Arterial pressure changes on cardiac function during hemodialysis

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ABSTRACT

Left ventricular (LV) diastolic function has been causally deteriorated on hypotensive episodes during hemodialysis (HD). The influence of intravascular volume deterioration and arterial pressure reduction on LV function in patients without hypotensive episodes during HD has not been adequately studied. 23 patients on HD were studied for 48 ± 35 months, whose mean age was 50 ± 12 years, their mean body mass index was 24±4kg/m² and their mean fluid retention between HD sessions 2.2±1.1 kgr. We used 2D and Doppler echocardiography before and after the same HD session. The following parameters were measured: a) end-diastolic (LVED) and end-systolic LV diameters, left atrial diameter (LA) and fraction shortening (FS) b) isovolumic relaxation (IVRT) and contraction (ICT) time, deceleration time (DT), E and A waves of the mitral inflow and ejection time (ET) of the LV outflow velocity. The following indices were calculated: E/A ratio, myocardial performance index (MPI) using the (IVRT+ICT)/ET formula, mean arterial pressure (MAP), the % reduction in MAP and the reduction in intravascular volume after HD. Stroke volume (SV) and corresponding cardiac output (CO), were calculated from the LV outflow velocity waveform. All patients demonstrated systolic arterial pressure at the level of ≥90mmHg and normal FS before HD, while they had demonstrated restrictive LV diastolic filling before HD. MAP, SV, CO, LVED, LA, E wave and E/A decreased while IVRT increased after HD (p<0.01). After HD, low MPI values (normal range <0.44) were related to greater ultrafiltration volume loss (UFV) (r=0.53, p<0.01) and greater % reduction in MAP (r=0.62, p<0.01). Conversely, abnormal MPI values were related to high MAP after HD (r=0,44, p<0.01). Increased UFV was related to greater % increase in IVRT (r=0.43, p<0.01) and % decrease in E/A (r=0.40, p<0.01) after HD. Patients with UFV >3kg (7/23) had lower MPI (0.47 ± 0.2 vs. 0.84 ± 0.3 , p<0.01) and greater % increase in IVRT and % reduction in E/A (p<0.01) than those with <3kgr. Fluid retention between HD sessions leads to restrictive LV diastolic filling as shown by an E/A>1 and a short IVRT in patients before HD. Reduction in the excess intravascular volume after HD normalises MAP and thus, improves LV performance.

KEY WORDS: Echocardiography assessment, Cardiac function, Heart rate, Arterial pressure, Intravascular volume, Ultrafiltration volume, Chronic renal failure, Hemodialysis.

INTRODUCTION

Chronically hemodialysed patients demonstrates a high incidence of cardiovascular disease (1). It is also known that while the majority of patients on HD have clinical signs of heart failure only few of them demonstrate systolic dysfunction in echocardiography (2). In these patients a large variation of intravascular volume and arterial blood pressure (BP) occurs during HD.

The diastolic function of the left ventricle (LV) is important for keeping a normal stroke volume with low intracavitary pressures. The study of parameters that could influence the balance of the cardiovascular system in patient on HD is very important. Doppler echocardiography of the LV is a reliable method of measuring LV diastolic function. Diastolic dysfunction of the LV induced by a reduction in the intravascular volume is responsible for the hypotension of those patients during HD (3-5). The effect of acute changes of intravascular volume and of the variability of BP on the diastolic and systolic function of the LV in patients who did not demonstrate hypotension during HD has not been fully investigated.

METHODS

Study population

The study included 23 patients (16 men and 7 women) aged 50 ± 12 years with chronic renal failure (CHD) who were undergoing HD, aged 50 ± 12 years and whose mean duration of HD was 48 ± 35 months (Tab. I). They did not suffer from coronary artery disease, respiratory disease, cardiac rhythm abnormalities or infection and have not used any inotropic or other cardioactive drugs for 5 drug half-lives before the study.

