Leaf Pulp of *Carpobrotus deliciousus* Attenuate Sodium Arsenate Induced Toxicity

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Abstract: *Carpobrotus deliciousus* (CD) is reported to have numerous health benefits. This study determined the influence of the oral and repeated administration of leaf pulp of *C. deliciousus* (LPCD) on sodium arsenate (SA) (Na_3AsO_4) induced toxicity in Wistar rats. Thirty male Wistar rats were distributed into five groups of six rats each. Group 1 was given 5 mg/kg body weight (*b.w*) of SA for 7 days; group 2 was administered 0.5 mL of LPCD for 14 days. Group 3 received 5 mg/kg b.w of SA for 7 days followed by 0.5 mL LPCD for another 7 days, while group 4 was treated with 0.5 mL LPCD for 7 days followed by 5 mg/kg *b.w* of SA for another 7 days. Group 5 served as the control and received deionized water. After the last treatment, the blood, kidneys, and liver were collected for biochemical analysis. The alkaline phosphatase (ALP), AST, and GPT levels were all significantly ($p < 0.05$) depleted in groups 1 and 2, while there was no significant ($p < 0.05$) change in groups 3 and 4 when compared to the control. The HDL, LDL, cholesterol, and triglyceride levels were not significantly ($p < 0.05$) altered for groups 3 and 4. These findings suggest that oral and repeated exposure to LPCD may ameliorate managing SA-induced toxicity.

Keywords: sodium arsenate; toxicity; *Carpobrotus deliciousus*; leaf pulp; organ damage

1. Introduction

Arsenic (As), a naturally-occurring metalloid, is not essential for plant growth, but it can accumulate in plants to toxic levels. As a result, it can enter the food chain and pose a health risk to humans [1]. It is found to occur ubiquitously in nature in the earth's crust. Major routes of exposure to humans may be via ingestion through drinking water, with relatively minor routes being inhalation and skin absorption [2]. A wide range of alterations has been investigated in arsenic poisoning. Arsenic has been reported to induce hematological and biochemical changes as well as oxidative stress in a chicken model of arsenic intoxication and other models [3]. Epidemiological and mechanistic experimental evidence of arsenic carcinogenesis in animals and humans are also available [4]. The mechanisms of arsenic toxicity have been reported to include the induction of oxidative stress, inhibition of enzyme

and mitochondrial function, and induction of stress-response genes [5,6]. Arsenic produces various clinic-pathological conditions, the major effects being cardiovascular, cerebrovascular, and peripheral vascular diseases, besides skin alterations and skin cancer, developmental anomalies, neurological and neurobehavioral disorders, diabetes, hearing loss, portal fibrosis, hematologic disorder (anemia, leukopenia, and eosinophilia) and carcinoma [7-10]. However, the clinical features of arsenic toxicity vary between individuals, population groups, and geographic areas. There seem to be variations in the clinical presentations of arsenic toxicity or the organs of the body targeted, and the factors responsible for this are not yet understood [11,12].

Some studies stated that chronic exposure to inorganic arsenic caused renal damage in humans and experimental animals [13-15]. Histopathological studies have shown that arsenic caused significant damage to the kidney [16,17].

The search for a specific, reliable, and safe treatment for arsenic toxicity continues [18]. Several medicinal plants and phytochemicals exhibited significant protection from experimentally induced arsenic toxicity in animal models [19]. The use of plant and plant products to treat diseases is as old as humanity. The major merits of plant-based medicine seem to be their perceived efficacy, low incidences of serious adverse effects, and low cost [20]. The Carpobrotus species are very well known for their medicinal abilities, particularly *Carpobrotus edulis,* due to its notorious antibacterial and antiseptic properties [21]. The leaf juice is astringent and mildly antiseptic. It is mixed with water and swallowed to treat diarrhea, dysentery, and stomach cramps. It is also used as a gargle to relieve laryngitis, sore throat, and mouth infections [22]. Leaf juice or a crushed leaf of the plant is a famous soothing cure for blue-bottle stings-being a coastal plant. It is luckily often on hand in times of such emergencies [23]. The leaf juice of the species has a gently calming effect on various forms of injuries that might occur at different parts of the body, such as skin surfaces. [24]. Sweet liquids prepared from the fruit relieve constipation and promote emptying the bowel. A concoction made from leaf juice and other ingredients such as honey has been effective against tuberculosis since time immemorial; the leaf juice also relieves the itch from mosquito, tick, and spider bites both for people and animal companions [23]. As part of investigations into the protective roles performed by *C. deliciousus* the present study sought to investigate the effects of the *C. deliciousus* on sodium arsenite-induced hematological and biochemical alterations in Wistar rats.

