Title:

When to Intervene the Pulmonary Artery: Importance of Anatomical Assessment in the Diagnosis of Pulmonary Artery Coarctation

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Glossary of Abbreviations:

CECT = contrast enhanced-computed tomography

MBTS = modified Blalock-Taussing shunt

PA = pulmonary artery

PACoA = pulmonary artery coarctation

PDA = patent ductus arteriosus

Central Picture Legend: (87 / 90 characters allowed)

Uneven pulmonary arteries branching increases the risk of pulmonary artery coarctation.

Central Message: (115/ 200 characters allowed)

Morphologic assessment by preoperative computed tomography should be considered in patients with pulmonary atresia.

Perspective Statement: (318/405 character allowed)

Pulmonary artery coarctation (PACoA) is a major problem that leads to repeated interventions. However, there is little evidence regarding the prediction of PACoA development. This study assessed the correlation between anatomical features and

PACoA development using preoperative contrast-enhanced computed tomography.

Structured Abstract: (216/250 words allowed)

Objective: Pulmonary artery coarctation (PACoA) is a major problem that increases the frequency of intervention. However, there is little evidence regarding the prediction of PACoA development.

Methods: A retrospective chart review was performed on 42 patients who underwent modified Blalock–Taussig shunt and preoperative contrast-enhanced computed tomography (CECT). An uneven PA branching was defined as an abnormal ductus arteriosus connection to the left PA distal to the PA branching on CECT.

Results: Nineteen (45.2%) of 42 patients were diagnosed with PACoA. The median diameters of the ductus on the aorta and PA sides were 4.1 mm and 3.6 mm in the PACoA group and 3.6 mm and 2.9 mm in the non-PACoA group, respectively (P=0.18, 0.51). Tortuous ductus was recognized in 7 (36.8%) patients in the PACoA group and 14 (60.8%) patients in the non-PACoA group [P=0.12]. PACoA was associated with pulmonary atresia (16 patients [84.2%] in the PACoA group and 12 patients [52.1%] in the non-PACoA group) [P=0.02]. All 19 (100%) patients had uneven PA branching in the PACoA group, whereas 5 of 23 (21.7%) patients had uneven PA branching in the non-PACoA group [P<0.001].

Conclusions: Uneven PA branching rather than the ductus arteriosus size was strongly

associated with PACoA development; therefore, morphologic assessment by CECT should be considered in patients with pulmonary atresia.

Keywords:

Pulmonary artery coarctation, congenital heart disease, neonate, pulmonary artery stenosis, anatomy, CT

INTRODUCTION

Pulmonary artery coarctation (PACoA) was first described by Mangers in 1802. This lesion was well recognized in the 1960s.[1,2] It is also known as "juxtraductal" pulmonary coarctation/obstruction based on the histological findings observed in patients with a discrete narrowing at the origin of the left pulmonary artery (PA), in which the ductal tissue was present in the stenotic segment.[3,4] The occluded PA after PACoA may lead to disproportionate PA growth and impaired development of the pulmonary vasculature and lungs.[5,6] To achieve definitive repair, such as the Fontan operation and biventricular repair, multiple interventions are required.[7]

Many factors including the right aortic arch, pulmonary atresia, muscular atresia, large, tortuous duct, and abnormal origin of the duct are considered associated with PACoA development.[7–13] Because the presence of a ductus arteriosus implanting away from the bifurcation on either the left or right pulmonary artery seems to be one of important anatomic features based on previous studies[9,14], we defined "uneven PA branching" as an abnormal ductus arteriosus connection to the left PA distal to the main PA's branching instead of being in direct continuity with the pulmonary trunk (Figure 1). However, there is little evidence regarding the correlation between these factors and PACoA. We retrospectively reviewed preoperative contrast-enhanced computed tomography (CECT)

scans to assess the anatomical features of patients who developed PACoA.

