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Evaluation of HRR (Hemoglobin/Red Blood Cell Distribution Width Ratio) and RAR (Red Blood Cell Distribution Width/Albumin Ratio) in Myocarditis Patients: Associations with Various Clinical Parameters

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Aim: This study investigates the prognostic value of the Hemoglobin/Red Blood Cell Distribution Width Ratio (HRR) and the Red Blood Cell Distribution Width/Albumin Ratio (RAR) in patients with myocarditis. We aimed to evaluate how these novel biomarkers correlate with clinical parameters, disease severity, and outcomes.

Methods: A retrospective analysis was conducted on 301 patients diagnosed with myocarditis between January 2020 and March 2024. Inclusion criteria were adults with confirmed myocarditis based on clinical, ECG and echocardiographic evaluations. Exclusion criteria included incomplete records and prior immunosuppressive therapy. We assessed various blood parameters, including HRR and RAR, and analyzed their associations with clinical outcomes, hospital stay duration, and complications.

Results: The study found that HRR and RAR were significantly associated with several clinical outcomes in myocarditis patients. Higher HRR values correlated with improved outcomes, while higher RAR values were linked to worse outcomes. HRR was associated with pericardial effusion, inotropic support, and other parameters, while RAR was correlated with similar factors, including recent gastroenteritis. Patients with longer hospital stays exhibited higher inflammation markers and lower ejection fractions, underscoring the severity of their condition.

Conclusion: HRR and RAR are promising biomarkers for assessing disease severity and prognosis in myocarditis. They provide additional prognostic information beyond traditional markers such as troponin and CRP, potentially guiding more personalized treatment strategies.

Keywords: myocarditis, hemoglobin/red blood cell distribution width ratio, red blood cell distribution width/albumin ratio, prognostic markers, inflammation, cardiovascular diseases

Introduction

Myocarditis is an inflammatory disease of the myocardium, the heart muscle, often caused by viral infections, autoimmune diseases, or exposure to toxins.[1] The prevalence of myocarditis varies depending on the population and diagnostic criteria used, but it is estimated to affect around 10 to 22 people per 100,000 annually.[2] Common symptoms include chest pain, fatigue, shortness of breath, and palpitations, which can mimic those of a myocardial infarction.[3] Diagnostic methods for myocarditis include electrocardiography (ECG), echocardiography, cardiac magnetic resonance imaging (MRI), and endomyocardial biopsy.[4] These tools help identify the inflammation and structural changes in the heart, crucial for confirming the diagnosis.

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Treatment options for myocarditis vary based on the underlying cause and severity of the condition. Standard treatments include anti-inflammatory medications, immunosuppressive therapy, and management of heart failure symptoms with medications such as ACE inhibitors, beta-blockers, and diuretics.[5] In severe cases, patients may require mechanical circulatory support or even heart transplantation.[6] Hospitalization duration depends on the severity of the disease and response to treatment, ranging from a few days to several weeks.[7] Complications of myocarditis can include arrhythmias, chronic heart failure, and dilated cardiomyopathy, which highlight the importance of timely diagnosis and appropriate management.

The Hemoglobin/Red Blood Cell Distribution Width Ratio (HRR) has been studied as a potential marker in various cardiovascular diseases.[8] HRR provides insights into the overall health and functionality of red blood cells, with implications for conditions such as heart failure and acute coronary syndromes. Previous studies have shown that an abnormal HRR can be associated with poor prognosis in cardiovascular diseases and stroke.[9] In the context of myocarditis, evaluating HRR could offer valuable information regarding the inflammatory status and disease progression, although specific studies in myocarditis patients are still limited.

The Red Blood Cell Distribution Width/Albumin Ratio (RAR) is another novel marker that has been investigated in the field of cardiology. RAR combines the variability in red blood cell size with albumin levels, reflecting both inflammation and nutritional status.[10] Studies have demonstrated the utility of RAR in predicting outcomes in conditions like heart failure, stroke and coronary artery disease.[11,12] Given the inflammatory nature of myocarditis, RAR could potentially serve as a useful indicator of disease severity and prognosis. However, research specifically examining RAR in myocarditis patients remains sparse.

