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Relationship Between Hematocrit (HCT) and Heart Failure

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العلاقة بين الهيماتوكريت (HCT) وفشل القلب

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Abstract
Heart failure is often accompanied by anemia, which can lead to impaired oxygen supply and serious consequences. Hematocrit (HCT) is an important measure of red blood cell volume and disease severity. This study aimed to determine the prevalence of low hematocrit in heart failure patients and its association with hemodilution, iron deficiency, functional status, and clinical outcomes. Fifty heart failure patients at Mitiga Heart Hospital in Tripoli, Libya, participated in a cross-sectional study. Data collected included demographics, NYHA classification, comorbidities, medications, laboratory markers (hematocrit, hemoglobin, ferritin, transferrin saturation, BNP), and echocardiography (LVEF, LVEDD). The relationship between hematocrit, iron status, hemodilution, and clinical outcomes was studied using descriptive statistics and percentages. Fifty-eight percent of patients had low hemoglobin levels (HCT) (<35%), with the majority being elderly (mean age 61.2 ± 11.4 years) and NYHA class III-IV patients (44%). Thirty-six percent (ferritin <100 ng/mL) and 28% (TSAT <20%) had iron deficiency. Forty percent of patients experienced hemodilation, often accompanied by edema and weight gain. Only 24% of patients received iron supplementation and 12% received erythropoietin despite following guidelines. Anemia was strongly associated with poor outcomes, as evidenced by increased hospitalization rates (32%) and all recorded deaths (6%) among patients with low HCT. Low HCT is very common in heart failure patients and is associated with worse clinical outcomes, iron deficiency, hemodilation, and disease progression. To improve prognosis, targeted therapies, iron status assessment, and routine HCT monitoring are crucial. Further research is needed to confirm this effect. 36% (ferritin <100 ng/mL) and 28% (TSAT <20%) were iron deficient. 40% of patients experienced hemolysis, often accompanied by edema and weight gain. To improve prognosis, targeted therapies, iron status assessment, and periodic monitoring with hematopoietic stem cell imaging (HCT) are crucial. Further research is needed to determine how anemia correction affects survival and functional capacity.

Keywords: NYHA Classification, Hematocrit, Heart Failure, Anemia, Iron Deficiency and Hemodilution.

ملخص
غالبًا ما يترافق فشل القلب مع فقر الدم (الأنيميا)، مما قد يؤدي إلى ضعف إمداد الأكسجين وعواقب وخيمة. يعتبر مكداس الدم) الهيماتوكريت (HCT - مقياسًا مهمًا لحجم خلايا الدم الحمراء وشدة المرض. هدفت هذه الدراسة إلى تحديد مدى انتشار انخفاض الهيماتوكريت لدى مرضى فشل القلب وارتباطه بتخفيف الدم(Hemodilution) ، ونقص الحديد، والحالة الوظيفية، والنتائج السريرية. شارك في الدراسة المقطعية خمسون مريضًا بفشل القلب في مستشفى ميليتا للقلب في طرابلس، ليبيا. شملت البيانات التي تم جمعها المعلومات الديموغرافية، وتصنيف جمعية القلب في نيويورك(NYHA) ، والأمراض المصاحبة، والأدوية، والعلامات المخبرية) الهيماتوكريت، الهيموغلوبين، الفيريتين، تشبع الترانسفيرين، (BNP، وتخطيط صدى القلبLVEF) ، .(LVEDDتمت دراسة العلاقة بين الهيماتوكريت وحالة الحديد وتخفيف الدم والنتائج السريرية باستخدام الإحصائيات الوصفية والنسب المئوية. كان لدى 58%من المرضى مستويات منخفضة من الهيموغلوبين (الهيماتوكريت (35%>، وكانت غالبيتهم من كبار السن (متوسط العمر 11.4$ \pm 61.2$سنة) ومرضى NYHA من الفئة الثالثة أو الرابعة .(44%) كان 36%يعانون من نقص الحديد (فيريتين 100>نانوغرام/مل) و) 28%تشبع الترانسفيرين (20%>يعانون من نقص الحديد. عانى 40%من المرضى من تخفيف الدم، والذي غالبًا ما كان مصحوبًا بالوذمة وزيادة الوزن. بالرغم من اتباع الإرشادات، لم يتلق سوى 24%من المرضى مكملات الحديد وتلقى 12%فقط الإريثروبويتين. ارتبط فقر الدم بشدة بسوء النتائج، كما يتضح من زيادة معدلات دخول المستشفى (32%) وجميع الوفيات المسجلة (6%) بين المرضى الذين يعانون من انخفاض الهيماتوكريت. يعد انخفاض الهيماتوكريت (HCT) شائعًا جدًا لدى مرضى فشل القلب ويرتبط بتدهور النتائج السريرية، ونقص الحديد، وتخفيف الدم، وتفاقم المرض. لتحسين التكهن بالمرض،

يعد العلاج المستهدف، وتقييم حالة الحديد، والمراقبة الروتينية لمكداس الدم (HCT) أمرًا بالغ الأهمية. هناك حاجة إلى مزيد من الأبحاث لتحديد كيفية تأثير تصحيح فقر الدم على البقاء على قيد الحياة والقدرة الوظيفية.

الكلمات المفتاحية :تصنيفNYHA ، الهيماتوكريت، فشل القلب، فقر الدم (الأنيميا)، نقص الحديد، وتخفيف الدم.

