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# Ultrasound-based clinical profiles for predicting the risk of intradialytic hypotension in critically ill patients on intermittent dialysis: a prospective observational study

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## Abstract

**Background:** Intradialytic hypotension, a complication of intermittent hemodialysis, decreases the efficacy of dialysis and increases long-term mortality. This study was aimed to determine whether different predialysis ultrasound cardiopulmonary profiles could predict intradialytic hypotension.

**Methods:** This prospective observational single-center study was performed in 248 critically ill patients with acute kidney injury undergoing intermittent hemodialysis. Immediately before hemodialysis, vena cava collapsibility was measured by vena cava ultrasound and pulmonary congestion by lung ultrasound. Factors predicting intradialytic hypotension were identified by multiple logistic regression analysis.

**Results:** Intradialytic hypotension was observed in 31.9% ( $n = 79$ ) of the patients, interruption of dialysis because of intradialytic hypotension occurred in 6.8% ( $n = 31$ ) of the sessions, and overall 28-day mortality was 20.1% ( $n = 50$ ). Patients were classified in four ultrasound profiles: (A) 108 with B lines  $> 14$  and vena cava collapsibility  $> 11.5$  mm  $m^{-2}$ , (B) 38 with B lines  $< 14$  and vena cava collapsibility  $\leq 11.5$  mm  $m^{-2}$ , (C) 36 with B lines  $> 14$  and vena cava collapsibility  $\leq 11.5$  mm  $m^{-2}$ , and (D) 66 with B lines  $< 14$  and vena cava collapsibility  $> 11.5$  mm  $m^{-2}$ . There was an increased risk of intradialytic hypotension in patients receiving norepinephrine (odds ratios = 15,  $p = 0.001$ ) and with profiles B (odds ratios = 12,  $p = 0.001$ ) and C (odds ratios = 17,  $p = 0.001$ ).

**Conclusion:** In critically ill patients on intermittent hemodialysis, the absence of hypervolemia as assessed by lung and vena cava ultrasound predisposes to intradialytic hypotension and suggests alternative techniques of hemodialysis to provide better hemodynamic stability.

**Keywords:** Ultrasound, Dialysis, Hypotension, Acute kidney injury, Critically ill patients, Profiles

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## Introduction

Ultrafiltration-induced fluid removal for fluid balance control is a major target of renal replacement therapy [1]. However, in critically ill patients, intradialytic hypotension (IDH) is a frequent complication of intermittent hemodialysis and it may decrease the efficacy of renal replacement therapy [2, 3].

The ultrafiltration rate is set to ensure fluid removal required to reduce fluid overload. However, assessment of fluid overload in critically ill patients may be a challenge, because pulmonary congestion is poorly correlated with clinical signs [4]. One alternative approach is the utilization of transthoracic lung ultrasound. A score based on the number of B lines accurately measures the degree of lung congestion and may guide ultrafiltration rate [4–6]. In addition, the rate of disappearance of B lines during intermittent hemodialysis shows a good correlation with the volume of ultrafiltration and dry weight [7, 8].

The pathogenesis of IDH includes the dialysis process itself and critically ill patient-related factors. It has been postulated that the ultrafiltration rate may lead to a reduction in preload, predisposing to hemodynamic instability. Moreover, the dialysis process may interfere with compensatory mechanisms, predisposing to an increased risk of hypotension [9]. As such, it is not clear whether IDH is directly related to preload dependence or to other factors. Clinical studies have been controversial. One study has reported that a positive passive leg raising test before starting dialysis predicts IDH during renal replacement therapy [10]. Another has shown that, in critically ill patients, the majority of hypotensive episodes occurring during intermittent hemodialysis are unrelated to preload dependence and likely related to vasomotor tone alterations [11]. Furthermore, clinical and radiological tools for assessing volume status are subjected to a wide variability of interpretation [12, 13]. As an alternative, the ultrasound measurement of inferior vena cava diameter has been reported to be an accurate method for the assessment of fluid status in critically ill patients [14] and in patients undergoing hemodialysis [15–17].

