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Original Research Article

LUNG ULTRASONOGRAPHY AND PRO BNP IN THE EVALUATION OF ACUTE DYSPNOEA AND THEIR RELATIONSHIP WITH ECHOCARDIOGRAPHIC FINDINGS

Ganesh Bhagwanrao Sonvane[1], Amit Vishwanath Patil[2], Mahesh Manikrao Shinde[3]

[1]*Assistant Professor, Department of General Medicine, MGM Medical College, Chh. Sambhajinagar, India.*
[2]*Associate Professor, Department of General Medicine, R.K. Damani Medical College, Shri Ramchandra Institute of Medical Sciences, Chh. Sambhajinagar, India.*
[3]*Assistant Professor, Department of General Medicine, MGM Medical College & Hospital, Chh.Sambhajinagar, India.*

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Corresponding Author:
Dr. Ganesh Bhagwanrao Sonvane, Assistant Professor, Department of General Medicine, MGM Medical College, Chh. Sambhajinagar, India
Email: Ganesh4umy@gmail.com

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ABSTRACT

Background: Differentiating cardiogenic from non-cardiogenic acute dyspnoea is a frequent diagnostic challenge. **Objective:** To evaluate the diagnostic performance of lung ultrasonography and Pro-BNP and their relationship with echocardiographic findings in adults with acute dyspnoea. **Materials and Methods:** This hospital-based cross-sectional study included 100 adults. Clinical assessment, lung ultrasonography, Pro-BNP measurement and two-dimensional echocardiography were performed. A cardiologist and an internist assigned the final diagnosis using clinical, laboratory, imaging and treatment-response findings. Sensitivity, specificity, predictive values and area under the receiver operating characteristic curve (AUROC) were assessed.
Results: Cardiogenic dyspnoea occurred in 67 patients and non-cardiogenic dyspnoea in 33. Lung ultrasound identified the cause in 92.5% of cardiogenic and 69.7% of non-cardiogenic cases (p=0.003). Lung ultrasound had sensitivity of 94%, specificity of 78%, positive predictive value of 96%, negative predictive value of 64% and AUROC of 0.91. Corresponding values for Pro-BNP were 86%, 83%, 90%, 74% and 0.87. Mean Pro-BNP was higher in cardiogenic than non-cardiogenic dyspnoea (11,921 ± 9,321 versus 316.97 ± 104.95; p<0.0001). Ultrasound positivity was associated with echocardiographic abnormality (p=0.017).
Conclusion: Lung ultrasound showed higher sensitivity and AUROC, while Pro-BNP showed higher specificity and negative predictive value. Both modalities may provide complementary information alongside clinical assessment and echocardiography in acute dyspnoea.
Keywords: Acute dyspnoea; lung ultrasonography; Pro-BNP; natriuretic peptides; echocardiography; cardiogenic dyspnoea.

INTRODUCTION

Acute dyspnoea presents a diagnostic challenge because cardiac, pulmonary and other disorders can produce overlapping symptoms and signs. Clinical examination helps narrow the differential diagnosis, but individual findings do not consistently discriminate between causes.[1] The limitations of examination are particularly relevant to congestion: Stevenson and Perloff showed that conventional physical signs may be absent despite elevated ventricular filling pressure in patients with chronic heart failure.[2] These observations support an assessment that combines the clinical presentation with objective investigations rather than relying on one symptom or sign.

Accurate recognition of acute heart failure requires evaluation of both the clinical syndrome and the underlying cardiac abnormality. The European Society of Cardiology acute heart failure guidance available before the study period described a diagnostic approach incorporating clinical

assessment, electrocardiography, chest imaging, laboratory testing and echocardiography.[3] This framework is relevant when a patient with breathlessness has more than one possible cardiopulmonary diagnosis.

Natriuretic peptides participate in cardiovascular homeostasis through natriuretic, diuretic and vasorelaxant actions.[4] Yasue et al demonstrated that BNP is predominantly secreted from the left ventricle and that ventricular secretion increases with the severity of left ventricular dysfunction.[5] Consequently, measurement of circulating natriuretic peptides can provide biochemical information about cardiac stress that complements examination and imaging.

