



ORIGINAL ARTICLE

Changes in Electrocardiographic Parameters in Seropositive horses with equine influenza

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ABSTRACT

This study was performed to examine the impact of influenza in horses on the electrocardiogram changes and cardiac arrhythmias. Samples were taken from 202 cases (1-3 years) from Tabriz area in Iran. After centrifugation, the serum was removed and then seroprevalence was investigated by ELISA method. All the horses were taken electrocardiogram using two-channel digital electrocardiography in base apex lead. From 202 horses, 19 samples (9.5%) were positive and 183 samples (90.5%) were negative. The electrocardiograms of all the horses were taken and the kind of cardiac arrhythmias were recorded. Average heart rate increased significantly in patient group ($p < 0.05$). 164 of 183 horses had heart's natural rhythm and 19 of them (10.38%) had arrhythmias in normal group but 12 of 19 horses had heart's natural rhythm in patient group and 7 of them (36.84%) had arrhythmias. Occurrence of cardiac arrhythmias was significantly greater in patient group ($p < 0.05$). The average of recorded duration and wave intervals in horses in two groups did not show significant changes. The average of T and P waves amplitudes had no significant change in two groups, but the QRS wave amplitude showed a significant increase in patient group ($p = 0.039$). The end result was that following equine influenza some electrocardiographic changes were created in horse and most of which are physiologic changes.

Key words: equine, influenza, electrocardiography

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INTRODUCTION

Equine influenza disease is attended by inflammation in upper respiratory tract. Most of epidemics in young horses is appeared less than 2 years of age, especially 2-6 month of age [1]. Horses that were maintained in unsuitable environment conditions cross disease period without any problem, but for horses which work or are used in transportation or exposure to unsuitable climate conditions, cough is sever and may led to disease such as Bronchitis, Pneumonia and Bound feet, but fortunately death rate is 1-3% [2, 3, 4]. The cause of this disease is type A virus, it is in two type A1 and A2. Disease transmission in horse is by air breathing that contaminated droplets of horse, breathing is propagated in air leading to disease transmission. For this reason, this virus is able to contaminate horses in a short time. Also transmission is possible by contaminated subjects. Generally horse races, horse fairs and places that horses, surgeries is performed, all of them are suitable places for transmission of this disease [5, 6, 7, 8]. This study was performed to examine the impact of influenza in horses on the electrocardiogram changes and cardiac arrhythmias.

MATERIALS AND METHODS

Samples were taken from 202 cases (1-3 years) from Tabriz area in Iran, during May to November of 2012. These cases were 410 males and 80 females. Ten millilitres of blood were collected from the jugular vein of each animal. The blood samples were allowed to clot and were centrifuged for 10 min at 3000g. After centrifugation, the serum was removed and stored at -20°C until ready for test. Serum samples were transmitted to Laboratory and then seroprevalence was investigated by ELISA method (IDVET kit). All the horses were taken electrocardiogram using two-channel digital electrocardiography in base apex lead. The

electrocardiograms of all the horses were taken and the kind of cardiac arrhythmias were recorded. SPSS13 statistical analysis software was used for analyzing obtained results and its results were expressed descriptively about electrocardiographic changes then they compared using chi-square method. In order to compare the mean of quantitative numbers, T-test statistical method was used.

RESULTS

From 202 horses, 19 samples (9.5%) were positive and 183 samples (90.5%) were negative. Seropositive horses tacked in patient group and non seropositive horses tacked in normal group. The number of heart rate per minute in normal group was 26 to 86 beats per minute; so, average heart rate was 42.41 ± 11.20 bpm. But in patient group, heart rate was 32 to 98 and the average heart rate was 54.22 ± 14.38 bpm. The difference between groups was significant ($p < 0.05$).

