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Prevalence and Risk Factors of Dilated Cardiomyopathy among Yemeni Patients with Heart Failure

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Abstract

Background: Idiopathic dilated cardiomyopathy (DCM) is a heart muscle disease of an undefined cause that commonly presents as congestive cardiac failure. The etiology of weakness of the heart muscle is often unknown, but many causal factors had been identified. **Aim:** This study aims to estimate the prevalence of DCM in heart failure patients in Dhamar Governorate, Yemen, and identify the risk factors associated with DCM among heart failure patients in Dhamar Governorate. **Method:** A prospective hospital-based observational study was undertaken during the period of 20 December 2021 to 20 January 2022 in two hospitals namely AL-Wahda Teaching Hospital and Taiba Consultative Hospital. After obtaining informed consent from the patients and their treating physicians, all patients diagnosed with heart failure were included in the study. A standardized questionnaire developed by the researcher was used to collect data on sociodemographic characteristics of patients; results of clinical, laboratory and radiography exams were recorded. **Results:** The prevalence of DCM among HF patients is 44%, which makes it the most common cause of heart failure. Ischemic heart disease follows as the second most common cause at 27%. Qat chewing was a major risk factor in 72%, HTN in 60%, smoking in 49%, DM in 32%, family history in 29%, and pregnancy in 2%. Male was affected more than female in 61%, and dyspnea was the most common presenting symptom in 96% of cases mostly (NYHA CLASS IV). **Conclusion:** This study about prevalence of DCM among HF patients registry for 100 cases (male, female), age group more than 16 years, duration from 20 December 2021 to 20 January 2022 in Al-Wahda Teaching Hospital and Taiba Consultative Hospital, Dhamar Governorate, Yemen. Our study findings show that DCM is the most prevalent cause of heart failure

among all other causes, with a prevalence of 44%. Additionally, in our study, several risk factors were identified among the patients with heart failure, including chewing Qat, smoking, irregular treatment for diabetes, family history, and pregnancy.

Subject Areas

Cardiology

Keywords

Dilated, Cardiomyopathy, Heart Failure, Yemen

1. Introduction

1.1. General Introduction

Dilated cardiomyopathy is a disease of the heart muscle that makes the muscle walls stretched and thin (dilated). The thinner walls are weakened, which means the heart can't squeeze (contract) properly to pump blood to the rest of the body. The World Health Organization (WHO) defines dilated cardiomyopathy as a ventricular chamber exhibiting increased diastolic and systolic volume (left ventricular end-diastolic size > 115% of that calculated for age and body surface area) and a low (<40%) ejection fraction.

Dilated cardiomyopathy (DCM) occurs mostly in adults. It affects the heart's ventricles and atria, the lower and upper chambers of the heart, respectively. Frequently, the disease starts in the left ventricle and the heart's main pumping chamber. The heart muscle begins to dilate, meaning it stretch and become thinner. Consequently, the inside of the chamber enlarges. The problem often spreads to the right ventricle and then to the atria. As the heart chambers dilate, the heart muscle doesn't contract normally and cannot pump blood very well. As the heart becomes weaker, heart failure can occur. Common symptoms of heart failure include shortness of breath, fatigue and swelling of the ankles, feet, legs, abdomen and veins in the neck. Dilated cardiomyopathy also can lead to heart valve problems, arrhythmias (irregular heartbeats) and blood clots in the heart. The exact cause of DCM is often unknown, although up to one-third of those affected inherit it from their parents. Some diseases, conditions and substances also can cause the disease such as coronary heart disease, heart attack, high blood pressure, diabetes, thyroid disease, viral hepatitis and HIV infections, especially viral infections that inflame the heart muscle. Peripartum cardiomyopathy as a complication during the last month of pregnancy or early months of birth can cause DCM. Certain toxins such as cobalt certain drugs (such as cocaine and amphetamines) and two medicines used to treat cancer (doxorubicin and daunorubicin) can cause DCM.

1.2. Study Justification

DCM is the most common cause of HF and a major health problem, even in young age groups, leading to disability. Many studies have been conducted worldwide, but to our knowledge, no such study has been done in Yemen. Therefore, we undertook this study to identify the most common risk factors associated with these diseases.

1.3. Study Objectives

This study aimed to:

- To determine the prevalence of DCM in heart failure patients in Dhamar Governorate, Yemen
- To identify the risk factors associated with DCM among heart failure patients in Dhamar Governorate.

2. Review of Literature

2.1. Background

Dilated cardiomyopathy is defined by the presence of left ventricular dilatation and contractile dysfunction. Genetic mutations involving genes that encode cytoskeletal, sarcomere, and nuclear envelope proteins, among others, account for up to 35% of cases. Acquired causes include myocarditis and exposure to alcohol, drugs and toxins, and metabolic and endocrine disturbances. The most common presenting symptoms relate to congestive heart failure, but can also include circulatory collapse, arrhythmias, and thromboembolic events. Secondary neurohormonal changes contribute to reverse remodeling and ongoing myocyte damage. The prognosis is worst for individuals with the lowest ejection fractions or severe diastolic dysfunction. Treatment of chronic heart failure comprises medications that improve survival and reduce hospital admission—namely, angiotensin converting enzyme inhibitors and β blockers. 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 stem-cell therapy are being investigated [1].

Cardiomyopathies are a heterogeneous group of myocardial diseases associated with mechanical or electrical dysfunction that usually exhibit inappropriate ventricular hypertrophy or dilatation [2]. Primary cardio myopathies are predominantly confined to heart muscle, whereas secondary cardiomyopathies are generally caused by systemic conditions with associated cardiac dysfunction. In the American Heart Association classification, cardiomyopathies are classified according to cause whereas the European Society of Cardiology classification is based on a combination of morphology and hemodynamics. Dilated cardiomyopathy is defined by the presence of left ventricular dilatation and contrac-

tile dysfunction, in the absence of abnormal loading conditions and severe coronary artery disease [3] [4].

2.2. Epidemiology

Dilated cardiomyopathy is one of the most common causes of heart failure and the most common indication for heart transplantation worldwide, with an estimated prevalence of 40 cases per 100,000 individuals and an annual incidence of 7 cases per 100,000 individuals [5]. Racial differences exist [3] [5], whereas sex-related differences are less consistent [3] [6]. This disorder accounts for around 60% of childhood cardiomyopathies [7] [8], with infants younger than 12 months having the highest incidence.

2.3. Etiology

Genetic: causes are important at all ages. Clinical and echocardiographic screening in families of affected individuals shows evidence of familial transmission in 20% - 35% of cases [6] [9] [10]. Acquired causes of cardiomyopathy include infectious agents, drugs and toxins, and endocrine disturbances [1]. In children, common causes of dilated cardiomyopathy are genetic mutations, myocarditis, and inborn errors of metabolism [11] [12].

Peripartum cardiomyopathy is defined by otherwise unexplained dilated cardiomyopathy, with onset in the last month of pregnancy or within 5 months of delivery [13]. Disease progression occurs in up to a half of cases and recovery occurs in less than a quarter. The fundamental cause is unknown and the highest incidence has been reported in sub-Saharan Africa [14]. In some cases, familial peripartum cardiomyopathy or familial dilated cardiomyopathy is present [15]. If heart function returns to normal, the risk of recurrence is low [16].

