Original Article

Serum and urine electrolyte changes following ligation of a left segmental renal artery in dogs

Veeranna Hoovinahole Bhadranna, Maqbool Ahmed[1]

Department of Physiology, Saptgiri Institutes of Medical science, [1]Bangalore Medical College, Bangalore, Karnataka, India

Abstract

Background and Aim: Ligation of renal artery has been reported to cause acute reduction of nephron population resulting in altered Na^+ and K^+ excretion pattern. However, no study has reported electrolyte balance following the ligation of a branch of left renal artery.

Methods: Segmental ligation of left renal arterial branch was performed in 18 dogs. Changes in the excretion pattern of Na^+ and K^+ and the urine flow rate by the remnant kidney have been observed. Serum and urine samples were collected before and after the ligation at 30-minute intervals for a period of two hours, and Na^+ and K^+ concentrations were determined along with the flow rate of urine.

Results: A considerable reduction in the urinary excretion rate of Na^+ and K^+ was observed in 30 minutes after the ligation. Subsequently, a gradual rise in the rate of excretion of Na^+ and K^+ towards pre-ligation values was observed by the end of 120 minutes. Statistically significant increase in K^+ concentration in urine was observed at the end of two hours (P = 0.023). The serum analysis for both the electrolytes in all experiments showed no significant changes in concentrations.

Conclusion: Findings of the present study suggest that ligation of an artery segment in the kidney though affects acute changes in K^+, it does not affect the overall homeostasis of either water or electrolytes. The maintenance of homeostasis might be achieved through the improvement of function by the remnant kidney.

Key words: Electrolytes, ligation, segmental renal artery

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INTRODUCTION

The term homeostasis refers to the maintenance of constancy of volume and composition of intercellular fluids that forms the internal environment of the body and cells.[1] The concentration of Na^+ and K^+ electrolytes in the extracellular fluid (ECF) is maintained by the transport mechanisms occurring across the cell membranes and by the kidneys.[1] The kidneys regulate the electrolyte concentration by altering their rate of excretion in the urine.[2] Kidneys besides possessing *de novo* mechanisms for the synthesis of hormones also become the target organ for many hormones.[2] These hormones act upon renal tubules facilitating either the re-absorption or secretion of these electrolytes into the tubular fluid. Thus, kidneys play a major role in the maintenance of *milieu interieur*.

Renal artery in dogs is divided into five segmental arteries and each of them is destined for one of the vascular segments of the kidney.[3] One of these segmental arteries courses posterior, while the remaining branches lie between the veins and the renal pelvis. These segmental arteries are not anastomosed with one another, except for small extra renal, capsular, and pelvic channels.[3] The present study was designed to assess the effects of the ligation of a branch of renal artery of one kidney in dogs in which it usually reduces the functional nephron population of a part of kidney, to (i) observe changes occurring in the excretion of sodium and potassium in urine (ii) observe changes occurring in the concentration

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Address for correspondence: *(present working address) Dr. Veeranna Hoovinahole Bhadranna, Department of Physiology, Sapthagiri Institute of Medical Sciences and Research Centre, Bangalore - 560 090, Karnataka, India. E-mail: vhb49@yahoo.com

of sodium and potassium in serum, and (iii) study the rate of flow of urine following ligation.

