Egyptian Journal of Surgery Vol. (22), No. (4), Oct., 2003

THE DENVER PERITONEOVENOUS SHUNT;THERAPEUTIC EFFECTIVENESS AND COMPLICATIONS.

By
Yehia M. Safwat, Hassan A. El Garem***
Department of Surgery* and Tropical Medicine **, Faculty of Medicine, Cairo University.

This study was performed on 42 patients with intractable ascites due to liver cirrhosis. All patients had a Denver peritoneovenous shunt placed. There was a marked reduction in the body weight, and abdominal distension together with an increase in urine output. Almost all patients developed subclinical DIC, but only 3 manifested it clinically. We had several severe complications including variceal bleeding in 3, shunt sepsis in 2, fluid overload in 2, ascitic leak in 3, upper limb deep venous thrombosis in 1, encephalopathy in 1, and shunt occlusion in 5 patients. In this paper we shall discuss the patient selection, operative technique and complications of the procedure with special stress on how to avoid them

Keywords: Denver shunt, peritoneovenous shunt, intractable ascites.

INTRODUCTION

The word ascites comes from the Greek word askos, in the meaning of bag or sac. It is a pathological accumulation of fluid in the peritoneal cavity [1]. Refractory ascites is defined as ascites unresponding to 400 mg of Spironolactone, or 30 mg of amiloride with or without 160 mg furosemide daily for 2 weeks despite being on sodium restriction of 50 mEq or less per day [2]. In 1974, LeVeen [3] devised a peritoneovenous shunt with a one-way valve for the treatment of ascites. In 1979, the Denver shunt was introduced which is made of a more inert material and has a compressible pump with one or two valves [4]. The aim of this study is to utilize the Denver peritoneovenous shunt in a group of patients with intractable ascites for evaluation of its therapeutic effectiveness as well as the various complications associated with this type of therapy.

PATIENTS AND METHODS

This study was performed on 42 patients, 28 males and 14 females fulfilling the criteria mentioned by Zervos and Rosemurgy [5] as an indication for peritoneovenous shunt. Patient's age ranged from 34-62 years with a mean of 40.9 + 10.6 years. All patients were Child-Pugh class "C" [5] cirrhotics with refractory ascites not responding to medical treatment. At the time of hospital admission, all patients underwent history and physical examination including measurement of body weight, abdominal girth and urine output, complete set of liver and kidney function tests, CBC, coagulation profile and electrolytes. Ascitic fluid cytological and bacteriological examination including ascitic fluid culture if the neutrophil count was more than 250/ml. The criteria for inclusion included:-

1) Massive disabling ascites that is refractory to sodium retention (2 g/day), bed rest, diuretics (Spironolactone up to 400 mg/day and furusemide up to 160mg/day) for 1 week, and repeated paracentesis with albumin infusion replacement.

2) Absence of spontaneous clinical encephalopathy.

3) No variceal bleeding within 30 days preoperatively.

4) Serum bilirubin less than 6 mg/dl.

5) Prothrombin time prolongation less than or equal to 5 seconds.

6) Serum creatinine less than or equal to 2 mg/dl.

7) A satisfactory cardiac function reserve.

8) Absence of spontaneous bacterial peritonitis, either clinically or proved by ascitic fluid culture.

Other contraindications [6] include:-

1) Peritonitis.

2) Any intraabdominal infection.

3) Any systemic infection.

Patients were prepared for surgery by repeated large-volume paracentesis (4-6 L/day) with intravenous albumin infusion plus standard diuretic therapy. The majority of ascitic fluid was removed 1-2 days before surgery to avoid entrance of ascitic fluid into the circulation through the shunt with the risk of DIC, and circulatory overload.

The Denver ascites shunt (Fig. 1) is constructed from medical grade silicone and consists of a fenestrated peritoneal catheter, a venous catheter, a flexible pump chamber containing either 1 or 2 one-way valve(s). In the double valved shunt, the second valve serves as a check that prevents reflux of blood into the distal end of the venous catheter when the shunt is manually pumped. We only used the double-valved shunt in our patients. Both shunt ends may be shortened for optimal positioning at the cavo-atrial junction and the peritoneal cavity. A barium sulfate stripe in the wall of both shunt ends permits visualization by fluoroscopy if needed [6].

In the operating room, the shunt container is opened in a sterile fashion and one would have to exercise extreme care to prevent the shunt from coming in contact with towels, drapes or talc as silicone is highly electrostatic and attracts airborne particles easily. It is then immersed in sterile heparinized saline, then with the peritoneal end of the shunt immersed in the saline and the venous end elevated, the pump chamber is repeatedly compressed until the entire shunt is filled with fluid and is free from air bubbles and a free flow is obtained (Fig. 2). The venous end is then dropped in the heparinized saline and left there until the patient is prepared for its insertion. When the catheter is taken out of the saline, it should be grasped by the venous end, and one should make sure that this end is always at a higher level than the rest of the shunt to prevent air bubbles from entering the catheter [6].

