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# "A Study of the Cardiovascular Risk Factors and Cardiovascular Complications in Systemic Hypertension".

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***ABSTRACT:***

***Aims and Objectives:***

***1. To know the clinical profile of the patients presenting with systemic hypertension.***

***2. To evaluate the patients for cardiovascular risk factors.***

***3. To study the prevalence of cardiovascular risk factors among the studied group.***

***4. To study the chest roentgenogram, electrocardiogram and echocardiographic finding of the same patients.***

***Design: Cross – sectional prospective study of 80 patients with Systemic Hypertension.***

***Setting and Method: The Study was done at Osh Regional Integrated Clinical Hospital and Osh City Territorial Clinical Hospital of The Kyrgyz Republic. Eighty patients fulfilling the criteria of high blood pressure on repeated measurement on two different occasions were enrolled in the study. They underwent routine and special investigations to look for cardiovascular risk factors and cardiovascular complications which included laboratory studies, chest x-ray electrocardiogram and echocardiography.***

***Conclusion: Hypertension is a major cardiovascular risk factor. Cardiovascular complications were significantly increased in the presence of other cardiovascular risk factors compared to patients without risk factors. Most common major clinical cardiovascular complication remains Left Ventricular Hypertrophy in uncontrolled hypertension and majority of patients had presented with angina or myocardial infarction and the other major CVS complication being heart failure.***

## INTRODUCTION:

An elevated arterial pressure remains one of the most important public health problems of developed countries and is rapidly becoming so in the developing countries much to the dismay of already burdened health budgets. It is common, asymptomatic, readily detectable, usually easily treatable, and often leads to lethal complications if left untreated or inadequately treated. In the representative sample of the U.S. population examined in the 1991-1994 National Health and Nutrition Examination Survey (NHANES III), only 27 percent of hypertensive had their blood pressure well controlled, as defined by a reading below 140/90 mm Hg.[1] Despite having been diagnosed only about half of the hypertensive are being treated. Moreover, cardiovascular mortality remains higher even in presumably well treated hypertensive than in nonhypertensives.[2] Clearly, more attention is being directed toward hypertension, but adequate hypertension control remains elusive in large part because of the asymptomatic nature of the disease for the first 15 to 20 years, even as it progressively damages the cardiovascular system.[3]

In view of these built barriers to effective control of the individual patient, population wise application of preventive measures becomes inherently more attractive. Although the specific mechanisms for most


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hypertension remain unknown, it is highly likely that the process could be slowed, if not prevented, by the prevention of obesity, moderate reduction in sodium intake, higher levels of physical activity, and avoidance of excessive alcohol consumption.[3]

Though understanding of the path physiology of raised arterial pressure has increased, in 90% to 95% of cases the etiology (and thus potentially the means of prevention or cure) is still largely unknown. Prevalence of other factors in hypertensive populations which increase the risk of having cardiovascular, cerebrovascular, renal, and peripheral vascular adverse events, independent of presence of high blood pressure, further escalates the chance of having target organ damage in such population. These factors have been defined in the Sixth report of Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI)[1] as Smoking, Dyslipidemia, diabetes Mellitus, Age more than sixty years, Male gender and Postmenopausal women, and a family history of cardiovascular disease before the age of fifty five in men and sixty five in women.

Target organ Damage or Clinical Cardiovascular Diseases has been defined as having Heart diseases (Left ventricular hypertrophy, angina, prior myocardial infarction, prior CABG, heart failure), Stroke or Transient Ischaemic Attack, Nephropathy, Peripheral Arterial Disease and Hypertensive Retinopathy.

Evidence of end organ damage is a risk factor for poor prognosis in hypertension. Patients with heart disease die prematurely and the most common cause of death is heart disease, with stroke and renal failure also frequent particularly in patients with significant retinopathy.

**Definition and Classification of Hypertension[4]:**

Blood pressure is a continuous variable, and whatever number is used to define hypertension will be arbitrary. Sixth Joint National Committee on Prevention, Detection, Evaluation Treatment of High Blood Pressure (JNC VI), and the British Hypertension Society of Hypertension (WHO/ISH), and the British Hypertension Society (BHS) use a more comprehensive system to define Hypertension[1,5,6] ( Table 1 ). These definitions are based on properly measured office readings.

**Table 1: Classification Of Blood Pressure For Adults Aged 18 Years And Older[1, 5]:**

| Category | Blood Pressure (mm Hg) | | |
|---|---|---|---|
| | **Systolic** | | **Diastolic** |
| **Optimal** | <120 | And | <80 |
| **Normal** | <130 | And | <85 |
| **High-normal** | 130-139 | And | 85-89 |
| **Hypertension** | | | |
| **Stage 1** | 140-159 | And | 90-99 |
| **Stage 2** | 160-179 | And | 100-109 |
| **Stage 3** | ≥ 180 | And | N ≥ 110 |

**Epidemiology and Risk[4] :**


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Physicians generally do not concern themselves with reducing BP when it is elevated because of the specific clinical problems they can attribute to that elevation. Instead, hypertension is treated because of the increased risk of mortality and cardiovascular disease that results from having an elevated BP[8-11].

These risks have been well documented in numerous epidemiologic, studies, beginning with the Framingham Heart Study and many others in the 1950s and 1960s and extending to the present .[8, 12-17] More recently, meta-analyses of pooled data have confirmed the robust, continuous relationship between BP level and cerebrovascular disease and coronary artery disease (CVD) in both western and eastern populations.[18,19] In addition, BP is directly related to left ventricular hypertrophy (LVH) and heart failure (HF), Peripheral vascular disease (PVD) carotid atherosclerosis, renal disease, and "sub clinical Daises."[10,11,20,21] Kannel and colleagues in the Framingham Heart Study have documented the fact that CV risk factors tend to cluster in hypertensives.[22] Hypertensive are more likely to have dyslipidemias, especially elevated serum triglyceride levels and low levels of high-density lipoprotein cholesterol (HDL-C), and type 2 DM. The common denominator may be insulin resistance, perhaps as a result of the frequent association of hypertension and obesity.[23] Systolic blood pressure has been recognized with increasing clarity in those over age 50 or 60 years and has given equal weight since the Fifth report of the Joint National Committee, Evaluation and Treatment of Hypertension.[24, 25, 26]

Pulse pressure, the difference between systolic and diastolic blood pressure, is and even better predictor risk than is systolic BP in most of the epidemiologic studies done to date.[27-32] A wide pulse pressure, unless it is a result of aortic insufficiency or an arteriovenous malformation, is a simple clinical indicator of stiffer and less compliant large central arteries and significant arterial damage. Pulse pressure measurements have not been included in classification systems in defining the risks of hypertension or in recommending treatment. Franklin and colleagues, again using data from the Framingham Heart Study cohort, showed that at all levels of systolic BP (even as low as 110 to 130 mmHg), risk is less with higher diastolic BPs.[32]

Hypertension may not be the asymptomatic condition it has long been thought to be. Different trials utilizing a wide variety of drugs to reduce BP have shown not only that lowering BP is safe but that hypertensive treated to lower levels feel better. In the Treatment of Mild Hypertension Study (TOMHS) and the Hypertension Optimum Treatment (HOT) trial, the group with the lowest BP had the fewest complaints.[33,34]

**Table 2: Components of Cardiovascular Risk Stratification in Patients with Hypertension and Target Organ Damage:**

| **Major Risk Factors** |
|---|
| Smoking |
| Dyslipidemia and Obesity |
| Diabetes mellitus |
| Age>60yr |
| Sex (men and postmenopausal women) |
| Family history of cardiovascular disease: |
| Women <65 yr or men >55 yr |
| Alcohol Consumption |
| **Target Organ Damage/Clinical Cardiovascular Disease** |
| Heart disease |
| -Left ventricular hypertrophy |


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| |
|---|
| -Angina or prior myocardial infarction<br>-Prior coronary revascularization<br>-Heart failure<br>Stroke or Transient Ischemic Attack<br>Nephropathy<br>Peripheral arterial disease<br>Retinopathy |
| *From The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. Arch Intern Med 157:2413. 1997 Copyright 1997, American Medical Association.* |

**Table 3: Risk Stratification and Treatment:**

| **Blood Pressure Stages (mmHg)** | **Risk Group A (No Risk Factors / No TOD, CCD)** | **Risk Group B (At Least 1 Risk Factor, Not Including DM / No TOD/CCD)** | **Risk Group C (TOD/CCD and / or DM, with or without Other Risk Factors** |
|---|---|---|---|
| **High normal (130-139/85-89)** | Life style modification | Life style modification | Drug therapy |
| **Stage 1 (140-159/90-99)** | Life style modification (up to 12 months) | Life style modification (up to 6 months) | Drug therapy |
| **Stages 2 and 3 (>160/>100)** | Drug therapy | Drug therapy | Drug therapy |

CCD=clinical cardiovascular disease; TOD=target organ disease. From The Sixth Report of the joint National Committee on Prevention, Detection Evaluation and Treatment of High Blood Pressure. Arch Intern Med 157:2413.1997 Copyright 1997. American Medical Association.

