JURNAL KOMPLIKASI ANESTESI
VOLUME 12 NOMOR 3, August 2025

CASE REPORT

Transform Your Heart Failure Treatment Approach: Conquering Svt and Pneumonia Challenges

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How to cite: Utami NP J, et al, Transform Your Heart Failure Treatment Approach: Conquering Svt and Pneumonia Challenges. Jurnal Komplikasi Anestesi 12(3)-2025.

Receive: July 20, 2024
Accepted: December 26, 2025

ABSTRACT

In this complex case, an 81-year-old female presented with acute decompensated heart failure (ADHF), pneumonia, and SVT-induced tachyarrhythmia. Upon admission, she received meticulous therapy in the intensive care unit (ICU). The treatment regimen was multifaceted, aiming to address each condition effectively. Fluid balance was closely monitored and maintained. Pharmacological interventions played a crucial role, with digoxin administered initially to manage SVT, alongside medications targeting heart failure including clopidogrel, atorvastatin, spironolactone, candesartan, and furosemide. Antibiotic therapy was initiated promptly following consultation with a pulmonologist, comprising ceftriaxone, levofloxacin, and methylprednisolone for pneumonia management. Additionally, regular nebulizations with salbutamol-ipratropium were administered. By the third day of hospitalization, significant improvement was observed, allowing for the patient's transfer to a general medical ward. This case underscores the importance of a comprehensive therapeutic approach in managing complex medical conditions, such as ADHF exacerbated by SVT and pneumonia, to achieve optimal patient outcomes.

Keywords: ADHF, arrhythmia, SVT, pneumonia

INTRODUCTION

Acute decompensated heart failure (ADHF) is a clinical syndrome characterized by the rapid onset or worsening of symptoms and signs of heart failure, necessitating urgent medical intervention.[1,2] It is frequently precipitated by various factors, the primary causes include noncompliance with medications and dietary restrictions; however, acute coronary syndrome, arrhythmias, uncontrolled hypertension, and infections such as pneumonia, which increase the metabolic demands on the heart and exacerbate the underlying cardiac dysfunction.[3] In primary care, respiratory infections and rapid AF are the most important precipitating factors for hospitalization and death within 30 d following an episode of heart failure decompensation.[4]

The interaction between arrhythmia and heart failure (HF) is intricate. It is evident that arrhythmia can aggravate HF, while HF concurrently increases the likelihood of developing arrhythmia.[5] Supraventricular tachycardia (SVT) is a common arrhythmia that can occur in patients with heart failure. SVT is characterized by an abnormally fast heart rhythm originating above the ventricles, which can further compromise cardiac output and precipitate or worsen heart failure symptoms.[6,7] The incidence and fatality rates of heart failure and cardiac arrhythmia are significantly elevated in the aging population aged 65 years and older. This demographic is projected to reach 973 million globally by the year 2030.[8] Pneumonia, through its systemic inflammatory response and hypoxic effects, can significantly aggravate heart failure, leading to a vicious cycle of worsening cardiac and pulmonary function.[9] The complex of ADHF worsening with SVT and pneumonia presents a particularly challenging clinical scenario due to the intricate and interdependent pathophysiological mechanisms involved.

This case report highlights the exacerbation of SVT in the setting of ADHF and pneumonia, illustrating the complex interplay between these conditions and the necessity for a comprehensive, multidisciplinary approach to management.

CASE

An 81-year-old female presented to the emergency department with a one-week history of progressively worsening dyspnea, which was exacerbated by lying down, and associated with chest tightness, fever, and bilateral lower extremity edema. The patient has a history of hypertension. On examination, the patient was in significant respiratory distress. Vital signs revealed a blood pressure of 130/90 mmHg, a heart rate of 138 beats per minute, a respiratory rate of 29 breaths per minute, oxygen saturation at 90% on room air, and a temperature of 36.3°C. Pulmonary auscultation disclosed decreased breath sounds bilaterally at the lung bases without adventitious sounds, while the cardiac examination demonstrated an irregular rhythm with murmurs auscultated at the fourth left intercostal space along the sternal border. Mild bilateral pitting pedal edema was also noted.

The 12-lead electrocardiogram (ECG) showed a narrow complex QRS tachycardia consistent with supraventricular tachycardia (SVT) accompanied by premature atrial complexes.(figure 1.a) A chest radiograph revealed cardiomegaly and infiltrates in the right lower lobe.(figure 2) Initial laboratory results included hemoglobin of 11.5 g/dL, leukocyte count of 12,900/µL, platelet count of 253,000/µL, blood urea nitrogen (BUN) of 12.2 mg/dL, urea of 26.13 mg/dL, and creatinine of 2.2 mg/dL. Four hours after administration of 0.5 mg of digoxin, follow-up ECG demonstrated irregular inverted P-waves followed by QRS complexes.(figure 1.b) Serum electrolytes revealed hyponatremia at 111.25 mEq/L, normokalemia at 4.25 mEq/L, and hypochloremia at 79.7 mEq/L.

