Drugs utilization and therapy outcome in patients with heart failure, Aden 2013

Ganna Mohammed Hussein[1] and Samira Abdulla Mahmood [2]
[1]Department of Medicine, Faculty of Medicine and Health Sciences, Aden University
[2]Department of Pharmacology and Toxicology, Faculty of Pharmacy, Aden University
DOI: https://doi.org/10.47372/uajnas.2015.n2.a16

Abstract

The study evaluated drug utilization in heart failure therapy among inpatients at Al-Gamhuoria Teaching Hospital, Aden, in 2013. This descriptive study was carried out among heart failure patients who were admitted at Al-Gamhoria Teaching Hospital, Aden, during the period April 30th to July 30th, 2013. After obtaining the verbal informed consent, the interviews have been done using a structured data collection form. It covered the patient's general characteristics such as age, gender, marital status, education level, residential area and income, characteristics about medical history(HF symptoms, type of the concomitant disease under treatment) and characteristics about medication history (type of drugs, number of medications, dose, frequency and side effects). The medical charts have been checked to verify the data. The study has included 40 heart failure patients; 33 (82,5%) of them were males and 7 (17.5 %) females. The mean age of the patients was 61.5 ± 10.3 (range 45 - 80) years. More than 79% (23/29) of the participants had low educational level and most of the participants came from Aden 67.5% followed by Abyan 22.5% .All admitted study patients (100%) showed the NYHA functional classical signs and symptoms of heart failure, including shortness of breath (dyspnea;87.5 %), Fatigue (57.5%), and peripheral edema (72.5%). Almost half of the study HF patients (47.5%) were classified as NYHA functional class ш The mean age increases from 61 ± 4.5 in NYHA functional class II to 68 ± 11.3 years in NYHA functional class IV. 31.6 % of the patients (12/38) had more than 100 beats per minute. Treatment of the study sample revealed 62.5% improvement, while one third of the participant (37.5) did not show improvement. Iischemia, non-ischemic heart disease or both made 7.5%, 52.5% and 37.5% of the patients, respectively. The preponderance was for hypertension and diabetes mellitus. 36.1 % of the total main drugs classes for HF treatment were diuretics, including aldosterone antagonist, followed by drugs blocking renin angiotensin system (RAS, 28.7%). Beta-blockers including carvedilol and bisoprolol made only 5.7% of the total drugs used. Diuretics and drugs blocking renin angiotensin system are the most used, but limited practice of carvedilol and bisoprolol. Hypertension and diabetes are the major comorbidities among the patients

Key words: Carvedilol, Heart failure, Diuretics, drugs blocking renin angiotensin system

Introduction

Heart failure (HF) is a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood. The cardinal manifestations of HF are dyspnea and fatigue, which may limit exercise tolerance, and fluid retention, which may lead to pulmonary and peripheral edema. [11] Both abnormalities can impair the functional capacity and quality of life of the affected individuals.

The goals of therapy in patients with heart failure have changed over time and vary with the severity and etiology of the syndrome. [12, 18] Drugs, originally, have been approved based on their ability to improve resting hemodynamics in the short term.

During the past 20 years, considerable growing knowledge in terms of pathophysiological mechanisms for the development and progression of the heart failure syndrome has been emerged. Thereafter, a number of therapeutic approaches have been proposed for the long-term management of chronic heart failure. It has been sufficiently documented that clinical heart failure resulting from impaired left ventricular systolic function is associated with a substantial mortality and

morbidity and that the more severe the clinical heart failure is, the worse the prognosis. To improve prognosis, it is essential to continue the search for newer therapies.

Drug therapies have focused on the endpoint components of this syndrome, volume overload (congestion) and myocardial dysfunction. [10]

Use of diuretics and cardiac glycosides that improved contractile performance, such therapies, have not been proven to improve survival. Then, greater insight into the induction and propagation of HF led to a conceptual framework in which heart failure is viewed as a consequence of disordered circulatory dynamics, and pathologic cardiac remodeling have come out. These developments have had a major positive impact on the treatment of HF.

