

# EVALUATION OF PERITONEOVENOUS DENVER SHUNT IN THE MANAGEMENT OF INTRACTABLE NON-MALIGNANT HEPATIC ASCITES

By

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Intractable ascites, refractory to medical therapy, occurs in approximately 10% of patients with ascites from cirrhosis and is almost always associated with a grave prognosis. The role of peritoneovenous Denver shunt in control of nonmalignant hepatic refractory ascites was assessed by clinical, anthropometeric, Doppler ultrasound and biochemical means.

The study was performed on twenty patients with ascites not responding to 400 mg spironolactone and 80 mg furosemide daily, with no bleeding attacks in last three months, serum bilirubin less than 4 mg%, compensated heart as well as renal functions and normal serum amylase level. Ascitic fluid sample revealed total leucocytic count less than 250/ml, no growth in culture, protein content less than 4.5 gm% and negative cytology.

These patients underwent peritoneovenous shunt under local anesthesia. Post-operatively, they were assessed by abdominal girth, body weight, and fluid balance. Hemoglobin, packed cell volume, platelets count and coagulation profile. Serum electrolytes, liver functions tests, total proteins and serum albumin. All these parameters were measured daily for two weeks and weekly for two months and monthly through out the study. Hepatic and renal duplex Doppler ultrasonography was carried in all patients before and 4 weeks after shunt operation as well as in twenty healthy controls. These data was analyzed statistically using T-test to compare between the ascitic and control groups while Paired t-test to compare pre and postoperative data. Operative mortality included all deaths within 30 days of surgery.

Regression of the tense ascites, improvement of the quality of life, improvement of the milieu interior and nutritional status were achieved in all patients with no operative related mortality. The mean weight and abdominal girth decreased significantly after shunt insertion (p<0.001). Haemoconcentration, urinary output and pulse pressure were markedly improved. Significant laboratory alterations in coagulation parameters (p<0.001) consistent with DIC were present in virtually all patients not associated with clinically evident DIC. Hospital stay was short (7-10) days. The mean resistive index of renal artery showed statistically significant reduction as compared to the pre-operative value (0.78 ±0.32 vs. 0.64 ±0.14 respectively P<0.05).

Few complications were described but they did not influence the general results. Complications related to shunt insertion were easily prevented and properly managed. Shunt occlusion occurred in six patients (30%) (Peritoneal catheter occlusion 20% and pump chamber occlusion 10%). Gastrointestinal bleeding occurred in four patients (20%) (Gastric erosions 10% - variceal bleeding 10%). Minor complications were observed as improper positioning of either the venous catheter in one patient (left innominate vein) or the peritoneal catheter in two patients (the supracolic compartment or subphrenic space). Also one patient had a small subcutaneous fluid collection after shunt obstruction.

In conclusion, Insertion of the peritoneo-venous Denver shunt seemed to be a minor operative procedure done under local anesthesia with minimal surgical stress. It provided good palliation for all patients with little morbidity and no operative related mortality. It improved renal haemodynamic as indicated by reduction of resistive index of the renal artery. Preoperative injection sclerotherapy as well as proton pump inhibitor prevent post-operative gastro-intestinal bleeding. Peritoneo-venous Denver shunt in association with chest tube drainage and pleurodesis 3 weeks after shunt insertion succeeded in controlling recurrent pleural effusions secondary to liver cirrhosis. So proper patient selection and careful surgical procedure seems to be mandatory for better results.

Key words: PERITONEOVENOUS DENVER SHUNT- INTRACTABLE -HEPATIC ASCITES

## INTRODUCTION

Ascites is the most common complication of cirrhosis. Its development is associated with a grave prognosis; 50% of patients die within 2 years of diagnosis. Ascites results from chronic liver disease, inflammatory bowel syndrome, malignancy as well as lymphatic obstruction by lymphoma, nodal deposits from carcinoma or intestinal lymphangioectasia. An understanding of the analysis of ascitic fluid is essential for the appropriate management of patients with liver disease and ascites. The macroscopic appearance of the fluid often indicates the nature of underlying disease and is described as serous, chylous, pseudochylous, bloodstained or myxomatous<sup>1</sup>.

Patients with advanced liver disease, Budd-Chiari syndrome or peritoneal carcinomatosis become refractory to medical treatment, these patients cease to respond to diuretics therapy and develop pre-renal azotaemia. Furthermore, patients with ascites are at risk for ascitic fluid infections and neurohormonal dysregulation that can lead to hepatorenal syndrome. The ascites is gross and tense; the elevated intra-abdominal pressure causes venous congestion of the abdominal wall veins and diminishes venous return from lower limbs, which aggravates the peripheral edema. It also induces a tense uncomfortably heavy abdominal sensation, respiratory distress results from splinting of the diaphragm, the umbilicus becomes evereted and existing hernias become distended<sup>1& 2</sup>.

