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Review Article

Review on Toxic Effects of Arsenic with relation to Clinical Signs in Different Animals

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Abstract

Arsenic is natural metalloid which is present in the universe everywhere. It is used for different insecticides and pesticides preparation. Poisoning of arsenic is a major issue that affects different species. Its occurrence is related to contamination of feed and water; therefore, it is matter of concern globally. However, its level of toxicity is increasing very rapidly in Asian countries especially Bangladesh, India and Pakistan. Its increasing level of arsenic in ground water is the major source of poisoning to human and animals. In addition, the contact of animals to arsenic lead to absorption body and accumulation in vital organs like liver and kidneys that may resulted to carcinogenic development. Elimination of absorbed arsenic in environment through excretions of animals is also one way to increase its level in water/soil. The objective of this review is to gather the information about arsenic toxicity occurrence by natural or experimentally ways and changes occurred in suffered animals related to pathological or biochemical.

Keywords: arsenic, lab animals, livestock and toxic effects

Introduction

Arsenic is a metalloid element and continuously eliminated in environment. The use of insecticides and pesticides, veterinary drugs and feed additives are the major sources for the exposure of animals (Friberg *et al*., 1986). Contamination of drinking water with arsenic is the principal source of exposure to livestock and human (Bode and Dong, 2002; Yih *et al*., 2002). The animals using the contaminated fodder and water are considered as the principal source of threat to human especially children through the secretion of arsenic in milk (Brahman *et al.*, 2016). The laboratory animals as well as large animals suffer many digestive and nervous signs due to arsenic poisoning. Arsenic commonly accumulates in hair, skin, liver and kidneys. The major clinical signs of toxicity in livestock are manifested in the form of diarrhea, in coordination and death of animals (Neiger *et al*., 2004).

Exposure of arsenic causes the cancer of bladder, lungs, skin, developmental abnormalities in reproductive and pancreas cells (Benbrahim-Tallaa and Waalkes, 2008; NRC, 1999). In acute cases, abdominal cramping, in coordination in extremities, electrocardiogram may develop in animals (Franzblau and Lilis, 1989). The chronic exposure of arsenic results in rise of liver enzymes in cattle (Rana *et al*., 2010).

No report is available about the complete toxicity of arsenic with relation to clinical signs. The objective of this review was to review the sources of arsenic, toxicity mechanism and clinical manifestations with special focus on livestock species. The efforts were also made about the available drugs for the treatment of arsenic toxicity.

Sources of Arsenic

The high arsenic concentration can be attributed to contamination of water from industrial activities and

pesticides, in addition to other anthropogenic sources (Nickson *et al.,* 2005). The contamination of fodder and water are considered as the common source of arsenic poisoning to livestock now a days. The frequent use of arsenic in dipping fluid to kill the ectoparasite and use as insecticides and pesticides are the major sources of arsenic contact to livestock. The use of contaminated water as well as the arsenic in natural source is also considered as the chronic source of arsenic accumulation in tissues and secretions of animals. In acute cases of arsenic toxicity, the clinical signs are manifested after 30 minutes. However, these signs can be delayed if the arsenic is given with food. The early clinical signs are present in acute cases are present in the form of muscular spasm, general weakness and flushing of skin (Franzblau and Lilis, 1989).

Mechanism of arsenic toxicity

After entry in body in acute cases, the first toxic signs are evidenced by severe vomiting, diarrhea, cramps in muscles and injuries in blood vessels **(**Rahman *et al.,* 2001). During the chronic cases the site of actions are kidney, liver and skin. The signs are manifested in the form of hyperkeratosis of skin and damages in kidney and liver (Wu *et al.,* 1989; ATSDR, 2000). These two organs are known as the first site of action for toxico pathologic manifestations as well as the changes in cellular morphology and functional activities (Nandi *et al.,* 2005). The absorption of ingested arsenic takes place in gastrointestinal tract and detoxification takes place in liver. The biotransformation of this metal takes place in liver through the oxidation of methyl (Vahter, 2002). This absorbed arsenite is transferred with in liver for metabolization and excretion in urine (Vahter, 2002).

Patho-clinical Signs due to Arsenic

These signs vary from species to species. The prognosis of acute arsenic toxicities is limited. Pronouncing sign of arsenic toxicities is death 6–12. The diagnosis of cattle is usually made on postmortem lesions.Arsenic causes the goiter in rats and malfunction of thyroid gland in man. The hronic exposure of this metal causes the cancerous growth in skin in the form of ofMulticentric basal cell and squamous cell carcinoma (Pershagen*et al.,* 1983).

The cattle suffering from arsenic toxicity results in ataxia with intense muscle fasciculation succeeding to recumbencewith bloody diarrhea Valentine *et al.* (2007). The clinical signs were appeared after 12 hour of arsenic ingestion to cattle in this case. The toxicity of arsenic in mice and rats results in diarrhea and difficult respiratory distress (Stevens *et al*., 1979). The clinical signs are manifested due to direct inflammation of gastrointestinal tract and by damaging the organs with the reduction of ATP requirements. The signs of toxicity in naturally and arsenic induced are summarized in table 1.

