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Original Research Article

Protective role of hydroxy citric acid (HCA) against lead induced toxicity in albino wistar rats

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ABSTRACT

The present study was designed to evaluate the role of herbal active constituent hydroxycitric acid obtained from Garcinia cambogia and Hibiscus subdariffa in heavy metal especially lead poisoning. The Lead acetate at 500ppm was used as an inducing agent in the present study. Sodium acetate (500ppm) used as control group is used as a baseline measure. The HCA at 100 mg/kg and 200mg/kg were used in the treatment of lead induced toxicity in rats. At the end of 28 days study period the blood levels of alpha ALAD activity was estimated in all the treatment groups. Lead inhibits the ALAD is more profound and its inhibition has been used clinically to gauge the degree of lead poisoning. Inhibition of ALAD results in the accumulation of aminolaevulinic acid, detectable in the plasma and urine even at blood lead levels of less than 10 μg/dl. It results that HCA showed significant elevated levels of ALAD activity, which was reduced by the lead intoxication. HCA showed significant reduction of liver enzymes (SGOT, SGPT and ALP). The markers like protein, bilirubin and creatinine were also found to be elevated in lead intoxicated rats and was found to be decreased significantly in a concentration dependent manner.

1. Introduction

Heavy metal toxicity has been proven to be a major threat and there is several health risks associated with it. The toxic effects of these metals, even though they do not have any biological role, remain present in some or the more other form harmful for the human body and its proper was functioning. There are 35 metals that are of concern. These heavy metals are commonly found in the environment and diet. In small amounts they are required for maintaining good health but in larger amounts they can become toxic or dangerous. Heavy metal toxicity can lower energy levels and damage the functioning of the brain, lungs, kidney, liver, blood composition and other important organs. Long-term exposure can lead to gradually progressing physical, muscular, and neurological degenerative processes that imitate diseases such as multiple sclerosis, Parkinson's disease, Alzheimer's disease and muscular dystrophy. Repeated long-term exposure of some metals and their compounds may even cause cancer. Thus utilization of heavy metal contaminated water results in high morbidity and mortality rates all over the world. Continuous exposure to heavy metals may lead to internal imbalance in body, they start accumulating in body where the body start using them as substitute of essential elements.[1–14]

Lead: Lead has major effects on different parts of the body. Lead distribution in the body initially depends on the blood flow into various tissues and almost 95% of lead is deposited in the form of insoluble phosphate in skeletal bones (Papanikolaou et al., 2005). Toxicity of lead, also called lead poisoning, can be either acute or chronic.

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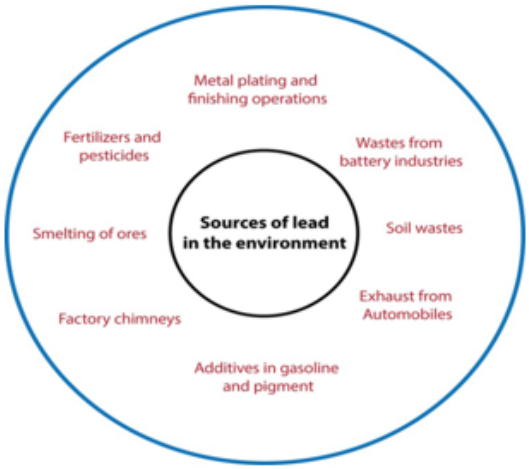


Table 1: Sources of lead toxicity

Acute exposure	Chronic exposure
Loss of appetite,	Mental retardation,
Headache,	Birth defects,
Hypertension,	Psychosis,
Abdominal pain	Autism,

1.1. Treatment for heavy metal poisoning

Medical treatment is one element of a comprehensive treatment for exposure to lead; removal source of lead exposure impairment. Chelation therapy is employed for the most severe case of lead of poisoning.

