Cardiology in the Young

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The prognostic significance of early troponin levels in patients undergoing aortic ridge surgery

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Abstract

Subaortic stenosis is a CHD that can lead to left ventricular hypertrophy, heart failure, and aortic valve damage if left untreated. The gold standard treatment for subaortic stenosis is septal myectomy. However, there is no clear consensus on the surgical margins required for adequate muscle resection. In this retrospective study, we reviewed the records of 83 patients who underwent subaortic stenosis surgery between 2012 and 2020 to investigate the effect of early troponin levels on prognosis. We excluded patients with additional cardiac pathologies, hypertrophic obstructive cardiomyopathy, and valvular aortic stenosis.

Troponin levels were recorded in the early post-operative period, and patients were monitored for complications such as ventricular arrhythmia, left ventricular systolic dysfunction, infective endocarditis, and pacemaker implantation. The troponin levels were significantly higher in the patients who had septal myectomy. The degree of myectomy affected the risk of complications in the early post-operative period and recurrence in the later period. However, when the gradient was substantially or completely removed by myectomy, patients experienced significant symptom improvement in the early post-operative period, and their late survival was equivalent to that of healthy individuals of the same age.

Our findings suggest that monitoring troponin levels in patients undergoing septal myectomy may be beneficial in predicting the risk of complications. However, further studies are needed to establish the optimal surgical technique and extent of muscle resection required for subaortic stenosis treatment. Our study adds to the existing knowledge of the benefits and risks associated with septal myectomy as a treatment option for subaortic stenosis.

In the presence of two equal ventricles, obstruction under the aortic valve and left ventricular outflow tract is called subaortic stenosis. Left ventricular outflow tract stenosis is present in 3–8% of CHDs, and subaortic stenosis is present in 8–20% of left ventricular outflow tract stenoses. The most common accompanying cardiac pathology is VSD with a rate of 20–65%. It has progressive and repetitive features. Patients are usually asymptomatic up to a certain gradient. Specific findings such as chest pain, syncope, palpitations, exertional dyspnoea, and heart failure occur in the advanced and late stages in direct proportion to the increasing aortic gradient. If severe forms of subaortic stenosis are left untreated, complications such as aortic valve damage, left ventricular hypertrophy, left ventricular systolic dysfunction, heart failure, ventricular arrhythmia, and infective endocarditis develop.[1–3]

Abnormal geometry in these patients causes chronic turbulence in the subvalvular region. Turbulent blood flow initiates fibrin formation and platelet deposition by damaging the endothelium. Subaortic stenosis develops as a result of endothelial transformation against flow stress, abnormal cell proliferation, and stimulation of growth factors.[4] The aim of surgery is to remove all structures that cause turbulence in the subaortic region and prevent complications such as ventricular hypertrophy, aortic valve injury, and infective endocarditis. Subaortic stenosis surgery encompasses complex surgery ranging from resection of fibrous tissue to septal myotomy or myectomy, reconstruction of the base of the heart, single or double valve replacement, use of an apicoaortic conduit, and cardiac transplantation.[5]

Although routine septal myectomy was recommended after 1966 in addition to performing isolated membrane resection, reports have been reported combining both isolated fibrous tissue resection and membrane resection with myectomy. The difference in surgical strategy still continues. Although there is no consensus on definitively performing myectomy in subaortic stenosis surgery, it is more prevalent to perform routine myectomy in every patient.[6]

Cardiac troponins are specific and sensitive enzymes for monitoring heart muscle function and damage. They are very sensitive markers for the detection of myocardial injury and have revolutionised the concepts of minor myocardial injury and infarction. In addition, they are strong prognostic indicators of future adverse cardiac events.[7,8] We will report with you the effect of troponin levels in the early post-operative period, which increases in direct proportion to the size of myocardial tissue resected, on prognosis, post-operative gradient change, and our own clinical experience in patients operated for aortic ridge.

