`

"ESTIMATION OF SERUM URIC ACID LEVEL IN NEWLY DIAGNOSED ESSENTIAL HYPERTENSION"

By

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Dissertation submitted to

BLDE (Deemed to be University), Vijayapura

In partial fulfilment of the requirements for the award of the degree of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

Under the guidance of

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2020

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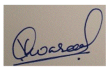

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Dr. HARSHVARDHAN NARUTE.

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LIST OF ABBREVIATIONS USED

AAP	4-Amino antipyrine
ACE	Angiotensin converting enzyme
ATP	Adenosine triphosphate
ARB	Angiotensin receptor blocker
BP	Blood Pressure
CCB	Calcium channel blocker
CHD	Coronary heart disease
CVD	Cardio Vascular Disease
DBP	Diastolic Blood Pressur
GFR	Glomerular Filtration rate
GMP	Guanylic acid
GNP	Guanosine monophosphate
HF	Heart Failure
HPRT	Hypoxanthine phosphoribosyl transferase
HTN	Hypertension
hUAT	Human uric acid transporter
IHD	Ischemic Heart Disease
IMP	Inosine Monophosphate
JNC	Joint National Committee
LV	Left Ventricle
LVH	Left ventricular hypertrophy
MI	Myocardial infarction
MRI	Magnetic resonance imaging
NaCl	Sodium chloride
NHANES	National health and nutrition survey

`

PRPP	Phosphoribosyl pyrophosphate
PRA	Phosphoribosylamine
RR	Relative risk
SAICAR	Succinylaminoimidazole Carboxamide Ribotate
SBP	Systolic blood pressure
SUA	Serum uric acid
URATI	Urate transporter 1.

`

ABSTRACT

Need of the study

The association of raised serum uric acid levels with various cardiovascular risk factors has often led to the debate of whether raised serum uric acid levels could be an independent risk factor in essential hypertension. Hence we carried out a study to examine the possibility of hyperuricemia causing hypertension, to see if there is a relationship between the serum uric acid levels and hypertension.

Methodology

The study was carried out in Shri B. M. Patil Medical College Vijayapur, the study period was of 18 months from November 2018 to June 2020 a total 93 patients of hypertension were studied. The patients were included if they satisfied the JNC VII criteria for hypertension. They were excluded if they were having any other condition known to cause raised serum uric acid levels & secondary hypertension.

Results

The study showed that serum uric acid (SUA) levels were raised in patients with newly diagnosed essential hypertension. out of ninety three hypertensive cases (male-sixty six, female-twenty seven). High serum uric acid level were observed in fourty nine patients (i.e- fifty two percentage of hypertension patents)

Interpretation & Conclusion

Based on the study carried out we concluded that there is association between between serum uric acid and blood pressure.

Key words:
Serum Uric Acid; Hypertension; JNC VII; Hyperuricemia.

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INTRODUCTION

Uric acid is a final result of purine metabolism, was found first in 1776. A Swedish researcher Scheele segregated it from a urinary stone. In 1797, a British researcher Wallaston identified uric acid in a tophus which was taken out from his own ear.

At that point About 50 years after the fact Alfred Baring Garrod, a british doctor appeared by compound confinement that uric acid was unusually high in gouty patients. In further investigations Garrod planned a reasonable connection among hyperuricemia and symptomatology of gouty patients[1].

In 1957, association between hypertension and raised uric acid was recognized when family with a unique and unfortunate pedigree attended Hammer Smith hospital. A family including, the dad and six of the seven kin had hyperuricemia, while the mother and all the kin had hypertension. this brought up the issue whether a raised serum uric acid was normal in patients with hypertension. So, this incidence raised a question whether raised serum uric acid levels were common in patients with hypertension[1].

`

OBJECTIVE OF THE STUDY

- To study the association of serum uric acid level with newly diagnosed essential hypertension.

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REVIEW OF LITERATURE

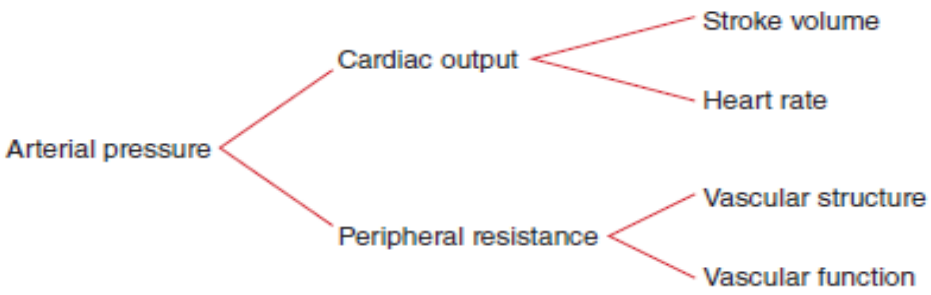
MECHANISM OF HYPERTENSION

A. INTRAVASCULAR VOLUME

Vascular volume is increased when NACL (sodium chloride) intake is more and kidney is unable to excrete sodium. vascular volume is directly proportional to cardiac output

BLOOD FLOW= PRESSURE ACROSS THE VASCULAR BED

VASCULAR RESISTANCE



Cardiac output= Stroke volume x Heart rate

Peripheral resistance = vascular structure x vascular function

Arterial pressure = cardiac output x peripheral resistance[2].

B. AUTONOMIC NERVOUS SYSTEM

Blood pressure raises over a short period of time because of adrenergic reflexes, Adrenergic receptors have 2 subtypes alpha and beta which are further divided into alpha 1, alpha 2 beta 1 and beta 2 receptors. Norepinephrine, epinephrine and dopamine plays an important part in regulating blood pressure.

Pheochromocytoma is most important example of raised blood pressure related to increased Catecholamines[2].

C. RENIN-ANGIOTENSIN ALDOSTERONE

`

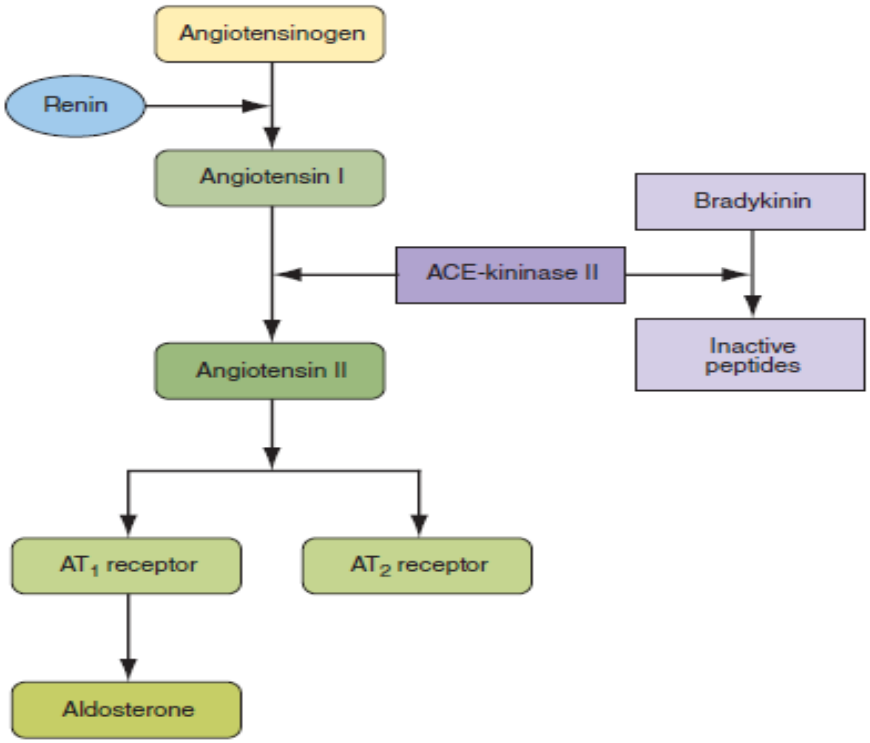
Renin angiotensin aldosterone system causes blood pressure regulation through

1) Vasoconstriction mediated by angiotensin II.
2) Sodium and water retention secondary to aldosterone,

Stimuli to renin synthesis-

1) Reduced Nacl transport to distal portion of thick ascending loop of Henle.
2) Reduced pressure in afferent arteriole (baroreceptor mechanism).
3) B1 receptor stimulation to activate sympathetic nervous system[2].

Renin –secreting Tumour causes hypertension, e.g: Wilms tumours.



VASCULAR MECHANISM

Radius of vessels and compliance of resistance arteries are important to arterial pressure[2].

`

IMMUNE MECHANISM , INFLAMMATION AND OXIDATIVE

This is also responsible in pathology of hypertension, patient with hypertension have circulating antibodies[2].

PATHOLOGIC CONSEQUENCES OF HYPERTENSION

HEART-

- Increased morbidity due to heart disease is more in hypertensive cases.
- Hypertensive heart disease leads to left ventricular hypertrophy, Congestive heart disease, stroke, cardiac arrhythmias, atherosclerosis in coronary heart disease.
- Left ventricular Hypertrophy occurs as a response to the increased afterload due to elevated systemic vascular resistance.
- Left ventricular hypertrophy can be prevented by aggressive management of hypertension to prevents cardiovascular disease[2,3].

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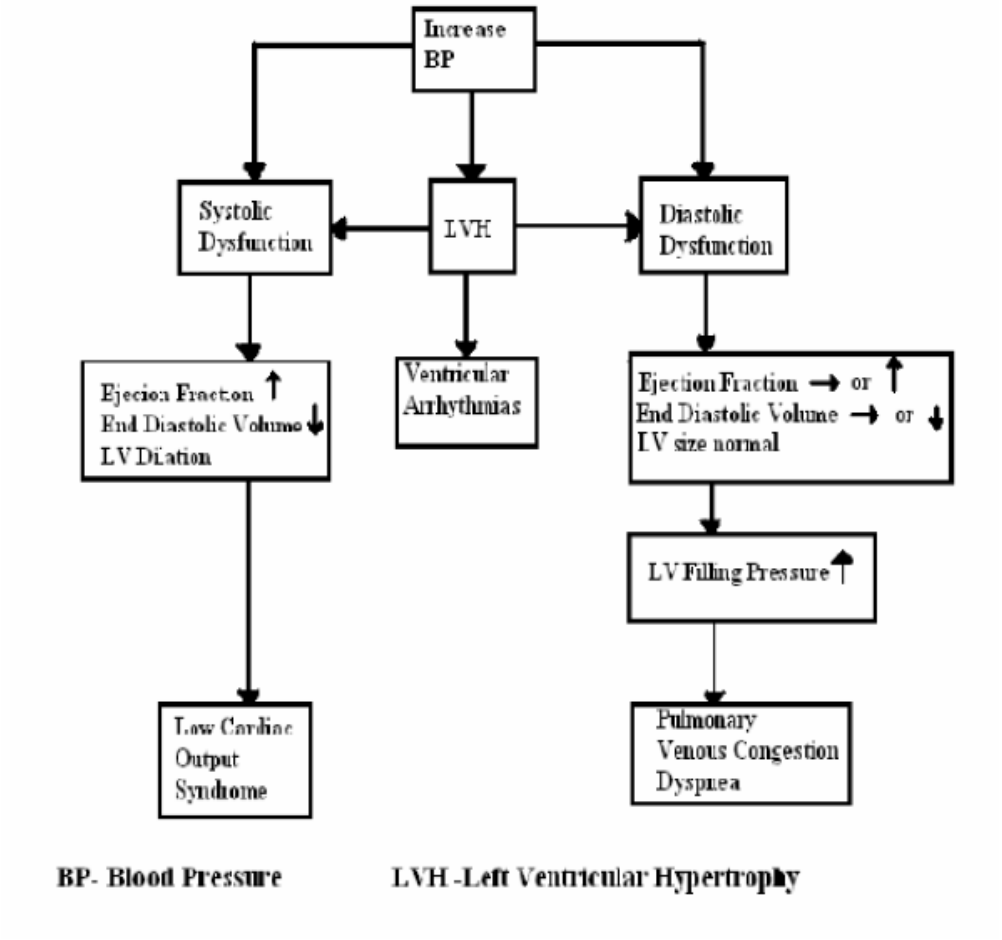

BRAIN

- Hypertension is a major risk factor for cerebral infraction and intracranial haemorrhage.
- About 85% stroke is because of infraction.
- 15% stroke because of intracerebral or subarachnoid haemorrhage.
- Hypertension also leads to impairment of cognition in ageing population.
- Also leading cause for dementia.
- Cerebral blood flow is well known for autoregulation but malignant hypertension and encephalopathy causes failure of autoregulation[8].

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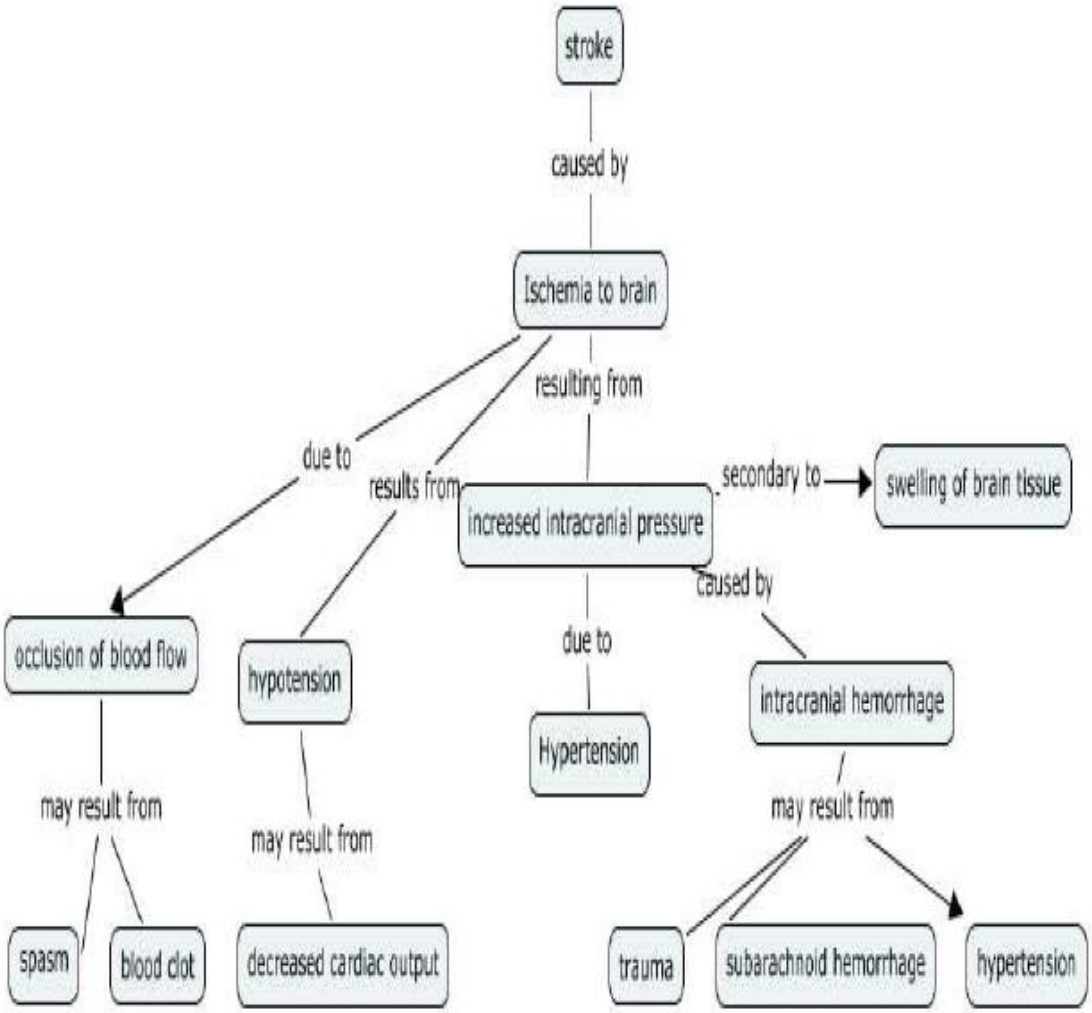

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KINDEY

- Hypertension also affects the kidney.
- Kidney diseases are also cause for secondary hypertension.
    1) Not able to excrete sodium
    2) Renin secretion related to volume stasis and sympathetic nervous systeme over activity.
- Hypertension is most important risk factor for end stage renal disease and renal injury.
- Hypertension causes ischemic changes in the glomeruli and post glomerular structures with glomerular sclerosis.
- Associated with microalbuminuria (random urine albumin/creatinine raitio-30-300mg/g) and macroalbuminuria more than 300mg/g are early markers of renal injury[8].