**Overall Cardiovascular Risk:**

The Framingham Study and other epidemiological surveys have clearly defined certain risk factors for premature cardiovascular disease addition to hypertension. For varying levels of blood pressure, the Framingham data (available in the Coronary and Stroke Risk Handbooks published by the American Heart Association) show the increasing likelihood of a vascular event over the next 10 years for both men and women at various ages as more and more risk factors are added.[36] For example, a 55 years-old man with a systolic blood pressure of 160 mm Hg who is otherwise at low risk would have a 13.7 percent chance of a vascular event in the next 10 years. A man of the same age with the same pressure but with all the additional risk factors (elevated serum total cholesterol, low high-density lipoprotein cholesterol, cigarette smoking, glucose intolerance, and LVH on the electrocardiogram) has a 59.5 percent chance. Obviously, the higher the overall risk, the more intensive the interventions should be. An interesting-and disturbing- connection between untreated hypertension and *hypercholesterolemia* has been noted in multiple populations.[46] This connection may be mediated through insulin resistance and hyperinsulinemia and anticipated in those with upper body obesity [47] but may also be found in nonobese hypertensives. Clearly, through this association, hypertensives are often burdened with an even greater risk than that imposed by their blood pressure alone.


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Gregory YH Lip and colleagues investigated atherogenic lipoprotein profile and endothelial dysfunction to cardiovascular risk by measuring LDL sub fractions and flow mediated dilation (FMD) reflecting endothelial dysfunction in a cohort of high-risk hypertensive patients. They studied 84 hypertensive patients (74 men; mean age, 64 years; SD 8). Mean LDL score was higher and FMD impaired in hypertensive subjects compared with control subjects. These indexes were significantly improved after 6 months of cardiovascular risk factor management. LDL score correlated significantly with the 10-year Framingham coronary heart disease risk score, with a negative correlation with FMD (both $p<0.001$). Abnormal parthenogenesis and endothelial dysfunction are both present in hypertension and appear to be related to each other, potentially leading to vascular complications. The abnormal LDL scores also correlate with the 10-year cardiovascular risk and can be positively influenced by cardiovascular risk management. [48]

The prevalence of sub clinical atherosclerosis and cardiovascular was evaluated by Kuller L and colleagues among the 5,201 adults aged > or =65 years in four communities participating in the Cardiovascular Health Study from June 1989 through May 1990. A combined index based on electrocardiogram abnormalities, carotid artery wall thickness and stenosis based on carotid ultrasound, decreased ankle-brachial blood pressure, and positive response to a Rose Questionnaire for angina or intermittent claudicating defined sub clinical disease. The prevalence of sub clinical disease was 36% in women and 38.7% in men and increased with age. Among women, low-density lipoprotein cholesterol, blood glucose, and cigarette smoking were positively associated, and high-density lipoprotein cholesterol negatively associated, with sub clinical disease. In men, systolic blood pressure, blood glucose, and cigarette smoking were independent risk factors in multiple logistic regression analysis. The risk factors for sub clinical disease are, therefore, similar to those for clinical disease at younger ages, especially among women. It is possible that older individuals with sub clinical disease are at very high risk of developing clinical disease and that more aggressive interventions to prevent clinical disease should be oriented to individuals with sub clinical disease.[49]

The central arteries stiffen with age, causing hemodynamic alterations have been associated with cardiovascular events. Changes in body fat with age may be related to aortic stiffening. The association between vascular stiffness and body fat was evaluated in 2488 older adults (mean age, 74 years; 52% female; 40% black) enrolled in the Study of Health, Aging, and Body Composition (Health ABC)., a prospective study of changes in weight and body composition. Clinical sites were located in Pittsburgh, Pa, and Memphis, Tenn. Aortic pulse wave velocity was used as an indirect measure of aortic stiffness. A faster pulse wave velocity indicates a stiffer aorta. Body fat measures were evaluated with dual energy x-ray absortiometry and computed tomography. Independent of age and blood pressure, pulse wave velocity was positively associated with weight, abdominal circumference, abdominal subcutaneous fat, abdominal visceral fat, thigh fat area, and total fat ($p<0.001$ for all). The strongest association was with abdominal visceral fat. Elevated pulse wave velocity was also positively associated with history of diabetes and higher levels of glucose, insulin, and hemoglobin A1c ($P<0.001$ for all). In multivariate analysis, independent positive associations with pulse wave velocity were found for age, systolic blood pressure, heart rate, abdominal visceral fat, smoking, HbAlc, and history of hypertension. The association between pulse wave velocity and abdominal visceral fat was consistent across turtles of body weight. Among older adults, higher levels of visceral fat are associated with greater aortic stiffness as measured by pulse wave velocity.[50] Excess body weight is associated with higher aortic stiffness in whites and African Americans as young as 20 to 30 years. The strength of the association, the early age at which it appears, and the prevalence of


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obesity among the young warm of substantially increased cardiovascular disease incidence as this cohort ages.[51]

J Pekkanen and Colleagues studied the associations of total, low-density lipoprotein (LDL), and high-density lipoprotein (HDL) cholesterol with mortality from coronary heart disease and cardiovascular disease. They studied 2541 white men who were 40 to 69 years old at base line and followed them for an average of 10.1 years. Seventeen percent had some manifestation of cardiovascular disease at base line, whereas the others did not. Among the men who had cardiovascular disease at base line, we found, after multivariate adjustment, that those with "high" blood cholesterol levels (above 6.10 mmol per liter) had a risk of death from cardiovascular disease, including coronary heart disease, that was 3.45 times higher (95 percent confidence interval, 1.63 to 1.33) for LDL cholesterol levels above 4.13 mmol per liter as compared with those below 3.35 mmol per liter, and 6.02 (95 percent confidence interval, 2.73 to 13.28) for HDL cholesterol levels below 0.90 mmol per liter as compared with those above 1.16 mmol per liter. All three lipid levels were significant predictors of death from coronary heart disease alone (P less than 0.005). Total cholesterol and LDL cholesterol levels were also significant predictors of death form cardiovascular and coronary heart disease in men without preexisting cardiovascular disease, although at a lower level of absolute risk of death. Thus, the 10-year risk of death from cardiovascular disease for a man with preexisting cardiovascular disease increased from 3.8 percent to almost 19.6 percent with increasing levels of total cholesterol from "desirable" to "high," whereas the corresponding risk for a man who was free of cardiovascular disease at base line increased from 1.7 percent. Our findings suggest that total, LDL, and HDL cholesterol levels predict subsequent mortality in men 40 to 69 years of age, especially those with preexisting cardiovascular deisease.[52]

A report by Kannel WB and McGee DL examines prospectively, in the Framingham cohort, the relation of diabetes and impaired glucose tolerance to each of the cardiovascular sequelae, taking into account age, sex, and associated cardiovascular risk factors. The incidence of cardiovascular disease, as well as the levels of cardiovascular Risk factors were found to be higher in diabetic than in non-diabetic men and women. The relative impact is greater for women. Present evidence suggests that alleviation of associated cardiovascular risk factors is the most promising course in reducing cardiovascular squeal in diabetic patients.[53]

An article by Kannel WB and colleagues presents prediction equation for several cardiovascular disease endpoints, which are based on measurements of several known risk factors. Subjects (n = 5573) were original and offspring subjects in the Framingham Heart Study, aged 30 to 74 years, and initially free of cardiovascular disease. Equations to predict risk for the following were developed: myocardial infarction, coronary heart disease (CHD), death from CHD, stroke, cardiovascular disease, and death from cardiovascular disease. The equations demonstrated the potential importance of controlling multiple risk factors (blood pressure, total cholesterol, high-density lipoprotein cholesterol, smoking, glucose intolerance, and left ventricular hypertrophy) as opposed to focusing on one single risk factor.[54]

As many as 30% of all coronary heart disease (CHD) deaths in the United States each year are attributable to cigarette smoking, with the risk being strongly dose-related.[55] [56] Smoking also nearly doubles the risk of ischemic stroke.[57] Smoking acts synergistically with other risk factors, substantially increasing the risk of CHD.[58] Smokers are also at increased risk for peripheral vascular disease, cancer, chronic lung disease, and many other chronic diseases. Cigarette smoking is the single most alterable risk factor contributing to premature morbidity and mortality in the United States, accounting for approximately 430000 deaths annually.[59]


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In patients with established coronary atherosclerosis, BMI, as well as CRP and number of coronary lesions, are independently associated with acute coronary syndromes. There is evidence of increased risk event at mildly elevated BMI levels.[60]

The results documented from a study done by Gerald BP and colleagues to determine whether any sex hormone abnormality might be a factor in the development of myocardial infarction in women, raise the possibility that in women an elevated FT level may be a risk factor for coronary atherosclerosis.[61] Hak AE et al found and independent inverse association between levels of testosterone and aortic atherosclerosis in men.[62]

**A Synthesis of Risk:**

As first clearly articulated by a group of New Zealand physicians, [63] the degree of risk from hypertension can be categorized with reasonable accuracy by taking into account (1) the level of blood pressure, (2) the biological nature of the hypertension based on the degree of target organ damage, and (3) the coexistence of other risks. The JNC-6 report provides a stratification of risk into three group based on known components or risk (Table 2) and levels of blood pressure, which are in turn, used as the basis for deciding upon initial treatment (Table 3). According to this stratification, active drug therapy is recommended for high-risk patients even if blood pressure is only high normal whereas life style modifications are recommended for low-risk patients even if blood pressure is as high as 159/99 mm Hg.

