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ORIGINAL ARTICLE

Effect of Acute Physical Stress on Serum Troponin-I, Inflammatory Cytokines, and Cardiovascular Parameters in Adults with Subclinical Anemia. A Clinical Investigation

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ABSTRACT

Background: Subclinical anemia is a borderline hematological condition often overlooked due to the absence of overt clinical symptoms. However, it may predispose individuals to exaggerated cardiovascular and inflammatory responses when subjected to physical exertion. Understanding this physiological vulnerability is crucial, especially in physically active or at-risk populations.

Objective: To evaluate the impact of acute physical stress on serum Troponin-I, inflammatory cytokines (IL-6, TNF-α, CRP), and cardiovascular parameters (heart rate, blood pressure, ECG changes) in adults with subclinical anemia compared to healthy controls.

Methods: This prospective clinical study included 100 participants aged 18–40 years, recruited from the Cardiology Departments of PNS Shifa Hospital, Karachi, and Pakistan Railway Hospital, Rawalpindi. Fifty participants with subclinical anemia and fifty age- and sex-matched controls underwent a standardized treadmill stress test using the Bruce protocol. Serum samples were collected pre- and one hour post-exercise to assess Troponin-I and cytokine levels. Cardiovascular parameters and ECG changes were monitored throughout.

Results: Post-exercise Troponin-I levels were significantly higher in the subclinical anemia group (0.064 ± 0.020 ng/mL) compared to controls (0.036 ± 0.012 ng/mL; $p < 0.001$). Inflammatory markers, including IL-6, TNF-α, and CRP, were also markedly elevated in the anemic group ($p < 0.05$). Cardiovascular responses such as peak heart rate and systolic blood pressure were more pronounced in anemic individuals. Transient ECG changes suggestive of myocardial ischemia were observed in 12% of the anemic group, but none in the controls.

Conclusion: Subclinical anemia is associated with a heightened cardiovascular and inflammatory response to acute physical stress, indicating increased vulnerability to myocardial strain and systemic inflammation. Early recognition and management of subclinical anemia may be critical in physically active populations to prevent stress-related cardiac complications.

Keywords: Subclinical anemia, Troponin-I, IL-6, Physical stress, Cardiovascular response, Inflammatory cytokines, ECG changes.

INTRODUCTION

Anemia, a common hematologic condition, is defined by a reduction in red blood cell count or hemoglobin concentration, leading to diminished oxygen-carrying capacity of the blood. While overt anemia is well-documented and readily diagnosed, a large population remains affected by subclinical or borderline anemia a condition in which hemoglobin levels are mildly reduced but do not yet meet the diagnostic threshold for clinical anemia[1]. Despite the absence of prominent symptoms, subclinical anemia may still impose physiological burdens, especially when the individual is exposed to acute stressors such as physical exertion. This latent burden is often underestimated in both clinical and research settings[2].

Acute physical stress, whether through exercise, occupational exertion, or emergency physiological demands, can trigger a complex cascade of hemodynamic and biochemical responses, including sympathetic activation, increased myocardial oxygen demand, systemic inflammation, and the release of cardiac biomarkers[3]. One such biomarker is cardiac Troponin-I (cTnI), a regulatory protein involved in myocardial contractility that is widely recognized for its specificity in detecting myocardial injury. Recent studies have shown that even non-pathological physical stress in healthy individuals can cause transient, low-level elevations in cTnI, suggesting that the myocardium is sensitive to short-term hemodynamic strain[4].

Alongside cardiac markers, inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP) are also activated in response to physical stress. These mediators, part of the body's innate immune response, have been implicated in both protective and pathological cardiovascular processes[5]. IL-6, for instance, plays a role in mediating the acute phase response and may contribute to endothelial dysfunction, while CRP is a well-known marker of low-grade inflammation and cardiovascular risk. TNF-α, on the other hand, promotes catabolic activity and has been associated with myocardial remodeling. The elevation of these cytokines is typically transient in healthy individuals but may become exaggerated in those with predisposing conditions such as anemia[6].

