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Effects of *Musa cavendish* on Selected Physiological Parameters and Haematological Changes in Isoproterenol-Induced Myocardial Infarction in Wistar rats

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ABSTRACT

Myocardial infarction (MI) is characterized by necrosis of heart muscle cells. Risk factors of MI include hypertension, diabetes mellitus and obesity which are derangements in blood pressure, blood glucose and body weight respectively. Haematological changes play a significant role in the pathophysiology and management of MI. *Musa cavendish* (MC) is known to exhibit some characteristics which may be protective in MI. The aim of this study was to investigate the effects of MC on selected physiological parameters and deranged haematological indices in isoproterenol (ISP) induced MI in Wistar rats. This study employed an experimental design. Aqueous extraction of MC was carried out. 60 rats were randomly assigned into ten groups (n=6). The groups were Normal control which received distilled water; MC only (100, 200 and 400 mg/kg respectively); MI control (ISP 85 mg/kg); Positive control (ISP + Propranolol 0.5 mg/kg); Aqueous extract of MC (ISP +100, 200 and 400 mg/kg). Blood samples were collected for biochemical analyses. Data analysis utilized one way ANOVA and tukey's post hoc test with significance set at $p<0.05$. MC did not cause any significant change in blood pressure, blood glucose and heart rate of healthy animals. There was a decrease in total white blood cells, neutrophils, lymphocytes, monocytes and platelet count and an increase in haemoglobin concentration after 4 weeks of treatment, when compared to control. MC thus reverses ISP induced derangements in PCV, Haemoglobin concentration, platelets, total white blood cell counts, neutrophils, lymphocytes and monocytes.

Keywords: Myocardial infarction, *Musa cavendish*, Risk Factors, Haematology, Platelet Count, Aqueous Extraction

Introduction

In sub-Saharan Africa, cardiovascular disease (CVD) remains the most prevalent cause of non-communicable disease (NCD) related deaths, with approximately 13% of all deaths and 37% of NCD deaths attributed while it is postulated that in the near future, CVDs account for more fatalities in low-income nations than infectious diseases.[1] The term myocardial infarction (MI) is applicable when there is evidence of necrosis of heart muscle cells in a patient with a clinical background consistent with ischaemia of the myocardium.[2] MI more familiarly known as 'heart attack' is predominantly instigated by the generation of atherosclerotic plaques in the walls of the arteries, thus compromising blood supply to the heart resulting in ischaemic alterations in cardiac muscles owing to insufficient oxygen delivery.[3] There are many risk factors for MI including hypertension, alcohol consumption, DM, tobacco use, inadequate physical activity, and obesity.[4] Correlating variables such as mechanical stress on blood arteries, which is a critical element in endothelial dysfunction brought on by hypertension, contributes to the pathophysiology of atherosclerosis and cardiac necrosis.[5]

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This predisposes to persistent microvascular blockage which increases irreversible capillary degeneration and leads to haemorrhage in the infarct zone, which independently predicts mortality or heart failure in the long term.[6,7] In the past, case fatality rates of patients presenting with both diabetes and myocardial infarction was quite high. However, the innovation of modern therapeutic interventions has reduced the fatality in such patients.[8] Despite the improvements, diabetes complicating MI is still responsible for a significantly higher case-fatality rate. With this rising prevalence in the Nigerian sub region, rates of NCDs such as MI and the mortality rates thereof are more than likely to escalate.[9,10] Obesity, often regarded as a modifiable cardiovascular risk factor, is associated with acute myocardial infarction,[11,12] and its prevalence is on the rise in the continent[13] Certain haematological indices are critical to the pathogenesis, management and monitoring of myocardial infarction. Anaemia in MI is well documented to adversely affect outcomes by causing an increased circulating catecholamine concentration which is directly linked to causing an aberration in the cardiac oxygen consumption balance. Anaemia also has a direct adverse effect on vascular healing, nitric oxide depletion and oxidative stress-mediated ischaemia-reperfusion injury as well as pathological ventricular remodelling.[14,15] Furthermore, myocardial infarction is said to develop from cardiac inflammation with expression of inflammation biomarkers as part of the hallmarks of the disease. Neutrophils, lymphocytes, tumour necrosis factor-α, interleukin 6 as well as products of the complement cascade are said to be affected.[16] Phytochemicals are useful in the discovery and development of new compounds and medications.[17,18] *Musa cavendish* forms an important constituent of diet in many countries around the world and has for long been researched and found to possess considerable amounts of biologically active compounds for example, flavonoids, carotenoids and phenols which have significant anti-inflammatory and antioxidant effects.[19,20] The

efficacy of *Musa* spp has been demonstrated as an anti-ulcerogenic agent[21] and also as having significant antioxidant potential.[22] Other researchers state that a single fruit of *Musa cavendish* can fulfil a significant proportion of the daily potassium requirement.[23] Recent research indicates that potassium supplementation may aid management of hypertension in persons with potassium deficiency and reduce the incidence of stroke.[24,25] *Musa cavendish* has also been shown to protect against renal and hepatic derangements in isoproterenol induced MI.[26] *Musa cavendish* is a modest source of health-enhancing flavonoids and polyphenolic antioxidants, including lutein and zeaxanthin. It contains tiny levels of β- and α-carotenes all of which function as anti-inflammatory agents as well as protective scavengers, neutralising oxygen-derived free radicals and reactive oxygen species (ROS).[27] The aim of the present study was thus to investigate the roles *Musa cavendish* may play in prevention or amelioration of changes in haematological indices in isoproterenol induced myocardial infarction as well as to establish the baseline effect of *Musa cavendish* on selected physiological parameters such as blood pressure, blood glucose and weight. There is as yet no existing study that investigates the possible role of MC in amelioration or prevention of haematological derangements in ISP induced MI.

Materials and Methods

Animals

Healthy adult male Wistar rats aged 12-15 weeks were randomly selected for the study. Animals were obtained from the Animal House of the Pharmacology Department, Delta State University, Abraka, Delta State, Nigeria. The rats were acclimatized for 14 days prior to the study under standard conditions of temperature and relative humidity with a 12:12 light:dark cycle. The rats were fed with standard animal pellet feeds and clean water ad libitum. Ethical approval was obtained from the Research, Ethics and Grants Committee of the Faculty of Basic Medical Sciences, Delta State University, Abraka (RBC/FBMC/DELSU/24/648) in compliance with the standard guidelines for the care and use of laboratory animals.

Plant Collection and Identification

Musa cavendish (MC) sample was obtained on the 23rd of December, 2023 from the Botanical Garden, Delta state University, Abraka located at Latitude $6^0$30'59.99"N and Longitude $3^0$23'5.99"E. The plant was identified and authenticated by Ozioma. E. Michael, a botanist with Delta State University; was registered with voucher Number DELSUH 303 and deposited in the herbarium of the Delta State University, Abraka.

