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Research Article
L-Arginine Oral Supplementation Reverses Hematological and Electrolytes Imbalances in Adrenaline-Induced Myocardial Injury in Rats

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Abstract

Background and Objective: L-arginine is a precursor for the synthesis of nitric oxide (NO) molecule that is used in the management of vascular disorders including myocardial ischemia. This study investigated the effect of L-arginine on some haematological parameters and serum electrolytes levels in adrenaline-induced myocardial injury rats. **Materials and Methods:** Wistar rats of both sexes were assigned to five groups of 10 rats each. The control group received rat chow and water daily. L-arginine+myocardial injury group received 200 mg kg^{-1} b.wt., of L-arginine orally first for 14 days followed by intramuscular induction of myocardial injury (MI) with 2 mg kg^{-1} of adrenaline for 2 days. In the myocardial injury+L-arginine group, myocardial injury was induced first with 2 mg kg^{-1} of adrenaline followed by administration of 200 mg kg^{-1} of L-arginine for 14 days. The L-arginine only group received 200 mg kg^{-1} of L-arginine for 14 days while the myocardial injury only group received 2 mg kg^{-1} of adrenaline for 2 days. **Results:** MI caused a significant ($p<0.01$) decrease in serum nitric oxide, RBC, hematocrit, haemoglobin, platelet and leukocytosis which was reversed by L-arginine supplementation. Serum electrolytes levels of Na^+, Cl^- and HCO_3^- were significantly ($p<0.05$) reduced in the MI group but was increased following treatment with L-arginine. **Conclusion:** L-arginine promotes erythropoiesis, raises serum nitric oxide level and maintains serum electrolytes level in adrenaline-induced myocardial injury in rats.

Key words: Electrolytes, L-arginine, myocardial injury, platelets, red blood cells

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Competing Interest: The authors have declared that no competing interest exists.

Data Availability: All relevant data are within the paper and its supporting information files.

INTRODUCTION

Myocardial Injury (MI) is a disease condition that is usually characterized by myocardial cell death arising from a prolonged lack of oxygen to cardiac tissues[1]. It is a common symptom of myocardial ischemia which occurs when cardiac injury surpasses a critical threshold, resulting in mortal cardiac damage[2]. During the MI attack, the formation of reactive oxygen species (ROS) are the underline cause of cardiac myocytes damage[3]. The MI is one of the major health problems leading to a high mortality and morbidity rate.[4] The prevalence of MI is approximately 1.5 M annually in the United States of America[5]. In Nigeria, cardiovascular-related death is estimated to be 51% while death associated with myocardial infarction is 9%[6]. The ischemic attack has been linked to vasospasm of the coronary vessels and vasodilation of the vessels has been associated with high availability of endogenous nitric oxide synthesis[7].

L-arginine is a precursor for the synthesis of the nitric oxide (NO) molecule. Nitric oxide is a vasodilator produced by the endothelial cells of the blood vessels through the enzymatic activity of nitric oxide synthase[8]. Sources of L-arginine include dietary protein, endogenous synthesis and proteins turnover. In disease conditions, nitric oxide is depleted because the endogenous synthesis of L-arginine is not sufficient to meet the body's needs[9]. Thus, L-arg also plays important role in many cellular processes such as cellular regeneration, wound healing, immunity and protein turnover[10] in addition to having antioxidant properties[11]. The NO synthase inhibition produces various cardiovascular and haematological abnormalities including ventricular contractile dysfunction[12]. L-arginine, NO donors or the precursor for NO synthesis, has been reported to ameliorate ischemic/ reperfusion injury in myocardial infarction[13].

Electrolytes, biochemical and haematological parameters are the main homeostatic systems of humans and animals. Haematological parameters such as red blood cells, hematocrit, white blood cells and platelets in humans and animals are major health index indices. Electrolyte imbalance is an abnormality in the concentration of electrolytes in the body. It plays a vital role in the maintenance of homeostasis within the body. Electrolyte imbalance can be caused by excessive ingestion, diminished elimination, diminished ingestion and excessive elimination of an electrolyte[14].

