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Original Article

Hematologic and electrocardiographic findings in sub-acute experimental monensin toxicosis in goats

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Abstract

Toxic effects of monensin, a polyether antibiotic mainly used as coccidiostat, have been described in a wide range of animals. The present study aimed to investigate the hematologic and electrocardiographic features of sub-acute monensin toxicosis in goats. For this purpose, seven adult goats were administered sodium monensin, 13 mg/kg, daily for 5 consecutive days via gastric gavage. Hematologic parameters including PCV, hemoglobin (Hb), total white blood cell (WBC) and differential count, total protein of plasma (TPP) and fibrinogen, were determined in baseline and daily blood samples for 10 days. Significant elevation of Hb at day 1, WBC at day 7, neutrophils percent at days 5 and 8, lymphocytes percent at days 5, 8 and 9, monocytes percent at days 1 and 3, absolute numbers of monocytes at days 1, 3, 6 and TPP at day 1, were observed in monensin exposed goats. At electrocardiography, sinus tachycardia, sinus bradycardia, S-T segment depression, and ventricular premature complexes and ventricular tachycardia were the most prominent findings. These findings suggest that sub-acute monensin toxicosis in goats, alters some hematologic parameters and causes a numbers of electrocardiographic abnormalities related to toxic cardiomyopathy in exposed goats.

Key words: Monensin, Ionophores antibiotic, Goats, Electrocardiography, Arrhythmia

Introduction

Monensin is an antibiotic produced as a byproduct of fermentation by *Streptomyces cinnamonensis* which belongs to a family of drugs known as polyether antibiotics or ionophores. It was discovered in 1967 by Agtarap et al. as a metabolite formed in a biosynthesis of aforementioned bacteria. Monensin was the first antibiotic that showed an effect at practical concentrations for incorporation in feed as an anticoccidial agent (Chapman et al, 2010). However, in the recent years it has widely been used as a feed additive to improve performance in livestock production systems (Duffield et al, 2012). Monensin has also minor additional uses in the treatment of ketosis, lactic acidosis, bloat and acute pulmonary edema and emphysema (Constable et al, 2017).

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Monensin has a low therapeutic index and may be fatal in certain species when used in excessive doses. Accidental monensin toxicosis, usually occurs after mixing errors that result in its inclusion in feeds of non-target species or in excessive concentration in the diets of target species (Novilla, 2007). Monensin toxicosis has been reported in cattle (Basaraba et al, 1999; Gonzalez et al, 2005), water buffaloes (Garcia et al, 2020; Silva et al, 2022), sheep (Jones, 2002; Mendes et al., 2003), horses (Peek et al., 2004; Gy et al 2020), swine (Miskimins and Neiger, 1996), chickens (Zavala et al, 2011), ostriches (Dedoussi et al, 2007), deer (Glover and Webeser, 1983), camels (Mu) and dogs (Vitello and Good 2023). Cases of accidental monensin intoxication have also been recorded in humans (Zhang et al, 2018). Monensin is approved for use in non-lactating goats (Smith and Sherman, 2009). However, field cases of monensin toxicosis rarely reported in goats (Anios et al, 2023). Experimental data has indicated that the LD_{50} for monensin in goats is 26.4 mg/kg body weight (Beasley, 1999).

Susceptibility to monensin toxicity varies considerably between species. Horses are usually sensitive to monensin and other ionophores intoxication and fish are the most tolerant to high levels of ionophores. The LD_{50} of monensin in horses is as low as 1.4 mg/kg, while its LD_{50} for chicken, the least sensitive species, is 214 mg/kg (Novilla, 2007). Monensin toxicity may also be potentiated by the concurrent use of various antibiotics, including tiamulin, oleandomycin, chloramphenicol, macrolides, and sulfa drugs (Barsaraba et al, 1999; Novilla, 2007). Electrocardiographic changes have not been reported in monensin intoxicated goats.

In this study, hematological and electrocardiographic features of sub-acute monensin intoxication were demonstrated in goats.

