Cardiology Research and Cardiovascular Medicine
Diallo M, et al. Cardiolog Res Cardiovasc Med 10: 274.
www.doi.org/10.29011/2575-7083.100274
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Research Article

Epidemiological and Phenotypic Profile of Primary Cardiomyopathies (PCM) using Magnetic Resonance Imaging (MRI) at the Dakar Military Training Hospital

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Citation: Diallo M, Diante D, Amar NI, Ndaw MDB, Diack A, et al. (2025) Epidemiological and Phenotypic Profile of Primary Cardiomyopathies (PCM) using Magnetic Resonance Imaging (MRI) at the Dakar Military Training Hospital. Cardiol Res Cardio vasc Med 10: 274. DOI: https://doi.org/10.29011/2575-7083.100274

Received Date: 09 February, 2025; **Accepted Date:** 18 February, 2025; **Published Date:** 21 February, 2025

Abstract

Introduction: Primary cardiomyopathies (PCM) are disorders in which the heart muscle is structurally and functionally abnormal in the absence of any other cardiovascular cause. They fall into four phenotypic groups: HCM (Hypertrophic Cardiomyopathy), DCM (Dilated Cardiomyopathy), RCM (Restrictive Cardiomyopathy) and ARVC (Arrhythmogenic Right Ventricular Cardiomyopathy). The assessment of cardiomyopathies is the first of the current indications for cardiac magnetic resonance imaging in the global cardiac MRI registry. The general objectives of this study are to assess the phenotypic aspects of CMP and specifically to define the epidemiological profile and describe the MRI semiology of the main CMPs. **Materials and Methods:** This is a retrospective, cross-sectional and descriptive study from January 2022 to March 2023, meaning it covers a period of 15 months, and it involves 46 patients. It was conducted in the medical imaging department of the Dakar Army Training Hospital (SENEGAL). **Results:** The mean age was 54 years with a sex ratio of 1.3. HCM and DCM were the most represented phenotypes with 46% and 39% respectively. 7 cases of phenotypic association were also pointed out. In HCM, the mean maximum end-diastolic thickness of the LV (Left Ventricle) was 20.2 mm, the topography of the parietal thickening was predominantly asymmetrical (90.5%) with an antero-septo-basal predominance. Alteration of LVEF (LV Ejection Fraction). (21%)? stenosis of the LV outflow tract (9.52%) and anterior systolic motion of the mitral valve (9.52%) were the most observed repercussions with dense intramyocardial fibrosis in 8 patients or 38.1%. The criteria for DCM were retained on the basis of LV dilation associated with an alteration of LVEF. The mean LV end-diastolic volumes were 222 ml and the mean LVEF alteration was 30.25%. This was associated with pericardial effusion and mitral reflux due to dilatation of the atrioventricular annulus in 28% and 17%, respectively. For RCM, of which amyloidosis is the typical example, LV myocardial hypertrophy was frequent with a mean of 17.4 mm. Diffuse interstitial fibrosis was objectified by T1 Mapping measurement, the mean of which was 1300 ms. The alteration of LVEF was 31% on average. A pericardial effusion was associated in 33.3% of patients. In ARVC, there was constant severe right ventricle (RV) dyskinesia with a mean RVEF (RV Ejection Fraction) of 16.25% and a mean RV end-diastolic volume of 88 ml/m^2. Regarding non-compaction of the LV, we found a mean ratio of non-compacted myocardium to compacted myocardium of 1.93 associated with an alteration of the LVEF with a mean of 31.5%. Late enhancement was present in 4 patients (66.7%). **Conclusion:** The profile of primary cardiomyopathies in our Senegalese population in MRI is not different from what is observed in the general population. MRI remains a benchmarking examination for the evaluation of cardiomyopathies.

Keywords: Cardiomyopathy; MRI; Military Training Hospital; Dakar; SENEGAL

Introduction

Primary Cardiomyopathies (PCM) are a structurally and functionally abnormal myocardium, in the absence of coronary artery disease, hypertension, valvular heart disease and congenital heart disease sufficient to cause the observed myocardial abnormality [1]. The European Society of Cardiology classifies cardiomyopathies according to the morphological and functional phenotype. The main types of cardiomyopathies are: hypertrophic cardiomyopathies (HCM), dilated cardiomyopathies (DCM), arrhythmogenic right ventricular cardiomyopathy (ARVC) and restrictive cardiomyopathies (RCM) [1,2]. Left Ventricular Noncompaction (LVNC) is no longer considered a PCM but a phenotypic trait. A new entity is to be noted: non-dilated left ventricular cardiomyopathy (NDLVC) [1]. New tools are now available for the diagnostic and prognostic management of patients. Therefore, CT and cardiac magnetic resonance imaging (MRI) have taken their place alongside ultrasound, nuclear medicine techniques (positron emission tomography and single-photon emission computed tomography) and coronary angiography for patient management [3]. Cross-sectional imaging, particularly MRI, is now essential for the diagnosis and monitoring of PCM [3]. The assessment of cardiomyopathies is the first of the current indications for MRI in the global cardiac MRI registry [4]. The practice of cardiac MRI in Senegal is recent due to a limited technical platform. Indeed, apart from the army training hospital, only two university and private hospitals have an MRI with cardiac activity. There is no study on the epidemiological and phenotypic aspects of primary cardiomyopathies (PCM) in Senegal, which serves as a ground for this retrospective study over a period of 15 months. The main objective of this work is to study the phenotypic aspects of CMP, its specific objectives are to define the epidemiological profile of CMP and to describe the MRI semiology of the main PCM.

