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CASE REPORT

Artificial Kidney / Dialysis

Continuous monitoring of changes in cerebral oxygenation during hemodialysis in a patient with acute congestive heart failure

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Abstract

A 71-year-old man undergoing hemodialysis (HD) was admitted to our hospital with congestive heart failure (CHF) and pneumonia. After admission, ultrafiltration with HD was urgently performed because of a lack of respiratory improvement despite the use of noninvasive positive pressure ventilation. During HD, cerebral regional saturation of oxygen (rSO_2) was monitored by INVOS 5100c oxygen saturation monitor (Covidien Japan, Japan) to evaluate changes in tissue oxygenation. At HD initiation, cerebral rSO_2 was very low at 34% under the fraction of inspiratory oxygen (FiO_2) of 0.4. Ultrafiltration was performed at the rate of 0.5 L/h thereafter, cerebral rSO_2 gradually improved even as inhaling oxygen concentration decreased. At the end of HD, cerebral rSO_2 improved at 40% under a FiO_2 of 0.28 as excess body fluid was removed. After pneumonia and CHF improved, he was discharged. Reports of the association between cerebral oxygenation and acute CHF status in patients undergoing HD are limited; therefore, in our experience with this case, cerebral oxygenation deteriorated with the CHF status but was improved by adequate body-fluid management during HD.

Keywords Acute heart failure · Cerebral oxygenation · Hemodialysis · Regional saturation of oxygen · Ultrafiltration

Introduction

The leading cause of death in hemodialysis (HD) patients is reportedly cardiovascular disease including heart failure (HF), which accounts for 25% of deaths [1]. Indeed, we sometimes experience HD patients with HF, induced by excess body-fluid in addition to ventricular dysfunction itself being a risk factor for HF [2]. Therefore, congestive HF (CHF) should be prevented in the clinical setting of HD therapy. Recently, near-infrared spectroscopy (NIRS) has been used to measure the regional saturation of oxygen (rSO_2), which is a tissue oxygenation marker [3–6]. To date, reginal tissue oxygenation was reportedly evaluated in HD patients [7–12]. In particular, cerebral evaluations using NIRS were performed to possibly confirm the changes of oxygenation, including in the acute phase of various disorders. However, to date, no studies have about the association between CHF status and changes in cerebral oxygenation in HD patients. We herein focused on changes in cerebral oxygenation during HD and evaluated the effect of ultrafiltration on cerebral oxygenation in a HD patient with CHF.

Case report

A 71-year-old man undergoing HD was admitted to our hospital with CHF and pneumonia. His past medical history included hypertension and diabetes mellitus. His vital signs and laboratory findings were as follows: blood pressure and pulse rate on admission, 135/67 mmHg and, 107 beats/min, respectively; white blood cells, 10,600 /μL;

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hemoglobin, 9.4 g/dL; serum albumin, 2.9 g/dL; blood urea nitrogen, 20 mg/dL; serum creatinine, 5.77 mg/dL; brain natriuretic peptide, 1840 pg/mL; partial pressure of arterial oxygen (PaO_2), 70.0 mmHg; and partial pressure of arterial carbon dioxide, 36.3 mmHg. On admission, ischemic heart disease was suspected; therefore, coronary angiography was performed, but no abnormalities in the coronary artery were confirmed. Thereafter, we started administering intravenous antibiotics and used noninvasive positive pressure ventilation (NPPV). However, his respiratory condition did not improve because of the excess body-fluid, as shown on chest X-ray (Fig. 1a). Therefore, to improve his body-fluid status, ultrafiltration with HD was urgently performed. In addition to the monitoring of saturation of percutaneous oxygen (SpO_2), cerebral rSO_2 was measured using INVOS 5100c oxygen saturation monitor (Covidien Japan, Japan) from HD initiation to end (Fig. 2). The patient provided informed consent prior to undergoing monitoring of his cerebral rSO_2 during HD. At the initiation of HD, his SpO_2 and PaO_2 were 96% and 77.2 mmHg, respectively, whereas cerebral rSO_2 was very low level of 34%, under the condition of the fraction of inspiratory oxygen (FiO_2) of 0.4. HD was performed under ultrafiltration of 0.5 L/h to correct body-fluid excess; then, cerebral rSO_2 gradually improved even with a decrease in inhaling oxygen concentration. At the end of HD, cerebral rSO_2 improved to 40%, the SpO_2 was maintained at 95%, and a PaO_2 of 89.3 mmHg was achieved under FiO_2 of 0.28 with excess water removal. As shown in Fig. 1b, after his pneumonia and CHF improved, he was discharged from our hospital. In another admission for an ophthalmic operation, we monitored his cerebral rSO_2 under the stable HD with ultrafiltration of 0.88 L/h. Comparison of cerebral rSO_2 between his CHF status and stable condition during HD revealed that cerebral rSO_2 at HD initiation was higher in stable HD than in CHF status, while those during stable HD were continuously maintained even with