MATERIALS AND METHODS

All animal experiments and procedures performed were approved by the institutional animal ethics committee. Experiments were performed on 18 dogs of either sex as described before by Cannon *et al*.[2] They were anesthetized by administering pentobarbital sodium at 30 mg/kg body weight intravenously. During the experiments, the constant body temperature of the dogs was maintained and 0.9% saline was infused to the dogs at the rate of 1.2 ml/min. A sub-coastal flank incision was made in the abdomen of all the animals to expose the left side kidney. The left renal artery and its branches were identified and one of the segmental branches was isolated and kept ready for ligation. In all experiments, the left ureter was catheterized just before the application of ligature. The control sample of urine was collected in a dried glass tube from the left ureter for every 30 minutes. Similarly, a control sample of 4-ml blood was drawn from the left renal vein. In all experiments, a silk thread was tied around the segmental branch. Blood samples were collected once in 30 minutes for two hours. They were centrifuged for 20 minutes at a rate of 3000 rpm, and sera were collected in different glass tubes and refrigerated. Urine samples were also collected in different test tubes at an interval of 30 minutes for a period of two hours, and the volume of each urine sample was measured before analysis. The rate of urine flow was calculated by dividing the volume of urine by the time taken for collection.[4] All urine and serum samples were analyzed for Na^+ and K^+ concentrations using flame photometer (Model CL 22A, Elico Pvt Ltd, Hydrabad). Stock solutions of the electrolytes were prepared for the construction of standard curves. Measurements before ligation are taken as control group for comparison with those after ligation.

Statistical analysis of data

Experimentally determined renal characteristics of flow rate of urine, excretion rates, and urine as well as serum concentrations of Na^+ and K^+ were subject to descriptive statistics. Analysis of variance (ANOVA) was performed to compare the control group with measured values at 30, 60, 90, and 120 min in order to determine statistical significance of the changes. $P < 0.05$ has been considered as statistically significant change after ligation. Data were analyzed using Microsoft Excel software version 2010 (Microsoft office, United States).

RESULTS

Variation in the urinary flow rate, Na^+ and K^+ concentrations in both urine and serum, and excretory pattern of these electrolytes in urine were noted at 30-minute intervals for two hours after ligation.

The urine flow rates observed before and for two hours after ligation are shown in Figure 1. The urine flow rate dropped considerably from the control value of 66.7 μL/min to 48.6 μL/min during the first 30 minutes, followed by a gradual increase to 52.3 μL/min by the end of two hours. However, these changes were not statistically significant ($P = 0.45$) [Figure 1].

The mean concentrations of Na^+ and K^+ observed in urine before and after ligation are presented in Figure 2, and their excretory rates are shown in Figure 3. The sodium concentration measured at 30 minutes after ligation also showed a considerable reduction from 130.3 mEq/L to 118.4 mEq/L. Subsequently, Na^+ concentration gradually increased and reached 136.7 mEq/L by the end of two hours. This change in Na^+ concentration was found to be statistically insignificant ($P = 0.74$). On the other hand, K^+ concentration levels showed a drop from the mean control value of 64.4 mEq/L to 61.6 mEq/L after ligation but the changes were statistically insignificant. Subsequently, K^+ concentration increased, in a statistically significant manner, at every 30-minute interval and reached the value of 79.3 mEq/L by the end of two hours ($P = 0.023$) [Figure 2].

After ligation, the mean rate of sodium excretion (through urine) observed during the first 30 minutes was reduced appreciably from mean control value of 9.40 μEq/L to 6.16 μEq/L as shown in Figure 3. The rate of excretion increased subsequently at each observation to reach a value of 8.19 μEq/L at the end of two hours. However, these changes were also found to be statistically insignificant when compared to the control values ($P = 0.57$). Similarly, the rate of potassium excretion decreased significantly from the mean control value of 4.9 μEq/L to 3.1 μEq/L during the first 30 minutes after the ligation. This was followed by a gradual increase to 4.2 μEq/L over a period of two hours, which was statistically insignificant when compared to the control values ($P = 0.32$) [Figure 3].

The concentrations of Na^+ and K^+ in serum were found to be practically unchanged, within 149.3 ± 7.7 and 5.5 ± 1.1 mEq/L, respectively, during all the time intervals before and after ligation ($P = 0.95$ for Na^+ and $P = 0.97$ for K^+) [Figure 4].

