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Clinical, Hematobiochemical and Cardiac Markers Evaluation of Horses Affected with Upper Respiratory Disease

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Abstract

EQUINE DISEASE of the upper respiratory tract is one of the more common medical conditions faced by horses. Therefore, this study aimed to conduct a survey on the most common cause of upper respiratory disease of horses in relation to clinical, haematological and biochemical changes. Special attention was given to the effect of inflammatory respiratory disease on cardiac function and muscle enzyme activity and their relations with trace element status. The study was applied to a total number of 120 horses (10 control and 110 diseased showed upper respiratory disease signs). Blood samples obtained from control and affected animals. Swabs from abscessed L. ns were collected from diseased animals. The most prevalent clinical findings that appeared on horses with upper respiratory tract affections in this study were inappetance, fever, pharyngitis, cough, dyspnea, dysphagia, serous, purulent and mucopurulent nasal discharge, painful swelling and abscesses in submandibular, parotid and retropharyngeal lymph nodes that occurred as complications of the disease in addition to guttural pouch empyema. Bacteriological results revealed isolation of *Streptococcus. equi* as a major causative agent confirmed by PCR and there were significant increases in white blood cells, neutrophils, platelets count, cardiac troponin I, creatine kinase-myocardial band, procalcitonin, tumor necrosis factor alpha, interleukein-6, total CK, aspartate aminotransferase, fibrinogen and ceruloplasmin. While significant decrease in red blood cells, hemoglobin, hematocrit, lymphocytes, eosinophils, monocytes, iron, zinc and selenium were recorded. In conclusion, these findings highlight the need of early detection and targeted treatment options for management of upper respiratory disease in horses.

Keywords: Cardiac troponin I (cTnI), Procalcitonin (PCT), Tumor necrosis factor alpha (TNF- α), Equine, Upper respiratory.

Introduction

Respiratory diseases considered an important cause of poor performance in horses [1]. Equine infectious upper respiratory tract disease (IURD) recognized as one of the more common medical conditions faced by equine practitioners nationwide [2]. It is an acute clinical disease syndrome that may be attributed to multiple respiratory viruses and bacteria, including equine herpesviruses, equine influenza virus and *Strep. equi subsp. equi* which is one of the most common pathogens recognised in this syndrome [3]. *Strep. equi subsp. equi* (SEE) which causes the disease commonly known as strangles is a gram-positive, capsulated β-hemolytic lancefield group C *Streptococcus*, only infects equines due to its high host adaptation and considered a bacterial infection that is both highly infectious and contagious affects upper respiratory tract in horses, mules and donkeys of any age [4]. The clinical signs of the disease in general characterised by inappetence, dysphagia and abrupt onset of pyrexia followed by pharyngeal swelling and abscess formation in the submandibular and retropharyngeal lymph nodes [5]. Numerous documented complications related to SEE infection typically arise from the local hematogenous or lymphatic dissemination of bacteria to various sites or as aconsequence of its toxin [6].

Actually, cardiac troponin I (cTnI) has been identified as the most effective biomarker for detecting diseases of myocardium in the field of

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equine medicine [7]. Moreover El-Deeb et al.[8] demonstrated increased cTnI levels in strangles affected horses .in addition,creatinine kinase myocardial band (CK-MB) which is another cardiac biomarker is predominantly located in the cells of the heart muscle. It has been noticed that there is an increase associated with exercise and with chest pain in human [9].

Elevation in plasma muscle enzyme levels, creatinine kinase (CK) and aspartate aminotransferase (AST) are often used as non-invasive indicators or biomarkers of muscle injury or irritation in horses [10]. Increases the permeability of the muscle membrane may lead to release of CK and AST, which is not exclusive to muscles. However, its increase is usually only due to muscle damage [11]. Arabian horses that were infected with *S. equi* demonstrated a notable rise in activity of serum AST that may be due to the local myositis that may be attributed to opened abscesses in the affected horse [12]. Procalcitonin (PCT) seems to serve as an initial indicator of systemic inflammatory response syndrome (SIRS) caused by bacterial infections in both human [13] and horses [14]. Besides, procalcitonin is regarded as a crucial indicator for monitoring the advancement of infections, particularly pneumonia and sepsis, as its levels may assist in directing antibiotic treatment [15].

