



JRAAS

Special Issues in Medicine & Surgery

www.internationalmedicalpublishing.com



Research Article

Section: Medicine

To Study Cardiovascular Changes in Patient of Anemia

Dr. Ravi Singh^{*1}, Dr. Shweta Sahai², Dr. Gaurav Kavi Bhargava³

¹Department of Medicine, Gajra Raja Medical College, Gwalior, Madhya Pradesh

²Professor, Department of Medicine, Gajra Raja Medical College, Gwalior, Madhya Pradesh

³Assistant Professor, Department of Cardiology, Gajra Raja Medical College, Gwalior, Madhya Pradesh

HIGHLIGHTS

- Anemia causes compensatory cardiovascular changes frequently
- Increased heart rate and stroke volume
- Cardiac murmurs and hypertrophy may develop
- Severe anemia leads to high-output failure
- Treatment reverses changes monitoring prevents complications

Key Words:

Anemia
Cardiovascular changes
Left ventricular hypertrophy
Cardiomegaly
Echocardiography

ABSTRACT

Introduction: Anemia is a widespread hematological disorder that significantly impacts cardiovascular health due to the reduced oxygen-carrying capacity of blood. Chronic anemia compels the cardiovascular system to initiate compensatory mechanisms, including tachycardia, increased stroke volume, and hyperdynamic circulation, which eventually predispose to structural and functional cardiac changes such as left ventricular hypertrophy (LVH), cardiomegaly, arrhythmias, and heart failure. **Aim and Objective:** The present study aimed to evaluate cardiovascular changes in anemic patients using electrocardiography (ECG), echocardiography, and chest X-ray, with objectives to assess the distribution of anemia types, their association with cardiovascular abnormalities, and the relationship between hemoglobin levels and cardiac function. **Materials and Methods:** This prospective observational study was conducted in the Department of General Medicine, G.R. Medical College, Gwalior, over 17 months (May 2023–September 2024). A total of 100 anemic patients (Hb <13 g/dL in males, <12 g/dL in females), aged 18–70 years, with equal gender representation, were included. Clinical evaluation, hematological investigations, ECG, echocardiography, and chest X-ray were performed. Data were analyzed using chi-square test to determine associations. **Results:** Microcytic hypochromic anemia was most common (51%), followed by normocytic normochromic (23%), macrocytic (21%), and dimorphic (5%). Cardiovascular involvement was evident, with LVH in 61%, cardiomegaly in 59%, and ECG abnormalities in 68%, mainly ST depression with T inversion and sinus tachycardia. Significant associations linked microcytic anemia with poor sanitation and worm infestation. **Conclusion:** Anemia, particularly iron deficiency type, exerts profound cardiovascular effects, with LVH, cardiomegaly, and ECG abnormalities predominating. Early recognition, correction of anemia, and routine cardiac evaluation are crucial for preventing long-term morbidity and mortality.



*Corresponding Author: Dr. Ravi Singh, E-mail: firingthakur54@gmail.com

Article History: Received 19 August 2025; Received in Revised form 20 September 2025; Accepted 22 September 2025

How To Cite: Ravi Singh, To Study Cardiovascular Changes in Patient of Anemia. *JRAAS: Special Issues in Medicine & Surgery*;2025;40(2),1-9

Anemia significantly influences the cardiovascular system because the reduced oxygen-carrying capacity of the blood compels the body to activate compensatory mechanisms in order to maintain adequate oxygen delivery to vital organs. The condition, defined by low red blood cell count or decreased hemoglobin concentration, means tissues and organs receive less oxygen than required. To counteract this deficit, the cardiovascular system increases cardiac output, which is achieved through tachycardia and sometimes by increasing stroke volume. This hyperdynamic circulation helps sustain oxygen delivery but imposes a considerable workload on the heart. If anemia persists, these compensations can precipitate long-term cardiac complications such as left ventricular hypertrophy (LVH) and eventually heart failure, particularly in individuals with pre-existing cardiovascular disease [1].