Early clinical evidence came from Davis et al, who studied elderly patients with acute dyspnoea and found that plasma BNP helped distinguish heart failure from primary pulmonary disorders.[6] The Breathing Not Properly Multinational Study subsequently demonstrated the diagnostic usefulness of rapid BNP measurement in emergency department patients with dyspnoea.[7] The PRIDE study established the usefulness of NT-proBNP for identifying and excluding acute congestive heart failure in the emergency setting.[8] McCullough et al further showed that BNP testing adds diagnostic information to clinical judgement.[9]

Lung ultrasound offers a bedside assessment of pulmonary and pleural findings. Lichtenstein and colleagues described comet-tail artefacts as a sonographic sign of alveolar-interstitial syndrome.[10] Jambrik et al evaluated ultrasound lung comets as a nonradiological indicator of extravascular lung water.[11] Agricola et al compared these images with pulmonary wedge pressure and independently measured extravascular lung water, providing further physiological support for their use in assessing pulmonary oedema.[12]

The BLUE protocol extended lung ultrasound interpretation to a systematic assessment of common causes of acute respiratory failure.[13] International consensus recommendations subsequently defined evidence-based applications of point-of-care lung ultrasound in emergency and critical care.[14] Practical guidance by Gargani and Volpicelli emphasizes the recognition of lung and pleural patterns and their interpretation within the clinical context.[15] These approaches support structured scanning, while also requiring clear criteria for what constitutes a positive examination.

Lung ultrasound, natriuretic peptide measurement and echocardiography provide different observations within the same diagnostic assessment. This study aimed to evaluate lung ultrasonography and Pro-BNP in adults with acute dyspnoea and examine their relationships with the cause of dyspnoea and two-dimensional echocardiographic findings.

MATERIALS AND METHODS

Study Design and Setting

This hospital-based cross-sectional study was conducted in the Department of Medicine of Government Medical College Nagpur from a period of November 2015 to August 2017.

Inclusion Criteria

1. Age >18 yrs.
2. Patients presenting with acute dyspnoea – NYHA 2-4
3. Informed consent

Exclusion Criteria

1. Patient not willing for consent
2. CPR less than 7 days
3. Cardiogenic shock
4. Dyspnoea after chest trauma
5. Advanced neoplasia
6. Chronic kidney disease requiring hemodialysis

Investigations

- ProBNP
- ECG
- ECHO
- CBC
- LFT
- KFT
- Chest X-Ray
- Lung Ultrasound

Data Collection

Patients were enrolled with convenience sampling method and after informed consent. On admission, after history taking and physical examination, 2D echocardiogram and a bedside ultrasound with 5.2 probe were performed, and a blood sample was drawn for laboratory examinations (including BNP levels) during the first 15 minutes of admission. The majority of patients were in sitting or supine position in accordance with their comfort position. For performing ultrasonography, each hemi thorax was divided into 2 anterior (upper and lower) and 2 lateral zones. Anterior zone was the area between sternum and anterior axillary line while lateral zone was between anterior and posterior axillary lines. These zones were divided into upper and lower zones by the third intercostal space as a border. Lungs were scanned for presence of pleural sliding, B-Lines and A-Lines. Pleural sliding is the back-and-forth movement of visceral and parietal pleura. B-Lines are vertical pleural-based discrete, laser-like, hyper-echoic reverberation artifacts. They move with respiration simultaneously and extend to the bottom of the screen without fading. A-Lines are horizontal hyperechoic reverberation lung artifacts that are separated from each other by an equal distance. For differentiating heart failure from pulmonary causes of acute respiratory distress, inferior vena cava (IVC) collapsibility index was measured. Final diagnosis of patients was decided based on clinical, laboratory, imaging and response to treatment findings by an expert cardiologist together with an internist.

Statistical Analysis

Data analysis was performed using SPSS software, version 26. Categorical variables were expressed as frequency and percentage and continuous variables as mean and standard deviation. Sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) of bedside ultrasound compared to BNP in differentiating cardiogenic causes of acute dyspnea were calculated. In addition, area under the receiver operating characteristic (ROC) curve was calculated for the two diagnostic tests. P-value < 0.05 was considered statistically significant.

Ethics

Institutional ethics committee clearance and permission from the appropriate authority were obtained before the study.

RESULTS

Clinical profile and cause of dyspnoea

There were 100 participants, including 53 males and 47 females. The largest age category was 61–70 years (31%). All presented with breathlessness; chest pain occurred in 28%, cough in 24% and fever in 23%. Cardiogenic dyspnoea was diagnosed in 67 patients and non-cardiogenic dyspnoea in 33. Participant characteristics and presenting symptoms are summarized in Table 1.

