“CORRELATION OF RED CELL DISTRIBUTION WIDTH WITH THE SEVERITY OF HEART FAILURE WITHOUT ANEMIA ”

BY

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THESIS MANUSCRIPT SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA

In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

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The Institutional Ethics Committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has examined and unanimously approved the Synopsis entitled **"Correlation of RDW with the Severity of Heart Failure without Anemia"** being investigated by Dr.HEMANTH KUMAR REDDY K & Dr. Prabhakar K in the Department of General Medicine at Sri Devaraj Urs Medical College, Tamaka, Kolar. **Permission is granted by the Ethics Committee to start the study.**

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ACKNOWLEDGEMENT

No academic work is single handedly accomplished. This work is no exception. Words fail me in expressing my heartfelt and humble gratitude to thank the almighty for showering his blessings on me. I sincerely thank my guide, **DR. PRABHAKAR K,** who have the substance of a genius and for their step-by-step guidance and constant extended support with the timely advices which helped me for this study. Their encouragement, sense of punctuality, research-oriented approach, the painstaking effort to weed out errors and their affection during the entire course of study leaves me permanently indebted to them.

I express my deep sense of gratitude and humble thanks to my senior Professors, **DR. RAVEESHA. A, DR. B.N. RAGHAVENDRA PRASAD, DR. VIDYASAGAR. C. R**, **DR. SRINIVAS. S. V** for their advice and encouragement throughout the study.

I am extremely indebted to and I thank **DR. VISHWANATHA REDDY. N, DR. KARTHIK. V, DR. MANJUNATH, DR ANITHA A**, Department of General Medicine for their constant support and encouragement throughout with patience and care. They have been my well-wisher and a source of inspiration throughout my study.

My heartfelt thanks to all my teachers throughout my life for having made me what I am today for their practical tips, invaluable advice and constant encouragement.

I extend my sincere thanks to my seniors **DR. SHASHI SHEKAR, DR. KISHORE, DR. DEEPTHI,** whose knowledge has guided and inculcated in me a sense of confidence. I am thankful for their valuable guidance and helping me with my dissertation.

I express my sincere thanks to my colleagues and dear friends **DR. APARNA C, DR. ATHISHAYA GV, DR. RAKESH G, DR. DHEERAJ A, DR. JAVERIA A, DR. DEEPAK D.** for their co-operation and help in carrying out this study.

I would express my deepest gratitude to my beloved grandparents **K. SUBBA REDDY** and **K. RAJAMMA, T. VENKAYAMMA** and my parents, **K. KESAVA REDDY** and **K. SITA**, whose love, blessings and sacrifices made me the person I am today. Without them I would have never reached to this level.

I am very much thankful to my wife **DR. SHALINI REDDY,** sister **K. SNEHA REDDY**, and my brother **K. VEDA KIRAN REDDY** and to all my beloved friends for their love and support towards me.

I thank all my interns and nurses of ICU, MICU and General ward for their help and assistance.

Last, but not the least, I thank my patients for providing me the opportunity to carry out my study.

DR. K. HEMANTH KUMAR REDDY

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LIST OF ABBREVIATIONS

Glossary	Abbreviations
6MWT	6-minute walk test
ACHD	Adult congenital heart disease
AHF	Antihemophilic factor
AR	Aortic regurgitation
AS	Ankylosing spondylitis
BMI	Body mass index
BNP	B-type natriuretic peptide
CAD	Coronary artery disease
CHF	Congestive heart failure
CI	Confidence interval
CV	Cardiovascular
DBP	Diastolic blood pressure
DHF	Diastolic heart failure
eGFR	Estimated glomerular filtration rate
GFR	Glomerular filtration rate
HbA1c	Hemoglobin A1c
HFimpEF	Heart failure with improved ejection fraction
HFmrEF	Heart failure with a mildly reduced ejection fraction
HFpEF	Heart failure with a preserved ejection fraction
HFREF	Heart failure with a reduced ejection fraction
LVEF	Left ventricular ejection fraction
NT-pro BNP	N-terminal pro-BNP
NYHD	New York heart association
RBC	Red blood cells
RDW	Red cell distribution width
SBP	Systolic blood pressure

ABSTRACT

BACKGROUND: Anisocytosis is measured by RDW, or "red cell distribution width." Increased RWD levels have been linked to negative outcomes in a variety of diseases, including chronic heart failure.

AIMS: To determine the correlation of “RDW” & severity of heart failure without anemia.

MATERIALS&METHODS: Prospective Observational Study over a period of 1 year from January 2020 to January 2021. The study had a total of 55 participants.

RESULTS: The average age was 54.15 ± 10.09 years. Males accounted for the majority of the cases, being 72.73%. “Coronary artery disease” & “ischemic heart disease” were the common etiologies identified in the study population with 49.09% and 45.45%. The mean RDW was 15.07 ± 1.52. The majority of the participants had NYHD class III with 34.55%, followed by class IV, II, and I with 27.27%, 21.82%, and 16.36%, respectively. LVEF was <30% in the majority of patients with 30.91%, followed by 30-44% with 29.09%. The mean RDW in NYHA classes I, II, III, and IV were identified with 13.26 ± 0.72, 13.88 ± 0.99. 15.24 ± 0.68 and 16.89 ± 0.53 respectively.

CONCLUSION: RDW can be used to forecast the likelihood of heart failure occurring and progressing.

INTRODUCTION

INTRODUCTION:

Heart failure is the inability of the heart to maintain an output which is adequate to meet the required metabolic demands of the body. Heart failure is caused by ischemic heart disease, diabetes, atrial fibrillation, hypertension, chronic renal disease, and valvular heart disease.[1]

In affluent countries, the prevalence of failure of heart in adults is between 1 and 2%. It has risen to more than ten percent among persons aged 70 and up. The causes of cardiac infarction are more, and these can differ in different parts of the world. A poor ejection fraction is found in half of all heart failure patients.[2]

The most common cause for the failure of the heart due to low ejection fraction is coronary artery disease. “Hypertension and diabetes” are considered as the contributing factors in the vast majority of cases. Other causes of HFREF consists of previous viral infection, alcohol abuse, chemotherapy, and idiopathic dilated cardiomyopathy.[2]

Failure of the heart with a "preserved ejection fraction" is known as diastolic heart failure. In comparison to HFREF, also known as systolic heart failure, these individuals are older, more typically female, and obese. They are less likely to develop coronary heart disease whereas more likely to develop hypertension and atrial fibrillation. Patients with HFPEF have a better prognosis as compared with those with HFREF.[3]

Heart failure patients with neurohormonal imbalances, low ejection fractions, ventricular arrhythmias, intraventricular conduction delays, reduced functional capacity, low SBP, and renal failure have a bad prognosis.[4]

RDW is the quantitative measure of anisocytosis. Higher values of RDW show that the size of red blood cells in a given sample of blood fluctuates more. The anemic differential diagnosis is narrowed by RDW. Generally, to differentiate iron deficiency anemia & thalassemia, RDW is more useful. The link between RDW and cardiovascular illnesses such as coronary artery disease and heart failure has recently gotten a lot of attention.[5] Red cell distribution width has newly been discovered as a novel prognostic marker in heart failure patients.

NEED OF THE STUDY:

Anisocytosis is measured by RDW. Higher RDW levels have been associated to unfavorable outcomes in a variety of diseases, including chronic heart failure. The role of RBC dispersion width for the prediction of "heart failure" has been mentioned in several studies, but the results of those studies are conflicting. The goal of the study was to discover if there was a link between the width of RBC dispersion and the severity of heart failure without anemia.

AIMS & OBJECTIVES

AIMS AND OBJECTIVES:

- To evaluate RDW in heart failure.
- To associate RDW with the severity of heart failure (NYHA functional class and LV ejection fraction).

REVIEW OF

LITERATURE

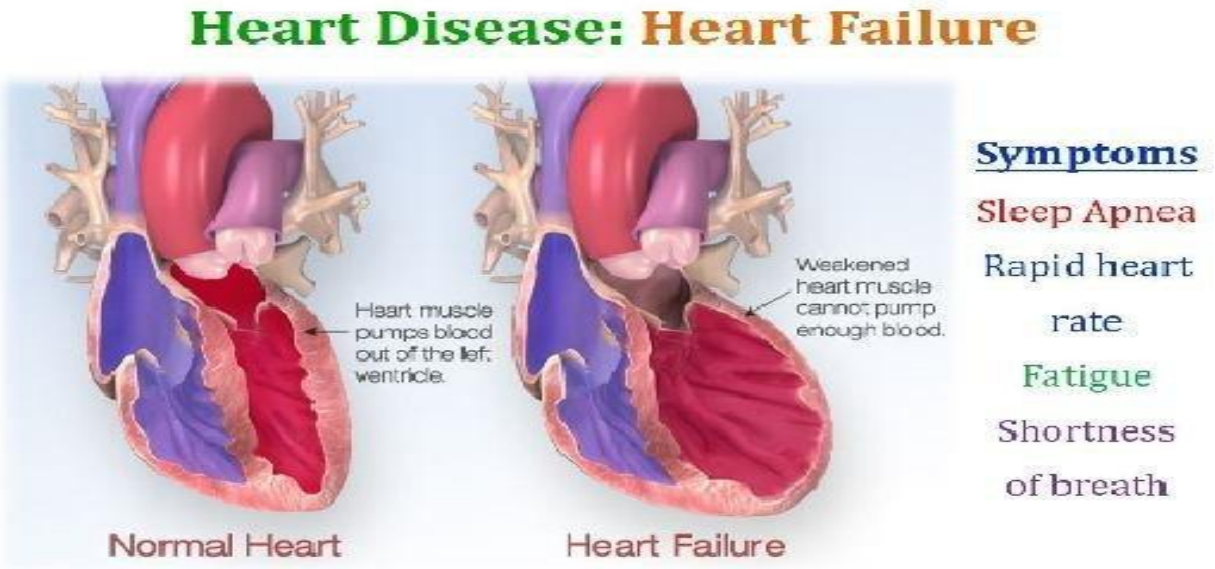
REVIEW OF LITERATURE:

1. Heart failure

a) Definition, classification

Heart failure is a complex clinical disease characterized by the heart's inability to perform its circulatory function as efficiently as it could due to structural and functional changes.

