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The Effect of Snakehead Fish on Superoxide Dismutase Levels and Gastric Histopathological Features in a Rat Model of Liver Cirrhosis

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ABSTRACT

Liver cirrhosis is a chronic disease that frequently leads to complications such as portal hypertensive gastropathy (PHG), which is characterized by increased oxidative stress and reduced activity of antioxidant enzymes such as Superoxide Dismutase (SOD). Snakehead fish (Channa striata) is known to contain high levels of albumin and amino acids that function as antioxidants and anti-inflammatory agents. This study aimed to analyze the effect of Channa striata extract on SOD levels and gastric histopathological features in a rat model of liver cirrhosis. This experimental research employed a randomized post-test-only control group design involving 30 male Wistar rats divided into five groups: normal control (K1), cirrhosis without treatment (K2), and three cirrhosis treatment groups receiving Channa striata extract at doses of 100 mg/kgBW (K3), 150 mg/kgBW (K4), and 200 mg/kgBW (K5). SOD levels were measured through tissue homogenization, while gastric histopathology was examined using Hematoxylin–Eosin staining. The results showed a significant increase in SOD levels with a dose–response pattern ($p < 0.001$) and a gradual improvement in gastric histopathology ($p > 0.05$). In conclusion, Channa striata extract has the potential to enhance antioxidant activity and improve gastric histological structure in rats with liver cirrhosis.

Keywords: liver cirrhosis, snakehead fish, portal hypertensive gastropathy, superoxide dismutase

INTRODUCTION

Liver cirrhosis is a disease or group of liver disorders characterized by the loss of its lobular structure due to fibrosis, parenchymal cell damage, and ongoing regenerative processes. Consequently, nodular structures form within the liver [1]. The leading cause of liver cirrhosis in Asia is chronic hepatitis B, accounting for 37.3%, followed by alcohol consumption (24.1%) and chronic hepatitis C (22.3%) [2].

Several complications may arise from liver cirrhosis, including gastrointestinal bleeding, ascites, hepatorenal syndrome, hepatic encephalopathy, spontaneous bacterial peritonitis, and hepatocellular carcinoma. These complications can increase mortality risk and cause significant discomfort for patients [3]. Mortality and morbidity associated with liver cirrhosis often result from complications of portal hypertension, which is frequently underestimated despite being preventable. Manifestations of portal hypertension may include gastroesophageal varices and portal hypertensive gastropathy [4].

Portal hypertensive gastropathy (PHG) is a common complication that is often overlooked in the clinical diagnosis of patients with liver cirrhosis. The incidence of PHG varies between 20% and 98% among cirrhotic patients. Gastrointestinal bleeding in cirrhotic patients exhibiting this manifestation accounts for approximately 10% of all upper gastrointestinal bleeding cases, making it the second most common cause after esophageal variceal bleeding [5].

In general, PHG is diagnosed through endoscopic examination, characterized by distinctive features on the gastric mucosa such as a mosaic pattern and red spots. Endoscopic diagnosis is typically based on these specific characteristics. A mosaic or “snakeskin” pattern usually indicates a mild condition, whereas the presence of additional red spots, often referred to as “cherry spots,” signifies a severe condition. In more advanced cases, these macroscopic signs may be accompanied by chronic symptoms such as bleeding and fluid discharge [6].

Bleeding is usually mild to moderate and rarely fatal. PHG is characterized by a hyperdynamic circulation caused by portal hypertension. This hyperdynamic state disrupts the gastric mucosal defense mechanisms, triggers the release of pro-inflammatory mediators, and inhibits growth factors, thereby making the gastric mucosa more susceptible to injury [7]. These histological findings should not be

relied upon for the diagnosis of PHG because they are nonspecific [8].

