Liggins Institute
University of Auckland
Auckland

School of Nursing, Midwifery and Health Practice
Victoria University of Wellington
Wellington, New Zealand

Philip J. Weston, MBChB
Newborn Intensive Care Unit
Waikato District Health Board
Hamilton, New Zealand

Greg D. Gamble, MSc
Jane E. Harding, DPhil
Liggins Institute
University of Auckland
Auckland, New Zealand

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Severe coronavirus disease 2019 in children and young adults

To the Editor:

DeBiasi et al[1] report that 3% of the pediatric patients who tested positive with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) at their center had a history of diabetes. Diabetes is currently listed by the Centers for Disease Control and Prevention as an underlying condition that places individuals at higher risk for severe illness. Two of the 5 patients with diabetes required hospitalization. Of the 2 patients, 1 patient with type 1 diabetes and brain injury required intensive care unit-level care. It would be informative to know the type of diabetes, duration of diagnosis, and glycemic control for those patients. Would the authors be able to provide these data, as well as whether the patients presented with symptoms related to diabetes vs symptoms related to SARS-CoV-2? Were any of the patients with SARS-CoV-2 newly diagnosed with diabetes?

At the Mount Sinai Kravis Children's Hospital, during the height of the pandemic in New York City, 10 pediatric patients (average age, 14.5 years; 8 female; 5 with new-onset diabetes) were treated in the emergency department or hospitalized with diabetes related complications. Eight were found to be in diabetic ketoacidosis when presenting to the emergency department. Three patients tested SARS-CoV-2 positive, and 3 patients had symptoms suggestive of SARS-CoV-2 and were in contact with sick family members. Compared with the prior 3 years at our children's hospital, no significant difference was noted in the number of patients with type 1 diabetes admitted or treated in the emergency department. There were also no significant differences in the number of newly diagnosed patients or severity of diabetic ketoacidosis.

To date, as part of the ongoing Type 1 diabetes COVID-19 Surveillance Study (www.t1dexchange.org/COVID19) coordinated by the T1D Exchange, there have been more than 20 reported cases of SARS-CoV-2 nationally in pediatric patients with type 1 diabetes.

Meredith Wilkes, MD
Rula Issa, MS
Anna Aluf, NP
Kara Beliard, MD
Mabel Yau, MD
Robert Rapaport, MD
Department of Pediatrics
Icahn School of Medicine at Mount Sinai
New York, New York

Osagie Ebekozien, MD, MPH
Diabetes Exchange
Boston, Massachusetts

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Reply

To the Editor:

In our interim report describing the first 177 severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)-positive symptomatic pediatric patients presenting for care at our institution, 5 patients (3%) had an underlying diagnoses of diabetes. All 5 of these patients were female, ranging from 13 to 20 years of age. Four of the 5 patients had type 2 diabetes and only 1 patient had type 1 diabetes. All of the patients had been diagnosed with diabetes from 1.5 to 9.0 years before their SARS-CoV-2 infection; none of these patients had new-onset diabetes and none presented in diabetic ketoacidosis.

Two of the 5 patients did not require hospitalization and both presented with symptoms referable to respiratory infection, rather than any exacerbation related to their underlying type 2 diabetes. One patient presented primarily with minor upper respiratory symptoms without hypoxia. This patient did not present with hyperglycemia or hypoglycemia, but had a history of poor glycemic control despite metformin therapy with consistently and markedly elevated A1C documented as recently as 3 months before infection with SARS-CoV-2. The second patient, who also had asthma as

a comorbidity, presented with fever, cough, and diarrhea, but no hypoxia. This patient had been managed with metformin for several years and had undergone bariatric surgery for morbid obesity 16 months before presenting with SARS-CoV-2 infection, but did not have acute glycemic control issues at the time of SARS-CoV-2 diagnosis.

