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# Impact of Atrial Fibrillation on Heart Failure in a Moroccan Population

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**Abstract** **Original Research Article**

***Introduction:*** Atrial fibrillation (AF) and heart failure (HF) represent two intrinsically interrelated entities, each promoting the development and exacerbation of the other. Their coexistence exposes patients to an increased risk of cardiovascular morbidity and mortality. The objective of this study was to analyze the impact of AF on the clinical, hemodynamic, and etiological profile of patients hospitalized for heart failure. ***Methods:*** This retrospective, descriptive, and analytical study was conducted between January 2022 and December 2024 at the Mohammed VI University Hospital Center (CHU) in Marrakech. It included 321 patients hospitalized or followed for documented HF. Characteristics were compared between patients with AF (AF+ group, n=104) and those in sinus rhythm (AF– group, n=217). ***Results:*** The prevalence of AF at admission was 32.4%. Patients in the AF+ group were significantly older (63.8 vs. 59.9 years; p=0.013) and had a higher prevalence of hypertension (49.0% vs. 26.3%; p<0.0001) and obesity (19.2% vs. 6.5%; p=0.001). Clinically, severe dyspnea (NYHA III–IV) and right ventricular failure predominated in the AF+ group. Paradoxically, the mean left ventricular ejection fraction (LVEF) was higher in the presence of AF (36.4% vs. 33.7%; p=0.039), with a higher prevalence of HF with preserved ejection fraction (HFpEF: 22% vs. 8.2%) and near-universal left atrial dilatation (84%). Ischemic cardiomyopathy predominated in sinus rhythm (69%), while valvular (26.9%) and arrhythmia-induced etiologies (8.7%) were significantly more frequent in the AF+ group. ***Conclusion:*** AF imparts a distinctive clinical and hemodynamic imprint characterized by a more severe presentation and a specific etiological profile, underscoring the central role of atrial myopathy.

**Keywords:** Atrial fibrillation; Heart failure; Atrial myopathy; Ejection fraction; Valvular heart disease.



## 1. INTRODUCTION

Heart failure (HF) and atrial fibrillation (AF) represent two cardiovascular pandemics of the twenty-first century, whose rising prevalence constitutes a major challenge for healthcare systems worldwide. With more than 64 million individuals affected globally by HF and over 59 million by AF, their frequent coexistence is not coincidental: these two conditions maintain a complex bidirectional relationship in which each inexorably worsens the other [1,2].

HF creates an anatomical and neurohumoral substrate conducive to the development of AF, through atrial dilatation and fibrosis, elevated filling pressures, and activation of the renin–angiotensin–aldosterone system (RAAS). Conversely, AF aggravates the clinical picture of HF by impairing cardiac output through loss of the atrial contribution to ventricular filling, tachycardia, and irregularity of the cardiac cycle [3,4].

Epidemiologically, the prevalence of AF in HF patients ranges from 10% to 50% across series, increasing with the severity of HF [5]. In Africa, and particularly in the Maghreb region, data remain sparse, with specific etiologies dominated by valvular heart disease—often related to acute rheumatic fever—and hypertensive cardiomyopathy [6].

The objective of the present study was to analyze the impact of AF on the demographic, clinical, hemodynamic, and etiological profile of patients admitted for heart failure at our center, in order to better characterize this population and identify particularities specific to our context.

## 2. PATIENTS AND METHODS

### 2.1. Study Design and Period

This is a retrospective, descriptive, and analytical study. It involved an exhaustive review of

medical records from 321 patients hospitalized or followed in outpatient consultation for documented heart failure (HF). The study was conducted over a three-year period, from January 2022 to December 2024, at the Department of Cardiology, Mohammed VI University Hospital Center (CHU) in Marrakech.

### 2.2. Study Population

Inclusion criteria: All adult patients, regardless of sex, with a confirmed diagnosis of HF according to the criteria of the European Society of Cardiology (ESC 2021) were included. Patients with a pacemaker or implantable cardioverter-defibrillator, those with atrial flutter or other supraventricular tachycardias, and those with incomplete clinical or paraclinical data were excluded.

