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The Effects Of Arsenic Accumulation In Clarias Gariepinus On The Activity Of The Enzyme Catalase

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Abstract:

Due to increased pesticide, sewage, and untreated industrial effluent discharges, heavy metals are prevalent environmental pollutants. Arsenic is one of these heavy metals, and it may be found in groundwater, warm springs, rivers, and lakes as well as in saltwater. The goal of the current research was to determine how arsenic buildup affected the catalase activity in the muscular tissues of fingerling African catfish Clarias gariepinus. The goal of the current investigation was to determine if calcium carbonate has any protective effects against arsenic's high toxicity to Clarias gariepinus. Some blood parameters and the hepatosomatic index (HSI) were elevated in fish exposed to arsenic spotlight, but the gonadosomatic index (GSI) and the intestinal index (ISI) were lowered. Fish exposed to arsenic showed plasma concentrations that were considerably higher than those of unsaturated fish, total bilirubin, direct bilirubin, total lipids, glucose, and total protein. Evidence suggests that arsenic poisoning causes an extreme upsurge in ALT, AST, and LDH, EC 1.1.1.27, as well as total protein and glycogen levels in these organs. Histological examination revealed degenerative changes in both the liver and the gills as a result of exposure to arsenic. Most of the arsenic-induced changes may be reversed by liming with calcium carbonate, especially in fish exposed to 1/20 LC50 of arsenic. Therefore, it could be viable to strengthen C. gariepinus with calcium carbonate to shield it against arsenic poisoning.

Keywords: Arsenic; Heavy Metals; *Clarias gariepinus*, sublethal concentrations, toxicity.

I. INTRODUCTION

One of the most crucial elements that significantly contributes to the buildup of heavy metals in animals is their metabolic activity. In recent years, humans have discovered and explored a wide range of alternative uses for heavy metals, both for the benefit of civilization and for personal gain. Heavy metals are major environmental contaminants in today's industrialized world, and their toxicity is a concern that is becoming more and more important for ecological, evolutionary, nutritional, and environmental reasons. This is because heavy metals have exceptional physiological and chemical qualities, which have expanded the spectrum of industrial processes in which they are used, including the manufacturing of fertilizer and biocides.. The biological food system may be further disrupted by the accumulating toxic metal components' negative impacts on aquatic life, which might endanger humans as the final consumer. Fish live in an aquatic environment, which is their natural home. Water pollution immediately affects these fish, putting humans at significant

danger since they consume these fish. an important way to keep track of the pollution levels in water bodies, and it can also be a useful way to learn more about the biological effects of heavy metals that are present in fish and other aquatic organisms at higher levels [1].

Because arsenic is water soluble, its concentrations are greater in aquatic environments than in the majority of terrestrial locations. Arsenic has become more prevalent in the environment recently due to human activities including the use of arsenical pesticides on agricultural land, the treatment of wood with. Arsenite is efficacious and regarded as safe for fish at doses of 1.5–3.8 mg/L. Arsenic concentrations in several fish species that inhabit waters contaminated with arsenic range from 1 to 10 g/g. Arsenic concentrations in fish are said to be more than 100 g/g at the bottom. Aquatic environments contain either arsenite (As3+) or arsenate (As5+) forms of arsenic, which are transformed into one another via redox and methylation processes. Under in vivo and in vitro circumstances, the arsenite one. Additionally, these two forms behave differently inside the cell, with arsenite attaching arsenate interfering with, may have a role in the toxicity of arsenic.

When looking at the consequences of pollution on aquatic ecosystems, fish are often the creature of choice. We created since we know that early detection of arsenic poisoning might be a useful tool in risk assessment. Fish body mass indexes and hematological characteristics have been utilized as markers of pollution levels in the water.

Fish blood and tissue enzyme activity assays (AST, ALT, and LDH) are also often employed in human and animal medicine. To further wreak havoc on the fish immune system, arsenic has been shown to inhibit macrophage function and development while also dampening the fish equivalent of antibodies. Liver fibrosis, hepatocellular damage, inflammation, localized necrosis, and hepatocellular cancer have all been linked to arsenic exposure, according to a number of studies.