Glomerulonephritis was the primary renal disease in 12 patients, whereas polycystic kidney disease was diagnosed in 4, reflux nephropathy in 2, diabetic nephropathy in 2, nephrolithiasis in 2, renal TBC in one and chronic pyelonephritis in one patient. Hemodialysis was performed for 4 hours, three times weekly with the use of bicarbonate dialysate, the blood flow rate ranging between 200-300 ml/min and the dialysate flow rate ranging between 400 and 500 ml/min. Their hematocrit was 35%. They were all receiving erythropoeitin therapy. They were also receiving phosphate binders, ferrum and folic acid per os. Parathyroid and thyroid function were within acceptable limits. Nine were smokers and 11 had hypertension

Moreover, these patients did not suffer from cardiac valve disease, cardiac muscle disease or infection. All patients had an echocardiogram half an hour before and immediately after HD. Their body weight, systolic, diastolic and mean arterial pressure (MAP) were measured. Heart rates (HR) were also assessed before and after HD. The ultrafiltration volume loss (UFV) was calculated by the reduction of body weight after HD. This change in weight was considered to represent the corresponding change of intravascular volume during HD. Institutional Ethics Committee approved the protocol and each subject gave an informed concept.

Echocardiographic measurements

The echocardiographic study was performed by means of a HP-Sonos 1500 and a 2.5MHz phase-array transducer.

The following indices were measured: a) left ventricular end-diastolic (LVED) and systolic LV diameters, left atrial diameter (LA) and fraction shortening (FS), b) isovolumic relaxation (IVRT) and contraction (ICT) time, deceleration time (DT), E

and A waves of the mitral inflow and ejection time (ET) of the LV outflow velocity. The following indices were calculated: E/A ratio, myocardial performance index (MPI) using the (IVRT + ICT)/ET formula, mean arterial pressure (MAP), the % reduction in MAP and the reduction in intravascular volume after HD. Stroke volume (SV) and corresponding cardiac output (CO), were calculated from the LV outflow velocity waveform utilizing the machine's software.

Statistical analysis

The distribution of the parameters examined was not normal, thus Mann-Whitney's non-parametric test was used for comparisons between groups. Spearman's correlation was used to assess relations between variables. A p value <0.05 was considered as statistically significant. All values are expressed as means \pm SD.

RESULTS

The clinical characteristics of our patients population are shown in Table I. Hemodialysis induced a significant reduction in MAP, SV, CO, LVED, LA, E, E/A, ET, with a concomitant increase in DT and IVRT, while A and MPI remained unchanged (Tab. II). The percentage (%) increase in IVRT and decrease in E/A correlated with a reduction in intravascular volume (r=0.60, p<0.01 (Fig. 1), r=0.51, p<0.05, respectively).

After HD, low MPI values (n value <0.44) were associated with greater UFV (r=0.53, p<0.01), greater % reduction in SV (r=0.41, p<0.05) and greater % reduction in MAP (r=0.62, P<0.01) (Fig. 2). Conversely, abnormal MPI values were related to high MAP after HD (r = 0.44, p < 0.01).

Patients with UFV loss > 3kg (7/23) had lower MPI and greater % increase in IVRT and % reduction in E/A (p<0.01) than those with < 3kg after HD (Tab. III, p<0.05). The two groups did not differ in age, duration of HD, hematocrit, history of hypertension, heart rate, cardiac output and in % reduction in BP after HD and baseline values of the echocardiographic indices before HD.

Table 1. Clinical characteristics of HD patients studied

Age (years)	50 ± 12							
Sex (M/F)	16 / 7							
BMI (kg/m^2)	24.4 ± 4							
Time on HD (in months)	48 ± 35							
UFV (kg)	2.2 ± 1							
MAP (mmHg)	98 ± 14							
HR (/min)	73 ± 10							
Hct (%)	36 ± 2							
Hypertension	11							
Smoking	9							
UFV= ultrafiltration volume, MAP=mean arterial pressure,								
HR=heart rate								

