

Case report

HIGH SULPHUR CONTENT OF WATER FROM DEEP
BORE WELLS AS A POSSIBLE CAUSE OF
POLIOENCEPHALITIS IN A CAMEL

A. AL-SWAILEM¹, M. A. AL-DUBAIB², G. AL-GHAMDI³, E. AL-YAMANI¹,
A. A. AL-NAEEM³, A. M. AL-MEJALI³, M. SHEHATA¹ & O. M. MAHMOUD²

¹ King Abdulaziz City for Science and Technology, ²College of Agriculture and Veterinary Medicine, Qassim University, ³ College of Veterinary Medicine and Animal Resources, King Faisal University; Saudi Arabia

Summary

Al-Swailem, A., M. A. Al-Dubaib, G. Al-Ghamdi, E. Al-Yamani, A. A. Al-Naeem, A. M. Al-Mejali, M. Shehata & O. M. Mahmoud, 2009. High sulphur content of water from deep bore wells as a possible cause of polioencephalitis in a camel. *Bulg. J. Vet. Med.*, 12, No 4, 265–270.

A case of a neurological disease affecting adult camels (locally called Altair) was described. A female camel was presented with typical clinical signs: weakness of legs, continuous head shaking especially when the animal was stressed and wry neck. There were no respiratory, cardiovascular or digestive abnormalities. The she-camel ate and drank normally. Complete blood count results and liver and kidney functions were normal. No visible macroscopic lesions were seen in the carcass except for meningeal congestion. Microscopic examination of the brain showed malacic lesions and the meninges were congested and oedematous. The concentration of sulphur in the drinking water was 13.56 ± 5.8 g/L which is considered very high for the nutrition of farm animals. The concentration of copper, molybdenum and selenium in water and feed were normal.

Key words: camels, deep bore wells, minerals, polioencephalomalacia, sulphur poisoning

Neurological diseases caused by excess sulphur in the diet or drinking water have been documented worldwide (Gooneratne *et al.*, 1989; Niles *et al.*, 2000). Central nervous system disease was observed in a herd of beef cattle drinking water with high sulphate content. Clinical signs, pathological findings, and high water sulphate levels confirmed the diagnosis (Haydock, 2003; Kul *et al.*, 2006). Experimental feeding of high concentrations

of sulphur to sheep was found to be associated with malacic lesions in the brain with consequent development of neurological signs (Gooneratne *et al.*, 1989; Olkowski *et al.*, 1992). It was suggested that effect of sulphur on the nervous system could be through its role in affecting vitamin B₁ metabolism as supplementation with dietary thiamine to sheep given high levels of sulphur prevented the development of nervous signs but did not

totally prevent the development of microscopic brain lesions (Olkowski *et al.*, 1992). The relationship between brain malacia and dietary sulphur intake was studied by Rousseaux *et al.* (1991). They concluded that inorganic sulphur was associated with brain malacia and that dietary thiamine might decrease the severity of the lesion in some affected areas of the central nervous system.

Most of the reports on the toxic effect of sulphur were carried out by controlled experimental feeding of diets containing high concentrations of the mineral to the animals. Only few reports on the natural toxicity of sulphur were reported in sheep and cattle feeding on diets or drinking water with naturally or artificially high content of the mineral (Mahmoud *et al.*, 1993; Jeffrey *et al.*, 1994; Low *et al.*, 1996).

Altair is a neurological disease of adult camels that is linked to drinking deep bore wells' water. Its treatment of choice is vitamin B₁ injections but its etiology remained unknown. This paper investigates the possible etiology of the disease.

A she-camel, of about 8 years of age, was admitted to the Veterinary Teaching Hospital of Qassim University for investigation of the etiology of a neurological disease, locally called Altair. History of the disease revealed that it started with slight gait staggering followed by shaking of the head after two to three weeks. The condition was chronic and had been going on for about 2 months. Wry neck developed after 6 weeks from appearance of clinical symptoms (Fig. 1). There were no systemic signs and the ability of the affected camel to eat and drink was good. The she-camel was diagnosed as being affected with Altair, a neurological disease of unknown etiology.

