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Effects of aqueous garlic extract on cadmium-induced cardiac damage in Wistar rats

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Abstract

Cadmium (Cd) is a widespread environmental toxicant that accumulates in the food chain and induces oxidative stress, DNA damage, and apoptosis. This study investigated the cardioprotective effects of aqueous *Allium sativum* (garlic) extract against cadmium-induced cardiac damage in adult male Wistar rats. Thirty-two rats were randomly assigned to four groups (n = 8): Group I (control) received tap water; Group II received CdCl? (5 mg/kg/day); Group III received aqueous A. sativum extract (200 mg/kg/day); and Group IV was pre-treated with A. sativum extract (200 mg/kg) 90 minutes before CdCl2 administration for 30 days. Blood and heart tissues were collected for biochemical and histopathological analyses. Cadmium exposure caused general weakness, reduced food and water intake, and significantly lower body weight gain compared with the control and treatment groups, although organ weights were unaffected. Lipid peroxidation was significantly elevated in the Cd-treated group, while the activities of antioxidant enzymes, including superoxide dismutase, catalase, and glutathione peroxidase, were significantly reduced ($p < 0.05$). Serum lactate dehydrogenase activity was also significantly increased, indicating cardiac injury. Histological examination revealed myocardial fiber necrosis, cytoplasmic vacuolization, myofibrillar disorganization, and structural irregularities in the Cd-treated rats. Pre-treatment with aqueous A. sativum extract markedly attenuated these biochemical and histological alterations, preserving cardiac architecture and improving antioxidant status. These findings demonstrate that cadmium induces oxidative cardiac damage in adult male Wistar rats and suggest that aqueous A. sativum extract exerts a protective effect by reducing oxidative stress and limiting myocardial injury, supporting its potential as a cardioprotective therapeutic agent against cadmium-induced toxicity.

Keywords: *A.sativum. Cadmium, Antioxidants, Oxidative stress, Wistar rats*

1. Introduction

The heart is a muscular organ that pumps blood through the circulatory and vascular systems which circulate

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it around the body. The rat heart has a quadrangular pyramidal form, which is very similar to the human heart (Anderson *et al.*, 2006). In vertebrates, it is in the anterior portion of the thorax and is encircled by a thin, transparent pericardium (Orheruata *et al.*, 2018). The heart is mesodermal in origin and develops from the splanchnopleuric mesoderm located anterior to the prechordal plate (Singh and Pal, 2007). The mammalian four-chambered heart has double circulation located in the thorax while the rat heart comprises two distinct pumps; the right side and left side of which are housed in the same organ due to their shared evolutionary history. The physical examination of the heart of the Wistar rat revealed that it has a structure typical of a mammalian heart (Orheruata *et al.*, 2018), with a clearly defined Atrioventricular (AV) groove separating the atria from the ventricles. The chordae tendineae of the mitral valve are attached to the anterior and posterior papillary muscles of the left ventricle, which have relatively long, slender apical parts that protrude into the ventricular lumen (Anderson *et al.*, 2006). The main sources of Cd poisoning in non-occupationally exposed populations are; food, drinking water, and cigarette smoking (Ferramola *et al.*, 2012). Occupational exposure to Cd occurs during mining, working with Cd-containing ores, pigments, and paints, manufacturing of electronics and batteries, and burning of fossil fuels (Tucker *et al.*, 2008). Soluble Cd2+ salts accumulate and injure various tissues, including the heart, kidney, liver, brain, lung prostate, and adenohypophysis depending on the exposure, time, and dose (Nazimabashir *et al.*, 2015). Studies on animals have shown that acute Cd exposure can cause cardiovascular problems by altering the antioxidant defense system (Ferramola *et al.*, 2012), resulting in the pathogenesis of hypertension, cardiotoxicity (Priya *et al.*, 2017), atherosclerosis (Messner *et al.*, 2009), and stroke (Tellez-Plaza *et al.*, 2013), myocardial infarction and peripheral arterial disease (Novelli *et al.*, 2000) but confirmatory evidence is sparse, and the mechanisms behind these interactions are still unknown (Messner *et al.*, 2009). Most of the Cd in the body is bound to Metallothionein (MT), a part of cysteine-rich, low molecular weight and ubiquitous intracellular proteins with high affinity for metals (Ferramola *et al.*, 2012). Cd forms a complex with metallothionein by durable combinations with the apoprotein thionein (Nazimabashir *et al.*, 2015). In doses as low as 0.1 μM, Cd may accumulate in the heart muscle and cause cardiotoxicity (Ferramola *et al.*, 2012), Cd also affects cardiac function, changing the composition, shape, structure of the heart (Nazimabashir *et al.*, 2015). Several medicinal plant products rich in antioxidant phytoconstituents have been reported to be beneficial against Cd-induced oxidative stress-mediated damages in cardiac tissues (Bhattacharjee *et al.*, 2019). The present study was designed to evaluate the efficacy of aqueous *A. sativum* extract against Cd-induced oxidative stress-mediated damages in the heart of male Wistar rats.

Garlic (*Allium sativum) i*s an aromatic herbaceous plant and one of the oldest and most important herbs that is consumed worldwide as food and traditional medicine for different diseases (Batiha *et al.*, 2020). Garlic has also been shown to improve vascular function (Bradley *et al.*, 2016) and has been reported to possess several biological properties including anticarcinogenic, antioxidant (Parvu *et al.*, 2019 no reference), antidiabetic, anti-atherosclerotic (Bradley *et al.*, 2016), antibacterial, and antifungal (Parvu *et al.*, 2019), and antihypertensive activities in traditional medicines.

2. Materials and methods

Thirty-two (32) adult male Wistar rats weighing between 180-250 g were randomly divided into four groups (n = 8 per group):

Group I (Control): Received only tap water.

Group II ($CdCl_2$-treated): Received cadmium chloride ($CdCl_2$) at 5 mg/kg body weight.

