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Intra-abdominal hypertension and abdominal compartment syndrome in acute pancreatitis, hepato-pancreato-biliary operations and liver transplantation

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Abstract

Intra-abdominal hypertension, even preceding the onset of abdominal compartment syndrome, is still recognized as an adverse prognostic factor. Unfortunately, most of the current supporting evidence within the critical care environment remains observational in nature. In acute pancreatitis, an active role for intra-abdominal hypertension early in the disease process follows a strong intuitive basis, and it is corroborated by preliminary evidence from animal models. Additional studies are needed to better characterize the optimal fluid resuscitation strategy, as well as the importance of intra-abdominal hypertension as an early therapeutic target. All critically ill patients with acute pancreatitis should be considered for routine intra-abdominal pressure monitoring. The prevalence and clinical relevance of intra-abdominal hypertension after elective major abdominal operations are underestimated in the literature. Hepato-pancreato-biliary surgery and liver transplantation represent high-risk surgical subspecialties, and routine intra-abdominal hypertension risk assessment to indicate postoperative intra-abdominal pressure monitoring can be recommended. Conservative management of intra-abdominal hypertension should be promptly initiated upon diagnosis. Although abdominal catheter drainage and decompressive laparotomy may be required in refractory cases based on expert clinical judgment, precise indications and timing are still unclear. Implementation of institutional protocols based on the Abdominal Compartment Society reference standards is crucial to optimize both clinical management and research in this evolving area.

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The effects of elevated intra-abdominal pressure (IAP) on renal and respiratory dysfunction have been well documented in animal models since the beginning of the twentieth century. Clinical interest in this topic, however, only evolved during the second half of the century, after major advances in critical care, the management of omphalocele, abdominal trauma, along with the development of laparoscopy [1]. During the 1970s and 1980s, refined characterization of the pathophysiology of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) leveraged the publication of multiple case-series and clinical reviews. The need for uniform definitions and standard recommendations to engage collaborative research efforts in this field became strongly apparent.

In 2004, the former World Society of Abdominal Compartment Syndrome (WSACS) — now known as the Abdominal Compartment Society — was founded to promote international collaborations for education and research. Since then, consensus definitions, standard recommendations and management algorithms have been published and updated by a panel of experts [2–5]. The WSACS recommends the indirect measurement of the IAP via a bladder catheter; with the IAP measured with a supine patient, by the end of expiration and in the absence of abdominal contractions. A maximum of 25 mL of saline (in adults) is instilled through a urinary catheter and the pressure reading is zeroed at the mid-axillary level. IAH is defined by a sustained or repeated

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pathological elevation in intra-abdominal pressure (IAP) ≥ 12 mm Hg while the diagnostic threshold for ACS relies on sustained IAP > 20 mm Hg in association with a new organ dysfunction. These concepts were widely accepted by the scientific community allowing for substantial improvement in the volume and quality of publications.

Unfortunately, studies still demonstrate a low awareness of practicing physicians with regard to WSACS reference standards, as well as a common underestimation of IAH prevalence. Lack of familiarity with current recommendations and a negative correlation with the institutional prevalence of IAH were demonstrated in a survey including 37 intensivists from three teaching hospitals and one regional emergency medical center in Southwestern China [6]. In another recent survey of over 2,000 members of the WSACS and three endorsing critical care societies, although 85% of respondents reported knowledge of IAP, IAH, and ACS concepts, only 28% were aware of their definitions. Moreover, reported IAP cut-off criteria for IAH and ACS were most often higher than recommended standards [7]. Proper dissemination of uniform definitions and recommendations remains a major challenge.

Additionally, improvements in survival with the application of evidence-based management guidelines also closely rely on timely assessment and intervention. As a result, routine monitoring of IAP based on risk assessment for IAH has been advocated in order to allow for early diagnosis in a variety of scenarios besides classic trauma, emergency surgery and critically ill medical patients with overt ACS. Early diagnosis in an expanding diversity of presenting scenarios puts greater emphasis on expert management of the underlying disease process. This has increased both the complexity of clinical judgment and the importance of actively involving different subspecialties.

Hepatopancreatobiliary (HPB) surgery has emerged over the past three decades as an expanding surgical subspecialty that manages not only severe acute pancreatitis, but also some of our most complex abdominal operations. The high risk of major abdominal morbidity in this area demands a precise understanding of the pathophysiology, diagnosis and management principles of IAH/ACS. This publication aims to provide a state-of-the-art review of IAH/ACS in the scope of HPB surgery. It includes a detailed discussion about the pathophysiology, prognostic relevance and management of IAH/ACS in acute pancreatitis, as well as distinctive aspects related to elective HPB operations and liver transplantation.

