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Chest Heart Journal
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EDITORIAL

Artificial Intelligence in the Radiological Diagnosis of Pulmonary Tuberculosis: Current Trends and Future Perspectives

Mohammed Mirazur Rahman

[Chest Heart J. 2025; 49(1) : 1-2]

Tuberculosis (TB) is an infectious disease caused by Mycobacterium tuberculosis. Although the diagnostic technology of pulmonary tuberculosis (PTB) has advanced, accurate diagnosis and excluding differentials are still challenging. In recent years, the rapid development of artifi-cial intelligence (AI) and its wide application in the medical fi-eld have provided new opportunities for diagnosing and treating TB and PTB. The machine learning model of AI has not only helped physicians improve diagnostic accuracy, but also enabled them to make early preventive strategies for individuals at increased risk of infection. Furthermore, AI can guide physicians to formulate targeted treatment strategies for PTB patients with different conditions.

Based on recent studies and meta-analyses, artificial intelligence (AI) has established a significant, high-performance role in the radiological diagnosis of pulmonary tuberculosis (PTB), particularly in screening and triage using chest X-rays (CXR) and computed tomography (CT) scans. AI tools, specifically deep learning-based computer-aided detection (CAD) systems, are now recommended by the World Health Organization (WHO) as an alternative to human readers for screening and triaging PTB in adults.

Key findings on the role of AI in PTB diagnosis

High diagnostic accuracy: AI systems show excellent performance, with studies reporting pooled sensitivity and specificity as high as 91–94% and 65–95% respectively[1].

Superior to human readers in screening: In high-burden areas, AI-powered CAD has demonstrated a significantly higher diagnostic yield (83.9%) compared to standard radiologist interpretation (25.6%)[2].This makes them invaluable for reducing missed diagnoses, especially in resource-limited settings with a shortage of qualified radiologists.

AI-assisted chest X-ray (CXR) imaging:

A lot of mature AI software has been applied to TB diagnosis in recent years. A previous meta-analysis showed that although the sensitivity and specificity of each software may differ under certain conditions, the accuracy of physician-only and computer-aided diagnosis (CAD) software-based diagnoses was similar, and the use of CAD software significantly reduced the variability caused by different experiences and work fatigue. Developing an AI-based automated screening platform in remote and resource-poor regions can significantly reduce the waiting time for patients to receive diagnostic reports, improve the efficiency of hospital outpatient clinics, and increase the accuracy of disease diagnosis. Research found that the sensitivity, specificity, positive predictive value, negative predictive value (NPV), and diagnostic accuracy of the AI-assisted system were higher than those of the physician alone[3]. In summary, AI-assisted CXR diagnosis has potential advantages in the rapid localization and quantification of PTB lesions, which can not only accurately identify microscopic lesions often missed by the human eye, but also assist in the diagnosis and improve the detection rate of PTB.

AI-assisted CT imaging diagnosis:

CT is superior to CXR in identifying parenchymal lesions and showing active features of PTB (e.g., cavities, parenchymal abnormalities, and lobular central nodules). In recent years, relevant AI

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systems have been developed to assist in reading chest CT images to help physicians improve the accuracy and sensitivity of CT diagnosis of PTB[4]. In addition, it has been suggested that AI can automatically identify pulmonary abnormalities 1000 times more efficiently than doctors.

Challenges and Future Directions

- Limited pediatric validation: AI tools are not yet recommended by the WHO for children and adolescents, as their performance is less robust in this demographic.
- Generalizability and data heterogeneity: Performance can vary based on the prevalence of the disease and the quality of the imaging equipment, highlighting the need for on-site calibration and validation.
- False positives: While AI is highly sensitive, its specificity can sometimes be lower than that of human experts, potentially increasing the need for unnecessary follow-up procedures if not used properly.
- Need for multimodal AI: Future research is focused on integrating AI with clinical symptoms and lab data to improve accuracy further.

Conclusions

AI is important in diagnosing PTB and can improve the diagnostic accuracy, sensitivity, and specificity of CXR- and CT-based tests. CAD dramatically improves the detection of PTB among symptomatic patients presenting to primary health care facilities. Meanwhile, CAD demonstrates significantly superior performance in township medical facilities compared to county medical facilities, providing validated evidence for implementing tiered diagnosis and treatment models in resource-limited regions.

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ORIGINAL ARTICLE

Association of Blood Urea Nitrogen to Serum Albumin Ratio with the Need for Non-invasive Ventilation in Patients with Exacerbation of Chronic Obstructive Pulmonary Disease

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Abstracts

Background: *Chronic Obstructive Pulmonary Disease (COPD) is a leading cause of chronic morbidity and mortality, with frequent exacerbations that can lead to acute respiratory failure. Non-invasive ventilation (NIV) is a key treatment for acute respiratory failure in COPD exacerbations. The Blood Urea Nitrogen (BUN) to serum albumin ratio has been proposed as a prognostic marker in various diseases, reflecting metabolic stress and systemic inflammation. Still, its association with the need for NIV in COPD exacerbations has not been widely studied.*

Objective: *This study aims to evaluate the association between the BUN/Albumin ratio and the need for NIV in patients with exacerbations of COPD.*

Methods: *This cross-sectional study was conducted in the inpatient Department of Respiratory Medicine at the National Institute of Diseases of the Chest and Hospital (NIDCH), Dhaka, Bangladesh, from June 2023 to December 2024. A total of 116 patients diagnosed with exacerbations of COPD were enrolled. The BUN/Albumin ratio was measured for each patient, and the need for NIV was assessed. Data were analysed to determine if there was a significant association between the BUN/Albumin ratio and the requirement for NIV.*

Results: *The mean BUN/Albumin ratio was significantly higher in patients who required NIV (9.1±2.8) compared to those who did not (5.3±1.8, p = 0.001). A progressive increase in the BUN/Albumin ratio was observed with worsening severity of COPD exacerbations, with severe cases showing the highest ratios (9.5±2.9). Multivariate regression analysis showed that the BUN/Albumin ratio was significantly associated with the need for NIV (adjusted OR 19.629, 95% CI 13.976-27.561, p = 0.001).*

Conclusion: *The BUN/Albumin ratio is significantly associated with the need for NIV in patients with exacerbations of COPD. Elevated BUN/Albumin ratios may serve as a useful biomarker for identifying patients at higher risk for respiratory failure and may help guide early intervention in clinical practice.*

Keywords: *Exacerbation of chronic obstructive pulmonary disease (ECOPD), Non-invasive ventilation (NIV).*

[Chest Heart J. 2025; 49(1) : 3-9]

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Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a leading cause of chronic morbidity and mortality globally, currently ranking among the top three causes of death worldwide. The disease burden is especially high in low- and middle-income countries, where approximately 90% of COPD-related deaths occur[1]. In Bangladesh, the prevalence of COPD is estimated to affect 12.5% of the population[2], with higher rates observed among the elderly, low-education groups, manual laborers, smokers, and those exposed to biomass fuels and indoor pollutants[3]. COPD is primarily caused by long-term exposure to harmful substances, particularly cigarette smoke, though non-smoking-related risk factors are also significant, especially in low- and middle-income regions.

COPD exacerbations (ECOPD), marked by a sudden worsening of symptoms, require medical intervention and are a major driver of hospital admissions and disease progression. Such exacerbations often lead to respiratory failure, which remains a major complication of the disease[4]. Non-invasive ventilation (NIV) is an effective treatment for acute respiratory failure in ECOPD patients, improving outcomes such as gas exchange and reducing hospital stay and mortality rates[5].

In COPD patients, both renal dysfunction and malnutrition are prevalent, with biomarkers such as Blood Urea Nitrogen (BUN) and serum albumin levels playing critical roles in assessing prognosis[6,7,8]. The BUN/Albumin ratio, which reflects both metabolic stress and inflammation, has been shown to predict the severity of diseases like community-acquired pneumonia and sepsis[9]. However, its potential as a predictive marker for NIV requirement in ECOPD patients remains unexplored.

This study aims to evaluate the association between the BUN/Albumin ratio and the need for NIV in patients with exacerbations of COPD, hypothesising that higher BUN/Albumin ratios may indicate greater severity of the exacerbation, thereby necessitating NIV. By identifying such biomarkers, the study could contribute to improving clinical decision-making in managing ECOPD exacerbations.

Objectives

General Objective:

To evaluate the association of the BUN/Albumin ratio with the need for non-invasive ventilation in patients with ECOPD.

Specific Objectives:

- To measure the BUN/Albumin ratio in patients with ECOPD.
- To assess the need for non-invasive ventilation in patients withECOPD.
- To determine an association of the BUN/Albumin ratio with the need for non-invasive ventilation in patients with ECOPD.

Materials and methods

This cross- sectional observational study was carried out in the Department of Respiratory Medicine of National Institute of Diseases of the Chest and Hospital (NIDCH), Mohakhali, Dhaka between June 2023 and December 2024.

Inclusion criteria were:

1. COPD patients admitted to NIDCH with exacerbation.

Exclusion criteria were:

1. Chronic Kidney Disease
2. Chronic Liver Disease
3. Pneumonia

Study procedure

One hundred sixteen patients admitted to NIDCH with chronic obstructive pulmonary disease (ECOPD) exacerbations were enrolled in this study based on inclusion and exclusion criteria. The study population was counselled regarding the study's aim, objectives, and usefulness. Written informed consent was obtained from each patient, and an interview was conducted using a semi-structured questionnaire. A proper history was taken, and the patient was thoroughly examined. Arterial blood (radial, brachial, or femoral) was collected as per standard protocol for ABG analysis and was kept horizontally within an icepack till analysis in a Radiometer ABL80 flex analyser. Venous blood for BUN and serum albumin was collected at the same time in an EDTA (Ethylene diamine tetra acetic acid) tube and analysed in an Automated Biochemistry Analyser Pentra-400 and checked manually. Appropriate medical management was given to all patients, following standard protocol. Finally, the BUN/Albumin ratio

was calculated, and an assessment was done to determine whether the BUN/Albumin ratio is associated with the need for NIV. It was done by multivariate binary logistic regression analysis.

Results

This cross-sectional study was conducted in the Department of Respiratory Medicine of the National Institute of Diseases of the Chest and Hospital (NIDCH), Mohakhali, Dhaka, from September 2023 to March 2025. During this period, 116 patients diagnosed with exacerbations of chronic obstructive pulmonary disease who fulfilled the inclusion and exclusion criteria were included in this study. The findings obtained from the data analysis are presented below:

Table-I

Distribution of the study population by socio-demographic characteristics (N=116)

Socio-demographic characteristics	Frequency	Percentage
Age (years)		
41-60	64	55.2
61-80	50	43.1
>80	2	1.7
Mean ±SD	59.8±9.8	
Range (min-max)	41.0-86.0	
Sex		
Male	110	94.8
Female	6	5.2
Occupational status		
Cultivator	33	28.4
Service holder	22	19.0
Businessman	21	18.1
Retired	20	17.2
Day labourer	10	8.6
Housewife	6	5.2
Others	4	3.4

Table-II

Distribution of the study population according to smoking status (N=116)

Smoking status	Frequency	Percentage
Current smoker	44	37.9
Ex-smoker	66	56.9
Never smoker	6	5.2
If smoker (n=110)		
≤20 pack year	26	23.6
>20 pack year	84	76.4
Mean±SD	25.2±6.8	

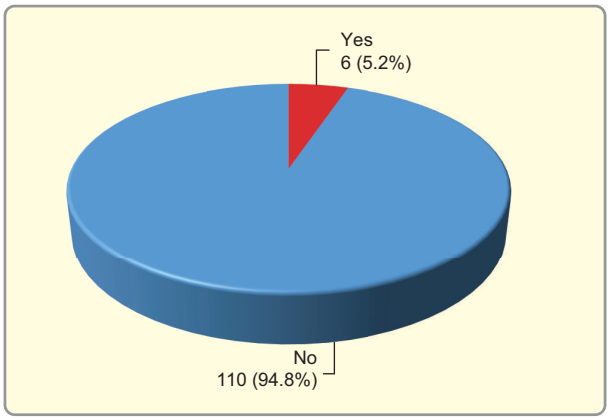

Figure 1: *Pie chart showing exposure to biomass fuel of the study population (N=116)*

Table-III

Distribution of the study population according to co-morbidities (N=116)

Co-morbidities	Frequency	Percentage
Hypertension	30	25.9
Diabetes mellitus	22	19.0
Ischemic heart disease	19	16.4
No co-morbidities	45	38.8

Table -IV

Distribution of the study population according to investigations (N=116)

Investigations	Mean±SD	
Blood urea nitrogen (mg/dl)	23.2±8.8	
Serum albumin (g/dl)	3.5±0.5	
BUN/Albumin ratio	6.9±3.0	
CRP		
<10 mg/l	41	35.3
≥10 mg/l	75	64.7

Table-V

Distribution of the study population according to ABG parameters (N=116)

ABG parameters	Mean±SD
pH 7.37±0.08	
PaCO2 (mmHg)	52.2±13.4
PaO2 (mmHg)	63.3±20.0
HCO3 (mmol/L)	30.0±6.5

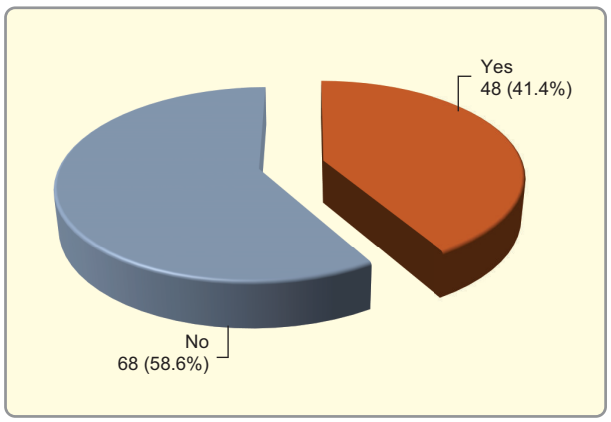

Figure 2: *Pie chart showing the need for NIV (non-invasive ventilation) of the study population (N=116)*

Table VI

Comparison between BUN/Albumin ratio based on the need for NIV (N=116)

	Need for NIV		p-value
	Yes(n=48) Mean±SD	No(n=68) Mean±SD	
BUN/Albumin ratio	9.1±2.8	5.3±1.8	0.001[s]
Range (min-max)	3.0-15.2	2.6-9.6	

s= significant
p-value reached from unpaired t-test

Table-VII

Comparison between severity of COPD exacerbation and BUN/Albumin ratio (N=116)

Severity of COPD exacerbation	Number of participants	BUN/ Albumin ratio Mean±SD	p-value
Mild	29	4.7±1.9	
Moderate	57	6.6±2.4	0.001[s]
Severe	30	9.5±2.9	

s= significant
p-value reached from ANOVA test

Table-VIII

Comparison between severity of COPD exacerbation based on the need for NIV (N=116)

Severity of COPD exacerbation	Need for NIV				p-value
	Yes (n=48)		No (n=68)		
	n	%	n	%	
Mild	0	0.0	29	42.6	
Moderate	18	37.5	39	57.4	0.001[s]
Severe	30	62.5	0	0.0	

s= significant
p-value reached from Chi-square test

Table-IX

Multivariate regression analysis for the need for NIV

	Regression coefficient (β)	Adjusted OR	95% CI		P value
			Lower	Upper	
Age	0.016	1.017	0.951	1.086	0.627[ns]
Smoking	0.200	1.222	0.508	2.940	0.655[ns]
IHD	1.320	0.744	0.534	1.139	0.954[ns]
DM	1.053	1.867	0.098	3.236	0.238[ns]
HTN	0.355	1.398	0.446	4.380	0.565[ns]
pH (<7.35 or >7.45)	1.317	3.734	1.053	13.246	0.041[s]
PCO2	1.093	2.098	1.010	3.194	0.004[s]
PO2	-0.047	0.954	0.916	0.994	0.007[s]
BUN/Albumin ratio	1.671	19.629	13.976	27.561	0.001[s]

s= significant, ns= not significant

p-value reached from multivariate analysis by binary logistic regression analysis

OR=Odd's Ratio

Discussion:

This cross-sectional study sought to assess the association of the blood urea nitrogen (BUN) to serum albumin ratio with the need for non-invasive ventilation (NIV) in patients experiencing exacerbations of chronic obstructive pulmonary disease (ECOPD). The results demonstrate significant relationships between clinical and biochemical parameters, highlighting the importance of the BUN/Albumin ratio. Below is a detailed discussion of the findings:

This study included 116 patients diagnosed with exacerbations of chronic obstructive pulmonary disease who fulfilled the inclusion and exclusion criteria from September 2023 to March 2025. The present study findings were discussed and compared with previously published relevant studies.

