Physician accuracy for diagnosing heart failure in unselected patients hospitalised with dyspnoea

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ABSTRACT

Aim: To examine variables associated with heart failure (HF) and determine accuracy of Emergency Department (ED) physicians to diagnose HF in a Norwegian teaching hospital without point-of-care natriuretic peptide testing.

Methods and results: We included 150 consecutive patients hospitalised for dyspnoea and collected the results of clinical examination and the probability of HF (0-100%) from the ED physicians. HF was adjudicated according to guidelines by two independent senior physicians. Of 150 patients, 68 patients (45%) were diagnosed with HF as the primary cause of the hospitalisation. HF patients were older (75.1 vs. 68.0 y, $p<0.001$) and more likely to be male (57% vs. 37%, $p=0.01$). There was no difference in New York Heart Association functional class or the duration of symptoms prior to hospitalization between HF patients and patients with non-HF dyspnoea. Several clinical variables previously reported to be predictive of HF were associated with HF by crude analysis in our patients, but only history of HF (HR 11.14 [OR 2.73-45.47], $p<0.001$), history of hypertension (HR 3.22 [OR 1.12-9.22], $p=0.03$), and atrial fibrillation (HR 3.22 [OR 1.21-10.58], $p=0.02$) were independently associated with HF in multivariate analysis. The area under the curve for ED physician diagnosis of HF was 0.85 (95% CI 0.79-0.91, $p<0.001$ vs. chance).

Conclusion: The accuracy of the ED physicians for diagnosing HF was sub-optimal in this cohort of mainly elderly subjects hospitalised for dyspnoea. Physician should be aware of the increased likelihood of HF in dyspneic patients with atrial fibrillation, history of hypertension, or history of HF.

INTRODUCTION

Dyspnoea is a cardinal symptom in several conditions associated with substantial morbidity and mortality, including heart failure (HF) and chronic obstructive pulmonary disease (COPD). Patients with dyspnoea and other symptoms of HF constitute a significant proportion of patients admitted to general medical wards[1]. The prevalence of HF related hospitalisations is especially high in elderly subjects[2], however, determining the correct diagnosis in patients with dyspnoea may be difficult, especially in the acute setting. With an increasing prevalence of both HF and COPD in the Western world due to an aging population[3,4,5] and the high prevalence of smoking in the last decades, the number of patients hospitalised for dyspnoea is likely to rise. Hence, differentiating between patients with HF and non-HF dyspnoea is of major clinical relevance.

Several clinical signs have been reported to be associated with a diagnosis of HF in the acute setting, including raised jugular venous pressure, hepatomegaly, pulmonary crackles and rales, tachypnoea, tachycardia, peripheral oedema and cardiac murmurs[6]. However, extrapolating from the non-acute setting, these established signs of HF may be less prominent in HF patients with preserved ejection fraction (HFpEF)[7]. Additional diagnostic tools like electrocardiogram (ECG), chest x-ray, arterial blood gas analysis, and general laboratory analysis may be helpful in identifying HF, while echocardiography, although a fundamental modality to diagnose myocardial dysfunction, may not be generally available in the Emergency Department (ED). The accuracy of the ED physician to diagnose HF has previously been examined in two large studies that also assessed the merit of the B-type natriuretic peptides as HF biomarkers[8,9], but due to the selection of patients and change in demographics, the age distribution in these studies may not be representative of the patients found in a general ED. Moreover, although recommended in most updated HF guidelines[6],

not all hospitals have implemented point-of-care natriuretic peptide testing due to local traditions and practical and financial reasons, which is expected to influence diagnostic accuracy[8,9]. Accordingly, in this study we wanted to (1) explore clinical variables associated with HF in mainly elderly subjects hospitalised for dyspnoea and (2) test the accuracy of ED physicians to diagnose HF in a European teaching hospital without point-of-care natriuretic peptide testing.

