Int J Burn Trauma 2026;16(4):153-160
www.IJBT.org /ISSN:2160-2026/IJBT0170144

Review Article

Cardiac biomarkers in acute burn injury: a systematic review of diagnostic and prognostic utility

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Received November 20, 2025; Accepted August 19, 2026; Epub August 25, 2026; Published August 30, 2026

Abstract: Objective: Cardiac biomarkers offer potential for early diagnosis and prognosis; however, their utility in burn care is not well established. This systematic review aimed to evaluate the current evidence regarding the diagnostic and prognostic values of cardiac biomarkers in patients with acute burn injuries. Methods: A systematic search of PubMed and Google Scholar (July 2015-September 2025) was conducted according to the PRISMA guidelines. We included prospective and retrospective observational studies that evaluated troponin (I or T), B-type natriuretic peptide (BNP), or N-terminal pro-BNP (NT-proBNP) in patients with acute burns and reported clinical outcomes. Case reports, reviews, and studies of patients with pre-existing cardiovascular diseases were excluded. Results: Seven original studies were included in this systematic review. The findings consistently demonstrated that elevated troponin levels were associated with larger burn sizes, sepsis, and increased mortality, reflecting systemic stress rather than primary ischemic events. BNP and NT-proBNP levels are strong independent predictors of mortality, sepsis, and toxic myocarditis. Notably, BNP demonstrated superior sensitivity to troponin for the early detection of myocarditis in patients with moderate-to-severe burns. Conclusions: Cardiac biomarkers are valuable prognostic indicators in acute burn injuries, strongly associated with mortality and major complications. The integration of these factors into clinical practice could significantly enhance early risk stratification.

Keywords: Burns, brain natriuretic peptide, troponin

Introduction

Burn injuries are a major global health burden, affecting an estimated 9 million people annually and resulting in approximately 115,000 deaths annually [1]. This indicates that a burn injury occurs every three seconds, while a fatality occurs every four minutes. Despite the rapid development of burn management, nutritional support, fluid resuscitation, and infection control over recent decades, the morbidity and mortality rates among patients with burns remain high [2]. Severe burns induce serious hemodynamic and cardiodynamic changes, which may be followed by sepsis, multiple organ failure, and death. Cardiac stress is considered a part of the acute-phase response, whereas the development of severe cardiac dysfunction carries a poor prognosis. Heart dysfunction can lead to organ hypoperfusion, deterioration of peripheral microcirculation, extension of the burn area, and loss of resistance to bacterial infection in the wound [2-4].

Intense inflammation, fluid extravasation, and catecholamine surges associated with burns may cause myocardial injury via mechanisms unrelated to traditional coronary artery disease. In patients with burns without coronary disease, increasing troponin levels could emanate from other sources, such as inflammatory cytokine-mediated injury or cellular hypoxia secondary to smoke inhalation and systemic stress. Emerging evidence challenges the concept of troponin as an isolated marker of acute coronary syndromes in these patients [5, 6].

Burn shock, severe hypovolemia, and abnormalities of the microcirculation, which lead to reduced coronary flow, are the early consequences of burns. Burn shock is caused by extensive capillary leak and fluid sequestration

https://doi.org/10.62347/ATYG1182

in the interstitial spaces, resulting in preload reduction, reduced cardiac output, increased peripheral resistance, and increased afterload [7]. Moreover, a hyperinflammatory condition with elevated amounts of TNF, interleukins, and reactive oxygen species contributes to myocardial depression through direct mechanisms that include cardiotoxicity, apoptosis, and disturbances in intracellular calcium homeostasis. In parallel, stress hormones, including catecholamines and cortisol, play a role through their cardiodepressant effect by increasing the heart rate and myocardial oxygen consumption, leading to myocardial ischemia. Stress-induced cardiomyopathy can also develop as a consequence [8]. In this case, myocardial injury is independent of obstructive coronary artery disease and resembles septic cardiomyopathy by a combination of oxygen mismatch, microinfarction, and toxic effects of inflammatory mediators [5].

