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ORIGINAL RESEARCH

Impact of Type 2 Diabetes Mellitus on the Epidemiological-Clinical and Paraclinical Characteristics of Acute Heart Failure Seen at the Soavinandriana Hospital Center, Antananarivo Madagascar

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Background: The association diabetes mellitus - acute heart failure (AHF) is frequent and a source of significant morbidity and mortality.

Objective: The present study aimed to determine the impact of type 2 diabetes (T2DM) on the epidemiological-clinical and paraclinical characteristics of acute heart failure (AHF).

Methods: This was a retrospective cross-sectional study, carried out over a period of 2 years. The diagnosis of diabetes mellitus was made according to the criteria of the American Diabetes Association. The diagnosis of AHF is established by the signs and symptoms of heart failure; increased levels of brain natriuretic peptide (BNP); and systolic and/or diastolic dysfunction on echocardiography.

Results: 63 T2DM and 120 non-T2DM consecutive patients were selected. Age (≥50 years in men and ≥60 years in women) (OR=2.08 [1.31–5.14]), dyslipidemia (OR=3.95 [1.82–8.75]), microalbuminuria (OR=6.06 [1.69–27.3]) and overweight/obesity (OR=3.32 [1.33–13.5]) were more frequent in T2DM. The clinical profile of T2DM was marked by the rise in mean systolic arterial pressure (p=0.0368), arterial oxygen desaturation (p=0.0214), New York Heart Association (NYHA) IV breathlessness (OR=2.06 [1.04–4.08]); and paraclinical by left ventricular hypertrophy (OR=2.67 [1.24–5.77]), segmental kinetic disorder (OR=1.96 [1.04–-3.67]) and ischemic heart disease (OR=1.98 [1.09–3.92]). Diabetics received more statin (OR=2.06 [1.05–4.03]) and less spironolactone (OR=0.29 [0.13–0.64]).

Conclusion: T2DM is associated with poor profile of AHF. Adequate management of cardiovascular risk factors, including diabetes, could thus minimize the occurrence of AHF and improve this profile.

Keywords: acute heart failure, cardiovascular risk factors, coronary artery disease, Madagascar, type 2 diabetes mellitus

Introduction

With a constantly increasing prevalence, diabetes mellitus is one of the global health emergencies of the 21st century.[1] The prevalence of its complications is bound to increase significantly. The risk of occurrence of cardiovascular accidents is significantly elevated in the presence of diabetes mellitus.[2] Heart failure (HF) is one of the cardiovascular pathologies that frequently manifests first in patients with type 2 diabetes mellitus (T2DM).[3]

In addition, T2DM and HF share common risk factors and comorbidities such as hypertension, coronary artery disease, kidney disease and obesity, may be responsible for the heart disease involved in HF,[4–6] and T2DM doubles the

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risk of occurrence of HF.[2,7] Moreover, HF itself is also a major cause of morbidity and mortality worldwide.[8] HF is said to be acute (AHF) if there is a rapid onset or worsening of its symptoms and/or signs.[9]

Thus, the concomitant presence of AHF and T2DM constitutes a major public health problem.[10] To compact the data on the extent of these two pathologies among the Malagasy population, we conducted this study to determine the impact of type 2 diabetes on the epidemiological-clinical and paraclinical characteristics of AHF.

Material and Methods

Study Design and Setting

This was a retrospective descriptive and analytical cross-sectional study, carried out in the Cardiovascular Diseases and Internal Medicine departments of the Soavinandrina Hospital Center (Military Hospital) in Antananarivo. These services are part of the references for the management of cardiovascular, internal medicine, metabolic and endocrine diseases in the capital and even the country (Madagascar). The study spanned a period of 2 years from November 2018 to February 2020.

Study Population

The study population (N) consists of the two groups of consecutive patients with AHF of which the 1st group were with T2DM (n1) and the 2nd group without T2DM (n2). The diagnosis of AHF is established by the signs and symptoms of heart failure; increased levels of brain natriuretic peptide (BNP); and systolic and/or diastolic dysfunction on echocardiography.[9] The diagnosis and typing of diabetes mellitus are confirmed by the criteria of the American Diabetes Association (ADA).[11]

Were excluded from this study: other types of diabetes mellitus; patients with diseases whose clinical manifestations could be confused with those of AHF such as anemia, chronic obstructive pulmonary disease, hepatic dysfunction, sleep apnea; and incomplete records.

