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RESEARCH ARTICLE

# The influence of the COVID-19 pandemic on pediatric hospitalizations for type 1 diabetes mellitus

Măriuca Mănescu[1], Ion-Bogdan Mănescu[2*], Alina Grama[1,3]

1. Department of Pediatrics, Emergency County Clinical Hospital of Targu Mures, Targu Mures, Romania
2. Department of Laboratory Medicine, George Emil Palade University of Medicine, Pharmacy, Science, and Technology of Targu Mures, Targu Mures, Romania
3. Department of Pediatrics, George Emil Palade University of Medicine, Pharmacy, Science, and Technology of Targu Mures, Targu Mures, Romania

**Objective**: In this retrospective cohort study, we aimed to provide a snapshot of how the pandemic has affected pediatric type 1 diabetes mellitus (T1D) admissions in our hospital.
**Methods**: This study included 117 patients aged 0-18 classified based on period (pre-pandemic vs. pandemic period 2020-2022) and type of diagnosis at admission: new-onset T1D (nT1D) or diabetic ketoacidosis (DKA)-decompensated T1D. We investigated the effect of the COVID-19 pandemic on the demographic, clinical, and laboratory characteristics of these patients.
**Results**: Out of all T1D-related admissions, the proportion of admissions for nT1D increased compared to the pre-pandemic period: 71.6% vs 53.4%, p=0.048. Unrelated to the pandemic, the type of diagnosis at admission was associated with 1) the sex distribution (males – more nT1D admissions, females – more frequent DKA admissions, p=0.01), and 2) hospitalization duration (longer for nT1D admissions than for DKA-decompensated T1D admissions, p=0.001). Blood glucose and HbA1c levels were influenced neither by the pandemic period nor by the type of diagnosis. During the pandemic, a change in the T1D seasonality became apparent. A potential association pattern between new COVID-19 cases, number of T1D admissions, and stringency of restrictions was observed.
**Conclusions**: During the COVID-19 pandemic, the proportion of nT1D admissions increased, as well as the severity of DKA-decompensated T1D cases. In addition, the pandemic period brought about notable shifts in the seasonality of pediatric T1D.

**Keywords**: COVID-19 pandemic, demographics, diabetic ketoacidosis, pediatrics, type 1 diabetes mellitus

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## Introduction

Type 1 diabetes (T1D) is a heterogeneous disorder caused by a complex interaction between genetic and environmental factors. T1D is most commonly observed in children and young adults, being one of childhood's most prevalent chronic diseases [1]. The International Diabetes Federation estimates that over 1.2 million children and adolescents worldwide are affected by T1D [1]. In most cases, an autoimmune-mediated process leads to the destruction of pancreatic beta cells, resulting in insulin deficiency and hyperglycemia.

Despite centuries of studies and therapeutic advancements, diabetes mellitus remains an incompletely understood incurable chronic disease. However, extensive research and robust studies have provided support for the relationship between genetic susceptibility and various environmental factors, including infections, ethnicity, weight, diet, vitamin D deficiency, geographic location, and microbiota [2, 3].

Although T1D has a strong genetic component, evidence suggests the substantial impact of environmental factors on the risk of developing the disease, such as the increasing incidence observed in recent decades, that is in the prepandemic period [4], the discordant onset and evolution of T1D in monozygotic twins [5], and the alignment of disease incidence in migrating populations with the rates of their destination regions [6].

Viral infections such as rubella, mumps, Coxsackie, and cytomegalovirus have been extensively studied as potential diabetogenic factors and have been associated with an increased risk of T1D. These viruses are often found in the pancreatic islets of individuals with T1D [2, 7, 8]. Recently, researchers have been raising awareness about the potential trigger role of SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2) for T1D. Observations that SARS-CoV-2 can enter and damage pancreatic beta cells, leading to diabetic-level of hyperglycemia [9, 10], have sparked discussions about new-onset diabetes in the context of COVID-19. Several studies have reported a rise in both the incidence and severity of pediatric T1D during the COVID-19 pandemic [11-14], but whether these findings are significant and/ or completely attributable to the SARS-CoV-2 virus itself is still unclear. As the COVID-19 pandemic loosens its grip, it is crucial to analyze its impact on the diabetic population and explore its relation to the T1D „silent epidemic".