The shunts were placed with the patients under a general anesthetic in 30 patients and under local anesthesia with sedation in 12 patients. Prophylactic third generation cephalosporin was given once the IV canula was inserted and continued for 3 days postoperatively. Patients were placed supine on the operating table with the head extended and tilted to the left side (Fig. 3), and a urinary catheter was placed. Patients were prepped and draped from the chin down to the symphysis pubis and from the midline to the right posterior axillary line.

The right internal jugular vein (more in line with the superior vena cava) is approached for venous access through a transverse incision located 1-2 cm above the clavicle between the two heads of the sternomastoid muscle. The incision is extended for 3-4 cm laterally from the medial head of the muscle. The vein is then isolated for 3-4 cm and two loose silk ties are placed around the vein proximally (cranially) and distally (caudally) for control. The wound is then packed gently with gauze.

A small incision 4-6 cm below the right costal margin just lateral to the midclavicular line is made with dissection carried down to the external oblique aponeurosis. At this stage, the pocket for the pump chamber is prepared using blunt dissection superiorly and laterally from the incision, separating the subcutaneous fat from the fascia overlying the muscle allowing the pump to lie immediately over the lower rib cage. Thereafter, the internal oblique and the transversus abdominis muscle fibers are split exposing the transversalis fascia and the peritoneum (Fig. 4). It is better, however, to avoid stripping the transverses abdominis from the peritoneum, as sometimes the peritoneum is too thin to hold the purse-string sutures by itself. Two purse-string sutures of nonabsorbable material are made in the peritoneum, the first 1.5 cm in diameter and the second about 2 cm in diameter. The peritoneum is then incised within the center of the purse-string and the remaining ascitic fluid should be suctioned out using the fenestrated-probe suction tip to reduce the risk of fluid overload and post-shunt coagulopathy. Half the suctioned amount is replaced with warm saline or lactated Ringer. Care should be taken when evacuating ascitic fluid, as rapid emptying may result in circulatory collapse. While maintaining an air-free shunt, the peritoneal catheter is then placed in the peritoneal cavity and directed towards the ipsilateral paracolic gutter. The purse-string sutures are then tied snugly around the catheter obtaining a water-tight closure to prevent ascitic fluid leak around the catheter, however, they should not be tied too tight to avoid blockage of the shunt. Pump suction is now checked by compressing the pump chamber which should lead to a free flow of fluid out of the venous end of the shunt. The muscles are then closed around the catheter and the peritoneal catheter clamped with a rubber-shod clamp, i.e. placing 2 pieces of rubber on a clamp, to avoid continuous ascitic leak in the operative filed.

The venous catheter is then tunneled subcutaneously from the abdomen to the neck incision using ideally a Denver Tunneler. We always used the longest artery clamp available, e.g. a Roberts clamp. The tip of the venous catheter attached to the clamp is advanced subcutaneously lateral to the breast then medially towards the neck incision. When a Roberts clamp is used, it usually cannot cut this whole distance in one shot, so a skin incision is made in the way and the tunneling is made in two stages (Fig. 5). The venous catheter is then pulled in the neck incision to allow the pump chamber to rest comfortably in the pocket already created. The venous catheter is then cut obliquely at the level of the second intercostal space maintaining a gentle curve at the clavicle (Fig. 6). This ensures placing the catheter tip at the cavo-atrial junction which ensures a pressure differential between the peritoneal and the venous catheter ends, which is necessary

for passive flow of ascitic fluid. Two fine atraumatic vascular clamps are placed proximally and distally over the internal jugular vein (Fig. 7) where a 5/0 monofilament arterial purse-string suture is now placed in the front wall of the vein (Fig. 8). A small longitudinal incision is made through which the venous catheter will be placed. Releasing the rubber-shod clamp from the peritoneal catheter will allow free flow of peritoneal fluid in the field. Insert the venous end of the shunt in the venotomy while releasing the distal clamp and direct it caudally till it sets in place (Fig. 9), then tie the purse-string suture. Manual compression of the valve is then done to verify that the shunt is working properly, if the valve did not yield to compression then this means that there is a distal (venous) obstruction, and if it fails to refill after compression, this means that there is a proximal (peritoneal) obstruction. The pump chamber is now sutured in place in its pocket using 2/0 nonabsorbable sutures placed through the suture pad of the pump (Fig. 10, 11), the two sides of the chamber should be sutured to avoid rotation. The proximal clamp and the ties are removed and all wounds are then closed in layers. The skin is marked over the pump chamber using intradermal methylene blue to facilitate its future use.