Ortal hypertensive gastropathy triggers excessive production of reactive oxygen species (ROS), particularly superoxide radicals (O_2^-), which disrupts the balance between gastric mucosal oxidation processes and the antioxidant defense system. This imbalance leads to damaging tissue peroxidation. Under normal conditions, ROS levels are tightly regulated by antioxidant enzymes such as Superoxide Dismutase (SOD) to maintain optimal physiological levels. However, in this condition, the balance is disturbed due to increased ROS generation and/or reduced antioxidant capacity, resulting in oxidative stress. Consequently, SOD levels in portal hypertensive gastropathy decrease. The harmful effects of this condition include oxidative stress, mitochondrial dysfunction in parietal cells, impaired energy production, damage to parietal cell function, inhibition of gastric acid secretion, and oxidative injury to the gastric mucosa [7].

In liver cirrhosis, a decrease in albumin levels is commonly observed. Albumin is a protein primarily produced by the liver and functions as an antioxidant that binds free radicals arising from oxidative stress in portal hypertensive gastropathy [9]. The amino acids contained in albumin also play a vital role in healing processes, supporting platelet function, neovascularization, lymphocyte formation, fibroblast proliferation, collagen synthesis, and wound remodeling [10].

Snakehead fish (Channa striata), known in Indonesia as ikan gabus, is a freshwater species commonly found in swamp areas [11]. Snakehead fish is known for several health benefits, particularly its ability to accelerate wound healing [12]. This effect is associated with its high protein content, especially albumin as the primary component [13]. The albumin and amino acid content in Channa striata plays an essential role in enhancing tissue regeneration, thereby accelerating the wound-healing process [14]. Albumin functions as a binding and transport molecule, regulates osmotic pressure, increases cellular permeability, and serves as an antioxidant [15]. As an exogenous antioxidant, albumin can enhance SOD activity. Several amino acids, including arginine and arginase, contribute to the body's immune response mechanisms. Meanwhile, lysine, aspartic acid, glutamic acid, and other amino acids function as antioxidants that act synergistically with fatty acids to maintain cellular oxidative balance [16]. Extracts of snakehead fish have been shown to increase SOD levels, demonstrating an improvement in antioxidant status following administration of Channa striata extract in Wistar rats [17]. Therefore, this study aims to analyze the effect of Channa striata extract on SOD levels and gastric histopathological features in a rat model of liver cirrhosis.

RESEARCH METHODS

This study was conducted at the Ellio Laboratory in Medan from May to September 2025 after obtaining ethical approval from the Ethics Committee. The snakehead fish (Channa striata) extract powder used in this study was a product of PT. Herbal Nusantara Solo and distributed by PT. Mega Medica Pharmaceutical, with distribution authorization POM HT.203 300 841. The powder was dissolved using a 0.5% CMC solvent to prepare a solution ready for administration to the test animals.

The experimental animals used in this study were male Wistar rats (Rattus norvegicus sp.) aged 6–8 weeks, weighing 150–250 grams, healthy, showing no anatomical abnormalities, and with no prior history of use in other studies. All animals were maintained under standard laboratory conditions with ad libitum access to food and drinking water. A total of 30 rats were divided into five treatment groups: a normal control group without induction (K1), a cirrhosis group induced with CCl_4 without therapy (K2), and three cirrhosis treatment groups receiving oral administration of snakehead fish extract at doses of 100 mg/kgBW (K3), 150 mg/kgBW (K4), and 200 mg/kgBW (K5). Cirrhosis induction was performed by administering CCl_4 diluted in coconut oil orally twice a week for six weeks at a dose of 1 mL/kgBW (10% CCl_4 in coconut oil, consisting of 0.1 mL CCl_4 and 0.9 mL coconut oil). During the seventh and eighth weeks, the treatment groups received snakehead fish extract according to their respective doses, while the cirrhosis control group received only 0.5% CMC. All rats were terminated at the end of the eighth week.

