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Electrocardiographic changes associated with hyperkalaemia in domestic cats#

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Abstract

Hyperkalaemia is a life-threatening electrolyte imbalance because it affects cardiac conduction and can lead to fatal arrhythmias if left untreated. The present study describes the occurrence of hyperkalaemia in cats and the electrocardiographic changes associated with this electrolyte imbalance. Hyperkalaemia was identified in 83.33 per cent of the study group subjects. Acute kidney injury and obstructive uropathy were the main clinical conditions associated with it. Electrocardiographic findings in hyperkalaemia in different cats under study included peaked T waves in lead II and the precordial lead CV6LL, atrial standstill and sino-ventricular rhythm, normal sinus rhythm, ventricular tachycardia, first-degree atrio-ventricular block, bradycardia, sinus tachycardia, and atrio-ventricular dissociation. Electrocardiography should always be performed in cases suspected of electrolyte imbalances, particularly hyperkalaemia, so as to identify any fatal arrhythmias and initiate treatment at the earliest.

Keywords: *Cat, hyperkalaemia, electrocardiography, cardiac arrhythmias*

Electrolyte imbalances are frequently encountered in cats, particularly in those presented to the critical care unit. A multivariable analysis found that electrolyte imbalances were associated with a higher mortality rate in cats. Therefore, the quantification of electrolytes was crucial in the assessment of critically ill feline cases (Goggs *et al.*, 2018). In a retrospective study, urinary disorders were found to be the most common cause of hyperkalaemia in cats (Hoehne *et al.,* 2019).

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Hyperkalaemia affects cardiac conduction which results in fatal arrhythmias. Thus, hyperkalaemia needs to be rapidly diagnosed and corrected. Electrocardiography was a quick, non-invasive and the initial test of choice for the diagnosis of cardiac arrhythmias, and should always be performed in cats suspected for potassium disorders.

Materials and methods

Twelve domestic cats, irrespective of age, sex and breed presented to TVCC, CVAS, Pookode, Wayanad, showing clinical signs suggestive of electrolyte disturbances such as anorexia, vomiting, poor body condition, muscular weakness, obtundation, oliguria, polyuria, stranguria, haematuria or ataxia were selected for the study. Six apparently healthy cats brought for routine health check-ups were taken as the control group for comparison of the parameters under study. The haematological evaluation was done, and biochemical parameters like serum creatinine, potassium and calcium were estimated using a semi-automatic biochemical analyzer (Agappe MISPAVIVA 2578-10/17).

Electrocardiography (ECG) was performed by placing the cat in right lateral recumbency on a rubber-coated foam mat, using minimally traumatic ECG clips (Fig. 1). The recordings were taken using standard bipolar limb leads, augmented unipolar limb leads and precordial chest leads at paper speeds, 25 mm/sec and 50 mm/sec, and voltage 10 mm/mV (Cote *et al.,* 2011).

Animals in the study group were treated for the primary cause of electrolyte disturbances. Specific treatments for the management of hyperkalaemia included five per cent sodium bicarbonate @ 1-2 mEq per kg body weight intravenously, 25 per cent dextrose @ 1 g per kg body weight intravenously, or insulin @ 0.5 IU per kg body weight subcutaneously, along with intravenous dextrose @ 2 g/IU of insulin (Riordan and Schaer, 2015). The treatment was decided according to the clinical condition.

Statistical analysis was done using SPSS software version 24.0, by applying independent t-test (Snedecor and Cochran, 1994).

Results and discussion

The results of the comparison of haematological findings between control and study groups are given in Table 1. The granulocyte count of the study group was significantly increased ($p<0.01$) as compared to the control group. This might be due to the inflammatory reaction of the urinary tract in the present study. The platelet count of the study group animals was significantly lower ($p<0.01$) as compared to the control group animals. This could be attributed to the consumptive loss of platelets due to haematuria in cats with obstructive uropathy (Ellis *et al.*, 2018).

Normal sinus rhythm was observed on the electrocardiogram of the control group animals, which was depicted in Fig. 3A, Fig. 3B and Fig. 3C.