**Complications of Hypertension:**

The higher the level of blood pressure, the more likely that various cardiovascular disease will develop prematurely through acceleration of atherosclerosis, the pathological hallmark of uncontrolled hypertension. If untreated, about 50% of hypertensive patients die of coronary heart disease or congestive failure, about 33 percent of stroke, and 10 to 15 percent of renal failure. Those with rapidly accelerating hypertension die more frequently of renal failure, as do those who are diabetic. Death is usually attributed to stroke or myocardial infraction instead of to the hypertension that was largely responsible.

In general, the vascular complications of hypertension can be considered as either "hypertensive" or atherosclerotic" (Table 4). The former are more directly caused by the increased blood pressure per se and can be prevented by lowering this level; the latter have more multiple causations. Although hypertension may represent the most significant of the known risk factors for atherosclerosis in quantitative terms, lowering blood pressure may not by itself halt the atherosclerotic process.

The path from hypertension to vascular disease probably involves three interrelated processes: pulsetile flow, endothelial cell dysfunction, and smooth muscle cell hypertrophy. Higher systolic pressures are probably more responsible for these changes than are lower diastolic levels, which provides an explanation for the closer approximation of cardio vascular risk to systolic pressure and pulse pressure. These three interrelated processes are probably responsible for the arteriolar and arterial sclerosis that is the usual consequence of longstanding hypertension. The subsequent target organ damage (see below) should be included in the overall assessment of hypertension risk. Beyond damage to the eyes, heart, brain, and kidney, large vessels such as the aorta may be directly affected and be at risk for aneurysms dissection.

**Target Organ Damage:**

The biological aggressiveness of a given level of hypertension varies among individuals. This inherent propensity to induce vascular damage can best be ascertained by examination of the eyes, heart, and kidney.


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**Funduscopic Examination:**

As described by Keith and associates in 1939, vascular changes in the fundus reflect both hypertensive retinopathy and arteriosclerotic retinopathy.[64] The two processes first induce narrowing of the arteriolar lumen (grade 1) and then sclerosis of the adventitia and/or thickening of the arteriolar wall, visible as arteriovenous nicking (grade 2) . Progressive hypertension induces rupture of small vessels, seen as hemorrhage and exudates (grade 3) and
eventually papilledema (grade 4). The grade 3 and 4 changes are clearly indicative of an accelerated-malignant form of hypertension, whereas the lesser changes have been correlated with other evidence of target organ damage. [65]

**Table 4:** Vascular Complications of Hypertension:

| **Hypertensive** | **Atherosclerotic** |
|---|---|
| Accelerated-malignant phase | Coronary heart disease |
| Hemorrhagic stroke | Sudden death |
| Congestive heart failure | Other arrhythmias |
| Atherosclerosis | Atherothrombotic stroke |
| Aortic dissection | Peripheral vascular disease |
| *Adapted from Smith WM: Treatment of mild hypertension. Results of a ten-year intervention trial. Circ Res 25 (Suppl 1):98, 1977. By permission of the American Heart Association* | |

**Cardiac Involvement:**

Hypertension places increased tension on the left ventricular myocardium that is manifested as stiffness and hypertrophy, which accelerates the development of atherosclerosis within the coronary vessels. The combination of increased demand and lessened supply increases the likelihood of myocardial ischemia and thereby leads to a higher incidence of myocardial infraction, sudden death, arrhythmias, and congestive failure in hypertensive.

**Abnormalities in Left Ventricular Function:**

Even before LVH develops, changes in both systolic and diastolic function may be seen. Those with minimally increased left ventricular muscle mass may have supernormal contractility as reflected by an increased inotropic state with a high percentage of fractional shortening and increased wall stress. [66] The earliest functional cardiac changes in hypertension are left ventricular diastolic dysfunction, with prolongation and incoordination of isovolumic relaxation, a reduced rate of rapid filling, probably caused by increased passive stiffness. 67
With increasing hemodynamic load either systolic or diastolic dysfunction may evolve and progress to different forms of congestive heart failure[68]. In addition, impaired coronary flow reserve and thallium perfusion defects may be observed in hypertensives without obstructive coronary disease. 69

**Left Ventricular Hypertrophy:**


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Hypertrophy as a response to the increased after load associated with elevated systemic vascular resistance can be viewed as necessary and protective up to a certain point. Beyond that point, a variety of dysfunctions accompany LVH.

In the past, LVH was recognized on electrocardiography by increased voltage of QRS complexes, intrinsicoid deflection over lead $V_2$ or $V_1$ greater than 0.06 seconds, and ST segment depression greater than 0.5 mm. increasingly, echocardiography is being used because it is much more sensitive in recognizing early cardiac involvement. By echocardiography, left ventricular mass is shown to progressively increase with increases in blood pressure.[70] Left ventricular mass is greater in those whose pressure does not fall during sleep because of a more persistent pressure load [71]

The pathogenesis of LVH involves a number of variables other than the pressure load, one of which is hemodynamic volume load. Devereux and colleagues [71] found a closer correlation between left ventricular stroke volume and left ventricular mass with diastolic than with systolic blood pressure. Other determinate are obesity, [72] levels of sympathetic nervous system and rennin-angiotensin activity, and whole blood viscosity, presumably by way of its influence on peripheral resistance. The correlation is much closer between LVH and pressure readings taken during the stress of work by ambulatory monitoring than between LVH and casual pressure readings.[73]

Different patterns of hypertrophy may evolve, often starting with asymmetrical left ventricular remodeling from isolated septal thickening, which has been noted in 22 percent of untreated hyprtensives with normal total left ventricular mass.[74] The pattern of LVH may have important prognostic implications. In a 10-year follow-up of 253 hypertensives, all-cause mortally was higher and cardiovascular events were most frequent in those with *concentric* LVH. [75] The degree of increased muscle mass is a strong and independent risk factor for cardiac mortality over and above the extent of coronary artery disease. [70] In addition, the risk of ventricular arrhythmias is increased at least twofold in the presence of LVH.[76] Echocardiographically determined left ventricular mass and geometry stratify risk in patients with essential hypertension independently of and more strongly than blood pressure or other potentially reversible risk factors and may help to stratify the need for intensive treatment.[77] LV hypertrophy is defined by arbitrary criteria, such as posterior wall thickness exceeding 1.1 cm, or better LV mass indeed exceeding 131 $g/m^2$ in men (110 $g/m^2$ in women). LV hypertrophy represents a remodeling process of the heart architecture to normalize wall stress. The particular pattern of hypertrophy is dependent on the type of load that is imposed on the LV. Increased after load leads to an increase in end-systolic and peak wall stress and the addition of sarcomas in parallel to an increase in LV wall thickness at the expense of chamber volume thus increasing relative wall thickness. [78] This pattern has been termed concentric remodeling' and LV mass is above the upper normal limit concentric hypertrophy. However, some hypertensive patients, especially those with concomitant volume overload states such as obesity or patent arteriole-venous fistula, develop eccentric hypertrophy, characterized by an increased left ventricular mass but normal relative wall thickness.[79] In contrast to physiologic hypertrophy' as encountered in athletes, pathologic forms of LV hypertrophy are accompanied by interstitial fibrosis. Due to decreased relaxation in early diastole, an impaired LV filling is the diagnostic criteria that favors the diagnosis pathologic' LV hypertrophy (decreased E: A ratio, i.e. the ratio of early to late diastolic filling), in contrast to a normal or even supernormal LV diastolic filling in physiologic' LV hypertrophy.

Since the presence of LVH may connote a number of deleterious effects of hypertension on cardiac function, a great deal of effort has been expended in showing that treatment of hypertension will cause LVH to regress. Treatment with all antihypertensive drugs except those that further activate sympathetic nervous system activity, e.g. direct vasodilators such as hydrolyzing when used alone, has been shown to cause LVH


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regression. [80] With regression, left ventricular function usually improves and cardiovascular morbidity decreases.[81]

**Features of Coronary Artery Disease:**

Hypertension is a major risk factor for myocardial ischemia and infarction. Moreover, in the Framingham cohort, the prevalence of silent myocardial infarction was significantly increased in hypertensive subjects, and they were also more susceptible to silent ischemia and
sudden death, [82] as well as having a greater risk for mortality after an initial myocardial infarction. [83] Beyond these multiple additional risks associated with hypertension, a higher incidence of cardiovascular mortality has also been recognized when elevated diastolic blood pressures are reduced to levels below 80 mm Hg.[84] This J- shaped curve probably reflects a reduction in perfusion pressure through coronary vessels either narrowed or having impaired vasodilator reserve in the presence of hypertrophied myocardium.

