Archives of Academic Emergency Medicine. 2025; 13(1): e64

OPEN ACCESS

ORIGINAL RESEARCH

Independent Predictors of Heat-Related Illness-Induced Acute Respiratory Failure: A Multicenter Cross-sectional Study

Wan-Yin Kuo[1-4], Chien-Cheng Huang[1,2,5], Chien-Chin Hsu[2], Hung-Jung Lin[2,6], Shih-Bin Su[3,7], Chung-Feng Liu[8], Mei-I Sung[8], Chi-An Chen[4], How-Ran Guo[4,9]*

1. School of Medicine, College of Medicine, National Sun Yat-sen University, Kaohsiung, Taiwan
2. Department of Emergency Medicine, Chi Mei Medical Center, Tainan, Taiwan
3. Department of Occupational Medicine, Chi Mei Medical Center, Tainan, Taiwan
4. Department of Environmental and Occupational Health, College of Medicine, National Cheng Kung University, Tainan, Taiwan
5. Department of Emergency Medicine, Kaohsiung Medical University, Kaohsiung, Taiwan
6. Department of Emergency Medicine, Taipei Medical University, Taipei, Taiwan
7. Department of Leisure, Recreation and Tourism Management, Southern Taiwan University of Science and Technology, Tainan, Taiwan
8. Department of Medical Research, Chi Mei Medical Center, Tainan, Taiwan
9. Department of Occupational and Environmental Medicine, National Cheng Kung University Hospital, Tainan, Taiwan

Received: June 2025; Accepted: July 2025; Published online: 5 August 2025

Abstract: **Introduction:**Acute respiratory failure (ARF) is a critical complication of heat-related illness (HRI). This study aimed to identify predictors of HRI-induced ARF in patients presenting to the emergency department (ED). **Methods:** Patients aged 20 years and above diagnosed with HRI (ICD-9-CM code 992 or ICD-10 code T67) who visited the EDs of the three hospitals in Tainan, Taiwan between January 2010 and October 2021, were included. Demographic characteristics, comorbidities, and laboratory data were collected. Logistic regression models using the backward elimination method were constructed to identify the independent predictors of HRI-induced ARF. **Results:** 820 patients with the mean age of 50.0 ± 18.4 years were studied (80.0% male). 29 (3.5%) cases experienced ARF. Patients with ARF were less likely to walk on arrival compared to those without it (27.6% vs. 61.8%, $p < 0.001$). Additionally, they had higher prevalence of Glasgow Coma Scale (GCS) scores ≤ 8 ($p = 0.003$), respiratory rate > 20 breaths/min ($p < 0.001$), body temperature ≥ 40°C ($p < 0.001$), hypertension ($p = 0.001$), cerebrovascular disease ($p = 0.001$), and chronic obstructive pulmonary disease ($p = 0.042$). The multivariable logistic regression revealed that body temperature ≥ 40 °C on arrival (odds ratio (OR): 7.76; 95% confidence interval (CI): 3.14–19.15), an initial respiratory rate > 20 breaths/min (OR: 8.19; 95% CI: 3.48–19.24), and history of hypertension (OR: 3.38; 95% CI: 1.52–7.52) were predictors of HRI-induced ARF. **Conclusion:** Elevated body temperature, respiratory rate, and a history of hypertension were key predictors of ARF in HRI patients, aiding in patient stratification for emergency care.

Keywords: Acute respiratory failure; Body temperature; Heat related illness; Hypertension; Respiratory rate

Cite this article as: Kuo W, Huang C, Hsu C, et al. Independent Predictors of Heat-Related Illness-Induced Acute Respiratory Failure: A Multicenter Cross-sectional Study. Arch Acad Emerg Med. 2025; 13(1): e64. https://doi.org/10.22037/aaemj.v13i1.2710.

1. Introduction

Heat-related illness (HRI) is an emergency condition that poses a major public health concern, particularly in the context of global warming (1). HRI occurs due to impaired thermoregulation resulting from the body's inability to dissipate heat effectively (2). The severity of HRI ranges from mild conditions such as heat rash, cramps, and fatigue, to moderate conditions such as heat exhaustion, and to severe conditions such as heat stroke (3). Treatment of HRI typically involves supportive care, rapid cooling interventions, and rehydration via oral or intravenous fluids (2, 4). While most patients presenting with mild to moderate HRI can be treated and discharged from the emergency department (ED), patients with severe HRI, the elderly, or those with comorbidities may require additional intervention or admission (4).

