

# **FACTORS IMPACTING ON LEFT VENTRICULAR HYPERTROPHY IN HAEMODIALYSIS PATIENTS**

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fulfilment of the requirements for the degree  
of  
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# DECLARATION

I, James Chabu declare that this thesis is my own work. It is being submitted for the degree of Doctor of Philosophy in the University of the Witwatersrand, Johannesburg. It has not been submitted previously for any degree or examination at this or other University.

.....

30<sup>th</sup> day of May, 2008.

I dedicate this work:

To my supportive and understanding wife Adore, my children, Kabanda, Chabu and  
Lweendo for bearing with my long absence from home

To my mother Mrs. Lucy Musonda Kabanda and to the memory of my late father Mr.  
Joseph Chabu, who originally inspired me to take up medicine as a career

To God who has been so gracious and kind to me and made it all happen

## **PUBLICATIONS AND PRESENTATIONS ARISING FROM THIS STUDY**

Chabu J, Naicker S, Manga P, Norton G, Olorunju S. Inferior vena cava diameter and collapsibility index in haemodialysis patients correlates with left ventricular geometrical adaptations. World Congress of Nephrology, April 21-25, 2007. Rio de Janeiro, Brazil. S-P0-0196, page 101, WCN 2007 Book of Abstracts.

## ABSTRACT

Left ventricular hypertrophy (LVH) and increases in large artery stiffness predict cardiovascular outcomes in patients with renal failure. What determines left ventricular mass index (LVMI) and large artery stiffness and the contribution toward LVH and large artery dysfunction is not entirely clear. Consequently, this cross sectional study was aimed at assessing the various factors impacting on LVH in haemodialysis (HD), to contribute toward our understanding of the pathophysiology of LVH and large artery dysfunction in 94 adult HD patients. Pre- and post-dialysis blood pressures (BPs) were determined over 12 sessions of dialysis and averaged. Pulse wave analysis performed at the carotid, femoral and radial arteries was employed to determine pulse wave velocity (PWV) and central augmentation index (Aic). Echocardiography was performed to determine left ventricular mass (LVM) indexed to body surface area (LVMI). Natriuretic peptides, procollagen type I c-peptide (PIP), c-terminal telopeptide of type I collagen (ICTP), matrix metalloproteinases and their inhibitors were studied.

The prevalence of LVH was 72.8 % (67/92) .On multivariate analysis pre- ( $p \leq 0.005$ ), post- ( $p < 0.05$ ) and averaged dialysis ( $p < 0.015$ ) systolic BP were associated with LVMI and PWV. 24 hour ( $r = 0.260$ ,  $p = 0.026$ ), day ( $r = 0.247$ ,  $p = 0.036$ ), and night ( $r = 0.241$ ,  $p = 0.042$ ) systolic BP were not more closely associated with LVMI than the averaged dialysis systolic BP ( $r = 0.272$ ,  $p = 0.010$ ). Similarly 24 hour ( $r = 0.41$ ,  $p = 0.0003$ ), day ( $r = 0.400$ ,  $p = 0.0005$ ), and night ( $r = 0.416$ ,  $p = 0.0003$ ) systolic BP were not more closely associated with PWV than the post-dialysis systolic BP ( $r = 0.39$ ,  $p = 0.0001$ ) indicating that these BP measurements are as effective as 24-hour ambulatory BP in predicting cardiovascular

target organ changes. No relationship between either PWV ( $r=-0.08$ ), or A1c ( $r=-0.10$ ) and LVMI, between PWV ( $r=-0.11$ ), or A1c ( $r=0.03$ ) and LV MWT was noted. IVCD was independently associated with LVMI (partial  $r$  adjusted for average dialysis SBP=0.27,  $p=0.014$ ; partial  $r$  adjusted for 24-hour SBP=0.29,  $p=0.013$ ), and LV mean wall thickness ( $p<0.01$ ), but not with LV relative wall thickness ( $p=0.18$ ), or LV end diastolic diameter ( $p=0.88$ ). An association between IVCD and A1c (partial  $r$  adjusted for average dialysis SBP=0.21,  $p<0.05$ ), but not PWV was noted. NT-proANP and NT-proBNP were independently associated with LVMI ( $p<0.0001$ ) but neither were associated with IVCD independent of LVMI suggesting a close association with LVMI in HD. Serum concentrations of matrix metalloproteinases 1, 2 and 9, and their tissue inhibitors (1 and 2) were not associated with LVMI, remodelling or PWV and neither procollagen I nor the C-terminal telopeptide of type I collagen (ICTP) were associated with LVMI. Thus, factors impacting on LVH in this study were systolic BP, NT-proANP, NT-proBNP and IVCD.