Because the pressure difference between both ends of the catheter is at its maximum when the patient is supine, this would result in maximum flow of ascitic fluid into the circulation. This flow can be stopped if the patient is placed in the sitting position. In the recovery room, patients remained with the head elevated at a 45 degrees angle for 30 minutes, 40 mg furosemide was given, and a blood sample was obtained for DIC profile (Platelet count, APTT, PT, Fibrinogen, and FDP's). On return of the DIC profile, patients whose results show less than a third drop in the platelet count and fibrinogen concentration from the preoperative levels were allowed to lie supine for 30 minutes before another blood sample was sent for a DIC profile. If at any time there was a decrease in the platelet count or fibrinogen concentration of a third or more, the patient was again placed with the head elevated at 45 degrees and kept in that position for eight hours, after which a DIC profile was obtained, and the sequence repeated.

In the postoperative period, daily assessment of the shunt patency was checked in the manner described above. Patients were instructed to depress the pump 20 times at bedtime and in the morning while lying in the supine position. This pumping is done by holding the pump firmly with the index and thumb of left hand and exerting pressure on the pump with the right index finger. A daily recording of the abdominal girth, body weight and urine output was done for three days. Measurement of PT, APTT, FDP's and Platelet count on the first and third postoperative days. Patients were discharged home or sent to the medical team after confirmation of shunt patency, a stable general condition, and stable serum electrolytes. Patients were instructed to pump their shunt for three times, three times daily. They were instructed of the warning signs as fever, melena, hematemesis, change in mental status, increase in abdominal size and rapid weight gain. They were strongly advised to avoid any knocks or falls and if that happens, they should come to check for shunt patency. It is important to advise these patients not to undergo any laparoscopic procedure to avoid carbon dioxide from entering the shunt. A monthly outpatient check was performed with questions about any previous pre-shunt symptoms like abdominal distension breathing difficulty, plus the previously mentioned warning signs and the shunt was examined for patency.

RESULTS

The Denver shunts were placed successfully in all patients without operative complications. The time taken to insert the shunt ranged from 40 – 90 minutes.

The duration of ascites was 14.1 ± 6.6 months. There was a history of variceal bleeding in three patients, the mean preoperative bilirubin was 4.4 ± 0.9 mg/dl, albumin was 3.0 ± 0.8 g/dl, creatinine 1.3 ± 0.5 mg/dl, and 16 patients were positive for hepatitis C virus.

The mean preoperative abdominal girth was 87.5 ± 5.6 cm. On postoperative day 1, the mean abdominal girth was 83.6 ± 5.4 cm, on day 3 was 80.9 ± 5.6 cm. This change was statistically significant (p=0.000). Again the body weight showed a significant (p=0.000) drop from the preoperative levels of 77.3 ± 7.3 to 75.7 ± 7.2 to 74.5 ± 7.4 kg on postoperative days 1 and 3 respectively. Also the urine output showed a significant (p=0.000) increase from the preoperative levels of 947 to 1169 to 1219 ml/day on postoperative days 1 and 3.

The mean preoperative PT was 17.6 ± 1.2 seconds, it rose to 37.7 ± 5.3 and to 40.9 ± 6.4 seconds on postoperative days 1 and 3 respectively (p=0.000). Also the mean preoperative APTT showed a significant (p=0.000) rise from the preoperative levels of 35.4 ± 5.7 to 78.7 ± 11.2 to 90.0 ± 12.8 seconds on postoperative days 1 and 3. Again the platelet count dropped significantly (p=0.000) from the preoperative levels of 196.3 ± 54.4 (x1000/cu mm) to 113.2 ± 41.2 and to 92.3 ± 34.6 on postoperative days 1 and 3 respectively. Also the FDP's were increased in all the patients on day 1. Therefore a subclinical DIC occurred in all patients after the shunt operation.

It was noted that the longer the duration of the ascites the faster the drop in body weight postoperatively ($p<0.05$). It was also noted in the study that patients with high serum bilirubin, particularly above 5 mg/dl, were more prone to develop a derangement in their coagulation profile

immediately postoperatively i.e. post-shunt coagulopathy.

The bilirubin level was above 5 mg/dl in 9 patients, these patients had a longer preoperative (p=0.013) and postoperative (p=0.034) PT on day 3, and a longer preoperative (p=0.000) and day 3 postoperative (p=0.023) APTT. The same relation was found with the platelet counts (p=0.007 and 0.009 respectively). When the same correlation was done for the serum albumin, there was no significant change in the coagulation profile between patients with a serum albumin < or > than 3 g/dl.

