

## Non-invasive home lung impedance monitoring in early post-acute heart failure discharge: Three case reports

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## Abstract

### BACKGROUND

Patients discharged after hospitalization for acute heart failure (AHF) are frequently readmitted due to an incomplete decongestion, which is difficult to assess clinically. Recently, it has been shown that the use of a highly sensitive, non-invasive device measuring lung impedance (LI) reduces hospitalizations for heart failure (HF); it has also been shown that this device reduces the cardiovascular and all-cause mortality of stable HF patients when used in long-term out-patient follow-ups. The aim of these case series is to demonstrate the potential additive role of non-invasive home LI monitoring in the early post-discharge period.

### CASE SUMMARY

All authors have read the CARE checklist (2016) and prepared and revised the manuscript in accordance with the CARE checklist (2016).

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We present a case series of three patients who had performed daily LI measurements at home using the edema guard monitor (EGM) during 30 d after an episode of AHF. All patients had a history of chronic ischemic HF with a reduced ejection fraction and were hospitalized for 6–17 d. LI measurements were successfully made at home by patients with the help of their caregivers. The patients were carefully followed up by HF specialists who reacted to the values of LI measurements, blood pressure, heart rate and clinical symptoms. LI reduction was a more frequent trigger to medication adjustments compared to changes in symptoms or vital signs. Besides, LI dynamics closely tracked the use and dose of diuretics.

### CONCLUSION

Our case series suggests non-invasive home LI monitoring with EGM to be a reliable and potentially useful tool for the early detection of congestion or dehydration and thus for the further successful stabilization of a HF patient after a worsening episode.

**Key words:** Heart failure; Home monitoring; Lung impedance; Case report; Pulmonary congestion

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**Core tip:** The monitoring of lung impedance (LI) using the edema guard monitor (EGM) seems to be a very sensitive tool for detecting an early increase in lung fluid volume. Non-invasive daily monitoring of LI with the EGM consistently reflects the changes in the dose of diuretics and responds to other treatment adjustments. The titration of the diuretic dose, according to LI values, may optimize patient stabilization in the early post-discharge period.

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## INTRODUCTION

Hospital readmissions are a challenge in the care of heart failure (HF) patients. Readmissions are stressful for patients and families and, at the same time, might have financial consequences for health care organizations. The Hospital Readmissions Reduction Program was recently established in the United States. This program involved a public reporting of hospitals' 30-d risk-standardized readmission rates and applied financial penalties for hospitals with higher readmissions. The results of such a health care policy show that focusing mainly on the financial part of health care may significantly worsen patient care and outcomes<sup>[1,2]</sup>. In the first 30 d, the patients seem to be most vulnerable for rehospitalization; therefore, extra attention during this time period is warranted.

The impedance-HF trial revealed that the use of lung impedance (LI) measurements for the guidance of the preemptive treatment of patients with chronic HF reduced all-cause and HF hospitalizations by 39% and 55%, respectively<sup>[3]</sup>. In that study, measurements of LI were done using the highly sensitive, non-invasive device edema guard monitor (EGM). The EGM is based on an algorithm calculating the chest wall impedance, which is the preponderant component of the total electrical thoracic impedance. The subtraction of the chest wall impedance from the latter yields the net LI, which is the impedance of interest. Decreasing LI values represent the increase of lung fluid<sup>[1]</sup>. In previous reports, EGM was used only in the hospital or during regular outpatient clinic visits. Since EGM seems to be very sensitive to evolving pulmonary congestion, we hypothesized that it could be an accurate tool for the cautious titration of the doses of medicines, especially diuretics, ensuring a smooth and swift transition to follow-up care<sup>[3,4]</sup>.

We present a case series of three patients after an episode of acute heart failure

(AHF), who had autonomously performed daily home LI measurements using the EGM during a 1-month follow up period. The aim of this case series is to demonstrate the potential additive role of non-invasive LI monitoring with EGM in patient stabilization in the early post-discharge period.