This study aims to provide a snapshot of how the pandemic has affected pediatric T1D admissions in our hospital. We aimed to describe the demographic, clinical, and laboratory characteristics of T1D patients admitted to the Department of Pediatrics during the COVID-19 era compared to previous years.

* Correspondence to: Ion-Bogdan Mănescu
E-mail: bogdan.manescu@umfst.ro

## Methods

### Study design and data collection

This single-center retrospective cross-sectional cohort study adheres to the STROBE research reporting guidelines. The study included all pediatric (age 0-18 years) T1D-related admissions to the Department of Pediatrics of a Romanian tertiary hospital between 2016-2022. All T1D-related admissions were classified as either new-onset T1D (nT1D), with or without diabetic ketoacidosis (DKA) at presentation, or DKA-decompensated T1D (previously diagnosed). The timeline was defined based on the first confirmed COVID-19 case in Romania, which was reported on February 26, 2020. Thus, the pandemic period was defined as starting from February 1, 2020, while the preceding years were considered the pre-pandemic period.

The diagnoses of T1D and DKA were established according to current guidelines set by The American Diabetes Association. DKA severity was determined based on the degree of acidosis using the 2009 consensus statement from the American Diabetes Association: mild (pH>7.25 and HCO3≥15 mmol/L), moderate (pH 7.00–7.24 and HCO3 10–15 mmol/L), and severe (pH<7.00 and HCO3<10 mmol/L).

Demographic/ clinical information and laboratory findings were retrospectively obtained from patient charts and electronic database records. No anthropometric data were collected. Overall, we included 74 patients from the pandemic period and 43 patients from the pre-pandemic period.

Data on the severity and evolution of government restrictions in Romania throughout the pandemic was expressed in the form of the Oxford COVID-19 Stringency Index, which "quantifies the strictness of government policies using nine metrics: school closures, workplace closures, cancellation of public events, restrictions on public gatherings, closures of public transport, stay-at-home requirements, public information campaigns, restrictions on internal movements, and international travel controls" [15].

This study was approved by the hospital's institutional Ethical Research Committee (approval no. 16139/2023).

### Statistical analysis

Microsoft Excel (Microsoft Corporation) was used for data storage and processing, adaptation of COVID-19 Stringency Index data [15], and generation of Figure 2. Statistical processing was performed using MedCalc® Statistical Software version 20.104 (MedCalc Software Ltd, Ostend, Belgium; 2022). All data sets were tested for normality using the Kolmogorov-Smirnov test. Based on the results, an initial comparison of the parameters shown in Table I as well as the comparison of blood parameters were performed using either the independent samples t-test or the Mann-Whitney test. The Chi-squared test was used to compare the diagnosis type at admission and DKA severity between periods, as well as the sex distribution based on the diagnosis. For the effect of period and diagnosis type on hospitalization length, the two-way analysis of variance (ANOVA) test was used. The significance threshold was set at $p<0.05$.

## Results

A total of 117 cases were classified based on period (pre-pandemic vs pandemic) and type of diagnosis at admission: nT1D with/ without DKA vs DKA-decompensated known T1D. Demographic and hospitalization data are presented in Table I.

In the pre-pandemic period, 53.4% of all T1D-related admissions were for nT1D, with 41.8% presenting with DKA and 11.6% without DKA. During the pandemic, 71.6% of T1D-related admissions were for nT1D, with 41.9% presenting with DKA (similar to the pre-pandemic period) and 29.7% without DKA (significantly higher). Overall, the proportion of nT1D cases among T1D-related admissions was significantly higher during the pandemic compared to the pre-pandemic period (chi-squared p=0.048) (Figure 1A). This increase was primarily due to a rise in nT1D admissions without DKA during the pandemic.