**Renal Function:**

Renal dysfunction too subtle to be recognized, may be responsible for the development of most cases of essential hypertension. Increased renal retention of salt and water may be a mechanism initiating primary hypertension, but the retention is so small that it escapes detection. Microalbuminuria in hypertensive has been correlated with both insulin resistances [85] and evidence of endothelial cell dysfunction.[86] As hypertension-induced nephrosclerosis proceeds, the plasma creatinine level begins to rise, and eventually, renal insufficiency with uremia may develop, thus making hypertension a leading cause of end-stage renal disease (ESRD), particularly in blacks, [87]

**Cerebral Involvement:**

Hypertension may accelerate cognitive deckling with age.[88] Hypertension, particularly systolic, is a major risk factor for initial and recurrent stroke and for transient ischemic attacks caused by extra cranial athrosclerosis.[89] Usually with, but sometimes without, hypertension, increasing left ventricular mass on echocardiography is associated with a progressively higher risk for stroke. [90] LVH and abnormal LV geometry are independently associated with increased stroke risk. LVH is strongly associated with ischemic stroke in all age, sex, and race-ethnic subgroups. Increased LV relative wall thickness imparts an increased stroke risk after adjustment for LV mass and is of additional value in stroke risk prediction.[91]
Blood pressure usually rises further during the acute phases of a stroke, and caution is advised in lowering blood pressure during this crucial period. On the basis of the aforementioned assessments of overall cardiovascular risk and severity of hypertension, it should be possible to determine the approximate risk status and prognosis for individual patients, which can most easily be accomplished with the Framingham data.

## 3) PATIENTS AND METHOD:

**Patients:**

Eighty patients fulfilling the criteria of high blood pressure on repeated measurement on two different occasions were enrolled in the study. The patients attending the Medical and Emergency Department of Osh Regional Integrated Clinical Hospital and Osh City Territorial Clinical Hospital were included in the study from September 2006 to August 2007. They include both in-patients and out-patients of Medical


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Department. The patients who presented initially in Emergency Department and got discharged were later followed up in Medical Out-Patient Department and who got admitted was followed up in the wards.

**Inclusion Criteria:**

* Patient should be above the age of 18 years.

* Patient may be a newly or previously diagnosed case of Hypertension (fulfilling the criteria for High Blood Pressure defined by JNC VI[1]).
* Patients who were previously diagnosed as hypertensive and taking treatment but not having adequate control of blood pressure.
* Patients presenting with complications attributable to High Blood Pressure.

**Exclusion Criteria:**

* Patients who were critically ill.

**Methods:**

All patients underwent a through evaluation including history, clinical examination, and laboratory investigations, chest x-ray, electrocardiography and echocardiography and it was according to the PROFORMA prepared for the evaluation of cases enrolled in this study.
History included all the present and medical illnesses with evaluation of past records and included family, personal and social history.

A detailed clinical examination was done with emphasis search for features suggestive of hypertensive complications and presence of cardiovascular risk factors. Blood pressure was taken according to guidelines described by JNC VI and was taken 3 times in each sitting on two different occasions.

Routine and specific laboratory tests were carried out using standard laboratory protocol and lipid profile was done after 6 weeks of acute event (on follow up). A posterior-anterior (PA view) chest x-ray was done to look for specifically Cardiomegaly (criteria: diameter of cardiac shadow more that half the diameter of thorax) and other features which could be sequel due to hypertension and its related complications.

**Electrocardiogram:**

Standard 12-lead ECGs were recorded at 25 mm/s and 1 mV/cm standardization with equipment. ECG criteria for the detection of LVH included QRS duration, Sokolow-Lyon voltage, sex-specific Cornell voltage (sum of the amplitude of the R wave in lead AVL and the amplitude of the S wave in lead $V_3$ adjusted by the addition of 800 µV in women).

**Echocardiography:**

All subjects underwent standard M-mode and two-dimensional echocardiography. All left ventricular dimensions were taken, LV ejection fraction measured, presence or absence of LV diastolic dysfunction and LV systolic dysfunction was noted and LV mass was calculated according to formula suggested by Devereux and Reicheck in 1977;
LV mass(g) = $1.04\ [(LVEDD+IVS+LVPWD)^3 - LVEDD^3] -14$


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And this was corrected for body surface area (which gives LV Mass Index). Values of 294 g and 198g were taken as LV mass for male and female respectively. Values of 131g/$m^2$ and 110 gm/$m^2$ were taken as LV Mass Index for male and female respectively.

## OBSERVATIONS AND DISCUSSION:

There were eighty (80) patients enrolled in the study. Forty-four (44) were male patients (55%) and thirty-six were female patients (45%) (Table 4-1). The mean age was 59.15 years and mean age range was 59+9.66 years. The youngest patient was of age 38 years and eldest was 80 years (Table 4-11). Among men 41 % were above 60 years of age and 42 % of women were above 60 years of age (Table 4-3).

The systolic blood pressure (SBP) was in the range of 130mmHg to 208mmHg. The mean SBP was 155.75mmHg and mean range was 155.75 +,- 15.88. The diastolic blood pressure (DBP) was in the range of 84 mmHg and to 134mmHg. The mean DBP was 100.78mmHg and mean range was 100.78 +,- 10.42.

Considering the risk factors (defined in JNC VI[1]) among the studied population (Table 4-3)(Figs 1& 2), smoking was highly prevalent with 70.45 % of men being smokers and 64 % of women being smokers. Diabetes mellitus was seen in 27.27 % of men and 19.44 % of women. Dyslipidemia was seen in the studied group in 22.72 % of men and 36.11 % of women. Among women 75 % were in postmenopausal age group. Alcohol consumption and Obesity are the another key risk factors among the studied population (Table 4.3) and (figs 1 and 2), being 75 % of men and 19.4 % of women alcohol consumers. Obesity was seen in 27.27 % of men and 33.33 % of women in the studied group.

Primary clinical presentation was attributable to cardiovascular disease in 38 cases (47.5%), cerebrovascular disease in 16 cases (20%), renal disease in 3 cases (3.8%), peripheral arterial disease in only one case (1.3%) and 22 cases (27.50%) were incidentally diagnosed when they consulted for non-hypertensive related conditions (Table 4-9) (Fig 3).

Clinical cardiovascular disease profile from the cohort was such that 50 (62.5%) cases had left ventricular hypertrophy, 20 (25%) cases had angina or myocardial infarction, none had prior coronary revascularization and 10 (12.5%) cases had heart failure (Table 4-10) (Fig 4).

Thirty-six percent cases were newly diagnosed cases. The mean duration of known hypertension was 2.99 years with a standard deviation of 3.20 (Table 4-13 and 4-12). There was significant cardiomegaly in chest x-ray between stage 1 hypertension and stage 2 hypertension ($p= 0.018$) and between stage 1 hypertension and stage 3 hypertension ($p=0.001$). No significance was found between stage 2 and 3 hypertension ($p=0.625$) (Table 4-14).

Left ventricular hypertrophy in electrocardiogram was significant between stage 1 hypertension and stage 2 hypertension ($p=0.012$) and highly significant between stage 1 and stage 3 ($p<0.001$). Again no significance was found between stage 2 and stage 3 hypertension ($p=0.135$) (Table 4-15).

Echocardiographic evaluation for left ventricular hypertrophy by measuring LV mass and LV mass index showed significant results between stage 1 and stage 2 hypertension ($p=0.016$ and $p=0.035$ respectively) and as well between stage 1 and stage 3 hypertension ($p<0.001$ in both). There was significance in the LV mass and LV mass index between stage 2 and stage 3 hypertension as well ($p=0.004$ and $p=0.003$ respectively) (Tables 4-24 and 4-26). Low LV ejection fraction was found in 20 cases and was found to be significant between stage l and stage 2 hypertension ($p=0.014$) and highly significant between stage 1 and stage 3 ($p<0.001$) but was insignificant between stage 2 and stage 3 ($p=0.497$) (Table 4-21 & 4-22).