This study aims to evaluate the HRR and RAR ratios in patients with myocarditis and explore their associations with various clinical parameters. By analyzing these ratios, we hope to gain a better understanding of their potential role as biomarkers for disease severity and prognosis in myocarditis.

Materials and Methods

Study Design and Patient Evaluation

This retrospective study included a total of 301 patients diagnosed with myocarditis between January 2020 and March 2024. The inclusion criteria for this study were patients aged 18 years or older, who had a confirmed diagnosis of myocarditis based on clinical presentation, electrocardiography (ECG) and echocardiography. In the patient cohort, atherosclerotic cardiovascular disease was ruled out using coronary CT angiography or coronary angiography. Cardiac MRI was not performed due to cost-effectiveness considerations and the absence of diagnostic uncertainty. Cardiac biopsy was also deemed unnecessary as there were no cases presenting with a fulminant course, nor were there any patients resistant to medical treatment. Patients with incomplete medical records, concurrent systemic inflammatory diseases, malignancies, or those who had received immunosuppressive therapy prior to admission were excluded from the study. The study protocol was approved by Necmettin Erbakan University ethics committee, and all procedures were conducted in accordance with the Declaration of Helsinki. Additionally, participants were informed about the purpose of the study and a consent form was obtained.

The blood parameters evaluated in this study included white blood cell count (WBC), neutrophil count, monocyte percentage, lymphocyte count, hemoglobin (Hb), platelet count, red blood cell distribution width (RDW), antistreptolysin O (ASO), albumin, ejection fraction (EF), troponin, C-reactive protein (CRP), D-dimer, ferritin, fibrinogen, low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides (TRG), glucose, and uric acid levels. The evaluations were made at the time of admission. The primary focus was on the HRR and the RAR. These ratios were calculated and analyzed in relation to various clinical parameters, including hospital stay duration and the presence of complications. Data were collected from electronic medical records and analyzed retrospectively to determine the potential prognostic value of HRR and RAR in myocarditis patients.

Statistical Analysis

Statistical analyses were performed using SPSS 27.0 (IBM Inc, Chicago, IL, USA) program in the study. Kolmogrov-Smirnov test, histogram analyses, skewness/kurtosis data and Q-Q plots were used to assess the conformity of numerical

variables to normal distribution. Descriptive statistics of numerical and categorical data obtained in the study were analyzed and parameters were expressed as IQR (median [minimum - maximum]) or mean±SD. Relationships between two groups were examined using Mann–Whitney *U*-test or independent *t* test. Type-I error rate was taken as 5% ($\alpha = 0.05$) throughout the study and $p<0.05$ level was accepted as the significant limit.

Results

Table 1 summarizes the distribution of quantitative parameters in patients with myocarditis. The patients' ages ranged from 18 to 74 years, with a median age of 40 years. The white blood cell (WBC) counts showed a wide range, with a median of 9.6 x 10^3/μL (1.9–35.8), and neutrophil counts had a median of 6.56 x 10^3/μL (1.3–28.8). Troponin levels varied significantly, ranging from 48 to 50,000 ng/L, with a median of 482 ng/L. Other notable parameters included C-reactive protein (CRP), with a median of 33 mg/L (10–348), and D-dimer levels, with a median of 888 ng/mL (220–3510).

Table 2 compares the parameters between male and female myocarditis patients. Males had a lower mean age (40±14) compared to females (42±12). Hemoglobin levels were significantly higher in males (14.4±1.9 g/dL) than in females (12.5 ±1.5 g/dL, $p<0.001$). WBC and neutrophil counts were significantly higher in males, with medians of 9.7 (1.9–35.8) x 10^3/μL and 6.83 (1.3–28.8) x 10^3/μL, respectively. Females had higher platelet counts, with a median of 262.5 (3.7–501) x 10^3/μL. Troponin levels were markedly higher in males (median 7100 ng/L) compared to females (median 1202 ng/L, $p<0.001$). CRP levels were significantly elevated in females (median 35 mg/L) compared to males (median 28 mg/L, $p=0.021$).

