The Thai Journal of Veterinary Medicine

Volume 43
Issue 2 *June, 2013*

Article 23

6-1-2013

ECG Quiz

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History

The 5 years old mixed breed spayed female weighing 15 kg was presented at the Small Animal Hospital, Faculty of Veterinary Science, Chulalongkorn University with history of severe depression, anorexia and vomiting. Ten months ago, the dog was azotemia with creatinine of 4.5 mg/dl and anemia with PCV of 11%. Dog was given supportive therapy, doxycycline and immuno-suppressive drugs. Treatment and monitoring had performed for five months, after which BUN and creatinine were declined to 43 mg/dl and 3.2 mg/dl, respectively while PCV was increased up to 34%. Chronic kidney disease was maintained although she was still alert. The dog had never seen the veterinarian after since. He then returned to the veterinarian with clinical signs of azotemia. He had epistaxis, which was treated two days before presenting to the hospital.

From physical examination, the dog had pale mucus membrane with normal heart and lung sound. At the hospital, dog had bloody diarrhea and anuria. Systolic blood pressure was 110 mmHg with bradycardia (heart rate was 45 beats/min). From blood results, the dog was positive for antibody against *Ehrlichia canis* using Snap 4Dx®. He had low red blood cell with PCV of 24%, 5.1 g/dl of total protein and leukocytosis (22,300 cell/mm^3). Blood chemistries showed azotemia with increased BUN and creatinine up to 104 mg/dl and 9.4 mg/dl, respectively. The concentration of inorganic phosphate was increased up to 10.1 mg/dl. Plasma Na and K were 142 and 4.53 mEq/L. Plasma HCO^{3-} was 18.6 mmol/L with pH of 7.391. Acute kidney injury was diagnosed and the dog was treated with furosemide, dopamine and mannitol to increase urine volume. Supportive fluid therapy, anti-emetic and antibiotics were also given. The dog was still oliguric with urine output between 0.05-0.34 ml/kg/hr. The BUN and creatinine measured 5 days after admission were increased to 191 mg/dl and 11.4 mg/dl, respectively. Plasma K was 6.46 mEq/L by the time the ECG was performed (Figure 1).

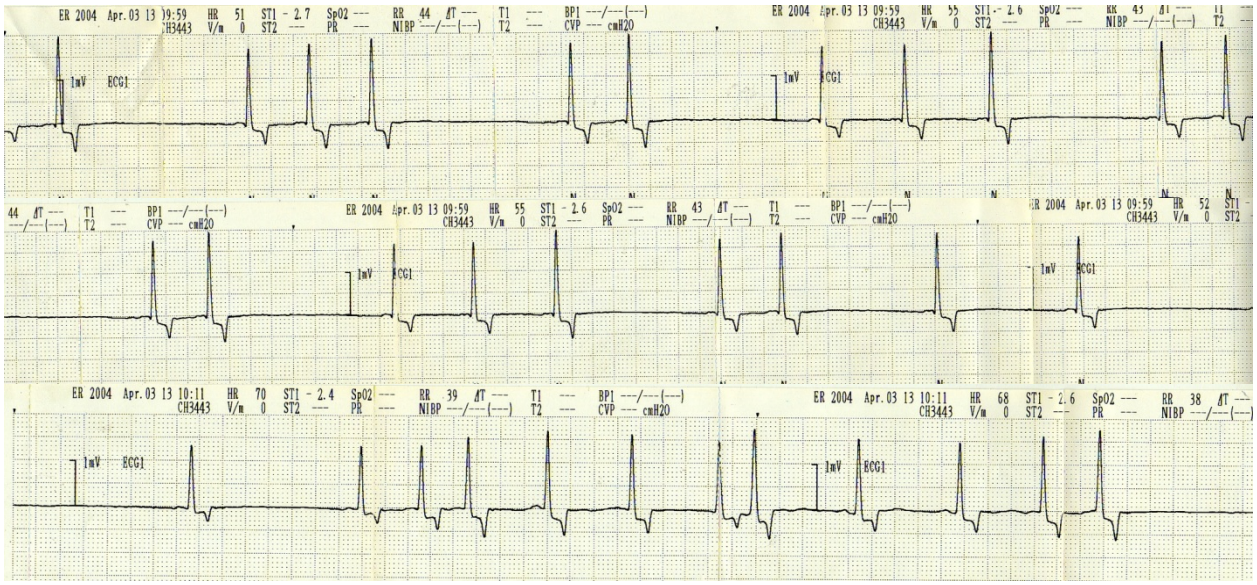

Figure 1 Electrocardiographic recording on dogs with acute kidney injury

Please answer before turning to the next page.

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Interpretation Sinus bradycardia without p-wave

The heart rate during resting was between 50-55 beats/minute. The shape of QRS complexes was similar to that originated from normal sinus node. However, the p-waves were very small or sometimes could not be detected suggesting that the conduction insight the atria may be slow. One supraventricular ectopic beat was found as shown at the 8th wave of the last panel of ECG tracing. Since the ECG tracing was performed by the telemetric ECG monitoring in the CCU unit, the conventional 10 leads of ECG should have been performed. The cause of bradycardia and loss of p-wave was due to electrolyte abnormalities especially potassium ion. Hyperkalemia in this case caused by oliguric kidney failure leads to bradycardia and many forms of ECG abnormalities. Besides p-wave abnormalities, QRS widening, fusion of QRS and T-wave, tall and tented T-wave are also seen when the hyperkalemia is more severe. Hyperkalemia causes Na^+ channel inactivation and speeds up membrane repolarization leading to tenting T-wave. Inactivation of Na channel causes slow conduction as shown by small or loss p-wave and QRS widening. Bradycardia is due to hyperpolarization by high potassium level, in which depolarization of phase 4 is prolonged. In severe hyperkalemia (K^+ > 9 mmol/L), the electrocardiographic sine-wave pattern will be seen. When arrhythmia is detected with serum potassium higher than 6.5 mmol/L, treatment should be warranted. Calcium chloride or calcium carbonate should be given to stabilize cardiac membrane potential. Insulin along with glucose can be given to accelerate the K^+ influx into the cell. Bicarbonate is another choice of drug that promotes K^+ influx and correct acidosis. Finally, if urine output is low and K^+ cannot be eliminated, hemodialysis or peritoneal dialysis should be considered.