Jpn J Apheresis **16** (1): 221-222, 1997

Continuous Hemofiltration and Continuous Hemodiafiltration in Patients with Septic Multiple Organ Failure

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Key words: continuous hemofiltration, continuous hemodiafiltration, hemodynamics, oxygen metabolism

Introduction

Septic multiple organ failure (MOF) is a common cause of death in critically ill patients. Septic MOF results in the activation of numerous mediators, defective oxygen utilization in peripheries and abnormal vascular tone.[1] The aim of this study is to determine the cause of death of patients with septic MOF treated with continuous hemofiltration (CHF) and continuous hemodiafiltration (CHDF) to eliminate causative MOF substances such as humoral mediators.[2,3]

Materials and Methods

A total of seventeen patients with septic MOF after digestive surgery were divided into two groups: Group S, seven survivors; and Group D, 10 patients who had died. There was no significant difference in age, number of failing organs or duration of CHF/CHDF between the two groups. Hemodynamics and oxygen metabolism can be readily and repeatedly measured using a systemic arterial catheter and a Swan-Ganz catheter, which can measure arterial and venous pressures of the systemic and pulmonary circulations, cardiac output, arterial and mixed venous gases, and hemoglobin. Measured and calculated hemodynamics included the cardiac index (CI), stroke volume index (SVI), left and right ventricular stroke work index (LVSWI and RVSWI), total peripheral vascular and pulmonary arteriolar resistance index (TPRI and PARI, respectively), oxygen delivery index (DO_2I), oxgen consumption index (VO_2I) and oxygen extraction ratio (O_2ER).[3,4] Blood purification by CHF/CHDF was begun when patients, after digestive surgery, developed MOF[3] including acute renal failure (15 patients) and acute respiratory failure (two patients). All values are expressed as the mean ± standard deviation. A p-value less than 0.05 was considered statistically significant by nonparametrics.

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Results

The CI increased significantly and SVI increased after CHF/CHDF as compared with before CHF/CHDF in Group S, whereas the CI and SVI remained unchanged in Group D (Table 1). The LVSWI increased significantly and RVSWI increased after CHF/CHDF as compared with before CHF/CHDF in Group S, whereas the LVSWI and RVSWI remained unchanged in Group D. Both the pulmonary arterial wedge pressure (PWP) and right atrial pressure (RAP) decreased after CHF/CHDF as compared with before CHF/CHDF in Group S, whereas the PWP and RAP remained unchanged in Group D. The TPRI decreased significantly and the PARI decreased after CHF/CHDF as compared with before CHF/CHDF in Group S, whereas both the TPRI and PARI remained unchanged in Group D. The DO_2I increased significantly and the VO_2I increased after CHF/CHDF as compared with before CHF/CHDF in Group S, whereas the VO_2I failed to increase despite an increase in the DO_2I in Group D.

Discussion

Compared to hemodialysis, CHF induces less hemodynamic instability, more gradual changes in osmolarity with minimal complement activation and, increasingly important, require fewer specialized personnel.[5] Furthermore, CHDF was developed. By the addition of a diffusive component to CHF, adequate metabolic control can be obtained in severely catabolic patients, while retaining the advantages of CHF.[2,3,5] In general, these techniques are indicated in acute renal failure (ARF) patients with hemodynamic instability or severe fluid overload.[5] These techniques are now widely applied for intesive care unit patients on many indications and frequently independent of the presence of ARF.[5] Effective removal of several humoral mediators (MW varying from 600 to 16,500 daltons) was demonstrated in MOF patients

Table 1 Hemodynamics and oxygen metabolism in blood purification (BP)

		Group S		Group D	
		before BP	after BP	before BP	after BP
CI	($l/min/m^2$)	3.94 ± 0.69	4.99 ± 1.08 *	4.99 ± 1.97	4.69 ± 1.6
SVI	(ml/m^2)	43.9 ± 9.4	54.6± 11.7	45.0 ± 15.3	44.3 ± 14.4
LVSWI	($g\text{-}m/m^2$)	51.3 ± 17.2	64.4 ± 17.3 *	43.3 ± 17.4	48.5 ± 21.2
RVSWI	($g\text{-}m/m^2$)	8.09 ± 2.41	9.34 ± 4.11	10.5 ± 5.4	10.67 ± 5.66
PWP	(mmHg)	12.7 ± 4.3	11.4 ± 3.95	11.2 ± 2.1	10.4 ± 4.4
RAP	(mmHg)	9.4 ± 4.0	8.3 ± 1.7	8.0 ± 2.8	8.0 ± 3.6
TPRI	($dyne\cdot sec\cdot cm^{-5}/m^2$)	1877 ± 406	1490 ± 368 *	1470 ± 667	1569 ± 557
PARI	($dyne\cdot sec\cdot cm^{-5}/m^2$)	223 ± 134	160 ± 70	250 ± 141	267 ± 145
MAP	(mmHg)	98 ± 6	98 ± 16	87.4 ± 24.2	92 ± 17
HR	(/min)	94 ± 9	93 ± 15	115 ± 23	107 ± 17
DO_2I	($l/min/m^2$)	554 ± 118	661 ± 195 *	582 ± 324	610 ± 252
VO_2I	($l/min/m^2$)	153 ± 25	172 ± 22	168 ± 55	162 ± 54
O_2ER	(%)	19.8 ± 8.1	26.6 ± 4.5	33.0 ± 12.5	26.7 ± 9.0

*statistically significantly difference ($p<0.05$) in the Group (mean ± SD)

treated with CHF/CHDF.[2,5] Coraim et al [6] reported that toxic substances related to shock, such as cardiodepressant factors, can be removed by CHF. Removal of these pathogenic factors during MOF as well as other toxic substances such as humoral mediators by CHF/CHDF, may account for the improvements we observed in the cardiac functions such as CI, SVI, LVSWI and RVSWI, of the survivors.[3] A decreased preload of PWP and RAP can be explained by fluid removal from the survivors. A disproportionate rise in oxygen consumption relative to cardiac index was observed in adult respiratory distress syndrome patients.[7] However, increased oxgen delivery and oxygen consumption were observed in the survivors, whereas hemodynamics and oxygen metabolism were not improved in the nonsurvivors. The differences between the survivors and nonsurvivors of the septic MOF patients treated with CHF/CHDF may be explained by differences in the underlying diseases or differences in the severity of the disease that led to the development of MOF.

Conclusion

We conclude that hemodynamics and oygen metabolisn may be useful markers of a better prognosis for patients with septic MOF treated with CHF/CHDF.

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