Surgery 166 (2019) 1128–1134

Contents lists available at ScienceDirect

Surgery

journal homepage: www.elsevier.com/locate/surg

Intra-abdominal hypertension in obese patients undergoing coronary surgery: A prospective observational study

Rakan Nazer, MD[a,*], Ali Albarrati, PhD[b], Anhar Ullah, MSc[a], Sultan Alamro, MD[a], Tarek Kashour, MD[a]

[a] Department of Cardiac Science, King Fahad Cardiac Center, College of Medicine, King Saud University, Riyadh, Kingdom of Saudi Arabia
[b] Department of Rehabilitation Science, College of Applied Medical Science, King Saud University, Riyadh, Kingdom of Saudi Arabia

ARTICLE INFO

Article history:
Accepted 27 May 2019
Available online 26 July 2019

ABSTRACT

Background: Coronary artery bypass grafting surgery has an increased risk of adverse events in obese patients. This increased risk might be explained in part by an increased intra-abdominal pressure and the development of intra-abdominal hypertension. Therefore, the objective of this study was to investigate the correlation between obesity and intra-abdominal hypertension and to evaluate its possible impact after coronary artery bypass grafting.

Methods: A total of 50 consecutive patients scheduled to undergo coronary artery bypass grafting at a single center were selected prospectively before undergoing elective coronary artery bypass grafting. Based on the body mass index, 25 obese (body mass index $\geq$ 30) patients were matched with 25 control patients. Each patient had intra-abdominal pressure taken at baseline followed by one measurment every 4 hours until 24 hours after coronary artery bypass grafting. The serum markers for liver and kidney functions were collected once a day for 7 days after coronary artery bypass grafting.

Results: Obese patients had a greater (mean ± SD) peak intra-abdominal pressure (15.4 ± 1.6 mm Hg versus 10.6 ± 1.6 mm Hg; $P = .011$) and mean change of intra-abdominal pressure from baseline (5.1 ± 3.3 mm Hg versus 2.2 ± 2.4 mm Hg; $P = .001$). The mean abdominal perfusion pressure was less in the obese group (63.0 ± 8.0 mm Hg versus 70.1 ± 11 mm Hg; $P = .017$). The liver dysfunction, as determined by the Schindl liver function scoring system between the obese and control groups, was not statistically significant (28% vs 8%; $P = .066$). More patients in the obese group developed renal injury based on the calculated glomerular filtration rate (32% vs 8%; $P = .034$). Obesity was highly associated with developing intra-abdominal hypertension (odds ratio: 2.99; 95% confidence interval: 1.92–3.53; $P < .001$).

Conclusion: Obesity is associated with the development of intra-abdominal hypertension after coronary artery bypass grafting. This effect might indirectly impair the renal and liver functions through a decrease in the abdominal perfusion pressure.

Introduction

Increased intra-abdominal pressure (IAP) is a frequent occurrence in critically ill patients and has been independently linked with mortality.[1,2] According to the World Society of Abdominal Compartment Syndrome (WSACS), intra-abdominal hypertension (IAH) is defined as a sustained or repeated pathologic increase of IAP $\geq$ 12 millimeters mercury (mm Hg).[3] Bedside measurement of IAP is best performed via transducing a urinary bladder catheter in accordance with an internationally standardized protocol.[4] A pathologically increased IAP in critical care patients is an important risk factor for multiorgan dysfunction, particularly in the visceral abdominal organs supplied by the splanchnic circulation.[5] Furthermore, the IAP in obese patients tends to be abnormally increased, because their body mass index (BMI) positively correlates with the measured baseline IAP and other obesity-related comorbidities.[6–8] Although the underlying mechanisms are not clearly understood, it has been speculated that the increase in the volume of the abdominal or retroperitoneal contents, presumably by fatty deposits, increases the IAP, which is transmitted to the adjacent organs and cavities. Even a small persistent increase in IAP

* Reprint requests: Rakan I. Nazer, MD, 3642 King Saud University, Riyadh 12372-7143, Kingdom of Saudi Arabia.
E-mail address: raknazer@ksu.edu.sa (R. Nazer).

https://doi.org/10.1016/j.surg.2019.05.038
0039-6060/

can adversely affect renal function, cardiac output, hepatic blood flow, the mechanics of breathing, splanchnic blood flow, and intracranial pressure.[5,9] Consequently, the presence of IAH in obese patients is associated with a decrease in visceral perfusion, which is evidenced by the strong correlation between the increased IAP and decreased splanchnic blood flow shown by a decrease in the intestinal intramucosal pH.[10] Although IAH and abdominal compartment syndrome have been investigated extensively in critically ill and general surgical patients,[9–13] limited studies have reported the impact of obesity and IAH in patients undergoing cardiac surgery.[14] The association between BMI and IAH can in part explain the increased adverse outcomes in obese patients after cardiac surgery.[15]

We hypothesized that obesity may have an important association with IAH in patients undergoing cardiac surgery, and that this in turn may have an adverse effect on the kidney and liver function in the postoperative period. Accordingly, the objective of the current study was to prospectively identify an association between obesity (BMI $\geq$ 30 kg/m^2) before "on-pump" coronary artery bypass grafting (CABG) and the incidence of IAH. We also aimed to quantify objectively the potential adverse effects of obesity on the postoperative hemodynamics liver and kidney functions.

