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# Right Heart Failure: Insight Into Its Epidemiological Profile and Management at Hassan II Regional Hospital Center in Agadir, Morocco

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Aymane Sebbar [1], Houssam Aitboutargante [1], Khalil Abderrahmane Elbaz [1], Wassim Beladel [2], Amine El Houari [1], Mohamed El Minaoui [1]

1. Cardiology Department, Mohammed VI University Hospital, Faculty of Medicine and Pharmacy, Ibn Zohr University, Agadir, MAR 2. Cardiology Department, Mohammed VI University Hospital, Faculty of Medicine and Pharmacy, Ibn zohr University, Agadir, MAR

**Corresponding author:** Aymane Sebbar, sebbaraymane0@gmail.com

## Abstract

### Background

Right-sided heart failure (RHF) is a distinct clinical syndrome with important prognostic implications and management challenges, particularly in settings where advanced diagnostic and device-based therapies may be limited. We aimed to describe the epidemiological profile, clinical presentation, etiologies, and in-hospital management of predominantly RHF in a tertiary regional hospital in southern Morocco.

### Methods

We conducted a descriptive retrospective study including adults (>18≥18 years) hospitalised for predominantly RHF in the Cardiology Department of Hassan II Hospital, Agadir, from June 2021 to May 2023. Patients with isolated left-sided or global heart failure and those with incomplete records were excluded. Demographic, clinical, electrocardiographic, echocardiographic, laboratory, etiological, therapeutic, and outcome data were extracted from medical charts and analysed using Microsoft Excel 2021 (Microsoft Corp., Redmond, WA, USA).

### Results

A total of 43 patients were included, with a mean age of 60.23 ± 17.51 years; 28 (65.1%) were men. Dyspnea was the most common presenting symptom, reported in 37 (86.0%) patients, while 25 (58.1%) were in New York Heart Association (NYHA) functional class IV. Echocardiographic assessment showed reduced left ventricular ejection fraction (LVEF; ≤40%) in 23 (53.5%) patients, echocardiographic pulmonary hypertension in 22 (51.2%), and right ventricular (RV) systolic dysfunction, reflected by low tricuspid annular plane systolic excursion, in 22 (51.2%). The leading etiologies were dilated cardiomyopathy in 17 (39.5%) patients and valvular heart disease in 12 (27.9%). Poor adherence to treatment or dietary measures was the most frequent precipitating factor, identified in 26 (60.5%) patients, followed by infection in eight (18.6%) and arrhythmia in six (14.0%). Laboratory abnormalities were common, including anemia in 25 (58.1%), renal impairment in 23 (53.5%), hyponatremia in 24 (55.8%), and elevated CRP in 33 (76.7%). At the end of hospitalization, all 43 (100%) patients received loop diuretics, whereas spironolactone, Angiotensin-converting enzyme inhibitors (ACEis), and beta-blockers were prescribed in 30 (69.8%), 27 (62.8%), and 31 (72.1%) patients, respectively. The mean length of hospital stay was 11.60 ± 6.81 days, and in-hospital mortality occurred in two (4.7%; 95% CI 0.6-15.8%) patients.

### Conclusions

In our RHF cohort, dilated cardiomyopathy and valvular disease predominated, and decompensation was most often related to potentially modifiable triggers, particularly poor adherence. Management relied mainly on diuretic-based decongestion, while advanced investigations and device therapies were limited by availability.

**Categories:** Cardiology, Epidemiology/Public Health
**Keywords:** echocardiography, epidemiological profile, heart failure, right-sided heart failure, right ventricular dysfunction

## Introduction

Right ventricle (RV) failure most commonly results from overload of the right heart chambers [1]. The most frequent mechanism is increased pulmonary vascular pressure, often secondary to left ventricular (LV) failure [2]. Symptoms are common but nonspecific, including dyspnea, lower limb edema, ascites, and

fatigue [3]. Physical examination may reveal elevated jugular venous pressure (JVP), an RV heave, a loud P2, a tricuspid regurgitation (TR) murmur, and hepatojugular reflux. ECG may suggest right atrial enlargement, right-axis deviation, or RV hypertrophy. Laboratory assessment, including BNP, may be useful to support the evaluation of RV dysfunction [4]. Transthoracic echocardiography is a key imaging modality to assess RV size and function and estimate pulmonary artery systolic pressure (PASP) [5]. Cardiac MRI provides a more precise assessment of RV size and RV ejection fraction (RVEF), supporting risk stratification [6]. Management depends on the dominant mechanism, focusing on optimizing preload, reducing afterload, and improving contractility, while treating the underlying cause [4]. In acute shock, fluid administration as a preload adjustment should be cautious. Afterload reduction may rely on targeted pulmonary vasodilators [7]. When contractility is impaired, urgent treatment of the cause is essential, with inotropes such as dobutamine when needed [4]. Studying this topic is important to emphasize that right-sided heart failure (RHF) should be assessed separately from global heart failure, as LV-directed therapies may not improve RV dysfunction, and RV failure may influence prognosis even when LV disease is successfully treated [8]. Our work aims to describe the epidemiological and therapeutic aspects of RHF in a population from southern Morocco and to compare it with published series.