The measurement of Superoxide Dismutase (SOD) levels was performed using a spectrophotometric method based on the procedure of Misra and Fridovich at a wavelength of 480 nm [18]. After the rats were anesthetized and euthanized, gastric tissue samples measuring 3 × 3 × 3 mm were collected by biopsy. A total of 0.2 grams of tissue was homogenized in 1 mL of physiological NaCl solution and then centrifuged at 8000 rpm for 20 minutes. A 500 µL aliquot of the supernatant was used for SOD level analysis.

Histopathological examination was performed on the fundus–corpus region of the stomach. Tissue samples measuring 1 × 1 × 1 cm were fixed in 10% Neutral Buffered Formalin (NBF), then processed through graded dehydration using 70%, 80%, 90%, and 96% alcohol, followed by absolute alcohol, toluene, and paraffin. The tissues were embedded using an embedding apparatus, sectioned with a microtome at a thickness of 4–5 µm, and stained using the Harris Hematoxylin–Eosin method. The slides were examined under a microscope at 400× magnification, and microscopic changes were recorded from five fields of view.

Statistical analysis of SOD levels was performed using a One-Way ANOVA followed by Tukey's post hoc test, while histopathological data were analyzed using the Kruskal–Wallis test. The research findings are presented in descriptive form, along with p-values and

data visualizations to strengthen the interpretation of results.

RESULT AND DISCUSSION

The results of this study showed that no stromal fibrosis was found in the gastric tissue of rats with liver cirrhosis. However, several histological abnormalities were observed, including capillary and venular dilation, inflammatory cell infiltration, gastric mucosal erosion, and lamina propria edema. In this study, we did not identify stromal fibrosis; instead, the main findings consisted of capillary and venular dilation, inflammatory cell infiltration or gastric mucosal erosion, and lamina propria edema. For clarity in presenting the results, these findings were categorized into two groups: lesion types and inflammation grades. Lesion types included capillary and venular dilation, gastric mucosal erosion, and lamina propria edema. Inflammation grade referred to the degree of inflammatory cell infiltration. A scoring system was used to assess the severity of both lesion types and inflammation grades, ranging from mild to severe. For lesion types, the scoring was defined as (0) None, (1) Epithelial erosion, (2) Ulcer, and (3) Perforation [19]. For inflammation grade, the scoring was defined as (0) None, (1) Mild, (2) Moderate, and (3) Severe [20]. During the study, one rat in the K2 group died. The analysis of SOD levels showed that the normal group (K1) had the highest levels, while the cirrhosis group (K2) had the lowest. The treatment groups (K3–K5) demonstrated a gradual dose-dependent increase in SOD levels. ANOVA revealed significant differences among groups ($p < 0.001$), and Tukey's test indicated that K2 differed significantly from all other groups, whereas K5 did not differ significantly from K1 but differed significantly from K2, K3, and K4 (Table 1).

Table 1. Mean ± SD of Gastric SOD Levels (U/mL) in Each Group

Group	Mean ± SD	p-Value	Tukey Notation
K1	15.17 ± 0.61	p <0.001*	a
K2	7.77 ± 0.49		b
K3	10.40 ± 0.44		c
K4	12.07 ± 0.32		d
K5	13.80 ± 0.62		a

*Statistical analysis performed using One-Way ANOVA. $p < 0.05$

Based on Table 1, the mean ± standard deviation (SD) of Superoxide Dismutase (SOD) enzyme levels in gastric tissue (U/mL) is shown for each treatment group. Statistical analysis using One-Way ANOVA yielded a p-value < 0.001, indicating a highly significant difference in SOD levels among the groups. Tukey's post hoc test was performed to identify which groups differed significantly. The results show that each group received a different letter notation (a–d), indicating meaningful differences between one group and another.

