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ORIGINAL RESEARCH ARTICLE

Body mass index and B-lines on lung ultrasonography in chronic and acute heart failure

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Abstract

Aims Increased body mass index (BMI) is common in heart failure (HF) patients and is associated with lower levels of N-terminal pro-brain natriuretic peptide (NT-proBNP). We evaluated the influence of BMI on lung ultrasonography (LUS) findings indicative of pulmonary congestion (i.e. B-lines) in patients with chronic and acute HF (AHF).

Methods and results We analysed ambulatory chronic HF (n = 118) and hospitalized AHF (n = 177) patients (mean age 70 years, 64% men, mean BMI 29 kg/m^2, mean ejection fraction 42%) undergoing echocardiography and LUS in eight chest zones. B-lines and chest wall thickness (skin to pleura) on ultrasound were quantified offline and blinded to clinical findings. NT-proBNP was available in AHF patients (n = 167). In chronic HF, B-line number decreased by 18% per 5 unit increase in BMI [95% confidence interval (CI) −35% to +5%, P = 0.11]. In AHF, the number of B-lines decreased by 12% per 5 unit increase in BMI (95% CI −19% to −5%, P = 0.001), whereas NT-proBNP concentration decreased by 28% per 5 unit increase in BMI (95% CI −40% to −16%, $P < 0.001$). For AHF, B-line number declined to a lesser degree than NT-proBNP concentration with increasing BMI (P = 0.020), and >6 B-lines were observed in half of AHF patients with severe obesity. There was an inverse relationship between B-line number and chest wall thickness, and this association varied by chest region.

Conclusions Despite an inverse relationship between B-lines and BMI, B-lines declined to a lesser degree than NT-proBNP with increasing BMI. These data suggest that LUS may be useful in patients with HF despite obesity.

Keywords Lung ultrasonography; B-lines; Body mass index; Heart failure

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Introduction

Diagnosis of heart failure (HF) in patients who are obese is difficult because obesity itself may lead to dyspnoea and peripheral oedema, and, conversely, clinical detection of pulmonary congestion is difficult in individuals who are overweight. Likewise, assessment of response to decongestive therapy is also challenging in these patients. Both problems are compounded by the inverse correlation between natriuretic peptide concentrations and body mass index (BMI).[1] Lung ultrasonography (LUS) offers an alternative approach to assessing pulmonary congestion in patients with suspected or proven HF. LUS enables identification of extravascular lung water by visualization of hyperechoic vertical lines originating from the pleural surface.[2] These are known as B-lines and, when quantified, provide a measure of the degree of pulmonary congestion. Prior research has demonstrated that quantification of B-lines facilitates the diagnosis of HF and may be useful for monitoring of HF therapy.[3–5] Furthermore, B-lines provide prognostic information regarding HF hospitalization and all-cause death.[6] LUS may therefore provide a weight-independent measure of congestion in HF.

However, body habitus could potentially also affect the visibility of the pleural surface[7] and, hypothetically, interfere

with the observed number of B-lines, although this possibility has not been studied previously in patients with HF.

Thus, we investigated the relationship between BMI and B-line prevalence in chronic HF and acute HF (AHF). We also compared the prevalence of B-lines with levels of natriuretic peptides and chest wall thickness in AHF.

Methods

Study population

We analysed data from two prospective, observational studies conducted at a single academic hospital in patients with chronic HF (n = 118) and AHF (n = 177) (*Figure 1*). Details of both cohorts have been published.[8,9] All patients underwent transthoracic echocardiography (TTE) and concomitant LUS. We collected clinical and demographic data from the patients' hospital records. N-terminal pro-brain natriuretic peptide (NT-proBNP) was measured in the patients with AHF (n = 167). We excluded subjects with BMI $>$ 50 kg/m^2 (n = 7) to allow for comparability of the two cohorts. All patients answered a questionnaire assessing dyspnoea on exertion and/or at rest. Our dichotomization of HF patients into those with reduced ejection fraction (HFrEF) and preserved ejection fraction (HFpEF) was based on left ventricular ejection fraction (LVEF) cut-off of 45%.[10]

