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Effect of Myricetin on Some Biochemical Parameters in Hemolytic Anemic Male Rats

Article Info.

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Abstract

Hemolytic anemia (HA) is a group of diseases characterized by the accelerated destruction of red blood cells (RBCs), resulting in various clinical manifestations such as anemia and jaundice. This study aimed to investigate the ameliorative effects of Myricetin on phenylhydrazine (PHZ)-induced hemolytic anemia in adult male rats. Forty rats were randomly divided into four groups. Group I received normal saline orally. Group II consisted of anemic rats administered normal saline orally. Group III included anemic rats treated with Myricetin (1 mg/kg body weight, i.p, once daily), and Group IV comprised anemic rats treated with folic acid (1 mg/kg body weight, orally, once daily). All treatments were continued for 30 consecutive days. Serum samples were collected on days 7, 15, and 30 and analyzed using biochemistry analyzer to assess hematological, inflammatory biomarkers. The results showed a significant, time-dependent improvement in key hematological and inflammatory parameters, including Ferritin, Serum Iron, Lactate dehydrogenase (LDH), C-Reactive Protein (CRP), and Indirect bilirubin. Collectively, these findings suggest that Myricetin may exert antioxidant, anti-inflammatory effects , that contribute to hematological improvement. This highlights its potential as a natural therapeutic candidate for the management of hemolytic anemia.

Keyword: Myricetin, Hemolytic anemia, Rats.

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Introduction

Hemolytic anemia (HA) is a group of diseases characterized by the accelerated destruction of red blood cells (RBCs), resulting in various clinical manifestations such as anemia and jaundice (1). In severe cases, hemolytic anemia may become life threatening. Extensive hemolysis reduces red blood cell count and hemoglobin levels, leading to impaired oxygen delivery (2,3). It is classified based on the origin of hemolysis-whether intracapsular due to intrinsic red cell pathology or extracorporeal from external factors (4,5,6). The etiology of this condition is diverse, including genetic abnormalities, autoimmune mechanisms, medications, or toxins (7,8) Laboratory investigations commonly reveal elevated levels of reticulocytes (RET) and lactate dehydrogenase (LDH) and decreased haptoglobin (9). RBCs are endowed with antioxidant enzymes to prevent HGB from being oxidized (10) Hemolysis compromises the protective enzymatic shield, leading to the release of free hemoglobin (FHb) into the bloodstream. This disruption initiates a cascade of oxidative reactions, producing reactive oxygen species (ROS) including superoxide radicals, hydrogen peroxide, and hydroxyl radicals, which have the potential to induce vascular oxidative stress and tissue injury (11,12). The release of FHb and iron following hemolysis into circulation, increasing ROS levels and resulting in systemic iron dysregulation, thereby worsening disease prognosis and increasing mortality (13,14).

Myricetin falls under the group of flavanols along with quercetin, kaempferol, and isorhamnetin and is mainly found in the Myricaceae, Anacardiaceae, Polygonaceae, Pinaceae, and Primulaceae families and from consumables such as vegetables, fruits, and tea. It has shown potent activity against free radicals even at low concentrations compared to other flavonoids (15). It is extracted as a solid form, appears yellow in color, has a molecular weight of about 318.23 g/mol, and has six hydrogen bond donors. Myricetin is a natural flavonoid with potent antioxidant and anti-inflammatory activities. Several studies have demonstrated its protective effects against oxidative stress-related disorders. (16).

Folate is a water-soluble vitamin essential for cell replication and DNA synthesis, repair, and methylation. Folate has been implicated in the pathophysiology of several human diseases, including anemia, thromboembolia, cardiovascular disease, neurologic diseases, and cancer (17,18).

PHZ has long been used to produce animal models of hemolytic anemia. The PHZ mechanism of hemolytic action was related to its interaction with RBCs. This interaction produces hydrogen peroxide and destroys the hemoglobin pigment through the formation of oxidized derivatives and free radicals of hydrazine (19). PHZ also induces Reactive Oxygen Species (ROS) formation, lipid peroxidation, and protein oxidation due to the reaction with the plasma membrane. Afterwards, the oxidative degradation of spectrin in the membrane cytoskeleton will result in hemolytic anemia (20). Based on reports, PHZ may cause oxidative damage to the liver, too. The formation of destructive free radicals during the microsomal oxidation of hydrazines is correlated with hepatotoxicity of hydrazine derivatives (21).

