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REVIEW ARTICLE

The Impact of the COVID-19 Pandemic on the Diagnosis and Epidemiology of Type 1 Diabetes in Children

HIGHLIGHTS ▸ Global pediatric T1D incidence increased by 9.5% to 35% during the pandemic, with significant regional variations linked to healthcare infrastructure and containment policies. ▸ Diabetic ketoacidosis (DKA) frequency at diagnosis peaked between 39.4% and 68.2%, driven primarily by indirect structural disruptions rather than direct viral cytopathic effects. ▸ Seasonal chronobiological patterns were disrupted, with unprecedented summer surges in new diagnoses and insulin prescriptions observed across US and European cohorts. ▸ Advanced metabolic decompensation was characterized by lower venous pH, higher HbA1c (13.0 ± 1.71%), and increased PICU admissions, particularly among children without prior family history of diabetes. ▸ National mRNA vaccination campaigns correlated with a subsequent decline in new-onset T1D rates among adolescent cohorts, suggesting a protective epidemiological association. ▸ Future resilience requires integrating remote telehealth networks, standardized emergency subcutaneous insulin protocols, and proactive community screening into baseline pediatric care.

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ABSTRACT

Background. The SARS-CoV-2 pandemic strained pediatric networks, disrupting the presentation and monitoring of chronic metabolic conditions.

Aim of the study. This evaluation compiles global evidence on shifts in new-onset pediatric type 1 diabetes (T1D) incidence, DKA severity, seasonal deviations, and entry parameters during the COVID-19 era.

Material and methods. Data were harmonized from 14 global cohorts, capturing clinical profiles for >150,000 pediatric and adolescent subjects across the US, UK, Germany, Italy, Spain, Israel, and Canada.

Results. Multi-country datasets confirmed a significant 9.5% to 35% baseline expansion in new-onset T1D, with acute DKA frequencies peaking between 39.4% and 68.2% at admission. Metabolic decompensation advanced during lockdowns, marked by a downward shift in venous pH, a high frequency of clinical shock, and a substantial rise in glycated hemoglobin (HbA1c) at 13.0 ± 1.71%. Interrupted time-series models confirmed that indirect structural disruptions—such as containment-driven care delays—operated as the primary driver of advanced decompensation, rather than direct viral pathways. Vaccination interventions subsequently lowered post-pandemic trends among adolescent groups.

Conclusions. The pandemic accelerated pediatric diabetes severity due to widespread care delays. Mitigating future global shocks requires optimized screening rules, flexible intensive care insulin protocols, and remote telehealth networks.

KEYWORDS type 1 diabetes; COVID-19 pandemic; pediatric epidemiology; diabetic ketoacidosis; beta-cell tropism

1. Introduction

The metabolic stability of the pediatric population relies entirely upon the cellular integrity of insulin-producing pancreatic beta cells situated within the islets of Langerhans, whose continuous immune-mediated destruction defines the progression of type 1 diabetes mellitus (T1D) [1, 9]. Historically, the clinical onset of childhood T1D has been modeled as a multi-stage process where environmental factors trigger autoimmune pathogenetic dynamics in genetically predisposed individuals [1, 6, 9]. Among these environmental triggers, common respiratory and gastrointestinal viral agents—specifically enterovirus strains and coxsackievirus variants—have been widely identified as contributors to pancreatic autoaggression or acute cytolytic transformation [1, 6, 7]. The sudden outbreak of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pandemic in early 2020 altered this

epidemiological baseline, exposing pediatric endocrinology frameworks to substantial clinical, structural, and administrative challenges [3, 5, 6, 9].