The patient was diagnosed with acute decompensated heart failure (ADHF), pneumonia, and SVT-induced tachyarrhythmia. She was admitted to the intensive care unit (ICU) for closer monitoring and management. Fluid balance was meticulously maintained at 1500 cc/24 hours, comprising 1000 cc oral intake and 500 cc intravenous administration of 0.9% sodium chloride. The therapeutic regimen included clopidogrel 75 mg daily, atorvastatin 20 mg daily, spironolactone 25 mg daily, candesartan 16 mg daily, and furosemide

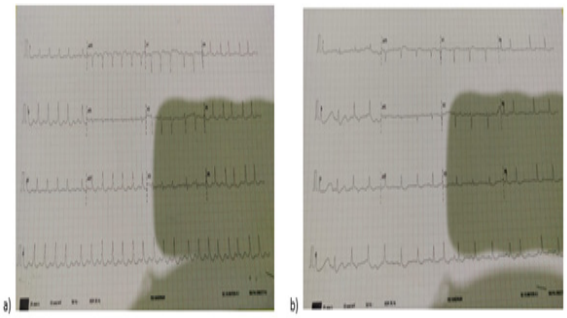

Figure 1. ECG during Admission 1b) ECG 4 Hours Post digoxin 0,5mg

20 mg thrice daily. Following a consultation with a pulmonologist, she received ceftriaxone, levofloxacin, methylprednisolone, and regular nebulizations with salbutamol-ipratropium for the management of pneumonia.

On the third day of hospitalization, the patient's ECG revealed sinus tachycardia without any pathological rhythms. Symptoms associated with acute decompensated heart failure (ADHF) had significantly improved; lower extremity edema was no longer present, and dyspnea had resolved, with only occasional coughing remaining. Consequently, the patient was transferred to a general medical ward. Antibiotic therapy was continued until the fifth day of hospitalization, after which the patient was discharged in stable condition.

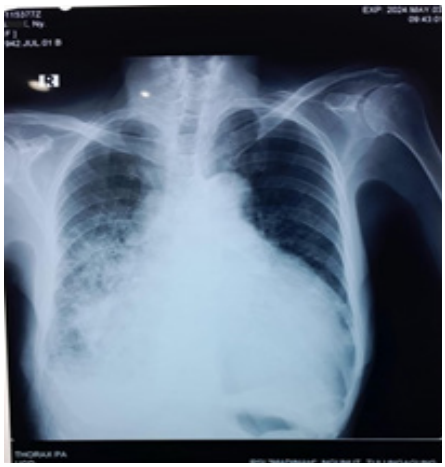

Figure 2. Chest X-ray showing Infiltrate and Cardiomegaly

DISCUSSION

The concurrence of acute decompensated heart failure (ADHF) and pneumonia, complicated by supraventricular tachycardia (SVT), presents a multifaceted clinical challenge due to the interrelated pathophysiological mechanisms that exacerbate each condition. This case highlights the intricate interplay between ADHF, pneumonia, and SVT, underscoring the need for a comprehensive and multidisciplinary approach to management.

In this case, the patient comes to the ER with classic signs and symptoms of ADHF10 These classic symptoms have proven to increase the probability of heart failure.[11] History of hypertension in this patient also strengthens the occurrence of ADHF. Hypertension causes concentric left ventricular hypertrophy (LVH), which subsequently leads to eccentric LVH and heart failure.[12] However, in this patient, ADHF is also exacerbated by the presence SVT and pneumonia, based on diagnostic examination findings. The 12-lead ECG showed no p-wave with narrow complex tachycardia (figure 1.a). Supraventricular tachycardia (SVT) is a type of tachycardias (atrial and/or ventricular rates >100 bpm at rest), with mechanisms involving tissue from the His bundle or above.[6] SVT can exacerbate ADHF by increasing the heart's workload and reducing cardiac output.

The increased heart rate associated with SVT shortens diastolic filling time, which can lead to a decrease in stroke volume and further compromise an already failing heart. This can result in worsened symptoms of heart failure, including pulmonary congestion and systemic hypoperfusion. The prevalence of arrhythmias, most AF, progresses with worsening HF with a prevalence of 10% in HF patients in the New York Heart Association (NYHA) Classes I and II, and up to 50% in NYHA Class IV.[13]

The patient's initial management included the administration of digoxin, which successfully converted the SVT to a more stable rhythm. Digoxin works primarily by inhibiting sodium-potassium ATPase, which increases intracellular calcium levels through the sodium-calcium exchanger. This results in a prolonged cardiac action potential, leading to a slower heart rate and enhanced myocardial contractility due to more calcium available for sarcomeric excitation-

contraction coupling. Additionally, digoxin has neurohormonal effects, improving baroreceptor sensitivity, reducing norepinephrine levels, and decreasing renin-angiotensin system activation. Electrophysiologically, digoxin exerts a parasympathetic influence on the sinoatrial node, reducing automaticity, and on the atrioventricular conduction system, decreasing conduction and extending effective refractory periods.[14] Digoxin was highly effective in lowering heart rate in patients experiencing acute heart failure due to tachyarrhythmia, demonstrating a good safety profile.[15]