PERIPHERAL ARTERIES-

- Long standing blood pressure also targets the blood vessels causes peripheral arterial disease.
- Hypertension with arterial disease of lower limb are a increased risk for cardiovascular disease.
- Ankle – brachial index help to evaluates peripheral arterial disease[8].

EFFECT OF HYPERTENSION ON THE EYE:

Vascular changes in the fundus reflect both arteriosclerotic retinopathy and hypertensive retinopathy. The hypertensive retinal changes are graded by the Keith – Wegner – Baker classification as[88].

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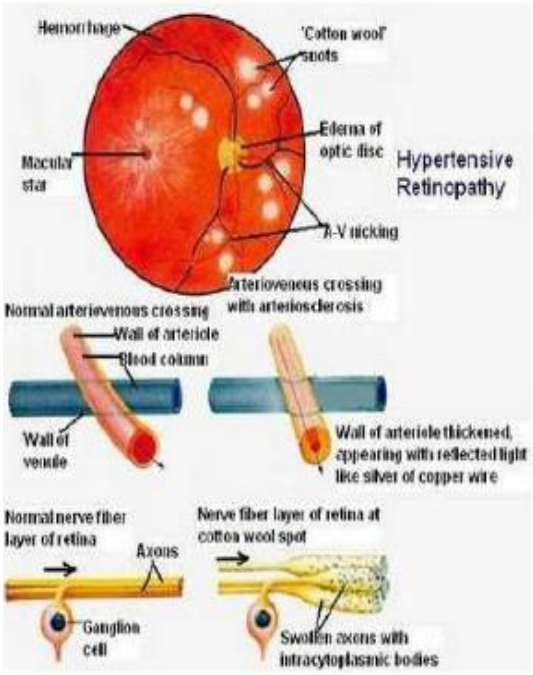

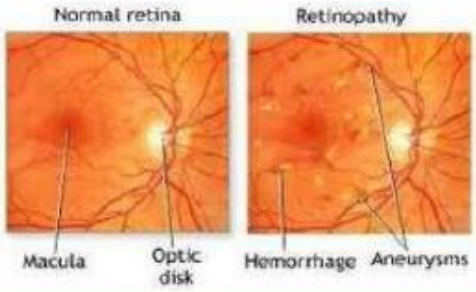

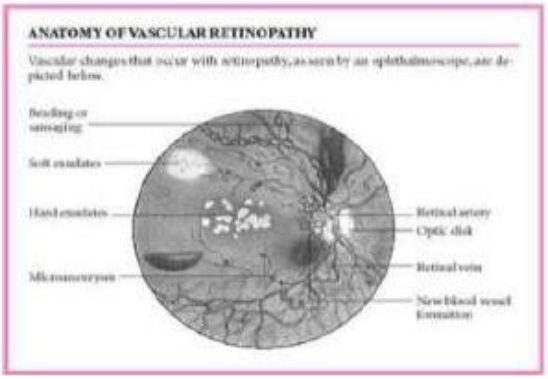

DEFINING HYPERTENSION

Clinically, hypertension may be defined as the level of blood pressure at which the institution therapy reduces blood pressure–related morbidity and mortality. Clinical criteria for defining hypertension generally have been based on the average of two or more seated blood pressure readings during each of two or more outpatient visits. One classification recommends blood pressure criteria for defining normal blood pressure, prehypertension, hypertension (stages I and II), and isolated systolic hypertension, which is frequent among the elderly

“In children and adolescents, hypertension generally is defined as systolic and/or diastolic blood pressure consistently greater than 95th percentile for age, sex, and height”.

`

In study called the Multiple Risk Factor Intervention Trial (MRFIT), which included more than 350,000 male members, which exhibited both systolic and diastolic blood pressure cause a persistent and reviewed impact on coronary heart disease(CHD) mortality, stretching out down to systolic blood pressure of 120 mmHg[2,4].

ACCURATE BLOOD PRESSURE MEASUREMENT

The precise estimation of (blood pressure) BP is important for successful management, the auscultatory and palpatory methods both should be used. Before checking Persons should be seated quietly for at least 5 minutes in a chair (rather than on an evaluation table), with feet on the floor, and arm maintained at heart level. substances like Caffeine, exercise and smoking ought to be stayed away from for at least 30 minutes preceding estimation. Estimation of BP in the standing position is indicated periodically, particularly in the individuals who report symptoms with diminished BP on standing.

To use appropriate sized cuff (cuff should encircle at least 80% of the arm). At least two measurements should be made and the average recorded.

For palpatory methods, radial pulse obliteration pressure should be used to estimate systolic blood pressure (SBP). The cuff to be inflated 20 to 30 mm Hg above this level for the auscultatory method of blood pressure measurement. Then cuff should deflate at the rate of 2 mm Hg per second. SBP is mark at the point where first of two or more Korotkoff sounds is heard (onset of phase 1), and the obliteration of Korotkoff sound (onset of phase 5) is mark as a Diastolic blood pressure (DBP).

In disease like Aortic Regurgitation, the diastolic BP will be 0 mm Hg so the occurrence of muffled sound is taken as diastolic BP. Blood pressure of elderly patient will have auscultatory gap.

`

JNC VII

Based on the seventh report of the Joint National Committee on prevention, detection, evaluation and treatment of high blood pressure (JNC VII report) BP is classified into the following stages[5].

TABLE 271-1 Blood Pressure Classification

BLOOD PRESSURE CLASSIFICATION	SYSTOLIC, mmHg	DIASTOLIC, mmHg
Normal	<120	*and* <80
Prehypertension	120–139	*or* 80–89
Stage 1 hypertension	140–159	*or* 90–99
Stage 2 hypertension	≥160	*or* ≥100
Isolated systolic hypertension	≥140	*and* <90

JNC 8 Hypertension guidelines[6].

The Joint National Committee (JNC 8) guidelines advised about the blood pressure goals and less use of several types of antihypertensive medications.

Important changes from the JNC 7 guidelines includes-

1. In patients 60 years or older who do not have diabetes or chronic kidney disease, the
2. goal blood pressure level is now <150/90 mm Hg.
3. In patients 18 to 59 years of age without major comorbidities, and in patients 60 years or older who have diabetes, chronic kidney disease (CKD), or both conditions, the new goal blood pressure level is <140/90 mm Hg.
4. First-line and later-line treatments should now be limited to 4 classes of medications: thiazide-type diuretics, calcium channel blockers (CCBs),

`

angiotensin converting enzyme (ACE) inhibitors, and angiotensin receptor blocker (ARBs).

5. Second- and third-line alternatives included higher doses or combinations of ACE inhibitors, ARBs, thiazide-type diuretics, and CCBs.

CLASSIFICATION OF HYPERTENSION:

There are two important type of hypertension, primary or essential hypertension and secondary hypertension about 80 to 95% cases are primary or essential hypertension where cause is unknown and the remaining 5–20% of hypertensive patients, a specific underlying disorder causing the elevation of blood pressure can be identified called as secondary hypertension[7].

Classification of Arterial Hypertension:

Systolic hypertension with wide pulse pressure,

- Decreased compliance of aorta
- Aortic regurgitation
- Thyrotoxicosis
- Hyperkinetic Heart Syndrome

Both Systolic and Diastolic Hypertension is seen in Increased peripheral vascular resistance

A.Renal

- Chronic Pyelonephritis can cause arteriolar nephrosclerosis.
- Glomerulonephritis includes both Acute and chronic.
- Polycystic kidney disease (PCOD)

`

B. Endocrine

- Oral Contraceptives
- Adrenocortical Hyperfunction
- Pheochromocytoma
- Thyroid- Myxedema

C. Neurogenic

- Psychogenic is important cause.
- Increased intracranial tension.
- Familial Dysautonomia (Riley-Day Syndrome)

IV. Miscellaneous

A. Coarctation of aorta

B. Increased intravascular volume related (excessive blood transfusion, and Polycythemia Vera)

C. Polyarteritis Nodosa.

GENETIC CONSIDERATION-

Essential hypertension is a polygenic disorder, it involves a multiple genes, which having little effects on blood pressure[8].

CLINICAL DISORDERS OF HYPERTENSION-

80–95% of hypertensive patients are diagnosed as having primary, or "essential," hypertension. In the remaining 5–20% of hypertensive patients, a specific underlying disorder causing the elevation of blood pressure can be identified[2].

`

TABLE 271-3 Secondary Causes of Systolic and Diastolic Hypertension	
Renal	Parenchymal diseases, renal cysts (including polycystic kidney disease), renal tumors (including renin-secreting tumors), obstructive uropathy
Renovascular	Arteriosclerotic, fibromuscular dysplasia
Adrenal	Primary aldosteronism, Cushing's syndrome, 17α-hydroxylase deficiency, 11β-hydroxylase deficiency, 11-hydroxysteroid dehydrogenase deficiency (licorice), pheochromocytoma
Aortic coarctation	
Obstructive sleep apnea	
Preeclampsia/eclampsia	
Neurogenic	Psychogenic, diencephalic syndrome, familial dysautonomia, polyneuritis (acute porphyria, lead poisoning), acute increased intracranial pressure, acute spinal cord section
Miscellaneous endocrine	Hypothyroidism, hyperthyroidism, hypercalcemia, acromegaly
Medications	High-dose estrogens, adrenal steroids, decongestants, appetite suppressants, cyclosporine, tricyclic antidepressants, monoamine oxidase inhibitors, erythropoietin, nonsteroidal anti-inflammatory agents, cocaine

NATURAL HISTORY OF UNTREATED HYPERTENSION:

Hypertensive disease can increase the risk of atherosclerotic vascular disease. The resultant increased pulse pressures have been generally answered to be the best prognostic marker of cardiovascular risk. In any case, an investigation of information from one million individual in 61 prospective studies found that, the SBP is somewhat more enlightening than DBP for foreseeing mortality from both stroke and coronary artery disease, and that pulse pressure is significantly less informative[9].

`

SYMPTOMS AND SIGNS:

In many cases the uncomplicated hypertension is quite often asymptomatic, with the goal that patient might be not aware of the subsequent progressive cardiovascular damage for up to 10 to 20 years.

Basic manifestations found in hypertensions are, headache, tinnitus, dizziness and fainting may be observed. Numerous manifestations inferable from the raised BP are psychogenic in cause, frequently reflecting hyperventilation instigated by anxiety[10].

Most common symptoms seen in hypertension is the headaches which is localized to the occipital region and are present when the patient wake up in the early morning but subsides spontaneously after several hours. Other complaints may also be present dizziness, easy fatigability, palpitations, and impotence.

Grievances referable to vascular illness incorporate epistaxis, haematuria, diminution of vision secondary to retinal pathology, scenes of dizziness because of transient cerebral ischemia, dyspnoea and angina pectoris because of cardiac failure . Dissection of aorta is an uncommon and rare side effect.

Secondary hypertension can be presented as a diabetes mellitus includes (polyuria, polydipsia), hypokalaemia secondary to primary aldosteronism causes muscle weakness and periodic paralysis, excessive fat deposition, skin changes, weight gain, and emotional lability seen in patients with Cushing’s syndrome. Patients with pheochromocytoma (catecholamine-secreting tumours) may associated with the episodic headaches, diaphoresis, palpitations and postural dizziness[11].

`

ASSOCIATION OF HYPERTENSION WITH OTHER CONDITIONS:

1. Obesity

There is association present between the hypertension and obesity ,(body mass index (BMI) more than 30 kg/m^2) , and obesity increases risk of ischemic heart disease, especially in central obesity patient Metabolic syndrome is leading and important cause of ischemic heart disease associated with hypertension.

In the study called Framingham the occurrence of hypertension was increases in 46% men and 75% in female who are overweight characterized as a BMI of 25.0 to 29.9 contrasted with ordinary weight persons[12].

2. Alcohol Intake

Alcohol in large quantity may increases the blood pressure, by mechanism such as arterial stiffness and finally atherosclerosis changes. A larger amounts of alcohol intake causes an increase in cardiac output and heart rate, possibly a consequence of increased sympathetic activity.

2. Physical Inactivity

Physical fitness can help forestall hypertension and people who are as of now hypertensive can bring down their BP by methods for high-impact work out. The relationship may include a reclamation age related decreases in endothelium subordinate vasodilatation[13].

4. Smoking

Smoking contains Nicotine which cause release of neurotransmitter from adrenergic nerve endings and increases sympathetic activity and increases the blood pressure. It also reduces the arterial compliance, and it is an independent risk for increase in blood pressure.

`

5. Sleep Apnea

Relief of sleep apnea may reduces the hypertension[14].

6. Hematological Findings:

Higher haematocrits are associated with increase in risk of hypertension and abnormal left ventricular filling on echocardiography[15].

7. Hypercholesterolemia:

Hypercholesterolemia frequently associated with hypertension at least in part because it causes endothelium dependent vasodilatation. Lipid lowering therapy like statins re-establishes the of nitric oxide, and reduces arterial stiffness and reduces the BP[16].

8. Hyperuricemia

Elevated serum uric acid concentrations is commonly seen in primary hypertension cases,

The hyperuricemia if not treated in time may associated with the reduction in renal blood flow and leads to hypertensive nephrosclerosis. In any case, antihypertensive medication regimens, US National Health and Nutrition Survey (NHANES III) demonstrated that raised serum uric acid was an independent risk factor for hypertension- associated morbidity and mortality.

Hypertensive people with elevated serum uric acid had associated with higher relative risk (RR) for both ischemic heart disease and stroke[17].

The renal complication associated with raised of serum uric acid may provide evidence to why hypertension-associated morbidity is closely linked to serum uric acid. It is well established that serum uric acid increases as arterial blood pressure rises and is associated with a reduction in renal blood flow.

`

High serum uric acid may cause increase renal reabsorption of sodium and causes hypervolemia and increase in blood pressure.

Hyperuricaemia may represent a multimetabolic syndrome in which insulin mediated renal haemodynamic abnormalities lead to hypertensive renal damage[18].

