

# Increased Uric Acid Levels in Patients with Hypertension: The Role of Calcium Channel Blockers and Beta Blockers

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## Abstract

**Background:** One of the most common issues that is arising globally is premature cardiovascular death, which is caused by hypertension. With the passage of time, the occurrence of cardiovascular diseases and hypertension is also increasing globally. Currently, the incidence of deaths that are caused by hypertension is about 33% worldwide. **Objective:** To do a comparison of the effects of calcium channel blockers and beta blockers on increased uric acid levels in people with hypertension. **Study design:** An analytical cross-sectional study. **Place and Duration:** This study was conducted in Sulaiman Roshan Medical College Hospital TandoAdam from May 2022 to May 2023. **Methodology:** There were a total of 80 people selected to be a part of this research. All of the participants were diagnosed with hypertension. All of these participants were equally divided into 2 groups, 40 patients each. One group included those patients who received Metoprolol as an antihypertensive (Group A), and the other group received Amlodipine as an antihypertensive. The participants in this research were aged from 40 to 50 years. SPSS version 22 was used to analyze the data. A T-test was applied. MS Excel and Word were used to generate tables and graphs. **Results:** This research was conducted on a total of 80 patients who were equally divided into 2 groups. All of the participants were between 40 and 50 years old. The average age was calculated to be 46 years. The average weight was 73 kg. The average HbA1c level was 5.1%. There were a total of 42 males and 38 females. **Conclusion:** In conclusion, there was no significant difference between amlodipine and metoprolol on serum uric acid levels in patients with hypertension.

**Keywords:** amlodipine, metoprolol, hypertension, hyperuricemia, adults

## 1. Introduction

One of the most common issues that is arising globally is premature cardiovascular death, which is caused by hypertension [1]. With the passage of time, the occurrence of cardiovascular diseases and hypertension is also increasing globally [2]. Currently, the incidence of deaths that are caused by hypertension is about 33% worldwide [3].

Due to certain factors, hypertension is still a severe disease that needs to be treated. One of the factors that are related to the increased incidence of hypertension is uric acid [4]. In 1879, it was the first

time that serum uric acid was found to be linked with hypertension. Later, there were other research studies conducted in which it was reported and confirmed that as uric acid levels increase, the incidence of hypertension increases [5, 6, 7]. Inflammation, activation of the renin-angiotensin-aldosterone system, oxidative stressors, and endothelial dysfunction are some procedures that are involved in contributing to the development of hypertension that is caused by rising levels of uric acid [8]. Moreover, there is also a link between hypertension and xanthine oxidoreductase polymorphism [9]. However, it is reported that there

is no link between the development of hypertension and the genetics of key urate transporters [10].

The regulation of uric acid and excretion processes is mediated by urate transporters, while the production of uric acid is mediated by xanthine oxidoreductase [11]. For the treatment of hypertension, amlodipine is used as the calcium channel blocker, and metoprolol is used as the cardio-selective beta-blocker [12]. According to some research studies, the safer and more beneficial option for the treatment of patients with hypertension who have raised serum uric acid levels is calcium channel blockers rather than beta blockers [13]. Hence, we have conducted this research to do a comparison of the effects of calcium channel blockers and beta blockers on increased uric acid levels in people with hypertension.

## 2. Methodology

There were a total of 80 people selected to be a part of this research. This sample was selected on a non-probability basis. All of the participants were diagnosed with hypertension. All of these participants were equally divided into 2 groups, 40 patients each. One group included those patients who received Metoprolol as an antihypertensive drug (Group A), and the other group received Amlodipine as an antihypertensive drug. The participants in this research were aged from 40 years to 50 years. The average weight of the patients was up to 80 kg. The patients had diastolic blood pressures of 100 to 110 mmHg and systolic blood pressures of 140 to 150 mmHg, serum uric acid levels of >6.5 mg/dl in females and >8 mg/dl in males, and normal HbA1c%. Informed written consent was obtained from every patient.

**Exclusion criteria:** Patients who are aged less than 40 years or more than 50 years were excluded from this research. Moreover, participants with cardiomyopathy, liver disorders, myocardial infarction, renal failure, and diabetes mellitus were also not a part of this research.

