Electrocardiographic manifestation of digoxin toxicity in a Pomeranian dog

Malmasi, A.[1]*, Selk Ghaffari, M.[2], Soroori S.[1]

[1]*Department of Clinical Sciences, Faculty of Veterinary Medicine, University of Tehran, Tehran- Iran.*

[2]*Department of Clinical Sciences, Faculty of Veterinary Medicine, Islamic Azad University-Karaj Branch, Karaj- Iran.*

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Abstract: **This case report describes several electrocardiographic findings in an 8-years old female Pomeranian dog that was referred with a history of digoxin overdose. Electrocardiographic abnormalities detected in this case could be classified as primary electrocardiographic disturbances, reflecting cardiac chamber enlargement and secondary ECG changes, attributed to digoxin toxicity. ECG manifestations of digoxin toxicity such as first and second degree AV blocks completely disappeared following digoxin withdrawal. Therefore, veterinarians should be aware of the possibility of digoxin toxicity due to its narrow therapeutic margins. The purpose of this report is to increase awareness of small animal practitioners about adverse cardiotoxic effects of cardiac glycosides. It also highlights the significance of electrocardiographic monitoring for diagnosis and management of digoxin induced cardiac arrhythmias and conduction disturbances in small animal patients.**

Key words: digoxin, digoxin toxicity, dog, electrocardiogram.

Introduction

Digoxin and digitoxin are the oral preparations of cardiac glycosides. They have multiple effects on cardiac muscle connective tissues. Both exert a positive inotropic effect on cardiac muscle by directly binding to the membrane sodium-potassium ATPase pump and impairing active transport of these ions. The increase in intracellular sodium causes a net influx of calcium, which results in an increased concentration of calcium in the sarcoplasm. This is supposedly responsible for increasing the force of myocardial contractions (Cote, 2005; Sharff, 1982). For drugs with narrow therapeutic windows, such as digoxin, toxicity may occur even during routinely prescribed doses (Woodland *etal*., 2003). Electrocardiography is a well-established technique in veterinary practice for detecting conduction disturbances and toxic effects of cardiac glycosides (Kittleson, 1998). The present report describes electrocardiographic manifestation of digoxin toxicity in a dog to increase awareness about unrewarding cardiotoxic effects of cardiac glycosides in small animals and also emphasizes on the electrocardiographic monitoring in cases which receive cardiac glycosides.

* **Corresponding author's email: Malmasia@vetmed.ut.ac.ir, Tel: 021- 61117151, Fax: 021-66933222, Mob: 09121932395**

Case Report

An 8-years old female Pomeranian dog was referred to our hospital with vomiting, anorexia and lethargy. The dog had a history of congestive heart failure (CHF) and had been receiving an inotropic agent (digoxin 0.003 mg/kg PO twice daily), an ACE inhibitor (Enalapril 0.25 mg/kg PO twice daily) and a loop diuretic (Furosemide 2 mg/kg PO once a day) since 6 months prior to her deterioration and was in a stable condition. It seemed that the dog's condition after a long walk with the owner deteriorated due to exercise intolerance and after a day, the owner without a veterinary specialist consultation doubled the dose of digoxin. After 72 hours the dog became anorexic and showed signs of vomiting (up to 10

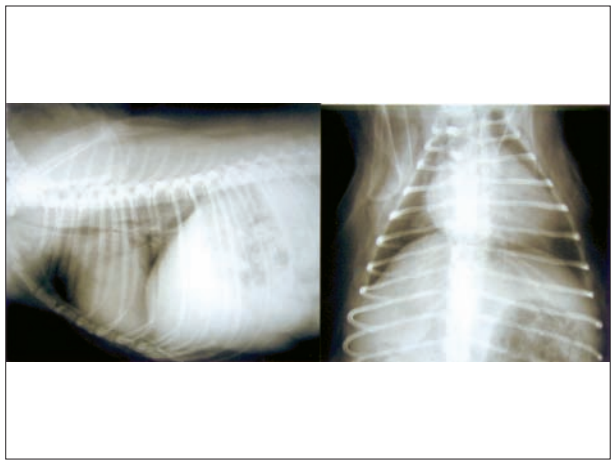

Fig. 1: Lateral radiograph shows bilateral heart enlargement. The trachea and caudal vena cava are elevated. The right heart border is rounded, and sternal contact is increased. On ventrodorsal view, both right and left cardiac borders become closer to the thoracic wall. Alveolar pattern is indicative of pulmonary edema.