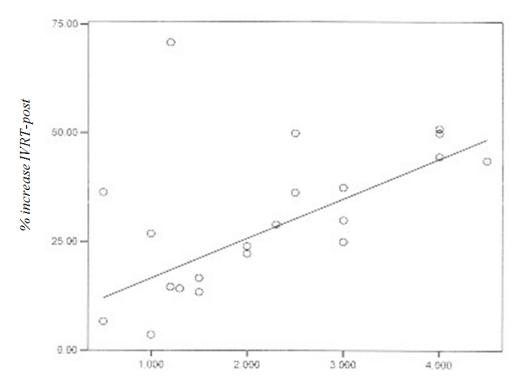
HD	MAP	HR	SV	CO	LVE	LV	LA	FS	Е	Α	E/A	DT	IVRT	ET	MPI
	mmH	pulse	ml	l/min	D	ES	mm	%	m/se	m/sec		msec	msec	msec	
	g	s/min			mm	mm			с						
PR	99±15	76±1	92±2	7±2	52±6	31±	43±5	$40\pm$	$0.8\pm$	0.8 ± 0	1.2±	184±	62±1	298±	0.6±0
Е		5	2			4		5	0.2	.2	0.2	52	4	85	.3
PO	86±12	83±2	73±2	6±2	48±6	30±	40±5	38±	0.6±	0.8±0	$0.8\pm$	211±	89±1	241±	0.7±0
ST		7	1			5		5	0.2	.2	0.2	60	9	54	.3
р	< 0.01	NS	< 0.01	< 0.0	< 0.0	NS	< 0.05	NS	< 0.0	NS	< 0.0	0.05	< 0.01	< 0.01	NS
				1	1				1		1				

TABLE 2. Results in all patients

TABLE 3. Patients with post-HD UFV

UFV	MAP mmHg	HR pulse s/min	SV ml	% DS V	CO l/min	LVE D mm	LV ES mm	LA mm	% FS	E m/se c	A m/se c	E/A	% DE/ A	DT mse c	IVR T mse	% DIV RT	ET mse c	M PI
										-	-			-	с			
<3	90±11	83±3	79±	15±	6.4±	50±	31.6	41±	37	0.6±	0.8±	0.9±	21±	223	92±	21±	239	0.8±
kg		1	20	9	3	6	±4	5	±4	0.2	0.2	0.6	20	±58	21	25	±61	0.3
n=16																		
≥3	76±10	84±1	60±	$30\pm$	5±1	44±	27.5	36±	39	$0.5\pm$	0.7±	0.6±	44±	184	85±	40±	244	0.47
kg		6	19	16		6	±5	5	±7	0.1	0.2	0.1	10	±60	9	35	±37	±0.2
n=7																		
р	< 0.01	NS	< 0.0	< 0.0	NS	0.03	0.06	<0.	NS	NS	NS	NS	< 0.0	NS	NS	0.02	NS	< 0.0
			5	5				05					1					1

%DSV: % difference SV,% DE/A : % difference E/A, %DIVRT=% difference IVRT.



Ultra filtration volume loss (UVF-kgr) post-HD Figure 1. *Correlation between ultrafiltration volume loss (UFV) and % increase IVRT-post-HD*.

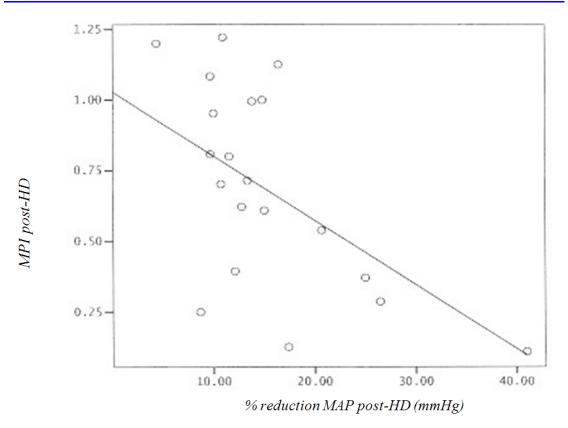


Figure 2. Correlation between % reduction MAP and MPI post-HD.

