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Ameliorative Effects of Bauhinia acuminata stem bark extract against chronic arsenicosis in rats

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Abstract

This study was conducted to evaluate the effect of *Bauhinia acuminata* (BA) stem bark against sodium arsenite ($NaAsO_2$) induced toxicosis in adult albino rats. Forty eight (48) adult albino rats having body weight 150-200 gm of either sex were randomly divided into four groups (A_0, A_1, A_2 and A_3) each containing twelve. Sodium arsenite was administered at 4mg/kg daily in drinking water for 90 days to all rats of group A_1, A_2 and A_3. The rats of group A_2 was orally treated with stem bark extract of BA ($^1/_{10}^{th}$ of LD_{50}) at 300mg/kg daily from 91 day to 120 day whilst stem bark extract of BA ($^1/_{20}^{th}$ of LD_{50}) at 150 mg/kg daily orally was administered in group A_3 from 91 day to 120 day. Only water was given to rats of group A_0 and considered as control.

Blood samples were collected at different days for analysis of haemogram, biochemical parameters like ALT, AST, BUN, CRT. Tissue samples were collected to study activity of SOD, MDA, GSH and catalase. Histopathology of some vital organs also conducted after completion of experiment.

The results reveal that stem bark extraction of *Bauhinia acuminata* may be effective to reduce the toxic effect of arsenic.

Key words : Arsenic, rats, *Bauhinia acuminata*, Histopathology, Antioxidant activity, Biochemical parameters, Haemogram.

Introduction

Arsenic is the naturally occurring metalloid ubiquitously present in the environment in both organic and inorganic forms. Exposure to arsenic occurs through both natural and anthropogenic sources. Human health in the past and present is influenced by the amounts and proportion of chemical elements to which humans have been exposed.

Recent circumstantial evidence, on the other hand, suggests that arsenic may be an essential ultra trace element in animals (Bogden *et al*, 2000). Arsenic deprivation has been induced in chickens, goats, hemsters, pigs and rats depressed growth, impaired fertility, with increased perinatal mortality in pigeons and goats with death during lactation, decreased serum triglycerides, myocardial damage; arsenic deprivation may also lead to changes in mineral concentration in various organs and kidney calcification in rats with high calcification inducing diets (Bogden *et al*, 2000).

Arsenic occurs in three forms : Neutral (Zero Valence), Trivalent and Pentavalent. For chemical convenience, arsenicals are grouped into organic and inorganic compounds in several important pharmacological aspects. The major toxic action of arsenicals in parasite and the host is inhibited of sulfhydroxyl (SH) enzyme. This was well reviewed by Harvey (Harvey SC, 1975). In general, inorganic arsenic is more toxic than the organic and trivalent arsenic is more toxic than pentavalent and zerovalent arsenic.

Chronic exposure to arsenic is associated with a wide range of toxic effects cancer of the skin, lung, kidney, liver and urinary bladder are the important cancers associated with these toxic effects (Mazumder *et al*, 2011; Srivastava *et al*, 2009, Suwalsky *et al* 2008). Inspite of this large body of information about the toxic effects of arsenic, the precise mechanisms of action for the many diseases end points following acute and chronic arsenic exposure, as well as the threshold for biologic effects and disease risks, remain enigmatic.

The metabolism of arsenic like other toxic metals is associated with the conversion of the most potent toxic form of this element to the less toxic form, following by cellular accumulation or excretion. Bio-methylation of arsenic is considered the primary detoxification mechanism, since the inorganic arsenics are more toxic to the living organisms (Yamauchi and Fowler, 1994).

Arsenic toxicity differs in a fundamental fashion from that of other “Protoplasmic poison” which act by denaturing and precipitating the cellular proteins.

Arsenicals can cause cellular damages through the generation of free radicals. Several studies suggest that arsenic compounds may also exert their toxicity through the generation of reactive oxygen species such as superoxide, hydroxyl radicals, hydrogen peroxide and nitric oxide during their metabolism in the cells (Hei and Filipic, 2004; Liu *et al*, 2005).

The plant *Bauhinia acuminata* L (Leguminosae:caesalpinioideae) commonly known as Dwarf white in English is a medium size, Oeciduous tree found in central India (Dhale 2011).

The bark and leaves in a decoetion helps relief biliousness. In Malaysia and Indonesia the plant is used in the treatment of common cold and cough (Timothy 1999).

In India the decoction of the leaves and bark is given for allergic asthmatic attack (Khare, 2007).

The plants of *Bauhinia acuminata* are widely distributed in India. The Indians made used of the bark and leaves in a decoetion to treat stones in the bladder, veneral diseases and leprosy (Khare, 2007).

Amongst the Mullu Kuruma tribe of Kerala the decoction of the bark is used in treating urinary discharge (gonorrhea). Paste of the leaves applied on the throat for throat troubles. It is applied externally to treat skin diseases (Silja *et al*, 2008). The roots is boiled in oil and applied to burns.

But no reports have been available for its effects on heavy metal toxicity in general and arsenic toxicity in particular considering above, the present study undertaken to explore its efficacy on induced arsenicosis in rats if any.

Materials and methods :

Chemicals : All chemicals and kits used in this present study were obtained from Bengalore Geni (India), Congent (India), Merck (Germany), Rankem (India) and Sigma Chemicals (USA).

Experimental Animals : Forty eight white albino rats of either sexes having body weight 150-200 gm were procured from registered animal breeder. They were caged in polypropylene cages and were acclimatized in experimental animal room for seven days before starting the experiment. The animals were maintained with standard pellet feed and provide drinking water *ad libitum*. The Institutional Animal Ethics Committee approved the technical programme.

Preparation of *Bauhinia acuminata* (BA) stem bark extract

At first the plant identification was made from BSI (Botanical Survey of India), Howrah, Kolkata and specimen number of BA was WBUAFS/KOL/2.

After collection of *Bauhinia acuminata*, stem barks were collected, washed with water, shade dried and powdered coarsely. Then 50 gm of bark powder was put into soxhlate apparatus and 200ml of ethanol (99.9%) were added and heated up at 60°C - 70°C.

Keeping this process upto 8 to 12 hrs. extraction was completed. After completion of extraction whole solution was condensed through rotary evaporation .Condensed solution were kept in room temperature for 2 to 3 days. This semi-solid solution was dissolved in water (triple distilled water) and feeded to rat.

Determination of LD_{50} of *Bauhina acuminata*

Healthy albino rats (either sex) were used to determine LD_{50} of *Bauhinia acuminata* stem bark extract described by Ghosh MN, 2008 and it was found to be 3000 mg/kg. Two dose levels i.e. $^1/_{10}{}^{th}$ (300 mg/kg) and $^1/_{20}{}^{th}$ (150mg/kg) LD_{50} of *Bahunia acuminata* were selected for the present study.

Experimental design :

Forty eight animals were randomly divided into four groups having twelve rats in each (groups A_0, A_1, A_2, A_3). Rats in group A_0 were given feed and water *ad libitum*. Each rat in groups A_1, A_2 and A_3 were treated with sodium arsenite (4mg/kg) daily in drinking water for 90 days and ethanolic extract of *B. acuminata* was administered at 300mg/kg and 150mg/kg dissolved in distilled water to animals of group A_2 and A_3 respectively from 91 to 120 days. Animals of group A_1 were considered as experimental control/untreated control group.

Collection of samples : Blood samples were collected on day 0, 14, 28, 42, 60, 90 and 120 from animals of each group. Tissue samples were collected on day 0, 90 and 120 after sacrificing four animals in each group.

Blood : Pooled blood samples were collected from the tail vein of 3 rats (1ml per rat) of each group according to procedure of Brown 2006 and kept 1ml blood in EDTA treated test tube for haemogram and 2ml into pre-marked centrifuge glass test tubes immediately after collection and was kept at room temperature for 1hr. without agitation to clot with a view to collect serum. The harvested sera were kept at -20°C until used for biochemical parameters.

Tissue : The rats were sacrificed by maintaining standard protocol using higher dose of ketamine. Pieces of liver, kidney, heart, spleen, lung and intestine from each rat were collected and fixed in 10% buffered formalin for his pathological examination, while sonal part of liver, kidney and heart were used for antioxidant status.

