Trop Anim Health Prod (2011) 43:1065–1068
DOI 10.1007/s11250-011-9818-0

BRIEF COMMUNICATION

# Acute oxalate intoxication associated to ingestion of eshnan (*Seidlitzia rosmarinus*) in sheep

**Mohammad Reza Aslani · Ahmad Reza Movassaghi · Vahid Najarnezhad · Hossein Janati Pirouz · Mohammad Heidarpour Bami**

Accepted: 13 March 2011 / Published online: 5 April 2011


**Abstract** An outbreak of acute oxalate intoxication in a sheep flock was associated to *Seidlitzia rosmarinus* (Chenopodiaceae) with a mortality rate of about 19%. Affected sheep showed marked azotemia and hypocalcemia. Post-mortem findings included congestion and hemorrhage in visceral organs, ruminitis frequently associated with precipitation of birefringent calcium oxalate crystals, and acute nephrosis with numerous birefringent calcium oxalate crystals in renal tubules. This is the first report of oxalate poisoning due to ingestion of *S. rosmarinus* in sheep.

**Keywords** Oxalate poisoning · Sheep · *Seidlitzia rosmarinus* · Acute nephrosis

## Introduction

Although oxalate intoxication in sheep has been associated with a variety of plant species, field reports of acute oxalate toxicity have been rare in recent years. In fact, cases of acute oxalate poisoning involve hungry and non-adapted stock grazing non-grass species such as chenopods with high concentrations of soluble oxalate (Radositits et al. 2007). Species of grasses in the genus of *Cenchrus*, *Panicum*, and *Setaria* which are widely cultivated in tropical and subtropical areas can also accumulate toxic amounts of oxalate (Maxie and Newman 2007). The most important plants involved in oxalate intoxication of ruminants include halogeton (*Halogeton glomeratus*), soursob (*Oxalis* spp.), rhubarb (*Rheum rhaponticum*), curly dock (*Rumex crispus*), purslane (*Portulaca oleracea*), lamb's quarter (*Chenopodium album*), bassia (*Bassia hyssopifolia*), greasewood (*Sarcobatus vermiculatous*), pigweed (*Amaranthus* spp.), Russian thistle (*Salsola kalis*), and sugar beets (*Beta vulgaris*) (Pickrell and Oehme 2004; Maxie and Newman 2007; Sargison and Angus 2007; Angus 2008).

In this report, we describe the clinical, laboratory, and post-mortem findings of an outbreak of acute oxalate poisoning associated with eshnan (*Seidlitzia rosmarinus*) in a flock of sheep from Iran.

## Case descriptions

In late January of 2010, a flock of 900 local-bred sheep, 500 pregnant ewes and 400 yearlings which had transported by trucks from Torbate Heidarieh in north-east to Boshrouvieh in central east of Iran (300 km distance), moved to a new winter pasture, prominently covered by eshnan. It was raining for 2 days and the plants were in bud stage. During the first night, the flock was returned to a fenced lot to avoid attack by predators. Signs of intoxication and mortality commenced from 8 pm and progressively increased, resulting in the death of 170 ewes within 24 h (mortality rate of 19%). Most of the affected ewes were in

M. R. Aslani (✉) · M. H. Bami
Department of Clinical Sciences and Excellence Center for Ruminant Abortion and Neonatal Mortality,
School of Veterinary Medicine, Ferdowsi University of Mashhad,
Mashhad, Iran
e-mail: mraslani@um.ac.ir

A. R. Movassaghi
Department of Pathobiology, School of Veterinary Medicine,
Ferdowsi University of Mashhad,
Mashhad, Iran

V. Najarnezhad · H. J. Pirouz
Veterinary Organization,
Mashhad, Iran

late pregnancy and 20% were pregnant with twins. Affected sheep were treated by a private veterinarian using atropine sulfate and laxatives without any noticeable effects.

The affected animals displayed signs of depression and weakness and were generally found in sternal or lateral recumbency and unable to rise even with assistance. Some of the recumbent ewes showed an S-form deviation of the neck and others turned their heads into their flanks (Fig. 1). Body temperature was within the normal range and respiration was dyspneic with excessive sero-mucus dripping from external nares. Some animals died suddenly without observable clinical signs.