DISCUSSION

The sudden decrease in the urinary flow rate observed for a period of 30 minutes soon after ligation could be due to the diminished blood flow to the kidney. Arrest of blood supply to a particular segment of the kidney causes reduction in the total glomerular filtration rate.[5] During 30-120 minute interval after ligation, a gradual rise in

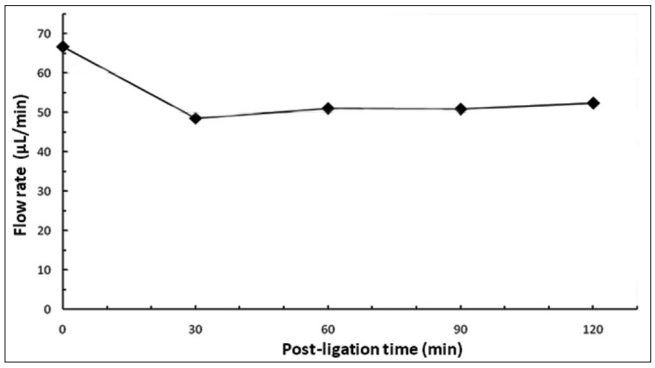

Figure 1: Rate of urine flow after ligation

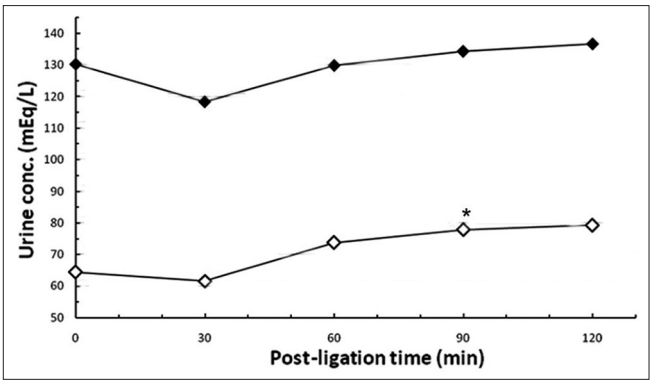

Figure 2: Concentration of Na^+ and K^+ in urine after ligation (♦ Na^+, ◊ K^+). *p<0.05

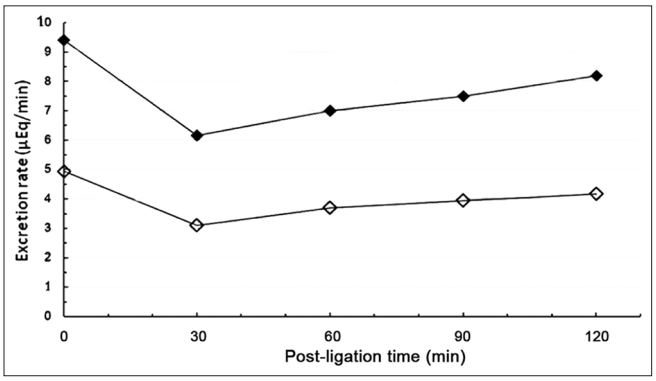

Figure 3: Excretion rates of Na^+ and K^+ in urine after ligation (♦ Na^+, ◊ K^+)

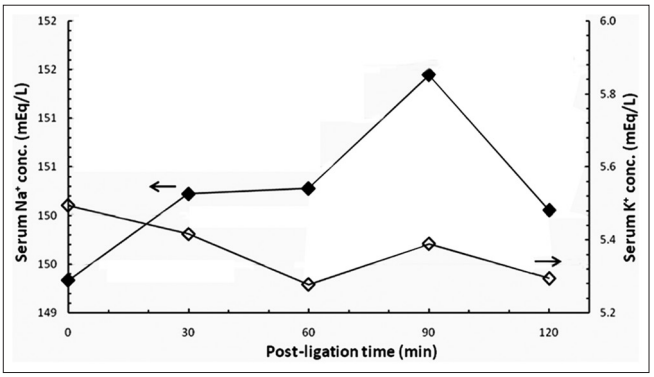

Figure 4: Concentrations of Na^+ and K^+ in serum after ligation (♦Na^+, ◊ K^+)

the rate of urine flow was observed, possibly due to the reabsorption of water in the proximal tubules of the surviving nephrons, secondary to the reduction in the proximal tubular reabsorption of sodium. This observation is consistent with the reports of the previous studies.[3,4]