Table 1: Participant characteristics and presentation

Characteristic	Category	n (%)
Age in years	Up to 20	2 (2.0)
	21–30	10 (10.0)
	31–40	9 (9.0)
	41–50	18 (18.0)
	51–60	15 (15.0)
	61–70	31 (31.0)
	Over 70	15 (15.0)
Gender	Male	53 (53.0)
	Female	47 (47.0)
Presenting symptoms	Breathlessness	100 (100.0)
	Chest pain	28 (28.0)
	Cough	24 (24.0)
	Fever	23 (23.0)
	Swelling	8 (8.0)
	Palpitations	5 (5.0)
Cause of dyspnoea	Cardiogenic	67 (67.0)
	Non-cardiogenic	33 (33.0)

Denominator is 100. Symptoms are multiple responses. The youngest category contains eligible adults aged over 18 years.

Lung ultrasound findings

Lung ultrasonography identified the cause of dyspnoea in 62 of 67 cardiogenic cases (92.5%) and 23 of 33 non-cardiogenic cases (69.7%). The cause was not identified in five cardiogenic and ten non-cardiogenic cases. Overall diagnostic yield was 85%. Diagnostic yield was significantly higher in cardiogenic than non-cardiogenic dyspnoea (χ^2=9.047, df=1; p=0.003; Table 2).

Among the 33 patients with non-cardiogenic dyspnoea, A-lines were present in ten (30.3%), a shred sign in 20 (60.6%) and a stratosphere sign in three (9.1%).

Table 2: Lung ultrasound diagnostic yield by cause of dyspnoea

Aetiological group	Recorded as diagnosed	Not diagnosed	Total
Cardiogenic	62 (92.5%)	5 (7.5%)	67
Non-cardiogenic	23 (69.7%)	10 (30.3%)	33
Total	85 (85.0%)	15 (15.0%)	100

Values are n (row %). Pearson χ^2=9.047; df=1; p=0.003.

Echocardiographic findings

Ejection fraction below 50% was present in 25 cardiogenic patients (37.3%) and 13 non-cardiogenic patients (39.4%). Hypokinesia, left ventricular hypertrophy and valvular disorders were present in 20, 19 and 16 cardiogenic cases, respectively; none was recorded in the non-cardiogenic group. Echocardiograms were within normal limits in 20 cardiogenic and 12 non-cardiogenic cases. [Table 3]

Table 3: Echocardiographic findings by aetiological group

Finding	Cardiogenic n=67	Non-cardiogenic n=33
Ejection fraction <50%	25 (37.3%)	13 (39.4%)
Hypokinesia	20 (29.9%)	0 (0.0%)
Left ventricular hypertrophy	19 (28.4%)	0 (0.0%)
Valvular disorder	16 (23.9%)	0 (0.0%)
Within normal limits	20 (29.9%)	12 (36.4%)

Values are n (% within each aetiological group). Echocardiographic findings are multiple responses.

Pro BNP and imaging relationships

Mean Pro-BNP was 11,921 ± 9,321 in patients with cardiogenic dyspnoea and 316.97 ± 104.95 in those with non-cardiogenic dyspnoea (p<0.0001). Mean values were also higher in patients with abnormal echocardiograms than in those with normal echocardiograms (12,812 ± 7,281 versus 288.92 ± 100.12; p<0.0001). Patients with abnormal lung ultrasound findings had higher mean Pro-BNP values than those with normal findings (11,516 ± 6,321 versus 269.18 ± 98.67; p<0.0001;.[Table 4]

Table 4: Pro-BNP values by cause of dyspnoea and imaging findings

Grouping variable	Category	Mean ± SD	p value
Cause of dyspnoea	Cardiogenic	11,921 ± 9,321	<0.0001
	Non-cardiogenic	316.97 ± 104.95	
Echocardiography	Normal	288.92 ± 100.12	<0.0001
	Abnormal	12,812 ± 7,281	
Lung ultrasound	Normal	269.18 ± 98.67	<0.0001
	Abnormal	11,516 ± 6,321	

Values are mean ± standard deviation. Biomarker measurement units: [confirm].

There were 68 abnormal and 32 normal echocardiograms. Lung ultrasound was positive in 62/68 patients with abnormal echocardiograms (91.2%) and 23/32 patients with normal echocardiograms (71.9%); it was negative in six (8.8%) and nine (28.1%) patients, respectively. Overall, 85 examinations were ultrasound-positive and 15 were negative. Ultrasound positivity was significantly associated with echocardiographic abnormality (Fisher exact p=0.017).