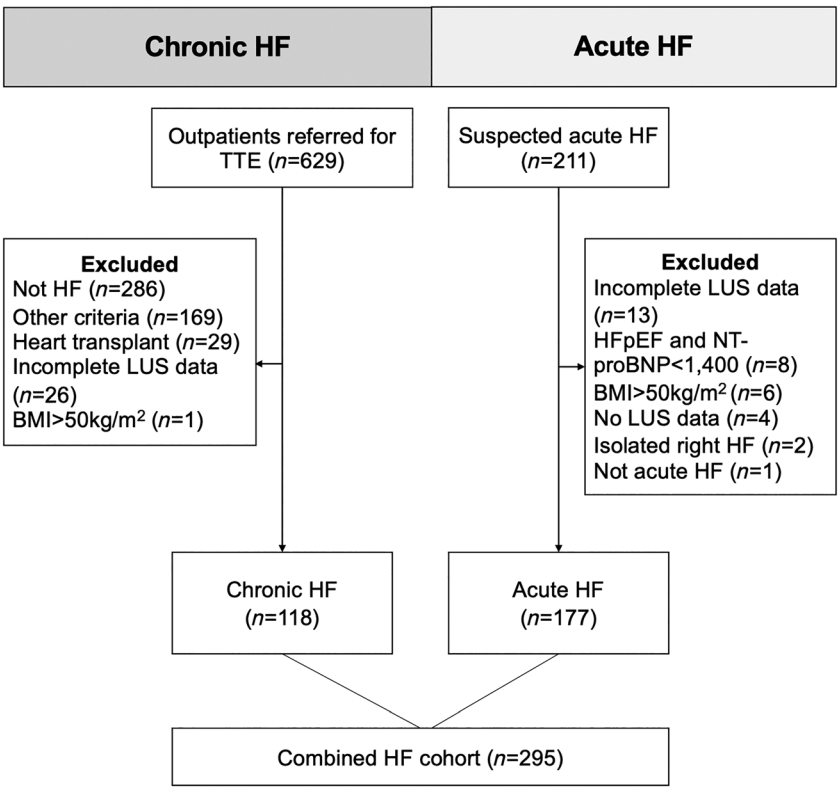
Chronic heart failure

We included prospectively enrolled patients referred for clinically indicated outpatient TTE between February 2014 and October 2015, as previously reported.[8] The TTE was conducted in the outpatient echocardiographic laboratory. Main exclusion criteria were interstitial lung disease, recent chest trauma, lung cancer, lung metastases $>$1 cm, prior lung or heart transplant, current chest drain, pregnancy, pneumothorax, pneumonia, dialysis, liver failure, and same-day admission to the hospital. A total of 119 patients with a prior clinical diagnosis of HF (defined according to the Framingham criteria[11] by review of electronic medical records) were identified from a larger cohort as previously described.[8]

Acute heart failure

We included patients ≥18 years old who were hospitalized for AHF between April 2015 and August 2017, irrespective of LVEF, and scheduled for a clinically indicated TTE early during their hospital stay [median 1 day, interquartile range (IQR) 1,

Figure 1 Flowchart. Consort diagram showing the inclusion of patients with chronic and acute heart failure from the two study populations. BMI, body mass index; HF, heart failure; HFpEF, heart failure with preserved ejection fraction; LUS, lung ultrasonography; NT-proBNP, N-terminal pro-brain natriuretic peptide; TTE, transthoracic echocardiography.



1 day since admission].[9] The TTE was performed in the inpatient units. We defined AHF as at least two signs and symptoms of HF (dyspnoea at rest, weight gain, crackles, peripheral oedema, ascites, elevated jugular venous pressure, and increasing abdominal distension) or one objective finding (chest X-ray with pulmonary oedema/vascular congestion/pleural effusion or LUS with bilateral B-lines/pleural effusions) and the need for ≥1 dose of intravenous diuretics. Additionally, patients with prior HFpEF diagnosis had to have BNP/NT-proBNP levels ≥350/≥1400 to be included. Main exclusion criteria were in intensive care unit at time of TTE, ventricular assist device, prior heart or lung transplant, isolated right HF, chest drain in place, current pneumothorax, recent significant chest trauma, current lung or pleural cancer, interstitial lung disease, current pneumonia, dialysis, liver failure, or pregnancy. Hence, 177 patients were included in our analyses.

All patients provided written informed consent. Both studies were approved by the Local Ethics Committee and complied with the Declaration of Helsinki.

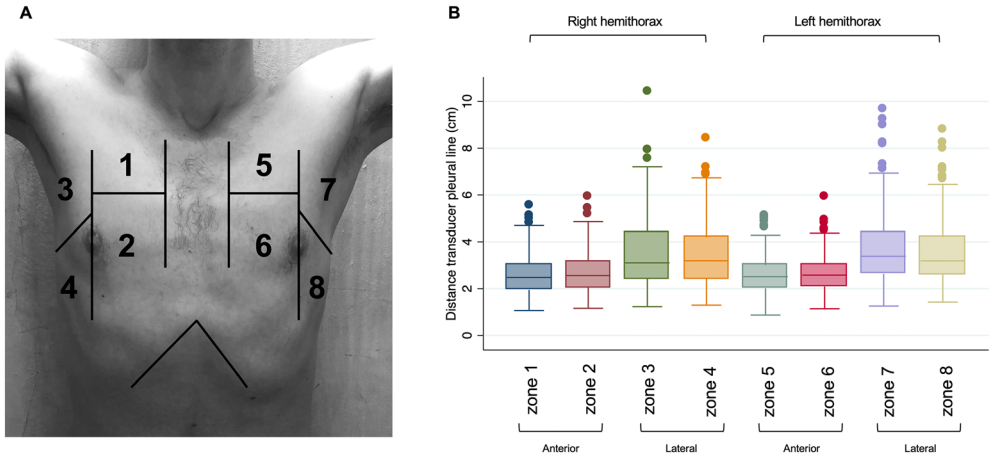
Echocardiography

Echocardiography was performed using 2–5 MHz phased array transducers with standard ultrasound equipment from three vendors (General Electric, Milwaukee, WI, USA; Philips, Bothell, WA, USA; and Siemens, Erlangen, Germany). Echocardiograms were stored offline and later analysed using echocardiographic software (Syngo Dynamics, Siemens, Malvern, PA, USA). All TTEs were analysed by trained investigators according to current guidelines by the American Society of Echocardiography[12] and blinded to all LUS data as previously reported.[8,9]

