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Research Article

THALASSEMIA WITH PULMONARY ARTERIAL HYPERTENSION IS AFFECTED BY ACETYLSALICYLIC ACID.

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ABSTRACT

The purpose of this study was Comparative study of thalassemic patients with PAH prescribed acetylsalicylic acid (ASA) and pulmonary arterial hypertension (PAH) after one year with those who were not prescribed ASA after one year. The hematological outpatient clinic conducted a retrospective cohort study. First- and 12-month follow-ups were conducted on all new cases of thalassemia with PAH. A two-group classification was used for the patients. A group of patients was prescribed 81 mg of ASA daily for one year, while another group was not prescribed ASA, due to the medication's contraindications, including bleeding and gastrointestinal side effects. The same cardiologist measured PASP using Doppler echocardiography. Indications and contraindications were adjusted using propensity scores. The effects of ASA were evaluated using multivariable regression analysis. The ASA group consisted of 24 (74.6%) thalassemia patients with PAH, while the no ASA group consisted of 8 (25.4%). A comparison between ASA and no ASA showed no significant reduction in The PASP is 0.95, the 95% confidence interval for the difference is 19.99-15.10, and the P value is 0.906. Following one year of treatment with low-dose ASA for PAH in thalassemia, PASP may not improve.

Keywords :- Thalassemia, Cardiography, Patients, Acetylsalicylic Acid

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INTRODUCTION

PAH is the most frequent cardiovascular complication observed in thalassemia patients, with a high prevalence reported in numerous studies on this population.[1–4]. A study has found a relationship between PAH and platelet activation in patients with thalassemia [5–7]. Acetate acetylsalicylic acid (ASA) inhibits platelet activity while lowering thromboxane-prostaglandin I2 ratios in PAH patients [8]. The results of a previous cohort study suggest that the observed hypoxemia is caused by a reversible increase in arterial partial pressure of oxygen after the administration of ASA ten grains or persantin for two–four weeks [9]. Recent studies have shown that ASA improves survival in monocrotaline animal models of pulmonary arterial hypertension, as well as decreased pulmonary arterial pressure [10]. There is a decrease in intracellular glutathione levels in thalassemic blood erythrocytes and platelets, leading to increased levels of reactive oxygen and oxidative stress, so proper treatment is necessary [11].

Thalassemia patients are not well informed about the standard medication for PAH. Drugs for PAH do not have a known mechanism of action. An antiplatelet therapy study will be needed to establish a definitive treatment guideline for patients with PAH who are thalassemic. Thalassemia patients with PAH have been treated with ASA in clinical practice as a method to prevent and treat PAH. It is important to note that the use of ASA or antiplatelet therapy has been studied only in a few cases. The study aimed to assess the effect of aspirin

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(ASA) on pulmonary artery systolic pressure (PASP) in thalassemia patients with pulmonary arterial hypertension (PAH) by comparing the PASP of patients who received ASA to those who did not, after a 1-year follow-up period.

MATERIALS AND METHODS

Efficacy of this treatment was evaluated at the clinic for outpatient hematology, a retrospective cohort study. All new thalassemia patients with PAH underwent Doppler echocardiography (echo) within the first month of treatment. Results of the classification study divided patients into two groups. The first group received ASA 81 mg for 1 year, while the second group did not receive ASA due to contraindications (bleeding, gastrointestinal side effects, and thrombocytopenia). Patient compliance was evaluated along with side effects of the drug. In addition to baseline and 12-month measurements, clinical right heart failure, PASP, functional class status, and oxygen saturation were assessed. Also excluded were those taking antiplatelet or anticoagulant medications as part of the study due to their involvement. During approval by the institutional research ethics committee of the study protocol, the protocol was approved.