Materials and Method

Our study was conducted in the medical imaging department of the Dakar Army Training Hospital. This is a retrospective, cross-sectional and descriptive study from January 2022 to March 2023, meaning a period of 15 months. We included all patients with PCM on cardiac MRI regardless of its indication, which aggregates to a total of 46 patients. All patients with cardiomyopathy without a certain phenotypic diagnosis based on the report were not included. Ischemic heart disease and myocarditis were excluded. The examinations were performed under cardiac monitoring with a 3 Tesla MRI from SIEMENS MAGNETOM Lumina using a phased array cardiac coil and ECG synchronization. The post-processing software was SyngoMR XA. The cardiomyopathy exploration protocol systematically included Ciné Trufi section plans in short axes, 04 cavities, 02 cavities or long axis right and left and three cavities. Parametric tissue characterization sequences (T1 and T2 Mapping) and late PSIR enhancement sequences performed 10 to 15 minutes after Gadolinium injection. The parameters studied were the epidemiological aspects (age and sex), the phenotypic distribution and the MRI semiology of PCM. For HCM, these were left ventricle (LV) wall thickness (value and topography), LV mass, LVEF, the presence of LV outflow tract stenosis, anterior systolic motion of the mitral valve, and late Gadolinium enhancement. For DCM, these were LV end-diastolic volume and LV ejection fraction (LVEF), and the presence of atrioventricular annulus dilatation, thrombus, and late Gadolinium enhancement. For amyloidosis, these were ventricular wall thickness, T1 Mapping values, and the presence of late Gadolinium enhancement. For ARVC, these were right ventricular (RV) end-diastolic volume and the presence of RV kinetics disorder. For LVNC, these were the ratio of noncompacted to compacted myocardium, LVEF, and late gadolinium enhancement. Data entry and analysis were performed using Microsoft® Excel software. Data are expressed as absolute values, percentages, mean, median, and were used to plot graphs.

Results

The mean age of our population was 54 years with extremes from 12 to 82 years. The age groups between 40-60 years and 60-80 years were the most represented with each 18 patients. (Figure 1). The distribution of ages according to the phenotypes was shown in Table 1 and the ages of the two patients diagnosed with ARCV were 23 years and 68 years. The distribution by sex showed a slight dominance of the male sex with a sex ratio of 1.3. Figure 2 shows the distribution of patients by sex and phenotypic aspect. HCM was the majority phenotype with 21 patients or 46%, followed by DCM with 18 patients or 39%. ARVC was the least represented with 2 patients (4%). Figure 3 shows the phenotypic distribution of PCM. There were also 7 cases of phenotypic association with 4 cases of DCM associated with non-compaction of the LV (57%), 2 cases of DCM associated with amyloidosis (29%) and one case of DCM associated with amyloidosis (14%).

	Average	Standard deviation	Median	Minimal	Maximum
HCM	58.0	12.9	62.0	30.0	78.0
DCM	49.3	18.6	48.5	15.0	82.0
Amyloidosis	64.8	12.7	68.0	49.0	79.0
NCLV	38.7	22.4	40.5	12.0	69.0

Table 1: Age distribution according to phenotype.

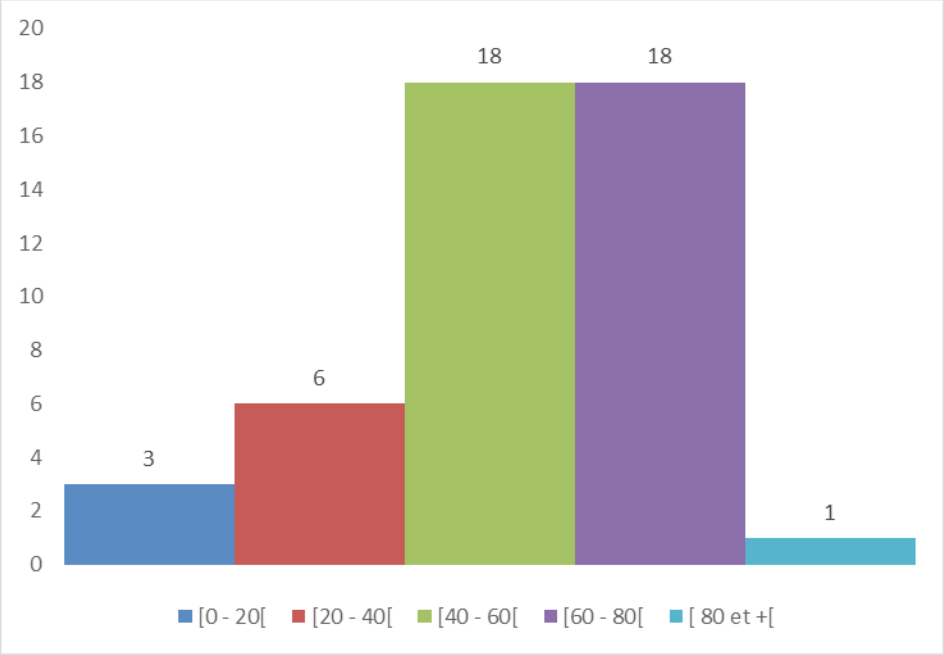

Figure 1: Distribution of patients by age group.

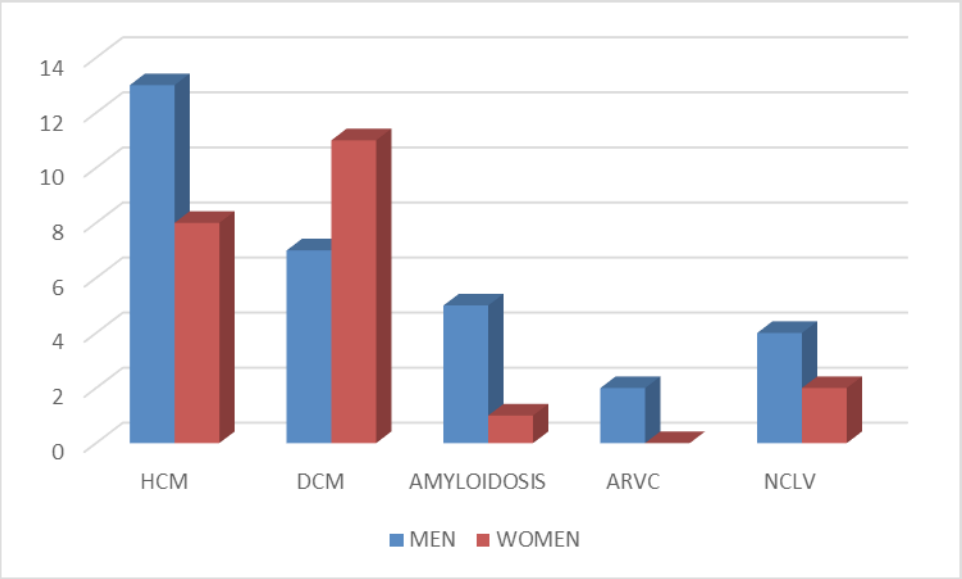

Figure 2: Distribution according to sex and phenotypic aspect of PCM.

