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NON-STEROIDAL ANTI-INFLAMMATORY DRUGS IN HYPERTENSIVE PATIENTS: A STUDY OF PRESCRIBING TRENDS AND SIDE EFFECT PROFILES

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Abstract

Hypertension (HTN) can affect all ages despite gender and ethnicity. In our study it was proved that HTN appears more often in women than in men. In this project, 80 patient cases with diagnosis of HTN were collected from which 75 patients had history of use of non-steroidal anti-inflammatory drugs (NSAID's). In elderly people, arthritis and other inflammatory problems become more frequent and a significant number of people are treated with NSAID. Below the age of 35 years, the prevalence of HTN was higher in males but after 35 years, it was higher in females. There was small BP increase in normotensive patients taking NSAIDs approximating +1.1 mm Hg. Patients with treated HTN showed variable increase with NSAID treatment, ranging up to + 14.3 mm Hg for SBP and +2.3 mm Hg for DBP. Most antihypertensive medications have decreased effects with concomitant NSAID administration, with the exception of calcium channel blockers. It appeared that NSAIDs increase BP in patients with controlled-HTN, but the quantity of this increase was variable.

INTRODUCTION

HTN is the most common public health problem in both economically developed and developing nations. (Jugal Kishore *et al.*, 2022) Approximately 50 million individuals in the United States (US) are affected by HTN. (Qureshi *et al.*, 2025) As per WHO reported, about 40% of people with age more than 25 years had HTN in 2008. (Jugal Kishore *et al.*, 2020) The prevalence of HTN in American adults has been estimated about 32%.(Stanley Snowden *et al.*, 2011) In 2004, WHO estimated that in the Eastern Mediterranean Region the average prevalence of HTN was 26% and affected approximately 125 million individuals. NSAIDs are among the most commonly used medications. In the US alone, 23% of adults over 18 years of age use ibuprofen and 3.5% use naproxen. NSAIDs are

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commonly used for minor injuries, headaches, fever, RA, and OA, among other indications. Therefore, quite possibly many patients will take NSAIDs at some point perhaps chronically even if they have HTN. (Stanley Snowden *et al.*, 2011) HTN is an important noteworthy worldwide concern due to its significant contribution to the global health burden and its role as a prominent risk factor for the development of a number of disease processes. In the year 2001, high BP accounted for 54% of stroke, 47% of ischemic heart disease, 75% of hypertensive disease, and 25% of other CVD worldwide. (Lawes, Hoorn *et al.*, 2023).

Definition

HTN is also known as high or raised BP, is a condition in which the blood vessels have persistently raised pressure. BP is measured in millimeters of mercury (mm Hg). According to the seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High BP (2004) defined and classified HTN in adults, as shown in Table 1.1. The diagnosis of HTN is made when the average of two or more DBP measurements on at least 2 subsequent visits is ≥90 mm Hg or when the average of multiple SBP readings on two or more subsequent visits is consistently ≥140 mm Hg. Isolated systolic HTN is defined as SBP 140 mm Hg and DBP 90 mm Hg. (Oscar. A *et al.*, 2020)

Table 1.1: Classification of BP for adults Adapted from JNC 7th. Report (2004)

BP Classification	SBP mmHg	DBP 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

Table 1.1 provides a classification of BP for adults 18 years and older. The classification is based on the average of two or more properly measured, seated, BP readings on each of two or more office visits.

Classification:

There are 2 basic types of HTN: primary (essential) HTN and secondary HTN. The majority of patients (90-95%) have essential HTN. (Richard E Klabunde, 2023).

Primary HTN is a heterogeneous disorder as different patients have a different factor that causes HTN. (Carretero *et al.*, 2020). The cause of essential HTN is still unknown but it is considered as the sum of interaction between genetic and multiple environmental factors. (Büssemaker *et al.*, 2023). Environmental factors including obesity, high alcohol intake, high salt intake, insulin resistance, low potassium intake, aging, sedentary lifestyles, stress, and low calcium intake contribute to the development of HTN. (Carretero *et al.*, 2020) Obesity is associated with more severe HTN along with a need for an increased number of antihypertensive medications. Mechanisms of obesity-induced HTN are complex and not fully elucidated but include impaired sodium excretion, increased sympathetic nervous system activity, and activation of the renin-angiotensin-aldosterone system. (Calhoun *et al.*, 2025)

Secondary HTN is greater in older patients because of a greater prevalence of sleep apnea, renal parenchymal disease, renal artery stenosis and possibly primary aldosteronism. Uncommon secondary causes of HTN include pheochromocytoma, Cushing's syndrome, hyperparathyroidism, aortic coarctation, and intracranial tumors. (Calhoun *et al.*, 2024) Temporary high BP cause by medications such as corticosteroids, NSAIDs, cold medicines and birth control pills. (Grossman *et al.*, 2024) NSAIDs

