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REVIEW ARTICLE

Electrocardiographic abnormalities among patients with LV systolic dysfunction

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Abstract

Background: Heart failure (HF) is a public health problems particularly affecting older males. Ischemic heart disease (IHD) and non-ischemic disease could cause HF although with different electrocardiographic (ECG) abnormalities.

Aim: The present study aimed to determine the frequency of common ECG findings in ischemic vs non ischemic systolic LV dysfunction.

Patients and Methods: This is a cross-sectional prospective study which was conducted at Baghdad Medical City/ Department of cardiology and IBN AL- BITAR specialized center for cardiac surgery. The study included a total of 250 adult patients admitted with HF during the period from January/ 2023 till December/ 2023. Sociodemographic characteristics, electrocardiography, echocardiography findings and medication were gathered. Patients were categorized according to two groups according to HF cause: ischemic and non-ischemic.

Results: the age of the studied population was 62.63±13.22years with males representing 65.2%. Left axis deviation, atrial fibrillation (AF), Q-wave, left atrial enlargement and low voltage were the most common ECG abnormalities accounting for 28.8%, 28.4%, 28.4, 27.6%, and 23.6% of the patients, respectively. Left ventricular dysfunction is very common, of which mildly reduced, moderately reduced, and severely reduced ejection fraction were reported in 42.8%, 38% and 12% of the patients, respectively. In the majority of cases (72.8%), the cause of HF was IHD while in only 27.2% the cause was non-IHD.

Conclusions: Most patients with HF were males in their fifth decade. Diabetes mellitus, dyslipidemia, hypertension are most risk factors associated with HF. The majority of cases with HF are due to ischemic causes, while non-ischemic diseases responsible for about one-fourth of cases. Atrial fibrillation is common in non IHD, while Q-wave is more common in ischemic than non-ischemic HF with significant differences.

Introduction

Heart failure (HF) is a major public health problem. Despite advances in treatment and improved survival in recent decades, the annual mortality for HF remains high, reaching proportions of all adult deaths of 40.5% in men and 59.5% in women [1]. The diagnosis of HF is frequently made late, only when patients develop acute symptoms, making non-invasive, accurate, and cost effective means of detection a priority [2]. According to the European Society of Cardiology (ESC), an electrocardiogram (ECG) for HF is the basic examination that should be performed routinely in patients with suspected or known HF. ECG abnormalities increase the likelihood of HF (89% sensitivity) but have low specificity [3].

The presence of ECG abnormalities, especially in patients with HF, may depend on many factors (e.g. ischemia, HF etiology, electrolyte disturbances, pharmacotherapy) and is often observed. They may be helpful in determining the HF etiology and making therapeutic decisions (e.g. anticoagulation in atrial fibrillation, pacing in bradycardia, cardiac resynchronization therapy [CRT] when QRS complex is prolonged) [4]

ECG Abnormalities in Ischemic Heart Disease

The ECG waveform findings vary considerably depending on four major factors: (1) the duration of the ischemic process, (2) its extent (size and degree of transmural involvement), (3) its topography (anterior versus inferior-posterior-lateral or right ventricular), and (4) the presence of other underlying abnormalities (e.g., prior infarction, LBBB, Wolff Parkinson-White syndrome, or pacemaker patterns) because they can alter or mask the classic patterns [5,6].

Repolarization (ST-T Wave) Abnormalities

The earliest and most consistent ECG finding during acute severe ischemia is deviation of the ST segment. In normal conditions, the ST segment usually is nearly isoelectric, because almost all healthy myocardial cells attain approximately the same potential during the plateau phase of the ventricular action potential.. As a result, leads overlying the ischemic zone will record a negative deflection during electrical diastole and

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produce depression of the TQ segment [7]. When acute ischemia is transmural, the overall ST vector (whether caused by diastolic or systolic injury currents, or both) usually is shifted in the direction of the outer (epicardial) layers, and ST-segment elevation and sometimes tall, positive (hyperacute) T waves are recorded over the ischemic zone. Reciprocal ST-segment depression can appear in leads reflecting the contralateral surface of the heart. Occasionally, the reciprocal changes can be more apparent than the primary ST-segment elevations [8].

QRS Complex Changes

Abnormal Q waves were considered markers of transmural myocardial infarction, whereas subendocardial (nontransmural) infarcts were thought not to produce Q waves[9]. In certain patients, fragmentation of the QRS complex, even without Q waves, may be a marker of myocardial scarring from ischemic or non-ischemic causes [10].

Other Ischemic ST-T Patterns

Reversible transmural ischemia, such as that caused by coronary vasospasm, may result in transient ST-segment elevation. Depending on the severity and duration of such non-infarction ischemia, the ST-segment elevation either can resolve within minutes or can be followed by T wave inversion that can persist for hours or even days [11].

