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HIV/AIDS Young Adult with Diabetes Mellitus Presenting with Newly Diagnosed Pulmonary Tuberculosis, Pneumonia Hypokalemia and Atrial Fibrillation: A Rare Case Report

Hesti Andika Putri, Reza Febryan
Universitas Riau, Indonesia
Email: hestiandikaputri@gmail.com, reza12feb1990@gmail.com

KEYWORDS

HIV, AIDS, Diabetes Mellitus, Pneumonia, Pulmonary Tuberculosis, Hypokalemia, Atrial Fibrillation, Young Adult

ABSTRACT

Pneumonia, pulmonary tuberculosis (TB), Human Immunodeficiency Virus (HIV)/Acquired Immunodeficiency Syndrome (AIDS), and metabolic disorders such as diabetes mellitus (DM) represent complex clinical challenges, particularly in young adults. This study reports and analyzes a rare case of a 25-year-old male with a unique combination of HIV infection, diabetes mellitus, clinical pulmonary TB, pneumonia, hypokalemia, and atrial fibrillation. The patient presented with shortness of breath for one day, a history of cough for more than three months, weight loss, fever, and prior insulin use five years earlier. Physical examination revealed pale conjunctiva and oral candidiasis, while laboratory results showed hemoglobin 10.8 g/dL, leukocytes 8,280/µL, platelets 296,000/µL, mean corpuscular volume (MCV) 89.4 fL, mean corpuscular hemoglobin concentration (MCHC) 33.9 g/dL, HbA1c 8.4%, potassium 2.7 mmol/L, lymphocytes 6.6%, and reactive HIV qualitative testing (R1, R2, R3). Electrocardiogram (ECG) demonstrated atrial fibrillation with normal ventricular response, chest X-ray revealed normal cardiac size with findings suggestive of bronchitis and suspected pneumonia, and thoracic CT scan with and without contrast showed features consistent with pneumonia and multiple bilateral paratracheal and subcarinal lymphadenopathy. This case underscores the importance of multidisciplinary management involving anesthesiology, pulmonology, internal medicine, cardiology, and pharmacy to ensure comprehensive care and optimize clinical outcomes, while preventing potentially fatal complications.

INTRODUCTION

Pulmonary tuberculosis (TB), Human Immunodeficiency Virus (HIV), and metabolic disorders such as diabetes mellitus (DM) often occur concomitantly and represent a significant clinical challenge due to overlapping pathophysiological mechanisms and their impact on immune function. In a study conducted in the North West Region, Cameroon (January 2017–December 2019), there were 1,115 pulmonary TB patients aged >14 years, showing that 38.6% of TB patients were also HIV-positive and 5.8% of TB patients had TB/HIV coinfection with *diabetes mellitus* (*TB/HIV/DM comorbidities*) (Sama et al., 2023). In patients with HIV–TB coinfection, dysregulation of the immune system increases susceptibility to opportunistic airway infections such as pneumonia and TB, while metabolic stress due to DM can worsen clinical outcomes. DM is also known to decrease immune function and increase the risk of active TB, while TB infection can impair glycemic control through pancreatic dysfunction (Peng et al., 2023).

Electrolyte disorders, particularly hypokalemia, are frequently reported in patients with TB–HIV coinfection and can be exacerbated by anti-TB treatment as well as systemic inflammatory responses. Hypokalemia also plays a critical role in the pathogenesis of cardiac arrhythmias and can significantly worsen morbidity (Adebimpe et al., 2015). Atrial fibrillation (AF), although rare in young adults, may arise from systemic inflammation, electrolyte imbalance, and lung disease. Pneumonia and hypokalemia are both risk factors for AF because they can trigger atrial electrical instability and structural remodeling of the heart (Corrales-Medina et al., 2018). In addition, DM itself contributes to AF risk through mechanisms such as cardiac fibrosis, autonomic dysfunction, and metabolic stress (Aune et al., 2018).