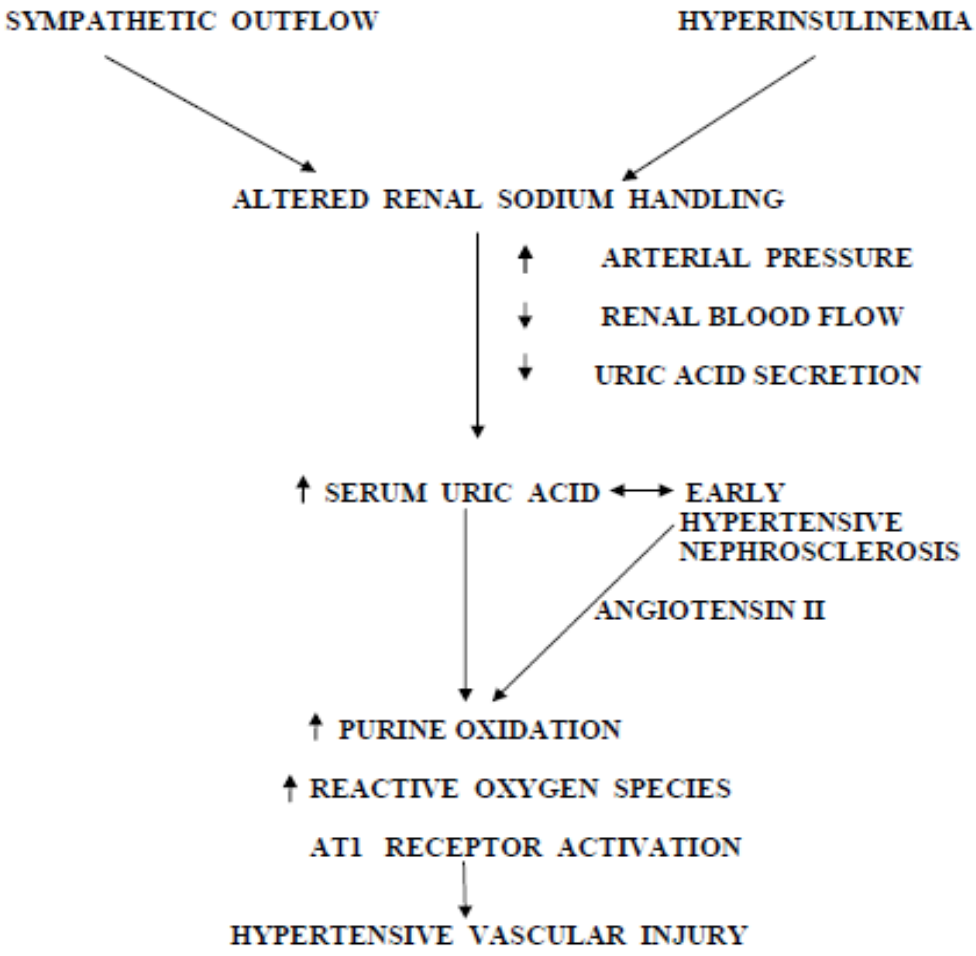

Figure 3 - Interaction between renal pathophysiology of hypertension and uric acid biochemistry

URIC ACID METABOLISM

Urates being the ionized types of uric acid are found conspicuously in significant amount in plasma ECF and synovial fluid, where it exists as monosodium urate at pH 7.4. Uric acid has likewise a significant impact on the pH of urine. Urate is

`

produced in tissues that contains xanthine oxidase fundamentally small intestine and liver.

Urate production depending on the purine content of person's diet, it's biosynthesis, degradation and salvage. Urate excretion takes place by kidney (around two third to three fourth) remaining by intestine. Urate is cleared from kidneys and thus physiological balance is being maintained by using specific organic anion transporters including urate transporter 1 (URATI) and human uric acid transporter (hUAT). URAT1 and other organic anion transporters (OATs), do help in carrying urate into tubular cells from apical side of lumen.

The methods of renal handling of urate /uric acid includes, glomerular filtration, secretion, tubular reabsorption, post secretory reabsorption urate transporter 1 (URATI) is present at the apical brush border of the proximal nephron. So Uric acid compounds directly inhibit URAT1 on the renal tubular cell which is called as a (so-called cis inhibition)[8].

The total-body urate pool is the net result between urate production and excretion.

Dietary intake of purine and novo-biosynthesis of purine from non purine precursors influences urate production thereby salvaging phosphoribosyl by urinary and intestinal routes.

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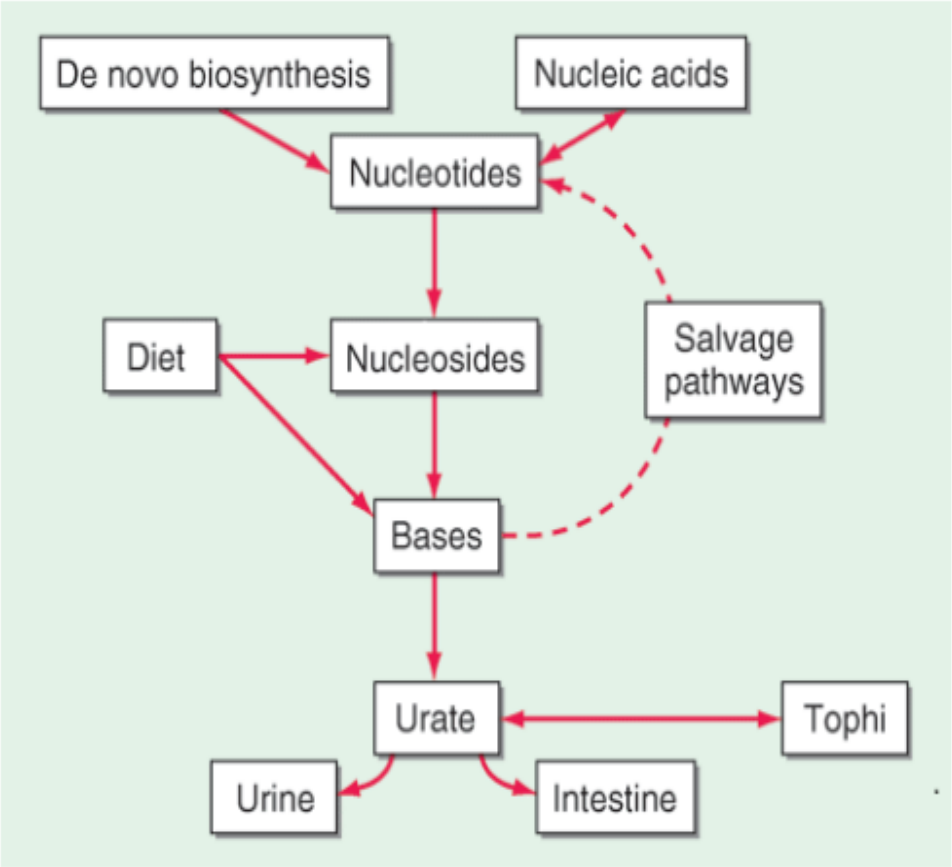

Fig.4-URIC ACID TURNOVER AND METABOLISM

HYPERURICEMIA

Hyperuricemia is defined as a plasma or serum uric acid level more than 420 umol/L or (7.0 mg/dL). In epidemiologic studies, hyperuricemia is defined as the mean plus 2 standard deviations of values determined from a randomly selected healthy population. When measured in non selected participant, 95% have serum urate concentrations less than 420 umol/L (7.0 mg/dL).

Finally, hyperuricemia can be defined in relation to the risk of disease. when urate levels >420 umol/L (7.0 mg/dL) the risk of developing gouty arthritis or urolithiasis increases and escalates in proportion to the degree of elevation.

`

Hyperuricemia is present in between 2.0 and 13.2% of ambulatory adults and somewhat more frequently in hospitalized individuals[19].

Causes of Hyperuricemia

Hyperuricemia may be classified as primary and secondary depending on whether the cause is innate or is the result of an acquired disorder. However it is useful to classify hyperuricemia in relation to the underlying pathophysiology, i.e., whether it results from increased urate production, decreased excretion, or a combination of two.

Classification of Hyperuricemia –

There are three mechanism to cause hyperurecemia

1) Urate overproduction
2) Decreased Uric acid Excretion
3) Combined Mechanism

1) Urate overproduction -

a. Primary idiopathic

b. Phosphoribosyl pyrophosphate (PRPP) synthetase overactivity

c. Hemolytic process

d. Polycythemia vera

e. Hypoxanthine phosphor ribosyl transferase (HPRT) deficiency

f. Rhabdomyolysis

g. Exersice and alcohol

`

2) Decreased Uric acid Excretion

a. Primary idiopathic

b. Renal disorder includes Polycystic kidney disease

c. Diabetic insipidus, diabetic ketoacidosis and hypertension.

d. chronic infection like Berylliosis and Sarcoidosis

e. Hyperparathyroidism and Hypothyroidism.

f. Bartter's syndrome, Down syndrome

g. Drug ingestion like Salicylates (>2g/d) and Diuretics

3) Combined Mechanism

a. Glucose-6- phosphatase deficiency

b. Alcohol

c. Fructose-1- phosphate aldolase deficiency

d. Shock

`

INCREASED URATE PRODUCTION

An exogenous source of purines is being provided by diet which accordingly further contributes to serum urate in proportion to it's purine content. The serum urate level can be maintained by restriction of purine rich diet, so with this we can reduces mean serum uric acid levels by about 60umol and urinary urate excretion by approximately 1.2mmol. .

Synthesis of purine nucleotide,

Synthesis of purine nucleotide by two pathways, Denovo purine synthesis which results in formation of purine ring from nonring structures in 11 step process which helps in formation of inosine monophosphate (IMP).

Initial step joins phosphoribosyl pyrophosphate (PRPP) and glutamine and is catalyzed by amido phosphoribosyl transferase (amido PRT). The rate of purine biosynthesis and urate creation are resolved, generally, by this enzymes.

AmidoPRT is managed by the substrate PRPP, which drives the response forward, and by the final results of biosynthesis (IMP and different ribonucleotides), which give input restraint.

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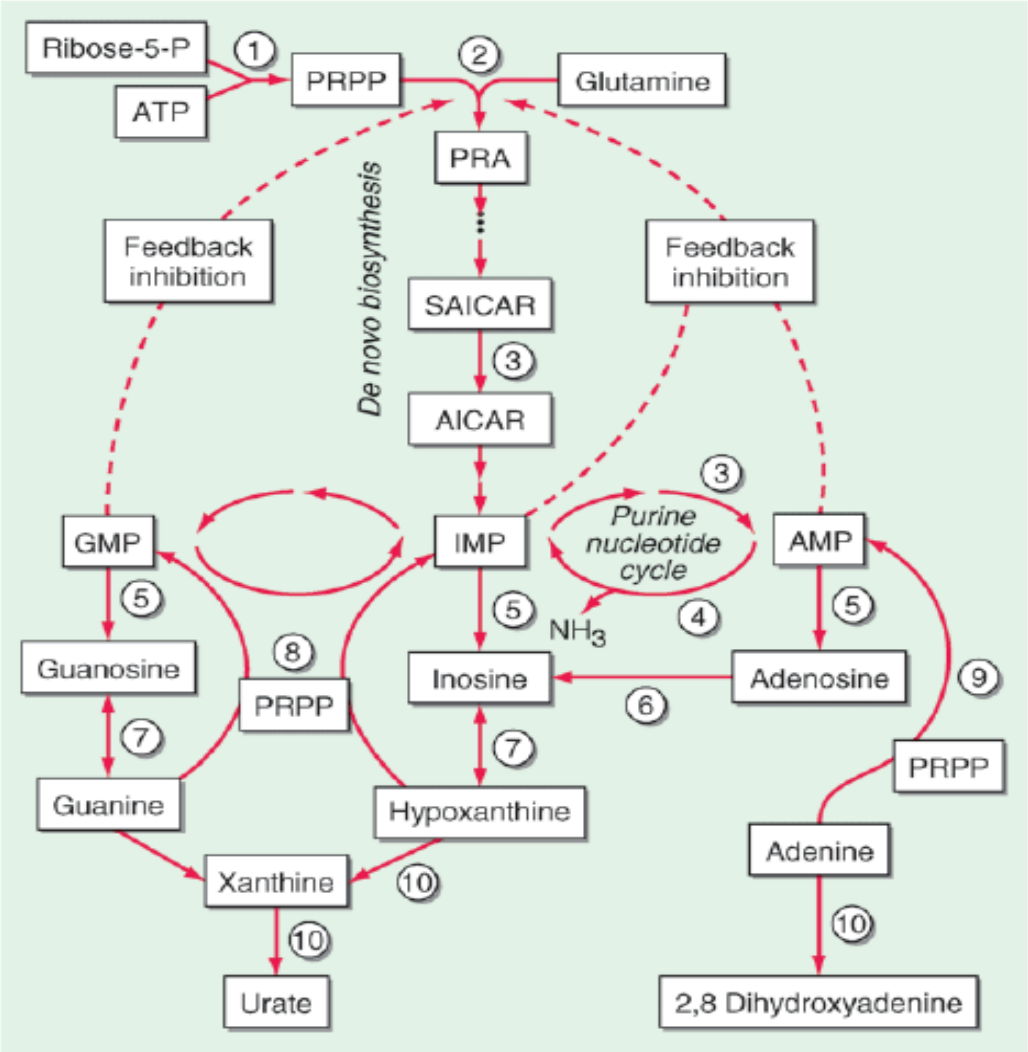

Fig.5- De novo biosynthesis and metabolism of Purine nucleotides

1. Phosphoribosyl pyrophosphate (PRPP) synthetase
2. Amido phosphoribosyl transferase (amidoPRT)
3. Adenyl succinate lyase
4. (myo-)adenylate (AMP) deaminase
5. 5'-nucleotidase
6. Adenosine deaminase
7. Purine nucleoside phosphorylase
8. Hypoxanthine phosphor ribosyl transferase (HPRT)
9. Adenine phosphoribosyl transferase (APRT)

`

10. Xanthine oxidase

A secondary regulatory pathway for purine synthesis is the salvage pathway in it purine bases by hypoxanthine phosphoribosyl transferase (HPRT). HPRT catalyzes the combination of the purine bases hypoxanthine and guanine with PRPP to form the respective ribonucleotides IMP and guanosine monophosphate (GMP). Increased salvage activity thus retards de novo synthesis by reducing PRPP levels and increasing concentrations of inhibitory ribonucleotides[31].

If there is increased PRPP synthetase activity and HPRT deficiency then it is associated with increased production of purines, hyperuricemia, and hyperuricaciduria.

Decreased Uric Acid Excretion

Uric acid excretion is affected theoretically by result from decreased glomerular filtration, decreased tubular secretion, or enhanced tubular reabsorption. about 90% of individuals with sustained hyperuricemia have a defect in the renal excretion of uric acid. So patients having gout excrete approximately 40% less uric acid than individuals who do not having gout,

patients with hyperuricemia is associated with chronic renal disease, the correlation between serum creatinine, urea nitrogen, and urate concentration is poor.

COMPLICATIONS OF HYPERURICAEMIA -

Hyperuricaemia and Gout:

Gout is primary and most important complication.The complications associated with gout correlate with both the duration and severity of hyperuricemia.

`

Hyperuricaemia and Renal System:

It includes -

1. Renal stone-
2. Uric acid nephropathy, leads to renal failure which is reversible in nature in it there is deposition of large amounts of uric acid stones in the renal collecting ducts, pelvis, and ureters.

Increased SUA (Serum uric acid) levels in Hypertension

The mechanisms underlying the increase in SUA and its prognostic implications in patients with essential hypertension are still not completely understood. Uric acid, is a final product of purine metabolism, it bounds 5% to plasma proteins, is freely filtered at the glomerulus and around 99% reabsorbed in the proximal tubule, and again secreted by the distal tubule, and subjected to considerable post secretory reabsorption. Total Fractional secretion of uric acid is about 7% to 10%. A direct association exists between SUA and renal vascular resistance in subjects with essential hypertension[20].

Uric acid is also commonly associated with hypertension. It is present in 25% of untreated hypertensive subjects, in 50% of subjects taking diuretics, and in more than 75% of subjects with malignant hypertension.

The increase in serum uric acid in hypertension may be due to the decrease in renal blood flow that accompanies the hypertensive state, since a low renal blood flow will stimulate urate reabsorption.

Hypertension causes microvascular disease, and this can lead to local tissue Ischemia[21]. In addition to the release of lactate that blocks urate secretion in the proximal tubule, ischemia also results in increased uric acid synthesis. With ischemia, adenosine triphosphate (ATP) is degraded to adenine and xanthine, and there is also increased generation of xanthine oxidase. The substrate (xanthine) and enzyme

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(xanthine oxidase) will be available to increase uric acid generation as well as oxidant (O2 -) formation. The finding that ischemia results in an increase in uric acid levels. may also explain why uric acid level increased in preeclampsia and congestive heart failure[22].

Other factors may also contribute to why uric acid is associated with hypertension, including alcohol abuse, lead intoxication, obesity and insulin resistance and diuretic use.

