

BRIEF REPORT

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Risk factors of early death in heat stroke and the challenges of emergency care in Hungary - a case series study

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Abstract

Background and aim The increasing frequency of heat waves is a major challenge for emergency care providers worldwide. The aim of this study was to analyze the clinical features, treatment options, and early outcomes of heat stroke patients presenting to a large academic emergency department (ED) in Hungary and to provide guidance in management for other emergency professionals.

Methods Patients presenting to the ED between June 1, 2024, and July 31, 2024, with a body temperature above 40 °C were analyzed in a retrospective cohort study. Data collection included demographic, clinical and laboratory parameters. Predictors of mortality were analyzed with Mann-Whitney U test.

Results Eight patients were included in our analysis. Three patients died in the ED (37.5%). Patients who died had significantly lower pH (7.07 vs. 7.4, $p=0.036$), higher potassium (7.3 vs. 3.2 mmol/L, $p=0.036$), higher calcium (1.19 vs. 0.97 mmol/L, $p=0.036$), higher lactate (10.9 vs. 3.5 mmol/L, $p=0.036$) and higher PaCO₂ (57.2 vs. 28 mmHg, $p=0.036$) at admission compared to those who did not die.

Conclusions The risk of heat stroke due to climate change-induced heat stress and the consequent thermoregulatory disruption may now be significant in temperate climate zones where it was not previously present. Standardization of differential diagnostic and therapeutic procedures could reduce mortality. pH, potassium, lactate and calcium levels may play an important role in predicting the outcome of heat stroke.

Keywords Emergency care, Heat stroke, Hyperthermia, Climate change, Cooling, Calcium metabolism disorders

Introduction

In recent years, Hungary has seen a noticeable rise in both the frequency and intensity of extreme heat waves, a clear sign of climate change [1]. Data from the temperate zone (45°–49° latitude) show a steady climb in summer temperatures, especially in July and August, where

highs often exceed 35 °C. In 2024, temperatures even topped 40 °C several times (Fig. 1A) [2]. These extreme heat events are more than just weather anomalies; they pose serious health risks, particularly for the elderly and those with chronic conditions [3]. By mid-July, the spike in temperatures coincided with a surge in emergency department visits for heatstroke (Fig. 1B).

Heat stroke is a life-threatening condition. The thermoregulatory system fails and core temperature exceeds 40 °C and if untreated, end-organ failure occurs. Immediate intervention is necessary to prevent irreversible damage [4]. The thermoregulatory disruption is a key

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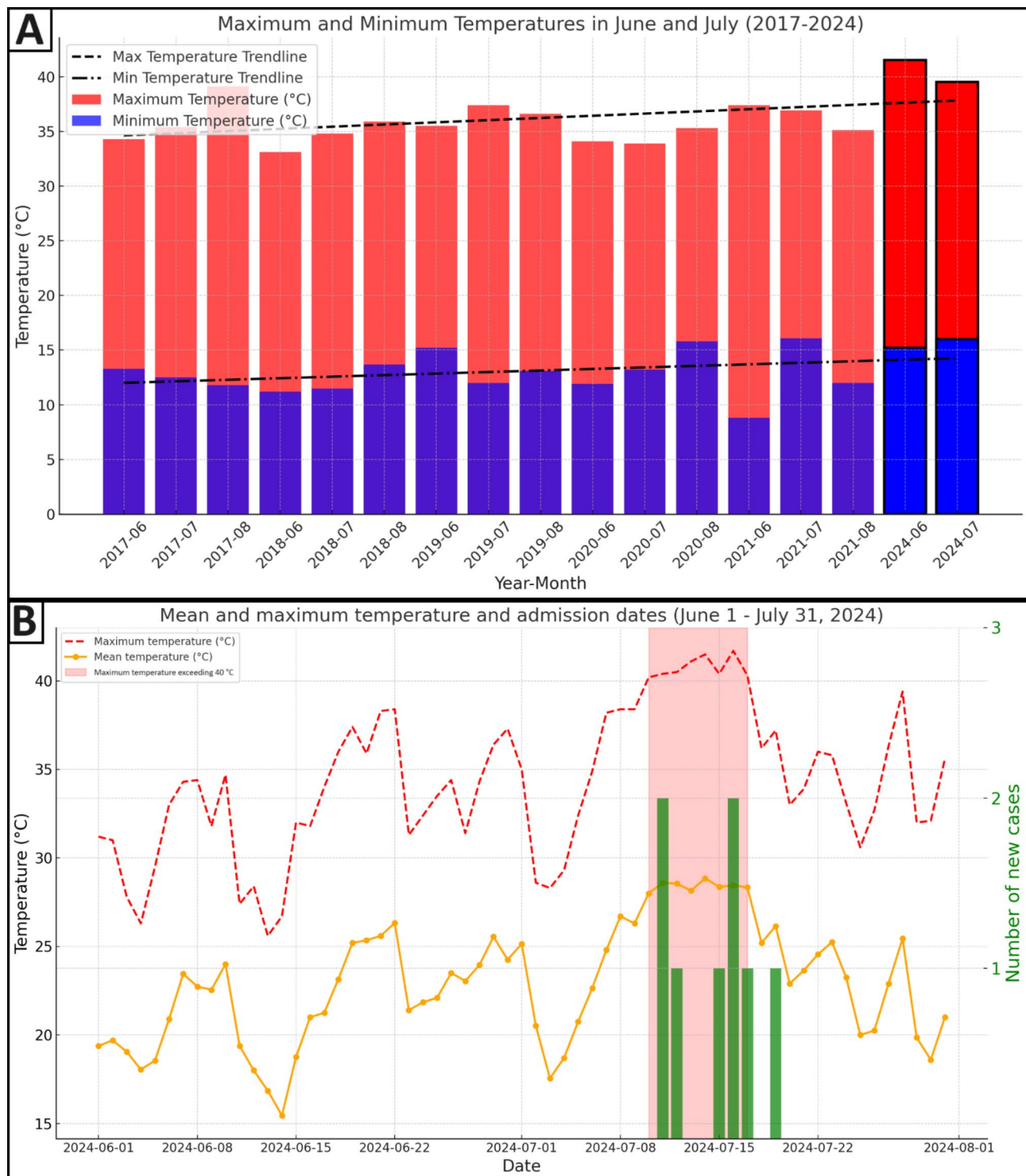


