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Sepsis in a Young Man with Thalassemia Major: A Case Report

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Abstract

A 23-year-old young man with a transfusion-dependent thalassemia major presented to internal medicine outpatient clinic of our institution. His complaints included fatigue and abdominal pain for 1-2 days. He needed blood transfusions in every two weeks. On admission, he had a fever that not responded to empiric antibiotics. General condition of the patient deteriorated rapidly and he died in intensive care unit of our institution. In conclusion, physicians should aware of serious infectious caused by such microorganisms in patients with iron overload and proper antimicrobial therapy should be initiate at once.

Keywords: ***Thalassemia major; Infection; Blood transfusion***

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Introduction

Thalassemia Major is a genetic disorder in which patients require serial blood transfusions. Iron chelation is needed due to iron overload in these subjects.

Infection is a serious risk in Thalassemia Major Subjects with increased in iron stores [1]. Here we aimed to present a Thalassemic young man that required blood transfusion as frequent as twice a month which developed sepsis and deceased despite aggressive treatment in intensive care.

Case Report

A 23-year-old young man with a transfusion-dependent Thalassemia Major presented to internal medicine outpatient clinic of our institution. His complaints included fatigue and abdominal pain for 1-2 days. The last transfusion was made about two weeks ago. He was on iron chelating agent therapy. There was no history of insect bites or toxic substance exposure. He was emigrated from Iran about 6 months ago. His brother has also been detected with Thalassemia major.

His blood pressure was 100 mmHg systolic and 80 mmHg diastolic, heart rate was 120 per minute, body temperature was 38°C, and respiratory rate was 14 per minute. There was no pathology except few crackles in lower region of right hemithorax, icteric appearance and epigastric tenderness on physical examination. Blood transfusion has planned due to hemoglobin level was 7

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g/dl and other blood tests were found normal.

Cultures of blood, urine, sputum and throat were obtained. Ampicillin Sulbactam 1.5 gram four times a day was initiated as empiric antibiotic. Abdominal sonography was normal. Trans-thoracic echocardiography was performed to investigate infective endocarditis and revealed normal echocardiography findings. Fewer did not resolve and his general condition deteriorated rapidly. Ribavirin, ceftriaxone, moxifloxacin initiated after consultation to infectious diseases department. His serum creatinine rised to 2.96 mg/dL, urea 47 mg/dL, potassium 3.4 mmol/L, erythrocyte sedimentation rate was 132 mm/hour, C-reactive protein was 7.4 mg/l, Hb: 7 g/dL, MCV was 82 fl, RDW was 17.2% platelet count was 175 K/uL, ferritin was 1449 ng/ml, lactate dehydrogenase was 184 U/L, indirect bilirubin was 7,6 mg/dL, direct bilirubin was 1,33 mg/dL. Urine analysis revealed prominent hematuria and proteinuria. The nephrology department suggested intravenous hydration and a list of autoimmune markers to be studied.

Control hemogram revealed with decreased platelet count to 82 K/uL and a CRP decreased to 4.7 mg/dL. Peripheral blood smear showed no signs of either hemolytic uremic syndrome or thrombotic thrombocytopenic purpura (no schistocytes). Direct and indirect coombs tests were performed and found negative and corrected reticulocyte was 0.5%.

D-dimer was 4.89 mg/dL (0-0.55), fibrinogen was 268 (180-350) mg/dL. Platelet count further decreased to 75 K/uL and disseminated intravascular coagulation due to sepsis is diagnosed. His general condition worsened and the patient was removed to the intensive care unit. Because the blood pressure decreased to 80/50 mm/Hg, the patient was initiated with noradrenalin and dopamine as a positive inotropes. Two hours of hemodialysis performed according to the suggestions of nephrologists since serum creatinine raised to 4.1 mg/dL and acidosis in blood gas analysis (blood pH was 7.16, lactate was 5 mmol/L and hco3 was 13 mmol/L). Endotracheal intubation and respiratory support was performed on the patients.

Patchy infiltrates detected in control thorax plain radiography (Figure 1). He deceased on second day of hospitalization despite of intensive care treatment. None of the cultures were positive for bacterial growth.

