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Original Article

The effect of cyanotic and acyanotic congenital heart disease on children's growth velocity

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Abstract

Background Congenital heart disease (CHD) can lead to failure to thrive. Decreased energy intake, malabsorption, increased energy requirements, and decreased growth factors (growth hormone/insulin-like growth factor 1 axis) are related to malnutrition and growth retardation in children with CHD.

Objective To compare the impact of cyanotic and acyanotic CHD on children's growth velocity (using the 2009 WHO growth velocity chart).

Methods This study was conducted in patients less than 24 months of age with CHD in the Pediatric Cardiology Specialist Unit Dr. Moewardi Hospital, Surakarta, Central Java, from December 2016 to February 2017. Subjects' weights were evaluated at the beginning of the study and two months later. Data were compared to the WHO Growth Velocity chart and analyzed by Chi-square test.

Results Of 46 patients with CHD (23 cyanotic, 23 acyanotic), 10 patients (21.7%) were identified with failure to thrive, i.e., $< 5^{th}$ percentile. Significantly more children with acyanotic CHD were in the >5th percentile for growth velocity than were children with cyanotic CHD (OR 5.600; 95%CI 1.038 to 30.204; P=0.032). Acute upper respiratory tract infection was not significantly associated with growth velocity (OR 2.273; 95%CI 0.545 to 9.479; P=0.253).

Conclusion Children with cyanotic CHD have 5.6 times higher risk of failure to thrive than children with acyanotic CHD. **[Paediatr Indones. 2017;57:159-62 doi: http://dx.doi.org/10.14238/pi57.3.2017.159-62]**.

Keywords: *congenital heart disease; growth velocity; failure to thrive*

Congenital heart disease (CHD) is the most prevalent structural malformation, constituting 25% of all congenital anomalies, and is on the forefront of global medical issues. CHD occurs in 0.5-0.8% of all births.[1-3] Children with CHD often face impaired growth and development. Chen *et al.* reported delays in growth and development among children suffering from CHD compared to their normal counterparts.[4] The so-called failure to thrive (FTT) is not a disease unto itself, but rather a symptom of a general pathway caused by one or more medical, psychosocial, or environmental issues leading to stunted growth in a child. Evaluation of children with slowed or stunted growth is a challenge to pediatricians.[5] The etiology of failure to thrive among CHD patients is still obscure. Many factors may contribute to such a condition, including caloric intake, malabsorption, increased use of energy, relative hypoxia, and endocrine adaptation.[5,6] We aimed to assess for a possible correlation between growth velocity and cyanotic and acyanotic CHD in children, using the 2009 WHO Growth Velocity Chart.

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Methods

This prospective cohort study was done to compare the impact of cyanotic and acyanotic CHD on children's growth velocity. This study was carried out in the Pediatric Cardiology Specialist Unit at Dr. Moewardi Hospital, Surakarta, Central Java, from December 2016 to February 2017. The target population was cyanotic and acyanotic CHD patients <24 months of age. Parents or guardians of patients who fulfilled the research criteria provided informed consent and participated by filling in the research forms. Patients with Down syndrome, immune deficiency, dysmorphia, severe sepsis, thyroid anomalies, and gastrointestinal congenital malformation, were excluded. All patients aged <24 months with cyanotic or acyanotic CHD who fulfilled the inclusion criteria were included consecutively. Subjects' weights were evaluated at the beginning of the study and taken prospectively two months later. The data were recorded in the 2009 WHO Growth Velocity Chart and failure to thrive (FTT) was defined to be < 5th percentile.

Data were processed and analyzed using *SPSS 20.0* software. Basic characteristics of the subjects (age, sex, type of congenital heart disease, and weight) were presented in numbers and percentages. Correlations between the independent and dependent variables were analyzed by Chi-square test. Confounding variables were analyzed by logistical regression multivariate statistical analysis.

Results

This study was performed on 46 patients diagnosed with cyanotic or acyanotic congenital heart disease. Of 56 initial patients, 10 were excluded due to Down syndrome and/or congenital hypothyroidism. **Table 1** shows that 58.7% of subjects were female and 41.3% were male. Most patients (65.2%) had no accompanying diseases (no acute upper respiratory tract infection, URI), whereas the remaining 34.8% had accompanying disease (acute URI). Most diagnoses of cyanotic CHD were tetralogy of Fallot (TOF) (30.4%), whereas most acyanotic CHD cases were diagnosed as atrial septal defect (ASD) (17.4%). In addition, 78.3% of patients did not have FTT, whereas the remaining 21.7% experienced FTT.

