The Thai Journal of Veterinary Medicine

Volume 41
Issue 3 *September, 2011*

Article 18

9-1-2011

ECG Quiz

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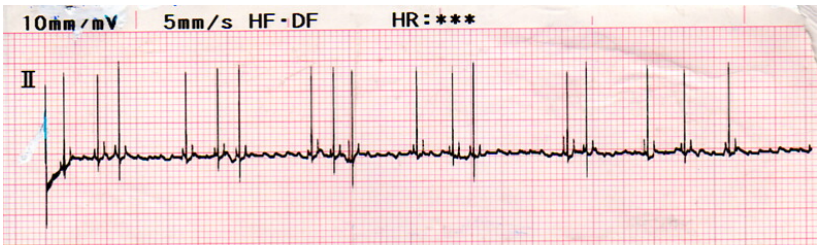
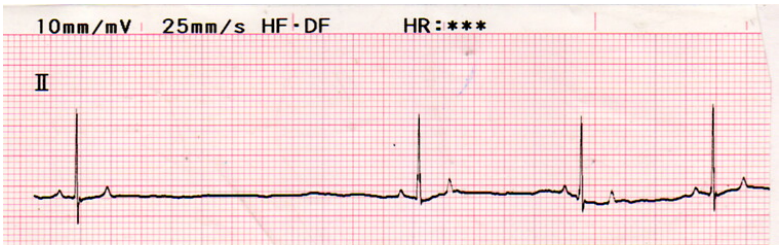
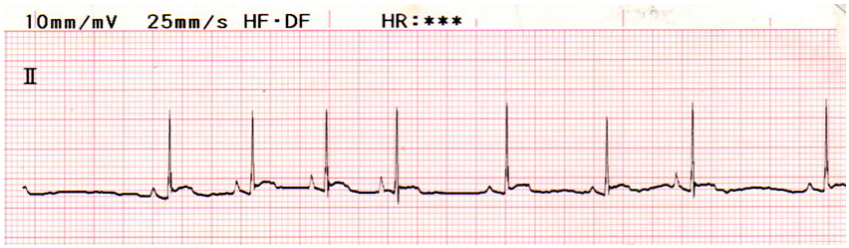
Tracing I - 5 mm/sec



Tracing II - 25 mm/sec



Tracing III - 25 mm/sec



History

A twelve years old female Shih Tzu weighing 5.4 kg came to the animal hospital with a history of exercise intolerance, insomnia, severe depression and vomiting for one week. The dog had previous history of chronic coughing and exercise intolerance while heart disease had been diagnosed at a private hospital. The thoracic radiograph showed enlargement of cranial mediastinal lymph node, mild interstitial and bronchial infiltration of lung and mild cardiomegaly. Twenty days earlier, the dog received the angiotensin converting enzyme inhibitor (enalapril; 0.23 mg/kg, bid), furosemide (1.8 mg/kg, bid), and antiobiotics (amoxycillin-clavulanate). A week later, the dog did not respond to treatment and digoxin was added at the dose of 0.116 mg/kg twice a day. Hematuria was also noticed by the owner at this time. Another week later, the bronchodilator, terbutaline was prescribed additionally. Blood work one week before the animal came to the hospital showed normal results (complete blood count, kidney and liver panels, plasma concentrations of protein, calcium phosphorus and bilirubin). At the day of admission, physical examination revealed severe depression with little response to external stimuli. The dog had normal heart sound with bradyarrhythmia. Abdominal ultrasonography showed normal liver, spleen, kidneys and bladder. Echocardiography showed thickening of interventricular septum and left ventricular wall with no mitral valve regurgitation. The mild left atrial enlargement was found with LA:Ao = 1.7. The electrocardiography showed in tracing I and II with different paper speed. The medications were stopped and the blood collection was performed to evaluate the plasma digoxin level. The supporting treatment with fluid therapy was given and dog's clinical signs were improved on the next day. Repeated measurement of electrocadiography was performed a week later after digoxin discontinuation and the result was shown in tracing III. Another positive inotropic agent, pimobendan was prescribed instead of digoxin along with furosemide and angiotensin converting enzyme inhibitor. The dog was alert and clinically normal thereafter.

Please answer before turning to the next page.

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Interpretation

Tracing I, II– sinus bradyarrhythmia
Tracing III – Respiratory sinus arrhythmia

On the day of presentation, heart rate of this dog was as low as 45 beats per minute as shown in tracing I and II. Since the dog showed sign of severe depression with insomnia, it was suggested that hypotension with low cardiac output may encounter. The results of blood chemistry profiles and ultrasonography did not indicate any systemic diseases. Therefore, digoxin toxicity was suspected in this case. Digoxin is a positive inotropic agent that is widely used to treat animals with low output due to congestive heart failure. This drug is rarely used in acute cardiac cases because of its slower onset of action and increased potential of intoxication due to narrow therapeutic index. The plasma digoxin level in this dog was 5.3 ng/ml, which was high compared with the normal range between 0.8-2.5 ng/ml. However, 1.0-1.8 ng/ml is considered safe in canine patient. Given high dose of digoxin in either patients with pulmonary disease or impaired renal function can cause digoxin toxicity with calcium overload. One mechanism of action of digoxin is a blockade of Na^+, K^+-ATPase activity of the myocardial cells. This effect causes the suppression of Na^+-Ca^{2+} exchanger resulting in high intracellular calcium. The clinical signs may include gastrointestinal symptoms such as anorexia, nausea, vomiting and diarrhea. The neurological signs are fatigue, insomnia and even syncope. For cardiac action potential, digoxin toxicity can cause delayed afterdepolarizations with high risk of ventricular ectopy typically bigeminy and paroxysmal atrial tachycardia. Another action of digoxin is excess vagal stimulation. Therefore, the bradyarrhythmia is found frequently. High vagal tone may cause second or third atrio-ventricular block. During admission, dog's electrocardiogram showed bradyarrhythmia with slightly prolonged PR interval (tracing I and II). Both characteristics were resolved after discontinuation of digoxin with heart rate was increased up to 100 beats per minute (tracing III). The treatment protocols depend on the clinical signs and severity. Withdrawal of digoxin and waiting for recovery with supportive care may be enough to resolve the problem. Lidocaine and potassium supplementations may be used for some type of arrhythmia but not in AV conduction block. Plasma potassium concentration should be evaluated in all cases suspected for digoxin toxicity since hypokalemia predisposed the patient for higher risk of toxicity. In this case, pimobendan was used to replace digoxin to enhanced cardiac contractility with lower risk of toxicity.