Methods

Study design

After the approval from our local institutional review board, potential candidates for the current, prospective, nonrandomized observational study were selected from among patients scheduled to undergo elective CABG. All listed patients had to have underlying ischemic heart disease and were scheduled to undergo isolated, on-pump CABG surgery. Patients were excluded from enrollment if they had end-stage renal dysfunction, creatinine clearance of <60 mL/min, active urinary tract infection, history of obstructive uropathy, history of bladder tumors, left ventricular ejection fraction <40%, valvular heart disease, or needed vasopressor support before CABG. All qualified patients were enrolled from a single center between January 2018 and August 2018. A total of 50 consecutive adult patients were selected for the study. Preoperatively, a cohort of 25 obese ($\geq$30 kg/m^2) patients were prospectively identified and were matched with a similar cohort of 25 controls (<30 kg/m^2) patients who had CABG within the same enrollment period. Each participant provided informed consent to participate in the study.

Conduct of CABG and cardiopulmonary bypass

All patients were induced under general anesthesia and intubated in the supine position. Anesthesia was obtained by administering 0.01 to 0.02 mg/kg fentanyl (Fentanyl, Polfa SA, Warsaw, Poland), 0.05 to 0.1 mg/kg midazolam (Dormicum, Roche, Basel, Switzerland), and 0.1 to 0.5 mg/kg etomidate (Hypnomidate, Janssen, Neuss, Germany). For muscle relaxation, a single dose of 0.08 0.1 mg/kg pancuronium (Pavulon, Organon-Teknika, Veedijk, Belgium) was administered. During cardiopulmonary bypass (CPB), anesthesia was maintained using an intravenous continuous infusion of fentanyl, midazolam, and fractionated doses of inhaled isoflurane (0.5–1 vol%; Forane, Baxter, Deerfield, IL, USA). The heart-lung machine was primed with a total of 1,500 to 1,700 mL of prime solution: 1,000 mL of Ringer's solution (Polfa SA), 250 mL of a 6% solution of hydroxyethylated starch (HAES, Fresenius-Kabi, Bad Homburg, Germany), 250 mL of 20% mannitol (Fresenius-Kabi,), 20 mL of sodium hydroxycarbonate (natrium bicarbonatum, Polpharma, Warsaw, Poland), and 75 mg heparin. Cardiac arrest protection was achieved by means of intermittent cold blood-potassium (1:4) cardioplegia. During CPB, the systemic temperature was maintained at 32°C, the blood flow was 2.4 L/min/m^2, the mean arterial pressure (MAP) was >50 mm Hg, and the target hematocrit was $\geq$20%.

After operation, all patients were kept in the intensive care unit (ICU) under mechanical ventilation with pressure-controlled ventilation (tidal volume 6–8 mL/kg, positive end-expiratory pressure 5–8 cmH_2O, respiratory rate 9–12 breaths/min) with adjustments to maintain normocapnia. Ventilation was weaned to pressure support, and extubation was accomplished once the patient was fully awake, rewarmed, hemodynamically stable, and with an acceptable range of blood gas analysis. All patients were monitored with a pulmonary artery catheter and received fluids or vasoactive drugs when deemed necessary to maintain MAP > 65 mm Hg and a cardiac index of >2.0 L/min/m^2. No nasogastric tubes were placed for gastric decompression in this series. Chest drains were removed within 24 to 48 hours once the total drainage was less than 80 mL per an 8-hour shift.

Measuring the IAP

The IAP was measured intermittently via a Foley urinary bladder catheter in accordance with the World Society of Abdominal Compartment Syndrome standardized technique.[4,5] The IAP measurement was taken at baseline once the catheter was inserted. This measurement was followed by serial once every 4 hours readings in the ICU for 24 hours after CABG. The IAP measurement technique was applied using the AbViser (Wolfe Tory Medical Inc, Salt Lake City, UT, USA) specialized kit.[5] The system is positioned aseptically between the urinary catheter and the collecting bag. The pressure transducer is zeroed and positioned on the iliac crest at the level of the midaxillary line. Before each measurement, 20 mL of sterile saline is injected into the bladder. A valve draining the bladder automatically closes to allow for the direct transducing of bladder pressure. Each reading lasts 1 to 3 minutes and is taken with the patient in the supine position, at the end of expiration, and in the absence of abdominal muscle contractions. The valve system then opens automatically to allow for normal bladder drainage, and the volume of saline utilized for injection is subtracted from the patient's urinary output in that hour. A patient was labeled to have IAH if the IAP was recorded to be $\geq$12 mm Hg on a minimum of 3 settings.

Data collection

For all the subjects enrolled in the study, the authors collected general demographics, pre-existing comorbidities, and operative and clinical outcome data from the time of enrollment and until the day of discharge. The MAP (mm Hg), central venous pressure ([CVP] mm Hg), abdominal perfusion pressure ([APP] defined as MAP − IAP = APP; mm Hg), cardiac index ([CI] L/m^2), mixed venous oxygen saturation (%SVO_2), urine output (mL/h), and fluid balance (mL) were recorded in the ICU every 4 hours until 24 hours after CABG.

