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RESEARCH ARTICLE

Decongestion strategies in patients presenting with acutely decompensated heart failure: A worldwide survey among physicians

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Aims Decongestion strategies for acute decompensated heart failure (ADHF) characterized by volume overload differ widely. The aim of this independent international academic web-based survey was to capture the therapeutic strategies that physicians use to treat ADHF and to assess differences in therapeutic approaches between cardiologists versus non-cardiologists.

Methods and results Physicians were invited to complete a web-based questionnaire, capturing anonymized data on physicians' characteristics and treatment preferences based on a hypothetical clinical scenario of a patient hospitalized with ADHF. A total of 641 physicians from 60 countries participated. A wide variation in the management of the patient was observed. There was conservative use of diuretics, i.e. only 7% started intravenous furosemide at a dose ≥2 times the baseline oral dose, and infrequent use of ultrasound in assessing congestion (20.4%). Spot urinary sodium was infrequently or never measured by ≥85% of physicians. A third considered a patient with ongoing oedema as being stabilized. There were significant differences between cardiologists and non-cardiologists in the management of ADHF, the targets for daily body weight loss and urine output, diuretic escalation strategies (66.3% vs. 40.7% would escalate diuresis by adding a thiazide) and assessment of response to treatment (27.0% vs. 52.9% considered patients with minimal congestion as stabilized).

Conclusions There is substantial variability amongst physicians and between cardiologists and non-cardiologists in the management of patients with ADHF, with regard to clinical parameters used to tailor treatment, treatment goals, diuretic dosing and escalation strategies.

Keywords Acute heart failure • Diuretics

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Introduction

Congestion represents a hallmark feature of acute decompensated heart failure (ADHF) and predominates the clinical presentation of nearly 85% of patients hospitalized for this diagnosis.[1] Although approaches to optimize volume status, such as modifying fluid and salt intake, may play a complementary role,[2] use of loop diuretics remains the mainstay of treatment for congested patients with ADHF, regardless of the underlying left ventricular ejection fraction (LVEF).[3] Characteristically, more than 85% of patients with ADHF receive an intravenous diuretic during hospitalization.[4] The 2021 European guidelines on heart failure (HF) recommend the use of diuretics for patients with signs and symptoms of congestion (recommendation class I, level of evidence C).[5] Moreover, a position statement by the Heart Failure Association (HFA) of the European Society of Cardiology (ESC) proposed an algorithm for diuretic use in ADHF reserving a central role for urine sodium measurement to tailor treatment and has been incorporated in the guidelines.[2] However, evidence-based data on optimal decongestion strategies in patients with ADHF are lacking; the few randomized controlled trials were performed several years ago and included small study populations.[6–8] Diuretics may exert a range of adverse effects including electrolyte depletion, hyperuricemia, orthostatic hypotension, neurohormonal activation and renal function deterioration.[3] Especially the latter, generally assessed by serum creatinine serial measurements, is a frequent reason for premature and inappropriate cessation of diuretic treatment and lack of initiation of evidence-based medical treatment with modulators of renin–angiotensin–aldosterone system, though it has been well documented that congestion is the main underlying mechanism of kidney function deterioration during ADHF.[9] Consequently, decongestion strategies in patients with ADHF seem to differ widely in terms of type and dose of diuretic used, administration of combinations of diuretics, and use of other pharmacological and non-pharmacological modalities with potential decongestive effects (thiazides, dopamine, mineralocorticoid receptor antagonists, acetazolamide, tolvaptan, ultrafiltration, etc.).[10] Hints for this are provided by the variable rates of residual congestion at hospital discharge among patients with ADHF enrolled in real-world registries,[11] albeit the well-established, detrimental association between residual congestion and outcomes in patients with both acute and chronic HF.[11–13]

This variation in treatment strategies may probably account, at least in part, for the variable outcomes of patients with ADHF across different countries in terms of mortality and HF hospitalizations.[14,15]

Thus, it would be of extreme scientific interest to record physician-related attitudes and preferences when faced with the treatment of a congested patient with ADHF.

The objective of the current survey was to register the therapeutic strategies that physicians implement to treat patients with ADHF presenting with volume overload. In particular, through this survey we (i) collected data on decongestive treatment preferences in a scenario of ADHF, (ii) assessed differences in therapeutic approaches among physicians, and (iii) investigated for differences in decongestive treatment preferences between physicians self-identified as cardiologists versus non-cardiologists.

Methods

Study design

The survey was constructed through a collaboration between the investigators from Royal Brompton and Harefiled Hospitals, part of Guys and St Thomas's NHS Foundation Trust, United Kingdom and the National Heart Failure Societies (NHFS) Committee of the HFA. In our survey, investigators disseminated the questionnaire to the physicians who treat patients with ADHF. The NHFS Presidents from ESC and affiliated countries were also contacted in order to distribute the questionnaire to such physicians. Physicians were contacted via phone, e-mail and social media and were provided a link to an online website (Survey Monkey, sponsored by the HFA of the ESC) to answer several open- and closed-ended questions regarding a hypothetical case of a patient with ADHF. Each physician was asked to complete the survey once and submission of a second response via the same computer IP was not permitted. Data collection started in October 2021 and ended in April 2022.

The survey complied with the European General Data Protection Regulation (GDPR) 2016/679. Any personal data processed in connection with this survey were anonymized and treated confidentially. Material and data collected from the survey will be analysed by the ESC and kept for a maximum of 24 months for analysis and quality control purposes.

Study parameters

The questionnaires were anonymized and captured data on (i) physicians' characteristics (Grade [consultant or trainee], years of training in general internal medicine and/or cardiology, specialty [recorded as 'cardiology' and 'non-cardiology'], country of practice, city of practice, type of hospital the physician works in [recorded as 'academic' and 'non-academic'], number of patients with ADHF they treated over the month preceding the survey, timing of their involvement in the treatment of patients with ADHF), and (ii) treatment preferences based on a hypothetical clinical scenario of a patient admitted to hospital with ADHF. The clinical scenario and the questionnaire can be found in online supplementary material.

Statistical analysis

Baseline characteristics and management strategies of physicians who participated in the survey are presented as counts (percentage) and compared with the chi-square test or Fisher's exact test (if chi-square was not applicable). A p-value <0.05 was considered statistically significant for all comparisons.

Results

From 14 October 2021 to 16 April 2022, 641 physicians from 60 countries completed the study questionnaire.

Participants' characteristics

Most answers were received by physicians in the United Kingdom (108 [16.8%]), followed by Turkey (99 [15.4%]), Romania

Table 1 Baseline characteristics of the study cohort ($n = 641$)

Cardiology specialty, *n* (%)	501 (78.2)
Consultant level, *n* (%)	523 (81.6)
Academic hospital, *n* (%)	448 (69.1)
Internal medicine experience (for non-cardiology consultants), *n* (%)	
<1 year	0 (0)
1–3 years	26 (28.0)
4–5 years	20 (21.5)
>5 years	47 (50.5)
Internal medicine training (for non-cardiology trainees), *n* (%)	
<1 year	8 (20.0)
1–3 years	19 (47.5)
4–5 years	10 (25.0)
>5 years	3 (7.5)
Cardiology experience (for cardiology consultants), *n* (%)	
<1 year	29 (6.7)
1–3 years	68 (15.8)
4–5 years	39 (9.1)
>5 years	294 (68.4)
Cardiology training (for cardiology trainees), *n* (%)	
<1 year	10 (14/7)
1–3 years	27 (39.7)
4–5 years	16 (23.5)
>5 years	15 (22.1)

(89 [13.9%]), and Greece (60 [9.4%]). The exact distribution of answers per countries is depicted in online supplementary *Table S1*. Among the participating physicians, 501 (78.2%) had cardiology as their primary specialty and 523 (81.6%) were at consultant level. The baseline characteristics of the study cohort are shown in *Table 1*.