1.2. Chelating therapy

Chelation therapy is a medical procedure that involved the administration of chelating agent to remove heavy metal or lead from the body. Chelating agents are administered under careful medical supervision duet to various inherent risks.[15–22]

1.3. Side effect of the treatment

Chelation therapy required the use of power full chelators that can be producing a variety of mild to severe side effects.

1. One of the common side effects of chelation therapy is burning sensation near the injections
2. Fever, Headache, Nausea and vomiting.
3. Severe hypersensitivity relation may to anaphylactic shock and even death.
4. Liver damage may be seen with some chelating agent and some patients may develop liver failure

1.4. Herbal drug s used for heavy metal poisoning

1. Herbal medicine, also known as herbals or botanical medicine is a medical system based on the use of plants or plant extracts. Since ancient times, herbal medicine has been used by many different cultures throughout the world to treat illness and to assist bodily functions.
2. India has a unique position in the world where a number of recognized Traditional system of medicine e g; Ayurveda, Siddha, Unani, Homeopath India has a very long, safe and continuous usage of many herbal drugs in the officially recognized alternative systems of health. Ayurveda, Yoga, Unani, Siddha, Homeopathy and Naturopathy.
3. The toxicity of trace metals on human health environment has attracted Heavy metals have a tendency to accumulate in the food chain. Heavy metals have low excretion rates through the kidney which could result in damaging effects on humans even at very low concentrations. Metals such as zinc, copper, iron, manganese, and chromium are essential nutrients; they are important for the physiological and biological functions of the human body. However, an increase in their intake above certain permissible limits can become toxic. In general, a number of health problems were linked to excessive uptake of dietary heavy metals including a decrease in immunological defences, cardiac dysfunction, fetal malformation, impaired psychosocial and neurological.

2. Aim and Objectives

2.1. Aim

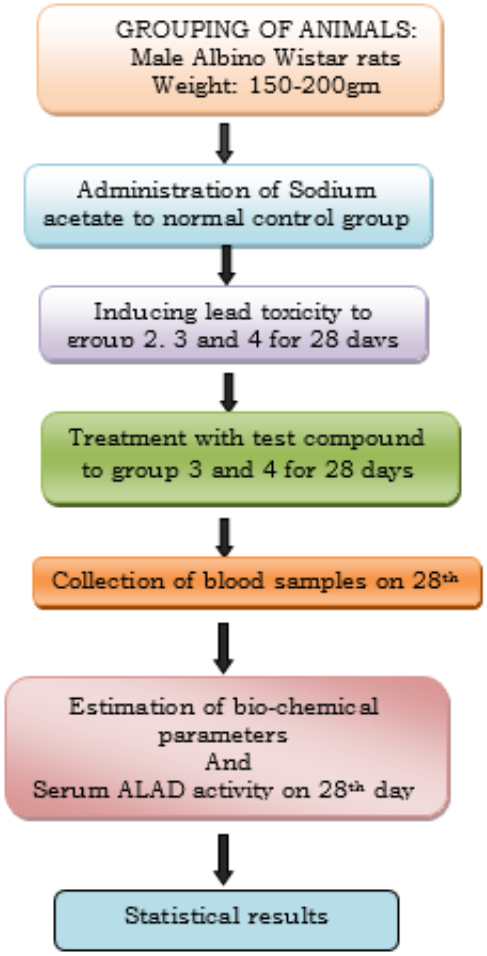
To evaluate the effects of hydroxyl citric acid (HCA) in lead induced toxicity in male albino Wistar rats.

2.2. Objectives

To study the lead induced toxicity on different organs in male albino Wistar rat.

1. To study the influence of hydroxyl citric acid (HCA) on the serum biochemical parameters in lead induced toxicity in male albino Wistar rat.
2. To study the influence of hydroxyl citric acid (HCA) on serum ALAD levels in lead toxicity induced male albino Wistar rats.