PATIENTS AND METHODS

This retrospective, nonrandomized, single-institution study recruited 72 patients who underwent modified Blalock–Taussig shunt (MBTS) for staged reconstruction of congenital heart diseases at Okayama University Hospital between 2014 and 2018. Forty-two consecutive patients with patent ductus arteriosus (PDA) who underwent preoperative CECT were included in this study (Figure 2). PACoA was diagnosed when patients had discrete stenosis of the PA with a diameter of <3 mm where the ductus arteriosus was connected or required surgical intervention.[7]

The institutional review board of our hospital approved this study and waived the requirement for written informed consent because of its observational nature (Institutional Review Board 2009-023, approved on October 23, 2020). Preoperative, intraoperative, postoperative, and follow-up data were collected from the clinical reports.

Our institutional approach for constructing MBTS involved 1) the size of shunt was decided based on the body weight (<2.5kg: 3.0mm, 2.5 – 3.0 kg: 3.5mm, >3.0kg: 4.0mm polytetrafluoroethylene (PTFE) graft), 2) the laterality of shunt was chosen

based on the side of aortic arch, i.e., right-sided for the left aortic arch, 3) cardiopulmonary bypass was used when systemic oxygenation was not maintained above 80% of SaO2 with the clamping of the PA or the need for PA plasty. In case bilateral PAs were hypoplastic, the shunt was anastomosed to main PA. The ductus arteriosus was routinely ligated or divided after the construction of MBTS unless MBTS was performed via right thoracotomy. Concomitant PA plasty was performed with MBTS in patients having a discrete narrowing of the PA where the ductus arteriosus drained into, with a diameter of less than 2.0mm. Whether or not resect the ductal tissue was decided by intraoperative inspection of the inside of the juxtaductal PA. Autologous fresh pericardium or 0.1mm ePTFE was used to augment the stenotic site whenever necessary and this was decided by intraoperative findings by each surgeon.[7]

CECT

CECT was performed if echocardiogram showed suspicious of PACoA or uncertainty of the anatomy. All CECT scans were evaluated by two experienced pediatric cardiac surgeons blindly and independently using multiplanar reconstruction. They determined the final anatomical characteristics after consultation. Uneven PA branching was defined as an abnormal ductus arteriosus connection to the left PA distal to the main PA's branching instead of being in direct continuity with the pulmonary trunk (Figure 1). In

patients with a right ductus arteriosus, uneven PA branching was defined as an abnormal ductus arteriosus connection to the right PA. A tortuous duct was defined as the ductus arteriosus that was bent at least twice in the course. The right and left PA were measured immediately proximal to the origin of the first branches. The PDA-to-PA size ratio was calculated by dividing the diameter of PDA by the diameter of PA to which the PDA was connected to.

Statistical Analysis

Continuous variables are reported as medians (interquartile ranges). Categorical variables are reported as absolute frequencies (percentages). Continuous variables were compared using the Mann–Whitney U test between patients with and without PACoA. Categorical variables were compared using Fisher's exact test and Pearson's chi-squared test. All statistical analyses were performed using Stata/SE version 17 (StataCorp, Texas, USA).

RESULTS

Demographics

Patient characteristics are shown in Table 1. The median age and weight of the patients at the time of the MBTS procedure were 32 (interquartile range, 24–61) days and 2.9

(interquartile range, 2.7–3.3) kg, respectively. Moreover, 19 (45.2%) of the 42 patients were diagnosed with PACoA.

Clinical Outcomes

The median follow-up period was 4.6 (3–6.4) years. One hospital death and six late deaths occurred. The lone operative death was caused by sepsis. The cause of death in six late mortalities were sepsis in one patient, acute encephalopathy in one patients and unknown cause in four patients. The mortality rate was higher in single ventricular patients than in biventricular patients (5 [31.2%] vs. 2 [7.6%], P=0.04). Four patients (21%) with PACoA and three patients (13%) without PACoA died (P=0.48). All nine patients who were diagnosed preoperatively (N=7) or intraoperatively (N=2) had concomitant pulmonary arterioplasty at the time of MBTS. The ductal tissue was completely resected, and end-to-end anastomosis was performed in two patients. Patch augmentation was performed with a pericardium or 0.1-mm PTFE sheet in seven patients. By contrast, 10 patients developed PACoA after MBTS, and the intervention was performed surgically (N=6) using a catheter (N=7). Four-teen patients (14/19 (73.6%) required an intervention after the initial operation. Of these 12/19 had a surgical intervention and 12/19 had a catheter intervention. Nine patients had both

catheter and surgical interventions (table 1). Two patients required surgical intervention mainly due to PACoA. Surgical interventions were performed at the time of elective surgeries with other surgical indications in 10 patients.