Group III (*A. sativum*-treated): Received aqueous extract of *Allium sativum* at 200 mg/kg body weight.

Group IV (Co-treatment): Pre-treated with *A. sativum* extract (200 mg/kg) 90 minutes prior to $CdCl_2$ (5 mg/kg) administration.

All treatments were administered via oral gavage once daily for 30 consecutive days.

Chemicals and plant extract preparation. Cadmium chloride ($CdCl_2$) was purchased from LOBA Chemical Laboratory, India, and sourced locally via Adfolak Chemical Shop, Ibadan, Nigeria. Fresh *Allium sativum* (garlic) bulbs were obtained from Bodija Market, Ibadan. The bulbs were washed, peeled, and 50 g was ground into a paste. This was mixed with 100 mL of distilled water, stirred, and filtered to obtain an aqueous extract

(stock solution). The extract was stored at 4 °C and freshly prepared weekly. The dose for administration was calculated using the formula:

Dose (mL) = [Required dose (mg/kg) × Body weight (kg)]/Concentration (mg/mL)

2.1. Sample collection

At the end of the treatment period, rats were fasted overnight and euthanized under light anesthesia. Blood samples were collected via cardiac puncture into plain tubes for cardiac enzyme assays. The hearts were excised, rinsed in ice-cold saline, and divided for biochemical and histopathological analyses.

2.2. Biochemical assays

Cardiac tissue was homogenized in Phosphate-Buffered Saline (PBS), centrifuged, and the supernatants were used for assessing: Lipid peroxidation measured as Thiobarbituric Acid Reactive Substances (TBARS) (Szymonik-Lesiuk *et al.*, 2003). Superoxide Dismutase (SOD) activity using the method of Misra and Fridovich (Paoletti and Mocali, 1990). Catalase (CAT) activity as per Aebi (1984).

Glutathione Peroxidase (GPx) activity following Rotruck *et al.* (1973). Lactate Dehydrogenase (LDH) levels were determined spectrophotometrically using a commercial kit.

2.3. Histopathological examination

Heart tissues were fixed in 10% neutral buffered formalin, dehydrated, and embedded in paraffin. Sections (5 µm) were stained with Hematoxylin and Eosin (H&E). Slides were examined under a light microscope for structural changes, including myofiber integrity, necrosis, cytoplasmic vacuolization, and myofibrillar arrangement.

2.4. Statistical analysis

All data were expressed as mean ± Standard Deviation (SD). Statistical comparisons were performed using one-way ANOVA, followed by Tukey's post hoc test for multiple comparisons via GraphPad Prism version 8.4. A p-value of < 0.05 was considered statistically significant.

3. Results

The behavioral observations throughout the study revealed distinct patterns among the experimental groups. Rats in the control and *A. sativum* groups exhibited high activity levels, while those in the *A. sativum* + Cd group displayed mild activity. Notably, the Cd-treated group (Cd group II) exhibited sullen behavior compared to other groups. Furthermore, the Cd-treated rats displayed signs of general body weakness, and reduced food and water intake, leading to a decrease in weight gain. Gait abnormalities, such as hyperabduction of the head, external rotation of limbs, and hind limb hyperabduction, were observed in some Cd-treated animals, suggesting potential neurological impacts (Table 1).

Table 1: Body weight (g) of rats

Group	Initial Body Weight (Day 1) Mean ± SD	Final Body Weight (Day 30) Mean ± SD	Weight Gain	% Weight Gain
Control	188.67 ± 27-95	248.50 ± 10.3	59.83	13.69
Cd	207.38 ± 8.345	222.88 ± 8.0	15.5[a]	3.6
A.sativum	208.63 ± 6.806	240.25 ± 6.8	31.62[ab]	7.04
A.sativum+cd	219.63 ± 11.03	238.38 ± 11.36	18.75[b]	4.09

The mean and SD of heart weight of the Control group I (0.69 ± 0.13), the Cd group II (0.76 ± 0.038), the *A. sativum* group III (0.66 ± 0.02), and the *A. sativum*+ Cd group IV (0.63 ± 0.01). However, further investigations into cardiac markers and biochemical parameters provided more insights (Figure 1). Protein concentration in the Cd group II significantly increased compared to the control group I and other treated groups at $P < 0.05$. In contrast, the *A. sativum* group III showed a significant decrease compared to the Cd group II. The *A. sativum* +