The heightened risk of COPD (Chronic Obstructive Pulmonary Disease) with advancing age is due to several factors. These include the natural decline in lung function and tissue repair as we age, the cumulative effects of environmental exposures (such as smoking or pollution), a weakening immune system, and the increasing presence of other health conditions. As a result, older adults are more likely to develop COPD, especially if they have encountered these risk factors throughout their lives. In this study, we found that 55.2% of participants were in the 41-60 age range, with a mean age of 59.8 ± 9.8 years. Similarly, a study by Han et al. (2023) reported a mean age of 67.48 ± 10.79 years[10]. These findings support the idea that advancing age is a significant risk factor for COPD.

Smoking is a major risk factor for COPD because it directly damages the lungs through inflammation, oxidative stress, and structural changes, leading to reduced lung function over time. In this study, we found that male patients were predominant (94.8%) with a male-female ratio of 18.3:1. We also found 44 (37.9%) patients were current smokers, 66 (56.9%) were ex-smokers with an average of 25.2 ± 6.8 pack-year smoking history. 6 (5.2%) were never smokers. Most patients (76.4%) had a history of smoking of over 20 pack years, which aligns with the established link between smoking and COPD. A study carried out by Islam et al. (2013) found that increasing age, male sex, and increasing duration of smoking were regarded as independent risk factors for COPD in Bangladesh[11].

Regarding the exacerbations of COPD patients in this study, we found that 29 (25.0%) had mild exacerbations, 57 (49.1%) had moderate exacerbations, and 30 (25.9%) had severe exacerbations. Specifically, no mild cases, 37.5% of moderate cases, and all severe cases necessitated NIV intervention. As ECOPD severity progressed from mild to severe, there was a corresponding increase in the proportion of cases requiring NIV therapy.

The mean BUN/Albumin ratio was significantly elevated in patients requiring NIV (9.1±2.8) compared to those without (5.3±1.8, $p = 0.001$). We observed a distinguishable pattern wherein the mean BUN/Albumin ratio level increased progressively as ECOPD severity intensified. Specifically, the mean BUN/Albumin ratio level was lowest among patients with mild ECOPD 4.7±1.9, followed by those with moderate ECOPD 6.6±2.4, and highest among severe ECOPD patients 9.5±2.9. This progressive increase in the BUN/Albumin ratio level suggests a potential association between worsening ECOPD severity and the need for NIV.

According to the study's findings, the degree of exacerbations and the requirement for NIV are related to the BUN/Albumin ratio level.

This pattern emphasises how useful the BUN/Albumin ratio level is as a possible gauge of the severity of the disease; larger values may be a sign of more severe exacerbations and a greater need for NIV.

We also evaluated the relationship between ABG and BUN/Albumin ratio; it was observed that the mean pH was 7.37 ± 0.08, mean PaCO2 was 52.2±13.4, mean PaO2 was 63.3±20.0 mmHg, and mean HCO3 was 30.0 ± 6.5 mmol/L and higher BUN/Albumin ratio level was observed in patients who had respiratory acidosis.

NIV is highly effective in treating hypercapnic respiratory failure in ECOPD. It has been shown to reduce mortality, morbidity, and hospital length of stay, making it a cornerstone of modern COPD care. In this study, among the 116 patients, 48 (41.4%) patients required NIV, while 68 (58.6%) patients did not. In a study conducted by Gomes et al. (2023), it was found that 35.3% of patients required NIV[12]. Another study conducted by Sun et al. (2021) found that 30.0% of patients needed NIV[13]. The above-mentioned findings are consistent with this study.

Based on multivariate regression analysis, pH (<7.35 or >7.45) with adjusted OR 3.734, 95% CI (1.053-13.246), PCO2 with adjusted OR 2.098, 95% CI (1.010-3.194), PO2 with adjusted OR 0.954, 95% CI (0.916-0.994), and BUN/Albumin ratio with adjusted OR 19.629, 95% CI (13.976-27.561) were significantly associated with the need for NIV. However, Age, Smoking, IHD, DM, and HTN were not significantly associated with the need for NIV.

This study shows a significant association between the BUN/Albumin ratio and the need for NIV in patients with ECOPD. The results suggest that as the severity of ECOPD exacerbations increases, the BUN/Albumin ratio also increases, reflecting a higher degree of metabolic stress and inflammation, which are key contributors to respiratory failure in COPD patients.

Conclusion:

The BUN/Albumin ratio is significantly associated with the need for NIV in patients with ECOPD. The higher the BUN/Albumin ratio, the greater the likelihood that a patient will require NIV. This association suggests that the BUN/Albumin ratio could serve as a useful clinical marker for early identification of patients at risk of respiratory failure and help guide timely intervention, especially in settings where ABG is not available.

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ORIGINAL ARTICLE

Eosinopenia as a Marker of Sepsis and Mortality in Intensive Care Unit patients

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Abstract

Background: *Sepsis is one of the most common causes of mortality and morbidity in the intensive care unit. Despite continuing advances in diagnosis and treatment, sepsis remains one of the important causes of higher mortality and morbidity.*

Purpose: This study was done to evaluate eosinopenia as a marker of sepsis and mortality in intensive care unit patients.

Materials and methods: *This cross sectional study was carried out in the Department of Clinical Pathology in collaboration with Department of Anaesthesia, Analgesia and Intensive care Medicine and Department of Microbiology and Immunology, Bangladesh Medical University, Dhaka.*

Observations and Results: Study population was 74 patients selected from intensive care unit, BMU. The sensitivity and specificity of AEC for diagnosis of sepsis was 73% and 62% respectively which was obtained by ROC curve. The area under ROC curve was 0.765 in current study. AEC decreased in patients who died due to sepsis. In infection group the mean AEC was20±11.4 cells/cumm in survivors and 9.2± 9.3 in died. In non infection group the mean AEC was140.0±57.7cells/cumm in survivors and 48.0±16.3 in died.

Conclusion: *This present data revealed that decreased AEC was significantly associated with sepsis and mortality.*

Key words: *Absolute eosinophil count (AEC), Sepsis, Intensive care unit (ICU), Receiver operating characteristic curve (ROC).*

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Introduction

Sepsis is one of most common causes of mortality and morbidity in the intensive care unit (Abidi et al., 2008). Early diagnosis of sepsis plays an integral role in the morbidity and mortality of patients admitted to the intensive care unit (Rey et al., 2007).

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Sepsis is a systemic inflammatory response to infection, manifested by two or more of the following condition as a result of infection: Temperature >38^0c or < 36^0c, Heart rate >90 beats/min, Respiratory rate > 20 breaths/min and white cell count >12,000/cumm,<4000/cumm, or >10% immature (band) forms (Abidi et al., 2008, Bone et al.,1992). 8

Sepsis was documented more than 35% of patients during their ICU stay (Vincent et al., 2006). The hospital mortality ranged from 16.9% for non-infected patients to 53.6% for patients who had infection at ICU (Curtiset al., 2002).

Normal eosinophil count is 40-400 cells/cu mm of blood (Firkin, 2006). Eosinopenia refers to a reduction in the normal number of circulating eosinophils (Bain, 2003). The level of eosinophils is normally tightly regulated (Abidi et al.,2008). Eosinophil production is regulated by IL-3, IL-5 and granulocyte macrophage colony stimulating factor (GM-CSF). Without these cytokines, eosinophil cannot survive. These cytokines are not significantly activated in patients with sepsis (Wibrowet al.,2011).

Sepsis and non-infectious systemic inflammatory response syndrome (SIRS) produce very similar clinical feature (Rey et al., 2007). Therapy and outcome differ greatly between patient with and those without sepsis (Gibot et al., 2004). The definitive diagnosis of sepsis is made by a positive culture, which requires a minimum of 48-72 hours (Reimer et al., 1997). As the culture procedure is costly and longer time required, other tests in the diagnosis of sepsis are required (Vincent et al., 2010). Several markers like C-reactive protein, procalcitonin, lactate, Interleukin-1 (IL-1), Interleukin-6 (IL-6), Tumor necroting factor (TNF), triggering receptor expressed on myeloid cells-1 (TREM-1) etc have been reported to predict sepsis (Shaban et al., 2010). Most of these markers are expensive, not easily accessible to clinicians, less sensitive and not ideal for early diagnosis of sepsis. Among these markers blood eosinophil count is simple, easy, quick, less expensive and reliable marker of sepsis (Shaabanet al., 2010, Rinaldo et al., 1999). It reduces widespread use of antibiotic, mortality and sepsis related complications. So this study was carried out to evaluate the diagnostic sensitivity of eosinopenia for detection of sepsis in ICU patients.

Materials and methods

This cross sectional study was carried out at the Department of Clinical Pathology in collaboration with Department of Anesthesia, Analgesia and Intensive care Medicine and Department of Microbiology and Immunology, Bangladesh Medical University (BMU), Dhaka from March 2012 to February 2013.Study population was 74 patients selected from intensive care unit, BSMMU, according to inclusion criteria. Prior to the commencement of this study, the research protocol was approved by the Institutional Review Board (IRB) of BSMMU, Dhaka. 2ml blood was collected for CBC including AEC which was done by automated cell counter and rechecked manually microscopically. Single set of culture was done for each patients. All data were recorded systematically in a preform data collection sheet and expressed as mean ± standard deviation (SD), t-test, Z test, Chi-square test. ROC curve and the respective area under curves was calculated for eosinophils. Sensitivity, specificity were calculated at the best cut off value. Statistical analysis was done by using statistical package for social science SPSS 17.0. P value <0.05 was considered as significant.

Result

In this study, 74 patients were divided into two group according to blood culture findings. Out of 74 patients, 34 patients were considered as proven sepsis by blood culture as infection group. The rest 40 patients were considered by blood culture as non infection group. Suspected case of sepsis and adult age group were included in the study. The area under the ROC curve was 0.765 in current study. In ROC curve the cut-off value of AEC < 40 cell/cumm. At this cut-off value the sensitivity and specificity of AEC in diagnosing infection were found to be 72.5% and 61.8% respectively. These findings were statistically significant (P < 0.001). In infection group the mean AEC was 20±11.4 cells/cumm in survivors and 9.2± 9.3 in died. In non infection group the mean AEC was 140.0±57.7 cells/cumm in survivors and 48.0±16.3 in died. The difference was statistically significant between two groups.

Table-I

Distribution of the study populations according to Absolute Eosinophil Count (AEC) (n=74)

AEC (cell/cumm)	Infection group (n=34)		Non infection group (n=40)		*p*Value
	n	%	n	%	
<40	25	73.5	13	32.5	
>40	9	26.5	27	67.5	
Mean± SD	18.3±11.4		145.0±57.7		0.001

P value reached from unpaired t-test.

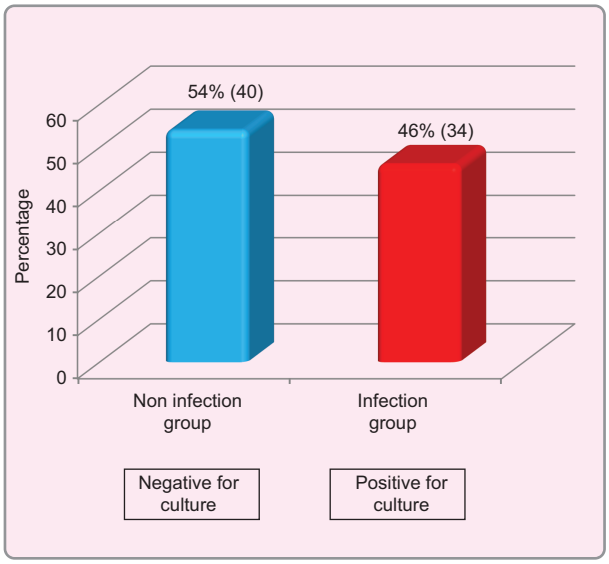

Figure 1: *Bar diagram showing distribution of bacterial growth in study patinets (n=74)*

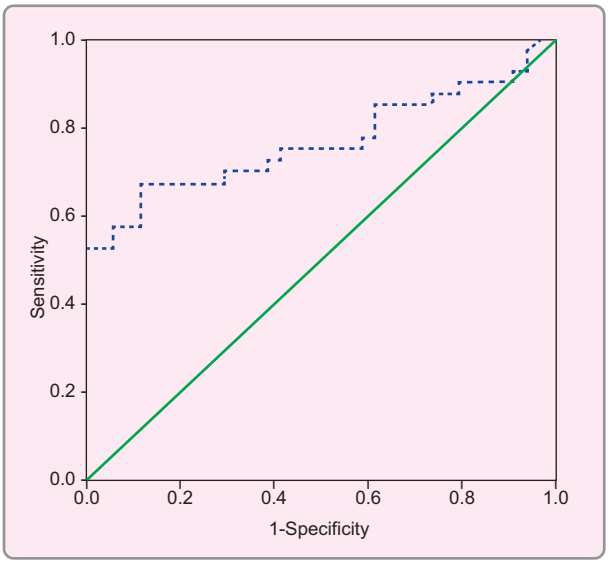

Figure 2: *Receiver-operating characteristic (ROC) curve of AEC for prediction of infection*

Fig. 1 shows blood culture was positive in 34 patients (46%) which indicate infection group and negative in 40 patients (54%) which indicate non infection group.

