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REPORTS OF ORIGINAL INVESTIGATIONS

Intra-abdominal pressure during and after cardiac surgery: a single-centre prospective cohort study

Pression intra-abdominale pendant et après la chirurgie cardiaque : une étude de cohorte prospective monocentrique

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Abstract

Purpose *While intra-abdominal hypertension (IAH) has been associated with adverse outcomes in multiple settings, the epidemiology and clinical implications of IAH in the context of cardiac surgery are less known. In this study, we aimed to describe the prevalence of IAH in patients undergoing cardiac surgery and determine its association with patient characteristics and postoperative outcomes.*

Methods *We conducted a single-centre prospective cohort study in which intra-abdominal pressure was measured in the operating room after general anesthesia (T1), after the surgical procedure (T2), and two hours after intensive care unit (ICU) admission (T3) in a subset of patients. Intra-abdominal hypertension was defined as intra-abdominal pressure (IAP) ≥ 12 mm Hg. Postoperative outcomes included death, acute kidney injury (AKI), and length of stay in the ICU and hospital.*

Results *A total of 513 IAP measurements were obtained from 191 participants in the operating room and 131 participants in the ICU. Intra-abdominal hypertension was present in 105/191 (55%) at T1, 115/191 (60%) at T2, and 31/131 (24%) at T3. Intra-abdominal pressure was independently associated with body mass index, central venous pressure, and mean pulmonary artery pressure but was not associated with cumulative fluid balance. Intraoperative IAH was not associated with adverse outcomes including AKI.*

Conclusions *Intra-abdominal hypertension is very common during cardiac surgery but its clinical implications are uncertain.*

Résumé

Objectif *Bien que l'hypertension intra-abdominale (HIA) ait été associée à des issues indésirables dans de multiples contextes, l'épidémiologie et les implications cliniques de l'HIA dans le contexte de la chirurgie cardiaque sont moins connues. Dans cette étude, nous avons cherché à décrire la prévalence de l'HIA chez les patients bénéficiant d'une chirurgie cardiaque et à déterminer son association avec les caractéristiques des patients et les issues postopératoires.*

Méthode *Nous avons mené une étude de cohorte prospective monocentrique dans laquelle la pression intra-abdominale a été mesurée en salle d'opération après une anesthésie générale (T1), après l'intervention chirurgicale (T2) et deux heures après l'admission à l'unité de soins intensifs (USI) (T3) dans un sous-ensemble de patients. L'hypertension intra-abdominale a été définie comme une pression intra-abdominale (PIA) ≥ 12 mmHg.*

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Les issues postopératoires comprenaient le décès, l'insuffisance rénale aiguë (IRA), et la durée du séjour à l'USI et à l'hôpital.

Résultats *Au total, 513 mesures de la PIA ont été obtenues auprès de 191 participants en salle d'opération et de 131 participants à l'USI. L'hypertension intra-abdominale était présente chez 105/191 patients (55 %) à T1, 115/191 (60 %) à T2 et 31/131 (24 %) à T3. La pression intra-abdominale était indépendamment associée à l'indice de masse corporelle, à la pression veineuse centrale et à la pression artérielle pulmonaire moyenne, mais n'était pas associée à un bilan hydrique cumulatif. L'HIA peropératoire n'était pas associée à des issues indésirables, y compris à l'IRA.*

Conclusion *L'hypertension intra-abdominale est très fréquente lors d'une chirurgie cardiaque, mais ses implications cliniques sont incertaines.*

Keywords Intra-abdominal pressure · Cardiac surgery · Abdominal hypertension

An increase in intra-abdominal pressure (IAP) is a physiopathological mechanism that can result in organ dysfunction in critically ill patients.[1,2] Organ dysfunction can occur when IAP reduces the sustained perfusion of abdominal organs for a prolonged period.[3] When intra-abdominal hypertension (IAH) is believed to contribute significantly to organ dysfunction, the term abdominal compartment syndrome (ACS) is employed.[4]