One of the earliest and most consistent changes in anemia is an elevated heart rate. By pumping faster, the heart attempts to circulate the limited oxygen supply more efficiently. Along with this, stroke volume may rise, increasing the total amount of blood propelled with each beat. Although this adaptation ensures short-term tissue oxygenation, it creates mechanical stress on the myocardium. Over time, the left ventricle enlarges and thickens in response to sustained demand, a process known as LVH. Initially adaptive, LVH eventually becomes maladaptive, resulting in impaired relaxation of the heart muscle and contributing to diastolic dysfunction and heart failure with preserved ejection fraction (HFpEF) [2].

Another consequence of anemia is myocardial ischemia, which occurs when the oxygen demand of the heart exceeds supply. Despite the heart's compensatory pumping, hemoglobin reduction limits oxygen availability. In patients with coronary artery disease, this mismatch can lead to angina and other ischemic events. Arrhythmias are also common due to structural changes such as LVH and ischemia, combined with hypoxia-induced disturbances in ionic balance within cardiac cells. This increases the risk of both atrial and ventricular arrhythmias, with atrial fibrillation being a frequent complication. Beyond myocardial effects, anemia can promote systemic vascular changes, including increased arterial stiffness and higher vascular resistance. Chronic hypoxia and inflammation contribute to endothelial dysfunction, further escalating the risk of hypertension, stroke, and myocardial infarction [3].

Clinically, patients with anemia present with symptoms that directly reflect these cardiovascular changes. Fatigue and weakness are hallmark features, stemming from inadequate oxygen supply to tissues. Shortness of breath occurs because the heart must work harder to compensate for oxygen deficiency, and in severe cases, dyspnea may even appear at rest. Palpitations, chest pain, and dizziness are frequently

reported, with angina being more pronounced in individuals with underlying coronary artery disease. In some cases, hypotension may develop due to low hemoglobin, while others may present with normal or elevated blood pressure depending on the type of anemia and changes in circulating blood volume. Additional symptoms such as cold extremities, syncope, and edema further illustrate how widespread the cardiovascular repercussions can be [4].

Diagnosis of these cardiovascular adaptations requires a combination of clinical evaluation, laboratory investigations, and imaging studies. A complete blood count provides the initial evidence of anemia, while serum iron, ferritin, and vitamin levels help identify the type. Cardiac involvement can be evaluated using ECG, which often reveals arrhythmias or ischemic changes. Echocardiography is particularly valuable in detecting LVH, diastolic dysfunction, or heart failure. Biomarkers such as BNP or NT-proBNP can signal heart failure, while elevated C-reactive protein may reflect systemic inflammation. Together, these investigations clarify both the hematological and cardiovascular aspects of the disease [5].

Management of cardiovascular changes in anemia requires addressing both the root cause of anemia and the secondary cardiovascular stress. Iron deficiency is treated with supplementation, either orally or intravenously, while deficiencies of vitamin B12 and folate are corrected with appropriate replacement. In chronic disease anemia, erythropoiesis-stimulating agents may be required, particularly in patients with kidney disease. Blood transfusions are sometimes used in severe cases to quickly restore hemoglobin levels and relieve cardiac strain. Alongside correcting anemia, cardiovascular complications must also be managed. Antihypertensives such as ACE inhibitors or ARBs can reduce LVH and lower afterload, while beta-blockers help control tachycardia and prevent arrhythmias. Diuretics are useful for treating fluid overload in heart failure, and statins and antiplatelet therapy may be indicated for patients with ischemic heart disease. Equally important are lifestyle modifications, including dietary adjustments, smoking cessation, and moderate physical activity, which collectively help improve cardiovascular resilience [6].

