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MINIREVIEWS

Intracranial pressure monitoring in the perioperative period of patients with acute liver failure undergoing orthotopic liver transplantation

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Abstract

Acute liver failure (ALF) may result in severe neurological complications caused by cerebral edema and elevated intracranial pressure (ICP). Multiple pathogenic mechanisms explain the elevated ICP, and newer hypotheses have been described. While invasive ICP monitoring (ICPM) may have a role in ALF management, these patients are typically coagulopathic and at risk for intracranial hemorrhage. ICPM is the subject of much debate, and significant heterogeneity exists in clinical practice regarding its use. Contemporary ICPM techniques and coagulopathy reversal strategies may be associated with a lower risk of hemor-rhage; however, most of the evidence is limited by its retrospective nature and relatively small sample size.

Key Words: Acute liver failure; Liver transplant; Hepatic encephalopathy; Intracranial hypertension; Brain edema

Core Tip: Despite its rare occurrence, acute liver failure generates academic interest from multiple disciplines because of its multiorgan involvement and high morbidity and mortality. Severe neurological complications may arise, requiring invasive monitoring with the potential risk of fatal intracranial bleeding. Newer strategies could decrease the risks while keeping the benefits.

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INTRODUCTION

Definition and incidence

Acute liver failure (ALF) is a rare syndrome caused by abrupt hepatocyte injury that can progress to a fatal outcome in days to weeks. The most widely accepted definition of ALF includes evidence of coagulopathy and any degree of mental alteration (*i.e.*, encephalopathy) within 26 wk in a patient without preexisting liver disease[1]. Classification according to etiology highlights associated prognostic value and disease-specific treatment. An alternative classification quantifies the interval between symptom onset and development of encephalopathy; hyperacute (0–7 d), acute (8–28 d), and subacute (1–3 mo)[2]. The incidence of ALF in the United States of America is thought to be close to 3000 cases per year[3].

Despite its rare occurrence, ALF generates academic interest from multiple disciplines because of its multiorgan involvement and high morbidity and mortality. The survival from ALF has improved in recent years through better knowledge of pathophysiology, advances in critical care management, and access to emergency liver transplantation (LT)[4].

Etiology, pathophysiology and multiorgan involvement

The pathophysiological process that leads to hepatocyte injury causes either direct toxic necrosis or immune apoptotic injury; the predominant cause for direct injury is acetaminophen toxicity, developing from hours to days[5]. The immune apoptotic injury is a slower injury process, led by hepatitis B infection/reactivation, autoimmune hepatitis, and drug-induced liver injury[6,7]. ALF is characterized by the development of hepatic encephalopathy (HE), and the loss of synthetic dysfunction in the form of coagulopathy. An elevated prothrombin time is a marker of synthetic dysfunction that occurs from the decrease in the vitamin K-dependent coagulation factors (II, VII, IX, X); prolongation of the INR more than 1.5 is considered a poor prognostic sign and a cornerstone of ALF diagnostic criteria.

The pathophysiology of ALF can be divided into primary liver injury specific to etiology and secondary multiorgan failure. The primary liver insult of acetaminophen-induced ALF has the best understood mechanism, namely glutathione depletion. The secondary multiorgan failure, severe systemic inflammation and microcirculatory alterations contribute to a clinical picture comparable to a distributive shock[8]. The vascular tone of the brain and kidneys are most vulnerable, leading to cerebral edema, encephalopathy, and functional renal failure[9].

NEUROLOGICAL DYSFUNCTION IN ALF

The central place of HE in the definition of ALF reflects its key prognostic impact, and its development reflects severely impaired liver function. A multiaxial definition of the syndromes of HE was developed for chronic liver disease by the International Society for Hepatic Encephalopathy and Nitrogen Metabolism based on the type of underlying hepatic abnormality, the time course, and severity of neurological manifestations[10]. The American and European Associations for the Study of Liver Diseases practice guidelines highlights the distinct features of HE in ALF and the association of HE with increased intracranial pressure (ICP)[11].

Cerebral edema and resulting intracranial hypertension (ICH) are the most severe neurological clinical manifestations in patients with ALF. In the past, cerebral edema was presumed to occur in up to 80% of patients with ALF. However, recent data from developed countries estimates a drop in the incidence to 20%-30%, probably due to earlier diagnosis and improved management[12].

