Paracentesis-induced circulatory dysfunction: are there albumin alternatives?

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Abstract

Background: Ascites is one of the main complications of advanced liver cirrhosis. It is defined as a pathological accumulation of free fluid in the peritoneal cavity.

Main body of the abstract: Ascites is a sign of decompensation in patients with liver cirrhosis and is associated with decreased survival. Ascites is associated with bad cosmetic figure and poor quality of life. Ascites is a predisposing factor for developing hydrothorax, hernias, diastolic dysfunction, spontaneous bacterial peritonitis, and renal impairment especially hepatorenal syndrome. The main treatment is salt restriction and diuretics. By the time the patient become non-responder and develop tense ascites, abdominal large volume paracentesis is the treatment of choice. Its advantages are rapid, cheap, and 1 day hospitalization. The main drawback is the development of paracentesis-induced circulatory dysfunction (PICD) if no volume expanding drugs are used. PICD is associated with dilutional hyponatremia, renal impairment, so it is considered the silent killer. Albumin infusion is the standard preventive measure but since costly to other alternatives such as colloids, vasoconstrictors or lowering the standard doses of the albumin was studied and is promising.

Conclusions: This review summarized the effectiveness of other alternative drugs.

Keywords: Ascites, Large volume paracentesis, Albumin, Paracentesis-induced circulatory dysfunction

Background

The ancient Egyptians identified liver disease and linked the presence of ascites to disease of the liver as early as the time of the Ebers papyrus, where paragraph 865 refers to a swelling of the abdomen with the “water going up and down” or freely moving, which is a very accurate description of ascites and how to detect it clinically [1, 2]. The recommended treatment was to pierce the abdomen with what is called a “hmmm” (supposedly a knife or sharp instrument) to drain the fluid, as in performing paracentesis. they also noted that “hardness of the liver” was a risk factor for ascites formation [3]. Hippocrates was the first one who use the term “ascites” which is derived from the Greek word (άσϰο; askos), i.e., a leather bag that was used to carry fluids [4]. Half of the cirrhosis patients develop ascites within 10 years. After the occurrence of ascites, the survival decreases dramatically where 50% of the patients survive for 2–5 years. The 1- and 5-year survival without transplantation is 85 and 55%, respectively [5].

Liver cirrhosis and portal hypertension account for 75% of all ascites cases [6]. Portal hypertension, arterial vasodilatation, neurohumoral activation, and renal dysfunction are the proposed pathogenesis factors [7]. Clinically, ascites can be divided into 3 grades in which the 1st and 2nd grade are treated by diuretics but the third grade is characterized by tense or marked ascites that requires large volume paracentesis (LVP) in addition to diuretics [8]. Salt restriction and diuretics (spironolactone and furosemide) are effective for ascites mobilization in 90% of
the patients. Twenty percent will become diuretic resistant, while 10% of the patients will not respond from the start [9, 10].

Main text
Refractory ascites was identified by the International Ascites Club in a consensus conference in 1996, and it was defined as “ascites that cannot be mobilized, or early recurrence of ascites which cannot be satisfactorily prevented by medical therapy” [11].

Modalities for tense ascites management
Till now, the two common tools for management of patients with tense or refractory ascites are LVP and transjugal intrahepatic portosystemic shunt (TIPS) [9, 10]. LVP is an outpatient maneuver that is highly effective for the rapid relief of ascites and related symptoms [12]. It is effective in removing ascites in one single tap using suction pump to decrease the time to 1–2 h [13]. Compared to diuretics, it is more effective in maintaining ascites-free state, shorter hospital stay, fewer complications, and mobilization of edema [14].

In fact, LVP is a known maneuver since ancient Greeks but it lost the interest with advent of diuretics but it returned back for use in mid-1980s [15]. Beethoven underwent LVP twice (11 liters and 22 liters) but the second time was infected and leaked, which resulted in his death [16]. He was excited when he saw the ascitic fluid coming out, and he shouted that it reminded him of Prophet Moses who struck the rock with his staff and made the water gush forth [17, 18].

LVP may be an indirect measure of patient compliance as regards sodium intake. About 1300 mmol sodium is removed by 10 L LVP. If the patient is consuming 88 mmol/day sodium without urinary sodium excretion and just 10 mmol/day nonurinary sodium, 10 L LVP will remove 17 days of retained sodium in a patient with no sodium excretion. As a result, if the frequency of LVP > 2 times/month, this means the patient is non-compliant with salt restriction [9, 19].

LVP is a safe maneuver, and the absolute contraindication is clinically evident fibrinolysis or clinically evident disseminated intravascular coagulation [20]. Pregnancy, severe bowel distension, and previous abdominal surgery are relative contraindication for non-ultrasound-guided (blind) paracentesis [21].

TIPS is an artificial bypass in which a shunt connects the portal vein to the hepatic vein through the liver. It was used in the past mainly for the treatment of variceal bleeding. Now, it is used for treatment of refractory ascites and hepatic hydrothorax [22]. Since the venous return increases with TIPS, the effective blood volume and the renal perfusion will improve with subsequent natriuresis. Hence, there is decreased rate of ascites formation or recurrence but unfortunately, the incidence of hepatic encephalopathy increases [23]. It was found that the nutritional status and quality of life improve markedly with TIPS [9].