Participants' responses to the clinical scenario

The responses of physicians regarding the management of the hypothetical clinical case are depicted in *Table 2*. A total of 588 (91.7%) participating physicians reported being aware of different HF phenotypes and 594 (92.7%) identified the patient in the scenario provided as a case of ADHF. The majority of physicians (79.2%) reported that they are typically involved in the management of ADHF patients within 12 h of their initial presentation to the emergency department. However, most participants did not manage ADHF patients frequently, as 478(74.6%) reported treating less than 10 patients with similar clinical profile over a 1-month period. Less than 25% of clinicians reported using lung ultrasound (LUS) and/or inspiratory variation of the inferior vena cava (IVC) to guide their management (*Table 2*, *Figure 1A*). In terms of initial diuretic dosing, only 7% of the participants would start the patient on intravenous furosemide at a dose ≥2 times the baseline oral dose. Among non-pharmacological measures for management of ADHF patients, more than one quarter of the respondents did not enforce fluid and salt restriction to the patients and more than half did not discuss with the patient strategies of managing increased urine output (*Table 2*, *Figure 1B*). Importantly, physicians seemed willing to maintain guideline-directed medical therapy (GDMT), as ≤10% would discontinue any of these medications with spironolactone being the agent more likely to be withheld. Although only 3.3% of the participating clinicians targeted a daily body weight (BW) loss of ≤0.5 kg (*Table 2*, *Figure 1C*), more than one third were reluctant to further increase furosemide dose when the patient's BW loss did not meet their target. On the other hand, approximately one fourth would reduce furosemide dose if serum creatinine increased, given that target daily drop in BW was achieved. When needing to further escalate diuresis, the most popular option was sequential nephron blockade with addition of a thiazide (58.3%), followed by addition or increase of spironolactone (22.5%) and increase of furosemide dose (14.8%). The most common measure of diuretic response was reportedly urine output (88.2%), followed by BW (75.1%), whereas spot urine sodium was infrequently or never measured by more than 85% of the responders. More than 85% of participants reported feeling confident in managing such patients, albeit almost one third of them considered patients with ongoing congestion as stabilized. The proportion of physicians who were aware of international and national guidelines on managing ADHF was 86.6% and 64.9%, respectively. Finally, less than half of the participants reported having a specific protocol of managing patients hospitalized with HF in their hospital/trust.

Participants' responses to the clinical scenario stratified by specific characteristics

Specialty

Significant differences in the responses to the ADHF clinical scenario were noted between clinicians with specialty of cardiology versus non-cardiology (*Table 3*). The former reported being more often aware of the different acute HF (AHF) phenotypes (94.2% vs. 84.9%, $p < 0.001$) and a higher proportion could correctly identify the scenario provided as a case of ADHF (93.8% vs. 88.6%, $p = 0.022$). As expected, clinicians with cardiology as specialty were more frequently involved ($p < 0.001$) in the management of such patients and even more so in the hyperacute setting (within 2 h of presentation, $p = 0.002$). Regarding patient management, cardiologists reported using N-terminal pro-B-type natriuretic peptide (NT-proBNP), LUS and/or inspiratory variation of the IVC more often and chest x-ray less often to guide their management compared with non-cardiologists ($p < 0.001$). On the other hand, cardiologists seem to abide less often to non-pharmacological measures, such as daily patient weights (78.8% vs. 90.7%, $p = 0.001$) and discussing with patients strategies to manage increased urine output (44.9% vs. 60.7%, $p < 0.001$) compared with non-cardiologists, while no significant difference was noted between them in the initial dosing of intra-hospital loop diuretics ($p = 0.066$). Cardiologists also seemed to target for higher daily weight loss (>1 kg) more often than non-cardiologists (60.5% vs. 40.0%, $p < 0.001$).

Table 2 Responses of physicians regarding the management of the hypothetical clinical case

	Missing values N(%)	N(%)			
Are you aware of different phenotypes of acute HF?	3 (0.5)				
Yes		588 (91.7)			
No		50 (7.8)			
What phenotype of acute HF would this patient fall into?	0 (0.0)				
Acute decompensated HF		594 (92.7)			
Acute coronary syndrome with decompensated HF		24 (3.7)			
Cardiogenic shock		5 (0.8)			
Acute pulmonary oedema		18 (2.8)			
How many patients with this similar presentation have you treated in the last month?	0 (0.0)				
0		40 (6.2)			
1–5		242 (37.8)			
5–10		196 (30.6)			
10–20		105 (16.4)			
>20		58 (9.0)			
When would you be first involved in this patient's care after the initial presentation to ED?	2 (0.3)				
Within 2 h		265 (41.3)			
Within 2–12 h		243 (37.9)			
Within 12–24 h		74 (11.5)			
After the first 24 h		57 (8.9)			
N/A		2 (0.3)			
What clinical signs do you use to guide your treatment?	0 (0.0)				
Clinical signs and CXR		282 (44.0)			
Clinical signs and LUS		22 (3.4)			
Clinical signs and NT-proBNP		206 (32.1)			
Clinical signs, LUS and inspiratory variation of IVC		131 (20.4)			
What diuretic regimen would you start this patient on?	0 (0.0)				
PO Furosemide 80 mg + 40 mg		10 (1.6)			
PO Furosemide 80 mg BD		7 (1.1)			
PO Furosemide 120 mg BD		3 (0.5)			
PO Bumetanide 2 mg + 1 mg		2 (0.3)			
PO Bumetanide 2 mg BD		3 (0.5)			
PO Bumetanide 3 mg BD		3 (0.5)			
IV Furosemide 120 mg BD		121 (18.9)			
IV Furosemide 160 mg/24 h		235 (36.7)			
IV Furosemide 240 mg/24 h		212 (33.1)			
IV Furosemide 360 mg/24 h		22 (3.4)			
IV furosemide 480 mg/24 h		23 (3.6)			
What non-pharmacological management options would you consider? (select all that apply)	0 (0.0)				
Daily weights		522 (81.4)			
Careful fluid balance and urine output monitoring		580 (90.5)			
Discuss with the patient the best strategies of managing increased urine output		310 (48.4)			
Fluid and salt restriction		473 (73.8)			
		No change	**Decrease**	**Increase**	**Discontinue**
At this point what changes to his regular medications would you do?					
Carvedilol 25 mg BD	8 (1.2)	436 (68.0)	102 (15.9)	62 (9.7)	33 (5.1)
Sacubitril/valsartan 97/103 mg BD	8 (1.2)	459 (71.6)	88 (13.7)	25 (3.9)	61 (9.5)
Spironolactone 25 mg OD	10 (1.6)	373 (58.2)	25 (3.9)	169 (26.4)	64 (10.0)
Atorvastatin 40 mg OD	6 (0.9)	574 (89.5)	23 (3.6)	11 (1.7)	27 (4.2)
Allopurinol 100 mg OD	10 (1.6)	544 (84.9)	14 (2.2)	8 (1.2)	65 (10.1)
Aspirin 75 mg OD	8 (1.2)	611 (95.3)	8 (1.1)	11 (1.7)	11 (1.7)
Omeprazole 20 mg OD	10 (1.6)	585 (91.3)	4 (0.6)	12 (1.9)	30 (4.7)

Table 2 (Continued)

