

Role of maslinic acid in ischemia-reperfusion-induced testicular injury in rats

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ABSTRACT

BACKGROUND: The pathophysiology of testicular ischemia-reperfusion is characterized by a marked increase in reactive oxygen species. Oxidative damage caused by reactive oxygen species to cellular components, including DNA, proteins and lipids, leads to injury of spermatogenic cells. Maslinic acid, a bioactive compound found in *Olea europaea*, hawthorn, and other medicinal plants, exhibits antioxidant properties. This study aimed to determine whether maslinic acid protects testicular sperm production following ischemia-reperfusion injury in a rat model.

METHODS: Male rats were randomly assigned to three groups: a control group (Group 1), an ischemia-reperfusion group (Group 2), and an ischemia-reperfusion + maslinic acid group (Group 3). Ischemia was induced in the left testis by two-hour torsion, followed by reperfusion via surgical detorsion. The treatment group received intraperitoneal administration of maslinic acid at the onset of detorsion procedure. Following detorsion, left orchietomy was performed at either four hours or three months. To comprehensively assess testicular oxidative stress and function, we measured key indicators: malondialdehyde concentration (reflecting reactive oxygen species levels); activities of superoxide dismutase and catalase, representing components of the cellular antioxidant system; and overall spermatogenic efficiency. These parameters were evaluated using biochemical assays and histological analysis with hematoxylin-eosin staining.

RESULTS: Testicular ischemia-reperfusion significantly increased malondialdehyde levels while suppressing key antioxidant defenses (superoxide dismutase and catalase) and impairing spermatogenic function ($p < 0.001$). Despite testicular damage induced by ischemia-reperfusion, maslinic acid treatment produced a partial restoration of these markers ($p < 0.01$).

CONCLUSION: In summary, maslinic acid mitigates ischemia-reperfusion-induced testicular injury by enhancing superoxide dismutase and catalase activities while reducing reactive oxygen species.

Keywords: Maslinic acid; ischemia-reperfusion; testicular torsion.

INTRODUCTION

Testicular torsion, defined as the rotation of the spermatic cord, is one of the most urgent emergencies in urology. Its estimated prevalence is approximately 1 in 4,000 males by the age of 25.^[1] Torsion compromises blood flow, depriving the testis of oxygen and resulting in ischemia. A critical period of

approximately six hours is available for diagnosis and surgical detorsion,^[2] during which restoration of blood flow can prevent irreversible testicular damage. Despite prompt intervention, testicular atrophy occurs in a substantial proportion of patients, with reported rates ranging from 25% to 60.2%.^[3,4] The pathogenesis of testicular ischemia-reperfusion injury is complex and multifactorial, involving the initial ischemic

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event, reperfusion-induced inflammation, oxidative stress, microvascular alterations, and multiple downstream mechanisms of injury. Among these, oxidative stress acts as a key driver of testicular damage.^[5] This condition is characterized by a marked increase in reactive oxygen species, including hydrogen peroxide, hydroxyl radicals, and superoxide anions.^[5] Reactive oxygen species attack cellular components, including DNA, proteins, and lipids, leading to damage of spermatogenic cells.^[5]

Currently, no effective pharmacological treatments are available for testicular ischemia-reperfusion injury in clinical practice. Maslinic acid, a bioactive compound found in *Olea europaea*, hawthorn, and other medicinal plants, has attracted attention due to its diverse biological activities.^[6-8] It has a molecular formula of C₃₀H₄₈O₄ and a molecular weight of 472.7 g/mol.^[7] Extensive pharmacological studies have demonstrated its potent antioxidant and anti-inflammatory effects.^[8,9] Maslinic acid has been shown to protect various organs from ischemia-reperfusion injury, including the kidney, brain, and heart.^[10-16] However, its potential protective role in testicular ischemia-reperfusion injury remains unexplored. In the present study, we employed a rat model of testicular ischemia-reperfusion to evaluate the therapeutic potential of maslinic acid by assessing oxidative stress markers and histopathological changes.

