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**Research Article**

# The impact of uric acid levels and hypertension on alcohol consumption patients in rural areas

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**ABSTRACT**

Hyperuricemia can raise the probability of various ailments, including gout, diabetes, cardiovascular disease, obesity, chronic renal disease, metabolic syndrome, hypertension, and other conditions.
**Aim**: The aim of the study is the impact of uric acid levels and hypertension on alcohol consumption patients in rural areas.
**Result**: There was significant difference in the age group, diastolic blood pressure, uric acid and glucose marker among the case group, when compared with the control group.
**Conclusion**: Advice to hypertensive patients to reduce or impede alcohol intake in totting up to other behavioural habit and the pharmaceutical interventions may give an additional advantage in terms of blood pressure reduction

**Keywords:** blood pressure, hypertension, uric acid, alcohol

## Introduction

The body eliminates uric acid, which is the final product of the breakdown of purines, through urine(1,2). Hyperuricemia, characterised by high Serum Uric Acid (SUA) levels, may occur due to increased SUA production or reduced excretion(3). The presence of hyperuricemia can increase the likelihood of several illnesses, including but not limited to gout, diabetes, cardiovascular disease, obesity, chronic kidney disease, metabolic syndrome, hypertension, and other disorders.
Hyperuricemia has become more prevalent worldwide in recent decades(4). There was an increase in the prevalence of hyperuricemia among Asian population, with rates rising from 11.1% in 2015-2016 to 14.0% in 2018-2019(5). Factors implicated in the development of hyperuricemia include diet, genetics, and lifestyle(6-10). Drinking alcohol has been reported by numerous investigators to increase the risk of hyperuricemia(11-14).
A meta-analysis demonstrated that alcohol had a larger influence on gout than hyperuricemia(15). A cross-sectional study among Americans found that drinking beer increased SUA levels more than drinking liquor (0.46 vs 0.29 mg/dL), while moderate wine consumption did not increase uric acid levels (-0.42 mg/dL, 95% CI: -0.62, -0.22). A prospective study of American men found that drinking beer increased the risk of gout more than drinking spirits (RR: 1.15, 95% CI: 1.04, 1.28), and moderate wine consumption had no effect on gout (RR: 1.04, 95% CI: 0.88, 1.22).

## Material and Methods:

The present case-control study was carried out at hospital in Erode after obtaining Institutional Human Ethics Committee approval and written informed consent from study subjects. The biochemical markers was investigated in 200 patients. The alcoholic patients (Case Group - 100 patients) and Non alcoholic patients (Control group - 100 patients) are recruited for study. Only males of age group between 18-70 are included for

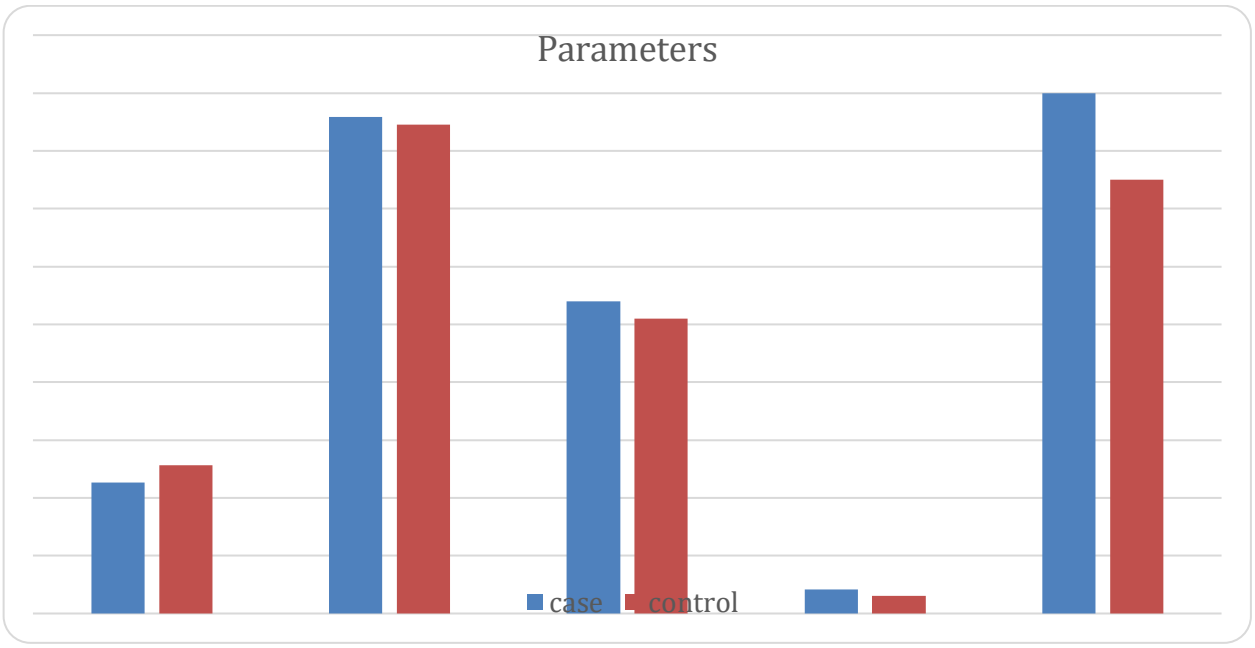
the study. A total of 2ml of venous blood was collected from all the subjects. Serum samples are used for biochemical analysis such as glucose and uric acid levels in subjects.

