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PERFORMANCE INDICATORS OF LEFT VENTRICULAR STRAIN, WALL STRESS AND SERUM BRAIN NATRIURETIC PEPTIDE LEVELS IN CHRONIC HEMODIALYSIS PATIENTS

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ABSTRACT

Background: Left ventricular (LV) global longitudinal strain (GLS) is a reliable determinant of LV systolic function. The precise relationship between LV wall stress and serum brain natriuretic peptide (BNP) concentrations in hemodialysis (HD) patients require clarification. BNP levels are raised in patients with end-stage renal disease (ESRD) and could reflect LV impairment amongst HD patients.

Objective: This study sought to evaluate the clinical utility of LV–GLS, wall stress and serum BNP levels in chronic HD patients. The correlations between BNP levels with both LV wall stress and LV–GLS were assessed.

Methodology: A total of 30 ESRD patients on regular HD – divided into 15 patients with LV ejection fraction (EF) <50% and 15 patients with LV EF > 50% – and 15 age-matched healthy subjects were assessed. LV function and structure were measured using conventional echocardiography, including LV meridional wall stress (LVMWS), LV mass index (LVM I) and two-dimensional speckle tracking echocardiography for determination of LV–GLS. Serum BNP levels were evaluated after HD sessions.

Results: There were significant increases in LVM SW (189.2 ± 81 vs. 72.2 ± 20.6 dynes/cm ^{2}2 1000, P < 0.0001), higher levels of BNP (1238 ± 1085.5 vs. 71 ± 23.4 pg/ml, P<0.0001), w hilst LV–GLS was significantly reduced (15.1 ± 3.1 vs. 20.8 ± 1.7%, P <0.0001) in HD patients, when compared to the controls. Higher values of LVMWS (246.9 ± 67.5 vs. 131.5 ± 43.6 dynes/cm ^{2}2 1000, P <0.0001) and BNP (1925.4 ± 1087 vs. 550.5 ± 496.5pg/ml, P < 0.0005) with further impairment of LV–GLS (13.8 ± 2.5 vs. 16.4 ± 5.4%, P < 0.05) were found in patients with LV EF 0 50% than those with LV EF > 50%. Serum levels of BNP were positively correlated with LVM I (r = 0.896, P < 0.0001) and LVMW S (r = 0.697, P < 0.0001), but negatively correlated with LV–GLS (r = 0 0.587, P < 0.0001).

Conclusion: LV–GLS and LVMWS are useful imaging markers for detection of LV dysfunction in HD patients. Serum BNP level is influenced by LV structural abnormalities, being regarded as a crucial hemodynamic biomarker in those patients.

Key words: Left Ventricular Strain, Global longitudinal strain, Serum Brain Natriuretic Peptide, LV wall stress, HD patients.

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1. INTRODUCTION

Cardiovascular disease (CVD) is a major reason of mortality and morbidity in patients suffering with chronic kidney disease (CKD) (1). Impairment of left ventricular (LV) morphology and functions are correlated with a poor cardiovascular prognosis, being frequently identified in CKD patients (2). In those patients, traditional echocardiography is unable to detect early deterioration of cardiacfunction.

Ejection fraction (EF) occurs in the majority of of CKD patients, despite some having a high prevalence of CVD and progressive heart failure symptoms (HF) (3). Moreover, several research projects have reported that less than a third of patients with end-stage renal disease (ESRD) demonstrated evidence of LV systolic dysfunction (4,5). However, this contradiction is related to the complex pathophysiology of CVD in CKD, taking into account the technical limitations of EF measurement as an additional factor. The standard method of EF measurement entails precise tracing of the endocardial border and is operator-, volume- and load-dependent, resulting in limited reproducibility (6-7).

There is a growing interest in the current literature for other echocardiographic modalities to assess LV function. This is particularly appropriate to those CKD patients w ho exhibit progressive cardiac remodelling. Global longitudinal strain (GLS) assessed using semi-automated speckle-tracking echocardiography (STE) is a new technique for detecting and quantifying subtle impairment in LV systolic function (8,9). GLS reflects the longitudinal contraction of the myocardium and its accuracy has been validated against tagged magnetic resonance imaging (MRI) (10). This method is operator-independent and more reproducible than EF, being easily measured and integrated with a standard echo-Doppler study (10). Several reports show that measuring GLS with automated function imaging softw are is robust, objective, efficient and reproducible, being used to measure LV systolic function promptly (8,9).

The B-type natriuretic peptide (BNP) is synthesised in the ventricular myocardium in response to ventricular stretching and wall stress (11). Serum BNP levels are associated with the severity of HF and LV function, being considered to be useful indicators of diagnosis,management, and prognosis in patients with normal renal function[11]. The prognostic potential of serum BNP concentrations has been investigated in several studies of patients with CKD and those on hemodialysis (5).

Although CKD is frequently associated with disturbances in CV hemodynamics, the mechanisms responsible for the increase of BNP circulation levels in this condition still remain to be elucidated (13). Furthermore, renal failure *per se* has also been shown to affect the circulating levels of BNP, this condition not being significantly altered by renal replacement therapy (14) .

There is evidence to suggest that circulating BNP levels could reflect the LV end-diastolic wall stress both in patients with systolic and diastolic HF, a correlation maintained even in the presence of significant renal failure (15). However, little is known about the association of serum BNP levels with LV–GLS and LV wall stress in those CKD patients receiving regular hemodialysis. The research aimed to evaluate the clinical utility of LV–GLS, wall stress and serum BNP levels in chronic hemodialysis patients. The correlations between BNP levels with both LV wall stress and LV–GLS were assessed.

