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Research Article

Congestive Heart Failure with Atrial Fibrillation

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Abstract

Atrial fibrillation (AF) in patients with heart failure is common and associated with an increased risk of stroke, heart failure hospitalizations and all causes mortality. AF is a common sustained arrhythmia in HF and primary related to atrial dilation and remodeling. Heart failure is a common co-morbidity in diabetes and patients with both conditions have particularly poor prognosis. Most clinical outcome trials investigating the effect of glucose lowering agent have excluded patient with heart failure. Patients with heart failure are frequently affected with AF and interrelation between these pathologies is complex. AF share same risk factors as heart failure. The exact causes of atrial fibrillation in HF are incompletely understood but data suggest that heart failure induced atrial fibrosis and atrial ionic remodeling are underlying abnormalities that facilitate atrial fibrillation. Therapeutic considerations for atrial fibrillation in patients with heart failure include risk factors modifications and guidelines directed medical therapy, anticoagulation, rate control and rhythm control. Although anticoagulation therapy and rhythm or rate control are the foundation of atrial fibrillation therapy. Here we report a case of 66 years old female patient diagnosed as a congestive heart failure with atrial fibrillation with type-2 Diabetes mellitus. This article will present some practical tips for managing heart failure.

Key words: Heart Failure, Atrial Fibrillations, Angiotensin Converting Enzyme Inhibitors, Angiotensin Receptors Blockers, Beta Blockers.

1. Introduction

Heart failure (HF) is a clinical syndrome characterized by symptom and signs caused by structural or functional abnormalities of the heart. Typical symptoms are breathlessness are ankle swelling, and fatigue. Typical signs are increased jugular venous pressure, third heart sound, peripheral edema, and pulmonary crackles; however, the condition can be present in the absence of these findings. It is important to address the underlying cause of heart failure because the specific etiology determines the choice of treatment. Common causes of heart failure are ischemic heart disease, dilated cardiomyopathy, valvular lesions, hypertension, and atrial fibrillation. The toxic impact of chemotherapy and high levels of alcohol consumption can also lead to systolic left ventricular failure (1). Some data suggest that type 2 diabetes and hyperinsulinemia promote a diabetic cardiomyopathy (2). Atrial arrhythmia is significant contributors to morbidity and mortality in all patient ages' groups (3, 4). The interaction between HF and atrial fibrillation is complex and one can predispose, precipitate, or complicate the others (4, 5). The management of cardiovascular disease has undergone much change in recent years in general, notably recent advances in management of acute coronary syndromes have significantly reduced both short term and long-term mortality (6). This factor has led to increased survival, and thus, it could be argued, an increasing number of individuals with myocardial damage at risk of developing heart failure. The medical and device treatment of patients with established heart failure has also improved considerably, reducing both morbidity and mortality (7-10). Both changes are thought to have led to an increase prevalence of heart failure. Thus, heart failure has become one of the most common cardiovascular diseases in the western world. Epidemiologic data show a prevalence of heart failure of 2%, among individuals older than 75 years, nearly 10% suffer from heart failure (11). Notably, the prevalence is even higher in patients with diabetes (12, 13) conversely; the prevalence of diabetes is very high in patients with heart failure with estimates of up to 40% in patients hospitalized with worsening symptoms (14, 15). Worldwide it is estimated that 26 million peoples are affected with heart failure (5), with over 6.5 million adults in the USA (16) and at least 15 million in Europe affected (17). The incidences of HF in the USA along are estimated to be close to 1 million annually (16).

2. Case description

Case presentation 1:

A 66-year's old female with known case of type-2 diabetes mellitus presented to the hospital OPD with chief complain of severe dyspnea at mild exertion (NYHA III), bilateral lower limb swelling and decreased urine output. Shortness of breath is associated with orthopnoea, paroxysmal nocturnal dyspnea. There is a no history of alcohol intake and smoking in past. On examination she was in sinus rhythm with pulse rates was 58 beats/minute, blood pressure was 150/70 mm Hg, respiratory rate was 26 times per minute and temperature were 98.6°F. On local examination, she has bilateral pitting edema, cyanosed and on systemic examination, she has an inspiratory basal crepations over the lung base on both sides.