		No change	Decrease	Increase	Discontinue
The patient has lost 0.5 kg weight over the last 24 h, Cr 186 μmol/L (2.1 mg/dl). BP remains unchanged and the patient is well perfused. What would you do next?	2 (0.3)				
Make no change		198 (30.9)			
Increase the diuretic dose		392 (61.2)			
Reduce the diuretic dose		44 (6.9)			
Discontinue diuretics		5 (0.8)			
Following the previous question, what weight loss would you target over a 24 h period?	3 (0.5)				
0–0.5 kg		21 (3.3)			
0.5–1 kg		258 (40.2)			
1–2 kg		260 (40.6)			
>2 kg		99 (15.4)			
The next day the patient achieved the desired weight loss, but remained clinically overloaded with an increase in Cr to 210 (2.4 mg/dl) from 186 μmol/L. BP remained stable. What would you do?	3 (0.5)				
Reduce the loop diuretic dose		147 (22.9)			
Keep the loop diuretic dose the same		410 (64.0)			
Discontinue diuretics		8 (1.2)			
Increase the loop diuretic dose		73 (11.4)			
The following day (day 3), the patient remains clinically overloaded and the weight has not dropped. What would you do?	1 (0.2)				
Make no change		27 (4.2)			
Increase the loop diuretic dose		144 (22.5)			
Add in thiazide diuretic		374 (58.3)			
Increase (or restart) spironolactone		95 (14.8)			
How do you assess diuretic response in a patient with decompensated HF?	0 (0.0)				
Measure daily urinary output and body weight		426 (66.5)			
Measure daily body weight		55 (8.6)			
Measure urinary spot sodium		21 (3.3)			
Measure urinary spot sodium and urinary output		139 (21.7)			
How common is it in your practice to measure urinary spot sodium?	0 (0.0)				
It is measured in each patient		11 (1.7)			
Very common		71 (11.1)			
Quite uncommon		199 (31.0)			
Never		360 (56.2)			
Which strategy for decongestion are you using in clinical practice to safely discharge a HF patient?	1 (0.2)				
Improving signs and symptoms, decreasing body weight and decreasing radiological congestion		375 (58.5)			
Improving dyspnoea, decreasing body weight and decreasing pulmonary congestion at LUS		110 (17.2)			
Improving signs and symptoms and decreasing NT-proBNP at discharge		146 (22.8)			
I am not using any strategy for decongestion		9 (1.4)			
When do you consider your patient 'stabilized'?	0 (0.0)				
No signs of congestion and preserved SBP		85 (13.3)			
No signs of congestion, preserved SBP and improved renal function (Cr/BUN)		347 (54.1)			
Minimal signs of congestion but preserved SBP and improved renal function		185 (28.9)			
Minimal signs of congestion but preserved SBP		24 (3.7)			

Table 2 (Continued)

		No change	Decrease	Increase	Discontinue
When would you refer this patient to HF service?	2 (0.3)				
Day 1		159 (24.8)			
Day 3		43 (6.7)			
After stabilized in-hospital		185 (28.9)			
Would not refer		14 (2.2)			
Would only refer if complication occurred		31 (4.8)			
Would refer as outpatient		83 (12.9)			
No HF service available		124 (19.3)			
Do you feel confident in managing patients with acute decompensated HF?	2 (0.3)				
Yes		550 (85.8)			
No		89 (13.9)			
Does your hospital/trust have a protocol on managing patients hospitalized with HF?	1 (0.2)				
Yes		300 (46.8)			
No		340 (53.0)			
Are you aware of any international guidelines on managing patients with HF and congestion?	0 (0.0)				
Yes		555 (86.6)			
No		86 (13.4)			
Are you aware of any national guidelines on managing patients with HF and congestion?	1 (0.2)				
Yes		416 (64.9)			
No		224 (34.9)			

Values are given as *n* (%).
BP, blood pressure; BD, twice daily; BUN, blood urea nitrogen; Cr, creatinine; CXR, chest x-ray; ED, emergency department; HF, heart failure; IV, intravenous; IVC, inferior vena cava; LUS, lung ultrasound; N/A, not available; NT-proBNP, N-terminal pro-B-type natriuretic peptide; OD, once daily; PO, per os; SBP, systolic blood pressure.

In regard to diuretic escalation preferences, cardiologists were more likely to add on a thiazide (63.3% vs. 40.7%) but less likely to increase (or restart) spironolactone (12.0% vs. 25.0%) or further increase the loop diuretic dose compared with non-cardiologists (20.6% vs. 29.3%, $p < 0.001$ for interaction; *Table 3*, *Figure 2*). There was a significant overlap of physicians who presented with good diuretic management practices (initial dosing ≥2 times the oral loop diuretic dose, escalation of dose when diuresis targets were not met and no de-escalation of dose when diuresis targets were met, albeit an increase in serum creatinine), a finding consistent in both cardiologists and non-cardiologists (*Figure 3*). Compared with non-cardiologists, cardiologists more often assessed response to treatment via urine output combined with spot urine sodium (24.4% vs. 12.1%) but less often via BW only (6.6% vs. 15.7%, $p < 0.001$ for interaction). A smaller proportion of cardiologists considered patients with ongoing signs of congestion as stabilized compared with non-cardiologists (27.0% vs. 52.9%, $p < 0.001$) (*Table 3*, *Figure 4*), while cardiologists reported that they would refer such patients to HF services less often and at a later stage compared with non-cardiologists ($p < 0.001$).

Cardiologists were more frequently aware of international (94.2% vs. 59.3%, $p < 0.001$) and national guidelines (68.9% vs. 50.7%, $p < 0.001$), and hospital/trust protocols (52.1% vs. 27.9%, $p < 0.001$) on managing ADHF compared with non-cardiologists (*Table 3*, *Figure 5*). Finally a higher proportion of cardiologists reported feeling confident in managing such patients compared with non-cardiologists (90.2% vs. 70.0%, $p < 0.001$).

Type of hospital

A few significant differences in the responses to the ADHF clinical scenario were noted between clinicians working in an academic versus a non-academic hospital (online supplementary *Table S2*). In particular, clinicians in academic centres preferred starting a 160 mg furosemide infusion as an initial dosing scheme, while clinicians in non-academic centres preferred a 240 mg furosemide infusion ($p = 0.003$ for interaction). Clinicians in academic centres were keener to escalate diuretic treatment compared with their colleagues when daily BW loss target was not reached (no change in diuretics in 27.7% vs. 37.1%, $p = 0.092$). Regarding methodology of assessing diuretic response, they were less likely to use BW change on its own (6.3% vs. 14%) and more likely to use urinary spot sodium and urinary output (23.9% vs. 16.7%, $p = 0.038$ for interaction). Finally, clinicians in academic centres tended to refer patients less often and at a later stage to HF services ($p = 0.059$) and were more often aware of hospital protocols for management of AHF (50.4% vs. 38.2%, $p = 0.073$).

