International Journal of Research in Medical Sciences
*Gupta AK et al. Int J Res Med Sci. 2025 Mar;13(3):1140-1146*
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20250679

**Original Research Article**

# Evaluation heparin binding protein as a prognostic biomarker for diagnosis of sepsis at tertiary care hospital, North India

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**Received:** 01 January 2025
**Revised:** 02 January 2025
**Accepted:** 03 February 2025

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## ABSTRACT

**Background:** Sepsis is a life-threatening condition marked by an uncontrolled inflammatory response to infection, leading to organ dysfunction and high mortality. In India, severe sepsis burdens ICU resources and impacts patient outcomes. This study aimed to evaluate heparin-binding protein (HBP) as a prognostic biomarker for assessing sepsis severity.
**Methods:** This prospective study was conducted at the department of medicine, G. S. V. M. medical college, Kanpur, from December 2022 to May 2024, including 113 adult patients suspected of sepsis or septic shock. Demographic data, HBP levels, and correlations with age, sex, disease severity, and other biomarkers C reactive protein (CRP and procalcitonin) were analyzed. Survival rates across different disease severities were also assessed.
**Results:** The mean age of participants was 53.2±19.3 years. Baseline HBP levels were significantly higher in infection cases compared to non-infection cases (11.21±5.51 ng/ml vs. 4.31±3.72 ng/ml, $p<0.001$). HBP levels decreased significantly over 72 hours but remained elevated in non-survivors (9.81±6.25 ng/ml vs. 7.17±5.18 ng/ml, $p=0.001$). HBP was more effective than CRP and procalcitonin in predicting infection severity and outcomes.
**Conclusions:** HBP is a promising biomarker for assessing sepsis severity and predicting survival. Elevated HBP levels correlate with increased infection severity and mortality. HBP offers an advantage in early diagnosis and prognosis, and further research is needed to optimize its use in sepsis management.

**Keywords:** HBP, Biomarkers, Disease severity, Prognostic indicator, C-reactive protein, ICU, Infection, Mortality

## INTRODUCTION

Sepsis is a critical condition that occurs when the body's response to an infection triggers widespread inflammation. This inflammation can cause damage to tissues and organs, leading to organ failure and possibly death. According to the sepsis alliance, sepsis is a complex syndrome that results from an unregulated inflammatory response to an infection, which can be caused by bacteria, viruses, fungi, or parasites.[1] Severe sepsis is defined as sepsis accompanied by organ dysfunction, hypoperfusion, or hypotension. This stage of sepsis involves the failure of one or more organs, which can result from the body's overwhelming response to infection.[2] Symptoms of severe sepsis may include difficulty breathing, changes in mental status, significantly decreased urine output, abnormal heart function, and unexplained metabolic acidosis.

Severe sepsis is a significant public health issue in India, with a notable prevalence in ICU admissions. The Indian intensive care case mix and practice patterns study (INDICAPS) in 2014 found that severe sepsis occurred in approximately 16.45% of ICU admissions, with a high mortality rate of 56.3%. A study published in the journal of global health estimated that India accounts for nearly one-third of global sepsis cases, largely due to the high

prevalence of infectious diseases and inadequate healthcare resources.[3] Whereas, in Uttar Pradesh, severe sepsis accounted for approximately 20% of ICU admissions, with a mortality rate exceeding 40%. This reflects the broader national trends of high sepsis burden and poor outcomes due to factors such as delayed diagnosis, antibiotic resistance, and limited healthcare resources.[4]

The pathophysiology of sepsis involves a complex interplay of the host immune response and the invading pathogens. Initially, the immune system detects the pathogen and activates a cascade of inflammatory responses to eliminate the infection. However, in sepsis, this response becomes dysregulated, leading to widespread inflammation, endothelial dysfunction, and increased vascular permeability.[2] This systemic inflammation results in impaired tissue perfusion, cellular injury, and multiple organ dysfunction. Key mediators such as cytokines, chemokines, and reactive oxygen species play crucial roles in this process, exacerbating the severity of the condition.[5]

