Natural Resources for Human Health
eISSN: 2583-1194

Original Research

Received 25 July 2026
Revised 24 Aug 2026
Accepted 12 Sep 2026

Natural Resources for Human Health 2026, Vol. 6 (20s): 1166–1172

KEYWORDS:
Exercise Therapy; Heart Defects, Congenital; Hypertension, Pulmonary; Inflammation.

Six-Week Exercise Training and Inflammatory Ratios in Adults with Pulmonary Hypertension and Congenital Heart Disease

M. Rheza Rivaldi Salam[1], Yulius Patimang[1], Zaenab Djafar[1], Akhtar Fajar Muzakkir[1], Melda Warliani[2], Andi Alfian Zainuddin[3]

[1]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia
[2]Department of Physical Medicine and Rehabilitation, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia
[3]Department of Public Health and Community Medicine, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia
Corresponding Author*: M. Rheza Rivaldi Salam. Department of Cardiology and Vascular Medicine, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia. Perintis Kemerdekaan Street Number. Km. 11, Tamanlarea, Tamanlarea District, Makassar City, South Sulawesi, Indonesia. Telp.: +6285397773536.
E-mail: eca121295@gmail.com.

ABSTRACT: Inflammation contributes to pulmonary hypertension associated with congenital heart disease. This study evaluated changes in PLR and NLR following six weeks of exercise training in adults with this condition. This single-arm pre–post interventional study enrolled 13 adults with pulmonary hypertension associated with congenital heart disease at Dr. Wahidin Sudirohusodo General Hospital, Makassar, Indonesia. Participants completed supervised low-intensity exercise training three times weekly for 30 minutes over six weeks. PLR and NLR were measured at baseline and after the intervention. Paired differences were assessed using the Shapiro–Wilk test and compared using two-sided paired t-tests. Mean PLR decreased from 139.75 ± 47.45 at baseline to 122.60 ± 35.59 after training, with a mean paired change of −17.16 ± 43.13 (95% CI, −43.22 to 8.91; p = 0.177). Mean NLR changed from 2.11 ± 0.83 to 2.09 ± 0.99, with a mean paired change of −0.03 ± 1.10 (95% CI, −0.69 to 0.64; p = 0.933). Although PLR decreased numerically, neither outcome showed a statistically significant pre–post change. Six weeks of low-intensity exercise training was not associated with statistically significant changes in PLR or NLR in adults with pulmonary hypertension associated with congenital heart disease.

1. INTRODUCTION

Pulmonary hypertension (PH) is a progressive vascular disorder characterized by elevated pulmonary vascular resistance and pulmonary arterial pressure, with inflammation contributing to vascular remodeling and right ventricular dysfunction [1]. In adults with congenital heart disease (CHD), persistent systemic-to-pulmonary shunting and increased pulmonary blood flow can promote PH development and substantially worsen morbidity and survival [2]. Although PH is reported in a relevant proportion of patients with CHD, evidence from Indonesia remains limited, underscoring the need for population-specific studies [3].

Inflammatory and hematologic alterations are implicated in PH pathobiology, including endothelial dysfunction, leukocyte activation, and platelet-mediated vascular responses [4]. The platelet-to-lymphocyte ratio (PLR) and neutrophil-to-lymphocyte ratio (NLR), derived from routine complete blood counts, are accessible biomarkers that may reflect systemic inflammation and prognostic risk in PH [5]. Exercise-based rehabilitation is considered safe and beneficial for appropriately selected, medically stable patients with PH, improving exercise capacity and quality of life [6].

Exercise may also attenuate inflammation through molecular and cellular mechanisms, including improved endothelial function, reduced oxidative stress, and anti-inflammatory myokine signaling [7]. Acute exercise has been associated with changes in immune-cell subsets and inflammatory cytokines in pulmonary arterial hypertension, but its effects on PLR and NLR after structured training remain unclear [8]. Therefore, this study evaluated changes in PLR and NLR before and after a six-week exercise-training program in adults with PH associated with CHD.

