Incidence of, and Risk Factors for, Acute Modified Blalock-Taussig Shunt Occlusion in Neonates and Infants

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Objective: *This study aimed to explore the incidence of acute shunt occlusion in neonates and infants within 24 hours after undergoing MBTS surgery and to determine the potential predisposing factors of acute shunt occlusion.*
Material and Method: *The electronic database of patients was reviewed retrospectively. The occurrence of acute shunt occlusion and the potential risk factors in neonates and infants who had undergone MBTS surgery between January 2009 and December 2015 were collected.*
Results: *One hundred and two patients were enrolled. The incidence of acute shunt occlusion was 13.7% (14 patients). Using a univariate analysis, a 3-mm shunt size (OR 11.88, 95% CI 2.24 to 63.11, p<0.01), a pulmonary artery size less than 4 mm (OR 4.82, 95% CI 1.32 to 17.54, p = 0.02), no concomitant ligation of ductus arteriosus (OR 14.50, 95% CI 1.22 to 172.31, p = 0.03) and the surgeon with low case volume (<6 cases per year) (OR 5.96, 95% CI 1.71 to 10.74, p<0.01) were significantly associated with acute MBTS occlusion postoperatively. The multiple logistic regression analysis revealed the surgeon with low case volume (<6 cases per year) (OR 8.90, 95% CI 1.74 to 45.66, p<0.01) and non-ligated patent ductus arteriosus (PDA) (OR 36.58, 95% CI 2.02 to 661.26, p = 0.02) were the significant risk factors contributing to acute shunt blockage.*
Conclusion: *A small volume of MBTS cases operated per year and non-ligated PDA are significant risk factors for acute shunt occlusion within 24 hours postoperatively.*

Keywords: *Blalock-Taussig procedure, Incidence, Infant, Risk factors, Thrombosis*

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Cyanosis is commonly associated with certain congenital cardiac diseases, estimated incidence is approximately 1 in 1,000 live births[1]. The pathology mostly results from decreased pulmonary blood flow, with ensuing inadequate oxygenation and hypotension. The severity of cyanotic congenital heart disease may vary from mild cyanosis to a life-threatening condition, depending on the degree of right-sided cardiac obstruction.

A modified Blalock-Taussig shunt (MBTS) is a common palliative procedure. It connects the systemic to the pulmonary circulation via a synthetic shunt from a subclavian artery to a pulmonary artery[2]. Therefore, it is an effective mean of providing an additional pulmonary blood flow and alleviating deleterious outcomes from desaturation in vulnerable patients for whom definite corrective surgeries were not feasible. However, certain complications after MBTS placement do exist[3]. For example, shunt occlusion or overflow remains important complications after MBTS surgery. Postoperative shunt blockage may cause a sudden deterioration as a result of profound hypoxemia and potential cardiac arrest, especially in an unstable patient. Prevention of the modifiable risk factors and early detection would prompt an immediate reoperation or a medical management, leading to a better outcome. The reported incidence of shunt occlusion varies from 2.5 to 20%[4-9]. The proposed perioperative risk factors described in previous studies include low body weight[10-12], low activated partial thromboplastin time (aPTT)[6], small pulmonary artery size[4,13], and polycythemia[7]. However, the published results were not uniform, and none of the studies had been conducted on Asian populations. This study was

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designed to explore the incidence of acute shunt occlusion within 24 hours among neonates and infants undergoing MBTS surgery, and to analyze the potential perioperative factors of acute shunt occlusion.

Material and Method

This was a retrospective cohort study. The Institutional Review Board (Si 765/2014) approval was obtained with an exemption of consent. The medical records of 102 patients aged less than 12 months and having undergone MBTS surgery as their first operation between January 2009 and December 2015 were retrieved from the hospital electronic database.

The patients' characteristics and demographic data were recorded. Documentation was made of the perioperative variables related to the increasing risk of shunt occlusions, such as the size of the polytetra-fluoroethylene (PTFE) shunt, the systemic (subclavian or innominate) and pulmonary artery sizes, the preoperative and postoperative hematocrit levels, the perioperative heparin administration, and the aPTT. Additionally, the postoperative complications that occurred during the index hospitalization and the detailed characteristics of those patients who had developed shunt occlusion were also documented.