Drugs and toxins: Alcohol abuse accounts for 21% - 36% of dilated cardiomyopathy cases in high-income countries [17]. The relationship between alcohol intake and clinical heart failure is influenced by various genetic, racial, and behavioral susceptibility factors [19]. The diagnosis is based on a history of heavy alcohol intake (>80 - 100 g/day for >10 years) [18] [19], in combination with otherwise unexplained cardiomyopathy [20]. Cocaine and methamphetamines are potent sympathomimetic drugs that induce heightened inotropic and chronotropic effects. Mechanisms of cardiac toxicity include myocardial ischemia from increased oxygen consumption, prothrombotic effects, coronary vasospasm, and accelerated coronary atherosclerosis [21] [22]. The prevalence of methamphetamine abuse is high among young adult patients with newly diagnosed dilated cardiomyopathy [23]. Anthracycline-induced cardiotoxicity can occur during treatment or many years afterward. The mechanisms of anthracycline-induced cardiotoxicity include oxidative stress, changes in mitochondrial membrane permeability, and suppression of respiratory chain activity [24] [25] [26]. Echocardiographic abnormalities are detected in between 25% and 50% of childhood cancer survivors within 20 years of treatment. The cumulative risk of

congestive heart failure 30 years after diagnosis is 8% in childhood cancer survivors who received a cumulative anthracycline dose of more than 250 mg/m^2 [27] [28]. Treatment with dexrazoxane, a free-radical scavenger, prior to anthracycline administration has been shown to diminish markers of acute myocardial damage [29]. The traditional histological criteria for myocarditis are based on an inflammatory cellular infiltrate, with or without myocyte necrosis [30] [31] [32] [33]. However, the clinical utility of these criteria is limited by both interobserver variability and low sensitivity; alternative criteria that rely on staining for various anti-CD surface antigens and antihuman leucocyte antigen might have greater sensitivity and improved prognostic value [30] [34]. Cardiac MRI can detect specific features of myocarditis without the sampling error associated with endomyocardial biopsy [35] [36]. The sequence of events leading to myocarditis typically starts with cardiac damage from viral infection (acute phase) resulting in exposure of intracellular antigens, leading to a T-lymphocyte-mediated inflammatory response (subacute phase). In some patients the inflammatory response can persist because of a misdirected immune response against endogenous heart antigens [30]. Myocarditis is an important cause of sudden death in adults younger than 35 years of age [37] and around 20% of patients with myocarditis develop a chronic dilated cardiomyopathy [38]. The typical manifestations of myocarditis are similar to those of dilated cardiomyopathy, but can vary from subclinical disease to arrhythmias, heart block, and sudden death, and can mimic myocardial infarction. In adults, fulminant lymphocytic myocarditis generally has a good prognosis [39]. Lymphocytic myocarditis accounts for around 10% of newly diagnosed adult-onset dilated cardiomyopathy, and is more common in children than adults. Myocarditis is most commonly caused by viral infection, although other infections can produce a similar clinical scenario [40] [41]. Coxsackievirus B, adenovirus, parvovirus B19, and human herpes virus 6 are common causes of myocarditis [41] [42]; many other viruses have also been implicated.

Other forms of myocarditis are caused by drug-induced hypersensitivity and systemic hyper eosinophilic syndromes, whereas giant-cell my carditis is primarily autoimmune in nature.

2.4. Clinical Features

Initial symptoms of heart failure are present in 80% of patients with dilated cardiomyopathy [43]. These symptoms include excessive sweating, ankle oedema, orthopnea, and fatigue after mild exertion. Abdominal discomfort, nausea, anorexia, and cachexia can be prominent in advanced cases. Circulatory collapse is the most severe manifestation of congestive heart failure. Some individuals have palpitations and syncope. Thromboembolic events and rarely, sudden death, might be the initial symptom, particularly in infants. Physical symptoms can include peripheral and sacral oedema, tachycardia, an elevated jugular venous pressure, pulmonary crepitations, an inferolateral displaced left ventricular apex

beat, a gallop rhythm, and a mitral regurgitant murmur. Pathophysiology Left ventricular dilatation is accompanied by remodeling, in which the left ventricle assumes a spherical shape. Pathophysiological changes include a decrease in stroke volume and cardiac output, impaired ventricular filling, and an increase in end-diastolic pressure. Compensatory changes in the vascular system include an increase in systemic vascular resistance, a decrease in arterial compliance, and an increase in venous pressure and circulating blood volume. Both cardiac preload and afterload are increased, with increased afterload resulting in elevated wall stress [44] [45]. Diastole involves both active relaxation (early diastole) and passive compliance (mid-to-late diastole). In dilated cardiomyopathy, diastolic dysfunction that affects both components can accompany the reduction in systolic function. Impaired ventricular relaxation results in reduced rapid ventricular filling. Reduction in ventricular compliance due to hypertrophy or fibrosis causes restrictive pathophysiology, with reduced ventricular filling and elevated end-diastolic pressures. Secondary neurohormonal changes include an increase in sympathetic adrenergic activity and a reduction in vagal activity to the heart. Neurohormonal changes include an increase in circulating catecholamines, an increase in vasopressin levels, and activation of the renin-angiotensin-aldosterone system. Nature tic peptide production is also increased. The combination of increased catecholamines, increased cardiac afterload, fluid retention, and tachycardia result in further elevation of wall stress and myocardial oxygen demand, as well as direct cardiotoxicity [46]. These secondary adaptations lead to ongoing myocyte damage with further reduction in myocardial performance. Compensatory hypertrophy allows more muscle fibers to share in wall tension for any given ventricular pressure and radius.

2.5. Diagnostic Investigations

The electrocardiogram (ECG) can only show non-specific repolarization abnormalities. Left ventricular hypertrophy, pathological Q waves, or poor R wave progression in the lateral chest leads might be observed. Prolongation of the PR interval might be the first manifestation of cardiomyopathy due to Lamin, emery, and SCN5A. Other abnormalities of conduction can mutations [47] [48] include atrioventricular block, left bundle branch block, and left anterior hemiblock. Chest radiographs usually show cardiomegaly and pulmonary venous redistribution, whereas pulmonary oedema is less common. Echocardiography typically shows global left ventricular hypokinesis; however, regional wall motion abnormalities might also exist. Left ventricular and atrial dilatation can be mild if the onset of the cardiomyopathy has been sudden. Right ventricular involvement is variable. Intracardiac thrombi and functional mitral regurgitation due to annular dilatation might also be noted. Doppler parameters can assist in quantifying the severity of diastolic dysfunction. Cardiac MRI provides accurate assessment of ventricular volumes, wall thickness, and contractile function, as well as tissue characterization. Delayed gadolinium enhancement can indicate

myocardial necrosis or scarring. Myocardial inflammation is considered likely when necrosis or scarring (late gadolinium enhancement) is detected in conjunction with oedema (high signal T2 intensity) and hyperemia (early gadolinium enhancement) [49] [50]. The presence of a pericardial effusion supports the diagnosis of myocarditis [36]. Electrocardiography, tissue Doppler imaging [51], and cardiac MRI can facilitate early detection of myocardial dysfunction in children with genetically determined cardiomyopathies, such as Duchenne muscular dystrophy, before the development of a typical dilated cardiomyopathy phenotype. Histological findings include irregular myocyte hypertrophy, with or without areas of fibrosis and myocyte damage. A lymphocytic infiltrate indicates the presence of inflammation that could be post-viral or immune mediated. Because of possible sampling error and variability in mechanisms of viral damage, the absence of these histological changes does not exclude the diagnosis of myocarditis. PCR can identify viral genome within the myocardium, even when inflammatory changes have resolved [41]. Cardiac histological examination can also help to identify specific disorders, such as sarcoidosis and haemochromatosis, and abnormal mitochondria, lysosomes, or myocardial inclusions, can indicate the presence of specific metabolic and storage disorders. Endomyocardial biopsy can be useful when a specific diagnosis is suspected that would influence therapy [52] [53] and is most helpful in patients with dilated cardiomyopathy who have recent onset of heart failure, ventricular arrhythmias, or Mobitz type II second or third degree atrioventricular heart block. Biomarkers, most commonly B-type natriuretic peptide (BNP) and N-terminal-BNP, are elevated in proportion to the severity of heart failure, leading to guidelines for their clinical use. In children with dilated cardiomyopathy, a BNP concentration greater than 300 pg/mL has been strongly associated with death, transplantation, or hospital admission due to heart failure.

