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Research Article

Clinical Profiles and One-Year Prognosis of Heart Failure in a Sub-Saharan Country of Africa

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ABSTRACT

Background: New classification of heart failure according to ejection fraction calls for exploring in black Africans.

Objectives: To determine our patient's characteristics and prognosis of the subtypes of heart failure.

Methods: We analysed data from consecutive black African patients hospitalised for heart failure at Heart Institute of Abidjan in 2018 and followed up for one year.

Results: Were considered 251 heart failure patients (age: 55.5 ± 16.3 years, 63.7% of males) with preserved (HFpEF) (18.7%), mid-range (HFmrEF) (17.6%), and reduced ejection fraction (HFrEF) (63.7%). HFpEF patients were older (p <0.0001) and had more frequently. acute pulmonary oedema. From an echocardiographic point of view, HFpEF patients had, on average, a smaller left ventricle than the other patients (p <0.001), but a larger left atrium (p <0.05). Clinically, these patients were admitted more often with acute pulmonary oedema (p = 0.01) and had more often comorbidities (p=0.00)4. However, survival was better with HFpEF patients than HFrEF patients (log-rank = 4.61; p = 0.032). HFmrEF patients have an intermediate profile.

Conclusion: In our context, although they have the same expression, HFrEF and HFPEF appear very different. We need further studies for a better understanding of HFmrEF.

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Introduction

Despite advances in its knowledge, heart failure with preserved ejection fraction (HFpEF) remains an enigma for cardiologists [1, 2]. To better understand this clinical entity, the European Society of Cardiology has introduced the concept of heart failure with mid-range ejection fraction (HFmrEF) since its last recommendations in 2016 [3, 4]. Some western authors have described regional and racial variations regarding HFpEF [5, 6]. These two subtypes of heart failure are less often described than heart failure with reduced ejection fraction (HFrEF) in Sub-Saharan Africa [7]. Recently, an article noticed the higher frequency of HFrEF [8]. The fact that heart failure presents some particularities in our regions could presage differences with the subtypes already described elsewhere. Our work aimed to analyse the characteristics and the prognosis of these different subtypes of heart failure classified according to the ejection fraction.

Methods

We carried out a prospective cohort study from January the 1st, 2018, to December the 31st 2019, on patients admitted at the Abidjan Heart Institute, the unique cardiology center of the country, in the medicine or intensive care units.

I Selection Criteria

The selection began on January the 1st and ended on December the 31st of 2018. Patients included were black African, adults, whose principal diagnosis at admission was heart failure, confirmed by the Framingham criteria [9].

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We did not include patients hospitalised for less than 24 hours, and those with postoperative heart failure. We classified the patients into three categories of heart failure: HFrEF (left ventricle ejection fraction (LVEF) <40%), HFmrEF (LVEF between 40 and 49%) and HFpEF (LVEF ≥ 50%). HFmrEF and HFpEF must also have at least two of the following three characteristics: NTproBNP elevation> 125 pg / mL, left ventricular or left atrial echocardiographic enlargement, and an abnormality in diastolic function (E / A <1 or E / E '> 15). We excluded from the group of HFpEF, severe valvular heart disease, isolated right heart failure, hypertrophic cardiomyopathy, infiltrative diseases, and pericarditis.

II Parameters Collection

We collected sociodemographic and clinical data within 48 hours of admission. The etiological diagnosis was that retained at the end of the patient's stay. The treatment took into account the main cardiovascular medications prescribed during the length of stay.

The comorbidities were extracted from the patient files. Comorbidity indicates a chronic medical condition or a risk factor existing simultaneously but independently with the heart failure in our patients. This condition or risk factor may have a negative impact on heart failure course. We have distinguished cardiovascular comorbidities (high blood pressure, atrial fibrillation, peripheral arterial disease, cerebrovascular disease) and non-cardiovascular ones (diabetes, anaemia, renal failure, chronic pulmonary disease, HIV infection, cancer, connectivitis, hyperuricemia, dyslipidaemia, dementia). The number of these comorbidities per patient was evaluated.