Although considered as first-line therapy in SVT, The vagal maneuver, such as Valsalva maneuver or carotid sinus massage, is not applicable in this case. Patients with ADHF usually have significantly impaired ventricular function, and vagal maneuvers can cause notable hemodynamic changes. Valsalva maneuver increases intrathoracic pressure, potentially reducing preload and cardiac output, which could worsen the hemodynamic status of the patient.[16] Vagal maneuvers can induce bradyarrhythmias that not be well tolerated by patients with already compromised ventricular function.[17] This could lead to further reduction in perfusion to vital organs. In this case a safer approach to managing SVT may involve pharmacological agents such as adenosine or beta-blockers, which can provide more controlled rhythm management without causing drastic hemodynamic changes.

The chest radiograph, showing cardiomegaly and infiltrates in the right lower lobe, in line with leukocytosis findings, suggests a diagnosis of pneumonia. Pneumonia showed an independent association with in-hospital mortality, yet it did not correlate with long-term mortality. The diagnosis of pneumonia and the decision regarding antibiotic therapy in cases of ADHF pose challenges, despite the presence of chest radiograph findings.[9] The management of pneumonia involves broad-spectrum antibiotics (ceftriaxone and levofloxacin), corticosteroids (methylprednisolone), and nebulization therapy to address the underlying infection and reduce systemic inflammation. The therapeutic regimen for ADHF included a combination of diuretics (furosemide), RAAS inhibitors (candesartan), and aldosterone antagonists (spironolactone). Despite extensive research and development over many years, the primary pharmacological treatments for ADHF continue to be diuretics, vasodilators, and inotropes.[18] Traditionally, GDMT (guideline-directed medical therapy) has been implemented by introducing the four drug classes in the sequential order of their inclusion in the guidelines. This involves starting with ACE inhibitors, followed by beta-blockers, then MRAs, transitioning from ACE inhibitors to ARNis, and finally initiating SGLT2 inhibitors.[19]

Intravenous loop diuretics are the cornerstone treatment for most patients hospitalized with ADHF, mainly alleviating symptoms by reducing venous congestion and volume overload. Although early research indicated that vasodilators might improve symptoms, a recent large-scale trial comparing an early, intensive vasodilator strategy to standard care (primarily involving intravenous diuretics) found no significant difference in all-cause mortality or rehospitalization rates for ADHF.[20] Inhibition of the RAAS is recommended to reduce morbidity and mortality for patients with HF. ARBs do not inhibit kininase, resulting in a much lower incidence of cough and angioedema. For patients who cannot tolerate ACE inhibitors due to cough or angioedema, ARBs are recommended. ARBs should be initiated at low doses and gradually increased, aiming to reach doses that have been shown to reduce the risk of cardiovascular events in clinical trials.[21]

Maintaining a meticulous fluid balance was vital in this patient, given the propensity for fluid overload in ADHF. In this case, the fluid intake was carefully regulated, balancing oral and intravenous fluids to optimize hydration without exacerbating pulmonary congestion. However, in a meta-analysis, patients with heart failure who take liberal fluid intake does not appear to negatively affect the rate of rehospitalization or all-cause mortality.[22] In addition to fluid management, electrolyte imbalances play a critical role in the prognosis of heart failure patients. For patients hospitalized due to

worsening heart failure, the serum sodium level at admission is an independent predictor of a longer duration of hospitalization for cardiovascular reasons and increased mortality within 60 days after discharge.[23] Improvement of hyponatremia in patients with ADHF is linked to a reduced risk of mortality during follow-up, particularly in the short term.[24] By the third day of hospitalization, the patient's ECG revealed sinus tachycardia without any pathological rhythms, and her symptoms of ADHF had significantly improved.

The resolution of lower extremity edema and dyspnea indicated effective management of her heart failure and pneumonia. She was subsequently transferred to a general medical ward and continued on antibiotic therapy until the fifth day of hospitalization, after which she was discharged in stable condition. This case underscores the importance of an integrated approach to managing patients with complex, overlapping conditions such as ADHF, pneumonia, and SVT.

Early identification and targeted treatment of each contributing factor are essential to improving patient outcomes. The successful management of this patient highlights the need for continuous monitoring, appropriate pharmacological intervention, and fluid management in the treatment of such multifactorial cases. Further research and clinical guidelines are needed to refine the management strategies for patients with concurrent ADHF, pneumonia, and SVT, to ensure optimal outcomes and reduce the risk of complications.

CONCLUSION

This case highlights the importance of an integrated approach to managing patients with complex, overlapping conditions such as ADHF exacerbated by SVT and pneumonia. Early identification and targeted treatment of each contributing factor are essential for improving patient outcomes.

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