Eight different types of complications occurred in our patients (Table 1), these include:

1) DIC: In addition to the subclinical DIC that developed in virtually all patients, three patients developed it clinically, manifested by bleeding from their wounds and hematomas on postoperative days 3, 4 and 9. The first 2 patients had their Denver shunt ligated urgently under a local anesthetic, but these 2 patients died in the ICU. The third patient survived and did not require shunt ligation.

2) Variceal hemorrhage: This occurred in 3 patients on postoperative day 7, and 3 and 5.5 months postoperatively. None of these patients had a clinical evidence of DIC in the early postoperative period. Resuscitation, injection sclerotherapy and shunt ligation was done for all of them. One died 2 weeks later and another 6 months later from liver cell failure.

3) Shunt infection: Two patients developed shunt infection and systemic sepsis on days 7 and 30 postoperatively. The first was treated preoperatively for 10 days from spontaneous bacterial peritonitis. Both received systemic antibiotics and had their shunts removed. It is worthy of notice that culture of the shunt revealed staphylococcus aureus that is an unusual organism in spontaneous bacterial peritonitis.

4) Fluid overload: Two patients developed fluid overload, one on day 4 and was treated as usual in addition to elevation of the head to decrease the amount of fluid shunted, he recovered fully. The second patient developed bilateral lower limb edema on postoperative day 5 that resolved completely by leg elevation and furosemide.

5) Ascitic fluid leak from the neck or abdominal incision occurred in three patients in the first few days after shunt placement, and were managed conservatively. In another patient the leak developed several months postoperative in the form of distention of the subcutaneous tunnel (Fig. 12), this patient had re-exploration of the abdominal incision with further placement of tight purse-string sutures.

6) Upper limb DVT occurred in one patient 18 days postoperatively, was confirmed with Color Doppler imaging and treated with heparinization.

7) Encephalopathy leading to death on the 7th postoperative day was encountered in 1 patient.

8) Shunt occlusion after 30 days developed in 5 patients, was clinically evident and confirmed by Color Doppler scanning. Three patients had their shunts removed, as the obstruction was at the level of the pump, with reinsertion of a new shunt in the left internal jugular vein. In the other two patients the peritoneal end of the shunt was simply cleared from omentum and replaced between the right lobe of the liver and the diaphragm as described by Bitzer et al.[7].

At the end of the 12 months we had 20 cases of both morbidity and mortality. Five patients (11.9%) had died (2 from DIC, 2 from variceal hemorrhage and 1 from encephalopathy), five had their shunts ligated (2 for DIC and 3 for variceal hemorrhage), and five had their shunts removed (2 from infection and 3 from shunt obstruction).

Table (1): ***Complications***.

	Number of patients	*Management*	*Outcome*
DIC	3	2→ ligated 1→ left	2 died
Variceal Bleeding	3	3→ ligated	2 died later
Infection	2	Shunt removal	
Fluid overload	2	Conservative	
Ascitic Leak	3	2→ Conservative 1→ Operative	
DVT	1	Conservative	
Encephalopathy	1	Conservative	Died
Shunt occlusion	5	3→ Removed 2→ Cleared	

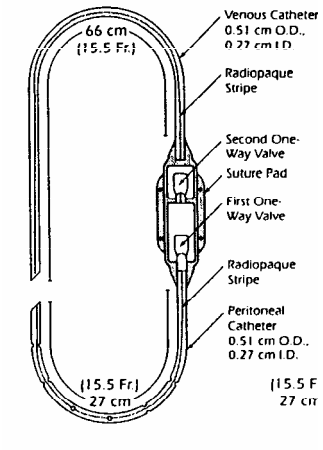

Fig. (1)

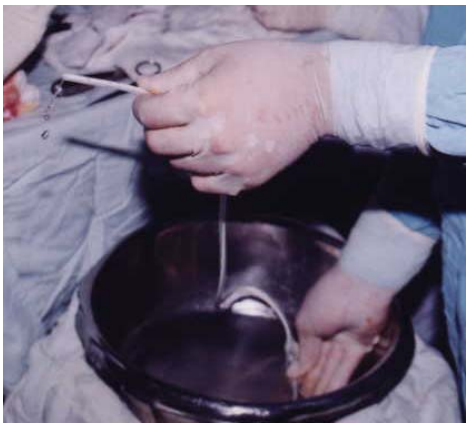

Fig.(2) : Preparing the shunt

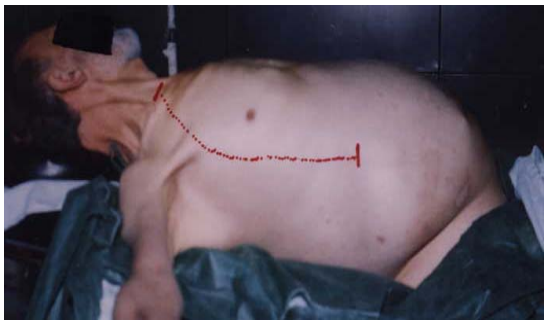

Fig. (3): Patient position, site of incisions and path of the shunt.