Thus, the 2009 American College of Cardiology/American Heart Association (ACC/AHA) updated guidelines [13, 30] , 2010 Heart Failure Society of America (HFSA) guidelines [16, 18] ,and the 2008 European Society of Cardiology (ESC)guidelines [6, 21], with varying levels of evidence, have recommend the following: *Angiotensin-convertingenzyme inhibitors*(ACEIs) and *Angiotensin receptor blockers*(ARBs) are used for neurohormonal modification, vasodilatation, improvement in LVEF, and survival benefit, *Hydralazine* and *nitrates* to improve symptoms, ventricular function, exercise capacity, and survival in patients who cannot tolerate an ACEI/ARB or as an add-on therapy to ACEI/ARB and beta-blockers in the black population for survival benefit, *Beta-adrenergic blockers* [25] for neurohormonal modification, improvement in symptoms and LVEF, survival benefit, arrhythmia prevention, and control of ventricular rate, *Aldosterone antagonists*, as an adjunct to other drugs for improved LVEF, and increase in survival, *Anticoagulants* to decrease the risk of thromboembolism, and *Inotropic agents* to restore organ perfusion and reduce congestion in decompensation. [21]

Assessment of the therapy is essential to lessen the burden on the patient activities. On the other hand, such evaluation might be helpful in revealing the faults and the factors that hinder the positive outcome. Several studies have evaluated the way of managing patients with HF as well as assessing the therapy outcomes. [7]

As far as we know, limited studies address the drug utilization in patients with heart failure in Yemen, particularly in Aden. As many worldwide heart failure patients, Yemeni heart failure patients admitted at Al-Gamhoria Teaching Hospital, Aden, receive medical care and required therapy. To assess drug utilization and treatment outcome of those patients, this work has been designed. Results of this work might be of benefit in this field.

Methodology

Study design and population

This descriptive study was carried out among heart failure patients who were admitted at Al-Gamhoria Teaching Hospital, Aden, during the period April 30thto July 30th, 2013.After obtaining the verbal informed consent, well trained students interviewed the patients using a structured data collection form.

Inclusion criteria

Heart failure (HF) patients (males and females) admitted to the medical wards at Al-Gamhoria Teaching Hospital, Aden, were enrolled in the study

Exclusion criteria

Acute decompensated HF patients, HF patients with chronic or acute Renal failure, and HF patients who had incomplete data were excluded from the study

Procedure

Patients were interviewed after obtaining informed verbal consent and data were collected through a questionnaire. The questionnaire covered the patient's general characteristics such as age, gender, marital status, education level, residential area and income, characteristics about medical history(HF symptoms/ signs, type of the concomitant disease under treatment) and characteristics about medication history (type of drugs, number of medications, dose, frequency and side effects). The interviewer checked the medical chart to verify the data.

Statistics

Data analysis**:** Patients' data were analyzed according to variables in the questionnaire, using frequency distributions, mean and percentages for quantitative variables. Data processing were performed by using computer facilities (SPSS).

Ethical consideration

This research was performed to achieve a social benefit. The interview with patients was conducted only after obtaining verbal informed consent from each patient. Patients have been given detailed explanation about the study objectives, and confidentiality of the information. In addition, the information obtained was used for the research purpose only.

Results

Patients characteristics

Out of 40studied heart failure inpatients; 33 (82.5%) were males and 7 (17.5 %) were females. The mean age of the patients was 61.5 ± 10.3 (range 45 - 80) years. More than 79% (23/29)of the participants have lower educational level (secondary school level and illiterate). Regarding the marital status, 77.5% of the admitted HF patients were married. According to area of residency, the study revealed that most of the participants came from Aden, followed by Abyan. The majority of the patients had poorly paid income

Medical characteristics

To find out the medical characteristics of the participants, patients were thoroughly investigated by their treating physicians. All study HF patients (100%) showed the NYHA functional classical symptoms of heart failure; including breathlessness (dyspnea; 87.5 %), Fatigue (57.5%), and peripheral edema (72.5%) (Figure 1). 82.5% of the patients experienced two or more of the classic symptoms. A proportion of patients with NYHA classes I, II, III and IV were 7.5%, 10.0%, 47.5% and 35.0%, respectively. Almost half of the study HF patients (47.5%) were classified as NYHA functional classes III (Figure 2).

The mean age increases from **61 ± 4.5** in NYHA functional **class II** to **68 ± 11.3** years in NYHA functional **class IV**. **31.6 %** of the patients **(12 /38)** showed heart rate with more than **100** beats per minute.