The prognosis of cardiovascular anemia is closely linked to timely recognition and effective treatment. Prompt correction of anemia improves oxygen delivery, alleviates symptoms such as fatigue, palpitations, and dyspnea, and restores exercise tolerance and quality of life. Long-term outcomes, however, depend on how well anemia and its cardiovascular consequences are managed. Persistent anemia can lead to progressive left ventricular hypertrophy, recurrent ischemic episodes, worsening heart failure, and higher morbidity and mortality, particularly in patients with chronic kidney disease or existing heart disease [7]. Early intervention helps relieve cardiac strain, lowers ischemic

risk, and prevents irreversible myocardial damage. Continuous monitoring of hemoglobin, cardiac biomarkers, and echocardiographic parameters is essential to guide therapy and minimize complications. A comprehensive, multidisciplinary approach that integrates correction of hematologic abnormalities, cardiovascular management, and lifestyle modification significantly improves survival, enhances overall prognosis, and safeguards long-term cardiovascular health in patients with anemia [8].

The present study aims to evaluate cardiovascular changes in patients with anemia through ECG, echocardiography, and chest X-ray. It further seeks to assess the distribution of anemia types among hospitalized patients, examine the correlation between different types of anemia and associated cardiovascular abnormalities, and analyze the relationship between hemoglobin levels and cardiac function, thereby providing insights into the impact of anemia on cardiovascular health and guiding effective clinical management.

MATERIALS AND METHODS

This prospective observational study was carried out in the

Department of General Medicine, G.R. Medical College, Gwalior (M.P.), over 17 months from May 2023 to September 2024. A total of 100 anemic patients, defined by WHO criteria (hemoglobin <13 g/dL in males, <12 g/dL in females), aged 18–70 years of both genders, were enrolled with equal gender distribution. Inclusion required confirmed anemia and consent, while exclusions were age limits, refusal, hematologic malignancies, or chemotherapy. Ethical clearance was obtained, written consent ensured, and patient confidentiality maintained, with voluntary participation and withdrawal rights safeguarded.

RESULTS

In the present study, most patients (65%) were in the age group of 31–45 years, followed by 16% in the 46–60 years group. A smaller proportion belonged to the 61–75 years (9%) and 15–30 years (10%) categories. Among the 100 participants, males and females were equally represented, with each accounting for 50% of the study population. This equal gender distribution highlights that both sexes were comparably affected, while the majority of cases clustered in early to middle adulthood.

Table 1: Distribution of Patients According to Type of Anemia

Type of Anemia	Count	Percentage
Microcytic Hypochromic	51	51.0
Normocytic Normochromic	23	23.0
Macrocytic	21	21.0
Dimorphic Anemia	5	5.0
Total	100	100.0

The table shows that microcytic hypochromic anemia was the most common type, affecting more than half of the patients (51%), indicating iron deficiency as a major cause. Normocytic normochromic anemia (23%) and macrocytic anemia (21%) were also relatively frequent, suggesting other

underlying nutritional or systemic factors. Dimorphic anemia was the least common (5%), reflecting mixed etiologies. Overall, the distribution highlights the predominance of iron deficiency anemia while also pointing to varied pathological mechanisms contributing to chronic anemia.

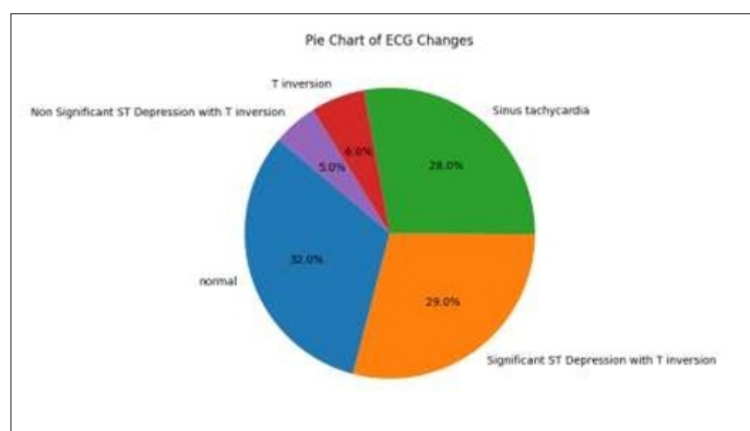


Figure 1: Distribution of Patients Based on ECG Changes

The ECG distribution shows that the majority of patients (32%) had normal findings, while significant ST depression with T inversion (29%) and sinus tachycardia (28%) were almost equally prevalent, reflecting common cardiac stress patterns in anemia. Less frequent abnormalities included

isolated T wave inversion (6%) and nonsignificant ST depression with T inversion (5%), indicating milder electrical disturbances. Overall, although one-third had normal ECGs, most patients demonstrated varying degrees of cardiac involvement, highlighting the cardiovascular impact of anemia.

