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Effect of Acarbose on Blood Sugar Levels in Male White Rats (Rattus norvegicus) With Pathologies of Liver Dysfunction and Renal Dysfunction

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Abstract

Background of study: Diabetes is caused by metabolic disorders that result in high blood glucose levels due to insulin deficiency and insulin resistance. This disease can trigger serious psychological, social, and physical problems and increase the risk of kidney and liver disease. Insulin resistance exacerbates liver damage through hyperinsulinemia and oxidative stress, which also affects kidney function. Research on acarbose suggests its potential to reduce diabetes complications, including liver and kidney dysfunction.
Aims and scope of paper: This study aims to determine the effect of acarbose on blood glucose levels in diabetic rats with induced liver and kidney dysfunction using an in vivo experimental model.
Methods: This study is an in vivo experiment using a male white rat (Rattus norvegicus) as a subject. The study began with acclimatization of mice, followed by induction with aloxan, CCl4, and gentamicin, and observation for 7 days. After that, the mice were given acarbose suspension according to the test group, and blood samples were analyzed.
Result: The results showed that acarbose was effective in lowering blood sugar levels in diabetic and liver-dysfunction mice, but had no effect in kidney-dysfunction mice because there was no kidney damage.
Conclusion: acarbose significantly lowered blood sugar levels in mice with diabetes and liver dysfunction, but not in those with kidney dysfunction.

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INTRODUCTION

Diabetes mellitus is a metabolic disorder characterized by elevated blood glucose levels due to insulin deficiency, insulin resistance, or increased glucose levels during pregnancy (Hardianto, 2021). This condition can cause a variety of serious problems, including significant psychological, social, and physical impacts, as well as increasing the risk of complications such as kidney and liver disease (Setyoningsih et al., 2025). Chronically high blood glucose levels can damage the kidney's filtering units and blood arteries, potentially leading to diabetic nephropathy. If left untreated, diabetes can progress to chronic kidney disease and kidney failure (Yamazaki et al., 2021). In addition, insulin resistance is the leading cause of liver damage in people with diabetes, as this resistance contributes to hyperinsulinemia and oxidative stress that damages the liver (Suastika, 2021; Jdir et al., 2017). Chronic hyperinsulinemia, caused by decreased hepatic insulin clearance, worsens the condition of diabetes by increasing levels of anti-insulin hormones such as glucagon, growth hormone, and cytokines (Marchisello et al., 2019). Diabetes mellitus can gradually affect the function of vital organs, particularly the kidneys and the liver. Persistent high blood glucose levels disrupt normal metabolic processes, leading to increased strain on these organs. Over time, this condition may result in decreased organ function and a higher risk of long-term complications.

Proper management of diabetes is essential to maintain organ health and improve the overall quality of life of individuals with diabetes (Banday et al., 2020).

Acarbose, an alpha-glucosidase inhibitor, has been identified as a potential agent to address diabetes complications by slowing carbohydrate digestion and intestinal carbohydrate absorption (Altay, 2022). Previous research has shown that acarbose is effective in managing diabetes complications, including liver and kidney dysfunction, without increasing the risk of organ damage (Chao et al., 2018). Acarbose has been widely used in the management of diabetes, particularly for controlling postprandial hyperglycemia. Several studies have reported that acarbose may contribute to improved metabolic profiles, including better glycemic stability and reduced oxidative stress, which are closely associated with the progression of diabetic complications. In addition, acarbose has been suggested to exert indirect protective effects on metabolic organs by reducing glucose fluctuations that exacerbate cellular injury. These characteristics make acarbose a relevant therapeutic agent to be further investigated in diabetic conditions complicated by liver and kidney dysfunction (Yousefi et al., 2023; Zamani et al., 2023)

Therefore, further research is needed to evaluate the effectiveness of acarbose in reducing blood glucose levels in diabetic conditions accompanied by organ dysfunction, particularly of the liver and kidneys. Considering the crucial roles of the liver and kidneys in glucose metabolism and drug clearance, understanding the effects of acarbose under conditions of impaired function of these organs is essential to ensure therapeutic safety and efficacy. This *in vivo* study is expected to provide a more comprehensive understanding of the potential of acarbose in the management of diabetes with organ complications and to serve as a scientific basis for the development of safer and more appropriate therapeutic strategies.