2. Materials and Methods

2.1. Plant material and extraction of the leaf pulp.

The fresh leaves of *C. edulis* were collected in March 2018 within the South Campus, Nelson Mandela University of Port Elizabeth, South Africa, and were identified by the curator of the Botanical garden of the same University. The leaves were placed in a juice extractor, and the juice extracted. The process was repeated to get the desired quantity needed.

2.2. Animal treatment.

Twenty male Wistar albino rats weighing 160–210 g were used in this study. They were housed in a well-ventilated animal house and were fed standard rat pellets, and allowed access to drinking water ad libitum. The animals were randomly assigned into four groups, comprising

five rats per group. The rats were allowed to acclimatize for 14 days before extract administration commenced.

GROUP 1:   Deionized water for 7 days (Control)

GROUP 2:   5 mg/kg of Na_3AsO_4 administered for 7 days (Arsenic Only)

GROUP 3:   Pretreated with 0.5 mL of LPCD for the first 7 days followed by an oral dose of sodium arsenite (5 mg/kg b.w) for 7 days (Pre-treated).

GROUP 4:   An administered oral dose of sodium arsenite (5 mg/kg b.w) for 7 days and 0.5 mL of LPCD rally for 7 days (Therapeutic)

2.3. Methods.

2.3.1. Determination of creatinine and urea**.**

Creatinine and urea levels in the blood were determined according to the methods described by Bartel *et al*. [25] and Fawcett and Scott [26], respectively.

2.3.2. Plasma bilirubin determination

The dimethyl sulfoxide method described by Tietz *et al*. [27] was used to determine bilirubin concentration in the plasma.

2.3.3. Determination of creatinine.

This was carried out according to the method described by Tietz *et al*. [27]

2.3.4. Determination of urea.

The method of Veniamin and Vakirtzi – Lemonias [28] was used to determine urea concentration.

2.3.5. Determination of bilirubin.

The dimethyl sulfoxide method described by Tietz *et al*. [27] was used for bilirubin determination.

2.3.6. Lipid profile.

Total cholesterol, triglyceride and HDL, and LDL – cholesterol The CHOD-PAD enzymatic colorimetric method of Trinder [27] was used.

2.3.7. Determination of Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities.

Activities of AST and ALT were determined following the principle described by Reitman and Frankel [30].

2.4. Statistical analysis.

Data generated from this study were expressed as means ± standard error of means (SEM) of three independent experiments and analyzed using the GraphPad Prism 6 software. The significance of difference was calculated using one way ANOVA with the level of significance set at $P \leq 0.05$

3. Results and Discussion

3.1. Blood chemistry and hematological parameters.

The result of hematological result is shown in Table 1. There was a significant decrease in the RBC level in all the groups at $p<0.05$ when compared to the control (8.76 ± 0.8). The RBC count for the arsenic only, Pre-treated and the therapeutic group are 6.23 ± 1.8, 6.62 ± 1.4, and 5.16 ± 0.6, respectively, while the WBC count increased in the arsenic only group (11.67 ± 0.4), the WBC count for the arsenic only, pretreated and the therapeutic group are 15.80 ± 0.2, 10.80 ± 0.3 and 10.20 ± 0.1 respectively. There was a significant decrease in the RBC level in all the groups at $p<0.05$ compared to the control. The mean Hb concentrations were 13.68 ± 0.1 g/dl, 9.18 ± 0.4 g/dl, 10.38 ± 1.4 g/dl, and 8.60 ± 1.1g/dl for the control, arsenic only, Pretreated and Therapeutic groups, respectively, the arsenic only and the therapeutic groups had a significantly ($P<.05$) higher Hb concentration when compared with the control. The PCV level was significantly decreased in the arsenic only (69.95 ± 1.4), the pretreated (73.60 ± 1.8) and the therapeutic group (76.83 ± 1.2) when compared with the control group (77.48 ± 1.2). The MCV of the Arsenic only group was significantly ($P<.01$) higher than that of the other groups. However, the MCV values of the Pretreated and therapeutic groups were not significantly different from that of the control group. The mean corpuscular hemoglobin (MCH) of the arsenic-only group is significantly elevated in the arsenic-only group compared to the other groups. The MCH value for the control group was 16.617 ± 1.5 (pg) while the values for the arsenic only, the pretreated and the therapeutic group were 15.550 ± 0.2 (pg), 15.817 ± 0.6 (pg) and 16.717 ± 0.3 (pg) respectively. Also, the MCHC of the control group was 28.683 ± 2.1 (g/dL) while the values for the Arsenic only, the pre-treated and the therapeutic group were 25.083 ± 0.8 (g/dL), 17.117 ± 0.5 (g/dL) and 17.450 ± 0.4 (g/dL) respectively. The MCH and MCHC of the control, pretreated and therapeutic groups were not significantly different. The Mean Platelet Volume (MPV) values were significantly reduced $p<0.05$ in the arsenic (7.650 ± 0.1 fL) and pretreated group (7.933 ± 0.4 fL) when compared with the control group (8.483 ± 0.1 fL), while there was no significant difference between the Control group and the therapeutic group (8.117 ± 0.1 fL).