In the context of subclinical anemia, where the oxygen delivery to tissues is subtly compromised, the effects of acute physical stress may be amplified. The heart, in an attempt to maintain perfusion and oxygenation, must increase cardiac output, which may in turn elevate myocardial workload and trigger stress-induced ischemic or inflammatory responses[7]. Although the cardiovascular system can adapt to mild hemoglobin reductions under resting conditions, during physical exertion this compensatory capacity may be exceeded, potentially leading to biomolecular indicators of myocardial strain, subtle ischemia, or even arrhythmogenic changes on electrocardiogram (ECG). However, this relationship remains poorly characterized in individuals with subclinical anemia, and few studies have explored the synergistic interaction between low-grade anemia, inflammatory stress, and myocardial function during acute physiological challenges[8].

Given the rising prevalence of sedentary lifestyles juxtaposed with intermittent intense physical activity (as seen in gym-goers, amateur athletes, or manual laborers), it is increasingly important to assess the hidden vulnerabilities of at-risk individuals, especially those with undiagnosed hematologic imbalances. In this context, subclinical anemia may serve as a “silent modifier” of

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stress response, potentially increasing susceptibility to adverse cardiovascular outcomes or long-term dysfunction[9].

Therefore, the present study was designed to investigate the effects of acute physical stress on serum Troponin-I, key inflammatory cytokines (IL-6, TNF-α, CRP), and cardiovascular parameters (heart rate, blood pressure, and ECG patterns) in adults with subclinical anemia, and to compare these responses with those in healthy, non-anemic individuals. By identifying early alterations in biochemical and physiological markers under controlled stress conditions, this study aims to bridge the existing gap in the literature regarding the cardiovascular and systemic risks posed by seemingly mild anemia, thus contributing to better screening, preventive care, and personalized recommendations in occupational, athletic, and general health settings[10, 11].

MATERIALS AND METHODS

Study Design and Setting: This prospective, comparative clinical investigation was conducted to evaluate the effect of acute physical stress on serum Troponin-I, inflammatory cytokines, and cardiovascular parameters in individuals with subclinical anemia. The study was carried out at two tertiary care centers in Pakistan: the Cardiology Department and Cardiac Unit of PNS Shifa Hospital, Karachi, and the Department of Cardiology at Pakistan Railway Hospital, Rawalpindi. Both institutions provided standardized clinical environments, diagnostic facilities, and trained personnel for cardiac evaluations and exercise stress testing.

Study Duration and Sample Size: The study was conducted over a period of 14 months, from January 2022 to February 2023. A total of 100 participants were enrolled and divided into two equal groups. Group A comprised 50 individuals diagnosed with subclinical anemia based on laboratory criteria, while Group B included 50 healthy, age- and sex-matched individuals with normal hemoglobin levels who served as the control group.

Inclusion and Exclusion Criteria: Participants included in the study were adults aged 18 to 40 years. Individuals in the subclinical anemia group were defined by hemoglobin levels of 10.5–12.5 g/dL for females and 11.5–13.5 g/dL for males. Exclusion criteria included clinically diagnosed anemia below the defined thresholds, history of cardiovascular disease, hypertension, diabetes mellitus, chronic inflammatory or autoimmune conditions, current infections, or use of any medications affecting the cardiovascular or immune systems. Smokers, individuals with obesity (BMI >30 kg/m²), or those with contraindications to exercise testing were also excluded.

Ethical Considerations: Ethical approval for the study was obtained from the Institutional Review Boards of both participating hospitals. All participants were informed about the objectives and procedures of the study, and written informed consent was obtained prior to inclusion.

Baseline Clinical Assessment: Prior to initiating the stress protocol, all participants underwent a comprehensive clinical examination including assessment of medical history, vital signs, anthropometric measurements (height, weight, and BMI), and a baseline 12-lead electrocardiogram (ECG). Complete blood count (CBC) was conducted to confirm hemoglobin levels and classify participants accurately into their respective study groups.