High blood pressure was selected according to WHO criteria [10]. Diabetes was considered when the patient was known diabetic or when his fasting blood sugar was higher than seven mmol/l twice. Atrial fibrillation was diagnosed using a 12-lead electrocardiogram. Any patient with intermittent claudication with the abolition of the pulse was considered to have a peripheral arterial disease. It was the same diagnosis for those who suffered from ischaemia or arterial thrombosis or an abdominal or a thoracic aneurysm. Brain damage included hemorrhagic, and established or transient ischaemic strokes. The obesity interested patients with a body mass index upper than 30 Kg /m^2 of body surface area. A glomerular filtration rate of less than 90ml / min / 1.73m^2 indicated a renal failure [11]. Haemoglobin below 130 g/l in men and 120 g /l in women defined anaemia. Hepatic dysfunction assumed a patient with a significant elevation of transaminases (≥ 3 times beyond standard value) or impairment of liver function. A level of uric acid beyond 60 mg /l defined hyperuricemia. Dyslipidaemia: was retained before a total cholesterol level greater than or equal to 6.2 mmol/l or LDL cholesterol greater than 4.1 mmol/l or HDL cholesterol less than 1.1 mmol/l or triglyceride greater than 2.26 mmol/l. We have subdivided the dyslipidemia group in three subgroups to compare their prevalence among our patients: Patient with hyper LDLemia (LDL cholesterol > 4.1 mmol/L), Patient with hypoHDLemia (HDL cholesterol < 1.1 mmol/L) and patient with hypertriglyceridemia (Triglycerides > 2.26 mmol/L). Was considered as HIV patients, any patients with HIV positive serology, whether or not he had AIDS. We considered as coronary artery disease's patients, those who experienced a documented acute coronary artery syndrome, patients with angina and with evidence of myocardial ischaemia on stress testing or resting electrocardiography or patients with at least 1 coronary artery stenosis of at least 70% at coronarography.

III Follow-up

Patients discharged from the hospital had scheduled visits every three months. We evaluated the monitoring over one year through the medical records, which recorded the date of the last visit. For patients absent from their 12-month appointment, we call them or their relatives. The patients lost to follow-up were those not seen at the 12-month visit and impossible to contact after three phone calls.

Prognostic variables: To assess the prognosis, we used all-cause of deaths in one year.

IV Statistical Analysis

The data was analysed using SPSS © version 23. Characteristics of the study population were presented using descriptive statistics. We explored the relationships between the different types of heart failure and the sociodemographic or clinical variables. The chi2 test or Fisher's exact test evaluated the categorical variables and analysis of variances (ANOVA) the continuous variables. The difference in time before death between the different subtypes of heart failure was analysed by the Kaplan-Meier curve associated with a bilateral log-rank test to compare the curves.

The Cox proportional hazard approach was used to model the death time and the different subtypes of heart failure, and eight other known prognostic factors for heart failure: age, systolic blood pressure, shock or low cardiac output, atrial fibrillation, serum creatinine> 265 µmol/L, glomerular filtration rate <60 ml/min/1.73 m^2 (moderate renal failure), serum sodium <130 mmol / L, and serum potassium> 5.5 mmol / l. After univariate Cox analysis, we have constructed a multivariate model in the input mode to eliminate the confounding factors. The analysis was done with the intent to treat. A value of $p < 0.05$ was considered to be statistically significant.

V Ethical Considerations

The design was approved by the ethics committee of the Abidjan Heart Institute (Ref: YYK/GK N° 021-2017/MSHP/ICA/DMS. We collected the data after a detailed explanation of the study to patients and their relatives. Verbal consent was obtained during hospitalisation and over the phone if necessary. The records were analysed following the laws of the data protection of individuals under the ethical principles from the declaration of Helsinki.

Results

Out of 302 previously selected patients, 51 patients didn't match to the definition of HFpEF and HFmrEF due to structural cardiac abnormalities (Figure 1). Of the 251 patients, 160 (63.7%) had HFrEF, 44 (17.6%) had HFmrEF, and 47 (18.7%) had HFpEF. The ejection fraction had a bimodal distribution (Figure 2) with the first mode at 0.30 and a second mode at 0,61.