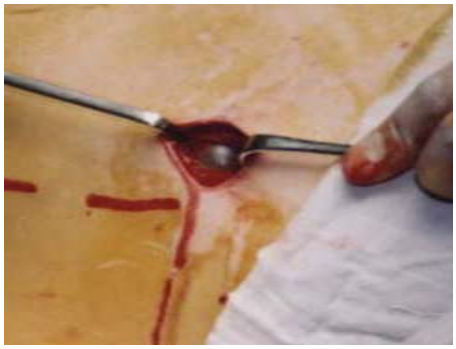

Fig. (4): Exposure of the peritoneum.

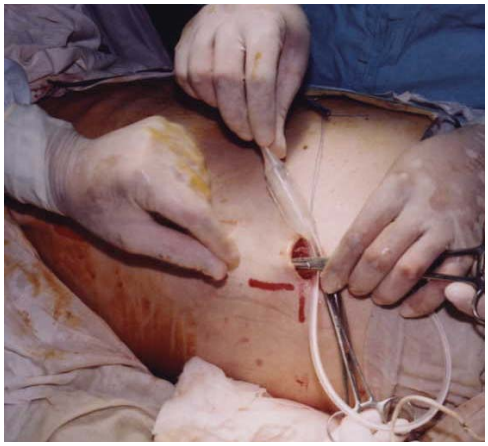

Fig.(5): Tunneling

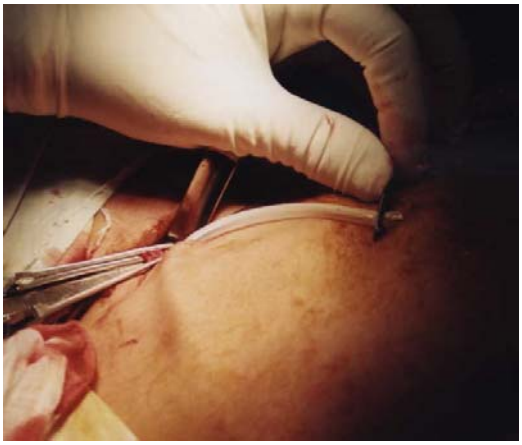

Fig.(6) : Cutting the catheter.

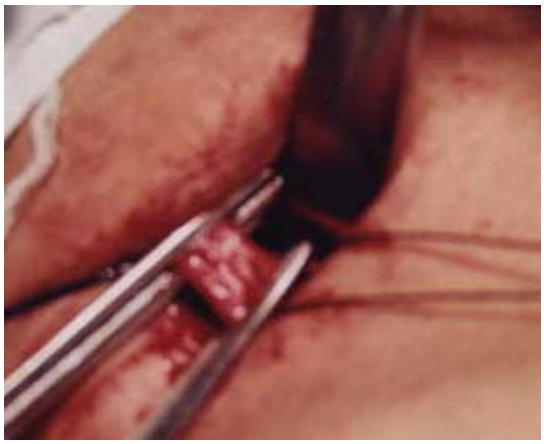

Fig. (7): Vascular clamps on the I.J.V.

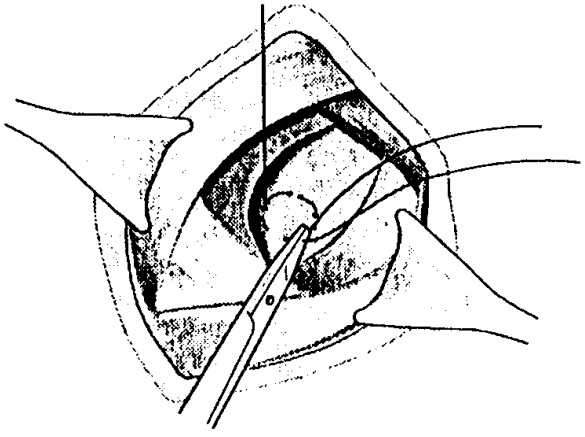

Fig. (8): Taking the venous purse-string suture (6).