Hyperkalaemia was identified in 10 out of 12 cats (83.33 per cent) of the study group. The mean ± SE values of serum creatinine, potassium and calcium of control and study group animals are given in Table 2. There was a significant increase ($p<0.01$) in the serum creatinine and potassium levels, and a significant decrease ($p<0.05$) in the serum calcium level observed in the study group, as compared to the control group (Table 2). Acute kidney injury and obstructive uropathy were identified as the main clinical conditions associated with hyperkalaemia, accounting for 40 per cent (4 out of 10 cats) and 60 per cent (6 out of 10 cats) of the cases, respectively.

Electrocardiographic changes associated with hyperkalaemia are presented in Table 4 and Fig. 2. Peaked T wave in the precordial lead CV6LL was found to be the most common ECG finding, in 50 per cent of the cases. In one cat (animal number S1), the peaked T wave was observed only in the precordial lead CV6LL and not in lead II, taken simultaneously, which highlighted the importance of recording precordial chest leads in these cases (Fig. 4). Other ECG findings related to hyperkalaemia were peaked T waves in lead II, atrial standstill and sino-ventricular rhythm, in 40 per cent of

Table 1. Comparison of haematological parameters between control group and study group

Parameters	Control group (n=6)	Study group (n=12)	t-value	P-value
TEC (10^6 /µL)	8.61 ± 0.41	9.2 ± 0.77	0.513^{ns}	0.615
Hb (g/dL)	11.82 ± 0.45	13.28 ± 1.05	0.949^{ns}	0.357
VPRC (%)	35.45 ± 1.44	39.85 ± 2.87	1.040^{ns}	0.314
MCV (fL)	41.52 ± 2.03	44.13 ± 0.95	1.345^{ns}	0.197
MCH (pg)	13.83 ± 0.94	14.53 ± 0.33	0.872^{ns}	0.396
MCHC (%)	33.58 ± 0.79	33.04 ± 0.49	0.612^{ns}	0.549
TLC (10^3/µL)	14.87 ± 2.08	19.55 ± 3.46	0.907^{ns}	0.378
Lymphocyte count (%)	39.85 ± 3.87	15.01 ± 3.92	1.36^{ns}	0.193
Monocyte count (%)	4.48 ± 0.35	3.51 ± 0.47	1.192^{ns}	0.251
Granulocyte count (%)	55.67 ± 4.11	81.5 ± 4.08	3.994**	0.001
Platelet count (10^3/µL)	260 ± 25.76	150.75 ± 22.95	3.886**	0.001

*** Significant at 0.01 level; ns non-significant*

Table 2. Comparison of serum biochemical parameters between control group and study group

Parameters	Control group (n=6)	Study group (n=12)	t-value	P-value
Creatinine (mg/dL)	1.18 ± 0.13	9.49 ± 1.49	5.565**	<0.001
Potassium (mEq/L)	4.6 ± 0.21	8.43 ± 0.89	4.185**	0.001
Calcium (mg/dL)	9.75 ± 0.33	6.93 ± 0.65	3.87**	0.001

*** Significant at 0.01 level*

Table 3. Comparison of values of ECG of lead II between control and study groups in case of hyperkalaemia

Parameters	Control group (n=6)	Study group (n=10)	t-value	P-value
P wave amplitude (mV)	0.108 ± 0.008	0.04 ± 0.021	3.048*	0.011
P wave duration (sec)	0.033 ± 0.003	0.011 ± 0.006	3.397**	0.005
PR interval (sec)	0.058 ± 0.003	0.018 ± 0.009	4.124**	0.002
R wave amplitude (mV)	0.583 ± 0.079	0.505 ± 0.168	0.344^{ns}	0.736
QRS duration (sec)	0.037 ± 0.002	0.055 ± 0.01	1.718^{ns}	0.117
T wave amplitude (mV)	0.158 ± 0.02	0.295 ± 0.099	1.357^{ns}	0.206
QT Interval (sec)	0.165 ± 0.006	0.227 ± 0.014	3.942**	0.002
Corrected QT interval (sec)	0.28 ± 0.009	0.347 ± 0.026	2.409*	0.034
Heart rate (beats per minute)	180.17 ± 8.67	156.3 ± 13.02	1.307^{ns}	0.212

*** Significant at 0.01 level; * Significant at 0.05 level; ns non-significant*

the cases. Normal sinus rhythm, ventricular tachycardia, and first-degree atrio-ventricular (AV) block were observed in 20 per cent of the cases. Sinus tachycardia, bradycardia and atrio-ventricular dissociation were the least common findings, observed only in 10 per cent of the cases.

