Water intoxication in cattle

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ABSTRACT
Water intoxication is a condition that is common in cattle, and has also been reported in other domestic animals and man. A comprehensive description of the condition is lacking. For a better understanding of the condition, this paper reviews work that has been reported previously by various authors.

Key words: adult cattle, calves, milk, salts, water intoxication.

INTRODUCTION
Water intoxication is a condition that occurs when excessive quantities of water are ingested by very thirsty animals. It commonly occurs in animals that have been subjected to severe exercise or high environmental temperatures.

Water intoxication was first reported as a distinct syndrome in man. Subsequently, naturally occurring cases have been reported in man, cattle, sheep, pigs, camels, calves, and adult cattle. Calves are the most commonly affected animals.

ANIMALS AFFECTED
In cattle, the condition has been reported to occur in all ages, but it is more common among calves 2–10 months of age and is most frequent in calves 3–5 months of age. All breeds of cattle are affected. The condition has no sex predilection.

PREDISPOSING FACTORS
A number of factors predispose calves to water intoxication. Young bucket-fed calves usually drink excessive quantities of water if it is offered to them in the manner in which they usually receive their milk. Under such circumstances, the limit of their consumption is apparently governed not by satiety, but by the physical limit of capacity of the gut. Another predisposing factor is the high ruminal capacity to hold ingested water as compared with total body size.

Chronic subclinical dehydration accompanied by increased environmental temperature, exercise or increased body water loss due to diarrhoea or fever also predisposes calves to water intoxication. Calves in an oliguric state or those that fail to rapidly develop diuresis after water overloading have a higher predisposition to water intoxication than normal calves.

More recent findings indicate that failure to provide calves with both water and salts from as early as 2 weeks of age to weaning predisposes them to water intoxication. Under experimental conditions, calves raised solely on milk from 2 weeks of age to weaning developed the condition while those raised on milk, water and salts did not. The salts used in the experiment included many minerals. However, the authors did not determine the role that each mineral component of the salt played in prevention of water intoxication.

Age has also been reported to predispose animals to the condition. Haemoglobinuria is found most frequently in calves 3–5 months of age. In adult cattle, the most common predisposing factor is chronic subclinical dehydration due to prolonged failure of water supply, accompanied by increased environmental temperature. They develop chronic oliguria and fail to rapidly develop diuresis after water overloading.

PATHOGENESIS
In Kenya, a similar case history is given by farmers in almost all cases reported (Clinical Records, Large Animal Clinic, Clinical Studies Department, University of Nairobi). Animals are exposed to a water source where they drink ad libitum after a long period of restriction and then start voiding ‘bloody’ urine. The farmer may report having raised calves on milk or milk substitutes alone, without access to additional water, followed by either unlimited water supply or an automatic water source. One or 2 calves are usually involved on small-holder farms, while several may be involved on large beef and dairy farms. In adult cattle, the commonly reported cases involve beef herds raised on rangelands that have had no water supply, followed by excess supply.

CLINICAL SIGNS
In a group of calves, water intoxication may be manifested in all or only a few of the animals. Clinically, the condition is characterised by haemoglobinuria and nervous signs. The nervous signs include hyperaesthesia, muscular tremors, myotis and lethargy. Mild cases recover in 3–4 hours. In severely affected animals, lethargy may progress to depression and coma, and death occurs in 24–48 hours. In some cases haemoglobinuria is the only clinical sign observed; in others, the nervous signs predominate and haemoglobinuria is not detected. Additional signs include hypothermia, salvation, severe ruminal tympany, colic, diarrhoea, arrhythmia, oedema of the eyelids and erection of body hairs.

In adult cattle, water intoxication is manifested clinically by haemoglobinuria. Other signs present in calves are rare. Unlike calves, bloody diarrhoea and abortion have not been recorded in cattle.

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The salt (Macik Super, Unga Feeds, Kenya) used had the following constituents: NaCl 27 %; Ca 18.51 %; P 11.00 %; Mg 3.90 %; Fe 0.50 %; Cu 0.16 %; Mn 0.40 %; Zn 0.50 %; S 0.40 %; Co 0.02 %; I 0.02 %; Se 0.0015 %; Mo 0.0002 %.

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down a concentration gradient into the cells. Cellular hydration occurs and in organised tissues, the cells increase in turgidity; this is evident in the brain, where oedema occurs, causing nervous signs2,22,23. In unorganised tissues, particularly the erythrocytes, hypotonicity of the blood leads to lysis of the cells and the resulting haemolysis may cause severe anaemia and haemoglobinuria.22,23,24 In calves, haemoglobinuria has also been attributed to fragility of erythrocytes. The osmotic fragility of erythrocytes has been found to be higher in calves suffering from water intoxication than those that do not suffer from the condition25. In adult cattle, fragility of erythrocytes has not been reported to play a role in the pathogenesis of water intoxication.

**POST MORTEM LESIONS**

In both calves and adult cattle, the carcass is usually in poor condition and shows severe ruminal tympany8,22,23. Grossly, there may be fluid in both the abdominal and thoracic cavities. The fat in the coronary grooves of the heart may be gelatinous. The urinary bladder usually contains red urine. The kidneys appear dark. In calves, the sulci on the surface of the brain are flattened, indicating a degree of oedema. This has not been reported in adult cattle. Unlike in the camel, rupture of the abomasum has not been observed in either calves or adult cattle. Ruptured abomasum has been reported to be the only significant post mortem finding in camels22,24.

Microscopic changes have been reported only in calves. Histological examination reveals oedema and haemorrhage of the brain parenchyma22. Cerebral veins and venules are congested and fill the Virchow-Robin spaces. The kidney may show marked lymphocytic infiltration of the cortex and fibrosis around the capillaries. The renal medulla may have areas of haemorrhage and congested capillaries. The epithelial cells of the kidney tubules contain fine, red granules. The urinary bladder may have areas of oedema, both beneath the stratified mucosa and in the muscular layer25.