Haemogram : Haemoglobin Total RBC, Total WBC, PCV and differential count were determined as per standard method (Coffin 1953).

Biochemical parameters : Serum AST and ALT activity was measured by the method of Reitman and Frankel (1957), BUN by Wybenga *et al* (1971) and CRT by Toro and Ackermann (1975).

Tissue Biochemical (antioxidant status) : Reduced glutathione (GSH) (Jollow *et al* 1974), Superoxide dismutase (SOD) (Marklund and Marklund 1974), MDA (Ohkawa et al 1979), Catalase (Aebi 1984) activity in liver, kidney and heart tissues were determined.

Statistical analysis : The results were expressed as Mean±SE. The data were analysed statistically by using Univariant General Linear Model with two ways ANOVA in SPSS 10 Version of software.

RESULTS AND DISCUSSION

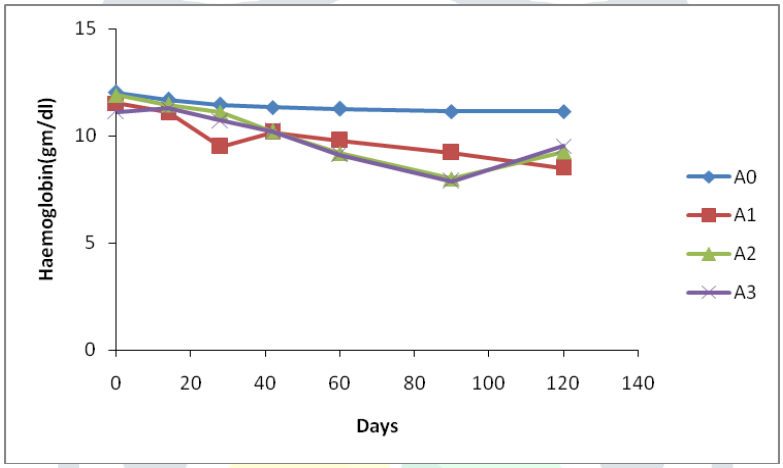

Fig.1:Effect of arsenic on haemoglobin (gm/dl) level in rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by *Bauhinia acuminata* stem bark extract after 90 days onwards.

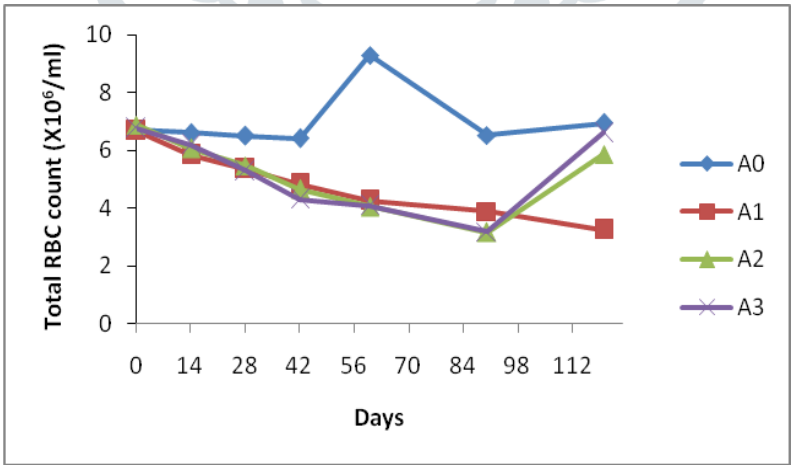

Fig. 2: Effect of arsenic on total RBC count ($X10^6$/µl) level in rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by *Bauhinia acuminata* stem bark extract after 90 days onwards.

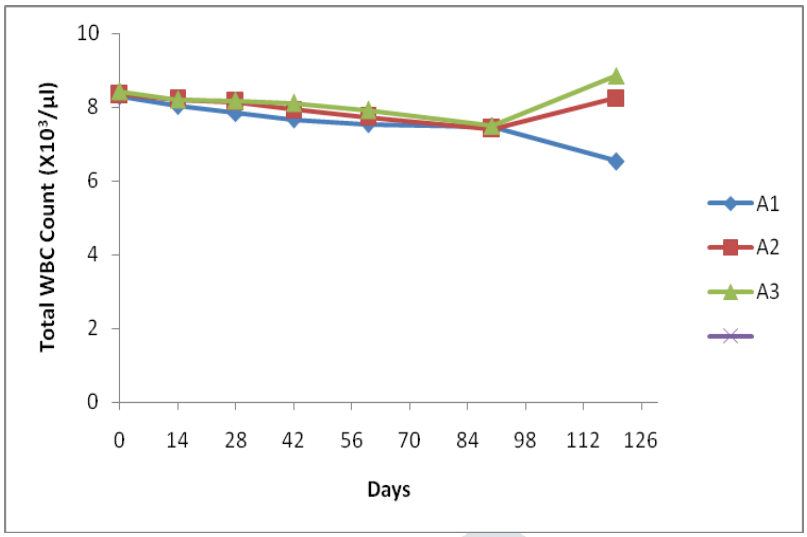

Fig.3: Effect of arsenic on total WBC count (x10^3/µl) in rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by *Bauhinia acuminata* stem bark extract after 90 days onwards.

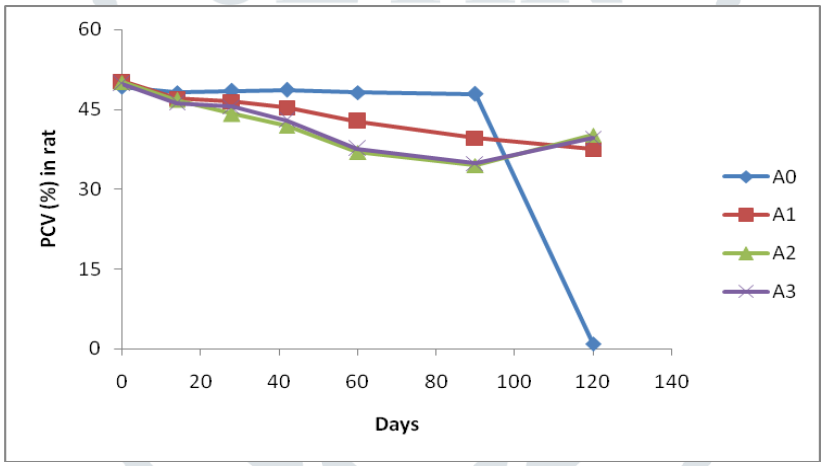

Fig.4: Effect of arsenic on PCV (%) in rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by *Bauhinia acuminata* stem bark extract after 90 days onwards.

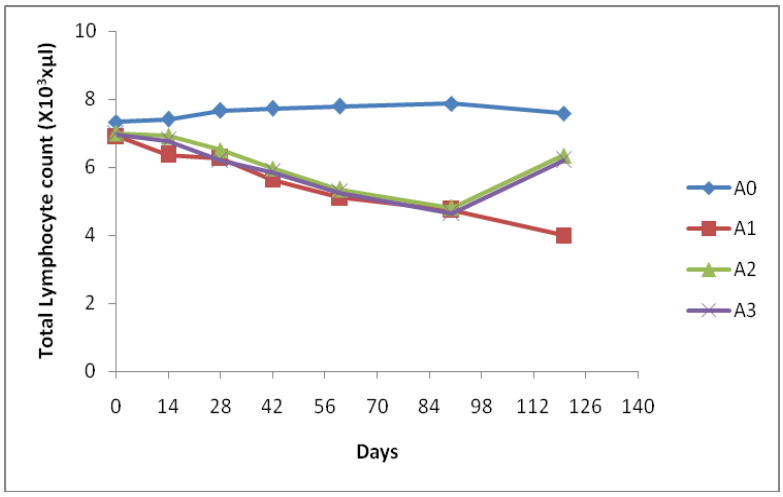

Fig5: Effect of arsenic on total lymphocyte count (X10^3/µl) level In rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by *Bauhinia acuminata* stem bark extract after 90 days onwards.