Lung ultrasound

At the time of TTE, trained investigators acquired LUS images in sagittal orientation using the same ultrasound equipment and transducers at an imaging depth of 18 cm in supine position for patients with chronic HF and semirecumbent position for AHF patients. Imaging frequencies were adjusted to optimize the visualization of the pleural line and B-lines. Six second clips in four zones on each hemithorax (eight LUS zones total) were recorded in all patients.[13] Analyses of LUS examinations were performed offline by trained investigators who were not involved in the LUS image acquisition and were blinded to all clinical and TTE data (K. H. D. and E. P.) The number of B-lines in a single intercostal space was counted for each zone and then summed for all eight zones (*Figure 2A*).[6] Intra-rater and inter-rater agreement has previously been described.[8] Data on B-lines were complete in the chronic HF cohort. As previously described, in the AHF cohort, we imputed B-line data from anatomically adjacent zones in patients with missing B-line data in ≤2 out of 4 zones who had undergone 8-zone LUS.[9] In order to further characterize the impact of obesity on LUS findings, we also obtained chest wall measurements from the LUS images described earlier. These were available in ≥7 zones for all AHF patients. The shortest and longest distance from the transducer to the pleural line was measured by a single investigator (V. S.) in order to calculate the mean chest wall thickness for each LUS zone (Supporting Information, *Figure S1*). Data were reported according to the

Figure 2 Lung ultrasound protocol and mean chest wall thickness by lung ultrasound zones in acute heart failure. (A) Example of lung ultrasound protocol with eight zones. Zones 1 and 2 are located on the medial area of thorax. Zones 3 and 4 are located on the lateral side of thorax. (B) Boxplot diagram of the mean chest wall thickness, assessed as distance from transducer to the pleural line (centimetres), in lung ultrasound zones 1–8 in patients with acute heart failure (n = 177).



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checklist for lung ultrasound studies in HF cohorts (Supporting Information, *Table S1*).[14]

Statistical analyses

Continuous variables are presented as means and standard deviations or median and IQR as specified. Characteristics for chronic HF and AHF were compared using Wilcoxon rank-sum test and ANOVA for continuous variables and Pearson's χ^2 test for categorical variables. We defined categories of BMI: lean (<25 kg/m^2), overweight (25 to 29.9 kg/m^2), obese (30 to 34.9 kg/m^2), and severely obese (35 to 50 kg/m^2).[15] Levels of NT-proBNP were assessed using a natural logarithmic transformation. Based on previous research in chronic HF patients, where B-lines ≥3 indicated increased risk of cardiac events, we divided patients according to three groups of B-lines: 0–2 B-lines, 3-6 B-lines and >6 B-lines.[6,16,17] The relationship between groups of B-lines and categories of BMI was assessed using Spearman's rho. B-lines in both chronic HF and AHF were not normally distributed and were highly dispersed. We therefore assessed B-lines as count data using negative binomial regression (NBR) models and reported incidence rate ratios with 95% confidence intervals (95% CIs). Cubic spline models based on NBR were used to illustrate the association between B-lines and BMI. The number of knots was selected according to the lowest values of Akaike information criterion. In an interaction analysis, we tested if type of HF modified the relationship between B-lines and BMI. Linear regression models were used to assess the association between BMI and NT-proBNP. Hausman's generalized specification test was used to compare the effect of BMI on levels of NT-proBNP and B-lines. Using a paired *t* test, we assessed the difference in B-lines and chest wall thickness between the anterior and lateral chest zones. NBR models were constructed to assess the association between chest wall thickness and B-lines. Two-sided significance levels of 0.05 were used for all analyses. Data were analysed using Stata SE, version 14.2 (StataCorp, College Station, TX, USA, 2015).

Results

Of the 295 patients included, 118 (40%) were ambulatory chronic HF patients and 177 (60%) were hospitalized for AHF. Baseline clinical characteristics are displayed in *Table 1*.

Table 1 Baseline clinical characteristics stratified by type of heart failure

	All patients	Chronic HF	Acute HF	*P*-value[a]
n	295	118	177	
Clinical data				
Age (years)	70 ± 14	68 ± 13	71 ± 14	0.10
Men, *n* (%)	188 (64%)	78 (66%)	110 (62%)	0.49
BMI (kg/m^2)	29 ± 6	28 ± 5	29 ± 7	0.29
Heart rate (b.p.m.)	75 ± 16	70 ± 14	79 ± 16	<0.001
Systolic BP (mmHg)	123 ± 22	127 ± 21	121 ± 22	0.021
Diastolic BP (mmHg)	68 ± 11	71 ± 11	66 ± 11	<0.001
Dyspnoea at rest, *n* (%)	171 (58%)	29 (25%)	142 (80%)	<0.001
Dyspnoea on exertion, *n* (%)	242 (82%)	69 (58%)	173 (98%)	<0.001
Medical history				
Hypertension, *n* (%)	249 (84%)	103 (87%)	146 (82%)	0.27
Diabetes, *n* (%)	114 (39%)	39 (33%)	75 (42%)	0.11
COPD, *n* (%)	41 (14%)	10 (8%)	31 (18%)	0.028
Myocardial infarction, *n* (%)	85 (29%)	27 (23%)	31 (18%)	0.08
CABG, *n* (%)	72 (24%)	27 (23%)	45 (25%)	0.62
Previous HF admission, *n* (%)	160 (54%)	58 (49%)	102 (58%)	<0.001
Atrial fibrillation, *n* (%)	151 (51%)	59 (50%)	92 (52%)	0.74
LVEF (%)	42 ± 14	45 ± 12	40 ± 15	0.014
Laboratory data				
Creatinine (mg/dL)	1.5 ± 0.8	1.2 ± 0.4	1.7 ± 0.9	<0.001
Sodium (mmol/L)	138 ± 4.4	140 ± 3.3	138 ± 4.7	0.002
Haemoglobin (g/dL)	11.8 ± 2.3	12.8 ± 2.2	11.4 ± 2.2	<0.001
Albumin (g/dL)	3.8 ± 0.5	4.1 ± 0.5	3.6 ± 0.5	<0.001
NT-proBNP (pg/mL) (*n* = 167)	—	—	4924 (2659, 11 211)	
Lung ultrasonography				
Chest wall thickness (cm)[b]	—	—	3.1 ± 1.0	