Extraction of Plant Material

The fruit of the plant was separated from the bark, washed, rinsed, and chopped into little pieces. The diced samples were dried at room temperature until they achieved a consistent weight. The dried *Musa cavendish* pulp was then crushed into powder and stored in an airtight plastic container at room temperature.

The powdered pulp (100 g) was weighed into a 1000 mL Pyrex glass beaker and dissolved in 200 mL of distilled water. The mixture was allowed to stand for 72 hours at room temperature, stirred regularly with a glass rod, and then filtered with Whitman number one filter paper to obtain the crude aqueous extract of the fruit of *Musa cavendish*. The filtrate was concentrated using a lyophilizer and preserved in the refrigerator when not being utilized.

Research design

The study employed the use of an experimental study design which proceeded in two phases. Phase one involved the determination of the effects of MC on body weight, blood glucose and blood pressure of healthy Wistar rats. 24 animals were divided into 4 groups (6 animals per group). Group 1 animals were administered 10 mL/kg of distilled water. Group 2, 3 and 4 animals were administered 100 mg/kg, 200 mg/kg and 400 mg/kg of the MC extract respectively.

Phase 2 involved determination of the effects of MC on haematological changes in isoproterenol-induced myocardial infarction. 36 animals were randomly divided into 6 groups (6 animals per group). Animals in group 1 were administered 10 mL/kg of distilled water. Animals in groups 2 to 6 were administered 85 mg/kg of subcutaneous ISP on days 28 to 30 of the study. Moreover, Group 3 animals were also administered 0.5 mg/kg of propranolol, orally. Animals in groups 4 to 6 were also administered with 100, 200 and 400 mg/kg of MC respectively. Unless otherwise stated, drug administrations were carried out for 30 consecutive days prior to sacrifice.

Measurement of weight

Body weight of animals was measured weekly in grams using an electronic weighing balance.

Measurement of blood pressure and heart rate

Blood pressure measurements (systolic blood pressure – SBP, diastolic blood pressure – DBP, and heart rate – HR) were recorded in the conscious rats weekly during daylight (between 8 am and 12 noon) by the same investigator, using a tail-cuff plethysmography (MRBP system, IITC Life Science, Woodland Hills, CA, USA), a computerized non-invasive blood pressure system which measures rat's tail blood pressure by means of volume pressure. The rat was placed in the restrainer (animal holder) leaving the tail outside and adjusted to the position with restricted movement. The BP monitoring sensor was placed round the tail root of rats. Following animal movement stability, the data of SBP, DBP and HR were recorded.

Measurement of blood glucose

Fasting blood sugar level (FBS) was measured using ACCU-CHEK® Active glucometer with compatible blood glucose test strips. FBS was measured in mg/dL on the first day of each new week of the study.[28].

Assay of haematological parameters

Blood collected into heparinized tubes was used for the determination of haematological parameters: packed cell volume (PCV), haemoglobin (Hb), mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), red blood cells (RBC), white blood cell (WBC), basophils, neutrophils, eosinophils, platelet, lymphocytes, and monocytes were analysed using automated sysmex- K1000.

Data analysis

All data obtained were expressed as mean ± SEM (standard error of mean), and analyzed by one-way analysis of variance (ANOVA) and followed by Tukey's post hoc test. Analysis was done using Statistical package for social sciences (SPSS-23) and GraphPad prism 9.0 (GraphPad software Inc, USA). P-values < 0.05 were taken as significant. Data were presented in tables and graphs.

Results and Discussion

The present study assessed the effects of *Musa cavendish* on haematological changes in isoproterenol-induced myocardial infarction as well as the effects on selected physiological parameters in Wistar rats. The effects of *Musa cavendish* on selected baseline physiological parameters (weight, blood pressure, heart rate and blood glucose) are important to establish, as *Musa cavendish* is a staple food in many areas globally with potential to positively or negatively affect cardiovascular risk factors.

Effects of aqueous extract of Musa cavendish (MC) on body weight of healthy Wistar rats

Body weights of animals increased across all groups after a 4-week period. There was no significant ($p > 0.05$) gain in body weights of animals administered with MC at 100, 200, and 400 mg/kg when compared with normal control (Table 1). *Musa cavendish* tended to increase the final body weight of the animals, although it was not significant when compared to control. This result is dissimilar to the results of a similar study,[29] which determined that there was a reduction in weight and BMI in diabetic male human subjects whose diets were supplemented with Musa spp. for 4 weeks; a reduction that was found to be statistically significant.

Table 1: Effect of aqueous extract of *Musa cavendish* (MC) on body weight of normal Wistar rats

Group	Initial weight (g)	Final weight (g)	Weight gain (%)
Normal Control	121.60 ± 3.67	232.40 ±34.18	47.68 ± 9.59
Extract 100 mg/kg	124.20 ± 7.66	245.20 ± 19.83	49.36 ± 6.33
Extract 200 mg/kg	120.80 ± 4.14	248.40 ± 21.27	51.74 ± 10.20
Extract 400 mg/kg	118.80 ± 4.21	248.20 ± 28.84	52.27 ± 7.99

Values are expressed as mean ± SEM (n = 6); * = P < 0.05 when compared with normal control. Extract = *Musa cavendish*.

Effect of aqueous extract of Musa cavendish (MC) on blood pressure and pulse rate in Wistar rats

There was no significant change in systolic blood pressure (SBP), diastolic blood pressure (DBP) and pulse rate (PR) across the groups from week 1 through week 4. When compared with the normal control group, the groups administered the extract at 100, 200, and 400 mg/kg showed no significant change (p>0.05) in the haemodynamic indices after 4 weeks (Table 2).

