EVALUATION OF DOG ELECTROCARDIOGRAPHY IN HYPOKALEMIA

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ABSTRACT

The objective of the work was to study Electrocardiography in normal dogs and in dogs treated with intravenous furosemide for 14 days.

In present study eight dogs in different sex and two years age used, serum potassium level determined using commercial kit and ECG evaluate twice daily pre and post furosemide use.

ECG tracing compared in the two groups (treated and control). when serum k⁺ reach (4.4 mEq/L ± 1.044) at day four from starting, and in the last five days of treatment the mean of serum potassium reach (3.2 mEq/L ± 0.504). the electrocardiographic changes shows features of hypokalemia T inversion or flatting of T wave in limb leads (I,II,III),avl, avf and most of the chest leads.

INTRODUCTION

The electrocardiogram (ECG) is the test of choice for diagnosis of abnormal heart rhythms and also can be useful for identification of cardiomegally or drug effect on the heart (1).

An electrocardiogram is a diagnostic test that records the electrical activity of the heart. Canine electrocardiographs are common procedures in a small animal veterinary practice. They are normally used as a diagnostic tool when canine heart disease is suspected and used to monitor heart function in the operating room (2).

There are typical six lead system (placement protocol) and III are called bipolar limb lead and detail the electrical voltage being produce by the heart between two lead (avr, avl, avf) compare cardiac electrical stimulation at one limb to a central point of the heart (3).
A vital element to an important group of blood minerals called electrolytes, potassium function in both cellular and electrical function, such as in the conduction of electrical charges in the heart, nerves and muscle. Therefore lower level of potassium in the bloodstream will compromise the normal functioning of these tissues (4).

Hypokalemia contributes to the pathogenesis of cardiovascular disease, and many cardiovascular disorders and drugs aggravate hypokalemia (5; 6). Hypokalemia is therefore a common, reversible factor in the natural history of cardiovascular disease.

Hypokalemia enhances the propensity for ventricular fibrillation in the normal, as well as the ischemic canine heart (7). Also hypokalemia induce hyper excitability and is clinically manifested by increase in supra ventricular and ventricular ectopy (8).

Diuretic inhibit chloride dependent sodium resorption and induce potassium excretion in a dose dependent manner (9).

Plasma potassium increase about 0.6 m Eq/L for each 0.1 unit decrease in blood pH. The reverse is true for pH increase, thus if an acidic animal has a normal plasma potassium level, it should be considered hypokalemia and corrective therapy should be initiated. Potassium loss may develop as a consequence of vomiting, diarrhea, using some drug such as diuretic, bad use for drug cortisone and using purgative (10).

The electrically active tissue of the heart is sensitive to change in extracellular concentration of K+. Hypokalemia produce flattening and or inversion of T wave, then appearance of U wave then prolongation of QT interval (normal QT<0.4 sec) (11). Hypokalemia cause cellular hyperpolarity increase resting potential, hastens depolarization and increase automaticity and excitability (12).

There was no study on canine electrocardiography, so the objective of this work is the determination of electrocardiograph changes in dogs in case of hypokalemia.

**MATERIAL AND METHODS**

**Animals:**

1- Eight dogs (different sex and about two years age) six dog treated with furosemide (40 mg/daily) and two as a control.
2- ECG used to evaluate all dogs twice daily pre and post administration of furosemide.

3- 5ml of blood collected for serum potassium determination using kit of potassium(Human Gmbh.65205,Wiesbaden,Germany).

ECG Procedure according (3)

1-place a blanket on the table.

2- Place the animal in right lateral decumbency, the spine should be straight and the forelimbs should be perpendicular to the spine and parallel to each other.

3- Shaving the hair ,spray alcohol and spray ECG conductivity solution over the alcohol to provide good contact metal surface, the chest ,or each other.

Red-left leg Green-Right leg
Black-Left arm White-Right arm

4-leads aVR, aVL ,aVF

- Attach the unipolar lead to elbow of the left front leg and the other unipolar lead to the elbow of the right front leg (lead aVR).

Clip the left unipolar lead to the elbow of the left rear leg and the other unipolar lead to the elbow of the left front leg (lead aVL).

Place the left unipolar lead on the elbow of the left front leg and the other unipolar lead on the elbow of the left back leg (lead aVF).

RESULTS AND DISCUSSION

The mean of serum potassium in control animal (5.2 m Eq/ L ±0.673) whereas the mean of potassium in treated animal in the first, second and third day of treatment with furosemide within normal limit (5.9 m Eq/L ± 0.408 ). At the fourth day the mean of serum potassium started to decline reach(4.4 m Eq/L ± 1.044 ) while in the last five days of treatment (9-13days) the mean serum potassium(3.2mEq/L ± 0.5042).