ACUTE PANCREATITIS

Most cases of acute pancreatitis (80%) are characterized by self-limited (peri)pancreatic inflammation and clinical resolution within the first week of conservative management. Disease severity increases with exacerbation of the inflammatory response, local complications and organ failure. Severe acute pancreatitis is defined by persistent organ failure (> 48 h) and is associated with high mortality (35% to 67%) [8–10].

Mortality is typically determined by multiple organ dysfunction syndrome (MODS) and follows a bimodal distribution. Early mortality (up to 60% of all deaths) [11] results from an overactive systemic inflammatory response syndrome (SIRS) over the first week, while the remainder deaths are secondary to local complications, infection and sepsis after the second week. Characterization of these patterns of severity progression, have not yet been translated into effective prediction of severe disease upon patient admission based on traditional parameters [12–14].

ACS has been clearly associated with worse outcomes in acute pancreatitis [15]. Over the past decade, most studies depicted ACS as a terminal prognostic event, with a reported incidence of IAH and ACS in severe disease of 59% to 84% and 25% to 56% respectively [15–21]. Wide variability within the literature has resulted from small study population sizes, heterogeneous diagnostic criteria, as well as inconsistent methodology for IAP measurement. Moreover, further imprecision in the interpretation of these estimates arose from biased case selection and changes in the Atlanta classification system for acute pancreatitis in 2012 [22].

More recently, an alternative landscape has emerged from the evolving idea of IAH as an early factor in the pathogenesis of severe disease. In a recent prospective cohort including 218 patients with acute pancreatitis [23], IAH was diagnosed in 17% of cases, most often on admission (30/36 patients). The diagnosis of IAH was a common sentinel event for clinical deterioration, and was associated with greater 30-day mortality (37% vs. 2%, *P*-value < 0.01). A mean IAP greater than 9 mm Hg was a good predictor of increased mortality (sensitivity 86%; specificity 87%; area under the ROC curve 0.81). In addition, Smit *et al.* [24] retrospectively analyzed a selected series of patients with severe acute pancreatitis using a Foley catheter. All 29 patients evolved with IAH, while 45% of them developed ACS, typically within the first week. Most cases of ACS (10/13) underwent decompressive laparotomy and presented with intra-abdominal ischemia (8/10).

Although a causative association between IAH and severe disease is still controversial, it is clearly supported by evidence from animal studies. Models of acute pancreatitis reveal IAP elevation as early as 6 hours post-induction [25], and incorporation of IAH into the severe acute pancreatitis model for 12 hours demonstrates detrimental effects on global hemodynamics, oxygenation and organ function [26]. More importantly, early decompression helped partially reverse some of these negative effects [27, 28]. Despite the

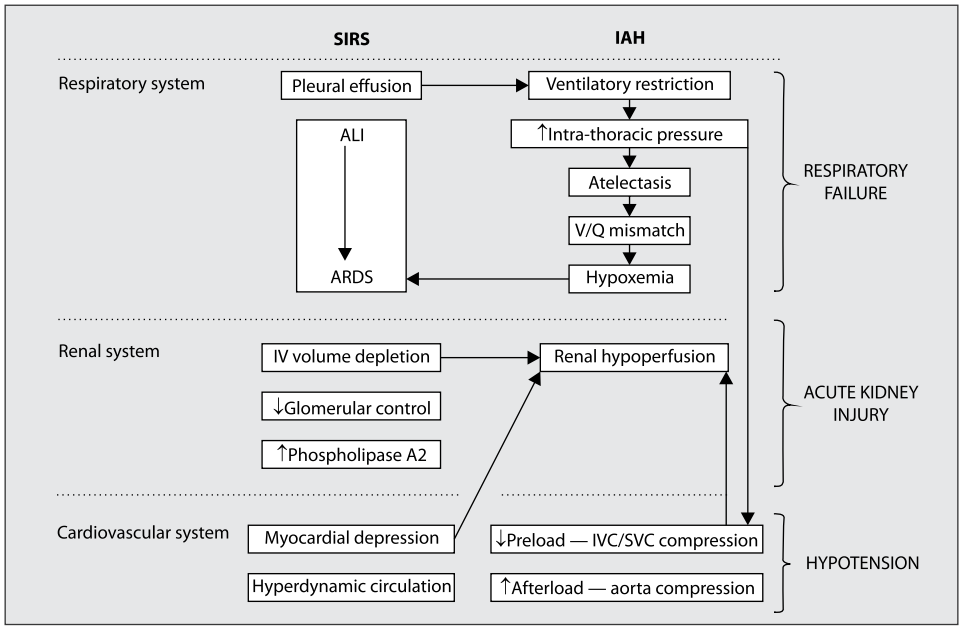

Figure 1. Schematic diagram of the roles of SIRS and IAH in pathogenesis of MODS in acute pancreatitis; abbreviations in the text