II. MATERIAL AND METHODS

Study Area

The study was carried out once every month for a year, from May 2020 to April 2021, along the Gomti River in Sultanpur and Jaunpur, particularly in the urban region. Sultanpur is a city in the Ayodhya division of the Indian state of Uttar Pradesh. Sultanpur is a district located 60 kilometers south of Ayodhya and 135 kilometers east of Lucknow, the state capital. Sultanpur, Uttar Pradesh, India, is located at latitude 26.264776 and longitude 82.072708. Sultanpur is situated in the cities place category in the country of India at the GPS coordinates 26015'53.1936"N and 8204'21.7488"E. The accompanying table includes the coordinates for Sultanpur,. One of Uttar Pradesh's major cities, Jaunpur is situated in the state's southeast. It was founded in the 14th century as a town called "Sheeraz-e-Hind." It is a very sizable medieval city. It was formerly a growing agricultural area with a lot of reputable colleges and schools. Jaunpur is home to more than 4.4 million people now, the majority of whom work in industry and services. In the city, there are a few interesting places to see, such as an ancient historic mosque and a historic bridge. The city of Jaunpur is 228 kilometers southeast of Lucknow, the state capital. Jaunpur is situated to the northwest of the Varanasi district. Jaunpur, formerly known as "Sultan," was built in 1359 was given the name Muhammad bin Tughlaq in honor of his cousin (nick name - Jauna Khan). Jaunpur's population is comparable to that of the surrounding Purvanchal region as a whole. Jaunpur, Uttar Pradesh, India is located at latitude 25.748695 and longitude 82.698441. Jaunpur is situated in the cities location category in the country of India at the GPS coordinates 25044'56.41"N and 82041'55.32"E. The following coordinates, which are for Jaunpur, Uttar Pradesh, India.

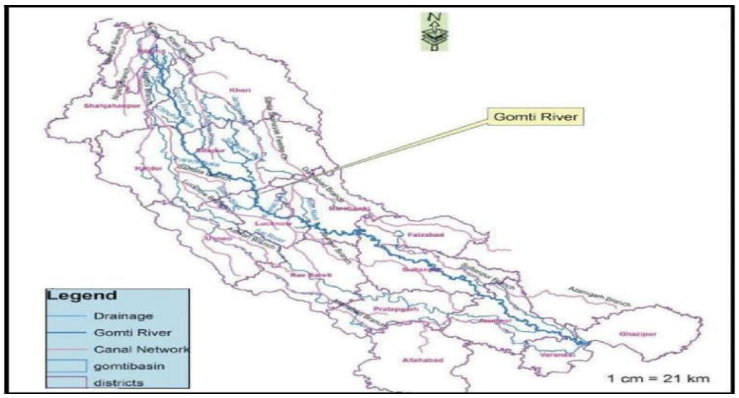

Figure 1: Study are of Jaunpur

2.1. Chemicals and Preparations of Stock Solutions

Egypt was able to provide us with some arsenous chloride. From banaras scientific centre bahelia tola varanasi u. p. was where I purchased my CaCO3, NaOH, and HCl. Additionally, the Cairo, Egypt-based Fura laboratory sold clove oil for use in cosmetics. Using the method recommended by Datta et al., arsenous chloride stock solution (100 mM) was created . The required concentration was maintained using a 10% CaCO3 stock solution.

2.2. Fish

Adult male C. gariepinus were shown at the central aquaculture laboratory in From banaras scientific centre bahelia tola varanasi u. p. They ranged in size from 23.1 to 3.0 cm in total length and weighed between 48.1 and 5.2 g. The fish used in this experiment seemed to be in excellent health. For a week, fish were acclimated in a lab environment. Along with a commercial fish meal, fish were fed minced chicken liver ad libitum (3% of their total body weight). The aquariums' water was changed every 24 hours to remove both soluble excretory products and faecal components. Fish were handled with care and according to standard laboratory practices.

2.3. Determination of LC50 for Arsenic

The limit 96-h LC50 of arsenic was established by administering the fish five escalating doses of arsenic (under static conditions). Cumulative death was defined after 96 hours, and the dead fish were then taken out of the water.

