Serial Sonographic Assessment of Pulmonary Edema in Patients With Hypertensive Acute Heart Failure

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Objectives—Objective measures of clinical improvement in patients with acute heart failure (AHF) are lacking. The aim of this study was to determine whether repeated lung sonography could semiquantitatively capture changes in pulmonary edema (B-lines) in patients with hypertensive AHF early in the course of treatment.

Methods—We conducted a feasibility study in a cohort of adults with acute onset of dyspnea, severe hypertension in the field or at triage (systolic blood pressure $\geq$ 180 mm Hg), and a presumptive diagnosis of AHF. Patients underwent repeated dyspnea and lung sonographic assessments using a 10-cm visual analog scale (VAS) and an 8-zone scanning protocol. Lung sonographic assessments were performed at the time of triage, initial VAS improvement, and disposition from the emergency department. Sonographic pulmonary edema was independently scored offline in a randomized and blinded fashion by using a scoring method that accounted for both the sum of discrete B-lines and degree of B-line fusion.

Results—Sonographic pulmonary edema scores decreased significantly from initial to final sonographic assessments ($P < .001$). The median percentage decrease among the 20 included patient encounters was 81% (interquartile range, 55%–91%). Although sonographic pulmonary edema scores correlated with VAS scores ($\rho = 0.64$; $P < .001$), the magnitude of the change in these scores did not correlate with each other ($\rho = -0.04$; $P = .89$).

Conclusions—Changes in sonographic pulmonary edema can be semiquantitatively measured by serial 8-zone lung sonography using a scoring method that accounts for B-line fusion. Sonographic pulmonary edema improves in patients with hypertensive AHF during the initial hours of treatment.

Key Words—chest/lune; congestive heart failure; critical care; dyspnea; emergency medicine; hypertension; hypoxia; pulmonary edema; sonography

Received March 27, 2017, from the State University of New York Downstate Medical Center, Brooklyn, New York USA (J.L.M., J.F.K., I.S.d., L.P., A.A., N.M., A.C., R.S.); and Stony Brook University Hospital, Stony Brook, New York USA (M.S.). Manuscript accepted for publication April 24, 2017.

This work was supported in part by New York University Clinical and Translational Science Award grant UL1 TR001445 from the National Center for Advancing Translational Sciences, National Institutes of Health.

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Abbreviations

AHF, acute heart failure; ED, emergency department; EMS, emergency medical services; IQR, interquartile range; VAS, visual analog scale

doi:10.1002/jum.14336

A primary diagnosis of acute heart failure (AHF) accounts for approximately 1 million annual emergency department (ED) visits in the United States.[1] Considering the high rates of 30-day readmission[2] and 30-day mortality[3] after AHF hospitalization, accurate assessment of treatment efficacy is critical. However, objective noninvasive measures of clinical improvement in patients with AHF are lacking. Changes in brain-type natriuretic peptide levels are unlikely to rapidly decrease,[4] and incorporating serial brain-type natriuretic peptide levels into inpatient management has not been shown to improve the hospital length of stay, 30-day mortality, or readmission rates.[5] The most common symptom prompting patients

with AHF to seek care is dyspnea,[6,7] and its improvement is an important measure for ascertaining therapeutic efficacy in clinical practice and clinical trials.[8–10] Dyspnea, however, is subjective and multifactorial, and the language used to describe dyspnea is subject to cultural and racial differences.[11]

A more objective measure of pulmonary edema, the primary pathophysiologic derangement underlying dyspnea in patients with AHF, might help clinicians better gauge therapeutic success. The chest radiograph has been the conventional imaging tool used to identify and follow changes in pulmonary edema. Radiographic signs of pulmonary edema, however, are insensitive,[12] are subject to variability in interpretation,[13] and may not change contemporaneously with rapid fluid changes occurring in the pulmonary interstitium. Lung sonography, in contradistinction, identifies pulmonary edema with high sensitivity[12] and has demonstrated utility in capturing changes in pulmonary edema in real time.[14,15] The dynamics of pulmonary decongestion using lung sonography in patients with AHF are not well defined and have not been studied during the earliest phases of treatment.