Materials and Methods

Animals: Forty Albino Wistar male rats, 12 weeks old, weighing 200-250g were used. The rats were housed in the animal house, College of Veterinary Medicine, University of Basrah, for a two-week acclimatization. The rats were housed under standard laboratory conditions, including a controlled temperature at 22±2°C and a fixed light/dark cycle (12 hours light/12 hours dark), with continuous access to food and water to ensure their well-being throughout the study.

Induction of Anemia: Thirty adult male rats were induced to anemia by administering 40 mg\kg of phenylhydrazine intraperitoneally for two days in a row. Before starting treatment, blood samples from the animals were taken before and after the injection for hematological analysis to assess the induction of anemia. Animals treated with phenylhydrazine whose hemoglobin concentration is below 11 g\dL are considered anemic (22-23).

Experiment design: A total of forty male rats were randomly allocated into four experimental groups, each comprising ten animals. Group 1 received normal saline orally daily. Group 2 consisted of anemic rats that were given normal saline orally. Group 3 included anemic rats treated with myricetin at a dose of 1 mg/kg body weight, administered intraperitoneally once daily for 30 days (24). Group 4 comprised anemic rats that received folic acid at a dose of 1 mg/kg body weight orally for 30 consecutive days (25).

Blood collection: Blood was collected from the lateral tail vein (26) in a gel tube at days 7, 15 and 30 of experiment and centrifuged for evaluation of biochemical parameters.

Parameters analysis: The biochemistry analyzer was used to measure Ferritin, Serum Iron, LDH, CRP, and Indirect bilirubin. All kits were from Spinreact™ company \ Spain, and performed with the Spin120™ company \ China.

Statistical analysis*:* Data were analyzed using SPSS version 22, two-way ANOVA was applied to evaluate the effects of treatments and time. Statistical significance was set at $p < 0.05$.

Results

Table 1 showed biochemical results of Ferritin, Serum Iron, LDH, CRP, and Indirect bilirubin for all study groups and in 7, 15, and 30 days of the experiment. For Ferritin, PHZ administration significantly increased serum ferritin levels in the anemic group compared with the control group ($p < 0.05$). Treatment with myricetin or folic acid resulted in a significant reduction in ferritin levels at day 30 compared with the untreated anemic group. In serum Iron results were significantly elevated in the PHZ- induced anemic group compared with controls ($p < 0.05$). Myricetin treatment significantly reduced serum iron levels from day 7 till the end of the experiment (30 days) compared with anemic group.

For LDH, the results showed a significant increase ($p < 0.05$) in anemia group compared with the control group. Both the myricetin group and the folic acid group significantly reduced LDH levels compared with the untreated anemic group, although values remained higher than those of the control, No significant differences ($P<0.05$) in serum CRP levels were observed among the control (anemic, myricetin and folic acid groups).

Finally, indirect bilirubin results showed a significant decrease ($p < 0.05$) on day 7 of the study in the anemia, myricetin, and folic acid groups compared to the control group, followed by a significant increase ($p < 0.05$) at days 15 and 30 for both the anemia and myricetin groups, while levels in the folic acid-treated group remained significantly lower.

Discussion

PHZ induces oxidative damage to erythrocytes by reacting with hemoglobin and leading to membrane damage and rapid hemolysis. Hemolysis liberates heme/iron into plasma and tissues, perturbing systemic iron handling; as a rapid protective response, cells and the liver upregulate ferritin to sequester labile iron and limit Fenton chemistry, so serum and tissue ferritin often rise after PHZ-induced hemolysis. This mechanistic sequence may explain the elevated ferritin levels observed in the PHZ-induced anemic group in the present study. So Myricetin: a polyhydroxy flavanol with potent antioxidant, anti-inflammatory and reported iron-modulating properties. By scavenging ROS, inhibiting inflammatory signaling and (in some models) limiting iron-driven tissue staining/accumulation, myricetin may reduce the oxidative stimulus that contributes to ferritin up-regulation and may help maintain erythrocyte integrity- thereby contributing to gradual reduction in ferritin levels over time. Recent reviews and in vivo work support myricetin's ability to blunt oxidative, inflammatory and iron-related pathology which agrees with (27).