METHODS

Study population

We included consecutive patients with dyspnoea admitted to the Division of Medicine, Akershus University Hospital, which is a Norwegian teaching hospital with a catchment area of 460 000 people. The physicians working in the ED were all junior staff within the first years of their residency. More experienced physicians were also on call, but not necessarily present in the ED. Patients were identified by dedicated study personnel who attended all briefings between sets. The criteria for study eligibility were age ≥18 years, dyspnoea considered as the primary cause for hospitalisation by the ED physician, and time from hospital admission to study inclusion <24 h. Exclusion criteria were dementia or other cause not enabling informed patient consent, disseminated malignant disease, and acute myocardial infarction, coronary intervention, or major surgery within the last 2 weeks. This study is also designed to explore biomarkers and patients with haemoglobin levels <10 g/dL or incomplete study baseline blood sampling due to technical issues were excluded. We included patients Monday-Thursday 8.00 a.m.-2 p.m. from June 2009-November 2010 and classified patients according to time from admission and approached the patients with shortest duration first. The study was conducted according to the Declaration of Helsinki, approved by the Regional Ethics Committee, and all patients provided informed consent before study commencement.

Data collection

Clinical information was recorded directly from the ED physician at the start of the briefings in between sets by the use of a standardised questionnaire. The questionnaire included questions on the duration of dyspnoea prior to admission, New York Heart Association (NYHA) functional class, respiratory frequency, cyanosis, raised jugular venous pressure, heart murmurs and third heart sound, attenuation over lungs, inspiratory rales, pathology in the ECG, hepatomegaly, ascites, and peripheral oedemas. The ED physicians were also requested to annotate the probability of acute HF as the primary cause of dyspnoea on a scale from 0% (unlikely) to 100% (very likely). Blood pressure, heart rate, and body temperature on hospital admission were collected directly from the patient records. Finally, clinical information was also obtained directly from the patients by dedicated study personnel according to a second and more comprehensive questionnaire, with questions relating to status prior to the admission, family medical history, co-morbidities, and smoking status. Previous medical history and medication were also checked against medical records. Paroxysmal, persistent and chronic atrial fibrillation were grouped together. Weight and height were collected directly from the patient or from the patient records, and body mass index was calculated by weight (Kg)/ [height (m)]^2. Left ventricular ejection fraction (LVEF) was collected from routine clinical echocardiography in the patient medical records that were performed <12 months of the hospitalisation. Creatinine and haemoglobin were determined on hospital admission by standard biochemical methods and creatinine clearance was calculated by the Cockcroft-Gault formula[10].

Adjudication of diagnosis

The final diagnoses of the index hospitalisation was determined by two independent senior physicians, who reviewed all medical records, including follow-up data. The admissions were

classified as (1) HF admission, (2) non-HF cause of dyspnoea for the index hospitalization but myocardial dysfunction (e.g. in the case of pneumonia in a HF patient), and (3) non-HF admission. For this study the latter two groups were merged as non-HF cause of dyspnoea, which was the pre-specified primary endpoint in studies on diagnostic accuracy. HF was based on the criteria proposed by the European Society of Cardiology requiring typical signs and symptoms of HF and objective evidence of structural or functional myocardial abnormality[6]. The endpoint committee also separated the HF group into HF with systolic dysfunction (LVEF<50%) and HFpEF (LVEF≥50%).

Statistics

Data are presented as median (quartile [Q]1-3) or absolute numbers and percentages. Continuous variables were assessed for normal distribution by the Kolmogorov-Smirnov one sample test and differences between groups examined by the Student´s *t*-test or the Mann-Whitney *U* test as appropriate. Categorical data were compared by the Chi-square test. Clinical and laboratory factors associated with a diagnosis of HF were examined by univariate and multivariate logistic regression analysis with odds ratios (OR) presented with 95% confidence intervals (CI). Variables associated with HF in univariate analysis were included in the multivariate model (backward selection of variables). Due to collinearity between history of HF and clinical signs of HF, we also performed a second multivariate analysis without past medical history. Diagnostic accuracy of HF was assessed by receiver-operating characteristics (ROC) curve analysis [25] with area under the curve (AUC) presented with 95% confidence interval (CI). A *P* value<0.05 was considered statistically significant. Statistical analyses were performed with SPSS for Windows version 16.0 (SPSS, Chicago, IL) with the exception of the comparison of ROC AUCs, which was performed with MedCalc for Windows, version 9.5.1.0 (MedCalc Software, Mariakerke, Belgium).