Table 1. Hematological parameters in Wistar rats exposed to sodium arsenite and leaf pulp of *C. deliciousus*

	Control	Arsenic Only	LPCD + As (Pre-treated)	LPCD + As (Therapeutic)
RBC x 10⁶μL	8.76 ± 0.8	6.23 ± 1.8^a	6.62 ± 1.4^a	5.16 ± 0.6^a
WBC x 10⁶μL	11.67 ± 0.4	15.80 ± 0.2^a	10.80 ± 0.3^b	10.20 ± 0.1^b
Hb g/dL	13.68 ± 0.1^b	9.18 ± 0.4^a	10.38 ± 1.4^b	8.60 ± 1.1^a
MPV(fL)	8.483 ± 0.1^b	7.650 ± 0.1^a	7.933 ± 0.4^a	8.117 ± 0.1^b
PCV (%)	50.85 ± 0.2^b	37.76 ± 0.5^a	38.73 ± 1.2^a	32.63 ± 1.1^a
LYM(%)	77.48 ± 1.2^b	69.95 ± 1.4^a	73.60 ± 1.8^b	76.83 ± 1.2^b
NEU(%)	20.16 ± 0.6^b	26.70 ± 0.8^a	23.00 ± 1.1^b	20.66 ± 0.6^b
MCV(fL)	58.05 ± 0.8^b	64.133 ± 1.3^a	58.46 ± 1.1^b	58.217 ± 1.7^b
MCH (pg)	16.61 ± 1.5^b	15.550 ± 0.2^a	15.55 ± 0.2^a	16.717 ± 0.3^b
MCHC(g/dL	16.71 ± 0.3^b	25.083 ± 0.8^a	17.11 ± 0.5^b	17.450 ± 0.4^b

KEY: Red blood cell (RBC), White Blood Cell (WBC), Hemoglobin (Hb), Mean Platelet Volume (MPV), Packed Cell Volume (PCV), Lymphocyte (LYM), Neutrophils (NEU) Mean Corpuscular Volume (MCV), Mean Corpuscular Haemoglobin (MCH) and Mean Corpuscular Haemoglobin Concentration (MCHC). All values are expressed as mean ± Standard Error of Mean (S.E.M), (n = 5); [a] values are significantly different when compared with the control group ($p<0.05$); [b] values are significantly different from the arsenate only group ($p<0.05$).

The result of the electrolyte level is shown in Table 2. Sodium (Na^+) and Potassium (K^+) level were elevated in the therapeutic group. The pretreated and the control groups are significantly different at $p<0.05$ compared with the arsenic-only group. While Chlorine (Cl^-) and Calcium (Ca^{2+}) level is significantly reduced in all the groups compared with the control group.

Table 2. Changes in Electrolyte level in Wistar rats exposed to sodium arsenite and leaf pulp of *C. deliciousus*.

S/N	Na^+	K^+	Cl^-	Ca^{2+}
Control	114.33 ± 5.81^b	3.86 ± 0.32	119.50 ± 10.77^a	15.97 ± 2.07^a
Arsenic only	118.33 ± 3.84^a	3.63 ± 0.11	116.66 ± 5.53^a	10.69 ± 0.20^b
As + LPCD (Therapeutic)	120.00 ± 2.65^{ab}	3.96 ± 0.32	104.50 ± 10.28^a	14.44 ± 0.48^a
LPCD+As (Pretreated)	114.50 ± 3.48^b	3.91 ± 0.26	101.83 ± 9.17^a	13.05 ± 1.17^a