Serum levels of aspirate aminotransferase (U/L), alanine aminotransferase ([ALT] U/L), total bilirubin (umol/L), lactate (mmol/L), international normalized ratio (INR), creatinine (umol/l), and estimated glomerular filtration rate ([eGFR] ml/min)[16] were recorded before operation and measured daily for up to 7 days postoperatively. A modified version of the Schindl liver function scoring system, which is based on the total bilirubin levels, INR, and lactic acid levels, was described recently to objectively quantify the postoperative transient liver dysfunction.[17–19] Other well-established scoring systems for liver function, such as the Model

Table I
Modified Schindl liver function score

Laboratory parameter	Score		
	0	1	2
Total serum bilirubin (umol/L)	<20	20–60	>60
International normalized ratio (INR)	<1.8	1.8–2.3	>2.3
Serum lactate (mmol/L)	<1.5	1.5–3.5	>3.5

Total score of ≥4 indicates clinically important liver dysfunction.

for End-stage Liver Disease or the Child-Pugh classification, were not suitable for this study because they were either designed to scale patients with end-stage liver disease or those with liver cirrhosis. The Schindl score (range, 0–8; Table I) for each patient in the cohort was calculated by averaging the 2 greatest serum values of each component in the first 7 days after CABG. Schindl scores ≥4 were used as a cutoff indicator of clinically relevant liver dysfunction after CABG.[20] Postoperative renal injury was defined as having a decrease in the calculated glomerular filtration rate (GFR) to less than 60 mL/min during the first 7 days after CABG.

Statistical analysis

Categorical data were summarized as absolute numbers and percentages. Numeric data were summarized as the mean and standard deviation or median and interquartile range. Comparison between groups was performed using the χ^2 test or the Fisher exact for categorical variables and using the independent sample *t* test or Mann–Whitney *U* test for continuous variables. The crude odds ratios with 95% confidence intervals (CIs) were estimated using univariate logistic regression and adjusted odds ratios with 95% CIs were estimated using multiple logistic regression analysis. Analysis was performed using SAS/STAT software, v 9.2 (SAS Institute, Cary, NC, USA.) and R software (Foundation for Statistical Computing, Vienna, Austria). A 2-sided *P* value of <.05 was considered statistically significant.

Results

The cohort of 25 obese patients (BMI ≥ 30 kg/m^2) was matched with another 25 non-obese (control group) patients who underwent the same procedure within the study timeframe. No differences were observed regarding age, sex, or history of myocardial infarction, left ventricular function, and symptoms of heart failure (Table II). There were no observable differences in baseline serum creatinine, liver transaminase, and total bilirubin values, or common morbid conditions. Neither group had patients with chronic obstructive lung disease. The measured baseline IAP was greater in the obese group at the start of the operation (10.3 ± 2.4 mm Hg vs 8.4 ± 2.0 mm Hg; $P = .003$; Table III). This difference continued to be observed in the serial postoperative measurements in which the peak IAP (15.4 ± 1.6 mm Hg vs 10.6 ± 1.6 mm Hg; $P = .011$) and the mean change from baseline to the peak IAP (5.1 ± 3.3 mm Hg vs 2.2 ± 2.4 mm Hg; $P = .001$) were greater in the obese group (Fig 1, *A*). More patients in the obese group developed IAH (84% vs 28%; $P < .001$) and spent a greater time on mechanical ventilation (7.1 ± 3.0 hours vs 5.7 ± 1.6 hours; $P = .045$). The CVP was slightly greater in the obese group (12.6 ± 1.9 mm Hg vs 10.6 ± 2.0 mm Hg; $P = .034$; Fig 1, *B*), and the mean APP was less in that group (63.0 ± 8.0 mm Hg versus 70.1 ± 11 mm Hg; $P = .017$). The CI was similar in the obese group (2.6 ± 0.3 L/min/m^2 vs 2.9 ± 0.6 L/min/m2; $P = .036$; Fig 1, *C*) but did not constitute clinical importance nor was the need for vasopressors or mixed venous oxygen in the ICU postoperatively different. There was a mild observable difference in the peak liver ALT transaminase (59.0 ± 15 U/L vs 46.5 ± 9 U/L; $P = .045$), with a greater peak in the obese group but not a greater postoperative liver dysfunction based on the Schindl score (28% vs 8%; $P = .066$). The calculated mean GFR was less in the obese group (61.3 ± 7.3 mL/min vs 73.0 ± 10 mL/min; $P = .002$) as was the median hourly urine output in the first 24 hours after CABG (35 mL/min vs 42 mL/min; $P = .023$). More patients in the obese group developed postoperative renal injury based on the calculated GFR (32% vs 8%; $P = .034$). No observable differences were observed in the postoperative adverse clinical outcomes (Table IV).

In this cohort, being obese was associated with the development of IAH in the postoperative period (OR: 2.99; 95% CI: 1.92–3.53; $P < .001$). The association remained after adjusting for the following competing factors: sex, left ventricular ejection fraction, CPB time, mechanical ventilation time, 24-hour fluid balance, and hourly urine output (OR: 2.32; 95% CI: 1.53–2.72; $P = .002$; Fig 2).