2.6. Treatment

Therapy of dilated cardiomyopathy is mainly directed at treatment of heart failure symptoms and prevention of disease progression and related complications, such as end organ dysfunction and stroke. Medical therapy remains the mainstay in patients with dilated cardiomyopathy and heart failure. Present oral regimen recommendations are outlined elsewhere [54] [55], and have led to significant improvements in survival and symptom relief. In short, inhibition of angiotensin converting enzymes and β blockade with or without diuretics continue to be standard options. Treatment of decompensated heart failure is focused on diuresis with loop diuretics for volume overload and afterload reduction. Modulation of afterload can be accomplished with use of vasodilator therapy such as nitroglycerin, nitroprusside, or ebiratide. In patients with heart failure and clinical evidence of hypotension associated with hypoperfusion and raised filling pressures, intravenous inotropic support or vasopressor therapy, or both, should be considered. We prefer to avoid inotropes that increase mechanical stress,

such as dopamine and dobutamine, in children, with milrinone continuing to be the most popular therapy. Once the need for other inotropic agents becomes clear because of deterioration in blood pressure and perfusion, we consider placement of an assist device. Some institutions use balloon pumps regularly in this instance. A comprehensive list of therapeutic strategies with supportive evidence is provided elsewhere [56]. Several clinical trials have assessed use of implantable cardioverter defibrillators in patients with low left ventricular ejection fractions, with results showing reduced mortality [57] [58]. For patients who have a left ventricular ejection fraction of less than 30% and symptomatic heart failure for which they are receiving optimum medical therapy, use of implantable cardioverter defibrillators is a class I indication, as outlined by the ACC/AHA guidelines. Treatment Therapy of dilated cardiomyopathy is mainly directed at treatment of heart failure symptoms and prevention of disease progression and related complications, such as end organ dysfunction and stroke.

3. Material and Methods

3.1. Study Design

Descriptive observational study design was conducted from Des 2021 to Jan 2022 at AL-Wahda Teaching Hospital and Taiba Consultative Hospital in Dahmer Governorate, Yemen

3.2. The Study's Inclusion Criteria

This study included:

- Patient diagnosed to have HF.
- Patient came with signs and symptoms of HF as (dyspnea, raise jugular venous pressure, congested hepatomegaly, bilateral basal lung crepitation, peripheral oedema).

3.3. The Study's Exclusion Criteria

This study excluded:

- Patients who were asymptomatic.
- Patients with severe renal or liver insufficiency.
- Patient under 16-year-old.

3.4. Study Area

This study was conducted at the Thamar University AL-Wahda Teaching Hospital (TUHTH) Mabar city and Taiba Consultative Hospital Dahmer Governorate, Yemen.

3.5. Study Sitting

This study was carried out at the outpatient cardiological clinic and internal medicine department of TUHTH and Taiba Consultative Hospital.

3.6. Study Population & Study Sample

Any patient diagnosed with heart failure in outpatient cardiological clinics or admitted to the Internal Medicine Department in AL-Wahda Teaching Hospital and Taiba Consultative Hospital during data collection period from Dec 2021 to Jan 2022 was included in this study, a total was 100 patients.

3.7. Technique for Data Collection

- On a sheet, include demographic information (Age, Residence, Occupation, etc.).
- The patient's clinical data as etiological factors and information's that pointed to risk factor as (smoking, obesity, hypertension, diabetes, and family history) all that were noted and record.
- The results of the patient's testing, including laboratory tests as (Renal function, lipid Profile, etc.) and other Investigation as (ECG, ECHO, Chest X-ray, etc.) were recorded.

3.8. Data Analysis

Data processing, statistical analysis and graph drawing were performed using Statistical Package for Social Sciences (SPSS) (V.20.0)

3.9. Ethical Consideration

- The protocol of this study was approved by Thamar University Medical Ethics Committee (TUMEC NO 22006).
- The consent was taken from patients before any examination, all of information was collected and kept strict confidently in order to follow the ethical and legal standards of the scientific investigate.

4. Results

4.1. Demographic Data

Attal of 100 patients with heart failure participated in this study according to our study most age group affected 42 (42)% were old age from (61 - 80 yrs.) and just 2 cases under 20-year, male mostly affected 61 (61%), married 91 (91%), 67 (67%) were from Dhamar city and half of the patients had duration of their illness of (1 - 5 yrs) 50 (50%). (Table 1)

4.2. Risk Factors

When taking of medical history of patients most risk factors in our study in patients were Qat chewers 72 (72%), hypertension present in 60 (60%) of cases, (40%) were smoker. D.M. as a risk factor was in (32%) of patients, 29 (29%) of cases have family history of heart disease, history of CAD was found in 30 (30%), but pregnancy was found in only 2 case (2%). (Table 2)

Table 1. Socio-demographic characteristics of patients with heart failure (n = 100).

Characteristics		Frequency	%
Age category	Less than 20	2	2%
	21 - 40 years	16	16%
	41 - 60 years	32	32%
	61 - 80 years	42	42%
	More than 81 years	8	8%
Gender	Male	61	61%
	Female	39	39%
Mitral status	Married	91	91%
	Not married	9	9%
Occupation	House wife	35	35%
	Farmer	18	18%
	Worker	14	14%
	Soldier	7	7%
	Other	8	8%
	No Job	18	18%
Residence	Dhamar	67	67%
	Yarim	10	10%
	Anis	6	6%
	Al-Hada'a	5	5%
	Other	12	12%
Duration of illness	Less than 1 year	21	21%
	1 - 5 years	50	50%
	6 - 10 years	6	6%
	not mentioned	23	23%

Table 2. Risk factor of heart failure patients (n = 100).

Characteristics	Frequency	%
Qat chewing	72	72.00%
HTN	60	60.00%
Smoking	49	49.00%
DM	32	32.00%
Hx of CAD	30	30.00%
Family history	29	29.00%
Shammah	16	16.00%
Pipe	14	14.00%
Pregnancy	2	2.00%

4.3. Clinical Features

Clinical features in Table 3 shows that most frequent symptoms was dyspnea in (96%), followed by lower limb oedema in 88(88%), then chest pain in 65 (65%) also palpitation was in 59 (59%), Rt. Hypochondrial pain in 54 (54%), syncopal attack 33 (33%) and less frequently abdominal distention in 30 (30%).