As mention earlier PHZ oxidatively damages RBCs, increases hemoglobin breakdown and iron release, and triggers compensatory erythropoiesis and hepcidin/erythroferrone axis changes that can transiently increase circulating iron this may explain the elevated serum-iron levels observed in anemic rats. Myricetin may limit oxidative erythrocyte damage and modulate systemic iron regulation - a dual action that explains early (day 7) normalization by its effect as antioxidant or cytoprotective effect. Myricetin is a potent flavonoid antioxidant and anti-inflammatory agent; by reducing oxidative membrane damage to RBCs, it can lower ongoing hemolysis, so less hemoglobin/iron is liberated into plasma, several experimental studies show myricetin suppresses hepatic hepcidin expression (BMP/SMAD pathway modulation) and alters iron distribution (reducing splenic iron, influencing serum iron and erythropoiesis). In a hemolytic context, this can speed restoration of iron homeostasis and RBC recovery. The combination of reduced ongoing release (less hemolysis) plus improved regulation of iron handling can produce measurable serum-iron improvement within about a week, all this matching with (28).

Table 1: Ferritin, Serum Iron, LDH, CRP, and Indirect Bilirubin evaluation for serum samples in 7,15, and 30 days of experiment

Parameter	Period In Days	Groups				Time Mean
		Control	Anemia	Myricetin	Folic Acid	
Ferritin µg/L	7	10.158 ±0.367	11.676 ±1.200	11.168 ±1.059	11.468 ±1.228	11.117 a ±1.126
	15	10.216 ±0.254	11.831 ±1.188	10.801 ±1.044	11.111 ±1.202	10.990 a ±1.107
	30	10.325 ±0.470	11.961 ±1.200	10.248 ±1.083	10.860 ±1.328	10.848 a ±1.220
Group Mean		10.233 c ±0.358	11.823 a ±1.130	10.739 bc ±1.071	11.146 bc ±1.205	
Serum Iron µg/dL	7	91.83 ±0.753	288.00 ±3.033	96.33 ±3.559	107.67 ±7.421	145.96 a ±84.079
	15	91.67 ±1.211	267.67 ±6.055	92.83 ±2.858	102.67 ±5.502	138.71 b ±76.290
	30	92.00 ±1.673	248.33 ±7.474	87.50 ±2.739	99.67 ±5.086	131.88 c ±68.972
Group Mean		91.83 c ±1.200	268.00 a ±17.540	92.22 c ±4.722	103.33 b ±6.651	
LDH U/L	7	1719.67 ±234.809	2390.00 ±123.194	2581.83 ±203.157	2236.83 ±120.184	2232.08 ±366.492
	15	1723.17 ±240.610	2391.50 ±121.558	2198.33 ±121.908	2128.33 ±100.671	2110.33 ±288.207
	30	1726.67 ±237.018	2365.17 ±110.177	2026.33 ±217.515	2105.33 ±102.804	2055.88 ±285.531
Group Mean		1723.17 c ±223.103	2382.22 a ±111.957	2268.83 a ±295.825	2156.83 b ±117.555	
CRP mg/L	7	0.371 ±0.021	0.388 ±0.044	0.385 ±0.025	0.430 ±0.112	0.393 a ±0.062
	15	0.380 ±0.020	0.418 ±0.037	0.368 ±0.020	0.400 ±0.095	0.391 a ±0.053
	30	0.373 ±0.018	0.405 ±0.037	0.333 ±0.020	0.380 ±0.123	0.372 b ±0.066
Group Mean		0.375 ab ±0.019	0.403 a ±0.039	0.362 b ±0.030	0.403 a ±0.106	
Indirect Bilirubin mg/dL	7	0.950 ±0.025	0.670 ±0.016	0.713 ±0.150	0.567 ±0.034	0.383 a ±0.323
	15	0.103 ±0.008	0.660 ±0.033	0.620 ±0.193	0.060 ±0.022	0.360 a ±0.300
	30	0.101 ±0.017	0.675 ±0.016	0.695 ±0.043	0.053 ±0.025	0.381 a ±0.311
Group Mean		0.100 b ±0.017	0.668 a ±0.023	0.676 a ±0.141	0.056 b ±0.026	