Introduction:

Despite significant advances in therapeutic techniques and diagnostic tools over the past few decades, heart failure (HF) remains a major health concern worldwide due to its consistently high morbidity and mortality rates. According to epidemiological estimates, HF is one of the leading causes of hospital admissions among people over 50 years of age, affecting approximately 64 million people globally to varying degrees (Ziaeian & Fonarow, 2016; Groenewegen *et al.,* 2020). The clinical significance of this syndrome stems from its chronic and progressive nature: structural and functional abnormalities of the myocardium impair the heart's ability to provide sufficient blood flow to meet the metabolic needs of the tissues by impairing both ventricular filling capacity and ejection capacity (McDonagh *et al.,* 2021). Hematocrit (HCT), the ratio of red blood cell volume to total blood volume, is one of the laboratory markers that is increasingly being studied in relation to heart failure (Rodak *et al.,* 2020). HCT readings are often low in heart failure patients, which may indicate anemia. This reduces the heart's ability to carry oxygen and increases stress on the damaged myocardium. A low HCT can result from blood thinning associated with vasodilation or from true anemia due to decreased red blood cell production or nutritional deficiencies, both of which are common in chronic heart failure (Silverberg *et al.,* 2001; Anand *et al.,* 2008). On the other hand, an elevated HCT may be a compensatory response to prolonged hypoxia, but it also increases vascular resistance and blood viscosity, potentially increasing cardiac stress and predisposing patients to stroke (Tistani *et al.,* 2014). A low red blood cell count is a significant prognostic factor associated with increased hospitalization and mortality rates in heart failure patients, as clinical research has repeatedly demonstrated (Ezekowitz et al., 2010; Westenbrink *et al.,* 2013). According to Andron *et al.* (2003), approximately 50% of patients with advanced heart failure experience significant dilution of red blood cell concentration, which negatively impacts cardiac function and exercise tolerance. Other studies (de Denus *et al.,* 2008; Zhou *et al.,* 2017) have shown that sequential changes in red blood cell count have prognostic significance independent of biomarkers such as left ventricular ejection fraction (LVEF) or beta-natriuretic peptide (BNP). The ratio of blood ejected from the ventricle with each systole to the end-diastolic volume is known as the left ventricular ejection fraction (LVEF) (Kumar *et al.,* 2018; Mann *et al.,* 2015)

the given formula: $\boldsymbol{EF = (StrokeVolume/End - DiastolicVolume) \times 100}$

The ratio of blood pumped by the left ventricle with each contraction (pulse volume) to the total blood volume in the ventricle before systole (end-diastolic volume) is known as the ejection fraction (EF) and is determined by this equation. A normal EF ranges from 55% to 70%; values below 30% indicate severe systolic dysfunction, values between 40% and 55% indicate mild dysfunction, and values between 30% and 40% indicate moderate dysfunction. Because stem cell angiography is a widely available and low-cost measure that can help determine the severity of heart failure and direct treatment options, understanding the relationship between stem cell angiography and cardiac function has significant clinical value. It is important to determine whether a decrease in hematocrit (HCT) is due to hemodilution or actual anemia, given the potential for different therapeutic approaches, such as adjusting diuretic therapy, treating iron deficiency, or promoting erythropoiesis (Anker *et al.,* 2009; McDonagh *et al*., 2021). Therefore, this study aims to evaluate the relationship between HCT levels and important indicators of cardiac function in heart failure patients, such as ejection fraction and BNP concentrations. Furthermore, it aims to ascertain whether changes in HCT can serve as a physiological indicator to aid in monitoring clinical progression and assessing the severity of heart failure.

Materials and Methods:

Between March and September 2025, a descriptive cross-sectional clinical study was conducted at Mitiga Heart Hospital (Tripoli, Libya) to evaluate the relationship between cardiac function and hematocrit (HCT) levels in patients with heart failure (HF). All participants provided written informed consent in accordance with the principles of the Declaration of Helsinki, and the study was approved by the local ethics committee (World Medical Association, 2013).

In accordance with ESC 2021 recommendations, the study population consisted of fifty adult patients (aged 30–85 years) with heart failure (McDonagh et al., 2021). HCT, hemoglobin, ferritin, renal indices, BNP/NT-proBNP, and echocardiographic confirmation of heart failure (LVEF ≤ 50%) were among the laboratory data available for eligible individuals. Individuals with hematological malignancies, current bleeding, or recent blood transfusions (less than three months prior) were excluded.

Data Collection:

A validated questionnaire encompassing identity information, HF features, comorbidities, medication history, cardiac tests, and laboratory results was used to gather clinical and demoFigureic data. To guarantee accuracy, all entries were examined and verified by licensed cardiologists.

Laboratory Analysis: Standard phlebotomy techniques were used to obtain venous blood samples.

- A Sysmex XN-Series analyzer was used to measure HCT and CBC.
- The Abbott Architect i1000SR chemiluminescent immunoassay was used to measure serum iron and ferritin.
- ECLIA (Roche Cobas e601) was used to measure BNP/NT-proBNP. • Enzymatic and ion-selective electrode techniques were used to examine renal indicators and electrolytes.
- Internal quality control and daily calibration were carried out in accordance with CLSI recommendations (CLSI, 2020).

EchocardioFigureic and Anthropometric Measurements: EchocardioFigureic and anthropometric measurements: A Philips EPIQ 7C echocardiogram was performed, and the Simpson biplane method was used to calculate left ventricular ejection fraction (LVEF) according to American Heart Association (ASE) recommendations (Lang et al., 2015). Body mass index was calculated using weight (kg)/height² (m²).**Statistical Analysis**: SPSS version 26 was used for statistical analysis. Categorical data were used as frequencies, while continuous variables were used as mean ± standard deviation. Pearson's correlation coefficient was used to assess the relationship between hematocrit (HCT) and cardiac/biochemical markers (EF, BNP, creatinine, ferritin, and hemoglobin). HCT categories were compared using an independent samples t-test. A p-value < 0.05 was set as the threshold for statistical significance (Pallant., 2020).

Results

1. Parameters of the Study Sample

The validated questionnaire and laboratory evaluation were completed by fifty clinically confirmed heart failure patients from Mitiga Heart Hospital in Tripoli, Libya. The sample represented an older population with a mean age of 61.4 ± 11.2 years and a mean body mass index of 28.7 ± 4.6 kg/m², indicating overweight. Males comprised 58% of the participants, while females comprised 42%. The majority of patients (72%) had chronic heart failure, and more than half (56%) were classified as Class III to IV according to the New York Heart Association (NYHA) classification, reflecting the severity of their advanced symptoms. Comorbidities were common, with 44% having diabetes, 62% having hypertension, and 28% having kidney disease. In addition, 48% of the patients had been hospitalized within the past twelve months, highlighting a clinically vulnerable population with a significant disease burden.