4.4. Physical Examination

Physical examination in our study in patient with heart failure most patient have lower limb oedema in 86 (86%), congested neck vein in 73 (73%), crepitation 67 (67%), hepatomegaly 33 (33%), and less frequently ascites 23 (23%). (Table 4)

4.5. Clinical Examination of Cardiovascular System

4.5.1. Cardiovascular System Examination

In examination of cardiovascular system 29 (29%) of cases have HTN, 15 (15%) have hypotension, the pulse was unpalpable in 20 (20%) of cases and the place of apical pulse was in 5th intercontinental space in 69 (86%) of cases. (Table 5)

4.5.2. Auscultation of Heart

During examination of cardiovascular system and auscultation of heart we found 40 (40%) of pts. Have abnormal heart sound also S1 was muffled in 70 (70%) of cases and S2 was muffled in 57 (57%) of cases. (Figure 1)

Table 3. Clinical features of heart failure patients (n = 100) patients.

Characteristics	Frequency	%
Dyspnea	96	96.00%
Oedema	88	88.00%
Chest pain	65	65.00%
Palpitation	59	59.00%
Rt. Hypochondrium pain	54	54.00%
Syncopal attack	33	33.00%
Abd. Distension	30	30.00%

Table 4. Physical examination of heart failure patients (n = 100).

Characteristics	Frequency	%
Lower limb oedema	86	86%
Congested neck vein	73	73.00%
Crepitation	67	67.00%
Hepatomegaly	33	33%
Ascites	23	23%

Table 5. Cardiovascular system examination of heart failure patients (n = 100).

Characteristics		Frequency	%
Blood pressure	Normal	56	56%
	Hypertension	29	29%
	Hypotension	15	15%
Apical heart sound	Palpable	80	80%
	Un palpable	20	20%
Place of apical heart rate	4th ICS	1	1%
	5th ICS MCL	69	86%
	6th ICS AAL	8	10%
	7th ICS AAL	2	3%

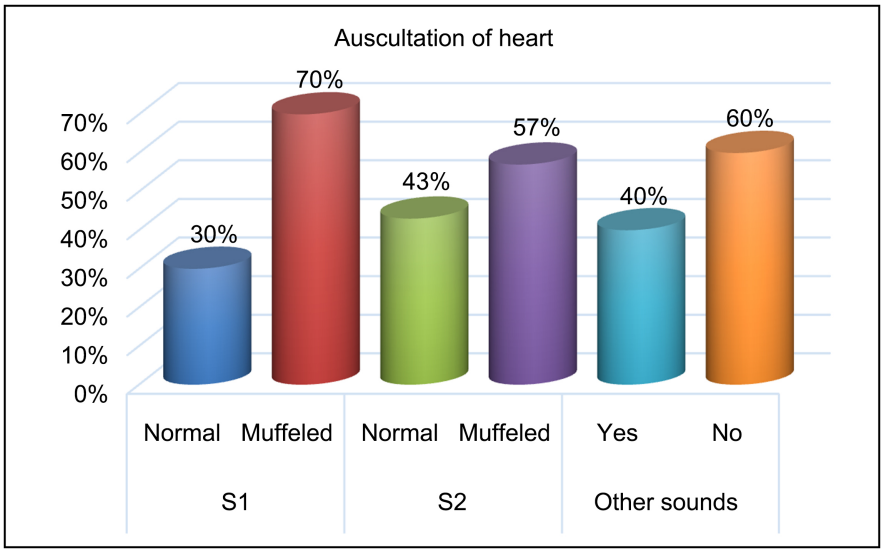

Figure 1. Auscultation of heart failure patients (n = 100).

4.5.3. Abnormal Heart Sound

Among 40 patients with abnormal sound 24 (60%) of cases have murmur and 11 (27.5%) of cases have S3, 3 (7.5%) of cases have S4 and gallop rhythm was found in 2 (5%) of cases. (Table 6)

4.5.4. Radial Pulse

Radial pulse in our study was normal in 78 (78%) of cases, the others 20 (20%) have tachycardia and bradycardia in 2 (2%) of cases. The pulse was irregular in 22 (22%) of cases, the volume of pulse was average in 58 (58%) of cases but small in 39 (39%) and large only in 3 (3%) of cases. Also, the pulse was equal in both sides in 100% of pts. (Table 7)

About radial pulse 8 (8%) of patients have special characters 5 (62.5%) have pulsus alternans and 3 (37.5%) have water hammer pulse. (Table 8)

Table 6. Abnormal heart sound of heart failure patients (n = 40).

Characteristics	Frequency	%
Murmur	24	60%
S3	11	27.50%
S4	3	7.50%
Gallop rhythm	2	5%

Table 7. Radial pulse of heart failure patients (n = 100).

Characteristics		Frequency	%
Heart rate	Normal	78	78%
	Tachycardia	20	20%
	Bradycardia	2	2%
Regularity	Regular	78	78%
	Irregular	22	22%
Volume	Small	39	39%
	Average	58	58%
	Large	3	3%
Equal	Yes	100	100%
	No	0	0%
Special characters	Yes	8	8%
	No	92	92%

Table 8. Special character of peripheral pulse of heart failure patients (n = 8).

Characteristics	Frequency	%
Pulsus alternans	5	62.5%
Water hammer pulse	3	37.5%

4.6. Laboratory Investigation

About laboratory investigation in our study, we did some investigations as CBC which was normal in most pts. 65 (65%) of cases, abnormal in 32 (32%) of cases and no data was present in only 3 (3%) of cases.

LFT no data was found in 79 (79%) of cases but in pts. Who did LFT 15 (15%) was normal and 6 (6%) was abnormal? RFT was normal in 65 (65%) of cases, but abnormal in 31 (31%) of cases and no data found in 4 (4%) of cases. Lipid profile no data found in 79 (79%) of cases among 21 case who did it 11 cases have abnormal and 10 cases have normal lipid profile. (Table 9)

4.7. Radiological Finding

In our study the radiological investigation was from the most important investigation we looked for to confirm the diagnosis of HF & DCM. In echocardiogram 100 (100%) of patients was abnormal, in ECG 4 cases only were normal, abnormal in 86 (86%) of cases but no data found in 10 (10%) of cases. In chest X-ray we found that the data was abnormal in 82 (82%) of cases, normal in 7 (7%) of cases and no data in 11 (11%) of cases. (Tables 10-12 and Figure 2)

4.8. Treatment

Most patient treated by diuretics mostly loop diuretics and spironolactone also many patients treated by beta blockers 80 (80%) of cases and ACE inhibitors 72 (72%) of cases but few patients use the new drugs which are SGLT inhibitors 72 (72%) and ARNI 2 (2%) of cases mostly because they are expensive. (Table 13 and Figure 3)

Table 9. Laboratory investigation of heart failure patients (n = 100).

Characteristics		Frequency	%
CBC	Normal	65	65%
	Abnormality	32	32%
	No data found	3	3%
LFT	Normal	20	20%
	Abnormality	6	6%
	No data found	79	79%
RFT	Normal	65	65%
	Abnormality	31	31%
	No data found	4	4%
Lipid profile	Normal	10	10%
	Abnormality	11	11%
	No data found	79	79%

Table 10. Radiological finding of heart failure patients (n = 100).

Characteristics		Frequency	%
Echocardiogram	Abnormality	100	100%
Electrocardiogram	Normal	4	4%
	Abnormality	86	86.00%
	No data found	10	10.00%
Chest X-ray	Normal	7	7.00%
	Abnormality	82	82.00%
	No data found	11	11.00%

Table 11. The common etiologies lead to heart failure regarding Echo finding (n = 100).

Characteristics	Frequency	%
Dilated cardiomyopathy	44	44%
Ischemic heart disease	27	27.00%
Hypertensive heart disease	15	15.00%
Rheumatic heart disease	11	11.00%
Congenital heart disease	3	3.00%

Table 12. ECG Abnormality of heart failure patients (n = 86).