On laboratory investigation total Red blood cell counts was 4.5 millions/mm^3(Normal range: 4.5-5.5 million/mm^3), Total white blood cells count was 10 thousand/mm^3(Normal range: 4-11 thousand/mm^3), Total platelet count was 2 lakh/mm^3(Normal range: 1.5-3 lakh/mm^3), Random blood sugar was 120 mg/dl(Normal

range: 70-110 mg/dl), Blood urea was 45mg/dl(normal range: 10-50 mg/dl), serum creatine was 1.2 mg/dl(Normal range: 0.5-1.5 mg/dl), sodium was 142 mmol/liter(normal range: 135-145 mmol/liter), potassium was 4.2 mmol/liter(normal range: 3.5-5 mmol/liter), CK-MB was 15 IU/L(Normal range: 5-25 IU/L), Troponin-I was 0.01 ng/ml(Normal range: < 0.04 ng/ml), Brain natriuretic peptide was 832.40 pg/ml(Normal range:< 300pg/ml)and thyroid function test is within normal limit. Chest x-rays show cardiomegaly with small bilateral pleural effusions (shown in **figure 1**). On Electrocardiogram finding, there is P-wave absent and rhythm is irregularly irregular that is R-R interval is irregular with left ventricular hypertrophy. Echocardiogram show enlarged and dilated left ventricle (shown in **figure 2**) with severe end diastolic dysfunction. Left ventricular ejection fraction is about 40%.

Medical therapy was started with diuretics (Furosemide 40 mg twice daily), Angiotensin receptors blockers (Tab Valsartan 160 mg daily), beta blockers (Tab Carvedilol 12.5 mg daily), and Tab Digoxin 0.25 mg daily, Tab Atrovastatin 20 mg daily, Tab Nifedipine 30 mg daily, Tab metformin 1000 mg twice daily and Tab Ranitidine 150 mg once daily. The patient showed clinical improvement and discharged after 10 days of hospital admission in oral medications.

