CARDIAC TROPONIN T (cTnT) INDICATES LESS CARDIAC DAMAGE IN DIALYSED PATIENTS WHO SWITCHED FROM LOW FLUX (LF) TO HIGH FLUX (HF) HEMODIALYSIS (HD)

Alicja E. Grzegorzewska 1, Krzysztof Cieszyński 2, Leszek Niepolski 3, Anna Sowińska 4

1Department of Nephrology, University of Medical Sciences, Poznań; 2Eurodial Dialysis Center, Ostrów Wlkp.; 3 B.Braun Avitum Dialysis Center, N. Tomyśl; 4Department of Statistics, University of Medical Sciences, Poznań, Poland

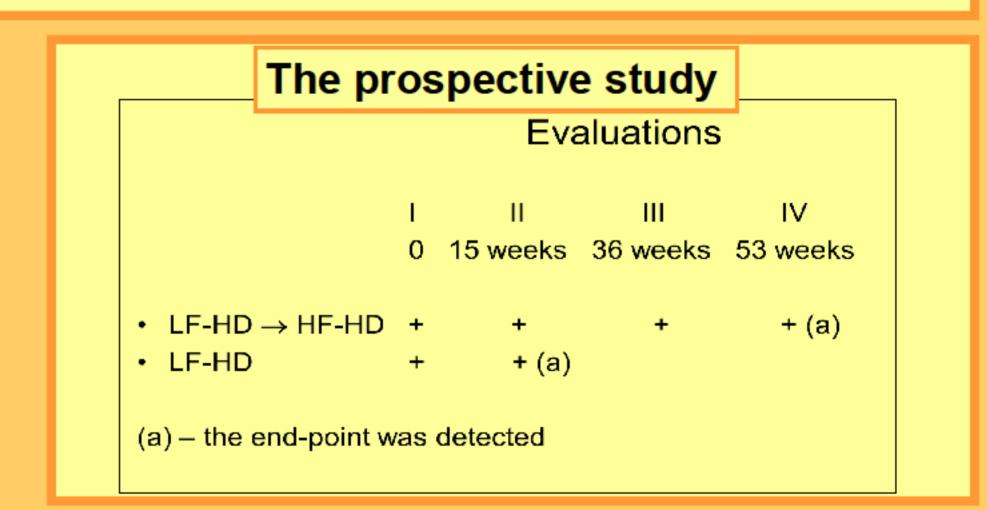
Objectives:

Methods:

Results:

CTnT is the established marker of myocardial damage. HF-HD has shown a minimal clearance of cTnT. Therefore, possible changes in cTnT in HF-HD patients are practically not related its transmembrane loss. More effective removal of uremic toxins may slow cardiac damage. Our aim was to show whether whether HF-HD may slow progressive cardiac damage evaluated by serum cTnT in comparison to the effect of LF-HD.

Group I of LF-HD patients (n=91) was switched to HF-HD (helixone membranes), whereas group II (n=65) continued LF-HD. Hs-cTnT (normal level <0.014 ng/mL) and clinical data were determined in each group at the start of the study and planned at 15, 36 and 53 weeks from the start. The end-point was a detection of increased cTnT levels compared to the initial results in patients who remained without cardiac events. In comparisons, results were adjusted for clinical and laboratory parameters, which differed both groups.