Characteristics	Frequency	%
Low voltage	24	24.50%
LVH	14	14.30%
P. Mitral	11	11%
Sinus tachycardia	10	10%
LAD	9	9%
RAD	6	6%
Old MI	5	5%
AF	5	5%
LBBB	4	4%
Inverted T wave	4	4%
RVH	3	3%
Premature beat	2	2%
Elevated ST segment	1	1%

Table 13. Pharmacological treatment of heart failure patients (n = 100).

Characteristics	Frequency	%
Diuretics	92	92.00%
Beta Blocker	80	80.00%
ACE Inhibitors	72	72.00%
Digitalis	46	46.00%
ARBS	43	43.00%
SGLT inhibitors	13	13%
ARNI	2	2%

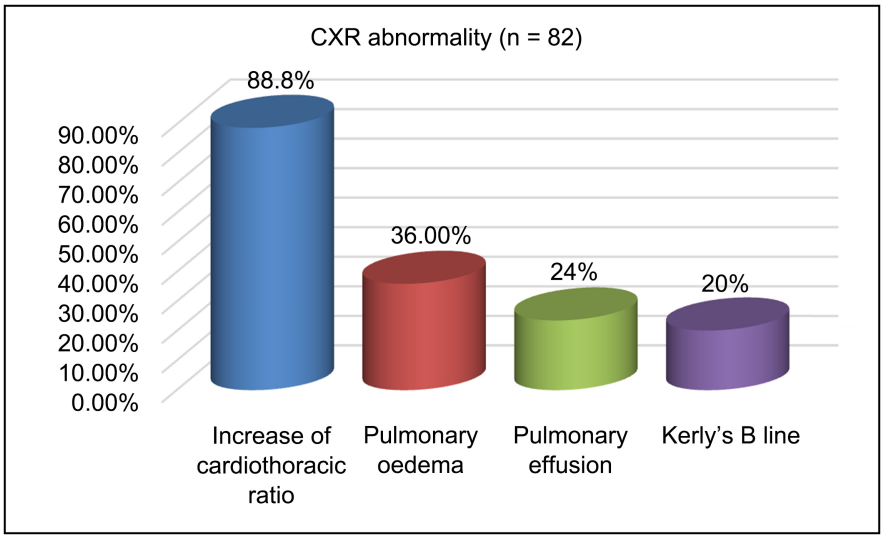

Figure 2. CXR abnormality of heart failure patients (n = 82).

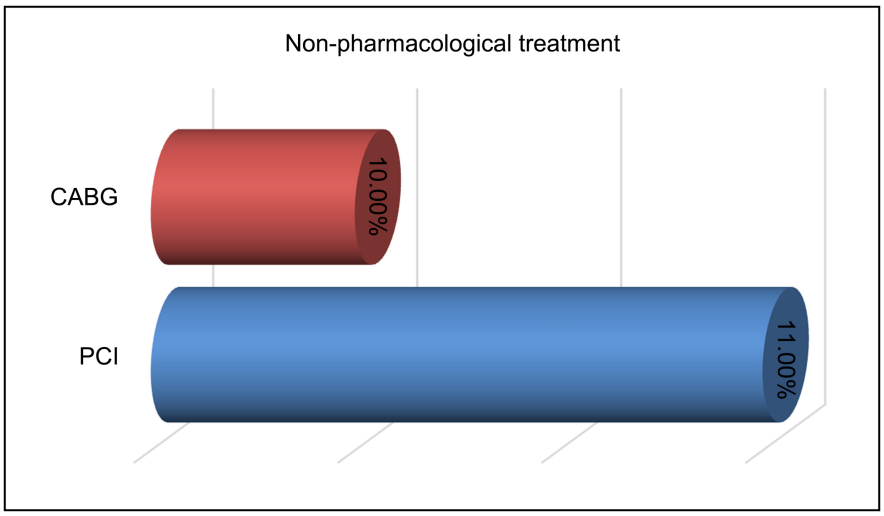

Figure 3. Non-pharmacological treatment of heart failure patients (n = 100).

5. Discussion

Using clinical and echocardiography criteria Our study showed that the Prevalence of dilated cardiomyopathy among heart failure patients in age group more than 16 years registry for 100 patients and conducted from 20 December 2021 to 20 January 2022 in Al-Wahdah Teaching Hospital and Taiba Consultative Hospital revealed that DCM is a major cause represent about 44% of causes of heart failure followed by ischemic heart diseases 27% and hypertensive heart diseases 15% these finding, similar result obtained from a prospective observational study design was conducted from January to April 2007 in two teaching hospitals (Academy Charity and Al-Shaab) of Khartoum, the research findings revealed that Dilated Cardiomyopathy (DCM) prevailed in Sudanese population with heart failure It had an overall prevalence of 43.0% (31 DCM out 72 heart failure) [59].

The prevalence of DCM among males with heart failure was significantly different when compared to that among females in our research, 61% for males and 39% for females, but in the previous study [60], which prevailed in Sudanese population with heart failure the prevalence of DCM among males with heart failure was not significantly different when compared to that among females with prevalence of 53% and 47% respectively, however in men being 3 folds more involved than women was stated in a study done in the United States. also, a similar study in the 1990 a Brazilian cohort study reported on 1220 outpatients followed in a specialized HF clinic, showing a mean age of 45 years. The idiopathic etiology was present in 37% as a commonest cause of HF [61].

In contrast, in the Argentinean GESICA study 40% of the patients had previous myocardial infarction; the idiopathic etiology was present in 20% and the chasmic in 10% [62]. In Minnesota, from a population sample of 45 patients with systolic HF accompanied at a HF clinic of a tertiary hospital, 47% of patients had ischemic etiology and 44% idiopathic etiology [63].

In a Brazilian report including 903 hospitalized patients from a tertiary center with diagnosis of HF, the medium age was 52 years, with the ischemic etiology being the most frequent (33%), followed by the idiopathic (26%), valvular (22%), hypertensive (7%) and chasmic etiology (6%); However, in a general hospital of Antigua , reporting on a population of 293 patients with HF as the diagnosis of discharge, hypertensive etiology was the most prevalent etiology (41%), followed by the ischemic etiology (33%). In sub-Saharan Africa, cardiomyopathies constitute 21.4% of heart failure cases, with dilated cardiomyopathy (DCM) being the most common form [63].

About clinical features of patients in our study most common symptoms are dyspnea 96% followed by lower limb oedema 88%, the chest pain represents about 65% and palpitation form about 59% of cases. In the study which prevailed in Sudanese population with HF the symptoms were in heart failure patients with DCM (n = 31), the symptoms the most often reported were palpitation (93.5%), orthopnea (80.6%), PND (74.1%) and chest pain (29.0%). The frequency of the symptoms revealed by patients of heart failure due to others causes (n = 41) were palpitations (95.1%), orthopnea (73.1%), PND (66.0%) and chest pain (32.0%).

In grading of dyspnea in our study most patients had exertional dyspnea 81.20, orthopnea 66.70%, PND 66.70% and about 43.80 of patients had dyspnea at rest. In the study conducted in Sudanese population with heart failure grading of dyspnea revealed that in heart failure with DCM (n = 31), dyspnea was absent in one patient (3.0%). In the remaining 97.0%, dyspnea was observed on severe exertion (grade 1 dyspnea) in 7.0% of the patients, on moderate (grade 2) and mild exertion (grade 3) respectively 32.0% and 42.0% while dyspnea occurring at rest (grade 4) was 16.0%.