BMI, body mass index; BP, blood pressure; CABG, coronary artery bypass graft; COPD, chronic obstructive pulmonary disease; HF, heart failure; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-brain natriuretic peptide.
[a]*P*-value for difference between chronic and acute HF populations.
[b]Mean chest wall thickness averaged over eight lung ultrasound zones.

Chronic heart failure

B-lines and body mass index

Patients with chronic HF had a mean BMI of 28 ± 5 kg/m^2, and there was no difference in BMI between patients with chronic and AHF (P = 0.29). The number of B-lines ranged from 0 to 17, with a median of 2 (IQR 0, 5). Categories of B-lines and BMI are displayed in *Figure 3*. In the categorical analysis, the association between B-line number and BMI was not significant (Spearman's r = −0.11, P = 0.25). In the continuous variable analysis, the reduction in number of B-lines was 18% per 5 unit increase in BMI (95% CI −35% to 5%, P = 0.11, *Figure 4A*).

Acute heart failure

B-lines and body mass index

Patients with AHF had a mean BMI of 29 ± 7 kg/m^2. The number of B-lines ranged from 0 to 36 with a median of 12 (IQR 6, 18). The relationship between categories of B-line number and categories of BMI is displayed in *Figure 3*. In this categorical analysis, B-line number decreased significantly with increasing BMI (Spearman's r = −0.21, P = 0.005). However, more than six B-lines could still be observed in half of severely obese patients with AHF. The association between B-lines and BMI, with both examined as continuous variables, is displayed in *Figure 4B*. The sum of B-lines decreased by 12% per 5 unit increase in BMI (95% CI −19% to −5%, P = 0.001). This association remained significant after adjusting for age and LVEF (−11%, 95% CI −18% to −3%, P = 0.006, per 5 unit increase in BMI).

B-lines and body mass index and N-terminal pro-brain natriuretic peptide

Median NT-proBNP was 4924 ng/L (IQR 2659, 11 211). NT-proBNP concentrations were inversely related to BMI (standardized β = −0.36, $P < 0.001$). There was an 18% increase in B-lines per log unit increase in NT-proBNP (95% CI +7 to +31, P = 0.001). There was a 28% decrease in NT-proBNP concentration per 5 unit increase in BMI (95% CI −40% to −16%, $P < 0.001$). The decrease in NT-proBNP with increasing BMI was greater than the decrease in B-lines with increasing BMI, as reported above (P = 0.020).

B-lines and body mass index and chest wall thickness

Among patients with AHF, the mean chest wall thickness on ultrasound was 3.1 ± 1.0 cm when averaged over eight zones (*Figure 2B* and Supporting Information, *Table S2*). Both B-line number and chest wall thickness were higher in the lateral zones compared with the anterior zones ($P < 0.001$ for both). In the absence of a reference standard for pulmonary congestion, we examined whether B-line number was influenced by BMI and chest wall thickness. In the lateral zones, B-line number was associated with both BMI (−11% per 5 unit increase in BMI) and chest wall thickness (−13% per 1 unit increase in thickness). In the anterior zones, B-lines were only associated with BMI (−14% per 5 unit increase in BMI) but not with chest wall thickness (0% per 1 unit increase in thickness) (Supporting Information, *Table S3*). The presence of ≥3 B-lines in one zone has been used to identify pulmonary

Figure 3 Patients by category of B-lines and body mass index. Bar chart displaying percentage of patients with chronic and acute heart failure according to categories of B-lines (0–2; 3–6; and >6) across four categories of body mass index (<25; 25–29.9; 30–34.9; and 35–50). BMI, body mass index; HF, heart failure.