Table I shows the AEC of the study patients. The mean AEC was found18.3±11.4 cells/cumm in infection group and145.0±57.7cells/cumm in non infection group. The difference was statistically significant (P<0.05) between two groups.

Receiver-operating characteristic (ROC) curve of AEC for prediction of infection

The area under the ROC curves for the infection predictors is depicted in fig 2.ROC were constructed using AEC of the patients with infection, which gave a AEC cut off value of (<40cells/cumm) as the value with a best combination of sensitivity and specificity for infection. At this AEC cut-off value of<40cells/cumm, the sensitivity and specificity of AEC in infection was 72.5% and 61.8%, respectively. These findings were statistically significant (P<0.001).

Table II shows the AEC of study patients. In infection group the Mean±SD AEC was found 20.3±11.4 cells/cumm in survivors and 9.2±9.3 in died. In no infection group the Mean±SD AEC was

Table-II

Distribution of absolute eosinophil count depending on outcome in groups.

Group	AEC (Cells/Cumm)		p-value
	Infection(Mean±SD)	Noninfection (Mean±SD)	
Survivors	20.3±11.4	140±57.7	0.001
Died	9.2±9.3	48.0±16.3	

found140±57.7 cells/cumm in survivors and 48.0±16.3 in died. The deference was statistically significant between two groups.

Discussion

This study was carried out to evaluated absolute eosinophil count for early diagnosis of sepsis compared with gold standard blood culture.

In this study mean value of AEC was 18±11.4 cells/ cu mm in infection group and 145±57.4 cells/ cu mm in non infection group. The mean AEC difference was statistically significant (p less than 0.001) between two groups which indicates sepsis was associated with decreased eosinophil count. Similar findings were observed in the study done by Abidi et al (2008), Shaban et al (2010), Kadir et al (2012), Gil et al (2003). From the study of Kadir et al (2012) mean AEC count were 23±46 cells/ cu mm in sepsis and 143±101 cells/ cu mm in patients without sepsis. This finding was similar to our study. In previous study the sensitivity of eosinopenia in sepsis patient had a reasonable range of variation. Our study showed sensitivity 75.5% which was consistent with the study of Abdi et al (2008). From the study of Abidi et al (2008), Bayram el al (2012), Lopez et al (2010), Gil et al (2003), sensitivity was 71%, 61.4%, 64.8%, 64% respectively. These results were nearly consistent with our study. The present study has also defined the specificity of eosinopenia in sepsis patients. Our study showed specificity of eosinopenia for diagnosis of sepsis was 61.8% which was consistent with the study of Shaban et al (2010). According to their observation specificity was 65%. From the study of Lopez et al (2010), Moura et al (2011) specificity was 70.9%, 71% respectively. These results were nearly consistent with our study. The area under ROC curve was 0.765 in current study. This is similar to the observation of the study done by Shaban et al (2010) which was 0.72. Considering the sensitivity specificity, this study implies that eosinopenia is reliable as a diagnostic tool for sepsis. The result of our study revealed, mortality was 41% in infection group and 22.5% in non infection group. The mortality rate in our study seems high and related to infection. On the other hand lower eosinophil count group had worse outcome than normal eosinophil count. This finding is consistent with the study done by Abidi et al(1008). From the study of Abidi et al (2008), ICU mortality was higher in infection group (42%) than non infection group(25%). In our study, the mean AEC was 20.3±11.4 cells/cumm in survivors and 9.2±9.3 cells/ cumm in died which was nearly consistent with the study of Abidi et al (2011). From the study of Abidi et al (2011), the AEC was 30 cells/cumm in survivors and o cells/cumm in died.

Conclusion

The result from our study support that AEC is significantly lower in patients with sepsis and eosinopenia associated with prognosis of outcome in critical condition. An early diagnosis of sepsis is made by absolute eosinophil count that can be obtained from routine laboratory test (Complete blood Count) which is simple, quick, cost effective and readily available.

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ORIGINAL ARTICLE

Pattern of Bacterial Infection in Acute Exacerbation of Chronic Obstructive Pulmonary Disease (COPD) in a Tertiary Care Hospital

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Abstract

Background: *Acute exacerbation of COPD is one of the important problems during management of COPD & has considerable impact on morbidity, mortality and quality of life. Acute exacerbation can occur due to bacterial or viral infection & non-infectious causes. Sputum culture & real-time PCR can identify the pattern of bacterial infection. Rational use of antibiotic can reduce the cost of treatment & possibility of antibiotic resistance.*

Material and Methods: *This cross sectional study was conducted on hospital admitted & outdoor attended acute exacerbation of COPD patients using sputum Gram stain, culture & Real time PCR.*

Results: *The mean age of the respondents was 60.02±10.55 years. Maximum patients 49(79.0%) age above 50 years followed by 13(21.0%) below 50 years. Most patients had dyspnea 54(87.1%) followed by increased sputum volume 31(50.0%), increased wheez or cough 23(37.1%), increased sputum purulence 19(30.6%). Considering sputum culture & RT-PCR, 29(46.8%) patients was found microbiologically positive. Klebsiella was isolated most frequently 10(34.5%) followed by Haemophilus 7(24.1%). Type 1 & type 2 AECOPD showed more bacteriological positivity than type 3 (85.7%, 80.8% & 6.9% respectively, p=<0.001). Bacteria isolation was significantly associated with increased sputum purulence & volume (89.5% & 67.7% respectively, p=<0.001) but not in case of dyspnea (48.1%, p=0.573).*

Conclusion: *In conclusion, bacterial isolation was more common in type 1 & type 2 AECOPD than type 3, Klebsiella infection was most common. Increased sputum purulence & volume significantly indicates bacterial infection in AECOPD.*

Keywords: *COPD, Acute exacerbation, Sputum culture, Real-time PCR.*

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Introduction

COPD is a common, preventable & treatable disease that is characterized by persistent symptoms & airflow limitation that is due to airway &/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases, according to GOLD 2019[1]. The chronic airflow limitation that is characteristic of COPD is caused by a mixture of small airways obstruction & parenchymal disease. Chronic inflammation causes structural changes, narrowing of the small airways & destruction of the lung parenchyma that leads to the loss of alveolar attachments to the small airways & decreases lung elastic recoil. In turn, these changes diminish the ability of the airways to remain open during expiration. A loss of small airways may also contribute airflow limitation & mucocilliary dysfunction is a characteristic feature of the disease[2]. COPD constitutes a major health problem[3]. COPD is currently the 4th leading cause of death in the world[4]. Prevalence of COPD in the total population ranged from a low of 7.8% in Mexico City, Mexico, to a high of 19.7% in Montevideo, Uruguay. The prevalence of COPD among Dhaka City population in Bangladesh is 11.4%[5]. Using operational severity criteria adopted from Global Initiative for Obstructive Lung Disease (GOLD) mild, moderate, severe and very severe COPD were found in 42.7%, 27.2%, 20.4% and 9.7% respectively[5]. COPD exacerbations are very common, affecting about 20% of COPD patients[6]. The estimated annual costs of COPD are $24 billion, and 70% are related to exacerbations requiring hospitalization[7]. Acute exacerbations of COPD (AECOPD) have considerable impact on morbidity, mortality and quality of life[8].

Common triggers for AECOPD include viral and/or bacterial infection of the tracheobronchial tree and air pollution, but the cause of approximately one-third of severe exacerbations cannot be identified[9]. 78% of patients with severe COPD exacerbations admitted to the hospital are associated with respiratory virus and/or bacterial infection. The presence of infection with viruses (48.4%) and/or bacteria (54.7%) at exacerbation was associated with both clinical and lung function indicators of increased exacerbation severity[10].

Importantly, the presence of co infection with both viruses and bacteria was found in 25% of exacerbations and these patients had more severe functional impairment and longer hospitalization[10]. A survey including 69,820 patients who had been hospitalized for exacerbations of COPD in 360 hospitals throughout the United States showed that 85% of all patients were given antibiotics, but not all patients equally experienced benefit from antibiotics, patients with signs of bacterial infection and more severe exacerbations seem to be benefitted from antibiotics, prescribing antibiotics for viral infections or noninfectious causes of AECOPD is ineffective and increases the risk of toxicity and the development of bacterial resistance[11]. In patients with COPD, the clinical manifestations of systemic inflammation due to infectious and non-infectious causes are similar. The differential diagnosis of these two conditions is very important in order to administer the correct treatment regimen and avoid unnecessary antibiotic use, thus reducing the morbidity, mortality and care-related costs. Nevertheless, antibiotic use in AECOPD is undoubtedly still excessive, exposing patients to considerable cost and potential adverse effects, and driving antibiotic resistance. In this context, a rapid, specific test to identify lower respiratory bacterial infections would be a major advancement, limiting the inappropriate use of antibiotics which is considered to be a main cause of the spread of antibiotic- resistant bacteria[12].

Culture from sputum yields pathogenic bacteria in 32.2% of AECOPD cases[13]. Sputum Gram stain & culture showed Gram negative bacteria caused exacerbation in 63.33% of the patients, Gram positive bacteria in 23.33% of them and atypical bacteria in 13.34% of the patients. *Haemophilus influenza* (30%) was the most common organism followed by *Streptococcus pneumoniae* (23.33%), *Pseudomonas aeruginosa* (23.33%), *Moraxella catarrhalis* (10%), *Mycoplasma pneumoniae* (6.67%), & *Chlamydia pneumoniae* (6.67%)[6].

Materials and Methods

This was a cross sectional study conducted between January 2019 to August 2019 in Department of Respiratory Medicine of Bangladesh Medical University (BMU).

COPD patients attended in Respiratory Medicine Department of BMU in inpatient & outpatient for acute exacerbation was selected. Ethical approval was taken from Institutional Review Board, BMU. The patients were informed about the purpose of the study and ethical issues. The following eligibility criteria were employed to select the study population.

Inclusion criteria:

All patients attended in Respiratory Medicine Department with acute exacerbation of COPD.

Exclusion criteria:

1. Patient with any type of immunosuppression-malignancy, organ transplantation, cytotoxic drugs.

2. Preexisting lung disease other than COPD.
3. Pulmonary tuberculosis.

Consecutive sampling was applied. Total 153 patients were screened. Among them 9 were excluded for pleural effusion, 12 for bronchiectasis, 6 for consolidation, 7 for bronchial carcinoma, 24 for below standard sputum, 13 for not giving consent & 20 for lost to follow up & 62 patients were selected as study sample.

Operational definitions were set according to GOLD 2019[1]. Acute exacerbation of COPD was defined & classified as Type 1: Increased dyspnea, sputum volume & sputum purulence, Type 2: When two of these symptoms were present, Type 3: When one of the three symptoms was present in addition to at least one of the following

- Upper respiratory infection within the past 5 days
- Fever without other cause
- Increased wheezing or cough
- Increase in respiratory rate or heart rate by 20% as compare with baseline[14]

Spontaneous expectorated sputum was collected in early morning in front of investigator & stained with Gram stain & cultured in appropriate media (Chocolate agar, Blood agar & McConkey's agar). Sputum was cultured when there was <10 epithelial cells & >25 leukocytes per low power field. ≥ 1000c.f.u./ml considered as significant growth. Real-time PCR for respiratory bacteria done by Seegene Allplex respiratory panel 4 & FTD Bacterial pneumonia_CAP which detects *Mycoplasma pneumoniae, Chlamydophila pneumoniae, Legionella pneumophila, Haemophilus influenzae, Streptococcus pneumoniae, Staphylococcus aureus, Moraxella catarrhalis, Bordetella pertussis*. Compatible instrument was CF×96tm Real-time PCR (Bio-Rad). After collection, the data was checked and rechecked for omission, inconsistencies and improbabilities. Means and standard deviations (SD) was used to summarize continuous variables, while percentages were used for categorical variables. Data analysis was performed by Statistical Package for Social Science (SPSS), version-22.

Results

This table shows Acute exacerbation type -3 was found in maximum cases (46.8%) followed by 41.9% patients' type 2 and 11.3% patients had type 1.

Table-I

Distribution of the study subjects by acute exacerbation type (N=62)

Acute exacerbation type	Frequency	Percentage (%)
Type-1	7	11.3
Type-2	26	41.9
Type-3	29	46.8
Total	62	100.0

Table-2 shows among 29 positive cases, gram positive 4 (13.8%) cases, gram negative 24(82.8%) cases, 1(3.4%) case was atypical and mixed 1 case.

Table-II

Distribution of the study subjects by type of isolated bacteria (RT-PCR and culture) (N=29)

Bacteria type	Organism	Frequency	Percentage (%)
Gram positive	*Streptococcus pneumoniae*	3	10.3
	Staphylococcus aureus	1	3.4
Total		4	13.8
Gram negative	*Klebsiella pneumonae*	10	34.5
	Haemophilus influenzae	7	24.1
	Moraxella catarrhalis	3	10.3
	Legionella pneumophila	2	6.9
	Pseudomonas aeruginosa	2	6.9
Total		24	82.8
Atypical	*Mycoplasma pneumoniae*	1	3.4
Total		29	100.0
Mixed	*Haemophilus influenzae* + *Streptococcus pneumoniae*	1	3.4

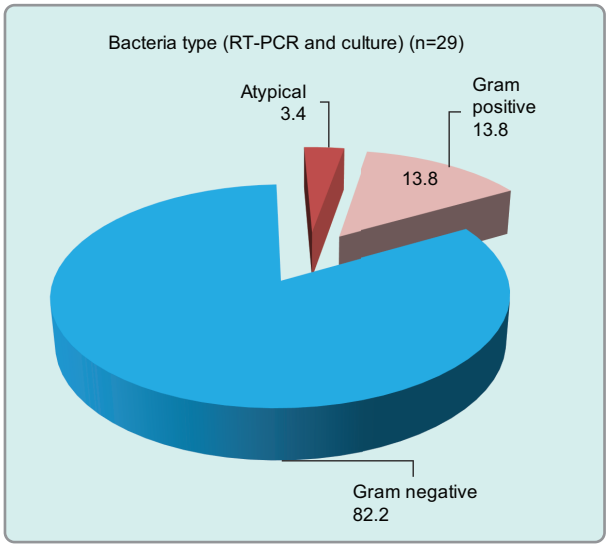

Figure-1: *Bacteria type*

Table-3 shows *Pseudomonas* was only isolated in type-1 exacerbation, *Klebsiella* was more frequent in type-1 & type-2 (33.3% in both) & *Haemophilus* in type-2 (28.6%).