## Materials And Methods

### Study design

This is a descriptive retrospective study conducted over two years, from June 2021 to May 2023, including 43 patients hospitalized in the Department of Cardiology at Hassan II Hospital in Agadir, Morocco. RHF was defined as a presentation dominated by systemic venous congestion (jugular venous distension, hepatomegaly, lower-limb edema, ascites), regardless of LV ejection fraction (LVEF); patients with combined left- and right-sided congestion were classified as global heart failure. Radiographic pulmonary congestion or pleural effusion did not lead to exclusion when systemic venous congestion dominated the clinical presentation.

Data were collected anonymously from medical records with the authorization of the head of the cardiology department.

### Inclusion criteria

We included in our study patients aged 18 years or older presenting with a syndrome of RHS.

### Exclusion criteria

We excluded from our study patients admitted for isolated left-sided heart failure or global (biventricular) heart failure, and patients with incomplete medical records. No patient was excluded for incomplete records (Figure *1*).

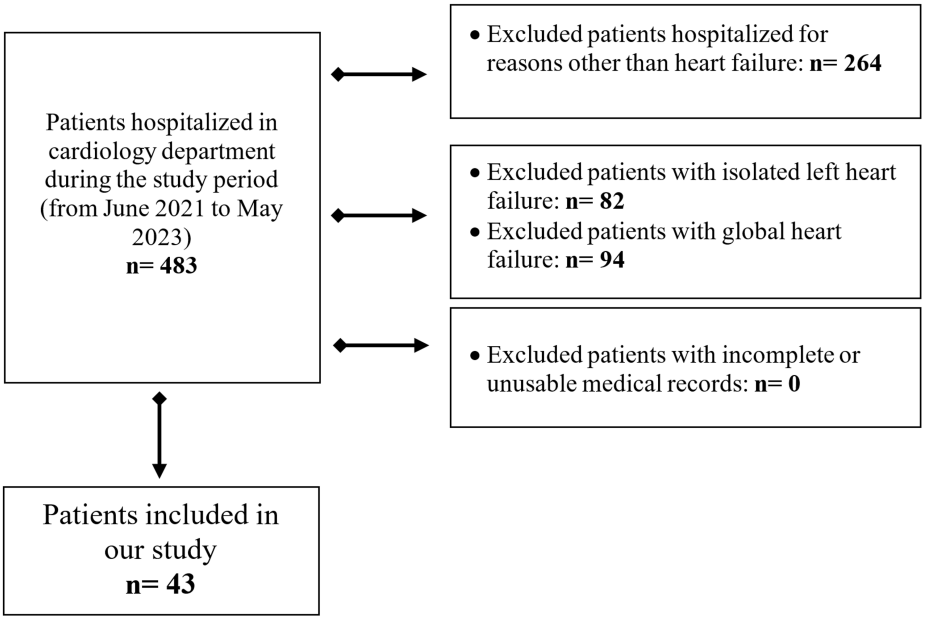


**FIGURE 1: Flow diagram showing study selection and inclusion and exclusion criteria**

### Data collection

Study data were extracted from hospital medical records, and for each patient meeting the inclusion criteria, including demographics (age and sex), cardiovascular risk factors (smoking, diabetes, hypertension, dyslipidemia, family history of coronary disease, and obesity), medical history and comorbidities, symptoms, admission clinical findings (heart rate, respiratory rate, blood pressure, oxygen saturation, and clinical signs of RHF), admission investigations (resting ECG and chest X-ray), transthoracic Doppler echocardiography findings, laboratory parameters, the etiology of RHF and its triggering factor, and the treatment initiated. Echocardiographic thresholds were: LV dilatation, end-diastolic diameter >58 mm (men) or >52 mm (women), RV dilatation, basal diameter >41 mm, RV hypertrophy, wall thickness >5 mm; left and right atrial dilatation, area >20 and >18 $cm^2$, dilated inferior vena cava (IVC), >21 mm, with reduced inspiratory collapse <50%, slow tricuspid annular plane systolic excursion (TAPSE), <17 mm, and low tissue Doppler S′, <9.5 cm/s [9]. PASP was estimated from the TR jet; pulmonary hypertension was graded as mild (40-50 mmHg), moderate (50-60 mmHg) or severe (>60 mmHg), without invasive confirmation, using the grading applied in our department, as guidelines provide no echocardiographic severity grading. Renal impairment was staged by estimated glomerular filtration rate (mild 60-89, moderate 30-59, severe 15-29, end-stage <15 mL/min/1.73 $m^2$), hyponatremia was defined as sodium <135 mmol/L, hyperkalemia as potassium >5 mmol/L, and moderate hepatic cytolysis as transaminases about twice the upper limit of normal.