Table 2: Distribution of Patients Based on Echo Structural Changes

Echo Structural Changes	Count	Percentage
LVH	61	61.0
Normal	39	39.0
Others (Regurgitant Lesions)	0	0.00
Total	100	100.0

The echocardiographic findings reveal that left ventricular hypertrophy (LVH) was the predominant structural change, observed in 61% of patients, reflecting the adaptive response of the heart to chronic anemia-induced increased cardiac workload. Normal echocardiographic patterns were noted in

39% of patients, while no cases of regurgitant lesions or other abnormalities were detected. These results suggest that LVH is the most significant structural cardiac alteration associated with anemia, emphasizing its role in long-term cardiovascular remodeling.

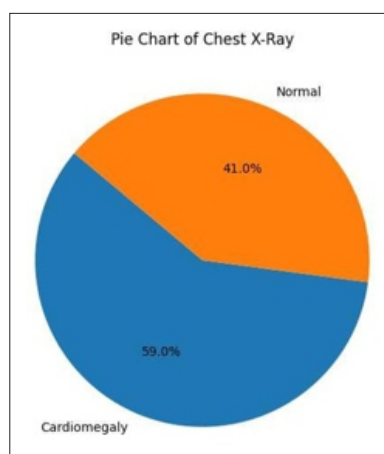


Figure 2: Distribution of Patients Based on Chest X-Ray Findings

The chest X-ray findings demonstrate that cardiomegaly was present in the majority of patients (59%), indicating cardiac enlargement likely due to chronic anemia-related hemodynamic stress. In contrast, 41% of patients had normal

chest X-rays, showing no evidence of structural changes. This distribution highlights the significant burden of cardiac remodeling in anemia, with cardiomegaly emerging as a common radiological manifestation.

Table 3: Association of Type of Anemia with Gender

Type of Anemia	Dimorphic anemia	Macrocytic	Microcytic Hypochromic	Normocytic Normochromic	Total
Gender					
Female	1 (2.0%)	13 (26.0%)	25 (50.0%)	11 (22.0%)	50 (100.0%)
Male	4 (8.0%)	8 (16.0%)	26 (52.0%)	12 (24.0%)	50 (100.0%)
Total	5 (5.0%)	21 (21.0%)	51 (51.0%)	23 (23.0%)	100 (100.0%)
'Chi-square test statistic: 3.0536, P-value: 0.3834'					

The table shows that microcytic hypochromic anemia was the most common type in both males (52%) and females (50%), followed by macrocytic and normocytic normochromic anemia. Dimorphic anemia was rare, with slightly higher

occurrence in males (8%) compared to females (2%). The chi-square test ($\chi^2 = 3.0536$, $p = 0.3834$) indicates no statistically significant association between gender and type of anemia. Thus, anemia types were distributed similarly across males and females in the study population.

Table 4: Association of Type of Anemia with History of Open Defecation

Type of Anemia	Dimorphic Anemia	Macrocytic	Microcytic Hypochromic	Normocytic Normochromic	Total
Hx of Open Defecation					
Absent	0 (0.0%)	16 (27.12%)	21 (35.59%)	22 (37.29%)	59 (100.0%)
Present	5 (12.2%)	5 (12.2%)	30 (73.17%)	1 (2.44%)	41 (100.0%)
Total	5 (5.0%)	21 (21.0%)	51 (51.0%)	23 (23.0%)	100 (100.0%)
'Chi-square test statistic: 29.2311, P-value: 0.0000'					

The table highlights a strong association between type of anemia and history of open defecation. Among patients with open defecation, microcytic hypochromic anemia was strikingly predominant (73.17%), compared to only 35.59% in those without such history. Conversely, normocytic

normochromic anemia was more frequent in the absence of open defecation (37.29% vs. 2.44%). The chi-square test ($\chi^2 = 29.2311$, $p = 0.0000$) confirms this relationship as statistically significant, suggesting poor sanitation and parasitic infestations may contribute to iron deficiency anemia in these patients.

