A case of using impedance cardiography-guided CVVH by critical care nephrology in the management of acute decompensated heart failure

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Abstract

Ultrafiltration by continuous venovenous hemofiltration (CVVH) is a well-established therapy for acute decompensated heart failure (ADHF). Aggressive fluid removal, however, may worsen the symptoms or even inflict *de novo* organ dysfunctions especially in the hemodynamically unstable patients. We described the incorporation of CVVH and impedance cardiography (ICG) in the management of an ADHF patient on maintenance hemodialysis. Simultaneous monitoring of the hemodynamic and fluid status using ICG helped optimize the CVVH prescription and improve the patient outcome. As such, assessment of the unique hemodynamic characteristics of each critically ill patient is indispensable, which may enhance the efficiency of the critical care unit. Selection of the ultrafiltration against diuretics is also discussed.

Key words: acute heart failure, CVVH, hemodynamic monitoring, impedance cardiography, critical care nephrology.

Introduction

Heart failure remains the single most common reason for admission in America among patients more than 65 years. (1) The majority of patients are admitted with fluid overload, often diuretic resistance and typically in the cardiomyopathy. Failure to adequately reduce total body sodium may progressively deteriorate the ventricular function, aggravate heart failure and increase morbidity. (2) Traditionally, diuretics are the mainstay for the treatment of acute decompensated heart failure (ADHF) yet currently this approach is under close scrutiny for several inherent limitations. (3) Ultrafiltration by continuous venovenous hemofiltration (CVVH) is believed to be a feasible alternative, (4) but not without concerns. (5) As such, hypovolemia due to aggressive fluid removal may inflict *de novo* renal impairment or exacerbate the pre-existing chronic kidney disease, stressing the importance of accurate assessment of volume status during the CVVH.

It is not easy, however, to ascertain the volume status of patients from review of the symptoms and physical examination alone. For this purpose, the impedance cardiography (ICG) is developed as a non-invasive method capable of yielding a wide spectrum of cardiocirculatory parameters. (6) Careful assessment of the data may help detect occult hypovolemia and titrate the optimal dose of fluid. Concurrently using the continuous venovenous hemodiafiltration and ICG, we have preciously treated acute renal failure caused by leukemia infiltration of the kidneys. (7)

We reported here the treatment of an ADHF patient on maintenance hemodialysis in our critical care nephrology (CCN) with the therapeutic CVVH accompanied by ancillary ICG. Introduction of the ICG appeared to facilitate the safe institution of effective CVVH.

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Case report

A 77-year-old female suffering from shortness of breath and dyspnea for the past 24 hours was admitted to our CCN. On admission, the patient complained general weakening, nocturnal cough with frothy sputum and paroxysmal orthopnea, was mostly in a state of lethargy. There was no fever, headache and chest pain. She was diabetic and had previously received CABG surgery with functional NYHA class III thereafter. Moreover, she was on regular maintenance hemodialysis for the past 1.5 years due to end stage renal disease, taking medications according to the guidelines of the National Kidney Foundation. The temperature was 36.5 ºC on presentation, pulse and respiratory rate were respectively 114 beats and 28 breaths per minute, blood pressure 96/52 mmHg. Auscultation of the chest revealed diffuse moderate rales on the base of the lungs and gallop rhythm of 114/min. There was also moderate jugular venous distention, subcostal hepatomegaly of 3 cm without tenderness, positive hepatojugular regurgitation and 3+ pitting edema of the lower extremities. The remainder of the physical exam was noncontributory.

Oxygen saturation was 91% by pulse oxymetry. Laboratory tests showed hemoglobin of 113 g/L and serum albumin of 30.6 g/L. Chest radiography found a CTR of 65.7% and bilateral infiltrates consistent with pulmonary edema. An ECG recorded sinus tachycardia, and non-specific ST changes that were present on a previous one. Her initial cardiac enzymes were negative.

CVVH was initiated with ICG (BioZ, CardioDynamics, CA, USA) performed at the initiation and cessation. The pre-CVVH ICG data confirmed the diagnosis of ADHF with myocardial impairment and fluid retention (**Table 1**): as indicated by the markedly decreased cardiac index (CI), stroke index (SI), velocity index (VI), prolonged systolic time ratio (STR) and elevated thoracic fluid content (TFC), respectively. Administration of vasoactive agents and fluid removal were expedited accordingly (**Table 2**). The patient began to have some symptomatic improvements in her dyspnea during the 1st CVVH session. Post-CVVH ICG showed improvement of the above parameters, consistent with general amelioration of the ADHF (**Table 1**). CVVH prescription was adjusted and medications tapered according to the ICG results (**Table 2**). After 5 sessions, the patient was discharged from the CCN with resolution of symptoms and signs. Regular hemodialysis was resumed then and the patient maintained a fragile yet tolerable cardiac function.

Discussion

Heart failure and chronic kidney disease indeed coexist in large numbers of patients and exert mutually worsening effects: the presence of chronic kidney disease in patients with heart failure adversely influences their survival and cardiovascular disease is the major cause of mortality in individuals with chronic kidney disease. (5) This detrimental interaction is exemplified in patients on maintenance hemodialysis, who usually exhibit volume overload and are hemodynamically more unstable thus susceptible to cardiac dysfunction. (8) Further, severe heart failure can also induce multiple organ failure which in turn requires multidisciplinary participation especially the critical care professionals.