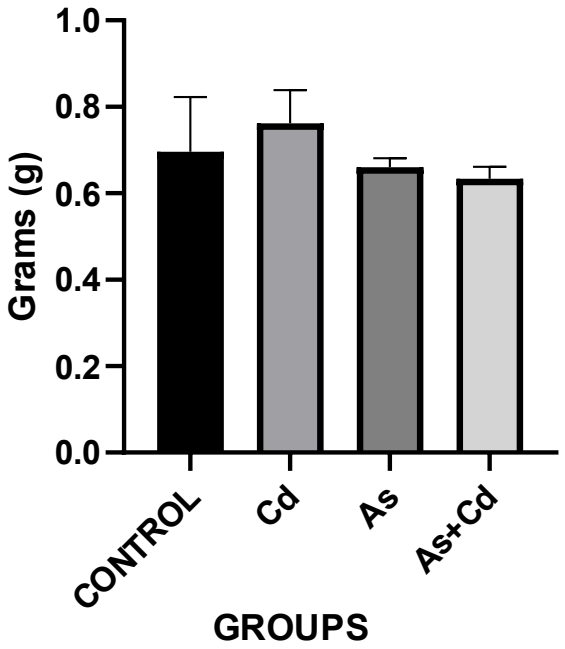

Figure 1: Heart Weight

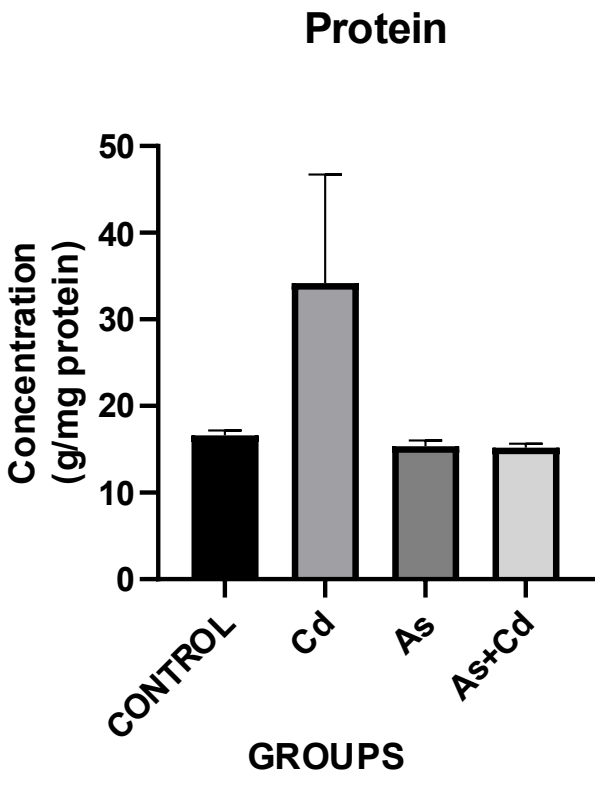

Figure 2: The difference in protein concentration among groups. The control group I, Cd group II, *A. sativum* group III, and *A. sativum* + Cd group IV. Values (n = 5) are presented as mean ± SD at P < 0.05. Cadmium group vs control (**) p = 0.0149, *Allium sativum* group vs Cadmium (@) p = 0.0064, and *Allium sativum*-cadmium group vs cadmium group (##) p = 0.0060

Cd group IV also displayed reduced protein concentration compared to the Cd group II at $P < 0.05$ (Figure 2). Superoxide Dismutase (SOD) activity showed a significant reduction in the Cd group II compared to the control and other treated groups. Conversely, the *A. sativum* group III exhibited increased SOD activity compared

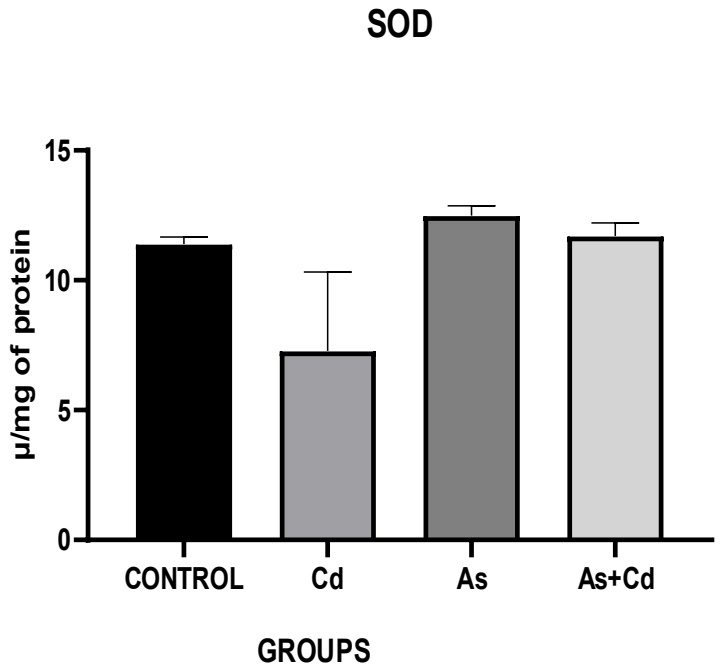

Figure 3: The activity of SOD level among the control group I, Cd group II, *A. sativum* group III, and *A. sativum* + Cd group IV. Values (n = 5) are presented as mean ± SD at P < 0.05. Cadmium group vs control (*) p = 0.0297, *Allium sativum* group vs Cadmium group (@) p = 0.0039, and *Allium sativum*-cadmium group vs cadmium group (#) p = 0.0121

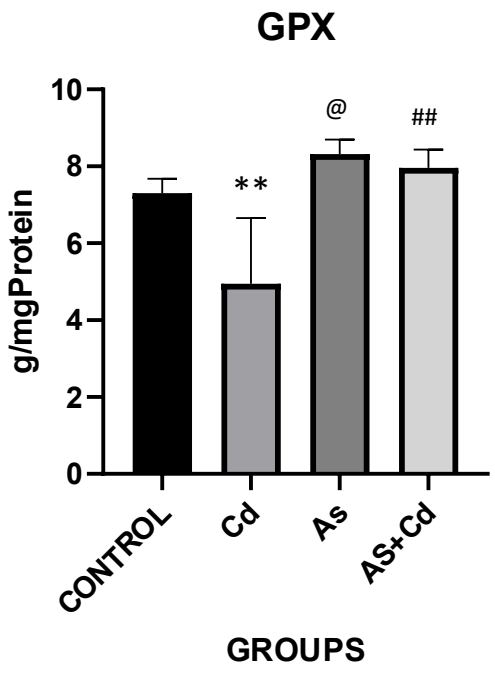

Figure 4: The mean GPx level among the control group I, Cd group II, *A. sativum* group III, and *A. sativum* + Cd group IV. Values (n = 5) are presented as mean ± SD at P < 0.05. Cadmium group vs control (**) p = 0.0305, *Allium sativum* group vs cadmium (@) p = 0.0032 and *Allium sativum*-cadmium group vs cadmium group (##) p = 0.0032

to the control and Cd group II at P < 0.05 (Figure 3). Glutathione Peroxidase (GPx) activity decreased significantly in the Cd group II compared to the control and other treated groups. However, the *A. sativum* group III displayed elevated GPx activity compared to the Cd group II. Additionally, the *A. sativum* + Cd group IV showed increased GPx activity compared to Cd group II (Figure 4).