The observation that an elevated uric acid is associated with subjects at cardiovascular risk may account for why hyperuricemia predicts the development of cardiovascular disease in the general population, in subjects with hypertension and in subjects with pre-existing cardiovascular disease.

Hyperuricemia also predicts stroke in diabetic and nondiabetic subjects and predicts the development of hypertension and renal disease in the general population[23,24]. Also, hyperuricemia is a novel, independent risk factor for heart failure[25].

Hyperuricaemia and Syndrome X:

Syndrome X or metabolic syndrome is characterized obesity, impaired glucose tolerance leads to hyper insulinaemia and type 2 diabetes mellitus, raised triglyceride level, increased low density lipoprotein cholesterol, decreased high density lipoprotein cholesterol, and raised uric acid.

hyperuricaemia resulting from euglycaemic hyper insulinaemia may causes the onset of type 2 diabetes, hypertension, coronary artery disease, and gout in individuals with syndrome X[19].

`

HISTORY OF URIC ACID AND HYPERTENSION

Uric acid is involved in the pathogenesis of hypertension is a fact that has been known since ages. This was furthermore established when a paper reported in 1879 stated that almost all the subjects with hypertension reported association with gout, hence proving that uric acid may be important in the development of essential hypertension[26] About ten years later, this hypothesis reappeared when Haig[27] proposed low-purine diets helps in prevention of hypertension and vascular disease. In 1909, the French academician Henri Huchard noticed that renal arteriolosclerosis was seen in three groups. Those with gout, a individuals who have a diet enriched with fatty meat and those with lead poisoning[28].

The proposal of an association between serum uric acid and systemic hypertension has been emphasised on repeatedly between 1950 to 1980, but sadly has not received the attention it deserves due to the absence of mechanistic reasoning[29-31].

Hyperuricemia has been described in 25-40 percent of cases with hypertension with the number only increasing with the consideration of the upper limit[7,8]. This association is as severe as 70 percent in pre-eclamptic women[32] n.

As the patient ages and the duration of onset of hypertension increases, the solidity of the association between uric acid levels and hypertension reduces, thereby suggesting the importance of this topic in the younger population[31].

Many Cross-sectional studies have been noted that untreated hypertension is associated with elevated serum uric acid level [33,34] and Serum UA levels have also been associated cross-sectionally with raised blood pressure[31,35,36] and longitudinally with hypertension incidence [37--41] and future increases in blood presuure[42].

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REMNANT KIDNEY MODEL

Several studies have demonstrated the effect of uric acid on renal injury, one such stated being the kidney model which stated that the hyperuricemic rats, which are made so by induction of 2 percent oxinic acid to the diet show higher incidence of blood pressure, proteinuria and creatinine[43,44]. Oxinic acid when added to cyclosporine as a part of the treatment schedule produced high uric acid levels, excessive hyalinisation of arteries, infiltration of macrophages and renal parenchymal damage in contrast to rats treated with cyclosporine alone[45]. Studies also essentialised the addition of allopurinol in liver transplant patients to improve renal functioning[46.47].

Mild hyperuricemia in the Rat - an animal model for essential hypertension

The concept of mild hyperuricemia had emerged but was also silenced due the requirement of an animal model for a better understanding of this concept. Oxinic acid, a pharmacologic inhibitor of urate oxidase was put to use to emphasize on the importance of sustained mild hyperuricemia. All of the performed studies proved in unison that the uric acid mediated hypertension was due to inflammation in the renal tissue, thereby activating the renin angiotensin system and deleterious effect on nitric acid production[48].

The study of URAT1, a urate transport channel present on the smooth muscle cells in humans was established by the northern and western analysis stated that HVSMC took up the C- urate, a process which is inhibited by probenecid, increases the uric acid mediated proliferation in a dose dependant manner[49].

RECENT EPIDEMIOLOGY: A CHANGE IN PERSPECTIVE

It was only after the year 1990 that the observation of valuating the level of uric acid was made and seen that 25 to 40 percent adults have serum uric acid concentration

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more than 6.5mg/dl and about 60 percent of them have concentration of 5.5mg/dl, proving an unilinear association between serum uric acid and hypertension[50].

There are Three reports which were published in 1990s showed that serum uric acid is an independent risk factor for hypertension [41,50,51] and a lot more were distributed in the previous 10 yrs[52-54].

Author	Year published	Study size	Follow-up	Relative Risk
Kahn et al.(27)	1972	10000 males	5 Years	2 - fold risk
Selby et al.(26)	1990	2062 adults	6 Years	3 - fold risk
Hunt et al.(29)	1991	1482 adults	7 Years	2 - fold risk
Jossa et al.(25)	1994	619 males	12 Years	2 - fold risk
Taniguchi et al.(42)	2001	6356 males	10 Years	2 - fold risk
Masuo et al.(30)	2003	433 males	5 Years	+27 mmHg in systolic BP per each 1-mg/dL change in uric acid
Nakanishi et al.(28)	2003	2310 males	6 Years	1.6 - fold risk
Nagahama et al.(49)	2004	4489 adults	13 Years	1.7 - fold risk
Alper et al.(43)	2005	577 children	11 Years	Increased risk
Sundstrom et al.(44)	2005	3329 adults	4 Years	1.6 fold risk

Table.3-Studies on Relative Risk for Hypertension in Hyperuricaemia

SIR BRADFORD HILL'S CRITERIA FOR URIC ACID AS A CAUSAL FACTOR IN HYPERTENSION[55]

1. **Strength**. Studies have demonstrated doubling of the incidence of hypertension in subjects with elevated uric acid levels in a period of 5 to 10 years[56-64].
2. **Specificity**. The risk for developing hypertension with an elevated uric acid level remains after controlling for other cardiovascular risk factors[57-64]. New onset essential hypertension in adolescents is also associated with an elevated

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uric acid (UA ≥ 5.5 mg/dL) in the vast majority (about 90%) of cases; whereas it is present in only 30% of secondary hypertension and is rare in normotensive and white-coat hypertensive adolescent subjects[65] .

3. **Consistency**. An elevated uric acid was found in all 9 studies to be predictive for hypertension[56-64].
4. **Dose-dependent**. The relationship of serum uric acid to future hypertension is continuous and dose-dependent[56-64].
5. **Temporality**. An elevated uric acid often precedes the development of hypertension, both in children and in adults[56-64].
6. **Plausibility.** A biological mechanism has been found by which raising uric acid may cause hypertension[66-69].
7. **Coherence.** The increase in hypertension frequency with Westernized diets corresponds to the rise in the frequency of hyperuricemia and gout[70].
8. **Experiment**. Experimental hyperuricemia causes hypertension in rats; preventing the rise in uric acid prevents the development of hypertension[66-68].

`

Koch's Postulates for Uric Acid as a Causal Factor of Hypertension

1. Raising serum uric acid in rats results in the hemodynamic, pathological, and clinical characteristics of essential hypertension in humans[66-68].
2. A plausible biological mechanism has been shown in which uric acid induces a salt-resistant hypertension by inhibition of endothelial function and activation of the renin-angiotensin system, and a later salt-sensitive renal dependent hypertension by inducing microvascular disease[66-68],these changes are consistent with studies of hypertension in humans[69].
3. Elevated uric acid is a definitive predictor for the development of hypertension[56-63].
4. Around 89 percent of novel hypertensives are associated with hyperuricemia and the level of uric acid matches closely with the systolic blood pressure[65].
5. Lowering uric acid in hyperuricemic rats prevents or treats new onset hypertension in rats[66]. Pilot studies in humans also suggest lowering uric acid may lower BP in new onset essential hypertension in adolescents[71].

Uric acid and essential hypertension in children

Like adult children's study also stated association between the uric acid and hypertension, The Moscow Children's Hypertension study found that uric acid level greater than 8.0 mg/dl in 9.5% of children with normal blood pressure, 49% of children with borderline hypertension, and 73% of children with moderate and severe hypertension[72].

The Hungarian Children's Health Study showed that total 17,624 children were followed for 13yrs and are found that risk factors for the development of hypertension were raised heart rate, early sexual maturity and hyperuricemia[73].

`

Gruskin stated that the hypertensive children had both elevated serum uric acid (mean greater than 6.5 mg/dl) and higher peripheral renin activity[74].

PROPOSED MECHANISM FOR URIC ACID MEDIATE HYPERTENSION IN HUMANS

The incidence of chronic hyperuricemia is due to increase in dietary uptake of fructose, purine rich meats, or low doses of lead [75].

Uric acid levels may be high in mothers either due to excessive dietary intake or due to illnesses like obesity, pre-eclampsia, pre-existing hypertension, which in turn leads to excessive uric acid in the baby producing teratogenic effects like IUGR, and a reduction in nephron number. On the other hand, children develop hyperuricemia as a consequence of genetic and environmental factors.

Persistent prolonged renal vasoconstriction leads to the development of salt sensitive hypertension despite correction of hyperuricemia, further stating that chronic disease will be resistant to uricosuric therapy with the hypertension being salt sensitive hence making it a challenge to treat.

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MATERIALS AND METHODS

The study "ESTIMATION OF SERUM URIC ACID LEVEL IN NEWLY DIAGNOSED ESSENTIAL HYPERTENSION" was conducted in Shri B M Patil Medical college Vijayapura, during the period from November 2018 to June 2020.

AIM-

The study was done with the following aim.-

1) To study the association of serum uric acid level with newly diagnosed essential hypertension.

Selection of cases: The study was done in patients admitted and diagnosed with essential hypertension" in Shri B M Patil Medical college Vijayapura

Period of Study: From November 2018 to June 2020.

Sample Size:

With 95% confidence level and margin of error of ±7.5%, a sample size of 92 subjects will allow the study to determine the "Estimation of serum uric acid level in newly diagnosed essential hypertension**"** with finite population correction.

By using the formula:

$$n = \frac{z^2 p(1-p)}{d^2}$$

where,

Z= z statistic at 5% level of significance

d is margin of error

p is anticipated prevalence

`

Study subjects:

A sample size of 93 subjects will allow the study to determine the Estimation of serum uric acid level in newly diagnosed essential hypertension in Shri B M Patil medical college hospital and research center vijayapura.

INCLUSION CRITERIA:

1. All patients above 16 years of age with newly diagnosed essential hypertension of both the genders will be included in the study.

The diagnosis of essential hypertension will be established according to JNC 7 criteria[6].

- Systolic blood pressure equal to or greater than 140 mm Hg.
- Diastolic blood pressure equal to or greater than 90 mm Hg.

EXCLUSION CRITERIA:

- Secondary hypertension .
- Ischemic heart disease, congestive cardiac failure.
- Gout.
- Hypothyroidism and hyperthyroidism.
- Renal insufficiency, glomerulonephritis, pyelonephritis, hereditary nephropathy.
- Patients on Drugs – Levodopa, Ethambutol, Pyrazinamide,Cytotoxic drugs , Low dose aspirin, Thiazide diuretics.

Consent

The study groups selected by the above criteria (inclusion and exclusion) were first informed about the nature of the study. Patients who are willing for the study were selected after getting an informed and written consent.

Thus, a total of 93 patients were taken up for study who satisfied the inclusion and exclusion criteria.

`

There was no conflict of interest and financial support was Nil. Urinary excretion and urate clearance were not done, only serum uric acid levels were analysed.

Patient profile-

Selected socio-demographic, clinical and laboratory data were collected from the cases.

1. Socio-demographic profile

- Age.
- Sex.
- Past history of major diseases.
- family history
- Cardiovascular risk factors – smoking.

2. Clinical profile

- weight
- Height
- Body mass index
- Pulse
- Systolic and diastolic blood pressure – Average of 3 BP measurements
- Clinical examination

3.Laboratory Data

Laboratory analyses, performed in Biochemical laboratory at the Shri B M Patil Medical college Vijayapura, included,

- Complete blood count.
- Urine examination.
- Random blood sugar.
- Serum lipid profile.

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- Serum creatinine.
- Serum uric acid.
- Echocardigraphy (2D ECHO).
- ECG to find left ventricular hypertrophy.
- Fundus examination was done for all subjects to rule out Hypertensive Retinopathy.

Data collection and measurements:

The clinical examination consisted of a medical history, a physical examination, blood pressure measurement and anthropometric measurements. Laboratory data included measurement of fasting serum uric acid levels and other parameters like Blood haemogram, Renal function tests (blood urea, serum creatinine), Electrocardiogram, Chest X-ray, Lipid profile (Total cholesterol, triglycerides, HDL-cholesterol, LDL- cholesterol), urine for protein and sugar.

The patients were asked to not to eat for 12 hours and to avoid smoking and heavy physical exercise for more than 2 hours before the examinations. After entering into the examination room patients were asked to take rest for 5 min a quiet room, systolic and diastolic blood pressures were measured in the sitting position for two time at an interval of a five minutes on the right arm with a standard mercury sphygmomanometer on three separate occasions[76].

Anthropometric measurements included height and body weight, which were measured while the subject was wearing light clothing without shoes. The body mass index was calculated as the weight in kilograms divided by the height in m2.

`

Statistical analysis

All characteristics were summarized descriptively. For continuous variables, the summary statistics of mean±standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries and diagrammatic presentation. Chi-square (χ^2) test was used for association between two categorical variables. Comparison of quantitative data was done using MANN Whitney U test and F test.

HYPERTENSION

Hypertension was defined according to the JNC VIII classification of hypertension as those with SBP of < 120 mm hg and DBP of < 80 mm hg as normal, those with SBP of 120- 139 mm hg or DBP of 80 - 89 mm hg were labelled pre-hypertensive were not taken up for the study, those with SBP 140 - 159 mm hg or DBP of 90 - 99 mm hg were labelled as having Stage 1 hypertension, and those with SBP ≥ 160 mm Hg or DBP ≥ 100 mm hg were labelled as Stage 2 hypertension.

Method of Uric Acid estimation

Principle

The principle for the determination of Serum Uric Levels was devised by Trivedi and Kabasakalian with a modified Trinder peroxidase method using TBHB.

URIC ACID + O2 + H2O Uricase Allantoin + CO2 + H2O2

H2O2 + 4- AAP + TBHB Peroxidase Quinoneimine + H2O

The Sample used was unhemolyzed serum or plasma separated from the cells as soon as possible. Recommended anticoagulants are heparin and EDTA. Uric acid is stable in serum or urine for 3 days at 20 - 25o C. The intensity of chromogen (Quinoneimine) formed is proportional to the uric acid concentration in the sample

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when measured at 510 nm (510 -550nm) on biochromatic analysers against reagent blank.

Reference Values for SUA levels -

In Males : 3.4 - 7.0 mg/dL

In females : 2.4 - 6.0 mg/dL

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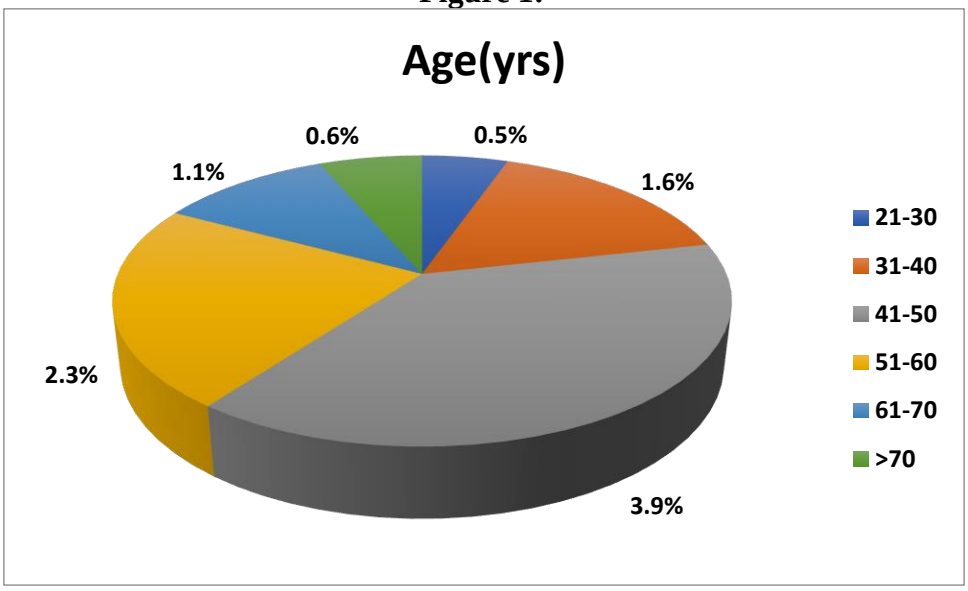
OBSERVATIONS AND RESULTS

This study was conducted in Shri B M Patil Medical College and Hospital, From November 2018 to June 2020, with 93 patients. It was a cross sectional study done in all the patients that satisfy the inclusion criteria.