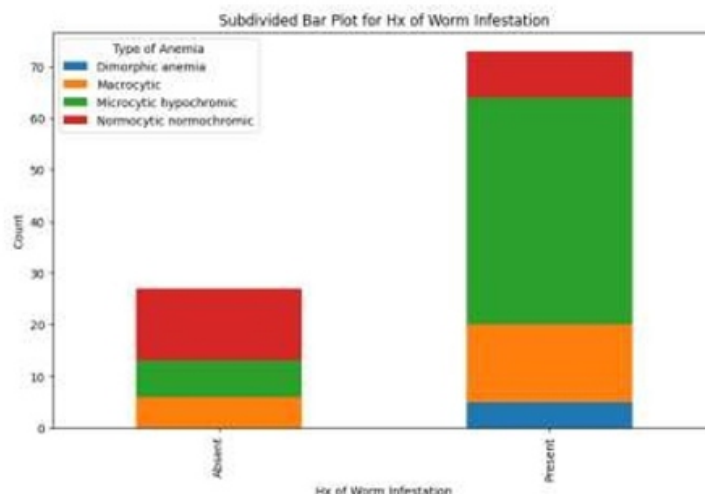


Figure 3: Association of Type of Anemia with History of Worm Infestation

The figure demonstrates a clear association between worm infestation and type of anemia. Patients with a history of worm infestation showed a markedly higher prevalence of microcytic hypochromic anemia, reflecting iron deficiency due to chronic blood loss and nutritional depletion caused by

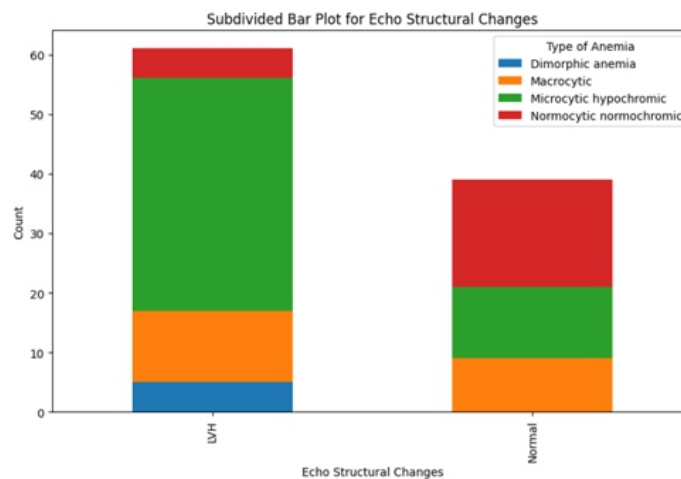
parasites. In contrast, those without infestation had relatively fewer cases and a more balanced distribution among macrocytic and normocytic anemia types. This trend highlights worm infestation as an important etiological factor in the development of iron deficiency anemia within the study population.

Table 5: Association of Type of Anemia with ECG Changes

Type of Anemia	Dimorphic Anemia	Macrocytic	Microcytic Hypochromic	Normocytic Normochromic	Total
ECG Changes					
Non-Significant ST Depression with Inversion	0 (0.0%)	1 (20.0%)	4 (80.0%)	0 (0.0%)	5 (100.0%)
Significant ST Depression with T Inversion	4 (13.79%)	1 (3.45%)	24 (82.76%)	0 (0.0%)	29 (100.0%)
Sinus Tachycardia	1 (3.57%)	8 (28.57%)	14 (50.0%)	5 (17.86%)	28 (100.0%)
T Inversion	0 (0.0%)	3 (50.0%)	0 (0.0%)	3 (50.0%)	6 (100.0%)
Normal	0 (0.0%)	8 (25.0%)	9 (28.12%)	15 (46.88%)	32 (100.0%)
Total	5 (5.0%)	21 (21.0%)	51 (51.0%)	23 (23.0%)	100(100.0%)
'Chi-square test statistic: 45.2992, P-value: 0.0000'					

