Type 1 diabetes onset triggered by COVID-19

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Short Report

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Abstract

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. Potential links between diabetes and COVID-19 infection were already described. Indeed Angiotensin-converting-enzyme 2 (ACE2) has been identified as the receptor for the coronavirus spike protein, and ACE is expressed on pancreatic beta cells. It was suggested that SARS-CoV2 could induce beta cell damage and new onset diabetes, but the phenotype of these new cases of diabetes has not been described.

This observation presented in that paper 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. 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.

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.

Introduction

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 [4]. 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.

Case Report

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 bicabonates 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/m²) 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 ferritine 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 0.37–1.47). Antoantibodies 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 [5]. 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) [6], but what about a patient with a massive weight loss in the first year after a bariatric surgery?

Conclusion

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.

Declarations

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Duality of interest

The authors declare that there is no duality 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 (to participate and to publish) was obtained from the patient for being included in this study.

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