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Histological Analysis of Kidney After Experimental Infection with *Klebsiella Pneumonia* in Rats

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Abstract

This study aimed to conduct a histological analysis of the kidney after experimental infection with *klebsiella pneumoniae*. *K. pneumonia* isolates obtained from master's students at Baghdad University in Iraq. Twenty rats were utilized as test subjects in this experiment. Their weight varied from 160 to 220 g, and their ages were between 2 and 2.5 months. Two groups of rats were used: Group A (10 rats) were given normal saline suspension as the control treatment. Group B: 10 rats underwent infection with 0.2 cc of *K. pneumonia* suspension orally. Animals were slaughtered, their kidney tissues were stored in 10% formalin for histopathological preparation after 7 and 14 days of infection, and the prepared slides were stained by hematoxylin and eosin. Kidney regions had substantial acute inflammatory alterations shortly after an infection, including dilated and blood-filled B.Vs, the presence of fibrin, edematous fluid in the interstitial tissues, neutrophilic infiltration, and liquefactive necrosis after seven days. At 14 days post-infection, significant histopathologic abnormalities included less vascular congestion, fibrinous exudate with PMN infiltration, and a lack of lymphocytes. In conclusion, *K. pneumoniae* can cause different pathological lesions in the kidneys of rats, such as necrosis and congestion of blood vessels as well as glomerulonephritis.

Key words: Histology, Kidney, *K. pneumoniae*

Introduction

Klebsiella pneumoniae is a gram-negative, immotile, rod-shaped, facultatively anaerobic, lactosefermenting bacterium with a distinctive polysaccharide capsule. Because of *K. pneumonia's* evolving ability to resist the effects of multiple drugs (Gerald et al. 2012), certain strains have been associated with episodes of bloody diarrhea that are difficult to cure today. Lipopolysaccharides, capsular polysaccharides, type 1 and type 3 fimbriae, and siderophores are the primary virulence

components of K. pneumonia that play critical roles in pathogenesis (Podschun et al. 2001). Klebsiella's pathogenicity could be explained by the bacteria's ability to produce a heat-stable enterotoxin. *K.pneumoniae* has lately received worldwide attention as a significant cause of nosocomial infections such as pneumonia, bacteremia, UTIs, and wound infections (Tawfick et al.2016). *K. Pneumonia* is characterized by an increased inflammatory response, significant lung damage, and elevated levels of pro-inflammatory cytokines; the etiological agent is Streptococcus pneumoniae (Zhang et al. 2000). Localized inflammation prevents the spread of the pathogen in such infections, and persistent hyperinflammation is often accompanied by mortality and chronic inflammatory diseases (Medzhitov 2008). *Klebsiella pneumoniae* isolated from a case of bloody diarrhea can bind to cytoskeletal proteins, whereas other researchers discovered that Henrietta lacked cells, such as actin, which collects at the bacterium-host interaction region (Guerin et al. 1998).

Antibiotic resistance in G-bacteria is linked to higher mortality, longer hospital stays, and higher medical costs (Schwaber et al.2006). *Klebsiella pneumoniae* and other species may colonize sterile wounds and urine and appear as normal flora in various organs, including the biliary tract, gut, and colon, in addition to inhabiting the human pharynx, digestive system, and skin (Abdallah et al.2014). K. Klebsiella pneumoniae is a common nosocomial pathogen that causes blood infections (sepsis) and biofilms that make antibiotics less effective. As a result, it can make people and animals sick with a wide range of diseases. As a result, researchers have concentrated on developing antigens and vaccines for *K. pneumoniae* (Unkel et al., 2012). This study aimed to perform a histological analysis of the kidney after an experimental infection with Klebsiella pneumoniae.

Materials and Methods

Isolates of K. pneumonia were obtained from master's students at the University of Baghdad, Iraq. Twenty rats were utilized as test subjects in this experiment. Their weight varied from 160 to 220 g, and their ages were between 2 and 2.5 months. Two groups of rats were used: Group A (10 rats) were given standard saline suspension as the control treatment. Group B: 10 rats underwent infection with 0.2 cc of *K. pneumonia* suspension orally. After 7 and 14 days of infection, the animals were slaughtered, and their kidney tissues were stored in 10% formalin for histopathological preparation. The prepared slides were stained with hematoxylin and eosin.

Results and Discussion

Kidney regions had substantial acute inflammatory alterations shortly after an infection, including dilated and blood-filled B.Vs, fibrin, edematous fluid in the interstitial tissues, and neutrophilic infiltration (Figure: 1), and liquefactive necrosis after seven days (Figure 2).

