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Clinical, Hematological, and Biochemical Alterations Associated With Early and Late Infection of Lumpy Skin Disease in Cattle in Egypt

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ABSTRACT

This study aimed to investigate clinical, hematological, and biochemical alterations associated with early (during the first week.) and late (after one month) of lumpy skin disease (LSD) infection in cattle infected during an outbreak of LSD in Egypt. Animals which were clinically examined directly after the clinical onset of LSD showed, firm elevated skin nodules that were distributed all over the body, fever (more than 40°C) associated with anorexia, general weakness, reduced milk yield, enlarged superficial lymph nodes, and edema in the brisket and/or limbs. Examination of the affected animals one month after the disease onset revealed marked weight loss, shrunk of skin nodules and formation of sit fast. There were significant increases in body temperature, pulse and respiratory rate in all animals infected with LSD with significant decrease of ruminal movement compared to control group. DNA extracted from skin samples collected from LSD infected animals showed bands at the expected size (172 base pairs) by partial amplification of viral attachment protein using PCR. Reduced RBCS count, Hb content, HCT, and MCHC values with a significant increase in MCV in all LSD infected cows compared to healthy control group, while leukogram analysis revealed leucopenia and lymphopenia during the 1st week post-infection, however, one month post-infection revealed granulocytic leukocytosis. In addition, all LSD infected cows showed inflammatory thrombocytopenia. Animals during early LSD infection showed significant reduction in total proteins, albumin, glucose and TAC and significant increase in ALT, AST, ALP, LDH, CPK, C-troponin-1, total and direct bilirubin, creatinine, K and MDA levels compared to the control group. While animals during late infection showed significant decrease in glucose, ALT, AST, LDH, CPK, C-troponin-1, direct bilirubin, K and MDA levels and significant increase in total protein, albumin and TAC compared to the early infection group. It can be concluded that, LSD infection in cattle revealed critical alterations in some hematological and serum biochemical parameters during both early and late stages of infection with negative impact on different organ functions including skin, muscle, liver, kidney and heart.

1. INTRODUCTION:

Lumpy skin disease (LSD) is an endemic infectious viral disease transmitted by blood-feeding vectors caused by Lumpy skin disease virus (LSDV) of *Capripoxvirus* genus, subfamily *Chordopoxvirinae*, family *Poxviridae* causing high morbidity and low-to-moderate mortality in cattle and buffaloes. (Gupta et al., 2020; Koirala et al., 2022). Cattle infected with LSDV shows characteristic clinical signs which include fever, ocular discharge, enlarged lymph nodes, edema, raised nodules on skin of the muzzle, nares, back, legs, scrotum, perineum and eyelids (Salib and Osman, 2011), with a serious complications that include corneal opacity, decreased milk production, recumbency, mastitis, cellulitis, phlegmon, myiasis

(permanent damage of skin) which causes lowering of their commercial value, abortion, dysentery, lameness, and pneumonia, thus LSD is associated severe economic losses (Abera et al., 2015). Post-mortem examination of the affected cattle may reveal widespread pox lesions throughout the gastrointestinal and respiratory tracts besides classical skin lesions, limb edema in one or more limbs (Babiuk et al., 2008). Characteristic signs, histopathology, virus isolation, polymerase chain reaction (PCR) as well as immunohistochemistry can all be used to diagnose LSD in infected cattle (Tuppurainen and Oura, 2012; Badr et al., 2022). The pathogenesis of LSD in cattle has been studied before, but there are few reports that demonstrated the full hematological, biochemical alterations observed during early and late LSD infection. Therefore, this study aimed to fully describe the clinical, hematological and biochemical alterations associated with early (within 1 week of the appearance of clinical signs) and late (one month later) LSD infection in cattle during an outbreak of LSD in Egypt with special attention to some serum biomarkers as indicators of tissue damage.

2. MATERIALS AND METHODS:

2.1. Animals and study area:

Clinical examination and collection of samples were performed during the outbreaks of Lumpy skin disease in Egypt during 2018 from cattle backyard at El-Behira province. Forty mixed breed Cattle (cross breeding of native cattle with the Holstein–Friesian cattle) were clinically examined early at the onset of the disease (within one week of appearance of clinical signs); in addition, the required clinical parameters (body temperature, pulse, respiratory rates and ruminal movements were measured according to (Kelly, 1984). After taking the required samples, ie. blood samples for hematological examination and serum samples for biochemical analysis and skin tissue for PCR detection, the animals were treated with a three days injection course of flunixin meglumine as an anti-inflammatory and antipyretic drug (Flamicure®, Pharma Sewede, Egypt), and broad-spectrum antibiotic preparation containing a combination of penicillin G and streptomycin (Pen & Strep®, Norbrook, United Kingdom) for prevention of secondary bacterial invasions. Fluid therapy and multivitamins were also administered as a supportive treatment. The animals were re-examined again for clinical signs, hematological and biochemical evaluation (after one month from the disease onset). Another ten apparently healthy normal cattle were enrolled as a control group.