The traditional monitoring of cardiac performance in patients with burns involves physical examination, electrocardiography, and echocardiography. However, these techniques usually cannot diagnose early myocardial dysfunction before clinical manifestations appear, thus delaying timely intervention [4]. Hemodynamic parameters, such as tachycardia, hypotension, and increased central venous pressure, cannot be reliably interpreted in this population because they can be a consequence of the injury perse, fluid resuscitation, or other complications such as sepsis [9].

Cardiac biomarkers have emerged as useful aids for the early detection and prognostic assessment of cardiovascular complications due to various clinical conditions [10-12]. Troponin isoforms (I and T) are considered the gold standard markers for myocardial necrosis and have been valuable in identifying cardiac involvement in critically ill patients who do not have primary heart disease [13]. However, the elevation of troponin is complex to evaluate in burned patients since its levels can be influenced by injury to skeletal muscle and systemic inflammation [14].

In cases with burns, BNP and NT-proBNP confer complementary advantages. Originally developed for the diagnosis of congestive heart failure, these peptides are released as a consequence of ventricular wall stress and volume overload that is a common finding in severe burns [5, 15]. Unlike troponins, which indicate mainly myocardial damage, BNP and NT-proBNP reflect functional cardiac impairment and are promising predictors of circulatory complications in the intensive care unit [5].

Despite a number of potential advantages, cardiac biomarkers have not been extensively studied or incorporated into routine clinical burn care. Many studies have single-center designs and differ significantly in protocols, biomarker thresholds, and outcomes measured, which delays the development of evidence-based guidelines on the use of cardiac biomarkers in burn patients. Key challenges persist, including the identification of optimal sampling times, the interpretation of results in the context of burn-related physiological alterations, and the incorporation of biomarkers into standard diagnostics [16].

Thus, early diagnosis of cardiac dysfunction in burn patients is highly relevant to management for optimization of fluid therapy, enhancement of hemodynamic monitoring, and cardioprotective strategies [17]. These biomarkers may provide prognostic value for risk stratification and guide decisions on escalation of intensive care and resource utilization.

This systematic review aimed to evaluate the current evidence regarding the diagnostic and prognostic utility of cardiac biomarkers in burn trauma.

Materials and methods

Search strategy

For this systematic review, we conducted a comprehensive search to identify relevant studies examining cardiac biomarkers in patients with burn injuries. The research was performed in compliance with the PRISMA criteria, Preferred Reporting Items for Systematic Reviews and Meta-Analyses, and the Flow Diagram is shown in **Figure 1**. The search was conducted in the PubMed and Google Scholar databases between July 2015 and September 2025. We used the Advanced Search Builder, and keywords were searched in [Title OR Abstract]. Filters were applied to include only research articles published in the English language, using the terms '(Acute burn [Mesh] OR Burn