Parameter	$LF-HD \rightarrow HF-HD^{1}$ $n = 91$	Only LF-HD ² (prospective) n = 65	P value
Caucasians (n, %)	88 (96.7)	65 (100)	0.139
Male gender (n, %)	45 (49.5)	36 (55.4)	0.467
Metrical age (years)	66.8 12.9	60.5 14.9	0.010
Diabetes mellitus (n, %)	30 (33.0)	15 (23.1)	0.179
Diabetic nephropathy (n, %)	20 (22.0)	15 (23.1)	0.871
Chronic glomerulonephritis (n, %)	17 (18.7)	21 (32.3)	0.051
Hypertensive nephropathy (n, %)	17 (18.7)	6 (9.2)	0.098
Chronic tubulointerstitial nephritis (n, %)	9 (9.9)	0 (0)	0.009
Coronary artery disease (n, %) - Myocardial infarction (n, %)	23 (25.3) 16 (17.6)	10 (15.4) 6 (9.2)	0.136 0.137
PTCA and stent (n, %) CABG (n, %)	1 (1.1) 5 (5.5)	0 (0) 0 (0)	0.396 0.055
Cardiomyopathies (n, %)	26 (28.6)	18 (27.7)	0.902
Mitral valvular disease (n, %)	12 (13.2)	15 (23.1)	0.107
Aortal valvular disease (n, %)	5 (5.5)	2 (3.1)	0.476
Atrial fibrilation (n, %)	11 (12.1)	3 (4.6)	0.106
Heart stimulation device (n, %)	4 (4.4)	1 (1.5)	0.310
NYHA class - no or I (n, %)	30 (33.0) 49 (53.8)	19 (29.2) 34 (52.3)	0.614 0.853
- II (n, %)	12 (13.2)	11 (16.9)	0.520
- III (n, %) - IV (n, %)	0 (0)	1 (1.5)	0.241
Administration of antihypertensive drugs due to arterial hypertension or other reasons (n, %)	88 (96.7) 20 (22.0)	26 (40.0) 5 (7.7)	<0.0001 0.016
Poor control of hypertension (n, %) Cerebral stroke (n, %)	7 (7.7)	3 (4.6)	0.436
COPD (n, %)	9 (9.9)	2 (3.1)	0.102
RRT vintage (years)	2.45 (0.37 - 25.92)	3.27 (0.07 - 19.15)	0.539
Dialysis access	74 (81.3)	53 (81.5)	0.975
- Arterio-venous fistula (n, %)	16 (17.6) 0 (0)	25 (38.5) 6 (9.2)	0.003 0.003
- Arm (n, %) - Cubital fossa (n, %)	58 (63.7)	22 (33.8)	0.0003
- Forearm (n, %)	17 (18.7)	12 (18.5)	0.975
Permanent catheter (n, %)	257 17	258 26	0.115
Dialysis session duration (min) BQ (mL/min)	291 50	290 46	0.115
eKt/V	1.28 0.23	1.30 0.24	0.886
Body weight (kg)			
- Before dialysis	75.1 14.3	81.6 21.2	0.062
- After dialysis - Difference	73.3 14.1 1.8 1.0	79.1 20.6 2.3 0.8	0.077 0.001
Dry body weight (kg)	73.3 14.1	78.1 18.7	0.077
BMI (kg/m2)	27.8 5.8	28.7 6.2	0.275
Positive HBsAg (n, %)	0 (0)	1 (1.5)	0.241
Positive anti-HBc (n, %)	23 (25.3)	7 (10.8)	0.024
Positive anti-HCV (n, %) Positive HCV RNA (n, %)	19 (20.9) 10 (11.0)	5 (7.7) 4 (6.2)	0.024
Positive ric v KtvA (n, %) Positive anti-HIV (n, %)	0 (0)	0 (0)	0.302
Albumin (g/dL)	41.5 4.0	41.2 3.5	0.327
Hs-CRP (mg/L)	15.4 7.8	14.2 7.6	0.685
β2-microglobulin (mg/dL)	3.98 (0.99 - 13.9)	2.74 (0.085 - 6.56)	0.0004
WBC (G/L)	7.7 3.2	6.6 2.0	0.044
Hb (g/dL)	11.3 1.2	11.4 1.7	0.577
ALT (U/L)	14.0 (4 - 263)	13.0 (4 - 164)	0.095
AST (U/L)	17.0 (7 - 116)	13.0 (5 - 106)	0.004
GGT (U/L)	23 (6 - 401)	36.0 (5 - 235)	0.051
Ca (mg/dL)	9.0 0.7	8.6 0.8 5.0 1.5	0.004
P (mg/dL) Ca x P (mg2/mL2)	5.1 1.7 46.0 14.7	42.9 13.2	0.852 0.247
PTH (pg/mL)	337 216	581 478	<0.0001
ALP (IU/L)	94.0 (39 - 725)	98.0 (47 - 365)	0.177
Blood pH	7.37 0.04	7.36 0.04	0.053
Bicarbonate (mmol/L)	22.0 2.5	21.5 1.6	0.101
Total cholesterol (mg/dL)	183.7 44.1	169.7 38.8	0.035
LDL-Ch (mg/dL)	119.4 77.4	95.5 28.4	0.113
HDL-Ch (mg/dL)	42.7 13.8	42.2 10.5	0.872
Triglycerides (mg/dL)	196.4 102.6	161.4 69.2	0.063

Prevalence of coronary artery disease, myocardial infarction, NYHA classes, cardiomyopathies, atrial fibrilation, valvular disease, cerebral stroke, PTCA and stenting, CABG, placement of heart stimulation device did not differ both groups. However, patients of group I showed AV fistula predominantly on the forearm and lower PTH, whereas patients of group II were younger, had better control of hypertension, lower prevalence of anti-HCV and anti-HBc positivity, lower β2-m, WBC, AST, and total cholesterol with similar BMI.