The occurrence of IAH and ACS is well documented in some contexts such as intra-abdominal hemorrhage, burns, and in the context of abdominal surgery.[5] Nevertheless, IAH is likely to occur in any medical or surgical conditions in which extensive fluid resuscitation and extravascular extravasation, such as tissue edema from endothelial dysfunction or third spacing, is often encountered.[6] Cardiac surgery is associated with a general systemic inflammatory response that may exacerbate the formation of interstitial edema, particularly when high venous pressure occurs from right ventricular failure or important fluid administration.[7] Furthermore, abdominal obesity and pre-existing heart failure with elevated central venous pressure (CVP) may heighten the risk of IAH during and after cardiac surgery.[8,9] A limited number of reports have suggested that IAH may occur in a significant proportion of patients after cardiac surgery, from 32% to up to 84% in obese patients.[8–12] Nevertheless, in clinical practice, IAH is rarely considered to be the underlying mechanism of organ dysfunction following cardiothoracic surgery and is seldom reported to be of clinical importance except in specific clinical circumstances such as acute mesenteric ischemia.[13]

The main objective of this study was to describe the evolution of IAP in the perioperative period in a cohort of cardiac surgery patients. Our secondary objectives were to determine the factors associated with IAH and the associations with postoperative outcomes.

Methods

Design and data collection

We conducted a prospective cohort study in adult patients undergoing urgent or elective cardiac surgery from April 2010 to June 2010, and from April 2011 to June 2011 at a single specialized tertiary academic urban centre. The study was approved by the ethics committee (#2010-1210) in April 2010 and written consent was obtained from all participants. Patients were excluded if the surgical procedure was too urgent to permit informed consent and IAP measurements. Convenience sampling based on the availability of the research staff was used when recruiting participants.

Intra-abdominal pressure was measured in the operating room after general anesthesia was induced (T1) and the chest was closed (T2). For measurement at T1 and T2, adequate paralysis was ensured by the administration of rocuronium (0.2 $mg \cdot kg^{-1}$). After IAP was measured in the first 60 patients, the protocol was amended to include IAP measurement two hours after intensive care unit (ICU) admission (T3) in the remaining patients.

Intra-abdominal pressure was measured according to the World Society of Abdominal Compartment Syndrome (WSACS)[1] as follows: 1) A pressure transducer connected to a T connector was placed at the mid-axillary level with the patient in dorsal decubitus position at 0° elevation. 2) A 500 mL normal saline bag was connected to the T connector with a 60 mL syringe connected to the other end of the connector with a rigid tubing attached to an 18G needle. 3) The correct position of the urinary catheter in the bladder was verified using point-of-care ultrasound and the urinary catheter was then clamped. 4) The system was connected to the urinary catheter and the needle was inserted through the aspiration port. 5) Sterile normal saline (25 mL) was injected into the bladder. 6) The T connector was then switched to the pressure monitor. 7) We waited one minute to ensure intravesical pressure was stable. 8) Intravesical pressure was measured in mm Hg while mechanical ventilation was briefly interrupted. 9) The system was removed and the urinary catheter clamp was removed. All measurements were taken by the same operator (E.R.S.).

The following data were collected: demographic data, baseline comorbidities and operative risk evaluated by the

EuroSCORE II,[14] procedure type, hemodynamic data at the time of measurements including mean arterial blood pressure, CVP, mean pulmonary arterial pressure (MPAP), pulmonary occlusion pressure (PAOP), preoperative creatinine value with estimated glomerular filtration rate (eGFR) by the Modified Diet in Renal Disease equation,[15] available echocardiographic information from perioperative echocardiography, and detailed information about intraoperative fluid balance including urine output. There was no loss to follow-up.