The management of patients with ascites involves a combination of dietary, medical, and surgical approaches; 10% of the patients die as a result of secondary morbidity associated with therapy <sup>2</sup>.

The medical management of intractable ascites (bed rest, diuretics, and fluid restriction) is impractical, ineffective and usually unrewarding as a long-term solution. It also leads to hypovolaemia, electrolyte imbalance, reduced renal blood flow and ultimately renal failure <sup>3</sup>.

Repeated large volume therapeutic paracentesis, the most often-employed initial treatment modality, may further compromise an already nutritionally depleted patient through the loss of protein and electrolytes. Also, repeated paracentesis is a source of patient discomfort, necessitates frequent hospital visits, and has been associated with a significant high rate of ascitic fluid infections and/or peritonitis <sup>4</sup>.

Although the portal-systemic shunt is efficient in clearing ascites, it does not improve the survival, which depends on liver function, and it is complicated by an important incidence of encephalopathy. Since the patients with refractory ascites and good hepatic risk are not usually many, it is possible to understand why derivative surgery has been disappointing with this indication <sup>5</sup>.

TIPS is an effective treatment for refractory ascites; it was reducing ascites and improving the functional renal impairment, however, it is a challenging procedure and has its serious complications (Portal vein thrombosis, peritoneal bleeding, acute renal failure, pneumothorax, hemoptysis, spontaneous bacterial peritonitis, and heart failure) <sup>6</sup>. TIPS creation was considered the cause of death in (10.4%). Primary patency was 23% at 1 year, but shunt obstruction was accessible for a second intervention. 21% had de novo encephalopathy after TIPS creation. A careful selection of patients is mandatory <sup>7</sup>

Early recognition of these complications allows therapeutic interventions that minimize further clinical deterioration in already chronically ill patients<sup>3</sup>.

#### Aim of work:

To assess the role of the peritoneo-venous Denver shunt in control of refractory ascites by clinical, anthropometeric, Doppler ultrasound and biochemical means as well as to study of the post-operative complications and their control.

## PATIENTS AND METHODS

This study was conducted on twenty patients with liver cirrhosis and non-malignant refractory hepatic ascites. They were selected according to these criteria:

• Failure of 400 mg spironolactone and 80 mg furosemide daily to controlascites.

• Development of encephalopathy or ureamia during diuretic therapy.

- No recent bleeding attack in the last three months.
- Serum bilirubin < 4mg%.
- Serum amylase within normal level.

• Good general condition esp. compensated heart as well as renal functions.

• Ascitic fluid sample for: total leucocytic count <250/ml culture: no growth and protein content <4.5 gm%.

Ascitic cytology revealed absence of malignant cells.

All patients underwent Denver peritoneovenous shunt (Denver Biomaterials, Inc.) under local anesthesia; patients were monitored throughout the procedure. A small supraclavicular incision made to expose the internal jugular vein. A further subcostal incision made to expose the transversus abdominus. Complete abdominal tapping of the ascitic fluid followed by half the volume replacement of saline solution. The shunt inserted through a subcutaneous tunnel between the internal jugular vein proximally and the peritoneum distally (figure 1a) 8.

Post-operatively, X-ray confirmed correct positioning of the peritoneal and venous catheter (figure 1b). The same dose of diuretic was administered for 7 days and tailed off gradually. The patients were taught to compress the shunt chamber three to four times per day.

## **POST-OPERATIVE ASSESSMENT:**

- Abdominal girth
- Body weight
- Fluid balance
- Hemoglobin and Packed cell volume
- Prothrombin time & conc.
- Partial tissue thromboplastin
- Platelets count
- Serum Fibrinogen level
- Serum Fibrin Degradation Products
- Serum total proteins and albumin.
- Serum electrolyte
- Liver functions tests
- Hepatic and renal duplex Doppler ultrasound

All these parameters were measured daily for two weeks and weekly for two months and monthly through out the study 9.

Hepatic and renal duplex Doppler ultrasound was carried preoperatively and 4 weeks post-operatively on all patients as well as 20 healthy controls. Both groups were studied after overnight fast and medication except diuretics had been stopped at least 4 days before study. Colorduplex ultrasound examination was performed with Hitachi 5000 scanner using 3.5 broadband convex probe. The portal blood mean velocity, portal blood flow and resistive index (RI) of both hepatic and renal artery were assessed<sup>10</sup>. Each result was the mean of five-repeated measurement. Doppler waveform was obtained at the lowest pulse repetition frequency possible without aliasing. This maximized the size of the Doppler spectrum and decreased the percentage of error of measurement.