The electrocardiogram of five animals (S1, S2, S4, S7 and S10) showed peaked T waves in lead II and precordial lead CV6LL. Peaked T waves were observed due to prolonged ventricular repolarization as a result of increased cell permeability to potassium (Cote *et al.,* 2011).

Atrial standstill and sino-ventricular rhythm were observed in four animals (S1, S2, S3 and S5). These findings were reported in cats with hyperkalaemia (Norman *et al.,* 2006; Hall *et al.,* 2010; Spalla *et al.,* 2014). The electrocardiogram of the animal S2, is depicted in Fig. 5. The atrial myocytes are more sensitive to hyperkalaemia, as compared to the specialized pacemaker cells of the sino-atrial node (SA node) and atrio-ventricular node (AV node). The amplitude of the P wave on the ECG progressively decreases and ultimately, there was a complete loss of the P wave which was known as an atrial standstill. The resulting rhythm was termed as a "sino-

Table 4. ECG changes in cats with hyperkalaemia

Animal numbers (within study group)	Potassium imbalance	ECG findings
S1	Hyperkalaemia (concurrent hypocalcaemia)	Atrial standstill, peaked T wave in precordial leads, QS pattern of QRS, right axis deviation
S2	Hyperkalaemia (concurrent hypocalcaemia)	Atrial standstill, peaked T wave in lead II and precordial leads, rS pattern with deep S wave, wide QRS complex, prolonged QT interval, right axis deviation
S3	Hyperkalaemia (concurrent hypocalcaemia)	Atrial standstill at presentation; first degree AV block, and prolonged QT interval after treatment with insulin and dextrose
S4	Hyperkalaemia (concurrent hypocalcaemia)	Atrio-ventricular dissociation at presentation; first degree AV block, peaked T waves and prolonged QT interval after intravenous calcium gluconate
S5	Hyperkalaemia (concurrent hypocalcaemia)	Atrial standstill, bradycardia
S6	Hyperkalaemia	Sinus tachycardia
S7	Hyperkalaemia (concurrent hypocalcaemia)	Ventricular tachycardia
S8	Hyperkalaemia (concurrent hypocalcaemia)	Normal sinus rhythm, prolonged QT interval
S9	Hyperkalaemia	Normal sinus rhythm
S10	Hyperkalaemia (concurrent hypocalcaemia)	Ventricular tachycardia

ventricular rhythm" as it originates in the SA node and is conducted to the AV node through the inter-nodal pathways, and subsequently to the ventricles, without causing atrial activation (Cote *et al.,* 2011).

Animal number S4 showed reduced amplitude of R wave, shortened PR interval and atrio-ventricular dissociation. Shortening of the PR and QT intervals were observed in the early stages of hyperkalaemia (Parham *et al.,* 2006).

Animal number S5 had bradycardia with a heart rate of 93 beats per minute (Fig. 6). Hyperkalaemia decreases the slope of diastolic depolarization (phase 4), decreases the normal pacemaker tissue activity, and subsequently slows the heart rate (Cote *et al.,* 2011).

Animal number S6 had sinus tachycardia, with a heart rate of 250 beats per minute (Fig. 7). Tachycardia might be attributed to increased sympathetic activity and catecholamine release in stress or pain (Cote *et al.,* 2011; Ashi *et al.,* 2022).

Animal numbers S7 and S10 showed ventricular tachycardia on the ECG, characterized by wide and bizarre QRS complexes (Fig. 8). Ventricular tachycardia in hyperkalaemia was previously reported by Tag and Day (2008) and Canei *et al.* (2021). As the serum potassium concentration increased, there was a depression of the sino-atrial node, which results in a junctional or ventricular rhythm. This could be considered as pre-agonal and followed by ventricular fibrillation and asystole (Spalla *et al.,* 2014).

Two animals (S8 and S9) showed normal sinus rhythm, which was consistent with the findings of Tag and Day (2008).