### ***The technique of LI measurements and patient monitoring***

The measurements were done with the help of the patients' caregivers once every day at the same time, attaching three EGM electrodes on the front and three EGM electrodes on back side of the chest wall, repeating the measurements 3 times (Figure 1). The LI values were being daily reported to a HF nurse via phone call or SMS, along with arterial blood pressure (BP), heart rate (HR) and body weight. Two patients performed all 30 measurements (100%) and one 29 d out of 30 (96.7%). Echocardiography and laboratory tests were performed before discharge and 1 month later.

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## **CASE SERIES PRESENTATION**

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We present three patients suffering from ischemic HF with a reduced ejection fraction, who were urgently hospitalized because of signs and symptoms of decompensation.

### ***Chief complaints***

At admission patients complained of progressing AHF symptoms: dyspnea at mild physical exertion or at rest, fatigue, palpitations and dizziness.

### ***History of present illness***

All patients had a history of chronic ischemic HF with a reduced ejection fraction for several years.

### ***History of past illness***

Patients' past illnesses are shown in Table 1.

### ***Personal and family history***

Patients' demographic characteristics are presented in Table 1; family history was unremarkable.

### ***Physical examination upon admission***

At admission physical examination of the patients revealed normal BP, tachycardia, leg edema, fine crackles in the lungs. In Patient 2 several paroxysms of ventricular tachycardia were seen on electrocardiogram, led by cold sweat, extreme weakness and decrease of BP.

### ***Laboratory examinations***

Laboratory tests parameters are summarized in Table 2.

### ***Imaging examinations***

Echocardiographic parameters are shown in Table 2.

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## **FINAL DIAGNOSIS**

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Based on clinical symptoms, signs and objective findings, acute decompensated HF was diagnosed in all patients.

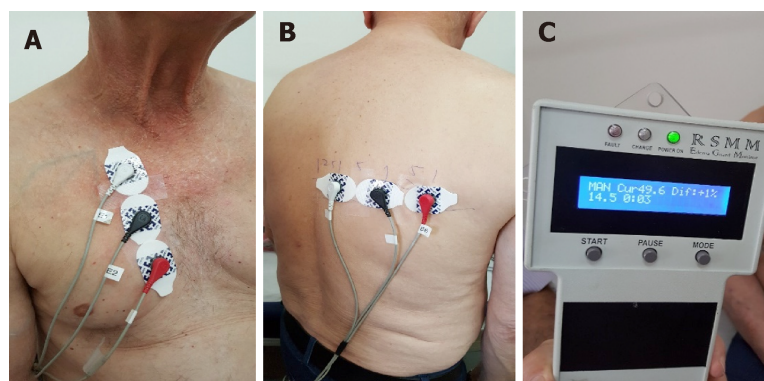
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## **TREATMENT**

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During hospitalization, patients were treated medically according to the ESC guidelines (Table 2). In Patient 2, several paroxysms of ventricular tachycardia were documented with a subsequent implantation of a biventricular defibrillator; Patient 3 was additionally treated with an implantation of drug-eluting stents in the left main and 2 other coronary arteries. Given the severe systolic dysfunction, mitral regurgitation, pulmonary hypertension and anticipated long duration of stenting, coronary intervention in Patient 3 was protected with extracorporeal membrane oxygenation. The dynamics of laboratory, echocardiographic parameters, LI, weight and discharge HF medications are presented in Table 2.

During the course of 30 d follow-up in all three patients, the dosages of medications were adjusted remotely with telephone calls or during four unplanned visits to the



**Figure 1** The technique of lung impedance measurements. A: Three electrodes were placed vertically on the front right side of the chest, 4.5 cm from the midline of the sternum with the upper electrode attached precisely under the clavicle; B: Another set of 3 electrodes was placed on the back along the horizontal line crossing the low edge of the right scapula, with the most leftward electrode placed at the crossing point of the horizontal line with the spine; C: The LI measurement result was displayed as a number on the screen.

outpatient department.

## OUTCOME AND FOLLOW-UP

The patients were asked to come for additional assessments due to deteriorating symptoms, and for laboratory assessments when an electrolyte imbalance or a worsening renal function were suspected. All patients were discharged in a better functional status, but they still remained in functional class III per the New York Heart Association classification.