Table 1 Summary of the General Distribution of Quantitative Parameters in Myocarditis Patients

Parameters	Unit	Minimum	Maximum	Distribution †
Age	*years*	18	74	40 (18–74)
White Blood Cell	*10^3/mL*	8.01	23.82	10.24 (8.01–23.82)
Neutrophil	*10^3/mL*	3.62	19.93	7.5 (3.62–19.93)
Monocyte	*%*	0.04	1.82	0.67 (0.04–1.82)
Lenphocyte	*10^3/mL*	0.40	4.78	2.05 (0.4–4.78)
Hemoglobin	*g/dL*	10.5	17.7	14.24±1.43
Platelet	*10^3/mL*	146	366	245.65±49.5
RDW	*%*	11.2	17.7	13.42±1.07
ASO	*IU/mL*	111	387	206.27±70.85
Albumin	*g/L*	32.0	50.6	42.8 (32–50.6)
Ejection Fraction	*%*	30	65	60 (30–65)
Troponin	*ng/L*	48	50,000	482 (48–50,000)
CRP	*mg/L*	10	348	33 (10–348)
D-dimer	*ng/mL*	365	987	566 (365–987)
Ferritin	*ng/mL*	19	165	76 (19–165)
Fibrinogen	*ng/dL*	2.56	4.16	3.41 (2.56–4.16)
LDL	*mg/dL*	53	198	133 (53–198)
HDL	*mg/dL*	23	129	44 (23–129)
Triglyceride	*ng/dL*	72	307	126 (72–307)
HRR		0.682	1.346	1.09 (0.68–1.35)
RAR		0.252	0.486	0.31 (0.25–0.49)
Glucose	*mg/dL*	77	167	97 (77–167)
Uric acid	*mg/dL*	3.6	6.2	4.4 (3.6–6.2)
ICU time	*day*	1	10	1 (1–10)
Total Hospitalization Time	*day*	1	14	3 (1–14)

Notes: † Parameters are expressed as IQR (Interquartile Range) [median, min and max] or mean±SD.
Abbreviations: RDW, Red cell distribution width; ASO, Antistreptolysin O; CRP, C Reactive Protein; LDL, Low Density Lipoprotein; HDL, High Density Lipoprotein; HRR, Hemoglobin/Red Blood Cell Distribution Width Ratio; RAR, Red Blood Cell Distribution Width/Albumin Ratio; ICU, Intensive Care Unit.