Chronic hypertension is a major risk factor of CVD and MI. One study,[30] previously determined that pharmacological management of blood pressure reduced risk of major CVD significantly. The current study found no significant difference in heart rate and systolic or diastolic blood pressure when the treatment groups were compared with each other; but the groups all showed a significant reduction in systolic and diastolic blood pressure, as well as heart rate after 4 weeks of treatment with MC. The existing data on the effects of Musa spp on blood pressure control over time have been conflicting. Some investigators,[31] determined in their study that Musa spp caused a significant decrease in week 3 heart rate and week 4 heart rate and blood pressure. Other studies have suggested that potassium supplementation and the relatively high potassium content of Musa *spp* is beneficial in management of hypertension.[32,33]

Conversely, another study[34] did not find any significant effects of Musa spp on blood pressure or heart rate. A few differences in the studies cited to reflect the effects of Musa spp on blood pressure are worth noting however. First is that while one of the studies[31] utilized *Musa cavendish* on spontaneously hypertensive rats, the latter study[34] involved ripe Musa paradisiaca fruits in otherwise normal humans. The index study was executed using ripe *Musa cavendish* in Wistar rats. Worth highlighting as well is that the therapeutic effect of potassium supplementation on blood pressure was more pronounced in hypertensives than those with normal blood pressure.[32]

Table 2: Effect of aqueous extract of *Musa cavendish* (MC) on blood pressure and pulse rate in healthy Wistar rats

Groups	SBP (Week 1)	SBP (Week 4)	DBP (Week 1)	DBP (Week 4)	PR (Week 1)	PR (Week 4)
Normal Control	104.80 ± 10.11	108.20 ± 8.99	62.00 ± 10.43	58.60 ± 20.55	84.80 ± 11.16	88.60 ± 10.04
Extract 100 mg/kg	99.60 ± 9.05	102.80 ± 8.98	60.00 ± 12.77	64.00 ± 26.42	78.20 ± 9.83	86.80 ± 8.94
Extract 200 mg/kg	101.80 ± 10.16	97.60 ± 8.70	62.60 ± 8.26	59.60 ± 36.75	77.00 ± 13.69	78.80 ± 9.96
Extract 400 mg/kg	106.60 ± 9.03	104.80 ± 13.16	60.20 ± 14.54	63.60 ± 12.26	83.80 ± 8.96	80.20 ± 9.29

Values are expressed as mean ± SEM (n = 6); * = P < 0.05 when compared with normal control. Extract = *Musa cavendish*; SBP = Systolic blood pressure; DBP = Diastolic blood pressure; PR = pulse rate.

Effect of aqueous extract of Musa cavendish (MC) on blood glucose level of healthy Wistar rats

Figure 1 shows the effect of aqueous extract of MC on blood glucose level of normal Wistar rats. There was non-significant (p>0.05) increase in the glucose level across the animal groups administered 100, 200, and 400 mg/kg when compared to normal control group. Furthermore, this study did not find any significant change in blood glucose. Lin[31] however found in their study, that *Musa cavendish* ameliorated blood glucose derangements in diabetic rats.

Effect of aqueous extract of Musa cavendish (MC) on PCV, Hb and platelets in isoproterenol-induced MI in Wistar rats

The precise effects exerted by various phytochemical containing substances on haematological indices have become the subject of much research in recent years. Furthermore, the effect of some co-morbidities on MI have also taken a more central role recently. The far-reaching effects of anaemia for example, has been established to worsen the mortality and morbidity of MI and lead to poor outcomes all round.[35] The same author postulates that one of the mechanisms by which this occurs is the activation of the sympathetic system by anaemia which worsens the myocardial supply imbalance.

Table 3 illustrates the effect of aqueous extract of MC on packed cell volume (PCV), haemoglobin (Hb) concentration and platelet count in isoproterenol-induced myocardial infarction. Administration of isoproterenol decreased PCV and Hb non-significantly (p>0.05) and significantly increased platelet counts when compared to normal control. Treatment with the extract had an increase in PCV and Hb which was significant (p<0.05) at 400 mg/kg when compared with isoproterenol group (ISP). Platelet count was significantly (p<0.05) reduced in groups that received the extract at 100, 200, and 400 mg/kg when compared with isoproterenol group.

One study[36] reveals that anaemia has the potential to worsen the myocardial ischaemic insult in AMI both by decreasing the oxygen content of the blood supplied to the jeopardized myocardium and by increasing myocardial oxygen consumption through increased cardiac metabolic activity. Carson *et. al.* [37] therefore suggests that low haemoglobin (Hb) concentrations at presentation convey a worse prognosis in patients with MI. Research using animal models have also demonstrated a protective effect of higher haemoglobin concentrations in the setting of coronary artery stenosis, even when said stenosis is said to be significant.[37]

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Table 3: Effects of *Musa cavendish (*MC) on PCV, Hb and platelets in isoproterenol-induced MI in Wistar rats

Group	PCV (%)	Hb (g/dl)	PLATELETS (10^9/L)
Normal control	40.17 ± 0.98	13.37 ± 0.33	76.00 ± 3.28
ISP	37.00 ± 0.89	12.40 ± 0.31	150.00 ± 1.10*
ISP + PRO 0.5 mg/kg	41.67 ± 2.66	13.87 ± 0.90	77.50 ± 6.02^{*a}
ISP + Extract 100 mg/kg	39.17 ± 1.72	13. 00 ± 0.57	119.50 ± 10.82^{*ab}
ISP + Extract 200 mg/kg	42.00 ± 0.89	13.97 ± 0.31	102.00 ± 1.38^{*ab}
ISP + Extract 400 mg/kg	46.50 ± 1.38^{*ab}	15.48 ± 0.46^{a}	99.67 ± 3.06^{*ab}

Values are expressed as mean ± SEM (n = 6); * = P < 0.05 when compared with normal control, ^{a} = P < 0.05 when compared with ISP, ^{b} = P < 0.05 when compared with ISP+PRO. Extract = *Musa cavendish*; ISP = isoproterenol; PRN = propranolol; PCV = packed cell volume; Hb = haemoglobin

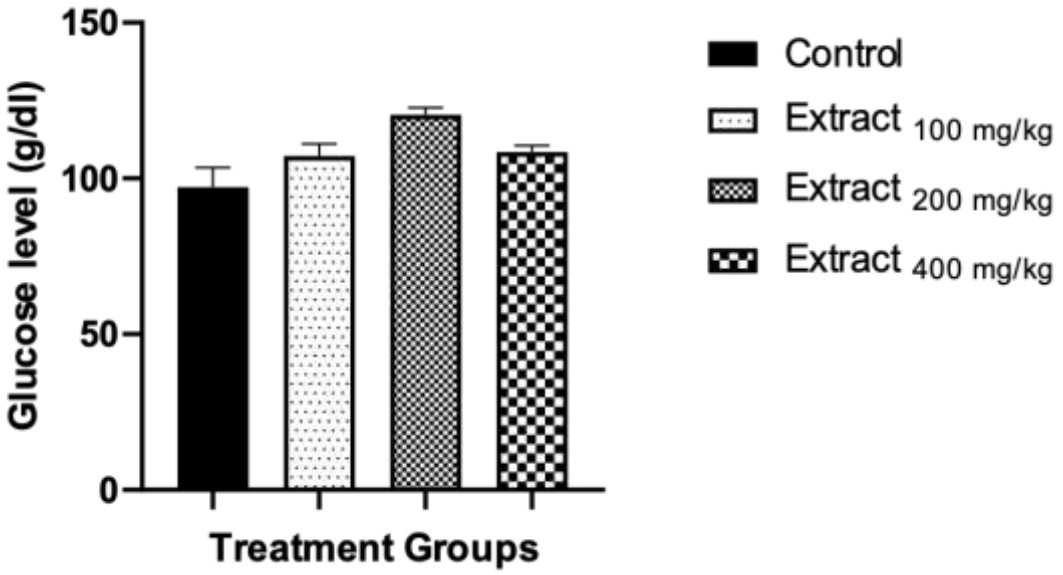

Figure 1: Effect of aqueous extract of *Musa cavendish* (MC) on blood glucose in Wistar rats.
Values are expressed as mean ± SEM (n = 6); Extract = *Musa cavendish*. Values are expressed as mean ± SEM (n = 6)