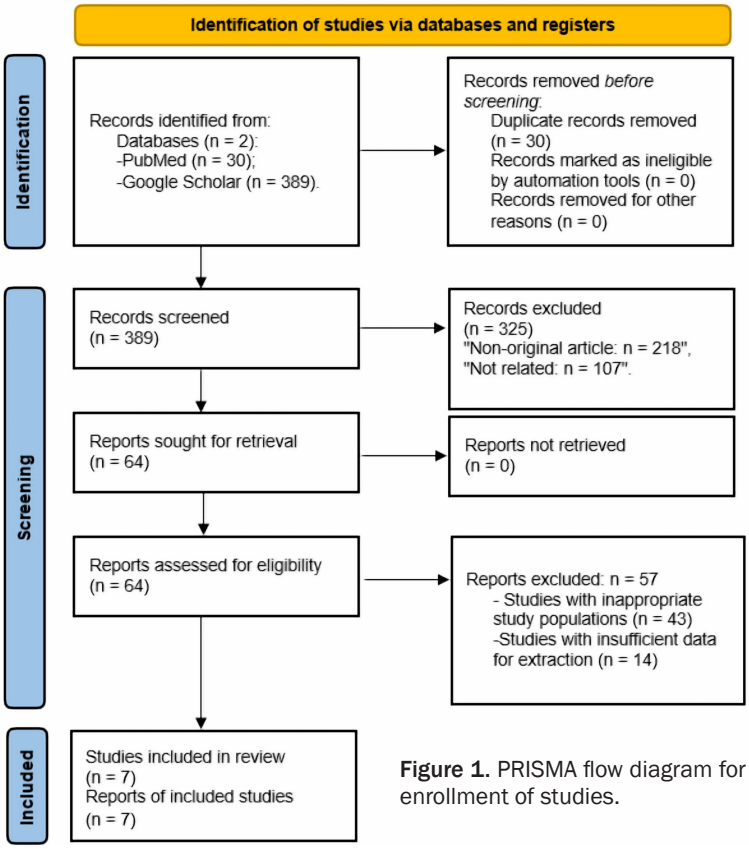

Figure 1. PRISMA flow diagram for enrollment of studies.

injury [Text word] OR Thermal injury [Text word]) AND (Cardiac biomarkers [Text word] OR Troponin [Mesh] OR Brain natriuretic peptide [Text word] OR BNP [Text word] OR NT-proBNP [Text word])'.

Inclusion and exclusion criteria

The studies were considered eligible for review if they included patients with any burn injury, regardless of severity, who had undergone cardiac biomarker testing in the acute phase of their injury, defined as within 72 h post-burn or during early hospitalization. Included studies had to be prospective observational studies, retrospective cohort studies, or case-control studies that could provide data on biomarker levels and their associations with burn severity, clinical outcomes, such as cardiac complications, sepsis, or mortality, or prognostic value. Studies that reviewed biomarker releases with regard to burn size (e.g., total body surface area [TBSA]), toxic myocarditis (TM), or sepsis in relation to burns would take precedence. The main outcome measures reviewed would be the incidence and significance of biomarker elevation and their possible relationships with mortality or cardiac events. Secondary outcomes included specific biomarker patterns, such as the threshold for troponin elevation or BNP sensitivity for myocarditis, and associations with TBSA or survival.

We would exclude studies that included patients with known previous cardiovascular disease, those already receiving cardiac medications at the time of injury, or those with confounding factors such as significant non-burn trauma that may confound biomarker interpretation. We would also exclude case reports, case series, review articles, editorials, and conference abstracts. Studies with data on cardiac biomarkers that were not extractable or without the necessary temporal criteria for assessment would be excluded. Additionally, we would exclude any studies not published in English alone to ensure consistent interpretation of data while minimizing potential translation bias that might affect data extraction and analysis.

Data extraction

The titles and abstracts were reviewed by A.M. After applying inclusion and exclusion criteria, data from studies were extracted based on the requirements of the review, focusing on study design, patient demographics, biomarker types, key findings on elevations and outcomes, and statistical associations. After scanning references in previously published review articles, any relevant studies were considered for inclusion. We obtained seven eligible published research articles in their final versions, which formed the basis of this systematic review.

Results

Study selection and characteristics

Seven original studies were included, featuring both retrospective and prospective designs,

with sample sizes ranging from 40 to 1,621 patients and encompassing adult and mixed-age populations with moderate to extensive burns (TBSA 10->70%). Most studies employed high-sensitivity assays for BNP, NT-proBNP, cardiac troponin I, or troponin T, and several reported other markers. **Table 1** showed the study characteristics of all evaluated studies.

Troponin (I and T) trends and outcomes

A total of four studies have examined cardiac troponin I (cTnI) or troponin T (cTnT) trends in acute-phase patients with burns. Alexander et al. [18] noted that more than a third (36.9%) of 1,621 patients with acute burns had a troponin test performed, which occurred at a higher rate among men, older age patients (≥50 years), and burns greater than or equal to 15% of TBSA; positive cTnI results within the first 24 hours were linked with the occurrence of acute cardiac events, extensive burn surface, and increased mortality in the hospital, and a high cTnI (>20% TBSA) concentration was an independent predictor for mortality. In the study of Segura et al. [19], 2,150 propensity-matched burn patients with increased levels of cTnI (>0.3 ng/mL) within the first 72 hours after burns led to a significant increase in mortality, sepsis, and myocardial infarction at 30 days.