Exercise Stress Testing Protocol: All participants underwent a standardized graded treadmill exercise test using the Bruce protocol. This test involved progressively increasing stages of speed and incline on the treadmill, with continuous monitoring of the heart rate and rhythm. The test was terminated when participants reached 85% of their predicted maximum heart rate, or earlier if they experienced symptoms such as chest discomfort, dyspnea, dizziness, or developed significant ECG abnormalities. The exercise was supervised by a cardiologist to ensure participant safety.

Blood Sampling and Biomarker Analysis: Venous blood samples were collected at two time points: one prior to the initiation of exercise (baseline) and the second one hour after the completion of the exercise test. Blood samples were processed and analyzed for cardiac and inflammatory biomarkers. The primary cardiac biomarker measured was serum Troponin-I, while the inflammatory biomarkers included interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP). All biomarker estimations were conducted using enzyme-linked immunosorbent assay (ELISA) kits in the central laboratories of the respective hospitals, following strict quality control protocols.

Cardiovascular Monitoring and Data Collection: Throughout the stress testing, cardiovascular parameters including heart rate and blood pressure were monitored continuously. Blood pressure measurements were recorded at rest, at peak exercise, and during the recovery period. Continuous ECG telemetry was used to monitor for ischemic changes, arrhythmias, or other abnormalities. Particular attention was paid to any ST-segment deviations or T-wave changes that could suggest stress-induced myocardial ischemia.

Statistical Analysis: All collected data were entered and analyzed using Statistical Package for the Social Sciences (SPSS) version 26.0. Continuous variables were expressed as mean ± standard deviation. Paired t-tests were used to compare pre- and post-exercise biomarker levels within each group, while independent t-tests were applied to compare outcomes between the subclinical anemia group and healthy controls. A p-value of less than 0.05 was considered statistically significant for all comparisons.

RESULTS

Baseline Characteristics of Study Population: A total of 100 participants were included in the final analysis, with 50 individuals categorized into Group A (subclinical anemia) and 50 individuals into Group B (healthy controls). The groups were well-matched for age, sex distribution, and BMI, ensuring comparability. The mean age in Group A was 28.8 ± 4.3 years, while in Group B it was 28.2 ± 4.7 years, with no statistically significant difference observed ($p = 0.56$). Gender distribution was nearly equal between the two groups, with 52% males and 48% females in Group A, and 50% males and 50% females in Group B ($p = 0.84$). The mean body mass index (BMI) was also comparable between the groups (23.4 ± 2.1 kg/m² in Group A vs. 23.1 ± 1.9 kg/m² in Group B; $p = 0.47$). However, the mean hemoglobin level differed significantly, as expected by design, with Group A showing lower mean hemoglobin (11.7 ± 0.5 g/dL) compared to Group B (14.2 ± 0.6 g/dL), and this difference was statistically significant ($p < 0.001$). These findings confirmed that the subclinical anemia group was appropriately identified and free from confounding demographic variables.

Table 1: Baseline Demographics and Hematological Parameters

Parameter	Group A (Subclinical Anemia)	Group B (Control)	p-value
Mean Age (years)	28.8 ± 4.3	28.2 ± 4.7	0.56
Male (%)	52%	50%	0.84
Female (%)	48%	50%	
BMI (kg/m²)	23.4 ± 2.1	23.1 ± 1.9	0.47
Hemoglobin (g/dL)	11.7 ± 0.5	14.2 ± 0.6	<0.001**

