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SNUFFLES DISEASE IN RABBITS: 4- PATHOLOGICAL STUDY

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ABSTRACT
Snuffles disease is a major problem in rabbits,for this reason this study aimed to identify all the pathological findings associated with this disease process experimentally. *Pasteurella multocida* is the causative agent their LD50 was 10^7 CFU/ml, infective dose was 10^2 CFU/ml. Then two groups of rabbits. 1st group was control (7), the 2nd group was infected group (27) were injected with 10^2 CFU/ml I/P whereas control group injected with pbs. All the pathological lesions were recorded at 4th, 8th, 12th, 16th and 20th days post infection. During these periods different pathological lesions grossly seen such as congestion, hemorrhage edema, all over organs especially liver, lungs, kidneys, spleen, lymph nodes and heart enlargement. Microscopically, multiple abscesses in liver and chollangitis and extensive fibrinous and suppurative bronchopneumonia and fibrino purulent pleuritis. Kidneys showed extensive embolic nephritis together with bacterial colonies distributed in the suppuration. Mesenteric and prefemoral lymph nodes and spleen showed reactive lymphoid hyperplasia and micro abscess in white pulp of spleen and in lymphoid follicles of the lymph nodes giving suppurative and edematous lymphadenitis feature. *P. multocida* induces extensive fatal pathological lesions in the rabbits organs.

KEYWORDS: Snuffles in rabbits – pathological findings.

INTRODUCTION

Snuffles disease a major problem in rabbits caused by various *Pasteurella multocida* serotypes, the disease occurs during the stress factor such as shipping, mating, experimental handling and malnutrition[1,2]. *P. multocida* already present in upper respiratory tract and tympanic bullae of rabbits and during seasonal influences and other stresses factor the *P. multocida* multiply rapidly and induce the disease in the lungs and upper respiratory tract such as pneumonia, otitis media and rhinitis together with septicemia[3]. For the importance of this disease problem, this study aimed to identify pathological parameters associated with this experiment disease process in rabbits.

MATERIALS AND METHODS

A local strain of *Pasteurellamultocida*were reidentified [4] and the LD50 and infective lethal dose were indentified [5], the LD50 was 10^7 CFU/ml and infective dose was 10^2 CFU/ml. Two groups of rabbits were taken, first group 7 (control group) injected I/P with pbs, the second group 27 (infective group) injected I/P with 10^2 CFU/ml *P. multocida* and 4 rabbits sacrificed at 4 days intervals (at 4th, 8th, 12th, 16th and 20th) days post infection. The other rabbits[7] were died during the experiment period. All the dead and sacrificed rabbits organs were examined and pieces for histopathological study were taken, fixed routinely and processed and examined under light microscope[6].

RESULTS & DISCUSSION

Gross Pathology

The Lungs: Showed extensive congestion, hemorrhage, fibrin and edema in pulmonary tissue at the 4th – 12th days post infection, the consolidation and emphysema appeared at 12th – 20th days post infection (Fig – 1) together with pleural adhesions.

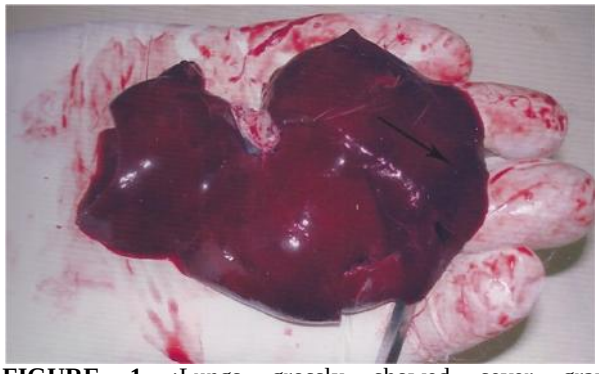

FIGURE 1 :Lungs grossly showed sever gray consolidation and emphysema (12th -16th) days of infection.

