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Moringa oleifera extract (Lam) attenuates Aluminium phosphide-induced acute cardiac toxicity in rats

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ABSTRACT

Background: Moringa oleifera extract (Lam) has many antioxidant and protective properties. Objective: to investigate the antioxidant activities of Lam in counteracting the high oxidative stress caused by acute sub-lethal aluminium phosphide (AlP) intoxication in rat heart. These activities will be detected by histopathological examination and some oxidative stress biomarkers.
Methods: a single sub-lethal dose of Alp (2 mg/kg body weight) was administered orally, and Lam was given orally at a dose (100 mg/kg body weight) one hour after receiving AlP to rats.
Results: aluminium phosphide caused significant cardiac histopathological changes with a significant increase in malondialdehyde (MDA); lipid peroxidation marker; and a significant depletion of antioxidant enzymes (catalase and glutathione reductase). However, treatment with Lam protected efficiently the cardiac tissue of intoxicated rats by increasing antioxidants levels with slight decreasing in MDA production compared to untreated group.
Conclusions: This study suggested that Moringa oleifera extract could possibly restore the altered cardiac histopathology and some antioxidant power in AlP intoxicated rats, and it could even be used as adjuvant therapy against AlP-induced cardiotoxicity.

1. Introduction

Aluminium phosphide (AlP) is one of agrochemical pesticides that is used to increase agriculture production [1]. Furthermore, it extensively misused as suicidal poison due to low cost availability. In Egypt, AlP is emerging as a common self-poisoning agent [2].

AlP multisystem toxic involvement has been connected with phosphine gas and oxidative stress [3]. Phosphine gas induces oxidative stress through inhibition of enzymatic antioxidants e.g. catalase (CAT), glutathione, glutathione reductase (GR) and superoxide dismutase (SOD) [4]. Inhibition of SOD, CAT and GR will produce superoxide radicals and reduce nitric oxide (NO) bioavailability. The reduced NO level increases neutrophil adherence to coronary vessels with subsequent vasoconstriction. On the other hand, excess superoxide radicals react with NO enhancing lipid oxidation [5,6].These alterations will lead to cellular injury and apoptosis through peroxidation of membrane lipids and disruption of membrane permeability [7,8].

Cardiac tissue is more vulnerable to AlP induced oxidative stress than other human tissues, as it has an elevated oxidative metabolic activity and an increased polyunsaturated fatty acids content [9] [10–12]. To the extent that seventy percent of AlP related deaths were attributable to cardiovascular complication [13]. Impairment of cardiac functions could be detected by several echocardiographic techniques and indices [9].

Moringa oleifera (Lam) is an umbrella shaped tree, and is known as 'the miracle tree' due to its health benefit effect [14]. Lam has many natural antioxidant compounds e.g. flavonoids, ascorbic acid, carotenoids, and phenolics. Moringa; as phenolic containing compound, has cardio-protective effect and prevents oxidative myocardial cell damage through enhancing oxidative stress defence enzymes, preventing lipid membrane peroxidation [15,16], and inhibiting the disruption of mitochondrial membrane [17].

Given the evidence that Lam may have a role in managing of AlP acute toxicity, we investigated antioxidant activities of Lam in counteracting the high oxidative stress induced by acute AlP intoxication in rat heart.

Abbreviations: AlP, aluminium phosphide; Lam, moringa oleifera extract; CAT, catalase; GR, glutathione reductase; SOD, superoxid dismutase; MDA, malondialdehyde (product of lipid peroxidation); ROS, reactive oxidative stress