To find out the outcome of therapy, the patients have been clinically assessed and reevaluated. The treatment of the study sample revealed **62.5%** improvement, while one third of the participants showed poor improvement, (Table 1). For searching for the concomitant disorders, the study showed **7.5%, 52.5%** and **37.5%** of the patients had ischemic , non-ischemic diseases or both, respectively, (Table 2). The preponderance was for hypertension and diabetes mellitus.

Analyzing the total utilized drugs by the HF patients has revealed that 36.1 % of the total main drug classes prescribed for HF patients were diuretics including aldosterone antagonist and followed by drugs blocking renin angiotensin system (RAS, 28.7%). Beta-blockers made only 5.7% of the total drugs prescribed (Table 3). On the other hand, 34 patients (85%) were given drugs blocking RAS, while 15% of the HF patients were utilized carvedilol and bisoprolol (beta-blockers) (Table 4).

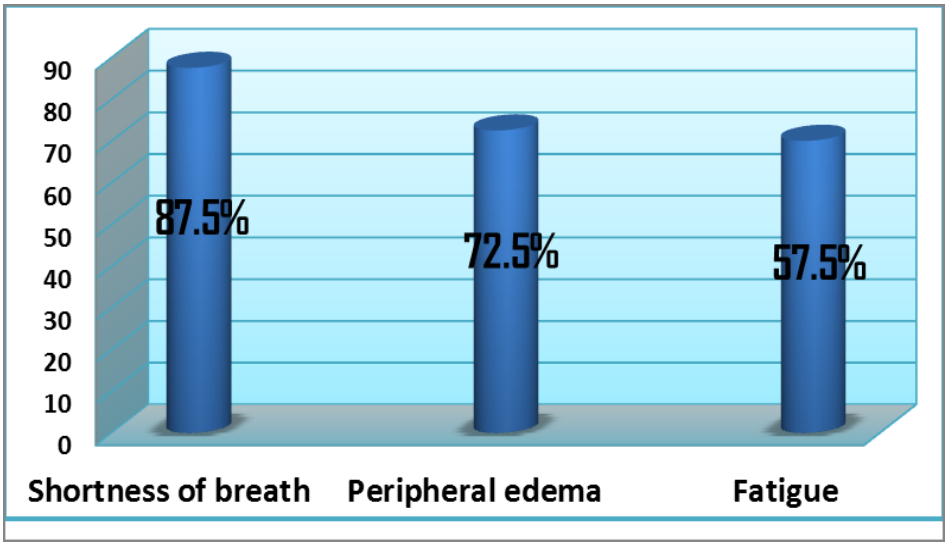

Figure 1: Frequency of individual signs and symptoms of study heart failure patients

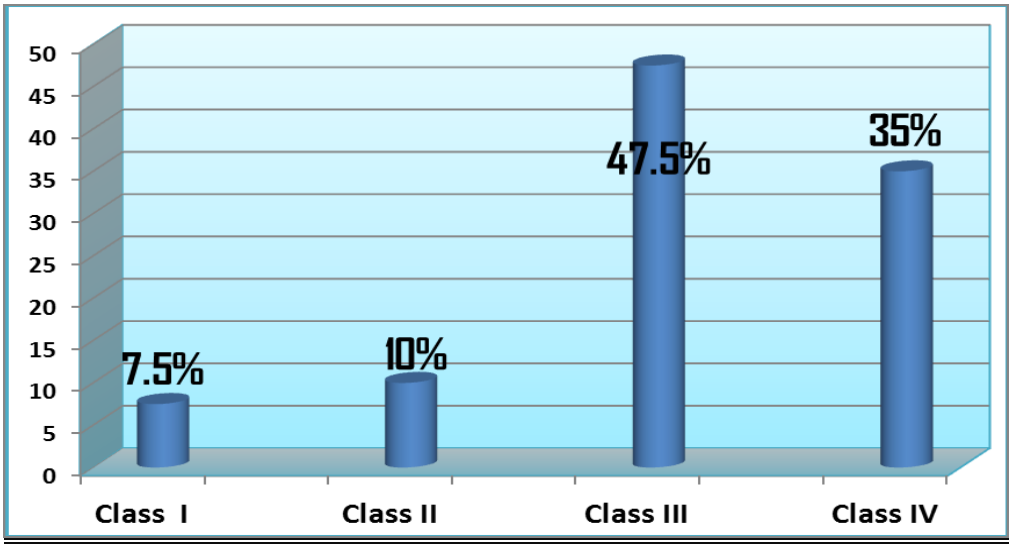

Figure 2: Classes of HF patients according NYHA functional classification
NYHA=New York Heart Association