2 PATIENTS AND METHODOLOGY

2.1. PATIENTS

A cross-sectional observational study was undertaken on 30 ESRD patients on regular HD through arteriovenous fistula (AVF) and 15 age-matched healthy subjects as controls. Patients were allocated from the nephrology unit of the internal medicine department, Alzahraa University Hospital, during the period November 2016–May 2017. Written informed consent was taken from all patients. They were receiving bicarbonate base dialysate using low flux dialyser with an average blood flow of 300–350ml/min, during four hour sessions, three times per week. All patients in the study were aged over 18 years.

Excluded patient groups comprised those suffering acute coronary syndrome during the past six months, moderate to severe valvular heart disease, chronic atrial fibrillation, congenital heart disease, pregnancy, liver failure, chronic systemic inflammatory conditions, and those whose conditions reaulted in inadequate echocardiography imaging quality.

2.2. METHODS

Demographic and clinical data including comorbidities, medical history, and current cardiovascular medication via careful review of each patient's medical record:

i. Evaluation of LV wall stress, funct ions and hypertrophy using transthoracic echocardiography

Both patients and control persons were evaluated using transthoracic echocardiography (TTE), the former being assessed immediately after the dialysis session.

The TTE examination was performed using a Vivid E9 GE, Vingmed Ultrasound Machine produced in Horten, Norway, using a M-5Sc matrix probe (1.5–3.6 MHz). Comprehensive trans-thoracic M-mode, 2D and Doppler tests were undertaken using standard views (parasternal long axis, parasternal short axis, apical four and two-chamber and long axis views). Images were obtained at a rate of 50 to 70 frames per second, and saved for off-line analysis (Using the EchoPac 201, produced by General Electric Medical Systems).

The effect of afterload and preload on GLS was evaluated using LV wall stress measurements. The LV meridional wall stress (LVMWS) was assessed using the validated formula: LVMWS= [0.334 2 systolic BP 2 LV end diastolic diameter]/[LV wall thickness in end diastole 2 (1 + LV wall thickness in end diastole/LV end diastolic diameter)] dynes/cm^2 2 10,000(16).

Cardiac chamber measurements were undertaken using the methodology suggested by the American Society of Echocardiography, including left atrial (LA) diameter, LV end-diastolic diameter (LVEDD), LV end-systolic diameter (LVESD), end-diastolic interventricular septum thickness (IVS) and end-diastolic LV posterior wall thickness (LVPWd), measured using 2D or M-mode images, taken from parasternal long axis view s of the LV (17).

Biplane disksummation was used to undertake LVEF (%) calculations from 2D images of the LV obtained from apical four and two-chamber views (18).

LV mass was calculated using the Devereux formula LV massðgÞ¼ 1:04 2 hðLVEDD þ IVSþ LVPW dÞ3 0 ðLVEDDÞi 3 0 13:6; where 1.04 (g/cm^2) is the specific gravity of the myocardium. The LV mass index (LVM I, gm/m^2) was defined as LV mass divided by body surface area (m^2). The reference upper limits of normal LVM I by linear measurements are 95gm/m^2 in women and 115gm/m^2 in men (18).

Pulmonary artery systolic pressure (sPAP) was estimated by multiplying the square of the peak tricuspid regurgitant flow velocity by four (modified Bernoulli equation) and adding

the right atrial pressure, as estimated from the change in inferior vena caval diameter during inhalation.

The mitral annular early diastolic velocity (LV. E0) using a pulsed wave tissue Doppler test was obtained from six mitral annular sites (lateral, septal, inferior, anterior, posterior and antroseptal) and then averaged to calculate the average of the early myocardial diastolic, wave from these six annular sites (Av.E0). The LV diastolic function was evaluated by obtaining the ratio of LVE wave of mitral flow by pulsed Doppler/Av.E0 (E/Av.E0).

LV–GLS was assessed using 2D speckle tracking echocardiography. The endocardial borders were traced in the end-systolic frame of the 2D images, derived from the three apical views. Speckles were tracked frame-by-frame throughout the LV wall during the cardiac cycle, and basal, mid, and apical regions were studied. The GLS was calculated as the mean strain of 17 segments. A cut off at –16.5% has been shown to provide important risk stratification and prognostic value (19). Therefore, in our study we defined impaired GLS as >0 16.5%. HD Patients, compared to 15 age-matched healthy volunteers as a control group, were classified into two groups, according to LVEF:

- Group I: including HD patients with LVEF >50%
- Group II: including HD patient s with LVEF <50%.

Laboratory investigations: 6ml of venous blood were collected and divided into 4ml in a serum separator tube, allowed to stand in room temperature for 30 mins and then centrifuged at 3000rpm for 20 min. Thr serum was used for measurement of urea, creatinine, cholesterol, triglycerides, calcium and phosphorus (using a COBAS INTEGRA 400 Plus Autoanalyser, Roche, Germany), parathormone hormone (PTH) (using an E-COBAS Analyser, Roche, Germany), where the estimated glomerular filtration rate (eGFR) was calculated using EPI. CKD eGFR calculator. The remaining serum was stored at <20 6 C until measurement of BNP by enzyme-linked immunosorbent assay (ELISA). The remaining 2ml were placed in a vacutainer tube containing disodium ethylenediaminetetraacetic acid (EDTA) to undertake a complete a blood count (using a Sysmex KX-21N Analyser, Japan).