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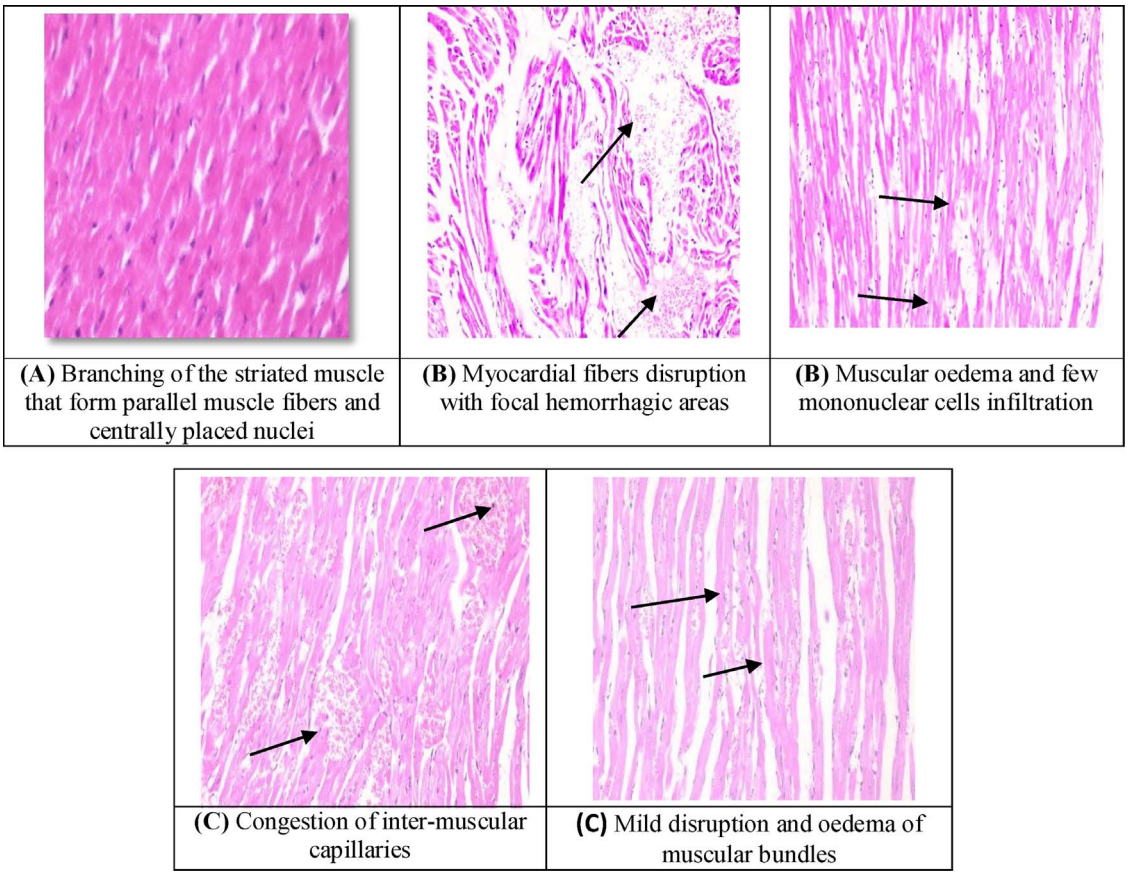

Fig. 1. Photomicrograph of heart section in group I (A), group II(B), and group III(C).

2. Materials and methods

In this study, the Lam antioxidant activities was detected histopathologically and biochemically through detection of malondialdehyde (MDA) concentration, (SOD), (CAT) and (GR) activities in rat heart. The study was ethically approved by the Institutional Animal Care and Use Committee (IACUC), Cairo University with number (**CU/III/S/41/17).**

2.1. Chemicals

Tablet form of aluminium phosphide (3 gm) was purchased from Sandhya Industries Pvt. Ltd., Gujarat, India. While Moringa extract (Lam) was purchased from Egyptian National Research Center (1 gm/mL aqueous preparations).

2.2. Animals and experimental design

Twenty-four male Wister rats weighting 100–135 g were used in the study. Animals were housed six cages (four rats/cage), kept under standard laboratory conditions; temperature was 25 ± 2 °C with 40% humidity and allowed free access on commercial diet and tap water provided *ad libitum*.

Rats were divided into three groups with eight animals each. Group I (control) was served as untreated rats and received 0.9% saline solution orally through gastric tube. Group II (AlP intoxicated rats) was given oral single sub-lethal dose of AlP (2 mg/Kg body weight) through gastric tube [18]. Group III (Lam treated group) was given oral single sub-lethal dose of AlP (2 mg/Kg body weight) and oral single dose of Lam (100 mg/Kg body weight) [4] one hour after receiving AlP dose. All groups were observed for 8 h then all rats were sacrificed under pentobarbital anaesthesia by decapitation.

2.3. Histopathological examination of heart tissue

Full thickness heart samples from each group were fixed in 10% neutral buffered formalin. The fixed specimens were then trimmed, washed and dehydrated in ascending grades of alcohol, cleared in xylene, embedded in paraffin, sectioned at 4–6U thickness and stained by hematoxylin and eosin dye for photo microscopic examination according to Bancroft et al.[19].

2.4. Assessment of oxidative stress biomarkers in heart tissue

Heart specimens were minced and homogenized (10%) in ice-cold 1.155 KCl-0.01 M sodium and potassium phosphate buffer (pH 7.4) in a Potter–Elvehjem glass homogenizer. The homogenate was centrifuged at 10,000 rpm for 20 min at 4 °C, and the resultant supernatant was separated and analyzed to estimate malondialdehyde (MDA) concentration, superoxide dismutase (SOD), glutathione reductase (GR), and catalase (CAT) activities.

Lipid peroxidation, (MDA) level, in heart homogenates was measured spectrophotometrically (Boeco S-20 Spectrophotometer, Hamburg, Germany) using Biodiagnostic kit (Egypt) following Okhawa et al. [20] method.

(CAT) (U/g), (SOD) (U/g) and (GR) (U/g) activities were detected spectrophotometrically (Boeco S-20 Spectrophotometer, Hamburg, Germany) using Biodiagnostic kit (Egypt) following Okhawa et al. [20], Aebi [21], Nishikimi et al. [22] and Goldberg and Spooner [23] respectively.

2.5. Statistical analyses

Data were coded and analyzed using the statistical package SPSS version 24. Quantitative variables were presented in mean and standard