Figure 1: Heart failure.[6]



The following is a revised classification of heart failure based on the left ventricular ejection fraction:[7]

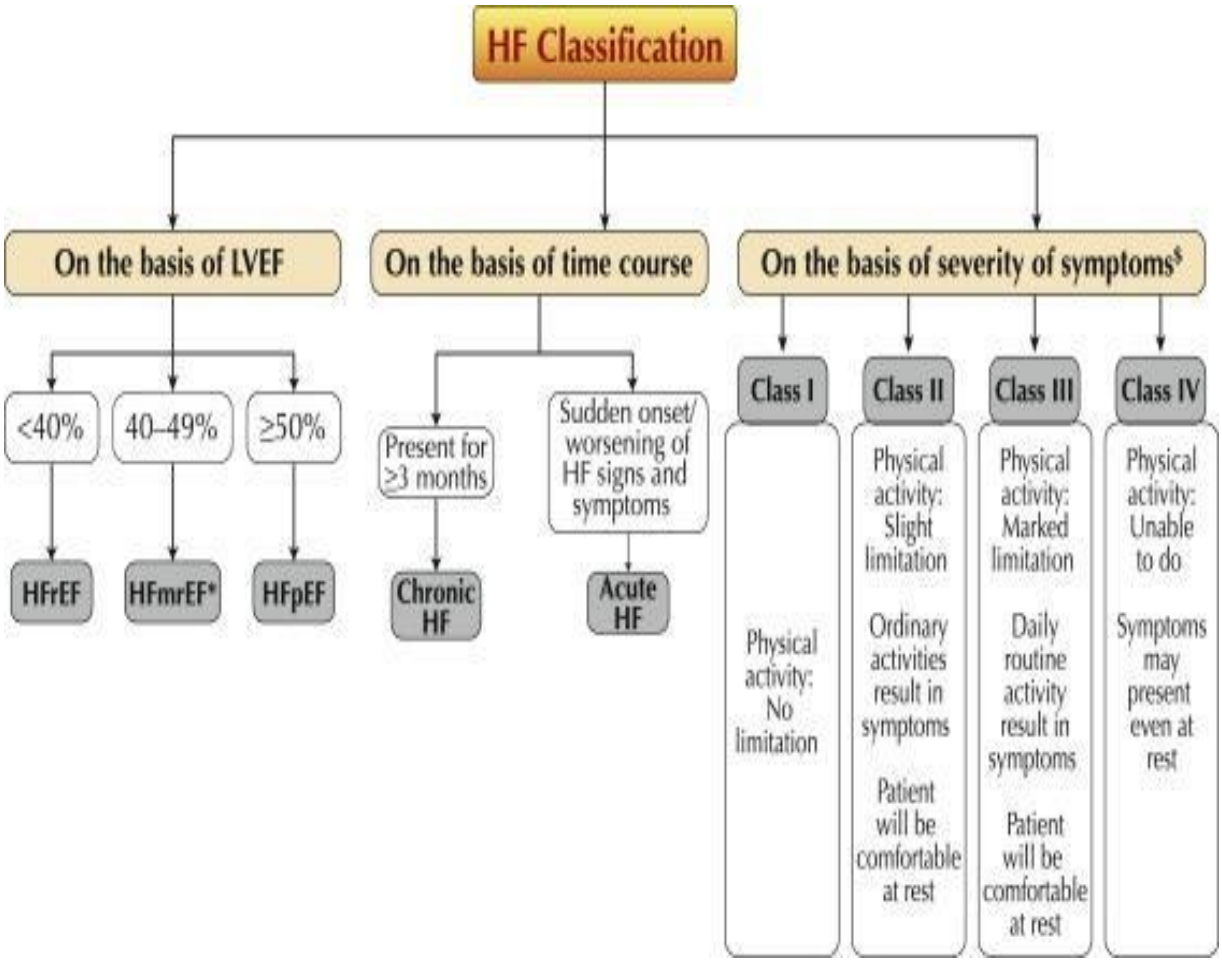
a. **"Heart failure with reduced ejection fraction (HFrEF)"**: Symptomatic heart failure is defined as an LVEF of less than 40%.

b. **"Heart failure with mildly reduced ejection fraction (HFmrEF")** – Symptomatic heart failure with a left ventricular ejection fraction (LVEF) of 41-49 percent.

c. **"Heart failure with preserved ejection fraction (HFpEF")** – This is symptomatic heart failure with a left ventricular ejection fraction (LVEF) of less than 50%.

d. **"Heart failure with improved ejection fraction (HFimpEF)"** - Defined as symptomatic heart failure with a baseline LVEF of 40%, an increase of 10% from baseline. The second measurement of LVEF >40% is known as the second measurement of LVEF.

Figure 2: Heart failure classification.[1]

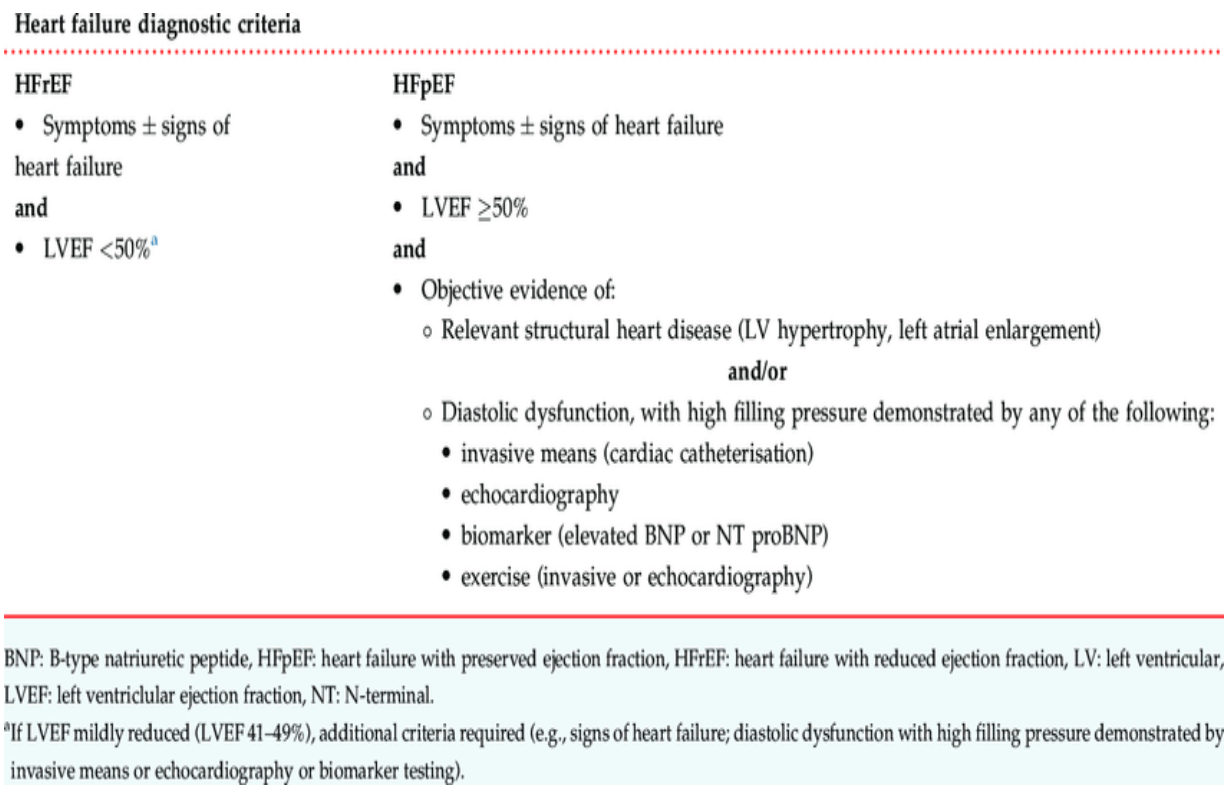


b) Epidemiology

The prevalence of failure of the heart is identified as >37.7 million globally. By the year 2030, the number of heart failure cases could increase by 25%. The longstanding risk of heart failure by the age of 55 years is observed as 33% for males and 28.5% for females.[1] Heart failure is common in developed countries, with rates ranging from 1% to 2%.[8]

c) Criteria for diagnosis

Figure 3: Criteria for diagnosis.[9]



Heart failure diagnostic criteria

HFrEF	HFpEF
• Symptoms ± signs of heart failure and • LVEF <50%[a]	• Symptoms ± signs of heart failure and • LVEF ≥50% and • Objective evidence of: ○ Relevant structural heart disease (LV hypertrophy, left atrial enlargement) **and/or** ○ Diastolic dysfunction, with high filling pressure demonstrated by any of the following: • invasive means (cardiac catheterisation) • echocardiography • biomarker (elevated BNP or NT proBNP) • exercise (invasive or echocardiography)

BNP: B-type natriuretic peptide, HFpEF: heart failure with preserved ejection fraction, HFrEF: heart failure with reduced ejection fraction, LV: left ventricular, LVEF: left ventriclular ejection fraction, NT: N-terminal.

[a]If LVEF mildly reduced (LVEF 41–49%), additional criteria required (e.g., signs of heart failure; diastolic dysfunction with high filling pressure demonstrated by invasive means or echocardiography or biomarker testing).

d) Types

Heart failure can be classified into three categories.[10]

- **Left-sided heart failure**: The most prevalent type of "heart failure" is this one in which the left ventricle of the heart is weak to pump adequate blood around the body, and as a result, the blood builds up in the pulmonary veins. Shortness of breath, trouble breathing, or coughing can be caused due to left-sided heart failure.
- **Right-sided heart failure**: Right ventricle of the heart no longer pumps an adequate amount of blood to the lungs. It causes the blood to build up in the veins. The increased pressure inside the veins can push fluid out of the veins into surrounding tissue and can lead to a build-up of fluid in the legs or, less commonly, in the genital area, organs, or the abdomen.

- **Biventricular heart failure**: Both sides of the heart are affected in biventricular heart failure. It causes the same symptoms as both left-sided and right-sided heart failure.

e) Clinical presentation

The following are the symptoms identified for heart failure:[1]

a. Dyspnea
b. Orthopnea
c. Paroxysmal nocturnal dyspnea
d. Reduced exercise tolerance
e. Fatigue and more time to recover post-exercise
f. Ankle swelling
g. Weight gain
h. Nocturia
i. Nocturnal cough
j. Puffy face in the morning
k. Bendopnea.

Heart failure symptoms include the following:

a. Elevated jugular venous pressure
b. Hepatojugular reflux
c. Third heart sound (gallop rhythm)
d. Clinically evident cardiomegaly
e. Tachycardia
f. Tachypnea
g. Hepatomegaly

h. Pleural effusion

i. Pedal edema

j. Rales

k. Mitral regurgitation

l. Cardiac murmur

m. Fourth heart sound

n. Diagnosis.