In smaller cohorts, Patel et al. [20] observed that admission cTnI >0.034 ng/mL in patients with ≥10% TBSA burns correlated positively with burn size (P=0.026) but showed no significant relationship with initial heart rate, blood pressure, or mean arterial pressure, and the higher mortality in the elevated troponin group (46.67% vs. 16%) did not reach statistical significance (P=0.065). Kimani et al. [14] reported that cTnT was raised in 61.4% of 83 burn patients at admission, peaked on day 3 (P<0.001), and remained significantly higher in non-survivors compared with survivors on days 0, 3, and 7; day-3 cTnT achieved an AUC of 0.81 (72% sensitivity, 81.1% specificity) for mortality prediction and correlated strongly with TBSA, age, and smoking status (P<0.001). Overall, these studies consistently indicate that early troponin elevation reflects burn severity and systemic stress and is associated with increased short-term mortality and complications, even when not clearly linked to overt hemodynamic instability.

BNP trends and outcomes

Two prospective studies evaluated BNP as a primary marker of ventricular dysfunction and prognosis after burn injury. In their study including 40 adult burn victims with 20-50% TBSA injuries, Abd Elaziz et al. [21] found TM in 16 patients using clinical and echocardiographic criteria; plasma BNP concentrations on day 1, 4 and 7 were significantly elevated in these patients (P<0.0001), and the sensitivity and AUC of day-1 BNP were greater than those of cTnI for early myocarditis diagnosis (sensitivity: 88.8-100%, 95% CI; AUC: 0.888-1.000). In another large study conducted by Hu et al. [22] involving 159 patients with ≥70% TBSA burns, sequential BNP concentrations over 56 days after burn injury were associated with death more strongly than any other markers evaluated (HR=6.831, 95% CI: 6.122-7.539, P<0.0001), and the probability of death was increased dramatically when patients showed BNP concentration ≥519.91 pg/mL in at least two consecutive measurements; this dynamic cut-off value was confirmed to be an independent predictor of survival through multivariate Cox regression analysis and an external validation cohort.

NT-proBNP trends and outcomes

There is a single study conducted to measure the role of NT-proBNP in patients having major burns along with sepsis. The study conducted by Wang et al. [23] involved a total of 204 patients with TBSA ≥50%; among them, 90 patients had sepsis, and NT-proBNP levels in these patients taken at 48 hours from microbiological proof of sepsis showed that there were significantly elevated levels in patients who did not survive compared to those patients who survived; their median levels being 2,900 pg/mL, and level >2,000 pg/mL was a predictor of significantly lower survival rate at 90 days, with mean of 15.08 days.

Discussion

The studies reviewed collectively demonstrate the prognostic and diagnostic value of cardiac biomarkers in patients with acute burns. Specifically, leadership among these biomarkers includes troponins (I and T), BNP, and NT-proBNP, which longitudinally correlate to adverse outcomes, including mortality, sepsis,

Table 1. Summary of studies investigating cardiac biomarkers as predictors of mortality and complications in burn patients

