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Hepatoprotective Effect of Kinar Leaf Juice on SGOT and SGPT Levels in a Preclinical Malaria Model

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Abstract
Malaria infection caused by *Plasmodium* parasites can induce live injury, characterized by elevated serum glutamic oxaloaceti transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT levels. Kinar leaves (*Kleinhovia hospita* L.) contain antioxidant bioactiv compounds with potential hepatoprotective properties. This laborator experimental study evaluated the effects of kinar leaf juice on SGOT an SGPT levels in *Plasmodium berghei* infected mice (*Mus musculus*) eighteen mice were divided into six groups: an infected untreate control, a quinine-treated group (0.037 mg/mouse/day), and thre treatment groups receiving kinar leaf juice at doses of 0.09, 0.18, an 0.36 mL/mouse/day for four consecutive days. SGOT and SGPT level were measured spectrophotometrically and analyzed using ANOV followed by Duncan's multiple range test ($P < 0.05$). Administration c kinar leaf juice reduces the number of *Plasmodium berghei* parasites Kinar leaf juice reduced SGOT levels to 29.3, 26.6, and 26.6 U/L corresponding to reductions of 41.4%, 46.8%, and 46.8% relative to th normal reference value. SGPT levels decreased to 25.0, 22.6, and 29.3 U/L, representing reductions of 9.09% and 17.82%, while the highes dose resulted in a value 6.55% above the normal reference. Thes findings suggest that kinar leaf juice has potential hepatoprotectiv effects against malaria-associated liver injury, although furthe biochemical and histopathological investigations are needed.

INTRODUCTION

Malaria remains a major global health problem, particularly in tropical regions like Indonesia (Ukratalo et al., 2023; Maatita et al., 2024; Ukratalo et al., 2025). Infection by the *Plasmodium parasite* not only causes hematological disorders but also affects various organs, including the liver (Kakisina & Ukratalo, 2011). The liver is the first organ infected in the early phase of the parasite's life cycle, thus playing a crucial role in malaria pathogenesis. Liver damage in malaria is characterized by impaired hepatocyte function due to inflammation, sinusoidal obstruction, and oxidative stress caused by the parasite infection (Khan et al., 2023).

Liver dysfunction in malaria is generally indicated by elevated transaminase enzymes, namely Serum Glutamic Oxaloacetic Transaminase (SGOT/AST) and Serum Glutamic Pyruvic Transaminase (SGPT/ALT) (Ukratalo, 2024). Elevated levels of these enzymes reflect liver cell damage due to leakage of hepatocyte membranes into the bloodstream. Several studies have shown that malaria patients experience significantly elevated SGOT and SGPT levels, even

reaching more than 10 times normal levels in certain conditions, making these parameters often used as indicators of disease severity (Cheaveau et al., 2019).

Furthermore, clinical studies show that approximately 69% of uncomplicated malaria cases have elevated liver enzymes, indicating that liver damage occurs not only in severe malaria but also in mild cases. This condition often receives less attention due to the focus on primary clinical symptoms such as fever and anemia. However, impaired liver function can worsen the prognosis and affect the metabolism of antimalarial drugs (Reuling et al., 2018; Dondorp et al., 2021).

Liver damage in malaria is largely triggered by oxidative stress resulting from increased free radical production during infection. Therefore, therapeutic approaches with antioxidant and hepatoprotective activity are crucial to prevent further damage to hepatocytes. Natural compounds from medicinal plants have potential as alternative therapies due to their relatively safe nature and diverse biological activities, including antioxidant and anti-inflammatory properties (Pangemanan et al., 2023; Kaihena et al., 2023; Loilatu et al., 2024; Hamsidar et al,, 2025; Kakisina et al., 2025).

One potential plant is the kinar leaf, which has been empirically used in traditional medicine and is thought to contain bioactive compounds such as flavonoids and polyphenols (Mainassy & Ukratalo, 2025). These compounds are known to ward off free radicals, stabilize cell membranes, and promote liver cell regeneration. However, to date, scientific research specifically evaluating the hepatoprotective effects of kinar leaf juice on malaria-induced liver damage, particularly using SGOT and SGPT parameters in preclinical models, is still very limited.

Based on these considerations, this study aims to evaluate changes in SGOT and SGPT levels following the administration of kinar leaf juice as biochemical indicators of liver function in a preclinical malaria mouse model. The findings are expected to provide scientific evidence regarding the potential of kinar leaves as a supportive therapeutic agent in mitigating malaria-induced liver damage, while also providing a foundation for the development of natural product-based phytopharmaceuticals.