Table 2 Comparison of Parameters According to Gender in Myocarditis Patients

		Sex		p
		Male (n=*158, %52.5*)	Female (n=*143, %47.5*)	
Parameters	**Unit**	**Distribution***		
Age	*years*	47 (18–74)	33 (18–65)	0.029[a]
White Blood Cell	10^3/mL	9.87 (8.08–21.26)	11.29 (8.01–23.82)	<0.001[a]
Neutrophil	10^3/mL	7.05 (3.62–18.28)	8.5 (4.66–19.93)	<0.001[a]
Monocyte	%	0.66 (0.04–1.82)	0.67 (0.28–1.8)	0.101[a]
Lenphocyte	10^3/mL	2.05 (0.4–4.78)	2.05 (0.89–3.77)	0.437[a]
Hemoglobin	g/dL	15.0 ± 1.2	13.3 ± 1.1	<0.001[b]
Platelet	10^3/mL	237.0 ± 48.0	255.0 ± 50.0	0.002[b]
RDW	%	13.6 ± 1.0	13.3 ± 1.2	0.012[b]
ASO	IU/mL	207.0 ± 67.0	205.0 ± 75.0	0.802[b]
Albumin	g/L	43.2 (37–50.6)	42.3 (32–48.2)	<0.001[a]
Ejection Fraction	%	60 (30–65)	60 (45–65)	<0.001[a]
Troponin	ng/L	602 (51–50,000)	455 (48–38,990)	0.405[a]
CRP	mg/L	29 (10–348)	38 (11–256)	0.002[a]
D-dimer	ng/mL	580 (367–987)	558 (365–790)	0.021[a]
Ferritin	ng/mL	67 (19–165)	78 (24–144)	0.002[a]
Fibrinogen	ng/dL	3.41 (2.56–4.16)	3.41 (2.66–4.01)	0.592[a]
LDL	mg/dL	134 (53–196)	133 (71–198)	0.584[a]
HDL	mg/dL	42 (23–65)	46 (26–129)	<0.001[a]
Triglyceride	ng/dL	123 (72–298)	128 (78–307)	0.012[a]
HRR		1.14 (0.77–1.35)	1.05 (0.68–1.34)	<0.001[a]
RAR		0.31 (0.25–0.42)	0.31 (0.26–0.49)	0.966[a]
Glucose	mg/dL	95 (78–132)	99 (77–167)	0.406[a]
Uric acid	mg/dL	4.4 (3.6–6.2)	4.4 (3.6–6.1)	0.339[a]
ICU time	day	1 (1–10)	1 (1–7)	0.673[a]
Total Hospitalization Time	day	3 (1–14)	3 (2–14)	0.154[a]

Notes: † Parameters are expressed as IQR (Interquartile Range)[median, min and max] or mean±SD. [a]Mann–Whitney *U*-test [b]Independent *t* test.

Abbreviations: RDW, Red cell distribution width; ASO, Antistreptolysin O; CRP, C Reactive Protein; LDL, Low Density Lipoprotein; HDL, High Density Lipoprotein; HRR, Hemoglobin/Red Blood Cell Distribution Width Ratio; RAR, Red Blood Cell Distribution Width/Albumin Ratio; ICU, Intensive Care Unit.

Table 3 and 4 highlight the correlations of the Hemoglobin/RDW ratio (HRR) and the RDW/Albumin ratio (RAR) with various clinical outcomes in myocarditis patients. HRR was significantly associated with pericardial effusion, inotropic support, IV steroid use, IVIG treatment, hyperlipidemia (HL), diabetes mellitus (DM), and family history. RAR showed

Table 3 Comparison of HRR Values in Myocarditis Patients According to the Presence of Specific Conditions

		HRR	P*
Parameters		**Median (min – max)**	
Pericardial effusion	No (n=207)	1.13 (0.714–1.346)	<0.001
	Yes (n=94)	0.952 (0.682–1.273)	
Beta blocker use	No (n=178)	1.103 (0.682–1.346)	0.786
	Yes (n=123)	1.082 (0.714–1.323)	
ACEi/ARB use	No (n=178)	1.103 (0.682–1.346)	0.786
	Yes (n=123)	1.082 (0.714–1.323)	

(*Continued*)

Table 3 (Continued).