Despite slight increases in the proportions of admitted males and age at admission during the pandemic period, the overall sex and age distribution did not show significant differences between the two periods (Table I). The distribution of age at onset for nT1D remained largely unchanged over the study period, with a small but non-signif-

Table I. Demographic and hospitalization data of pediatric admissions for T1D

| | | Pre-pandemic period (n=43) | Pandemic period (n=74) |
|---|---|---|---|
| Sex (male) | | 41.9% | 47.3% |
| Age at admission (years, mean ± SD) | | 9.7 ± 5.0 | 10.2 ± 4.5 |
| Age at onset (years, mean ± SD) | | 8.5 ± 4.7* | 9.1 ± 4.4* |
| Symptoms before admission (days, median [IQR]) | nT1D | 14.0 [7.0-28.0] | 21.0 [7.7-55.5] |
| | DKA | 1.0 [1.0-3.0] | 1.0 [1.0-2.2] |
| Admission for nT1D | no DKA | 11.6% | 29.7% |
| | with DKA | 41.8% | 41.9% |
| Admission for DKA only | | 46.6% | 28.4% |
| Hospitalization duration (days, median [IQR]) | nT1D | 8.0 [6.2-9.0] | 6.0 [5.0-7.0] |
| | DKA | 5.0 [2.5-5.5] | 3.0 [3.0-4.0] |

All T1D-related admissions were divided in categories based on the time of admission (pre- or during the pandemic) and diagnosis (nT1D ± DKA or DKA-decompensated previously diagnosed T1D). Abbreviations: DKA – diabetic ketoacidosis, IQR – interquartile range, n – number of patients, (n)T1D – (new onset) type 1 diabetes mellitus, SD – standard deviation. *The new onset T1D subgroup consists of 76 patients (23 in the pre-pandemic period, 53 in the pandemic).

icant increase in the pandemic period (Table I). However, sex-related differences non-related to the pandemic were observed depending on the diagnosis (Figure 1B). Overall, more male patients were admitted with nT1D, but had fewer consequent presentations for DKA, while female patients had a less favorable clinical profile after diagnosis, with more frequent DKA-decompensated T1D admission (chi-squared p=0.010).

The duration of symptoms before nT1D presentation seemed to be longer in the pandemic period, but the difference did not reach statistical significance. Two-way ANOVA analysis revealed no significant effect of the pandemic period on the duration of hospitalization. Conversely, the diagnosis was shown to significantly influence the duration of hospitalization regardless of the period, with nT1D patients having significantly longer hospitalizations than those admitted with DKA-decompensated T1D (p=0.001, see Figure 1C). The clinical presentation features and frequency of symptoms reported by nT1D patients were as follows: polyuria 85.5%, polydipsia 85.5%, weight loss 72.4%, fatigue 43.4%, polyphagia 23.7%, nausea/ vomiting 21.1%, abdominal pain 18.4%, somnolence/ lethargy 15.8%, loss of appetite 14.5%, headache 7.9%, dizziness 6.6%, shortness of breath 6.6%, arthralgia/ myalgia 2.6%, urinary tract infection 2.6%, diarrhea 1.3%, blurred vision 1.3%, genital infection 1.3%.

The COVID-19 pandemic had no significant effect on the concentrations of relevant blood parameters such as glucose, HbA1c, pH, bicarbonate, base excess, potassium, and lactate (see Table II). Also, there were no significant associations between these analytes and the type of diagnosis at admission (data not shown).

Despite the relative decrease in DKA-decompensated T1D admissions, the percentage of cases with moderate and severe DKA was significantly higher during the pandemic (Figure 1D). In DKA, bicarbonate, potassium, and lactate levels did not show significant differences between the two periods (data not shown).

Regarding seasonal variability, pre-pandemic T1D admissions seemed to peak during winter and spring, with an additional increasing trend in June and July. Most T1D admissions were noted in April, followed by December, while May and September had the fewest admissions. During the COVID-19 pandemic, a change in the seasonality of T1D became apparent, with an additional peak frequency of admissions in September. The monthly variation of T1D admissions in the pre-pandemic and pandemic periods is presented in Figure 2, alongside the daily average number of confirmed COVID-19 cases for each month and the Oxford COVID-19 Stringency Index. Based on visual indications from Figure 2, which suggest a 1-month lagged increase in the percentage of T1D-related admissions following a rise in new COVID-19 cases, we conducted a correlation analysis with progressively increasing lag times from 0 to 6 months. The results of this lagged correlation analysis are as fol-