The index research found a significant decrease in pack cell volume (PCV) and haemoglobin (Hb) concentration in the MI control group which appeared to be corrected by treatment with the MC extract at 100 mg/kg, 200 mg/kg and 400 mg/kg. It is well established that oxidative stress is able to cause anaemia by oxidation of the red blood cell membrane thus increasing fragility and auto-oxidation thus leading to a reduction in PCV and Hb concentration.[38] There appears to be multiple clinical studies which confirm the presence of anaemia at presentation of patients with MI with numbers varying anywhere between 30% and 60%.[35,39,40] The cause and mechanism of this anaemia has not been definitively defined as while some authors believe it could have predated the MI,[40] others suggest it could be caused by the inflammation and pathological processes that lead to MI including haemodilution.[39] This study further demonstrated a significant increase in haemoglobin concentration and pack cell volume in treatment groups when compared with the normal and negative controls. This is at variance with other similar studies carried out on the effects of Musa spp on haematological parameters which did not find any significant difference[41] and even a decrease in haemoglobin concentration.[42] However, the other studies focused on other species of Musa other than cavendish and also on other parts of the plant such as bark and stem respectively.

Platelets have a primary function of stopping haemorrhage from vascular endothelium or tissue following an injury, but pathologically, their apportioned physiological function of plugging endothelial defects, platelet activation in response to plaque disruption, resulting in occlusive thrombosis, is the base upon which acute MI could occur.[43] The researchers[43] further comment that there is a remarkably higher platelet count in MI compared with the normal population while other authors revealed that the mean platelet volume is significantly elevated in MI.[44] It is in fact established that both platelet count and mean platelet volume are important indices in prediction of long-term mortality after MI.[45] Platelet activation results in the production of pro-inflammatory, mitogenic, and pro-apoptotic chemicals, as well as cytotoxic substances, which interact with leukocytes and endothelial cells, initiating and exacerbating cardiac ischaemia/reperfusion injury.[46] The results of the index study showed a significant elevation in platelet count in the MI control group when compared with the normal control group. Furthermore, this significant decrease of platelets in the ISO treatment group appeared to be ameliorated in a non-dose dependent manner.

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Effect of aqueous extract of Musa cavendish (MC) on white blood cell (WBC), monocyte, neutrophil, and lymphocyte count in isoproterenol-induced MI in Wistar rats

The changes in white blood cells (WBC) count in myocardial infarction has also been the subject of much research. All AMI studied groups had significantly higher WBC count compared with controls mainly due to increased neutrophils.[47] There is documented evidence confirming that a rise in WBC counts conveys a higher mortality as well as prolonged stay in hospital as regards patients being managed for unstable angina or AMI.[48,49] These effects were seen in all sub groups; women, men, elderly patients or younger patients as well as smokers and non-smokers.

Figures 2-5 show the effect of aqueous extract of MC on white blood cell (WBC), monocyte, neutrophil, and lymphocyte counts. Induction with isoproterenol significantly $p<0.05$) increased WBC count, as well as monocyte, neutrophil, and lymphocyte count when compared to normal control. Treatment with MC at 100, 200, and 400 mg/kg significantly ($p<0.05$) reduced the elevated WBC count and the differentials when compared to isoproterenol (ISP).

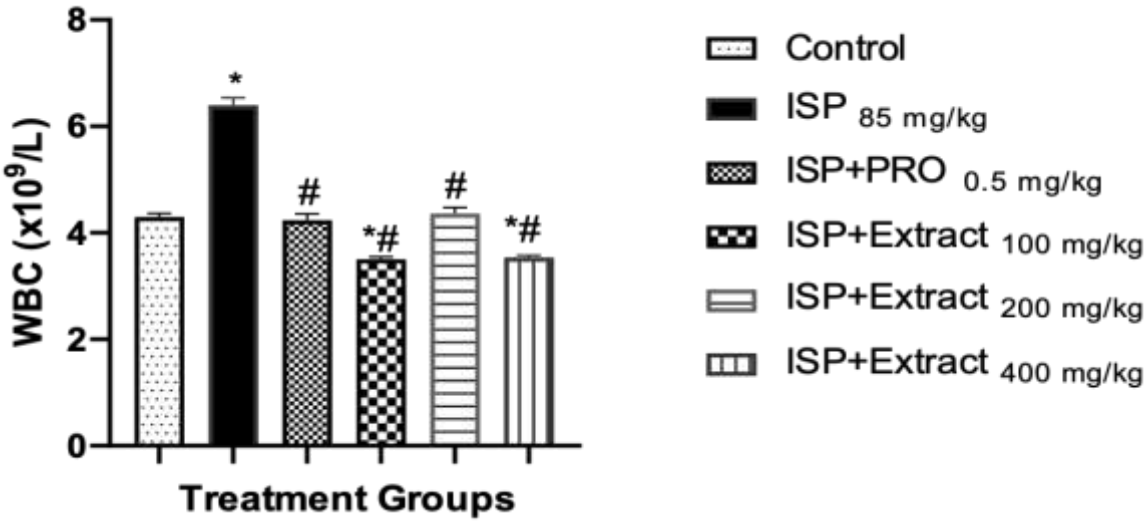

Figure 2: Effect of aqueous extract of *Musa cavendish* (MC) on white blood cell (WBC) count on isoproterenol-induced myocardial infarction in Wistar rats. Values are expressed as mean ± SEM (n = 6); * = P < 0.05 when compared with normal control, # = P < 0.05 when compared with ISP.