About signs of heart failure in our study, lower limb oedema represents about 86% of cases and congested neck vein about 73% and basal crepitation about

67%, hepatomegaly and ascites represent about 33% & 23% respectively. in study conducted in Sudanese population with heart failure the signs of heart failure in DCM patients (n = 31) were hepatomegaly (87.1%), bilateral basal crepitation (90.3%) and lower limb edema (93.5%) while in patients of heart failure due to other causes hepatomegaly, bilateral basal crepitation and lower limb edema were respectively 80.5%, 97.6% and 65.9% [61].

6. Conclusion and Recommendation

6.1. Conclusion

This study could be concluded that dilated cardiomyopathy is the most common cause of HF (44%) similar to many studies worldwide. Dilated cardiomyopathy was more common in male than female and the major risk factor included was Qat chewing among Yemeni patients followed by hypertension and smoking. Also, pregnancy is a risk factor in 2 cases in our study.

6.2. Recommendation

From the findings of this study:

1) We recommend further studies in order to understand the exact cause and pathophysiology of DCM, determine the impact of them on clinical outcomes, identify effective treatments for their symptoms, and provide clinicians with the evidence for practical guidelines.

2) We need further studies to determine the effect of Qat chewing on cardiac diseases.

3) Educate the patients about the effect of bad habits such as Qat chewing and smoking on cardiac disease.

4) Educate the patient about the natural course of the disease and the importance of taking medication regularly.

Conflicts of Interest

The authors declare no conflicts of interest.

References

[1] Maron, B.J., Towbin, J.A., Theine, G., *et al.* (2006) Contemporary Definitions and Classification of the Cardiomyopathies: An American Heart Association Scientific Statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention. *Circulation*, **113**, 1807-1816. https://doi.org/10.1161/CIRCULATIONAHA.106.174287

[2] Elliott, P. andersson, B., Arbutin, E., *et al.* (2008) Classification of the Cardiomyopathies: A Position Statement from the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *European Heart Journal*, **29**, 270-276. https://doi.org/10.1093/eurheartj/ehm342

[3] Yancy, C.W., Jessup, M., Bozkurt, B., *et al.* (2013) 2013 ACCF/AHA Guideline for

the Management of Heart Failure: Executive Summary: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Journal of the American College of Cardiology*, **62**, 1495-1539. https://doi.org/10.1016/j.jacc.2013.05.020

[4] Pinto, Y.M., Elliott, P.M., Arbutin, E., *et al.* (2016) Proposal for a Revised Definition of Dilated Cardiomyopathy, Hypokinetic Non-Dilated Cardiomyopathy, and Its Implications for Clinical Practice: A Position Statement of the ESC Working Group on Myocardial and Pericardial Diseases. *European Heart Journal*, **37**, 1850-1858. https://doi.org/10.1093/eurheartj/ehv727

[5] Manolios, T.A., Baughman, K.L., Roedeer, R., *et al.* (1992) Prevalence and Etiology of Idiopathic Dilated Cardiomyopathy (Summary of a National Heart, Lung, and Blood Institute Workshop). *American Journal of Cardiology*, **69**, 1458-1466. https://doi.org/10.1016/0002-9149(92)90901-A

[6] Taylor, M.R., Carnie, E. and Mastroeni, L. (2006) Cardiomyopathy, Familial Dilated. *Orphanet Journal of Rare Diseases*, **1**, Article No. 27. https://doi.org/10.1186/1750-1172-1-27

[7] Lipshultz, S., Sleeper, L., Tobin, J., *et al.* (2003) The Incidence of Pediatric Cardiomyopathy in Two Regions of the United States. *The New England Journal of Medicine*, **348**, 1647-1655. https://doi.org/10.1056/NEJMoa021715

[8] Nugent, A.W., Wilkinson, L.C., Dabney, P.E.F., *et al.* (2003) The Epidemiology of Childhood Cardiomyopathy in Australia. *The New England Journal of Medicine*, **348**, 1639-1646. https://doi.org/10.1056/NEJMoa021737

[9] Michel's, V.V., Moll, P.P., Miller, F.A., *et al.* (1992) The Frequency of Familial Dilated Cardiomyopathy in a Series of Patients with Idiopathic Dilated Cardiomyopathy. *The New England Journal of Medicine*, **326**, 77-82. https://doi.org/10.1056/NEJM199201093260201

[10] Grundig, E., Tasman, J.A., Kucherov, H., Franz, W., Kubler, W. and Katsu, H.A. (1998) Frequency and Phenotypes of Familial Dilated Cardiomyopathy. *Journal of the American College of Cardiology*, **31**, 186-194. https://doi.org/10.1016/S0735-1097(97)00434-8

[11] Tobin, J.A., Lowe, A.M., Colin, S.D., *et al.* (2006) Incidence, Causes, and Outcomes of Dilated Cardiomyopathy in Children. *JAMA*, **296**, 1867-1876. https://doi.org/10.1001/jama.296.15.1867

[12] Hsu, D.T. and Canter, C.E. (2010) Dilated Cardiomyopathy and Heart Failure in Children. *Heart Failure Clinics*, **6**, 415-432. https://doi.org/10.1016/j.hfc.2010.05.003

[13] Pearson, G.D., Vielle, J.C., Kahtoola, S., *et al.* (2000) Peripartum Cardiomyopathy: National Heart, Lung, and Blood Institute and Office of Rare Diseases (National Institutes of Health) Workshop Recommendations and Review. *JAMA*, **283**, 1183-1188. https://doi.org/10.1001/jama.283.9.1183

[14] Hilfiger-Kleiner, D., Silwan, K. and Drexler, H. (2008) Peripartum Cardiomyopathy: Recent Insights in Its Pathophysiology. *Trends in Cardiovascular Medicine*, **18**, 173-179. https://doi.org/10.1016/j.tcm.2008.05.002

[15] Ware, J.S., Li, J., Malaika, E., *et al.* (2016) Shared Genetic Predisposition in Peripartum and Dilated Cardiomyopathies. *The New England Journal of Medicine*, **374**, 233-241. https://doi.org/10.1056/NEJMoa1505517

[16] Celta, F. and Michel's, V.V. (1995) The Natural History and Spectrum of Idiopathic dilated Cardiomyopathy, Including HIV and Peripartum Cardiomyopathy. *Current*

Opinion in Cardiology, **10**, 332-338.
https://doi.org/10.1097/00001573-199505000-00015

[17] Maisch, B., Nutrias, M., Ruppert, V., Richter, A. and Panduit, S. (2012) Cardiomyopathies: Classification, Diagnosis, and Treatment. *Heart Failure Clinics*, **8**, 53-78. https://doi.org/10.1016/j.hfc.2011.08.014

[18] Lao Nigro, I., Correlate, M., Di Biased, M. and Alto mare, E. (2009) Alcohol Abuse and Heart Failure. *European Journal of Heart Failure*, **11**, 453-462.
https://doi.org/10.1093/eurjhf/hfp037

[19] Fernandez-Sola, J. (2015) Cardiovascular Risks and Benefits of Moderate and Heavy Alcohol Consumption. *Nature Reviews Cardiology*, **12**, 576-587.
https://doi.org/10.1038/nrcardio.2015.91

[20] Klosky, A.L. (2009) Alcohol and Cardiovascular Diseases. *Expert Review of Cardiovascular Therapy*, **7**, 499-506. https://doi.org/10.1586/erc.09.22

