Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Diabetes & Metabolism 47 (2021) 101162

Available online at
ScienceDirect
www.sciencedirect.com

Elsevier Masson France
EM|consulte
www.em-consulte.com

Research letter

COVID-19 symptoms masking inaugural ketoacidosis of type 1 diabetes

A 31-year-old white European male, with no known previous medical history, arrived at the emergency department with a 5-day fever, fatigue and vomiting. He mentioned having had contact with two COVID-19 cases [infection caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)] within the past 5 days. His temperature was 38.7 °C, with a respiratory rate of 28 breaths/min, heart rate of 132 bpm and oxygen saturation of 93% with an ambient air breathing apparatus. Pulmonary auscultation found bibasilar crackles. Chest computed tomography (CT) showed basal bilateral ground-glass opacities and air-space consolidation that was highly suggestive of COVID-19 injury (graded as minor, affecting < 10% of lung volume). An oropharyngeal swab for COVID-19 was positive. Blood gas testing found a low pH (7.25) with bicarbonate (8 mmol/L) suggestive of metabolic acidosis. Besides these findings, the patient's capillary blood glucose was 23.7 mmol/L with elevated ketonaemia (5.2 mmol/L), which confirmed the diagnosis of diabetic ketoacidosis (DKA). Results of his laboratory findings are detailed in Table 1.

Table 1
Patient's laboratory findings.

White blood cell counts (per mm^3)	7800
Differential counts (per mm^3):	
Total neutrophils	5830
Total lymphocytes	1000
Total monocytes	910
Haemoglobin (g/dL)	18.0
Platelet count (per mm^3)	233,000
Glucose (mmol/L)	19.8
Sodium (mmol/L)	133
Potassium (mmol/L)	4.5
Protide (g/L)	84
Creatinine (μmol/L)	83
eGFR (mL/min/1.73 m^2)	93.5
C-reactive protein (mg/L)	16
High-sensitivity cardiac troponin I (ng/L)	< 4
Arterial blood gas:	
pH	7.28
PO2 (mmHg)	85.8
PCO2 (mmHg)	18.5
CO2 (mmol/L)	9.3
Bicarbonate (mmol/L)	8

eGFR: estimated glomerular filtration rate; PO2/PCO2: partial pressure of oxygen/carbon dioxide.

The treatment administered for DKA included intravenous (IV) insulin infusions, and correction of fluid loss and electrolyte disturbances. DKA remission was rapidly obtained and subcutaneous (sc) insulin therapy was started 24 h after admission.

The diagnosis of immune-mediated type 1 diabetes (T1D) was confirmed by the finding of positive autoantibodies to glutamic acid decarboxylase (GAD) and to zinc transporter 8 (ZnT8; 118.4 U/mL and 304.2 U/mL, respectively), whereas islet-cell (ICA) and insulinoma-associated-2 (IA2) autoantibodies were negative [1].

Regarding the COVID-19 infection, the patient required passive oxygen therapy with 2–9 L/min for 5 days, while no antibiotherapy, antiviral or immunosuppressive therapies were administered. His oxygen requirement decreased progressively and was stopped on day 7. The patient was discharged at day 8 with sc insulin treatment only.

This was a case of an inaugural DKA revealing T1D in a young man with COVID-19 infection. In the emergency room, his COVID-19 symptoms were at the forefront while the DKA diagnosis was incidental. This case highlights the fact that clinicians must be aware of other acute diseases that can be veiled by COVID-19 symptoms.

In this particular case, COVID-19 most probably triggered the DKA, but was not causal in the occurrence of T1D. Indeed, in the course of T1D, the cell-mediated autoimmune destruction of β cells in the pancreas leading to hyperglycaemia begins some time (0.5–1.5 years) before the onset of its symptomatic manifestations [2]. However, that COVID-19 could exacerbate DKA as a precipitating factor cannot be excluded, as it has been demonstrated that SARS-CoV-2, through binding to its ACE-2 receptor expressed in β cells, can damage islets and, thus, reduce insulin release [3].

Disclosure of interest

The authors declare that they have no competing interest.

References

[1] American Diabetes A. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2020. Diabetes Care 2020;43(Suppl. 1):S14–31.
[2] Insel RA, Dunne JL, Atkinson MA, Chiang JL, Dabelea D, Gottlieb PA, et al. Staging presymptomatic type 1 diabetes: a scientific statement of JDRF, the Endocrine Society, and the American Diabetes Association. Diabetes Care 2015;38:1964–74.
[3] Yang JK, Lin SS, Ji XJ, Guo LM. Binding of SARS coronavirus to its receptor damages islets and causes acute diabetes. Acta Diabetol 2010;47:193–9.

https://doi.org/10.1016/j.diabet.2020.05.004

L. Potier[a,*], J.B. Julla[b], R. Roussel[a], P. Boudou[c], D.C. Gauthier[b], C. Ketfi[b], J.F. Gautier[b]
[a]*Bichat Hospital, AP–HP, Université de Paris, Paris, France*
[b]*Lariboisière Hospital, AP–HP, Université de Paris, Paris, France*
[c]*Saint-Louis Hospital, AP–HP, Université de Paris, Paris, France*

[*]Corresponding author at: Département de diabétologie, endocrinologie et nutrition, Hôpital Bichat, 46, rue Henri Huchard, 75018 Paris, France
E-mail address: louis.potier@gmail.com (L. Potier).

Received 3 May 2020
Received in revised form 14 May 2020
Accepted 15 May 2020
Available online 21 May 2020