ORIGINAL ARTICLE

ROLE OF LUNG ULTRASOUND DURING WEANING IN PATIENT WITH MITRAL REGURGITATION POST ANGIOPLASTY

Mujtaba Hassan, Muhammad Imran Ansari, Jawed Abubaker, Madiha Umair, Komal Baloch, Musa Karim, Nawal Salahuddin

National Institute of Cardiovascular Diseases (NICVD), Karachi-Pakistan

Background: We hypothesize that a change in lung ultrasound score (LUS) can assist in the early diagnosis of weaning-induced respiratory failure (RF). The objective of this study was to determine the utility of LUS in weaning patients with mitral regurgitation (MR) from mechanical ventilation (MV). **Methods:** This prospective observational study included patients with acute coronary syndrome (ACS) who required invasive MV after angiography/angioplasty. Echocardiography was performed and MR was recorded. When the patient was considered ready for extubation, a spontaneous breathing trial (SBT) was performed and pre- and post-SBT LUS was calculated. Patients who successfully passed the SBT were extubated and followed up for 48 hours for the signs of RF and outcomes. **Results**: We enrolled 215 patients, out of which MR occurred in 51(23.7%) patients. On post-SBT lung ultrasound, patients with MR were more likely to have B2 lines compared to those without MR; 15.7% vs. 3.7%; p=0.002 and mean LUS was significantly higher for patients with MR as compared to patients without MR; 2.75±3.21 vs. 1.37± 2.02; p<0.001. Post-extubation RF and mean CCU stay were significantly higher in MR patients, 49.0% (25) vs. 32.3% (53); p=0.030 and 3.53±1.54 days vs. 2.41±1.1 days; p<0.001 respectively. However, re-intubation and coronary care unit (CCU) mortality rate were not significantly different between patients with and without MR; 7.8% (4/51) vs. 3.7% (6/164); p=0.215, and 5.9% (3/51) vs. 3% (5/164); p=0.35 respectively. **Conclusions:** Bedside LU is a convenient tool to detect changes in cardiopulmonary interactions during weaning for patients with MR post-ACS.

Keywords: Acute coronary syndrome; Mechanical ventilation; Mitral regurgitation; Lung ultrasound; Spontaneous breathing trial; Respiratory failure

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INTRODUCTION

Mitral regurgitation (MR) is a common sequela of left ventricular pathological remodelling with chronic coronary artery disease (CAD).[1] MR is defined as mitral regurgitation caused by changes of LV structure and function due to ischemic heart disease, the underlying process is primarily non valvular and represents the valvular consequences of increased tethering forces and reduced closing forces.[1] MR is reported in approximately one-fifth of patients following AMI and one-half of those with congestive heart failure and associated with poor prognosis.[2,3] Weaning failure is encountered in approximately 42% of mechanically ventilated patients, and is independently associated with poor outcomes.[4] In patients intubated with acute myocardial infarction (AMI), weaning and liberation can be especially challenging when stunned myocardium and ischemic mitral valve regurgitation occur.[5–8] Weaning-induced pulmonary oedema (WIPO) is well recognized complication occurring during a spontaneous breathing trial (SBT) in patients after acute coronary syndrome, depicting worsening cardiac loading conditions due to heart–lung interactions.[9,10] MR becomes clinically significant after the cardioprotective effects of mechanical ventilation are removed, which leads to cardiogenic pulmonary oedema and respiratory failure.[10] Timely detection of WIPO allows bedside clinicians to recognize opportunities to intervene and reduce risk of extubation failure.

Detection of WIPO can be by measuring changes in left ventricular end-diastolic pressures, pulmonary artery occlusion pressure and other hemodynamic indices, but they require invasive monitoring with its own complications.[11,12] Measuring cardiac biomarkers like BNP and NT-BNP is helpful but requires a lab run time of 18–20 minutes.[13] Transthoracic echocardiography can accurately detect changes in hemodynamic induced by SBT and identify patients at high risk of cardiac-related weaning failure. This requires training in critical care echocardiography, because image acquisition and interpretation is frequently challenging.[14,15] Bedside lung ultrasound in critically ill patients is a non-invasive tool proven to be effective for the diagnosis of cardiogenic pulmonary oedema.[16,17] We hypothesize that a change in lung ultrasound score can assist the bedside clinician in the early diagnosis of weaning-induced pulmonary oedema in this high-risk patient group.