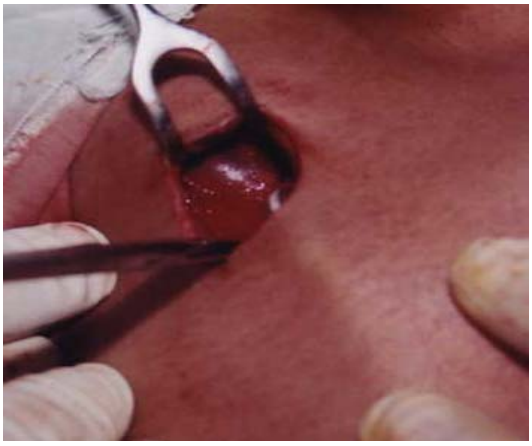

Fig. (9): The shunt in place

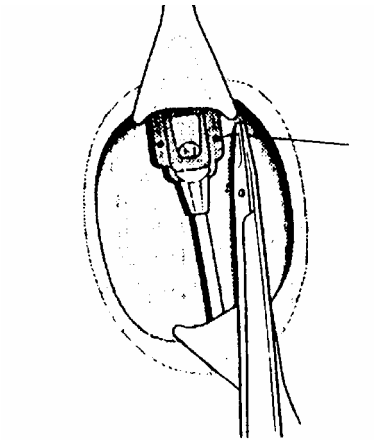

Fig. (10) : Suturing the pump (6).

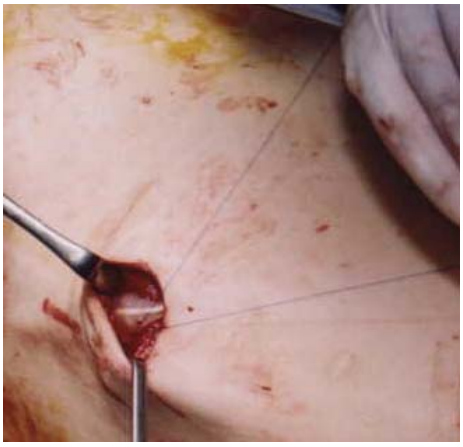

Fig.(11): Suturing the pump.

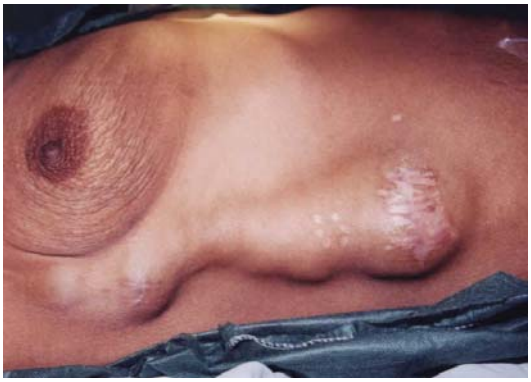

Fig.(12): Ascitic fluid accumulating subcutaneously along the tunnel.

DISCUSSION

At a late stage of liver cirrhosis, many patients are suffering from ascites, and the average 2-year survival is around 50% when ascites appears. Ninety percent of these patients can be managed conservatively, but there remains around 10% that will require some surgical procedure to relieve the ascites [8, 9]. These procedures include, saphenoperitoneal shunts [10, 11], peritoneovenous shunts [12], transjugular intrahepatic shunts [13, 14, 15], portocaval shunts [16], and liver transplantation [5]. Indeed, the only definitive treatment of ascites is liver transplantation [17], but the others methods are meanwhile applicable.

Peritoneovenous shunt is a simple and effective procedure for the control of refractory ascites but it will not prolong the life of the patients [9]. The LeVeen shunt was the first clinically used design of peritoneovenous shunts [3], but the postoperative mortality and morbidity were reported to be 20% and 50% respectively. More recently, the Denver shunt became increasingly popular because of its subcutaneous compressible valve chamber that can be manually flushed, and occlusion can be easily diagnosed at the bedside and a better short and long-term patency rate. Though the effects of these shunts are promising, they are not perfect designs however, as shunt dysfunction occurs frequently and revision procedures are needed. Removal of the shunt is sometimes necessary due to shunt infection and severe coagulopathy [18, 19].

In this study, the common symptoms reported by patients with intractable ascites such as abdominal distension, respiratory distress and diminished mobility were markedly improved after insertion of the shunt. Patients also noticed improvement of appetite and an increase in muscle mass. Re-infusion of the ascitic fluid into the circulation leads to expansion of the intravascular compartment, increase in the venous return and cardiac output and an increase in the renal blood flow and aldosterone level [20].

Although our patients were Child-Pugh class "C", those with a serum bilirubin above 6 mg/dl were excluded from our study. It has been shown [21] that the total serum bilirubin is a predictor of operative mortality. In addition, it was the only predictor of late postoperative survival. They also mentioned that patients with a serum bilirubin greater than 5 mg/dl or prothrombin time prolonged more than 5 seconds over the control value, were at the greatest risk for severe consumptive coagulopathy. When we used the cutoff of 5 mg/dl for serum bilirubin, we found a statistically significant affection of the coagulation profile.