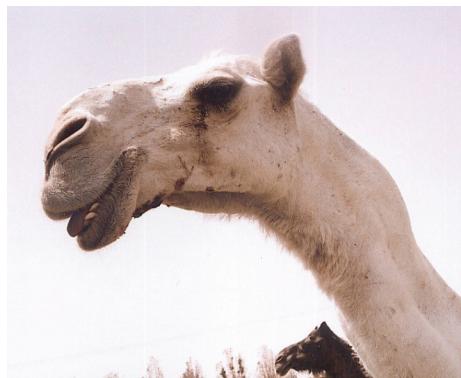


Fig. 1. Wry neck in a she-camel with Altair disease.

Blood samples were obtained from the jugular vein for haematological studies, and biochemical analysis. A blood smear was made and stained with Giemsa stain for parasitological examination. Complete blood counts (CBC) were done by manual methods. Activity of the enzyme aspartate amino transferase (AST) and the concentrations of serum bilirubin and creatinine were determined by an automated analyzer (Reflotron, Switzerland) using commercial assay kits.

Clinical examination showed no respiratory, cardiovascular or digestive manifestations. Blood was negative for parasites and haematology results were normal (Hb: 122 g/L, PCV: 0.32 L/L). Serum analysis results showed normal liver (AST activity of 15.2 IU/L, and bilirubin concentrations of 1.1 µmol/L) and kidney functions (normal creatinine concentration).

The animal was then humanely euthanized with intravenous injection of 50 mL pentobarbital sodium (Vetoquinol, 200 mg/mL, UK) and necropsied. Pieces of tissues from the liver, kidneys and brain were fixed in 10% formol saline, processed in wax, sectioned at 5 µm and

stained with haematoxylin and eosin (H&E) for histopathological examination.

Five samples of drinking water and five from alfalfa normally fed to camels were collected from the area for mineral analysis. The concentrations of sulphur, copper, molybdenum and selenium in water and feed were determined by the methods described in the Ministry of Agriculture, Fisheries and Food Bulletin No. 74 (1973).

Sulphur concentration in drinking water was 13.56 ± 5.8 g/L (Table 1) which is considered too high for the nutrition of ruminants. The concentrations of copper, molybdenum and selenium in the drinking water and fodder were within normal range.

Table 1. Mineral concentration in deep-bore wells drinking water and alfalfa (mean \pm SD)

Mineral	Water (n=5)	Alfalfa, DM basis (n=5)
Sulphur	$13.56 \pm$ 5.8 g/L	$1.46 \pm$ 0.68 g/kg
Copper	$0.005 \pm$ 0.001 ppm	$5.66 \pm$ 1.80 ppm
Selenium	$0.013 \pm$ 0.004 ppm	$1.04 \pm$ 0.28 ppm
Molybdenum	$0.070 \pm$ 0.02 ppm	$0.229 \pm$ 0.04 ppm

Postmortem examination showed absence of gross lesions except for meningeal congestion. Histopathological examination showed malacic lesions in the brain seen as vacuoles in the nervous tissue (Fig. 2). The meningeal blood vessels were congested and there was perivascular and pericellular oedema (Fig. 3).

The authors are not aware of a previous report describing sulphur-induced polioencephalomalacia in Arabian camels. Brain malacia could be naturally or

experimentally induced in animals by a number of factors including high dietary level of sulphur, lead poisoning or the anti-coccidian amprolium. These would directly or indirectly interfere with the metabolism of thiamine (Gould *et al.*, 1991; Tanwar *et al.*, 1993; Wernery *et al.*, 1998). The histopathological picture of the brain and the results of water analysis in this study pointed to the effect of high concentration of sulphur as a cause of the brain malacia seen in the she-camel affected with Altair.

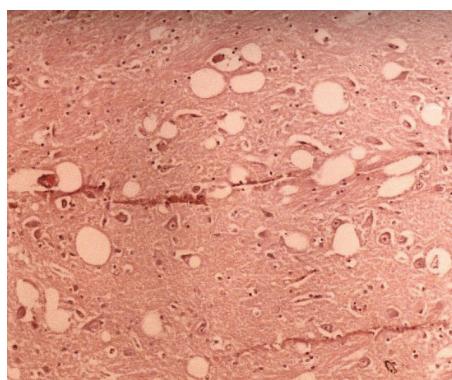


Fig. 2. Vacuolization of the brain. H&E, $\times 125$.