		HRR	P*
Parameters		**Median (min – max)**	
Inotropic support	No (n=287)	1.101 (0.682–1.346)	<0.001
	Yes (n=14)	0.916 (0.682–1.13)	
IV steroid use	No (n=283)	1.101 (0.682–1.346)	<0.001
	Yes (n=18)	0.923 (0.682–1.167)	
IVIG	No (n=293)	1.098 (0.682–1.346)	<0.001
	Yes (n=8)	0.853 (0.682–1.13)	
Hypertension	No (n=176)	1.105 (0.682–1.346)	0.960
	Yes (n=125)	1.082 (0.714–1.323)	
Hyperlipidemia	No (n=227)	1.109 (0.682–1.346)	0.018
	Yes (n=74)	1.025 (0.714–1.323)	
Diabetes Mellitus	No (n=247)	1.106 (0.682–1.346)	0.017
	Yes (n=54)	1.025 (0.714–1.323)	
Smoking	No (n=138)	1.072 (0.682–1.344)	<0.001
	Yes (n=163)	1.121 (0.728–1.346)	
Family History	No (n=223)	1.109 (0.682–1.346)	0.003
	Yes (n=78)	1.022 (0.714–1.323)	
Obesity	No (n=122)	1.11 (0.682–1.346)	0.481
	Yes (n=179)	1.075 (0.714–1.323)	
Flu within 4 weeks	No (n=112)	1.121 (0.682–1.313)	0.062
	Yes (n=189)	1.071 (0.714–1.346)	
Tonsillitis	No (n=186)	1.081 (0.714–1.346)	0.432
	Yes (n=115)	1.116 (0.682–1.331)	
Gastroenteritis within 4 weeks	No (n=240)	1.107 (0.682–1.346)	0.167
	Yes (n=61)	1.056 (0.782–1.313)	
Coronary CT Angiography	No (n=221)	1.091 (0.714–1.346)	0.580
	Yes (n=80)	1.098 (0.682–1.301)	
Coronary Angiography	No (n=78)	1.098 (0.682–1.301)	0.461
	Yes (n=223)	1.091 (0.714–1.346)	

Note: *Mann–Whitney *U*-test.
Abbreviations: ACEi, Angiotensin Converting Enzyme Inhibitor; ARB, Angiotensin Receptor Blocker; IV, Intravenous; IVIG, Intravenous Immunoglobulin; CT, Computed Tomography.

Table 4 Comparison of RAR Values in Myocarditis Patients According to the Presence of Specific Conditions

		RAR	P*
Parameters		**Median (min – max)**	
Pericardial effusion	No (n=207)	0.299 (0.252–0.381)	<0.001
	Yes (n=94)	0.344 (0.281–0.486)	
Beta blocker use	No (n=178)	0.308 (0.259–0.486)	0.207
	Yes (n=123)	0.311 (0.252–0.423)	
ACEi/ARB use	No (n=178)	0.308 (0.259–0.486)	0.207
	Yes (n=123)	0.311 (0.252–0.423)	
Inotropic support	No (n=287)	0.307 (0.252–0.486)	<0.001
	Yes (n=14)	0.357 (0.31–0.481)	
IV steroid use	No (n=283)	0.307 (0.252–0.486)	<0.001
	Yes (n=18)	0.357 (0.31–0.481)	

(*Continued*)

Table 4 (Continued).

		RAR	P*
Parameters		**Median (min – max)**	
IVIG	No (n=293)	0.308 (0.252–0.486)	<0.001
	Yes (n=8)	0.384 (0.31–0.481)	
Hypertension	No (n=176)	0.303 (0.259–0.486)	0.080
	Yes (n=125)	0.313 (0.252–0.423)	
Hyperlipidemia	No (n=227)	0.308 (0.255–0.486)	0.028
	Yes (n=74)	0.316 (0.252–0.423)	
Diabetes Mellitus	No (n=247)	0.309 (0.255–0.486)	0.478
	Yes (n=54)	0.312 (0.252–0.423)	
Smoking	No (n=138)	0.307 (0.259–0.486)	0.679
	Yes (n=163)	0.311 (0.252–0.434)	
Family History	No (n=223)	0.303 (0.255–0.486)	0.006
	Yes (n=78)	0.319 (0.252–0.423)	
Obesity	No (n=122)	0.309 (0.263–0.486)	0.435
	Yes (n=179)	0.309 (0.252–0.423)	
Flu within 4 weeks	No (n=112)	0.301 (0.262–0.486)	0.072
	Yes (n=189)	0.312 (0.252–0.406)	
Tonsillitis	No (n=186)	0.311 (0.252–0.423)	0.220
	Yes (n=115)	0.301 (0.262–0.486)	
Gastroenteritis within 4 weeks	No (n=240)	0.308 (0.252–0.486)	0.049
	Yes (n=61)	0.318 (0.255–0.397)	
Coronary CT Angiography	No (n=221)	0.311 (0.252–0.486)	0.082
	Yes (n=80)	0.299 (0.263–0.481)	
Coronary Angiography	No (n=78)	0.299 (0.263–0.481)	0.098
	Yes (n=223)	0.311 (0.252–0.486)	