2.3. MEASUREMENT OF SERUM BNP

Human BNP Serum levels were measured using the quantitative sandwich ELISA kit (Bioassay, England/China, Cat# E1287Hu), according to the manufacturer'sinstructions. The detection range of the kit is 5–2000ng/ml. Each sample was run in duplicate and compared with a standard curve. The mean concentration was determined for each sample.

2.4. STATISTICAL ANALYSIS

Results were analysed using the SPSS softw are (version 16.0; SPSS Inc., Chicago, IL, USA). Descriptive statistics were presented as percentages, means and standard deviations (SD), where parameters with normal distribution were expressed as the mean 1SD. Univariate analysis for group comparisons was performed using the Student's t-test and one-w ay analysis of variance (ANOVA). The associations between variables were assessed by Pearson and Spearman's *r* correlation analysis. P <0.05 was accepted as statistically significant.

3. RESULTS

3.1. BASELINE DEMOGRAPHIC CLINICAL DATA AND SERUM BNP

The demographics of the patients and controls are provided in Table 1. No statistically significant difference was found between both groups regarding age (P > 0.05). The

participants were predominantly female, accounting for 53.3% of the patients' group and 80% of the control group. The patients' group had a high prevalence of hypertension (63.3%), hypercholesterolemia (56.7%) and diabetes mellitus (20%). In the HD patients, it was noted tht chronic hypertension represents the most common cause of ESRD (50%), followed by diabetes mellitus (16.7%).

Table 1.

Clinical and laboratory data of HD population.		
Demographic Data	HD patients (n = 30)	Control (n = 15) P-value
Age in years	51.2 ± 10.2	48.9 ± 6.6 NS
Sex:		
5 Male	14 (46.7%)	3 (20%)
5 Female	16 (53.3%)	12 (80%)
Risk factors:		
5 HTN	19 (63.3%)	
5 Hyperlipidemia	17 (56.7%)	
5 DM	6 (20%)	
Causes of CKD (no& %):		
5 HTN	15 (50%)	
5 DM	5 (16.7%)	
5 Analgaesic	1 (3.3%)	
5 UTI	1 (3.3%)	
5 FMF	1 (3.3%)	
5 SLE	1 (3.3%)	
5 Unknown	6 (20%)	
Duration of dialysis (months)	74.8 ± 35.6	
Blood pressure:		
5 SBP (mmHg)	130 ± 16.6	111.3 ± 8.3 <0.0001
5 DBP (mmHg)	79 ± 8	70 ± 8.5 <0.005
BNP (ng/ml)	1238 ± 1085.5	71 ± 23.4 <0.0001
S. creatinine (mg/dL)	10.1 ± 2.4	0.9 ± 0.2 <0.0001
eGFR ((mL/min/1.73 m^2)	12.5 ± 3	–

The values in the table were presented as a number (n) with the percentage in square brackets and the mean ± standard deviation (SD). HTN = hypertension, DM = diabetesmelitus, UTI = urinary tract infection, FMF = familial mediterranean fever, SLE = systemic lupus erythematosis, BNP = brain natriuretic peptide, S = serum, eGFR = estimated glomerular filtration rate.

The median duration of HD before the study was 74.8 ± 35.6 months. There were statistically signifi cant higher serum levels of BNP in the HD patients' group, when compared to the healthy group (P < 0.0001). Conventional, TDI echocardiographic and GLS parameters were analysed. Table 2 summarises the results of the LV dimensions, LV mass, LA, sPAP, and LV systolic and diastolic functions.

Table 2.

Echocardiographic parameters in HD patients' and control groups.		
Echocardiographic parameters	HD patients n = 30	Control n = 15 P value
LVEDD (mm)	53.1 ± 10.1	44.3 ± 4.4 <0.0005
LVESD (mm)	37.4 ± 9	27.5 ± 3 <0.0001
IVS (mm)	9.3 ± 2	9.1 ± 1.7 NS
LVPWd (mm)	8.9 ± 1.5	7.4 ± 1.1 <0.0005
LVMI (gm/m*2*)	115.3 ± 53	64.6 ± 14.8 <0.0001
LVEF-MM (%)	56.5 ± 9.5	67.1 ± 4.1 <0.0001
LV FS (%)	28.8 ± 7.2	37.2 ± 4.1 <0.0001
LVEDV4 (ml)	116.8 ± 46.7	81.3 ± 33.6 <0.01
LVESV4 (ml)	57.1 ± 27.3	31.4 ± 14.3 <0.001
LVEDV2 (ml)	104.8 ± 41.8	73.3 ± 22.9 <0.005
LVESV2 (ml)	50.6 ± 28.3	27.5 ± 8.7 <0.0005

There was a significant increase in the LV internal diastolic and systolic group. LVM I was increased significantly in HD patients (P < 0.0001). The sPAP and LA diameter were significantly higher in HD patients, when compared to the controls (P < 0.0001 for both).

The echocardiographic study demonstrated significant impairment of the cardiac functions of HD patients whencompared to healthy controls (Table 2). The LV–GLS was

significantly lower in HD patients, when compared with the controls (P< 0.0001), which denoted systolic dysfunction (Table 2 note: the more negative value of GLS, the better the LV systolic function will be). Furthermore, Table 2 shows a significant increase in LVMSW in HD patients, when compared to the healthy group (P<0.0001).