Catalase (CAT) activity significantly decreased in the Cd group II compared to the control and other treated groups. Both *A. sativum* groups (III and IV) displayed increased CAT activity compared to the Cd group II at P < 0.05 (Figure 5). Malondialdehyde (MDA) levels significantly increased in the Cd group II compared to the control and other treated groups. Conversely, the *A. sativum* group III exhibited decreased MDA levels compared

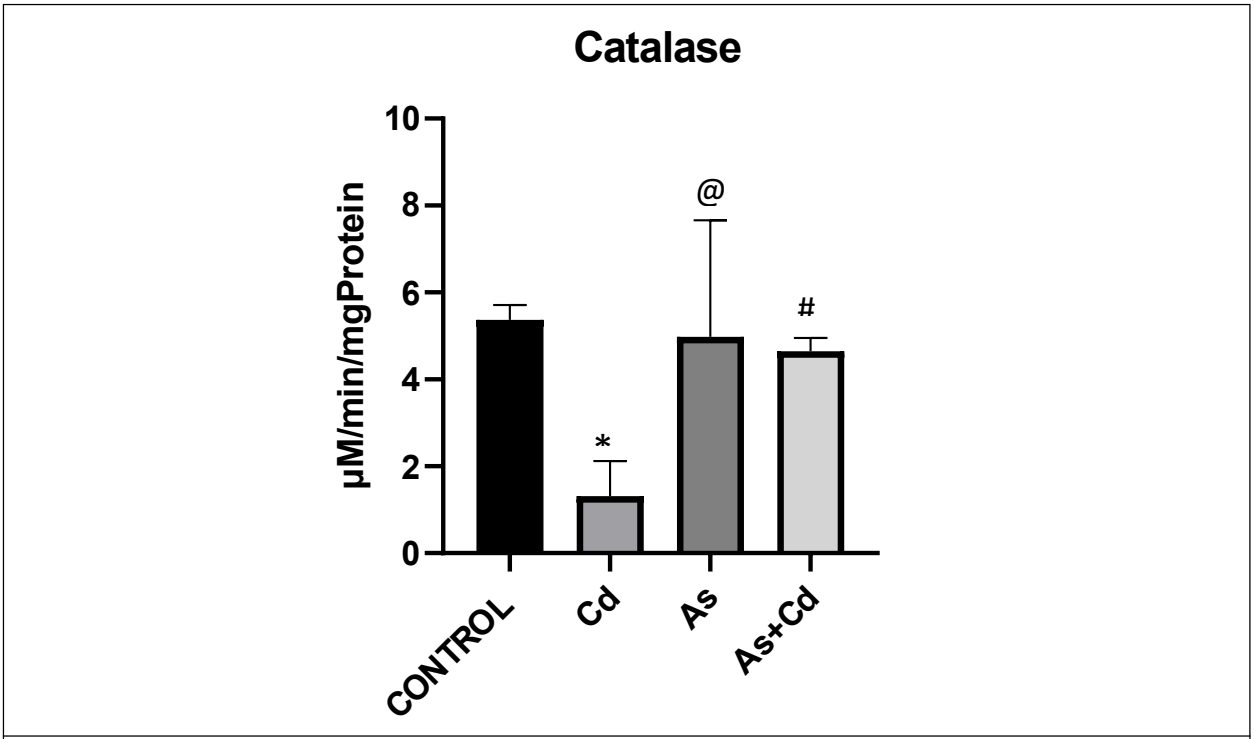

Figure 5: The difference in the activity of CAT among the control group I, Cd group II, *A. sativum* group III, and *A. sativum* + Cd group IV. Values (n = 5) are presented as mean ± SD at P < 0.05. Cadmium group vs control (*) p = 0.0160, *Allium sativum* group vs cadmium group (@) p = 0.0278, *Allium sativum*-cadmium group vs cadmium group (#) p = 0.0453