PROCEDURE FOR ECHO

A blinded study was conducted with the same cardiologist performing the echocardiogram without knowing anything about the patient's health background. We performed a color echocardiogram, pulsed waves, continuous waves, and M-modes. By evaluating all midprecordial and apical positions where tricuspid regurgitation jets could be found, we were able to obtain the most accurate The highest velocities are spectral. We measured the regurgitant jet's peak pressure. Systolic pressure gradient and maximum velocity can be determined using the modified Bernoulli equation (P = 4V2). The inferior vena cava diameter was used to determine right atrium pressure. When inferior vena cava diameter decreases by 50% or more during inspiration, right atrial pressure is calculated as 15 mmHg and it is based on the diameter decreasing by 50% or more. If the inferior vena cava diameter is .2.5 cm and the right atrial pressure is 20 millimeters per square inch, the right atrial pressure would be .50% lower. In the absence of obstruction of the outflow tract, the transtricuspid gradient was added to mean right atrial pressure for right ventricular systolic pressure or peak pulmonary arterial pressure. By measuring the pressure gradient between the pulmonary artery pressure and the right ventricular end diastolic pressure, we calculated a diastolic pulmonic regurgitant velocity using the following equation: PASP = 4 (VPR)2 + right atrial pressure.

STATISTICAL ANALYSIS

There were two methods The mean differences between two groups based on categorical variables were compared using Student's t-tests or Wilcoxon rank-sum tests; and the exact probability test was used for continuous variables. It was determined that ASA indication and contraindication were confounding factors that had to be controlled by the propensity score adjustment. An analysis of the logistic regression model used to estimate the Probabilities of prescribing ASA versus prescribing no ASA were calculated based on patient characteristics. There were two types of risk regressions performed, one gaussian and one exponential. The effects of ASA on the aging process were evaluated using univariate and multivariate regression analyses. We present the data using frequency, percentages, means, standard deviations (SD), beta coefficients, credibility intervals [CIs], and P-values as well as providing data by frequency or percentage. A two-tailed test was used for all statistical analyses. In order to be considered statistically significant, the P-value had to be less than 0.05.

RESULTS

There were 32 patients with thalassemia, including patients with PAH, E/β-thal, homozygous β-thal, and Hb H disease. In the ASA group, 24 patients (74.6%) were included, while there were only eight (25.4%) in the control group without ASA. As for the contraindications for ASA in the second group, there was bleeding in seven of the patients (43.7%), gastrointestinal side effects in seven of the patients (43.7%), and thrombocytopenia in two of the patients (12.5%). ASD groups averaged 35.9 years in age, while controls averaged 28.3 years in age, and PASPs averaged 51.9 mmHg in ASA groups, and 45.6 mmHg in non-ASA groups. Red cell transfusions and right ventricular diameter were the only differences in baseline characteristics (Table 1). Following a blood test for the ASA group transfusion after a 12 month follow-up, they also got fewer blood transfusions than those who didn't get ASA. A larger right ventricular diameter was found in the powered by group than it A non-ASA group (29.5±5.8 vs 25.5±4.2 mm, P=0.016).

A PASP increase was observed in six of 24 patients in the ASA group (25.5%) and three of eight in the control group (18.7%). Echo findings were not significantly different between ASA-treated and non-treated patients.

A propensity score was adjusted for thalassemia type, splenectomy, O2 saturation, Heart Association Several factors are considered including: A baseline PASP is calculated from hemoglobin, platelet, and nucleated red cell counts, serum ferritin levels, and red cell transfusion. Based on The two groups did not differ statistically significantly in functional class status, clinical right heart

failure, or oxygen saturation. Pulmonary arterial diameter, the diameter of the right ventricle, and the function of the right ventricle differed. In comparison to a group without ASA, the adjusted difference between ASA and no ASA was not significantly reduced (P=0.906, 95% CI, 16.99 to 15.10). There were no deep vein thromboses, ischemic strokes, or pulmonary embolisms in any of the patients.