2. MATERIALS AND METHODS

2.1 Study Design, Setting, and Participants

This single-arm pre–post interventional study was conducted at the Integrated Heart Center, Dr. Wahidin Sudirohusodo General Hospital, Makassar, Indonesia, from June to December 2025. PLR and NLR were assessed at baseline and after a six-week exercise-training program. Adults aged ≥18 years with PH associated with CHD were recruited from the outpatient clinic. PH was confirmed by right-heart catheterization, defined as mean pulmonary arterial pressure ≥25 mmHg, pulmonary artery wedge pressure ≤15 mmHg, and pulmonary vascular resistance >3 Wood units. CHD was confirmed by echocardiography and/or right-heart catheterization. The minimum sample size was calculated using the formula for paired means, $n = [(Z\alpha/2 + Z\beta) \times \sigma/\Delta]^2$, with $Z\alpha/2 = 1.96$ (two-sided $\alpha = 0.05$), $Z\beta = 0.67$ (power of approximately 75%), an assumed standard deviation of the paired difference (σ) of 70, and an expected mean difference (Δ) of 52.7; this yielded 12.2, which was rounded up to 13 participants. Eligible patients were enrolled until the required sample size was reached.

Patients were excluded if they had left ventricular disease, severe lung disease, recent acute coronary syndrome, recent percutaneous coronary intervention or coronary artery bypass grafting, intracardiac thrombus or recent embolism, resting heart rate >90 beats/min, exercise-induced arrhythmia or conduction disturbance, acute myocarditis or pericarditis, hypertrophic cardiomyopathy, hemoglobin <11.0 g/dL, severe musculoskeletal or neurological disability, thrombophlebitis, uncontrolled major non-cardiac illness, active malignancy, aortic aneurysm, severe psychiatric disorder, or inability or unwillingness to participate. Participants were considered dropouts if adherence was <80% or if they did not undergo the post-intervention assessment.

2.2 Baseline Assessment

Demographic data, medical history, medication history, educational level, occupation, and income were obtained from medical records and participant interviews. Body weight, height, body mass index, resting heart rate, and blood pressure were measured at baseline. Left ventricular diastolic function was assessed by echocardiography using a GE Vivid E95 system. All participants were then referred to the cardiorespiratory subdivision of the Department of Physical Medicine and Rehabilitation, where baseline blood samples for PLR and NLR were obtained.

2.3 Exercise-Training Program

All participants received standardized cardiac-rehabilitation education addressing medication adherence, stress management, smoking cessation, healthy diet, risk-factor control, and exercise. Participants received a 1-kg dumbbell, incentive spirometer, digital blood-pressure monitor, pulse oximeter, exercise logbook, and video-based cardiac-exercise materials. The program consisted of supervised low-intensity exercise three times weekly for 30 minutes per session over six weeks (18 sessions in total). Each session combined low-intensity cardiac exercise following the video-based materials, light resistance exercise using the 1-kg dumbbell, and breathing exercise with the incentive spirometer. Low intensity was defined according to the Indonesian Heart Association (PERKI) classification as a rating of perceived exertion of 10–11 on the 6–20 Borg scale, corresponding to 20–39% of heart-rate reserve. Sessions were delivered by the cardiac-rehabilitation team at the Integrated Heart Center. To ensure consistency, all participants received the same standardized education, equipment, and exercise prescription. For safety, participants used the provided digital blood-pressure monitor and pulse oximeter to monitor blood pressure, heart rate, and

peripheral oxygen saturation during training. Exercise completion was recorded in the logbook to assess adherence, calculated as the percentage of the 18 prescribed sessions that were completed.

2.4 Outcome Measures

The primary outcomes were PLR and NLR, calculated from complete blood counts obtained before and after the intervention. PLR was calculated by dividing the absolute platelet count by the absolute lymphocyte count, whereas NLR was calculated by dividing the absolute neutrophil count by the absolute lymphocyte count. Post-intervention blood sampling was performed at least 48 hours after the final exercise session, following adequate rest.