The table reveals a significant association between type of anemia and ECG changes. Microcytic hypochromic anemia showed the highest prevalence of abnormalities, particularly significant ST depression with T inversion (82.76%) and sinus tachycardia (50%). Macrocytic anemia was more often linked with sinus tachycardia (28.57%) and isolated T wave

inversion (50%), while normocytic normochromic anemia was predominantly associated with normal ECGs (46.88%). The chi-square test ($\chi^2 = 45.2992$, $p = 0.0000$) confirms this relationship as statistically significant, highlighting that different anemia types predispose to distinct cardiac electrical

Figure 4: Association of Type of Anemia with Echo Structural Changes

The bar plot shows a strong association between anemia type and echocardiographic changes. Left ventricular hypertrophy (LVH) was predominantly seen in patients with microcytic hypochromic anemia, followed by macrocytic and dimorphic types, indicating a higher burden of structural remodeling in

these groups. In contrast, patients with normal echocardiographic findings were more often associated with normocytic normochromic anemia. This pattern highlights that severe and chronic forms of anemia, especially iron deficiency, are more likely to cause cardiac hypertrophy compared to other anemia types.

Table 6: Association of Type of Anemia with Chest X-Ray Findings

Type of Anemia	Dimorphic Anemia	Macrocytic	Microcytic Hypochromic	Normocytinormochromic	Total
Chest X-Ray					
Cardiomegaly	5 (8.47%)	11 (18.64%)	38 (64.41%)	5 (8.47%)	59 (100.0%)
Normal	0 (0.0%)	10 (24.39%)	13 (31.71%)	18 (43.9%)	41 (100.0%)
Total	5 (5.0%)	21 (21.0%)	51 (51.0%)	23 (23.0%)	100 (100.0%)
'Chi-square test statistic: 22.1273, P-value: 0.0001'					

The table shows a significant association between type of anemia and chest X-ray findings. Cardiomegaly was most frequent in microcytic hypochromic anemia (64.41%), followed by macrocytic (18.64%) and dimorphic (8.47%) cases, reflecting the strong link between iron deficiency and cardiac enlargement. On the other hand, normal chest X-rays were more common in normocytic normochromic anemia (43.9%) and macrocytic anemia (24.39%). The chi-square test ($\chi^2 = 22.1273$, $p = 0.0001$) confirms this association as statistically significant, suggesting that microcytic hypochromic anemia carries the highest risk for structural cardiac changes detectable on X-ray.

DISCUSSION

Anemia, defined as a reduction in hemoglobin (Hb) concentration or red blood cell (RBC) count, impairs oxygen delivery and contributes significantly to cardiovascular morbidity and mortality worldwide. The cardiovascular system compensates for reduced oxygen supply by increasing cardiac output through tachycardia and stroke volume. While initially protective, prolonged anemia induces pathological changes such as left ventricular hypertrophy (LVH), heart failure, and ischemic complications, especially in individuals with pre-existing cardiovascular disease [9].

The mismatch between myocardial oxygen supply and demand in anemia worsens ischemic heart disease and may precipitate angina or myocardial infarction [10]. Patients with comorbidities, particularly chronic kidney disease (CKD), diabetes, and hypertension, face an amplified cardiovascular burden. CKD-associated anemia is strongly linked to heart failure with preserved ejection fraction (HFpEF), highlighting the interplay between renal dysfunction, anemia, and cardiac remodeling. Mechanistically, iron deficiency anemia reduces hemoglobin synthesis and oxygen transport, while anemia of chronic disease arises from inflammatory cytokines impairing erythropoiesis, both pathways exacerbating cardiac dysfunction [11].