Anatomical assessment by CECT

The anatomical features in CECT images are shown in Table 2. Pulmonary atresia was more common in patients with PACoA than in those without PACoA (16 [84.2%] vs. 12 [52.1%], P=0.02). Seven patients with PACoA were diagnosed with muscular atresia (36.8%), whereas three patients (29.1%) without PACoA were diagnosed with muscular atresia (P=0.07). CECT was conducted at an earlier age in patients with PACoA than in those without PACoA (6 days [1–8] vs 26 days [5–60], P=0.002). All 19 (100%) patients in the PACoA group had uneven PA branching, whereas 5 of 23 (21.7%) patients in the non-PACoA group had uneven PA branching [P<0.001]. The median diameters of the ductus on the aorta and PA sides were 4.1 mm and 3.6 mm in the PACoA group, respectively, and 3.6 mm and 2.9 mm in the non-PACoA group, respectively. Tortuous ductus was recognized in 7 (36.8%) patients in the PACoA group and 14 (60.8%) patients in the non-PACoA group [P=0.12]. Table 3 shows the anatomical features in CECT of patients with uneven PA branching.

DISCUSSION

The adverse effects of PACoA include disproportionate PA growth and increased frequency of interventions. Previous studies have implied a correlation between anatomical features and PACoA development.[7–13] This study demonstrated that uneven PA branching on preoperative CECT was strongly associated with PACoA. Among patients with normal PA branching, none developed PACoA during our observation period. This study also indicated that patients with PACoA are likely to have pulmonary atresia, which is consistent with the findings of previous studies. Many other factors are believed to be associated with PACoA development including right aortic arch, large tortuous duct, and muscular atresia; however, compared with uneven PA branching and pulmonary atresia, these factors do not seem to be strong indicators of PACoA development.

Another key finding is that only one-third of the 19 patients with PACoA were diagnosed preoperatively, and more than half of the patients developed PACoA after MBTS. This is important in clinical practice when deciding for surgical pulmonary arterioplasty and post-MBTS follow-up.

Anatomical characteristics in PACoA development

From a histological point of view, PACoA is believed to be caused by a ductal tissue that migrates into the PA wall, which is often associated with the displacement of the entry site of the ductus into the pulmonary trunk towards one of the PA.[8] A recent study showed that the primary problem is stunted development of the pulmonary push extension leading to pulmonary stenosis or atresia and subsequent lack of the proper incorporation of the ventral segment into the aortic sac, which causes an abnormal connection between the left PA and pulmonary arch artery (future ductus arteriosus).[14] From this point of view, it may be more accurate to describe this anatomy as an abnormal left PA origin from the ductus rather than an abnormal ductus connection to the left PA (Figure 3). Therefore, we defined this anatomy as uneven PA branching that was strongly associated with PACoA development.

Although the majority of patients with uneven PA branching developed PACoA, 20.8% of the patients with uneven PA branching did not develop PACoA during follow-up. We analyzed the anatomical features of these patients and found a relatively large right PA in patients who did not develop PACoA (3.8 mm vs. 5.5 mm, P=0.02). This may be caused by the limited blood flow from the PDA to the right PA due to PACoA, which restricts right PA growth. However, in this theory, it is strange that it becomes obvious

only when we compared patients with uneven PA branching. This may be due to multiple comparisons.