### Statistical analysis

Statistical analysis was performed using Microsoft Excel 2021 (Microsoft Corp., Redmond, WA, USA).

Quantitative variables are reported with the sample size, mean +/- standard deviation, and range. For qualitative and ordinal variables, results are presented as the number of cases and the frequency (percentage) for each category (Figure *1*). Variables available in only part of the cohort are reported with the number of patients tested. The 95% confidence interval (CI) of in-hospital mortality was calculated using the exact Clopper-Pearson method.

## Results

### Demographic characteristics

Among the 43 patients with RHF, the mean age was 60.23 ± 17.51 years, ranging from 18 to 110 years. Age distribution showed six (14.0%) patients younger than 40 years, 11 (25.6%) aged 40-59 years, 23 (53.5%) aged 60-79 years, and three (7.0%) aged ⩾80 years (Figure *2*). There was a male predominance, with 28 (65.1%) men and 15 (34.9%) women (sex ratio 1.87).

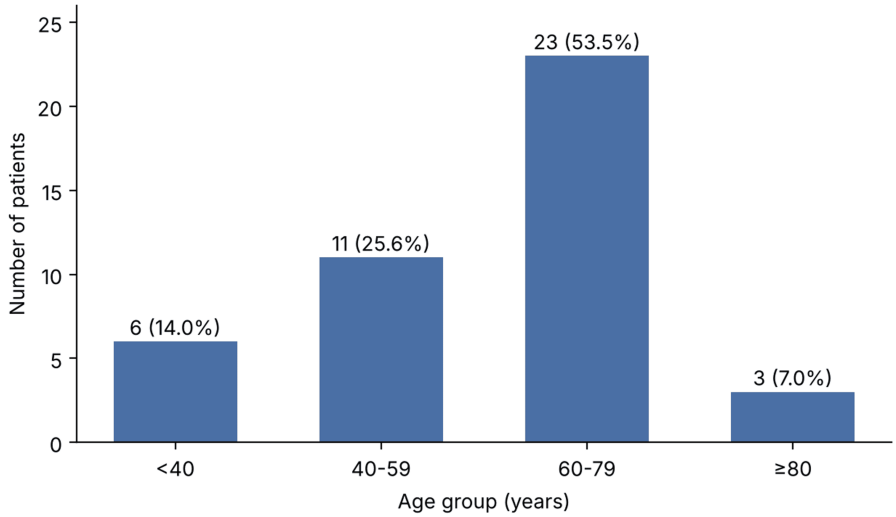


**FIGURE 2: Age-wise distribution of the study population.**

### Cardiovascular risk factors

Type 2 diabetes was present in 16 (37.2%) patients; among diabetic patients, 11 (68.8%) were on insulin, four (25.0%) were on oral antidiabetic drugs, and one (6.3%) was managed with diet alone. Hypertension was reported in eight (18.6%), and smoking in 13 (30.2%). Dyslipidemia (total cholesterol >2.2 g/L and/or low-density lipoprotein cholesterol >0.7 g/L) was documented in seven (16.3%), although lipid profiling was not performed in all patients at admission (number tested not recorded), and obesity (defined by a BMI ⩾30

kg/m²) was recorded in three (7.0%).

## Past medical history and comorbidities

Regarding cardiac history, documented categories included valvular heart disease in 16 (37.2%) patients, self-reported, undocumented heart disease in 13 (30.2%), congenital heart disease in three (7.0%), chronic cor pulmonale in one (2.3%), and pericardial disease in one (2.3%). Reported comorbidities included anemia in four (9.3%), thyroid dysfunction in two (4.7%), chronic kidney disease in two (4.7%), and neoplasia in one (2.3%).

## Clinical presentation and physical evaluation

Dyspnea was the predominant symptom, present in 37 (86.0%) patients, with New York Heart Association (NYHA) functional class distributed as follows: class I, six (14.0%); class II, three (7.0%); class III, nine (20.9%); and class IV, 25 (58.1%). Atypical chest pain occurred in three (7.0%), palpitations in three (7.0%), and syncope or lipothymia in one (2.3%). The most frequent physical sign was lower-limb edema, found in 31 (72.1%) patients. On physical examination, tachycardia (heart rate >100 bpm) was noted in 18 (41.9%), while 25 (58.1%) had a normal frequency, and no bradycardia was noted among those patients. On admission, four (9.3%) were hypertensive (systolic blood pressure (SBP) >140 mmHg), and 39 (90.7%) had a normal blood pressure (SBP 90-140 mmHg), with a mean SBP of 114.88 ± 13.72 mmHg. Low blood oxygen ($SpO_2$ <90%) was observed in four (9.3%) patients.

