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Some biochemical and immunological parameters in rats infected with *Strongyloides stercoralis*

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Abstract

Strongyloidiasis, caused by *Strongyloides stercoralis*, is a globally distributed parasitic infection with significant public health concerns. Due to its ability to establish chronic auto-reinfection, understanding its physiological impact on host biochemistry, immune response, and metabolic homeostasis is crucial. This study aims to assess the systemic effects of *S. stercoralis* infection in a murine model using a combination of biochemical, immunological, and metabolic analyses. Male Wistar rats (n=30) were divided into three groups: control (uninfected), low-intensity infection (500 larvae), and high-intensity infection (2000 larvae). Infection was established via oral administration of infective third-stage larvae (L3). After 30 days, blood, urine, and fecal samples were collected for biochemical assays, immune profiling, and metabolic analysis. Serum markers of inflammation (IL-6, TNF-α, and IFN-γ), as well as oxidative stress markers (malondialdehyde, catalase, glutathione peroxidase), were measured using enzyme-linked immunosorbent assays (ELISA) and spectrophotometry. Renal and hepatic function was assessed via blood urea nitrogen (BUN), creatinine, ALT, and AST. The work was performed from November 1st to December 1st, 2024, in the Laboratory of Parasitology at the College of Veterinary Medicine, University of Al-Qadisiyah. Infected rats displayed significant increases in IL-6, TNF-α, and IFN-γ levels. Oxidative stress markers were elevated, with higher MDA levels and reduced catalase and glutathione peroxidase activity in infected groups. Kidney and liver function tests revealed increased BUN, creatinine, ALT, and AST levels, suggesting renal and hepatic dysfunction. This study provides a comprehensive biochemical and immunological characterization of *S. stercoralis* infection, demonstrating significant systemic alterations.

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Introduction

Prolonged infection with *Strongyloides stercoralis* as the culprit of persistent clinical manifestations was first documented in 1876. The infection can profoundly impact the host's physiology (1). *Strongyloides stercoralis* is the causative agent of strongyloidiasis, a parasitic infection of medical importance across both the tropics and increasingly in non-endemic regions. Global estimates of people affected remain farcically broad due to limited epidemiologic data, whilst diagnostic tools with adequate sensitivity are both insufficient and largely unavailable. *S. stercoralis* has attracted relatively little research attention. Major concern remains the rising prevalence of strongyloidiasis following infection in a greater number of immunocompromised patients. More efficient management will depend upon an enhanced understanding of both how the host responds to infection and what particular host predispositions facilitate dramatic consequences (2). *Strongyloides stercoralis* has been known as an emerging silent killer owing to its ability to cause persistent or long-lasting infection accompanied by various complications and high mortality rates. *S. stercoralis*

