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# ARSENIC INDUCED CLINICO-HEMATO-PATHOLOGICAL ALTERATIONS IN BROILERS AND ITS ATTENUATION BY VITAMIN E AND SELENIUM

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Present study was carried out to know the arsenic (As) induced toxico-pathological alterations in broiler chicks and their attenuation with vitamin E (Vit E) and selenium (Se). A total of 90 day-old broiler chicks were equally distributed into 5 groups. Groups 1-4 were administered As@50 mg/kg BW daily through feed for 30 days. In addition to $A_s$, groups 2 to 4 received Vitamin E@150 mg/kg BW, selenium@0.25 mg/kg BW and Vitamin E plus selenium, respectively. Group 5 (Control) received normal drinking water for 30 days. Dullness, depression, open mouth breathing, increased thirst; ruffled feathers, pale comb, skin irritation and watery diarrhea were the most striking clinical signs. The body weight and feed intake was significantly decreased in treated birds. The erythrocyte counts, hemoglobin concentration and packed cell volume decreased ($P<0.05$) in treated broilers with As or As with Se and Vit E. Grossly pale and hemorrhagic liver and swollen kidneys were observed in As treated birds. Arsenic treated groups showed significant decrease in serum. Histopathologically, liver exhibited congestion and cytoplasmic vacuolation. In kidneys, condensation of tubular epithelium nuclei, epithelial cell necrosis, increased urinary spaces, sloughing of tubules from basement membrane and cast deposition were observed. In conclusion As induced toxico-pathological alterations and vitamin E and selenium partially ameliorate the toxic effects in broilers chicks.

**Keywords:** Arsenic, broilers, clinico-hematological, pathological

## INTRODUCTION

The livestock sector plays an important role in the economy of Pakistan by contributing more than 40% to the value addition in the agriculture sector and almost 11% to the Pakistani GDP (Abubakar *et al*., 2011). Among livestock sector, poultry industry has its own importance which plays an significant role in generating animal proteins most efficiently and economically within the shortest possible time (Hosseinzadeh *et al*., 2010) and affords good employment sources (Mahmud *et al*., 2011). The production of total poultry birds, including domestic and commercial poultry birds, was 518 million and total meat production remained 601 thousand tons (Ghafoor *et al*., 2010). At the end of egg production, about 27.4 and 8.0 million layer birds and breeders, respectively also contribute more than 46,000 metric tons of poultry meat per annum in Pakistan (Javaid *et al*., 2012). Poultry meat being a high quality animal protein source plays significant role in maintaining the health and nutrition of the people (Shahzad *et al*., 2011). The basic role of poultry production is turning feed stuffs into meat (Hafez, 2011), but this production is hampered by the presence of arsenic in drinking water or the infectious diseases.

Arsenic is a ubiquitous and one of the most potent toxic metalloids in environment. Globally millions of people are being exposed to inorganic arsenic through consumption of contaminated drinking water and food (Silbergeld *et al.*, 2008). Inorganic forms of arsenic are more toxic than organic forms. Arsenic and its compounds are considered as potent carcinogen (Wang *et al*., 2006). Arsenic (As) is a metalloid that occurs in organic and inorganic forms in water and soil throughout the world especially in Bangladesh, India and several other countries of Southeast Asia (Bhatacharya *et al*., 2009). Although it is rare in nature as a pure element however, the organic form exists as arsinobetaine in most of the microbiota, plants and in other biological system. Reduced form of as $A_s$ ($As^{+3}$and $As^{+5}$) are frequently present in industrial products, agricultural wastes and in surface water.

