STUDIES ON CONTAGIOUS SKIN NECROSIS AND TRYPANOSOMOSIS IN CAMELS

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SUMMARY

Camels showed clinical signs of contagious skin necrosis (CSN), with or without trypanosomosis, were subjected to study. The following samples were collected; sterile bacteriological swabs from skin necrosis area, whole blood samples for hematological analysis and for diagnosis of trypanosomosis, and serum for measuring lipid peroxidation product (Malondialdehyde, MDA). The bacteriological examination of collected swabs from dermal lesion of CSN revealed that Staphylococcus aureus was the predominant bacterial isolate alone in 6 cases and coupled with other bacteria in the remained 4 cases, coupled with coagulase negative staphylococci 3 cases and coupled with Streptococcus agalactiae in one case. Trypanosoma evansi infection was identified using polymerase chain reaction in 5 camels that had CSN. Malondialdehyde showed significant increase in camels affected with CSN that associated with trypanosomosis. The current study revealed that Staphylococcus aureus was the predominant bacterial isolate, camels may be infected with both trypanosomosis and CSN, lipid peroxidation products increased in the blood of camels with CSN that associated with trypanosomosis, it is recommended to supply camels with antioxidants to overcome the deterioration of blood antioxidants.

Keywords: Camels, CSN, Trypanosoma, MDA

INTRODUCTION

Contagious skin necrosis (CSN) is a chronic inflammation of the skin primarily caused by Staphylococcus aureus, occurred mainly in young dromedaries and localized mostly in the shoulder and neck regions [8]. The disease began with signs of folliculitis, which frequently progressed to a furunculosis with individual or grouped small abscesses. These abscesses could become large and when lanced yield whitish-green pus, and the disease could be chronic and difficult to treat medically depending on the pathogenicity of the staphylococcal strain present [7, 12, 13, 20]. Arthropods were incriminated as transmitting vectors of the various types of the isolated bacteria [17]. Trypanosomosis in camel caused by Trypanosoma evansi is still a serious problem in camel husbandry, causes considerable economic losses in many camel-rearing regions of the world [6, 15]. The course of the infection is often chronic and the parasitological diagnosis is usually difficult, because the parasitaemia is low or no trypanosomes are found in the blood [21]. With the introduction of molecular diagnostic techniques, several diagnostic assays based on the detection of trypanosomal DNA by PCR have been developed [1]. The goals of the present study are to identify the causative microorganism of CSN in the dromedary camels and to evaluate the effect on the health status of camels.

MATERIAL AND METHODS

The investigated camels were clinically inspected [14] for the presence of skin necrosis or abscesses, the suspected cases were subjected to detailed clinical examination and samples collection. The following samples were collected; sterile bacteriological swabs from skin necrosis area, whole blood samples for hematological analysis [5], and for diagnosis of trypanosomosis, and serum for measuring lipid peroxidation product (Malondialdehyde, MDA).

Sterile bacteriological swabs were taken from the opened cutaneous abscesses of the infected camels after disinfection also the necrosed skin was detached and the underlaying tissue was swabbed. These swabs were taken for identification of the bacterial pathogens following standard bacteriological techniques [4, 16]. Serum malondialdehyde (MDA) was measured by using commercial kits (Bio-diagnostic, Egypt) and by means of Digital VIS/Ultraviolet Spectrophotometer (Cecil instruments, Cambridge, England, Series No. 52.232).

For detection of trypanosoma infection 2-5 thin blood films from each camel were prepared [5]. Negative cases were subjected to molecular diagnosis using PCR technique, the PCR technique was performed according to established method [2]. Primers of Trypanosoma evansi were as follow: Primer 1: 5’-CGATGAATATTAAACTGGCAGT-3’, and Primer 2: 5’-AGAACATTAGTTTGTTGC-3’.
Statistical analysis

Data were expressed as mean ± SD. Statistical analysis was conducted using SPSS 16.0 for windows (SPSS, Chicago, USA). The difference in the blood constituents among the investigated groups were compared using one way analysis of variance followed by least significant difference (LSD) post-hoc analysis, significant difference was considered at \( p<0.05 \).