The normal control group (K1) showed the highest SOD level at 15.17 ± 0.61 U/mL, indicating optimal antioxidant activity in the gastric tissue. In contrast, the second group (K2) exhibited the lowest SOD level, 7.77 ± 0.49 U/mL, reflecting a decline in SOD enzyme activity due to increased oxidative stress. An increase in SOD levels was observed in groups K3 to K5, with values of 10.40 ± 0.44 U/mL, 12.07 ± 0.32 U/mL, and 13.80 ± 0.62 U/mL, respectively, demonstrating a protective effect of the administered treatment. These findings indicate that the treatments given to groups K3, K4, and K5 enhanced SOD activity compared to the untreated oxidative stress group (K2). This suggests the potential role of the treatment in reducing free radicals and improving the antioxidant defense system in gastric tissue.

The results further show that gastric Superoxide Dismutase (SOD) levels differed significantly among treatment groups. K1 exhibited the highest SOD level (15.17 ± 0.61 U/mL), while the cirrhosis group without treatment (K2) showed a marked decrease (7.77 ± 0.49 U/mL). This reflects an increase in oxidative stress due to liver cirrhosis, which reduces endogenous antioxidant capacity [21].

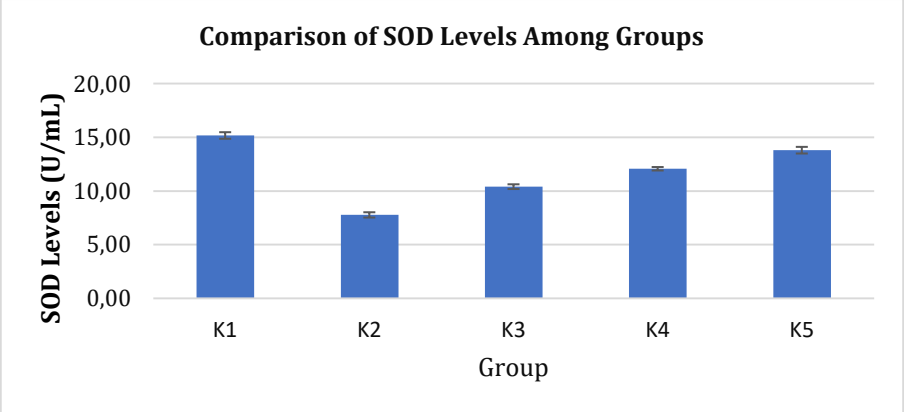

Figure 1. Comparison of SOD Levels Among Rat Groups

K1 (Normal rat group without induction), K2 (Rats in this group were induced with CCl_4 + coconut oil orally (p.o.) twice a week for 6 weeks, followed by administration of 0.5% CMC solution, 1 mL orally, during

weeks 7–8), K3 (Rats in this group were induced with CCl_4 + coconut oil orally (p.o.) twice a week for 6 weeks, followed by administration of snakehead fish extract at 100 mg/kg BW orally during weeks 7–8), K4 (Rats in this group were induced with CCl_4 + coconut oil orally (p.o.) twice a week for 6 weeks, followed by administration of snakehead fish extract at 150 mg/kg BW orally during weeks 7–8), K5 (Rats in this group were induced with CCl_4 + coconut oil orally (p.o.) twice a week for 6 weeks, followed by administration of snakehead fish extract at 200 mg/kg BW orally during weeks 7–8).

Overall, these findings indicate that snakehead fish extract provides a protective effect against gastric damage caused by liver cirrhosis and enhances the levels of the antioxidant enzyme SOD, with the most optimal effect observed at a dose of 200 mg/kg BW, which approaches normal conditions. The Kruskal–Wallis test for the variables of lesion type and degree of inflammation yielded $p > 0.05$, indicating no significant differences among groups. The minimum and maximum scores show that the cirrhosis group without treatment (K2) and the 100 mg/kg BW dose group (K3) experienced the most evident damage, with lesion and inflammation scores of 1–2, whereas the 200 mg/kg BW dose group (K5) exhibited the mildest damage. The data visualization demonstrates a dose–response relationship, in which increasing doses of snakehead fish extract correspond to a reduction in lesions and inflammation.