Pathogenesis of brain edema in ALF

The pathogenesis of cerebral edema in ALF is complex and only partially understood, and its occurrence is related to the severity of encephalopathy. Cerebral edema is occasionally observed in patients with grade I-II encephalopathy; moreover,the risk of edema increases to 25% to 35% with progression to grade III, and 65% to 75% or more in patients reaching grade IV coma[13].

Potential contributing factors include cytotoxicity due to osmotic effects of ammonia, glutamine, and proinflammatory cytokines, vasogenic edema due to disruption of the blood-brain barrier with the rapid accumulation of low molecular substances, and the loss of the cerebral blood flow autoregulation.

Multiple studies support astrocyte swelling and cytotoxic edema as major contributors to cerebral edema in ALF[12,14,15]; the evidence is most compelling in the central role of ammonia causing astrocyte swelling. The ammonia-glutamine hypothesis has persisted over years, describing an excess of ammonia in the brain which is converted to glutamine with resulting osmotic effects on astrocytes. New studies have challenged this hypothesis, concluding that astrocyte swelling may not be the result of glutamine's direct osmotic effect; instead, a "Trojan horse" hypothesis is proposed in which glutamine may function as a carrier of ammonia into the mitochondria where its accumulation can lead to oxidative stress and ultimately cellular swelling[16]. Oxidative stress has been implicated as an important factor in the pathophysiology of ammonia-induced neurotoxicity through the formation of free radicals which may result in mitochondrial permeability transition[17].

Other studies have suggested that neuroinflammatory mediators, particularly proinflammatory cytokines such as the interleukins (IL)-1β and IL-6 and tumor necrosis factor-α, play an essential role in the development of brain edema and ICH[18,19]. Neuroinflammation is now widely considered the result of a direct interaction between microglia and ammonia. The released proinflammatory cytokines from activated microglia cells and ammonia appear to act synergistically to induce cytotoxic cerebral edema in which the blood-brain barrier is preserved.

Research combining brain imaging in the context of ALF demonstrates evidence of interstitial brain edema in addition to cytotoxic brain edema, implying the presence of vasogenic edema, in which the blood-brain barrier would be compromised[20,21]. Although a generalized breakdown of the blood-brain barrier cannot be demonstrated, some studies propose the "leaky" theory, in which there are subtle changes in the integrity of the tight junctions of the blood-brain barrier. The exact mechanism of how cytotoxic, vasogenic, and neuroinflammation interact to bring brain edema in ALF remains unknown.

The role of ICP monitoring in ALF

The Brain Trauma Foundation guidelines explicitly recommend ICP monitoring (ICPM) for patients with severe traumatic brain injury to minimize mortality[22]; however, recommendations for ICPM in patients with non-traumatic brain injury are lacking. The rationale for using monitors to measure the pressure inside the cranium in ALF considers the potential benefit of early identification and management of ICH. In addition, continuous ICP measurements contribute to the decision-making process for emergency LT; intraoperative ICPM facilitates active neurological management in the setting of rapid fluid shifts and hemodynamic instability.

Invasive ICPM remains the gold standard for the measurement of ICP[23], which may reveal occult elevations in ICP in comatose patients with ALF[24]. Despite the proposed benefits, invasive ICPM in this unique patient population raises concern due to the risk of life-threatening intracranial hemorrhage in the setting of coagulopathy.

Noninvasive ICPMs offer an alternative solution in this specific group of patients, employing techniques of optic nerve ultrasound and transcranial doppler. However, current evidence does not support its use to accurately identify patients with ICH. One study evaluated noninvasive ICPM techniques in comparison to the gold standard of invasive ICPM; the authors concluded that neither optic nerve ultrasound nor transcranial doppler pulsatility index correlated with the gold standard[25]. Another standard noninvasive option is cerebral computerized tomography, yet, evidence demonstrates this method's failure to consistently detect brain edema in patients with elevated ICP[23]. In addition, the complexity of intrahospital transport for critically ill patients should not be underestimated.

With invasive ICPMs identified as the most accurate modality to identify ICH in patients with ALF, several invasive options exist. Transducers may be placed in the brain parenchyma, ventricular system, epidural or subdural spaces. Epidural devices have lower complication rates than subdural or intraparenchymal monitors[26]. A ventricular system has the potential to be diagnostic and therapeutic as cerebrovascular fluid can be drained; however, intraventricular placement may be associated with severe and potentially fatal hemorrhage.

