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DIABETIC KETOACIDOSIS TRIGERED BY SEPSIS AN INITIAL PRESENTATION OF TYPE 2 DIABETES MELITUS: A CASE REPORT

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INTRODUCTION

Diabetic Ketoacidosis, a life-threatening complication of diabetes, is most common in type 1 DM but **occurs in 10%-30% of newly diagnosed type 2 DM cases, often triggered by factors like infection.**

CASE DESCRIPTION

A 57 y.o man with no diabetes history presented to the ER with dyspnea but with stable condition. He had a recent 3-day fever, 1-week diarrhea (>5x/day), abdominal pain, vomiting (3x/day), and a month-long history of weight loss, polydipsia, polyphagia. There was also yellow discharge from a prior hand amoutaion site, suggesting infection. On exam vital sign stable.

While on observation patient suddenly litte bit Kussmaul breathing, tachycardia, desaturation (94% to 66%) requiring immediate intubation. After couple of time Lab and CXR result glucose 28,4 mmol/L, WBC 40,61 10^9/L, Potasium 6,7 mmol/L , Asidosis metabolic with pH 6,9, PCO2 43,0 mmHg, HCO3 10,2 mmol/L and Keton positit 2+ confirmed the diagnosis of DKA, septic and pneumonia so treat patient with DKA, hyperkalemia, sepsis and acidosis received 1500 ml isotonic fluids, insulin bolus and 0.1 IU/kg/hr drip, calcium gluconate, and sodium bicarbonate. Transferred to the ICU, they improved by day three, were extubated on day four, and recovered in the ward until discharge on day ten.

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DISCUSSION

The patient presented with **shortness of breath and clinical signs of infection** involving a prior hand amputation site, pneumonia, and gastrointestinal infection. The combination of these infections initiated a systemic inflammatory response that **progressed to sepsis,** which subsequently **triggered a metabolic disturbance leading to diabetic ketoacidosis** (DKA). The physiological stress of sepsis, through the rise of **counter-regulatory hormones and insulin resistance**, can **worse hyperglicemia** and **trigger ketogenesis**, especially in individuals with existing or newly diagnosed diabetes

Although the patient **initially appeared clinically stable** and showed no signs suggestive of DKA, clinical features of acidosis **gradually emerged during clinical observation**. The progression of this condition required **close clinical monitoring, with laboratory evaluation playing a crucial role** in determining disease severity and guiding appropriate management.

Treatment for DKA focuses on **restoring circulatory volume, gradually reducing blood glucose, correcting electrolyte imbalances, and addressing underlying causes like infections**. Fluid therapy is crucial to counter volume depletion, boosting intravascular volume and urine output. Insulin, given in physiologic doses and adjusted by glucose levels, is vital. DKA causes hyperkalemia due to acidosis and insulin deficiency, while insulin corrects it and calcium stabilizes cardiac membranes.

CONCLUSSION

This case highlights **sepsis** as a **trigger for DKA in newly diagnosed type 2 DM**, complicating diagnosis and early management**. Accurate clinical assessment by "eagle's eyes" needed to bring effective treatment.** Beside, **early recognition** and **timely intervention** in the ER are **critical to prevent life-threatening complications and improve outcomes** although there is a challenge while in limited source.