Intra-abdominal hypertension in postoperative cardiac surgery patients

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Intra-abdominal hypertension (IAH) is associated with multi-organ failure and mortality in critically ill patients.[1-3] The incidence and relevance of IAH have been extensively reported in general surgical patients,[3,4] but few studies have investigated patients in the perioperative period of cardiac surgery.[5-7] General risk factors for developing IAH in the absence of intra-abdominal pathological changes or a need for surgery include age, obesity, undergoing mechanical ventilation (MV) with high positive end-expiratory pressure (PEEP), high severity of disease and massive fluid resuscitation. Specific risk factors for developing IAH in patients undergoing cardiac surgery with the use of extracorporeal circulation (ECC) include splanchnic ischaemia–reperfusion with increased endothelial permeability, extravascular fluid losses, organ oedema and gastrointestinal dysmotility.[8-10] The extent of an intra-abdominal pressure (IAP) increase after ECC seems related to the degree and duration of hypothermia, haemodilution, polytransfusion, coagulopathy and the release of pro-inflammatory mediators.

The World Society of the Abdominal Compartment Syndrome (WSACS) defines IAH as a sustained increased in a patient's IAP of over 12 mmHg.[11] This condition can initiate, prolong or aggravate cardiac, respiratory and renal dysfunction, all of which are commonly observed to some degree in patients after cardiac surgery. Treatment for IAH begins with early recognition at a stage when the degree of organ functional impairment is limited and reversible. If left unrecognised and untreated, IAH can progress to the abdominal compartment syndrome (ACS) that in its most malignant course might necessitate decompressive laparotomy. The addition of open laparotomy in patients with a recent sternotomy is associated with significant increases in morbidity and mortality.[12,13] Since many of the risk factors identified in the consensus guidelines for IAH[11] do not directly apply to the postcardiac surgery population, the aim of our study was to investigate the incidence, severity and evolution of IAP in such patients, with particular emphasis on determining which clinical variables that are routinely available within the first 24 hours of admission to the intensive care unit may be used for early screening for IAH risk.

Methods

Our project was approved by the South Western Sydney Local Health District Research and Ethics Office (LNR/12/

ABSTRACT

Objective: To investigate screening variables identifying patients at risk of developing intra-abdominal hypertension (IAH) after cardiac surgery.

Design and setting: Prospective observational study in a tertiary general intensive care unit.

Participants: One hundred and eight patients admitted to the ICU after cardiac surgery, with measurements of intra-abdominal pressure (IAP).

Main outcome measures: Routinely collected clinical, physiological and biochemical variables were analysed with at least twice-daily measurements of IAP during the postoperative stay in the ICU. Variables available within 24 hours of admission to the ICU were evaluated against the incidence of IAH using logistic regression analysis to develop a set of screening criteria to identify patients at risk.

Results: Fifty patients (46%) developed IAH during their stay in the ICU and were ventilated for longer, needed more vasopressors and stayed one more day in the ICU. Plasma albumin concentration, central venous pressure, minimal abdominal perfusion pressure, cardiopulmonary and aortic cross-clamp times and the presence of abdominal distension within the first 24 hours were associated with the occurrence of IAH. A logistic regression model using these variables correctly identified 85% of patients who developed IAH.

Conclusions: A set of screening criteria routinely available within the first 24 hours of admission to the ICU after cardiac surgery could correctly identify most patients at risk of IAH.

LPOOL/330). All patients admitted to the ICU after cardiac surgery between February and May 2013 were consecutively screened for eligibility (the presence of an indwelling urinary catheter (IDC) without any obstruction and the absence of bladder dystony or chronic renal failure needing haemodialysis).

Cardiopulmonary bypass

Cardiopulmonary bypass (CPB) was performed using standard cannulation and the Maquet HL-20 CPB machine was

primed with 2 L of Hartmann solution and 200 mL of 20% albumin solution. A flow rate of 2.4 L/min/m^2 was used and after aortic clamping, cardiac arrest and protection was achieved by intermittent antegrade, with or without retrograde, cold blood cardioplegia. A target haematocrit above 20% was used during CBP and vasoactive and inotropic drugs were administered as appropriate to maintain the mean arterial pressure (MAP) > 50 mmHg.

