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A TEN YEAR STUDY OF MANAGEMENT OF CHRONIC HEART FAILURE IN A TERTIARY HOSPITAL IN THE SOUTH WEST NIGERIA.

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ABSTRACT

Chronic Heart Failure (CHF) occurs when the heart is incapable of maintaining sufficient blood flow to accommodate tissue perfusion and metabolic requirements. A retrospective study of ten years of management of chronic heart failure (CHF) at the University College Hospital Ibadan was made. The primary aim of this study was to assess the rational pharmaco-therapeutic approach in the management of chronic heart failure at the University College Hospital Ibadan during a period of ten years. A total number of 202 case notes in the medical records of the outpatients and inpatients of the Cardiology Unit were reviewed. The study population was made up of 109 males (54%) and 93 females (46%). The mean age of subjects was 56.01 years (± 14.74). A review of the drug management indicated that the prescribed drugs included; loop diuretics 176 (87.1%), thiazide diuretics 19 (9.4%), potassium sparing diuretics 162 (80.2%), amiloride and hydrochlorothiazide 71 (35.1%), digoxin 33 (16.3%), captopril 13 (6.4%), lisinopril 109 (54.0%), enalapril 42 (20.8%), ramipril 28 (13.9%), antibiotics 140 (69.3%), antiplatelet 128 (63.4%), anticoagulant 17 (8.4%), calcium channel blocker 68 (33.7%), methyldopa 46 (22.8%), beta blocker 16 (7.9%), enalapril and hydrochlorothiazide 19 (9.4%), oral hypoglycemic agent 6 (3.0%), non-steroidal anti-inflammatory drugs (NSAIDS) 37 (18.3%), while other prescribed drugs was 148 (73.3%). Side effects documented include cough 32 (15.8%), dyspnoea 9 (4.5%), pain 37 (18.3%), headache 11 (5.4%), hypotension 5 (2.5%), gastrointestinal effects 24 (11.9%), visual effects 4 (2.0%), insomnia 32 (15.8%), tinnitus 3 (1.5%), paraesthesia (18) (8.9%), itching (10) (5.0%), and other side effects 27 (13.4%).

Drug tolerability could not be inferred solely from the documented side effects because some might be an extension of the signs and symptoms of chronic heart failure. Clinical laboratory tests and drug interaction should be properly monitored to ascertain rational pharmaco-therapy in the management of chronic heart failure. The findings from this study will positively enhance the long-term management of patients with chronic heart failure in this environment.

Key Words: Chronic heart failure, Patients, Management, Pharmacotherapy, Side effects.

INTRODUCTION

Chronic heart failure, (CHF) can be defined as progressive complex clinical syndrome characterized by dyspnea, fatigue and fluid retention[1]. Over the course of the syndrome, patients may not always have symptoms of congestion. Therefore, the term "congestive heart failure" is beginning to fall out of favor; rather, "chronic heart failure" is being used to describe this population[2]. It occurs when the heart is incapable of maintaining sufficient blood flow to accommodate tissue perfusion and metabolic requirements. Forty to fifty percent of patients with symptoms of heart failure may have preserved systolic function[3]. These patients are more likely to have hypertension, left ventricular hypertrophy (LVH), and isolated diastolic dysfunction (IDD) [4, 5].

The final stage in almost any type of heart disease is chronic heart failure (CHF) in which the heart muscle weakens and is unable to pump enough blood to the body [6]. In the early stages of CHF, the muscle may enlarge in an attempt to contract more vigorously, but after a time, this enlargement of the muscle simply makes the heart inefficient and unable to deliver enough blood to the tissues [6]. In response to this shortfall, the kidneys conserve water in an attempt to increase blood volume and the heart is stimulated to pump harder. Eventually, excess fluid seeps through the walls of tiny blood vessels and into the tissues [7]. Fluid may collect in the lungs, making breathing difficult, especially when patient is lying down at night. Many patients with CHF find it very difficult to do physical activity[8].

Almost any condition that overworks or damages the heart muscle can eventually result in CHF[6]. The most common cause of CHF is coronary heart disease [8]. It may develop when the death of heart muscle in a heart attack leaves the heart with less strength to pump blood, or simply as a result of long-term oxygen deprivation due to narrowed coronary arteries[9]. Hypertension or malfunctioning valves that force the heart to work harder

over extended periods of time may also lead to CHF[9]. Viral or bacterial infections, alcohol abuse, and certain chemicals including some lifesaving drugs used in cancer chemotherapy can all damage the heart muscle and result in CHF[10].