Left ventricular diastolic dysfunction was found in total 46 cases and was seen to be highly significant between stage 1 and 3 ($p<0.001$) and significant between stage 2 and 3 hypertension ($p=0.013$). LVDD was found to be insignificant between stage 1 and stage 2 hypertension ($p=0.127$) (Table 4-27a & b). Left


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ventricular systolic dysfunction was found in total of 22 cases and was significant between stage 1 and 2 hypertension (p=0.029) and highly significant between stage 1 and 3 (p<0.001) but not significant between stage 2 and stage 3 (p=0.195) (Table 4-28 a & b). Cardiovascular complications were significant between stage 1 and stage 3 hypertension (p=0.023) but so between stage 1 and stage 2 (p=0.965) or stage 2 and stage 3 (p=0.056) (Table 4-29).
Cardiovascular risk factors when combined were seen to cause significant cardiovascular disease compared with absence of risk factors (p=0.036) (Table 4-30).

## 5) CONCLUSION:

Hypertension remains a common diagnosis among patients presenting to Medical Department of Osh Regional Integrated Clinical Hospital and Osh City Territoral Clinical Hospital of The Kyrgyz Republic. It leads to considerable amount of morbidity and mortality. In the cohort, 27.5% of patients were asymptomatic, having been incidentally diagnosed as hypertensive.

Hypertension is a major cardiovascular risk factor. To add to the misery, these patients also tend to harbour other cardiovascular risk factors which significantly increases cardiovascular morbidity and mortality. Cardiovascular complications (47.5% patients among the cohort presenting with cardiovascular disease) were significantly increased in the presence of the risk factors compared to patients without risk factors. This study showed high prevalence of such risk factors among the cohort. Smoking was prevalent to a level of concern, being in the range of 70.45 % in men and 64 % in women. Diabetes mellitus was found in 27.27 % of men and 19.44 % of women, in whom strict blood pressure management is a key to prevent vascular complications. Dyslipidemia was seen in 22.72 % of men and 36.11 % of women. Around one half of the patients in each gender group were above 60 years age and among female patients 75 % were in the postmenopausal age group. Alcohol consumption and Obesity are the another key risk factors among the studied population, being 75 % of men and 19.4 % of women alcohol consumers. Obesity was seen in 27.27 % of men and 33.33 % of women in the studied group.

Most common clinical cardiovascular complication was left ventricular hypertrophy being present in 62.5% of total population and majority of patients had presented with angina or myocardial infarction (25%) and the other major complication being heart failure (10.5%).

Among the cardiovascular parameters studied significant values were found in relation to different stages of hypertension specially between Stage 1 and Stage 3 hypertension. Cardiomegally in chest x-ray, LVH in electrocardiogram, low LV ejection fraction, increased LV mass, increased LV mass index, LV diastolic dysfunction, LV systolic dysfunction and overall cardiovascular complications were significant between these two stages of Hypertension. Increase in LV mass, increase in LV mass index and LV diastolic dysfunction were significant between stage 2 and stage 3 but otherwise not much significance was found between stage 2 and 3 hypertension.
Hence, **Left Ventricular Hypertrophy** remains a major complication of uncontrolled hypertension and remains central to significant further cardiovascular adverse events.

## OBSERVATIONS:

**The relevant results and statistical analysis of the study is given below in tabulated forms:**


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**Table 4.1 General Information:**

| Sex | No. of Patients | Stages of Blood Pressure | | | | Total No. of Patients |
|---|---|---|---|---|---|---|
| | | Stage I (140-149/90-99) | Percentage | Stage II & III (≥160/≥100) | Percentage | |
| Male (55%) | 44 | 13 | 29.5 % | 31 | 70.45 % | 44 |
| Female (45%) | 36 | 9 | 25 % | 27 | 75 % | 36 |
| Total No. Patients | 80 | 22 | | 58 | | 80 |
| Percentage | | 27.5 % | | 72.5 % | | |

**Table 4.2 Prevalence of Cardiovascular Risk Factors in the Study Population, Stratified by both Sexes (Whole No.):**

| Total No. Sex / Pt. | Stages of Blood Pressure (HTN) | No. of Patients | Cardiovascular Risk Factors | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | Smoking | DM | Dis.Lip | Obesity | Age> 60 | Alc. Con. | Post. Menopa. |
| Male / No. 44 | Stage I | 13 | 8 | 2 | 5 | 2 | 5 | 10 | _ |
| | Stage II & III | 31 | 23 | 10 | 5 | 10 | 13 | 23 | _ |
| Total No. of Male Patients | | 44 | 31 | 12 | 10 | 12 | 18 | 33 | _ |
| Female / No. 36 | Stage I | 9 | 6 | 2 | 3 | 2 | 1 | 1 | 7 |
| | Stage II & III | 27 | 17 | 5 | 10 | 10 | 14 | 6 | 20 |
| Total No. of Female Patients | | 36 | 23 | 7 | 13 | 12 | 15 | 7 | 27 |
| Grand Total No. of Patients (Male + Female) | | 80 | 54 | 19 | 23 | 24 | 33 | 40 | 27 |

**Table 4.3 Prevalence of Cardiovascular Risk Factors in the Study Population, Stratified by both Sexes,**
**Percentage (%):**

| Total No. Sex / Pt. | Stages of Blood Pressure (HTN) | No. of Patients | Cardiovascular Risk Factors ( % ) | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | Smoking | DM | Dis.Lip | Obesity | Age > 60 | Alc. Con. | Post. Menopa. |
| Male / No. 44 | Stage I | 13 | 62 | 15.4 | 38.5 | 15.4 | 38.5 | 77 | _ |
| | Stage II & III | 31 | 74.19 | 32.25 | 16.12 | 32.25 | 42 | 74.2 | _ |
| Total No. of Male Patients | | 44 | 70.45 | 27.27 | 22.72 | 27.27 | 41 | 75 | _ |
| Female / No. 36 | Stage I | 9 | 67 | 22.22 | 33.33 | 22.22 | 11.11 | 11.1 | 78 |
| | Stage II & III | 27 | 63 | 19 | 37.03 | 37.03 | 52 | 22.2 | 74.07 |
| Total No. of Female Patients | | 36 | 64 | 19.44 | 36.11 | 33.33 | 42 | 19.4 | 75 |
| Grand Total No. of Patients (Male + Female) | | 80 | 68 | 24 | 29 | 30 | 41.25 | 50 | |


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**Table 4.4 Prevalence of Cardiovascular Risk Factors in the Study Population, Stratified by Male (Whole No.):**

| Total No. Sex / Pt. | Stages of Blood Pressure (HTN) | No. of Patients | Cardiovascular Risk Factors | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | Smoking | DM | Dis.Lip | Obesity | Age> 60 | Alc. Con. | Post. Menopa. |
| Male / No. 44 | Stage I | 13 | 8 | 2 | 5 | 2 | 5 | 10 | _ |
| | Stage II & III | 31 | 23 | 10 | 5 | 10 | 13 | 23 | _ |
| Total No. of Male Patients | | 44 | 31 | 12 | 10 | 12 | 18 | 33 | _ |

**Table 4.5 Prevalence of Cardiovascular Risk Factors in the Study Population, Stratified by Male, Percentage (%):**

| Total No. Sex / Pt. | Stages of Blood Pressure (HTN) | No. of Patients | Cardiovascular Risk Factors (% ) | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | Smoking | DM | Dis.Lip | Obesity | Age> 60 | Alc. Con. | Post. Menopa. |
| Male / No. 44 | Stage I | 13 | 62 | 15.4 | 38.5 | 15.4 | 38.5 | 77 | _ |
| | Stage II & III | 31 | 74.19 | 32.25 | 16.12 | 32.25 | 42 | 74.2 | _ |
| Total Male Patients | | 44 | 70.45 | 27.27 | 22.72 | 27.27 | 41 | 75 | _ |

