

Myocardial Dysfunction and Left Ventricular Thrombus in a Young Thalassemia Patient with Diabetic Ketoacidosis: A Rare Case Report

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Abstract

A 20-year-old male with a history of β -thalassemia major presented with altered sensorium, abdominal pain, and rapid breathing. He was recently diagnosed with diabetes mellitus, likely secondary to iron overload due to poor compliance with chelation therapy. On admission, he was found to be in diabetic ketoacidosis (DKA). During management, the patient developed recurrent supraventricular tachycardia (SVT) and hypotension. Cardiac evaluation revealed regional wall motion abnormalities in the anterolateral wall, left ventricular ejection fraction of 40%, elevated troponin I,

and a large apical thrombus measuring 2.5×1.5 cm. The patient subsequently went into cardiogenic shock with concurrent sepsis. Intensive care management included insulin infusion, electrolyte correction, inotropic support, anticoagulation, antiarrhythmic therapy, and broad-spectrum antibiotics. Gradual stabilization was achieved, with partial resolution of the thrombus and improved cardiac function on follow-up echocardiography. This case underscores the devastating impact of iron overload and non-compliance to chelation in thalassemia patients, leading to multi-organ complications including endocrine dysfunction, DKA, and severe cardiac disease.

Early recognition and multidisciplinary management are vital to improve outcomes in such complex scenarios.

Keywords: Diabetic Ketoacidosis, Endocrine Dysfunction, Hyperglycemia

Introduction

Thalassemia major is a hereditary hemoglobinopathy requiring lifelong transfusion support. Chronic transfusions, if inadequately managed with iron chelation therapy, result in systemic iron overload, predisposing patients to cardiac, hepatic, and endocrine dysfunctions^{1,2}. Cardiac involvement, particularly cardiomyopathy, arrhythmias, and thromboembolic events, remains the leading cause of mortality in thalassemia³.

Secondary diabetes mellitus occurs in approximately 20–30% of thalassemia patients, resulting from pancreatic siderosis⁴. The coexistence of diabetes-related metabolic crises such as diabetic ketoacidosis (DKA) with iron-induced cardiomyopathy is rare but potentially fatal. Here, we present a case of a young male with thalassemia major, poorly compliant with chelation therapy, who developed DKA complicated by myocardial infarction, arrhythmias, left ventricular thrombus, and sepsis.

Case Presentation

A 20-year-old male, known case of β -thalassemia major, presented with abdominal pain, vomiting, and altered sensorium. He was transfusion-dependent since childhood and had a history of poor compliance with desferrioxamine therapy. Recently diagnosed with diabetes mellitus, he was not on regular treatment. On admission, he was tachypneic with Kussmaul's respiration, hypotensive, and drowsy. Laboratory investigations revealed hyperglycemia (478 mg/dL),

ketonuria, and high anion gap metabolic acidosis, confirming DKA.

During treatment with insulin and IV fluids, the patient developed recurrent SVT and worsening hemodynamic instability. Troponin I was elevated.

Echocardiography Findings

2D Echocardiography Findings:

- Mild concentric LVH
- Regional wall motion abnormality: Septo-apical, apical, and distal anterolateral wall hypokinesia
- Moderate LV systolic dysfunction, EF 40%
- Grade I LVDD
- No MS/Mild MR
- No AS/AR
- Moderate TR (mild pulmonary artery hypertension)
- LV apical clot measuring 2.5×1.5 cm

Blood counts showed leukocytosis, and cultures suggested sepsis.

Management included ICU admission, insulin infusion, fluid and electrolyte correction, antiarrhythmic therapy, inotropes for cardiogenic shock, anticoagulation (LMWH), and broad-spectrum antibiotics. Iron chelation therapy was restarted.

Over one week, the patient stabilized with partial thrombus resolution and improved cardiac function on repeat echocardiography.

Discussion

Iron overload cardiomyopathy is the most lethal complication of thalassemia major⁵. Excess cardiac iron deposition leads to conduction abnormalities, arrhythmias, cardiomyopathy, and predisposition to intracardiac thrombus formation^{6,7}.

The occurrence of DKA in thalassemia patients is attributed to pancreatic siderosis, which impairs β -cell function⁸. DKA itself exerts additional stress on an

already compromised myocardium through electrolyte imbalances and catecholamine surges, precipitating arrhythmias and cardiac dysfunction.

In our case, poor chelation compliance led to a cascade of complications—secondary diabetes, DKA, arrhythmias, myocardial infarction, LV thrombus, and sepsis. Such a constellation in a young adult is rare but carries significant mortality risk.

Management is highly challenging and requires a multidisciplinary approach involving intensivists, cardiologists, endocrinologists, and hematologists. This case highlights the need for strict chelation adherence, regular screening for endocrine and cardiac complications, and high clinical suspicion when thalassemia patients present with acute metabolic or cardiac symptoms.

Key Takeaways

- Iron overload in thalassemia can lead to diabetes, cardiomyopathy, arrhythmias, and thrombus formation.
- Poor compliance with chelation therapy is the single most important modifiable risk factor for complications.
- DKA in thalassemia patients may unmask underlying cardiac dysfunction and worsen prognosis.
- Early echocardiography and troponin testing should be considered in high-risk thalassemia patients presenting with DKA.
- Multidisciplinary care is crucial for survival in such complex presentations.

Conclusions

This case demonstrates the complex interplay of metabolic, infectious, and cardiovascular complications in thalassemia major. Early recognition of endocrine and

cardiac involvement, aggressive supportive management, and lifelong adherence to chelation therapy are vital to prevent such catastrophic outcomes.

Legend Figures



Figure 1



Figure 2

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