Despite its ominous name, CHF can sometimes be reversed and can often be effectively treated for long periods with a combination of drugs[11]. Addressing possible underlying causes of CHF is the first step in the management of the disease. Non-drug interventions are important, including dietary changes such as reducing salt and alcohol intake and exercise and smoking cessation[10]. The principal aims of drug therapy are to reduce mortality, control symptoms, prevent hospital admissions, delay disease progression and minimize the adverse effects of therapy[11]. Medications such as Digitalis are often prescribed to increase the heart's pumping efficiency, while B-blockers may be used to decrease the heart's workload. Drugs known as vasodilators relax the arteries and veins so that blood encounters less resistance as it flows. Diuretics stimulate the kidneys to excrete excess fluid[12,13].

A last resort in the treatment of CHF is heart transplantation, in which a patient's diseased heart is replaced with a healthy heart from a person who has died of other causes[14]. The purpose of this study was to assess the rational pharmaco-therapeutic approach in the management of chronic heart failure (CHF) at the University College Hospital Ibadan in Nigeria during the period of 1995 to 2005 with the goal of providing and enhancing pharmaceutical care.

PATIENTS AND METHODS

A ten-year retrospective cohort review of the 202 case notes from the Medical Records Department of the University College Hospital, Ibadan, in Southwestern Nigeria was made.

The case notes reviewed were randomly selected. Each patient's case note which outturned the patient's medical profile was reviewed. The study populations of 202 patients were outpatients and inpatients at the Cardiology Unit who had continuously registered at the University College Hospital, Ibadan between August 1995 and August 2005.

The New York heart association (NYHA) definition was used to define chronic heart failure [1].

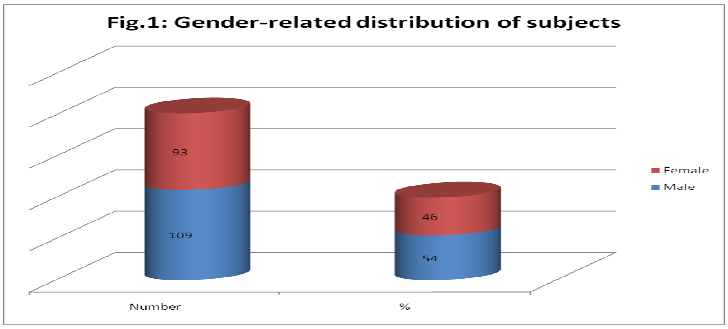
The past medical history of the subjects was used to elicit the presence of prior myocardial infarction, hypertension, valvular disease, kidney disease, liver disease and other co-morbidities. The drug management was

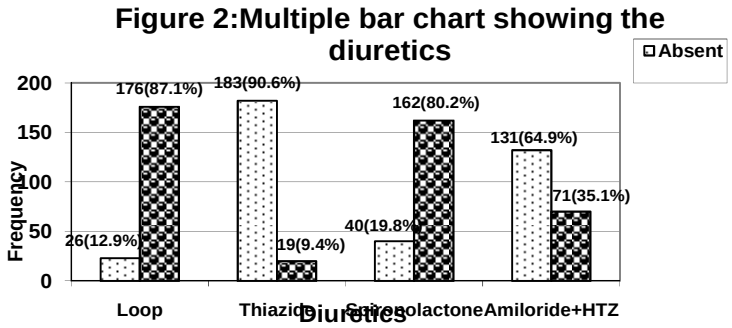
reviewed by noting the types of drugs prescribed, documentation of lifestyle modification such as restricted diet and exercise programs. Demographics of subjects (age and sex) were collected. The range of the reported adverse reactions was used as a measure of the tolerability to the regimen. Statistical analysis was done using the SPSS version 11.0 software programme for frequency distribution and cross-tabulations. Test for statistical significance included chi-square for categorical data and bivariate regression to model predictor-criterion relationships.

Permission to conduct the study was given by the authorities of the University College Hospital (U.C.H.) Ibadan, Nigeria. Ethical approval was granted based on the understanding that the study has the potential to improve the overall pharmaceutical care received by the chronic heart failure patients at the University College Hospital Ibadan in particular and Nigeria in general.