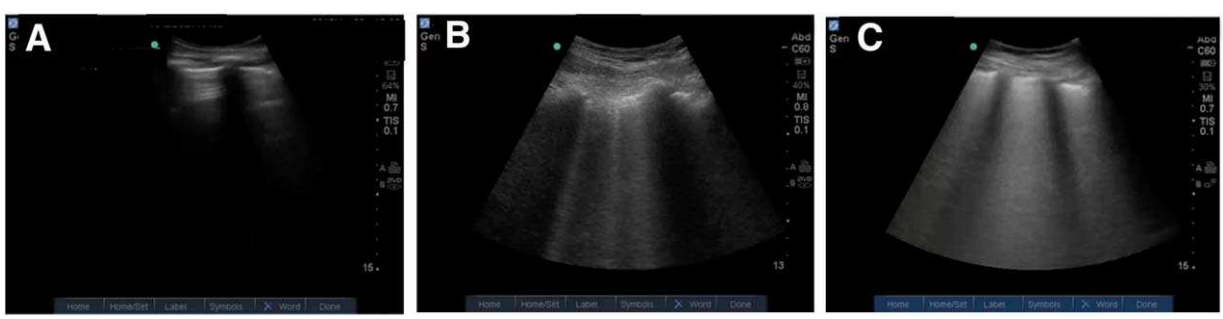
Semiquantitative measures of sonographic pulmonary edema have been based on the grand sum of discrete vertical artifacts called B-lines in an intercostal space from each of 28 thoracic zones.[14–20] When pulmonary edema is severe, B-line quantification becomes less straightforward because B-lines fuse together (Figure 1). An alternative method for quantifying B-lines based on estimates of B-line confluence has been proposed[21] and found to have good inter-rater reliability,[22] but it has not been applied to serial sonographic measures of pulmonary edema. The objective of this study was to demonstrate the feasibility of measuring sonographic pulmonary decongestion in patients with AHF during the course of their treatment in the ED with a simplified 8-zone scanning protocol and a measure of B-lines that accounts for B-line fusion. We chose to study patients with hypertensive AHF who presented with acute onset of dyspnea to ensure that lung sonography could capture the most rapid changes in pulmonary edema. In this phenotype of AHF, dyspnea is often severe and improves dramatically in response to treatment targeting cardiac loading conditions.[23,24]

Materials and Methods

Study Population

We conducted a prospective observational cohort study designed to evaluate the extent to which sonographic B-lines disappear with treatment in a convenience sample of patients presenting to the ED with presumed hypertensive AHF and acute dyspnea. Enrollment took place between October 2015 and September 2016 in 2 academic EDs with a combined annual census of 195,000 patients. This study was conducted in accordance with the amended Declaration of Helsinki and was approved by the Institutional Review Boards at both hospitals. Patients with an early working diagnosis of hypertensive AHF were selected on the basis of an initial systolic blood pressure of 180 mm Hg or higher obtained either in the prehospital setting or at ED triage, acute onset of dyspnea, and the presence of pathologic B-lines ($\geq$3 lines per intercostal space) in both lungs on rapid anterior 2-region lung sonography.[21,25] Exclusion criteria were fever, ST-elevation myocardial infarction, primary lung malignancy, pulmonary fibrosis, sarcoidosis, a working diagnosis of acute lung disease (chronic obstructive pulmonary disease, asthma, or pneumonia) as the primary cause of dyspnea, pneumothorax, acute respiratory distress syndrome, acute pulmonary embolism, and end-

Figure 1. Still images obtained from lung sonographic video clips. **A**, Absence of B-lines. **B**, Discrete B-lines. **C**, Complete B-line fusion.



stage renal disease. The study sample was limited to those patients who could be scanned within 45 minutes of ED triage. Patients were not excluded if they received prehospital vasodilator or diuretic therapy or if noninvasive ventilation was initiated by emergency medical services (EMS) prior to ED arrival.