RESULTS

Patient characteristics stratified by diagnosis

Patients diagnosed with HF were older and more likely to be male compared to the patients admitted with non-HF dyspnoea (Table 1). The prevalence of previous cardiovascular events and interventions, atrial fibrillation, hypertension, and diabetes mellitus were higher among patients hospitalised with HF, while the prevalence of COPD was lower. Patients with HF had lower creatinine clearance and more frequent use of β-blockers, angiotensin-converting enzyme inhibitors (ACEI), loop diuretics, statins, warfarin, acetyl salicylic acid (ASA), and digitalis compared to the other patients, while the use of short- and long-acting β_2-agonist for inhalation (ipratropium bromide and tiotropium bromide) and theophylline were more prevalent among patients with non-HF dyspnoea. Heart murmurs, peripheral oedema, pathology in the ECG, and reduced creatinine clearance were also more prevalent in patients with HF, while more patients in the non-HF group had cough on admission (Table 2). In patients diagnosed with HF, mean LVEF was 41±2% and 25 (37%) of the patients were classified as HFpEF.

Clinical factors associated with a diagnosis of HF

Heart murmurs, peripheral oedema, pathology in the ECG, and creatinine clearance were positively associated with a diagnosis of HF in univariate analysis, while cough on admission was associated with non-HF related cause of dyspnoea (Table 3). Moreover, history of HF, previous acute myocardial infarction, coronary arterial bypass grafting, coronary artery disease, hypertension, diabetes mellitus, and patients with atrial fibrillation were more likely to be diagnosed with HF, while more patients with history of COPD were hospitalised due to a non-HF cause (Table 3). In multivariate analysis, history of HF, history of hypertension, and atrial fibrillation were independently associated with HF (Table 3). Excluding past medical

history from the model, male gender, heart murmurs, peripheral oedema, and pathology in the ECG were all associated with a diagnosis of HF in multivariate analysis.

ED physician accuracy for diagnosing HF

ED physicians scored a higher probability of HF in patients classified as HF hospitalisations compared to patients hospitalised for non-HF dyspnoea: 55.4±3.5% vs. 19.5±1.7%, p<0.001 (Table 2). The AUC of the ED physicians for diagnosing HF in the whole cohort (n=150) was 0.85 (95% CI 0.79-0.91), p<0.001 vs. chance (Figure). Examining physician assessment in patients with systolic dysfunction (n=43) and HFpEF (n=23) separately, ED physicians scored probability of HF higher in patients with systolic dysfunction than in patients with HFpEF: 60.5±4.4% vs. 46.8±5.4%, p=0.054. The AUC of the ED physicians to differentiate HF patients with systolic dysfunction from patients with non-HF related dyspnoea (AUC 0.91 [95% CI 0.83-0.95] was also different from the AUC of HFpEF patients vs. non-HF related dyspnoea (AUC 0.84 [95% CI 0.74-0.91]). Moreover, excluding patients with history of HF (n=48), the AUC of the ED physicians to identify *de novo* HF (n=28) was 0.79 (95% CI 0.69-0.86).

DISCUSSION

The principal result of our study is the sub-optimal accuracy of the ED physicians to diagnose HF in our cohort of mainly elderly patients hospitalised for dyspnoea. We identify several clinical variables associated with HF in univariate analysis, but only atrial fibrillation, history of hypertension, and history of HF were independently associated with HF in our patients.