The objective of this study was to determine the utility of lung ultrasound in weaning patients with ischemic MR from mechanical ventilation.

MATERIAL AND METHODS

This prospective, observational study was conducted at the coronary care unit (CCU) of a tertiary care cardiac center. The study period was from November 2020 to May 2021. The study included patients with acute coronary syndrome who required at least 24-hours of invasive mechanical ventilation (MV) after angiography/angioplasty.

This protocol was reviewed and approved by the ethical review committee of the institution. (ERC approval number: ERC-61/2020). Consent for participation and publication was obtained from the patients' legal caretaker. Patients placed on non-invasive ventilation (NIV) for post extubation stridor and patients with "do not intubate" and/or "do not resuscitate" order at the time of extubation were also not included. Patients were evaluated for liberation from ventilator based on hemodynamic and metabolic profile, ventilatory requirements, and clinical condition as per our approved ICU protocol. When the patient was considered ready for extubation, a SBT was performed by a T-piece trial for 30 minutes using a standard ICU protocol. The decision to stop SBT was made by the primary intensivist. The patient was rested for a minimum of 30 minutes to prevent exhaustion by weaning efforts.[18] Hemodynamic and biological variables recorded before and at the end of SBT were respiratory rate (bpm), pulse oxygen saturation (%), heart rate (bpm), arterial pressure (mmHg), arterial blood gas (ABG) analysis, and haemoglobin concentration. Pre- and post-SBT bedside transthoracic echocardiography (TTE) was performed and ischemic mitral regurgitation (IMR) was noted along with left ventricular ejection fraction (%) and diastolic dysfunction. Pre- and post-SBT lung ultrasound was performed in all the patients and the total Lung Ultrasound Score (LUS) was calculated was calculated as per the criteria described by Soummer *et al.*[19]

Patients who successfully passed the SBT were extubated and followed up for 48 hours by the team for signs of respiratory failure. Requirement of NIV was assessed by the assigned ICU Intensivist based on following signs of respiratory failure.1) Hypoxemic respiratory failure; room air saturation <90%, PaO2 <60 mmHg, an increase in FiO2 requirement, or decrease in PaO2 >30 mmHg from baseline if the patient was on oxygen. 2) Hypercarbic respiratory failure; if PaCO2 >45 mmHg and pH<7.30. 3) A more than 4-point increase in the base deficit over an hour observation period from the baseline level. 4) Tachypnoea; respiratory distress (using accessory muscles of respiration) or respiratory rate more than 30 bpm. Vital signs, pulse oximetry was continuously monitored for all patients and re-intubation and length of stay was recorded. Patients were categorized into two groups based on presence of moderate- severe MR as diagnosed on TTE. Study results are summarized as mean ± standard deviation or frequency (%) as appropriate. Demographic, clinical, hemodynamic, and metabolic variables along with the primary outcome (post-extubation RF, re-intubation, and length of CCU stay) were compared between the two groups using the Independent Sample T-test or Chi-square test. A two-sided *p*-value ≤0.05 was considered statistically significant. Statistical analysis was performed using IBM SPSS version 21.0.

RESULTS

We enrolled 215 ACS patients, out of which ischemic mitral regurgitation occurred in 51 (23.7%) patients. Majority (69.8%) were male and the mean age was 60.5 (±11.37) years. Assessment of co-morbid conditions revelled, 151 (70.2%) patients were hypertensive and 89 (41.4%) were on vasopressor support. A total of 213 (99%) patients underwent revascularization with angioplasty. Mean LV systolic dimensions (39.41±8.38 mm vs. 35.02±6.73 mm; *p*<0.001) and mean RV dimensions (21.49±5.34 mm vs. 20.12±3.14 mm; *p*=0.024) were significantly higher in patients with MR as compared to non-MR patients respectively. Biventricular dysfunction (27.5% vs. 15.2%; *p*=0.048) was more common in MR group as compared to non-MR groups respectively (Table-1).