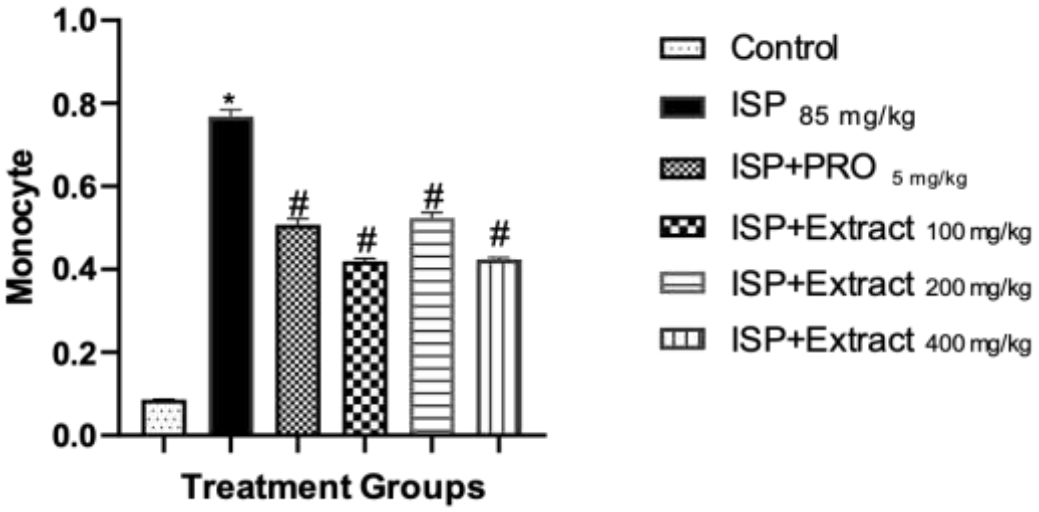

Figure 3: Effect of aqueous extract of *Musa cavendish* (MC) on monocyte count on isoproterenol-induced myocardial infarction in Wistar rats. Values are expressed as mean ± SEM (n = 6); * = P < 0.05 when compared with normal control, # = P < 0.05 when compared with ISP. Control = Normal control; ISP = Isoproterenol; PRO = Propranolol; Extract = *Musa cavendish* (MC)

The results of this research found a significantly higher WBC count in the MI control compared to the normal control. This is in keeping with established knowledge of pattern of rise in WBC in MI as quoted above. Furthermore, there was also a significant decrease in the MC treatment

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groups, when compared to the ISP treatment groups. Human studies in STEMI patients demonstrated higher WBC counts on account of a higher neutrophil count at hospitalisation compared with stable angina patients. Interventions such as percutaneous coronary intervention (PCI) and tocilizumab (a biological immunosuppressant) also showed a gradual decline in neutrophils which was found to be associated with improved myocardial recovery and lower peak troponin values.[50] This finding was similar to the results of the index study which also found a significant decrease in the neutrophil count in MC treated groups compared to the ISP group suggesting an improved myocardial performance. The trend with total white blood cell count was also noted with neutrophils and monocytes. There was a significant increase in neutrophils and monocytes in the negative control group, a trend reversed by the administration of the plant extract. The elevation in monocytes and neutrophils strengthens the evidence for inflammation as a mechanism of cardiac necrosis and also MC for its anti-inflammatory effects in reversing the ischaemic necrosis induced. This is further confirmed in the study[51] which assessed the pro-inflammatory role played by neutrophils and monocytes by the release of proteolytic enzymes and reactive oxygen species that exacerbate the injury by harming myocytes that survived the ischaemic period.

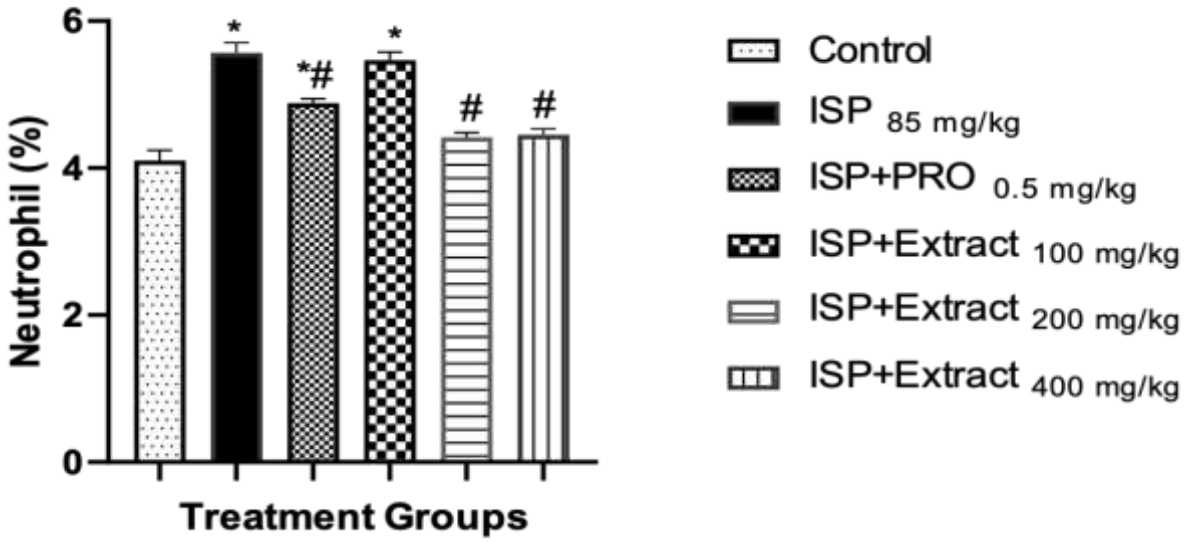

Figure 4: Effect of aqueous extract of *Musa cavendish* (MC) on neutrophil count on isoproterenol-induced myocardial infarction in Wistar rats. Values are expressed as mean ± SEM (n = 6); * = P < 0.05 when compared with normal control, # = P < 0.05 when compared with ISP. Control = Normal control; ISP = Isoproterenol; PRO = Propranolol; Extract = *Musa cavendish* (MC)