DISCUSSION

In the present study we have shown that patients with end-stage renal failure (ESRF) and normal systolic LV function before HD, demonstrate a restrictive LV diastolic fill possibly due to fluid retention between sessions. MAP, LVED, LA, E wave and E/A decreased, IVRT increased after HD, while the systolic BP has remained \geq 90mmHg throughout each session. Patients with a UFV of >3kg (7/23) had better MPI and greater % increase in IVRT and % reduction in E/A than those with <3kg.

Relaxation of the contracted myocardium occurs at the onset of diastole. The rapid pressure decay and the concomitant "untwisting" and elastic recoil of the LV produce a suction effect that augments the LA to LV pressure gradient, thereby promoting diastolic filling. During the later phases of diastole, the normal LV is composed of completely relaxed myocytes and is very compliant and easily distensible, offering minimal resistance to LV filling over a normal volume range. This can normally be accomplished by very low filling pressures in the left atrium and pulmonary capillary pressure (<12 mmHg) and the contribution of atrial contraction is relatively small.

Loss of normal LV diastolic relaxation and distensibility due to either structural (eg, LVH) of functional (ischemia, volume overload) causes impairs LV filling. This results in increases in LV diastolic, LA and pulmonary venous and pulmonary capillary pressures. The net effect is a relative shift of LV filling to the later part of diastole, with a greater dependence on atrial contraction.

They also present left ventricular hyperthrophy and dilatation. Coronary artery disease is also common in this population. For these reasons patients with ESRF may demonstrate systolic and diastolic dysfunction of the left ventricle (2). Other studies documented the presence of the LV filling abnormalities in patients on HD and correlated them with the clinical occurrence of hypotensive episodes during dialysis. This is possible due to the requirement of a higher preload to maintain adequate filling and cardiac output in a stiff ventricle (11). The reduction in intravascular volume during HD causes a concomitant reduction in pre and after load of LV. An inordinate decrease in preload could cause deterioration of LV diastolic filling and hypotension. Our findings are in agreement with those of Punzengruber et al (3) who found prolonged isovolumic relaxation time and increased peak velocity of the late ventricular filling, in patients on chronic maintenance HD. Rozich et al reported a significant prolongation of isovolumic relaxation time and reduction in early filling after HD. This appears to be so due to the reduction in preload suggested by these investigators.

Previous studies have evaluated the influence of HD induced altered loading conditions on Doppler echocardiographic indices of the LV in patients with CRF. It has been proposed that most of Doppler-derived indices of diastolic dysfunction are preload dependent. The use of MPI as a Doppler index of the global myocardial performance in diastolic dysfunction patients was introduced in 1995 by Tei et al. In a recent publication by Koga et al it was suggested that the MPI index is a reliable diagnostic tool in the evaluation of myocardial performance in dialysis patients (9). This index is preload dependent.

MPI is a new Doppler index (10), combining systolic and diastolic time intervals as an expression of global myocardial performance, has been shown to be of prognostic value in patients with dilated cardiomyopathy (6), cardiac amyloidosis, primary pulmonary hypertension (13) and Ebstein anomaly (14). This Doppler index of cardiac function was proposed and defined as the sum of isovoluic contraction and relaxation times divided by the ejection time. In addition, this index is not influenced by changes in ventricular geometry, in heart rate or blood pressure, which might alter the left ventricular filling pattern and affect the assessment of diastolic Doppler indices (13,16). MPI is an important incremental measurement in a comprehensive hemodynamic workup of patients with cardiac dysfunction since it is reproducible and easy to use (17-20).

After HD, low MPI values (n values <0.44) (12) were related to greater UFV loss and greater % reduction of MAP, thus confirming the beneficial role in the reduction of pre and afterload in LV function. Conversely, abnormal MPI values were related to high MAP after HD, suggesting the exacerbating effect on afterload in diastolic LV function.

In conclusion, fluid retention between HD sessions leads to restrictive LV diastolic filling as shown by a E/A>1 and a short IVRT in patients before HD. Reduction in the excess intravascular volume after HD normalises the MAP and thus, improves diastolic LV performance.

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