Materials and Methods

Animals and treatments

Seven clinically normal local-breed, female, non-lactating and non-pregnant goats that weighed 35-40 kg and aged 2-4 years, were purchased from a local market for use in the study. Prior to commencement of the experiment, goats were dewormed by subcutaneous injection of ivermectin and oral administration of rafoxanide (at the dose of 0.22 and 7.5mg/kg, respectively) and fed for 14 days to ensure proper acclimation. Monensin powder (Monensin 10%) was obtained from Razak Co., Iran. Fresh water was available all the time. Monensin was administered orally via orogastric tube at the dose of 13 mg/kg body weight daily for 5 days.

Blood collection and serum biochemistry

Venous blood samples were collected in the tubes containing anticoagulant (EDTA) for hematology and determination of plasma total protein (TPP) and fibrinogen, on day before monensin administration and daily until day 10. Hemoglobin (Hb) and total white blood cells (WBC) was measured by an automated cell counter (Nihon kohden, MEK 64500). Packed cell volume (PCV) was determined by using microhematocrit method. Differential count of white blood cells (WBC) were undertaken by using the manual method. The total protein was determined by refractometry and the fibrinogen concentration was measured by the heat-precipitation and refractometry method.

Electrocardiography

The baseline of the electrocardiogram (ECG) was recorded using base-apex leads (Constable et al, 2017). Electrocardiography was done using an electrocardiograph (Kenz ECG 110, Suzuken co- Ltd, Japan) on the day before starting the experiment and daily until the day 10.

Statistical analysis

Statistical analysis was conducted using SPSS for windows (release 16, SPSS Inc, Chicago, USA). Based on Kolmogorov–Smirnov normality test, non- parametric Friedman and Wilcoxon Signed tests were used to investigate significant differencees within group for measured parameters. For all comparisons, P≤0.05 was considered as significant.

Results

Hematology

Friedman test showed a significant difference in Hb and TPP levels during the experiment. Wilcoxon signed test showed the following results when the data was compared with the baseline levels: Hb was significantly increased (P<0.05) at day 1. Elevation of WBC at day 7 was significant (P<0.05) (Table 1). There was a significant elevation of neutrophils (P<0.05) at days 5 and 8. Lymphocytes was significantly increased (P<0.05) at days 5, 8 and 9 (Table 2). Monocytes was significantly increased (P<0.05) at days 1 and 3. Also, absolute numbers of monocytes were significantly increased (P<0.05) at days 1, 3 and 6. There was a noticeable increase in total protein of plasma at day 1 (P<0.05) (Table 3). Monensin exposure did not cause any significant changes in eosinophil counts and plasma fibrinogen concentration (Tables 4 & 5).

Table 1: Levels of PCV, Hb and WBC in goats with sub-acute monensin intoxication

Day	N	PCV (%)			P ★ value	Hgb (gram/deciliter)			P ★ value	WBC (per/microliter)			P ★ value
		25th	50th	75th		25th	50th	75th		25th	50th	75th	
0	7	32	35	37	-	8.2	8.5	9.2	-	12000	12600	13100	-
1	7	36	38	44	0.063	9.3	9.7	10.9	0.018	12400	12600	13000	0.499
2	7	31	36	40	0.344	7.7	8.9	10.2	0.600	12000	13200	15400	0.075
3	7	31	36	43	0.611	7.2	8.5	10.1	0.600	12300	13000	13800	0.612
4	7	27	30	35	0.093	7.5	8.5	10.2	0.500	11000	12000	14700	0.735
5	6	28	32	35	0.225	7.725	8.7	9.55	0.674	12500	13600	14500	0.116
6	6	28.75	31.5	32.75	0.080	8.075	8.55	9.15	0.400	12100	13200	14300	0.279
7	6	30.75	32	33.75	0.104	8.25	8.85	9.1	0.528	12200	13250	14000	0.046
8	6	32	34.5	36.5	0.916	8.35	9.3	9.9	0.686	13000	14150	14900	0.116
9	6	31.5	35	37.5	0.915	8.4	9.25	9.725	0.917	12600	13550	14600	0.115
10	6	32.25	35.5	38.25	0.686	8.25	9.5	10.225	0.465	12100	13850	14200	0.249
P value ★ ★		0				0.051				0.193			

Table 2: Neutrophils and Lymphocytes in goats with sub-acute monensin intoxication