**Fig: 1.**

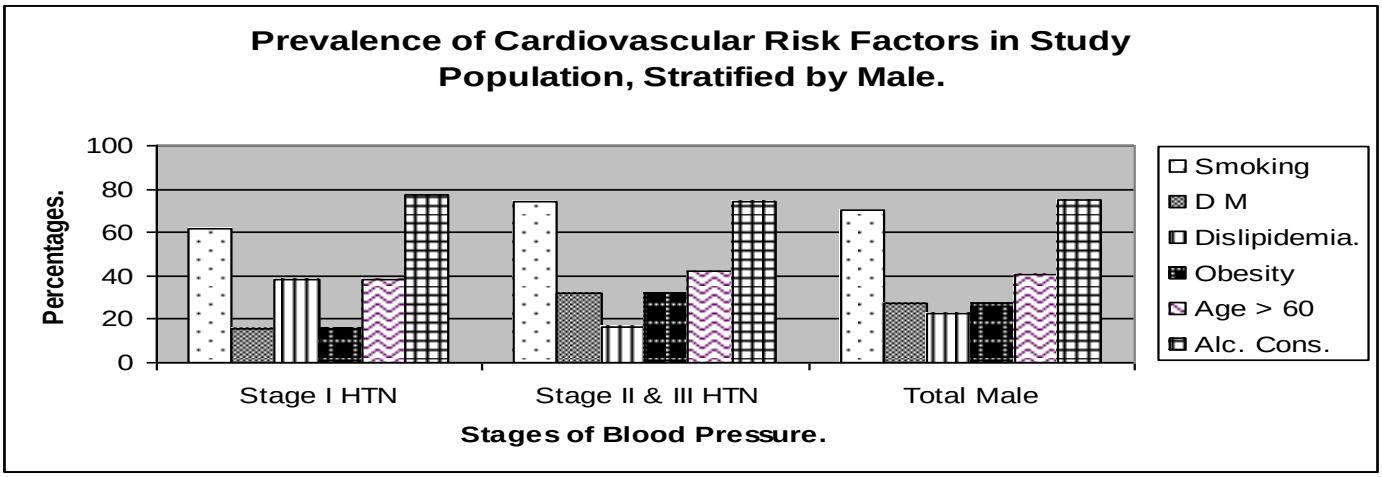


**Table 4.6 Prevalence of Cardiovascular Risk Factors in the Study Population, Stratified by Female, (Whole No.):**

| Total No. Sex / Pt. | Stages of Blood Pressure (HTN) | No. of Patients | Cardiovascular Risk Factors | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | Smoking | DM | Dis.Lip | Obs. | Age> 60 | Alc. Con. | Post. Menopa. |
| Female / No. 36 | Stage I | 9 | 6 | 2 | 3 | 2 | 1 | 1 | 7 |
| | Stage II & III | 27 | 17 | 5 | 10 | 10 | 14 | 6 | 20 |
| Total No. of Female Patients | | 36 | 23 | 7 | 13 | 12 | 15 | 7 | 27 |


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**Table 4.7 Prevalence of Cardiovascular Risk Factors in the Study Population, Stratified by Female, Percentage (%):**

| Total No. Sex / Pt. | Stages of Blood Pressure (HTN) | No. of Patients | Cardiovascular Risk Factors | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | Smoking | DM | Dis.Lip | Obs. | Age> 60 | Alc. Con. | Post. Menopa. |
| Female / No. 36 | Stage I | 9 | 67 | 22.22 | 33.33 | 22.22 | 11.11 | 11.1 | 78 |
| | Stage II & III | 27 | 63 | 19 | 37.03 | 37.03 | 52 | 22.2 | 74.07 |
| Total Female Patients | | 36 | 64 | 19.44 | 36.11 | 33.33 | 42 | 19.4 | 75 |

**Fig: 2.**

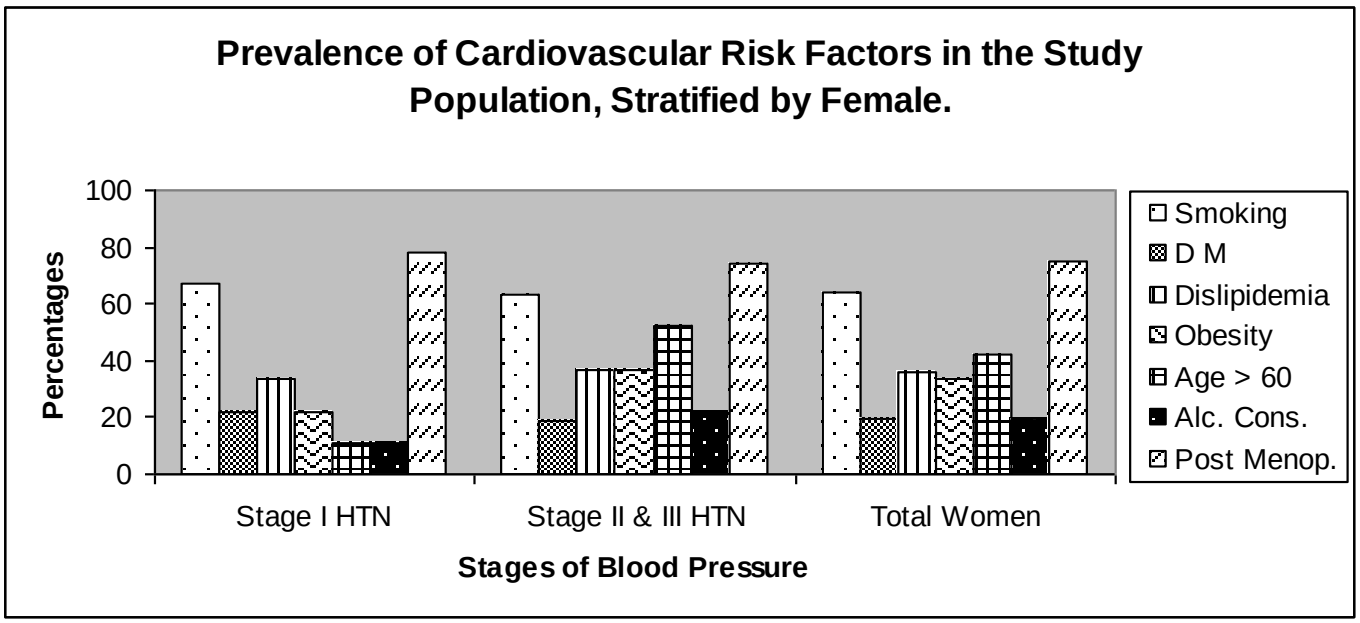


**Table 4.8 CRP Positive in Hypertensive Cases:**

| Sex | Total No. of Cases | Total No. of Cases, CRP Positive | Percentage % (Male & ) Female |
|---|---|---|---|
| **Male** | 44 | 9 | 20.45 |
| **Female** | 36 | 4 | 11.11 |
| **Total No. of Cases** | 80 | 13 | |
| **Percentage (In total)** | | 16.25 % | |

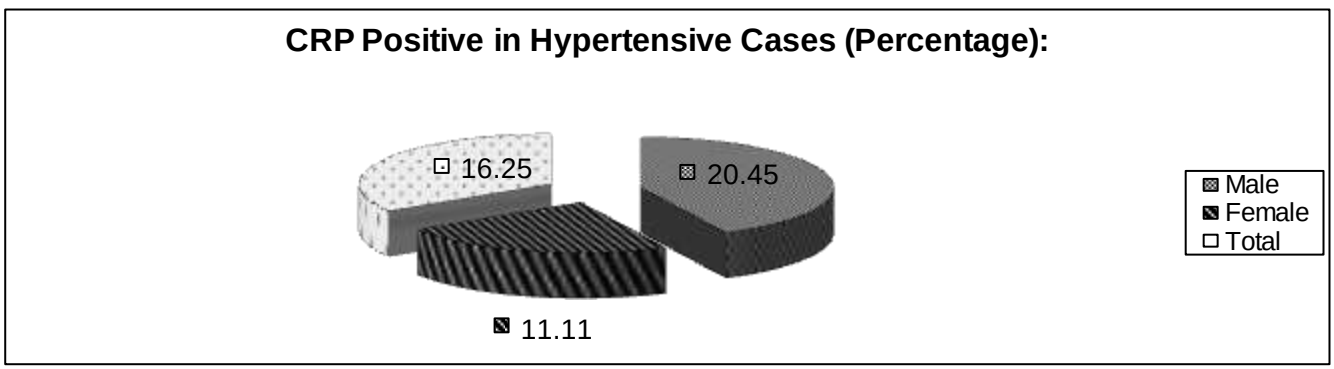



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**Table 4.9 Clinical Presentation of Hypertension in the Study:**

| S. N. | Features Primarily Attributable To | No. of Cases | Percentage (%) |
|---|---|---|---|
| 1. | Cardiovascular Diseases | 38 | 47.5 |
| 2. | Cerebro vascular Diseases | 16 | 20.0 |
| 3. | Renal Diseases | 3 | 3.8 |
| 4. | Peripheral arterial Diseases | 1 | 1.3 |
| 5. | Asymptomatic (Incidentally Diagnosed) | 22 | 27.5 |
| Total No. of Cases | | 80 | 100 |