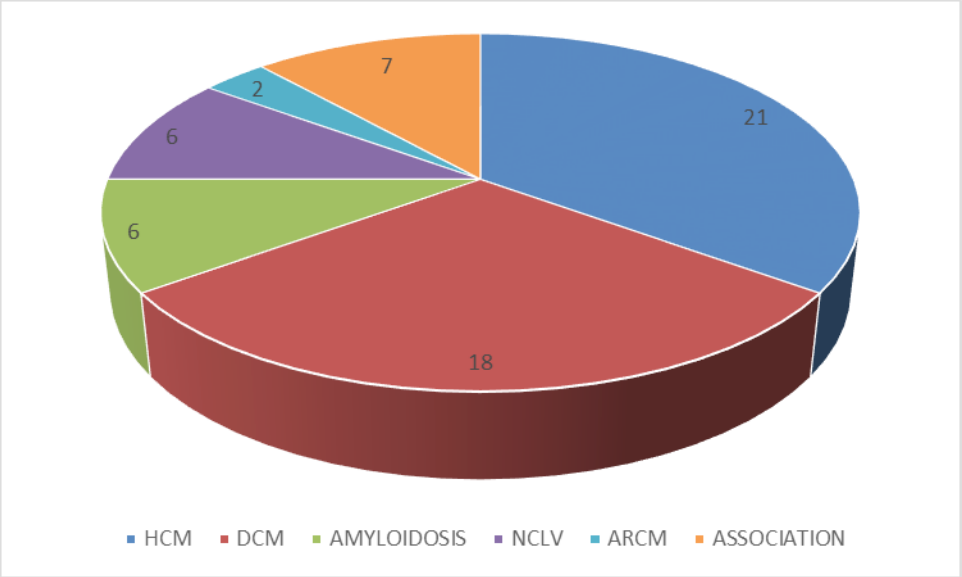

Figure 3: Distribution of patients according to cardiomyopathy phenotype.

Regarding the semiological aspects of MRI of HCM, the maximum end-diastolic thickness of the LV was greater than 14.3 mm with an average of 20.2 mm and a maximum of 27 mm. The left ventricular mass was reported in 19 patients with an average of 143g +/- 52.4g. The topography of this parietal thickening was mainly asymmetrical representing 90.5% with a predominance in antero-septo-basal followed by septal and mid-ventricular septal localizations (Figure 4). Alteration of the LVEF was observed in 4 patients or 21%. Stenosis of the LV outflow tract was present in 2 patients or 9.52%. Left intraventricular obstruction was observed in one patient (4.76%) and was associated with dilation of the left atrium. Anterior systolic motion of the mitral valve was present in 2 cases (9.52%). Dense intramyocardial fibrosis was observed in 8 cases (38.1%) in the form of late gadolinium retention. Figure 5 illustrates the MRI signs of this phenotype. And regarding DCM, a mean LV end-diastolic volume of 222 ml was noted with an alteration of the LV ejection fraction, the mean of which was 30.25%. Pericardial effusion was found in 5 patients or 28% and mitral reflux by dilation of the atrioventricular

ring in 3 patients or 17%. Late gadolinium retention was noted in one patient and with a linear intramyocardial septal topography. Figure 6 illustrates a DCM.

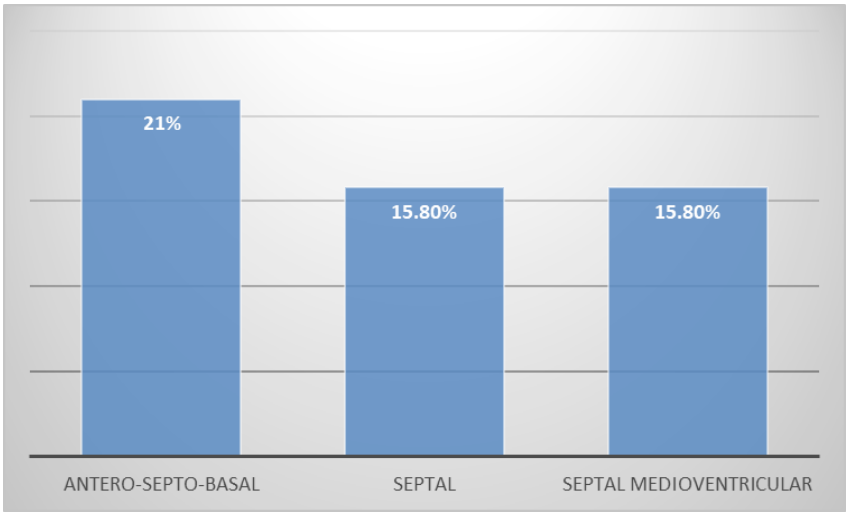

Figure 4: Distribution of segmental localization of HCMs.