RESULTS

Two hundred and two case notes of patients diagnosed to have CHF were reviewed. One hundred and nine (54%) were males while ninety three (46%) were females as shown in Figure 1. The mean age was 56.01 years, (S.D$\pm$14.74). Of the two hundred and two subjects 34.7% of the patients had diabetes mellitus, 37.6% were hypertensive, 5% had vascular disease, 8.9% had kidney disease, 5.9% had liver disease, 6.4% had myocardial infarction and 4.5% had corpulmonale. Analysis of the medication subjects were on showed that; 87.1% were on loop diuretic, furosemide, with the most common dosage being between 40 – 80mg daily, 19% were on thiazide and hydrochlorothiazide (25mg twice daily), 80.2% on aldosterone antagonist and spironolactone (25mg twice daily), 35.1% on potassium-sparing diuretic, amiloride combined with hydrochlorothiazide (1 tablet daily). Fig. 2 shows the number of patients who were on loop diuretics to be 176 (87.1%) while patients who were on thiazide diuretics, spironolactone and amiloride + HTZ were 19 (9.4%), 162 (80.2%) and 71 (35.1%) respectively. The number of patients who were not on loop diuretics 26 (12.9%), thiazide diuretics 183 (90.6%), spironolactone 40 (19.8%) and amiloride +HTZ 131 (64.9%) is also shown in fig. 2. At one point or the other, 16.3% of the patients were placed on digoxin while 6.4% of the patients were prescribed captopril, 54% on lisinopril, 20.8% on enalapril, 13.9% on ramipril and 9.4% on enalapril and hydrochlorothiazide.





Additional therapies used included antibiotics (69.3%), antiplatelets (acetylsalicylic acid) (63.4%), and anticoagulant (8.4%) while 29.7% of the patients were on anti-hypertensive. The calcium channel blockers were given to 33.7% of the patients, methyldopa to 22.8%, beta blocker to 0.99%, oral hypoglycaemic agent (OHA) to 6%, non steroidal anti inflammatory drugs (NSAIDs) to 18.3% and chlorpheniramine maleate, diazepam and antacids to 73.3%.

Side effects recorded included; cough (15.8%), dyspnoea (4.5%), arrhythmia (0%), pain (18.3%), headache (5.4%), hypotension (2.5%), gastro-intestinal effects which includes abdominal pain, nausea, vomiting, loss of appetite (11.9%), blurred vision (2.0%), insomnia (15.8%), tinnitus (1.5%), paraesthesia (8.9%), itching (5%) and other effects such as irrational behavior, palpitation, tiredness, somnambulism, fatigue and tremor were (13.4%). A list of observed side effects is shown in table 4. The regression of the ACEI on cough as side effect was not significant (P>0.05) as analyzed from the data obtained as shown in table 5. Similarly the regression of the loop diuretics on tinnitus as a side effect was not significant (P.0.05) as shown in table 6 and the regression of enalapril on liver disease as a side effect was not significant (P>0.05) as analyzed from the data obtained in table 7.

Table-4: Side effects documented among subjects with CHF.

Side effect	*Frequency	Percentage (%)
Cough	32	15.8
Dyspnoea	9	4.5
Arrhythmia	0	0
Pain	37	18.3
Headache	11	5.4
Hypotension	5	2.5
GI effect	24	11.9
Visual effect	4	2
Insomnia	32	15.8
Tinnitus	3	1.5
Paraesthesia	18	8.9
Itching	10	5.0
Other effects	27	13.4

*Multiple responses

Table-5: Regression of the ACEIs on cough Model summary.

Model	R	R Square	Adjusted R Square	Std. error of the Estimate
1	0.185[a]	0.034	0.009	0.36431

a. Predictors: (Constant): Enalapril + Hydrochlorothiazide, Ramipril, Captopril, Enalapril, Lisinopril

ANOVA[b]

Model	Sum of Squares	df	Mean square	F	Level of Significance
1 Regression	0.917	5	0.183	1.382	0.233[a]
Residual	26.014	196	0.133		
Total	931	201			

a. Predictors: (Constant): Enalapril + Hydrochlorothiazide, Ramipril, Captopril, Enalapril, Lisinopril

b. Dependent variable: Cough

Table-6: Regression of loop diuretic on tinnitus Model summary.

Model	R	R Square	Adjusted R Square	Std. error of the Estimate
1	0.047[a]	0.002	-0.003	0.12143

a Predictors: (Constant): Loop diuretic

ANOVA[b]

Model	Sum of Squares	df	Mean square	F	Sig
Regression	0.007	1	0.007	0.446	0.505[a]
Residual	2.949	200	0.015		
Total	2.955	201			

a Predictors: (Constant): Loop diuretic
b Dependent variable: Tinnitus

Table-7: Cross tabulation of liver disease and Enalapril Chi-Square tests.