Surveillance metrics recorded across multiple global healthcare jurisdictions highlighted a sharp escalation in both the absolute registration of new pediatric diabetes cases and the clinical severity of children presenting to emergency and intensive care units [1, 11, 12]. This baseline shift initiated a comprehensive scientific discussion regarding whether SARS-CoV-2 operates as a direct diabetogenic modifier accelerating beta-cell failure via specific direct cellular tropism, or whether the advanced clinical decompensation represents indirect collateral damage. Widespread public health containment measures, structural limitations inside primary healthcare networks, strict societal lockdowns, and parental hesitation to visit clinical facilities due to infection transmission fears may have extended the temporal window between initial metabolic disruption and formal clinical evaluation [3, 12, 13]. Systematically evaluating these direct biological pathways against indirect organizational barriers is critical for defining modern pediatric health policies and standardizing clinical networks ahead of future global healthcare emergencies [6, 9].

2. Literature Search Methodology

To ensure a robust and comprehensive synthesis of the empirical evidence, a structured literature search was conducted across the PubMed, Scopus, and Web of Science databases for scientific articles published between January 2020 and December 2025. The search architecture utilized precise combinations of the following Medical Subject Headings (MeSH) and text keywords: ("Type 1 Diabetes" OR "T1D") AND ("COVID-19" OR "SARS-CoV-2") AND ("pediatric" OR "children" OR "adolescents") AND ("incidence" OR "diabetic ketoacidosis" OR "DKA").

Studies evaluated in this review were required to meet the following pre-specified inclusion criteria: • Original peer-reviewed research, multicenter registry reports, longitudinal cohort studies, or systematic meta-analyses evaluating new-onset pediatric T1D during the core pandemic window (2020–2022) [1-15]. • Target study cohorts comprising pediatric, adolescent, or young adult populations within the age range of 0 to 26 years [3, 5, 7, 12, 13]. • Inclusion of a well-defined pre-pandemic historical control baseline spanning longitudinal control intervals (e.g., 2006–2019 datasets) to calculate long-term linear trends [3, 7, 12, 13].

Exclusion criteria comprised case reports or brief case series with fewer than five patients, narrative opinion pieces lacking empirical datasets, and studies focused exclusively on adult populations or the management of established, pre-existing T1D. A total of 14 major global datasets met these criteria and were selected for critical synthesis [1-14]. These data sources included an international multicenter database compiling records from 13 countries (encompassing Australia, Austria, Czechia, Denmark, Germany, Italy, Luxembourg, New Zealand, Norway, Slovenia, Sweden, Wales, and the USA) [3], the T1D Exchange Quality Improvement Collaborative (T1DX-QI) representing seven large U.S. clinical centers [12], the German Longitudinal Prescription Database (LRx) [13], the Israel National Childhood T1D Registry [7], the Canadian Alberta Children's Hospital and Stollery Children's Hospital multi-clinic databases [11], and the North Central and North West London Pediatric Diabetes Networks in the United Kingdom [8, 10].

3. Epidemiological Variations and Comparative Incidence

A synthesis of global data reveals a multi-regional increase in pediatric T1D presentations during the pandemic, though notable discrepancies exist between geographical regions, ethnic sub-populations, and study designs. A global random-effects meta-analysis compiling tracking metrics across the initial phase of the pandemic confirmed an absolute 9.5% increase in pediatric new-onset T1D registrations, with the overall baseline incidence shifting from a pre-pandemic mean of 19.73 cases per 100,000 children in 2019 to 32.39 per 100,000 during the pandemic observation window [1].

At the national registry level, substantial adjustments were verified, though their magnitude varied significantly by data capture methods and regional tracking frameworks: • **Italy:** An Italian tertiary referral unit registry (Bambino Gesù, Rome) recorded a 22% to 35% baseline expansion in new diagnoses during 2021 compared to the 2017–2020 mean, which was temporally concentrated within the final four months of 2021, tracking the trajectory of the national fourth viral wave [2]. • **Israel:** The Israel National Childhood T1D Registry demonstrated that the overall annual incidence rose from a pre-pandemic mean of 12.9 per 100,000 children to 17.7 per 100,000 in 2020 and 16.7 per 100,000 in 2021 across both sexes, particularly within prepubertal and pubertal age brackets [7]. • **Spain:** A regional multicenter database in the province of Tarragona identified a 60.8% case increase during 2020 compared to 2019, noting a highly uneven ethnic distribution marked by an increase in incidence among

the Maghrebi pediatric population (132.1 per 100,000 in 2020 vs. 52.2 per 100,000 in 2019) [4].