MATERIALS AND METHODS

Experimental Subjects and Ethical Considerations

Male Sprague–Dawley rats (250–300 g) were obtained from SLAC (Shanghai, China). Animals were housed under controlled conditions with a temperature of 20–22°C, relative humidity of 50–60%, and a 12-hour light/dark cycle. Standard laboratory chow and water were provided ad libitum. All experimental procedures were conducted in accordance with institutional ethical guidelines and were approved by the Ethics Committee at Zhejiang Shuren University (Date: August 16, 2024; Approval no: 20240816-01).

Experimental Design and Induction of Testicular Ischemia-Reperfusion

Rats were randomly assigned to three groups: control (Group 1, n=20), testicular ischemia-reperfusion (Group 2, n=20), and testicular ischemia-reperfusion plus maslinic acid treatment (Group 3, n=20). General anesthesia was induced with ketamine (50 mg/kg, intraperitoneally; Sigma-Aldrich, Saint Louis, MO, USA). Before surgery, the left ilioinguinal and scrotal regions were shaved and disinfected with 10% povidone-iodine solution. The left testis was exposed via a left ilioinguinal incision in all groups. In the control group, an 11/0 noninvasive suture was passed through the tunica albuginea, and the testis was returned to the scrotum without performing torsion-detorsion.^[17] The incision was closed with 4/0 silk sutures. Testicular ischemia-reperfusion was induced by rotating the left testis 720° counterclockwise along

the spermatic cord axis.^[17] Ischemia was maintained for two hours by fixing the rotated testis to the scrotal wall using an 11/0 noninvasive suture to prevent spontaneous detorsion. Following the ischemic interval, testicular blood flow was restored by detorsion and repositioning of the testis. At the onset of reperfusion, the maslinic acid-treated group received maslinic acid (20 mg/kg, intraperitoneally; Sigma-Aldrich). The selected dose was based on previous studies demonstrating its efficacy against ischemia-reperfusion injury in multiple organs, particularly the kidney and heart.^[10-14] To assess the acute-phase response, 10 rats from each group were sacrificed four hours after reperfusion. Left testicular tissues were promptly excised for the analysis of malondialdehyde levels and the activities of superoxide dismutase and catalase. The remaining animals (n=10 per group) were sacrificed at three months post-reperfusion. Left testicular tissues were collected to evaluate spermatogenic function.

Measurement of Malondialdehyde Levels in Testicular Tissue

Testicular tissue (100 mg) was rapidly homogenized in 1 mL of malondialdehyde lysis buffer at 4°C. The homogenate was centrifuged at 5,000 × g for 15 minutes at 4°C, and the supernatant was collected for analysis. Malondialdehyde levels were measured according to manufacturer's instructions using a kit from Jiancheng Bioengineering Institute (Nanjing, China).

Determination of Superoxide Dismutase and Catalase Activities

Testicular tissue was homogenized in normal saline at 4°C and centrifuged at 2,000 × g for 10 minutes at 4°C. The resulting supernatant was used to determine superoxide dismutase and catalase activities according to manufacturer's protocols (Jiancheng Bioengineering Institute, Nanjing, China).

Evaluation of Spermatogenic Function

Spermatogenic function was assessed based on seminiferous tubule diameter, Johnsen score, testicular weight, and seminiferous epithelium layer number.^[18] After harvesting and weighing the rat testes, tissue samples were fixed in Bouin's solution for four hours for pathological evaluation. The testicular tissue was dehydrated using a series of progressively concentrated ethanol solutions. Xylene was then used to clear the specimens prior to paraffin embedding. Microtome sectioning of the paraffin-embedded blocks produced sections of 5 micrometers in thickness. The sections were dewaxed with xylene, hydrated through graded alcohols, and stained with hematoxylin and eosin (Sigma-Aldrich) for routine histopathological examination. For each section, a blinded pathologist randomly selected 20 seminiferous tubules, which were examined under a light microscope at 200× magnification. The diameter of the seminiferous tubules was measured using a microscope eyepiece fitted with a micrometer. Testicular spermatogenesis was evaluated using the 10-point Johnsen scoring system.^[19] The lowest score (1) indicates seminiferous tubules lacking epithelial cells,^[19] whereas the highest

score (10) represents complete and orderly spermatogenesis with abundant spermatozoa and a patent lumen.^[19] The stratification of the seminiferous epithelium was determined by counting the number of cell layers from the basement membrane to the lumen.

