Clinical Investigation

Influence of Body Mass Index

on Outcomes of Patients Undergoing Surgery for Acute Aortic Dissection: A Propensity-Matched Analysis

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To determine whether body mass index ≥30 kg/m² affects morbidity and mortality rates in patients undergoing surgery for type A acute aortic dissection, we conducted a retrospective study of 201 patients with type A dissection. Patients were divided into 2 groups according to body mass index (BMI): nonobese (BMI, <30 kg/m²; 158 patients) and obese (BMI, ≥30 kg/m²; 43 patients). Propensity score matching was used to reduce selection bias.

The overall mortality rate was 19% (38/201 patients). The perioperative mortality rate was higher in the obese group, both in the overall cohort (33% vs 15%; P=*0.01) and in the propensity-matched cohort (32% vs 12%;* P=*0.039). In the propensity-matched cohort, patients with obesity had higher rates of low cardiac output syndrome (26% vs 6%;* P=*0.045) and pulmonary complications (32% vs 9%;* P=*0.033) than those without obesity. The overall 5-year survival rates were 52.5% ± 7.8% in the obese group and 70.3% ± 4.4% in the nonobese group (*P=*0.036). In the propensity-matched cohort, the 5-year survival rates were 54.3% ± 8.9% in the obese group and 81.6% ± 6.8% in the nonobese group (*P=*0.018).*

Patients with obesity (BMI, ≥30 kg/m²) who underwent surgery for type A acute aortic dissection had higher operative mortality rates and an increased risk of low cardiac output syndrome, pulmonary complications, and other postoperative morbidities than did patients without obesity. Additional extensive studies are needed to confirm our findings.
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The prevalence of obesity has been increasing in all industrialized countries.[1] Obesity is associated with a wide array of pathophysiologic changes, such as impairment of cardiac, pulmonary, and immunologic functions, and with a high rate of comorbidities.[2,3] However, the role of obesity as an independent risk factor for perioperative complications and death has not been clarified. Investigators studying cardiac surgical populations have generally focused on patients undergoing coronary artery bypass grafting or valve surgery, and have reported mixed results. In older studies, obesity was identified as an important risk factor that can influence surgical outcomes, but patients with obesity have not experienced worse outcomes than those without obesity in recent series.[4-11] Type A acute aortic dissection (AAD) is a life-threatening condition, with operative mortality rates ranging from 8% to 34%.[12,13] There is a lack of studies that specifically address the impact of obesity on surgical outcomes of AAD repair.

The aim of this study was to determine whether body mass index (BMI) ≥30 kg/m² affects morbidity and short- and midterm mortality rates in patients undergoing surgery for type A AAD.

Patients and Methods

We conducted a retrospective cohort study of 201 consecutive patients with type A AAD who underwent emergency surgery at our hospital from January 2006 through July 2013. The hospital ethics committee approved this study and waived the requirement for obtaining informed consent. Body mass index was calculated in standard fashion (BMI = weight in kg / height in m²). Patients were divided into 2 groups according to the World Health Organization definition of obesity[1]: nonobese group

(BMI, <30 kg/m^2; 158 patients) and obese group (BMI, ≥30 kg/m^2; 43 patients).

For the purpose of this study, AAD was defined as a dissection managed surgically within 14 days after onset of symptoms. Visceral ischemia was defined as a reduction in blood flow to the gastrointestinal tract caused by the blockage of a blood vessel, which was diagnosed clinically and confirmed by computed tomography. Low cardiac output syndrome (LCOS) was defined as a severe reduction in cardiac index (<2.2 L/min/m^2) or a need for prolonged inotropic support (>48 hr). Pulmonary complications referred to the need for prolonged mechanical ventilation (>48 hr), including noninvasive ventilation or reintubation for respiratory insufficiency or pneumonia. Infection referred to any localized or systemic infection, including chest, urinary, wound, and graft infection, as well as septicemia.

The primary endpoints were perioperative morbidity and mortality (defined as any death occurring within 30 d after surgery or before discharge from the hospital) and midterm survival.