Animal, S3, had an initial serum potassium concentration of 9.95 mEq/L and atrial standstill and sino-ventricular rhythm in ECG at presentation. Treatment with insulin and dextrose resulted in a lowering of the serum potassium concentration to 6.25 mEq/L. The ECG revealed sinus rhythm with

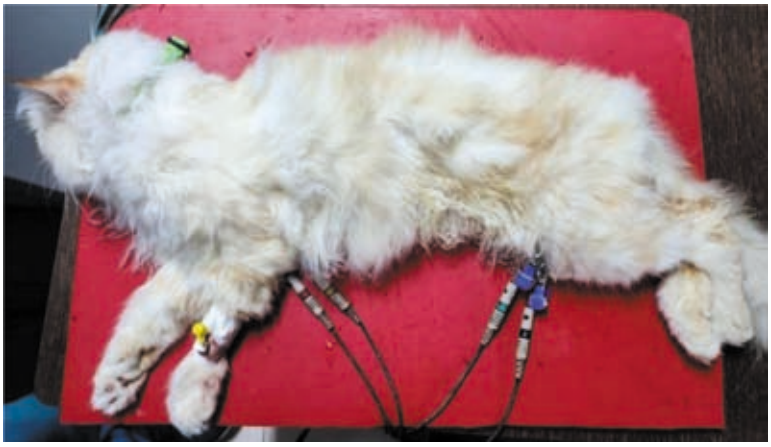

Fig. 1. Positioning of cat for electrocardiography

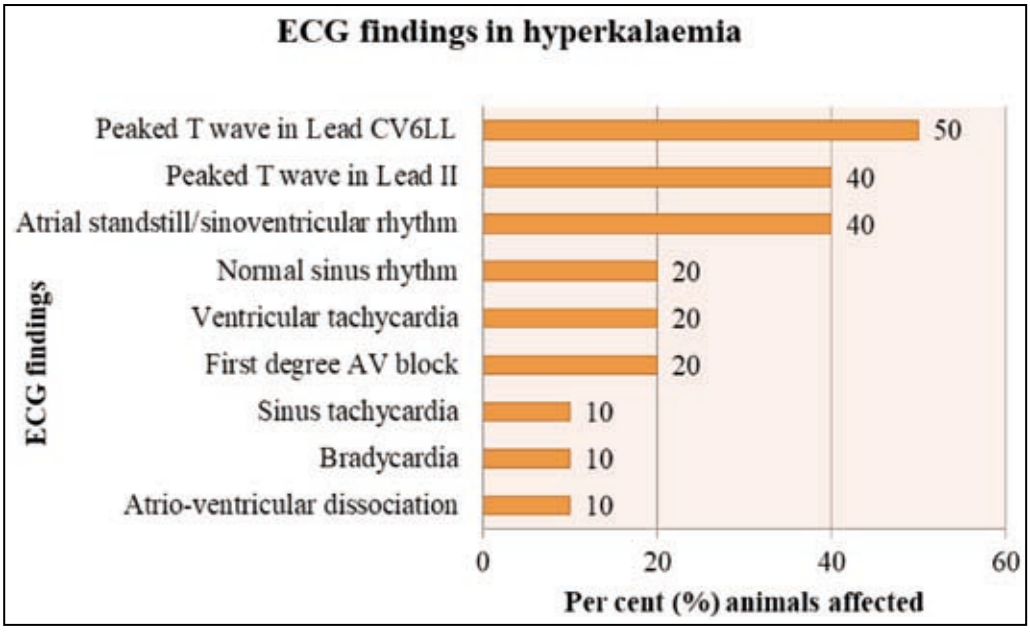

Fig. 2. Electrocardiographic findings associated with hyperkalaemia in cats

first-degree AV block (Fig. 9) after initiation of therapy. First-degree AV block, a less serious disturbance, had been previously reported with hyperkalaemia, which might be attributed to the reduced conduction velocity through the AV node (Spalla *et al.,* 2014).

The ECG values obtained from the analysis of lead II values of control and study groups were given in Table 3.