3.2. PATIENTS' CLASSIFICATION ACCORDING TO LV SYSTOLIC FUNCTION

Patients were categorised into two groups, according to the LV systolic function, which were measured using a modified Simpson's method with EF cut-off point of 50%. Group I comprised patients with a reduced LV systolic function (n = 15; of w hom 8 were female, EF <50%) with a mean age of 51.8 ± 10 years. Group II accounted for patients with preserved LV systolic function (n = 15; of w hom 8 were female, EF > 50%), with a mean age of 50.7 ± 10.7 years. There were no significant differences between the two dialysis groups (Group I and Group II) with respect to such conventional clinical measurements as age, diabetes mellitus, and hypertension.

Table 3 showed that, in this study, there were no significant differences in the majority of laboratory data between both groups. Patients with LV systolic impairment (Group I) were more likely to have lower serum calcium levels, when compared to those with preserved LV systolic function (Group II). There were no differences in the level of serum creatinine, parathormone hormone, phosphate and total cholesterol between both groups. It should be noted that BNP serum levels in patients with longitudinal strain LV meridional wall stress (LVMSW) (Group I) were significantly elevated, when compared to patients with a preserved LV systolic function (Group II) (P < 0.0005) (Table 3).

LVEF-biplane (%)	52.4 ± 8.8	62.2 ± 3.1	<0.0001
sPAP (mmHg)	43.4 ± 1.5	25.7 ± 4.9	<0.0001
E/A	1.1 ± 0.5	1.5 ± 0.4	<0.005
LV E/av.E0	19.2 ± 7.2	7.8 ± 1.2	<0.0001
LA (mm)	41.9 ± 7.3	30.8 ± 2.5	<0.0001
LV-GLS (%)	15.1 ± 3.1	20.8 ± 1.7	<0.0001
LVMWS (dynes/cm^2 2 1000)	189.2 ± 81	72.2 ± 20.6	<0.0001

* Values in the table were presented as mean ± standard deviation (SD). HD = hemodialysis, LVEDD = left ventricular end diastolic dimension, LVESD = LV end systolic dimension, IVS = LV inter-ventricular septum, LVPWd = LV posterior wall, LVM = LV mass, LVMI = LV mass index, LVEF-MM = LV ejection fraction by M-mode, LVEDV = LV end diastolic volume, LVESV = LV end systolic volume, 4 = four chamber view, 2 = two chamber view, sPAP = pulmonary arterial systolic pressure, E/A = ratio of early diastolic flow to late diastolic flow across the mitral valve, LV E/av.E0 = early diastolic wave velocity/average of early myocardial diastolic wave velocity at six mitral annulus sites, LA = left atrium, LV–GLS = LV – global longitudinal strain, LVMSW = LV meridional wall stress.

Systolic dysfunction was identified in patients with ESRD using decreased EF (either measured by M-mode or modifi ed Simpson's method) (P <0.0001 for both) and fractional of shortening (FS) (P < 0.0001) in conventional echocardiography, whilst a severe grade of diastolic dysfunction in the HD patients group was evident from the high E/Av.E0 ratio (P <0.0001), when compared to the healthy controls.

Table 4: Comparison of echocardiographic data of HD patients' groups (Group I and Group I

Echocardiographic Parameters	G1 (n = 15)	G2 (n = 15)	P value
LVEDD (mm)	59.4 ± 7.4	46.7 ± 8.4	<0.0001
LVESD (mm)	44.2 ± 5.7	30.7 ± 6	<0.0001
IVS (mm)	10.9 ± 1.8	8.5 ± 1.4	<0.0005
LVPWd (mm)	10 ± 1.6	8.5 ± 1.14	<0.01
LVEDV4 (ml)	143.4 ± 43.1	90.2 ± 33.9	<0.01
LVESV4 (ml)	76.9 ± 20.5	37.3 ± 16.7	<0.0001
LVEDV2 (ml)	132.1 ± 36.7	77.5 ± 25.9	<0.0001
LVESV2 (ml)	70.1 ± 26.4	31 ± 12	<0.0001

Values in the table were presented as a number (n) with the percentage in square brackets and the mean ± standard deviation (SD). LVEDD = left ventricular end diastolic dimension, LVESD = LV end systolic dimension, IVS = LV inter-ventricular septum, LVPWd = LV posterior wall, LVM = LV mass, LVMI = LV mass index, LVEF-MM = LV ejection fraction by M-mode, LVEDV = LV end diastolic volume, LVESV = LV end systolic volume, 4 = four chamber view, 2 = two chamber view, sPAP = pulmonary arterial systolic pressure, E/A = ratio of early diastolic flow to late diastolic flow across the mitral valve, LV E/av.E0 = early diastolic wave velocity/average of early myocardial diastolic wave velocity at 6 mitral annulus sites, LA = left atrium, LV–GLS = LV systolic dysfunction.

In Table 4, LV global conventional, TDI echocardiographic and GLS parameters have been summarised. The Group I patients had increased LV internal dimensions in diastole, LV end diastolic volumes, and the LA diameter, when compared to Group II patients. LVMI and sPAP were significantly higher in Group I patients (P <0.0001 and <0.05; respectively) than in Group II. Furthermore, both groups experienced a reversed E/A ratio, and high E/Av. E0 values, indicating the presence of increased LV filling pressure, which is indicative of LV diastolic dysfunction. E/Av.E0 values were statistically higher in Group I patients, w hen compared with patients in Group II (P <0.01).

LV–GLS was significantly reduced in Group I, unlike Group II (Table 4) (Fig.1). A significant increase in LVM WS was noted in Group I patients, when compared to those in Group II (P < 0.0001).

Group II patients were also assessed in relation to the healthy controls. There was no significant difference between both groups regarding age. Significantly higher levels of BNP serum were found in the Group II patients (P <0.005), when compared to the controls.