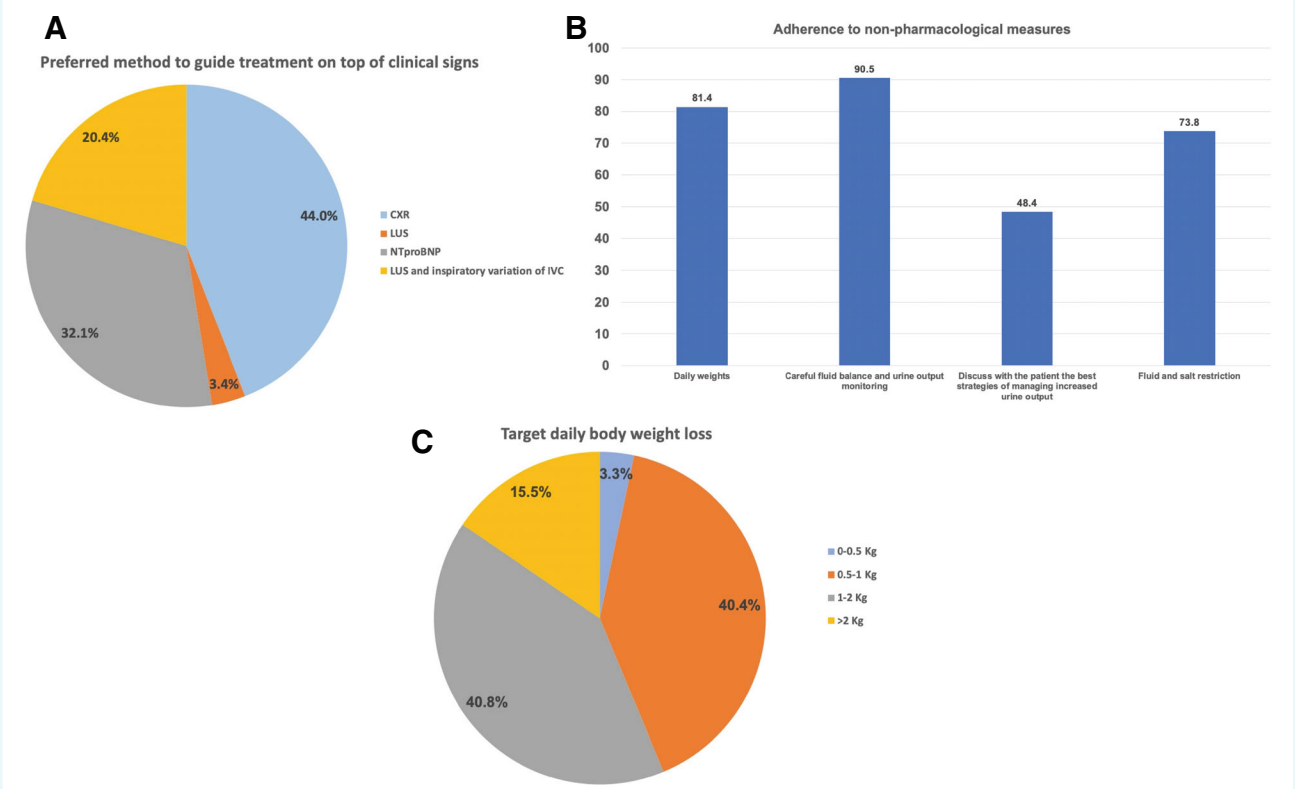

Figure 1 (*A*) Preferred method to guide treatment on top of clinical signs, given as percentage of the 641 physicians. (*B*) Adherence to various non-pharmacological measures, given as percentage of the 641 physicians. (*C*) Target daily body weight loss, given as percentage of the 641 physicians. CXR, chest x-ray; IVC, inferior vena cava; LUS, lung ultrasound; NTproBNP, N-terminal pro-B-type natriuretic peptide.

Years of experience

Among cardiology consultants.

A few significant differences in the responses to the ADHF clinical scenario were noted between cardiology consultants with less versus more than 5 years of experience (online supplementary *Table S3*). In particular, less experienced consultants were more likely to be guided in their management by LUS and inspiratory variation of the IVC but less likely to be guided by NT-proBNP compared with more experienced consultants (27.2% vs. 21.8% and 26.5% vs. 37.8%, respectively, $p = 0.045$). They were also more likely to discontinue sacubitril/valsartan ($p < 0.001$) and spironolactone ($p = 0.024$) but less likely to discontinue the beta-blocker ($p = 0.014$) compared with their more experienced colleagues (online supplementary *Table S3*). Interestingly, they were less aware of hospital protocols on AHF (44.9% vs. 56.5%, $p = 0.025$) and international guidelines on HF and congestion (90.4% vs. 95.6%, $p = 0.038$).

Cardiology versus non-cardiology consultants with >5 years of experience.

Some differences in the responses to the ADHF clinical scenario were demonstrated between cardiology and non-cardiology consultants with >5 years of experience. Specifically, cardiologists were more frequently aware of the different HF phenoytpes (94.9% vs. 84.8%, $p = 0.01$) and more likely to be guided in their management by LUS, inspiratory variation of the IVC and NT-proBNP (61.9% vs. 40.4%) but less likely to be guided by chest x-ray on top of clinical signs compared with non-cardiologists (38.1% vs. 59.6%, $p = 0.008$). They were less likely to discontinue sacubitril/valsartan at admission (3.4% vs. 17%, $p = 0.002$) but more likely to aim at a daily BW loss >1 kg (58.8% vs. 32%) and to add a thiazide to overcome diuretic resistance (60.2% vs. 38.3%, $p = 0.034$) compared to non-cardiologists. Moreover, they were more often aware of national (70.4% vs. 48.9%, $p = 0.012$) and international guidelines (95.6% vs. 72.3%, $p < 0.001$) and hospital protocols (56.5% vs. 29.8%, $p < 0.001$) compared with non-cardiologists.

Cardiology versus non-cardiology consultants with <5 years of experience.

A few significant differences in the responses to the ADHF clinical scenario were noted between cardiology and non-cardiology consultants with less than 5 years of experience. Namely, cardiologists were more likely to be guided in their management by LUS, inspiratory variation of the IVC and NT-proBNP (59.6% vs. 28.3%) but less likely to be guided by chest x-ray on top of clinical signs compared with non-cardiologists (40.4% vs. 71.7%, $p = 0.002$). They were more likely to add a thiazide to overcome

Table 3 Responses to the clinical scenario among clinicians with specialty of cardiology vs. non-cardiology

	Missing values	Non-cardiology	Cardiology	*p*-value
Are you aware of different phenotypes of acute HF?	3 (0.5)			<0.001
Yes		118 (84.9)	470 (94.2)	
No		21 (15.1)	29 (5.8)	
What phenotype of acute HF would this patient fall into?	0 (0.0)			0.022
Acute decompensated HF		124 (88.6)	470 (93.8)	
Acute coronary syndrome with decompensated HF		5 (3.6)	19 (3.8)	
Cardiogenic shock		2 (1.4)	3 (0.6)	
Acute pulmonary oedema		9 (6.4)	9 (1.8)	
How many patients with this similar presentation have you treated in the last month?	0 (0.0)			<0.001
0		19 (13.6)	21 (4.2)	
1–5		63 (45.0)	179 (35.7)	
5–10		36 (25.7)	160 (31.9)	
10–20		17 (12.1)	88 (17.6)	
>20		5 (3.6)	53 (10.6)	
When would you be first involved in this patient's care after the initial presentation to ED?	2 (0.3)			0.002
Within 2 h		46 (32.9)	219 (43.7)	
Within 2–12 h		73 (52.1)	170 (33.9)	
Within 12–24 h		12 (8.6)	62 (12.4)	
After the first 24 h		8 (5.7)	49 (9.8)	
N/A		1 (0.7)	1 (0.2)	
What clinical signs do you use to guide your treatment?	0 (0.0)			<0.001
Clinical signs and CXR		91 (65.0)	191 (38.1)	
Clinical signs and LUS		4 (2.9)	18 (3.6)	
Clinical signs and NT-proBNP		34 (24.3)	172 (34.3)	
Clinical signs, LUS and inspiratory variation of IVC		11 (7.9)	120 (24.0)	
What diuretic regimen would you start this patient on?	0 (0.0)			0.066
PO Furosemide 80 mg + 40 mg		4 (2.9)	6 (1.2)	
PO Furosemide 80 mg BD		3 (2.1)	4 (0.8)	
PO Furosemide 120 mg BD		1 (0.7)	2 (0.4)	
PO Bumetanide 2 mg + 1 mg		1 (0.7)	1 (0.2)	
PO Bumetanide 2 mg BD		1 (0.7)	2 (0.4)	
PO Bumetanide 3 mg BD		1 (0.7)	2 (0.4)	
IV Furosemide 120 mg BD		32 (22.9)	89 (17.8)	
IV Furosemide 160 mg/24 h		34 (24.3)	201 (40.1)	
IV Furosemide 240 mg/24 h		54 (38.6)	158 (31.5)	
IV Furosemide 360 mg/24 h		6 (4.3)	16 (3.2)	
IV Furosemide 480 mg/24 h		3 (2.1)	20 (4.0)	
What non-pharmacological management options would you consider? (select all that apply)	0 (0.0)			
Daily weights		127 (90.7)	395 (78.8)	0.001
Careful fluid balance and urine output monitoring		130 (92.9)	450 (89.8)	0.279
Discuss with the patient the best strategies of managing increased urine output		85 (60.7)	225 (44.9)	<0.001
Fluid and salt restriction		102 (72.9)	371 (74.1)	0.776