### Case 1: The dependence of LI values on diuretic intake

Torsemide 50 mg was prescribed in Patient 1 every other day during the first week. There were 5 d when the patient did not take torsemide at all. **Figure 1** illustrates the high dependence of the LI value on the use of a diuretic: each missed diuretic dose has caused the mean drop of LI value by 9 points, or 9.5% (maximal LI value was 97.8  $\Omega$ , minimal 82.6  $\Omega$ ). The concomitant fluctuation of the patient's weight was negligible, ranging between 0 and 500 g, mostly decreasing (**Figure 2**).

### Case 2: The adjustment of treatment in relation to the LI measurement

For Patient 2, the decrease of LI by -9% and -18%, compared with the initial LI, was twice treated by increasing the dose of torsemide from 50 to 100 mg daily. During the follow-up period, the Patient 2's weight fluctuated between 103.3 to 105.7 kg. In 4 d, when LI had decreased the most, the patient's weight increased by 200 g (0.2%) averagely, as compared with the previous day (**Figure 3**).

### Case 3: Dehydration and congestion reflected by LI changes

In Patient 3, the LI value at discharge was 101.8  $\Omega$ , while during the next 3 d, it was high and still increasing (101- > 112- > 117 $\Omega$ ; +15%), pointing to decreasing lung fluid, yet the patient felt poorly. The patient reported a shortness of breath at rest and during night. On the 4<sup>th</sup> d, the patient was invited to an unscheduled cardiologist visit; acute renal failure with hyperkalemia and hypochloremia were diagnosed. The patient was readmitted for 3 d and treated with intravenous fluid and electrolyte infusion. An acute kidney injury was likely associated with contrast-induced damage after the complex percutaneous coronary procedure. Subsequently, during a three-day period (from the 17<sup>th</sup> to the 20<sup>th</sup> of the month), Patient 3 gained 800 g in weight, felt increased dyspnea on the 20<sup>th</sup> and 21<sup>st</sup> d in parallel with a decrease of LI. The negative dynamics of NT-proBNP on the 30<sup>th</sup> d were concordant with a gradual progressive decline of LI during the follow-up (**Figure 4**).

### The correlation between weight changes and LI decrease

In Patient 1 and Patient 3, clinically relevant inverse correlations (a decrease of LI and an increase in weight) were found. LI had decreased 1 d before the increase of weight of Patient 1 with a cross-correlation coefficient equal to -0.738 ( $P < 0.001$ ); in Patient 3, LI and weight has had the maximum cross-correlation at the same day with a coefficient of -0.830 ( $P < 0.001$ ).

**Table 1** Patients' demographics, medical history and length of hospital stay

	Patient 1	Patient 2	Patient 3
Age	66	62	83
Gender	Male	Male	Female
Medical history			
Arterial hypertension	+	+	+
Kidney disease	+	+	+
Myocardial infarction	+	+	+
Revascularization	+	+	+
Atrial fibrillation	-	+	-
Implanted devices	Biventricular pacemaker	Biventricular defibrillator	-
Length of stay (d)	6	17	15

In the course of a 30 d-follow-up, the dosages of medications were adjusted remotely via telephone calls reacting to the changes in symptoms, BP, HR or LI in all three patients. The patients were asked to come for four unplanned visits to the outpatient department when the symptoms had been deteriorating, LI had decreased but an electrolyte imbalance or a worsening renal function had been concomitantly suspected. Among other clinical parameters, the values of LI were the main triggers for adjusting treatment, especially for the dosage of diuretics (Table 3).

## DISCUSSION

A variety of tools that quantify changes in lung fluid content have been evaluated to aid in the early detection of impending HF exacerbation, but in clinical practice, the prediction of pulmonary congestion is still a challenge. Invasive hemodynamic monitoring of PA pressure using a permanently implanted pressure sensor and the titration of diuretics according to pressure values have been reported to decrease hospitalizations for acute HF during 6 mo<sup>[5]</sup>. Monitoring of LI is also possible through the use of OptiVol feature and implanted cardioverter defibrillator or biventricular pacemaker. Although adding OptiVol alerts to HF management in observational studies was shown to improve patient prognosis as well, the positive predictive value for HF exacerbations was found to be only moderate<sup>[6,7]</sup>.