Materials and methods

In our study, 83 patients who were operated for subaortic stenosis in our cardiac surgery clinic from 2012 to 2020 were retrospectively analysed. The patients' age, gender, weight, diagnosis, past operations, and applied operation were obtained from the hospital archive and hospital data processing system. Our study is a retrospective, observational, single-centre study. Troponin T values of the patients were recorded in the post-operative period. Patients with additional cardiac pathologies, HOCM, and valvular aortic stenosis were excluded, and only patients with isolated subvavular aortic stenosis were included.

In the pre-operative period, echocardiography was performed to measure the mean gradients in the doppler mode, and the severity and location of the stenosis were evaluated by transcatheter diagnostic angiography, when necessary. Surgery was planned for patients with subaortic stenosis with a gradient greater than 30 mmHG on echocardiography, and for patients with left ventricular hypertrophy, newly developing aortic regurgitation or symptoms.

Vasoactive inotrope score was calculated as follows: Dopamine dose (mg/kg/min) + dobutamine dose (mg/kg/min) + 100 × dose of epinephrine (mg/kg/min) + 100 × dose of norepinephrine (mg/kg/min) + 10,000 × dose of vasopressin (U/kg/min) + 10 × milrinone dose (mg/kg/min).

Median sternotomy was used as incision in all patients, and all cases were performed under cardiopulmonary bypass by cannulating the aorta and atrium. During surgery, the patient was cooled to 28–32°C (moderate hypothermia). The right upper pulmonary vein was used for venting. Antegrade blood, del nido, or custadiol cardioplegia were applied according to the surgeon's preference. After cross-clamp, transverse aortotomy was performed. Aortic valves and subvalvular region were dissected and examined. Fibroelastic tissue was completely excised. In cases with tunnel-shaped complex stenosis, the resection was expanded by removing the right/left lower muscle tissue as well as the fibrous tissue. Gradients of all patients were evaluated with transesophageal echocardiogram in the post-operative period. A more aggressive approach to stenosis was used in reoperations. Fibrotic tissues and muscle tissue were resected more extensively. The Ross–Konno procedure was applied in the presence of severe subaortic obstruction and unrepairable aortic valve disease and was not included in the study.

It was done retrospectively, in accordance with the Declaration of Helsinki, taking into account the ethical rules. Informed consent was obtained from the patients and their relatives before the operation.

NCSS (Number Cruncher Statistical System) 2007 (Kaysville, Utah, USA) programme was used for statistical analysis. While evaluating the study data, descriptive statistical methods (mean, standard deviation, median, frequency, ratio, minimum, maximum) as well as the distribution of the data were evaluated with the Shapiro–Wilk test. Kruskall–Wallis test was used in the comparison of quantitative data of three or more groups that do not show normal distribution; Mann–Whitney U test was used to compare two groups that did not show normal distribution. Friedman test was used for comparisons of three or more periods, and Wilcoxon test was used for comparisons of two periods. Spearman's correlation analysis was used to determine the relationship between quantitative data. Significance was evaluated at $p < 0.01$ and $p < 0.05$ levels.

Results

While 55.4% (n = 46) of the patients were male, 44.6% (n = 37) were female. In the pre-operative echocardiography of the patients included in the study, 26.5% (n = 22) of the aortic valves were biscuspid, while 73.5% (n = 61) were tricuspid. While 14.5% (n = 12) of the cardioplegia solutions used during the operation is custadiol, 14.5% (n = 12) is delnido and 71% (n = 59) is blood. ECMO was required in 3.6% (n = 3) of the patients. Mortality during hospital stay was calculated as 7.2% (n = 6). 19.2% of the patients (n = 16) were reoperation cases. Two patients with death were in the reoperation group.

Pacemaker implantation was required due to AV block in five of the patients who underwent septal myectomy (6%). There was no statistically significant difference between the post-operative troponin T values of the patients who developed AV complete block and the other patients ($p > 0.05$).

Age at the time of the operation (years), body mass index (m^2/cm^2), body surface area, TROP-T, length of hospital stay (days), cross clamp time (minutes), cardiopulmonary bypass time (minutes), total operation time (minutes), VIS score, drainage (cc), intubation time (days), ECMO time (days), and intensive care hospitaliwation (days) values are given in Table 1.