LT in ALF

With high-grade HE identified as an independent predictor of mortality in patients with ALF, LT is a potentially life-saving intervention[27]. Access to emergency LT has improved survival rates for patients that fulfill criteria for a poor prognosis. The King's College Criteria remains the most clinically useful prediction tool, with disease-specific modeling for paracetamol and non-paracetamol categories[28]. Post-LT outcomes in this population are high with one- and three-year patient survival rates reported as 91% and 90% respectively[28].

Consensus guidelines for ICP monitor use

A review of the current literature highlights the lack of consensus regarding the use of ICPM in patients with ALF. The Acute Liver Failure Study Group guidelines does not recommend the use of external ventricular devices to monitor ICP for all patients with ALF; however, they recognize that most centers will place ICPM in patients with advanced encephalopathy[29,30]. A survey of 24 centers in the United States of America demonstrated that a minority (approximately 30%) of centers utilized ICPM[31].

Invasive ICPM use in Europe is more prevalent with 55% of centers surveyed reporting use of this monitoring modality[26]. In both surveys, invasive ICPM was reserved for patients with advanced encephalopathy according to The West Haven criteria; the type of invasive monitor use was not specified. The American Association for the Study of the Liver recommends invasive ICPM in patients with ALF awaiting LT and in centers with expertise[28]; The European Association for the Study of the Liver recommends monitoring only in a select group of patients including those with advanced encephalopathy at risk of ICH, hyperammonemia, and renal or vasopressor support[32]. Table 1 summarizes the current large-society recommendations.

Robust data regarding the impact on long-term neurological consequences of cerebral edema and ICH in patients with ALF is scarce. Similarly, evidence reporting outcomes associated with the use of ICPM in this patient population is also lacking. Karvellas *et al*[33] reported a multicenter retrospective cohort study involving 140 patients managed with ICPM *vs* 489 controls without ICPM; the mortality at 21 d was not significantly different[33].

The incidence of spontaneous intracranial hemorrhage in ALF

The estimated risk of spontaneous intracranial hemorrhage in overt encephalopathy grade III and IV is 25%-35% and 65%-75% respectively[34]. The incidence of intracranial hemorrhage has decreased over many years. Bernal *et al*[4] reported a series of 3300 patients, in which intracranial hemorrhage occurred in more than 70% of patients on initial analysis with a dramatic reduction in incidence to only 20%, with a corresponding reduction in mortality, 20 years later[4]. The same author reported 29% incidence of intracranial hemorrhage in a series of more than 160 patients with overt encephalopathy[14]. The risk factors for intracranial hemorrhage include hyperacute presentation, younger age, and requirements of vasopressors or renal replacement therapy[14,35].

Risk of bleeding and outcomes from the use of invasive ICPM in ALF

The general incidence of hemorrhagic complications from ICPM is approximately 10%-20% with fatal hemorrhage reported in 1%-5% of patients[31,36]. The risk of intracranial bleeding is related to the type of device and location of the ICPM placement. Some authors claim a reduction in bleeding risk by a meticulous insertion technique and targeted peri-procedural transfusion (*e.g.*, recombinant factor VIIa prior to the placement of the ICPM)[37]. A literature search from 1992 to 2017 shows eleven studies reporting the use of ICPM in ALF;only four of these studies described an institutional protocol to correct the coagulopathy prior to the insertion of ICPM. Variable use of peri-procedural blood product transfusion was observed.

Another potential complication associated with ICPM insertion is infection. The general risk of infection is approximately 1%-20%[38]. To our knowledge, ALF patients have no associated increase in infection risk; however, data is limited. Multiple small case series demonstrated a low incidence of ICPM-related infections[24,37,39]. Reported rates of infection ranged from 0%-7%. A common practice to reduce infection risk is the administration of prophylactic intravenous antibiotics to cover the typical skin flora prior to ICPM placement.

TECHNICAL ASPECTS FOR ICPM

It is important to acknowledge that regardless of the transducer selected, the management of ICP should be guided by the cerebral perfusion pressure. The cerebral perfusion is estimated by the difference between the mean cerebral arterial pressure and the ICP. To ensure accurate measurement of cerebral arterial pressure, it is recommended that the arterial line transducer should be positioned at the external auditory meatus, level with the middle cranial fossa[40].

