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Case Report

Patients with Rapid Atrial Fibrillation in Congestive Heart Failure with Bilateral Pleural Effusion Complication: Case Report and Literature Review

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ABSTRACT

Atrial fibrillation (AF) is the most common form of arrhythmia. The prevalence of AF in patients with systolic left ventricular dysfunction and CHF is around 35% in the patient population with NYHA II-IV. In this case, we will discuss a case report of a patient with AF with complications of CHF and Bilateral Pleural Effusion. **Case Summary.** A female patient, 62 years old, came to the emergency room of dr. Soegiri Hospital with complaints of sudden shortness of breath since morning. There were crackles in the right lung field during the auscultation and the chest x-ray shows cardiomegaly with a CTR> 50% with bilateral effusion and atelectasis. On the electrocardiogram examination performed at the ICCU, an irregular rhythm was obtained with an HR of 100x/minute, with p waves that cannot be distinguished, normal QRS waves, and T waves that cannot be assessed. The patient was diagnosed with congestive heart failure (CHF) with rapid atrial fibrillation (AF) accompanied by complications of bilateral pleural effusion with a differential diagnosis of acute decompensated heart failure (ADHF) with pneumonia and acute lung edema (ALO). **Discussion.** Patients with AF and CHF have a poorer prognosis than patients with a single diagnosis of AF or CHF alone. A rhythm control strategy consisting of antiarrhythmic drugs and electrical cardioversion in stable patients with AF and CHF adds no benefit to a heart rate control strategy.

Highlights:

1. In the setting of AF in patient with AHF, conventional treatment strategies do not convert the rhythm into sinus rhythm.
2. Newly emerged therapies such as catheter ablation, or atrioventricular node ablation with biventricular pacing might be more beneficial rather than the conventional rhythm and rate control strategies.

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Introduction

Atrial fibrillation (AF) is the most common form of arrhythmia. The AHA (American Heart Association) statistics show the incidence of Atrial fibrillation (AF) in men is 20.6 per 100,000 population (age 15-44 years) to 1077.4 per 100,000 population (age ≥85 years). In women, the incidence is 6.6/100,000 population (age 15-44 years) to 1203.7/100,000 population (age ≥85 years).[1]

Heart failure (HF) is a health problem with a high incidence worldwide. Mortality and morbidity caused by heart failure are increasing annually with a prevalence of around 23 million people worldwide. Data from the World Health Organization (WHO) in 2013 found that 17.5 million people worldwide died from cardiovascular disease, around 31% of the world's population. The incidence of heart failure in the United States is approximately 550,000 cases per year. In developing countries, the incidence of heart failure is higher with around 400,000 to 700,000 cases per year.[2,3]

The prevalence of AF in patients with systolic left ventricular dysfunction and CHF ranges from 6% for patients without symptoms or for those with minimal symptoms, to between 15% and 35% for patients with New York Heart Association (NYHA) class II-IV symptoms. In two large epidemiological studies, the prevalence of AF was greater in patients with CHF with preserved ejection fraction than with left ventricular systolic dysfunction, although AF rates were similar in patients with systolic versus diastolic CHF in one clinical trial.[4]

In this case, we will discuss a case report of a patient with AF with complications of CHF and Bilateral Pleural Effusion.

Case Presentation

A female patient, 62 years old, came to the emergency room of dr. Soegiri Hospital with complaints of sudden shortness of breath since morning. The patient had no complaints of cough, runny nose, fever, and weakness. There was no history of diabetes mellitus and hypertension. The patient had no history of hospitalization and surgery. The patient is not currently taking any other treatment. On admission the patient looked lethargic with vital signs of blood pressure 110/60 mmHg, pulse 160x/minute, respiratory rate 30x/minute, peripheral oxygen saturation (SpO2) 98% with non-rebreathing mask, and temperature 36.6oC. The patient appeared to have difficulty breathing (dyspnea). The patient's CRT is > 2 seconds. There were crackles in the right lung field during the auscultation of the patient. The chest x-ray found cardiomegaly with a CTR> 50%, bilateral effusion, and atelectasis. (Figure 1).

