Diabetic ketoacidosis precipitated by Covid-19 in a patient with newly diagnosed diabetes mellitus - Ying Jie Chee - Khoo Teck Puat Hospital, Singapore

Ying Jie Chee

Abstract
There is scarce data on diabetic ketoacidosis (DKA) in Covid-19 infection. We report a case of DKA precipitated by Covid-19 in a patient with newly diagnosed diabetes mellitus. A 37 year-old, previously healthy man presented with 1 week history of fever, vomiting, polydipsia and polyuria. On admission, his temperature was 38.5°C. He was a haemodynamically stable but mildly tachycardic. He did not display Kussmaul’s breathing and did not require supplemental oxygen. His body mass index was 22.6 kg/m^2 with no evidence of insulin resistance. Given positive contact history, he was tested and confirmed to be infected with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Laboratory investigations (Table 1) were significant for hyperglycemia, high anion gap metabolic acidosis and ketonemia, confirming the diagnosis of DKA. He received 6 L of intravenous fluids and intravenous insulin infusion in the first 24 h. Serum electrolytes were closely monitored. DKA resolved the following day and he was tran-sitioned to subcutaneous insulin therapy. DKA occurs as a result of insulin deficiency and increased counterregulatory responses, which favour the production of ketones. The interactions between SARS-CoV-2 and the renin-angiotensin-aldosterone system (RAAS) might provide another mechanism in the pathophysiology of DKA. Angiotensin-converting enzyme 2 (ACE2), a key enzyme in the RAAS, catalyzes the conversion of angiotensin II to angio-tensin (1–7)[1]. ACE2 is highly expressed in the lungs, pancreas and serves as the entry point for SARS-CoV-2[1]. After endocytosis of the virus complex, ACE2 expression is down-regulated[2]. There are 2 implications of these interactions. Firstly, entry of SARS-CoV-2 into pancreatic islet cells may directly aggravate beta cell injury[3]. Secondly, downregulation of ACE2 after viral entry can lead to unopposed angiotensin II, which may impede insulin secretion[4]. These 2 factors might have contributed to the acute worsening of pancreatic beta cell function and precipitated DKA in this patient. In addition, the relationship between SARS-CoV-2 and the RAAS can complicate DKA management. Excessive fluid resuscitation may potentiate acute respiratory distress syndrome as angiotensin II increases pulmonary vascular permeability and worsens damage to lung parenchyma[5]. Furthermore, angiotensin II stimulates aldosterone secretion, potentiating the risk of hypokalemia, which may necessitate potassium supplementation in order to continue intra-venous insulin to suppress ketogenesis. In conclusion, it is possible that SARS-CoV-2 may aggravate pancreatic beta cell function and precipitate DKA. Further studies will help delineate the pathophysiology. We also highlight the pertinent clinical considerations in the con-current management of two life-threatening conditions – DKA and Covid-19.
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The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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