Study Flow

Immediately after eligible patients consented to participate in the study, the investigator performed initial 8-zone lung sonographic and dyspnea assessments and measured concurrent vital signs. Thereafter, dyspnea severity was repeatedly assessed at fixed intervals during the ED course until the patient showed a 2-cm improvement on a visual analog scale (VAS). A second lung sonographic examination and set of vital signs were obtained at the time of this initial substantial improvement. A third and final lung sonographic examination, dyspnea assessment, and set of vital signs were obtained at the time the treating clinician decided on the patient's disposition (regardless of the patient's score on the VAS). The treating clinicians were blinded to sonographic pulmonary edema scores and not present in the room for the repeated 8-zone lung sonographic examinations. The treating team was able to perform its own lung sonographic examinations if it wished to. In a few cases in which the treating clinician had not yet performed his or her own diagnostic lung sonography, the clinician was in the room for the rapid anterior 2-region lung sonography used to determine study eligibility performed by the investigator. The treating clinicians were not asked to follow any specific treatment protocol.

Dyspnea Assessment

Dyspnea severity was evaluated by an uncalibrated horizontal 10-cm VAS that has been previously used in heart failure research.[26] Patients were asked to mark the line based on their self-perceived proximity to the 2 statements flanking the line. If patients reported feeling "less short of breath" (they were asked at 20-minute intervals from the time of the initial sonogram), they were given the same VAS that they initially marked and asked to make a second mark. A second sonographic examination was performed if and when a 2-cm improvement was measured on the VAS. This level of improvement has previously been defined as a clinically meaningful difference.[27] Dyspnea severity was also serially determined by a 5-point Likert scale at the of each lung sonographic examinations. Measures on this ordinal scale have previously been shown to correlate with B-line severity.[28]

Lung Sonography

Investigators performing lung sonography were board-certified emergency physicians with variable levels of formal training in point-of-care sonography. All investigators underwent a 30-minute training session on the lung sonographic protocol before enrollment. As described by Volpicelli et al,[29] lung sonography was performed by scanning 8 thoracic zones (2 anterior and 2 lateral per hemithorax) with the head of the stretcher at a minimum of 45 °. Scans were performed with a 3.5-MHz curvilinear transducer (SonoSite, Inc, Bothell, WA) on abdominal settings set to a depth of 11 to 15 cm. A 6-second video clip of an intercostal space from each of the 8 thoracic zones was recorded for future offline interpretation.

Video segments were recorded as QuickTime mp4 files (Apple Corporation, Cupertino, CA), which were then numbered and arranged by a random-sequence generator before compilation into a single randomized file for review after patient enrollment was completed. Two investigators who were fellowship trained in sonography (J.F.K. and M.S.) independently analyzed the deidentified video clips and recorded their assessments in separate spreadsheet files (Excel 2014, version 15.18; Microsoft Corporation, Redmond, WA). Each investigator analyzed every video clip, and comparisons of the scores for all clips were used to measure inter-rater reliability. A subset of 40 video clips from the data set was randomly selected by a random-number generator and then duplicated and mixed into the data set to measure intra-rater reliability. The investigators performing clip analysis were blinded to patient identity, clinical information, and timing of video clip acquisition.

B-lines were defined as vertical hyperechoic lines arising from the pleural line, extending down to the bottom of the screen, and moving synchronously with respirations. A sonographic pulmonary edema score was based on the greatest number of B-lines (or degree of B-line fusion) noted in a single intercostal space at any instant during each video clip. Confluent B-lines were scored by dividing the percentage of the pleural line occupied by confluence by 1021. This number was added to any other discrete B-lines that might have been simultaneously noted in the intercostal space. A score of 10 would be applicable if the entire pleural line were

occupied by B-line confluence.[22] The overall sonographic pulmonary edema score for each 8-zone scan was defined as the sum of individual scores. Final sonographic pulmonary edema scores were based on the average sum between the 2 investigators' independent scores. Median imputation was performed for video clips that were missing or classified as uninterpretable.