2.5 Statistical Analysis

Data were analyzed using SPSS version 27. The normality of paired differences was assessed using the Shapiro–Wilk test. Baseline and post-intervention PLR and NLR were compared using two-sided paired t-tests. Results are presented as mean ± standard deviation, mean paired difference, 95% confidence interval, and p value. Statistical significance was set at $p < 0.05$. Analyses included all participants who completed both assessments (complete-case analysis). Because the study had a single-arm design, the paired comparison estimates within-participant change over time and cannot distinguish an intervention effect from regression to the mean, biological variability, or other time-dependent factors. The 95% CI of the mean paired difference was therefore emphasized as a measure of the precision of each estimate, and nonsignificant results were interpreted as inconclusive rather than as evidence of no effect. As an exploratory analysis, absolute neutrophil, lymphocyte, and platelet counts were also compared between baseline and six weeks using paired t-tests; these analyses were not adjusted for multiple comparisons and were interpreted descriptively.

3. RESULTS AND DISCUSSION

3.1 Participant Characteristics

Thirteen adults with PH associated with CHD completed the six-week exercise-training program and underwent both baseline and post-intervention measurements. The mean participant age was 32.54 ± 8.21 years, and most participants were women (10/13, 76.9%). ASD was the predominant underlying CHD diagnosis, affecting 10 participants (76.9%), while VSD and PDA were present in two (15.4%) and one participant (7.7%), respectively. The cohort had a mean body mass index of 19.97 ± 2.73 kg/m² and a mean baseline peripheral oxygen saturation of 94.38 ± 3.78% (Table 1). Because participants with adherence below 80% were classified as dropouts, all analyzed participants completed at least 80% of the prescribed sessions.

Table 1. Baseline Characteristics of Participants

Characteristic	Value (n = 13)
Age, years	32.54 ± 8.21
Female sex, n (%)	10 (76.9)
Body mass index, kg/m²	19.97 ± 2.73
Peripheral oxygen saturation, %	94.38 ± 3.78
Underlying CHD diagnosis, n (%)	
ASD	10 (76.9)
VSD	2 (15.4)
PDA	1 (7.7)

Data are presented as mean ± standard deviation or n (%).

3.2 Changes in PLR and NLR

The distributions of paired pre–post differences did not significantly deviate from normality for PLR (Shapiro–Wilk $p = 0.935$) or NLR (Shapiro–Wilk $p = 0.325$); therefore, paired t-tests were used for the primary analyses. PLR decreased from 139.75 ± 47.45 at baseline to 122.60 ± 35.59 after six weeks of exercise training. The mean paired change was −17.16 ± 43.13,

corresponding to a 95% CI of −43.22 to 8.91. Although the direction of change indicated a reduction in PLR, the confidence interval included the null value and the difference was not statistically significant (p = 0.177).

Similarly, NLR changed minimally, from 2.11 ± 0.83 at baseline to 2.09 ± 0.99 after the intervention. The mean paired change was −0.03 ± 1.10 (95% CI, −0.69 to 0.64), with no statistically significant difference between measurements (p = 0.933). Collectively, these findings indicate that six weeks of low-intensity exercise training was not associated with statistically significant pre–post changes in either inflammatory ratio in this sample (Table 2). Individual trajectories for PLR and NLR are presented in Figure 1.

Table 2. Changes in PLR and NLR After Six Weeks of Exercise Training

Outcome	Baseline	Six weeks	Mean paired change*	95% CI	*p* value
PLR	139.75 ± 47.45	122.60 ± 35.59	−17.16 ± 43.13	−43.22 to 8.91	0.177
NLR	2.11 ± 0.83	2.09 ± 0.99	−0.03 ± 1.10	−0.69 to 0.64	0.933

Data are presented as mean ± standard deviation. Values were compared using two-sided paired t *tests. Change was calculated as the six-week value minus the baseline value.*

