Comparison of Blood Pressure in Beta-Thalassemia Major Patients with that of Control

Bhatkar Sneha M, Shivalkar Surendra S, Mulkutkar Sachin H.
Assistant Professor, Professor, Professor and Head, Department of Physiology, Grant Government Medical College, Mumbai, India.

Abstract

Introduction: β-thalassemia major patients need to have repeated blood transfusions throughout their life for survival, which leads to various complications. Heart disease is the most critical complication and the primary determinant of survival in these patients. Our study shows a comparison of blood pressure between these patients and that of healthy subjects. Methods: The present study was a cross-sectional type of study and consisted of 100 healthy subjects (control group) and 100 patients of beta-thalassemia major (study group). Blood pressure was recorded in both the groups using a mercury sphygmomanometer and statistical analysis of the observations was done using the Z test. Results: There was no statistically significant difference in the Systolic blood pressure (SBP) of the study group as compared to the control group, whereas there was statistically significant difference (p-value <0.0001) in the Diastolic blood pressure (DBP) of the study group. Conclusion: There was an overall reduction in the blood pressure of the study group as compared to the control group, but the difference in SBP was statistically not significant, whereas that of DBP was statistically significant.

Keywords: Beta thalassemia major; Diastolic blood pressure; Systolic blood pressure.

Introduction

The incidence of β-thalassemia in different regions of India varies from 3% to 17%, with a mean prevalence of 4% [1]. Although in the past year’s notable improvements in the therapy of thalassemia major have been realized, constant and progressive cardiac impairment still leads to irreversible cardiac failure, which remains the primary cause of death for these patients [2,3]. It is responsible for more than half of the deaths. It may take the form of cardiomyopathy, pulmonary hypertension, heart failure, arrhythmias, pericarditis, and myocarditis [4,5].

The cardiac output maintains the blood pressure and the total peripheral resistance. These two show a significant inverse relationship; that is, the higher the cardiac output, the lower is the vascular resistance [6].

Spritro et al. reported that blood pressure measured by traditional cuff sphygmomanometer at the time of the doppler examination was within the normal range but slightly and significantly lower in the thalassemia major patients compared with control subjects [7]. In contrast, in the study done by Tabatahaie and co-workers, a relatively high prevalence of elevated blood pressure was reported in β-thalassemia major patients [8]. Some studies state that, in addition to cardiac complications (such as iron deposition in myocytes), this disease probably has a protective role against heart diseases by lowering blood pressure [9,10].

The present study was done to find out the presence of impairment of blood pressure in beta-thalassemia major patients without any clinical evidence of heart disease.

Materials and Methods

The present study was a cross sectional analytical type of study conducted in the Department of Physiology and Department of Paediatric Medicine, Grant Government Medical College, Mumbai and in the Thalassemia unit of St. George Hospital, Mumbai. Before commencement of the project, approval was taken from the Institutional Ethical Committee. Written informed consent was taken before the clinical examination of the subject.

The study design involved 200 individuals who were divided into two groups of 100 normal subjects (control group) and 100 patients of beta-thalassemia major (study group) between the age group of 8 to 20 years involving both, males and females receiving regular blood transfusions at least since 5 years. Patients with concomitant sickle cell anaemia or with associated congenital or acquired heart disease, diabetes mellitus, thyroid disorders or any other endocrine disorder or on long term medications for any other chronic disease were excluded from the study. Written informed consent was taken before the clinical examination of the subject.

The subjects were asked to refrain from ingesting any beverages containing caffeine and alcohol for at least 12 hours prior to the study. They were asked to report between 10 a.m. to 12 p.m. in the lab after an adequate night’s sleep followed by light breakfast. A detailed recording of medical, personal and family history and clinical examination of the subjects was carried out. Body weight (in kg) and height (in cm) were taken and BMI was calculated. The Arterial blood pressure of each subject was recorded in the right arm in sitting position using mercury sphygmomanometer. The subject was seated quietly in a chair with back support, with both feet flat on the floor. The patient was instructed to relax as much as possible and to not talk during the measurement procedure [11].

