Correlation between Oxygen Saturation and Hemoglobin and Hematokrit Levels in Tetralogy of Fallot Patients

Farhatul Inayah Adiputri,[1] Armijn Firman,[2] Arifin Soenggono[3]
[1]Faculty of Medicine Universitas Padjadjaran, [2]Department of Child Health Faculty of Medicine, Universitas Padjadjaran/Dr. Hasan Sadikin General Hospital Bandung, [3]Department of Anatomy and Cell Biology Faculty of Medicine Universitas Padjadjaran

Abstract

Background: Hemoglobin and hematocrit levels increase in Tetralogy of Fallot (TOF) but the oxygen saturation declines. Reduced hemoglobin in circulating blood as a parameter of cyanosis does not indicate rising hemoglobin due to the 'not-working' hemoglobins that affect the oxygen saturation. Increasing hematocrit is the result of secondary erythrocytosis caused by declining oxygen level in blood, which is related to the oxygen saturation. This study was conducted to find the correlation between oxygen saturation and hemoglobin and hematocrite levels in TOF patients.
Methods: This study was undertaken at Dr. Hasan Sadikin General Hospital in the period of January 2011 to December 2012 using the cross-sectional analytic method with total sampling technique. Inclusion criteria were medical records of TOF patients diagnosed based on echocardiography that included data on oxygen saturation, hemoglobin, and hematocrite. Exclusion criteria was the history of red blood transfusion.
Results: Thirty medical records of TOF patiens from Dr. Hasan Sadikin General Hospital Bandung were included in this study. Due to skewed data distribution, Spearman correlation test was used to analyze the data. There was a significant negative correlation between oxygen saturation and hematocrit level (r= -0.412; p=0.024) and insignificant correlation between oxygen saturation and hemoglobin (r=-0.329; p= 0.076).
Conclusions: There is a weak negative correlation between oxygen saturation and hematocrite levels. **[AMJ.2016;3(1):152–5]**

Keywords: Hematocrit, hemoglobin, oxygen saturation, tetralogy of fallot

Introduction

Tetralogy of Fallot (TOF) is the most frequent disease found in patients with cyanotic congenital heart disease. Nevertheless, not all TOF patients experience cyanosis because it depends on the severity of the disease.[1] Cyanosis occurs due to several factors, including decreased pulmonary blood flow resulting in the obstruction of the pulmonary valve, known as pulmonary stenosis, making oxygen distribution to the tissue inadequate and increasing right to left shunt.[2] In addition, another factor that may cause cyanosis is drastically reduced hemoglobin level in circulating blood that affects oxygen saturation.[3] Oxygen saturation indicates the amount of oxygen bound by hemoglobins in a percentage of maximum binding at the time of measurement. One of the variables that affect oxygen saturation is hemoglobin concentration. However, it is not sure whether there is a correlation between oxygen saturation and hemoglobin in TOF.

Hematocrit increases in TOF patients as a response to hypoxia. The oxygen delivery to tissues depends on many factors, particularly the total number of circulating erythrocytes, systemic arterial oxygen tension (PaO2), oxygen saturation, oxygen-hemoglobin dissociation curve position, cardiac output, and regional blood flow.[4,5] Theoretically, rising hematocrit with declined oxygen saturation indicate increase in erythrocyte production to supply increased demand of oxygen in the tissue.[5] It is postulated that there might be a correlation between oxygen saturation and hematocrit. This study aimed to find the

Correspondence: Farhatul Inayah Adiputri, Faculty of Medicine, Universitas Padjadjaran, Jalan Raya Bandung-Sumedang Km.21, Jatinangor, Sumedang, Indonesia, Phone: +6285720471165 Email: tijannisa@gmail.com

correlation between oxygen saturation with hemoglobin and hematocrit levels in patients with TOF.

Methods

This study was performed in October 2013 through the use of secondary data, i.e. medical records. This study has been approved by the Health Reasearch Ethics Committee, Faculty of Medicine, Universitas Padjadjaran and Dr. Hasan Sadikin General Hospital Bandung. The inclusion criterion in this study was medical records from patients with tetralogy of Fallot diagnosed by echocardiography that includes information on hemoglobin, hematocrit, and saturation oxygen levels. The exclusion criteria were incomplete medical record and TOF patients who received packed red cell transfusion. In this study, data were not classified by age or sex.