## ECG and chest X-ray

Resting ECG was performed in all patients and showed microvoltage in six (14.0%), LV hypertrophy in 14 (32.6%), RV hypertrophy in three (7.0%), left atrial enlargement in three (7.0%), and right atrial enlargement in one (2.3%). Rhythm disorders were present in 19 (44.2%), including atrial fibrillation in 17 (39.5%) and atrial flutter in two (4.7%); right bundle branch block was found in 10 (23.3%) and left bundle branch block in eight (18.6%); ventricular extrasystoles occurred in five (11.6%), and necrosis sequelae were reported in 16 (37.2%). Chest X-ray was also performed in all patients, showing cardiomegaly in 39 (90.7%), with a mean cardiothoracic index of 0.595, pleural effusion in 20 (46.5%), pulmonary congestion in 29 (67.4%), and an interstitial pattern in 22 (51.2%).

## Echocardiography and other imaging techniques findings

All patients underwent at least one transthoracic echocardiography. LVEF was reduced (⩽40%) in 23 (53.5%), mildly reduced (41-49%) in 4 (9.3%), and preserved (⩾50%) in 16 (37.2%). LV contractility abnormalities were seen in 31 (72.1%), including global hypokinesia in seven (16.3%), isolated segmental hypokinesia or akinesia in 11 (25.6%), and combined global and segmental abnormalities in 13 (30.2%). Valvular lesions included mitral regurgitation in 19 (44.2%), being minimal in 15 patients (34.9%), moderate in three patients (7.0%), severe in one patient (2.3%), TR in 18 (41.9%), severe mitral stenosis in six (14.0%), aortic regurgitation in eight (18.6%), and aortic stenosis in seven (16.3%). Other echocardiographic findings included elevated LV filling pressures in 17 patients (39.5%), LV dilatation in 26 (60.5%), RV dilatation 23 (53.5%), RV hypertrophy in four (9.3%), left atrial dilatation 18 (41.9%), right atrial dilatation in eight patients (18.6%), dilated IVC in 29 patients (67.4%) with reduced inspiratory collapse in 21 patients (48.8%), pericardial effusion in nine (20.9%), pulmonary hypertension in 22 patients (51.2%), low TAPSE in 22 (51.2%), and low tissue Doppler S′ in 22 (51.2%); parameters such as RV fractional shortening, pulmonary acceleration time, RV strain, and RVEF were not systematically available due to technical limitations. Among those with echocardiographic pulmonary hypertension, seven (16.3%) were mild, six (14.0%) moderate, and nine (20.9%) were severe, with a mean estimated PASP in the whole cohort of 36.33 ± 23.36 mmHg. Additional testing was limited by technical availability: coronary angiography was performed in three (7.0%) (with significant coronary disease reported), while 0 (0.0%) underwent right-heart catheterization or cardiac CT/MRI (Table *1*).

| Parameter | Category | n (%) |
|---|---|---|
| RV | RV dilatation | 23 (53.5%) |
| | RV hypertrophy | 4 (9.3%) |
| LV | Global hypokinesia | 7 (16.3%) |
| | Isolated segmental hypokinesia/akinesia | 11 (25.6%) |
| | Combined global + segmental abnormalities | 13 (30.2%) |
| | Elevated LV filling pressure | 17 (39.5%) |
| | LV dilatation | 26 (60.5%) |
| | Reduced LVEF (≤40%) | 23 (53.5%) |
| | Mildly reduced LVEF (41-49%) | 4 (9.3%) |
| | Preserved LVEF (≥50%) | 16 (37.2%) |
| LA | Left atrial dilatation | 18 (41.9%) |
| RA | Right atrial dilatation | 8 (18.6%) |
| MV | Minimal regurgitation | 15 (34.9%) |
| | Moderate regurgitation | 3 (7.0%) |
| | Severe regurgitation | 1 (2.3%) |
| | Severe mitral stenosis | 6 (14.0%) |
| TV | Tricuspid regurgitation | 18 (41.9%) |
| AV | Aortic regurgitation | 8 (18.6%) |
| | Aortic stenosis | 7 (16.3%) |
| IVC | Dilated IVC | 29 (67.4%) |
| | Reduced inspiratory collapse | 21 (48.8%) |
| Pericardium | Pericardial effusion | 9 (20.9%) |
| Pulmonary hypertension | Mild | 7 (16.3%) |
| | Moderate | 6 (14.0%) |
| | Severe | 9 (20.9%) |
| TAPSE | Low | 22 (51.2%) |
| Coronary angiography | Performed (significant CAD reported) | 3 (7.0%) |

