Biochemical and Histopathological Investigation of *Pseudomonas Aeruginosa* Infection on The Spleen, Kidneys and Liver of Rabbit *Oryctolagus Cuniculus*

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Abstract
For the purpose determine the association between biochemical indicators and severity of histopathological lesions, this study used 20 rabbits, 10 in each of the T1 control and T2 groups, to isolate, identify, and investigate the effects of *Pseudomonas aeruginosa* illness on the spleen, kidneys, and liver of rabbits. *P. aeruginosa* isolation and identification from an infected rabbit. A thin fibrous capsule, a red pulp, and white pulp were visible in the microscopic sections of the normal group's spleen, which normally lacks the organized nodules seen in mammals. In contrast, the infected group's splenic sinuses showed significant dilatation and congestion of the red pulp with a mild hemorrhagic concentration, Melano macrophage accumulation, bleeding, lymphocytosis, an and proliferation of white pulp follicles, which are indicators of oxidative damage and an active immune system response. Vacuolation, necrosis, and sloughing of the renal tubules' epithelial cells were signs of severe cellular degeneration, and the primary lesion in the kidney was thrombus in the blood vessels accompanying inflammatory cells. In the normal group, the liver is enveloped by a thin capsule alongside simple squamous epithelium and is composed of polyhedral hepatocytes with sinusoids and a central vein, while in group T2 showed hepatocyte granular and degeneration, capillary endothelium activation, and Kupffer cells, Vascular edema, hemorrhage, degeneration, congested fibrosis, and inflammation of hepatocytes, a sinusoid contraction, and bile duct hyperplasia defined the histopathological changes. Spleen, liver, and kidney function measures were all altered in the experimental rabbits. Urea levels were markedly elevated, while rabbits inoculated with *P. aeruginosa* had elevated levels of the uric acid, creatinine levels, alanine aminotransferase, and aspartate aminotransferase. In conclusion, *Pseudomonas aeruginosa*-induced infections in experimental rabbits showed histopathologic lesions and abnormalities in the liver, kidneys, and spleen. Bacterial infections are one of the main issues that laboratory animals face. Due to the dangers and inefficiency of drugs, an effective treatment without negative side effects is needed for these common illnesses.

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Introduction

Numerous bacterial, fungal, parasitic, and viral agents can be found in laboratory rats, mice, and rabbits. These organisms often do not exhibit obvious symptoms of illness [1]. Nevertheless, a lot of these lab animals' natural infections have the potential to change the physiology of the host, making it unsuitable for a variety of experimental applications. Even though the quantity and frequency of these diseases have significantly decreased, many still occur in lab animals and serve as undesirable factors in

studies [2]. The rabbit is a widely dispersed animal species that is frequently employed economically and in laboratories. It serves as a model for many clinical trials and is widely utilized in education, particularly in disciplines where laboratory procedures are carried out [3, 4]. *Pseudomonas aeruginosa* is a significant Gram-negative opportunistic bacterium that produces a high morbidity and mortality rate of up to 40% in both acute and chronic infections. P. aeruginosa is a particularly difficult infection because of its strong intrinsic and developed resistance to several of the antibiotics now in use [5-8]. They are widespread in nature, found in soil, water, and vegetation, and they are often connected to animals. The bacterium can survive in infectious processes while other more vulnerable organisms have been eradicated by treatment due to its relative drug resistance and tolerance to a wide range of antibiotics [9-12]. *Pseudomonas aeruginosa* infections pose a serious threat to medical professionals because of their high virulence, resistance to antibiotics, and capacity to induce systemic infections [13]. This bacterium has the ability to infiltrate several organs, causing serious structural and metabolic changes that could impair organ function. Improving therapeutic approaches, illness management, and diagnostic indicators all depend on an understanding of the degree of organ harm and the corresponding biochemical alterations [14-17]. Thus, a thorough analysis of the clinical and biochemical effects of P. aeruginosa infections in rabbits is required. In order to provide information that may help with early detection, prevention, and therapy of P. aeruginosa infections, this study will examine the impact of Pseudomonas aeruginosa an infection on specific organs of *Oryctolagus cuniculus* by evaluating changes in serum biochemical parameters and identifying structural and changes in cells in spleen, kidneys, and liver tissues.

Materials and method

Ethical approval: Regulations pertaining to animal welfare were followed in every procedure. From August to December 2025, the Department of Dialysis Technology at Al-Furat Al-Awsat University-Al-Samawah Technical Institute used the animals' organs for study after they were killed in compliance with ethical and animal care standards.

