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Review Article

THE INCIDENCE OF INTRAABDOMINAL HYPERTENSION (IAH) AND ABDOMINAL COMPARTMENT SYNDROME (ACS), FOLLOWING CARDIAC OPERATIONS

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A B S T R A C T

Aims: To focus through a related literature research and the authors' personal experience on the issue of gastrointestinal and abdominal compartment syndrome complications after cardiac surgery and Cardiopulmonary bypass(CPB).
Method: our experiences in general surgery, thoracic surgery, cardiac surgery, trauma surgery, anaesthesiology, intensive care unit and 104 articles review
Background: Most commonly, compartment syndrome involves the extremities; tissue edema below the fascial layer causes ischemia and eventual muscle necrosis. For the last 20 years, there has been more awareness among surgeons and intensivists of Abdominal compartment syndrome (ACS) being a distinct disease entity, but still widespread ignorance prevails. ACS can be acute, chronic and acute on chronic. Initial diagnosis is clinical, confirmed by measurement of IAP. ACS is a systemic syndrome involving derangement in cardiovascular haemodynamic, respiratory and renal functions because of sustained increase in (IAP) ending in multi-organ failure. It is a life-threatening emergency and requires prompt action and treatment. ACS occurs whenever increasing pressure within a confined anatomic space undermines the normal cellular functions of the tissues contained within that space.
Conclusion: ACS should be suspected in all critically ill patients, particularly those on ventilatory support in intensive care units who are haemodynamically not improving despite adequate resuscitation.Patients with long-time cardiopulmonary bypass with postoperative heart failure , with high dose of catecholamine are under high risk ACS. Decompressive laparotomy is the mainstay of treatment if the patient is to be saved from multiorgan failure and death despite which mortality is high.

INTRODUCTION

ACS occurs whenever increasing pressure within a confined anatomic space undermines the normal cellular functions of the tissues contained within that space.1,2

The peritoneal cavity is another confined anatomic space. Although the notion is not new, trauma clinicians are becoming more aware that increased pressure within the abdomen impairs organ function.3-5

This condition is known as intra-abdominal hypertension and can lead to the development of ACS, with potentially devastating consequences.5-7

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Etiology and Pathophysiology

Any abnormality that elevates the pressure within the abdominal cavity can induce intra-abdominal hypertension. In some situations, such as acute pancreatitis or ruptured abdominal aortic aneurysm, retroperitoneal processes are potential causes.3

Ileus, mechanical obstruction of the bowel, and abdominal growths can all be precipitating events.4 However, blunt abdominal trauma with intra-abdominal bleeding from splenic, hepatic, and mesenteric injuries is the most common cause of intra-abdominal hypertension.4,5

Surgical placement of abdominal packing to control hemorrhage may also increase pressure within the peritoneal space.8 Finally, bowel distention, because of hypovolemic shock and massive volume replacement, is an important cause of intraabdominal hypertension, and subsequent ACS, in trauma patients.2,5,9

Table 1 Burch grading system for intra-abdominal hypertension and abdominal compartment syndrome. 9

Grade	Bladder pressure; cmH_2O	Recommendation
I	10–15	Maintain adequate intravascular volume
II	16–25	Maintain adequate intravascular volume and closely monitor
III	26–35	Consider decompression
IV	>35	Perform surgical decompression

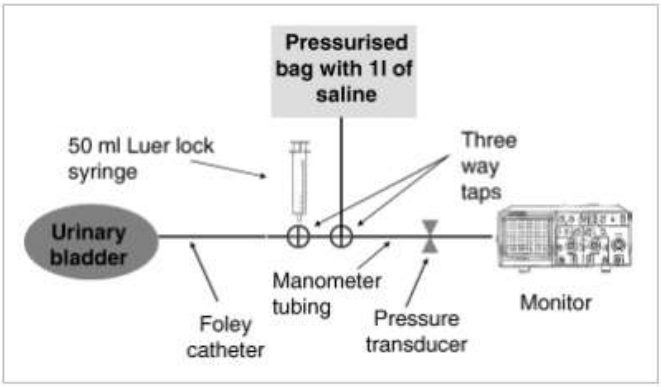

Fig 1 Modification of the Kron method for the intravesical measurement of intra-abdominal pressure.10

Intra-abdominal hypertension causes significant dysfunction of almost all organ systems. These physiological derangements become more pronounced and clinically significant when abdominal pressure is greater than 20 mmHg.