The main disadvantages of these techniques are invasiveness, relatively high cost and inapplicability on a routine basis. Non-invasive transthoracic impedance (TI) measurements are associated with chest congestion, as fluid increases the electrical conductivity of the tissue<sup>[8,9]</sup>. The use of conventional electrical TI equipment for monitoring pulmonary congestion was found to be insufficiently sensitive and did not guarantee reliable monitoring of lung fluid content in the individual patient<sup>[10]</sup>. This may be explained by the fact that TI consists of the target net LI, which is only a small fraction of the overall TI, plus the high impedance of the chest walls.

In this case series, we report three patients with acute HF, who were monitored with the help of the EGM – a highly sensitive, non-invasive, LI measuring device. An arrangement of three electrodes on each side of the chest allows additional electrical circuits between electrodes, which enables calculation of the chest wall impedance and its subtraction from TI; this approach increases the sensitivity of the device to measure changes in lung fluid content by approximately 25 times<sup>[3]</sup>. As a result, preemptive treatment of an evolving pulmonary congestion can be initiated very early, a therapeutic policy that has proven its effectiveness in patients with ST elevation myocardial infarction<sup>[11]</sup>. Our experience with these patients suggests the EGM to be a practical tool that can be used for monitoring of lung fluid, especially while adjusting the dose of diuretics. We have applied a threshold of approximately 10% for reduction of LI (from the initial value measured on discharge) for therapy adjustment. This value is based on previous publications showing the LI dynamics during HF hospitalization and our own experience<sup>[12,13]</sup>. The presented example of Patient 1 clearly illustrates a high dependence of the LI value on the use of diuretics, reflecting an increase in congestion following after the day when the medication was not taken.

Importantly, the measurement of LI with the EGM at home requires the help of a caregiver to attach electrodes to the chest. Though not technically difficult, this dependence on family members may be considered a disadvantage of the method. An

**Table 2** The dynamics of laboratory tests, echocardiography parameters, lung impedance, weight and medications throughout 30 d