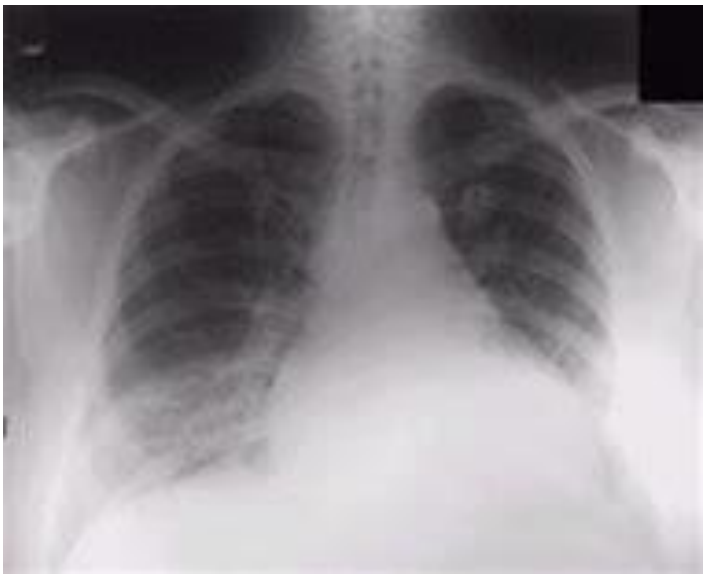

Figure 1

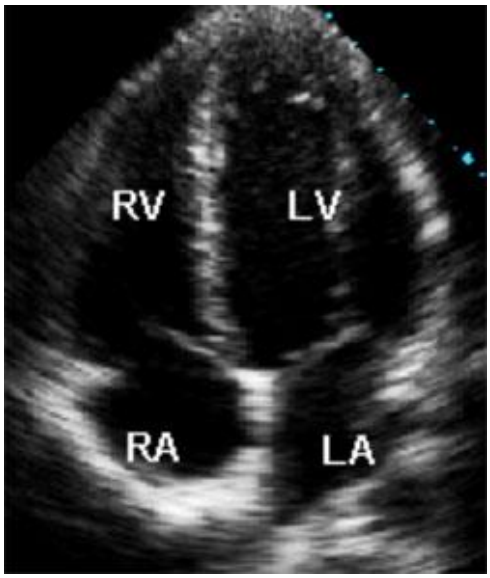

Figure 2

Figure 1: Chest x-ray showing Cardiomegaly with LVH.

Figure 2: Echocardiography, apical four chamber views showing Left ventricular hypertrophy. RA (right atrium), LA (Left atrium), LV (left ventricle), RV (right ventricle).

Case presentation 2:

A 65-year female patient presented to emergency department with several dyspnea at mild exertion (NYHA III) and history of myocarditis, type 2 diabetes mellitus and chronic atrial fibrillation. Shortness of breath is associated with bilateral lower limb swelling and decreased urines output. There is a history of smoking for last 20 years but no history of alcohol intake in past. On examination, she looks ill, with pulse rate 120 b/minute, respiratory rate 24 time/minute, blood pressure was 110/70 mm of Hg. On chest

examination there is crepitations is present whole over lung in both sides.

On laboratory investigation total Red blood cell counts was 5.3 million/mm^3(Normal range: 4.5-5.5 million/mm^3),Total white blood cells count was 8 thousand/mm^3(Normal range: 4-11 thousand/mm^3),Total platelet count was 1 lakh/mm^3(Normal range: 1.5-3 lakh/mm^3), Random blood sugar was 105 mg/dl(Normal range: 70-110 mg/dl), Blood urea was 43 mg/dl(normal range :10-50 mg/dl), serum creatine was 1.1 mg/dl(Normal range: 0.5-1.5 mg/dl), sodium was 140 mmol/liter(normal range: 135-145 mmol/liter), potassium was 3.2 mmol/liter(normal range: 3.5-5 mmol/liter), CK-MB was 12 IU/L(Normal range: 5-25 IU/L), Troponin-I was 0.01 ng/ml(Normal range: < 0.04 ng/ml), Brain natriuretic peptide was 945.40 pg/ml(Normal range:< 300pg/ml)and thyroid function test is within normal limit. Chest x-rays show cardiomegaly. On Electrocardiogram finding, there is P-wave absent and rhythm is irregularly irregular that is R-R interval is irregular with left ventricular hypertrophy. Echocardiogram show enlarged and dilated left ventricle with severe end diastolic dysfunction. Left ventricular ejection fraction is about 38%.

Medical therapy was started with diuretics (Furosemide), ACE inhibitors, Digoxin, Metformin. Patients feel better and after 8 days of admission patient was discharge on oral medication.

Case presentation 3:

A 72-year male patients with known case of type 2 diabetes mellitus, hypertension and stage 3 chronic kidney disease came in our clinics with complain of shortness of breath, chest pain and decreased urine output for two weeks. On examination patients is looking ill and there is a bilateral lower limb massive swelling. The blood pressure was 100/60 mm of Hg; pulse rate was 105 beat/minutes, respiratory rate was 18 time/minutes and SPo2 was 90 %. On chest examination there was crepitations was present on base of the both lung and decreased breath sound is heard on right lower part of chest.