**Group allocation: To address the study objective, the study population was divided into two distinct groups based on the admission electrocardiogram (ECG):**

- AF group: Patients presenting with atrial fibrillation at admission or with a known history of permanent/paroxysmal AF. (AF+, n=104)
- SR group: Patients admitted in regular sinus rhythm. (AF–, n=217)

### 2.3. Data Collection

Data were systematically extracted from electronic and/or physical medical records. Variables collected and analyzed included:

- Sociodemographic data and cardiovascular risk factors: age, sex, hypertension, diabetes mellitus, obesity, smoking, dyslipidemia.
- Clinical data: NYHA functional class, predominant type of HF (left, right, biventricular), presence of palpitations.
- Electrocardiographic data: baseline rhythm (AF or sinus).
- Transthoracic echocardiographic data: LVEF (classified per ESC 2021 as HFrEF <40%, HFmrEF 40–49%, HFpEF ≥50%), left atrial (LA) and left ventricular (LV) dimensions.
- Laboratory data: hemoglobin (Hb), estimated glomerular filtration rate (eGFR) using the CKD-EPI equation.
- Etiological profile: ischemic, valvular, arrhythmia-induced (tachycardia-mediated cardiomyopathy), hypertrophic, immunologic, or undetermined.

### 2.4. Statistical Analysis

Data entry was performed using Microsoft Excel. Statistical analysis was carried out using SPSS version 26.0 and R.

**Descriptive analysis:** Qualitative (categorical) variables were expressed as frequencies and percentages (%). Quantitative (continuous) variables were expressed as means ± standard deviations (SD) for normally distributed data, or as medians with interquartile ranges otherwise.

**Univariate and comparative analysis:** Comparisons between the AF group and the SR group were performed using appropriate statistical tests:

- The Chi-square ($\chi^2$) test or Fisher's exact test was used for comparison of proportions (categorical variables).
- Student's t-test or the non-parametric Mann–Whitney U test was used for comparison of means (continuous variables).
- Significance threshold: A p-value <0.05 was considered statistically significant for all analyses.

## 3. RESULTS

### 3.1. Demographic Characteristics and Cardiovascular Risk Factors

Between January 2022 and December 2024, 321 patients were enrolled (mean age 61.1 ± 12.9 years; 66.9% male). At admission, 32.4% presented with AF (AF group) and 67.6% were in sinus rhythm (SR group). Comparative analysis revealed that patients in the AF group were significantly older (63.8 vs. 59.9 years; p=0.013). Regarding cardiovascular risk factors, hypertension (49.0% vs. 26.3%; p=0.0001) and obesity (19.2% vs. 6.5%; p=0.001) were highly significantly associated with the arrhythmia. No statistically significant differences were observed for sex, diabetes mellitus, smoking, or dyslipidemia (Table 1).

**Table 1. Demographic characteristics and cardiovascular risk factors (* p<0.05 significant; SD: standard deviation; HTN: hypertension)**

| Characteristic | Total population (N=321) | AF+ group (n=104) | AF– group (n=217) | p-value |
|---|---|---|---|---|
| Age (years), mean ± SD | 61.1 ± 12.9 | 63.8 ± 13.6 | 59.9 ± 12.3 | 0.013* |
| Male sex, n (%) | 214 (66.9%) | 63 (61.2%) | 151 (69.6%) | 0.115 |
| Hypertension, n (%) | 108 (33.6%) | 51 (49.0%) | 57 (26.3%) | <0.0001* |
| Diabetes mellitus, n (%) | 129 (40.3%) | 50 (48.1%) | 79 (36.6%) | 0.113 |
| Obesity, n (%) | 34 (10.6%) | 20 (19.2%) | 14 (6.5%) | 0.001* |
| Smoking, n (%) | 137 (42.7%) | 40 (38.5%) | 97 (44.7%) | 0.348 |
| Dyslipidemia, n (%) | 135 (42.1%) | 41 (39.4%) | 94 (43.3%) | 0.588 |