These data was analyzed statistically using T-test to compare between the ascitic and control groups while Paired t-test to compare pre and postoperative data. Operative mortality included all deaths within 30 days of surgery.

## RESULTS

This study was carried on twenty patients with refractory ascites in THEODOR BILHARZ RESEARCH INSTITUTE Referred from the tropical medical department. They were seventeen males and three females. Their ages ranged between 42-58 years with a mean of 48 ±4.2 years. They were thirteen bleeders (with past history of haematemisis and/or melena) and seven non-bleeders. Eighteen patients had ascites secondary to liver cirrhosis and portal hypertension confirmed by abdominal ultrasonography while two patients had ascites secondary to veno-occlusive disease (Budd-Chiari syndrome) diagnosed by abdominal duplex. They were usually presented with anorexia, compromise of respiratory functions, and difficulty with ambulation. 10% of these patients had right plural effusion associated with their tense ascites (n=2) and 40% had hernia symptoms (n=8).

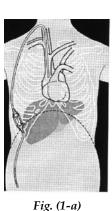
Regression of the tense ascites, subjective improvement of the quality of life, improvement of the milieu interior and nutritional status were achieved in all patients with no operative related mortality (figure 2).

The abdominal girth showed early decrease of 10% in the second post-operative day because of complete tapping & saline replacement done during surgical procedure. two weeks later, 20% decrease of the pre-operative abdominal girth was achieved denoting shunt function. After which equilibrium was achieved as the rate of ascitic formation equal ascitic drained through the shunt (figure 3).

Body weight revealed abrupt decrease in the second day post-operative as a result of abdominal tapping. Further decrease still present in the first two weeks denoting functioning of the shunt till reach to the equilibrium. Later on, the body weight started to increase gradually as nutritional improvement was noticed and not ascitic fluid reaccumulation (figure 4).

Pre-operative urine output (UOP) ranged between 550-800 cc urine/24 hours in spite of maximum dose of diuretic intake but shortly after introduction of the shunt marked increase of urine output 2500-3000 cc with the same dose of diuretics, the high UOP persisted after decrease of the dose of diuretics 2 weeks later till reach the state of equilibrium after 2 weeks when the UOP reached a range between 1500-2000 cc (figure 5).

As regards Doppler ultrasound, portal blood mean velocity as well as portal blood flow was statistically



significantly decreased in cirrhotic patients with refractory ascites than controls. Moreover the resistive index of both hepatic artery and renal artery were statistically significantly increased in the former group as compared to healthy controls (table 1). After 4 weeks post-operatively, only the resistive index of the renal artery showed a significant decrease when compared with the pre-operative index (P<0.05) (table 1).

Serum albumin showed early post-operative dilutional decrease followed by gradual slowly increase allover the time of study without intra-operative or post-operative exogenous albumin infusion (figure 6).

Prothrombin concentration as well as partial tissue thromboplastin did not show significant changes allover the study while the decrease of serum fibrinogen and concomitant increase of serum FDPs post-operatively revealed a state of DIC on laboratory ground and not in clinical ground (figure 7). Significant laboratory alterations in coagulation parameters consistent with DIC were present in virtually all patients with functioning shunt. These were evidenced by a statistically significant decline in platelet count and serum fibrinogen level (p<0.01). Furthermore, all patients with patent shunt demonstrated a marked increase in the circulating level of FDP (P<0.001). These alterations of coagulation parameters were not associated with clinically evident DIC.

Also, all hernias either umbilical or inguinal were deflated markedly from ascitic fluid and were easily repaired later on (figure 8). Peritonea-venous Denver shunt in association with chest tube drainage and pleurodesis 3 weeks after shunt insertion succeeded in controlling recurrent pleural effusions secondary to liver cirrhosis in two patients which was unsuccessfully treated medically, or by repeated thoracentesis. Chest tube decreased the intra-thoracic pressure and improved the functions of the Denver shunt (figure 9).

Complications related to shunt insertion were summarized in table (2). The most common complication was shunt occlusion in six patients (30%) (peritoneal catheter occlusion 20% and pump chamber occlusion 10%). It was easily prevented with regular pumping of the shunt and also easily treated with shunt revision (restoration of the flow through the shunt by removal of the obstructing agent). In the early phase of this series, Gastrointestinal bleeding occurred in four patients 20% (gastric erosions 10% - variceal bleeding 10%). these four patients were among the first seven patients but in the remaining thirteen patients pre-operative injection sclerotherapy as well as proton pump inhibitor prevent this complication.

Low grade febrile episodes (37.5-38.2 C), of short duration (less than 4 days) and required no treatment, were noted in all patients with functioning shunt in the early post-operative period. Neither shunt-related infectious complications, heart failure, arrhythmia or fluid overload occurred.