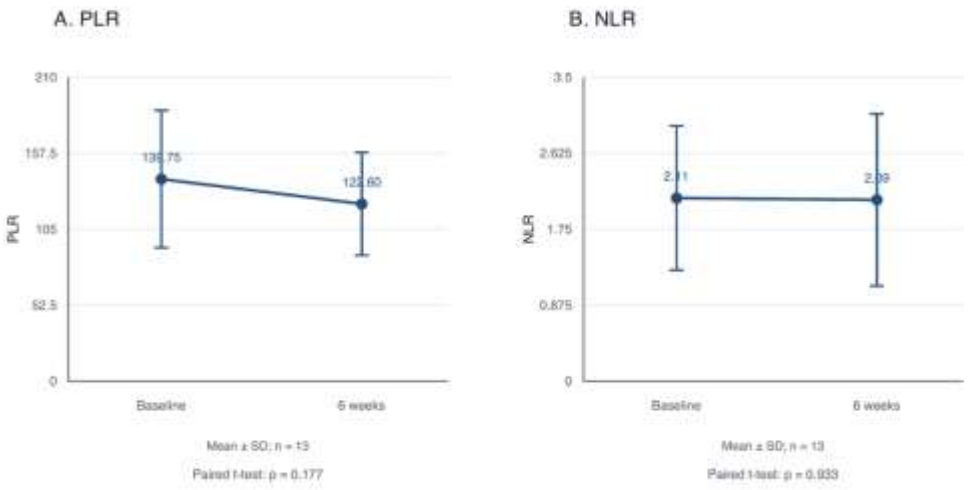


Figure 1. Individual Changes in PLR and NLR From Baseline to Six Weeks of Exercise Training. Panel A shows PLR, and panel B shows NLR. Each line represents one participant and connects the baseline and six-week measurements; the superimposed bold line indicates the group mean. Neither outcome showed a statistically significant change on two-sided paired *t*-testing.

3.3 Exploratory Changes in Hematologic Components

To aid interpretation of the ratio-based outcomes, absolute neutrophil, lymphocyte, and platelet counts were compared before and after training. Mean neutrophil and lymphocyte counts increased descriptively, whereas the mean platelet count decreased slightly. However, none of these changes was statistically significant: neutrophils increased from 3.81 ± 1.22 to 4.14 ± 1.65 × 10^9/L (p = 0.368), lymphocytes increased from 1.90 ± 0.50 to 2.06 ± 0.50 × 10^9/L (p = 0.298), and platelets decreased from 247.92 ± 64.55 to 241.46 ± 50.12 × 10^9/L (p = 0.547). These exploratory findings provide no evidence of a statistically significant change in the blood-cell components contributing to PLR or NLR (Table 3).

Table 3. Exploratory Changes in Absolute Blood-Cell Counts After Six Weeks of Exercise Training

Variable, ×10^9/L	Baseline	Six weeks	*p* value
Absolute neutrophil count	3.81 ± 1.22	4.14 ± 1.65	0.368

Absolute lymphocyte count	1.90 ± 0.50	2.06 ± 0.50	0.298
Platelet count	247.92 ± 64.55	241.46 ± 50.12	0.547

Data are presented as mean ± standard deviation. V alues were compared using two-sided paired t *tests. This was an exploratory analysis.*

3.4 Discussion

The present study found no statistically significant pre–post change in PLR or NLR following six weeks of low-intensity exercise training in adults with PH associated with CHD. The directionally lower PLR should not be interpreted as evidence of reduced systemic inflammation because neither the ratio nor its underlying hematologic components changed significantly. Likewise, the essentially unchanged NLR indicates that this intervention was not associated with a measurable shift in the circulating neutrophil-to-lymphocyte balance. Importantly, a nonsignificant result does not demonstrate the absence of an effect. The 95% CI for the change in PLR (−43.22 to 8.91) was wide and compatible with both a substantial reduction and a small increase, whereas the 95% CI for NLR (−0.69 to 0.64) was centered close to zero. Thus, within the limits of this small sample, the data are inconclusive regarding an effect of short-term exercise training on PLR and suggest little change in NLR.