Which is the better, LVP or TIPS? It is a continuous debate but generally, we should begin with LVP first but if the patient needs > 3 sessions per months, we should assess for TIPS [10].

Paracentesis-induced circulatory dysfunction:
Paracentesis-induced circulatory dysfunction (PICD) is a serious complication of performing LVP without a plasma expander. It is defined as 50% increase in the plasma renin activity (PRA) over the baseline on the sixth day after treatment, up to a value > 4 ng/mL per hour [24].

Without the use of plasma expander, the following hemodynamics were recorded. Immediately after paracentesis, there is decreased abdominal pressure and increased venous return with suppression of the renin-angiotensin-sympathetic nervous systems [25]. Unfortunately, opposite changes occur after 12 h with higher levels compared to before LVP [26]. In such case, the incidence of PICD was 75% [27], and it may persist for months [28].

PICD is an asymptomatic condition but it is associated with serious effects as rapid re-accumulation of ascites, dilutional hyponatremia, increased incidence of hepatorenal syndrome, and short survival [29, 30]. These consequences are noted if LVP is more than 5 L [27]. The development of PICD is not related to the paracentesis flow rate [31] unlike older studies [32]. Recently, β blockers were accused as a risk factor for PICD. Sersté et al. [33] performed a self-control cross-over study. Ten patients with cirrhosis and refractory ascites treated with β blockers were selected. When patients were given β blockers, the prevalence of PICD was 80%. After the withdrawal of β blockers, the prevalence of PICD dropped to 10%. Therefore, the authors concluded that the use of β blockers may be associated with a high risk of PICD in patients with cirrhosis and refractory ascites.

PICD prevention
PICD can be prevented by multiple measures as albumin infusion, synthetic colloids, extracorporeal ultrafiltration and reinfusion, vasoconstrictors (midodrine, terlipressin, and noradrenaline), and recently low-dose albumin (Fig. 1).

All the studies compared a test drug to the standard of care (standard dose albumin infusion) but our study [29] was the first head-to-head multiple drug comparison. Synthetic colloids showed contradictory results. Gines et al. [28] did not find dextran 70 useful. The same result was reported by El-Ashry et al. [34] and Nasr et al. [35]. Other studies found dextran 40 [36] and 3.5%
polygeline (Haemaccel®) [37] were also ineffective drugs. Recently, hydroxy-ethyl starch (HES 6%) was found to be effective (6–8 g/L) [29, 38]. Christidis et al. [39] found that HES could increase the portal pressure as it filled the Kupffer cells (lysosomal storage), and this would increase the risk of variceal bleeding.

Terlipressin is a splanchnic vasoconstrictor. It was studied for PICD prevention [29, 40–42]. Moreau et al. [40] evaluated 20 patients with tense ascites, 10 received terlipressin (total 3 mg; 1 g IV before, 8 h, and 16 h after paracentesis) and the other 10 patients received albumin replacement (8 g/L ascites removed; 50% infused within 2 h after paracentesis and 50% after 6 h). In each group, 3 patients developed PICD. There was significant weight reduction and no renal impairment development in both groups. Singh et al. [41] assigned 40 patients with tense ascites into 2 groups as Moreau et al. [40]. In each group, 2 patients developed PICD. There was significant weight reduction, but in each group, one patient developed renal impairment [41]. Lata et al. [42] studied 49 patients but he used different doses. Twenty-four received terlipressin (1 mg every 4 h for 48 h; the first dose given immediately after paracentesis), and the other twenty five received albumin replacement (8 g/L ascites removed; 50% infused with 2 h after paracentesis and 50% after 6 h). The results in both treatment procedures suggested a similar efficacy for PICD prevention. In both groups, on the first 3 days, there was a tendency to improve hemodynamics reflected by the renin-angiotensin-aldosterone system activity. In the terlipressin group, this tendency approached statistically significant levels. All these studies found that terlipressin may be used as a measure for PICD prevention [29, 40–42].

Noradrenaline was assessed only in one study by Singh et al. [43] with promising results but no further studies. Midodrine is an α1-agonist that increases the systemic and splanchnic blood pressure, circulating blood volume, and renal perfusion [44]. Midodrine is beneficial in hypotensive cirrhotic patients with refractory ascites [45].