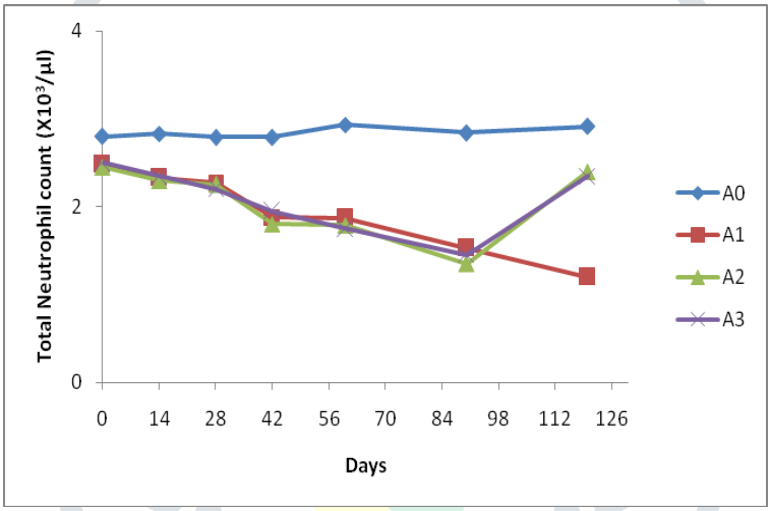

Fig. 6: Effect of arsenic on total Neutrophil count (X10^3/µl) level in rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by *Bauhinia acuminata* stem bark extract after 90 days onwards.

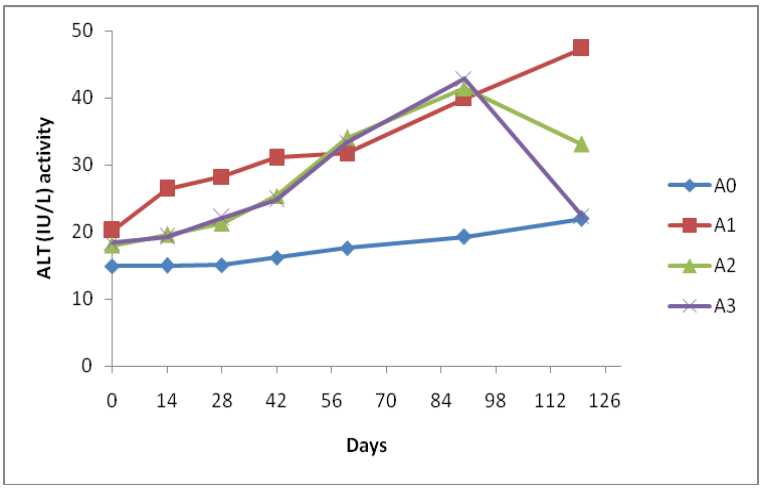

Fig.7: Effect of arsenic on ALT (IU/L) activity in rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by *Bauhinia acuminata* stem bark extract after 90 days onwards.

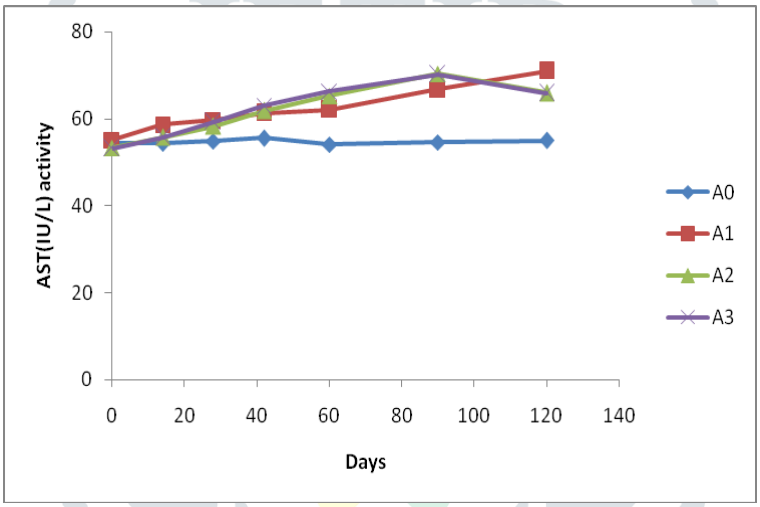

Fig. 8: Effects of arsenic on AST (IU/L) activity in rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by *Bauhinia acuminata* stem bark extract after 90 days onwards.

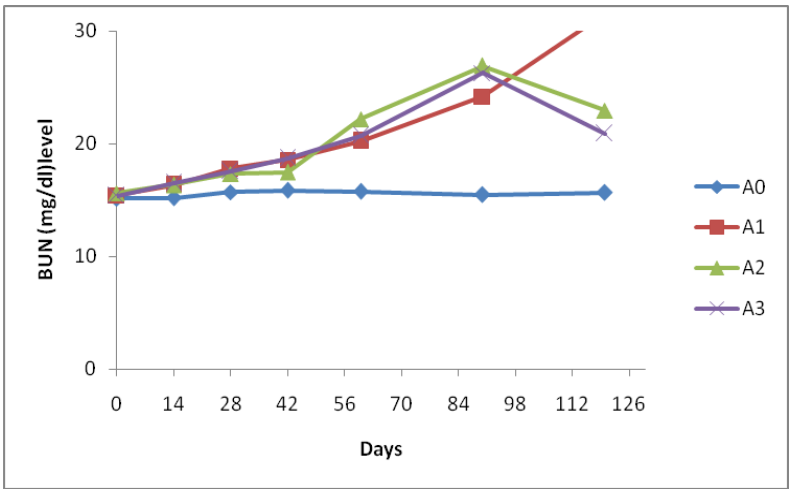

Fig.9: Effect of arsenic on BUN (mg/dl) level in rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by *Bauhinia acuminata* stem bark extract after 90 days onwards.

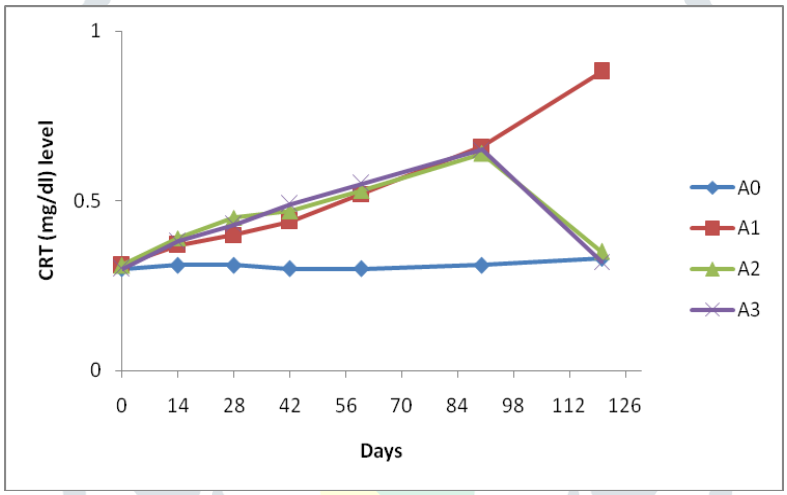

Fig.10: Effect of arsenic on CRT (mg/dl) level in rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by *Bauhinia acuminata* stem bark extract after 90 days onwards.

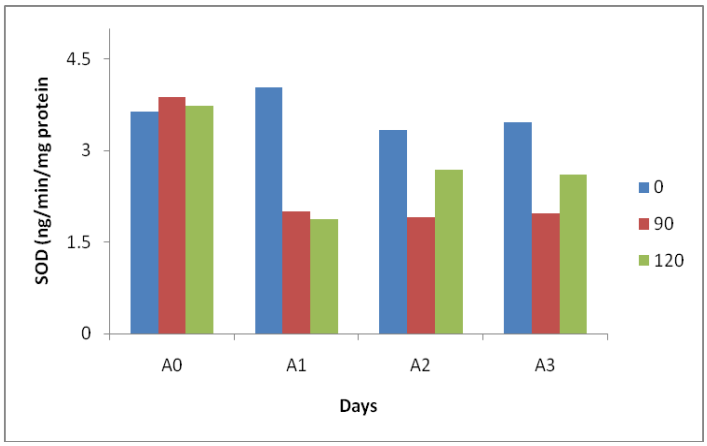

Fig.11: Effect of arsenic on SOD (ng/min/mg prtein) in heart of rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by BA stem bark extract after 90 days onwards.