Fig. 1 Temperature extremes and their impact on admissions during the summer months. **A:** Maximum (red) and minimum (blue) temperatures for June and July from 2017 to 2024. **B:** Daily temperatures (maximum in red, mean in orange) with hospital admissions (green bars) from June 1 to July 31, 2024. Data from KSH [1], Időkép [2]

factor in the escalation. This imbalance triggers immune and coagulation disturbances, increased oxidative stress, cell injury, and disruptions in the microbiome, leading to progression [5]. The severity is influenced by individual

biological variability and combination of predisposing factors (Fig. 2) [6]. Classical heat stroke develops more slowly and mainly affects older, immobilized patients, whereas endogenous, exertional heat stroke may develop

Predisposing factors, comorbidities and other agents involved in the development of heat stroke [13]

External causes	Activity & Lifestyle	Comorbidity	Agents
<ul style="list-style-type: none">• High, sustained high ambient temperatures• High relative humidity• Light wind movement• Warm enclosed space• Faulty air conditioning	<ul style="list-style-type: none">• Physical overload• Insulating clothing• Lack of acclimatization• Inadequate fluid intake• Poor adaptive skills, abuse disorder• Fatigue, lack of sleep• Poor physical fitness• Ignoring prevention options	<ul style="list-style-type: none">• Extreme age• Anamnestic hyperthermia• Obesity• Dehydration• Fever of infectious origin• Cardiovascular disease• Autonomic neuropathy, convulsive disorders• Thyreotoxicosis• Skin diseases (e.g. anhidrosis, ectodermal dysplasia)	<ul style="list-style-type: none">• Ethanol and other abusers• Sympathomimetics• Anticholinergic agents• Antihistamines• Serotonergic agents• Drugs with negative inotropic effects (e.g. beta-blockers, calcium channel blockers)• Hallucinogens• Phenothiazines• Salicylates• Lithium• MAO inhibitors

Fig. 2 Predisposing factors, comorbidities and other agents involved in the development of heat stroke [9]

during intense physical activity, especially in younger, active individuals. Heat stroke triggers secondary damage that contributes to patient deterioration and mortality. Common complications include central nervous system damage, cardiovascular, hepatorenal dysfunction and coagulopathies [7]. Hyperthermia-associated multi-organ failure is a significant risk, so rapid and accurate differential diagnosis are keys to effective treatment. Pathologies to be excluded include sepsis, neuroleptic malignant syndrome, serotonin syndrome, and toxic conditions [8].

Aims

The aim of study was to report and analyze the clinical manifestations and treatment options of heat stroke based on the cases of patients treated at the Department of Emergency Medicine, Semmelweis University in the summer of 2024. A further aim was to draw attention to the diagnostic and therapeutic moments of the first 24 h and the importance of standardizing protocols. The analyses aimed to identify differences between survivors and descendants to identify predictors of mortality.