**TABLE 1: Summary of the main structural and functional abnormalities identified in the study population.**

RV: Right Ventricle; LV: Left Ventricle; LA: Left Atrium; RA: Right Atrium; IVC: Inferior Vena Cava; MV: Mitral Valve; TV: Tricuspid Valve; AV: Aortic Valve; LVEF: Left Ventricular Ejection Fraction; TAPSE: Tricuspid Annular Plane Systolic Excursion; CAD: Coronary Artery Disease

## Laboratory findings

NT-proBNP was measured in five (11.6%) patients and was positive in four (80.0%), with a reported mean concentration of 13160 ± 8808.14 pg/mL. Anemia on laboratory testing was reported in 25 (58.1%), including iron deficiency in 11 (44.0%) of anemic patients. Renal function was normal in 20 (46.5%) and impaired in 23 (53.5%), distributed as mild (nine, 20.9%), moderate (12, 27.9%), severe (one, 2.3%), and end-stage (one, 2.3%). Electrolyte abnormalities included hyponatremia in 24 (55.8%) and hyperkalemia in 10 (23.3%), with no hypokalemia. Inflammation (CRP >10 mg/L) was present in 33 (76.7%), moderate hepatic cytolysis in 11 (25.6%), and thyroid dysfunction in five (11.6%), including hyperthyroidism in two (4.7%) and hypothyroidism in three (7.0%) (Table *2*).

| Laboratory finding | | Result (n = 43) |
|---|---|---|
| NT-proBNP | Measured | 5 (11.6%) |
| | Positive | 4/5 (80.0%) |
| | Mean concentration (pg/mL) | 13,160 ± 8,808.14 |
| Anemia | | 25 (58.1%) |
| Renal function impairment | Mild | 9 (20.9%) |
| | Moderate | 12 (27.9%) |
| | Severe | 1 (2.3%) |
| | End-stage renal disease | 1 (2.3%) |
| Electrolytes | Hyponatremia | 24 (55.8%) |
| | Hyperkalemia | 10 (23.3%) |
| | Hypokalemia | 0 (0.0%) |
| Inflammation (CRP > 10 mg/L) | | 33 (76.7%) |
| Moderate hepatic cytolysis | | 11 (25.6%) |
| Thyroid dysfunction | Hyperthyroidism | 2 (4.7%) |
| | Hypothyroidism | 3 (7.0%) |

**TABLE 2: Summary of laboratory abnormalities in the study population.**

## Etiologies and triggering factors

The main etiologies of RHF in our study were dilated cardiomyopathy in 17 (39.5%), including two (4.7%) rhythm-related cardiomyopathy secondary to thyrotoxicosis, valvular heart disease in 12 (27.9%), ischaemic cardiomyopathy in five (11.6%), chronic cor pulmonale in four (9.3%), congenital heart disease in four (9.3%), and chronic constrictive pericarditis in one (2.3%) (Table *3*).

| Etiologies | n (%) |
|---|---|
| Dilated cardiomyopathy | 17 (39.5%) |
| Valvular heart disease | 12 (27.9%) |
| Ischemic cardiomyopathy | 5 (11.6%) |
| Chronic cor pulmonale | 4 (9.3%) |
| Congenital heart disease | 4 (9.3%) |
| Chronic constrictive pericarditis | 1 (2.3%) |

**TABLE 3: Etiologies of right-sided heart failure counted in our population.**

The most frequent precipitating factor for decompensation was dietary non-adherence to salt restriction or treatment discontinuation or poor adherence in 26 (60.5%), followed by infection in eight (18.6%), arrhythmia (atrial fibrillation/flutter) in six (14.0%), de novo decompensation without an identified trigger in four (9.3%), pulmonary embolism in two (4.7%), hyperthyroidism in two (4.7%), and chronic obstructive pulmonary disease or asthma exacerbation in one (2.3%) (Table *4*). A patient could have more than one factor (49 factors in 43 patients).

| Triggering factors | n (%) |
|---|---|
| Poor adherence to treatment or salt restriction | 26 (60.5%) |
| Infection | 8 (18.6%) |
| Arrhythmia (AF/flutter) | 6 (14.0%) |
| De novo decompensation (no trigger identified) | 4 (9.3%) |
| Pulmonary embolism | 2 (4.7%) |
| Hyperthyroidism | 2 (4.7%) |
| COPD/asthma exacerbation | 1 (2.3%) |

**TABLE 4: Causes of decompensation identified in our population.**

COPD: Chronic Obstructive Pulmonary Disease; AF: Atrial Fibrillation.