infection can occur almost worldwide, especially in tropical and subtropical regions. It is estimated that the global prevalence of *S. stercoralis* infection could range from 30 to 100 million. The prevalence of Strongyloides infection might be underestimated, and underdiagnosis can also lead to a lack of epidemiological data. Hence, detecting indirect or non-invasive forms can contribute to a comprehensive understanding of S. stercoralis, especially within the host, for future studies. If left untreated, an uncomplicated *S. stercoralis* infection can develop into a fatal hyperinfection syndrome (3). This nematode has five developmental stages: rhabditiform, filariform, free-living adult male, free-living adult female, and parasitic adult female. The infectious form for host invasion is the third-stage infective filariform larvae (iL3) (4). This durable characteristic of Strongyloides species contributes to its strong adaptability to the environment, as well as a complex life cycle involving free-living and parasitic generations. Although *S. stercoralis* and *S. fuelleborni* share the same parasitic patterns as human parasites, *S. fuelleborni* exclusively parasitizes non-human primates (humans are the exclusive host of *S. stercoralis*) (4). Strongyloides species might secrete certain secretory fluids from its infective stages and from the host to support the completion of each parasitic generation. Evasion strategies, especially the strong adaptability of Strongyloides species to the adverse host microenvironment (temperature and immunological response), may also contribute to the parasitism of S. stercoralis. Ever since this threadworm was first reported, gradually accumulating evidence suggests that slight skin penetration by filariform larvae causes host invasion. Dyspnea, bilateral leg edema, and eosinophilia can also occur (5). *S. stercoralis* is a soil-transmitted helminth and one of the most overlooked in the group of neglected tropical diseases. Although it has a global distribution, little is known about the prevalence or risk factors in most regions (5). Infections with S. stercoralis are common in impoverished, immunocompromised societies with poor sanitation and close proximity to faecal soils (6). *S. stercoralis* is the sole identified nematode capable of reproducing both parasitically and liberally. In the parasitic niche, the adults live within the small intestine, generally in the duodenum. Rather uniquely, the parasitic females reproduce in the absence of male parasites by parthenogenesis, generating eggs that give rise to first-stage larvae (F3), which are subsequently passed in human feces (7). There, they develop into infective filariform third-stage larvae (iL3), which either disperse in the soil or the host. It is the iL3 that is responsible for host infection; they are equipped with a sensory apparatus that detects carbon dioxide gradients near the skin's surface. After detecting a human host, the larvae are chemically triggered to start the host penetration. Host entry may occur in two main ways: the classical percutaneous penetration, followed by dissemination to the lungs via the bloodstream, or the snap-like invasion via oral ingestion (8). There is a clear predilection for the prior, notwithstanding clinical examples demonstrating the ramifications of oral entry (9). The spotlight on human *S. stercoralis*, given its clinical burden, is warranted. The nematode repeatedly infects the host through its unique autoinfection mode, leading to indefinite infection until it is effectively treated. The problematic nature of this autoinfective cycle is reflected in its contribution to heavy infection syndrome. Here, the large worm burden escalates uncontrollably, favoring dissemination to extra-intestinal sites and engendering a far higher mortality rate (10).

This study aims to assess the systemic effects of *S. stercoralis* infection in a murine model using a combination of biochemical, immunological, and metabolic analyses.

Materials and methods

Ethical approval

This study was approved by the Institutional Animal Care and Use Committee, numbered 1878 dated 29/04/2025.

Experimental animals

All procedures involving animals were performed in accordance with internationally accepted guidelines. Thirty male Wistar rats (8 weeks old, weighing 200-250 g) were randomly used for the study. Animals were housed in temperature-controlled conditions (22°C±2°C), with a 12-hour light/dark cycle, and provided with standard rat chow (23.9% crude protein, 5.0% fat, and 6.0% fiber) and water ad libitum.

Parasite infection

Rats were randomly assigned to three groups: control (uninfected), low infection (500 larvae), and high infection (2000 larvae). Infection was induced by oral administration of infective third-stage larvae (L3) of *S. stercoralis*, which were isolated from stool cultures of dogs at 25-28°C for 5-7 days. Then, using the Baermann technique, they were collected under a microscope. Using a previous pilot study conducted by the current researchers, low and high infection levels were induced at 500 L3 and 2000 L3, respectively.

Sample collection

At 30 days post-infection, animals were euthanized using CO_2 asphyxiation, and blood, urine, liver, and kidney tissues were collected. Serum was separated by centrifugation (3000 rpm for 10 min) and stored at -80°C until further analysis.

Biochemical and immunological assays

Serum markers of inflammation, including IL-6, TNF-α, and IFN-γ, were measured using commercial ELISA kits following the manufacturer's protocol. Oxidative stress markers, including malondialdehyde (MDA), catalase, and glutathione peroxidase (GPx), were quantified using spectrophotometry.

Renal and hepatic function analysis

Blood urea nitrogen (BUN), creatinine, alanine aminotransferase (ALT), and aspartate aminotransferase (AST) levels were measured using an automated biochemical analyzer.

Statistical analysis

All data were analyzed using a One-Way ANOVA with Bonferroni post hoc tests. Statistical significance was set at $P<0.05$.