Sodium (Na^+), Potassium (K^+), Chlorine (Cl^-) and Calcium (Ca^{2+}). All values are expressed as mean ± Standard Error of Mean (S.E.M), (n = 5)

The Urea level is elevated in the arsenic (17.36 ± 2.17 mg/L) and therapeutic group (17.83 ± 1.42 mg/L) when compared to the control group (15.63 ± 1.81 mg/L), while in the pretreated group (15.63 ± 1.81 mg/L), Urea level declined. Creatinine level was significantly elevated in the Arsenic group (1.42 ± 0.31 g/L) group when compared with the control (0.90 ± 0.35 g/L), the pre-treated (0.82 ± 0.21 g/L) and the therapeutic (0.89 ± 0.29 g/L) group. The total bilirubin level in the arsenic only group (7.57 ± 0.89 g/L) is significantly elevated than the control (5.87 ± 1.42 g/L), therapeutic group (6.52 ± 1.38 g/L), and the pretreated group (6.00 ± 2.27 g/L).

Table 3. Kidney function test in Wistar rats exposed to sodium Arsenite and leaf pulp of *C. deliciousus*.

S/N	Urea (mg/L)	Creatinine (g/L)	TB (g/L)	DB (g/L)
Arsenic Only	17.36 ± 2.17^a	1.42 ± 0.31^a	7.57 ± 0.89^a	0.73 ± 0.22^a
As + LPCD (Therapeutic)	17.83 ± 1.42^a	0.89 ± 0.29^b	6.52 ± 1.38^{ab}	0.70 ± 0.24^a
LPCD+As (Pretreated)	15.63 ± 1.81^b	0.82 ± 0.21^{ab}	6.00 ± 2.27^b	0.93 ± 0.30^{ab}
Control	16.95 ± 1.99^b	0.90 ± 0.35^b	5.87 ± 1.42^b	0.78 ± 0.19

TB-Total Bilirubin, DB- Direct Bilirubin. All values are expressed as mean ± Standard Error of Mean (S.E.M), (n = 5)

[a]-values are significantly different from the control group, [b]-values are significantly different from the arsenate only group

The results of oral administration of 0.5 ml of leaf pulp of *C. deliciousus* and 5 mg/kg of Sodium Arsenite on the levels of Aspartate Transaminase (AST), Glutamic pyruvic transaminase (GPT), and Alkaline Phosphatase (ALP) are shown in Figure 1. There is an increase in the Arsenic only group when compared with the control group. A significant ($p < 0.05$) reduction in AST level was seen in the pretreated group compared with the control group and the group given As only, the AST level in the therapeutic group was significantly reduced compared with the Arsenic group. There was a reduction in the GPT activity in both the therapeutic and Pretreated groups compared to the control group and the group that received arsenic only. ALP level is significantly elevated in all the groups compared to the control, but significantly, there was a significant reduction in the pretreated and therapeutic groups compared with the Arsenic only group.
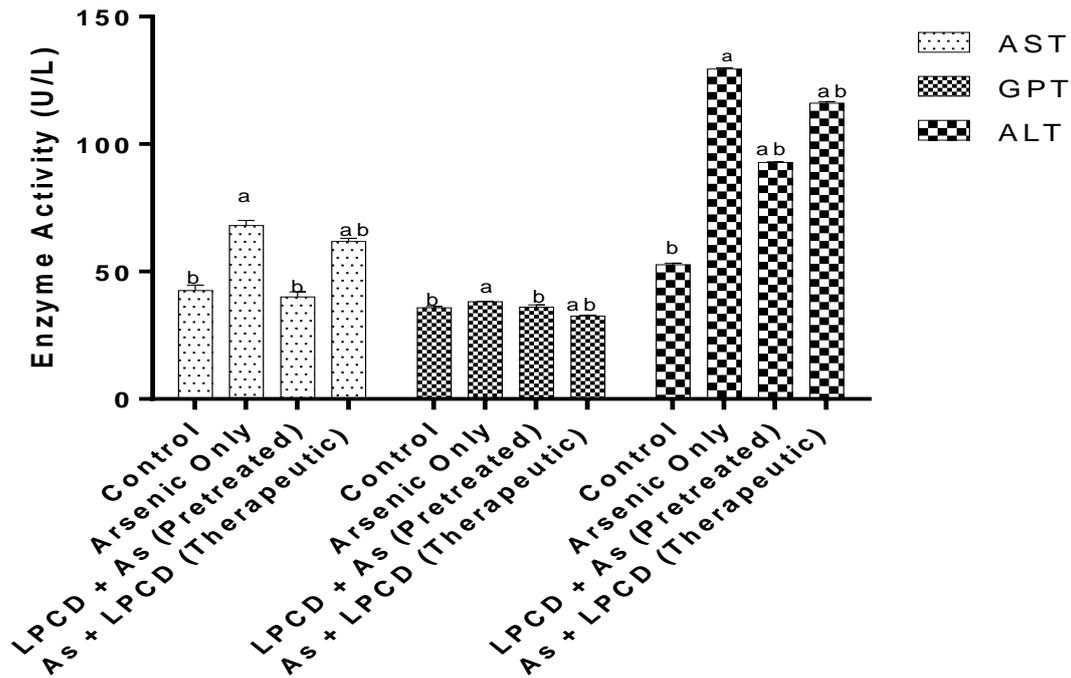
Figure 1. Liver Enzyme activity in Wistar rats exposed to sodium arsenite and leaf pulp of *C. deliciousus*.