The demographic profile of this cohort is clinically plausible. ASD predominated, consistent with its often-delayed clinical presentation and the gradual development of pulmonary vascular disease after prolonged left-to-right shunting [9,10]. The predominance of women is also consistent with previous Indonesian data in adults with CHD-associated PH [11]. Although female predominance in PH has been linked to the complex and context-dependent effects of estrogen signaling on pulmonary vascular and right ventricular biology, the underlying mechanisms remain incompletely understood [12,13].

Direct evidence on exercise-induced changes in PLR and NLR among adults with PH associated with CHD remains sparse. The present findings differ from studies in other clinical populations, in which longer or more intensive exercise interventions have produced inconsistent ratio-based inflammatory responses. Nazari et al. (2023) reported a reduction in PLR after 16 weeks of combined aerobic and resistance training in children with type 1 diabetes, whereas Joisten et al. (2021) observed reduced NLR but unchanged PLR after high-intensity interval training in multiple sclerosis [14,15]. Similarly, Wang et al. (2011) found a reduction in NLR after a diet-and-exercise intervention in overweight adolescents [16]. Differences in disease mechanisms, training modality, intervention duration, and inflammatory phenotype may explain why these observations cannot be directly extrapolated to PH associated with CHD.

The current findings are broadly in line with Grünig et al. (2012)[17], who demonstrated improved exercise capacity, functional status, and quality of life after structured exercise training in PH without a significant change in C-reactive protein. This pattern suggests that functional benefits of exercise may occur independently of detectable changes in conventional systemic inflammatory biomarkers. However, exercise may still influence inflammatory pathways not captured by PLR and NLR. In idiopathic pulmonary arterial hypertension, acute cycling exercise was associated with reductions in Th17 lymphocytes and pro-inflammatory cytokines without aggravating systemic inflammation [8]. Experimental studies similarly suggest that aerobic training may attenuate pulmonary vascular remodeling and inflammatory signaling through effects on cytokine balance, oxidative stress, and right ventricular adaptation [18,19].

Biologically, regular exercise may exert anti-inflammatory actions through myokine-mediated signaling, including muscle-derived IL-6, subsequent induction of IL-10 and IL-1Ra, and suppression of TNF-α activity [20,21]. Exercise may also reduce platelet reactivity through improved nitric oxide bioavailability and redox balance, thereby affecting platelet activation without necessarily altering peripheral platelet counts or PLR [22,23]. Therefore, the absence of significant changes in PLR and NLR does not exclude biologically relevant anti-inflammatory or vascular effects of exercise. Rather, it indicates that these ratios may not be sufficiently responsive markers of short-term exercise-related immunomodulation in adults with PH associated with CHD.

This study used a single-arm pre–post design without a control group; therefore, changes over time cannot be attributed solely to the exercise intervention. Regression to the mean, biological variability, and changes in concurrent therapy over the study period cannot be excluded. The small sample size (n = 13) limited statistical power, and the study may have been underpowered to detect modest but clinically relevant changes in PLR and NLR, as reflected by the wide confidence intervals. The single-center setting and the predominance of women and of ASD may further limit generalizability. Furthermore, detailed

hemodynamic parameters, WHO functional class, and PH-specific therapy were not systematically reported, which limits characterization of PH severity in this cohort. In addition, these ratios are non-specific inflammatory biomarkers and may be influenced by concurrent clinical conditions, medication use, intercurrent infection, hydration status, and biological variation. Measurements were obtained only at baseline and after six weeks, precluding assessment of temporal trajectories or the effects of different training durations. Finally, CRP, cytokines, and other molecular biomarkers were not measured; thus, potential exercise-related inflammatory mechanisms could not be comprehensively evaluated.

4. CONCLUSION

This single-arm pre–post study found that six weeks of low-intensity exercise training was not associated with statistically significant changes in PLR or NLR among adults with PH associated with CHD. PLR showed a numerical decrease (mean paired change, −17.16) and NLR remained essentially unchanged (mean paired change, −0.03); however, neither change was statistically significant. These numerical differences should therefore not be interpreted as evidence of an intervention effect or of improved systemic inflammation. Taken together, the findings indicate that a six-week exercise program may be insufficient to produce consistent changes in PLR and NLR in this population, despite the established potential of exercise to improve functional and vascular outcomes in PH. Future controlled studies with larger samples, longer training periods, repeated biomarker assessments, and complementary inflammatory markers are needed to clarify the anti-inflammatory effects of exercise in PH associated with CHD.