Intestinal Ischemia associated with ECC/CPB

A broader study was conducted by *Mangi et al*. 11 who included 8,709 heart-operated patients in their analysis. They calculated the overall incidence of gastrointestinal complications to be 0.53%, while intestinal ischemia presented in 31 of the patients (0.3 %).

The incidence of intestinal ischemia was estimated to be 0.3% by *Vassiliou et al*. in a series of 3,724 retrospectively analysed. Intestinal ischemia is a rare but usually devastating complication, which may affect any part of the small or large intestine. 12

It is attributed to hypoperfusion during extracorporeal circulation or perioperative haemorrhage, and of course to thromboembolic episodes occasionally influenced by heparin induced thrombocytopenia.

Acute cholecystitis post cardiac surgery associated with ECC/CPB

Passage et al. 13 performed a retrospective study on 16,576 patients and found an incidence of 0.03% for the calculous and a 0.08% for the acalculous form.

Overall mortality rate reached 20% for the calculous and 23% for the acalculous form; all lethal cases regarded patients submitted to surgery. *Yilmaz et al*. 14concluded to a comparable incidence of this complication to *Passage et al*. The 60% of patients with gallstones and 46.1% of patients with acalculous cholecystitis had to be operated.

Acute cholecystitis both calculous and acalculous cholecystitis, occurs more frequently and affects more morbid patients. It is attributed both to the systemic hypoperfusion and the systemic inflammatory response which is associated with extracorporeal circulation and is characterised, among others, by coagulation disorders, fluid removal towards the interstitial space, leukocyte count elevation and complement factors activation.

Acute pancreatitis associated with ECC/CPB, post cardiac surgery

Acute pancreatitis have same pathophysiological background with acalculous cholecystitis, rare but severe complication. Hypoperfusion owed to the application of the extracorporeal circulation or due to perioperative bleeding and the systemic inflammatory response are the basic causes of triggering acute pancreatitis with high mortality rate about 20%.

Mangi et al. 11 and *Vassiliou et al*. 12,13 reported 3 and 4 cases out of 3,724 and 8,709 patients, respectively.

Poirier et al. studied 11,405 patients and calculated the incidence of gastrointestinal complications to 1.2%, about 9% of which was attributed to postoperative pancreatitis. 15

Visceral hypoperfusionpost cardiac surgery, associated with ECC/CPB

Visceral hypoperfusion during the perioperative period is the main recognized factor that leads to systemic inflammatory response syndrome (SIRS) 16,17.

Liver failure associated with ECC/CPB

Ascione et al, estimated in their trials postoperative hepatocellular injury was increase with CPB time. Severe consequences of liver failure consist of hypo-albuminemia, malabsorption of hepatically metabolised drugs and malfunction of coagulation factors. 18

Main causes include side effects of anesthetic drugs and inotropic agents, Proper conservative treatment entails control of fluids and electrolytes and replenishment of nutritive and coagulation factors. Liver dysfunction in a broad sense, cases with significant liver enzymes' elevation of patients and can affect up to 25% of heart operated patients. Mechanical pressure from a low-placed vena cava inferior cannula for ECC, has also been reported cause of live failure.

Ileus associated with ECC/CPB, post cardiac surgery

A broader study was conducted by *Simic et al*. 19 paralytic ileus accented as one of the most frequent complications in a series of 3,312 patients. Perioperative fasting, the effect of anaesthetic drugs and patients' immobilisation during the first postoperative days, contribute to a temporary intestinal paralysis, which in most of cases regresses automatically after the initiation of alimentation.

