Diabetology

Extended Abstract

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

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Abstract

There is scarce data on diabetic ketoacidosis (DKA) inCovid-19 infection. We report a case of DKA precipitated byCovid-19 in a patient with newly diagnosed diabetes mellitus.A 37 year-old, previously healthy man presented with1 week history of fever, vomiting, polydipsia and polyuria.On admission, his temperature was 38.5°C. He washaemodynamically stable but mildly tachycardic. He did notdisplay Kussmaul's breathing and did not require supplemen-tal oxygen. His body mass index was 22.6 kg/m2with no evi-dence of insulin resistance.Given positive contact history, he was tested and con-firmed to be infected with severe acute respiratory syndromecoronavirus 2 (SARS-CoV-2). Laboratory investigations(Table 1) were significant for hyperglycemia, high anion gapmetabolic acidosis and ketonemia, confirming the diagnosisof DKA.He received 6 L of intravenous fluids and intravenous insu-lin infusion in the first 24 h. Serum electrolytes were closelymonitored. DKA resolved the following day and he was tran-sitioned to subcutaneous insulin therapy.DKA occurs as a result of insulin deficiency and increasedcounterregulatory responses, which favour the production ofketones. The interactions between SARS-CoV-2 and the renin-angiotensin-aldosterone system (RAAS) might provideanother mechanism in the pathophysiology of DKA.Angiotensin-converting enzyme 2 (ACE2), a key enzyme inthe RAAS, catalyzes the conversion of angiotensin II to angio-tensin (1–7)[1]. ACE2 is highly expressed in the lungs, pan-creas and serves as the entry point for SARS-CoV-2[1]. Afterendocytosis 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 maydirectly aggravate beta cell injury[3]. Secondly, downregula-tion of ACE2 after viral entry can lead to unopposed angioten-sin II, which may impede insulin secretion[4]. These 2 factorsmight have contributed to the acute worsening of pancreaticbeta cell function and precipitated DKA in this patient.In addition, the relationship between SARS-CoV-2 and theRAAS can complicate DKA management. Excessivefluid resuscitation may potentiate acute respiratory distress syndrome as angiotensin II increases pulmonary vascularpermeability and worsens damage to lung parenchyma[5].Furthermore, angiotensin II stimulates aldosterone secretion,potentiating the risk of hypokalemia, which may necessitatemore potassium supplementation in order to continue intra-venous insulin to suppress ketogenesis.In conclusion, it is possible that SARS-CoV-2 may aggra-vate pancreatic beta cell function and precipitate DKA. Fur-ther studies will help delineate the pathophysiology. Wealso highlight the pertinent clinical considerations in the con-current management of two life-threatening conditions –DKA and Covid-19.

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This work is partly presented at 52th Annual Congress on Neuroscience and stroke 2020, December 14, 2020

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