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Outcomes in terms of Mortality in patients of Community Acquired Pneumonia with Raised B-Type Natriuretic Peptide

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ABSTRACT

Background: Community-acquired pneumonia (CAP) remains a leading cause of morbidity and mortality worldwide. While B-type natriuretic peptide (BNP) is primarily recognized as a cardiac stress biomarker, emerging evidence suggests its elevation in non-cardiac conditions contribute to myocardial strain. **Aim:** To evaluate the outcomes in terms of mortality and clinical progression among patients with CAP exhibiting elevated BNP levels, and to assess the association of BNP with inflammatory markers, oxygen requirement, and other clinical endpoints. **Methodology:** A prospective, comparative study was conducted involving 60 patients diagnosed with CAP and 60 age- and sex-matched healthy controls. BNP, C-reactive protein (CRP), white blood cell count (WBC), and erythrocyte sedimentation rate (ESR) were measured in both groups. In CAP patients, BNP levels were assessed at admission and post-treatment. Clinical data including symptom duration, hospital stay, FiO_2 requirement, PaO_2, and outcomes such as mortality, ICU admission, and mechanical ventilation were recorded. Statistical analyses included t-tests, correlation coefficients, and p-value assessments for group comparisons and prognostic associations. **Results:** CAP patients demonstrated significantly elevated BNP at admission (84.56 ± 22.58 pg/ml) compared to controls (31.50 ± 7.12 pg/ml, $p < 0.0001$), with post-treatment reduction to 41.06 ± 9.58 pg/ml ($p < 0.001$). BNP was significantly correlated with FiO_2 requirement ($r = 0.313$, $p = 0.0149$) and mechanical ventilation ($r = 0.256$, $p = 0.0479$), while weak positive correlations with mortality and ICU admission did not reach statistical significance. Mortality occurred in 3.3% of cases, exclusively within the high BNP (>100 pg/ml) subgroup. Patients with BNP ≤47 pg/ml had an uncomplicated clinical course without adverse outcomes. Inflammatory markers (CRP, WBC, ESR) were also significantly higher in CAP patients compared to controls ($p < 0.0001$). **Conclusion:** Elevated BNP levels in CAP patients are strongly associated with respiratory severity and clinical deterioration, particularly oxygen dependency and risk of mechanical ventilation. While not an independent predictor of mortality in this sample size, BNP provides meaningful stratification of clinical risk, with low values reliably indicating favorable outcomes. BNP measurement may thus serve as a valuable adjunct for early prognostication and management decisions in CAP.

INTRODUCTION

Community-acquired pneumonia (CAP) is one of the most frequently occurring infectious diseases that could be life threatening and play a major role in the morbidity and mortality occurrence in various groups of populations (Lu et al., 2020). The World Health Organization (WHO) indicates that in 2019 globally, CAP led to the death of about 2.5 million people and was the fourth leading cause of death in lower respiratory tract infections (Huang et al., 2024). CAP has an impact on people across all age groups but the highest burden on elderly patients, people with comorbid conditions and immunocompromised patients (Malézieux-Picard et al., 2021). In spite of the (potent) antimicrobials and other supportive care, CAP remains a significant clinical challenge because of its heterogenous presentation and testing, course, and spectrum of pathogens and perspectives of infection (Ali et al., 2021). Consequently, the ability to confirm the severity and outcomes of CAP early on in the clinical course by identifying reliable prognostic markers remains a research vocal point (Yang et al., 2021).

The prevalence of CAP is especially shocking in low and middle-income countries (LMICs), such as Pakistan, in which structural health system issues, delays in access to care, and poor vaccination rates compound the severity and outcomes of the disease (Adams et al., 2021). In a countrywide surveillance study in Pakistan, pneumonia ranked as the most common infectious cause of mortality in children below five years of age and the annual estimate of deaths related to lower respiratory tract infections recorded (Ullah et al., 2022). CAP is also common in adults, particularly in adults with comorbid lung or cardiovascular diseases, diabetes and malnutrition (Ali et al., 2021). Another regional risk factor is the overpopulation, air pollution, the lack of proper use of vaccines, and the prevalence of tobacco consumption. Nevertheless, little is known about systematic studies of prognostic factors in CAP in the population of Pakistani adults (Ahsan et al., 2023).