Blood samples were collected from four intoxicated sheep for hematology and serum analysis. Results of hematology showed mild elevation of packed cell volume (0.37–0.38 L/L) and neutrophils ($6.18–8.13\times10^9$/L), plasma total protein (76 to 83 g/L), and fibrinogen concentration (5 to 7 g/L). Serum analysis revealed marked hypocalcemia (0.73 to 0.97 mmol/L), relatively high magnesium (1.03 to 2.021 mmol/L), phosphorous (1.52 to 3.55 mmol/L) and glucose (2.42 to 10.77 mmol/L), and marked elevation of BUN (26.95–35.03 mmol/L) and creatinine (314.70–476.47 mmol/L) concentrations. The activity of serum AST, ALT, AP, and GGT were in normal range but there was high activity of serum CK (440 to 4971 U/l).

Gross pathology findings at post-mortem examination included general congestion of subcutaneous tissues, congestion, and some hemorrhages on rumen mucosa and other parts of the gastrointestinal tract, moderate hydrothorax, and hydropericardium, as well as pulmonary edema and hemorrhage. Kidneys were mottled with pale areas, swollen, and congested. Rumen contents were prominently plant residues of eshnan.

The most significant histopathological finding was a heavy deposit of typical oxalate crystals in the cortical and medullary tubules of kidneys. Numerous tubules throughout the kidneys contained clear to slightly yellow acicular or radiating refractile crystals (Fig. 2). Severe diffuse renal tubular necrosis characterized by eosinophilic tubular epithelial cells with pyknotic nuclei, and sloughed epithelial cells in tubular lumina occurred to various degrees in affected kidneys with oxalate deposits. There were also multifocal to diffuse hydropic degeneration and necrosis of ruminal epithelial cells with precipitation of crystalline material and infiltration of inflammatory cells in lamina propria (Fig. 3). There was mild to moderate congestion with vacuolar degeneration in the livers. There was no remarkable lesion in other tissues and organs.

The plant collected from the pasture (Fig. 4) was determined as *S. rosmarinus* by the Herbarium of Ferdowsi University of Mashhad. It was in the budding stage. Other plant samples analyzed for oxalate content, revealed 5.3% of soluble oxalate in the dry matter.

**Fig. 1** An oxalate-intoxicated sheep due to *S. rosmarinus* showing clinical features of hypocalcemia

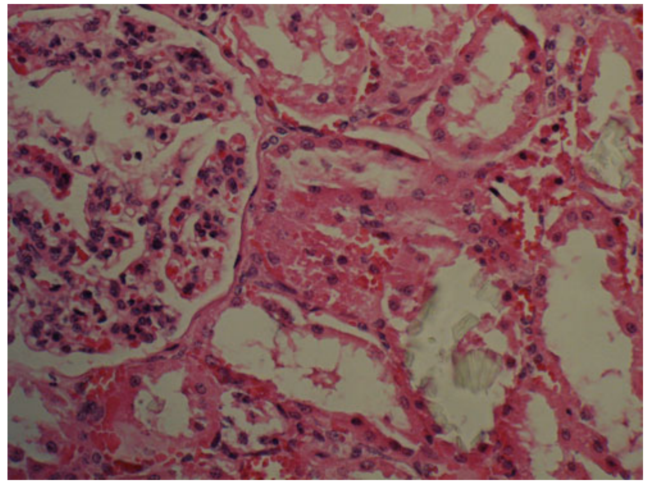

**Fig. 2** Accumulation of typical oxalate crystal (*arrows*) in necrotic renal tubules. Hematoxylin and eosin, ×640

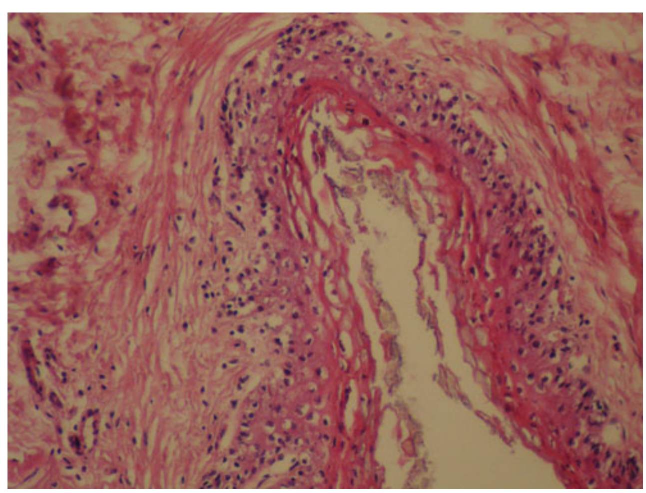

**Fig. 3** Hydropic degeneration of ruminal epithelium with accumulation of crystalline material in superficial layer (*arrows*). Hematoxylin and eosin, ×320