Surgical Techniques

A standard median sternotomy was routinely performed in patients with AAD. Cardiopulmonary bypass (CPB) was established by cannulating the axillary or femoral artery or the ascending aorta and the venae cavae. Myocardial protection was achieved by using intermittent antegrade blood cardioplegia or single-dose crystalloid cardioplegia. Left ventricular venting was performed through the right superior pulmonary vein. Core body temperature was monitored in the bladder with a Foley catheter and in the esophagus with a nasopharyngeal probe. Hypothermia was used in all cases. The core temperature was allowed to drift down to between 21 °C and 33 °C, depending on the anticipated period of circulatory arrest. Generally, when circulatory arrest was projected to last up to 10 min, a temperature of 30 °C to 33 °C was considered acceptable; if a period of arrest >30 min was anticipated, a core temperature between 21 °C and 25 °C was considered safe. Continuous cerebral perfusion was used during circulatory arrest periods >20 min. Unilateral cerebral perfusion was achieved by clamping the origin of the brachiocephalic artery and using axillary arterial inflow, whereas bilateral cerebral perfusion was established through the Kazui technique, in which the brachiocephalic and left common carotid arteries were cannulated and perfused at a rate of 10 mL/(kg·min) by a single pump. In all cases, the aortic arch was inspected and distal anastomosis was performed under circulatory arrest. The decision to perform concomitant aortic root surgery, arch repair, or both was based on each patient's condition, intimal tear site, or diameter of the aortic root and arch, but was ultimately left to the discretion of the attending surgeon.

Follow-Up

Midterm outcomes were determined from clinical records, when available, or from telephone interviews with patients or family members, when necessary. All follow-up data were collected, and no patients were lost to follow-up. Median and mean duration of follow-up were 23 months (interquartile range, 0–51 mo) and 28 ± 27 months (range, 0–96 mo), respectively.

Statistical Analysis

Continuous data were presented as mean ± SD or as median with the interquartile range, and categorical data were expressed as percentages. The Student *t* test was used to compare continuous variables when normal distribution was present, as confirmed by the Kolmogorov-Smirnov test. The Mann-Whitney test was used for nonnormally distributed variables. Categorical variables were compared with the χ^2 test. The Fisher exact test was used for small group sizes (n <5). Kaplan-Meier curves were generated to provide survival estimates at postoperative points in time. Differences between the 2 groups were determined by log-rank tests. These estimates included operative deaths. All reported *P* values were 2-sided, and *P* values <0.05 indicated statistical significance.

To reduce selection bias, a propensity score was calculated by logistic regression, with obesity (BMI, ≥30 kg/m^2) as the dependent variable. All the variables listed in Table I (except for BMI class) and several others (for example, site of cannulation, type of cerebral perfusion, and concomitant surgical procedures) were included in the analysis. A propensity score-matched cohort was constructed by 1:1 nearest neighbor matching without replacement. We calculated standardized differences for variables to evaluate postmatch balance. A standardized difference <10% was considered an acceptable imbalance. All statistical analyses were conducted using SPSS 15.0 (SPSS, an IBM company).

Results

Overall Cohort

Table I shows the baseline characteristics of the 201 patients with type A AAD. No patients were underweight. The obese group had a higher percentage of male patients. There were no differences in the prevalence of diabetes mellitus, hypertension, history of smoking, left ventricular ejection fraction, or other preoperative factors.

Table II shows the operative variables for all patients. Cardiopulmonary bypass time was significantly longer in the obese group than in the nonobese group (*P*=0.03).

Table III shows the postoperative results. The perioperative mortality rates were 33% in the obese group and 15% in the nonobese group (*P*=0.01). Postoperative LCOS occurred significantly more frequently in the