Methods

The cohort search was conducted at the Department of Emergency Medicine, Semmelweis University amongst patients admitted to the ED between 1 June and 31 July 2024. We included patients who presented with a body temperature of at least 40 °C and had an ICD-10 diagnosis code between T67.0 and T67.9, indicating hyperthermia. For the retrospective study, patient data were collected from the Hospital Information System (HIS), eMedSolution version 2024/Q2/3 (20240604085251)

and paper nursing records. Twenty-two patients met the ICD-10 criteria of whom a total of 8 patients had a core body temperature of at least 40 °C at admission. No additional exclusion criteria were applied (Fig. 3). Demographic data, etiology and predisposing factors, clinical characteristics, and results of laboratory and imaging tests were recorded. Patients were followed up until their length of ED stay for survival. We also examined the cooling techniques used and the reduction in body temperature achieved in the first four hours after admission. Data were analyzed using IBM SPSS Statistics version 28.0. Due to the small sample size and non-normal distribution in some variables, as indicated by Shapiro-Wilk tests, non-parametric tests were used for continuous variables and chi-square tests for categorical ones. Histograms and Q-Q plots for key variables are in the supplementary materials (Supplementary Figs. 1–4). Data were categorized into two groups: patients who died during their ED stay (*n*=3) and those who did not die (*n*=5). The significance threshold was set at *p*=0.05. We reported 95% confidence intervals (CI) for each variable and used Cliff’s delta to measure the effect size when comparing medians between groups. Missing data were excluded rather than imputed, paper nursing charts were consulted where possible to address gaps from electronic health records.

Results

Demographic and clinical characteristics

Among the 8 patients included in the study, 3 died (37.5%) and 5 survived during their ED care in the first 24 h of admission. (Table 1). Individual patient

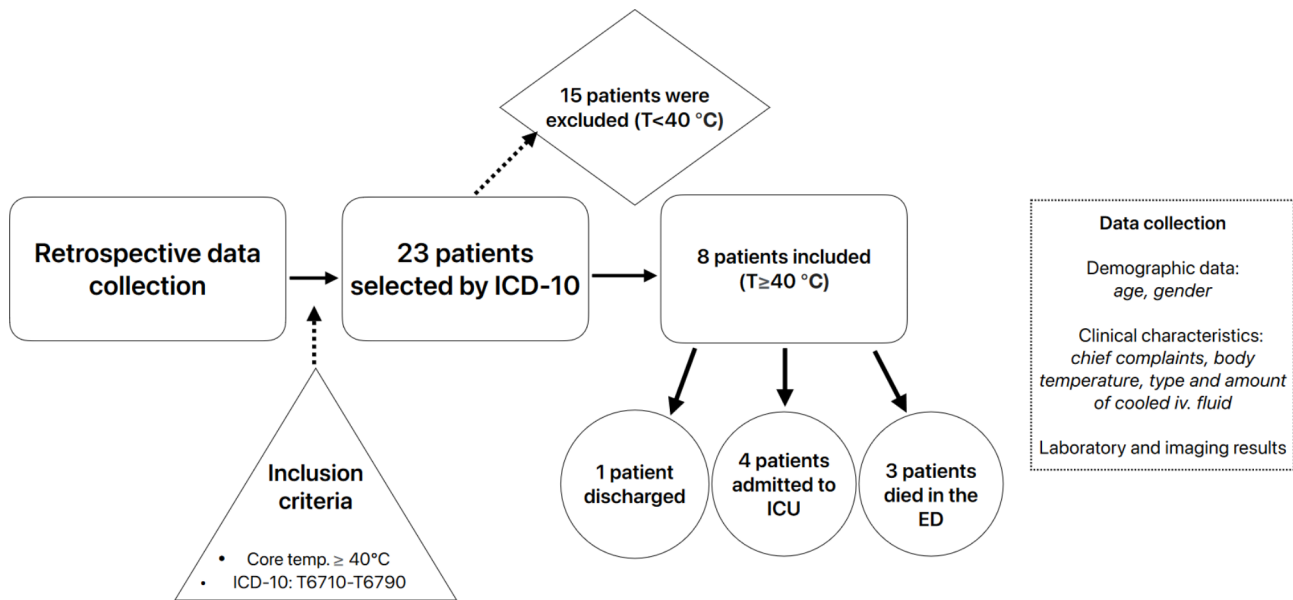


Fig. 3 Patient selection and data collection

Table 1 Comparison of characteristics of patients who died vs. not died in the emergency department