On lung ultrasound, patients with MR were more likely to have B2 lines compared to those without MR; 11.8% vs. 3.0%; *p*=0.014 at baseline and after SBT; 15.7% vs. 3.7%; *p*=0.002. Similarly, LUS, both pre- and post-SBT, were also significantly higher for patients with MR as compared to patients without MR; 2.16±2.77 vs. 1.0±1.73; p<0.001 and 2.75±3.21 vs. 1.37±2.02; *p*<0.001 for post-SBT respectively. Among other hemodynamic and metabolic parameters, post-SBT heart rate was found to be significantly higher in the MR group 100.4±13.9 bpm vs. 95.8±14.4; *p*=0.046 respectively (Table-2).

Post-extubation respiratory failure (RF) was significantly higher in MR patients 49.0% (25) vs. 32.3% (53); *p*=0.030 respectively. Mean duration of CCU stay was significantly higher for patients with MR; 3.53±1.54 days vs. 2.41±1.1 days; *p*<0.001. Re-intubation and CCU mortality rate were not significantly different between patients with MR patients compared to patients without MR; 7.8% (4/51) vs. 3.7% (6/164); *p*=0.215, and 5.9% (3/51) vs. 3% (5/164); *p*=0.35 respectively (Figure-1).

Table-1: Baseline clinical characteristics, echocardiogram, and ventilator settings stratified by the presence of mitral regurgitation

Characteristics	Mitral Regurgitation		*p*-value
	No	Yes	
Total (N)	164 (76.3%)	51 (23.7%)	-
Male	122 (74.4%)	28 (54.9%)	0.008*
Female	42 (25.6%)	23 (45.1%)	
Age (years)	60.82 (±11.07)	59.73 (±12.34)	0.548
22 to 50 years	29 (17.7%)	12 (23.5%)	0.636
51 to 65 years	84 (51.2%)	25 (49%)	
> 65 years	51 (31.1%)	14 (27.5%)	
Co-morbid conditions			
Hypertensive	117 (71.3%)	34 (66.7%)	0.524
Diabetes mellitus	24 (14.6%)	6 (11.8%)	0.605
Coronary artery disease	26 (15.9%)	12 (23.5%)	0.209
Chronic obstructive pulmonary disease	24 (14.6%)	6 (11.8%)	0.605
Chronic kidney disease	14 (8.5%)	4 (7.8%)	0.876
Arrhythmias	37 (22.6%)	9 (17.6%)	0.455
Vasopressors	70 (42.7%)	19 (37.3%)	0.492
20 to 30%	69 (42.1%)	26 (51%)	0.502
30 to 45%	85 (51.8%)	23 (45.1%)	
>45%	10 (6.1%)	2 (3.9%)	
Ventricular septal rupture	2 (1.2%)	2 (3.9%)	0.212
LV dysfunction	148 (90.2%)	50 (98%)	0.072
RV dysfunction (mm)	30 (18.3%)	14 (27.5%)	0.157
LV dimension: systolic	35.02 (±6.73)	39.41 (±8.38)	<0.001*
LV dimension: diastolic	46.34 (±7.02)	47.96 (±8.13)	0.168
RV dimension	20.12 (±3.14)	21.49 (±5.34)	0.024*
Biventricular dysfunction	25 (15.2%)	14 (27.5%)	0.048*
PF Ratio	338.02 (±109.04)	347.16 (±105.81)	0.599
≤300	78 (47.6%)	23 (45.1%)	0.758
>300	86 (52.4%)	28 (54.9%)	
Oxygen saturation (SO_2)	97.81 (±6.21)	97.31 (±8.38)	0.648
FiO_2	40.06 (±7.28)	40.33 (±8.77)	0.825

*Significant at 5%. RV= right ventricle, LV = left ventricle, COPD = chronic obstructive pulmonary disease, CXR = chest X-ray, CPR = cardiopulmonary resuscitation, PEEP = positive end expiratory pressure, PSV = pressure support ventilation, FiO2 = fraction inspired oxygen

Table-2: Comparison of pre- and post-spontaneous breathing trial lung ultrasound score, hemodynamic, and metabolic parameters for patients with and without ischemic mitral regurgitation