When patients are compared according to biscuspid or tricuspid valve; patients' age (years), body mass index (m^2/cm^2), body surface area, TROP-T, length of hospital stay (days), VIS score, drainage (cc), intubation time (days), ECMO duration (days), and intensive care hospitalisation duration (days) do not show a statistically significant difference ($p > 0.05$).

When the patients are compared according to whether they have a biscuspid or tricuspid valve, there is a statistically significant difference in the values of cross clamp time (minutes), cardiopulmonary bypass time (minutes), total operation time (minutes), and post-operative intubation time (days) ($p < 0.05$).

As shown in Table 2, there is no statistically significant difference in age at the time of operation (years), body mass index (m^2/cm^2), body surface area, TROP-T, VIS score, intubation time (days) according to cardioplegia type ($p > 0.05$). When compared according to cardioplegia type, there were statistically significant difference between the values of patients' length of hospital stay (days), cross clamp time (minutes), cardiopulmonary bypass time (minutes), total operation time (minutes), ECMO time (days), intensive care hospitalisation (days), and drainage (cc) ($p < 0.05$).

Lvedd value does not show a statistically significant difference according to the periods ($p > 0.05$) The IVSD value does not show a statistically significant difference according to the periods ($p > 0.05$) The Ivedd value shows a statistically significant difference according to the periods ($p = 0.023$; $p < 0, 05$). It was found statistically significant that the third measurement value was higher than the other periods ($p = 0.001$; $p < 0.01$) (Table 3).

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Table 1. Measurement averages

	Ort±Ss	Min-Max (Median)
Age at operation (years)	7.43 ± 5.33	1–22 (6)
Body mass index (m^2/cm^2)	17.67 ± 4.69	9.55–39.16 (16.64)
Body surface area (m^2)	0.92 ± 0.42	0.2–1.75 (0.87)
TROP-T (ng/mL)	3.86 ± 2.99	0.3–10 (2.8)
Length of hospital stay (days)	18.87 ± 127.48	–359–1103 (7)
Cross clamp time (minutes)	73.47 ± 49.71	14–290 (62)
Cardiopulmonary bypass time (minutes)	109.82 ± 77.17	28–526 (90)
Total operation time (minutes)	231.19 ± 90.38	90–585 (225)
VIS	13.98 ± 7.98	0–32 (15)
Drainage (cc)	172.77 ± 121.15	20–650 (150)
Intubation period (days)	8.86 ± 10.81	0.5–70 (4)
ECMO duration (days)	0.57 ± 3.09	0–21 (0)
Intensive care Hospitalisation (days)	38.96 ± 30.69	4–139 (22)

In the pre-operative and post-operative comparison, it was statistically significant that the htc value of the second measurement was lower than the first measurement ($p = 0.001$; $p < 0.01$). The gradient value in the second measurement in the subaortic area was found to be lower than the first measurement ($p = 0.001$; $p < 0.01$) (Table 4). In the pre-operative and post-operative comparison, it was statistically significant that the htc value of the second measurement was lower than the first measurement ($p = 0.001$; $p < 0.01$). The gradient value in the second measurement in the subaortic area was found to be lower than the first measurement ($p = 0.001$; $p < 0.01$) (Table 4).

According to Spearman correlation, there is a weak and positive correlation between troponin-t and total operation time ($r = 0.231$, $p < 0.05$). There is a positive and weakly significant relationship between trop-t and drainage ($r = 0.223$, $p < 0.05$). There is a positive and weakly significant correlation between Trop-t and intubation time ($r = 0.262$, $p < 0.05$). There is a positive and weakly significant correlation between trop-t and ecmo duration ($r = 0.245$, $p < 0.05$). There is no statistically significant relationship between Trop-t and total hospital stay, cross clamp time, cardiopulmonary bypass time, ultrafiltration, overall balance, milrinone, adrenaline, dopamine, dobutamine, vis, length of stay in ICU, initial gradient pressure, second gradient pressure, and gradient difference ($p > 0.05$). According to Spearman correlation, there is a weak and positive correlation between Troponin-t and total operation time ($r = 0.231$, $p < 0.05$). There is a positive and weakly significant relationship between trop-t and drainage ($r = 0.223$, $p < 0.05$). There is a positive and weakly significant correlation between Trop-t and intubation time ($r = 0.262$, $p < 0.05$). There is a positive and weakly significant correlation between trop-t and ecmo duration ($r = 0.245$, $p < 0.05$). There is no statistically significant relationship between Trop-t and total hospital stay, cross clamp time, cardiopulmonary bypass time, ultrafiltration, overall balance, milrinone, adrenaline, dopamine, dobutamine, vis, length of stay in ICU, initial gradient pressure, second gradient pressure, and gradient difference ($p > 0.05$).