Statistical Analysis

A sample size of 20 patients was calculated with a 2-tailed α of .05, a β error of 0.1, and estimations of the effect size and the standard deviation of the effect size. On the basis of prior studies that measured changes in B-line sums across 28 scanned thoracic regions in response to inpatient treatment, we estimated that the average initial B-line score in our study would be smaller in value (30) and decrease by 50% from the time of the initial scan to self-reported improvement in dyspnea.[17,30] An estimated SD of the change in the effect size of 20 was based on standard deviations reported for raw B-line scores in these studies.

Continuous variables are expressed as median (interquartile range [IQR]), as appropriate. Categorical variables are presented as count (percentage). Comparisons among the 3 sonographic pulmonary edema scores and among the 3 dyspnea VAS scores were performed by the Friedman test. Spearman analysis was used to correlate sonographic pulmonary edema scores with VAS scores and the change in sonographic pulmonary edema scores with the change in VAS scores. Inter-rater and intra-rater reliability were determined by intraclass correlation coefficients with absolute agreement, 2-way random effects. Clips that 1 or both raters deemed to be uninterpretable were excluded from intraclass correlation coefficient estimates. The statistical analysis was performed with SPSS version 20 software (IBM Corporation, Armonk, NY).

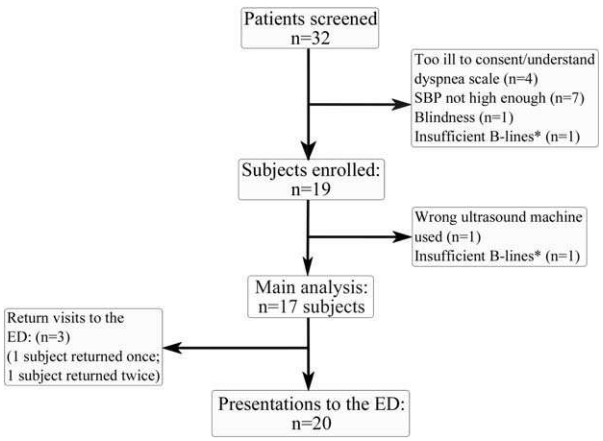
Results

A total of 19 patients were enrolled, and 2 patients were excluded: 1 patient was scanned with a different ultrasound machine than the one selected for the study protocol; another patient was determined not to have initial bilateral pathologic B-lines. Two patients returned to the ED during the enrollment period, contributing more than once to the 20 total ED presentations that were analyzed (Figure 2).

Clinical characteristics of the patients included in the final analysis are shown in Table 1. All patients were African American, and 6 of the 17 patients were male. The mean arterial pressure at the time of ED triage was high (139 mm Hg [IQR, 123–152 mm Hg]) in spite of the fact that prehospital treatment was administered before half of the ED encounters (Table 2). Noninvasive ventilation was administered during every patient encounter. Based on data from formal inpatient echocardiograms after enrollment (n = 16), 8 patients had a reduced ejection fraction (<40%); 4 had a midrange ejection fraction (40%–49%), and 4 had a preserved ejection fraction (≥50%; Table 1). Four patients had echocardiography performed within the 6 months preceding study enrollment. The ejection fraction was preserved in 3 of these patients and reduced in 1. Evidence of diastolic dysfunction was present on the echocardiogram for each patient with a midrange or preserved ejection fraction.

Lung sonography was performed by 5 of the study investigators and was feasible in every patient. Two video clips (zones 4 and 6 on the initial sonographic study of a patient) were inadvertently not recorded. Of the 924 video clips reviewed by the 2 investigators (each reviewed the same data set of 462 recorded clips), 824 (97%) could be analyzed and scored. Inter-rater reliability (0.83 [95% confidence interval, 0.75–0.88]; n = 439 clips) and intra-rater reliability (intraclass correlation coefficient, 0.97 [95% confidence interval, 0.96–0.98]) for B-line scoring were excellent. The median time from

Figure 2. Study flow chart.
*Insufficient B-lines: less than 3 B-lines per intercostal space, bilaterally. SBP indicates systolic blood pressure.