## Results:

**Table 1: Anthropometric and Biochemical markers of Case and Control groups**

| | Case (Alcoholic ) | Control (Non-Alcoholic) | P value |
|---|---|---|---|
| Age | 45.2 ± 7.8 | 51.2 ± 8.3 | < 0.001 |
| SBP | 171.8 ± 18.2 | 169 ± 16.9 | 0.42 |
| DBP | 108 ± 12.1 | 102 ± 11.9 | 0.00 |
| UA | 8.4 ± 1.4 | 6.2 ± 1.1 | <0.001 |
| Glucose | 180 ± 22.4 | 150 ± 17.2 | <0.001 |

**Fig 1: Bar diagram of Anthropometric and Biochemical markers of Case (alcoholic) and Control (Non-alcoholic) groups**




The anthropometric and biochemical measures of both case and control groups were presented in Table 1. There was significant difference in the age group, diastolic blood pressure, uric acid and glucose marker among the case group, when compared with the control group.

There was significant difference in the age group (P = <0.001), diastolic blood pressure (p =0.01), uric acid (<0.001) and glucose (<0.001) marker among the case group, when compared with the control group.

## Discussion:

Alcohol (particularly, ethanol) use can raise the SUA by increasing its production and slowing its removal from the body(15). Alcohol use promotes ATP breakdown, resulting in the creation of adenosine and adenine(16). Adenosine can be further degraded to hypoxanthine and inosine(17). The enzyme xanthine oxidase then turns hypoxanthine to xanthine, which is eventually converted into uric acid(18). As a result, increased ATP breakdown would lead to increased purine and uric acid synthesis. Furthermore, alcohol metabolism raises lactic acid levels in the blood, which hinders uric acid excretion through the renal tubules.

Higher average daily alcohol intake was related with marginally but substantially higher SBP and DBP in a population of Black and White men and women with hypertension, and these correlations were not demonstrated to be mediated by other hypertension treatment behaviours. However, this study is unusual in that it examines whether alcohol intake affects blood pressure in ways other than these direct biological linkages. We found no evidence for such indirect interactions. Such findings are clinically significant because they emphasise the importance of screening persons with hypertension for alcohol consumption.

Several recent studies have revealed that most persons with hypertension receive recommendations to restrict dietary salt and promote exercise (66-76%), but little more than one-third receive guidance to minimise alcohol

use (19). Dietary purines account for around one-third of the body's daily blood uric acid synthesis; the remainder is derived from endogenous sources. Elevated uric acid can also be associated with increased purine breakdown in high cell turnover conditions (e.g., hemolysis, rhabdomyolysis, tumour lysis) and reduced excretion (e.g., hereditary diseases, renal failure, metabolic syndrome). The kidney excretes around two-thirds of uric acid, while the gastrointestinal system excretes the remaining one-third. However, these proportions can fluctuate based on drugs or failure in the renal or GI systems (20). For the past two decades, Hyperuricemia has been well studied on revealing mechanistic insights into UA homeostasis. Some epidemiological and experimental evidence suggests that both hyperuricemia and hypouricemia can lead to cardiorenal pathologies respectively(21).

There was significance between alcoholic and non-alcoholic patients such as Age group, (P = <0.001), diastolic blood pressure (p =0.01), uric acid (<0.001) and glucose (<0.001) marker among the case group, when compared with the control group. Yang X, Lin Y et al., studies showed that, the significance present between case and control group. Beilin LJ et al., research revealed that the same outcome had been reproduced.

## Conclusion:

Advice on Hypertensive patients to impede or reduce the alcohol intake in addition to other behavioural habits and the pharmaceutical interventions also gives an additional advantage in terms of blood pressure reduction.

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