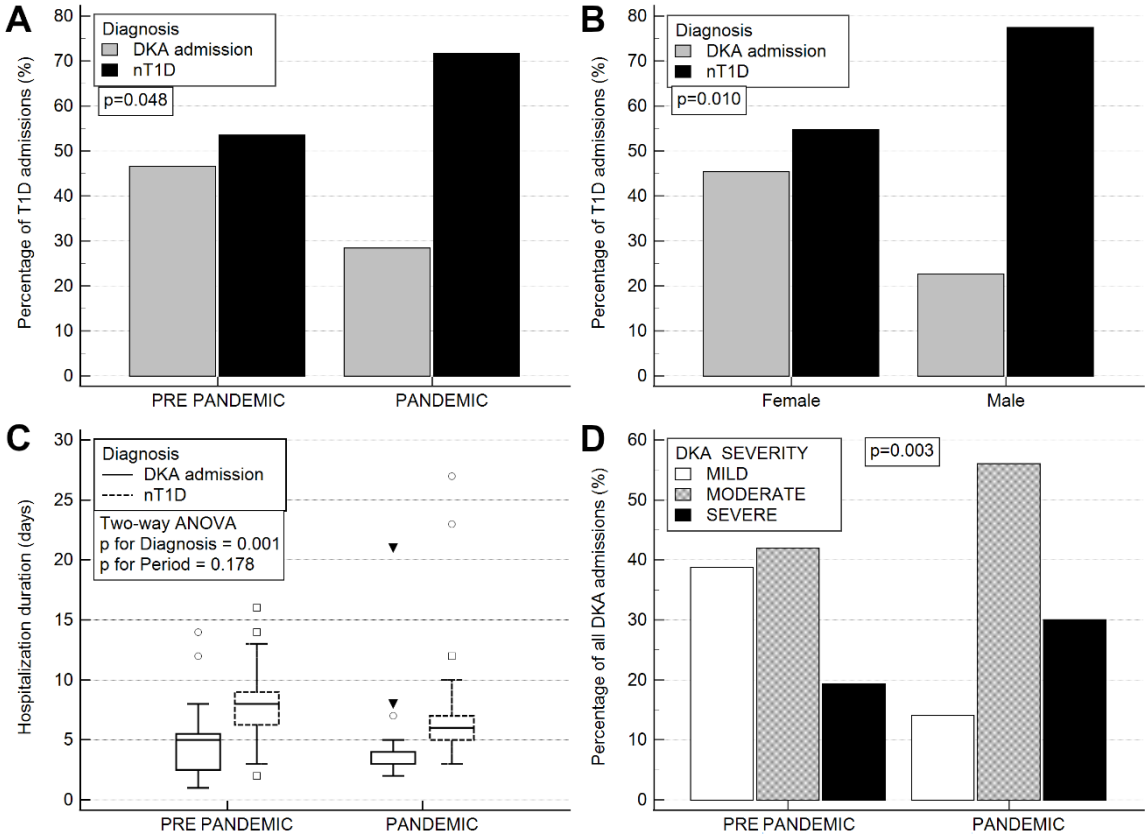


Fig. 1. Graphical representation of the statistical comparisons performed. A – between-period comparison of type of diagnosis at admission. B – between-sex comparison of type of diagnosis at admission (overall period, not pandemic-related). C – two-way analysis of variance (ANOVA) for the influence of period and type of diagnosis on hospitalization duration. D – between-period comparison of diabetic ketoacidosis (DKA) severity.

Table II. Comparison of laboratory findings between the two periods

| | Pre-pandemic period | Pandemic period | p value |
|---|---|---|---|
| Glucose (mg/dL) | 420.5 [306.0-518.0] n=42 | 421.0 [320.0-498.0] n=74 | 0.92 |
| HbA1c (%) | 11.4 [10.4-12.7] n=31 | 11.8 [10.3-13.1] n=69 | 0.57 |
| pH value | 7.17 [7.05-7.29] n=26 | 7.17 [7.05-7.34] n=66 | 0.47 |
| Bicarbonate (mmol/L) | 10.4 [7.2-16.4] n=26 | 9.2 [4.7-18.2] n=66 | 0.49 |
| Base excess (mmol/L) | -16.9 [-24.1 to -10.1] n=26 | -18.8 [-25.3 to -7.3] n=66 | 0.92 |
| Potassium (mmol/L) | 4.00 [3.75-4.55] n=21 | 4.19 [3.80-4.87] n=63 | 0.34 |
| Lactate (mmol/L) | 1.79 [1.11-3.11] n=18 | 2.05 [1.10-3.25] n=40 | 0.84 |