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particularly are associated with modest but predictable increases in BP. Meta-analyses of the effects of NSAIDs have indicated average increases in mean arterial pressure (MAP) of approximately 5.0 mm Hg. Additional studies indicated that NSAIDs can blunt the BP lowering effect of several antihypertensive medication classes, including diuretics, ACE inhibitors, ARBs, and β-blockers, (Calhoun *et al.*, 2023) except for calcium antagonist and central-acting drugs (Grossman *et al.*, 2022). NSAIDS such as indomethacin, naproxen and piroxicam were the greatest that involves in the increasing of BP, while rofecoxib raise SBP more than celecoxib (Grossman *et al.*, 2022). Other medication classes that worsen BP control include sympathomimetic compounds such as decongestants and certain diet pills and amphetamine-like stimulants. (Calhoun *et al.*, 2024) Cold medicines such as pseudoephedrine hydrochloride that used for upper respiratory decongestant elevates BP in hypertensive patients (Grossman *et al.*, 2025). Glucocorticoids, such as prednisone cause sodium and fluid retention and Corticosteroids (e.g. cortisone, hydrocortisone) produces the greatest amount of fluid retention that results in significant increase in BP. (Calhoun *et al.*, 2024) Intake of birth control pills contributes in the increasing of BP particularly in women above 35 years old that overweight and smokers. (Grossman *et al.*, 2022)

HTN is also referred as the "silent killer". Most people who have high BP usually do not have symptoms. In some cases, people with HTN may have a pounding feeling in their head or chest, a feeling of lightheadedness or dizziness. Other signs include ear noise or buzzing, irregular heartbeat, nose bleeding, tiredness and vision changes. If there are no warning signs, people with HTN may go years without knowing that they have the condition (Siddiqua *et al.*, 2024).

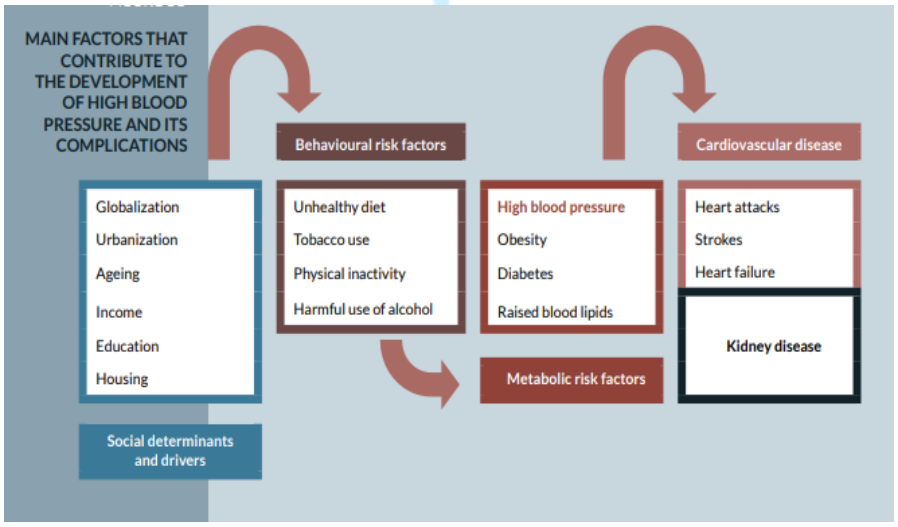

Figure 1.1: Showing the main risk factors that contribute to the development of high BP and its complications by WHO, 2013.

Various antihypertensive drugs such as β-blocking agents, Diuretics, calcium antagonist, ACE inhibitors, Angiotensin II receptor antagonists and α-receptor blocking agents were usually used to control HTN and its alleviate symptoms clinically. Two or more antihypertensive drugs from different categories usually were combined to achieve optimal results. (Campbell *et al.*, 2020)

Thiazide diuretics increase salt and water excretion primarily by inhibiting mechanisms for electroneutral sodium and chloride transport by distal convoluted tubule cells. (Velázquez H.,2024). This leads to depletion of extracellular fluid volume (ECF) and decrease renal blood flow for fall of BP followed by decreased peripheral vascular resistance due to a direct vasodilator action of the drug for example: hydrochlorothiazide. (Edward D. Freis *et*