Parameter (median)	Died (n=3)	Not died (n=5)	Reference range	p-value
Age (years)	47 (IQR=17.5)	69 (IQR=10)	-	0.393
Sex	100% male	Females 40%	-	0.673
Core body temperature (°C)	40.6	42.2	35–37.1	0.294
pH	7.067	7.4	7.35–7.45	0.036
PaO ₂	121	200	80–100 mmHg	0.393
PaCO ₂	57.2	28	35–45 mmHg	0.036
HCO ₃	16.4	19	22–26 mmol/L	0.571
Base excess	-13.8	-6	-2–+2 mmol/L	0.071
Na	131	126	135–145 mmol/L	0.571
K	7.3	3.2	3.5–5.1 mmol/L	0.036
Ca	1.19	0.97	1.0–1.2 mmol/L	0.036
Lactate	10.9	3.5	0.5–1.5 mmol/L	0.036

characteristics are presented in Supplementary Table 1. Patients who died were younger, with a median age of 47 years, compared to 69 years for those who survived ($p=0.393$). Patients who died were all males. Several patients developed hyperthermia after prolonged exposure to elevated environmental temperatures during outdoor activities or labor. Others experienced heat stroke in their homes or public spaces. Common metabolic, cardiovascular and psychiatric predisposing comorbidities increased patient's susceptibility to heat stroke.

Laboratory findings

Patients who died had significantly lower pH (7.067 vs. 7.4; $p=0.036$, Cliff's delta=1.0, 95% CI [-0.395, -0.015])

and higher potassium (7.3 vs. 3.2 mmol/L; $p=0.036$, Cliff's delta = -1.0, 95% CI [3.23, 4.77]) and calcium levels (1.19 vs. 0.97 mmol/L; $p=0.036$, Cliff's delta = -1.0, 95% CI [0.06, 0.38]) on initial blood gas analysis. Lactate was elevated in both groups but significantly higher in those who died (10.9 vs. 3.5 mmol/L; $p=0.036$, Cliff's delta = -1.0, 95% CI [4.80, 20.13]). Median base excess was lower in non-survivors (-13.8 vs. -6.0 mmol/L; $p=0.071$, Cliff's delta=0.87, 95% CI [-15.4, -2.0]), though this difference did not reach statistical significance. Creatine kinase, D-dimer, and liver enzymes showed differences but were not statistically significant due to limited data (Supplementary Table 1).

Imaging

Head CT scans were performed on six out of eight patients. One patient who died shortly after admission, exhibited signs of diffuse brain edema and herniation on the CT scan. No abnormalities were detected in the scans of the surviving patients (Fig. 4).

Effectiveness of cooling techniques

All patients in both groups were given cooled intravenous infusions using a balanced crystalloid solution. There was no significant difference in the volume of cooled intravenous infusions between survivors and non-survivors (2000 ml vs. 1500 ml; $p=0.544$, Cliff's delta=1.0 95% CI [189, 3411]). The body cavity lavage procedure was not performed on any of the survivors except one, while it was carried out on all but one of the patients who died (Table 2).



Fig. 4 Representative CT images (axial and coronal slices; ST: 5 mm) demonstrating diffuse cerebral edema; loss of gray-white matter differentiation, compression of internal and external liquor spaces with effacement of basal cisterns

Discussion

Managing severe hyperthermia is especially difficult in regions where heat stroke is uncommon. Quick decisions on cooling and electrolyte correction become crucial as cases rise and treatment options remain limited [10]. The success of cooling methods varied; although physical cooling was applied to all patients, the rate of temperature reduction differed, likely influenced by the severity of heat stroke and how each patient responded. In the most severe cases, invasive body cavity lavage was used, and some evidence suggests it may be more effective than non-invasive techniques when applied quickly and skillfully [11]. Two of the three patients who died arrived in cardiac arrest, while the third was near arrest and died within the first hour. Their critical condition likely contributed to the fatal outcomes. All had severe mixed acidosis, a life-threatening complication that severely disrupts normal body functions [4, 8]. Laboratory results showed differences in pH, PaCO₂, potassium, calcium, and lactate between the two groups. Low pH, insufficient respiratory compensation, lactatemia from poor tissue perfusion and severe hyperkalemia appeared as major predictors of death [8]. Calcium abnormalities may be clinically significant, as dysregulation contributes to complications. Hyperthermia damages muscle cells, causing calcium release and paradoxical hypocalcemia due to binding or precipitation in damaged tissues [12]. This triggers endoplasmic reticulum stress, apoptosis,

worsening neuromuscular function [13]. One patient had abnormally high CK levels, suggesting severe muscle injury and rhabdomyolysis. Muscle breakdown releases myoglobin and CK, which can lead to renal damage by blocking renal tubules. Elevated CK in deceased patients likely reflects severe rhabdomyolysis and multi-organ failure, marking a higher risk of death [14].