3. Study Protocol



3.1. General antidotes or chelating agent of Ayurveda

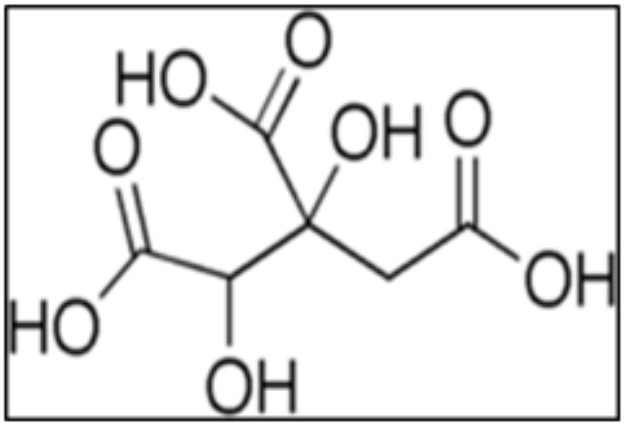
1. The Ayurvedic system of medicine has a great antiquity, dating back to about 5000 years B.C.

2. The advent of Rasa Shastra, use of certain metals and minerals in Ayurvedic therapeutics increased.

3. Tankan (borax), Gandhak (sulphur), Saindhav Lavana (rocksalt), Churnodaka (lime water), Triphala Kwath, ginger juice, Arjuna (Terminalia arjuna), turmeric, lemon juice, Maricha (black pepper), Kumari Swarasa (aloe vera juice), coriander, honey, cow's milk, goat's milk, cow's ghee, cow's urine, common antidotes which are used to subside the toxic symptoms and to remove the toxic effects of administration of improper processed metals and minerals.

4. Ayurveda have the potential to detoxify the body from metal toxicity. Now chelating agenta now days of Ayurveda to prove their potency for detoxifying the body from metal toxicity,and they will detoxify the body without causing agent any harm to other essential mineral of the body.

(-)-Hydroxycitric acid (HCA): It is a main component of the dried fruit rind of garciniacambogia, a native Southeast Asian plant commonly known as brindle berry or Malabar tamarind.



Molecular formula: $C_6H_8O_8$

Sources of Hydroxy citric acid: Garcinia cambogia, Hibiscus subdariffa.

4. Plant Description

Garciniacambogia; (Family: Guttiferae); is a small or medium sized tree with a rounded crown and horizontal or drooping branches. The fruit have six to eight seeds surrounded by a succulent aril and the tree is distributed commonly in the ever green forests of western Ghats, from konkan southward to Travancore and in the forests of Nilgiris up to an altitude of 6000 ft. It flowers during the hot season and fruits ripen during the rainy season. The fruit is harvested, dried and ground into a powder. Garciniacambogia extract is the calcium salt of hydroxyl citric acid (Both 50% and 60% hydroxy citric acids are available), which is obtained from water extract of Garciniacambogia fruit.

4.1. Mechanism of action

HCA is a competitive inhibitor of the enzyme ATP citrate-lyase, an extra-mitochondrial enzyme which is involved in the initial steps of de novo lipogenesis in the body. Consequently, HCA reduces the transformation of citrate into acetyl coenzyme A, a step necessary for the formation of fatty acids in the liver. In addition, there is an increased

production of hepatic glycogen in the presence of HCA, which may activate glucoreceptors leading to a sensation of fullness and reduced appetite. The increased bioavailability of serotonin is thought to be related to appetite supressing effects of supplemental HCA. Another possible mechanism of action may be HCA's ability to down-regulate leptin, an amino acid hormone that influences obesity and body weight.

Chemical constituents: Garcinol (anti-inflammatory and potent anti-cancer), Succinic acid (anti-bacterial), tartaric acid (anti-oxidant)

Resin: It contains cambogimal and cambogin. From this, the gutta gum is made, with purging

Uses: Food, sour, astringent, thermo genic, constipating and digestive.