Our study highlighted significant structural and functional cardiac alterations in anemic patients. Electrocardiographic (ECG) analysis revealed sinus tachycardia (28%), ST depression with T inversion (29%), and isolated T inversions (6%), consistent with ischemic strain from inadequate oxygen delivery. Prior studies confirm a high prevalence of ECG abnormalities in anemia, linking them to clinical severity and adverse prognosis [12]. Echocardiography demonstrated LVH in 61% of cases, with none showing regurgitant lesions. This aligns with reports by Broughton et al. (2019) and Carson et al. (1996), which emphasize LVH as a hallmark adaptive response to chronic anemia [12,13]. Radiological evaluation revealed cardiomegaly in 59% of patients, corroborating findings by Haider et al. (2020) and others, where chronic anemia predisposed to heart enlargement and remodeling [14].

Mortality in our cohort was modest (2–4%), primarily in patients >70 years. Cohen et al. (2017) reported higher mortality, particularly in heart failure patients, where anemia accelerates decompensation [15]. Gender distribution of anemia types was not statistically significant, though earlier studies by Kotecha et al. (2019) suggested male predisposition to microcytic anemia-related cardiac remodeling [16].

Environmental and parasitic factors strongly influenced anemia severity. A significant association was observed between microcytic hypochromic anemia and both open defecation (73%) and worm infestation (60%), reflecting nutritional and infectious etiologies. Historical studies by Varat et al. (1972) and Carson et al. (1996) similarly reported that parasitic infestations worsen anemia and, in turn, increase cardiovascular strain [12,17].

Organ involvement was notable, with hepatomegaly in 61% and hepatosplenomegaly in 2% of microcytic anemia patients, findings comparable to Varat et al. (1972) [17]. Mean hemoglobin (7.3 g/dL) and reduced ejection fraction (52.7%) reflected severe anemia's impact on myocardial function. Lanser et al. (2021) linked such biochemical derangements with impaired oxygenation and cardiac efficiency [18].

Anemia severity further influenced cardiovascular outcomes. Severe anemia (43%) was associated with higher rates of LVH (68.8%), cardiomegaly (67.8%), and reduced ejection fraction, consistent with Anand et al. (2004) and Kaiafa et al. (2015) [19,20]. Gender-wise, severe anemia predominated in men (58%), whereas moderate anemia was more common in women (60%), supporting previous findings that men present with more severe forms due to underlying chronic disease [20].

our findings demonstrate that anemia, particularly microcytic hypochromic type, is strongly associated with cardiovascular alterations including LVH, cardiomegaly, ECG changes, and reduced ejection fraction. These results reinforce prior evidence [14], underscoring the dual necessity of managing anemia and monitoring cardiac function to mitigate morbidity and mortality.

CONCLUSION

This study underscores the significant interplay between anemia and cardiovascular alterations, revealing how microcytic hypochromic anemia (51%) and dimorphic anemia are strongly associated with left ventricular hypertrophy, cardiomegaly, and abnormal ECG changes, particularly in severe cases with reduced ejection fraction. Conducted on 100 patients with equal gender distribution, predominantly aged 31–45 years, the findings highlight anemia's systemic impact beyond hematology, with sanitation and nutrition playing major roles in severity. Importantly, cardiac changes correlated inversely with hemoglobin levels, independent of liver or renal dysfunction, emphasizing the need for early diagnosis, routine cardiac screening, and integrated public health interventions.