**Fig. 4** *S. rosmarinus* (eshnan) attributed to oxalate intoxication in sheep

## Discussion

The clinical and pathological findings agree with previous descriptions of oxalate poisoning in sheep in which hypocalcemia, azotemia, and nephropathy associated with precipitation of calcium oxalate crystals in renal tubules are characteristic findings (McKenzie et al. 1988; Jacob and Peet 1989; Panciera et al. 1990). The availability and evidence of considerable consumption of large quantities of *S. rosmarinus* as well as considerable contents of oxalate in the plant would appear to establish *S. rosmarinus* as the source of oxalate intoxication.

Plants are the main source of oxalate poisoning in animals. High concentrations of oxalate are present in about 70 species of plants (Radostits et al. 2007). Many species of those plants are found in Iran (Aslani 2005), but none of them have been reported as a cause of animal intoxication and losses. This is thought to be the first report of oxalate poisoning due to ingestion of *S. rosmarinus* in animals. *S. rosmarinus* is a perennial shrub that belongs to the Chenopodiaceae family. Oxalate poisoning associated with ingestion of many members of the Chenopodiaceae has been reported in the literature (Pickrell and Oehme 2004; Naudė and Naidoo 2007; Radostits et al. 2007). *S. rosmarinus* is indigenous in Iran and other countries in the Middle East regions. The plant grows up 1.5 to 2 m tall with a canopy diameter of 1.5 m. The leaves are cylindrical, milky-white, and succulent, measuring 5–33 mm long. It tolerates high levels of salts in the soil and also thrives very well in soils with high alkalinity. It is also a drought-resistant plant and grows very well in low-rainfall desert regions. *S. rosmarinus* is a dominant plant in saline soils in the central part and south-west of Iran (Asadi 2001; Hadi 2009). High levels of mineral element such as iron, copper, magnesium, and potassium are found in *S. rosmarinus* (Towhidi 2007). The plant has been used as forage, particularly for camels, for a long time (Hadi 2009).

Moderate concentration of soluble oxalate (5.3%) was determined in *S. rosmarinus*. The oxalate content of some plants may be as much as 28–30% on a dried weight basis (Jones et al. 1997; Osweiller 1996). The amount of oxalate content above which required to induce acute intoxication in ruminants is quite variable. Acute poisoning and death in sheep can occur in pastures containing as little as 2% soluble oxalate by dry matter (McKenzie et al. 1988). However, induction of acute oxalate intoxication depends on several factors including the rate of consumption, the amount and quality of other feed consumed concurrently, total amount of oxalate consumed, and adaptation to diet containing oxalate (Burrows and Tyrl 1989; Pickrell and Oehme 2004; Radositits et al. 2007). Ruminants in general tolerate relatively more oxalate in their diet than other animals because ruminal microfloras readily metabolize a portion of ingested oxalate, thereby preventing the absorption of soluble oxalate. In addition, the population of ruminal oxalate-degrading microflora increases with gradual exposure to higher concentration of oxalate for 3–4 days and such animals are able to consume considerably greater quantities of oxalate-bearing plants than are animals rapidly consume a single comparable dose (Allison et al. 1977; Burrows and Tyrl 1989; Radositits et al. 2007). Indeed, hungry and unadapted ruminants are the most susceptible ones to oxalate intoxication. Pregnant and lactating animals also are probably more susceptible than others (Radositits et al. 2007).

Introduction of hungry travelling and unadapted sheep to eshnan pasture resulted to high morbidity and mortality associated with acute oxalate intoxication. The plant, *S. rosmarinus*, was in budding stage when grazed by sheep. It has been suggested that many oxalate-bearing plants contain the highest amount of oxalate salts during young, rapidly growing stages and the amount decreases with maturity and drying of the plant (Pickrell and Oehme 2004; Radositits et al. 2007). Such phenomenon has not been demonstrated in *S. rosmarinus* and remained to be elucidated.