Critical Methodological Appraisal of Incidence Discrepancies

When appraising these findings critically, a clear methodological dichotomy emerges between European/Israeli datasets and certain North American cohorts. For instance, an interrupted time-series (ITS) analysis conducted in the US Mid-Atlantic region involving a cohort of 288,574 pediatric patients showed that the monthly rate of type 1 diabetes diagnosis remained stable and did not exhibit statistically significant changes (level change IRR = 0.97, p = 0.82) [5]. Concurrently, this same population experienced a parallel increase of 128% in the incidence of pediatric type 2 diabetes (T2D) and 142% for prediabetes [5].

This discrepancy can be explained by two distinct factors: 1. **Selection Bias vs. Population-Wide Sampling:** The Italian and Spanish data relied heavily on tertiary referral hospital registries [2, 4], which inherently capture acute presentations and are susceptible to selection bias. Conversely, the US Mid-Atlantic study analyzed a broad electronic health record (EHR) database within an integrated healthcare network [5], capturing a wider spectrum of milder presentations that did not require tertiary care. 2. **True Regional Variations in Confounding Lifestyle Shifts:** The rapid acceleration of childhood BMI velocities during lockdowns in the United States served as a strong driver for type 2 diabetes and prediabetes expression [5], potentially shifting the clinical presentation of metabolic disease compared to European cohorts where lockdown characteristics and baseline population genetics differed.

4. Metabolic Profiles and Diabetic Ketoacidosis Presentation Dynamics

The metabolic presentation of pediatric new-onset T1D during the pandemic was characterized by increased clinical severity at presentation [3, 6, 10, 11]. Diagnostic and biochemical protocols across all harmonized registries followed parameters established by the International Society for Pediatric and Adolescent Diabetes (ISPAD), where diabetic ketoacidosis (DKA) was defined biochemically by a plasma glucose concentration exceeding 200 mg/dL, a venous blood gas pH below 7.30, or a serum bicarbonate concentration below 15 mmol/L paired with documented ketonemia or ketonuria [1, 10, 11, 14]. DKA severity was stratified into mild (venous pH 7.20–7.30), moderate (pH 7.10–7.20), and severe (venous pH < 7.10 or serum bicarbonate < 5 mmol/L) [3, 11].

An international multicenter registry audit compiling data from 104,290 individuals across 13 countries established that the adjusted observed prevalence of DKA at initial diagnosis rose to 39.4% in 2020 and 38.9% in 2021 [3]. These figures significantly exceeded the projected benchmarks of 32.5% (2020) and 33.0% (2021) derived from long-term pre-pandemic linear trends ($p < 0.0001$), reflecting deep systemic disruptions in the continuity of preventive care [3].

Biochemical analysis across regional multicenter networks confirmed a downward shift in mean venous pH and serum bicarbonate metrics: • In a United Kingdom cohort (North Central London), new T1D presentation profiles showed a lower mean pH (7.09 ± 0.21 vs. 7.30 ± 0.13, $p = 0.001$) and a significantly higher presenting HbA1c concentration ($13.0 \pm 1.71\%$ vs. $10.4 \pm 3.21\%$, $p = 0.008$) during the first wave compared to pre-pandemic months, resulting in an absolute escalation in severe DKA rates (47% vs. 10%) [10]. • In Alberta, Canada, retrospective audits documented an absolute 22.6% increase in DKA frequency at T1D onset (68.2% in 2020 vs. 45.6% in 2019, $p < 0.001$), alongside a doubling of severe DKA rates (27.1% vs. 13.2%, $p = 0.01$) and a rise in critical care resource utilization via PICU admissions (17.8% vs. 7.69%, $p < 0.001$) [11].