Cardiorespiratory support in ICU

Patients were admitted to the ICU with morphine or fentanyl and propofol infusions, and were ventilated using synchronised intermittent mandatory ventilation at 6–8 mL/kg tidal volume and a PEEP of 6–8 cmH$_2$O. Ventilation was weaned to pressure support and patients were evaluated for extubation within 6 hours of admission. All patients were monitored with a pulmonary artery catheter and fluids, and vasoactive and inotropic drugs were administered if necessary to maintain the MAP > 70 mmHg and the cardiac index > 2.5 L/min/m^2, unless the surgeon's postoperative orders specified other targets.

Measurement of IAP

The IAP was measured via the IDC with a maximum installation volume of 25 mL of sterile saline with the patient in the supine position and ensuring that there were no abdominal muscle contractions. The pressure transducer was zeroed at the iliac crest at the level of the midaxillary line. At least two measurements per day were made in every patient. Sustained (lasting more than 4–6 hours) IAH (as defined above), if present, was graded between I and IV according to the WSACS consensus guidelines.[11] Patients were classified into one of two cohorts according to the presence or absence of IAH at any time during their ICU stay.

Data capture

Data recorded on admission to the ICU were demographic data (age, sex and body mass index [BMI]), the Acute Physiology and Chronic Health Evaluation (APACHE) III score, the duration of CBP time and risk factors for IAH (according to the consensus guidelines: abdominal distension, bowel dysmotility, coagulopathy, hepatic dysfunction, hypotension, hypothermia, sepsis, pneumonia, and vasopressor use[11]). Apart from the IAP, which was recorded at least twice daily, the following variables were recorded once daily from the day of admission to and until discharge from the ICU: maximum IAP (highest daily value), minimum abdominal perfusion pressure (APP) (defined as MAP minus IAP), central venous pressure (CVP), PEEP, peak airway pressure, Pao$_2$, Pao$_2$/Fio$_2$ ratio, pH, haemoglobin, platelet count, international normalised ratio, activated partial thromboplastin time, lactate, albumin, aspartate amino-

transferase (AST), alanine aminotransferase, bilirubin, creatinine, estimated glomerular filtration rate,[14] fluid intake (type and amount), fluid losses (type and amount), fluid balance and abbreviated sequential organ failure assessment (SOFA) score (except the Glasgow Coma Scale component). The duration of MV and the length of stay in the ICU and in hospital were also recorded.

Statistical analysis

We present our results as means and SDs, or as medians and interquartile ranges (IQRs), as appropriate for normally and non-normally distributed data. We made comparisons between patients with and without IAH using the unpaired t test or the Mann–Whitney test, and we analysed categorical data with the χ^2 test. Changes over time were assessed by two-way analysis of variance (ANOVA) tests, using the factors of time and presence or absence of IAH. Statistical significance was set at $P < 0.05$. We performed a stepwise logistic regression analysis with the presence of IAH at any time during ICU stay as the dependent variable. Any potential screening variable available within the first 24 hours of admission to the ICU was entered into the regression analysis if its receiver–operator characteristic (ROC) against the presence of IAH at any time generated an area under the curve (AUC) > 0.5, with $P < 0.25$. The associated criterion was based on the Youden J statistic. In the final stepwise regression analysis, all variables associated with IAH at $P < 0.05$ were entered with an α-to-remove of 0.1. The goodness-of-fit was analysed using the Hosmer–Lemeshow (HL) test.
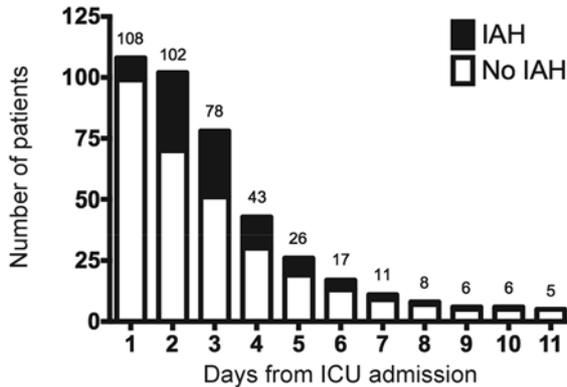
Detailed data were analysed for the first 5 days in the ICU, after which any events represented < 10% of the study cohort and no further analyses were pursued. The incidence of IAH was analysed for the entire stay in the ICU. All statistical analyses were performed using MedCalc, version 12.3 (MedCalc Software).