Heat stroke, the most severe condition of HRI, manifests with a core body temperature ≥ 40°C, is often accompanied by central nervous system (CNS) dysfunction, presenting as delirium, convulsions, or coma (4, 5). In addition to elevated core body temperature, the patients typically exhibit other vital sign abnormalities including sinus tachy-

*Corresponding Author: How-Ran Guo; Department of Environmental and Occupational Health, College of Medicine, National Cheng Kung University, 138 Sheng-Li Road, Tainan, 70428, Taiwan. Phone: +886-6-235-3535 ext. 5802, Fax: +886-6-275-2484, Email: hrguo@mail.ncku.edu.tw, ORCID: https://orcid.org/0000-0003-3343-3446.

cardia, tachypnea, and hypotension (6). Complications of heat stroke may include acute respiratory distress syndrome (ARDS), disseminated intravascular coagulation (DIC), acute kidney injury, hepatic injury, rhabdomyolysis, and seizures (6). Acute respiratory failure (ARF) stands out as a critical and life-threatening complication among these conditions, which calls for the need of patient stratification (7).

Despite the pressing need for mitigating ARF in HRI patients, research specifically targeting its predictors remains limited. Previous studies have predominantly concentrated on predicting outcomes such as mortality (8-10), admission rates (11, 12), or neurological complications (13). A few studies have reported that lower Glasgow Coma Scale (GCS) scores and decreased standard base excess levels upon admission were associated with a higher risk of ARF among patients with heat stroke. However, these studies evaluated ARF as a secondary outcome among multiple complications and did not focus on early clinical predictors in an HRI population in general (13, 14). Therefore, this study aimed to fill the research gap by comprehensively identifying and analyzing the predictors associated with ARF in patients with HRI presenting to the ED.

2. Methods

2.1. Study design and setting

We conducted a multicenter cross-sectional study involving patients who presented to the EDs between January 2010 and October 2021. Data were retrieved from electronic medical records of three hospitals: Chi Mei Medical Center, Chi Mei Liouying Hospital, and Chi Mei Chiali Hospital, which comprised the largest healthcare system in Tainan, Taiwan. The primary outcome was HRI-induced ARF, and was defined by either: (1) ICD-9-CM codes 518.81, 518.84, or ICD-10 code J96 in the diagnoses, or (2) the use of a non-rebreathing mask, bi-level positive airway pressure, or endotracheal intubation. Accordingly, patients were classified into two groups, namely with and without ARF, and independent predictors of HRI-induced ARF were evaluated using multivariate regression analysis.

This study was carried out according to the ethical principles of the Declaration of Helsinki and approved by the Institutional Review Board of Chi Mei Medical Center (approval code: 11210-007). The Board waived the need for informed consent from participants because the dataset consists of de-identified data. This waiver does not affect the rights and welfare of the participants.

2.2. Participants

All patients aged 20 years and above, who received a final diagnosis of HRI, identified by the ICD-9-CM code 992 or ICD-10 code T67 were included. Patients who were transferred to other hospitals were excluded.

2.3. Data gathering

We collected demographic characteristics, comorbidities, vital signs on arrival, and laboratory data from electronic medical records to identify potential predictors. Demographic characteristics included age and sex. We stratified age into two groups: < 65 years and ≥ 65 years by the definition of the elderly. Comorbidities included hypertension (ICD-9-CM: 401–405 or ICD-10: I10–I16), diabetes (ICD-9-CM: 250 or ICD-10: E08–E13), coronary artery disease (CAD, ICD-9-CM: 410–414 or ICD-10: I20–I25), congestive heart failure (CHF, ICD-9-CM: 428.0 or ICD-10: I50), cerebrovascular disease (ICD-9-CM: 430–438 or ICD-10: I60–I69), chronic obstructive pulmonary disease (COPD, ICD-9-CM: 490–496 or ICD-10: J44), liver disease (ICD-9-CM: 570–576 or ICD-10: K70–K77, K80–K83), chronic kidney disease (CKD, ICD-9-CM: 585 or ICD-10: N18), malignancy (ICD-9-CM: 140-208 or ICD-10: C00-C69), and mental disorder (ICD-9-CM: 290–319 or ICD-10: F01–F99). We also collected the patient's body mass index (BMI) and stratified it into two groups: <24 and ≥24 Kg/m2 according to the definition of overweight set by the Taiwanese government. As for the condition on arrival, studied variables included walking on arrival (yes/no), GCS score, blood pressure (including systolic blood pressure [SBP] and diastolic blood pressure [DBP]), heart rate (HR), respiratory rate (RR), body temperature (BT), and pulse oximetry (SpO2). The values of vital signs were further stratified into two groups based on previous studies or definitions of clinical abnormalities such as hypotension, tachycardia, and tachypnea (15, 16). Laboratory data included all blood chemistry, except for variables with more than 70% missing data, such as base excess level. For included variables, we imputed missing data with the mean of each variable based on the outcome. For instance, if a patient experienced ARF and had missing SBP at the initial assessment, the mean for the group with ARF was assigned to this patient. An exception was made for BMI, which was imputed based not only on the outcome but also on the patient's sex.

