Paediatrica Indonesiana

p-ISSN 0030-9311; e-ISSN 2338-476X; Vol.59, No.2(2019). p.87-91; doi: http://dx.doi.org/10.14238/pi59.2.2019.87-91

Original Article

Diagnostic value of electrocardiography for ventricular septal defects

Besse Sarmila, Burhanuddin Iskandar, Dasril Daud

Abstract

Background Congenital heart disease (CHD) in children requires attention from medical practitioners, because CHDs that are diagnosed early and treated promptly have good prognoses. Ventricular septal defect (VSD) is the most common type of congenital heart disease.

Objective To compare the accuracy of electrocardiography (ECG) to echocardiography in diagnosing VSD.

Methods This diagnostic study was conducted from November 2013 until July 2015. It involved patients with acyanotic CHDs who were suspected to have VSD at Dr. Wahidin Sudirohusodo Hospital, Makassar, South Sulawesi.

Results Of 114 children screened, 97 were included and analyzed. The frequency of positive VSD was 69.1% based on ECG, and 99% based on echocardiography. There was a significant difference between ECG and echocardiography (P=0.000). However, when small VSDs were excluded, there was no significant difference between the two diagnostic tools [(P=1.000), Kappa value was 0.66, sensitivity was 98.5%, specificity was 100%, positive predictive value (PPV) was 100%, and negative predictive value (NPV) was 50%].

Conclusion There were significant differences between the ECG and echocardiography, for diagnosing VSD. However, if small VSDs were not included in the analysis, there was no difference between the two examinations, suggesting that ECG might be useful for diagnosing VSD in limited facilities hospitals. **[Paediatr Indones. 2019;59:87-91; doi: http://dx.doi.org/10.14238/pi59.2.2019.87-91]**.

Keyword: *acyanotic CHD; VSD; electrocardiography; echocardiography*

Pediatric congenital heart disease (CHD) requires attention from medical practitioners, especially physicians, because early diagnosis of CHD followed by prompt treatment results in improved prognoses. Late detection of congenital heart disease in infants or children delays their treatment, not only causing children to suffer, but prolonging hardship for parents and families. Therefore, diagnosis and management of CHD should be done as early as possible, so that the child survives with a better future.[1]

Although more sophisticated diagnostic tools such as echocardiography for CHD diagnosis exist, the roles of history, physical examination, and diagnostic tools such as electrocardiography (ECG) are not less important. Electrocardiography is a non-invasive, practical, as well as inexpensive diagnostic tool, and is available in a variety of places. However, examinations using an ECG should still be based on history and

From the Department of Child Health, Universitas Hasanuddin Medical School/Dr. Wahidin Sudirohusodo Hospital, Makassar, South Sulawesi, Indonesia.

Corresponding author: Besse Sarmila, Department of Child Health, Hasanuddin University Medical School/Dr. Wahidin Sudirohusodo Hospital, Jl. Perintis Kemerdekaan Km. 10, Tamalanrea Makassar 90245, South Sulawesi, Indonesia. Telp. (62)411584461; Email: bessesarmila07@gmail.com.

Submitted November 26, 2018. Accepted March 29, 2019.

physical examination, because some CHDs present similarly by electrocardiogram.[1] In ventricular septal defects (VSD), hemodynamic changes occur due to an interventricular defect. Due to left-to-right shunt, the left cardiac output changes. Those circumstances lead to left atrial enlargement (LAE) and left ventricular hypertrophy (LVH). If these conditions are continuous, the pressure in the right ventricle will also increase, possibly leading to right ventricular hypertrophy (RVH). Electrocardiography can record the presence of LAE, LVH, and RVH.[2] Therefore, it is important to compare the findings of ECG to those of echocardiography.

In children with prolonged undetected and untreated CHD, 50% may die in the first month of life or 70% may die in the first year of life.[3,4] Echocardiography is the standard diagnostic tool for VSD, but it can be performed only in certain places by experts. Therefore, we aimed to investigate the usefulness of ECG in diagnosing VSD, for early detection and treatment, in order to reduce morbidity and mortality rates. To our knowledge, the diagnostic value of electrocardiography for VSD has never been studied in Indonesia. As such, we aimed to assess the accuracy of electrocardiography in diagnosing ventricular septal defect compared to echocardiography.