Taking into account these interrelated factors, a young male patient with newly diagnosed HIV, DM, clinical pulmonary TB, pneumonia, hypokalemia, and AF represents a rare but important clinical condition that underscores the necessity of early detection and comprehensive multidisciplinary management. Previous studies have highlighted the clinical complexity of TB when coexisting with HIV and DM. For instance, Sama et al. (2023) reported that among 1,115 pulmonary TB patients in Cameroon, 38.6% had TB–HIV coinfection and 5.8% had *TB/HIV/DM comorbidities*, emphasizing the epidemiological burden but without addressing the deeper pathophysiological interactions or complications such as electrolyte imbalance and arrhythmias. Similarly, Khalil et al. (2021) described severe hypokalemia as a rare manifestation in disseminated pulmonary TB, proposing tubular dysfunction as a possible mechanism, yet their single case report did not explore how combined TB/HIV/DM *comorbidities* may exacerbate such metabolic disturbances or contribute to AF.

The objective of this research is to describe the clinical manifestations, underlying mechanisms, and therapeutic considerations in such a complex case, while its significance lies in advancing understanding of the interplay between infectious diseases, metabolic dysfunction, and cardiac complications, ultimately reinforcing the importance of early detection and multidisciplinary management strategies.

RESEARCH METHOD

Case Report

A 25-year-old man presented to the emergency room of Arifin Achmad Hospital with complaints of shortness of breath for one day prior to *SMRS*. The shortness of breath was severe and accompanied by cough, unrelated to weather, dust, or food. The patient also reported coughing for two months, producing yellowish-white sputum. A low-grade fever had occurred two months earlier but never reached high temperature, and had since subsided. The patient experienced loss of appetite, weight loss from 80 kg to 65 kg, and night sweats without physical activity. *Mencret* was denied, and there was no nausea or vomiting. The patient had a history of pulmonary embolism and diabetes mellitus with insulin use five years earlier. He worked as an *admin cargo*, reported a history of sexual relations through anal *sodomy*, and was unmarried. There was no family history of similar illness.

On examination, the patient appeared moderately ill but was cooperative and conscious. Vital signs were blood pressure 109/77 mmHg, pulse 87 beats per minute, respiratory rate 22 breaths per minute, temperature 36.7 °C, and oxygen saturation (SpO_2) 95% on room air,

increasing to 98% with nasal cannula 3 L/min. His height was 158 cm, weight 65 kg, and body mass index (BMI) 31, consistent with level II obesity. Oral examination revealed candidiasis (+).

Physical examination showed pale conjunctiva, non-icteric sclera, positive light reflexes (+/+), oral candidiasis (+), absence of dry mucosa, no pale lips, no atrophic papillae, and no bleeding gums. Other systemic examinations were unremarkable. Laboratory results revealed Hb 10.8 g/dL, leukocytes 8,280/µL, platelets 296,000/µL, erythrocytes 3,570,000/µL, hematocrit 31.9%, MCH 30.3 pg, MCV 89.4 fL, MCHC 33.9 g/dL, random blood glucose (GDS) 162 mg/dL, sodium (Na^+) 142 mmol/L, potassium (K^+) 2.7 mmol/L, chloride 100 mmol/L, urea 25.7 mg/dL, creatinine 0.72 mg/dL, CA 19-9 12.01 U/mL, D-dimer 0.57 µg/mL, basophils 0.1%, eosinophils 0.8%, neutrophils 86.9%, lymphocytes 6.6%, monocytes 5.6%, albumin 2.9 g/dL, AST 44 U/L, ALT 56 U/L, HbA1c 8.4%, total cholesterol 203 mg/dL, HDL 27 mg/dL, LDL 143 mg/dL, triglycerides 165 mg/dL. Hepatitis B surface antigen (HBsAg) was non-reactive, while qualitative HIV testing was reactive in R1, R2, and R3. Additional values included sodium (Na^+) 137 mmol/L, potassium (K^+) 2.7 mmol/L, calcium 1.04 mmol/L, lactate 0.90 mmol/L, pH 7.47, pCO_2 46 mmHg, pO_2 146 mmHg, HCO_3^- 34 mmol/L, TCO_2 34 mmol/L, base excess (BE) 10, and SO_2c 99%. RT-PCR for SARS-CoV-2 was negative. Electrocardiogram (ECG) demonstrated atrial fibrillation with normal ventricular response.