Rinds: treatment of inflammatory ailments, for rheumatic pains and bowel complaints, piles, hemorrhoids, colic problems, ulcers, inflammations, treat sores, dermatitis, diarrhoea, dysentery, ear infection, to facilitate digestion and to prevent over perspiration or hyper perspiration

Fruit: Anti-helmintic and cardiotonic

Other therapeutical uses: Antifungal Effects, Free Radical Scavenging Properties, Anti-Lipid Peroxidation and Anti-Carbonyl Activities, Anti -aging Activities, Gastro protective Effects, Anti-diabetic Effects, Antineoplastic and Chemo preventive Effects, Lowering Lipid Effect, Anti-Obesity Activity.

5. Therapeutic Dosages

The usual dosage for Garcinia is 300 to 500mg tablets three times daily taken half an hour before meals with water.

6. Collection of Blood Samples From Rats

6.1. Materials

1. Micro-centrifuge tubes (1.5 ml capacity)
2. Micro-capillary tubes (1 mm diameter).
3. Absorbent cotton

Blood was collected from the retro orbital plexus of rats. It is the best method, if small amounts (0.1 to 0.5 ml) of blood samples are required. A fine capillary is inserted at 45 degree angle and over the bony socket to rupture the fragile venous capillaries of the ophthalmic venous plexus. The passage is about 10 mm. The tip of the capillary is slightly retracted and the blood collected in the orbital cavity flows out from the capillary which is collected in a micro-centrifuge tube. After collecting the desired volume, capillary is removed with simultaneous release of pressure by fore finger and thumb. Any residual blood droplet around the eye ball is wiped off by absorbent cotton swab. In this study unanaesthetized animals were used because, anesthesia causes hyperglycemia by various mechanisms. Ether increases blood glucose levels by glycogenolysis in liver (Dohm et al., 1983). Halothane increases blood glucose by inhibiting release of insulin from pancreas, inhibit the effect of insulin on tissues and decreased rate of glycogen synthesis in liver.

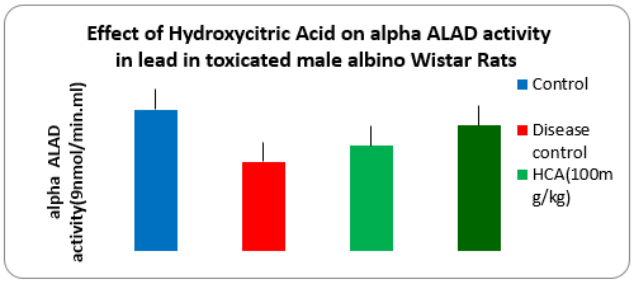


Figure 1: Effect of Hydroxycitric Acid on alpha ALAD activity in lead intoxicated male albino Wistar Rats (N=6) Effect of Hydroxycitric Acid on serum SGOT of Lead Intoxicated Rats (N=6)

After collection of blood samples, the following were estimated parmeters in rats:

1. Alad
2. Creatinine in serum /urine
3. Total protein in serum
4. Sgot & sgpt
5. Alp
6. Bilirubin

7. Results

7.1. Erum profile parameters

7.1.1. Estimation of lead induced toxicity profile parameters

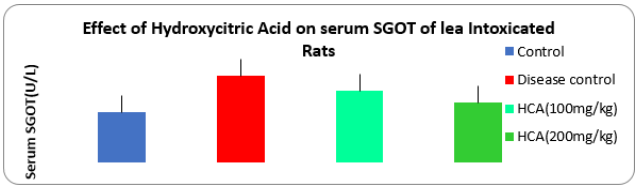


Figure 2: Effect of hydroxycitric acid on serum SGOT of lead intoxicated rat (N=6)(N=6)

All the values represents MEAN±SEM, n=6, ***P<0.001, **P<0.01, *P<0.05. When compared to Disease Control, #p<0.001compared to normal control group using One way ANOVA- Dunnett s multiple comparison test.

