



# Fluid Management in Liver Failure

# 21

Michaël Mekeirele and Alexander Wilmer

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M. Mekeirele (✉)

Department of Critical Care, Vrije Universiteit Brussel (VUB), Universitair Ziekenhuis Brussel (UZB), Jette, Belgium

e-mail: [Michael.Mekeirele@uzbrussel.be](mailto:Michael.Mekeirele@uzbrussel.be)

A. Wilmer

Medical Intensive Care Unit, Katholieke Universiteit Leuven (KUL), Universitair Ziekenhuis Gasthuisberg, Leuven, Belgium

e-mail: [alexander.wilmer@uzleuven.be](mailto:alexander.wilmer@uzleuven.be)

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**IFA Commentary (PN)**

Critically ill patients with liver disease are known to experience hemodynamic alterations, which can lead to systemic hypotension, reduced cardiac output, and altered vascular resistance. These changes can be due to a variety of factors such as changes in the hepatic vasculature, reduced synthesis of vasodilatory and vasoconstrictive factors, and alterations in the activity of the renin-angiotensin-aldosterone system. Fluid therapy is a vital component of managing critically ill patients with liver disease, as these patients are prone to developing complications such as fluid and electrolyte imbalances, bleeding, and hypotension. The goals of fluid therapy in these patients are to restore effective circulating volume, improve organ perfusion, and prevent complications such as acute kidney injury. The best monitoring tools to guide fluid therapy depend on the patient's individual circumstances, but some commonly used tools include: first, hemodynamic monitoring that involves the use of various methods to directly measure cardiovascular parameters such as cardiac output, systemic vascular resistance, and central venous pressure. Commonly used techniques include pulmonary artery catheterization, echocardiography, and arterial waveform analysis. Second, fluid responsiveness assessment that involves assessing the patient's response to fluid administration, which can help guide further therapy. Various methods can be used to assess fluid responsiveness, such as dynamic variables (such as pulse pressure variation) and passive leg raising. Finally, serum biomarkers that measure serum markers such as lactate, central venous oxygen saturation, and hepatic venous pressure gradient can help assess the patient's hemodynamic status and guide fluid therapy. One key consideration in fluid therapy for liver disease patients is the potential for fluid (hypovolemia and hypervolemia) and electrolyte imbalances. This can be exacerbated by the presence of ascites, which may require treatment with diuretics or paracentesis. Additionally, patients with liver disease may have impaired renal function, making careful monitoring of fluid balance and renal function essential. In terms of the type of fluid given, there is ongoing debate about the optimal fluid, with some studies suggesting that balanced crystalloids (without lactate buffer) may be preferable to saline in reducing the risk of kidney injury and other complications. However, individual patient factors must be considered when selecting a fluid type. The type, dose, and use of albumin is a subject of ongoing research and debate. Some studies suggest that albumin can improve hemodynamics and reduce mortality in patients with liver disease, while others have found no significant benefit. Overall, fluid therapy in critically ill patients with liver disease is a complex and dynamic process that requires careful monitoring and individualized management to avoid potential complications and optimize patient outcomes.

**Learning Objectives**

After reading this chapter, you will:

1. Be able to describe the hemodynamic alterations specific to cirrhosis.
2. Be able to integrate the hemodynamic parameters of a patient with liver failure.
3. Be able to choose the right tools to guide fluid therapy in liver failure.

**Case Vignette**

A 45-year-old male with CHILD C liver cirrhosis is admitted to the ICU following a variceal hemorrhage. During stabilization, he received 1 L of balanced crystalloids. The bleeding was stopped after variceal ligations. High-dose PPI and Terlipressin were initiated. At this time, he is still hypotensive 80/35 mmHg (MAP 50 mmHg) and has marked peripheral edema, ascites, and an  $S_{c,v}O_2$  of 75%. He feels cold peripherally and has a mottled skin. When applying a passive leg raise, his blood pressure increases to 90/40 (MAP 59 mmHg). The lactate level is 15 mmol/L.