About 60% of death has been attributed to arrhythmia or myocardial failure resulting from myocardial ischemia[15]. Myocardial injury can result from acute adrenaline administration due to overproduction of reactive oxygen species[16,17]. L-arginine has been reported to be actively involved in mediating these problems through the vasodilatory effect of NO but there is no sufficient data regarding the effect of L-arginine on some haematological parameters and serum electrolytes levels. Thus, this research investigated the effect of L-arginine supplementation on some haematological parameters and electrolytes levels in adrenaline-induced myocardial infarction rats. The study investigated the haematological parameters and serum electrolyte levels in adrenaline-induced myocardial injury rats treated with L-arginine.

MATERIALS AND METHODS

Study area: This study was carried out at the Department of Physiology, University of Calabar, Calabar Nigeria from October, 5th to December 19th, 2019.

Animals and diet: Ethical approval was obtained from the Faculty of Basic Medical Sciences Animal Ethical Committee (039PHY2619). Adult Wistar rats of both sexes weighing 120-250 g were used for this experiment. The animals were obtained from the animal house of the Department of Physiology, University of Calabar, Nigeria and were divided into 5 groups of 10 rats each in Table 1, separated and housed in different cages. They were kept under 12 hrs dark/day cycle in a serene environment. The animals were acclimatized for seven days and were fed with rat feed and water.

Induction of myocardial injury and treatment: Myocardial injury was induced by subcutaneous injection of adrenaline at a dose of 2 mg kg^{-1} body weight following a previously reported method[18]. Group 2 received 200 mg kg^{-1} of L-arginine dissolved in water by gavage method for 14 days and then induced with myocardial injury for 2 days. Group 3 was induced with myocardial injury for two days followed by administration of L-arginine (200 mg kg^{-1}). Group 4 received only L-arginine (200 mg kg^{-1}) for 14 days while group 5 was the myocardial injury group without treatment. At the end of the treatment period, all animals were euthanized using thiopentone sodium at the dose of 35 mg kg^{-1} body weight. Blood samples were collected by cardiac puncture into EDTA bottles for haematological analysis and plain sample bottles for biochemical analysis. The blood samples in the plain tubes were allowed to stand for two hours and then centrifuged at 3000 rpm for ten minutes to obtain the serum. The serum was stored at -20°C for subsequent use for biochemical analysis.

Determination of body weight: The initial and final body weight of each animal was obtained using an electronic

Table 1: Different experimental groups and their treatment

Groups	Number of rats	Dosage (mg kg^{-1} b.wt.)	Duration
1	10	Control (Untreated)	14 days
2	10	L-arginine (200 mg kg^{-1})	14 days
3	10	(L-arginine +MI) 200 mg kg^{-1} of L-arginine+2 mg kg^{-1} of adrenaline	14 days
4	10	(MI+L-arginine) 2 mg kg^{-1} of adrenaline+200 mg kg^{-1} of L-arginine	14 days
5	10	Adrenaline (2 mg kg^{-1})	14 days

weighing balance. The rats were weighed every day before drug administration. The final body weight was obtained before the animals were sacrificed.

Determination of haematological parameters: Haematological parameters were determined using a mid ray haematological auto-analyzer (model BC5300 Germany). Values for red blood cell count, packed cell volume, haemoglobin concentration, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC) were automatically calculated by the machine The result for each parameter was obtained in a print out from the machine.

Serum electrolyte assay: The serum sodium ion (Na^+) and potassium ion (K^+) concentrations were measured using the flame photometry method while the chloride ion (Cl^-) concentration was determined using the titrimetric, mercuric nitrate method. Bicarbonate ion (HCO_3^-) was measured using the back titration method.