Table 1: Distribution of Cases according to Age

Age(yrs)	N	Percent
21-30	5	5.4
31-40	15	16.1
41-50	36	38.7
51-60	21	22.6
61-70	10	10.8
>70	6	6.5
Total	93	100

Figure 1:



- As shown in table number 1 Among the total studied population around three fourth of individual's fall in 40 to 70years of age groups.
- Rest one fourth of population fall in extreme age group.

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Table 2:

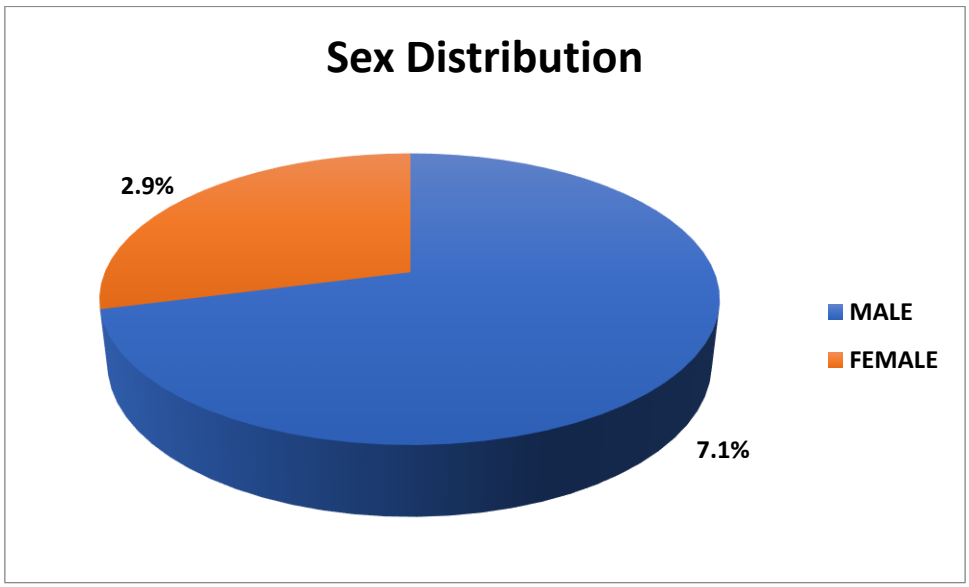
Descriptive Statistics	Min	Max	Mean	SD
Age(yrs)	25	86	51.5	12.3

- In the studied population minimum age found is 25 years, and maximum found is 86 year.
- Mean age group of this study is 51.5 years.

Table 3: Distribution of Cases according to Sex

Sex	N	Percent
Male	66	71
Female	27	29
Total	93	100

Figure 2: Distribution of Cases according to Sex



- Among the studied population, total number of male patients were 66 so it was 71% and total number of female patients were 27 (29%) of 93 patients.
- Male participant are more than female in this study.

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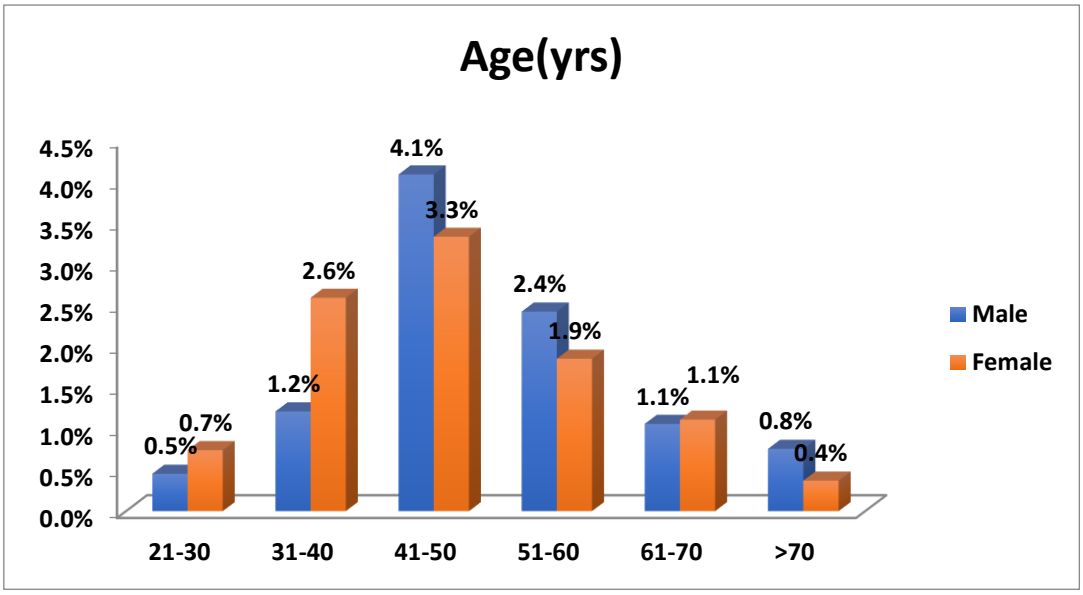
Table 4: Association of Age and Sex

Age(yrs)	Male		Female		p value
	N	%	N	%	
21-30	3	4.5%	2	7.4%	0.613
31-40	8	12.1%	7	25.9%	
41-50	27	40.9%	9	33.3%	
51-60	16	24.2%	5	18.5%	
61-70	7	10.6%	3	11.1%	
>70	5	7.6%	1	3.7%	
Total	66	100.0%	27	100.0%	

Figure 3: Association of Age and Sex



- The study had maximum number of males between the age group of 41 – 50 years, accounting to 27 (40.9%) in number and maximum number of female subjects were in the age group of 41 – 50 years, accounting to 9(33.3%) in number.

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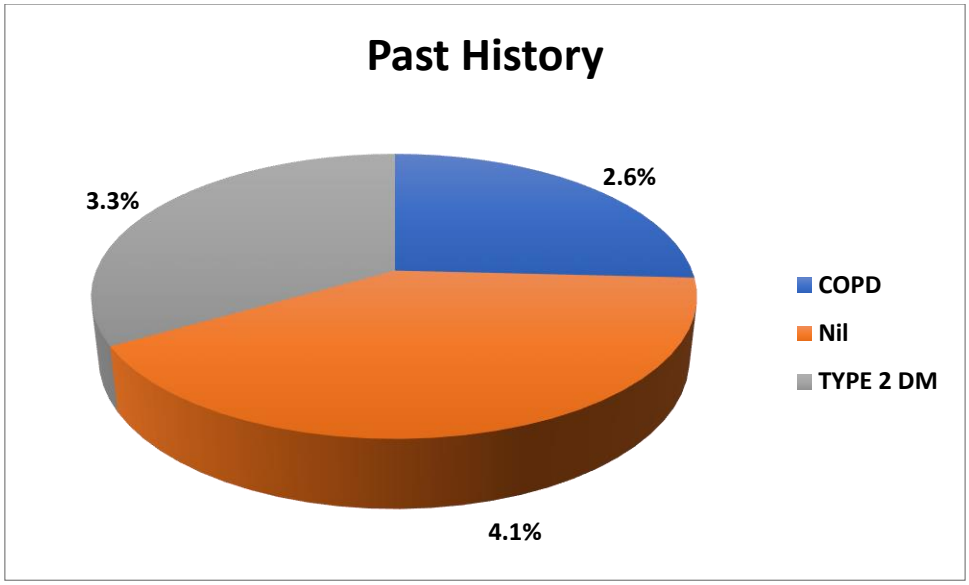
Table 5: Distribution of Cases according to Past History

Past History	N	Percent
COPD	24	25.8
Nil	38	40.9
TYPE 2 DM	31	33.3
Total	93	100

Figure 4: Distribution of Cases according to Past History



- In the study conducted, maximum number of subjects did not have any significant past history, noted in 38 individuals, while 31 patients had type II diabetes and 24 patients had COPD.

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Table 6: Descriptive Statistics of Study Variables

Descriptive Statistics	Min	Max	Mean	SD
SBP (mmHg)	140	200	163.66	14.25
DBP (mmHg)	90	110	93.83	5.272
SUA LEVEL (mg/dl)	3.10	9.70	6.24	1.38969

- In the studied population minimum systolic and diastolic blood pressures are 140 mmHg and 90 mmHg respectively, and maximum systolic and diastolic blood pressures are 200mmHg and 110mmHg. It was also observed that the minimum uric acid level in the study was 3.10mg/dl and the maximum uric acid level was 9.7mg/dl, giving a mean value of 6.24mg/dl.

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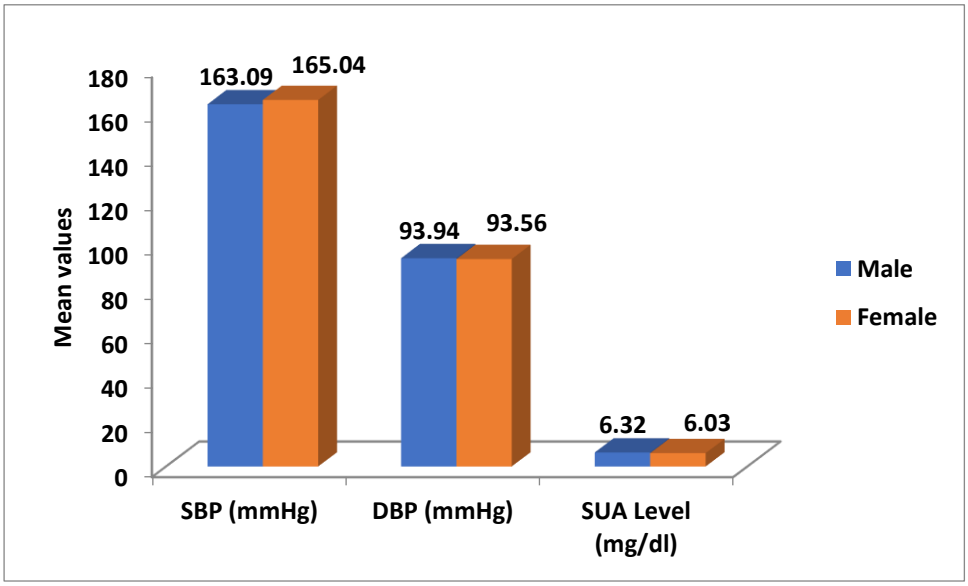
Table 7: Distribution of Study Variables according to Sex

Parameters	Male		Female		Mann Whitney U test	p value
	Mean	SD	Mean	SD		
SBP (mmHg)	163.09	14.5	165.04	14.0	U=833.0	0.6261
DBP (mmHg)	93.94	5.262	93.56	5.39	U=837.50	0.6511
SUA Level (mg/dl)	6.32	1.43	6.03	1.28	U=724.0	0.1588

Figure 5: Distribution of Study Variables according to Sex



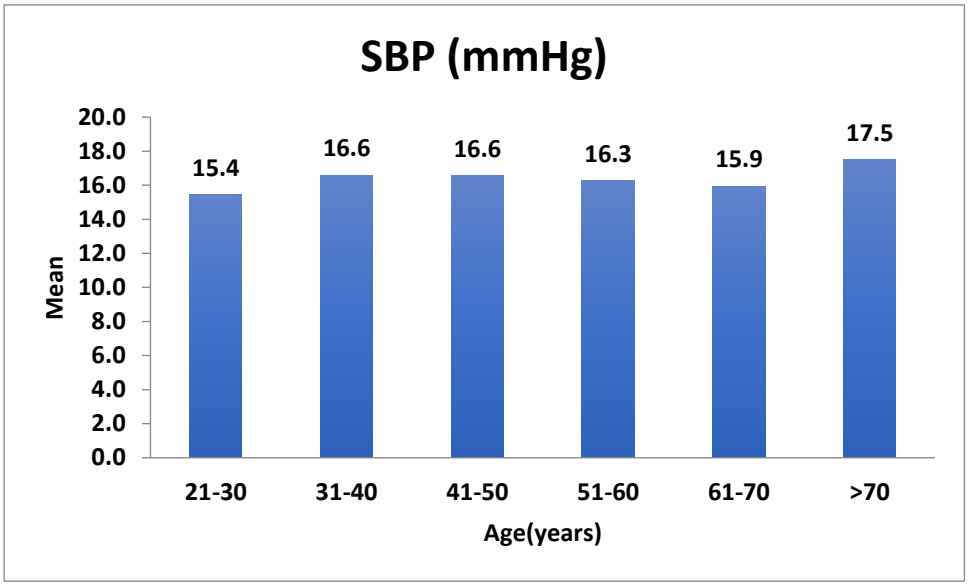
- Among the studied population in males, mean serum uric acid level is 6.32mg/dl with mean systolic blood pressure and diastolic blood pressure being 163.9mmHg and 93.94mmHg respectively.
- And in females, serum uric acid level is 6.03mg/dl with mean systolic blood pressure and diastolic blood pressure being 165mmHg and 93.56mmHg respectively.

`

Table 8: Mean SBP according to Age

Age(yrs)	SBP (mmHg)		p value
	Mean	SD	
21-30	154.4	20.3	0.173
31-40	165.7	16.0	
41-50	165.6	12.4	
51-60	162.6	12.7	
61-70	159.4	16.5	
>70	175.0	13.8	
Total	164.3	14.3	

Figure 6: Mean SBP according to Age



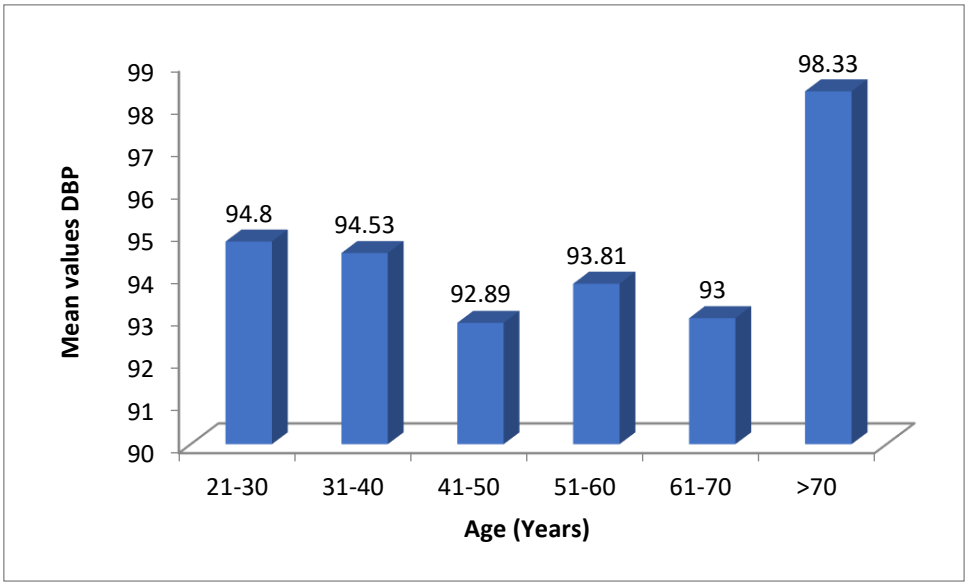
- Maximum Mean systolic blood pressure is 175mmHg seen in age greater than 70 years, with standard deviation 13.8, and the minimum mean systolic blood pressure was seen in the age group of 21-30 years, the value being 154.4mmHg.