**Questions**

- Q1. Would you administer this patient an extra fluid bolus?
- Q2. What could help you decide between the administration of fluids or the application of either inotropes or vasopressors.
- Q3. What would you do if the lactate level decreased to only 10 mmol/L after 6 h despite your best efforts?

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**Introduction**

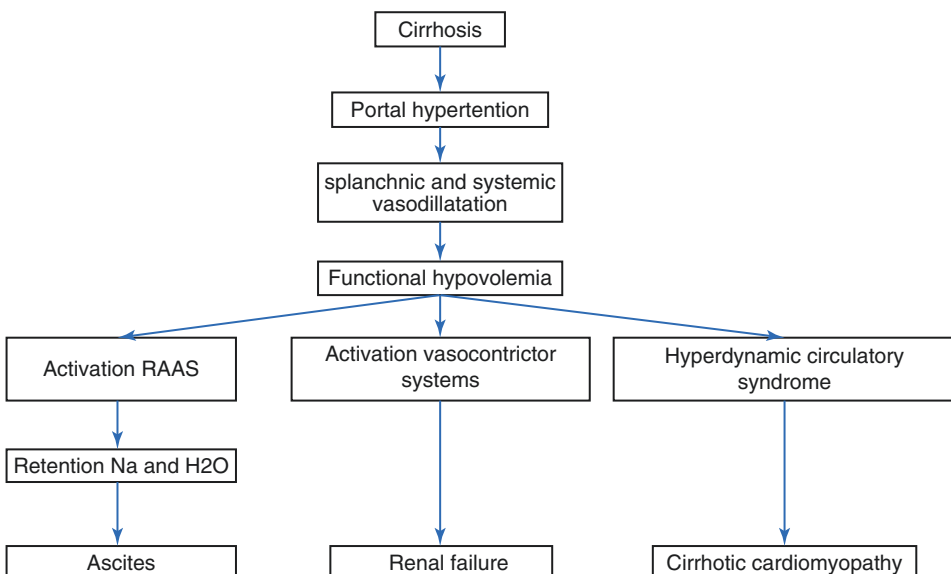
The general concepts of fluid management also apply to patients with liver failure. However, both acute liver failure (ALF) and acute-on-chronic liver failure (ACLF) are associated with marked hemodynamic changes due to inflammation, portal hypertension, and diminished clearing capacity of the liver for vasoactive substances. These hemodynamic changes are characterized by decreased systemic vascular resistance, increased cardiac output, central functional hypovolemia, increased arterial compliance, and peripheral vasodilatation. It is important to take these changes into account to determine the timing and volume of fluid administration and/or application of vasopressors and when interpreting the results obtained by hemodynamic monitoring devices. Although complex, the importance of adequate fluid management in liver failure cannot be overstressed since both hypervolemia and hypovolemia can further compromise residual liver function [1]. The choice of fluids in liver failure is relevant and perhaps more specific given the undeniable beneficial effects of albumin in certain indications. Particularly in acute on chronic liver failure, the advantages of albumin exceed those of mere volume expansion [1–5].

In this chapter, we will first discuss fluid management in acute on chronic liver failure. This syndrome is characterized as an acute liver decompensation in patients with a chronic underlying liver disease (mostly cirrhosis) with a high short-term mortality due to multiple organ failure. Second, we will discuss the scarce data on fluid management in patients with acute liver failure without underlying liver disease. This chapter will focus on adult patients, and more information on fluid therapy in children can be found in Chap. 20. Some other chapters will discuss fluids in specific populations: sepsis (Chap. 14), heart failure (Chap. 15), trauma (Chap. 16), neurocritical care (Chap. 17), perioperative setting (Chap. 18), burns (Chap. 19), abdominal hypertension (Chap. 22), and COVID-19 (Chap. 26).