Outcome definition

Intra-abdominal hypertension was defined as an IAP of $\geq$ 12 mm Hg and was further classified according to the grading proposed by the WSACS as follows:[16] stage 1 = 12–15 mm Hg; stage 2 = 16–20 mm Hg; stage 3 = 21–25 mm Hg; and stage 4 $>$ 25 mm Hg. Postoperative information included length of stay in the ICU and hospital, in-hospital mortality, and creatinine values on postoperative days 0 and 1. Acute kidney injury (AKI) was defined per the Kidney Diseases: Improving Global Outcomes creatinine criteria as an increase of more than 26 $\mu mol \cdot L^{-1}$ or 50% from baseline during the postoperative period.[17]

Statistical analysis

The number of patients (%) with IAH at the three studied timepoints is presented. The IAP at each timepoint is also presented as a median [interquartile range (IQR)] or mean (standard deviation [SD]), as appropriate. Categorical variables were compared between groups using the Chi square test or Fisher's exact test if cell sizes were < 5. For continuous variables, normality was assessed through visual inspection of normal Q-Q plots. Normally distributed variables were compared between groups using Student *t* test. In the presence of serious non-normality, non-parametric tests (Mann–Whitney for comparison between two groups or Kruskal–Wallis for comparison between more than two groups) were used. To determine whether there was a significant change in IAP between the studied timepoints, we used a Friedman paired samples test and, if the result was statistically significant, proceeded to perform multiple comparisons with Bonferroni correction. Descriptive baseline information is presented based on whether patients had IAH (stage 1 or higher) at T1.

Association between clinical variables and repeated IAP measurements at different timepoints were assessed using general linear mixed models with random intercepts. This type of analysis accounts for the repeated measures design, implying that the sample was not independent and produces estimates (β) with 95% confidence intervals (CI) that can be interpreted as linear variation coefficients. Variable selection was performed *a priori*. A variance component was specified for the covariance structure of the random intercept effect. Missing data were not replaced or imputed. A summary of missing data is presented in Electronic Supplementary Material (ESM) eTable 1.

The difference in the length of hospital stay, length of ICU stay, and rates of AKI between patients with IAH categorized as no IAH, stage 1 IAH, and stage $\geq$ 2 IAH were assessed using the Kruskal–Wallis test (continuous variables) and the Chi square test (categorical variables). In an exploratory analysis, the association between IAP and postoperative AKI at each timepoint was assessed by logistic regression with IAP as a continuous variable. A multivariable model was used to adjust for known risk factors for postoperative AKI including pre-existing chronic kidney disease (eGFR $<$ 60 $mL \cdot min^{-1} \cdot 1.73\ m^{-2}$) and preoperative risk evaluated by the EuroSCORE II. Adjustment variables were chosen a priori without data-driven selection and were revised during peer-review. The STROBE checklist for cohort studies can be found in ESM eTable 2 and statistical reporting was based on published guidelines.[18] A sample size of 191 was determined based on a linear regression model that would include five covariates with a model fit of $r^2 = 0.25$. This sample size would enable the detection of an increase in r^2 of 0.03 per added variable with a power of 80% considering a 0.05 significance level.

Results

A total of 191 patients were included in the study as presented in Fig. 1. Baseline characteristics are presented in Table 1. Patients were predominantly male with a median [IQR] age of 66 [58–72] yr and a mean (SD) body mass index (BMI) of 29.1 (5.2) $kg \cdot m^{-2}$. Less than 50% of patients had isolated coronary artery bypass grafting.

After the induction of general anesthesia (T1), 105/191 (55%) patients had IAH, including 61/191 (32%) patients with stage 1 IAH, 39/191 (20%) patients with stage 2 IAH, and 5/191 (3%) patients with stage 3 or 4 IAH. Patients with IAH at T1 had higher weight, BMI, and body surface area than patients who did not have IAH did (Table 1). When considering hemodynamic parameters at the start of surgery, patients with IAH also had higher CVP, PAOP, and MPAP. Nevertheless, no significant differences in other baseline characteristics were observed.