Significant changes were recorded in the P amplitude, P duration, PR interval, QT interval and corrected QT interval in lead II of cats with hyperkalaemia. Among the ECG values of lead II, there was a significant decrease ($p<0.05$) in the P amplitude of the study group as compared to the control group. The P duration and PR interval of the study group were significantly lower ($p<0.01$) than the control group.

The QT interval of the study group was significantly increased ($p<0.01$) as compared to the control group. There was a significant increase ($p<0.05$) in the corrected

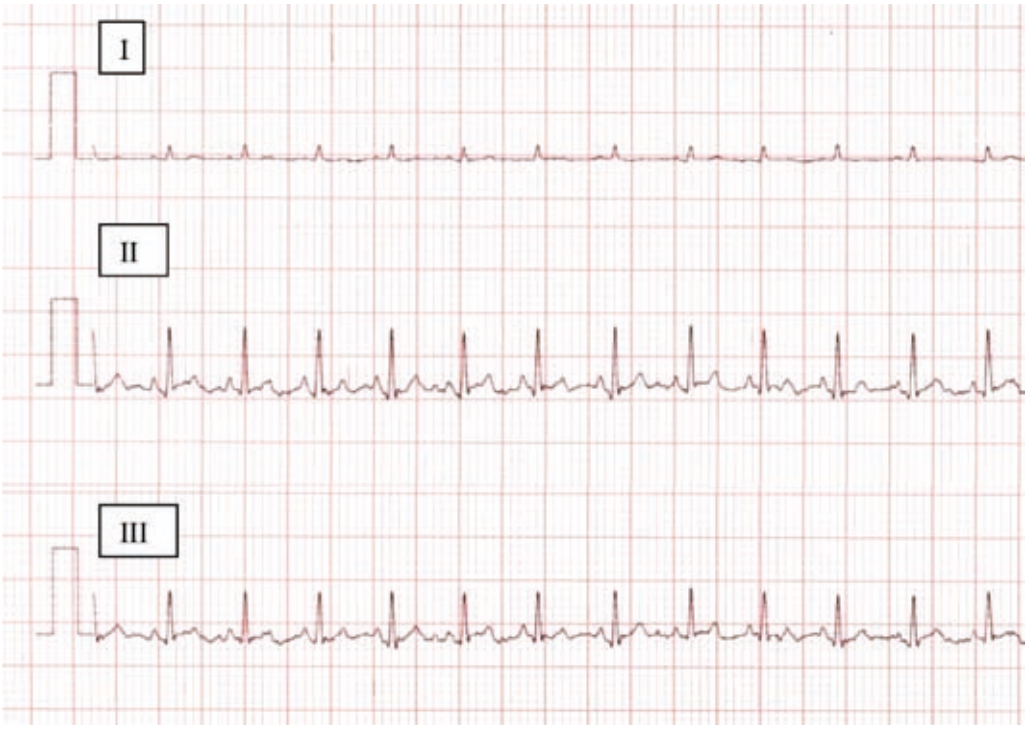

Fig. 3A. Normal sinus rhythm (bipolar limb leads) (Paper speed – 25 mm/sec)