The phenomenon of increased intra-abdominal pressures (IAP) and the resultant physiologic compromise were first described inthelate1800s. 20,21

More than a century later, this pathophysiology was recognized as an uncommon but morbid sequela of abdominal vascular surgery and was termed the "abdominal compartment syndrome" (ACS).22,23

Cardiovascular and ECC/CPB

Twelve separate retrospective studies since 1980 have documented high mortality (13% to 67%) associated with cardiopulmonary bypass-induced intraabdominal complications (IAC).24-35An intra-abdominal pressure >20 mmHg has a profound influence on the cardiovascular

system.20Haemodynamic compromise is due to complex alterations in preload, afterload and intrathoracic pressure. A decrease in cardiac output is observed due to an increase in afterload, in turn due to mechanical compression of the abdominal vascular beds, and a decrease in preload due to direct compression of the inferior vena cava and portal vein. Increased intra-abdominal pressure causes elevation of the diaphragm, which causes an increase in pleural and hence intrathoracic pressure 13, 20.

The decrease in preload is exacerbated by this increase in intrathoracic pressure, which not only hinders venous return but also causes cardiac compression, leading to a decrease in ventricular end-diastolic volume. A compensatory tachycardia is usually observed in response to the decrease in stroke volume.Cardiac surgery itself is a risk factor for intra-abdominal organ injury 9. According to data emerged from medical literature, the incidence of gastrointestinal complications ranges from 0.4% to 3.7%, with mortality ranging from 11% to 85%.36, 37, 38, 39These complications are frequently reported in high risk patients. The reported incidence varies from 0.2-14% 40-43. The mortality rate varies widely. It ranges from 13.9% to 100% in various series 44-46

CPB with membrane oxygenation

CPB with membrane oxygenationis known to induce a variety of metabolic, hematologic, and neurohumoral effects, most of which are reversible unless CPB is excessively prolonged. A common effect is post-CPB vasoconstriction, which occasionally necessitates vasodilator administration and is due to temporary elevations in several vasoactive substances, including catecholamines, serotonin.47see tab-2Complication of CPB organised according to organ Systems.

Cardiopulmonary bypass (CPB) can be complicated by a systemic inflammatory response characterized by profound vasodilation.48This vasodilatory shock syndrome, which is especially noted after extended CPB,49 is attributed to endothelial injury50and the release of cytokines and other inflammatory mediators.51Pressor catecholamines are commonly administered to support systemic arterial pressure in these cases,52but their effectiveness is limited by frequent catecholamine resistance 53and by significant toxic effects at high doses.54Alternative pressor agents could therefore be useful.

Cardiac surgeons have become more familiar with the operative management of various congenital and acquired malformations, and as improvements have been made in methods of preoperative preparation, the conduct of cardiopulmonary bypass, and in postoperative management. Because of this, direct mortality associated with operations upon the heart and great vessels has steadily declined.**9** As it is obvious from literature, complications secondary to the operation, usually occurring in the early postoperative period, and those complication may still represent serious threats to the patient's survival.51 These complications may involve disturbances in the contractile state, cardiac rhythm, electrolyte central nervous system, renal functions,liver functions, pulmonary, functions, gastrointestinal and hematopoietic.48

The incidence of gastrointestinal (GI) complications after cardiac surgery is low, but the mortality and morbidity, as well as the cost in terms of prolonged hospitalization, are all considerable. Gastrointestinal (GI) complications occurring after cardiac surgery are considered as "second line" complications due to their scarcity. Delayed recognition of these complications leads to a high incidence of morbidity and mortality, therefore prompt diagnosis and treatment are essential.44

Cardiac surgeons, ICU staff and nursing personnel are usually aware and suspicious of cardiac-related mishappenings after heart surgery; however, they often tend to underestimate potentially lethal manifestations involving the gastrointestinal track, or organs of the abdomen and subsequent complications because of their relative infrequency and the fact that they lack a "*visible connection*" to the primary target organ of the operation.55

These complications range from a simple, temporary paralytic ileus, to graver, highly perilous conditions, such as gastrointestinal hemorrhage, acalculous cholecystitis, acute pancreatitis, liver failure or mesenteric ischemia. A total mortality rate of 12-14.5% speaks for itself regarding the gravity of these nosologically entities 56-62.

