

## Mass Cyanide Intoxication in Sheep

MR Aslani, M Mohri  
Department of Clinical Sciences

M Maleki  
Department of Pathobiology

K Sharifi, GR Mohammadi  
Department of Clinical Sciences, School of Veterinary Medicine,  
Ferdowsi University of Mashhad, PO Box 91775-1793, Mashhad, Iran

M Chamsaz  
Department of Chemistry, Faculty of Science,  
Ferdowsi University of Mashhad, PO Box 91775-1436, Mashhad, Iran

**ABSTRACT.** An outbreak of cyanide poisoning that killed 56 ewes and 2 goats is reported. Fluid released into a ditch contained 1 g cyanide/l and produced toxicity in 3 ewes experimentally dosed with the liquid waste.

Cyanide is a potent and ubiquitous toxicant; many naturally occurring substances and industrial products contain cyanide (1). The most important source of cyanide to animals is plant material. Over 1000 species of higher plants contain hydrocyanic acid either free or, more common, in the form of cyanogenic glycoside, an organic compound containing a sugar and capable of yielding cyanide on hydrolysis (2, 3). Hydrogen cyanide and cyanide salts are used in a wide variety of common industrial processes: electroplating techniques, photographic processes, newspaper printing, metal cleaning and finishing, gold and silver mining, and plastic processing and tanning. Hydrogen cyanide gas is used as a fumigant rodenticide. Combustion of many plastic compounds produces hydrogen cyanide gas and as a result, many smoke inhalation victims suffer from cyanide toxicity (4, 5).

### CASE REPORT

In November 2002, a flock of 350 ewes of Kuordish breed and 25 goats of local breed were exposed to a yellow-green watery waste, which had been released beside a not frequently visited road near Mashhad. The fluid was drunk by some of the animals and a few minutes later they became prostrate and had severe convulsive movements. Fifty-six ewes and 2 goats died or were slaughtered in a short period of time after exposure to the toxic material. Two ewes survived after transient ataxia and dyspnea. On clinical examination these animals had tachycardia and tachypnea. The urine of these ewes was acidic when tested by urine strip (Combi-Screen, Germany).

Postmortem examination revealed congestion of abomasum and congestion and various degrees of hemorrhagic fossa in kidneys and epicardium. Tissue samples from different organs were collected and fixed in 10% buffered formalin, embedded in paraffin, and sectioned and stained with hematoxylin and eosin. There was congestion and various degrees of hemorrhage in the various organs.

A sample of the suspected source of intoxication, the waste fluid, was taken for evaluation for toxic compound(s). It had a

special bitter almond smell, and cyanide was suspected as cause of intoxication. The sample was highly positive in the potassium isopropionate test and potentiometry assay determined 1 g/L of cyanide.

The toxic fluid was used for experimental induction of poisoning by administering it orally to 3 ewes 5, 8 or 10 ml/kg body weight. The ewes that received 5 or 8 ml/kg body weight of the fluid showed transient incoordination, dyspnea, restlessness, weakness, staggering and depression. Their mucous membranes were bright red in color. Venous blood taken from these ewes for gas analysis (ABL 50 Radiometer, Denmark) revealed marked elevations of Po<sub>2</sub>. The 3<sup>rd</sup> ewe, which received 10 ml of the toxic fluid /kg body weight, became prostrate 2 min after dosing and had convulsions, opisthotonos, paddling, and gasping. Four minutes after dosing, respirations ceased but the heart had normal rhythm for 10 min later. Postmortem findings in this ewe were the same as in the earlier cases.

### DISCUSSION

Cyanide poisoning occurs in most countries because of the common occurrence of plant containing toxic quantities of cyanogenic glycosides. Most affected animals die and, although the overall economic effects are not great, the losses may be heavy on individual farms (6) as in this case. Intoxication with cyanide of industrial origin is not common in farm animals. Carelessness in waste product management containing cyanide compounds leads to environmental pollution and is hazardous for all life. When such pollutants flow to rivers they cause rapid and large-scale mortality of aquatic organisms and animals living close to the poisoned rivers, establishing an ecological disaster (7, 8).

Cyanide is well known for its acute impact. It is rapidly absorbed from the gastrointestinal tract and produces acute anoxia, especially of the central nervous system by inhibition of cytochrome oxidase of the tissue respiratory chain (2, 3). Cessation of tissue respiration causes elevation of venous blood oxygen, which manifests clinically by a bright red color of the

visible mucous membranes and blood. High levels of venous blood  $P_{O_2}$  may be helpful for a tentative diagnosis of cyanide poisoning. Although many antidotes are available and efficient against cyanide intoxication (6, 9), the peracute nature of toxicity and very short disease course often makes treatment of cyanide intoxicated animals not possible.

Cyanide does not bioaccumulate, but environmental spills of industrial material containing cyanide are very dangerous for all living organisms. Such contaminated industrial materials must be discarded or destroyed in a secure manner to avoid any environmental pollution.

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