Animals: Twenty rabbits weighing between 1.5 and 2.0 kg were used in this experiment; they were acquired from the sciences collage farm in Al-Samawah university. Ten rabbits each were split into two groups: the T1 control group and the T2 group, which received an intraperitoneal inoculation of isolated Pseudomonas aeruginosa. Experimental animals were kept on a 12-hour light/dark cycle in air-conditioned rooms with temperatures 20 degrees Celsius and relative humidity between 60 and 65 percent. Morphological, cultural, and biochemical assays were used for isolation and diagnosis, each bacterial species was cultivated on nutrient agar for 24 hours at 37ºC to create some of the bacterial inoculums. Five to seven colonies were then transferred to a tube containing five milliliters of sterile normal saline. To create a bacterial suspension having turbidity equal to 0.5 percent McFarland's standard solution, or 5×108 colony-forming units (CFU/mL), the tubes were vortexed. Sterilized syringes were used to gently inject 2 milliliters of a bacterial suspension from each sample intraperitoneally into each experimental animal. Prior to the study, each rabbit was examined bacteriologically by creating a face culture, and rabbits free of *P. aeruginosa* were employed. The animals were kept in metallic cages. P. aeruginosa was added to Trypticase soy broth (Oxoid) and incubated for 24 hours at 37°C. Using PBS, various broth dilutions were produced to yield (6.5×105CFU/ml) using the conventional procedure outlined in (6,14). MacConkey agar was used to diagnose the bacteria, which are isolated using a variety of culture media and colony shapes. Media was incubated at 35 degrees for 24 to 48 hours (7,15). Experts in animal handling kept an eye out for any clinical indications of disease in rabbits. Rabbits were put to death by an intravenous injection on 0.4 ml per kg of body weight. 50 milligrams of mebezonium iodide and 5 mg/ml of tetracaine hydrochloride. The animals were killed in order to obtain new blood specimens; the serum was separated by centrifugation above 3000 rpm to biochemical analysis and liver and kidney function assessment. Human Diagnostic Tests (Human GmbH, Wiesbaden, Germany) provided diagnostic kits including serum ALT, AST, uric acid, urea and creatinine, which were used in accordance with the manufacturer's instructions. Histopathological Techniques Tissue specimens from the experimental rabbits' liver, kidneys, and spleen were immediately fixed in a formalin solution containing 10% dehydrated in increasing ethyl alcohol concentrations, cleared in xylene, then blocked in paraffin, and sectioned as 5µm using a rotary microtome. Hematoxylin and eosin used to stain the obtained tissue slides [18].

Results and discussion

The microscopic sections of the spleen in groupT1 revealed no obvious pathological findings, with the capsule, red pulp, splenic sinuses, and white pulp all appearing normal. In contrast, group T2's spleen sections revealed dilation and congestion of the splenic sinuses with a mild hemorrhagic focus, Melano macrophage aggregation, especially in the red pulp, and congestion, within red pulp. Indications of oxidative stress with an active immunological response include bleeding, lymphocytes, and the development of white pulp vesicles (Fig. 1A-F). A thin capsule, red pulp supporting blood filtering, and white pulp for immunological responses make up the spleen, an essential hematopoietic and immune organ that usually lacks the ordered nodules present in mammals [19]. Numerous ellipsoids (specialized wrapped capillaries) and Melano macrophage centers (aggregates of pigment-filled macrophages) are important characteristics (3). In addition to progressive lesions and cellular reduction within the lymphoid tissue, microscopic analysis of the spleen showed a decrease in white pulp, with red pulp significantly outnumbering white pulp (Fig. 1A-F). Immunosuppression and elevated immune demand were indicated by lymphoid loss and congestion in the spleen. These alterations show the body's attempt to fight infection and also potential immune cell depletion because the spleen is crucial to immunological responses [20, 17] refers to the spleen is commonly regarded as an organ of storage that maintains the blood cells that contributes to immunological responses, while it may have a number of other functions. The breakdown of irregular erythrocytes and spleen's hyperfunction in response to strong erythropoiesis are most likely the causes of these alterations. The spleen, a well-known peripheral lymphoid organ, is essential for immune complex removal and defense against infection. Additionally, it triggers adaptive immune responses [21]. Furthermore, it functions as a specific circulatory system filter that promotes lymphocyte growth in humoral and cellular immunity [22, 23].

