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A comparative study of treatment with ciprofloxacin and sodium propionate for non-O157 STEC (IQ1) infected mice

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Abstract

This study aimed to identify non-O157 STECs by taking fecal samples from infected humans and calves. A new non-O157 STEC type (IQ1) was isolated to infect mice experimentally. The study involved four groups: a control group, an infected group, an infected group treated with ciprofloxacin, and an infected group treated with sodium propionate. The study aimed to evaluate the effect of bacteria on the internal organs, specifically the liver, kidneys, and intestines, and to compare the effectiveness of different treatments. The affected group showed severe histopathological changes, while the organic acid-treated group showed a significant reversal of the changes in the kidneys and intestines and moderate liver changes. Mice were infected with a new Shiga toxin-producing *Escherichia coli* (STEC) strain and treated with organic acids or ciprofloxacin. Organic acid showed a better response than ciprofloxacin. Blood samples were taken, and the organs were examined for histopathological changes. The livers of the infected mice showed hepatocyte disorder, degeneration, thrombosis, and infiltration of mononuclear cells. The kidney had thickening of the renal capsule, glomeruli atrophy, and severe granulomatous inflammation. The intestines had thickening of the external muscular layer, loss of villi, and areas of necrosis. The sodium propionate group significantly changed villus length and mild hepatic alteration. The infected group showed histopathological changes in the liver, kidneys, and intestines, while those treated with antibiotics or organic acids showed minimal changes. The organic acid-treated group showed a significant reversal of histopathological changes in the kidneys and intestines, and the evolution of intestinal villi length was remarkable. The non-O157 STEC (IQ1) has histopathologic effects on the liver, kidney, and intestine and causes alteration in some blood parameters. The organic acid treatment showed faster and better results than the antibiotic treatment.

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Introduction

For the past decade, the non-O157 Shiga toxin-producing *E. coli* (STEC) predominance and due to the change in methodology of molecular-based assays, as well as to mandatory requirements to report non-O157 STEC cases had become evident in many countries (1-5). The non-O157 STEC infections have been widely reported (1-9); recently, about 96 534 O157 infections and 168 698 non-O157 infections have been estimated to occur each year (10). The increase in the incidence of non-O157 STEC noted by the Foodborne Diseases Active Surveillance Network in the years between 2000 and 2010 and from 0·12 to 0·95/100 000; in addition, during the previous ten years period, six serogroups were reported to cause about 83% of the non-O157 STEC infections; and the remainder of the

non-O157 STEC infections were associated with the other 65 serogroups (11), The liver, kidneys, and intestines suffered the most incredible infection-related damage. Histopathological analysis reveals that the liver eliminates toxins as one of its tasks. Yet, it is susceptible to pathological alterations in its blood pressure due to the presence of two separate blood vessels. The kidney changed the histological structure of Bowman's capsule and coagulative necrosis of the epithelial cells lining the renal tubules. Still, the extent of the damage depended on several factors, including the status of the nutrition of hepatic cells and the amount of toxins by bacteria. There was significant glomerular bleeding compared to the control group (12). Histopathological abnormalities in the kidneys suggest necrosis caused by endotoxin. The cytoplasm and nucleus of a cell undergo alterations that lead to acute tubular necrosis. Where the deviant behavior first appears, epithelial cells of the renal tubules are highly specialized cells, and even subtle shifts in their physical location can affect the function of the kidneys (13). Widespread inflammation, including inflammatory cell infiltration and proliferation in lamina propria and crypt abscesses, necrosis of epithelial cells lining the intestinal glands, sloughing, erosion, and total necrosis of the villi, were all observed in the gut (14). *Escherichia coli* potentially spreads disease through contaminated food. *Escherichia coli* has become a significant public health concern. Previous research has found that using PCR technology is a rapid and accurate test for *E. coli* (15). Meat, poultry, milk, and other animal products have been the subject of much research over the past few decades due to the prevalence of *E. coli* (16,17). Pathogenicity in *E. coli* may be acquired in several ways, including the dissemination of virulence factors or antibiotic-resistant genes; the latter poses a greater risk of spreading antibiotic resistance to other strains of *E. coli* (15). Essential oils found in organic acids can be defined as volatile oils derived from different plants and have anti-microbial, antiviral, and antifungal properties, immunomodulatory action, hypolipidemic effect, digestive stimulation effect, and property to alleviate heat stress (18). These essential oils are recently being used in animals because the digestion process and performance have improved. This may occur due to the positive modulation of gastrointestinal microbiota (19). Organic acids also found in organic acids are another alternative that scientists have attracted because of their antibacterial effect against different pathogenic microorganisms. The interlinked phenomena behind these compounds' synergism may be the positive modulation of gastrointestinal microflora. The hydrophobic nature of essential oils makes the bacterial cell membrane more permeable, resulting in an increased influx of organic acids into the cellular cytoplasm. Hence, the organic acids in their un-dissociated form make cellular pH more acidic, resulting in hampered bacteria's cellular metabolism (20).

Study the hematological, biochemical, and histological changes of mice infected by the isolated non-O157 STEC Comparison between the best antibiotic with organic acids for treatment.

Materials and methods

Samples

Non-O157 STECs were isolated and identified by taking fecal samples from humans and calves that showed clinical signs of infection with these bacteria. The results showed that a new non-O157 STEC type (IQ1) was registered according to (GenBank: LC738862.1).

Experimental design.

Twenty-eight mice were used and divided into four groups (control, infected, infected, and treated with an antibiotic, infected and treated with organic acids, the mice were given the effective dose of the bacteria (1x108 C.F.U/ml) according to Camundongos (21). The mice were started with an adequate amount. Clinical signs appeared during the first week after infection, such as nausea, fever, dehydration, diarrhea, sometimes bloody diarrhea, abdominal cramps, tiredness, fatigue, discomfort, and colic. After the appearance of clinical signs, start the treatment with Ciprofloxacin (22) and sodium propionate. During the second week after the infection, blood samples were taken to measure ALT, AST, gamma-glutamyl transferase (GGT), creatinine, and urea levels. The experimental mice were euthanized, and the internal organ (liver, kidney, intestine) for histopathological study.

Histological section

The histological sections for all groups were fixed by the paraffin and stained with hematoxylin and eosin stains (H&E staining) to detect the histopathological changes due to the infection under the light microscope (23).