In previous years, CAP severity has been determined through composite scoring system including but not limited to, Pneumonia Severity Index (PSI) and CURB-65 which are based on clinical, laboratory, and radiologic measures (Pranata et al., 2020). Although these tools generate reasonable mortality and ICU prediction accuracy, they demand several variables, which become less sensitive in the early stages of disease. More recently an increased interest has been in the usefulness of single, rapidly measurable biomarkers that might be added to clinical judgment. Among them there are inflammatory markers, i.e., C-reactive protein (CRP), procalcitonin (PCT), or white blood cell (WBC) count that have been investigated to a great extent (Han et al., 2022). Nevertheless, they are not very specific in distinguishing severe disease and in predicting respiratory failure or death. Therefore, the outlook has been to switch to other indicators of systemic, rather than local physiological stress, of which cardiac strain is one, i.e., B-type natriuretic peptide (BNP) (Ghilencea et al., 2022).

The BNP is a neurohormone produced by the ventricular myocytes which react towards the expansion of the volume and escalated wall tension in the heart and is commonly used to diagnose and treat heart failure (Guo et al., 2020). Interestingly, increased levels of BNP have been also considered in other non-cardia diseases, such as sepsis, pulmonary embolism, and pneumonia, indicating that BNP could be released in reaction to non-cardia diseases, inflammation, and hypoxic stress (Chung et al., 2021). In CAP, increased BNP has been suggested to measure the cardiopulmonary burden that the alveolar-capillary inflammatory storm imposes which can be hypoxemia and right heart strain due to pulmonary vasoconstriction (Farhat et al., 2024). However, the exact prognostic value of BNP in CAP i.e., prognostic value in predicting mortality, respiratory failure, and requirement of an intensive care unit is not well-defined, and some discrepancies in the literature are noted (Kim et al., 2021).

A number of clinical reports have shown that patients with CAP who have high BNP levels correlate with the severity of disease as well as poor outcome. As an example, Shen et al., (2021) noticed that the levels of BNP were many times higher in patients with CAP who had to be admitted to the ICU than in those who were placed on general wards. In the same regard, BNP >100 pg/ml was linked to higher in-hospital mortality and long hospital stay (Shen et al., 2021). Li et al. performed a meta-analysis on the topic of BNP and NT-proBNP in pneumonia (2017), who concluded that raised BNP and NT-proBNP were independent predictors of mortality and adverse events in pneumonia. However, some contradicting facts are present. Other scholars claim that the increase of the BNP in CAP can be misled by subclinical heart disorder, fluid accumulation or kidney damage and consequently restricts its specificity (Wu et al., 2025). Moreover, dissimilarities exist between the studies employed in the cut-off level of BNP, inclusion criteria of patients and consideration of comorbidity, making it hard to apply the findings across studies (Takeshima et al., 2023; Zhu et al., 2021).

Since CAP in Pakistan is a burden and due to early, cost-effective risk stratification tools being needed in resource-limited settings, BNP measurement becomes an appealing alternative. BNP tests are fast, commercially widely available in most tertiary care facilities, and have a relatively low expense as compared to advanced imaging or procalcitonin testing. Notably, the BNP holds the prospect of having patients who are at risk of respiratory failure or fatality as early as they do not necessarily have overt cardiac disease, which is especially significant where the echocardiography or intensive hemodynamic observation may not be readily available. Regardless of these benefits, limited data on the prognostic value of BNP in CAP among Pakistanis exist and there is no agreement about the optimum BNP cut off levels in this regard. Thus, the present study aimed to examine clinical outcomes especially death, hypoxia, and mechanical ventilation requirement in patients with community-acquired pneumonia and a high level of BNP.

METHODOLOGY

Study Design and Setting

This was a descriptive case series study conducted over a six-month period from 25 Jan 2024 to 25 July 2024 at the Department of General Medicine, Omar Hospital & Cardiac Centre, Lahore. The aim was to evaluate the prognostic significance of B-type natriuretic peptide (BNP) levels in hospitalized patients diagnosed with community-acquired pneumonia (CAP), and to compare them with healthy individuals as controls. Ethical approval for the study was obtained from the Research Evaluation Unit of CPSP, and written informed consent was obtained from all participants prior to enrolment.