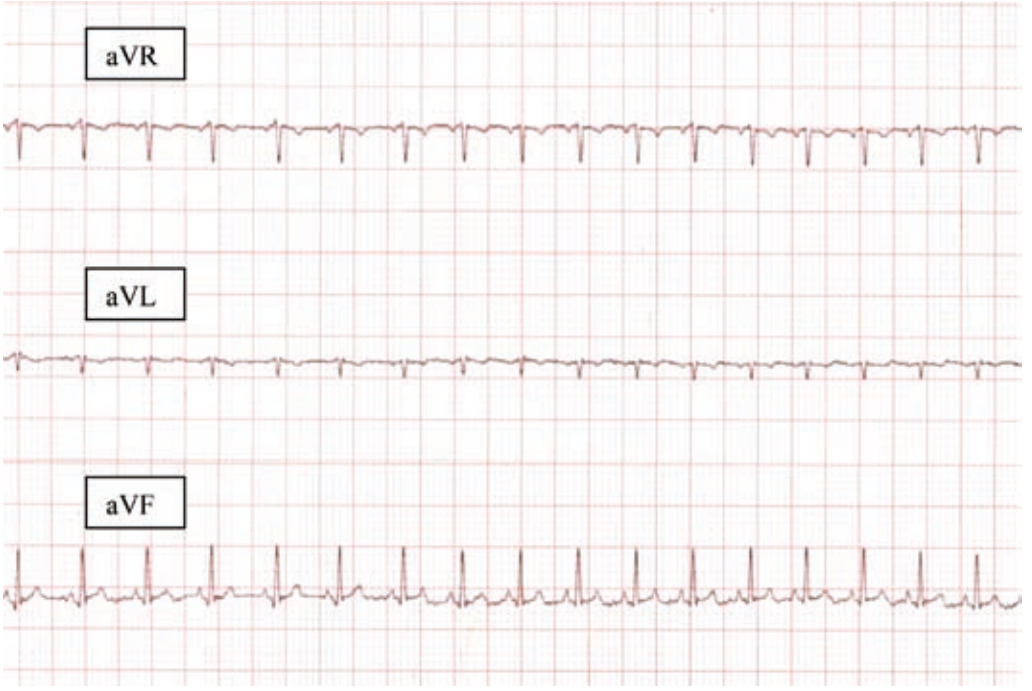

Fig. 3B. Normal sinus rhythm (augmented unipolar limb leads) (Paper speed – 25 mm/sec)

QT interval of the study group as compared to the control group. Prolongation of QT interval might be attributed to the widening of QRS complexes or due to hypocalcaemia, which occurred concurrently in a majority of these cases. Hypocalcaemia caused prolongation

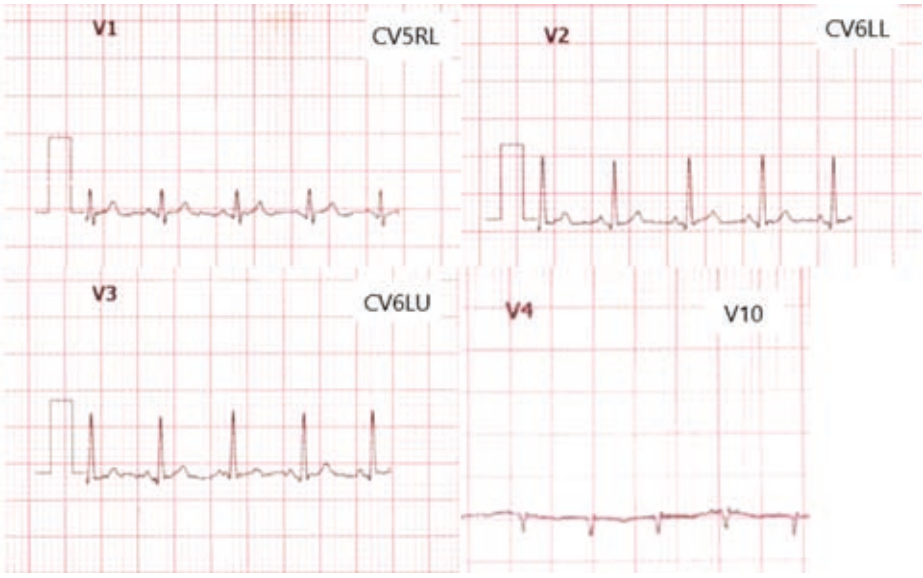

Fig. 3C. Normal sinus rhythm (precordial chest leads) (Paper speed – 25 mm/sec)