Ethics approval

The current work has never been published in any language, nor is it being considered for publication in a similar or identical form by any other peer-reviewed journal. The animal experiments were carried out with the approval of the Ethical Committee of the College of Veterinary Medicine (UOK. VET. HE. 2021. 0.43).

Results

Blood test

The blood results showed that ALT and AST values were raised in infected groups as well as the urea and creatinine, while the GGT (Gamma-glutamyl transpeptidase) recorded variable results and the values decreased in the infected group, which was treated with an antibiotic (ciprofloxacin) while the lowest values recorded in the infected group that treated with sodium propionate (Table 1).

Table 1: Values of some biochemical parameters of study groups

Group	Urea	Creatinine	ALT	AST	GGT
Normal	28.40±1.14 ^{c}	0.28±0.06 ^{b}	64.80±2.77 ^{c}	519.40±18.58 ^{b}	4.00±0.01 ^{a}
Infected	39.60±1.51 ^{a}	0.44±0.08 ^{a}	84.80±8.04 ^{a}	809.80±17.94 ^{a}	2.80±0.45 ^{b}
Infected and treated with ciprofloxacin	36.20±1.09 ^{b}	0.29±0.01 ^{b}	76.20±3.70 ^{ab}	420.40±16.13 ^{c}	1.20±0.44 ^{c}
Infected and treated with organic acid	29.80±1.09 ^{c}	0.26±0.01 ^{b}	73.80±2.16 ^{b}	307.80±11.14 ^{d}	2.40±0.89 ^{b}

Different letters in the same column represent a significant difference at ($P<0.05$).

Liver histopathology

In control group the liver shows normal hepatocytes, hepatic architecture, usual central vein, and granular cytoplasm (Figures 1 and 2). In infected group; post-infection, the infected group with non-O157 STEC for several days showed histopathological changes; the hepatocytes were disorganized, degenerated, and had remarkable necrosis, alteration, and massive congestion of the central vein (thrombosis), sinusoids, infiltration of inflammatory cells (mononuclear cells), and severe interstitial inflammation (Figures 3 and 4). While in infected treated by an antibiotic (ciprofloxacin), the liver showed mild reversible changes of hepatic architecture, slight congestion of the central vein, with significant and severe hepatocyte degeneration, mononuclear inflammatory cell infiltration around the congested central vein, and mild interstitial infiltration of inflammatory cells (Figures 5 and 6). In infected treated by sodium propionate, the liver section for organic acid-treated mice after *E. coli* infection showed mild hepatic alterations, normal central vein, and sinusoids, significant hepatocytes organized cords with mild degeneration, slight mononuclear inflammatory cells infiltration, and apparent bi nucleated hepatocytes (Figures 7 and 8).

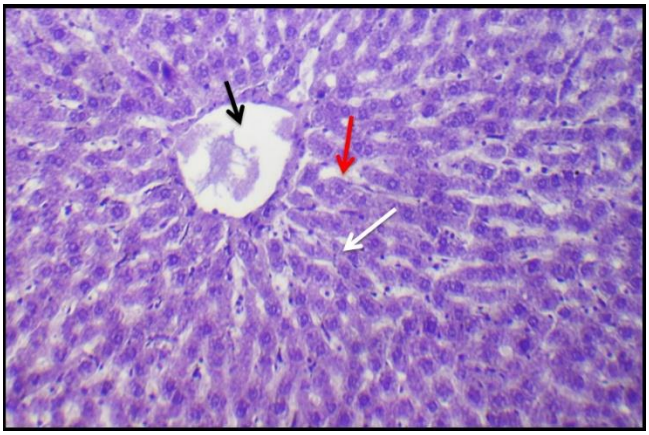

Figure 1: The liver section of the control group showed normal hepatic architecture, remarkable hepatocyte cord arrangements (white arrow) radiating around the normal central vein (black arrow), and normal sinusoids separating the hepatocyte cords (red arrow) (H and E, 10 X).

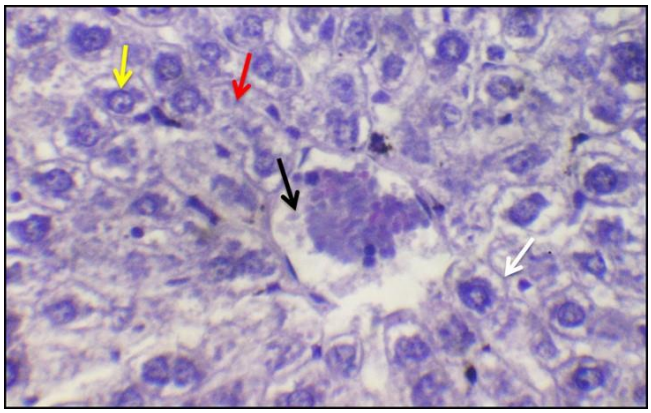

Figure 2: The liver section of the control group showed the normal hepatic architecture, remarkable hepatocyte cords arranged (white arrow) around the normal central vein (black arrow), with significant prominent nuclei (yellow arrow) and granular cytoplasm (red arrow) (H and E, 40 X).

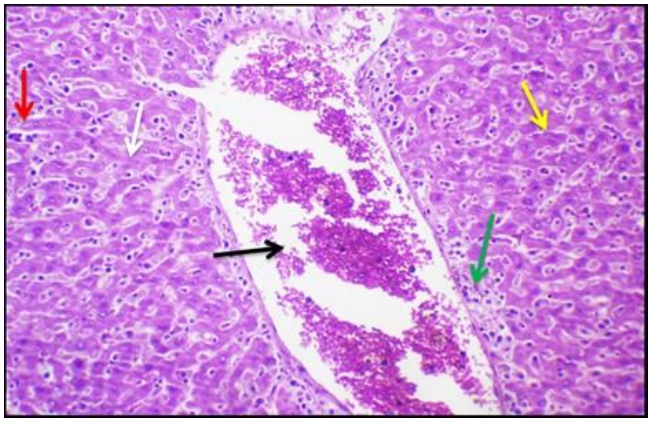

Figure 3: The liver section of a male mouse showed severe hepatic pathological alterations, massive congestion of central vein and sinusoids (black arrow), significant hepatocyte disorganization, and severe degeneration (yellow arrow), with remarkable necrosis (white arrow), intensive perivascular mononuclear inflammatory cells infiltration (green arrow) and severe interstitial inflammation (red arrow) (H and E, 10 X).