Statistical analysis: The data obtained from the result was subjected to statistical testing using one-way ANOVA followed by Tukey test using GraphPad Prisms software 6.0. Data were expressed as Mean±Standard error of the mean (SEM) and $p<0.05$ was considered significant.

RESULTS

Bodyweight changes: The results for bodyweight changes are presented in Table 2. The mean initial body weight for each group at the start of the study was 157±5.6 g in the control group, it was 155±7.7 g in L-arginine only group, 156±6.8 g in L-arginine+MI treated group, 155±8.9 g in MI+L-arginine treated group and 155±5.3 g in MI only group. No statistical significance was observed in the mean initial body weights when comparing the groups. At the end of the experiment, the mean final body weight in the control group was 170±6.5 g, administration of L-arginine significantly increased ($p<0.05$) in L-arginine only group (176±2.6 g), when compared with L-arginine+MI, treated group (154±4.8 g), MI+L-arginine treated group (148±4.7 g) and MI only group 128±5.5 g. Also, the percentage weight gain in the control group (8.3%) was not significant when compared with L-arginine only group (7.1%), There was a significant ($p<0.05$) reduction of percentage weight gain in L-arginine+MI (-1.3%) and MI+L-arginine, (-1.9%) treated groups when compared with MI only group (-27 %) in MI only group. At the end of the study, the result showed that there was a significant ($p<0.05$) decrease in body weight in the MI only group, L-arginine+MI and MI+L-arginine treated groups when compared to the control group and L-arginine only group. No significant change was observed in the L-arginine group when compared to the control group.

Red blood cell parameters: The result for red blood cell (RBC) is presented in Fig. 1. Administration of L-arginine significantly ($p<0.05$) increased RBC count in L-arginine only group ($7.4\pm0.17\times10^{12}$ cell L^{-1}) when compared with the control group ($6.8\pm0.16\times10^{12}$ cell L^{-1}). Also in the two treated groups L-arginine+MI ($6.8\pm0.076\times10^{12}$ cell L^{-1}) and MI+L-arginine ($6.0\pm0.11\times10^{12}$ cell L^{-1}) the RBC was significantly higher when compared with MI only group ($5.1\pm0.063\times10^{12}$ cell L^{-1}). There was a significant ($p<0.05$) improvement in RBC count in L-arginine+MI treated group ($6.8\pm0.076\times10^{12}$ cell L^{-1}) when compared with MI+L-arginine treated group ($6.0\pm0.11\times10^{12}$ cell L^{-1}). Induction of MI in MI only group ($5.1\pm0.063\times10^{12}$ cell L^{-1}) resulted in a significant ($p<0.01$) decrease in RBC count when compared with the control group.

The result for hematocrit is presented in Fig. 2. The result for the control group was 48±0.98%, it was 50±0.65% in L-arginine only group, administration of L-arginine to L-arginine+MI (46±0.41%) in and MI+L-arginine (44±0.48%) treated rats significantly raised hematocrit level when compared with MI only group (41±0.41%) in MI only group. Similarly, hematocrit level was observed to be significantly ($p<0.05$) increased in the control group when compared with the MI only group.

The result for haemoglobin concentration is presented in Fig. 3. The haemoglobin concentration in the control group was 16±0.42 g dL^{-1}, it was 19±0.29 g dL^{-1} in L-arginine only group, 16±0.44 g dL^{-1} in L-arginine+MI treated group, 14±0.53 g dL^{-1} in MI+L-arginine treated group

Table 2: Bodyweight changes of control rats and rats treated with L-arginine before and after MI

Parameters	Mean initial body weight (g)	Mean final body weight (g)	Bodyweight gain (%)
Control	157±5.6	170±6.5	8.3[c]
L-arginine	155±7.7	176±2.6	7.1[c]
L-arginine+MI	156±6.8	154±4.8[a]	-1.3[ac]
MI+L-arginine	151±8.9	148±4.7[ac]	-1.9[ac]
MI only	155±5.3	128±5.5[ab]	-27[ab]