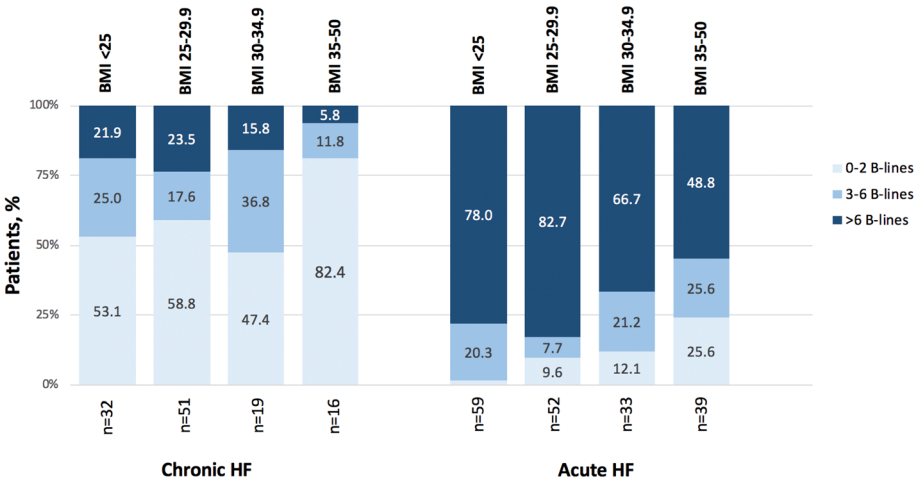

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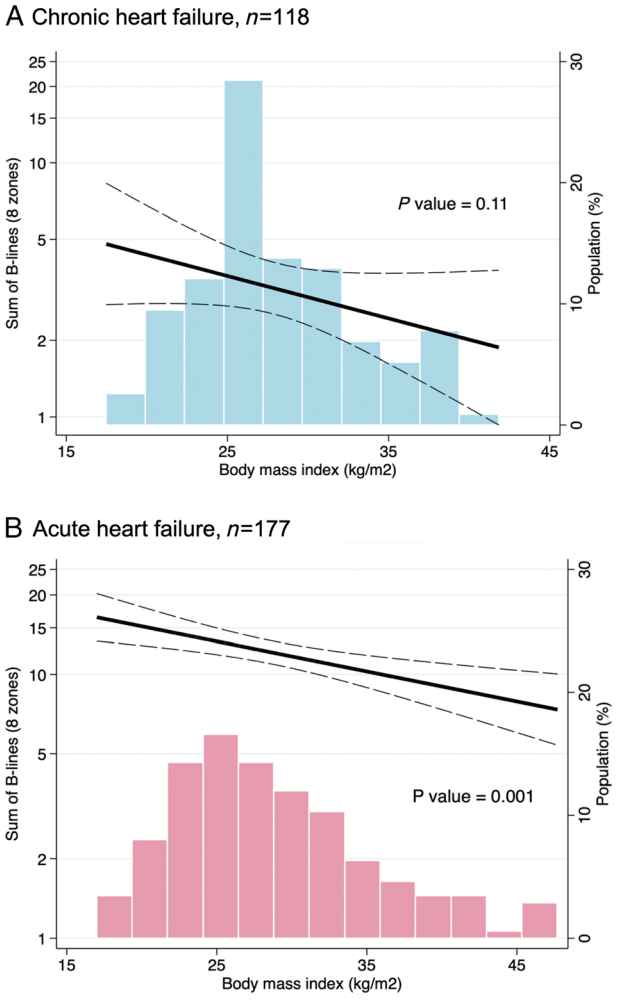
Figure 4 Association between sum of B-lines and body mass index. Cubic spline plots demonstrating the association between sum of B-lines and body mass index in (A) chronic heart failure and (B) acute heart failure. Black and blue lines: association correlate with 95% confidence intervals; histogram shows the distribution of body mass index.



congestion in dyspnoeic patients presenting to the Emergency Department.[18,19] We found that the maximum chest wall thickness at which ≥3 B-lines can be observed in the lateral zones 4 and 8 were 6.2 and 7.1 cm, respectively.

Combined heart failure cohort

B-lines and body mass index

In the combined HF cohort, the sum of B-lines decreased by 17% per 5 unit increase in BMI (95% CI −30% to −1%, P = 0.035). The type of HF (chronic vs. acute) did not affect this relationship (P for interaction = 0.58). Scatterplots of B-line number related to BMI in patients with chronic HF and AHF stratified by HFpEF and HFrEF are shown in Supporting Information, *Figure S2a* and *S2b*

Discussion

Obesity is increasingly common and poses a major diagnostic challenge in patients with suspected HF, in part because of the lower natriuretic peptide levels found in individuals with a very high BMI.[1,20–23] We hypothesized that LUS, an alternative method for assessing congestion, might be useful in the assessment of obese patients with chronic HF and AHF. We found that B-lines assessed by LUS were inversely associated with BMI, and in AHF, the number of B-lines declined to a lesser degree than NT-proBNP with increasing BMI.

Because higher BMI is associated with more thoracic subcutaneous adipose tissue,[24] the distance between the skin and the pleural line is greater in obese individuals, and this could impede the identification of B-lines during LUS examination. In this study, we found that higher BMI was associated with fewer B-lines in patients with HF. However, whether this was because LUS imaging was suboptimal or because obese patients had less congestion is impossible to say, in the absence of a gold standard for pulmonary congestion. Two findings suggest that impaired image quality was probably not the main explanation. Firstly, chest wall regions that were thickest (lateral zones 4 and 8) were also the zones in which the highest number of B-lines was found in AHF patients. Secondly, more than six B-lines were observed in half of AHF patients with severe obesity. This is important because ≥3 B-lines in ≥2 zones in each hemithorax is a recognized diagnostic cut-off for pulmonary oedema in patients presenting to the Emergency Department with undifferentiated dyspnoea.[13,18] The other key finding of relevance was that B-line number declined less steeply than NT-proBNP concentration with increasing BMI in AHF. Collectively, these observations suggest that LUS, a quick and non-invasive examination, probably has a useful role in evaluation of obese individuals with suspected (and proven) HF.