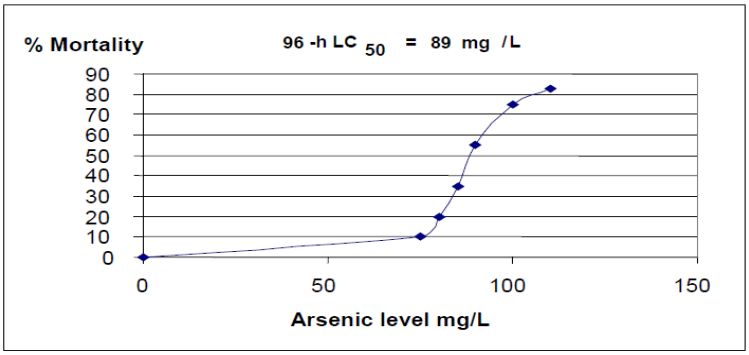

Figure 2: Graphic evaluation of 96- h LC50 of arsenous chloride\

2.4. Experimental Groups

The testing was done in glass aquariums with dimensions of 40 cm in height at a static system. Each of the 9 test group consisted of 5 acclimatized fish. These are the many types of experimental groups:

Group 1: Arsenic-free and calcium carbonate-free fish (control).

Group 2: Arsenic exposure at 1/10th the LC50 level with no calcium carbonate added results in death in fish.
Group 3: Fish given a concentration of arsenic equivalent to 1/20 of the maximum tolerated level (LC50) but no calcium carbonate at all.
Group 4: Arsenic at 50 mg/L and no calcium carbonate in the water is toxic to fish.
Group 5: Fish exposed to 1/10th the LC50 concentration of arsenic and 50 milligrams per liter of calcium carbonate.
Group 6: Fish given a calcium carbonate solution with 50 milligrams per liter and a twentieth of the LC50 of arsenic.
Group 7: Effects of calcium carbonate concentrations of 100 mg/L and 0 mg/L on fish.
Group 8: Calcium carbonate (100 mg/L) and arsenic (1/10 LC50) were administered to fish.
Group 9: Toxic effects of calcium carbonate and arsenic (1/20 LC50) in fish at 100 mg/L.

2.5. Somatic Indices

The fish were divided into groups and their organs, including the liver, gonads, and intestines, as well as their guts, were weighed. The hepatosomatic index was determined as a proportion of the total body weight rather than just the weight after being gutted.

2.6. Haematological Parameters

Following the protocol described by Ribas et al. (2007), 1 ml/L of clove oil was used to anesthetize the fish before collection 20 days later. Blood was drawn veins and arteries using a hypodermic syringe. The blood was then transferred to a tube treated with lithium heparin to avoid clotting. After diluting the blood with saline solution (0.75 percent NaCl), the RBC count was taken using a Neubauer haemocytometer slide. Blood was drawn into microhaematocrit capillary tubes and spun for 5 minutes to get Hct readings. Hct were determined by dividing PCV by total blood volume.

Henry's technique was used to calculate the hemoglobin concentration (Hb, g/100 ml) in the blood (1964). After centrifuging the remaining blood.

2.7. Biochemical Analysis

From each fish, the liver in addition to the white muscle at the epiaxial axis were taken. A glass homogenizer was used to blend the tissues while they were submerged in cold distilled water. The tissue homogenates were centrifuged twice at a speed of 4000 revolutions per minute for a total of five minutes. The enzyme activity and metabolite contents of the tissue supernatants were determined after separating them. Following Reitman and Frankel's approach from 1957, the AST and ALT activity were measured colorimetrically in plasma and tissue. Only in tissue was the activity of LDH kinetically measured at 340 nm. Blood plasma was examined to assess its total and direct bilirubin, Alkaline tissue digestion was used to assess the amount of glycogen in the muscle and liver, and then glucose was measured enzymatically.

2.8. Histology

The fish's liver and gills were removed before it was dissected. These organs were cut up into tiny pieces, preserved in neutral buffered formalin, dried down, and then embedded in paraffin. Using a 6 m tissue segment, hematoxylin and eosin staining was applied. Using a trinocular light microscope (Bio-Med), pictures of the dyed tissue slices were taken and then connected using software. Windows version of Image-Pro Plus. The severity of tissue lesions was assessed semi-quantitatively using the degree of tissue change (DTC). The following progressive ordering were used to classify the modifications in the examined organs: Stage I changes nothing about how the tissue functions normally, stage II is more severe and interferes with how the tissue functions normally, and stage III is very severe and results in irreversible tissue damage. The DTC value was determined by using the

method and tallying the total number of tissue lesions that were present in each animal at stages I, II, and III:

DTC = (1×Σ I) + (10 Σ II) + (100×Σ III).

No harm to the organ was indicated by a DTC value of 0-10, mild damage by 11-20, moderate modifications by 21-50, severe damage by 50-100, and permanent damage by 100 and above.

III. STATISTICAL ANALYSIS

The statistical analysis in this research was performed in spss (Version 10). Means and standard deviations were used to present the data in this article. In order to find out whether there was a notable change between the control and experimental groups, a series of paired t-test comparisons were performed.