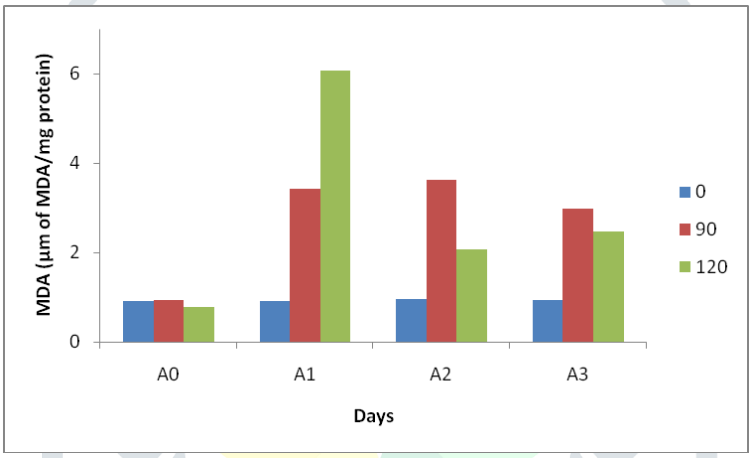

Fig.12: Effect of arsenic on MDA (µm of MDA/mg prtein) in heart of rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by BA stem bark extract after 90 days onwards.

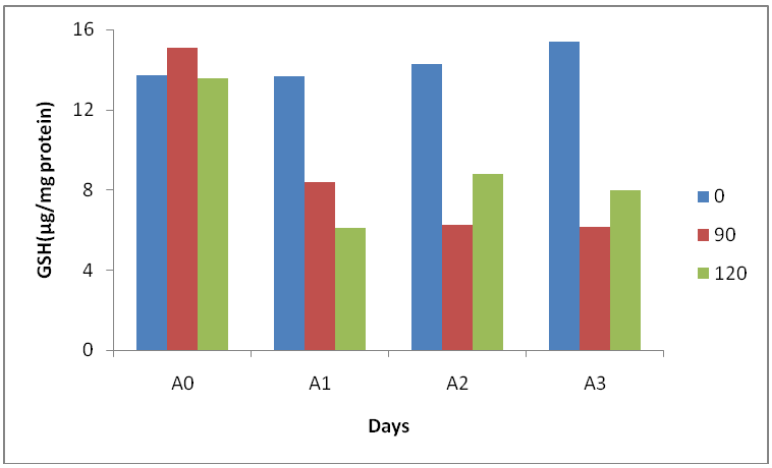

Fig. 13: Effect of arsenic on GSH (µg/mg prtein) in heart of rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by BA stem bark extract after 90 days onwards.

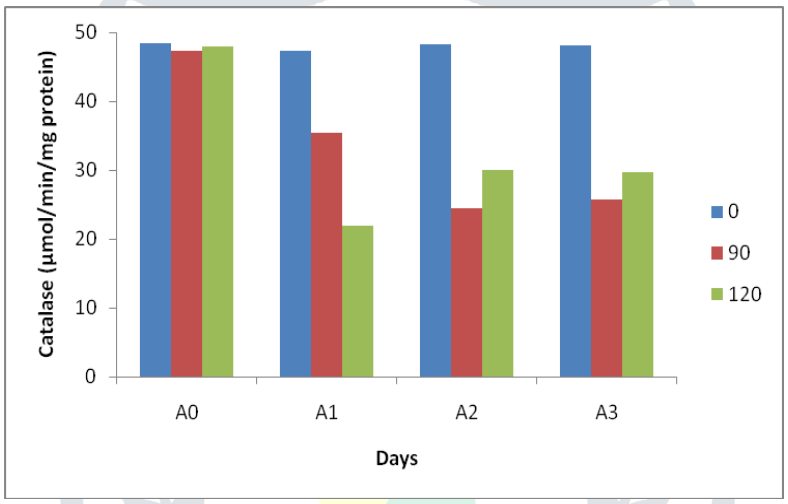

Fig.14 : Effect of arsenic on catalase (µmol/min/mg prtein) in heart of rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by BA stem bark extract after 90 days onwards.

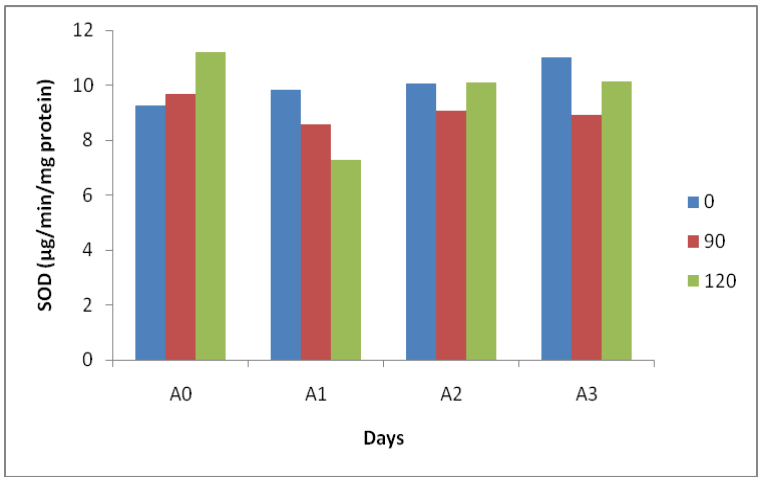

Fig.15: Effect of arsenic on SOD (µg/min/mg prtein) in liver of rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by BA stem bark extract after 90 days onwards.

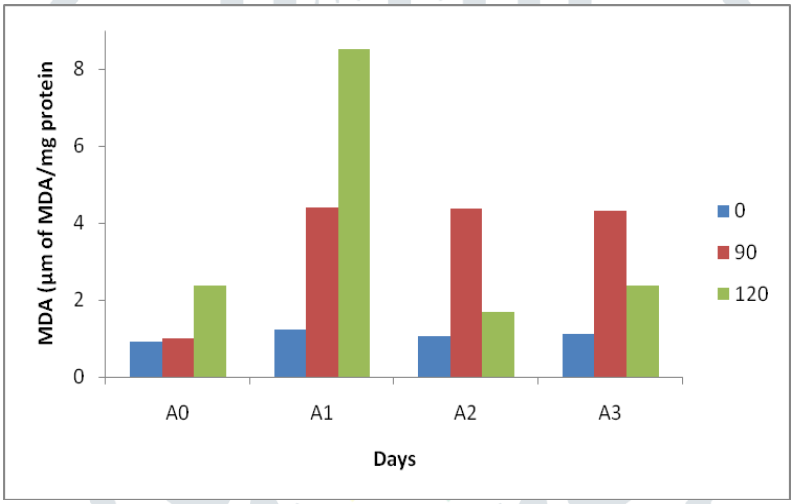

Fig.16: Effect of arsenic on MDA (µm of MDA/mg prtein) in liver of rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by BA stem bark extract after 90 days onwards.

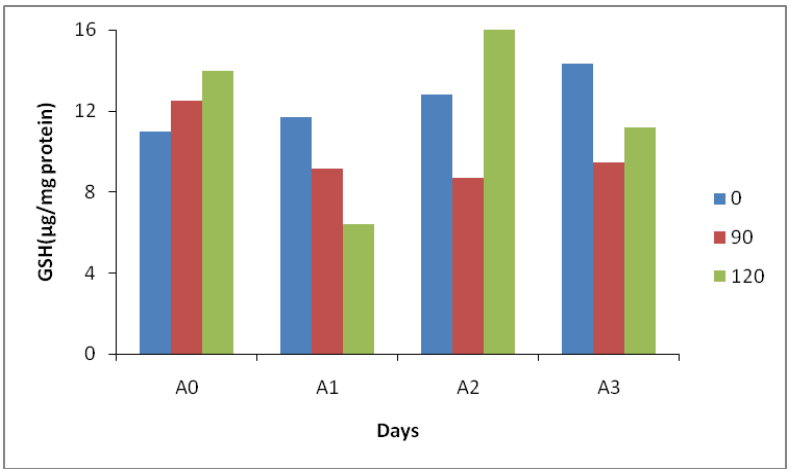

Fig. 17: Effect of arsenic on GSH (µg/mg prtein) in liver of rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by BA stem bark extract after 90 days onwards.