The results of oral administration of 0.5 ml of leaf pulp of *C. deliciousus* and 5 mg/kg of Na_3AsO_4 on the lipid profile are shown in Figure 2. The total cholesterol significantly decreased at $p < 0.05$ in the pretreated and the therapeutic groups compared to the arsenic-induced group, while there was no significant difference between the pretreated and control groups. The triglyceride level was significantly reduced in all the groups compared to the arsenic group, while there was no significant difference between the control and therapeutic groups at $p < 0.05$. The HDL level significantly increased in all the groups compared with the Arsenic only group. No significant difference was seen in-between groups for the control, pretreated, and therapeutic groups. The LDL cholesterol significantly decreased $p < 0.05$ in all the groups compared with the arsenic-induced group.
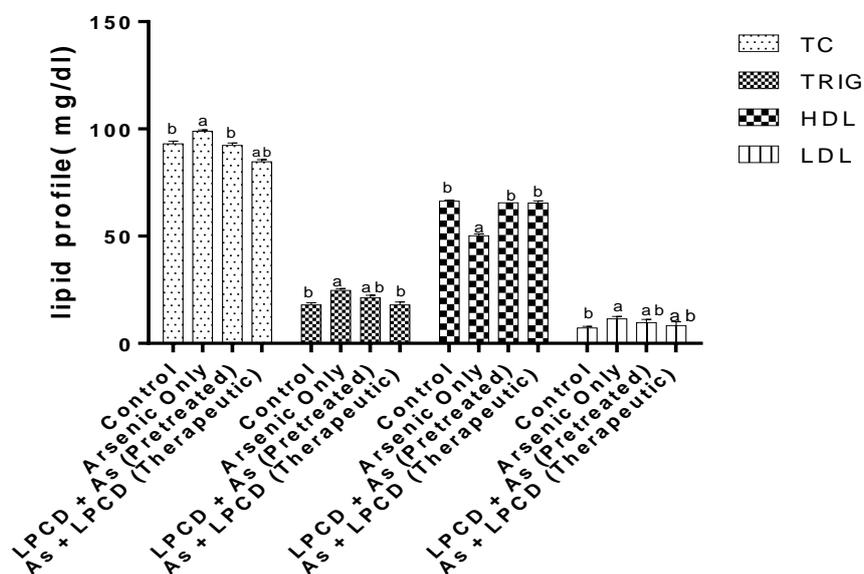

Figure 2. Lipid profile of Wistar rats exposed to sodium arsenite and leaf pulp of *C. deliciousus*.

