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The Relationship Between KCl Infusion and Changes of ECG, Electrolytes of Plasma and K Content of Donkey's Red Blood Cells

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Abstract: Sudden marked increases in the serum potassium concentration, up to 8 to 9 meq L^{-1}, are the result of transcellular movement of potassium and are associated with profound electrocardiographic abnormalities and fluid shifts. Electrocardiographic changes associated with hyperkalemia have been described in the cat, the dog, the horse and are well documented in man. Since there is no research about the effects of hyperkalemia on electrocardiogram of donkey, the purpose of this study was to induce the experimentally hyperkalemia in donkey and studying the relationship between KCl infusion and changes of ECG, electrolytes of plasma and K content of donkey's red blood cells. This research was carried out in seven clinically healthy female donkeys, injected with 0.35 molar potassium chloride solutions to jugular vein. Results indicated that potassium content of RBC at 30, 45, 60, 75 and 90 min, blood potassium level at 30, 45, 60, 75 and 90 min, blood calcium at 60, 75, 90 and 105, increased significantly and blood magnesium decreased significantly at 135 min. Electrocardiographic changes were first degree A-V block, wandering pacemaker, inversion of negative to positive T-wave, flattening of the P-wave, ventricular premature beat, sinus arrest, bradycardia and sinus tachycardia.

Key words: Infusion, potassium chloride, electrocardiographic changes and donkey

INTRODUCTION

Hyperkalemia may develop *in vitro* as a result of hemolysis or leakage of erythrocyte potassium after storage of whole blood (Smith, 2002). The release of potassium from leukocytes or platelets into the serum after clot formation is a potential cause of hyperkalemia if marked leukocytosis or thrombocytosis is present (Cunningham, 1992). Hyperkalemia also result from renal potassium retention in Addison's disease, acute renal failure and renal shutdown (Radostits *et al*., 2000). A number of factors contributed to movement of interacellular potassium into ECF, resulting in hyperkalemia. Hyperkalemia has been reported in animals with massive muscle necrosis, but neither hyperkalemia nor a metabolic acidosis is a common feature in horses with exertional rhabdomyolysis (Swenson *et al*., 1996). Episodic hyperkalemia and muscular weakness are associated with the condition known as hyperkalemic periodic paralysis. Sudden marked increases in the serum potassium concentration, up to 8 to 9 meq L^{-1} are the result of transcellular movement of potassium and are associated with profound electrocardiographic abnormalities and fluid shifts. It has been anticipated that state of hyperkalemia may affect electrocardiogram (Williamson, 1974). Electrocardiographic changes associated with hyperkalemia have been described in the cat (Schaer, 1977; Norman and Barrett, 2006), the dog (Feldman and Ettinger, 1977), the horse (Epstein, 1984) and are well documented in man (Gorantitou *et al*., 2005). As a result of any research about the effects of hyperkalemia on electrocardiography in donkey has not been done yet, It was therefore, the purpose of this study to induce the experimentally hyperkalemia in the donkey.

MATERIALS AND METHODS

The study was carried out in the Department of large animal internal medicine and surgery, Veterinary Faculty of Shahrekord University, Iran, in February 2006.

Seven healthy female non-pregnant donkeys four years old with average weigh of 221.42±10.1 Kg were used for this study. Jugular vein was cannulated (14 g 20 cm polyethylene catheter). Potassium chloride were prepared as 0.35 molar solutions (26.09 g potassium chloride added to 1 L sterile water) and infused into jugular vein at a rate

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of 0.11 cc kg^{-1} body weight. Before commencing infusion and at 15 min intervals thereafter, 10 mL blood samples were collected and electrocardiograph was recorded. Five milliner of blood samples placed in plastic tubes containing the anticoagulant heparin, centrifuged and the plasma separated. Potassium, sodium, calcium and magnesium concentrations were estimated by Atomic absorption (Spectrometer Atomic Absorption model 939 Unicam). Another 5 mL of blood samples placed in plastic tubes containing the anticoagulant heparin, centrifuged and erythrocytes separated. The potassium content of erythrocytes measured with Atomic absorption.

Base apex technique (the left electrode placed at the heart area, the right electrode applied to the jugular furrow and the earth electrode placed at the left flank) used to recording electrogarciograph as lead I.