Myocardial Infarction with Bundle Branch Blocks

The diagnosis of myocardial infarction (MI) often is more difficult when the baseline ECG shows a bundle branch block pattern or when bundle branch block develops as a complication of the MI, because LBBB alters the early and the late phases of ventricular depolarization and produces secondary ST-T changes. The diagnosis of Q wave infarction usually is not impeded by the presence of right branch bundle block (RBBB), which affects primarily the terminal phase of ventricular depolarization [12].

The following points summarize the ECG signs of MI in LBBB:

1. ST-segment elevation with tall, positive T waves frequently is seen in the right precordial leads with uncomplicated LBBB. Secondary T wave inversions are characteristically seen in the lateral precordial leads. However, the appearance of ST-segment elevations in the lateral leads or ST-segment depressions or deep T wave inversions in leads V1 to V3 strongly suggests underlying ischemia. More marked ST-segment elevations (>0.5 mV) in leads with QS or rS waves also may be caused by acute ischemia, but false-positive findings occur, especially with large-amplitude negative QRS complexes. Use of the ratio of the absolute amplitude of the STsegment to S wave, determined in any relevant lead of greater than 0.25 has been proposed as having a greater accuracy [13].
2. The presence of QR complexes in leads I, V5, or V6 or in II, III, and aVF strongly suggests underlying MI.
3. Chronic MI also is suggested by notching of the ascending part of a wide S wave in the midprecordial leads or the ascending limb of a wide R wave in lead I, aVL, V5 , or V6

Abnormalities in Dilated Cardiomyopathy

The most common ECG findings include left ventricular hypertrophy (LVH), T-wave inversions (TWI), left axis deviation, pathological Q waves, and conduction alterations. While such ECG abnormalities were long considered non-specific for DCM patients, newly acquired knowledge on genotype–phenotype correlations has now made the ECG a useful tool to guide the etiological diagnosis (“red flags”) and provide prognostic stratification [14]. Maximum P-wave duration and P-wave dispersion (PWD), defined as the difference between maximum and minimum P-wave duration, have been found to be higher in patients with DCM than in healthy control subjects [15]. These anatomical and structural abnormalities underlie the increased risk of developing atrial fibrillation (AF) in patients with DCM [14]. In literature, the first-degree atrioventricular (AV) block has a prevalence of 10%– 23% in the DCM population, although advanced AV blocks can also be found in these patients [16].

Right bundle branch block has a prevalence of 2%–6% among patients with DCM and is frequently associated with neuromuscular disorders [17]. Left bundle branch block is far more common than RBBB. LBBB is present in approximately one-third of DCM patients [18]. Several studies have previously demonstrated the negative prognostic impact of LBBB, with an increased risk of all-cause mortality, by determining an asynchronous contraction of the LV and a progressive worsening of systolic function [19]. Left posterior fascicular block (LPFB), which is uncommon in the general population, has been associated in a recent small study with extensive LV scarring and an increased risk of sudden death [20].

Low electrocardiographic QRS voltages (LQRSV) are defined in the literature as a nadir-to-peak QRS amplitude of <5 mm in all limb leads and <10 mm in all precordial leads [21]. In DCM patients, LQRSV have been described in 6% and may be observed only in limb leads (most frequently), in precordial leads, or both [22]. Several criteria are used to diagnose the presence of Q waves: Q-wave duration of ≥40 ms, absolute depth of >3 mm, or amplitude of ≥25% of the ensuing R-wave (23). Q waves have been described more frequently in anterior and lateral leads in DCM, despite normal coronary arteries [24].

Fragmented QRS is a narrow QRS complexes with the presence of an additional R-wave (R’) or notching in the nadir of the R-wave or the S wave or the presence of >1 R’ (fragmentation) in two contiguous leads. fQRS is a marker of depolarization abnormality present in a significant number of patients with DCM (more than 20%) [25]. The fQRS has also been shown to be associated with significant intraventricular dyssynchrony in patients with non-ischemic cardiomyopathy, narrow QRS, and sinus rhythm; such findings suggest the possibility of using fQRS as a predictor in identifying patients who can benefit from CRT, but these data have not yet been confirmed [26].

T wave inversion (TWI) was described as a T inversion of ≥0.1 mV in depth in ≥2 contiguous leads, in the absence of LBBB. The prevalence of TWI in the DCM population, as reported by the literature, is 15%–45%. The leads most frequently presenting TWI are inferior, antero-lateral, and inferolateral. The QT interval, while generally normal in DCM patients, has been shown to be of potential use in sudden cardiac death risk stratification in DCM when abnormal [27].