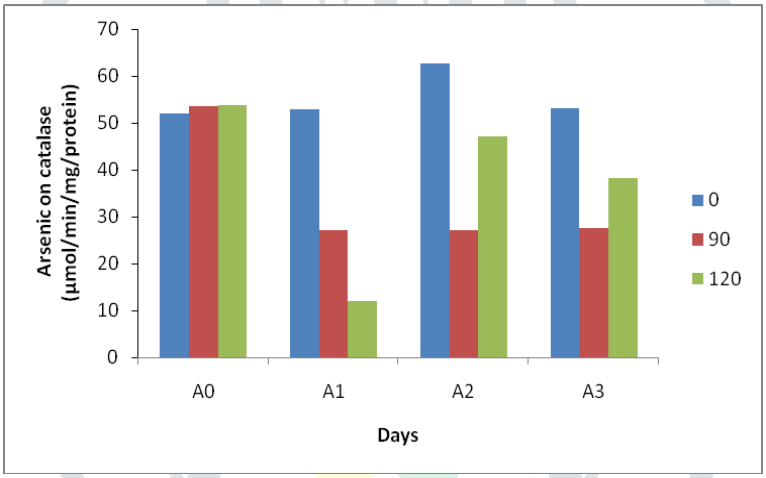

Fig. 18 : Effect of arsenic on catalase (µmol/min/mg prtein) in liver of rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by BA stem bark extract after 90 days onwards.

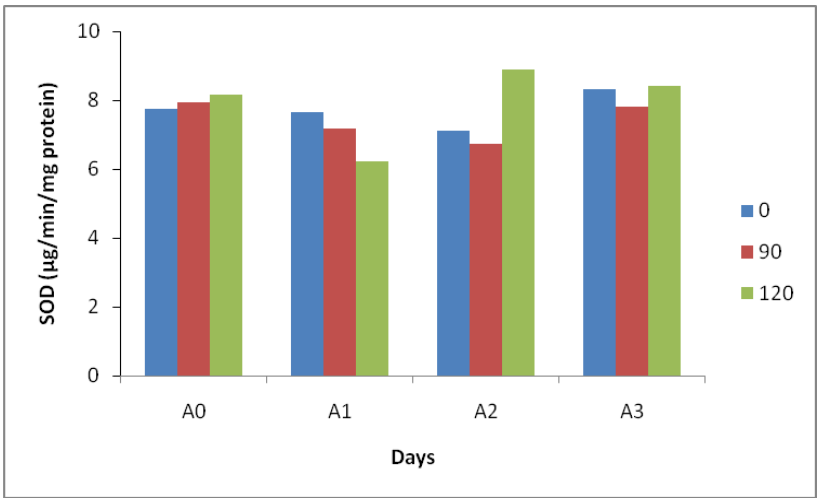

Fig. 19: Effect of arsenic on SOD (µg/min/mg prtein) in kidney of rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by BA stem bark extract after 90 days onwards.

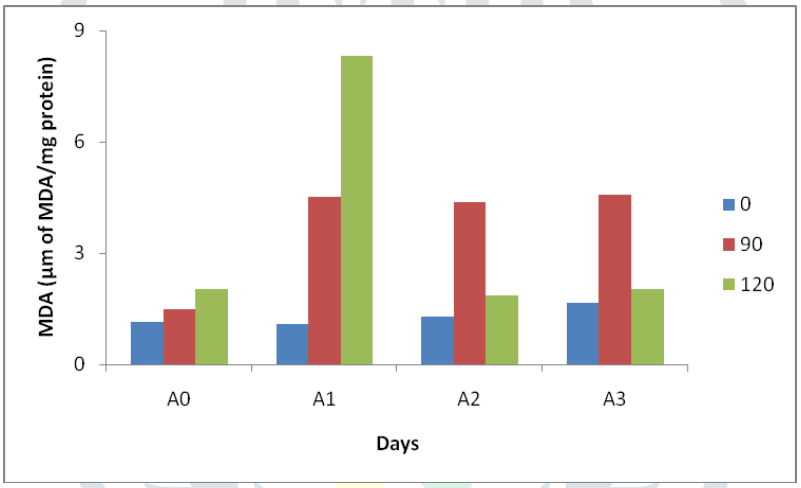

Fig. 20: Effect of arsenic on MDA (µm of MDA/mg prtein) in kidney of rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by BA stem bark extract after 90 days onwards.

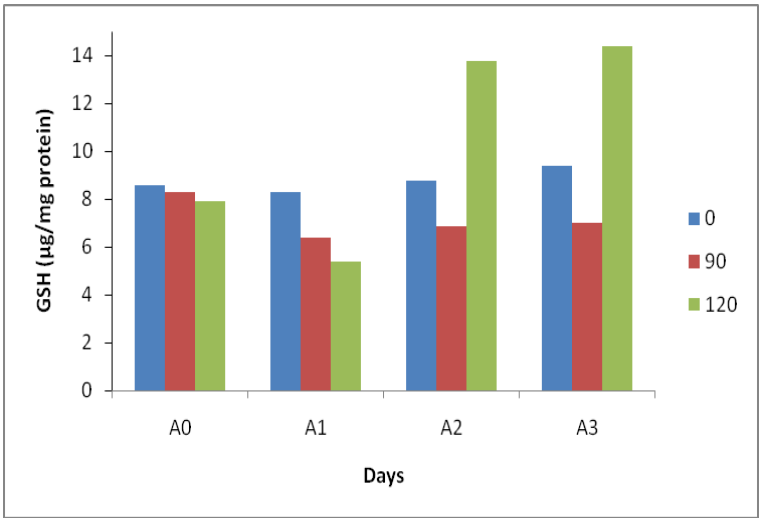

Fig.21: Effect of arsenic on GSH (µg/mg prtein) in kidney of rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by BA stem bark extract after 90 days onwards.

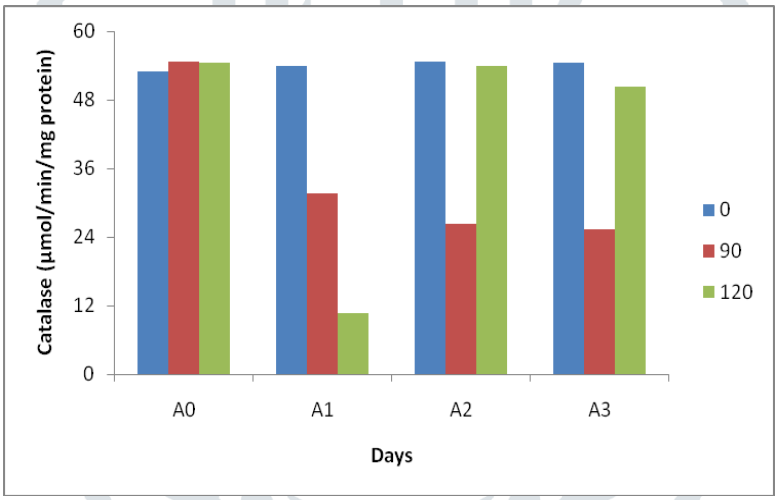

Fig.22 : Effect of arsenic on catalase (µmol/min/mg prtein) in kidney of rat after daily single oral administration of sodium arsenite at 4mg/kg for consecutive 90 days and ameliorative effect by BA stem bark extract after 90 days onwards.