When to intervene

The indications and optimal timing of PACoA surgery are still debated. Various strategies and procedures for PACoA are available. Brink et al. performed excision of the localized ridge in the PA and primary end-to-end anastomosis of the posterior wall with anterior patch reconstruction. Their indication for the surgery is based on the presence of a large ductus arteriosus implanting away from the bifurcation on either the left or right PA.[9] Kim et al. demonstrated low reintervention rates using the main flap technique.[15] Sakamoto et al. performed PA plasty with excision of all ductal tissues and end-to-end anastomosis without patch augmentation for all monoventricular patients who have ductal-dependent pulmonary blood flow.[10] On the contrary, Choi et al. implied the importance of the "shunt-only" strategy to avoid PA plasty at the time of MBTS.[16]

In this study, only nine patients were diagnosed with PACoA preoperatively or intraoperatively, whereas 10 patients developed PACoA postoperatively, and more than half of the patients with PACoA were diagnosed after MBTS. All patients had uneven

PACoA with an underlying diagnosis of pulmonary atresia. Based on this finding, close follow-up in outpatient clinics is recommended for patients with pulmonary atresia with uneven PA branching. Concomitant PA plasty with MBTS can be an option for such patients if the surgical procedure is feasible. However, because PA plasty in small neonates is technically challenging and may require repeated reinterventions during staged repair, it may be reasonable to not perform PA plasty at the time of MBTS in small neonates based on surgeon's experience. We believe that the ultimate goal for this patient group is to achieve definitive repair with good PA conditions. For future study, we also need to address the effect of PA plasty at the time of MBTS in patients with uneven PA branching comparing patients who did not underwent PA plasty.

LIMITATION

This study assumes the typical limitations of any retrospective study: selection bias and lack of randomization. As we only analyzed patients who underwent CECT, this population was also highly specialized. Additionally, the timing of CECT differed between patients who developed PACoA and those who did not.

CONCLUSIONS

Uneven PA branching rather than the size of the ductus arteriosus was strongly associated with PACoA development; therefore, morphological assessment by CECT should be considered in patients with pulmonary atresia. Surgical intervention should be considered when CECT shows an uneven PA branching.

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Figure 1. “Uneven pulmonary arteries branching” was defined as an abnormal ductus arteriosus connection to the left PA distal to the main PA’s branching instead of being in direct continuity with the pulmonary trunk.

Figure 2. A retrospective chart review was performed on 72 patients who had a modified Blalock-Taussig shunt as a first palliation between 2014 and 2018. 42 consecutive patients who underwent CECT preoperatively and had patent ductus arteriosus were included in this study. All 19 (100%) patients had uneven PA branching in the PACoA group, whereas 5 of 23 (21.7%) patients had uneven PA branching in then non-PACoA group [P<0.001]. CECT, contract enhanced-computed tomography; PA, pulmonary artery; PACoA; pulmonary artery coarctation.

Figure 3. a. b. PACoA is believed to be caused by a ductal tissue that migrates into the PA wall, which is associated with the displacement of the entry site of the ductus into the pulmonary trunk towards one of the PA. In patients with uneven branching pulmonary arteries, left PA arises from the ductus arteriosus. c. d. In patients with a right ductus arteriosus, uneven PA branching was defined as an abnormal ductus arteriosus connection to the right PA. DA, ductus arteriosus.

336 Table 1. Patients' characteristics and clinical course

	PACoA (+) N=19	PACoA (-) N=23	P
Diagnosis			
Functional single ventricle (%)	7 (36.8)	9 (39.1)	0.87
Biventricle (%)	12 (63.1)	14 (60.8)	0.87
Body weight, kg (IQR)	3.2 (2.8-3.3)	2.9 (2.6-3.2)	0.14
Shunt laterality			
Right (%)	10 (52.6)	17 (73.9)	0.15
Left (%)	7 (36.8)	5 (21.7)	0.28
Main Pulmonary artery (n)	2 (10.5)	1 (4.3)	0.43
Timing of PACoA diagnosis			
preoperative	7 (36.8)	-	
intraoperative	2 (10.5)	-	
postoperative surgical catheter	10 (52.6) 6 7	-	
Concomitant PA plasty	9 (47.3)	0 (0)	
End-to-end anastomosis	2 (10.5)	-	
Patch augmentation	7 (36.8)	-	
Post-MBTS PACoA intervention	14 (73.6)		
Surgical	12 (63.1) Pre or intraoperatively diagnosed PACoA N=9 6 (66.7)	Postoperatively diagnosed PACoA N=10 6 (60)	
Catheter	12 (63.1)		
	Pre or intraoperatively diagnosed PACoA N=9 5 (77.8)	Postoperatively diagnosed PACoA N=10 7 (70)	
Death	4 (21)	3 (13)	0.48
Achievement of definitive repair	16 (84.2)	18 (78.2)	0.36