Table 1: Treatment outcome of study HF patients

Therapy outcome	Frequency n-=40	Percentage %
Improved	**25**	**62.5**
Poorly improved	**15**	**37.5**

Table 2: Distribution of concomitant diseases of study heart failure patients

Concomitant diseases	Frequency n-=40	Percentage %
Ischemic	**03**	**07.5**
Non-ischemic	**21**	**52.5**
Both	**15**	**37.5**
Unidentified	**01**	**02.5**

Table 3: Main drug classes utilized by HF patients in relation to total drugs prescribed

Main Drug Classes	**NO**	**%**
Renin Angiotensin Aldosterone system n=35		**28.7**
Angiotensin converting enzyme inhibitors		
lisinopril	23	**17.8**
captopril	7	**05.4**
ramipril	2	**01.6**
enalapril	2	**01.6**
Angiotensin receptor blocker		
losartan	1	**00.7**
Beta Blockers n=07		**05.7**
Carvidillol	3	**05.4**
Bisoprolol	3	**04.7**
Propranolol	1	**00.7**
Diuretics n=44		**36.1**
Furosemide	22	**17.1**
Torsemide	4	**03.1**
Spironolactone	18	**13.**9
Cardiotonic n=16		**13.1**
Digoxin	16	**12.4**
Vasodilator n=20		**16.4**
Nitroglycerin	05	**03.9**
Isosorbidmononitrate	10	**07.**7
Amlodipine	05	**03.9**
Total	**122**	**100**

Table 4: Distribution of ACE-Inhibitors and beta-blockers in relation to study sample

Drug Class	**Patients No n=40**	**%**
ACE-Inhibitors	**34**	**85.0**
lisinopril *(cepril)*	**23**	**57.5**
captopril*(capoten)*	**07**	**17.5**
ramipril	**02**	**05.0**
enalapril	**02**	**05.0**
Beta-Blockers	**06**	**15.0**
Carvedilol	**03**	**07.5**
Bisoprolol	**03**	**07.5**

Discussion

The data of this study revealed that **62.5%** of the studied HF patients were improved and **87.5%** of the patients have used drugs blocking renin angiotensin aldosterone system, but only 32.5% of the total patients used beta-blockers (carvedilol and bisoprolol). This finding is dissimilar to study

by Komajda et al [17] in which treatment of HF was under-utilized. However, data about the effect of Heart failure (HF) disease management programs on outcome have been inconsistent. [15]

Biologic changes, associated with age, may be an important factor underlying the association between increasing age and the prevalence of diseases. [19] It has been found that the prevalence of cardiovascular and non-cardiovascular comorbidities increases linearly with advancing age. [23] Concerning the study sample, males were more than women. The mean age was almost 60 years. This is similar to that reported by Albert et al [1] and lower than that found in Dahlstrom et al [5] in which the mean age was 79 years. The mean age increases from 61 ±4.5 in NYHA functional class I to 68 ±11.3 years in NYHA functional class IV. This tendency is equal to that reported by Dahlstrom et al. [5] Most of the participants came from Aden. The majority of the patients were married, had low income and low educational level.

All admitted studied patients had the NYHA based classical symptoms/signs of heart failure. Virtually, half of the patients were in class III. Because of the lack of (reliability) availability of certain clinical data, analysis of the diagnostic measure echocardiography was not possible.

A higher usage of RAAS blockade and a lower practice of BB was found in this study. These results are similar to the study of Dahlstrom et al [5] in which a much higher use (74%) of RAAS blockade was reported. The lower utilization of beta-blockers has been mentioned in many other studies [2,4,9 ,28,36] . A remote explanation for the lower use of beta-blockers might be the limited prevalence of ischemia among our study population

Hallmarks of congestive heart failure (CHF) are sodium and water retention, and the resulting symptoms are related to congestion. Management of the disease is directed to those parameters. The European Society of Cardiology guidelines for the management of CHF related to left ventricular (LV) systolic dysfunction *emphasize* in particular the *beneficia*l effects of angiotensin-converting enzyme-inhibitors (ACE-I), beta-blockers, angiotensin II receptor blockers (ARB), and aldosterone antagonists on mortality and morbidity, based on large outcome trials. [3, 8, 20] In spite of such advance, mortality and morbidity rate are still high. [29, 24]