Table-6 shows that dyspnea, increased sputum volume and increased sputum purulence relatively more in *Klebsiella pneumoniae*

Table-III

Distribution of the organism on the basis of sputum culture and RT-PCR by acute exacerbation type (N=29)

Acute exacerbation type	n	Gram negative					Gram positive		Atypical
		Klebsiella pneumonae	*Pseudomonas aeruginosa*	*Haemophilus influenzae*	*Moraxella catarrhalis*	*Legionella pneumophila*	*Streptococcus sp*	*Staphylococcus aureus*	*Mycoplasma pnemoniae*
Type-1	6	2(33.3%)	2(33.3%)	0(0.0%)	0(0.0%)	0(0.0%)	2(33.3%)	0(0.0%)	0(0.0%)
Type-2	21	7(33.3%)	0(0.0%)	6(28.6%)	3(14.3%)	2(9.5%)	1(4.8%)	1(4.8%)	1(4.8%)
Type-3	2	1(50.0%)	0(0.0%)	1(50.0%)	0(0.0%)	0(0.0%)	0(0.0%)	0(0.0%)	0(0.0%)

This table shows that bacteria isolation was significantly more in type-1 AECOPD compared to type-2 and type-3.

Table-IV

Association of sputum RT-PCR and culture positivity with type of acute exacerbation of COPD (N=62)

Type of acute exacerbation of COPD	n	Sputum RT-PCR and culture positivity		Chi-square test
		Negative	Positive	
Type I df=2 p= <0.001*	7	1(14.3%)	6(85.7%)	χ^2 =34.86
Type 2	26	5(19.2%)	21(80.8%)	
Type 3	29	27(93.1%)	2(6.9%)	
Total	62	33(53.2%)	29(46.8%)	

*significant

This table shows that bacteria isolation was significantly more in increased sputum volume and purulence.

Table-V

Association of sputum RT-PCR and culture positivity with symptoms of acute exacerbation of COPD (N=62)

Type of acute exacerbation of COPD	n	Sputum RT-PCR and culture positivity		Chi-square test
		Negative	Positive	
Dyspnea	54	28(51.9%)	26(48.1%)	χ^2 =0.317, df=1 p= 0.573^{ns}
Increased sputum volume	31	10(32.3%)	21(67.7%)	χ^2 =10.95, df=1 p=< 0.001*
Increased sputum purulence	19	2(10.5%)	17(89.5%)	χ^2 =20.06, df=1 p=<0.001*

*significant, ns= not significant

Table-VI

Distribution of the organism on the basis of symptoms

Symptoms	n	Gram negative					Gram positive		Atypical
		Klebsiella pneumonae	*Pseudomonas aeruginosa*	*Haemophilus influenzae*	*Moraxella catarrhalis*	*Legionella pneumophila*	*Streptococcus sp*	*Staphylococcus aureus*	*Mycoplasma pnemoniae*
Dyspnea	26	8(30.8%)	2(7.7%)	6(23.1%)	3(11.5%)	2(7.7%)	3(11.5%)	1(3.8%)	1(3.8%)
Increased sputum volume	21	8(38.1%)	2(9.5%)	3(14.3%)	3(14.3%)	2(9.5%)	2(9.5%)	0(0.0%)	1(4.8%)
Increased sputum purulence	17	7(41.2%)	2(11.8%)	4(23.5%)	0(0.0%)	0(0.0%)	3(17.6%)	1(5.9%)	0(0.0%)

Discussion

This cross sectional study conducted in the Department of Respiratory Medicine, Bangladesh Medical University, Dhaka including 62 acute exacerbation of COPD patients to assess the pattern of bacterial infection.

In the present study we found type-3 AECOPD is most common among study samples (46.8%) because most of AECOPD usually mild[15]. Bacterial infection causes inflammation & recruitment of neutrophil in airway. In current study sputum purulence is associated with more bacterial isolation (89.5%). In present study, predominant Gram negative infection may indicate micro aspiration of oral secretions. El-Halim and Sayed (2015) [6] reported in their study Gram negative bacteria caused COPD exacerbation in 66.33% of the patients, Gram positive bacteria in 23.33% of them and atypical bacteria in 13.34% of the patients . Eller et al. (1998) [16] reported on patients with acute exacerbations of COPD who require mechanical ventilation, 64% of the bacteria isolated by a protected specimen brush were Gram-negative bacteria. Miravitlles et al. (1999) [17] indicated higher prevalence of infection in severe AECOPD. Current study also found higher incidence of bacterial isolation (85.7%) from type-1 or severe AECOPD.

The common organisms in gram positive bacteria were *Streptococcus* (10.3%), *Staphylococcus* (3.4%), in gram negative pathogen common organisms were *Klebsiella* (34.5%), *Haemophilus* (24.2%), *Moraxella* (10.3%) & *Pseudomonas* (6.9%) in present study. Nair et al. (2019) [18] observed predominantly isolates of *Pseudomonas* in 10.1% of patients. Chawla et al. (2008)[19] also showed *Pseudomonas* in 25.95% cases. These findings are similar to current study because they found *Pseudomonas* predominantly in severe exacerbations & present study showed isolation of *Pseudomonas* only in type-1 AECOPD. In an Indian study done by Patel et al. (2015) [20] in Gujarat in 2011-2012, *Streptococcus pneumonia* was found to be the commonest pathogen. Difference in bacterial isolation may represent the geographical variation of microorganisms.

Klebsiella pneumonia was found as the predominant isolate in study by Madhvi et al. (2012)[21]. This study also found *Klebsiella* as the commonest pathogen.

Kuwal et al. (2018)[22] demonstrated bacterial pathogens (PPMs) were identified in 47% of all hospitalized patients, based on sputum culture, *Pseudomonas* and *Klebsiella* were the most frequently isolated organisms. Boixeda et al. (2012) [23] reported *Pseudomonas* in 38.23% of cases and Eller et al. (1998)[16] reported *Pseudomonas* sand Enterobacteriaceae spp in 48.2% patients of their study. Thus, this is in line with the previous studies. The higher proportion (34.5%) of isolation of *Klebsiella* in the present study also deserves special mention. Kuwal et al. (2018) [22] reported *Klebsiella* in 29.41% cases. Lin et al. (2007)[24] reported high prevalence of infection with K. pneumoniae (19.6%) and P. aeruginosa (16.8%) among hospitalized patients of AECOPD. However, the isolation of *Klebsiella* was predominated in mild COPD.

Conclusion

Bacterial isolation was more common in more severe AECOPD (type-1 & type-2 than type-3), Klebsiella infection was most common. Increased sputum purulence & volume significantly indicates bacterial infection in AECOPD.

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ORIGINAL ARTICLE

Clinical Conditions, Mortality, and Associated Risk Factors in Patients with Flail Chest Injuries: A Tertiary Care Experience

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Abstract

Background: *Flail chest is a severe thoracic injury associated with significant morbidity and mortality. Understanding the clinical profile, outcomes, and risk factors is crucial for effective management. This study aimed to evaluate the clinical conditions, outcomes, and predictors of mortality in patients with flail chest injuries at a tertiary care center.*

Method:*This cross-sectional study was conducted in the Department of Thoracic Surgery, National Institute of Diseases of Chest & Hospital (NIDCH), Dhaka, Bangladesh, from January 2010 to December 2010. This study included 60 patients with flail chest injuries who were admitted at this study institution.*

Results: *The mean age of patients was 39.85 ± 16.3 years, with a majority over 40 years (45%). Males predominated (72%), with a male-to-female ratio of 2.5:1. Central flail chest was the most common type (63.3%), and road traffic accidents were the leading cause of injury (80%). All patients presented with chest pain, while 63.3% had respiratory distress and 25% exhibited cyanosis. The mean number of fractured ribs was 7 ± 1.5. Pneumonia developed in 31.7% of patients, and adult respiratory distress syndrome occurred in 8.3%. The mean hospital stay was 12.2 ± 1.25 days, and the overall mortality rate was 10%. Significant predictors of mortality included age e"60 years (83.3% of non-survivors vs. 7.4% of survivors, p < 0.0001), pneumonia (66.7% vs. 11.1%, p = 0.0006), and requirement for mechanical ventilation (83.3% vs. 5.6%, p < 0.0001).*

Conclusion: *Flail chest is associated with substantial morbidity and mortality. Older age, pneumonia, and mechanical ventilation are significant predictors of mortality, highlighting the need for early recognition and intensive management of high-risk patients.*

Keywords: *Flail chest, Thoracic trauma, Mortality, Risk factors, Mechanical ventilation*

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Introduction

Flail chest remains one of the most challenging conditions for thoracic surgeons and is widely recognized as a serious injury, with reported mortality rates ranging from 5% to 36% [1,2]. It is defined as the fracture of three or more adjacent ribs, each fractured in at least two places, resulting in a free-floating segment of the chest wall. Although relatively rare, flail chest represents the most severe form of blunt chest wall trauma and is an independent predictor of poor outcomes in patients with blunt chest injuries[3,4]. The injury typically results from a high-impact blunt force, often accompanied by underlying pulmonary contusions. Fractured ribs may lacerate lung tissue, leading to hemothorax or pneumothorax of varying severity[5]. Patients with severe chest wall injuries exhibit high rates of morbidity and mortality, with studies indicating that only 43% of affected patients return to full-time employment. Long-term complications frequently include chronic pain, subjective dyspnea, chest wall deformities, and reduced quality of life [3,4,6,7]. Isolated unilateral flail chest carries a relatively low mortality rate of around 6%, suggesting that higher mortality rates are often attributable to concomitant extra thoracic injuries [8]. Chest trauma encompasses a broad spectrum of injuries, ranging from minor abrasions and contusions to life-threatening insults. These injuries are a major contributor to morbidity and mortality, particularly in developing countries. Rapid urbanization and the proliferation of high-speed motor vehicles have further increased the incidence of traumatic chest injuries[9-11]. Chest trauma is a strong predictor of pulmonary complications, with the risk being highest among older patients, who experience longer ventilator dependence, higher rates of pneumonia, and greater incidence of acute respiratory distress syndrome compared to younger individuals. Bilateral chest involvement also signifies severe trauma, with nearly 50% of fatalities occurring in these patients [12-14]. Several factors have been identified as predictors for the need for mechanical ventilation in flail chest patients, including bilateral costochondral separation, male sex, development of pneumonia, pulmonary contusion, and the requirement for tracheostomy[5,7]. Morbidity is similarly influenced by high injury severity scores (ISS), shock, concurrent pulmonary contusions, and bilateral costochondral separation [5,15]. Despite the known association between flail chest and pulmonary contusions, short-term morbidity and mortality have not significantly improved over the past three decades. Pulmonary contusions, combined with pain, often result in shallow tidal volumes, alveolar collapse, arteriovenous shunting, and hypoxemia, ultimately leading to respiratory insufficiency [16,17]. Despite advances in the management of major chest injuries, flail chest remains a significant concern. The paradoxical motion of the flail segment reduces vital capacity and impairs effective ventilation, leading to pulmonary insufficiency[2]. Management strategies focus on preventing ventilation–perfusion mismatch, maintaining pulmonary hygiene, providing adequate fluid resuscitation, optimizing pain control, and considering surgical stabilization of the chest wall when appropriate [3,18]. In the present study, we aimed to evaluate the clinical conditions, outcomes, and predictors of mortality in patients with flail chest injuries at this tertiary care center.

Methodology & Materials

This cross-sectional study was conducted in the Department of Thoracic Surgery, National Institute of Diseases of Chest & Hospital (NIDCH), Dhaka, Bangladesh, from January 2010 to December 2010. In this study, we included a total of 60 patients who were admitted with flail chest injuries at this study institution.

These were the following criteria for eligibility as study participants:

Inclusion Criteria

a) Patients aged more than 18 years;

b) Patients admitted with a radiologically and clinically confirmed diagnosis of flail chest injury;

c) Patients who were willing to participate were included in the study

Exclusion Criteria

a) Patients with extensive burns with chest injuries;

b) Patients with pelvic and limb injuries;

c) Patients with pre-existing chronic respiratory conditions (e.g., COPD, interstitial lung disease) or with acute illnesses such as CKD, pancreatic

disease, asthma or Cardiac failure were excluded from the study.

Injury Severity Assessment: The Injury Severity Score (ISS) was used to quantify injury severity, following the method described by Copes et al. (1987). The ISS is derived from the Abbreviated Injury Scale (AIS-90). The three most severely injured body regions were identified, their AIS scores were squared, and the sums were obtained to calculate the ISS. In cases of multiple injuries, the grade was advanced by one level up to Grade III.

To specifically evaluate chest wall trauma, the Chest Wall Injury Scale was applied:

Based on ISS, patients were classified into three categories:

- Moderate injury: ISS 9–14
- Severe injury: ISS 16–24
- Critical injury: ISS >25

Data Collection Procedure: Informed written consent was obtained after an explanation of the study procedure. Data were collected prospectively using a structured questionnaire through interviews, observation, clinical examinations, and investigations. Baseline demographic details, cause of injury, clinical presentation,associated thoracic conditions and Injury Severity Score (ISS) were recorded for each patient. Radiological investigations, including chest radiography and computed tomography (CT) scans of the thorax (when indicated), were performed for diagnosis.

Clinical Management and Outcomes: All patients received standard trauma care, including airway management, pain control, oxygen supplementation, and mechanical ventilation when indicated. Associated injuries, complications such as pneumonia or acute respiratory distress syndrome (ARDS), hospital stay, and mortality were recorded.

Statistical Analysis: All data were systematically recorded in a pre-designed data collection form. Quantitative data are presented as mean and standard deviation, while qualitative data are shown as frequency and percentage. The data were analyzed using the chi-square test. A p-value <0.05 was considered significant. Statistical analysis was conducted using SPSS 19 (Statistical Package for Social Sciences) for Windows version 10. This study received ethical approval from the Institutional Review Committee of the National Institute of Diseases of Chest & Hospital (NIDCH).

Grade	Injury Type	Description	AIS-90 Score
I	Contusion	Any size	1
	Laceration	Skin and subcutaneous	1
	Fracture	< 3 ribs, closed; nondisplaced clavicle, closed	1–2
II	Laceration	Skin, subcutaneous, and muscle	1
	Fracture	e≥3 adjacent ribs, closed	2–3
		Open or displaced clavicle	2
		Nondisplaced sternum, closed	2
		Scapular body, open or closed	2
III	Laceration	Full-thickness, including pleural penetration	2
	Fracture	Open or displaced sternum, flail sternum	2
		Unilateral flail segment (< 3 ribs)	3–4
IV	Laceration	Avulsion of chest wall tissues with underlying rib fractures	4
	Fracture	Unilateral flail chest (≥3 ribs)	3–4
V	Fracture	Bilateral flail chest (≥3 ribs on both sides)	5

* This scale is confined to the chest wall alone and does not reflect associated internal thoracic or abdominal injuries.