Hypoperfusion and Hypoxia in CPB/ECC

Increased IAP leads to compromised blood flow through the gut and a decrease in venous return to the heart. Extreme elevation of IAP can progress to multiple-system organ failure. In fact, ACS has been defined as the "cardiovascular, pulmonary, renal, splanchnic, abdominal wall and intra-cranial disturbances resulting from elevated intraabdominal pressures" 63.

This value derives from the deleterious effects on renal, cardiac and gastrointestinal functions witnessed at IAP levels between 10 and 15 mmHg 64–68.

Previous studies in patients undergoing cardiopulmonary bypass (CPB) have documented gastric mucosal hypoperfusion and hypoxia.69

K. Ohri,et al 1997 In their study examine the influence of the CPB protocol on the adequacy of gut blood flow and oxygenation. Twenty-four patients were prospectively randomized into one of four CPB groups: nonpulsatile hypothermic (NP 28); pulsatile hypothermic (P 28); nonpulsatile normothermic (NP 37); and pulsatile normothermic (P 37). Gastric wall blood flow was assessed using laser Doppler flow measurement and gastric mucosal oxygenation (intramucosal pH), using tonometry**.**

K. Ohri,et al 1997 found that perfusion protocol can influence mucosal blood flow, but other overriding factors that operate during and after CPB act to cause mucosal hypoxia. These findings, particularly the timing of mucosal hypoxia, may have implications for centres contemplating early extubating or "fast tracking" of patients after CPB.

This syndrome is presently being seen with an increasing frequency and in a wide variety of critically ill patients.This escalation in occurrence has been closely paralleled by a significant increase in the number of publications related to intra-abdominal hypertension (IAH; defined as IAP >12mmHg) and ACS in recent years 70. (Fig-2)

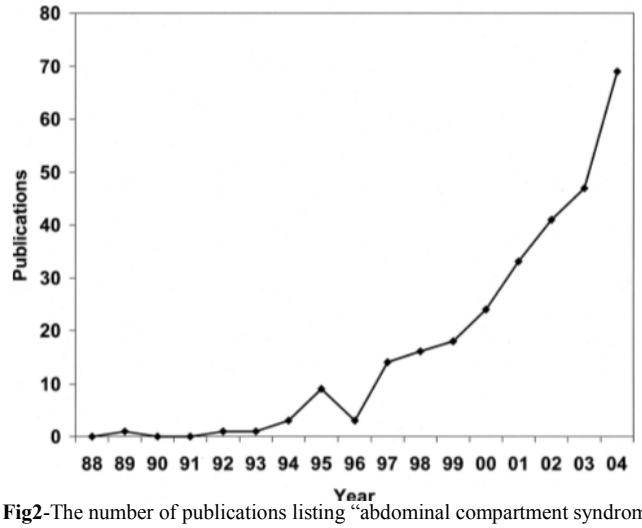

Fig2-The number of publications listing "abdominal compartment syndrome" or " intra-abdominal hypertension" within the PubMed search criteria (limited by English language in human subjects), by publication years 1988 to 2004.

Significant increases in IAP can be seen in the setting of *systemic inflammation* with *capillary leak* resulting in third spacing of fluid, followed by visceral, mesenteric, and *retroperitoneal edema*, and intra-abdominal free fluid.

Resuscitation in the setting of systemic inflammatory response syndrome (SIRS) resulting in IAH/ACS has been seen in a wide variety of disease processes including sepsis, pancreatitis, trauma, ischemia reperfusion injury, burns, peritonitis, bowel ischemia, and disseminated intravascular coagulation 71, 72–80.

Other clinical conditions, without a significant inflammatory component, can lead to increased IAP including ascites, pregnancy, hemorrhage, pneumoperitoneum, gastric dilation, ileus, bowel obstruction, organomegaly, tumours, repair of large hernias, gastroschisis, and omphaloceles 81–89.