IV. RESULTS

4.1. Somatic indices

In fishes exposed to both arsenic concentrations (1/10 & 1/20 LC50), only the HSI values rose considerably, while the GSI and ISI values decreased. These modifications were shown to be dose-related. There were no discernible changes in the assessed somatic indices after exposing fishes to calcium carbonate (50 or 100 mg/L). Both the calcium carbonate and arsenic levels assessed did not substantially affect the somatic indices of fish when compared to the control group. (Table 1).

4.2. Blood Parameters

Significant decreases in RBCs, Hb contents, and Hct values were seen in fish exposed to both concentrations of arsenic evaluated when compared to control group fish (Table 2). Variables in blood pressure, heart rate, and oxygen saturation were measured in fish treated to calcium carbonate alone or in combination with other treatments.

Table 1. Exposed C. gariepinus for 20 days to two concentrations of arsenic (As) or calcium carbonate (

Groups	HSI	GSI	ISI
Control	0.551±0.023	0.312±0.049	7.291±0.423
1/10 LC_{50} of As	0.836±0.031*	0.174±0.017*	6.124±0.621*
1/20 LC_{50} of As	0.646±0.056*	0.245±0.071*	6.561±0.742*
50 mg/L $CaCO_3$	0.591±0.072	0.291±0.042	7.123±0.562
50 mg/L $CaCO_3$ + 1/10 LC_{50} of As	0.660±0.042	0.284±0.042	6.924±0.452
50 mg/L $CaCO_3$ + 1/20 LC_{50} of As	0.585±0.067	0.324±0.092	7.492±0.821
100 mg/L $CaCO_3$	0.574±0.027	0.320±0.121	7.801±0.721
100 mg/L $CaCO_3$ + 1/10 LC_{50} of As	0.571±0.022	0.304±0.049	7.321±0.561
100 mg/L $CaCO_3$ + 1/20 LC_{50} of As	0.585±0.054	0.974±0.072	7.421±0.762

Table 2. Variations in a number of blood parameters of the Nile catfish were observed (C. gariepinus)

Groups	RBCs ($X10^6/mm^3$)	Hb (g/L)	Hct (%)
Control	2.124±0.121	90.421±5.120	25.513±1.123
1/10 LC_{50} of As	1.421±0.215*	71.412±8.125*	18.149±1.052*
1/20 LC_{50} of As	1.6214±0.425*	80.120±6.240*	20.389±2.256*
50 mg/L $CaCO_3$	2.121±0.231	91.023±6.121	25.670±2.490
50 mg/L $CaCO_3$ + 1/10 LC_{50} of As	1.954±.316	85.431±6.132	23.124±1.624
50 mg/L $CaCO_3$ + 1/20 LC_{50} of As	2.121±0.215	89.124±7.181	24.769±1.351
100 mg/L $CaCO_3$	2.212±0.265	93.561±8.921	25.812±2.012
100 mg/L $CaCO_3$ + 1/10 LC_{50} of As	2.104±0.371	91.246±7.213	24.921±1.941
100 mg/L $CaCO_3$ + 1/20 LC_{50} of As	2.156±0.213	95.121±8.923	25.637±2.141

4.3. Plasma Biochemical Parameters

Enzyme activities (AST, ALT, and GC) and other plasma biochemical parameters were measured in arsenic-exposed fish. The levels of total bilirubin, total lipids, glucose, and total protein were all considerably higher in the experimental group compared to the levels in the control group (Table 3).

This result was linked to the total concentration of arsenic in the environment. There were no discernible differences found between the fish that were given the control substance and those that were given the tested levels of calcium carbonate alone or 50 mg/L calcium carbonate combined with 1/20 LC50 arsenic. All of the biochemical parameters were significantly elevated in the exposed group of fish compared to the control group after they were given either 50 or 100 mg/L of calcium carbonate and 1/10 of the LC50 concentration of arsenic. However, after being exposed to 100 mg/L of calcium carbonate and 1/20 of the LC50 concentration of arsenic, the observed biochemical parameters did not alter dramatically; the only exception was the plasma glucose level, which rose significantly.