The Mann-Whitney test was applied for all comparisons. Results are expressed as median value with [interquartile range].

lows: r=0.19, p=0.27 with no lag; r=0.42, p=0.01 with a 1-month lag; r=0.09, p=0.61 with a 2-month lag; r=-0.15, p=0.37 with a 3-month lag; r=-0.06, p=0.71 with a 4-month lag; r=0.13, p=0.44 with a 5-month lag; and r=0.05, p=0.76 with a 6-month lag.

## Discussions

T1D is one of the most challenging health problems of our century, with a continuously increasing incidence, a great economic burden, issues of misdiagnosis or late diagnosis, a high risk of complications, and premature mortality. To address these challenges, untangling and better understanding the complex etiology of T1D are becoming priorities for clinical practice and public health.

The most widely accepted theory is that viral infections enhance or induce autoimmune diseases such as T1D. Extensive research suggests that viral infections play a significant role in the development of T1D [7, 8, 16, 17]. However, while there is considerable evidence of viral presence in the pancreas of T1D patients, viral exposure is not always detrimental and may, in fact, have protective effects depending on the virus and patient susceptibility to infections [18, 19]. Given the increasing incidence of viral infections, it is important to sort through the sometimes-divergent information regarding the autoimmunity-promoting effect of viruses.

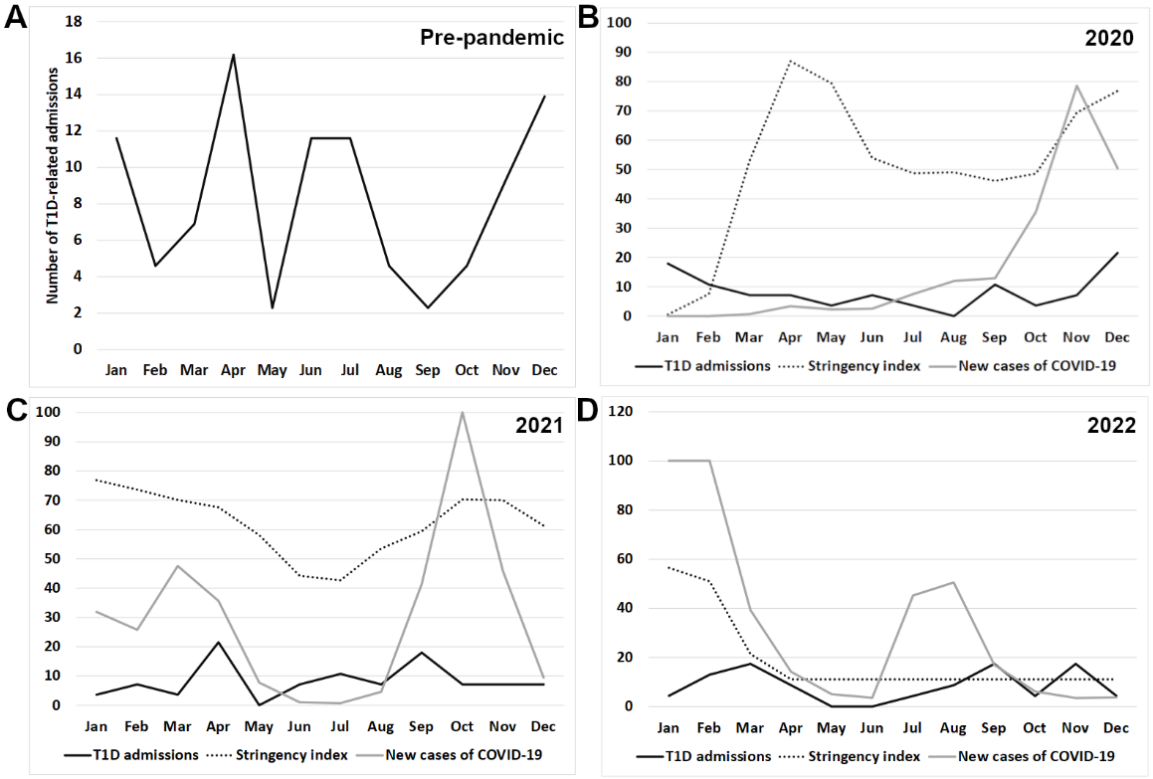


Fig. 2. Seasonal variation of T1D in the pre-pandemic and pandemic periods. Monthly T1D admissions are expressed as percentage (%) of yearly total admissions. The monthly stringency index (value from 0 to 100, 100 being the strictest) is expressed as the average of daily stringency indices (according to Mathieu et al. [15]). For scaling purposes, monthly new cases of COVID-19 are expressed as $10^{-2}$ × the average of daily new cases of COVID-19 (according to Mathieu et al. [15]) and scaled values were capped at 100, which did not affect the visible trends and patterns.

SARS-CoV-2 is a coronavirus that emerged in late 2019, causing the COVID-19 pandemic. SARS-CoV-2's genome revealed its close relation to SARS-CoV-1 and Middle East Respiratory Syndrome-related Coronavirus (MERS-CoV), which were responsible for the 2003 and 2012 respiratory outbreaks, respectively [20]. During these prior coronavirus epidemics, a causal relationship between coronaviruses and insulin-dependent diabetes was suggested due to high expression of ACE2 receptors in endocrine pancreatic tissues [20-22]. Several SARS-CoV-1 studies found that patients with no history of diabetes, who received no corticosteroids during the course of the disease, had developed insulin-dependent diabetes, sometimes with atypical presentation [23, 