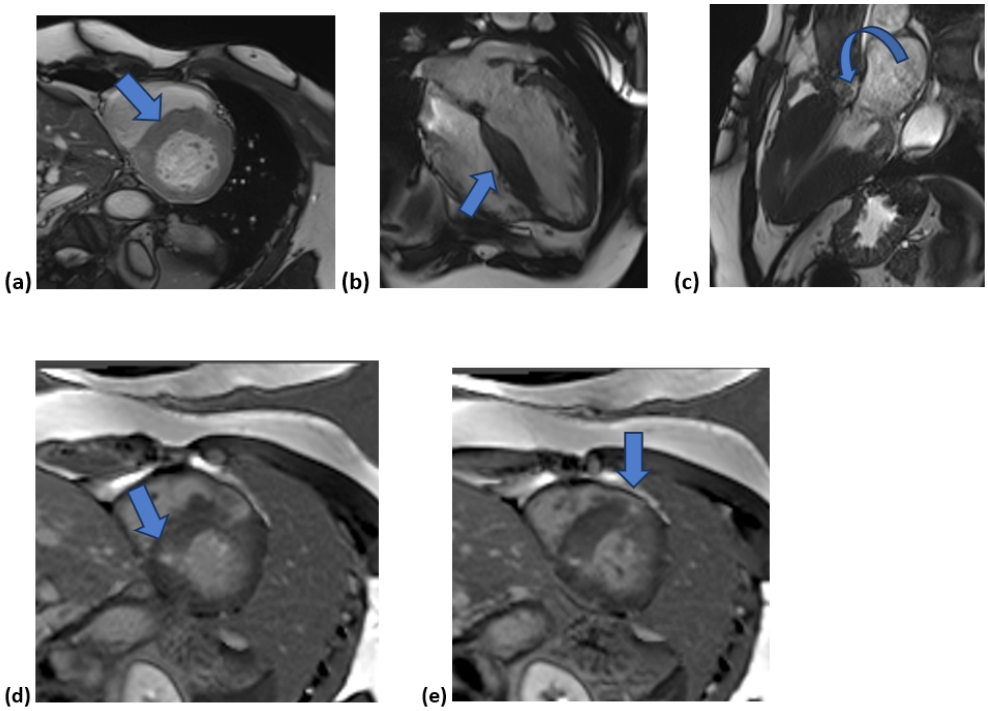

Figure 5: HCM: Short axis Trufi cine section, 04 cavities and 03 left cavities (a, b, and c). Short axis PSIR late enhancement sequences (d and e). HCM with asymmetric myocardial hypertrophy with septal predominance (solid arrow) with maximum thickness of 20 mm at the basal and medial crowns (a and b). Displacement of the anterior pillar of the mitral valve during LV systole: MAS, reducing the LV outflow tract (curved arrow in c). Dense fibrosis in the form of late and diffuse gadolinium retention at the RVLV attachment points (arrows in d and e).

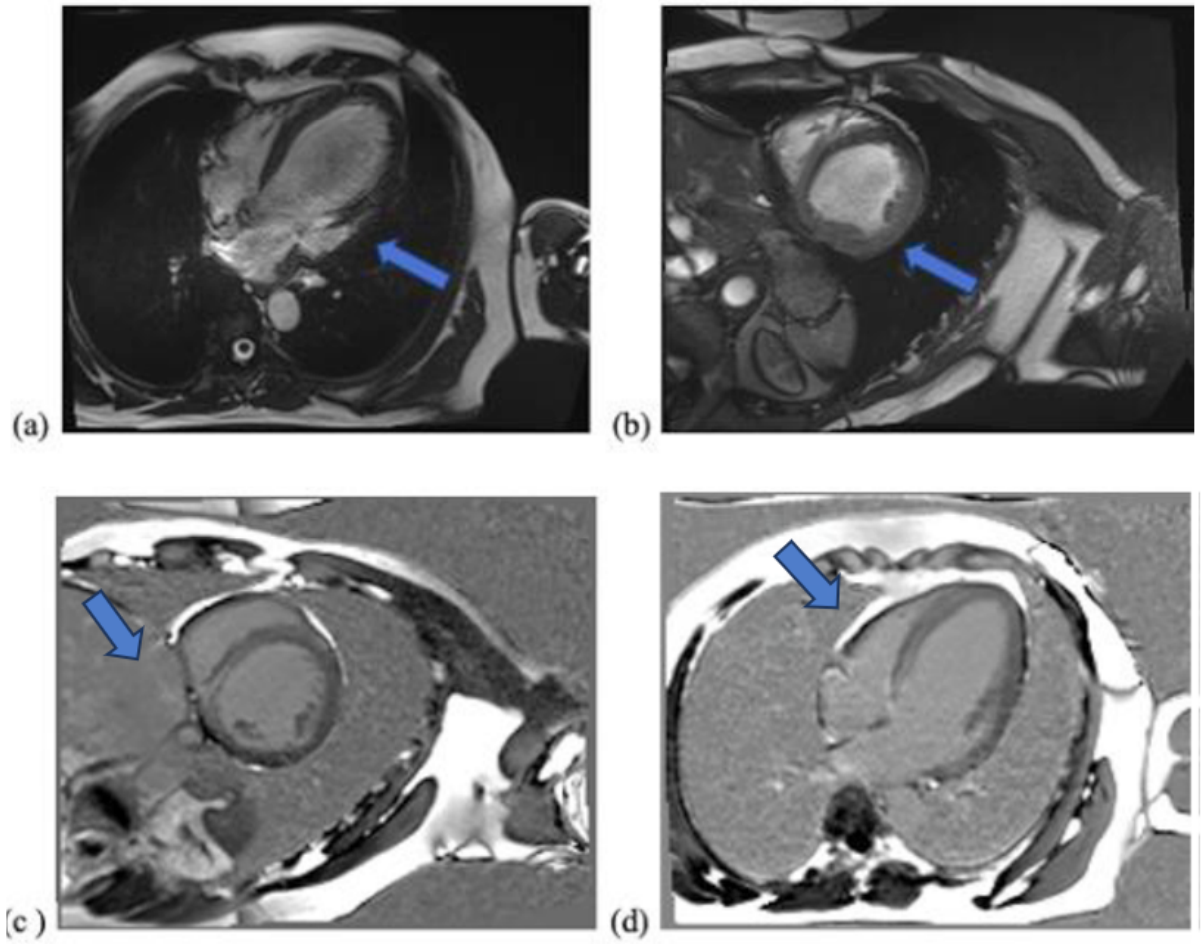

Figure 6: DCM: Ciné Trufi 04 cavities and short axis (a and b). Late enhancement PSIR short axis and 04 cavities (d and e)**.** LV dilation (solid arrow in a and b) with an indexed end-diastolic volume measured at 150 ml. Late linear intramyocardial retention at the interventricular septum (arrow in c and d).

As far as the amyloidosis concerned, the wall thickness was increased at the level of the LV and symmetrically in 5 patients (83.3%) and at the level of the RV in one patient (16.7%). The average wall thickness was 17.4 mm. The alteration of the LVEF was noted in 5 patients (83.3%) with an average of 31%. The late enhancement was noted in 5 patients (83.3%) with a transmural predominance and mainly involved all the walls of the cardiac cavities (Figure 7). The elevation of the T1 Mapping was present in 3 patients (50%) with an average of 1300 ms. In the other 3 patients there is no information on the T1 Mapping. The pericardial effusion was noted in 2 patients (33.3%).