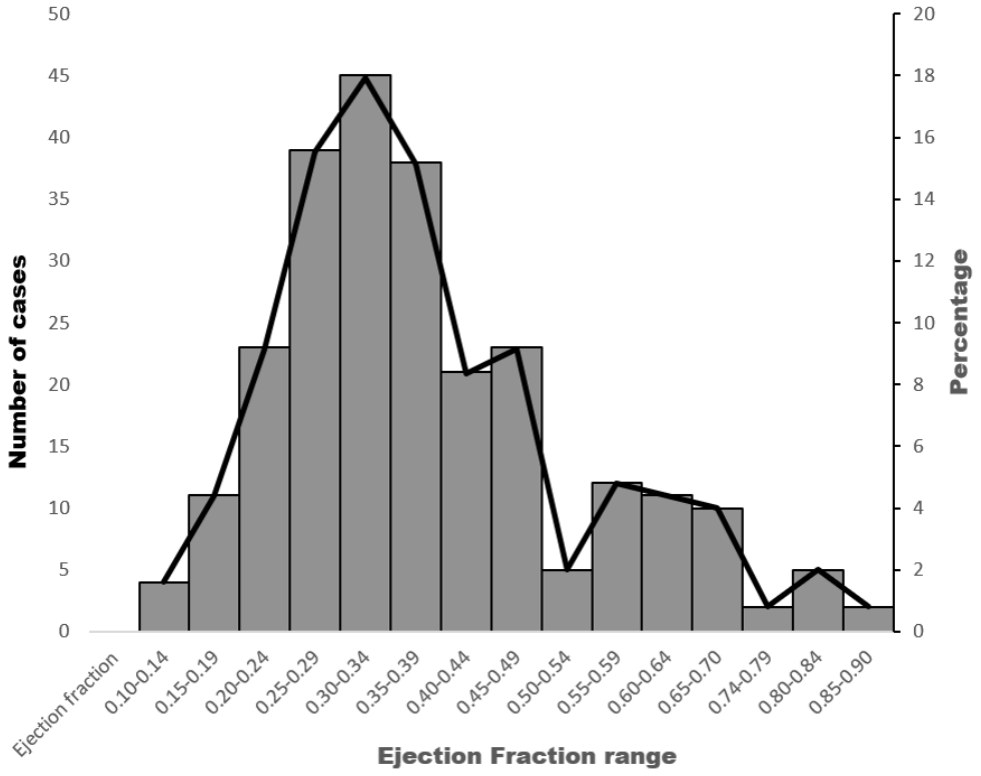


Figure 1: Distribution of left ventricular ejection fraction of heart failure patients by range of 0.05.

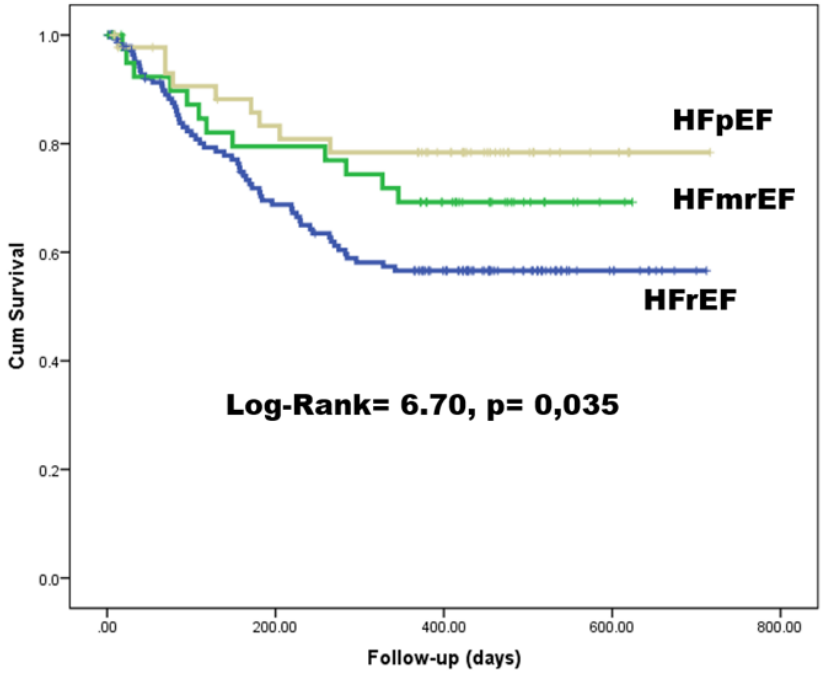


Figure 2: Kaplan-Meier survival curves for all-cause mortality in cohorts with HFpEF, HFmrEF, HFrEF.

