RESULTS

The present study was conducted on 40 patients with esophageal varices either with history of bleeding or activly bleeding varices admitted to pediatric endoscopy unit. Liver Institute, Menoufiya University from May, 1996 to January, 1998.

Patients included in the study werer divided into two groups randomly according to the method of varices treatment:

Group I: Endoscopic variceal Sclerotherapy (EVS).

Group I: Endoscopic variceal Ligation (EVL).

Analysis of the results showed that regarding age; group I ranged from 0.6-13 years with a mean of 6.5 years, group II ranged from 3-14 years with a mean of 7.2 years which was statistically insignificant (Table 1).

Regarding the sex; there was a girl preponderance 55% in group I, while in group II there was a boy preponderance 60%, but this was statistically insignificant (**Table 2**).

Regarding the presenting symptoms, hematemesis and melena were the commonest; 50 % in group I and 55% in group II. Hematemesis alone occured in 35% in group I and 30% in group II, while melena alone occured in 15% in each group (**Table 3**).

Regarding number of bleeding episodes; the majority of cases were in their first bleeding episode, 85% in group I and 90% in group II. Only 5 cases (15% in group I and 10% in group II) were showed in the second episode (**Table 4**).

Among 5 cases with second attach, 4 cases received no specific therapy (endoscopic or even tamponade), 3 of them had severe attaches were hospitalized and received blood transfusion and one with mild attach received no treatment. Lastly, one patient gave history of endoscopic injection seclerotherapy for previous attack but more than six months ago.

Regarding physical findings in the patients; clinically normal liver cellular status indentified by the absence of jaundice, ascites, edema of the Lower Limbs, encephalopathy and bleeding tendency was recorded in 75% in group I compared to 80 % in group II. The remaining patients had decompensated cellular function; 25% in group I and 20% in group II, but this was insignificant statistically as shown in (**Table 5**).

As regard the local abdominal examination; normal liver size occured in 65% in group I and 60% in group II. Shrunken cirrhotic liver was found in 25% of cases in group I and 30% in group II and lastly hepatomegaly was found in 10% in each group (**Table 6**). The liver, when felt, was firm in consistency, not tender or pulsating with sharp border and smooth surface.

Table (7) compared both groups as regard the size of the spleen. All patients showed splenomegaly with an average size of the spleen 4.1 fingers in group I and 3.7 fingers in group II, but this was statistically insignificant. As for the liver, in all patients where the spleen could be felt, it was firm with smooth surface and not tender.

Concerning the laboratory investigations they revealed no statistical significance between both groups. As for the complete blood picture; the mean hemoglobin level in grams was 6.8 in group I and 6.6 in group II. The white cell count was 8.3 thousands in group I and 6.6 in group II. The

platelet count was 213 thousands in group I and 168 in group II, all these data were statistically insignificant (**Table 8**).

As for the Live function tests, there was no statistically significante difference between both groups regarding total and direct bilirubin, aminotransferases, alkaline phosphatase, prothrombin time and concentration and the A/G ratio as was shown in (**Table 9**).

In patients with shrunken liver (11 patients), the splenic size was maximum and the incidence of cellular decompensation and enzymatic changes were more than other groups [(those with normal liver size (25 patients) and those with hepatomegaly (4 patients)]. The difference as regard liver enzymes, total bilirubin and the splenic size was significant statistically (**Table 10**).

Patients were classified according to their Child's classification that includes measurement of the serum albumin, bilirubin, prothrombin time as well as clinical encephalopathy and ascites, into 3 grades: A, B, C with the majority of cases falling in group A with better condition (75% in group I and 80% in group II). Grade B was noted in 15% in group I and 10% in group II and only 2 patients (10%) were grade C in each group, but this was insignificant statistically (**Table 11**).

Regarding abdominal ultrasonography which was done for all patients as shown in (**Table 12**), portal vein thrombosis stands out as the most important cause of bleeding and was found in 50% in group I and 55% in group II. Portal vein cavernoma was found in 15% and 5% for group I and II respectively.

Cirrhosis diagnosed by the presence of extensive fibrosis and nodularity was shown in 25% in group I and 30% in group II. Intrahepatic cholestasis was encountered in only one case (5%) in group I. One patient (5%) in group I and two patients (10%) in group II with hepatomegaly and renal enlargement were diagnosed as congential hepatic fibrosis and confirmed by liver biopsy (**Table 12**).