Correspondence: Bhatkar Sneha M, Room No.11, Saidham Building, N. M. Joshi marg, Lower Parel, Mumbai, Maharashtra, India. Email id: snehabhatkar6353@gmail.com

© Authors; 2020. International Journal of Current Research in Physiology and Pharmacology. Sumathi Publications. This is an Open Access article which permits unrestricted non-commercial use, provided the original work is properly cited. (CC BY-NC-SA 4.0)
Statistical analysis of the observations was carried out using Z test. Statistical significance was tested at 5% & expressed in terms of ‘p’ value with p<0.05 as statistically significant.

**Results**

Table 1: General growth parameters, Comparison of Systolic and Diastolic blood pressure of subjects

| Study group | Control group | P value |
|-------------|---------------|---------|
| Mean age (years) | 13.42±4.43 | 14.84±3.92 | 0.0928 |
| Mean height (cm) | 133.45±16.27 | 148.61±18.05 | <0.0001* |
| Mean weight (kg) | 31.05±10.90 | 41.1±13.89 | <0.0001* |
| Mean BMI (kg/m²) | 17.01±3.21 | 18.05±3.74 | 0.1381 |
| SBP (mm Hg) | 101.4±7.16 | 102.8±8.8 | 0.385 |
| DBP (mm Hg) | 57.72±9.54 | 69.64±6.23 | <0.0001* |

Statistical analysis using ‘Z’ test, * Significant (BMI – Body mass index, SBP – Systolic blood pressure, DBP – Diastolic blood pressure)

Comparison of general growth parameters between study group and control group is as shown in table 1. The mean Body mass index (BMI) was 17.01±3.21 and 18.05±3.74 kg/m² in the study and control group respectively. As shown in table there was no statistically significant difference (p>0.05) in systolic blood pressure of study group as compared to control group. There was a difference in diastolic blood pressure of study group and control group which was statistically significant (p value <0.0001).

**Discussion**

The systolic blood pressure of study group and control group was 101.4±7.16 and 102.8±8.8 mmHg respectively and the difference was statistically not significant. However the diastolic blood pressure of study group and control group was 57.72±9.54 and 69.64±6.23 mmHg respectively and the difference was statistically significant. This is in accordance with study done by Veglio et al who observed that in thalassemia major patients, the overall amplitude of systolic BP and diastolic BP was significantly lower than in controls [12].

This may be because the chronic anemics of thalassemia have a high cardiac output state caused by a low systemic vascular resistance. This has been hypothesised to result from vasodilation secondary to reduced inhibition of endothelium derived relaxing factor by low circulating haemoglobin [13]. Nitya Nand et al has shown that patients with severe chronic anemia had significantly low blood pressure as compared to control subjects [14]. Aniruddha et al shown similar findings in iron deficiency anemia patients [15]. According to them this fall in blood pressure may be the result of decreased peripheral resistance due to anemia in iron deficiency anemia.

In β-thalassemia major, endothelial dysfunction is also one of the fundamental pathophysiological mechanisms that play a role in progression of cardiovascular involvements. Many studies have showed there is an endothelial dysfunction in different vascular beds in these patients.

Gullu found that diastolic peak flow velocity of the left anterior descending coronary artery was significantly higher in the β-thalassemia major group at baseline, however, coronary flow reserve (reflecting of microvascular function), is impaired and is significantly lower than control group [16]. Endothelial dysfunction may be because of alteration of nitric oxide synthesis and consumption, inflammation or may be due to hypercoagulable state [17,18].

In contrast to above studies, study done by Tabatabaie and co-workers reported a relatively high prevalence of elevated blood pressure (16.7%) and no dipper statues (56.7%) in β-thalassemia major patients [8]. These findings may be consequent to arterial stiffness, endothelial dysfunction, and autonomic neural dysfunction. Also, study done by Kazemi Jahromi M et al shown that there was no significant difference in blood pressure between thalassemia major patients and normal subjects [19].

**Conclusion**

There was an overall reduction in blood pressure in thalassemia major patients as compared to controls but the difference in SBP was statistically not significant whereas that of DBP was statistically significant. This may be because of chronic anemia resulting into peripheral vasodilation secondary to tissue hypoxia leading to lower vascular resistance or may be due to endothelial dysfunction. Thus, there was impairment of blood pressure in beta thalassemia major patients in a pre-clinical phase of heart disease.