Clinical and Laboratory Data

The parameters studied were demographic data (gender, age); T2DM (duration, glycated hemoglobin and associated degenerative complications); other cardiovascular risk factors (hypertension, age, smoking, dyslipidemia, menopause, microalbuminuria, overweight/obesity); the history of heart disease; and AHF (blood pressure, heart rate and arterial oxygen saturation in ambient air on admission, clinical manifestations, echocardiographic signs, decompensation factors, drug treatment received during hospitalization and the intra-hospital outcomes).

The enzymatic method was used for the determinations of glycated hemoglobin (Hb A1c) and serum lipids. Diabetes was said to be controlled if the Hb A1c was less than 7% (<53 mmol/mol). The estimated glomerular filtration rate (eGFR) (mL/min/1.73 m²) was calculated according to the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation. The diagnosis of diabetic kidney disease was confirmed in the presence of microalbuminuria ≥30 mg/24 hours and/or a reduction in eGFR <60 mL/min/1.73 m², in the absence of signs or symptoms of other primary causes of kidney damage, associated with long-term diabetes, with or without diabetic retinopathy.[12] The diagnosis of retinopathy is made in the presence of abnormalities on examination of the vitreous and the fundus after pupillary dilation. Peripheral diabetic neuropathy is suggested by the presence of symmetrical distal sensory symptoms beginning in the lower limbs and/or impaired foot sensitivity on examination with a 10 g monofilament.[13] The arteriopathy of the lower limbs was confirmed by arterial Doppler of the lower limbs. The presence of hypertension was certified by blood pressure ≥140/90 mmHg (based on an average of ≥2 measurements obtained on ≥2 occasions) or taking antihypertensive medication. Cardiovascular risk was estimated according to the criteria of the European Society of Cardiology (ESC) 2019. Low-density lipoprotein cholesterol (LDL-C) must be < 1.4 mmol/L (<55 mg/dL) in subject's very high risk; <1.8 mmol/L (<70 mg/dL) if at high risk; <2.6 mmol/L (<100 mg/dL) if at moderate risk and <3.0 mmol/L (<116 mg/dL) if at low risk.[14] Patients with LDL-C outside these targets or taking a lipid-lowering drug were considered to have dyslipidemia. The body mass index was calculated as the weight in kilograms (kg) divided by the square of the height in meters (m²). Overweight and obesity are defined by body mass index ≥ 25kg/m² and ≥ 30kg/m², respectively.

Statistical Analysis

Data was collected from a pre-established survey form from patient medical records. Then, they were used using Epi Info™ version 3.5.4 software (United States Centers for Disease Control and Prevention in Atlanta, Georgia). Qualitative and quantitative variables were respectively expressed as proportion and median with interquartile range [25% and 75%]. The chi-square test with a significance threshold less than 0.05 and the odds ratio (OR) were used to comparing the different variables between patients with and without T2DM. This OR is affected by a 95% confidence interval [95% CI].

Ethical Considerations

Before carrying out the study, a request for authorization to collect data was sent and granted by the General Director of the hospital and the head of department. Patients' anonymity and confidentiality were respected. Review Board of Soavinandriana Hospital approved this study (December 12, 2020). No written consent from patients is required as the data was collected retrospectively from patient medical record. The study adhered to the principles outlined in the Declaration of Helsinki.

Results

During the study period, there were 1494 patients hospitalized at the study sites. Among them, 201 were admitted for AHF, including 18 excluded from the study. Of the remaining 183 (N) patients, 63 (n1) were with T2DM and 120 (n2) without T2DM.