Interventions for managing brain edema and ICH in ALF are out of the scope of this article. However, standard measures are to maintain adequate sedation, head elevation at 30 degrees, target plasma sodium levels of 145 to 155 mEq/L, maintain normocapnia with a CO_2 of 35 mmHg, a plasma osmolarity of 320 mOsml/L, a mean arterial blood pressure of 75-80 mmHg, and temperature between 32-34 °C for 10-14 h in candidates for LT[41].

SINGLE-CENTER EXPERIENCE

Using the limited evidence and large-society guidelines, a protocol was developed and implemented to guide management of severe neurological consequences of ALF in our center. Integral to this document is the recommendation for the use of invasive ICPM in carefully selected patients. Protocol development engaged representatives from all multidisciplinary stakeholders including hepatology, anesthesia, critical care, and surgery. Explicit clinical criteria outlined patients appropriate for invasive ICPM use.

As outlined in this protocol, patients with high-grade HE (grade III and IV) in the context of ALF, with the possibility of recovery from medical intervention and/or LT, warrant ICPM insertion.

Table 1 Summary of recommendations for intracranial pressure monitor in patients with acute liver failure

Society	Recommendation	Quality of evidence
AASLD 2005[1]	ICPM is mainly considered for patients who are listed for transplantation. In the absence of ICPM, frequent evaluation for signs of intracranial hypertension is needed to identify early evidence of uncal herniation	Evidence level III
AASLD Revised 2011[28]	The use of recombinant factor rVIIa may be considered	NA
ALSFG 2007[30]	Insufficient data to recommend ICPM placement in all patients with ALF. However, most members of the ALFSG place ICPM in patients with advanced (stage III/IV) hepatic encephalopathy	NA
EASL 2017 [32]	ICPM should be considered in a highly selected subgroup of patients, who have progressed to grade 3 or 4 coma, are intubated and ventilated and deemed at high risk of intracranial hemorrhage, based on the presence of more than one of the following variables: (1) Young patients with hyperacute or acute presentations; (2) ammonia level over 150–200 lmol/L that does not drop with initial treatment interventions (RRT and fluids); (3) renal impairment; and (4) vasopressor support (> 0.1 lg/kg/min)	(Evidence level II-3, grade of Recommendation 1)

AASLD: American Association for the Study of Liver Diseases; ALSFG: United States Acute Liver Failure Study Group; EASL: European Association for the study of the Liver; ICPM: Intracranial pressure monitor; RRT: Renal replacement therapy; ALF: Acute liver failure; rFVIIa: Recombinant factor VIIa; NA: Not available.

Due to the risk of severe brain edema, eventually obliterating the ventricles, our neurosurgical team is reluctant to use external ventricular devices, in addition to the increased risk of periprocedural hemorrhage. In our protocol, we use the Codman Microsensor™ intraparenchymal monitor, which measures ICP *via* a strain gauge microchipat the catheter's tip. Pressure is reflected as an electrical voltage transmitted to the proximal end of the catheter through nylon-encapsulated copper wires. The proximal end of the catheter is connected to the Codman Express™ monitor, which displays the ICP value. Following baseline brain imaging andbefore insertion, the ICPMis zeroed at atmospheric pressure; after insertion, opening pressure is determined, and real-time display and longitudinal recordings are obtained.

To minimize the risk of ICPM-associated hemorrhage, coagulation correction is frequently undertaken prior to device insertion. It is generally accepted that conventional coagulation tests (*e.g.*, INR and platelet count) provide a limited perspective of *in vivo* clot formation in patients with liver disease. With the growing popularity of viscoelastic testing (VET) to guide coagulation management during LT, VET has been proposed as a more comprehensive tool to facilitate invasive procedures such as ICPM insertion. Our protocol utilizes a combined approach of VET and conventional laboratory testing. In our early institutional experience, directed administration of recombinant factor VIIa, fibrinogen, platelets, and desmopressin has enabled intraparenchymal monitor insertion, maintenance, and removal without hemorrhagic complications.

CONCLUSION

Future prospective studies are necessary to address the existing gaps in knowledge outlined in this review. Given the rarity of ALF, and the broad spectrum of presentation, it is unlikely that single-center studies will provide robust evidence. Most of the protocols currently in place, including the one used in our center, are derived from retrospective observational and expert consensus statements. It is paramount to define the specific population of patients in which insertion of ICPMs changes outcomes and standardized transfusion protocols to minimize the associated risk of bleeding.

FOOTNOTES

Author contributions: All authors contribute to the review of literature, first author Mendoza Vasquez LE wrote the initial manuscript that was extensively reviewed and changed by the other two authors.

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