Analysis of Protective Factors and Regional Anomalies

Crucially, the data identified specific protective parental and biological factors. Across the Canadian and U.K. cohorts, the rate of severe presentation and advanced DKA was lower among children with a documented first-degree relative with diabetes mellitus (33.3% vs. 75.2% in Canada, $p = 0.031$) [11]. Similarly, children who utilized home capillary blood glucose testing presented with significantly lower metabolic derangement, as parental awareness or incidental testing truncated the delay window.

Paralleling these findings, a critical limitation in generalizing these DKA increases is illustrated by a single-center retrospective audit at the Akdeniz Reference Center in Turkey, which revealed a unique preservation of baseline clinical patterns [14]. The local DKA frequency remained static (53.5% in 2020 vs. 56.4% in 2019, $p = 0.94$), and severe DKA proportions showed no significant deviation (43.3% vs. 45.4%, $p = 0.95$) [14].

This institutional anomaly can be explained by a statistical ceiling effect. Because the baseline pre-pandemic DKA frequency at this Turkish center was already exceedingly high (>55%) due to historical regional delays in healthcare access, there was very little statistical margin for a

further upward shift during the pandemic [14]. This contrasts sharply with the UK cohort, where the baseline DKA rate was low (10%), leaving a wide margin for a pandemic-induced surge [10].

5. Chronobiological Seasonality and Longitudinal Prescribing Patterns

The introduction of pandemic containment protocols coincided with a disruption of established chronobiological and seasonal configuration curves for pediatric T1D onset [12, 13]. Historically, newly diagnosed T1D exhibits a winter-dominant cyclical variation, with a high concentration of diagnoses between the months of December and February, and a predictable decline during summer intervals. While pre-pandemic control periods followed these trends, the 2020–2021 pandemic era showed a disruption of this cyclical structure [12, 13].

Multicenter data from the U.S. T1D Exchange Quality Improvement Collaborative showed that, between March and September 2020, considerably more children received a new T1D diagnosis than during the equivalent months of 2019 ($p < 0.001$), and a higher share of these children reached the clinic in a state of DKA within that same window ($p = 0.006$) [12]. This summer-phase inflation was verified in Germany through the IQVIA LRx database, which evaluates a population of 23,785 pediatric subjects [13]. Time-series models (ARMA) built on pre-pandemic datasets demonstrated that the expected seasonal drops during the summer months vanished during the pandemic [13]. The observed insulin prescription incidence—used as a surrogate metric for clinical T1D onset—was 44% higher in the summer of 2020 and 65% higher in the summer of 2021 than predicted by baseline historical trends [13]. The incidence rate ratios (IRR) for the summer periods of 2020 and 2021 rose to 1.44 (95% CI: 1.30–1.58) and 1.65 (95% CI: 1.49–1.82), respectively [13].

Limitations of Prescription Databases

A critical limitation of the German LRx dataset is its reliance on longitudinal prescription data as a surrogate for clinical onset [13]. While this method provides an immense sample size and eliminates hospital selection bias, it lacks granular clinical confirmation. It cannot definitively rule out cases where transient stress-induced hyperglycemia or early-stage type 2 diabetes cases were initially misclassified as T1D and prescribed insulin during acute viral stress, potentially inflating the documented summer incidence ratios.

6. Pathophysiological Interpretations: Direct Viral Tropism vs. Indirect Structural Pathways

The mechanisms linking the COVID-19 pandemic to alterations in pediatric metabolic presentation operate through two distinct modalities discussed in the literature: direct biological pathogenetic mechanisms and indirect structural or organizational healthcare dynamics [1, 3, 6, 9, 14].

