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Original Article

Ankle brachial index as a predictor for cardiovascular risk factors in patients with chronic kidney disease

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ABSTRACT

Objectives: To determine association between chronic kidney disease with abnormal ankle brachial index and cardiovascular risk factors in patients having abnormal ankle brachial index.

Materials and methods: The study was designed as a cross sectional descriptive observational study. All the consecutive patients of CKD with estimated glomerular filtration rate (eGFR) less than 60 ml/min/1.73 m^2 was tested for the ABI. The ABI was calculated by measuring the brachial systolic pressure in both arms and posterior tibial systolic pressure in both legs. The ratio of the systolic pressure in the leg to the higher of the brachial systolic pressure defined the leg specific ABI and lower of leg specific ABI was used for analysis.

Results: The study showed 28% of patients had low ABI less than 0.9 and 4% had high ABI more than 1.4 so cumulative prevalence of abnormal ABI was 32%. The association between abnormal ABI and different stages of CKD was nonlinear and not significant. Among these patients with abnormal ABI there was significant association with left ventricular hypertrophy, calcium–phosphorus product is equal or more than 55 and highly sensitive C reactive proteins (hs-CRP) more than 5 mg/l.

Conclusion: There was high prevalence of abnormal ABI in patients of CKD but the association between different stages of CKD with abnormal ABI was not significant. In patients with abnormal ABI, there were significant association with left ventricular hypertrophy, calcium–phosphorus product $\geq$55 and highly sensitive C reactive protein (hs-CRP) $>$ 5 mg/l. The high hs-CRP denotes inflammation is one of the factor linking CKD and cardiovascular event.

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1. Introduction

Chronic kidney disease (CKD) is a worldwide threat to public health. It is expected that the overall prevalence of CKD is 10.6% in urban areas of Nepal. Three most common causes of end stage renal disease (ESRD) in Nepal are believed to be diabetes mellitus, glomerulonephritis and hypertension.[1]

Chronic kidney disease, affects approximately 13% of adults in the U.S, and is strongly associated with cardiovascular disease events.[2] At each stage of CKD the risk of cardiovascular disease (CVD) mortality is several-fold higher than the risk of progression to end-stage renal disease .[3–5]

Arterial calcification is one mechanism linking CKD and CVD. Arterial calcification is highly prevalent in maintenance dialysis patients. Its presence predicts all-cause mortality. Medial arterial calcification is prevalent in distal arteries of the lower extremities and is correlated with higher pulse-wave velocity, left ventricular hypertrophy, and mortality in maintenance dialysis populations.[6]

Vascular calcification may be caused by either intimal or medial arterial calcification. In atherosclerotic process, calcium is deposited within the tunica intima with lipid-rich plaque causing focal arterial narrowing, medial arterial calcification is limited to tunica media, has a uniform character resembling a ring in vessel cross-section directly contributes to arterial stiffness· The study shows that cardiovascular remodeling and extracellular fluid excess occurred at a very early stage of CKD.[7] The ankle brachial index (**ABI)** is a noninvasive measure of subclinical CVD. It determines the predominant pattern of arterial disease in the lower limbs. A low **ABI** is sensitive and specific for angiographically determined atherosclerosis of the lower extremities and is strongly associated with CVD events.[8]

There is non-linear association between ABI and cardiovascular mortality. Both groups (high and low ABI) were at approximately 2-fold mortality risk compared with subjects with intermediate **ABI** scores.[9] The association between high ABI and mortality was similar to that of low ABI and mortality, highlighting a U-shaped association between this noninvasive measure of peripheral arterial disease and mortality risk.[10]

Inflammation in chronic renal disease may play a central role in the predisposition of patients with chronic renal failure to vascular calcifications. Inflammation is basic component of the process of atherosclerosis and the increased levels of the inflammation markers, especially C – protein, are present in patients with renal failure in predialytic stage. About 30–50% of the patients with chronic renal disease show increased values of the inflammatory markers. The rise of the mediators of inflammation is due to oxidative stress.[11]

The study shows that cardiovascular remodeling and extracellular fluid excess occurred at a very early stage of CKD. The independent association between fluid excess and cardiac and vascular remodeling and hypertrophy may be instrumental in the increased cardiovascular risk in CKD patients. Early therapeutic control of extracellular fluid may reduce cardiovascular events in CKD patients.[12]

Chronic kidney disease (CKD) has been recently recognized as an independent cardiovascular risk factor. The National Health and Nutrition Examination Study II reported a 1.68-fold increase in the risk of cardiovascular death in patients with mild renal failure while the Atherosclerosis Risk in Communities Study indicated a continuum of the effect of renal failure with a 10 ml/min decrease in glomerular filtration rate (GFR) linked to a 7% increase in the risk of *de novo* atherosclerosis and cardiovascular disease.[13,14]

Chronic kidney disease is strongly associated with cardiovascular disease (CVD) events and these associations are not fully explained by traditional CVD risk factors. Recently, nontraditional risk factors for CKD, including inflammation, oxidative stress and metabolism of minerals, have generated more attention.[15]

Peripheral arterial disease (PAD) is more frequent in older adults, with a 1.5- to twofold increase in risk for every 10-year increase in age. Smoking or DM increases the risk of PAD independently by approximately three fold.[16,17]

An ABI of less than 0.9 has not only been established as a reliable diagnostic marker for PAD with high sensitivity and specificity, but also a strong predictor for overall and cardiovascular mortality and overall mortality in advanced CKD patients in hemodialysis patients. Early detection of PAD identifies a group of patients who would benefit from aggressive cardiovascular risk factor modification and from antiplatelet therapy.[18,19]

2. Aims and objectives

- To determine association between chronic kidney disease with high or low ankle brachial index (abnormal ABI).
- To determine the cardiovascular risk factors in chronic kidney disease with high or low ankle brachial index (abnormal ABI).

3. Materials and methods

This Cross sectional descriptive observational study conducted from February 2010 to February 2011 at B.P. Koirala Institute of Health Sciences (BPKIHS), in Eastern Nepal. All the consecutive patients of CKD with estimated glomerular filtration rate (eGFR) <60 ml/min/1.73 m^2 who visited OPD, admitted in the ward under the department of internal medicine fulfilling the inclusion criteria were tested for the ankle brachial index. Estimated GFR (ml/min/1.73 m^2) was calculated using MDRD study equation. Approval for the project was received from the ethical committee of B.P. Koirala Institute of Health Sciences. Informed consent was obtained from each participant before the collection of any data and we sought to design and conduct the project in the line with the Helsinki Declaration & subsequent amendments.

3.1. *Inclusion criteria*

1. All Subjects above the age of 15yrs who were diagnosed to have CKD with GFR less than 60 ml/min/1.73 m^2.
2. Patients giving informed consent.