Continuous variables were transformed into categorical variables, including white blood cell (WBC) count, hemoglobin, platelet count, sodium (Na), potassium (K), blood urea nitrogen (BUN), random glucose, alanine aminotransferase (ALT), creatine kinase total (CK-Total), and myoglobin. The cut-off points for each variable were determined based on previous studies (17, 18) or the normal range defined by the laboratory.

2.4. Statistical analysis

Demographic and clinical characteristics were analyzed as frequencies and percentages for categorical variables. We evaluated differences between the two groups (with and without ARF) using either the Pearson chi-square test or Fisher's exact test. Logistic regression analyses were performed to investigate the independent factors. Initially, univariate analysis was performed. Subsequently, multivariable analysis was carried out, which included variables with a p-value < 0.15 in the univariate analysis. Then, a model was

constructed using the backward elimination method (with a p-value set at 0.15) to identify independent factors and evaluate their effects. This threshold was commonly used to avoid prematurely excluding variables that may become significant when included in a multivariable model. Our approach was consistent with established practices in logistic regression modeling in the literature (19, 20). The odds ratio (OR) for each variable and its corresponding 95% confidence interval (CI) were calculated. Correlation coefficients between variables included in the multivariable analysis were also examined. Model performance was evaluated using the Hosmer–Lemeshow goodness-of-fit test, with a p-value > 0.05 indicating acceptable fit. For sensitivity analysis, we applied multiple imputations to handle missing data and compared the results to those from the primary analysis using mean imputation. We carried out all analyses using the Statistical Analysis Software Version 9.3 (SAS Inc., Raleigh, NC, USA) and Statistical Product and Service Solutions Version 17.0 (SPSS Inc., Chicago, IL, USA) at the significance level of 0.05 (two-tailed).

3. Results

3.1. Baseline characteristics of the study population

820 patients with the mean age of 50.0 ± 18.4 (range: 20.3–96.3) years were included (80.0% male). During the ED visit, 29 (3.5%) patients experienced ARF. Table 1 compares the baseline characteristics between cases with and without HRI-induced ARF. Patients with ARF were less likely to walk on arrival compared to those without it (27.6% vs. 61.8%, $p < 0.001$). Additionally, they had higher prevalence of GCS scores ≤ 8 ($p = 0.003$), RR > 20 breaths/min ($p < 0.001$), BT ≥ 40°C ($p < 0.001$), hypertension ($p = 0.001$), cerebrovascular disease ($p = 0.001$), and COPD ($p = 0.042$). No significant differences were observed in laboratory data between patients with and without ARF.

3.2. Independent predictors of HRI-induced ARF

Table 2 shows the results of multivariable logistic regression analysis. Based on this analysis, two independent predictors were identified: RR > 20 breaths/min (OR = 8.19; 95% CI: 3.48–19.24) and hypertension (OR = 3.38; 95% CI: 1.52–7.52) (Model 1). When only these two independent variables were included (Model 2), RR > 20 breaths/min was associated with an OR of 10.96 (95% CI: 4.99–24.06) and hypertension was associated with an OR of 3.67 (95% CI: 1.66–8.14). The examination of correlation coefficient revealed a good correlation between RR > 20 breaths/min and BT ≥ 40°C (correlation coefficient = 0.734). Accordingly, we constructed another model in which RR > 20 breaths/min was replaced by BT ≥ 40°C (Model 3) and found that BT ≥ 40°C was also an independent predictor of ARF in HRI patients. All three logistic regression models demonstrated good calibration according to the Hosmer–Lemeshow goodness-of-fit test. The p-values were 0.847 for Model 1, 0.515 for Model 2, and 0.449 for Model 3, indicating acceptable fit of all the models. To better illustrate the model development process and the identification of key predictors, a summary flowchart was provided (Figure 1).

When employing multiple imputations instead of single imputation for missing data, the results of multivariable analysis and subsequent model refinement remained consistent.

4. Discussion

This multicenter study encompassed patients with all types of HRI in the EDs and examined various variables, including baseline characteristics, vital signs, comorbidities, and laboratory data, to identify potential predictors of ARF. As a result, BT ≥ 40°C and RR > 20 breaths/min on arrival, as well as a history of hypertension were identified as independent predictive factors for ARF.