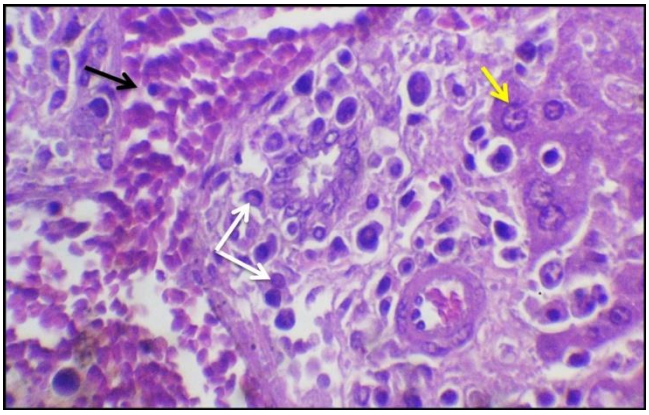

Figure 4: The liver section of a male mouse showed severe hepatic pathological alterations, severe congestion of the portal vein with thrombosis (black arrow), significant hepatocyte degeneration (yellow arrow), and severe perivascular mononuclear inflammatory cell infiltration (white arrow) (H and E, 40 X).

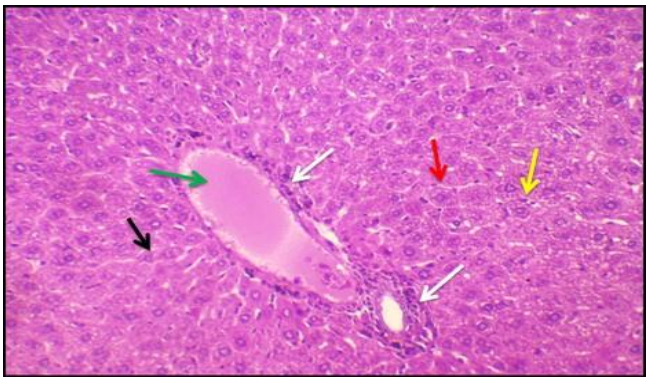

Figure 5: The liver section of a mice treated with an antibiotic revealed mild reversible changes of hepatic architecture, hepatocytes normal arrangements (black arrow), with significant and severe hepatocyte degeneration (red arrow), perivascular and surrounding bile ductile mononuclear inflammatory cells aggregation (white arrow), congested portal blood vessels (green arrow), mild interstitial infiltration of inflammatory cells (yellow arrow) (H and E,10 X).

Kidney histopathology

The kidneys of a normal group show normal renal cortical, normal glomerular tuft, bowman capsule, and appearance of bowman space and normal collecting tubes (Figures 9 and 10). In infected group, the renal capsule is thick due to severe granulomatous inflammation, severe cellular injuries, glomeruli atrophied, severe tubular epithelia, necrosis and interstitial inflammatory cells, and congestion of blood vessels (Figures 11 and 12). In Infected group treated by an antibiotic (ciprofloxacin), kidney section for an antibiotic treated mice post-infection with *E. coli* showed mild histological reversible changes, normal glomeruli with inflammatory cells around Bowman's capsule, severe degeneration and swelling of tubular epithelia appeared as star-shaped lumen, slight inflammatory cells interstitial infiltration (Figures 13 and 14). In Infected group treated by sodium propionate, the kidney section for organic acids treated mice post-infection with *E. coli* revealed mild histological changes, significant reverse change of renal capsule due to normal, remarkable normal glomeruli, semi-normal tubular epithelia, with slight interstitial inflammatory cells infiltration (Figures 15 and 16).

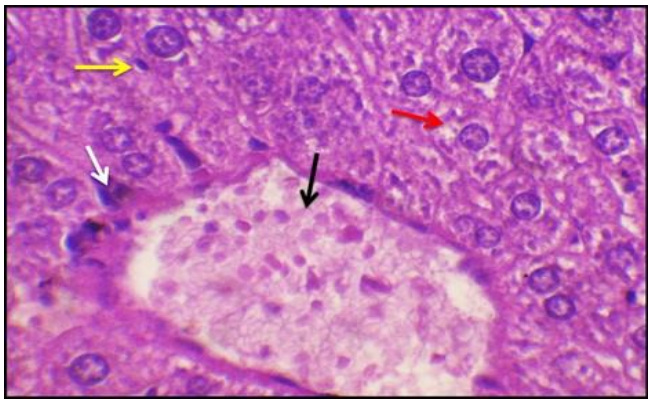

Figure 6: The liver section of a mice treated with an antibiotic showed mild reversible changes of hepatic architecture, slight congestion of the central vein (black arrow), with significant and severe hepatocyte degeneration (red arrow), mononuclear inflammatory cells infiltration around congested central vein (white arrow), mild interstitial infiltration of inflammatory cells (yellow arrow) (H and E, 40 X).

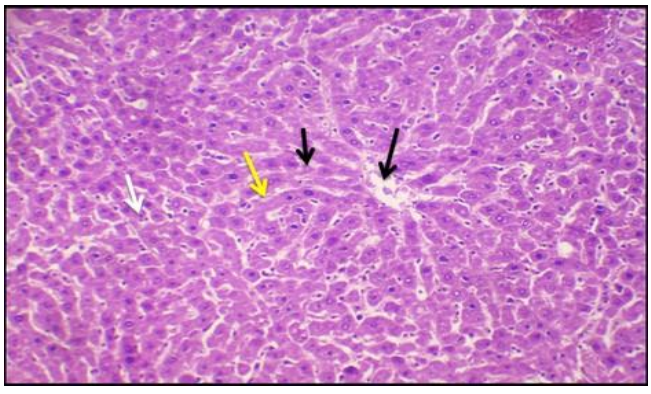

Figure 7: The liver section for organic acid-treated mice showed mild hepatic alterations, normal central vein, and sinusoids (black arrow), significant hepatocytes organized cords with mild degeneration (yellow arrow), slight mononuclear inflammatory cells infiltration (white arrow) (H and E,10 X).