From the medical records, we obtained data on oxygen saturation and hematologic examination (hemoglobin level and hematocrit level) in which the results of hematologic data were taken from the Clinical Pathology Department of Dr. Hasan Sadikin General Hospital, Bandung. Oxygen saturation was measured using pulse oximeter by the examiner. Oxygen saturation value collected was the first value noted in the medical record by the examiner. Data were analyzed using Statistical Product and Service Solutions (SPSS) with a normality test by Shapiro-Wilk. Since two of the variables did not have normal distribustion, Spearman correlation test was then used.

Table 1 Baseline Characteristics of the Subjects

Characteristics	n (%)
Age	
0-28 days	1
1-12 months	10
1-2 years	4
2-6 years	3
6-12 years	12
Sex	
Boy	14
Girl	16

Results

There were thirty medical records of TOF in Dr. Hasan Sadikin General Hospital Bandung during the period of January 2011–December 2012 that were included in this study. One patient was excluded because he received packed red cell transfusion. The age range of the subjects when they first visited the hospital was very large. The youngest was 15 days old and the oldest was 12 years old. The average age of the samples was 4.44 years. The sample consisted of 14 boys and 16 girls. Before the correlation was analyzed, data were tested for the the normality of their distribution using Shapiro-Wilk test. It was revealed that the data distribution of hematocrit was normal, but the data on oxygen saturation and

Table 2 Mean and Standard Deviation of the Variables

Variable	Mean	Standard Deviation
Oxygen Saturation (%)	82.45	9.88
Hemoglobin (g/dL)	15.55	11.34
Hematocrit (%)	48	10.63

Table 3 Spearman Correlation Test between Oxygen Saturation and Hemoglobin and Hematocrit Levels

		Hemoglobin (g/dL)	Hematocrit (%)
Oxygen Saturation	r	-0.329	-0.412
	p	0.076	0.024
	n	30	30

Note: r = correlation coefficient; p = p-value; n = sample size

hemoglobin level were not normal. Data were then analyzed using bivariate analysis.

Most of the subjects were in 6–12 years old category. The mean for the oxygen saturation was low with the normal range of healthy children's oxygen saturation of 96–100%.[7]

Table 2 shows that the oxygen saturation correlated negatively with hemoglobin (r=-0.329); however, the the correlation was not statistically significant (p>0,05). There was a significant correlation between the oxygen saturation and hematocrit (p<0.05). Both correlative values revealed negative and moderate correlations.

Discussion

The statistic results showed that oxygen saturation and hematocrit correlated inversely. This is in line with a study that reveals that the best correlation was obtained between oxygen saturation and hematocrit, r=-0.74.[5] The increased hematocrit level due to secondary erythrocytosis usually occurs in cyanotic congenital heart disease (CCHD) because of the physiological response when the tissue experiences hypoxia. Patients experience hypoxemia due to reduced oxygenated blood which is represented as the oxygen saturation. Consequently, this stimulates bone marrow to produce erythrocytes by first excreting erythropoietin from kidney. Increased erythrocyte level, known as erythrocytosis, will increase red blood cell mass, hematocrit, and viscosity. This condition causes hyperviscosity in cyanotic congenital heart diseases, marked by increased hematocrit.[4] Iron deficiency anemia also becomes a preciptitating factor for hyperviscosity.[9] Finally, hiperviscosity can decrease blood flow to the tissue; thus, the amount of oxygen delivered to the tissue declines.

Furthermore, in TOF patients, the pulmonary blood flow (PBF) decreases because of the anatomy malformation in the development of heart during pregnancy, i.e. pulmonary stenosis. It causes reduced perfussion to the tissue. The manifestation includes cyanosis, in addition to the rising of hematocrit.[10] Therefore, the inverse correlation between the oxygen saturation and hematocrit level in Tetralogy of Fallot was moderate in this study.