Alveolar ventilation, a product of RR and tidal volume, is typically meticulously regulated by central and peripheral chemoreceptors along with lung receptors. It is influenced by both the arterial partial pressure of oxygen (PaO2) and the arterial partial pressure of carbon dioxide (PaCO2), with PaCO2 being the primary driver. The body responds to hypoxemia and hypercapnia by increasing both tidal volume and respiratory rate, making RR a useful metric for detecting these conditions (21). Exposure to heat stress prompts an elevation in body core temperature, leading to increased ventilation regardless of metabolic factors. This increased ventilation results in a decrease in arterial CO2 pressure (hypocapnia) (22). If unchecked, this condition may progress to ARDS due to hyperventilation and pulmonary vasodilation and hyperpermeability through hyperpnea and the effects of cytokines (23). Previous studies have shown that an abnormal RR was a predictor of potentially serious clinical events, such as cardiac arrest, admission to an intensive care unit (ICU), and respiratory failure (21, 24). Nevertheless, RR has not been identified as an independent predictor of ARF in HRI patients in the EDs in previous studies.

In this study, a BT of 40°C or higher upon arrival was not only indicative of ARF in HRI patients but also strongly associated with tachypnea. Enzyme denaturation initiates once the BT reaches 40°C at the cellular level; at 41°C, decreases in mitochondrial function disrupt oxidative phosphorylation (the process of intracellular energy production), leading to organ dysfunction (23). Clinically, heat stroke typically manifests with a core temperature elevated to at least 40°C and the presence of CNS dysfunction (5). Heat stroke can potentially culminate in multi-organ failure, including respiratory failure. Both experimental and clinical evidence elucidated BT≥40°C as a crucial factor in predicting ARF in HRI patients. Previous studies have indicated that some underlying illnesses contribute to the risk of HRI. While many comorbidities were evaluated in this study, only a history of hypertension was identified as an independent predictor for ARF in HRI patients. Studies have also shown that individuals

with hypertension faced greater challenges in adapting to extreme environmental conditions and were at an elevated risk of heat-related complications compared to those with normal blood pressure. This susceptibility may be attributed to increased peripheral vascular resistance and associated circulatory changes, including vascular smooth muscle hypertrophy, reduced blood vessel density, and decreased baroreflex sensitivity in hypertensive individuals. These alterations can disrupt blood flow regulation to the skin, consequently impacting core temperature control. During exercise in hot conditions, hypertensive individuals often exhibit heightened blood pressure responses and may experience greater thermal strain than those with normal blood pressure (25). However, the clinical implications of pulmonary responses during heat exposure in people with hypertension remain unclear. Further studies are warranted to elucidate this matter.

Some predictors have been identified, and various assessment tools have been developed, in previous studies to predict clinical outcomes in patients with HRI, primarily focusing on mortality, hospitalization, or neurological sequelae (8-12). However, research specifically targeting predictors of ARF remains limited. One study assessing the association between standard base excess levels upon admission and outcomes in heat stroke patients found that, along with in-hospital mortality, standard base excess level was significantly linked to ARF, as well as admission to the ICU, acute kidney failure, sepsis, and coagulation impairment (14). Although base excess level was initially included in this study, it was later excluded due to more than 70% of missing data, indicating that it is not routinely checked in HRI patients in the ED. However, metabolic acidosis in heat stroke with low standard base excess levels may prompt an increase in RR, which was identified as an independent predictor for ARF in this study. Another study on heat stroke patients demonstrated that the initial GCS score was an independent prognostic factor for ARF, as well as neurological sequelae at discharge and ICU admission (13). Despite the association between lung injury and brain damage in heat stroke, the GCS score on arrival was not an independent predictor of ARF in HRI patients after adjustment for other factors in this study, although it appeared to be associated with ARF in the univariate analysis. Several reasons might account for this disparity. Firstly, patient population in this study differed from that of the previous study, as we included all types of HRI rather than solely focusing on heat stroke, in which CNS dysfunction is typically evident. Secondly, the low prevalence (3.7%) of low GCS scores (GCS ≤ 8) in our study population may decrease the statistical power. Nonetheless, the low prevalence also indicates that it cannot be applied to most cases in clinical practice. Furthermore, the multifactorial nature of HRI suggests that while neurological impairment assessed by the GCS score is essential, other factors such as underlying illnesses or altered vital signs may contribute more to respiratory failure. Notably, our multivariable analysis included RR and hypertension, which differed from the variables used in the previous study.

The strength of this study lied in its specific focus on ARF in HRI, different from most studies, in which it was considered a secondary outcome. By encompassing all types of HRI, rather than focusing on specific types, this study offered clinicians valuable insights, considering HRI as a continuum of conditions when differentiation can be challenging initially. Moreover, this study comprehensively examined various aspects of HRI, including demographic characteristics, comorbidities, vital signs, and laboratory data, and thus provides a holistic understanding of predictors for ARF.