Results

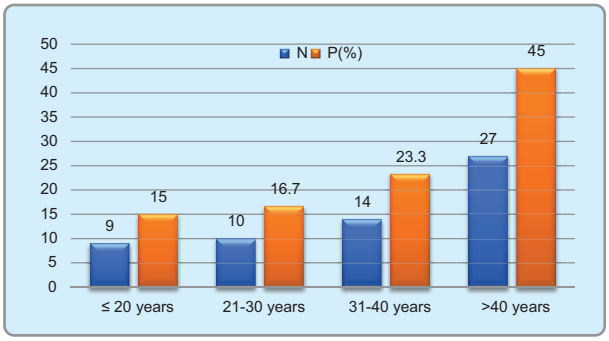

Figure 1: *Age Distribution of Study Patients*

In Figure 1, the age distribution shows that majority (45%) of patients were over 40 years, followed by 23.3% were 31–40 years, 16.7% were 21–30 years and 15% of patients were ≤20 years.

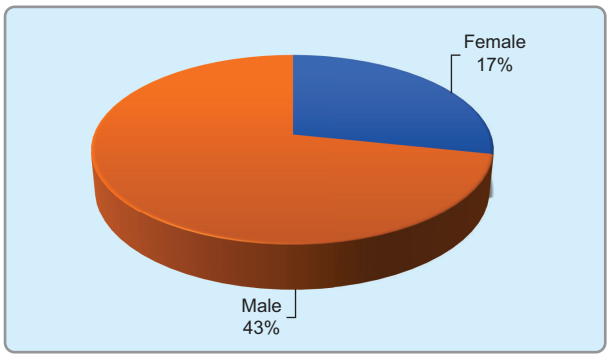

Figure 2: *Gender Distribution of Study Patients*

The pie chart shows that most of the study patients were males (72%) and 28% were females. The male-to-female ratio was 2.5:1 in this study.

Table-I

Baseline Characteristics of Study Patients with Flail Chest Injury (N = 60)

Variable	Frequency (n)	Percentage (%)
Age (years)		
Mean ± SD (years)	39.85±16.3	
Types of flail chest injury		
Central flail	38	63.3
Other types of flails	22	36.7
Cause of injury		
Road traffic accident	48	80
Fall from height	10	16.7
Others	2	3.3
Injury severity score (ISS)*		
Moderate (9 – 14)	2	3.3
Severe (16 – 24)	16	26.7
Critical (>25)	42	70

Table I summarizes the baseline characteristics of the study population. The mean age of patients was 39.85 ± 16.3 years. Central flail chest was the most common type of injury (63.3%), while other flail patterns accounted for 36.7%. Road traffic accidents were the leading cause of injury (80%), followed by falls from height (16.7%) and other causes (3.3%). Based on the Injury Severity Score (ISS), 3.3% of cases were categorized as moderate (9–14), 26.7% as severe (16–24), and 70% as critical (>25).

Table-II

Clinical Presentation, Associated Conditions and Outcomes in Flail Chest Patients (N = 60)

Clinical Presentation	Frequency (n)	Percentage (%)
Chest pain	60	100.0
Respiratory distress	38	63.3
Cyanosis	15	25.0
Number of fractured ribs	7±1.5	
Associated Condition		
Haemothorax	16	26.7
Haemopneumothorax	33	55.0
Lung contusion	22	36.7
Surgical emphysema	22	36.7
Clinical Outcomes		
Pneumonia	19	31.7
Adult respiratory syndrome	5	8.3
Hospital Stay	12.2±1.25	
Mortality	6	10.0

Table II shows that all patients presented with chest pain (100%), while respiratory distress was observed in 63.3% and cyanosis in 25%. The mean number of fractured ribs was 7 ± 1.5. Common associated conditions included haemopneumothorax (55%), lung contusion (36.7%), surgical emphysema (36.7%), and haemothorax (26.7%). Regarding clinical outcomes, pneumonia developed in 31.7% of patients, and 8.3% experienced adult respiratory distress syndrome. The mean duration of hospital stay was 12.2 ± 1.25 days, and the overall mortality rate was 10%.

Table-III

Risk Factors Associated with Mortality in Flail Chest Patients (N = 60)

Risk Factor	Survivors (n=54)	Non-survivors (n=6)	p-value
Age ≥60 years	4 (7.4%)	5 (83.3%)	< 0.0001
Male sex	36 (66.7%)	4 (66.7%)	1.000
Pneumonia	6 (11.1%)	4 (66.7%)	0.0006
Lung contusion	12 (22.2%)	2 (33.3%)	0.545
Mechanical ventilation	3 (5.6%)	5 (83.3%)	< 0.0001

Table III shows that out of 60 patients with flail chest, 6 (10%) died during hospitalization. Older age was found to be a significant predictor, as patients aged e"60 years accounted for 83.3% of non-survivors (5 of 6), compared to only 7.4% of survivors (4 of 54) ($p < 0.0001$). Pneumonia was also significantly more common among non-survivors (66.7%, 4 of 6) than survivors (11.1%, 6 of 54) ($p = 0.0006$). Similarly, the requirement for mechanical ventilation was strongly associated with mortality, being present in 83.3% of non-survivors (5 of 6) compared to just 5.6% of survivors (3 of 54) ($p < 0.0001$). On the other hand, male sex and lung contusionshowed no association with mortality.

Discussion

This study evaluated the clinical conditions, mortality, and associated risk factors in patients with flail chest injuries admitted to a tertiary care center. Flail chest, a severe manifestation of blunt thoracic trauma, is recognized as an independent predictor of poor outcomes. It is frequently complicated by respiratory compromise, ventilatory dependence, and secondary complications such as pneumonia and acute respiratory distress syndrome. In our series, chest pain and respiratory distress were the predominant clinical presentations, while haemo-pneumothorax and pulmonary contusion were the most common associated conditions. Among the 60 patients included, the mean age was 39.85 ± 16.3 years, with a male predominance of 72%, and road traffic accidents were the leading mechanism of injury (80%).

A study from the United Arab Emirates reported a mortality rate of 7.2% among chest trauma patients. Road traffic accidents accounted for 66% of cases, followed by falls (23.4%). Approximately 36.5% of patients sustained isolated chest injuries, while the remainder had associated head (27.4%) and extremity injuries (50%). Mortality was significantly associated with severe head trauma ($p < 0.0001$) and hypotension on admission ($p = 0.027$) [19].In the present study, the Injury Severity Score (ISS) proved useful in predicting morbidity and mortality. However, direct comparisons between studies are limited by methodological differences in assessing severity. Some studies considered ISS, the presence of bilateral flail segments, or pulmonary contusions as key indicators [20,21]. It is clinically logical that increasing injury severity correlates with higher mortality, though defining a universal threshold could better guide clinicians and facilitate standardized treatment protocols.

The overall mortality in our series was 10%. This rate is lower than that reported by Baru et al. (27.6%), Massaga et al. (24.2%), and Sanjay Datey et al. (22.23%) [9,22,23], but higher than findings from Al-Koudmani et al., Al-Eassa et al., and Khursheed et al. [10,19,24]. These differences may be attributed to variations in healthcare infrastructure, early intervention strategies, and perioperative care standards.

Consistent with other literature, most of our patients sustained injuries from road traffic accidents. However, in some reports, interpersonal violence was the leading cause of chest trauma. For instance, one study noted assault-related injuries in 52.5% of cases, higher than the 41% reported by Al-Koudmani et al. [25]. Regional sociocultural differences may explain this variability.Regarding the associated conditions, haemo-pneumothorax was most frequent (55%), followed by lung contusion and surgical emphysema (36.7%) and isolated haemothorax (26.7%). This is comparable to the findings from Addis Ababa, where haemo-pneumothorax was also the most common lesion [26].The mean hospital stay

in our series was 12.2 ± 1.25 days. Although the length of stay has been proposed as a predictor of mortality in flail chest patients, evidence is inconsistent. Some studies report a significant association [17,27], while others highlight the influence of confounding factors such as concomitant head or pelvic injuries in elderly patients, which may prolong admission independent of thoracic trauma severity.Only 13.3% of our patients required mechanical ventilation, which is lower than the 18.68% reported in an Indian cohort [2]. This variation may reflect differences in injury severity or institutional ventilation protocols. Importantly, the need for mechanical ventilation was a significant predictor of mortality ($p<0.0001$), consistent with the findings of Refaely Yael et al. [28].Pneumonia emerged as the most frequent complication, affecting 66.7% of non-survivors compared to 11.1% of survivors ($p = 0.0006$). This aligns with prior reports identifying pneumonia as the leading cause of morbidity in flail chest patients. However, lower incidences were documented in studies from Uganda, possibly due to differences in diagnostic capacity or patient characteristics [29].

Limitations of the study

This study was a single-center study. This study took a small sample size due to the short study period, which may limit the generalizability of the findings. The cross-sectional design did not allow long-term follow-up to evaluate functional recovery, quality of life, and late complications.

Conclusion and recommendations

Flail chest remains a serious thoracic injury with substantial morbidity and mortality. In this study, older age, development of pneumonia, and the need for mechanical ventilation were identified as significant predictors of mortality. The findings highlight the importance of early recognition of high-risk patients and aggressive management strategies, including careful monitoring, prompt respiratory support, and prevention of pulmonary complications, to improve clinical outcomes. Despite advances in trauma care, flail chest continues to pose a challenge, underscoring the need for multidisciplinary care and targeted interventions to reduce complications and mortality.

Further study with a prospective and longitudinal study design, including a larger sample size, needs to be done to validate the findings of this study.

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ORIGINAL ARTICLE

Status of Haematocrit Level in COPD Patient Admitted in Rajshahi Medical College Hospital

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Abstract

Introduction: *COPD is a common, preventable and treatable disease which is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.[1–3] It is a leading cause of chronic morbidity and mortality worldwide that includes an economic and social burden which is both substantial and increasing.[1,4]*

Aim of the study: *The aim of the study was to observe the haematocrit status in COPD patients admitted in Rajshahi Medical College Hospital.*

Methods: *This cross-sectional study was performed in the Department of Medicine, Rajshahi Medical College Hospital, Bangladesh, from March 2022 to February 2023. After careful history taking, examination and appropriate investigations fulfilling inclusion and exclusion criteria, total 100 clinically diagnosed COPD patients with a FEV1/vital capacity (FVC) ratio <70% and a FEV1 <80% predicted, were included in this study irrespective of their age, sex, race and ethnic group.*

Result: *Mean age of all patients was 55.42±7.05 years (median: 55, range: 40-71 year), wherein maximum patients belonged to 50-59 years of age (51%). Major part of the study patients was male (90%) with a male: female ratio 9:1. Maximum patients were hailed from rural area (70%). Most of the patients were illiterate or had below primary level of education (61%).Maximum patients were farmer or day laborer (51%).Of all, 58% patients were from lower socio-economic status. Most of the study patients were underweight (65%). More than*

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90% of the study patients were smoker. Maximum patients had severe COPD (60%) followed by very severe (22%) and moderate (18%) COPD. Mean FEV1 of all patients was 40.52±13.27% predicted. Average duration of COPD of study patients was 6.37±2.70 years. Mean haematocrit level of all patients was 48.49±3.92% wherein male had a mean of 48.97±3.64% and female had 44.20±3.91% haematocrit. Mean haematocrit level was significantly higher among very severe COPD patients (52.91±2.41%) compared to moderate and severe COPD (43.83±3.07 and 48.27±2.65%, respectively) as p value <0.001.Pearson correlation model showed significant moderate negative association between FEV1 and haematocrit level among COPD patients (R= -0.736, p value <0.001).

Conclusion: *Based on these research findings, mean haematocrit level was significantly higher among very severe COPD patients compared to moderate and severe COPD, which suggest that haematocrit level was significantly associated with severity of COPD.*

Keywords: *Haematocrit,COPD, Haemoglobin*

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Introduction

Chronic obstructive pulmonary disease (COPD) is a widespread, preventable, and treatable illness that is characterised by chronic respiratory symptoms and airflow limitation brought on by abnormalities of the airways and/or alveoli, which are typically brought on by prolonged exposure to harmful particles or gases[1,3]. It is a major contributor to chronic morbidity and mortality globally, with a significant and growing economic and social cost.[1,4] With a global incidence of 11.7%, it ranks as the third most common cause of death[1,5]. The main risk factors for developing COPD are genetic predisposition, smoking, and biomass exposure. It is not a pulmonary illness that occurs alone.The clinical significance of systemic effects and other chronic illnesses linked to COPD has gained more attention in recent years due to their substantial influence on the disease's progression[6,7].Significant extrapulmonary consequences of COPD include skeletal muscle weakness, dietary problems, and weight loss[3]. COPD patients' morbidity and death are negatively impacted by co-morbidities, which are significant occurrences in the disease's natural history. Patients with COPD frequently have co-morbid conditions such as diabetes, hypertension, cardiovascular disease, lung cancer, osteoporosis, and depression[8,9]. This complex pathophysiology is taken into consideration by newly developed prognostic algorithms that include both lung-specific and systemic characteristics[10].One of the most accurate ways to assess the severity of anaemia or polycythaemia is to use the haematocrit. It shows how many red blood cells there are in 100 millilitres of blood (a haematocrit of less than 39% indicates anaemia, while a haematocrit of more than 55% indicates polycythaemia)[6,11,12]. According to the World Health Organisation, anaemia in the general population is defined as a haemoglobin concentration of less than 13.0 g/dL in men and less than 12.0 g/dL in women. This classification is suitable for older adults. There is no particular haemoglobin concentration level that may be used as a cutoff score to identify which is clinically significant in people with COPD[13]. In several populations of COPD patients, it has been demonstrated that haemoglobin and hemocrit concentrations offer prognostic information.[7] Although the distribution varies greatly depending on the population under study, erythropoiesis in COPD can be influenced by a number of factors and present as either anaemia or polycythaemia. Patients with nocturnal desaturation or a PaO2 of less than 55 mm Hg frequently have an increased haematocrit[5,14,16]. Despite the fact that polycythaemia is "traditionally" linked to COPD, there is mounting evidence linking the two conditions[17]. Secondary polycythaemia was a prominent hallmark of COPD in the past, particularly prior to the advent of domiciliary oxygen. However, the prevalence appears to be low today, ranging from 6% to 18.1%. Rather, anaemia, which has a documented frequency of 6.2%–46.3%, may be more prevalent[4,7,10,18].The balance between inflammatory and hypoxic stimuli determines whether anaemia or polycythaemia develops.[9]These studies, however, only examined a small number of patients or short-term survival, or included patients who needed hospitalisation or were on long-term oxygen therapy. Although COPD is frequently diagnosed in our nation's hospitals, there aren't many studies examining the haematocrit's prognostic significance in connection to COPD. The aim of the study was to observe the haematocrit status in COPD patients admitted in Rajshahi Medical College Hospital.

Methods

This cross-sectional study was performed in the Department of Medicine, Rajshahi Medical College Hospital, Bangladesh, from March 2022 to February 2023. After careful history taking, examination and appropriate investigations fulfilling inclusion and

exclusion criteria, total 100 clinically diagnosed COPD patients>18 years of agewith a FEV1/vital capacity (FVC) ratio <70% and a FEV1 <80% predicted and patients participated willingly were included in this study irrespective of their age, sex, race and ethnic group. However, patients who had active pulmonary tuberculosis, bronchiectasis, malignancies, bronchial asthma, patients who had complications of COPD like pneumonia, pneumothorax and suspected pulmonary embolism, seriously ill patients and patients who had symptoms of less than two years were excluded from the study.