### Therapeutic management and in-hospital outcomes

On admission, loop diuretics were used in 15 (34.9%) patients, whereas at discharge, 43 (100%) received loop diuretics (furosemide), with daily discharge doses of 40 mg in 29 (67.4%), 80 mg in 10 (23.3%), 250 mg in three (7.0%), and 500 mg in one (2.3%). Mineralocorticoid receptor antagonists (spironolactone) were used in nine (20.9%) at admission and 30 (69.8%) at discharge. No patient received pulmonary vasodilators. Angiotensin-converting enzyme inhibitors (ACEi) were prescribed in 11 (25.6%) at admission and in 27 (62.8%) at discharge, including ramipril in 23 (85.2%) of ACEi users, perindopril in four (14.8%), while angiotensin II receptor blockers (ARBs) were absent at admission and used in one (2.3%) at discharge (valsartan). Beta-blockers increased from 12 (27.9%) at admission to 31 (72.1%) at discharge, predominantly bisoprolol in 27 (87.1% of beta-blocker users). Angiotensin receptor-neprilysin inhibitor (ARNI) therapy was used in one (2.3%) at admission, and two (4.7%) at discharge; SGLT2 inhibitors were absent at admission and prescribed to eight (18.6%) at discharge. Ivabradine increased from one (2.3%) to three (7.0%), and digitalis from one (2.3%) to four (9.3%); iron-deficiency anemia was managed with oral iron supplementation in 11 (44.0% of anemic patients). Interventional management remained limited: diagnostic coronary angiography was performed in three (7.0%) with no case of revascularization, 19 (44.2%) had a potential indication for an implantable cardioverter-defibrillator (ICD), and 19 (44.2%) for cardiac resynchronization therapy (CRT), related to concomitant LV systolic dysfunction and to be confirmed after at least three months of optimal medical therapy, but none received device therapy because implantation was not available in our centre. Mean hospital length of stay was 11.60 ± 6.81 days; heart failure was stabilized in 41 (95.3%), complications occurred in 6 (14.0%), and in-hospital mortality was two (4.7%; 95% CI 0.6-15.8%), with complications mainly consisting of cardiogenic shock in five (11.6%) and embolic events in one (2.3%).

## Discussion

### Definition

RHF is a clinical syndrome related to impaired right-heart function [10, 11]. It results from inadequate blood delivery to the pulmonary circulation and/or elevated systemic venous pressure [12]. RHF is a broad clinical concept that refers to dysfunction involving any component of the right heart, whereas RV failure is more specific and denotes failure of the RV itself [11]. RHF involves mechanisms that differ from LV failure [11]. It reflects the inability of the right ventricle to pump deoxygenated blood into the lungs and is most often associated with LV failure [13]. In this context, the incidence and severity of RV dysfunction (RVD) correlate with the degree of LV systolic dysfunction, with severe RVD more frequently observed in patients with low LVEF [14].

### Epidemiology

RV failure represents a smaller proportion of heart failure admissions, with a substantial proportion occurring secondary to LV failure [13]. In the Moroccan series by Aitboutargante et al., conducted in our centre (Hassan II Hospital, Agadir) during the same period (2021-2023), the mean age of patients admitted for heart failure was 62.35 ± 14.1 years (range: 18-110), with 57.1% men [15]. As our patients belong to this cohort, a similar profile is expected: a mean age of 60.23 ± 17.51 years (range: 18-110 years) and a male predominance of 65.1%. Regarding major cardiovascular risk factors, diabetes affects roughly 40% of heart failure patients (ADHERE 44% [10], Aitboutargante 42.9% [15], Kheyi 43% [16]). In our series, diabetes was present in 37.2%, which remains within the range reported in the literature. Hypertension is very common in general heart failure cohorts (Framingham 75% [17]) but was less frequent in our RHF cohort (18.6%), compared with 35.2% in Aitboutargante et al. [15] and 46% in Kheyi et al. [16]. Smoking is frequent in

African cohorts (66% Adoubi et al. [18]) and remains substantial in Moroccan series (32.9% Aitboutargante [15]; 45% Kheyi [16]). Obesity shows high variability between studies (29.2% Aitboutargante [15]; 35% Framingham [17]; 23.4% Mali Medical [19]; 80% Kingue et al. [20]). In our series, obesity was 7.0% and known dyslipidemia 16.3%, suggesting these were less prominent risk factors in our population than in some reports of general heart-failure cohorts.

## Pathophysiology

Chronic RV afterload initially leads to increased contractility, followed by hypertrophy, and later dilation with rising filling pressures and functional TR. This, in turn, aggravates volume overload and reduces forward output. In contrast, an acute rise in afterload may precipitate rapid RV decompensation [21].

## History and physical examination

Assessment begins with a careful history and physical examination to determine severity, rapidity of evolution, and potential causes [22]. Symptoms are largely related to hypoxemia and systemic venous congestion and may include shortness of breath, chest discomfort, palpitations, and peripheral swelling. Physical findings may include jugular venous distension, hepatosplenomegaly and hepatojugular reflux, peripheral edema, ascites, an RV heave, a TR murmur, and signs of concomitant LV failure. Signs of low peripheral perfusion such as syncope, cool extremities, and oliguria may be observed in more severe cases [21].