Results

Infected rats exhibited a significant increase in pro-inflammatory cytokines, including IL-6, TNF-α, and IFN-γ, compared to controls (Figure 1). Infected rats showed a significant increase in pro-inflammatory cytokines, including IL-6, TNF-α, and IFN- γ, compared with controls. IL-6 levels were 80 pg/mL in the low-infection group. They escalated to 150 pg/mL in the high-infection group, showing a progressive inflammatory response (Figures 2 and 3). Oxidative stress parameters revealed an elevation in MDA levels, with infected rats demonstrating a 3–4-fold increase. Concurrently, catalase and GPx activity were markedly reduced, suggesting an overwhelmed antioxidant defense system (Figures 4-6). Renal function parameters, BUN and serum creatinine, were significantly elevated in infected groups (Figures 7 and 8). BUN levels doubled in low-infection rats, 35 mg/dL, and escalated to 50 mg/dL in the high-infection group. A similar increase in creatinine was observed, rising from 0.6 mg/dL (control) to 2.0 mg/dL (high-infection), indicating significant nephropathy. Liver enzyme analysis showed a 2.5 to 3-fold increase in ALT and AST levels (Figures 9 and 10). ALT levels rose to 80 U/L (low infection) and 120 U/L (high infection). In contrast, AST followed a similar pattern, increasing to 130 U/L in heavily infected animals (Table 1).

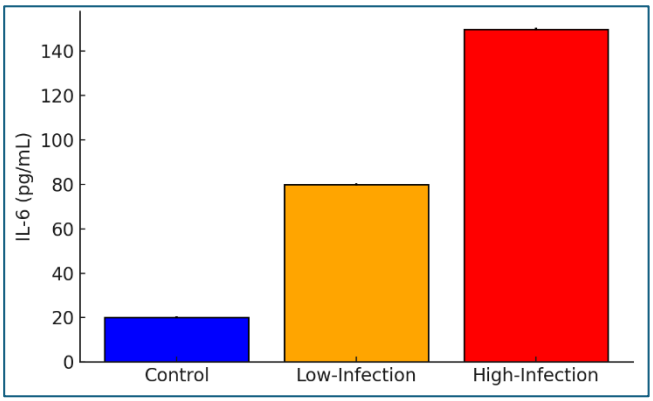

Figure 1: IL6 levels in different groups of rats infected with *S. stercoralis*.

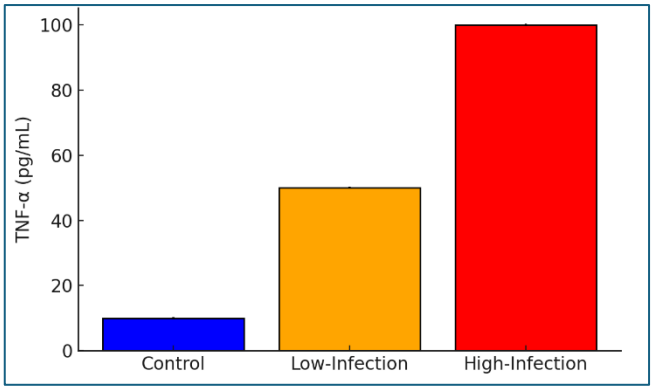

Figure 2: TNF-alpha levels in different groups of rats infected with *S. stercoralis*.

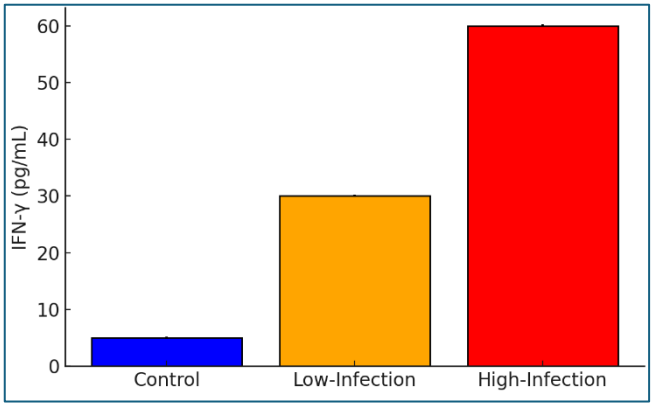

Figure 3: IFN-gamma levels in different groups of rats infected with *S. stercoralis*.