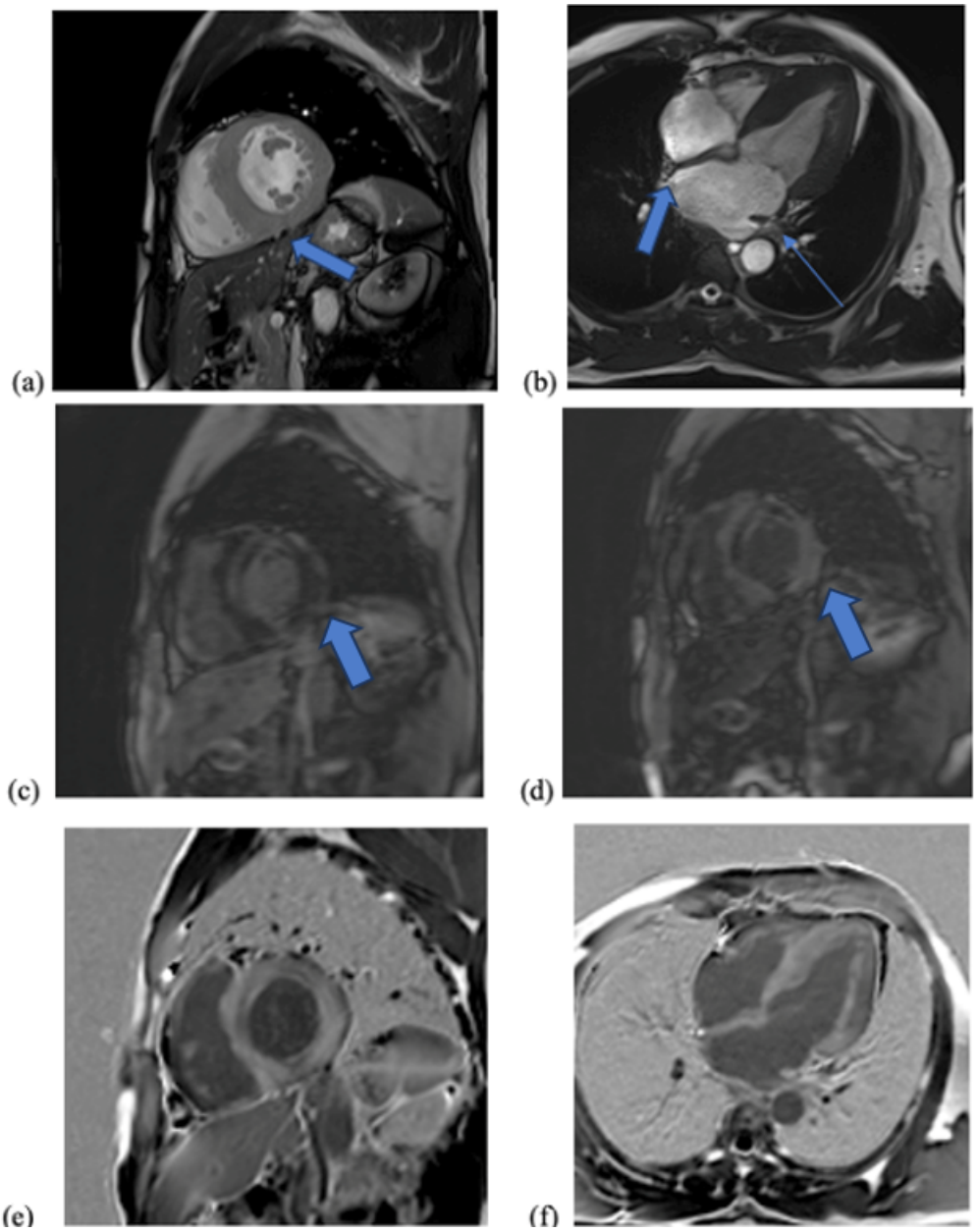

Figure 7: Amyloidosis. Cine Trufi short axis and 04 cavities (a and b), Ti scout (c and d) and late enhancement PSIR short axis and 04 cavities (e and f). Hypertrophy of the interventricular and interatrial septa (solid arrow) and dilation of the left atrium (thin arrow). Cancellation of the myocardial signal before that of the blood on the Ti scout sequence (d and e). Late and diffuse retention of Gadolinium affecting the 04 cavities and the interatrial septum in c and d.

For LVNC, elevated noncompacted to compacted myocardium ratio with a mean of 2.8 and impaired LVEF with a mean of 31.5% were present in all patients with this phenotype. Late enhancement was observed in 4 patients (66.7%). There were 2 cases of left ventricular dilatation and 2 cases of left intraventricular thrombus. Similarly, there was one case of interatrial septal aneurysm, one case of minimal pericardial effusion, and two cases of mitral and tricuspid valve leak. An illustration of this phenotype is shown in Figure 8.