Although several studies have confirmed the emergent role of biomarkers such as natriuretic peptides [18], copeptin, and bioimpedance vector analysis (BIVA) in the management of volume overload, bedside lung ultrasound appears a sensitive tool for evaluating changes in extravascular lung water in dialysis patients [18, 19]. The primary aim of the present study is to determine whether different predialytic cardiopulmonary profiles, defined on sonographic findings, could predict IDH in critically ill patients undergoing intermittent hemodialysis. The secondary aim was to identify risk factors for IDH in this population.

## Methods

### Study design

This was a prospective observational single-center study performed between January 1, 2015, and April 30, 2018, in a 30-bed medical intensive care unit (ICU), at Hospital Português, a tertiary hospital in Salvador, Brazil. The study was approved by the Ethical Committee from Centro de Estudos Egaz Muniz (CAAE: 89428318.000005029). Written informed consent was waived for this observational and non-interventional study. Critically ill patients were included in the study if they fulfilled the following criteria: (i) age > 18 years, (ii) acute kidney injury (AKI) defined by KDIGO 3, and (iii) treatment by intermittent hemodialysis. Patients with right ventricle dysfunction, valvar heart disease, lung hyperinflation, increased abdominal pressure, marked inferior vena cava respiratory translational motion, and the use of compression stockings and patients with inadequate transthoracic window were excluded from the study.

### Intermittent hemodialysis sessions

The nephrology team in charge of patient care was responsible for the timing of initiation of dialysis and prescription. Intermittent hemodialysis sessions were performed on the basis of standard clinical guidelines, including AKI with hemodynamic stability, ongoing hypercatabolism, hyperkalemia, severe acidosis, presumed volume overload, and respiratory distress. The indication for intermittent hemodialysis refers to patients without vasopressors or in low dose of vasopressors (norepinephrine dose  $\leq 0.3 \text{ mg kg}^{-1} \text{ min}^{-1}$ ) for at least 6 h before initiation of dialysis with mean arterial pressure (MAP)  $\geq 65 \text{ mmHg}$ . For each patient, clinical, laboratory, and hemodynamic variables were used to inform clinical decision-making of ultrafiltration rate. Intermittent hemodialysis was performed with Fresenius 4008 S (Gambro Hospal, Meyzieu, France), and dialysate concentrate solutions with 1.75 mmol/L calcium concentration. Intradialytic hypotension (IDH) was defined as the occurrence of a MAP below 65 mmHg during the dialysis session [11].

### Ultrasound procedures and classifications

Vena cava collapsibility measurement and B line determination were performed by physicians with expertise in cardiac and lung ultrasound for critically ill patients [7, 20–22] (see Additional file 5).

### Statistical analysis

Statistical analysis was performed using SPSS version 19.0 for Windows (SPSS Inc., Chicago, IL). Data were tested for normality by visual inspection and the use of Kolmogorov-Smirnov test. Continuous variables were expressed as median (interquartile range, IQR) and were compared by a nonparametric Mann-Whitney test.

Results are expressed as mean ± SD or median (IQR). Proportions were compared by the  $\chi^2$  test. Variables were compared between groups of sessions (with hypotension vs without hypotension). The binary classification (“no intradialytic hypotension” vs “intradialytic hypotension”) was used as an outcome variable in a way that “no hypotension” and “hypotension” were coded as 0 and 1, respectively. Quantitative and qualitative variables associated with hypotension with a *p* value below 0.05 in univariate analysis were selected for inclusion in a multivariable logistic regression model. Logistic regression with both categorical and continuous independent variables was used to build predictive models for the occurrence of hypotension. A one way-ANOVA was used for comparison between groups of patients according to ultrasound profiles. The four “ultrasound profiles” were compared for association with hypotension, dialysis

discontinuation, or mortality in 28 days, using  $\chi^2$  analysis. Statistical significance was assumed at the 5% level.

**Results**

From January 2016 to March 2018, 248 AKI patients requiring intermittent hemodialysis were considered eligible. Median age was 68, and the age range was 58–76; 149 (60.1%) patients were male; the median Charlson score was 10 (8–12); the median APACHE II score was 15 (12–18); and SOFA score was 8 (6–10).