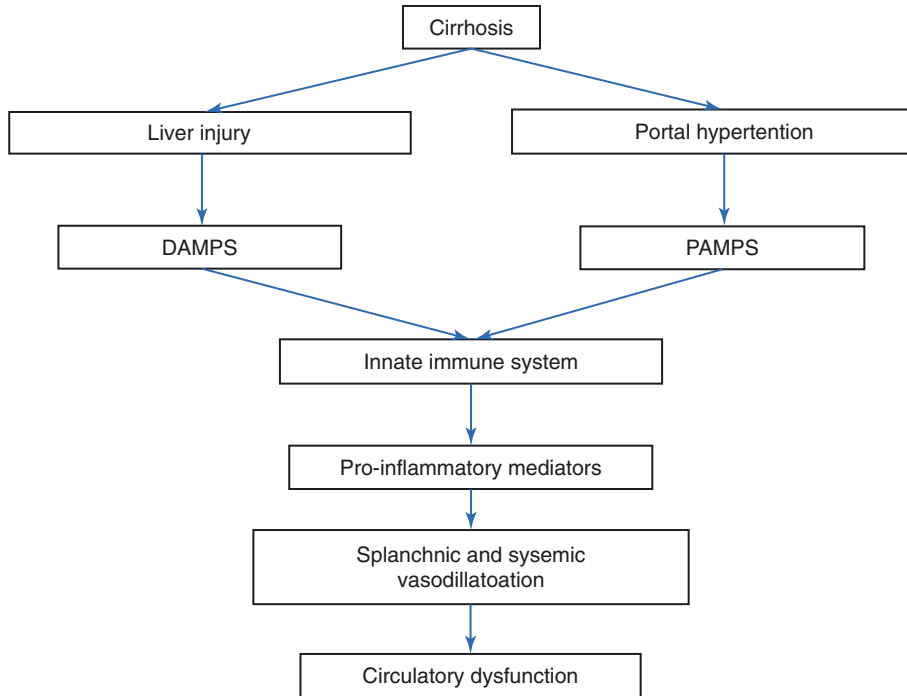
## Acute-On-Chronic Liver Failure (ACLF)

### Pathophysiology of Circulatory Dysfunction in Patients with Cirrhosis

While the changes in hemodynamics of patients with cirrhosis have been described for years, a paradigm shift has occurred recently concerning the pathogenesis of these hemodynamic alterations. The “classical” view is that cirrhosis obstructs portal flow leading to portal hypertension which in turn induces splanchnic and systemic vasodilatation (Fig. 21.1). This vasodilatation leads to a state of functional hypovolemia with three important aspects. First, this activates the renin-angiotensin-aldosterone (RAAS) cascade leading to ascites formation via the retention of sodium and water. Second, this functional hypovolemia activates vasoconstrictor systems, including renal vasoconstriction. This can lead to renal failure although the patient will remain in a functional hypovolemic state



**Fig. 21.1** Classical view on hemodynamic alterations due to cirrhosis



**Fig. 21.2** New view on hemodynamic alterations due to cirrhosis

despite the activation of RAAS. Third, this hypovolemic state can induce a hyperdynamic circulatory syndrome potentially leading to a cirrhotic cardiomyopathy. The latter, in its milder form, blunts the contractile responsiveness to stress and alters diastolic relaxation [1, 6, 7]. The new view on the pathogenesis of the hemodynamic alterations emphasizes the importance of inflammation in cirrhosis (Fig. 21.2). In this hypothesis, cirrhosis leads to both liver injury and portal hypertension increasing plasma concentrations of damage associated molecular patterns (DAMPS) and pathogen associated molecular patterns (PAMPS). These DAMPS and PAMPS activate the innate immune system, causing the release of proinflammatory mediators which in turn cause splanchnic and systemic vasodilation and circulatory dysfunction [1, 8]. It is important to recognize that due to fluid retention the average circulating blood volume in patients with cirrhosis is higher than in healthy persons [7]. However, the volume is unevenly distributed between central and abdominal compartment [9, 10]. With increasing severity of cirrhosis, this uneven distribution is magnified and becomes relevant for fluid administration [7].