Figure 4: Algorithm for the diagnosis of heart failure.[11]

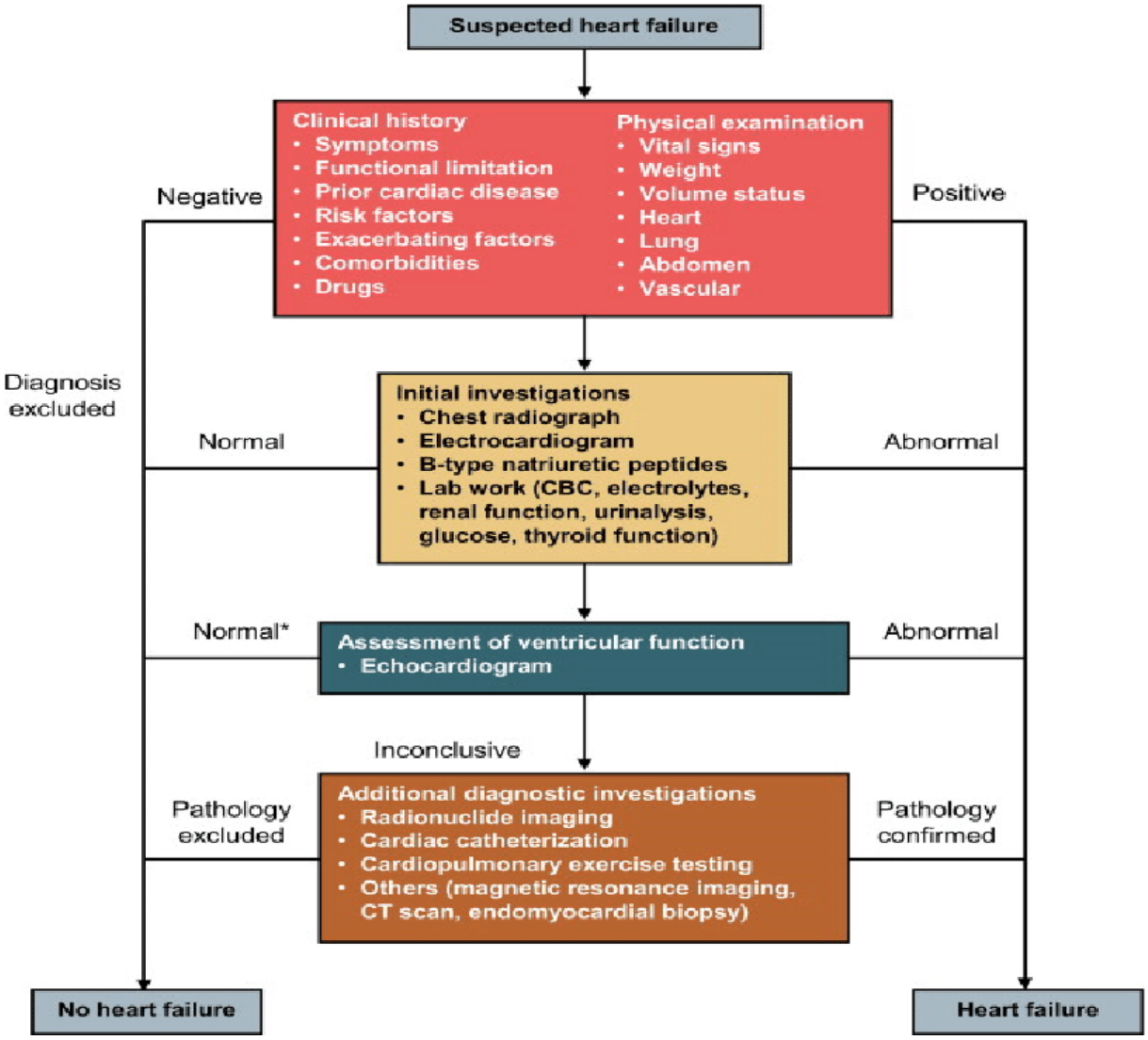
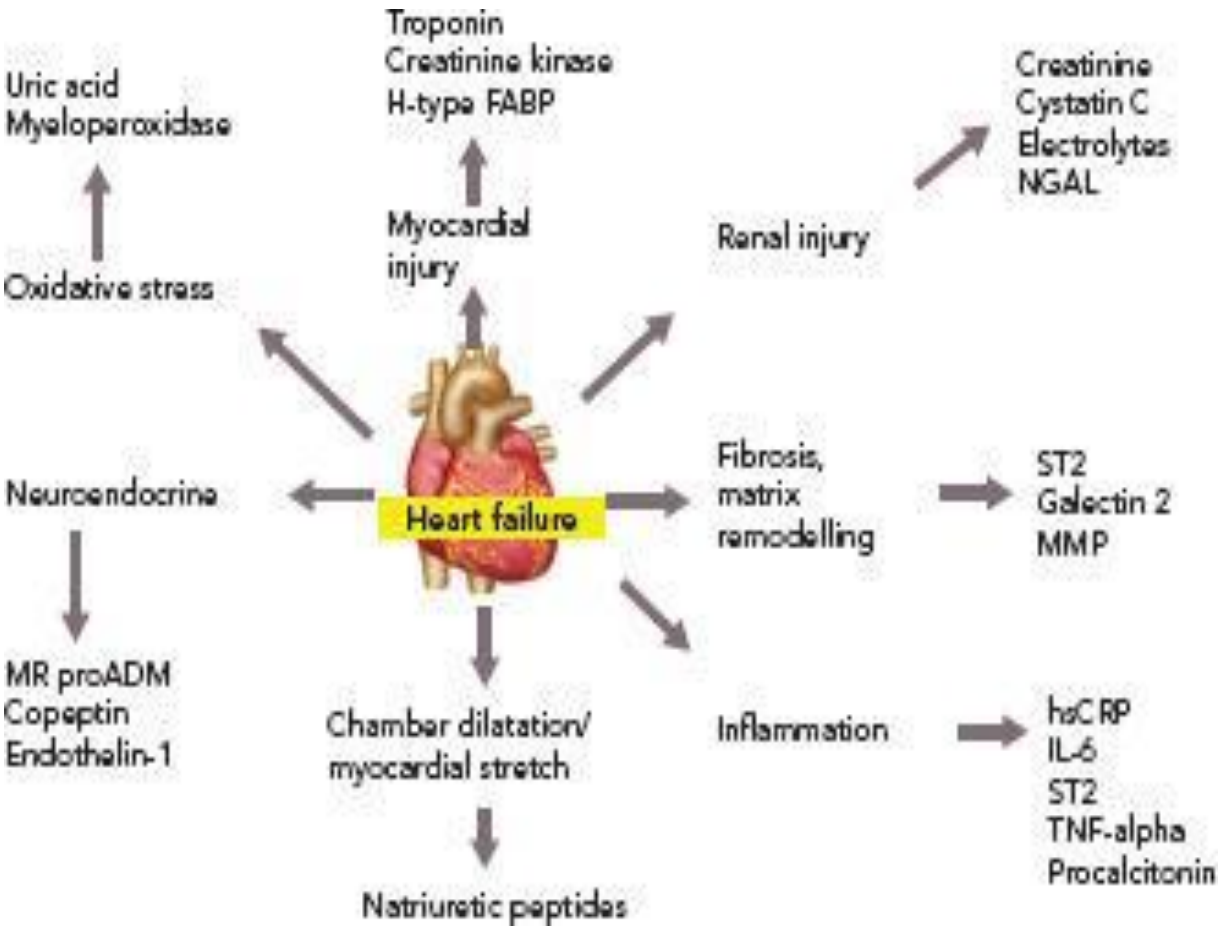


Figure 5: Different Potential Biomarkers in the "Diagnosis & Management" of Heart Failure.[12]



f) Risk factors, causes, and pathogenesis

Common risk factors in heart failure are the following:[1]

- Ischemic heart disease
- Diabetes mellitus
- Atrial fibrillation
- Chronic kidney disease
- Valvular heart disease
- Hypertension.

Figure 6: Causes of heart failure.[13]

TABLE 3. **Resume of the Main Etiological Factors in Heart Failure With the Most Common Examples Found in Clinical Practice***

Cause		Examples
Predisposing causes		
Etiological		Coronary artery disease, congenital heart disease
Probably etiological		AHT, diabetes, history of rheumatic fever
Non-etiological		Age, masculine sex, obesity, tobacco use
Determining causes		
Cardiomyopathy	Primary	Cardiomyopathy dilated, hypertrophic, and restrictive cardiomyopathy
	Secondary	Ischemic, infectious, toxic, and metabolic cardiomyopathy
Ventricular overload	Pressure	AHT, aortic/pulmonary stenosis, pulmonary hypertension
	Volume	Valve insufficiency, shunts
Altered ventricular filling		Ventricular hypertrophy, mitral/tricuspid stenosis, tumors, cardiac tamponade, constrictive pericarditis
Arrhythmias		Bradycardia, tachycardia, tachycardiomyopathy
Precipitant causes		
Cardiac		Arrhythmias, ischemic cardiomyopathy, negative inotropic drugs: calcium antagonists, beta-blockers, antiarrhythmics, others
Extracardiac		Infections, non-completion of treatment, pulmonary embolism, anemia, drugs (NSAIDs), surgery, effort, toxic substances

*AHT indicates arterial hypertension; NSAIDs, non-steroid anti-inflammatory drugs.

g) Complications

The following are heart failure complications:[14]

- Arrhythmias— Atrial fibrillation, ventricular arrhythmias (ventricular tachycardia & ventricular fibrillation), and bradyarrhythmia.
- Thromboembolism - Stroke, peripheral embolism, deep venous thrombosis, and pulmonary embolism.
- Gastrointestinal - Hepatic congestion & hepatic dysfunction and malabsorption
- Musculoskeletal - Muscle wasting
- Respiratory - Pulmonary congestion, respiratory muscle weakness, and pulmonary hypertension (rare).

2. **Prognosis in Heart failure**

HbA1c, eGFR, LVEF, 6MWT, and NT-proBNP are the commonly used prognostic markers. They can help to predict mortality in patients with heart failure.

"Left Ventricular Ejection Fraction": It indicates the extent of structural and functional abnormalities of the ventricle. It is used to describe poor prognosis, morbidity, and mortality. A gross functional abnormality is defined as a lvef of less than 20%. It has a connection to a dilated Left Ventricle chamber which is observed as a structural abnormality.[15]

The survival rates are declined by the simultaneous presence of diastolic dysfunction in heart failure patients.[16]

Hyponatremia: Hyponatremia is considered as a serum sodium concentration of < 135 mmol / L. In persons with acute heart failure, it is one of the predictors of poor outcomes. Serum sodium levels, on the other hand, are linked to mortality in patients with CHF.[17] In-hospital and 60 days mortality rates are more in patients with decreased sodium levels on admissions. Also, after 60 days of follow-up, a rise in serum sodium of 5 meq / l reduced mortality rates by 25%.[17]

Estimated glomerular filtration rate: Drugs used to treat heart failure can have a variety of side effects on renal function. It can lead to a decreased Glomerular Filtration Rate and a poor prognosis.[18]

The rate of mortality was identified as high in patients with decreased eGFR or decreasing eGFR. Renal impairment is most commonly related with venous congestion and reduced renal

perfusion. These are commonly influenced by compromised cardiac functioning.[19] According to the findings of the Testani et al.[20] study early GFR decline is linked to higher fatality rates among patients of heart failure.

NT-pro BNP: Natriuretic peptides such as BNP (B-type natriuretic peptide) and NT-proBNP (N-terminal pro-BNP) are produced mostly in the heart's left ventricle and discharged into the circulation under pressure or volume overload. It's a quick diagnostic test that also aids in identifying people who have heart failure and dyspnea. Serial natriuretic peptides testing is useful in establishing the efficacy of heart failure management as well as defining the prognosis of heart failure patients.[21–23]

All-cause mortality is very high in "acute heart failure" patients whose NT-proBNP levels are increased during the admission process. It is considered a reliable and powerful mortality predictor at 30 days and 1 year after admission. The optimal cutoff level of NT-proBNP used to predict 30 days and 1-year mortality has an increased specificity and sensitivity.[24] For acute heart failure patients, it can also help with risk stratification.[25]

Six-minute walk test: It is an exercise test used to figure out the functional capacity and therapeutic effects of the intervention.[26,27] 6-MWT is performed pre-rehabilitation and post-rehabilitation in order to compare the distances walked in patients of heart failure who are enrolled for cardiac rehabilitation. An increase in the distance travelled is regarded as a positive sign, while failure to do so can result in an increase in mortality.[28] A cut-off value of 300 m is considered as a comparison level. A patient who covers a distance of < 300 m has a poor prognosis. Reliability, low cost, and easy to conduct are the advantages of 6-MWT. Age,

stature, and BMI are the factors influenced by 6-MWT.[29] This is applicable for the cardiovascular mortality risk stratification.