Discussion

Obese patients who require CABG have increased mortality, major morbidity, and high cost for hospital care.[15] In the present observational study, we found that even in a small but balanced cohort of obese and control patients, the impact of obesity on the IAP was obvious. Both at baseline and in the postoperative period, the obese patients had a statistically greater measured IAP and developed more frequent IAH after CABG (84% vs 20%). From a pathophysiologic viewpoint, having IAH could seemingly explain the subclinical impact on the postoperative hemodynamics, which might explain in part the impairment in abdominal perfusion pressure and liver and renal functions. Although none of the obese patients showed an observable difference in adverse clinical outcomes, except for more surgical site infections in the obese group (12% vs 0%), notable subclinical trends were observed. During the critical care phase after CABG, the obese group had a series of distinct patterns that are consistent with the results of other studies in similar subjects: greater requirement of mechanical ventilation, higher CVP, lower mean APP, and lesser cardiac index. Although a higher BMI is associated with a greater IAP in patients undergoing general surgery procedures,[21] only 1 study of 186 consecutive adult patients undergoing cardiothoracic surgery established a positive correlation between BMI and IAP ($r^2 = 0.05$, $P = .003$),[14] which is consistent with our results. Furthermore, the utilization of the CPB machine during extracorporeal circulation can initiate an inflammatory response that affects the splanchnic microcirculation, thereby leading to a shift in fluids and interstitial edema, which may explain the documented increase in the IAP after CABG.[22] In a simple cohort of 45 adult patients undergoing "on-pump" CABG, Dabrowski et al[23] measured the transbladder IAP at 7 points during and after the operation and found the increase in IAP occurred immediately after CABG and presisted up to 18 hours after CABG. This correlated with a decrease in the APP while maintaining a steady MAP postoperatively. Our present study confirms the findings, demonstrating that obesity can amplify the apparent increase in IAP and decrease the APP during the first 24 hours after CABG. The postoperative increase in CVP in patients who develop IAH after cardiac surgery has also been demonstrated elsewhere.[23–25] Although the dynamics between IAP and CVP are not clear, it is speculated that the increase of the diaphragm caused by increased IAP results in an increase in intrapleural pressure, inspiratory pressure, and vascular resistance in the lung microcirculation, all of which affect CVP.[26]

Table II
Preoperative characteristics

	Obese group (BMI ≥ 30)	Control group (BMI < 30)	*P* value
	n = 25	*n* = 25	
Demographics			
Age (y) ± SD	56.3 ± 6.2	53.2 ± 6.7	.105
Female (%)	7 (28%)	3 (12%)	.157
Mean body mass index (kg/m^2)	32.9 ± 5.0	25.1 ± 2.2	<.001
Myocardial infarction (STEMI/NSTEMI) (%)	17 (68%)	20 (80%)	.333
Median postoperative stay (days) (IQR)	5.0 (5-6.5)	5.0 (5-6)	.732
NYHA Class III–IV (%)	2 (8%)	1 (4%)	.500
Left ventricular EF%	50.8 ± 8.0	53.1 ± 7.1	.279
Redo CABG (%)	0	0	
Morbidities with baseline liver and kidney markers			
Preoperative serum creatinine (umol/L)	96.8 ± 20.8	97.1 ± 25.6	.971
Preoperative AST (U/L) ± SD	24.7 ± 18.2	30.6 ± 25.0	.353
Preoperative ALT (U/L) ± SD	40.0 ± 12.8	38.5 ± 12.9	.687
Preoperative total bilirubin (umol/L) ± SD	10.4 ± 9.0	10.8 ± 5.3	.865
Preoperative INR	1.14 ± 0.04	1.06 ± 0.11	.091
Diabetes (%)	18 (72%)	16 (64%)	.544
HbA1C %	7.8 ± 1.4	8.1 ± 1.7	.508
Still smoking (%)	7 (28%)	5 (20%)	.508
Chronic lung disease (%)	0	0	
History of stroke (%)	1	1	1.000
Peripheral vascular disease (%)	5 (20%)	1	.189

BMI, body mass index; *IQR*, interquartile range; *STEMI*, ST-elevation myocardial infarction; *NSTEMI*, non ST-elevation myocardial infarction; *NYHA*, New York Heart Association classification; *EF*, ejection fraction; *CABG*, coronary arteries bypass grafting; *AST*, aspartate aminotransferase; *ALT*, alanine aminotransferase; *INR*, international normalized ratio; *HbA1C*, glycosylated hemoglobin A1C.