3.2. Antioxidant activity of C. deliciousus.

In the liver, the antioxidant enzymes (SOD and CAT) activity significantly increased $p<0.05$ in all the groups compared with the arsenic group. There was no difference in the pretreated and the therapeutic groups compared with the control group. The GSH activity was only significantly elevated in the therapeutic group compared with the arsenic group. There was no significant difference in the control and the pretreated groups compared with the arsenic group. While in the kidney, the activity of SOD, CAT, and GSH was significantly elevated in all the groups compared with the arsenic group.
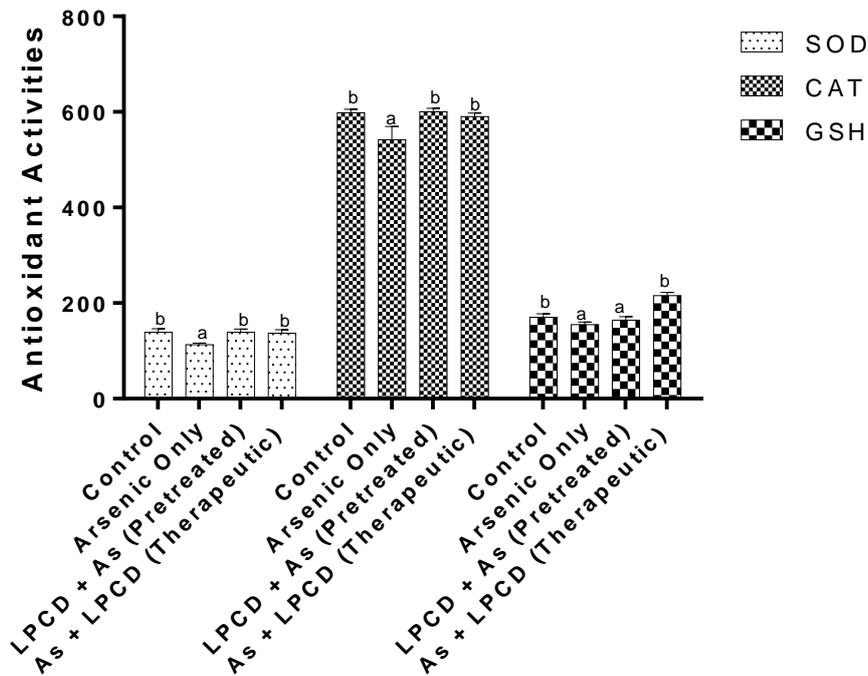

Figure 3. Antioxidant activities in the Liver of Wistar rats exposed to sodium arsenite and leaf pulp of *C. deliciousus.*
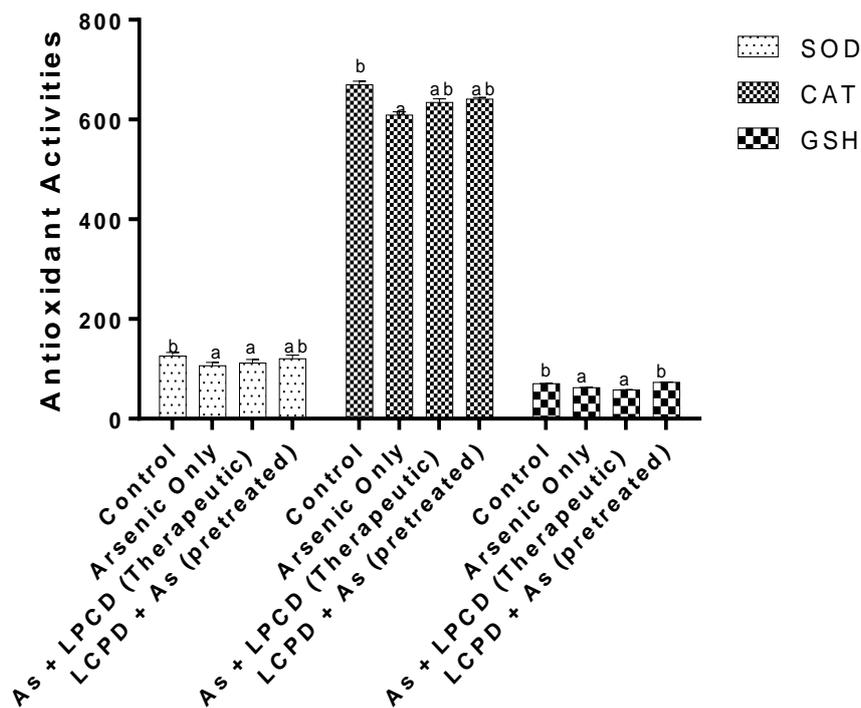

Figure 4. Antioxidant activities in the Kidney of Wistar rats exposed to sodium Arsenite and leaf pulp of *C. deliciousus.*