**Intradialytic hypotension: incidence and predictive factors**

Patients’ characteristics are shown in Table 1 and hemodialysis sessions’ characteristics in Table 2. IDH was observed in 31.9% (*n* = 79) of the patients, interruption of dialysis because of IDH occurred in 6.8% (*n* = 31) of the sessions, and overall 28-day mortality was 20.1%.

**Table 1** Clinical, ultrasound, and biological characteristics of patients

	All patients <i>N</i> = 248	Intradialytic hypotension		<i>p</i>
		No 169 (68.1%)	Yes 79 (31.9%)	
<b>Clinical characteristics</b>				
Gender				0.684
- Male, <i>n</i> (%)	149 (60.1%)	103 (60.9%)	46 (58.2%)	
- Female, <i>n</i> (%)	99 (39.9%)	66 (39.1%)	33 (41.8%)	
Age (years)	68.0 (58.2–76)	66 (55–76)	70 (64–76)	0.018
Charlson score	10 (8–12)	10 (8–12)	10 (8–12)	0.116
APACHE II	15 (12–18)	14 (12–18)	16 (13–18)	0.215
SOFA	8 (6–10)	8 (6–10)	8 (7–10)	0.057
Sepsis, <i>n</i> (%)	123 (49.6%)	71 (42%)	52 (65.8%)	< 0.001
Use of norepinephrine, <i>n</i> (%)	37 (14.9%)	5 (3%)	32 (40.5%)	< 0.001
Mechanical ventilation, <i>n</i> (%)	34 (13.7%)	14 (8.3%)	20 (25.3%)	< 0.001
<b>Predialytic arterial pressure</b>				
Systolic blood pressure, mmHg	132 (114.2–152)	140 (123–160)	114 (103–135)	< 0.001
Diastolic blood pressure, mmHg	70 (60.2–80.7)	71 (64–86)	64 (58–73)	< 0.001
Mean blood pressure, mmHg	89.5 (79.0–105.0)	94 (84–109)	81 (75–90)	< 0.001
<b>Biological characteristics</b>				
Hemoglobin, g/dL	9 (7.6–10.1)	9 (7.4–10.1)	9 (7.8–10.2)	0.839
Bicarbonate, mEq/L	20(18–23)	20 (18–23)	20 (16–22)	0.321
Sodium, mEq/L	138 (138–140)	138 (135–141)	139 (134–141)	0.565
Urea, mg/dL	143 (114.2–194)	143 (110–195)	142 (119–194)	0.659
Lactate, mmol/L	1.4 (1.1–1.9)	1.3 (1.1–1.7)	1.7 (1.2–2.2)	< 0.001
Fluid balance, mL	1772 (1540–2320)	1800 (1535–2320)	1750 (1540–2350)	0.762
<b>Ultrasound characteristics</b>				
Pulmonary congestion, <i>n</i> (%)	144 (58.1%)	106 (62.7%)	38 (48.1%)	0.030
IVC collapsibility, <i>n</i> (%)	74 (29.8%)	14 (8.3%)	60 (75.9%)	< 0.001

APACHE Acute Physiology, Age, Chronic Health Evaluation; SOFA Sequential Organ Failure Assessment score; IVC inferior vena cava; IQR interquartile. Data are expressed as number (percentage) for categorical variables or median (1st quartile–3rd quartile) for continuous variables

**Table 2** Dialysis sessions' characteristics

Dialysis characteristics	All patients N = 248	Intradialytic hypotension		p
		No	Yes	
Duration, min	240 (180–240)	240 (180–240)	210 (150–240)	0.140
Ultrafiltration, mL	1000 (200–2000)	1.242.01	893.03	0.020
Blood flow, mL/min	300 (250–300)			0.010
- 200		24 (14.2%)	24 (30.4%)	
- 250		47 (27.8%)	16 (20.3%)	
- 300		98 (58%)	38 (48.1%)	
- 350		0 (0%)	1 (1.3%)	
Dialysate flow, mL/min	500 (500–500)			0.002
- 300		12 (7.1%)	17 (21.5%)	
- 320		0 (0%)	1 (1.3%)	
- 500		157 (92.9%)	61 (77.2%)	
Temperature, °C	36 (36–36)	36.0 (35.5–36.5)	36.0 (35.5–36.5)	0.416
Sodium, mEq/L	138 (138–140)	138 (138–140)	138 (135–141)	