ECG abnormalities and arrhythmias

Any variety of ventricular arrhythmia can be found in DCM patients, from premature ventricular contractions (PVCs) and non-sustained and sustained monomorphic ventricular tachycardia (NSVT and SVT, respectively) to polymorphic ventricular tachycardia and ventricular fibrillation (VF). PVCs and NSVT may be found in up to 40% of patients with DCM, but their role is not clear in the literature [28]. It is well established that the frequency of arrhythmias increases with the severity of heart failure, worsening of the ejection fraction, and the New York Heart Association (NYHA) class. Recent data suggest that both genetic

and MRI findings can contribute to risk stratification [29].

ECG Abnormalities in mitral valve disease

When significant regurgitation is found, however, ECG signs of left ventricular enlargement associated with an abnormal P wave or atrial fibrillation are usually observed [30]. In mitral valve prolapse, repolarization abnormalities are frequently found in II, III, aVF, and left precordial leads. Atrial arrhythmias are common, especially frequent premature atrial complexes and atrial fibrillation, and ventricular arrhythmias might be seen, especially when mitral prolapse is severe, in which case the ECG is rarely normal [31].

ECG Abnormalities in aortic valve disease

In the early stages of left ventricular enlargement, there is usually a pattern of qR morphology with positive T wave in left lateral leads, which is more evident with aortic regurgitation then with aortic stenosis (a deeper "q" wave and a taller T wave). This pattern has been considered a result of diastolic hemodynamic overload. In advanced cases both aortic stenosis and aortic regurgitation generally show a similar morphology, known as "strain pattern" (somewhat depressed ST segment followed by a negative and asymmetric T wave). Although the ST segment/T wave pattern is similar in both cases, in aortic regurgitation, the R wave is often still preceded by a usually small Q wave, which usually decreases over time, whereas in aortic stenosis, QRS complex morphology tends to be a pure R wave. Sometimes, a mixed pattern is seen, T wave more negative than usual and/or more symmetrical, or a more marked decrease of ST segment due to an added primary factor, such as associated ischemia or myocardial compromise or due to drug effects.

The so called pattern of diastolic overload is observed in the early stages of any type of ventricular enlargement due to aortic valve disease, but not in the ventricular enlargement of isolated aortic regurgitation. The "strain pattern," is a pattern of systolic overload observed in more advanced stages of aortic valve disease, regardless of the predominance of stenosis or regurgitation. On the other hand, it has been proven that the presence or absence of a "Q" wave in V5–V6 is more directly related to the degree of septal fibrosis (more fibrosis, less "Q" wave) than the type of lesion [32]. Patients with advanced aortic valvular disease frequently present with ventricular arrhythmias and intraventricular blocks and AV block (calcification of the aortic valve), even more than those with advanced mitral valve disease, but the latter more frequently present atrial fibrillation [33].

Patients and Methods

This is a cross-sectional prospective study which was conducted at Baghdad Medical City/ Department of cardiology and Ibn-Al-Baitar Center for Cardiac Surgery. The study included a total of 250 adult patients admitted with HF during the period from January/ 2023 till December/ 2023. Definite heart failure was defined as a combination of the presence of at least one of the typical signs or symptoms of heart failure, such as breathlessness at rest or during exertion, ankle oedema and pulmonary crepitations, and confirmation by objective evidence of cardiac dysfunction (chest X-ray, echocardiography). This definition is in accordance with the criteria of the European Society of Cardiology. The study was approved by Iraqi Council for Medical Specializations.

Inclusion criteria for ischemic group

- Patients aged 18 years and above form both sexes
- Documented diagnosis of systolic LV dysfunction, defined as an ejection fraction (EF) For those with HFmrEF, with LVEF between 41% and 49%, and HFrEF(EF<40% according to ESC guidelines
- Evidence of ischemic heart disease, including at least one of the following:

a. History of myocardial infarction.

b. Coronary angiography showing ≥70% stenosis in at least one major coronary artery.

c. Prior revascularization procedure (e.g., percutaneous coronary intervention or coronary artery bypass grafting) for significant coronary artery

Inclusion criteria to non-ischemic group

1. Absence of significant coronary artery disease, confirmed by one or more of the following:

a. Coronary angiography showing <50% stenosis in all major coronary arteries.

b. Absence of prior myocardial infarction or revascularization procedure.

c. Negative stress test without evidence of myocardial ischemia

2. Presence of other etiologies associated with non-ischemic systolic LV dysfunction, such as:

a. Dilated cardiomyopathy.

b. Hypertrophic cardiomyopathy.

c. Valvular heart disease.

Exclusion Criteria

- Newly diagnosed women with HF
- Patients with acute myocardial infarction or unstable angina
- Patients with paced ventricular rhythm
- Patients with severe comorbidities that may confound the interpretation of ECG findings or impact clinical outcomes, such as end-stage renal disease requiring dialysis, advanced liver disease, or terminal cancer
- Inadequate ECG quality: such as significant artifact or technical issues affecting waveform interpretation, may be excluded from analysis.

