Venous thromboembolism is a well described, though infrequently reported, complication of diabetic ketoacidosis (DKA) (Anhalt et al. 1996; Gill et al. 2006). Studies have shown that systemic prothrombotic changes occur during diabetic ketoacidosis, including endothelial activation, decreased levels of free protein S and protein C activity, and conformational changes to the vascular endothelial cells, which change their hemostatic profile and result in a prothrombotic state (Carl et al. 2003). Case reports of pulmonary embolism (PE), one of the most serious potential complications of venous thromboembolism, secondary to the hypercoagulable state of DKA, have been noted in the literature (Anhalt et al. 1996; Quigley et al. 1994). Most instances of venous thromboembolism are managed with anticoagulation therapy consisting of heparin, Coumadin®, or both. We present a case of an individual with a PE secondary to DKA who was successfully treated with tissue plasminogen activator (tPA). Whereas the goal of heparin and Coumadin is to improve outcomes by preventing further clot formation, tPA acts by lysis of the active thrombosis. In massive pulmonary embolus, immediate thrombolysis can be a lifesaving measure.

A 55-year-old male, with no significant past medical history, presented to the emergency department at Long Island Jewish Hospital with xerostomia, polydypsia, polyuria, and progressive confusion during the previous week. His physical exam was significant for tachycardia and dry mucous membranes. Admission laboratory results showed sodium 118 mmol/L, chloride 78 mmol/L, bicarbonate 18 mmol/L, and glucose 786 mmol/L. The calculated anion gap was 22 and the venous pH was 7.35. A diagnosis of diabetic ketoacidosis was made. The patient was given intravenous insulin and two liters of intravenous normal saline in the emergency department before being transferred to the intensive-care unit, where normal saline was administered at 250 cc per hour and an insulin drip was begun. Subcutaneous heparin, 5,000 units every eight hours, was administered in the emergency department and the intensive-care unit for deep-vein thrombosis prophylaxis.

On hospital day two, the patient's symptoms resolved and he was tolerating a diabetic diet. His anion gap narrowed to 12 and his serum glucose decreased to 178 mmol/L. Shortly after transfer to a general medical unit, the patient became acutely dyspneic and tachypneic. He was noted to be tachycardic with a heart rate of 111 bpm and his previously normal oxygen saturation decreased to 78 percent on room air. The suspected diagnosis of PE was confirmed after CT angiography showed massive right- and left-pulmonary artery thromboses as well as thromboses of the bilateral superficial femoral and right popliteal veins. Severe right-ventricular dysfunction was demonstrated by a transthoracic echocardiogram. The patient was initially treated with intravenous tPA, 100mg over two hours. His symptoms resolved and his oxygen saturation rose to normal. Subsequent anticoagulation with intravenous heparin and oral Coumadin (10 mg) was started. The patient was discharged home when a therapeutic international normalized ratio was achieved.

This case appears to be an incident of PE precipitated by diabetic ketoacidosis. The patient had no prior history of venous or arterial thromboses, no recent surgery or vascular intervention, and only a short period of bed rest during which prophylactic subcutaneous heparin was given. Despite the absence of obvious risk factors, the patient developed a PE, which was successfully treated with thrombolysis using tPA. This case illustrates the necessity of a high index of suspicion for thrombosis in patients with DKA. Equally important, this case illustrates that thrombolysis by tPA offers an alternative to the traditional treatment of life-threatening venous thromboembolic events.

REFERENCES