## Evaluation

ECG may show signs of RV volume overload, including right axis deviation, RV strain patterns, P-pulmonale, and atrial arrhythmias [23]. D-dimer can be used when pulmonary embolism is suspected. BNP and troponin may help evaluate myocardial injury and severity. Imaging is essential to evaluate right-heart structure and function, particularly RV size and performance, which has strong prognostic value [12]. Echocardiography assesses RV and RA enlargement, TAPSE, RV free wall strain, myocardial performance index, and IVC size and collapsibility to estimate RA pressure [24]. Three-dimensional echocardiography can further improve estimation of RV volumes and EF. Cardiac MRI is performant when evaluating RV volumes and function, perfusion, and regurgitant volumes. CT can estimate RV volumes and EF but has limitations and radiation concerns. Catheterization of the RH is useful to provide chamber pressures and pulmonary vascular resistance [21].

## Etiology

Multiple causes can lead to right ventricular congestion, including acute pulmonary embolism (PE) [21]. When associated with RVD, PE is linked to worse prognosis in up to 60% of cases, with mortality multiplied by up to 3.5 when RVD is present. Chronic pressure overload causes include primary and secondary pulmonary hypertension [12], defined by a mean pulmonary artery pressure >20 mmHg at rest [25]. Congenital heart diseases (e.g., pulmonary stenosis, RV outflow tract obstruction, Tetralogy of Fallot, hypoplastic left heart) may cause pulmonary hypertension and RH overload [21]. Volume overload may occur due to right-sided valvular lesions and shunts, including TR and pulmonary regurgitation [12]. Intrinsic RV myocardial disease may result from infarction, most often linked to inferior STEMI (up to 50% of cases) [26], infiltrative disease such as amyloidosis, and genetic cardiomyopathies. Additionally, RVD has been reported to be more frequent in non-ischaemic compared with ischaemic cardiomyopathy [27]. RV filling impairment may also cause RV failure and is typically related to constrictive pericarditis or tamponade, tricuspid stenosis, and superior vena cava syndrome [21]. Other causes include myocarditis due to inflammatory processes affecting the myocardium [12].

In our study, the leading etiology of RHF was dilated cardiomyopathy (39.5%), followed by valvular heart disease (27.9%), predominantly tricuspid and mitral lesions, while ischemic cardiomyopathy accounted for 11.6%, possibly underestimated given that only three patients had coronary angiography. Chronic cor pulmonale and congenital heart disease each represented 9.3%, and chronic constrictive pericarditis was least frequent (2.3%). In contrast, the Framingham study reported hypertension and coronary artery disease as major drivers of heart failure [28]. In the Moroccan study by Kheyi et al., etiologies were dominated by ischemic heart disease (59.91%), with primary dilated cardiomyopathy (16.27%) and valvular disease (12.74%) less frequent [16]. Similarly, Aitboutargante et al. also identified ischemic heart disease as the main etiology, contrasting with our cohort in which dilated cardiomyopathy and valvular disease predominated [15].

## Treatment and management

Acute RV failure management focuses on early recognition and treatment of triggers such as sepsis and arrhythmias, with etiology-specific interventions (e.g., revascularization for infarction and reperfusion for high-risk PE) [29]. Management principles include optimizing preload, improving RV contractility, and reducing RV afterload. This may involve cautious fluid administration in hypotensive patients with low filling pressures, diuresis for congestion, sodium restriction, and close monitoring to maintain euvolemia. Rhythm and rate control are important, with restoration of sinus rhythm when feasible. Vasopressors

(notably norepinephrine) and inotropes (dobutamine, milrinone, levosimendan) can be used to improve hemodynamics, alongside optimization of oxygenation and ventilation. Interventional therapies depend on the cause and clinical status, including extracorporeal membrane oxygenation (ECMO), right ventricular assist device (RVAD), left ventricular assist device (LVAD), and pulmonary thromboendarterectomy for chronic thromboembolic pulmonary hypertension (CTEPH), embolectomy for massive PE, balloon atrial septostomy in refractory RV failure, and transplantation for advanced disease [21].

In our series, therapeutic patient education and systematic reinforcement of lifestyle and dietary measures were provided to all patients by the treating physician, reflecting a consistent emphasis on non-pharmacological management as part of routine care. Loop diuretics were used frequently across studies, confirming their central role in heart failure management. In our study, 100% of patients received furosemide, underscoring that congestion control is central to RHF management. Furosemide was prescribed in 78.6% at follow-up in Kambiré et al. [30], and 84.3% of patients received diuretics in Maggioni et al. [31]. Moroccan series reported similarly high rates: 95.5% in Aitboutargante et al. [15] and 99% at admission (54% at discharge) in Kheyi et al. [16].