Tumor necrosis factor (TNF-α) is an essential component of the pro-inflamatory process,it affects immune cells and has the potential to induce cell death .in addition, IL-6 is aversatile cytokine that plays asignificant role in initiating the acute phase response subsequent to injury and infection. While certain effects of this cytokine and other pro-inflammatory cytokines can be beneficial, but excessive expression can be detrimental, resulting in the worsening of the condition or even mortality[16]. Increased TNF-α concentrations were found after ex vivo stimulation with bacterial products in recurrent airways obstruction affected horses [17]. In addition, IL-6 was increased in foals and horses affected with upper and upper complicated with lower respiratory diseases [18].

Fibrinogen is classified as an acute phase reactant protein its levels rise 24 hours following the onset of inflammation and might not peak for 2-3 days [19]. Moreover, reported hyperfibrinogenaemia in horses with strangles [20].

The plasma protein ceruloplasmin (Cp), an acute-phase reactive protein was identified as being present at elevated levels during the intermediate or later stages of acute inflammation in horses [21]. Ceruloplasmin is essential for iron metabolism. With ceruloplasmin–bound copper constituting 95% of copper metabolism in plasma.additionally,it is recognized that ceruloplasmine levels rises in response to both sterile and surgical inflamations [22].Previous research has demonstrated that trace minerals exhibit adynamic mechanism during the progression of infectious diseases [23].Some researchs indicated that systemic inflammation is linked to reduced levels of certain trace minerals [24].

Thus, the aim of the study was to conduct a survey on the most common cause of upper respiratory disease of horses in relation to clinical, haematological and biochemical changes. Special attention was given to effect of inflammatory respiratory disease on cardiac function and muscle enzyme activity, and their relations with trace element status.

Material and Methods

Animals

This current study was conducted on 120 (110 diseased and 10 control) horses aged from (2±1) year with average body weight (85-250) kg. Sporadic cases were brought to Mashtoul Alsouq Veterinary Training Center, Sharkia governorate, Egypt in the period from January 2023 to January 2024 were used in the present study. The study was carried out in accordance with Benha University Ethics Committee's approval with approval number (BUFVTM 36-09-23). Clinical and physical examinations were applied on all selected cases for determination of body temperature, heart and respiration rates, mucous membranes and examination of superficial lymph nodes. Auscultation of heart and lung sounds was applied [25]. According to physical examination, the animals were classified into two groups. First group was the control group which included 10 apparently healthy horses. The second group includes 110 horses clinically infected with *Strep. equi subsp. equi* that were confirmed by PCR.

Samples

Blood Samples

Only 30 affected horses were selected for sampling based on physical examination and clinical signs of upper respiratory infection. 5 ml of blood was withdrawn from the jugular vein in a sterile vacuum tube (with EDTA) for hematological evaluation. Another 5 ml of whole blood was withdrawn in a sterile vacuum tube containing (potassium citrate) for separation of plasma. An additional 5 ml blood sample was collected into a clean, dry test tube without anticoagulant. The sample was allowed to clot at room temperature and then centrifuged for 10 minutes at 3000 rpm to extract clean non-hemolyzed serum. The serum and plasma samples were then transferred into a clean, dry and sterilized Eppendorf tube and stored at -20 °C until they were needed for biochemical analysis.

Nasal Swabs

Nasal swab samples from abcessiated draining lymph nodes were cultured for bacterial growth.