The diagnosis of acute shunt occlusion was based upon a combination of clinical signs and investigations, including the absence of an audible shunt murmur, a decrease in the partial pressure of arterial oxygen (PaO_2), or an oxygen saturation (SpO_2) of more than 20% from baseline. The incidence of acute shunt occlusion was recorded after the confirmative diagnosis was established from the echocardiographic evidence for poor shunt flow. All patients were treated with the re-operation according to the hospital protocol.

Statistical analysis

The primary objective of this study was to identify the incidence of acute shunt occlusion. The sample size was calculated on the assumption of a 15% incidence of acute shunt occlusion, based on estimated rates between 2.5 and 20% in previous publications[4-9]. A sample size of 100 patients was needed to achieve a 95% confidence interval (CI) with a 7% margin of error.

Demographic data was analyzed using descriptive statistics. The mean and standard deviations were shown for continuous variables with normal distribution; otherwise, the median with minimum and maximum was presented. For a univariate analysis, an independent sample t-test and a binary logistic regression were used for continuous and categorical variables, respectively. As for examining the risk factors of acute shunt occlusion, factors with the clinical feature of interest or factors with $p<0.15$ from the univariate analysis were entered into a multiple logistic regression model. The crude odds ratio (OR), the adjusted OR and the 95% CI were reported to determine the strength of the association between the possible factors and the occurrence of an acute shunt occlusion. A *p*-value of less than 0.05 was used as the cutoff value for statistical significance. The statistical analysis was performed using PASW Statistics for Windows, 18.0 Chicago: SPSS Inc.

Results

One hundred and two patients were enrolled in this study. The patient's median age was 1 month ranged from 1 day to 11 months, mean body weight was 4.3$\pm$1.8 kg. There were 19 patients (18.6%) had prior endotracheal intubation due to severe cyanosis. General anesthesia was conducted on all patients as a standard practice. No major intraoperative adverse events were noted. The blood pressure was maintained above the normal baseline value via fluid and inotrope(s) administration. Eighteen patients (17.6%) were extubated at the conclusion of surgery. Most patients underwent MBTS surgery via a thoracotomy incision, whereas one patient required a median sternotomy approach because of severe hemodynamic instability that necessitated the use of a cardio pulmonary bypass. PTFE grafts were used for the interposition tube graft in all patients. The shunt location was predominately the right MBTS. The other demographic data is shown in Table 1.

Fourteen patients (13.7%) had an acute shunt blockage within the first 24-hour period after the operations, with 11 patients (78.6%) having the occlusion within 6 hours. The univariate analysis of the possible risk factors related to acute shunt occlusion are demonstrated in Table 2. According to the univariate analysis, a 3-mm shunt size (OR 11.88, 95% CI 2.24 to 63.11, $p<0.01$), no concomitant ligation of ductus arteriosus (OR 14.50, 95% CI 1.22 to 172.31, $p = 0.03$), a small pulmonary artery size (OR 4.28, 95% CI 1.32 to 17.54, $p = 0.02$) and the surgeon with low case volume of less than 6 cases per year (OR 5.96, 95% CI 1.71 to 10.74, $p<0.01$) were associated with acute shunt blockage significantly. While using the multiple logistic regression analysis, the surgeon with low case volume (<6 cases per year) (OR 8.90, 95% CI 1.74 to 45.66, $p<0.01$) and non-ligated patent ductus arteriosus