Liver biopsy was done in only 33 cases of total 40 (82.5%). The remaining 7 cases either refused to do the biopsy, (4 cases of prehepatic group, 2 lost during follow up and another 2 cases were continued follow up but still refuse to do the biopsy) or biopsy was contraindicated due to bleeding tendency of coagulation defect or thrombocytopenia (3 cases of hepatic group, one lost during follow up and another 2 cases were died).

Normal liver biopsy was found in 45% in group I and 35% in group II. Fatty degeneration was found in 10% in group I and 5% in group II and hydropic degeneration in one case (5%) in each group. All these cases were found to have P.V.T, P.V.C by abdominal ultrasonography (Table 13-14).

Chronic active hepatitis and cirrhosis was found in 10% in group I and 15% in group II. Micronodular cirrhosis was found in 5% in group I only. Congenital hepatic fibrosis was found in 5% in group I and 10% in group II. Bilharizial hepatic fibrosis and cirrhosis on top was found in 5% in group I and 10% in group I and 10% in group II (Table 13).

Upper endoscopic examination revealed that the majority of cases were grade III and IV varices (50% and 45% respectively) in group I and (55% and 40%) in group II. Esophageal varices grade II were only

encountered in 5% in each group, but this difference was insignificant (Table 15).

Reagarding the causes of portal hypertension, we found the majority of patients in both groups were secondary to prehepatic causes (65% in group I and 60% in group II) (**Table 16**).

Table (1): Comparison between the studied groups regarding age.

Studied groups	Group I*	Group II*
Age (ys)	(n = 20)	(n = 20)
Range Mean ± SD	$0.5 - 13$ 6.5 ± 2.8	3 - 14 7.2 ± 2.5
t	0.79	94
P	> 0.05 (N.S.*)

* Group I: Injection Sclerotherapy (EVS)

* Group I : Banding (EVL) * N.S. : Not Significant

Table (2): Sex distribution of the studied groups.

Studied groups	Group	I(n = 20)	Group II	Group II $(n = 20)$		
Sex	No.	%	No.	%		
Boys	9	45	12	60		
Girls	11	55	8	40		
Adjusted X ²	0.902					
Р		> 0.	05 (N.S.)			

Table (3): The presenting symptoms among the studied groups.

Studied groups	Group I ((n = 20)	Group I	I(n = 20)	
Presenting Symptoms	No.	%	No.	%	
Hematemesis	7	35	6	30	
Melena	3	15	3	15	
Hematemesis and melena	10	50	11	55	
Adjusted X ²	0.125				
P		> 0	.05 (N.S.)		

Table (4): Comparison between both groups as regard number of bleeding episodes before treatment.

Studied groups	Group I	(n=20)	Group	II (n = 20)	
No. of bleeding episodes	No.	%	No.	%	
1st episode	17	85	18	90	
2nd episode	3	15	2	10	
Adjusted X ²	0.229				
P		> 0.	05 (N.S.)		

Table (5): Cellular decompensation in both groups by clinical parameters.

Studied groups	Group I	(n=20)	Group II (n = 20)		
Clinical parameters	No.	%	No.	%	
Present	5	25	4	20	
Absent	15	75	16	80	
Adjusted X ²	0.143				
P		> 0	.05 (N.S.)		

Table (6): Comparison between both groups as regard the Liver size.

Studied groups	Group I	(n = 20)	Group I	I (n = 20)	
Liver size	No.	%	No.	%	
Normal	13	65	12	60	
Shrunken Liver	5	25	6	30	
Hepatomegly	2	10	2	10	
Adjusted X ²	0.131				
P		> 0	.05 (N.S.)		

Table (7): Comparison between both groups as regard the size of Spleen.

Studied groups	Group I*	Group II*		
Size of Spleen	(n = 20)	(n = 20)		
Range	1 - 7 fingers	1 - 7 fingers		
Mean ± SD	4.1 ± 1.2 3.7 ± 1.1			
t	1.099			
P	> 0.0	5 (N.S.)		

Table (8): Complete blood picture in both groups.