### 3.2. Clinical Presentation

AF+ patients exhibited significantly more severe symptomatology. NYHA class III–IV dyspnea was present in 64.4% of the AF+ group versus 47.5% of the AF– group (p=0.004). AF was also associated with a higher prevalence of right ventricular failure (32.7% vs. 21.7%; p=0.033), biventricular failure (39.4% vs. 27.2%; p=0.027), and palpitations (72.1% vs. 28.1%; p<0.001) (Table 2).

**Table 2. Clinical presentation of patients with heart failure**

| Clinical parameter | AF+ (n=104) | AF– (n=217) | p-value |
|---|---|---|---|
| NYHA III-IV dyspnea, n (%) | 67 (64.4%) | 103 (47.5%) | 0.004* |
| Right ventricular failure, n (%) | 34 (32.7%) | 47 (21.7%) | 0.033* |
| Biventricular failure, n (%) | 41 (39.4%) | 59 (27.2%) | 0.027* |
| Palpitations, n (%) | 75 (72.1%) | 61 (28.1%) | <0.001* |

### 3.3. Echocardiographic and Laboratory Parameters

Mean LVEF was significantly higher in the AF+ group (36.4 ± 12.0% vs. 33.7 ± 8.3%; p=0.039). The prevalence of HFpEF (LVEF ≥50%) was significantly higher in the presence of AF (22% vs. 8.2%; p<0.001). Left atrial dilatation was near-universal in the AF+ group (84% vs. 59.4%; p<0.001). Regarding laboratory parameters, AF+ patients had a lower eGFR (56.9 ± 35.0 vs. 78.0 ± 42.8 mL/min/1.73m²; p<0.001) and lower hemoglobin levels (11.0 ± 2.0 vs. 12.1 ± 2.4 g/dL; p<0.001) (Tables 3 and 4).

**Table 3. Echocardiographic parameters**

| Parameter | AF+ (n=104) | AF– (n=217) | p-value |
|---|---|---|---|
| LVEF (%), mean ± SD | 36.4 ± 12.0 | 33.7 ± 8.3 | 0.039* |
| LVEF ≤40% (reduced), n (%) | 62 (59%) | 153 (70%) | 0.053 |
| LVEF 41–49% (mildly reduced), n (%) | 19 (18%) | 46 (21%) | 0.53 |
| LVEF ≥50% (preserved), n (%) | 23 (22%) | 18 (8.2%) | <0.001* |
| LV dilatation, n (%) | 51 (49%) | 103 (47%) | 0.74 |
| LA dilatation, n (%) | 88 (84%) | 129 (59.4%) | <0.001* |

**Table 4. Admission laboratory parameters**

| Laboratory parameter | AF+ (n=104) | AF– (n=217) | p-value |
|---|---|---|---|
| eGFR (mL/min/1.73m²), mean ± SD | 56.9 ± 35.0 | 78.0 ± 42.8 | <0.001* |
| Hemoglobin (g/dL), mean ± SD | 11.0 ± 2.0 | 12.1 ± 2.4 | <0.001* |

### 3.4. Etiology of Heart Failure

Ischemic cardiomyopathy predominated in the SR group (69% vs. 36.5%; p<0.001). Conversely, valvular heart disease (26.9% vs. 10%; p<0.001) and arrhythmia-induced cardiomyopathy (tachycardia-mediated; 8.7% vs. 0.9%; p=0.001) were significantly more frequent in the AF+ group (Table 5).