From the initial hospital the patient received a 500cc/24 hours infusion of Ringer lactate (RL), 3x1 injection of Metamizole 2ml/ampule, 2x1 injection of Ranitidine 50mg/2ml, 1 ampule 20cc of Digoxin

given over in 20 minutes, 25 nano/KgBB/hour of Norepinephrine, 25 mCq drip of KCl, and 1x1 Warfarin sodium clathrate 2mg.

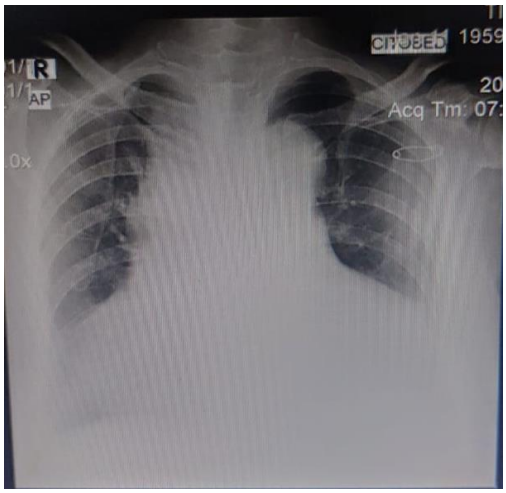

Figure 1. Patient's chest x-ray. CTR> 50% with bilateral effusion

While in the ICCU, the patient's symptomps was relieved, with a blood pressure of 108/74mmHg, heart rate of 75x/minute, respiration rate 23x/minute, oxygen saturation (SpO2) of 98% with a non-rebreathing mask of 13 lpm, and patient's temperature of 36.0oC. On thoracic examination, the patient's JVP was 8cm, with symmetrical breathing movements, dull percussion on the distal part of the thorax, and rhonchi in the auscultation. The patient had swelling in the right and left legs. The patient's input fluids record was 500cc 20ml/hour of 0.9% NaCl infusion, 200cc from drinking, and output fluid record from 1500 cc of 24-hour urine output and 100cc of IWL. The patient's serum electrolytes can be seen in table 1.

Table 1. Patient's electrolyte examination while in the ICCU

Electrolyte	Value	Reference range
Chloride	104 mmol/l	97-111 mmol/l
Potassium	3,7 mmol/l	3.5-5.1 mmol/l
Natrium	143 mmol/l	136-146 mmol/l

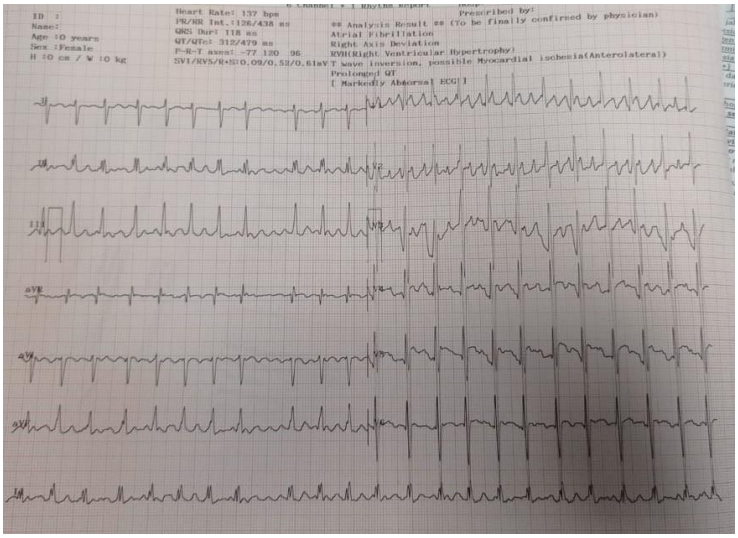

Figure 2. Patient's ECG while in the ICCU

On the electrocardiogram examination performed at the ICCU, shows an irregular rhythm with HR over 100x/minute. There were many p waves that could not be distinguished, normal QRS waves, and T waves that could not be assessed. The ECG shows Atrial Fibrillation with Rapid Ventricular Response 75-137 beat per minute, with normal axis. (Figure 2). Patients received 500cc/24 hours of PZ infusion, Warfarin sodium clathrate 1 x 2mg, Furosemide 2 x ½ 40mg, Bisoprolol 1 x ½ 2.5 mg, injection of Metamizole 3 x 2ml/ampule, Injection of Ranitidine HCL 2 x 50mg/2ml and Ondansetron 3 x 4 mg.