Table 3 (Continued)

	Discontinue	Decrease	No change	Increase	Discontinue	Decrease	No change	Increase	
At this point what changes to his regular medications would you do?									
Carvedilol 25 mg BD	8 (1.2)	12 (8.6)	12 (8.6)	92 (65.7)	22 (15.7)	21 (4.2)	90 (18.0)	344 (68.7)	40 (8.0)	0.002
Sacubitril/valsartan 97/103 mg BD	8 (1.2)	23 (16.4)	12 (8.6)	95 (67.9)	8 (5.7)	38 (7.6)	76 (15.2)	364 (72.7)	17 (3.4)	0.006
Spironolactone 25 mg OD	10 (1.6)	17 (12.1)	4 (2.9)	67 (47.9)	49 (35.0)	47 (9.4)	21 (4.2)	306 (61.1)	120 (24.0)	0.039
Atorvastatin 40 mg OD	6 (0.9)	7 (5.0)	1 (0.7)	128 (91.4)	2 (1.4)	20 (4.0)	22 (4.4)	9 (1.8)	446 (89.0)	0.286
Allopurinol 100 mg OD	10 (1.6)	15 (21.0)	2 (1.4)	113 (80.7)	2 (1.4)	44 (8.8)	12 (2.4)	431 (86.0)	6 (1.2)	0.280
Aspirin 75 mg OD	8 (1.2)	3 (2.1)	0 (0.0)	130 (92.9)	5 (3.6)	8 (1.6)	0 (0.0)	481 (96.0)	6 (1.2)	0.268
Omeprazole 20 mg OD	10 (1.6)	6 (4.3)	1 (0.7)	129 (92.1)	2 (1.4)	24 (4.8)	3 (0.6)	456 (91.0)	10 (2.0)	0.989
The patient has lost 0.5 kg weight over the last 24 h, Cr 186 µmol/L (2.1 mg/dl). BP remains unchanged and the patient is well perfused. What would you do next?	2 (0.3)									0.338
Make no change		42 (30.0)				156 (31.1)				
Increase the diuretic dose		91 (65.0)				301 (60.1)				
Reduce the diuretic dose		6 (4.3)				38 (7.6)				
Discontinue diuretics		0 (0.0)				5 (1.0)				
Following the previous question, what weight loss would you target over a 24 h period?	3 (0.5)									<0.001
0–0.5 kg		6 (4.3)				15 (3.0)				
0.5–1 kg		76 (54.3)				182 (36.3)				
1–2 kg		45 (32.1)				215 (42.9)				
>2 kg		11 (7.9)				88 (17.6)				
The next day the patient achieved the desired weight loss, but remained clinically overloaded with an increase in Cr to 210 (2.4 mg/dl) from 186 µmol/L. BP remained stable. What would you do?	3 (0.5)									0.941
Reduce the loop diuretic dose		31 (22.1)				116 (23.2)				
Keep the loop diuretic dose the same		90 (64.3)				320 (63.9)				
Discontinue diuretics		1 (0.7)				7 (1.4)				
Increase the loop diuretic dose		17 (12.1)				56 (11.2)				
The following day (day 3), the patient remains clinically overloaded and the weight has not dropped. What would you do?	1 (0.2)									<0.001
Make no change		6 (4.3)				21 (4.2)				
Increase the loop diuretic dose		41 (29.3)				103 (20.6)				
Add in thiazide diuretic		57 (40.7)				317 (63.3)				
Increase (or restart) spironolactone		35 (25.0)				60 (12.0)				

Table 3 (Continued)

		Discontinue	Decrease	No change	Increase	Discontinue	Decrease	No change	Increase	
How do you assess diuretic response in a patient with decompensated HF?	0 (0.0)									<0.001
Measure daily urinary output and body weight				96 (68.6)				330 (65.9)		
Measure daily body weight				22 (15.7)				33 (6.6)		
Measure urinary spot sodium				5 (3.6)				16 (3.2)		
Measure urinary spot sodium and urinary output				17 (12.1)				122 (24.4)		
How common is it in your practice to measure urinary spot sodium?	0 (0.0)									0.740
It is measured in each patient				2 (1.4)				9 (1.8)		
Very common				13 (9.3)				58 (11.6)		
Quite uncommon				48 (34.3)				151 (30.1)		
Never				77 (55.0)				283 (56.5)		
Which strategy for decongestion are you using in clinical practice to safely discharge a HF patient?	1 (0.2)									0.001
Improving signs and symptoms, decreasing body weight and decreasing radiological congestion				96 (68.6)				279 (55.7)		
Improving dyspnoea, decreasing body weight and decreasing pulmonary congestion at LUS				23 (16.4)				87 (17.4)		
Improving signs and symptoms and decreasing NT-proBNP at discharge				16 (11.4)				130 (25.9)		
I am not using any strategy for decongestion				4 (2.9)				5 (1.0)		
When do you consider your patient 'stabilized'?	0 (0.0)									<0.001
No signs of congestion and preserved SBP				17 (12.1)				68 (13.6)		
No signs of congestion, preserved SBP and improved renal function (Cr/BUN)				49 (35.0)				298 (59.5)		
Minimal signs of congestion but preserved SBP and improved renal function				68 (48.6)				117 (23.4)		
Minimal signs of congestion but preserved SBP				6 (4.3)				18 (3.6)		
When would you refer this patient to HF service?	2 (0.3)									<0.001
Day 1				58 (41.4)				101 (20.2)		
Day 3				13 (9.3)				30 (6.0)		
After stabilized in-hospital				36 (25.7)				149 (29.7)		
Would not refer				0 (0.0)				14 (2.8)		
Would only refer if complication occurred				5 (3.6)				26 (5.2)		
Would refer as outpatient				17 (12.1)				66 (13.2)		
No HF service available				10 (7.1)				114 (22.8)		

Table 3 (Continued)

		Discontinue	Decrease	No change	Increase	Discontinue	Decrease	No change	Increase	
Do you feel confident in managing patients with acute decompensated HF?	2 (0.3)									<0.001
Yes				98 (70.0)				452 (90.2)		
No				42 (30.0)				47 (9.4)		
Does your hospital/trust have a protocol on managing patients hospitalized with HF?	1 (0.2)									<0.001
Yes				39 (27.9)				261 (52.1)		
No				100 (71.4)				240 (47.9)		
Are you aware of any international guidelines on managing patients with HF and congestion?	0 (0.0)									<0.001
Yes				83 (59.3)				472 (94.2)		
No				57 (40.7)				29 (5.8)		
Are you aware of any national guidelines on managing patients with HF and congestion?	1 (0.2)									<0.001
Yes				71 (50.7)				345 (68.9)		
No				69 (49.3)				155 (30.9)		

Values are given as *n* (%).
BP, blood pressure; BD, twice daily; BUN, blood urea nitrogen; Cr, creatinine; CXR, chest x-ray; ED, emergency department; HF, heart failure; IV, intravenous; IVC, inferior vena cava; LUS, lung ultrasound; N/A, not available; NT-proBNP, N-terminal pro-B-type natriuretic peptide; OD, once daily; PO, per os; SBP, systolic blood pressure.