Methods

This diagnostic study was conducted at Dr. Wahidin Sudirohusodo Hospital, Makassar, South Sulawesi, from November 2013 to July 2015 with primary and secondary data, as well as measurements performed by cross-sectional method. This study compared ECG and echocardiography as the gold standard. The study subjects were the population of children with acyanotic congenital heart disease and suspected VSD who met the inclusion criteria, which were acyanotic CHD presenting with pansystolic murmur on ICS III-IV with or without other murmur, aged 3 months to 15 years, had complete medical records, and parents' willingness to participate in the study. Patients were excluded if their ECG recording could not be read. Primary and secondary data were grouped according to destination and type, then were analyzed by univariate analysis, McNemar test, or association coefficient. The result of electrocardiography would be interpreted by verificator. There were validity test and reliability test among verificators with McNemar analysis. Patients with VSD diagnosis were grouped into small VSD (diameter <3mm), moderate VSD (diameter 3-8 mm), and large VSD (diameter ≥8mm).

This study was approved by the Research Ethics Committee of the Universitas Hasanuddin Medical School.

Results

From November 2013 to July 2015, there were 114 CHD patients who were suspected to have VSD. However, 17 children were excluded resulting in a total of 97 subjects. **Table 1** shows the subjects' characteristics: age, sex, nutritional status, major defect, and type of defect. Of 97 subjects, 50 (51.5%) were female. The mean age of the subjects was 4 years and 15 days, with a range of 3 months to 13 years and 3 months. Subjects' nutritional status types were poorly nourished in 41 (42.3%), undernourished in 36 (37.1%), and good nutrition in 20 (20.6%) subjects. The VSD sizes were small in 28 (28.8%) subjects, moderate in 42 (43.2%) subjects, and large in 27 (28%) subjects. Isolated VSD was found in 82 (84.5%) subjects, while the complex VSD was found in 15 (15.5%) subjects.

Intra-examiner reliability and validity in assessing ECG was analyzed in 10 subjects with McNemar and Kappa tests (**Table 2** and **Table 3**).

Table 1. Study subjects' characteristics

Characteristics	Total (N=97)
Age, years	
Mean (SD)	4.04 (3.59)
Median (range)	3.00 (0.25 – 13.25)
Sex, n (%)	
Female	50 (51.5)
Male	47 (48.5)
Nutritional status, n (%)	
Well-nourished	20 (20.6)
Undernourished	36 (37.1)
Poorly-nourished	41 (42.3)
Defect size, n (%)	
Small VSD	28 (28.8)
Moderate VSD	42 (43.2)
Large VSD	27 (28)
Type of VSD, n (%)	
Isolated	82 (84.5)
Complex	15 (15.5)

Table 2. Analysis of intra-examiner ECG interpretation reliability

	Verificator I		Total
	LVH	Not LVH	
Verificator II			
LVH	6	0	6
Not LVH	0	4	4
Total	6	4	10

Table 3. Analysis of inter-examiner ECG interpretation validity

	Verificator I		Total
	LVH	Not LVH	
Verificator II			
LVH	6	0	6
Not LVH	0	4	4
Total	6	4	10

McNemar analysis revealed no significant differences among examiner readings and the Kappa value was 1. Hence, intra-examiner reliability and validity were sufficient.

Table 3 shows inter-examiner validity in assessing ECG, as analyzed by McNemar and Kappa tests. McNemar analysis no significant differences and the Kappa value was 1. Hence, inter-examiner assessments had a strong degree of conformity.

The diagnostic value of ECG was compared to echocardiography. VSD was detected in 67 (69.1%) subjects using ECG and 96 (99%) subjects using echocardiography (**Table 4**). McNemar analysis revealed a significant difference between the two examinations (P=0.000), with a Kappa value of 0.045, indicating a weak degree of conformity between the two examinations.

After the small VSD subjects were excluded from the analysis, the frequency of VSD occurrence based on ECG was 66 (97.1%) subjects and 67 (98.5%) subjects based on echocardiography. McNemar analysis revealed a no significant difference between the two examinations (P=1.000), with a Kappa value of 0.66, indicating that the degree of conformity between the two examinations was moderate (**Table 5**). For diagnosing VSD, electrocardiography sensitivity was 98.5%, specificity was 100%, positive predictive value was 100%, and negative predictive value was 50%.