I Patients Characteristics Overall

Our cohort had an average age of 55.5 ± 16.3 years with a male predominance (64.5%) (Table 1). 47.4% of patients were at their first episode of heart failure. Patients were most often admitted in biventricular heart failure (65.4%) at the NYHA IV stage (92.8%). High blood pressure was the leading cause of heart failure (48.6%) and was also the prominent comorbidity (53.8%). The exit treatment for these patients included in first-line, diuretics (98.4%), renin-angiotensin blockers (89.6%), and aldosterone antagonists (70.1%) (Table 2). 52.6% of patients were taking beta-blockers at discharge. The mean length of stay was 8.5 ± 5.2 days.

Table 1: Demographic and clinical characteristics overall and by heart failure subtypes.

Study type	Overall N=251	HFpEF n=47	HFmrEF (n= 44)	HFrEF (n= 160)	P
Demography/history					
Sex (Males) (%)	162 (64.5)	30 (63.8)	24 (54.5)	108 (67,5)	0.28
Age (Years)	55.5 ± 16.3	62.1 ± 17*	60 ± 15.2	52.2 ±15.5 §	< 0.001
Age > 70 years	51 (20.3)	18 (38.3)*	12 (27.3)	21 (13.1)) §	< 0.001
First episode of HF (%)	119 (47.4)	28 (59.6)*	26 (59.1)	65 (40.6) §	0.017
History of hospitalisation	46 (18.3)	8 (17)	8 (18.2)	30 (18.8)	0.96
Tobacco (%)	24 (9.6)	2 (4.3)	1 (2.3)	21 (13.1)	0.037
Physical exam					
NYHA 4	233 (92.8)	150 (93.8)	42 (95.5)	41 (87.2)	NS
SBP (mm Hg)	138.9 ± 43.4	159.8 ± 37.8*	145 ± 36.9	130.9 ± 44	<0.001
SBP ≥ 140 mm Hg	128 (53.3)	32 (71.1)	27 (65.9)	69 (44.8)	0.002
SBP < 100 mm Hg	300 (12.1)	1 (2 .1)	2 (4.5)	27 (16.9)	0.006
DBP (mm Hg)	90.9 ± 26.4	95.8 ± 22.8	94.3 ± 23.2	88.5 ± 26.4	0.16
Pulse pressure (mm Hg)	48 ± 24.7	64.1 ± 27.9*	51.1 ± 22.7 †	42.4 ± 22 §	< 0.001
Heart Rate (bpm)	101.3 ±28.5	96.7 ± 27.2	106.7 ± 28.8	101.2 ± 28.8	
Heart Rate > 120 bpm	62 (25.2)	9 (19.9)	14 (32.6)	39 (24.8)	0.36
Acute Pulmonary Edema (%)	35 (13.9)	13 (27.7)*	4 (9.2) †	18 (11.3)	0.01
Biventricular heart failure	164 (65.3)	20 (42.6)*	33 (75) †	111 (69.4)	0.001
Cardiogenic shock	23 (9.2)	1 (2.1)*	2 (4.5)	20 (12.5) §	0.04
Echocardiography					
LVEDD (mm)	62.3 ± 10	50 ± 8.1*	59.1 ± 7.6†	66.7 ± 7.3 §	< 0.001
LVESD (mm)	50.6 ± 12.1	32.2 ± 7.3*	45.5 ± 7.1†	57.3 ± 7.3 §	< 0.001
LVEF	0.38 ± 0.15	0.63 ± 0.08*	0.45 ± 0.03†	0.29 ± 0.06 §	< 0.001
Left atrium (mm)	47.2 ± 8.3	46.3 ± 11.2	46.7 ± 8.7	47.7 ±7.3	0.6
Left atrium enlargement	44 (17.9)	16 (34.8)	9 (20.9)	19 (12.1)	0.002