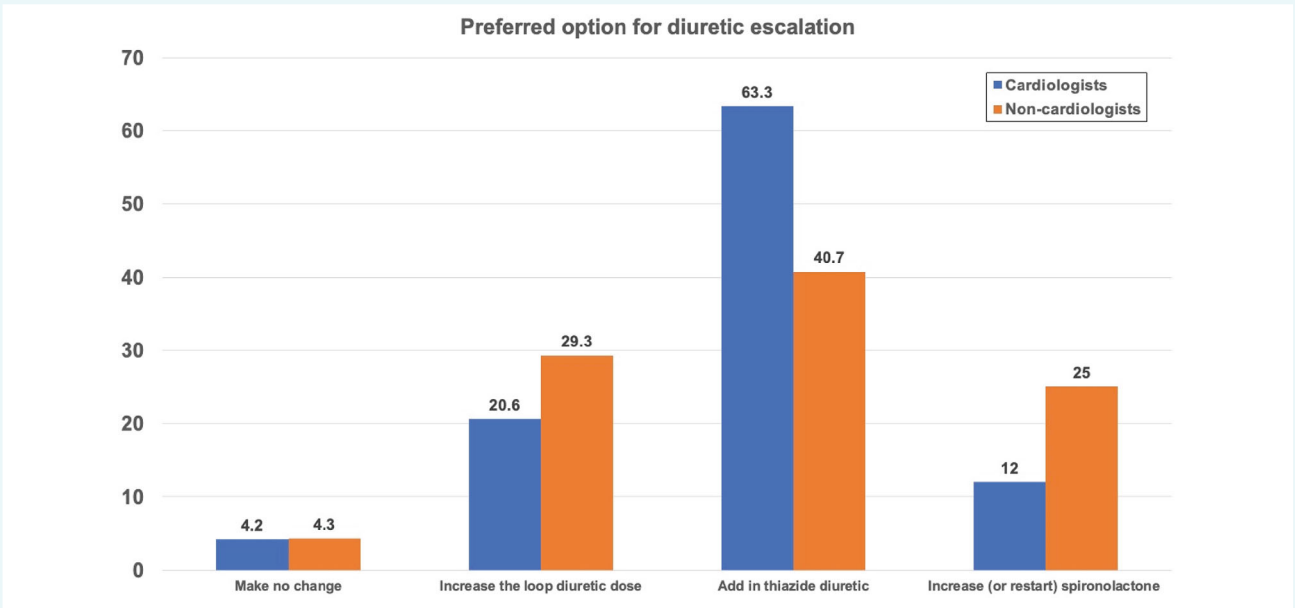

Figure 2 Differences between cardiologists and non-cardiologist in their preferred option for diuretic escalation, given as percentages.

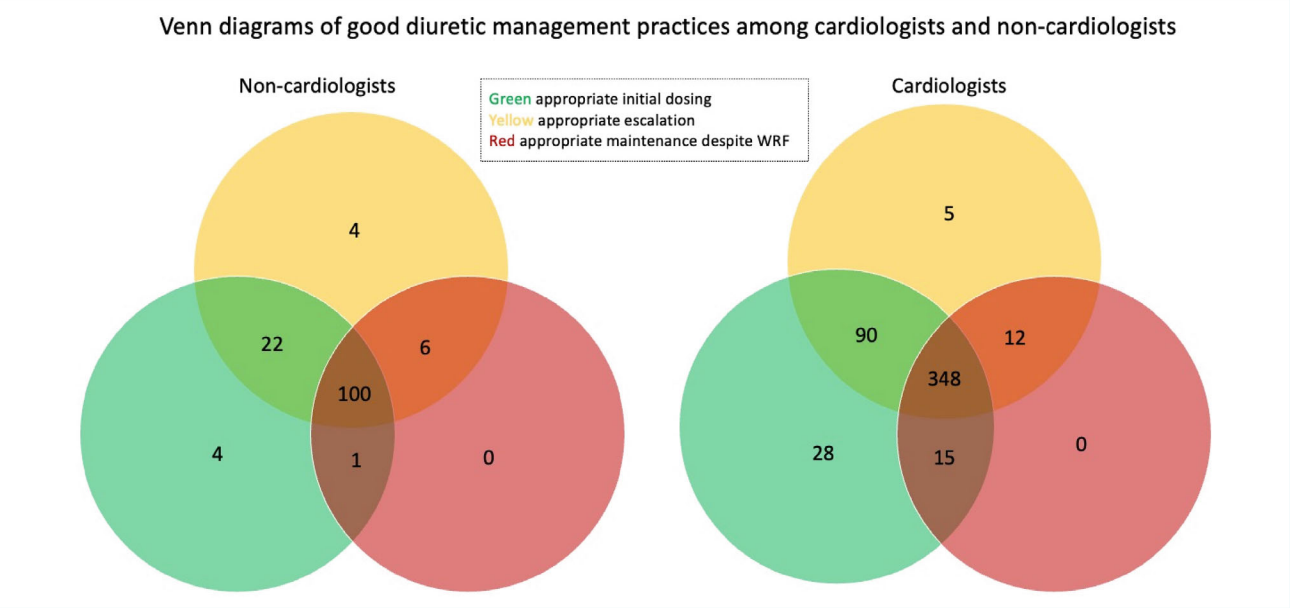

Figure 3 Displays the overlap in good diuretic management practices among cardiologists and non-cardiologists, given as absolute numbers. WRF, worsening renal function. (The same figure is provided in percentage format in online supplementary *Figure S1*).