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## **ABBREVIATIONS**

BP Ambulatory BP  
ABP Ambulatory BP  
ACE Angiotensin-converting enzyme inhibitors  
ACORD Anaemia correction in Diabetes study  
AIc Central augmentation index  
AIp Radial augmentation index  
ANP Atrial natriuretic peptide  
AVA Arterio-venous access  
BMI Body mass index  
BNP Brain natriuretic peptide  
BP Blood pressures  
BSA Body surface area  
CAD Coronary artery disease  
CHF Congestive heart failure  
CHOICE Choices for Healthy Outcomes in Caring for ESRD  
Ci Collapsibility index  
CKD Chronic kidney disease  
CRP C-reactive protein concentrations  
cSBP Central systolic BP  
cSBP Central systolic BP  
CV Coefficient of variation  
CVD Cardiovascular disease  
DBP Diastolic blood pressure  
Deceleration time deceleration time of (e)  
ECG Electrocardiography  
ECM Extracellular collagen matrix  
EF Ejection fraction  
EIA Enzyme immunoassay  
Epoetin erythropoietin  
ESRD End-stage renal disease  
Evaluation and Treatment of High Blood Pressure  
FS fractional shortening  
GFR Glomerular filtration rate  
HCU Hand-carried ultrasounds  
HD Haemodialysis  
Hypertension=1  
ICTP C-terminal telopeptide of type I collagen  
IHD Ischaemic heart disease  
IL Interleukin  
ISH Isolated systolic hypertension  
IVCD Inferior vena cava diameter  
IVCmin Minimum IVC diameter  
IVSTd Interventricular wall thickness

JNC VII Seventh Report of the Joint National Committee for the Prevention, Detection,  
 JVP Jugular venous pressure  
 K/DOQI Kidney Disease Quality Outcomes Initiative  
 Kt/V averaged equilibrated urea clearance  
 Left ventricular dilatation Cavity volume index  $>90,EF>/+50\%$   
 LV MWT Left ventricular mean wall thickness  
 LVCVI left ventricular cavity volume index  
 LV-Dilation = left ventricular dilation  
 LVEDD Left ventricular internal dimensions in end-diastole  
 Lvesd left ventricular end systolic diameter  
 LVESD Left ventricular internal dimensions in end-systole  
 LVH Left ventricular hypertrophy  
 LVM Left ventricular mass  
 LVMI Left ventricular mass index  
 LV-RWT Left ventricular relative wall thickness  
 LVSD left ventricular systolic dysfunction  
 MAP Mean arterial pressure  
 MHD Maintenance haemodialysis  
 MMP Matrix metalloproteinase  
 MMP-1 Human Matrix Metalloproteinase -1  
 MMP-2 Human Matrix Metalloproteinase -2  
 MMP-9 Human Matrix Metalloproteinase -9  
 NHLS National Health Laboratory  
 NT-proANP N-terminal -proanp  
 NT-proBNP N-terminal fragment of probnp  
 PDGF Platelet-derived growth factor  
 PIP Procollagen Type I C-Peptide  
 PP Pulse pressure  
 PPc Central pulse pressures  
 PPp Peripheral pulse pressures  
 PTH Parathyroid hormone  
 PVD Peripheral vascular disease  
 PWV Pulse wave velocity  
 RAAS Renin angiotensin aldosterone system  
 RLU Relative light units  
 RRT Renal replacement therapy  
 SBP Systolic blood pressure  
 SBP Systolic BP  
 TGF Transforming growth factor  
 TGF- $\beta$  Transforming growth factor beta)  
 TIMP Tissue inhibitors of metalloproteinase  
 TIMP-1 Human Tissue inhibitor of Matrix Metalloproteinase 1  
 TIMP-2 Human Tissue inhibitor of Matrix Metalloproteinase 2

## **PREFACE**

The incidence of cardiovascular disease in chronic kidney disease population has been described as reaching epidemic proportions. Cardiovascular mortality in patients on renal replacement therapy is 10–30 times more common than in the general population. Left ventricular hypertrophy (LVH) and large artery dysfunction are prevalent intermediate cardiovascular changes in patients receiving HD. Volume and pressure related risk factors impact LVH and large artery dysfunction. Measurements of circulating natriuretic peptides and their N-terminal pro-hormones and inferior vena cava diameter (IVCD) have been used recently to aid in assessment of appropriate circulating volume. Studies have suggested that changes in collagen I markers, matrix metalloproteinase 1, 2, 9 and their tissue inhibitors (1 and 2) may predict changes in the cardiovascular system. However, what is not entirely clear is exactly what determines these, and the extent to which each factor contributes toward LVH and large artery dysfunction in HD.