The research protocol was approved by the ethical committee of Faculty of Veterinary Medicine, Alexandria University and Damanhour university. All animal handling and procedures were carried out in accordance with the national guidelines for animal care and welfare.

2.2. Samples Collection and preparation:

Two blood samples (with and without EDTA) were aseptically collected from the jugular vein of healthy, early infected and late infected animals via traumatic venipuncture using Vacutainer tubes. Whole blood samples with EDTA were used for hematological analysis, and samples without EDTA were centrifuged at 3000 rpm for 15 min for serum separation and stored at −20°C for biochemical analysis.

Skin tissues were aseptically collected from the erupted skin nodules of cattle showing clinical signs of LSD (n=5) to confirm clinical diagnosis of LSD, transported into the laboratory, stored at at 4 ℃ then DNA extraction was performed by using QIAamp DNA mini-Kit (51304, QIAGEN, Hilden, Germany).

2.3. Viral detection by PCR:

PCR was performed using primers specific to the gene encoding for the viral attachment protein of Capri poxviruses (ORF074) **(Ireland and Binepal, 1998)**. The forward primer (5' TTTCCTGATTTTTCTTACTAT 3'), and the reverse primer (5' AAATTATATACGTAAATAAC 3') were used. These primers amplify a 172 base pair fragment. For PCR mixture, a final volume of 25 µl reaction mixture containing 2.5 µl of 10× PCR buffer for KOD –Plus- Neo (Toyobo), 1.5 µl of MgSo4 (25 mM), 2.5 µl of dNTPs (2 mM), 0.75 µl of the forward primer, 0.75 µl of the reverse primer, 1 µl of DNA template, 0.25 µl (0.25 U) of KOD –Plus- Neo DNA polymerase (Toyobo), and 15.75 µl of nuclease-free water, was prepared. Negative and positive controls were included by using distilled water and DNA of sheep pox (extracted from sheep pox vaccine- Servac Capri-C, Veterinary Serum and Vaccine Research Institute, Cairo, Egypt), respectively. The thermal profile was as follows: an initial cycle at 98 °C for 2 minutes, and then 35 cycles of 98 °C for 10 seconds (s), 50 °C for 30 s and 68 °C for 10 s and a final extension step of 68 °C for 7 min. PCR products were electrophoresed using 1.5% agarose gel and images visualized using UV trans-illuminator.

2.4. Hematological analysis:

Complete blood cell counts including total white blood cells (WBCs), red blood cells (RBCs), hemoglobin (Hb), hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC) and platelets count (PLT)

were performed with EDTA-treated blood collected from healthy control, early and late LSD infected animals using a fully automated veterinary hematology analyzer (Exigo, Boule medical AB., Sweden) in the central laboratory, Faculty of Veterinary Medicine, Alexandria University. Blood smears were prepared, fixed with absolute methanol (95%) and stained with Giemsa stain to determine differential leukocyte counts (DLCs) (Feldman et al., 2000).

2.5. Biochemical analysis:

Serum total proteins, albumin, globulin (by subtracting the albumin from the total proteins), alanine amino transferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), lactate dehydrogenase (LDH), total and direct bilirubin, glucose, creatinine, and blood urea nitrogen (BUN) were measured by using commercial test kits (Bio-labo, France). Malondialdehyde (MDA) and total antioxidant capacity (TAC), as well as serum sodium, potassium, and chloride were measured using commercial test kits (Biodiagnostics, Egypt). Serum cardiac troponin I (cTn I) was analyzed by using an enzyme-linked immunosorbent assay (ELISA) kit (Monobind Inc, Lake Forest, CA, USA). Meanwhile, creatine phosphokinase (CPK) activity was measured using commercially available test kits (Spectrum diagnostics, Germany). All parameters were analyzed following standard methods mentioned in the leaflet of the manufacturer.

2.6. Statistical analysis:

One-way analysis of variance (ANOVA) with Tukey Kramer post-hoc testing was performed in Graph Pad Prism 5 software (San Diego, CA, U.S.A.) to investigate clinical examination, hematological and biochemical parameters. All comparisons were considered significant at $P \leq 0.05$.

3. RESULTS:

3.1. Clinical examination:

Animals were clinically examined directly after the clinical onset of LSD. Eruption of firm elevated skin nodules that were distributed all over the body was the constant clinical feature of LSD (Fig. 1A, B, and C). The number of nodules varied. High fever (more than 40℃) associated with anorexia, general weakness, and reduced milk yield were observed. Other clinical manifestations were enlarged superficial lymph nodes, and edema in the breast and/or limbs.