Results

One hundred and eight patients were studied, including 12 patients (11%) admitted after emergency surgery. Four patients underwent surgery without CPB. An intra-aortic balloon counterpulsation catheter was inserted in five patients to facilitate weaning them from CPB, and all those patients developed IAH. Fifty patients (46%) developed IAH at any time during their ICU stay. The incidence of IAH was highest on Day 2 and Day 3 of the ICU admission (Figure 1) and the mean duration of IAH was 1.57 days (SD, 0.91 days).
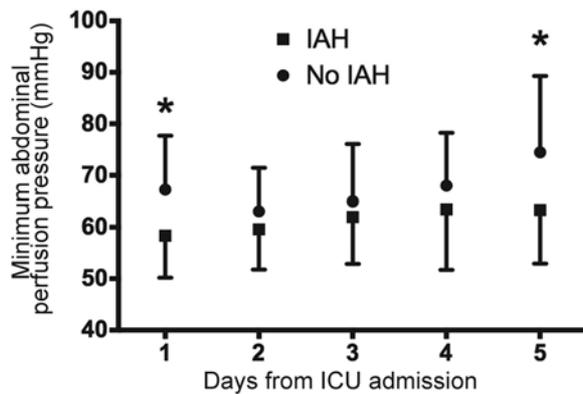
Sex, age and BMI were not significantly different when we compared patients who developed IAH and patients without IAH (Table 1). The abbreviated SOFA score recorded at ICU admission was higher in patients who developed IAH, and the

Figure 1. Daily incidence of IAH from ICU admission (Day 1)

IAH = intra-abdominal hypertension. ICU = intensive care unit.

Figure 2. Daily minimum abdominal perfusion pressure from admission to ICU (Day 1).

ICU = intensive care unit. IAH = intra-abdominal hypertension. * For patients with IAH v patients without IAH, $P < 0.05$. Error bars = SD.

Table 1. Patient characteristics and duration of interventions

Characteristic and duration	Without IAH ($n = 58$)	With IAH ($n = 50$)	P
Men/women	44/14	38/12	0.99
Age, years*	66 (13)	66 (10)	0.96
BMI*	29 (5.7)	30 (5.6)	0.26
APACHE III score[†‡]	45 (36–62)	52 (43–79)	0.12
SOFA score[†‡]	6 (4–7)	7 (6–8.5)	0.002
PPV time, days[†]	0.5 (0.5–1)	1 (0.7–2.5)	< 0.001
CPB time, minutes[†]	93 (77–125)	120 (91–139)	0.01
Aortic XC time, minutes[†]	70 (53–89)	80 (64–106)	0.02
ICU LOS, days[†]	2 (1.5–3.5)	3 (2–5)	0.005
Hospital LOS, days[†]	12.5 (7–21)	14 (7–22)	0.72

IAH = intra-abdominal hypertension. BMI = body mass index. APACHE = Acute Physiology and Chronic Health Evaluation. SOFA = Sequential Organ Failure Assessment. PPV = Positive pressure ventilation. CPB = cardiopulmonary bypass. XC = cross-clamp. ICU = intensive care unit. LOS = length of stay. * Means with SDs. † Medians with interquartile ranges. ‡ On ICU admission.

patient survived to hospital discharge (0.9% total inhospital mortality).

No patient developed ACS. Grade II IAH (16–20 mmHg) was the highest observed in any patient and occurred in 13 patients (26% of patients who developed IAH, and 12% of total patient population). The minimum APP was lower in patients with IAH and the curves were significantly separated over time (Figure 2).

