*Available online at* www.scientiaresearchlibrary.com

**Scientia Research Library**

**ISSN 2348-0416**
**USA CODEN: JASRHB**

**Journal of Applied Science And Research, 2019, 7 (5):25-27**

(*http://www.scientiaresearchlibrary.com/arhcive.php*)

# Reduction of hyperuricemia by potassium supplementation in a hypertensive patient

**R. LOUMINGOU*, G. MAHOUNGOU**

***Service de Néphrologie C.H.U de Brazzaville***

## ABSTRACT

*The beneficialeffects of potassicsupplementation on health are welldocumented. We report an observation thatsuggest a probable in fluence of potassium intake on the regulation of uric of in a hypertensive patient. Wediscuss a relevant linkbetween variations of the potassium and uricacidthrough variations in sodium and stimulation of the reninangiotensinaldosteron system.*

**Keywords :** potassicsupplementation, uricacidregulation.

## INTRODUCTION

Disturbances of potassium and uric acid can lead to cardiovascular, renal and metabolic abnormalities (1,2). Potassium is an essential mineral for the body. Numerous clinical studies have proven the beneficial effect of potassium intake on lowering blood pressure, cardiovascular mortality, sodium reduction, renal protection, prevention of osteoporosis and urolithiasis (3,4).

Hyperuricemia is considered a cardiovascular risk factor (5,6) and is frequently associated with glucose intolerance, obesity, and high blood pressure (7). We report an observation that suggests the likely beneficial role of potassium supplementation in decreasing uricemia.

## MATERIALS AND METHOD

A 45 year old hypertensive man known for 8 months was treated with captopril, replaced by a hydrochlorothiazide-amlodipine combination due a persistent dry cough.

He was received on an outpatient nephrology consultation for physical asthenia, cramps, pain and swelling of the big toe associated with gouty arthritis. The blood pressure was 105 / 60mmHg, biologically there was a hyperuricemia 108mg / l ( normal 40 - 70), hyponatremia 121 mg / l (normal 135-145), hypokalaemia 2.4 mmol / l (normal 3.5 - 5), uricosuria was low 80mmol / 24h, the clearance of creatinine according to MDRD at 85ml / min. Hydrochlorothiazide was stopped. The prescribed allopurinol was stopped after 15 days due to the appearance of generalized pruritus attributed to toxiderma which was resolved a few days after the discontinuation of allopurinol.

Mixed potassium supplementation, medicated (potassium gluconate) and dietetic (diet rich in fruit and vegetables) was prescribed for 1 month. The recommended fluid intake of 1.5 to 2 liters per day was respected.

The potassium level increased to 4.6mmol / l, the decrease in uricemia to 60mg / l, the normalization of uricosuria to 420mmol / 24h, the kaliuresis to 80mmol / 24h. Twenty-five days after stopping potassium supplementation, the potassium level decreased to 3.2mmol / l, the uricemia rose to 76mmol / l. The re-introduction of potassium intake was accompanied by a normalization of Kalemia at 4mmol / l, uricemia at 65mg / l natremia at 133mol / l, natriuresis at 180mmol / 24h, uricosuria 380mmol / 24h.

## RESULTS AND DISCUSSION

Potassium is an essential mineral for the body whose absorption provides

demonstrated health benefits (8). The harmful effects of the potassium deficit have also been described; increased cardiovascular mortality, glucose intolerance (7) increased stroke and exacerbation of hypertensive salt sensitivity (9). Hyperuricemia also found in high cardiovascular risk situations (10) is frequently associated with hypertension in 25% of untreated hypertensives, 50% of hypertensives treated with diuretics and

75% of patients with malignant hypertension (11). Hypotheses have been proposed to explain this association; the development of arteriolopathy following high blood pressure induces tissue ischemia and activation of the renin angiotensin aldosterone system (12). Other authors mention the decrease in renal perfusion in connection with high blood pressure which would promote the reabsorption of sodium-coupled uric acid (13). Arguments argue in favor of a relevant link between potassium supplementation and the variations in uric acid found in our observation. The increased potassium intake led to an increase in the excretion of uric acid in the urine and a decrease in serum uric acid levels. The increase in uricemia was noted when potassium supplementation was stopped. We hypothesize that potassium supplementation is likely to lead to a reduction in uricemia by the sodium reduction consecutive to hypoaldosteronism induced by potassium load and to the increase in natriuresis which is coupled with the increase uricosuria.

## CONCLUSION:

Disturbances of potassium and uric acid are frequently associated with cardiovascular and metabolic risk situations.

There seems to be a relevant link between potassium supplementation and the decrease in uric acid through the renin angiotensin aldosterone system.

Potassium supplementation can be systematically offered to hyperuricemic patients, especially in cases of intolerance to conventional hypouricemic agents.

## REFERENCE

[1] Hoeiggen A, Alderman MH, Kjelden SE et al. The impact of serumuricacidon cardiovascular out comes in the LIFE study.Kidney international **2004**; 65: 1041 – 9.

[2] Alper AB, Campbell RC et al. A propensithy – matchedstudy of lowserum potassium and mortality older adults with chronic heart failure. *Int J cardiol* **2009** sep 11; 137 (1): 1 – 8.

[3] GudHeJ et al. Effet of potassium supplemantation on blood pressure inchinese arandomized palce bo – controlled trial. *J Hypertens* **2001** Jul; 19 (7): 1325 – 31.

[4] Appel LJ et al. American society of hypertension writting group ASH position paper: Dietary approaches to lover blood pressure. J clin Hypertens (Greenwich) **2009** Jul; 11 (7): 358 – 68.

[5] D. wietlisbach v, Bovet P et al. Prevalence of hyperuricemia and relation of serumuric acid with cardiovascular risk factors in a developing country.BMC Public health **2004**; 8.

[6] Niskanem, Laaksonen DE, Nyyssonen K et al. Uricacidlevel as a risk factor for cardiovascular and all cause mortality in middle – aged men : A prospective cohortstudy. *Arch intern Med* **2004**; 26: 1545 – 51.

[7] Nakanishi N, Okamoto M, Yoshida A et al. Serumuricacid and risk for development of hypertension and impairedfasting glucose or type II diabetes in Japanese male office workers. *Eur J epidemiol* **2003** : 18: 523 – 30.

[8] China Salt substitute study collaborative Group. Salt substitution : a low – coststrategy for blood pressure control among rural chinesse.A randomized, controlled trial. *J Hypertens*. 2007 oct: 25 (10): **2011** - 8.

[9] Brancati FL, Appel LJ et al. Effet on potassium in AfricanAmericans on a low-potassium diet. A randomized double-blind placebo – controlled trial. *Arch inter Med* **1996** Jan 8; 156 (1): 61-7.

[10] Johnson RJ, Kang DH, Feig D et al. Is there a pathogenicrole for uricacid in hypertension and cardiovascular an renaldisease ? Hypertension **2003** ;41: 1183 – 90.

[11] Franse LV et col. Serumuricacid, diuretictreatment and the risk of cardiovascularevents in the systolic hypertension in the Elderly program (SHEP). *J Hypertens* **2000**; 18: 1149 – 54.

[12] Mazzali M, Hughes J, Kim Y et al. Elevateduricacidincreaseblood pressure in the rat by a novelcrystalindependantmechanism. *Hypertension* **2001**; 28: 1001 – 6.

[13] Messerli FH, Frohlich ED, Dreslinki GR al. Serumuric in the essential hypertension : An indicator of renalvascularinvolvement. *Ann Int Med* **1980**; 98: 817 – 21.