Table III
Perioperative outcome variables

	Obese group (BMI ≥ 30)	Control group (BMI < 30)	*P* value
	n = 25	*n* = 25	
CPB time (minutes) ± SD	101 ± 14	96 ± 12	.222
Ventilation time (hours) ± SD	7.1 ± 3.0	5.7 ± 1.6	.045
Median tidal volume during ventilation ml (IQR)	600 (550-650)	550 (500-700)	.441
Baseline intra-abdominal pressure (mm Hg)	10.3 ± 2.4	8.4 ± 2.0	.003
Peak intra-abdominal pressure (mm Hg)	15.4 ± 1.6	10.6 ± 1.6	.011
Mean change in intra-abdominal pressure (mm Hg)	5.1 ± 3.3	2.2 ± 2.4	.001
Intra-abdominal hypertension (%)*	21 (84%)	5 (20%)	<.001
Circulation			
Mean arterial pressure (mm Hg)	80.3 ± 8.0	78.3 ± 5.0	.270
Peak central venous pressure (mm Hg)	12.6 ± 1.9	10.6 ± 2.0	.034
Mean abdominal perfusion pressure (mm Hg)	63.0 ± 8.0	70.1 ± 11	.017
Postoperative cardiac index (mean) ± SD	2.6 ± 0.3	2.9 ± 0.6	.036
Lowest mixed venous oxygen saturation% (SVO2)	55.5 ± 4.6	60.0 ± 4.3	.130
Requirement for vasopressor support (%)	8 (32%)	7 (28%)	.758
Liver			
Peak ALT (U/L) ± SD	59.0 ± 15	46.5 ± 9	.045
Peak AST (U/L) ± SD	80.0 ± 47	60.6 ± 25	.075
Peak total bilirubin (umol/L) ± SD	19.3 ± 8.9	14.2 ± 3.5	.170
Peak lactic acid (mmol/L) ± SD	7.1 ± 1.4	5.4 ± 2.3	.350
Peak INR	1.3 ± 0.2	1.2 ± 0.08	.130
Schindl score ≥ 4 (%)	7 (28%)	2 (8%)	.066
Kidney			
Peak creatinine (umol/L)±SD	124 ± 23	114 ± 33	0.241
Mean glomerular filtration rate (mL/min)	61.3 ± 7.3	73.0 ± 10	0.002
Median urine output in the first 24 hours (mL/h) [IQR]	35 [30-40]	42 [37-60]	0.023
Median fluid balance in the first 24 hours (mL) [IQR]	+513 [430-900]	+408 [-104 to 652]	0.062
Postoperative renal injury (GFR < 60 mL/min)†	8 (32%)	2 (8%)	0.034

BMI, body mass index; *CPB*, cardiopulmonary bypass; *IQR*, interquartile range; *AST*, aspartate aminotransferase; *ALT*, alanine aminotransferase; *INR*, international normalized ratio; *GFR*, glomerular filtration rate.

* Intra-abdominal pressure of ≥ 12 mm Hg on at least 3 settings.

† Calculated GFR < 60 mL/min during the first 7 days postsurgery.

The incidence of IAH after cardiac surgery varies between 27% and 83%.[14,24,25,27] This difference is caused by a wide range of factors that influence IAP during cardiac surgery. Dalfino et al[24] recorded the IAP for up to 24 hours in 69 consecutive adult patients admitted in the ICU after undergoing cardiac surgery. One third of the patients developed IAH with a linear correlation between IAP changes and total fluid balance in the on-pump group.[24] Unlike the results in the present study, BMI was not found to