Results

Table-I

Distribution of sociodemographic characteristics of study patients (N=100)

Demographic characteristics of patients	Number of patients	Percentage (%)
Distribution of patients according to age		
40-49	22	22.00%
50-59	51	51.00%
60 or more	57	57.00%
Distribution of patients according to sex		
Male	90	90.00%
Female	10	10.00%
Distribution of patients according to residence		
Rural	70	70.00%
Urban	30	30.00%
Distribution of patients according to education status		
Illiterate	23	23.00%
Below primary	38	38.00%
Primary passed	16	16.00%
Below SSC	13	13.00%
SSC and above	10	10.00%
Distribution of patients according to occupation		
Service holder	17	17.0%
Businessman	16	16.0%
Farmer	28	28.0%
Day laborer	23	23.0%
Unemployed	16	16.0%
Distribution of patients according to socioeconomic status		
Lower Class	58	58.00%
Middle Class	33	33.00%
Higher Class	9	9.00%
Distribution of patients according to BMI status		
Underweight	65	65.00%
Normal	35	35.00%
Distribution of patients according to smoking history		
Current	42	42.00%
Ex-smoker	50	50.00%
Never	8	8.00%

Mean age of all patients was 55.42±7.05 years (median: 55, range: 40-71 year), wherein maximum patients belonged to 50-59 years of age (51%).Major part of the study patients was male (90%) with a male:female ratio 9:1.Maximum patients were hailed from rural area (70%).Most of the patients were illiterate or had below primary level of education (61%).Maximum patients were farmer or day laborer (51%).Of all, 58% patients were from lower socio-economic status.Most of the study patients were underweight (65%).More than 90% of the study patients were smoker, of whom 50% were ex-smoker and 42% were current smoker.

Table-II

Disease profile of study patients (N=100)

Disease profile	Frequency (n)	Percentage (%)
Severity of COPD		
Moderate	18	18
Severe	60	60
Very severe	22	22
FEV1 (in % predicted)		
Mean±SD	40.52±13.27	
Median	38	
Range (min-max)	21-78	
IQR (Q1-Q3)	30.25-47.75	
Disease duration (in year)		
Mean±SD	6.37±2.70	
Median	6	
Range (min-max)	1-13	
IQR (Q1-Q3)	5-8	

Maximum patients had severe COPD (60%) followed by very severe (22%) and moderate (18%) COPD. Mean FEV1 of all patients was 40.52±13.27% predicted. Average duration of COPD of study patients was 6.37±2.70 years.

Table-III

Haematocrit level of study patients (N=100)

Haematocrit (%)	Male n=90	Female n=10	All n=100
Mean±SD	48.97±3.64	44.20±3.91	48.49±3.92
Median	49	43.5	49
Range (min-max)	41-57	38-50	38-57
IQR (Q1-Q3)	46-52	41-48	46-51

Mean haematocrit level of all patients was 48.49±3.92% wherein male had a mean of 48.97±3.64% and female had 44.20±3.91% haematocrit.

Table-IV

Association between haematocrit level and severity of COPD (N=100)

Haematocrit (%)	Male n=90	Female n=10	All n=100
Mean±SD	48.97±3.64	44.20±3.91	48.49±3.92
Median	49	43.5	49
Range (min-max)	41-57	38-50	38-57
IQR (Q1-Q3)	46-52	41-48	46-51

P value was determined by one-way ANOVA test.

Mean haematocrit level was significantly higher among very severe COPD patients (52.91±2.41%) compared to moderate and severe COPD (43.83±3.07 and 48.27±2.65%, respectively) as p value <0.001.

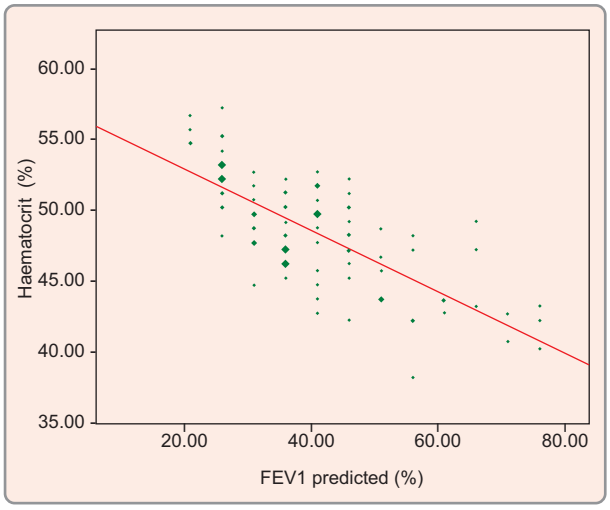

Figure 1: *Correlation between FEV1 and haematocrit level among COPD patients (n=100)*

Pearson correlation model showed significant moderate negative association between FEV1 and haematocrit level among COPD patients (R= -0.736, p value <0.001).

Discussion

Chronic obstructive pulmonary disease (COPD) is highly prevalent and associated with substantial morbidity and mortality[17]. Increased haematocrit level in COPD patients is associated with development of pulmonary hypertension, and leads to pulmonary endothelial dysfunction, reduced cerebral blood flow, hyperuricemia and gout, and increased risk of venous thromboembolic disease[5]. However, there is limited information in the current literature describing the distribution of haemoglobin (Hb) and its impact on outcomes in the COPD population. Therefore, this study was conducted with an aim to observe the haematocrit level in COPD patients. Total 100 clinically diagnosed COPD patients with a FEV1/vital capacity (FVC) ratio <70% and a FEV1 <80% predicted, were included in this study after careful history taking, examination and appropriate investigations fulfilling inclusion and exclusion criteria.In this study, average age of all patients was 55 years (range: 40-71 year), wherein maximum patients belonged to 50-59 years of age (51%). Similarly, previous two Bangladeshi studies by Alam et al. and Islam et al. also reported age >50 years as a significant risk factor for COPD[19,20]. Alam et al. observed that 50-59 years-aged people and 60-69 years-aged people were two times and five times more likely to develop COPD, respectively, compared to 40-49 years-aged people (50 to 59 years, OR: 2.2, 95% CI, 1.6-3.0; $p < 0.001$; 60 to 69 years, OR: 4.7, 95% CI, 3.5-6.4; $p < 0.001$)[19]. In current study, major part of the study patients was male (90%) with a male:female ratio 9:1. Similarly, several other studies also found male predominancy in COPD patients[19-23]. In this study maximum patients were hailed from rural area (70%). Here, most of the patients were illiterate or had below primary level of education (61%).Maximum patients were farmer or day laborer (51%).Of all, 58% patients were from lower socio-economic status. And from this study it was found that most of the study patients were underweight (65%). However, the number of COPD patients is higher among men, primarily because men are more exposed to tobacco smoking and outdoor activities in dusty environment or high intensity chemicals from air pollution. In this study, more than 90% of the study patients were smoker. Of whom 50% were ex-smoker and 42% were current smoker. Previous Bangladeshi literatures also found that smoking was significantly associated with an increased prevalence of COPD in Bangladeshi people[5,19,20,22,24].Hasshim et al. observed that smoking has a direct influence on the haematocrit of the patient and found significant correlation of smoking pack years with both severity of COPD and haematocrit level.[5] Maximum patients had severe COPD (60%) followed by very severe (22%) and moderate (18%) COPD. Mean FEV1 of all patients was 40.52±13.27% predicted. Mean haematocrit level of all patients was 48.49±3.92% wherein male had a mean of 48.97±3.64% and female

had 44.20±3.91% haematocrit. Previous related studies had also measured almost similar haematocrit and FEV1 level among their enrolled COPD patients[5,14]. Chabellan et al found that mean hematocrit of their studied COPD patients was 45.9 ±7.0% in men and 43.9±6.0% in women, where haematocrit was negatively correlated with FEV1% predicted. In this study, haematocrit level was also negatively correlated with FEV1% predicted, wherein mean haematocrit level was significantly higher among very severe COPD patients (52.91±2.41%) compared to moderate and severe COPD (43.83±3.07 and 48.27±2.65%, respectively). Similarly, Hasshim et al. also found a negative correlation between haematocrit level and FEV1% predicted among COPD patients[5]. Besides, Guo et al. observed that FEV1 level was significantly lower among those with elevated haematocrit level compared with those without polycythemia[25]. In addition, Xu et al. stated that high haematocrit level was associated with an increased risk of in-hospital death in the COPD population[26]. In this study, Pearson correlation model showed significant moderate negative association between FEV1 and haematocrit level among COPD patients (R= -0.736, p value <0.001).The causes for high haematocrit level are increase in the total number of red cells in the body due to excessive stimulation of normal erythroid precursors by the physiological regulator, erythropoietin, in states such as chronic hypoxemia (secondary erythrocytosis)[27]. However, El-Korashy et al. showed that almost half of the COPD patients had low haematocrit level, whereas increased haematocrit level found only in the advanced stages of COPD[12].

Limitations of the Study

This was a single center study where sample size was not representative and this study could not evaluate the prognosis value of the dynamics of the hematocrit over time. So, the results may not represent the whole community.

Conclusion

Based on these research findings, mean haematocrit level was significantly higher among very severe COPD patients compared to moderate and severe COPD, which suggest that haematocrit level was significantly associated with severity of COPD. These results correspond with the findings of other similar studies with slight variations. However, further study could give us more information about the changes of hematocrit level in COPD patients, thus helping the clinicians to manage those patients in better way.

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Recommendation

Further larger cohort study is required to get the exact scenario.

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Chest Heart Journal
Vol. 49, No. 1, January 2025

REVIEW ARTICLE

Recent Advancement in Chest Ultrasonography in Pleural Diseases: From Morphological Imaging to Artificial Intelligence Driven Precision Diagnostics

Md. Mahbub Islam[1], Md. Zahirul Islam[2], Golam Sarwar Liaquat Hossain Bhuiyan[2]

Abstract:

Pleural disease comprises a major diagnostic and therapeutic challenge in pulmonology, oncology, critical care, and emergency medicine. Over the past decades, Chest Ultrasonography (CUS) has undergone a paradigm shift from a supplementary bedside tool to a central, decision-making imaging modality in pleural diseases. This evolution has been driven by technological innovations including high-frequency probes, contrast-enhanced ultrasonography (CEUS), elastrography, three-dimensional reconstruction, and artificial intelligence(AI)- alongside standardized scanning protocols and increasing procedural integration. This state-of-the-art review synthesizes contemporary evidence on advanced pleural ultrasonography, focusing on diagnostic accuracy, tissue characterization, procedural optimization, future directions towards precision pleural medicine.

Keywords:Pleural ultrasound, Chest ultrasound, Pleural effusion, Elastrography, Contrast enhanced ultrasound, Point-of-care ultrasound, Artificial intelligence, Radiomics.

[Chest Heart J. 2025; 49(1) : 35-39]

Introduction:

Pleural diseases represent a significant healthcare burden, affecting over 350 000 patients annually in the USA alone and requiring accurate diagnostic approaches for optimal management [1]. The traditional diagnostic modalities rely on chest radiograph (CXR), computed tomography (CT), and invasive pleural procedures. These modalities have significant limitations- radiation exposure, logistic constraints, limited bedside application, and delayed diagnosis. Chest ultrasonography (CUS) has emerged as a game changer in pleural medicine. Once restricted to effusion detection, modern pleural ultrasound now enables morphologic, functional, hemodynamic, and biomechanical assessment of pleural pathology. The integration of CUS into pleural pathways has reshaped diagnostic algorithms, procedural safety standards, and follow-up strategies. This review aims to provide detailing of advanced ultrasound techniques in pleural disease, critically evaluate evidence beyond conventional B-mode imaging, highlight newer technologies including AI and radiomics, and identify knowledge gaps and future research scopes.

Physical and Biophysical Basis of Pleural Ultrasonography

Acoustic Interface of the Pleura

The pleural space represents a unique acoustic environment. The interface between pleura and aerated lung produces characteristic reverberation artifacts, while pleural fluid creates an acoustic

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window permitting visualization of deeper structures[2]. Advances in probe frequency modulation and beamforming algorithms have improved spatial resolution at this interface, allowing detection of submillimeter pleural abnormalities

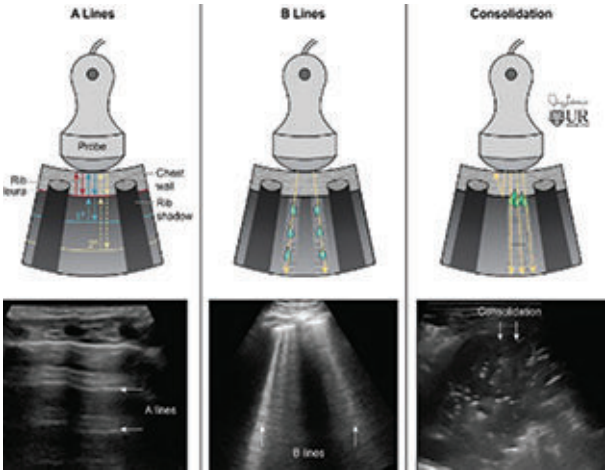

Figure 1: *Physics of lung US. (Left) A-line reverberation artifacts. (Center) B-line artifacts obliterating A-lines. (Right) Consolidation. (Reprinted, with permission, from University of Rochester, Rochester, New York © 2021; medical illustration by Jane Lichorowic)*

High Frequency and Matrix Array Transducers

Modern high- frequency linear probes (10-18 MHz) enable exquisite visualization of pleural line irregularity, sub pleural micro-nodularity, and early fibrin deposition. Matrix array probes further permit volumetric data acquisition, facilitating 3D reconstruction of pleural surfaces[3].

Advanced Morphologic Characterization and Volume Estimation of Pleural Effusions

Traditional classification of pleural effusions into anechoic, complex non-septated, complex septated, and homogenously echogenic patterns[4]has now been refined through quantitative echotexture analysis[5]. Heterogeneity indices derived from grayscale histogram analysis correlate with biochemical exudate marker, microbial burden in empyema, and malignant cellularity.Advanced volumetric models integrate interpleural distance, thoracic curvature correlation, and patient positioning algorithms. Machine-learning-assisted volume estimation shows superior reproducibility compared to conventional formula based approaches, with implications for procedural planning and outcome prediction[6].

Ultrasound in Pleural infection and Empyema

Sonographic Phenotyping of Pleural infection

Chest Ultrasonography enables dynamic phenotyping of para- pneumonic effusions. Fibrin strand density, loculation architecture, and diaphragmatic involvement are main differentiating features [7]. These features predict failure of simple thoracentesis, need for intra pleural fibrinolytic, and surgical referral timing.