	Value	df	Asymp. Sig (2-sided)	Exact Sign. (2-sided)	Exact Sig. (1-sided)
Pearson chi-square	3.376[b]	1	0.066		
Continuity correction[a]	2.163	1	0.141		
Likelihood ratio	2.872	1	0.090		
Fisher's exact test				0.133	0.077
Linear-by-linear asso	3.359	1	0.067		
No of valid cases	202	1			

a. Computed only for a 2x2 table
b. 1 cells (25.0%) have expected count less than 5. The minimu expected count is 2.50

DISCUSSION

Proper management of CHF should include attempts to modify or correct systemic diseases causing or precipitating CHF in approximately 90% of patients[15]. Coronary artery disease, (CAD), is the cause of CHF in approximately two-thirds of CHF patients in the United States[15] CAD, if present, may impair ventricular function that may be secondary to myocardial infarction. Among the cardiovascular co-morbidities reviewed in this study, it is clear that hypertension (37.6%) predisposed patients more to CHF. In addition to hypertension other cardiovascular co-morbidities reviewed were valvular disease (5%) and acute myocardial infarction (3.5%) as shown in table 1.

Table-1: Co-morbidities associated with chronic heart failure.

Co-morbidity	Number	Percentage (%)
Diabetes Mellitus	70	34.7
Hypertension	76	37.6
Valvular disease	10	5
Renal disease	18	8.9
Liver disease	12	5.9
Acute myocardial Infarction	7	3.5
Corpulmonale	9	4.5
Total	202	100

Hypertension, valvular disease and acute myocardial infarction had been established among the generalized pathophysiologic conditions that lead to CHF. Hypertension causes CHF due to pressure overload of the heart, while acute myocardial infarction leads to CHF due to loss of functional myocardial tissue and valvular disease cause CHF due to volume overload of the heart[2]. Hypertensive patients may benefit from the same antihypertensive drugs used to treat CHF while patient with acute myocardial infarction may benefit from beta blockers and patient with valvular disease may benefit from attempt to surgical repair or replacement of defective myocardial valves[16]. In this study 202 case notes consisting of 54% males and 46% females were

reviewed. The random selection of case notes is supported by a study carried out in 2002 in the United States of America where the prevalence of CHD was estimated to be 4.9 million with nearly equal number of men and women [17]. The Kidney is one of the target organs that are complicated in arterial hypertension. Untreated hypertension resulting from renal complication can result in CHF[2]. Diabetes mellitus is a major cardiovascular risk factor in hypertension that complicates CHF[2]. In this study, renal disease 8.9% and diabetes mellitus 34.7% were

co morbidities. The influence of some drugs such as captopril, lisinopril, ramipril and enalapril, which are angiotensin converting enzyme inhibitors (ACEI) on the kidney function, could be quite significant. Proper electrolyte and urea concentrations (E&U) tests should be carried out to monitor the kidney function. Ideally, E&U test should be done before and during treatment[16]. Reduction in renal blood flow and glomerular filtration rate (GFR) are reflected by increases in both serum creatinine concentration and blood urea nitrogen (BUN) level. In this study, it was noted that the test, when carried out, was recorded once, most times before commencing the treatment which made it difficult to determine to what extent the kidney was compromised prior to therapy as well as determine if the observed kidney problem was as a result of drug use or deteriorating co-morbidity.

Numerous laboratory abnormalities are observed in patients with CHF. Simple laboratory abnormality tests such as serum electrolytes, serum creatinine and blood urea nitrogen, body weight and chest radiographs are used most frequently in monitoring ambulatory patients with CHF. The patient can monitor body weight at home every day and serum electrolytes are checked about every 1 to 2 months provided that there are no changes in therapy.

In this study, the conventional treatment for CHF was a combination of a diuretic most especially a loop and a potassium-sparing diuretics. Loop diuretics was 87.1%, thiazide 9.4%, spironolactone, an aldosterone antagonists 80.2% while, 35.1% were prescribed amiloride and hydrochlorothiazide, 6.4% were prescribed captopril, 54% lisinopril, 20.8% enalapril, 13.9% ramipril and 9.4% enalapril and HTZ as a fixed dose as shown in table 2.

Table-2: Anti hypertensives and digoxin prescribed.