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al., 2020) Loop diuretics act promptly by blocking sodium and chloride reabsorption in the kidneys even in the patient with poor renal functions or those who have not responded to thiazide diuretics. They cause decreased vascular resistance for example: furosemide. (R.A. Harvey *et al.*, 2023). Potassium sparing diuretics i.e. Amiloride and triamterene (inhibitors of epithelial sodium transport at the late distal and collecting ducts) as well as spironolactone (aldosterone receptor antagonists) reduce potassium loss in the urine. (T.A. Panavelil *et al.*, 2020) The antihypertensive action of beta blockers depends upon a reduction in plasma renin activity (James Conway, 2023). Inhibition in the release of renin from the kidneys decreases the formation of angiotensin II and the secretion of aldosterone and decreased aldosterone causes the sodium water retention and decrease in blood volume resulting in decrease in BP. (R.A. Harvey *et al.*, 2020). The reduction in cardiac output and heart rate also contribute to the fall in BP for example: propranolol, Nebivolol. (James Conway, 2021). ACE inhibitors are used in HTN where thiazide diuretics and beta-blockers are contraindicated. They act by inhibiting the conversion of angiotensin (Ang) I to Ang II and kinin hydrolysis resulting in vasodilation thus decreasing the BP for example: Enalapril, Lisinopril. (Hongmei Peng *et al.*, 2025). Calcium channel blockers (CCBs) inhibit the flow of extracellular calcium through ion-specific channels. When inward calcium flux is inhibited, vascular smooth muscle cells relax, resulting in vasodilation and a lowering of BP. (William J. Elliott *et al.*, 2011) There are three chemical classes i.e. Diphenylalkylamines (verapamil), Benzodiazepines (diltiazem), Dihydropyridine (nifedipine,felodipine, nicardipine, amlodipine). (S. D. Seth *et al.*, 2020) ARBs, such as losartan block the AT1 receptors, decreasing the activation of AT1 receptors by Ang II. Their pharmacologic effects are similar to those of ACE inhibitors in that they produce arteriolar and venous dilation and block aldosterone secretion, thus lowering BP and decreasing salt and water retention. ARBs do not increase bradykinin levels (R.A. Harvey.*et al.* 2023).

NSAIDs:

NSAIDs are the class of drugs that provide analgesic and antipyretic effect and in higher doses, anti-inflammatory effects. The analgesic and anti-inflammatory properties act by inhibiting COX 1 and COX 2. The pharmacodynamic action of these drugs is mostly mediated by inhibition of COX2, while the adverse reactions are largely due to COX1 inhibition. (Charles Ntungwen *et al.*, 2023). NSAIDs are widely used drugs with potential effects on systemic BP. NSAIDs act by inhibiting synthesis of PGs from arachidonic acid via COX-1 and COX-2, the 2 isoforms of COX. NSAIDs may affect BP via the renin–angiotensin pathway, alterations in sodium and water retention in the kidneys, inhibition of vasodilating PGs, and production of various vasoconstricting factors, including endothelin-1 and P450–mediated metabolite so far achidonic acid (Frishman W.H, 2022)

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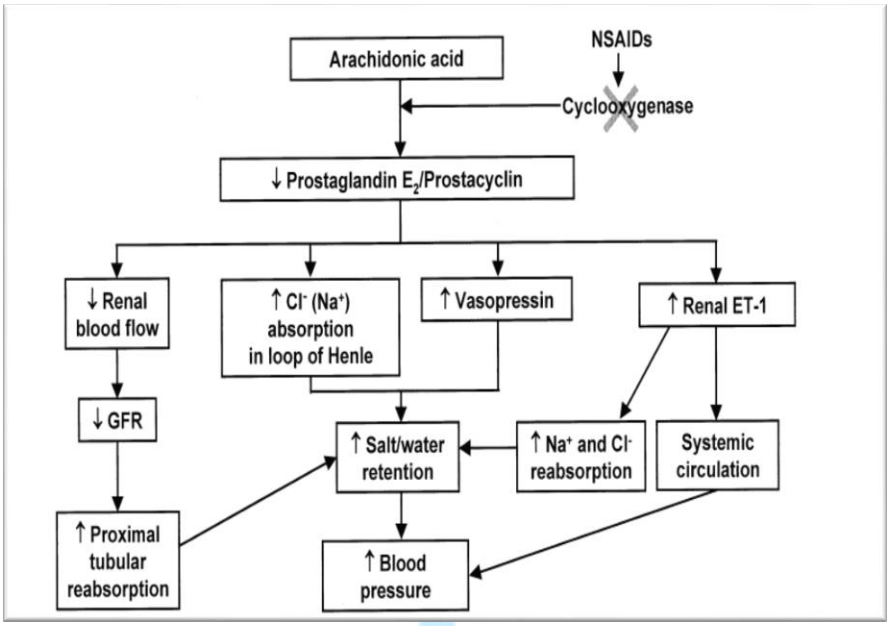

Figure 1.2 Potential mechanisms of NSAIDs induced systemic HTN. Cl-= chlorine; ET= endothelin; GFR= glomerular filtration rate; Na =sodium. (Frishman W.H, 2022).