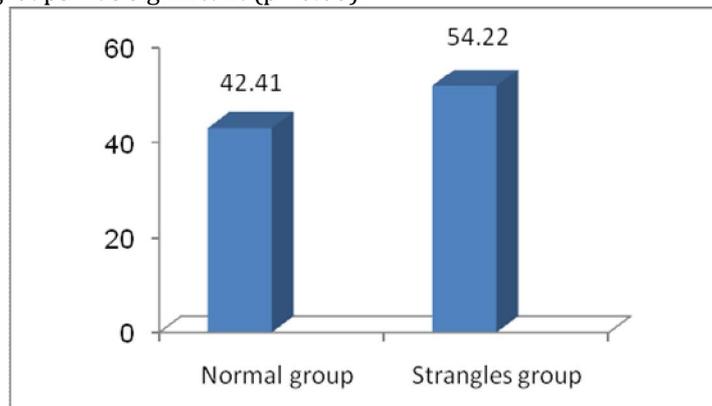


Diagram 1: Mean of heart rate in normal and patient groups

Assessing the electrocardiographic changes in normal group cleared that 164 of 183 under studied horses had a normal heart rhythm and physiological cardiac arrhythmias were observed in 19 other horses (10.38%); in 12 cases sinus tachycardia, in one case atrioventricular block I and 2 cases atrioventricular block II and in 4 cases sinus arrhythmia were recorded. But in patient group, 12 of 19 horses had natural cardiac rhythm and 7 horses (36.84%) had arrhythmias that all of which were physiologic cases, such that in 4 cases sinus tachycardia, in 1 case sinus arrhythmias and 2 cases atrioventricular block II were recorded. The occurrence of cardiac arrhythmias in patient group was significantly greater than the normal group ($p < 0.05$).

Table 1: Frequency of different heart rhythm in horses at normal and patient groups

group	No.	Heart rhythm	No.	%	P-Value
Normal	183	Normal rhythm	164	89.62	0.003
		Sinus tachycardia	12	6.56	
		Atrioventricular block I	1	0.55	
		Atrioventricular block II	2	1.09	
		Sinus arrhythmia	4	2.18	
Patient	19	Normal rhythm	12	63.16	
		Sinus tachycardia	4	21.05	
		Atrioventricular block I	-	-	
		Atrioventricular block II	2	10.53	
		Sinus arrhythmia	1	5.26	

The mean and standard deviation for P waves and QRS duration and PR and QT interval per seconds between both groups are given in Table 2. Comparison of the numbers in the table indicates that the time intervals and waves duration are in the normal range and there is no significant difference between the two averages.

Table 2: Mean of duration and wave intervals (s) in normal and patient groups

Wave and Wave intervals	Group	Mean	Standard deviation	P-Value	
P-wave	normal	0.11	0.03	0.965	
	patient	0.11	0.01		
QRS-wave	normal	0.11	0.01		0.406
	patient	0.13	0.06		
P-R intervals	normal	0.31	0.05	0.870	
	patient	0.30	0.02		
S-T segment	normal	0.41	0.08	0.623	
	patient	0.39	0.12		

The mean and standard deviation of the amplitude of waves P, QRS and T in terms of MV in both groups are given in Table 3. P and QRS average amplitude in patient group was greater than normal group that in the P-wave the mean difference between two groups was nonsignificant, but in the QRS wave it was significant ($p=0.021$). T- Wave amplitude in normal group was less than another group, non-significantly ($p=0.811$).

Table 3: Mean and standard deviation of wave amplitude (mv) in normal and patient groups

Wave	Group	Mean	Standard deviation	P-Value
P	normal	0.30	0.04	0.834
	patient	0.32	0.10	
QRS	normal	1.87	0.09	0.021
	patient	2.14	0.43	
T	normal	1.02	0.06	0.811
	patient	1.05	0.01	

Wave forms recorded in normal group are shown in Table 4. P-wave was recorded two-stage and single stage in 89 and 94 cases, respectively. QRS wave was recorded as qrs, QRS, qRS and QrS in 39, 86, 33 and 25 cases, respectively. T- Wave was recorded as negative, negative-positive and positive in 123, 35, and 25 cases, respectively.