**Source of funding: Self**

**Conflict of Interest: Nil**

**References**

[1] Sood SK, Madan N, Colah R, Sharma S, Apte SV. Collaborative Study on thalassaemia. Report of ICMR Task Force Study. Indian Council of Medical Research. 1993;20-27

[2] Engle MA. Cardiac involvement in Cooley’s anemia. Ann NY Acad Sci. 1969;119:694-702

[3] Zurlo MG, De Stefano, Borgna-Pignatti C, Di Palma A, Piga A, Melevendi C et al. Survival and causes of death in thalassemia major. Lancet. 1989;2:27-30

[4] Eldor A, Rachmilewitz EA. The hypercoagulable state in thalassemia. Blood. 2009;99(1):36-43

[5] Samira ZS, Basma AA, Amr SA. The early cardiac involvement in patients with β-thalassemia major. The Egyptian Heart Journal. 2013;1-7

[6] Roy SB, Bhatia ML, Mathur VS. Hemodynamic effects of chronic severe anemia. Circulation. 1963;28:346-56

[7] Spirit P, Lupi G, Melevendi C, Vecchio C. Restrictive diastolic abnormalities identified by Doppler echocardiography in patients with thalassemia major. Circulation. 1990;82:88-94

[8] Tabatabaie M, Hooman N, Arjmandi-Rafsanjani K, Isafatfeshi R. Ambulatory blood pressure monitoring for children with Beta-thalassemia major: a preliminary. Iran J Kidney Dis. 2013;7:299-303.
Surendra et al. ■ Comparison of Blood Pressure in Beta-Thalassemia Major Patients

[9] Vyssoulis G, Karpanou E, Kyvelou SM, Tzamou V, Triantafyllou A, Theodosiadis G et al. Ambulatory Blood Pressure Profile in Hypertensive Patients with β-Thalassemia Minor. Hypertens Res. 2011;34 (2):253-56

[10] Karimi M, Marvasti VE, Motazedian S, Sharifian M. Is beta-Thalassemia Trait a Protective Factor Against Hypertension in Young Adults? Ann Hematol. 2006;85(1):29-31

[11] Rund D, Rachmilewitz E. Beta-thalassemia. N Engl J Med. 2005;353:1135–46

[12] Veglio F, Melchio R, Rabbia F, Molino P, Genova G, Maritini G et al. Blood pressure and heart rate in young thalassemia major patients Am Heart J. 1998;11:539-47

[13] Anand IS, Chandrashekhar Y, Wander GS. Endothelium-derived relaxing factor is important in mediating the high output state in chronic severe anemia. See comment in PubMed Commons below J Am Coll Cardiol. 1995;25(6):1402-7

[14] Nand N, Mohan R, Khosala SN, et al. Autonomic function test in chronic severe anemia. J Assoc Physicians. Ind. 1989;37(8):508-10.

[15] Aniruddha Jibhkate, Sonali Pande. Assessment of Autonomic Nervous System Status in Severe Iron Deficiency Anemia Patients Using Valsalva Maneuver Asian Journal of Biomedical and Pharmaceutical Sciences. 2014;4(34):54-8

[16] Gullu H, Caliskan M, Caliskan Z, et al. Coronary Microvascular function, Peripheral Endothelial Function and Carotid IMT in beta-thalassemia minor. Thromb Res. 2013;131:247-52

[17] Aggeli C, Antoniades C, Cosma C. Endothelial dysfunction and inflammatory process in transfusion dependent patients with beta-thalassemia major. Int J Cardiol. 2005;105:80-4

[18] Tantawy AAG, Adly AAM, Ismail EAR, Habeeb NM, Farouk A. Circulating platelet and erythrocyte microparticles in young children and adolescents with sickle cell disease: Relation to cardiovascular complications. Platelets. 2012:1-10

[19] Kazemi Jahromi M, Shahriari Ahmadi A, Mousavikani K. Major Beta-thalassemia: Protective or predisposing Factor for Cardiovascular Diseases. Int J Hematol Oncol Stem Cell Res. 2011;5(1):30-3.