Table 3: Laboratory data of HD groups (Groups I and Group II)

Laboratory parameters	GI (n = 15)	GII (n = 15)	P value
HB (g/dl)	10.3 ± 1.6	9.7 ± -0.9	NS
WBCs (cel/mm^3)	8.1 ± 7.3	6.6 ± 2.7	NS
Platelets (cel/mm^3)	181.3 ± 57.3	220 ± 73.8	NS
BUN (mg/dL)	143.6 ± 39.6	165.3 ± 32.3	NS
S. creatinine (mg/dL)	10 ± 2.5	10.1 ± 2.3	NS
S. Ca (mg/dL)	8.9 ± 0.4	8.3 ± 0.7	<0.01
eGFR (mL/min/1.73m^2)	12.9 ± 3.2	12 ± 2.9	NS
S.Ph (mg/dL)	5.9 ± 2	5.4 ± 1.7	NS
PTH (pg/mL)	800.3 ± 581.4	436.8 ± 408.5	NS
Cholesterol (mg/dL)	141.1 ± 40.6	169.6 ± 52.4	NS
Triglycerides (mg/dL)	132 ± 53.3	155 ± 83.4	NS
BNP (ng/ml)	1925.4 ± 1087	550.5 ± 496.5	<0.0005

Values in the table were presented as a number (n), with the percentage in square brackets, and the mean ± standard deviation (SD). S= serum, BUN = blood urea nitrogen, Ca = calcium, Ph = phosphorous, PTH = parathormone hormone, BNP= brain

natriuretic peptide, eGFR = estimated glomerular filtration rate.

Regarding echocardiographic indices, Group II patients had significantly increased LA diameter (P <0.005), LVPWd (<0.05), E/A ratio (P <0.005), sPAP (P <0.01), E/Av.E0 (P<0.0001) and LVM WS (P <0.0001), with significant impairment of LV–GLS (P<0.0001). However, the authors could not find any significant difference between both groups in LV dimensions, volumes or EF when using either M-mode or biplane.

By comparing Group I HD patients with LV systolic dysfunction to healthy participants, we found no significant difference between both groups regarding age. We found statistically significant higher levels of serum BNP in Group II patients (P <0.0001), when compared to the controls.

As expected, the Group II HD patients with LV systolic dysfunction had a significantly increased LA diameter (P <0.0001), IVS (P <0.01), LVPWd (<0.0001), LV dimensions, volumes and LVEF (by M-mode and bi-plane) (P <0.0001). Group II HD patients had significantly higher levels of sPAP (P <0.0001), LVM WS (P <0.0001), more impairment of LV diastolic function defined by E/Av.E0 (P <0.0001), and significantly reduced LV systolic function detected by LV–GLS (P <0.0001).

3.3. CORRELATION OF SERUM BNP CONCENTRATIONS WITH RENAL FUNCTIONS AND DIFFERENT ECHOCARDIOGRAPHIC INDICES

No correlation was found between serum BNP levels and renal functions, either in serum creatinine or eGFR in HD patients.

We found significant positive correlations between serum BNP concentrations with LV Av.E/E0 (r = 0.512, P < 0.01), LVM I (r = 0.869, P < 0.0001) and LVM WS (r = 0.697, P<0.0001), whilst BNP serum levels correlated negatively with LV EF-biplane (r = 0 0.642, P < 0.001) and LV-GLS (r = 0 0.587, P <0.0001) (Figure 2).

4. DISCUSSION

The incidence of chronic kidney disease (CKD) is increasing across the world, being exponentially related to the risk of cardiovascular morbidity and mortality (20).

(A) LV–GLS = 10% (B) LV–GLS = 14.8% (C) LV–GLS = 21.3%

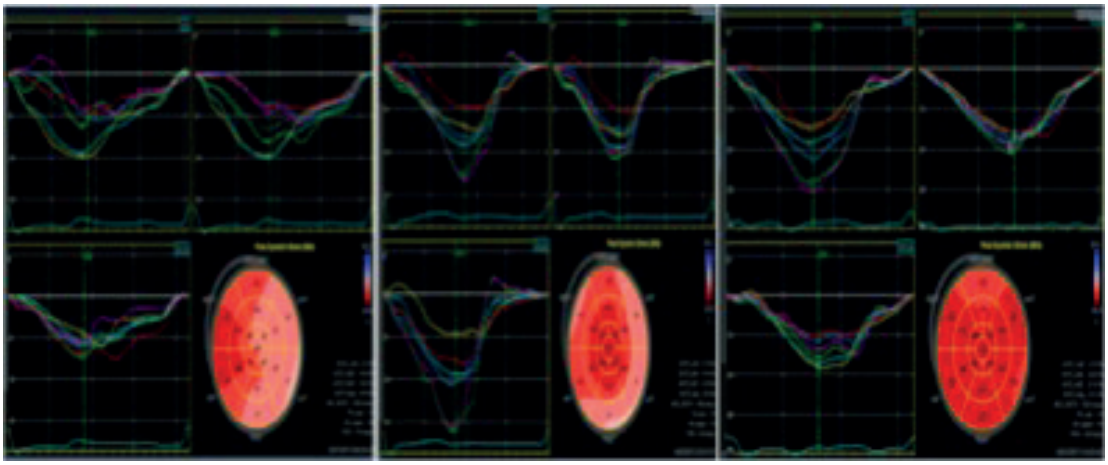

Figure 1: Ball's eye for 17 segments of LV–GLS in (A) Group I patient, (B) Group II patient and (C) control volunteer