Table 1 presents the general characteristics of the population studied. Gender was not significantly different between the two groups (p = 0.3987). Mean ages of patients with and without T2DM were 61.6 ± 10.2 years and 60.9 ± 15 years,

Table 1 General Characteristics of the Population Studied

Variables	With T2DM (n1=63)	Without T2DM (n2=120)	OR [95% CI]	p Value
Male gender, n (%)	39 (61.9)	78 (65.0)	1.14 [0.57–2.24]	0.39876
Age groups, years				
[24–44], n (%)	4 (6.3)	18 (15.0)	0.38 [0.90–1.24]	0.0666
[45–64], n (%)	34 (54.0)	50 (41.7)	1.63 [0.84–3.17]	0.0763
[65–91], n (%)	25 (39.7)	52 (43.3)	0.86 [0.43–1.67]	0.3763
Glycated haemoglobin ≥ 7%, n (%)	49 (77.8)	—	—	—
Associated degenerative complications				
None, n (%)	26 (41.3)	—	—	—
Nephropathy, n (%)	22 (34.9)	—	—	—
Ischemic stroke, n (%)	6 (9.5)	—	—	—
Retinopathy, n (%)	3 (4.8)	—	—	—
Peripheral neuropathy, n (%)	3 (4.8)	—	—	—
Arteriopathy of the lower limbs, n (%)	1 (1.6)	—	—	—
Associated cardiovascular risk factors				
Age (≥50 years in men and ≥60 years in women), n (%)	53 (84.1)	86 (71.7)	2.08 [1.31–5.14]	0.0431*
Hypertension, n (%)	50 (79.4)	83 (69.2)	1.71 [0.79–3.85]	0.0721
Dyslipidemia, n (%)	25 (39.7)	17 (14.2)	3.95 [1.82–8.75]	0.0001*
Tobacco, n (%)	24 (38.1)	40 (33.3)	1.23 [0.62–2.42]	0.3147
Menopause, n (%)	21 (33.3)	28 (23.3)	1.63 [0.78–3.38]	0.1017
Microalbuminuria, n (%)	11 (17.5)	4 (3.3)	6.06 [1.69–27.3]	0.0001*
Overweight/obesity, n (%)	8 (12.7)	5 (4.2)	3.32 [1.33–13.5]	0.0363*
≥ 3 risk factors, n (%)	58 (92.1)	90 (75.0)	3.84 [1.36–13.4]	0.0021*
History of known heart disease, n (%)	19 (30.2)	37 (30.8)	0.97 [0.46–1.97]	0.5323

Note: *Statistically significant (p value <0.05).
Abbreviations: CI, Confidence Interval; OR, Odds ratio; T2DM, Type 2 diabetes mellitus.

respectively (p = 0.2346). T2DM was newly diagnosed in 19 patients (30.2%) and previously diagnosed in 44 patients (69.8%) whose mean duration of evolution was 5.5 ± 4.5 years (extremes: 0.5–23 years). Mean Hb A1c was 8.2 ± 1.6% (range: 6.8–15.4%). Apart from coronary artery disease, nephropathy was the most common chronic degenerative complication in 31.7% of cases. Note that a patient could have one or more complications.

Table 2 shows the clinical characteristics of acute heart failure. Mean systolic blood pressure was significantly higher in patients with than without T2DM (p value = 0.0368). Arterial oxygen saturation in ambient air (O2SaAA) below 90% was significantly more frequent in T2DM (OR = 2.75 [1.32–5.78]). Breathlessness was significantly more severe (New York Heart Association [NYHA] IV) in patients with than without T2DM (OR = 2.06 [1.04–4.08]), and cardiac murmur less present (OR = 0 0.28 [0.08–0.73]).

Table 3 presents paraclinical characteristics, aetiology and decompensation factors of acute heart failure. The mean BNP was 2066.9 ± 1076.4 ng/l and 1945.2 ± 876 ng/l in patients with and without diabetes, respectively. On transthoracic echocardiography, left ventricular filling pressure was elevated in both groups. Left ventricular hypertrophy, segmental kinetic disorder and ischemic heart disease were significantly more frequent in patients with T2DM (p = 0.0034; 0.0378 and 0.0367; respectively). The etiology related to hypertension was also significantly more frequent in patients with than without T2DM (55.6% versus 35%; OR = 2.31 [1.18–4.53]). In both groups, infections, acute coronary syndrome and tachyarrhythmias were the main factors of decompensation. No significant correlation was found.