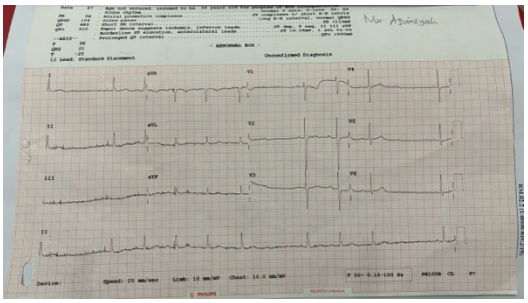


Figure 1. ECG

In the thorax photo, casts were found within normal limits and the pulm had susp bronchitis. Pneumonia.

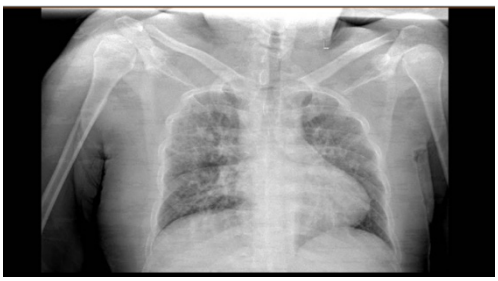


Figure 2. Photo Thorax

CT scan of the thorax without and with contrast was obtained leading to a picture of pneumonia and multiple lymphadenopathy in the bilateral and subcarinal paratracheal.

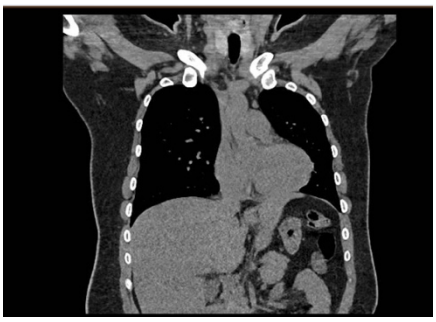


Figure 3. Ct Scan Thorax without and with contrast

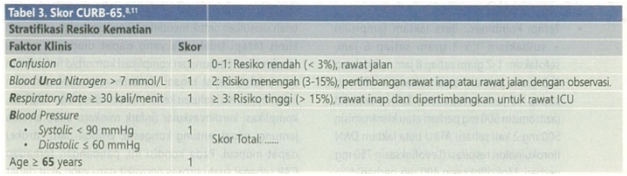
Based on the anamnesis, physical examination and examination of the patient was diagnosed as Community Acquired Pneumonia (CAP) + clinical pulmonary TB new cases with HIV status not yet ARV+ HIV AIDS Stage 3 + AF NVR + Hypokalemia + DM Type 2.

Patients received O2 therapy 3 lpm NK, IVFD NACL 0.9 % 500 cc/8 hours, inf. Lovofloxacin 1 x 750 mg, drip resfar 1 x 2.5 grams, Paracetamol infusion 3 x 1 grams, inj. Omeprazole 2 x 40 mg, KCL Correction 25 mEq, OAT FDC 1 x 4 tab, atorvastatin 1 x 20 mg, nystatin 4 x 1 iu, sitagliptin 1 x 10 mg, cotrimoxazole tab 1 x 960 mg, simarc 1x1 tab.