TABLE I. Baseline Characteristics of the 201 Patients

Variable	Overall Cohort			PSM Cohort		
	Obese (n=43)	Nonobese (n=158)	*P* Value	Obese (n=34)	Nonobese (n=34)	*P* Value
Age (yr)	60 ± 13	63 ± 12	0.18	59 ± 11	58 ± 13	0.27
Female	6 (14)	48 (30)	0.031	4 (12)	3 (9)	1.0
Body mass index (kg/m^2)	33.95 ± 5.19	25.15 ± 2.59	<0.001	33 ± 3.81	25.31 ± 2.76	<0.001
Underweight (<18.5)	0	0	—	0	0	—
Normal (18.5–24.9)	0	104 (66)	<0.001	0	23 (68)	<0.001
Overweight (25–29.9)	0	54 (34)	<0.001	0	11 (32)	<0.001
Class I obesity (30–34.9)	33 (77)	0	<0.001	29 (85)	0	<0.001
Class II obesity (35–39.9)	4 (9)	0	0.002	2 (6)	0	0.15
Class III obesity (≥40)	6 (14)	0	<0.001	3 (9)	0	0.076
Diabetes mellitus	2 (5)	7 (4)	1.0	2 (6)	2 (6)	1.0
Dyslipidemia	8 (19)	10 (6)	0.012	6 (18)	5 (15)	0.74
Hypertension	37 (86)	138 (87)	0.82	30 (88)	30 (88)	1.0
History of smoking	14 (33)	49 (31)	0.85	12 (35)	10 (29)	0.6
Recent MI	1 (2)	3 (2)	1.0	1 (3)	1 (3)	1.0
Renal dialysis	0	3 (2)	1.0	0	0	—
LVEF	0.46 ± 0.13	0.51 ± 0.05	0.19	0.45 ± 0.16	0.49 ± 0.02	0.42
Previous operations	3 (7)	6 (4)	0.41	2 (6)	1 (3)	1.0
Cardiogenic shock	5 (12)	10 (6)	0.24	3 (9)	2 (6)	0.54
Malperfusion						
Coronary	5 (12)	15 (9)	0.68	3 (9)	3 (9)	1.0
Cerebral	8 (19)	24 (15)	0.58	6 (18)	5 (15)	0.74
Iliofemoral	4 (9)	13 (8)	0.76	3 (9)	3 (9)	1.0
Gastrointestinal	2 (5)	7 (4)	1.0	1 (3)	1 (3)	1.0

LVEF = left ventricular ejection fraction; MI = myocardial infarction; PSM = propensity score-matched

Data are presented as mean ± SD or as number and percentage. $P<0.05$ was considered statistically significant.

obese group (21% vs 8%; *P*=0.018). In addition, patients with obesity had higher rates of visceral ischemia (11% vs 4%; *P*=0.077) and renal failure necessitating continuous venovenous hemofiltration (CVVH) (18% vs 9%; *P*=0.096), but these differences did not reach statistical significance. Mean intensive care unit stay and hospital stay were similar for the 2 groups.

There were 31 late deaths, including 18 related to cardiovascular events; 6 deaths (14%) occurred in the obese group and 25 (16%) in the nonobese group (*P*=0.76). Overall 5-year survival rates were 52.5% ± 7.8% in the obese group and 70.3% ± 4.4% in the nonobese group (*P*=0.036) (Fig. 1).

Propensity Score-Matched Cohort

Propensity score-matching resulted in 34 pairs with similar baseline characteristics and similar operative variables (Tables I and II). The propensity-matched cohorts were equal-sized and well matched, and there were no differences in covariates between the 2 groups.

In the propensity-matched cohort, the operative mortality rates were 32% in the obese group and 12% in the nonobese group (*P*=0.039) (Table III). Patients with obesity had higher rates of LCOS (26% vs 6%; *P*=0.045) and pulmonary complications (32% vs 9%; *P*=0.033). Renal failure necessitating CVVH (21% vs 6%; *P*=0.15) and infections (12% vs 0; *P*=0.11) were more frequent in patients with obesity, but the differences were not statistically significant. Ventilation time (*P*=0.062) and mean intensive care unit stay (*P*=0.05) were longer in the obese group.

Of the 6 late deaths, 4 (12%) occurred in the obese group and 2 (6%) in the nonobese group (*P*=0.34). The 5-year survival rates were 54.3% ± 8.9% in the obese group and 81.6% ± 6.8% in the nonobese group (*P*=0.018) (Fig. 2).