5. Limitations

Several limitations warranted consideration in this study. First, the retrospective design limited the variables available for analysis and might introduce information bias. Nevertheless, the key predictors identified—RR > 20 breaths/min, BT ≥ 40°C, and a history of hypertension—were routinely recorded and objectively measured, which reduced the likelihood of systematic misclassification. Additionally, the consistency of these predictors across three logistic regression models and in sensitivity analyses using multiple imputations supported the robustness of the associations. Any misclassification, particularly of the outcome variable, was likely to be non-differential, which would bias results toward the null rather than generate spurious associations. Second, the primary analysis employed mean imputation for missing data, which might underestimate the variance and could introduce bias if the assumption of data being missing completely at random was violated. To mitigate this limitation, we conducted a sensitivity analysis using multiple imputations, a method that preserved data variability, reduced bias, and yielded more valid statistical inferences by generating multiple plausible values for missing data based on observed patterns. The consistent identification of key predictors across both imputation methods supported the reliability of our conclusions. Third, some variables were excluded due to a large proportion of missing data, such as DIC profiles and base excess levels, limiting our ability to evaluate their effects in this study. Fourth, despite adjusting for various demographic and clinical factors, residual confounding from unmeasured variables or unexplored interactions may persist. Finally, while utilizing multicenter data has enhanced the generalizability of study results, this study was conducted within a specific healthcare system in Taiwan, potentially restricting the applicability of findings to other populations or healthcare settings.

6. Conclusions

Based on the findings, RR > 20 breaths/min, BT ≥ 40°C, and a history of hypertension were among the independent predictors of HRI-induced ARF. These findings provided a practical reference for emergency physicians to identify high-risk

patients early. Incorporating these readily available predictors into ED triage protocols may enhance early recognition and prompt initiation of critical care, potentially improving patient outcomes.

7. Declarations

7.1. Acknowledgments

None.

7.2. Funding

This study was supported by grant CMFHR11096 from Chi Mei Medical Center. The funder had no role in the study design, data collection, analysis, publishing decision, or manuscript preparation.

7.3. Authors' contributions

Wan-Yin Kuo: conceptualization, methodology, software, formal analysis, writing—original draft. Chien-Cheng Huang: conceptualization, methodology, supervision, writing—reviewing & editing. Chien-Chin Hsu: data curation, supervision, writing—reviewing & editing. Hung-Jung Lin: data curation, supervision, writing—reviewing & editing. Shih-Bin Su: conceptualization, supervision, writing—reviewing & editing. Chung-Feng Liu: data curation, writing—reviewing & editing. Mei-I Sung: data curation, writing—reviewing & editing. Chi-An Chen: software, formal analysis, writing—reviewing & editing. How-Ran Guo: conceptualization, methodology, writing—reviewing & editing, supervision. All authors read and approved the final version of manuscript.

7.4. Ethical approval

This study was carried out according to the ethical principles of the Declaration of Helsinki and approved by the Institutional Review Board of Chi Mei Medical Center (approval code: 11210-007). The Board waived the need for informed consent from participants because the dataset consists of de-identified data. This waiver does not affect the rights and welfare of the participants.

7.5. Consent to participate

Informed consent was waived due to the retrospective nature of the study.

7.6. Conflict of Interest

The authors have no conflict of interests related to this paper.

7.7. Data availability

The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request.

References

1. Sankoff J. Heat illnesses: a hot topic in the setting of global climate change. Aust Fam Physician. 2015;44(1-2):22-6.
2. Gauer R, Meyers BK. Heat-Related Illnesses. Am Fam Physician. 2019;99(8):482-9.
3. Sorensen C, Hess J. Treatment and Prevention of Heat-Related Illness. N Engl J Med. 2022;387(15):1404-13. https://doi.org/10.1056/NEJMcp2210623 .
4. Santelli J, Sullivan JM, Czarnik A, Bedolla J. Heat illness in the emergency department: keeping your cool. Emerg Med Pract. 2014;16(8):1-21; quiz -2.
5. Atha WF. Heat-related illness. Emerg Med Clin North Am. 2013;31(4):1097-108. https://doi.org/10.1016/j.emc.2013.07.012.
6. Morris A, Patel G. Heat Stroke. 2023 Feb 13. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan–. PMID: 30725820.
7. Bouchama A, Abuyassin B, Lehe C, Laitano O, Jay O, O'Connor FG, et al. Classic and exertional heatstroke. Nat Rev Dis Primers. 2022;8(1):8. https://doi.org/10.1038/s41572-021-00334-6
8. Shimazaki J, Hifumi T, Shimizu K, Oda Y, Kanda J, Kondo Y, et al. Clinical characteristics, prognostic factors, and outcomes of heat-related illness (Heatstroke Study 2017-2018). Acute Med Surg. 2020;7(1):e516. https://doi.org/10.1002/ams2.516
9. Argaud L, Ferry T, Le QH, Marfisi A, Ciorba D, Achache P, et al. Short- and long-term outcomes of heatstroke following the 2003 heat wave in Lyon, France. Arch Intern Med. 2007;167(20):2177-83. https://doi.org/10.1001/archinte.167.20.ioi70147.
10. Hifumi T, Kondo Y, Shimazaki J, Oda Y, Shiraishi S, Wakasugi M, et al. Prognostic significance of disseminated intravascular coagulation in patients with heat stroke in a nationwide registry. J Crit Care. 2018;44:306-11. https://doi.org/10.1016/j.jcrc.2017.12.003 .
11. Hess JJ, Saha S, Luber G. Summertime acute heat illness in U.S. emergency departments from 2006 through 2010: analysis of a nationally representative sample. Environ Health Perspect. 2014;122(11):1209-15. https://doi.org/10.1289/ehp.1306796 .
12. Pillai SK, Noe RS, Murphy MW, Vaidyanathan A, Young R, Kieszak S, et al. Heat illness: predictors of hospital admissions among emergency department visits-Georgia, 2002-2008. J Community Health. 2014;39(1):90-8. https://doi.org/10.1007/s10900-013-9743-4 .
13. Chen L, Lu L, Fang Y, Ren J, Yang X, Gong Z, et al. Glasgow Coma Scale on admission as predictor of neurological sequelae at discharge and acute respiratory failure in patients with heatstroke. Postgrad Med J. 2023;99(1178):1237-45. https://doi.org/10.1093/postmj/qgad071.
14. Chen L, Yang X, Zhao J, Xu S, Zhang Y, Feng X. The association of standard base excess upon