Studied groups	Group I	Group II	t	P
Complete blood picture	(n = 20)	(n = 20)		
Hemoglobin (grams) Range Mean ± SD	3.4 - 10.1 6.8 ± 1.8	3.6 - 11 6.6 ± 2	0.333	> 0.05 (N.S.)
W.B.Cs (thousands) Range Mean ± SD	3.1 - 15.3 8.3 ± 4.6	2.4 - 13.6 6.6 ± 2.8	1.652	> 0.05 (N.S.)
Platelet count (thousands) Range Mean ± SD	45 - 463 213.1 ± 101.2	66 - 335 174.52 ±71.683	1.705	> 0.05 (N.S.)

Table (9): Liver function tests in both groups.

Studied groups	Group I	Group II	t	Р
Liver	(n = 20)	(n = 20)		
function tests				
Total Bilirubin (mg/dL)		:		
Range	0.3-7.2	0.2-6.3		> 0.05
Mean ± SD	1.7±1.5	1.5±1.4	0.500	(N.S.)
Direct Bilirubin (mg/dL)				
Range	0.1-5.4	0.1-3.7		> 0.05
Mean ± SD	0.6±0.5	0.5±0.5	0.094	(N.S.)
ALT (IU/litre)				
Range	30-525	35-490		> 0.05
Mean ± SD	110±97.6	106.1±97.5	0.129	(N.S.)
AST (IU/litre)				
Range	40-468	40-480		> 0.05
Mean ± SD	98.7±69.5	101.0±80.9	0.094	(N.S.)
ALP (IU/litre)				
Range	70-545	60-720		> 0.05
Mean ± SD	265.0±73.7	288.2±96.2	1.360	(N.S.)
Prothrombin conc.				
Range	30-100%	44-100%		> 0.05
Mean ± SD	73.4±23	81.6±17.7	1.264	(N.S.)
A/G ratio				
Range	0.6-1.7	0.6-1.8		> 0.05
Mean ± SD	1.3±0.3	1.4±0.3	1.053	(N.S.)

Table (10): Clinical and Laboratory data in the studied groups.

	Clinical Data	ata				Laborat	Laboratory Data	
Liver size	Splenic size	Joindice Ascites Enceph.	Ascites	Enceph.*	T.B	ALT	AST	ALP
Normal (25)	3.5 ± 0.5	1	1	ì	0.9 ± 0.1	65.6 ± 9.4	65.2 ± 9.7	218 ± 23.3
Shrunken (11)	5.5 ± 0.9	7	4	2	3.4 ± 1.7	220.8±129	190.4±94.4 329±127.6	329±127.6
Hepatomeyloly (4)	1.8 ± 1.0	-			1.4 ± 1.1	63.7±9.5	67.5±8.7	195±34.9
ᅜ	54.984				27.178	21.098	25.112	10.662
Ь	<0.01 (H.S.)*	·			< 0.01 (H.S.)*	< 0.01 (H.S.)*	<0.01 (H.S.)*	<0.05 (S.)

* Enceph. * H.S.

: Encephaopathy : Highly Significant

Table (11): Child's grade in both groups.

Impic ()						
Studied groups	Group	I(n = 20)	Group II	(n = 20)		
Child's grade	No.	%	No.	%		
	15	75	16	80		
A	3	15	2	10		
В	2	10	2	10		
Adjusted X ²	0.232					
P		> 0.0)5 (N.S.)			

Table (12): Abdominal ultrasonographic findings of the two groups of studied cases.

	8					
Studied groups	Grou (n =			up II = 20)	Z	P
Abdominal	No.	%	No.	%		
ultrasonography P.V.T.	10	50	11	55	0.317	> 0.05 (N.S.)
P.V.C.	3	15	1	5	1.054	> 0.05 (N.S.)
Cirrhosis	5	25	6	30	0.354	> 0.05 (N.S.)
Cholestasis	1	5	0	0	1.013	> 0.05 (N.S.)
Liver fibrosis	1	5	2	10	0.600	> 0.05 (N.S.)

P.V.T: Portal Vein Thrombosis

P.V.C: Portal Vein Cavernoma

Table (13): Liver biopsy findings of the two groups of studied cases.