HbA1c: It is identified that the increased levels of HbA1c are association with increased rates of hospitalization in patients with heart failure.[16]

3. RDW in heart failure

a) RDW in heart failure without anemia

Felker et al.[5] established the clinical usefulness of monitoring RDW in heart failure. In total standard deviation increase in RDW was connected to a 17 percent increased risk of cardiovascular death or hospitalization in failure heart patients and a 12 percent increased risk of all-cause mortality in this study. Furthermore, each one standard deviation increase in RDW was connected to a 29% increase in the risk of all-cause mortality.

According to the Tonelli et al.[30] study, every 1% increase in RDW value was linked to a 14% increase in all-cause mortality. Furthermore, during the follow-up period, each 1% increase in RDW was connected to a 15% greater risk of having symptomatic heart failure.

According to Hou et al.[31] each 1% increase in RDW value was linked to an 11 percent increased risk of mortality in heart failure patients, as well as an 11 percent increased risk of heart failure in patients with a prior history of cardiovascular disease.

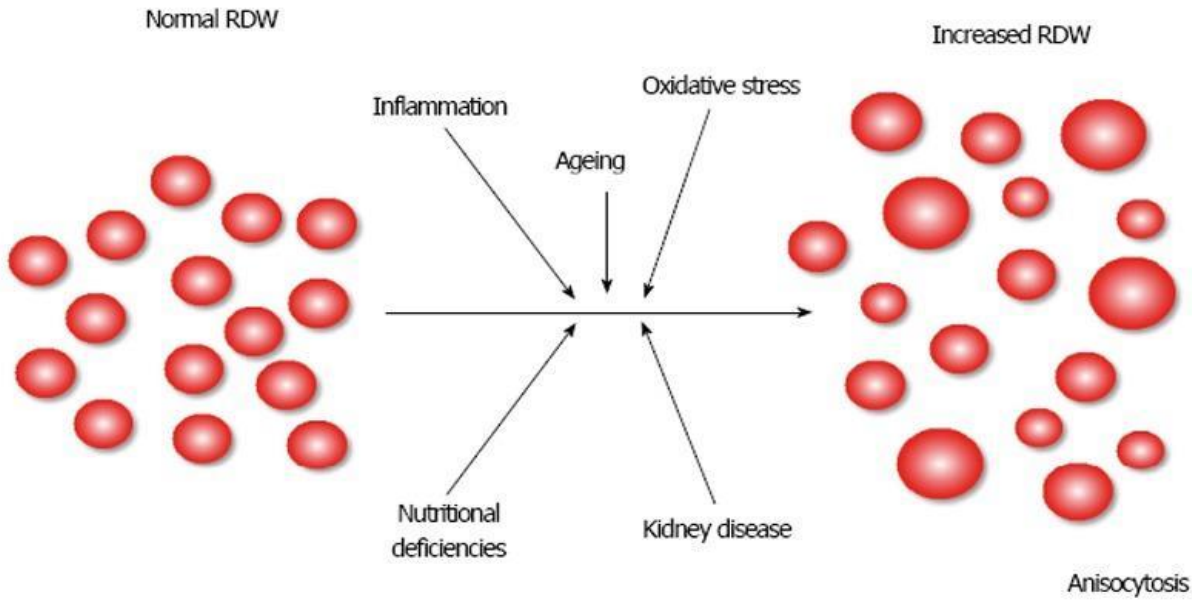
According to Huang and colleagues' meta-analysis study[32], in heart failure patients, each one percent raise in “RDW” value was associated with a 10% increase in the chance of future death events. No substantial difference was identified between the retrospective and

prospective studies, whereas a higher risk was identified in studies with a follow-up period of greater than two years as compared to those with a shorter follow-up period.

In a meta-analysis study, Shao et al.[33] discovered that each 1% increase in RDW was associated with a 19% increase in severe adverse cardiovascular events, a 12% increase in death, and a 9% increase in hospitalization in heart failure patients. The connection between RDW value and mortality was slightly stronger in CHF patients than in "acute heart failure" patients.[34]

b) Specific Mechanisms for the elevation of RDW in heart failure without anemia

Figure 7: Pathophysiological mechanisms causing anisocytosis.[35]



c) Relationship between RBC distribution width and the severity of" heart failure" in the absence of anemia.

To calculate red cell distribution, divide the standard deviation of RBC size by the mean corpuscular volume. It is a powerful predictor of poor outcomes in those with CAD, acute heart failure, and CHF. It is used to determine the size variability of red blood cells. It's a low-

cost, easily accessible marker that's frequently reported in automated cell counts. The link between a higher RDW and a higher risk of cardiovascular disease is not well understood.
RDW is a composite marker for a variety of chronic pathophysiological conditions. Comorbidities such as dietary deficits, inflammation, renal dysfunction, hepatic congestion, and bone marrow dysfunction may raise RDW values in chronic heart failure.[35]

Increased levels of RDW are correlated with mortality and cardiovascular events in known myocardial infarction patients but no symptomatic heart failure. In a retrospective study, Felker et al.[36] found that RDW is one of the most effective indicators of morbidity and mortality in heart failure patients.

Baggen VJM et al.[37] conducted a prospective cohort study on 602 participants. The researchers wanted to see if there was any correlation between RDW and cardiovascular events in persons with congenital heart disease. The "median age of the participants" was identified as 33 [25-41] years. The majority of the participants, 58 percent, were men. Around 98% of the subjects belonged to NYHA I. The primary endpoint (death, heart failure, hospitalization, arrhythmia, thromboembolic events, and cardiac intervention) was identified in 33% of the subjects. Median RDW was 13.4% in the patients with the primary endpoint, while it was 12.9% in patients without a primary endpoint. When age, sex, clinical risk factors, CRP, and NT-proBNP were taken into account, there was an association between RDW and the primary goal. The C-index of the model, including RDW, was observed as higher as compared to the model without. The study concluded the association between RDW and cardiovascular events in ACHD.

Dai Y et al.[38] performed a cross-sectional study on 521 patients. The researchers wanted to assess the short- and long-term prognostic values of RBC distribution width and hemoglobin in individuals with acute congestive heart failure. The mean Hgb levels in those who succumbed or remained alive were identified as 11.0±1.8g/l and 11.8±2.6 g/l. While the median values of RDW were identified as 16.2% and 14.4%, respectively. During the follow-up period, the mean Hgb levels in groups with and without endpoints were 11.42.5 and 12.52.4 g/dl, respectively, while the median RDW values were 14.9 and 13.8 percent. In-hospital mortality, RDW, "New York Heart Association" functional class IV, estimated glomerular filtration rate, and C-reactive protein were all found to be linked. The RDW, left ventricular ejection fraction, age, and NYHA functional classes III/IV were found to be independent risk factors of long-term outcomes. The study concluded that the increased levels of RDW in acute CHF patients during the admission process can be associated with the worse short- and long-term outcomes.

A retrospective analysis of 179 individuals was undertaken by Liu S et al.[39] Its goal was to see how important baseline red blood cell distribution width was in predicting the severity of chronic heart failure. In comparison to class I, RDW was shown to be higher in classes III and IV. Areas under ROCs of RDW and NT-ProBNP for class IV heart failure were identified as 0.817 and 0.840. RDW in the mortality group was identified as higher as compared with the survival group. RDW's predictive value was shown to be lesser than that of NT-ProBNP. RDW and NT-proBNP showed decreased predictive values for the repeated admission of ≥3. This study concluded that the RDW increased significantly in class III, IV, and in the mortality group.

Wołowiec Ł, et al.[40] conducted a study in 165 patients. The goal was to determine the "prognostic significance" of "red cell parameters" determined in a routine blood count in chronic heart failure. At one year of follow-up Hb level, RDW and N-terminal pro-B-type natriuretic peptide levels were related with the occurrence of the study endpoint. While, at 2 years of follow-up, it was identified as left ventricular ejection fraction, NYHA class, RDW, and NT-proBNP level. RDW and NT-proBNP levels were the independent predictors of mortality at one year of follow-up. While it was NT-proBNP level and NYHA class at two years of follow up. The baseline RDW and Hb level in patients admitted with NYHA class II-IV CHF were found to be predictors of mortality in this study.

Vizzardi E et al.[41] conducted a study on 30 participants. The purpose of the study was to examine the prognostic efficacy of echocardiographic indicators in patients with chronic heart failure. The optimal cut-off for RDW on the ROC curve was found to be 14.45 percent. Mitral regurgitation grade, left ventricular ejection fraction, posterior wall thickness, LV mass index, and RDW>14.45 percent were all linked to the primary target. LVEF, PWT, and RDW>14.45% were identified as the factors which predict the primary endpoint. According to the findings, "RDW" is a better predictor of worse outcomes in "patients with chronic heart failure."

YL H et al.[32] conducted a thorough examination and meta-analysis study. The goal of the research was to see how RDW affected heart failure prognosis. The heart rate for the effect of a 1% increase in baseline RDW on ACM was identified as 1.10. Heart failure participants with higher RDW had a worse prognosis than those with a lower RDW, according to the data.

Trinath M. et al.[42] performed a study on 210 participants. The study's goal was to see if RDW had any predictive value in heart failure. There was a link found with RDW at the start and mortality after a year. Male sex, diabetes mellitus, NYHA class III/IV, and atrial fibrillation were all revealed to be major predictors of death. Similarly, an association was identified between increased RWD, worse NYHA class, ischemic etiology, and diabetes mellitus. This study concluded that "RWD" has a strong predictive value in predicting rates of "mortality" in "patients with heart failure."

Celik A, et al.[43] recruited 71 patients for his study. The researchers wanted to see if there was a link between "RDW" and "echocardiographic characteristics" in people with "diastolic heart failure." RDW and "N-terminal pro-B-type natriuretic peptide" were identified as higher in the cases as compared to the cases. The DHF group had greater "echocardiographic measures" assessing "diastolic function." RDW levels were observed to be higher in the DHF group. According to the findings, elevated RDW levels in DHF patients are linked to "increased neurohormonal activity," decreased renal functioning, and "elevated filling pressure."

Rudresh M G. et al.[3] conducted a cross-sectional study on 70 subjects. The study's aim was to see if RDW levels might be utilized as a "prognostic indicator in heart failure patients." Results of the study revealed 15.763 ± 2.609 and 13.17± 0.75 as the mean RDW in patients and controls. Of the 44 subjects of HFREF, 33 had RDW> 13.6 while 11 had RDW ≤ 13.6. Similarly, Of the 26 patients of HFPEF, 20 had RDW > 13.6 while 6 had RDW ≤ 13.6. The researchers found that RDW is a simple test with a strong predictive value.