Statistical Analysis

Statistical analysis of all biochemical and histopathological data was performed using GraphPad Prism 4 (San Diego, CA, USA). The Shapiro–Wilk test confirmed that the data were normally distributed. Data are presented as mean ± standard deviation. Comparisons among the three experimental groups were performed using one-way analysis of variance, followed by the Student–Newman–Keuls post hoc test. A *p* value <0.05 was considered statistically significant.

RESULTS

Maslinic Acid Reduced Malondialdehyde Levels in Testicular Tissue Following Ischemia-Reperfusion Injury

As shown in Figure 1, malondialdehyde levels were significantly higher (*p*<0.001) in the ischemia-reperfusion group compared with the control group. Treatment with maslinic acid significantly decreased malondialdehyde levels (*p*<0.001) compared with the untreated ischemia-reperfusion group.

Maslinic Acid Increased Superoxide Dismutase and Catalase Activities in Testicular Tissue Following Ischemia-Reperfusion Injury

As illustrated in Figure 2, the activities of the antioxidant enzymes superoxide dismutase and catalase were significantly lower (*p*<0.001) in the ischemia-reperfusion group than in the control group. Statistical analysis revealed that maslinic acid treatment significantly restored the activities of super-

oxide dismutase and catalase (*p*<0.001) in testicular tissue affected by ischemia-reperfusion injury.

Maslinic Acid Improved Spermatogenic Function Following Ischemia-Reperfusion Injury

Data revealed a pronounced impairment in spermatogenic function following testicular ischemia-reperfusion (Figs. 3, 4), characterized by significantly reduced (*p*<0.001) seminiferous tubule diameter, Johnsen score, testicular weight, and seminiferous epithelium layer number compared with the control group. Maslinic acid treatment demonstrated a significant ameliorating effect on testicular spermatogenic function (seminiferous tubule diameter: *p*<0.01; Johnsen score, testicular weight, and seminiferous epithelium layer number: *p*<0.001), partially counteracting the adverse effects of ischemia-reperfusion.

DISCUSSION

The primary concern in testicular torsion is the need for urgent treatment (detorsion) to restore testicular perfusion and prevent irreversible infarction. A strong negative correlation exists between time to detorsion and testicular survival rates. Clinically, these rates are approximately 90–100% and 50% for interventions performed within 4–8 hours and 12–24 hours, respectively.^[20] Despite successful detorsion, postoperative testicular atrophy has been reported in 25% to 60.2% of cases.^[3,4] The long-term consequences of two-hour testicular torsion, as demonstrated in our study, were evident three months after detorsion. Testicular impairment included significant reductions in seminiferous tubule diameter, Johnsen score, testicular weight, and seminiferous epithelium layer number (Figs. 3, 4).

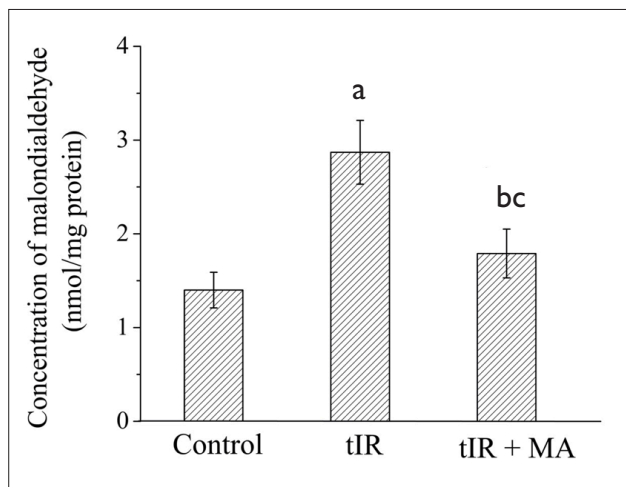


Figure 1. Maslinic acid (MA) administration reduced malondialdehyde levels in the testes. Malondialdehyde concentrations in testicular tissue were measured using a biochemical assay in control, testicular ischemia-reperfusion (tIR), and MA-treated groups. Data are presented as mean ± standard deviation (n=10). a: *p*<0.001 vs. control group; b: *p*<0.01 vs. control group; c: *p*<0.001 vs. tIR group.