While the mitral regurgitation grade was mild in 22.9% (n = 19) of the patients preoperatively, 18.1% (n = 15) had moderate, 4.8% (n = 4) had advanced, and 54.2% (n = 45) had no mitral regurgitation. While the postoperative mitral regurgitation grade was mild in 36.1% (n = 30) of the patients, 6% (n = 5) had moderate, 1.2% (n = 1) had advanced, and 56.6% (n = 47) had no mitral regurgitation.

While the pre-operative aortic regurgitation grade was mild in 41% (n = 34) of the patients, 28.9% (n = 24) had moderate, 3.6% (n = 3) had advanced, and 26.5% (n = 22) had no aortic regurgitation. In the post-operative period, while the aortic regurgitation grade was mild in 47% (n = 39) of the patients, 21.7% (n = 18) had moderate, 3.6% (n = 3) had advanced, and 27.7% (n = 23) had no aortic regurgitation.

Postoperatively, 8.4% (n = 7) of the patients had mild aortic stenosis, 10.8% (n = 9) had moderate, 18.1% (n = 15) had advanced, and 62.7% (n = 52) had no aortic stenosis.

The parameters of the alive and death patients are compared in Table 5. Statistically significant differences were found as age at operation (years), body mass index (m^2/cm^2), body surface area (m^2), Trop-T (ng/mL), length of stay in hospital (days), cardiopulmonary bypass time (min), operation time (min), drainage (cc), intubation duration (days), and ecmo duration (days).

Discussion

Despite successful results with surgical treatment in children with subaortic stenosis, it is a rare congenital lesion that frequently recurs. The disease has a wide spectrum ranging from a simple short lesion to complex forms that require complex surgical reconstructions.[9] Although it is a low-mortality surgical operation, morbidities such as total AV heart block due to conduction system damage, aortic and mitral valve damage, and iatrogenic VSD formation can be seen at different rates in patients during myectomy performed to widen the left ventricular outflow tract, depending on the experience of the centre.

Carlson et al. investigated pre-operative risk factors in recurrent subaortic stenosis. They found that age, distance between the aortic valve and aortic ridge tissue, and additional left heart lesion were risk factors in recurrent subaortic stenosis. It has been stated that patients younger than 2 years of age pose a risk for recurrent aortic ridge.[10] Age was found to be an important risk factor for mortality in the living and ex-patient group in our study group, which supports the literature.

The jet stream hitting the aortic valve due to turbulence and systolic ejection damages the leaflets of the valve, causing thickening of the leaflets and insufficient coaptation, resulting in aortic valve insufficiency. Up to 15% of untreated patients with subaortic stenosis develop aortic regurgitation 1–10 years after diagnosis. Therefore, early surgery for subaortic stenosis is helpful in preventing the development and progression of significant aortic regurgitation.[2] Post-operative aortic regurgitation grade was mild in 47% (n = 39) of our patient series, moderate in 21.7% (n = 18), advanced in 3.6% (n = 3). 27% of them (n = 23) did not have aortic insufficiency.