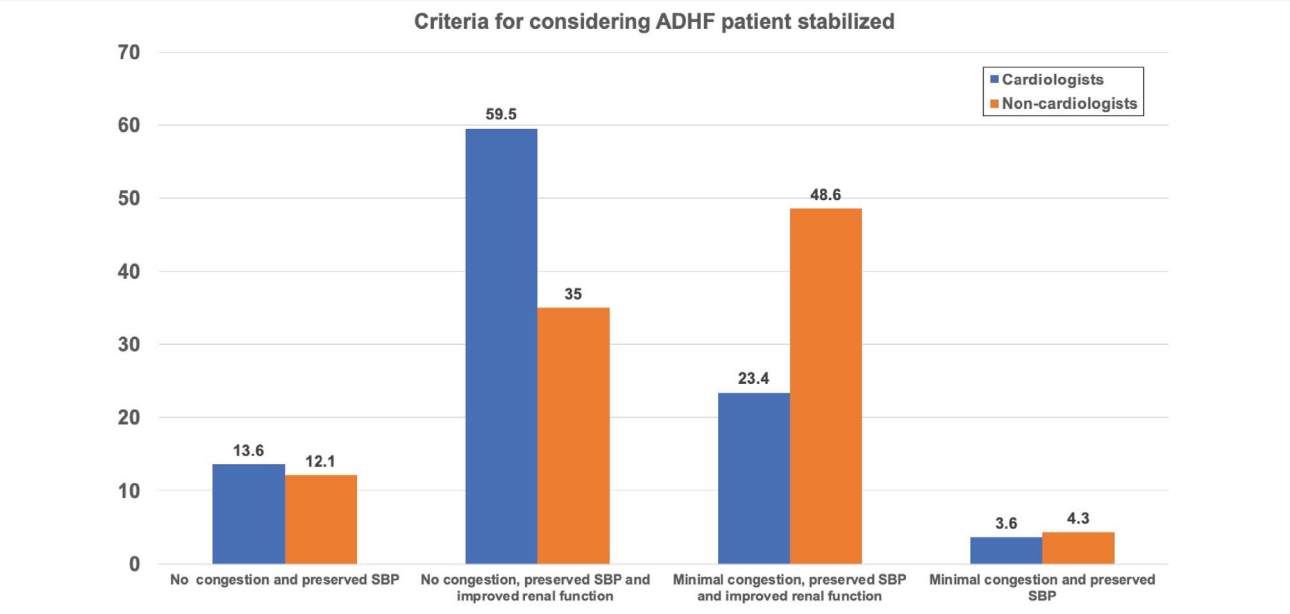

Figure 4 Differences between cardiologists and non-cardiologist in their criteria for considering acute decompensated heart failure (ADHF) patients as being stabilized, given as percentages. SBP, systolic blood pressure.

diuretic resistance (64.7% vs. 39.1%, $p = 0.012$) and also regarded no signs of congestion as a requirement to consider a patient stabilized (71.7% vs. 41.3%, $p = 0.002$). They were more confident about their management (89% vs. 73.9%, $p = 0.013$), more often aware of international guidelines (90.4% vs. 56.5%, $p < 0.001$) and hospital protocols (44.9% vs. 15.2%, $p < 0.001$) compared with non-cardiologists.

Continent

Minimal differences in the responses to the ADHF clinical scenario were noted between clinicians from different continents. The most important differences regarded the higher tendency of physicians from Asia to discontinue mineralocorticoid receptor antagonists on admission (20% vs. 9.1% for America, 9.2% for Europe and 0% for Africa, $p = 0.046$) and lower likelihood of physicians from

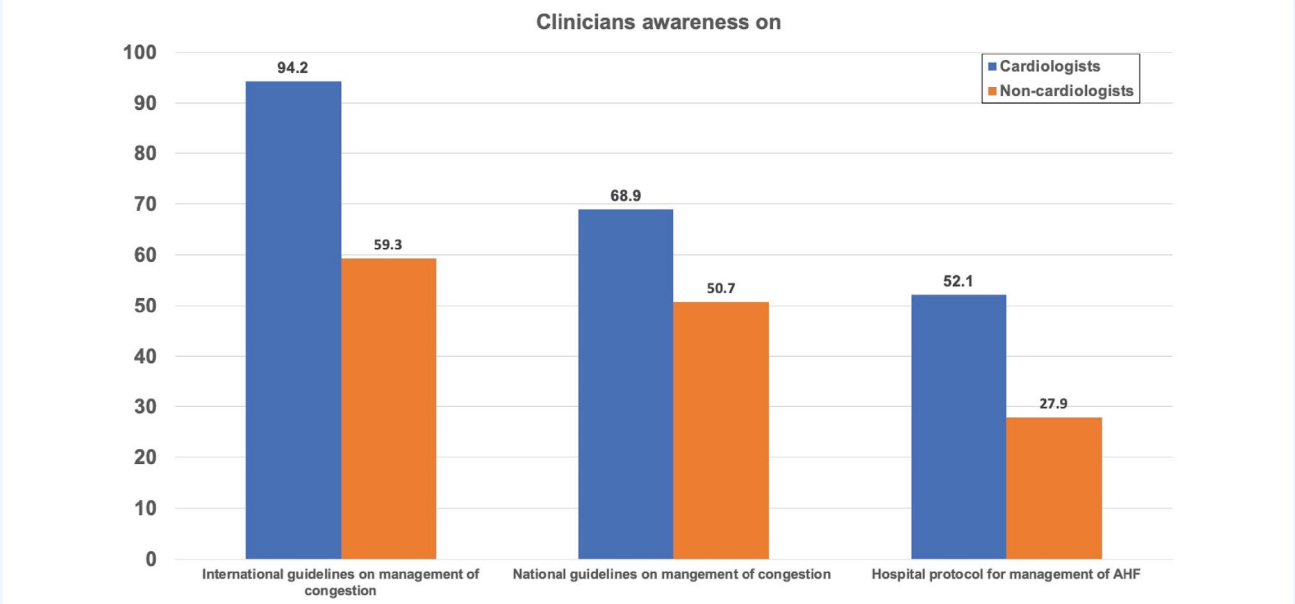

Figure 5 Differences between cardiologists and non-cardiologist in their awareness of international and national guidelines and the presence of localized hospital protocol for the management of heart failure. AHF, acute heart failure.

America to reduce the dose or discontinue diuretics in the scenario of worsening renal function albeit decongestion was achieved (9.1% vs. 37.8% for Asia, 24.1% for Europe and 14.3% for Africa, $p = 0.03$). Physicians from America were less frequently aware about international guidelines (66.7% vs. 97.8% for Asia, 87% for Europe and 100% for Africa, $p = 0.001$), likely indicating higher adherence to national guidelines.

Characteristics of physicians with optimal management

We defined optimal management of the clinical scenario as the combination of the following responses: initial administration of at least 2× the per of loop diuretic dose (≥160 mg furosemide or its equivalent intravenously daily), application of all non-pharmacological measures, non-discontinuation of GDMT but no increase of beta-blockers and/or sacubitril/valsartan on admission, a target daily BW loss of >0.5 kg, non-discontinuation or decrease of loop diuretics when decongestion targets were achieved, increase of loop diuretics when decongestion targets were not achieved and considering for discharge only patients who had no residual congestion. This combination of answers was provided by 93 (14.5%) physicians. When comparing the physicians with optimal versus the physicians with sub-optimal management of the clinical scenario, the only significant factor that could be appreciated was the frequency of managing such patients with 63.4% of physicians with optimal strategy versus 54.7% of physicians with sub-optimal strategy managing at least five such patients per month ($p = 0.001$). No other differences could be noted with regard to timing of managing these patients, medical specialty, years of experience, confidence, type of hospital, continent, presence of relevant hospital protocol and knowledge of national or international guidelines.

Discussion

In this large survey including 641 physicians across 60 countries around the world, (i) a wide variation of clinical management of patients with ADHF was overall noted; (ii) more elaborate approaches of assessing congestion (LUS, inspiratory variation of the IVC) and diuretic response (spot urine sodium) are infrequently used; (iii) physicians seem to be rather conservative in the use of diuretics; and (iv) significant differences in the management of ADHF patients exist between cardiologists and non-cardiologists, including, but not restricted to, indices/modalities for guiding treatment, ways of assessing response to treatment, treatment goals and diuretic escalation strategies. Furthermore differences in clinical management were reported between doctors in academic versus non-academic hospitals and between levels of experience of more and less than 5 years.