absence of definitive evidence of causality, the available data clearly support the recommendation of both routine IAP monitoring and early management of IAH in severe acute pancreatitis.

PATHOPHYSIOLOGY

The severity of acute pancreatitis and escalation of IAP are directly related to the magnitude of the inflammatory cascade within the pancreas. However, while the pathophysiology of severe disease directly relates to systemic effects of inflammatory mediators, IAP elevation results from both local and systemic events. IAH represents an inability to accommodate an increasing volume of abdominal contents — both intra-luminal (ileus, gastric and colonic distension) and extra-luminal (visceral and retroperitoneal edema, peripancreatic collections, and ascites). In this mechanic model, decreased compliance due to abdominal wall oedema accelerates IAP rise in response to increasing abdominal content.

Over the first week, an overactive systemic inflammatory response typically causes diffuse lung inflammation in the spectrum of acute lung injury and acute respiratory distress syndrome. Progression to MODS is characterized by cumulative involvement of the renal (largely due to intravascular volume depletion and impaired glomerular control) and cardiovascular systems (hyperdynamic circulatory state with myocardial depression).

In this setting, IAH directly affects the respiratory and renal systems by restricting ventilatory incursions and decreasing abdominal perfusion pressure [29]. Moreover, cardiac function is impaired by decreased pre-load (compression of the inferior vena cava) and increased systemic vascular resistance (elevated intra-thoracic and intra-abdominal pressures). As secondary events, hypoxemia due to ventilation-perfusion mismatch and bowel ischemia potentiate systemic inflammatory response syndrome (SIRS) and complete this vicious cycle (Fig. 1).

After the first week, clinical deterioration is usually determined via a secondary insult (e.g. local complications, infection, thromboembolic events). Prolonged invasive support (e.g. central-line, mechanic ventilation, urinary catheter) and development of peri-pancreatic collections and necrosis increases the risk of infectious complications. Bacterial translocation is the most common source of necrosis infection, with gut barrier dysfunction being reported in up to 59% of patients with acute pancreatitis [29]. Late onset ACS may result from intra-abdominal infection, haemorrhage or ischemic complications [15, 30], and portends an even worse prognosis [23].

FLUID THERAPY

Disturbance of water homeostasis is a central event in the pathogenesis of severe acute pancreatitis. It involves the inflammatory process, hemodynamics, and abdominal compartment mechanics and results from increased capillary permeability, third-spacing and intra-vascular volume depletion. No pharmacotherapy is available to directly address this intricate biological system. Even though patients often present early in the disease process, remediation of intra-vascular volume depletion is still the basis of emergency treatment.

Aggressive fluid resuscitation has been supported by early evidence of increased risk of pancreatic necrosis and organ failure with persistent hemoconcentration at 24 h after admission [31]. Subsequent studies comparing fluid resuscitation strategies have demonstrated decreased risk of SIRS, organ failure and mortality in patients receiving more intensive regimens [32–34]. Recommendation for aggressive fluid therapy — largely based on small retrospective studies — has been further supported by evidence extrapolated from patients with sepsis [35].

More recent prospective studies however, have shown an increased risk of local complications and persistent organ failure with more than 4.1 L of intravenous fluid during the initial 24 h [36], as well as a higher incidence and earlier onset of sepsis, and greater mortality with rapid hemodilution (hematocrit < 35%) within 24 h of admission, in patients with severe acute pancreatitis [37]. The increased risk of IAH with large volume fluid resuscitation has also been demonstrated in a recent systematic review [38]. The possibility of reverse causality bias seriously compromises any definitive conclusions based on observational studies. In two randomized controlled trials from the same institution, aggressive fluid therapy was further incriminated for worse outcomes in terms of APACHE II scores, rates of mechanical ventilation, risk of sepsis, ACS and mortality [39, 40]. However, a systematic review recently defended the equipoise of evidence surrounding aggressive fluid therapy and the need for additional randomized controlled studies [41]. While better evidence awaits, judicious volume expansion and attention to the potential harms of aggressive fluid therapy are recommended.