Ultrasound-Guided Precision Drainage

Advanced Doppler and CEUS techniques allow identification of viable vs. necrotic pleural tissue, optimal catheter trajectory, and residual loculations post- drainage. CUS- guided empyema management reduces hospital stay and intervention failure compared to CT-guided or blind approaches[8].

Malignant Pleural Disease: From Pattern Recognition to Tissue Characterization:

Sonographic Markers of Malignancy:

Key high-risk ultrasound features include[9]nodular pleural thickening (>10mm), irregular pleural margins, diaphragmatic infiltration, reduced pleural sliding with preserved lung aeration.

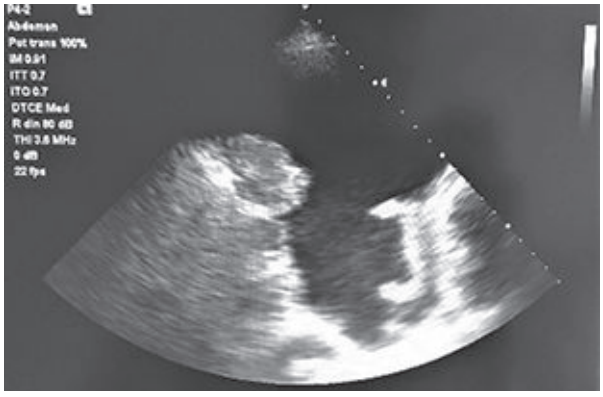

Figure.2: *USG image of pleural effusion and Pleural thickening*

Elastography in Malignant Pleural Disease

Shear-wave elastography quantifies pleural stiffness[10] where malignant pleura demonstrates

significantly higher Young's modulus values and elastrographic heterogeneity correlates with histological aggressiveness. This opens avenues for non-invasive pleural malignancy risk.

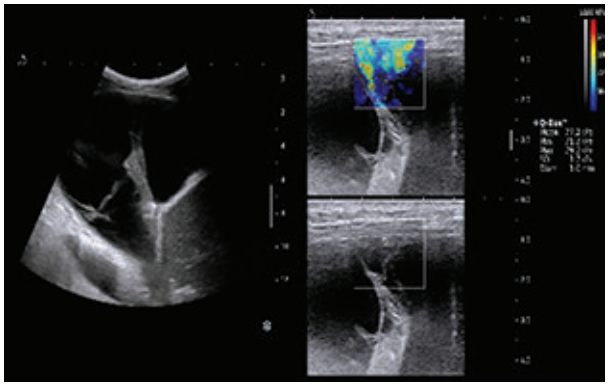

Figure 3: *USG features of pleural nodules, thickening and measurements of pleural stiffness by shear wave elastography*[11].

Contrast Enhanced Ultrasound (CEUS):

CEUS reveals neovascularization pattern, wash-in /wash out kinetics, and tumor associated angiogenesis. These features assist in targeted biopsy site selection, differentiation between malignant vs. benign pleural thickening, monitoring response to pleurodesis or oncologic therapy[12].

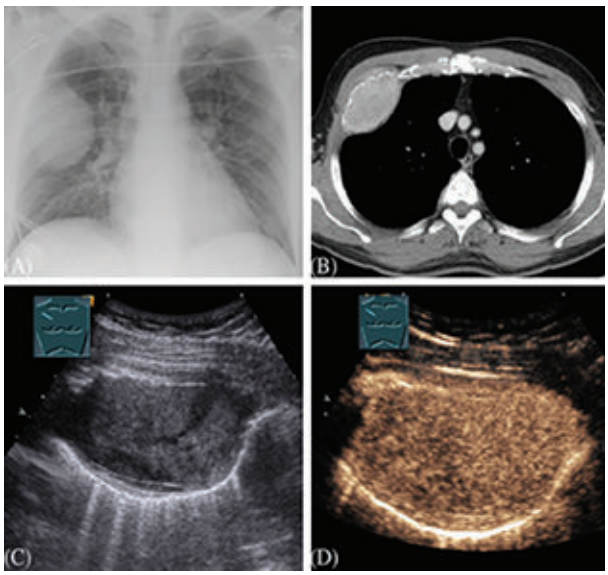

Figure 4: *Contrast-enhanced ultrasound reveals a hyperechoic, homogeneous enhancement of the lesion after 31 s. An ultrasound-guided biopsy was performed, with the diagnosis of plasmocytoma. (Courtesy of Prof. Dr. Mahnken, Department of Radiology, University hospital Marburg)*

Interventional Pleural Ultrasound: A New Standard of Care

Ultrasound guided pleural proceduresinclude thoracentesis, pleural biopsy, intercostal Chest drain insertion, and indwelling pleural catheter placement. Studies [13]demonstrate 50% reduction in pneumothorax risk, lower hemorrhagic complications, and improved success rate compared to blind approaches [14].Pre-thoracoscopy ultrasound mapping diminishes port site infection, avoidance of adhesions, diagnostic yield. Real time ultrasound guidance during thoracoscopy is an emerging technique under study [1516].

Artificial Intelligence and Radiomics in Pleural Ultrasound

AI models trained on pleural ultrasound datasets achieve high accuracy in effusion detection, automated B-line quantification, real-time pleural line abnormality detection. Extraction of high-dimensional ultrasound features enables prediction of pleural malignancy, differentiation of tuberculous vs. malignant effusion, and outcome prediction following pleural interventions [17]. Integration of radiomics with clinical and biochemical data represents a major step toward precision pleural medicine.

Pleural USG in Critical Care and Emergency Medicine

CUS has become essential in acute respiratory failure algorithms, shock differentiation, rapid pneumothorax detection[18].

Dynamic pleural assessment enables continuous bedside monitoring which is not possible with static imaging modalities.

Training, Credentialing, and Global implementation

Despite its advantages, barriers remain including operator dependency, variability in training standards, and limited access to advanced technology in resource-constrained settings. Societies now advocate for competency based certification, structured pleural ultrasound curriculum, and tele-mentoring and cloud based image review.

Limitations and Pitfalls

Key challenges include limited penetration for mediastinal pleura, overlap of benign and malignant features, learning curve for advanced modalities,

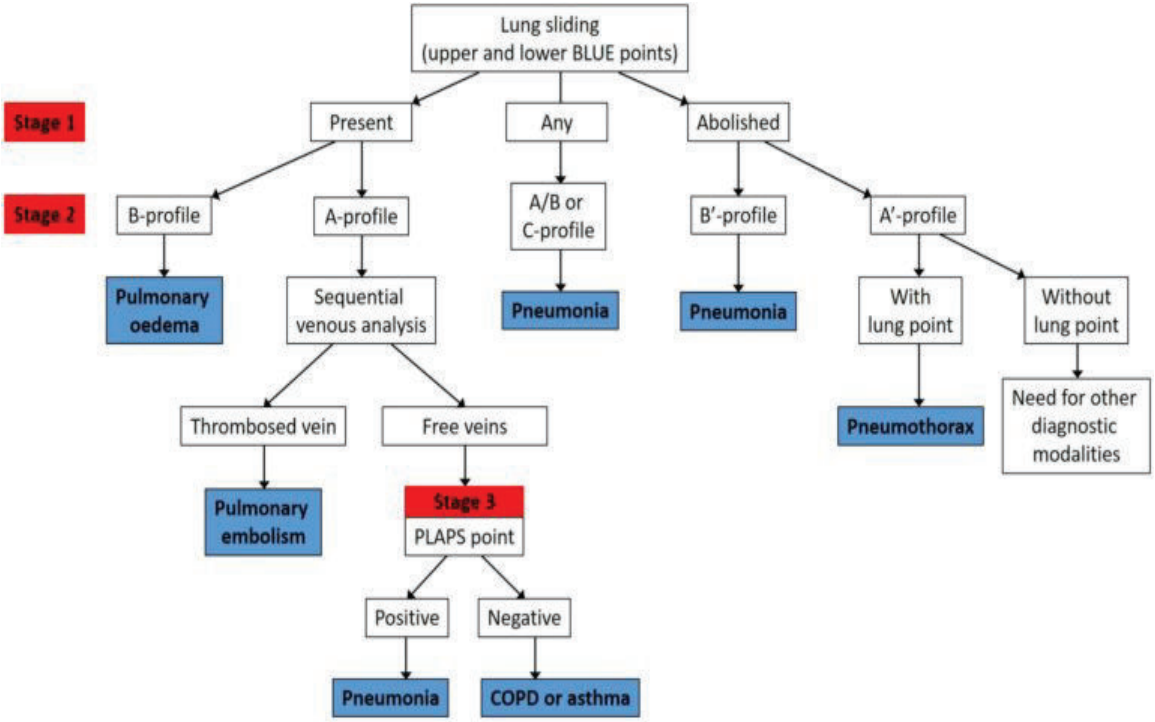

Figure 5: *Schematic of the BLUE protocol*

and lack of universal reference standards. These reinforce the need for multimodal integration, not replacement of histology or CT.

Future Directions and Research Agenda

Key future priorities include large multicenter trials validating elastrography and CEUS, AI-driven decision support systems, standardization of pleural ultrasound reporting, integration with molecular pleural diagnostics, and development of portable high-resolution devices.

Conclusion

Chest Ultrasonography has become a high resolution, multi parametric, and interventional imaging platform central to modern pleural disease management. Advances are ongoing in the field of technology and analytics which has transformed pleural ultrasound a descriptive modality into a quantitative, predictive, and precision guided tool. Continued innovation, rigorous validation, and standardized training will determine its ultimate impact on global pleural disease care.

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CASE REPORT

The Missing Lung: A 33-Year-Old's Battle with Congenital Agenesis and Cor Pulmonale

Md. Hamza[1], Raj Kiron Saha[2], Tazrin Farhana[3], Sarder Mohammed Golam Sarwor[4], Kazi Fawzia Afreen[5], Habib Uddin Ahmed[6]

Abstract

Unilateral lung agenesis is a rare congenital disorder, often diagnosed in childhood due to recurrent infections. We present a 33-year-old male with left lung agenesis, manifesting as progressive dyspnea, pedal edema, and severe pulmonary hypertension (PH). Clinical examination revealed absent left lung sounds, mediastinal shift, and signs of right heart failure. Imaging confirmed left lung agenesis, while echocardiography demonstrated severe PH (PASP 110 mmHg). Management included oxygen therapy, diuretics, and antibiotics for superimposed infection. This case underscores the importance of early recognition and multidisciplinary management in congenital lung anomalies to prevent PH and cor pulmonale.

[Chest Heart J. 2025; 49(1) : 40-43]

Case Presentation

A 33-year-old male, with no history of consanguinity or familial congenital disorders, presented with progressive exertional dyspnea (NYHA class III) for seven months and bilateral pedal edema for two months. He reported recurrent chest infections since childhood but had no prior cardiopulmonary evaluation. On admission, he was dyspneic (SpO, 90% on room air), tachycardic (HR 104 bpm), and hypotensive (BP 90/60 mmHg). Physical examination revealed elevated jugular venous pressure (JVP), a loud P, , palpable P, , and left parasternal heave. Respiratory findings included left-sided chest flattening, absent breath sounds, and dull percussion on the left hemithorax, with tracheal and mediastinal shift to the left.

Investigations confirmed the diagnosis. Chest X-ray showed complete left hemithorax opacification with rib crowding and compensatory right lung hyperinflation. CT chest revealed left lung agenesis, mediastinal shift, and right lower lobe ground-glass opacities with bronchiectasis. Fiberoptic bronchoscopy confirmed the absence of the left bronchial tree. ECG demonstrated P pulmonale and right ventricular hypertrophy (RVH) with strain, while echocardiography estimated a pulmonary artery systolic pressure (PASP) of 110 mmHg with preserved left ventricular function (LVEF 62%). Sputum culture grew *Klebsiella pneumoniae*, sensitive to ceftriaxone.

The patient was initiated on long-term oxygen therapy (LTOT), intravenous antibiotics (empirical

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followed by culture-directed), diuretics, and pulmonary vasodilators (Ambrisentan 5 mg/day) for severe PH. After 3 months of follow-up, his symptoms showed modest improvement: SpO, 94% on 2 L/min LTOT, reduced JVP elevation, and mild resolution of bipedal edema. Repeat echocardiography demonstrated a decline in PASP to 87 mmHg, though right heart strain persisted. He was advised to continue current therapy with close cardiopulmonary monitoring.

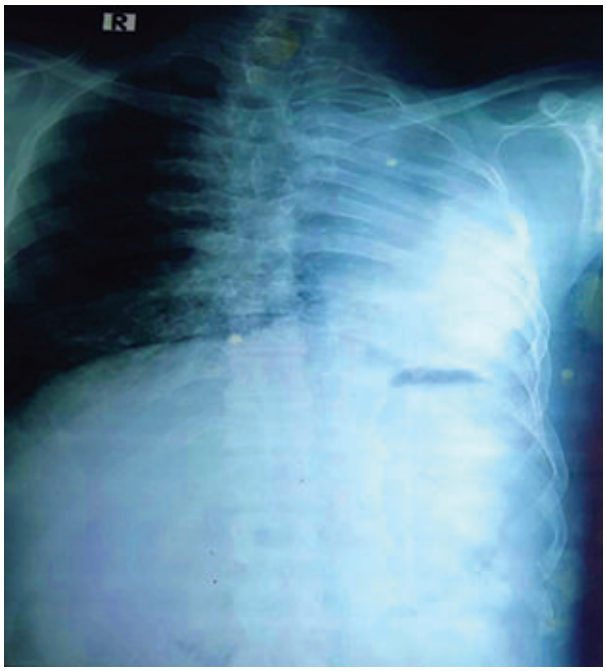
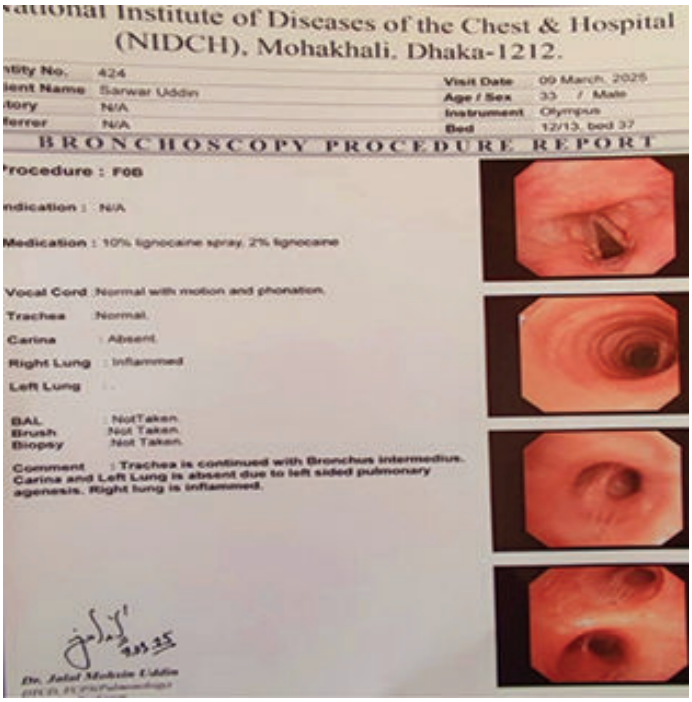


Figure 1: *Chest X-ray demonstrating left hemithorax opacification, rib crowding, and right lung hyperinflation.*

Figure 2: *CT scan revealing left lung agenesis, mediastinal shift, and right lower lobe Ground glass opacity."*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BRONCHOSCOPY PROCEDURE REPORT

Procedure : FOB

Indication : N/A

Medication : 10% lignocaine spray, 2% lignocaine

Vocal Cord : Normal with motion and phonation.

