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SHORT COMMUNICATION

Type 1 diabetes onset triggered by COVID-19

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The epidemic of coronavirus disease-2019 (COVID-19) is caused by the severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) virus. Some data describing characteristics and prognosis of patients with COVID-19 and diabetes are now available, for example, for hospitalized patients in the CORONADO study [1]. Potential links between diabetes and COVID-19 infection were already described [2]. Indeed, angiotensin-converting enzyme 2 (ACE2) has been identified as the receptor for the coronavirus spike protein [3], and ACE is expressed on pancreatic beta cells [2]. It was suggested that SARS-CoV2 could induce beta cell damage and new onset diabetes [2], but the phenotype of these new cases of diabetes has not been described.

Here we report the case of a 29-year-old woman with a medical history of gastric by-pass one year earlier and family history of diabetes (aunts with type 2 diabetes; a cousin with type 1 diabetes diagnosed at the age of 7 years). She presented two months earlier (20 March 2020) severe asthenia, fever, stiffness and dyspnea. Then, she presented anosmia and ageusia, with anorexia (25 march). She was admitted at the emergency department, symptomatic treatment was delivered for a suspected COVID-19 infection, and she was discharged (glycemia was normal at this time). Two weeks after, she did no longer have any symptoms. But one month after her first symptoms of COVID-19 (24 April), she presented acute polyuria–polydipsia syndrome. Diabetes mellitus was diagnosed (12 may) with a glycemia of 3.7 g/l (20.5 mmol/l), non-significant ketosis (0.7 mmol/l) and normal bicarbonate level (26 mmol/l). HbA1c level was 11.8% (105 mmol/mol). Her weight was 120 kg before gastric by-pass, 65 kg before COVID-19 and 57 kg (BMI of 21.5 kg/m^2) at diabetes diagnosis. The diabetes was immediately insulin requiring, and she was treated with basal bolus regimen. She did not present metabolic comorbidities and markers (no hypertension, negative CRP (< 0.6 mg/l, Hdlc 0.46 g/l, Ldlc 0.43 g/l, triglycerides 0.42 g/l, normal ALT, AST, gGT and ferritin levels, no liver steatosis at the CT scan). Lipase and TSH levels were normal; pancreatic CT scan was normal. C-peptide was low at 0.07 pmol/ml (normal values between 0.37 and 1.47). Autoantibodies against pancreatic beta cells were tested, and finally, glutamic acid decarboxylase-65 autoantibodies (GAD-65A) were positive (93UI/ml, N < 17) in favor of immune-mediated type 1 diabetes, whereas tyrosine phosphatase IA2 antibodies (IA2A) and zinc transporter 8 antibodies (ZnT8A) were negative.

SARS-CoV2 serology was positive (Elecsys®, Roche), confirming previous COVID-19 infection.

This observation highlights the fact that COVID-19 infection may also trigger type 1 diabetes onset. Viral infection, in particular, by enteroviruses but also by coronaviruses, is a well-known environmental trigger for the development of type 1 diabetes [4]. In the case presented herein, there was a short delay between COVID-19 infection and diabetes onset. It remains to determine if the hyperinflammation/cytokine storm described with this infection could accelerate the onset of type 1 diabetes in genetically susceptible individuals.

In addition, the patient was obese before undergoing gastric bypass one year earlier. Obese patients have higher risks to develop viral infection like influenza (with more complications) [5], but what about a patient with a massive weight loss in the first year after a bariatric surgery?

In conclusion, the relationship between SARS-CoV2 exposition and autoimmune diabetes development must be further studied, and incidence of type 1 diabetes will be carefully observed in the next months.

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Compliance with ethical standards

Conflict of interest The authors declare that there is no conflict of interest associated with this manuscript.

Statement of human and animal rights All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008.

Statement of informed consent Informed consent was obtained from all patients for being included in the study.

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