The usefulness of LUS appears to be true in both chronic HF and AHF, as well as HFrEF and HFpEF. Although the relationship between number of B-lines and BMI was not significant in patients with chronic HF, this is probably explained by the small number of very obese patients in the chronicHF cohort, and acuity of HF did not statistically modify the relationship between BMI and B-line count. We also found a similar relationship between BMI and B-lines in both HFrEF and HFpEF. This is important because approximately 80% of patients with HFpEF in the USA are overweight or obese.[25] These patients can be particularly difficult to diagnose and are the individuals in which natriuretic peptide levels can be less helpful as they may have levels within the normal range.

Prior research has demonstrated that B-line prevalence may vary according to chest zones.[26,27] We found that regional differences across the chest applied to B-lines and

their association with chest wall thickness and BMI. As these findings are from a relatively small cohort of AHF patients, we suggest that both parameters (BMI and chest wall thickness) should be taken into consideration when examining obese patients with suspected HF. This consideration is important as there are several different LUS imaging protocols involving different areas of the chest which, based on our data, may be differentially affected by chest wall thickness.

Limitations

This was a single centre study with a relatively small but well-defined cohort. We did not have natriuretic peptide measurements in most patients with chronic HF. Although patients in our AHF cohort had a clinical diagnosis of HF and had been admitted to hospital for treatment of HF, it is possible that the diagnosis was incorrect in some patients given the difficulties alluded to earlier. We did not standardize transducer frequencies (2–5 MHz) in our study protocol but rather allowed investigators to optimize imaging frequencies for each patient. Lower ultrasound frequencies can penetrate subcutaneous fat better and allow more easy visualization of deeper structures.[28] Positioning is known to affect the observed number of B-lines,[29] and this varied between the chronic HF (supine) and AHF (semirecumbent) cohort.

Conclusions

Among patients with HF, B-line number decreased with higher BMI and greater chest wall thickness. However, B-lines declined to a lesser degree than NT-proBNP with increasing BMI. These data suggest that LUS may be useful in patients with HF despite obesity.

Conflict of interest

Dr Brainin reports grants from Gangsted Foundation, grants from Herlev and Gentofte Hospitals internal funds, and grants from The Lundbeck Foundation, from The Augustinus Foundation, from The European Society of Cardiology, and from A.P. Møllers Lægefond, during the conduct of the study. Dr Biering-Sørensen reports nonfinancial support from Amgen, personal fees from Sanofi Pasteur, and personal fees from Novartis, during the conduct of the study. Dr Cheng reports grants from NIH, during the conduct of the study. Dr Solomon reports grants and personal fees from Alnylam, grants and personal fees from Amgen, grants and personal fees from AstraZeneca, grants from Bellerophon, grants and personal fees from BMS, grants from Celladon, grants and personal fees from Gilead, grants and personal fees from GSK, grants from Ionis, grants from Lone Star Heart, grants from Mesoblast, grants and personal fees from MyoKardia, grants from NIH/NHLBI, grants and personal fees from Novartis, grants from Sanofi Pasteur, grants and personal fees from Theracos, personal fees from Akros, grants and personal fees from Bayer, personal fees from Corvia, personal fees from Ironwood, personal fees from Merck, personal fees from Roche, personal fees from Takeda, personal fees from Quantum Genomics, personal fees from AoBiome, personal fees from Janssen, personal fees from Cardiac Dimensions, grants from Eidos, grants and personal fees from Cytokinetics, personal fees from Tenaya, personal fees from Daichi-Sankyo, personal fees from Cardurion, and personal fees from Eko.Ai, outside the submitted work. Dr Platz reports grants from NIH/NHLBI, during the conduct of the study.

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Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Figure S1. Lung ultrasound image with measurements of chest wall thickness.
Figure S2. B-lines and body mass index in chronic HF (a) and acute HF (b) stratified by HFpEF and HFrEF.
Table S1. Checklist for reporting of lung ultrasound studies in heart failure cohorts.
Table S2. Chest wall and B-line measurements by LUS zone in patients with acute HF (n=177).
Table S3. Association between B-lines and BMI and chest wall thickness in patients with acute HF (n=177).