[a]p<0.05 compared to control, [b]p<0.05 compared to L-arginine+MI and [c]p<0.05 compared to MI

Table 3: Electrolytes and nitric oxide levels of control rats and rats treated with L-arginine before and after MI

Parameters	Na^+ (mMol L^{-1})	K^+ (mMol L^{-1})	Cl^- (mMol L^{-1})	HCO_3^- (mMol L^{-1})	NO (mMol L^{-1})
Control	142±1.2	3.8±0.3	109±1.2	24±0.58	28±2.3
L-arginine	133±0.67[a]	3.6±0.1	102±1.5[a]	21±0.1[a]	38±1.1[a]
L-arginine+MI	135±0.41[a+]	3.3±0.13[+]	111±1.5[a+]	21±0.6[a+]	29±0.47
MI+L-arginine	122±2.2[c]	4.5±0.3[c]	89±1.9[abc]	17±1.7[abc]	30±1.1
MI only	128±0.58[ab]	5.4±0.1[ab]	98±1.0[b]	21±1[a]	13±0.99[ab]

[a]p<0.05 compared to control, [b]p<0.05 compared to L-arginine+MI, [c]p<0.05 compared to MI, [+]p<0.05 vs MI+L-arginine and n = 10

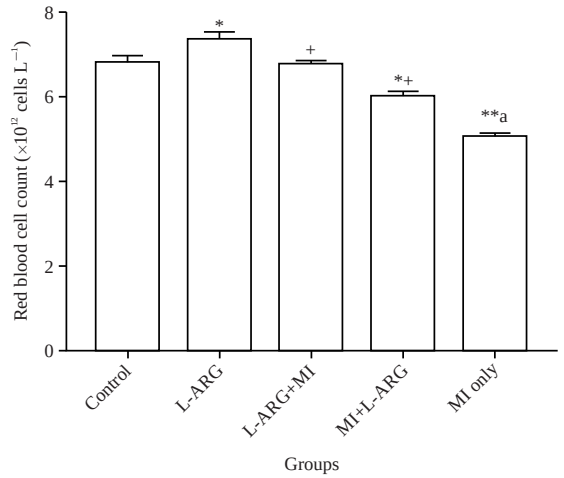

Fig. 1: Red blood cell count of normal control rats and rats treated with L-arginine before and after MI

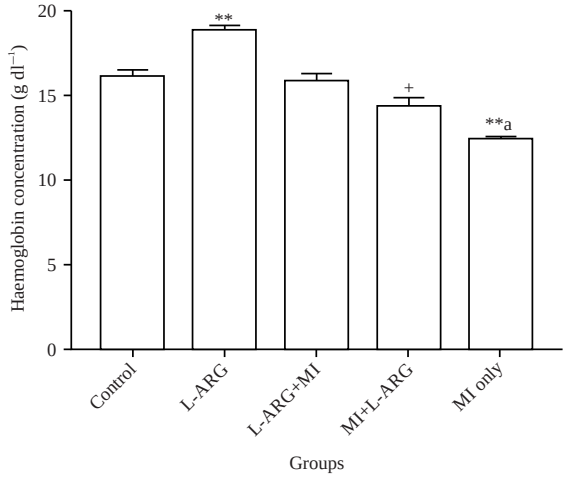

Fig. 3: Hemoglobin concentration of normal control rats and rats treated with L-arginine before and after MI