[21] Afonso, L., Mohammad, T. and Theta, D. (2007) Crack Whips the Heart: A Review of the Cardiovascular Toxicity of Cocaine. *American Journal of Cardiology*, **100**, 1040-1043. https://doi.org/10.1016/j.amjcard.2007.04.049

[22] Parritz, E.D., Cunningham, N.J. and MacIsaac, A.I. (2016) The Cardiac Complications of Methamphetamines. *Heart, Lung and Circulation*, **25**, 325-332.
https://doi.org/10.1016/j.hlc.2015.10.019

[23] Yeo, K.K., Wijetunga, M., Ito, H., *et al.* (2007) The Association of Methamphetamine Use and Cardiomyopathy in Young Patients. *The American Journal of Medicine*, **120**, 165-171. https://doi.org/10.1016/j.amjmed.2006.01.024

[24] Schultz, S.E., Adams, M.J., Colin, S.D., *et al.* (2013) Long-Term Cardiovascular Toxicity in Children, Adolescents, and Young Adults Who Receive Cancer Therapy: Pathophysiology, Course, Monitoring, Management, Prevention, and Research Directions: A Scientific Statement from the American Heart Association. *Circulation*, **128**, 1927-1995. https://doi.org/10.1161/CIR.0b013e3182a88099

[25] Lip Shultz, S.E., Lipsitz, S.R., Salla, S.E., *et al.* (2005) Chronic Progressive Cardiac Dysfunction Years after Doxorubicin Therapy for Childhood Acute Lymphoblastic Leukemia. *Journal of Clinical Oncology*, **23**, 2629-2636.
https://doi.org/10.1200/JCO.2005.12.121

[26] Sorensen, K., Levitt, G.A., Bull, C., Droop, I. and Sullivan, I.D. (2003) Late Anthracycline Cardiotoxicity after Childhood Cancer: A Prospective Longitudinal Study. *Cancer*, **97**, 1991-1998. https://doi.org/10.1002/cncr.11274

[27] van der Pal, H.J., van Dalen, E.C., Hauptmann, M., *et al.* (2010) Cardiac Function in 5-Year Survivors of Childhood Cancer: A Long-Term Follow-Up Study. *Archives of Internal Medicine*, **170**, 1247-1255. https://doi.org/10.1001/archinternmed.2010.233

[28] van Dalen, E.C., van der Pal, H.J., Kook, W.E., Caron, H.N. and Kremer, L.C. (2006) Clinical Heart Failure in a Cohort of Children Treated with Anthracyclines: A Long-Term Follow-Up Study. *European Journal of Cancer*, **42**, 3191-3198.
https://doi.org/10.1016/j.ejca.2006.08.005

[29] Lip Shultz, S.E., Rifai, N., Dalton, V.M., *et al.* (2004) The Effect of Dexrazoxane on Myocardial Injury in Doxorubicin-Treated Children with Acute Lymphoblastic Leukemia. *The New England Journal of Medicine*, **351**, 145-153.
https://doi.org/10.1056/NEJMoa035153

[30] Cooper, L.T. (2009) Myocarditis. *The New England Journal of Medicine*, **360**, 1526-1538. https://doi.org/10.1056/NEJMra0800028

[31] Aretz, H.T., Billingham, M.E., Edwards, W.D., *et al.* (1987) Myocarditis. A Histo-

pathologic Definition and Classification. *The American Journal of Cardiovascular Pathology*, **1**, 3-14.

[32] Chow, L.H., Radio, S.J., Sears, T.D. and McManus, B.M. (1989) Insensitivity of Right Ventricular Endomyocardial Biopsy in the Diagnosis of Myocarditis. *Journal of the American College of Cardiology*, **14**, 915-920. https://doi.org/10.1016/0735-1097(89)90465-8

[33] Baughman, K.L. (2006) Diagnosis of Myocarditis: Death of Dallas Criteria. *Circulation*, **113**, 593-595. https://doi.org/10.1161/CIRCULATIONAHA.105.589663

[34] Linderman, I., Linderman, M., Kandloff, R., *et al.* (2008) Predictors of Outcome in Patients with Suspected Myocarditis. *Circulation*, **118**, 639-648. https://doi.org/10.1161/CIRCULATIONAHA.108.769489

[35] Abdel-At, H., Boyes, P., Zagorsk, A., *et al.* (2005) Diagnostic Performance of Cardiovascular Magnetic Resonance in Patients with Suspected Acute Myocarditis: Comparison of Different Approaches. *Journal of the American College of Cardiology*, **45**, 1815-1822. https://doi.org/10.1016/j.jacc.2004.11.069

[36] Friedrich, M.G., Sachem, U., Schulz-Manger, J., *et al.* (2009) Cardiovascular Magnetic Resonance in Myocarditis: A JACC White Paper. *Journal of the American College of Cardiology*, **53**, 1475-1487. https://doi.org/10.1016/j.jacc.2009.02.007

[37] Fabre, A. and Sheppard, M.N. (2006) Sudden Adult Death Syndrome and Other Non-Ischemic Causes of Sudden Cardiac Death. *Heart*, **92**, 316-320. https://doi.org/10.1136/hrt.2004.045518

[38] Ambrosio, A., Patti, G., Manzoni, A., *et al.* (2001) The Fate of Acute Myocarditis between Spontaneous Improvement and Evolution to Dilated Cardiomyopathy: A Review. *Heart*, **85**, 499-504. https://doi.org/10.1136/heart.85.5.499

[39] McCarthy, R.E., Boehmer, J.P., Aruban, R.H., *et al.* (2000) Long-Term Outcome of Fulminant Myocarditis as Compared with Acute (No Fulminant) Myocarditis. *The New England Journal of Medicine*, **342**, 690-695. https://doi.org/10.1056/NEJM200003093421003

[40] Maisch, B. and Panduit, S. (2013) Standard and Etiology-Directed Evidence-Based Therapies in Myocarditis: State of the Art and Future Perspectives. *Heart Failure Reviews*, **18**, 761-795. https://doi.org/10.1007/s10741-012-9362-7

[41] Linderman, I., Barth, C., Mahfud, F., *et al.* (2012) Update on Myocarditis. *Journal of the American College of Cardiology*, **59**, 779-792. https://doi.org/10.1016/j.jacc.2011.09.074

[42] Kuhl, U., Bauschinger, M., Nutrias, M., *et al.* (2005) High Prevalence of Viral Genomes and Multiple Viral Infections in the Myocardium of Adults with "Idiopathic" Left Ventricular Dysfunction. *Circulation*, **111**, 887-893. https://doi.org/10.1161/01.CIR.0000155616.07901.35

[43] Dec, G.W. and Foster, V. (1994) Idiopathic Dilated Cardiomyopathy. *The New England Journal of Medicine*, **331**, 1564-1575. https://doi.org/10.1056/NEJM199412083312307

[44] Opie, L.H., Comerford, P.J., Gersh, B.J. and Pfeffer, M.A. (2006) Controversies in Ventricular Remodeling. *The Lancet*, **367**, 356-367. https://doi.org/10.1016/S0140-6736(06)68074-4

[45] Alter, P., Rupp, H., Stoll, F., *et al.* (2012) Increased End Diastolic Wall Stress Precedes Left Ventricular Hypertrophy in Dilative Heart Failure—Use of the Volume-Based Wall Stress Index. *International Journal of Cardiology*, **157**, 233-238. https://doi.org/10.1016/j.ijcard.2011.07.092

[46] Sayer, G. and Bhat, G. (2014) The Renin-Angiotensin-Aldosterone System and Heart Failure. *Cardiology Clinic*, **32**, 21-32. https://doi.org/10.1016/j.ccl.2013.09.002

[47] Bharuch, T., Lee, K.J., Dabney, P.E., *et al.* (2015) Sudden Death in Childhood Cardiomyopathy: Results from a Long-Term National Population-Based Study. *Journal of the American College of Cardiology*, **65**, 2302-2310.