Minor complications were observed as improper positioning of either the venous catheter (left innominate vein) in one patient (figure 10) or the peritoneal catheter (the supracolic compartment or subphrenic space) in two patients. Readjustment of the proper position of catheter was done easily under fluoroscopy. Also one patient had a small subcutaneous fluid collection after shunt obstruction around the subcostal incision in the standing position (figure 11) shifted to the cervical incision when patient lying down (figure 12). It almost occurs in association with shunt occlusion and it is treated by shunt revision

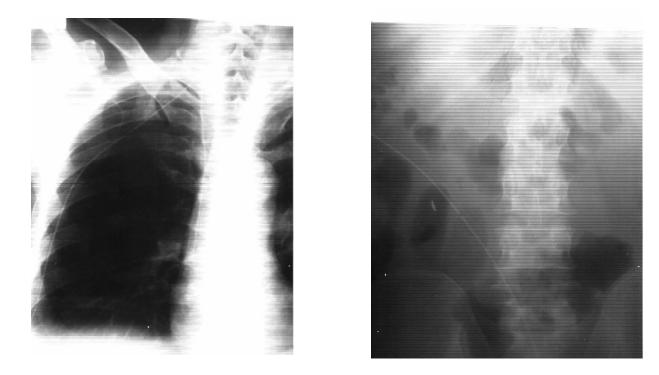
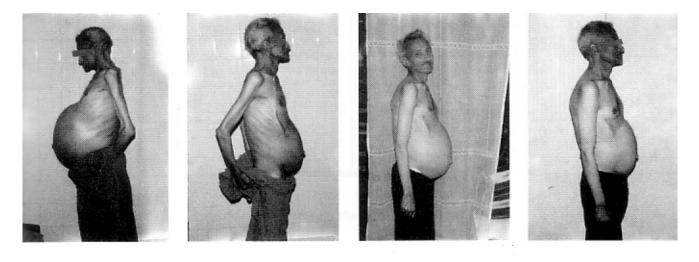


Figure (1-b): the exact position of the venous and peritoneal catheters of the peritoneo venous shunt.



Pre-op

2 weeks

2 months

6 months

Figure (2): The effect of peritoneo venous shunt on intractable ascites at different intervals

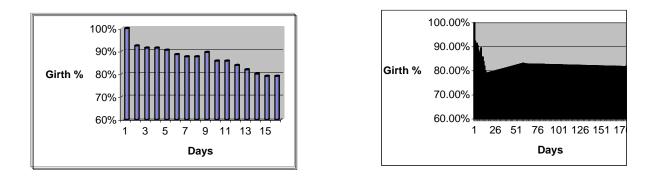
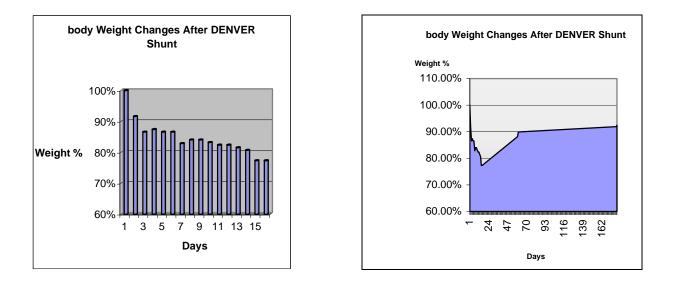
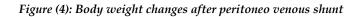
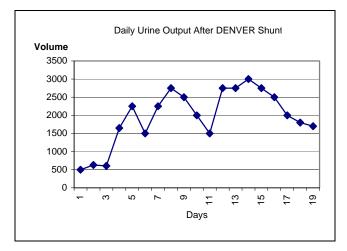
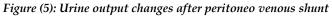


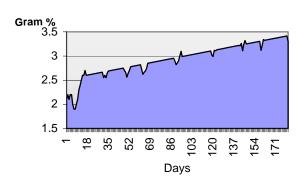
Figure (3): Abdominal girth changes after peritoneo venous shunt