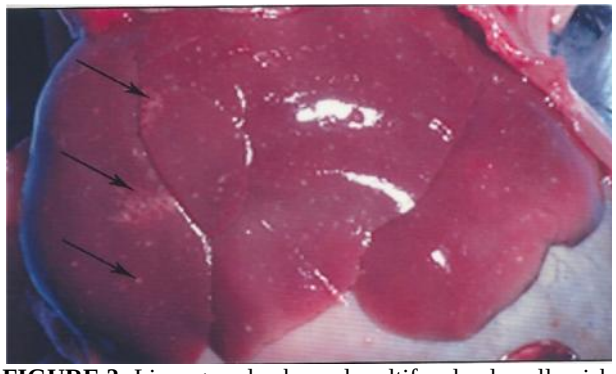

FIGURE 2: Liver grossly showed multifocal pale yellowish foci with rounded borders of liver parenchyma.

The Liver: Showed extensive congestion, edema and hemorrhage at the 4^{th} – 12^{th} days post infection, multiple pale yellow foci appeared on liver tissue at 12^{th} – 20^{th} days post infection together with swelling of gall bladder (Fig – 2)

The Kidney: Showed extensive congestion of the cortex and medulla, edema and hemorrhage and the kidney is soft in consistency along the period of experiment(Fig – 3).

The Lymph Nodes: All the mediastinal, mesenteric and prefemorallymph nodes showed edematous enlargement, congestion and hemorrhage along the experiment period (Fig – 3).

The Spleen: Showed sever congestion and hemorrhage together with edema in the whole splenic tissue (Fig – 4).

The Heart: Showed extensive hemorrhage and congestion between the muscle fibers and cardiac hypertrophy (Fig – 5).

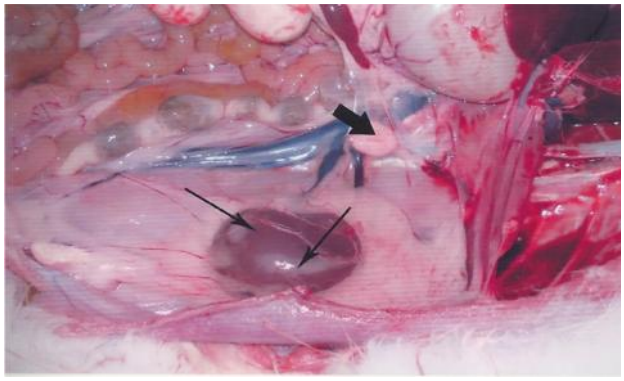

FIGURE 3: Kidney and mesenteric lymph node grossly showed enlargement and multi edematous areas.

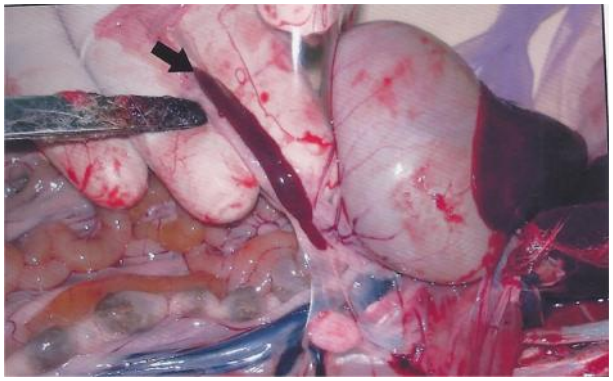

FIGURE 4: Spleen, grossly showed congestion and enlargement at 4^{th} day of infection.