Table 1. Demographic data

	Total (n = 102)
Gender: Male	57 (55.9)
GA (wk)	37.9±1.5
Birth weight (kg)	2.8±0.5
Age (d)	1 (1.0,330.0)
Body weight (kg)	4.28±1.80
Preexisting endotracheal tube	9 (18.6)
Diagnosis	
Pulmonary atresia with ventricular septal defect	25 (24.5)
Common atrium common ventricle with pulmonary stenosis or atresia	17 (16.7)
Univentricular heart with pulmonary atresia	18 (17.6)
Tricuspid atresia with pulmonary atresia	11 (10.8)
Tetralogy of Fallot	15 (14.7)
Others	16 (15.7)
Incision site	
Thoracotomy	101 (99.0)
Median sternotomy	1 (1.0)
Shunt location	
Right	88 (86.3)
Left	14 (13.7)
Shunt size (mm)	
3.0	9 (8.8)
3.5	51 (50.0)
≥4	42 (41.2)
Pulmonary artery size (mm)	5.7±2.3
Subclavian or innominate artery size (mm)	4.0±1.2
PDA ligation	3 (2.9)
Inotrope administration	
Preoperative	20 (19.6)
Postoperative	97 (95.1)
Number of postoperative inotrope administration	
0	5 (4.9)
1	80 (78.4)
≥2	16 (15.7)
Hematocrit (%)	
Preoperative	49.0±7.6
Postoperative	46.1±6.9
Activated partial thromboplastin time (sec)	
Preoperative	31.4±5.1
Postoperative	57.5±49.6
Oxygen saturation (%)	
Preoperative	81.7±8.5
Postoperative	91.8±6.5
Heparin administration	
Intraoperative	79 (77.5)
Postoperative	77 (75.7)
Time to extubation	
In operating room	18 (17.6)
<6 hour	16 (15.7)
6 to 24 hour	33 (32.4)
>24 hour	35 (34.3)

Data presented as mean ± SD, median (min, max) or n (%)

Table 1. Cont.

	Total (n = 102)
Surgeon (occlusion/total cases)	
1	4/66 (6.1)
2	4/8 (50.0)
3	2/13 (15.4)
4	4/15 (26.7)

Data presented as mean ± SD, median (min, max) or n (%)

(PDA) (OR 36.58, 95% CI 2.02 to 661.26, *p* = 0.02) appeared to be the significant risk factors, as presented in Table 3.

The postoperative complications are shown in Table 4. Three patients developed cardiac arrests and died within 24 hours, in which one of those 3 patients was included in the shunt occlusion group. The detailed characteristic of the patients with a shunt occlusion are shown in Table 5.

Discussion

A MBTS occlusion is a disastrous complication after shunt surgery. The incidence of shunt blockage has been recognized as ranging from 2.5% to 20%[4-9]. Prior publications predominantly included the shunt occlusion occurring at any time during the entire hospital stay or during the later follow-up period. The current study focused on the incidence of MBTS occlusion within the 24-hour period after surgery, which was observed in 13.7% of the patients. This result is comparable with a study by Gedicke et al, which demonstrated a rate for the first 24-hour shunt blockage of 11.8%[9]. Furthermore, this study found that approximately 80% of the patients with early shunt occlusion (within 24 hour) developed shunt occlusion within the first six hours. This implies that it is particularly important to closely monitor for, and to prevent, the development of a shunt blockage during the first six hours after surgery. All potential factors should be identified and eliminated in order to prevent an acute shunt occlusion from occurring.

Although several publications have investigated the risk factors for shunt occlusion, the results were incongruent. Theoretically, the causes of shunt blockage could be either a mechanical obstruction or a thrombosis. Mechanical obstructions might be related to patients' anatomical variations, the surgical techniques or the surgeons' experience. The shunt thrombosis component could plausibly be precipitated by a variety of factors. For instance, stasis of blood flow and hypercoagulability play a major role in contributing to thrombosis. A poor shunt flow could also occur as a result of a low cardiac output or a competitive flow from a PDA. In addition, a low flow state can be provoked by increased shunt resistance due to an increase in the length of the graft tube, hyperviscosity, a small shunt size, a small pulmonary artery size, or an increased pulmonary vascular resistance.

A small shunt size was generally presumed as a predisposing factor for acute shunt occlusion as it increases a greater resistance to blood flow and, consequently, a risk of thrombosis. However, this study was unable identify a statistical significance of this factor with a multiple logistic regression analysis. The finding is consistent with the other study[6]. Their incidence of the occluded shunts was also around 10% similar to the current study, which was probably too small to detect a significance of shunt size.