Serum Troponin-I Response to Acute Physical Stress: Baseline Troponin-I levels were within the normal reference range in both groups, with no significant difference at rest. In Group A, the baseline Troponin-I level was 0.019 ± 0.008 ng/mL, while in Group B it was 0.017 ± 0.006 ng/mL ($p = 0.23$). However, one hour after undergoing the treadmill stress test, Troponin-I levels rose significantly in both groups, with a substantially greater increase in the subclinical anemia group. Post-exercise levels in Group A reached 0.064 ± 0.020 ng/mL compared to 0.036 ± 0.012 ng/mL in Group B ($p < 0.001$). This marked elevation in Group A reflects subclinical myocardial strain that is more pronounced in the presence of borderline anemia. The difference between pre- and post-exercise values was also statistically significant within each group ($p < 0.01$), indicating a true physiological response rather than random fluctuation. These findings suggest that Troponin-I is

a sensitive biomarker for detecting stress-induced myocardial changes, and that individuals with subclinical anemia may have a reduced threshold for ischemic stress.

Table 2: Serum Troponin-I Levels (ng/mL) Before and After Exercise

Timepoint	Group A (Subclinical Anemia)	Group B (Control)	p-value
Baseline	0.019 ± 0.008	0.017 ± 0.006	0.23
1 Hour Post-Exercise	0.064 ± 0.020	0.036 ± 0.012	<0.001**

Post-Exercise Inflammatory Cytokine Response: Inflammatory cytokines demonstrated a significantly exaggerated response in participants with subclinical anemia. The mean post-exercise IL-6 level in Group A was 12.4 ± 2.6 pg/mL, while in Group B it was 8.2 ± 1.7 pg/mL ($p < 0.01$), indicating a hyper-inflammatory state following exertion. Similarly, CRP levels increased to 3.2 ± 0.8 mg/L in Group A compared to 1.9 ± 0.6 mg/L in Group B ($p < 0.01$), again pointing toward enhanced systemic inflammation in anemic individuals. TNF-α levels, while elevated in both groups, were significantly higher in Group A (7.5 ± 1.4 pg/mL) than in Group B (6.1 ± 1.2 pg/mL; $p = 0.04$). These elevations reflect a pro-inflammatory response that is disproportionate in individuals with subclinical anemia when exposed to acute physiological stress. The amplified release of cytokines such as IL-6 and TNF-α suggests an exaggerated immune response that could contribute to endothelial dysfunction and cardiovascular risk if such stressors are sustained or repeated.

Table 3: Post-Exercise Inflammatory Cytokine Levels

Biomarker	Group A (Subclinical Anemia)	Group B (Control)	p-value
IL-6 (pg/mL)	12.4 ± 2.6	8.2 ± 1.7	<0.01**
TNF-α (pg/mL)	7.5 ± 1.4	6.1 ± 1.2	0.04*
C-Reactive Protein (mg/L)	3.2 ± 0.8	1.9 ± 0.6	<0.01**

Cardiovascular Parameters and Electrocardiographic Changes: Cardiovascular parameters recorded during and after the exercise test showed exaggerated responses in Group A compared to Group B. The peak heart rate achieved by participants in the subclinical anemia group was significantly higher (172 ± 9 bpm) compared to controls (161 ± 7 bpm; $p < 0.001$). Systolic blood pressure rose more sharply in anemic individuals, with an average increase of 24.3 ± 5.2 mmHg in Group A versus 17.1 ± 4.7 mmHg in Group B ($p = 0.01$). This increased cardiac workload may reflect the compensatory mechanisms attempting to overcome the reduced oxygen-carrying capacity in anemic patients. Importantly, 12% (6 out of 50) of participants in the subclinical anemia group developed transient electrocardiographic abnormalities, including ST-segment depression and T-wave inversion, which were suggestive of subclinical myocardial ischemia. No such changes were observed in the control group (0%; $p = 0.02$). These findings emphasize that even mild reductions in hemoglobin levels can significantly alter cardiovascular response to stress, placing individuals at potential risk of cardiac events during exertion.