**Table 5. Etiological distribution of heart failure**

| Etiology | AF+ (n=104) | AF– (n=217) | p-value |
|---|---|---|---|
| Ischemic, n (%) | 38 (36.5%) | 151 (69%) | <0.001* |
| Valvular, n (%) | 28 (26.9%) | 22 (10%) | <0.001* |
| Arrhythmia-induced (CMP), n (%) | 9 (8.7%) | 2 (0.9%) | 0.001* |
| Undetermined, n (%) | 21 (20.2%) | 32 (14%) | 0.18 |
| Hypertrophic CMP, n (%) | 5 (4.8%) | 4 (1.8%) | 0.14 |
| Immunologic/Inflammatory, n (%) | 3 (2.9%) | 6 (2.7%) | 0.94 |

## 4. DISCUSSION

### 4.1. Prevalence, Demographic Profile, and Risk Factors

The prevalence of AF in our study was 32.4%, a figure consistent with international literature. The CHARM (Candesartan in Heart failure: Assessment of Reduction in Mortality and morbidity) trial reported an AF prevalence of 29% in HF patients across all disease stages [7], while more recent registries, such as the ESC Heart Failure Long-Term Registry, report prevalences ranging from 33% to 44% [8]. This prevalence increases with HF severity, being estimated at less than 10% in NYHA class I and reaching 40–50% in NYHA class IV [5].

The older age of AF+ patients (63.8 ± 13.6 years vs. 59.9 ± 12.3 years; p=0.013) is a consistent finding across all studies. Aging induces structural and electrophysiological atrial changes—fibrosis and shortening of the refractory period—that promote the initiation and perpetuation of AF [9]. In a meta-analysis

of 6,553 patients by Kotecha *et al.,*(2016), age was an independent predictor of AF in HF [10], and the risk of developing AF doubles with each decade beyond the age of 50 years.

The strong association between hypertension and AF in our study (49.0% vs. 26.3%; p<0.0001) is well-documented. Hypertension is recognized as the most important population-attributable risk factor for AF, accounting for approximately 14–22% of attributable cases [11]. The mechanisms involved include pressure overload leading to atrial hypertrophy and fibrosis, diastolic dysfunction, and activation of the RAAS, which exerts direct arrhythmogenic effects on the atrial myocardium [12]. The 2024 ESC Guidelines on AF identify hypertension as an essential comorbidity to manage within the framework of the 'ABC pathway' (Anticoagulation, Better symptom control, Cardiovascular risk factor management) [13].

Obesity was significantly more prevalent in the AF+ group (19.2% vs. 6.5%; p=0.001). The Framingham Heart Study established that each one-unit increase in body mass index (BMI) is associated with a 4% increase in AF risk [14]. The underlying mechanisms are multifactorial: epicardial adipose tissue infiltration into atrial tissue, increased intrathoracic pressure, RAAS activation, and a chronic pro-inflammatory state [15]. More recently, multiple studies have demonstrated that active weight loss in obese patients significantly reduces arrhythmia burden, supporting a causal role for obesity [16].

Regarding diabetes mellitus (48.1% vs. 36.6%; p=0.113), although our study did not reach statistical significance, the observed trend is consistent with meta-analyses reporting a relative risk of AF of 1.28 (95% CI [1.14–1.43]) in diabetic patients [17]. Diabetes promotes atrial fibrosis through the accumulation of advanced glycation end-products and oxidative stress, impairing atrial electrical conduction.

### 4.2. Clinical and Hemodynamic Impact

#### 4.2.1. Impact on Functional Tolerance and Cardiac Output

The increased clinical severity observed in our AF+ group corroborates international literature. The prevalence of NYHA III–IV dyspnea (64.4% vs. 47.5%; p=0.004) illustrates the direct detrimental impact of AF on exercise tolerance. The underlying pathophysiological mechanisms are multiple and synergistic. First, loss of atrial systole—the 'atrial kick'—reduces cardiac output by 15–30% depending on the degree of ventricular dysfunction [18]. Second, tachycardia and irregularity of the cardiac cycle shorten ventricular filling time, increasing filling pressures and congestive symptoms [19].

The high prevalence of palpitations in the AF+ group (72.1% vs. 28.1%; p<0.001) logically reflects the nature of the arrhythmia, but also underscores the importance of this symptom as a clinical warning sign, justifying systematic electrocardiographic monitoring in any HF patient presenting with this complaint. Early detection of AF, including in its paroxysmal forms, is now further supported by wearable devices and cardiac implants [21].