RESEARCH METHODS

Research Design

This study employed a laboratory experimental design using a Completely Randomized Design (CRD), consisting of six treatment groups with three replicates per group (n = 3 per group). A total of 18 male mice (Mus musculus), aged 2–3 months and weighing approximately 20 g, were used as experimental animals and randomly assigned to six treatment groups, with three mice in each group.

Time and Location of the Study

This study was conducted from March to December 2025 at the Zoology Laboratory, Department of Biology, Faculty of Science and Technology, Pattimura University, Ambon.

Instruments and Materials

Tool Which used in study This are Erlenmeyer flasks, measuring cups, electronic scales, knives, mouse cages, analytical balances, syringes, surgical instruments (scalpels), tweezers, scissors, object glasses, microscopes, probes, volumetric pipettes, test tubes, purple-capped tubes (EDTA tubes or blood tubes), object glasses, and micropipettes. While the materials which used in This research uses kinar leaves (*Kleinhovia hospita L.), Plasmodium berghei* culture, mice (*Mus musculus*) aged 2-3 months weighing 20 grams, methanol, distilled water, aluminum foil, tissue, cotton, albumin, glycerin, and Giemsa.

Research Variables

The study variables consisted of independent and dependent variables. The independent variable was the administration of kinar leaf juice at three different doses: 0.09 mL, 0.18 mL, and 0.36 mL. The dependent variables were serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT) levels in mice with a preclinical malaria model.

Research Procedure

Stage Preparation

A total of 18 mice were randomly assigned to six treatment groups, with three mice in each group, to evaluate the percentage of parasitemia. Each group was housed in a plastic cage equipped with a wire-mesh cover and lined with rice husks. During the maintenance period, the mice were provided with food and drinking water ad libitum, while the cage environment was maintained in a clean and dry condition. Before the experimental treatments began, all mice were acclimatized to the laboratory environment and housing conditions for seven days.

Stages of Making a Malaria Model with Plasmodium berghei Injection

Inoculation of donor mice with *Plasmodium berghei* was performed via intraperitoneal injection at a volume of 200 µL (Ukratalo & Lateke, 2023). One day after inoculation, blood samples were collected from the donor mice to assess parasitemia. The inoculum was prepared using blood obtained from *P. berghei*-infected donor mice. Parasitemia levels were determined by examining thin blood smears stained with Giemsa under a microscope to calculate the percentage of infected erythrocytes relative to the total number of erythrocytes counted. Once parasitemia reached 20%, 0.2 mL of blood was collected from the donor mice and used as an inoculum to infect the experimental mice via intraperitoneal injection. Subsequently, parasitemia levels in the experimental mice were monitored daily. When parasitemia reached 2%, the mice in each treatment group were orally administered kinar leaf extract using an oral gavage needle at doses of 0.09 mL, 0.18 mL, and 0.36 mL for four consecutive days.

Stages of Making Kinar Leaf Juice

The method for making kinar leaf juice is as follows:

a. The freshest and greenest kinar leaves are chosen.
b. Wash the kinar leaves until clean and cut into small pieces.

c. To make 100% concentration: obtained from 100 grams of kinar leaves which are weighed and ground using a mortar.
d. The kinar leaves that have been ground are squeezed using three to five layers of gauze.
e. The juice that has been separated from the dregs is filtered again.
f. After being filtered, the juice that is clean of pulp is ready to be used by taking a dose of the juice using a syringe.
g. The dose taken is 100 ml and calculations are carried out for increasing and decreasing doses using a calculation method using a formula.
h. Then, the dose of squeezed kinar leaves is taken and ready to be used according to the calculated dose.

Stage Treatment

Administration of quinine medicine and squeezed quinar leaves is carried out for 4 days with the following treatment:

KN : Normal group that received no treatment
K+ : Group control positive, namely malaria model mice group
K- : Group control negative that is group of malaria model mice given quinine 0.037 mg/head/day for 4 days.
P1 : Group Malaria model mice were given kinar leaf juice at a dose of 0.09 ml/head/day for 4 days.
P2 : Group Malaria model mice were given kinar leaf juice at a dose of 0.18 ml/head/day for 4 days.
P3 : Group Malaria model mice were given kinar leaf juice at a dose of 0.36 ml/head/day for 4 days.