METHOD

This study used an in vivo experimental method with male white rats (Rattus norvegicus) as test subjects. The research process began with acclimatization of mice, followed by induction with aloxane, CCl4, and gentamicin to induce the desired conditions. After a 7-day observation period, the mice were divided into test groups and given acarbose suspensions as assigned. Blood samples are taken for analysis to evaluate the effects of the treatment. The tools used in the study included analytical scales (Ohaus®), animal scales (Sf-400), measuring cups (Pyrex®), glucometers (Easy Touch®), and photometers (Clinical Chemistry®) for measuring glucose levels and diabetes complications in mice. In addition, rat cages, lumpang and pestles, oral sondes, capillary pipes, surgical scissors, one cc and three cc syringes (Onemed®), watch glasses, and other general laboratory equipment from the STIKES Harapan Ibu Jambi Pharmacology Laboratory were also used. The ingredients used included male white rat (Rattus norvegicus), acarbose 50 mg, Na CMC 0.5%, aquadest (Amidis), aloxan, CCl4 (Merck), injectable gentamicin, alcohol swab, wood husk, as well as food and beverage for rats.

RESULTS AND DISCUSSION

Result

Table 1. Blood Sugar Level

Group	Average Blood Sugar Level (mg/dl) ± SD			
	Day 0	Day 1	Day 3	Day 5
Kel 1	109±6.65	101 ± 11.01	87 ± 11.59	102 ± 7.50
Kel 2	451±35.92	502±44.79	448±91.03	365±107.28
Kel 3	412±144.35	461±46.45	333±49.32	257±122.93
Kel 4	290±54.16	261±53.29	284±37.80	188±52.55
Kel 5	321±118.87	239±99.63	188±105.40	113±12.01

Table 2. SGPT and Creatinine

Group	SGPT (mg/dl)	Creatinine (mg/dl)
Group 1 (Negative)	21 ± 3.05	0.6 ± 0.17
Group 2 (Aloksan + CCl4)	80 ± 21.65	0.9 ± 0.37
Group 3 (Aloksan + CCl4 + Acarbose)	44 ± 10.06	1.6 ± 1.55
Group 4 (Aloksan + CCl4 + Gentamisin)	99 ± 18.52	1.4 ± 1.44
Group 5 (Aloksan + CCl4 + Gentamisin + Acarbose)	88 ± 19.56	0.4± 0.23

Discussion

The study showed a significant difference in blood sugar levels during the 5 days of alloxan administration. This could occur because of physiological differences among rats, allowing alloxan to act more effectively (Fitrianita & Musir, 2018). Aloxan works by attacking pancreatic beta cells that produce insulin. Once the allocator enters the beta cell via the glucose transporter GLUT2, it triggers the formation of oxygen-free radicals that damage essential cellular components, including the cell's membranes, proteins, and DNA. This damage leads to beta cell death, resulting in decreased insulin production and secretion (Hasim et al., 2020) a condition classified as hyperglycemia when blood sugar levels exceed 135 mg/dl (Jiwintarum et al., 2017).

In the measurement of blood sugar levels in the diabetes and liver dysfunction groups, SGPT levels were previously measured in test animals. Blood samples were collected via orbital sinus (eye) puncture, then centrifuged at 3000 rpm for 15 minutes. SGPT levels were measured using a clinical photometer. The normal range of SGPT levels is 17.5-30.2 U/L (Daipadli et al., 2024). The results showed that the average SGPT level in the treatment group increased above normal (> 30.2 U/L), except in the negative group, which received only Na CMC. The administration of CCl4 was induced for 1 day via intraperitoneal injection; after 1 day, SGPT levels increased to 103.30 U/L. Continuous use of CCl4 can damage the liver because it is a hepatotoxic substance (Sengupta et al., 2011). So that there is an increase in SGPT levels caused by extrahepatic conditions beyond the researchers' control (Sari et al., 2025).