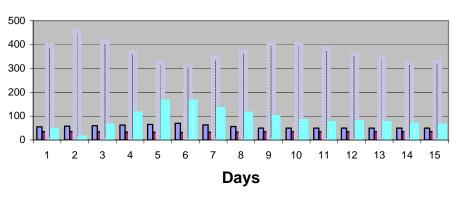






Serum Albumin Changes After DENVER Shunt

Figure (6): Serum albumin changes after peritoneo venous Denver shunt



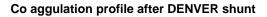


Figure (7): Coaggulation parameters after peritoneo venous Denver shunt

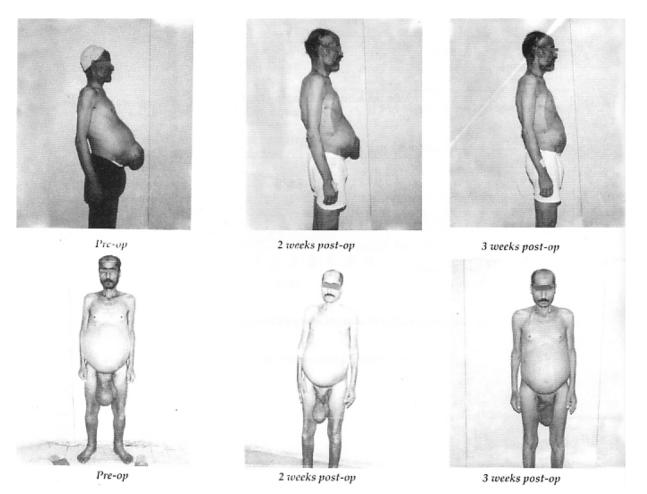
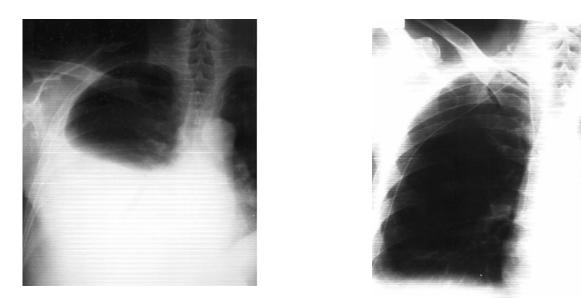


Figure (8): inguinal and umbilical hernias deflated after control of ascites

PROTH.C PTT FIBRINOGEN FDPs



Pre-op.4 weeks post-operativeFigure (9): Right pleural effusion well controlled after control of ascites with peritoneovenous shunt



Fig. (10) showed the venous catheter in the lefty innominate vein

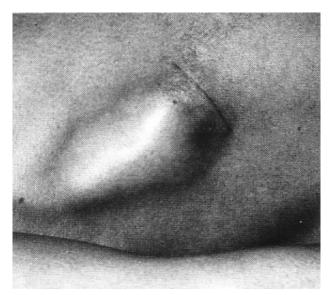


Fig. (11) subcutaneous fluid collection of the subcostal incision

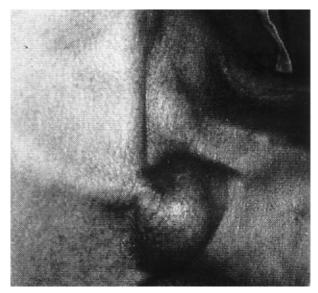


Fig. (12) subcutaneous fluid collection of the cervical incision

Table (1): Hepatic and renal Doppler measurements in controls and cirrhotic patients before and 4 weeks Following
Peritoneovenous Denver Shunt

	Control	Cirrhotic before surgery	P value	Cirrhotic 4 weeks after surgery	P value
Hepatic artery RI	0.57 ±0.21	0.74 ±0.43	0.002	0.69.3 ±0.12	0.11
Portal blood mean velocity (cm/sec)	19.2 <b>±</b> 4.2	9.5 <b>±</b> 3.1	0.001	10.2 <b>±</b> 2.1	0.21
Portal blood flow (ml/min)	1257 <b>±</b> 357	865 <b>±</b> 325	0.002	895 <b>±</b> 415	0.24
Renal artery RI	0.54 ±0.24	0.78 ±0.32	0.001	0.64 ±0.14	0.05

#### Table (2): Complications Following Peritoneovenous Denver Shunt

Complication	Number of cases		Percentage	
Shunt occlusion	6		30%	
Peritoneal catheter occlusion		4	20%	
Pump chamber occlusion		2	10%	
Gastrointestinal bleeding	4		20%	
Gastric erosions		2	10%	
Variceal bleeding		2	10%	

## DISCUSSION

Medically refractory ascites is a clinical entity for which exists few effective therapeutic options. Available treatment modalities include diuresis and sodium restriction, large volume paracentesis, surgical shunts as Porto-systemic shunts, Lympho-venous shunt or Peritoneo-venous shunts (Sapheno-peritoneal shunt, Le veen shunt and Denver shunt), transjugular intrahepatic portosystemic shunts, and liver transplant. The absence of a single, effective therapy in the management of refractory ascites speaks to the complex nature of this complication. Although most patients will respond to medical management, thoughtful application of available therapeutic options in patients who fail not only makes decisions regarding their care easier but also provides the best palliation in a vexing clinical scenario<sup>11</sup>.

Peritoneo-venous shunting has been cited in the literature as providing several physiologic benefits: Increases effective blood volume and renal blood flow, retains nutrients and improves nutritional status, increases diuresis, improves mobility and respiration, relieves massive or refractory ascites, reduces the risk of infection and reduces hospitalization. In cirrhotic, it specifically increases natriuretic response to diuretics, reduces activity of the rennin and angiotensin system and reduces sodium retention <sup>12</sup>.