Common symptoms of sepsis include fever or hypothermia, tachycardia (heart rate >90 beats per minute), and tachypnoea (respiratory rate >20 breaths per minute), all indicative of the body's effort to fight the infection. Symptoms of severe sepsis may include difficulty breathing, changes in mental status, significantly decreased urine output, abnormal heart function, and unexplained metabolic acidosis.[2] Organ dysfunction becomes more pronounced, marked by signs such as acute respiratory distress syndrome (ARDS), significant jaundice, and severe lactic acidosis. The systemic inflammation causes widespread endothelial damage and capillary leakage, leading to multiple organ failure.[6] Approximately 20-30% of sepsis patients do not show typical symptoms of organ dysfunction at admission but progress to severe sepsis within 24 hours of admission.

Sepsis is diagnosed through a combination of clinical evaluation and laboratory tests. Clinicians assess signs of infection, systemic inflammation, and organ dysfunction. Common tests include blood cultures to identify the causative pathogen, complete blood count (CBC) to check white blood cell count, lactate levels to assess tissue hypoxia, and markers like CRP and procalcitonin to detect inflammation.[7] The SOFA score is used to determine the level of organ dysfunction in patients with sepsis. A new quantitative index, the change in SOFA score, analyzes the fluctuations in organ function by measuring the change in SOFA score.[8] Identifying reliable biomarkers for sepsis has been challenging, but multiomics offers hope for a personalized approach.[9] HBP, also known as azurocidin or cationic antimicrobial protein of 37 KDa (CAP37), is a promising candidate. Stored in neutrophil granules, HBP is rapidly released in response to bacterial structures and inflammatory stimuli, making it one of the earliest detectable markers of infection. HBP functions as a chemoattractant, particularly for monocytes, and induces vascular leakage and edema contributing to hypotension and organ dysfunction.[10] These attributes make HBP a critical biomarker for early diagnosis, severity assessment, and prognostication in sepsis management. The present study was designed to evaluate HBP as a diagnostic parameter for the diagnosis of severe sepsis.

### *Aim and objectives*

Aim of the study was to evaluate HBP as a prognostic biomarker for analyzing severity of sepsis.

Objectives of the study were to evaluate demographic characteristics of enrolled patients, to evaluate correlation of HBP with age group and sex of patients, to evaluate HBP for analysing disease severity, to compare HBP with other biomarker (CRP and procalcitonin) and to observe the survival rate of enrolled patients in different disease severities.

## METHODS

This prospective and analytical study was conducted in the department of medicine at G. S. V. M. Medical College, Kanpur, from December 2022 to May 2024. The study included 113 patients over 18 years old, suspected of sepsis or septic shock, presenting to the emergency department. Inclusion criteria were respiratory rate >25 breaths/min, heart rate >120 beats/min, altered mental status, systolic blood pressure <100 mmHg, and oxygen saturation <90% without oxygen or <93% with oxygen. Exclusion criteria included patients under 18, those not consenting, prior antibiotic treatment within 24 hours before admission, neutropenia, primary coagulation abnormalities, haematological malignancy, immunosuppressive therapy, chronic infections like tuberculosis, and those on haemodialysis. Data were collected using a predetermined proforma after obtaining informed consent. Data were entered and analysed using MS excel and SPSS Version 20.0. Continuous variables were expressed as means and standard deviation or median and interquartile range, while categorical variables expressed as percentages. Statistical analyses included Chi-square tests for categorical variables, one-way ANOVA for parametric continuous variables, and Pearson correlation for continuous variables, with significance threshold of $p<0.05$.

## RESULTS

The study analysed the age, sex, clinical complaints, vital signs, co-morbidities biochemical and haematological parameters, organ dysfunction, intubation need and HBP levels along with its comparison with other biomarkers across different patient groups to determine significant association with the diagnosis of severe sepsis.