Studied groups	Grou (n =	- 1	Grou (n =		z	P
Liver biopsy	No.	%	No.	%		0.05
Normal	9	45	7	35	0.645	> 0.05 (N.S.)
Fatty changes	2	10	1	5	0.600	> 0.05 (N.S.)
Hydropic changes	1	5	1	5	<u>-</u>	- L
CAH +¢	2	10	3	15	0.478	> 0.05 (N.S.)
Micronodular ¢	1	5	0	0	1.013	> 0.05 (N.S.)
C.H.F	1	5	2	10	0.600	> 0.05 (N.S.)
Bilharzial fibrosis + ¢	1	5	1 2	10	0.600	> 0.05 (N.S.)
Not done	3	15	4	20	0.416	> 0.05 (N.S.)

CAH : Chronic active hepatitis

¢ : Cirrhosis

C.H.F.: Congenital hepatic fibrosis

Table (14): Liver biopsy Vs abdominal ultrasonography

				I iver h	Liver hionsy			
Abdominal						1	OUF	Not
	Mormol	W + HVU	Fattv	Hydropic	Mirco.	Bilharziai	C.II.F.	
ultrasonograpny Normal	Norman	٠ ١٠٥٥	fam.			fibrosis + ¢		done
								(r
PVT. On	15		2					,
(17)								
PVC.	1			_				
È : : : : :						7		^
Cirrhosis (11)		5 7			_	<u> </u>		1
(11)								
Cholestasis (1)								
							3	
HSM (3))	

P.V.T. : Portal vein thrombosis

P.V.C. : Portal vein cavernoma

HSM : Hepatosplenomegaly

: Cirrhosis

CAH : Chronic active hepatitis

Micro. ¢ : Micronodular cirrhosis

C.H.F. : Congenital hepatic fibrosis

Probably ulceration occured in all patients but healed before the second endoscopy session.

The only significant difference between both treatment techniques was the development of esophageal stricture that was related to the depth of the ulcers and happened in 17.6% compared to 0% in group I and II respectively, but responded rapidly to dilatation (**Table 26**).

All patients with esophageal stricture were complaining of variable grade of dysphagia and had endoscopic evidence of narrowing.

Recurrence of varices which is an endoscopic finding was noted to be more common in the EVL group (group II); 35.3% compared to 20% in the EVS group (group I), with no statistical difference. However, recurrence was more delayed in EVL group than the EVS group (4.5 months Vs 3.7 months), but also, with no statistically significant difference (Table 27-28).

All recurrent varices (grade II-III) underwent repeated sessions and were easily eradicated with further fewer sessions.

Regarding rebleeding that occured either before or after eradication, it was noted in 35.3% of group I and 22.2 in group II (Table 29).

The main causes of rebleeding among both groups were variceal rebleeding (66.66% in group I and 50% in group II), to be followed in group I by ulcer induced rebleeding and bleeding from fundal varices (16.67% of each) and in group II by ulcer induced rebleeding and bleeding from congestive gastropathy (25% of each) (**Table 30**).

Rebleeding were effectively treated either conservatively or by further sessions.

In **Table (31)** patients, classified according to the cause of portal hypertension, were compared as regard the outcome of the treatment. It was shown that there was no statistical difference between both groups in any of the studied parameter except for the survival which is more in the prehepatic patients as all mortalities were secondary to severe liver cell failure.

This study also, compared variceal grades according to **Thakeb et al.**, (1987), against the outcome of endoscopic treatments and showed that survival and eradication were more in the smaller variceal grades II, III than grad IV with the reverse happening in all other parameters, but without statistical significance (**Table 32**).

Table (20): Eradication of the varices in the studied cases.

Studied groups	Group I (n = 17)		Group II $(n = 18)$		
Eradication	No.	%	No.	%	
Eradication	15	88.2	17	94.4	
Non eradication	2	11.8	1	5.6	
Adjusted X ²	0.410				
P	> 0.05 (N.S.)				

Table (21): Number of sessions required for reduction one grade of variceal size.

	Group I (n = 17)	Group II $(n = 18)$			
Mean ± SD	1.8 ± 1.6	1.2 ± 0.4			
t	1.	1.500			
P	> 0.05	> 0.05 (N.S.)			

Table (22): Number of sessions required to eradicate the varices.

Studied groups	Group I (n = 15)	Group II (n = 17)	
No. of sessions Range	2 - 7	2.7	
Mean ± SD	4.4 ± 1.2	3.8 ± 1.3	
t	1.342		
P	> 0.0	5 (N.S.)	

Table (23): Amount of sclerosant used to eradicate varices in group (1).