[48] Lakdawala, N.K., Winterfield, J.R. and Funke, B.H. (2013) Dilated Cardiomyopathy. *Circulation*: *Arrhythmia and Electrophysiology*, **6**, 228-237. https://doi.org/10.1161/CIRCEP.111.962050

[49] McNair, W.P., Sinatra, G., Taylor, M.R., *et al.* (2011) SCN5A Mutations Associate with Arrhythmic Dilated Cardiomyopathy and Commonly Localize to the Voltage-Sensing Mechanism. *Journal of the American College of Cardiology*, **57**, 2160-2168. https://doi.org/10.1016/j.jacc.2010.09.084

[50] Oromo, J., Kure, S., Shiba, T., *et al.* (2005) Electrophysiological and Histopathological Characteristics of Progressive Atrioventricular Block Accompanied by Familial Dilated Cardiomyopathy Caused by a Novel Mutation of Lamin A/C Gene. *Journal of Cardiovascular Electrophysiology*, **16**, 137-145. https://doi.org/10.1046/j.1540-8167.2004.40096.x

[51] Friedrich, M.G. and Marcotte, F. (2013) Cardiac Magnetic Resonance Assessment of Myocarditis. *Circulation*: *Cardiovascular Imaging*, **6**, 833-839. https://doi.org/10.1161/CIRCIMAGING.113.000416

[52] Marold, H., Boedecker, C., Wagner, A., *et al.* (2004) Cardiovascular Magnetic Resonance Assessment of Human Myocarditis: A Comparison to Histology and Molecular Pathology. *Circulation*, **109**, 1250-1258. https://doi.org/10.1161/01.CIR.0000118493.13323.81

[53] Gyotakus, N., Kigali, M., Stephens, D., Dawson, D., Muntons, F. and Muntons, P. (2006) Cardiac Tissue Velocities and Strain Rate in the Early Detection of Myocardial Dysfunction of Asymptomatic Boys with Duchenne's Muscular Dystrophy: Relationship to Clinical Outcome. *Heart*, **92**, 840-842. https://doi.org/10.1136/hrt.2005.067710

[54] Ashford, M.W., Liu, W., Lin, S.J., *et al.* (2005) Occult Cardiac Contractile Dysfunction in Dystrophin-Deficient Children Revealed by Cardiac Magnetic Resonance Strain Imaging. *Circulation*, **112**, 2462-2467. https://doi.org/10.1161/CIRCULATIONAHA.104.516716

[55] Dubuc, D., Mesne, C., Ledebur's, G., Decaux, J.Y., Waksman, G. and Became, H.M. (2005) Effect of Perindopril on the Onset and Progression of Left Ventricular Dysfunction in Duchenne Muscular Dystrophy. *Journal of the American College of Cardiology*, **45**, 855-857. https://doi.org/10.1016/j.jacc.2004.09.078

[56] Cooper, L.T., Baughman, K.L., Feldman, A.M., *et al.* (2007) The Role of Endomyocardial Biopsy in the Management of Cardiovascular Disease: A Scientific Statement from the American Heart Association, the American College of Cardiology, and the European Society of Cardiology. Endorsed by the Heart Failure Society of America and the Heart Failure Association of the European Society of Cardiology. *Journal of the American College of Cardiology*, **50**, 1914-1931.

[57] Kirk, R., Dip Chand, A.I., Rosenthal, D.N., *et al.* (2014) The International Society for Heart and Lung Transplantation Guidelines for the Management of Pediatric Heart Failure: Executive Summary. *The Journal of Heart and Lung Transplantation*, **33**, 888-909. https://doi.org/10.1016/j.healun.2014.06.002

[58] Ichida, F., Hanamachi, Y., Miyawaki, T., *et al.* (1999) Clinical Features of Isolated

Noncompaction of the Ventricular Myocardium: Long-Term Clinical Course, Hemodynamic Properties, and Genetic Background. *Journal of the American College of Cardiology*, **34**, 233-240. https://doi.org/10.1016/S0735-1097(99)00170-9

[59] Richardson, P., McKenna, W., Bristow, M., *et al.* (1996) Report of the 1995 World Health Organization/International Society and Federation of Cardiology Task Force on the Definition and Classification of Cardiomyopathies. *Circulation*, **93**, 841-842. https://doi.org/10.1161/01.CIR.93.5.841

[60] Baffa, G.M., Theine, G., Nava, A. and Dalla Volta, S. (1991) Cardiomyopathy: A Necessary Revision of the WHO Classification. *International Journal of Cardiology* Jan, **30**, 1-7. https://doi.org/10.1016/0167-5273(91)90117-8

[61] Gillum, R.F. (1986) Idiopathic Cardiomyopathy in the United States, 1970-1982. *American Heart Journal*, **111**, 752-755. https://doi.org/10.1016/0002-8703(86)90111-0

[62] Codd, M.B., Sugrue, D.D. and Gersh, B.J. (1989) Epidemiology of Idiopathic Dilated and Hypertrophic Cardiomyopathy. A Population-Based Study in Olmsted County, Minnesota, 1975-1984. *Circulation*, **80**, 564-572. https://doi.org/10.1161/01.CIR.80.3.564

[63] Codd, M.B., Sugrue, D.D., Gersh, B.J. and Melton 3rd, L.J. (1989) A Population-Based Study in Olmsted County, Minnesota, 1975-1984. *Circulation*, **80**, 564-572. https://doi.org/10.1161/01.CIR.80.3.564

List of Abbreviations

HF	Heart Failure
MI	Myocardial Infraction
NYHA	New York Heart Association
WHO	World Health Organization
CAD	Coronary Artery Disease
BNP	Brain Natriuretic Peptide
MRI	Magnetic Resonance Image
HIV	Human Immunodeficiency Virus
CD	Cluster of Differentiation
PCR	Polymerase Chane Reaction
BNP	B type Natriuretic peptide
ACC/AHA	American College of Cardiology/American Heart Association
Pt	Patient
Yrs.	Years
Rt	Right
CV	Cardiovascular
n	number
ECG	Electrocardiogram
ACE	Angiotensin-Converting Enzyme inhibitor
ARBs	Angiotensin Receptors Blockers
ARNI	Angiotensin Receptors Neurolysin Inhibitor
SPSS	Statistical Package for Social Sciences
RHD	Rheumatic Heart Disease
HTN	Hypertension
DM	Diabetes Mellitus
AF	Atrial Fibrillation
Hx	History
Ab	Abdominal
ICS	Intercostal Space
MCL	Midclavicular Line
S1	First heart sound
S2	Second heart sound
S3	Third heart sound
S4	Fourth heart sound
P	P Wave
LAD	Left Atrial Dilatation
RAD	Right Atrial Dilatation
DCM	Dilated Cardiomyopathy
PND	Paroxysmal Nocturnal Dyspnea
CXR	Chest X-ray
ECHO	Echocardiography
LVH	Left Ventricle Hypertrophy

RVH	Right Ventricle Hypertrophy
CBC	Complete Blood Count
LFT	Liver Function Test
RFT	Renal Function Test
PCI	Percutaneous Coronary Intervention