Case Report

A case of DKA with Pulmonary Thromboembolism in a newly diagnosed young diabetic male

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Abstract

Diabetic Ketoacidosisis [DKA] is a risk factor for developing venous thromboembolism which can be fatal if not recognised and treated on time. We report a case of DKA with pulmonary thromboembolism in a newly diagnosed diabetic patient. A 30 year old male patient presented to our emergency department with history of generalised weakness, shortness of breath, and dry cough for 10 days. His blood sugars were high and ketone bodies were positive. CT pulmonary angiography showed irregular filling defects in right and left pulmonary arteries and multiple pulmonary infarcts. Patient was treated with insulin and low molecular weight heparin (enoxaparin). The patient condition improved in 5 days and he was discharged with oral anticoagulants (rivoroxaban) and insulin.

In view of normal B12 deficiency, normal homocysteine levels and various other normal laboratory parameters in this patient, DKA can be considered as a single risk factor for the development of pulmonary thromboembolism. This case establishes the need for further studies and discussion about prophylactic anticoagulation in DKA patients to prevent venous or pulmonary thromboembolism.

Keywords: Diabetic ketoacidosis, Pulmonary thromboembolism, Venous thromboembolism, DKA, Heparin

Introduction

Diabetic Ketoacidosis (DKA) is the most common complication that can present acutely when the patient has uncontrolled high blood sugars. Diagnosis of DKA can be made when the patient has high blood sugar levels, positive urine ketone bodies, and high anion gap metabolic acidosis.[1] Acute Pulmonary thromboembolism is a common complication of Diabetic Ketoacidosis (DKA), and if not managed promptly on time, can be fatal. Venous thromboembolism can present as deep vein thrombosis or pulmonary thromboembolism or both. The incidence of venous thromboembolism is 0.75 to 2.7 per 1000 population globally, and it increases to 7 per 1000 in elderly age group (>70 yrs). [2] There is less scientific data available in india about incidence and prevalence of pulmonary thromboembolism, but a study conducted in india showed the prevalence of Pulmonary thromboembolism was significantly high in diabetic patients when compared to non-diabetic patients.[3] In a retrospective registry of indian patients with venous thromboembolism, patients with acute deep vein thrombosis without pulmonary embolism were 64%, acute deep vein thrombosis with pulmonary embolism were 23%, and pulmonary embolism alone were 13%. [4] Literature says deaths among patients with pulmonary thromboembolism is 18 fold higher than those with deep vein thrombosis. [5] CT pulmonary angiography is the choice of diagnosis to detect pulmonary embolism. The symptoms and few diagnostic tests like high D dimer

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levels, high NT pro BNP levels, S1Q3T3 pattern in ECG can give more clues towards the diagnosis.[5]

The levels of cogulation markers like clotting factors (Fibrinigen, factor VII, factor VIII, factor XI, factor XII, kallikrein, and thrombin-anti-thrombin complexes) are elevated in diabetes, and the level of anticoagulants like protein C are decreased. The fibrinolytic system (increased level of plasminogen activator inhibitor factor-1) is also inhibited in diabetic patients which further adds to the hypercoagulable state.[6] The other factors which contribute to the development of pulmonary thromboembolism in DKA patients is high serum viscosity, severe dehydration, and thrombosis secondary to low cardiac output. [7]

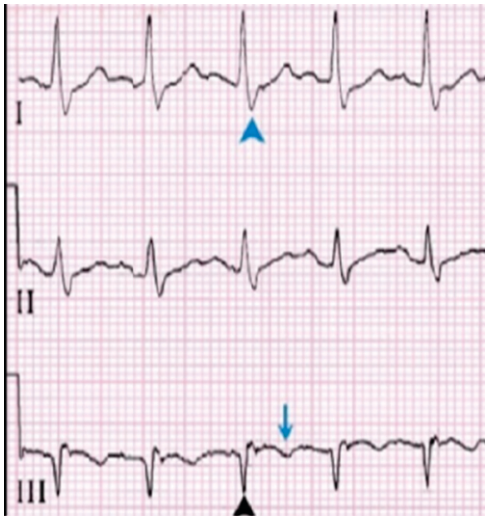
Case Report

We report a case of DKA with Pulmonary thrombo-embolism in a newly diagnosed diabetic patient. A 30 year old male admitted to emergency ward with history of generalised weakness, breathlessness, dry cough for 10 days. The symptoms almost started at the same time, they were insidious in onset, gradually progressive. Patient was weak throughout the day. Patient had generalized muscle pains. Breathlessness was aggravated on physical activity and relieved on rest, more in the night. Breathlessness was not associated with chest pain. No history of orthopnea or paroxysmal nocturnal dyspnea. Cough was insidious in onset, gradually progressive, not associated with sputum, no hemoptysis. Cough was aggravated on physical activity and relieved on rest, more in the day. No history of fever or other significant symptoms. Patient had no history of medical conditions like diabetes, hypertension, TB, seizures, etc. in the past and he was not on any medications. He had not undergone any surgery. Patient is not a smoker, not an alcoholic. Father was a type 2 diabetic, diagnosed at the age of 52 years. No other significant family history.

On physical examination, BP was 120/90 mm hg, SpO_2 – 86% @ RA, maintained at 98% with 4 lit O_2, Pulse rate was 98/min, he was tachypneic (RR - 30/min), he was afebrile and GRBS was 460 mg/dl. Bilateral pitting type of pedal edema extended to the level of ankles was noted. Systemic examination was normal. ECG showed $S_1Q_3T_3$ Pattern [Figure 1].

