ISSN 2250-3153

Effect of Caffeine on Serum and Urinary Electrolytes

G. Geethavani [1], M. Rameswarudu [2], R. Rameswari reddy [3], S. Babu rao [4], D. Moulali [5]

[1] Tutor, Department of Physiology, SVS Medical College, Mahabubnagar, A.P.
[2] Professor of Physiology & Dean, SVS Medical College, Mahabubnagar, A.P.
[3] Professor & HOD, Department of physiology, SVS Medical college, Mahabubnagar, A.P.
[4] Assistant professor, Department of Anatomy, Santhiram Medical college, Nandyal, A.P.
[5] Tutor, Department of BioChemistry, SVS Medical College, Mahabubnagar, A.P.

Abstract- In order to establish a relationship between caffeine and serum and urinary electrolytes, 30 healthy males were given an oral dose of 400 mg caffeine over a period of 120 min in four divided doses and electrolyte concentrations were measured during control and experimental periods. Serum potassium decreased significantly($P<0.01$) from 4.4±0.2 (control period) to 3.9±0.2 (experimental period at 400 mg of caffeine). Increased urinary sodium (i.e from 139±3 in the control period to 209±8) and increased chloride concentrations (i.e from 46±7 in the control period to 124±9) associated with increased urinary output was noticed significantly($P<0.01$) at 400 mg of caffeine. These results helps in establishing a dose response relationship between caffeine and electrolytes.

Index Terms- caffeine, serum potassium, urinary sodium, urinary output.

I. INTRODUCTION

Caffeine is a xanthine alkaloid most commonly consumed by humans. In North America, United Kingdom, and Denmark 82 to 95% of adults regularly consume caffeine (1, 2). Caffeine is generally considered as safe under moderate consumption (according to FDA). In increased doses, caffeine causes a significant change in renal and cardiovascular indices (3). Caffeine is associated with changes in serum and urinary electrolytes. We know that electrolytes are important in regulating the amount of water present in the body, acid- base balance and muscle function etc. Very few studies (3, 4) were reported in literature regarding the effect of caffeine on electrolytes. There is a need for study to determine the relationship between the dose of caffeine ingested and serum and urinary electrolytes for two reasons. Firstly, they are reported with inadequate sample size. Secondly, no report is published based on Indian population.

This study mainly aims at establishing a dose response relationship between caffeine and serum and urinary electrolytes, with adequate sample size in Indian population. It serves as a clinical database for Indian population.

II. MATERIALS AND METHODS

Thirty healthy male volunteers of age group between 19-22 years and weight between 45-60 kg were recruited for this study. They were non-smokers and not suffering from any cardiac or pulmonary diseases. All the participants gave the informed written consent for the study. The study was approved by Institutional Human Ethical Committee on 26/03/2010. The study was conducted according to the guidelines of SVS Medical College, Mahabubnagar.

All the participants were instructed not to take coffee or tea atleast four days prior to the test. They were instructed to take normal diet and were restricted to eat foods high in sodium and potassium content. Once they were in the lab they were permitted to take rest for 30 minutes. Blood pressure and heart rate were recorded for all the individuals using sphygmomanometer and pulse rate.

On stabilisation, blood and urine samples were collected from all the participants and sent to laboratory and labelled as controls. All the participants were given 100 mg of caffeine in gelatine capsules. This was repeated at 30- minute intervals for a total period of 120 minutes which accounts to a total amount of 400 mg caffeine. At each interval, blood and urine samples were collected before the caffeine intake and sent to laboratory. In order to maintain the water balance and to ensure a brisk urine flow rate, each participant is allowed to take an equal amount of water load after voiding. This procedure was followed to eliminate total body water.

Analyses:

The blood samples in the laboratory were centrifuged by 3000rpm to separate serum and allowed to estimate the electrolytes by selective ionic exchange method. Urine samples were also allowed to estimate the electrolytes by selective ionic exchange method. The data was tabulated and analysed by using SPSS 16 statistical soft ware. Values are represented as means ± SD.

III. RESULTS

1. Serum electrolytes:

Serum potassium was found to decrease significantly from 4.4±0.2 in the control period to 3.9±0.2 at 400 mg of caffeine intake (Table 1). Serum sodium and chloride levels decreased slightly but not significant.

Table 1: Serum electrolyte concentrations (means ± SD; n = 30 subjects)

	Control period (30 min)	Experimental period (Total 120 min)				significance
		100 mg caffeine (30 min)	100 mg caffeine (30 min)	100 mg caffeine (30 min)	100 mg caffeine (30 min)	
Na^+ (mmol/L)	139±2	139±2	138±2	138±2	138±2	NS (p=0.5)
K^+(mmol/L)	4.4±0.2	4.4±0.2	4.4±0.2	4.3±0.2	3.9±0.2	P < 0.01, S
Cl^-(mmol/L)	106±2	106±2	106±2	105±2	105±2	NS (p=0.5)

2. Urine output & electrolytes:

Increased urinary sodium (i.e from 139±3 in the control period to 209±8) and increased chloride concentrations (i.e from 46±7 in the control period to 124±9) associated with increased urinary output was noticed significantly at 400 mg of caffeine intake (Table 2). Urinary potassium concentrations increased slightly but not significant.