Bacterial Culturing, Isolation and Identification

30 swabs from opened abscess lymph nodes were cultured firstly on on plates containing selective Edward media for streptococcus and incubated at 37°C for 24h then β-hemolytic colonies were subcultured on blood agar media and incubated at 37°C for 24 h under anaerobic conditions. Typical colonies of beta hemolytic *streptococci*-like appear as dew drops were detected then further confirmation for identification of the colony characteristics. The pure isolates of bacteria were identified by using Gram stain according to Collee et al.,[26] we conducted biochemical tests in accordance with Quinn et al., [27]. The differentiation of *Strep. equi subsp. equi* from *Strep. equi subsp. zooepidemicus* was based on the characteristic that *Strep. zooepidemicus* is capable of fermenting sorbitol and lactose. While *Strep. equi subsp. equi* cannot ferment sorbitol and lactose and ferment salicin and sucrose [28] .

DNA Extraction and Convential PCR

For the purpose of extraction of DNA from samples, the QIAamp DNA Mini kit Catalogue no.51304 (Qiagen, Germany, GmbH) was utilized after some modifications occurred to the manufacturer's instructions. The oligonucleotide primers were provided by Metabion (Germany) and the PCR primer sequences are stated in Table (1). Preparation of PCR_Master Mix for cPCR_according to Emerald Amp GT PCR mastermix (Takara) Code No.RR310Akit. The PCR products were analyzed following the method described by Hamouda et al., [29].

Hematological Analysis

Complete blood count (CBC) including red blood cells (RBCs) count, hemoglobin (Hb) concentration, hematocrit (Hct), platelets (PLTs) count, total leukocytic count (WBCs), MCH , MCHC and MCV were evaluated by Hematological analyzer (Model No. 93-91098-00-GF) [31].

Biochemical Analysis

Cardiac troponin I (cTnI) levels were assessed in serum using fullautomated biochemical analyser (VetScan i-STAT® 1, Abaxis, CA, USA) according to the manufacturer's instructions. Chemistry analyzer (VetScan HM5, Abaxis, CA, USA) was used to assess the levels of total CK and AST in serum. Monitoring the activities of CK-MB was performed using colorimetric method and test kits provided by Beckman Coulter inc. (USA) following the method described by Hughes, [32]. according to the instructions of the kit's producer serum ceruloplasmin (Cp) was determined by commercially available kit (catalog no. 4096-1000; Biovision Inc, CA) in accordance with the method outlined [33]. Procalcitonin was measured using a commercial ELISA kit designed for equine species (Horse Procalcitonin ELISA kit; My BioSource San Diego, USA). In addition, determination of the concentration of TNF-α in serum was done by using an equine ELISA immunoassay kit (Genorise Scientific, USA). The concentration of IL-6 was estimated in serum by using equine commercial kits (Egyptian Company for Biotechnology (S.A.E), Obour city industrial area, Cairo, Egypt). Additionally, fibrinogen was determined in citrated plasma according to the procedure described by Campbell et al.,[34]. Estimation of selected trace minerals (iron, copper, selenium and zinc) levels using commercially available kits (Spectrum Diagnostic-Egypt).

Statistical analysis

The statistical analysis was performed using SPSS software (IBM, SPSS Statistics, Version 22, USA). The descriptive data are expressed as mean ± standard error (SE). The obtained results were analysed using one-way ANOVA test; all data are listed as mean ± SE. The significance level was set at $P < 0.05$.

Results

Clinical Findings

The most prevalent clinical findings appeared on horses with upper respiratory tract affections were inappetence, fever, pharyngitis, cough, serous, purulent and mucopurulent nasal discharge, painful swelling and abscesses in submandibular, parotid and retropharyngeal lymph nodes, dyspnea and dysphagia. In addition to guttural pouch empyema (Fig. 1). Moreover, abnormal inspiratory noises identified as stridor were heard during tracheal auscultation. The auscultation of the lung revealed normal lung sounds confirmed by ultrasonography. Evaluation of the heart rate indicated presence of tachycardia and respiratory rate showed tachypnoea. The frequency of most prevalent clinical findings showed in Table (2). The more common clinical finding was l.ns enlargement then nasal discharge and fever.