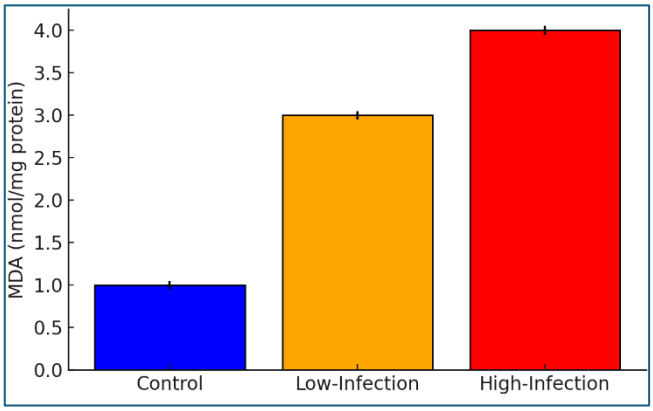

Figure 4: MDA levels in different groups of rats infected with *S. stercoralis*.

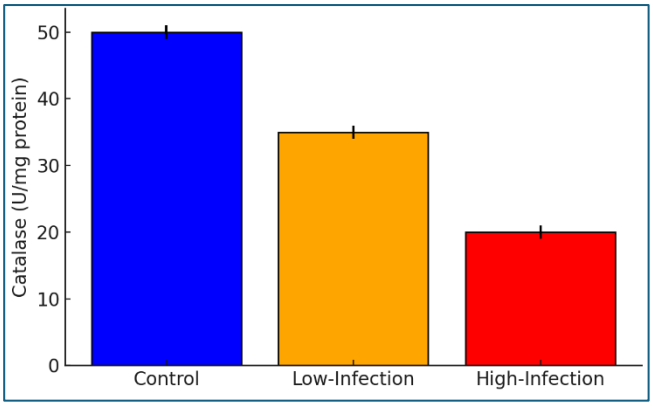

Figure 5: Catalase levels in different groups of rats infected with *S. stercoralis*.

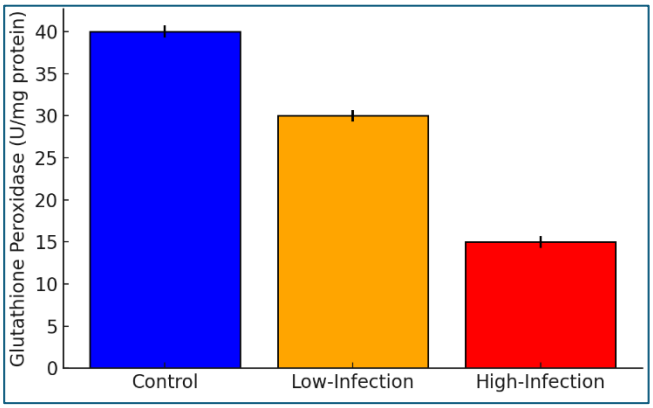

Figure 6: Glutathione peroxidase (GPx) levels in different groups of rats infected with *S. stercoralis*.

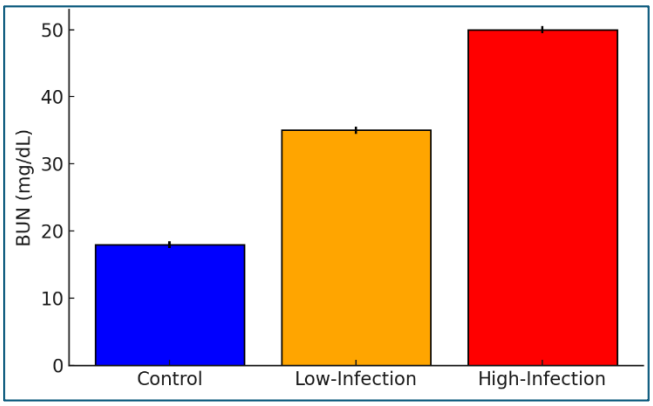

Figure 7: BUN levels in different groups of rats infected with *S. stercoralis*.