Ashitha B et al.[3] conducted a cross-sectional study on 100 patients. The researchers wanted to see if there was a link between "RDW" and the failure of the Heart. The study results revealed

16.177±3.247 and 13.67±2.156 as the mean RWD in cases and controls. RWD was >14.5 in 73% of cases in the HFrEF group and 50% of cases in the HFpRF group. In patients of heart failure, RWD values rise with the nature of the condition, according to the study.

Uemura Y et al.[44] conducted a study on 229 patients. The study's purpose was to see if there was a link between changes in RDW values during hospitalization and long-term prognosis in patients with acute decompensated heart failure. Subjects with greater RDW levels during hospitalization were found to have higher all-cause and cardiac-based mortality post-heart cardiac arrest, as per the findings. During hospitalization, changes in RDW levels were linked to both all-cause and cardiac-based death.

Xanthopoulos A et al.[45] performed a study on 218 study participants. Determination of the "prognostic value of RDW in HF and DM patients" was the main moto of the study. In both HF patients with and without DM, a link was discovered between RDW and a higher even rate. The longitudinal changes were identified to be significantly different between the two groups of HF patients.

LA A et al.[46] performed a study on heart failure. The study's goal was to see if RDW had any predictive value in heart failure. RDW was identified as a strong independent predictor of "adverse outcome" in chronic heart failure. This study concluded that RDW can indicate inflammatory stress and impaired iron mobilization.

CA C et al.[47] conducted a study on 6,159 ambulatory "chronic heart failure patients." The main moto of the study is if consecutive RDW examination could predict chronic heart failure. The median baseline RDW was identified as 14.9%. There was an association

identified between RDW >16% at baseline and an increased mortality rate. At the 12-month follow-up period, the majority of the patients showed rising RDW and increased risk for all-cause mortality. This study concluded that the baseline and serial increase in RDW are associated with poor long-term outcomes.

M OJ et al.[48] performed a study on 1,190 patients. The study's goal was to see if there was a link between “RDW” and “long-term mortality” in people with acute heart failure. The mortality rate was identified as 38% in the study population. This study concluded that increased levels of RDW are associated with increased long-term mortality.

R M. et al.[49] performed a retrospective cohort observational study on 451 patients. The goal of the research was to determine the “prognostic role” of “red cell distribution width” in admitted “patients of acute heart failure”. Males made up the majority of the participants (52%). Patients with an increased RDW were identified more comorbidities and greater Charlson Index. During the follow-up period, the mortality rate was 44 percent. In the cohort with decreased RDW,36.4% had died, while in the cohort with increased RDW, 63.7% had died. This study concluded that the age, etiology of heart failure, anemia, hyponatremia, estimated glomerular filtration rate, NT-proBNP levels, Charlson comorbidity score, atrial fibrillation, functional status, therapy with renin-angiotensin-aldosterone system inhibitors, beta-blockers. RDW is the powerful marker of worse long-term outcomes in acute heart failure.

W H et al.[50] performed a study on 128 participants. The researchers wanted to find out if RDW and N-terminal pro-B-type natriuretic peptides had any bearing on short-term clinical outcomes in acute heart failure. The 30 day and 90-day CV event rates were identified as 16.4% and 35.9%. NT-proBNP was more in patients with cardiovascular events at 30 and 90-day time points, while RDW was more at the 90-day time point. The independent predictive factors of a 90-day cardiovascular event were identified as RDW and NT-proBNP. Subjects with an RDW level > 14.5% and NT-proBNP > 1471.5 pg/mL were identified at highest risk for a cardiovascular event. The RDW and NT-proBNP were supposed to act as independent

risk factors of 90-day cardiovascular events in hospitalized patients with AHF. The study concluded that the RDW can add prognostic value to NT-proBNP for predicting early cardiovascular events.

LACUNAE OF LITERATURE:

In India, there are limited investigations on the link between "RDW and heart failure." There hasn't been any research comparing RDW & N-terminal pro-B-type natriuretic peptides for predicting short-term clinical outcomes in people with “acute heart failure.” The usefulness of RDW in predicting diastolic heart failure has only been demonstrated in one study.

MATERIALS & METHODS

MATERIALS AND METHODS:

Study site: The research was carried out in the Department of General Medicine at Sri Devaraj Urs Academy of Higher Education and Research, Tamaka, Kolar- 563101

Study population: All the eligible patients admitted with heart failure in the General Medicine opd at Sri Devaraj Urs Academy of Higher Education and Research were considered as the study population.

Study design: It was a Prospective Observational Study in this case.

Sample size:

Sample correlation coefficient = -0.41

Sample correlation coefficient = -0.41

Population correlation coefficient = .1

Power (%) = 80

Alpha Error (%) = 1

Sided = 2

Required sample size = 55

Alpha Error (1%) Power (90%) Sample Size (n=55)

Formula

$$n = \frac{\left(Z_{1-\frac{\alpha}{2}} + Z_{1-\beta}\right)^2}{[FZ(\rho_1) - FZ(\rho_0)]^2} + 3$$

$$FZ(\rho_1) = \frac{1}{2}\ln\left[\frac{1+\rho_1}{1-\rho_1}\right] \qquad FZ(\rho_0) = \frac{1}{2}\ln\left[\frac{1+\rho_0}{1-\rho_0}\right]$$

Where,

ρ_0 : Population correlation coefficient

ρ_1 : Sample correlation coefficient

$Z_{1-\alpha/2}$: Desired confidence level

$1-\beta$: Power

Sampling method: All the eligible subjects were enlisted into the study consecutively by convenient sampling till the sample size is reached.

Study duration: The data collection for the study was done between January 2020 to January 2021 for a period of 1 year.

Inclusion Criteria:

- Patients aged between 18 and 80.
- Heart failure patients.
- Heart failure patients both with preserved as well as reduced EF.

Exclusion criteria:

1) Liver disease.
2) Renal disease.
3) Blood transfusion within past 3 months.
4) Hematological malignancy.
5) Patients with hemoglobin <12g/dl.

Methodology:

Patients who satisfy inclusion criteria were included into the study.

All cases were subjected to a detailed history taking and clinical examination

Patients who satisfy Framingham's diagnostic criteria of heart failure were included into the study.

2D ECHO was performed for LV ejection fraction.

5ml of venous blood sample was drawn for RDW estimation.

Patients were divided into 2 groups of "reduced and preserved ejection fraction."

The groups were further divided according to the "severity of heart failure" (NYHA).

Age, sex, and other co-morbidities were matched.

Then RDW was correlated (positive) with the severity of the disease

FRAMINGHAM'S DIAGNOSTIC CRITERIA OF HEART FAILURE [51]

Major criteria	Minor criteria
Acute pulmonary oedema	Ankle oedema
Cardiomegaly	Dyspnoea on exertion
Hepatojugular reflex	Pedal Oedema
Neck vein distension	Hepatomegaly
PND or Orthopnoea	Nocturnal cough
Rales	Pleural Effusion
Third Heart sound gallop	Tachycardia (>120 beats/minute)

Heart failure is diagnosed when two major or one major and two minor criteria are met.

"New York Heart Association classification"[52]

- Class I: Symptoms only at levels of activity that would produce symptoms in normal individuals; ordinary physical activity does not cause undue dyspnea or fatigue
- Class II: Symptoms on ordinary exertion, resulting in mild limitation of physical activity
- Class III: Symptoms on less than ordinary exertion, resulting in marked limitation of physical activity
- Class IV: Symptoms at rest or minimal exertion, resulting in an inability to carry on any physical activity without discomfort

Following investigations were done on all the patients in the study group.

1) Chest x-ray
2) 2D ECHO
3) Electrocardiography
4) CBC
5) RFT
6) Serum Electrolytes
7) Liver-function tests
8) Measurement of creatine phosphokinase, B-type natriuretic peptide, D-dimer, as well as troponin I.

Ethical considerations: The study was approved by the institutional human ethics committee. Written and Informed consent was taken from all the study participants, and only those participants willing to sign the informed consent were recruited in the study. The risks and benefits involved in the study and the voluntary nature of participation were explained to the participants before obtaining consent. The study participants' anonymity was protected.

Data collection tools: All the relevant parameters were documented in a structured study proforma.

STATISTICAL METHODS:

NYHA class, RDW, etc., were considered as outcome parameters.

Descriptive statistics were used to analyze data in keeping with the study's objectives. Data were expressed as the mean, 95% confidence interval (CI; lower and upper bounds), median, minimum and maximum, and percentage, where appropriate. Data was also represented using appropriate diagrams like bar diagrams and pie diagrams. All Quantitative variables were checked for normal distribution. For normally distributed Quantitative parameters, the mean values were compared between study groups using ANOVA (>2 groups). P-value < 0.05 was considered statistically significant. Data were analyzed by using SPSS software, V.[53]

OBSERVATIONS AND RESULTS

RESULTS:

A total of 55 subjects were enlisted in the final analysis

Table 1: Descriptive analysis of age in study population (N=55)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
Age (in years)	54.15 ± 10.09	53.00	27.00	80.00	51.42	56.87

The mean age was 54.15 ± 10.09 years, ranged from 27 to 80 years. (Table 1)

Table 2: Descriptive analysis of gender in the study population (N=55)

Gender	Frequency	Percentages
Male	40	72.73%
Female	15	27.27%

Among the study population, 40(72.73%) were male, and 15(27.27%) were female. (Table 2 & Figure 8)

Figure 8: Pie chart of gender in the study population (N=55)

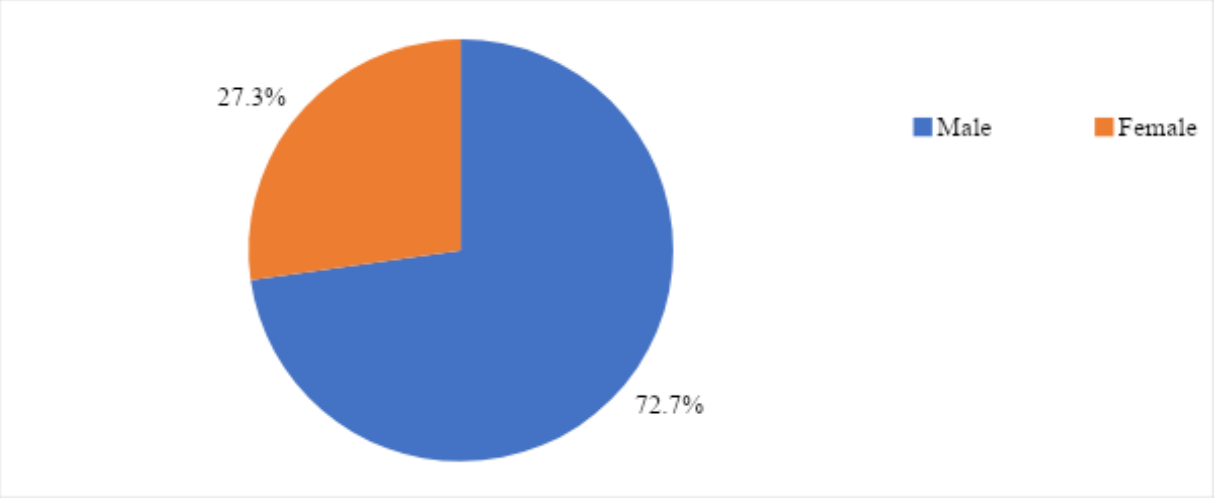


Table 3: Descriptive analysis of anthropometric parameter in study population (N=55)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
Height (in cm)	166.67 ± 9.39	168.00	148.00	180.00	164.14	169.21
Weight (in kg)	65.87 ± 7.93	65.00	50.00	86.00	63.73	68.02
BMI (in kg/m2)	23.83 ± 2.25	23.97	20.06	28.90	23.22	24.44