Examination of the affected animals one month after the disease onset revealed marked weight loss. Skin nodules shrunk, with hair loss in the center of the nodules which formed thick scab (sit fast) (Fig. 1D), that detached easily leaving wound (Fig. 1E), which heals with periodical using of mild anti-septic on the exposed wound. Occasionally, the small adjacent wounds coalesce, forming large wounds (Fig. 1F).

There was a significant elevation of body temperature, pulse and respiratory rates in animals infected with LSD in both early and late stages of infection compared to the control group ($P \leq 0.05$) (Table 1).

Also, the effect of LSD infection on ruminal movements is shown in Table1; animals infected with LSD in both early and late stages of infection have a significantly reduced ruminal movement compared to the control group ($P \leq 0.05$).

3.2. Viral detection by PCR:

DNA extracted from skin samples collected from LSD infected animals showed bands at the expected size (172 base pairs) by partial amplification of the gene encoding viral attachment protein (ORF074) (Fig. 2).

3.3. Hematological analysis:

As shown in **Table (2)**, there was a significant decrease in total RBCs count, Hb content, HCT and MCHC values in animals infected with LSD in both early and late stages of infection compared to the control group ($P \leq 0.05$). In contrast, there was a significant increase in MCV value in all animals infected with LSD compared to control group ($P \leq 0.05$).

Early LSD infected group showed a significantly reduced number of total WBCs, lymphocytes and neutrophils compared to the control group ($P \leq 0.05$). However, in late LSD infected group, there were a significant increase in the number of total WBCs and lymphocytes compared to early LSD infection groups ($P \leq 0.05$). Also, there was a significant increase in the number of neutrophils in animals in the late stage of LSD infection compared to both control and early LSD infected groups ($P \leq 0.05$).

As for other types of cells less frequently observed in the complete blood count (MID cells), late LSD infected group showed a significant increase in the number of these cells compared to control and early LSD infected groups ($P \leq 0.05$). For platelets count, early and late LSD infected groups showed a significant decrease compared to control group ($P \leq 0.05$).

Table.1. Clinical parameters of cattle at early lumpy skin disease infection (within one week of clinical onset) and late infection (one month later) compared to control group:

Group	Body Temperature^{0}C	Respiratory rate/ min.	Pulse rate /min.	Ruminal movement / 2 min.
Control group N=10	38.72 ± 0.370	23.40 ±3.209	65.80±6.419	3.200 ±0.447
Early LSD infection group N=40	40.10 ±0.628 *	42.40±7.635*	89.60±8.562*	1.600 ±0.547 *
Late LSD infection group N=40	40.62 ±0.683 *	46.80 ±7.918 *	92.20±16.35*	1.200 ±0.447 *

*****Significantly different from the control group at P≤0.05.

Table 2: Hematological evaluation of cattle at early lumpy skin disease infection (within one week of clinical onset) and late infection (one month later) compared to control group:

Group	RBCs ($X10^6$/µl)	Hb (mg/ 100ml)	HCT (%)	MCH (Pg.)	MCHC (g/dl)	MCV (fl)	WBC ($X10^3$/µl)	Lymph. ($X10^3$/µl)	MD ($X10^3$/µl)	Neutrophils ($X10^3$/µl)	Platelets ($X10^3$/µl)
Control group	6.700 ± 0.50	9.590 ± 0.655	31.26 ± 1.262	13.10 ± 0.435	33.36 ± 0.413	47.49 ± 0.665	10.33 ± 0.577	5.320 ± 0.399	1.197 ± 0.205	3.963 ± 0.040	325.2 ± 18.76
Early LSD infection group	5.967 ± 0.251*	7.683 ± 0.815 *	30.76 ± 0.488 *	12.92 ± 0.951	30.53 ± 1.524 *	49.07 ± 0.940 *	6.000 ± 1.0 *	2.510 ± 0.418 *	0.90 ± 0.14	2.347 ± 0.319 *	186.8 ± 17.48 *
Late LSD infection group	5.740 ± 0.461*	7.613 ± 0.57 *	29.48 ± 0.627 *	11.61 ± 0.432	29.76 ± 0.431 *	50.70 ± 0.574 *	13.67 ± 0.577 #	5.683 ± 0.518 #	2.387 ± 0.45 * #	5.173 ± 0.243 * #	228.7 ± 19.02*#

*Significantly different from control group at P≤0.05.
#Significantly different from early LSD infected group at P≤0.05.

