

A222

Title: A RAPID INCREASE IN SODIUM IS ASSOCIATED WITH CPM AFTER LIVER TRANSPLANTATION

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Central pontine myelinolysis (CPM) is a demyelinating degeneration of the central part of the pons that can cause dysarthria, dysphagia, quadriplegia, and death. A rapid rise in serum sodium (Na) level has been described as a cause of CPM.¹ CPM has been diagnosed in 14 to 28% of adult patients submitted to postmortem neuropathologic examination after orthotopic liver transplantation (OLT).^{2,3} The purpose of this retrospective study was to evaluate the relationship between abnormalities in Na and histologically confirmed CPM after OLT.

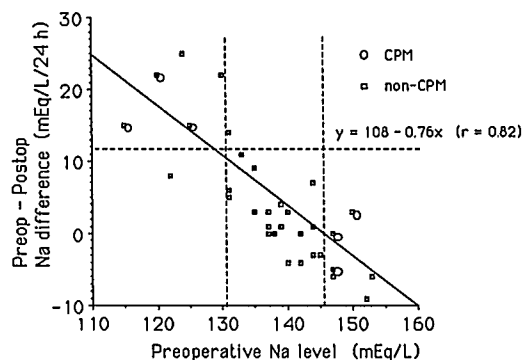
METHODS: The records of all adult patients who underwent OLT at the University of Pittsburgh from 1983 through 1988 and had a neuropathologic examination after death were reviewed. Na level was measured daily during hospitalization and hourly during surgery. Hyponatremia was defined as Na < 130 mEq/L and hypernatremia as Na > 145 mEq/L. When Na increased more than 12 mEq/L/24 h, the increase was considered rapid.¹ The patients were divided into two groups based on histopathologic findings: Group 1 patients (n = 6) had CPM and Group 2 patients (n = 29) did not. Data were analyzed using Fisher exact test and Pearson coefficient.

RESULTS: The two groups were similar regarding demographic variables and etiology and clinical course of liver disease. Preoperatively, Group 1 patients had a higher incidence of abnormal Na levels than Group 2 patients (hyponatremia, 3/6 vs 2/29 and hypernatremia, 3/6 vs. 3/29) (p < 0.01). A rapid perioperative increase in Na was observed in 3/6 in Group 1 patients and in 3/29 in Group 2 patients. The difference in Na level between preoperative and postoperative values correlated negatively with preoperative Na level (r = 0.82, Fig. 1) and positively with NaHCO₃ dose administered (150 vs 280 mEq) (r = 0.7). A rapid increase in Na was observed at least 48 h before death in 5/6 of Group 1 patients and 5/29 of Group 2 patients. CPM was diagnosed in 3/6 of patients with a rapid rise in Na compared with 1/29 of patients without this increase (p < 0.01). The amount of blood products and fluids given during the surgery were similar in the two groups. The survival time after OLT was similar in both groups, with sepsis the major cause of death.

DISCUSSION: In this study, preoperative hyponatremia and a rapid increase in Na at least 48 h before death appeared to be important risk factors for development of CPM. To prevent this rapid increase in Na, hyponatremia should be corrected slowly before OLT. During OLT, correction of metabolic acidosis by administration of THAM (tris-hydroxymethyl-aminomethane), instead of NaHCO₃, would reduce Na load. The metabolic effects of THAM in these patients, however, require further examination.

REFERENCES

1. Am J Med 88:557-560, 1990.
2. Neurology 39:493-498, 1989.
3. Transplantation 48:1006-1012, 1989.



A223

Title: RIGHT VENTRICULAR FUNCTION AFTER ORTHOTOPIC HEART TRANSPLANTATION

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Acute right ventricular failure (RVF) occurs frequently after orthotopic heart transplantation (OHT).^{1,2} The aim of this study was to evaluate the right ventricular (RV) function immediately after OHT and to determine whether preoperative pulmonary vascular resistance (PVR) and myocardial ischemic time (ISCH) could influence the postoperative RV function.

Methods: Twelve unpremedicated males (47±14 yrs; mean ± SD) were studied following informed consent and institutional approval. All patients were in sinus rhythm and none had clinical evidence of tricuspid regurgitation. Myocardial protection of the donor heart was identical for all patients. All received isoproterenol maintaining heart rate (HR) between 100 and 120 bpm. The following protocol was used for standard hemodynamic measurements: baseline (arrival in the ICU), 4 (T4), 12 (T12), 24 (T24) and 48 (T48) hours after the end of cardiopulmonary bypass (CPB). Cardiac output (CO), right ventricular ejection fraction (RVEF) were determined using a Swan-Ganz thermodilution catheter with a rapid response thermistor (93A-431H-7.5-Fr, Edwards Lab.) and an ejection fraction/cardiac output computer (REF-1™, Edwards Lab.). Cardiac index (CI), RV stroke volume index (RVSVI), RV end diastolic volume index (RVEDVI) and RV end systolic volume index (RVESVI) were calculated according to standard formulae. Statistical analysis: ANOVA for repeated values and least square linear regression between preoperative PVR and CI, RVEF, RVEDVI at each time, and between ISCH and CI, RVEF, RVEDVI at each time. Data are expressed as mean ± SD. p<0.05 was considered significant.

Results: Preoperative PVR was low in all the patients (2±0.7 Wood Units, Wu). Mean ISCH was 165 min (range: 94 to 193). Hemodynamic parameters are summarized on table. There was no significant correlation between preoperative PVR and CI, RVEF, RVEDVI at each time, and between ISCH and CI, RVEF, RVEDVI at each time. No significant changes occurred throughout the study.

Discussion: it was found that the RV function remained altered over the first two days after OHT, without being influenced by preoperative PVR and ISCH. A previous study indicates significant improvement of CI and RVSVI in patients with preoperative PVR less than 2 Wu, but not in these with preoperative PVR greater than 3 Wu.² In the present study the preoperative PVR was less than 3 Wu and CI was kept in a normal range. These results showed that RVF could occur after OHT despite normal preoperative PVR.

References

- 1-J Heart Transplant 9: 644-651, 1990.
- 2-Crit Care Med 17: S14, 1989.

	baseline	T4	T12	T24	T48
HR(1)	125±12	120±15	111±14*	112±9*	109±9*
MAP(2)	71±11	78±11	77±9	84±9*	87±12*
RAP(2)	9±6	8±6	8±4	10±4	10±4
MPAP(2)	22±4	21±6	19±4*	23±5	22±5
PCWP(2)	11±4	9±4	9±2	12±4	13±4
CI(3)	3.8±1.5	3.8±1.3	3.6±0.8	3.9±0.8	4.1±1.2
RVESVI(4)	82±35	86±35	85±43	91±42	92±3
RVEDVI(4)	119±43	120±31	123±43	124±44	129±36
RVEF(5)	31±13	30±13	32±11	29±10	30±9

(1):bpm;(2):mmHg;(3):l.min⁻¹.m⁻²;(4):ml.m⁻²;(5):%.

*p<0.05 vs baseline.