4.4. Carbohydrate Metabolism

The level of LDH activity in the livers of fish that had been exposed to arsenic of fish in the control group (Table 4). In a manner comparable to this, exposure to arsenic increased the activity of LDH in the muscle. The increase in its activity was not statistically different in fish that were treated to 1/10 the LC50 level of arsenic, in contrast to. The activities of LDH that were exposed to both amounts of calcium carbonate did not differ considerably from one another. In the tissues that are the focus of this inquiry, the LDH activity is unaffected by the administration of calcium carbonate at either of the two levels of exposure that were examined in conjunction with 1/20 of the LC50 concentration of arsenic. Surprisingly, fish who were given calcium carbonate (at both tested levels) together with arsenic at 1/10 the LC50 level showed a much higher level of LDH activity in their livers and muscles compared to the group that served as the control. The arsenic levels that were tested caused a significant decrease in the glycogen levels seen in both the liver and the muscles. Calcium carbonate at a concentration of 50 mg/L was used, and arsenic levels were reduced at the same time. However, there was no discernible change to the glycogen levels in the liver or the muscles as a consequence of this treatment. However, when paired with a greater quantity of arsenic, it caused the glycogen levels in the specified tissues to drop significantly.

Table 3. Changes in several biochemical parameters after 20 days of exposure or their mixtures in the blood plasma of C. gariepinus

Groups	AST (U/L)	ALT (U/L)	Total bilirubin (mg/L)	Direct bilirubin (mg/L)	Total lipids (g/L)	Glucose (g/L)	Total proteins (g/L)
Control	81.24±2.12	54.34±1.26	1.86±0.13	1.02±0.25	17.34±0.92	0.88±0.03	18.45±1.40
1/10 LC_{50} of As	120.12±4.65*	85.63±6.24*	2.91±0.12*	1.85±0.56*	23.46±1.26*	0.12±0.05*	22.47*±1.07
1/20 LC_{50} of As	97.435±7.62*	65.46±3.92*	2.30±0.07*	1.54±0.42*	21.56±0.83*	1.03±0.05	20.45*±1.12
50 mg/L $CaCO_3$	84.62±8.24	56.23±5.16	1.72±0.21	1.12±0.42	17.63±0.69	0.90±0.07	18.72±0.95
50 mg/L $CaCO_3$ + 1/10 LC_{50} of As	99.24±6.24*	71.21±4.59*	2.36±0.20*	1.56±0.52*	20.19±0.89*	0.98*±0.07	19.02±2.10
50 mg/L $CaCO_3$ + 1/20 LC_{50} of As	84.27±3.92	56.42±4.58	1.91±0.14	1.37±0.67	18.04±0.67	0.92±0.09	18.98±0.95
100 mg/L $CaCO_3$	83.46±5.92	55.92±6.12	1.90±0.26	1.21±0.37	17.94±1.29	0.91±0.07	18.94±1.04
100 mg/L $CaCO_3$ + 1/10 LC_{50} of As	103.69±6.43*	75.12±9.22*	2.42±0.62*	1.62±0.32*	21.45±1.33*	1.03±0.07*	21.86±1.47*
100 mg/L $CaCO_3$ + 1/20 LC_{50} of As	83.627±5.024	56.17±7.92	2.05±0.46	1.13±0.42	18.02±2.76	0.95±0.07*	19.15±1.12

Table 4. LDH activity (units/min/g fresh tissue) and glycogen content (mg glucosy glucose/g fresh tissue)
Nile catfish (C. gaeiepinus) liver and muscle subjected to arsenic, calcium carbonate, or both. combinations

Group	LDH		Glycogen	
	Liver	Muscle	Liver	Muscle
Control	2.452±0.236	105.243±4.563	1.361±0.035	0.516±0.029
1/10 LC_{50} of As	3.185±0.314*	145.412±5.129*	0.953±0.029*	0.468±0.046*
1/20 LC_{50} of As	2.946±0.261*	109.374±6.274	1.167±0.041*	0.495±0.032
50 mg/L $CaCO_3$	2.514±0.394	107.139±7.149	1.349±0.046	0.498±0.089
50 mg/L $CaCO_3$ + 1/10 LC_{50} of As	2.853±0.426*	131.721±6.293*	1.224*±0.051*	0.408±0.056*
50 mg/L $CaCO_3$ + 1/20 LC_{50} of As	2.568±0.367	110.426±7.014	1.314±0.063	0.499±0.067
100 mg/L $CaCO_3$	2.638±0.528	106.943±6.981	1.328±0.072	0.521±0.078
100 mg/L $CaCO_3$ + 1/10 LC_{50} of As	2.863±0423*	126.166±7.142*	1.301*±0.057*	0.453±0.049*
100 mg/L $CaCO_3$ + 1/20 LC_{50} of As	2.416±0612	109.624±7.753	1.402±0.082	0.497±0.066