Characteristics	Mitral Regurgitation		*p*-value
	No	Yes	
Total (N)	**164 (76.3%)**	**51 (23.7%)**	**-**
Lung ultrasound score before SBT			
B1 lines	49 (29.9%)	22 (43.1%)	0.079
B2 lines	5 (3%)	6 (11.8%)	0.014*
Consolidation	0 (0%)	1 (2%)	0.072
Normal	110 (67.1%)	22 (43.1%)	0.002*
Lung ultrasound score at end of SBT			
B1 lines	59 (36%)	22 (43.1%)	0.357
B2 lines	6 (3.7%)	8 (15.7%)	0.002*
Consolidation	0 (0%)	1 (2%)	0.072
Normal	99 (60.4%)	20 (39.2%)	0.008*
Lung Ultrasound Score (LUS)			
Pre-SBT	1 (±1.73)	2.16 (±2.77)	<0.001*
Post- SBT	1.37 (±2.02)	2.75 (±3.21)	<0.001*
Heart Rate (HR)			
Pre- SBT	95.6 (±17.05)	99 (±13.72)	0.196
Post- SBT	95.77 (±14.38)	100.37 (±13.87)	0.046*
Systolic Blood Pressure (SBP)			
Pre- SBT	124.88 (±18.94)	122.59 (±16.1)	0.435
Post- SBT	123.12 (±17.09)	123.94 (±17.99)	0.766
pH			
Pre- SBT	7.44 (±0.06)	7.44 (±0.06)	0.381
Post- SBT	7.39 (±0.39)	7.42 (±0.04)	0.632
Base deficit (BE)			
Pre- SBT	-0.17 (±3.5)	0.35 (±4.47)	0.384
Post- SBT	-0.13 (±3.37)	1.01 (±3.42)	0.038*
Carbon Dioxide (CO_2) gap			
Pre- SBT	8.96 (±4.69)	8.81 (±3.6)	0.837
Post- SBT	8.66 (±4.45)	8.1 (±3.41)	0.404
Mixed Venous Oxygen Concentration (ScvO2)			
Pre- SBT	66.7 (±11.57)	65.38 (±8.74)	0.455
Post- SBT	67.54 (±9.86)	66.69 (±10.45)	0.599
Carbon Dioxide (CO_2)			
Pre- SBT	34.8 (±5.47)	34.49 (±5.72)	0.726
Post- SBT	37.28 (±6.49)	38.33 (±6.5)	0.314