There are many clinical signs reported to be associated with HF, including jugular vein distension, cardiac murmurs and third heart sound, pulmonary attenuation and rales, and peripheral oedema[6]. However, in our study, only cardiac murmurs and peripheral oedema were found to be associated with HF. In general, two factors may have influenced the association between the other clinical variables and HF in our patients. First, we explored an aging population in which substantial co-morbidity may discern the association between pulmonary attenuation and rales and HF. This could be particularly relevant for COPD patients with pulmonary rales due to fibrosis. Moreover, aging in itself may also be associated with pulmonary fibrosis. Secondly, we included a large proportion of patients with HFpEF. Most previous studies examining clinical signs of HF have been performed in patients with systolic dysfunction, hence the clinical signs considered indicative of HF could be different in patients with HFpEF. This is supported by recent data from a Dutch HF outpatient registry with equal distribution of systolic dysfunction and HFpEF, in which 32% of the patients diagnosed with HF did not have typical clinical signs of HF[7]. The inclusion of history of HF to the model, a factor that by nature is closely correlated to clinical signs of HF, will also influence the performance of clinical variables in multivariate regression analysis. Hence, we also performed analysis without past medical history in the model and found male gender, cardiac murmurs, peripheral oedema, and ECG pathology to be associated with HF in multivariate analysis. All of these variables have previously also been found predictive of

HF[6,9], but still, we cannot preclude that the physicians in our study failed to detect important clinical signs that could have helped them in identifying HF. We did not provide special instructions regarding clinical assessment or during the annotation of ED physician probability, thus our results should be valid for the real-life situation of mainly young and less experienced physicians performing the initial evaluation in the ED.

We found that the ED physicians in our study demonstrated sub-optimal accuracy in diagnosing HF. Similar to our study, two large studies have previously examined clinical variables associated with, and the accuracy of clinical assessment in the evaluation of HF in patients with dyspnoea. In the Rapid Measurement of B-type Natriuretic Peptide in the Emergency Diagnosis of Heart Failure (Breathing Not Properly) study the utility of BNP to diagnose HF was compared to the accuracy of the NHANES and the Framingham criteria[11], two commonly used algorithms to assess HF probability. In line with our results of sub-optimal physician accuracy to diagnose HF, the NHANES and Framingham criteria performed poorly in the Breathing Not Properly Study yielding AUCs of 0.67 and 0.73, respectively[8]. Moreover, analogous to our results, history of HF was found to be independently associated with HF in multivariate regression analysis together with several clinical signs of HF and a BNP level≥100 pg/mL. Compared to the Breathing Not Properly study, our patients were older and with more co-morbidity, which may influence the association between results on clinical evaluation and HF. The N-terminal Pro-BNP Investigation of Dyspnea in the Emergency Department (PRIDE) study also examined diagnostic accuracy of HF in patients with dyspnoea. In the PRIDE study, analogous to our study, ED physicians annotated the probability of HF on a scale from 0-100%. The PRIDE study was a single-centre study performed at Massachusetts General Hospital (Boston, MA) and reported an AUC of 0.90 to differentiate HF from non-HF dyspnoea based on clinical