4.5. Protein Metabolism

Fish exposed to both measured arsenic levels showed a considerable advantage in their AST and ALT activity (Table 5). Fish exposed to either dose of calcium carbonate had enzyme activities that were not noticeably different from those of control fish measured at the same time. The levels of AST and ALT activity in the liver and muscles of fish that were given either 50 or 100 mg/L of calcium carbonate and 1/20 of the LC50 dose of arsenic did not change. When compared to controls, fish that were given calcium carbonate and 1/10 of the LC50 amount of arsenic exhibited much better behavior, as evaluated by AST and ALT levels in the liver and muscle, respectively. The level of liver ALT activity in fish that had been exposed to 100 mg/L calcium carbonate and 1/10 LC50 of arsenic was not substantially different from that of the fish in the control group.

Exposure to arsenic results in a significant decrease in the total protein content of the species studied. while there is no detectable effect on fish tissues from exposure to any of the dosages of calcium carbonate that have been tested. Both low and high doses of arsenic and calcium carbonate have little impact. Changes in the liver's total protein composition are reversible. Regular levels of activity were maintained in only those fish fed the highest concentrations of calcium carbonate and the least arsenic.

4.6. Histological Alterations

After being subjected to any quantity of arsenic, liver cells finally perished after undergoing deformation, vacuolization, and necrotization. This was due to the fact that certain cytoplasmic and nuclear material had partially precipitated (Table 6 and Figure 2). It was also shown that exposure to arsenic was the primary factor in the development of branchial alterations in the gills (Table 6 and Figure 3). Comparing the arsenic-exposed fish to the control group, the mean DTC rates for the liver in the arsenic-exposed fish remained 54.79, which indicated severe damage, and 31.46, which indicated moderate damage (Table 7). The mean DTC findings indicated that fish exposed to the measured amounts of arsenic had moderate gill damage. (40.26 and 24.72).

Table 5. Whether there were shifts in AST, alanine ALT, or a combination of the two throughout the course of 20 days.

Groups	AST		ALT		Total proteins	
	Liver	Muscle	Liver	Muscle	Liver	Muscle
Control	48.432±5.121	25.625±1.432	40.592±6.927	20.463±0.982	90.465±8.241	21.928±0.592
1/10 LC_{50} of As	65.513±4.152*	33.156±1.914*	62.692±4.212*	29.614±1.842*	60.246±9.356*	15.331±1.014*
1/20 LC_{50} of As	58.724±5.136*	29.723±1.634*	51.937±5.214*	25.147±2.361*	70.861±6.725*	17.853±1.126*
50 mg/L $CaCO_3$	50.312±6.724	26.481±1.378	42.371±4.125	21.793±2.146	92.645±9.325	20.784±1.214
50 mg/L $CaCO_3$ + 1/10 LC_{50} of As	60.261±5.243*	30.482±2.163*	55.792±4.132*	26.615±1.792*	70.149±7.632*	17.124±1.146*
50 mg/L $CaCO_3$ + 1/20 LC_{50} of As	51.429±6.241	26.372±2.017	45.629±6.731	22.069±1.998	84.937±7.642*	20.982±1.219
100 mg/L $CaCO_3$	52.371±5.432	25.894±2.146	44.671±5.291	21.137±1.984	92.561±9.146	20.947±1.461
100 mg/L $CaCO_3$ + 1/10 LC_{50} of As	61.425±4.938*	29.824±2.263*	45.463±5.241	25.431±2.413*	80.426±6.293*	16.894±2.143*
100 mg/L $CaCO_3$ + 1/20 LC_{50} of As	52.604±7.125	27.093±2.087	43.921±4.231	22.101±2.141	85.739±7.426	20.024±1.635

Table 6. Histological changes in the liver and gills of C. gariepinus treated to arsenic or calcium carbonate for 20 days.