The study's age distribution is as follows: 18-30 years (21.2%), 31-40 years (9.7%), 41-50 years (10.6%), 51-60 years (19.5%), 61-70 years (20.4%), and over 70 years (18.6%). The mean age is 53.2±19.3 years, with the highest representation in the 18-30 and 61-70 age groups.

The mean age of the study cases was 53.2±19.3 years. The distribution of cases by sex is nearly equal, with 56 males (49.6%) and 57 females (50.4%). This balance ensures the study findings are representative of both genders.

On admission, 69.9% (79 individuals) did not have OD, while 30.1% (34 individuals) did. Within 72 hours, those without OD decreased to 56.6% (64 individuals), and those with OD increased to 43.4% (49 individuals), indicating a rise in OD occurrence within the first 72 hours.

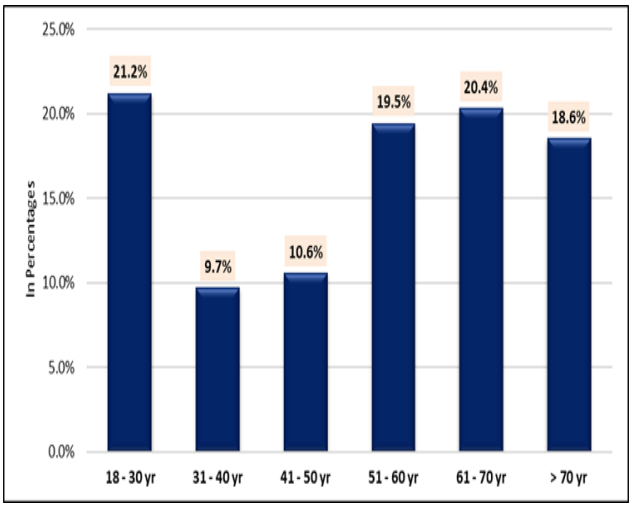


**Figure 1: Distribution of cases according to age.**

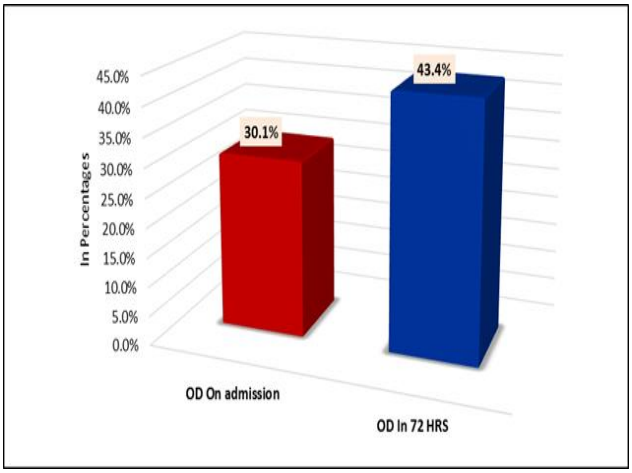


**Figure 2: Cases are distributed according on the OD status.**

**Table 1: Case distribution by outcome.**

| Outcome | N | Percentage (%) |
|---|---|---|
| **Expired** | 42 | 37.2 |
| **Survive** | 71 | 62.8 |

The outcome data revealed that out of the total cases, 42 individuals (37.2%) expired, whereas 71 individuals (62.8%) survived. This shows a survival rate of nearly two-thirds, with slightly more than one-third of the cases resulting in death.

The distribution of cases based on the infection status showed that 54 cases (47.8%) were confirmed infections, while 20 cases (17.7%) were categorized as probable infections. Additionally, 11 cases (9.7%) were identified as viral infections. There were 4 cases (3.5%) where infection was probable but not confirmed, and 24 cases (21.2%) where no infection was detected.