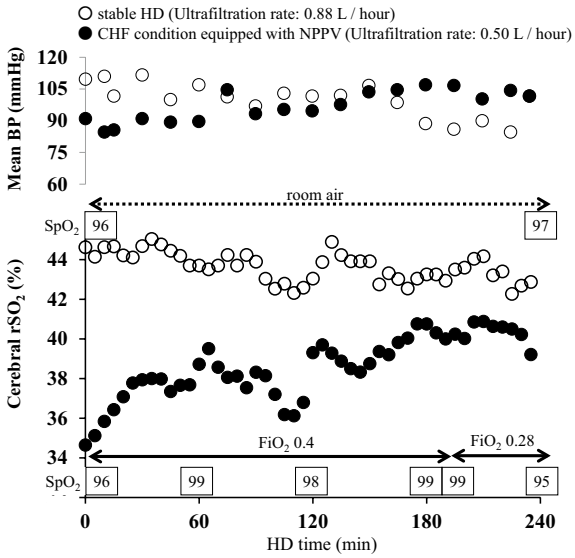

Fig. 2 Changes in cerebral rSO_2 during HD with a stable HD and CHF status equipped with NPPV. *BP* blood pressure, *rSO_2* regional saturation of oxygen, *HD* hemodialysis, *CHF* congestive heart failure, *NPPV* non-invasive positive-pressure ventilation, *SpO_2* saturation of percutaneous oxygen, *FiO_2* fraction of inspiratory oxygen