Study	Year	Study Design	Sample Size	Mean of age, year ± SD, IQR	Burn Severity (Mean TBSA %)	Biomarkers Assayed	Measurement Timing	Mortality	Conclusion
Abd Elaziz et al. [21]	2025	Prospective study	40 patients	21-60	All patients were between 20% and 50% TBSA.	BNP, cTnI	Days 1, 4, and 7 post-admission.	10 (TM group) and 11 (non-TM group)	BNP can serve as an effective and early predictor of TM in patients with severe burn injuries, outperforming cTnI in the initial post-burn period.
Hu et al. [22]	2025	Retrospective study with external validation	159 (primary cohort) and 144 (validation cohort)	19-60	≥70% for all patients.	BNP, proBNP, CK-MB, IL-6, Myoglobin, D-dimer, cTnI	Within 56 days post-burn.	74 (primary cohort) + 61 (validation cohort)	In patients with massive burns, BNP is a powerful predictor of mortality. A new dynamic threshold (≥519.91 pg/ml) can effectively stratify high-risk patients.
Segura et al. [19]	2024	Retrospective study	4,300 patients (2,150 with elevated cTnI, 2,150 with non-elevated cTnI)	~ 56±16.6	Mild (<20%) and severe (>20%) TBSA.	cTnI	Within 24 and 72 hours of injury.	343 (elevated cTnI group) and 147 (non-elevated cTnI group)	An elevated cTnI level (>0.3 ng/mL) shortly after a burn injury is a powerful predictor of increased 30-day mortality, MI, and sepsis, independent of burn size.
Kimani et al. [14]	2022	Cross-sectional study	91 patients	20.62±15.3	TBSA ≥15 (n=64) TBSA ≤15 (n=27).	cTnT	9 hours after burns and before 12 days form the day of burns.	-	This study has demonstrated that burns patients have elevated serum cardiac troponin T compared to the general population.
Wang et al. [23]	2022	Retrospective study	204 patients (90 with sepsis and 114 without sepsis)	~37.14	69.56%	NT-proBNP, Procalcitonin, Platelet count.	48 hours after a positive pathogenic culture confirmed sepsis.	17.74% (Sepsis group) and 13.16% (non-sepsis group)	For patients with NT-proBNP >2000 pg/mL, the mortality rate was 91.67%. Therefore, NT-proBNP is a valuable and independent prognostic marker for mortality in patients with major burns complicated by sepsis.
Patel [20]	2021	Prospective study	40 (25 had normal TnI and 14 had elevated TnI)	~51.87±18.8	~31%	cTnI	-	Elevated TnI: 46.67%; Normal TnI: 16%	The increase in TnI levels following burn injury does not seem to align with the clinical indicators of cardiac performance, such as heart rate and blood pressure. Moreover, a positive correlation was observed between TBSA and elevated TnI levels.
Alexander et al. [18]	2018	Retrospective study	285 patients	50.8±19.3	21.3±18.5%	cTnI	Within 24 hours of admission.	13 (4.6%)	Raised cTnI is common after significant burns (>20% TBSA) and is an independent predictor of mortality.

TBSA: total body surface area, BNP: B-type natriuretic peptide, cTnI: cardiac troponin I, cTnT: cardiac troponin T, proBNP: pro-B-type natriuretic peptide, NT-proBNP: N-terminal pro-B-type natriuretic peptide, CK-MB: creatine kinase-MB, IL-6: interleukin-6, TM: toxic myocarditis, MI: myocardial infarction, SD: standard deviation, IQR: interquartile range, TnI: troponin I, pg/mL: picograms per milliliter, and ng/mL: nanograms per milliliter.

TM, and other cardiac issues, independent of pre-existing coronary artery disease. Troponins are elevated in the first 24-72 hours following an acute burn, indicating myocardial stress or injury due to systemic inflammation, fluid shifts, and catecholamines, not, as is traditionally thought, ischemia.

Troponin has emerged as a reliable marker of burn severity and poor prognosis. Patel et al. [20] demonstrated that increased admission troponin I (>0.034 ng/mL) was significantly related to a greater TBSA burned (P=0.0264) but had no relation to hemodynamic parameters like heart rate, blood pressure, or mean arterial pressure (P>0.05). This indicates that the elevation of troponin in burns can be of importance, above all, in the context of the overall injury and the systemic stress in those patients, rather than because of direct cardiac dysfunction. This is supported by the nonsignificant trend for higher mortality in the group with increased troponin levels (46.67% vs. 16%, P=0.0654). Likewise, peak troponin T levels on the third day after burning were found by Kimani et al. [14] to be the strongest predictor of mortality (AUC: 0.81; sensitivity 72%, specificity 81.1%), while the association of peak TnT with TBSA, age, and smoking was highly significant (P<0.001). All these findings run against the traditional consideration of troponin as an exclusive marker of acute coronary syndromes, therefore pointing out other etiologies in burnt patients, including inflammatory cytokines and hypoxia due to smoke inhalation.

From a clinical standpoint, our view is that routine troponin evaluations should be prioritized in burn populations that are at a high risk for early detection of cardiovascular complications. This includes individuals with TBSA ≥15-20%, older individuals, and suspected inhalation injuries. Routine troponin levels can be beneficial because they can assist clinicians in determining whether they need to escalate their monitoring and provide additional support to patients who have an elevated value, even when there are few changes to vital signs or echocardiographic findings that can be used to establish hemodynamic instability. In addition, troponin levels alone should never be used for the diagnosis of acute coronary syndrome in this population, but should instead be evaluated in conjunction with clinical findings, ECG changes, and imaging in order to avoid unnecessary invasive procedures.