`

Table 9: Mean DBP according to Age

Age(yrs)	DBP (mmHg)		F test p value
	Mean	SD	
21-30	94.80	5.020	F=1.259 P=0.289
31-40	94.53	4.565	
41-50	92.89	4.153	
51-60	93.81	5.896	
61-70	93.00	6.749	
>70	98.33	7.528	
Total	92.6	6.6	

Figure 7: Mean DBP according to Age



- Maximum mean diastolic blood pressure is 98.3mmHg seen in age greater than 70 years, and the minimum mean diastolic blood pressure is 92.89mmHg observed in the age group of 41-50years.

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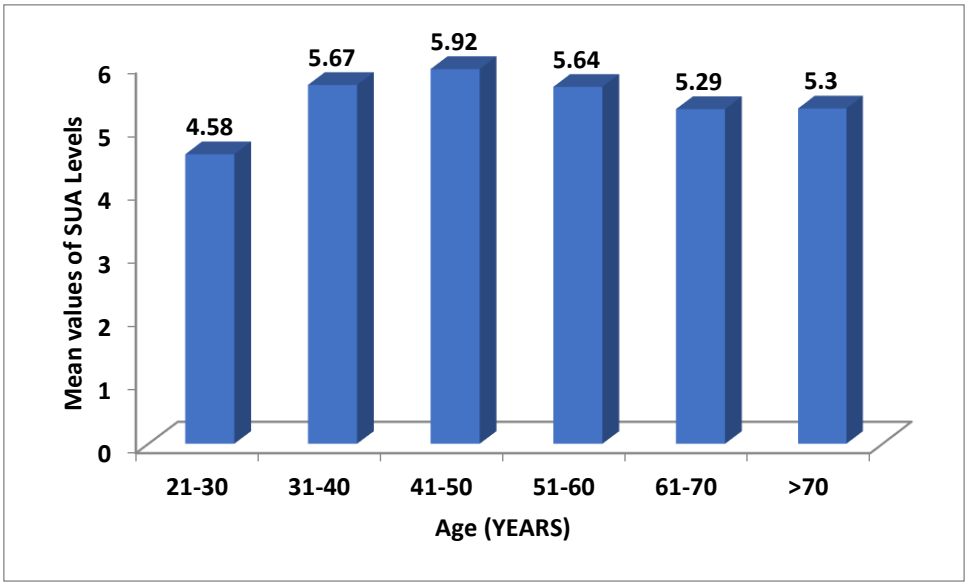
Table 10: Mean SUA according to Age

Age(yrs)	SUA Level (mg/dl)		F test p value
	Mean	SD	
21-30	4.58	2.382	F=0.657 P=0.657
31-40	5.67	2.080	
41-50	5.92	1.554	
51-60	5.64	1.733	
61-70	5.29	1.791	
>70	5.30	1.889	
Total	5.64	1.760	

Figure 8: Mean SUA according to Age



Maximum mean serum uric acid level in newly diagnosed essential hypertension cases was found to be 5.92 with standard deviation 1.6 seen in the age

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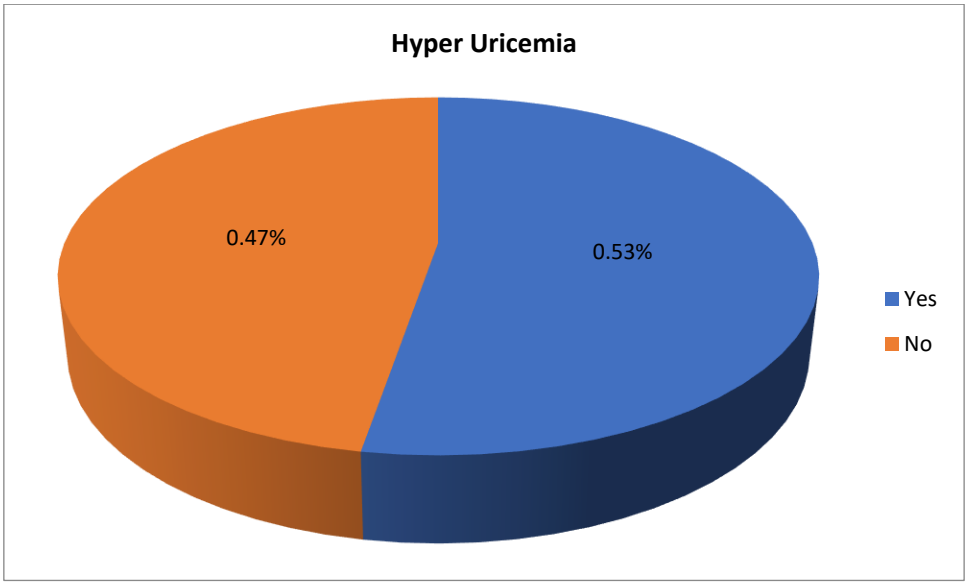
group 41-50, and minimum mean serum uric acid level was 4.58 seen in the age group of 21-30 years.

Table 11: Distribution of Cases according to Hyper Uricemia

Hyper Uricemia	N	Percent
Yes	49	52.7
No	44	47.3
Total	93	100

Figure 9: Distribution of Cases according to Hyper Uricemia



In this study total number of newly diagnosed essential hypertension patients was 93 in number out of which hyperuricemia was seen in 49(52.7%) patients while rest 44(47.3%) hypertensive patients showed normal uric acid level.

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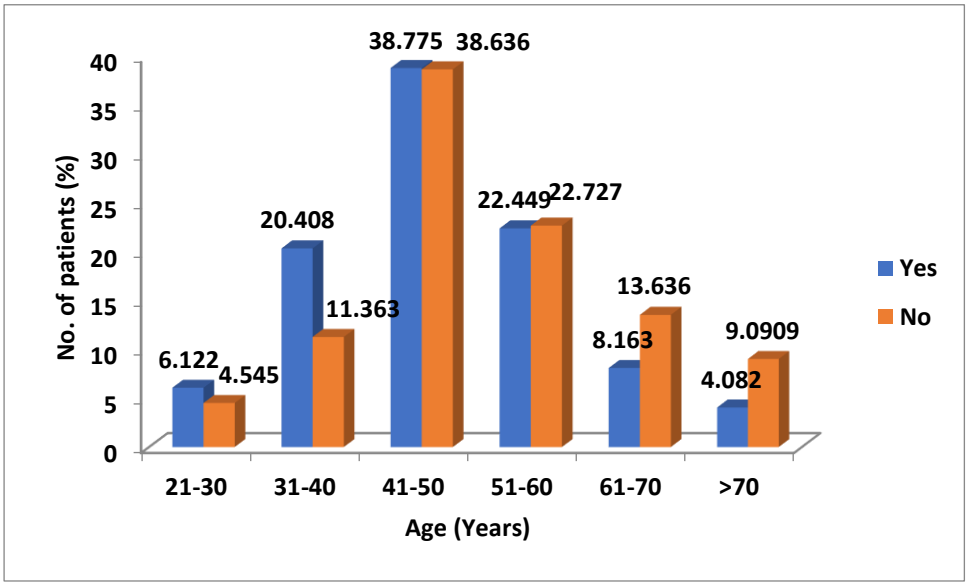
Table 12: Distribution of Age according to Hyper Uricemia

Age(yrs)	Hyper Uricemia				Chi square test p value
	Yes		No		
	N	%	N	%	
21-30	3	6.122	2	4.545	X^2=0.831 P=0.7260
31-40	10	20.408	5	11.363	
41-50	19	38.775	17	38.636	
51-60	11	22.449	10	22.727	
61-70	4	8.163	6	13.636	
>70	2	4.082	4	9.0909	
Total	49	100.0%	44	100.0%	

Figure 10: Distribution of Age according to Hyper Uricemia



In this study among maximum number of hyperuricemic patients were seen in the age group of 41-50 years, accounting to 38.7%. Among the normouricemic patients, the maximum number was in the age group of 41-50 years, accounting to 38.63%.

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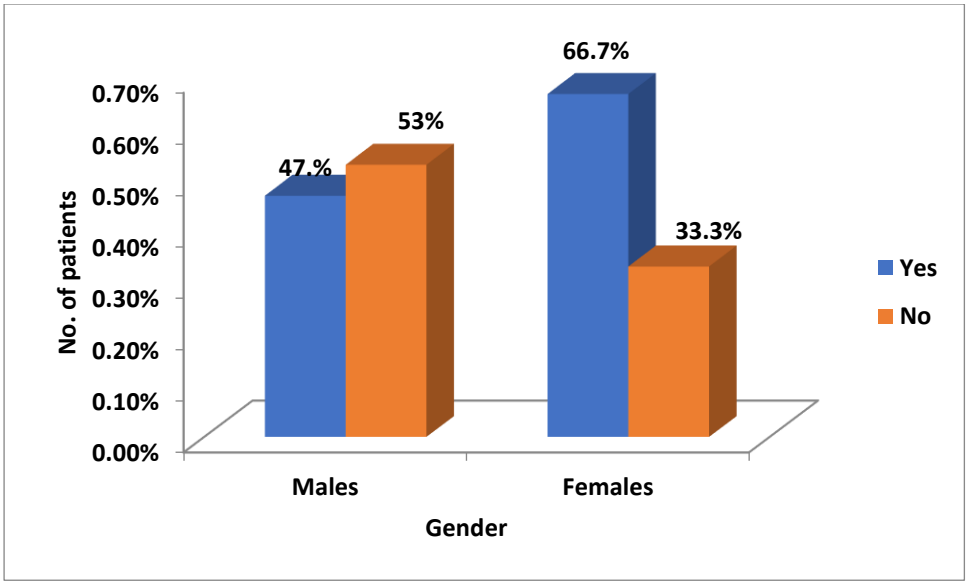
Table 13: Distribution of Sex according to Hyper Uricemia

Sex	Hyper Uricemia				Total	Chi square test p value
	Yes		No			
	N	%	N	%		
Male	31	47.0%	35	53%	66(100%)	X^2=2.982 0.0842
Female	18	66.7%	9	33.3%	27(100%)	
Total	49		44	100.0%		

Figure 11: Distribution of Sex according to Hyper Uricemia



Among the hyperuricemic patients, the male patients were 31 accounting to 47%, while the female patients were 18 in number, accounting to 66.7%.

Among the normouricemic patients, 35 patients were male and 9 were female.

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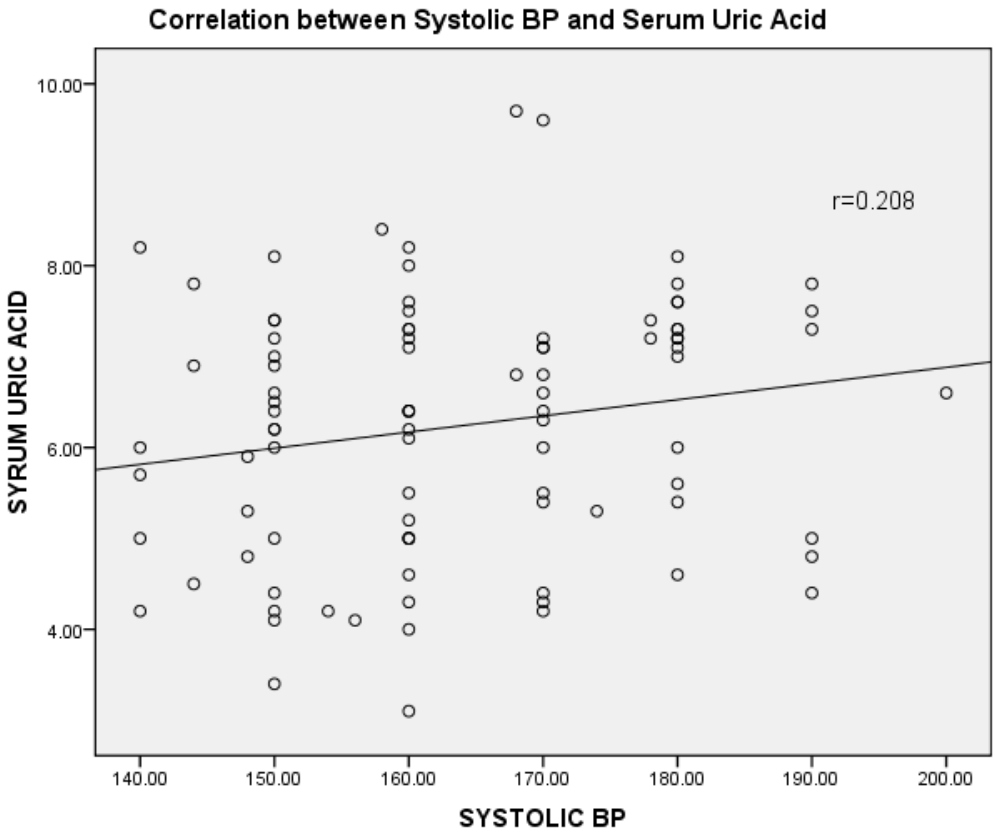
Table 14: Correlation between Systolic BP and Serum Uric Acid

Correlation between	Spearman's rho Correlation coefficient	P value	Remark
Systolic BP and Serum Uric Acid	r=0.208	P=0.047*	Mild positive correlation
*:Correlation is significant			

Figure:12



To assess the correlation between the above discussed parameters, the Spearman's correlation coefficient and p values were studied to find out the significance of such an association. The correlation between the systolic blood pressure and the serum uric acid showed significant mild positive correlation.

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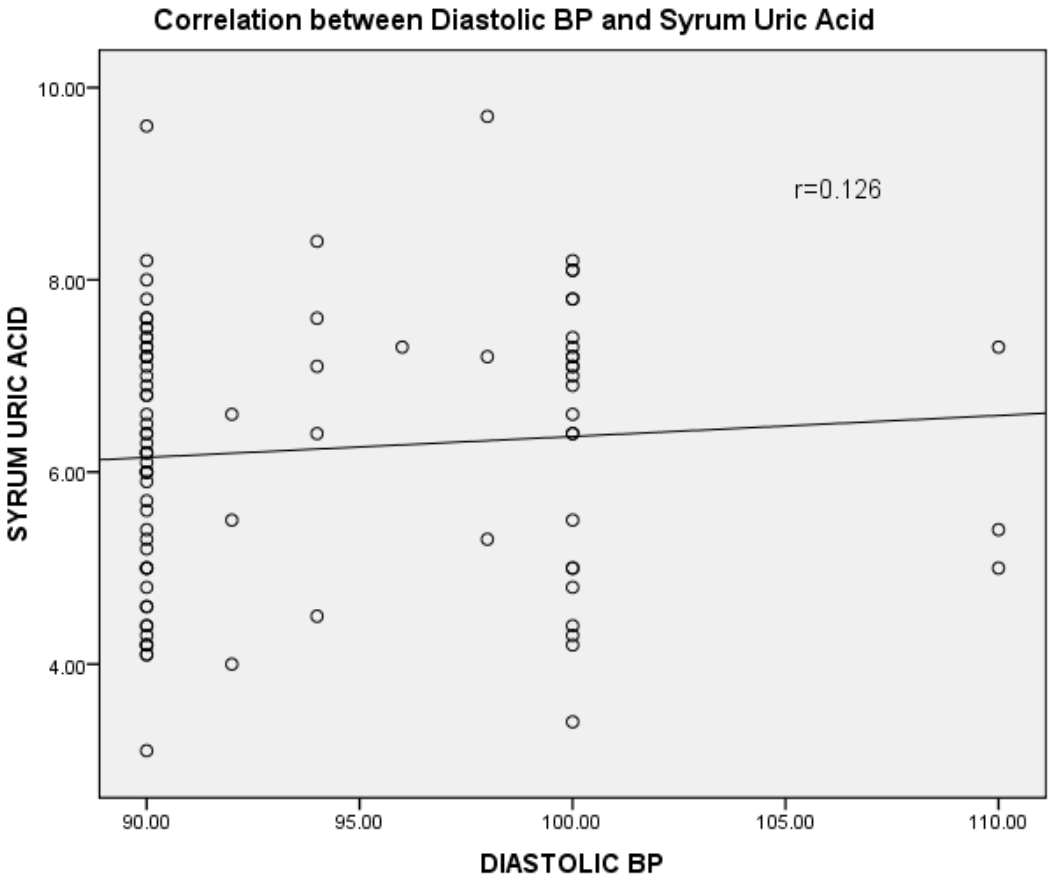
Table 15: Correlation between the Diastolic BP and Serum Uric Acid

Correlation between	Spearman's rho Correlation coefficient	P value	Remark
Systolic BP and Serum Uric Acid	r=0.126	P=0.268	Mild positive correlation
*:Correlation is Insignificant			

Figure: 13



In a similar manner the association between the serum uric acid and diastolic blood pressure was measured and showed a mild positive correlation.

