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Idiopathic Dilated Cardiomyopathy at the Hospital De La Paix in Ziguinchor: About 79 Cases

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Abstract

Idiopathic dilated cardiomyopathies are the most frequent cardiomyopathies and represent a major public health problem. The aim of this work was to study the epidemiological, clinical, paraclinical, therapeutic and evolutionary aspects of idiopathic dilated cardiomyopathies at the Hospital de la Paix in Ziguinchor. We had conducted a retrospective and descriptive study at the cardiology department of the Hospital la Paix of Ziguinchor. We included in the study all cases of confirmed cardiomyopathies by echocardiography with diagnostic criterias as left ventricular dilatation ≥ 59 mm in men and 53 mm in women, associated with left ventricular dysfunction with an ejection fraction <45% without detectable etiology. We selected 79 patients with an average age of 61.51 years, there SAS a male predominance of 60.8%. Idiopathic dilated cardiomyopathies accounted for 35% of the causes of cardiomyopathies. Clinically, all our patients were in heart failure and exercise dyspnea was the main symptom. In the chest X-ray, cardiomegaly was found in 94.9% of cases and electrocardiogram left ventricular hypertrophy was the most common abnormality (41.8%). On transthoracic echocardiography Doppler, the average telediastolic diameter of the left ventricle was 63.08 mm and the average left ventricular ejection fraction was 32.46%. Medical treatment relied mainly on diuretics and angiotensin-converting enzyme inhibitors. The mortality rate was 19%. Idiopathic dilated cardiomyopathy is the most common type of cardiomyopathy. In the absence of adequate means of exploration in our developing countries, the diagnosis is based on simple anamnestic, clinical and paraclinical arguments, of which the Doppler echocardiogram is the cornerstone. The mortality rate remains high despite the therapeutic advances made in recent years.

Keywords: Idiopathic dilated cardiomyopathies; Heart failure; Left ventricular hypertrophy; Left ventricular fraction ejection; Cardiomegaly; Peace Hospital; Ziguinchor

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Introduction

Dilated cardiomyopathies (DCM) are characterized by direct damage to the heart muscle that causes dilation and impairment of left ventricular (LV) contraction or both ventricles [1,2]. They constitute the evolutionary term of a heterogeneous group of cardiac and extra cardiac pathologies. Their etiology may be primitive or secondary. The term Idiopathic DCM refers when etiology is not known and it is the most common form of cardiomyopathy. It is a major public health problem due to its morbidity and mortality [2,3]. Developing countries in the tropics are undergoing an epidemiological transition marked by an increase in chronic noncommunicable diseases while infectious diseases remain significant. In this context, DCMs are the main cause of heart failure in our regions. Idiopathic DCMs appear to be common in Africa, but epidemiological data are often lacking. We report the results of this study whose objective was to study the epidemiological, clinical, paraclinical, therapeutic and

evolutionary aspects of idiopathic DCMs in Ziguinchor, Southern region of Senegal.

Methods

This was a retrospective and descriptive study over a period of 27 months (01st of April 2015 to 30th of July 2017) at the Cardiology Unit of the Hospital de la Paix in Ziguinchor.

We included in the study all patients of both sexes hospitalized for DCM confirmed by Doppler echocardiography and without detectable etiology (coronary, valvular, hypertensive or congenital).

The diagnostic criteria were diastolic LV enlargement ≥ 59 mm (31 mm/m2) in men and 53 mm (32 mm/m2) in women with LV systolic dysfunction (ejection fraction <45%). We excluded from the study all DCMs that did not meet the criteria defined above, secondary cardiomyopathies and other cardiomyopathies. The study focused on the analysis of individual hospitalization records and we prepared a card that served as a support. The card included socio - demographic, clinical, paraclinical, therapeutic and evolutionary data. The data was analyzed using SPSS software in version 22.

Results

We had a total of 79 patient records during the study period. The average age of our patients was 61.51 years (range 33-93 years) and male patients were predominant (60.8%). The frequency of idiopathic DCMs in hospital during the study period was 1.46%. Idiopathic DCMs accounted for 35% of the causes of cardiomyopathy during the study period and 19.6% of the causes of hospitalization for heart failure. The majority of our patients were unemployed (29.1%) and of low socioeconomic status (60.8%). The average consultation time was 44.92 days (range 1 to 190 days).

Stage IV dyspnea was the most common functional sign **(Table 1)** and all our patients had cardiac failure on admission. Edema of the lower extremities was the main sign found at the clinical examination **(Table 2).**

The pattern of heart failure was progressive in most cases (91.1%) and was most often congestive heart failure (54.4%). On the electrocardiogram, left ventricular hypertrophy (LVH) was the most common abnormality **(Table 3)**.