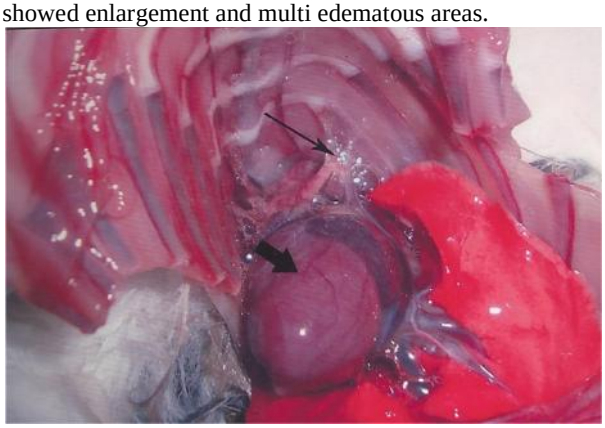

FIGURE 5 : Heart grossly showed enlargement and adhesion of the lung with thoracic cavity

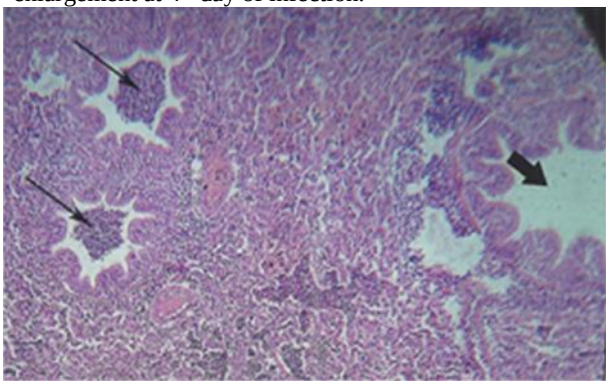

FIGURE 6: Lung tissue microscopically showed suppurative bronchopneumonia et the 12^{th} -16^{th} days of infection (H&E) X100

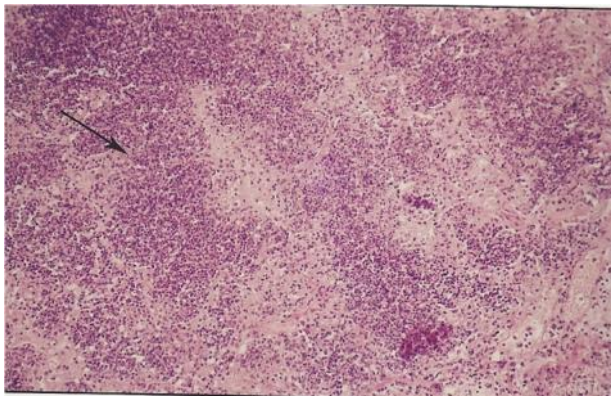

FIGURE 7: Lung tissue microscopically showed streaming macrophages exudate together with fibrin .(H&E) X100

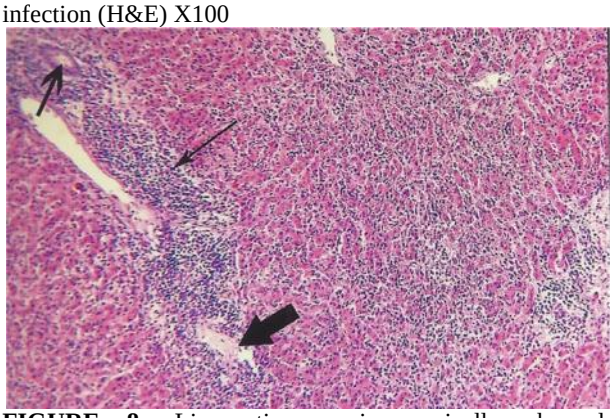

FIGURE 8: Liver tissue microscopically showed suppurative cholangitis , thrombus and perivascular leukocyte cuffing (H&E)X100

HISTOPATHOLOGY

The Lungs: It showed congestion of blood vessels, edema, fibrin and emphysema at the beginning and thickening of pulmonary interstitial tissue at 8^{th} day post infection, also there is fibrinous pneumonia and suppurative bronchopneumonia (Fig – 6) characterized by infiltration of neutrophils in bronchi, bronchioles and alveoli together with fibrin and edema. Steaming macrophages exudates

(Fig – 7) were seen in some alveolar spaces, hyperplasia of peribronchial lymphoid tissue. Also there is fibrino purulent pleuritis later on.

The Liver: The lesions began as congestion, perivascular leukocytes cuffing, thrombus formation and suppurative cholangitis (Fig – 8).Also multinecrotic areas in liver tissue lead to liver abscess formation (Fig – 9). Hypertrophy of hepatocytes lead to sinusoidal disappearance later on.