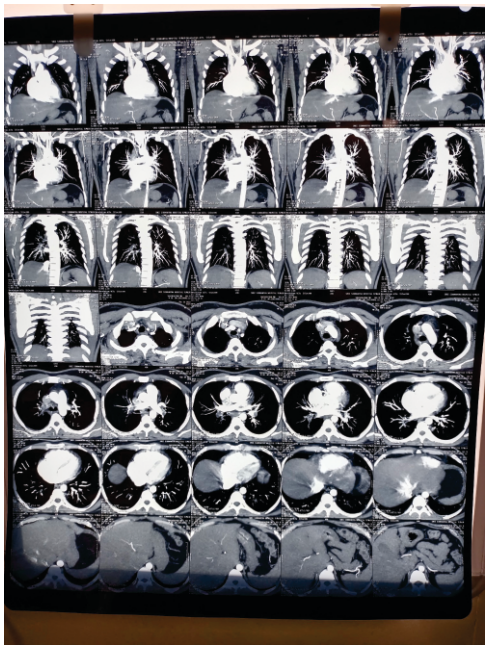
Patient was admitted in ICU for further follow up and treatment. On further investigations, Urine routine examination showed urine ketone bodies positive (+3), glucose present. Na^+ 127 mmol/l, K^+ 4.8 mmol/, Cl^- 98 mmol/, HCO_3^- 13.8 mmol/, in ABG, Anion gap was 20, PH was 7.21. Liver function tests were normal. Urea, serum creatinine, serum uric acid were in normal range.

Figure 1: $S_1Q_3T_3$ pattern of ECG seen in Pulmonary Embolism



HRCT: HRCT showed multiple pulmonary infarcts. CT Pulmonary Angiography showed irregular filling defect in right and left pulmonary arteries and bilateral interlobar and subsegmental branches suggesting

Figure 2: CT pulmonary angiography report of the patient



pulmonary thrombo embolism (Figure 2). Ultrasound abdomen was normal. 2D echo showed – Dialted RV/RA, (LVEF- 60%),

D dimer: 3.09 mg/l, (Normal Values: 0.2 to 0.7 mg/l) NT-PRO BNP – 9410 pg/ml (Normal < 125 pg/ml), HBA1C – 10.8%, Hb – 12.5 gm/dl. WBC – 10.62 x 103 U/lt, Platelets: 1.2 lacks/cu mm. Troponin I – 0.57 ng/ml (Normal – 0.2 to 0.8ng/ml). Urine culture and blood culture were negative. APTT – 27.4 sec (30–40 sec) Prothrombin Time: 21.6 sec (Normal : 11-16sec) INR : 1.7.

Syphilis card test was non-reactive. Serum Homocystine and vitamin B12 levels were in normal range. Serology was negative for Hepatitis B, Hepatitis C, Hepatitis E and HIV. Venous doppler of both lower limbs showed no features of DVT. RT-PCR was negative. P – ANCA, C-ANCA, ANA were negative.

Patient was given 4 to 5 litres of oxygen and SpO_2 was maintained >95%. Patient was given enough IV fluids and Insulin infusion intially. After achieving normoglycemia and closure of anion gap, subcutaneous insulin was given to maintain blood sugars. Patient was treated with Low mlecular weight Heparin, Enoxaparin S/C 60mg 12 hourly for 5 days. No major side effects were noted except for mild epistaxis on the third day of Heparin. Major clinical improvement was noticed in patient in 5 days. CT pulmonary angiogram was not repeated before discharge due to financial constraints. Patient was discharged with insulin and direct oral anticoagulants (Rivoroxaban 15mg twice a day for a week and 20mg once daily for 3 weeks) and asked to follow up after a week.

Discussion

In our case, the patient was diagnosed with DKA and pulmonary thromboembolism correlating laboratory and clinical parameters. The patient was a newly diagnosed diabetic presented with DKA and with no earlier co-morbidities. The common causes noted in patients were prolonged immobilization, recent surgery, malignancy and any systemic illness. DKA can be explained as one of the associated or aggrvating factors contributing to the development of pulmonary thromboembolism. As there are very few studies reported that have shown DKA as the single factor to cause pulmonary thromboembolism especially in the setting of development of DKA and pulmonary thromboembolism in a newly diagnosed diabetic patient, our case study becomes the rare one. Among the very few reported studies, in a seven case series study published by Irini Scordi-Belloet al. In 1996, four out of seven patients who had fatal pulmonary thromboembolism with Diabetic ketoacidosis (DKA) were newly diagnosed diabetics with out prior significant medical and family history.[8] This shows high level of unpredictability in early identification and treatment of pulmonary thromboembolism especially in newly diagnosed diabetics. In our patient, treatment was given at the right time for DKA with Insulin and anticoagulation with Low molecular weight Heparin was started for Pulmonary thromboembolism. Though the efficacy of unfractionated heparin and low molecular weight heparin is same in patients with pulmonary thromboembolism, the risk of hemorrhage and thrombocytopenia is less with low molecular weight heparin comparitively.[9,10] Fibrinolytic therapy can be given for massive pulmonary embolism,[11] though in our case it was not given. The continuation of oral anticoagulants can be done for 3 to 6 months ideally, and can be stopped if the risk is low with treatable risk factors and less number of co-morbidities.[12]

Conclusion

In our patient, diabetic ketoacidosis (DKA) is the predominant and single causative factor noted for the development of pulmonary thromboembolism. Dehydration and hyperosmolar states can lead to elevated plasma clotting factors, increased platelet aggregation, decreased coagulation inhibitors in diabetic ketoacidosis (DKA). These procoagulant mechanisms in DKA can go unrecognised and can cause life threatening pulmonary thromboembolism. Early diagnosis of pulmonary thromboembolism in DKA is always a challenge. Currently there are no clear recommendations for prophylaxis of pulmonary thromboembolism in DKA patients. There is a need for further studies and discussion on this topic to decrease mortality in DKA patients due pulmonary thromboembolism.

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