Day	N	Neutrophils (%)				Neut (per/Microliter)				Lymphocytes (%)			
		25th	50th	75th	P ★ value	25th	50th	75th	P ★ value	25th	50th	75th	P ★ value
0	7	27	37	38	-	3406	4560	4736	-	59	61	71	-
1	7	30	35	36	0.611	3458	4030	4464	0.735	57	61	68	1
2	7	29	35	38	1.000	3696	4576	4837	0.237	59	64	68	0.833
3	7	28	35	36	0.233	3861	4428	4550	0.866	60	62	70	0.715
4	7	27	29	40	0.498	3300	3822	4635	0.735	59	66	70	0.463
5	6	41.025	48.89	53.8	0.028	4102.5	4889.5	5380	0.463	78.995	86.08	90.5	0.028
6	6	30.25	35.5	38.5	0.768	3516	4631.5	5370.5	0.753	57.75	61.5	68	0.752
7	6	27.75	31.5	35.5	0.207	3689.3	4035	4661.5	0.917	62.25	64.5	68.5	0.249
8	6	24.75	30.5	35	0.043	3298	4319.5	4856.3	0.917	61.75	66	72.75	0.043
9	6	28.5	30.5	33.75	0.116	3913.5	4041.5	4547.3	0.917	81.705	88.685	94.618	0.028
10	6	28.75	31	35.5	0.207	3672.3	4352	4875.5	0.463	67	67	68.5	0.072
P value ★ ★		0.385				0.854				0.658			

Table 3: Monocytes and TPP in goats with monensin intoxication

Day	N	Monocytes (%)				Monocytes (per/microliter)				TPP (gram/deciliter)			
		25th	50th	75th	P ★ value	25th	50th	75th	P ★ value	25th	50th	75th	P ★ value
0	7	1	1	1	-	125	128	133	-	7.3	7.5	7.7	-
1	7	1	2	3	0.039	130	248	378	0.028	7.9	8.4	8.9	0.018
2	7	0	1.31	4.8	0.176	0	131	480	0.310	7.4	7.7	8.6	0.207
3	7	1	3	5	0.041	123	390	585	0.028	7.1	7.7	8.1	0.553
4	7	1	1	4	0.102	118	153	441	0.128	7	7.5	7.9	0.734
5	6	1	2	2.25	0.059	88.5	267	330.75	0.116	7.2	7.4	8.125	1
6	6	1	1.5	3.25	0.109	125	227	425.25	0.028	7.2	7.6	8	0.590
7	6	0	2	3	0.330	0	252	406.5	0.173	7.275	7.75	8.35	0.340
8	6	0.75	2	2.25	0.157	123	247	321	0.116	7.325	7.9	8.45	0.144
9	6	0	0.5	2	0.783	0	63	282.5	0.917	7.15	7.3	7.825	0.595
10	6	0	2	3	0.336	0	50.5	425.25	0.686	7.05	7.5	8.075	0.917
P value★★		0.626				0.627				0.002			

Table 4: Lymphocyte and Eosinophil in goats with monensin intoxication

Day	N	Lymph (per/Microliter)				Eosinophil (%)				Eosinophil (per/Microliter)			
		25th	50th	75th	P ★ value	25th	50th	75th	P ★ value	25th	50th	75th	P ★ value
0	7	7320	7560	9301	-	0	1	4	-	0	126	304	-
1	7	7182	7625	8844	0.735	0	3	4	0.680	0	273	496	0.753
2	7	6720	9372	9856	0.237	0	1	1	0.713	0	120	132	0.686
3	7	7020	7930	9660	0.612	0	0	1	0.144	0	0	130	0.225
4	7	7150	7920	9996	0.398	0	1	3	0.680	0	110	360	0.600
5	6	7899.5	8608	9050	0.249	0	0.5	1.25	0.168	0	66	179.5	0.249
6	6	7312.3	8396.5	9184.5	0.345	0	0	1.25	0.131	0	0	163.75	0.225
7	6	7944.8	8565	9282.5	0.075	0	1.5	2.75	1	0	189.5	381.25	0.917
8	6	7939.5	9156	10154	0.075	0	2	3.5	1	0	281.5	540.25	0.463
9	6	8170.5	8868.5	9461.8	0.116	0	0	3.5	0.414	0	0	458.5	0.500
10	6	7527.5	8817.5	9508	0.249	0	0.5	1.75	0.197	0	70	249	0.463
P value★★		0.114				0.452				0.427			