P-wave, in patient group, in 10 of 19 under studied was recorded as two-stage and in 9 horses it was recorded as single-stage. QRS was recorded as qrs, QRS, qRS and QrS in 8, 6, 2 and 3 cases, respectively. T wave was recorded as negative, negative-positive, -negative and positive in 7, 3, 5 and 4 cases, respectively (Table 5).

Table 4: Recorded wave forms in normal group

Wave	Wave form	No.	%
P	Positive two-phase	89	48.63
	Positive single-phase	94	51.34
QRS	qrs	39	21.31
	QRS	86	46.99
	qRS	33	18.04
	QrS	25	13.67
T	-	123	67.21
	-,+	35	19.13
	+,-	-	-
	+	25	13.66

Table 5: Recorded wave forms in strangles group

Wave	Wave form	No.	%
P	Positive two-phase	10	52.63
	Positive single-phase	9	47.37
QRS	qrs	8	42.11
	QRS	6	31.58
	qRS	2	10.52
	QrS	3	15.79
T	-	7	36.84
	-,+	3	15.79
	+,-	5	26.32
	+	4	21.05

DISCUSSION AND CONCLUSION

In this study, the mean heart rate increased significantly in patient group ($p < 0.05$). The fever and pain of strangles is a sympathetic stimulation increases the heart rate. Pain, due to sympathetic stimulation, increases exhaust of atrial sinus node impulse and thus raise the heart rate [9]. 164 of 183 normal group horses (89.62%) had normal heart rhythm, but in another group, 12 of 30 under studied cases (63.16%) had normal heart rhythm that a significant difference was observed between them ($p=0.003$). Pain, due to Performance increase and cardiac contractility power, makes some changes in electrocardiogram and

heart rhythm that are considered; such a result was obtained in the present study, as well as decrease in the number of normal heart rhythm. Sinus tachycardia incidence was increased due to diseases. Sinus tachycardia represents an increase in heart rate in response to factors associated with pain, agitation, anxiety and other stressors. Pathological tachycardia may have originated as a secondary problem because of general diseases or as a primary form due to specific disorders of the heart itself. Fever, hypoxia, bleeding and anemia are causative factors of pathological sinus tachycardia, and some cases such as congestive heart failure, myocardial Infarcts are causative factors of primary pathological sinus tachycardia. It has been documented that in single-hoof animals and cattle poisoning due to eating high levels of Nerium Oleander, sympathetic stimulation causes sinus tachycardia [10]. Sinus arrhythmia is a kind of Physiological arrhythmias in which the exhausting impulses from atrial sinus node become slow and fast that in some animals, including horses, this type of arrhythmia is correlated to animal respiration (inspiration and expiration), and change in their vagus nerve tone [11]. Of course, some electrolyte changes, exercise, excitement and stress are also involved in its occurrence. Especially because of the anatomical location of SA node in horses and its large size, its affectability of the vagus is greater. In normal and patient groups were atrioventricular block I and II. Atrioventricular block I and II are considered as physiological arrhythmias in the horse, which could make some problems in animal exercise that in this case they should be considered as pathological arrhythmias. Since, there wasn't any problem in animal exercise ability, so it can be said that all recorded cases are considered as physiological atrioventricular blocks and their occurrence can be correlated with electrolyte changes. Nowadays, the continuous block of grade II in the horse that is resistant in repeated ECGs, are considered abnormal, and must be considered. However what is certain is that the atrioventricular conduction disturbances due to the imbalance of electrolytes, excesses in the administration of calcium salts, poisoning by Digitalines and low levels of Nerium Oleander, cardiomyopathy and myocarditis coupled with nutritional and infective diseases are some causes of II grade A-V block. Two of the arrhythmia cases have been observed in sheep infected by Sarcosporidiosis [12]. In a recent study, ECG strip was obtained from 50 horses that have had exploratory celiotomy surgery: 8 horses (23%) had premature ventricular beats, 4 horses (11%) had sudden ventricular tachycardia, and 11 horses (31%) showed supraventricular premature beats [13]. Researchers of the mentioned study believe that the most important causes of secondary arrhythmias are the effect of endotoxin on myocardium, autonomic neuropathy, acid-base disturbances, and electrolyte imbalance [12].