Data are expressed as number (percentage) for categorical variables or median (1st quartile–3rd quartile) for continuous variables

The presence of sepsis, the use of norepinephrine, a low predialysis MAP, a high predialysis lactate level, the use of mechanical ventilation, and elderly age were factors significantly associated with IDH (Table 1). There was a difference in median ultrafiltration volume (Table 1) between patients with and without IDH: 893 ml (0–1500) in patients with IDH vs 1242 ml (675–2000) in patients without IDH ( $p = 0.020$ ).

Regarding ultrasound findings, pulmonary congestion (as defined by a B line score  $\geq 14$ ) was found in 38 (48.1%) patients with IDH and 106 (62.7%) patients without IDH ( $p = 0.030$ ). Inferior vena cava collapsibility (as defined by a VCDi  $\leq 11.5 \text{ mm m}^2$ ) was found in 60 (75.9%) patients with IDH and 14 (8.3%) patients without IDH ( $p < 0.001$ ).

The predictive multiple logistic regression model of IDH demonstrated that MAP, use of norepinephrine, and inferior vena cava collapsibility were associated with the occurrence of IDH (Table 3). Specifically, pulmonary congestion (a B line score  $\geq 14$ ) and higher MAP appeared protective against IDH (Table 3).

**Ultrasound profiles**

Based on lung ultrasound and VCDi, we could classify patients in four distinct profiles (Fig. 1) (Additional files 1, 2, 3, and 4): (A) 108 patients had pulmonary congestion and hypervolemia, (B) 38 patients did not have pulmonary congestion nor hypervolemia, (C) 36 patients had pulmonary congestion without hypervolemia, and (D) 66 patients had hypervolemia without pulmonary congestion. Baseline

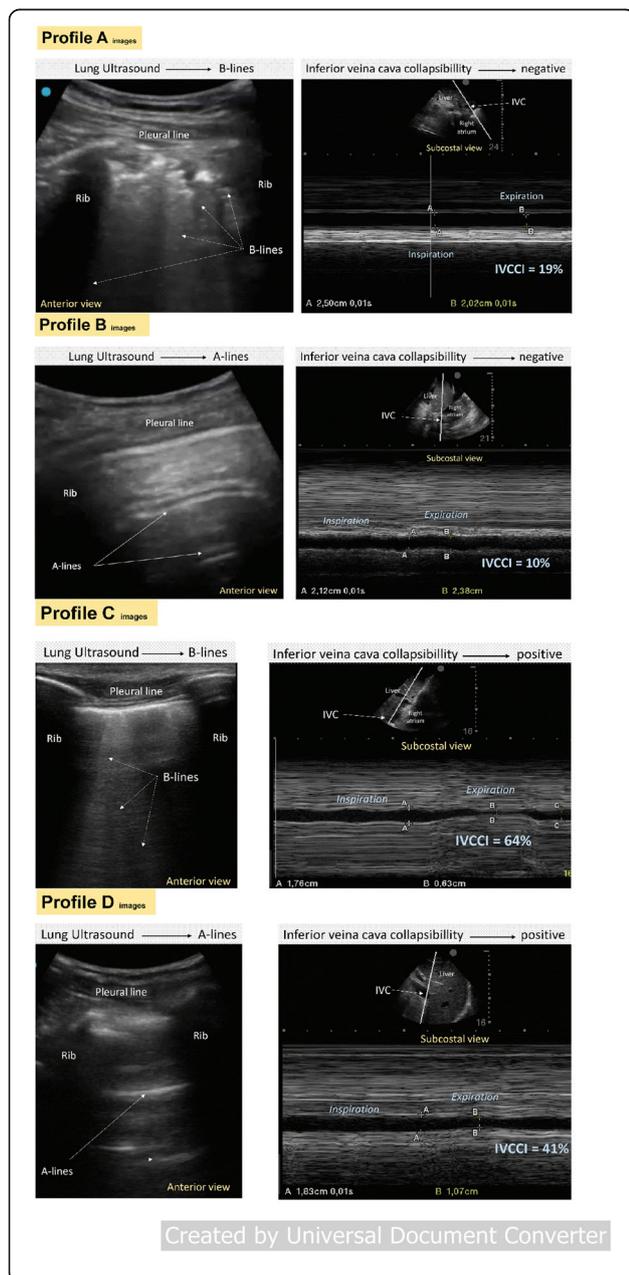
**Table 3** Multivariate logistic regression analysis for the occurrence of intradialytic hypotension