Angiotensin-converting-enzyme (ACE) inhibitors reduce morbidity and mortality in patients with chronic heart failure and systolic left-ventricular dysfunction as well as in patients who have had a myocardial infarction. [26 , 33] The benefits of ACE inhibitors have been mostly attributed to blockade of angiotensin II production and/or to a *decrease in the breakdown of bradykinin*. Bradykinin has been shown to have beneficial effects associated with *the release* of *nitric oxide and prostacyclin*, which may contribute to the haemodynamic effects of ACE inhibition. Bradykinin may, however, also be responsible for some of the adverse reactions to ACE inhibitors; such as cough, angioedema, renal dysfunction, and hypotension, and these side effects may explain in part why ACE inhibitors are used in less than 30% of patients with heart failure despite the proven clinical benefit of these agents.[32] ACE inhibitors reduce the activity of sympathetic nervous system as angiotensin II promotes the release of noradrenaline and inhibits its re-uptake. Moreover, they improve β-receptor density (causing their up regulation) and function, variation in heart rate, and autonomic function (including vagal tone); so ACE inhibitors modify cardiac remodeling.

Orally active, non-peptide angiotensin II type 1 receptor antagonists, such as losartan, can block this receptor specifically without increasing bradykinin levels, and, since angiotensin II may be produced by alternate pathways [22, 34], such drugs may have additional advantages over ACE inhibitors where blockade of the effects of angiotensin II is incomplete. Losartan is licensed for the treatment of hypertension in many countries, and in earlier studies in patients with symptomatic heart failure, oral losartan produced beneficial haemodynamic effects both acutely and with chronic dosing. [33]

β-Adrenoceptor blockers have *been traditionally avoided* in patients with heart failure due to their *negative inotropic* effects. However, there is now considerable clinical evidence to support the use of β blockers in patients with **chronic stable** heart failure

resulting from left ventricular systolic dysfunction. Recent randomized controlled trials in patients with chronic heart failure have reported that combining β blockers with conventional treatment

with diuretics and angiotensin converting enzyme inhibitors results in *improvements* in left ventricular function, symptoms, and survival, as well as a *reduction in admissions* to hospital.[37]

Potential mechanisms and benefits of β blockers could be improved left ventricular function; reduced sympathetic tone; improved autonomic nervous system balance; up regulation of β adrenergic receptors; reduction in arrhythmias, ischemia, further infarction, myocardial fibrosis, and apoptosis. More than that, β blockers antagonize the activity and action of other neurohormonal system which are indirectly activated by sympathetic nervous system. [25]

Clinical-based evidence has also established the effectiveness of spironolactone or aldactone in HF management. [7] The use of potassium-sparing diuretics PSDs in HF patients is associated with a reduced risk of death from, or hospitalization for, progressive HF or all-cause or cardiovascular death, compared with patients taking only a non-PSD.[7] In 1999, the Randomized Aldactone Evaluation Study (RALES) demonstrated *a 30% decrease* in mortality for treating CHF patients with spironolactone [27] . The benefit was seen in patients already receiving background therapy with ACE inhibitors. Previously used in heart failure to promote diuresis, spironolactone was readily accepted as an inexpensive agent to treat severe systolic dysfunction. [14, 35] However, the potassium-sparing effects of spironolactone pose a great risk for retaining potassium and subsequent fatal arrhythmias, especially in patients taking other medications affecting the renin-angiotensin-aldosterone system, therefore, laboratory monitoring in patients treated with Spironolactone is recommended. [31]

In conclusion, the study showed a short term improvement of inpatients with heart failure. A proper utilization of angiotensin converting enzyme blocking drugs, but underutilization of beta-blockers, carvedilol and bisoprolol

References:

1. Albert N, Trochelman K, Li J, Lin S. (2010)Signs and Symptoms of Heart Failure: Are You Asking the Right Questions? **American Journal of Critical Care** 2010; 19 (5): 443-452
2. Ceia F, Fonseca C, Mota T, Morais H, Matias F, Costa C, Oliveira AG.(2004) Aetiology, comorbidity and drug therapy of chronic heart failure in the real world: the EPICA substudy. **Eur J Heart Fail** 6:801–806.
3.CIBIS-II investigators and Committees.(1999) The Cardiac Insufficiency Bisoprolol Study II (CIBIS-II): a randomised trial. **Lancet** 353:9–13.
4. Cleland JG, Cohen-Solal A, Aguilar JA, Dietz R, Eastaugh J, Follath F, Freemantle N, (2002) Improvement of Heart Failure Program Committees and Investigators. Improvement programme in evaluation and management; Study Group on Diagnosis of the Working Group on Heart Failure of The European Society of Cardiology. Management of heart failure in primary care (the IMPROVEMENT of heart failure program): an international survey. **Lancet** 360:1631–1639
5. Dahlstrom U, Hakansson J, Swedberg K, Waldenstrom A. (2009)Adequacy of diagnosis and treatment of chronic heart failure in primary health care in Sweden **European Journal of Heart Failure** 11, 92–98
6.Dickstein K, Cohen-Solal A, Filippatos G .(2008)Guideline for the Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2008 of the European Society of Cardiology. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2008: the Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2008 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association of the ESC (HFA) and endorsed by the European Society of Intensive Care Medicine (ESICM). **Eur Heart J**. 29(19):2388-442
7.Domanski M, Norman J, Pitt B, Haigney M, Hanlon S, Bethesda EP, Arbor A. (2003) Diuretic Use, Progressive Heart Failure, and Death in Patients in the Studies of Left Ventricular Dysfunction (SOLVD**) Journal of the American College of Cardiology** 42 (4):705-708.

8. Granger CB, McMurray JJ, Yusuf S, Held P, Michelson EL, Olofsson B, Ostergren J, Pfeffer MA, Swedberg K.(2003) Effects of candesartan in patients with chronic heart failure and reduced left-ventricular systolic function intolerant to angiotensin-converting-enzyme inhibitors: the CHARM-Alternative trial. **Lancet** 362:772–776.
9.Halling A, Berglund J. (2003)Diagnosis and treatment of heart failure in primary health care among elderly patients with non-insulin-dependent diabetes mellitus, with special reference to use of echocardiography. **Scand J Prim Health Care** 21:96–98
10. Harvey RA, (2009), Pharmacology, 4th edition, Lippincott Williams & Wilkins, **New Delhi**, 183-274.
11. Hess OM and Carroll JD. (2007)Clinical assessment of heart failure. In: Libby P, Bonow RO, Mann DL, Zipes DP, eds. Libby: Braunwald's Heart Disease: **A Textbook of Cardiovascular Medicine**. 8th ed. Saunders; chap 23.
12.Hildebrandt P. (2006)Systolic and non-systolic heart failure: equally serious threats. **JAMA.** 296(18):2259-60.
13.Hunt SA, Abraham WT, Chin MH, Xu AM. (2009) The American College of Cardiology Foundation; American Heart Association. 2009 Focused update incorporated into the ACC/AHA 2005 guidelines for the diagnosis and management of heart failure in adults: a report of the American College of Cardiology Foundation/American Heart Association Task Force on practice guidelines developed in collaboration with the International Society for Heart and Lung Transplantation. **J Am Coll Cardiol**. 53(15):e1-e90
14.Ikram H, Webster MW, Nicholls MG, Lewis GR, Richards AM, Crozier IG.(1986) Combined spironolactone and converting-enzyme inhibitor therapy for refractory heart failure**. Aust N Z J Med** 16:61–3.
15. Jaarsma T, van der Wal MH, Lesman-Leegte I, Luttik ML, Hogenhuis J, Veeger NJ, Sanderman R. (2008) Effect of Moderate or Intensive Disease Management Program on Outcome in Patients With Heart Failure Coordinating Study Evaluating Outcomes of Advising and Counseling in Heart Failure (COACH) **Arch Intern Med.** 168(3):316-324
16. Joann L, Nancy MA, Debra KM, John PB . (2010) Heart Failure Society of America (HFSA) practice guidelines. HFSA 2010 Comprehensive Heart Failure Practice Guideline. **J Card Fail** 5:357e82.
17. Komajda M, Hanon O, Hochadel M, Follath F, Swedberg K, Gitt A, Cleland JGF. (2007) Management of octogenarians hospitalized for heart failure in Euro Heart Failure Survey I **European Heart Journal** 28: 1310–1318
18. Lindenfeld J, Albert NM, Boehmer JP.(2010)Guideline for the Heart Failure Society of America. Executive summary: HFSA 2010 comprehensive heart failure practice guideline. **J Card Fail**. 16(6):e1-194
19. Masoudi FA, Havranek EP, Smith G, Fish RH, Steiner JF, Ordin DL, Krumholz HM. (2003)Gender, Age, and Heart Failure With Preserved Left Ventricular Systolic Function . Am Coll Cardiol 41:217–23
20. McMurray JJ, Ostergren J, Swedberg K, Granger CB, Held P, Michelson EL, Olofsson B, Yusuf S, Pfeffer MA. (2003) Effects of candesartan in patients with chronic heart failure and reduced left-ventricular systolic function taking angiotensin-converting-enzyme inhibitors: the CHARM-Added trial. **Lancet** 362:767–771.
21.McMurray JJV, Granger CB, Held P, Michelson EL, Olofsson B, Yusuf S, Pfeffer MA. (2012) ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. **European Heart Journal** 33;1787–1847
22.Miura S, Ideishi M, Sakai T. (1994)Angiotensin II formation by an alternative pathway during exercise in humans. **J Hypertension** 12**:** 1177–81.
23. Mogensen UM, Ersoll M, anrersson C, Hassager C, Torp-Pedersen C.(2011) Clinical characteristics and major comorbidities in heart failure patients more than 85 years of age compared with younger age groups. **European Journal of Heart Failure** 13: 1216–1223