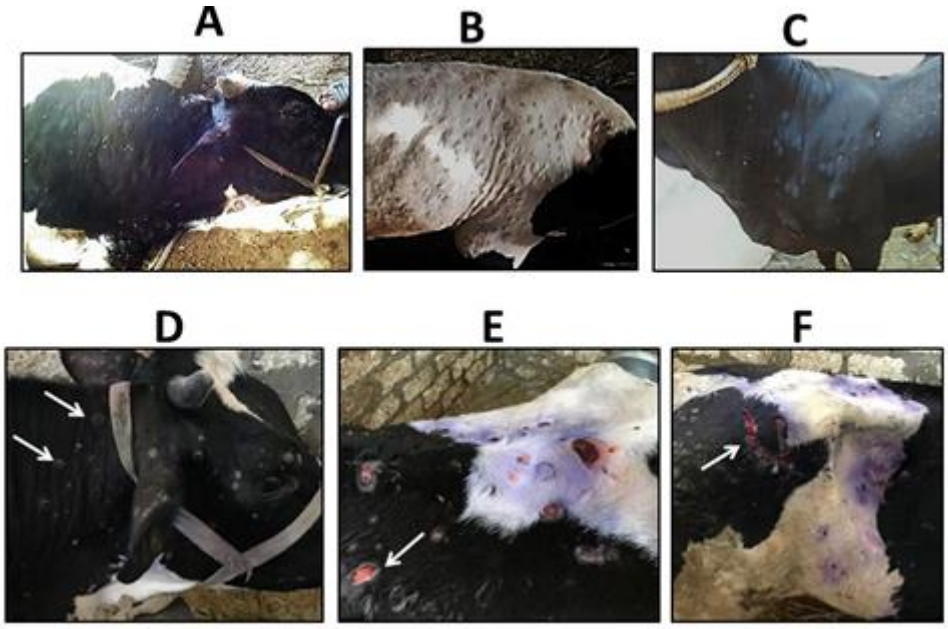

Fig. 1: Lumpy skin disease-induced skin lesions at the early stage of infection (A, B, and C) as well as at the late stage (one month after disease onset) (D, E, and F): formation of easily detached thick scab (sit fast) (D); exposed skin after detachment of the scab (E); coalesce of adjacent exposed skin lesions to form a large wound (F). White arrows refer to the explained lesions.

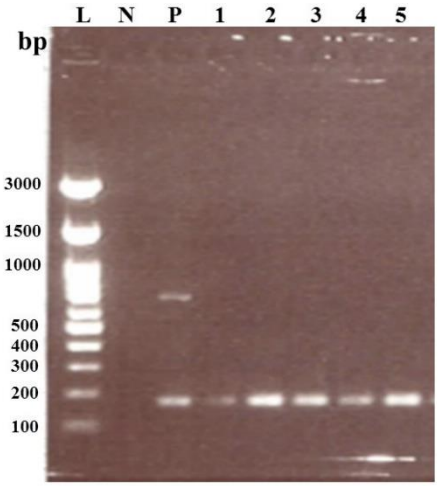

Fig. 2: Detection of gene encoding viral attachment protein (172 base pairs) of capripoxviruses in the five collected skin samples. Lanes: L, 100 bp ladder (DM003-R500, GeneDirex, Taiwan); N, negative control; P, positive control; 1-5, DNA extracted from the five skin samples. Sizes of some ladder bands are shown at left in base pairs.

3.4. Biochemical analysis:

Early LSD infected group showed a significant decrease in total protein and albumin levels compared to the control group (P≤0.05). However, the late LSD infected group that showed a significant increase in total protein and albumin level compared to early LSD infection (P≤0.05**)** (Fig. 3A & B). Neither Early LSD infected group nor late LSD infected group had any significant change in globulin level (Fig. 3C).

Early LSD infected group exhibited a significantly elevated ALT, AST, and ALP activities compared to control group (P≤0.05). Meanwhile, the late LSD infected group showed a significant reduced ALT and AST activities compared to early LSD infection (P≤0.05) (Fig. 4A, B, C). However, the late LSD infection has no significant change in ALP activity (Fig. 4C). Early LSD infected group showed a significant increase in LDH activity compared to control group (P≤0.05). While, late LSD infected group that showed significant decrease in LDH activity compared to early LSD infected group (P≤0.05) (Fig. 4D)

Early LSD infected group showed a significant increase in CPK and C-troponin-1 levels compared to

control group (P≤0.05). Whereas, late LSD infected group showed significant decrease in CPK and C-troponin-1 levels compared to early LSD infected (P≤0.05) (Fig. 5 A, B). Regarding the effect of LSD infection on total (T.) and direct (D.) bilirubin, early LSD infected group showed a significant increase in T. bilirubin and D. bilirubin levels compared to the control group (P≤0.05). However, the late LSD infected group showed non-significant changes on T. bilirubin level with a significantly decreased D. bilirubin levels compared to early LSD infected group (P≤0.05) (Fig. 5 C, D). Also, early LSD infected group showed a significant increased creatinine level compared to control group (P≤0.05). However, late LSD infected group showed non-significant changes in creatinine level. BUN levels showed non-significant changes in both early LSD and late LSD infected groups as compared to control group (Fig. 5 E, F).