Table 2 Clinical Characteristics of Acute Heart Failure

Variables	With T2DM (n1=63)	Without T2DM (n2=120)	OR [95% CI]	p Value
Mean SBP, mmHg	136.1 ± 24	130.6 ± 29	—	0.0368*
Mean DBP, mmHg	80.8 ± 18.3	80.9 ± 15.5	—	0.0791
Mean heart rate, bpm	93.1 ± 20.9	92.8 ± 20.8	—	0.7014
Mean arterial oxygen saturation, %	89.3 ± 7.3	92.3 ± 5.6	—	0.0214*
Arterial oxygen saturation <90%, n (%)	25 (39.7)	23 (19.2)	2.75 [1.32–5.78]	0.0018*
Breathlessness, n (%)	58 (88.9)	109 (90.8)	0.81 [0.26–2.06]	0.4287
NYHA IV, n (%)	29 (46.0)	35 (29.2)	2.06 [1.04–4.08]	0.0128*
NYHA III, n (%)	15 (23.8)	32 (26.7)	0.86 [0.39–1.82]	0.4077
NYHA II, n (%)	12 (19.1)	39 (32.5)	0.49 [0.21–1.07]	0.0377*
NYHA I, n (%)	0 (0)	2 (1.7)	ND	0.4287
Ankle swelling, n (%)	45 (60.8)	73 (71.4)	1.61 [0.79–3.31]	0.1031
Fatigue/tiredness, n (%)	16 (25.4)	23 (19.2)	1.43 [0.64–3.14]	0.2142
Gallop rhythm, n (%)	33 (52.4)	53 (44.2)	1.38 [0.72–2.68]	0.1835
Hepatojugular reflux, n (%)	33 (52.4)	63 (52.5)	0.99 [0.51–1.92]	0.5554
Elevated jugular venous pressure, n (%)	29 (46.0)	48 (40.0)	1.27 [0.65–2.47]	0.2646
Pulmonary crepitations, n (%)	50 (79.4)	94 (78.3)	1.06 [0.47–2.46]	0.5157
Hepatomegaly, n (%)	10 (15.9)	21 (17.5)	0.89 [0.34–2.15]	0.4773
Cardiac murmur, n (%)	6 (9.5)	33 (27.5)	0.28 [0.09–0.74]	0.0031*
Ascites, n (%)	5 (7.9)	7 (5.8)	1.13 [0.33–5.34]	0.3981

Note: *Statistically significant (p value <0.05).
Abbreviations: bpm, beat per minute; CI, Confidence Interval; DBP, Diastolic blood pressure; NYHA, New York Heart Association; ND, not defined; OR, Odds ratio; SBP, systolic blood pressure; T2DM, Type 2 diabetes mellitus. *p value < 0.05.

Table 3 Paraclinical Characteristics, Aetiology and Decompensation Factors of Acute Heart Failure