## Hemodynamic Approach During Decompensation

### General Background

Classical signs of decompensated liver failure include ascites and edema. Large volumes of extracellular fluid may accumulate in the form of ascites and edema, while the patient

may be volume depleted intravascularly. This implies that over-resuscitation with IV fluids can worsen the situation by aggravating ascites formation, edema, hyponatremia, cardiac dysfunction, and intra-abdominal hypertension. Fluid accumulation and overload can also increase portal hypertension and induce gastro-intestinal bleeding in this population [1].

Assessment of the intravascular volume status in these patients is not easy. To understand what measures should be taken, it is important to know that during resuscitation we aim to rebalance the scales of supply and demand. To achieve this goal, we try to optimize the cardiovascular function. This can be done by manipulating preload (mainly through fluids), cardiac contractility (using inotropes), or afterload (mainly through vasopressor titration). To fully address this issue a more accurate evaluation should focus on the dynamics of change after interventions rather than on data limited to blood pressure, heart rate, or urine production. More complex, but more robust dynamic tests include the use of thermodilution methods or echocardiography. These tools allow a better distinction between intravascular and extravascular fluid status, cardiac function, and vasodilatation. A thorough review of these tools can be found in the first chapters of this book. In this section, we will further discuss the pitfalls in the interpretation of the hemodynamic status in ACLF. In addition, we will review different types of shock in ACLF since these determine the appropriate choice of fluid as well as the timing to start vasopressors or inotropes.

### **Special Considerations During Resuscitation**

The decompensated patient with cirrhosis often has a lower blood pressure at baseline. Some authors state that a mean arterial pressure of 60 mmHg could suffice during resuscitation, while others stress the importance of mean arterial pressure (MAP) above 65 mmHg given the high incidence of renal failure in cirrhosis [11]. We prefer a MAP above 65 mmHg. Beyond the monitoring of blood pressure and heart rate, successful resuscitation in intensive care is frequently evaluated by changes in central venous oxygen saturation ( $S_{cv}O_2$ ) and lactate clearance.  $S_{cv}O_2$  is used as a surrogate marker for cardiac output. Since patients with ACLF are generally hyperdynamic,  $S_{cv}O_2$  is usually normal or slightly elevated even in the presence of hypovolemia. An approach to volume resuscitation based on lactate clearance should consider that a damaged liver will clear lactate at a slower pace. This implies that in patients with cirrhosis, the evolution of lactate over time is more important than the absolute value [12].

In patients with cirrhosis, the normal values of dynamic preload indices such as pulse pressure variation (PPV) and stroke volume variation (SVV) can be altered [13]. On the one hand, a low systemic vascular resistance can theoretically alter the aortic compliance and in this way alter the PPV [14, 15]. On the other hand, some authors warn that due to ascites intra-abdominal hypertension could influence these values [13]. However, in our experience this is rarely the case. When aware of these additional limitations, changes in PPV during resuscitation in ACLF remain a useful tool.

More informative tests include thermodilution methods (static) or echocardiography (dynamic). Invasive “calibrated” monitoring devices like PICCO (Getinge, Solna, Sweden)

or a Swan Ganz catheter cannot be completely replaced by noninvasive or “uncalibrated”-derived measurements [16, 17]. Only transthoracic echocardiography offers a completely noninvasive alternative for holistic hemodynamic monitoring [1]. In the absence of adequate transthoracic imaging, some authors advocate the use of transesophageal echocardiography claiming a good safety profile in patients with varices up to grade 2 without recent upper gastro-intestinal bleeding [18]. We would however suggest being prudent in this population and would opt for a calibrated intravascular monitoring device in this situation such as PICCO.

### **Superimposed Shock Syndromes**

The two most prevalent types of shock in patients with cirrhosis are septic shock and hemorrhagic/hypovolemic shock [19, 20]. The correct differentiation guides the choice of fluid and timing for the introduction of vasopressors.