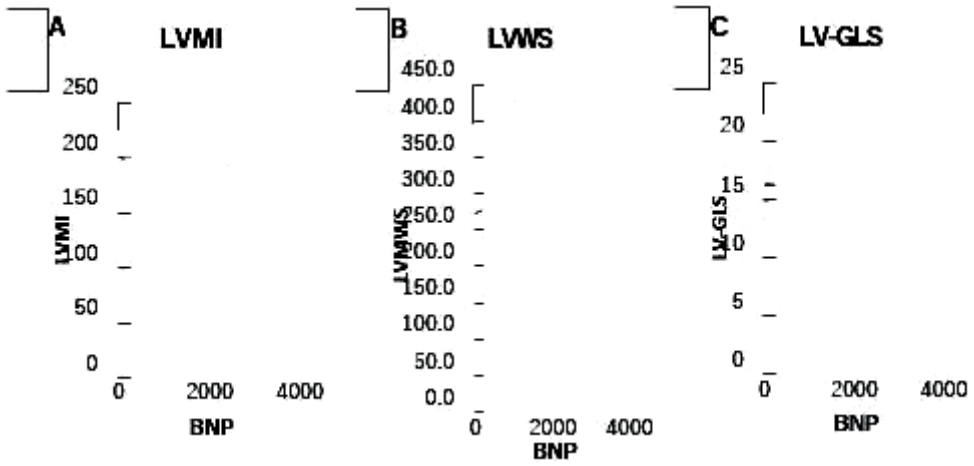

Figure 2: BNP serum concentrations and (A) LVMI, (B) LVMWS and (C) LV–LS. BNP correlated positively with LVMI (r = 0.869, P< 0.0001) and LVMWS (r = 0.697, P<0.0001). Serum BNP levels demonstrated a negative correlation with LV–GLS (r = 0 0.587, P<.0001). BNP = brain natriuretic peptide, LVMI = left ventricular mass index, LVMWS = LV meridional wall stress and LV-GLS = left ventricular global longitudinal strain.

CKD is associated with structural and functional LV remodelling, as a consequence of pressure and volume overload and nonhemodynamic factors (21). Pressure overload is the result of chronic hypertension and vascular stiffness, where as anaemia, arteriovenous fistulas, and sodium and water retention lead to volume overload. To keep LV wall stress close to normal, the LV responds to pressure and volume overload w th hypertrophy and dilatation (22). As LVH progresses, the interstitial space also increases with the accumulation of collagen (interstitial or replacement fibrosis), potentially causing a reduction in contractility. In addition, LVH increases the myocardial oxygen demand, which causes myocardial hypoperfusion, cardiomyocyte loss, and further interstitial fibrosis (23).

Furthermore, non-hemodynamic factors are associated with CKD, such as inappropriate renin-angiotensin-aldosterone system activation, oxidative stress, inflammation, and stimulation of prohypertrophic and profibrogenic factors, all of which contribute to LV remodelling (23). These structural changes cause impaired LV contractility, which can be detected by LV–GLS in addition to LV wall stress. Furthermore, it has been established that LV–GLS is a more sensitive marker of LV systolic dysfunction than LV ejection fraction (24). In the current study, mean LVEF in HD patients was >50% (assessed either by using M-mode or biplane methods) despite the significant increase of LV dimensions and volumes. However, mean LV–GLS amongst those patients was significantly reduced, suggesting that the LV contractility is significantly reduced, probably due to the ongoing LV remodelling that was evidenced by increased LVMWS, LVM I and a severe grade of LV diastolic dysfunction detected by the LV E/Av.E0 ratio.

Wang *et al*. (25), demonstrated the reliability of GLS in detecting subclinical systolic dysfunction in patients with LVH, when compared to the control group. Elevated serum BNP concentrations closely correlated with a significantly elevation of LVM I and LVM WS and LV E/Av. E' (marker of diastolic dysfunction), in addition to a significant decline of LV–GLS and LV EF in both patients' groups.

However, BNP levels were not found to be correlated with renal functions (neither serum creatinine, nor eGFR).

Confirming these results, Sanjuan *et al*., (27) observed a significant rise in the BNP concentration in patients on chronic dialysis. Renal insufficiency by itself does not appear to explain the serum BNP levels. A general consensus indicates that BNP is related to the stretching of myocardial fibres, following volume overload (28).

However, since BNP levels remain elevated after significant fluid loss following each dialysis session for both types of HD patient, other factors must also be involved (27). Bavbek *et al*., (29) and Sanjuan *et al*., (27) ascertained that there was an excellent correlation between BNP and LVM I, with LVM I being indicative of the BNP increase.

Sanjuan *et al*. (27), concluded that, in asymptomatic patients, marked increases in BNP levels may reflect very early stages of the pathological processes that precede the development of apparent cardiac signs (such as measurable LVH) in patients on extrarenal dialysis. Only echocardiographic parameters of cardiac dysfunction should be used as diagnostic criteria. The reverse was indicated in the study by Cataliotti *et al*. (30), which stated that BNP concentrations in HD patients without cardiovascular anomalies, hypertensive cardiopathy or ventricular dysfunction did not differ from those obtained from healthy subjects unafflicted by cardiovascular or renal pathology. Similar results were found by Akiba *et al*. (31), who found no differences in BNP levels in asymptomatic patients with and without renal insufficiency; consequently, renal insufficiency by itself does not appear to explain the increased serum BNP levels.