Isolation and Identification of Bacterial Culture and Confirmation by PCR

Typical colonies of *Strep. equi* were detected and isolated then molecular differentiation of both subspecies of *Strep. equi* from closely related species by molecular assays which target specific genetic markers sodA gene, and the seel gene which is specific for *Strep. equi subsp. equi*. Samples give positive result for reaction of sodA gene, which is specific for *streptococcu*s spp, and give positive

reaction with seeI gene, which is one of the genes specific for *streptococcus. equi subsp. equi* (Fig. 2).

Hematological Findings÷

Hematological analysis showed that there were significant ($p \leq 0.05$) reduction in RBCs count, Hb concentration, Hct , lymphocytes, monocytes and eosinophils with significant ($p \leq 0.05$) elevation in total WBCs, neutrophils, MCH ,MCHC and PLT counts in diseased group compared to control group (Table, 3). In addition, the significant increase in MCH and MCHC with no change in MCV indicate normocytic hyperchromic anemia.

Biochemical Findings

The result of cardiac and inflammatory biomarkers showed that there were significant ($p \leq 0.05$) elevation in concentrations of cTnI, CK-MB, PCT, TNF- α and IL-6 in diseased horses compared to control group (Table, 4). Significant ($p \leq 0.05$) elevation in total CK and AST activities and concentrations of fibrinogen and ceruloplasmin were recorded in diseased group compared to control group (Table, 5). In addition, analysis of serum trace mineral showed that there were significant ($p \leq 0.05$) reduction in Fe, Zn and Se levels with non-significant increase in Cu in diseased group compared to control levels (Table, 6).

Discussion

Acute infectious upper respiratory tract disease (IURD) represents an important disease syndrome in equine [3]. Even though *Streptococcus equi subsp. equi* (SEE) infection holds a relatively low mortality and high morbidity rates. It possesses a significant global challenge owing to its infectious characteristics, the potential for quarantine measures and the considerable economic impact on the equine industry [35]. The streptococcal species Streptococcus equi subsp zooepidemicus and Streptococcus equi subsp. equi, which are closely related, were identified through polymerase chain reaction (PCR) utilizing oligonucleotide primers that were specifically designed based on unique regions of the super oxide dismutase A encoding gene sodA [36]. The Superoxide dismutase A (sodA) gene region is commonly found in *Streptococcus* species. Therefore, this region was incorporated into the assays to achieve a positive or negative result at the genus level [37].

The results of the present study detected isolation and molecular identification of *Streptococcus equi* which produced amplified products for seeI and sodA genes at 520 and 235 base pairs (bp) respectively. This result was coincided with Farhan and Yousseff [38]. On the other hand, the recorded clinical signs in our study were in line with Fridberg et al. [39]. Likewise, lymphadenopathy considered a major clinical sign of *Strep. equi* infection which was reported by Sweeny et al. [40]. Regarding to the haematological changes, normocytic hyperchromic anemia was reported in the present study .This was cleared with the significant decrease in erythrocytic count, Hb concentration and Hct value with significant increase in MCH and MCHC and normal value of MCV. This could be as a result of reduction in iron level due to the block of its release from reticuloendothelium storages because of the inflammatory process induced by the infectious pathogen which inturn became inaccessible for Hb synthesis leading to the inhibition of erythropoiesis [12].

The observed leucocytosis due to neutrophilia associated with lymphopenia, eosinopenia and monocytopenia resulted from *Strep. equi* infection in equines, was like to those recorded by Ijaz et al. [20], Farhan and Yousseff, [38]. Results of our study revealed a significant neutrophilia which is usually associated with the inflammatory processes or infectious diseases [31].