Both early LSD and late LSD infected groups showed a significant reduced blood glucose levels compared to control group (P≤0.05) (Fig. 6A). Regarding the effect of LSD on serum electrolyte levels; early LSD infected group showed significantly increased serum K level compared to healthy control group (P≤0.05). However, the late LSD infected group showed a significant decrease in K levels compared to early LSD infected group (P≤0.05) (Fig. 6B). Noteworthy, changes in serum Na and Cl levels were non-significant in either early or late stage groups (Fig. 6B, C, D).

Concerning the effect of LSD infection on TAC level, early LSD infected group showed a significant decrease in TAC level compared to control group (P≤0.05); nonetheless, the late LSD infected group showed significant increase in TAC levels compared to early LSD infected group (P≤0.05) (Fig. 7A). On the level of lipid peroxidation, early LSD infected group showed significant increase in MDA levels compared to control groups (P≤0.05); nevertheless, the late LSD infected group showed significant decrease in MDA levels compared to early LSD infected group (P≤0.05) (Fig. 7A, B).

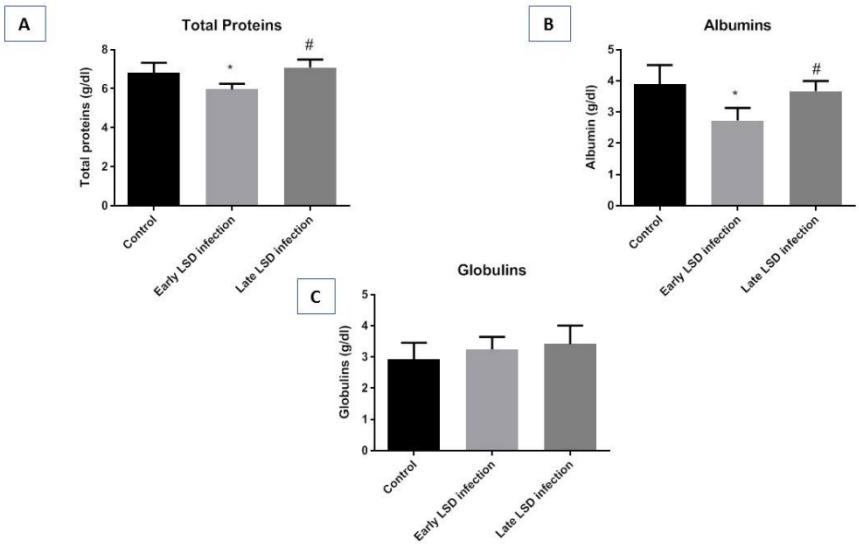

Fig. 3. Serum total proteins, albumins, and globulins (g/dl) levels of infected animals (Early LSD infection and late LSD infection) compared to the control group. (*) significantly different from the control group; (#) significantly different from the early LSD infection group at P≤0.05.

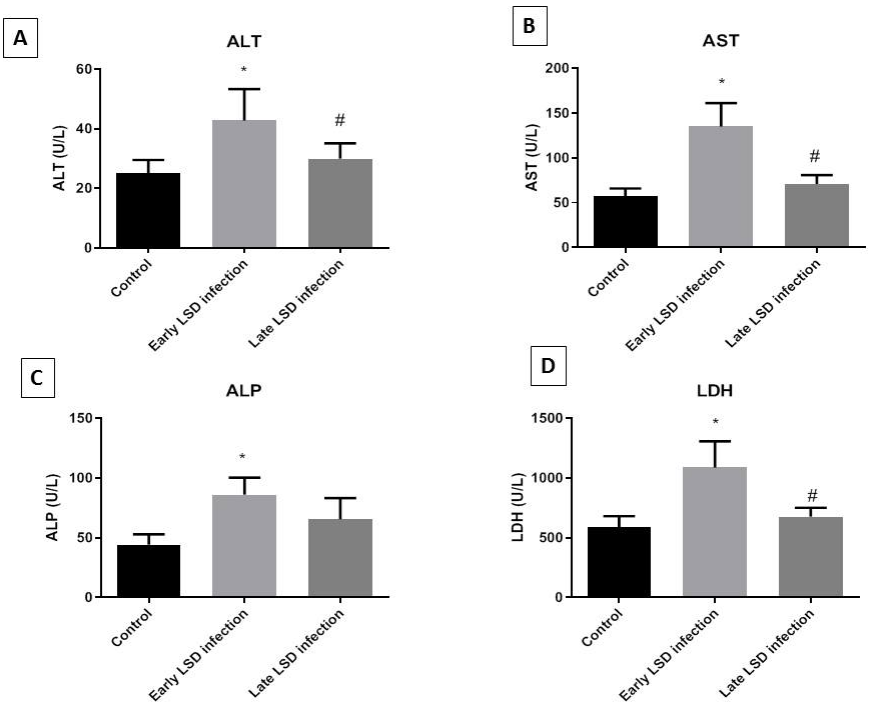

Fig. 4. Serum ALT, AST, ALP, and LDH (U/L) levels of infected animals (early LSD infection and late LSD infection) compared to the control group. (*) significantly different from the control group; (#) significantly different from the early LSD infection group at P≤0.05.