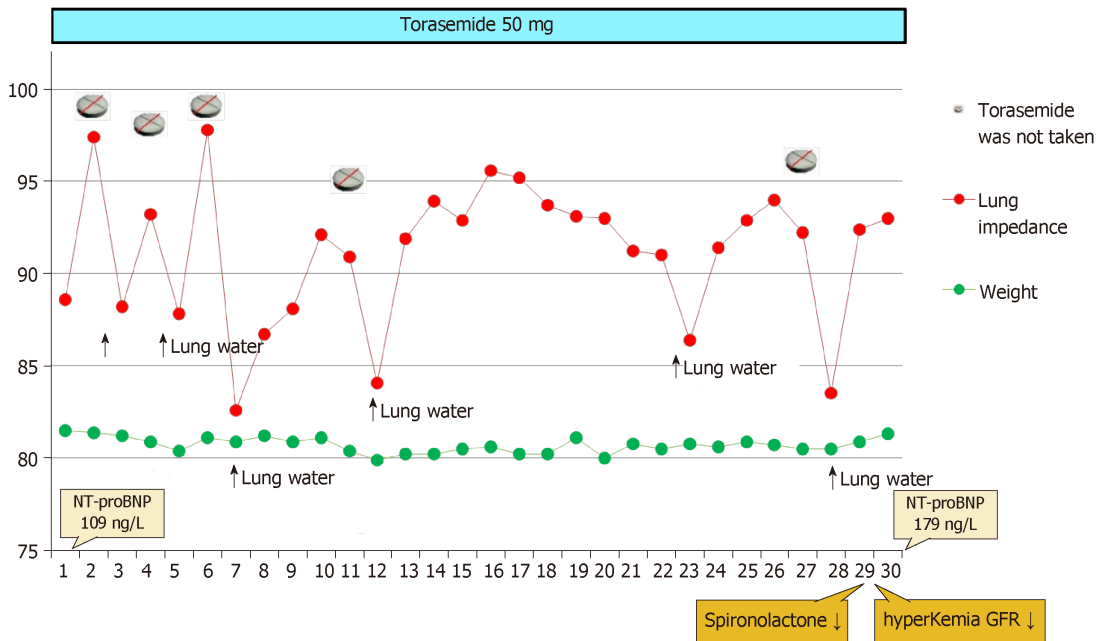
Characteristics	Patient 1		Patient 2		Patient 3	
	Discharge	After 30 d	Discharge	After 30 d	Discharge	After 30 d
Laboratory tests						
NT-proBNP (ng/L)	109	179	3485	2061	2927	5398
Troponin I (ng/L)	13.5	15.4	50.2	20.8	168.0	25.1
Potassium (mmol/L)	5.2	6.3	4.7	4.0	5.1	4.2
Sodium (mmol/L)	139	138	139	139	137	143
Chlorine (mmol/L)	100	95	101	98	98	103
Creatinine (mkmol/L)	133	282	96	103	111	118
eGFR (mL/min per 1.73 m <sup>2</sup> )	48	19	73	68	40	37
Echocardiography						
LV diastolic diameter (mm)	59	56	76	76	71	71
LV ejection fraction 2D (%)	29	27	20	28	23	30
LV ejection fraction 3D (%)	26	29	19	25	35	23
Cardiac output (L/min) 2D	3.1	3.43	4.14	4.68	4.1	3.9
Cardiac output (L/min) 3D	1.7	2.2	4.4	4	4.3	2.2
LV stroke volume 2D (mL)	36	47	41	52	48	58
PCWP (by Nagueh, mmHg)	9.34	12.7	21.0	17	13.0	8.2
Global longitudinal 2D strain (%)	-7	-8.8	-6	-3.6	-6.2	-7.7
Right ventricular diameter (cm)	3.3	3.8	5.2	5.3	2.3	1.9
RV S' (cm/s)	9	11	7	10	15	14
TAPSE (cm)	1.7	1.9	0.6	1.6	2.1	1.9
RV FAC (%)	32.3	35	16.6	28.8	49.6	74.8
Lung impedance ( $\Omega$ )	88.6	93.0	107.9	97.1	101.8	88.1
Weight (kg)	80	81.3	104	105	60.0	61.8
Medicatio						
Percentage of target dose of beta-blocker	25%	50 %	100 %	100%	25%	25%
Percentage of target dose of ACEI	100%	100%	12.5%	25%	50%	25%
Percentage of target dose of Spironolactone	50%	-	50%	100%	100%	-
Torsemide daily dose (mg)	50 mg (e.s.d.)	10 mg	50 mg	100 mg	25 mg	10 mg

NT-proBNP: N-terminal pro B-type natriuretic peptide; eGFR: Estimated glomerular filtration rate; LV: Left ventricular; PCWP: Pulmonary capillary wedge pressure; RV S': Systolic velocity of tricuspid valve; TAPSE: Tricuspid annular plain systolic excursion; FAC: Fractional area change; ACEI: Angiotensin-converting enzyme inhibitor.

essential aspect is the availability of healthcare professional who daily accepts and reacts to LI values. Considering data on reduction of HF hospitalizations using this kind of congestion monitoring<sup>[3]</sup>, financial savings with EGM may be highly significant due to the relatively low cost of the device and regular service.

Significant fluctuations of LI were noticed in all these cases; moreover, the LI change was the most important trigger for medication adjustment compared to standard monitoring variables, such as BP, HR, symptoms and markers of renal function. Though the monitoring of weight changes caused by fluid retention is routinely recommended for HF patients<sup>[13]</sup>, several studies showed that many episodes of worsening HF did not appear to be associated with weight gain. For example, in a case-control study 54% of patients hospitalized due to AHF gained  $\leq 1$  kg during the month prior to admission<sup>[14]</sup>. This suggests that volume overload incompletely characterizes the pathophysiology of AHF and redistribution of volume may also contribute to the development of signs and symptoms of congestion<sup>[15,16]</sup>.