Statistical Analysis

All statistical analyses were performed using SPSS software. Categorical data are presented as numbers of patients and percentages, continuous data are presented as mean and standard deviation. Categorical data were compared by the Pearson's $\chi 2$ test or Fisher's exact test as required. Continuous data were compared by student t-test. A P value less than 0.05 is considered as a statistically significant difference.

Results

Baseline Characteristics of the Patients at Presentation

As indicated in table 1, the age of the studied population was 62.6313.22±years (range =17-93). The majority were males (65.2%). The most common NYHA class was III accounting for 151 patients (60.4%), followed by class II (25.6%) The majority of patients (82.4%) have PMH of HTN, 75% with CAD, 56.4% with DM, 25.2% with renal disease, 7.2% with hyperlipidemia, and another 7.2% with hyperthyroidism.

Table 1: Demographic characteristics the study population

Variables	Value
Age, years	
Mean±SD	62.6313.22±
Range	17-93
≤65	162(64.8%)
>65	88(35.2%)
Sex	
Male	163(65.2%)
Female	87(34.8%)
NYHA classification of HF	
I	12(4.8%)
II	64(25.6%)
III	151(60.4%)
IV	23(9.2%)
Past medical history*	
Hypertension	206(82.4%)
CAD	206(82.4%)
DM	141(56.4%)
Renal disease	63(25.2%)
Hyperlipidemia	18(7.2%)
Thyroid disease	18(7.2%)

SD: standard deviation; NYHA = New York Heart Association

*the patient might have more than one comorbidity.

Echocardiographic findings

According to the echocardiographic findings, left atrial size was enlarged in 111 patients (44.4%) while the right atrial size was enlarged in 26 patients (10.4%) patients. Similarly, the left ventricle was dilated in 101 patients(40.4%) and the right ventricle was dilated in 25(10%). The mean left ventricular ejection fraction was 36.97%±8.26 (range =20-55%). Mildly reduced, moderately reduced, and severely reduced ejection fraction were reported in 48%, 38% and 12% of the patients, respectively.

The mean fraction shortness of the study population was 17.62±3.78 (range =8-26). Arrhythmias were common and including 28.4% with AF, 13.6% with premature ventricular conduction, 6.4% with Ashman phenomenon, 2.8% with premature atrial conduction and 0.8% with ventricular tachycardia. According to valvular involvement, 32% have mild MR, 17.6% with moderate MR, 11.2% with severe TR, 10.4% with mild AR, 6.8% with moderate TR, and 2.8% with aortic stenosis. On the other hand, 30% have no valvular disease as shown in Table 2.

Table 2: Echocardiography

Variables	Value
Left atrial size	
Normal	139(55.6%)
Enlarged	111(44.4%)
Right atrial size	
Normal	224(89.6%)
Enlarged	26(10.4%)
Left ventricular dimension	
Normal	149(59.6%)
Dilated	101(40.4%)
Right ventricular dimension	
Normal	225(90%)
Dilated	25(10%)
Left ventricular ejection fraction, %	
Mean±SD	36.97±8.26
Range	20-55
Mildly reduced	120(48%)
Moderately reduced	95(38%)
Severely reduced	30(12%)
Arrhythmias	
Atrial fibrillation	71(28.4%)
Premature ventricular conduction	34(13.6%)
Ashman phenomenon	16(6.4%)
Premature atrial conduction	7 (2.8%)
Ventricular tachycardia	2(0.8%)
Presence of valvular disease	
None	75(30%)
Mild mitral regurgitation	80(32%)
Moderate mitral regurgitation	44(17.6%)
Severe tricuspid regurgitation	28(11.2%)
Mild aortic regurgitation	26(10.4%)
Moderate tricuspid regurgitation	17(6.8%)
Aortic stenosis	7(2.8%)

Medication

Patients of this study were prescribed different medications (solely or in combination), in descending order, loop diuretics in 58.4%, beta blockers in 57.2%, aspirin in 54.8%, statin in 52.8%, antiplatelet in 49.2%, aldactone in 37.2%, SGLT-2 in 36.4%, heparin in 32.8%, ACEI in 16.8%, pantor in 10.8%, insulin injections in 10.4%, digoxin in another 10.4%, isordil in 9.2%, cordarone in 6%, amplodipine in 4.8%, vastarel in 4%, and other

drugs in 12% of patients as presented in Table 3.