Of the 3 patients with diabetes who required hospitalization, all had systemic symptoms attributable to both SARS-CoV-2 infection, as well as diabetes. One had longstanding type 1 diabetes and prior central nervous system injury secondary to diabetic ketoacidosis/cerebral edema; this patient presented with fever, chest pain, and increased oxygen requirement from baseline. She had a history of good glycemic control (last A1C 7.4%), but had hyperglycemia, without diabetic ketoacidosis at the time of SARS-CoV2 infection. The second patient presented with shortness of breath and chest pain, but no hypoxia, and had concomitant nausea, vomiting, and hypoglycemia. This patient had been diagnosed 1.5 years earlier at an external facility with type 2 diabetes and treated with short-acting insulin rather than oral therapy. However, upon review at our institution, she was deemed to be more consistent with prediabetes rather than diabetes and she was discharged on oral therapy. The third patient had type 2 diabetes with longstanding poor glycemic control despite insulin therapy. She initially presented with fever, chills, nausea, and headache, without respiratory symptoms or acute change in her glycemic control and was managed as an outpatient. However, after improvement in her symptoms, she was admitted 10 days later (after the date of our interim report) with chest pain and shortness of breath, and determined to have pulmonary embolism, as well as significant hyperglycemia (300-400) without diabetic ketoacidosis, requiring initial intravenous insulin therapy. All 3 of these patients had stabilization of their blood glucose levels relatively quickly after hospitalization.

Roberta L. DeBiasi, MD, MS
Division of Pediatric Infectious Diseases
Children's National Hospital/Children's National Research Institute
Department of Pediatrics and Microbiology, Immunology and Tropical Medicine
The George Washington University School of Medicine and Health Sciences
Washington, District of Columbia

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Body mass index rebound, weight gain in puberty, and risk of cardiovascular disease

To the Editor:

Yuan et al identified 3 groups with distinct trajectories in body mass index (BMI) related to cardiovascular disease risk in adulthood by analyses of a dataset with 30 years of follow-up in a population in northern China.[1] Among the 3 groups characterized as low-, moderate-, and high-increasing, based on the rate of BMI increase, the high-increasing group (11.9% of 2789 total participants) had the highest rate of cardiovascular disease in adulthood. This group had relatively moderate initial BMI levels, but BMI then increased steeply from about age 12 years and exceeded BMI in the moderate-increasing group at age 18 years.

In the pathway to obesity, adolescent obesity forms with acceleration of BMI in childhood following early adiposity rebound, which refers to an early increase in BMI before 4 years of age.[2-4] As these authors mentioned, the present study lacks data on children before age 6 years, which makes it impossible to look at the relationship between timing of adiposity rebound and BMI trajectories into adolescence.[1]

In this study, the high-increasing group had lower average BMI at 6 years of age compared with the moderate group, and their BMI then began to increase after entering adolescence.[1] This change is unlikely to be attributable to early adiposity rebound. This position is supported by previous studies showing that early adiposity rebound is associated with high BMI at 6-8 years of age, and children who develop adolescent obesity have a higher rate of change of BMI from early childhood.[5-7]

Thus, it seems that there is a distinct subgroup in which BMI increases rapidly after onset of puberty, increasing the cardiovascular disease risk owing to increasing adiposity. Pubertal maturation worsens obesity after puberty, but social background is also thought to be involved in the development of obesity.[8-10] Although efforts to alter trajectories that are predictive of adult obesity should commence before preschool age, a prevention strategy is also needed for obesity developing during puberty as a second critical window.

Osamu Arisaka, MD, PhD
Department of Pediatrics
Nasu Red Cross Hospital
Ohtawara, Tochigi-ken, Japan

Go Ichikawa, MD, PhD
Department of Pediatrics
Nasu Red Cross Hospital
Ohtawara, Tochigi-ken, Japan

Department of Pediatrics
Dokkyo Medical University School of Medicine
Mibu, Tochigi-ken, Japan

Satomi Koyama, MD, PhD
Department of Pediatrics
Dokkyo Medical University School of Medicine
Mibu, Tochigi-ken, Japan

Toshimi Sairenchi, PhD
Department of Public Health
Dokkyo Medical University School of Medicine
Mibu, Tochigi-ken, Japan

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