Direct Biological Mechanisms

In vitro and in vivo studies have confirmed that human pancreatic islet cells show susceptibility to direct SARS-CoV-2 infection [1, 6, 9]. The direct molecular pathway requires the binding of the viral spike (S) glycoprotein to the cell-surface angiotensin-converting enzyme 2 (ACE2) receptor, co-localized with the transmembrane serine protease 2 (TMPRSS2) [6, 9]. Advanced transcriptional profiling has shown that beta cells demonstrate a high expression of alternative entry co-receptors, including neuropilin-1 (NRP-1), transferrin receptor protein 1 (TFRC), and FURIN, compared to neighboring alpha cells.

The activation of these receptors by SARS-CoV-2 entry initiates intracellular stress signals, triggering the phosphorylation and up-regulation of stress-response Mitogen-Activated Protein (MAP) kinases, specifically the c-Jun N-terminal kinase (JNK), p38 pathways, and p21-activated kinases (PAK) [6, 9]. This signaling cascade induces subcellular damage, characterized by vacuolization of the endoplasmic reticulum-Golgi complex, leading to beta-cell death via intrinsic apoptotic and necroptotic pathways [6, 9]. Concurrently, viral entry is associated with beta-cell transdifferentiation through a protein kinase R (PKR) and eukaryotic translation initiation factor 2a (eIF2a)-mediated integrated stress response, causing functional, insulin-secreting beta cells to lose their phenotypic identity and transdifferentiate into glucagon-producing alpha cells or trypsin-secreting acinar cells [6, 9]. Furthermore, down-regulation of functional ACE2 expression blocks the degradation of angiotensin II, altering the renin-angiotensin-aldosterone system (RAAS) to drive adrenal aldosterone release and subsequent renal potassium wasting, which explains the severe, refractory hypokalemia documented in some pediatric COVID-19-positive DKA admissions [8, 10].

The Epidemiological Discrepancy

When evaluating these cellular findings against population-level data, a significant epidemiological discrepancy arises. If direct viral cytopathic destruction were the primary driver of the documented T1D incidence spikes, time-series cross-correlation analytics would demonstrate a direct, time-lagged association between weekly regional COVID-19 viral infections and the corresponding velocity of new T1D diagnoses [3, 6, 13]. However, rigorous mathematical modeling across large-scale systems, such as the German LRx database analysis, showed an absence of direct cross-correlations or time-lagged synchronization between national viral infection density and new-onset curves [13].

This lack of correlation shifts scientific focus toward the indirect structural pathway. Multivariable log-binomial regression models have demonstrated a significant, direct correlation between the likelihood of advanced DKA presentation and public health containment stringency, as measured by the Oxford Stringency Index ($p < 0.0001$ in 2020) [3]. Societal containment lockdowns, school closures, and the systemic reorganization of healthcare facilities away from routine preventive care created structural bottlenecks [3, 6, 9, 11]. Widespread parental reluctance to visit hospitals due to transmission fears ("corona-phobia") further widened the diagnostic window. Consequently, mild osmotic indicators went undetected, allowing early-stage hyperglycemia to progress unmonitored over weeks, as evidenced by significantly elevated HbA1c levels (13.0%) and high proportions of severe, late-stage DKA at clinical presentation [10, 11, 12].

Additionally, the psychological impact of prolonged containment isolation stimulated the hypothalamic-pituitary-adrenal axis, driving a sustained release of counter-regulatory stress hormones (cortisol, epinephrine) that accelerated hepatic gluconeogenesis and peripheral insulin resistance [5]. This neuroendocrine activation, combined with rapid containment-driven lifestyle shifts that increased childhood BMI velocities, accelerated metabolic collapse in children with pre-existing, pre-clinical autoantibody positivity, precipitating clinical decompensation [5].

7. Clinical Framework Modifications, Technology, and Vaccine Associations

The challenge of handling severe metabolic crises alongside active or prior viral exposure required structural modifications within pediatric intensive care environments. The severe clinical presentation profiles of pandemic-era DKA required precise fluid and electrolyte replacement strategies to mitigate the risk of cerebral edema associated with profound acidosis [6, 9, 11]. Crucially, in resource-limited or emergency settings where intensive care infrastructure faced capacity constraints, clinical trials validated the safety and efficacy of rapid-acting subcutaneous insulin regular or analog regimens as a viable alternative to continuous intravenous infusions for managing mild-to-moderate pediatric DKA states [6, 9].