Natural sources of As include arsenate, sulfide, arsenite, arsenide, oxides, silicates, assinopyrites which result in ground water pollution. Iron arsenate is also found near mineral mines including coal mines and oil ore extraction. Many industries such as leather, textile, oil refineries, treated lumber, metal extraction and purification, fertilizer production, insecticides, herbicides and fossil fuel release considerable amount of arsenic into environment (Jomova *et al*., 2011). Elevated levels of $A_s$ have been reported in groundwater in several countries like United States (Korte *et al.*, 1991), Thailand (Williams *et al.*, 1996), Mexico (Hernández-Zavala *et al*., 1998) and China (Liu *et al*., 2000) where millions of people are currently exposed to $A_s$ compounds than the permissible (<10 µg/L) levels (Anonymous, 2001). Ground water contamination with $A_s$ is

the highest in developing countries particularly in West Bengal, India and central and southern parts of Pakistan (Islam *et al*., 2009). Southern area mainly consisted of parts of Sindh province like Hyderabad, Gambit, Thari Mirwah, Manchar Lake, Khairpur, Jamshoro, etc. In these parts, the As levels in drinking water have been reported to be 3-30 fold higher than the permissible level (Wadhwa *et al*., 2011; Baig *et al*., 2011; Baig *et al*., 2012).

High residual concentration of As has been found in the tissues of many marine organisms, wild and sea birds (Fairbrother *et al.,* 1994). Arsenite reacts with the cells and cause free radical injury. Persistent exposure either due to geochemical enrichment or due to industrial process induces severe biochemical, toxicological and pathological changes (Liu *et al.,* 2000). Few reports are available about the toxicological effects of As in broilers (Garbarino *et al.,* 2003) and livestock (Monies, 1999).

Vitamin E (α-tocopherol) is considered as chain-breaking micronutrient antioxidant. Owing to antioxidant properties, vitamin E and selenium have major impact on immunity and enhance intracellular generation of super oxide dismutase and glutathione, thus reduce free radical induced lethal injury (Selvaraj *et al*., 2012). In the published literature, reports about role of vitamin E and selenium in detoxifying $A_s$ toxicity in broilers are scanty; therefore, the present study was designed and executed to know the role of vitamin E and selenium in detoxifying detrimental belongings of arsenic in broiler birds.

## MATERIAL AND METHODS

***Experimental birds and management*:** This study was carried out on a total of 90 day-old broiler chicks of mixed gender procured from local hatchery. After 2 days of acclimatization, chicks were randomly divided into five equal groups (1-5). Basel feed with 16% protein and clean water was offered *ad libitum*. Chicks were vaccinated against Newcastle Disease (ND) on day $2^{nd}$ and $23^{rd}$, infectious bursal disease (IBD) on $8^{th}$ and $21^{st}$ and hydropericardium syndrome (HPS) on day $19^{th}$ by using live vaccines. The treatments mixed in feed in this experiment took place at 3 days age and continual till 33 days age. In this experiment, 1-4 groups received As@ (50 mg $kg^{-1}$ BW) daily for 30 days. Groups 2-4 also received Vit E (400 IU/g; dl-alpha-tocopheryl acetate); M/S Alpharma Inc. 440 Route 22, Bridgewater, New Jersey 08807@ (150 mg $kg^{-1}$ BW), Se @ (0.25 mg $kg^{-1}$ BW) and Vit E + Se (150 + 0.25 mg $kg^{-1}$ BW), respectively. Respective treatment was administered for 30 days with skip a day strategy. Control group was 5 which received no treatment except normal feed and water. Monitoring of the birds were carried out two times daily to note clinical signs which were then categorized on the basis of severity (Ehtisham *et al*., 2011) into very mild (+), mild (++), moderate (+++) and severe (++++). On days 0, 15 and 30 of experiment, feed intake and body weight of all broiler chicks was recorded and analyzed.

***Hematological parameters*:** From each group, 6 birds were selected randomly and killed humanely on experimental days 0, 15 and 30 and blood with $Na_2$ EDTA; 1mg $ml^{-1}$ was collected for RBC and TLC determination (Jalees *et al*., 2011). Hematocrit was dogged by microhematocrit capillary tubes and Hb. concentration by spectrophotometrically. Erythrocyte indices i.e., MCHC and MCV were calculated (Njidda and Isidahomen, 2011). All blood smears were subjected to determination of DLC by staining slides with Wright-Giemsa following the method described by Benjamin (1978).

***Gross and histopathological studies*:** Collected morbid tissues were preserved in 10% buffered formalin. About 4-5µm thick sections were cut and processed for histopathological examination using the standard method of dehydration in ascending grades of ethanol, clearing in xylene and stained with hematoxylin and eosin (Hassan *et al*., 2012).