BNP and NT-proBNP, due to their association with ventricular wall tension and hemodynamic instability, offer specific promise for early diagnosis of complications such as sepsis and TM. Abd Elaziz et al. [21] documented BNP's superior sensitivity (88.8-100%, 95% CI) compared to troponin I for predicting TM in moderate-to-severe burns (20-50% TBSA), with an AUC of 0.888-1.000 on day 1 (P<0.0001). The follow-up work by Hu et al. [22] identified BNP as the biomarker with the strongest mortality association for extensive burns (≥70% TBSA). A threshold of ≥519.91 pg/mL across consecutive measurements predicted a significantly higher risk of mortality (P=0.005) with strong discriminative power (AUC: 0.800-0.909 at 8-12 weeks). Wang et al. [23] further supported the utility of NT-proBNP, showing that levels >2000 pg/mL serve as an independent risk factor for poor 90-day survival among patients with sepsis and major burns (≥50% TBSA). These natriuretic peptides collectively allow clinicians to predict sepsis or myocardial damage, thus enabling timely intervention with antimicrobial therapy or echocardiography.

This information implies that natriuretic peptides should be regarded as valuable markers for diagnosing cardiocirculatory failure after burns in terms of recognizing TM or sepsis-associated heart dysfunction. Testing BNP or NT-proBNP becomes essential in situations where tachycardia and hypotension may be caused by inadequate fluid resuscitation, excessive resuscitation, or development of septic shock. When the above-mentioned signs develop, high concentrations of natriuretic peptides can facilitate early diagnosis of heart failure and justify conducting an echocardiography, determining the best approach to fluid and drug administration, and monitoring a patient in the intensive care unit even in absence of evident heart failure. Furthermore, using a dynamic threshold for BNP established by Hu et al. [22] demonstrates additional advantages of repeated BNP testing over one-time measurement.

Physiologically, myocardial dysfunction following thermal injury is characterized by slowed isovolemic relaxation, impaired contractility, and decreased left ventricular diastolic compliance [3, 24]. This is manifested primarily by a

decrease in cardiac output and metabolic rate, with compensatory increments in heart rate and peripheral vascular resistance, which increase myocardial oxygen demand, ultimately leading to right and left heart deficits [25]. Depending on the extent of the burn, this deficit may result in cardiogenic shock, which has been identified as a major cause of failed resuscitation [26, 27].

The integration of these biomarkers into clinical practice may further enhance risk stratification and improve outcomes. Routine testing of biomarkers such as troponin in high-risk populations (e.g., ≥15-20% TBSA, elderly, or smoke inhalation) could indicate the early employment of hemodynamic monitoring, while BNP/NT-proBNP may guide sepsis management in severe cases. However, challenges persist, including heterogeneous cutoff thresholds across studies; for instance, troponin I cutoffs ranged from 0.034 ng/mL to >0.3 ng/mL, which prevents standardization.

Future research involving prospective multicenter cohorts will define burn-specific thresholds through serial measurements, compare isoforms (e.g., troponin I vs. T), and apply advanced imaging modalities (e.g., cardiac magnetic resonance) for validation. Underrepresented groups include pediatrics and those suffering from severe inhalation injuries. Finally, machine learning models incorporating biomarkers may allow for personalized, real-time prognostication, where randomized controlled trials will be necessary to evaluate biomarker-guided therapies.

Conclusion

In conclusion, cardiac biomarkers, especially troponin, BNP, and NT-proBNP, are useful prognostic tools for understanding the cardiovascular consequences of acute thermal injury and are associated with mortality and significant complications in burn patients. While they could serve as an important mechanism for early risk stratification and prognostication, the routine application of these biomarkers will require further research demonstrating sound methodology beyond the current evidence's shortcomings. Our review confirms the promising value of biomarker-guided management and underscores the urgent need for immediate, focused investigations to optimize the use and benefit of biomarkers for outcomes of this uniquely high-risk patient group.

Disclosure of conflict of interest

None.

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