Table 4: Cardiovascular Parameters and ECG Changes During Stress Test

Parameter	Group A (Subclinical Anemia)	Group B (Control)	p-value
Peak Heart Rate (bpm)	172 ± 9	161 ± 7	<0.001**
Increase in SBP (mmHg)	+24.3 ± 5.2	+17.1 ± 4.7	0.01*
Participants with ECG Changes (%)	12% (6/50)	0% (0/50)	0.02*

Overall Interpretation of Findings: The overall results of this clinical investigation demonstrate a consistent and statistically significant pattern across multiple domains. Individuals with subclinical anemia showed elevated levels of Troponin-I, heightened inflammatory cytokine activity, and exaggerated cardiovascular responses following acute physical stress. The presence of transient ECG abnormalities in a subset of the subclinical anemia group further strengthens the clinical relevance of these findings. These results suggest that even in the absence of overt anemia, individuals with borderline low hemoglobin levels are at a physiologically disadvantaged position when exposed to acute exertional stress, likely due to the compromised oxygen delivery, increased cardiac workload, and activated inflammatory pathways.

These findings have important implications for screening and managing individuals who may be engaging in physical activities, such as athletes, manual laborers, or military personnel, who are unknowingly affected by subclinical anemia. Preventive strategies including early identification, nutritional optimization, and tailored exercise programs may be warranted to mitigate cardiovascular risks in this overlooked population.

DISCUSSION

This clinical investigation provides novel insights into the physiological and biochemical responses to acute physical stress in adults with subclinical anemia. The key findings of the study demonstrate that individuals with borderline low hemoglobin levels exhibit significantly heightened cardiac and inflammatory responses compared to healthy controls, despite being clinically asymptomatic at rest. These results underline the previously underestimated cardiovascular vulnerability in individuals with subclinical anemia, especially during periods of physical exertion[12, 13].

One of the most striking observations in this study was the significant post-exercise elevation in serum Troponin-I among the subclinical anemia group. Although all values remained within the diagnostic range for non-infarction, the magnitude of the increase suggests subclinical myocardial strain. Troponin-I is well-established as a sensitive and specific biomarker for myocardial injury, and even modest elevations are now recognized to carry prognostic significance[14]. The exaggerated response in anemic individuals may be due to the limited oxygen-carrying capacity of their blood, which forces the myocardium to work harder to meet oxygen demands during exercise, potentially leading to transient ischemia or stress-induced cardiomyocyte injury[15].

In addition to cardiac biomarkers, our findings revealed a significantly amplified inflammatory cytokine response in the subclinical anemia group. IL-6 and CRP levels, in particular, rose sharply post-exercise[16]. IL-6 plays a dual role in both initiating the acute-phase inflammatory response and mediating protective metabolic adaptations; however, chronically elevated IL-6 is linked to endothelial dysfunction and atherosclerosis. CRP, as a downstream product of IL-6 signaling, has been implicated in predicting adverse cardiovascular outcomes. The elevated TNF-α levels in the anemic group further suggest activation of pro-inflammatory and catabolic pathways. Taken together, these results imply that subclinical anemia primes the immune system for a more aggressive response to stress, which may predispose individuals to inflammatory complications or vascular injury during repeated or sustained physical activity[17].

Cardiovascular parameters such as heart rate and systolic blood pressure increased more significantly in the subclinical anemia group, reflecting greater sympathetic nervous system activation and cardiovascular workload. This aligns with physiological expectations, as the heart compensates for reduced blood oxygen content by increasing output[18]. However, the fact that a subset of participants developed ECG abnormalities particularly ST-segment depressions and T-wave inversions highlights the potential for even transient exertion to unmask ischemic changes in this population. These findings are especially relevant in occupational and athletic settings where individuals are exposed to repetitive physical demands without routine hematological screening[19].

When contextualized within the existing literature, our results support and extend previous studies that linked anemia to

increased cardiovascular risk. However, unlike prior research which primarily focused on overt anemia, this study highlights the significant physiological burden of subclinical anemia a condition often dismissed in clinical practice. Moreover, the integration of biomarker analysis with ECG monitoring and hemodynamic profiling offers a multidimensional perspective on how borderline anemia can influence cardiovascular resilience[20].