24]. The same phenomenon occurred again during the COVID-19 pandemic, reigniting the theories about coronavirus-mediated islet cell damage. Given its global scale, the COVID-19 pandemic has provided undeniable proof of association between coronaviruses and diabetes. Diabetes rates increased significantly during the pandemic and many cases of COVID-associated diabetes were classified as T1D [13, 14, 25, 26]. The course of the disease was variable, sometimes with transitory, self-resolving episodes, leading at other times to potentially life-threatening DKA and persistent diabetes. Moreover, several studies reported increased severity of T1D during the pandemic, both in nT1D and pre-existing diabetes [13, 27, 28].

**Newly-diagnosed T1D**

It should be noted that the study was conducted in a tertiary hospital, which, in addition to routine hospitalizations, handles all emergency cases from several counties, thereby serving a large population. Specifically, this includes pediatric emergencies related to T1D as well. Our findings were consistent with the literature as the percentage of pediatric nT1D cases admitted to our hospital significantly increased during the pandemic period. The large proportion of nT1D patients, as well as the increasing severity of the cases, suggest a potential diabetogenic effect of COVID-19. In light of previous experiences with coronaviruses, it may be speculated that SARS-CoV-2 could alter glucose metabolism through three main mechanisms: 1) an important severe illness-associated stress response, leading to excessive release of hyperglycemic hormones; 2) an inflammatory response associated with increased insulin resistance; 3) hyperglycemic medications used in the treatment of COVID-19 (e.g. corticosteroids). Several studies are now trying to establish the exact mechanisms through which COVID-19 induces or alters metabolic disorders, such as T1D, but the results are not yet conclusive. Currently, there is no substantial evidence to suggest that the virus itself can directly induce T1D [29-31]. Unfortunately, epidemiological data on SARS-CoV-2 infection and/ or vaccination was not available for all patients included in this study.

The indirect effects of COVID-19 should also be considered, as the pandemic has brought significant lifestyle changes, which can potentially affect glycemic control. During illness or periods of containment/ restriction, individuals may have experienced altered eating patterns, leading to a negative impact on blood sugar levels. Other factors such as disrupted sleep patterns, daily routine changes, lack of physical activity, and emotional stress due to social distancing, anxiety, grief, and loss can potentially trigger the onset or exacerbation of the condition in genetically predisposed individuals.