For mineralocorticoid receptor antagonists, 69.8% of our patients received spironolactone, similar to Maggioni et al. (68.3%) but higher than Moroccan reports (42% in Aitboutargante [15] and 58% in Kheyi [16]). This may reflect our cohort profile with prominent congestion, where spironolactone is often used for symptomatic benefit, while acknowledging that evidence supporting its routine use in isolated RHF remains limited.

For renin-angiotensin system inhibitors, ACEis were prescribed in 62.8% of our patients, similar to Aitboutargante et al. (62.5%) [15], and consistent with other reports (Maggioni [31] and Kheyi [16]). This high rate likely reflects concomitant standard indications such as LV dysfunction, systemic hypertension, or ischemic heart disease. In our cohort, ramipril was the most commonly used ACEi (85.2%), followed by perindopril (14.8%); Maggioni et al. reported ramipril in 43.9%, enalapril in 27.5%, and perindopril in 14.5% [31]. Only 2.3% of our patients received an ARB, possibly because of good ACEi tolerance and the absence of a specific validated indication for ARBs in isolated RHF. Beta-blockers were prescribed in 72.1% of our patients, reflecting frequent coexisting guideline-based indications rather than RHF alone. SGLT2 inhibitors and ARNIs were rarely prescribed despite 23 patients with LVEF ⩽40%, probably because of cost and availability. Digoxin was used in 9.3% of cases, suggesting selective use, consistent with the absence of strong evidence supporting routine digoxin therapy in isolated RHF. No patient underwent CRT or received an ICD; these indications came from concomitant LV dysfunction, as device therapy has no validated indication in isolated RHF. Regarding in-hospital outcomes, the mean length of stay in our series was 11.60 ± 6.81 days, comparable to Moroccan studies in global heart failure (Aitboutargante 10 ± 5 days [15]; Kheyi 12.1 ± 6.6 days [16]). In-hospital mortality was 4.7%, also similar to Moroccan reports (4.5% Aitboutargante [15]; 6.1% Kheyi [16]).

For improved outcomes, early recognition and coordinated care involving cardiologists, critical care specialists, interventional cardiology, and cardiothoracic surgery is advisable.

### Study limitations

This study has several limitations. Its single-centre, retrospective design and modest sample size limit precision and restrict the generalizability of our findings. RHF was classified clinically, and many patients had concomitant LV dysfunction and radiographic pulmonary congestion or pleural effusion, so some biventricular involvement cannot be excluded. Etiologies and triggers were taken from the charts; the patients overlapped with a previously described cohort [10], and there was no follow-up after discharge. Reliance on chart review entailed incomplete or missing data, and some echocardiographic parameters, notably RVEF, were not systematically available because of technical constraints. The resource-limited setting curtailed access to advanced diagnostics such as cardiac magnetic resonance imaging and right heart catheterization, as well as to device-based therapies, which may have led to misclassification or underestimation of certain etiologies and disease severity. These limitations should be addressed in larger, multicentre, prospective studies.

## Conclusions

In this single-centre retrospective study of 43 adults hospitalized for predominantly right-sided heart failure in southern Morocco (2021-2023), dilated cardiomyopathy and valvular heart disease were the leading etiologies, and decompensation was most often related to potentially modifiable factors, particularly poor adherence, followed by infection and arrhythmia. Echocardiography frequently showed reduced LVEF, pulmonary hypertension, and RV systolic dysfunction. Management relied mainly on diuretic-based decongestion, while advanced investigations and device-based therapies were limited by availability. By providing real-world data from a resource-limited setting where RHF remains under-described, our study highlights the burden, dominant causes, and modifiable triggers of RHF and suggests potential priorities, notably structured patient education and adherence interventions and better access to specialized diagnostics and advanced therapies.

# Additional Information

## Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

**Concept and design:** Aymane Sebbar, Mohamed El Minaoui, Houssam Aitboutargante, Khalil Abderrahmane Elbaz, Wassim Beladel, Amine El Houari

**Acquisition, analysis, or interpretation of data:** Aymane Sebbar, Mohamed El Minaoui, Houssam Aitboutargante, Khalil Abderrahmane Elbaz, Wassim Beladel, Amine El Houari

**Drafting of the manuscript:** Aymane Sebbar, Mohamed El Minaoui, Houssam Aitboutargante, Khalil Abderrahmane Elbaz, Wassim Beladel, Amine El Houari

**Critical review of the manuscript for important intellectual content:** Aymane Sebbar, Mohamed El Minaoui, Houssam Aitboutargante, Khalil Abderrahmane Elbaz, Wassim Beladel, Amine El Houari

**Supervision:** Aymane Sebbar, Mohamed El Minaoui, Houssam Aitboutargante, Khalil Abderrahmane Elbaz, Wassim Beladel, Amine El Houari

## Disclosures

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