*Significant at 5%. SBT= post-spontaneous breathing trial

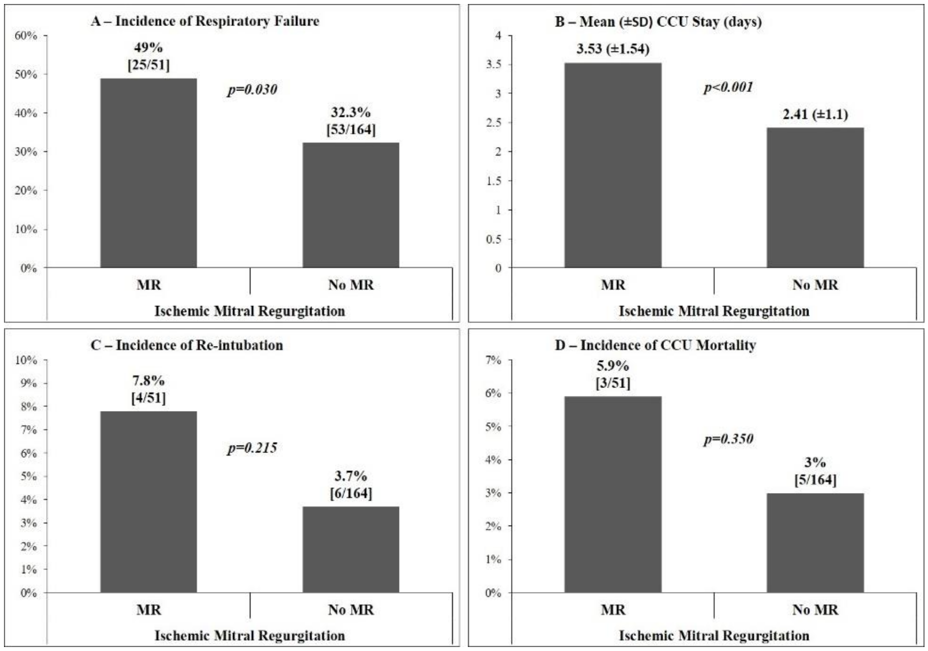

Figure-1: Respiratory failure, CCU stay, re-intubation, and CCU mortality rate stratified by the presence or absence of ischemic mitral regurgitation

DISCUSSION

Our study found that in patients after acute coronary syndrome, lung ultrasound is a useful bedside tool to assist in weaning patients from mechanical ventilation. We found significant increases in the LUS in patients with post-ACS MR before and after a SBT. This increase in the lung ultrasound score after the 'stress test' of a breathing trial depicts the development of pulmonary oedema and can therefore assist the clinician to avert post-extubation cardiorespiratory failure.

Mitral regurgitation (MR) may become clinically significant after the cardio protective effects of mechanical ventilation are removed. This may lead to cardiogenic pulmonary oedema and respiratory failure.[20,21] Ruiz-Bailén M *et al*. looked at worsening mitral regurgitation during stress echo in 37 mechanically ventilated patients who failed SBT. However, echocardiography requires expertise and requires technical expertise.[22] Rasanen J *et al*. described hemodynamic changes and increased work of breathing during SBT associated with MR.[23]

Lung ultrasound is increasingly used as a bedside tool to identify increased extravascular lung water. Alexis Ferré *et al*. found that among the 33 cases with spontaneous breathing trial failure, lung ultrasound diagnosed pulmonary oedema with a sensitivity of 88% (64–98) and a specificity of 88% (62–98) with AUC of 0.91 (0.75–0.98).[16] Soummer A *et al*.[19] reported that in a cohort of 100 critically ill, mechanically ventilated patients, a LUS after a spontaneous breathing trial was found to be highly predictive of post extubation distress with a positive likelihood ratio of 11.8.These studies were in medical ICU patients, in comparison with our data which is in patients with post-ACS MR.

Lung ultrasound is easier to learn and perform compared to echocardiography. It provides a non-invasive and real time assessment of extravascular lung water by detecting the early phase of pulmonary oedema. To the best of our knowledge this is the first time a study was conducted to look at the utility of Bedside ultrasound pre and post SBT specifically in patients with post-ACS MR. Our study demonstrates lung ultrasound as a useful monitoring tool during SBT, as cardiac decompensation and pulmonary oedema can become evident during stress. The results of this study could be generalized to other critically ill patients who fail weaning with occult cardiovascular disease[22] or latent heart failure. Our study identified through lung ultrasound that patients apparently clinically stable enough to be extubated, were at higher risk of extubation failure. In the light of our results further studies should be conducted to detect and subsequently optimize patients undergoing weaning related cardiopulmonary dysfunction.

The strengths of our study are that we looked at a hitherto unstudied population (post-ACS MR), that the ICU team was blinded to the lung ultrasound results and therefore could not have affected the outcomes. Our limitations are that this is relatively small, single center study.

CONCLUSION

Bedside Lung ultrasound is a convenient tool to detect changes in cardiopulmonary interactions during weaning for patients with mitral regurgitation post-acute myocardial infraction. Larger trials are needed to further explore its potential benefit for this cohort.

AUTHORS' CONTRIBUTION

HM, MIA, JA, MK, and NS contributed to the concept and design of study, HM, MIA, MU, and KB contributed to the collection, analysis and interpretation of data, HM, MIA, MU, KB, and MK contributed to the drafting of the manuscript, and JA and NS critically analyzed for content. All authors have read and approved the manuscript

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Address for Correspondence:

Mujtaba Hassan, National Institute of Cardiovascular Diseases (NICVD), Karachi, Pakistan
Rafiqui (H.J.) Shaheed Road, Karachi- 75510-Pakistan
Cell: +92 343 261 3622
Email: mujtaba_1986@yahoo.com