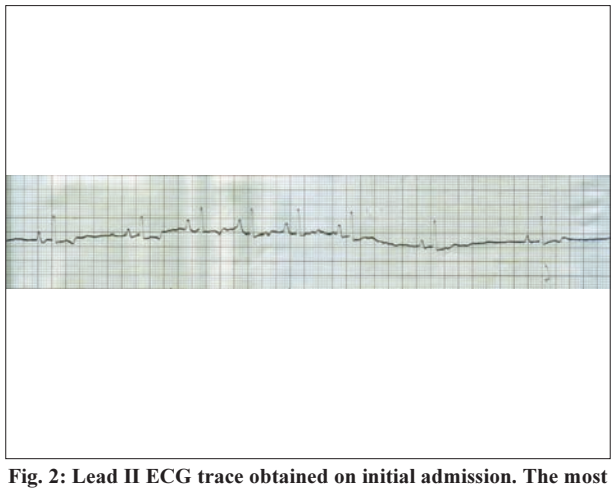

Fig. 2: Lead II ECG trace obtained on initial admission. The most important findings in the first P- QRS-T complex include tall P wave (0.5 mv) and Ta wave. In the second complex, P wave becomes wide and tall (0.05 sec; 0.6 mv). Furthermore P-R interval is prolonged (0.18 second) which indicate first degree AV block. The P wave that follows second beat is not conducted to ventricle (second degree AV block) (Paper speed: 50 mm/second; 1 cm =1mV).

times a day), depression and lethargy. The dog was then referred to the Small Animal Hospital of Tehran University for further diagnostic evaluation and treatment. At initial presentation, the dog seemed to have a moderate respiratory distress. Thoracic examination indicated increased respiratory sounds. Cardiac auscultation revealed a heart rate of 80 beats/minute and a marked irregularity in the heart rhythm. Thoracic radiographs showed bilateral cardiac enlargement. Vertebral heart scale was estimated to be 11 (normal

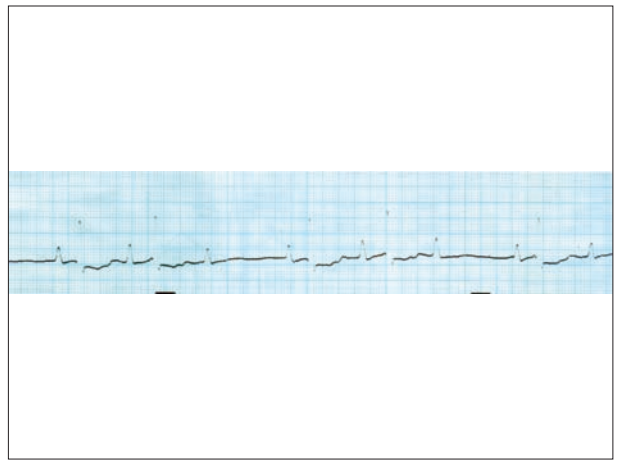

Fig. 3: Lead II electrocardiogram trace recorded in the fourth day of presentation. All ECG variables are within normal limits (paper speed: 50 mm/second; 1 cm = 1mV).

range is between 8.5-10.5 vertebral bodies). A marked increasing in lung opacity was presented in the hilar region which is indicative of alveolar pattern (Fig.1). Results of blood tests were unremarkable. Electrocardiography was then performed. The first P-QRS-T complex was normal except for a tall P wave (0.5 mv), and the presence of Ta wave. The second complex had an abnormally prolonged and tall P wave (0.05 sec; 0.6 mv), and a considerably prolonged P-R interval (0.18 second; reference range: 0.06-0.13 second), which was indicative of first degree AV block. Following the first and second P-QRS-T complexes, a P wave appeared on the ECG trace, which was not followed by QRS-T complex. This ECG criterion indicated second degree AV block. The atrioventricular conduction ratio was 2:1. Repeated electrocardiograms again showed similar ECG findings (Fig. 2). Based on the expressions of the owner on overdosing the digoxin tablets and the presence of clinical signs and electrocardiographic findings, the possibility of digoxin toxicity was considered. All the medications including digoxin promptly discontinued for 48 hours and instead, supportive therapy considered. All the unrewarding clinical signs disappeared gradually and animal's general condition improved later. Four days following admission, the clinical examination and the recorded electrocardiogram showed no previous conduction abnormalities including AV blocks, which resolved promptly after discontinuation of overdosed medication (Fig. 3). The treatment with digoxin was started again. The owner advised about careful dosing of

prescribed digoxin. No recurrence of AV blocks had been observed during follow up period up to now.