emergency admission with outcomes in patients with heat stroke. Am J Emerg Med. 2023;72:7-15. https://doi.org/10.1016/j.ajem.2023.06.048

15. Huang CC, Hsu CC, Guo HR, Su SB, Lin HJ. Dengue fever mortality score: A novel decision rule to predict death from dengue fever. J Infect. 2017;75(6):532-40. https://doi.org/10.1016/j.jinf.2017.09.014.
16. Park SB, Khattar D. Tachypnea. 2023 Feb 13. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan–. PMID: 31082106.
17. Varghese GM, John G, Thomas K, Abraham OC, Mathai D. Predictors of multi-organ dysfunction in heatstroke. Emerg Med J. 2005;22(3):185-7. https://doi.org/10.1136/emj.2003.009365.
18. Wu M, Wang C, Zhong L, Liu Z. Serum myoglobin as predictor of acute kidney injury and 90-day mortality in patients with rhabdomyolysis after exertional heatstroke: an over 10-year intensive care survey. Int J Hyperthermia. 2022;39(1):446-54. https://doi.org/10.1080/02656736.2022.2046183.
19. Bursac Z, Gauss CH, Williams DK, Hosmer DW. Purposeful selection of variables in logistic regression. Source Code Biol Med. 2008;3:17. https://doi.org/10.1186/1751-0473-3-17.
20. Cheng YY, Huang NC, Chang YT, Sung JM, Shen KH, Tsai CC, et al. Associations between arsenic in drinking water and the progression of chronic kidney disease: A nationwide study in Taiwan. J Hazard Mater. 2017;321:432-9. https://doi.org/10.1016/j.jhazmat.2016.09.032.
21. Cretikos MA, Bellomo R, Hillman K, Chen J, Finfer S, Flabouris A. Respiratory rate: the neglected vital sign. Med J Aust. 2008;188(11):657-9. https://doi.org/10.5694/j.1326-5377.2008.tb01825.x.
22. Tsuji B, Hayashi K, Kondo N, Nishiyasu T. Characteristics of hyperthermia-induced hyperventilation in humans. Temperature (Austin). 2016;3(1):146-60. https://doi.org/10.1080/23328940.2016.1143760.
23. Miyake Y. Pathophysiology of heat illness: Thermoregulation, risk factors, and indicators of aggravation. Japan Medical Association Journal. 2013;56:167-73.
24. Chen L, Chen L, Zheng H, Wu S, Wang S. Emergency admission parameters for predicting in-hospital mortality in patients with acute exacerbations of chronic obstructive pulmonary disease with hypercapnic respiratory failure. BMC Pulm Med. 2021;21(1):258. https://doi.org/10.1186/s12890-021-01624-1.
25. Kenny GP, Yardley J, Brown C, Sigal RJ, Jay O. Heat stress in older individuals and patients with common chronic diseases. Cmaj. 2010;182(10):1053-60. https://doi.org/10.1503/cmaj.081050.