These cases illustrate that LI measurements may represent a more sensitive method for the evaluation of fluid retention compared to weight and subjective symptoms. In two out of these three patients, we found a clinically and statistically significant correlation (lag -1; 0) of weight increase with the drop of LI. It was shown previously that the sensitivity of LI for HF hospitalization and the ambulatory adjustment of diuretics was twice as high as of body weight (83.3% vs 43.9%), and the unexplained detection rate per patient-year was 1.6 vs 4.8, respectively<sup>[17]</sup>. The case of Patient 3 illustrates that the LI measurements can sometimes even reflect excessive



**Figure 2** The dynamics of lung impedance, weight and diuretic dose for Patient 1. The N-terminal pro B-type natriuretic peptide level was not high at the day of discharge, but the patient had clear signs and symptoms of congestive heart failure (HF) on admission (shortness of breath, bilateral rales in the lungs, leg edema, poor left ventricular function on echocardiography). HF with reduced ejection fraction developed after an acute myocardial infarction more than 4 years ago; then, his BNP was 2611 ng/L, but 2 years after, it had reduced to the level of 200 ng/L. NT-proBNP: N-terminal pro B-type natriuretic peptide; GFR: Glomerular filtration rate.

dehydration, assisting in the detection of not only an under- but also over-dosage of diuretics.

## CONCLUSION

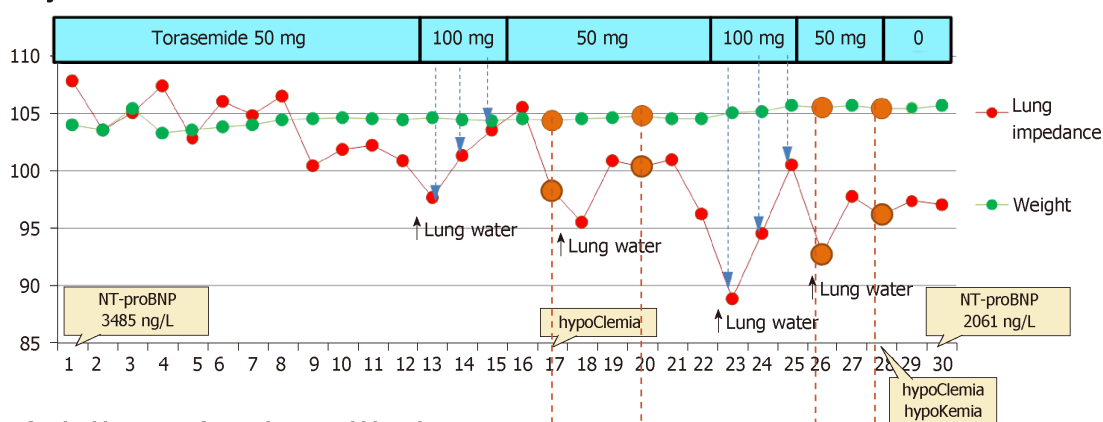
Our first experience with taking LI measurements using the EGM implies the high sensitivity and potential clinical utility of this tool consistently reflected the changes in the dose of diuretics. Non-invasive daily monitoring of LI may become an important component of successful transitions from acute to stable phases of HF, but more clinical experience is needed in order to find the best algorithms for the reactions of health care professionals to different LI changes.