The patient was diagnosed with congestive heart failure (CHF) with rapid atrial fibrillation (AF) accompanied by complications of bilateral pleural effusion with a differential diagnosis of acute decompensated heart failure (ADHF) with pneumonia and acute lung edema (ALO).

Discussion

In atrial fibrillation, electrical impulses do not start from the SA node, but from other parts of the atria or near the pulmonary veins. This will generate rapid and erratic impulses so that the atria will beat in a precise and erratic manner. When the electrical impulse arrives at the AV node, the AV node will continue the impulse, although not as fast as the initial impulse so the ventricles will also beat fast but not as fast as the atria. Therefore, the atria and ventricles no longer beat together. This causes blood in the atria not to pump into the ventricles as it should.[1]

In patients with AF, some changes in blood pressure (hypertension or hypotension); pulse may be irregular/deficit, irregular heart sounds and rhythms, pale skin, cyanosis, sweating, edema, and decreased urine output if there is a severe decrease in cardiac output. Patients with AF may also come to the hospital with syncope, throbbing dizziness, headache, disorientation, confusion, lethargy, and pupillary changes. In this case, the patient came with shortness of breath with a respiration rate of 30 times per minute. The normal rate of respiration is 12-16 times per minute, therefore this patient indicates difficulty in breathing (dyspnea).[5]

Complications that can be caused are stroke and heart failure. A stroke occurs due to the release of a blood clot (thrombus) in the atrium, which then clogs the brain's blood vessels. Heart failure occurs when the heart can't pump enough blood to meet the body's needs. The ECG shows irregular waves, a narrow QRS complex, and a speed of >300x/minute.[1]

The temporal relationship of AF and CHF was examined in a study of 1470 patients with new-onset AF or CHF from the Framingham Heart Study. In patients diagnosed with AF, the incidence of developing CHF is approximately 33 patients per 1000 patients annually. In contrast, among

individuals diagnosed with CHF, the prevalence of developing AF is 54 per 1000 patients per year.[6]

The development of AF in patients with HF is associated with a poorer prognosis. The Framingham Heart Study reported that AF complicated by HF was associated with an increase in all-cause mortality (men: HR, 2.7 [95% CI, 1.9–3.7]; women: HR, 3.1 [95% CI, 2,2– 4.2]). 12 Similarly, in individuals with HF developing AF was subsequently associated with an increase in all-cause mortality (men: HR, 1.6 [95% CI, 1.2–2.1]; women: HR, 2.7 [95% CI, 2.0-3.6]). In a meta-analysis that included seven RCTs and nine observational studies, AF was associated with increased all-cause death in heart failure with an odds ratio of 1.40 (95% CI, 1.32-1.48) in randomized trials and 1.14 (95% CI 1.03-1.26) in an observational study.[7]

Persistent AF disease can lead to arrhythmia-induced cardiomyopathy (AIC) and heart failure reduced ejection fraction (HFrEF), important and potentially reversible conditions requiring a high index of suspicion to be diagnosed. An aggressive diagnostic approach is recommended in cases of AIC, especially with a rhythm control strategy. The process is mediated by altered cellular and neurohumoral factors, as well as extracellular remodeling, and is more likely by the presence of underlying structural heart disease[14]. In a study of 19 patients with suspected AIC (84% with AF), endomyocardial biopsies showed evidence of macrophage-mediated inflammation, including increased expression of histocompatibility complex class II, CD68+ macrophage infiltration, and absent or low levels of CD3+ T cells.[8[

AIC disease and HFrEF may manifest in patients with adequate HR rates suggesting that patients and their associated arrhythmia mechanisms contribute to AIC with higher rapid ventricular rates. In this situation, restoration and maintenance of sinus rhythm have been shown to increase LVEF. In AF disease accompanied by HFrEF, one should be suspicious of AIC, which requires aggressive treatment of both conditions.[9] The European Society of Cardiology/European Association of Cardio-Thoracic Surgery in 2020 on AF guidelines provides class I recommendations for catheter ablation (CA) in patients with AF and HFrEF because AF-induced cardiomyopathy is very likely.[10]