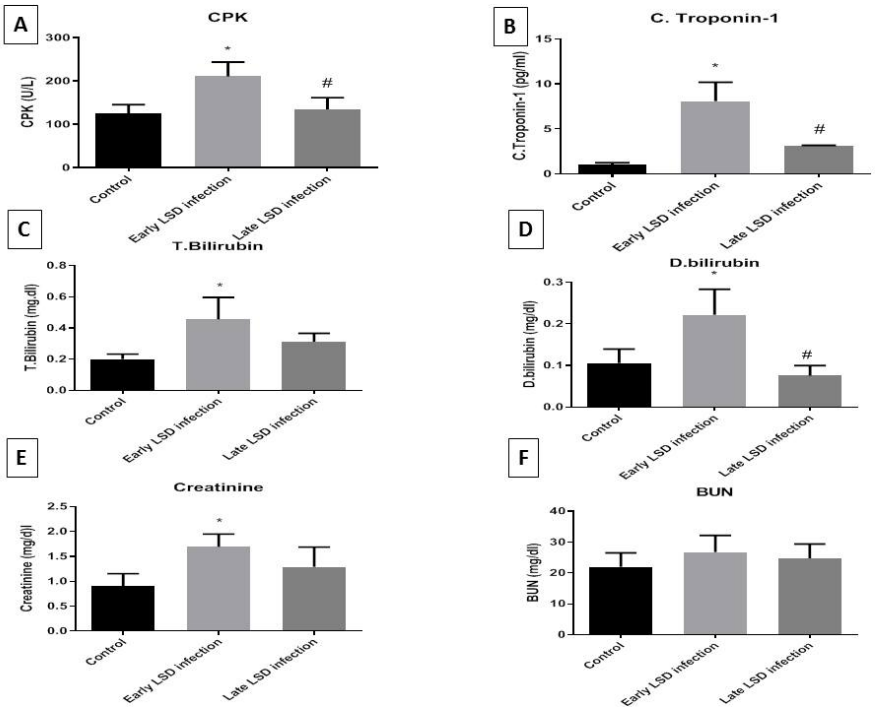

Fig.5. Serum CPK (U/L), C. troponin-1 (pg/ml), total (T) and direct (D) bilirubin (mg/dl), creatinine (mg/dl), and BUN (mg/dl) levels of infected animals (early LSD infection and late LSD infection) compared to the control group.
(*) significantly different from the control group; (#) significantly different from the early LSD infection group at P≤0.05.

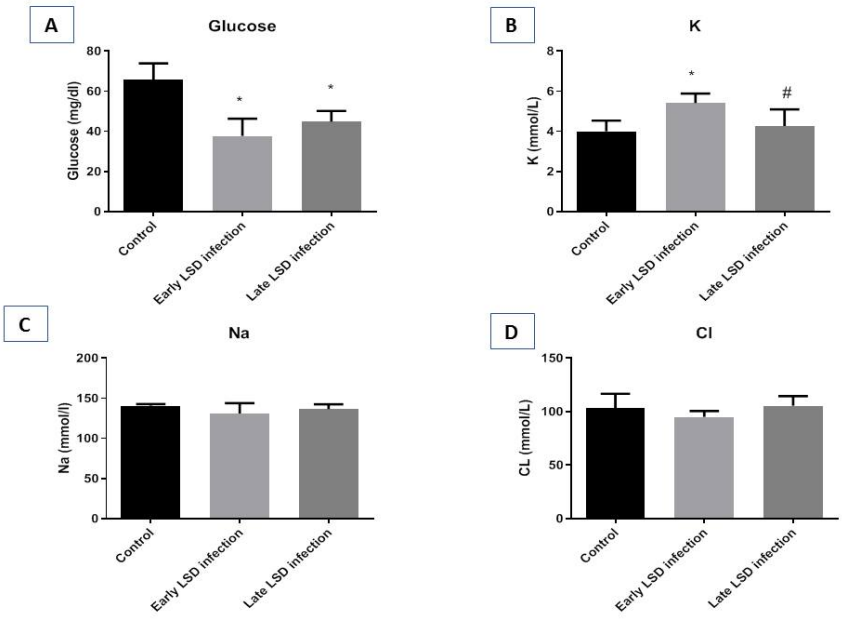

Fig. 6. Serum glucose (mg/dl), K, Na, and Cl (mmol/l) levels of infected animals (early LSD infection and late LSD infection) compared to the control group. (*) significantly different from the control group; (#) significantly different from the early LSD infection group at P≤0.05.