The administration of snakehead fish extract gradually increased SOD levels, with the highest dose of 200 mg/kg BW (K5) nearly matching the value observed in the K1 group. This finding is consistent with studies in other animal models reporting that snakehead fish extract can enhance antioxidant status and reduce MDA levels, a marker of lipid peroxidation and oxidative stress[9]. Snakehead fish extract has also been shown to improve overall antioxidant status and increase the activity of antioxidant enzymes such as SOD [22], [23].

The effects of snakehead fish (Channa striata) extract can be explained by its content of albumin and amino acids, such as glutamate[16]. Albumin possesses radical-scavenging properties and helps reduce oxidative burden [16], [24]. Glutamate serves as a primary precursor of glutathione, an endogenous antioxidant that works synergistically with the enzyme Superoxide Dismutase (SOD) to protect cells from damage [22], [25]. In addition to glutamate, several amino acids—including arginine and arginase—contribute to immune response mechanisms. Meanwhile, lysine, aspartic acid, and other amino acids function as antioxidants that act synergistically with fatty acids to maintain cellular oxidative balance[16]. Therefore, the increased SOD levels observed in groups K3, K4, and K5 indicate that snakehead fish extract acts as a natural antioxidant agent capable of effectively restoring redox homeostasis in the gastric tissue of cirrhotic rats.

In addition to changes in SOD levels, this study showed that the normal control group (K1) exhibited neither lesions nor inflammation in the gastric mucosa, whereas the cirrhotic group without treatment (K2) demonstrated mucosal damage characterized by higher lesion and inflammation scores. These findings are consistent with the mechanism of gastric hyperplastic pathology (GHP) in cirrhotic conditions, which is marked by increased oxidative stress contributing to tissue injury [26]. The following figure presents the histopathological features of the gastric tissue.

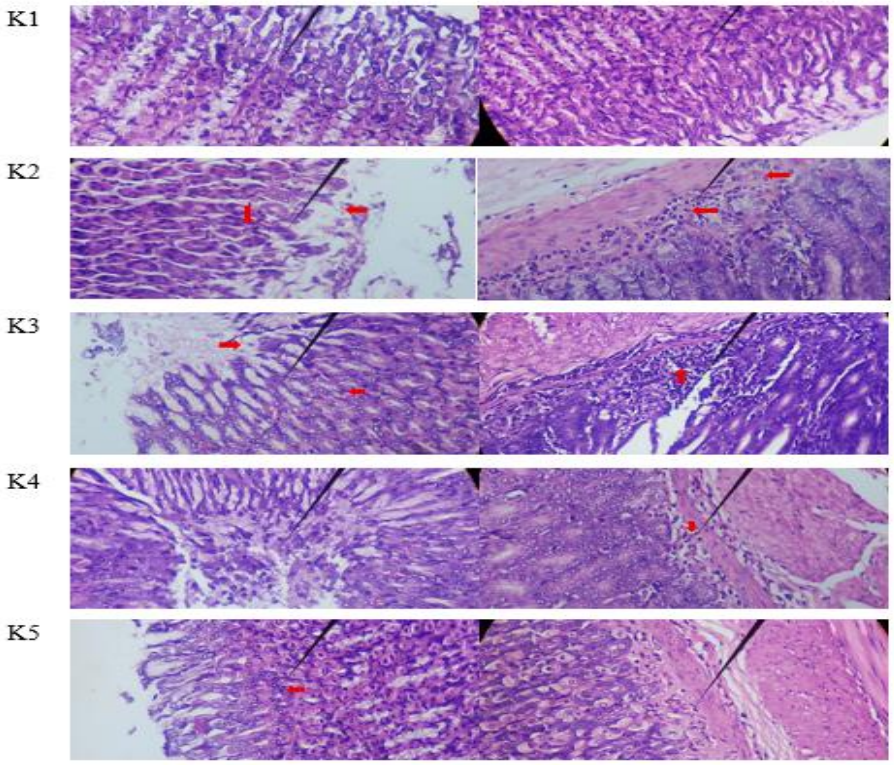

Figure 2. Histopathology of Gastric Tissue with HE Staining (10×40 Magnification)

