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New-Onset Type 1 Diabetes Mellitus Triggered by COVID-19 with Severe DKA and Asymmetric Neuropathy: A Case Report

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Introduction

Type 1 diabetes mellitus (T1DM) is a chronic autoimmune disorder characterized by progressive destruction of pancreatic β-cells, resulting in absolute insulin deficiency. Its pathogenesis is multifactorial, involving genetic susceptibility interacting with environmental factors, particularly viral infections. Several studies have identified specific viral agents—such as enteroviruses and influenza viruses—as potential triggers for autoimmune β-cell injury, which may explain seasonal peaks and increased incidence of newly diagnosed T1DM in pediatric populations [1].

The emergence of the COVID-19 pandemic, caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has intensified scientific interest in viral-induced disturbances of endocrine function. Accumulating evidence suggests that SARS-CoV-2 infection may precipitate new-onset T1DM in both children and adults through multiple mechanisms, including direct cytopathic effects on pancreatic tissue, dysregulated immune activation, and molecular mimicry leading to autoimmune β-cell targeting [2,3].

Patients with pre-existing diabetes are known to have a higher likelihood of developing severe COVID-19 manifestations, including diabetic ketoacidosis (DKA). Recent epidemiological reports also indicate an increase in newly diagnosed T1DM and DKA episodes among children following SARS-CoV-2 infection [4,5]. When COVID-19 precipitates T1DM, the disease may present abruptly with severe metabolic derangements, including DKA, and may involve multiorgan dysfunction such as acute kidney injury (AKI) or early neurological complications [6]. However, neuropathic manifestations occurring at the onset of COVID-19–associated T1DM have not been well documented in the scientific literature, and available evidence remains extremely limited [8].

We describe a rare pediatric case of SARS-CoV-2–associated new-onset T1DM presenting with severe DKA, AKI, and asymmetric diabetic neuropathy. This case underscores the multifaceted impact of COVID-19 on endocrine and immune systems, while also highlighting potential neurological and renal complications in newly diagnosed T1DM.

Case Presentation:

A previously healthy 13-year-old female presented to the emergency department after a sudden loss of consciousness, preceded by two days of fever and cough. Her history included progressive polyuria and polydipsia for approximately 10 months, without prior assessment of blood glucose levels. On arrival,

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she was unresponsive with a Glasgow Coma Scale score of 3/15, severely dehydrated, and in hypovolemic shock.

Initial arterial blood gas analysis demonstrated severe metabolic acidosis with a pH of 6.68 (reference: 7.35–7.45) and bicarbonate (HCO_3^-) of 2.9 mmol/L (reference: 22–26 mmol/L). Laboratory investigations showed marked hyperglycemia with a blood glucose concentration of 37 mmol/L (reference fasting range: 3.9–5.6 mmol/L; random <11.1 mmol/L), and HbA1c >14%, consistent with chronic, untreated hyperglycemia. Indicators of acute kidney injury (AKI) were present, including elevated urea (15 mmol/L) and serum creatinine (135 µmol/L) (reference ranges: urea 2.5–7.1 mmol/L; creatinine 44–88 µmol/L). SARS-CoV-2 infection was confirmed by rapid polymerase chain reaction testing.

The patient underwent immediate resuscitation and was subsequently intubated prior to transfer to the Pediatric Intensive Care Unit (PICU). She was diagnosed with severe diabetic ketoacidosis (DKA), COVID-19 pneumonia, encephalopathy, and AKI. Management was provided by a multidisciplinary team including pediatric endocrinology, nephrology, neurology, infectious disease, immunology, and PICU critical care specialists.

Treatment included a standardized DKA management protocol, mechanical ventilation, and continuous renal replacement therapy (two sessions) due to renal impairment. Neuroprotective strategies were initiated after brain computed tomography revealed cerebral edema. Her clinical course was complicated by severe metabolic acidosis, electrolyte disturbances, and challenges in fluid management due to co-existing AKI. She required high doses of vasopressor agents and advanced ventilatory support.

With comprehensive supportive management, the patient progressively stabilized. Cerebral edema resolved, renal function improved, and she was successfully weaned from mechanical ventilation. No clinical or laboratory findings suggested an underlying immunodeficiency.