Table 5: Fibrinogen in goats with monensin intoxication

Day	N	Fibrinogen (milligram/deciliter)			P value★
		25th	50th	75th	
0	7	300	300	400	-
1	7	300	400	400	0.157
2	7	300	400	400	0.180
3	7	400	400	400	0.059
4	7	400	400	400	0.157
5	6	375	400	400	0.317
6	6	375	400	400	0.157
7	6	375	400	400	0.157
8	6	300	400	425	0.157
9	6	375	400	400	0.157
10	6	300	400	400	0.655
P value★★		0.899			

Electrocardiography

At electrocardiography, sinus arrhythmias, sinus tachycardia, sinus bradycardia, ventricular tachycardia, ventricular premature complexes, S-T segment depression and increase in T wave amplitude were the most prominent findings. Sinus arrhythmias and sinus tachycardia were seen in 6 goats (Figure 1-1). Sinus bradycardia in one goat (Figure1-2), ventricular premature complexes in two goats (Figure 1-3), ventricular tachycardia in one goat (Figure 1-4) and S-T segment depression were observed in three goats (Figure 1-3).

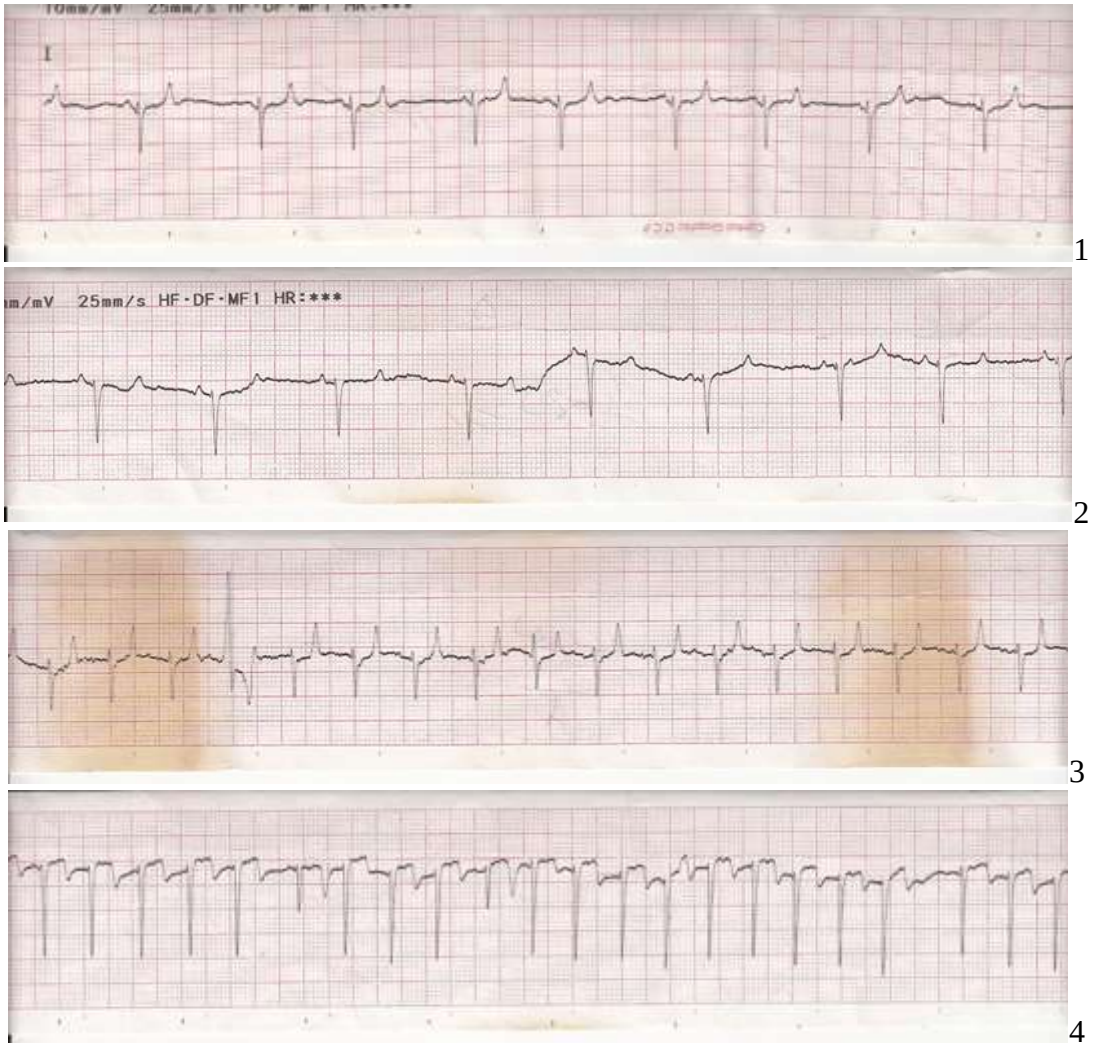

Figure 1: Electrocardiograms of goats intoxicated with monensin. 1. Sinus arrhythmia, 2. Sinus bradycardia, 3. Ventricular premature complex, 4. Ventricular tachycardia (non-sustained)

Discussion

During the past decades, it has been an increasing data of monensin beneficial and toxic effects in cattle and sheep (Bourqe et al, 1986; Duffield et al, 2012; Gonzalez et al, 2005; Mendes et al, 2003; Wang et al, 1990). However, while monensin has been approved for using in goats (Smith and Sherman, 2009), similar data for this animal are rather limited and to our knowledge, this study is the first report regarding the hematologic and electrocardiographic features in monensin exposed goats.

In this study, monensin exposure of goats did not result in noticeable changes in hematologic parameters. It is in accordance with Bouque et al, (1987) and Gonzalez et al, (2005) reports in which monensin intoxication in sheep had no effect on CBC. In this study, elevation of neutrophil percent at days 5 and 8 was significant. Elevation of blood neutrophils in these goats may be related to pain due to myopathy and associated stress Mild neutrophilia has been reported in cases of acute monensin intoxication in horses (Peek and et al, 2004). Janzen et al, (1981) also observed a mild neutrophilia in a group of monensin intoxicated bulls.