Table 4. Analysis of VSD occurrence based on ECG and echocardiography

	Echocardiography		Total
	VSD	Not VSD	
Electrocardiography			
VSD, n (%)	67 (69.1)	0 (0)	67 (69.1)
Not VSD, n(%)	29 (29.9)	1 (1)	30 (30.9)
Total, n(%)	96 (99)	1 (1)	97 (100)

There was one subject in our study with an echocardiogram that showed ASD, and it ECG result confirmed that the subject had no VSD because there was RVH on electrocardiogram recordings. The subject was included in the sample, as this was due to misinterpretation of a murmur.

Discussion

In this study, suspected VSD in acyanotic CHD was more frequent in girls than boys (51.5% *vs.* 48.5%, respectively). Similarly, a previous study in Canada reported a slightly higher CHD incidence in girls (52%).[5] The average age of patients with suspected VSD was 4 years and 15 days, with the highest occurrence in 3-year- olds. In contrast, Shah *et al.* showed that the age of 1 month to 1 year (46.4%) was most common in terms of suspected VSD, while there were only 9.5% in neonates.[6] Neonates were not included in our study criteria since heart murmurs at such an age are difficult to detect because of the high pressure in the right heart chamber. Nonetheless, the average age of the subjects was quite high (4 years and 15 days), indicating that in general, early detection ability was still poor.[1]

The VSD types found in our subjects were isolated VSD (82 subjects, 84.5%) and VSD with other CHDs such as atrial septal defect (ASD), patent

Table 5. Analysis of moderate-large VSD occurrence based on ECG and echocardiography

	Echocardiography		Total
	VSD	Not VSD	
Electrocardiography			
VSD, n (%)	66 (97.1)	0 (0)	66 (97.1)
Not VSD, n(%)	1 (1.4)	1 (1.5)	2 (2.9)
Total, n(%)	67 (98.5)	1 (1.5)	68 (100)

ductus arteriosis (PDA), and transposition of the great arteries (15 subjects, 15.5%). Hariyanto in Padang also observed isolated VSD (16 cases, 45.7%) and VSD with other CHDs [ASD (7 cases, 20%) and PDA (3 cases, 8.5%)].7

The CHD patients mostly poorly nourished (42.3%), however, 20.6% of patients had good nutritional status, and 37.1% had undernourished Likewise, another study in Bandung showed that VSD patients tended to poorly nourished, where 24% of VSD patients had severe malnutrition, 16% had moderate malnutrition, 33% had mild malnutrition, and 27% patients had good nutrition.[8] Malnutrition was mostly found in patients with large VSDs. The presence of pulmonary hypertension was a risk factor related to the occurrence of malnutrition. Patients with increased blood flow to the lung and pulmonary hypertension had an increased chance of malnourishment and stunted growth.[9] Malnourished children are more prone to infection, which further aggravates their condition. Furthermore, anorexia, inadequate nutritional intake, hypoxemia, hyper-metabolic status, acidemia, cation imbalances, reduced peripheral blood flow, chronic decompensated heart disease, malabsorption, protein loss, recurrent respiratory infection, hormonal factors, and genetics may also eventually lead to malnutrition in CHD patients.[10]

In our study, the frequency of VSD based on ECG was 69.1%, while the frequency of VSD based on echocardiography was 99%. Statistical analysis revealed that the two tests were significantly different (P=0.000). In general, ECG capability in diagnosing VSD is not comparable to echocardiography because of the high frequency of small VSDs (28.8%) with normal ECG. Biologically, small VSDs produce normal electrocardiograms, because the light volume overload in the left heart is undetectable in electrocardiogram recordings.[1]