Table 3: Medications prescribed to the patients

Drugs	Frequency(%)
Loop diuretics	146(58.4%)
Beta-blockers	143(57.2%)
Aspirin	137(54.8%)
Statin	132(52.8%)
Antiplatelet	123(49.2%)
Aldactone (Spironolactone)	93(37.2%)
Sodium-glucose co-transporter-2 inhibitors (SGLT-2i)	91(36.4%)
Heparin	82(32.8%
Angiotensin Converting enzyme inhibitors	42(16.8%)
Pantor (Pantoprazole)	27(10.8%)
Insulin	26(10.4%)
Digoxin	26(10.4%)
Isordil (Isosorbide dinitrate)	23(9.2%)
Cordarone (Amiodarone hydrochloride)	15(6%)
Amlodipine	12(4.8%)
Vastarel (Trimetazidine)	10(4%)
Others	30(12%)

Electrocardiographic changes of the study population

Table 4 show the ECG changes of the study population. Only 7 patients (2.8%) had normal ECG findings. Conduction defects were common and appeared as left axis deviation in 71 patients (28.8%), LBBB in 26 patients (10.4%), complete heart block in 15 patients (6%), RBBB in 11 patients (4.4%) and 1st degree heart block in 9 patients(3.6%). Seventy-two patients (28.8%) presented with left axis deviation, , 28.4% with Q-wave, 27.6 with LAE, 23.6% with low voltage, 14.4% with prolonged QT interval, 9.2% with lateral ischemia, 8.8% with left ventricular hypertrophy, 10.8% with poor R wave progression. Accordingly the Goldberg's triad was found in 12 patients (4.8%). A 11.6% of patients had intraventricular conduction delay, , another 8% with ST elevation, 5.2% with complete heart block, 4.4% with right BBB, 4% with sinus tachycardia, 3.6% with first degree heart block, 8% with ST depression, 2.8% with sustained ST elevation, and another 2.8% with other changes. On the other hand, the ECG was normal in 2.8% of patients.

Table 4: Electrocardiographic changes

ECG changes	Frequency(%)
Normal ECG	7(2.8%)
Conduction defects	
Left axis deviation	72(28.8%)
Intraventricular conduction delay	29(11.6%)
Left bundle branch block	26(10.4%)
Complete heart block	15(6%)
Right bundle branch block	11(4.4%)
1st degree heart block	9(3.6%)
Right axis deviation	6(2.4%)
Evidence of myocardial fibrosis	
Q-wave	71(28.4%)
Low voltage	59(23.6%)
Fragmented QRS	12(4.8%)
Left atrial enlargement	69(27.6%)
Low voltage	59(23.6%)
Prolonged QT interval	36(14.4%)
Lateral ischemia	23(9.2%)
Left ventricular hypertrophy	22(8.8%)
Poor R wave progression	28(10.8%)
ST elevation	20(8%)
Goldberg's triad	12(4.8%)
Sinus tachycardia	10(4%)
ST depression	20(8%)
Sustained ST elevation	7(2.8%)
Right atrial enlargement	6(2.4%)

Association of demographic data with causes of heart failure

Patients with IHD as a cause of HF were significantly older than those of non-IHD cause 63.81±10.81 years versus 59.46±17.89 years. The NYHA classification of HF was significantly different between those with and without IHD (p <0.001). Moreover, the PMH of HTN, CAD, and DM were significantly higher (p <0.001) in IHD group as compared to those with non-IHD group (Table 5).

Table 5: Association of demographic characteristics with cause of HF

Variables	IHD (n=182)	Non-IHD (n=68)	p-value
Age, years			
Mean±SD	63.81±10.81	59.46±17.89	0.020
Range	39-87	17-93	
≤65	114(62.64%)	48(70.59%)	
>65	68(37.36%)	20(29.41%)	

Sex			
Male	116(63.74%)	47(69.12%)	0.427
Female	66(36.26%)	21(30.88%)	
NYHA classification of HF			
I	10(5.5%)	2(2.94%)	
II	57(31.32%)	7(10.29%)	0.004
III	99(54.4%)	52(76.47%)	
IV	16(8.79%)	7(10.29%)	
Past medical history			
Hypertension	166(91.21%	40(58.82%)	<0.001
CAD	166(91.21%)	22(32.35%)	<0.001
DM	118(64.84%)	23(33.82%)	<0.001
Renal disease	50(27.47%)	13(19.12%)	0.176
Thyroid disease	15(8.24%)	3(4.41%)	0.297
Hyperlipidemia	13(7.14%)	3(4.41%)	0.432
LVEF, %			
Mean±SD	38.87±7.32	31.57±8.65	<0.001
Range	20-55	20-50	
Mildly reduced	78(42.86%)	52(76.47%)	
Moderately reduced	93(51.1%)	14(8.33%)	
Severely reduced	11(6.04%)	2(1.19%)	
Arrhythmias			
Atrial fibrillation	37(20.33%)	34(50%)	<0.001
PVC	22(12.09%)	12(17.65%)	0.254
Ashman phenomenon	9(4.95%)	7(10.29%)	0.124
PAC	5(2.75%)	2(2.94%)	0.934
VT	1(0.05%)	1(1.47%)	0.112
Valvular disease			
None	64(35.16%)	11(61.18%)	0.004
Mild MR	67(36.81%)	13(19.12%)	0.008
Moderate MR	27(14.84%)	17(25%	0.060
Severe TR	14(7.69%)	14(20.59%)	0.004
Mild AR	20(11%)	6(8.82%)	0.609
Moderate TR	4(2.2%)	13(19.12%)	<0.001
Aortic stenosis	3(1.65%)	4(5.88%)	0.090