At tissue levels, necrotic myopathy and cardiomyopathy are the main features of monensin and other ionophers intoxications in different species of animals as well as in humans (Omidi et al, 2010; Mousas and El-Hamamsy, 2013; Zhang et al, 2018;

Pavarini et al, 2018; Pistán et al, 2020). Monensin, an ionopher antibiotic, has been approved to be used in domestic ruminants and widely mentioned as coccidiostat and growth promotant. However, because it has a narrow safety index in target and non-target species, intoxication occurs as a result of inappropriate use or accidental contaminations of ration (Hall, 2004). All ionophores including monensin facilitate transmembrane ion fluxes and dissipation of ion gradients, which are exaggerated at toxic levels. Cells respond to the metabolic insult by expending energy to maintain homeostasis. When homeostatic mechanisms are exceeded, toxicity ensues from excessive influxes of sodium and calcium ions leading to degeneration and necrosis of cardiac and skeletal muscle cells. Monensin also causes release of catecholamines from cultured adrenal chromaffin cells. Catecholamines and toxic oxidation products have been implicated in myocardial necrosis through greater influx of Ca^{++} and formation of free radicals (Novilla, 2012). Although cardiac arrhythmia has been mentioned in cases of monensin intoxication (Peek et al, 2004; Anjos et al, 2023), there are no details about the type of those disorders.

Monensin exposure of goats in the present study resulted in sinus tachycardia in 6 goats. This finding has also been reported in accidental monensin intoxication in goats (Anjos et al, 2023). Pain due to monensin intoxication induced sinus tachycardia. Sinus bradycardia or simple bradycardia is used to describe a decrease in heart rate due to a decreased rate of discharge from the sino-atrial node. In this study, sinus bradycardia was observed in one goat and it was accompanied by sinus arrhythmia which may be due to anorexia and hypoglycemia. Sinus arrhythmia is usually present in animals with sinus bradycardia (Constable et al, 2017).