Limitations

Key limitation is the small sample size, which makes it harder to draw strong conclusions because random variations could have a greater impact. The short follow-up period limits our ability to see long-term outcomes. Since the data was collected retrospectively, there were challenges with incomplete records and unaccounted factors. Excluding cases with missing data may have affected the results and the different conditions of the patients made it harder to assess cooling techniques. Because the study was done at just one center in Hungary, the findings might not apply generally. A larger, multicenter study with longer follow-up period would help confirm these results.

Conclusions

Heat stroke is more common when the outside temperature exceeds 40 °C. Climate change induced temperature surges are increasing in temperate countries and can lead to increased occurrence of disruptions

Table 2 Basic characteristics and cooling methods and efficiency

#	Age	Sex	Outcome	Admission temp. °C	Etiology	Predisposing factor	Chief complaint	Cooled inf. ml	Inf. °C *	Phys. cooling	Cavital lavage	1st hour	2nd hour	3rd hour	4th hour	Disposition
1	69	M	Not died in the ED	41.8	Found confused at home	Diabetes, hypertension, depression	drowsy, chest pain, shortness of breath	2000	7	+	-	40.9	40.3	39.9	37.9	Discharged
2	76	F	Not died in the ED	42	Collapsed at home, found unconscious	Asthma/COPD, diabetes, pulmonary embolism, thyroid cancer, spine surgery	unconscious, vomiting blood	3000	6	+	-	41.3	40.8	40.4	39.8	Admitted to the ICU
3	35	M	Not died in the ED	42.2	Prolonged outdoor exposure, multiple layers of clothing	Psychiatric history, substance use, homelessness	unconscious, shocked	2500	6	-	-	42.1	41.2	40.6	40.1	Admitted to the ICU
4	76	M	Not died in the ED	42.1	Outdoor activity, collapsed with seizure, hyperthermic state	spine degeneration, recent surgeries	unconscious, grand mal	1500	5	+	+	41.1	40.9	40.4	40.1	Admitted to the ICU
5	66	F	Not died in the ED	40.1	Worked in high heat as a chef	Diabetes mellitus, cirrhosis, insulin use, recent history of high blood sugar	disoriented	2000	5	+	-	39.1	38.3	37.3	37.1	Admitted to the ICU
6	47	M	Died in the ED	41.8	Worked in the sun all day	n/a	circulatory arrest, grand mal,	3000	5	+	+	40.8	39.8	38.5	38.1	Died in the ED
7	70	M	Died in the ED	40.3	Collapsed at train station	n/a	circulatory arrest	1500	6	+	+	39.9	39.2	38.3	38	Died in the ED
8	35	M	Died in the ED	40.6	Collapsed at work after seizure, found without circulation	No known relevant history	disoriented, unconscious	7500	6	+	+	n/a**	-	-	-	Died in the ED

**died in the first hour

in thermoregulation. Emergency professionals need to be prepared to treat patients from the most vulnerable groups, even in countries where this pathology has been less frequent [15]. Prevention, rapid diagnosis and urgent intervention alongside the development of uniform guidelines may be key to reducing mortality. The target of therapeutic cooling is to reach a core temperature below 40 °C in the first hour. Normalization of volume-electrolyte balance is also essential to maintain end-organ function. Further studies are recommended to identify markers of mortality, such as predictive values of calcium abnormalities also focusing on making cooling techniques more efficient and on fine-tuning differential diagnostics.

Abbreviations

BE	base excess
CK	creatinine kinase
ED	Emergency Department
HIS	Hospital Information System
ICU	Intensive Care Unit

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12245-024-00743-v>.

Supplementary Material 1
Supplementary Material 2
Supplementary Material 3
Supplementary Material 4
Supplementary Material 5

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Author contributions

Á.K. designed the study, managed the data collection and wrote the manuscript. T.B. hosted the idea of the study and assisted during the preparation of the manuscript. D.M. assisted in the manuscript's language editing and formatting and gave critical revisions. B.F. assisted in the manuscript's language editing and formatting and gave critical revisions. Sz.G. provided language review and ensured formal requirements. Cs.V. helped with data interpretation and contributed to the critical review of the manuscript. All authors read, reviewed and approved the final manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Ethical approval was provided by the Semmelweis University Regional and Institutional Review Board (SE-RKEB 198/2021).

Consent for publication

Not applicable. This manuscript does not contain any individual person's data in any form (including individual details, images, or videos) that would require consent for publication.

Competing interests

The authors declare no competing interests.

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