Association of echocardiographic data with causes of heart failure

Table 6 show the echocardiographic data of those with and without IHD. The left atrial size, left ventricular dimension, right ventricular dimension, LVEF%, and fraction shortness were significantly different (p <0.001) between patients with and without IHD groups. Moreover, right atrial size was also significantly different (p =0.006) between those with and without IHD. Furthermore, mild MR, moderate and severe TR was significantly different between the two groups.

Table 6: Association of echocardiography findings with cause of HF

Variables	IHD (n=182)	Non-IHD (n=68)	p-value
Cardiac Chambers			
Left atrial size			
Normal	126(69.23%)	13(19.12%)	<0.001
Enlarged	56(30.77%)	55(80.88%)	
Right atrial size			
Normal	169(92.86%)	55(80.88%)	0.006
Enlarged	13(7.14%)	13(19.12%)	
LV dimension			
Normal	127(69.78%)	22(32.35%)	<0.001
Dilated	55(30.22%)	46(67.65%)	
RV dimension			
Normal	172(94.51%)	53(77.94%)	<0.001
Dilated	10(5.49%)	15(22.05%)	

Association of medication used and causes of heart failure

In patients with IHD and non-IHD, those who use loop diuretics (98 versus 48), beta blockers (102 versus 14), aspirin (116 versus 21), statin (111 versus 21), antiplatelet (109 versus 14), aldactone (60 versus 33), ACEI (36 versus 6), digoxin (11 versus 15), and isordil (23 versus none) with significant difference (Table 7).

Table 7: Association of different medications with cause of HF

Drugs	IHD (n=182)	Non-IHD (n=68)	p-value
Loop diuretics	98(53.85%)	48(70.59%)	0.017
Beta-blockers	102(56.04%)	14(20.59%)	<0.001
Aspirin	116(63.74%)	21(30.88%)	<0.001
Statin	111(61%)	21(3088%)	<0.001
Antiplatelet	109(59.89%)	14(20.59%)	<0.001
Aldactone	60(32.97%)	33(48.53%)	0.023
SGLT2	62(34.07%)	29(42.65%)	0.210
Heparin	56(30.77%)	26(38.24%)	0.263
ACEI	36(19.78%)	6(8.82%)	0.039
Pantor	23(12.64%)	4(5.88%)	0.170
Insulin	23(12.64%)	3(4.41%)	0.064
Digoxin	11(6.04%)	15(22.05%)	<0.001
Isordil	23(12.64%)	0(0%)	<0.001
Caradrone	10(5.49%)	5(7.35%)	0.582
Amlodipine	10(5.49%)	2(2.94%)	0.401
Vastarel	7(3.85%)	3(4.41%)	1.00
Others	23(12.64%)	7(10.29%)	0.312

Association of electrocardiographic changes and causes of heart failure

Atrial fibrillation was present in 20.33% of those with IHD and 50% with those with non-IHD with significant difference. Similarly, left BBB was present in 6.04% of those with IHD 22.05% of those with non-IHD with significant difference. Q v1-3 was present in 35 versus 6 in IHD versus non-IHD, respectively with significant difference. Lateral ischemic changes was present in 9.34% versus 0% of IHD and non-IHD, respectively with significant difference as indicated in Table 8.

Table 8: Association of ECG changes with cause of HF

ECG changes	IHD (n=182)	Non-IHD (n=68)	p-value
Normal ECG	4(2.2%)	3(4.41%)	0.394
Conduction defects			
Left axis deviation	55(30.22%)	17(25%)	0.438
Intraventricular conduction delay	22(12.09%)	7(10.29%)	0.693
Left bundle branch block	11(6.04%)	15(22.05%)	<0.001
Complete heart block	9(4.95%)	4(5.88%)	0.766
Right bundle branch block	7(3.85%)	4(5.88%)	0.485
1st degree heart block	7(3.85%)	2(2.94%)	0.733
Right axis deviation	6(3.3%)	0(0%)	0.194
Evidence of myocardial fibrosis			
Q-wave	60(32.97%)	11(16.18%)	0.009
Poor R wave progression	21(11.54%)	6(8.82%)	0.272
Fragmented QRS	9(4.95%)	3.(4.41%)	1.00
Prolonged QT	25(13.74%)	11(16.18%)	0.625
Left atrial enlargement	55(30.22%)	14(20.59%)	0.153
Left ventricular mass	14(7.69%)	8(11.76%)	0.538
ST elevation	15(8.24%)	5(7.35%)	0.805
Goldberger's triad	11(6.04%)	1(1.47%)	0.132
Sinus tachycardia	6(3.3%)	4(5.88%)	0.467
ST depression	13(71.4%)	7(10.29%)	0.342
Sustained ST elevation	7(3.85%)	0(0%)	0.195
Low voltage	41(22.53%)	18(26.47%)	0.272