**The severity of diabetic ketoacidosis**

Another interesting observation was the rreduced number of DKA hospitalizations during the pandemic, but with increased severity (Figure 1A, 1D). We hypothesize that public health measures enacted during the pandemic have reduced exposure to other seasonal viruses associated with T1D decompensation, resulting in a decrease in DKA cases. Additionally, school closures may have had a beneficial impact on maintaining a healthy diet and a regular distribution of meals, resulting in better glycemic control in some patients. However, the significantly increased DKA severity may reflect delays in seeking medical care due to various reasons such as fear of exposure, restricted access, or overwhelmed healthcare systems. Moreover, as mentioned earlier, COVID-19 itself can disrupt glycemic control, leading to an increased risk of ketoacidosis [32].

**The demographics of T1D patients**

The age distribution of nT1D patients remained largely unchanged in the two studied periods, with a small, but not significant, increase in the age at onset during the pandemic, and a peak age at diagnosis overlapping with the pubertal onset period in children. Thus, the role of sex hormones in the relevant pathways associated with the pathophysiology of T1D should be further studied. While the exact reasons for this trend are not fully understood, several factors may contribute to the increased diagnosis of T1D during puberty: 1) the characteristic hormonal changes and fluctuations – increased levels of growth hormone, sex hormones, and adrenal hormones; 2) the physiologically increased insulin resistance, which can potentially expose underlying genetic predispositions; 3) exposure to certain environmental factors or infections.

In terms of sex distribution, males account for most nT1D admissions, regardless of the studied period, while females tend to have more severe clinical features, with a higher prevalence of DKA. These sex-related disparities have also been observed on a population level in other studies [33-36]. A 2023 systematic review noted that sex disparities exist in certain aspects of care, such as glycemic control, treatment adherence, DKA frequency, and quality of life, with young female individuals exhibiting less favorable outcomes compared to males [36]. Future research is needed to obtain a clearer understanding of the complex interplay between biological, hormonal, genetic, environmental, and psychosocial factors contributing to this gender gap in T1D clinical manifestations.

### Hospitalization duration and glycemic control

Our study also provides valuable insights into the clinical characteristics of T1D during the pandemic compared to the pre-pandemic period. It highlights potential differences in the duration of symptoms prior to admission, suggesting a potential delay in seeking medical attention, possibly influenced by various factors related to the pandemic, such as restricted access to healthcare or concerns about exposure to COVID-19. Regarding hospital stay, we observed a trend towards shorter hospitalization during the pandemic, but the difference did not reach statistical significance. This may be attributed to several factors, such as the need to minimize exposure to the virus, changes in treatment approaches and clinical management, limited hospital bed availability, and limited resources. All these factors may have influenced the decision to discharge patients earlier, provided their condition was stable.

Although our study does not indicate a significant effect of the COVID-19 pandemic on blood glucose and HbA1C levels, the existing literature presents various findings. While some studies also suggest no significant effect [37, 38], others have reported improvements in glycemic control during the pandemic [39, 40], while others still indicate a significant increase in HbA1C and blood glucose levels [41, 42]. The available evidence suggests a substantial variability in the effects of COVID-19 on glycemic control, which can be influenced by various factors such as age, comorbidities, socioeconomic status, differences in healthcare access, and clinical management. Therefore, it is essential to adopt a more nuanced approach when interpreting and generalizing the impact of COVID-19 on glycemic control.

### The seasonality shift of T1D

Our research provides insights into the seasonality of T1D, revealing a distinct pattern in disease development (Figure 2). Consistent with other pre-pandemic studies, we found pre-pandemic incidence peaks during the winter, spring, and summer months [43, 44]. Several factors, including viral exposure, environmental triggers, and sun exposure-driven vitamin D variations, have been proposed as potential contributors to this seasonal trend [45-47]. However, during the pandemic period, notable pattern changes were observed. We noticed a new peak of T1D admissions in September, which seemed unaffected by the level of restrictions implemented in our country as it appears under moderate-to-high restrictions in 2020-2021, but also under minimal restrictions in 2022. The surge in T1D cases observed during this period may be primarily attributed to the influence of the virus itself and its transmission dynamics within the population. Several contributing factors may have played a role in this increase, such as the reopening of schools which facilitated viral transmission among children and adolescents. Additionally, the easing of restrictions during the summer months (especially August) for travel and tourism, might have led to a higher rate of COVID-19 infections, with many people returning from vacations by the beginning of September. As a result, the combination of increased viral transmission, certain delayed effects of reduced vigilance, and overcrowding during summer vacations could have collectively contributed to the peak in COVID-19 cases during autumn, subsequently impacting the incidence of T1D cases.