Table 1: Comparing the baseline characteristics of the study population between cases with and without heat-related illness-induced acute respiratory failure

Variable	Overall (n=820)	Acute respiratory failure		P value
		No (n=791)	Yes (n=29)	
Age (years)				
≥ 65	190 (23.2)	181 (22.9)	9 (31.0)	0.307
Sex				
Male	656 (80.0)	633 (80.0)	23 (79.3)	0.925
BMI (Kg/m2)				
≥ 24	633 (77.2)	613(77.5)	20 (69.0)	0.282
Triage vital signs				
Walking on arrival	497 (60.6)	489 (61.8)	8 (27.6)	<0.001
GCS ≤ 8	30 (3.7)	25 (3.2)	5 (17.2)	0.003
SBP < 90 (mmHg)	57 (7.0)	53 (6.7)	4 (13.8)	0.136
DBP < 60 (mmHg)	80 (9.8)	76 (9.6)	4 (13.8)	0.517
HR > 100 (/min)	340(41.5)	326(41.2)	14 (48.3)	0.448
RR > 20 (/min)	114 (13.9)	97 (12.3)	17 (58.6)	<0.001
BT ≥ 40 (°C)	41 (5.0)	33 (4.2)	8 (27.6)	<0.001
SpO2 < 95 (%)	83 (10.1)	78 (9.9)	5 (17.2)	0.204
Comorbidities				
Hypertension	173 (21.1)	160 (20.2)	13 (44.8)	0.001
Diabetes mellitus	114 (13.9)	111 (14.0)	3 (10.3)	0.786
Coronary artery disease	56 (6.8)	52 (6.6)	4 (13.8)	0.129
Congestive heart failure	16 (2.0)	16 (2.0)	0 (0.0)	> 0.95
Cerebrovascular disease	64 (7.8)	57 (7.2)	7 (24.1)	0.001
COPD	56 (6.8)	51 (6.4)	5 (17.2)	0.042
Liver disease	85 (10.4)	83 (10.5)	2 (6.9)	0.759
Chronic kidney disease	31 (3.8)	30 (3.8)	1 (3.4)	> 0.95
Malignancy	27 (3.3)	25 (3.2)	2 (6.9)	0.247
Mental disorder	127 (15.5)	119 (15.0)	8 (27.6)	0.067
Laboratory data				
WBC ≥ 12 (103/uL)	248 (30.2)	239 (30.2)	9 (31.0)	0.925
Hemoglobin < 12 (g/L)	66 (8.0)	63 (8.0)	3 (10.3)	0.502
Platelet < 150 (103/uL)	61 (7.4)	58 (7.3)	3 (10.3)	0.470
Na < 135 (mEq/L)	180 (22.0)	170 (21.5)	10 (34.5)	0.097
K < 3.5 (mEq/L)	203 (24.8)	195 (24.7)	8 (27.6)	0.719
Glucose (random) > 200 (mg/dL)	94 (11.5)	90 (11.4)	4 (13.8)	0.565
BUN > 20 (mg/dL)	214 (26.1)	203 (25.7)	11 (37.9)	0.140
Creatinine > 1.3 (mg/dL)	507 (61.8)	488 (61.7)	19 (65.5)	0.677
ALT > 40 (U/L)	223(27.2)	216 (27.3)	7 (24.1)	0.706
CK-Total > 1000 (U/L)	108 (13.2)	104 (13.1)	4 (13.8)	0.785
Myoglobin > 1000 (ng/mL)	501 (61.1)	482 (60.9)	19 (65.5)	0.619

Data are presented as number (%). BMI: body mass index; GCS: Glasgow Coma Scale; SBP: systolic blood pressure; DBP: diastolic blood pressure; HR: Heart rate; RR: Respiratory rate, BT: body temperature; SpO2: pulse oximetry; COPD: chronic obstructive pulmonary disease; WBC: white blood cell; Na: sodium; K: potassium; BUN: blood urine nitrogen; ALT: alanine aminotransferase; CK-Total: creatine kinase total.

Table 2: Independent predictors of acute respiratory failure in patients with heat-related illness based on logistic regression analysis

Variables	95% confidence interval	P value
Crude model (selecting variables with p<0.15)		
Waking on arrival	0.24 (0.10–0.54)	0.001
Glasgow Coma Scale ≤ 8	6.38 (2.25–18.11)	<0.001
Respiratory rate > 20 (/minute)	10.14 (4.70–21.87)	<0.001
Body temperature ≥ 40 (°C)	8.75 (3.61–21.22)	<0.001
Hypertension	3.20 (1.51–6.80)	0.002
Cerebrovascular disease	4.10 (1.68–10.00)	0.002
Coronary artery disease	2.27 (0.76–6.78)	0.140
Chronic obstructive pulmonary disease	3.02 (1.11–8.25)	0.031
Mental disorder	2.15 (0.93–4.97)	0.073
Na < 135 (mEq/L)	1.92 (0.88–4.21)	0.102
Blood urine nitrogen > 20 (mg/dL)	1.77 (0.82–3.81)	0.144
Model 1 (backward elimination method)		
Waking on arrival	0.49 (0.20–1.24)	0.132
Respiratory rate > 20 (/minute)	8.19 (3.48–19.24)	<0.001
Hypertension	3.38 (1.52–7.52)	0.003
Model 2 (adjusted for respiratory rate and hypertension)		
Respiratory rate > 20 (/minute)	10.96 (4.99–24.06)	<0.001
Hypertension	3.67 (1.66–8.14)	0.001
Model 3 (adjusted for body temperature and hypertension)		
Body temperature ≥ 40 (°C)	7.76 (3.14–19.15)	<0.001
Hypertension	2.84 (1.31–6.16)	0.008