RESULT AND DISCUSSION

Pneumonia is a form of respiratory infection of the lung parenchyma. In healthy individuals, the alveoli are filled with air, while in pneumonia the alveoli are filled with fluid (Perhimpunan Dokter Spesialis Penyakit Dalam Indonesia [PAPDI], 2025). These patients came with complaints of shortness of breath since 1 day, severe shortness of breath accompanied by cough, shortness of breath not affected by weather, dust and food. The patient also complained of coughing since 2 months, coughing with yellowish-white phlegm and a fever that disappeared. On the physical examination, RR was obtained 22 x/min and other physical examinations there were no abnormalities. In the laboratory results, leukocytes were 8,280 uL, platelets 296,000 uL and CRP 377.92. CRP is a protein secreted by hepatic cells in response to an increase in the cytokines interleukin-6, interleukin-1B and TNF-a. CRP is the lymphocytes, monocytes, neurons and plaques of atherosclerosis and CRP levels increase with each inflammatory process. On thorax photon, it was found to be leading to pneumonia and CT scan of the thorax without and with contrast it was found to indicate pneumonia. In this patient, it is classified as community pneumonia (CAP) because it is <= 48 hours and is not obtained in the hospital. History of hospitalization in patients in April with acute bronchitis and pulmonary embolism. Risk factors for pneumonia in this patient include a history of type 2 DM, and immunocompromise. Overall, streptococcus pneumoniae (pneumococcus) is the most frequent pathogen in CAP. However, in this patient, a staining examination of Gram Sputum contaminated with saliva has been carried out, (the quality of the specimen is not good). The morphology of Gram-Positive and Gram-Negative Bacillus bacteria is the normal flora of the oral cavity and upper respiratory tract that was taken during sampling. The morphology of Gram-Negative bacillus bacteria is impressive and has not been convincing as a pathogen causing infection. In patients with a diagnosis of CAP, an important step in management is to determine the severity of the disease based on clinical assessment and scoring systems, namely

PSI/PORT score or CUR-65. In these patients, a CURB-65 score is used, which is another alternative that is more widely used by clinicians because it is easy to use.



Tabel 3. Skor CURB-65.[8,11]		
Stratifikasi Resiko Kematian		
Faktor Klinis	Skor	
Confusion	1	0-1: Resiko rendah (< 3%), rawat jalan
Blood Urea Nitrogen > 7 mmol/L	1	2: Risiko menengah (3-15%), pertimbangan rawat inap atau rawat jalan dengan observasi.
Respiratory Rate ≥ 30 kali/menit	1	≥ 3: Risiko tinggi (> 15%), rawat inap dan dipertimbangkan untuk rawat ICU
Blood Pressure • *Systolic* < 90 mmHg • *Diastolic* ≤ 60 mmHg	1	Skor Total: .....
Age ≥ **65** years	1	

Figure 4. Curb Score-658

In patients, a total score of 1 was obtained, namely urea 25.7 (> 7 mmol/l or . 20 m/dL with outpatient indications. However, in this patient, they are still treated because they have a history of other comorbidities such as atrial fibrillation, hypokalemia, new clinical pulmonary TB and HIV. This patient was given 3L/hour of cannulal nasal oxygen therapy, Inf. Lovefloxacin 1 x 750 mg, drip resfar 1 x 2.5 grams in NACL 0.9 % 100 cc exhausted in 4 hours, and paracetamol infusion of 3 x 1 gram (if fever). This is in accordance with the theory that patients with moderate pneumonia without risk factors Methicillin - Resistant Staphylococcus Aureus (MRSA) or Pseudomonas Aeruginosa receive inf monotherapy. Levofloxation 1 x 750 mg per day.

