

Case Report

Severe hypoalbuminemia in patient with pulmonary edema and pleural effusion: a case report

Martika Esty Wulandary*, Aditya Azwar Sofandi

Department of Interna, Bima Regional General Hospital, West Nusa Tenggara, Indonesia

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*Correspondence:

Dr. Martika Esty Wulandary,

E-mail: ulanmartika@gmail.com

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ABSTRACT

Hypoalbuminemia is a recognized risk factor for mortality and poor outcomes in various clinical contexts. This case report aims to discuss the case and management of severe hypoalbuminemia in a 30-year-old female patient with pulmonary edema and pleural effusion at RSUD Bima. A 30-year-old female with a history of tuberculosis was admitted with worsening shortness of breath, nosebleed, abdominal and breast enlargement, hair loss over the past 2 months, tired, joint pain, swelling in both legs, ulcers on the oral mucosa, and butterfly rash. She exhibited signs of anaemia, hypoalbuminemia, and ascites. X-ray thorax showed cardiomegaly, pulmonary edema, and bilateral pleural effusion. Diagnoses included dyspnea due to pulmonary edema, severe hypoalbuminemia, anaemia, thrombocytopenia, and suspected SLE. Treatment involved oxygen, IV octalbin 25% 100cc, medications, and an extra protein diet. After three days, her albumin levels improved. Hypoalbuminemia can cause pleural effusion by reducing oncotic pressure, leading to fluid buildup. In SLE, it may result from inflammation, malnutrition, or protein loss. Management includes addressing causes, nutritional support, and careful albumin administration.

Keywords: Hypoalbuminemia, Pulmonary edema, Pleural effusion, Systemic lupus erithematosus

INTRODUCTION

Hypoalbuminemia in adults is defined as an intravascular albumin level below 3.5 g/dl, while in children under three years, it is defined as below 2.9 g/dl. The condition can result from decreased synthesis (due to liver disease or protein malnutrition), increased tissue catabolism (such as in sepsis), renal loss (nephrotic syndrome), gastrointestinal loss (protein-losing enteropathy), or changes in distribution (sequestration).^{1,2} Hypoalbuminemia is frequently observed in clinical practice, especially in severely ill or malnourished patients, and serum albumin levels are routinely measured. It can develop rapidly in acute disease or after trauma and resuscitation in previously healthy individuals and is also seen in chronic inflammatory diseases despite adequate nutrition intake. However, it is often mistakenly viewed as an indicator of poor nutrition intake that can be corrected solely through nutritional support.³ Hypoalbuminemia is a recognized

risk factor for mortality and poor outcomes in various clinical contexts, including wellness promotion, acute hospitalization, end-stage renal disease (ESRD), and decompensated heart failure. In surgical patients, it is associated with the need for reoperation, prolonged hospital stays, wound complications, renal failure, gastrointestinal dysfunction, and mortality.⁴

Serum albumin constitutes about 80% of the colloid oncotic pressure in the circulation. Reduced serum albumin levels (hypoalbuminemia) and the subsequent decrease in colloid osmotic pressure are significant features of critical illness. This reduction in oncotic pressure can contribute to the formation of pulmonary edema by decreasing the forces that retain fluid in the microvasculature.^{5,6} This case report aims to discuss the case and management of severe hypoalbuminemia in a 30-year-old female patient with pulmonary edema and pleural effusion at RSUD Bima.

CASE REPORT

A 30-year-old female patient was referred from Puskesmas to the emergency department of RSUD Bima due to worsening shortness of breath over the past week. The patient experienced a nosebleed early this morning (active bleeding), progressive abdominal enlargement over the past 3 months, breast enlargement with a lump in the umbilical area, hair loss over the past 2 months, tired, joint pain, swelling in both legs, ulcers on the oral mucosa, and butterfly rash. She has had a cough for the past 3 months with sputum production and a history of tuberculosis 3 months ago. Her appetite has decreased, and she feels she has lost weight. She has a history of hospitalization in January and a history of blood transfusion.

Upon examination, the patient had a glasgow coma scale score of 15. Her blood pressure was 140/110 mmHg, heart rate was 82 beats per minute, respiratory rate was 30 breaths per minute, body temperature was 36.5°C, and oxygen saturation was 90%. The patient's face appeared swollen, she was non-icteric, and anaemic. Bilateral basal crackles were heard in the lungs. The heart had a regular rhythm with no murmurs. In the abdomen, a soft, mobile tumor was palpable in the umbilical area with tenderness, and ascites was present. Both legs showed pitting edema, and urine output was less than 100 ml.

Lab results are shown in table 1. Routine blood work results showed high WBC, low HGB, low HCT, high neutrophil count. Electrolyte count showed high chloride levels. Albumin levels were severely decreased.



Figure 1: X-ray thorax showing cardiomegaly, pulmonary edema, and bilateral pleural effusion.