Variable	Parameter estimated	Standard error	Odds ratio	95% CI	p
Age	0.018	0.015	1.018	0.989–1.049	0.228
Blood flow rate	0.006	0.007	1.006	0.993–1.019	0.379
Dialysate flow rate	- 0.007	0.004	0.993	0.984–1.001	0.096
Ultrafiltration	0.001	0.004	1.000	1.000–1.001	0.136
Mean blood pressure	- 0.039	0.015	0.962	0.934–0.991	0.010
Lactate	- 0.187	0.398	0.829	0.380–1.810	0.638
Use of norepinephrine	2.736	0.679	15.425	4.078–58.353	0.001
Pulmonary congestion	- 1.025	0.462	0.448	0.106–0.888	0.027
Inferior vena cava collapsibility	3.573	0.487	35.635	13.722–92.538	0.001
Mechanical ventilation	- 0.803	0.737	0.359	0.106–1.900	0.276
Sepsis	0.584	0.466	1.793	0.719–4.470	0.210
Constant	1.825				

characteristics of the patients in the four ultrasound profiles are listed in Table 4. The presence of IDH was different between profiles ( $p < 0.001$ ). All other variables but age were similar between the four different profiles.

**Clinical outcomes vs ultrasound profiles**

Table 5 shows the risk for IDH, early interruption of intermittent hemodialysis, and 28-day mortality. Patients in profile D appeared to have a lower risk for all outcomes. There was an increased risk of IDH, dialysis discontinuation, and 28-day mortality in patients with profiles B and C.



**Fig. 1** Vena cava and lung ultrasound profiles. (Profile A) 66-year-old patient admitted to the intensive care unit for community-acquired pneumonia with acute kidney injury KDIGO 3 requiring hemodialysis. The patient had a positive fluid balance of 6.5 L and did not receive vasoactive support. In the right upper anterior thoracic region, multiple coalescent B lines issued from justapleural consolidations typical of bronchopneumonia are visible. As shown in the corresponding video file (Additional files 1, 2, 3, and 4), lung sliding is nearly abolished caused by inflammation/infection of pleural layers. The vena cava appears well filled and does not show any significant collapsibility with respiratory movements. (Profile B) 28-year-old patient admitted to the intensive care unit for a urinary tract infection, and acute kidney injury KDIGO 3 requiring dialysis was diagnosed. The patient had a positive fluid balance of 2.5 L, and he was on vasoactive support. In the right upper anterior thoracic region, predominant A lines are visible. As shown in the corresponding video file (Additional files 1, 2, 3, and 4), lung sliding is normal. The vena cava appears filled and shows not significant collapsibility with respiratory movements. (Profile C) 53-year-old patient admitted to the intensive care unit for an alcoholic acute pancreatitis. AKI (KDIGO) 2 was diagnosed, and intermittent dialysis was initiated. The patient had a positive fluid balance of 8.5 L, and he was on vasoactive support. In the right upper anterior thoracic region, white lines from the pleural line to the bottom (B-lines-comets) are visible. As shown in the corresponding video file (Additional files 1, 2, 3 and 4), lung sliding is normal. The vena cava appears collapsed with respiratory movements. (Profile D) 73-year-old patient admitted to the intensive care unit for an acute mesenteric ischemia treated by an extended right hemicolectomy. The patient developed AKI KDIGO 3, and intermittent dialysis was started. The patient had a positive fluid balance of 5.5 L and did not receive vasoactive support. In the right upper anterior thoracic region, horizontal lines (A lines) are visible. As shown in the corresponding video file (Additional files 1, 2, 3, and 4), lung sliding is normal. The vena cava appears collapsed with respiratory movements

**Discussion**

In this study, we describe four clinical profiles based on ultrasound findings and their relation to IDH, interruption of dialysis, and overall mortality at 28 days. Patients with pulmonary congestion and hypervolemia (profile A) had a lower risk of IDH. In contrast, patients without pulmonary congestion and hypervolemia had the highest incidence of IDH. Sepsis, the use of norepinephrine, a low predialysis MAP, lactate level, and mechanical ventilation were significantly associated with IDH.