#### 4.2.2. Right Ventricular Failure and Atrial Functional Tricuspid Regurgitation

The higher frequency of right ventricular failure (32.7% vs. 21.7%; p=0.033) and biventricular failure (39.4% vs. 27.2%; p=0.027) reflects the impact of AF on right ventricular function. AF leads to chronic elevation of left atrial pressures, which propagates retrogradely to the pulmonary circulation, generating postcapillary pulmonary hypertension. This imposes pressure overload on the right ventricle, progressively impairing its systolic function [20].

More recently, the literature has identified a distinct and clinically significant mechanism linking AF directly to right-sided failure: atrial functional tricuspid regurgitation (AFTR) [22,23].

Unlike ventricular tricuspid regurgitation (which results from right ventricular dilatation secondary to pulmonary hypertension), AFTR is the direct consequence of right atrial remodeling induced by chronic AF. Permanent atrial fibrillation causes massive dilatation of the right atrium, which stretches the tricuspid annulus, leading to leaflet coaptation failure, even in the absence of left heart pathology or severe pulmonary hypertension [24].

This massive tricuspid regurgitation causes right ventricular volume overload and a marked rise in central venous pressure (CVP). Clinically, it manifests as severe right-sided failure (edema, ascites, hepatomegaly), as observed in our AF+ group. The development of AFTR represents a turning point with adverse prognostic implications, often inducing strong diuretic resistance [25].

### 4.3. The Ejection Fraction Paradox and Atrial Myopathy

#### 4.3.1. Paradoxically Higher LVEF in the Presence of AF

One of the most striking findings of our study is the paradoxically higher LVEF in the AF+ group (36.4 ± 12.0% vs. 33.7 ± 8.3%; p=0.039), with a higher prevalence of HFpEF (22% vs. 8.2%; p<0.001).

This phenomenon is consistent with large international registries reporting that HF patients with AF tend to have smaller end-diastolic volumes, more pronounced concentric hypertrophy, and less impaired overall systolic function compared to those in sinus rhythm [26]. Several pathophysiological and

epidemiological paradigms explain this close association between AF and higher LVEF spectra:

- ***Intolerance to Loss of Atrial Systole in HFrEF*:** Patients with severe HFrEF are critically dependent on the atrial contribution for filling their dilated, hypokinetic ventricle. The onset of AF often triggers immediate hemodynamic collapse (cardiogenic shock) and early mortality. Conversely, a heart with preserved or mildly reduced LVEF (HFmrEF) tolerates this loss better in the short term, allowing the arrhythmia to become established chronically and to be diagnosed during routine admissions [27].
- ***Echocardiographic Measurement and Variability*:** Recent publications incorporating artificial intelligence for echocardiographic cycle analysis highlight that LVEF may be underestimated in the presence of AF due to extreme R–R cycle variability. Once rhythm is regularized, the true LVEF is often found to be higher than initially estimated, suggesting that a portion of the systolic dysfunction attributed to AF may represent a measurement artifact or transient depression [28].

#### 4.3.2. HFpEF and Atrial Fibrillation: The Twin Expressions of Atrial Myopathy

HFpEF and atrial fibrillation share a common pro-inflammatory substrate. Systemic inflammation driven by comorbidities (obesity, hypertension, diabetes mellitus) stiffens the left ventricle, causing a dramatic rise in filling pressures that propagates to the left atrium [29]. Unable to tolerate this mechanical stress, the atrium undergoes massive dilatation and fibrosis—the process termed 'atrial myopathy.' AF is the direct electrical expression of these mechanical lesions [30]. Our data confirm this concept (atrial dilatation in 84% of AF+ patients), demonstrating that the atrium loses its reservoir function and becomes an inert, highly thrombogenic conduit.