After extubation and initiation of rehabilitation, the patient developed right foot drop and a high-stepping gait. Electromyography and nerve conduction studies revealed:

- Severe partial injury of the right peroneal nerve,
- Severe right sural neuropathy, and
- Moderate axonal sensorimotor neuropathy in the left lower limb.

These findings were consistent with a combination of diabetic neuropathy and critical illness–associated neuropathy. The patient continued follow-up care with endocrinology, neurology, nephrology, and physical therapy teams, and received structured diabetes self-management education.

Discussion:

This case highlights the potential of SARS-CoV-2 infection to precipitate new-onset type 1 diabetes mellitus (T1DM) in a previously healthy child, rapidly progressing to severe diabetic ketoacidosis (DKA). Growing evidence indicates that the COVID-19 pandemic has been associated with increased incidence of T1DM in the pediatric population. Two principal mechanisms have been proposed: [1] direct viral insult to pancreatic β-cells through angiotensin-converting enzyme 2 (ACE2) receptors expressed on islet tissue, and [2] immune dysregulation that promotes autoimmune β-cell destruction [9,10]. Large cohort analyses and meta-analyses have reported a higher rate of new T1DM diagnoses and greater frequency of DKA episodes in children following SARS-CoV-2 infection [4,5].

The prolonged history of polyuria and polydipsia in this patient suggests a pre-existing autoimmune process that may have been subclinical until the viral infection accelerated β-cell failure. Similar reports describe viral infection serving as a trigger that transforms latent autoimmunity into overt diabetes in genetically susceptible children

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[11,12]. The level of acidosis observed in this case (pH <6.8) is exceptionally severe and is associated with a higher risk of cerebral edema and systemic organ dysfunction [13]. The combined effect of viral invasion, cytokine-driven inflammation, and disruption of the renin–angiotensin–aldosterone system contributes to profound insulin deficiency and marked metabolic instability in COVID-19–associated T1DM [2,3].

Reports indicate that DKA occurring during or shortly after SARS-CoV-2 infection may be more severe and frequently necessitates intensive care support [5,14]. In parallel, acute kidney injury (AKI) is a well-recognized complication of critical COVID-19. Proposed mechanisms include renal hypoperfusion, cytokine-mediated injury, microvascular thrombosis, and possible direct infection of renal tubular cells expressing ACE2 receptors [15,16]. The development of AKI in this patient necessitated renal replacement therapy, reflecting the degree of multisystem involvement.

A particularly unusual feature of this case was the onset of acute peripheral neuropathy at the time of diabetes diagnosis. Classically, diabetic neuropathy develops years after T1DM onset; however, acute neuropathic injury can occur in the context of severe DKA, profound metabolic disturbances, or critical illness [17]. Increasing literature reports peripheral and autonomic neuropathies associated with COVID-19, including cases occurring alongside diabetic neuropathy or critical illness neuropathy [8]. The overlapping injury from hyperglycemia-related oxidative stress and SARS-CoV-2–related neuromuscular complications complicate rehabilitation and prolongs recovery [7,8].

This patient's clinical course demonstrates how SARS-CoV-2 can contribute simultaneously to endocrine, neurological, and renal dysfunction. Recognition of this association is essential because timely intervention—intensive metabolic correction, neuroprotective measures, and renal support—may reduce morbidity. Optimal outcomes require coordinated multidisciplinary care involving endocrinologists, neurologists, nephrologists, infectious disease physicians, and critical care specialists [18,19].

Conclusion:

This case highlights the ability of SARS-CoV-2 infection to precipitate new-onset type 1 diabetes mellitus in a child, presenting with severe diabetic ketoacidosis, acute kidney injury, and asymmetric sensorimotor neuropathy. The rapid clinical deterioration emphasizes the importance of early recognition of hyperglycemia and DKA in pediatric patients with COVID-19. Optimal outcomes require timely metabolic stabilization and coordinated, multidisciplinary management. Further research is essential to clarify the mechanisms by which SARS-CoV-2 accelerates β-cell autoimmunity and contributes to acute neurological complications in new-onset T1DM.

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