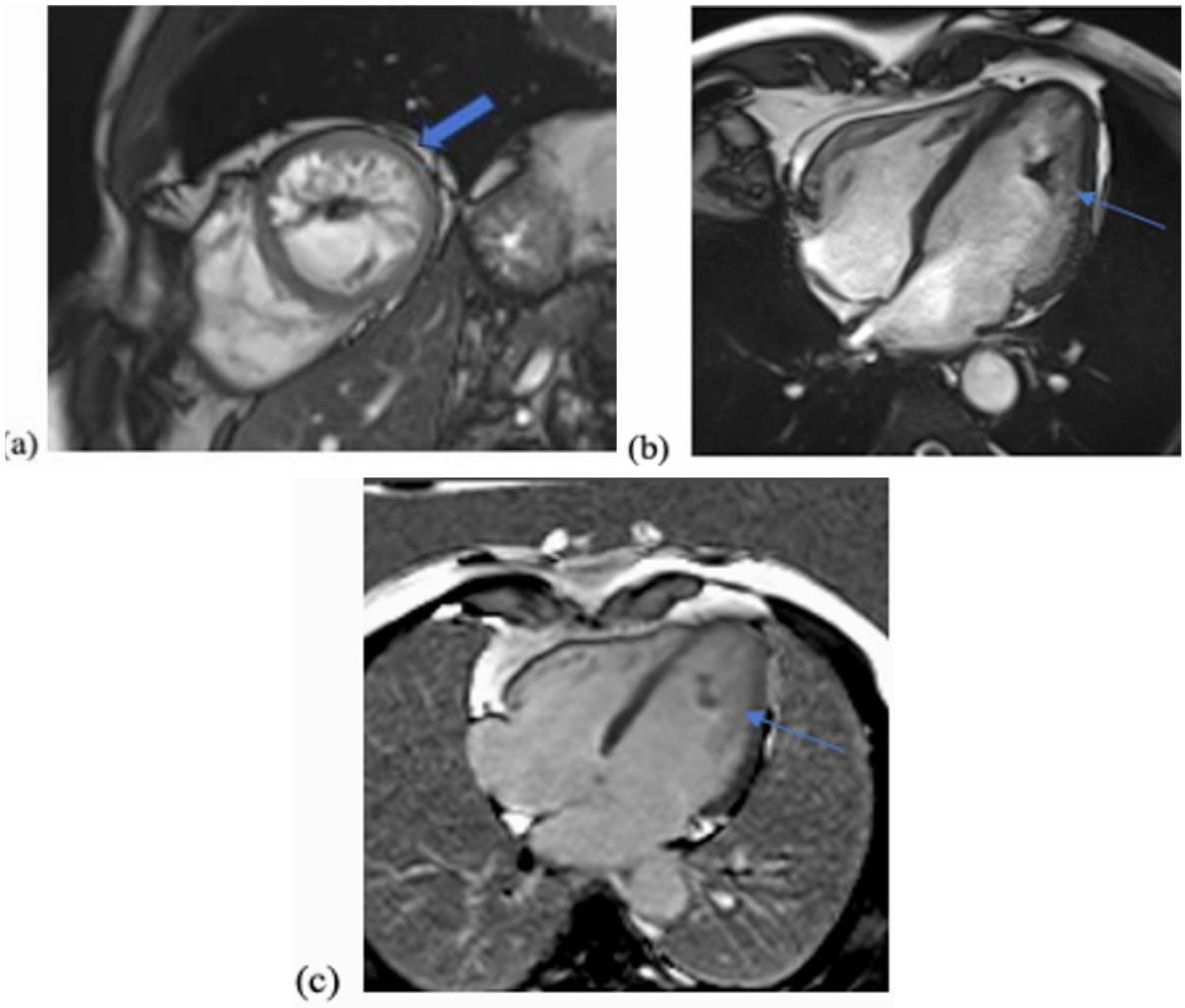

Figure 8: LVNC: Ciné Trufi short axis section and 04 cavities (a and b) and late PSIR enhancement 04 cavities (c)**.** Non-compaction of the LV: excess trabeculation of the lateral wall of the LV with non-compacted layer/compacted layer ratio estimated at 3.48 (solid arrow). Thrombus (thin arrow) in hypointense and not enhanced late.

In both cases of ARVC (Figure 9), right ventricular dyskinesia was noted with RVEFs of 14.5% and 18%. Right ventricular end-diastolic volumes were 68 ml and 108 ml. One of them had additionally moderate left ventricular dysfunction.

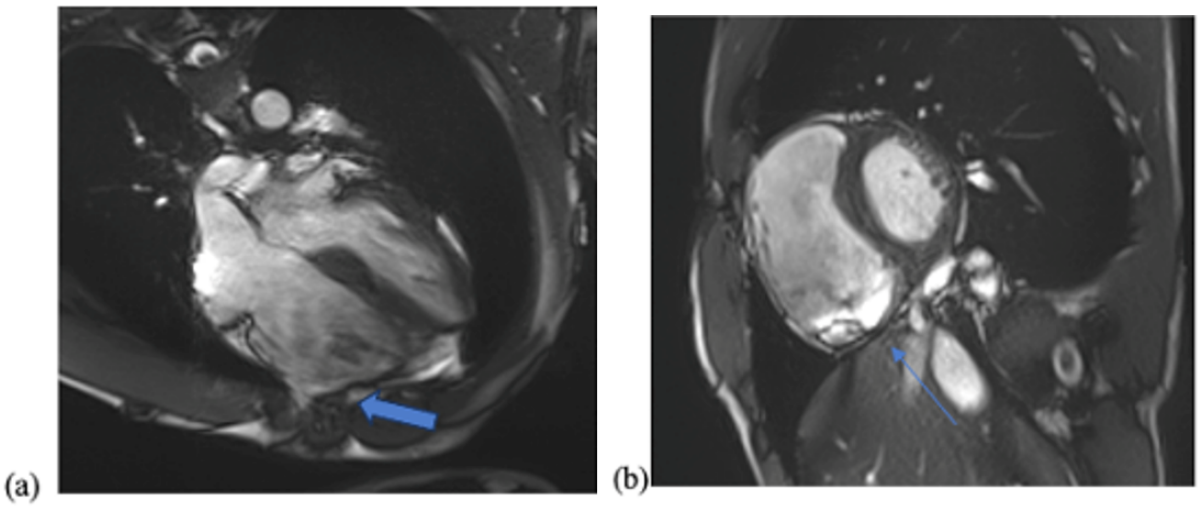

Figure 9: ARVC: Ciné Trufi sections 04 cavities (a) and 02 cavities (b). ARVC with major criterion in a 40-year-old man: Free wall dyskinesia (solid arrow) and RV dilatation (thin arrow) with an indexed end-diastolic volume measured at 115 ml.

Discussion

Hypertrophic Cardiomyopathy (HCM)

First identified in 1957 at St George's Hospital, London, in 08 patients with asymmetric left ventricular septal thickening, HCM has since been recognized worldwide [2]. It is an autosomal dominant, clinically heterogeneous heart muscle disease with a hereditary origin, mainly by mutations in genes encoding cardiac sarcomere myofilament proteins [5-6]. Sarcomere mutations are found in 60-70% of adult and pediatric patients with a family history of HCM and in 30-40% of apparently sporadic cases [6].

A large number of genetic mutations in HCM have been described, including those affecting proteins important for sarcomere function (a sarcomere is a basic unit of repeating contractile proteins that make up muscle cells) [2]. Mutations in the genes encoding beta-myosin heavy chain and myosin-binding protein C account for the majority of cases; less commonly affected genes are cardiac troponins I and T, tropomyosin alpha-1 chain, and myosin light chain [5]. Other etiologies have been described, including other genetic disorders: inherited metabolic and neuromuscular diseases, chromosomal abnormalities, and genetic syndromes and nongenetic disorders that mimic genetic forms [5].