Diagnostic performance

Lung ultrasonography had sensitivity of 94%, specificity of 78%, PPV of 96%, NPV of 64% and AUROC of 0.91. Pro-BNP had sensitivity of 86%, specificity of 83%, PPV of 90%, NPV of 74% and AUROC of 0.87 (Table 5). Lung ultrasound showed numerically higher sensitivity, PPV and AUROC, whereas Pro-BNP showed higher specificity and NPV.

Table 5: Diagnostic performance of lung ultrasonography and Pro-BNP

Measure	Lung ultrasonography	Pro-BNP
Sensitivity	94%	86%
Specificity	78%	83%
PPV	96%	90%
NPV	64%	74%
AUROC	0.91	0.87

PPV, positive predictive value; NPV, negative predictive value; AUROC, area under the receiver operating characteristic curve.

DISCUSSION

This study evaluated lung ultrasonography, Pro-BNP and echocardiographic findings in 100 adults with acute dyspnoea. Cardiogenic dyspnoea accounted for 67% of cases. Lung ultrasound showed sensitivity of 94%, specificity of 78% and AUROC of 0.91, compared with 86%, 83% and 0.87 for Pro-BNP. Lung ultrasound also had a higher diagnostic yield in cardiogenic than non-cardiogenic dyspnoea. Pro-BNP values were higher in patients with cardiogenic dyspnoea and abnormal imaging findings, while ultrasound positivity was associated with echocardiographic abnormality. The different diagnostic profiles support considering both modalities alongside clinical assessment and echocardiography.

The predominance of cardiogenic dyspnoea in this cohort is comparable with the hospital-based study by Gargani et al, in which 122 of 149 patients with acute dyspnoea had a cardiogenic cause.[16] Differences in the proportions across studies may reflect eligibility criteria and the clinical setting. In the present study, recruitment through emergency and intensive care services and exclusion of patients with shock or dialysis-dependent kidney disease may have influenced the distribution of diagnoses. The observed proportion therefore describes the study population rather than the community incidence of cardiogenic dyspnoea.

Lung ultrasound had sensitivity of 94% and specificity of 78% in this study. In a prehospital study of 218 patients, Prosen et al evaluated heart failure against COPD or asthma and reported lung ultrasound sensitivity of 100% and specificity of 95%.[17] The higher specificity in their study may reflect differences in the patient population, diagnostic spectrum and ultrasound criteria. Our diagnostic yield of 92.5% in cardiogenic and 69.7% in non-cardiogenic cases describes the proportion in whom ultrasound identified the cause of dyspnoea and is distinct from sensitivity and specificity.

The AUROC was numerically higher for lung ultrasound than for Pro-BNP (0.91 versus 0.87), indicating good discrimination by both tests. Golshani et al similarly reported higher ROC areas for bedside ultrasonography than for BNP in acute dyspnoea (0.864 versus 0.663).[18] In a separate pilot study, Glöckner and colleagues compared point-of-care B-line ultrasound with NT-proBNP for

screening acute heart failure.[19] Differences in assays, sonographic criteria and reference diagnoses must be considered when comparing studies. The numerical AUROC difference in our study does not establish statistically significant superiority because no formal comparison of the two ROC areas is presented.

The non-cardiogenic group showed A-lines, shred signs and stratosphere signs, illustrating the range of pulmonary and pleural patterns encountered in acute dyspnoea. Consensus recommendations and practical ultrasound guidance emphasize interpretation of these signs within the whole clinical examination.[14,15] The sonographic pattern must therefore be considered together with history, examination and other investigations; an individual sign alone does not establish the underlying disease.

Pro-BNP values were substantially higher in cardiogenic than non-cardiogenic dyspnoea. This finding is consistent with the clinical diagnostic literature on BNP and NT-proBNP, including the studies by Davis et al, the Breathing Not Properly investigators and the PRIDE investigators.[6,7,8] The wide dispersion in the cardiogenic group indicates substantial variation between patients. A difference in mean values supports a group-level association but does not by itself establish an individual diagnostic threshold or superiority over lung ultrasound.

Lung ultrasound had a PPV of 96% and NPV of 64%, compared with 90% and 74% for Pro-BNP. The higher sensitivity of ultrasound was accompanied by lower specificity, while Pro-BNP showed the reverse pattern. Neither test had a sufficiently high NPV to exclude a cardiogenic cause on its own in this cohort. Predictive values also depend on disease prevalence and their interpretation should account for the 67% proportion of cardiogenic dyspnoea in the study population.