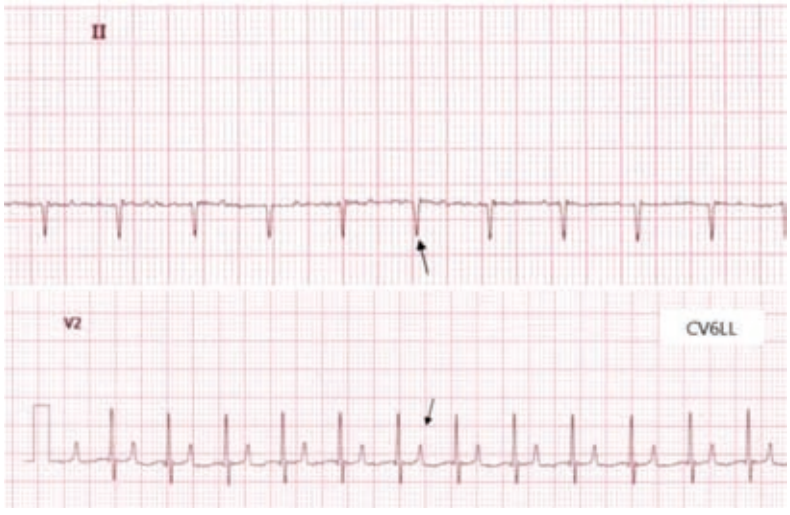

Fig. 4. ECG of animal number S1, showing QS pattern in lead II (arrow), Peaked T wave in precordial lead CV6LL (arrow) (Paper speed – 25 mm/sec)

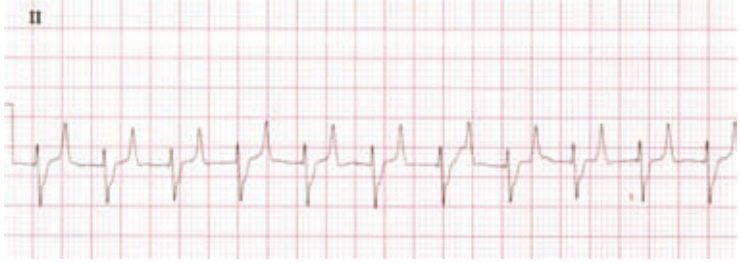

Fig. 5. ECG of animal number S2, showing atrial standstill and sino-ventricular rhythm, rS pattern of QRS complex with deep S waves, peaked T waves, wide QRS complexes and prolonged QT interval (Paper speed – 25 mm/sec)

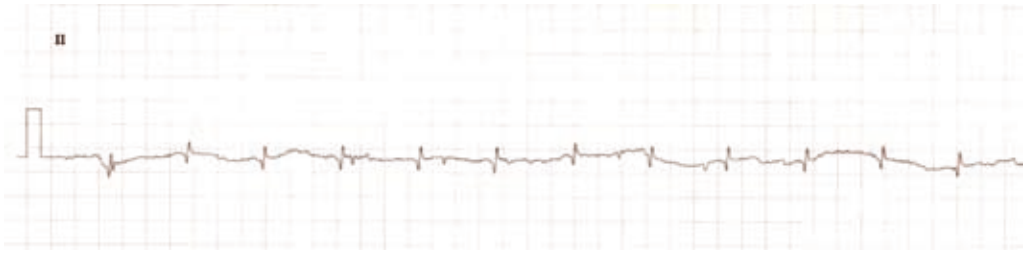

Fig. 6. ECG of animal number S5 depicting atrial standstill and sino-ventricular rhythm along with bradycardia (heart rate = 93 beats per minute) (Paper speed – 25 mm/sec)

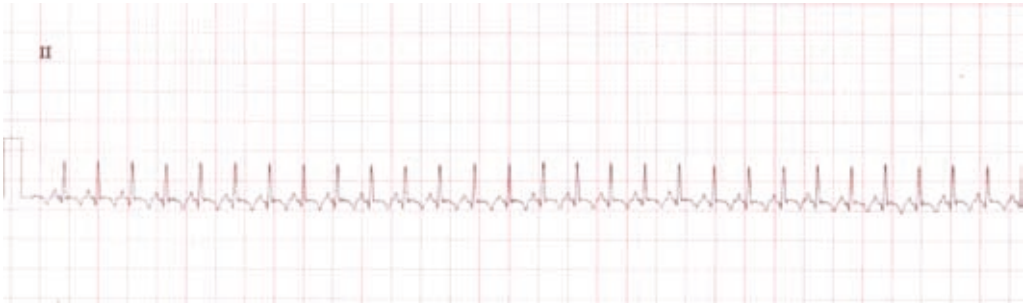

Fig. 7. ECG of animal number S6 depicting sinus tachycardia (Heart rate = 250 beats per minute) (Paper speed – 25 mm/sec)