Dalfinoa L etal; estimated in their trials, IAH develops in one-third of cardiac surgery patients and is strongly associated with higher baseline IAP values, higher central venous pressure, positive fluid balance, extracorporeal circulation, use of vasoactive drugs and AKI. Determinants of IAH should be accurately assessed before and after surgery, and patients presenting risk factors must be monitored properly during the perioperative period. 90

Emilie Richer Séguin et al in their study on 191 patients conclude that, more than half of the patients going under cardiac surgery have IAH. IAH is associated with obesity, higher filling pressure and pulmonary hypertension. Further studies with larger population would be required to determine the clinical impact of IAP measurements in cardiac surgery.91

Management

As ACS evolves, a positive feedback loop is created whereby increases in intra-abdominal pressure promote further elevations in intra-abdominal pressure.

Because the underlying problem is that the abdominal compartment has become too small, the immediate solution is to enlarge the size of the compartment. Consequently, current treatment of patients with ACS is urgent decompressive laparotomy, either in the operating room or at the

Tab 2 Complication of CPB organised according to organ Systems

Organ System	*Complication*	*Aetiology*
Respiratory	Left lower lobe' collapse	Phrenic nerve neuropraxia, due to cold slush cardioplegia Poor reinflation following restoration of circulation
	Pulmonary hypertension	Due to increased pulmonary vascular resistance (protamine)
	Acute Lung Injury	SIRS due to bypass circuit-associated complement activation
Cardiovascular	Myocardial stunning	Due to direct effects of cardiotomy and cardioplegia
	Myocardial infarction	Coronary graft ischaemia (air embolism)
	RV dysfunction	Due to pulmonary hypertension related to protamine
	Arrhythmias	Due to electrolyte disturbances and hypothermia
	Heart block	Due to hypothermia or direct conduction system trauma
	Systemic MODS	Hypoperfusion and end-organ ischaemia related to non-pulsatile flow and/or air/atheroma embolism
Neurological	-Stroke -Watershed infarcts - Neurocognitive impairment	All thought to be due to the sluggish low-flow state following the recommencement of bypass, as well as due to air emboli microemboli and possibly microemboli from the bypass circuit itself
Electrolytes and Endocrine	Hypothermia	Due to intra-operative cooling and delayed re-warming
	Hyperglycaemia	Due to hypothermia-related insulin resistance Due to circulating endogenous catecholamines
	Electrolyte derangement	Haemodilution
Renal	Post-op diuresis	"Cold diuresis" due to intra-operative cooling and delayed re-warming
	Post-op renal failure	Low flow, and thromboembolic events
Gastrointestinal	Splanchnic ischaemia	Low flow, and thromboembolic events
	Hepatic dysfunction	Low flow, and thromboembolic events
	Pancreatitis	Low flow, and thromboembolic events
Haematological	Coagulopathy	Due to consumption of clotting factors by the bypass circuit Due to residual anticoagulation Due to dilutional coagulopathy
	Platelet dysfunction	Due to antiplatelet agents, and due to the SIRS response
	Anaemia	Due to haemodilution and haemolysis
	Haemolysis	Due to mechanical destruction by the bypass pump, as well as due to MAHA and SIRS
Metabolic	Hypothermia	Due to intra-operative cooling and delayed re-warming
	Hyperglycaemia	Due to hypothermia-related insulin resistance Due to circulating endogenous catecholamines
Immune	Coagulation cascade activation	Due to blood contact with non-biological surfaces and blood-gas interface
	SIRS	Due to complement activation by circuit components
	Anaphylaxis	A reaction to protamine

Little A.G, Merril W.H. 2007, 2nd ed. Chapter 4 by Creswell and Karis ,Complications in cardiothoracic surgery: avoidance and treatment.

bedside.92,93,94 This procedure provides rapid relief of intra-abdominal hypertension.95 The peritoneal cavity is usually left open postoperatively, and the exposed contents are covered with a sterile dressing such as an iodine impregnated plastic adhesive drape.96,97 Closure of the incision is attempted when the swelling subsides.98

Unfortunately, once ACS is well advanced, surgical decompression may have serious adverse effects resulting in massive washout of anaerobic products, profound hypotension, and asystolic arrest.**99** In a recent survey, ***Mayberry et al*** assessed the knowledge level, experience, and current ACS management techniques of expert trauma surgeons in the United States. **100**

Nursing Care

Patients at high risk for ACS are those who have either undergone open heart procedures with long-time cardiopulmonary bypass and postoperative heart failure or experienced any event that predisposes them to increased intra-abdominal pressure.