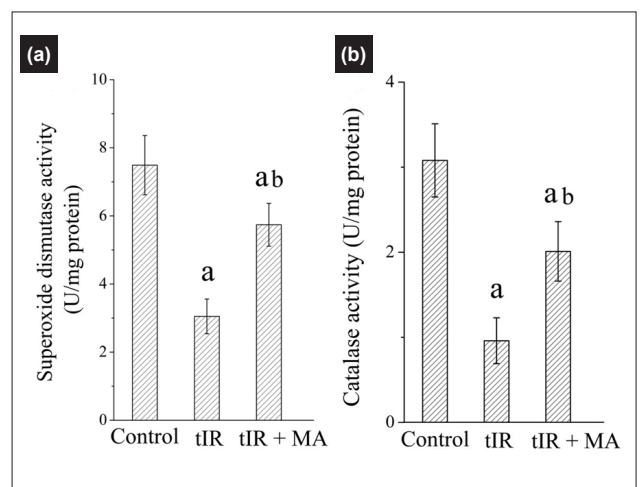


Figure 2. Maslinic acid (MA) administration increased the activities of (a) superoxide dismutase and (b) catalase in the testes. Enzyme activities were measured using biochemical assays in control, testicular ischemia-reperfusion (tIR), and MA-treated groups. Data are presented as mean ± standard deviation (n=10). a: *p*<0.001 vs. control group; b: *p*<0.001 vs. tIR group.

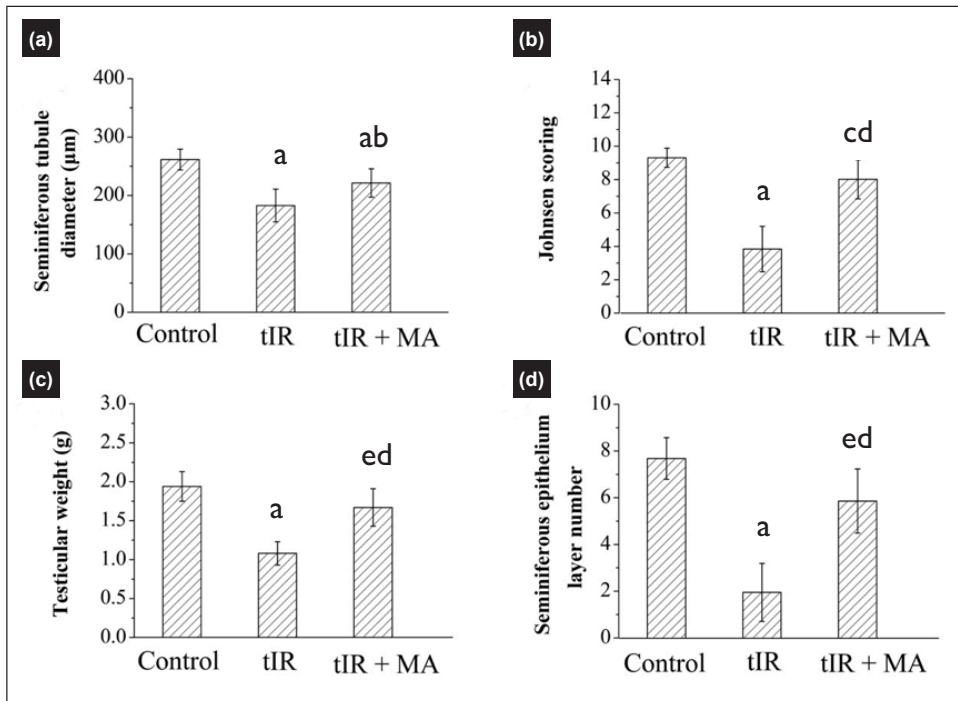


Figure 3. Maslinic acid (MA) administration improved spermatogenic function, as reflected by (a) seminiferous tubule diameter, (b) Johnsen score, (c) testicular weight, and (d) seminiferous epithelium layer number. These parameters were evaluated through histomorphological analysis in control, testicular ischemia-reperfusion (tIR), and MA-treated groups. Data are presented as mean \pm standard deviation (n=10). a: $p < 0.001$ vs. control group; b: $p < 0.01$ vs. tIR group; c: $p < 0.05$ vs. control group; d: $p < 0.001$ vs. tIR group; e: $p < 0.01$ vs. control group.