Histopathological Findings

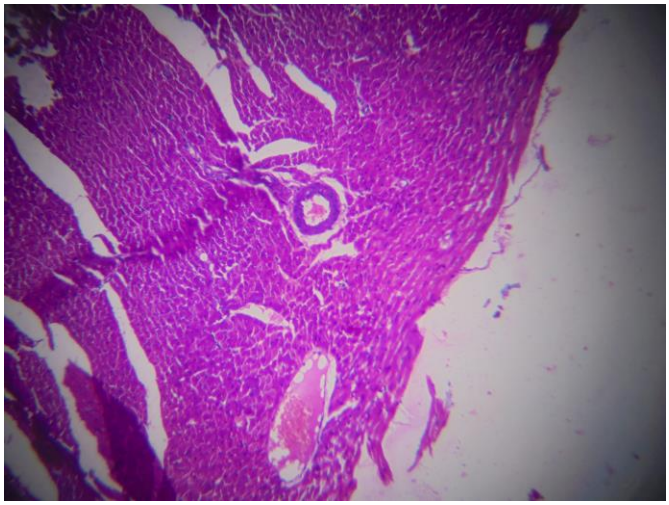

Fig.23:Cross section of heart shows congested blood vessels in the myocardium in arsenicosis induced rats(H &E10X)

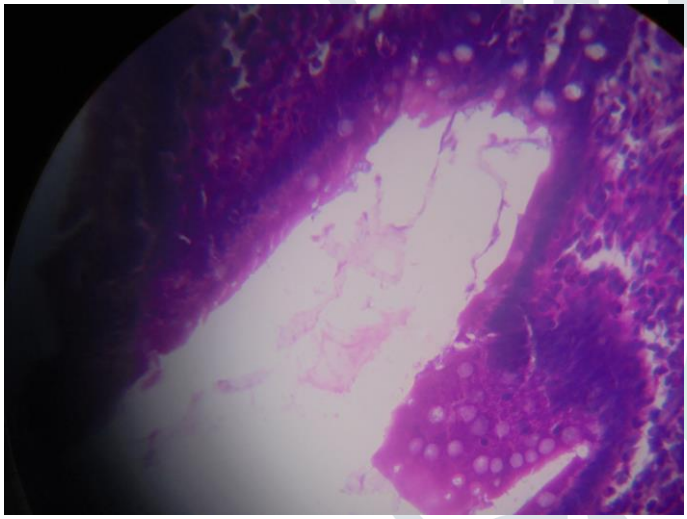

Fig.24:Crosssection of intestine shows excessive mucous secretion in arsenicosis induced rats (H &E 10 X).

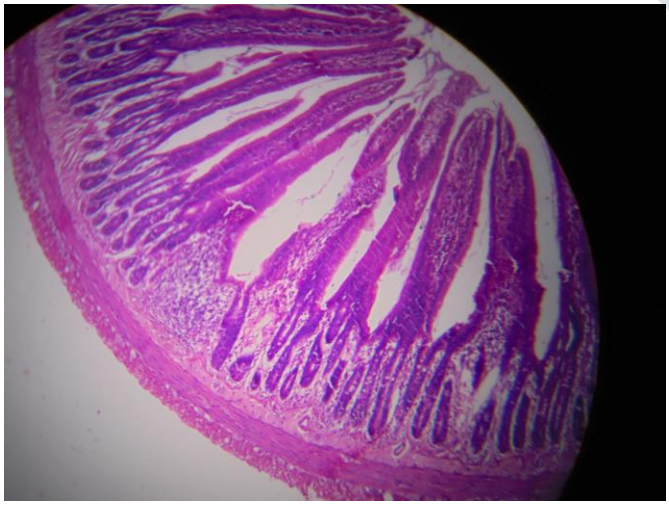

Fig.25:Cross section of intestine of BA 1/10 rats (H & E 10 X) indicate some emoliant action in the intestinal mucosa.

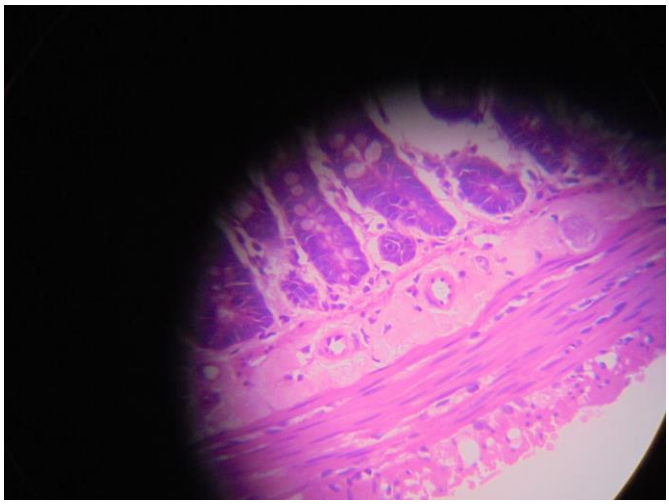

Fig.26:Cross section of intestine of BA 1/20 rats (H & E 40 x) reduced the inflammatory condition of the intestine through oedema in the submucosa and muscularis mucosa.

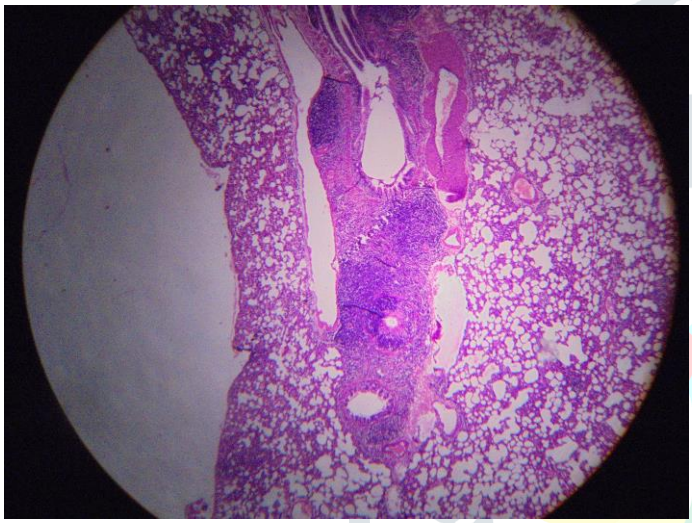

Fig.27:Cross section lungs shows local pneumonic lesion in arsenicosis induced rats (H & E 10X)

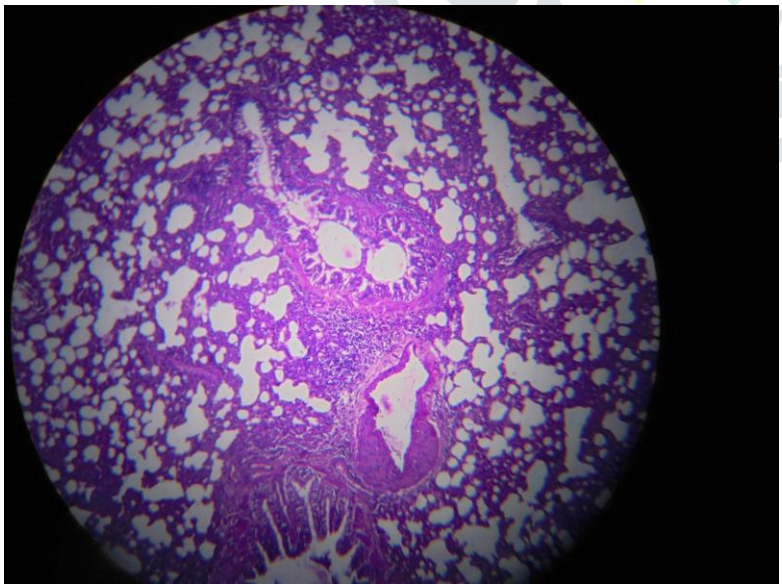

Fig.28:Cross section of lungs shows pneumonic lesions in BA1/10th treated rats (H & E 10X).