This transition was supported by the adoption of digital technologies. Interstitial continuous glucose monitoring (CGM) systems, real-time sensor arrays, and integrated smart insulin pumps served as tools for tracking volatile glycemic metrics, enabling remote monitoring and reducing physical contact requirements in isolation wards [6, 9]. Concurrently, outpatient diabetes management expanded toward telemedicine and virtual-first care networks, which proved effective for stabilizing glycemic control, conducting remote nutritional counseling, and reducing diabetes-related distress among families during lockdowns [6, 9].

Finally, longitudinal tracking into the later phases of the pandemic revealed a significant epidemiological shift following the introduction of national preventive countermeasures. Population-level monitoring across the Israel National Childhood Registry and European networks established that the implementation of national mRNA-BNT-162b2 immunization campaigns was strongly linked to a subsequent decline in new-onset pediatric T1D presentation rates [2, 7]. Specifically, among pubertal and adolescent cohorts (the 10–14 and 15–17 age groups), who maintain the highest national vaccination coverage rates, the incidence of new T1D presentations dropped significantly during 2022 compared to the pre-vaccination waves of 2020–2021 ($p = 0.03$ and $p = 0.02$ respectively) [7]. This epidemiological correlation indicates that by truncating the systemic inflammatory cascade and severe outcomes associated with native viral exposure, active immunization rollouts were chronologically associated with a modification in the presentation velocity of childhood diabetes, though direct causal links remain to be definitively established through prospective mechanistic trials [6, 7, 9].

8. Discussion and Future Regenerative Perspectives

The extensive data synthesized across these 14 global cohorts underscores a critical vulnerability in pediatric healthcare: the extreme dependency of metabolic homeostasis on a highly finite population of functional beta cells [1-13]. When public health shocks disrupt early screening networks, the diagnostic window widens, resulting in severe metabolic decompensation at onset [3, 10, 11]. The profound degree of beta-cell damage and transdifferentiation observed under viral or environmental stress highlights the limitations of current insulin-centric therapeutic models, which manage symptoms without addressing the underlying loss of cellular mass [6, 9, 15].

Consequently, contemporary diabetology literature increasingly emphasizes the necessity of developing long-term regenerative interventions to rescue or replace destroyed islet architectures [15, 16]. The clinical surges in T1D severity recorded during the pandemic highlight the value of advanced regenerative strategies, such as stem cell therapeutics and cell-replacement engineering [16]. Specifically, recent progress in the differentiation of human pluripotent stem cells (hPSCs) into functional, insulin-secreting beta-like cells provides a prospective pathway to mitigate the permanent metabolic consequences of accelerated autoaggression [16]. Integrating these emerging stem cell platforms with advanced biomaterials to prevent immune-mediated destruction represents a primary objective for future clinical practice [16]. Transitioning from reactive intensive care protocols to active cell-replacement frameworks may provide a definitive shield for vulnerable pediatric cohorts against future global environmental or epidemiological provocations.

9. Conclusions

The COVID-19 pandemic coincided with epidemiological instability and an escalation in presentation severity within the pediatric type 1 diabetes landscape globally, exposing vulnerabilities in early clinical screening mechanisms. The documented clinical shifts were strongly associated with indirect structural pathway breakdowns—most notably lockdown containment bottlenecks, parental care avoidance, and delayed clinical contact windows—rather than direct cytopathic viral pathways. Proactive community screening campaigns, standardized emergency subcutaneous insulin care pathways, and integrated remote digital

telehealth networks must be permanently incorporated into baseline pediatric practice to protect vulnerable childhood cohorts during future global health emergencies.

Disclosure

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