Trachea : Normal.

Carina : Absent.

Right Lung : Inflammed

Left Lung :

BAL : NotTaken.
Brush : Not Taken.
Biopsy : Not Taken.

Comment : Trachea is continued with Bronchus intermedius. Carina and Left Lung is absent due to left sided pulmonary agenesis. Right lung is inflammed.

Dr. Jalal Mohsin Uddin

Figure 3: *Bronchoscopic view confirming left bronchial agenesis.*

Discussion

Unilateral lung agenesis is an exceedingly rare congenital anomaly resulting from the complete absence of lung parenchyma, bronchi, and vasculature on one side. Left-sided agenesis, as seen in this case, is slightly more common than right-sided agenesis and is often associated with a better prognosis due to less severe mediastinal shift and cardiac compression.[1] However, the remaining lung undergoes compensatory hyperinflation, increasing susceptibility to recurrent infections, bronchiectasis, and progressive respiratory insufficiency.[2] In our patient, chronic hypoxia due to reduced pulmonary vascular bed and recurrent infections likely contributed to the development of severe pulmonary hypertension (PH), a well-documented complication in such cases.[3]

The pathophysiology of PH in lung agenesis involves multiple mechanisms. The absence of pulmonary vasculature in the affected side increases blood flow to the contralateral lung, leading to endothelial dysfunction, vascular remodeling, and elevated pulmonary vascular resistance.t Chronic hypoxia further exacerbates PH by inducing vasoconstriction and vascular smooth muscle proliferation.u Over time, this results in right ventricular hypertrophy (RVH), right heart failure, and cor-pulmonale, as evidenced by our patient's echocardiographic findings (PASP 110 mmHg) and clinical signs of right-sided congestion (elevated JVP, pedal edema).v

Diagnosis of lung agenesis requires a high index of suspicion, particularly in adults with a history of recurrent respiratory infections and unexplained dyspnea. Imaging plays a crucial role, with chest X-ray revealing mediastinal shift and hemithorax opacification, while CT provides definitive confirmation of absent lung tissue.w Fiberoptic bronchoscopy is useful to rule out bronchial atresia or other obstructive pathologies.x In our case, echocardiography was pivotal in assessing PH severity and right heart function, though right heart catheterization remains the gold standard for PH diagnosis.y

Management of lung agenesis with PH is primarily supportive. Long-term oxygen therapy (LTOT) is essential to alleviate hypoxia and reduce pulmonary vasoconstriction.[1]p Diuretics are used for volume overload, while pulmonary vasodilators (e.g., phosphodiesterase-5 inhibitors, endothelin receptor antagonists) may be considered in selective cases, though evidence in congenital lung anomalies is limited.[11] Infection prevention through vaccinations and prompt antibiotic therapy for respiratory infections is critical to prevent further lung damage. In advanced cases, lung transplantation may be the only definitive treatment, though comorbidities such as severe PH and right heart failure pose significant challenges.[12]

This case highlights the delayed presentation of lung agenesis and underscores the importance of early recognition and multidisciplinary management. Regular follow-up with cardiopulmonary assessment, PH monitoring, and preventive care are vital to improving long-term outcomes in such patients.

Conclusion

Unilateral lung agenesis, though rare, can lead to severe complications such as pulmonary hypertension and cor pulmonale in adulthood. A high clinical suspicion, combined with imaging and hemodynamic assessment, is essential for diagnosis. Multidisciplinary management, including oxygen therapy, diuretics, and infection control, remains the cornerstone of treatment. Early intervention may mitigate disease progression and improve quality of life.

Consent

Verbal consent has been taken from the patient regarding publication of this report.

Conflict of Interest

No conflict of interest was declared by the authors.

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CASE REPORT

A Case Report of a less Anticipated Presentation : SVCO in Adenocarcinoma Lung

Syeda Tohara Tuz Alam[1], Shibly Noman[2], Azmain Mahtab[2], Nabo Kanto Roy[2], Pallob Saha[3], Mehedi Hasan[3], Zarin Tasnim[4], Rubayat Rahman[4], Mohammed Mirazur Rahman[5]

Abstract:

*Superior vena cava obstruction (SVCO) can cause significant mortality and morbidity. When complicated by stridor it becomes a medical emergency. Among the Non-small cell lung cancer (NSCLC), squamous cell cancer presents most commonly due to its central location, while adenocarcinoma stays as a rare cause of SVCO.We present this case of a 38 years old Bangladeshi male smoker patient who has been diagnosed as a case of adenocarcinoma lung following a history of pain in right chest, arm,hand with swelling of face and neck for 1 month.Following admission,meticulous regular physical examination revealed a right supraclavicular lymph node measuring 2*2 cm that played a vital role in reaching the diagnosis. His CECT scan of chest showed a mass in right upper lobe merging with mediastinum compressing SVC. FNAC from the supraclavicular lymph node revealed metastatic adenocarcinoma.*

Keywords: *adenocarcinoma, lung mass, Superior vena cava obstruction.*

[Chest Heart J. 2025; 49(1) : 44-46]

Introduction

To date,70% of SVCO occurs due to malignancies[1], NSCLC being the commonest cause (50%).An obstruction to SVC causes SVCO syndrome, when malignant it can occur due to compression of tumor itself or by enlarged mediastinal lymph node or by direct invasion of tumor in SVC. It is categorized into 4 domains. These are- 1.neurological (headache, blurred vision, papilloedema, altered consciousness), 2.laryngopharyngeal(cough, tongue swelling, dyspnea, stridor), 3.facial(nasal stuffiness, conjunctival edema, periorbital plethora) 4. Venous distension (dilated capillaries at upper extremities) [3].About 2-4% of lung cancer patients develop SVCO at any point of their clinical course[4].Early presentation of SVCO can be misleading if other examinations and investigations are inconclusive which our case all about is. However any presentation of SVCO warrants prompt diagnosis and management.Here we present a case of SVCO occurring due to lung adenocarcinoma.

Case description

This 38-year-old normotensive businessman from a low socioeconomic condition presented to us with the complaints of cough for last 3 months, pain in right side of chest, shoulder and arms for 1 month and swelling of face and neck for 1 week. He is an active smoker of 20 pack year. For last 3 months,

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he has been experiencing cough mostly dry without any sputum or hemoptysis.He is also having pain in his right upper chest, shoulder, arms which is severe, dull in nature, persistent, relieves slightly by analgesics. His swelling of face and neck is not associated with any headache, altered consciousness, high grade fever with night sweat, any loss of weight. Upon clinical examination, upper chest, neck and face appears plethoric with some dilated superficial veins directed above downwards(Fig-1), respiratory rate 20/min, BP-120/70mm hg, a 2*2 cm right supraclavicular lymph node which is nontender, firm to hard, mobile, auscultation of lungs- reduced breath sound with reduced vocal resonance in right upper and part of mid zone.

Figure 1: *Patient's swollen face and neck with superficial venous engorgement*

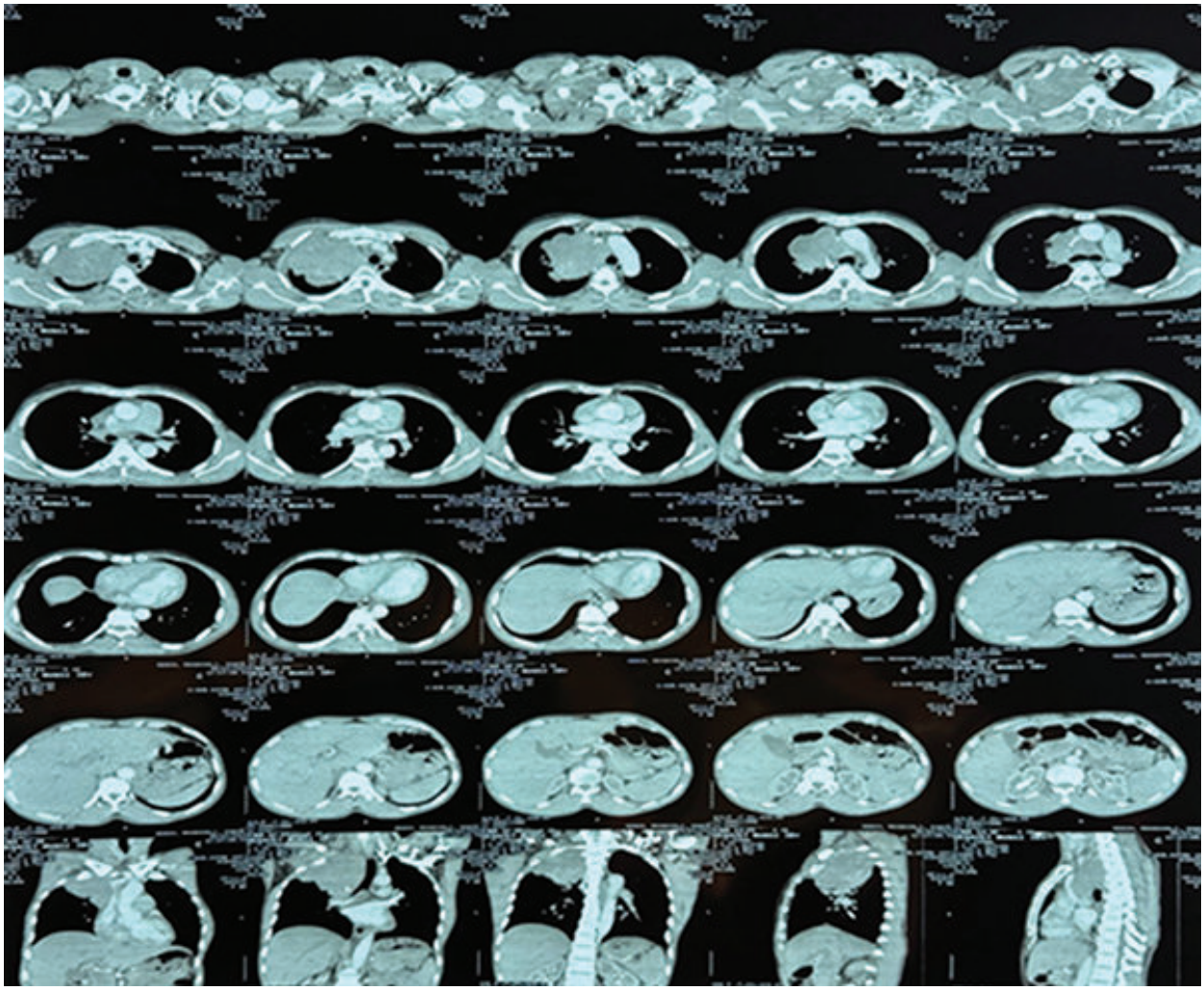

Figure 2: *CECT scan Chest showing right lung mass merging with mediastinum and compressing SVC.*

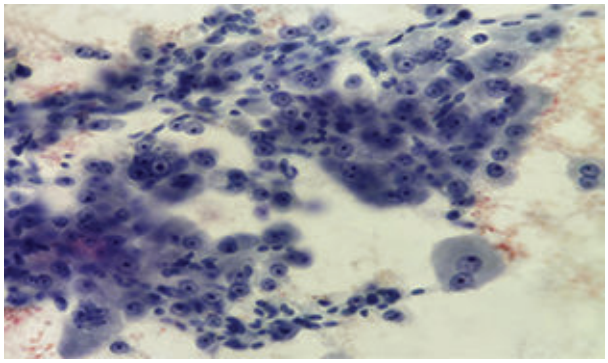

Figure 3: *Stained cytology slide showing atypical polygonal,round to oval cells in cluster and singly and multinucleation present.*

All his routine investigations appeared normal. CXR P/A view showed an ill-homogeneous opacity in right upper and midzone, USG of chest showed mixed echoic lesion with central hyper-echogenicity. CECT scan of chest showed mass lesion in right upper lobe merged with mediastinum and mediastinal lymphadenopathy encasing and compressing SVC(Fig:2). FOB was done which showed normal endobronchial tree.His CT-guided FNAC of the lung showed a lung abscess and FNAC from right supraclavicular lymph node that had become palpable after admission revealed metastatic adenocarcinoma(Fig:3). Then we referred the patient to cancer hospital for better and specific management.

Discussion

An intrathoracic malignancy is responsible for 60 to 85 percent of cases of SVC syndrome, and SVC obstruction is the presenting symptom of a previously undiagnosed tumor in up to 60 percent of these cases[4]. Non-small cell lung cancer (NSCLC) is the most common malignant cause of SVC syndrome, accounting for approximately 50 percent of all cases[4], followed by small cell lung cancer (SCLC; approximately 25 to 35 percent of all cases) and non-Hodgkin lymphoma (NHL; 10 to 15 percent of cases). SVCO due to adenocarcinoma of lung is very rare. That's why we presented this case.Symptoms and signs of thoracic central venous obstruction can include swelling, chest pain, respiratory symptoms, or neurologic manifestations[5].Regardless of etiology, face or neck swelling and dyspnea are common presenting symptoms.Our patient presented with the same symptoms, but he was young. His CT-guided FNAC of the lung showed a lung abscess; therefore, we performed a USG-guided FNAC from the right supraclavicular lymph node. From this, we confirmed the diagnosis.The best choice of treatment depends on the underlying type of cancer, extent of disease, and overall prognosis, which is closely linked to histology and whether prior therapy has been administered[6].An endovenous stent is particularly appropriate for rapid symptom palliation in patients with tumors for which response to chemotherapy and/or RT is intermediate or poor.SVC obstruction is a strong predictor of poor prognosis in patients with NSCLC, with a median survival of only five months in one series[7].We referred this patient to the National Institute of Cancer Research & Hospital (NICRH) for better and more appropriate management.

Conclusion

Adenocarcinoma is a rare malignancy associated with SVCO.US guided FNAC and ROSE give favorable outcomes with less invasiveness. Management of SVCO needs multidisciplinary approach but endeavor should be to find out primary cause of SVCO rather than going for urgent radiotherapy or chemotherapy without proper evaluation.

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INSTRUCTION TO AUTHORS ABOUT UNIFORM MANUSCRIPT WRITING

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Cancer in South Africa (editorial). S Afr Med J 1994; 84-15.

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The Cardiac Society of Australia and New Zealand. Clinical exercise stress training. Safety and performance guideline. Med J Aust 1996; 164 : 282-4.

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