TABLE II. Comparison of Operative Characteristics

Variable	Overall Cohort			PSM Cohort		
	Obese (n=43)	Nonobese (n=158)	*P* Value	Obese (n=34)	Nonobese (n=34)	*P* Value
Femoral artery cannulation	19 (44)	73 (46)	0.79	18 (53)	18 (53)	1.0
Axillary artery cannulation	20 (47)	70 (44)	0.82	13 (38)	14 (41)	0.8
Ascending aortic cannulation	4 (9)	15 (9)	0.87	3 (9)	2 (6)	0.54
CPB time (min)	193 ± 99	155 ± 75	0.03	165 ± 93	165 ± 92	0.99
Cardiac ischemic time (min)	93 ± 42	94 ± 47	0.84	98 ± 48	100 ± 43	0.84
Duration of CA (min)	23 ± 24	23 ± 32	0.99	18 ± 25	18 ± 26	0.97
Temperature at CA (°C)	25 ± 7	23 ± 9	0.38	23 ± 9	20 ± 13	0.14
Unilateral antegrade perfusion	13 (30)	34 (22)	0.23	8 (24)	6 (18)	0.55
Bilateral antegrade perfusion	13 (30)	48 (30)	0.98	9 (26)	10 (29)	0.78
Concomitant procedures						
Bentall	11 (26)	28 (18)	0.25	9 (26)	9 (26)	1.0
AV replacement	4 (9)	13 (8)	0.76	4 (12)	2 (6)	0.67
AV repair	0	6 (4)	0.34	0	0	—
CABG	5 (12)	8 (5)	0.12	3 (9)	3 (9)	1.0
Hemiarch repair	12 (28)	45 (28)	0.94	8 (24)	8 (24)	1.0
Total arch repair	10 (23)	24 (15)	0.21	7 (21)	5 (15)	0.52
Frozen elephant trunk	3 (7)	1 (0.6)	0.031	0	0	—

AV = aortic valve; CA = circulatory arrest; CABG = coronary artery bypass grafting; CPB = cardiopulmonary bypass; PSM = propensity score-matched

Data are presented as number and percentage or as mean ± SD. $P<0.05$ was considered statistically significant.

Discussion

The role of obesity in the prognosis of patients undergoing cardiac surgery remains unclear. In the past, obesity was considered a risk factor in cardiac patients, as demonstrated by its inclusion as a criterion in the Parsonnet system for stratifying risk for perioperative death.[14,15] More recent findings have suggested that patients with obesity who undergo coronary artery bypass grafting or valve surgery may not have worse outcomes than underweight or normal-weight patients who undergo similar procedures.[7,8,10-11]

Investigators have described the relationship between BMI and postoperative death as an obesity paradox,[8] reporting the highest risk for postoperative death in underweight patients and in those with morbid obesity (BMI, ≥40 kg/m^2).[5,6] Moreover, some researchers have reported a survival benefit for patients with obesity, whereas others have found no association between obesity and long-term survival after cardiac surgery.[7,9]

Our understanding of the role that BMI plays in shaping outcomes of cardiac surgery remains incomplete, and there is currently a lack of studies that specifically address the impact of obesity on the surgical outcomes of patients undergoing surgery for type A AAD. Therefore, we investigated the effect of obesity on morbidity and short- and midterm mortality rates in patients with type A AAD who underwent surgical treatment at our hospital.

An important finding of our study was that AAD did not occur in underweight patients; none of the patients admitted to our hospital had a BMI <18.5 kg/m^2. This result may be attributed to the lower incidence of hypertension in underweight patients.

The overall operative mortality rate in our cohort was 19% (38/201), and it was significantly higher in patients with obesity (BMI, ≥30 kg/m^2) than in those without obesity (33% vs 15% in the overall population, P=0.01; 32% vs 12% in the PSM cohort, P=0.039). In addition, patients with obesity had a higher incidence of severe postoperative complications. In particular, LCOS occurred more frequently in patients with obesity, both in the entire cohort and in the propensity-matched population. The association between obesity and LCOS has been previously reported.[2,16,17] There were no differences in CPB or cardiac ischemic times between the 2 groups in our study. Theories about the mechanisms underlying cardiac postoperative complications in patients with obesity include an increased myocardial workload, inadequate myocardial protection of the hypertrophied heart,