The rabbit kidney in group T1 had a recognizable cortex and medulla, as well as a thin fibrous capsule. Renal pyramid, collecting duct, as well as loops of Henle are found in the inner, lighter medulla, while kidneys corpuscles and convoluted tubules are found in the outer, darker cortex. The structure of the nephron consists of proximal tubules containing brush border and a glomerular tuft with podocytes. in group T2; showed the vacuolation, necrosis, and sloughing of the renal tubules' epithelial lining cells with cellular debris within the lumen were signs of severe cellular degeneration. The primary lesion in the kidney was a thrombus forming in the blood vessels alongside inflammatory cells, primarily neutrophils, appearing in the blood vessel lumen (Fig. 2A-D), as results of [24]. These results imply that group T2's kidneys suffered severe damage, which most likely led to impaired filtration and reabsorption functions. Tubular necrosis and thrombosis together suggest that kidney impair is caused by ischemia and inflammatory mechanisms.

In the normal group, the liver is enveloped by a thin capsule alongside simple squamous epithelium and is composed of polyhedral hepatocytes alongside symmetrical sinusoids and a central vein. Histological examination of the liver in group T2 showed hepatocyte granular and seldom vacuolar degeneration, capillary endothelium activation, and Kupffer cells, Vascular edema, hemorrhage, degeneration, congested tissue fibrosis, edema and inflammation of hepatocytes, a sinusoid contraction, and bile duct hyperplasia defined the histopathological changes (Fig. 3 A-D). [25] mention edema around bile ducts, mononuclear cell perivascular infiltration, bile duct growth, and minor vascular congestion, whereas the liver showed hepatocyte coagulative necrosis with inflammatory cells, primarily neutrophils and macrophages, in the necrotic area. Large-scale bile duct hyperplasia was the principal cause of the lesions found in liver tissue of infected rabbits, which were mostly limited to the liver and bile ducts. Similar to adenomatous hyperplasia, the bile ducts were noticeably enlarged and bordered with hyperplastic epithelial cells that were thrown into many arborizing papillary fronds reaching into the ductal lumina. Additional slices showed bile ducts were extremely dilated, with flattened epithelium that protruded little or not at all to the lumen, which is packed with many thin walls. A substantial quantity of connective tissue packed with mononuclear cells encircled the hyperplastic bile ducts [26, 27]. Furthermore, Biliary epithelial cells that had been shed filled the ducts. In as well as vacuolar degeneration and hepatocyte death, the hepatic parenchyma displayed dilatation, congestion, and rupture of the endothelium lining the sinusoids and central veins. Mononuclear cells penetrated the liver parenchyma and portal sections, and there was also significant bleeding in the necrotic area. In other regions, the parenchyma was replaced by fibrous connective tissue that was invaded by mononuclear cells and had a propensity to generate new bile ductulus. This observation aligns with the previously published findings [28, 29].

The preference and growth of aeruginosa inside the epithelium may be the cause of bile duct epithelium's proliferation. The widespread sinus dilatation, which is linked to fibrosis around cords, may be caused by the hepatic blood flow being blocked, particularly in the portal veins, by the bile ducts' massive proliferation and dilatation. Additionally, hepatocyte cellular degradation and cord atrophy would ensue from the blood flow standstill.

These histopathological findings concur with those reported by others [8, 30, 7, 31] described granulomatous hepatitis during aeruginosa infection, which causes a significant portion of the hepatic lobules to be destroyed and fibrotic. Massive parenchymal necrosis was the cause of the fibrosis seen in the hepatic parenchyma. bile pigment deposit resulted from local edema obstructing the main ductal system. The hepatocytes had regions of the fibrosis and mononuclear cell aggregates along with degenerative alterations to necrosis [32].