In this study, a small pulmonary artery size was another significant risk factor contributing to acute shunt occlusion according to the univariate analysis, but not the multiple logistic regression analysis. A small pulmonary artery can cause shunt thrombosis as a result of a limited blood flow to the pulmonary vascular bed. Guzetta et al. also described a significant association of shunt thrombosis with a smaller pulmonary artery size[4]. Their finding is consistent with the study by Al Jubair et al that shunt failure was more prevalent if the pulmonary artery diameter was less than 4 mm[13].

The steal phenomenon from a competitive flow through non-ligated PDA attenuates the shunt flow and increases the risk of thrombosis. The present study is able to identify a PDA preservation as a significant risk factor for acute shunt occlusion which is correlated with the study by Gedick et al[9]. The increased incident of shunt thrombosis was also

Table 2. Possible risk factors associated with acute shunt occlusion: univariate analysis

	Modified Blalock-Taussig shunt		Univariate analysis	
	Occlusion (n = 14)	Non-occlusion (n = 88)	Crude OR (95% CI)	*p*-value
Gestational age (wk)				
<37	1 (8.3)	11 (91.7)	0.52 (0.06, 4.36)	0.54
≥37	13 (14.9)	74 (85.1)	1	
Birth weight (kg)				
<2.5	3 (14.3)	18 (85.7)	0.93 (0.23, 3.68)	0.91
≥2.5	11 (15.3)	61 (84.7)	1	
Body weight (kg)				
<3	6 (20.0)	24 (80.0)	2.06 (0.53, 8.12)	0.30
3-4	4 (11.4)	31 (88.6)	1.07 (0.25, 4.63)	0.93
>4	4 (10.8)	33 (89.2)	1	-
Shunt size (mm)				
3.0	5 (55.6)	4 (44.4)	11.88 (2.24, 63.11)	<0.01
3.5	5 (9.8)	46 (90.2)	1.03 (0.26, 4.12)	0.96
≥4.0	4 (9.5)	38 (90.5)	1	-
Pulmonary artery size (mm)				
<4	5 (35.7)	9 (64.3)	4.82 (1.32, 17.54)	0.02
≥4	9 (10.3)	78 (89.7)	1	
Ligation of patent ductus arteriosus				
No	12 (12.1)	87 (87.9)	14.50 (1.22, 172.31)	0.03
Yes	2 (66.7)	1 (33.3)	1	
Perioperative heparin administration				
No	1 (7.1)	13 (92.9)	2.25 (0.28, 18.73)	0.45
Yes	13 (14.8)	75 (85.2)	1	
Polycythemia				
Preoperative				
No	12 (14.6)	70 (85.4)	1	0.59
Yes	2 (10.0)	18 (90.0)	0.65 (0.13, 3.16)	
Postoperative				
No	12 (13.6)	76 (86.4)	1	0.93
Yes	2 (14.3)	12 (85.7)	1.06 (0.21, 5.31)	
Postoperative inotrope administration				
No	0 (0)	5 (100.0)		1.00
Yes	14 (14.4)	83 (85.6)		
Postoperative aPTT (sec)				
≤35	2 (10.0)	18 (90.0)	0.51 (0.09, 2.95)	0.45
>35	5 (17.9)	23 (82.1)	1	
Surgeon annual MBTS case volume				
<6	10 (27.8)	26 (72.2)	5.96 (1.71, 10.74)	<0.01
≥6	4 (6.1)	62 (93.9)	1	

Data presented as n (%)
aPTT = activated partial thromboplastin time

demonstrated by Zahorec et al[14], even though the outcome did not yield a clinical significance. Currently, PDA closure is still controversial and performed according to the surgeon's preference[12]. PDA has a great benefit as it can be an immediate rescue in the presence of shunt blockage, allowing some degree of pulmonary blood flow. MBTS surgery is commonly performed via a right thoracotomy approach, as right MBTS is more prevalent (88% of patients in this series). In order to ligate a PDA, a left thoracotomy is required.