The mean height was 166.67 ± 9.39 cm, ranged from 148 to 180, the mean weight was 65.87 ± 7.93 kg, ranged from 50 to 86, and the mean BMI was 23.83 ± 2.25 kg/m2, ranged from 20.06 to 28.90 in the study population. (Table 3)

Table 4: Descriptive analysis of blood pressure in study population (N=55)

Parameter	Mean ± SD	Median	Minimum	Maximum
SBP (In mmhg)	122.73 ± 17.37	120.00	90.00	160.00
DBP (In mmhg)	80.18 ± 10.63	80.00	60.00	100.00

The mean systolic blood pressure was 122.73 ± 17.37 mmHg, ranged from 90 to 160, and the mean diastolic blood pressure was 80.18 ± 10.63 mmHg, ranged from 60 to 100 in the study population. (Table 4)

Table 5: Descriptive analysis of renal function parameter in study population (N=55)

Parameter	Mean ± SD	Median	Minimum	Maximum
Blood urea nitrogen (in mg/dl)	35.75 ± 15.88	30.00	16.00	90.00
Serum Creatinine(mg/dL)	0.98 ± 0.33	1.00	0.50	2.10

The mean blood urea nitrogen was35.75 ± 15.88 mg/dl, ranged from 16 to 90, and the mean serum creatine was 0.98 ± 0.33 mg/dl, ranged from 0.50 to 2.10 in the study population. (Table 5)

Table 6: Descriptive analysis of etiology parameter in the study population (N=55)

Etiology parameter	Frequency	Percentages
Alcoholic cardiomyopathy	3	5.45%
Calcific AS/AR	8	14.56%
Corpulmonale	5	9.09%
Dilated cardiomyopathy	10	18.18%
Ischemic heart disease	25	45.45%
Rheumatic heart disease	2	3.64%
Coronary artery disease	27	49.09%

Among etiology parameters, 3(5.45%) had alcoholic cardiomyopathy, 8(14.56%) had calcific AS/AR, 5(9.09%) had corpulmonale,10(18.18%) had dilated cardiomyopathy, 25(45.45%) had ischemic heart disease,2(3.64%) rheumatic heart disease and 27(49.09%) had coronary artery disease. (Table 6)

Table 7: Descriptive analysis of "red cell distribution width" in study population (N=55)

Parameter	Mean ± SD	Median	Minimum	Maximum
"Red cell distribution width"	15.07 ± 1.52	15.00	12.00	18.00

The mean "red cell distribution width" was 15.07 ± 1.52, ranged from 12 to 18 in the study population. (Table 7)

Table 8: Descriptive analysis of NYHA in the study population (N=55)

NYHA	Frequency	Percentages
Class I	9	16.36%
Class II	12	21.82%
Class III	19	34.55%
Class IV	15	27.27%

Among NYHA class, 9(16.36%) were class I, 12(21.82%) were class II, 19(34.55%) were class III, and 15(27.27%) were class IV. (Table 8 & Figure 9)

Figure 9: Bar chart of NYHA in the study population (N=55)

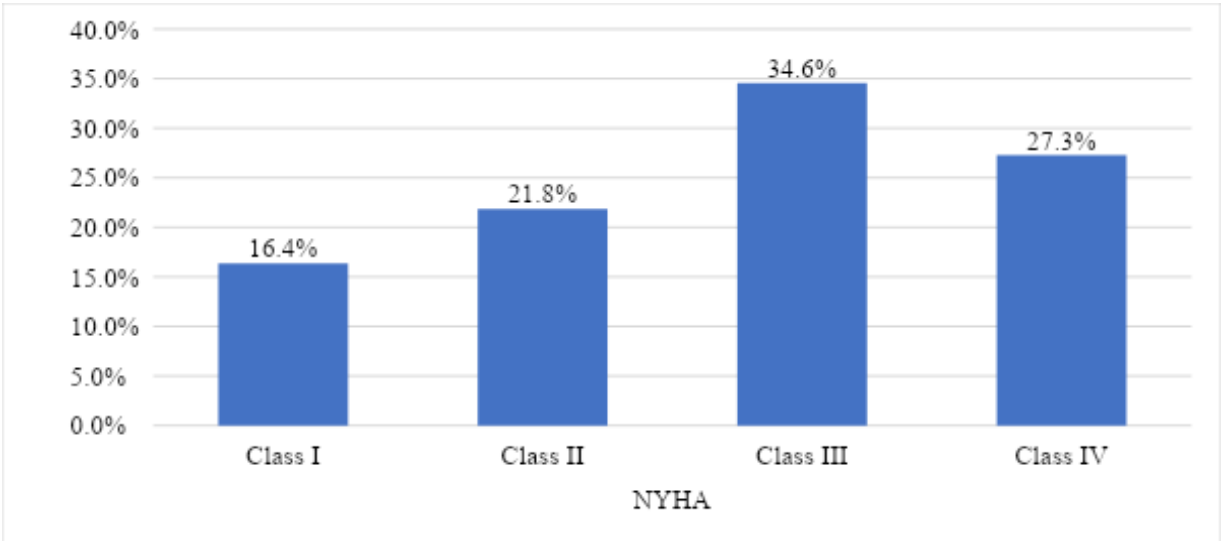


Table 9: Descriptive analysis of LVEF in the study population (N=55)

LVEF	Frequency	Percentages
<30%	17	30.91%
30-44%	16	29.09%
45-54%	13	23.64%
>54%	9	16.36%

Among LVEF, 17(30.91%) were <30%, 16(29.09%) were belongs to 30 to 44%, 13(23.64%) were 45 to 54% and 9(16.36%) were >54%. (Table 9 & Figure 10)

Figure 10: Bar chart of LVEF in the study population (N=55)

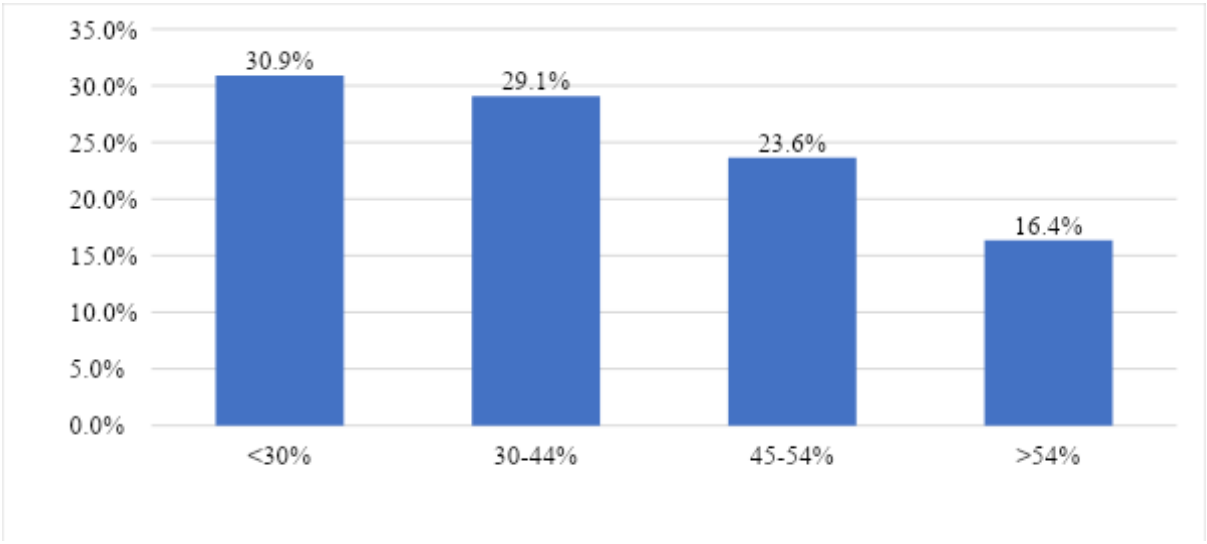


Table 10: Comparison of mean RDW across the study groups (N=55)

NYHA	"Red blood cell distribution width" Mean ± SD	Mean difference	95% CI		P-value
			Lower	Upper	
Class I	13.26 ± 0.72				
Class II	13.88 ± 0.99	0.62	-0.03	1.27	0.060
Class III	15.24 ± 0.68	1.98	-1.39	2.57	<0.001
Class IV	16.89 ± 0.53	3.63	-3.01	4.25	<0.001

The Mean "red cell distribution width" within NYHA class I was 13.26 ± 0.72, it was 13.88 ± 0.99 class II, it was 15.24 ± 0.68 class III, and it was 16.89 ± 0.53 in class IV. Taking class, class I as a baseline, the mean difference of red cell distribution width (0.62) in class II was statistically significant (P-value 0.060), and in-class III (1.98) and class IV (3.63) was also statistically significant (P-value <0.001). (Table 10)

DISCUSSION

DISCUSSION:

Increased RDW levels are connected to adverse outcomes in several conditions, such as chronic heart failure. Several research has looked into the significance of red blood cell distribution width in heart failure prognosis; however, the results have been mixed. The present study had done to describe the correlation of "red cell distribution width" with the" severity of heart failure" without anemia. A total of 55 subjects were enlisted in the final analysis.

In the present study, 54.15 ± 10.09 years was observed as the mean age of participants.

Rudresh M G. et al.[54] conducted a cross-sectional study on 70 subjects in which 54.86 ± 11.75 years was the mean age of the participants. In another study by AtacCelik, et al.[43] the mean of age was 57.09 ± 7.43 years in the study population.

Rudresh M G. et al.[54] AtacCelik, et al.[43] and our study showed similar results in terms of the mean of age.

Table 11: Comparison of mean of age between different studies.

Studies	Population	Mean of age (years)
Present study	55	54.15 ± 10.09
Rudresh M G. et al.[54]	70	54.86 ± 11.75
AtacCelik, et al.[43]	71	57.09 ± 7.43

In the present study, 72.73% were males, and 27.27% were females. Domingo A. et al.[55] performed a study on 628 patients in which males and females were identified with 68% and

32%, respectively. Similarly, in Ryszard Targoński. Et al.[56] studies 54.7% were males, and 45.3% were females.

In another study by Andrew Xanthopoulos et al.[45] proportions of males and females were identified with 60.6% and 39.4%, respectively.