Within the first 24 hours of ICU admission, patients in the IAH group had a higher IAP, lower pH, higher CVP, higher peak airway pressures, lower Pao_2 and lower Pao_2/Fio_2 ratios (Table 2).

Patients in the IAH group (when compared with patients who did not have IAH) did not receive more blood products ($P = 0.46$), albumin ($P = 0.68$), crystalloids (mean, 3.3 L [SD, 1.2 L] v mean, 2.9 L [SD, 1.1 L]; $P = 0.06$) or total fluid volume (mean, 4.4 L [SD, 1.4 L] v mean, 3.8 L [SD, 1.7 L]; $P = 0.09$) within the first 24 hours. Neither was their fluid balance different compared with patients without IAH (mean, 2.9 L [SD, 1.4 L] v mean, 2.3 L [SD, 1.6 L]; $P = 0.06$). The differences in fluid balance between patients with and without IAH did not differ significantly over time (Figure 3).

The pH of patients was lower on admission to ICU in the IAH group and remained lower throughout the ICU stay (ANOVA time $P < 0.001$; group $P = 0.01$). A delayed and sustained elevation in AST was observed in patients with IAH (ANOVA time $P = 0.01$; group $P = 0.01$) although the admission values were not different when compared with patients without IAH.

APACHE III score was not significantly different. The differences in SOFA scores between patients with and without IAH did not differ significantly over time. Patients who developed IAH at any stage had longer CPB and aortic cross-clamp times. The intraoperative use of noradrenaline ($P = 0.54$) and milrinone ($P = 0.09$) was not significantly different in patients who developed IAH compared with patients who did not develop IAH. Patients in the IAH group were ventilated for longer and stayed in the ICU one day more (as measured by the median) compared with patients who did not develop IAH. The use of vasopressors was significantly more common in patients with IAH on the first and second day of ICU admission, but similar among patients who stayed in the ICU beyond this time. All patients were discharged alive from the ICU and all but one

Table 2. Intra-abdominal pressure, blood analyses and cardiorespiratory variables of patients within the first 24 hours of ICU admission

Variable*	Without IAH (n = 58)	With IAH (n = 50)	P
IAP, mmHg	9 (2.9)	13 (3.6)	< 0.001
Lactate, mmol/L	2.4 (1.7)	2.9 (1.8)	0.18
pH	7.30 (0.05)	7.27 (0.06)	0.01
Platelets, 10^9/L	153 (50)	151 (47)	0.82
INR	1.3 (0.2)	1.3 (0.2)	0.19
APTt, sec	41 (12)	41 (24)	0.9
Albumin, g/L	32 (3.8)	31 (3.8)	0.052
AST, U/L	50 (22)	57 (34)	0.54
ALT, U/L	31 (22)	30 (21)	0.85
Bilirubin, μmol/L	14 (9.6)	14 (8.3)	0.95
Creatinine, μmol/L	95 (41)	122 (119)	0.13
eGFR, mL/min/1.73m²	56 (8.7)	53 (13)	0.28
CVP, mmHg	15 (3.1)	18 (4)	< 0.001
PEEP, cmH$_2$O	6.4 (1.8)	7.1 (2.2)	0.06
P$_{aw}$, cmH$_2$O	23 (3.4)	25 (5.3)	0.004
PaO$_2$, mmHg	112 (69)	85 (57)	0.03
PaO$_2$/FIO$_2$	231 (143)	171 (102)	0.02

ICU = intensive care unit. IAH = intra-abdominal hypertension. IAP = intra-abdominal pressure. INR = international normalised ratio. APTt = activated partial thromboplastin time. AST = aspartate aminotransferase. ALT = alanine aminotransferase. eGFR = estimated glomerular filtration rate. CVP = central venous pressure. PEEP = positive end-expiratory pressure. P$_{aw}$ = peak airway pressure. PaO$_2$ = arterial partial pressure of oxygen. FIO$_2$ = fraction of inspired oxygen. * All are means with SDs.