Table 3. Results of multiple logistic regression analysis of acute shunt occlusion

	Adjusted OR (95% CI)	*p*-value
Surgeon with low case volume (<6 cases/yr)	8.90 (1.74, 45.66)	<0.01
Shunt size <3 mm	4.50 (0.72, 28.02)	0.11
Pulmonary artery size <4 mm	2.22 (0.46, 11.83)	0.35
Non-ligated patent ductus arteriosus	36.58 (2.02, 661.26)	0.02
No perioperative heparin administration	1.24 (0.12, 12.56)	0.85

Table 4. Postoperative complications

	Total (n = 102)
No. of patients with shunt occlusion	
$\leq$24 hr	14 (13.7)
>24 hr	2 (2.0)
Shunt overflow	1 (1.0)
Reoperation	18 (17.6)
Shunt revision/replacement/thrombectomy	16 (88.8)
Shunt banding	1 (5.6)
Stop bleeding	1 (5.6)
Cardiac arrest	3 (2.9)
Death	3 (2.9)

Data presented as n (%)

This technical difficulty makes it less appeal for surgeons to ligate a PDA since it can also be spontaneously obliterated after prostraglandin E1 (PGE1) is discontinued. Nonetheless, as a result of a retrospective study, data regarding the existent of preoperative and postoperative PDA are limited. Even though non-ligated PDA is a statistically significant factor for early shunt occlusion, it is unable to completely assumed that there is a competitive flow through a PDA in every single patient.

This study did not find an association between preoperative or postoperative polycythemia and the incidence of acute shunt obstruction. Hyper-viscosity from polycythemia is a major factor for higher resistance to blood flow, leading to shunt thrombosis. A hemoglobin level over 18 g/dL was needed to show a significant relationship to shunt blockage in the previously published study[9].

Theoretically, heparin administration should be able to prevent the development of shunt thrombosis as it could oppose the effects of the hypercoagulable state that is well-known to aggravate the risk of thrombosis. When shunt occlusion was suspected, the heparin infusion was usually started while awaiting the echocardiographic confirmation. However, this study did not find any correlation between the perioperative heparin administration and the incidence of acute shunt occlusion. The antithrombotic therapy in neonates and children guidelines by Monagle et al suggested the routine use of intraoperative heparin following MBTS placement[15], but the use of post-operative heparin is still under debate[16]. The dosage and timing of the heparin administration varied widely among the surgeons; therefore, the result of the study could be confounded. One retrospective series reported no significant differences in early shunt failure between the heparin and the no-heparin groups[15].

MBTS surgery requires a skillful surgeon. There were technical difficulties regarding the complicated cardiac anomaly, limited surgical approach and small blood vessels in neonates and infants. In order to obtain a good result, experience from repetitive case exposure is necessary. Therefore, the authors selectively used an average number of MBTS cases operated per year as a surrogate for a surgical competency. To date, there is no recommendation on the number of cases per year to maintain a sufficient surgical skill. In this study, four surgeons, with the occlusion rate of over 15%, performed MBTS surgery 1 to 5 cases per year. Therefore, MBTS surgery of less than 6 cases per year was used as a cutoff value for a small case volume. Evidently, the surgeon's MBTS case volume of less than or equal to 6 cases per year has an approximately 9 times greater risk for early postoperative shunt occlusion. However, the result should be interpreted with caution as there were other confounding factors, such as the complexity of congenital heart disease, anatomical variants, a proficiency of teamwork, etc.

Acute shunt failure was observed more frequently in a conventional thoracotomy (20%) compared with the sternotomy approach (8%) in a study by Odim et al[17]. They recommended the sternotomy over the thoracotomy approach to MBTS for neonates and infants. In addition to a lesser rate of shunt occlusion, the sternotomy approach was found to