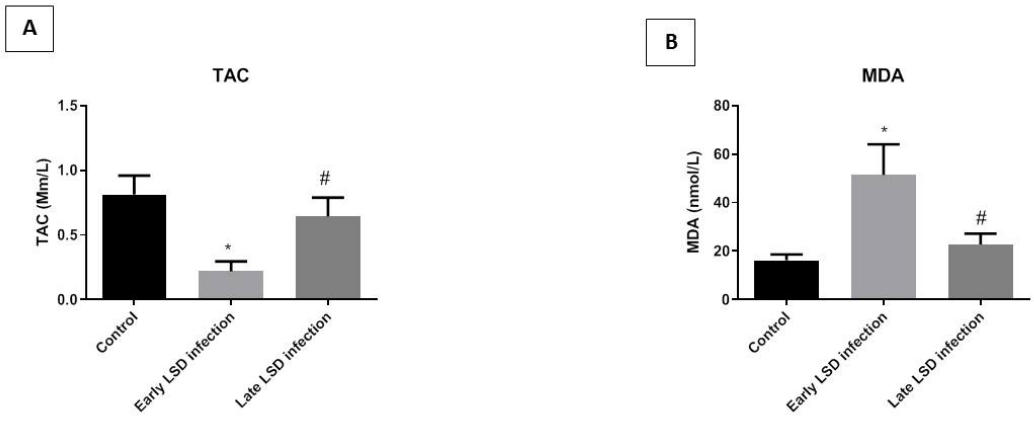

Fig. 7. Serum total antioxidant capacity (TAC) (Mm/l) and malondialdehyde (MDA) (nmol/l) levels of infected animals (early LSD infection and late LSD infection) compared to the control group. (*) significantly different from the control group; (#) significantly different from the early LSD infection group at P≤0.05.

4. DISCUSSION:

LSD has been attracting much attention in the last few years, due to its invasion into new regions and the high economic losses which it induces to cattle industry.

By clinical examinations of the infected animals directly after the onset of clinical signs revealed; eruption of firm elevated skin nodules which were distributed all over the body, high fever (more than 40℃) associated with anorexia, general weakness, and reduced milk yield, enlarged superficial lymph nodes, and edema in the breast and/or limbs. After one month, skin nodules shrunk, with hair loss in the center of the nodules which formed thick scab (sit fast) that detaches easily leaving wound and occasionally, the small adjacent wounds coalesce forming large wounds. These findings are typical for LSD and consistent with (Rouby et al., 2021; Jafarsab et al., 2022b; Badr et al., 2022). Detection of viral DNA in skin samples collected from LSD infected cattle by PCR confirmed the clinical diagnosis of LSD infection. Significantly elevated body temperature, pulse and respiratory rate is in

accordance with (Jafarsab et al., 2022a; Jafarsab et al., 2022b). This significant increase in pulse rate may be due to anemia and heart affection which will be discussed later. Prolonged fever observed in late LSD infected animals may be due to secondary bacterial infection (Coetzer and Tustin, 2004). Significant decreased ruminal movement in both early and late LSD infected animals may be due to occurrence of fever and anorexia among them (Van Miert, 1987). LSD not only affected clinical situation of infected cattle but also affected significantly internal hemogram including reduced RBCS count, Hb content, HCT, and MCHC values with a significant increased MCV values in all LSD infected cows compared to healthy control one. Leukogram analysis revealed leucopenia and lymphopenia during the early infection, however, after one month of infection granulocytic leukocytosis was observed. In addition, all LSD infected animals showed inflammatory thrombocytopenia.

Erythrogram results coincides with that of (Neamat-Allah, 2015) who stated that lumpy skin diseased cows were suffered from macrocytic hypochromic anemia which mostly developed due to anorexia with simultaneous decrease in serum iron (Latimer, 2011; Morceau et al., 2009). This type of anemia is mild and slowly progressive (Abutarbush, 2015) and considered as anemia of inflammatory type which is usually associates chronic diseases like LSD and is mainly caused by increased inflammatory cytokines causing lower bone marrow responsiveness to erythropoietin (Jalali et al., 2017). Leukogram analysis revealed marked leucopenia and lymphopenia during the early infection, which are in agreement with the findings of (Neamat-Allah, 2015; El-Mandrawy and Alam, 2018) which is mainly returned to viral infection especially associating viral pathogenesis including systemic spread and environmental viral shedding. In addition to that lymphopenia in infected animals may be due to release of excess quantity of endogenous corticosteroids (Neamat-Allah, 2015) with lymph node sequestration of peripheral blood lymphocytes (Latimer, 2011). While after one month of infection, granulocytic leukocytosis was detected, with intensified production of neutrophils associating mainly secondary pyogenic bacterial infection, these findings were previously confirmed by (Smith, 2002; Neamat-Allah, 2015). Regarding thrombocytopenia recorded in all infected cows involved in this study may be attributed to shortened life span of platelets and marked platelets consumption due to widespread vasculitis and vascular thrombosis of infected cows according to (El-Mandrawy and Alam, 2018) .