The significant reduction in the urinary concentration of Na^+ and its excretion noted in the first 30 minutes of ligation is due to sudden arrest of blood flow to the experimental segment. Consequently, sodium excretion rate gradually increased towards pre-ligation values between the intervals of 30-120 minutes. Peter[3] and Allison *et al.,*[6] have also observed similar rise in the rate of sodium excretion in their experiments. This increase in the sodium excretion is attributed to a decrease in the tubular sodium reabsorption by the surviving nephrons. Diezi *et al.,*[7] have also reported a decline in the fractional sodium reabsorption in proximal tubules and they have identified the rise in the peritubular hydrostatic pressure, as the cause of glomerular imbalance, acting across proximal tubule. Bricker[8] had attributed the observed "natriuresis" to a factor known as natriuretic hormone, which modulates sodium excretion in the surviving nephrons.

Although the urine concentration of K^+ measured between 0-30 minutes interval was found to be reduced, it was not statistically significant, whereas the decrease in its excretion rate was significant. This reduction in K^+ concentration in urine occurred due to sudden arrest of blood supply to the particular kidney segment under experimentation. Subsequently, K^+ concentration and its excretion increased significantly from 30-120 minutes and are attributed to the increased secretion of the ion at the collecting tubules. A previous experimental study by Schultze *et al.,*[9] also observed a four-fold increase in the potassium excretion per nephron in dogs. This increase occurred within 18 hours of nephron reduction. However, the research team could not identify the factor regulating the kaleuresis. By conducting micro-puncture and clearance experiments in rat models following 3/4th of nephrectomy, Bank *et al.,*[10] have noted a large increase in tubular fluid potassium content between the end of the distal tubule and the final urine. Based on these results, they have concluded that the collecting duct is responsible for the enhanced potassium excretion in the surviving nephrons. Similar animal model study by Malnik *et al.,*[11] have identified cortical collecting tubule as the site of both active K^+ and Na^+ excretions in the isolated perfused tubule preparation. It may be concluded from the above findings that the ligation of a branch of renal artery results in the initial fall in the potassium excretion rate. The subsequent gradual rise is due to the increased

potassium secretion, in exchange for the increased sodium load arriving at the collecting tubular level.

The concentrations of Na^+ and K^+ in serum after ligation remained almost constant compared to pre-ligation control values. Although slight variations were noticed, they are found to be statistically insignificant, similar to studies reported earlier.[9,10,12]

The renin-angiotensin-aldosterone system plays an important role in the regulation of sodium balance and ECF volume. Negative or positive changes in sodium balance are commonly accompanied by iso-osmotic contraction or expansion of blood ECF volumes. Plasma sodium concentration does not change accordingly with changes in sodium balance because of the tendency to preserve iso-osmoticity by the removal or addition of appropriate amounts of water to the ECF. The mechanisms involved in the regulation of sodium and potassium excretion in the urine are aimed at maintaining the concentration of both the electrolytes in plasma despite the reduction in nephron population.

Though there are reports[9,10,13] that half of one kidney is adequate to maintain the renal functions of an individual, till date no study has been conducted to assess the effect of ligation of an artery segment of the kidney on the kidney functions. Usually, the arterial ligations alter the electrolytic balance and affect the body fluid homeostasis. However, the present study demonstrates that ligation of such segmental artery is not functionally injurious to kidney. However, future research should be done in patients suffering from renal vascular disease to detect if such segmental renal arterial disease produces electrolyte and fluid imbalance in the body.

Limitations of the study

In the present study, the interstitial pressure has not been recorded following ligation. Recording of blood pressure and its correlation with the electrolyte concentrations would have given more evidence to substantiate the results of the present work.

CONCLUSION

Segmental ligation of a renal arterial branch might lead to acute reduction in nephron population, which may change the excretion pattern of Na^+, K^+, and the urine flow rate. Though, the present study showed that the ligation of segmental artery of the left kidney does not influence the electrolyte and fluid homeostasis, a study in larger sample size could confirm it. Further experiments are required to study the mechanisms on interstitial pressures and starling forces, that act as the driving factors for the renal reabsorbtion and secretion of electrolytes.

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