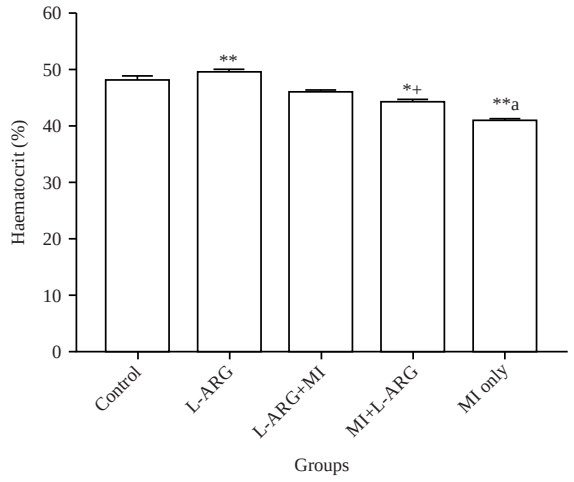

Fig. 2: Hematocrit of normal control rats and rats treated with L-arginine before and after MI

and 12±0.14 g dL^{-1} in MI only group. The concentration of haemoglobin in the L-arginine only group (19±0.29 g dL^{-1}) was significantly ($p<0.01$) increased when compared to the control group (16±0.42 g dL^{-1}) and MI only group (12±0.14 g dL^{-1}). Also, administration of L-arginine in the L-arginine+MI group (16±0.44 g dL^{-1}) significantly improved ($p<0.05$) haemoglobin concentration when compared to MI+L-arginine treated group (14±0.53 g dL^{-1}).

White blood cell count: The result for white blood cell count is presented in Fig. 4. The result showed a significant ($p<0.01$) increase in WBC in the MI only group (11.0±0.31×10^9 cells L^{-1}) when compared to the control (7.7±0.14×10^9 cells L^{-1}), L-arginine+MI (7.5±0.41×10^9 cells L^{-1}) and MI+L-arginine (8.3±0.16×10^9 cells L^{-1}) treated groups. No significant change was observed in L-arginine

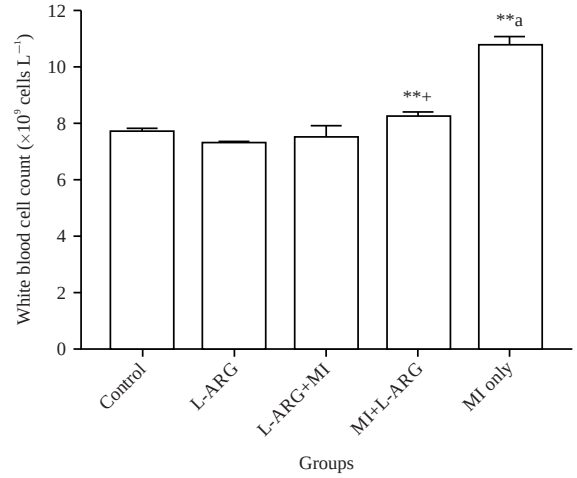

Fig. 4: White blood cell count of normal control rats and rats treated with L-arginine before and after MI

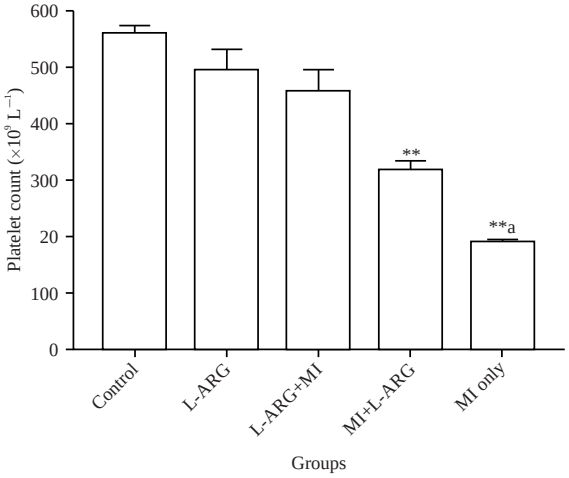

Fig. 5: Platelet count of normal control rats and rats treated with L-arginine before and after MI

only group ($7.3\pm0.058\times10^9$ cells L^{-1}). when compared with the control group ($7.7\pm0.14\times10^9$ cells L^{-1}) and L-arginine+MI treated group ($7.5\pm0.41\times10^9$ cells L^{-1}).

