respiratory tract infections. Less commonly, AdV causes gastrointestinal, ophthalmologic, and genitourinary diseases. Serotype F 40 and 41 account for 5–10% of acute gastroenteritis in infants, with 6–9 days of symptom duration. Severity and prognosis due to different serotype are not specified.[4]

Although AdV enteritis is self-limiting, it can cause severe symptoms and reactivation in immunocompromised hosts. Cidofovir for immunocompromised hosts, or those with severe disease,[5] is not authorized in Japan. It is a poor prognostic factor for patients after hematopoietic stem cell / solid organ transplantation but conditions occurring with malignancies are quite rare.[2]

The AdV enteritis did not develop in our patient following a transplantation; however, the acute diarrhea induced hypovolemic shock with a prolonged recovery time. Nausea, caused by anti-cancer drugs and gastroenteritis, prevented oral intake of water, resulting in a large free water deficit. Sodium intake via fluid infusion may also have contributed to the exacerbation of hypernatremia. In our patient, intestinal mucosal damage due to chemotherapy, and immunosuppression due to bone marrow suppression, might have resulted in the severe AdV enteritis. There were also no suspected individuals with gastroenteritis around him, suggesting his illness arose from reactivation of AdV. It should be noted that, while rare, there is a risk of AdV reactivation and life-threatening infection in children undergoing chemotherapy for malignancy without transplantation.

In conclusion, our patient, a young boy, developed life-threatening AdV enteritis following chemotherapy for AML. Physicians should be watchful for signs of infection, especially in patients with intestinal mucosal injury and severe myelosuppression, even without prior transplantation.

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Disclosure

Informed consent from the patient's parents and approval from the institutional review board of the University of Tsukuba Hospital were obtained (H27-167). The authors declare no conflict of interest.

Author contributions

A.Y.S. and H.F. treated the patient. A.Y.S. wrote the article with support from H.F. R.S., Y.Y., and H.T. critically reviewed the manuscript. All authors read and approved the final manuscript.

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COVID-19 in a child with severe propionic acidemia

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Over 60 million people globally have been infected with severe acute respiratory syndrome coronavirus 2 (SARS-CoV2), of which over 1 400 000 have died.[1] In children with coronavirus disease 2019 (COVID-19), clinical manifestations appear to be less severe than in adults.[2] However, there is a lack of reports regarding individuals with acutely decompensating metabolic disorders diagnosed with the virus. Due to

the risk of developing a metabolic crisis secondary to a SARS-CoV2 infection, metabolic disease centers have been cautious handling these cases.

We present a 7-year-old female of Hispanic and Caucasian ancestry with propionic acidemia who was recently diagnosed with, and recovered from, COVID-19. Propionic acidemia is a well characterized inborn error of metabolism in which patients may experience encephalopathy, seizures, or a coma resulting in death.[3] Despite early intervention, she has global developmental delay, seizures, chronic constipation, pancytopenia, and recurrent pancreatitis. An echocardiogram in the spring of 2020 demonstrated normal cardiac function. She is G-Tube dependent with limited oral intake. Her diet meets energy recommendations but exceeds protein recommendations.[4] Despite increased protein intake, she has low levels of essential amino acids. Prior to 2020, she was hospitalized 3–4 times a year. This year, she has been admitted monthly with various indications. During admissions, her arterial blood gas pH has ranged from 7.01 to 7.45 with an anion gap between 4 and 27. The patient's white blood cell count has been between 1–2 $(10)^3$/uL, and she has required platelet transfusions. Ammonia levels were 17–105 micromol/L, which is within her normal range.

Earlier this year, we received a call from her mother stating that she had been lethargic for 2 days; however, she was tolerating her feeds, afebrile, and having normal bowel movements. At her local emergency room, critical labs, including ammonia level, were consistent with the patient's baseline. Due to concerns from previous admissions, the patient was transferred to our hospital. She then experienced recurrent emesis and was made nil per os (NPO). Total parenteral nutrition was started thereafter. On hospital day 2, the patient's routine SARS-CoV2 test resulted positive. She did not exhibit any respiratory symptoms, and her breathing was stable on room air. She experienced a one-time fever to 38.1 °C, which resolved spontaneously. The virus was treated with supportive care, and the use of novel treatments, such as remdesivir, was not employed. The patient was empirically started on the antibiotics vancomycin and cefepime due to her high risk of bacterial infection. Regular feeds without protein restriction were restarted on hospital day 3, and she tolerated them well. Her lethargy also began to improve. She was monitored closely for the next 2 days with continued improvement and was discharged home on hospital day 6. We have since followed up with her, and she was back at her baseline and does not appear to have any lingering symptoms of COVID-19. A repeat polymerase chain reaction (PCR) test for SARS-CoV2 25 days after the initial test was positive during another admission to her local hospital for emesis; however, a repeat test 33 days after her initial positive test was negative. She did not present with any symptoms of COVID-19 on the second admission. We do not believe that this was a new SARS-CoV2 infection, but rather this test had remained positive from her first admission.

Prior to the pandemic, the patient attended a prescribed pediatric extended center (PPEC), but she had not gone since spring 2020. A second-degree relative of the patient had symptoms of fever and myalgia prior to the early July admission and subsequently tested positive for SARS-CoV2. Her family believes this was likely the exposure that led to the patient's positive test as they had quarantined for several months. After her admissions, the rest of her family tested negative.

It is well documented that children with COVID-19 do not present with severe complications, and fatalities have been seen in only 0.05% of pediatric patients.[2] Some children do present with fever, pneumonia, or a rare multisystem inflammatory syndrome.[2] A case report by Caciotti *et al*. described a 1 year old with propionic acidemia and COVID-19 who exhibited fever up to 38.5 °C, emesis, skin pallor, and dyspnea. A chest X-ray demonstrated small subpleural thickenings. Anabolic support and supportive care were provided, and he improved within 7 days of admission and was discharged home.[5]

Our observations indicate that having a severe form of a metabolic disorder is not necessarily associated with a worse clinical outcome. Perhaps the anabolic support and reversal of catabolism provided by both our team and the team in Italy prevented the patients' decompensation. It is important to note that this is a small sample size. It is possible that children with metabolic disease who contract COVID-19 may be treated similarly to other patients with decompensating metabolic diseases; however, several more cases need to be observed and treated to confirm this. The prevention of metabolic decompensation in patients with inborn errors of metabolism is likely a crucial component in the outcome of COVID-19 cases.[5]

Disclosure

The authors declare no conflicts of interest.

Author contribution

M.S.N. and M.T. and S.H. conceptualized and designed the study, collected data, drafted the initial manuscript, and reviewed and revised the manuscript. D.B. and W.T. carried out the initial analyses, and critically reviewed and revised the manuscript. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work. Written consent was obtained from the patient.

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