Sepsis is the most prevalent reason for admission to ICU of patients with cirrhosis and the index of suspicion of septic shock should always be high [19, 20]. Given the impaired immunity of these patients, early start of antibiotics is suggested when an infection is suspected. Antibiotic prophylaxis in variceal bleeds is highly recommended to avoid spontaneous bacterial peritonitis and subsequent deterioration of ACLF [21]. Fluid resuscitation remains a cornerstone in the management of septic shock as well as in patients with cirrhosis. However, with increasing severity of liver disease, a larger amount of the administered volume will pool in the splanchnic compartment [7]. Therefore, the impact on improved circulatory function will be less than in noncirrhotic patient populations with septic shock. Earlier start of vasopressors to restore perfusion pressure and avoid fluid overload seems appropriate.

Variceal bleeding is the second most common cause of ICU admission in this population, often presenting as a hemorrhagic shock [19, 20]. Aside from attaining source control, correction of coagulation and hemoglobin levels take priority. Red blood cell transfusion above a target of 7 g/dL and platelet transfusion above 50,000 platelets per microliter is recommended [22, 23]. More controversial and more relevant to fluid management is the optimization of coagulation.

While fresh frozen plasma contains all clotting factors, it takes 1 mL/kg of plasma to correct the PT for 1% [24]. This implies that generally a huge volume would be required to normalize coagulation, potentially fueling the variceal bleeding. Given this fact, we would recommend the administration of a much smaller volume of concentrated clotting factors. One should however be aware that specific concentrates lack several clotting factors and consider administration of some fresh frozen plasma to supplement these missing factors.

At the time of writing, there is no validated endpoint for restoration of coagulation. In cirrhosis production of both pro- and antithrombotic, fibrinogenic, and fibrinolytic factors are impaired due to liver injury. During compensated cirrhosis, a new equilibrium is established while PT and aPTT remain impaired [25]. The role of functional testing like TEG or ROTEM in this population during decompensation is still to be determined [26, 27].

## Choosing the Right Fluid

As in the general population crystalloids should be used as a first-line treatment in resuscitation. In particular balanced solutions should be preferred to “normal” saline given the known risk of acidosis and kidney failure due to hyperchloremic metabolic acidosis [28].

Although little data exist studying specifically patients with cirrhosis, it seems even more reasonable to opt for balanced solutions in this population given the fact that patients with cirrhosis are prone to developing additional renal failure. It is also known that “normal” saline worsens the formation of ascites and induces other extra-vascular fluid accumulation. It should be noted though that some authors advise against the use of specific balanced solutions such as Ringer lactate or acetate-containing solutions given the decreased metabolic clearance in patients with cirrhosis. Only limited data support these concerns and they appear to be only true for Ringer lactate [29].

As stated in previous chapters there is no place for starches anymore aside from perhaps a perioperative surgical bleed in a non-infectious patient. Resuscitation with starches in septic shock or during variceal bleeding is known to be associated with worsening of hepatic function and renal failure potentially increasing mortality [21].

Albumin has always been a molecule of interest in cirrhosis (Table 21.1). It is produced by the liver and has numerous functions including influencing oncotic pressure, binding, and transport of endogenous and exogenous substances, antioxidant, antithrombotic, immunomodulatory, anti-inflammatory properties, and endothelial stabilization [3]. Albumin is not recommended as a nutritional support. It should also not be administered to correct hypo-albuminemia per se in the absence of hypovolemia [3]. There are two well-established evidence-based indications for albumin in cirrhosis. First, in the prevention of postparacentesis circulatory dysfunction and prevention of renal vasoconstriction [4, 12]. The AASLD guidelines of 2012 suggest 6–8 g albumin per liter of ascites for paracentesis above 5 L [30]. Second, as part of the treatment of hepatorenal syndrome. A dose of 1 g/kg albumin for 2 days is advised [8]. To limit the fluid load, albumin 20% is preferred to lower concentrations.