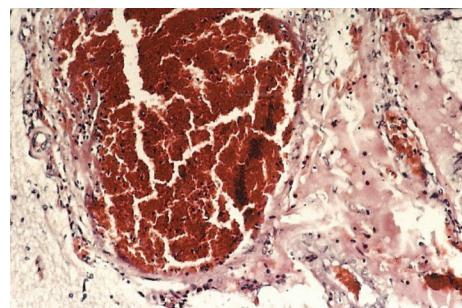


Fig. 3. Congestion and perivascular and pericellular oedema of the meninges. H&E, $\times 250$.

Natural sulphur toxicity is so far unknown among camels in Saudi Arabia but Mahmoud *et al.* (1993) reported malacic lesions in the brain of sheep drinking

deep bore well's water high in sulphur in certain farms at central Saudi Arabia. Affected sheep suffered complete paralysis and massive liquefactive necrosis of the brain.

Cattle, drinking water high in sulphur are reported to develop nervous signs and malacic brain lesions (Gould *et al.*, 1991; 1997; Gould, 1998).

In the past all brain malacic diseases were blamed on primary thiamine deficiency causing cerebrocortical necrosis (Jones *et al.*, 1997). Many cases with malacic lesions had a low content of thiamine in blood and brain, and thiamine administration is often therapeutic.

Vitamin B₁ injection was found effective in the treatment of malacic conditions of the brain caused by sulphur excess (Raisbeck, 1982; Gooneratne *et al.*, 1988). Some controversy exists however as to whether high concentration of sulphide is the primary cause of sulphur-induced polioencephalomalacia (PEM). High levels of sulfur have been implicated in decreased levels of thiamine (Brent & Bartley, 1984). Excess sulphur may decrease the levels of thiamine, either directly and/or through the stimulation of the production of thiaminase (Olkowski *et al.*, 1992). It has also been suggested that the transient sulphite that is produced during the reduction of sulphate to sulphide, could have a direct impact on the brain tissue itself (Brent & Bartley, 1984; Olkowski *et al.*, 1992; Oliveira *et al.*, 1996). Sulphite-derived radicals have been postulated to cause lipid peroxidation and to damage biological membranes. Because of the high lipid content of the brain, and its inability to be efficiently repaired, it becomes apparent why lesions are first seen in its tissue (Olkowski *et al.*, 1992).

The incidence of Altair in camel herds is usually very low (1–2%) even when the

whole herd was exposed to the high level of sulphur in drinking water. Gould *et al.*, (1991) showed that the experimental ingestion of toxic amounts of sulphur failed to induce clinical signs in all treated calves. Different reasons could be involved in this failure, such as previous dietary sulphur level, production of hydrogen sulphide by rumen microflora, ruminal pH affecting thiamine metabolism and amount of easily digestible polysaccharides in the diet (Gould *et al.*, 1991).

Copper, molybdenum or selenium deficiencies might cause clinical signs similar to those induced by sulphur excess (Jones *et al.*, 1997). Yet, our results have showed that the histopathological lesions seen in the necropsied she-camel were inconsistent with those seen in copper or selenium deficiencies. Further, the concentrations of these minerals in the diet and drinking water consumed by the affected camel were normal and thus, the possibility for them to cause Altair disease was excluded.

In conclusion, our results obtained for Altair disease as a high dietary sulphur-associated PEM were similar with those reported for other animals worldwide. The high concentrations of sulphur in the drinking water in this study were suggested to cause the lesions of Altair disease of the camel, as its concentrations in blood or tissues were not determined. Since most of the camels in central Saudi Arabia drink water from deep-bore wells, analysis of its sulphur content is necessary to safeguard the health of camels as well as sheep in the area.

ACKNOWLEDGEMENTS

This work was financed by King Abdulaziz City for Science and Technology (KACST), Riyadh, Saudi Arabia. It is part of a research

programme investigating the etiology of neurological diseases of camels in the northeastern region of the country.