The distribution of IAP was significantly different between the three studied timepoints with multiple comparisons showing significant differences between IAP measured after anesthesia induction (T1) and 2 hours after

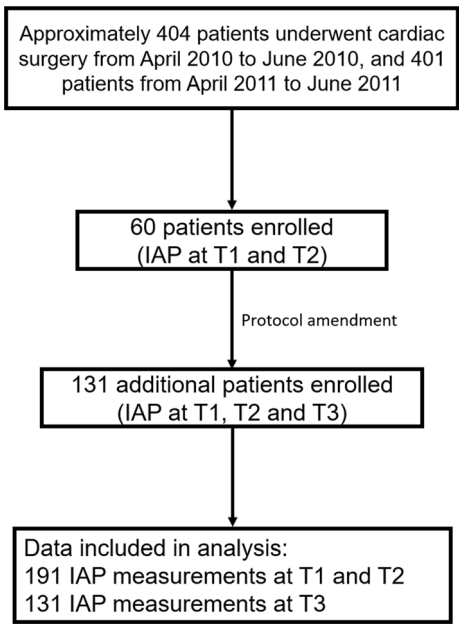


Fig. 1 Flowchart of patient inclusion. Abdominal pressure measured in the operating room, after general anesthesia (T1), after the surgical procedure (T2), and two hours after intensive care unit admission (T3). IAP = intra-abdominal pressure.

ICU admission (T3) (median IAP at T1: 13 [10–16] mmHg *vs* median at T3: 8 [6–11] mmHg, $P < 0.001$), as well as between IAP measured after chest closure (T2) and 2 hours after ICU admission (T3) (median IAP at T2: 13 [10–15] mmHg *vs* median at T3: 8 [6–11] $P < 0.001$) (Fig. 2). However, no significant difference was observed between T1 and T2 ($P = 0.12$). When considering variables associated with repeated IAP measurements, the time of assessment, BMI and CVP, and MPAP remained were independent predictors, while age, sex and intraoperative fluid balance were not (Table 2).

After cardiac surgery, patients had a median length of stay in the ICU and hospital of 2 [2–4] days and 8 [6–12] days, respectively. A total of 54/191 patients (28%) patients developed AKI and 4/191 (2%) patients died. Regarding postoperative outcomes, when comparing patients with no IAH, IAH stage 1, and IAH stage ≥ 2 at each timepoint (T1 to T3), there were no significant differences between hospital length of stay, ICU length of stay, incidence of AKI or death (Table 3). In exploratory analysis, IAP measurement as a continuous variable at T3 was associated with an increased risk of AKI in the post-operative period (OR: 1.09 (CI: 1.001 to 1.18) $P = 0.048$), while IAP measurements at T1 and T2 were not (Table 4). This association remained after adjustment for the presence of pre-existing chronic kidney disease and pre-operative risk estimated by EuroSCORE II (Table 5).

Discussion

In this prospective cohort of cardiac surgery patients, we observed that a significant proportion of patients had IAH when IAP was measured before and after chest closure and that IAP generally decreases in the early postoperative period compared with measurements taken in the operating room. BMI and CVP were the clinical variables most associated with IAP measurements. We did not observe any associations between IAH grade and postoperative outcomes. Only the exploratory analysis revealed an association between IAP measurements performed two hours after ICU admission and a higher risk of AKI in the postoperative period.

Data about IAP monitoring in adult cardiac surgery have been reported in case reports and in retrospective and prospective studies that included small to moderate samples of 1–186 patients with single to multiple IAP measurements (ESM eTable 3).[8–12,19–23] The prevalence of IAH was reported to be significant in all studies but varied widely from 32%[10] to 84% in a cohort of obese patients.[9] Abdominal obesity is likely to be a major risk factor for IAH in the cardiac surgery setting.[9,22] In support of this, BMI was observed to be associated with IAH in our cohort and others[9,21,22] in the perioperative period. Furthermore, IAH is also common before surgery in obese patients,[9,22] with baseline IAP being one of the determinant factors predicting IAH in the perioperative period.[10] While chronic IAH associated with abdominal obesity is not usually associated with ACS, it could heighten the risk of developing this condition if there is an acute increase in IAP in the setting of cardiac surgery.[12,23] This may represent one mechanism mediating the relationship between waist circumference and adverse outcomes in this population.[24] Some authors have noted that general anesthesia tends to decrease IAP in other settings and in cardiac surgery, suggesting that baseline IAP may have been even higher than what was measured after general anesthesia in some patients.[8] Nevertheless, in our cohort, the majority of patients had IAH after anesthesia was induced and after surgery but only a quarter of patients had IAH after ICU admission. This may be related to change in position and liberation from mechanical ventilation during the early postoperative period in most patients at our institution, but we did not collect information on this.