***Statistical analysis*:** The data collected from the present experiment were analyzed statistically by applying 2-Factor factorial keeping two factors, i.e. group and time as factors. The means were compared using analysis of variance technique and Duncan multiple range (DMR) test. Significance level was ($P<0.05$).

## RESULTS AND DISCUSSION

Arsenic exposure is one of the major and most important environmental health risks in many regions of the world. Non occupational exposures to inorganic arsenic compounds are associated with contaminated drinking water from ground-water (Silbergeld *et al.,* 2008). Therapeutic potential of organic arsenical and arsenic trioxide has been reported in treating syphilis, coccidiosis in poultry and for treatment of promyelocytic leukemia and multiple myeloma (Sasaki *et al*., 2007).

***Physical Parameters*:** Various clinical signs (Table 1) including depression, dullness, emaciation, open mouth breathing; ruffled feathers, pale comb and hyper-excitability were observed in all treated groups. However the severity of these clinical signs varied in As treated birds. These clinical signs were partially ameliorated with Vit E and Se. The feed intake and body weight was significantly decreased in treated birds (Table 1). Hyper-excitability in present study may be due to significant inhibition of superoxide dismutase, catalase, glutathione-S-transferase which causes the lipid peroxidation of neuronal plasma membrane with the generation of lethal reactive oxygen and reactive nitrogenous species (Das *et al*., 2010). Similar nervous excitability symptoms have been reported in mice (Wu *et al*., 2008); however, Sajan *et al*. (2009) reported no nervous excitability

**Table 1. Clinical signs displayed by As treated broiler chicks only or with Se and Vit E in contrast to control birds**

| Clinical signs | Groups* | | | |
|---|---|---|---|---|
| | 1 | 2 | 3 | 4 |
| Depression | (++++) | (++) | (+++) | (+) |
| Dullness | (++++) | (++) | (++) | (++) |
| Emaciation | (++++) | (++) | (++) | (++) |
| Open mouth breathing | (++++) | (++) | (+++) | (+) |
| Ruffled feathers | (++++) | (++) | (+++) | (+) |
| Pale comb | (++++) | (++) | (+++) | (++) |
| Hyper excitability | (++++) | (+++) | (++) | (+) |
| Decreased body weight | (++++) | (+++) | (+++) | (+) |
| Decreased feed intake | (++++) | (++) | (+++) | (+) |

*Groups 1-4 received As@ (50 mg $kg^{-1}$ BW) daily for 30 days. Groups 2-4 also received Vit E (400 IU/g or 150 mg $kg^{-1}$ BW dl-alpha-tocopheryl acetate), Selenium @ (0.25 mg $kg^{-1}$ BW) and Vit E + Se (150 + 0.25 mg $kg^{-1}$ BW), respectively. Group 5 served as control and no signs were observed.

in cockerels after administration of As at dose of 50 and 100 ppm in feed.

In the present study, less feed intake and decrease body weight was recorded following administration of As in present experiment (Table 2). Similar reduction in body weight gain and feed intake have also been recorded after oral administration of different levels of As in broiler chicks (Vodela *et al.,* 1997), dullness and depression, rough body coat with erected hairs, profound muscular weakness, and in coordination in goats at the dose rate of 75, 100, 125, 150 mg/kg sodium arsenate goat (Halder *et al*., 2007). However, prominent gastrointestinal symptoms were observed during initial toxic phase of As in human (Xu *et al*., 2008). Similarly, Selby *et al.* (1977) reported that symptoms of As poisoning in animals manifested by intense abdominal pain, staggering gait, extreme weakness, trembling, salivation, vomiting (in dogs, cats, pigs, and perhaps even cattle), diarrhea, fast, feeble pulse, prostration, rumen atony, normal to subnormal temperature, collapse, and death. In subacute As poisoning, animals may live for several days, exhibiting depression, anorexia, watery diarrhea, increased urination at first followed by anuria, dehydration, thirst, partial paralysis of the rear limbs, trembling, stupor, cold extremities, subnormal temperature and death. The watery diarrhea may contain shreds of intestinal mucosa and blood. Convulsive seizures have been reported but are not an expected manifestation.