8. Discussion & Conclusion

Heavy metals have the ability to bioaccumulate in food chain, and children can be chronically exposed to them

Table 2: Effect of hydroxycitric acid on alpha alad activity in lead intoxicated male albino wistar rats (N=6)

Groups	Alpha ALAD activity(9nmol/min.ml)						Mean±Sem
	R1	R2	R3	R4	R5	R6	
Control	0.35	0.36	0.40	0.39	0.33	0.32	0.35±0.01
Disease control	0.22	0.24	0.22	0.22	0.23	0.24	0.22±0.01#
HCA(100mg/kg)	0.25	0.26	0.27	0.26	0.27	0.25	0.26±0.01*
HCA(200mg/kg)	0.30	0.31	0.31	0.32	0.31	0.32	0.31±0.01***

All the values represents MEAN±SEM, n=6, ***P<0.001, **P<0.01, *P<0.05. When compared to Disease Control, #p<0.001compared to normal control group using One way ANOVA- Dunnett s multiple comparison test.

Table 3: Effect of hydroxycitric acid on serum SGOT of leadIntoxicated rats (N=6)

Groups	Serum SGOT(U/L)						MEAN±SEM
	R1	R2	R3	R4	R5	R6	
Control	28.01	30.21	29.32	31.12	29.05	32.15	29.97±.0.12
Disease control	51.34	53.24	52.71	51.02	50.09	51.24	51.60±0.40$^{\#}$
HCA(100mg/kg)	41.27	45.23	43.28	45.24	42.26	40.37	42.64±0.51***
HCA(200mg/kg)	34.01	38.27	35.62	37.23	33.42	36.21	35.79±0.47***

All the values represents MEAN±SEM, n=6, ***P<0.001, **P<0.01, *P<0.05. When compared to Disease Control, #p<0.001compared to normal control group using One way ANOVA- Dunnett s multiple comparison test.

Table 4: Effectof hydroxycitric acid on serum sgpt of lead intoxicated rats (N=6)

Groups	Serum SGP T(U/L)						MEAN±SEM
	R1	R2	R3	R4	R5	R6	
Control	35.51	29.03	30.31	27.54	31.27	29.53	30.16±1.10
Disease control	37.23	36.51	49.61	39.54	40.17	49.39	41.66±2.38#
HCA(100mg/kg)	20.81	28.24	40.31	33.02	15.34	26.20	27.01±3.65**
HCA(200mg/kg)	23.81	25.37	27.51	25.07	29.21	39.15	24.33±3.41**

Table 5: Effect of hydroxycitric acid on serum alp of lead intoxicated rats(N=6)

Groups	Serum ALP (U/L)						MEAN±SEM
	R1	R2	R3	R4	R5	R6	
Control	21.32	20.21	19.12	20.25	21.51	22.61	20.83 ± 0.50
Disease control	32.18	42.51	42.47	41.61	39.71	42.24	40.12±0.24$^{\#}$
HCA(100mg/kg)	35.23	30.29	33.23	31.51	34.62	32.35	32.78±0.07***
HCA(200mg/kg)	24.32	28.21	26.51	28.65	25.20	27.57	26.74±0.21***

All the values represents MEAN±SEM, n=6, ***P<0.001, **P<0.01, *P<0.05. When compared to Disease Control, #p<0.001compared to normal control group using One way ANOVA- Dunnett s multiple comparison test.

Table 6: Mean changes difference between SGOT, SGPT and ALP

Group	Mean ±SEM		
	SG0T	SGPT	ALP
Control	29.97±0.12	30.16±1.10	20.83±0.50
Disease control	51.60±0.40$^{\#}$	41.66±2.38$^{\#}$	40.12±0.24$^{\#}$
HCA (100 mg/kg)	42.64±0.51***	27.00±3.65**	32.78±3.07***
HCA (200 mg/kg)	37.79±0.47***	24.33±3.41**	26.74±0.21***

All the values represents MEAN±SEM, n=6, ***P<0.001, **P<0.01, *P<0.05. When compared to Disease Control, #p<0.001compared to normal control group using One way ANOVA- Dunnett s multiple comparison test.