Observation of parasitemia

Parasitemia was assessed microscopically using thick and thin blood smears stained with Giemsa and examined under a light microscope at 1,000× magnification using an oil immersion objective. Thick blood smears were used to detect the presence of *Plasmodium berghei* parasites, as the erythrocytes undergo lysis during the staining process, allowing the parasites to become concentrated and more easily detectable. Meanwhile, thin blood smears were used to identify parasite morphology and species, as well as to determine parasitemia levels. The percentage of parasitemia was determined by counting the number of infected erythrocytes relative to the total number of erythrocytes examined, with approximately 1,000 erythrocytes counted in each thin blood smear. Parasitemia was subsequently calculated as the proportion of infected erythrocytes to the total number of erythrocytes examined, expressed as a percentage.

Measurement of SGOT and SGPT levels in mice blood

Blood samples were taken from mice using microhematocrit through the retroorbital plexus. Blood samples were placed in a test tube without anticoagulant to obtain serum. The test tube containing blood without anticoagulant was left for 60 minutes at room temperature.

Then it was centrifuged at 1500 rpm for 15 minutes. The clear liquid above the clotted blood cells was then taken with a micropipette and placed in an Eppendorf tube. Then, SGOT and SGPT levels were measured using a regen kit according to the photometric system method. The SGOT reagent consists of reagent I: TRIS, L-aspartate, malate dehydrogenase, lactate dehydrogenase and reagent II: 2-oxalogglutarate, NADH. The SGPT reagent consists of reagent I: TRISS, L-alanine, lactate dehydrogenase and reagent II: 2-oxalogglutarate, NADH.

SGOT levels were measured using a reagent kit (Siauta et al., 2021). Blank cuvettes were filled with 100 µL of distilled water and 1000 µL of reagent I, then incubated for 5 minutes at 37°C. Next, 250 µL of reagent II was added to each cuvette and incubated again for 1 minute at the same temperature. The absorbance value (optical density/OD) was measured using a spectrophotometer at a wavelength of 365 nm. The absorbance readings were taken three times with 1-minute intervals to obtain the Δ absorbance/minute value. This value was then multiplied by a conversion factor of 3971 to determine SGOT levels.

SGPT levels were measured using a reagent kit (Siauta et al., 2021). The first cuvette was used as a blank containing 100 µL of distilled water and 1000 µL of reagent I. The mixture was incubated for 5 minutes at 37°C. Next, 250 µL of reagent II was added to each cuvette and incubated for another minute at the same temperature. The absorbance value was measured using a spectrophotometer at a wavelength of 365 nm. The absorbance reading was taken three times with 1-minute intervals to obtain Δ absorbance/minute. This value was then multiplied by a conversion factor of 3971 to determine the SGPT level.

Calculation of SGOT and SGPT uses the following formula:

SGOT (U/L) = ΔAbs./min x 1746

SGPT (U/L) = ΔAbs./min x 1768

Description: Δ Abs is the change in absorbance measured during an enzyme assay. This measurement is important for assessing liver damage due to parasitic infection.

Data Analysis

The obtained SGOT and SGPT data were statistically analyzed using Analysis of Variance (ANOVA). When the ANOVA results indicated statistically significant differences, the analysis was followed by Duncan's Multiple Range Test (DMRT) at a significance level of $\alpha = 0.05$. All statistical analyses were performed using SAS software. SGOT and SGPT levels are presented as mean ± standard deviation (SD), with differences among treatments considered statistically significant at $p < 0.05$.

RESULTS AND DISCUSSION

Based on Table 1, administration of kinar leaf extract resulted in a reduction in parasitemia percentage in *Plasmodium berghei*-infected mice (*Mus musculus*) throughout the observation period. On day 4, the group of mice infected with *P. berghei* without treatment exhibited the highest parasitemia value (0.42 ± 0.01), whereas the quinine-treated control group showed a value of 0.22 ± 0.02. Administration of kinar leaf extract at doses of 0.09 mL, 0.18 mL, and 0.36 mL resulted in parasitemia values of 0.11 ± 0.01, 0.10 ± 0.01, and 0.07 ± 0.02, respectively. On day 6, parasitemia values in these treatment groups decreased further to 0.05 ± 0.01, 0.03 ±

0.01, and 0.01 ± 0.01, respectively. Meanwhile, the quinine-treated control group showed a parasitemia value of 0.13 ± 0.03, whereas the group of mice infected with *P. berghei* without treatment exhibited an increase in parasitemia from 0.42 ± 0.01 to 0.47 ± 0.02. Statistical analysis revealed significant differences among the treatment groups ($p < 0.05$).