It has been long known that clinical symptoms and signs, and chest x-ray findings have low sensitivity and specificity for diagnosing congestion in patients with ADHF.[14] Therefore, contemporary guidelines for AHF recommend a multi-faceted approach which encompasses chest x-ray, LUS, natriuretic peptides and echocardiography.[5] However, only the latter is thought to bear major diagnostic value in AHF and is absolutely recommended, whereas the rest have confirmatory or complementary value.[5] The lack of a single marker or modality that is able to provide an accurate assessment of the patients' congestion status is the main contributor of this variation in clinical practice.[14]

The significant differences in the selection of methodology to guide treatment between cardiologists and non-cardiologists may also indicate a lower availability of specialized modalities such as echocardiography, LUS and natriuretic peptides to the

latter, as has been at least inferred by the lower rates of LVEF and NT-proBNP availability among AHF patients treated by non-cardiologists versus cardiologists.[15–17] The reported, higher compliance of non-cardiologists to non-pharmacological measures might represent compensatory activities in light of their presumed limited access to more elaborate modalities.

The algorithm of diuretic use in patients with ADHF proposed by the relevant working group of the HFA and incorporated in the most recent ESC guidelines recommends spot urine sodium as a key marker for assessing diuretic response and guiding treatment in this setting.[2,5] Nonetheless, this is a novel approach, the utility of which is supported by clinical research published over the last few years and is yet to be proven in a clinical trial setting.[18–20] Thus, the slower uptake of this new, specialized knowledge by non-cardiologists (vs. cardiologists), as indicated by the herein reported lower rates of awareness of national and international guidelines on HF and congestion, may in part explain the lower use of spot urine sodium among the former group, albeit its use was overall infrequent. The recent results of the Efficacy of a Standardized Diuretic Protocol in AHF study (ENACT-HF), which was an open-label study without randomization, used spot urine sodium to assess natriuretic response as part of a standardized diuretic protocol and demonstrated better diuresis and natriuresis, and shorter hospital stay compared to usual care.[21] Hopefully, these promising results together with other studies which are also currently investigating the utility of spot urine sodium, may help expand its use among physicians treating patients with ADHF.[22] Lower awareness of relevant guidelines, hospital protocols and different AHF patient profiles, alongside less frequent management of patients with similar presentation may explain the variation observed in the management of ADHF amongst non-cardiologists.

In terms of initial loop diuretic dosing, clinicians were rather conservative and in line with contemporary recommendations (intravenous dose 1–2 times the oral dose),[2,5] but only 7% of the participants would start the patient on intravenous furosemide at a dose approximating the one tested in the Diuretic Optimization Strategies Evaluation (DOSE) trial (intravenous dose 2.5 times the oral dose).[6] Furthermore, a disappointingly high proportion of physicians, which approximated 30% and was similar between cardiologists and non-cardiologists, was reluctant to escalate diuretic treatment in the setting of ongoing congestion and sub-optimal diuretic response. This proportion equals the reported proportion of AHF patients with residual congestion at discharge in the ESC-EORP HF Long-Term Registry.[23] Thus, beyond clinical predictors of residual congestion at hospital discharge, such as severe tricuspid regurgitation, anaemia, diabetes, etc., our findings indicate that physicians' misperception about congestion may also explain suboptimal clinical practice patterns. On the other hand, almost one fourth of clinicians would reduce furosemide dose due to increases in serum creatinine, albeit target daily BW loss was achieved, and one third would consider a patient with ongoing congestion as stabilized. All the above findings indicate that a considerable proportion of clinicians are unaware of the pathophysiology of renal dysfunction in decompensated HF,[9] and the fact that complete decongestion of AHF patients is associated with favourable outcomes,[24,25] even with the trade-off of worsening renal function, which does not reflect permanent nephron loss but likely altered haemodynamic state in this setting.[24–27] When facing diuretic resistance physicians were keen to adopt several established options, such as further loop diuretic dose increases, initiation/up-titration of spironolactone and addition of a thiazide.[28,29] The preference of non-cardiologists towards the two first options rather than the latter might have to do with the paucity of large studies assessing the efficacy and safety of combined use of loop and thiazide diuretics,[30] alongside their greater familiarity with use of high doses of mineralocorticoid receptor antagonists, as used in liver cirrhosis.[28,29] The results of the Safety and Efficacy of the Combination of Loop with Thiazide-type Diuretics in Patients with Decompensated Heart Failure (CLOROTIC) trial suggest using combination of loop and thiazide diuretic regimens in patients with ADHF to achieve better diuresis.[31] Furthermore, albeit not included in the diuretic escalation options of the survey, intravenous acetazolamide may be a reasonable option in this clinical scenario, as the recently published Acetazolamide in Decompensated Heart Failure with Volume Overload (ADVOR) trial demonstrated that its addition to a standardized intravenous loop diuretic regimen enhances diuresis and leads to higher rates of decongestion at 3 days with reduction in length of stay by a median 1.1 day compared with placebo.[32,33]

Finally, both cardiologists and non-cardiologists seemed to be aware of the prognostic benefits of maintaining GDMT at the highest tolerated doses during a HF hospitalization,[34–36] rarely opting to discontinue them and overall keeping them at a dose at least equivalent to the admission one in more than 75% of cases. The recent published Safety, Tolerability and Efficacy of Rapid Optimization, Helped by NT-proBNP Testing, of Heart Failure Therapies (STRONG-HF) trial further supports the prognostic benefit of intensive strategy for rapid up-titration to high-dose GDMT and close follow-up after acute decompensated hospitalization,[37] whereas the association between higher doses of GDMT and favourable outcomes even in patients with HF and worsening renal function has also been demonstrated and advocates for the maintenance of GDMT at the highest doses possible in the in-hospital setting.[38]

All in all, the findings above represent an important knowledge-practice gap that cannot be ignored. These data showing wide heterogeneity in treatment of ADHF highlight the need for better education and diffusion of current algorithms that are poorly known and adopted. There is a need of targeted initiatives to ensure physicians' education and certification in HF, as recently recommended by the HFA.[39] Namely, the initial step would be to make sure that all physicians that have to manage patients with ADHF are aware of the current HF guidelines. This is particularly relevant for non-cardiologists that are unaware of international and/or national guidelines in >40% of cases. Other knowledge that needs to be more effectively disseminated to physicians managing patients with ADHF are: the significance of maintaining GDMT during the admission for ADHF, the non-prognostic significance of transient worsening of renal function during aggressive diuresis which should not lead to de-escalation of treatment, and the utility of spot urine sodium to guide diuretic treatment in these cases.

Limitations

This study has several limitations. As dissemination was performed via phone, email and social media the response rate cannot be assessed. Furthermore, there was higher participation from some versus other countries. Nonetheless, our population of physicians is large and diverse in terms of geographical variation, specialty, grade, experience and type of hospital, likely providing a representative sample of clinicians managing ADHF patients. However, generalizability to other countries and settings must be done with caution. Moreover, physicians' responses were specific to the clinical scenario provided, so extrapolation of their answers to other AHF patient cases should be performed with extreme cautiousness. Finally, as in any survey, it is impossible to exclude differences between actual practice and survey responses.

Conclusions

There is substantial variability in the clinical management of patients with ADHF. This pertains not only to tools for tailoring treatment, but also to compliance with non-pharmacological measures, treatment targets and diuretic dosing and escalation strategies. Significant differences were noted between cardiologists and non-cardiologists, likely reflecting gaps in evidence and training, and reinforcing the notion that the management of ADHF, at least during the first days of hospitalization, must be protocolized to ensure uniform patient trajectories and optimal outcomes. These inter-specialties differences should be considered by ESC professional associations when intending to improve guideline implementation and may serve to tailor distinct educational programmes.

Supplementary Information

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

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