The pathogenesis of IDH includes the dialysis process itself and critically ill patient-related factors such as sepsis and sedation. Cardiac output and peripheral vasomotor tone are the main determinants of arterial pressure. In critically ill patients, dialysis hypotension is a result of the imbalance of these two variables [23]. Sepsis is the main etiology of AKI and causes a decrease in the peripheral vasomotor tone, induced by the release of vasodilating inflammatory mediators [24, 25]. During the ultrafiltration process, plasma volume decreases leading to an increase in protein level [26]. The resulting increase in oncotic pressure induces a water shift from interstitial and intracellular compartments toward

**Table 4** Baseline characteristics of patients according to the ultrasound profile

Profile	A, n = 108	B, n = 38	C, n = 36	D, n = 66	p
Hypotension, n (%)	8 (7.4%)	30 (78.9%)	30 (83.3%)	11 (16.7%)	0.001
Gender					0.346
Male, n (%)	67 (62%)	26 (68.4%)	22 (14.8%)	34 (51.4%)	
Female, n (%)	41 (41.4%)	12 (31.6%)	14 (38.9%)	32 (48.5%)	
Age (years)	65.0 ± 15.2	69.9 ± 11.8	66.0 ± 12.4	66.0 ± 18.1	0.016
Charlson score	10.3 ± 2.7	10.6 ± 2.8	10.7 ± 2.6	10.0 ± 2.8	0.113
APACHE II	15.2 ± 3.7	15.6 ± 4.3	15.9 ± 3.3	15.4 ± 5.6	0.470
SOFA	8.2 ± 2.3	8.4 ± 2.6	9.2 ± 2.6	8.4 ± 2.7	0.677
Sepsis, n (%)	59 (54.6%)	17 (44.7%)	15 (41.7%)	32 (48.5%)	0.495
Use of norepinephrine, n (%)	19 (17.6%)	6 (15.8%)	4 (11.1%)	8 (12.1%)	0.694
Mechanical ventilation, n (%)	16 (14.8%)	8 (21.1%)	3 (8.3%)	7 (10.6%)	0.353
Fluid balance (milliliters)	1952.2 ± 702.0	1953.0 ± 673.5	1899.8 ± 667.5	1951.8 ± 592.9	0.469
Mortality 28 days, n (%)	19 (17.6%)	6 (15.8%)	5 (13.9%)	20 (30.3%)	0.112
Dialysis data					
Duration, min	212.4 ± 47.5	202.6 ± 53.1	195.14 ± 81.1	212.7 ± 40.7	0.958
Ultrafiltration, mL/min	1197.2 ± 836.6	1072.4 ± 923.3	813.9 ± 818	1228.8 ± 40.7	0.428
Blood flow rate, mL/min	273.15 ± 35.80	248.7 ± 48.6	262.5 ± 42.0	274.24 ± 35.3	0.420
Dialysate flow rate, mL/min	485.18 ± 56.62	447.9 ± 88.4	455.5 ± 84.3	487.9 ± 48.1	0.628
Sodium, mEq/L	138.66 ± 1.28	138.6 ± 0.9	138.8 ± 1.5	138.8 ± 1.9	0.841
Systemic data					
Temperature	36.06 ± 0.25	36.0 ± 0.3	36.0 ± 0.3	36.0 ± 26.1	0.326
Systolic blood pressure, mmHg	139.1 ± 28.5	126.0 ± 0.3	123.0 ± 22.5	144.4 ± 26.1	0.855
Diastolic blood pressure, mmHg	72.8 ± 15.8	68.6 ± 16.9	67.2 ± 12.7	73.9 ± 16.8	0.831
Mean blood pressure, mmHg	94.9 ± 18.4	87.8 ± 18.3	85.8 ± 13.6	97.4 ± 17.5	0.793
Blood test data					
Hemoglobin, /dL	9.1 ± 2.2	9.2 ± 2.2	8.6 ± 2.2	8.6 ± 2.2	0.262
Bicarbonate, mEq/L	19.7 ± 3.8	18.8 ± 3.8	20.1 ± 5.2	20.1 ± 5.2	0.767
Sodium, mEq/L	138.2 ± 4.5	138.9 ± 8.5	137.4 ± 5.4	137.0 ± 6.6	0.396
Urea, mg/dL	157.6 ± 59.3	159.9 ± 50.3	157.5 ± 47.8	150.1 ± 61.7	0.802
Lactate, mmol/L	1.5 ± 0.6	1.8 ± 0.85	2.0 ± 1.1	1.5 ± 0.5	0.242