In the measurement of blood sugar levels in the diabetes and kidney dysfunction group, creatinine levels were previously measured. In a test to measure creatinine levels, rats treated with gentamicin received an intraperitoneal dose of 80 mg/kg for 7 days (Mitra et al., 2021). intraperitoneally to damage his kidneys. The average result on the fifth day of the treatment group showed kidney damage, with creatinine levels above the normal range (>0.8 mg/dl). Only the treated diabetes, liver, and kidney groups did not experience kidney damage or had creatinine levels below the normal range (<0.4 mg/dl). Where normal creatinine levels in mice are 0.2-0.8 mg/dl (Tandi et al., 2020), this is due to researchers' errors in inducing gentamicin, so that creatinine levels do not rise or the kidneys are not damaged. During induction with gentamicin and aloxan, a phenomenon occurs in rats. Death in rats can also be caused by gentamicin-induced inflammation; gentamicin-induced kidney damage can lead to the formation of cast tubules (kidney stones in the drains), damage to the tubule cell layer, bleeding in the cells, and inflammation of the cells (Muthmainnah et al., 2015). However, the possible cause is the interaction between aloxan and gentamicin, but no research has been conducted on this interaction.

Implications

The findings of this study provide important implications for the use of acarbose in diabetic conditions accompanied by organ dysfunction. The results indicate that acarbose effectively reduced blood glucose levels in diabetic rats with liver dysfunction, suggesting that its antihyperglycemic activity remains preserved despite hepatic impairment. This supports the potential clinical relevance of acarbose as a therapeutic option for diabetic patients with concurrent liver dysfunction (Papazafiropoulou & Melidonis, 2019). However, the absence of a blood glucose lowering effect in the kidney dysfunction group highlights the importance of confirming organ pathology when evaluating pharmacological efficacy. These findings emphasize that the effectiveness of antidiabetic agents should be interpreted in relation to the integrity of target organs involved in glucose

metabolism and drug elimination (Masiani et al., 2024). Overall, this study underscores the need for careful experimental validation when assessing antidiabetic therapies under conditions of multi-organ involvement.

Research Contribution

This study contributes to the existing body of knowledge by experimentally evaluating the effect of acarbose in diabetic conditions complicated by liver and kidney dysfunction using an in vivo rat model. Unlike previous studies that mainly focused on the general antidiabetic effects of acarbose, this research specifically examines its performance under organ impaired conditions. The study provides evidence that acarbose can significantly reduce blood glucose levels in diabetic rats with liver dysfunction, while also revealing the limitations of its observed effect in the kidney dysfunction model due to unsuccessful induction. These findings add to the understanding of acarbose's pharmacological behavior in complex pathological states and offer preliminary data that may inform future preclinical and clinical investigations involving diabetes with organ complications.

Limitations

Several limitations should be acknowledged in this study. First, the induction of kidney dysfunction using gentamicin was not consistently successful, as indicated by creatinine levels that remained within or below the normal range in some treatment groups. This limitation restricts the interpretation of acarbose's effect in diabetic rats with confirmed renal impairment (Masiani et al., 2024). Second, the study did not include histopathological examination of liver and kidney tissues, which would have provided stronger confirmation of organ damage and treatment effects. Third, the sample size and short duration of observation may limit the generalizability of the findings and the ability to detect long-term effects of acarbose administration. Finally, potential interactions between alloxan and gentamicin were not explored, which may have influenced the outcomes of organ induction (Salehi et al., 2020).

Suggestions

Future studies are recommended to optimize and validate kidney dysfunction induction protocols to ensure consistent renal damage prior to treatment evaluation. Incorporating histopathological analysis alongside biochemical markers such as SGPT and creatinine would strengthen the assessment of organ injury and therapeutic effects (Fagbohun et al., 2020). Further research should also explore different doses and durations of acarbose administration to evaluate dose response relationships under organ-impaired conditions. In addition, investigating potential interactions between diabetogenic agents and nephrotoxic compounds would be valuable to improve experimental accuracy. Expanding sample size and extending observation periods may also enhance the reliability and translational relevance of future findings (Saputra et al., 2018).

CONCLUSION

Based on research using data on results, it can be concluded that acarbose did not affect lowering blood sugar levels in diabetic rats with liver dysfunction pathology, compared to the diabetic group with kidney dysfunction.

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AUTHOR CONTRIBUTION STATEMENT

Aisa Dinda Mitra designed the research and calculated blood sugar levels; Sry Nur Admida calculated creatinine and SGPT levels; and Rizky Yulion designed the research and calculated blood sugar, SGPT, and creatinine levels.

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