Table 9: Comparison of ECG and Echo findings

Variables	ECG	Echo	p-value
LAE	59(23.6%)	111(44.4%)	<0.001
RAE	6(2.4%)	26(10.4%)	<0.001
LVmass	22(8.8%)	101(40.4%)	<0.001

In this table, p-values indicate the statistical significance of the correlation between each ECG finding and the corresponding echocardiographic measure of chamber enlargement.

Table 10: Association of ECG findings with the type of LV dysfunction

ECG changes	Mildly reduced (n=107)	Moderately reduced (n=95)	Severely Reduced (n=13)	p-value
Conduction defects				
Int. conduction delay	13(12.15%)	6(6.32%)	0(0%)	0.001
LBBB	11(10.28%)	7(7.37%)	1(7.7%)	0.144
CHB	6(5.61%)	4(4.21%)	1(7.7%)	0.829
RBBB	8(7.48%)	2(2.11%)	0(0%)	0.239
1st degree HB	2(1.87%)	3(3.16%)	0(0%)	0.058
Left axis deviation	25(23.36%)	34(35.79%)	4(30.8%)	0.817
Atrial fibrillation	28(26.17%)	24(25.26%)	5(38.5%)	0.180
Prolonged QT	20(18.69%)	9(9.47%)	2(15.4%)	0.088
LAE	24(22.43%)	31(32.63%)	3(23.1%)	0.861
PVC	16(14.95%)	12(12.63%)	0(0%)	0.285
Q-wave	29(27.1%)	36(37.89%)	1(7.7%)	0.050
LV mass	7(6.54%)	12(12.63%)	0(0%)	0.517
Low voltage	22(20.56%)	23(24.21%)	2(15.4%)	0.395
Ashman phenomenon	8(7.48%)	1(1.05%)	0(0%)	0.233
ST elevation	7(6.54%)	12(12.63%)	0(0%)	0.517
Goldberger's triad	7(6.54%)	4(4.21%)	0(0%)	0.461
Sinus tachycardia	3(2.8%)	4(4.21%)	1(7.7%)	0.145
ST changes				
ST depression	3(2.8%)	13(13.68%)	1(7.7%)	0.077
Sustained ST elevation	0(0%)	7(7.37%)	0(0%)	0.022
PAC	4(3.74%)	3(3.16%)	0(0%)	0.557
Right atrial enlargement	4(3.74%)	2(2.11%)	0(0%)	0.451
Right axis deviation	0(0%)	1(1.05%)	0(0%)	<0.001
Others	3(2.8%(	6(6.32%)	2(15.4%)	0.234

Discussion

Many comorbid diseases were encountered in those with HF of the current study, intracardiac like CAD and extracardiac like hypertension, DM, renal disease, hyperlipidemia, and thyroid disease. Many studies document such finding like Levy D, Garrison RJ et al [34], This study from the Framingham Heart Study cohort investigates the prognostic implications of hypertension, a major contributor to left ventricular hypertrophy, in patients with LV systolic dysfunction. Ashoka devkota et al [35] another study it shows the HTN was 82.8% and DM 49% prevalent in LV systolic dysfunction. In the current study the number of patients were having diabetes was 141(56.4%). DM is considered one of the strongest risk factors for cardiovascular disease, including IHD. The age of patients in this study was 62.6313.22± years (range =17-93). Most of the burden of heart failure occurs in people aged 65 years or over. It was documented that the prevalence of HF rises steeply with age [36].

A larger proportion of the patients (65.2%) of the current study were males. Also, our study showed predominance of males over females with affection by IHD. In their studies, Lund and Mancini [37] and Khan et al. [38] shows men were more

commonly affected than women by IHD. The present study showed that IHD was prevalent as a cause of HF in 72.8%. A finding that is indicated by other researchers. IHD is recognized was the main etiological factor in more than 50% of HF patients in North America and Europe [39]. Moreover, Abdissa et al. [40] in an Ethiopian study found the same trend In the present study, echocardiographic data revealed enlarged left atrium and ventricle in the majority of patients with HF as compared to the right atrium and ventricle. This is in agreement with many studies worldwide [40,41].

In our study the normal ECG found in only 7 (2.8%) and this with agreement with kamilu u sani et al, (2008 Electrocardiographic abnormalities in patients with heart failure), in which only 2 patients (1.8%) had normal ECG. In the current study the LBBB was seen in 26 (10.4%),while in kamilu et al the prevalence of LBBB was seen in 6 patients (8.5%) out of 71 and this due to difference in number of patients enrolled in current study. In current study the Q wave significantly seen in 60 (32.97%) of IHD group($p<0.009$) as demonstrated by Lin et al [42] study.