Reactive oxygen species (ROS) production increases following detorsion of a torsed testicle.^[21] The oxidative modification of proteins, lipids, and DNA by reactive oxygen species represents a major mechanism of damage in spermatogenic cells.^[5] Direct measurement of reactive oxygen species in testicular tissue is challenging due to their high reactivity and short lifespan.^[17] Reactive oxygen species induce cellular membrane damage through lipid peroxidation.^[22] Malondialdehyde, a byproduct of lipid peroxidation, is therefore widely used as a marker of reactive oxygen species.^[23] In the present study, ischemia-reperfusion significantly increased malondialdehyde levels and was associated with impaired spermatogenesis (Figs. 1, 3, 4). These findings suggest that excessive reactive oxygen species generation during ischemia contributes to spermatogenic dysfunction. Maslinic acid administration significantly reduced testicular malondialdehyde levels and improved spermatogenic function compared with the ischemia-reperfusion group (Figs. 1, 3, 4). These results indicate that maslinic acid mitigates oxidative stress following testicular ischemia-reperfusion, thereby promoting recovery of spermatogenesis. Maslinic acid has been reported to be safe and beneficial in clinical settings, including in elderly women with knee osteoarthritis and in water polo athletes experiencing exercise-induced fatigue and muscle soreness.^[24,25] Therefore, maslinic acid may represent a promising therapeutic candi-

date for testicular ischemia-reperfusion injury. However, the mechanism by which maslinic acid mitigates oxidative stress has yet to be determined.

For normal cell function to be maintained, a balance must exist between the production of reactive oxygen species and their elimination.^[26] Superoxide dismutase and catalase are key components of the intracellular antioxidant defense system that protect cells from reactive oxygen species.^[27] Superoxide dismutase catalyzes the dismutation of superoxide anions into hydrogen peroxide, which is subsequently decomposed into water and molecular oxygen by catalase.^[28,29] In pathological conditions such as ischemia-reperfusion, increased reactive oxygen species production leads to oxidative damage that overwhelms endogenous antioxidant defenses, resulting in cellular injury and death.^[30,31] In the present study, testicular ischemia-reperfusion disrupted this balance, as evidenced by increased malondialdehyde levels and decreased activities of superoxide dismutase and catalase, along with impaired spermatogenesis (Figs. 1-4). These findings support that excessive reactive oxygen species production during testicular ischemia-reperfusion depletes antioxidant defenses, thereby contributing to spermatogenic dysfunction. Data from Figures 1-4 indicate that maslinic acid treatment increased superoxide dismutase and catalase activities, reduced malondialdehyde levels, and improved spermatogenic func-

