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SUDDEN FATAL ANAPHYLACTIC REACTION TO HUMAN ALBUMIN INFUSION IN A PATIENT WITH RHEUMATIC HEART DISEASE: A CASE REPORT
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SUDDEN FATAL ANAPHYLACTIC REACTION TO HUMAN ALBUMIN INFUSION IN A PATIENT WITH RHEUMATIC HEART DISEASE: A CASE REPORT

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SUMMARY

Human serum albumin (HSA) is widely used to manage critical conditions that need fine fluid homeostasis, such as hypoalbuminemia, cirrhosis, and heart failure. While generally safe, rare cases of hypersensitivity and anaphylaxis have been reported. We report a case of fatal anaphylaxis following HSA infusion in a 49-year-old woman with rheumatic heart disease, acute decompensated heart failure, and congestive hepatopathy. After initial infusion, she developed urticaria, abdominal pain, diarrhea, dyspnea, and hypotension, consistent with anaphylaxis. Despite stopping the infusion and treatment, a second administration led to a more severe reaction, resulting in cardiac arrest and death. Anaphylaxis related to albumin is extremely rare and can be difficult to diagnose in patients with underlying conditions. The mechanisms are not fully understood, but they may involve both IgE-mediated and non-IgE-mediated pathways. Treatment follows standard anaphylaxis protocols, with epinephrine as first-line therapy. No standardized protocols exist for albumin allergy, emphasizing the need for heightened clinical awareness. This case highlights the potential for fatal anaphylactic reactions to albumin, especially upon re-exposure, and underscores the importance of vigilance and avoidance of repeat administration after initial hypersensitivity.

INTRODUCTION

Human serum albumin (HSA) is the most abundant extravascular protein in human blood [1]. It has several essential physiological properties, including maintenance of oncotic pressure, transport of endogenous and exogenous molecules, acid-base balance, and inflammation [1]. Given these physiological functions, albumin is a cornerstone in managing critical conditions such as hypoalbuminemia, ascites, cirrhosis, nephrotic syndrome, heart failure, and others, where maintenance of fluid homeostasis is vital [1].

Although albumin is generally considered very safe, patients can occasionally develop serious hypersensitivity reactions or even life-threatening anaphylaxis. These reactions are rare but have been documented, demonstrating that albumin is not completely free from immunologic risk [2]. Data on the true rates of hypersensitivity and anaphylaxis are limited, but available reports suggest that such events are extremely uncommon.

We report a case of fatal anaphylaxis following HSA infusion in a patient with rheumatic heart disease, acute decompensated heart failure, and congestive hepatopathy. To our knowledge, this is the first report of such an outcome in the adult cardiac patient population.

CASE PRESENTATION

A 49-year-old Kenyan woman with a 16-year history of rheumatic heart disease (RHD) and right ventricular dysfunction presented on 19 October 2025 with generalized body swelling for one week. She reported progressive generalized oedema involving the abdomen, face, and lower limbs. She described marked abdominal distension and facial swelling with a sensation of "being choked." She had dyspnea on exertion (NYHA III), orthopnea, and paroxysmal nocturnal dyspnea. She denied a history of coughing, vomiting, and bleeding tendencies and reported normal urine output and bowel habits. She had experienced an upper respiratory tract infection approximately one month earlier, which was managed adequately with antibiotics.

Her past medical history included long-standing RHD with multiple prior admissions for acute decompensated heart failure (the most recent occurring in May 2025). That admission was precipitated by medication non-adherence and a concurrent upper respiratory tract infection, and her management included intravenous (IV) diuresis with Spironolactone and Furosemide and IV Levofloxacin. Her surgical history consisted of two previous mitral valve repair surgeries (2009 and 2016). A thorough review of her records confirmed she had never received blood products, colloid infusions, or human serum albumin during any prior admission or surgery. She was HIV-positive on antiretroviral therapy (Tenofovir disoproxil, Lamivudine, Dolutegravir). Her chronic medications included warfarin, carvedilol, enalapril, furosemide, spironolactone, and digoxin. She had no known drug allergies before this admission.

On examination, she appeared ill and in respiratory distress, with bilateral lower-limb oedema and facial puffiness. Jugular venous pressure was elevated. Cardiac auscultation revealed a grade III/VI pansystolic murmur at the apex (mitral area), a grade III/VI holosystolic murmur at the lower left sternal border (tricuspid area), and a grade III/VI diastolic murmur at the right upper sternal border (aortic area). Respiratory examination demonstrated bibasal crepitations. The

abdomen was moderately distended with shifting dullness on percussion, and deep palpation elicited marked tenderness in the right upper quadrant, where a firm liver edge was palpable. Neurological examination was unremarkable.

Laboratory investigations revealed hypoalbuminemia (22.9 g/L). Other results included Aspartate aminotransferase (AST): 39.9 U/L, Gamma-glutamyl transferase (GGT): 117 U/L, sodium 121 mmol/L, potassium 7.5 mmol/L, chloride 95.3 mmol/L, and urea 6.2 mmol/L. A prior abdominal ultrasound had shown congestive hepatopathy.