The role of early goal-directed fluid therapy in acute pancreatitis has been investigated in small and sparse studies. Wu *et al.* [29] randomly assigned 40 patients with acute pancreatitis to either goal-directed fluid therapy (based on blood urea nitrogen levels) or standard fluid therapy. No significant decrease in the incidence of SIRS or C reactive protein blood levels were demonstrated after 24 hours. A more comprehensive protocol was recommended by Reddy *et al.* [42] for the medical management of post-ERCP pancreatitis with early fluid expansion guided by vital signs and hematocrit. Less severe disease, shorter hospital length-of-stay, decreased use of antibiotics and computed tomography imaging were retrospectively demonstrated in 32 patients managed by their protocol. It is interesting to notice, however, that the protocol also included specific standard recommendations for admission, analgesia, nutritional support, imaging and antibiotic therapy; moreover, specific reasons for the management of 13 patients outside the protocol were not reported. Finally, a retrospective analysis of the fluid resuscitation profiles of 32 patients with acute pancreatitis revealed the poor ability of central venous pressure (CVP) to reflect intravascular volume or to follow variations in cardiac index [43]. Non-survivors presented significantly higher CVP values and received less crystalloid and more vasopressors. Additional blunting of pre-load parameters is a specific concern in patients with IAH [44].

MANAGEMENT

Routine monitoring of IAP and early management of IAH are recommended in all critically ill patients with acute pancreatitis. Despite the lack of proper validation, these recommendations are strongly supported (GRADE 1C) by the high prevalence of IAH in this patient population [38, 45], the simplicity and low cost of intravesical IAP measurement, along with the unreliability of clinical examinations to detect IAP elevation [44]. This strategy also offers additional lead-time to potentially prevent adverse outcomes associated with sustained IAH.

Initial management of IAH/ACS in patients with acute pancreatitis is not different from that of other etiologies, while conservative measures should be promptly considered upon diagnosis (Table 1). Adequate analgesia, neuromuscular blockade and mechanical ventilation may help increase abdominal compliance. Gastrointestinal distension can be addressed by utilization of promotility agents and nasogastric and colonic decompression. Although a positive fluid balance should be avoided, the role of adjunctive measures to relieve the oedematous state (diuretics, albumin, and renal replacement therapy) still remain unclear. Drainage of intra-abdominal fluid collections is the initial invasive procedure to consider in refractory cases amenable to percutaneous intervention [46].

In trauma patients, the placement of a peritoneal lavage catheter in a series of 12 patients with ACS — defined by IAP > 20 mm Hg or abdominal perfusion pressure < 50 mm Hg — was followed by an average decrease of IAP of 8.0 mm Hg ($P = 0.01$), with only 2 out of 12 patients requiring surgery [47]. However, more rigorous characterization of its impact on clinical outcomes is still needed, while some distinct clinical aspects of acute pancreatitis should be considered. The high incidence of IAH (60% to 80%) early in the course of severe acute pancreatitis [30] is largely secondary to retroperitoneal oedema and gastrointestinal distension. Considerable amounts of ascites amenable to percutaneous drainage may not be present, while percutaneous intervention on evolving peri-pancreatic fluid collections are often ineffective and associated with increased risk of secondary infection. The indication of more aggressive management of sustained IAH in the setting of severe systemic inflammation and progressive organ failure thus represents a particularly challenging scenario.

One clinical trial [48] reported improved abdominal pain control and hospitalization time in patients with acute pan-

Table 1. Non-operative measures to consider during initial management of intra-abdominal hypertension

Evacuate intra-abdominal content	
Intra-luminal	Nasogastric tube
	Minimize/discontinue enteral nutrition
	Promotility drugs
	Rectal tube
	Enemas
	Colonoscopic decompression
Extra-luminal	Paracentesis
	Percutaneous drainage of fluid collections
Improve abdominal compliance	
	Analgesia
	Sedation
	Neuromuscular blockade
	Remove constrictive dressings
	Reverse *Trendelenberg* position