Tabel 1. Reduction in Parasitemia (Mean ± SD) in Mice (*Mus musculus*) Infected with *Plasmodium berghei* Following Administration of Kinar Leaf Extract

Jumlah Parasitemia	Treatment (Mean ± SD)					
	Normal	*Plasmodium berghei*	Obat Kina	Kinar leaf extract		
				0.09 ml	0.18 ml	0.36 ml
The fourth day	0.00 ± 0.01^{e}	0.42 ± 0.01^{a}	0.22 ± 0.02^{b}	0.11 ± 0.01^{c}	0.10 ± 0.01^{cd}	0.07 ± 0.02^{d}
The six day	0.00 ± 0.01^{d}	0.47 ± 0.02^{a}	0.13 ± 0.03^{b}	0.05 ± 0.01^{c}	0.03 ± 0.01^{cd}	0.01 ± 0.01^{d}

Description: Numbers followed by different letters in the same row indicate a significant difference ($P<0.05$).

These findings indicate that increasing the dose of kinar leaf extract tends to enhance its ability to suppress parasite development. This activity is presumed to be associated with the secondary metabolites present in kinar leaves, such as flavonoids, alkaloids, tannins, and phenolic compounds, which may exhibit antiplasmodial activity by inhibiting parasite development and exerting antioxidant effects. A previous study on *Sida acuta* by Adesina et al. (2020) demonstrated that alkaloid, flavonoid, and phenolic extracts were able to reduce parasitemia in *Plasmodium berghei* infected mice (Adesina et al., 2020).

Changes in SGOT levels in mice (*Mus musculus*) infected with *Plasmodium berghei* showed a striking difference between the normal and infected groups. In the normal group, SGOT levels of 50.0 U/L reflected a stable physiological liver condition without any cell damage. However, after *Plasmodium berghei infection*, SGOT levels increased sharply to 168 U/L. This increase indicates hepatocellular damage due to parasite invasion in the liver stage, where the parasite develops in hepatocytes before entering the bloodstream. This process disrupts the structure and function of liver cells, resulting in the release of intracellular enzymes such as SGOT into the bloodstream. This is in line with research suggesting that malaria infection can trigger increased liver enzymes due to tissue damage and a systemic inflammatory response (Chughlay et al., 2020). Furthermore, the mechanism of *Plasmodium berghei infection* in the liver stage plays a crucial role in the occurrence of organ damage. Parasites are known to invade hepatocytes and develop within specialized vacuoles before being released back into the bloodstream, causing oxidative stress and cellular damage (Schmuckli-Maurer et al., 2024;Schepis et al., 2025). This condition strengthens the rationale for the increased SGOT levels in the infected group. Liver damage is not only caused by direct parasite invasion, but also by the body's immune response and the production of free radicals that exacerbate cell membrane damage. *Plasmodium berghei infection* can exacerbate liver damage through inflammatory mechanisms and the release of mediators that accelerate hepatocellular injury (Zhang et al., 2023 ; Prenen & Van den Steen, 2025).

Table 2. Average changes in blood SGOT levels (mean ± SD) in mice *(Mus musculus*) infected with *Plasmodium berghei*.

Parameters (U/L)	Treatment (Mean ± SD)					
	Normal	*Plasmodium berghei*	Quinine Medicine	Kinar leaf juice		
				0.09 ml	0.18 ml	0.36 ml
SGOT levels	50.0 ± 1.00 [c]	168 ± 3.46 [a]	30.6 ± 3.05 [b]	29.3 ± 2.30 [b]	26.6 ± 1.52 [b]	26.6 ± 3.05 [b]

Description: Numbers followed by different letters in the same row indicate a significant difference (P<0.05).

Quinine therapy significantly reduced SGOT levels to 30.6 U/L, even lower than the normal group. This decrease indicates that quinine is effective in inhibiting parasite growth and reducing liver tissue damage. Antimalarial drugs are known to work by inhibiting parasite metabolism, thereby reducing parasitemia and improving organ physiology. Recent studies have also emphasized the importance of using antimalarial agents in controlling parasite development and improving host health (Azmi et al., 2024).