### 4.4. Etiological Profile of Heart Failure

Etiological analysis reveals profoundly different profiles between the two groups, with major mechanistic and therapeutic implications.

#### 4.4.1. Predominance of Ischemic Etiology in Sinus Rhythm

Although ischemic cardiomyopathy is the leading overall cause of HF globally (reflecting the increasing prevalence of risk factors in Morocco) [31], it is paradoxically less frequent in the AF+ group (35.6% vs. 69% in sinus rhythm). This suggests that isolated macrovascular ischemia is a less potent trigger for chronic AF compared to prolonged mechanical overloads (such as severe hypertension or valvular disease), which induce far deeper and more arrhythmogenic atrial remodeling.

#### 4.4.2. Valvular Heart Disease and AF: A Historic Association

Valvular heart disease is markedly more frequent in the AF+ group (26.9% vs. 10% in sinus rhythm). This finding reflects the epidemiological reality of Morocco and North Africa, still heavily burdened by the sequelae of acute rheumatic fever (ARF)—in contrast to high-income countries. Rheumatic involvement (primarily mitral stenosis) combines a mechanical obstruction with autoimmune-mediated inflammation that destroys the left atrial architecture. This remodeling leads almost inevitably to permanent AF, substantially worsening the prognosis of these patients, who face a major risk of cardioembolic stroke [32]. The 2021 ESC Guidelines on valvular heart disease emphasize the importance of early correction of severe valvular lesions to prevent AF development [33].

#### 4.4.3. Arrhythmia-Induced Cardiomyopathy

Arrhythmia-induced cardiomyopathies (tachycardia-mediated cardiomyopathy) are significantly more frequent in the AF+ group, illustrating the bidirectional relationship between AF and heart failure. Rapid, poorly controlled AF can independently induce ventricular dysfunction through myocardial metabolic exhaustion (ATP depletion, calcium handling abnormalities, oxidative stress) [34]. Identifying this entity is clinically crucial, as—unlike other cardiomyopathies—this dysfunction is largely reversible if strict, prompt rate or rhythm control is implemented [35].

Recent data from the CASTLE-AF trial and the meta-analysis by Turagam *et al.,* demonstrate that catheter ablation of AF significantly improves LVEF and quality of life in this patient subgroup, with reductions in mortality and HF hospitalizations [36,37].

### 4.5. Study Limitations

Several limitations inherent to the study design must be acknowledged. The retrospective, single-center nature of the study introduces selection bias and limits generalizability to other centers or regions. The sample size limits statistical power to detect subtle associations (e.g., diabetes mellitus). The absence of systematic prolonged ECG monitoring may have underestimated the prevalence of paroxysmal or subclinical AF. The unavailability of BNP/NT-proBNP measurements across the entire cohort limits prognostic stratification. Finally, the lack of longitudinal follow-up precludes assessment of the impact of AF on mortality and rehospitalization rates.

## 5. DECLARATIONS

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**Conflicts of interest:** The authors declare no conflicts of interest related to this article.

**Author contributions:** All authors contributed equally to the drafting of the manuscript and approved the final submitted version.

**Data availability:** The data supporting this study are available from the corresponding author upon reasonable request.

## 6. CONCLUSION

This study confirms that atrial fibrillation imparts a distinctive clinical, hemodynamic, and etiological imprint on the profile of patients with heart failure. AF+ patients are older, more hypertensive, more obese, clinically more severe, and present an etiological profile dominated by valvular heart disease and arrhythmia-induced cardiomyopathy. The paradox of a higher LVEF in the AF+ group, combined with near-universal left atrial dilatation, underscores the concept of atrial myopathy as the central substrate of this complex relationship.

These findings advocate for an integrated management approach, in which treatment of AF cannot be dissociated from that of HF, with rigorous control of modifiable cardiovascular risk factors, optimization of guideline-directed medical therapy for HF, and, where eligible, a rhythm control strategy through catheter ablation. Prospective multicenter studies and national registries are warranted to better characterize this interaction within our specific epidemiological context.

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