Data are presented as mean ± SD (n = 10). Different small letters denote significant differences between groups and time ($p<0.05$).

Serum LDH rose significantly in the PHZ-treated anemia group, consistent with acute hemolysis and intracellular enzyme release described in PHZ models and clinical hemolytic states. LDH is a well-established, sensitive marker of hemolysis and tissue injury, although it is not specific and should be interpreted together with bilirubin, haptoglobin and reticulocyte data. The partial but significant reduction of LDH in both the myricetin- and folic-acid–treated groups (vs. the untreated anemia group) suggests that the treatments attenuated PHZ-induced erythrocyte membrane damage and cell lysis rather than fully preventing it. Myricetin's effect is biologically plausible: multiple recent studies and reviews report that myricetin has potent antioxidant and anti-inflammatory actions (including activation of Nrf2 and suppression of oxidative lipid and protein damage), mechanisms that protect erythrocytes from oxidant-mediated hemolysis and can reduce LDH release. Folic acid likely acted indirectly by supporting erythropoiesis and accelerating recovery of circulating red cell mass, which over time lowers hemolytic biomarkers; however, folate itself is not primarily a membrane-stabilizing antioxidant. Because LDH elevation is nonspecific, the observed reductions in LDH should be read alongside the full panel of hemolysis markers and bone-marrow/erythropoietic indices to conclude the extent and mechanism of protection. Overall, our data indicate that myricetin provides measurable protection against PHZ-induced erythrocyte injury and that folic acid accelerates hematologic recovery, both producing statistically significant decreases in serum LDH compared with untreated anemia (29) and (30).

In the PHZ-induced hemolytic anemia model, serum C-reactive protein (CRP) did not differ significantly between control, anemia, myricetin and folic acid groups. This finding is compatible with the biology of CRP in rats: unlike in humans, where CRP is a major acute-phase reactant that can rise hundreds-to-thousands-fold, CRP in rats is a moderate/minor acute-phase protein with relatively high basal concentrations and only modest (≈two-fold or less) increases after injury or inflammation. Because of this species difference, small or transient inflammatory signals may not produce statistically detectable CRP changes in standard rat experiments (31), so both myricetin (a flavonoid with well-documented antioxidant and anti-inflammatory properties) and folic acid have been reported to modulate inflammatory markers in other settings, but effects on CRP are context-dependent. Recent comprehensive reviews and experimental work show myricetin reduces oxidative stress and inflammatory signaling in multiple models, yet short-term rodent studies do not always translate into measurable reductions in circulating CRP because of assay sensitivity and species-specific acute phase dynamics. Similarly, clinical meta-analyses find folic-acid supplementation can lower CRP in humans under some conditions, but those human findings cannot be directly extrapolated to a short PHZ rat protocol. Therefore, the lack of significant changes in CRP levels does not contradict known anti-inflammatory actions of myricetin or folic acid — it likely reflects the model, timing and biomarker chosen (27) and (32).