Table 5. Detailed patient characteristics in shunt occlusion group

No.	Gender	Age	Wt (kg)	Diagnosis	Location	Shunt size (mm)	PDA ligation	Hematocrit (%)		Oxygen saturation (%)		Peri-operative heparin	Time at occlusion (hr)	Remark
								Preop	Postop	Preop	Postop			
1	M	11d	2.8	Ebstein's anomaly, PA, IVS	Right	3.0	No	46.0	43.0	90	90	Yes	23	Thrombectomy, shorten mBTS
2	F	11d	2.8	CACV, PA	Right	3.5	Yes	51.1	44.7	80	40	Yes	During transfer	Shunt revision via sternotomy, graft compressed by ascending aorta
3	F	11d	2.2	TA, PA	Right	3.5	No	42.1	42.0	80	94	Yes	24	Shunt revision
4	F	13d	2.5	CACV, PA	Right	3.0	No	56.1	43.2	82	99	Yes	2	Shunt revision
5	M	26d	2.2	DORV, PA, VSD	Left	3.0	Yes	52.3	51.0	87	95	Yes	5	Replace shunt to 3.5
6	F	29d	3.1	Ebstein's anomaly, PA	Right	4.0	No	48.1	48.0	90	73	Yes	4	Shunt revision with atrial septectomy* Dead*
7	M	1mo	3.8	CACV, severe PS	Right	3.5	No	48.7	50.0	50	80	Yes	4	Replace shunt to 4
8	F	1mo	3.0	PA, VSD	Right	3.0	No	47.2	39.2	80	90	Yes	6	Replace shunt to 3.5
9	M	2mo	5.9	dTGA,VSD	Right	3.5	No	49.7	47.1	80	90	Yes	3	Replace shunt to 4
10	F	2mo	3.5	TOF	Right	4.0	No	43.6	55.8	95	95	Yes	6	Shunt revision
11	M	3mo	5.0	dTGA	Right	3.5	No	49.0	35.5	78	90	Yes	3	Replace shunt to 4 via sternotomy
12	M	4mo	5.1	CACV, PA, TA, TAPVR	Right	4.0	No	46.8	39.0	83	70	Yes	11	Replace shunt to 5, right to left
13	F	4mo	2.4	TOF	Right	3.0	No	47.0	45.8	74	90	Yes	2	Shunt revision
14	F	8mo	7.4	TOF	Right	4.0	No	57.8	58.0	85	90	Yes	3	Replace shunt to 5

M = male; F = female; CACV = common atrium common ventricle; DORV = double-outlet right ventricle; IVS = intact ventricular septum; PA = pulmonary atresia; PDA = patent ductus arteriosus; PS = pulmonic stenosis; TA = tricuspid atresia; TAPVR = total anomalous pulmonary venous return; dTGA = dextro-transposition of great arteries; TOF = tetralogy of Fallot; VSD = ventricular septum defect be superior in terms of reduced morbidity and mortality as it provided more hemodynamic stability, was technically easier to perform, avoided lung compression, including the possibility to ligate a PDA simultaneously. Because of the limited number of sternotomy incisions in this report, the surgical approach was not included for statistical analysis.

Currently, there is insufficient evidence to recommend a specific therapy for acute shunt thrombosis[15]. The management is at the surgeon's discretion and varies greatly. The treatment options for a shunt occlusion include reoperation with a shunt revision, a shunt take down and replacement, a catheter-based intervention, or thrombolysis. At the author's hospital, the protocol is to re-operate on all patients. All patients who were diagnosed with an acute shunt occlusion in this study had re-operations to do a shunt revision of the same size; to upsize to a larger shunt; or to perform a thrombectomy.

The advancements in early diagnosis, surgical techniques, neonatal cardiac anesthesia and intensive care have led to a decline in postoperative MBTS morbidity and mortality. The overall mortality rate after MBTS was reported as 2 to 18.2%[5,6,11]. According to a study by Kucuk et al., 2% of mortality was associated with thrombotic occlusion, and the patient died during a shunt revision[6]. Others expired due to cardiac decompensation in the 48-hour postoperative period, sepsis and pneumonia. The overall mortality is this series was 2.9%, but the mortality rate in the acute-shunt failure group was as high as 7.1% suggested the significance of the prevention of acute shunt occlusion.

The retrospective design had become a major limitation. The available database was not able to provide adequate information. Since the primary research question was to determine the incidence of acute shunt occlusion, the statistical power of the sample size might be insufficient to identify the risk factors. A future, prospective study or a multi-center study should be carried out to provide more accurate data and improve the quality of the evidence.