From a pathophysiological standpoint, the interplay between reduced hemoglobin, increased cardiac workload, and systemic inflammation likely creates a pro-ischemic, pro-inflammatory environment. These factors may contribute not only to acute decompensation during stress but also to long-term cardiovascular remodeling and endothelial damage if left unaddressed. Hence, this study emphasizes the need for proactive identification and management of subclinical anemia, particularly in individuals who routinely engage in physical labor or athletic training[21].

Nonetheless, the study is not without limitations. The sample size, though statistically adequate, may not capture all interindividual variations, and the findings may not be generalizable to older or comorbid populations. Moreover, we did not assess long-term cardiovascular outcomes or perform imaging to detect structural cardiac changes. Future studies incorporating echocardiography, cardiac MRI, or longitudinal follow-up would be valuable to elucidate the clinical impact of repeated stress in this population[22].

CONCLUSION

This study demonstrates that adults with subclinical anemia experience significantly exaggerated cardiovascular and inflammatory responses to acute physical stress, as evidenced by elevated serum Troponin-I, increased levels of IL-6, CRP, and TNF-α, heightened heart rate and blood pressure, and a higher frequency of transient ischemic changes on ECG. These findings underscore the hidden physiological burden of subclinical anemia and its potential to compromise cardiac function during exertion.

Given the prevalence of subclinical anemia in the general population, particularly in developing countries like Pakistan, these results hold important implications for preventive cardiology, occupational health, and sports medicine. Early screening, nutritional correction, and tailored exercise programs for individuals with borderline anemia may be crucial in mitigating preventable cardiac stress and long-term complications.

In conclusion, subclinical anemia should no longer be regarded as a benign or clinically irrelevant condition. Instead, it warrants attention as a modifiable risk factor, especially when individuals are subjected to regular physical stress. Further research should focus on long-term outcomes and interventional strategies to protect this vulnerable population.

Availability of Data and Materials: The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Competing Interests: The authors declare that they have no competing interests.

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Authors' Contributions: JA and MTHK conceptualized and designed the study. AI and MM were involved in data collection and laboratory analysis. KP contributed to statistical analysis and interpretation of results. AIJ supervised the study and critically revised the manuscript. All authors reviewed and approved the final draft.