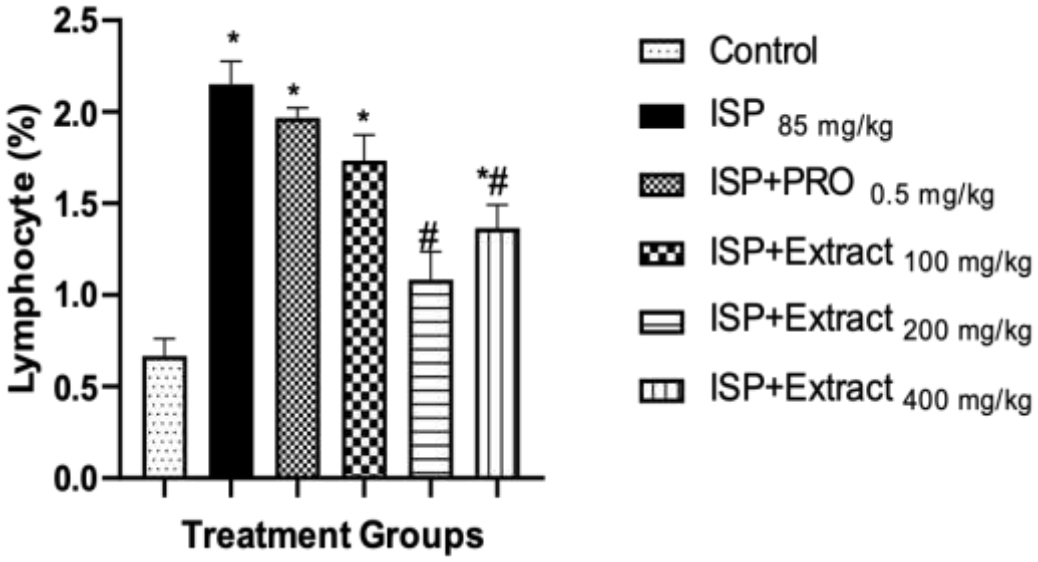

Figure 5: Effect of aqueous extract of *Musa cavendish* (MC) on lymphocyte count on isoproterenol-induced myocardial infarction in Wistar rats. Values are expressed as mean ± SEM (n = 6); * = P < 0.05 when compared with normal control, # = P < 0.05 when compared with ISP. Control = Normal control; ISP = Isoproterenol; PRO = Propranolol; Extract = *Musa cavendish* (MC)

Effects of Musa cavendish on Neutrophil /Lymphocyte Ratio, Platelet/Lymphocyte Ratio and Neutrophil+ Monocyte /Lymphocyte Ratio in isoproterenol induced myocardial infarction in Wistar rats

Table 4 shows that there was a significant increase in Neutrophil /Lymphocyte Ratio (NLr), Platelet/Lymphocyte Ratio (PLr) and Neutrophil+ Monocyte /Lymphocyte Ratio (N+M/Lr) following isoproterenol induction when compared to normal control. Treatment with MC extract at 100, 200, and 400 mg/kg significantly decreased NLr, PLr and N+M/Lr values when with ISP. There is also evidence as to the use of the neutrophil/lymphocyte ratio in the prediction of severity in subjects with MI. One study[52] investigated the predictive value of the neutrophil to lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR), in patients with non-ST-elevated myocardial infarction (NSTEMI). A high NLR indicates systemic inflammation and is said to be a major predictor of poor clinical outcomes. High NLR values are associated with increased risk of complications such as heart failure and mortality after acute MI.

The results of this study were indicative of a significantly higher NLR and PLR for the negative control groups than for the treatment groups suggestive of a protective role for MC in the course, prognosis and severity of MI.

Table 4: Effects of *Musa cavendish* on NLr, PLr and N+M/Lr in isoproterenol-induced myocardial infarction in Wistar rats

	Neutrophil /Lymphocyte Ratio (NLr)	Platelet /Lymphocyte Ratio (PLr)	Neutrophil + Monocyte /Lymphocyte Ratio (N+M/Lr)
Normal control	2.00 ± 0.26	88.47 ± 2.05	1.62 ± 0.03
ISP	3.00 ± 0.26*	118.90 ± 3.98*	3.44 ± 0.09*
ISP + PRN 5 mg	1.40 ± 0.52#	74.19 ± 1.64#	1.96 ± 0.04#
ISP + Extract 100 mg/kg	1.87 ± 0.08#	98.60 ± 4.52#	2.03 ± 0.02#
ISP + Extract 200 mg/kg	1.40 ± 0.08#	93.73 ± 2.87#	2.01 ± 0.04#
ISP + Extract 400 mg/kg	2.00 ± 0.26#	102.80 ± 2.46#	2.01 ± 0.02#

Values are expressed as mean ± SEM (n = 6); * = P < 0.05 when compared with normal control, # = P < 0.05 when compared with ISP. Extract = *Musa cavendish*; ISP = isoproterenol; PRN = propranolol

Conclusion

Administration of MC to healthy test animals did not cause any significant change in blood pressure, pulse rate, weight or blood glucose in this study. Reductions in haemoglobin concentration, pack cell volume and elevations in platelet count caused by induction of MI with isoproterenol were reversed by treatment with MC. Furthermore, it was also observed that induction of MI with isoproterenol was associated with elevation in total white blood cells as well as neutrophils, monocytes and lymphocytes. These derangements were drastically reversed by pre-treatment with MC; an amelioration that was significant enough to cause a better prognostic profile as evidenced by a significantly lower neutrophil/lymphocyte ratio, platelet/lymphocyte ratio and neutrophil+monocyte/lymphocyte ratio. An overall protective and beneficial effect of MC in isoproterenol induced MI was thus determined, without any significant effects on blood pressure, heart rate and blood sugar.

Conflict of Interest

The author declares no conflicts of interest.

Authors' Declaration

The authors hereby declare that the work presented in this article is original and that any liability for claims relating to the content of this article will be borne by them.

References

1. Yuyun MF, Sliwa K, Kengne AP, Mocumbi AO, Bukhman G. Cardiovascular Diseases in Sub-Saharan Africa Compared to High-Income Countries: An Epidemiological Perspective. Glob Heart. 2020; 15(1): 15.
2. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, White HD; Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction. Fourth Universal Definition of Myocardial Infarction (2018). J Am Coll Cardiol. 2018; 72(18): 2231-2264.
3. Saito Y, Oyama K, Tsujita K, Yasuda S, Kobayashi Y. Treatment strategies of acute myocardial infarction: updates on revascularization, pharmacological therapy, and beyond. J Cardiol. 2023; 81(2): 168-178.
4. Essien OE, Andy J, Ansa V, Otu AA, Udoh A Coronary artery disease and the profile of cardiovascular risk factors in South South Nigeria: a clinical and autopsy study. Cardiol Res Pract. 2014; 1: 804751.
5. Rakugi H, Yu H, Kamitani A, Nakamura Y, Ohishi M, Kamide K, Nakata Y, Takami S, Higaki J, Ogihara T. Links between hypertension and myocardial infarction. Am Heart J. 1996; 132(1 Pt 2 Su): 213-221.
6. Eitel I, Kubusch K, Strohm O, Desch S, Mikami Y, De Waha S, Gutberlet M, Schuler G, Friedrich MG, Thiele H. Prognostic value and determinants of a hypointense infarct core in T2-weighted cardiac magnetic resonance in acute reperfused ST-elevation–myocardial infarction. Circ Cardiovasc Imaging. 2011; 4(4): 354-362.
7. Carrick D, Haig C, Maznyczka AM, Carberry J, Mangion K, Ahmed N, Yue May VT, McEntegart M, Petrie MC, Eteiba H, Lindsay M. Hypertension, microvascular pathology, and prognosis after an acute myocardial infarction. Hypertens. 2018; 72(3): 720-730.
8. Nedkoff L, Knuiman M, Hung J, Briffa TG. Improving 30-day case fatality after incident myocardial infarction in people with diabetes between 1998 and 2010. Heart. 2015; 101(16): 1318-1324.