On laboratory investigation total Red blood cell counts was 4.1 millions/mm^3(Normal range: 4.5-5.5 million/mm^3),Total white blood cells count was 7 thousand/mm^3(Normal range: 4-11 thousand/mm^3),Total platelet count was 1.7 lakh/mm^3(Normal range: 1.5-3 lakh/mm^3), Random blood sugar was 90 mg/dl(Normal range: 70-110 mg/dl), Blood urea was 25mg/dl(normal range :10-50 mg/dl), serum creatine was 0.8 mg/dl(Normal range: 0.5-1.5 mg/dl), sodium was 138 mmol/liter(normal range: 135-145 mmol/liter), potassium was 3.2 mmol/liter(normal range: 3.5-5 mmol/liter), CK-MB was 12 IU/L(Normal range: 5-25 IU/L), Troponin-I was 0.01 ng/ml(Normal range: < 0.04 ng/ml), Brain natriuretic peptide was 1112.40 pg/ml(Normal range:< 300pg/ml)and thyroid function test is within normal limit. Chest x-rays show cardiomegaly with small bilateral pleural effusions. On Electrocardiogram finding, there is P-wave absent and rhythm is irregularly irregular with left ventricular hypertrophy. Echocardiogram show enlarged and dilated left ventricle with severe end diastolic dysfunction. Left ventricular ejection fraction is about 36%.

Medical therapy was started with diuretic (Furosemide), potassium sparing diuretics, Angiotensin receptors blockers (Telmisartan), metformin, Digoxin, Statin, and Calcium channel blockers. There was improve in symptom of patients and after 12 days of admission patient was discharge on oral medication.

3. Discussion

Heart failure is a complex syndrome in which abnormal heart function results in, or increases the subsequent risk of, clinical symptoms and signs of low cardiac output or pulmonary or systemic congestion. Heart failure (HF) represents a heterogeneous population of patients defined by multiple etiologies and characteristics sharing, however, a common clinical outcome characterized by disabling symptoms and chronic congestion leading to recurrent hospital admissions (1, 18, 19). Heart failure is a common condition in primary care with 1% of the population self- reporting this condition. Mortality is substantial, approaching 40% to 50% over 5 years. Atrial fibrillation (AF) is the most common cardiac arrhythmia (20) and is found most frequently in patients with obesity, hypertension, diabetes mellitus, coronary artery disease or obstructive sleep apnea (21). HF shares these same risk factors, which contribute to coexistence of atrial fibrillation (21). The data suggest that AF is most often risk factors for HF than the reverse (10). The prevalence of new AF is high in patients with HF, ranging from 13 to 27 % and increasing as HF becomes more symptomatic(4). The pathways leading to AF in HF can be grouped into structural substrate for reentry and atrial ionic remodeling (22). The structural substrate for AF is atrial fibrosis, as the volume and composition of extracellular matrix has been corrected with persistence of AF (23). Atrial fibrosis is found in a variety of AF promoting conditions such as advanced age(24), dilated cardiomyopathy(25), mitral dysfunction(26), HF induces atrial fibrosis (27), which interfere with local conduction without significantly altering the effective period (28). In patients with HF, electrophysiology studies have confirmed these structural and conductive abnormalities, also without changes in atrial refractoriness (29). Ionic abnormalities are also found in patients with HF, which lead to calcium overload and prolonging of the atrial action potentials, particularly at faster heart rate. The result is an increased risk of arrhythmogenic delayed after depolarization (30). The changes described above are secondary to elevated left ventricular filling pressure (30). The pro-fibrotic milieu is strongly dependent on neurohormonal activation by renin-Angiotensin- aldosterone system (RAAS)(30). The RAAS inhibition is effective in primary prevention of AF in patients with heart failure(31). Angiotensin II induces myocardial apoptosis and fibrosis through the mitogen activated protein kinase pathways(32), in HF Models, it is associated with myocardiocyte apoptosis and atrial fibrosis(33). Angiotensin II also induces oxidative changes in sinoatrial nodes through the nicotinamide adenine dinucleotide phosphate oxidase, lead to cell apoptosis, which in turn lead to sinus dysfunction by preventing normal impulse formation and propagation(34). The circles Closes when AF results in impaired diastolic relaxation, leading to clinical HF and further atrial remodeling. Tachycardia induced cardiomyopathy may promote systolic and diastolic dysfunction as well, resulting additional remodeling (22).