After small VSDs were excluded in our analysis, VSD occurrence based on ECG was 97.1%, while the VSD occurrence based on echocardiography was 98.5%. Statistical analysis revealed no significant difference between the frequencies of positive VSDs from the two examinations. In other words, ECG capability in diagnosing VSD was comparable to echocardiography, if the defect size was moderate-large. ECG sensitivity compared to echocardiography as the gold standard in moderate-large VSDs was 98.5%, which means that ECG could be used to precisely diagnose moderate-large VSDs. ECG specificity in moderate-large VSD was 100%. As such, ECG could be used to rule out the possibilty of moderate-large VSDs. The positive predictive value was 100%, indicating that a positive VSD result based on ECG was 100%. However, the negative predictive value of the ECG for diagnosing moderate-large VSDs was 50%, indicating that possibly half of patients with negative VSD results based on ECG might actually have a VSD. Hence, the benefits of the application for diagnostics lies in the positive predictive value. If a patient is clinically suspected of having VSD with acyanotic CHD and the ECG shows signs of LVH, then it strongly suggests that the patient has a moderate-large VSD and must be treated. The negative predictive value of only 50% means that if the patient's ECG is normal, then the patient should be immediately referred to tertiary care to be further evaluated by echocardiography. Results of this study can only be applied to acyanotic CHD patients with suspected VSD. Acyanotic VSD patients who have pulmonary hypertension should be referred to tertiary care for echocardiography and timely treatment, as pulmonary hypertension can lead to Eisenmenger syndrome.

Biologically, ventricular septal defect shows signs of LVH in electrocardiogram recordings because of volume overload in the left ventricle, while RVH is caused by volume overload in the right heart and pulmonary stenosis is found in ASD.[1] In addition, a subject with moderate VSD by echocardiography but normal electrocardiogram was considered to be a false negative. Conceptually, this VSD with a normal electrocardiography result was because in moderate VSD, significant hemodynamic disturbances occur in defects > 5 mm.[11]

The strength of this study was the large sample size. Determination of the number of samples by using the VSD occurrence frequency is high at around 30%. Electrocardiography and echocardiography results were interpreted by a pediatric cardiologist, followed by verification of their validity and reliability. Moreover, this study was conducted at Dr. Wahidin Sudirohusodo Hospital, which is the national referral hospital in Eastern Indonesia. Therefore, the data are representive of acyanotic congenital heart disease in Eastern Indonesia. A limitation of this study was that the capability of ECG to diagnose VSD

accompanied by cyanotic congenital heart disease was not performed.

In conclusion, there are significant differences between ECG and echocardiography for diagnosing VSD in children with acyanotic CHD. If small VSDs are not included in the analysis, there is no significant difference between the two examinations. As such, ECG may be useful for identifying moderate-large VSDs.

Conflict of Interest

None declared.

Funding Acknowledgment

The authors received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

References

1. Park MK. The Pediatric Cardiology Handbook. 4th ed. Philadelphia: Mosby Elsevier; 2010. p.140-44.
2. Bernstein D. Acyanotic congenital heart disease: Kliegman, Berhman, Jenson, Stanton, editors. Nelson textbook of pediatrics. 19th ed. Philadelphia: Saunders Elsevier. 2011. p.1571.
3. Meberg A, Otterstad JE, Froland G, Lindberg H, Sorland SJ. Outcome of congenital heart defects- a population based study. Acta Paediatr. 2000;89:1344-51.
4. Cahyono A, Rachman MA. The cause of mortality among congenital heart disease patients in Pediatric Ward, Soetomo General Hospital (2004-2006). Indones J Kardiologi. 2007;28:279-84.
5. Marelli AJ, Mackie AS, Ionescu-Ittu R, Rahme E, Pilote L. Congenital heart disease in the general population: changing prevalence and age distribution. Circulation. 2007;115:163-7.
6. Shah GS, Singh MK, Pandey TR, Kalakheti BK, Bhandari GP. Incidence of congenital heart disease in tertiary care hospital. Kathmandu Univ Med J. 2008;6:33-6.
7. Hariyanto D. Profil penyakit jantung bawaan di instalasi rawat inap anak RSUP Dr. M. Djamil, Padang Januari 2008 – Februari 2011. Sari Pediatri. 2012;14:152-7.
8. Rahayuningsih SE. Hubungan antara defek septum ventrikel dan status gizi. Sari Pediatri. 2011;13:137-41.
9. Shrivastava S. Malnutrition in congenital heart disease. Indian Pediatr. 2008;45:535-6.
10. Fyler DC, Geggel RL. History, growth, nutrition, physical examination and routine laboratory test. In: Keane F.J, Lock JE, Fyler DC, editors. Nada's pediatric cardiology. 2nd ed. Philadelphia: Elsevier Saunders; 2007. p. 129-44.
11. Kimball TR, Daniels SR, Meyer RA, Hannon DW, Khoury P, Schwartz DC. Relation of symptoms to contractility and defect size in infants with ventricular septal defect. Am J Cardiol. 1991;67;1097-1102.