More interesting results were seen in the treatment groups with quinine leaf juice (P1, P2, and P3), which showed SGOT levels of 29.3 U/L; 26.6 U/L; and 26.6 U/L, respectively. Based on the measurement results, SGOT levels of 29.3 U/L, 26.6 U/L, and 26.6 U/L corresponded to 58.6%, 53.2%, and 53.2% of the normal reference value of 50.0 U/L, respectively. These findings indicate that all SGOT values were below the established normal reference value, with relative reductions of 41.4%, 46.8%, and 46.8%, respectively. This suggests that SGOT levels in all three observations were lower than the reference value. These values were lower than those of the normal group and the group given only quinine. This indicates that quinine juice has a potentially stronger hepatoprotective effect, possibly due to its content of bioactive compounds such as alkaloids and antioxidants that can suppress oxidative stress and accelerate hepatocyte regeneration. A decrease in SGOT below normal levels can also be interpreted as an optimal recovery condition, where cell damage has been minimized and enzyme activity has stabilized (Odedra et al., 2022).

Malaria infection is known to trigger high oxidative stress due to increased production of reactive oxygen species (ROS), which can damage liver cell membranes. Antioxidant compounds in plants have the potential to neutralize ROS, thereby protecting hepatocytes from further damage. This is supported by research showing that natural antioxidant compounds can lower liver enzyme levels by reducing oxidative stress and inflammation (Chughlay et al., 2020). Furthermore, other studies have shown that the hepatoprotective activity of medicinal plants is closely related to their ability to enhance the endogenous antioxidant defense system (Apsari et al., 2024).

The quinine leaf juice (P1, P2, and P3) treatment resulted in SGOT levels of 29.3 U/L; 26.6 U/L; and 26.6 U/L, respectively. These values were lower than those of the normal group (50.0 U/L) and the quinine-only group (30.6 U/L), indicating a more optimal liver protection effect. Physiologically, the decrease in SGOT levels reflects a reduction in enzyme leakage from hepatocytes into the blood circulation, thus assuming that the integrity of the liver cell membrane is maintained. This condition indicates that the quinine leaf juice intervention is able to suppress the level of cell damage caused by malaria infection, which is generally known to

increase liver enzymes such as SGOT due to hepatocellular damage. This hepatoprotective effect is most likely related to the content of bioactive compounds in the quinine leaf juice, especially alkaloids such as quinine and antioxidant compounds (Ahmed et al., 2024). Malaria infection, including by *Plasmodium berghei*, is known to trigger oxidative stress by increasing the production of reactive oxygen species (ROS), which can damage cell membranes and hepatocyte organelles. Recent research suggests that an imbalance in the antioxidant system, such as decreased glutathione, plays a key role in malaria pathogenesis and tissue damage (Kotepui et al., 2023). Furthermore, another study reported that *Plasmodium berghei infection* can exacerbate liver damage through inflammatory mechanisms and the release of mediators that accelerate hepatocellular injury (Zhang et al., 2023). Therefore, the presence of antioxidant compounds in cinchona juice is thought to neutralize ROS, reduce inflammation, and protect hepatocytes from further damage.

Furthermore, the decrease in SGOT levels below normal values in groups P1, P2, and P3 can be interpreted as an indication of optimal liver function recovery. This indicates not only inhibition of cell damage but also the possibility of a more effective hepatocyte regeneration process. Several recent studies in mouse models infected with *Plasmodium berghei* have shown that administering plant-based compounds can improve liver antioxidant status and repair tissue damage through regenerative mechanisms (George-Opuda et al., 2023). In addition, phytotherapeutic interventions have also been reported to reduce parasitemia while improving immune and inflammatory responses in malaria-infected mice (Quadros Gomes et al., 2024 ; Zothantluanga et al., 2025). Thus, kinar leaf juice has the potential not only as an antimalarial agent but also as an effective hepatoprotector in supporting liver function recovery after infection.

The results showed that SGPT levels in the normal group were 27.5 U/L, then increased significantly in the group of mice infected with *Plasmodium berghei* to 75.3 U/L. This increase indicates liver cell (hepatocyte) damage, because SGPT is an enzyme that is more specific to the liver than SGOT. Malaria infection is known to cause impaired liver function through parasite invasion and an excessive immune response, resulting in the release of liver enzymes into the blood circulation. A recent study reported that in mice infected with *Plasmodium berghei*, there was an increase in liver enzymes, including ALT as an indicator of liver dysfunction due to infection and inflammation (Chuljerm et al., 2021). In addition, metabolic changes and immune responses in the liver during malaria infection also contribute to hepatic tissue damage (Hu et al., 2023).