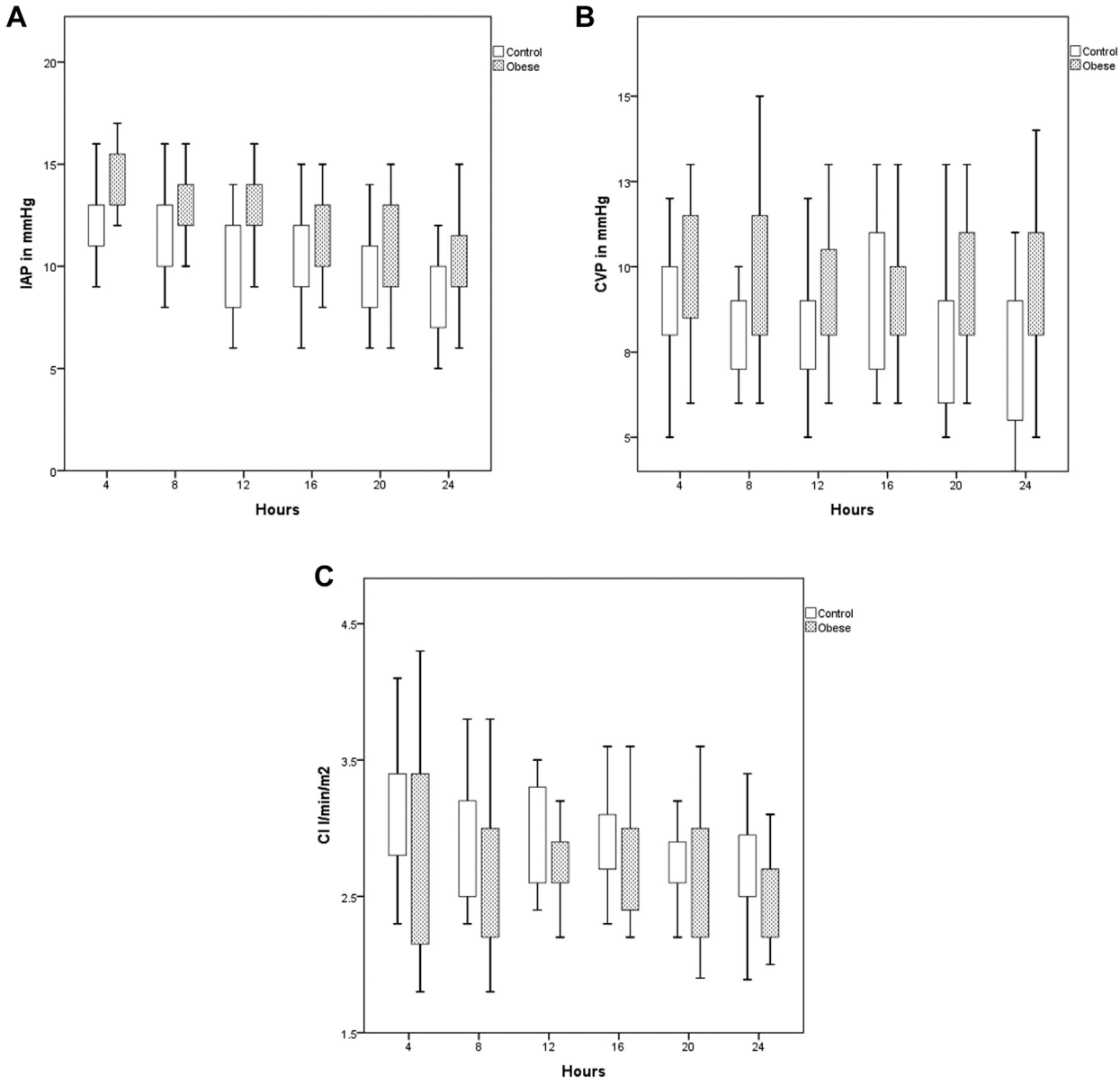

Fig 1. Comparing the (*A*) IAP, (*B*) CVP, and (*C*) CI between the obese versus the control groups at 4-h intervals in the first 24-h CABG.

Table IV
Clinical outcomes

Complications	Obese group (BMI ≥ 30)	Control group (BMI < 30)	*P* value
	n = 25	*n* = 25	
Blood and blood products transfusion	17 (68%)	15 (60%)	.556
Intra-aortic balloon pump support (%)	0	2 (8%)	.490
Myocardial infarction (%)	0	1	1.000
Low output syndrome (%)	2 (8%)	0	.490
New postoperative dialysis (%)	0	0	
Atrial fibrillation (%)	3 (12%)	1	.609
Pulmonary complications (%)	5 (20%)	2 (8%)	.417
Re-exploration surgery (%)	1	0	1.000
Stroke/TIA (%)	0	1	1.000
Surgical site infection (%)	3 (12%)	0	.235
Number of deaths (%)	0	0	

BMI, body mass index; *TIA*, transient ischemic attack.

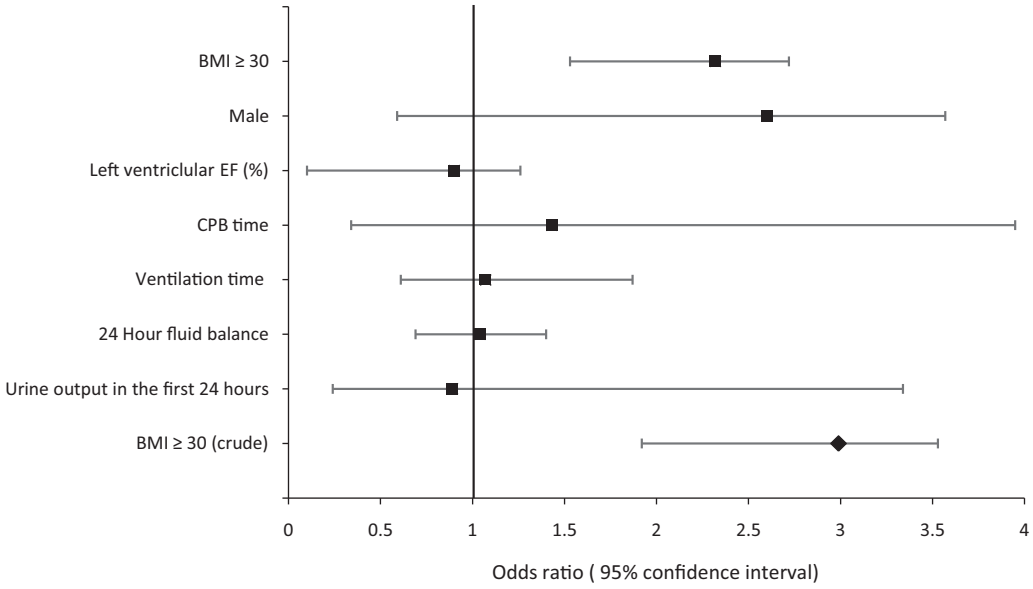

Fig 2. The crude and adjusted odds ratio of having a BMI ≥ 30 and its association with intra-abdominal hypertension.

predict IAH, likely because most patients were not obese (mean BMI in the IAH group: 26.7 ± 4 vs control group: 25.6 ± 3). Other factors found to predict IAH after cardiac surgery were CPB time, ventilation time, plasma albumin concentration, need for vasopressors, high CVP, low APP, and abdominal distension.[25] Establishing a direct association between obesity and IAH after adjusting for confounders was a key element of the current study; however, this does not imply that other important factors do not affect the development of IAH. It is likely that obesity alone seemed to overshadow other important predictors, owing to the relatively small cohort of subjects.