References

1. Wang TJ, Larson MG, Levy D, Benjamin EJ, Leip EP, Wilson PWF, Vasan RS. Impact of obesity on plasma natriuretic peptide levels. *Circulation* 2004; **109**: 594–600.
2. Picano E, Pellikka PA. Ultrasound of extravascular lung water: a new standard for pulmonary congestion. *Eur Heart J* 2016; **37**: 2097–2104.
3. Platz E, Merz AA, Jhund PS, Vazir A, Campbell R, McMurray JJ. Dynamic changes and prognostic value of pulmonary congestion by lung ultrasound in acute and chronic heart failure: a systematic review. *Eur J Heart Fail* 2017; **19**: 1154–1163.
4. Pivetta E, Goffi A, Lupia E, Tizzani M, Porrino G, Ferreri E, Volpicelli G, Balzaretti P, Banderali A, Iacobucci A, Locatelli S, Casoli G, Stone MB, Maule MM, Baldi I, Merletti F, Cibinel GA, Baron P, Battista S, Buonafede G, Busso V, Conterno A, Paola DR, Ferrera P, Pecetto PF, Moiraghi C, Morello F, Steri F, Ciccone G, Calasso C, Caserta MA, Civita M, Condo' C, D'Alessandro V, Del Colle S, Ferrero S, Griot G, Laurita E, Lazzero A, Curto FL, Michelazzo M, Nicosia V, Palmari N, Ricchiardi A, Rolfo A, Rostagno R, Bar F, Boero E, Frascisco M, Micossi I, Mussa A, Stefanone V, Agricola R, Cordero G, Corradi F, Runzo C, Soragna A, Sciullo D, Vercillo D, Allione A, Artana N, Corsini F, Dutto L, Lauria G, Morgillo T, Tartaglino B, Bergandi D, Cassetta I, Masera C, Garrone M, Ghiselli G, Ausiello L, Barutta L, Bernardi E, Bono A, Forno D, Lamorte A, Lison D, Lorenzati B, Maggio E, Masi I, Maggiorotto M, Novelli G, Panero F, Perotto M, Ravazzoli M, Saglio E, Soardo F, Tizzani A, Tizzani P, Tullio M, Ulla M, Romagnoli E. Lung ultrasound-implemented diagnosis of acute decompensated heart failure in the ED. *Chest Elsevier* 2015; **148**: 202–210.
5. Prinz C, Voigt J-U. Diagnostic accuracy of a hand-held ultrasound scanner in routine patients referred for echocardiography. *J Am Soc Echocardiogr Mosby* 2011; **24**: 111–116.
6. Platz E, Lewis EF, Uno H, Peck J, Pivetta E, Merz AA, Hempel D, Wilson C, Frasure SE, Jhund PS, Cheng S, Solomon SD. Detection and prognostic value of pulmonary congestion by lung ultrasound in ambulatory heart failure patients. *Eur Heart J* 2016; **37**: 1244–1251.
7. Alpert MA, Kelly DL. Value and limitations of echocardiography in the assessment of obese patients. *Echocardiography* 1986; **3**: 260–272.
8. Dwyer KH, Merz AA, Lewis EF, Claggett BL, Crousillat DR, Lau ES, Silverman MB, Peck J, Rivero J, Cheng S, Platz E. Pulmonary congestion by lung ultrasound in ambulatory patients with heart failure with reduced or preserved ejection fraction and hypertension. *J Card Fail Elsevier Inc* 2018; **24**: 219–226.
9. Platz E, Campbell R, Claggett B, Lewis E, Groarke J, Docherty K, Lee M, Merz A, Silverman M, Swamy V, Lindner M, Rivero J, Solomon S, McMurray J. Lung ultrasound in acute heart failure: prevalence of pulmonary congestion and short- and long-term outcomes. *J Am Coll Cardiol HF* 2019; **7**: 859–861.
10. McMurray JJV, Adamopoulos S, Anker SD, Auricchio A, Böhm M, Dickstein K, Falk V, Filippatos G, Fonseca C, Gomez-Sanchez MA, Jaarsma T, Køber L, Lip GYH, Pietro MA, Parkhomenko A, Pieske BM, Popescu BA, Rønnevik PK, Rutten FH, Schwitter J, Seferovic P, Stepinska J, Trindade PT, Voors AA, Zannad F, Zeiher A, ESC Committee for Practice Guidelines. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012: The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology. Developed in collaboration with the Heart. *Eur Heart J* 2012; **33**: 1787–1847.
11. McKee PA, Castelli WP, McNamara PM, Kannel WB. The natural history of congestive heart failure: the Framingham study. *N Engl J Med* 1971; **285**: 1441–1446.
12. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, Flachskampf FA, Foster E, Goldstein SA, Kuznetsova T, Lancellotti P, Muraru D, Picard MH, Rietzschel ER, Rudski L, Spencer KT, Tsang W, Voigt J-U. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr* 2015; **28**: 1–39.e14.
13. Volpicelli G, Elbarbary M, Blaivas M, Lichtenstein DA, Mathis G, Kirkpatrick AW, Melniker L, Gargani L, Noble VE, Via G, Dean A, Tsung JW, Soldati G, Copetti R, Bouhemad B, Reissig A, Agricola E, Rouby JJ, Arbelot C, Liteplo A, Sargsyan A, Silva F, Hoppmann R, Breitkreutz R, Seibel A, Neri L, Storti E, Petrovic T. International evidence-based recommendations for point-of-care lung ultrasound. *Intensive Care Med* 2012; **38**: 577–591.
14. Platz E, Jhund PS, Girerd N, Pivetta E, McMurray JJV, Peacock WF, Masip J, Martin-Sanchez FJ, Miró Ò, Price S, Cullen L, Maisel AS, Vrints C, Cowie MR, DiSomma S, Bueno H, Mebazaa A, Gualandro DM, Tavares M, Metra M, Coats AJS, Ruschitzka F, Seferovic PM, Mueller C. Expert consensus document: reporting checklist for quantification of pulmonary congestion by lung ultrasound in heart failure. *Eur J Heart Fail* 2019: 1–8.
15. Sturm R. Increases in morbid obesity in the USA: 2000–2005. *Publ Health WB Saunders* 2007; **121**: 492–496.
16. Gustafsson M, Alehagen U, Johansson P. Imaging congestion with a pocket ultrasound device: prognostic implications in patients with chronic heart failure. *J Card Fail Elsevier Inc* 2015; **21**: 548–554.
17. Coiro S, Rossignol P, Ambrosio G, Carluccio E, Alunni G, Murrone A, Tritto I, Zannad F, Girerd N. Prognostic value of residual pulmonary congestion at discharge assessed by lung ultrasound imaging in heart failure. *Eur J Heart Fail* 2015; **17**: 1172–1181.
18. Pivetta E, Goffi A, Lupia E, Tizzani M, Porrino G, Ferreri E, Volpicelli G, Balzaretti P, Banderali A, Iacobucci A, Locatelli S, Casoli G, Stone MB, Maule MM, Baldi I, Merletti F, Cibinel GA. Lung ultrasound-implemented diagnosis of acute decompensated heart failure in the ED: a SIMEU multicenter study. *Chest* 2015; **148**: 202–210.
19. Pivetta E, Goffi A, Nazerian P, Castagno D, Tozzetti C, Tizzani P, Tizzani M, Porrino G, Ferreri E, Busso V, Morello F, Paglieri C, Masoero M, Cassine E, Bovaro F, Grifoni S, Maule MM, Lupia E, Paolo B, Alessia B, Giuseppina B, Andrea C, Andrea C, Ottavio D, Paola DR, Andrea E, Paolo FP, Patrizia F, Daniela F, Francesca G, Grazia GM, Sara G, Alda L, Davide L, Franca M, Franco M, Corrado M, Giulia N, Luca P, Sonia P, Paolo Q, Claudia S, Elisa S, Elisabetta S, Flavia S, Fabio S, Michela S, Francesca S, Cristina T, Grazia VM, Marco A, Laura B, Sofia B, Ernesta B, Barbara C, Matteo C, Melisenda C, Beatrice D, Grazia F, Roberto F, Giuseppe G, Chiara G, Simona G, Eriola H, Juri M, Federico M, Andrea N, Maddalena O, Andrea P, Giuseppe P, Stefano P, Michele R, Francesca S, Francesco TP, Federica T, Vanni S, Gabriele V. Lung ultrasound integrated with clinical assessment for the diagnosis of acute decompensated heart failure in the emergency department: a randomized controlled trial. *Eur J Heart Fail* 2019; **21**: 754–766.
20. Owan TE, Hodge DO, Herges RM, Jacobsen SJ, Roger VL, Redfield MM. Trends in prevalence and outcome of heart failure with preserved ejection fraction. *N Engl J Med* 2006; **355**: 251–259.
21. Nadruz W, Claggett BL, McMurray JJ, Packer M, Zile MR, Rouleau JL, Desai AS, Swedberg K, Lefkowitz M, Shi VC, Prescott MF, Solomon SD. Impact of body mass index on the accuracy of N-terminal pro-brain natriuretic peptide and brain natriuretic peptide for predicting outcomes in patients with chronic heart failure and reduced ejection fraction. *Circulation* 2016; **134**: 1785–1787.