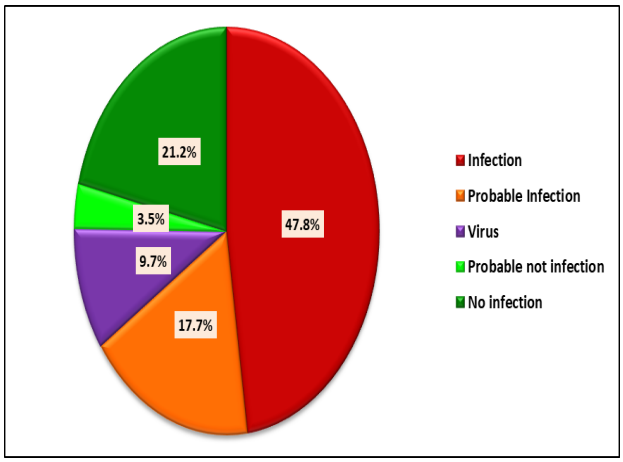


**Figure 3: The case distribution by groups.**

The outcome distribution among patients with different diagnoses was as follows. In the infection group, 20 individuals (37.0%) expired, while 34 individuals (63.0%) survived. Among those with probable infection, 7 individuals (35.0%) expired and 13 individuals (65.0%) survived. For patients with a viral infection, 4 individuals (36.4%) expired, whereas 7 individuals (63.6%) survived. In the probable not infection group, all 4 individuals (100%) survived, with no deaths reported. Among patients with no infection, 11 individuals (45.8%) expired and 13 individuals (54.2%) survived. The chi-square value was 3.18 with a p-value of 0.528, indicating no significant association between the outcome and patient diagnosis.

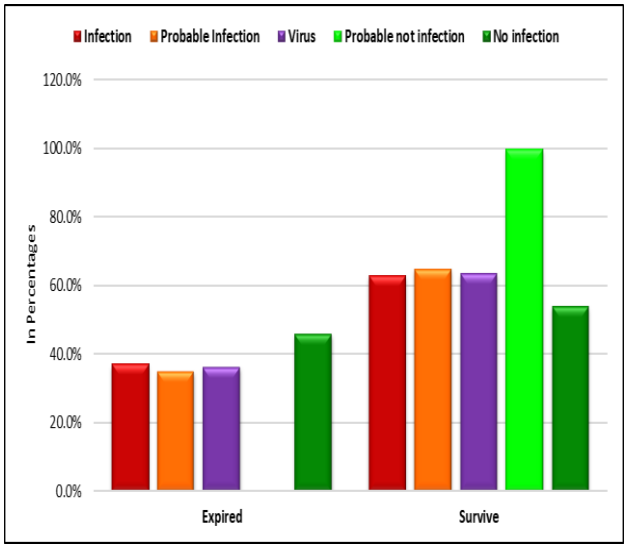


**Figure 4: Association of outcome with patient diagnosis.**

**Table 2: Association of HBP different category of patient diagnosis.**

| HBP level | Infection | | Probable infection | | Viral infection | | Probable not infection | | No infection | | ANOVA | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | Mean | SD | Mean | SD | Mean | SD | F value | P value |
| **Baseline** | 11.21 | 5.51 | 7.37 | 4.41 | 6.63 | 5.22 | 4.73 | 2.15 | 4.31 | 3.72 | 7.58 | <0.001 |
| **At 72 Hr** | 5.55 | 3.05 | 4.77 | 2.60 | 4.18 | 2.95 | 3.67 | 1.43 | 2.16 | 1.14 | 3.67 | 0.008 |

Association of HBP levels with patient diagnosis across different categories-infection, probable infection, virus, probable not infection, and no infection-was examined using ANOVA. At baseline, HBP levels varied significantly across groups: 11.21 (SD=5.51) in infection group, 7.37 (SD=4.41) in probable infection, 6.63 (SD=5.22) in virus, 4.73 (SD=2.15) in probable not infection, and 4.31 (SD=3.72) in no infection (f=7.58, p<0.001).

Similarly, at 72 hours, HBP levels showed significant variation: 5.55 (SD=3.05) in Infection, 4.77 (SD=2.60) in probable infection, 4.18 (SD=4.95) in virus, 3.67 (SD=1.03) in probable not infection, and 2.16 (SD=1.14) in no infection (f=3.67, p=0.008). These findings indicate that HBP levels are significantly associated with different diagnostic categories, suggesting its potential utility as a biomarker for distinguishing between infection statuses in clinical settings.