Table 2: Urine output and electrolyte concentrations (means ± SD; n = 30 subjects)

	Control period (30 min)	Experimental period (Total 120 min)				significance
		100 mg caffeine (30 min)	100 mg caffeine (30 min)	100 mg caffeine (30 min)	100 mg caffeine (30 min)	
Na^+ (mEq/L)	139±3	144±3	150±4	177±5	209±8	P<0.01, S
K^+(mEq/L)	18±3	18±3	19±3	20±3	21±3	NS (p=0.75)
Cl^-(mEq/L)	46±7	51±7	58±7	87±8	124±9	P<0.01, S
Output(ml)	104±4	109±4	113±4	120±4	142±2	P<0.01, S

IV. DISCUSSION

Present study states that with increasing doses of caffeine i.e at 400mg, a significant acute increase in urinary sodium and chloride concentrations were noticed. At this dose of caffeine, a significant increase in urinary output was also noticed. This clearly suggests that, with moderately high doses of caffeine it causes acute natriuresis associated with diuresis. The same was supported by Shirley D.G. et al[4] which states that caffeine causes an acute substantial increase in sodium excretion with accompanying diuresis at moderately high doses. According to Passmore AP et al[3], caffeine doses greater than 90mg will significantly increase the urinary sodium excretion but urinary volume was increased by 360 mg of caffeine only. No significant change in urinary output was noticed with low oral doses of caffeine (45, 90 and 180 mg). Reviews of scientific literature[5,6] stated that caffeine induces acute diuretic effect in greater dosages [i.e >250 mg/d (5); >680 mg/d (6)]. Another study by Gonzalez-Alonso[7] also suggested that caffeine causes an acute diuretic effect when consumed in moderately high doses.

The intrarenal mechanisms responsible for the natriuretic and diuretic effects of caffeine remains to be determined. Many conflicting views exist over the role of altered renal haemodynamics and tubular reabsorption. D.G., Shirley et al (3,8) states that the caffeine induced natriuresis resulted largely from inhibition of fractional tubular reabsorption with out effecting the renal plasma flow, since hemodynamic effects are involved in natriuresis. However based on scientific reviews[4,8] the probable causes are: 1. Inhibition of proximal tubular sodium reabsorption due to A_1-adenosine receptor antagonism of caffeine.[9-11] 2. Reduction of distal sodium reabsorption due to A_2-adenosine receptor antagonism of caffeine, but the underlying mechanism behind this remain unexplained.

The study states that caffeine also causes slight increase in urinary potassium concentrations which are not significant. This was supported by Shirley D.G. et al[4] who states that no major significant changes are observed in urinary potassium concentrations with caffeine ingestion.

Present study states that caffeine decreases the concentrations of serum sodium and chloride levels to a lesser extent which are not significant. Scientific reviews[3,4] substantiated that no major changes were observed in the concentrations of serum sodium and chloride levels.

Caffeine also causes a significant decrease in the concentrations of serum potassium at doses of 400 mg. Passmore AP et al[3] also stated that Serum potassium was significantly reduced by 360 mg of caffeine.

V. CONCLUSION

The present study confirmed and established a dose response relationship between caffeine and serum and urinary electrolytes with adequate sample size. It also provides a clinical database for Indian population. This data suggest the importance of regulating the caffeine intake in human beings.

REFERENCES

[1] Barone, J.J., and H.R. Roberts. Caffeine consumption. Food Chem. Toxicol. 34:119-129, 1996.

[2] Gilbert, R.M. Caffeine consumption. In: The methylxanthine beverages and foods:chemistry, composition, and health effects. New York: Alan R. Liss Co., 1984, pp.185-213.

[3] Passmore, A.P., G.B. Kondowe, and G.D. Johnston. Renal and cardiovascular effects of caffeine: a dose-response study. Clin. Sci. 72:749-756, 1987.

[4] Shirley, D. G., Walter, S. J., and Noormohamed, F. H. Natriuretic effect of caffeine: assessment of segmental sodium reabsorption in humans. Clin. Sci. 103:461-466, 2002

[5] Nehlig,A., and G.Debry. Caffeine and sports activity: a review. Int. J. Sports Med. 15:215-223, 1994

[6] Armstrong, L.E. Caffeine, body fluid-electrolyte balance, and exercise performance. Int. J. Sport Nutr. Exerc. Metab 12:189-206, 2002

[7] Gonzalez-Alonso, J., C.L. Heaps, and E.F. Coyle. Rehydration after exercise with common beverages and water. Int. J. Sports Med. 13:399-406, 1992

[8] Arnaud, M.J. (1997) The pharmacology of caffeine. Prog. Drug Res. 31, 273-313

[9] Knight, R.J., Browmer, C.J. and Yates, M. S. (1993) The diuretic action of 8-cyclopentyl-1,3-dipropylxanthene, a selective A1 adenosine receptor antagonist. Br. J. Pharmacol. 109, 271-277

[10] Mizumoto, H. and Karasawa, A. (1993) Renal tubular site of action of KW-3902, a novel adenosine A1-receptor antagonist, in anesthetized rats. Jpn. J. Pharmacol. 61, 251-253

[11] Wilcox, C. S., Welch, W.J., Schreiner, G. F. and Belardinelli, L. (1999) Natriuretic and diuretic actions of a highly selective adenosine A1 receptor antagonist. J. Am. Soc. Nephrol. 10, 714-720

AUTHORS

First Author – G. Geethavani, Tutor, Department of Physiology, SVS Medical College, Mahabubnagar, A.P.
Second Author – M. Rameswarudu, Professor of Physiology & Dean, SVS Medical College, Mahabubnagar, A.P.
Third Author – R. Rameswari reddy, Professor & HOD, Department of physiology, SVS Medical college, Mahabubnagar, A.P.
Fourth Author – S. Babu rao, Assistant professor, Department of Anatomy, Santhiram Medical college, Nandyal, A.P.
Fifth Author – D. Moulali, Tutor, Department of BioChemistry, SVS Medical College, Mahabubnagar, A.P.