22. Frankenstein L, Remppis A, Nelles M, Schaelling B, Schellberg D, Katus H, Zugck C. Relation of N-terminal pro-brain natriuretic peptide levels and their prognostic power in chronic stable heart failure to obesity status. *Eur Heart J* 2008; **29**: 2634–2640.
23. Horwich TB, Hamilton MA, Fonarow GC. B-type natriuretic peptide levels in obese patients with advanced heart failure. *J Am Coll Cardiol Elsevier Masson SAS* 2006; **47**: 85–90.
24. Secombe P, Sutherland R, Johnson R. Body mass index and thoracic subcutaneous adipose tissue depth: possible implications for adequacy of chest compressions. *BMC Res Notes BioMed Central* 2017; **10**: 1–5.
25. Kitzman DW, Brubaker P, Morgan T, Haykowsky M, Hundley G, Kraus WE, Eggebeen J, Nicklas BJ. Effect of caloric restriction or aerobic exercise training on peak oxygen consumption and quality of life in obese older patients with heart failure with preserved ejection fraction: a randomized clinical trial. *JAMA* 2016; **315**: 36–46.
26. Volpicelli G, Caramello V, Cardinale L, Mussa A, Bar F, Frascisco MF. Bedside ultrasound of the lung for the monitoring of acute decompensated heart failure. *Am J Emerg Med* 2008; **26**: 585–591.
27. Cortellaro F, Ceriani E, Spinelli M, Campanella C, Bossi I, Coen D, Casazza G, Cogliati C. Lung ultrasound for monitoring cardiogenic pulmonary edema. *Intern Emerg Med Springer Milan* 2017; **12**: 1011–1017.
28. Modica MJ, Kanal KM, Gunn ML. The obese emergency patient: imaging challenges and solutions. *Radiographics* 2011; **31**: 811–823.
29. Frasure SE, Matilsky DK, Siadecki SD, Platz E, Saul T, Lewiss RE. Impact of patient positioning on lung ultrasound findings in acute heart failure. *Eur Heart J Acute Cardiovasc Care* 2015; **4**: 326–332.