Table 1 Shows Baseline DemoFigureic and Clinical Characteristics (n = 50)

Variable	Mean ± SD / n (%)
Age (years)	61.4 ± 11.2
BMI (kg/m²)	28.7 ± 4.6
Sex (Male)	29 (58%)
Sex (Female)	21 (42%)
Chronic HF	36 (72%)
NYHA III–IV	28 (56%)
Diabetes Mellitus	22 (44%)
Hypertension	31 (62%)
Kidney Disease	14 (28%)
Recent Hospitalization (12 months)	24 (48%)

2. Distribution of cases by gender

The sex distribution of the study population shows that males represented 58% of the sample, while females accounted for 42%, indicating a modest predominance of male participants.

Table 2 Shows Distribution of cases by gender

Sex	Frequency (n)	Percentage (%)
Male	29	58%
Female	21	42%

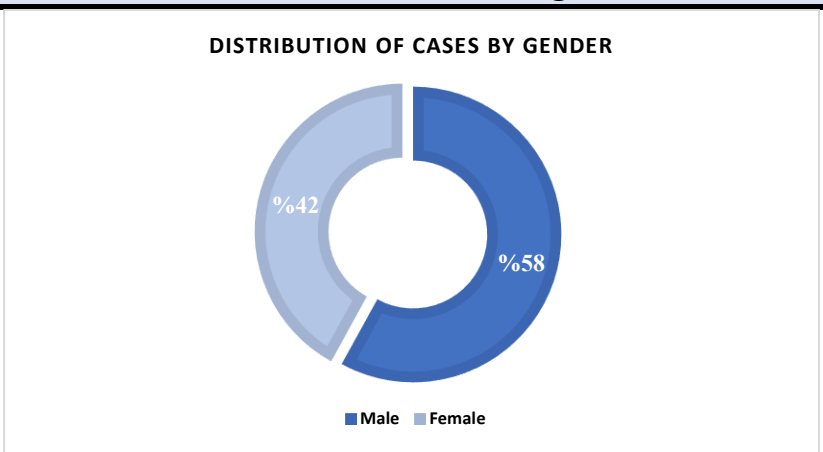

Figure 1 Shows Distribution of cases by gender.

3. Age Distribution of Study Participant

The age distribution shows that the sample was predominantly older, with 36% of participants aged 60-69 and 26% aged 70 or older. Younger individuals were less represented, with 14% under 50 and 24% aged 50-59. This pattern suggests that the group consisted mainly of middle-aged and elderly patients.

Table 3 Shows Age Distribution of Study Participants.

Age Group (years)	Frequency (n)	Percentage (%)
< 50	7	14%
50–59	12	24%
60–69	18	36%
≥ 70	13	26%

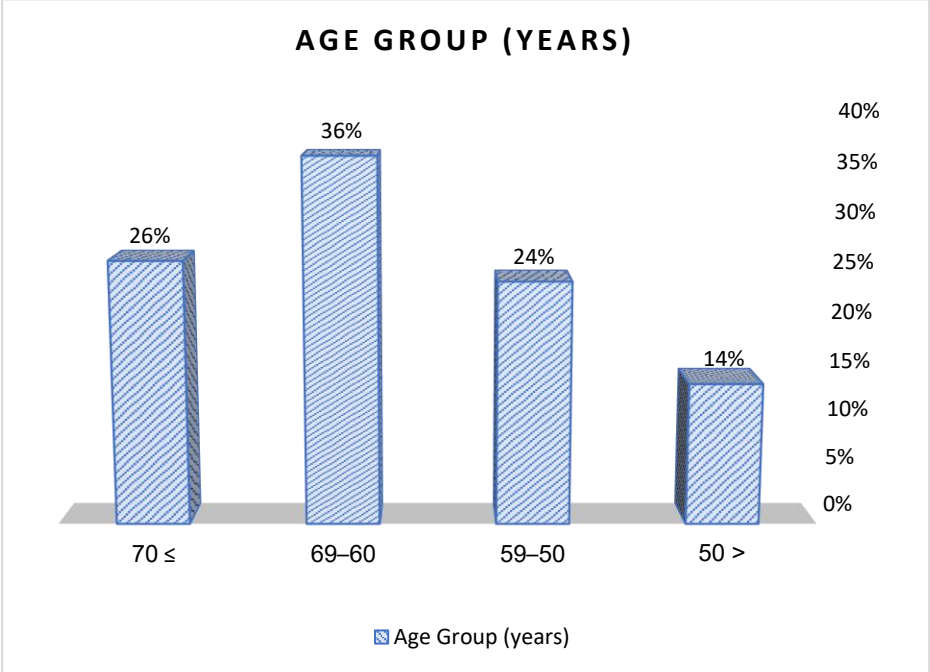

Figure 2 Shows Distribution of cases by gender**.**

4. Distribution of BMI Categories Among the Study Sample

The BMI distribution shows that most participants were above the normal weight range, with 42% classified as overweight and 36% as obese. Only 22% fell within the normal BMI category, indicating that excess body weight was common in the studied population.

Table (4) Shows Distribution of BMI Categories Among the Study Sample.

BMI Category	Frequency (n)	Percentage (%)
Normal (18.5–24.9)	11	22%
Overweight (25–29.9)	21	42%
Obese (≥30)	18	36%

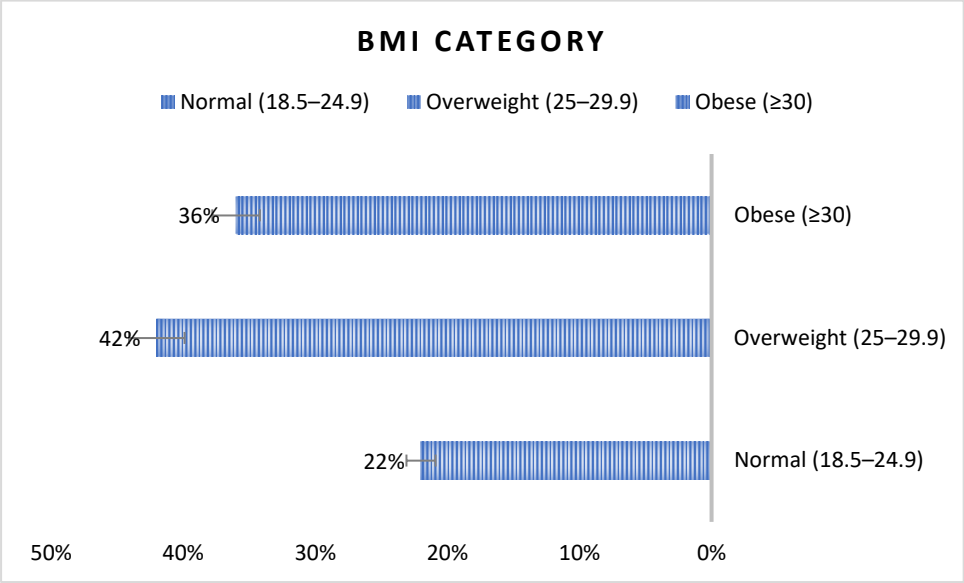

Figure 3 Shows Distribution of BMI Categories Among the Study Sample.