Variables	With T2DM (n1=63)	Without T2DM (n2=120)	OR [95% CI]	p Value
Mean brain natriuretic peptide, ng/l	1945.2 ± 876	2066.9 ± 1076.4	—	0.6157
Transthoracic echocardiography				
High LVFP, n (%)	63 (100)	120 (100)		
Left atrial dilation, n (%)	30 (47.6)	49 (40.8)	1.31 [0.67–2.54]	0.2344
Left ventricular hypertrophy, n (%)	22 (34.9)	20 (16.7)	2.67 [1.24–5.77]	0.0034*
Segmental kinetic disorder, n (%)	28 (44.4)	36 (30.0)	1.96 [1.04–3.67]	0.0378*
Global kinetic disorder, n (%)	16 (25.4)	40 (33.3)	0.68 [0.32–1.41]	0.1743
Mean LVEF, %	36.2 ± 15.4	33.3 ± 14.9	—	0.3123
LVEF reduced, n (%)	44 (69.8)	86 (71.7)	0.91 [0.44–1.91]	0.4623
LVEF preserved, n (%)	12 (19.1)	18 (15.0)	1.33 [0.54–3.18]	0.3074
LVEF mildly reduced, n (%)	7 (11.1)	16 (13.3)	0.81 [0.26–2.25]	0.4295
Dilated stage heart disease, n (%)	26 (41.3)	64 (53.3)	0.61 [0.31–1.19]	0.0812
Ischemic heart disease, n (%)	24 (38.1)	29 (24.2)	1.98 [1.09–3.92]	0.0367*
Hypertrophic cardiomyopathy, n (%)	7 (11.1)	10 (8.3)	1.37 [0.41–4.24]	0.3571
Valvular heart disease, n (%)	0 (0)	7 (5.9)	—	0.0489*
Others, n (%)	6 (9.5)	10 (8.3)	1.92 [0.13–7.11]	0.4269
Aetiology				
Coronary diseases, n (%)	47 (74.6)	83 (69.2)	1.31 [0.62–2.80]	0.2764
Hypertension, n (%)	35 (55.6)	42 (35.0)	2.31 [1.18–4.53]	0.0041*
Tachyarrhythmias, n (%)	16 (25.4)	24 (20.0)	1.35 [0.61–2.96]	0.2555
Heart valve disease, n (%)	5 (7.5)	17 (14.2)	0.52 [0.14–1.58]	0.1606
Metabolic disorders, n (%)	6 (9.5)	7 (5.8)	1.69 [0.44–6.19]	0.2625
Bradyarrhythmias, n (%)	1 (1.16)	4 (3.3)	0.47 [0.01–4.87]	0.4374
Toxic causes, n (%)	1 (1.6)	2 (1.7)	0.95 [0.02–18.62]	0.7204
Others, n (%)	1 (1.6)	1 (0.8)	1.91 [0.02–151.9]	0.5712
Decompensation factors				
Infections, n (%)	57 (47.5%)	31 (49.2%)	1.07 [0.56–2.06]	0.4743
Acute coronary syndrome, n (%)	26 (21.7%)	15 (23.8%)	1.13 [0.51–2.46]	0.4387
Tachyarrhythmia, n (%)	22 (18.3%)	9 (14.3%)	0.74 [0.28–1.83]	0.3177
Metabolic/hormonal disturbances, n (%)	11 (9.2%)	9 (14.3%)	1.64 [0.56–4.67]	0.2084
Hypertensive crisis, n (%)	4 (6.3)	8 (6.7%)	0.70 [0.12–3.06]	0.4381

Note: *Statistically significant (p value <0.05).
Abbreviations: CI, Confidence Interval; LVEF, left ventricular ejection fraction; LVFP, left ventricular filling pressure; OR, Odds ratio; T2DM, Type 2 diabetes mellitus.

Table 4 Drug Treatment During Hospitalization for Acute Heart Failure and Outcome

Variables	With T2DM (n1=63)	Without T2DM (n2=120)	OR [95% CI]	p Value
Drug treatment				
ACE inhibitors, n (%)	47 (74.6)	91 (75.8)	0.93 [0.44–2.04]	0.4949
Angiotensin receptor blocker, n (%)	15 (23.8)	28 (23.3)	1.03 [0.46–2.21]	0.5402
Diuretics, n (%)	63 (100)	120 (100)	–	–
Beta-blockers, n (%)	24 (38.1)	35 (29.2)	1.49 [0.74–2.97]	0.1444
Spironolactone, n (%)	12 (19.0)	53 (44.2)	0.29 [0.13–0.64]	0.0003*
Antiplatelet agents, n (%)	42 (66.7)	69 (57.5)	1.47 [0.74–2.95]	0.1475
Statins, n (%)	32 (50.8)	40 (33.3)	2.06 [1.05–4.03]	0.0118*
Preventive anticoagulation, n (%)	8 (12.7)	27 (22.5)	0.50 [0.18–1.23]	0.0778
Curative anticoagulation, n (%)	34 (54.0)	52 (43.3)	1.53 [0.79–2.96]	0.1124
Inotropes and/or vasopressors, n (%)	2 (3.2)	9 (7.5)	0.41 [0.04–2.05]	0.2032
Outcome				
Mean length of in-hospital stays, days	15.5 ± 11.1	14.4 ± 6.5	—	0.1097
Released with improvement, n (%)	53 (84.1)	107 (89.2)	0.64 [0.24–1.76]	0.2266
Released against medical advice, n (%)	6 (9.5)	5 (4.2)	2.41 [0.58–10.4]	0.1320
Deceased, n (%)	4 (6.3)	8 (6.7)	0.94 [0.20–3.72]	0.6018

Note: *Statistically significant (p value <0.05).
Abbreviations: ACE, Angiotensin-converting enzyme; CI, Confidence Interval; OR, Odds ratio; T2DM, Type 2 diabetes mellitus.