ACKNOWLEDGEMENTS

The authors thank the participants and the cardiac-rehabilitation team at the Integrated Heart Center, Dr. Wahidin Sudirohusodo General Hospital, Makassar, Indonesia.

AUTHOR CONTRIBUTIONS

Conceptualization: M.R.R.S., Y.P., Z.D., A.F.M., M.W., and A.A.Z. Methodology: M.R.R.S., Y.P., Z.D., M.W., and A.A.Z. Formal analysis: M.R.R.S. and A.A.Z. Investigation: M.R.R.S., Y.P., Z.D., A.F.M., and M.W. Data curation: M.R.R.S. Writing—original draft: M.R.R.S. Writing—review and editing: M.R.R.S., Y.P., Z.D., A.F.M., M.W., and A.A.Z. Supervision: Y.P., Z.D., A.F.M., M.W., and A.A.Z. All authors approved the final manuscript.

CONFLICT OF INTEREST

The authors declare that they have no competing interests.

ETHICS STATEMENT

The study protocol was approved by the Health Research Ethics Committee, Faculty of Medicine, Hasanuddin University, Indonesia (approval no. 957/UN4.6.4.5.31/PP36/2026; protocol no. UH26060731; approved 12 August 2026). Written informed consent was obtained from all participants before enrollment, and participant confidentiality was maintained throughout the study.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to applicable ethical and privacy requirements.

REFERENCES

[1] Zhao H, Song J, Li X, et al. The role of immune cells and inflammation in pulmonary hypertension: mechanisms and implications. Front Immunol. 2024;15:1374506. doi:10.3389/FIMMU.2024.1374506/XML

[2] Kaemmerer H, Gorenflo M, Huscher D, et al. Pulmonary Hypertension in Adults with Congenital Heart Disease: Real-World Data from the International COMPERA-CHD Registry. J Clin Med. 2020;9(5):1456. doi:10.3390/JCM9051456

[3] Jone PN, Ivy DD, Hauck A, et al. Pulmonary Hypertension in Congenital Heart Disease: A Scientific Statement From the American Heart Association. Circ Heart Fail. 2023;16(7). doi:10.1161/HHF.0000000000000080

[4] Gursoy M, Salihoglu E, Hatemi AC, Hokenek AF, Ozkan S, Ceyran H. Inflammation and congenital heart disease associated pulmonary hypertension. Heart Surg Forum. 2015;18(1):038. doi:10.1532/HSF.1228