**Fig: 3a.**

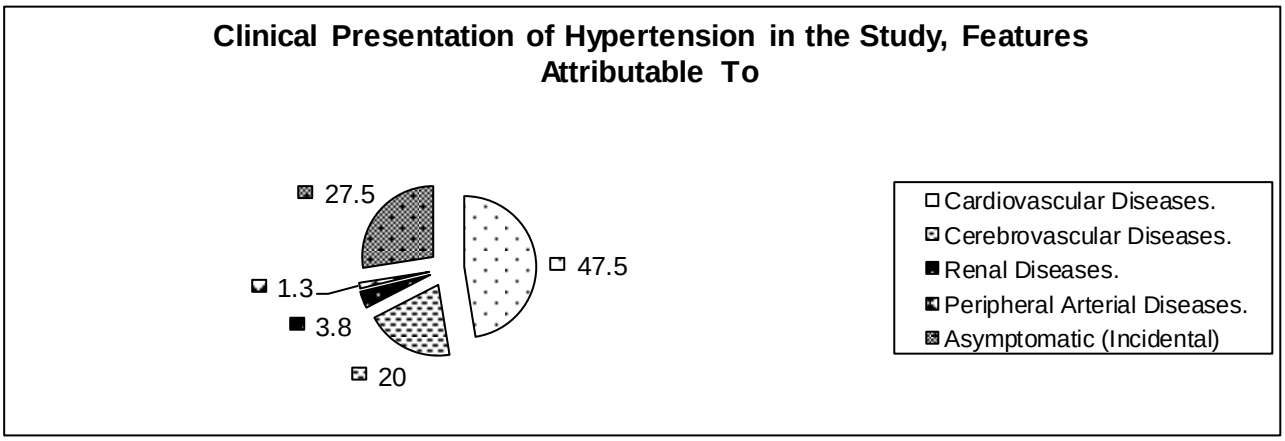


**Fig: 3b.**

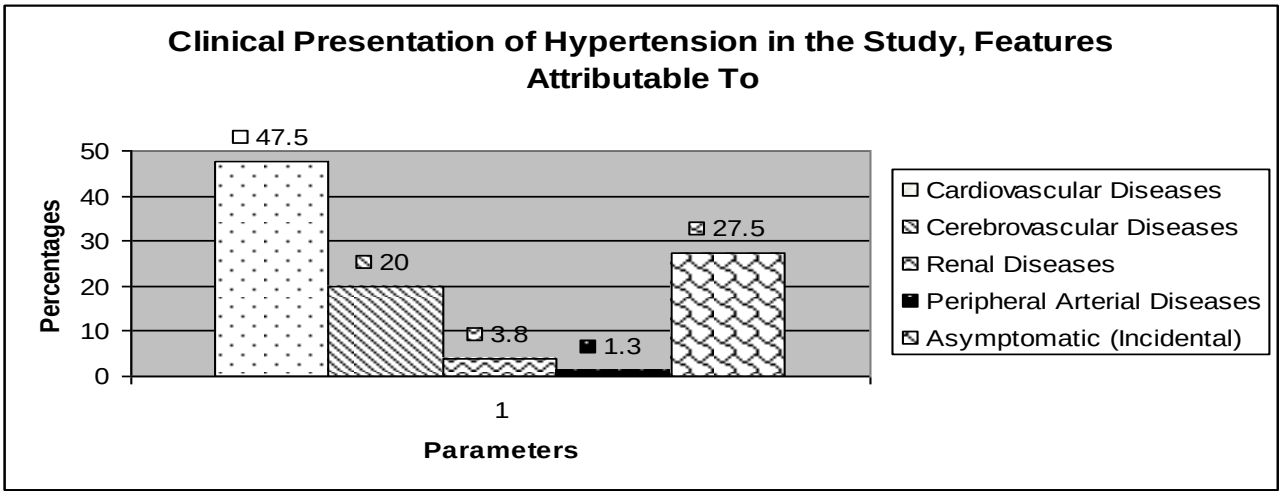


**Table 4.10 Clinical Cardiovascular Diseases:**

| S. N. | Cardiovascular Diseases | No. of Cases | Percentage (%) |
|---|---|---|---|
| 1. | Left Ventricular Hypertrophy | 50 | 62.5 |
| 2. | Angina / Myocardial Infarction | 20 | 25.0 |
| 3. | Prior Coronary Revascularization | 0 | 0.0 |
| 4. | Heart Failure | 10 | 12.5 |
| Total No. of Cases | | 80 | 100 |


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*** % (Percentage) age is from total no of cases.**

**Fig: 4.**

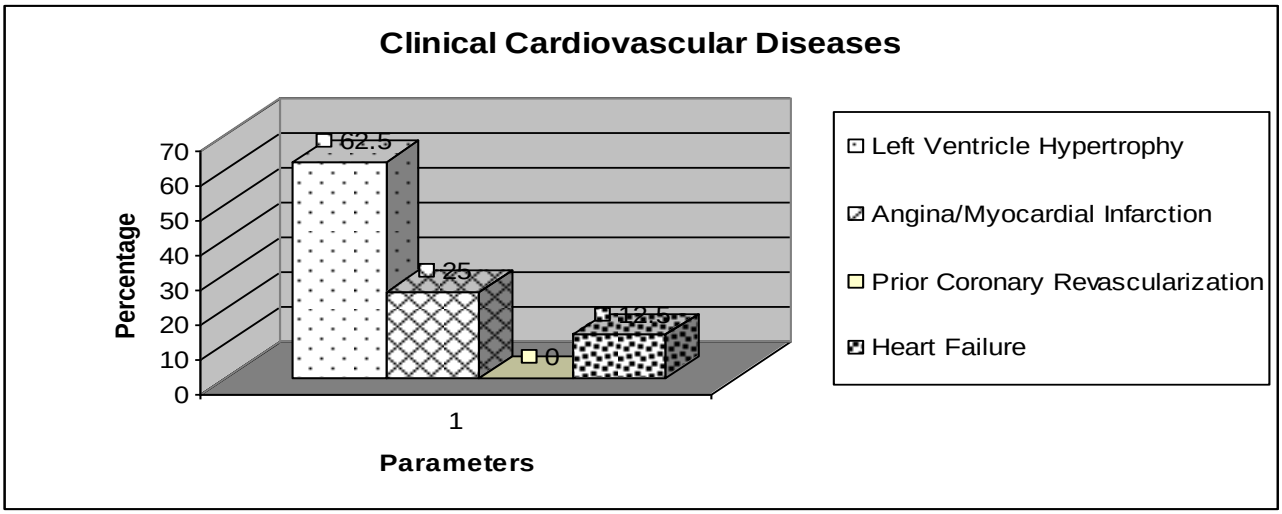


**Table 4.11 Demographic and Laboratory Data:**

| S. N. | Parameters | Number | Range | Minimum | Maximum | Mean | S D |
|---|---|---|---|---|---|---|---|
| 1. | Age (Year) | 80 | 42 | 38 | 80 | 59.15 | 9.66 |
| 2. | Systolic B.P. (mmHg) | 80 | 78 | 130 | 208 | 155.75 | 15.88 |
| 3. | Diastolic B.P. (mmHg) | 80 | 50 | 84 | 134 | 100.78 | 10.42 |
| 4. | BMI (Kg / $m^2$) | 80 | 11.59 | 19.86 | 31.45 | 24.66 | 2.68 |
| 5. | BSA (Body Surface Area) | 80 | 0.7 | 1.26 | 1.96 | 1.59 | 0.14 |
| 6. | Blood Glucose (mmol / l) | 80 | 13.4 | 4.8 | 18.2 | 8.29 | 3.12 |
| 7. | Cholesterol (mmol / l) | 80 | 4.7 | 3.1 | 7.8 | 5.09 | 1.1 |
| 8. | HDL Cholesterol (mmol / l) | 80 | 0.8 | 0.6 | 1.4 | 0.92 | 0.20 |
| 9. | LDL Cholesterol (mmol / l) | 80 | 4.8 | 1.8 | 6.6 | 3.79 | 1.09 |
| 10. | Triglyceride (mmol / l) | 80 | 3.2 | 0.6 | 3.8 | 1.94 | 0.74 |
| 11. | CCR (ml / min) | 80 | 127.3 | 7.1 | 134.4 | 58.34 | 28.84 |

***BMI*** *= Body Mass Index,* ***BSA*** *= Body Surface Area,* ***CCR*** *= Creatinine Clearance ,*

***S D*** *= Standard Deviation.*

**Table 4.12 Times since Diagnosis of Hypertension:**

| Years | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|
| 0 | 29 | 33.7 | 36.3 | 36.3 |
| 0.5 | 2 | 2.3 | 2.5 | 38.8 |
| 1 | 3 | 3.5 | 3.8 | 42.5 |
| 1.5 | 2 | 2.3 | 2.5 | 45 |
| 2 | 4 | 4.7 | 5 | 50 |
| 2.5 | 3 | 3.5 | 3.8 | 53.8 |
| 3 | 4 | 4.7 | 5 | 58.8 |
| 3.5 | 3 | 3.5 | 3.8 | 62.5 |
| 4 | 4 | 4.7 | 5 | 67.5 |
| 4.5 | 2 | 2.3 | 2.5 | 70 |


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| | | | | |
|---|---|---|---|---|
| 5 | 8 | 9.3 | 10 | 80 |
| 5.5 | 1 | 1.2 | 1.3 | 81.3 |
| 6 | 3 | 3.5 | 3.8 | 85 |
| 7 | 3 | 3.5 | 3.8 | 88.8 |
| 7.5 | 1 | 1.2 | 1.3 | 90 |
| 8 | 2 | 2.3 | 2.5 | 92.5 |
| 8.5 | 1 | 1.2 | 1.3 | 93.8 |
| 9 | 1 | 1.2 | 1.3 | 95 |
| 10 | 2 | 2.3 | 2.5 | 97.5 |
| 12 | 2 | 2.3 | 2.5 | 100 |