Table 4 shows drug treatment received during hospitalization for AHF and outcome. Almost all patients had received a renin-angiotensin-aldosterone system (RAAS) blocker and a diuretic. Spironolactone was prescribed significantly less in patients with than without T2DM (19% versus 44.2%; OR = 0.29 [0.13–0.64]). However, patients with T2DM more often benefited from a statin than without T2DM (50.8% versus 33.3%; OR = 2.06 [1.05–4.03]). Mean length of in-hospital stay was slightly longer in patients with than without T2DM (15.9 ± 11.1 days versus 14.4 ± 6.5 days), with respective mortality rates of 6.3% and 6.7%. Statistical tests were not significant.

Discussion

The problem of AHF, in the general population and even more so in the diabetic population, is undeniably taking on growing importance. Knowledge of the impact of type 2 diabetes on the epidemiological-clinical and paraclinical characteristics of AHF would therefore seem important. Recent evidence suggests that the evaluation of the hemodynamic HF phenotypes at hospital admission may allow an important prognostic risk stratification of each HF patient.[15]

In our study, gender and age of diabetics were not statistically different from non-diabetics. However, Targher et al had found a male predominance (p = 0.002) and a young age (p <0.001) in diabetics than in non-diabetics.[16] On the one hand, authors had found that women had a lower risk of AHF than men.[17] On the other hand, other authors have objected that diabetic women have an extremely high risk of coronary artery disease, stroke, cardiac mortality and all-cause mortality compared to diabetic men.[18] Moreover, in the Framingham study, diabetic subjects over the age of 65 years were at greater risk of developing cardiovascular diseases such as HF.[19] The absence of correlations in our study could be explained by the small size of our sample. Nevertheless, the search for AHF in a diabetic population, and vice versa, should be done without distinction of gender or age.

Regarding diabetes mellitus, our results concurred with those of Mwita et al in Botswana, where 44.4% of patients had newly diagnosed diabetes during hospitalization for AHF.[20] In a European cohort, diabetes was newly diagnosed only in 19.5%.[16] In fact, in low-income countries like ours, the diagnosis of diabetes mellitus is often delayed due to the difficulties encountered in systematic screening. The glycemic control of our patients was identical to that of the Botswana study who's average Hb A1c of their patients was 8.2%.[20] Diabetic nephropathy was the most common associated degenerative complication in our study and in the literature. Indeed, AHF and chronic renal failure are often associated in T2DM, mutually worsen and exert synergistic effects to increase the risk of cardiac and renal events.[21] This trio forms a vicious circle that must be broken to improve patient prognosis.[22]

Hypertension remains the cardiovascular risk factor most associated with T2DM.[16,20] As such, it represents a well-known risk factor for cardiac decompensation. And by chronically increasing afterload, it leads first to left ventricular hypertrophy and diastolic dysfunction and then to various structural abnormalities that culminate in AHF. Dyslipidemia, microalbuminuria and overweight or obesity were significantly frequent in our diabetics as in those of other authors.[16,23] Indeed, obesity is considered to be an independent factor of HF. The excessive accumulation of triglycerides in the cardiomyocytes of the obese subject is likely to lead to dysfunction of myocardial contractility and even structural abnormalities.[24] In addition, microalbuminuria is one of the main signs of diabetic nephropathy whose role in HF has already been described previously.[21,22] Finally, our diabetics significantly accumulated at least three cardiovascular risk factors. Thus, detection and early and adequate management of these other factors are essential to minimize the occurrence of heart failure in subjects with diabetes.