TABLE III. Comparison of Postoperative Results

Variable	Overall Cohort			PSM Cohort		
	Obese (n=43)	Nonobese (n=158)	*P* Value	Obese (n=34)	Nonobese (n=34)	*P* Value
Hospital death	14 (33)	24 (15)	0.01	11 (32)	4 (12)	0.039
Reexploration for bleeding	4 (9)	25 (16)	0.34	4 (12)	3 (9)	1.0
Permanent neurologic injury	6 (14)	9 (6)	0.068	4 (12)	0	0.11
Transient neurologic injury	1 (2)	9 (6)	0.69	1 (3)	1 (3)	1.0
LCOS	9 (21)	13 (8)	0.018	9 (26)	2 (6)	0.045
Acute myocardial infarction	2 (5)	1 (0.5)	0.12	1 (3)	1 (3)	1.0
Acute renal failure	10 (23)	28 (18)	0.41	8 (23)	5 (15)	0.35
Postoperative CVVH	8 (19)	15 (9)	0.096	7 (21)	2 (6)	0.15
Visceral ischemia	5 (12)	7 (4)	0.077	4 (12)	2 (6)	0.67
Pulmonary complications	13 (30)	33 (21)	0.19	11 (32)	3 (9)	0.033
Tracheostomy	5 (12)	11 (7)	0.31	4 (12)	1 (3)	0.35
Infection or sepsis	5 (12)	10 (6.5)	0.24	4 (12)	0	0.11
Atrial fibrillation	9 (21)	35 (22)	0.86	7 (21)	9 (26)	0.57
Ventilation time (hr)	105 ± 192	104 ± 204	0.98	113 ± 200	36 ± 50	0.062
ICU stay (d)	7.5 ± 11	7 ± 11	0.76	8 ± 12	3 ± 2	0.05
Hospital stay (d)	15 ± 17	13 ± 12	0.58	16 ± 18	12 ± 12	0.22
RBC units (n)	8 ± 8	7 ± 7	0.65	6 ± 8	6 ± 7	0.9
Platelet units (n)	4 ± 4	4 ± 5	0.83	3 ± 3	4 ± 4	0.56
FFP units (n)	5 ± 4	5 ± 4	0.57	4 ± 4	4 ± 3	0.73

CVVH = continuous venovenous hemofiltration; FFP = fresh frozen plasma; ICU = intensive care unit; LCOS = low cardiac output syndrome; PSM = propensity score-matched; RBC = red blood cell

Data are presented as mean ± SD or as number and percentage. $P < 0.05$ was considered statistically significant.

and an imbalance between myocardial oxygen demand and supply.[17] Obesity has also been linked to left ventricular morphologic changes and impaired diastolic and systolic function, even in the absence of coronary artery disease.[2] Moreover, the high risk of LCOS in patients with obesity may be attributed to gene–environment interactions that alter cardiovascular metabolism.[16]

Another finding of our study is the association between obesity and pulmonary complications. Obesity has been associated with impaired pulmonary function, including increased residual lung volume, decreased lung compliance and increased chest wall impedance, ventilation-perfusion abnormalities, depressed ventilatory drive, and bronchospasm.[18] Similar to the findings in our series, the Society of Thoracic Surgeons database study showed that obesity and morbid obesity were associated with prolonged ventilation, with respective adjusted odds ratios of 1.49 and 1.70.[5]

In general, patients with obesity have a greater risk for developing acute kidney injury related to underlying structural changes that occur in the kidneys despite normal serum chemistry.[19,20] Morbid obesity has been associated with an increased risk of acute renal failure after CPB.[11,19] In our study, the rate of postoperative renal failure and the need for CVVH were slightly higher in the obese group, but the differences were not statistically significant.

Higher rates of infection or sepsis have also been reported in patients with obesity compared with patients without obesity. This correlation may be related to an underlying impairment of the immune responsiveness of lymphocytes in patients with obesity, which might contribute to increasing rates of infection.[3]

In our study, patients with obesity showed no increased hemorrhagic tendency. There were no differences between the 2 groups in reexploration for bleeding, and patients with obesity did not need more frequent transfusions of blood products than patients without obesity. The need for blood transfusions in patients with obesity undergoing cardiac surgery has been evaluated in only a few studies with small cohorts, and the results have been mixed. Uva and colleagues[21] found an increased transfusion rate in this population, but Ranucci and associates[22] found that the transfusion rate decreased.