Our study demonstrated elevated levels of cTnI in diseased horses, which may suggest a possible potential myocardial injury, and this was coincided with El-Deeb et al. [8]. In this study, elevated levels of cTnI in affected horses could indicate a possible myocardial injury. Auscultation of the heart alone is insufficient to distinguish pre-existing heart disease. Therefore, the existence of an initial myocardial condition or pre-existing heart disease cannot be disregarded. Elevated values of cTnI have been reported in non-cardiac disorders, such as sepsis in foals [41]. In addition, the increased levels of cTnI in the current study could be attributed to an inflammatory origin (sepsis). The mechanism of increased levels of cTnI in cases of sepsis is not fully acknowledged [42]. Moreover, it thought that antigens of SEE trigger inflammation of the myocardium, causing electro-cardiographic abnormalities in convalescent horses [35]. On the other hand, in this study, the levels of myocardial depressants such as TNF-α and IL-6 were significantly elevated in the serum of horses with strangles. The release of TNF-α increase the permeability of endothelial monolayers to macromolecules at the level of myocardial cells results in release of cytoplasmic cTnI without cardiomyocyte necrosis, that was agreed with De'Ath et al. [43].

In addition, our results revealed a significant elevation in creatine kinase-myocardial band (CK-MB) in *Strep. equi* infected horses and these results consider one of the few papers that measured levels of CK-MB in SSE infected horses. CK-MB is an enzyme variant predominantly located in cardiac muscle cells . It is typically evaluated alongside cTnI or utilized in its absence. In human,CK-MB serves as ales sensitive biomarker compared to cTnI for the evaluation of myocardial injury [44]. With this aspect, increased levels of both cTn-I and CK-MB

have been reported in horses after a long-distance endurance ride [45]. Furthermore, it was reported that certain horses that were diagnosed with myocarditis might have aprior history if upper respiratory illness based on aclinical association between prior disease and cardiac disease . This conclusion is derived from the findings reported in humans [46].

The result of the current study showed increase the levels of total CK and AST that was in line with Farhan and Yousseff, [38]. Which could be due to myositis resulted from the effects of the toxins of SEE on muscle tissue, which have been hypothesised by Boyle et al. [35]. Alternatively, it may be due to local myositis that resulted from opened abscesses [12]. Moreover**,** Hassanpour & Fartashvand, [47] reported increase the levels of AST and CK in *Strep. equi* infected horses.

The results of the current study revealed increased levels of procalcitonin (PCT) in diseased horses. PCT is released from numerous tissues and cell types in the body [48]. The observed increase of PCT within 3 to 6 hours suggests that it may serve as an early indicator of systemic inflammatory response syndrome (SIRS) triggered by bacterial infections . This rapid elevation is particularly notable in cases of bacterial infections and endotoxemia indicating its potential role as an early marker for these conditions in human patients [13]. It was reported that adult horses, foals and cattle showed elevated levels of PCT during abnormal medical conditions caused by bacteria or by the translocation of bacteria and/or their products into the bloodstream [49]. PCT was increased in horses suffered from systemic inflammatory response syndrome (SIRS) [14], and in foals suffered from acute respiratory distress syndrome [50].

The current study revealed significant increase in TNF-α concentration that was agreed with El-Deeb et al. [8]. TNF-α plays a fundamental role in the pro-inflammatory process [16]. The release of TNF-α has been reported to play a crucial role in initiating early pro-inflammatory cytokine and immune responses during the host defence mechanisms in both horses and humans [51]. Increased TNF-α concentrations were found after ex vivo stimulation with bacterial products in recurrent air ways obstruction affected horses [52].

Our results revealed significant elevation in concentrations of IL-6, and this was in line with El-Deeb et al. [8]. There are conflicting studies that suggested that IL-6 has both anti-inflammatory and pro-inflammatory roles [53]. On the other hand, Pihusch et al. [54] reported that interleukin (IL-6) and PCT levels were increased after infectious complications.

The current work revealed hyperfibrinogenemia, and this was agreed with Ijaz et al, [20] and Sweeny et al., [40]. Fibrinogen constitutes the predominant fraction of plasma proteins produced during an acute phase response. It is regarded as a relatively unresponsive marker of inflammation, attributed to its extensive reference range and prolonged response time [31]. Fibrinogen was considered an indicator of active inflammation. In horses with severe equine asthma, fibrinogen was demonstrated to exceed the levels observed in the control group [55].

The results of the current study reported elevated levels of ceruloplasmin (Cp) that was in line with Rad et al., [56]. Ceruloplasmin (Cp) is characterized as an acute-phase reactive protein, which is observed at increased concentrations during the intermediate or later stages of acute inflammation in equines [21].