assessment alone. Several factors may explain the apparent superior accuracy of physician assessment in the PRIDE study compared to our study. First, the patients admitted with non-HF dyspnoea were considerably younger in the PRIDE study (mean 56.9 y) compared to our patients with non-HF dyspnoea (mean 68.0 y). Secondly, the proportion of patients with a diagnosis of COPD were considerably lower in the PRIDE study compared to our study, not least pertaining to 47% of our HF patients having a history of COPD compared to 25% in the PRIDE study. The prevalent co-morbidity between COPD and HF in our study is expected to influence accuracy by enhancing the complexity of the diagnosis in these patients[12]. In the PRIDE study, patients with renal insufficiency were also excluded, while we excluded very few patients based on co-morbidity. The lower morbidity of the non-HF dyspnoea patients in PRIDE compared to our study is also evident from lower prevalence of loop diuretics (16% vs. 30%) and medication blocking the angiotensin system (15% vs. 32%) in the PRIDE study. Moreover, the prevalence of HFpEF is not reported in the PRIDE study, but a low prevalence of HFpEF in the PRIDE study will account for some of the discrepancy. Of note, the AUC of the ED physicians in our study to discriminate HF patients with systolic dysfunction from patients with non-HF dyspnoea was comparable to the AUC of the PRIDE study. Finally, analogous to our results and the Breathing Not Properly study, history of HF seemed also in the PRIDE study to be a strong indicator of new HF hospitalisations. The difference in prevalence of hypertension in HF patients and non-HF patients in the PRIDE study was also comparable to our results, while the PRIDE study did not report prevalence of atrial fibrillation.

The high prevalence of HFpEF and elderly patients in our study makes this study different from previous work on ED physician accuracy of HF in patients with dyspnoea. This is also relevant for the interpretation of our results. Hypertension and atrial fibrillation are prevalent

in and considered causal factors of progress to diastolic dysfunction and HFpEF[13]. Extrapolating this to the clinical setting, physicians responsible for the assessment of elderly patients with dyspnoea should be especially alert to a diagnosis of HFpEF in patients with chronic hypertension and atrial fibrillation. Given the lower accuracy to detect HFpEF compared to systolic dysfunction, this could represent a possible improvement in the evaluation of elderly patients with dyspnoea. Finally, although the BNPs are implemented in guidelines as important for diagnosing HF, the utility of these peptides in elderly patients with HFpEF and substantial co-morbidity seem more limited[14,15] and this should be tested and validated in new cohorts that may compare more to the patients in most general EDs.

In conclusion, a high proportion of elderly patients hospitalised with HF have preserved ejection fraction, and this influences the clinical variables associated with a diagnosis of HF. Moreover, the ED physicians demonstrate sub-optimal accuracy in separating HF patients from patients with non-HF dyspnoea, which may be improved by special focus on HFpEF in patients with atrial fibrillation, history of hypertension, and history of HF.

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DISCLOSURES

There are no disclosures relating to this manuscript.

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Table 1. Descriptive statistics of patients hospitalised for dyspnoea

	Acute HF (n=68)	Non-HF related dyspnoea (n=82)	p
Age, years (mean ± SEM)	75.1±1.2	68.0±1.3	<0.001
Male sex (n, %)	39 (57%)	30 (37%)	0.01
Body mass index	26.4±0.8	25.5±0.9	0.47
Duration from start of dyspnoea (n, %)			0.28
0-6 hrs	6 (8.8%)	7 (8.6%)	
6-12 hrs	4 (5.9%)	7 (8.6%)	
12-24 hrs	2 (2.9%)	7 (8.6%)	
1-2 days	6 (8.8%)	7 (8.6%)	
3-7 days	19 (27.9%)	30 (37%)	
> 7 days	31 (45.6%)	23 (28.4%)	
NYHA functional class (n, %)			0.70
II	11 (16%)	13 (16%)	
III	23 (34%)	33 (40%)	
IV	34 (50%)	36 (44%)	
LVEF, % (mean ± SEM) (n=61)	41±2		
HF, systolic dysfunction	43 (63%)		