Study Population and Sampling

Sample size was 120 patients being calculated by taking 95% confidence level, 6% margin of error and taking expected frequency of mortality as (12%). Patients were divided into two groups. Total of 60 patients diagnosed with CAP, selected through consecutive non-probability consecutive sampling. Additionally, 60 age- and sex-matched healthy individuals without respiratory, cardiac, or systemic inflammatory conditions were recruited as the control group. Patients were enrolled within 24 hours of admission, and data were collected both at the time of

presentation and after clinical recovery. Patients were eligible if they were aged 18 years or above, had a radiologically confirmed diagnosis of CAP, and exhibited clinical signs consistent with pneumonia (fever, cough, dyspnea, and/or sputum production). Only those without signs of sepsis or circulatory shock were included to eliminate confounding from systemic critical illness. Exclusion criteria included pre-existing congestive heart failure, chronic kidney disease, chronic liver disease, myocardial infarction, pulmonary embolism, recent major surgery, or any active malignancy. Patients on diuretics or those with known elevated BNP for other reasons were also excluded to ensure BNP elevation was pneumonia-related.

Data Collection

At admission, detailed history and physical examination findings were recorded for each patient, including duration of symptoms, vital signs, comorbidities, and medication history. Clinical severity was assessed using oxygen saturation, respiratory rate, and need for supplemental oxygen. The duration of hospital stay and the total time to symptom resolution were documented. A standardized follow-up was conducted either at discharge or within 14 days to assess radiological and clinical recovery. Venous blood samples were drawn at two time points: within 24 hours of admission (pre-treatment) and at the time of clinical recovery (post-treatment). Serum BNP was measured using an enzyme-linked immunosorbent assay (ELISA)-based method with a reference range of <35 pg/ml. Inflammatory markers including C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and white blood cell count (WBC) were also measured using standard automated laboratory techniques. Arterial blood gas (ABG) analysis was performed to determine partial oxygen pressure (PaO_2) and arterial oxygen saturation (SaO_2) at admission. All laboratory tests were conducted in the hospital's central laboratory by personnel blinded to clinical outcomes.

Outcome Definitions

The primary outcome was in-hospital mortality, defined as death occurring during the index hospital admission due to pneumonia-related complications. Secondary outcomes included duration of illness (defined as number of days from symptom onset to complete clinical recovery), length of hospital stay (days from admission to discharge), radiological resolution rate (percentage of patients with clearing on follow-up imaging), and oxygen requirement during hospital stay (FiO_2 percentage or need for advanced respiratory support). BNP levels were stratified into low (≤47 pg/ml), intermediate (48–100 pg/ml), and high-risk (>100 pg/ml) categories based on literature and receiver operating characteristic (ROC) analysis.

Statistical Analysis

All data were analyzed using SPSS version 28. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Independent sample t-tests were used to compare means between CAP patients and controls, while paired t-tests compared pre- and post-treatment values within patients. Pearson's correlation coefficient was applied to assess the relationship between BNP levels and variables such as CRP, WBC, ESR, and PaO_2. The chi-square test was used for categorical variables such as mortality and radiological resolution. A p-value <0.05 was considered statistically significant. ROC curve analysis was conducted to determine the optimal cut-off value of BNP for predicting mortality and poor outcomes.

RESULTS

Demographic Variables

The mean age of the 60 CAP patients was 52.14 ± 10.90 years, closely comparable to the 60 healthy controls (51.96 ± 10.38 years, p = 0.9244), indicating no significant age difference. Male participants comprised 53.3% of the CAP group and 43.3% of the control group, with a male-to-female ratio of 1.14:1 in CAP and 0.76:1 in controls.

Table 1

Demographics of CAP Patients and Controls

Parameter	CAP Patients (n=60)	Controls (n=60)	p-value
Age (years)	52.14 ± 10.90	51.96 ± 10.38	0.9244
Male (%)	53.3% (n=32)	43.3% (n=26)	
Female (%)	46.7% (n=28)	56.7% (n=34)	

Clinical and Diagnostic Variables

In patients with CAP, the mean symptom duration before hospitalization was 8.19 ± 2.62 days, and the average hospital stay was 5.67 ± 1.80 days. Most patients presented with fever (95%), cough (93%), and dyspnoea (88%). The mean arterial oxygen pressure (PaO_2) was 71.54 ± 11.94 mmHg, while FiO_2 requirements averaged 37.99 ± 7.68%, reflecting moderate hypoxia and oxygen support needs.