Tuberculosis is an infectious disease caused by infection with the Mycobacterium tuberculosis complex. Clinically diagnosed TB patients are TB patients who do not meet the criteria for bacteriological diagnosis, but based on other strong evidence, they are still diagnosed and managed as TB by the treating doctor. Included in the classification are BTA negative pulmonary TB patients with thoracic photo examination results supporting TB, BTA negative pulmonary TB patients with no clinical improvement after being given non-OAT antibiotics and having TB risk factors, extrapulmonary TB patients who are clinically or laboratory-diagnosed and histopathological without bacteriological confirmation. New cases of TB are cases who have never received anti-tuberculosis drugs (OAT) or have swallowed OAT with a total dose of less than 28 days (PDPI, 2021). In this patient, a new case of clinical pulmonary TB is evidenced from the anamnesis of cough with phlegm >= 2 weeks, shortness of breath, weight loss, decreased appetite, subfebris fever for more than 1 month, Sweating at night without physical activity. There were no abnormalities in the physical examination. At the supporting examination in the form of BTA negative sputum, Ronsen Thorax was found to have pneumonia bronchitis susp. Pneumonia CT scan of the thorax found multiple lymphadenopatic diaphagea bilateral and subcarinal. Bacteriological examination in ODHA often gives negative results on microscopic sputum, so the enforcement of TB diagnosis is prioritized using the Molecular Rapid Test (TCM). The picture of the thorax in ODHA is also not specific, especially in advanced statidums. Patients are given OAT 4 FDC 1 x 4 tabs. Within 8 weeks of giving OAT, ARV (anti-retro virus) administration was continued. The patient also has a history of DM, but the OAT guidelines and duration of treatment are in principle the same as TB without DM, provided that blood sugar levels are controlled.

The problem of HIV/AIDS is a major problem that threatens Indonesia and many countries in the world. UNAIDS, the WHO agency that deals with AIDS, estimates the number

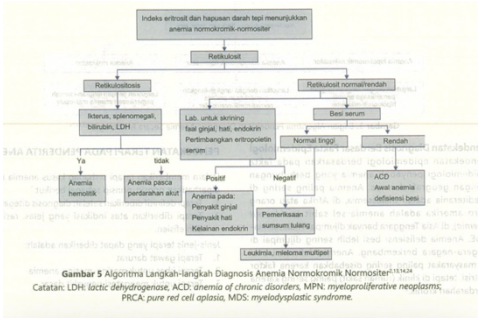
of people with HIV/AIDS (ODHA) worldwide at the end of 2021 to be 38.9 million orgs. Acquired Immunodeficiency Syndrome (AIDS) can be interpreted as a collection of symptoms or diseases caused by decreased immunity due to infection by the Human Immunodeficiency Virus (HIV) which belongs to the family retroviridae.8 This patient is included in stage 3 due to a weight loss of > 10 kg, oral candidiasis and pulmonary tuberculosis. However, this patient has not been given a tearapi regimen, because the patient also has a new case of clinical pulmonary TB. Patients were given cotrimoxazole prophylaxis 960 mg/24 hours until clinically stable. Pulmonary TB medication is given first and then continued with ARV as soon as possible within the first 8 weeks of TB treatment. In a state of immunosuppression (CD4 <50 cells/micro L) should be received ARV therapy within the first 2 weeks of TB treatment. The preferred regimen of HIV with TB coinfection is TDF + 3TC + EFV. In the patient, there is oral candidiasis so that the patient is given therapy in the form of 4 x 10 drops of suspension nystatin for 7-14 days.