X-ray thorax results showed cardiomegaly, pulmonary edema, and bilateral pleural effusion (Figure 1). The patient was diagnosed with the conditions like dyspnea due to pulmonary edema, severe hypoalbuminemia with

ascites, anaemia, thrombocytopenia; and suspected SLE (Systemic Lupus Erythematosus).

The treatment plan included administering oxygen via mask at 6 liters per minute, IV fluids (Ringer's Lactate), IV furosemide 20 mg three times daily, IV ceftriaxone 1 g twice daily with a skin test, IV ranitidine 50 mg twice daily, IV paracetamol 1 g three times daily, IV ondansetron 4 mg three times daily, IV tranexamic acid 500 mg, and IV octalbin 25% 100cc. Additionally, the patient was advised to follow an extra protein diet with extra egg whites. Follow up on the patient showed improvements in albumin levels after four days treatment in the hospital (Table 2).

Table 1: Laboratorium analysis.

Analysis	Results	Reference value
Hematology		
WBC	↑11.710×10 ³ /μl	4.50-11.00×10 ³ /μl
HGB	↓7.7 g/dl	11.0-16.0 g/dl
HCT	↓22%	37.0-54.0 %
Neutrophil count	↓8.3×10 ³ /μl	138-146×10 ³ /μl
Electrolyte		
Chloride (Cl)	↑112 mmol/l	98-100 mmol/l
Albumin	↓1.3 g/dp	3.5-5.2 g/dp

Table 2. Albumin levels improvement.

Analysis	Albumin levels	Reference value
Admission		
4/7/2024	1.3 g/dp	3.5 – 5.2 g/dp
Follow up		
6/7/2024	↑ 2.0 g/dp	3.5 – 5.2 g/dp
8/7/2024	↑ 3.5 g/dp	3.5 – 5.2 g/dp

DISCUSSION

Hypoalbuminemia can cause pleural effusion, particularly transudative pleural effusion. This condition is recognized as a potential cause of pleural effusion, particularly of the transudative type. This condition involves a low serum albumin level, which can lead to fluid accumulation in the pleural space due to decreased oncotic pressure in the blood vessels. Although hypoalbuminemia is associated with pleural effusion, it is considered a less common cause compared to other factors.^{7,8}

A 1999 study by Eid et al, confirmed hypoalbuminemia as a cause of pleural effusion, though it is considered rare because low serum albumin levels often occur alongside other potential diseases. Additional clinical evaluations are needed to determine the specific cause of pleural effusion in patients with hypoalbuminemia.^{9,10}

In this case report, the underlying cause of hypoalbuminemia are suspected to be systematic lupus erimathosus (SLE) and anemia. Bilateral pleural effusion,

which is the accumulation of fluid in the space between the lungs and chest wall on both sides, is a significant complication for patients with SLE.¹¹ In SLE, bilateral pleural effusion can arise from inflammation of the pleura, which is often due to the immune system's attack on healthy tissues, including the pleura. This inflammation can result in fluid leakage into the pleural space.¹²

Albumin is crucial for maintaining fluid balance and transporting essential substances like hormones and vitamins. Additionally, decreased appetite and difficulty eating due to SLE symptoms can lead to malnutrition and low protein intake, further contributing to hypoalbuminemia.¹³ Protein-losing enteropathy, a condition causing excessive protein loss through the intestines, may also be a factor due to chronic inflammation or damage.^{14,15}

The consequences of hypoalbuminemia in SLE patients are severe. Reduced albumin levels decrease osmotic pressure, leading to fluid leakage from blood vessels into the interstitial space, which causes edema in the legs, face, and abdomen (ascites).¹⁶ In this patient, ascites and general edematous state were observed. Hypoalbuminemia can also lead to hypotension due to decreased blood volume and disrupt liver, kidney, and immune system functions, increasing infection risk.¹⁷

Managing hypoalbuminemia involves treating the underlying causes. Addressing malnutrition with dietary counseling or supplements, and treating protein-losing enteropathy by targeting its root cause, is also essential.¹⁸ In this case, the patient was given intravenous and oral corticosteroid to manage the underlying inflammation and protein losing enteropathy. Dietary counselling was given with extra protein diet to increase the level of albumins.

Intravenous albumin administration can help increase serum albumin levels and mitigate hypoalbuminemia-related complications like edema and hypotension. However, this treatment must be carefully managed to avoid potential risks such as fluid overload or heart failure.¹⁹ Effective management of hypoalbuminemia in SLE requires a thorough understanding of its causes and a comprehensive approach, including medication and albumin replacement, to improve patient outcomes and quality of life.²⁰

CONCLUSION

Hypoalbuminemia can cause pleural effusion by reducing oncotic pressure, leading to fluid buildup. In SLE, it may result from inflammation, malnutrition, or protein loss. Management includes addressing causes, nutritional support, and careful albumin administration.

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