While intraoperative detection of IAH does not seem to carry important prognostic significance, we observed an increased risk of AKI associated with increasing levels of IAP in the early postoperative period. This has also been observed in previous work.[9–12,23] Some authors have reported an association between IAP and a positive fluid balance in the perioperative period[8,25] while others have not reported this.[11] While intraoperative fluid balance did

Table 1 Characteristics of included patients according to the presence of intra-abdominal hypertension after general anesthesia before cardiac surgery

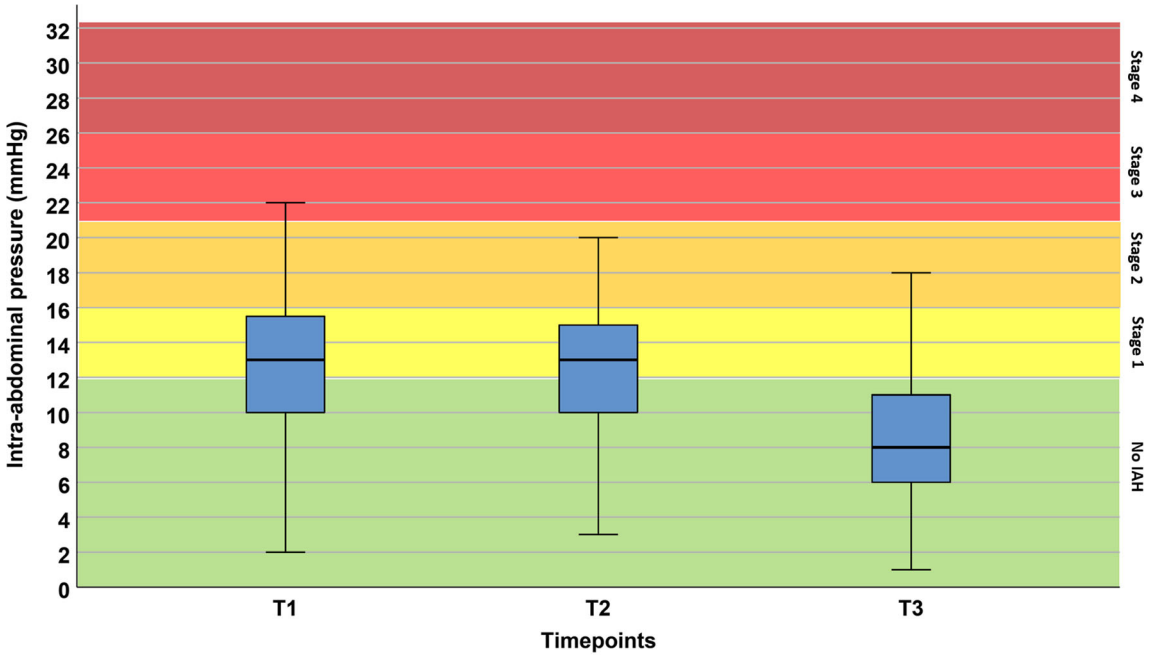
Variables	No IAH after anesthesia induction (T1) (N = 86)	IAH after anesthesia induction (T1) (N = 105)	P value
Male	65/86 (76%)	86/105 (82%)	0.29
Age (yr), median [IQR]	65 [55–71]	67 [61–72]	0.19
Weight (kg), mean (SD)	74.0 (14.5)	88.8 (15.6)	< 0.001
Height (cm), mean (SD)	168 (10)	168 (8)	0.65
Body mass index ($kg \cdot m^{-2}$), mean (SD)	26.3 (4.1)	31.4 (5.0)	< 0.001
Body surface area (m^{-2}), mean (SD)	1.8 (0.2)	2.0 (0.2)	< 0.001
Chronic kidney disease (eGFR < 60 $mL \cdot min^{-1} \cdot 1.73\ m^{-2}$)	7/86 (8%)	13/105 (12%)	0.34
Known hypertension	57/86 (66%)	79/105 (75%)	0.17
Diabetes	22/86 (26%)	40/105 (38%)	0.07
Left ventricular hypertrophy	10/86 (12%)	10/105 (9%)	0.64
Recent myocardial infarction	14/86 (16%)	19/105 (18%)	0.74
Peripheral arterial disease	14/86 (16%)	17/105 (16%)	0.99
Baseline LVEF			
> 50%	76/86 (88%)	87/105 (84%)	0.58
30–50%	9/86 (10%)	15/105 (14%)	
< 30%	1/86 (2%)	3/105 (3%)	
Type of surgery			
- Isolated CABG	41/86 (48%)	51/105 (49%)	0.91
- Isolated non-CABG procedure	19/86 (22%)	26/105 (25%)	
- Multiple procedures or other	26/86 (30%)	29/105 (28%)	
EuroSCORE II (%), median [IQR]	4.65 [2.53–7.59]	4.65 [2.71–7.50]	0.98
Parsonnet, median [IQR]	10 [6–16]	11 [8–18]	0.22
Intra-abdominal pressure at T1 (mm Hg), median [IQR]	9 [7–10]	15 [13–17]	< 0.001
Heart rate ($beats \cdot min^{-1}$), median [IQR]	53 [47–62]	54 [47–64]	0.55
Mean arterial pressure (mm Hg), mean (SD)	73 [65–77]	71 [66–81]	0.33
Central venous pressure (mm Hg), median [IQR]	12 [10–15]	16 [14–18]	< 0.001
Pulmonary artery occlusion pressure (mm Hg), median [IQR]	16 [14–19]	20 [17–22]	< 0.001
Mean pulmonary artery pressure (mm Hg), median [IQR]	25 [20–29]	28 [25–32]	< 0.001