Ventricular premature complexes may arise from an irritable process anywhere within the ventricular myocardium and are often seen in association with structural heart disease (Cha et al, 2012). In this situation, normal rhythm is interrupted by a beat that occurs earlier than expected but the initial rhythm is established following a compensatory pause. On the electrocardiogram, ventricular premature complexes are characterized by bizarre QRS morphology (Constable et al, 2017). In this study, two of monensin intoxicated goats showed ventricular premature complexes in the electrocardiograms. Premature complexes of all site origins are indicative of myocardial disease (Cha et al, 2012; Constable et al, 2017). Ventricular premature beats have also been reported in experimentally induced salinomycin, an ionophore antibiotic, toxicosis in sheep (Hesseini et al, 2013).

Ventricular tachycardia may produce either a regular heart rate or an irregular heart rate or rhythm. When the discharge rate of the irritant focus exceeds that of the sino-atrial pacemaker, the ectopic focus will take over completely as the pacemaker of the heart (Constable et al, 2017). Monensin exposure of goats resulted in ventricular tachycardia in one goat. Ventricular tachycardia is an evidence of severe cardiac disease and is usually accompanied by signs of acute heart failure (Reef and McGuirk, 2015). Myocarditis, nutritional cardiomyopathy and myocardial neoplasia could primarily cause ventricular tachycardia. Ventricular tachycardia is often a life-threating arrhythmias (Delesalle et al, 2002).

In conclusion, this study demonstrated that sub-acute monensin intoxication caused particular electrocardiographic features. However, further investigations are needed to clarify the relationship between those changes and doses of monensin in goats.

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Conflict of Interest

The authors declare that they have no conflict of interest.

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مسمومیت تحت حاد تجربی با مونسین در بزها: یافته‌های خون‌شناسی و الکتروکاردیوگرافی

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چکیده

اثرات سمی مونسین، یک آنتی‌بیوتیک پلی‌اتری که غالبا به عنوان کوکسیدیواستات به کار می‌رود، در طیف وسیعی از حیوانات توصیف شده است. مطالعه حاضر برای بررسی چهره خون‌شناسی و الکتروکاردیوگرافیک مسمومیت تحت حاد با مونسین در بزها انجام شد. برای این منظور، مونسین با دز ۱۳mg/kg روزانه به مدت ۵ روز متوالی به ۷ رأس بز بالغ با لوله معدی تجویز شد. فراسنجه‌های خون‌شناسی شامل هماتوکریت، هموگلوبین، گلبول‌های سفید و شمارش تفریقی آن‌ها، پروتئین تام پلاسما و غلظت فیبرینوژن آن در روز قبل از تجویز مونسین و ۱۰ روز متوالی اندازه‌گیری شدند. الکتروکاردیوگرافی نیز روزانه به روش استاندارد اخذ شد. نتایج افزایش معنی‌دار هموگلوبین در روز ۱، گلبول‌های سفید در روز ۷، درصد نوتروفیل‌ها در روزهای ۵ و ۸، در صد لمفوسیت‌ها در روزهای ۵، ۸ و ۹ و درصد منوسیت‌ها در روزهای ۱ و ۳ را نشان داد. همچنین تعداد مطلق منوسیت‌ها در روزهای ۱، ۳ و ۶ و پروتئین تام پلاسما در روز ۱ افزایش معنی‌دار را نشان دادند. یافت‌های غالب در الکتروکاردیوگرافی دام‌های مورد آزمایش تاکی کاردی سینوسی، برادی کاردی سینوسی، آریتمی سینوسی، پایین افتادن قطعه S-T، ضربان‌های زودرس بطنی و تاکی کاردی بطنی بودند. بر اساس مطالعه حاضر مسمومیت تحت حاد با مونسین در بزها تغییرات اندکی در فراسنجه‌های خونی ایجاد می‌کند ولی اختلات الکتروکاردیوگرافی چندی را باعث می‌شود که می‌تواند به کاردیومیوپاتی توکسیک مربوط باشد.

کلمات کلیدی: مونسین، آنتی‌بیوتیک‌های پونوفر، بز، الکتروکاردیوگراف، آریتمی

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