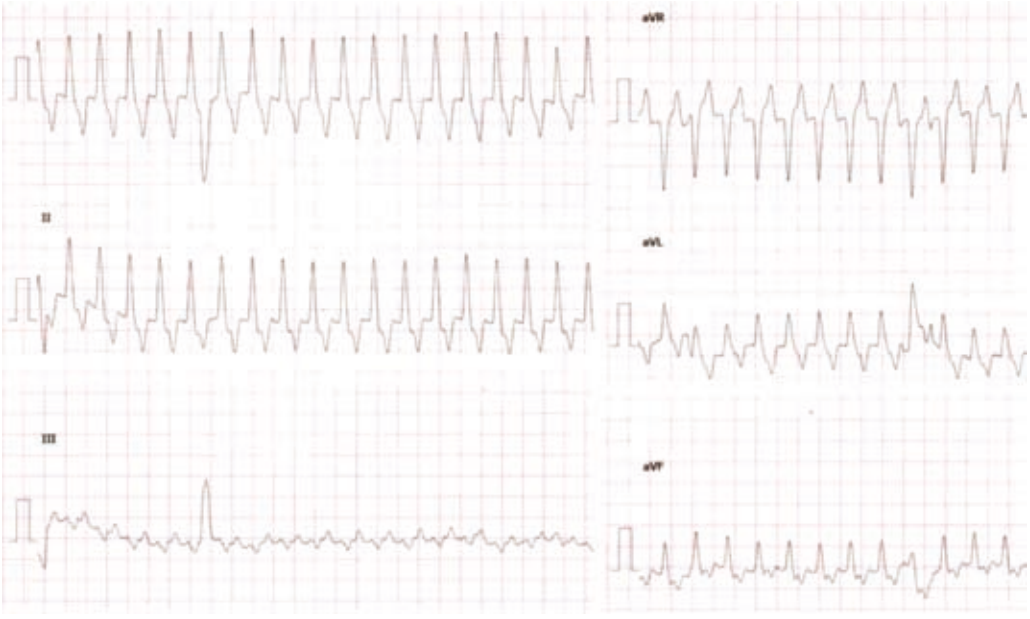

Fig. 8. ECG of animal number S7 showing ventricular tachycardia (Heart rate = 214 beats per minute) (Paper speed – 25 mm/sec)

of the QT interval by prolongation of phase 2 of the cardiac action potential (Dusky, 2001). In the current study, cats with concurrent hyperkalaemia and hypocalcaemia had more severe electrocardiographic abnormalities because the cardiac effects of hyperkalaemia might be exacerbated by hypocalcaemia (Simon *et al.,* 2022).

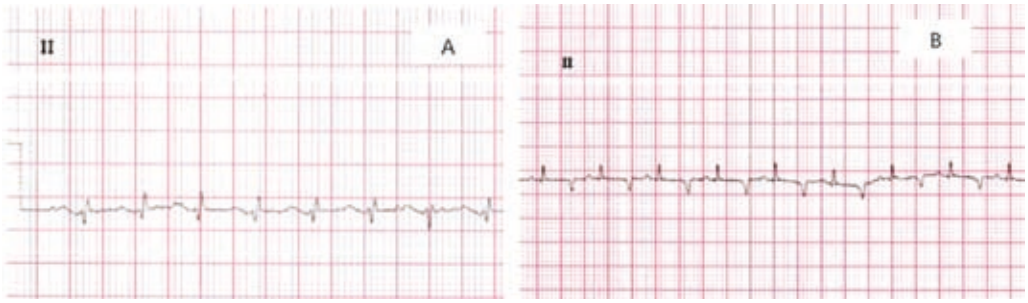

Fig. 9. ECG of animal number S3 before and after treatment (Paper speed – 25 mm/sec). A – Atrial standstill and sino-ventricular rhythm before treatment. B – Sinus rhythm with first-degree AV block and prolonged QT interval (lengthening of ST segment) after treatment

Conclusion

Electrocardiography is helpful in the early diagnosis of electrolyte imbalances, particularly hyperkalaemia so that appropriate treatments shall be instituted quickly even before getting the serum biochemical results. Intravenous sodium bicarbonate or intravenous dextrose, alone or in combination with insulin, was effective in the treatment of hyperkalaemia.

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Conflict of interest

The authors declare no conflict of interest.

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