Given the severe hypoalbuminemia, congestive hepatopathy, and acute decompensated heart failure, she was given 100 mL of 20% human serum albumin (Albucel, India) (100 mL of 20% HSA twice daily for three days). Approximately fifteen minutes after the infusion began, she developed generalized urticaria, abdominal pain with diarrhea, dyspnea, and hypotension, consistent with an acute anaphylactic reaction. The infusion was immediately stopped, and hydrocortisone was administered, leading to temporary stabilization. Blood levels showed an elevated level of eosinophils in the blood.

A second course of albumin was planned and scheduled for 23 October 2025. Five days following the initial reaction, the infusion was initiated. Approximately ten minutes later, she developed difficulty breathing, diarrhea, generalized urticaria, and severe hypotension (BP 67/33 mmHg), with subsequent clinical deterioration.

One day after the second albumin infusion was attempted (24 October 2025), she became unresponsive with no palpable pulses. Cardiopulmonary resuscitation was initiated at 00:10 and continued for 20 minutes without return of spontaneous circulation. She was pronounced dead at 00:30, marking the third day of her hospital admission. The working diagnosis was fatal anaphylactic shock secondary to albumin infusion. Consent for publication was obtained from the next of kin.

DISCUSSION

Anaphylaxis is a severe, systemic hypersensitivity reaction characterized by the rapid onset of multisystem involvement and potential progression to cardiovascular collapse. According to the Ring and Messmer classification, anaphylactic shock corresponds to Grade III–IV reactions, defined by life-threatening bronchospasm, severe hypotension, or cardiac arrest [2]. Diagnosis is primarily clinical and is supported by the presence of acute mucocutaneous symptoms accompanied by respiratory compromise, gastrointestinal manifestations, or hemodynamic instability.

The most common clinical features of anaphylaxis include generalized urticaria, angioedema, dyspnea or wheezing, abdominal pain, diarrhea, vomiting, hypotension, syncope, and impaired consciousness. Our patient's presentation of generalized urticaria, respiratory distress, gastrointestinal symptoms, and profound hypotension fulfilled criteria for Ring and Messmer Grade IV anaphylaxis [3].

The mechanisms underlying HSA-induced anaphylaxis remain incompletely understood. Proposed pathways include IgE-mediated hypersensitivity to native or modified albumin proteins; non-IgE–mediated mast cell degranulation, potentially triggered by complement activation; hypersensitivity to stabilizers, preservatives, or manufacturing-related contaminants; and cross-reactivity with prior exposures to blood products. These mechanisms highlight that even a human-

derived protein can elicit significant immunologic reactions in sensitized or predisposed individuals [2], [4].

Diagnosis of albumin-induced anaphylaxis poses particular challenges. Early manifestations such as rash or mild hypotension may be attributed to the patient's underlying cardiac disease, volume overload, or sepsis. Furthermore, albumin is generally perceived as a safe therapeutic agent, which may delay recognition of hypersensitivity reactions. Although laboratory tests such as serum tryptase and eosinophil counts can support the diagnosis, they are not always readily available, and management must rely on immediate clinical judgment.

Management follows established international guidelines for anaphylaxis. Intramuscular epinephrine is the first-line treatment, followed by airway stabilization, high-flow oxygen, aggressive isotonic fluid resuscitation, and adjunctive corticosteroids and antihistamines.[3]. Severe reactions may require continuous epinephrine infusion, vasopressor support, and intensive care monitoring. Crucially, re-exposure to the inciting agent must be avoided, as subsequent reactions can be more rapid and severe, as was evident in this case[5].

Despite the widespread clinical use of albumin, no standardized protocols exist for diagnosing or managing confirmed albumin allergy. Literature is limited to isolated case reports, and no validated skin-testing kits, desensitization regimens, or predictive biomarkers have been established. As a result, management is guided by general principles of drug hypersensitivity, discontinuation of the offending agent, comprehensive documentation, and selection of alternative colloid or crystalloid solutions. Given the rarity of albumin-induced anaphylaxis, heightened clinical vigilance is warranted, especially in patients with multiple comorbidities or prior unexplained reactions to biologic products.

This case underscores the need for increased awareness of this rare but potentially fatal complication and highlights the importance of avoiding re-exposure once an initial reaction has occurred.

However, reactive management is insufficient. Prevention must begin at a policy level. We recommend hospitals develop institutional protocols for screening patients with a history of multiple surgeries, transfusions, or prior colloid exposure for potential sensitization. For high-risk patients with a documented but necessary indication for albumin, supervised desensitization protocols should be considered and readily available. Currently, no such standardized guidelines exist, highlighting a significant gap in patient safety frameworks.

CONCLUSION

Human serum albumin is an essential therapeutic agent for managing fluid imbalance in various critical illnesses. However, clinicians should be aware of the rare but potentially fatal risk of anaphylaxis associated with its use. Early recognition and prompt treatment of allergic reactions are vital, particularly in patients with complex cardiac conditions where symptoms may overlap. Importantly, once a hypersensitivity reaction to albumin has occurred, re-exposure must be avoided to prevent fatal outcomes. Increased awareness and careful monitoring during albumin infusion can help reduce the risk of such adverse events.

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