Table 1: Signs of Arsenic Toxicity in various animal species

Species	Dose	Signs	Reference
Cattle	Not reported	The toxic signs were present in the form of severe diarrhea and enteritis with hemorrhages. There was loss of appetite, muscular weakness, ataxia, general weakness and recurrent convulsion.	(Riviere *et al.*, 1981)
Goats	Sodium arsenite was fed with dose of 75 and 100 mg/kg body weight for six weeks.	Toxicity was present in gastrointestinal and kidney after 12 hours of feeding sodium arsenite. The general appearance of animal was dull and depressed, with reddish color of urine. The postural defects appear in the form of loss ability to rise and postural defects like (knee-based posture, Fig. 1). There was increase in respiratory and heart rate in this case.	(Biswas *et al*., 2000).
Goats	(NaAsO2 4 mg/kg body wight daily orally	Body weight was reduced	(Akter *et al.*, 2010)
Cattle	Arsenic pentoxide, 780 mg/kg ash	Toxicity appeared in the form of sudden death. The concentration of arsenic in liver, kidney and rumen was 13.9, 23.7 and 25.8 mg As /kg respectively.	(Thatcher *et al*., 1985)
Pigs	Arsenic was 100 mg arsanilic acid/kilogram of feed for time of 6 weeks.	Feed intake was decreased.	(Morrison and Chavez, 1983)
Pigs	1 gram of arsanilic acid/kilogram of diet.	The toxic signs were appeared in the form of loss of appétit.	(Hapke, 1988).
Hen	15 mg arsenic oxide/kg	Egg mass was reduced	(Holeman *et al*., 2001).
Chickens	As (50 mg/kg BW)	Decrease in body weight and feed intake. The other signs were general dull in behavior, breathing with open mouth and thirst was increased.	(Sharaf *et al.*, 2013)
Quails	30 mg sodium arsenite/kg of feed	No sign reported	(El Begearmi *et al*., 1982).
Rabbits	Sodium arsenite with dose of 0, 0.19, 0.75 or 3.0 mg/kg body weight /day for 6-18 days of gestation and sacrificed on day 29.	There was loss in weight gain, diarrhea and terminal convulsions with increasing the dose of arsenic.	(Nemec *et al*., 1998)
Dogs	As(III) with1.2 mg/kg body weight /day and As(V) 3 mg/kg body weight/day	Mortality was present without any sign.	(Byron *et al*., 1967)
Rainbow trout	arsenic trioxide with dose of 180 mg /kg feed and disodium arsenate heptahydrate137 mg /kg of feed for 56 days	Feed intake and body growth was reduced.	(Cockell and Hilton, 1988).
Buffalo	Buffalo 50 mg/L	The toxicity was present in the form of loss of body weight. The other external signs were general weakness, dehydrated, anorexic and diarrhea with blood. There was also generalized body signs were; anemia, hemorrhages and congestion was present in intestine, liver and kidneys.	(Rana *et al.*,2008)
Cattle	Contamination of Environment	Sudden death, diarrhea, ataxia, dehydration and respiratory distress and marked hemolysis were developed due to arsenic. These abnormalities lead to severe icterus as shown in (Fig 3)	(Bertin *et al.*, 2013)
Sheep	Drinking of contaminated water with (ug/L) 310 level of arsenic	The average value of body weight, body temperature, heart rate, and respiration rate in As-exposed vs control sheep were (30.70 kg) vs (48.30 kg), (39.22C) vs (38.90C) and (77.60/min vs 37.46/min), The skin was severely in the form of hyperkeratonised and pustule formation (Fig. 2).	(Keshavarzi *et al.*, 2015)

It is evident from Table 1 that different forms of arsenic manifest variety of clinical signs in different animals. The main sites for the changes of arsenic are skin, liver and kidneys.

Fig: 1 Case of hyperkeratosis and pustule formation in the skin of sheep due to arsenic (Keshavarzi*et al.*, 2015)

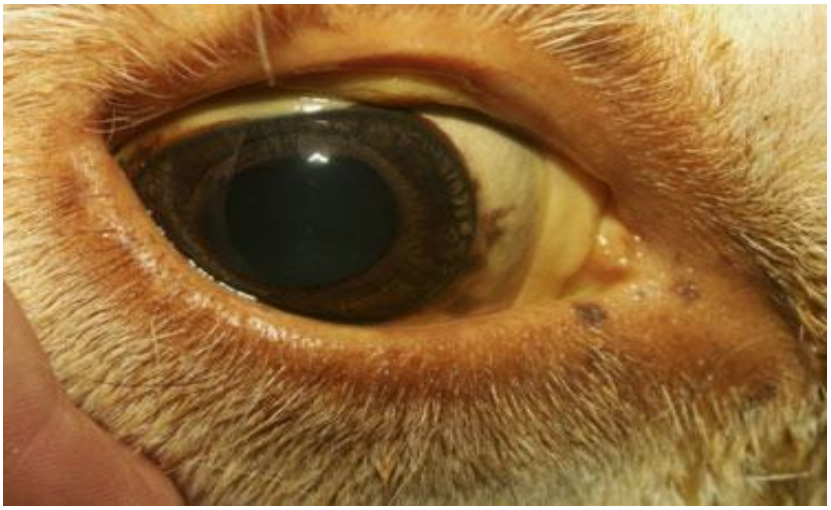

Fig: 2 A case of severe icterus in cattle due to arsenic toxicity (Bertin *et al.*, 2013)

Treatment

The administration of sodium thiosulfate with dose of 40 mg/ kg intravenous (IV)for 8 hour has the useful effects in removing the arsenic from the body and maintenance of renal insufficiency (Bertin *et al.*, 2013). However, the antioxidants like vitamin E can also be used to ameliorate its toxic effects (Momeni *et al.*, 2012).

Conclusion

This review is source of awareness to progressive livestock farmers about the clinical signs of arsenic toxicity and its management. Although, no such case is reported in Pakistan, but these reports will helpful in future for the control of toxicity. The environmental pollution is continuously increasing in the Pakistan. The source of such cases could not be overlooked in future.

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