The malondialdehyde activity was significantly elevated in the kidney in all the treatment groups compared with the arsenic group. At the same time, it was only significantly elevated in the pretreated group in the liver, while the therapeutic group showed a decrease in malondialdehyde activity.
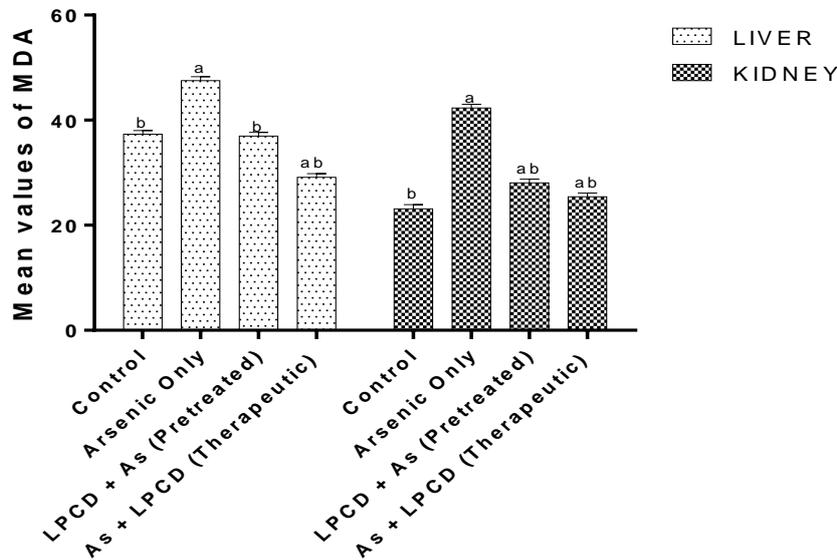

Figure 5. Antioxidant activities in the Liver of Wistar rats exposed to Sodium Arsenite and leaf pulp of *C. deliciousus.*

Treatment of disorders caused by arsenic toxicity or poisoning remains a challenge due to the lack of effective options [31]. It has been discovered that some medicinal plants display effective relief from arsenic-mediated toxicity without any side effects [32]. These plants contain secondary metabolites that help eliminate arsenic from the biological system and, therefore, be more effective than conventional therapeutic agents in ameliorating arsenic-mediated toxicity [33]. This study investigated the effects of the *C. deliciousus* on sodium arsenite-induced hematological and biochemical alterations in Wistar rats. There was a significant decrease in the RBC, Hb, and PCV levels in all the groups at p<0.05 compared to the control. The inhibition of porphyrin or heme synthesis by sodium arsenite could be responsible for the decrease in PCV, Hb and RBC observed in this study. Arsenic inhibits aminolevulinic acid dehydratase (ALAD) activity by binding to its sulfhydryl groups [34], resulting in changes in the pathway leading to the synthesis of heme and RBC, ultimately leading to anemia; this anemia may also be partly due to the effect of free radical generated by the Sodium arsenite on the RBC. The rats in the Arsenic-only group showed significant leukocytosis due to an increase in the white blood cell count. This is a normal occurrence since increased WBC is known to be a reaction to foreign molecules; this leukocytosis was significantly reversed in the animals in the pretreated and the therapeutic group. Leukocytosis, resulting from the mobilization of leukocytes into the circulation, can result from the immune system's promptness against infectious agents or chemicals [35,36]. MCV and MCHC in the arsenic group increased contrary to the decreased erythrocyte count, hemoglobin, and PCV levels. These values can be a marker of anemia with subsequent inhibition of erythropoiesis in the hemopoietic system. Also, the reason for an increase in the MCV and MCHC values may be macrocytic type anemia [37]. However, this elevated level of MCV and MCH was reversed in the Pretreated and the therapeutic group.

Arsenic toxicity leads to a disturbance in lipid metabolism; this is usually associated with increased low-density lipoprotein (LDL), Triglyceride (TG), and total cholesterol, along with decreased high-density lipoprotein (HDL) [38]. Elevated LDL and total cholesterol levels are considered prime risk factors of cardiovascular disorders (CVD). In contrast, elevated high-density lipoprotein shuttles cholesterol from the periphery to the liver, thereby reducing the danger of CVD [39]. This study witnessed a significantly higher concentration of serum lipids scores with a decrease in serum high-density lipoprotein concentration in arsenic-only exposed animals compared to control. Oral administration of *C. deliciousus* as pretreatment and therapeutic agent reversed arsenic-induced dyslipidemia, reduced the degree of atherogenesis, and improved high-density lipoprotein.

