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Scientific Report

Nitrate intoxication due to ingestion of pigweed red-root (*Amaranthus retroflexus*) in cattle

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Summary

Nitrate toxicosis associated with consumption of pigweed red-root (*Amaranthus retroflexus*) was diagnosed in a small herd of cattle in the suburb of Mashhad, northeastern Iran. The clinical signs were ruminal tympany, incoordination, tachycardia, dyspnea, head pressing, aggressive movements and jugular distension. Seven animals including 3 young calves and 4 cows died of intoxication; other affected cattle were treated by intravenous administration of epinephrine and ascorbic acid. No further mortality was observed after the treatment. One of the treated cows aborted a 6-month-old fetus more than three days after intoxication. Two samples of suspected plants were analysed for nitrate. The nitrate content of those plants was 6.6% and 10.4% in dry matter.

Key words: *Amaranthus retroflexus*, Nitrate intoxication, Cattle

Introduction

Among animals, ruminants are especially vulnerable to nitrate intoxication because of the nitrate-reducing potential of rumen microorganisms. The reduction of nitrate (NO_3^-) to more toxic nitrite (NO_2^-) is an intermediate step in the bacterially biochemical sequence of the formation of fully reduced ammonia (NH_3) which is used to form microbial protein. Since the amount of ammonia produced is rate limiting, toxicosis by ammonia dose not occur (Casteel, 1997; Valli, 1998). If ruminants however, rapidly ingest food containing high levels of nitrate, nitrite will accumulate in the rumen. Then, the nitrite ions are absorbed into the blood. Nitrite ions easily enter erythrocytes in exchange for chloride ions. Nitrite oxidizes haemoglobin to form methaemoglobin which is incapable of carrying oxygen to tissues and results in hypoxia. Signs of hypoxia develop when 30–40% of haemoglobin is converted to methaemoglobin and death occurs when there is more than 80% methaemoglobin (Bahri *et al*., 1997; Casteel and Evans, 2004).

The most common source of nitrate toxicosis is plants containing accumulated toxic levels of nitrates. The plants which are most frequently incriminated include cereal crops—either green or as hay—the leaves of sugar beets and turnips and other Brassicacea, *Sorghum* spp., and a number of weeds particularly variegated thistle (*Silybum marianum*), pigweed red-root (*Amaranthus retroflexus*), jimson weed (*Datura stramonium*), and lamb's-quarter (*Chenopodium album*). The greatest potential for toxic levels of nitrates exist in unripe plants and plant that is regrown after harvest. Factors related to plants and animals have important roles in the development of toxicosis and its outcome (Bahri *et al*., 1997; Radostits *et al*., 2000).

Case report

In early June, 2005, a small herd of

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cattle comprising 26 cattle of different ages in Mashhad suburb, northeastern Iran, were fed by pigweed red-root herb. The herb was cut from a farm cultured sorghum but whatever has grown was pigweed red-root with rare sorghum plant (Fig. 1). Urea and potassium nitrate soil fertilizers had been used during the herb growth in the field. About four hrs after plant consumption, 10 animals showed clinical signs of poisoning

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Fig. 1: *A. retroflexus* leaves and green cluster flowers

Fig. 2: Red roots of *A. retroflexus*

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including ruminal tympany, foam in mouth, weakness, unsteady gait and incoordination, head pressing, aggressive movements and biting to objects. Three calves (5- to 7-month-old) and four cows died following a short course of disease. Three cases—one 6-month-old calf and two cows—which their intoxication was not peracute, showed jugular distention, ruminal tympany, hypermetria, incoordination, tachycardia, dyspnea and sunken eyes. Their mucous membranes were pale to purple. Presumptive diagnosis of nitrate poisoning was made on the basis of acute nature, clinical signs and history of pigweed red-root ingestion.

Venous blood samples were obtained from the affected calf and a cow. The colour of blood samples was deep chocolate. Two samples of plants—a fresh one from the field and a sample from the hay prepared for animal feeding—were taken for chemical analysis for nitrate content. Sorghum sample was also collected from the farm for nitrate content.