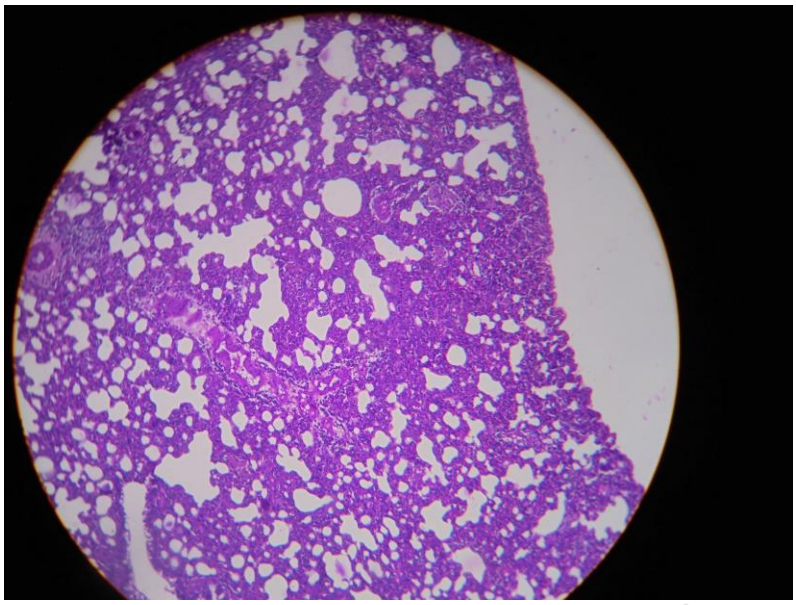

Fig.29:Cross section of lungs shows pneumonic lesions in BA1/20th treated rats (H & E 10X).

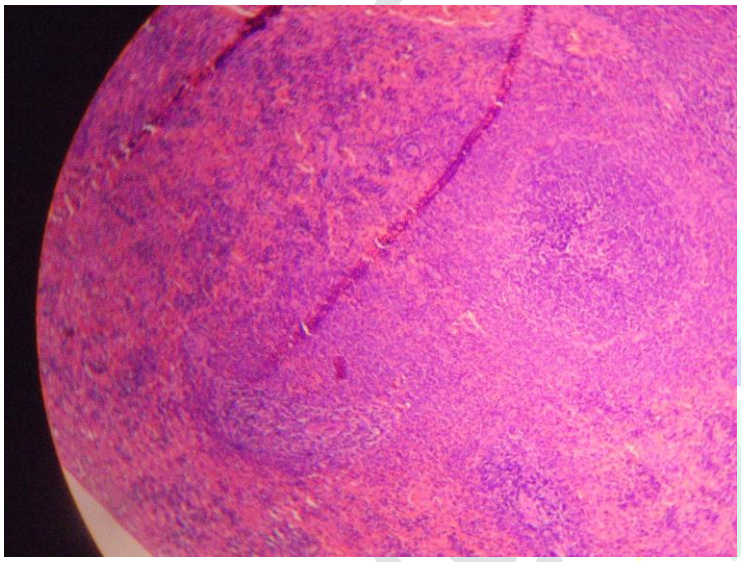

Fig.30:Cross section of spleen shows degeneration of the lymphocytes in the germinal foci of white pulp in arsenicosis induced rats(H & E 10X).

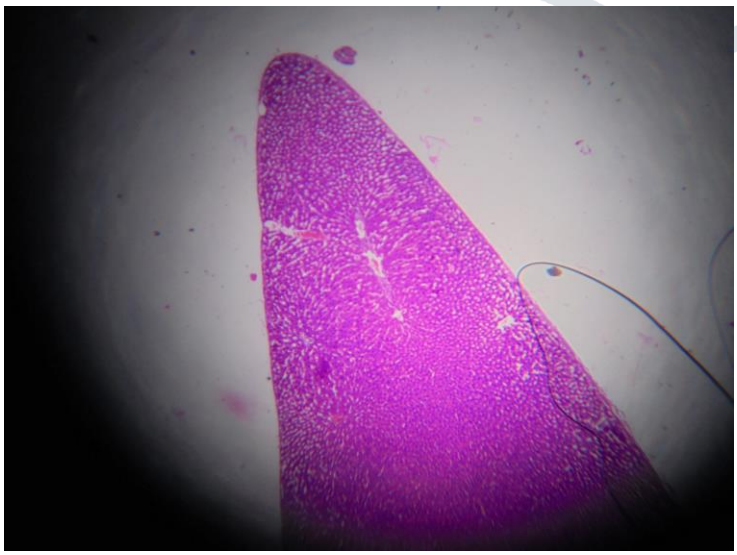

Fig.31: Cross section of liver shows massive fatty changes at the periphery and of the lobule in arsenicosis induced rats (H & E 10 X).

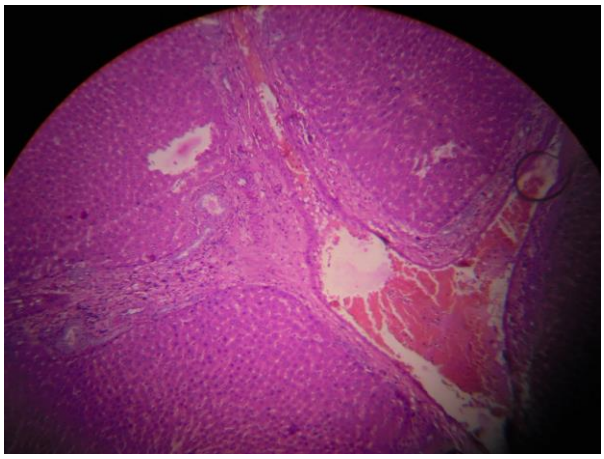

Fig.32:Cross section of liver shows reparative changes around the lobular septa in BA(1/20 of LD_{50}) treated rats (H & E 40 X).

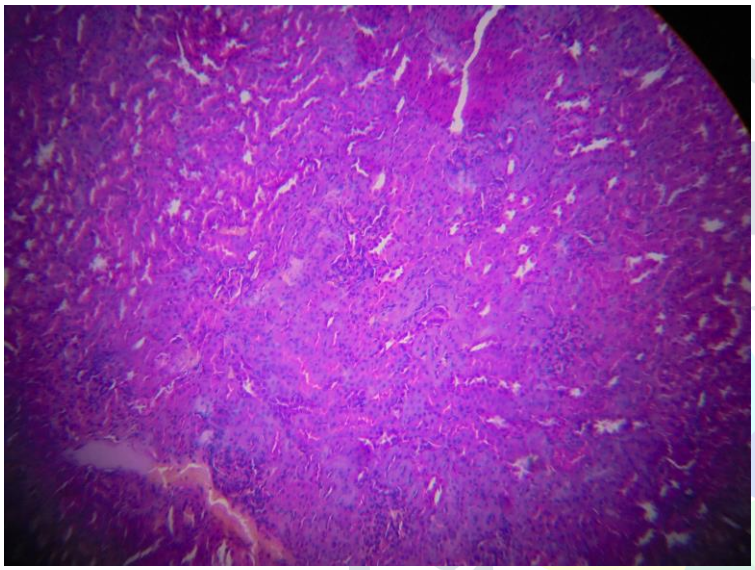

Fig.33: Cross section of kidney shows damaged Bowmen capsule in the cortex in arsenic induced rats (H & E 10 X).

It is clear from fig.1, 2 and 4 that the level of haemoglobin, total RBC count and PCV did not alter on respective days for group A_0 animals but the (Group A_1, A_2 and A_3) above values significantly ($P<0.05$) decreased till 90 days with respect to '0' day value in A_1, A_2 and A_3 groups. The values significantly ($P<0.05$) decreased on day 120 in group A1 significantly ($P<0.05$) but increased in A2 and A3 treated with *Bauhinia acuminata* stem bark extract.

It is depicted from the figures 3,5, 6 that WBC, lymphocyte and neutrophil counts significantly decreased ($P<0.05$) till 90 day in A_1, A_2 and A_3 compared to '0' day but the value significantly ($P<0.05$) decreased on day 120 in group A_1 but increased in A_2 and A_3 treated with *Bauhinia acuminata* stem bark extract.

The reduction in haemoglobin content may be related to decrease amount of RBC number which indicates hemolysis, hemorrhage and reduced erythropoiesis in pathophysiological condition.

In the present study, there is an increase in the total WBC count for group A_2 and A_3 animals on day 120. The cause of enhancement of WBC count may effect the defensive mechanism against the pathophysiological conditions in the body. The findings are similar with the findings of Charles 2014.