Platelet count: The result for platelet count is presented in Fig. 5. The result showed a significant ($p<0.01$) reduction in platelet count in MI only group ($190\pm4.3\times10^9$ cells L^{-1}) when compared to the control group ($559\pm15\times10^9$ cells L^{-1}). The platelet count was significantly ($p<0.05$) increased in L-arginine+MI ($456\pm39\times10^9$ cells L^{-1}) and MI+L-arginine ($318\pm16\times10^9$ cells L^{-1}) treated groups when compared to the MI only ($190\pm4.3\times10^9$ cells L^{-1}) group. No significant change was observed in L-arginine only group when compared to the control group.

Electrolytes and nitric oxide: The results for electrolytes and nitric oxide (NO) levels are presented in Table 3. The result showed a significant ($p<0.05$) decrease in serum Na^+ level in the MI only group (128 ± 0.58 mMol L^{-1}) when compared to the control group (142 ± 1.2 mMol L^{-1}), L-arginine only (133 ± 0.67 mMol L^{-1}) and L-arginine+MI treated group (135 ± 0.41 mMol L^{-1}). A significant ($p<0.05$) decrease in Na^+ level was also observed in the MI+L-arginine group (122 ± 2.2) when compared to the L-arginine+MI group (135 ± 0.41 mMol L^{-1}).

The result for serum K^+ level in MI only group (5.4 ± 0.1 mMol L^{-1}) showed a significant ($p<0.05$) increase when compared to the control group (3.8 ± 0.3 mMol L^{-1}), L-arginine only (3.6 ± 0.1 mMol L^{-1}) and L-arginine+MI treated group (3.3 ± 0.13 mMol L^{-1}). Also, there was a significant increase of K^+ level in the MI+L-arginine group (4.5 ± 0.3 mMol L^{-1}) when compared to L-arginine+MI treated group (3.3 ± 0.13 mMol L^{-1}).

The serum Cl^- level revealed a significant decrease ($p<0.05$) in MI only group (98 ± 1.0 mMol L^{-1}) when compared to the control (109 ± 1.2 mMol L^{-1}), L-arginine only group (102 ± 1.5 mMol L^{-1}) and L-arginine+MI treated group (111 ± 1.5 mMol L^{-1}). Similarly, there was a significant ($p<0.05$) decrease in MI+L-arginine treated group (89 ± 1.9 mMol L^{-1}) when compared with L-arginine+MI treated group (111 ± 1.5 mMol L^{-1}). The result for HCO_3^- in the control group was 24 ± 0.58 mMol L^{-1}, it was 21 ± 0.1 mMol L^{-1} in the L-arginine only group, 21 ± 0.6 mMol L^{-1} in L-arginine+MI treated group, 17 ± 1.7 mMol L^{-1} in MI+L-arginine treated group and 21 ± 1 in MI only group. The HCO_3^- level in the MI only group (21 ± 1 mMol L^{-1}) decreased significantly ($p<0.05$) when compared to the control group (24 ± 0.58 mMol L^{-1}). The HCO_3^- level increased significantly ($p<0.05$) in L-arginine+MI treated group (21 ± 0.6 mMol L^{-1}) when compared to MI+L-arginine treated group (17 ± 1.7 mMol L^{-1}). The NO level in the control group was 28 ± 2.3 mMol L^{-1}, it was 38 ± 1.1 mMol L^{-1} in L-arginine only group, 29 ± 0.47 mMol L^{-1} in L-arginine+MI treated group, 30 ± 1.1 mMol L^{-1} in MI+L-arginine treated group and 13 ± 0.99 mMol L^{-1} in MI only group. The NO level in the MI only group (13 ± 0.99 mMol L^{-1}) decreased significantly ($p<0.05$) when compared to the control group (28 ± 2.3 mMol L^{-1}). In the L-arginine only group (38 ± 1.1 mMol L^{-1}), the NO level increased significantly ($p<0.01$) when compared to the control group (28 ± 2.3 mMol L^{-1}). In rats pretreated with L-arginine before MI induction L-arginine+MI (30 ± 1.1 mMol L^{-1}), NO level was significantly ($p<0.05$) improved when compared to MI only group (13 ± 0.99 mMol L^{-1}).