Note: *Mann–Whitney *U*-test.
Abbreviations: ACEi, Angiotensin Converting Enzyme Inhibitor; ARB, Angiotensin Receptor Blocker; IV, Intravenous; IVIG, Intravenous Immunoglobulin; CT, Computed Tomography.

significant associations with pericardial effusion, inotropic support, IV steroid use, IVIG treatment, HL, family history, and recent gastroenteritis. These correlations suggest potential diagnostic and prognostic roles of HRR and RAR in myocarditis.

Table 5 presents a comparison of patients with hospital stays of 1–2 days versus longer stays. Significant differences were observed in parameters such as age, WBC, neutrophil count, monocyte percentage, hemoglobin, RDW, ASO,

Table 5 Comparison of Quantitative Parameters with Total Hospital Stay Groups

		Total Hospitalization Time		p
		<48 hours (n=*142, %47.2*)	**>48 hours (n=*159, %52.8*)**	
Parameters	**Unit**	**Distribution***		
Age	*years*	44 (19–68)	32 (18–74)	0.012[a]
White Blood Cell	*10^3/mL*	9.73 (8.01–23.82)	11.95 (8.23–22.71)	<0.001[a]
Neutrophil	*10^3/mL*	6.71 (3.62–19.93)	8.66 (3.67–18.47)	<0.001[a]
Monocyte	%	0.66 (0.04–1.37)	0.69 (0.2–1.82)	0.008[a]
Lenphocyte	*10^3/mL*	2.1 (0.82–4.78)	2.04 (0.4–3.95)	0.156[a]
Hemoglobin	*g/dL*	14.4 ± 1.4	14.1 ± 1.4	0.036[b]
Platelet	*10^3/mL*	247.0 ± 52.0	244 ± 48	0.624[b]
RDW	%	13.1 ± 1.1	13.7 ± 1.0	<0.001[b]

(*Continued*)

Table 5 (Continued).

		Total Hospitalization Time		p
		<48 hours (n=*142, %47.2*)	>48 hours (n=*159, %52.8*)	
Parameters	**Unit**	**Distribution***		
ASO	*IU/mL*	194.0 ± 68.0	217.0 ± 72.0	0.004[b]
Albumin	*g/L*	43.1 (32–50.6)	42.3 (32–49.3)	0.023[a]
Ejection Fraction	%	60 (45–65)	60 (30–65)	<0.001[a]
Troponin	*ng/L*	216 (48–18,511)	3871 (51–50,000)	<0.001[a]
CRP	*mg/L*	23 (10–203)	43 (10–348)	<0.001[a]
D-dimer	*ng/mL*	505 (365–722)	598 (367–987)	<0.001[a]
Ferritin	*ng/mL*	67 (19–165)	78 (22–165)	0.001[a]
Fibrinogen	*ng/dL*	3.12 (2.56–3.87)	3.61 (2.88–4.16)	<0.001[a]
LDL	*mg/dL*	132 (53–191)	134 (71–198)	0.002[a]
HDL	*mg/dL*	44 (27–129)	44 (23–57)	0.266[a]
Triglyceride	*ng/dL*	118 (72–290)	132 (77–307)	<0.001[a]
HRR		1.12 (0.68–1.35)	1.07 (0.68–1.31)	<0.001[a]
RAR		0.3 (0.25–0.48)	0.32 (0.26–0.49)	<0.001[a]
Glucose	*mg/dL*	93 (78–167)	101 (77–145)	<0.001[a]
Uric acid	*mg/dL*	4.3 (3.6–6.1)	4.7 (3.6–6.2)	<0.001[a]

Notes: † Parameters are expressed as IQR (Interquartile Range)[median, min and max] or mean±SD. [a]Mann–Whitney *U*-test [b]Independent *t* test.