HF, preserved ejection fraction	25 (37%)		
History of (n, %)			
HF	40 (59%)	8 (10%)	<0.001
Coronary artery disease	36 (53%)	21 (26%)	0.001
Myocardial infarction	29 (43%)	20 (24%)	0.018
PCI	20 (29%)	14 (17%)	0.07
CABG	12 (18%)	1 (1%)	<0.001
Hypertension	37 (54%)	26 (32%)	0.005
Chronic or paroxysmal atrial fibrillation	30 (44%)	12 (15%)	<0.001
Diabetes mellitus	16 (24%)	9 (11%)	0.04
COPD	32 (47%)	57 (70%)	0.005
Medication (n, %)			
β-blocker	39 (57%)	28 (35%)	0.005
Ca^{2+} channel blocker	13 (19%)	13 (16%)	0.62
ACEI	30 (44%)	15 (19%)	0.001
ARB	13 (19%)	11 (14%)	0.36
ACEI or ARB	40 (59%)	26 (32%)	0.001
Aldosterone antagonist	9 (13%)	5 (6%)	0.14
Loop diuretics	46 (68%)	24 (30%)	<0.001
Statin	36 (53%)	22 (27%)	0.001
Warfarin	21 (31%)	12 (15%)	0.02
ASA	31 (46%)	23 (28%)	0.03
Clopidogrel	7 (10%)	6 (7%)	0.53

Digitalis	10 (15%)	0 (0%)	<0.001
Amiodarone	1 (2%)	1 (1%)	0.90
Slow release nitrate	6 (9%)	6 (7%)	0.75
Proton pump inhibitor	11 (16%)	17 (21%)	0.45
Short-acting β_2-agonist, inhalation	20 (29%)	39 (48%)	0.02
Ipratropium bromide	18 (27%)	35 (43%)	0.03
Long-acting β_2-agonist, inhalation	23 (34%)	37 (46%)	0.14
Tiotropium bromide	4 (6%)	14 (17%)	0.03
Corticosteroids, inhalation	19 (28%)	31 (38%)	0.18
Corticosteroids, oral	8 (12%)	15 (19%)	0.26
Theophylline	0 (0%)	9 (11%)	0.005
Insulin	9 (13%)	5 (6%)	0.14
Anti-diabetic medication, oral	9 (13%)	5 (6%)	0.14

Abbreviations: NYHA: New York Heart Association; HF: heart failure; LVEF: left ventricular ejection fraction; PCI: percutaneus coronary intervention; CABG: coronary artery bypass grafting; COPD: chronic obstructive pulmonary disease; ACE: angiotensin-converting enzyme inhibitor; ARB: angiotensin II receptor blocker; and ASA: acetyl salicylic acid.

Table 2. Clinical characteristics on admission for patients hospitalised for dyspnoea

	Acute HF (n=68)	Non-HF related dyspnoea (n=82)	P
Heart rate, b.p.m. (mean±SEM)	91.37±2.92	94.09±2.34	0.46
Systolic BP, mmHg (mean±SEM)	148±3.6	150±3.1	0.61
Diastolic BP, mmHg (mean±SEM)	82±2	80±1.7	4.21
Respiration frequency (/min)			0.40
<12	1 (1.5%)	0 (0%)	
12-16	14 (20.6%)	14 (17.3%)	
16-30	47 (69.1%)	54 (66.7%)	
>30	6 (8.8%)	13 (16%)	
Cyanosis			0.44
Yes	7 (10.4%)	13 (16.7%)	
No	60 (89.6%)	65 (83.3%)	
Orthopnoea			0.43
Yes	41 (60.3%)	42 (53.8%)	
No	27 (39.7%)	36 (46.2%)	
Fever (>38°C), (n, %)			0.72
Yes	5 (7.4%)	7 (9%)	
No	63 (92.6%)	71 (91%)	
Raised jugular venous pressure			0.54