Table 1.2: Common drug interactions with NSAIDs (Charles Ntungwe *et al.*, 2023).

Drugs	**Results**
Diuretics	Decrease diuresis
B-blockers	Decrease antihypertensive effect
ACE inhibitors	Decrease antihypertensive effect
Anticoagulants	Increase GI bleeding

The most prominent members of this group are aspirin, ibuprofen, naproxen, that are available as over the counter (OTC) drugs in most countries. Paracetamol (acetaminophen) is generally not considered an NSAID because it has only little anti-inflammatory activity. It treats pain mainly by blocking COX-2 mostly in the central nervous system, but not much in the rest of the body. (Charles Ntungwen *et al.*, 2018). In elderly people, arthritis and other inflammatory problems become more frequent and a significant number of people are treated with NSAID. NSAID cause salt and water retention and this may be the reason they elevate BP in both normotensive and hypertensive people. If a person is taking an NSAID and is being treated for HTN then the dose of the drug will be increased or the drug altered so that the HTN is controlled. However, if a person intermittently takes a NSAID, the BP may be controlled when they are not taking the NSAID and go out of control when NSAID is used. (Trefor O. Morgan *et al.*, 2023) A review of the current literature regarding this topic yielded 2 meta-analyses and 10 randomized controlled trials. There is evidence of small BP increase in normotensive patients taking NSAIDs approximating +1.1 mm Hg. Patients with treated HTN show variable increases with NSAID treatment, ranging up to + 14.3 mm Hg for SBP and +2.3 mm Hg for DBP. Most antihypertensive medications seem to have decreased effects with concomitant NSAID administration, with the exception of CCB. (Stanley Snowden *et al.*, 2021) In common with other NSAIDs, concomitant use of diclofenac with diuretics or β-blockers, ACE inhibitors causes a decrease in their antihypertensive effect. Ibuprofen, Diclofenac and celecoxib blocks (COX)-1 and COX-2 enzymes which leads to

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reduction in prostaglandin formation so causes dose related increase in sodium and water retention (Darrell Hulisz *et al.*, 2024). Among the various NSAID's, indomethacin, naproxen, and piroxicam were also associated with greatest increase in B.P (Grossman *et al.*, 2022) Aspirin and sulindac do not appear to elevate BP significantly, even in hypertensive patients. Ibuprofen and other NSAIDs appear to have an intermediate BP effect. COX-2 inhibitors such as refecoxib and celecoxib have been shown to cause mild elevations in BP (Brook RD *et al.*, 2020). It appears that NSAIDs increase BP in patients with controlled-HTN, but the quantity of this increase is variable. (Stanley Snowden *et al.*, 2021).

Objectives:

Our objectives were to:

(1) Examine the association between NSAIDs and BP in patients with HTN.

(2) Compare the effects of various NSAIDs on BP in patients with HTN.

(3) Examine changes in antihypertensive therapy after starting NSAIDs.

Methodology:

Study design:

The methodology applied for this project is non-experimental quantitative study in which community-based survey which was done to evaluate the effect of NSAIDS in HTN patients taking antihypertensive drugs. A questionnaire is designed for the purpose of collection of all the relevant information i.e. the patient's knowledge about the HTN, lifestyle and behavioral assessment of patient along with the list of prescribed antihypertensive and NSAIDs medications. The questionnaire contains all the parameters which are required for the fulfillment of project work. A questionnaire was filled by a volunteer Interviewer, exploring a range of demographic and health-related issues including age, sex, self-prescribed medication, current tobacco and alcohol usage and past medical history. The patient's data was collected from different clinical setups and Jinnah Hospital, Lahore. For comparison and evaluation Research articles, Journals and books were considered. A literature search was conducted using PubMed with the following search terms: "hypertension" and "non-steroidal anti-inflammatory drugs". These terms were cross referenced with each other. Additional articles were found by reviewing the references of relevant search results. For analysis purposes, we entered the data in the database (Microsoft Excel). Results were presented below on corresponding graphs.

Study population:

The patients recruited to the study were men and women, above 25 years, with essential hypertension well controlled taking antihypertensive medication. This control was assessed by BP measurement 24 hours after the drug administration.

Sample Size:

The study was carried out on outdoor and indoor hypertensive patients in various hospitals in Lahore. Data was collected from 80 patients. The study was designed with an approach to learn about the effects of use of NSAIDs in hypertensive patients.