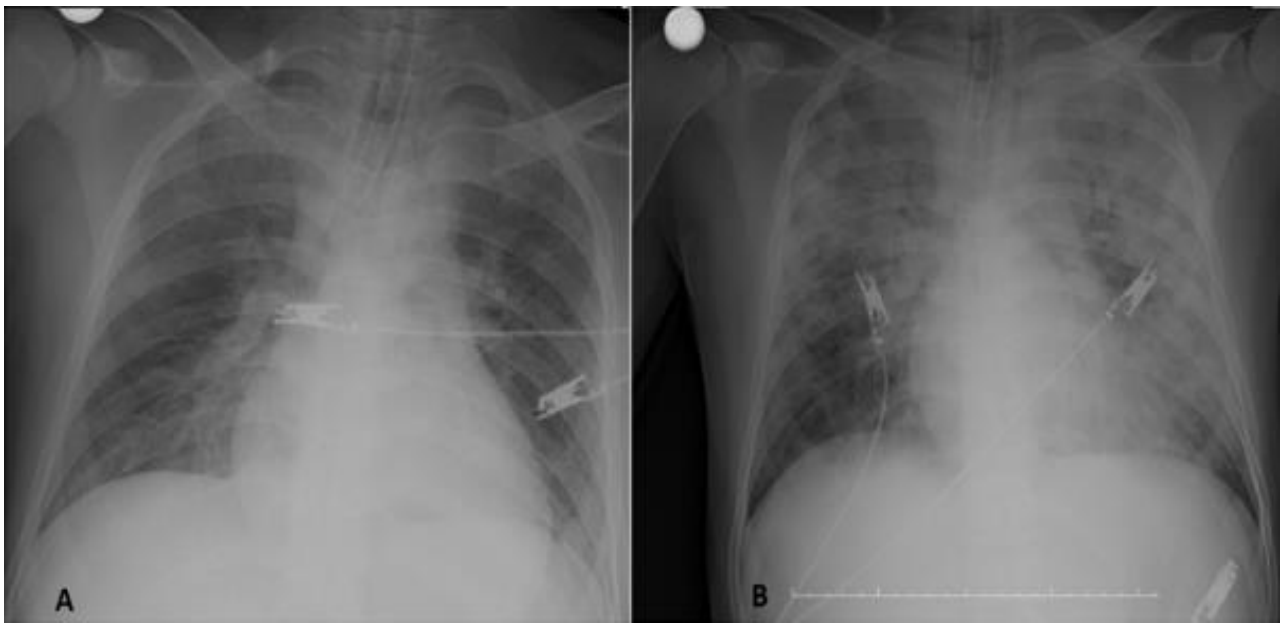

Figure 1: Chest X-ray of the patient on ICU admission (A) and in following hours of treatment (B) Increased patchy

infiltrates in both hemithorax.

Discussion

We reported a sepsis case in a young man with thalassemia major who did not respond to the treatment despite aggressive antibiotic and supportive care in intensive care unit.

Iron overload deteriorates anti-infectious capacities of leukocytes. Accumulated iron in serum may act as a growth factor for microorganisms that necessitate iron for reproduction [1]. Ferritin levels of the present patient were extremely high despite chelating therapy. Another risk factor for infection in Thalassemia is splenectomy, which increases the risk for infection with encapsulated bacteria i.e., Pneumococci [2]. However, present patient has not received splenectomy operation.

Patients with iron overload are at higher risk of infection with certain microorganisms [3]. Patients with iron accumulation has specific iron complex in their serum which bacteria can easily utilize [4]. *Yersinia Enterocolitica* and *Vibrio vulnificus* are two bacteria that can rapidly grow in serum with excess of iron. About half of the case with liver abscess caused by *Yersinia Enterocolitica* was associated with iron overload conditions [5]. Authors reported that homozygous beta-thalassemia subjects were at increased infection risk including *Yersinia Enterocolitica*, since infection with this microorganism usually prefer subjects with iron accumulation [6]. Indeed, Abbott et al. reported *Yersinia* infection in a patient whom substantially diagnosed with hemochromatosis [7]. Not only iron overload but also chelation therapy may increase infection risk in such patients. Chelating agents such as Desferrioxamine may increase bioavailability of serum iron for the bacteria [6]. Present case was on iron chelation therapy, too.

In conclusion, physicians should aware of serious infectious caused by such microorganisms in patients with iron overload and proper antimicrobial therapy should be initiated at once.

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