Pseudomonas aeruginosa infections in rabbits have been shown to cause considerable hepatotoxicity, which is characterized by elevated liver enzymes, inflammatory damage to tissues, and metabolic changes. This is especially true in the laboratory models of septicemia. Severe liver lesions, such as necrosis, bleeding, and the development of microabscesses, are frequently caused by the infection. Histogram (1) showed Serum biochemical parameters were significantly altered in rabbits infected with *Pseudomonas aeruginosa* as when compared to the control group. Hepatocellular damage is indicated by a marked increase in the ALT and AST values, Increased serum levels of these enzymes, which are typically found in hepatocytes, indicate that the liver cells have been damaged and are leaking into the circulation. Additionally, cholestasis or biliary dysfunction may be linked to the noted rise in alkaline phosphatase levels. Increased ALP is frequently associated with reduced bile flow or biliary epithelial damage, which can be a consequence of inflammation or infection, this similar to results of [33, 34]. Overall, our biochemical results indicate that *Pseudomonas aeruginosa* infection causes severe hepatic stress, which may result in biliary dysfunction as well as hepatocellular damage. This lends credence to the idea because an infection has pathological effects outside of the initial infection site and these the elevated in the levels may indicate biliary dysfunction.

Hepatocellular injury is indicated by the substantial increase in ALT and AST concentrations seen in infected rabbits. After liver cell damage, those enzymes leak into the bloodstream. Additional evidence of liver injury comes from histopathological findings such necrosis and inflammatory infiltration (Histogram 1). This could be explained by systemic inflammatory reactions brought on by *P. aeruginosa* and bacterial toxins. elevated amounts of creatinine and urea in the blood, which indicate compromised renal function. reduction in albumin and total protein levels, possibly as a result of enhanced protein catabolism or liver disease. After receiving an intraperitoneal injection of Pseudomonas for two weeks, the level of urea increased more significantly. Reduced renal function is suggested by elevated urea and creatinine values [35]. These biochemical findings are corroborated by histological observations, such as tubular necrosis as well as glomerular injury. The dispersion of germs through the bloodstream as well as ensuing colonization of the kidney, resulting in tissue degradation, could be the cause of renal impairment [2].

Elevated Liver Function Enzymes: Hepatocellular injury is indicated by a significant increase in blood liver enzymes during experimental infection, such as Alanine Aminotransferase (ALT), and Aspartate Aminotransferase (AST). Histopathological alterations such as central venous congestion, hepatocyte vacuolar degeneration,

and mononuclear cell infiltration (lymphocytes, macrophages) accompany biochemical damage (Histogram 1), this agrees with (9). Histopathological facts provide significant support for the biochemical results. Central venous congestion is indicative of reduced hepatic flow of blood and potential liver circulation problems. Hepatocyte vacuolar degeneration also indicates cellular damage, which is frequently linked to metabolic problems and intracellular fluid buildup. Moreover, a continuous immunological response to infection is indicated by the invasion of mononuclear cells, such as macrophages and lymphocytes. By releasing cytokines, this inflammatory response exacerbates hepatic injury and causes tissue damage. Overall, the presence of both histological changes and increased liver enzymes indicates experimental infection causes substantial hepatic damage. A complex mechanism of liver damage involving mainly direct pathogenic effect and host immunological responses is highlighted by the combination of vascular abnormalities, cellular degradation, and inflammatory infiltration [18, 30].

Immunization Effects: It has been demonstrated that immunization by *P. aeruginosa* entire sonicated antigen modulates these responses, leading to less severe liver damage and milder clinical symptoms when compared to immunized controls (Fig 1). Hospital-acquired as well as other medically related bacteremia cases are largely caused by *Pseudomonas aeruginosa* [3]. The decrease of liver damage indicates immune response is more prepared to identify and react to the virus, hence preventing its spread and minimizing tissue damage. Additionally, vaccinated individuals typically show less severe clinical symptoms, suggesting a general improvement in the course of the illness. Increased behavioral and cellular immune system responses, which aid in neutralizing bacterial toxins and more successfully controlling infection, may be responsible for this protective impact. A well-known opportunistic bacterium, *Pseudomonas aeruginosa* has a substantial contributor to nosocomial (hospital-acquired) infections, especially bacteremia. It is often linked to infections in people with impaired immune systems, protracted hospital stays, and invasive medical procedures. The need of creating efficient preventive measures, including immunization, is highlighted by the high frequency of P. aeruginosa-related bacterial infections in clinical settings. The bacterial activity as well as the documented histological abnormalities in the liver and kidneys may be the cause of the elevated urea, ALT, and AST levels (Fig 1). This was additionally verified in the past. Infection causes dehydration and decreased renal perfusion, which raises the level of urea. Alanine aminotransferase and aspartate aminotransferase may be caused by bacterial activity. Increased tissue breakdown throughout infection, decreased kidney excretion, elevated uric acid, reduced kidney filtration, sepsis, toxic consequences of infection, and severe infection leading to liver stress are all indicated. When systemic disease occurs, AST may increase more than ALT.