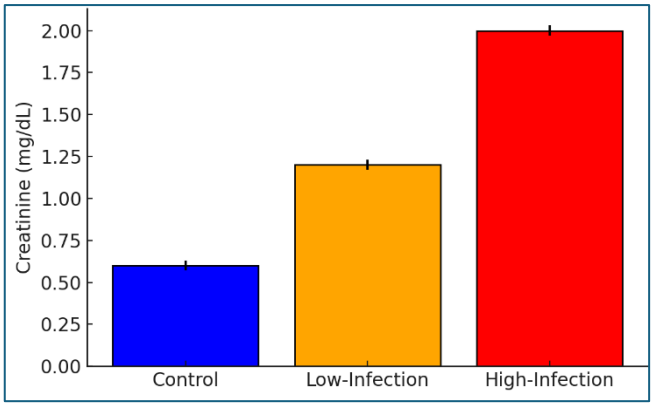

Figure 8: Creatinine levels in different groups of rats infected with *S. stercoralis*.

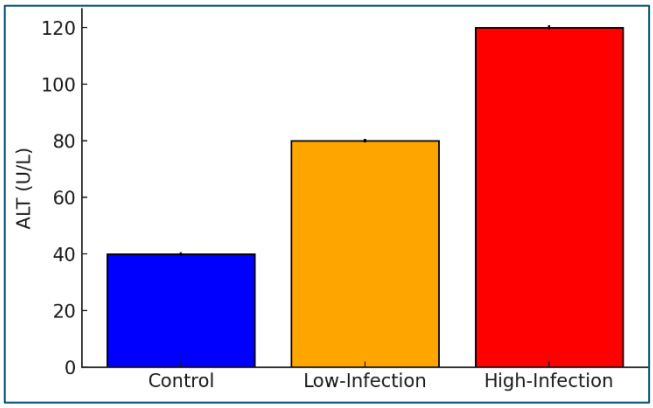

Figure 9: ALT levels in different groups of rats infected with *S. stercoralis*.

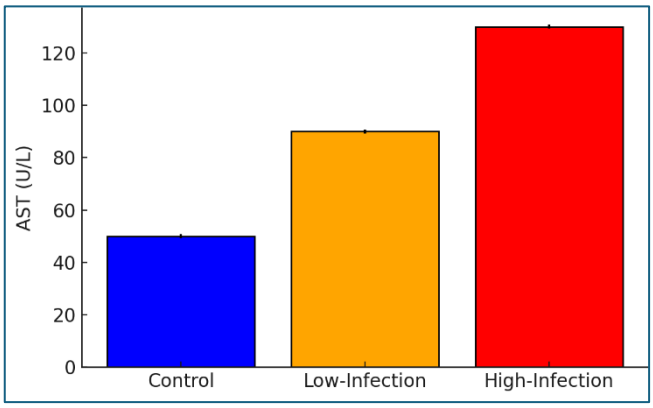

Figure 10: AST levels in different groups of rats infected with *S. stercoralis*.

Table 1: Details of statistical analysis

Parameter	Control	Low-Infection	High-Infection	Control vs Low	Control vs High
IL-6 (pg/mL)	20±0.5	80±0.8	150±1.2	<0.03	<0.0001
TNF-α (pg/mL)	10±0.3	50±0.6	100±1.0	<0.01	<0.0001
IFN-γ (pg/mL)	5±0.2	30±0.5	60±0.7	<0.03	<0.0001
MDA (nmol/mg)	1.0±0.05	3.0±0.07	4.0±0.09	<0.05	<0.001
Catalase (U/mg)	50±1.0	35±0.9	20±0.8	<0.05	<0.003
GPx (U/mg)	40±0.7	30±0.6	15±0.5	<0.02	<0.005
BUN (mg/dL)	18±0.5	35±0.8	50±1.0	<0.05	<0.0009
Creatinine (mg/dL)	0.6±0.03	1.2±0.05	2.0±0.06	<0.05	<0.0001
ALT (U/L)	40±0.6	80±0.9	120±1.2	<0.01	<0.007
AST (U/L)	50±0.7	90±1.0	130±1.3	<0.01	<0.001