Liver	Gills	Liver	Gills
Stage I		Stage II	
-Nuclear hypertrophy	-Hyperplasia of gill	-Cytoplasmic degeneration	-Rupture of epithelial cells
-Cellular hypertrophy	epithelium	-Cell rupture	with haemorrhage
-Cytoplasmic vacuolation	-Epithelial lifting of gill	-Nuclear degeneration	-Comlete fusion of lamellae
-Nuclear atrophy	lamellae		-Rupture of pillar cells
-peripheral nuclei	-Lamellar fusion		

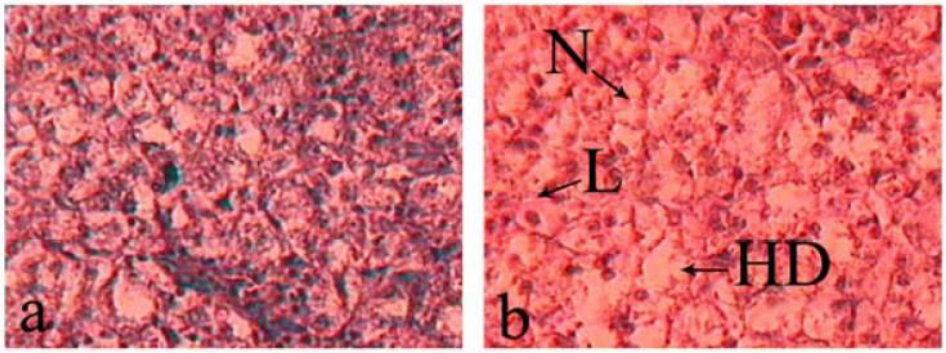

Figure 2. 400X magnification of a 6-m-thick C. gariepinus liver section.

Necrosis, enlarged hepatocytes, and hepatocyte degeneration were seen in fish exposed to 1/10 LC50 As. (A) Fish exposed to water (control); b) As-treated fish.

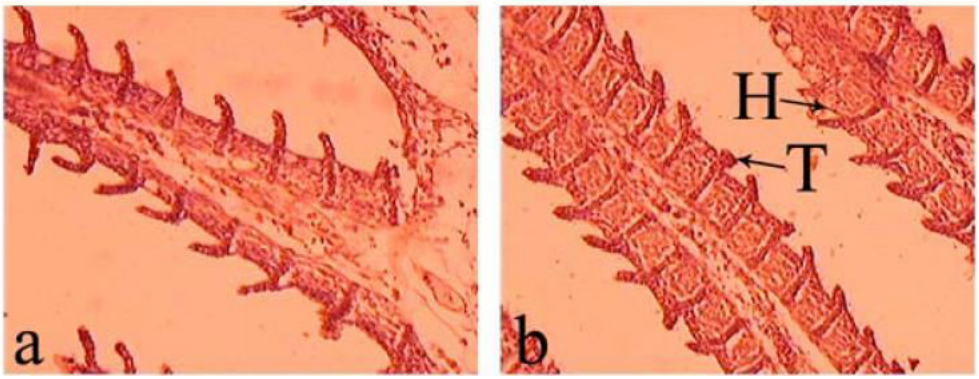

Figure 3. a photomicrograph of a C. gariepinus liver segment (6 m) stained with hematoxylin and eosin (100X) shows normal structure in fish exposed to water (control) and 1/10 LC50 of As (40X).

Table 7. The outcomes are the average of two exposures to arsenic, calcium carbonate, or both in the liver and gills of a single fish.

Group	Liver	Gills
Control	14.62±1.12	9.71±1.52
1/10 LC_{50} of As	54.792±5.34*	40.26±5.19*
1/20 LC_{50} of As	21.46±3.69*	24.72±4.27*
50 mg/L $CaCO_3$	14.49±3.69	12.48±4.85
50 mg/L $CaCO_3$ + 1/10 LC_{50} of As	24.13±5.17*	25.14±6.13*
50 mg/L $CaCO_3$ + 1/20 LC_{50} of As	16.73±5.32	14.98±4.68
100 mg/L $CaCO_3$	12.12±4.29	19.86±5.97*
100 mg/L $CaCO_3$ + 1/10 LC_{50} of As	20.76±5.92*	33.14±3.96*
100 mg/L $CaCO_3$ + 1/20 LC_{50} of As	15.01±3.14	17.56±6.12*

When comparing the DTC values of the livers of fish treated with either of the two calcium carbonate concentrations with those of untreated fish, The results showed no statistically significant variations. Fish given 50 milligrams per liter of calcium carbonate had DTC levels that were within normal limits.\, but DTC levels in fish treated with 100 mg/L of calcium carbonate were 14.86. In both the liver and the gills, the DTC values were not substantially different following exposure to 50 mg/L calcium carbonate and 1/20 LC50 of arsenic as compared to the matching control. The similar pattern is seen in the liver of fish given 1/20 LC50 arsenic and 100 mg/L calcium carbonate.