**Table 3: Comparison of HBP, CRP and procalcitonin level between no infection and infection group.**

| Parameters | No infection | | Infection | | Unpaired t test | |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | T value | P value |
| **HBP** | 4.31 | 3.72 | 11.21 | 5.51 | 4.78 | <0.001 |
| **HBP at 72 hr** | 2.16 | 1.14 | 5.55 | 3.05 | 3.09 | 0.002 |
| **CRP** | 12.89 | 23.43 | 75.42 | 44.82 | -1.21 | 0.031 |
| **Procalcitonin** | 1.62 | 2.20 | 12.29 | 27.95 | -0.53 | 0.008 |

The comparison of HBP, CRP, and procalcitonin levels between patients with and without infection reveals significant differences as determined by unpaired t tests. In the infection group, HBP levels were markedly higher with a mean of 11.21 (SD=5.51), compared to 4.31 (SD=3.72) in the no infection group, resulting in a substantial t=4.78 (p<0.001). At 72 hours, HBP levels remained elevated in the infection group (mean=5.55, SD=3.05) compared to the no infection group (mean=2.16, SD=1.14), yielding a t=3.09 (p=0.002). Conversely, CRP levels were slightly higher in the infection group (mean=75.42, SD=44.82) than in the no infection group (mean=12.89, SD=23.43), although this difference reach statistical significance (t=-1.21, p=0.031). Procalcitonin levels showed significant difference between the infection (mean=12.29, SD=27.95) and no infection (mean=1.62, SD=2.20) groups, with a t=-0.53 (p=0.008). These results underscore the utility of HBP as a potentially valuable biomarker for distinguishing infection status, while CRP and procalcitonin may have nuanced roles in this context.

In comparing the levels of HBP, CRP, and Procalcitonin between patients who expired and those who survived, several key observations emerge from the unpaired t-tests conducted. For HBP, the mean levels were 9.81 (SD=6.25) in expired cases and 7.17 (SD=5.18) in survived cases, with a significant t-value of 0.59 (p=0.001). Similarly, HBP levels at 72 hours showed means of 5.11 (SD=3.66) and 3.43 (SD=2.20) in expired and survived cases, respectively, yielding a t=0.84 (p=0.008). CRP levels were 74.29 (SD=53.93) and 68.25 (SD=47.06) for expired and survived cases, with a t=0.62 (p=0.533), indicating no significant difference. Procalcitonin levels were virtually identical between expired (mean=9.58, SD=22.95) and survived (mean=9.57, SD=23.03) cases, resulting in a negligible t=0.00 (p=1.000). These findings suggest that there were statistically significant differences in these biomarker levels between patients who survived and those who did not, indicating that these HBP markers may dependently predict survival outcomes in this cohort.

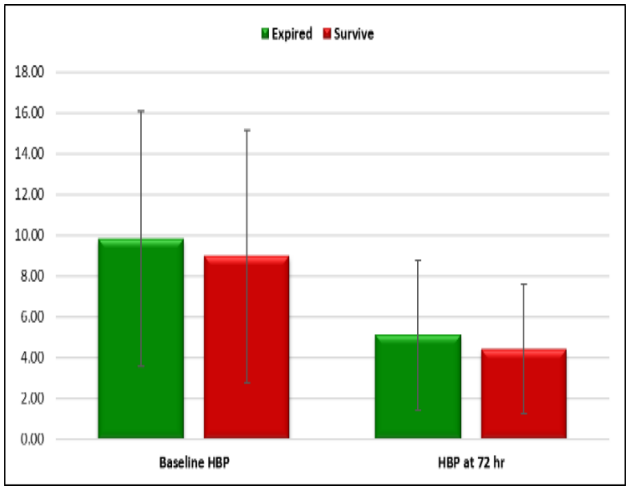


**Figure 5: Comparison of HBP, CRP and procalcitonin level between expired and survived cases.**

In comparing the levels of HBP, CRP, and procalcitonin between patients who has probable infection and those