Discussion

The infection caused by *S. stercoralis* represents a significant but neglected threat to global health, especially in tropical and subtropical areas with insufficient diagnostic capabilities. The nematode's capacity to maintain infection in humans for decades through autoinfection creates complex obstacles for disease control, particularly affecting immunocompromised patients (11). Patients who undergo immunosuppressive therapy, including those with HIV/AIDS or organ transplant recipients, need better early diagnostic methods and treatment options because of the chronic infection that can lead to heavy infection and dissemination. This investigation aimed to thoroughly examine the biochemical, immunological, and physiological changes triggered by *S. stercoralis* infection with a specific focus on inflammatory cytokine responses, oxidative stress markers, and organ dysfunctions.

Infected hosts exhibit substantial increases in pro-inflammatory cytokines IL-6, TNF-α, and IFN-γ, showing that *S. stercoralis* drives continuous immune activation that could result in ongoing inflammation (12). Previously conducted research showed that prolonged exposure to helminths, such as S. stercoralis, creates an immune environment (13). Research has shown that S. stercoralis infection induces immunomodulatory changes that may cause some affected individuals to develop regulatory or Th2-dominant immune responses (14). The altered immune responses make it hard to entirely eliminate the parasite, particularly among patients with established infections.

The current research demonstrated significant oxidative stress, as evidenced by elevated malondialdehyde (MDA) levels and reduced catalase and glutathione peroxidase activity in parasite-infected groups. Persistent immune activation and inflammatory responses during helminth infections lead to oxidative damage, which aligns with prior research findings (15). Long-term oxidative stress worsens tissue damage in organs involved in detoxification and metabolic regulation, such as the liver and kidneys. The study demonstrates that *S. stercoralis* infection causes severe systemic damage since liver (ALT, AST) and kidney (BUN, creatinine) function markers rise significantly.

Infected animals showed renal impairment, which supports existing evidence that strongyloidiasis leads to renal complications, especially in heavy infection syndrome cases (16). Researchers have theorized that immune complex deposition and inflammation-related renal damage might occur. Yet, direct kidney toxicity caused by the parasite is still not well-defined (17). The global systematic review of strongyloidiasis cases found that patients with concurrent bacterial or viral infections are at higher risk of kidney damage because these additional infections weaken the immune system. These findings demonstrate the need to include renal assessment in the clinical evaluation of patients with chronic strongyloidiasis.

The current study indicates significant liver involvement during infection, as evidenced by observed hepatic changes, including elevated ALT and AST levels. Studies showed that disseminated strongyloidiasis leads to hepatobiliary complications, which include cholestatic liver injury and hepatocellular dysfunction from continuous immune activation (18). Intestinal nematodes, such as S. stercoralis, cause bacterial endotoxin translocation, which worsens liver inflammation through the gut-liver axis during parasitic infections (19-30). The results demonstrate that liver function monitoring is essential for patients with chronic strongyloidiasis who reside in areas where hepatitis B or C infections might influence disease progression.

Strongyloidiasis's ability to worsen other diseases highlights its significance as a neglected tropical disease. Recent research shows that *S. stercoralis* can coexist with other enteric pathogens, leading to increased intestinal dysbiosis and worsening patient conditions. Research indicates that *S. stercoralis* infections have been found alongside hookworms, protozoa, and *Mycobacterium tuberculosis,* suggesting that this parasite can modify host vulnerability to other infectious organisms (31-37). The current research results support the idea of integrated parasitic disease control strategies that require routine multi-pathogen screening, especially for vulnerable populations

such as immunocompromised patients and residents of endemic regions.

A primary obstacle in strongyloidiasis research is limited access to reliable epidemiological data. Endemic countries often lack comprehensive surveillance systems, resulting in lower case reporting and an inaccurate assessment of disease prevalence (38-41). The latest meta-analysis findings show that *S. stercoralis* prevalence estimates face challenges because diagnostic methods and sampling strategies do not match up (42-49). To bridge these gaps, we must implement global initiatives to standardize diagnostic techniques, expand screening programs, and enhance public education about the risks of this parasitic infection (50).