Group I (LF-HD \rightarrow HF-HD)
Patients free from symptomatic cardiac events
during the entire study (n = 58)

0.4

0.3

0.268

0.2

0.1

0.0047

0.0048

0.0057

0.0055

0.0050

0.012

0.011

0.0012

0.012

0.012

0.012

0.012

0.012

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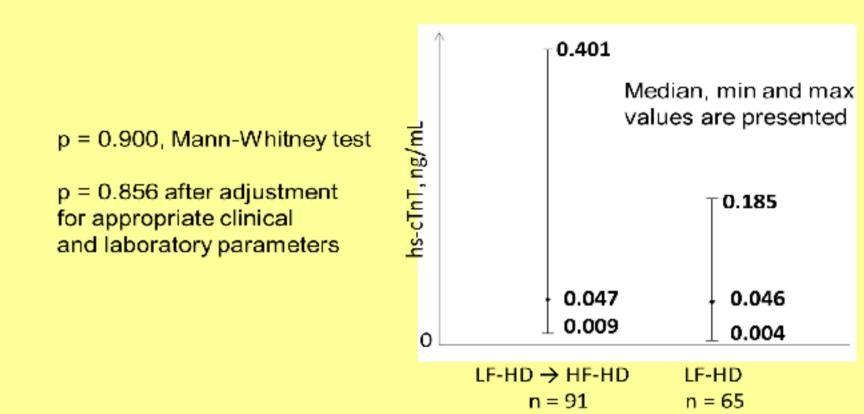
0.012

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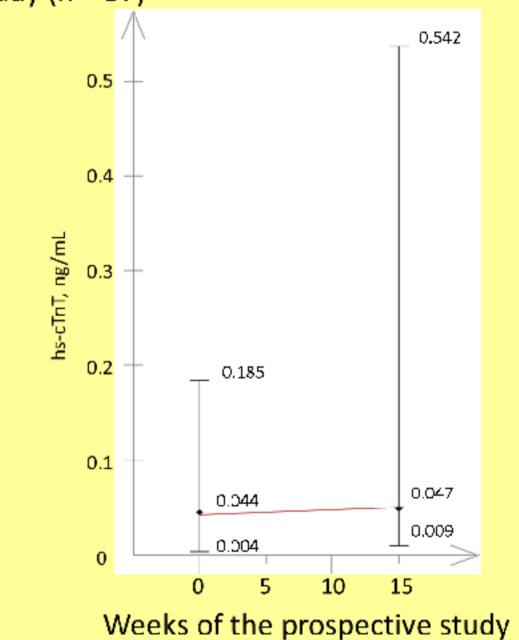
In patients of group I, who were free from symptomatic cardiac events, serum cTnT increased after 53 weeks since the start (n = 58, 0.047, 0.009 - 0.268; 0.048, 0.120 - 0.348; 0.057, 0.011 - 0.330; 0.055, 0.012 - 0.382 ng/mL in consecutive evaluations, p = 0.022 for the course, p< 0.05 i-IV).).

Hs-cTnT in HD patients at the beginning of the prospective study



At the start, groups did not differ in hs-cTnT.

Group II (LF-HD), Patients free from symptomatic cardiac events during the study (n = 57)



In group II, this increase was shown already after 15 weeks (n=57, 0.044, 0.004-0.185 vs 0.047, 0.009-0.542 ng/mL, p=0.028).

HF-HD (n = 91)
8 patients died
3 received renal graft
3 became unstable at 15 week of the study

LF-HD (n = 65)
3 patients died p = 0.492 p = 0.748*
1 received renal graft p = 0.641 p = 0.606*
0 became unstable at 15 week of the study p = 0.266 p = 0.999*

after adjustment for appropriate clinical and laboratory parameters

An increase in eKt/V (1.27 ± 0.23 vs 1.47 ± 0.23 , p<0.001) in group I was associated with a decrease in $\beta2m$ (3.8, 0.99-13.9 vs 2.8, 0.80-5.5 mg/dL, p<0.001) and P (5.0 ± 1.7 vs 4.3 ± 1.2 mg/dL, p<0.001), and an increase in blood pH (7.38 ± 0.04 vs 7.41 ± 0.05 mmol/L, p<0.001) and bicarbonate (21.7 ± 2.3 vs 24.6 ± 2.7 mmol/L, p<0.001), whereas in group II despite a slight increase in Kt/V (1.30 ± 0.24 vs 1.35 ± 0.28 , p=0.017) serum P (5.1 ± 1.6 vs 5.4 ± 1.5 , p=0.028) and WBC (6.6 ± 2.0 vs 7.2 ± 2.2 G/L, p=0.015) also increased.

HF-HD, n = 91

7 patients had cardiac events but returned to stability
Acute coronary syndrome, NSTEMI (4 patients)
Exacerbation of chronic heart failure (3 patients)

LF-HD (prospective),

n = 65

4 patients had cardiac events but returned to stability

Myocardial infarction (1 patient)
Exacerbation of chronic heart failure (1 patient)

Placement of artificial heart valves complicated with cardiac tamponade (1 patient)

Pulmonary oedema in the course of hypertensive crisis (1 patient)

p = 0.429 after adjustment for appropriate clinical and laboratory parameters

There were no significant differences between groups in the total death rate, cardiac death rate, frequency of non-fatal cardiac events, or unstable condition at completion of the 15th study week.

Conclusions:

An increase of hs-cTnT is more evident in stable LF-HD patients than in HF-HD ones. Changes induced by switch from LF-HD to HF-HD slow progressive cardiac damage evaluated by serum hs-cTnT. We propose hs-cTnT as a marker of progressive cardiac damage in the course of HD treatment.