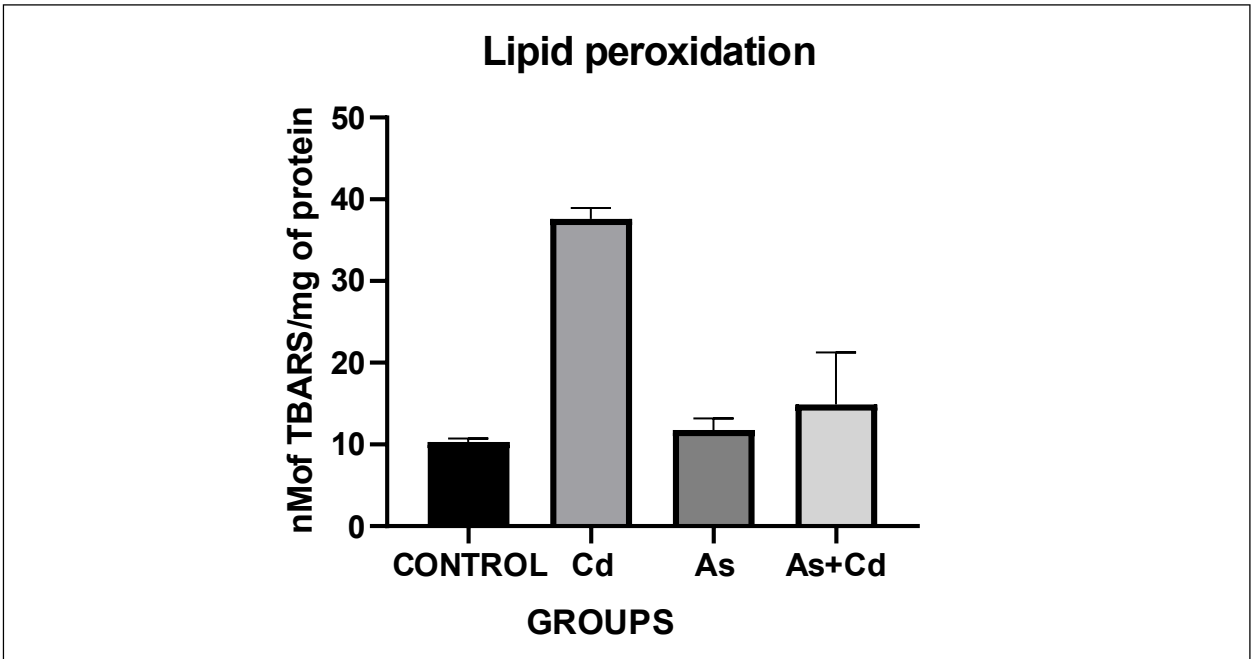

Figure 6: The mean MDA level among the control group I, Cd group II, *A. sativum* group III, and *A. sativum* + Cd group IV. Values (n = 5) are presented as mean ± SD at P < 0.05. Cadmium group vs control group (****) p < 0.0001, *Allium sativum* group vs cadmium group (@) p = <0.0001, *Allium sativum*-cadmium group vs cadmium group (###) p = 0.0003

to the Cd group II. The *A. sativum* + Cd group IV also displayed reduced MDA levels compared to Cd group II at P < 0.05 (Figure 6). Lactate Dehydrogenase 1 (LDH1) concentration in the serum significantly increased in the Cd group II compared to the control and *A. sativum* group III. However, the *A. sativum* group III showed a significant decrease in LDH activity compared to the Cd group II at P < 0.05 (Figure 7).

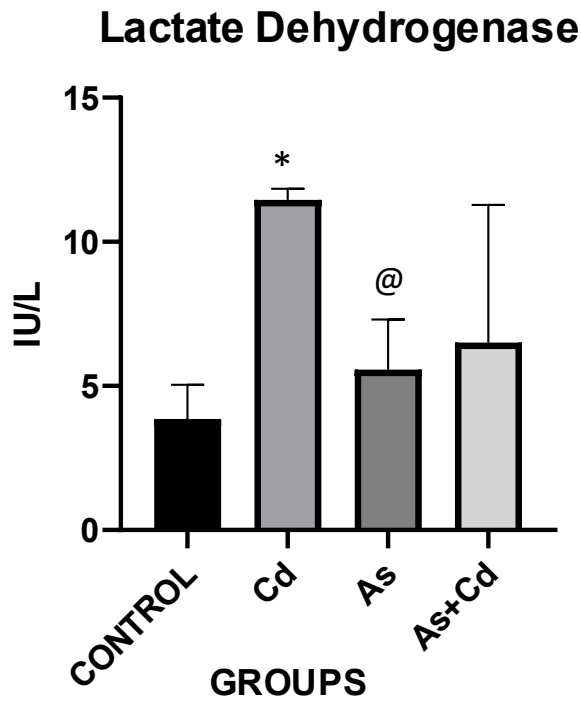

Figure 7: The mean LDH1 concentration among the control group I, Cd group II, *A. sativum* group III, and *A. sativum* + Cd group IV. Values (n = 5) are presented as mean ± SD at P < 0.05. Cadmium group vs control (*) p = 0.0166, *Allium sativum* group vs Cadmium group (@) p = 0.0455

PLATE 1: Photomicrograph of longitudinal sections of the left ventricle of the heart of adult Wistar rats stained with H&E for X100. (A) Control group I displays a normal architecture of the left ventricle, with branched cardiac muscle fibers containing centrally placed nuclei. (B) Cd group II shows myocardial fiber waviness (blue arrow) and disorganization of cardiac muscle fibers. (C) *A. sativum* group III displays a normal architecture of the myocardium with a regular arrangement of muscle fibers, the presence of blood vessels, and centrally located nuclei. (D) *A. sativum* + Cd group IV shows some restoration of myocardial fibers with no vacuolated cytoplasm and the presence of capillaries among the muscle fibers (N-Nucleus, MF-Cardiac muscle fiber, CA-Capillary).

PLATE 2: Photomicrograph of transverse sections of the left ventricle of the heart of adult Wistar rats stained with H&E for X400. (A) Control group I displays the normal architecture of the left ventricle with branching and anastomosing cardiac muscle fibers and central elongated nuclei. (B) Cd group II shows disarrangement of cardiac muscle fibers, hypertrophic muscle fibers (blue arrow), and cytoplasmic vacuolated cells (yellow arrow). (C) *A. sativum* group III displays the normal architecture of the myocardium with the regular alignment of muscle fibers and nuclei. (D) *A. sativum* + Cd group IV shows some restoration of myocardial fibers (N-Nucleus, MF-Cardiac muscle fiber, CA-Capillary, BV-Blood vessel).

PLATE 3: Photomicrograph of longitudinal sections of the left ventricle of the heart of adult Wistar rats stained with H&E for X400. (A) Control group I displays the normal architecture of the left ventricle with a regular arrangement of muscle fibers, intercalated discs connecting the fibers, and intact cells. (B) Cd group II shows disorganization of cardiac muscle fibers, hypertrophic muscle fibers (blue arrow), and myocardial fiber waviness (yellow arrow). (C) *A. sativum* group III displays the normal architecture of the myocardium with a regular arrangement of muscle fibers and capillaries present among cardiac muscle fibers. (D) *A. sativum* + Cd group IV shows restoration of some myocardial fibers (O-Nucleus, MF-Cardiac muscle fiber, ID-Intercalated disc, BV-Blood vessel, CA-Capillary).