Conclusion

The research demonstrates essential findings on the systemic impacts of Strongyloides stercoralis infection, including extensive alterations in immune responses and oxidative stress levels that compromise organ health. The concurrent rise in pro-inflammatory cytokines and oxidative stress markers, together with hepatic and renal dysfunction, necessitates a multidisciplinary approach to properly diagnose and manage Strongyloidiasis. The primary focus of upcoming research must be on understanding parasite-host interaction mechanisms in detail while developing better methods for early detection and creating specific treatments to reduce the long-term effects of chronic infections. Recognizing strongyloidiasis as a significant global health threat allows us to implement better control measures to lower disease-related deaths and illnesses among those affected.

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Conflict of interest

The authors have not received any funding or benefits from industry, an agency, or a financing source, or elsewhere, to conduct this study.

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بعض المؤشرات البيوكيميائية والمناعية في الجرذان المصابة بالأسطوانية البرازية

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الخلاصة

الإصابة بالدودة الخيطية، والتي تُسببها دودة الأسطوانية البرازية، هي عدوى طفيلية منتشرة عالميا وتشكل مصدر قلق صحي عام كبير. بسبب قدرتها على إحداث عدوى مزمنة وإعادة العدوى الذاتية، فإن فهم تأثيراتها الفسيولوجية على كيمياء الجسم والاستجابة المناعية والتوازن الأيضي للمضيف يُعد أمرا بالغ الأهمية. يهدف هذا البحث إلى تقييم التأثيرات الجهازية للإصابة بدودة الأسطوانية البرازية في نموذج فأري باستخدام مجموعة من التحليلات البيوكيميائية والمناعية والأيضية. تم تقسيم ذكور الجرذان من سلالة ويستار (عددها ٣٠) إلى ثلاث مجموعات: مجموعة ضابطة (غير مصابة)، ومجموعة إصابة منخفضة الشدة (٥٠٠ يرقة)، ومجموعة إصابة عالية الشدة (٢٠٠٠ يرقة). تم إحداث العدوى عن طريق الإعطاء الفموي ليرقات المرحلة الثالثة المعدية. وبعد ٣٠ يوما، تم جمع عينات الدم والبول والبراز لإجراء التحاليل البيوكيميائية والتوصيف المناعي والتحليل الأيضي. تم قياس مؤشرات الالتهاب في المصل الإنترلوكين السادس وعامل تنخر الورم الفا، والانترفيرون كاما، ومؤشرات الإجهاد التأكسدي (المالونديالديهيد، الكاتالاز، والكلوتاثيون) باستخدام اختبارات المقايسة المناعية المرتبطة بالإنزيم وتقنية المطيافية. تم تقييم وظائف الكلى والكبد من خلال قياس مستويات نيتروجين اليوريا في الدم، الكرياتينين، وإنزيمات الكبد. أُجري العمل خلال الفترة من ١ نوفمبر إلى ١ ديسمبر ٢٠٢٤، في مختبر الطفيليات بكلية الطب البيطري، جامعة القادسية. أظهرت الجرذان المصابة زيادات ملحوظة في مستويات الإنترلوكين السادس وعامل تنخر الورم الفا والانترفيرون كامل، كما ارتفعت مؤشرات الإجهاد التأكسدي مع زيادة مستويات المالونديالديهيد وانخفاض نشاط الكاتالاز وإنزيم الجلوتاثيون بيروكسيداز في المجموعات المصابة. كشفت اختبارات وظائف الكلى والكبد عن ارتفاع في مستويات نيتروجين اليوريا في الدم، والكرياتينين، وأنزيمات الكبد مما يشير إلى وجود خلل في وظائف الكلى والكبد. يوفر هذا البحث توصيفا شاملا للتغيرات البيوكيميائية والمناعية الناتجة عن الإصابة بدودة الأسطوانية البرازية مما يبرز التغيرات الجهازية الهامة الناتجة عن العدوى.