Arsenic is a toxic element for human and livestock causing serious health hazards. Biochemical and haemogram indices are the reliable parameters for assessment of the health status of humans and animals in arsenic toxicity (Ohaeri *et al*, 2011; Sexena DP *et al*, 2011).

It is observed from Fig.12, 16 and 20 that MDA of heart, liver and kidney did not alter significantly ($P<0.05$) in group A_0 at different days compared to its '0' day value. Again those values significantly ($P<0.05$) increased on day 90 for all groups but decreased on day 120 for group A_2 and A_3 animals and increased for group A_1 animals.

It is also clear from Fig.11, 15, 19, 14, 18, 22, 13, 17, 21 that SOD activity, catalase and GSH level of heart, liver, kidney, did not alter significantly ($P<0.05$) in group A_0 at different days compared to its day '0' value whilst the activity was decreased significantly ($P<0.05$) on day 90 and day 120 in arsenic treated animals (group A_1) but the activity was increased on day 120 in rats of groups A_2 and A_3 administered with extract of BA at two dose levels.

Adequate level of the cellular GSH pool required not only for maintaining the cellular redox status by keeping sulfhydryl groups of cytosolic proteins in their reduced form. A decrease in cellular GSH concentration has been inversely correlated with lipid peroxidation in the liver, therefore, an increased GSH concentration by the BA treatment could presumably protect the organ from arsenic induced lipid peroxidation (Maiti *et al*, 2001).

Anti-oxidant enzymes are considered to be the first line of cellular defense against oxidative damage. SOD is an anti-oxidant metallo-enzyme that reduces super oxide radicals to water and molecular oxygen (Cord *et al* 1976). CAT is a hemo-protein, which reduces hydrogen peroxide to molecular oxygen and water (Gutteridge, 1995).

In contrast to this experiment, significantly increase of SOD and CAT activities with increased LPO levels have been documented in the blood, liver and kidneys of arsenic treated rats (Nandi D *et al* 2008).

Arsenic causes toxicity through its interaction with sulfhydryl groups of proteins and enzymes to denature the proteins and enzymes within the cells and also through an increase of ROS in the cells, consequently

causing cell damage (Charles, 2014). It has been reported that arsenic induced hematotoxicity is associated with As induced oxidative stress, imbalance o antioxidant system, increased lipid peroxidation resulting heme dysfunction through influencing heme biosynthesis pathway (Kiran *et al* 2007; Mishra *et al* 2008; Sinan *et al* 2013; Vijay *et al*, 2009). The present results also showed that exposure to As significantly increase the oxidative stress which is supported with the increase level of lipid peroxidation and decreased level of non enzymic and enzymic antioxidants. But stem bark extract of BA has significantly improved all these altered parameters in arsenic intoxicated rat.

It has been reported that arsenic induced hematotoxicity is associated with As induced oxidative stress-imbalance of anti-oxidant system, lipid peroxidation was increased by high arsenic level and duration.

Arsenic induced MDA production could be due to impairement of cells natural protective system. The present results also showed that exposure to As significantly increase the oxidative stress whch is supported with the increased level of lipid peroxidation and decreased level of non enzymatic and enzymatic anti-oxidants. But stem bark extract of BA has significantly improved all these altered parameters in arsenic intoxicated rat.

Increased oxidative stress represents an imbalance between intracellular production of free radicals and the cellular defense mechanisms; notably, MDA is one of the most important markers of oxidative stress (Charles, 2014). Extensive research demonstrated that arsenic causes oxidative stress in a dose and time-dependent manner (Lantz *et al*, 2006), and increase the levels of MDA, deprete GSH and decrease activities of antioxidant enzymes such as SOD and CAT. The stem bark extract of BA was found to produce a significant less lipid peroxides than arsenic-treated rats.

Section of heart of group A_1 animals revealed that blood vessels were congested in the myocardium with focal loss of striations of myocardial muscles and no necrotic changes were observed (Fig.23). Section of heart of group A_2 and A_3 animals did not reveal any significant changes apart from congestion of myocardial vessels following treatment with BA stem bark extract at two dose levels.

Section of intestine of group A_1 animals showed an excessive mucous secretion (Fig.24). Rats feed with sodium arsenite treated with BA 1/10 (Fig.25) reduce the congestion and mucous secretion of the intestine which indicate some emoliant action in the intentional mucosa. The rats treated with BA 1/20 (Fig.26) reduced the inflammatory condition of the intestine through oedema in the sub mucosa and muscularis mucosa reveal exudation from vessels might have some flashing activity of the toxic substances locally.

The local pneumonic lesion was evident in the lungs of arsenic treated animals (A_1) (Fig.27), whilst pneumonic lesions were evident with oedema and epithelization of the alveoli in animals of group A_2 and A_3 (Fig. 28 & Fig.29).

Section of spleen of group A_1 animals shared the follicular proliferation of the lymphocytes of the white pulp extending to red pulp. Degeneration of the lymphocytes in the germinal foci of white pulp was found (Fig.30). Section of spleen of group A_2 and A_3 animals showed moderate follicular lymphocytic proliferation infiltrating the red pulp.

It may be observed that BA stem bark extract acted as scavenger of superoxide and hydroxyl radical. The results also showed that arsenic exposure to rats caused a significant reduction in GSH level and decreased activities of SOD suggesting arsenic-induced oxidative stress. The treatment with Ba stem bark extract was able to restore the activities of SOD, catalase and increased GSH level.

Figures 7,8,9 and 10 showed the activity of ALT, AST and level of BUN and CRT in different groups of animals. These values are higher till 90 days in arsenic treated animals suggesting same damage of bath liver and kidney which is corroborated with the findings of carles (2014). BA play an important role to reduce ALT, AST, BUN and creatinine level. The increase in liver marker enzyme (AST and ALT) is responsible for the hepatotoxicity in arsenicosis which was improved by BA administration.

Section of liver of arsenic treated group (A_1) animals showed massive fatty changes at the periphery and of the lobule of the liver (Fig.31).

Degenerative changes was reduced and confined to sub capsular area of the peripheral lobule of the group A_2 animals. In group A_3 animals (Fig.32) degenerative changes in the parenchyma of the liver is not noticed but the reparative changes were marked in the form of proliferation of fibrous tissues around the lobular septa.

The significant increase in urea and creatinine values in arsenicosis in rats suggest renal impairment which may be corrected by BA treatment. It is observed from histopathological findings that section of kidney of the experimental control group (A_1) was damaged by arsenic (Fig.33) which was actually excreted out through the kidney resulting partially damaged Bowmen capsule in the cortex as well as loops of Henley's. The lesions in the kidney of group A_2 and A_3 animals did not reveal any significant changes or curative activity.

The increased level of serum creatinine after sodium arsenite intoxication is due to enhanced formation of metabolic waste product of muscle metabolism.

Further, creatinine is an hydride of creatinine. Muscle contains phospho-creatinine which undergoes spontaneous cyclization with loss of inorganic phosphorous to form creatinine.

Increased level of creatinine was reported by Faires (2004) in arsenic intoxicated cattle. Patel and Kalia (2010) also obtained higher level of serum creatinine in arsenic induced rats. Nandi *et al* (2008) and Rana et al (2008) also suggested that arsenic is a potential nephrotoxic agent – Arsenic acts on renal capillaries, tubules and glomerule to cause several renal damage (Klassen, 2006).

Conversion of phosphor creatinine to creatinine is a non-enzymatic irreversible process. Due to affinity for thiol group of various proteins found in the cell membrane of muscles, arsenic damages the cells due to which the enzyme CPK (creatinine phosphokinase) gets released from the cells which is responsible for the conversion of phosphocreatine in to creatinine. Thus increases the level of creatinine. Toxic effect of arsenic on hepatic parenchymal cells reflucted by elevation of liver enzymes AST and ALT in blood. BA plays an important role to reduce ALT, AST, BUN and creatinine level.

Conclusion :

Bauhinia acuminata stem bark extract has some ameliorative effects in chronic arsenicosis in rats.

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