Inclusion criteria

Patients were eligible for inclusion if they had received a prescription for any NSAID, aged 25 years or older and had a clinical diagnosis of arterial HTN. All the subjects were patients with essential HTN and their SBP or DBP were higher than 140/90 mmHg respectively at the baseline period and had at least one measurement of sitting SBP. Outpatients were eligible to participate in the study if they met the following criteria: (1) OA of the hip, knee, or hand (2) stable, controlled systemic HTN; and (3) potential benefit from daily NSAID therapy to control arthritis symptoms. To be included, patients were required to have taken the same fixed dose of antihypertensive medication(s) for ≥3 months before screening.

Exclusion criteria:

The following patients were excluded from the study:

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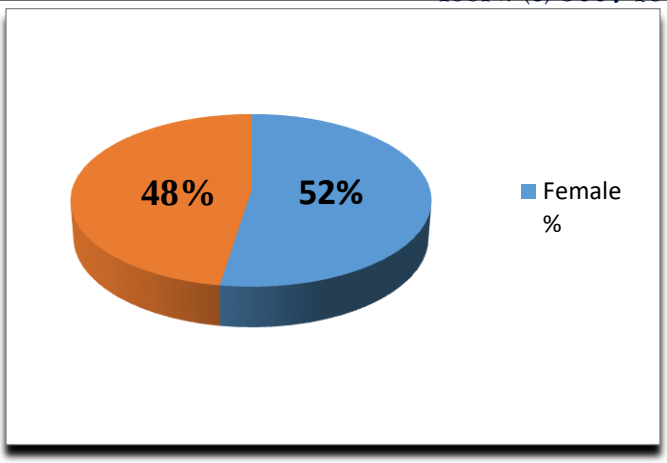

Patients with evidence of a cardiovascular or cerebrovascular episode within the previous 6 months or with unstable angina. Patients with cardiac failure, history of peptic ulceration or gastrointestinal bleeding, renal or hepatic dysfunction, Unconscious patients and pregnant women were also excluded from this study.

Ethical consideration:

Each selected subject was given explanation about the procedure and objectives of the study. Informed written consent was obtained from the study participants. Confidentiality was assured and maintained throughout the study. Names of participants were not captured on questionnaires. All participants were given health education on hypertension after the interview. Previously undiagnosed hypertensive participants who had raised Blood Pressure were referred to clinics for full medical evaluation and subsequent treatment.

Duration of study:

Study period was 6 weeks in duration.

Data analysis:

Data analysis was done using SPSS version 16. The results were explained in simple proportions. In order to analyze the selected patient data, we stored all the collected information into the evaluation database (Microsoft Excel) and analyzed the antihypertensive agents and frequency of used NSAIDS was preliminary analyzed. Results are presented below on corresponding graphs.

Socio-demographic characteristics in Table 4.1 showed the comparison of hypertensive patients' gender that about 42 (52.5%) participants of the total examined population with HTN were female and 38 (47.5%) were male.

Gender	Total no.	Percentage %
Female	42	52.5%
Male	38	47.5%

Table 4.1: shows the comparison of hypertensive patients' gender, where 100% is the total number of patients' population (N=80).

Graph 4.1: The pie graph shows the comparison of hypertensive patients' gender, where 100% is the total number of patients' population (N=80).

The female participants for the age groups 24-34 years, 35-44years, 45-54years and 55 years and above were 3.8%, 12.5%, 18.8% and 17.5% respectively.

The male participants for the age groups 24-34 years, 35-44years, 45-54years and 55 years and above were 7.5%, 10%, 13.8% and 16.3% respectively. Sixty (75%) participants were married and the majority (41.2%) of these being females. About 78.8% of study participants belonged beyond the primary education level. The socio-demographic characteristics of study participants are shown on Table 4.2 below.

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Table 4.2: Frequency distribution of socio-demographic characteristics

Variable	Males (n = 38)	Females (n = 42)	Overall (n = 80)
Age (years)			
25–34	6 (7.5%)	3 (3.8%)	9 (11.3%)
35–44	8 (10.0%)	10 (12.5%)	18 (22.5%)
45–54	11 (13.8%)	15 (18.8%)	26 (32.5%)
55+	13 (16.3%)	14 (17.5%)	27 (33.8%)
Marital Status			
Married	27 (33.8%)	33 (41.2%)	60 (75.0%)
Non-married	11 (13.8%)	9 (11.2%)	20 (25.0%)
Level of Education			
Primary	8 (10.0%)	9 (11.3%)	17 (21.3%)
Secondary	10 (12.5%)	15 (18.8%)	25 (31.3%)
Tertiary	20 (25.0%)	18 (22.5%)	38 (47.5%)