Discussion

The electrocardiographic abnormalities during initial admission in this case could be classified as the primary electrocardiographic disturbances, reflecting cardiac chamber enlargement and secondary ECG changes attributed to digoxin overdose. The P wave represents atrial depolarization. Atrial enlargement may result in an increase in width or height of the P waves recorded in lead II (Tilley and Good win, 2001). Our findings in regard to concurrent wide and tall P waves in the electrocardiograms indicated biatrial enlargement. The presence of Ta waves was a very interesting finding on initial electrocardiogram. This ECG criterion was also related to cardiac chamber enlargement. Ta waves are characterized by slight depression of baseline following the P waves. It is sometimes seen in atrial enlargement. This represents atrial repolarization. The Ta wave can also be seen in very rapid heart rates (Tilley, 1992). The estimated heart rate in ECG trace of this case was 80 beats/minute, thus the presence of Ta waves seemed to be directly related to biatrial enlargement. We concluded that the concurrent first and second degree AV blocks were directly correlated to digoxin toxicity, as these conduction abnormalities disappeared completely after digoxin withdrawal. Abnormalities of AV conduction can be caused by excessive vagal tone, drugs (e.g., Digoxin, opioids), and organic disease of AV node (Nelson, 2003). Digitalis glycosides produce indirect and direct anti-arrhythmic effects. Most of the indirect antiarrhythmic actions of these agents are due to an increase in parasympathetic tone and sympathetic inhibition. Toxic doses of digitalis cause cellular depolarization and marked slowing of conduction in atrial and ventricular specialized fibers and AV node. These actions can produce a wide variety of conduction disturbances, including first, second, and potentially third-degree AV blocks, right and left bundle branch blocks, and reentrant atrial or ventricular arrhythmias (Fox, 1999). Based on the results of first ECG, the AV conduction ratio was 2:1. This finding is similar to those human cases of digoxin overdose. This pattern was well-described by several researchers (6). The type of presented second degree atrioventricular block in our case was Mobitz Type I. In this type of AV block, there is progressive prolongation of PR interval, followed by occurrence of second degree AV block (Tilly, 1998). In this dog, we did not consider specific therapy for detected conduction abnormalities. Conduction disturbances and bradyarrhythmias usually require only digitalis withdrawal, but ventricular arrhythmias are generally treated aggressively, especially when ventricular tachycardia is present (Kittleson, 1998), which were absent in our case. Results of recorded electrocardiogram in the fourth day of admission did not reveal previous cardiac chamber enlargement patterns. This finding was in agreement with the opinion that the major shortcoming of the ECG is its lack of sensitivity for inferring cardiac chamber enlargement or hypertrophy by changes in the P-QRS-T voltage (Fox, 1999). It is often normal, even with demonstrable chamber enlargements; also the left atrial enlargement may result in a wide and sometimes notched P wave; however, these abnormalities may be detected in as few as 40% of patients with moderate-to-severe enlargement (Kittleson, 1998).

In conclusion, the clients should be informed about adverse effects of digoxin overdosing. In such cases, early diagnosis of digoxin toxicity is highly advised for the practitioners in order to prevent unexpected fatal complications of digitalis glycosides overdoses. The potential clinical and cardiac complications require careful clinical and electrocardiographic monitoring.

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یافته های الکتروکاردیوگرافی مسمومیت با دیگوکسین در یک قلاده سگ پومرانین

عبدالعلی ملماسی [۱*] مسعود سلک غفازی [۲] سارنگ سروری [۳]

۱) گروه علوم درمانگاهی، دانشکده دامپزشکی دانشگاه تهران، تهران-ایران.

۲) گروه علوم درمانگاهی، دانشکده دامپزشکی دانشگاه آزاد اسلامی-واحد کرج، کرج-ایران.

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چکیده

در این گزارش یافته‌های الکتروکاردیوگرافی در اثر مسمومیت ناشی از دیگوکسین در یک قلاده سگ هشت ساله پومرانین ماده مورد بحث قرار گرفت. مجموعه یافته‌های الکتروکاردیوگرافی در این بیمار در دو زیر مجموعه شامل تغییرات اولیه که بیانگر بزرگ شدگی حفرات قلب بودند و تغییرات الکتروکاردیوگرافی ثانویه ناشی از مسمومیت با دیگوکسین ، دسته‌بندی شد. علایم الکتروکاردیوگرافی مربوط به مسمومیت با دیگوکسین مانند بلوک‌های درجه اول و دوم دهلیزی بطنی متعاقب قطع دارو کاملا برطرف گردید. دامپزشکان باید از احتمال مسمومیت با دیگوکسین مطلع باشند چرا که دیگوکسین دارای حاشیه درمانی بسیار محدود می‌باشد. هدف از این مقاله افزایش آگاهی دامپزشکان فعال در زمینه دام‌های کوچک در رابطه با اثرات کاردیوتوکسیک گلیکوزیدهای قلبی می‌باشد. علاوه بر این اهمیت بررسی‌های الکتروکاردیوگرافی در تشخیص و مدیریت آریتمی‌های قلبی و اختلالات هدایتی ناشی از دیگوکسین در دام‌های کوچک مورد تاکید قرار می‌گیرد.

واژه‌های کلیدی: دیگوکسین، مسمومیت با دیگوکسن، سگ، الکتروکاردیوگرام.

*) نویسنده مسؤول: تلفن همراه: ۰۹۱۲۱۹۳۲۳۹۵ نمابر: ۶۶۹۳۳۲۲۲-۰۲۱ Email:Malmasia@vetmed.ut.ac.ir