The calculated APP is largely affected by the presence of obesity/IAH. This might be one hypothetic reason for the apparent morbid outcomes in obese subjects undergoing CABG. We speculate that the evident decrease in APP plays a fundamental role in understanding acute kidney injury (AKI) and the possible liver dysfunction after CABG in this select population. Using various criteria for determining AKI, Mazzeffi et al[27] took serial IAP readings of 43 consecutive adult cardiac surgery patients in the ICU. Of the 42 patients, 35 developed IAH, with 100% sensitivity for predicting postoperative AKI, using the RIFLE criteria. By using calculated GFR in the present study as an indicator for AKI, we observed that obese patients developed AKI in the obese group more often than in the controls (32% vs 2%; $P = .034$). In a recent, large scale, collaborative meta-analysis of more than 5 million individuals from 39 general populations, Chang et al[28] found an association between BMI higher than 25 and the increased risk of a decrease in GFR during 8 y of mean follow-up. In contrast, there appeared to be a difference in liver function gauged by using the validated Schindl liver scores for liver dysfunction (28% vs 8%; $P = .066$), but there was a clear difference in leakage of the liver enzyme ALT in the obese group after surgery, which is a more specific indicator of ischemic liver injury.[29] None of the patients enrolled in this study developed abdominal compartment syndrome (defined as sustained IAP ≥ 20 mm Hg) nor any active intervention was sought to prevent the development of IAH. The current study has identified obesity as a risk factor for increased IAP after CABG. This finding warrants close monitoring of IAP in such patients. Although studies demonstrating the effectiveness of nonsurgical interventions in this group after CABG is lacking, general measures, such as the judicious use of fluids for resuscitation, gastric decompression via nasogastric suctioning, sedation, neuromuscular blockade, reverse Trendelenburg body position, active diuresis, and hemofiltration, are reported in the literature.[13] Operative abdominal decompression remains the definitive treatment for abdominal compartment syndrome during or after CABG.[30,31]

The strength of the present study is that it is the first to present the association between obesity and the potential development of IAH after on-pump CABG. It also presents a pathophysiologic foundation for how the kidneys and possibly the liver might be impaired because of the low APP caused by the apparent increase in the IAP in obese individuals after CABG. The current study, however, is limited by its observational nature and small cohort of selected subjects. The small sample size might have underpowered some of the more overt clinical outcomes and the competing confounding variables in the regression analysis. Moreover, the study did not seek to demonstrate any active interventions to mitigate the hazardous effects of IAH. In addition, the data collectors were not blinded to the presence of obesity in the participating subjects, which may have led to some bias.

In summary, obesity in patients undergoing CABG is generally linked with multiple adverse outcomes. Multiple factors can influence the development of IAH after cardiac surgery, but preexisting obesity seems to play a key role. The development of IAH in obese patients can lead to AKI and possible liver dysfunction. Anticipating IAH in vulnerable patients and taking measures to carefully monitor the IAP in the perioperative period may help those at risk. Nevertheless, future studies are needed to validate therapeutic interventions in such patients.

Funding/Support

Supported by the College of Medicine Research Centre, Deanship of Scientific Research, King Saud University, Riyadh, Kingdom of Saudi Arabia.

Conflict of interest/Disclosure

The authors have no disclosures.