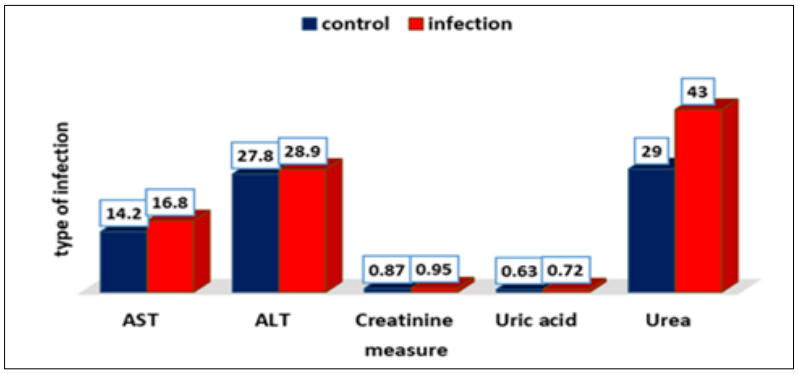
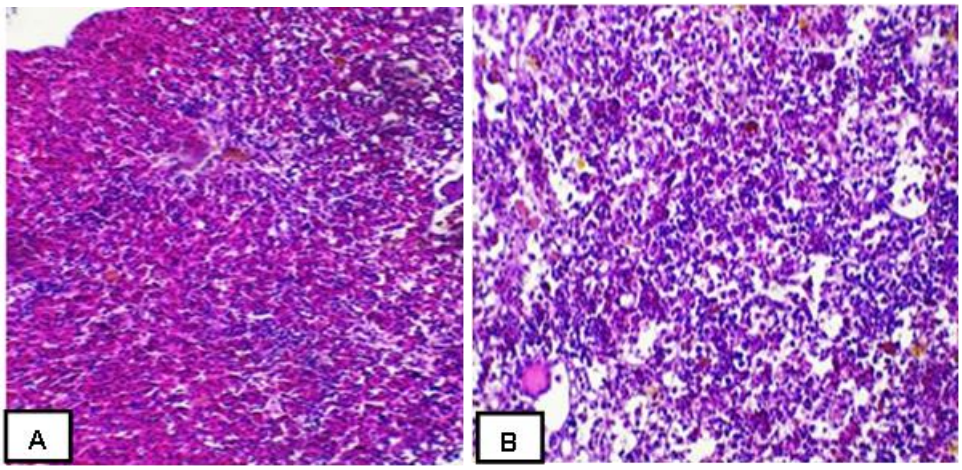


Fig 1: measurement of ALT and AST (U/L), urea, uric acid and creatinine mg/dL in rabbit