RESULTS

Contagious skin necrosis

Ten camels showed characteristic dermal lesions of contagious skin necrosis. These lesions were demonstrated on different parts of the animal body, as an area of skin necrosis, in which the skin looked black in color and not covered with hair. These areas sharply separated from the surrounding healthy skin, and they were cold and very hard in consistency. When an area of necrosed skin was detached, circular ulcer of varying diameter, usually 2-10 cm, remained and clearly demarcated from surrounding healthy tissue (Fig. 1). This ulcer was filled with large amounts of whitish pussy material tinged with blood and may reach up to 10cm in depth. The bacteriological examination of collected swabs from dermal lesion of CSN revealed that *Staphylococcus aureus* was the predominant bacterial isolate alone in 6 cases and coupled with other bacteria in the remained 4 cases, coupled with coagulase negative staphylococci 3 cases and coupled with *Streptococcus agalactiae* in one case.

![Figure 1: Camels with CSN](image)

*Trypanosoma evansi* infection was identified using polymerase chain reaction in 5 camels that had CSN.

Comparing data from camels with CSN groups with the control group revealed significant decrease in total RBCs count \( (p<0.05) \) in camels suffering from CSN alone. Serum malondialdehyde showed significant increase in camels affected with CSN with trypanosomosis \( (p<0.01) \), when compared with the control healthy camels.

| Table 1. Measured blood constituents and MDA levels in investigated camels |
|-----------------|------|-----|---------|------|------|------|-----|
|                | RBCs (x10^6/ul) | Hb (g/dl) | PCV (%) | MCV (pg) | MCH (fl) | MCHC (g/dl) | MDA (nmol/ml) |
| Control        | 8.35±1.21a      | 12.2±1.73a | 28.5±2.3a | 34.7±5.6a | 14.7±1.6a | 43.3±7.5a     | 1.0±0.6a      |
| CSN            | 6.2±1.8b        | 10.7±2.2a  | 25.4±3.8a | 40.0±6.5a | 15.7±3.3a | 39.4±5.3a     | 3.2±1.7ab     |
| Tryp.+ CSN     | 7.2±1.3ab       | 11.2±2.2a  | 26.8±3.3a | 37.9±7.5a | 15.8±4.1a | 41.6±5.3a     | 5.0±2.6b      |

In each column, different letter means significant, Tryp.: Trypanosomosis.

DISCUSSION

In the current study, lesions of CSN were found on the back of the animal, sides, shoulder region, gluteal region and in the ventral aspect of the neck. Similar field observation was previously recorded \([7, 11]\), who noted that lesions of staphylococcal dermatitis were usually situated in the gluteal, perineal and lower cervical regions. The main clinical findings in camels suffering from CSN are agreed with previous studies \([7, 11, 14, 20]\).

The obtained results revealed that the main isolated bacterial species from CSN affected camels was *S. aureus* (60.61%), while coagulase negative staphylococci
represented 27.27% and *Streptococcus agalactiae* was 12.12%, these results greed with previous studies [20], which stated that staphylococcal dermatitis primarily caused by *S. aureus*. The obtained results are in harmony with previous studies [7, 8, 14], they reported that CSN in camels caused by a number of bacteria including *S. aureus*. In contrast to other studies [9], which concluded that the main isolated bacteria from CSN was *Streptococcus agalactiae* followed by *S. aureus*. Lipid peroxidation in biological samples [19] and the metabolic fate of malondialdehyde has been extensively studied [3, 18, Hjelle and Petersen 1983]. Unlike reactive free radicals, aldehydes are rather long lived and, therefore, can diffuse from their site of origin (i.e., membranes) to reach and attack other targets intracellularly or extracellularly [10]. The increased MDA in the current study indicated increased oxidative stress in blood of camels with CSN coupled with trypanosomosis, and this may attributed to decreased antioxidant levels or may be due to excessive release of free radicals.

**CONCLUSIONS**

The current study revealed that *Staphylococcus aureus* was the predominant bacterial isolate, camels may be infected with both trypanosomosis and CSN, lipid peroxidation products increased in the blood of camels with CSN and it is recommended to supply camels with antioxidants to overcome the deterioration in blood antioxidants status.

**REFERENCES**

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