All numbers are n/group N (%) unless otherwise specified. P values are from the Chi square or Fisher's exact test for categorical variables, or Student's t test or Mann–Whitney test for continuous variables.

CABG = coronary artery bypass grafting; IAH = intra-abdominal hypertension; IQR = interquartile range; LVEF = left ventricular ejection fraction; SD = standard deviation.

not appear to be associated with IAP in our cohort, it must be noted that IAP measurements were performed early in the postoperative period while fluid expansion may occur later in the course of ICU stay. We observed that CVP and MPAP were significantly associated with IAP in our cohort as reported in previous work.[8,10,11,20] Because decompensated heart failure may be responsible for IAH,[26] patients with inadequately compensated heart failure before surgery, or with acute right ventricular dysfunction in the perioperative period may be at risk of developing ACS. Nevertheless, it must be noted that an elevation in IAP may itself cause an elevation in CVP.

This study has strengths. For example, we collected repeated measurements during the perioperative period using a standardized methodology at the bedside. Furthermore, this is the largest reported study on IAH in



	T1 (After anesthesia)	T2 (After procedure)	T3 (2 hours after ICU admission)
Median IAP	13 [9-15]	13 [10-15]	8 [6-11]
No IAH	86/191 (45.0%)	76/191 (39.8%)	100/131 (76.3%)
Stage 1 IAH	61/191 (31.9%)	73/191 (38.2%)	20/131 (15.3%)
Stage 2 IAH	39/191 (20.4%)	40/191 (20.9%)	8/131 (6.1%)
Stage 3 IAH	4/191 (2.1%)	0/191 (0%)	2/131 (1.5%)
Stage 4 IAH	1/191 (0.5%)	2/191 (1.0%)	1/131 (0.8%)