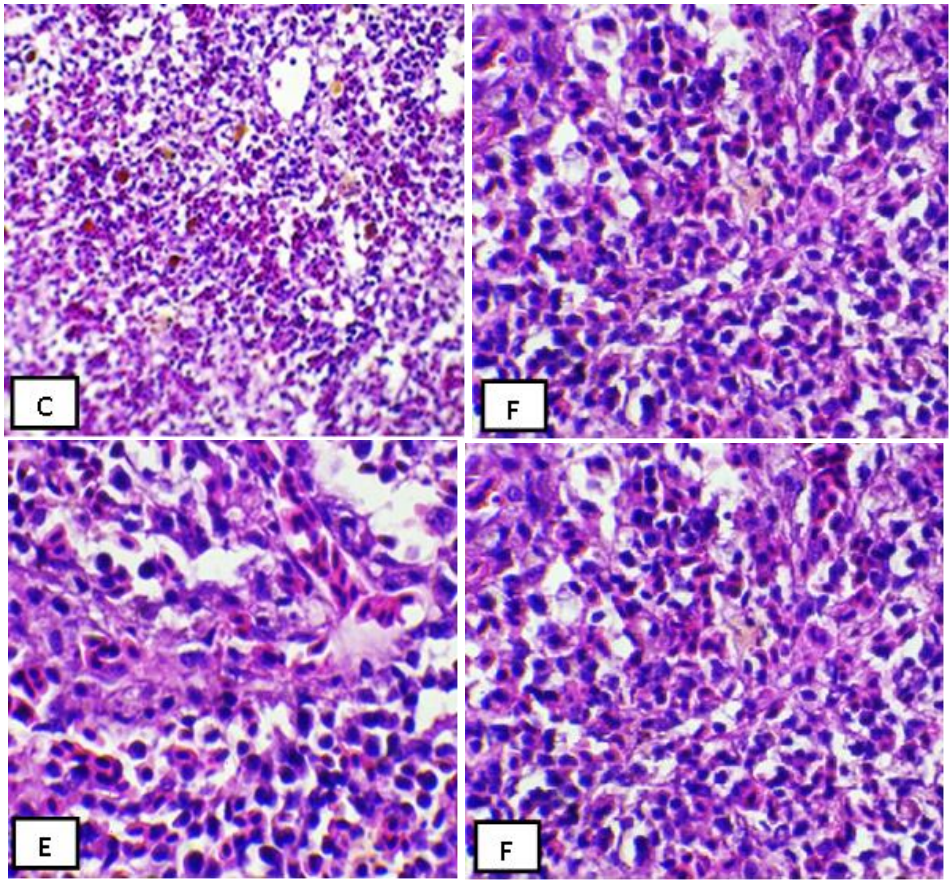


Fig 2: (A) Histological section of the spleen of rabbit showed the normal structure of capsule, white pulp and red pulp; H & E 400×. **(B).** dilation and congestion of the splenic sinuses with a mild hemorrhagic focus; H & E 400×.; **(C)** represents the cells hyperplasia and congestion, H & E stain, 400 ×. **(D)** nuclei contraction with necrosis of tubular cells demonstrates inflammation; H & E 400×.**(E)** Melano macrophage aggregation, especially in the red pulp, and congestion within red pulp; H & E 400×., **(F)** Indications of oxidative stress with an active immunological response include bleeding, lymphocytes, and the development of white pulp vesicles; H & E 400×.

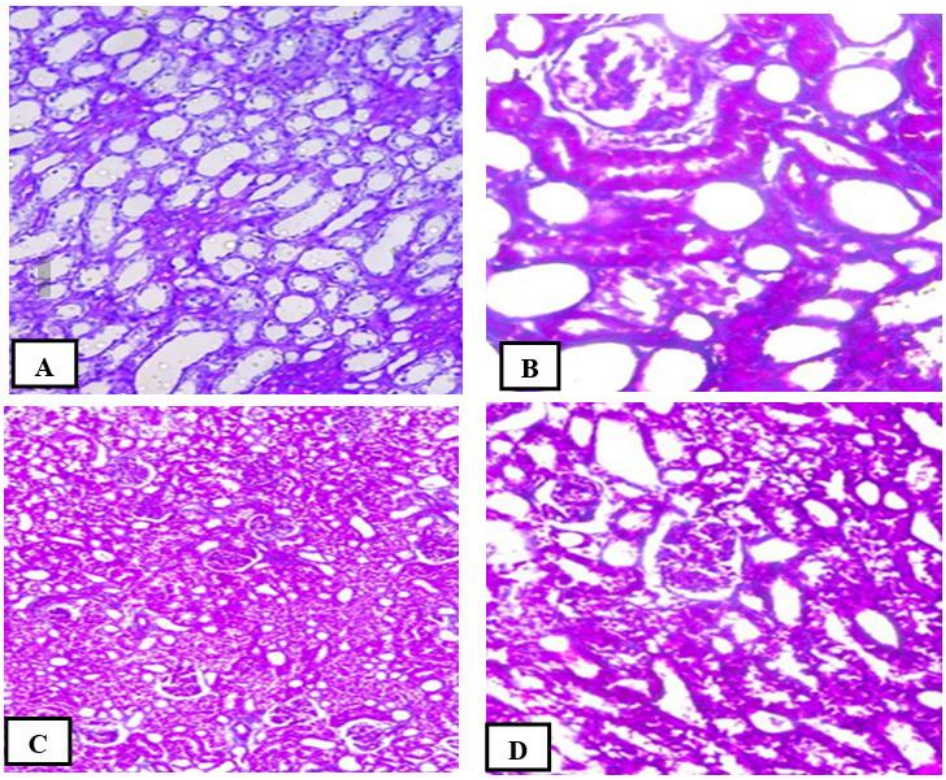


Fig 3: (A) Histological section of the kidney of rabbit showed the kidney had a recognizable cortex and medulla, thin fibrous capsule. Renal pyramid, and collecting duct; H & E 400×. **(B).** showed the vacuolation, necrosis, and sloughing of the renal tubules' epithelial lining cells; H & E 400×.; **(C)** Tubular necrosis and thrombosis; H & E stain, 400 ×. **(D)** severe damage and a thrombus forming in the blood vessels alongside, and primarily neutrophils, appearing in the blood vessel lumen. H & E 400×.