The mean systolic blood pressure of patients with T2DM was significantly higher than that of without T2DM in our study as in the literature[16] and mean heart rate was indifferent between the two groups.[25] Mean O2SaAA was lower in our patients with than without T2DM. This was not the case in the study by De Groote et al.[26] Patients education about the onset of symptoms remains essential to avoid reaching this advanced stage of the disease where there is a deterioration of vital parameters. In the present study, breathlessness was significantly more severe in patients with than without T2DM. Similarly, Targher et al had objectified that the proportion of NYHA III–IV breathlessness was 86.8% in diabetics and 83% in non-diabetics ($p < 0.001$).[16] The significantly lower proportion of heart murmurs in our patients with T2DM could be explained by the high frequency of valvular pathologies in patients without T2DM.

Biological markers were indifferent in diabetics than in non-diabetics, in our study as in another study.[16] However, in the study by Sarma et al, diabetics had a lower average serum BNP value than non-diabetics (p value = 0.05).[27]

On transthoracic echocardiography, ventricular hypertrophy, segmental kinetic disorder and ischemic heart disease were significantly more frequent in our patients with T2DM. This could be explained by the predominance of coronary diseases as etiologies of heart failure, especially in our diabetic patients. In the present study, the difference in mean LVEF of two groups was not statistically significant. However, Greenberg et al had also objectified that their diabetics had a slightly higher average LVEF compared to non-diabetics ($39.7 \pm 17.2\%$ versus $38.5 \pm 18\%$; $p < 0.0001$).[28] According to the literature, ischemic heart disease and hypertension remain the main aetiology of heart failure in developed countries, and rheumatic heart disease in low-income countries in Africa and Asia.[29] However, in our study, the most representative etiology of heart failure was coronary disease in both groups. Under the probable influence of globalization, the progressive Westernization of the lifestyle of subjects living in low-income countries like ours, could be the origin of this etiological profile. In addition, diabetes carries a relative risk of atherosclerosis of 2 to 4 for coronary artery disease.[30]

Even in the absence of a significant association, infections, acute coronary syndrome and tachyarrhythmias were the main decompensation factors of AHF for our entire study population. However, in the ALARM-HF study, the most common decompensation factor in diabetics was acute coronary syndrome, followed by arrhythmias, non-compliance with therapy and infections ($p < 0.0001$; 0.002 0.008 and 0.563, respectively).[31]

In our study as in the EVEREST study,[27] spironolactone was prescribed significantly less in diabetics compared to non-diabetics. This could be explained by the high frequency of renal failure in diabetics than in non-diabetics, most often contraindicating the prescription of spironolactone. Moreover, our diabetics benefited significantly more from statins than our non-diabetics, as in another study.[16]

As outcome, in our study, the in-hospital mortality rates of diabetics and non-diabetics were identical and statistically insignificant. However, in the ALARM-HF cohort, it was significantly higher in diabetics than in non-diabetics.[31] Indeed, diabetes mellitus is an independent factor of mortality in patients with heart failure.[32]

The present study has limitations. Its retrospective nature did not make it possible to collect important information which was not included in all medical records. The hemodynamic HF phenotypes were not evaluated among T2DM and not T2DM patients. Also due to the monocentricity of the study, the results we obtained cannot be extrapolated to the entire general diabetic population in Madagascar.

Conclusion

To conclude, patients with T2DM accumulated significantly more cardiovascular risk factors, represented mainly by age, arterial hypertension, dyslipidemia, microalbuminuria and overweight/obesity. T2DM were more prone to elevated systolic blood pressure, arterial oxygen desaturation and severe breathlessness. They significantly presented with left ventricular hypertrophy, segmental kinetic disorder and ischemic heart disease. Their therapeutic profile was marked by the more frequent prescription of statin and less frequent of mineralocorticoid receptor antagonist.

Early, adequate and multidisciplinary management of associated cardiovascular risk factors could thus minimize the occurrence of heart failure and improve its clinical and paraclinical characteristics in patients with diabetes mellitus. Given its limitations, conducting a prospective, multicenter study is useful to identify other parameters.

Disclosure

The author(s) report no conflicts of interest in this work.

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