References

1. Malbrain ML, Chiumello D, Pelosi P, et al. Incidence and prognosis of intra-abdominal hypertension in a mixed population of critically ill patients: A multiple-center epidemiological study. *Crit Care Med*. 2005;33:315–322.
2. Malbrain ML, Chiumello D, Cesana BM, et al. A systematic review and individual patient data meta-analysis on intra-abdominal hypertension in critically ill patients: the wake-up project. World initiative on Abdominal Hypertension Epidemiology, a Unifying Project (WAKE-Up!). *Minerva Anestesiol*. 2014;80:293–306.
3. Kirkpatrick AW, Roberts DJ, De Waele J, et al. Intra-abdominal hypertension and the abdominal compartment syndrome: Updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. *Intensive Care Med*. 2013;39:1190–1206.
4. Cheatham ML, Malbrain ML, Kirkpatrick A, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. II. Recommendations. *Intensive Care Med*. 2007;33:951–962.
5. Lee RK. Intra-abdominal hypertension and abdominal compartment syndrome: A comprehensive overview. *Crit Care Nurse*. 2012;32:19–31.
6. Varela JE, Hinojosa M, Nguyen N. Correlations between intra-abdominal pressure and obesity-related co-morbidities. *Surg Obes Relat Dis*. 2009;5:524–528.
7. Sugerman HJ. Effects of increased intra-abdominal pressure in severe obesity. *Surg Clin North Am*. 2001;81:1063–1075. vi.
8. Noblett KL, Jensen JK, Ostergard DR. The relationship of body mass index to intra-abdominal pressure as measured by multichannel cystometry. *Int Urogynecol J Pelvic Floor Dysfunct*. 1997;8:323–326.
9. Schein M, Ivatury R. Intra-abdominal hypertension and the abdominal compartment syndrome. *Br J Surg*. 1998;85:1027–1028.
10. Ivatury RR, Diebel L, Porter JM, Simon RJ. Intra-abdominal hypertension and the abdominal compartment syndrome. *Surg Clin North Am*. 1997;77:783–800.
11. Reintam A, Parm P, Kitus R, Kern H, Starkopf J. Primary and secondary intra-abdominal hypertension—Different impact on ICU outcome. *Intensive Care Med*. 2008;34:1624–1631.
12. Malbrain ML, Chiumello D, Pelosi P, et al. Prevalence of intra-abdominal hypertension in critically ill patients: A multicentre epidemiological study. *Intensive Care Med*. 2004;30:822–829.
13. An G, West MA. Abdominal compartment syndrome: A concise clinical review. *Crit Care Med*. 2008;36:1304–1310.
14. Smit M, Werner MJM, Lansink-Hartgring AO, Dieperink W, Zijlstra JG, van Meurs M. How central obesity influences intra-abdominal pressure: A prospective, observational study in cardiothoracic surgical patients. *Ann Intensive Care*. 2016;6:99.
15. Ghanta RK, LaPar DJ, Zhang Q, et al. Obesity increases risk-adjusted morbidity, mortality, and cost following cardiac surgery. *J Am Heart Assoc*. 2017;6.
16. Johnson DW, Jones GR, Mathew TH, et al. Chronic kidney disease and automatic reporting of estimated glomerular filtration rate: New developments and revised recommendations. *Med J Aust*. 2012;197:224–225.
17. Nazer RI, Alburikan KA, Ullah A, Albarrati AM, Hassanain M. Transient liver dysfunction increases surgical site infections after coronary surgery. *Asian Cardiovasc Thorac Ann*. 2018;26:439–445.
18. Hassanain M, Simoneau E, Madkhali A, et al. Post-transplant liver function score as an early surrogate marker of long-term outcome. *Ann Transplant*. 2015;20:198–205.
19. Hassanain M, Metrakos P, Fisette A, et al. Randomized clinical trial of the impact of insulin therapy on liver function in patients undergoing major liver resection. *Br J Surg*. 2013;100:610–618.
20. Fisette A, Hassanain M, Metrakos P, et al. High-dose insulin therapy reduces postoperative liver dysfunction and complications in liver resection patients through reduced apoptosis and altered inflammation. *J Clin Endocrinol Metab*. 2012;97:217–226.
21. De Keulenaer BL, De Waele JJ, Powell B, Malbrain ML. What is normal intra-abdominal pressure and how is it affected by positioning, body mass and positive end-expiratory pressure? *Intensive Care Med*. 2009;35:969–976.
22. Dabrowski W. Changes in intra-abdominal pressure and central venous and brain venous blood pressure in patients during extracorporeal circulation. *Med Sci Monit*. 2007;13:CR548–CR554.
23. Dabrowski W, Wacinski P, Visconti J. Abdominal perfusion pressure and coronary arterial perfusion pressure in patients undergoing coronary artery bypass graft surgery. *Exp Clin Cardiol*. 2009;14:e84–e88.
24. Dalfino L, Sicolo A, Paparella D, Mongelli M, Rubino G, Brienza N. Intra-abdominal hypertension in cardiac surgery. *Interact Cardiovasc Thorac Surg*. 2013;17:644–651.
25. Iyer D, D'Amours S, Aneman A. Intra-abdominal hypertension in postoperative cardiac surgery patients. *Crit Care Resusc*. 2014;16:214–219.
26. Malbrain ML, Cheatham ML, Kirkpatrick A, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. *Intensive Care Med*. 2006;32:1722–1732.
27. Mazzeffi MA, Stafford P, Wallace K, et al. Intra-abdominal hypertension and postoperative kidney dysfunction in cardiac surgery patients. *J Cardiothorac Vasc Anesth*. 2016;30:1571–1577.
28. Chang AR, Grams ME, Ballew SH, et al. Adiposity and risk of decline in glomerular filtration rate: Meta-analysis of individual participant data in a global consortium. *BMJ*. 2019;364:k5301.
29. Ascione R, Talpahewa S, Rajakaruna C, et al. Splanchnic organ injury during coronary surgery with or without cardiopulmonary bypass: A randomized, controlled trial. *Ann Thorac Surg*. 2006;81:97–103.
30. De Wolf A, Poelaert J, Herck I, De Waele JJ. Surgical decompression for abdominal compartment syndrome after emergency cardiac surgery. *Ann Thorac Surg*. 2008;85:2133–2135.
31. Rabbi JF, Valaulikar G, Appling NA, Bee TK, Ostrow BF, Weiman DS. Secondary abdominal compartment syndrome causing failure to wean from cardiopulmonary bypass. *Ann Thorac Surg*. 2012;93:e99–e100.