DIC has been suggested to be due to pouring of ascitic fluid into the circulation which was found to have procoagulants like factor X and collagen [22], in addition to the preoperative liver-related DIC [23]. In our study we had a rate of 7.1 % for clinical DIC despite the significant derangement in coagulation profile in most of the patients as agreed to by others [24, 25, 26]. DIC can be minimized by suction of as much as possible of the ascitic fluid before placement of the shunt, and the selection of patients with as low a serum bilirubin as possible, because patients with higher serum bilirubin are more prone to develop DIC as described above. If DIC occurs it becomes mandatory to ligate the venous end of the shunt under local anesthesia to stop its propagation.

A combination of DIC and a rise in portal pressure owing to infusion of ascitic fluid and increased circulatory volume after shunt insertion has been blamed as a cause of post-shunt variceal bleeding [23]. The incidence of post-shunt variceal bleeding varies in the literature, and ranged from 7% [27], to 11% [28], to 30% [21]. This complication can be lessened by: the exclusion of patients with recent history of variceal bleeding, preoperative lowering of the portal blood pressure [6], trying to suction as much as possible of the ascitic fluid during the operation, decreasing the rate of ascites infusion via positioning the patient in the upright position postoperatively, and by careful monitoring of DIC parameters during the early postoperative period. Late variceal hemorrhage can still occur and its relation to shunting at this time remains controversial [23].

We had a 4.7% incidence of shunt sepsis. Others [29] had a 2% infection rate requiring shunt removal. The causative organism is staphylococcus aureus in half of the cases, which is the same organism isolated from our shunts. The patients with intractable ascites are at an end-stage liver disease and are immunocompromised, in addition to the fact that fewer bacteria are necessary to cause infection in the presence of a foreign body than in normal tissues [30], so the strictest aseptic precautions are required in these patients together with care in handling the shunt and the perioperative prophylactic antibiotics. In the future cases we shall use adhesive opsite over the whole prepped area to avoid contamination of the catheter.

Five of our patients (11.9%) had shunt occlusion, it was salvaged in 2 and replaced in three. If we compare our shunt failure rate with previous studies, we find that some [9] mentioned a high occlusion rate of 48.2% at 7 months. The reason for the high failure rates in these two studies might be because of inclusion of patients with malignant ascites. Hu and Lee [9] introduced some salvaging procedures for shunt occlusions, these include; lengthening of the peritoneal end of the shunt with a peritoneal dialysis catheter so it would rest in Douglas pouch in cases obstructed at the peritoneal end.

They also introduced a reservoir that can be placed instead of the pump, should the later be clogged by

proteinaceous material.

The overall morbidity in our series was high, reaching 47.61%, and a mortality rate of 11.9% (5 patients). Despite these terrifying figures, one cannot blame all of these complications on the shunt, some are due to natural course of patients with intractable ascites. Medical treatment of ascites, by itself, has its complications in the form of water and electrolyte imbalance, renal failure and encephalopathy [31]. Patients with intractable ascites carry a 50% mortality rate in 6 months with or without treatment [17] and ascites is mentioned to be a sign of death in patients with cirrhosis [32], so we can expect to get a significant amount of complications whether we treat or do not treat these patients.

CONCLUSION

Peritoneovenous shunting for patients with intractable ascites is a symptomatic operation that helps improve the patients' quality of life in the form of decreased abdominal distension and body weight together with increased muscle mass. It, however, does not affect the natural course of cirrhosis with its dreadful complications and ending. It can be suitable for patients with intractable ascites and good liver function tests especially a bilirubin level not more than 5 mg/dl, in these patients we expect to get a high incidence of post-shunt complications especially DIC which is the most dreadful sequel of peritoneovenous shunting.

REFERENCES

1. Runyon BA: Management of adult patients with ascites caused by cirrhosis. Hepatology 1998;27:264

2. Pisani CA; Intra M; Borzio M: Role of surgical therapy in the treatment of refractory ascites. Minerva Chir; 1997 52(11):1339-48

3. LeVeen HH; Christoudas G; Moon JP: Peritoneovenous shunting for ascites. Ann Surg 1974;180:580-591

4. LaVilla G; Arroyo V: Pathophysiology and treatment of ascites in cirrhosis. Hepatology 1990;12(3):465-7

5. Zervos EE; Rosemurgy AS: Management of medically refractory ascites. Am J Surg 2001;181(3):256-64

6. www.denverbiomedical.com

7. Bitzer LG; Tulman SA; Doerr RJ: Improving peritoneal venous catheter patency. Surg Gyn Obst 1993;177:415-6

8. Dulai G; Vora D; Hage A; Martin P: A novel complication of peritoneovenous shunt. Am J Gastroentrology;1997 93(8):1379-80