***Hematological parameters*:** The total erythrocyte counts, hemoglobin concentration and packed cell volume decreased ($P<0.05$) in As treated broiler chicks only or with Se and Vit E in contrast to control birds (Table 2). MCHC and MCV also decreased significantly in As treated broiler chicks only or with Se and Vit E (Table 3) than birds in control. Increased ESR in As treated broiler chicks only or with Se and Vit E in contrast to control birds. Leukopenia was observed at days 15 and 30 of the experiment in all treated groups as compared to the birds of control group. At days 15 and 30 of experiment, total leukocyte counts (TLC), eosinophil, basophil, monocyte and heterophil significantly ($P<0.05$) decreased in As treated broiler chicks only or with Se and Vit E in contrast to control birds. The lymphocyte population was significantly increased in all treated chicks throughout the experiment (Table 4). The reduction in hematological parameters in present study could be due to the mylotoxic effects of As to blood forming tissues (Meisner *et al*., 1992). Rubina *et al*. (2008) reported

**Table 2. Live body weight (g) of broiler chicks in different groups administered arsenic, vitamin E and selenium in different combinations**

| Groups | Age (days) | | |
|---|---|---|---|
| | **0** | **15** | **30** |
| G1 (As) | 43.0±5.7 | 292.7±49.5b | 827.6±59.9b |
| G2 (As + Vit. E) | 45.3±4.6 | 449.2±49.5a | 1172.7±85.2a |
| G3 (As + Se) | 41.5±5.4 | 428.5±79.1a | 869.3±60.3b |
| G4 (As + Vit. E + Se) | 43.0±6.1 | 467.6±132.0a | 846.7±70.5b |
| G5 (Control) | 47.2±3.0 | 457.6±60.1a | 1225.8±111.6a |

Values bearing different alphabets in a row differ significantly ($P<0.05$). Groups 1-4 received As@ (50 mg $kg^{-1}$ BW) daily for 30 days. Groups 2-4 also received Vit E (400 IU/g or 150 mg $kg^{-1}$ BW dl-alpha-tocopheryl acetate), Selenium @ (0.25 mg $kg^{-1}$ BW) and Vit E + Se (150 + 0.25 mg $kg^{-1}$ BW), respectively. Group 5 served as control and no signs were observed.

**Table 3. Erythrocyte indices in arsenic treated broiler chicks only or with vitamin E and selenium in contrast to control birds**