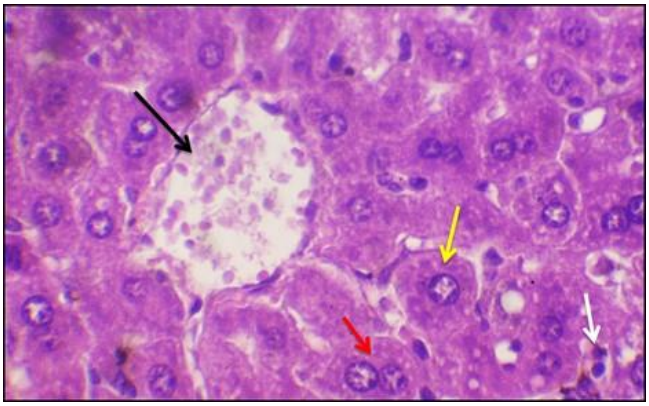

Figure 8: The liver section for organic acid-treated mice showed mild hepatic alterations, normal central vein, and sinusoids (black arrow), significant hepatocytes organized cords with mild degeneration (yellow arrow), slight mononuclear inflammatory cells infiltration (white arrow) and apparent bi nucleated hepatocytes (red arrow) (H and E,40 X).

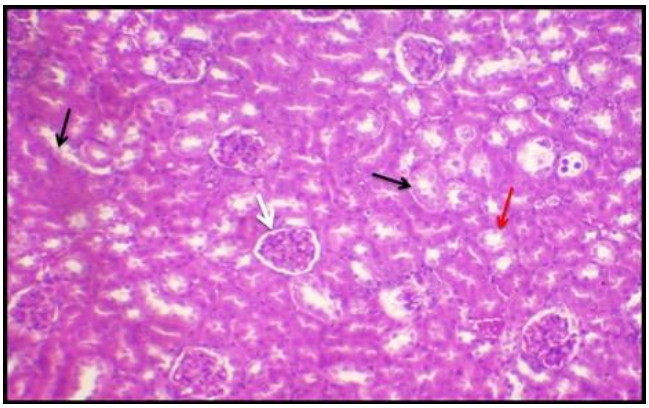

Figure 9: The kidney section of the control group revealed the normal renal cortical architecture, significant normal glomerular tuft (white arrow), normal distal convoluted tubules (black arrow), and normal collecting tubules (red arrow) (H and E,10 X).

Intestine histopathology

In control group, the photomicrograph of the intestine shows a normal intestine wall, with significant villi shape and length, normal sub mucosa, and muscular external layer. Payer patches also appear clearly (Figures 17 and 18). In the Infected groups the intestine showed external muscular layer is thickness, damage, and loss in intestinal villi, granulomatous inflammation in sub mucosa, and necrosis of the lamina propria layer (Figures 19 and 20). In the infected group treated by an antibiotic (ciprofloxacin), the intestinal transverse section of an antibiotic-treated mice post-infection with *E. coli* revealed remarkable reversible changes, represented by the significant villi length, areas of necrosis, with severe mucosal degeneration and moderate to severe inflammatory cell infiltration in mucosa and submucosa (Figures 21 and 22). In infected group treated by sodium propionate, a transverse section of intestine for organic acid treated group animal after *E. coli* infection showed remarkable villous length alteration, lamina propria with mild inflammatory cells infiltration, and presence of payer patches, with mild inflammatory cells infiltration and degeneration of enterocytes with proliferation of goblet cells (Figures 23 and 24).

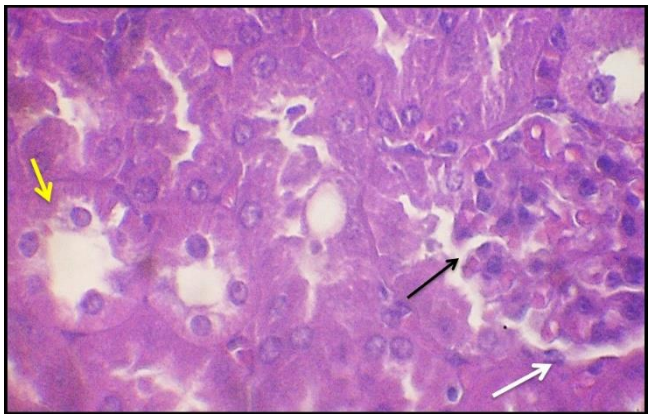

Figure 10: The kidney section of the control group revealed the normal renal cortical architecture, significant normal glomeruli with flattened epithelial lining of Bowman's capsule (white arrow), an apparent Bowman's space (black arrow), and normal collecting tubules (yellow arrow) (H and E,40 X).

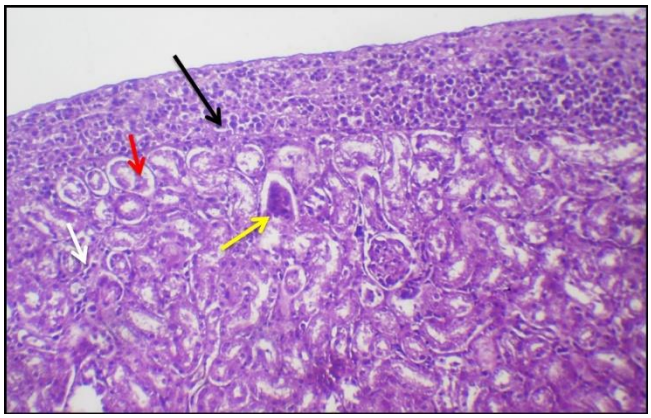

Figure 11: The kidney section of a male mouse showed severe cellular injuries, the significant thickness of the renal capsule due to severe granulomatous inflammation (black arrow), remarkable atrophied glomeruli (yellow arrow), severe tubular epithelial necrosis (red arrow) with interstitial inflammatory cells infiltration (white arrow) (H and E, 10 X).