In conclusion, the incidence of acute MBTS shunt occlusion within 24-hour postoperatively was comparable to previous literature. A surgeon with low case volume and non-ligated PDA are the significant predisposing factors associated with acute MBTS occlusion. The currently available data are still inhomogeneous; thus, there is no single best-predictor for acute shunt occlusion in neonates and infants undergoing MBTS surgery. Vigilant postoperative monitoring and early recognition are essential for immediate treatment to correct the devastating condition and improve patient's outcome.

What is already known on this topic?

Acute modified Blalock-Taussig shunt (MBTS) occlusion is catastrophic. The risk factors for shunt obstruction vary in published literature.

What this study adds?

The incidence of acute shunt occlusion is not uncommon. A surgeon with low case volume (<6 cases per year) and non-ligated PDA are significant risk factors for acute shunt occlusion within 24 hours postoperatively.

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Potential conflicts of interest

None.

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อุบัติการณ์และปัจจัยเสี่ยงของการอุดตันแบบเฉียบพลันในผู้ป่วยทารกแรกเกิดและผู้ป่วยเด็กอายุน้อยกว่า 1 ปี ที่มารับการผ่าตัด Modified Blalock-Taussig Shunt

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วัตถุประสงค์: *เพื่อศึกษาอุบัติการณ์และปัจจัยเสี่ยงของการอุดตันแบบเฉียบพลัน (ภายใน 24 ชั่วโมง) ในผู้ป่วยทารก แรกเกิดและผู้ป่วยเด็กอายุน้อยกว่า 1 ปี ที่มารับการผ่าตัด Modified Blalock-Taussig Shunt (MBTS)*

วัสดุและวิธีการ: *เป็นการศึกษาย้อนหลังจากเวชระเบียนอิเล็กทรอนิกส์ที่อยู่ในระบบโดยเก็บข้อมูลการเกิดการอุดตันของ MBTS แบบเฉียบพลัน และปัจจัยเสี่ยงที่น่าจะมีผลต่อการเกิดการอุดตันของ MBTS ในผู้ป่วยทารกแรกเกิด และผู้ป่วยเด็กอายุน้อยกว่า 1 ปี ที่มารับการผ่าตัด MBTS ตั้งแต่เดือนมกราคม พ.ศ. 2552 ถึงเดือนธันวาคม พ.ศ. 2558*

ผลการศึกษา: *จากประชากรศึกษาจำนวน 102 คน พบอุบัติการณ์ของการเกิดการอุดตันของ MBTS ร้อยละ 13.7 (14 คน) การวิเคราะห์ผลโดยใช้วิธี univariate พบว่า shunt ขนาด 3 มม. (OR 11.88, 95%CI 2.25-63.11, p<0.01) ขนาดหลอดเลือดแดงพัลโมนารีเล็กกว่า 4 มม. (OR 4.82, 95%CI 1.32-17.54, p = 0.02) การไม่ได้ผูก ductus arteriosus ร่วมด้วย (OR 14.50, 95%CI 1.22-172.3, p = 0.03) และจำนวนผู้ป่วยที่ศัลยแพทย์ผ่าตัด MBTS น้อยกว่า 6 รายต่อปี (OR 5.96, 95%CI 1.71-10.74, p<0.01) เป็นปัจจัยเสี่ยงที่มีผลต่อการเกิด MBTS อุดตันภายใน 24 ชั่วโมงหลังผ่าตัด ขณะที่วิเคราะห์ผลด้วยวิธี multiple logistic regression พบว่าจำนวนผู้ป่วยที่ศัลยแพทย์ผ่าตัด MBTS น้อยกว่า 6 รายต่อปี (OR 8.90, 95%CI 1.74-45.66, p<0.01) และการไม่ได้ผูก ductus arteriosus ร่วมด้วย (OR 36.58, 95%CI 2.02-661.26, p = 0.02) เป็นปัจจัยเสี่ยงที่ทำให้เกิด MBTS อุดตัน*

สรุป: *ศัลยแพทย์ที่ผ่าตัด MBTS ต่อปีน้อย และการไม่ได้ผูก ductus arteriosus เป็นปัจจัยเสี่ยงที่ทำให้เกิดการอุดตัน แบบเฉียบพลัน (ภายใน 24 ชั่วโมง) ของ MBTS หลังผ่าตัด*