9. Nesto RW, Zarich S. Acute myocardial infarction in diabetes mellitus: lessons learned from ACE inhibition. Circulation. 1998; 97(1): 12-5.
10. Johansson S, Rosengren A, Young K, Jennings E. Mortality and morbidity trends after the first year in survivors of acute myocardial infarction: a systematic review. BMC Cardiovasc Disord. 2017; 17(1): 53.
11. Yusuf S, Hawken S, Ounpuu S, Bautista L, Franzosi MG, Commerford P, Lang CC, Rumboldt Z, Onen CL, Lisheng L, Tanomsup S, Wangai P Jr, Razak F, Sharma AM, Anand SS; INTERHEART Study Investigators. Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. Lancet. 2005; 366(9497): 1640-1649.
12. Şaylık F, Çınar T, Hayıroğlu Mİ. Effect of the obesity paradox on mortality in patients with acute coronary syndrome: a comprehensive meta-analysis of the literature. Balkan Med J. 2023; 40(2): 93.
13. Alexandratos, N. The Mediterranean diet in a world context. Public Health Nutr. 2006; 9(1a):111-117.
14. Levy PS, Quigley RL, Gould SA. Acute dilutional anemia and critical left anterior descending coronary artery stenosis impairs end organ oxygen delivery. J Trauma. 1996; 41(3): 416-423.
15. Mamas MA, Kwok CS, Kontopantelis E, Fryer AA, Buchan I, Bachmann MO, Zaman MJ, Myint PK. Relationship Between Anemia and Mortality Outcomes in a National Acute Coronary Syndrome Cohort: Insights From the UK Myocardial Ischemia National Audit Project Registry. J Am Heart Assoc. 2016; 5(11): e003348.
16. Matter MA, Paneni F, Libby P, Frantz S, Stähli BE, Templin C, Mengozzi A, Wang YJ, Kündig TM, Räber L, Ruschitzka F, Matter CM. Inflammation in acute myocardial infarction: the good, the bad and the ugly. Eur Heart J. 2024; 45(2): 89-103.
17. Chihomvu P, Ganesan A, Gibbons S, Woollard K, Hayes MA. Phytochemicals in Drug Discovery-A Confluence of Tradition and Innovation. Int J Mol Sci. 2024; 25(16): 8792.
18. Elufioye TO, Habtemariam S. Drug Development for the Management of Corona Viruses: Insights from Natural Antiviral Agents. 2020; 4(9): 490-511.
19. Speisky H, Shahidi F, Costa de Camargo A, Fuentes J. Revisiting the oxidation of flavonoids: Loss, conservation or enhancement of their antioxidant properties. Antioxidants. 2022; 11(1): 133.
20. Jia JY, Zang EH, Lv LJ, Li QY, Zhang CH, Xia Y, Zhang L, Dang LS, Li MH. Flavonoids in myocardial ischemia-reperfusion injury: Therapeutic effects and mechanisms. Chin Herb Med. 2020;13(1):49-63.
21. Madueke AC, Ugwu DO, Nwanelo VO, Anosike AC, Anajemba RC, Onoh PA. Anti-Ulcerogenic Effect of Unripe Plantain (*Musa* paradisiaca) Pulp on Indomethacin Induced Ulcer in Wistar Rat. Trop J Nat Prod Res. 2020; 4(10): 784-789.
22. Ahmed MU, Bala U, Umaru IJ. Antioxidant and Alpha Amylase Inhibitory Potential of Methanol Extract of Unripe *Musa* paradisiaca Fruit with Husk. Trop J Nat Prod Res. 2022; 6(8): 1283-1285
23. Afzal MF, Khalid W, Akram S, Khalid MA, Zubair M, Kauser S, Abdelsamea Mohamedahmed K, Aziz A, Anusha Siddiqui S. Bioactive profile and functional food applications of banana in food sectors and health: a review. Int J Food Prop. 2022; 25(1) :2286-2300
24. Arnett DK, Blumenthal RS, Albert MA, Buroker AB, Goldberger ZD, Hahn EJ, Himmelfarb CD, Khera A, Lloyd-Jones D, McEvoy JW, Michos ED, Miedema MD, Muñoz D, Smith SC Jr, Virani SS, Williams KA Sr, Yeboah J, Ziaeian B. 2019 ACC/AHA Guideline on the Primary Prevention of Cardiovascular Disease: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2019;140(11):e563-e595.
25. Kumar N, Ved A, Yadav RR, Prakash O. A comprehensive review on phytochemical, nutritional, and therapeutic importance of *Musa*. Int J Curr Res Rev. 2021; 13: 114-124.
26. Umukoro EK, Akponah E, Elijah OB, Moke EG, Ohaju-Obodo, JO, Georgewill, OA. *Musa cavendish* ameliorates isoproterenol induced renal and hepatic injury in Wistar rats. J Oral Public Health. 2025; 6(7): e1267
27. Nataraj L. Antioxidant capacity and phenolic content of different solvent extract *Musa* paradisiacal and mustai (Rivea hypocrateriformis). Food Sci Biotech. 2010; 9(5): 1251-1258.
28. Moke EG, Omogbai EKI, Osagie-Eweka SDE, Uchendu AP, Omogbiya AI, Ben-Azu B, Eduviere AT, Edje KE, Umukoro EK, Anachuna KK, Asiwe JN, Ahante E, Oghoghovwe IJ. Co-administration of metformin and/or glibenclamide with losartan reverses NG-nitro-l-arginine-methyl ester-streptozotocin-induced hypertensive diabetes and haemodynamic sequelae in rats. Microvasc Res. 2023;147:104497.
29. Ble-Castillo JL, Aparicio-Trápala MA, Francisco-Luria MU, Córdova-Uscanga R, Rodríguez-Hernández A, Méndez JD, Díaz-Zagoya, JC. Effects of native banana starch supplementation on body weight and insulin sensitivity in obese type 2 diabetics. Int J Environ Res Public Health. 2010; 7(5): 1953-1962.
30. Blood Pressure Lowering Treatment Trialists' Collaboration. Pharmacological blood pressure lowering for primary and secondary prevention of cardiovascular disease across different levels of blood pressure: an individual participant-level data meta-analysis. Lancet. 2021; 397(10285): 1625-1636.
31. Lin HL, Tseng YH, Yeh CC, Chen YM, Shen KP. Effect evaluation of banana on improving hyperglycemia and hypertension in diabetic spontaneously hypertensive rats. Nat Prod Comm, 2023; 18(11):1-10
32. Filippini T, Naska A, Kasdagli MI, Torres D, Lopes C, Carvalho C, Moreira P, Malavolti M, Orsini N, Whelton PK, Vinceti M. Potassium Intake and Blood Pressure: A Dose-Response Meta-Analysis of Randomized Controlled Trials. J Am Heart Assoc. 2020; 9(12):e015719.
33. Yanti, E., Nofia, VR. The effect of mosquito banana (*Musa* paradisiaca) on blood pressure in hypertension patients.In Proceeding International Conference Syedza Saintika. 2020; 1 (1)
34. Rafi IK, Sanyal A, Hoque M. The Impact of Banana Consumption on Bangladeshi Rickshaw Pullers' Assessing Cholesterol, Liver and Blood Pressure Functions. Middle East Res J Biological Sci. 2023; 3(2): 24-28.
35. Padda J, Khalid K, Hitawala G, Batra N, Pokhriyal S, Mohan A, Cooper AC, Jean-Charles G., Pokhriyal SC. Acute anemia and myocardial infarction. Cureus. 2021; 13(8).
36. Pei J, Wang X, Chen P, Zheng K, Hu X. Hb Levels and Sex Differences in Relation to Short-Term Outcomes in Patients With Acute Myocardial Infarction. Front Cardiovasc Med. 2021; 8: 653351.
37. Carson JL, Brooks MM, Hébert PC, Goodman SG, Bertolet M, Glynn SA, Chaitman BR, Simon T, Lopes RD, Goldsweig AM, DeFilippis AP, Abbott JD, Potter BJ, Carrier FM, Rao SV, Cooper HA, Ghafghazi S, Fergusson DA, Kostis WJ, Noveck H, Kim S, Tessalee M, Ducrocq G, de Barros E Silva PGM, Triulzi DJ, Alsweiler C, Menegus MA, Neary JD, Uhl L, Strom JB, Fordyce CB, Ferrari E, Silvain J, Wood FO, Daneault B, Polonsky TS, Senaratne M, Puymirat E, Bouleti C, Lattuca B, White HD, Kelsey SF, Steg PG, Alexander JH; MINT Investigators. Restrictive or Liberal Transfusion Strategy in Myocardial Infarction and Anemia. N Engl J Med. 2023; 389(26): 2446-2456.
38. Orrico F, Laurance S, Lopez AC, Lefevre SD, Thomson L, Möller MN, Ostuni MA. Oxidative Stress in Healthy and Pathological Red Blood Cells. Biomolecules. 2023; 13(8): 1262.
39. Anker SD, Voors A, Okonko D, Clark AL, James MK, von Haehling S, ... & Dickstein K. Prevalence, incidence, and prognostic value of anaemia in patients after an acute myocardial infarction: data from the OPTIMAAL trial. Eur Heart J. 2009; 30(11): 1331-1339.
40. Bolińska S, Sobkowicz B, Zaniewska J, Chlebińska I, Boliński J, Milewski R, ... & Musiał W. The significance of anaemia in patients with acute ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention. Pol Heart J (Kardiologia Polska). 2011; 69(1): 33-39.