Clinicians should carefully address LVOT. Anatomy, especially whether the subaortic membrane enters the aortic or mitral valve, the distance between the aortic valve and the subaortic membrane, and the mean LVOT gradient must be calculated, as these help predict the progression of LVOT obstruction. A subaortic membrane closer to the aortic valve is more likely to cause progression of stenosis in the valves, while a membrane farther from the aortic valve is more likely to progress to aortic and mitral

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Table 2. Comparison of measurements by cardioplegia

	N		*Ort±Ss*	*Min-Max (Median)*	*p value*
Age of operation (year)	Custadiol	12	8.17 ± 6.03	1–22 (7)	0.457
	Delnido	12	6.08 ± 5.87	1–22 (4.5)	
	Kan	59	7.56 ± 5.12	1–20 (6)	
Body mass index (m^2/cm^2)	Custadiol	12	17.41 ± 3.75	11.72–25.57 (16.74)	0.368
	Delnido	12	17 ± 5.51	12.62–32.08 (15.5)	
	Kan	59	17.85 ± 4.75	9.55–39.16 (16.67)	
Body surface area (m^2)	Custadiol	12	0.95 ± 0.42	0.33–1.52 (1.01)	0.198
	Delnido	12	0.74 ± 0.39	0.33–1.73 (0.61)	
	Kan	59	0.95 ± 0.42	0.2–1.75 (0.9)	
Trop-T (ng/mL)	Custadiol	12	4.95 ± 3.78	0.6–10 (3.5)	0.608
	Delnido	12	4.31 ± 3.76	0.4–10 (3.91)	
	Kan	59	3.54 ± 2.62	0.3–10 (2.7)	
Length of stay in hospital (day)	Custadiol	12	11.08 ± 6.17	3–27 (11)	0.035*
	Delnido	12	12.08 ± 8.94	5–37 (9)	
	Kan	59	21.83 ± 151.4	7–300 (7)	
Cross clamp time (minute)	Custadiol	12	141.92 ± 60.63	78–290 (122.5)	0.001**
	Delnido	12	87.08 ± 32.91	37–139 (82.5)	
	Kan	59	56.78 ± 36.06	14–180 (47)	
Cardiopulmoner bypass time (minute)	Custadiol	12	227.75 ± 113.03	125–526 (193.5)	0.001**
	Delnido	12	116.33 ± 51	55–240 (107.5)	
	Kan	59	84.51 ± 44.33	28–220 (70)	
Surgery duration (minute)	Custadiol	12	343.5 ± 109.57	235–585 (315)	0.001**
	Delnido	12	240.17 ± 88.22	132–360 (210)	
	Kan	59	206.53 ± 67.46	90–370 (195)	
	Kan	59	0.35 ± 0.69	0–3 (0)	
VIS score	Custadiol	12	17.67 ± 6.79	8–31 (17)	0.356
	Delnido	12	14.08 ± 4.89	7–21 (14.5)	
	Kan	59	13.2 ± 8.56	0–32 (15)	
Drainage (cc)	Custadiol	12	325 ± 179.62	100–650 (260)	0.001**
	Delnido	12	169.17 ± 103.52	30–400 (155)	
	Kan	59	142.54 ± 82.89	20–450 (140)	
Intubation time (day)	Custadiol	12	15.71 ± 18.88	2.5–70 (8.25)	0.056
	Delnido	12	10 ± 13.41	1.5–47 (5)	
	Kan	59	7.23 ± 7.12	0.5–23 (3.5)	
Ecmo duration (day)	Custadiol	12	2.5 ± 6.37	1–21 (9)	0.032*
	Delnido	12	0 ± 0	0–0 (0)	
	Kan	59	0.29 ± 2.21	2–17 (4)	
Intensive care hospitalisation (day)	Custadiol	12	57.83 ± 33.3	19–118 (53.5)	0.012*
	Delnido	12	55.54 ± 44.42	12–139 (45.5)	
	Kan	59	31.75 ± 23.74	4–116 (22)	

Kruskall–Wallis test *$p < 0.05$ **$p < 0.01$.

insufficiency.[11] In this patient group, the LV undergoes remodelling with the removal of the stenosis in front of the LV. In our series, LVesd (LV end systolic diameter) changed significantly in the postoperative period, even though there was no statistically significant change in LVsd (LV systolic diameter) and LVedd (LV end diastolic diameter).

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Table 3. Comparison of measurements by periods.