Our study demonstrated significant decrease in Zn and Se levels with non-significant increase in Cu levels, which was agreed with Rad et al. [56]. There is evidence that the presence of systemic inflammation is linked to reduced serum concentrations of Fe [24]. Moreover, It was suggested that in strangles because of inappetence, serum levels of these elements will be faced with deficiency.

Conclusion

These findings highlight the need of early detection and targeted treatment options for the effective management of bacterial upper respiratory disease in horses by demonstrating the disease's substantial impact on the immune system and inflammatory response by evaluation of hematological parameters, cardiac and inflammatory biomarkers, muscle enzymes, acute phase proteins and trace elements status. This early diagnosis could be useful to overcome secondary complications including guttural pouch empyema and subsequent respiratory distress.

Conflicts of Interest

The authors have no conflicts of interest to declare.

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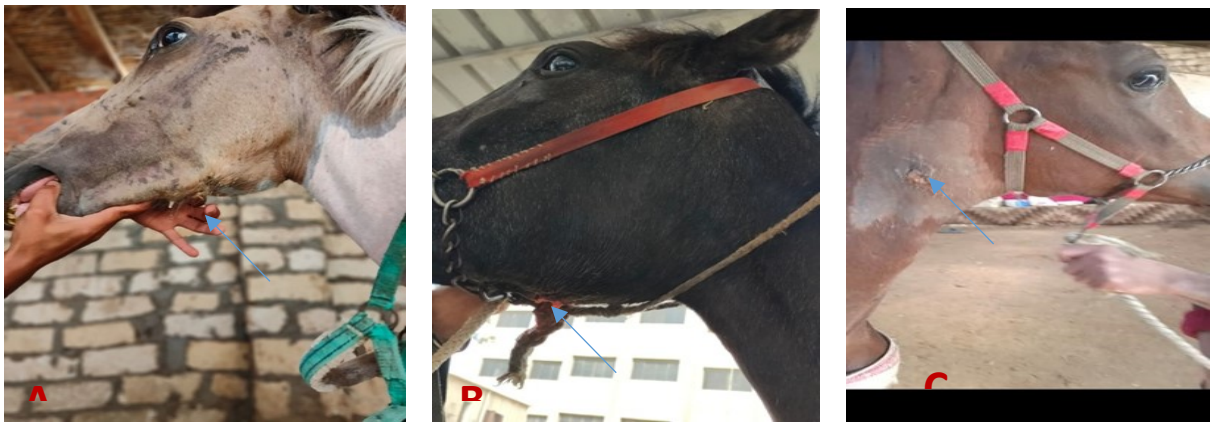

Fig. 1. Showing clinical manifestations of upper respiratory tract infection in horse that include pus oozing from submandibular l.n (A), enlarged and opened mandibular L.n (B), guttural pouch empyema(C).

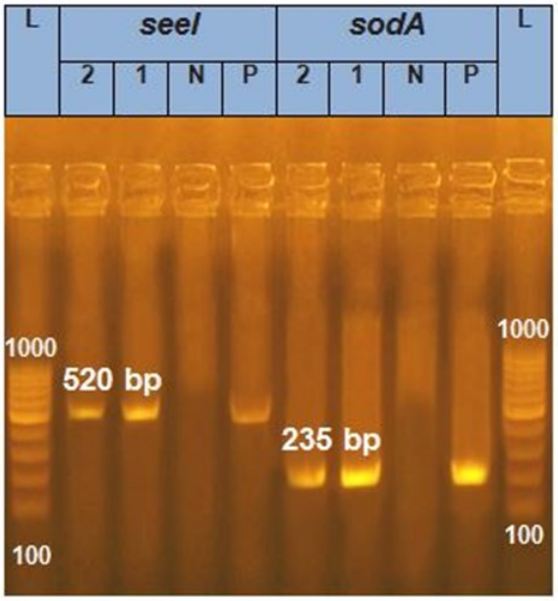

Fig. 2. Agar gel electrophoresis for Streptococcus spp. The product of sodA reaction (235 base pairs, bp) demonstrating specificity of sodA primer for Streptococci. The bands reaction at (520) bp for the SeeI gene indicates the presence of *S. Equi. Equi.*
Key: L = DNA ladder, N. = negative control, P. =positive control.