Contemporary management of the critically ill patients requires accurate assessment of the volume status and maintenance of an appropriate fluid balance to optimize patient outcomes. (9) In ADHF patients, fluid removal should be targeted to a specific preload, stroke volume and/or cardiac output rather than to a specific mean blood pressure. During the pursuit of this end-goal directed therapy, caution should be exercised against volume depletion and the resultant hypotension, prerenal azotemia, and collateral renal injury. The timely information of SI and TFC may therefore not only help secure the appropriate ultrafiltration setup by taking into account of the cardiac function, but allow the early use of vasopressor. (6) These two actions are vital in our patient and the likes manifesting low blood pressure, as they may swiftly alleviate the circulatory crisis and create opportunity for later use of vasodilators and diuretics. In addition, ultrafiltration can simultaneously clear through convection pro-inflammatory cytokines that have a deleterious role in heart failure and restore diuresis. (10) Irrespective of the anuric state, expeditious application of the ICG-guided CVVH and the ensuing fluid removal are pivotal in improving the clinical conditions of ADHF patients.

Two therapeutic options are available to relieve venous

congestion in ADHF: diuretics and ultrafiltration (ie CVVH in our case). The latter is more effective in diuretic-resistance, severe, congestive heart failure. (11) Interestingly, a tenet was recently brought forward by a cardiologist who believes ultrafiltration is the only rational initial treatment for volume overload in ADHF. (4) We are more inclined to agree with this opinion. From our previous work on the tubuloglomerular feedback mechanism (TGF), (12) sodium removal by ultrafiltration may be fundamentally different from, and superior to, diuretic therapies by avoiding increased sodium delivery to the distal nephron which is the signal to activate the TGF. By abolishing the augmented TGF response, ultrafiltration may prevent the subsequent maladaptive diuretic resistance, stimulation of the renin-angotensin-aldosterone cascade and activation of sympathetic nervous system. (13) Moreover, ultrafiltration removes more total body sodium (the main determinant of extracellular fluid volume) than do diuretics for similar volume of fluid removal (14) since ultrafiltration and diuretics elicit the removal of isotonic plasma water and excretion of hypotonic urine, respectively. (15) Despite the advantages of the CVVH, however, a number of safety issues pertinent to the extracorporeal circuit could not be neglected. Critical care units, with higher level of competence in managing patients with life-threatening illnesses, could be readily called upon to meet the challenges as predicted by Bellomo and Ronco a decade ago. (16)

In summary, ultrafiltration is a reliable and effective treatment of ADHF with volume overload and the use of ICG makes it safer and more efficacious. Incorporation of CVVH and ICG may therefore enable the CCN as the ″second front″ unremittingly waging and expectantly winning the battle against ADHF, an inexorable disease which may otherwise be fatal.

Table 1. Hemodynamic parameters from the impedance cardiography monitoring in the CCN

Hemodynamic parameters	1st session		2nd session		5th session		14 days after onset		Normal range and Unit
	pre-CVVH	post-CVVH	pre-CVVH	post-CVVH	pre-CVVH	post-CVVH	pre-dialysis	post-dialysis	
Heart Rate	98	72	76	74	79	82	94	88	58-86 beats/min
Systolic blood pressure	84	76	86	95	106	100	76	79	100-140 mmHg
Diastolic blood pressure	38	53	44	57	58	46	45	52	60-90 mmHg
Cardiac index	1.6	1.8	1.7	1.5	2.3	2.5	1.9	1.9	2.5-4.2 L/min/m^2
Stroke index	16	25	22	20	29	30	20	21	35-65 mL/m^2
Systemic vascular resistance index	950	1511	1176	2346	1635	1408	1164	1621	1337-2483 dyne. s / cm^5/m^2
velocity index	21	32	25	23	31	27	31	32	33-65 /1000 second
Thoracic fluid content	66.0	60.4	55.3	42.8	43.5	34.0	43.5	33.7	30.0-50.0 L /kilo ohm
Systolic time ratio	0.70	0.43	0.64	0.71	0.59	0.49	0.71	0.67	0.30-0.50

CCN: critical care nephrology; CVVH: continous venovenous hemofiltration.

Table 2. Timely changes in the CVVH prescription and vasoactive regimen

	1st session	2nd session	5th session	14th day*	Unit
CVVH					
Ultrafiltration	7300	6800	4700	1500	mL
Net fluid removal	6000	5000	2500	0	mL
CVP (pre- and post-CVVH)	31/27	25/20	17/15	16/12	cmH2O
Duration	37	26	23	4	H
Vasopressors					
Dopamine (pre- and post-CVVH)	20/15	15/10	Cessation	None	µg/kg/min
Dobutamine (pre- and post-CVVH)	16/13	13/7	Cessation	None	µg/kg/min

Legend: CVP=central venous pressure; *=time after onset and regular hemodialysis

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