4. Distribution of Heart Failure Types Among the Study Sample

The distribution of heart failure types indicates that the majority of patients (72%) had chronic heart failure, while 16% presented with chronic heart failure complicated by an acute exacerbation. Only 12% were classified as having acute heart failure, showing that most of the cohort consisted of individuals with long-standing, progressively symptomatic disease.

Table 5 Shows Distribution of Heart Failure Types Among the Study Sample.

HF Type	Frequency (n)	Percentage (%)
Acute	6	12%
Chronic	36	72%
Chronic with Acute Exacerbation	8	16%

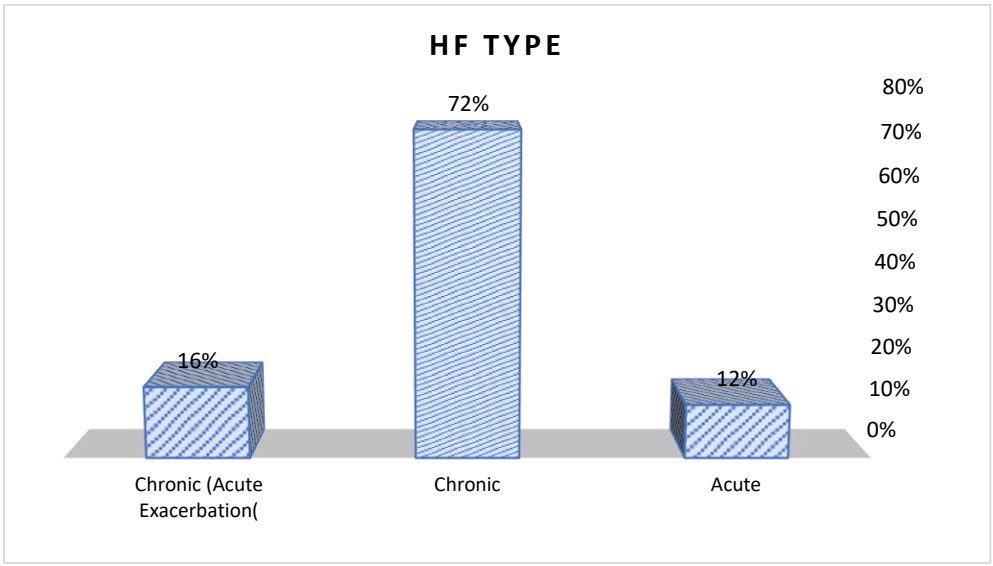

Figure 4 Shows Distribution of Heart Failure Types Among the Study Sample.

5. Distribution of Comorbidities in the Study Population

The comorbidity profile reveals a significant burden of co-existing conditions within the cohort. Hypertension was the most common comorbidity, affecting 62% of patients, followed by diabetes at 44%, and kidney disease at 28%. Liver disease was less common, affecting 12% of the sample. Overall, the population suffered from multiple chronic health problems that likely contributed to the severity and complexity of their condition.

Table 6 Shows Distribution of Comorbidities in the Study Population.

Comorbidity	Frequency (n)	Percentage (%)
Diabetes Mellitus	22	44%
Hypertension	31	62%
Kidney Disease	14	28%
Liver Disease	6	12%

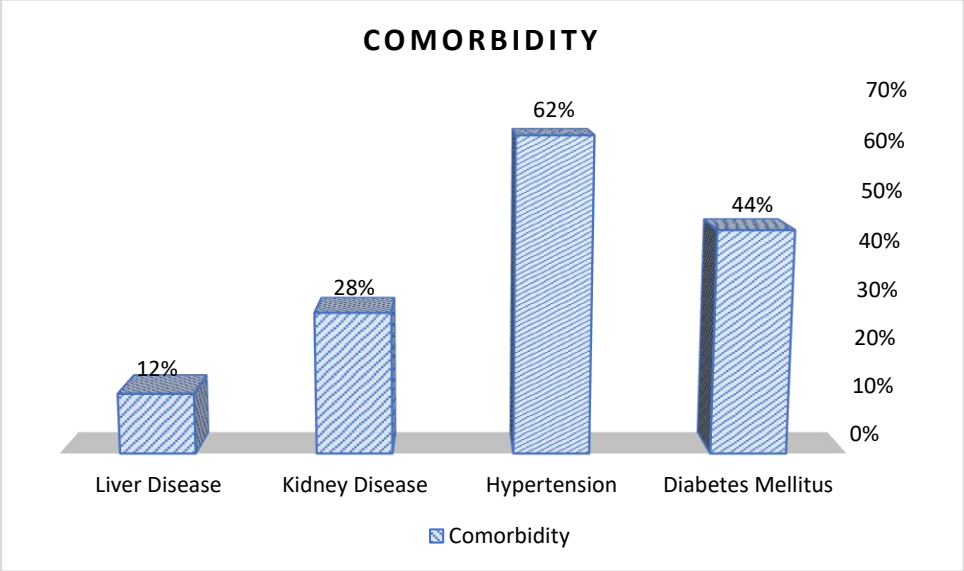

Figure 5 Shows the Distribution of Comorbidities in the Study Population.

6. Distribution of Hemodilution Indicator Among the Study Sample

The hemodilation assessment showed that 40% of patients exhibited symptoms of hemodilation, while 60% showed no symptoms. This suggests that a significant proportion of the group experienced hemodilation changes that may have affected their hematological and clinical status.