9. Hu RH; Lee PH: Salvaging procedures for dysfunctional peritoneovenous shunt. Hepato-Gastroenterology 2001;48:794-7

10. Vercruysse GA; Reed DN: Saphenoperitoneal shunts for patients with intractable ascites associated with chronic liver disease. Br J Surg 2002 Feb;89(2):245

11. Narayanan R: Saphenoperitoneal shunts for patients with intractable ascites associated with chronic liver disease. Br J Surg 1999 Dec;86(12):1588-9

12. Mabrut JY; de la Roche E; Adham M; Ducerf C; Baulieux J: Peritoneovenous diversion using the LeVeen shunt in the treatment of refractory ascites after liver transplantation. Ann Chir 1998;52(7):612-7

13. Hwang S; Park KM; Lee SG; Sung KB; Lee YJ; Choi DN; Ahn CS; Min PC: Transjugular intrahepatic portosystemic shunt for intractable posthepatectomy ascites. Hepatogastroenterology 2002 Nov-Dec;49(48):1669-72

14. Bahramipour PF; Festa S; Biswal R; Wachsberg RH: Transjugular intrahepatic portosystemic shunt for the treatment of intractable ascites in a patient with polycystic liver disease. Cardiovasc Intervent Radiol 2000 May-Jun;23(3):232-4

15. Waggershauser T; Muller-Schunk S; Holl J; Reiser M: TIPS in patients with therapy refractory ascites and kidney dysfunction. Radiologe 2001 Oct;41(10):891-4

16. Rodes J: Intractable ascites management: the role of side-to-side portacaval shunt. HPB Surg 1999;11(3):200-4

17. Choudhury J; Sanyal AJ: Treatment of Ascites. Curr Treat Options Gastroenterol 2003 6(6):481-491

18. Hillaire S; Labiance M; Borognova G; Smadja C: Peritoneovenous shunting of intractable ascites in patients with cirrhosis; Improving results and predictors of failure. Surgery 1993;113:373

19. Arroyo V; Gines P; Jimenez W; Rhodes J: Ascites, renal failure and electrolyte disorders in cirrhosis. In: McIntyre N; Benhamou JP; Bircher: Oxford Textbook of Clinical Pathology. Oxford University Press. 1991:429-70

20. Massari R; Fulgente R; Marivellis S; Romessis M: Surgical treatment of refractory ascites with peritoneovenous shunt. Chir Itl 1995;47:57-60

21. Fulenweider JT; Galambos JD; Smith RB: LeVeen versus Denver peritoneovenous shunt for intractable ascites of cirrhosis. A randomized prospective study. Arch Surg 1986;12:351-5

22. Ragni MV; Lewis JH; Spero JA: Ascites-induced LeVeen shunt coagulopathy. Ann Surg 1983;198:91

23. Moskovitz M: The peritoneovenous shunt: expectations and reality. Am J Gastroenterology 1990;85(8):917-29

24. Jaques W: Disseminated intravascular coagulation. In. Jaques W: Interpretation of Diagnostic Tests. Little Brown and Co. 1996:442-6

25. Schwartz ML; Swain WR; Vogel SB: Coagulopathy following peritoneovenous shunting. Surgery 1979;85:6712-6

26. Puig JG; Anton FM; Fernandez FA; Aguado AG; Rodriguez JJV: Peritoneovenous shunting for ascites. N Eng J Med 1980;303:461-5

27. Zervos EE; McCormick J; Goode SE; Rosemurgy AS: Peritoneovenous shunts in patients with intractable ascites. Am Surg 1997;63(2):157-62

28. Smadja C; Franco D: The LeVeen shunt in the elective treatment of intractable ascites in cirrhosis. A prospective study on 140 patients. Ann Surg 1985;201:488-93

29. Lund RH; Mortiz MW: Complications of Denver peritoneovenous shunting. Arch Surg 1982;117(7):924-8

30. Nocholas RL: Bacteriology in surgery. In: Nyhus LM and Baker RJ: Mastery of Surgery. Little Brown and Company. Boston 1992:83-98

31. Borie DC; Vaillant JC; Breton S; Hannoun L: Role of surgery in the treatment of refractory ascites in cirrhotic patients. Ann Chir 1999;53(10):966-72

32. Nordin A; Makisalo H: Ascites--a sign of death? Duodecim 2000;116(19):2074-83