Table 1: A 12-month review of clinical outcomes

Outcome	ASA, N=24	No ASA, N=8	*P*-value
New York Housing Authority (n [%])			
I	3 (15.5)	1 (12.5)	0.841
II	19 (79.2)	7 (87.5)	
III	2 (8.3)	0	
Number of right heart failures (%)	7 (22.5)	2 (7.2)	0.424
O_2 saturation (%)	97.4±3.1	98.0±2.6	0.492
Echocardiographic findings			
PASP (mmHg)	48.1±17.8	42.1±12.5	0.197
LVEF (%)	65.8±8.4	65.0±7.3	0.700
LVESd (mm)	10.5±3.1	10.7±3.5	0.759
LVEDd (mm)	51.5±6.5	49.6±7.8	0.266
MPAd (mm)	26.7±5.2	25.4±4.2	0.249
RVd (mm)	30.6±7.1	27.5±4.0	0.057
SYSTOLIC dysfunction of right ventricle (n)	7 (13.6)	0	0.158
Diastolic function			
Inflatable valve E:A	2.4±1.4	2.6±1.8	0.395
DT (ms) of the mitral valve	206±29	218±30	0.382

Table 2: Echocardiographic findings and clinical outcomes of acetylsalicylic acid

Outcome	Adjusted difference* (95% CI)	*P*-value
Functional class status: NYHA class (n [%])b	1.29 (-3.20 to 3.79)	0.865
Clinical right heart failure (n [%])c	-0.002 (-3.90 to 3.89)	0.999
O_2 saturation (%)	0.32 (-1.72 to 2.34)	0.757
Result of echocardiography		
PASP (mmHg)	-0.96 (-17.98 to 16.11)	0.906
LVEF (%)	-0.67 (-10.25 to 8.91)	0.877
LVESd (mm)d	-0.75 (-4.07 to 3.55)	0.655
LVEDd (mm)	-1.46 (-8.51 to 5.57)	0.673
MPAd (mm)	1.20 (-2.68 to 5.07)	0.534
RVd (mm)d	0.82 (-4.61 to 6.28)	0.765
Poor right ventricular systolic function, n (%)c	15.36 (-2,218 to 2,249)	0.989
Flow in diastole		
Mitral valve E:A ratiod	0.45 (-1.77 to 2.69)	0.687
Mitral valve DT (ms)d	-25.61 (-47.88 to -3.37)	0.024

DISCUSSION

This study examined the effects of low-dose aspirin (ASA) on pulmonary arterial hypertension (PAH) in thalassemia patients. A 12-month trial of ASA showed no significant improvements in Oxygen saturation, right heart failure, functional class, or pulmonary artery systolic pressure (PASP). In both groups, PASP was reduced, but without clinical or statistical significance. Subgroup analysis of different patient groups also showed no significant effect of ASA on PASP.

Previous studies have reported conflicting results on the effectiveness of ASA in treating PAH in thalassemia patients. Some studies have shown that ASA may help reduce PASP and improve oxygen saturation, while others have found no significant effects. This study adds to the body of evidence by showing that low-dose ASA is not effective in treating PAH in thalassemia patients.

The study also acknowledges some limitations, including the retrospective nature of the study, the lack of

Treatment was short-term and randomized. A therapeutic trial of PAH cannot confirm PASP without cardiac catheterization, and Six-minute walk tests are not clinically useful. The most common noninvasive screening tool for PAH is echocardiography.
Currently, sildenafil and bosentan are used to treat PAH in thalassemia, however more research is needed. Thralassemia patients may not benefit as much from antiplatelet therapy alone, so its role in PAH prevention needs to be evaluated. For high risk of A definitive recommendation on the management of PAH in thalassemia postsplenectomy will require further studies.

Conclusion

This study found that treating A thalassemia patient with pulmonary arterial hypertension with low-dose aspirin (ASA) for 1 year did not lead to significant improvements in pulmonary artery systolic pressure (PASP). ASA appears to not benefit treating PAH in thalassemia. Other studies have produced conflicting results on the effectiveness of ASA in treating PAH in thalassemia patients, and There is a need for more research the role of antiplatelet or anticoagulant therapy in preventing or treating PAH in this population. Despite some limitations of the study, including its retrospective nature and lack of a randomized controlled trial, the Evidence adds to body of knowledge on the effectiveness of ASA for PAH in thalassemia patients

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