Functional single ventricle (N=16)	5 (71.4)	5 (55.5)	0.63
Biventricle (N=26)	11 (91.6)	13 (92.8)	0.9

337 MBTS, modified Blalock–Taussig shunt; PA, pulmonary artery; PACoA; pulmonary
338 artery coarctation
339
340 Table 2. Anatomic features

	PACoA (+) N=19	PACoA (-) N=23	P
CECT age, days (IQR)	6 (1-8)	26 (5-60)	0.002
Diagnosis			
Pulmonary atresia	16 (84.2)	12 (52.1)	0.02
Muscular atresia	7 (36.8)	3 (13)	0.07
Uneven PA branching	19 (100)	5 (21.7)	<0.001
Diameter of the duct			
PA side, mm (IQR)	3.6 (2.8-4.3)	2.9 (2-3.9)	0.28
Aorta side, mm (IQR)	4.1 (3.7-4.5)	3.6 (2.4-4.6)	0.07
PDA-to-PA size ratio			
PA side (IQR)	0.93 (0.66-1.08)	0.78 (0.58-1.12	0.51
Aorta side (IQR)	1.04 (0.91-1.32)	0.97 (0.59-1.22)	0.18
Tortuous duct	7 (36.8)	14 (60.8)	0.12
Right duct	4 (21)	2 (8.6)	0.19
Right aortic arch	6 (31.5)	4 (17.3)	0.28
Diameter of the right PA, mm (IQR)	3.8 (3.2-4.7)	4.2 (3-5.2)	0.47
Z score (IQR)	-1.3 (-2.17- -0.71)	-0.97 (-2.2-0)	0.45
Diameter of the left PA, mm (IQR)	3.9 (3-4.3)	3.9 (3.1-4.6)	0.69
Z score (IQR)	-1.32 (-2.13- -0.36)	-1 (-2- -0.37)	0.42

341 CECT, contract enhanced computed tomography; PA, pulmonary artery; PACoA;
342 pulmonary artery coarctation; PDA, patent ductus arteriosus
343
344 Table 3. Patients with uneven PA branching

	PACoA (+) N=19	PACoA (-) N=5	P
Diagnosis			
Pulmonary atresia	16 (84.2)	2 (40)	0.04
Muscular atresia	7 (36.8)	1 (20)	0.04
Functional single ventricle	7 (36.8)	2 (40)	0.89
CECT age, days (IQR)	6 (1-8)	12 (5-24)	0.29
Diameter of the duct			
PA side, mm (IQR)	3.6 (2.8-4.3)	3 (2.9-3.5)	0.43
Aorta side, mm (IQR)	4.1 (3.7-4.5)	3.6 (3.6-4.9)	0.54
PDA-to-PA size ration			
PA side (IQR)	0.93 (0.66-1.08)	0.81 (0.69-0.88)	0.39
Aorta side (IQR)	1.04 (0.91-1.32)	1.04 (0.79-1.19)	0.46
Tortuous duct	7 (36.8)	3 (60)	0.35
Right duct	4 (21)	0 (0)	0.23
Right aortic arch	6 (31.5)	1 (20)	0.61
Diameter of the right PA, mm (IQR)	3.8 (3.2-4.7)	5.5 (4.75-5.9)	0.02
Z score (IQR)	-1.3 (-2.17- -0.71)	0.23 (-0.43-0.52)	0.02
Diameter of the left PA, mm (IQR)	3.9 (3-4.3)	4.1 (3.95-4.4)	0.31
Z score (IQR)	-1.32 (-2.13- -0.36)	-1 (-1.3- -0.69)	0.35

345 CECT, contract enhanced computed tomography; PA, pulmonary artery; PACoA;
346 pulmonary artery coarctation; PDA, patent ductus arteriosus
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