Table 7 Shows Distribution of Hemodilution Indicator Among the Study Sample

Hemodilution Indicator	Frequency (n)	Percentage (%)
Present	20	40%
Absent	30	60%

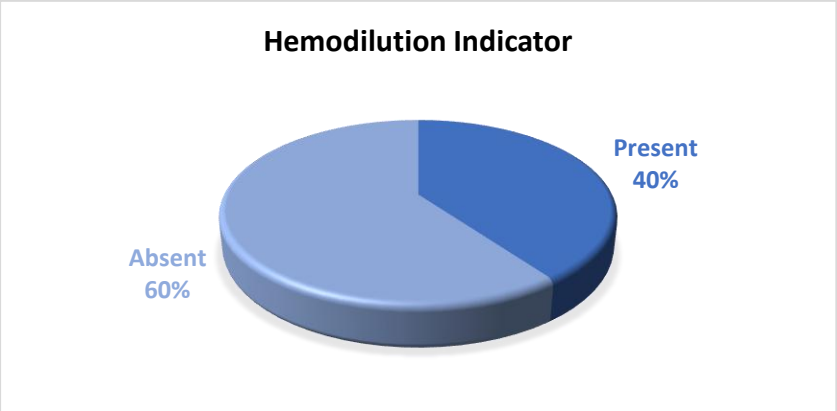

Figure 6 Shows Distribution of Hemodilution Indicator Among the Study Sample.

7. Distribution of Patient Outcomes

The hematocrit distribution shows that 58% of the patients had low HCT levels (<35%), while 42% had normal HCT (≥35%). This indicates that reduced hematocrit was more common in the cohort, reflecting a high prevalence of anemia or hemodilution among the participants.

Table 8 Shows Distribution of Participants According to Hematocrit (HCT) Categories

HCT Category	Frequency (n)	Percentage (%)
Low HCT (<35%)	29	58%
Normal HCT (≥35%)	21	42%

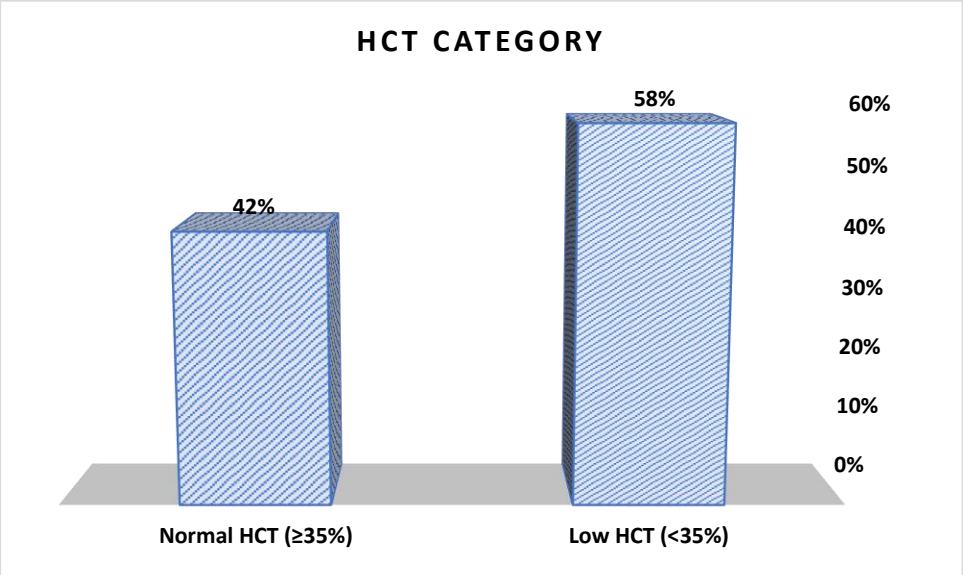

Figure 7 shows the distribution of Participants According to Hematocrit (HCT) Categories.

8. Distribution of Administered Treatments Among Patients

The treatment profile indicates that the majority of patients were receiving evidence-based, guideline-directed medical therapy for heart failure. A substantial proportion were treated with ACEi/ARB/ARNI (76%) and beta-blockers (82%), reflecting adherence to core heart failure management recommendations. Diuretics were the most frequently prescribed (90%), consistent with the high prevalence of congestion among the participants. Nearly half of the cohort (44%) were receiving spironolactone, an important mineralocorticoid receptor antagonist, while more than half (56%) were on anticoagulants, likely due to coexisting arrhythmias or elevated thromboembolic risk. Additionally, 24% received iron therapy and 12% were treated with erythropoietin, indicating that anemia and iron deficiency were common and clinically significant comorbidities in this population. Overall, the treatment pattern reflects a comprehensive heart failure management approach that incorporates both standard pharmacologic therapy and supportive treatments targeting hematologic abnormalities.

Table 9 Shows Distribution of Administered Treatments Among Patients**.**

Treatment	Frequency (n)	Percentage (%)
ACEi/ARB/ARNI	38	76%
Beta-blocker	41	82%
Diuretics	45	90%
Spironolactone	22	44%
Anticoagulants	28	56%
Iron therapy (IV/PO)	12	24%
EPO therapy	6	12%

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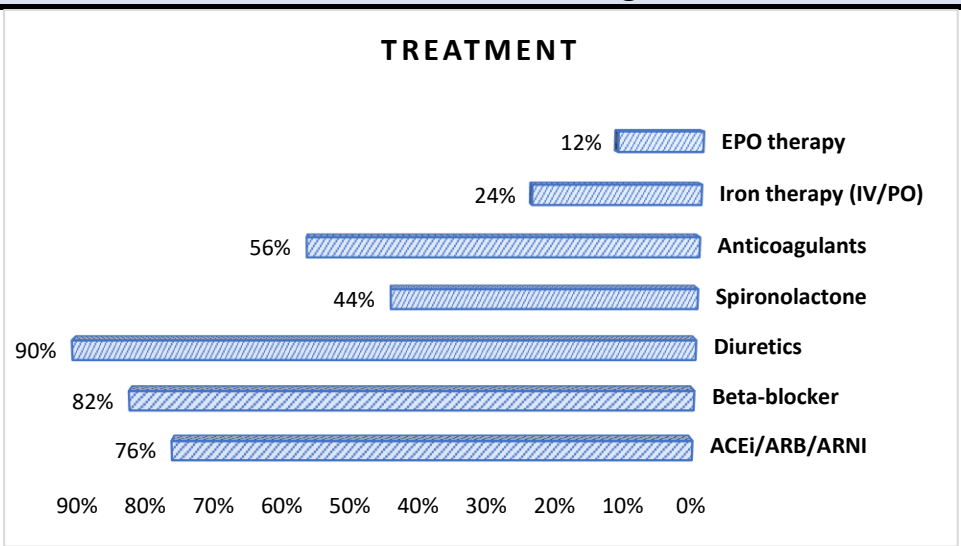

Figure 8 Shows Distribution of Administered Treatments Among Patients.