The role of albumin during resuscitation is still heavily debated. The ALBIOS trial did not show a mortality benefit at 28 and 90 days after a resuscitation strategy including Albumin 20% administration in a general ICU population to correct serum albumin levels up to 30 g/L. On the other hand, post hoc subgroup analysis in patients with septic shock at enrollment did show a survival advantage in the group treated with albumin at 90 days

**Table 21.1** Common reasons for applying albumin. Well-studied indications are in green, indications with soft or conflicting evidence in orange, and wrongful indications in red

Indication albumin	Recommendation
Prevention post paracentesis renal vasoconstriction and circulatory dysfunction	6–8 g albumin per liter of ascites for paracentesis above 5 L
Treatment of a hepatorenal syndrome	1g/kg albumin for 2 days using albumin 20%
Spontaneous bacterial peritonitis to decrease renal impairment	1.5 g/kg on day one and 1 g/kg on day three
Volume resuscitation with albumin 4%	Potential mortality benefit in septic shock compared to saline 0.9%
Aiming at a serum albumin >30 g/L during resuscitation by applying Albumine 20%	Potential mortality benefit in septic shock
Scavenging bacterial products	Evidence is lacking
Keep albumin above 30 g/L in decompensated liver cirrhosis patients with persistent ascites despite diuretic therapy	Mortality benefit in ANSWER trial, but not in MACHT and ATTIRE
Nutritional support to keep albumine >30 g/L	Not recommended for this indication



[31]. The SAFE study compared the administration of fluid boluses of either saline 0.9% or albumin 4% in patients with sepsis. Again at first no difference was observed while a post hoc analysis again showed a trend toward mortality benefit in the subgroup with septic shock. However, all patients in the SAFE study were included after the initial resuscitation phase [32]. This implies there is currently still no evidence supporting the unique use of albumin as a resuscitation fluid.

Recent studies also suggest three potential additional indications for albumin infusion. First, it is claimed that albumin administration can restore the capacity of scavenging bacterial products [4]. Second, albumin administration decreased renal impairment and mortality in patients with spontaneous bacterial peritonitis. A dose of 1.5 g/kg on day one and 1 g/kg on day three is suggested [5]. Finally, the ANSWER trial showed a decreased 18-month mortality in decompensated patients with liver cirrhosis with persistent ascites despite diuretic therapy who were treated with IV albumin on a weekly basis [33]. However, the data of this ANSWER trial were not confirmed by the MACHT study that used lower albumin doses [34]. Furthermore, the recently published ATTIRE study showed no additional benefit of increasing the albumin level above 30 g/L using albumin 20% compared to usual care in patients hospitalized on the normal ward with decompensated liver cirrhosis [35].

At the time of writing a lot of promising studies are awaited. The PRECIOSA study tries to answer the question of which patients can benefit most from long term administration of albumin. This is an unanswered question given the conflicting evidence of the ASNWER trial, the MACHT study, and the ATTIRE study. Furthermore addition of plasmapheresis and DIALIVE (a new device aimed at removing damage associated molecular patterns (DAMPS) and pathogen associated molecular patterns (PAMPS)) are studied and could extend the appropriate indications of albumin [2].

## Acute Liver Failure (ALF)

### Special Considerations During Resuscitation

Many patients with acute liver failure are admitted to the ICU with intravascular volume depletion due to impaired oral intake caused by vomiting and/or encephalopathy. On the other hand, similar to the patient with ACLF, both volume depletion and liver congestion can lead to hypoxic hepatitis worsening residual liver function or inducing multiple organ failure. Specific to ALF, fluid overload can also increase intracranial hypertension and lead to brain edema and death [36].