creatitis randomized to receive abdominal catheter drainage as compared to conservative measures alone. Interestingly, a mortality decrease from 20.7% to 10% in the intervention group was not statistically significant, while primary study outcomes were not defined in the manuscript raising concerns about sample size estimation. Drainage volume correlated to intra-abdominal pressure which, in turn, correlated to the APACHE II score and hospital length-of-stay. Interim analysis of another randomized clinical trial comparing percutaneous drainage to decompressive laparotomy for ACS in severe acute pancreatitis suggested lower incidence of sepsis and mortality in the drainage group [49]. However, a systematic review of cohorts reporting management of ACS in severe acute pancreatitis [50] identified seven studies with a total of 271 cases of acute pancreatitis, and 103 cases of ACS (38%). Most patients with ACS (87 patients) received an initial invasive intervention, most commonly surgical decompression (76 patients). Upfront catheter drainage was performed in only 11 patients, while additional surgical intervention was required in 8 of them. Although the limited clinical utility of catheter drainage for ACS in acute pancreatitis was therefore suggested, the heterogeneity and moderate-to-low methodological quality of the included studies was also highlighted.

A retrospective series of ACS in severe acute pancreatitis also indicates decreased mortality in patients undergoing early operation. Mentula *et al.* [51] analysed 26 cases of ACS in severe acute pancreatitis treated with surgical decompression in a tertiary care hospital [51]. Decreased mortality was demonstrated with surgery within 4 days from disease onset (18% vs. 100%). In another retrospective cohort including 45 cases of severe acute pancreatitis admitted to the ICU [52], 16 patients required surgical decompression. On average, surgery was performed 3.1 hours after the diagnosis of ACS, while mortality in this group was not significantly different from cases not requiring surgery (24% vs. 25%, $P = 0.9$).

Although a full-thickness midline laparotomy is the most commonly utilized incision, alternative approaches have been proposed [53–55]. Early retroperitoneal debridement finds no support in the literature, and it has actually been associated with a high risk of postoperative haemorrhage and mortality [16]. Utilization of negative pressure wound therapy, and attempts to obtain early or, at least, same-hospitalization fascia closure have been recommended [4].

HPB OPERATIONS

Similar to other complex abdominal operations, the clinical relevance of IAH in HPB surgery is better demonstrated in cases with multiple postoperative complications, such as haemorrhage, anastomotic leakages, infected intra-abdominal collections and pronounced gastrointestinal distension. In these cases, elevated IAH acts as a "second-hit" [56] to further decrease visceral perfusion in the already hypotensive patient with visceral vasoconstriction. No specific distinction is made on the management of overt ACS in HPB surgery; IAP monitoring is recommended in critically ill patients, and management should follow the same general principles and standard recommendations of other cases. The prognostic value of isolated IAH during an otherwise uneventful recovery also remains unclear.

In a study by Scollay *et al.* [57], patients undergoing major abdominal operations (duration > 2 h, and expected blood loss > 500 mL) were enrolled in a prospective protocol for 72 h IAP assessment, while measurement was performed according to WSACS recommendations. From a total of 42 major abdominal surgeries (including 29 HPB operations), IAH was diagnosed in 5 cases (12%), two of which corresponded to HBP surgeries (1 Whipple procedure and 1 hepatectomy). Although the non-IAH group resumed an oral diet on average 4 days earlier than IAH group (6 vs. 10 days, $P = 0.017$), no other significant differences were found between the two groups in terms of acute kidney injury, duration of ventilatory support, hospital length-of-stay and operative severity scores. The small study population size, however, seriously undermines definitive conclusions.

Although additional literature concerning ACS in HPB surgery is limited to sparse case reports, an important aspect of abdominal compartment mechanics has been depicted in two publications. Nissen *et al.* [58] reported a case of

fulminant hepatic failure due to a subcapsular hematoma caused by a liver biopsy, three years after a liver transplant. In this case, a thickened fibrotic liver capsule and peri-hepatic adhesions confined a 15 cm large subcapsular hematoma with an ischemia-prone transplanted liver within a small abdominal sub-compartment, with this creating a greater than 90% parenchyma necrosis. The clinical importance of non-hydrostatic pressure gradients due to shearing forces within the abdominal cavity was further explored in a case of delayed post-hepatectomy haemorrhage recently reported by the authors of this review. Limited hemoperitoneum (less than 2 liters) was restricted to the upper abdomen by severe lower abdominal adhesions, while its detrimental compressive effects on the diaphragm and renal perfusion were not reliably transmitted to the intravesical pressure probe by the interposed fixed viscera [59]. In this illustrative case, the terminology "abdominal *intra*-compartment syndrome" was proposed.