Mean	SD
48.4	13.9

Table (24): Number of bands used to eradicate varices in group (II).

Mean	SD
10	3.8

Table (25): Ulcerations due to endoscopic techniques.

Studied groups	Group I	(n = 17)	Group I	I (n = 18)
Ulceration	No.	%	No.	%
Yes	9	52.9	12	66.7
No	8	47.1	6	33.3
X ²	0.686			
P	> 0.05 (N.S.)			

Table (26): Stricture formation among studied cases.

Studied groups	Group I (n = 17	Group II (n = 18)		
Stricture formation	No.	%	No.	%	
Yes	3	17.6	0	0	
No	14	82.4	18	100	
Z	1.964				
Р	< 0.05 (S.)				

* S. : Significant

Table (27): Recurrence of the varices among 2 groups of studied patients after treatment.

Studied groups	Group I	(n=15)	Group II (n = 17)		
Recuurence of varices	No.	%	No.	%	
No recurrence	12	80	11	64.7	
Recurrent varices	3	20	6	35.3	
Adjusted X ²	0.922				
Р	> 0.05 (N.S.)				

Table (28): Onset of recurrence of varices in months among 2 groups of studied patients after treatment.

	Group I $(n = 3)$	Group II $(n = 6)$
Mean ± SD	3.7 ± 0.6 months	4.5 ± 1.1
t	1.4	13
P	> 0.05	(N.S.)

Table (29): Rebleeding among the studied cases.

Studied groups	Group I	(n = 17)	Group I	I(n=18)
Rebleeding	No.	%	No.	%
No	11	64.7	14	77.8
Yes	6	35.3	4	22.2
Adjusted X ²	0.732			
P	> 0.05 (N.S.)			

Table (30): Causes of rebleeding among the studied cases.

Studied groups	Gro (n =	-	Grou (n =	up II = 4)	Z	P
Causes of rebleeding	No.	%	No.	%		
Ulcer induced	1	16.67	1	25	0.323	> 0.05 (N.S.)
Variceal bleeding	4	66.66	2	50	0.527	> 0.05 (N.S.)
Fundal varices	1	16.67	0	0	0.861	> 0.05 (N.S.)
Congestive gastropathy	0	0	1	25	1.291	> 0.05 (N.S.)

Table (31): Comparison between prehepatic and hepatic groups as regard the outcome.

Variant	Prehe group	_	Hep	atic (n=15)	Z	Р
v arrant	No.	%	No.	%		
Follow up						
No	2	8	1	6.7	0.155	> 0.05
Yes	23	92	14	93.3		(N.S.)
Survival			·			
No	0	0	2	14.3	1.964	< 0.05
Yes	23	100	12	85.7		(S.)
Eradication						
No	1	4.3	2	16.7	1.236	> 0.05
Yes	22	95.7	10	83.3		(N.S.)
Ulceration		20.1	_	11.7		
No	9	39.1	5	41.7	0.145	> 0.05
Yes	14	60.9	7	58.3		(N.S.)
Rebleeding						
No	17	73.9	8	66.7	0.450	> 0.05
Yes	6	26.1	4	33.3		(N.S.)
Recurrence					,	
No	16	72.7	7	70	0.159	> 0.05
Yes	6	27.3	3	30		(N.S.)

Table (32): Outcome of cases according to the variceal grading.

Grade	Survival	ival	Eradic	Eradication	Ulcera	Ulcerations	Recur	Recurrence Rebleeding	Reble	eding	Stricture	ture
	No.	%	No.	%	No.	%	No. %	%	No.	%	No.	%
11 (2)	2	100	2	100	1	95	0	0	0	0	0	0
(61) III	18	94.7	17	94.4	6	20	4	23.5	4	22.2		5.6
IV (16)	15	93.8	13	86.7	11	11 73.33	5	5 38.5	9	40	2	13.3
Z	0.1	0.198	1.5	1.542	1.3	1.394	8.0	0.893	1.2	1.296	0.872	7.2
Ч	\ \ \ \ \ \ \	> 0.05 (N.S.)	0 ×	> 0.05 (N.S.)	0 <	> 0.05 (N.S.)	> 0.05 (N.S.)	.05 S.)	ر ا ا	> 0.05 (N.S.)	\ \ \ \ \	> 0.05 (N.S.)