Table 1. Basic characteristics of subjects

Characteristics	N=46
Gender, n(%)	
Female	27 (58/7)
Male	19 (41.3)
CHD, n(%)	
Acyanotic	23 (50)
Cyanotic	23 (50)
Growth velocity, n(%)	
≤ 5th percentile	36 (78/3)
> 5th percentile	10 (21/7)
Accompanying disease, n(%)	
No URI	30 (65/2)
URI	16 (34/8)
Diagnoses	
Cyanotic CHD, n(%)	
TOF	14 (61)
TGA	3 (13)
Single atrium, single ventricle	1 (4)
Tricuspid atresia	1 (4)
DORV	1 (4)
Type-A truncus arteriosus	2 (9)
TAPVR	1 (4)
Acyanotic CHD, n(%)	
VSD	7 (30)
PDA	6 (26)
ASD	8 (35)
AVSD	1 (4)
PS	1(4)

ToF=tetralogy of Fallot, TGA=transposition of the great arteries, DORV=double outlet right ventricle, TAPVC=total anomalous pulmonary venous connection, VSD=ventricular septal defect, PDA=persistent ductus arteriosus, ASD=atrial septal defect, AVSD=atrial-ventricular septal defect,

In subjects with cyanotic CHD, 34.8% had <5th percentile growth velocity, while in the acyanotic group, only 8.7% had similarly poor growth. Chi-square analysis revealed a significant correlation between the cyanotic CHD and impaired growth velocity. Also, patients with cyanotic CHD had 5.6 times higher risk of FTT (OR 5.600; 95%CI 1.038 to 30.204; P=0.032) compared to acyanotic CHD patients (**Table 2**).

As shown in **Table 3**, 31.3% of URI patients had <5th percentile growth velocity compared to 16.7% of those without URI. Chi-square test revealed no significant association between URI and FTT (OR 2.273; 95%CI 0.545 to 9.479; P=0.253).

Table 2. Cyanotic and acyanotic CHD and growth velocity

Types of congenital heart disease	Growth velocity		Total (N=46)	OR (95%CI)	P value
	> 5th percentile (n=10)	≤5th percentile (n=36)			
Cyanotic (n=23)	8	15	23	5.600 (1.038 to 30.204)	0.032
Acyanotic (n=23)	2	21	23		

Table 3. URI and growth velocity

Accompanying disease	Growth velocity		Total (N=46)	OR (95%CI)	P value
	> 5th percentile (n=10)	≤5th percentile (n=36)			
URI (n=16)	5	11	16	2.273 90.545 TO 9.479)	0.253
No URI (n=30)	5	25	30		

Discussion

Children's growth velocity can be determined by the 2009 WHO Growth Velocity Chart.[7] The chart can also be used to assess children's vulnerability to FTT. A child is diagnosed with FTT if his/her weight is < 5th percentile. We compared FTT in children with cyanotic to acyanotic CHD. Failure to thrive in CHD patients has no clear etiology, but may be due to multiple contributing mechanisms, including inadequate caloric intake, decreased appetite, malnutrition caused by hypoxia, malabsorption due to venous congestion, increased use of energy, relative hypoxia, increased need for oxygen, endocrine adaptation, and recurring respiratory infection. Hypoxia is a result of an imbalance between the demand and supply of oxygen. In addition, CHD causes chronic hypoxia, resulting in low levels of IGF-1 in the patient's endocrine system.[6-11]

This analytical study was done in patients who underwent routine medical check-ups at the Pediatrics Cardiology Specialist Unit at Dr. Moewardi Hospital, Surakarta. In our study, there were more female CHD patients (58.7%) than males (41.3%). In contrast, Mahapatra et al. reported that more male patients (54.5%) suffered from CHD compared to females, with a ratio of 1.2 to 1. However, Batte *et al.* found more females with CHD (57.2%) than males.[12,13]

The most common cause of acyanotic CHD is VSD (35-30%), and that of cyanotic CHD is TOF (5-7%).[12] In our study, the most of our cyanotic CHD subjects had TOF (61%), and most of our acyanotic subjects had ASD (35%). Nasiruzzaman et *al.* reported that the most prevalent cyanotic CHD etiology was TOF (26%). Similar to our findings, Atwa *et al.* reported that the frequency of ASD in acyanotic CHD was higher (28.8%) than VSD (28.2%). However, Mahapatra et al. in India reported VSD as the most prevalent acyanotic CHD (36.3%), and TOF as the most prevalent cyanotic CHD (11.25%).[12,14,15]

In our study, 80% children with cyanotic CHD suffered from FTT. A previous study reported that 55.9% patients with CHD suffered from FTT.[16] Another study also reported FTT in 55% of neonates with VSD and TOF CHD.6 In addition, Harshangi *et al.* reported FTT complications among 56% of CHD patients.[17] Furthermore, Batrawy *et al.* reported that 60% cyanotic CHD patients suffered from growth anomalies.[9] Artiko *et al.* reported that patients with acyanotic CHD patent ductus arteriosus suffered from growth anomalies before a catheterization action was taken.[18] However, Nasiruzzaman *et al.* reported FTT occurrence in only 13% of children with CHD.[19]

We found that cyanotic CHD patients have a 5.6 times higher risk of FTT (OR 5.600; 95%CI 1.038 to 30.204) compared to acyanotic CHD patients. Batte et al. in Uganda found that CHD patients who suffered from growth anomalies, as determined by the 2006 WHO Nutritional Chart, also showed similar findings of increased FTT.[13]