Regarding the association of ECG abnormalities with type of LV dysfunction in the current study interventricular conduction delay was prevalent in mildly reduced LVEF($p<0.001$) in contrast to Kamilu et al 66 in which the IVCD not prevalent 4(5.6%) out of 71 participant this could be explained by small cohort , so those patient can be targeted as for follow up for early detection of LV remodeling and function deterioration. In our study the 55 (80.88) patients were have dilated LA on electrocardiography in non IHD group while in Kamilu M Karaye, Mahmoud et al were found the dilated LA was in 45 (63.4%) out of 71 patients with IHD and reduced EF.

Many types of valvular diseases were evident in patients with HF in the current study. Falk et al. [43], document that Valvular heart diseases are major causes of acute and chronic heart failure. In the majority of this study cases, mitral regurgitation represents the most frequent valvular disease. This finding was in harmony with Iung et al. [44] who finds mitral regurgitation represent the most frequent etiology of severe native valvular heart disease, frequently associated with congestive HF (50%). In recent study, Pokhrel Bhattarai et al., showed a high prevalence of long PR interval, left ventricular hypertrophy, pathological Q waves, long JTc, Q waves, and LBBB in HF with reduced EF. However, they didn't report ECG changes in HF with mildly reduced EF [45] . A 6.04% of IHD patient in the current study exhibited LBBB. In Jain et al.[46] study, the prevalence of LBBB was 4% . In Lopes et al. [47] study, LBBB was present in 1.7%.

In our study, incidence of patients with AF was higher in patients with IHD. Similar finding was demonstrated in Kannel et al. study [48] that showed patients with CHD tend to develop AF. About 30% of patients with AF have CAD. Conversely, 15% of subjects with IHD experience at least one episode of AF during their lifetime. In the present study, AF was more frequent in patients with non-ischemic heart disease (50%) than those with IHD (20.33%).

In contrast to the present study, Kamal et al. [49] included 50 Egyptian patients with HF (30 ischemic and 20 non- ischemic) they found that AF was more frequent in ischemic than non-ischemic patients. This variation could be explained by the variation in general characteristic of patients between the our and Egyptian study, and the small sample size of the Egyptian study. In the present study, fragmented Q was more frequent in patients with IHD than those without IHD. FQRS has also been reported in a variety of cardiac conditions such as ischemic and dilated cardiomyopathy, sarcoidosis, myocarditis, arrhythmogenic ventricular dysplasia..

In the present study, all cases of sustained ST-elevation were within ischemic HF group, although the difference was not significant. Sustained ST-segment elevation, beyond the acute phase of myocardial infarction, is indicative of ongoing myocardial injury or ischemia and is associated with a heightened risk of adverse cardiac events.

In the present study, PVC and PAC were reported in 13.6% and 2.8% of patients with HF and did not differ significantly between ischemic and non-ischemic type. PVCs and PACs are ectopic beats originating from the ventricles and atria, respectively, and their presence may indicate underlying electrical instability or structural heart disease. Isolated PVCs and PACs are common and often benign. In the present study, low voltage QRS accounted for 23.6% of patients and there was no significant difference in this abnormality between ischemic and non-ischemic HF. While low voltage QRS complexes are nonspecific findings, they have been associated with an increased risk of adverse cardiac events. Therefore, the identification of low voltage QRS complexes in our study population may serve as a marker for further evaluation and risk stratification, guiding clinical management decisions and prognostic assessment [50].

Conclusion

Most patients with HF were males in their fifth decade. Diabetes mellitus, dyslipidemia, hypertension are most risk factors associated with HF. The majority of cases with HF are due to ischemic causes, while non-ischemic diseases responsible for about one-fourth of cases. Left ventricular dysfunction is very common, of which mildly reduced, moderately reduced, and severely reduced ejection fraction were reported in 42.8%, 38% and 12% of the patients, respectively. Loop diuretics, beta blockers, aspirin, statin, and antiplatelet are the most common medications prescribed for patients with HF. The vast majority of patients with HF display ECG changes with left axis deviation, AF, Q-wave, LVE and low voltage are the most common abnormal findings. Atrial fibrillation commonest electrocardiographic abnormalities among patients with non IHD, while Q-wave is more common in ischemic than non-ischemic HF.

Recommendation

ECG abnormalities are the stigma of heart failure whether ischemic or non-ischemic. Therefore, ECG should be the first tool in patients referred to the cardiac outpatient clinic on suspicion of heart failure. Further studies including the association of ECG abnormalities with type of LV dysfunction are required in order the investigate the ECG abnormalities in those patients Further study to compare the ECG abnormalities in ischemic LV dysfunction vs LV dysfunction with preserve EF.

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