4. Role of cardiac resynchronization therapy in the pathogenesis of AF in patient with HF:

Cardiac resynchronization therapy (CRT) is indicated for patients with HF and left ventricular ejection fraction (LFEF) ≤ 35 % who have evidences of delayed intraventricular conduction (QRS duration ≥ 120 ms), NYHA functional class III/IV despite receiving optimal pharmacological for at least 3 months and have a life expectancy of ≥ 1 years with good functional status(35,36). Recipients of CRT can be classified as a responders or non-responders based on reverse cardiac remodeling. The presence of reverse atrial remodeling is thought to be the reason behind the

atrial anti- arrhythmic effect of CRT(37), mainly secondary to improved atrial hemodynamic(38) and reduced atrial stretch as a consequences of improved mitral and ventricular hemodynamic.

Patients with HF who have undergone CRT placement appear to have an annual incidence of AF of 2.8 % compared with 10.2 % who did not undergo CRT placement (39). The incidences of AF after 3 years follow up was significantly lower in CRT responder than non-responders, determined by degree of reverse cardiac remodeling (40). In MADIT- CRT trials CRT responders had a 53 % risk reduction for atrial tachyarrhythmia compared with patients who had no undergone CRT placement (41).

5. AF therapeutics in patients with heart failures

In addition to risk factors modification, therapeutics considerations in patients with AF are broadly categorized into anticoagulation, rate control and rhythm control (4). Cardioversion should be performed if there is a hemodynamic compromise. Targeting an initials heart rate of 110 beat per minute is recommended. Early consideration should be given to rhythm control.

5.1. Anticoagulation Therapy

Anticoagulation therapy is not indicated in all patients with AF. The current guidelines recommended risk stratification using clinical scores to assess the probability of thromboembolic events compared with hemorrhagic events (42, 43). The risk of thromboembolic events in patients with AF is best assessed by using CHA_2DS_2-VASc score (44).

a. The current guidelines recommended anticoagulation in patients with AF if the CHA_2DS_2-VASc score is $\geq$ 2 (43). Anticoagulation can be achieved with either warfarin or direct acting oral anticoagulation (43).

b. If the CHA_2DS_2-VASc score is 1, anticoagulation, aspirin or anti-thrombotic therapy are feasible options, though anticoagulation is preferred in male patients.

c. If the CHA_2DS_2-VASc score is 0, anti-thrombotic therapy can be omitted.

5.2. Rate control versus rhythm control strategies

It is still unclear whether patients with HF benefit from a rhythm control strategy over a rate control approach (45). Both are accepted therapeutic option (46) and rate control should be the initial approach. Both strategies led to improvement in LVEF and functional status (47). The reversal to sinus rhythm has been associated with improvement in quality of life and LVEF (48). The studies show that rate control is non-inferior to rhythm control, outcomes might be better with rhythm control, as cardiovascular mortality, bleeding and hospitalizations for HF is less frequent (49). The AF catheter ablation is superiors to rate control in improvement of LVEF, functional capacity and quality of life (50).

The preferred target heart rate is to be 11o bpm (51); however, ACC/AHA/ARS guidelines follow the target heart rate to be 80 bpm. The beta blockers and non-dihydropyridine calcium channel blockers are first lines option for rate control. The ESC recommended digoxin as a first lines medication. Amiodarone and Atrioventricular node/junction ablation with permanent ventricular pacing are reserved for patients unable to achieve target heart rate. Amiodarone is for rhythm control is reserved for patients with significant systolic dysfunction or second lines agents. Digoxin has been associated with an increased risk of death in patients with AF (52), although the effect appears to be lower in patients with HF than without heart failure (53).

The use of carvedilol is associated with LVEF improvement (54), and beta blockers lead to reduction in resting heart rate are associated with reduction in all causes mortality with HF(55).