Group A patients were given Metoprolol tablets 50mg per day and group B patients were given Norvasc tablets 5mg per day. A digital sphygmomanometer was used to measure the blood pressure. For the estimation of serum uric acid level, a 5 cc blood sample was drawn. Troponin T tests and an ECG were also done to assess any cardiac pathology. SPSS version 22 was used to analyze the data. A T-test was applied. MS Excel and Word were used to generate tables and graphs.

## 3. Results

This research was conducted on a total of 80 patients, who were equally divided into 2 groups. All of the participants were between 40 to 50 years old. The average age was calculated to be 46 years. The average weight was 73 kg. The average HbA1c level was 5.1%. There were a total of 42 males and 38 females.

**Table No. 1: frequency of males and females in the groups.**

Gender	Group A	Group B
Female	18	21
Male	22	19

The findings were recorded initially at level 0 and then at level 1 (after three months of antihypertensive medications).

**Table No. 2: findings of group A at level 0 and level 1.**

Parameters	Level 0	Level 1
Mean Serum uric acid (Males)	7.4 mg/dl	7.1 mg/dl
Mean Serum uric acid (Females)	6.2 mg/dl	6 mg/dl
Mean Systolic blood pressure	146 mmHg	126 mmHg
Mean Diastolic blood pressure	106 mmHg	89 mmHg

**Table No. 3: findings of group B at level 0 and level 1**

Parameters	Level 0	Level 1
Mean Serum uric acid (Males)	7.2 mg/dl	7 mg/dl
Mean Serum uric acid (Females)	6.1 mg/dl	5.9 mg/dl
Mean Systolic blood pressure	147 mmHg	124 mmHg
Mean Diastolic blood pressure	106 mmHg	86 mmHg

## 4. Discussion

Uric acid is a mild acid, and urate ions are present in normal blood. XOR is an enzyme that breaks down and changes metabolites like xanthine and hypoxanthine into urate compounds. This happens when purines are broken down. XOR comes in two forms: (A) xanthine dehydrogenase (the main form) and (B) Xanthine oxidase [14, 15]. Hyperuricemia is defined as elevated uric acid levels in the bloodstream, with serum uric acid levels surpassing 6 mg/dl in females and 7 mg/dl in males.

The major cause of hyperuricemia is a decrease in uric acid removal. Renal failure, hypothyroidism, and the use of beta blockers in hypertensive individuals are all common causes of reduced uric acid excretion in urine. Furthermore, during ischemic heart disease and times of stress, blood uric acid levels may rise due to xanthine oxidase stimulation [16].

Several things can lead to high blood pressure caused by uric acid, such as changes in the renin-angiotensin-aldosterone system (RAAS), endothelial dysfunction, stress, and inflammation throughout the body [17]. Certain studies imply that ischemia in renal arterioles may be a contributing factor to hypertension in hyperuricemia patients, albeit the exact process is unknown. Furthermore, there is a significant biological link between genders, particularly in postmenopausal women, where the female gender has a larger correlation with hyperuricemia than premenopausal women.

According to Liu J et al. and J. Tang et al., beta blockers increased blood uric acid levels while calcium channel blockers decreased serum uric acid levels [18, 19]. This research contradicts our findings. There were no significant effects of beta-blockers or calcium channel blockers on blood uric acid levels in our research sample. However, our study time may be limited. That was just for three months, but these scientists conducted their experiments over the course of a year. Our findings are comparable with

those of Lutfu Askin et al., who reported that using beta blockers to treat hypertension has no effect on blood uric acid levels [20].

There are some limitations to this study that should be acknowledged. The study was relatively short in duration, implying the necessity for a more extensive and protracted research period with a larger sample size. Notably, our study primarily included premenopausal women, with postmenopausal women underrepresented. A study examining the effects of hypertensive medications on blood uric acid levels in both premenopausal and postmenopausal women might be valuable in future research.

## 5. Conclusion

In conclusion, there was no significant difference between amlodipine and metoprolol on serum uric acid levels in patients with hypertension.

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