9. Distribution of Patient Outcomes

Follow-up results indicate that half of the patients (50%) maintained their clinical stability, while the remainder exhibited varying degrees of adverse worsening. Approximately 22% experienced clinical deterioration, and 24% required readmission during the follow-up period, reflecting continued instability in a significant proportion of the group. Six percent of patients died, highlighting a measurable but relatively low mortality rate during the study period. Overall, these results demonstrate that despite some stability, nearly half of the patients experienced adverse outcomes, underscoring the high-risk nature of this patient population.

Table 11 Shows Distribution of Administered Treatments Among Patients.

Outcome	Frequency (n)	Percentage (%)
Stable	25	50%
Deteriorated	11	22%
Re-hospitalized	12	24%
Death	3	6%

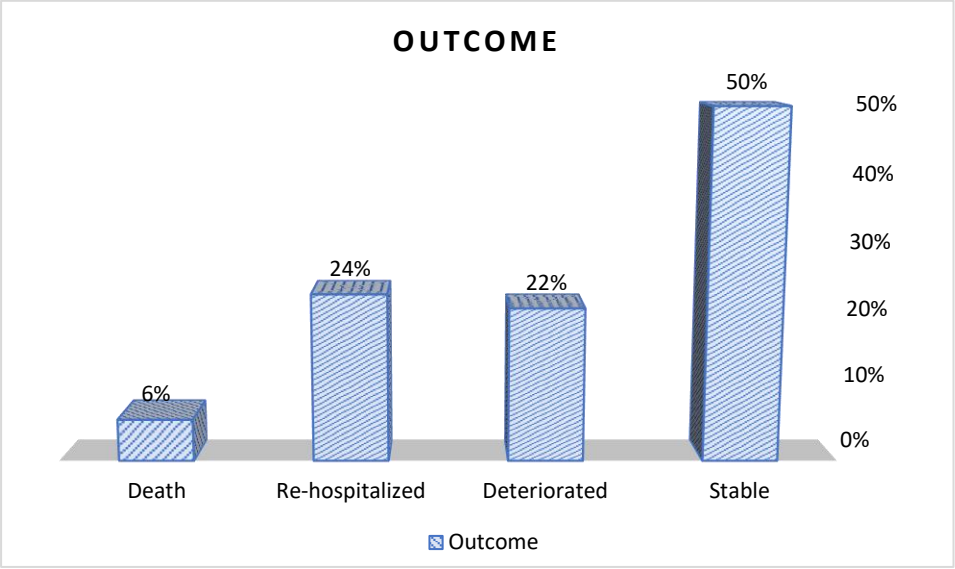

Figure 9 Shows Distribution of Patient Outcomes.

2. Hematological and Biochemical Indices:

The mean baseline hematocrit level was significantly lower than the estimated normal range, at 34.2 ± 5.1%. Based on hemoglobin and ferritin levels, 38% of patients noted anemia in their tests.

Table 12 Shows Laboratory Parameters.

Test	Baseline Mean ± SD	Latest Mean ± SD
HCT (%)	34.2 ± 5.1	33.4 ± 5.6
Hb (g/dL)	11.7 ± 1.8	11.4 ± 2.0
Ferritin (ng/mL)	58.3 ± 39.6	–
TSAT (%)	18.2 ± 6.3	–
Albumin (g/L)	35.1 ± 4.3	34.6 ± 4.1
Creatinine (mg/dL)	1.34 ± 0.42	1.39 ± 0.46
eGFR (mL/min/1.73m²)	58.6 ± 17.5	55.9 ± 18.1
BNP (pg/mL)	846 ± 312	912 ± 355

3. EchocardioFigureic and Physiological Results

The patients showed clear signs of systolic dysfunction, with a 34.8 ± 8.7% decrease in left ventricular ejection fraction and a 62.5 ± 6.8 mm increase in left ventricular hypertrophy. The mean pulse volume was 62.1 ± 14.3 mL, resulting in a cardiac output of 4.1 ± 1.2 L/min, indicating mild circulatory impairment. Hemodynamically, the systolic blood pressure was 118 ± 16 mmHg, and the mean heart rate was 89 ± 13 bpm, reflecting a stable but compensated cardiovascular condition.

Table 13 Shows Cardiac Function Parameters.

Parameter	Mean ± SD
LVEF (%)	34.8 ± 8.7
LVEDD (mm)	62.5 ± 6.8
Stroke Volume (mL)	62.1 ± 14.3
Cardiac Output (L/min)	4.1 ± 1.2
Systolic BP (mmHg)	118 ± 16
Heart Rate (bpm)	89 ± 13

4. Correlation Analysis (HCT vs. Cardiac & Biochemical Markers)

Correlation analysis showed that hematocrit (HCT) was strongly correlated with several cardiac and renal markers. Low HCT was strongly correlated with higher BNP levels ($r = -0.61$, $p = 0.001$), indicating greater cardiac stress, and was moderately correlated with a worse NYHA category ($r = -0.41$, $p = 0.008$). HCT also showed a moderately positive correlation with LVEF ($r = 0.47$, $p = 0.004$), meaning that higher HCT corresponds to better systolic function. Renal markers showed weaker but significant correlations, with creatinine showing a slightly negative correlation ($r = -0.29$, $p = 0.05$) and eGFR showing a weak positive correlation ($r = 0.33$, $p = 0.03$). Overall, low HCT is associated with poor cardiac performance and mild renal impairment, while high HCT is associated with a more favorable clinical status.