		1. Measurement (preoperative)	*2. Measurement (1st postoperative day)*	*3. Measurement (1st postoperative month control)*	p value
Ivedd (mm)	Ort ± Ss Min–Max (Median)	3.45 ± 0.87 1.02-4.96 (3.51)	3.61 ± 0.74 1.8-5.19 (3.64)	3.84 ± 0.68 2.48-5.8 (3.7)	0.089
Ivsd (mm)	Mean ± Sd Min–Max (Median)	1.17 ± 0.71 0.4-4.23 (0.99)	1.59 ± 2.94 0.58-18.6 (1.02)	1.07 ± 0.37 0.6-2 (1)	0.551
Ivesd (mm)	Mean ± Sd Min–Max (Median)	1.96 ± 1.17 0.75-6 (1.65)	1.83 ± 0.52 1.25-2.7 (1.75)	2.21 ± 0.62 1.1-3.6 (2.2)	0.023*

Friedmann Test **p < 0.01.

Table 4. Comparison of measurements by periods.

		1. Measurement (preoperative)	*2. Measurement (postoperative Day 1)*	p value
HTC	Mean±Sd Min–Max (Median)	35.39 ± 4.92 24-48 (35)	34.42 ± 33.31 21-330 (30.5)	0.001**
Mean gradient in the subaortic area	Mean±Sd Min–Max (Median)	71.47 ± 28.41 17-148 (70)	29.35 ± 22.94 0-90 (25)	0.001**

Wilcoxon Test **p < 0.01.

The risk of AV block is higher in patients undergoing aggressive resection and patients undergoing reoperation. It has been reported that 80% of patients (four out of five) who developed AV block developed AV block during reoperation due to aggressive surgical resection, but only 3.7% had AV block at the first surgery.[12] In our series, pacemaker implantation was required in five patients due to AV block, and the rate of AV block was calculated as 6%. There was no statistically significant difference between troponin-t values of patients with or without AV block. We think that this may be due to the limited number of patients.

Surgical resection of SAS usually yields good results with low mortality in experienced centres. An operative mortality rate close to 0% is reported in SAS.[13] However, Serraf et al. reported 3% operative mortality between 1980 and 1997 in their study on 160 patients.[14] Tunnel-type SAS, accompanying mitral stenosis, CoA, and hypoplastic aortic annulus were risk factors for overall mortality. In our series, 3.6% (n = 3) of the patients required ECMO. Mortality was calculated as 7.2% (n = 6).

Despite the absence of obvious echocardiographic findings in early childhood, a sharper ventriculoaortic angle has been described in patients with discrete subaortic stenosis. It causes increased wall stress on the ventricular surface. Increased wall stress triggers the pathological process and stimulates endothelial proliferation in the LVOT. Membrane resection and myectomy result in a decrease in the LVOT pressure gradient, and a change in the geometry of the ventricles occurs.[15] In our series, the gradient value in the subaortic area decreased from an average of 71.47 ± 28.41 mmHg to 29.35 ± 22.94 mmHg according to the first measurement, and this decrease was statistically significant (p = 0.001; p < 0.01).

Binsamalah et al. reported that septal myectomy in patients with isolated subaortic membranes was associated with a longer time required for reoperation. Septal myectomy relieves left ventricular stenosis and reduces turbulent flow in the left ventricle and restores the outflow tract and normal left ventricular outflow tract, but there is a risk of damage to the conduction bundle. Therefore, they suggested evaluating the operative findings and making an intraoperative decision for septal myectomy.[16] Based on our own experience, we believe that whether septal myectomy will be performed and how much myectomy will be done should be decided on a patient basis and the surgeon's own experience. In our series, troponin t values, which we think are correlated with myectomy, were found to be significantly higher in the Death group. Although wide resection is thought to reduce the risk of reoperation, it increases major complications and mortality.

The progression of discrete subaortic stenosis and the risk of reoperation are considered unpredictable. A large retrospective study reported slower progression in adults compared to children. In the same study, they said that care should be taken in terms of the risk of recurrence, since adults can be selected as having less severe disease.[17] In our patient group, the mean age was calculated as 7.43 ± 5.33 years.