5.3. Cardioversion and devices therapy

In hemodynamic compromised patients, cardioversion is recommended to restore the normal sinus rhythm (56). The intra-cardiac defibrillator (ICD) is used in patients with HF to prevent deadly ventricular arrhythmias. Recently, it has been proposed that cardioversion of hospitalized hemodynamic compromised patients with HF and AF can be achieved through ICD defibrillation (57).

CRT is to be beneficial in the primary prevention of AF with HF. Patients with HF and established AF, who qualifies for CRT placement also derive AF related benefits from CRT. About 10 % of patients with HF, who have permanent AF, may convert to normal sinus rhythm after CRT placement (38).

Catheter ablation is recommended for patients with atrial fibrillation who remain symptomatic despite anti- arrhythmic medications (58). In patients with HF and AF, catheter ablation appears to be associated with preservation of normal sinus rhythm (59). It is also associated with improvement in LVEF (60), quality of life, and exercise tolerance with modest side effect. More recently catheter ablation is associated with reduction in ventricular and atrial size in patient without ventricular fibrosis (61).

6. Heart Failure therapy

The foundation for all HF therapy includes nonpharmacologic and pharmacological management (62). Dietary, lifestyle and over-the-counter nonsteroidal anti-inflammatory drug indiscretions are common sources of HF exacerbations (62, 63). Lifestyle measures facilitate HF management. Communicating information about exercise and salt and fluid intake to patients is essential for optimal management.

(a) The patient can exercise aerobically 3 to 5 times per week (30 to 40 minutes per session) for New York Heart Association class I to III HF (64).

(b) All patients need to restrict salt intake to 2 to 3 g (0.5 tsp) per day. Patients with unremitting fluid retention or advanced cardiac failure (ejection fraction less than 35%) require restriction to less than 2 g (approximately 0.25 tsp) of salt per day(62).

(c) Have patients report any weight gain of 2 lb (1 kg) in1 to 2 days or 5 lb (2 kg) in 1 week. Selected patients might be suitable candidates to self-adjust their Furosemide doses, doubling Furosemide until normal weight is restored or holding Furosemide if weight decreased by 1 kg (62).

(d) Patients, especially those with renal dysfunction or hyponatremia, should restrict fluid intake to 1.5 to 2.0 L per day.

Diuretics are useful in providing symptom relief, especially acutely, but do not prevent long-term mortality (63). Overreliance on diuretics often results in hypotension and electrolyte abnormalities, limiting the use of other agents that reduce mortality. Loop diuretics are preferred for congestive symptoms. Once symptoms are relieved, use the lowest effective maintenance dose. Multiple daily dosing can be used to improve diuretic effect, especially if higher doses are needed. If persistent volume overload continues with optimal Furosemide therapy, add a low-dose thiazide diuretic or metolazone (most effective if given 30 minutes before Furosemide) (65). Remember to monitor daily weight and regularly measure creatinine, urea, potassium, and magnesium levels. Furosemide useful for congestive symptom relief, once congestion

resolve, reduces to lowest effective dose or stop so that agent with mortality evidence can be optimized.

Table 1: Nonpharmacologic management of heart failure (62).

Following are the Nonpharmacologic way of management of heart failure.
• Smoking cessation.
• Influenza and pneumococcal vaccination.
• No more than 1 alcoholic drinks per day.
• Fluid intake 1.5 to 2 liter per day.
• Daily monitoring weight.
• Exercise after stress test assessment.
• No added salt diet [2 to 3 g of salt per day].

Spironolactone (12.5 to 25 mg) should be considered for patients with an ejection fraction of less than 30% and severe symptoms of HF, despite treatment optimization (10). Although the target dose in the RALES (Randomized Aldactone Evaluation Study Investigators) trial was 50 mg, 25 mg daily was the average dose reached (10). Remember to watch out for Hyperkalemia. Isosorbide dinitrate or a nitroglycerin patch is additional options, especially for nocturnal dyspnea. Primary prevention of AF has also demonstrated with eplerenone (66).