Insertion of the peritoneo-venous Denver shunt seemed in this study and others<sup>13</sup> - to be a minor operative procedure done under local anesthesia with minimal surgical stress.

It provided good palliation for all patients as regression of the tense ascites, improvement of the quality of life, improvement of the milieu interior and nutritional status were achieved in all patients with little morbidity and no operative related mortality in agreement of Utikal et al <sup>14</sup>.

Saphenous-peritoneal shunt appears a simple, safe, and effective method of achieving long-term control of refractory ascites. The use of a biological shunt is an added advantage over prosthetic shunts for drainage of ascitic fluid but the procedure was performed under general anesthesia. Thirty-day mortality was 10%. Morbidity included transient hepatic encephalopathy in 36%, minor wound hemorrhage in 27%, fluid leakage in 64%, and wound infection in 64%. Hospital stay was16 days (range 11 to 23)<sup>15</sup>.

Compared with the Saphenous-peritoneal shunt, hospital stay in the present study was significantly shorter (7-10) days, The mean weight and abdominal girth decreased significantly after shunt insertion (p<0.001), Haematocrit value decreased to <40% significantly earlier, haemoconcentration, urinary output and pulse pressure were markedly improved, Discomfort due to massive ascites diminished promptly and did not recur in all patients, The serum concentration of protein was maintained, The mean values of several parameters in the coagulation-fibrinolysis system did not change significantly<sup>15&16</sup>.

Malnutrition is common in patients with decompensated cirrhosis and refractory ascites. Nutritional state was markedly improved in our patient after peritoneovenous shunt guided by the increase of the body weight not associated with ascitic fluid reaccumulation and increased serum proteins partly from improved appetite and partly from absence of protein losses in repeated therapeutic paracentesis. Post-operative photographs 2 and 6 months after implantation of the shunt in figure (2) verify the nutritional improvement. Other studies experienced this improvement after TIPS placement <sup>17</sup>.

In the current prospective study, Doppler ultrasound showed that in cirrhotic patients with refractory ascites, resistance to hepatic and renal arterial blood flows were increased in agreement with other studies <sup>18,19,20&21</sup>. The increase of arterial resistances can be caused by local factors such as liver fibrosis and regenerative nodules or release of local vasoconstrictors <sup>22</sup>.

The resistive index of hepatic artery correlated directly with that of the renal artery, suggesting that in addition to local factors, a general vasoconstricting activity such as activation of sympathetic nervous system and rennin angiotensin system may contribute to increase the resistances of the two vascular beds simultaneously <sup>23</sup>. On the other hand portal mean velocity as well as portal blood flow showed marked reduction in cirrhotic patients with refractory ascites than controls, which can be ascribed to high vascular resistance in cirrhotic liver <sup>24</sup>. 4 weeks after shunting renal artery resistive index was the only measurement that showed statistically significant improvement, which could be attributed to improvement of effective blood volume leading to suppression of various hormones and thus improves renal haemodynamics.

Few complications were described but they did not influence the general results. Complications related to shunt insertion were easily prevented and properly managed. Shunt occlusion was 30% in this study (peritoneal catheter occlusion 20% and pump chamber occlusion 10%) while in other series was 60%<sup>25</sup> (peritoneal catheter occlusion 20%, pump chamber occlusion 20%&venous catheter occlusion 20%). Zervos et al, reported loss of function due to occlusion in 52% patients, infection in 18%, and ligation for disseminated intravascular coagulation in 6% <sup>26</sup>.

Lengthening of the peritoneal catheter with a peritoneal dialysis catheter so that the catheter tip could rest in the Douglas pouch decreased the incidence of peritoneal catheter occlusion <sup>25</sup>. The incidence of venous catheter

occlusion was prevented when the catheter tip was positioned in the cavo -atrial junction (opposite to the right third intercostals space) where high blood velocity decreases the incidence of catheter tip thrombosis. Salvaging procedures we used in our series could restore a dysfunctional Denver shunt easier, safer and with a higher cost saving than a total revision of the shunt.

Gastrointestinal bleeding occurred in four patients 20% (gastric erosions 10% - variceal bleeding 10%). While in other series, it was 35.7% <sup>27</sup>.Variceal bleeding might be due to either part of systemic DIC or pressure alteration after peritoneo venous shunt with resultant venous congestion. Gastric erosions may be due to surgical stress. Most likely, pre-operative injection sclerotherapy as well as proton pump inhibitor prevent this complication. So patient selection and careful surgical procedure seems to be mandatory for better results.