SOFA Sequential Organ Failure Assessment, APACHE Acute Physiology and Chronic Health Evaluation. Data are expressed as mean ± standard deviation

intravascular compartment, which limits ultrafiltration-induced hypovolemia [27]. When ultrafiltration rate surpasses refilling rate, reduction in preload induces a fall in stroke volume that predisposes to hemodynamic instability [23, 27]. Normally, two physiological mechanisms are activated to maintain cardiac output and arterial pressure despite the reduction of stroke volume: an increase in heart rate and an increase in systemic vascular resistance. The dialysis process, however, interferes with this compensatory process [28]. Several mechanisms have been incriminated [27]: diffusion-induced changes in osmolality impairing baroreceptor activation, dialysis of plasma norepinephrine, calcium dialysate concentrations, and dialysate temperature. The hemodynamic impact of ultrafiltration is higher in

critically ill patients with preload dependence and altered vasomotor tone [29].

Application of ultrasound has improved the care of critically ill patients with kidney diseases by providing an accurate estimation of volume status [14, 15] and pulmonary congestion [22]. It is also an accurate monitoring of treatment efficiency: the rate of disappearance of B lines following hemodialysis correlates with ultrafiltration and dry weight [5, 8]. Ultrasound determination of IVC expiratory diameter and collapsibility combined with clinical parameters have been used to monitor volume unloading during hemodialysis [29–32]. In the present study, critically ill patients treated by norepinephrine without hypervolemia were at high risk of IDH.

**Table 5** Ultrasound profiles vs outcomes

Profile	Parameter estimated	Standard error	Odds ratio	95% CI		<i>p</i>
Hypotension						
A	- 2.530	0.404	0.08	0.036	0.176	0.001
B	2.511	0.430	12.321	5.304	28.624	0.001
C	2.811	0.476	16.633	6.544	42.277	0.001
D	- 1.093	0.364	0.335	0.164	0.684	0.003
Dialysis discontinuation						
A	- 2.608	1.039	0.074	0.010	0.565	0.012
B	4.516	0.526	4.516	1.601	12.743	0.004
C	1.863	0.526	6.444	2.298	18.071	0.001
D	- 1.835	1.041	0.160	0.021	1.228	0.780
Mortality 28 days						
A	- 1.072	0.360	0.342	0.169	0.694	0.003
B	0.580	0.399	1.786	0.817	3.907	0.146
C	1.284	0.385	3.612	1.697	7.688	0.001
D	- 0.172	0.368	0.842	0.410	1.731	0.640

We identified two profiles based on lung and vascular ultrasound able to predict the risk of IDH and guide the type of dialysis session. Critically ill patients with VCDi  $\leq 11.5 \text{ mm m}^{-2}$ , an ultrasound value ruling out hypervolemia, were at risk of IDH in the presence or absence of lung congestion. At the opposite, hypervolemia defined as a VCDi  $\geq 11.5 \text{ mm m}^{-2}$  was protective against IDH in the presence or absence of lung congestion.