ED triage to the first lung sonographic examination was 12.5 minutes (IQR, 3.25–24.5 minutes).

Sonographic pulmonary edema scores decreased from the initial assessment to the time of disposition decision in every patient (Figure 3). The presence of B-line fusion was observed in 40% of video clips. Median sonographic pulmonary edema scores for initial, second, and third sonographic studies were 47 (IQR, 40–51), 25 (IQR, 11–35), and 8 (IQR, 4–19), respectively. The decrease in the number of B-lines among the 3 lung sonographic studies was statistically significant ($\chi^2 = 31.6$ [2, n = 18]; $P < .001$). Sonographic pulmonary edema scores decreased by a median of 81% (IQR, 44%–99%) from the initial to the final lung sonography.

Vital signs improved during the course of ED treatment in this cohort of patients (Table 3). All patients showed a minimum improvement of 2 cm on the 10-cm VAS between triage and disposition decision. The

Table 1. Clinical Characteristics of the Study Population

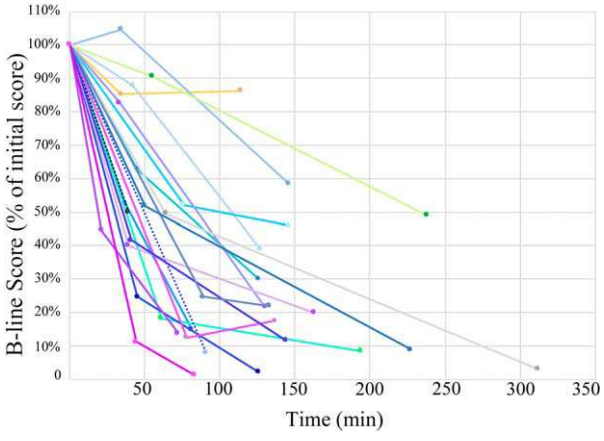
Characteristic	Value
Demographics	
Age, y	67.5 (57.8–77.3)
Male, n (%)	6 (35)
African American, n (%)	17 (100)
Body mass index, kg/m^2	29 (24.6–32.0)
Medical history, n (%)	
Hypertension	16 (94)
Diabetes mellitus	10 (59)
Congestive heart failure	13 (76)
Myocardial infarction	4 (24)
Coronary artery bypass graft	2 (12)
Percutaneous coronary intervention	3 (18)
Pacemaker	1 (6)
Automatic implantable cardioverter defibrillator	4 (24)
Renal insufficiency	3 (18)
Stroke	3 (18)
Atrial fibrillation	1 (6)
Chronic obstructive pulmonary disease	4 (24)
Asthma	1 (6)
EMS data	
Systolic blood pressure (n = 12), mm Hg	202 (190–213)
Diastolic blood pressure (n = 12), mm Hg	133 (109–141)
Room air oxygen saturation (n = 9), %	89 (88–90)
Triage vital signs	
Heart rate, beats/min	101 (90–113)
Systolic blood pressure, mm Hg	188 (159–199)
Diastolic blood pressure, mm Hg	113 (97–134)
Mean arterial pressure, mm Hg	139 (123–152)
Respiratory rate, breaths/min	30 (23–32)
Laboratory data	
Brain-type natriuretic peptide, pg/mL	894 (483–1414)
Sodium, mEq/L	139 (136–141)
Blood urea nitrogen, mg/dL	24 (17.3–31.8)
Creatinine, mg/dL	1.4 (1.0–2.3)
Hemoglobin, g/dL	12.1 (11.0–13.7)
pH	7.34 (7.25–7.37)
Lactate, mg/dL	1.6 (1.3–3.2)
Echocardiographic data (n = 16), n (%)	
Reduced ejection fraction (<40%)	8 (50)
Midrange ejection fraction (40%–49%)	4 (25)
Preserved ejection fraction (≥50%)	4 (25)

Data are presented as median (IQR) unless otherwise indicated.