Diabetes mellitus is a group of metabolic diseases with hyperglycemia characteristics that occur due to abnormalities in insulin secretion, insulin action or both. Insulin resistance in muscle cells and liver, as well as pancreatic beta cell failure which is the central damage pathophysiology of type 2 DM. The criteria for diagnosis of Diabetes mellitus are fasting plasma glucose examination >= 126 mg/dL or plasma glucose >= 200 mg/dL 2 hours after TTGO with a load of 75 grams. Or plasma glucose when >= 300 mg/dL with classic symptoms (polyuria, polydipsia, polyphagia and unexplained weight loss) or HBAIC examination >=6.5%.10 In patients there was a history of insulin use 5 years ago and in the HBAIC examination it was found to be 8.4%, then the patient was diagnosed as type 2 DM. The comparative diagnosis of type 2 diabetes in this patient is Maturity Onset Diabetes of the Young (MODY), which is a group of autosomal dominant monogenic diabetes characterized by early onset <25 years, usually without signs of autoimmune or obesity and derived from a single mutation of a gene that interferes with pancreatic B cell function. Diagnosis of MODY with onset before 25 years of age but can appear up to 35-50 years of age, autosomal inheritance with a family history of similar diseases, no autoantibodies (GAD, IA-2), detectable C-Peptides, mild to moderate hyperglycemia and metabolic signs. As with obesity, insulin resistance is usually absent.11 This patient was 25 years old and diagnosed with DM at the age of 20, but this patient did not have GAD, IA-2 and C peptide tests so it was not possible to rule out whether the patient was type 2 or MODY DM. Therapy in this patient was given an inhibitor group of the enzyme dipeptidyl peptidase-4 (DPP-4), namely sitagliptin tab 25-100 mg. DPP-4 is a serine protease that is widely distributed in the body. DPP-4 inhibitors inhibit the binding site of DPP-4 so that it will prevent inactivation of glucagon-like peptide (GLP)-1. This inhibition process maintains GLP-1 and glucose-dependent insulinotropic polypeptide (GIP) levels in the circulating active form, thereby improving glucose tolerance, improving insulin response and reducing glucagon secretion (Deacon, 2019). Sitagliptin is safe to administer to patients with HIV comorbidities, pneumonia, clinical pulmonary TB, and atrial fibrillation. In general, DPP-4 inhibitors such as sitagliptin do not increase the risk of severe infection (pneumonia) (Chen et al., 2021). However, one Taiwanese cohort study reported that high doses of DPP-4 were associated with a higher risk of TB infection (Dzhemileva et al., 2025).

Dyslipidemia is a lipid metabolism disorder characterized by an increase or decrease in lipid fractions in plasma. The main lipid fraction abnormalities are an increase in total cholesterol levels, Low-Density Lipoprotein (LDL), Triglycerides (TG), and a decrease in High Density Lipoprotein (HDL). In this patient, the total cholesterol results were 203 mg/dL, (borderline/slightly high), HDL 27 (low), LDL 143 (borderline/slightly high), triglycerides 165 (borderline/slightly high). In these patients, there is a risk of obesity, diabetes, and having heart disease in the form of atrial fibrillation. Non-pharmacological management is in the form of physical activity and weight loss. Pharmacological management in this patient was given atorvastatin 1 x 20 mg. Statins inhibit cholesterol biosynthesis, increase LDL receptor regulation in the liver, increase LDL clearance, and decrease lipoprotein secretion from the liver, thereby lowering LDL production. Statins are the main pillars of governance. Dyslipidemia and. Useful for all types of hyperlipoproteinemia with elevated LDL (Adam & Wildan, 2025). Dyslipidemia in DM generally increases TG and LDL and lowers HDL called atherogenic patterns, which will accelerate cardiovascular risk. In addition, it also increases inflammatory stress which will worsen insulin resistance. The association of dyslipidemia with HIV therapy found a prevalence of 69% with 39-40% experiencing an increase in LDL/TG and a 36% decrease in HDL. Meanwhile, in pneumonia, according to US longitudinal data, as many as > 20 people who were followed up for 20 years showed that low HDL and high TG were associated with an increased risk of hospitalization due to pneumonia. In addition, in severe pneumonia, there is a decrease in HDl as a response to severe inflammation. The association of dyslipidemia with atrial fibrillation according to studies that low HDL and metabolic syndrome increase the risk of atrial fibrillation (Brake et al., 2025; Belete et al., 2024; Baluku et al., 2024).

Atrial non-valvular fibrillation (AF NVR) is atrial fibrillation in patients without rheumatic heart valve disease. This type is most common with a prevalence of > 50 million per case. These cases increase with aging, hypertension, obesity, DM, systemic inflammation. This AF NVR arises as a result of atrial remodeling caused by pressure, fibrosis, inflammation and atrial electrical irritation. This AF NVR has a 5 times higher risk of stroke. The characteristics of AF NVR on an ECG are fibrillation waves (350-600 bpm), irregular interval R, and a narrow QRS complex without the usual P waves. The 2023 ACC/AHA/HRS guide emphasizes lifestyle modification, early detection and a multidisciplinary approach. In this patient simarc (warfarin) 1x1 tabs were given. This is in accordance with the theory that walfarin is a vitamin K antagonist that prevents the formation of clotting factors II, VII, IX and X. Walfarin is effective in reducing the risk of stroke by 64% and mortality by 26% in AF patients (Sagris et al., 2022).