Nurses caring for any of these patients must remain vigilant to prevent ACS by watching closely for the hallmark renal, pulmonary, cardiovascular, and neurological signs that indicate the development of this syndrome.101,93

Patients who have low urinary output and hypotensive shock unresponsive to fluid resuscitation or who have increased peak airway or intracranial pressures should be considered at risk for ACS.104However, patients with pancreatitis, bowel obstructions, abdominal aortic aneurysm rupture, pregnancy, large tumours, circumferential full-thickness burns of the abdomen, and those who have received massive volume resuscitation for whatever reason are also at risk for ACS. 2,93,18 The definitive assessment finding for ACS, however, is not a constellation of ambiguous clinical signs and symptoms but rather the presence of elevated intra-abdominal pressure. Animal studies in which a catheter was placed directly into the peritoneal cavity indicated that normal intra-abdominal pressure is atmospheric or even sub atmospheric (≤0 mm Hg).3-5Elevations in intra-abdominal pressure are classified as mild (10-20 mm Hg), moderate (20-40 mm Hg), and severe (>40 mm Hg).2 The exact level at which intraabdominal hypertension requires intervention is not well established. However, most experts recommend abdominal decompression in symptomatic patients whose intra-abdominal pressure reaches 30 mm Hg.5

The well-recognized technique is used to determine bladder pressure.2,5,8In an indwelling urinary (Foley) catheter is inserted into the bladder and the bladder is emptied. The drainage bag is then clamped off, and 50 to 100 mL of sterile isotonic sodium chloride solution is instilled through the catheter tubing. When the volume in the bladder is in this range, the organ acts as a passive diaphragm.5

With the patient supine and the symphysis pubis as a zero-reference point, a pressure transducer is connected to the sampling port of the Foley catheter and the pressure (in millimetres of mercury) is displayed on the bedside monitor.2,4,5

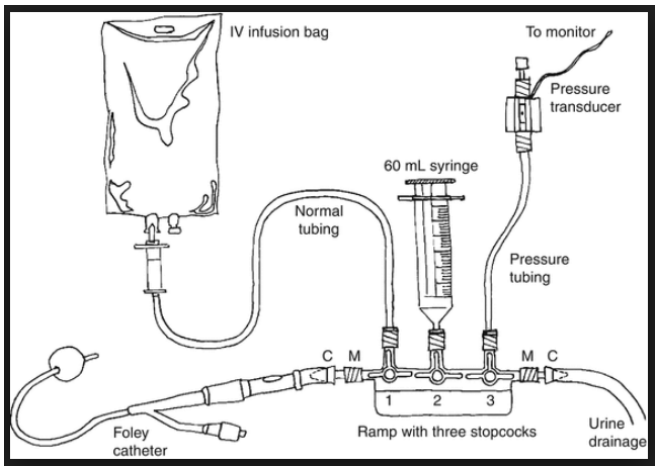

Fig 3 The well-recognized technique is used to determine bladder pressure

CONCLUSION

ACS is a potentially lethal condition caused by any event that produces intra-abdominal hypertension and causes ischemia of the peritoneal organs. ACS should be suspected in all critically ill patients, particularly those on ventilatory support in intensive care units who are haemodynamically not improving despite adequate resuscitation. ACS is a distinct clinical entity developing in the wake of a serious insult to the body affecting multiple organs in a progressive manner. The classical clinical setting remains of major trauma, massive fluid resuscitation and prolonged operation. The gut mucosa is affected first, and gut ischemia develops before renal and cardiopulmonary derangements. Hemodynamic, respiratory, renal, and neurological abnormalities are classic findings. Patients with long-time (CPB/ECC) with postoperative heart failure, and administration of high doses catecholamine can be under high risk of abdominal compartmental syndrome. Decompressive laparotomy is the mainstay of treatment if the patient is to be saved from multiorgan failure and death despite which mortality is high.

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