Fig. 2 Median intra-abdominal pressure (IAP) values during the perioperative period including after general anesthesia in the operating room (T1), after chest closure (T2), and two hours after admission to the intensive care unit (T3) with distribution according to intra-abdominal hypertension (IAH) staging. There was a significant difference in the distribution of IAP between timepoints ($P < 0.001$, Kruskal–Wallis test). Multiple comparisons showed significant differences between T1 and T3 ($P < 0.001$) and between T2 and T3 ($P < 0.001$) but not between T1 and T2 ($P = 0.12$).

cardiac surgery to date. The study also has limitations that must be considered. Intra-abdominal pressure was only measured in the early period after cardiac surgery, and IAH may have later deteriorated during ICU stay. It is well-established in the IAH literature that the highest risk patients are those undergoing emergent surgery.[1] Because our study required consent and our research team was only available during the day, we excluded those patients. Furthermore, IAP was only measured after ICU admission in two thirds of patients. We did not obtain measures later on when IAH pressure might have increased. The IAH has been reported to peak within few hours of ICU admission,[10,12] which we captured. Nevertheless, our population was mostly at risk of secondary compartment syndrome and the IAP values were significantly lower in the ICU. In addition, the sample size limits the ability to detect associations with postoperative outcomes. Several risk factors for kidney failure have been reported in cardiac surgery,[27–29] but the relative role of abdominal perfusion pressure and IAH has not been explored in many of these studies. For this reason, analyses of postoperative outcomes remain exploratory. It must be noted that the association between IAP measured two hours after admission to the ICU was not a prespecified analysis in this study; therefore, our findings on this may be false positive and should be considered hypothesis-generating.

In summary, we observed that IAH occurs in most patients undergoing cardiac surgery and in almost a quarter of patients in the early postoperative period. While we did not observe that IAH during surgery was associated with

Table 2 Factors associated with intra-abdominal pressure

	Univariable Estimates (CI); *P* value	Multivariable Estimates (CI); *P* value
Time of assessment		
- After anesthesia induction (T1)	Ref	Ref
- After chest closure (T2)	0.4 (−0.3 to 1.0); $P = 0.25$	0.3 (−0.7 to 1.2); $P = 0.61$
- 2 hr after ICU admission (T3)	−3.6 (−4.3 to −2.9); $P < 0.001$	−2.6 (−3.6 to −1.5); $P < 0.001$
Age (per 10 yr increments)	0.2 (−0.3 to 0.7); $P = 0.45$	−0.1 (−0.5 to 0.4); $P = 0.75$
BMI (per $kg \cdot m^{-2}$ increments)	0.4 (0.3 to 0.5); $P < 0.001$	0.3 (0.2 to 0.4); $P < 0.001$
Male sex	1.1 (−0.3 to 2.5); $P = 0.12$	0.4 (−0.7 to 1.5); $P = 0.50$
CVP (per 10 mm Hg increments)	4.3 (3.5 to 5.0); $P < 0.001$	2.4 (1.6 to 3.3); $P < 0.001$
MPAP (per 10 mm Hg increments)	2.9 (2.3 to 3.5); $P < 0.001$	0.8 (0.05 to 1.5); $P = 0.03$
Intraoperative fluid balance (% of body weight)	−0.4 (−0.6 to −0.2); $P < 0.001$	−0.2 (−0.4 to 1.3); $P = 0.29$

Data from 513 IAP measurements in 191 patients including 191 before anesthesia, 191 after the procedure and 131 two hours after ICU admission. Mixed linear models with random intercept. The estimates represent the estimated mean variation in intra-abdominal pressure per unit of increase in the variable (fixed effects).

BMI = body mass index; CI = 95% confidence interval; CVP = central venous pressure; IAP = intra-abdominal pressure; ICU = intensive care unit; MPAP = mean pulmonary artery pressure.