In this patient, normochrome moderate anemia is obtained. Reticulocytes are important to be examined to find the etiology of anemia in this patient, but in this patient no reticulocyte examination is performed (Bae et al., 2021).



Gambar 5 Algoritma Langkah-langkah Diagnosis Anemia Normokromik Normositer[1,13,14,24]

Catatan: LDH: *lactic dehydrogenase*, ACD: *anemia of chronic disorders*, MPN: *myeloproliferative neoplasms*; PRCA: *pure red cell aplasia*, MDS: *myelodysplastic syndrome*.

Figure 9. Algorithm diagnosis of anemia normocytic normochrome.8

Potassium in this patient is 2.7 mmol/L which is included in moderate hypokalemia (2.3 - 3.0 mmol/L). The patient had no history of diuretic consumption, vomiting, diarrhea.22 However, the patient had a low food intake. The patient was given therapy with a correction of KCL 25 mEq dissolved in 100 cc of NACL at a rate of 10 meq/hour and then potassium rose to 2.8 mmol/L. Then the patient was given oral therapy in the form of KSR tab 3 x 600 mg. Hypokalemia (<3.5 mmol/L) was found in 20% of inpatients and was significantly associated with an increase in NVR AF. A cohort study in Rotterdam revealed that hypokalemia increases the risk of AF. Hypokalemia triggers arrhythmias through two mechanisms, namely prolonging the duration of potential action, weakening repolarization and increasing after depolarization and as an inhibition of Na+/K+-ATPase (Pukenyte et al., 2023). Both of these mechanisms cause changes in atrial conduction and facilitate the triggered activity phase in contributing to NVR AF.

In HIV-positive patients with TB on OAT and in these patients, AST 44 U/L (10-40) ALT 56 U/L (10-40) is advised with caution because it can cause the phenomenon of DILI due to a combination of toxic metabolites from OAT, HUV inflammation, malnutrition and genetic predisposition. Therefore, management strategies are needed including monitoring transaminase, improving nutritional status, and stopping OAT (Kim et al., 2023).

This patient had a 3-month history of pulmonary embolism and was treated at Arifin Achmad Hospital with good cardiac function echocardiography, EF 90%. Normokinetic D-Dimer 0.57 means that D-Dimer reflects the ongoing process of clot formation and destruction. In pulmonary thromboembolism I was characterized by simultaneous activation of coagulation and fibrinolysis in the lungs (Vyas et al., 2024). The plasmin then breaks down the clot fibrin tissue in the pulmonary artery and produces circulating D-dimers. Thus the rise of the D-dimer directly reflects the presence of an active clot and its destruction (Thu Kyaw & Maung, 2022).

On the 6th day of treatment, the patient had type 1 respiratory failure with PH/PCO2/PO2/ 7.31/36/32. Type 1 respiratory failure is characterized by PaO2 <60 mmHg and normal or low PaCO2, which reflects a failure of oxygenation with adequate ventilation. There is a difference between ventilation and alveolar perfusion that causes the lungs to not get enough oxygen even though the blood flow is good (physiological shunt). This occurs in the conditions of pneumonia, pulmonary edema and Acute Respiratory Distress Syndrome (ARDS).

ARDS is an extreme form of type 1 respiratory failure with pathophysiological first activation of alveolar macrophages so as to cause the release of inflammatory cytokines and then to make neutrophil recruitment so as to cause damage to the alveolar epithelium and endothelial and the formation of edema. Second, it causes lung volume and compliance to decrease so that many alveoli collapse (atelectasis) and there is a heterogeneity of ventilation/perfusion, causing more severe oxygen diffusion and persistent hypoxemia. This patient was given 15 lpm NRM oxygen therapy. According to acute care and ICU in 2024 for severe hypoxemia, immediately use NRM with flow >= 15 lpm to achieve the > saturation target of 92%.