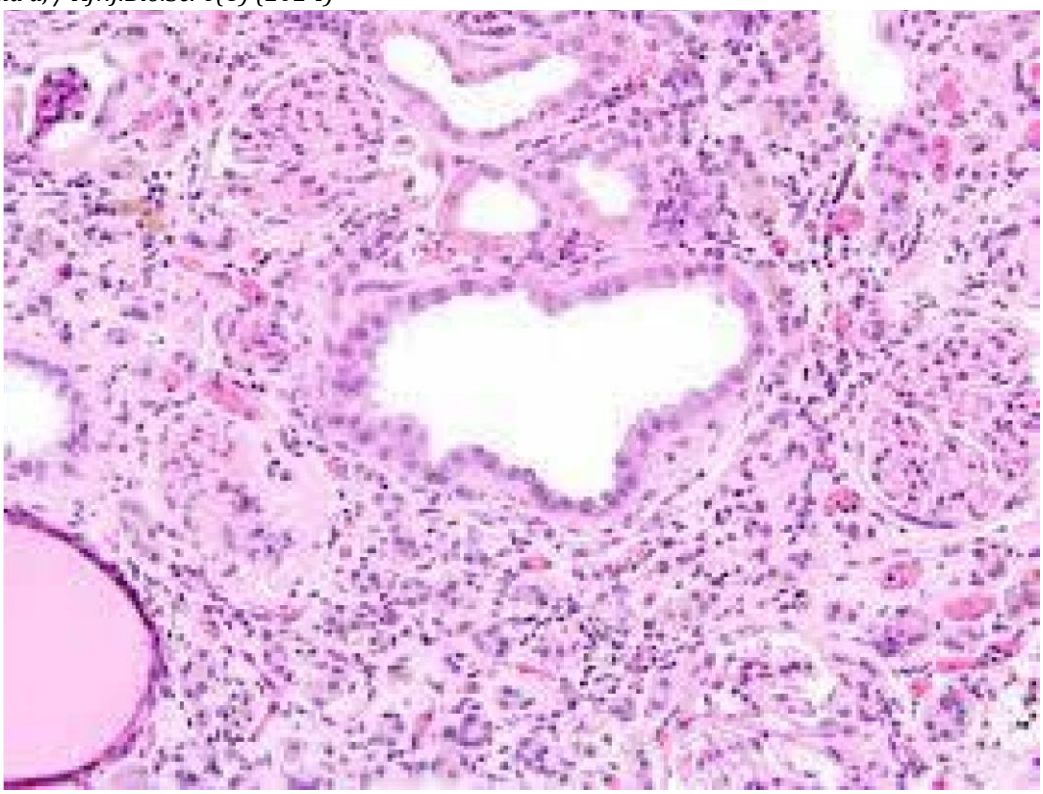

Figure 1. The kidney of the infected group shows inflammatory changes, including dilated and blood-filled B. Vs, fibrin, edematous fluid in the interstitial tissues, and neutrophilic infiltration. H&E, 400x.

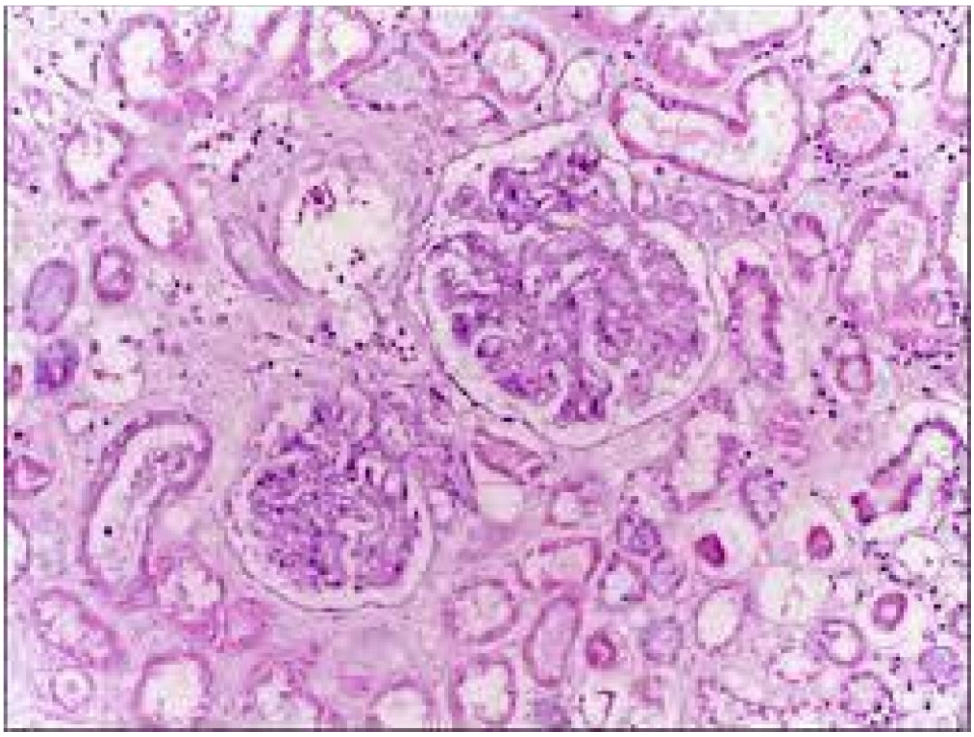

Figure 2. The kidney of the infected group shows liquefactive necrosis H&E, 400x.