**DIFFERENTIAL DIAGNOSES**

Water intoxication needs to be distinguished from other diseases that cause nervous signs and haemoglobinuria. Numerous diseases and intoxications can cause nervous signs in cattle of various ages. Babesiosis is the most usual cause of haemoglobinuria in adult cattle, and may sometimes be observed in calves.

**TREATMENT**

Hypertonic saline (5 %) given intravenously has been recommended for treatment of water intoxication26. However, this is effective only in mild to moderate cases. In severe cases, use of both 5 % dextrose-saline solution intravenously and a tranquiliser such as acetylpromazine has been found to achieve a better response in calves1,22.

**PREVENTION**

Prevention of water intoxication can be achieved by provision of salts and water ad libitum to calves from as early as 2 weeks of age. The salts may be provided as blocks or powder licks for the calves to lick ad libitum27.

**WATER INTOXICATION AND SALT POISONING**

Whereas water intoxication results from a state of positive water balance within the body, salt poisoning occurs when excessive amounts of salt are ingested or animals have been drinking saline water without access to fresh water. The salt ingested causes irritation of the gastrointestinal tract. This is manifested as diarrhoea with mucous in the faeces, depression of body temperature, abdominal pain and anorexia. Some salt is absorbed and may cause involvement of the central nervous system. The signs in this case are similar to those of water intoxication in calves. However, a nervous syndrome due to salt poisoning is present in both calves and adult cattle, while that due to water intoxication is common only in calves. Haemoglobinuria is the most common clinical sign in cases of water intoxication but has not been reported in salt poisoning. In salt poisoning, the plasma Na+ is elevated while in water intoxication it is lower than in healthy animals.

Cattle that die of salt poisoning show marked congestion of the mucosa of the omasum and abomasum. Fluid faeces that are dark in colour may be present.

Treatment of salt poisoning involves removal of the toxic feed or water and provision of fresh water in small quantities. In cases of nervous involvement, treatment is similar to that for water intoxication in calves with nervous signs.

**REFERENCES**

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One of the recurring questions – largely rhetorical – within the veterinary fraternity in South Africa concerns the relative impact and cost of some of the major epidemic and endemic diseases that confront us, particularly those caused by ticks and tick-borne diseases (TTBD). This excellent compendium of 16 papers dealing with the economics of disease control makes 2 points on this issue. First, that the question itself is not relevant to the debate as far as the economics of control is concerned. The reason, as pointed out by R S Morris in the introductory chapter, is that the guiding principle (equimarginal returns) should be ‘to allocate funds progressively, moving resources to wherever the return on the next investment dollar is highest’, and away from activities that generate a lower return. Second, if the reference list of the relevant chapter is a reliable measure, South Africa has contributed nothing significant to the much-studied economics of TTBD. This is clearly not true but it may be so that South Africa’s vast experience and knowledge in this field is not easily accessible to the international community. One suspects that the answer lies in differences of approach – our essentially pragmatic/trial-and-error perspective versus a more systematic and academic approach espoused by Australians and Europeans operating in Africa. In the modern world, where the economics of production are increasingly important and complex, we probably have a lesson to learn from the latter group. Current plans to establish an integrated epidemiology unit at Onderstepoort that includes capacities for impact and risk analysis makes this compendium extraordinarily opportune from the South African perspective.

A particularly interesting statement, also by R S Morris, contends that the net economic benefit obtained from investment funds directed towards controlling animal diseases is commonly in the range of 200–1500 %, while the yields derived from investment in other activities in the livestock sector are commonly about 20 %. These are the sort of data that those of us in public service need to attract the attention of the purse-string holders. The coordinator (B D Perry), the editorial staff of the OIE and especially the authors themselves, have produced a high-quality volume with an appropriate balance of subject matter. Rushton, Thornton and Otte’s chapter covering methods (partial budgets, break-even analysis, cost/benefit analysis etc.) is the only one that deals in any detail with the technicalities of control economics. Other chapters are concerned mostly with the application, using interesting examples, of control economics in both the developing and developed world and at different geographic levels, namely regions, countries, zones within countries and individual farms with different production systems. For that reason very few veterinarians dealing with production animals would not find this volume both interesting and instructive. There are many informative illustrations and no irritating editorial errors or inconsistencies were encountered. Two of the papers are not in English (Spanish and French) but they do have English summaries.

The appeal of different chapters in this volume will depend very much on the interests of the reader. To provide some indication as to the types and range of chapters the following are given as examples of the subject matter:

- Control and eradication of epidemic diseases.
- Endemic diseases and disease control programmes.
- Health and productivity in smallholder livestock systems in developing countries.
- Delivery of veterinary services.
- Implications of greater global trade in livestock and livestock products.
- Rinderpest control in Africa.
- Health and productivity in the commercial dairy.
- Privatising veterinary services in Africa.

The chapter on trends and predictions for global trade in livestock and livestock products by Leslie and Upton contains important considerations for South Africa. For example, they deduce that increasing global trade in livestock products will disadvantage net food importers while favouring high-income countries. South Africa is, fortunately, a net food exporter but on the livestock side the reverse is the case. We are 8th, according to tables contained in the chapter, among world importers of ovine meat (29 600 tonnes). They also predict that grassland-fed ruminant meat and milk is likely to capture market share from pig and poultry producers, presumably as a result of BSE-type issues currently troubling consumers in Europe.

For anyone involved in production animal health where financial considerations are important (and where is that not the case?) this volume will provide an invaluable source of information.

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