Pulmonary Hypertension	126 (50.4)	14 (30.4)	20 (47.5)	92 (57.5)	0.004
Biology					
Glycemia (mmol/L)	6.88 ± 4.05	6.94 ± 3.94	7.44 ± 6.10	6.71 ± 3.50	0.61
Haemoglobin (g/dl)	12.5 ± 2.4	12.2 ± 2.7	11.8 ±2.6	12.8 ± 2.2	0.03
Natremia (mmol/L)	135.2 ± 6.1	136 ± 6.4	135.8 ± 5.3	134.8 ± 6.1	0.32
Natremia < 130 mmol/L	41 (16,3)	3 (6.4) *	3 (6.8)	35 (21.9) §	0.007
Kaliemia (mmol/L)	4.2 ± 0.7	4.3 ± 0.9	3.9 ± 0.5	4.2 ± 0.7	0.02
Kaliemia > 5.5 mmol/L	11 (4.4)	5 (10.6)	0 (50)	6 (3.8)	0,038
Creatinin (µmol/L)	129.9 ± 103.4	178.6 ± 187.4*	116.7 ± 63.6†	119.3 ± 68.9	0.01
eGRF (ml/min)	65.1 ± 30.5	62.1 ± 33.5	62.1 ± 23.6	66.8 ± 30.6	0.51
Severe renal failure [1]	30 (12)	10 (21.7)	2 (4.8)	18 (11.8)	0.05
Aetiology					
Dilated CM	66 (26.3)	1 (2.1)*	5 (11.4)	60 (37.5) §	< 0.001
Hypertensive CM	129 (48.6)	29 (61.7)*	24 (54.5)	69 (43.1)§	< 0.001
Coronary Heart Disease	24 (9.6)	8 (17.6)	6 (13.6)	10 (6.3)	0.52
comorbidities					
High Blood Pressure	136 (53.8)	36 (74.5)*	30 (68.2)	70 (43.8)§	< 0.001
*Diabetes Mellitus (%)	41 (16.3)	12 (25.5)*	10 (22.7)	19 (11.8) §	0.038
PAD	10 (4%)	1 (2.8)	3 (6.8)	6 (.3.8)	NS
Obesity	31 (12.4)	14 (29.8)*	4 (9.1) †	13 (8.1)	< 0.001
Dyslipidemia	95 (37.8)	19 (40.4)	20 (45.5)	56 (35)	0.41
HyperLDLemia	18 (7.2)	6 (12.8))	6 (13.6))	17 (10.6)	0.82
Hypo HDLemia	66 (26.3)	12 (17.5)	15 (34.1)	39 (24.4)	0.43
Hyper triglyceridemia	4 (1.6)	2 (4.3)	0 (0)	2 (1.3)	#
Hyperuricemia	86 (34,3)	19 (40.4)	13 (29.5)	54 (33.8)	0.53
*Renal failure	111 (44.2)	22 (42.5)	21 (47.7)	68 (48.8)	0.76
Anemia	101 (40.1)	22 (46.8)	19 (43.2)	60 (37.5)	0.47
VIH infection	29 (11.6)	5 (10.6)	2 (4.5)	22(13.8)	0.23

Atrial Fibrillation	70 (27.9)	17 (36.2)	15(34.1)	38 (23.8)	0.15
Stroke	25 (10)	3 (6.4)	3 (6.8)	19 (11.9)	0.4
Hepatic dysfunction	67 (26.7)	5 (10.6)*	8 (18,8)	53 (33.8) §	0.003
*Number of Comorbidities	3.6 ± 1.8	4.2 ± 1.7*	3.9 ± 2.1	3.3 ± 1.7	0.004
Length of stay in hospital	8.5 ± 5.2	7.1 ± 2.9	7.8 ± 4.6	8.4 ± 5.8	0.31

HFmrEF, heart failure with mid-range ejection fraction; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; SBP, systolic blood pressure; DBP, diastolic blood pressure; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; LVEF, left ventricular ejection fraction; eGFR, estimated glomerular filtration rate; CM: cardiomyopathy; PAD: peripheral artery disease. 1.eGRF < 30 ml/min/1.73 m2. # Comparison cannot be performed. Number of cases insufficient.