BNP is released from ventricular myocytes in response to LV wall stress and is a marker of cardiac distress (32). Serum levels of BNP are indicative of a reduction in LV function, and BNP levels correlate negatively with left ventricular ejection fraction, both in nonrenal and HD patients (33). BNP also reflects diastolic dysfunction (32).

Charfeddine *et al.* (34) concluded that, in patients with ESRD, the longitudinal and radial systolic functions are reduced, although the LVEF may remain within normal limits. This could be explained by the preservation of the circumferential functions. Two-dimensional speckle tracking echocardiography measures e-Val e T, according to Edvardsen T, *et al*. Non-invasive myocardial strain has the potential to detect the severity of uraemic cardiomyopathy in the early stages of the disease and may provide useful information for risk stratification in ESRD patients with preserved LVEF.

In agreement with our results, Niizuma *et al*. (35), found that BNP concentrations increased progressively with the grade of LV end diastolic wall stress (EDWS) in both the normal and CKD groups.

In the ESRD group, there were no significant differences between the low and middle EDWS groups. However, patients with ESRD and high EDWS indicated the highest serum BNP concentrations.

Furthermore, they concluded that renal dysfunction and high levels of log EDWS were both independent determinants of BNP concentrations.

Several limitations should be considered when interpreting these results. Firstly, the study population was relatively small. Secondly, only serum BNP concentrations after dialysis sessions were considered in our study. The authors suggest that it would give a more effective reading if BNP concentrations before and after dialysis were assessed.

5. CONCLUSIONS

The results suggested that LV–GLS and LVMWS, together with a degree of LV E/Av.E0 ratio, could be useful imaging markers for the detection of LV dysfunction (either systolic or diastolic) in HD patients. Serum BNP levels are influenced by LV structural and preserved EF in HD patients. They concluded that myocardial function was impaired not only in the longitudinal direction, but also in the circumferential direction, despite the preserved LVEF.

Liu *et al*. (26) demonstrated that, despite the preserved LV systolic function revealed by conventional echocardiographic parameters and TDI, deteriorating renal function is associated with a reduction of systolic function, as reflected by the decline of LV–GLS, circumferential strain and strain rate.

Our results revealed that HD patients (either with preserved or reduced LV EF) experienced a marked elevation of serum levels of BNP functional abnormalities, rather than the renal functions that w ould be a crucial hemodynamic biomarker in these high-risk patients.

CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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This study is not associated with any thesis or dissertation work.

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XÜLASƏ

Xroniki Hemodializ xəstələrində sol mədəciyin yüklənməsinin, divar gərginliyinin və serum BNP səviyyəsinin göstəriciləri

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İlkin məlumat: Sol mədəciyin (SM) uzununa genişlənməsi (UG) sol mədəciyin sistolik funksiyasının etibarlı determinantıdır. Hemodializli (HD) pasientlərdə sol mədəcikdə divar gərginliyi və beynin qan zərdabında natriuretik peptid (BNP) konsentrasiyaları arasındakı dəqiq qarşılıqlı əlaqəyə aydınlığın gətirilməsi tələb olunur. Beynin qan zərdabında natriuretik peptid səviyyələri böyrək xəstəliyinin son həddində (BXSH) olan pasientlərdə baş qaldırır və hemodializli pasientlərində sol mədəciyin funksiya pozuntusunu büruzə verə bilər.

Məqsəd: Bu araşdırmada xroniki hemodializ pasientlərində sol mədəciyin uzununa genişlənməsinin kliniki əhəmiyyəti, xroniki hemodializ pasientlərində ürək divar gərginliyi və beynin qan zərdabında natriuretik peptid səviyyələrini dəyərləndirməyə səy göstərilmişdir. Beynin qan zərdabında natriuretik peptid səviyyələrinin sol mədəcikdə divar gərginliyi, həmçinin uzununa genişlənmə ilə birlikdə qiymətləndirilmişdir.

Metodologiya: Müntəzəm hemodializdə olan cəmi 30 BXSM pasient sol mədəciyin ifraz nisbətinin (İN) müvafiq olaraq <50% və > 50% olduğu 15 nəfərdən ibarət iki qrupa ayrılmışdır və 15 nəfərdən ibarət yaş uyğunluğu olan sağlam şəxslər qiymətləndirilmişdir. Sol mədəcik funksiyası və quruluşu adi exokardioqrafiya, eləcə də sol mədəciyin meridional divar gərginliyi (SMMDG), sol mədəciyin kütlə indeksi (SMKİ) və ikiölçülü spekl trakinq exokardioqrafiyasından istifadə edilərək ölçülmüşdür. Sol mədəciyin genişlənmə səviyyəsini müəyyən etmək üçün beyində qan zərdabı səviyyələri hemodializ sessiyalarından sonra dəyərləndirilmişdir.