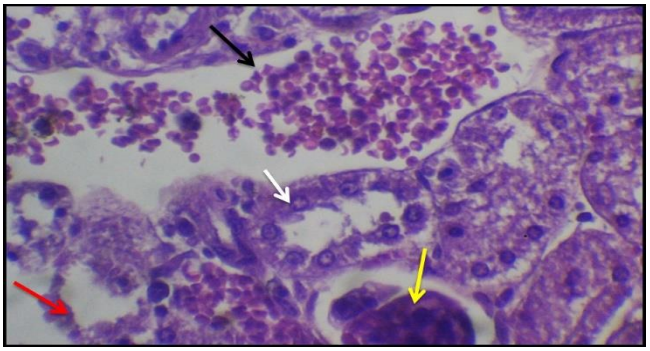

Figure 12: The kidney section for a male mouse showed severe renal injuries, significant congestion, and dilatation of renal blood vessels (black arrow), remarkable atrophied glomeruli (yellow arrow), severe tubular epithelial necrosis (red arrow) and degeneration (white arrow) (H and E, 40 X).

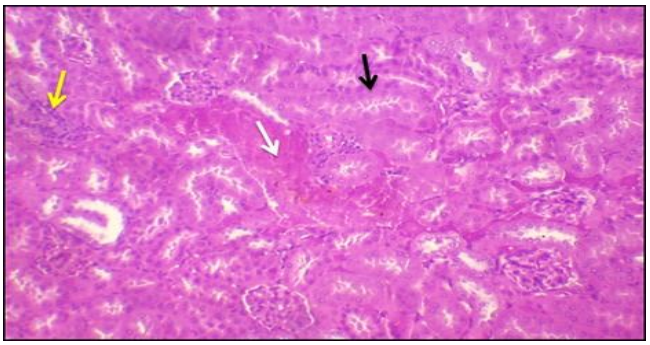

Figure 13: kidney section for an antibiotic-treated mouse revealed mild histological reversible changes, normal glomeruli with severe degeneration and swelling of tubular epithelia appeared as a star-shaped lumen (black arrow), remarkable focal granulomatous inflammatory cells aggregation scattered interstitially (yellow arrow), with slight interstitial hemorrhage (white arrow) (H and E,10 X).

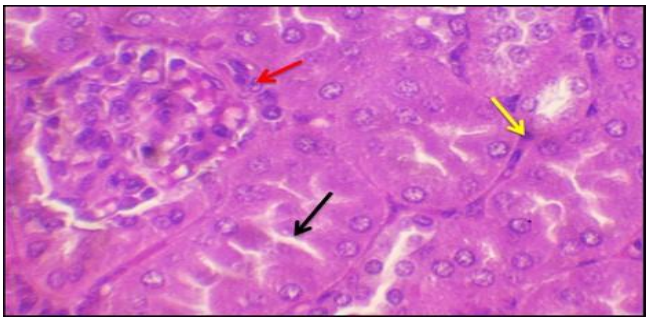

Figure 14: The kidney section for an antibiotic-treated mouse showed mild histological reversible changes, normal glomeruli with inflammatory cells around Bowman's capsule (red arrow), severe degeneration and swelling of tubular epithelia appeared as star-shaped lumen (black arrow), slight inflammatory cells interstitial infiltration (yellow arrow) (H and E,40 X).

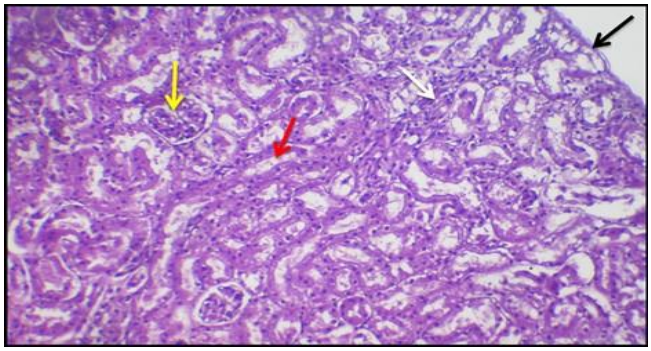

Figure 15: kidney section for organic acid-treated mice revealed mild histological changes, significant reverse change of renal capsule due to normal (black arrow), remarkable normal glomeruli (yellow arrow), semi-normal tubular epithelia (red arrow) with slight interstitial inflammatory cells infiltration (white arrow) (H and E, 10 X).

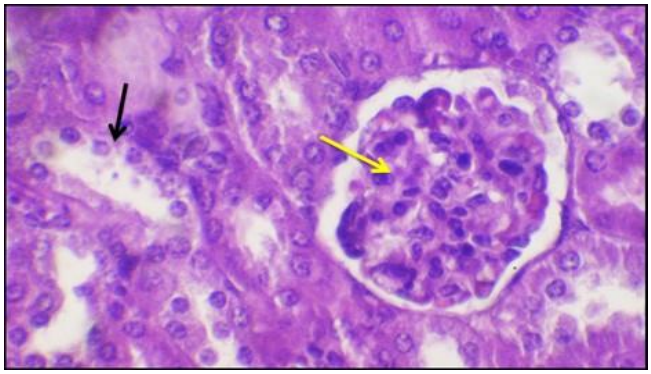

Figure 16: The kidney section for organic acid-treated mice revealed mild histological changes, remarkable normal glomeruli (yellow arrow), and semi-normal tubular epithelia (black arrow) (H and E,40 X).

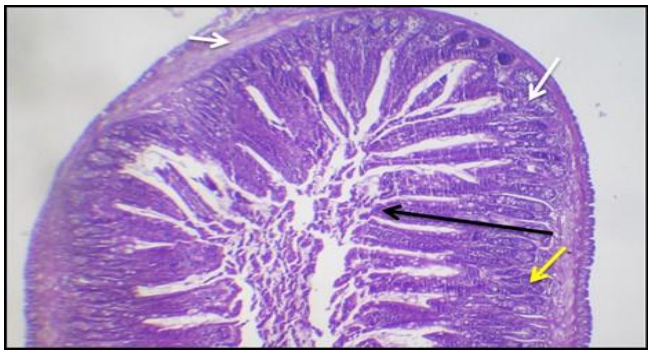

Figure 17: The transverse section of the intestine for a control group revealed the normal intestinal wall histological architecture, normal and significant villi shape and length (black arrow), normal submucosa and muscularis externa layer (white arrow), and apparent Payer Patches (yellow arrow) (H and E, 4X).