*p <0.05 for HFpEF vs. HFrEF

†p <0.05 for HFmrEF vs. HFpEF.

§p <0.05 for HFmrEF vs. HFrEF

Table 2: Treatment characteristics overall and by heart failure subtypes.

Treatment	Overall N=251	HFpEF n=47	HFmrEF (n= 44)	HFrEF (n= 160)	P
Diuretics (%)	247 (98.4)	45 (95.7)	44 (100)	158 (98.8)	0.23
ACE I / ARB drugs	225 (89.6)	42 (89.4)	40 (90.9)	144 (89.4)	0.95
BetaBlockers (%)	132 (52.6)	18(38.3)*	26 (59.1)	88 (55)	0.08
Digitalis (Digoxin) (%)	124 (49.4)	12 (25.5)*	18 (40)	94 (58.8) §	< 0.001
Nitrates (%)	139 (55.4)	35 (74.5)*	26 (59.4)	78 (48) §	0.007
Aldosterone antagonist(%)	176 (70.1)	25 (53.2*	32 (72.2)	119 (74.4)	0.019
Length of stay in hospital	8.5 ± 5.2	7.1 ± 2.9	7.8 ± 4.6	8.4 ± 5.8	0.31

HFmrEF, heart failure with mid-range ejection fraction; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction. ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker.

*p <0.05 for HFpEF vs. HFrEF

†p <0.05 for HFmrEF vs. HFpEF.

§p <0.05 for HFmrEF vs. HFrEF

II Comparison of HFpeF, HFmrEF, and HFrEF

HFpEF patients were more often older than HFrEF patients (p <0.001) (Table 1). They were most often at their first episode of heart failure (59.6 vs 40.6%, p <0.05). Clinically, their systolic blood pressure and pulse pressure were significantly higher than those of HFrEF patients (p <0.001). Acute pulmonary oedema was more common in this first group. From an echocardiographic point of view, HFpEF patients had, on average, a smaller left ventricle than the other patients (p <0.001), but a larger left atrium (p <0.05). In terms of comorbidities, high blood pressure (74.5% vs 43.5%, p <0.001), diabetes (25.5% vs 11.8%, p =0.038) and obesity (29.8% vs 8.1%, p < 0.001) predominated in the HFpEF group compared to the HFrEF group. Overall, the number of comorbidities was significantly higher in the first group cited. Compared to HFrEF patients, HFpEF patients received more often nitrates (p = 0.007) but less often digitalis (p <0.001) and aldosterone antagonist p <0.05) (Table 2).

The HFmrEF patients presented globally intermediate data between those of HFpEF patients and those of HFrEF patients. Their data were broadly comparable to that of HFpEF patients except for the clinical presentation of heart failure. Biventricular heart failure was more frequent, and acute pulmonary oedema less prevalent in the HFmrEF group compared to the HFpEF group (p <0.05)). But HFmrEF patients were significantly different from HFrEF patients, particularly concerning age, history of heart failure, pulse pressure, high blood pressure, diabetes, aetiologies, digitalis and nitrates (p <0.05).

III Prognosis of Heart Failure

During the one-year follow-up, 79 patients (31.5%) died (67.1 male). Thirty-five patients (13.9%) were lost to follow-up. We performed a univariate and multivariate analysis with the different subtypes of heart failure according to the ejection fraction, and factors are known to be associated with the prognosis of heart failure (Table 3). In a univariate analysis, it appears that the risk of death was associated with the three subtypes of heart failure grouped: RR = 1.52; 95% CI [1.1 - 2.1], p = 0.012 (not in the table). Divided into three categories, they retained their high mortality risk (p = 0.0041). HFpEF patients had the lowest risk. That of the HFmrEF patients was not significantly different from those HFpEF patients (p = 0.37). However, the risk of HFrEF patients were significantly greater: RR = 2.290; 95% CI [1.134 - 4.623].

