients with and without H.E.

Blood ammonia level H.E.	X (mean)	± SD	t	P
Present	93.8	± 34.5	2.681	< 0.05
Absent	64.0	± 25.4		

Table (14) shows higher level of blood ammonia in patients with H.E.

90 80-70-60-90 80-70-30 20 10-D.L.C. Controls Studied groups

Fig. (10): Blood ammonia level among the studied groups.

100-90-80-70-60-50-40-30-20-10-

Hepatic Encephalopathy

Absent

Fig. (11): Blood amonia level of patients with and without Hepatic Encephalopathy.

Present

Table (15): Gastric ammonia level of the studied group.

Gastric ammonia level Studied group	X mean	±SD	t	P
DLC	3.42	± 1.91	7.397	< 0.01
Controls	0.21	± 0.15	,.371	. 0.01

Table (15) shows a significant rise of gastric ammonia level in patients with DLC in comparison to control group.

Table (16): Gastric ammonia level in patients with and without HE.

Gastric ammonia level	X mean	±SD	t	P
Present	3.82	± 1.91		İ
			2.088	< 0.05
Absent	2.50	± 1.63		

Table (16) shows a significant high levels of gastric ammonia in patients with H.E.

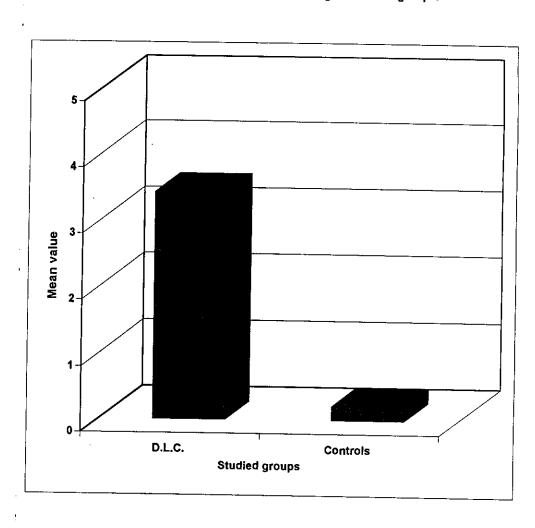


Fig. (12): Gastric ammonia level among the studied groups.

Present Absent Hepatic Encephalopathy

Fig. (13): Gastric amonia level in patients with & without Hepatic Encepahlopathy.