RESULTS AND DISCUSSION

Mean value (±SE) for constituents measured in plasma electrolytes and potassium content of erythrocytes in the eight donkeys are shown in Table 1. The electrocardiographic abnormalities are demonstrated in Fig. 1-8. Results indicated that potassium content of erythrocytes at 30, 45, 60, 75 and 90 min, blood potassium level at 30, 45, 60, 75 and 90 min, blood calcium at 60, 75, 90 and 105, increased significantly and blood magnesium decreased significantly at 135 min. Electrocardiographic changes was first degree A-V block, wandering pacemaker, inversion of negative to positive T-wave, flattening of the P-wave ventricular premature beat, sinus arrest and bradycardia, sinus tachycardia.

The present finding will be compared largely with those experiment observed previously in animals. The results of rapid potassium chloride infusion are similar to those recorded in calves (Bergman and Seller, 1953) in which infusion lead to clinical signs and death at plasma levels of 8.1 to 12.7 mmol L^{-1}. Plasma potassium level of 8 to 10.1 mmol L^{-1} in these experiments caused cardiac toxicity and probably approach the upper limits compatible with life in the horse.

Tolerance to high potassium diets has been demonstrated in the rat (Wright and Fiebisch, 1971) and the cow (Pickering, 1965). Tolerance to high potassium diet would also be expected in the horse because its diet is similar to the bovine. These experiments demonstrated the very high infusion rates required to elevate plasma potassium and the rapid fall immediately infusion was supported. Rapid infusion of potassium chloride had a great effect on the ECG. As reported by Surawicz and Mazzouleril, 1967), it is the alternation in the transmembrane potassium gradient rather than the absolute amount of potassium present which alters the ECG. Alternation of the transmembrane action potential because of the altered ECG potassium concentration is reflected electrocardiographically. High potassium affects the myocardium in a similar manner to that of experimentally induced hyperkalemia in the dog, the cat (Epstin, 1984), the calf (Bergman and Seller, 1954) and man. The atria are the most sensitive to hyperkalemia (Fisch and Knoubl, 1966) and widening and flattening of the P wave is a constant experimental finding. This is accompanied by alternations in ventricular repolarization reflected in altered T wave, which become progressively more peaked. Further increase hyperkalemia have been associated with arrhythmia and eventual death (Epstein, 1984). Clinically, electrocardiographic changes associated with hyperkalemia have been most extensively studied in man. The first diagnostic signs are peaking of the T wave which may be associated with widening of the QRS complex. Broadening and flattening of the P wave also occurs (Wood, 1968).

The effect of heart rate on the equine T wave has been well documented (Steel, 1963; Holmes and Rezakhani, 1975; Epstein, 1984). Hyperkalemia affects myocardial depolarization and it is possible that this facilities inversion of the T wave (Epstein, 1984). A similar founding in electrocardiographic changes reported by Epstein (1984) in horse injected with potassium chloride. MacKenzie (2007) recorded the presence of widened QRS complexes in hyperkalemia according to this research. Trof and Brouwer (2004) investigated the effects of hyperkalemia in two men, aged 19 and 64, with chronic renal insufficiency and subacute symptoms of malaise and weakness of the leg muscles, broad QRS complexes were seen in the ECG. Norman and Barrett (2006) described three cats with moderate to severe hyperkalemia and concurrent rapid heart rate. In each cat, the serum potassium concentration was > or =7.5 meq dL^{-1} with a concurrent heart rate > 200 beats min^{-1}. In each cat, nine-lead electrocardiograms demonstrated an absence of P waves and a wide-complex tachycardia.

Table 1: Measurement of blood and erythrocytes parameters (mean±SE) in experimentally induced hyperkalemia in the donkey