Methylene blue, the antidote of nitrate poisoning, was not available then and three affected animals were therefore treated with intravenous injection of ascorbic acid at a dose of 5 mg/kg body weight as well as 2 ml of norepinephrine. Fifty ml of 10% oxy-tetracycline was also administered intraruminally.

Serum samples were strongly positive for nitrate when tested by urine test strip (Combi-Screen, Germany). Analysis of plant samples by radiopotentiometry revealed 6.6% potassium nitrate in dry mater (DM) of fresh red-root pigweed sample, 10.4% in DM of pigweed red-root hay and 13% in DM in fresh sorghum.

There was no further mortality. In the day after poisoning, three intoxicated animals were completely recovered. On the third day, one cow aborted a 6-month-old fetus.

Discussion

Nitrate poisoning occurs commonly in cattle raised in western countries (Haliburton, 1999). However, in spite of abundance of nitrate accumulator plants (Aslani, 2004), it has rarely been reported in Iran (Gorjidouz *et al*., 2002). Nitrate is found in many plants in varying amounts, but under certain conditions, it can accumulate in plants. During night and prolonged periods of cloudy weather, nitrate tends to accumulate in the plants (Haliburton, 1999). On the other hand, application of excessive nitrogen containing fertilizers leads to accumulation of nitrate in forages. In the present report, repeated use of such fertilizers has been resulted in high levels of nitrate in the plants. Nitrate levels higher than 15,000 ppm in DM in forages are toxic for cattle and sheep (Bahri *et al*., 1997; Radostits *et al*., 2000). Outbreaks of nitrate poisoning are usually recorded in forage which contains 3–7% in DM of potassium nitrate (Valli, 1998). The toxicity of nitrate in ruminants depends on several factors including the rate of nitrate consumption, species, the amount of carbohydrate in the diet and the stage of adaptation to nitrate. The most important factor influencing susceptibility however, appears to be the rate of ingestion of the nitrate-containing plants. Cattle on high-energy ration can also tolerate more nitrates (Bahri *et al*., 1997; Radostits *et al*., 2000).

Among different species of nitrate accumulator plants, *A. retroflexus* is quite common as a weedy herb and found in and around farmyards and in cultivated and waste areas (Aslani, 2004). The plant is annual, erect with simple, egg-shaped, wavy-marginated, alternate leaves. The red lower stem, branches and taproot give rise to the plant's common name (Fig. 2). Flowers of the plant are borne in short, compact clusters along with green spines (Fig. 1).

In addition to nitrate toxicosis, a nephrotoxicosis with unknown etiology has been associated with consumption of *A. retroflexus* in cattle, pigs and sheep (Casteel *et al*., 1994; Rae and Binnington, 1995; Torres *et al*., 1997).

Clinical signs of nitrate poisoning reflect tissue oxygen deprivation and become apparent when 30–40% of haemoglobin is converted to methaemoglobin. (Bahri *et al*., 1997; Casteel and Evans, 2004). Clinical signs such as head pressing, aggressive behaviour and hypermetria which are due to central nervous system (CNS) dysfunction and mentioned in the present report may also

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be related to the severe CNS hypoxia. Abortion in ruminants due to the fetal hypoxia is a sequela of nitrate intoxication and as it was observed, it usually occurs three to seven days after exposure of dams to the toxic levels of nitrate (Bahri *et al*., 1997; Casteel and Evans, 2004).

Diagnosis of nitrate intoxication is based on observed clinical signs, chocolate-brown blood and exposure to the toxic plant. The diagnosis can also be confirmed by the analysis of suspected plant(s) or body fluids including serum and urine in live animals and aqueous humour or cerebrospinal fluid in dead animals (Bahri *et al*., 1997; Casteel and Evans, 2004).

Methylene blue is the antidote of choice for nitrate intoxication, particularly in ruminants. An alternative antidote is ascorbic acid, but its action is slow. Intravenous administration of a vaso-constrictor such as epinephrine is also very effective in treating hypotention even in cases that are at the point of death (Bahri *et al*., 1997). Intraruminal administration of antibiotics also suppresses the reduction of nitrate to nitrite.

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