Drugs	*Frequency	Percentage (%)
Loop diuretics	176	87.1
Thiazide diuretics	19	9.4
Spironolactone	162	80.2
Amiloride + HTZ	71	35.1
Captopril	13	6.4
Lisinopril	109	54
Enalapril	42	20.8
Ramipril	28	13.9
Enalapril + HTZ	19	9.4
Calcium channel blockers	68	33.7
Beta blockers	16	7.9
Methyldopa	46	22.8
Digoxin	33	16.3

*Multiple responses

As the understanding of the pathophysiology of CHF has developed, there have been a number of major advances in its pharmacotherapy. The evidence-based drug used in improving CHF includes ACEIs, beta-blockers and spironolactone[18]. ACEIs have been described as the cornerstone of CHF management and have been shown to significantly reduce mortality, and morbidity. Based on several studies, ACEIs are to be considered for all patients with symptoms of CHF that result from left ventricular dysfunction.

In this study, a good number of the patients diagnosed with CHF were prescribed ACEI. This is in line with the current guideline recommending the use of ACEI as first line therapy [2].

Products incorporating an ACEI with thiazide diuretics are now available. (for example Enalapril and HTZ). In this study 9.4% of such was on ACEI with thiazide diuretics. The use of this combination product should be reserved for patient whose blood pressure has not responded to thiazide diuretic or an ACEI alone.

Despite publication of data as early as 1979 to support the use of beta-blockers, many clinicians have been reluctant to use them in the management of CHF. As observed also in this study, 7.9% patients were prescribed beta-blocker, such as atenolol. Large-scale studies were however published in the late 1990s demonstrating significant reduction in mortality during treatment with beta-blockers. For example, in their second report, the Cardiac Insufficiency Bisoprolol study II (CIBIS-II) randomized 2,647 patients to receive either bisoprolol or placebo in combination with standard care ACEI with or without diuretic and/or digoxin. Bisoprolol treatment was associated with 34% reduction in mortality, a 32% reduction in hospital admission and significant improvements in symptom control. These benefits were evident across all the subgroups assessed including people with diabetes, the elderly and those with renal dysfunction[19].

Most of previously published reports have studied the benefit of beta-blockers in patients with New York Heart Association (NYHA) class II or III, heart failure. There has been some controversy over the benefit and risk associated with treating patients with more severe symptomatic NYHA class IV. This subgroup of patients was investigated in the Copernicus trial, which reported an overall reduction in mortality of 35% with carvedilol treatment compared to placebo[20].

Copernicus indicated that this agent could be prescribed safely to patients with an EF<25% with 74% achieving target dose and the majority reporting significantly improved symptoms. A 23% reduction in mortality was reported in Copernicus study looking at Carvedilol therapy in patient with left ventricular dysfunction, post-myocardial infarction (PMI), indicating that the beta-blockers are safe and effective in PMI[21].

B-blockers should therefore be considered for all patients with NYHA class II, III or IV heart failure. This agent should be initiated when patients are clinically stable and optimized on first line therapy primarily by ACEI and diuretic. B-blocker therapy must not be initiated during the acute phase of CHF management, but may be considered in the hospital environment when symptoms of fluid overload have been adequately addressed[18].

B-blockers are contra-indicated in patients with severe bradycardia, acute CHF, severe asthma or bronchospasm and peripheral vascular disease. Although the Committee of Medicines has warned against the use of B-blockers in patients with a history of asthma or bronchospasm, a recent Cochrane Collaboration Review has concluded that there is no strong evidence to support withholding cardio selective b-blockers from patients with mild to moderate reversible airway disease[1,2].

Spironolactone therapy is indicated for the treatment of NYHA class IV heart failure or class III heart failure where there has been a recent class IV episode and it should be added to optimized ACEI therapy. There is, however, little data on the use of ACEI, beta-blocker and spironolactone combinations currently. In this study, 80.2% patients were prescribed spironolactone as shown in table 2. Spironolactone is contraindicated in hyperkalaemia, hyponatraemia and Addison's disease. Again the importance of carrying out the electrolyte level test is emphasized. Renal function and serum potassium should be monitored carefully throughout therapy.