**Table 4.13 Times the Diagnosis of Hypertension:**

| | | |
|---|---|---|
| No. | Valid | 80 |
| | Mean | 2.99 |
| | Standard Deviation | 3.20 |

**Table 4.14 Cardiomegally in Chest X – Ray:**

| S. N. | Parameters | Significance |
|---|---|---|
| **1.** | Stage 1 HTN versus Stage 2 HTN | 0.018 |
| **2.** | Stage 1 HTN versus Stage 3 HTN | 0.001 |
| **3.** | Stage 2 HTN versus Stage 3 HTN | 0.625 |
| **The Mean Difference is Significant at 0.05 Level.** | | |

**Table 4.15 LVH (Left Ventricle Hypertrophy) in ECG:**

| S. N. | Parameters | Significance (P – Value) |
|---|---|---|
| **1.** | Stage 1 HTN versus Stage 2 HTN | 0.012 |
| **2.** | Stage 1 HTN versus Stage 3 HTN | 0.000 |
| **3.** | Stage 2 HTN versus Stage 3 HTN | 0.135 |
| **The Mean Difference is Significant at 0.05 Level.** | | |

**Table 4.16 Echocardiographic Measurements:**

| S. N. | Parameters | Mean | Standard Deviation |
|---|---|---|---|
| **1.** | Left Ventricular End – Diastolic Diameter (LVEDD) | 5.359 | 0.882 |
| **2.** | Left Ventricular End – Systolic Diameter (LVESD) | 3.842 | 1.026 |
| **3.** | Inter Ventricular Septal Diameter (IVSD) | 0.924 | 0.178 |
| **4.** | Left Ventricle Posterior Wall Diameter (LVPWD) | 1.292 | 0.213 |

**Table 4.17 Left Ventricular (L V) End – Diastolic Diameter:**

| S. N. | Parameters | Significance (P – Value) |
|---|---|---|
| **1.** | Stage 1 HTN versus Stage 2 HTN | 0.009 |
| **2.** | Stage 1 HTN versus Stage 3 HTN | 0.000 |
| **3.** | Stage 2 HTN versus Stage 3 HTN | 0.006 |
| **The Mean Difference is Significant at 0.05 Level.** | | |


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**Table 4.18 Left Ventricular (L V) End Systolic Diameter:**

| S. N. | Parameters | Significance (P – Value) |
|---|---|---|
| 1. | Stage 1 HTN versus Stage 2 HTN | 0.003 |
| 2. | Stage 1 HTN versus Stage 3 HTN | 0.000 |
| 3. | Stage 2 HTN versus Stage 3 HTN | 0.029 |
| The Mean Difference is Significant at 0.05 Level. | | |

**Table 4.19 Inter – Ventricular Septal Diameter (IVSD):**

| S. N. | Parameters | Significance (P – Value) |
|---|---|---|
| 1. | Stage 1 HTN versus Stage 2 HTN | 0.085 |
| 2. | Stage 1 HTN versus Stage 3 HTN | 0.000 |
| 3. | Stage 2 HTN versus Stage 3 HTN | 0.143 |
| The Mean Difference is Significant at 0.05 Level. | | |

**Table 4.20 Left Ventricular Posterior Wall Diameter (LVPWD):**

| S. N. | Parameters | Significance (P – Value) |
|---|---|---|
| 1. | Stage 1 HTN versus Stage 2 HTN | 0.591 |
| 2. | Stage 1 HTN versus Stage 3 HTN | 0.058 |
| 3. | Stage 2 HTN versus Stage 3 HTN | 0.405 |
| The Mean Difference is Significant at 0.05 Level. | | |

**Table 4.21 Total No. of Cases With Low L V Ejection Fraction:**

| S. N. | Parameters | No. of Patients |
|---|---|---|
| 1. | Stage 1 HTN | 1 |
| 2. | Stage 2 HTN | 7 |
| 3. | Stage 3 HTN | 12 |
| | Total No. of Patients | 20 |
| | Percentage (%) | 25 |

**Table 4.22 Left Ventricle Ejection Fraction:**

| S. N. | Parameters | Significance (P – Value) |
|---|---|---|
| 1. | Stage 1 HTN versus Stage 2 HTN | 0.014 |
| 2. | Stage 1 HTN versus Stage 3 HTN | 0.000 |
| 3. | Stage 2 HTN versus Stage 3 HTN | 0.497 |
| The Mean Difference is Significant at 0.05 Level. | | |

**Table 4.23 Left Ventricle Mass:**


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| S. N. | Parameters | No. of Patients | Mean | Standard Deviation |
|---|---|---|---|---|
| 1. | Stage 1 HTN | 30 | 214.64 | 88.99 |
| 2. | Stage 2 HTN | 25 | 291.04 | 113.92 |
| 3. | Stage 3HTN | 25 | 383.60 | 96.44 |
| Total | | 80 | 291.32 | 120.84 |

**Table 4.24 Left Ventricle Mass by Echocardiography:**

| S. N. | Parameters | Significance (P – Value) |
|---|---|---|
| 1. | Stage 1 HTN versus Stage 2 HTN | 0.016 |
| 2. | Stage 1 HTN versus Stage 3 HTN | 0.000 |
| 3. | Stage 2 HTN versus Stage 3 HTN | 0.004 |
| The Mean Difference is Significant at 0.05 Level. | | |

**Table 4.25 Left Ventricle Mass Index:**

| S. N. | Parameters | No. of Patients | Mean | Standard Deviation |
|---|---|---|---|---|
| 1. | Stage 1 HTN | 30 | 137.027 | 59.013 |
| 2. | Stage 2 HTN | 25 | 180.836 | 69.636 |
| 3. | Stage 3HTN | 25 | 241.732 | 63.015 |
| Total | | 80 | 183.438 | 76.508 |

**Table 4.26 Left Ventricle Mass Index by Echocardiography:**

| S. N. | Parameters | Significance (P – Value) |
|---|---|---|
| 1. | Stage 1 HTN versus Stage 2 HTN | 0.035 |
| 2. | Stage 1 HTN versus Stage 3 HTN | 0.000 |
| 3. | Stage 2 HTN versus Stage 3 HTN | 0.003 |
| The Mean Difference is Significant at 0.05 Level. | | |

**Table 4.27a Total No. Cases with Left Ventricular (LV) Diastolic Dysfunction:**

| S. N. | Parameters | No. of Patients | Percentage (%) |
|---|---|---|---|
| 1. | Stage 1 HTN | 8 | 10 |
| 2. | Stage 2 HTN | 16 | 20 |
| 3. | Stage 3 HTN | 22 | 27.5 |
| Total | | 46 | 57.5 |
| Total No. of Cases = 80 | | | |

**Table 4.27b Left Ventricular (LV) Diastolic Dysfunction:**

| S. N. | Parameters | Significance (P – Value) |
|---|---|---|
| 1. | Stage 1 HTN versus Stage 2 HTN | 0.127 |
| 2. | Stage 1 HTN versus Stage 3 HTN | 0.000 |
| 3. | Stage 2 HTN versus Stage 3 HTN | 0.013 |
| The Mean Difference is Significant at 0.05 Level. | | |


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**Table 4.28a Total No. Cases with Left Ventricular (LV) Systolic Dysfunction:**

| S. N. | Parameters | No. of Patients | Percentage (%) |
|---|---|---|---|
| **1.** | Stage 1 HTN | 1 | 1.25 |
| **2.** | Stage 2 HTN | 8 | 10 |
| **3.** | Stage 3 HTN | 13 | 16.25 |
| **Total** | | 22 | 27.5 |
| **Total No. of Cases = 80** | | | |

**Table 4.28b Left Ventricular (LV) Systolic Dysfunction:**

| S. N. | Parameters | Significance (P – Value) |
|---|---|---|
| **1.** | Stage 1 HTN versus Stage 2 HTN | 0.029 |
| **2.** | Stage 1 HTN versus Stage 3 HTN | 0.000 |
| **3.** | Stage 2 HTN versus Stage 3 HTN | 0.195 |
| **The Mean Difference is Significant at 0.05 Level.** | | |

**Table 4.29 Cardiovascular Complications:**

| S. N. | Parameters | Significance (P – Value) |
|---|---|---|
| **1.** | Stage 1 HTN versus Stage 2 HTN | 0.965 |
| **2.** | Stage 1 HTN versus Stage 3 HTN | 0.023 |
| **3.** | Stage 2 HTN versus Stage 3 HTN | 0.056 |
| **The Mean Difference is Significant at 0.05 Level.** | | |

**Table 4.30 CVS Risk Factors Vs Cardiovascular System Diseases:**

| S. N. | Parameters | Significance (P – Value) |
|---|---|---|
| **1.** | Without all Risk Factors Versus With all Risk Factors | 0.036 |
| **The Mean Difference is Significant at 0.05 Level.** | | |

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