The higher Pro-BNP values in patients with abnormal lung ultrasound findings are biologically plausible because both measurements may reflect consequences of cardiac dysfunction. Gargani et al found a correlation between ultrasound lung comet count and NT-proBNP ($r=0.69$; $p<0.0001$), with ROC areas of 0.893 for ultrasound lung comets and 0.978 for NT-proBNP.[16] Our analysis compared biomarker values across imaging categories and did not quantify the continuous relationship between B-line burden and Pro-BNP. The findings are therefore consistent with a complementary assessment, without establishing the strength of a continuous correlation.

Lung ultrasound positivity was associated with echocardiographic abnormality ($p=0.017$). Frassi et al similarly reported an association between ultrasound lung comets and left ventricular diastolic and/or systolic dysfunction in patients undergoing comprehensive echocardiographic assessment.[20] These observations provide a physiological basis for concordance between pulmonary and cardiac imaging. Nevertheless, lung ultrasound assesses pulmonary findings while echocardiography evaluates cardiac structure and function; an association between their findings does not establish interchangeability.

Reduced ejection fraction occurred in both aetiological groups, while some patients with cardiogenic dyspnoea had echocardiograms classified within normal limits. This pattern emphasizes the distinction between a cardiac imaging finding and the cause of the current episode of breathlessness. The consensus statement by Paulus et al describes heart failure with normal left ventricular ejection fraction as requiring symptoms or signs and evidence of diastolic dysfunction.[21] Consequently, ejection fraction alone is insufficient for aetiological classification and echocardiographic findings should be interpreted within the complete clinical assessment.

Renal function is another consideration when interpreting natriuretic peptide values. Chenevier-Gobeaux et al specifically examined the influence of renal function on BNP and NT-proBNP in emergency department patients presenting with dyspnoea.[22] Excluding dialysis-dependent chronic kidney disease does not establish that all remaining participants had normal renal function. The present data therefore do not support attributing every elevated biomarker value exclusively to the assigned cause of dyspnoea and no adjustment for renal function has been performed.

Diagnostic usefulness and improvement in clinical management are separate questions. Mueller et al studied 452 patients with acute dyspnoea in a randomized trial and found that a strategy incorporating rapid BNP testing reduced time to discharge and treatment costs.[23] In the 500-patient IMPROVE-CHF trial, Moe et al found that adding NT-proBNP results to clinical assessment improved diagnostic accuracy and selected management outcomes.[24] These findings support further evaluation of clinically integrated biomarker testing. The present cross-sectional study, however, did not measure treatment time, cost, readmission or mortality and cannot establish those benefits.

No combined ultrasound–biomarker decision rule was evaluated in this study. The association of both tests with cardiogenic dyspnoea does not establish that their combination improves diagnostic accuracy. Studies using prespecified thresholds, a consistent reference diagnosis and paired patient-level measurements are needed to determine the incremental value of combined testing.

Strengths and limitations

A strength of this study is the assessment of clinical findings, lung ultrasonography, Pro-BNP and echocardiography in the same cohort of patients with acute dyspnoea. The single-centre design and sample size of 100, including only 33 non-cardiogenic cases, limit generalizability. Because clinical adjudication incorporated laboratory and imaging findings, bias cannot be excluded. Interobserver agreement was not assessed in the analysis.

Although diagnostic performance was assessed for each modality, continuous relationships between Pro-

BNP and B-line burden, adjusted models and the performance of a combined diagnostic rule were not assessed. Renal function was not included in an adjusted analysis, although dialysis-dependent chronic kidney disease was excluded. Treatment outcomes, readmission, mortality and prognostic endpoints were not assessed. The small single-centre sample limits the precision and generalizability of the accuracy estimates. These limitations restrict conclusions about the incremental value of combined testing and its effect on patient outcomes.

CONCLUSION

Lung ultrasonography and Pro-BNP demonstrated useful diagnostic performance in acute dyspnoea. Lung ultrasound had higher sensitivity, positive predictive value and AUROC, whereas Pro-BNP had higher specificity and negative predictive value. Pro-BNP values were higher in cardiogenic dyspnoea and in patients with abnormal imaging findings and ultrasound positivity was associated with echocardiographic abnormality. Both modalities may contribute complementary information to clinical assessment and echocardiography. Further studies are needed to establish the incremental diagnostic value of combined testing.

Declarations

Ethics approval and consent: Institutional ethics committee approval was obtained and all participants provided written informed consent.

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Conflicts of interest: None.

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