At 14 days after the infection, significant histopathological changes included less vascular congestion, fibrinous exudate with PMN infiltration, and a lack of lymphocytes (Figure 3).

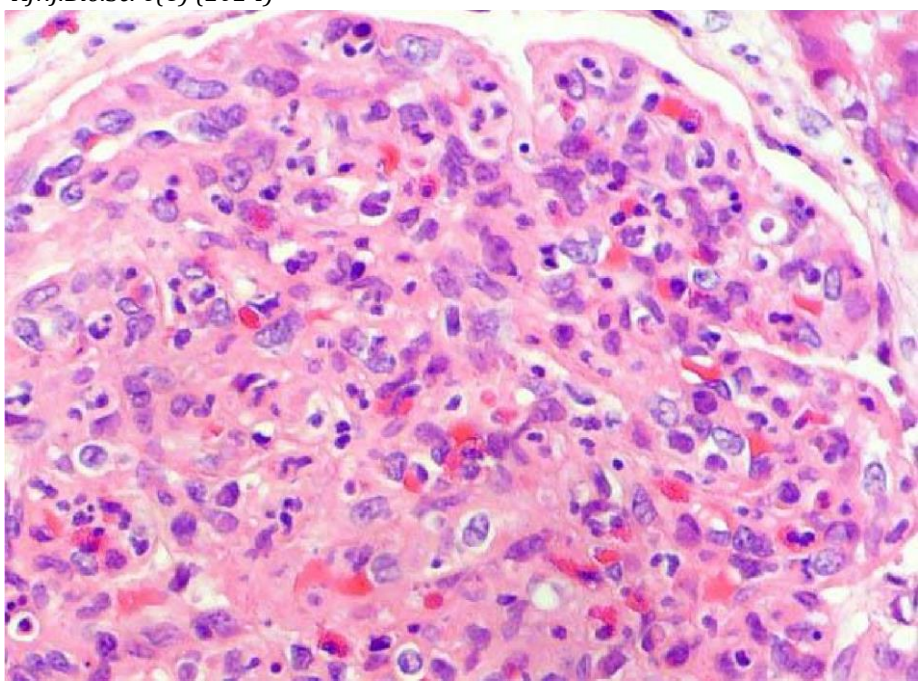

Figure 3. The kidney of an infected group shows glomerulonephritis H&E, 400x.

It was noted that the glomeruli were affected at the site of the lesion of the peritoneal cyst and pelvic peritoneum, either by thinning their walls due to the weakening of the interstitial tissue surrounding them or by their atrophy and eventual disappearance. Parietal of Bomana's capsule and calcium deposition around intimate outgrowths in quantity, which can be attributed to the difference in the type of bacteria that caused the infection in the cyst and pelvis as the length of the infection period (Glassock et al. 1974).

The pathological changes in the ureters and bladder were less severe, even if they increased in the advanced stages of the experiment with severe infiltration Lymphocytes with few polymorphous cells, severe epithelium shedding, and persistence of cellular infiltrates even after the inflammatory lesion has healed (Gillies and Dodds 1976). According to biochemical tests, the bacteria K. pneumoniae, a weak product of yeast urease (Sanford et al. 1962), causes the formation of any stone in the urinary system, as opposed to the bacteria Proteus, its strong product. According to (Abadullah and Zghair, 2016), kidney disease caused by *K. pneumoniae* infection is characterized by a buildup of blood in the blood vessels, bleeding, inflammatory cells in the interstitial tissue an increase in the size of the glomerular space. The histological alterations of the kidneys revealed varying degrees of inflammation within and between the infected group, particularly in the kidney specimens that demonstrated pyelonephritis in rats (Ibrahim 2008).

Conclusion

K. pneumoniae can cause different pathological lesions in the kidneys of rats, such as necrosis and congestion of blood vessels, as well as glomerulonephritis.

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Declarations:

The author declares the university of Fallujah, College of Veterinary Medicine.

Conflict of interest

The authors declare that they have no competing interest.

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