Table 2

Clinical and Diagnostic Parameters in CAP Patients (n=60)

Variable	Mean ± SD or % (n)
Symptom Duration (days)	8 ± 2
Hospital Stay (days)	5 ± 1
Fever	95% (n=57)
Cough	93% (n=56)
Dyspnoea	88% (n=53)
PaO_2 (mmHg)	71.54 ± 11.94
FiO_2 (%)	37.99 ± 7.68

Laboratory Variables

Significant differences were observed in biomarker profiles between the two groups. CAP patients had markedly elevated levels of BNP (84.56 ± 22.58 pg/ml) versus controls (31.50 ± 7.12 pg/ml, $p < 0.0001$). Similarly, CRP, WBC, and ESR were significantly higher in patients than in healthy controls ($p < 0.0001$ for all), reflecting inflammatory burden. After treatment, BNP levels in patients decreased to 41.06 ± 9.58 pg/ml, supporting its utility in tracking clinical improvement.

Table 3

Biomarker Comparison Between Groups

Biomarker	CAP Patients	Controls	p-value
BNP (pg/ml)	84.56 ± 22.58	31.50 ± 7.12	<0.0001
CRP (mg/L)	62.85 ± 15.55	5.5 ± 1.5	<0.0001
WBC (10^9/L)	11.95 ± 3.13	6.5 ± 1.2	<0.0001
ESR (mm/hr)	45.38 ± 7.62	12 ± 3	<0.0001

Recovery and Mortality Outcomes

The mortality rate was 3.3% (n=2). ICU admission was required in 10%, mechanical ventilation in 7%, and

complications occurred in 20%. The mean time to recovery was 6.5 ± 1.4 days, and readmissions within 30 days occurred in 5%. These outcome patterns help stratify severity levels and healthcare resource usage.

Table 4

Outcomes Among CAP Patients (n=60)

Recovery Outcome	Frequency / Value
In-hospital Mortality	3.3% (n=2)
ICU Admission	10% (n=6)
Mechanical Ventilation	7% (n=4)
Complications	20% (n=12)
Time to Recovery (days)	6.5 ± 1.4
Readmission Within 30 Days	5% (n=3)

Correlation Outcomes

Correlation analysis was performed between BNP at admission and major outcome indicators. BNP was positively and significantly correlated with FiO_2 requirements (r = 0.313, p = 0.0149), suggesting that higher BNP reflects increased oxygen needs. BNP was significantly associated with mechanical ventilation (r = 0.256, p = 0.0479), indicating a link with disease severity. BNP was weakly correlated with ICU admission (r = 0.185) and mortality (r = 0.204), though not statistically significant at $p < 0.05$. These results demonstrate that BNP levels are significantly elevated in CAP and correlate with oxygen dependency and respiratory severity, especially in those requiring mechanical ventilation. While not an absolute predictor of mortality, BNP is a valuable adjunct to traditional inflammatory markers like CRP and WBC, helping clinicians identify high-risk patients early, especially those likely to require ICU-level care or ventilatory support.

Table 5

Pearson Correlation Between BNP and Clinical Outcomes (n = 60)

Outcome Variables	Correlation (r)	*p*-value
FiO_2 Requirement	0.313	0.0149
Mechanical Ventilation	0.256	0.0479
Mortality	0.204	0.118
ICU Admission	0.185	0.1577
Complications	0.103	0.4348
Time to Recovery (days)	0.040	0.7624
Hospital Stay (days)	0.063	0.6312

DISCUSSION

The primary aim of this study was to evaluate the utility of B-type natriuretic peptide (BNP) as a prognostic biomarker in patients with community-acquired pneumonia (CAP), with particular emphasis on its association with mortality, hypoxia, and treatment response. By comparing 60 CAP patients with 60 healthy controls, the investigation explored how BNP levels correlate with inflammation markers, clinical severity (including oxygen requirement and hospitalization), and ultimate patient outcomes such as mortality, ICU admission, and mechanical ventilation. The study findings showed that BNP levels were significantly elevated in CAP patients at admission (84.56 ± 22.58 pg/ml) compared to controls (31.50 ± 7.12 pg/ml, p < 0.0001), and that they decreased significantly following recovery (41.06 ± 9.58 pg/ml, p < 0.001), thereby affirming the hypothesis that BNP is a dynamic marker reflecting disease burden and treatment progress.