TABLE 1. Oligonucleotide Primers Sequences

Target gene	Primer sequence (5'-3')	Length of amplified product (bp)	Reference
***S. equi* sodA**	CAG CAT TCC TGC TGA CAT TCG TCA GG CTG ACC AGC CTT ATT CAC AAC CAG CC	**235**	[30]
***S. equi subsp. equi* seeI**	GAA GGT CCG CCA TTT TCA GGT AGT TTG GCA TAC TCT CTC TGT CAC CAT GTC CTG	**520**	

TABLE 2. Frequency of Clinical Signs in Upper Respiratory Tract Affected Horses

Clinical symptoms	No. of animals N=110	%
Fever	75	68.1
Anorexia	45	40.9
Dysphagia	23	20.9
Serous nasal discharge	5	4.5
Purulent nasal discharge	35	31.8
Mucopurulent nasal discharge	48	43.6
Dry cough	6	5.4
Moist cough	47	42.7
Dyspnea	28	25.4
Tachycardia	65	59
Increased respiratory rate	25	22.7
Submandibular L. n enlagement	68	61.8
Parotid L. n enlagement	10	9.09
Retropharyngeal L. n enlagement	20	18.1
Mucous membrane congestion	37	33.6
Guttural pouch empyema	10	9.09

TABLE 3. Hematological Findings in Control and Diseased Horses

Parameters	Control group N=10	Diseased group N=30
RBCs (10^3/µl)	6.32±0.35[a]	4.90±0.22[b]
Hb (g/dl)	13.90±0.10[a]	11.55±0.12 [b]
HCT%	38.83±1.01[a]	30.17±0.70 [b]
MCV (f L)	61.43±0.08[a]	61.55±0.21[a]
MCH(pg)	21.99±.003 [b]	23.50±.007[a]
MCHC(g/dl)	35.79±.021 [b]	38.26±.014[a]
PLT (10^3/µl)	153.83±3.11[b]	398.00±25. 51[a]
WBCs (10^3/µl)	9.22±0.31[b]	16.17±0.95[a]
Neutrophil (10^3/µl)	49.47±1.49[b]	83.50±0.76[a]
Bands (10^3/µl)	0.00±0.00[b]	2.00±0.37[a]
Segmented (10^3/µl)	49.47±1.49[b]	81.50±1.02[a]
Lymphocytes (10^3/µl)	36.95±1.53 [a]	13.00±0.73 [b]
Monocytes (10^3/µl)	10.97±0.89 [a]	1.83±0.31 [b]
Eosinophils (10^3/µl)	2.48±0.49[a]	1.67±0.21 [b]

Data in table represent value ± SE. Values with different superscripts letters within the same row were statistically significant at ($P<0.05$) (one way a nova, test n= 2).

TABLE 4. Cardiac and Inflammatory Biomarkers in Control and Diseased Horses

Parameters	Control group N=10	Diseased group N=30
cTnI (mg/dl)	0.02±0.002 [b]	0.45±0.31[a]
CK-MB (ng/ml)	2.15±0.02 [b]	2.87±0.06 [a]
PCT (ng/ml)	0.05 ±0.004[b]	0.17 ±0. 006[a]
TNF-alpha (pg/ml)	35.67±1.05 [b]	118.66±1.86[a]
IL-6 (pg/ml)	2.50±0.06 [b]	5.1±0.24[a]

Data in table represent value ± SE. Values with different superscripts letters within the same row were statistically significant at ($P<0.05$) (one way a nova, test n= 2).