Table 2. Treatment and Disposition

Characteristic	Value
EMS treatment, n (%)	
Administration of nitroglycerin (tab or spray)	9 (45)
Initiation of noninvasive ventilation	4 (20)
Administration of furosemide	2 (10)
ED treatment, n (%)	
Nitroglycerin (tab or spray)	11 (55)
Nitroglycerin continuous infusion	12 (60)
Non-invasive ventilation	20 (100)
Enalapril (intravenous)	13 (65)
Hydralazine	3 (15)
Furosemide (intravenous)	7 (35)
Disposition, n (%)	
Coronary care unit	7 (35)
Step-down unit	1 (5)
Telemetry	6 (30)
Floor	6 (30)
Hospitalization	
Median length of stay (IQR), d	5 (3.0–7.5)

Figure 3. Percentages of initial B-line score over time. Dotted lines represent the 2 patient encounters that were scanned only twice (failed to improve by >2 cm on the dyspnea scale before treatment disposition).



improvement in VAS scores was statistically significant ($\chi^2 = 36$ [2, n = 18], $P < .001$). Two patients failed to show at least 2 cm of improvement at a time earlier than disposition decision. Lung sonography was performed only at triage and disposition in these 2 patients. In 1 of the 2 patients, the sonographic pulmonary edema score improved by 91% in spite of only a 2.8-cm improvement in dyspnea at the time of disposition. The other patient had a low initial VAS score (2.3 cm) in spite of a high initial sonographic pulmonary edema score (50). A disposition decision was made early into the ED course of this patient (39 minutes after the initial sonographic study), at which time the sonographic pulmonary edema score improved by 50%. Although there was a correlation between the B-line score and dyspnea as measured by the VAS ($\rho = 0.64$; $P < .001$), there was no correlation between the magnitude of change in sonographic pulmonary edema and VAS scores (final score – initial score; $\rho = -0.04$; $P = .89$).

Discussion

Our study demonstrates that serial lung sonography can capture rapid changes in pulmonary edema in a cohort of hypertensive patients presenting to the ED with acute pulmonary edema. Reliable estimates of sonographic pulmonary edema were obtained by using an 8-zone scanning approach and a scoring system that incorporated estimates of B-line fusion. This study adds to previous work investigating changes in sonographic pulmonary edema in response to longer durations of AHF treatment. In a cohort of 25 patients with systolic heart failure presenting to an ED with clinical signs of pulmonary congestion, Facchini et al[30] performed 28-zone lung sonographic assessments before and after a 24-hour infusion of diuretic therapy. B-line scores decreased by 41% (mean ± SD, 53.4 ± 17.2 to 31.7 ± 13.5). Gargani et al[20] also demonstrated partial pulmonary decongestion in response to inpatient treatment of AHF using the same scanning protocol. B-line scores in this cohort of 100 patients decreased by 58% (48 ± 48 to 20 ± 23) from hospital admission to discharge. Using a simplified lung sonographic score based on the number of positive thoracic areas (a positive area defined as ≥3 B-lines), Volpicelli et al[31] demonstrated a decrease in the median number of positive lung zones from 8 to 1 (of the 11 thoracic zones interrogated) from hospital admission to discharge (after 4.2 ± 1.7 days) among 70 patients with AHF. Dichotomizing individual sonographic pulmonary edema scores for each of the 8 lung zones interrogated in our study (positive defined as a sonographic pulmonary edema score ≥3) produces similar results: a decrease from a median of 8 to 1 positive lung zones (of the 8 interrogated) from triage to ED disposition (after a median of 138 minutes [IQR, 103.5–170.5 minutes). Although this scoring method might be most easily incorporated into clinical practice, it might lack the sensitivity to detect more gradual and less dramatic improvement in pulmonary decongestion during the initial phase of AHF treatment.