Table 7: Effect of hydroxy citric acid on serum total protein of lead intoxicated rats (N=6)

Groups	Serum total protein(G/D L)						Mean±Sem
	R1	R2	R3	R4	R5	R6	
Control	8.51	9.09	8.07	8.61	8.12	8.31	8.45 ±0.15
Disease control	13.00	14.21	13.54	14.94	15.34	15.32	14.42 ±0.41$^{\#}$
HCA(100mg/kg)	11.21	12.54	11.32	11.54	12.56	11.94	11.85 ±0.24***
HCA(200mg/kg)	9.00	9.54	9.24	9.25	10.21	9.21	9.40 ±0.17***

All the values represents MEAN±SEM, n=6, ***P<0.001, **P<0.01, *P<0.05. When compared to Disease Control, #p<0.001compared to normal control group One using way ANOVA- Dunnett s multiple comparison test.

Table 8: Effect of hydroxylcitric acid on serum bilirubin of lead intoxicated rats (N=6)

Groups	Serum Bilirubin (mg/d l)						MEAN±SEM
	R1	R2	R3	R4	R5	R6	
Control	3.1	3.2	3.4	3.1	3.5	3.7	3.28±0.07
Disease control	7.8	7.1	7.7	7.4	7.9	7.6	7.31±0.17$^{\#}$
HCA(100mg/kg)	5.2	5.7	5.1	5.3	5.3	5.5	5.33±0.09***
HCA(200mg/kg)	4.2	4.1	4.2	4.4	4.2	4.2	4.21±0.04***

All the values represents MEAN±SEM, n=6, ***P<0.001, **P<0.001, *P<0.05. When compared to Disease Control, #p<0.001compared to normal control group using One way ANOVA- Dunnett s multiple comparison test.

Table 9: Effect of hydroxycitric acid on serum creatinine of lead intoxicated rats (N=6)

Groups	Serum creatinine(mg/d l)						Mean±SEM
	R1	R2	R3	R4	R5	R6	
Control	1.24	1.26	1.26	1.21	1.23	1.25	1.24±0.01
Disease control	1.45	1.49	1.48	1.47	1.48	1.47	1.47±0.08$^{\#}$
HCA(100mg/kg)	1.36	1.38	1.41	1.39	1.38	1.37	1.35±0.08***
HCA(200mg/kg)	1.31	1.33	1.31	1.31	1.33	1.32	1.29±0.04***

All the values represents MEAN±SEM, n=6, ***P<0.001, **P<0.01, *P<0.05. When compared to Disease Control, #p<0.001compared to normal control group One using way ANOVA- Dunnett s multiple comparison test.

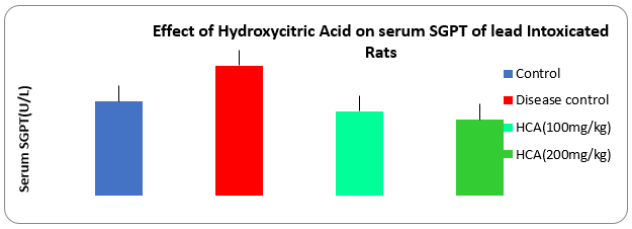


Figure 3: Effect of hydroxycitric acid on serum sgpt of lead intoxicated rats (N=6)

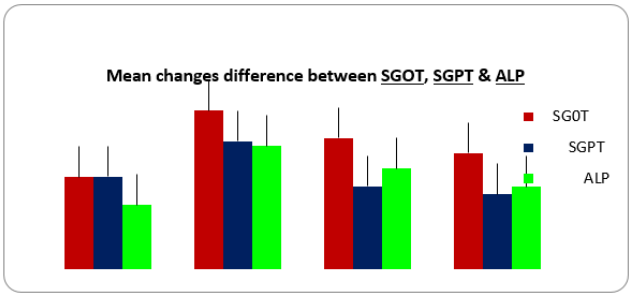


Chart 1:

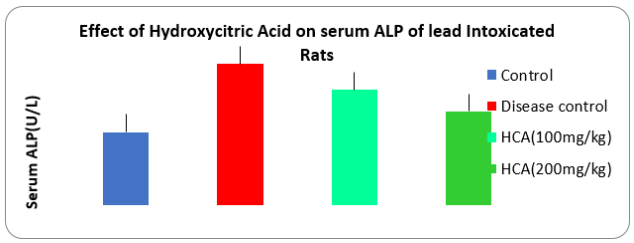


Figure 4: Effect of Hydroxycitric Acid on serum ALP of lead Intoxicated Rats (N=6)

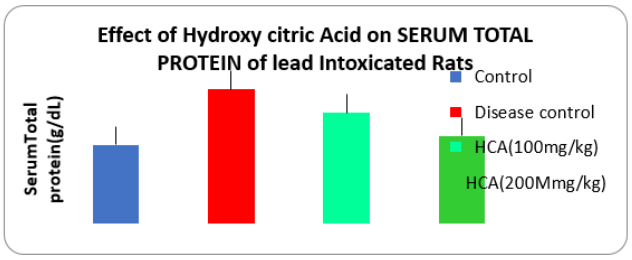


Figure 5: Effect of hydroxycitric acid on serum total protein of lead intoxicated rats (N=6)

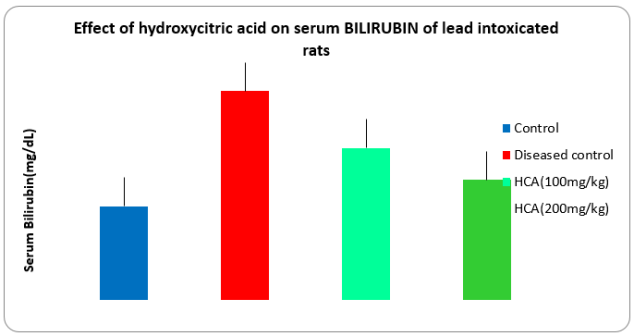


Figure 6: Effect of hydroxycitric acid on serum BILIRUBIN of lead intoxicated rats (N=6)

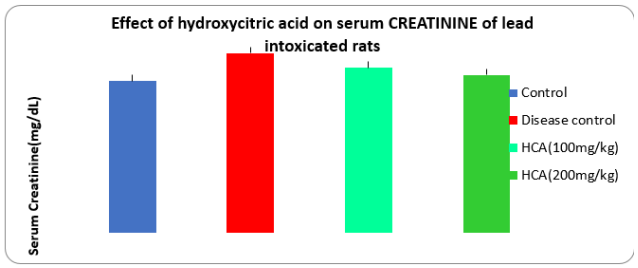


Figure 7: Effect of hydroxycitric acid on Sof lead intoxicated rats (N=6)

Table 10: Mean changes difference betweentotal protein, creatinine and bilirubin

	Mean ±SEM		
Group	**Total protein**	**Creatinine**	**Bilirubin**
Control	8.45±0.15	1.24±0.01	3.28±0.07
Disease control	14.42±0.41$^{\#}$	1.47±0.08$^{\#}$	7.31±0.17$^{\#}$
HCA (100 mg/kg)	11.85±0.24***	1.35±0.08***	5.33.±0.09***
HCA (200 mg/kg)	9.40±0.17***	1.29±0.04***	4.21±0.04***

All the values represents MEAN±SEM, n=6, ***P<0.001, **P<0.01, *P<0.05. When compared to Disease Control, #p<0.001compared to normal control group using One way ANOVA- Dunnett s multiple comparison test.