Figure 3. Daily fluid balance from ICU admission (Day 1)
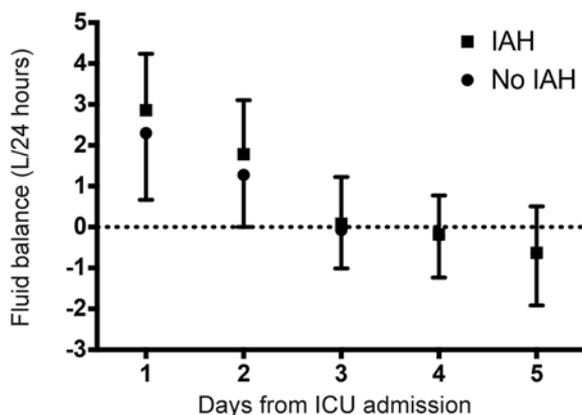
ICU = intensive care unit. IAH = intra-abdominal hypertension. Error bars = SD.

Sixteen screening variables and their cut-off criteria (as in the ROC analysis) were entered into the logistic regression analysis (Table 3). Six screening variables remained in the regression analysis with a significant goodness-of-fit (using the HL test, $P = 0.22$); these included the presence of abdominal distension, minimal APP, albumin, CPB time, cross-clamp time and CVP. The model correctly identified 85% of patients who developed IAH and the ROC generated an AUC of 0.89 (95% CI, 0.78–0.95).

Discussion

In our prospective, observational study of postoperative cardiac surgery patients, the incidence of IAH at any time during the ICU admission was high, at almost 50%. Twelve per cent of patients developed Grade II IAH, the highest grade of IAH observed, and no patients developed ACS or required a decompressive laparotomy. It is well established that ACS has dramatic pathophysiological implications that require urgent surgical intervention, and several studies of critically ill patients have also shown IAH to be associated with poorer outcomes.[1,3,15] In our study, patients with IAH

Table 3. Screening variables with an AUC > 0.5, and their P values

Variable	AUC	P*	Criterion	P†
Abdominal distension	0.66	< 0.001	na	0.01
Albumin	0.63	0.02	⩽ 32	0.04
APTt	0.70	< 0.001	⩽ 61	0.01
AST	0.65	0.04	> 46	
CPB	0.66	0.01	> 121	0.02
Aortic XC time	0.66	0.01	> 75	< 0.001
Amount of crystalloids	0.61	0.065		
CVP	0.70	< 0.001	> 15	0.02
eGFR	0.56	0.207		
Fluid balance	0.59	0.104		
Amount of fluids given	0.60	0.090		
PaO$_2$/FIO$_2$	0.59	0.106		
PaO$_2$	0.59	0.136		
P$_{aw}$	0.62	0.04	> 23	
pH	0.58	0.177		
SOFA score (Day 1)	0.65	0.01	> 6	

AUC = area under the curve. na = not applicable. APTt = activated partial thromboplastin time. AST = aspartate aminotransferase. CPB = cardiopulmonary bypass. XC = cross-clamp. CVP = central venous pressure. eGFR = estimated glomerular filtration rate. PaO$_2$ = arterial partial pressure of oxygen. FIO$_2$ = fraction of inspired oxygen. P$_{aw}$ = peak airway pressure. SOFA = Sequential Organ Failure Assessment. * Variables with $P < 0.05$ had the cut-off criterion calculated and were entered into the logistic regression analysis. † After logistic regression analysis, six variables had significant P values.

were ventilated for longer, required vasopressor support more frequently and had their median ICU stay prolonged by 1 day, but did not develop additional renal or hepatic impairment when compared with patients without IAH.