As is the case with cirrhotic patients' PT and aPTT values correlate poorly with the bleeding risk given the fact that both procoagulant and anticoagulant factors are impaired. Also in ALF a new equilibrium is often attained [37, 38]. Usually, there is no need for aggressive correction of the deranged coagulation with blood products in order to reduce the bleeding risk. In addition, in the cases listed for liver transplant, the administration of plasma might compromise the eligibility of the patient for transplantation.

For similar reasons as in patients with ACLF, the diagnostic value of  $S_{cv}O_2$  or lactate clearance is diminished in ALF. Due to the hyperdynamic circulation, the  $S_{cv}O_2$  can be normal or slightly elevated in the presence of hypovolemia [12]. Given the decreased liver function, lactate clearance can be slower and could be more useful as a marker for liver injury [1].

More reliable for fluid management are dynamic maneuvers such as the passive leg raising test, thermodilution methods, pulse contour analysis, or echocardiography. However, during dynamic maneuvers one should be aware of the potential risk of increasing intracranial hypertension [1].

In a cohort study including 35 ALF patients SVV (obtained by PICCO), PPV, and respiratory change in peak left ventricular outflow tract velocity were evaluated for their accuracy to predict fluid responsiveness. In this study, SVV and echocardiographic parameters (inferior vena cava distensibility and LVOT) were poor predictors while PPV using a cut-off of 9% predicted fluid responsiveness with moderate accuracy (area under the receiver operating characteristics AUROC curve 0.75). Of note is that the accuracy of PPV was decreased (AUROC 0.72) in the presence of intra-abdominal hypertension (intra-abdominal pressure above 12 mmHg), which was present in 12 of the 15 patients in whom the abdominal pressure was measured [39]. Before transplantation for ALF, monitoring for intra-abdominal pressure seems to be indicated as rapid formation of ascites can occur with further compromise of the renal function. After transplantation, intra-abdominal hypertension has been described as an independent risk factor for renal failure [40].

### **Superimposed Shock Syndromes**

Patients with acute liver failure are often admitted in a dehydrated state due to impaired oral intake caused by vomiting and encephalopathy [36]. In addition, these patients develop an inflammatory response that is associated with systemic vasodilatation, capillary leak, and increase in insensible fluid loss aggravating this hypovolemia [1]. Given the immune dysfunction in acute liver failure, patients with ALF are at a high risk of combined septic/hypovolemic shock [1]. However, unmasking an infection can be daunting, since patients with acute liver failure are often hyperdynamic at baseline. In this case, it is preferred to introduce a vasopressor at an early stage rather than administering only fluids.

### **Choosing the Right Fluid**

An important complication in ALF is intracranial hypertension. Although hard data are lacking this implies hypotonic fluids could be harmful. To sustain sufficient cerebral perfusion pressure crystalloids are preferred above colloids. Given the decreased metabolizing capacity some authors warn also in ALF against the use of Ringers lactate and acetate-containing balanced solutions. Only limited data support these concerns and they appear to be only true for Ringer lactate [29]. The role of albumin has not been studied in ALF.

**Case Vignette**

A 45-year-old male with a CHILD C liver cirrhosis is admitted to the ICU following a variceal hemorrhage. During stabilization, he received 1 L of balanced crystalloids. The bleeding was stopped after variceal ligations. High-dose PPI and Terlipressin were initiated. At this time he is still hypotensive 80/35 mmHg (MAP 50 mmHg), and has marked peripheral edema, ascites, and an  $S_{cv}O_2$  of 75%. He feels peripheral cold and has mottled skin. When applying a passive leg raise his blood pressure increases to 90/40 (MAP 59 mmHg). The lactate level is 15 mmol/L.

**Conclusion**

Acute liver failure and acute on chronic liver failure are characterized by decreased systemic vascular resistance, increased cardiac output, central functional hypovolemia, increased arterial compliance, and peripheral vasodilatation. The importance of adequate fluid management in liver failure cannot be overstressed since both hypervolemia and hypovolemia can further compromise the residual liver function.