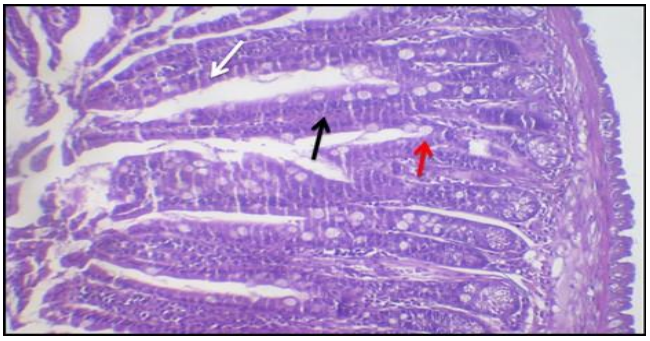

Figure 18: The transverse section of the intestine for a control group revealed the normal intestinal wall histological architecture, normal and significant crypts with supportive lamina propria (black arrow), remarkable crypts epithelial enterocytes (white arrow) with distinguished goblet cells (red arrow) (H and E, 10X).

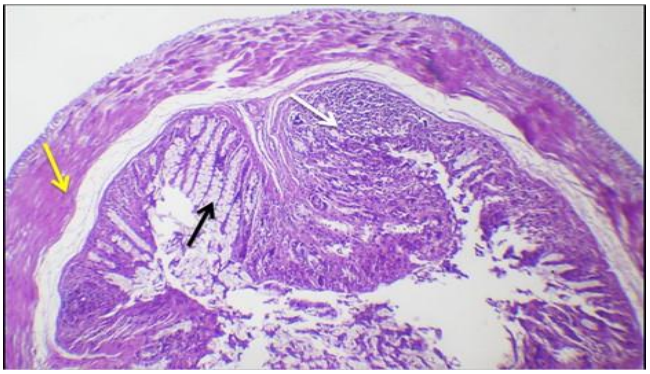

Figure 19: The transverse section of the intestine for the *E. coli* infection group revealed damage and loss in intestinal villi (black arrow), significant granulomatous inflammation in the submucosa (white arrow) and thickness of muscularis externa layer (yellow arrow) (H and E, 4X).

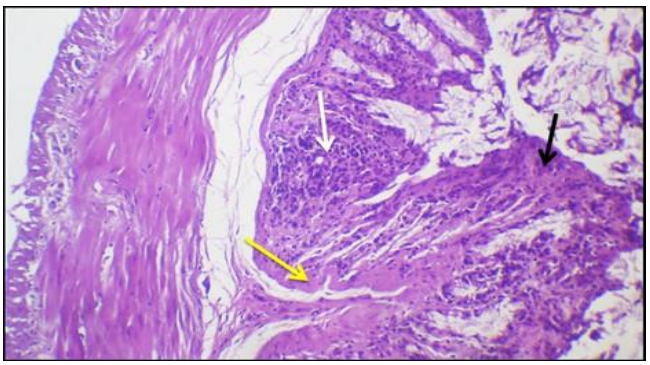

Figure 20: The transverse section of the intestine for the *E. coli* infection group revealed damage and loss in intestinal villi (black arrow), significant granulomatous inflammation in the submucosa (white arrow), and thickness of muscularis externa layer (yellow arrow) (H and E, 4X).

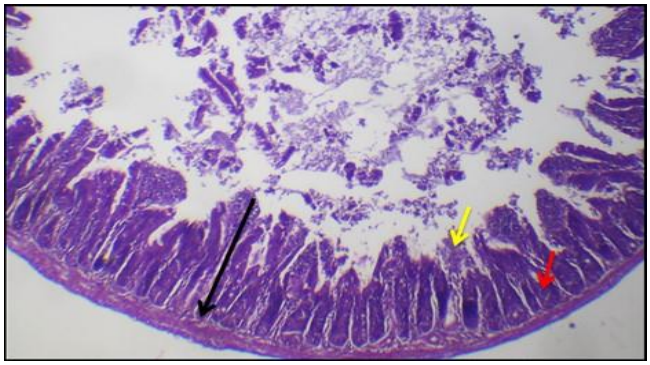

Figure 21: The intestinal transverse section of antibiotic-treated mice revealed remarkable reversible changes, represented by the significant villi length (black arrow) areas of necrosis (yellow arrow) with severe mucosal degeneration (red arrow) (H and E, 4 X).

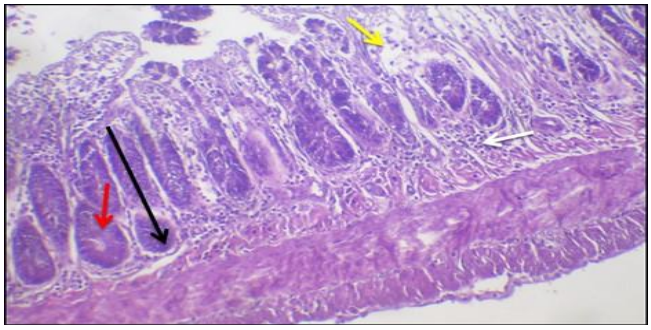

Figure 22: The intestinal transverse section of antibiotic-treated mice showed remarkable reversible changes, represented by the significant villi length (black arrow), areas of necrosis (yellow arrow), with severe mucosal degeneration (red arrow), and moderate to severe inflammatory cells infiltration in mucosa and submucosa (white arrow) (H and E, 10 X).

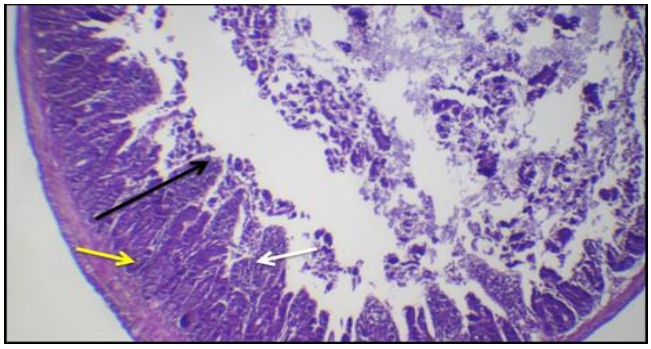

Figure 23: The transverse section of the intestine for the organic acid-treated group showed remarkable villous length alteration (black arrow), lamina propria with mild inflammatory cell infiltration (white arrow), and the presence of payer patches (yellow arrow) (H and E, 4X).