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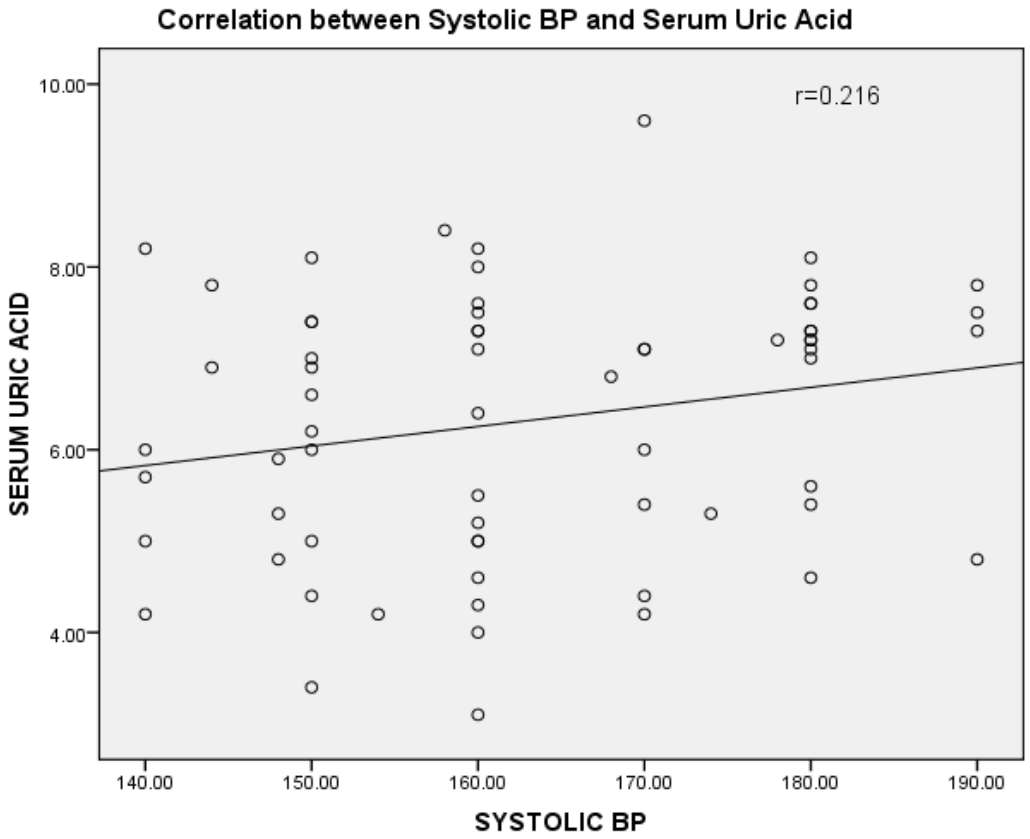
Table 16: Correlation between the Systolic BP and Serum Uric Acid of Males

Correlation between	Spearman's rho Correlation coefficient	P value	Remark
Systolic BP and Serum Uric Acid	r=0.216	P=0.096	Mild positive correlation
*:Correlation is Insignificant			

Figure: 14



The above displayed correlation shows that there is a mild positive correlation between the systolic blood pressure and the mean serum uric acid level among the male patients.

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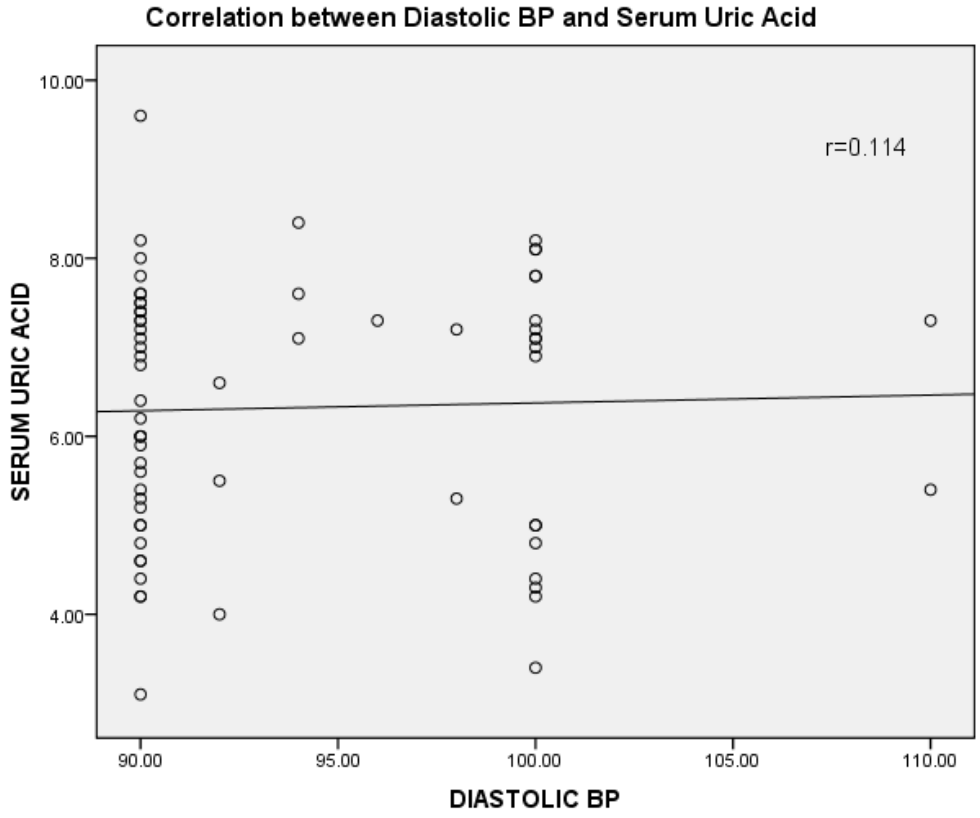
Table 17: Correlation between the Diastolic BP and Serum Uric Acid of Males

Correlation between (Males)	Spearman's rho Correlation coefficient	P value	Remark
Diastolic BP and Serum Uric Acid	r=0.114	P=0.096	Mild positive correlation
*:Correlation is Insignificant			

Figure:15



Similar to the above showed correlation, the association between the serum uric acid level and the diastolic blood pressure among males was studied and it also showed a mild positive correlation.

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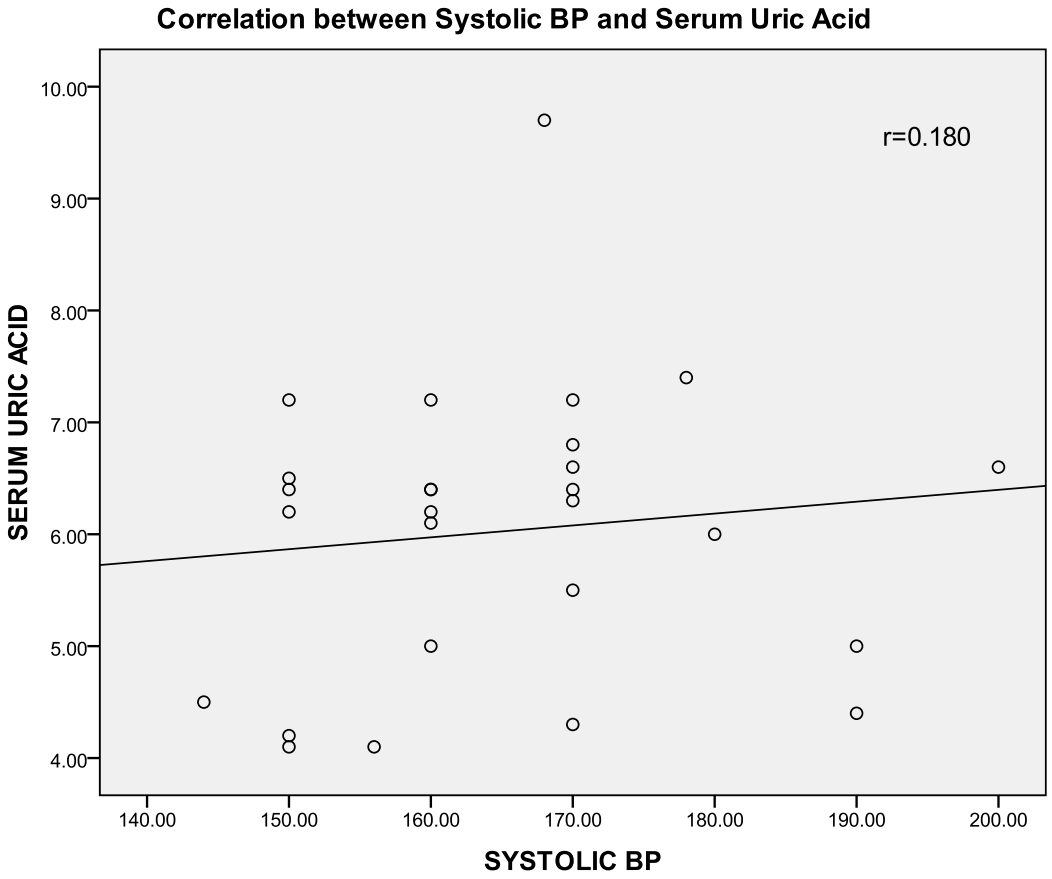
Table 18: Correlation between the Systolic BP and Serum Uric Acid of Females

Correlation between (Females)	Spearman's rho Correlation coefficient	P value	Remark
Systolic BP and Serum Uric Acid	r=0.180	P=0.370	Mild positive correlation
*:Correlation is Insignificant			

Figure: 16



The female patients were also studied for such similar correlations. A mild positive correlation was found between the serum uric acid and systolic blood pressure in females.

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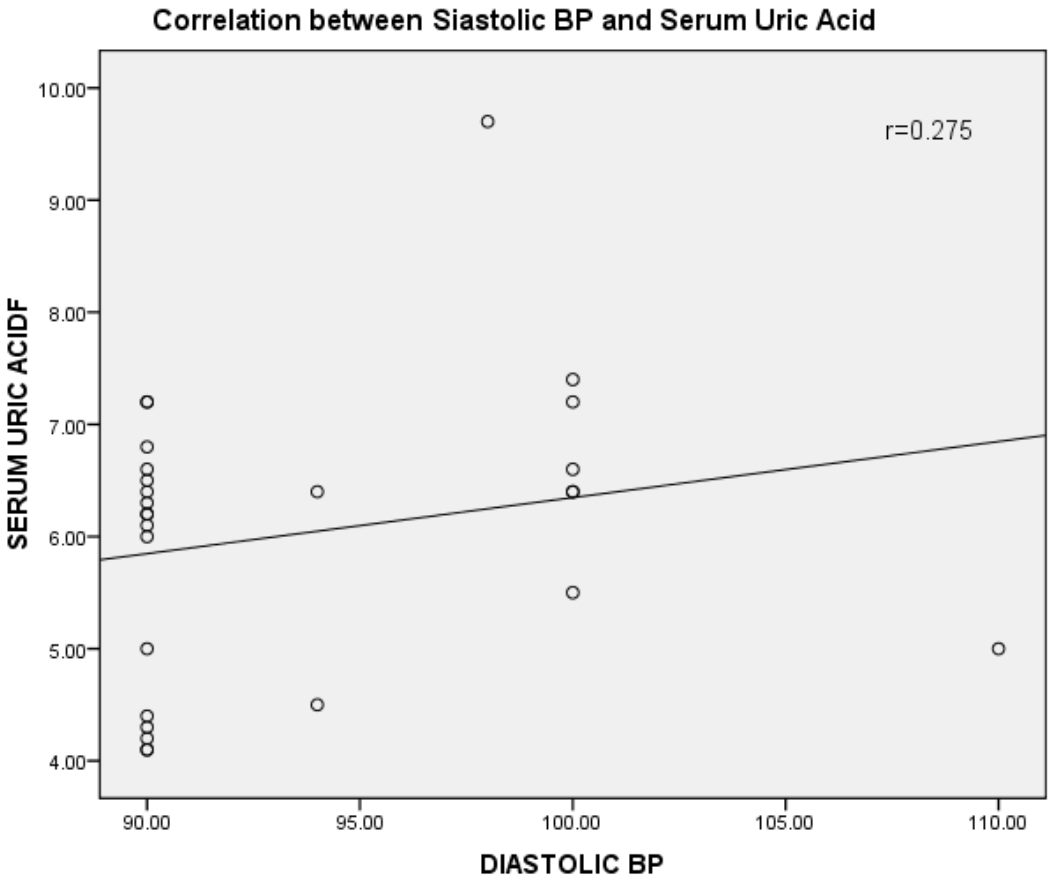
Table 19: Correlation between the Diastolic BP and Serum Uric Acid of Females

Correlation between (Females)	Spearman's rho Correlation coefficient	P value	Remark
Diastolic BP and Serum Uric Acid	r=0.275	P=0.164	Mild positive correlation
*:Correlation is Insignificant			

Figure: 17



The correlation was seen between the diastolic blood pressure in females and the serum uric acid levels. A mild positive correlation was observed.

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DISCUSSION

This study was conducted in Shri B M Patil Medical College and Hospital, with 93 patients. It was a cross sectional study done in all the patients that satisfy the inclusion criteria. The following studies are compared on the basis of various parameters as described below :

Table 20:

Bibek at el	**TOTAL NUMBER**	**HYPERURICEMIA**
MALE	115	33 (28.4%)
FEMALE	89	26 (29.2%)
This study		
MALE	66	31 (47%)
FEMALE	27	18 (66.7%)

In a study conducted by Bibek Poudel et al, the mean age of the patient was 44 years obtained by observing 205 newly diagnosed essential hypertensive cases with 115 male patients and 89 female patients. Hyperuricemia was observed in 33 (28.4%) male and 26 (29.2%) female patients[78]. This study showed that mean age of the patients was 51.5 years with total 93 newly diagnosed essential hypertension patients were included, of 66 male and 27 female, hyperuricemia was observed in 31 (47%) male and 18 (66.7%) female.

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Table 21 :

	Total (N)	MALE	FEMALE	Mean SUA in mg/dl
Dr Rohit et al	80	58	22	Male-5.49 Female-4.8
This study	93	66	27	Male-6.32 Female-6.03

In a study conducted by Dr Rohit Poondru Reddy et al, total 80newly diagnosed essential hypertension cases studied consisting of 58 male and 22 female, the mean serum uric acid in male is 5.49mg/dl. and in female 4.86mg/dl[79]. In this study total number of newly diagnosed essential hypertension patients was 93 in number consisting male 66 in number and female 27 in number, the mean serum uric acid was 6.32mg/dl in male and 6.03mg/dl in female.