Indirect (unconjugated) bilirubin showed an unexpected transient decline on day 7 across the PHZ, myricetin and folic-acid groups, followed by later increases in the PHZ and myricetin groups at days 15 and 30, whereas folic-acid-treated animals maintained lower levels. PHZ models produce an acute hemolytic insult followed by compensatory erythropoiesis; the temporal course of

bilirubin depends both on the balance of hemoglobin breakdown and hepatic conjugation/clearance capacity. Early decreases in UCB might reflect transient up-regulation of hepatic uptake/conjugation or assay/volume effects, whereas later increases likely reflect ongoing hemolytic turnover overwhelming clearance. Myricetin's known antioxidant and hepatoprotective actions could explain the initial suppression of UCB but may be insufficient to prevent later bilirubin accumulation if hemolytic drive persists; conversely, folic acid treatment may have contributed to reduced bilirubin over time. These interpretations are consistent with recent PHZ animal model work and current preclinical literature on myricetin and folate in hemolytic states, but confirmatory measures (total/direct bilirubin, LDH, haptoglobin, reticulocyte counts, and hepatic function) are required to distinguish altered production from altered clearance (33) and (34).

Conclusion

Based on the presented biochemical findings, phenylhydrazine (PHZ)–induced hemolytic anemia markedly disrupted iron metabolism and hemolysis-related markers in male rats, as evidenced by increased ferritin, serum iron, and LDH levels, while CRP remained unchanged, indicating minimal systemic inflammation. Treatment with myricetin significantly ameliorated these alterations by reducing serum iron, ferritin, and LDH levels throughout the experimental period, reflecting its protective role against hemolysis and iron overload. Although indirect bilirubin increased at later stages in anemic and myricetin-treated groups, myricetin showed partial modulation compared with untreated anemia. Overall, myricetin demonstrated comparable, though not identical, efficacy to folic acid, suggesting its potential as a supportive therapeutic agent in managing biochemical disturbances associated with hemolytic anemia.

Conflicts of interest

The authors declare that there is no conflict of interest.

Ethical Clearance

This work is approved by The Research Ethical Committee.

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تأثير الميريسيتين على بعض المؤشرات البيوكيميائية في ذكور الجرذان المصابة بفقر الدم الانحلالي

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الخلاصة

فقر الدم الانحلالي هو مجموعة من الأمراض التي تتميز بتدمير خلايا الدم الحمراء بشكل متسارع، مما يؤدي إلى ظهور أعراض سريرية متنوعة مثل فقر الدم واليرقان. هدفت هذه الدراسة إلى التحقق من التأثيرات المُحسِّنة للميريسيتين على فقر الدم الانحلالي المُستحث بالفينيل هيدرازين في ذكور الجرذان البالغة. تم تقسيم أربعين جرذًا عشوائيًا إلى أربع مجموعات. تلقت المجموعة الأولى محلول ملحي عادي عن طريق الفم. ضمت المجموعة الثانية جرذانًا مصابة بفقر الدم تلقت محلولًا ملحيًا عاديًا عن طريق الفم. شملت المجموعة الثالثة جرذانًا مصابة بفقر الدم عولجت بالميريسيتين (1 ملغم/كغم من وزن الجسم، عن طريق الحقن داخل الصفاق، مرة واحدة يوميًا). ضمت المجموعة الرابعة جرذانًا مصابة بفقر الدم عولجت بحمض الفوليك (1 ملغم/كغم من وزن الجسم، عن طريق الفم، مرة واحدة يوميًا). استمرت جميع العلاجات لمدة 30 يومًا متتالية. جُمعت عينات من مصل الدم في الأيام 7 و15 و30، وحُللت باستخدام محلل الكيمياء الحيوية لتقييم المؤشرات الحيوية الدموية والالتهابية. أظهرت النتائج تحسناً ملحوظاً ومتناسباً مع الوقت في مؤشرات الدم والالتهاب الرئيسية، بما في ذلك الفيريتين، وحديد المصل، وإنزيم نازعة هيدروجين اللاكتات(LDH) ، والبروتين المتفاعل(CRP) C ، والبيليروبين غير المباشر. تشير هذه النتائج مجتمعةً إلى أن الميريسيتين قد يمتلك خصائص مضادة للأكسدة والالتهاب، مما يساهم في تحسين حالة الدم. وهذا يُبرز إمكاناته كعلاج طبيعي محتمل لإدارة فقر الدم الانحلالي.

الكلمات المفتاحية :ميريسيتين، فقر الدم الانحلالي، الجرذان.