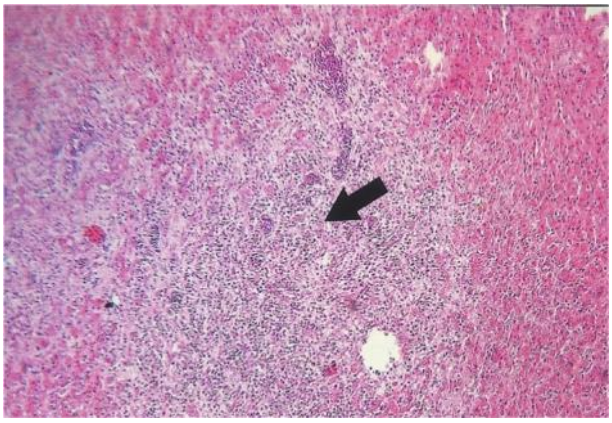

FIGURE 9: Liver tissue microscopically showed diffuse abscess formation (H&E)X100

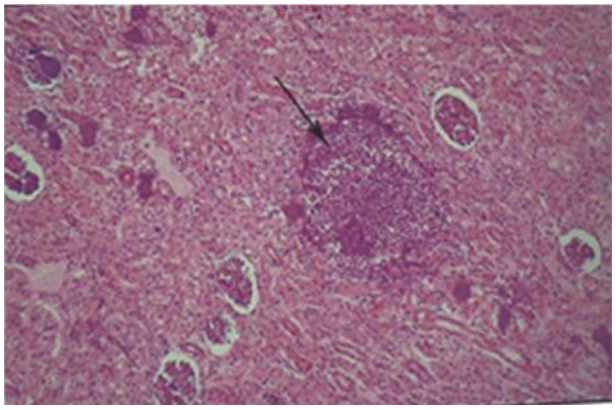

FIGURE -10 : Kidney tissue microscopically showed embolic nephritis with abscess formation adjacent glomeruli , also there is bacterial colonies in glomeruli and periglomerular tissue (H&E)X100

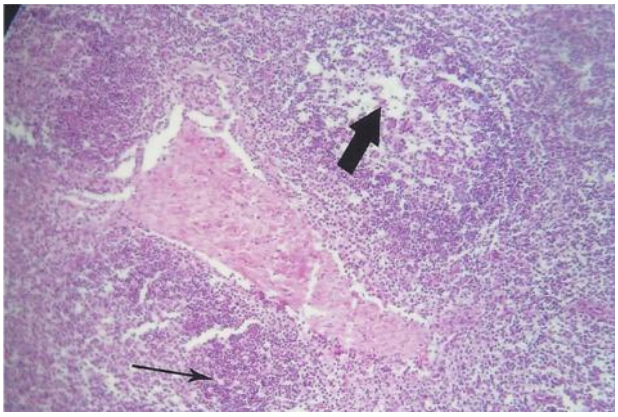

FIGURE 11 : Lymph node microscopically showed suppurative lymphadenitis together with localized microabscess in the lymphoid follicles (H&E)X100

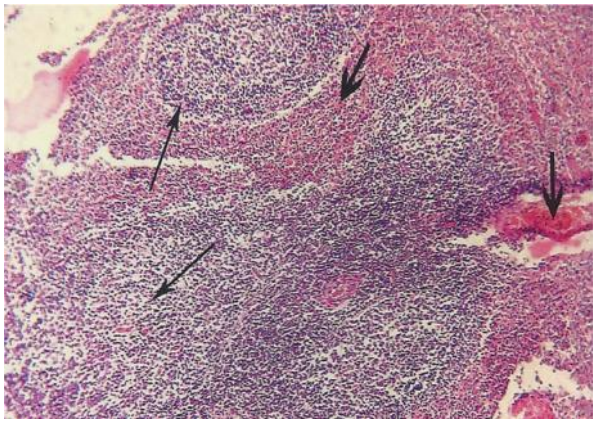

FIGURE 12: Spleen microscopically showed extensive reactive lymphoid hyperplasia together with localized microabscess in white pulp. (H&E)X100

The Kidneys: The lesions began as congestion of renal blood vessels, infiltration of neutrophils between the renal tubules and glomeruli lead to embolic nephritis (Fig – 10). Also bacterial colonies were seen in the neutrophils exudates in the renal tissue. At the advance stage atrophies of glomerular tuft and dilation of Bowman's space.