The mortality rate was 0% in our study while in other series it was 14.3% <sup>27</sup>and 83% when done by interventional radiologist <sup>28</sup>. Causes of death included bleeding from preexisting varices, sepsis, hepatic failure, rupture of hepatoma, and disseminated intravascular coagulation <sup>28</sup>.

This wide discrepancy in the mortality rate may be due to the preventive measures used in our series as preoperative injection sclerotherapy, better orientation with the antiseptic techniques between surgeons and the interventional radiologists, better patient selection as well as discarding the peritoneal fluid and replacing it with saline solution.

Incidence of pleural effusion secondary to liver cirrhosis in the present search was 10%. Peritonea-venous Denver shunt in association with chest tube drainage and pleurodesis 3 weeks after shunt insertion succeeded in controlling recurrent pleural effusions secondary to liver cirrhosis who was unsuccessfully treated medically, or by repeated thoracentesis. Successful management of these patients is challenging, as many of the treatment options can be associated with increased morbidity. It was also successfully treated after transjugular intrahepatic portosystemic shunt placement <sup>29</sup>.Another therapeutic option was originally used by insertion of LeVeen peritoneo venous shunt. Ascites completely resolved but pleural effusion was continuously and severely reaccumulating. A Denver inverted shunt was subcutaneously inserted from pleural to peritoneal cavity. After operation CPAP was applied and pump device activated; pleural effusion gradually disappeared clearing completely the pleural space. The patient was discharged and followed for one year without ascites and pleural effusion <sup>30</sup>.

Recently, peritoneovenous shunt is a therapeutic option for the treatment of intractable ascites after liver transplantation <sup>31</sup>, after hepatic resection <sup>32</sup> or following a Whipple procedure <sup>33</sup>. It can be used to treat chylous ascites due to lymphangiomyomatosis <sup>34</sup>, or associating with radical retroperitoneal lymph node dissection and venacavectomy <sup>35</sup>.

In conclusion, Insertion of the peritoneo-venous Denver shunt seemed to be a minor operative procedure done under local anesthesia with minimal surgical stress. It provided good palliation for all patients with little morbidity and no operative related mortality. It improved renal haemodynamic as indicated by reduction of resistive index of the renal artery. Pre-operative injection sclerotherapy as well as proton pump inhibitor prevent post-operative gastro-intestinal bleeding. Peritonea-venous Denver shunt in association with chest tube drainage and pleurodesis 3 weeks after shunt insertion succeeded in controlling recurrent pleural effusions secondary to liver cirrhosis. So proper patient selection and careful surgical procedure seems to be mandatory for better results.

#### REFERENCES

- Garcia, N., Jr. and A. J. Sanyal (2001). "Minimizing ascites. Complication of cirrhosis signals clinical deterioration." Postgrad Med 109(2): 91-6, 101-3.
- 2. Runyon, B. A. (1998). "Management of adult patients with ascites caused by cirrhosis." Hepatology 27(1): 264-72.
- Watanabe, A. (1997). "Management of ascites. A review." J Med 28(1-2): 21-30
- Smith, D. A., D. W. Weaver, et al. (1989). "Peritoneovenous shunt (PVS) for malignant ascites. An analysis of outcome." Am Surg 55(7): 445-9.
- Pisani Ceretti, A., M. Intra, et al. (1997). "[Role of surgical therapy in the treatment of refractory ascites]." Minerva Chir 52(11): 1339-48.
- 6. Waggershauser, T., S. Muller-Schunk, et al. (2001). "[TIPS in patients with therapy refractory ascites and kidney dysfunction]." Radiologe 41(10): 891-4.
- Peron, J. M., K. Barange, et al. (2000). "Transjugular intrahepatic portosystemic shunts in the treatment of refractory ascites: results in 48 consecutive patients." J Vasc Interv Radiol 11(9): 1211-6.
- Weaver, D. W., R. G. Wiencek, et al. (1990). "Percutaneous Denver peritoneovenous shunt insertion." Am J Surg 159(6): 600-1.
- Guardiola, J., X. Xiol, et al. (1995). "Prognosis assessment of cirrhotic patients with refractory ascites treated with a peritoneovenous shunt." Am J Gastroenterol 90(12): 2097-102.
- Taylor, K. J. and S. Holland (1990). "Doppler US. Part I. Basic principles, instrumentation, and pitfalls." Radiology 174(2): 297-307.