DISCUSSION

L-arginine is a semi-essential amino acid involved in a myriad of significant beneficial bio-functions including the synthesis of proteins and human growth hormones secretion[19]. L-arginine as a precursor for protein and nitric oxide synthesis enhance dilation of blood vessels and blood supply to organs. The risk factors involved in myocardial injury are hypertension, sedentary lifestyle, increase in alcohol intake, increase blood cholesterol, smoking, diabetes and obesity[20]. Sedentary lifestyle has been associated with a high incidence of myocardial infarction[21]. The result of this study showed that adrenaline-induced myocardial injury resulted in a decreased body weight. This result agrees with a previous study where adrenaline was used to induce myocardial injury[16]. There was significant weight loss in myocardial injury rats without treatment and in myocardial injury rats before treatment with L-arginine when compared to the control group. The body weight of rats in the L-arginine treated group before inducing myocardial injury was significantly lower when compared to L-arginine only group. This result is in line with an earlier study that reported that dietary intake of L-arginine increases muscle gain with a decrease in body fat mass[22,23]. The reduction in body weight in myocardial injury rats is in agreement with a previous study that reports a depletion of cardiac and skeletal muscle glycogen in normal rats due to the adrenaline effect[24,25].

Blood parameters could reflect the physiological or pathological responsiveness of the body systems to the presence of drugs, chemicals, pathogens, toxicants or nutrients. The red blood cell count decreased significantly in the L-arginine+MI group compared to L-arginine only group. In the two treated groups, the result showed a significant increase in red blood cell count in the group given L-arginine before inducing myocardial injury (L-arginine+MI group) compared to the group treated with L-arginine after inducing myocardial injury (MI+L-arginine). The red blood cell count was also seen to decrease significantly in the myocardial injury group (MI only) without treatment when compared with the MI+L-arginine group. The increase in red blood cell count observed in L-arginine before MI (L-arginine+MI) group is an indication that L-arginine has the protective potential against conditions that may result in decreased red blood cells in myocardial injury rats. Myocardial injury was reported to cause a significant reduction in RBC count[26]. The decreased RBC count in myocardial injury rats could be due to the inhibitory effect of the inflammatory response to red cell production during the acute phase of myocardial injury on the bone marrow. It has been shown that myocardial injury is associated with a decrease in RBC and haematocyte levels[27]. The present result shows the ameliorating potential and improved RBC count of L-arginine against the possible anaemic state due to myocardial injury. This is in line with earlier studies that reported the beneficial effect of L-arginine against the destruction of a red blood cell[28,29]. Haemoglobin is very important in the transport of oxygen. The result of the hematocrit showed a significant increase in the L-arginine only group when compared to the control group. It also decreases significantly in myocardial injury rats without treatment when compared to the control group and MI+L-arginine group. In the two treated groups, the hematocrit value increased significantly in L-arginine+MI when compared to MI+L-arginine. The result is in agreement with a previous study that reported an increased hematocrit in rats treated L-arginine[30]. The result for haemoglobin concentration showed a significant decrease in MI only group when compared to the control group. Also, haemoglobin concentration was significantly higher in L-arginine only group when compared to the control group. In the two treated groups, the result shows a significant increase in the L-arginine+MI group when compared with the MI+L-arginine group. Whereas, haemoglobin concentration was seen to decrease significantly in MI only group when compared with MI+L-arginine treated group. In previous studies[31,32]. Both HB and PCV values increased significantly following administration of L -arginine. The results of our study are in agreement with these previous studies. The observation results could probably signify that there was an increase in erythropoiesis in animals treated with L-arginine. The lowered haemoglobin (Hb) and packed cell volume (PCV) counts in adrenaline-induced myocardial injury rats was significant compared to the control group. This may be due to insufficient blood flow due to the effect of oxidative stress in myocardial injury. It has been documented that reactive oxygen species might have a toxic effect on the erythroid maturation[33].