Table 3. Average changes in blood SGPT levels (mean ± SD) in mice *(Mus musculus*) infected with *Plasmodium berghei*.

Parameters (U/L)	Treatment (Mean ± SD)					
	Normal	*Plasmodium berghei*	Quinine Medicine	Kinar leaf juice		
				0.09 ml	0.18 ml	0.36 ml
SGPT levels	27.5 ± 1.00 [b]	75.3 ± 3.05 [a]	36.3 ± 4.04 [b]	25.0 ± 1.00 [b]	22.6 ± 2.08 [b]	29.3 ± 2.51 [b]

Description: Numbers followed by different letters in the same row indicate a significant difference (P<0.05)

Quinine administration in the treatment group resulted in a decrease in SGPT levels to 36.3 U/L, approaching normal values. This indicates that quinine is effective in suppressing parasite development and reducing liver damage. Mechanistically, antimalarial drugs work by inhibiting parasite metabolism, thereby reducing parasitemia, which ultimately reduces oxidative stress and inflammation in liver tissue. Research shows that antimalarial therapy can reduce liver enzyme levels such as ALT and AST, which are previously elevated due to infection (Chuljerm et al., 2021). Therefore, the decrease in SGPT in this group reflects improved liver function following pharmacological intervention.

More optimal results were seen in the treatment groups with quinine leaf juice (P1, P2, and P3), with SGPT levels of 25.0 U/L; 22.6 U/L; and 29.3 U/L, respectively. Based on the measurement results, SGPT levels of 25.0 U/L, 22.6 U/L, and 29.3 U/L corresponded to 90.91%, 82.18%, and 106.55% of the normal reference value of 27.5 U/L, respectively. These findings indicate that two SGPT values were below the normal reference value, with relative reductions of 9.09% and 17.82%, respectively, whereas the SGPT level of 29.3 U/L was 6.55% above the normal reference value. These results indicate variations in SGPT levels relative to the established reference value. These values were not only lower than those in the infected group, but also partially below normal values. This condition indicates that quinine juice has a potentially stronger hepatoprotective effect. This is likely due to the content of bioactive compounds such as alkaloids and antioxidants that can suppress oxidative stress caused by malaria infection. *Plasmodium infection* is known to cause an imbalance in the antioxidant system, such as a decrease in glutathione, which plays a role in liver cell damage (Kotepui et al., 2023). Other studies have also shown that plant-based compounds can act as antiplasmodial (Hadi et al., 2025), by increasing the antioxidant defense system and reducing liver damage in mice infected with *Plasmodium berghei* (George-Opuda et al., 2023).

Overall, these data indicate that *Plasmodium berghei infection* significantly increases SGPT levels, an indicator of liver damage. While administration of quinine, and especially quinine juice, effectively reduces SGPT levels. A decrease to near or even below normal levels indicates optimal hepatocyte recovery and regeneration. Thus, quinine juice has potential not only as an antimalarial agent but also as a hepatoprotective agent, supporting liver function recovery after infection.

CONCLUSION

Based on the research results, it can be concluded that:

1. Administration of kinar leaf juice reduces the number of *Plasmodium berghei* parasites.
2. Administration of kinar leaf juice reduced SGOT levels to 29.3, 26.6, and 26.6 U/L at doses of 0.09, 0.18, and 0.36 mL/mouse/day, respectively. These values were 41.4%, 46.8%, and 46.8% lower than the normal reference value.
3. Kinar leaf juice reduced SGPT levels to 25.0, 22.6, and 29.3 U/L at doses of 0.09, 0.18, and 0.36 mL/mouse/day, respectively. The first two doses resulted in values 9.09% and 17.82% below the normal reference value, whereas the highest dose resulted in a value 6.55% above the reference value.

4. Overall, kinar leaf juice demonstrated potential hepatoprotective effects in *Plasmodium berghei*-infected mice, as indicated by changes in SGOT and SGPT levels. Further biochemical and histopathological studies are required to confirm its hepatoprotective activity and elucidate the underlying mechanisms.

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DECLARATIONS

Author Contributions

R.R.N. and A.J.A.U. designed the research methodology and conducted the experiment. R.R.N. was responsible for data processing and analysis. A.K. and L.E. contributed as core researchers in the study. E. S. contributed to the research execution, writing, and drafting of the manuscript. All authors have read and approved the final version of the manuscript.

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Declaration of Interest

The authors declare no conflict of interest.

Data Sharing Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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