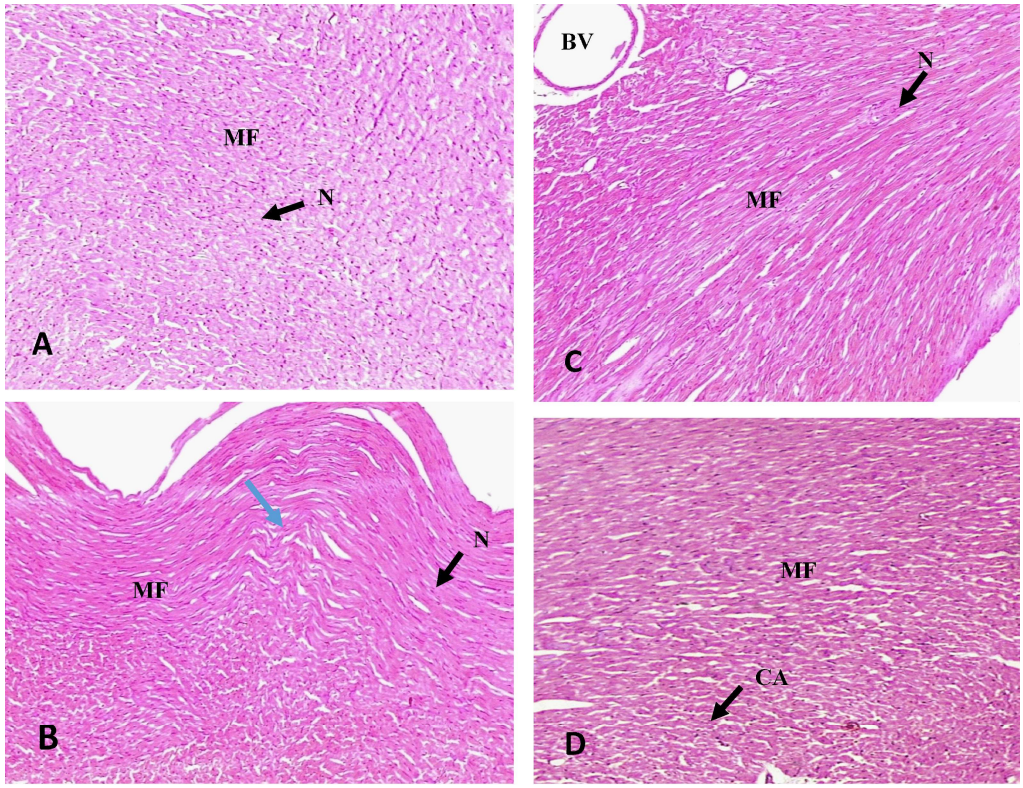

PLATE 1: Photomicrograph of longitudinal sections of the left ventricle of the heart of adult Wistar rats, H&E, X100

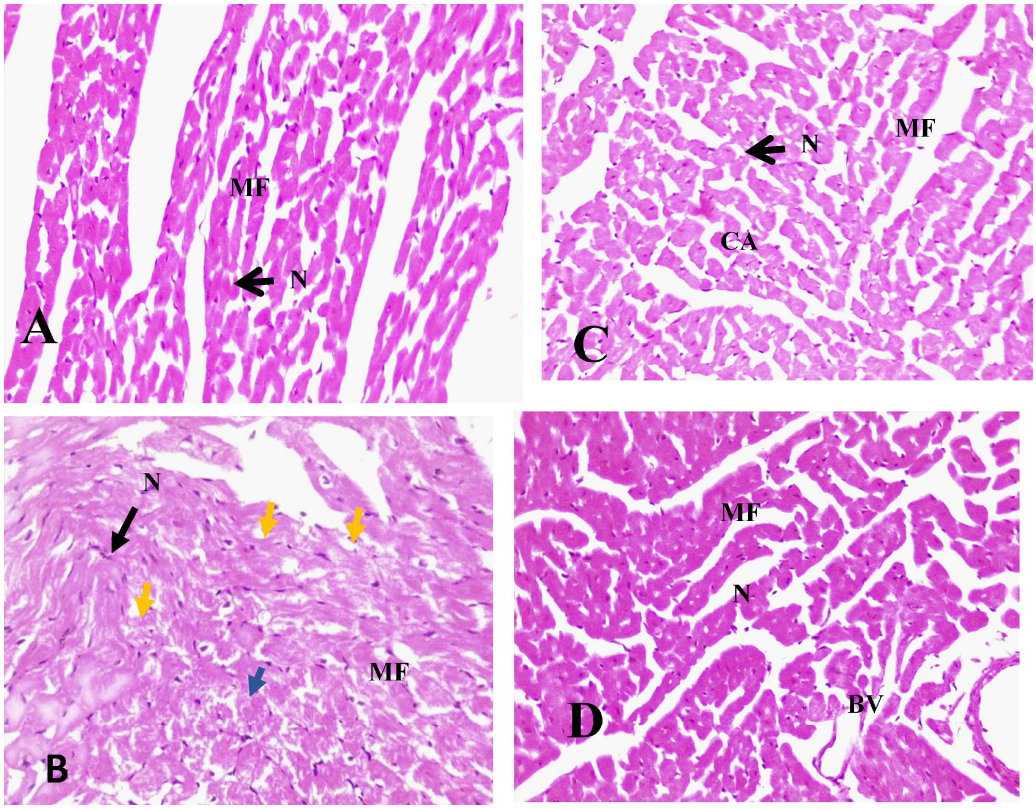

PLATE 2: Photomicrograph of transverse sections of the left ventricle of the heart of adult Wistar rats, H&E, X400