Studies were contradictory as regards midodrine as a preventive measure for PICD [24, 29, 46]. Singh et al. [46] randomized 20 patients to receive midodrine (titrated dosage 5–10 mg/8 h to maintain a mean arterial pressure 10 mmHg above baseline for 72 h) and the other 20 patients to receive albumin (8 g/L ascitic fluid removed). No patient developed PICD in the midodrine group versus 2 in the albumin group. In contrast, in a study of 24 patients, Appenrodt et al. [24] randomized 11 patients to receive midodrine (12.5 mg every 8 h for 2 days) and the other 13 patients to receive albumin (8 g/L ascitic fluid removed). In the midodrine group, one patient developed HRS precipitated by pneumonia, and he was excluded as he received terlipressin as treatment and eventually died. Six patients out of 10 (60%) developed PICD in the midodrine versus 4 patients out of 9 (31%) in the albumin group. There was marked increase in the plasma renin in the midodrine group. The authors did not recommend utilization of midodrine for the prevention of PICD. Notably, they gave fixed dose of midodrine for relatively shorter period, the patients did not stop diuretics, and finally, they did not exclude the diabetic patients. Hamdy et al. [47] found that patients in the midodrine arm had more incidence of renal impairment and PICD. Seven patients died in the midodrine arm in contrast to none in the albumin group.

Being much cheaper than albumin and terlipressin and much easier to administer, midodrine may be worth more trials to assess its use instead of albumin.

Albumin infusion is the approved standard-of-care measure for PICD prevention [48, 49]. Gines et al. [27] found that albumin infusion decreased the incidence of PICD from 80 to 15–20%. A study by Tan et al. [50] had the same conclusion.

On one hand, there is some controversy regarding albumin infusion as being very expensive, causing inhibition of the endogenous albumin synthesis and increasing its degradation [51, 52]. On the other hand, albumin has many beneficial functions that reinforce its use apart from being plasma-expanding drug. Albumin detoxifies harmful reactive oxygen, stabilizes the endothelium, prevents platelets aggregation, and can modulate the immune and inflammatory responses [53].

In 2012, a meta-analysis conducted by Bernardi et al. [54] found that albumin infusion caused 60% decrease of PICD development. Furthermore, albumin infusion
decreased the hyponatremia and mortality. In 2017, Kütt ing et al. [55] conducted another meta-analysis with contradictory results. Kütt ing et al. found that albumin infusion decreased the incidence of hyponatremia and PICD development in patients without hepatocellular carcinoma. Albumin infusion in this meta-analysis unfortunately did not decrease mortality. Even in patients with hepatocellular carcinoma, the survival did not increase with albumin infusion. Their final conclusion was against albumin infusion.

The albumin dose is 6–8 g per liter of ascitic fluid removed [48, 49, 56]. It can be given once the session completed [57] or giving half the dose immediately after the paracentesis and the other half 6 h later [28, 29, 58].

Low-dose albumin infusion was studied by Alessandria et al. (4 g/L ascites removed) [59] and Alsebaey et al. (2 g/L ascites removed) [29]. Thirty-five patients were randomized by Alessandria et al. [59] for half dose albumin (4 g/L ascites) and 35 patients to full dose albumin (8 g/L ascites removed). PICD developed in 5 patients (14%) in the half dose albumin group and in 7 patients (20%) in the full dose albumin group with no significant difference. They concluded that albumin in half dose is equivalent to full dose albumin in prevention of PICD. Alsebaey et al. [29] found same finding despite lower doses.

Our study [29] was the first head-to-head comparison of different drugs for PICD prevention. It included 125 patients randomized to 5 equal groups. In the albumin group, the dose was 6 g/L. In the low-dose albumin group, the dose was 2 g/L. Hydroxyethyl starch (HES 130/0.4) was given as 8 g/L. Terlipressin dose was 1 mg IV at the onset then every 8 h for a maximum of 3 g per day. Midodrine was administered orally for 3 days (5–10 mg 8 hourly to maintain the MAP at 10 mmHg > the baseline). All groups were statistically comparable as regards the age, sex, the etiology of the liver disease, and the Child-Pugh score. Most patients had hepatitis C-related liver cirrhosis. The average volume of ascitic fluid removed was 13 ± 0.14 L. PICD occurred in two patients in both terlipressin and HES 130/0.4 groups. In each albumin group, three patients developed PICD. Five patients in the midodrine group developed PICD. Comparing all groups showed no significant difference. We concluded that low-dose albumin, terlipressin, and HES 130/0.4 were good alternatives to the standard-of-care albumin dose.

We published another new study [60] focusing on the use of low-dose albumin 2 g/L ascitic fluid removed (n = 85). The incidence of PICD development was comparable to the standard dose albumin. In the same study, we did again comparison of the new number of the low-dose albumin to the other old arms, namely, terlipressin, midodrine, HES 130/0.4, and standard dose albumin. All the groups again were the same for PICD development.

The recent European Association for the Study of the Liver guidelines [56], however, still recommend albumin (8 g/L ascites removed) and did not recommend other alternatives.

**Conclusions**

PICD is a harmful complication of large volume paracentesis that should be prevented by plasma expanders. Albumin infusion in the standard dose is the standard drug till now but other alternatives are promising.

**Abbreviations**

LVP: Large volume paracentesis; TIPS: Transjugular intrahepatic portosystemic shunt; PICD: Paracentesis induced circulatory dysfunction

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**Authors’ contributions**

AA put the plan of the review article, searched the PubMed and google scholar for relevant studies, summarized them, and wrote the manuscript. ER and IW revised the manuscript. All authors have read and approved the final manuscript.

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