41. Osinowo O, Odubo TC, Izah SC. Hematopoietic Potentials of *Musa* paradisiaca Stem Sap. ES General. 2024; 6: 1139.
42. e Silva AF, Copatti CE, de Oliveira EP, Bonfá HC, de Melo FVST, de Silva Camargo AC, Melo JFB. Effects of whole banana meal inclusion as replacement for corn meal on digestibility, growth performance, haematological and biochemical variables in practical diets for tambaqui juveniles (*Colossoma macropomum*). Aquac Rep. 2020; 17: 100307.
43. Amraotkar AR, Song DD, Otero D, Trainor PJ, Ismail I, Kothari V, Singh A, Moore IV JB, Rai SN, DeFilippis AP. Platelet count and mean platelet volume at the time of and after acute myocardial infarction. Clin Appl Thromb Hemost. 2017; 23(8): 1052-1059.
44. Chu SG, Becker RC, Berger PB, Bhatt DL, Eikelboom JW, Konkle B, Mohler ER, Reilly MP, Berger JS. Mean platelet volume as a predictor of cardiovascular risk: a systematic review and meta-analysis. J Thromb Hemost. 2010; 8(1): 148-156.
45. Azab B, Torbey E, Singh J, Akerman M, Khoueiry G, Mcginn Jr JT, Widmann WD, Lafferty J. Mean platelet volume/platelet count ratio as a predictor of long-term mortality after non-ST-elevation myocardial infarction. Platelets. 2011; 22(8): 557-566.
46. Fuentes E, Moore-Carrasco R, de Andrade Paes AM, Trostchansky A. Role of Platelet Activation and Oxidative Stress in the Evolution of Myocardial Infarction. J Cardiovasc Pharmacol Ther. 2019; 24(6): 509-520.
47. Avramakis G, Papadimitraki E, Papakonstandinou D, Liakou K, Zidianakis M, Dermitzakis A, Mikhailidis DP, Ganotakis ES. Platelets and white blood cell subpopulations among patients with myocardial infarction and unstable angina. Platelets. 2007; 18(1): 16-23.
48. Dragu R, Huri S, Zukermann R, Suleiman M, Mutlak D, Agmon Y, Kapeliovich M, Beyar R, Markiewicz W, Hammerman H, Aronson D. Predictive value of white blood cell subtypes for long-term outcome following myocardial infarction. Atherosclerosis. 2008;196(1):405-412.
49. Shen XH, Chen Q, Shi Y, Li HW. Association of neutrophil/lymphocyte ratio with long-term mortality after ST elevation myocardial infarction treated with primary percutaneous coronary intervention. Chin Med J (Engl). 2010; 123(23): 3438-3443.
50. Huse C, Anstensrud AK, Michelsen AE, Ueland T, Broch K, Woxholt S, Yang K, Sharma K, Tøllefsen IM, Bendz B, Amundsen BH, Damås JK, Berg ES, Bjørkelund E, Quiles-Jiménez A, Bjerkeli V, Bendz C, Kleveland O, Stensaeth KH, Opdahl A, Kløw NE, Andersen GØ, Wiseth R, Halvorsen B, Gullestad L, Seljeflot I, Aukrust P, Osnes L, Dahl TB. Interleukin-6 inhibition in ST-elevation myocardial infarction: Immune cell profile in the randomised ASSAIL-MI trial. EBioMedicine. 2022; 80:104013.
51. Peet C, Ivetic A, Bromage DI, Shah AM. Cardiac monocytes and macrophages after myocardial infarction. Cardiovasc Res. 2020; 116(6): 1101-1112.
52. Tudurachi BS, Anghel L, Tudurachi A, Sascău RA, Stătescu C. Assessment of Inflammatory Hematological Ratios (NLR, PLR, MLR, LMR and Monocyte/HDL-Cholesterol Ratio) in Acute Myocardial Infarction and Particularities in Young Patients. Int J Mol Sci. 2023; 24(18): 14378.