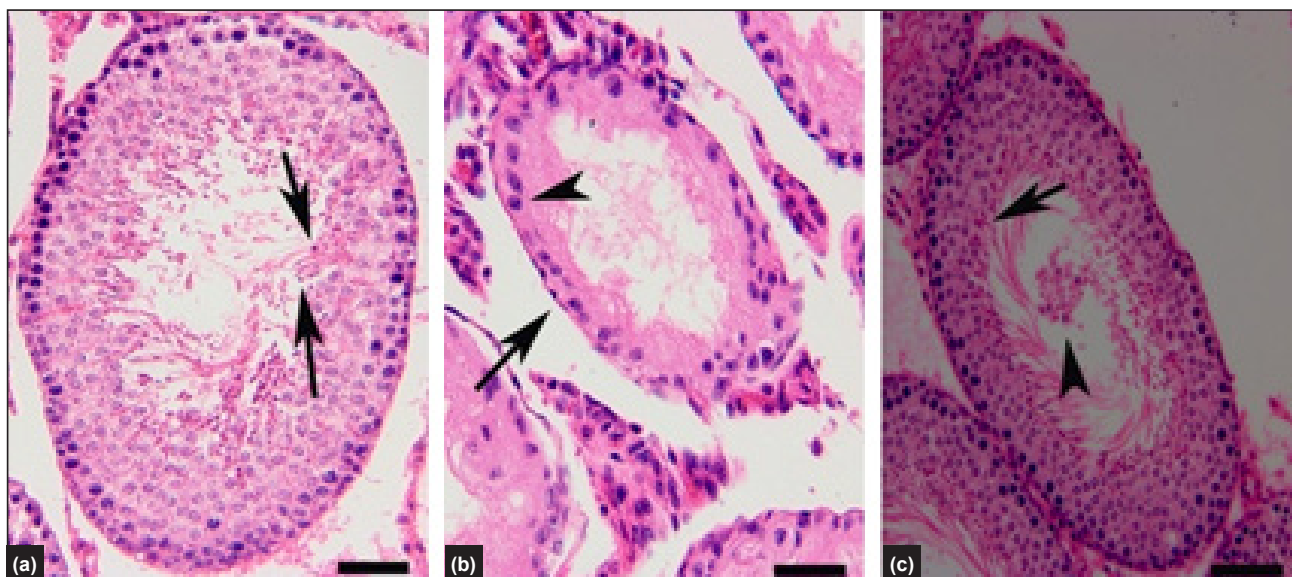


Figure 4. Representative hematoxylin and eosin-stained sections of testicular tissue. **(a)** Control group shows normal architecture, with well-organized epithelial layers containing numerous sperm (↑). The tubules exhibit clear, unobstructed lumina and normal diameter. **(b)** Following ischemia-reperfusion injury, the testes display substantial damage to the seminiferous tubules (↑), characterized by thinning of the epithelial lining (▲), absence of sperm, and a marked reduction in cross-sectional diameter. **(c)** Maslinic acid treatment restores the architecture and function of the seminiferous tubules to a state closely resembling that of the control group, as evidenced by complete spermatogenesis (presence of sperm, ↑), a comparable number of epithelial layers, and similar tubule diameter. However, exfoliated epithelial cells (▲) are observed within the tubular lumen, posing a potential risk of tubular blockage. Images were captured at 200× magnification. Scale bar = 40 micrometers.

tion in testicular tissue subjected to ischemia-reperfusion. These results suggest that maslinic acid alleviates oxidative stress by enhancing the activities of superoxide dismutase and catalase, thereby improving spermatogenic function.

Our study did not evaluate dose-dependent effects of maslinic acid in testicular ischemia-reperfusion injury. Therefore, the optimal therapeutic dose remains to be determined in future studies.

To evaluate spermatogenesis in the testes, hematoxylin and eosin-stained tissue sections were examined in this study. Histopathological evaluation was based on key parameters, including seminiferous tubule diameter, Johnsen score, and seminiferous epithelium layer number. These criteria are widely used in studies of testicular ischemia-reperfusion injury due to their effectiveness in reflecting tissue damage.^[1,23] Additional markers, including germ cell apoptosis (assessed by methods such as the Terminal deoxynucleotidyl transferase dUTP nick end labeling (TUNEL) assay), integrity of the tubular basement membrane, interstitial edema, inflammatory cell infiltration, and ultrastructural alterations, also serve as valuable indicators of testicular injury. We plan to incorporate these markers into future studies.

Using an experimental model of testicular torsion-detorsion, Karli et al.^[32] evaluated the effects of medical ozone and hyperbaric oxygen treatment. Compared with the ischemia-reperfusion group, both treatments significantly reduced malon-

dialdehyde levels, indicating decreased oxidative damage. Additionally, both groups showed significantly higher glutathione peroxidase activity, an antioxidant enzyme, compared with the ischemia-reperfusion group. Compared with both the ischemia-reperfusion and medical ozone groups, hyperbaric oxygen administration resulted in significantly increased superoxide dismutase activity. Consequently, hyperbaric oxygen demonstrated a stronger antioxidant effect than medical ozone, particularly with respect to superoxide dismutase activity. Histopathological assessment of the testicles was performed using the Cosentino scoring system. Although both medical ozone therapy and hyperbaric oxygen treatment were effective in reducing testicular ischemia-reperfusion injury, they demonstrated comparable results, with no significant difference between their effects. These findings suggest that both therapies may serve as antioxidant treatments for testicular torsion. Beyond malondialdehyde and superoxide dismutase, the study by Karli et al.^[32] differed from ours in terms of other evaluated parameters and pharmacological interventions. In future studies, we will incorporate measurements of glutathione peroxidase and the Cosentino score, and compare the efficacy of maslinic acid, medical ozone, and hyperbaric oxygen in the treatment of testicular ischemia-reperfusion injury to determine the most effective agent.