Time (min)	0	15	30	45	60	75	90	105	120	135
Blood parameters										
Potassium	423.57±3.40	453.50±3.40	892.14±2.85*	996.42±2.60*	1093.57±4.32*	1196.14±3.42*	1094.28±3.52	552.85±3.24	515.00±3.53	510.00±3.53
Magnesium	13.59±0.11	13.19±0.41	13.14±0.23	13.20±0.12	13.95±0.21	14.70±0.45	14.88±0.64	12.39±0.30	11.71±0.08	11.46±0.04*
Sodium	2620.00±4.08	3120.00±4.08	3245.00±4.08	3367.00±3.91*	3620.00±4.08*	4000.00±2.44*	3455.00±2.08*	3245.00±3.84	3117.00±4.63	2997.00±4.33
Calcium	103.45±0.14	111.32±0.18	113.48±0.13	118.34±0.18	160.44± 12*	155.37±0.11*	138.40±0.13*	125.20±0.15*	110.32±0.13	104.42±2.16
Erythrocytes parameter										
Potassium	1040.00±4.08*	1120.00±3.68*	1169.00±4.55*	1186.00±3.40*	1175.00±4.08*	1251.00±4.84*	1161.00±4.84*	1150.00±5.28*	1043.00±4.63*	1003.00±2.39*

*: Significant at $p<0.05$

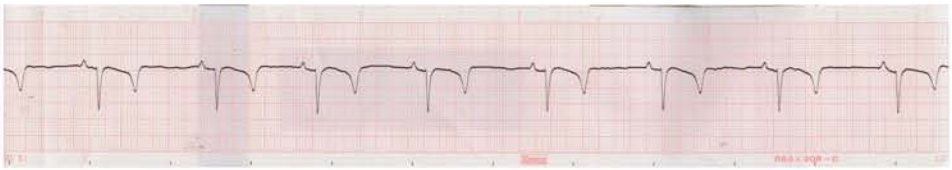

Fig. 1: Normal heart rate before injection of potassium chloride

Fig. 2: Normal with higher heart rate

Fig. 3: Ventricular tachycardia

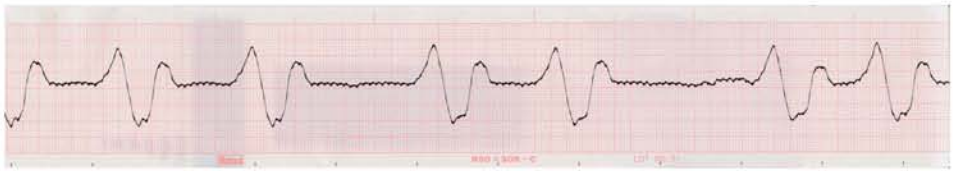

Fig. 4: Ventricular fibrillation

Fig. 5: Sinus bradycardia

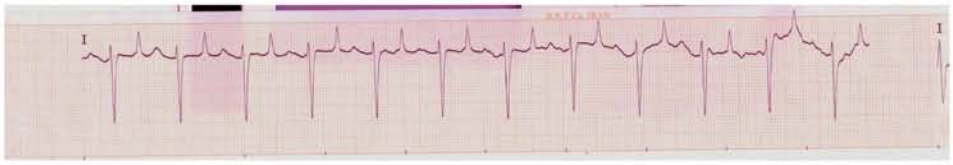

Fig. 6: AV Block I

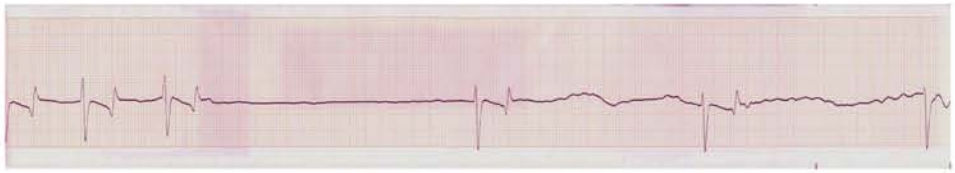

Fig. 7: Cardiac arrest

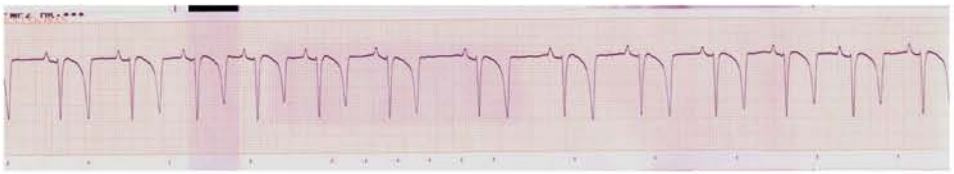

Fig. 8: Sinus tachycardia

CONCLUSION

In conclusion, as a result of potassium content of erythrocytes in horse and donkey is higher than other animals, they tolerate to high potassium infusion and use of isotonic solutions that contain potassium chloride in these animals might be at low life threatening.

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