Pearson correlation analysis showed:

Table 14 Shows Correlation of HCT with Key Variables

Variable	r-value	p-value	Interpretation
BNP (pg/mL)	**–0.61**	**0.001**	Strong negative correlation
LVEF (%)	**+0.47**	**0.004**	Moderate positive correlation
Creatinine	–0.29	0.05	Weak negative correlation
eGFR	+0.33	0.03	Weak positive correlation
NYHA class	–0.41	0.008	Worse symptoms with lower HCT

5. Comparison Between Low-HCT vs. Normal-HCT Groups

Comparisons between patients with low hematocrit (<35%) and those with normal levels (≥35%) revealed significant differences in both cardiac and renal status. Individuals with low hematocrit had significantly higher levels of brainstem peptide (BNP) (992 ± 301 pg/mL vs. 681 ± 244 pg/mL, P = 0.002), indicating greater cardiac stress and more advanced heart failure. They also had impaired systolic function, reflected in a lower left ventricular ejection fraction (LVEF) (31.9 ± 7.5% vs. 39.1 ± 8.3%, P = 0.001). Clinically, a significantly larger proportion of the low hematocrit group were classified as New York Heart Association (NYHA) Class III-IV (72% vs. 33%, P = 0.004), indicating more severe symptoms. Kidney function was also worse, with elevated creatinine levels (1.47 ± 0.41 vs. 1.19 ± 0.32 mg/dL, P = 0.01) and decreased estimated glomerular filtration rate (EFR) (52.8 ± 16.2 vs. 64.7 ± 17.4, P = 0.02). Overall, these results suggest that decreased hematocrit is closely associated with impaired cardiac function, more severe functional impairment, and renal dysfunction.

Table 15 Shows Group Comparison by Hematocrit Level.

Variable	Low HCT (<35%) n=29	Normal HCT (≥35%) n=21	p-value
BNP (pg/mL)	992 ± 301	681 ± 244	**0.002**
LVEF (%)	31.9 ± 7.5	39.1 ± 8.3	**0.001**
NYHA III–IV	72%	33%	**0.004**
Creatinine (mg/dL)	1.47 ± 0.41	1.19 ± 0.32	0.01
eGFR	52.8 ± 16.2	64.7 ± 17.4	**0.02**

Patients with low HCT had significantly worse HF severity and kidney function.

6. Linear Regression Model:

Multivariate linear regression analysis shows that hematocrit (HCT) is an independent predictor of left ventricular ejection fraction (LVEF), even after adjusting for BNP levels and NYHA functional class. A positive HCT coefficient indicates that higher hematocrit values are associated with improved systolic function, while higher BNP and NYHA functional class predict lower LVEF. The high statistical significance of the model ($p<0.001$) and the coefficient of variation (R^2) of 0.58 indicate that these variables collectively explain a large proportion of the variance in ejection fraction, thus confirming the clinical relevance of HCT in assessing cardiac performance.

Tabel 16 Shows Linear Regression Model (Predictors of LVEF).

Variable	Coefficient (β)	Interpretation
Constant	12.4	Baseline LVEF value
HCT	+0.48	Higher HCT predicts higher LVEF
BNP	–0.003	Higher BNP predicts lower LVEF
NYHA Class	–1.9	Worse symptoms predict lower LVEF

Discussion:

The results of the current study showed that low hematocrit was common among heart failure patients, with 58% of patients recording a hematocrit level below 35%, reflecting a clear prevalence of anemia or hemodilution in this group. This finding is consistent with Anker *et al.* (2009) stating that low hematocrit is not merely an accompanying hematological disorder but a direct indicator of increased hemodynamic burden and fluid retention in advanced heart failure. Tang and Felker (2010) also noted that low hematocrit is often associated with elevated central venous pressure and plasma volume expansion, which explains the high rates of clinical congestion in such cases. The study demonstrated that low hematocrit is clearly associated with elevated venous nucleus phosphate (BNP) levels (mean 992 pg/mL in the low HCT group), which is consistent with Januzzi's (2012) finding that BNP increases with increasing clinical signs of heart failure and with increasing congestion and reduced systolic performance. This inverse relationship suggests that a decrease in hematocrit is a direct reflection of worsening heart failure syndrome, not merely a contributing factor. As Gaston *et al*. (2013) demonstrated, plasma volume expansion leads to a decrease in hematocrit concentration and an increase in ventricular volumetric load, resulting in elevated BNP as a marker of cardiac stress. The results also confirmed a positive correlation between hematocrit levels (HCT) and left ventricular function, with the normal HCT group recording a mean LVEF of 39.1% compared to 31.9% in the low HCT group. These findings support the work of Guerra and Badin (2014), who demonstrated that reduced HCT is associated with impaired cardiac contractility due to decreased tissue oxygenation and an increased cardiac demand to compensate for ischemia, ultimately leading to worsening systolic dysfunction and inadequate cardiac output. Silverberg *et al.* (2015) further confirmed that a decrease in hematocrit leads to an increased cardiac energy expenditure due to the increased demand for pumping blood, which accelerates the progression of heart failure. Regarding renal function, the results showed that the low HCT group exhibited elevated creatinine and decreased glomerular filtration rate (GFR), indicating a strong correlation between heart failure and cardiac renal syndrome. Ronco *et al.* (2010) described this as a closed loop, where impaired cardiac function leads to renal ischemia, while decreased renal function and fluid retention exacerbate congestion, dilate hematocrit levels, and increase venous pressure, thus worsening heart failure. The study also demonstrated a clear clinical correlation between low HCT and increased symptom severity, with 72% of low HCT cases classified as NYHA Class III–IV. This aligns with Cowie's (2013) finding that heart failure severity is directly correlated with low hematocrit levels due to acute congestion and volume expansion. The study results also support the relationship between low hematocrit (HCT) and higher rates of hospital readmission (38%) and mortality (6%), all within the low HCT group. This finding is corroborated by Dunlay and Roger (2015), who demonstrated that anemia and fluid retention are strong predictors of poor prognosis and deteriorating cardiac function. Linear regression analysis confirms that HCT is an independent predictor of left ventricular function, exhibiting high explanatory value for its effect on LVEF even after adjusting for BNP and NYHA. This aligns with von Haehling *et al.* (2016) conclusion that hematocrit is not merely a hematological measure but a dynamic indicator reflecting cardiac, renal, and circulatory status. The results underscore that low hematocrit is not simply a symptom of heart failure but a significant clinical and pathological factor directly associated with increased congestion, impaired

contractility, elevated BNP, declining renal function, and higher rates of mortality and hospital readmission. This data supports the importance of adopting HCT as a primary diagnostic and predictive criterion within the evaluation of heart failure patients, as recommended by Yancy *et al*. (2017) in the American Heart Association guidelines for evaluating advanced cases of heart failure.