While diuretics help symptoms, b-blockers (BBs) and Angiotensin-converting enzyme inhibitors (ACEIs) have the best demonstrated evidence for mortality and morbidity outcomes in HF (67-69). ACEIs used in all asymptomatic patients with LVEF < 35% and all patients with symptom of HF and LVEF < 40 %. ACEIs also used in all patients after AMI and continue in LVEF < 40 % or if AHF complicated the AMI. The maximum tolerated target dose of ACEI should be used in all HF patients with ejection fractions of less than 40% (70). ACEIs can be used in combinations with diuretics.

If ACEI intolerance develops, an Angiotensin receptor blocker (ARB) can be used (71, 72). Generally, ACEIs and ARBs should not be combined, as adverse effects increase with little extra benefit (73). Exceptions might include symptomatic patients with class III or IV HF on optimum ACEI and BB treatment, or those unable to tolerate beta blockers (62). Blood pressure serum creatinine and potassium should be monitored. To titrate ACEIs or ARBs, start at low doses and then double the dose at 1- to 2-week intervals until the target dose is reached or until intolerable side effects persist. Trial evidence for best HF outcomes has been with relatively high doses (74, 75).

B-Blockers have strong evidence for mortality reduction (67)and beta blockers start when heart failure is stable and euvolemic. Start at low doses and double the dose every 2 to 4 weeks. Warn patients to expect some symptom worsening, initially. Start with Bisoprolol 1.25 mg daily or Carvedilol 3.125- 6.25 mg twice daily. Used in all patients of heart failure with LVEF ≤ 40 % and if NYHA class IV. Beta blockers will improve left ventricular function, patient's well-being, decrease hospitalizations and treat atrial fibrillation. Avoid abrupt withdrawal, if necessary, can titrate the dose down in AHF and titrate up once stabilized. Both the drug Bisoprolol and

Carvedilol have mortality benefit (28,76). When pursuing maximum tolerated doses of ACEIs or

Beta blockers a heart rate as low as 50 beats per minute or a blood pressure as low as 80/50 mm Hg might not require any change in therapy, as long as the patient is not showing symptoms of hypotension (dizziness and falls).

Combination of Isosorbide dinitrate and Hydralazine should be considered in addition to standard therapy for African Americans patients with systolic dysfunction to decrease the mortality (77) and for HF patients unable to tolerate other standard treatment or who have chronic renal insufficiency. A nitroglycerin patch can be substituted for oral nitrates.

While patient dietary indiscretions often occur, we must also be aware of prescriber indiscretions that can exacerbate HF. Specifically, medications such as nonsteroidal anti-inflammatory drugs, antiarrhythmic agents, diltiazem, verapamil, stimulants, glitazones, corticosteroids, tumor necrosis factor blockers, and numerous cancer chemotherapeutic agents are implicate (63).

6.1. Patients receiving advanced HF therapies

AF is already present in 39 % of patients who are started on home inotrope therapy (78). Approximately 46-52 % of patients who undergoes left ventricular assist device (LVAD) placement already have AF (79) and new AF develops in approximately 11 % (79). AF is present in 27 % of patients referred for cardiac transplant but it does not appear to affect event free survival (80).

Table 2: Heart failure drugs with dose (62).