In a heterogeneous cohort of patients with AHF, Cortellaro et al[32] evaluated the dynamics of pulmonary decongestion over the initial 24 hours of treatment using a simplified scoring system that incorporated B-line

Table 3. Clinical and Sonographic Profiles of Patients at ED Triage, Initial Self-Reported Improvement of Dyspnea, and Disposition

Characteristic	Phase 1 (ED Triage)	Phase 2 (Improved Dyspnea)	Phase 3 (Disposition)
n	20	18	20
Time from triage, min	12.5 (3.3–24.5)	56.5 (42.0–67.8)	138 (103.5–170.5)
Vital signs			
Systolic blood pressure, mm Hg	192 (157–208)	153 (114–185)	150 (122–180)
Diastolic blood pressure, mm Hg	106 (95–126)	86 (74–100)	83 (72–100)
Heart rate, beats/min	101 (85–115)	92 (79–104)	83 (77–98)
Respiratory rate, breaths/min	32 (27–34)	25 (21–28)	26 (22–28)
Lung sonography			
Raw B-line score	47 (40–51)	25 (11–35)	8 (4–19)
Proportion of initial B-line score, %	1	50 (25–84)	19 (9–45)
Dyspnea scores			
VAS	7.5 (5.5–9.0)	3.0 (1.8–4.5)	0.7 (0.0–1.8)
5-point Likert scale	4 (3–4)	2 (2–3)	2 (1–2)

Data are presented as median (IQR) unless otherwise indicated.

coalescence (0, no B-lines; 1, multiple B-lines; and 2, confluent B-lines) across 11 lung zones. At 3 hours from admission, hemodynamic and respiratory vital signs improved significantly, and the B-line score improved by 54%. The most commonly identified trigger for the patients in this cohort was hypertension (defined in this study as diastolic pressure >120 mm Hg). This study further supports the feasibility of performing serial lung sonography throughout the acute phase of treatment of AHF and in quantifying rapid changes in pulmonary edema using a simplified scale.

The rate of improvement in sonographic pulmonary edema in our study was more rapid than that observed by Cortellaro et al,[32] likely because of differences in our studied populations. Although our study was not designed to evaluate the rate of sonographic pulmonary edema improvement, it is notable that the median time for an 80% or greater reduction in sonographic pulmonary edema (in the 10 patients who attained this degree of improvement in the ED), was 86 minutes (IQR, 69–149 minutes). Acute pulmonary edema in the hypertensive phenotype of AHF that we studied is thought to result from sympathetically mediated vascular redistribution of fluid from vasoconstricted splanchnic and peripheral vascular beds into the pulmonary circulation rather than from volume overload.[24,33] Based on this conceptual model of disease, therapies aimed at reducing cardiac filling pressures are likely to redistribute the blood volume and decrease pulmonary edema. In addition to its pulmonary benefits, noninvasive ventilation may be particularly useful in hypertensive AHF through its hemodynamic effects on reducing cardiac loading conditions.[34,35] Noninvasive ventilation alone has been shown to resolve pulmonary edema in a patient with both congestive heart failure and end-stage renal disease over several hours.[36] Every patient received noninvasive ventilation during the ED treatment course. Vasodilator therapy in the form of nitrates can rapidly improve cardiac filling pressures and redistribute blood volume. Nitrate therapy was administered by EMS or in the ED to all but 1 patient enrolled in this study. This patient had the least amount of sonographic pulmonary edema improvement (14%). Patients seemed to benefit from vasodilator therapy and noninvasive ventilation regardless of their ejection fraction. An 86% reduction in sonographic pulmonary edema was observed in the 11 patients in this study who did not receive loop diuretic therapy.