Yes	6 (9%)	5 (6.2%)	
No	61 (91%)	75 (93.8%)	
Heart murmurs			0.003
No murmur	51 (76.1%)	76 (95%)	
Systolic murmur	15 (22.4%)	3 (3.8%)	
Diastolic murmur	1 (1.5%)	1 (1.2%)	
Gallop rhythm			0.27
Yes	1 (1.5%)	0 (0%)	
No	65 (98.5%)	80 (100%)	
Pulmonary attenuation			0.13
None	50 (73.5%)	70 (86.4%)	
Lower third	14 (20.6%)	7 (8.6%)	
Midfield	4 (5.9%)	3 (3.7%)	
Apex	0 (0%)	1 (1.2%)	
Pulmonary rales			0.18
None	23 (34.3%)	39 (50%)	
Lower third	28 (41.8%)	20 (25.6%)	
Midfield	13 (19.4%)	15 (19.2%)	
Apex	3 (4.5%)	4 (5.1%)	
Couch on hospital admission			0.026
Yes	38 (56.7%)	60 (74.1%)	
No	29 (43.3%)	21 (25.9%)	
Hepatomegaly			0.11
Yes	4 (6.1%)	1 (1.2%)	

No	62 (93.9%)	80 (98.8%)	
Ascites			0.88
Yes	1 (1.5%)	1 (1.2%)	
No	64 (98.5%)	80 (98.8%)	
Peripheral oedema			0.002
Yes	37 (54.4%)	24 (29.6%)	
No	31 (45.6%)	57 (70.4%)	
Normal ECG			0.013
Yes	13 (20%)	31 (39.2%)	
No	52 (80%)	48 (60.8%)	
Creatinine clearance (ml/min) (mean±SEM)	65.7±4.4	82.6±3.4	0.003
Haemoglobin, g/dL (mean±SEM)	13.6±0.2	13.7±0.2	0.72
Cigarette smoking			0.19
No, never	17 (26.6%)	11 (14.3%)	
Yes, including the last 3 months	15 (23.4%)	20 (26.0%)	
Yes, quit more than 3 months ago	32 (50%)	46 (59.7%)	
ED physician probability of acute heart failure as cause of hospitalisation (mean±SEM)	55.4±3.5	19.5±1.7	< 0.001

Abbreviations: BP: blood pressure; NYHA: New York Heart Association; HF: heart failure; ED: Emergency Department; ECG: electrocardiogram.

Table 3. Clinical variables associated with a diagnosis of heart failure

Univariate analysis

	Odds ratio	95 % CI.	P value
Age (y)	1.06	1.03-1.10	< 0.001
Male	2.33	1.21-4.50	0.012
Body mass index	1.02	0.97-1.06	0.47
Duration of dyspnoea	0.61	0.28-1.36	0.23
NYHA functional class 1-3 vs. class 4	1.28	0.67-2.44	0.46
History of			
HF	13.21	5.51-31.69	<0.001
Coronary artery disease	3.27	1.64-6.50	0.001
Myocardial infarction	2.31	1.15-4.63	0.019
PCI	2.02	0.93-4.40	0.08
CABG	17.36	2.19-137.31	0.007
Hypertension	2.57	1.32-5.01	0.005
Chronic or paroxysmal atrial fibrillation	4.61	2.12-10.02	< 0.001
Diabetes mellitus	2.50	1.02-6.08	0.044
COPD	0.39	0.20-0.76	0.006
Angina pectoris	1.41	0.51-3.88	0.51
Asthma	0.73	0.23-2.36	0.60
Pulmonary disorder, other	0.51	0.15-1.73	0.28
Cyanosis	0.58	0.22-1.56	0.28
Orthopnoea	1.30	0.67-2.52	0.43

Heart rate, b.p.m	1.00	0.98-1.01	0.46
Systolic BP, mmHg	0.92	0.48-1.76	0.79
Diastolic BP, mmHg	0.91	0.44-1.85	0.78
Respiration frequency (/min)	1.24	0.55-2.83	0.61
Fever (>38° C)	0.81	0.24-2.66	0.72
Raised jugular venous pressure	1.48	0.43-5.07	0.54
Heart murmurs	5.96	1.88-18.86	0.002
Pulmonary attenuation	2.29	1.00-5.27	0.051
Pulmonary attenuation > basis	1.20	0.29-5.00	0.80
Pulmonary rales	1.91	0.98-3.75	0.06
Pulmonary rales > basis	0.97	0.45-2.10	0.95
Cough on hospital admission	0.46	0.23-0.92	0.028
Hepatomegaly	5.16	0.56-47.34	0.15
Ascites	1.25	0.08-20.38	0.88
Peripheral oedemas	2.84	1.44-5.57	0.002
Abnormal ECG	0.39	0.18-0.83	0.014
Haemoglobin (g/dL)	0.96	0.78-1.19	0.72
Creatinine clearance, mL/min	0.40	0.18-0.87	0.021
Current smoker	0.87	0.40-1.89	0.73
Previous smoker	0.67	0.35-1.32	0.25