Male predominance was identified in Domingo A. et al.[55] Ryszard Targoński. et al.[56] Andrew Xanthopoulos, et al.[45] Yusuke Uemura. Et al.[44] Mohamed Abdirahman Abdinur. et al.[57] and our study.

Table 12: Comparison of gender between various studies.

Study	Population	Gender
Present study	55	Males (72.73%) Females (27.27%)
Yusuke Uemura. Et al.[44]	229	Males (56.8%) Females (43.2%)
Mohamed Abdirahman Abdinur. et al.[57]	230	Males (67.4) Females (32.6)

In the present study, the mean of height (cm), weight (kg), and BMI (kg/m2) were identified as 166.67 ± 9.39, 65.87 ± 7.93, and 23.83 ± 2.25 respectively. Ryszard Targoński. et al.[56] conducted a study on 170 patients in which 83.1 ± 19.5 (kg) and 30.9 ± 6.5 (kg/m²) were identified as the mean of weight and BMI in the study population, which was an increased mean as compared to our study.

In our study, the mean of systolic blood pressure (mmHg) and diastolic blood pressure (mmHg) were identified with 122.73 ± 17.37 and 80.18 ± 10.63, respectively.

In Ryszard Targoński. et al.[56] studies 136.8 ± 26.5 and 80.8 ± 15.2 were identified as the mean of systolic blood pressure (mmHg) and diastolic blood pressure (mmHg), which resembles to our study results.

AtacCelik, et al.[43] performed a study on 71 participants in which 139.79 ± 20.06 and 86.41 ± 11.75 were observed as the mean of systolic blood pressure (mmHg) and diastolic blood pressure (mmHg) in the study population, which was an increased mean as compared to our study results.

Table 13: Comparison of blood pressure between various studies.

Study	Population	Blood pressure (mmHg)
Present study	55	SBP (122.73 ± 17.37) DBP (80.18 ± 10.63)
Ryszard Targoński. et al.[56]	170	SBP (136.8 ± 26.5) DBP (80.8 ± 15.2)
AtacCelik, et al.[43]	71	SBP (139.79 ± 20.06) DBP (86.41 ± 11.75)

In the present study, the mean of blood urea nitrogen (mg/dl) and serum creatinine (mg/dl) were observed as 35.75 ± 15.88 and 0.98 ± 0.33, respectively. In AtacCelik, et al.[43] studies, 0.78 ± 0.23 mg/dl was identified as the mean of creatinine. In another study by Rudresh M G. et al.[54] the mean of creatinine was identified as 1.2 ± 0.4 mg/dl in the study population.

The mean of creatinine was identified as similar in AtacCelik et al.[43] Rudresh M G. et al.[54] and our study.

In our study, coronary artery disease, ischemic heart disease, dilated cardiomyopathy, calcific AS/AR, corpulmonale, alcoholic cardiomyopathy, and rheumatic heart disease were the common etiologies identified in the study population with 49.09%, 45.45%, 18.18%, 14.56%, 9.09%, 5.45%, and 3.64%. In Rudresh M G. et al.[54] studies, ischemic heart disease,

hypertensive heart disease, rheumatic heart disease, and nonischemic cardiomyopathy were identified with 60%, 27.14%, 10%, and 2.86%, respectively.

Velan SB. Et al.[58] conducted a study on 100 patients in which IHD, RHD, Cor pulmonale, DCM idiopathic, alcoholic cardiomyopathy, and calcific AS/AR were the etiologies identified in the study with 47.0%, 14%, 12%, 9%, 3%, and 4% respectively.

Rudresh M G. et al.[54] Velan SB. et al. [58] and our study showed similar results in terms of etiology.

In the current study, the mean of red cell distribution width was identified as 15.07 ± 1.52. In Rudresh M G. et al.[54] studies, 15.763 ± 2.609 was identified as the mean RDW in patients.

In another study by Ryszard Targoński. et al.[56] the mean RDW was noted as 15.0 ± 1.8 (%) Rudresh M G. et al.[54] Ryszard Targoński. et al.[56] Andrew Xanthopoulos, et al.[45] Yusuke Uemura. et al.[44] showed similar results in terms of the mean of RDW with our study.

Table 14: Comparison of mean of RDW between various studies.

Study	Population	Mean of RDW (%)
Present study	55	15.07 ± 1.52
Andrew Xanthopoulos, et al.[45]	218	15.2 ± 1.6
Yusuke Uemura. et al.[44]	229	15.0 ± 1.9

In the present study, most of the participants had NYHD class III with 34.55%, followed by class IV, II, and I with 27.27%, 21.82%, and 16.36%, respectively. In Andrew Xanthopoulos et al.[45] study majority of the patients belonged to NYHA class IV with 71.6%% followed by NYHA III with 26.6%.

In Velan SB. Et al.[58] studies, most of the patients were belonged to NYHA class III with 60.0%, followed by class IV and II with 18.0% and 15.0%, respectively.

Andrew Xanthopoulos, et al.[45] Velan SB. Et al.[58] and our study showed similar results in terms of NYHD class.

Table 15: Comparison of NYHD class between various studies.

Study	Population	NYHD class
Present study	55	Class I (16.36%) Class II (21.82%) Class III (34.55%) Class IV (27.27%)
Domingo A. et al.[55]	628	Class I (26%) Class II (36%) Class III (27%) Class IV (11%)

In the present study, LVEF was <30% in the majority of patients with 30.91%, followed by 30-44% and 45-54% with 29.09% and 23.64%, respectively. In Velan SB. et al.[58] study LVEF was < 30% in 26.0% whereas, it was between 30 – 44 in 27.0%, 45 – 54 in 13.0% and > 54 in 34% of cases which resembles to our study results.

In the present study, the mean of RDW in NYHA classes I, II, III, and IV were identified with 13.26 ± 0.72, 13.88 ± 0.99. 15.24 ± 0.68 and 16.89 ± 0.53 respectively. In Velan SB. et al.[58] studies, the mean of red cell distribution width in NYHA class I, II, III, and IV were identified with 40.2 ± 1.06, 43.6 ± 2.7, 49.1 ± 3.2, and 61.5 ± 4.03, respectively which resembles to our study results.

SUMMARY:

Heart Failure is defined as the inability of the heart to maintain an output which is adequate to meet the required metabolic demands of the body. Ischemic heart disease, diabetes mellitus, atrial fibrillation, chronic kidney disease, valvular heart disease, and hypertension are the risk factors associated with heart failure.

Anisocytosis is measured in terms of RDW. RDW readings that are higher suggest that the size of red blood cells in a particular sample of blood varies more. RDW is used to limit the number of anemia diagnoses that can be made. It's frequently used to distinguish between iron deficiency anemia and thalassemia. The link between RDW and cardiovascular illnesses like heart failure and coronary artery disease has recently gotten a lot of attention. In heart failure patients, the width of red blood cell dispersion has recently been revealed as a new prognostic feature.

Anisocytosis is measured by the width of red cell distribution. Increased RWD levels have been linked to negative outcomes in a variety of diseases, including chronic heart failure. The importance of RDW in heart failure prognosis has been studied in several studies; however, the results have been inconsistent. The goal of this study was to see if there was a link between the width of red cell distribution & the severity of heart failure without anemia.

The final analysis comprised a total of 55 participants. The mean age of the participants was identified as 54.15 ± 10.09 years; most of the participants were males with 72.73%. The mean of height (cm), weight (kg) and BMI (kg/m2) were identified as 166.67 ± 9.39, 65.87 ± 7.93 and 23.83 ± 2.25 respectively.

The mean of systolic blood pressure (mmHg) and diastolic blood pressure (mmHg) was identified with 122.73 ± 17.37 and 80.18 ± 10.63, respectively. Coronary artery disease, ischemic heart disease, dilated cardiomyopathy, calcific AS/AR, corpulmonale, alcoholic cardiomyopathy, and rheumatic heart disease were common etiologies identified with

49.09%, 45.45%, 18.18%, 14.56%, 9.09%, 5.45%, and 3.64%. The mean RCD width was 15.07 ± 1.52

The majority of the participants had NYHD class III with 34.55%, followed by class IV, II, and I with 27.27%, 21.82%, and 16.36%, respectively. LVEF was <30% in the majority of patients with 30.91%, followed by 30-44% and 45-54% with 29.09% and 23.64%, respectively. The mean of red cell distribution width in NYHA classes I, II, III, and IV were identified with 13.26 ± 0.72, 13.88 ± 0.99. 15.24 ± 0.68 and 16.89 ± 0.53 respectively.

The width of the red cell distribution is an excellent prognostic indicator. The assessment of RDW can be used to predict the risk of progressing heart failure.

CONCLUSIONS:

The width of the red cell distribution is an excellent prognostic indicator. The assessment of RDW can be used to predict the risk of developing and progressing heart failure.

LIMITATIONS:

The sample size of the study was relatively small. Vitamin B_{12} and folate levels are the potential causes of increased levels of RDW, which are not measured in the study. The present study is conducted in a single center.

RECOMMENDATIONS:

Larger studies can be conducted with a longer duration. Risk factors, follow up and mortality can also be considered in future studies.

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ANNEXURES

STUDY PROFORMA

CORRELATION OF RED CELL DISTRIBUTION WIDTH WITH THE SEVERITY OF HEART FAILURE WITHOUT ANEMIA

PROFORMA

Name:

Age / Sex:

Residential Address:

Mobile No:

Case History:

Other known Illness:

BP: Pulse rate:

CVS-

RS-

P/A-

CNS-

Outcome Measures:

EJECTION FRACTION(EF)	RDW
Reduced EF	
Normal EF	

2D ECHO	Red cell distribution width
LVEF<30%	
LVEF30-44%	
LVEF45-54%	
LVEF>54%	

PATIENT INFORMATION SHEET

Study Title: CORRELATION OF RED CELL DISTRIBUTION WIDTH WITH THE SEVERITY OF HEART FAILURE WITHOUT ANEMIA

Study site: R.L Jalappa hospital, Tamaka, Kolar.

Aim: To evaluate RDW in heart failure.

To correlate RDW with severity of heart failure

Recently Red cell distribution Width was found to be elevated in many heart failure patients.It is considered as a measure of variability in RBC size. It is an easily available investigation as most of the hematology instruments measure measure RBC volume and give RDW.An elevated RDW can predict mortality and morbidity in heart failure. Various postulates and theories have been put forth by many researchers for the cause for elevated RDW in the context of heart failure.

Blood samples will be taken for the study to determine CBC, LFT, RFT and serum electrolytes. ECG, Chest X-Ray, SOB profile and 2D ECHO will also be done. This information is intended to give you the general background of the study. Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study we will collect information (as per proforma) from you or a person responsible for you or both. Relevant history will be taken. This information collected will be used only for publication. Principal investigator will be paying for dengue serology, serum sodium and serum potassium levels.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For any further clarification you can contact the study investigator:

Dr. K.Hemanth Kumar Reddy

Mobile no: 8095708798

Email:hemanthreddy56789@gmail.com

INFORMED CONSENT FORM

I ---------- participant, hereby give consent to participate in the study entitled "CORELATION OF RED CELL DISTRIBUTION WIDTH WITH THE SEVERITY OF HEART FAILURE WITHOUT ANEMIA"

I have been explained that;

1. I would have to provide a blood sample for the study purpose.
2. I have to answer the questionnaires related to project.
3. I do not have to incur any additional expenditure on my inclusion into the study.
4. The data generated from my clinical examination and laboratory tests and other reports will be used in the study (which may be subsequently published) without revealing my identity in any manner.