The average cardio - thoracic ratio (CTR) was 0.63 (range 0.51 to 0.85) and radiological cardiomegaly was found in 94.9% of cases. On echocardiography, the average telediastolic diameter of the LV was 63.08 mm (range 55-85 mm) and the average left ventricular ejection fraction was 32.46% (range 15-44%). **Table 4** summarizes the main abnormalities found on the cardiac Doppler ultrasound. The medical treatment was essentially based on diuretics and angiotensin-converting enzyme inhibitors **(Table 5).** The average duration of hospitalization was 6.8 days (range 02 to 21 days). Mortality rate was 19% during hospitalization. LV diameter > 70 mm, Pulmonary Arterial Hypertension (PAH) with Pulmonary Arterial Pressure (PAP)> 55 mmHg, and the presence of intra-cavitary thrombi were poor prognostic factors of our study.

Table 1 Functional signs.

Signs	Number	Percentage %
Stage III dyspnea	34	43
Stage IV dyspnea	45	57
Precordialgia	26	32.9
Asthenia	18	22.8
Hemoptysis	5	6.3

Table 2 Physical signs.

Signs	Number	Percentage %
Turgid jugular veins	50	63.3
Tachycardia	42	53.2
Heart murmur	21	26.6
Gallop heart sound	6	7.6
Crackling rattles	47	59.5
Hepatomegaly	41	51.9
Ascites	19	24.1
Lower limbs edema	59	74.7

Table 3 ECG signs.

Signs	Number	Percentage %
Atrial fibrillation	10	12.7
Left branch block	19	24.1
Left ventricular hypertrophy	33	41.8
Appearance pseudo – necrosis	9	11.4
Atrial flutter	1	1.3
Ventricular extrasystole	1	1.3

Table 4 Doppler echocardiogram.

Signs	Number	Percentage %
Mitral insufficiency ≥ 3/4	29	36.7
Left ventricular high pressures	51	64.6
Right ventricle systolic dysfunction	21	26.6
Pulmonary arterial hypertension	36	45.6
Intracardiac thrombus	4	5.1
Pericardial effusion	21	26.6

Table 5 Medical treatment.

Treatment	Number	Percentage %
Diuretics	79	100
Beta – blockers	13	16.5
Converting enzyme inhibitors	75	94.9
Angiotensin II receptor antagonists	2	2.6
Digoxin	23	29.1
Anticoagulants	14	17.7
Antiaplatelet	37	46.8

Discussion

Idiopathic DCMs are an entity that groups together all the myocardial dysfunctions related to a structural or functional

Table 6 Main identified genetic mutations involved in familial dilated cardiomyopathies.

Gene	Chromosomal location	Additional possible phenotype
Autosomal dominant transmission		
Cardiac actin	15q14	Any
Desmin	2q35	Peripheral myopathy
B and d sarcoglycan	Sq33- 35	Muscular dystrophy
Heavy chain β myosin	14q1	Any
Cardiac Troponin T	1q32	Any
A – tropmyosin	15q22.1	Any
Lamine A/C	1p1 - q21	Anomaly of the conduction system
X – linked transmission		
Dystrophin	Xp21	Peripheral myopathy
Taffazine	Xp28	Small size and neutropenia
Autosomal recessive inheritance		
Desmoplakin	6p24	Hirsitusm and keratoderma

anomaly of the cardiac muscle not linked to a coronary, valvular, hypertensive or congenital disease [2,3]. The classification proposed until recent years was that of WHO. There are currently new classifications proposed by the American Heart Association and the European Society of Cardiology [4-6]. During the period of our study, idiopathic DCMs accounted for 1.6% of hospitalizations. Our results are close to those reported by BOUAKEZ [7] and GOEH-AKUE [8]. Idiopathic DCM appears to be a low-prevalence condition that affects approximately 2% of cardiac hospitalized patients according to LENEGRE [9]. The epidemiology of idiopathic DCM has been modified by the improvement of its diagnosis. It is currently being detected earlier, which explains the increase in its frequency and makes it the most frequent cardiomyopathy. It accounted for 35% of the causes of cardiomyopathies in our study. The average age of our patients was 61.51 years, higher than the other series [7,8-9] with a male predominance reported by all series.

The disease mainly affects young adults aged 30 to 40 years and more often men than women [1,3]. Clinically, all our patients were in heart failure and exercise dyspnea was the master-symptom. This clinical presentation is reported by several authors [7,8] and seems to be the circumstance of discovery of idiopathic DCM. According to the series, idiopathic DCMs account for 20 to 50% of hospitalization causes for heart failure [10,11]. On the chest x-ray, cardiomegaly was almost constant with an average ICT of 0.63 comparable to the results of BOUAKEZ [7] and GOEH - AKUE [8]. Chest X-ray is still of interest because the discovery of a large cardiomegaly is a guiding element in the diagnosis of DCM. The majority of electrocardiographic abnormalities found in our study were LVH (33%), left branch blocks (24.1%) and atrial arrhythmias (12.7%). Right bundle branch block (RBBB) was an independent predictor of all-cause mortality, and the combination of RBBB and Left ventricular end diastolic dimension (LVEDD) provided more clinically relevant information than RBBB alone for assessing the risk of all-cause mortality in patients with IDCM [12]. Electrocardiographic abnormalities are not specific however. The Doppler echocardiogram is the key examination to highlight the two main abnormalities of DCM: left cavitary dilatation and impairment of LV systolic function secondary to diffuse and homogeneous hypokinesia of parietal kinetics.