| Parameters/Days | Groups | | | | |
|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 |
| **Total erythrocyte count (× $10^6$/µl)** | | | | | |
| 0 | 3.63±0.39 | 3.71±0.41 | 3.41±0.39 | 3.48±0.36 | 3.83±0.36 |
| 15 | 2.89±0.56b | 3.95±0.52a | 2.89±0.69b | 3.28±0.56ab | 3.82±0.36a |
| 30 | 3.02±0.56b | 3.95±0.51a | 2.86±0.68b | 3.28±0.55ab | 3.85±0.36a |
| **Hemoglobin concentration (g/dl)** | | | | | |
| 0 | 9.79±1.60 | 9.73±1.37 | 10.82±1.31 | 10.34±1.72 | 10.23±1.66 |
| 15 | 9.42±0.83a | 7.25±0.26b | 8.04±0.57b | 7.54±0.31b | 10.23±1.66a |
| 30 | 3.88±1.67b | 3.86±1.81b | 2.50±0.614b | 4.30±0.78b | 10.23±1.66a |
| **Packed cell volume (%)** | | | | | |
| 0 | 129.0±3.9 | 31.0±7.1 | 30.83±5.6 | 31.17±8.0 | 31.83±6.7 |
| 15 | 24.33±3.01c | 31.67±4.76ab | 27.50±4.09bc | 26.83±2.13c | 34.50±4.28a |
| 30 | 17.83±4.17c | 23.0±5.90bc | 20.33±5.68c | 27.83±3.31ab | 31.67±4.97a |
| **Mean corpuscular volume (fl)** | | | | | |
| 0 | 181.38±19.44 | 94.77±18.49 | 92.27±30.58 | 92.14±20.61 | 88.73±22.87 |
| 15 | 71.51±31.374d | 80.53±11.01bc | 99.77±26.82ab | 84.92±20.13c | 100.38±5.57a |
| 30 | 61.37±21.8c | 62.19±16.44c | 70.71±34.52bc | 83.63±20.84ab | 88.89±35.07a |
| **Mean corpuscular hemoglobin concentration (g/dl)** | | | | | |
| 0 | 160.17±20.15 | 59.60±55.9 | 65.00±41.0 | 56.26±32.89 | 64.04±43.87 |
| 15 | 24.54±17.90c | 22.84±49.67d | 59.67±45.8b | 58.78±42.08b | 63.35±56.90a |
| 30 | 26.78±24.98c | 23.89±29.45c | 64.75±43.87a | 60.56±46.67b | 66.56±48.78a |
| **Erythrocyte sedimentation rate (mm/h)** | | | | | |
| 0 | 6.33±1.63 | 6.16 ±1.94 | 6.50 ± 1.87 | 5.83±2.31 | 6.76 ±1.63 |
| 15 | 6.56±1.63b | 6.83 ±1.94b | 6.33 ±2.58b | 7.66 ±2.06b | 12.67 ±3.08a |
| 30 | 6.67±1.63e | 25.66 ±2.16a | 13.33 ±2.58d | 19.33 ±1.96c | 21.83 ±1.94b |

Values bearing different alphabets in a row differ significantly (P<0.05). Groups 1-4 received As@ (50 mg $kg^{-1}$ BW) daily for 30 days. Groups 2-4 also received Vit E (400 IU/g or 150 mg $kg^{-1}$ BW dl-alpha-tocopheryl acetate), Selenium @ (0.25 mg $kg^{-1}$ BW) and Vit E + Se (150 + 0.25 mg $kg^{-1}$ BW), respectively. Group 5 served as control and no signs were observed.

**Table 4. Leukogram indices in arsenic treated broiler chicks only or with vitamin E and selenium in contrast to control birds**

| Parameters/Days | Groups | | | | |
|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 |
| **Total leukocytic count (× $10^3$/µl)** | | | | | |
| 0 | 20.87±5.16 | 21.43±5.16 | 23.04±5.97 | 22.43±4.84 | 24.56±4.98 |
| 15 | 12.13±0.87c | 22.28±2.73a | 13.50±1.28c | 16.92±1.01b | 22.45±2.19a |
| 30 | 11.98±1.12b | 21.05±2.26a | 14.87±2.06b | 14.98±1.74b | 23.98±1.94a |
| **Lymphocytes (%)** | | | | | |
| 0 | 54.33±8.07 | 57.5±8.26 | 57.83±6.31 | 52.16±5.6 | 60.33±4.23 |
| 15 | 85.0±1.9ab | 72.66±16.23c | 88.16±2.32a | 77.33±3.93bc | 59.45±4.21d |
| 30 | 89.0±1.41a | 81.66±5.6bc | 86.16±2.79ab | 78.16±5.084c | 58.3±4.17d |
| **Heterophils(%)** | | | | | |
| 0 | 13.33±5.72b | 15.83±7.91ab | 18.0±4.05ab | 16.33±5.43ab | 20.50±2.74a |
| 15 | 6.83±2.32c | 13.33±6.86b | 5.16±.75c | 12.67±5.32b | 23.5±2.19a |
| 30 | 7.16±1.17cd | 11.66±3.88b | 5.16±0.75d | 9.50±4.68bc | 24.50±2.74a |
| **Monocytes(%)** | | | | | |
| 0 | 20.87±5.16 | 21.43±5.16 | 23.04±5.97 | 22.43±4.84 | 24.56±4.98 |
| 15 | 12.13±0.87c | 22.28±2.73a | 13.50±1.28c | 16.92±1.01b | 22.45±2.19c |
| 30 | 11.98±1.12b | 21.05±2.26a | 14.87±2.06b | 14.98±1.74b | 23.98±1.94a |
| **Eosinophils(%)** | | | | | |
| 0 | 0.50±0.84 | 0.167±0.41 | 0.24±0.97 | 0.16±0.41 | 0.80±0.75 |
| 15 | 5.16±1.17bc | 11.83±7.39a | 4.33±.28bc | 7.16±2.56b | 0.85±0.75c |
| 30 | 0.50±0.84c | 3.83±2.32b | 4.16±0.75b | 6.33±2.88a | 0.823±0.75c |
| **Basiphils(%)** | | | | | |
| 0 | 0.83±1.33 | 0.33±0.82 | 0.66±1.21 | 0.50±1.22 | 1.83±1.17 |
| 15 | 0.16±0.41b | 0.66±0.82ab | 0.16±0.41ab | 1.16±1.01ab | 1.83±2.19a |
| 30 | 0.16±0.41b | 0.33±0.82ab | 0.57±2.55ab | 2.16±2.99a | 1.83±1.17a |