Table 4.3: Distribution of blood pressure (BP) where both mean systolic blood pressure (SBP) and mean diastolic blood pressure (DBP) increase with age

Age Group (Years)	Mean Systolic BP ± SD (mmHg)	Mean Diastolic BP ± SD (mmHg)
<25	122.6 ± 12.0	73.1 ± 9.6
25–34	126.0 ± 11.7	79.3 ± 8.1
35–44	131.1 ± 16.0	82.3 ± 16.2
45–54	140.3 ± 20.5	87.2 ± 13.0
55+	145.3 ± 21.4	89.1 ± 11.0

Prevalence of HTN was higher in the older age groups as shown on graph 4.2. Below the age of 25 years, the prevalence of HTN was 0.0%. it raised to 11.25%, 22.5%, 32.5%% and 33.75% for the age groups 24-34 years, 35-44years, 45-54years and 55 years and above respectively. Below the age of 35 years, the prevalence of HTN was higher in males but after 35 years, it was higher in females as shown on Figure 4.2.

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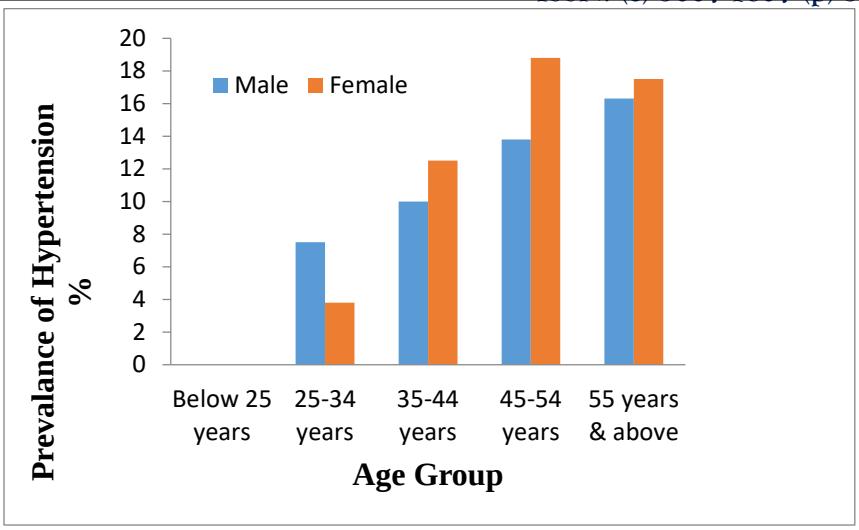
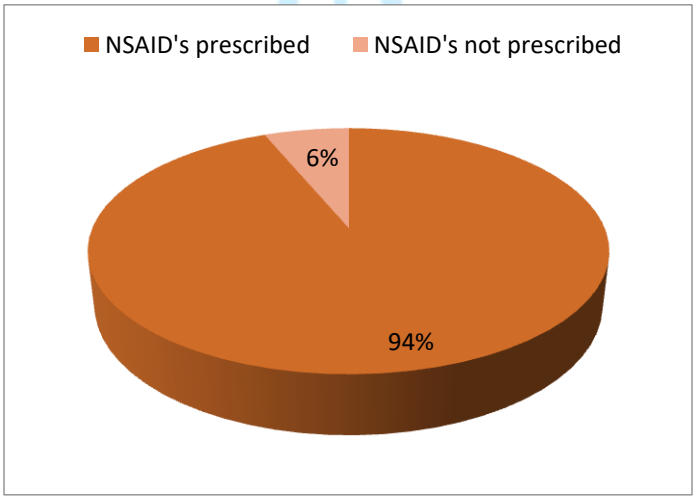


Graph 4.2: Age and sex distribution of the prevalence of HTN

Category	No. of patients	Percentage
NSAID's prescribed	75	93.8%
NSAID's not prescribed	5	6.3%

Table 4.4: Calculation of patients either NSAID's prescribed or not.