V. DISCUSSION

The current investigation showed that arsenic decreased GSI levels in C. gariepinus. Male catfish (Pangasianodon hypophthalamus) exposed to metal tainted water shown this behavior in the past. They found that the exposed fish's spermatogonia and sertoli cells had necrotized and became vacuolated. In this experiment, ISI values dropped after being exposed to arsenic. This finding

coincides with those previously reported for similar toxins. It's possible that a lack of hunger is to blame for this, which would explain why the person's overall body weight has dropped. Several studies have looked at ISI fluctuations as a proxy for physiological status and body weight loss due to decreased feed intake. In the current work, exposing C. gariepinus to arsenic and calcium results in reversible somatic indices. Observing how haematological parameters vary over time may be a good indicator for fish health. This research documents a decrease in blood parameters caused by arsenic exposure. Pollutants may be causing hemolysis and/or hemorrhaging in the fish, which would explain the phenomenon. When it comes to metabolic processes, the fish liver is one of the more delicate organs involved. So, the liver is generally the organ to first show signs of a chemical's effects.

Arsenic exposure has been linked to a decrease in liver function, which has led researchers to utilize this metric as a proxy for the extent of the damage., bilirubin, total lipids, and total protein were measured to assess arsenic-induced hepatotoxicity. The research showed that after being exposed to arsenic, these indicators rose to unhealthy heights. This may be an indication of arsenic toxicity-related liver damage. There are two possible explanations for the increase in bilirubin concentrations observed: haemolysis and fluctuations in bilirubin absorption and conjugation by hepatocytes. Also, the observed dramatic increase in plasma total lipids is consistent with data on similar trends for other environmental contaminants. As a result, it's possible that this might have a similar impact to that of hazardous agents and environmental contaminants. In this research, arsenic exposure resulted in hyperglycemia, which might be a sign of hepatopancreatic degeneration.

Following treatment with calcium carbonate, it was shown that the majority of the plasma biochemical parameters evaluated were reversible in fishes that had been exposed to 1/20 LC50 of arsenic. This was demonstrated in the study that is being presented here. On the other hand, same compounds showed irreversibility when calcium carbonate was used in conjunction with 1/20 of the LC50 of arsenic. Since the toxicity of arsenic varies depending on both the amount and the species, this might very well be the case. Because of this, there was a reduction reported for the total quantity of protein in the report. Given the enormous energy demand that pollutants cause, it's probable that the tissue proteolysis previously observed for several fish species that were exposed to pollution would provide light on how protein is used as an alternate source of energy. This is because proteins may be disassembled into their individual amino acids, which can then be utilized as fuel. This would be the case because of the rising energy consumption brought on by environmental toxins.

VI. CONCLUSION

As a consequence of being exposed to arsenic, the liver and gills of C. gariepinus exhibited considerable histopathological changes in the current experiment. It is possible that cellular damage and hepatic disorders are present when there is an abundance of increased DTC in the liver. This conclusion is given further weight by the data that were provided in this article, which indicated greater AST and ALT levels in plasma as a result of arsenic exposure. The changes in the liver's histology that arsenic causes are a reflection of the organ's high vulnerability to contaminants. These changes, which might be useful for environmental monitoring, are induced by arsenic. Additionally, the gills are an essential component in the process of fish breathing and the elimination of waste. Gills of C. gariepinus that had been exposed to arsenic over the course of the present study exhibited evidence of the development of histological alterations. This is accomplished to some degree by the fact that the DTC values of exposed fish are much greater than those of control fish. It has been shown that exposure to arsenic causes hyperplasia of the gills, telangiectasia, and lamellar fusion; these results are comparable with those for other environmental contaminants.

Arsenic causes branchial lesions in the gills of C. gariepinus, which leads to a disruption in the normal functioning of the gills. Gill hyperplasia and metal pollution-induced hypoxia were conditions that were connected to one another. These changes, which were brought on by exposure to metal, might be a defensive reaction designed to prevent metal from entering the gill cells. It is possible to demonstrate that C. gariepinus's vulnerability to arsenic led to the development of undesirable characteristics. Instead, the deleterious impact was mitigated when calcium carbonate and arsenic at a concentration of 1/20 LC50 were mixed together. As a consequence of this, it has the potential to be useful as a protection against the proper harm caused by arsenic.

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