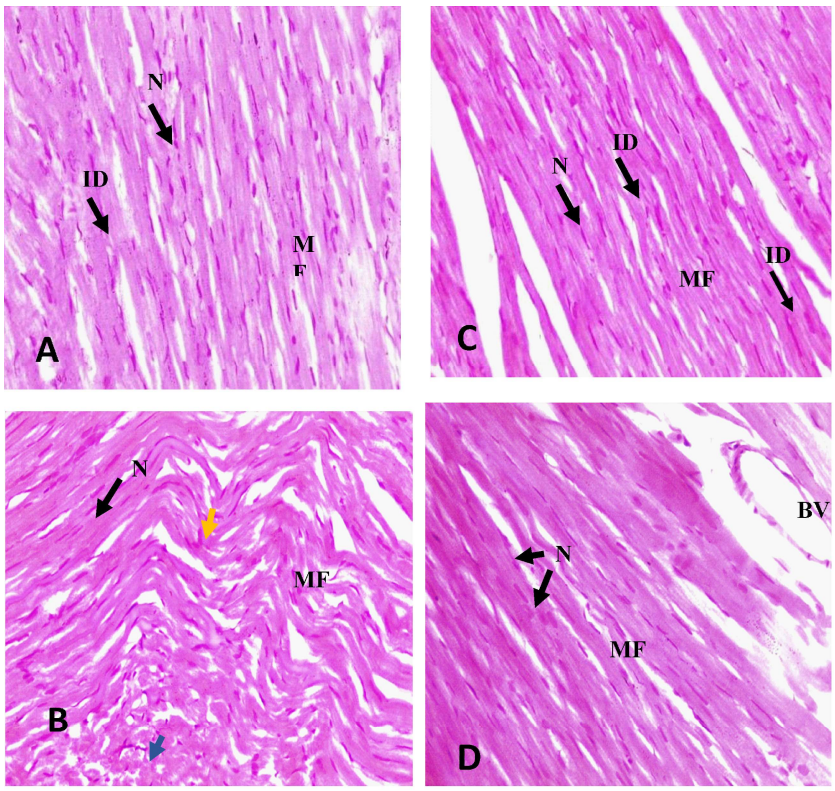

PLATE 3: Photomicrograph of longitudinal sections of the left ventricle of the heart of adult Wistar rats, H&E, X400

4. Discussion

In this study, toxicity of the Cd is associated with oxidative stress-mediated damages which may be associated with loss in the total body weight, distortion of the cellular antioxidant defense system, and increase in the serum level of LDH1. These changes observed in Cd-treated rats were attenuated with *A. sativum* aqueous extract (200 mg/kg body weight) because it possesses antioxidant properties that inhibit ROS activity in the body. Findings in this study showed a significant reduction in the percentage weight gain of the Cd group II (15.5 g) when compared to the control group I (59.83 g) and *A. sativum* group III (31.62 g). There was a slight increase in the percentage weight gain of the *A. sativum*+ Cd group IV (18.75 g) compared to Cd group II, and alteration in body weight induced by Cd intoxication was slightly restored upon pre-administration of aqueous extract of *A. sativum*. The weight gain in the Control group I is the highest. Values (n = 5) are presented as mean ± SD at $P < 0.05$. This result agrees with (Nazimabashir *et al.*, 2014) who suggested that Cd accumulation causes disturbances in the total body weight of rats and may be due to tissue damage and reduction in their functions. Changes in body weight serve as a marker that is used for checking the animal health status, and a reduction in body weight serves as an indicator of the deterioration in the health of the experimental rats during the experiment (Nazimabashir *et al.*, 2014). The decreased body weight recorded in the Cd-treated groups may be a result of a reduction in food and water intake following the administration of Cd. The retardation in growth may also be attributed to the direct effect of Cd on growth, as Cd-induced toxicity involves the induction of oxidative stress resulting in alterations in the antioxidant status, thereby leading to severe metabolic disorders and inability to gain weight (Poli *et al.*, 2022). There was no statistically significant difference in the organ weight (g) of animals across the groups. Although the Cd group II (0.76 ± 0.038), has a higher mean organ weight compared to other groups, this aligns with the report of Bhattacharjee *et al.* (2019), they reported that Cd administration elevates the weight of the heart tissues which may lead to organomegaly. The present dose of Cd (5 mg/kg body weight) resulted in three animal mortalities during the treatment period. In this study, there was a significant decrease in the level of SOD (μ/mg of protein) at $P < 0.05$ in the Cd group II (7.28 ± 1.52). However, a statistically significant increase at $P < 0.05$ was recorded in rats pre-treated with an aqueous extract of *A. sativum* (12.50 ± 0.37), the levels were elevated near the control levels. Under normal

physiological conditions, the mammalian body produces *ROS* that are generated inside the cells and are removed by endogenous antioxidants. An imbalance between the antioxidant defense and oxidants leads to oxidative stress (Priya *et al.,* 2017). The reduced activity of SOD in the Cd-treated group II may be due to the replacement of Zn^{2+} ions by Cd. The direct binding of Cd ions to the active sites of this enzyme or utilization of more enzymes in scavenging the oxidants produced during Cd toxicity can be said to be another reason why there was loss of catalytic function of SOD (Priya *et al.,* 2017). However, when the rats were pre-treated with an aqueous extract of *A. sativum* for 30 days, they demonstrated an increased level of activity of this enzyme up to control group I animals. This aligns with the result of the studies of Kilikdar *et al.* (2013), who reported that an aqueous extract of *A. sativum* can exert its antioxidant effect directly via free radical scavenging or indirectly by upregulating cellular antioxidant enzymes such as SOD. The SOD activity in the cardiac tissues of rats of the control group I and *A. sativum* group III exhibited no significant changes indicating not only that the extract has the potential to alleviate oxidative stress but also may be considered safe for consumption as a natural antioxidant. Catalase decomposes hydrogen peroxide and converts it to water and diatomic oxygen. In this study, a significant reduction in the activity of CAT (μM/min/mg protein) was recorded in the Cd group II (1.32 ± 0.40; $P < 0.05$), and an increase in the CAT activity level when rats were pre-administered with *A. sativum* before Cd administration (4.64 ± 0.31). The CAT activity in the cardiac tissues of rats of the control group I (5.37 ± 0.34) and *A. sativum* group (4.98 ± 1.54) exhibited no significant changes. This result aligns with the studies of Shaikh *et al.* (1999), who reported that Cd-induced alterations in the CAT activity in cardiac tissue can be described as an adaptive response to oxidative stress. Reduction in the activity of CAT in the cardiac tissues of rats in the Cd-treated group may be caused by the deprotonation of the nitrogen atom of the imidazole ring in His-74 due to Cd interaction (Wang *et al.,* 2015). However, Cd-induced alterations on CAT enzyme activity were protected when the rats were pre-administered with an aqueous extract of *A. sativum* showing the potent antioxidant properties of *A. sativum* as reported by the studies of (Kilikdar *et al.,* 2013), that *A. sativum* can exert its antioxidant effect directly via free radical scavenging or indirectly by upregulating cellular antioxidant enzymes such as CAT. This study further demonstrates that after Cd treatment, the levels of GPx decreased significantly in the rat cardiac tissue of Cd group II compared with the control group I and other experimental groups, there was a significant increase in the GPx activity level up to control levels when rat were pre-administered with *A. sativum* before Cd administration at $P < 0.05$. A statistically significant increase in the activity of GPx was also noted in the rats treated with aqueous extract of *A. sativum* only when compared to the Control group I (Figure 4). This aligns with the studies of Mitra *et al.* (2015), who reported a decrease in the activity of the GPx enzyme of the Cd-Cd-treated group compared with the control group and other experimental groups. This shows that the glutathione metabolizing pathway is disturbed in Cd-treated rats (Mitra *et al.,* 2015). GPx oxidizes GSH to GSSG by utilizing hydrogen peroxide as its substrate (Priya *et al.,* 2017).