[5] Shahmoradi Z, Malekmohammad M, Najafi G, et al. Prognostic Value of Platelet to Lymphocyte Ratio (PLR) and Neutrophil to Lymphocyte Ratio (NLR) in Patients with Pulmonary Hypertension. Tanaffos. 2022;21(4):480-486. Accessed October 16, 2026. https://pubmed.ncbi.nlm.nih.gov/37583772/
[6] Wojciuk M, Dzięcioł-Anikiej Z, Kaniewska K, Ciołkiewicz M, Moskal-Jasińska D, Kuryliszyn-Moskal A. Exercise Training as a Non-Pharmacological Therapy for Patients with Pulmonary Arterial Hypertension: Home-Based Rehabilitation Program and Training Recommendations. J Clin Med. 2022;11(23). doi:10.3390/JCM11236932
[7] Poojary G, Morris N, Joshi MB, Babu AS. Role of Exercise in Pulmonary Hypertension: Evidence from Bench to Bedside. Pulse (Basel). 2024;12(1):66-75. doi:10.1159/000539537
[8] Waller L, Krüger K, Conrad K, Weiss A, Alack K. Effects of Different Types of Exercise Training on Pulmonary Arterial Hypertension: A Systematic Review. J Clin Med. 2020;9(6):1689. doi:10.3390/JCM9061689
[9] Toyono M. Pulmonary arterial hypertension in adults with atrial septal defect. J Cardiol Cases. 2012;6(1):e32. doi:10.1016/J.JCCASE.2012.04.005
[10] Kuijpers JM, Mulder BJM, Bouma BJ. Secundum atrial septal defect in adults: a practical review and recent developments. Neth Heart J. 2015;23(4):205-211. doi:10.1007/S12471-015-0663-Z
[11] Tjan W, Purwandini D, Purnamasari CB. Pulmonary Hypertension in Patient with Acyanotic Congenital Heart Defects in Abdul Wahab Sjahranie General Hospital, Samarinda from 2015-2016. Cardiovascular and Cardiometabolic Journal (CCJ). 2020;1(2):45-49. doi:10.20473/CCJ.V1I2.2020.45-49
[12] Umar S, Rabinovitch M, Eghbali M. Estrogen paradox in pulmonary hypertension: current controversies and future perspectives. Am J Respir Crit Care Med. 2012;186(2):125-131. doi:10.1164/RCCM.201201-0058PP
[13] Rodriguez-Arias JJ, García-Álvarez A. Sex Differences in Pulmonary Hypertension. Frontiers in Aging. 2021;2:727558. doi:10.3389/FRAGI.2021.727558/FULL
[14] Nazari M, Shabani R, Dalili S. Effect of Concurrent Resistance-Aerobic Training on Neutrophil-Lymphocyte Ratio and Platelet-Lymphocyte Ratio in Pediatric Type 1 Diabetes: A Randomized Trial. Iranian Journal of Diabetes and Obesity. 2023;15(1):51-58. doi:10.18502/IJDO.V15I1.12211
[15] Joisten N, Proschinger S, Rademacher A, et al. High-intensity interval training reduces neutrophil-to-lymphocyte ratio in persons with multiple sclerosis during inpatient rehabilitation. Mult Scler. 2021;27(7):1136-1139. doi:10.1177/1352458520951382
[16] Wang R, Chen PJ, Chen WH. Diet and exercise improve neutrophil to lymphocyte ratio in overweight adolescents. Int J Sports Med. 2011;32(12):982-986. doi:10.1055/S-0031-1283185
[17] Grünig E, Lichtblau M, Ehlken N, et al. Safety and efficacy of exercise training in various forms of pulmonary hypertension. Eur Respir J. 2012;40(1):84-92. doi:10.1183/09031936.00123711
[18] Moreira-Gonçalves D, Ferreira R, Fonseca H, et al. Cardioprotective effects of early and late aerobic exercise training in experimental pulmonary arterial hypertension. Basic Res Cardiol. 2015;110(6). doi:10.1007/S00395-015-0514-5
[19] Nogueira-Ferreira R, Moreira-Gonçalves D, Silva AF, et al. Exercise preconditioning prevents MCT-induced right ventricle remodeling through the regulation of TNF superfamily cytokines. Int J Cardiol. 2016;203:858-866. doi:10.1016/j.ijcard.2015.11.066
[20] Nara H, Watanabe R. Anti-Inflammatory Effect of Muscle-Derived Interleukin-6 and Its Involvement in Lipid Metabolism. Int J Mol Sci. 2021;22(18). doi:10.3390/IJMS22189889
[21] Małkowska P, Sawczuk M. Cytokines as Biomarkers for Evaluating Physical Exercise in Trained and Non-Trained Individuals: A Narrative Review. Int J Mol Sci. 2023;24(13). doi:10.3390/IJMS241311156
[22] Barale C, Melchionda E, Tempesta G, Morotti A, Russo I. Impact of Physical Exercise on Platelets: Focus on Its Effects in Metabolic Chronic Diseases. Antioxidants. 2023;12(8):1609. doi:10.3390/ANTIOX12081609
[23] Russo I, Barale C, Melchionda E, Penna C, Pagliaro P. Platelets and Cardioprotection: The Role of Nitric Oxide and Carbon Oxide. Int J Mol Sci. 2023;24(7). doi:10.3390/IJMS24076107