Class	Drugs	initial dose
1. ACEIs	Ramipril	1.25-2.5 mg twice daily or 5 mg twice daily
	Lisinopril	2.5-5 mg daily (20-40 mg daily)
	Enalapril	1.25- 2.5 mg twice daily or 10 mg twice daily
	Captopril	6.25-12.5 mg 3 time a day (25-50 mg TDS)
2. BBs	Bisoprolol	1.25 mg daily or 10 mg daily
	Carvedilol	3.125- 6.25 mg twice daily
	Metoprolol-SR	12.5- 25 mg daily
3. ARB	Candesartan	4 mg daily (32 mg daily)
	Valsartan	40 mg twice daily (160 mg twice daily)
	Losartan	25- 50 mg daily (< 150 mg daily)
4. Aldosterone antagonist	Spironolactone	12.5 mg daily (12.5- 25 mg daily)
5. Vasodilators	Isosorbide dinitrate	20 mg three time a day
	Hydralazine	37.5 mg three times a day (75 mg TDS)
6. Diuretics	Furosemide	20- 40 mg daily to twice daily
	Hydrochlorothiazide	12.5- 25 mg daily to twice daily
	Metolazone	2.5- 5 mg daily
7. Cardiac glycoside	Digoxin	0.0625- 0.125 mg daily

The presence of AF in patients who received LVAD has been associated with an increased risk of death, particularly if it is permanent AF (79). The patients with HF and AF who undergo LVAD have AF related benefits. LVAD is associated with reduced left atrial size and volume index, which results in beneficial electrical remodeling (79). Consequently, 43 % of patients with preoperative AF had no evidence of post LVAD AF during follow up.

7. Learning point

a. Patient education is key (consider referral to an interprofessional HF clinic where available).
b. Make sure all patients with reduced ejection fraction are on the maximum tolerated dose of BB and ACEI or ARB.
c. After HF is controlled, titrate BB dose gradually (every 2 to 4 weeks); patient will feel worse before feeling better; ACEI dose should be titrated every 1 to 2 weeks.
d. To optimize ACEI and BB dose, decrease dose of diuretics nitrates, and other antihypertensive.
e. Consider adding a third drug (spironolactone, digoxin, nitrate) if the patient is still symptomatic on ACEI and BB.
f. Ensure ongoing communication among health care providers.

Conclusion:

Heart failure is an imprecise term used to describe the state that develops when heart cannot maintain adequate cardiac output. Almost all forms of heart disease can lead to heart failure and most frequently due to coronary artery disease. AF frequently coexists with HF due to HF induced atrial remodeling. It is associated with increase morbidity and mortality with corresponding decrease in quality of life. HF is usually presenting with a sudden onset of dyspnea at rest that progress to acute respiratory distress, orthopnoea, paroxysmal nocturnal dyspnea and inspiratory crepitations over the lung base. Educating patients on lifestyle measure support medication management. The cornerstones of AF management–anticoagulation therapy and rate or rhythm control remain essential in patients with HF and AF along with conventional HF treatment. Ensuring patients approach the maximal tolerated doses of ACEIs and BBs improve mortality and morbidity. Finally, RAAS inhibition is a promising field of study in patients with HF and AF, as it provides structural benefits that affect both pathologies.

Abbreviations:

HF: Heart failure.
AHF: Acute heart failure.
ACC: American college of cardiology.
AHA: American Heart association.
ARH: American Rhythm Society.
NYHA: New York heart associations.
AMI: Acute myocardial infarction.
LVEF: Left ventricular ejection fraction.
RAAS: Renin Angiotensin Aldosterone system
ACEIs: Angiotensin converting enzyme inhibitors.
ARBs: Angiotensin Receptors Blockers.
BBs: Beta blockers.
CRT: Cardiac resynchronization therapy.
ICD: Intracardiac Defibrillators.

Declarations

1) Consent to publication

We declare that all authors agreed to publish the manuscript at this journal based on the signed Copyright Transfer Agreement and followed publication ethics.

2) Ethical approval and consent to participants

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by Editors in chief of this journal.

3) ***Disclosure of conflict of interests***

We declare that no conflict of interest exists.

4) ***Funding***

None

5) ***Availability of data and material***

We declare that the data supporting the results reports in the article are available in the published article.

6) ***Acknowledgement***

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7) ***Author contribution***

Authors contributed to this paper with the design (LND and YS), literature search (LND), revision LND and YS), editing (LND and YS) and final approval (LND).

8) ***Authors' biography***

None

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