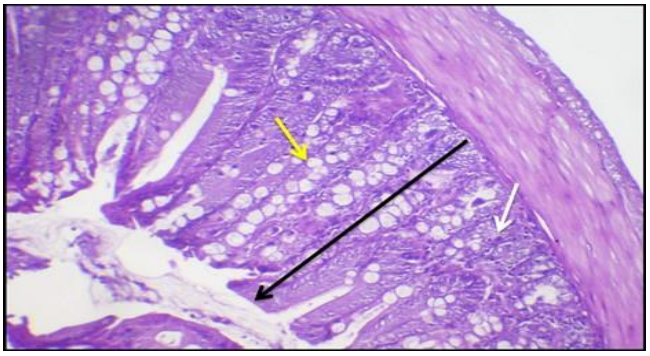

Figure 24: The transverse section of the intestine for the organic acid-treated group showed significant villous structures (black arrow), lamina propria with mild inflammatory cell infiltration (white arrow), and degeneration of enterocytes with proliferation of goblet cells (yellow arrow) (H and E, 10X).

Discussion

The non-0157 IQ1 serotype was given orally to the experimental animal (mice) in does $1x10^8$ C.F.U/ml according to Camundongos (21); after administration, the affected mice clinically were suffering poor appetite, foul smell of urea, difficulty breathing, tremors, diarrhea or bloody diarrhea and some were dead as mentioned in 24-26 after the appears of symptoms we give treatment, one group given ciprofloxacin and after 12 hours the animals start to show health signs, the second group treated with organic acids after 6 hours the health signs began to show up, it was better than antibiotic treatment, and our histopathological section insure that result, A wide variety of alternative antibiotics agents have been investigated for the treatment of diarrhea and seem to be effective agents for the improvement the acute Diarrheal cases in children when used therapeutically (24-27). The alternative antibiotics appear perfect for preventing and controlling recurrent bacterial and antibiotic-associated diarrhea (28,29).

Organic acids (found in keprofix) may prevent infectious diarrhea in several ways, such as by reducing luminal pH, producing bacteriocins, and encouraging mucus production (commensal pathogen cross-talk). It is possible that the superiority of probiotic therapy in viral diarrhea can be attributed to the ability of pathogenic bacteria to manufacture mucinase (30). Organic acids aid in pathogen elimination by increasing innate immunity and improving intestinal motility. Short-chain fatty acids produced by lactobacilli and bifidobacteria exert trophic effects on the intestinal epithelium, regulating cell proliferation and differentiation; they also promote bifidobacteria and oppose urease-producing strains, keeping the gut barrier intact (31).

Liver damage is most reliably detected by elevated levels of alanine aminotransferase and aspartate aminotransfer (32). The hepatic enzymes ALT and AST are found mainly in the cytoplasm and mitochondria, respectively. Transaminases are released into the circulation when the hepatocyte membrane becomes more permeable after liver damage. Serum transaminase levels are highly correlated with the severity of liver damage. Consistent with our findings, a previous study found that serum ALT and AST levels in mice were elevated following the administration of non-O157 *E. coli* (33).

This rise in BUN and creatinine may be due to the effect of bacteria and their toxins on the kidneys, and the findings agree with (34-36). The present study's findings corroborate those of (37), who found an increase in serum creatinine in rabbits infected with pathogenic *E. coli*, suggesting that these bacteria contribute to kidney failure.

Liver and kidney damage because the *E. coli* infection show degeneration and necrotic area in the liver cell and kidney as the primary target for the infection is the villi that are ulcerated and damaged due to bloody and pus diarrhea (38).

Several of the animals in this research had *E. coli* infections, and their internal organs revealed signs of a supportive inflammatory response and a tiny, numerous granulomatous lesions. The study's findings suggested that the microorganisms utilized were hazardous. These findings corroborated those of Contour *et al.* (39), who described *E. coli* infection as resulting in lesions of the intestine characterized by hyperplasia of goblet cells, mucin in the lumen, and inflammatory cells (primarily neutrophils and macrophages) in the lamina propria of atrophic villi, all of which indicate invasion of the epithelial cells of the intestinal mucosa and subsequent dissemination to the bloodstream of internal organs and When exposed to this infection, many hosts react with inflammation. Intestinal epithelial cells may respond to inflammation by producing more goblet cells. Some researchers believe that the increased number of goblet cells seen in other types of damage is an adaptive response that permits mucin released by goblet cells to form a thick gel that traps bacteria and irritants and prevents their access to the epithelium (40). Both mucin expression and secretion increased as chemically induced intestinal inflammation worsened (41). Mucin overproduction and secretion have also been linked to inflammation due to bacterial infection (42).

Among the many effects of *E. coli* infection on male mice blood parameters was a significant increase in total white blood cell count in the infected group to reach 10.15 in comparison with 8.52 in the control group. This increase may be attributable to *E. coli* infection, which has been shown to increase total leukocyte count, which agrees with (43).

Lymphopenia, characterized by a moderate to considerable absolute drop in lymphocytes, may be triggered by stress, as the data shows a decrease in lymphocytes in the infected group (43). Erythrocytes may be broken down by hemolysis enzymes produced by *E. coli* (41), leading to a drop in the number of erythrocytes, which in turn leads to a

decrease in hemoglobin concentration (44) and packed cells volume (45). The number of thrombocytes in the infected population is also much lower. Platelets may be greatly destroyed after antigen-antibody interactions on the platelets' surface membrane, which may explain the platelet drop in this research (46). This is likely the origin of the bulk of so-called idiopathic thrombocytopenia in animals (47). The attachment of bacteria to intestinal cells causes a lesion that progresses with effacement of the microvillus and changes the shape of the cells, as shown on histological inspection of infected groups (48).

The liver showed vacuolation and fatty degeneration in the hepatic cells, in addition to a proliferation of Kuepfer cells and an aggregation of mononuclear cells surrounding the significant veins. Multiple granulomatous lesions of varying sizes dispersed throughout the liver, along with central venous congestion and neutrophils in the vein lumen. There was a noticeable color difference between the two kidneys, with the left being noticeably paler than the right. Fibrin deposition and inflammatory cells were also seen in the capsular area of the cortical and medullary cross-sections under the microscope. At the same time, renal tubule dilatation and aggregation of mononuclear cells in the interstation with Bowman's space enlargement were also observed. Cystic dilatation with contact proteinaceous debris and atrophic glomerular tuft were also identified. These modifications may primarily be defined by an extensive actin cytoskeleton polymerization within the inebriated cell, leading to a paler-than-normal appearance of the kidneys (49), examined many bacterial variables thought to promote UPEC translocation across renal epithelial cells.