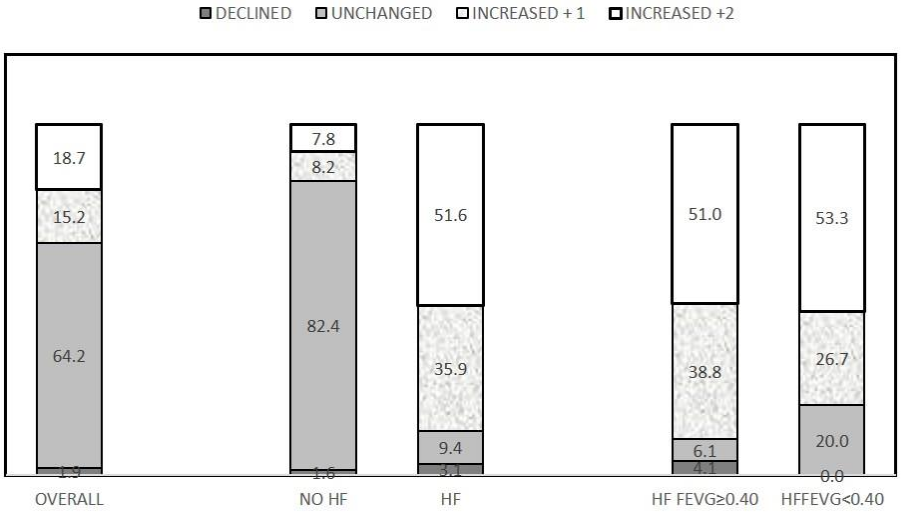
Table 3: Univariate and Multivariate predictors of death in patients with heart failure according to Cox model analysis.

	Univarate Analysis			Multivariate analysis		
	p	RR	95%CI	p	RR	95%CI
Age	0.559	1.004	(0.991 -1.017)	0.034	10.018	(1.001 - 1.034)
Systolic Blood Pressure	0.049	0.995	(0.990 -1.000)	0.674	0.998	(0.992 - 1.006)
Shock/ low cardiac output	0.003	2.665	(1.406 - 5.053)	0.556	1.364	(0.485 - 3.834)
Atrial Fibrillation	0.059	0.581	(0.331 - 1.020)	0.114	0.602	(0.321 - 1.130)

Creatininemia > 265 mmol/l	<0.001	4.861	(2.471 – 9.561)	<0.001	5.109	(2.155 - 12.109)
eGRF< 60 ml/min	0.015	1.755	(1.115 - 2.762)	0.767	1.083	(0.639 - 1.837
Natremia< 130 mmol/l	0.001	2.483	(1.479 – 4.170))	0.042	1.865	(1.022 - 3.405
Kaliemia >5.5 mmol/l	0.003	3.239	(1.486 – 7.061)	0.021	2.838	(1.173 - 6.867)
HFpEF	0.041			0.031		
HFmEF	0.37	1.485	(0.626 - 3.524)	0.056	2.658	(0.976 -7.235)
HFrEF	0.021	2.290	(1.134 - 4.623	0.009	3.276	(1.352 - 7.934)

HFmrEF, heart failure with mid-range ejection fraction; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction.

The other risk factors for death apart from age and atrial fibrillation were confirmed (p <0.05). In multivariate analysis, the risk of death was always associated with the different subtypes of heart failure (p = 0.031) with an RR = 3.2276; 95% CI [1.352 - 7.934], p = 0.009. In this last analysis, age (p = 0.034), plasma creatinine level> 265 µmol / L, serum <130 mmol / L and serum potassium> 5.5 mmol / l remained also predictors of all-cause mortality in heart failure. Analysis of survival according to the category of heart failure using Kaplan Meier curves compared with a Log Rank test, shows that survival decreased significantly with these different subtypes of heart failure (Log- Rank = 6.695; p = 0.035) (Figure 3). Compared two by two, the HFpEF curve was significantly different from that of HFrEF (Log-rank= 4.99, p=0.025), which demonstrated a more reduced survival. But HFpEF curve and HFmrEF curve (Log-rank=1.57; p=0.21) and HFmrEF curve and HFpEF curve Log-rank=0.27; p=0.6) were similar.