who has probable not Infection, several key observations emerge from the unpaired t-tests conducted. For HBP, the mean levels were 7.37 (SD=4.41) in probable infection cases and 4.73 (SD=2.1) in probable not infection cases, with a not significant t-value of 0.59 (p=0.321). Similarly, HBP levels at 72 hours showed means of 4.77 (SD=2.60) and 3.67 (SD=1.43) in probable infection and probable not infection cases, respectively, yielding a t=0.34 (p=0.520). CRP levels were 69.29 (SD=57.08) and 22.99 (SD=14.06) for probable infection and probable not infection cases, with a t=0.42 (p=0.033), indicating significant difference. Procalcitonin levels were virtually identical between probable infection (mean=7.57, SD=17.01) and probable not infection (mean=2.73, SD=3.74) cases, resulting in a significant difference t=0.61 (p=0.040). These findings suggest that there were statistically significant differences in these biomarker levels between patients who probable infection and probable not infection, indicating that these HBP markers may not dependently predict probable infection outcomes in this cohort.

**Table 4: Comparison of HBP, CRP and procalcitonin level between probable infection and probable not infection.**

| Parameters | Probable infection | | Probable not infection | | Unpaired t test | |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | T value | P value |
| **HBP** | 7.37 | 4.41 | 4.73 | 2.1 | 0.59 | 0.321 |
| **HBP at 72 hr** | 4.77 | 2.60 | 3.67 | 1.43 | 0.34 | 0.520 |
| **CRP** | 69.29 | 57.08 | 22.99 | 14.06 | 0.42 | 0.033 |
| **Procalcitonin** | 7.57 | 17.01 | 2.73 | 3.74 | 0.61 | 0.040 |

**Table 5: Comparison of HBP, CRP and procalcitonin level between virus and no infection.**

| Parameters | Virus | | No infection | | Unpaired t test | |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | T value | P value |
| **HBP** | 6.63 | 5.22 | 4.31 | 3.72 | 2.49 | 0.648 |
| **HBP at 72 hr** | 4.18 | 2.95 | 2.16 | 1.14 | 2.39 | 0.690 |
| **CRP** | 47.84 | 58.24 | 12.89 | 8.12 | 7.43 | 0.008 |
| **Procalcitonin** | 5.38 | 3.49 | 1.62 | 2.20 | 0.63 | 0.040 |

In comparing the levels of HBP, CRP, and procalcitonin between patients who has virus and those who has no infection, several key observations emerge from the unpaired t tests conducted. For HBP, the mean levels were 6.63 (SD=5.22) in virus cases and 4.31 (SD=3.72) in no infection cases, with a not significant t-value of 2.49 (p=0.648). Similarly, HBP levels at 72 hours showed means of 4.18 (SD=2.95) and 2.16 (SD=1.14) in virus and no infection cases, respectively, yielding a t=2.39 (p=0.690). CRP levels were 47.84 (SD=58.24) and 12.89 (SD=8.12) for virus and no infection cases, with a t=7.43 (p=0.008), indicating significant difference.

Procalcitonin levels were virtually identical between virus (mean=5.38, SD=3.49) and no infection (mean=1.62, SD=2.20) cases, resulting in a significant difference t=0.63 (p=0.040).

These results reveal that there were statistically significant changes in these biomarker levels between patients with and without viral infection, suggesting that these HBP indicators may not be a reliable indicator of the likelihood of infection in this population.

## DISCUSSION

The present study entitled HBP as a prognostic biomarker for diagnosis of sepsis was carried out in, KPS post graduate institute of medicine, G.S.V.M. medical college, Kanpur from December 2022 to May 2024.