Values bearing different alphabets in a row differ significantly (P<0.05). Groups 1-4 received As@ (50 mg $kg^{-1}$ BW) daily for 30 days. Groups 2-4 also received Vit E (400 IU/g or 150 mg $kg^{-1}$ BW dl-alpha-tocopheryl acetate), Selenium @ (0.25 mg $kg^{-1}$ BW) and Vit E + Se (150 + 0.25 mg $kg^{-1}$ BW) respectively. Group 5 served as control and no signs were observed.

decreased hemoglobin and red blood cells in rats. The erythrocyte sedimentation rate increased invariably in broiler given As. The cause of change in hematological values might be due to the toxic effect on hematopoietic system which is responsible for such alterations in hematological parameters. However, the toxic effects of arsenic on bone marrow may be responsible for erthrocytopenia. Total leukocyte counts, heterophils, monocyte, eosinophil and basophil significantly decreased while lymphocyte population was significantly increased in birds of all treated groups. The alteration in hematological parameters might have occurred due to direct depression of bone marrow and impaired folic acid absorption (Sajan *et al*., 2009). In addition, As also acts as a capillary poisons and increases the fragility of red blood cells (Biswas *et al*., 1998).

***Pathological observations*:** Liver of birds in groups 1-6 (Arsenic 50mg/kg) grossly revealed no significant lesions; however, microscopically liver parenchyma exhibited degenerative and necrotic changes (intracytoplasmic vacuolation) and various morphological changes in nucleus including hyperchromasia, pyknosis, fragmentation and cells without nucleus were observed (Fig. 1). Portal areas showed infiltration of mononuclear cells particularly around the portal vein and there was increased number of bile ducts. In addition to the above changes, increased proliferation of connective tissue has been reported after oral intoxication of As in mice (Wu *et al*., 2008). In Nile cat-fish with the administration of As, Abdel-Hameid (2009) reported chronic and degenerative changes in liver such angiomatiod cysts in hepatic parenchyma (peliosis hepatis) and mononuclear cell infiltration chronic within the sinusoidal spaces and portal area. These necrotic and degenerative changes observed in present study could be due to lipid peroxidation as a result of reactive oxygen species (ROS) generation (Das *et al*., 2010). Mononuclear infiltration in the portal area in association with biliary hyperplasia could be due to production of IL-33, IL-1α and production of intra and extracellular damage associated molecules from the necrotic cells in the liver those are recognized by the macrophages and generated the cytokines for the recruitment and removal of the necrotic cells from the liver.