REFERENCES

- Brent, B. E. & E. E. Bartley, 1984. Thiamin and niacin in the rumen. *Journal of Animal Science*, **59**, 813–822.
- Gooneratne, S. R., A. A., Olkowski & D. A. Christensen, 1989. Sulphur-induced polioencephalomalacia in sheep: Some biochemical changes. *Canadian Journal of Veterinary Research*, **53**, 462–467.
- Gould, D. H., M. M. McAllister, J. C. Savage & D. W. Hamar, 1991. High sulphide in rumen fluid associated with nutritionally-induced polioencephalomalacia in calves. *American Journal of Veterinary Research*, **51**, 1164–1169.
- Gould, D. H., B. A. Cummings & M. Hamar, 1997. *In vivo* indicators of pathologic ruminal sulphide production in steers with diet-induced polioencephalomalacia. *Journal of Veterinary Diagnostic Investigations*, **9**, 72–76.
- Gould, D. H. 1998. Polioencephalomalacia. *Journal of Animal Science*, **76**, 309 – 314.
- Haydock, D. 2003. Sulphur-induced polioencephalomalacia in a herd of rotationally grazed beef cattle. *Canadian Veterinary Journal*, **44**, 828 – 829.
- Jeffrey, M., J. P. Duff, R. J. Higgins, V. R. Simpson, R. Jackman, T. O. Jones, S. C. Mechic & C. T. Livesey, 1994. Polioencephalomalacia associated with ingestion of ammonium sulphate by sheep and cattle. *The Veterinary Record*, **134**, 343–348.
- Jones, T. C., R. D. Hunt & N. W. King, 1997. Diseases caused by viruses. In: *Veterinary Pathology*, 6th edn, William & Wilkins, Philadelphia, USA., p. 197.
- Kul, O., S. Karahan, M. Basalan, & N. Kabakci, 2006. Polioencephalomalacia in cattle: A consequence of prolonged feeding barley malt sprouts. *Journal of Veterinary Medicine*, **53**, 123–128.
- Low, J. C., P. R. Scott, F. Howie, M. Lewis, J. Fitzsimons & J. A. Spence, 1996. Sulphur-induced polioencephalomalacia in lambs. *The Veterinary Record*, **138**, 327–329.
- Mahmoud, O. M., E. M. Haroun & A. Sulman, 1993. Encephalomyelomalacia in lambs drinking deep-bore water high in sulphur content. SVS, Edinburgh, UK, pp. 205–206.
- Ministry of Agriculture, Fisheries and Food, 1974. The Analysis of Agricultural Material. Bulletin No. 74, Her Majesty's Stationery Office, London.
- Niles, G. A., S. E. Morgan & W. C. Edwards, 2000. Sulphur-induced polioencephalomalacia in stocker calves. *Veterinary and Human Toxicology*, **42**, 290–291.
- Oliveira de, L. A., C. V. D. Jean-Balin, V. Corso, A. Benard, A. Durix & S. Komisarczuk-Bony. 1996. Effect of high sulfur diet on rumen microbial activity and rumen thiamine status in sheep receiving a semi-synthetic, thiamine-free diet. *Reproduction Nutrition Development*, **36**, 31–42.
- Olkowski, A. A., S. R. Gooneratne, C. G. Rousseaux & A. Christensen, 1992. Role of thiamine status in sulphur induced polioencephalomalacia in sheep. *Research in Veterinary Science*, **52**, 78–85.
- Raisbeck, M. F., 1982. Is polioencephalomalacia associated with high sulphate diets? *Journal of the American Veterinary Medical Association*, **180**, 1303–1305.
- Rousseaux, C. G., A. A. Olkowski, A. Chauvet, S. R. Gooneratne, & D. A. Christensen, 1991. Ovine polioencephalomalacia associated with dietary sulphur intake. *Zentralblatt für Veterinärmedizin*, **38**, 229–239.
- Tanwar, R. K., K. S. Malik & J. R. Sadana, 1993. Polioencephalomalacia induced with amprolium in buffalo calves: Pathologic changes of the central nervous system. *Journal of Veterinary Medicine Series A*, **40**, 58–66.
- Wernery, U., J. Hayden-Evans & J. Kinne, 1998. Amprolium-induced cerebro-cortical necrosis (CCN) in dromedary racing ca-

mels. *Journal of Veterinary Medicine B*,
45, 335–343.

Paper received 13.04.2009; accepted for
publication 08.06.2009

Correspondence:

Prof. O. M. Mahmoud
Department of Veterinary Medicine,
Faculty of Agriculture and
Veterinary Medicine,
Qassim University,
P. O. Box 6622 Buraydah, Qassim,
Saudi Arabia.
e-mail: oabdelbari@gmail.com