**Table 3 Triggers for treatment adjustments reacting to clinical and monitoring parameters**

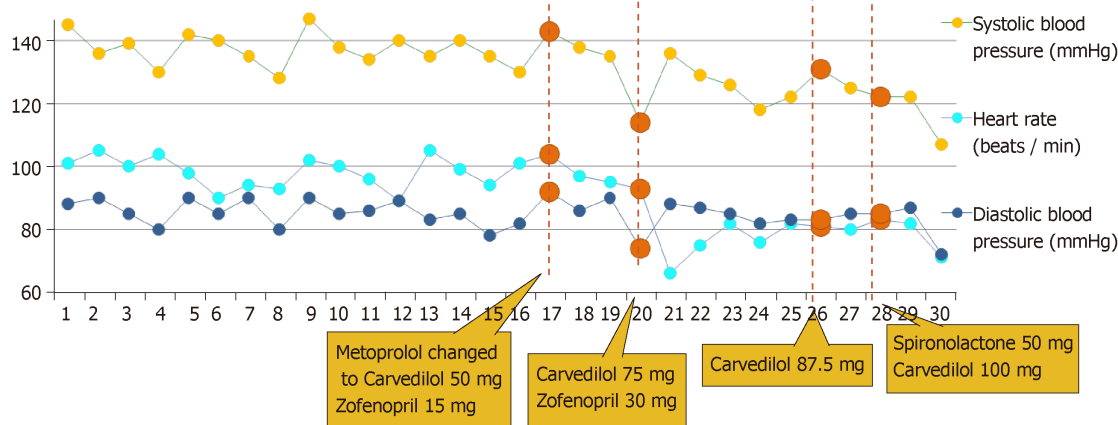
Trigger	Intervention	Frequency of intervention, times
Symptoms (weakness)	Hospitalization because of revealed renal failure, hyperkalemia	1
High HR	Increasing the dose of beta-blockers	3
	Administration of Ivabradine	1
Uncontrolled BP	Increasing the dose of ACE inhibitors	1
Electrolyte imbalance	Increasing the dose of Spironolactone and decreasing the dose of Torasemide	1
LI decrease	Increasing the dose of beta-blockers and Spironolactone	1
	Increasing the dose of ACE inhibitors and beta-blockers	1
	Increasing the dose of Torasemide	5
Weight gain	-	0

HR: Heart rate; BP: Blood pressure; LI: Lung impedance; ACE: Angiotensin-converting enzyme.

**Adjustment of diuretic dose**

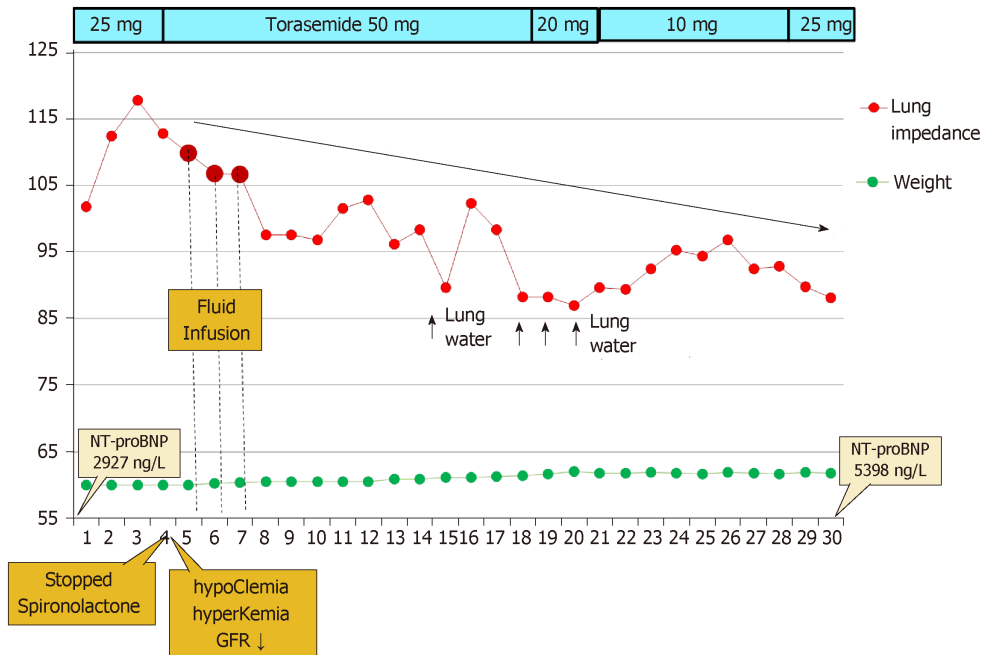


**Gradual increase of neurohormonal blockade**



**Figure 3** The dynamics of lung congestion, weight, hemodynamics and medication dosage for Patient 2. NT-proBNP: N-terminal pro B-type natriuretic peptide.





**Figure 4** The dynamics of lung congestion, weight and diuretic dose for Patient 3 (in this figure, the legend is different than the others (LI). NT-proBNP: N-terminal pro B-type natriuretic peptide; GFR: Glomerular filtration rate.

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