***Parameters of study population***

*Age and sex of patients and aetiology of sepsis*

Kahn et al studied 718 emergency department sepsis patients, with 194 males and 524 females, all over 18 years old.[11] In a subset of 113 patients, the average age was 53.2±19.3 years. Age distribution was as follows: 20.4% between 61-70 years, 18.6% over 70 years, 21.2% between 18-30 years, 9.7% between 31-40 years, 10.6% between 41-50 years, and 19.5% between 51-60 years. Gender distribution was nearly equal, with 49.6% males and 50.4% females.

*Chief complaint related to sepsis*

According to Zuo et al there were 326 sepsis patients in total. Fever was the most prevalent complaint, with 54 reports-or 47.8% of total-being made.[12] Breathlessness, which afflicted 50 people/44.2% of participants, came next. Cough and sputum recorded by 18 people (15.9%), and altered sensorium was observed in 28 instances (24.8%). Less often reported symptoms nausea/ vomiting (10.8%) and abdominal discomfort (14.4%), respectively.

*How sepsis cases are distributed according to the state of infection*

Kahn et al identified 524 sepsis patients: 18.3% confirmed infections, 16.03% probable, 7.44% viral, 45% no

infection, and 13.16% likely but unconfirmed.[11] Our study found 47.8% confirmed, 17.7% probable, 9.7% viral, 3.5% likely but unconfirmed, and 21.2% no infection.

*Association between the patient's infection condition, OD, and requirement for intubation*

In our investigation an analysis of the relationship between organ dysfunction (OD) and patient diagnosis found no evidence of a significant relationship. Fourteen people (25.9%) had an infection at the time of admission, 5 people (25.0%) had a probable infection, 5 people (45.5%) had a virus, 1 person (25.0%) was most likely not infected, and 9 people (37.5%) had no infection. In this comparison, the chi-square value was 2.60, and the p=0.627, meaning that there was no significant correlation.

In the span of 72 hours, 21 people (38.9%) had an infection, 8 people (40.0%) had a likely infection, 5 people (45.5%) had a virus, 0 people were probably not infected, and 15 people (62.5%) had no infection. There was also no significant correlation found between the patient's diagnosis and organ failure within 72 hours, as indicated by the chi-square value of 7.19 and p=0.126 for this comparison.

There were notable correlations between individuals with various illnesses and the requirement for intubation. Of those who had an infection, 42 (77.8%) did not need to be intubated, while 12 (22.2%) did. Out of the patients who had a suspected infection, 18 (90.0%) did not require intubation, whereas 2 (10.0%) did. Of the patients suffering from a viral infection, 8 (72.7%) did not require intubation, while 3 (27.3%) did. Intubation was not necessary for any of the four patients (100%) who had a suspected non-infection. Twelve (50.0%) of those without an infection did not require intubation, whereas twelve (50.0%) did.

*HBP level: a descriptive summary and its association with patient diagnosis*

In our investigation HBP levels were assessed both at baseline and after 72 hours. The baseline HBP level was 11.28 ng/mL on average, with a 5.57 standard deviation. After 72 hours, the mean HBP level decreased to 6.68 ng/mL with a standard deviation of 3.39, indicating a significant decrease in HBP levels over time.

At baseline and after 72 hours, the levels of HBP in the various patient groups were examined. The mean HBP level at baseline was 4.73 ng/ml (SD=2.15) in the group that was probably not infected, and 6.63 ng/ml (SD=5.22) in the virus group. The mean HBP level was 11.21 ng/ml (SD=5.51) for the infection group, 7.37 ng/ml (SD=4.41) for the probable Infection group, and 4.31 ng/ml (SD=3.72) for the no infection group. With an f=7.58 and a p<0.001, the ANOVA analysis revealed a significant difference, suggesting variability among the groups.

All groups' HBP levels dropped after 72 hours. At 4.18 ng/ml (SD=2.95), the mean HBP level in the virus group and lowest in the no infection group (2.16 ng/mL, SD=1.14). The average results for the infection, probable Infection group and probable not Infection group were 5.55 ng/ml (SD=3.05), 4.77 ng/ml (SD=2.60), and 3.67 ng/ml (SD=1.43), respectively. With an f=3.67 and a p=0.008, the ANOVA analysis for the 72-hour data did not reveal any significant differences, indicating that there was no significant variation in the HBP levels across the groups at this time.