Graph 4.3: Showing the pie chart of percentage of patients prescribed NSAID's and percentage of patients not prescribed NSAID's

Drugs	Number of patients	Percentage of the study group
Aspirin	20	26.7%
Paracetamol	13	17.3%
Ibuprofen	12	16%
Diclofenac	10	13.3%
Naproxen	8	10.6%

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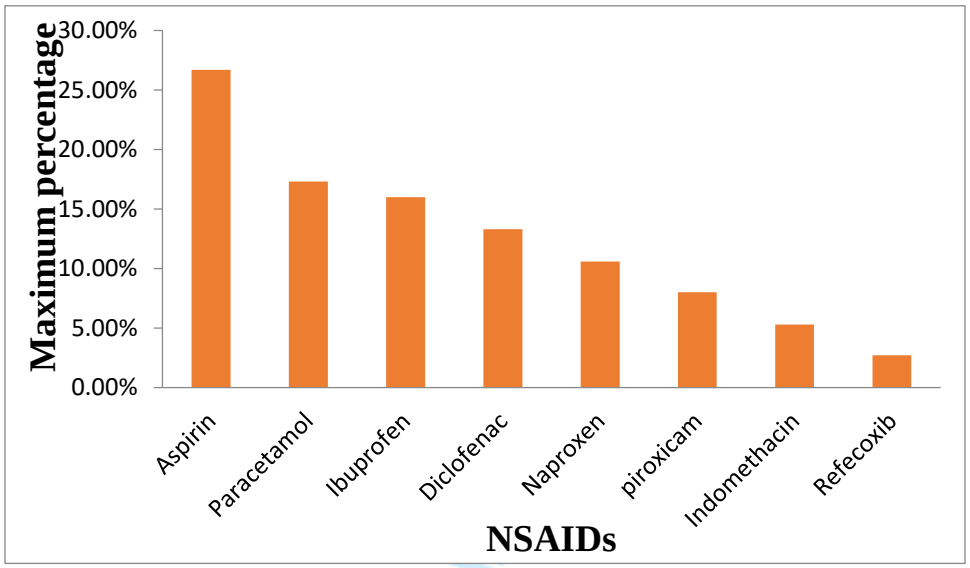
Piroxicam	6	8%
Indomethacin	4	5.3%
Refecoxib	2	2.7%

Table 4.5: Frequency of NSAIDs used by the patients.

Table 4.5 shows that among 80 participants who were previously diagnosed with HTN from which 75(93.8%) were using NSAID's for some reasons like aspirin (26.7%), Paracetamol (17.3%), Ibuprofen (16%), Diclofenac (13.3%), Naproxen (10.6%), Piroxicam (8%), Indomethacin (5.3%) and refecoxib (2.7 %),



Graph 4.4: shows Frequency of NSAIDs used by the patients

We had evaluated 20 patients within age group 45-54 years with BP 160/92 mm Hg prescribed with aspirin 650mg daily and 13 patients with paracetamol 1g 8th hourly concurrently taking thiazide diuretics and B-adrenoceptor antagonists so according to J.P. Chalmers, Changes in BP were minimal in the low dose aspirin study and the paracetamol study. Aspirin caused 2 mm Hg increase in supine DBP ($p < 0.05$). Changes in BP were again only slight in the patients completing the paracetamol study in which the only significant changes were increases of 4 mm Hg in supine and standing SBP during the paracetamol phase. (J.P. Chalmers, *et al.*, 2025). According to Isabella Sudano, acetaminophen at doses used in daily clinical practice may increase BP in patients with CAD so the use of acetaminophen should be as rigorously evaluated as all traditional NSAIDs, particularly in patients at increased cardiovascular risk (Isabella Sudano, *et al.*, 2023).

We had evaluated 12 patients within age group 45-54 years with BP 139/88 mm Hg were prescribed with ibuprofen 400 mg O.D due to arthritis. Robert Palmer in his research article stated that Ibuprofen caused significantly greater increase in systolic and diastolic (BPs) in hypertensive patients, stabilized on an ACE inhibitor (Robert Palmer et al., 2023) and Alan Morrison found that the mean change in BP (in mmHg) from baseline to end of study for trials of ibuprofen was 3.54 for SBP and 1.16 for DBP. (Alan Morrison *et al.*, 2025) Aljadhey found that Ibuprofen was associated with a 3 mmHg increase in SBP compared to naproxen and a 5-mmHg increase compared to celecoxib. The SBP increase was 3 mmHg in patient's concomitantly prescribed with ACE inhibitors or CCB and 6 mmHg among those prescribed a beta- blocker. (Aljadhey *et al.*, 2022)

10 patients were evaluated within age group 45-54 years prescribed with antihypertensive medication and concurrently taking diclofenac. Krum *et al*,

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demonstrated that etoricoxib 60–90mg once daily raises BP to a greater extent than diclofenac 150 mg/day in patients with OA and RA. He further added CCB were associated with the least increase in BP upon administration of anti-inflammatory drugs, suggesting that they could be considered as first-line therapy for HTN in patients receiving ns-NSAIDs or COX-2 inhibitors (Krum *et al.*, 2021). Whelton. A stated that the conventional NSAIDs such as Diclofenac increases the risk of the CVD and HTN (Whelton, A., 2022)