Oral digoxin is clearly indicated in patients with atrial fibrillation and CHF for the control of ventricular rate. Although there is no overall mortality benefit conferred by digoxin therapy in patient with normal sinus rhythm; trial data indicate improved symptom control and reduced hospital admission, at the expense of an increase in cardiac arrhythmia[22]. Also, the currently limited information on the use of digoxin in CHF patients does not justify its routine use in this setting. Digoxin may therefore be considered for patients who remain symptomatic despite optimized doses of diuretic, ACEIs, beta-blockers and spironolactone[23,24]. In this study 16.3% of the patients were on digoxin, (Table 2). Majority were given for a short period and discontinued.

In CHF, the major defect is a decrease in cardiac output, which results in poor tissue perfusion and stable blood pressure. Neurohormones mainly norepinephrine and angiotensin II by their vasoconstricting effects increase peripheral resistance, decrease cardiac output and heart rate. This is the pathological basis and focal point in the use of vasodilators in CHF. Vasodilator therapy shows new promise in CHF treatment. These agents through with varied mechanisms have been shown to alter the capacitance (preload) and resistance (after load) of vessels, either directly or indirectly[25]. These include the calcium channel blockers such as nifedipine, verapamil, diltiazem and amlodipine.

Nifedipine, amlodipine, verapamil and diltiazem have some degree of negative isotropic effect which could worsen the left ventricular function in CHF. Amilodipine is a potent peripheral vasodilator with minimal effect on heart rate. Nifedipine is the most studied of this class of drugs and is indicated in CHF with dilated cardiomyopathy. In this study, amlodopine a calcium channel blocker was prescribed more often (33.7%). Calcium antagonists are useful alternatives in some patients that may have CHF alone or in addition to other cardiovascular diseases such as angina pectoris, arrhythmias, cardiomyopathy and hypertension[26]. Since CHF may be worsened by the use of some of these calcium antagonists, patient selection, gradual administration of the drugs and close monitoring should be the guide in the use of these drugs in patients with CHF.

Non-steroidal anti-inflammatory drugs, (NSAIDs), inhibit cyclo-oxygenase activity, leading to a reduction in vasodilatory prostaglandins, which oppose the renal and systemic effects of all the patients with CHF[27.] Administering NSAIDs to patients with CHF produces a reduction in glomerular filteration rate (GFR) and renal blood flow and an increase in sodium and water retention. Chronic use of NSAIDs in older adults with CHF is associated with increased hospitalization rates secondary to acute CHF decompensation[28]. In this study, 18.3% were prescribed NSAIDs as shown in table 3. Improved education about the possible detrimental effects of chronic use of NSAIDs in patients with CHF is important. There is the need to pay more attention to these precipitating factors which could significantly reduce the number of hospitalizations and ease the clinical and economic burden of CHF on patients.

Table-3: Concomitant drugs prescribed.

Drugs	*Frequency	Percentage (%)
Antibiotics	140	69.3
Antiplatelets (ASA 75mg-150mg)	128	63.4
Anticoagulant	17	8.4
Anti hypertensive	60	29.7
Oral hypoglycemic agents	6	3.0
NSAIDs	37	18.3
Others	148	73.3

*Multiple responses

The tolerability of the drugs could not be significantly measured from this study because most of the time they were not documented. A clear distinction could not be made between side effects which were an extension of the signs and symptoms of the diseases conditions or as a result of the drugs as shown in table 4. The regression of the ACEIs on cough was not significant (P>0.05) as analyzed from the data obtained as shown in table 5. Similarly, the regression of the loop diuretic on tinnitus as a side effect was not significant (P>0.05) as shown in table 6. Although not statistically significant it could however be said that the drugs were clinically tolerated by the patients. It is imperative to know the co-morbidities associated with hypertension, being the major risk factor of CHF, so as to rationalize the choice of drug to be prescribed. In this study, it was noted that 41.7% of the patients had liver disease and were prescribed enalapril, a prodrug that has to be metabolized by the liver to the active angiotensin converting enzyme (ACE). The regression of the enalapril on liver disease was however not significant (P>0.05) as analyzed from the data obtained in table 7. The influence of the medical representatives of pharmaceutical industries on the prescribers was so obvious that too frequent switching from one brand of a particular class of drug product to other classes of drug products was too prominent. It s important however that such switching be based on potential clinical advantage as well as positive effect of the new drug on the quality of life of the patient.

CONCLUSION

The results of the present work show that drug tolerability could not be inferred solely from the documented side effects because some might be an extension of the signs and symptoms of chronic heart failure. Clinical laboratory tests and drug interaction should be properly monitored to ascertain rational pharmaco-therapy in the management of chronic heart failure. These findings will optimize the treatment of patients with chronic heart failure.

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