Hemorrhagic colitis results from intestinal injury. Samples of the intestines from individuals who presented with severe symptoms and had hemicolectomy, biopsy, or autopsy were analyzed for macroscopic and microscopic alterations. They may represent the most potent forms of intestinal involvement. Edema, hyperemia, bleeding, fibrinous exudates, vascular thrombosis, necrosis of the mucosa, and the creation of a pseudomembrane were all identified as pathological abnormalities (50).

Some antibiotics may increase the risk for complications, such as acute renal failure in HUS. Antibiotic therapy was associated with an elevated incidence of HUS in a prospective cohort study of 71 children hospitalized with diarrhea (51). During the last decade, alternative medicine has garnered much interest due to its potential as an effective treatment method. One of the recommended treatment techniques involves the utilization of chosen alternative antibiotics that have proven substantial potential for supporting quick recovery from STEC-producing diarrhea. A Meta-analysis of randomized controlled trials gave evidence of the efficacy of lactic acid-generating bacteria (Lactobacillus and Bifidobacterium) for both the prevention and treatment of acute diarrhea in newborns and young children (52). The immunological and pathological changes were also studied in mice infected with this strain (53). Between 1983 and 2002, the *E. coli* serogroup was responsible for 22% of all non-O157 STEC infections (54).

Conclusion

The study included the hematological, biochemical and histological changes of mice infected with non-O157 STEC isolates isolated from stool samples of infected humans and calves and compared the best antibiotics with organic acids for treatment. After experimentally infecting mice. The experimental study included four groups: ciprofloxacin and sodium propionate were used as comparator treatments. After observing the effect of bacteria on the internal organs, specifically the liver, kidneys, and intestines, and comparing the effectiveness of the treatments. The infected group showed severe histopathological changes, while the group treated with organic acids showed noticeable adverse changes in the kidneys and intestines and moderate changes in the liver. Blood samples were taken, and the organs were examined for histopathological changes. The livers of affected mice showed hepatocyte disruption, degeneration, thrombosis, and mononuclear cell infiltration. The kidney had renal capsular thickening, glomerular atrophy, and severe granulomatous inflammation. The intestine had thickening of the outer muscular layer, loss of villi, and areas of necrosis. The sodium propionate group significantly changed villus length and mild hepatic change. The infected group showed pathological histopathological changes in the liver, kidneys, and intestines, while the group treated with antibiotics or organic acids showed minor changes. The organic acid-treated group showed a marked reversal of histopathological changes in the kidneys and intestines, and the development of intestinal villus length was notable. It causes changes in some blood parameters. Treatment with organic acids has shown faster and better results than treatment with antibiotics.

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Conflict of interests

The authors declare no conflicts of interest regarding this manuscript's publication or funding.

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دراسة مقارنة للعلاج بسيبروفلوكساسين وبروبيونات الصوديوم الى الايشيريكيا القولونية غير و ١٥٧ المنتجة لسموم الشيجا ـ١ في الفئران المصابة

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الخلاصة

هدفت هذه الدراسة إلى التعرف على الإيشريكيا القولونية غير و ١٥٧ المنتجة لسموم الشيجا عن طريق أخذ عينات برازية من البشر والعجول المصابة. تم عزل نوع جديد من الإيشريكيا القولونية غير العترة و ١٥٧ المنتجة لسموم الشيجا ١ واستخدم لإصابة الفئران تجريبيا. اشتملت الدراسة على أربع مجموعات، بما في ذلك مجموعة السيطرة، والمجموعة المصابة، والمجموعة المصابة التي عولجت بالمضادات الحيوية (سيبروفلوكساسين)، والمجموعة المصابة التي عولجت بالحمض العضوي (بروبيونات الصوديوم). لدراسة وتقييم تأثير البكتيريا على الأعضاء الداخلية، وتحديدا الكبد والكلى والأمعاء، ومقارنة فعالية المضادات الحيوية المختلفة. أظهرت المجموعة المصابة تغيرات نسيجية مرضية شديدة، بينما أظهرت المجموعة المعالجة بالحمض العضوي (بروبيونات الصوديوم) انعكاسا ملحوظا للتغيرات في الكلى والأمعاء وتغيرات معتدلة للكبد. أصيبت الفئران بنوع جديد من الإيشريكيا القولونية غير و ١٥٧ المنتجة لسموم الشيجا ١ وعولجت بالحمض العضوية (بروبيونات الصوديوم) أو سيبروفلوكساسين. الحمض العضوية (بروبيونات الصوديوم) أظهر استجابة أفضل من المضادات الحيوية. تم أخذ عينات الدم وتم فحص الأعضاء من أجل ملاحظة التغيرات النسيجية المرضية. أظهر كبد الفئران المصابة اضطرابا في خلايا الكبد، وتنكسا، وتجلطا، وتسللا إلى الخلايا أحادية النواة. كانت الكلية تعاني من سماكة الكبسولة الكلوية، وضمور الكبيبات، والتهاب حبيبي شديد. كان للأمعاء سماكة في الطبقة العضلية الخارجية وفقدان الزغابات ومناطق النخر. أظهرت مجموعة الحمض عضوي (بروبيونات الصوديوم) تغيرا ملحوظا في طول الزغابات وتغير كبدي خفيف. أظهرت المجموعة المصابة تغيرات نسيجية مرضية في الكبد والكلى والأمعاء، بينما أظهرت المجموعات التي عولجت بالمضادات الحيوية أو حمض عضوي (بروبيونات الصوديوم) تغيرات طفيفة. أظهرت المجموعة المعالجة بالحمض عضوي انعكاسا كبيرا للتغيرات النسيجية المرضية في الكلى والأمعاء، وكان تغيير طول الزغابات في الأمعاء ملحوظا. الإشريكية القولونية غير و ١٥٧ المنتجة لسموم الشيجا ١ له تأثير نسيجي على الكبد والكلى والأمعاء ويسبب تغيرا في بعض مقاييس الدم. أظهرت المعالجة بالحمض العضوية (بروبيونات الصوديوم) نتائج أسرع وأفضل من العلاج بالمضادات الحيوية.