Another intriguing observation was the absence of the March-April peak in 2020, despite its recurrent presence before the pandemic and in the subsequent pandemic years of 2021-2022. The cancellation of this peak might be attributed to various social factors present only at the beginning of the pandemic when data on the SARS-CoV-2 virus (e.g. transmissibility, mortality) were scarce and oftentimes exaggerated or contradictory. Thus, fear of exposure to the virus and mass panic may have resulted in excessive voluntary social isolation and delays in seeking medical attention.

Upon analyzing Figure 2, we emphasize a possible evolution pattern between the number of new COVID-19 cases, the stringency of containment measures, and the incidence of T1D admissions at our hospital. Figure 2 visually suggests a one- or two-month delay between the increase in SARS-CoV-2 infections and the subsequent rise in the T1D admission rate, followed by an increase in the stringency of restrictions, thus indicating a potential cause-effect relationship between these factors. Following this line of inquiry, we conducted lagged correlation analyses spanning from 0 to 6 months. We observed a moderate yet statistically significant correlation only when considering a 1-month lag between the surge in new COVID-19 cases and the subsequent uptick in T1D-related admissions. As reported in the Results section, this correlation greatly outperformed those observed at other lag intervals. Notably, this finding resonates with the broader pathophysiological and epidemiological framework suggesting a potential link between viral infections and the onset of T1D. As mentioned above, it is well known that viral infections can trigger or exacerbate diabetes, but there is a delay between exposure to the virus and the onset of pancreatic disease. Additionally, we cannot ignore the fact that some individuals who recovered from COVID-19 may have experienced „long COVID" or „post-COVID" syndrome, with long-term complications that may increase the risk of developing diabetes or exacerbate pre-existing diabetes. Given that the implementation of restrictions was reactive rather than proactive, it is not surprising that the peak of COVID-19 infections, closely followed by the peak of T1D admissions, led to increased restrictions. Consequently, the number of admissions remained high during the initial part of the restriction periods, and the effect of these measures started to become apparent only after a certain period of delay. This phenomenon is commonly referred to as „the lag effect" and has important implications during an outbreak, helping epidemiologists better understand the dynamics of the disease and predict future trends [48]. Future studies

of the lag effect in the context of the pandemic could provide valuable insights for better intervention planning and evidence-based decision-making.

Several studies have reported on the influence of the COVID-19 pandemic on pediatric T1D patients after studying various periods of the pandemic. However, to our knowledge, the present study is the first to investigate an almost three-year period of the pandemic, spanning the critical years of 2020-2022. Thus, we delved into the most active and consequential period of the pandemic, capturing a relatively wide range of variations induced by this period. These encompass not only epidemiological factors but also clinical and demographic aspects, shedding light on the intricate interplay between the disease and its multifaceted consequences. Thus, this study adds significant insights to the existing body of knowledge, enhancing our understanding of the complex dynamics between the COVID-19 pandemic and pediatric T1D.

### Study limitations

However, several important limitations require consideration. First, the data collected do not represent the general pediatric population, but rather a subset affected by the disease. Therefore, it is important to acknowledge that the findings may not be generalizable to the entire population. Second, due to the reliance on data from a single center, the study's sample size is relatively small, which may limit its ability to capture the full diversity and variability of the disease across different settings and regions.

## Conclusion

During the COVID-19 pandemic, the proportion of nT1D admissions increased, as well as the severity of DKA-decompensated T1D cases. In addition, the pandemic period brought about notable shifts in the seasonality of pediatric T1D.

## Authors' contribution

MM (Data curation; Formal analysis; Investigation; Writing – original draft)

IBM (Formal analysis; Visualization; Software, Writing – original draft)

AG (Conceptualization; Methodology; Supervision; Writing – review & editing)

## Conflict of interest

None to declare.

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