A 32% incidence of IAH within 24 hours after cardiac surgery has previously been reported.[7] Other studies have shown a significant increase in IAP intraoperatively and within 18 hours of surgery.[5,6] Our study corroborates the common occurrence of IAH within the first 24 hours of surgery and highlights the importance of consecutive measurements, since a comparable incidence was observed in Day 2 and Day 3 after surgery. Similarly to previous reports, this study identified the association between CPB and aortic cross-clamp times and subsequent development of IAH. The pathophysiological events linking CPB and IAH involve the redistribution of blood flow away from the splanchnic organs during CPB[9] (this also occurs during off-pump cardiac surgery[16]). Mesenteric injury after CPB may develop as a result of mucosal ischaemia and hypoxia, inflammation with neutrophil sequestration and complement activation. The autoregulatory capacity of mesenteric vessels is further limited after CPB, making the gut susceptible to any haemodynamic instability in the postoperative period.[10] In our study, a CPB time of over 121 minutes and an aortic cross-clamp time of over 75 minutes were significantly associated with the incidence of IAH, in isolation and in the logistic regression analysis.

Intraoperative use of noradrenaline and milrinone was not different between patients with and without IAH, but the postoperative use of vasopressors was increased in IAH patients. This was consistent with an increased degree of haemodynamic instability. This study cannot discriminate causality from association but it is interesting to note that the minimal APP was also independently associated with the development of IAH. It remains possible that targeting APP might avoid or ameliorate the evolution of IAH.

Massive fluid resuscitation and polytransfusion are well recognised independent risk factors for IAH.[1-4,11] In our study, the administration of fluids and blood products within the first 24 hours of ICU admission was not significantly different between patients who did and did not develop IAH. Neither was the overall fluid balance different between the groups, although that variable had a borderline P value of 0.06. This is different from a previous report,[7] although similar net fluid balances were observed on admission to the ICU. Importantly, our study showed an early and progressive decrease in fluid balance so that a neutral balance was achieved in most patients by the third day in the ICU. This is distinctly different from the progressive increase observed in previous studies reporting an association between fluid balance and the occurrence of IAH.[5-7] From a therapeutic point of view, this is of particular interest since it would appear that proper

attention to the management of fluid balance could dissociate this parameter from the development of IAH. Current guidelines on the management of IAH highlight the importance of targeting a negative fluid balance.[2,11] The administration of fluids was not associated with IAH, but the serum albumin level was a significant factor in the logistic regression analysis. This is consistent with a decreased colloid osmotic pressure resulting in increased extravasation of fluid, especially in the setting of deranged mesenteric capillary permeability. A low albumin level can be expected as a consequence of the normovolaemic haemodilution that occurs on CPB. The inflammatory response triggered by prolonged CPB times might further reduce serum albumin levels.

Early detection of low-grade IAH is important in preventing the development of higher grades of IAH and ACS, and IAH is associated with pathophysiological changes and increased morbidity and mortality.[1-3,15] Hence our study aimed to identify the variables, observable on admission, that could be used to screen for patients at risk of developing IAH. The model achieved correct identification of patients who developed IAH in 85% of cases, with an ROC statistic that further supported its applicability. While prolonged CPB and aortic cross-clamp times are not amenable to any change on admission to the ICU, albumin levels and minimal APP can be manipulated.

The correlation between clinical examination to estimate IAP and the obtained intravesical measurement of IAP is poor,[17] but this does not mean that clinical examination for IAP is not important, since the presence of abdominal distension is still a significant warning sign. Similar to previous reports, CVP was associated with IAH.[5-7] It is well recognised that CVP measurement is a poor gauge of intravascular volume but in the setting of IAH, a high CVP is important as a contributor to splanchnic venous stasis. It might also cause further haemodynamic compromise by decreasing the venous return pressure gradient and hence cardiac output.

The strengths of our study include the relatively large cohort studied, the prospective design that minimised selection bias and the pragmatic approach to data selected for analysis. The limitations of our study include its observational design, which precluded any direct inferences to causality and hence therapeutic options. The low number of patients studied after emergency surgery means that caution is warranted in extrapolating the results to this population.

In conclusion, IAH developed in almost half the patients admitted to the ICU after cardiac surgery. It was possible to screen patients at risk for IAH with clinically useful sensitivity and specificity, using a model based on data available within the first 24 hours of admission to the ICU. Such screening enables early identification of patients at risk of IAH who warrant specific attention and management.

Competing interests

None declared.

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