Nəticələr: Sol Mədəciyin meridional divar gərginliyində nəzərəçarpan artımlar (189.2 ± 81-yə qarşı 72.2 ± 20.6 dina/sm 2 2 1000, P < 0.0001), beynin qan zərdabında yüksək natriouretik peptid səviyyələri (1238 ± 1085.5-yə qarşı 71 ± 23.4 pg/ml, P<0.0001) müşahidə edildiyi halda, nəzarət tədbirləri ilə müqayisədə hemodializli pasientlərdə sol mədəciyin uzununa genişlənməsi əhəmiyyətli surətdə azalmışdır (15.1 ± 3.1-yə qarşı 20.8 ± 1.7%, P <0.0001). Sol mədəcikdə ifraz nisbətinin > 50% olduğu pasientlərlə müqayisədə ifraz nisbətinin 50% olduğu pasientlərdə sol mədəciyin meridional divar gərginlyi (246.9 ± 67.5-yə qarşı 131.5 ± 43.6 dina/sm 2 2 1000, P <0.0001), beynin qan zərdabında natriouretik peptid səviyyəsi (1925.4 ± 1087-yə qarşı 550.5 ± 496.5pg/ml, P < 0.0005) və sol mədəciyin uzununa genişlənməsi (13.8 ± 2.5-yə qarşı 16.4 ± 5.4%, P < 0.05) aşkar edilmişdir. Beyin qan zərdabında natriouretik peptid səviyyələri sol mədəciyin kütlə indeksi (r = 0.896, P < 0.0001) və sol mədəciyin meridional divar gərginliyi (r = 0.697, P < 0.0001) ilə müsbət əlaqəli olsa da, sol mədəciyin uzununa genişlənməsi (r = 0 0.587, P < 0.0001) ilə qarşılıqlı əlaqə mənfi olmuşdur.

Yekun: Sol mədəciyin uzununa genişlənməsi və sol mədəciyin meridional divar gərginliyi hemodializli pasientlərdə sol mədəciyin funksiya pozuntusunu aşkarlamaq üçün faydalı təsvir göstəriciləridir. Beyində qan zərdabında natriuretik peptid səviyyəsinə bu cür pasientlərdə mühüm hemodinamik biomarker hesab edilən sol mədəciyin struktur anormallıqları təsir edir.

Açar sözlər: Göstərici, Sol mədəciyin uzununa genişlənməsi, BNP, LV ürək divar gərginliyi, HD xəstələr.

РЕЗЮМЕ

Расширение левого желудочка, напряжение сердечной стенки и показатели сыворотки крови в мозге. Уровни натрийуретических пептидов у пациентов с хроническим гемодиализом

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Предварительная информация: Продольная дилатация (ПД) левого желудочка (ЛП) - надежный детерминант систолической функции левого желудочка. Пациенты, находящиеся на гемодиализе (ГД), должны выяснить точную взаимосвязь между натяжением стенки левого желудочка и концентрацией натрийуретического пептида (БНМ) в сыворотке мозга. Уровни церебрального натрийуретического пептида в сыворотке крови повышены у пациентов с терминальной почечной недостаточностью (ТПН) и могут проявляться дисфункцией левого желудочка у пациентов, находящихся на гемодиализе.

Цель: Это исследование было направлено на оценку клинического значения продольной дилатации левого желудочка у пациентов с хроническим гемодиализом, напряжения сердечной стенки у пациентов с хроническим гемодиализом и уровней натрийуретического пептида в сыворотке мозга. Уровни натрийуретического пептида в церебральной сыворотке оценивали в сочетании с натяжением стенки левого желудочка, а также с продольной дилатацией. Методология.

В общей сложности 30 пациентов с ТПН с секскреции левого желудочка (LVR) <50% и> 50%, соответственно, находящихся на регулярном гемодиализе, были разделены на две группы по 15 человек и сравнены с 15 здоровыми людьми, соответствующими по возрастным критериям. Функцию и структуру левого желудочка измеряли с помощью стандартной эхокардиографии, а натяжение меридиональной стенки левого желудочка (НМСЛЖ) и индекс массы левого желудочка (ИМЛЖ) также измеряли с помощью двумерной эхокардиографии со спектральным отслеживанием. Уровни сыворотки в головном мозге оценивали после сеансов гемодиализа, чтобы определить степень дилатации левого желудочка.

Результаты: В случае значительного увеличения меридионального натяжения стенки левого желудочка (72,2 ± 20,6 дина / см 2 2 · 1000 против 189,2 ± 81, P <0,0001), высокого уровня натрийуретического пептида в сыворотке мозга (71 ± 23,4 пг / против 1238 ± 1085,5). Мл. , P <0,0001), по сравнению с контрольными мероприятиями у пациентов, находящихся на гемодиализе, продольная дилатация левого желудочка была значительно снижена (20,8 ± 1,7% против 15,1 ± 3,1, P <0,0001). У пациентов относительной секрецией 50% по сравнению с пациентами с относительной секрецией > 50% были выявлены напряжение меридиональной стенки левого желудочка (131,5 ± 43,6 дина / см 2 2 1000, P <0,0001 против 246,9 ± 67,5), уровни натрийуретического пептида в сыворотке крови (1925.4 ± 1087 против 550.5 ± 496.5pg/ml, P < 0.0005) и продольной дилатации левого желудочка (16,4 ± 5,4% против 13,8 ± 2,5, P <0,05). Хотя уровни натриоуретического пептида в сыворотке положительно коррелировали с индексом массы левого желудочка (r = 0,896, P <0,0001) и меридиональным натяжением стенки левого желудочка (r = 0,697, P <0,0001), но взаимодействие с продольной дилатацией левого желудочка (r = 0 0,587, P <0,0001) была отрицательной.

Заключение. Продольная дилатация левого желудочка и меридиональное натяжение стенки левого желудочка являются полезными показателями визуализации для выявления дисфункции левого желудочка у пациентов, находящихся на гемодиализе. На уровень натрийуретического пептида в сыворотке крови головного мозга влияют структурные аномалии левого желудочка, которые являются важным гемодинамическим биомаркером у таких пациентов.

Ключевые слова: Индикатор, продольная дилатация левого желудочка, церебральная сыворотка, натяжение стенки сердца ЛЖ, пациенты с ГБ.