White blood cells are greatly involved in the immune functions of the body. The result of this study showed a significant increase in white blood cell count in the myocardial injury rats. Administration of L-arginine into L-arginine +MI and MI+L-arginine treated rats significantly reversed the increase of white blood cells to normal. White blood cell count has been known to be elevated in myocardial infarction and has a positive relationship with cardiac events[34]. The increase in WBC count in the MI group may reflect mobilization of the WBC to protect the body against infection.

Activation of NO synthase occurs when platelet is stimulated in humans. In this study, platelet count was seen to

decrease significantly in MI only group when compared with all other groups. In the two treated groups, platelet count increase significantly in the L-arginine+MI group when compared to the MI+L-arginine group. The increased platelet production seen in the L-arginine+MI group when compared with the MI+L-arginine group and MI only group is an indication that L-arginine can maintain the homeostatic role of blood platelet in pathological conditions such as myocardial injury. The generation of NO by L-arginine regulates platelet reactivity by increasing cyclic GMP. Thus, alteration in platelet activity via this pathway may have pathophysiological, physiological and therapeutic significance[35] as seen in myocardial infarcted rats in this study.

The results showed that the serum level of sodium and bicarbonate ions were significantly reduced while the level of potassium was raised in the MI only group when compared to the control group. Nitric oxide level was significantly reduced in MI only group when compared with the control. However, pre-treatment with L-arginine reversed hyponatremia, hyperkalaemia and normalized chloride ion and nitric oxide levels in MI rats. Hyponatremia and hyperkalaemia noted in this study reflects an imbalance in electrolyte handling and has been associated with acute myocardial injury. This study is in agreement with previous studies that reported hyponatremia and hyperkalaemia in acute myocardial infarction that was attributed to altered renal handling of electrolytes[36]. L-arginine has also been shown to induce fluid secretion and regulate electrolyte balance[37]. Electrolytes imbalance is an indication of abnormal conditions such as diabetes mellitus, stress, anaemia and myocardial infarction. L-arginine is a nitric oxide donor, probably helped to maintain water and electrolytes transport by modulating the absorptive capacity of the ileum and the kidney thereby regulating and maintaining serum electrolyte level[38,39]. MI results in a decrease in red blood cell count, red cell indices and electrolyte imbalance in addition to a reduction in body weight and serum level of nitric oxide. Administration of L-arginine reversed these alterations, especially as a preventive therapy. It is concluded that L-arginine, a precursor for nitric oxide, promotes erythropoiesis, raised serum nitric oxide level and maintains serum electrolytes while it has ameliorative effects on haematological parameters and serum electrolytes imbalance in myocardial injury rats.

CONCLUSION

This study revealed the importance of haematological parameters and serum electrolytes in myocardial injury. It also emphasized the changes in these parameters which was ameliorated by the administration of L-arginine. This study suggests that L-arginine, a precursor for nitric oxide, a vasodilator, prevents alterations in electrolytes and hematopoietic functions caused by adrenaline-induced myocardial injury in rats.

SIGNIFICANCE STATEMENT

This study discovered the importance of L-arginine in regulating electrolytes and haematological imbalances caused by myocardial injury in the body. The study shows that L-arginine can be beneficial in improving the haematological parameters and increasing the level of nitric oxide, a vasodilator. This study will help the researchers to uncover the critical areas of preventing electrolyte imbalance in myocardial injury that many researchers were not able to explore. Thus a new theory on preventing altered electrolyte imbalance in myocardial injury may be arrived at.

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