This figure presents a comparative overview of the histological condition of the gastric mucosa across the treatment groups, showing variations in tissue damage from normal morphology to gradual epithelial improvement. Observed alterations include epithelial disorganization, inflammatory cell infiltration, edema, and dilation of capillaries and venules, with each panel (K1–K5) demonstrating different degrees of inflammation that reflect the progression and recovery of gastric tissue following treatment. K1 exhibits normal gastric mucosa, whereas K2 shows marked damage characterized by disorganized epithelium, dense inflammatory infiltration, edema, and vascular dilation. In K3, epithelial structure begins to recover but moderate inflammation remains, while K4 displays more organized epithelium with reduced infiltration and mild degeneration. K5 appears nearly normal with minimal inflammation and minor vascular dilation. To determine the effect of Snakehead fish (Channa striata) extract on gastric histopathology, the Kruskal–Wallis test was applied as a non-parametric method to assess differences in lesion type and inflammation severity across groups, with the results summarized in Table 2.

Table 2. Kruskal–Wallis Test Results for Gastric Histopathology

Variable	Kruskal-Wallis	p-Value
Types of Lesions	6.396	0.171*
Degree of Inflammation	4.375	0.358*

Based on the Kruskal–Wallis test results presented in Table 2, both the lesion type and inflammation grade variables showed *p-values > 0.05. This indicates that there were no statistically significant differences among the treatment groups. Nevertheless, the descriptive histological observations revealed progressive improvement in the gastric mucosal structure with increasing doses of Snakehead fish extract, suggesting a protective trend despite the absence of statistically significant differences.

Administration of Snakehead fish (Channa striata) extract in the cirrhosis groups demonstrated a clear dose–response relationship. The protective effect was not yet consistent at the lower dose of 100 mg/kg BW (K3), but a more stable reduction in inflammation was observed at 150 mg/kg BW (K4). The most optimal effect occurred at the 200 mg/kg BW dose (K5), where most samples showed no lesions or inflammation, resulting in a condition that closely resembled the normal group. This pattern indicates that higher doses of Snakehead fish extract confer greater protective effects on gastric mucosa damaged by cirrhosis. These findings align with the known anti-inflammatory properties of Snakehead fish and its documented ability to improve histological tissue structure in various inflammation models [23].

Channa striata exhibits suppressive effects on inflammatory mediators such as IL-1β, TNF-α, and IL-6 [16], [25]. Similar findings have been reported in other studies, which demonstrated that the protein and oil fractions of Snakehead fish can reduce inflammation and accelerate tissue regeneration through fibroblast stimulation and enhanced angiogenesis [27]. Therefore, the observed reduction in lesion and inflammation scores, accompanied by increased SOD levels, indicates that Snakehead fish extract exerts its effects through both antioxidant and anti-inflammatory mechanisms [28].

Overall, the findings of this study strengthen the evidence that Snakehead fish extract possesses potential as a gastroprotective agent. The protective effects demonstrated a clear dose–response pattern, with the 200 mg/kg BW dose providing the most optimal outcome, approaching normal physiological conditions. This result is consistent with international literature indicating that natural bioactive compounds play a crucial role in promoting tissue repair and reducing oxidative stress in liver-related diseases [29].

CONCLUSION

The findings of this study demonstrate that Snakehead fish extract effectively protects the gastric tissue through both antioxidant and anti-inflammatory mechanisms. This is indicated by the increased SOD levels accompanied by reduced lesions and inflammation in the gastric tissue of the experimental rats. The protective effect increased in a dose-dependent manner, with the most optimal outcome observed at 200 mg/kg BW, which closely approximated normal physiological conditions. These results highlight the role of bioactive albumin and amino acids in facilitating tissue repair and reducing oxidative stress.

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