LIVER TRANSPLANTATION

Even for non-complicated elective cases, liver transplantation involves multiple risk factors for postoperative IAH. Abdominal content volume can significantly increase due to the oedematous state associated with SIRS and the massive infusion of fluids and blood products, the intra-peritoneal accumulation of ascites and blood, gastrointestinal distension, as well as visceral congestion secondary to portal vein clamping. At the same time, extensive surgical incisions and inadequate analgesia can further compromise abdominal compliance. These events are expected early in the postoperative course, typically during the initial 72 hours.

In a consecutive series of 108 liver transplants [60], average post-operative IAP was 21.5 mm Hg, while 79% of cases presented IAP > 18 mm Hg as assessed every 8 hours during the initial three postoperative days. This study predates the publication of WSACS standard recommendations, and IAP readings were obtained after infusion of 100 ml of saline in the urinary catheter. Although this likely overestimated pressure measures by current standards, high IAP (defined as sustained IAP > 25 mm Hg) was present in 31% of cases and was associated with significantly increased risk of renal failure (OR 5.4; 95% CIs 1.8–16.3). It is important to note that liver transplant patients are already predisposed to acute kidney injury secondary to preoperative renal dysfunction, intra-operative vena cava clamping, intra-operative hypotension, as well as polytransfusion. Moreover, post-operative renal failure has been reported in up to 95% of patients after liver transplantation [61] and has a major impact on short- and long-term operative outcomes [62]. Adverse outcomes with high IAP were also demonstrated in terms of prolonged ventilatory support (extubation within 3 h of 59% vs. 86%, $P < 0.01$), primary graft dysfunction (15% vs. 3%, $P < 0.05$) and mortality (15% vs. 4%, $P < 0.05$). Special concern with IAH is raised by the underlying critical risk of graft hypoperfusion due to loss of vascular auto-regulation, interrupted collateral vessels, as well as the absence of contra-lateral shunting in cases of split grafts.

In a prospective series of 18 paediatric liver transplant recipients [63], elevated IAP (IAP > 15 mm Hg for age under 8 years; IAP > 20 mm Hg thereafter) within 72 h from surgery was also associated with renal failure (50% vs. 0%, $P = 0.04$), hemodynamic instability (75% vs. 14%, $P = 0.04$), metabolic acidosis (100% vs. 14%, $P < 0.01$), as well as graft dysfunction ($P = 0.04$). In a study conducted by Shu *et al.* [64], an IAP greater than 20 mm Hg (assessed every 8 hours over 72 hours after liver transplantation) was associated with an increased incidence of acute kidney injury (45.8% vs. 7.9%, $P = 0.01$), in a retrospective series of 62 cases. More studies are required to fully appreciate the true impact of IAP monitoring on clinical outcomes. Although liver transplantation is not specifically mentioned in WSACS standard recommendations, all liver transplant patients present risk factors for IAH and, therefore, clearly conform to the current recommendation of routine IAP monitoring.

Particularly in paediatric transplantation, ACS may also result from large-for-size liver grafts. A graft-to-recipient weight ratio greater than 4% (particularly for recipients weighing less than 10 kg) should raise suspicion for liver graft/abdominal cavity size discrepancy; while it increases the risk for IAH, vascular complications and ischemia-reperfusion injury [65, 66]. Doppler ultrasonography should be performed upon fascia closure and followed closely thereafter, along with IAP monitoring.

CONCLUSIONS

Over the past three decades, the clinical relevance of IAH/ACS has expanded from a terminal condition in critically ill patients to a much more prevalent adverse prognostic factor in a variety of additional scenarios. Data supporting the association of IAH and worse clinical outcomes largely derives from observational studies. Causal inference, and thus the suitability of subclinical IAH as a therapeutic target, still requires improved characterization.

These uncertainties do not invalidate routine IAP monitoring in selected cases, or the early indication of conservative measures upon a diagnosis of IAH. Unfortunately, the precise indications and optimal timing of surgical decompression remain poorly characterized, and are better indicated in cases with overt ACS. HPB surgery and liver transplantation address complex abdominal pathologies and involve involve a high risk of intra-abdominal complications, and therefore IAH and ACS. IAH is particularly concerning in severe acute pancreatitis and patients with post-operative abdominal complications. In liver transplantation, studies

suggest a more incisive adverse effect of IAH, even in non-complicated elective operations. IAP monitoring based on IAH risk assessment should be routinely considered, while a protocolized approach based on WSACS definitions, recommendations and management guidelines represent a powerful strategy to optimize patient care by current standards and facilitate research in this evolving area.

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