The Lymph Nodes: The lesions began as a reactive hyperplasia of lymphoid follicles and congestion of sub capsular and medullary sinuses and edema all over the lymph node tissue together with infiltration of neutrophils and macrophages lead to supportive and edematous lymphadenitis later on (Fig – 11).

The Spleen: The lesions began as congestion, reactive hyperplasia of white pulp and at the 12^{th} – 20^{th} days post infection there is abscessation inside white pulp and reticuloendothelial cells hyperplasia in red pulp together with hemorrhage (Fig – 12).

The Heart: The lesions began as congestion and hemorrhage between muscle fibers together with thrombus. Also clotted blood was seen in heart cavities during the experiment period.

All the pathological lesions were associated with extensive neutrophils infiltration occurred as a result of bacterial effects that lead to leukocytes recruitment mainly neutrophils and some lymphocytes and macrophages and these inflammatory cells induce parenchymal cell injury in the body organ [7,8,9 10]. Thrombus is mostly common in all the organs due to effect of endotoxins increase the adherence of platelets on the endothelial cells with the expression of adhesion molecules[11]. Neutrophils are the main inflammatory cells infiltrated the all over the organs, these inflammatory cells have powerful effects on production and release of cytokines and have also be shown to exert potent proinflammatory and anti-

inflammatory effects of animal model of inflammatory disease[12]. Lipopolysaccharide (endotoxins) of *P. multocida* have a role in endothelial damage of blood vessels and increase their permeability and activate the alternative pathway of the complement as well as to increase chemotactic activity of all the inflammatory cells into the organs which more evident in this experiment. The degree of neutrophils activation increased with the degree of tissues injury occurred as a result of effect of bacterial endotoxins [12] which lead to endothelial damage so hemorrhage and fibrin together with edema infiltrated in all the organs, other lesion, focal abscessations occurred in all organs later on as a result of tissue injury caused by bacterial endotoxins and bacterial proliferation, since necrosis and neutrophils infiltration lead to abscess formation in the lungs, liver, spleen as well as cholanitis which more observed in this study, similar finding reported by[13,14]. Necrosis of the tissue in all examined organs was occurred also as a result of ischemia due to partial obstruction of blood vessels at inflammatory areas in all the organs[15]. Emphysema in the lungs tissue occurred as a result of neutrophils infiltration caused damage of alveoli and developed in to emphysema which is observed in all pulmonary tissues in this study similar to [16]finding. Streaming macrophages were seen in all pulmonary tissue, this feature considered a pathognomonic findings for pneumonic pasteurellosis[17]. Peribronchial lymphoid tissue hyperplasia in lungs, lymphoid hyperplasia of cortical resion in lymph node and in white pulp of the spleen occurred as a result of persistence stimulation of these lymphoid tissues by lipopolysaccharide and outer membranes protein of *P. multocida* [18,19]. Other lesions were observed in this study dilation of Bowman's space, shrinkage of glomeruli, whereas, other glomeruli showed increase cellularity, these lesions were related to immune complex deposition (*P. multocida* endotoxins and antibodies)[14] which more evident in this study together with the embolic nephritis which occurred as a result of hematogenous route of dissemination of *P. multocida* so reach into glumeruli and infect periglomerular interstitial tissue so localized abscesses seen between glomeruli and renal tublules. The abscesses and necrosis occurred in all the organs as a result of several mechanisms of tissue injuries such as reactive oxygen species, lytic enzymes, inflammatory cytokines and modulators of hemostasis [20, 21].

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