Because hypocalcemia is the most significant feature of acute oxalate intoxication, parenteral injection of calcium salts is the specific treatment. For this purpose, subcutaneous or intravenous administration of 50–100 ml of 25% calcium borogluconate is effective and results in rapid recovery (Radositits et al. 2007). On the other hand, oral administration of dicalcium phosphate for binding of soluble oxalate in the gut and fluid therapy for prevention of nephrosis caused by precipitation of oxalate crystals in the kidneys, are recommended as supportive treatment (Pickrell and Oehme 2004).

Sheep may efficiently utilize large quantities of oxalate-bearing plants if proper management practices are followed.

Eshnan must be introduced to sheep gradually and adequate levels of water and good forage variety must be provided.

## References


Allison, M. J., Littledike, E. T. and James, L. F., 1977. Changes in ruminal oxalate degradation rates associated with adaptation to oxalate ingestion. Journal of Range Management, 45, 1173–1179.

Angus, K. W., 2008. Plant poisoning in Britain and Ireland. In: I. D. Aitken (eds), Diseases of Sheep, 4th edn, (Blackwell, Oxford), 405–423.

Asadi, M., 2001. Flora Iranica, No 38 Chenopdiaceae, (Research Institute of Forest and Rangelands, Tehran).

Aslani, M. R., 2005. Poisonous plants of Iran and their effects on animals. 2nd ed, (Ferdowsi University Publication, Mashhad).

Burrows, G. E. and Tyrl, R. J., 1989. Plants causing sudden death in livestock. Veterinary Clinics of North America: Food Animal Practice, 5, 263–289.

Hadi, M. R., 2009. Biotechnological potentials of *Seidlitzia rosmarinus*: a mini review. African Journal of Biotechnology, 11, 2429–2431.

Jacob, R. H. and Peet, R. L., 1989. Acute oxalate toxicity of sheep associated with slender iceplant (*Mesembryanthemum nodiflorum*). Australian Veterinary Journal, 66, 91–92.

Jones, T. C., Hunt, R. D. and King, N. W., 1997. Veterinary Pathology, 6$^{th}$ ed, (Williams & Wilkins, Baltimore).

Maxie, M. G. and Newman, S. J., 2007. Urinary system. In: M.G. Maxie (eds.), Pathology of Domestic Animals, 5th ed, (Saunders-Elsevier, Edinburgh), 425–522.

McKenzie, R. A., Bell, A. M., Storie, G. J., Keenan, F. J., Cornack, K. M. and Grant, S. G., 1988. Acute oxalate poisoning by buffel grass (*Cencheus cilliaris*). Australian Veterinary Journal, 65, 26.

Naudė, T. W. and Naidoo, V., 2007. Oxalate-containing plants. In: R. C. Gupta (eds), Veterinary Toxicology, Basic and Clinical Principle, (Elsevier, Amsterdam), 880–891.

Osweiller, G. D., 1996. Toxicology. (William & Wilkins, Philadelphia).

Panciera, R. J., Martin, T., Burrows, G. E., Taylor, D. S. and Rice, L. E., 1990. Acute oxalate poisoning attributable to ingestion of curly dock (*Rumex crispus*) in sheep. Journal of American Veterinary Medicine Association, 196, 1981–1984.

Pickrell, J. A. and Oehme, F. (2004) Soluble oxalate. In: K. H. Plumlee (eds), Clinical Veterinary Toxicology (Mosby, St. Louis), 345–346.

Radositits, O. M., Gay, C. C., Hinchcliff, K. W. and Constable, P. D., 2007. Veterinary Medicine. 10th edn (Saunders-Elsevier, London).

Sargison, N. D. and Angus, K. W., 2007. Diseases of the urinary system. In: I. D. Aitken (eds), Diseases of Sheep, 4th edn (Blackwell, Oxford), 393–423.

Towhidi, A., 2007. Nutritive value of some herbage for dromedary camel in Iran. Pakistan Journal of Biological Sciences, 10, 167–170.