Concerning serum biochemistry, significant decreased serum total protein and albumin levels during early LSD infection is in accordance with (Neamat-Allah, 2015; El-Mandrawy and Alam, 2018) this might be due to hepatic injury, decreased protein synthesis and higher catabolic rate (Hassan et al., 2011). However significant increase of total protein and albumin during late LSD infection compared to early infection is in accordance with (El-Mandrawy and Alam, 2018). This may be attributed to dehydration which causes an increase in total protein level. Also, might be returned to inflammatory changes in the body in addition to activation of immune response of the host following infection (Abutarbush, 2015; Jafarsab et al., 2022b). Significantly elevated serum activities of ALT, ALP and AST during early LSD infection compared to healthy control group may be due to primary hepatic injury by LSDV or secondary to anoxic necrosis of periacinar hepatocytes (Cockcroft) (Hassan et al., 2011). AST activity also may be elevated due to affection of cardiac muscles (Şevik et al., 2016). Elevation of cardiac troponin activity confirms the heart affection because it is more sensitive for cardiac muscles injury. This is in accordance with (Helal et al., 2019; Jafarsab et al., 2022b), so in our study, heart affection during early LSD infection is confirmed by elevated serum levels of both AST and cardiac troponin 1. Also, Serum LDH and CPK activities were significantly elevated during early LSD may be due to myocardial injury. These findings may reflect myocardial damage (Marmor et al., 1988). In addition, LDH activity may be elevated due to muscle damage resulting from muscular rigidity or activity during fever episodes (El-Mandrawy and Alam, 2018). These findings were in consistent with recent research recorded by (Jafarsab et al., 2022a; Jafarsab et al., 2022b). Significantly elevated serum levels of T. and D. bilirubin during early infection were in accordance with (El-Mandrawy and Alam, 2018; Jalali et al., 2017). Total and direct hyperbilirubinemia was recorded in early infected animals; this may attributed to the presence of LSDV in the blood, which may have damaged hepatocytes surrounding the bile duct, also may have been induced by intrahepatic cholestasis and existing biliary disorder (Scott and Stockham, 2013). Serum creatinine was significantly elevated in early LSDV infection which may be returned to wide spread muscle injury and damaging effects of LSDV on the kidneys (Coles, 1986). As reduced renal blood flow, decreased glomerular filtration and increased protein catabolic rate resulting in renal impairment with elevated serum creatinine level (Helal et al., 2019) . These results also in agreement with (El-Mandrawy

and Alam, 2018; Jafarsab et al., 2022b) and not in agreement with (Jalali et al., 2017) who recorded decreased serum creatinine level. Non-significant change in BUN in both early and late LSD infection and comparing to other researchers may be returned to differences in the stage of the disease, breed, and body condition of animal. Hypoglycemia in both early and late LSD infection could be attributed to prolonged anorexia associated with reduced ruminal activity and subsequent hepatic damage (Jafarsab et al., 2022b). Disturbance of some electrolytes balance are associated with LSD infection, hyperkalemia in both animals infected early and late with LSD virus may be due to the muscular damage that is associated with LSD infection which result in release of intracellular potassium content into the blood (Smith, 2002; Radostitis, 2010). This in accordance with (Abutarbush, 2015). In our study there were non-significant changes in serum level of both Na and Cl in early and late LSD infection. LSD infection is not usually associated with hyponatremia or hypocholermia. Only one study on LSD infection reported hypercholermia which was likely proportional and mainly caused by reduced water intake (Abutarbush, 2015). There are few available studies that's describe total blood antioxidants and lipid peroxidation status in LSD infection of cattle. In our study, there was a significant elevation in serum MDA in early LSD infected group compared to healthy control group. This result may be returned to oxidative stress injury due to excess free radical production from tissue damage and higher lipid peroxidation rate with exhaustion of antioxidants in the body (Ratnam et al., 2006) and (Elsayed et al., 2016). Also, there was a significant reduced serum levels of TAC during early infection compared to healthy control group, while there was a significant increase in serum levels of TAC during late infection as compared to early infection. These findings are in agreement with those recorded by (Helmy and Ahmed, 2017) and (El-Mandrawy and Alam, 2018) who reported reduced TAC in LSD-infected animals associated with excessive MDA production. Such imbalance between oxidants and anti-oxidants status can occur in different aspects of viral pathogenesis as replication, reduced immune cell reproduction, and function with cellular apoptosis (Neamat-Allah and Mahmoud, 2019).

5. CONCLUSION:

It can be concluded that infection of cattle with LSD resulted in sever alterations in some hematological and serum biochemical parameters during both early and late stages of infection. Collectively, clinical, hematological, and biochemical analysis of some specific parameters confirm skin, muscle, liver, kidneys, and heart affections. This study revealed a better understanding of lumpy skin viral pathogenesis for further insight to improve treatment strategies.

6. ACKNOWLEDGMENT:

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7. REFERENCES:

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