24. Owan TE, Hodge DO, Herges RM, Jacobsen SJ, Roger VL, Redfield MM. (2006)Trends in prevalence and outcome of heart failure with preserved ejection fraction. **N Engl J Med** 355:251–259.
25. Packer M, Coats AJ, Fowler MB, Katus HA, Krum H, Mohacsi P, Rouleau JL, Tendera M, Castaigne A, Roecker EB, Schultz MK, DeMets DL. (2001) Carvedilol Prospective Randomized Cumulative Survival Study Group. Effect of carvedilol on survival in severe chronic heart failure. **N Engl J Med.** 344:1651–1658
26. Pfeffer MA, Braunwald E, Moye LA. on behalf of the SAVE Investigators. (1992) Effect of captopril on mortality and morbidity in patients with left ventricular dysfunction after myocardial infarction: results of the Survival and Ventricular Enlargement Trial. **N Engl J Med** 327**:** 669–77.
27.Pitt B, Zannad F, Remme WJ. (1999)The effect of spironolactone on morbidity and mortality in patients with severe heart failure. Randomized Aldactone Evaluation Study Investigators. **N Engl J Med** 341:709 –17.
28.Rutten FH, Grobee DE, Hoes AW.(2003) Differences between general practitioners and cardiologists in diagnosis and management of heart failure: a survey in every-day practice. **Eur J Heart Fail** 5:337–344.
29.Schaufelberger M, Swedberg K, Koster M, Rosen M, Rosengren A. (2004) Decreasing one-year mortality and hospitalization rates for heart failure in Sweden; data from the Swedish Hospital Discharge Registry 1988 to 2000. **Eur Heart J** 25:300–307.
30.Seharon Ann Hunt, William T. Abraham . (2009) Focused Update Incorporated Into the ACC/AHA 2005 Guidelines for the Diagnosis and Management of Heart Failure in Adults: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines **Circulation.** 119:e391-e479.
31. Shah KB, Rao K, Sawyer R, Gottlieb SS. (2005) The Adequacy of Laboratory Monitoring in Patients Treated With Spironolactone for Congestive Heart Failure Baltimore, **J Am Coll Cardiol** 46:845–9
32. Stafford RS, Saglam D, Blumenthal D. (1996) Low rates of angiotensin converting enzyme inhibitor use in congestive heart failure. **Circulation** 94**:** 1–194
33. The SOLVD Investigators.(1992) Effect of enalapril on mortality and the development of heart failure in asymptomatic patients with reduced left ventricular ejection fractions. **N Engl J Med** 327**:** 685–91.
34.Urata H, Strobel F, Ganten D. (1994) Widespread tissue distribution of human chymase. **J Hypertension** 12**:** S17–S22.
35. van Vliet AA, Donker AM, Nauta JP, Verheugt FW. (1993) Spironolactone in congestive heart failure refractory to high-dose loop diuretic and low-dose angiotensin-converting enzyme inhibitor. **Am J Cardiol** 71:21–8A.
36. Watz R, Ekstrand AB, Engelbrektsson V, Beermann B. (2005) Treatment with angiotensin II antagonists and beta-blockers in an unselected group of patients with chronic heart failure. **Eur J Clin Pharmacol** 61:209–214.
37. Willenheimer R, van Veldhuisen DJ, Silke B, Erdmann E, Follath F, Krum H .(2005) Effect on Survival and Hospitalization of Initiating Treatment for Chronic Heart Failure With Bisoprolol Followed by Enalapril, as Compared With the Opposite Sequence Results of the Randomized Cardiac Insufficiency Bisoprolol Study (CIBIS) III **Circulation** 112;2426-2435.