Discussion

We find in our series the general characteristics of HFpEF patients: they are older subjects, with more comorbidities than HFrEF patients, but with a slightly more favourable prognosis. HFmrEF patients are intermediate subjects in clinical presentation and prognosis. But they resemble more to HFpEF patients. For over 20 years, cardiologists have been interested in HFpEF, formerly known as diastolic heart failure, because of its high prevalence, its pathophysiology still poorly understood, and its difficult management [2, 12, 13]. Indeed, no therapeutic class has proven its effectiveness regarding this disease's morbidity and mortality. Our work, through these results, has allowed us to explore this affection in black Africans.

Several hypotheses raised about the pathogenesis of HFpEF. It would be the joint manifestation of different syndromes. However, HFpEF is most often considered as a single entity characterised by a systemic inflammatory state and microvascular endothelial dysfunction [14]. This entity has several phenotypes [15]. Western epidemiological data report a high prevalence of HFpEF exceeding 50% in some studies compared to 17.6% in our study [15, 16]. However, these results are to be qualified, most studies using only the LVEF as diagnostic criteria without sometimes excluding HFmrEF and often heart diseases which do not come under the new diagnostic criteria.

However, we must recognise that in sub-Saharan Africa, HFrEF predominates widely, sometimes reaching 75% in some studies [17-19]. Only Bonsu *et al.* in Ghana find a high level of HFpEF for the above reasons [20]. The comorbidities in our work were significantly more numerous in the HFpEF group. Samson *et al.* distinguish four phenotypes in HFpEF patients: the elderly and hypertensive, obese, coronary, and pulmonary phenotypes [14]. These last two phenotypes were uncommon in our cohort due to the small proportion of these aetiologies in our heart failure population. Clinically, we found higher pulse pressure in the HFpEF group. It results in an increase in arterial stiffness which contributes to the onset of heart failure [13].

We noticed a low prescription of aldosterone antagonists, and a high prescription of nitrates due to the high frequency of acute lung oedema in the HFpEF group. These attitudes appear to be contrary to good medical practice. We know that aldosterone antagonists are the only therapies to have partially improved HFpEF patients in studies and that nitrates are not recommended [13, 21, 22]. Concerning prognosis, some studies recognise that HFpEF has a slightly better prognosis than HFrEF,

others not [23, 24]. In our study, although, they have more comorbidities, HFpEF patients have a better prognosis. There is evidence which supports improved outcome in HFrEF with appropriate use of anti-remodelling therapy [25, 26]. In our case, HFrEF patients and HFpEF patients used the same proportion of ACE inhibitors/ Antagonist receptors of angiotensin 2 and Beta-blocker. Unfortunately, this data indicate treatment at discharge (and not after optimisation) and no dosage of drugs was evaluated.

The HFmrEF has an intermediate profile between the HFpEF and the HFrEF. But does this entity exist? The bimodal distribution of the LVEF that we found in our work and described in other studies would be in favour of 2 subtypes of heart failure and not 3 [16]. Then, HFmrEF appears closer to HFpEF in our work as in that of Koh *et al*. [27]. But it seems closer to HFrEF in other works [28, 29]. We believe that this proximity depends on the aetiologies of heart failure. We hypothesise that in this group, we have HFpEF patients who have a relatively low LVEF and HFrEF patients who slowly degrade their systolic function. Long-term cohort studies may help us better understand this condition.

Limitations

The relatively small number of patients, the number of patients lost to follow-up, and the severity of heart failure make the results difficult to be generalised. However, the forward-looking nature of this work and its original approach comparing the different subtypes of heart failure give unique pieces of information on African characteristics. Although a better prognosis than HFrEF, HFpEF and HFmrEF are severe conditions with limited symptomatic treatment. Active management of comorbidities could help to control and better, to prevent these subtypes of heart failure.

Conflicts of Interest

None.

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