INTRODUCTION

Hypertension is a disease characterized by end-organ complications, leading to high morbidity and mortality in many cases. People with untreated or uncontrolled hypertension often run the risk of developing complications directly associated with the disease. Left ventricular hypertrophy (LVH) has been shown to be a significant risk factor for adverse outcomes both in patients with hypertension and in the general population. The existence of left ventricular hypertrophy (LVH) identifies patients at particularly high risk for cardiovascular complications in essential hypertension. Hypertension is the fundamental trigger to the sequence of biologic events that lead to the development of LVH. Volume load, inotropy, and arterial compliance also are important determinants of the development and the degree of LVH. Less is known about the relation between in-treatment LVH and risk of cardiovascular events. It is a cardinal manifestation of preclinical cardiovascular disease that strongly predicts cardiovascular events in hypertensive patients as well as in the general population. The prevalence of Echocardiographic left ventricle hypertrophy has been reported to almost 50 percent in patient with mild to be moderate hypertension. Concentric hypertrophy is the form most often found in hypertensive patients. However, a high prevalence of septal hypertrophy has been reported in patients with borderline hypertension. Eccentric or dilated hypertrophy also has been reported to occur in hypertension and usually is associated with a decrease in cardiac function. It is stated that septal thickness is greater than the thickness of the left ventricular posterior wall in hypertensive patients. The high prevalence of hypertrophy in hypertensive patients suggests that blood pressure levels are directly related to the degree of hypertrophy. However, inappropriate left ventricular hypertrophy may occur. It also reflects the increased afterload imposed on the left ventricle, although other important determinants are demographic characteristics (e.g., age, gender, race), neurohumoral and growth factors, and underlying genetic factors because, they are associated with increased plasma volume and cardiac output. Ageing per se increases left ventricular overload due progressive stiffening of central arteries.

A large amount of data, obtained in both animal and human studies, seem to indicate that the degree of LVH correlates with abnormalities in left ventricular function and carries with it an unfavourable clinical prognosis. At some point in the natural history of hypertension, the compensatory increase in LVH is not beneficial anymore. In fact, it becomes a preclinical disease and an independent risk factor for congestive heart failure, ischemic heart disease, arrhythmia, sudden death, and stroke. Echocardiography is now frequently used in the evaluation of the hypertensive patient. It has been shown to be reliable and reproducible technique in the assessment of cardiac anatomy and function. Echocardiographic assessment requires a comprehensive assessment in several imaging planes with careful attention to correct beam alignment in order to minimize errors in the measurement of LV wall thickness and appropriate identification of hypertrophy with an unusual distribution.
We planned this study to; See the degree of left ventricular remodelling in chronic uncontrolled hypertensive patients and to compare their remodelling with the similar in the western part of the world.

MATERIAL AND METHODS
We carried out this study in the Physiology Department of College Medicine, Northern Borders University with Collaboration of Cardiology Department of Central Hospital Ar’ar during 1st Nov 2010 to 31st Dec 2010. In this study we include 50 consecutive patients with the diagnosis of HTN-induced LVH (based on echocardiography findings and ruling out other possible aetiologies of LVH) were evaluated. All patients were newly diagnosed and untreated. We also included 50 age and sex match none obese normotensive and healthy volunteers were included in this study as controls. We excluded the cases with Echocardiographic features or positive family history indicative of hypertrophic cardiomyopathy; and the presence of any other known disease, which was completely or partially responsible for renal dysfunction or retinal abnormality (such as diabetes mellitus and genetic or autoimmune diseases affecting retinal or renal integration).

The blood pressure (BP) was measured with an arm cuff and a mercury sphygmomanometer after the patient had been resting in a sitting position for five minutes. Systolic and diastolic BP measurements were taken as the first and fifth phase of the Korotkoff sounds, respectively. According to American Heart association classification of the hypertension (HTN) is defined as a systolic BP (SBP) ≥135 mmHg and/or diastolic BP (DBP) ≥85 mmHg on two measurements taken with a one-day interval. The mean arterial pressure (MAP) was measured by the formula

\[
MAP = \frac{1}{3} \text{SBP} + \frac{2}{3} \text{DBP}
\]

M-mode, two-dimensional echocardiographic examinations were performed for all the subjects in the partial left decubitus position, using a Philips machine and a 2.5–3.5 MHz electrical transducer. End-diastolic left ventricular internal diameter (LVIDd), septal wall thickness (IVST) and posterior wall thickness (PWTd) were calculated from the two-dimensionally-guided M-mode tracings and measured in five consecutive cardiac cycles, according to the Penn Convention.

RESULTS
The results of the study are summarised in Table-1 and 2.

**Table-1: Comparison of means of demographic values of patients and healthy controls**

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Patients (Mean±SD)</th>
<th>Controls (Mean±SD)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (Years)</td>
<td>50</td>
<td>52.48±13.38</td>
<td>50.92±13.38</td>
<td>&lt;0.05***</td>
</tr>
<tr>
<td>Body Mass Index (Kg/m²)</td>
<td>50</td>
<td>28.66±5.71</td>
<td>26.3±3.76</td>
<td>&lt;0.05***</td>
</tr>
<tr>
<td>Mean Arterial Pressure (mmHg)</td>
<td>50</td>
<td>111.62±7.76</td>
<td>89.04±5.19</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>50</td>
<td>156.22±14.14</td>
<td>18.06±8.29</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>50</td>
<td>88.96±6.23</td>
<td>74.54±6.31</td>
<td>&lt;0.001**</td>
</tr>
</tbody>
</table>

*Highly Significant, **Significant, ***Non-significant

**DISCUSSION**

The present study showed a positive correlation between left ventricular remodelling and Hypertension. This important correlation can be due to a long duration of undiagnosed HTN (even though all the patients were newly diagnosed). This possible explanation of the study findings should be kept in mind in future investigations.

Left ventricular remodelling is one of the most serious complications of HTN, and it has been strongly associated with an increased incidence of heart failure, coronary artery disease, myocardial infarction, cardiac arrhythmias and sudden death. Based on these facts, its early detection is mandatory in order to obtain left ventricular remodelling regression, using certain adequate antihypertensive drugs. Some investigators concluded that diastolic BP was not a reliable parameter in association to left ventricular remodelling and others suggested that although the office or home BP did not have a remarkable correlation with LV structural indices.
The results from the current research were consistent with the majority of the previous studies that showed positive associations between the Mean arterial pressure, Systolic and Diastolic BPs and PWTd, IVSTD and LVIDd in hypertensive patients (Table-1, 2). Our investigation confirmed that the more severe and uncontrolled the BP, the more severe the PWTd, IVSTD and LVIDd in hypertensive patients.

CONCLUSION
The degree of left ventricular remodelling in chronic uncontrolled hypertensive patients depends largely upon how much hypertension is control. While comparing our study it is almost in the agreement of western studies. For the time being we could not find/establish any relationship of Left ventricular remodelling with BMI. Therefore it is suggested that further specific study may be conducted with different parameters to find this relationship.

REFERENCES

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