Pleural fluid accumulation in heart failure is more associated with left ventricular failure than right ventricular failure. A recent study described pleural effusion caused by right heart failure occurring in 19 of 147 patients (13%) with idiopathic or familial pulmonary hypertension. Nearly two-thirds of the pleural effusions were small and were located unilaterally on the right side in 11 patients (58%), left-sided unilateral in three patients (16%), and bilateral in five patients (26%). Four out of five patients who undergo thoracentesis have a transudate.[11]

Pharmacological management of AF depends on clinical presentation, hemodynamic status, ventricular rate, choice of therapy, risk (based on age and comorbidities), and type of AF (paroxysmal, persistent, or permanent). The patient's clinical presentation with acute or chronic HF or AF must be considered. A diagnostic and therapeutic approach should be taken to treat HF and AF according to the severity of each condition. During the acute phase, this may require control of the ventricular rate in cases of AF and medical therapy according to the guidelines for HFrEF.[12]

Treatments for heart failure, including angiotensin-converting enzyme inhibitors, angiotensin receptor inhibitors, angiotensin-neprilysin receptor inhibitors, blockers, diuretics, mineralocorticoid receptor antagonists, and sodium-glucose cotransporter-2 inhibitors, can improve HF outcomes and have an effect on improving heart failure. AF. In a meta-analysis, in four trials evaluating angiotensin-converting enzyme inhibitors and angiotensin receptor inhibitors, there was a relative risk reduction of 44% (95% CI, 15%-63%; P=0.007) in AF patients (in various pathways) with heart failure. Subjects in the study with the most impaired LV function had the greatest reduction in AF (reduction in RR, 78%).[13]

Long-term medical management of HFrEF depends on evidence of structural myocardial damage and ventricular function with normalization of rate or rhythm. Long-term medical management of heart failure in patients with tachycardia due to cardiomyopathy secondary to AF is controversial but may be beneficial in patients with persistent structural abnormalities. Thus, implantable cardioverter defibrillators should not be considered a therapeutic option until the patient is stabilized and reevaluated from the standpoint of HFrEF and AF (rate or rhythm control). After treatment for heart rate control, the next step is determining the need to return the patient to normal sinus rhythm, which is often accomplished by direct current cardioversion. Further management, whether rate control, rhythm control or both, must take into account the expected efficacy of the approach and the avoidance of futile and unnecessary therapy (eg, frequent short-term cardioversion).[14]

In terms of antiarrhythmic drug therapy, only two drugs have been evaluated and recommended in patients with HFrEF: amiodarone and dofetilide.[21] Dofetilide, is more effective than placebo in maintaining sinus rhythm in patients with heart failure. Concerns about serious side effects and proarrhythmic exist with many antiarrhythmic drugs, especially in patients with heart failure.[14]

Heart rate control is difficult to achieve with pharmacological therapy alone. Although β-blockers can control ventricular rate in patients with HFrEF and AF, the benefit of β-blockers in all-cause mortality has only been seen in patients with HF in sinus rhythm. Whereas digoxin in combination with a

β-blocker may help control heart rate levels in patients with AF, digoxin has been associated with increased mortality.

Calcium antagonists for control of the rate of AF in HFrEF are not recommended. The European Society of Cardiology/European Association of Cardio-Thoracic Surgery recommends “a resting heart rate” of <110 beats per minute as the initial target heart rate for rate control therapy in AF (Class IIa; Level of Evidence B). More stringent heart rate control should be performed in patients with AF in the presence of symptoms or worsening LVEF. The exact and optimal ventricular rate in AF at rest and with activity in patients with heart failure is uncertain.[15]

Conclusion

Patients with AF and CHF have a poorer prognosis than patients with a single diagnosis of AF or CHF alone. The pathophysiological mechanisms of AF in patients with CHF are complex. Therefore, it is unlikely that current treatment strategy aimed at either of these mechanisms alone will restore the patient to sinus rhythm. Newly emerged therapies such as catheter ablation for AF, and rate control therapies such as atrioventricular node ablation with biventricular pacing, shows potential alternatives to conventional rhythm and rate control strategies.

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