Abbreviations: Hx: history; HF: heart failure; CABG: coronary artery bypass grafting; PCI: percutaneous coronary intervention; COPD: chronic obstructive pulmonary disease; BP: blood pressure; ECG: electocardiography; NYHA: New York Heart Association.

Multivariate analysis, all variables

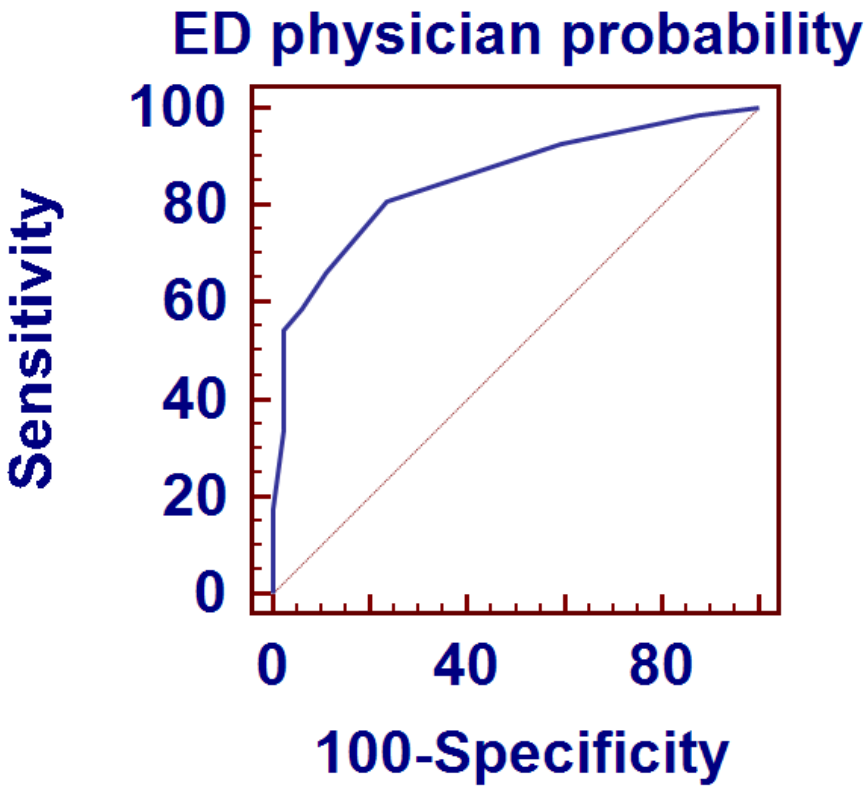
	Odds ratio	**95 % CI.**	***P* value**
History of			
HF	11.14	2.73-45.47	0.001
Hypertension	3.22	1.12-9.22	0.03
Chronic or paroxysmal atrial fibrillation	3.57	1.21-10.58	0.021

Multivariate analysis, past medical history excluded

	Odds ratio	**95 % CI.**	***P* value**
Male gender	3.81	1.64-8.83	0.002
Heart murmurs	4.54	1.18-17.43	0.028
Peripheral oedemas	3.03	1.31-7.02	0.010
Normal ECG	0.36	0.15-0.90	0.029

Abbreviations: HF: heart failure; ECG: electrocardiography.

Figure.


ED physician probability
Sensitivity
100
80
60
40
20
0
0
40
80
100-Specificity