استعمال الأدوية وحصيلة علاج مرضى القصور القلبي، عدن 2013

جنة محمد حسين[1] و سميرة عبدالله محمود [2]
[1]قسم باطني كلية الطب والعلوم الصحية، جامعة عدن
[2]قسم علم الادوية والسموم، كلية الصيدلة، جامعة عدن
DOI: https://doi.org/10.47372/uajnas.2015.n2.a16

الملخص

تهدف هذه الدراسة إلى تقييم استعمال الأدوية بين المرضى المصابين بالقصور القلبي في هيئة مستشفى الجمهورية العام التعليمي، عدن، 2013.أجريت الدراسة الوصفية بين مرضى قصور القلب الذين أدخلوا مستشفى الجمهورية العام، عدن خلال الفترة 30 أبريل حتى 30يوليو، 2013. بعد الحصول على موافقة شفهية، تمت مقابلات المرضى وتسجيل البيانات في الاستبيان الذي غطي الخصائص العامة للمريض مثل السن والجنس، الحالة الزوجية، ومستوى التعليم، والمنطقة السكنية والدخل الشهري، الخصائص عن التاريخ الطبي (أعراض قصور القلب، نوع الأمراض المصاحبة تحت العلاج) والخصائص عن تاريخ الدواء (نوع الدواء، وعدد جرعات الأدوية والآثار الجانبية لها. تم التأكد من خطة العلاج الطبي للتحقق من البيانات. شملت الدراسة 40 مرضى قصور القلب ؛ 33 (82,5 ٪) منهم من الذكور و 7 من الإناث (17.5%). كان متوسط عمر المرضى 61.5 ± 10.3 (المدى45-80) سنة. أكثر من 79% (23من29) من المشاركين كان المستوى التعليمي لديهم متدني، وكان معظم المرضى من عدن 67.5% تليها أبين 22.5%. وأظهر جميع المرضى (100%) علامات وأعراض قصور القلب الكلاسيكية حسب تصنيف NYHA، بما في ذلك ضيق في التنفس (ضيق التنفس؛ 87.5 في المائة)، والتعب (57.5%)، واستسقاء طرفي (72.5%). ما يقرب من نصف مرضى الدراسة (47.5%) صنفوا في المستوى الثالث. لقد وجد أنَّ متوسط العمر يتزايد من 61 ± 4.5 في المستوى الثاني إلى 68 ± 11.3عاماً في المستوى الرابع لتصنيف NYHA. اظهرت الدراسة أن31.6% من المرضى (12من38) كان لديهم أكثر من 100 نبضة في الدقيقة. علاج عينة الدراسة كشف تحسن بنسبة62.5 في المائة، في حين أنَّ أكثر من ثلث المشاركين (37.5) لم يظهروا تحسناً وكانت نسب وجود الامراض المصاحبة ischemia, non-ischemic heart diseaseأو كليهما هي 7.5%، 52.5% و 37.5% على التوالي. وكانت السيادة لارتفاع ضغط الدم ومرض السكري. 36.1% من المجموعات الرئيسية لعلاج قصور القلب كانت مدرات البول بما في ذلك مضاد الالدوستيرون، تليها أدوية حظر نظام إنزيم الرينين انجيوتنسين (RAS، 28.7%). حاصرات بيتا (بما في ذلك كارفيديلول وبيسوبرولول) عملت فقط 5.7% من مجموع الأدوية المستخدمة. مدرات البول وأدوية حظرنظام إنزيم الرينين انجيوتنسين كانت الأكثر استخداماً لكن وجد استعمال محدود لكل من الكارفيديلول وبيسوبرولول. ارتفاع ضغط الدم والسكري من الأمراض الرئيسية المصاحبة لقصور القلب.

الكلمات مفتاحية: كارفيديلول، القصور القلبي، مدرات البول، أدوية حظر نظام الرينين انجيوتنسين.