In present study, kidneys were swollen, congested and were protruded out from there bony sockets in groups 1 to 4. Microscopically, moderate to severe necrosis of tubular epithelial cells characterized by pyknotic nuclei and cytoplasmic vacoulation were observed. Atrophy of glomeruli with massive lobulation and fragmentation of capillary tufts along with mononuclear infiltration and increased urinary spaces were observed in kidneys of birds in groups 1 and 3 (Fig. 2). Most of the tubules were collapsed and lumens were obliterated, however, some tubular lumens showed eosinophilic proteinous material aggregated was observed in As treated birds. According to Nandi *et al.* (2004), kidneys are organ more vulnerable to arsenic toxicity where it induces lethal effects on renal tubular epithelium in dose dependent manner in the cockerels (Sajan *et al*., 2010). Exposure of inorganic arsenic during early age potentially responsible for neoplastic transformation of hepatocytes and kidney cells as a result of continuous production of ROS and reactive nitrogenous species in these tissues where induce damage to the thiamine nucleotide of DNA strand (Tokar *et al*., 2012).

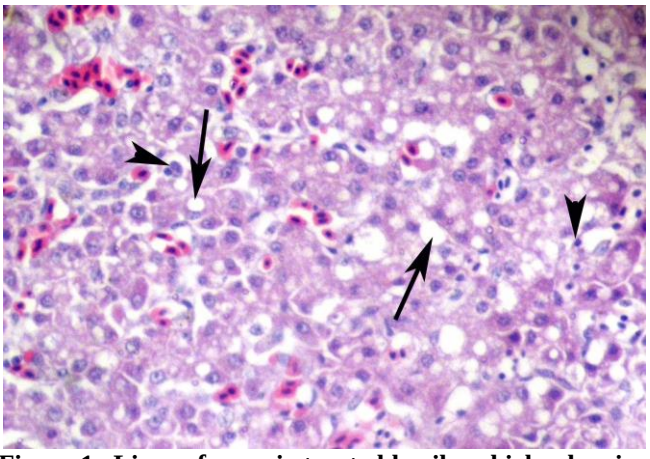

**Figure 1. Liver of arsenic treated broiler chicks showing vacuolation (arrow), congestion and condensed nuclei (arrow head). H & E. 200X.**

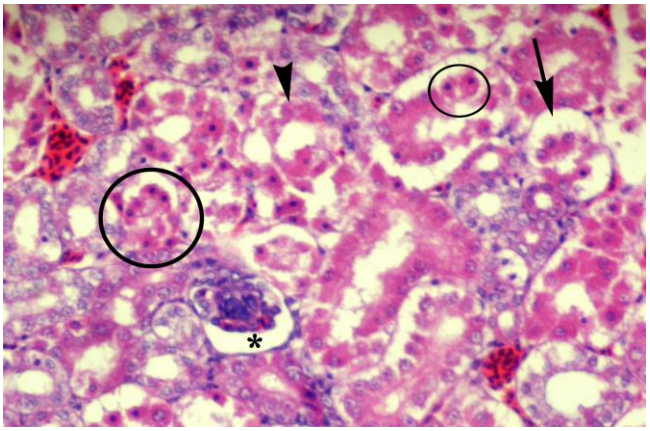


**Figure 2. Kidney of arsenic treated broiler chicks showing sloughing of epithelium from the basement membrane of renal tubule (arrow), necrosis of renal tubule (arrow head), atrophied glomerulous & increased urinary space (*) and hyperchromatic nuclei (circle). H & E. 400X.**

In the present study, addition of vitamin E and Se partially ameliorated the toxic effects of As in the form of reduced severity of clinical signs and pathological lesions and even improved hematological parameters. Vitamin E and Se amelioration could be due to their major impact on immunity

and their antioxidant properties through enhancing the intracellular generation of glutathione and super oxide dismutases that minimize the free radical induced lethal injury (Politis *et al*., 1995; Selvaraj *et al*., 2012). It has also been reported that the free radicals generated by As feeding, attack the double bonds of polyunsaturated fatty acids and thereby initiating a chain reaction which affect membrane integrity and cellular function (Das *et al*., 2012). This chain reaction is inhibited by vitamin E by reacting with free radicals and converting itself into a-tocopheroxyl radical which is not harmful, thus vitamin E checks lipid peroxidation (Buettner, 1993).

***Conclusion*:** On the basis of the results of present experiment it can be concluded that arsenic induces clinico-hematological toxicity in broilers and administration of vitamin E and selenium alone or in combination prove to be good modulators of the toxicity induced by the administration of arsenic.

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