Finally, survival rates were significantly lower in the obese group than in the nonobese group. The overall

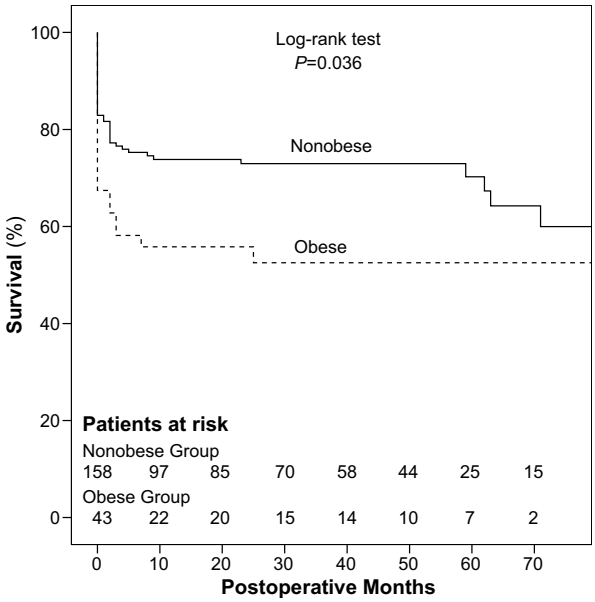

Fig. 1 *Kaplan-Meier survival curves in the overall cohort: overall 5-year survival rates were 52.5% ± 7.8% in the obese group and 70.3% ± 4.4% in the nonobese group (*P*=0.036).* P *<0.05 was considered statistically significant.*

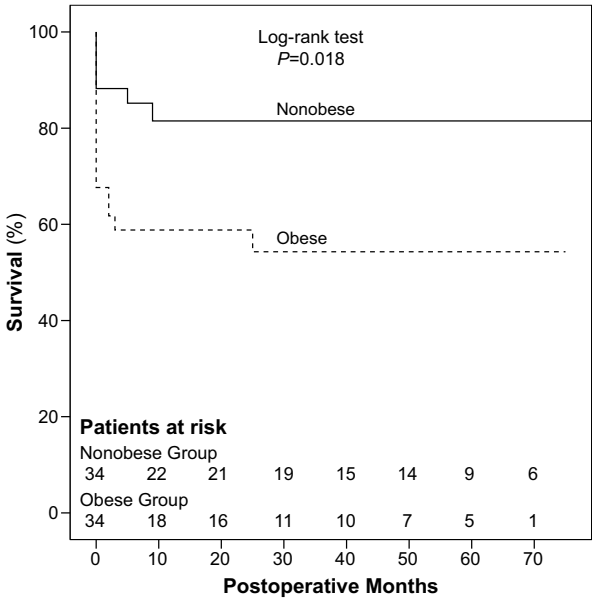

Fig. 2 *Kaplan-Meier survival curves in the propensity score-matched cohort: 5-year survival rates were 54.3% ± 8.9% in the obese group and 81.6% ± 6.8% in the nonobese group (*P*=0.018).* P *<0.05 was considered statistically significant.*

5-year survival rates were 52.5% ± 7.8% in the obese group and 70.3% ± 4.4% in the nonobese group (*P*=0.036). In the propensity-matched cohort, the 5-year survival rates were 54.3% ± 8.9% in the obese group and 81.6% ± 6.8% in the nonobese group (*P*=0.018). This difference may be related to the higher perioperative mortality rates in patients with obesity. Our results showed that the incidence of late death was similar in the 2 groups, with no statistically significant differences. However, in the propensity-matched population, the association between obesity and late mortality was greater. We speculate that obesity could emerge as a predictor for late death, but additional data, as well as longer follow-up periods, are needed to confirm this assumption.

Study Limitations

The main limitations of our study are the retrospective nature of the analysis and the small sample size. In our study cohort, only a small number of patients had class II and III obesity, with most patients being categorized in class I. For this reason, we chose to treat obesity as a dichotomous variable and did not use BMI as a continuous variable. In addition, our study had a median follow-up period of 23 months. Larger studies with longer follow-up periods may provide additional insight into the impact of obesity on long-term outcomes. Although propensity-score matching was performed to exclude potential confounding factors, unmeasured confounders and the relatively small sample size may have resulted in bias. Moreover, it is important to consider that BMI does not discriminate between fat and lean mass, and, therefore, BMI does not adequately reflect adiposity. Other measures, such as waist circumference, might be more informative.

Conclusions

Patients with obesity (BMI, ≥30 kg/m^2) who underwent surgery for type A AAD had higher rates of perioperative mortality and postoperative morbidity than did those without obesity, with a statistically significant increase in the incidence of LCOS and pulmonary complications. Additional, larger studies are necessary to confirm our findings.

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