Table 22:

	male	Female	Mean SBP in mmHg	Mean DBP in mmHg
Dr Yeon et al	79 (139)	60 (139)	168.4	114.5
This study	66 (93)	27 (93)	163.6	93.8

In a study conducted by Yeon Ho Kim et al total 139 patients having essential hypertension were studied, consisting of 79 male and 60 female, the mean systolic blood pressure was 168.45mmhHg and mean diastolic blood pressure was 114.5mmHg, and mean age patients was 45.7years[80]. In this study total 93 patients having essential hypertension were studied, consisting of 66 male and 27 female, the mean systolic

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blood pressure was 163.6mmhHg and mean diastolic blood pressure was 93.8 mmHg, and mean age patients was 51.5 years.

Table 23:

	Total (N)	Mean SUA	SD
Gulab et al	50	6.938	1.143
This study	93	6.24	1.38

In a study conducted by Gulab kanwar et al A total of 50 essential hypertensive patients studied consisting of 30 male and 20 female and mean serum uric acid was found to be 6.938mg with standard deviation of 1.143[81]. In a study by L.K Dash et al consisting of 150 essential hypertensive cases the mean serum uric acid level was 6.428[87]. In this study A total of 93 essential hypertensive patients studied consisting of 66 male and 27 female and mean serum uric acid was found to be 6.24 and standard deviation of 1.38

Table 24:

	Male	Female	Uric acid level
Rizwan Ansari et al	51	49	65%
This study	66	23	52.7%

In a study done by Rizwan Ansari et al total 100 hypertension cases studied of 51 male and 49 female with average of 65% having hyperuricemia. A few study have shown that serum uric acid level raised in hypertensive patients[82], A study conducted by Kinsey in 1961, he reported a 46% of incidence of raised serum uric acid level seen in 400 hypertensive individuals[83], and the Kolbe in 1965, reported that 26 patients with increased serum uric acid are found out of 46 hypertensive patients, Which is around 56 %[84].

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Ramsay et al reported 18 patients with raised SUA out of 73 patients which is 25%[85], and Messerli et al had incidence of 72% raised SUA in their population of 39 established hypertensives[86].

In a study by Tykarski (1991), he showed SUA concentration and the prevalence of hyperuricemia were significantly higher in hypertensive patients[87].In this study total 93 hypertension cases studied of 66 male and 27 female with average of 52.7% having hyperuricemia.

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SUMMARY

- This study was conducted in BLDE University's, Shri B.M. Patil medical college, hospital & research centre, Vijayapura.
- In 93 patients, who have satisfied the inclusion criteria and have given due consent, Study included patients between the age group of 25 to 86 year, consisting 66 male patients and 27 female. Most of the patients, were attributed to the age group of 41 to 50 years which incidentally was also the age group for maximum mean serum uric acid level of 5.92.
- All the patients in this study were diagnosed as essential hypertension and other comorbidities seen commonly were type 2 diabetes mellitus followed by chronic obstructive pulmonary disease.
- As the study included subjects with blood pressure recorded more than 140 mmHg systolic and more than 90 mmHg diastolic, the mean systolic blood pressure was 163.66mmHg and mean diastolic blood pressure was 93.83mmHg in male subjects and mean serum uric acid level was 6.32mg/dl.

  In female patients, the mean systolic blood pressure was 165mmHg and mean diastolic blood pressure was 93.56mmHg and mean serum uric acid level was 6.03mg/dl.
- Among the 93 newly diagnosed hypertensive patients studied, 49 patients were found to have hyperuricemia with female predominance of 66.7% and male 47%.
- To study the association of the abovesaid parameters in combination, the Spearman's correlation coefficient was applied and concurrently the p values was also noted. The association displayed a significant positive correlation between the systolic blood pressure and serum uric acid. The same association

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was studies between the diastolic blood pressure and serum uric acid and was found to have a mild, but statistically insignificant correlation.

- The correlation coefficient was applied even to assess the relation between the serum uric acid levels in male and female patients with the systolic an diastolic blood pressures. The result was a mild positive but statistically insignificant correlation.

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CONCLUSION

In this study of the total 93 newly diagnosed essential hypertensive patients, 52.7% of them were associated with hyperuricemia. This study confirmed that the relationship between serum uric acid and blood pressure, hyperuricemia and hypertension is quite different in each age group. Among the hyperuricemic patients female proportion was predominant. 41 to 50 years age group was mainly associated with hyperuricemia. The present study indicates the specific age/gender groups which would obtain maximal benefit from the treatment of hyperuricemia for the prevention and treatment of hypertension. Statistically significant correlation was seen between the serum uric acid levels and systolic blood pressures. Therefore, serum uric acid measurement aids in support the diagnosis of newly diagnosed essential hypertension in middle age group.

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ANNEXURE -I

ETHICAL CLEARENCE CERTIFICATE

B.L.D.E (Deemed to be University)
SHRI.B.M.PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE
VIJAYAPUR – 586103

IEC. no-286/18
17/11/2018

INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 13-11-2018 at 03-15 PM scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.

Title : Estimation of serum uric acid level in newly diagnosed essential hypertension.

Name of P.G. Student : Dr Harshvardhan Narute.
Department of General Medicine.

Name of Guide/Co-investigator: Dr.Vijaykumar.G.Warad, Professor of General Medicine.

DR RAGHAVENDRA KULKARNI
CHAIRMAN
Institutional Ethical Committee
BLDE(U)'s Shri B.M. Patil
Medical College,BIJAPUR-586103.

Following documents were placed before E.C. for Scrutinization:

1) Copy of Synopsis/Research Project
2) Copy of informed consent form.
3) Any other relevant documents.

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ANNEXURE II

INFORMED CONSENT FORM

TITLE OF RESEARCH: "**ESTIMATION OF SERUM URIC ACID LEVEL IN NEWLY DIAGNOSED ESSENTIAL HYPERTENSION".**

GUIDE : **Dr. VIJAYKUMAR. G. WARAD.**
M.D. MEDICINE

P.G. STUDENT : **Dr. HARSHVARDHAN NARUTE.**

All aspects of this consent form are explained to the patient in the language understood by him or her.

PURPOSE OF STUDY:

To study the association of serum uric acid level with newly diagnosed essential hypertension.

PROCEDURE:

I understand that I will undergo detailed history and clinical examination and investigations.

BENEFITS:

I understand that my participation in this study will have no direct benefit to me other than the potential benefit of treatment which is planned to prevent further morbidity and mortality in me.

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of hospital record and will be subjected to confidentiality and privacy regulation of hospital. If the data is used for publication the identity will not be revealed.

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REQUEST FOR MORE INFORMATION:

I understand that I may ask for more information about the study at any time.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or withdraw from study at any time.

(Signature of Guardian) (Signature of patient)

STUDY SUBJECT CONSENT FORM:

I confirm that **Dr. HARSHVARDHAN NARUTE.** has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all above in detail in my own language and I understand the same. I agree to give my consent to participate as a subject in this research project.

DATE SIGNATURE Of PARTICIPANT

DATE SIGNATURE OF WITNESS

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ANNEXURE III

PROFORMA

"ESTIMATION OF SERUM URIC ACID LEVEL

IN NEWLY DIAGNOSED ESSENTIAL HYPERTENSION"

SCHEME OF CASE TAKING :

IDENTIFICATION DETAILS:

Name/Age/Sex	
OP/IP NO.	
Occupation	
Address	

PRESENTING COMPLAINTS:

PAST HISTORY:

TREATMENT HISTORY:

FAMILY HISTORY:

PERSONAL HISTORY:

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GENERAL PHYSICAL EXAMINATION:

SYSTEMIC EXAMINATION :

I. CARDIOVASCULAR SYSTEM:

II. RESPIRATORY SYSTEM:

III. ABDOMINAL AND GENITO URINARY SYSTEM:

IV. CENTRAL NERVOUS SYSTEM:

INVESTIGATIONS :

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COMPLETE BLOOD COUNT-

URINE EXAMINATION-

RANDOM BLOOD SUGAR-

SERUM CREATININE-

FASTING LIPID PROFILE-

SERUM URIC ACID-

ECG-

2D ECHO-

FUNDOSCOPY-

RESULT-

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MASTER CHART

NAME	AGE	SEX	I/P NUMBER	PAST H/0		FAMILY H/0	SYSTO BP	DYSTO BP	SUA LEVEL
							mmhg	mmhg	mg/dl
SHASHIDHAR	68	MALE	13784	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	160	90	7`6
SHOBA RAMANNA	70	MALE	12277	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	148	90	5`9
SANGAMESH	37	MALE	43238	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	160	90	5`2
ASHOK	50	MALE	18273	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	160	92	5`5
PANDURANG	52	MALE	18352	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	160	100	5
HAMU	65	MALE	18248	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	144	90	7`8
MANJUNATH	25	MALE	13366	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	148	90	5`3
PARSHURAM	64	MALE	18377	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	140	90	5`7
ASHOK	58	MALE	12574	K/C/O COPD	1	NOTHING SIGNIFICANT	150	100	8`1
RAMU	56	MALE	13333	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	180	110	5`4
SHANTABAI	86	FEMALE	7107	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	190	110	5
VIJAYAKUMAR	35	MALE	13368	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	160	100	5
PAVITRA	30	FEMALE	13461	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	144	94	4`5
NEELAMMA	68	FEMALE	5405	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	170	100	5`5
ISMAIL	48	MALE	7114	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	170	90	6
NIGAPPA	60	MALE	5230	K/C/O COPD	1	NOTHING SIGNIFICANT	170	100	7`1
BHAVARAVVA	58	FEMALE	4915	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	178	100	7`4
DHULAPPA	75	MALE	7115	K/C/O COPD	1	NOTHING SIGNIFICANT	180	90	7`6
DUNDAPPA	76	MALE	6706	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	180	100	7`2
PARAPPA	70	MALE	7060	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	190	110	7`3
SHIVAJI	43	MALE	17178	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	170	100	7`1
SAHEBAGOUDA	76	MALE	5395	K/C/O COPD	1	NOTHING SIGNIFICANT	170	100	4`4
SHANTABAI	65	FEMALE	13359	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	150	90	6`5
KALPA	60	MALE	13435	K/C/O COPD	1	NOTHING SIGNIFICANT	150	100	7
SUDEEP	40	MALE	12945	K/C/O COPD	1	NOTHING SIGNIFICANT	150	100	3`4
GADIGEPPA	58	MALE	13433	K/C/O COPD	1	NOTHING SIGNIFICANT	144	90	6`9
GANGAWWA	73	MALE	11575	K/C/O COPD	1	NOTHING SIGNIFICANT	150	100	6`9
PRADHU	46	MALE	12560	K/C/O COPD	1	NOTHING SIGNIFICANT	160	100	8`2
SUNIL	56	MALE	13055	K/C/O COPD	1	NOTHING SIGNIFICANT	150	90	6`2

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RAMESH	25	MALE	12911	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	140	90	8`2
CHANDRAWWA	49	FEMALE	13564	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	150	90	6`2
MALLIKARJUN	52	MALE	12382	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	160	90	7`3
GURAWWA	46	MALE	12345	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	160	90	7`5
SARADAR	58	FEMALE	12308	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	170	90	6`3
KALAVATI	48	FEMALE	15809	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	170	90	6`6
SHREEPAL	50	MALE	15755	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	178	98	7`2
FARIDA	50	FEMALE	15763	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	160	90	6`1
CHINANAD	50	MALE	15525	K/C/O COPD	1	NOTHING SIGNIFICANT	170	90	9`6
MALAKAPPA	48	MALE	15766	K/C/O COPD	1	NOTHING SIGNIFICANT	174	98	5`3
RESHMA	35	FEMALE	15836	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	160	90	6`2
MALLIKARJUN	48	MALE	12303	K/C/O COPD	1	NOTHING SIGNIFICANT	160	90	4`6
VIJAYAKUMAR	49	MALE	13368	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	140	90	5
MAHAVDEVI	38	FEMALE	12337	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	168	98	9`7
ISHWAR	50	MALE	15540	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	180	90	7`1
AMEENA	40	FEMALE	14594	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	160	94	6`4
MAHADEVI	35	FEMALE	15212	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	150	90	6`4
MALLIKARJUN	56	MALE	13952	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	180	90	7`2
AMBAWWA	48	FEMALE	13741	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	190	90	4`4
SAHEBAGOUDA	45	MALE	14499	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	150	90	5
LAXMAN	40	MALE	13725	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	190	90	7`5
BASAVARAJ	50	MALE	12302	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	170	90	5`4
SHREESHAI	45	MALE	14241	K/C/O COPD	1	NOTHING SIGNIFICANT	180	100	8`1
GOPAL RAO	35	FEMALE	15485	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	200	100	6`6
THAVARU	40	MALE	15240	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	140	90	6
IRANAGOUDA	50	MALE	16755	K/C/O COPD	1	NOTHING SIGNIFICANT	160	100	4`3
SAHEBAGOUDA	48	MALE	14499	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	180	94	7`6
JAGADEVI	35	FEMALE	14262	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	160	90	7`2
HINAKOUSAR	50	MALE	14395	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	180	90	7
REVANSIDDA	40	MALE	14116	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	160	92	4
SHARANAGOUD	45	MALE	15250	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	150	90	4`4
NIMBEWWA	48	FEMALE	14906	K/C/O COPD	1	NOTHING SIGNIFICANT	170	90	4`3

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PUTALABAI	50	FEMALE	14884	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	150	90	4`2
CHANNAKKA	56	FEMALE	14696	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	160	90	5
SHANTAPPAGOUDA	55	MALE	14667	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	150	90	7`4
ANNAPPA	60	MALE	15607	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	160	90	8
PURAVVA	56	MALE	14756	K/C/O COPD	1	NOTHING SIGNIFICANT	170	100	4`2
RAJENDRA	54	MALE	15569	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	190	90	4`8
GANGAWWA	45	FEMALE	16852	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	180	90	6
AMBIKA	30	MALE	14151	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	190	100	7`8
SUSILBAI	50	MALE	13685	K/C/O COPD	1	NOTHING SIGNIFICANT	160	96	7`3
SHRISHALI	50	MALE	13726	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	180	100	7`3
SHASHIKALA	56	FEMALE	12272	K/C/O COPD	1	NOTHING SIGNIFICANT	150	90	4`1
BHEERAPPA	40	MALE	13422	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	180	100	7`8
SANGAVVU	65	FEMALE	18841	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	156	90	4`1
MABUBBI	50	MALE	13329	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	148	100	4`8
SUSMITA	30	FEMALE	13089	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	150	100	7`2
LAXMAN	60	MALE	18588	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	154	90	4`2
HAMU CHAVAN	65	MALE	18248	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	160	90	3`1
HANUMANT	68	MALE	43052	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	140	90	4`2
SHIVAPUTRAYYA	58	MALE	15852	K/C/O COPD	1	NOTHING SIGNIFICANT	168	90	6`8
SHIVAJI	50	MALE	13633	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	160	90	6`4
PAWADEPPA	56	MALE	14539	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	150	90	7`4
GIRIJABAI	50	MALE	14534	K/C/0 COPD	1	NOTHING SIGNIFICANT	180	90	7`3
SUMANGAL	40	FEMALE	14485	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	170	100	6`4
HANUMTAPPA	45	MALE	14083	K/C/O COPD	1	NOTHING SIGNIFICANT	180	90	4`6
SANGAMMA	50	FEMALE	13648	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	170	90	7`2
VIJAYAKUMAR	50	MALE	13452	K/C/O COPD	1	NOTHING SIGNIFICANT	150	92	6`6
SUMITRA	48	FEMALE	13409	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	160	100	6`4
PARASAPPA	50	MALE	13505	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	150	90	6
SAIBANNA	40	MALE	13533	K/C/O COPD	1	NOTHING SIGNIFICANT	158	94	8`4
PARVATI	60	FEMALE	12289	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	170	90	6`8
MUDAKKAPPA	48	MALE	14646	NOTHING SIGNIFICANT	3	NOTHING SIGNIFICANT	160	94	7`1
SURESH	86	MALE	15949	K/C/O TYPE 2 DM	2	NOTHING SIGNIFICANT	180	90	5`6