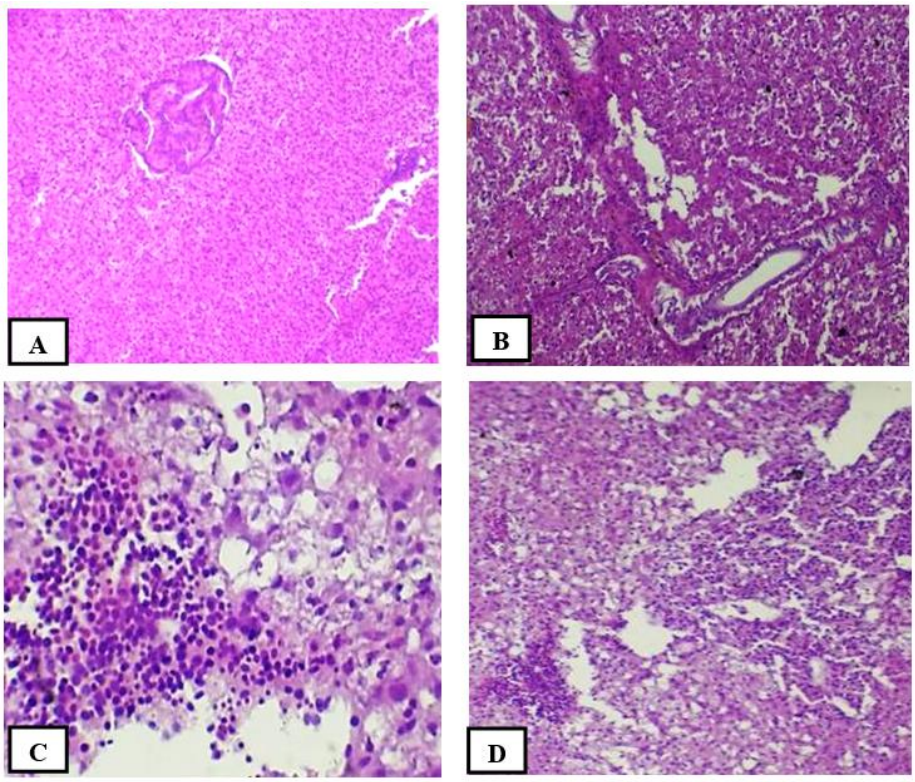


Fig 4: (A) Histological section of the liver of rabbit showed the liver is encompassed by a thin capsule alongside simple squamous epithelium and is composed of polyhedral hepatocytes with nsinusoids and a central vein; H & E 400×. **(B).** showed the Vacuolation, hemorrhage, extreme degeneration, edema and inflammation of the hepatocytes; H & E 400×.; **(C)** showed the sinusoid edema, and bile duct hyperplasia; H & E stain, 400 ×. **(D)** showed the necrosis, congestion, fibrosis of hepatocytes; H & E 400×.

Conclusion

During the two weeks of the experimental infection, rabbits acted physically normally, but biochemical and histological analysis revealed minor systemic disruptions. However, for the first four days following an intraperitoneal injection of germs, there were noticeable symptoms of illness. Experimental rabbits infected with *Pseudomonas aeruginosa* showed histopathologic lesions and abnormalities in their spleen, liver, and kidney functions. The histopathology results showed that the germs had entered the interior organs. Given these findings, recommend that future studies be conducted to develop diagnostic methods for identifying dangerous bacteria in Iraq, that would aid in the better management and prevention of aquaculture diseases. Bacterial infections are one of the main issues laboratory animals faces. Due to the dangers and inefficiency of drugs, an effective treatment without negative side effects is necessary for many common illnesses. Overall, the study demonstrates that a rabbit's liver, kidneys, and spleen suffer significant structural and functional damage as a result of P. aeruginosa. The biochemical and histological results together offer a thorough insight of the course of the disease and its consequences on individual organs. Hepatocellular necrosis and elevated liver enzymes were correlated. Increased kidney indicators were associated with glomerular and tubular impairment.

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Conflict of Interest

The writers of the essay assert which no conflicts of the interest surfaced while it was being drafted.

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