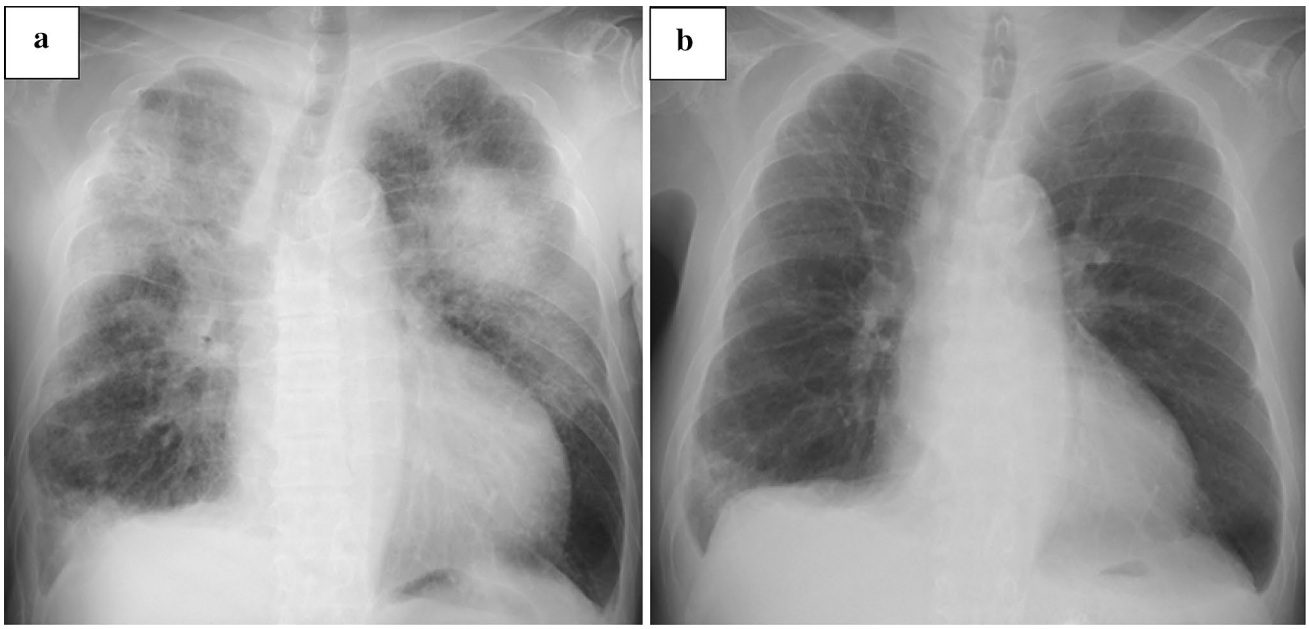

Fig. 1 Chest X-ray findings. **a** On admission. **b** At discharge

the greater ultrafiltration, unlike the previous cerebral rSO_2 monitoring under the CHF status.

Discussion

Patients with HF and low ejection fraction have generally their poor prognosis compared with those without cardiac dysfunction [13]. In addition, many HD patients are likely to represent CHF status induced by the body-fluid excess, leading to the deterioration of systemic oxygenation including the brain. Therefore, adequate body-fluid management is necessary to prevent the deterioration of cardiac function and improve the prognosis of patients undergoing HD.

Several recent reports have detailed continuous and non-invasive monitoring of regional tissue oxygenation using NIRS in the field of dialysis therapy [7–11]. Cerebral rSO_2 levels in HD patients were lower than those in healthy subjects [7, 8]. Furthermore, cerebral rSO_2 deterioration was confirmed in addition to the decrease in SpO_2 in an HD patient with sleep apnea [12]. Recent studies reported that cerebral rSO_2 values did not change before versus after HD [7, 9, 11]. The rSO_2 of this patient was maintained under stable HD status without CHF, consistent with previous reports. However, there was a remarkable impairment in cerebral oxygenation under the CHF status associated with excess body-fluid in this patient, and ultrafiltration during HD induced the cerebral oxygenation improvement even under the decrease of the inhaling oxygen concentration. Thus far, reports about the changes in cerebral oxygenation during HD with CHF status were limited; therefore, it would be meaningful to describe the changes in cerebral rSO_2 during HD with ultrafiltration.

Intermittent hypoxia caused by HF or sleep apnea could generally induce reactive oxygen species or oxidative stress, and increased oxidative stress causes vascular injuries [14]. Additionally in HD patients, vascular calcification could progress because of mineral and bone disorders, diabetes mellitus and hypertension [15, 16]. Such vascular calcification would be correlated with cerebral rSO_2 values according to a previous report [17]; therefore, they might mutually impact each other. Additionally, low rSO_2 level in the brain was affected by various factors, including low serum albumin, anemic state, HD duration, and hypotension [8, 18–22]. In particular, intradialytic hypotension could decrease cerebral oxygenation [20, 21], and repeated hypotension events could cause brain atrophy [23], which might be associated with cognitive impairment. Cerebral rSO_2 recently showed a significant and positive correlation with cognitive assessment in non-dialyzed CKD patients [24] as well as HD patients [25]. Furthermore, in the field of cardiovascular disease, the management of cardiac function and body-fluids would be important to prevent the deterioration of cognitive function because of the progression of cognitive impairment in patients with CHF [26]. Therefore, the recurrent decrease of cerebral oxygenation accompanied by CHF status should be avoided to maintain cognitive function. However, the association between cerebral oxygenation decrease induced by the CHF status and cognitive impairment in HD patients remains uncertain; therefore, further studies are needed to clarify the association between the two factors.

In conclusion, in our experience treating an HD patient with acute CHF, cerebral oxygenation deteriorated with CHF status and could be improved by adequate body-fluid management during HD.

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Compliance with ethical standards

Conflict of interest The author(s) declare that they have no conflict of interest.

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