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REFERENCES

1. Sarocchi M, Grossi F, Arboscello E, Bellodi A, Genova C, Dal Bello MG, et al. Serial troponin for early detection of nivolumab cardiotoxicity in advanced non-small cell lung cancer patients. The oncologist. 2018;23(8):936-42.
2. Lee EC, Fragala MS, Kavouras SA, Queen RM, Pryor JL, Casa DJ. Biomarkers in sports and exercise: tracking health, performance, and recovery in athletes. The Journal of Strength & Conditioning Research. 2017;31(10):2920-37.
3. Akwe J, Halford B, Kim E, Miller A. A review of cardiac and non-cardiac causes of troponin elevation and clinical relevance part II: non cardiac causes. J Cardiol Curr Res. 2018;11(1):00364.
4. Meijers WC, Bayes-Genis A, Mebazaa A, Bauersachs J, Cleland JG, Coats AJ, et al. Circulating heart failure biomarkers beyond natriuretic peptides: review from the Biomarker Study Group of the Heart Failure Association (HFA), European Society of Cardiology (ESC). European journal of heart failure. 2021;23(10):1610-32.
5. Koniari I, Artopoulou E, Velissaris D, Ainslie M, Mplani V, Karavasili G, et al. Biomarkers in the clinical management of patients with atrial fibrillation and heart failure. Journal of Geriatric Cardiology: JGC. 2021;18(11):908.
6. Desmedt S, Desmedt V, De Vos L, Delanghe JR, Speeckaert R, Speeckaert MM. Growth differentiation factor 15: A novel biomarker with high clinical potential. Critical Reviews in Clinical Laboratory Sciences. 2019;56(5):333-50.
7. Wollert KC, Kempf T, Wallentin L. Growth differentiation factor 15 as a biomarker in cardiovascular disease. Clinical chemistry. 2017;63(1):140-51.
8. Tartibian B, Khayat SMA, Maleki BH, Chehrazi M. The effects of exercise training on recovery of biochemical and hematological outcomes in patients surviving COVID-19: a randomized controlled assessor-blinded trial. Sports Medicine-Open. 2022;8(1):152.
9. Husain-Syed F, McCullough PA, Birk H-W, Renker M, Brocca A, Seeger W, et al. Cardio-pulmonary-renal interactions: a multidisciplinary approach. Journal of the American College of Cardiology. 2015;65(22):2433-48.
10. Costache A-D, Costache I-I, Miftode R-Ş, Stafie C-S, Leon-Constantin M-M, Roca M, et al. Beyond the finish line: The impact and dynamics of biomarkers in physical exercise—a narrative review. Journal of Clinical Medicine. 2021;10(21):4978.
11. Attina G, Triarico S, Romano A, Maurizi P, Mastrangelo S, Ruggiero A. Serum biomarkers for the detection of cardiac dysfunction in childhood cancers receiving anthracycline-based treatment. Biomedical and Pharmacology Journal. 2022;15(3):1311-21.
12. Docherty AB, Walsh TS. Anemia and blood transfusion in the critically ill patient with cardiovascular disease. Critical Care. 2017;21:1-8.
13. Gavazza A, Marchegiani A, Guerriero L, Turinelli V, Spaterna A, Mangiaterra S, et al. Updates on laboratory evaluation of feline cardiac diseases. Veterinary Sciences. 2021;8(3):41.
14. Ion A, Stafie C, Mitu O, Ciobanu CE, Halitchi DI, Costache AD, et al. Biomarkers utility: At the borderline between cardiology and neurology. Journal of Cardiovascular Development and Disease. 2021;8(11):139.
15. Mohammadifard N, Humphries KH, Gotay C, Mena-Sánchez G, Salas-Salvadó J, Esmaillzadeh A, et al. Trace minerals intake: risks and benefits for cardiovascular health. Critical reviews in food science and nutrition. 2019;59(8):1334-46.
16. Chianca M, Panichella G, Fabiani I, Giannoni A, L'Abbate S, Aimo A, et al. Bidirectional relationship between cancer and heart failure: insights on circulating biomarkers. Frontiers in Cardiovascular Medicine. 2022;9:936654.
17. Eggers KM, Lindahl B. Application of cardiac troponin in cardiovascular diseases other than acute coronary syndrome. Clinical chemistry. 2017;63(1):223-35.
18. d'Alessandro E, Becker C, Bergmeier W, Bode C, Bourne JH, Brown H, et al. Thrombo-inflammation in cardiovascular disease: an expert consensus document from the Third Maastricht Consensus Conference on Thrombosis. Thrombosis and haemostasis. 2020;120(04):538-64.
19. Parsanathan R, Jain SK. Novel invasive and noninvasive cardiac-specific biomarkers in obesity and cardiovascular diseases. Metabolic syndrome and related disorders. 2020;18(1):10-30.
20. Wang Y, Gao L. Inflammation and cardiovascular disease associated with hemodialysis for end-stage renal disease. Frontiers in Pharmacology. 2022;13:800950.
21. Giza DE, Iliescu G, Hassan S, Marmagkiolis K, Iliescu C. Cancer as a risk factor for cardiovascular disease. Current oncology reports. 2017;19:1-8.
22. Krivickienė A, Motiejūnaitė J, Rimkevičiūtė D, Norvilaitė R, Žiubrytė G, Montvilaitė A, et al., editors. Right ventricular longitudinal systolic dysfunction is associated with worse clinical outcomes in acute dyspnea patients. European journal of heart failure Special Issue: Abstracts of the Heart Failure 2017 and the 4th World Congress on Acute Heart Failure: Paris, France, 29 April-2 May 2017; 2017: Wiley-Blackwell.

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