Clinically, the increased hemoglobin can decrease the degree of right to left shunt and systemic vascular resistance as well as increasing the pulmonary blood flow, oxygen transport to the tissue, and aortic oxygen saturation as the physiologic responses to cyanosis.[11] Thus, oxygen saturation often relates to hemoglobin as the compensation because decreased oxygen saturation will led to higher erythrocyte production to comply to the oxygen need. However, the statistical test results showed no correlation between oxygen saturation and hemoglobin level despite the fact that studies on cyanotic congenital heart diseases reveals that correlation between oxygen saturation and hemoglobin has the same pattern as the correlation between oxygen saturation and hematocrit.[5] This means that there was a correlation between oxygen saturation and hemoglobin level albeit insignificant.

In another study, cyanosis affects the rise in hemoglobin level but no significant change in oxygen saturation. The increased hemoglobin level will increase the oxygen capacity binding without any change in oxygen saturation and provide greater oxygen supply for the tissue. However, clinical manifestations, such as cyanosis, still exists in an even more severe state despite the clinical improvement in this phase. This is known as relative anemia phenomenon.[3] This occurrs due to many factors. Not all TOF patients experience cyanosis or relative anemia. If cyanosis presents, the degree of cyanosis is different among patients depending on the severity of the obstruction pulmonary valve, the degree of right ventricular outflow tract obstruction (RVOTO), and the status of the systemic vascular resistance relative to the degree of right ventricular obstruction.[2] The ratio of pulmonary to systemic blood flow leads to patient's condition, which can be fully saturated, cyanotic, or severely cyanotic.[3] The irregular changes in oxygen saturation and hemoglobin level result in different degree of cyanosis.

In conclusion, this study shows that TOF oxygen saturation correlates negatively with hemoglobin and hematocrit levels. Patient's condition can be monitored and the cause of cyanosis, whether it is due to heart disease or not, can be determined using this correlation. Further research on oxygen saturation, hemoglobin, and hematocrit levels as the parameters for selecting the best management for TOF patients, either by surgery or blood transfusion, is needed.

References

1. Bailliard F, Anderson RH. Tetralogy of fallot. Orphanet J Rare Dis. 2009, 4:2

2. Qu JZ. Congenital heart diseases with right-to-left shunts. Int Anesthesiol Clin 2004;42(4):59–72.
3. Rudolph AM, Nadas AS, Borge WH. Hematologic adjustment to cyanotic congenital heart disease. Pediatricts. 1953;11: 454–464.
4. Puspitasari F, Harimurti GM. Hyperviscoucity in cyanotic congenital heart disease. Jurnal Kardiologi Indonesia. 2010;31:41–7.
5. Haga P, Cotes P, Till J, Minty B, Shinebourne E. Serum immunoreactive erythropoietin in children with cyanotic and acyanotic congenital heart disease. Blood.1987;70(3):822–6.
6. Soetjiningsih S. Tumbuh kembang anak. Jakarta: EGC; 2007.
7. Balfour-Lynn DJF, Gringras P, Hicks B, Jardine E, Jones RC, Magee AG,, et al. BTS guidelines for home oxygen in children. Thorax. 2009;64(Suppl 2):ii1–26.
8. Adamson JW, Longo DL. Anemia and polycythemia. In: Longo DL, Fauci AS, Kasper DL, Hauser AL, Jameson JL, Loscalzo J. Harrison's principles of internal medicine. 18th ed. New York: Mc Graw Hill; 2012. p. 450.
9. Rose SS, Shah AA, Hoover DR, Saidi P. Cyanotic congenital heart disease (CCHD) with symptomatic erythrocytosis. J Gen Intern Med. 2007;22(12):1775–7
10. Murphy PJ. The fetal circulation. Contin Educ Anaesth Crit Care Pain. 2005;5(4):107–12.
11. Beekman RH, Tuuri DT. Acute of hemodynamic effects of increasing hemoglobin concentration in children with right to left ventricular and relative anemia. J Am Coll Cardiol. 1985;5(2 Pt1):357–62.