Discrete subaortic stenosis is a complex disease that is largely unexplained. The unpredictability of recurrence after resection combined with the risks of surgery adversely affects the course of the disease. In clinical imaging techniques, computational and experimental fluid dynamics and mechanobiology offer new opportunities to elucidate the underlying mechanisms and pathogenesis of discrete subaortic stenosis. Clinicians and engineers must work together to implement this new strategy.[18,19] In our clinical series, the treatment is completely based on surgical experience. Although there is no definitive method and method to predict mortality and morbidity in this patient group, we think that early troponin-t values may be important in this regard.

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Table 5. Comparison of measurements by mortality status.

	N		*Mean±Sd*	*Min–Max (Median)*	*p value*
Age of operation (year)	Alive	77	7.82 ± 5.32	1–22 (7)	0.007**
	Death	6	2.5 ± 1.76	1–5 (2)	
Body mass index (m^2/cm^2)	Alive	77	17.95 ± 4.73	9.55–39.16 (16.89)	0.004**
	Death	6	14.04 ± 1.7	10.91–15.7 (14.59)	
Body surface area (m^2)	Alive	77	0.95 ± 0.41	0.27–1.75 (0.9)	0.01*
	Death	6	0.49 ± 0.23	0.2–0.76 (0.48)	
Trop-T (ng/mL)	Alive	77	3.47 ± 2.69	0.3–10 (2.7)	0.001**
	Death	6	8.79 ± 2.19	4.43–10 (9.8)	
Length of stay in hospital (day)	Alive	77	18.78 ± 132.37	−359–1103 (7)	0.032*
	Death	6	20 ± 13.65	3–42 (18.5)	
Cross clamp time (min)	Alive	77	69.97 ± 42.09	14–197 (62)	0.370
	Death	6	118.33 ± 104.9	26–290 (78)	
Cardiopulmoner bypass time (min)	Alive	77	99.79 ± 54.8	28–251 (84)	0.026*
	Death	6	238.5 ± 174.92	70–526 (209)	
Ultra filtration (cc)	Alive	77	314.94 ± 386.37	0–1150 (0)	0.105
	Death	6	1016.67 ± 1175.44	0–3100 (750)	
Balance (cc)	Alive	77	54.35 ± 461.46	−1070–2200 (−20)	0.225
	Death	6	−164.17 ± 323.58	−550–290 (−97.5)	
Surgery duration (min)	Alive	77	220.05 ± 74.54	90–382 (210)	0.009**
	Death	6	374.17 ± 151.9	195–585 (365)	
	Ex	6	0 ± 0	0–0 (0)	
VIS score	Alive	77	13.92 ± 8.24	0–32 (15)	0.846
	Death	6	14.67 ± 3.44	10–20 (15)	
Drainage (cc)	Alive	77	160.91 ± 103.52	20–600 (150)	0.047*
	Death	6	325 ± 218.7	110–650 (270)	
Intubation duration (day)	Alive	77	7.49 ± 8.22	0.5–47 (3.5)	0.002**
	Death	6	26.33 ± 22.2	9–70 (18.5)	
Ecmo duration (day)	Alive	77	0 ± 0	0–0 (0)	0.001**
	Death	6	7.83 ± 9.41	1–21 (4.5)	
Intensive care hospitalisation (day)	Alive	77	39.44 ± 31.36	4–139 (22)	0.771
	Death	6	32.83 ± 21.33	15–70 (24.5)	

Mann–Whitney U test *$p < 0.05$. **$p < 0.01$.

Conclusion

In the practice of congenital heart surgery, LVOTO is one of the pathologies that every surgeon will encounter frequently. It is necessary to have comprehensive knowledge about the pathophysiology of this stenosis, which is caused by various factors and pathologies. Septal myectomy remains the gold-standard treatment in Subaortic Membrane surgery, but the learning curve is challenging, and although many techniques are available, the precise surgical margins required to achieve a complete muscle resection are lacking. When the gradient is completely removed by myectomy, symptoms resolve, and late survival is equivalent to the healthy paediatric population of the same age. Although the risk of recurrence decreases as the degree of myectomy increases in these patients, the risk of complications such as complete atrioventricular block increases in the early period. Also, understanding the nuances involved in patient selection and operation, interpretation of imaging, and the tricks and pitfalls of surgical techniques are critical to success.

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