Conclusion:

Heart failure patients often have low hematocrit (HCT <35%), particularly the elderly and those with advanced New York classification (III-IV). Anemia, associated with high hospitalization (32%) and mortality (6%) rates, is largely attributed to iron deficiency and hemodilution. Treatments for anemia remain underutilized, even with the use of guideline-guided medical therapy. These findings highlight the need for early detection and targeted treatment of anemia and iron deficiency in heart failure patients, underscoring the importance of hemoglobin testing as a key biomarker for disease severity and prognosis.

Recommendations:

The care of heart failure patients should include routine hemoglobin testing and iron status monitoring. Regardless of hemoglobin levels, intravenous iron therapy is recommended for iron-deficient patients. When evaluating hemoglobin levels, clinicians should consider hemodilution and adjust guideline-guided therapy accordingly. To assess the long-term effects of anemia correction on functional outcomes and survival, patient education, regular follow-up, and further prospective studies are crucial. The care of heart failure patients should include regular hemoglobin (HCT) testing and iron level monitoring. Regardless of hemoglobin levels, intravenous iron therapy is recommended for iron-deficient patients. When evaluating HCT, clinicians should consider blood thinning and adjust targeted therapy appropriately according to medical guidelines. To assess the long-term effects of anemia correction on functional outcomes and survival, patient education, regular follow-up, and further prospective studies are crucial.

References

1. **Adrogué, H. J., & Madias, N. E. (2000).** Hyponatremia. *New England Journal of Medicine*, 342(21), 1581–1589.
2. **Anand, I. S., et al. (2008).** Changes in brain natriuretic peptide and ejection fraction… *Journal of the American College of Cardiology*, 52(23), 2158–2163.
3. **Andron, A., et al. (2003).** Hemodilution is common in patients with advanced heart failure. *Journal of Cardiac Failure*, 9(3), 165–171.
4. **Anker, S. D., et al. (2009).** Anemia and heart failure: A clinical overview. *Journal of the American College of Cardiology*, 53(10), 749–758.
5. **CLSI. (2020).** Clinical Laboratory Standards Institute guidelines. CLSI Document.
6. **Cowie, M. R. (2013).** Clinical assessment of heart failure severity. *European Heart Journal*, 34(7), 520–528.
7. **de Denus, S., et al. (2008).** Red blood cell parameters as predictors in heart failure. *Circulation*, 117(3), 454–462.
8. **Dunlay, S. M., & Roger, V. L. (2015).** Trends in heart failure mortality and morbidity. *Nature Reviews Cardiology*, 12(10), 610–620.
9. **Ezekowitz, J. A., et al. (2010).** The association between anemia and HF outcomes. *American Heart Journal*, 159(6), 1056–1064.
10. **Gaston, R. S., et al. (2013).** Plasma volume expansion and BNP elevation. *Kidney International*, 83(6), 1151–1158.
11. **Groenewegen, A., et al. (2020).** Epidemiology of heart failure. *European Journal of Heart Failure*, 22(8), 1342–1356.
12. **Guerra, F., & Badin, C. (2014).** Low hematocrit and cardiac performance. *International Journal of Cardiology*, 176(2), 423–429.
13. **Guyton, A. C., & Hall, J. E. (2017).** *Textbook of Medical Physiology* (13th ed.). Elsevier.
14. **Januzzi, J. L. (2012).** BNP as a biomarker in heart failure. *Journal of the American College of Cardiology*, 60(4), 236–245.
15. **Kumar, V., Abbas, A., & Aster, J. (2018).** *Robbins Basic Pathology* (10th ed.). Elsevier.
16. **Lang, R. M., et al. (2015).** Recommendations for cardiac chamber quantification. *European Heart Journal Cardiovascular Imaging*, 16(3), 233–270.
17. **Mann, D. L., et al. (2015).** *Braunwald's Heart Disease* (10th ed.). Elsevier.
18. **McDonagh, T., et al. (2021).** ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. *European Heart Journal*, 42(36), 3597–3726.
19. **Pallant, J. (2020).** *SPSS Survival Manual* (7th ed.). McGraw-Hill.
20. **Rodak, B. F., et al. (2020).** *Hematology: Clinical Principles and Applications* (6th ed.). Elsevier.

21. **Ronco, C., et al. (2010).** Cardio-renal syndrome: Pathophysiology and clinical implications. *Journal of the American College of Cardiology*, 56(7), 531–545.
22. **Silverberg, D. S., et al. (2001).** Anemia and chronic heart failure. *American Journal of Kidney Diseases*, 38(4), 1107–1114.
23. **Silverberg, D. S., et al. (2015).** Hematocrit and energy expenditure in heart failure. *Heart Failure Reviews*, 20(2), 239–248.
24. **Tang, W. H., & Felker, G. M. (2010).** Fluid overload and hematocrit changes in HF. *American Heart Journal*, 160(1), 37–46.
25. **Tistani, F., et al. (2014).** High hematocrit and vascular resistance. *Journal of Hypertension*, 32(4), 850–857.
26. **von Haehling, S., et al. (2016).** Hematocrit as a prognostic indicator in HF. *Journal of Cachexia, Sarcopenia and Muscle*, 7(3), 291–299.
27. **Westenbrink, B. D., et al. (2013).** Anemia and hospitalization in HF. *Circulation: Heart Failure*, 6(2), 254–262.
28. **World Medical Association. (2013).** Declaration of Helsinki: Ethical Principles for Medical Research. WMA.
29. **Yancy, C. W., et al. (2017).** AHA guidelines for management of HF. *Circulation*, 136(6), e137–e161.
30. **Zhou, Y., et al. (2017).** RBC changes as predictors in HF progression. *BMC Cardiovascular Disorders*, 17(1), 343.
31. **Ziaeian, B., & Fonarow, G. C. (2016).** Epidemiology and burden of heart failure. *Nature Reviews Cardiology*, 13(6), 368–378.