CONCLUSION

The role of maslinic acid in alleviating testicular ischemia-re-

perfusion injury has not been previously investigated, and this study aimed to address this gap. Our findings demonstrate that maslinic acid treatment improves spermatogenesis following ischemia-reperfusion injury. Its protective effects are mediated by antioxidant mechanisms, including increased superoxide dismutase and catalase activities and reduced reactive oxygen species levels. These findings suggest that maslinic acid may represent a promising therapeutic option for testicular damage caused by ischemia-reperfusion. However, further studies are required to confirm its tolerability and clinical efficacy before translation into clinical practice.

Ethics Committee Approval: This study was approved by the Ethics Committee at Zhejiang Shuren University (Date: 16.08.2024, Decision No: 20240816-01).

Authorship Contributions: Concept: S.M.W., Y.M.H.; Design: S.M.W., Y.M.H.; Supervision: S.M.W., Y.M.H.; Resource: S.M.W.; Materials: S.M.W.; Data collection and/or processing: S.M.W., Y.M.H.; Analysis and/or interpretation: S.M.W., Y.M.H.; Literature review: S.M.W., Y.M.H.; Writing: S.M.W., Y.M.H.; Critical review: S.M.W., Y.M.H.

Informed Consent: Not applicable, as this study was conducted on experimental animals and did not involve human participants.

Conflict of Interest: None declared.

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DENEYSEL ÇALIŞMA - ÖZ

Sıçanlarda iskemi-reperfüzyonun neden olduğu testis hasarında maslinik asidin rolü

AMAÇ: Testis iskemi-reperfüzyonunun patofizyolojisi, reaktif oksijen türlerinde belirgin bir artışla karakterize edilir. Reaktif oksijen türlerinin DNA, proteinler ve lipitler dahil olmak üzere hücrel bileşenlere verdiği oksidatif hasar, spermatogenik hücrelerin hasar görmesine yol açar. *Olea europaea*, alıç ve diğer şifalı bitkilerde bulunan biyoaktif bir bileşik olan maslinik asit, antioksidan özellikler sergiler. Bu çalışma, maslinik asidin sıçan modelinde iskemi-reperfüzyon hasarı sonrası testis sperm üretimini koruyup korumadığını belirlemeyi amaçlamıştır.

GEREÇ VE YÖNTEM: Erkek sıçanlar rastgele üç gruba ayrılmıştır: Bir kontrol grubu (Grup 1), bir iskemi-reperfüzyon grubu (Grup 2) ve bir iskemi-reperfüzyon + maslinik asit grubu (Grup 3). İskemi, sol testiste iki saatlik torsiyonla induklendi, ardından cerrahi detorsiyon yoluyla reperfüzyon sağlandı. Tedavi grubuna, detorsiyon prosedürünün başlangıcında intraperitoneal maslinik asit uygulandı. Detorsiyonun ardından, dört saat veya üç ay sonra sol orşiektomi gerçekleştirildi. Testislerdeki oksidatif stresi ve fonksiyonu kapsamlı bir şekilde değerlendirmek için, temel göstergeleri ölçtük: malondialdehit konsantrasyonu (reaktif oksijen türlerinin seviyelerini yansıtır); hücrel antioksidan sistemin bileşenlerini temsil eden süperoksit dismutaz ve katalaz aktiviteleri; ve genel spermatogenik verimlilik. Bu parametreler, biyokimyasal testler ve hematoksilen-eozin boyaması ile histolojik analiz kullanılarak değerlendirildi.

BULGULAR: Testis iskemi-reperfüzyonu, malondialdehit düzeylerini önemli ölçüde artırırken, temel antioksidan savunma mekanizmalarını (süperoksit dismutaz ve katalaz) baskıladı ve spermatogenez fonksiyonunu bozdu ($p<0.001$). İskemi-reperfüzyonun neden olduğu testis hasarına rağmen, maslinik asit tedavisi bu belirteçlerde kısmi bir düzelmeye sağladı ($p<0.01$).

SONUÇ: Özetle, maslinik asit, reaktif oksijen türlerini azaltırken süperoksit dismutaz ve katalaz aktivitelerini artırarak iskemi-reperfüzyonun neden olduğu testis hasarını hafifletmektedir.

Anahtar sözcükler: Maslinik asit; iskemi-reperfüzyon; testis torsiyonu.

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