Lipid peroxidation is a potent indicator of oxidative damage (Weisberg *et al.,* 2003), MDA is the breakdown product of the major chain reactions leading to the oxidation of polyunsaturated fatty acids and thus serves as a reliable marker of oxidative stress-mediated LPO in myocardial tissue (Sahma *et al.,* 2003). Biochemical assays in this study show that MDA (nMol of TBARS) was increased in cardiac tissues of Cd group II (37.62 ± 1.71) indicating accentuated oxidative damage. There was a significant decrease in the MDA levels of rats in *A. sativum*+ Cd group IV (14.92 ± 3.77). There was no significant difference in the MDA level in the *A. sativum* group III (11.76 ± 1.41) and control group I (10.30 ± 0.43) at $P < 0.05$. The result of this study correlates with Alpsoy *et al.* (2014) and Priya *et al.* (2017), in their studies, they observed a significant increase in the cardiac content of MDA following Cd administration. Previous studies have reported that Cd-induced lipid peroxidation was diminished by plant extracts (Alpsoy *et al.,* 2014). Pre-treatment with an aqueous extract of *A. sativum* successfully decreased the levels of lipid peroxidation possibly by scavenging the harmful effects of free radicals generated during Cd toxicity, thereby protecting the tissues from undergoing oxidative damage, this is consistent with Cha (1987), who reported on the heavy metal chelating activity of *A. sativum*. Results from this study prove that the rat hearts were susceptible to ROS-mediated damage, evident from the higher levels of MDA found in the cardiac tissues. Lactate dehydrogenase 1 is an enzyme that serves as an indicator of myocardial damage upon the loss of functional integrity of membrane architecture resulting in the leakage of this enzyme from the heart into the bloodstream (Priya *et al.,* 2017). In this study, biochemical estimations of this marker enzyme showed a profound increase in the serum Cd-treated animals following Cd administration

(11.45 ± 0.39) at $P < 0.05$, this is consistent with the result of Bhattacharjee *et al.* (2019). However, rats pre-treated with aqueous extract of *A. sativum* protected the cardiac tissues since the levels of LDH1 in their serum were significantly reduced (6.50 ± 2.39, $P < 0.05$). The antioxidant property of *A. sativum* might have strengthened the heart tissue membrane integrity, thereby preventing Cd-induced membrane damage indicating that this extract may have the capacity to protect the rat myocardial tissue (Priya *et al.,* 2017).

About histological alterations, Hematoxylin and eosin-heart tissue sections of Cd intoxicated rats revealed that the Cd administration with the present dose for the thirty consecutive days brought about significant alterations to the myocardium of the heart. These alterations included loss of cardiac myofibers, vacuolization of cytoplasm, hypertrophy of muscle fibers, irregularity, and disarrangement of the myofibrils in cardiac tissues. Here again, the histological appearance of rats pre-treated with aqueous extract of *A. sativum* was found to be significantly improved. This result is consistent with a previous report by (Alpsoy *et al.,* 2014), that cardiac tissue is susceptible to Cd-induced cardiotoxicity, reinforced by another research, Mitra *et al.* (2015) in their work, they revealed that the myocardium is highly susceptible to damages caused by Cd after Cd treatment every alternate day for 15 days. The histoarchitecture of the control group I and *A. sativum* groups appeared normal with no damage to the tissue morphology proving that the extract not only can protect cardiac tissues against oxidative stress but also does not cause tissue damage, indicating again that the *A. sativum* may be considered safe for human consumption in future (Kilikdar *et al.,* 2013).

5. Conclusion

The study highlights the diverse effects of Cd exposure and *A. sativum* treatment on behavior, biochemical parameters, and cardiac markers in rats. The observed alterations in oxidative stress markers and enzyme activities suggest potential cardioprotective effects of *A. sativum* against Cd-induced toxicity. The study emphasizes the importance of further research to elucidate the underlying mechanisms and clinical relevance of these findings.

As it may serve as a possible nutritional intervention and the extract itself or fractions that may be obtained from it may be used as a future nutritional supplement to fight oxidative stress-induced cardio-toxicity in persons exposed to Cd.

Recommendation

Further studies are required for adjudging the long-term effects of the *A. sativum* extract on animal study. It is worth elucidating the molecular mechanisms that go beyond *A. sativum* extract cardio-protection in Cd intoxication.

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