Zuo et al the HBP values of the infection, sepsis, septic shock, and control groups were 18.0 (9.9-32.1), 24.0 (14.1-56.4), 45.7 (24.8-107.9), and 69.0 (33.8-150.9) ng/ml, on average (p<0.001). Using HBP, it may be possible to distinguish between patients who have an infection or who do not have sepsis.

*HBP, CRP, and procalcitonin level comparison in the infection and no infection groups*

In comparing HBP, CRP, and procalcitonin levels between infection and no infection groups, significant differences were found. HBP was notably higher in the infection group (mean 11.21, SD=5.51) compared to the no infection group (mean 4.31, SD=3.72), with a significant t=4.78 (p<0.001). At 72 hours, HBP levels remained significantly higher in the infection group (mean 5.55, SD=3.05) than in the no infection group (mean 2.16, SD=1.14; t=3.09, p=0.002). CRP levels were marginally higher in the infection group (mean 75.42, SD=44.82) compared to the no infection group (mean 27.89, SD=13.43; t=-1.21, p=0.031), while procalcitonin levels also significantly differed (infection mean 12.29, SD=27.95; no infection mean 6.62, SD=11.20; t=-0.53, p=0.008). Kahn et al reported similar findings, with higher HBP (mean 8.97 vs. 3.01), procalcitonin (mean 5.46 vs. 1.57), and CRP (mean 38.7 vs. 1.57) in the infection group compared to those with organ dysfunction.

*HBP, CRP, and procalcitonin level comparison in expired and survived cases*

Unpaired t tests comparing HBP, CRP, and Procalcitonin levels between patients who survived and those who expired revealed significant findings for HBP. Mean HBP levels were higher in expired patients (9.81, SD=6.25) compared to survivors (7.17, SD=5.18; t=0.59, p=0.001). At 72 hours, HBP levels were again higher in expired patients (5.11, SD=3.66) versus survivors (3.43, SD=2.20; t=0.84, p=0.008). CRP levels showed no significant difference (expired mean 74.29, SD=53.93; survived mean 68.25, SD=47.06; t=0.62, p=0.533), and procalcitonin levels were almost identical (expired mean 9.58, SD=22.95; survived mean 9.57, SD=23.03; t=0.00, p=1.000).

These results suggest that HBP may be a useful predictor of survival outcomes in this cohort.

## CONCLUSION

The study evaluating HBP as a prognostic biomarker for sepsis severity reveals its potential as a significant indicator of infection status and disease outcome. The demographic analysis highlights a diverse patient population with a mean age of 53.2 years, a near-equal gender distribution, and varying comorbidities. Fever and shortness of breath were the most prevalent symptoms, with a considerable proportion of patients requiring intubation and experiencing organ dysfunction. The study found a higher mortality rate among those with elevated HBP levels, particularly at baseline, where HBP was significantly higher in infection cases compared to no infection cases. This trend persisted at 72 hours, indicating that HBP could effectively differentiate between infection statuses and predict severe outcomes. Although other biomarkers like CRP and procalcitonin also varied with infection status, HBP showed a distinct pattern, with higher levels correlating with both infection presence and poorer survival outcomes. While CRP and procalcitonin levels did not significantly predict mortality, HBP emerged as a promising prognostic tool, potentially aiding in the early identification and management of severe sepsis. The findings underscore the need for further research to refine HBP's role in clinical practice and confirm its utility in predicting sepsis severity and patient survival.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: The study was approved by the Institutional Ethics Committee*

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**Cite this article as:** Gupta AK, Verma RK, Giri R, Gupta AC, Gupta S. Evaluation heparin binding protein as a prognostic biomarker for diagnosis of sepsis at tertiary care hospital, North India. Int J Res Med Sci 2025;13:1140-6.