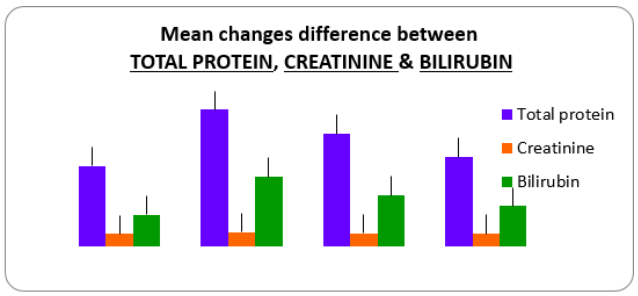


Chart 2: Mena changes diffrence between

from different sources as air, water and food leading to their accumulation in body tissues of children and causing various diseases because they act as systemic toxins with specific neurotoxic, cardiotoxicity, hepato toxicity, nephrotoxic, and teratogenic effects and they can directly influence behavior and impair mental and neurological functions. chelation was originally used to treat conditions like lead poisoning, chelation therapy is now claimed to protect against heart disease and other major health problems. The safe chelating agents available are Dimercaprol pencilamine, Dimercaptosuccinic acid EDTA. Side effects commonly associated with chelation therapy include: diarrhea, headache, high blood pressure, loose stools, low blood sugar, nausea, poor appetite, skin rash, and vomiting. In some cases, chelation therapy may trigger serious side effects such as kidney damage and abnormally. Children, pregnant women, and people with heart or kidney failure should not receive chelation therapy. Hence, herbal plants are the alternative therapy for the treatment of lead induced toxicity.

Herbal plants are show anti antioxidant activities against the free radicals and reactive oxygen species (ROS) reducing lead effect. Different medicinal plant are used for the treatment of reducing free radicals.

Hydroxycitric acid has gained recent popularity as a weight loss aid. Due to its lipid lowering properties, hydroxycitric acid may also help to lower triglyceride and low-density lipoprotein, LDL levels.

Based on the above reports on the herbal based drug therapy for lead induced toxicity, the present study was designed to evaluate the role of herbal active constituent hydroxycitric acid obtained from Garcinia cambogia and Hibiscus subdariffa.

The Lead acetate at 500ppm was used as an inducing agent in the present study. Sodium acetate (500ppm) used as control group is used as a baseline measure. The control group is identical to all other subjects that you are examining with the exception that it does not receive the treatment and as the conjugate base of acetic acid, a solution of sodium acetate and acetic acid can act as a buffer to keep a relatively constant pH level. The HCA at 100 mg/kg and 200mg/kg were used in the treatment of lead induced toxicity in rats. At the end of 28 days study period the blood levels of alpha ALAD activity was estimated in all the treatment groups. Lead inhibits the ALAD is more profound and its inhibition has been used clinically to gauge the degree of lead poisoning. Inhibition of ALAD by lead leads to anemia primarily because it both inhibits heme synthesis and shortens the lifespan of circulating red blood cells, but also by stimulating the excessive production of the hormone erythropoietin, leading to inadequate maturation of red cells from their progenitors. Inhibition of ALAD results in the accumulation of aminolevulinic acid, detectable in the plasma and urine

even at blood lead levels of less than 10 μg/dl (Ahamed et al., 2005). The Table 5 indicates the effect HCA on alpha ALAD activity. It result that HCA showed significant elevated levels of ALAD activity, which was reduced by the lead intoxication.

Several animal investigations have been performed on hepatotoxicity of lead, although clinical studies concerning the manifestations of lead induced liver toxicity in humans and the absorbed lead accumulates mostly in soft tissues, bones and liver (Dongre NN., 2010). Lead induces overproduction of reactive oxygen species, which can result in peroxidation damage to hepatic cell membranes. Damage to hepatic cells leads to release of transaminases and increases their levels in serum (Aziz II., 2006). Hence, the study was conducted to estimate the liver enzymes in lead induced intoxication in rats.

HCA showed significant reduction of liver enzymes (SGOT, SGPT and ALP) in a dose dependent manner in lead intoxicated rats. The markers like protein, bilirubin and creatinine were also found to be elevated in lead intoxicated rats and was found to be decreased significantly in a concentration dependent manner.

9. Source of Funding

None.

10. Conflict of Interest

None.

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