Patients are susceptible to sudden cardiac death with an estimated mortality of 1% per year [5-4]. Thus, the assessment of the risk of sudden death has evolved to integrate two risk factors: a late enhancement (LE) of more than 15% of the left ventricular mass and an LVEF < 50% [1].

Its prevalence is 0.2% in the general population [2-6]. In the 2014 ESC recommendations, a number of methodologically diverse studies in North America, Europe, Asia and Africa report a prevalence of HCM of the order of 0.02 to 0.23% in adults [5]. Brieller J, et al., describe hypertrophic cardiomyopathy as the most common cardiomyopathy with a prevalence, in various geographical areas, of 0.2% of the general population [7]. Almeida et al find a higher prevalence of HCM between 1 in 200 to 500 people [8].

Similarly, Diop A. et al., in Senegal, found a predominance of HCM of 14.7% with PCM constituting 32.8% of their cohort [9]. This is similar to our results where hypertrophic cardiomyopathy is the most represented phenotype. Sarr et al., found a mean age of 53.2 years with extremities of 27 and 79 years and a male predominance of HCM in their study with a sex ratio of 1.66 [10]. These values are very close to those we obtained in our study.

The first-line imaging technique for initial assessment and follow-up remains transthoracic echocardiography. Yet, MRI is indicated in patients with poor acoustic windows or when certain regions of the LV are poorly visualized such as the anterolateral wall, the LV apex and the RV [4,5]. A consensus of European experts even recommends performing MRI in any patient with HCM [4].

Cardiovascular MRI is more sensitive than TTE in detecting LV apical and anterolateral hypertrophy, aneurysms, and thrombi [4,5].

Cardiovascular MRI encompasses several sequences that provide detailed information on cardiac morphology, ventricular function, and myocardial tissue characteristics [5].

The diagnosis of HCM is made when the wall thickness of the left ventricle is ≥ 15 mm on any segment of the myocardium that cannot be explained solely by loading conditions. In the presence of a family history of HCM or a positive genetic test for sarcomeric protein mutations, the diagnosis of hypertrophy is made from 13 mm [8-1-4]. The maximum wall thickness of patients diagnosed with HCM in the Diop A. et al study was 33 mm [9]. These diagnostic criteria are met in our study, where we had a maximum wall thickness of the LV greater than 15 mm in all patients of the phenotype except for one patient (14.5 mm). For the latter, a comparison with a family investigation was recommended. Almeida et al., found in their study that the distribution of HCM, in most of their patients, was asymmetrical with often an interposition of areas of normal parietal thickness.

The anterior free wall and the basal anterior ventricular septum were the most common locations of HCM [8]. This undergirds our results where we found more asymmetric distribution (90.5%) and with a predominance in antero-septo-basal (21%) followed by septal and septo-medio-ventricular locations (15.8%).

Other MRI signs found in our study, such as impaired LVEF, LV outflow tract stenosis, left intraventricular obstruction, left atrial dilation, anterior systolic motion of the mitral valve, and intramyocardial fibrosis marked by the presence of late enhancement, are described in the literature as signs of repercussion, without giving their prevalence.

Dilated Cardiomyopathy (DCM)

Dilated cardiomyopathy is conventionally defined by the presence of left ventricular or biventricular dilatation and systolic dysfunction in the absence of abnormal loading conditions or coronary artery disease sufficient to cause ventricular remodeling. However, this definition has been recognized as too restrictive, as left ventricular hypokinesia without dilatation may be the initial presentation of dilated cardiomyopathy [11-12].

The diagnosis, treatment and prognosis of dilated cardiomyopathies have been the subject of considerable progress. A comprehensive etiological assessment makes it possible to distinguish primary DCM, which are of genetic or non-genetic origin and characterized by solely cardiac involvement, and DCM secondary to numerous etiopathogenic mechanisms [11]. Approximately 25% to 35% of cases are familial, these cases being mainly inherited in an autosomal dominant manner [13]. It is the most common cause of heart failure [8]. Its treatment combines that of systolic heart failure with possible specific etiological management [10]. DCM is the main indication for heart transplantation [7].

According to Galinier et al., DCM was the most common cardiomyopathy with an estimated prevalence of 36 per 100,000 inhabitants [11]. This is not consistent with our results. This difference could stem from the fact that in the study by Galinier et al., screening was early, often at a subclinical stage, whereas in our study, most of our patients already had their diagnosis at the time of cardiac MRI.

In the European pilot cardiomyopathy registry, DCMs were the second most common PCM in terms of frequency (31%) and their incidence was estimated at between 5 and 8 cases per 100,000 inhabitants [14]. Similarly, Ikama et al., in Congo, found a hospital frequency of 32.1% [15] and Diop A. et al found a prevalence of DCM of 8.8%, lower than that of HCM in their study [9]. These results are similar to ours where we found a prevalence of CMD of 38%. DCM mainly affects young adults, with ages between 30 and 40 years for Galinier et al [11].

For Brieller J et al., the most represented age group was between 40 and 59 years [7]. Similarly, Ikama et al found a mean age of 52.9 ± 17.1 years [15]. The results of these studies are similar to those of our work where we had a mean age of 49.3 ± 18.6 years. Ikama et al. found a female predominance with 62% women for 38% men for patients with DCM [15]. These results are superimposable to our work where we noted a female prevalence with 11 women or 61% of patients diagnosed with DCM. On the other hand, Galinier et al. found a male prevalence three times higher than that of women [11]. Similarly, Omair Shah et al. found 24 men against 11 women [16]. These results are inversely proportional to ours.

Ventricular dilation and systolic dysfunction with normal myocardial thickness are the main characteristics of this phenotype. Typically, late gadolinium retention can be noted, with a linear shape and intramyocardial topography involving the interventricular septum as found in one of our patients [1-4]. The three patients diagnosed with DCM in the study by Diop A. et al presented with global hypokinesia and a minimal LVEF of 11% [9]. These same signs allowed the diagnosis in our study. Indeed, the mean end-diastolic volume and the mean LVEF in our study were respectively 222.29 ml and 30.25%.

Amyloidosis

Cardiac amyloidosis is the most common infiltrative cardiomyopathy. It is characterized by an extracellular deposition of insoluble fibrillar proteins that progressively invade the myocardium, creating a false “hypertrophy” appearance [4].