These patients also lead to Pneumocystis Jirovecii Pneumonia (PCP). PCP is caused by pneumocystis jiroveci, which is an opportunistic fungus that is asymptomatic in the lungs. In HIV patients with CD4 <400 cells/mL, especially <200 cells/mL, cellular immunity is weakened, resulting in over proliferation and extensive damage. In the alveoli so that it causes hypoxia and gas exchange disorders. In addition, disturbed macrophages and B cells exacerbate the infection. Population studies suggest that PCP mortality remains significant especially in patients with a late diagnosis of HIV. In Tongkok, the prevalence of PCP in HIV is 8-26% with a high mortality rate. Clinical symptoms are high fever, dry cough, chest pain during inspiration and progressive dyspnea. Supporting examination found on the thoracic photo there was a bilateral infiltrate in the perihiller area, sometimes solitary or multiple nodules were found, CT-Scan showed a picture of ground glass, in the serum lab 1-3 beta-D-glucan was positive, LED increased. LDH rises, sputum examination found pneumocystis Jiroveci, PCR, cyst wall painting Grocott Gomori methenamine silver, Gram-Weigert, cresyl violet or toluidine blue. Therapy in PCP patients was given Trimethoprim-Sulfamethoxazole (TMP-SMX)/cotrimoxsazole 15-20 mg/kg/day divided into 3-4 doses per day for 14-21 days.28 In severe cases, prednisone was added 2 x 40 mg for the first 5 days, 1 x 40 mg 5 days later followed by 20 mg/day until completion. In this patient, cotrimoxazole therapy was given a tab of 2 x 960 mg. This patient should have been given 4 x 960 mg. So that in this patient it becomes stage 4 HIV/AIDS with rapid worsening progression.

In this case report, a patient aged 25 years with multiple comorbidities consisting of HIV/AIDS, CAP, clinical pulmonary tuberculosis on OAT, Dm type 2, hypokalemia and atrial non-valvular fibrillation. With this young age, it is important to have early detection and comprehensive multidisciplinary treatment. Treatment of these patients requires disciplinary teams from anesthesia, pulmonology, internal, cardiology and pharmacy to ensure comprehensive and multidisciplinary management to optimize clinical outcomes. Early detection as an interaction and complication is the key for therapy to run effectively and safely.

CONCLUSION

This case report describes a 25-year-old male patient with HIV/AIDS and diabetes mellitus who was newly diagnosed with pulmonary tuberculosis (TB), pneumonia, hypokalemia, and atrial fibrillation, representing a rare and complex clinical presentation. The patient was initially managed with oxygen therapy, intravenous infusions, and medications to stabilize his condition. However, despite early interventions, he developed severe

complications, including community-acquired pneumonia, profound hypokalemia, and type 1 respiratory failure. These complications highlighted the need for a multidisciplinary team—comprising anesthesiologists, pulmonologists, internists, cardiologists, and pharmacists—to design a comprehensive treatment plan aligned with clinical practice guidelines.

During hospitalization, the patient's condition continued to deteriorate, with persistent shortness of breath, cough, fever, and marked weight loss. Laboratory findings indicated ongoing infection with persistent hypokalemia, while thoracic CT scans demonstrated pneumonia and lymphadenopathy. Despite therapeutic efforts—including the administration of antibiotics, antidiabetic agents, and strict clinical monitoring—the patient ultimately suffered respiratory and cardiac arrest, resulting in death. The family's request for *Do Not Resuscitate (DNR)* marked the end of his treatment course, and the patient was pronounced dead in the presence of his family and the ICU nursing team.

This case underscores the complexity of managing patients with HIV/AIDS and multiple severe *comorbidities*, and reinforces the importance of early detection, holistic care, and coordinated multidisciplinary approaches to optimize outcomes in such high-risk clinical scenarios.

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