REFERENCES

- Obeagu EI, Obeagu GU. Anemia and cerebrovascular disease: pathophysiological insights and clinical implications. *Annals of Medicine and Surgery*. 2025 Jun 1;87(6):3254-67.
- BUILDER V. Cardiovascular Pathologies and Disorders. *Mosby's Pathology for Massage Professionals-E-Book: Mosby's Pathology for Massage Professionals-E-Book*. 2021 Sep 5;234.
- Manolis AS, Koulouris S, Triantafyllou K, Sakellariou D, Pastromas S, Melita H. Anemia and cardiovascular disease. *Alternatives to Blood Transfusion in Transfusion Medicine*, Second Edition. Chichester: Wiley-Blackwell. 2011.
- Obeagu EI, Ali MM, Alum EU, Obeagu GU, Ugwu PO, Bunu UO. An Update of Aneamia in Adults with Heart Failure.
- Garcia-Casal MN, Dary O, Jefferds ME, Pasricha SR. Diagnosing anemia: Challenges selecting methods, addressing underlying causes, and implementing actions at the public health level. *Annals of the New York Academy of Sciences*. 2023 Jun;1524(1):37-50.
- Van Veldhuisen DJ, Anker SD, Ponikowski P, Macdougall IC. Anemia and iron deficiency in heart failure: mechanisms and therapeutic approaches. *Nature Reviews Cardiology*. 2011 Sep;8(9):485-93.
- Qaseem A, Humphrey LL, Fitterman N, Starkey M, Shekelle P. Treatment of anemia in patients with heart disease: a clinical practice guideline from the American College of Physicians. *Annals of internal medicine*. 2013 Dec 3;159(11):770-9.
- Goel H, Hirsch JR, Deswal A, Hassan SA. Anemia in cardiovascular disease: marker of disease severity or disease-modifying therapeutic target? *Current atherosclerosis reports*. 2021 Oct;23(10):61.
- Wolanskyj-Spinner AP, Go RS. New Developments in the Understanding and Treatment of Autoimmune Hemolytic Anemia, An Issue of Hematology/Oncology Clinics of North America, E-Book: New Developments in the Understanding and Treatment of Autoimmune Hemolytic Anemia, An Issue of Hematology/Oncology Clinics of North America, E-Book: Elsevier Health Sciences; 2022.
- Kaiafa G, Kanellos I, Savopoulos C, Kakaletsis N, Giannakoulas G, Hatzitolios AIJoc. Is anemia a new cardiovascular risk factor? 2015;186:117-24.
- Lin S. *Comprehensive Frontier of Kidney Disease*: World Scientific; 2024.
- Carson JL, Duff A, Poses RM, Berlin JA, Spence RK, Trout R, et al. Effect of anaemia and cardiovascular disease on surgical mortality and morbidity. 1996;348(9034):1055-60.
- Broughton, H. M., & Vassalotti, J. A. (2019). Anemia in chronic kidney disease: Current diagnosis and management. *Journal of Clinical Medicine*, 8(11), 1789.
- Haider, Z. M., & Shaikh, S. (2020). Iron deficiency anemia and cardiovascular health: The bidirectional relationship. *Cardiovascular Research*, 115(3), 423-432.
- Cohen, J. A., & Kriegel, M. (2017). Cardiovascular effects of anemia: A review of pathophysiology and clinical management. *European Heart Journal*, 38(2), 113-120.
- Kotecha, D., & Dunning, J. (2019). Anemia and cardiovascular disease: The importance of early diagnosis and management. *American Journal of Cardiovascular Disease*, 9(1), 35-44.
- Varat MA, Adolph RJ, Fowler NO. Cardiovascular effects of anemia. *American heart journal*. 1972 Mar 1;83(3):415-26.
- Lanser L, Fuchs D, Scharnagl H, Grammer T, Kleber ME, März W, Weiss G, Kurz K. Anemia of chronic disease in patients with cardiovascular disease. *Frontiers in cardiovascular medicine*. 2021 Aug 12; 8:666638.
- Anand I, McMurray JJ, Whitmore J, Warren M, Pham A, McCamish MA, Burton PB. Anemia and its relationship to

clinical outcome in heart failure. *Circulation*. 2004 Jul 13;110(2):149-54.

20. Kaiafa G, Kanellos I, Savopoulos C, Kakaletsis N, Giannakoulas G, Hatzitolios AI. Is anemia a new cardiovascular risk factor? *International journal of cardiology*. 2015 May 1; 186:117-24.