Before starting intermittent hemodialysis, the identification of patients with cardiopulmonary profiles increasing the risk of intradialytic hypotension should incite the clinician to select an alternative dialysis technique. Implementation of practice guidelines for intermittent hemodialysis can lessen hemodynamic instability [2]. Continuous renal replacement therapies (CRRT) provide hemodynamic stability, but the continuous session lasting 24–96 h requires expertise, anticoagulation, and alarm vigilance [33]. Extended daily dialysis is associated with similar outcomes to CRRT; however, further high-quality randomized controlled trials are desirable [34]. A technique of dialysis personalized to the risk of intradialytic hypotension is of critical importance, since intradialytic hypotension can increase mortality [35]. Recently, it has been shown that a high ultrafiltration rate ( $> 25 \text{ ml/kg/day}$ ) over the period of RRT significantly reduces the risk of 1-year mortality in 1075 patients with AKI and fluid overload  $> 5\%$  of body weight [36]. Interestingly, MAP was lower for duration of RRT and cumulative norepinephrine dose higher in patients treated with low or moderate ultrafiltration rate, suggesting that IDH increased the mortality risk. However, it was impossible to conclude whether tolerating intensive ultrafiltration is simply a marker of recovery or a mediator [37]. Our results are similar: it is likely that the increased

28-day mortality observed in the 79 critically ill patients who experienced intradialytic hypotension was partly due to the hypotensive episodes, but the finding of increased mortality in patients with dialytic hypotension does not necessarily mean that hypotension causes mortality.

Our study has some limitations. It is a single-center study, and our findings have to be reproduced and our hypothesis tested in multicenter randomized controlled trials. We have only performed ultrasound studies just before hemodialysis initiation. The vena cava collapsibility was assessed either in patients with or without mechanical ventilation. The patients on mechanical ventilation, however, were lightly sedated and partly spontaneous breathing.

**Conclusion**

In critically ill patients with objective indications of emergent initiation of hemodialysis, the absence of hypervolemia with or without pulmonary congestion as assessed by vena cava and lung ultrasound predisposes to intradialytic hypotension. Further studies should be considered to validate these results and personalize the techniques of dialysis according to the risk of intradialytic hypotension.

**Supplementary information**

Supplementary information accompanies this paper at <https://doi.org/10.1186/s13054-019-2668-2>.

- Additional file 1.** Profile A. Patient with B lines  $> 14$  and VCDi  $> 11.5 \text{ mm.m}^{-2}$ .
- Additional file 2.** Patient with B lines  $< 14$  and VCDi  $\leq 11.5 \text{ mm.m}^{-2}$ .
- Additional file 3.** Patient with B lines  $> 14$  and VCDi  $\leq 11.5 \text{ mm.m}^{-2}$ .
- Additional file 4.** Patient with B lines  $< 14$  and VCDi  $> 11.5 \text{ mm.m}^{-2}$ .
- Additional file 5.** Ultrasound procedures and classifications

**Abbreviations**

IDH: Intradialytic hypotension; ICU: Intensive care unit; AKI: Acute kidney injury; MAP: Mean arterial pressure; IVC: Inferior vena cava; IVC<sub>max</sub>: Maximal IVC diameter; IVC<sub>min</sub>: Minimum IVC diameter; IVCCI: IVC collapsibility index; IQR: Interquartile range; CRRT: Continuous renal replacement therapies

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**Authors' contributions**

RHP was responsible in the conception and design of the work, acquisition and interpretation of data, drafting the work, and performing ultrasound examinations. JCR was responsible in the interpretation of data and drafting the work. JGRR was responsible in the interpretation of data and drafting the work. EBSGM was responsible in the acquisition of data. MPDR was responsible in the acquisition of data and performing ultrasound examinations. MFCA was responsible in the acquisition of data and drafting the work. PBPB was responsible in the interpretation of data and drafting the work. OHCM was responsible in the acquisition of data. AMCF was responsible in the acquisition of data. EM was responsible in the interpretation of data and drafting the work. JJR was responsible in the interpretation of data and drafting the work. All authors read and approved the final manuscript.

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**Availability of data and materials**

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

**Ethics approval and consent to participate**

The study was approved by the Ethical Committee from Centro de Estudos Egaz Muniz (CAAE: 89428318.000005029). Written informed consent was waived for this observational and non-interventional study.

**Consent for publication**

All authors reviewed the manuscript and consented to its publication.

**Competing interests**

The authors declare that they have no competing interests.

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