There are several types of amyloidosis. The most common form is light chain (AL) amyloidosis. The second type of amyloidosis is mutated transthyretin (ATTR) amyloidosis and the third type is wild-type transthyretin amyloidosis, known as senile, occurring with advanced age [16].

The diagnosis of cardiac amyloidosis is based on endomyocardial biopsy, which is, however, invasive and associated with complications. Non-invasive diagnostic methods include magnetic resonance imaging and scintigraphy. However, the use of the latter has been found only in the amyloid transthyretin variant, while its sensitivity and specificity in light chain amyloidosis are limited [16].

MRI is a powerful tool for the diagnosis and prognosis of cardiac amyloidosis. It provides information on the presence, distribution and localization of hypertrophy, visualization of cardiac amyloid infiltration with late enhancement and measurement of cardiac amyloid burden with T1 Mapping [8]. Cardiac MRI demonstrates diastolic dysfunction (low end-diastolic volume), restrictive physiology with late depressed systolic function, arrhythmias and heart failure [17]. Hypertrophy of the right ventricular and papillary muscles, bi-atrial dilation, thickening of the interatrial septum, thickening of the valve leaflets and pericardial effusion are also frequently found. T1 Mapping values are generally very high and revolve around 1300 to 1400 ms [18]. The myocardial inversion time lengthens and reverses in advanced forms with cancellation of the myocardial signal before that of the blood on the T1 scout sequence as found in our patient who served as an illustration. The 2021 ESC guidelines indicate that cardiac amyloidosis should be suspected in any patient with HF and preserved LV ejection fraction [14]. These T1 Mapping values are very close to ours where we had a mean T1 Mapping of 1300 ms.

In our study, there were 5 patients (83.3%) with increased LV wall thickness and one patient at the RV level. This coheres with the results of Omair Shah et al., who found increased LV and RV free wall thickness, accompanied by thickening of the interatrial septum in 31 patients or 88%. However, the LV ejection fraction was normal in most of their patients, unlike us who found impaired LVEF in only 5 patients (83.3%). In the cohort of Omair Shah et al., 92% had late enhancement and the pattern was either uneven multifocal, global subendocardial, or transmural [16]. These results are similar to ours where late enhancement was noted in 83.3% with a transmural predominance. Pleural pericardial effusion was noted in 21 patients (60%) and 27 patients (77%) respectively [16]. This is significantly higher than our results. This difference can be explained by the small population size for this phenotype in our study. The age of diagnosis is late and our results are similar to those of Omair Shah et al. who found a mean age of 58.3 years ± 5.6 years with extremes of 48 years and 65 years [16].

Left Ventricular Noncompaction (LVNC)

LVNC was considered until recently as a rare congenital cardiomyopathy, initially described in association with anomalies such as cyanotic heart disease. It was in 1984 that it was individualized as a distinct entity, in the absence of any other cardiac involvement [19]. Anatomically, it is characterized by the presence of a double layer of myocardium with prominent trabeculations and deep intertrabecular recesses [6].

Diagnosis is based on echocardiographic or cardiac magnetic resonance criteria [20]. Cardiac MRI allows a more precise and reliable assessment of the extension of the non-compacted zone and provides additional morphological information, particularly at the level of the apex and the lateral wall of the LV [20].

The diagnosis of LVNC in MRI is made on the basis of the ratio of the thicknesses of the non-compacted and compacted areas significant beyond a value of 2.3 from measurements made in diastole in 02-cavity incidence. [21-22-23] or on the use of a very sensitive and specific criterion: a trabeculated left ventricular mass representing more than 20% of the total mass of the LV [24-21]. The presence of cardiac fibrosis identified by late gadolinium enhancement could be a prognostic argument [21-19]. Thrombi can form between the trabeculations as found in our illustrative patient.

The ratio of non-compacted to compacted myocardium allowed the diagnosis to be made in all our patients. Similarly, late enhancement was observed in 4 patients (66.7%). Paule P et al. had found in the 3 patients in his study the existence of LV dilation with severe alteration of LVEF [19]. In our case, all patients had an alteration of LVEF but dilation of the left cavities was observed in only 2 patients. However, the recent ESC recommendations of 2023 no longer consider LVNC as a cardiomyopathy strictly speaking [1]. They define it rather as a phenotypic trait integrated into the different cardiomyopathies [1].

Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

ARVC is an inherited disease of desmosomal proteins and has a prevalence of 1 in 1000 to 5000 according to Jay Brieller et al [13]. It had a prevalence of 5% in the European Cardiomyopathy Pilot Registry [25].

ARVC is structurally characterized by progressive myocardial atrophy with fibro-fatty replacement of the right ventricular myocardium that may predispose to ventricular arrhythmias, unexplained syncope and/or sudden death. Lesions may also be present in the left ventricular myocardium [8-1]. Its diagnosis is based on a bundle of arguments. A diagnostic score has been established, with major (2 points) and minor (1 point) criteria, taking into account clinical, paraclinical, personal and family parameters [26].

Diagnostic criteria for ARVC on cardiac MRI are defined by the presence of segmental akinesia, dyskinesia or asynchrony of RV contraction associated with RV dilation defining a major criterion and a minor criterion. A major criterion defined by the association

of an RV kinetic disorder with an indexed right ventricular end-diastolic volume ≥ 110 ml/m^2 in men or ≥100 ml/m^2 in women or an RVEF ≤ 40% [26]. A minor criterion is defined by the association of a kinetic disorder with an indexed right ventricular end-diastolic volume ≥100 and <110 ml/m^2 in men or ≥90 and <100 ml/m^2 in women or an RVEF >40% and ≤45% [26]. These criteria allowed the diagnosis in our study. ARVC was noted in 2 male patients with RVEFs of 14.5% and 18% associated with RV kinetics disorders and RV end-diastolic volumes of 68 ml and 108 ml respectively: thus, classifying ARVC with major criterion.

Conclusion

MRI is the examination of choice for the evaluation of cardiomyopathies. It includes several sequences that provide detailed information on cardiac morphology, ventricular function and myocardial tissue characteristics. The profile of primary cardiomyopathies in our Senegalese population in MRI is not different from what is observed in the general population. The diagnostic criteria erected as a frame of reference by learned societies were generally respected in our study.

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