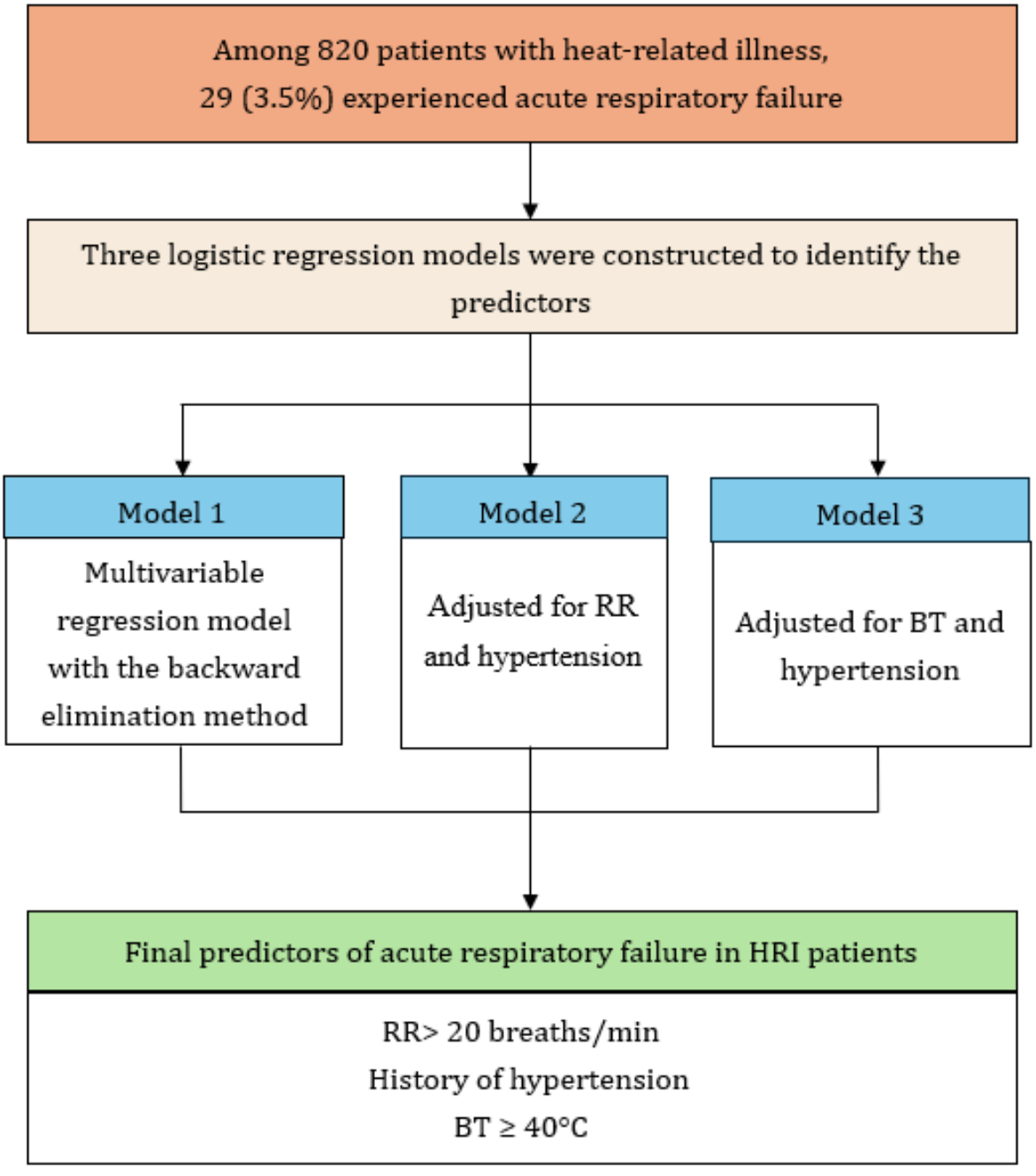

Figure 1: Flowchart summarizing the modeling strategy and key findings. Three logistic regression models were constructed to evaluate predictors of acute respiratory failure among patients with heat-related illness (HRI). Final predictors identified across the models included RR > 20 breaths/min, history of hypertension, and BT ≥ 40°C. RR: respiratory rate; BT: body temperature.