Table 3 Post-operative outcomes according to intra-abdominal hypertension (IAH) measured at studied timepoints

	No IAH	IAH stage 1	IAH stage ≥ 2	*P* value
IAP measured after anesthesia (T1 – 191 patients)				
Hospital length of stay (days), median [IQR]	9 [6–13]	8 [6–12]	8 [7–14]	0.66
ICU length of stay (days), median [IQR]	2 [2–3]	2 [2–3]	2 [2–4]	0.52
Acute kidney injury	27/86 (31%)	14/61 (23%)	13/43 (30%)	0.51
In-hospital mortality	2/86 (4%)	0/61 (0%)	2/44 (4%)	0.27
IAP measured after chest closure (T2 – 191 patients)				
Hospital length of stay (days), median [IQR]	9 [6–14]	8 [6–10]	9 [7–13]	0.19
ICU length of stay (days), median [IQR]	2 [2–3]	2 [2–3]	2 [2–4]	0.60
Acute kidney injury	20/76 (26%)	19/73 (26%)	15/41 (37%)	0.42
In-hospital mortality	1/76 (1%)	2/73 (3%)	1/42 (2%)	1.00
IAP measured 2 hr after ICU admission (T3 – 131 patients)				
Hospital length of stay (days), median [IQR]	9 [7–14]	7 [6–10]	11 [7–24]	0.10
ICU length of stay (days), median [IQR]	2 [2–4]	2 [2–3]	3 [2–5]	0.32
Acute kidney injury	31/100 (31%)	7/20 (35%)	5/11 (45%)	0.62
In-hospital mortality	2/55 (4%)	0/43 (0%)	1/33 (3%)	0.29

All numbers are *n*/group N (%) unless otherwise specified. *P* values are from the Chi square or Fisher's exact test for categorical variables, or Kruskal-Wallis test for continuous variables.

IAP = intra-abdominal pressure; ICU = intensive care unit; IQR = interquartile range.

adverse outcomes, our study reports an elevated prevalence of IAH in cardiac surgical patients,[30] which may represent a risk factor for AKI.[12,26,31] Additional studies are required to explore whether strategies aimed at reducing IAH in the postoperative period may influence the risk of complications in selected patients.

Table 4 Association between acute kidney injury in the postoperative period and intra-abdominal pressure according to the different time of assessment

	OR (95% CI)	*P* value
IAP after anesthesia induction (T1)	1.03 (0.96 to 1.11)	0.38
IAP after chest closure (T2)	1.04 (0.97 to 1.12)	0.22
IAP 2 hr after ICU admission (T3)	1.09 (1.001 to 1.18)	0.048

Data from 513 IAP measurements in 191 patients including 191 before anesthesia, 191 after the procedure, and 131 two hours after ICU admission. Univariate logistic regression, OR are presented per 1 mm Hg of increase in IAP.

CI = confidence interval; IAP = intra-abdominal pressure; ICU = intensive care unit; OR = odds ratio.

Table 5 Association between intra-abdominal pressure measured two hours after admission to the intensive care unit after cardiac surgery and acute kidney injury in the postoperative period

	Univariable OR (95% CI); *P* value	Multivariable OR (95% CI); *P* value
IAP 2 hr after ICU admission (T3)	1.09 (1.001 to 1.18); $P = 0.048$	1.13 (1.03 to 1.25); $P = 0.01$
Pre-existing chronic kidney disease (eGFR < 60 mL·min^{-1}·1.73 m^{-2})	2.22 (0.67 to 7.3); $P = 0.19$	1.60 (0.43 to 5.97); $P = 0.48$
EuroSCORE II (%)	1.06 (1.01 to 1.11); $P = 0.02$	1.05 (0.99 to 1.10); $P = 0.09$
Duration of CPB	1.55 (0.95 to 2.53); $P = 0.08$	1.33 (0.77 to 2.29); $P = 0.30$
Blood transfusion during surgery	3.02 (1.18 to 7.70); $P = 0.02$	2.09 (0.65 to 6.65); $P = 0.21$

Data from 131 patients who had available IAP measurements two hours after ICU admission. Univariable and multivariable logistic regression, odds ratio (OR) are presented per 1-unit increase.

CI = confidence interval; CPB = cardiopulmonary bypass; IAP = intra-abdominal pressure; ICU = intensive care unit; eGFR = estimated glomerular filtration rate; OR = odds ratio.

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