In cirrhosis, the average circulating blood volume is higher than in a healthy person and it is more unevenly distributed between the central and abdominal compartments with increasing severity of cirrhosis. Assessment of the intravascular volume status in these patients is not simple. Focus on the dynamics of change is important. Resuscitation techniques based on changes in lactate and  $S_{cv}O_2$  are less useful in patients with liver failure. The most robust dynamic tests include the use of thermodilution methods or echocardiography. The two most prevalent types of shock in patients with cirrhosis are septic shock and hemorrhagic shock. The correct differentiation guides the choice of fluid and timing for the introduction of vasopressors. Generally, strategies that reduce the need for large fluid volume administration via the earlier start of vasopressors or by administration of clotting factor concentrates seem more appropriate. As in the general population, balanced crystalloids should be used as a first-line treatment in resuscitation. There is some evidence suggesting against the use of ringer lactate. Albumin should also not be administered to correct hypo-albuminemia per se. The benefits of albumin are undeniable when applied in the setting of paracentesis or treatment of hepatorenal syndrome. The role of albumin during resuscitation is still heavily debated. At the time of writing promising studies are performed that will further impact the indications for albumin use in this population.

In acute liver failure many patients are admitted to the ICU with intravascular volume depletion due to impaired oral intake caused by vomiting and/or encephalopathy. Specific in ALF fluid overload can also increase intracranial hypertension and lead to brain edema and death. Dynamic maneuvers such as the passive leg raising test, thermodilution methods, pulse contour analysis, and echocardiography are most suitable to evaluate the fluid status and fluid responsiveness. Of these tests, the passive leg raising test could increase intracranial pressure. Although hard data are lacking hypotonic fluids could theoretically increase intracranial pressure in ALF and might be better avoided. Balanced crystalloids are generally the fluid of choice in ALF.

### Questions and Answers

Q1. Would you administer this patient an extra fluid bolus?

A1. Yes, the hypotensive state with positive passive leg raising test and cold mottled skin suggest an unresolved functional hypovolemia and fluid responsiveness. The high  $S_{cv}O_2$  can be attributed to the hyperdynamic circulation due to cirrhosis. Patients with cirrhosis can be intravascularly volume-depleted while showing signs of edema and ascites. However, the effect after fluid administration should be evaluated since administering too much fluid can aggravate ascites formation, edema, cardiac dysfunction, and even increase portal hypertension and restart gastro-intestinal bleeding.

Q2. What could help you decide between the administration of fluids or application of either inotropes or vasopressors.

A2. Advanced hemodynamic monitoring can be applied when in doubt. As a non-invasive technique, an echocardiography can be performed. Alternatively, calibrated invasive techniques such as PICCO or Swann Ganz can be applied.

Q3. What would you do if the lactate level decreased to only 10 mmol/L after 6 h despite your best efforts?

A3. Continue surveilling the patient, but wait patiently as long as lactate is decreasing and there are no signs of evolving end organ failure. Lactate could be a marker for the severity of liver disease rather than a marker of unresolved shock. A slowly decreasing lactate level should not necessarily lead to continued aggressive administration of fluids.

### Take Home Messages

- Liver cirrhosis is characterized by decreased systemic vascular resistance, increased cardiac output, central functional hypovolemia, increased arterial compliance, and peripheral vasodilatation.
- Over-resuscitation with IV fluids can aggravate ascites formation, edema, hyponatremia, cardiac dysfunction, and intra-abdominal hypertension. Fluid accumulation and overload can also increase portal hypertension and induce gastrointestinal bleeding in this population.
- Most static parameters are less useful in cirrhosis. Given the generally hyperdynamic state of cirrhotic patients  $S_{cv}O_2$  is less useful to assess cardiac output in cirrhosis. Likewise lactate clearance is often impaired due to liver damage.
- Advanced hemodynamic monitoring techniques favoring dynamics of change are valuable tools to optimize fluid therapy. These include the “calibrated” PICCO and Swann Ganz as well as transthoracic echocardiography.

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