We had evaluated that 8 patients with age of 55 above used 250 mg naproxen T.D for RA. Their BP were 142/95 mmHg previously diagnosed with HTN and prescribed with Beta blockers. C. Ljungmana *et al* analyzed an association between the dose of naproxen and an increase in SBP when comparing the reference dose of 250mg with higher doses. Accordingly, to his study naproxen was associated with a dose dependent increase in SBP ,7mm Hg for the 1000mg dose of naproxen. (C. Ljungmana *et al.*, 2023)

We had evaluated 6 patients within age group 45-54 years prescribed with antihypertensive medication and concurrently taking piroxicam. A meta-analysis was performed by Janet E. Pope to determine the hypertensive effects of NSAIDs. According to Pope, the effects of NSAIDs on BP were found solely in hypertensive subjects. His findings concluded that piroxicam increased MAP by 2.86 mm Hg but after adjusting salt-intake level in these hypertensive subjects, Piroxicam increased MAP by 0.49mm Hg (Janet E. Pope, *et al.*, 2021).

We had evaluated 4 patients within age group 45-54 years were prescribed with indomethacin 50mg T.D. They were diagnosed with HTN had using ACE inhibitors for BP control. Their BP were 141/92 mmHg. According to the researcher Trefor O. Morgan Indomethacin was associated with 10.1/4.9 mm Hg increase in BP in people taking enalapril (Trefor O. Morgan *et al.,* 2000) J.P. Chalmer in his research article stated that there was 9 mm Hg increase in mean BP in hypertensive patients due to incident use of indomethacin. (J.P. Chalmers *et al.*, 2021)

We had evaluated 2 patients with edema and OA used rofecoxib 25 mg/day while their BP were 159/98 mmHg >65 years of age who were treated with fixed antihypertensive regimens. According to Andrew Whelton Patients receiving rofecoxib had a mean 24-hour SBP increase of 4.7 mm Hg compared with placebo. Rofecoxib caused the greatest increase in systolic BP in patients receiving ACE inhibitors or Beta blockers. (Andrew Whelton, *et al.*, 2002).

DISCUSSION

In this project, 80 patient cases with diagnosis of HTN were collected from which 75 patients had history of use of NSAID's. For this study, HTN was defined as SBP ≥140 mmHg and DBP ≥90 mmHg, or previous diagnosis of HTN or current use of antihypertensive medication or any combination of the above. Furthermore, control of HTN was defined as SBP <140 mmHg and DBP <90 mmHg. Despite the study limitations, all the results were given in the form of graphs.

Firstly, regarding findings, the comparison of hypertensive patients" gender and age showed that about 42 (52.5%) participants of the total examined population with HTN were female and 38 (47.5%) were male. The hypertensive group was significantly higher in individuals more than 35 years than those less than 35 years. The prevalence of HTN was found to be strongly linked to age. The female participants for the age groups 24-34 years, 35-44years, 45-54years and 55 years and above were 3.8%, 12.5%, 18.8% and 17.5% respectively. The male participants for the age groups 24-34 years, 35-44years, 45-54years and 55 years and above were 7.5%, 10%, 13.8% and 16.3% respectively. . Below the age of 25 years, the prevalence of HTN was 0.0%. it raised to 11.25%, 22.5%, 32.5%% and 33.75% for the age groups 24-34 years, 35-44years, 45-54years and 55 years and above respectively. Below the age of 35 years, the prevalence of HTN was higher in males but after 35 years, it was higher in females. The prevalence of HTN is increased by age, accounting higher ratios in women after the age of 65. The higher prevalence in females may be due to the high level of obesity and physical inactivity was more common in among females than males. The increasing prevalence of HTN with age represents the biological effect of increased arterial resistance due to thickening arterial wall that comes with age. Sixty (75%) participants were married and the majority (41.2%) of these being females. About

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78.8% of study participants belonged beyond the primary education level. Among 80 participants 75(93.8%) were previously diagnosed with HTN were using NSAID's for some reasons like aspirin (26.7%), Paracetamol (17.3%), Ibuprofen (16%), Diclofenac (13.3%), Naproxen (10.6%), Piroxicam (8%), Indomethacin (5.3%) and refecoxib (2.7 %).

CONCLUSION

HTN is a common disease in our community and the prevalence increases as the population ages. In our study, it was observed that NSAIDs increase BP in patients with controlled-HTN, but the quantity of this increase was variable. Below the age of 35 years, the prevalence of HTN was higher in males but after 35 years, it was higher in females. So NSAIDs should be prescribed with precaution in hypertensive patients.

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