I affirm that I have been given full information about the purpose of the study and the procedures involved and have been given ample opportunity to clarify my doubts in my mother tongue. In giving my consent, I have not faced any coercion. I have been informed that, notwithstanding this consent given, I can withdraw from the study at any stage.

For any further clarification you can contact the study investigator:

Dr. K.Hemanth Kumar Reddy

Mobile no: 8095708798

Email:hemanthreddy56789@gmail.com.com

Signature of participant: Place:

Name of participant: Date:

ಸಮ್ಮತಿ ಪತ್ರ:

ಈ ಕೆಳಗೆ ಸಹಿ ಮಾಡಿರುವ ---------- ಆದ ನಾನು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲೆಗೊಳಳುವ ಸಲುವಾಗಿ ವೈದಯಕೀಯ ಪರಯೀಗ ಪರೀಕ್ಷೆಗೆ ಒಳಪಡಲು ನನನ ವೈಯಯಕಿಕ ವಿವರಗಳನುನ ನೀಡಲು ಸಮ್ಮತಿಸಿರುತೆ್ತೀನೆ. ಈ ಅಧ್ಯಯನದ ಉದೆದೀಶ, ಅಧ್ಯಯನದ ಸಂದಭ೯ದಲ್ಲಿ ನೀಡುವ ಮ್ತ್ತು ಸಂಗರಹಿಸುವ ಮಾಹಿತಿಯ ಗೆಗೀಪಯತೆಯ ಬಗೊ ನನಗೆ ನನನ ಸಥಳೀಯ ಭಾಷೆಯಲ್ಲಿ ಓದಿ ಹೇಳಲಾಗಿದೆ/ವಿವರಸಲಾಗಿದೆ ಮ್ತ್ತು ನಾನು ಇದನುನ ಅರ್ಥ ಮಾಡಿಕೆಗಂಡಿರುತೇನೆ. ಈ ಅಧ್ಯಯನದ ವಿವಿಧ್ ಅಂಶಗಳ ಬಗೊ ಪರಶ್ನಗಳನುನ ಕೇಳಳವ ಅವಕಾಶವನುನ ನನಗೆ ನೀಡಲಾಗಿದೆ ಮ್ತ್ತು ನನನ ಪರಶ್ನಗಳಗೆ ತ್ೃಪ್ತಿಕರವಾದ ಉತ್ತರಗಳಳ ದೆಗರೆತಿರುತ್ತೆ. ಈ ಅಧ್ಯಯನದ ಮ್ಗಲಕ ಸಂಗರಹಿಸಿರುವ ಮಾಹಿತಿಯನುನ ಸಂಶ್ೀಧ್ನೆಯ ಉದೆದೀಶಕೆ ಮಾತ್ರ ಬಳಸತ್ಕೆದುದ.

ಈ ಅಧ್ಯಯನದಿಂದ ಯಾವುದೇ ಸಂದಭ೯ದಲ್ಲಿ ಹಿಂದೆ ಸರಯುವ ಸ್ವಾತ್ಂತ್ರ್ಯ ನನಗಿದೆ ಎಂಬುದನಗನ, ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲೆಗೊಳಳುವುದರಂದ ನನಗೆ ಯಾವುದೇ ಹೆಚ್ುವರ ವೆಚ್ು ತ್ಗಲುವುದಿಲಿವೆಂಬುದನುನ ತಿಳದಿರುತೆ್ತೀನೆ.

ಪರೀಕ್ಷಾರ್ಥಿ೯ಯ ಹೆಸರು ಮ್ತ್ತು ಸಹಿ/ಹೆಬ್ೆ್ಟ್ಟಿನ ಗುರುತ್ು

ಸ್ವಾಕ್ಷಿಗಳ ಹೆಸರು ಮ್ತ್ತು ಸಹಿ

1. ದಿನಾಂಕ:

2. ದಿನಾಂಕ:

ಸಂದಶಥಕರ ಹೆಸರು ಮ್ತ್ತು ಸಹಿ ಪರಧಾನ ಪರೀಕ್ಷಕರ ಹೆಸರು ಮ್ತ್ತು ಸಹಿ

ದಿನಾಂಕ: ದಿನಾಂಕ:

MASTER SHEET

S.no	Age	Gender	Height	Weight	BMI	SBP	DBP	BUN (in mg/dl)	S. crea(mg/dL)	Etiology	RDW	NYHA	LVEF
1	55	Male	173	64	21.30	100	80	24	0.70	IHD/CAD	16.80	Class III	<30%
2	49	Male	169	70	24.50	90	60	18	0.80	IHD/CAD	17.00	Class IV	<30%
3	65	Male	170	71	24.60	120	80	21	1.10	DCM	15.90	Class III	30-44%
4	56	Female	153	58	24.70	130	90	26	1.30	IHD/CAD	16.00	Class III	<30%
5	40	Male	168	70	24.80	150	90	28	0.90	RHD	15.00	Class II	45-54%
6	50	Male	159	69	27.20	130	80	21	1.10	IHD/CAD	14.00	Class I	>54%
7	49	Female	151	59	25.80	120	90	19	1.00	DCM	14.30	Class I	>54%
8	67	Male	167	65	23.30	130	90	27	1.20	DCM	14.70	Class II	45-54%
9	60	Female	153	53	22.60	120	90	17	0.60	CAD	13.10	Class I	>54%
10	58	Male	165	62	22.70	130	90	18	1.10	CAD	13.00	Class I	>54%
11	52	Female	152	59	25.50	130	90	28	0.50	DCM	14.80	Class II	45-54%
12	49	Male	172	67	22.70	120	90	29	1.00	IHD/CAD	15.90	Class III	30-44%
13	54	Female	165	65	23.80	150	80	30	0.80	DCM	16.00	Class III	30-44%
14	50	Male	167	64	23.00	130	90	31	0.70	IHD/CAD	15.40	Class III	45-54%
15	53	Male	176	62	20.06	120	90	21	1.00	IHD/CAD	14.00	Class I	>54%
16	69	Female	153	55	23.50	130	80	26	0.90	CORPULMONALE	15.00	Class II	45-54%
17	80	Male	159	67	26.50	140	80	48	1.20	CORPULMONALE	14.90	Class II	45-54%
18	70	Male	178	70	22.15	100	60	30	1.30	CALCIFIC AS/AR	16.70	Class IV	<30%
19	45	Male	176	75	24.27	90	70	60	0.90	IHD/CAD	17.10	Class IV	<30%
20	77	Male	169	70	24.56	140	80	20	1.10	CALCIFIC AS/AR	16.00	Class IV	<30%
21	52	Male	180	80	24.60	160	90	19	0.70	IHD/CAD	15.00	Class III	30-44%
22	54	Male	172	79	26.70	130	90	26	1.00	IHD/CAD	14.90	Class III	30-44%
23	41	Male	172	71	24.06	130	80	38	0.80	ALCOHOLIC CARDIOMYOPATHY	13.00	Class II	45-54%
24	45	Male	176	65	21.00	120	80	42	1.00	DCM	16.50	Class IV	<30%
25	45	Female	149	50	22.50	110	70	18	0.60	IHD/CAD	18.00	Class IV	<30%
26	67	Male	166	59	21.45	140	90	44	1.10	CORPULMONALE	12.90	Class I	>54%
27	43	Male	178	79	25.00	160	80	52	0.70	RHD	13.90	Class II	45-54%
28	27	Male	176	86	27.80	130	80	49	0.90	ALCOHOLIC CARDIOMYOPATHY	13.90	Class II	45-54%
29	53	Male	171	70	23.97	140	90	45	1.10	CALCIFIC AS/AR	14.80	Class III	30-44%
30	50	Male	163	76	28.60	100	70	23	0.50	DCM	12.00	Class I	>54%
31	47	Male	179	78	24.30	120	70	39	1.30	IHD/CAD	12.50	Class II	45-54%
32	67	Male	177	69	22.04	120	70	72	0.80	IHD/CAD	12.90	Class II	45-54%
33	50	Male	177	68	21.70	120	90	37	1.00	DCM	16.70	Class IV	<30%

34	60	Male	165	74	27.20	150	90	51	0.70	CALCIFIC AS/AR	14.60	Class III	30-44%
35	43	Male	164	60	22.30	140	80	16	1.20	ALCOHOLIC CARDIOMYOPATHY	13.00	Class I	>54%
36	59	Female	152	59	25.50	110	70	57	1.00	IHD/CAD	17.30	Class IV	<30%
37	59	Female	151	58	25.40	100	80	25	0.80	IHD/CAD	17.30	Class IV	<30%
38	68	Female	148	50	22.80	120	90	24	0.70	DCM	15.00	Class III	30-44%
39	60	Male	175	63	20.58	130	90	50	1.30	DCM	14.00	Class III	30-44%
40	49	Male	176	62	25.80	110	60	44	0.60	CORPULMONALE	14.60	Class III	30-44%
41	55	Female	160	57	22.20	130	70	27	0.90	CALCIFIC AS/AR	15.10	Class III	30-44%
42	59	Female	159	54	21.40	130	90	37	0.60	CALCIFIC AS/AR	14.30	Class III	30-44%
43	54	Male	165	75	27.50	120	70	45	0.90	IHD/CAD	16.80	Class IV	<30%
44	41	Female	158	60	24.09	130	70	59	1.30	IHD/CAD	13.00	Class I	>54%
45	64	Male	159	73	28.90	100	70	66	1.60	CALCIFIC AS/AR	17.00	Class IV	<30%
46	52	Male	167	69	24.80	130	100	90	2.10	IHD/CAD	15.00	Class III	30-44%
47	57	Female	149	60	27.00	120	90	22	0.70	CALCIFIC AS/AR	13.30	Class II	45-54%
48	47	Male	176	66	21.30	110	70	28	0.80	IHD/CAD	15.90	Class IV	<30%
49	49	Female	168	57	20.21	130	70	42	1.90	IHD/CAD	12.60	Class II	45-54%
50	59	Male	175	76	24.80	140	100	35	1.40	CORPULMONALE	15.50	Class III	30-44%
51	40	Male	175	70	22.80	100	70	32	0.60	IHD/CAD	15.60	Class III	30-44%
52	55	Male	178	68	21.50	100	80	28	1.00	IHD/CAD	16.60	Class IV	<30%
53	69	Male	171	64	21.90	120	80	56	0.70	IHD/CAD	15.10	Class III	30-44%
54	48	Male	173	63	21.07	90	60	53	1.50	IHD/CAD	17.20	Class IV	<30%
55	42	Male	172	60	20.30	90	60	33	1.10	IHD/CAD	17.20	Class IV	<30%