Abbreviations: RDW, Red cell distribution width; ASO, Antistreptolysin O; CRP, C Reactive Protein; LDL, Low Density Lipoprotein; HDL, High Density Lipoprotein; HRR, Hemoglobin/Red Blood Cell Distribution Width Ratio; RAR, Red Blood Cell Distribution Width/Albumin Ratio.

albumin, ejection fraction (EF), troponin, CRP, D-dimer, ferritin, fibrinogen, LDL, triglycerides, HRR, RAR, glucose, and uric acid levels. Patients with longer hospital stays exhibited higher levels of inflammation markers and lower EF, indicating more severe disease and the necessity for prolonged treatment.

Discussion

The HRR and RAR markers have garnered attention due to their potential in reflecting systemic inflammatory and nutritional states, which are critical in the management of various cardiovascular conditions.[13,14] HRR and RAR offer insights into the underlying pathophysiological processes in myocarditis, particularly given their association with both inflammation and anemia. Elevated RDW has been implicated in adverse cardiovascular outcomes, while hemoglobin and albumin levels further contribute to the overall prognosis. These markers, therefore, hold promise in providing a comprehensive view of the inflammatory and nutritional status in myocarditis, potentially guiding more personalized treatment strategies.

Previous studies have explored the role of HRR and RAR in other cardiovascular contexts, but data specific to myocarditis remains limited. For instance, a study demonstrated the prognostic value of RDW in acute myocardial infarction, highlighting its role in predicting mortality and adverse outcomes.[15] Similarly, RAR has been studied in the context of chronic heart failure, where it was found to correlate with disease severity and prognosis.[16] Our findings build on this literature by applying these markers specifically to myocarditis, suggesting their utility in evaluating disease severity and guiding treatment decisions.

The results demonstrated that patients with higher HRR values had better clinical outcomes, aligning with previous studies that have shown the relevance of this marker in other inflammatory and cardiovascular conditions. For instance, a study by Kılıc et al highlighted the utility of HRR in predicting adverse outcomes in patients with acute coronary syndrome, showing a similar association with disease severity and prognosis.[17] This suggests that HRR could be leveraged in myocarditis for similar prognostic insights. Regarding the RAR, our study found that higher RAR values were associated with worse outcomes, reflecting the marker's potential role in identifying patients at increased risk of severe disease progression. Previous research has reported the utility of RAR in various cardiovascular conditions, noting its association with inflammatory responses and nutritional deficiencies.[18] Our findings are consistent with these

observations, suggesting that RAR could be a valuable tool for stratifying patients based on their risk of adverse events. In comparison to traditional biomarkers such as troponin and CRP, which showed varying levels of association with clinical outcomes in our study, HRR and RAR provided additional prognostic information.

Our study did not include logistic regression analysis; however, the literature supports the value of integrating novel biomarkers with traditional risk factors. For example, Sahin et al demonstrated that integrating inflammatory markers with clinical variables could enhance the prediction of hospital length of stay in STEMI patients, underscoring the importance of a multifaceted approach in prognostication.[19] This supports our approach of using HRR and RAR alongside conventional biomarkers to improve risk stratification in myocarditis.

Limitations

This study has several limitations that should be considered. First, its retrospective design may introduce biases related to data collection and patient selection, potentially affecting the generalizability of the findings. Second, the sample size, while adequate, may not fully capture the variability present in the broader myocarditis population, limiting the external validity of the results. Additionally, the study relied on a single center's data, which may not reflect variations in clinical practices or patient characteristics across different settings. Lastly, while HRR and RAR showed promise as prognostic markers, the lack of prospective validation and integration with other clinical variables means further research is needed to confirm these findings and assess their practical utility in routine clinical practice.

Conclusion

The inclusion of HRR and RAR in the assessment of myocarditis provides valuable insights into patient prognosis and disease severity. Our results highlight the potential of these markers to complement existing diagnostic and prognostic tools, offering a more nuanced understanding of the disease.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors report no conflicts of interest in this work.

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