- Zervos, E. E. and A. S. Rosemurgy (2001). "Management of medically refractory ascites." Am J Surg 181(3): 256-64.
- Massari R. Flugente R. et al. (1995). "Surgical treatment of refractory ascites with peritoneovenous shunt." Chir Ital 47(1): 57-60.
- Spreafico, C., G. Patelli, et al. (2001). "Percutaneous implant of Denver peritoneovenous shunt: a new opportunity for the interventional radiologist." Radiol Med (Torino) 102(3): 154-8.
- Utikal P. Kral V. et al. (1997). "Peritoneovenous shunt in the surgical treatment of ascites in patients with liver cirrhosis." Rozhl Chirg. 76(10): 497-501.
- Deen, K. I., A. P. de Silva, et al. (2001). "Saphenoperitoneal anastomosis for resistant ascites in patients with cirrhosis." Am J Surg 181(2): 145-8.
- Koike, T., S. Araki, et al. (2000). "Clinical efficacy of peritoneovenous shunting for the treatment of severe ovarian hyperstimulation syndrome." Hum Reprod 15(1): 113-
- 17. Allard, J. P., J. Chau, et al. (2001). "Effects of ascites resolution after successful TIPS on nutrition in cirrhotic patients with refractory ascites." Am J Gastroenterol 96(8): 2442-7.
- Chojkier, M. and R. J. Groszmann (1981). "Measurement of portal-systemic shunting in the rat by using gamma-labeled microspheres." Am J Physiol 240(5): G371-5.
- Fernandez-Munoz, D., C. Caramelo, et al. (1985). "Systemic and splanchnic hemodynamic disturbances in conscious rats with experimental liver cirrhosis without ascites." Am J Physiol 249(3 Pt 1): G316-20.
- Platt, J. F., J. H. Ellis, et al. (1994). "Renal duplex Doppler ultrasonography: a noninvasive predictor of kidney dysfunction and hepatorenal failure in liver disease." Hepatology 20(2): 362-9.
- Rivolta, R., A. Maggi, et al. (1998). "Reduction of renal cortical blood flow assessed by Doppler in cirrhotic patients with refractory ascites." Hepatology 28(5): 1235-40.
- 22. Rockey, D. C. and R. A. Weisiger (1996). "Endothelin induced contractility of stellate cells from normal and cirrhotic rat liver: implications for regulation of portal pressure and resistance." Hepatology 24(1): 233-40.
- 23. Floras, J. S., L. Legault, et al. (1991). "Increased sympathetic outflow in cirrhosis and ascites: direct evidence from intraneural recordings." Ann Intern Med 114(5): 373-80.
- 24. Kroeger, R. J. and R. J. Groszmann (1985). "Increased portal venous resistance hinders portal pressure reduction during the administration of beta-adrenergic blocking agents in a portal hypertensive model." Hepatology 5(1): 97-101.
- Hu, R. H. and P. H. Lee (2001). "Salvaging procedures for dysfunctional peritoneovenous shunt." Hepatogastroenterology 48(39): 794-7.

- 26. Zervos, E. E., J. McCormick, et al. (1997). "Peritoneovenous shunts in patients with intractable ascites: palliation at what price?" Am Surg 63(2): 157-62.
- Arciero, G., V. Di Blasio, et al. (1996). "[Early complications and long-term results of the LeVeen peritoneo-venous shunt in the treatment of refractory ascites]." Minerva Chir 51(11): 897-901
- Park, J. S., J. Y. Won, et al. (2001). "Percutaneous peritoneovenous shunt creation for the treatment of benign and malignant refractory ascites." J Vasc Interv Radiol 12(12): 1445-8.]
- 29. Conklin, L. D., A. L. Estrera, et al. (2000). "Transjugular intrahepatic portosystemic shunt for recurrent hepatic hydrothorax." Ann Thorac Surg 69(2): 609-11.
- Montanari, M., P. Orsi, et al. (1996). "Hepatic hydrothorax without diaphragmatic defect. An original surgical treatment." J Cardiovasc Surg (Torino) 37(4): 425-7.
- Mabrut, J. Y., E. de la Roche, et al. (1998). "[Peritoneovenous diversion using the LeVeen shunt in the treatment of refractory ascites after liver transplantation]." Ann Chir 52(7): 612-7.
- Maeda, T., M. Shimada, et al. (1995). "Strategies for intractable ascites after hepatic resection: analysis of two cases." Br J Clin Pract 49(3): 149-51
- Takahashi, T. and A. Kakita (1995). "Temporary use of peritoneovenous shunting for treatment of tense ascites following a Whipple procedure." Dig Dis Sci 40(9): 1946-50.
- Kimura, M., T. Morikawa, et al. (1996). "[Lymphangiomyomatosis with chylous ascites treatment successfully by peritoneo-venous shunting]." Nihon Kyobu Shikkan Gakkai Zasshi 34(5): 557-62.
- 35. See, W. A., T. F. Kresowik, et al. (1996). "Peritoneal venous shunting for the treatment of lymphatic ascites following retroperitoneal lymph node dissection." Urology 48(5): 783-5.