Increased urea and creatinine concentrations are used to study the nephrotoxic effect of drugs in animals and men [40]. In this study, arsenate interfered with kidney functions, as seen by elevated urea value in the arsenic and the therapeutic groups. Urea, a waste product of protein catabolism, usually rises when the kidney is faulty [41]. When the rate of serum urea production is greater than the rate of its clearance, serum urea will accumulate, and this will ultimately lead to a faulty kidney [42]. Increased urea and creatinine levels observed in the arsenic-only group may indicate nephrotoxicity by sodium arsenite. This agrees with the findings of [15] that arsenite toxicity causes several metabolic disorders, including urea and creatinine elevation following proximal tubule damage and glomerular injury, respectively. Pretreatment with *C. deliciousus* had reversal effects on these parameters. This indicates that the pretreatment with *C. deliciousus* is more effective than the therapeutic administration in eliciting its nephroprotective effects in arsenite-induced toxicity. Bilirubin is a waste product primarily produced by the normal breakdown of heme [43]. The red cells contain heme, an essential part of the hemoglobin. The liver is the site for bilirubin metabolism and excretion. [44]. The state of a person's liver could be determined by checking the concentration of bilirubin in the blood and diagnosing anemia caused by RBC destruction [43]. The total bilirubin content increased in the arsenic-only group, indicating liver damage caused by sodium arsenate. The ameliorating effect shown by the therapeutic and the pretreated group proves the hepatoprotective effect of *C. deliciousus*.

AST, ALP, and GPT activities are usually used as indicators of liver damage since they are linked to the function of liver cells. An increase in the activities of these enzymes in the group given arsenic only is a clue to a potential hepatic membrane impairment, which will later lead to the release of cytoplasmic content into the circulation [44]. There was an increase in AST, ALP, and GPT activities compared with the control. This might have resulted from sodium arsenite-induced oxidative stress-related damages to the membrane and leakage of the hepatocyte of hepatic transaminases into extracellular spaces, ultimately finding their way into the blood from the liver. The results showed that the pretreated and the therapeutic groups significantly reduced these enzymes' activity compared to the arsenic-only group. This further supports the hepatoprotective activity of *C. deliciousus*.

SOD is an antioxidant enzyme that reduces superoxide radicals to H_2O_2 and water. Metabolism of arsenic leads to increased production of superoxide [5]. GSH is a crucial component of the antioxidant defense mechanism. It functions as a direct, reactive free radical scavenger or as a cofactor of several detoxifying enzymes against oxidative stress, e.g., GPx, GST, and others [5]. GSH is able to regenerate the most important antioxidants, vitamins C and E, back to their active forms [45]. In this study, the depletion in GSH levels was observed after arsenic treatment, which an adaptive response to oxidative stress could explain, hence

GSH plays a fundamental role in detoxifying arsenic species as well as stimulates the excretion of methylated arsenic compounds, or GSH may be oxidized due to the interaction with the free radicals induced by arsenic [45]. The observed rise of MDA concentration in the Arsenic only group indicates lipid peroxidation provoked by arsenic intoxication. Arsenic can cause elevation of MDA levels. Arsenic exposure enhances lipid peroxidation and leads to oxidative stress due to ROS production and lipid peroxidation of membranes, thereby causing degradation of phospholipids and, finally cellular deterioration [46]. It was reported that the increased MDA level was an index of enhanced lipid peroxidation [47]. In addition, the increase of hepatic MDA of As-treated rats is possibly related to the decline in the GPx activity, which scavenges hydroperoxides and lipid peroxides. The therapeutic and the pretreated group were able to reverse this effect, supporting the antioxidant activity and the free radical scavenging effect of *C. deliciousus*.

4. Conclusions

Arsenic toxicity has been reported to cause grave health issues. Arsenic pollution has been documented from different regions globally, and therapeutic interventions for arsenic toxicity are very expensive and are often inaccessible as detoxification should be done promptly. Arsenic contamination can be found in groundwater and food at varying concentrations. The Carpobrotus species have several medicinal values that are reported in the literature. *Carpobrotus edulis* is known for its potent antioxidant and antibacterial properties. *Carpobrotus deliciousus* is believed to have many health benefits and is used indiscriminately by the natives, and from time to time, included in foods; however, there was no study depicting the effect of this plant in the treatment of arsenic toxicity. Evidence regarding the relationship between *Carpobrotus deliciousus* and the toxicity of sodium arsenate was not yet reported in the literature. Information on the effect of the plant on arsenic-induced toxicity on organs such as the liver and kidney was also absent in the literature. This work was probably the first time the effect of the leaf pulp of *C. deliciousus* was being investigated against arsenic toxicity in rats. The findings from this study suggest that oral and repeated exposure to LPCD may possess an ameliorative effect in the management of SA-induced toxicity. Further research should be carried out to investigate the mechanism of action of the leaf pulp of the plant in ameliorating arsenate induced toxicity in organs of rats

Conflicts of Interest

The authors declare no conflict of interest.

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