Measures of pulmonary edema after therapeutic intervention in the ED have the potential to help risk stratify patients and assist in disposition decisions. Patients with hypertensive AHF who present with the most severe dyspnea and degree of pulmonary edema may have complete resolution of pulmonary edema in a short time. Persistent pulmonary edema after treatment, however, may indicate a need for further therapeutic intervention. Pulmonary decongestion is incomplete in some patients with AHF even after inpatient treatment, and residual pulmonary edema at hospital discharge has been shown to predict rehospitalization.[19,20]

Symptomatic improvement is an important clinical goal in treating patients with AHF and improved dyspnea, as measured by reduced VAS scores, and has been used as an outcome measure in major clinical trials.[8,26] Despite a correlation between raw VAS scores and sonographic pulmonary edema scores in our study, we did not find a correlation between the magnitudes of change in these measures. The severity of dyspnea that patients presented with (7.5 cm [95% confidence interval, 5.5–9.0 cm]) likely made this self-assessment more challenging than it would have been in patients who were in less respiratory distress. Self-perceived relief of dyspnea may also partially reflect decreased underlying anxiety and adrenergic tone and thus might be exaggerated in patients who are reassured by treating clinicians of their anticipated clinical improvement and averted intubation. One patient reported an 8-cm improvement on the VAS despite minimal improvement in sonographic pulmonary edema (14%). In most of the patients, the percentage of change on the VAS exceeded that observed for sonographic pulmonary edema. Changes in the VAS might be larger than those observed on lung sonography if, as suggested by previous studies,[19,20] subclinical sonographic pulmonary edema is significant. Persistent sonographic pulmonary edema was observed in the 6 patients in our study who reported complete resolution of dyspnea on the VAS.

Our study had several important limitations. The small sample size of this feasibility study precluded a subgroup analysis. Analyses of sonographic pulmonary edema resolution stratified by different treatment approaches (eg, administration of a diuretic and dosage of nitroglycerin) and clinical variables (eg, ejection fraction) in future studies would be informative. Because our study objective was to capture rapid and dramatic changes in sonographic pulmonary edema, we chose to

limit our studied population to hypertensive patients with AHF and acute dyspnea. Our results are therefore not generalizable to patients with AHF who have lower presenting blood pressures or gradual dyspnea. The presence of bilateral pulmonary edema was an inclusion criterion in this study, and a selection bias may have played a role in selecting patients with a more severe degree of pulmonary edema on the initial examination and subsequently greater improvement in sonographic pulmonary edema. The dyspnea scale used in this study, whereas used in other heart failure research, is not a validated instrument, so correlations between pulmonary decongestion and dyspnea improvement may be affected by limitations of the scale itself. Another methodological limitation in this study was the allowance for a variable image depth as part of our scanning protocol. Most (87%) of our video clips were set to a depth of 15 cm, and we view it unlikely that image depths several centimeters short of this value would lead to an overestimation of the B-line number. This study does not reflect true before-and-after changes in sonographic pulmonary edema after therapeutic intervention, as half of the enrolled patients received prehospital treatment by EMS. A strength of this study, however, was the short time between ED triage and initial lung sonography. The same sonographer performed each of the serial scans for a single patient during the treatment course, which may have introduced some degree of observer bias. To limit the effects of observer bias, sonographic pulmonary edema scoring was performed in a blinded fashion, and video clips were randomized by patient and scan sequence. Our results would be more applicable to clinical practice if scoring were concurrent with scanning, and both scoring and scanning were performed by clinicians lacking advanced training in sonography.

In conclusion, this study demonstrates the dynamics of pulmonary edema in dyspneic patients with hypertensive AHF early in their treatment course. Real-time feedback from serial sonographic assessments of pulmonary edema may be informative to the clinician titrating AHF treatments and making disposition decisions. Validation of this simplified scanning protocol and semiquantitative approach to measuring changes in sonographic pulmonary edema in a larger cohort of patients with different phenotypes of AHF is needed. The reliability of measuring pulmonary edema while scanning in comparison to offline analysis also merits study.

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