TABLE 5. Muscle Enzymes and Acute Phase Proteins in Control and Diseased Horses

Parameters	Control group N=10	Diseased group N=30
Total CK (U/L)	169.17±2.76[b]	283.17±6.20[a]
AST (U/L)	290.67±2.08[b]	361.17±9.87[a]
Fibrinogen (mg/dl)	299.50±18.31 [b]	527.00±20.32[a]
Ceruloplasmin (mg/dl)	31.83±1.45 [b]	49.67±1.94[a]

Data in table represent value ± SE. Values with different superscripts letters within the same row were statistically significant at (P<0.05) (one way a nova, test n= 2).

TABLE 6. Trace Minerals (Fe, Cu, Zn and Se) in Control and Diseased Horses

Parameters	Control group N=10	Diseased group N=30
Fe (µg/dl)	97.33±2.73[a]	56.50±5.41[b]
Cu (µg/dl)	104.33±0.88[b]	107.00±0.93 [b]
Zn (µg/dl)	74.67±0.88[a]	59.17±1.30[b]
Se (µg/dl)	0.14±0.004[a]	0.11±0.003[b]

Data in table represent value ± SE. Values with different superscripts letters within the same row were statistically significant at (P<0.05) (one way a nova, test n= 2).

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تقييم العلامات الإكلينيكية والكيميائية الدموية والقلبية للخيول المصابة بأمراض الجهاز التنفسي العلوي

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الملخص

تعتبر أمراض الجهاز التنفسي العلوي من أكثر الأمراض شيوعاً بين الخيول، لذلك هدفت هذه الدراسة إلى إجراء مسح حول السبب الأكثر شيوعاً لأمراض الجهاز التنفسي العلوي لدى الخيول فيما يتعلق بالتغيرات الإكلينيكية والدموية والكيميائية الحيوية. وقد تم إعطاء إهتمام خاص لتأثير أمراض الجهاز التنفسي الإلتهابية على وظائف القلب ونشاط إنزيمات العضلات وعلاقتها بحالة العناصر النادره. وقد أجريت الدراسة على إجمالي عدد (120) حصاناً (10 من المجموعة الضابطة و110 من الخيول المريضة التى أظهرت علامات التهاب الجهاز التنفسي العلوي). وكانت النتائج السريرية الأكثر انتشاراً على الخيول المصابة بأمراض الجهاز التنفسي العلوي في هذه الدراسة هي فقدان الشهية والحمى وإلتهاب البلعوم والسعال وإفرازات الأنف المصلية والصديدية والمخاطية الصديدية والتورم المؤلم والخراجات في الغدد الليمفاوية تحت الفك السفلي والغدة النكفية والبلعوم الخلفي كمضاعفات للمرض وضيق التنفس وصعوبة البلع. بالإضافة إلى إحتقان الجيب الحنجري. تم الحصول على عينات الدم من الحيوانات السليمة ومن الحيوانات المريضة وجمع مسحات من الغدد الليمفاوية المصابة بخراريج مفتوحة فى الحيوانات المريضة. أظهرت النتائج البكتريولوجية عزل العقدية الخيلية كمسبب رئيسي تم تأكيده بواسطة تفاعل البوليميراز المتسلسل، وكانت هناك زيادات كبيرة في كريات الدم البيضاء والنيتروفيل والصفائح الدموية وتروبونين القلب Tumor necrosis factor alpha وبروكالسيتونين والفيبرينوجين Ck-MB وIL6 وCK وAST و Cp ومع إنخفاض كبير فى كرات الدم الحمراء والهيموجلوبين ووالهيماتوكريت والخلايا الليمفاوي والسيلينيوم والزنك والحديد eosinophils و خلايا الإزينوفيل والمونوسيت.

وفي الختام، تسلط هذه النتائج الضوء على الحاجة إلى الكشف المبكر وخيارات العلاج المستهدفة لإدارة أمراض الجهاز التنفسي العلوي في الخيول.

الكلمات الدالة: التروبونين القلبي (cTnI) I، البروكالسيتونين (PCT)، Tumor necrosis factor alpha (TNF- α)، الخيول، الجهاز التنفسي العلوي.