The mean rate of duration and intervals of waves recorded for under studied horses in two groups did not show significant difference. Non-significant reduction of the wave intervals can be correlated to increase heart rate following sympathetic stimulated disease. The mean of P-waves' amplitude between two groups did not change significantly and was no significant difference in the T wave, but QRS wave amplitude increased significantly in patient group (from 1.87 mV to 2.14 mV) ($p=0.021$). Changes in QRS wave amplitude can be attributed to increase ventricular contractility following disease. Fregin has been reported QRS wave height in a study on Thoroughbred horses as 1.86 mV [14].

Also, in the present study the wave forms were compared between normal and patient groups. In most cases, P-wave of the large animals such as horses is two-stage which is not considered arrhythmia but in a small animal, we should deal with this kind of deformation of the P wave as premature atrial contraction or atrial tachycardia, and in advanced cases it must be considered as atrial fibrillation [10]. QRS complex in control group was as qrs, QRS, QrS and qRs in 39, 86, 33 and 25s cases, respectively. But, in the patient group it was as qrs, QRS, QrS, and qRs in 8, 6, 2, and 3 case, respectively. Fregin didn't measure the QRSs frequency in his study [14]. Positive or negative T-wave is not considered Arrhythmia and can be recorded in any form in a horse. The end result is that Following equine influenza, some electrocardiographic changes are created most of which are physiological changes.

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REFERENCES

1. Burrows R, Goodridge D, Denyer M, (1982) Equine influenza infections in Great Britain. *Vet Rec* 1982; 110:494-497.
2. Gerber H, (1970) Equine influenza: Clinical features, sequelae and epidemiology of equine influenza. In: *Proceedings of the 2nd Int Conf Eq Inf Dis*. 63-80.
3. Glass K, Wood JLN, Mumford JA, (2002) Modelling Equine Influenza Infection. *Epidemiol Infect*. 128:491-502.
4. Goto H, Shimizu k, Abe T, Kanamitsu M, (1976) Sero- epidemiological study on equine influenza in Japan. *J. Clin. Microbiol.* 2: 89-93.

5. Guo Y, Wang M, Zhang G-S, (1995) Seroepidemiological and molecular evidence for the presence of two H3N8 equine influenza viruses in China in 1993-94. *J Gen Virol.* 76:2009-2014.
6. Gupta AK, Yadav MP, Uppal PK, (1993) Characterisation of equine influenza isolates from the 1987 epizootic in India by nucleotide sequencing from the HA1 gene. *Eq Vet J.* 25:99-102.
7. Ilobi CP, Nicolson C, Taylor J, (1998) Direct sequencing of the HA gene of clinical equine H3N8 influenza virus and comparison with laboratory derived viruses. *Arch Virol.* 143:891-901.
8. Livesay GJ, O'Neill TO, Hannant D, (1993) The outbreak of equine influenza (H3N8) in the United Kingdom in 1989: diagnostic use of an antigen capture ELISA. *Vet Rec.* 133:515-519.
9. Rezakhani A, Yazdanmehr MM, (1977) The normal electrocardiographic parameters of the domestic donkey (*Equus asinus*). *Zentralblatt Veterinarmedizin A* 24, pp. 672-679.
10. Robertson, SA, . (1990) Practical use of ECG in the horse. In *Practice*, March issue: 59-67.
11. Radostitis OM, Gay CC, Hinchcliff KW, Constable PD, (2007) *Veterinary Medicine, a text book of the disease of cattle, sheep, pigs and horses.* 10th edition, Elsevier publishing, pp: 1526-1532.
12. Robert R, Sonnehblick EH, Hurst s, (2007) *The heart Arteries and Veins.* 9th Ed McGraw-Hill Comp pp: 873-941.
13. Stephen M, Reed B, Warwick M, Bayly DC, Sellon S. (2004) *Equine Internal Medicine.* Second Edition, pp:429-432.
14. Fregin GF, (1982) The equine electrocardiogram with standardized body and limb positions. *The Cornell Veterinarian.* 72 (3): 304-324.

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