In the ≥ 5th percentile category, 69.4% of patients did not suffer from acute URI and did not experience FTT, whereas 50.0% of patients in the < 5th percentile category suffered from URI

and experienced FTT. We noted that URI as an accompanying disease increased the tendency to have FTT (OR 2.273; 95%CI 0.545 to 9.479) but there was no statistically significant correlation between URI and growth velocity (P=0.253). Gabriela *et al.* reported that CHD patients who suffered from acute lower respiratory tract infection (ALRTI) cited bronchopneumonia as the most prevalent disease (86.6%).19 In addition, Medrano et al. reported that 13.5% of CHD patients were hospitalized due to respiratory tract infection.[20]

A limitation of our study was that other factors may cause failure to thrive, such as low body weight at birth, short pregnancy period, socioeconomic issues and malnutrition, none of which were evaluated in this study. In conclusion, cyanotic CHD poses higher risk of failure to thrive compared to acyanotic CHD.

Conflict of Interest

None declared.

References

1. Khan I, Muhammad A, Muhammad T. Pattern of congenital heart disease at Lady Reading Hospital Peshawar. Gomal J Med Sci. 2011;9:174-7.
2. Van der Linde D, Konings EE, Slager MA, Witsenburg M, Helbing WA, Takkenberg JJ, *et al.* Birth prevalence of congenital heart disease worldwide: a systematic review and meta-analysis. J Am Coll Cardiol. 2011;58:2241-7.
3. Richards AA, Garg V. Genetics of congenital heart disease. Curr Cardiol Rev. 2010;6:91-7.
4. Chen CW, Li CY, Wang JK. Growth and development of children with congenital heart disease. J Adv Nurs. 2004;47:260-9.
5. Irwanto, SA, Narendra MB. Penyimpangan tumbuh kembang anak. 29 July 2006. [cited 2016 January 17]. Available from: http://old.pediatrik.com/pkb/061022022956-57x6138.pdf.
6. Noble K, Forsey J. An analysis of growth failure in children with congenital heart disease. Welsh Paediatr J. 2010;32:13-6.
7. WHO. The WHO multicenter growth reference study (MGRS). 2009.
8. Hubschman LE. Malnutrition in congenital heart disease: management to improve outcomes. Infant, Child, and Adolescent Nutrition. 2013;5:170-6.
9. Batrawy SRE, Tolba OARE, El-Tahry AM, Soliman MA, Eltomy M, Habsa A. Bone age and nutritional status of toddlers with congenital heart disease. Res J Pharm Biol Chem Sci. 2015;3:940-9.
10. Eren E, Cakir EDP, Bostan O, Saglam H, Tarim O. Evaluation of the endocrine functions in pediatric patients with cyanotic congenital heart disease. Biomedical Res. 2013;24:77-81.
11. Al-Asy HM, Donia AA, El-Amrosy DM, Rabee E, Bendary AA. The levels of ghrelin in children with cyanotic and acyanotic congenital heart disease. J Pediatr Sci. 2012;6:e209.
12. Mahapatra A, Sarangi R, Mahapatra PP. Spectrum of congenital heart disease in a tertiary care centre of Eastern india. Int J Contemporary Pediatr. 2017;4:1-3.
13. Batte A, Lwabi P, Lubega S, Kiguli S, Otwombe K, Chimoyi L, et al. Wasting, underweight and stunting among children with congenital heart disease presenting at Mulago Hospital, Uganda. BMC Pediatr. 2017;17:10.
14. Nasiruzzaman AHM, Hussain MZ, Baki MA, Tayeb MA, Mollah MN. Growth and developmental status of children with congenital heart disease. Bangladesh Med J. 2011;40:54-57.
15. Atwa ZT, Safar HH. Outcome of congenital heart diseases in Egyptian children: is there gender disparity? Egypt Pediatr Assoc Gazette. 2014;62:35-40.
16. Vaidyanathan B, Nair SB, Sundaram KR, Babu UK, Shivaprakasha K, Rao SG, *et al.* Malnutrition in children with congenital heart disease (CHD) determinants and short term impact of corrective intervention. Indian Pediatr. 2008;45:541-6.
17. Harshangi SV, Itagi LN, Patil V, Vijayanath V. Clinical study of congenital heart disease in infants in tertiary care hospital. JPSI. 2013;2:15-8.
18. Artiko B. Percepatan pertumbuhan anak dengan patent ductus arteriosus sebelum dan sesudah kateterisasi penutupan [thesis]. [Surakarta]: Fakultas Kedokteran Universitas Sebelas Maret; 2015.
19. Gabriela K, Kuswiyanto RB, Dwiyatnaningrum F. Clinical characteristic and outcome of acute lower respiratory tract infection in children with congenital heart disease. Althea Med J. 2015;2:403-8.
20. Medrano Lopez C, Garcia-Guereta Silva L. Respiratory infections and congenital heart disease: two seasons of the CIVIC study. An Pediatr. 2007;67:329-36.