Potassium level in blood decrease in some case like diarrhea, persistent vomiting, diuretic use, use of steroid. potassium play an important role in cell metabolism and excitability, disorders of potassium balance can have profound clinical effects, particularly on the cardiovascular and neuromuscular system(4).

Potassium is the major intracellular cation in mammalian cells and is largely responsible for maintenance of intracellular volume. Serum potassium concentrations slightly exceed plasma concentrations because potassium is released from platelets.
during the clotting process. Potassium is responsible for maintaining resting cell membrane potential. Therefore, disorders of potassium concentration affect excitable membranes (13).

The ECG recorded daily for any evidence of hypokalemia in the first day of trial was normal in control and treated animals. As in figure (1). While in the fourth day ECG tracing start to show features of hypokalemia. T inversion or flating of T wave in the limb leads (I,II,III) aVL, aVR and aVF and most of the chest leads as shown in figure (2). In the following days of treatment the rhythm continue to be sinus figure(3), but diffuse T wave inversion continue to appear and suspicious U wave seen in lead II (V5) QT interval continue to be normal figure(4).

Hypokalaemia was also associated with changes in the morphology of the T wave recorded in CV5, in particular, with a flattening and/or a notching of the wave (14).

Potassium is the most abundant intracellular cation and hypokalemiais the most commont electrolyte abnormality encountered in clinical practice. The most significant ECG manifestation of hypokalemia is a prominent U wave. Several cardiac and non cardiac drugs are known to suppress the HERG K+ channel and hence the IK, and especially in the presence of hypokalemia, can result in prolonged action potential duration (15).

An electrocardiogram, is a non-invasive way to look at the electrical activity of the heart. The various spikes, bumps and waves on the ECG tracing follow a specific pattern, this may be a signal that something is wrong with the heart. In the case of a patient with hypokalemia, there's an extra "bump" on the ECG tracing, called a "U" wave, it is not clear what electrical activity is occurring in the heart when the "U" wave appears on the ECG tracing. What is known is that a prominent "U" wave is often an indication of hypokalemia (16).

The U wave is a low amplitude deflection following the T wave which is mostly observed in V2 and V3 precordial leads. The mechanism behind the U wave form is still being investigated but it has been identified in cases of hypokalemia (17).

and (18) show Pathologic U waves may be seen in ischemic heart disease where they sometimes point to acute ischemic events. The large U waves of hypokalemia are most likely not true U waves but rather the terminal deflection in abifid T wave.

The adverse association between hypokalemia and arrhythmia in animal models appears to be more significant in the presence of acute myocardial ischemia (19).
Figure 1
Normal ECG
Calibration: 20 mm (2mv)
Speed: 25 mm/s
All ECG waves are represented: P, QRS, and T wave

Figure 2
Calibration: 20 mm (2mv)
4 days after IV Frusemide 20 mg twice daily
T wave inversion in leads II, III, AVF, and chest leads VI, VII, VII (hypokalaemia)
Figure 3
Sinus rhythm. T inversion in lead I, II, AVL, AVF and minimal T wave changes in leads VI, VII, and VIII

Figure 4
Sinus rhythm. Inverted T in leads II, III, AVF, VI, VII and flattened T in V5, V6
تقييم مخطط القلب الكهربائي في الكلاب في حالة انخفاض البوتاسيوم

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الخلاصة

هدفت الدراسة إلى تقييم تخطيط القلب الكهربائي في الحالة الطبيعية وفي حالة انخفاض نسبة البوتاسيوم في الدم بعد إعطاء عقار furosemid بجرعة 40 ملغ يومًا لمدة 14 يوم.

استخدمت في الدراسة ثمان كلاب عمر سنتين وجمعية مختلفة. تم تقييم مستوى البوتاسيوم في مصل الدم ومقارنة التغير في تخطيط القلب الكهربائي في مجموعة السيطرة ومجموعة المعالمة بعقار furosemid.

وحسب انخفاض مستوى البوتاسيوم في الدم في اليوم الرابع حيث كان (4.4 ± 1.044 mg/L) من 3.2 mg/L (± 0.504). وتم إجراء العملية القدرة الأخرى من المعالمة كان معدل البوتاسيوم في البتبيب furosemid المعالمة بعقار تجريح في وظائف القلب الكهربائي حسب تطور وانخفاض سالب للموجة T في أقطاب الاطراف ومعظم الأقطاب الصدر.

REFERENCES


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