The etiopathogenesis of DCM is multifactorial and involves 3 main mechanisms: genetic abnormalities, infections (viral infections in particular) and autoimmune reactions. These 3 mechanisms can coexist [11].

The familial and monogenetic forms of DCM are characterized by a great heterogeneity. Their frequency has long been underestimated whereas they represent about 30% CMD. Their pathophysiology essentially involves a deficit of generation of myocardial force or a defect of transmission of this force. Different modes of transmission are possible whose dominant autosomal forms predominate **(Table 6)** [1,13-16].

Non-genetic dilated cardiomyopathies account for the majority of cases, about 70%. The causes are multifactorial. All infectious myocarditis can progress to CMD. In Europe, viral causes predominate whereas in Africa parasitic etiologies are more common. The progression from myocarditis to CMD is reported with a frequency varying from 7% to 52% of cases. Serological examinations and myocardial biopsy allow the pathogen to be investigated using molecular biology techniques. The viral genome can also be sought by polymerase chain reaction. New cardiac radiology techniques, including MRI, provide quality information and allow an anatomical approach to the myocardium [1,17].

Toxic causes are also incriminated including chronic and excessive consumption of alcohol and iatrogenic causes due to the side effects of antimitotics [17,18].

Inflammatory causes related to a systemic disease or an immune etiology may also occur in the genesis of DCM. Circulating anti-heart autoantibodies are recovered in 30% to 40% of patients with CMD. The main auto-antigens found are the myosin alpha and beta heavy chains, troponin I, mitochondrial antigens, beta 1 adrenergic receptors, M2 muscarinic receptors and Na-K adenosine triphosphatase (ATPase). Antibodies directed against these last three antigens could promote the occurrence of ventricular or atrial rhythm disorders [1,17-19].

It is difficult to assert the primacy of a DCM in the absence of coronary angiography or endomyocardial biopsy, as these invasive tests are not very accessible in developing countries. Diagnosis of idopathic DCM is often established after eliminating the causes of cavitary dilatation on simple anamnestic, clinical and paraclinical arguments.

In our study, the treatment was mainly based on diuretics, ACE inhibitors and a small proportion of patients who had benefited from beta - blockers because they were not available and were relatively expensive.

In the absence of etiological treatment, the treatment of idiopathic DCM is common to the treatment of any systolic heart failure and is perfectly codified by learned societies [Galinier, Logeart, Gweintraub, Meyer]. It relies on diuretics, in the presence of congestive signs, ACE inhibitors or angiotensin II receptor antagonists (ARA II) and beta-blockers concomitantly with hygiene and dietary measures.

Other interventions include enrolment in a multidisciplinary heart failure service, and device therapy for arrhythmia management and sudden death prevention. Patients who are refractory to medical therapy might benefit from mechanical circulatory support and heart transplantation. Treatment of preclinical disease and the potential role of stemcell therapy are being investigated [17].

The evolution was favorable in the majority of the cases but the mortality rate was 19%. Idiopathic DCMs are associated with a mortality rate ranging from 15 to 50% according to series [Galinier, Bouakez-Goeh, Tesson F] due to refractory heart failure or sudden death by rhythm disorder [20,21]. They are the main cause of heart transplantation in Europe.

Current therapeutic advances and earlier diagnosis of the disease have improved the prognosis with a survival rate of up to 80% at 5 years [20].

Conclusion

Idiopathic DCMs are the most common cardiomyopathies with a caricatural clinical expression. In the absence of invasive tests for formal diagnosis, simple clinical and paraclinical arguments are available to make the diagnosis in our developing countries. In our study, our patients were predominantly elderly and male. In addition, there were very long consultation periods and all our patients were in heart failure. Diagnosis was based primarily on anamnestic, clinical and echocardiographic data. The treatment is well codified and the evolution is currently improved by an earlier and adapted management. In our study, the use of beta blockers remained low and should be encouraged to improve the survival of our patients. This study highlights the difficulties encountered in the diagnosis and management of CMDs in our developing countries and encourages us to adopt a preventive policy against the cardiovascular risk factors but also the infectious diseases big suppliers of CMD.

Conflicts of Interest

The authors do not declare any conflict of interest.

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