In line with earlier studies, these findings substantiate the argument that BNP elevation in CAP is not merely cardiac in origin but also reflects systemic inflammation and pulmonary stress. For instance, studies by Polovina et al., (2023) and Dumanli and Karadağ (2025) both reported elevated BNP in patients with severe lower respiratory tract infections, where BNP levels showed significant correlation with CRP and IL-6 (Polovina et al., 2023; Dumanli and Karadağ, 2025). In our study, BNP levels showed a weak but positive correlation with CRP (r = 0.133), suggesting overlapping inflammatory pathways. Although this correlation was not statistically strong, it parallels the findings by Seo et al., (2020), who noted that BNP could rise due to cytokine-mediated myocardial stress even in patients without primary heart disease. Moreover, the significant reduction in BNP after treatment aligns with the results of Lu et al., (2020), who reported a post-treatment fall in BNP that correlated with radiologic resolution and symptom improvement (Seo et al., 2020; Lu et al., 2020).

An important novel finding of the present study is the statistically significant correlation between BNP and FiO_2 requirements (r = 0.313, p = 0.0149), underscoring BNP's role in reflecting hypoxic burden. This reinforces earlier hypotheses proposed by Choi et al., (2020), who argued that increased right ventricular afterload from pulmonary vasoconstriction in pneumonia may lead to cardiac stretch and BNP release (Choi et al., 2020). Moreover, the BNP levels were moderately correlated with the need for mechanical ventilation (r = 0.256, p = 0.0479), suggesting that BNP may serve as an early warning marker of respiratory decompensation. This observation is consistent with the findings of Tazón-Varela et al., (2022), who demonstrated that patients with BNP >100 pg/ml were more likely to experience respiratory failure requiring ventilatory support. In our study, both in-hospital deaths (3.3%) occurred in the high-BNP subgroup (>100 pg/ml), further supporting its prognostic potential (Tazón-Varela et al., 2022).

However, not all outcome correlations with BNP were statistically significant. Although mortality, ICU admission, and complications showed weak positive correlations with BNP (r = 0.204, 0.185, and 0.103 respectively), their p-values exceeded the 0.05 threshold. These non-significant results might be attributed to the relatively low number of adverse outcomes (e.g., only 2 deaths, 6 ICU admissions), limiting statistical power. Similar limitations have been reported in other studies with modest sample sizes, such as in the work of Liu et al., (2023), where BNP levels were associated with disease severity scores but did not achieve significance for hard endpoints due to small event rates. Nevertheless, the directional trends in our data support a hypothesis of BNP as a marker for poorer prognosis, warranting confirmation in larger multi-center trials (Liu et al., 2023). A critical strength of this study lies in its clear demonstration of BNP's decline post-treatment (from 84.56 to 41.06 pg/ml), positioning it as a useful marker for monitoring therapeutic response. This dynamic behavior of BNP was echoed in research by Huang et al., (2021), who reported that serial measurement of pro-BNP provided

superior prognostic information compared to single time-point readings (Huang et al., 2021).

Our results further add to this body of evidence by showing that those with BNP ≤47 pg/ml had no mortality or major complications, suggesting that a low BNP at presentation could help stratify patients for outpatient care or early discharge, reducing healthcare costs (Handargal and Usman, 2024; Lee et al., 2021). Despite these strengths, the study has several limitations. First, while BNP was statistically associated with several indicators of severity, causality cannot be inferred due to the observational design. Second, the exclusion of patients with sepsis or pre-existing cardiac disease, though methodologically appropriate, may limit generalizability. Third, the study did not include NT-proBNP, which some guidelines consider superior to BNP in terms of diagnostic stability. Nonetheless, given that BNP assays are widely available and cost-effective, the findings retain strong clinical relevance, especially in low-resource settings.

CONCLUSION

This study critically explored the outcomes in terms of mortality and disease severity in patients with CAP presenting with elevated BNP levels. The findings revealed that patients with raised BNP not only had significantly higher levels than healthy controls at baseline but also demonstrated a meaningful decline following treatment, indicating its utility as a dynamic biomarker of disease progression and recovery. While the in-hospital mortality rate was modest (3.3%), both deaths occurred in the subgroup with BNP levels exceeding 100 pg/ml, reinforcing BNP's potential as a mortality risk indicator. Moreover, statistically significant correlations between BNP and respiratory parameters—particularly FiO_2 requirements and need for mechanical ventilation—highlight its role in identifying patients at risk of respiratory compromise. Although correlations with ICU admission and mortality did not reach statistical significance, their consistent positive direction suggests clinical relevance, particularly in larger cohorts. Importantly, patients with BNP levels ≤47 pg/ml experienced no major adverse outcomes, supporting the threshold's value in stratifying low-risk individuals. Future studies should aim to include larger cohorts and stratify by pneumonia severity scores (e.g., CURB-65) to determine BNP's role in validated risk models.

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