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## Water intoxication in adult cattle

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### ABSTRACT

**Water intoxication is a common disorder in calves and is usually characterized by transient hemoglobinuria. In contrast, the condition is very rare in adult cattle, with few reports on naturally occurring cases. In the present report, four female Japanese Black cattle, aged 16-25 months, showed neurological signs when they drank water following a water outage. Hemoglobinuria was not grossly observed, while severe hyponatremia was revealed by laboratory tests. Autopsy indicated cerebral edema with accumulation of serous fluid in expanded Virchow-Robin spaces. These results indicate the possibility of water intoxication associated with cerebral edema due to severe dilutional hyponatremia in adult cattle.**

Key Words: cattle, cerebral edema, water intoxication

Water intoxication is a relatively well-known disorder in calves, which occurs when calves are exposed to ad libitum access to water after a certain period of restriction<sup>9)</sup>. The condition is characterized by transient hemoglobinuria and hemoglobinemia. It is considered that excessive water ingestion causes a temporary extreme drop in plasma osmotic pressure in the capillary veins of the intestinal wall, where intravascular hemolysis may occur<sup>11)</sup>. In addition, depending on the amount of absorbed water, hemodilution in systemic veins may also occur, and consequently, various degrees of hyponatremia are observed<sup>8,9)</sup>. There have been many reports on naturally occurring cases of water intoxication in

calves<sup>2,3,5,14)</sup>, while there are few reports of this condition in adult cattle<sup>4)</sup>. Recently, we experienced a case of water intoxication characterized by neurological signs in adult cattle that drank their fill after a relatively short period of water outage.

The case described in the present report occurred on a fattening farm in Yamagata, Japan, in which about 850 Japanese Black cattle, aged 7-32 months, were raised in groups of two or three animals. This farm consisted of four cow houses. Two of them used tap water as drinking water, and the others used well water.

In 2013, on July 18, the Yamagata prefecture

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sustained torrential rain, and around noon on July 21, due to a troubled water filtration plant, the water supply was discontinued in the area around the farm. On the afternoon of July 22, the water in the tanks of two cow houses, in which about 700 cattle were housed, was depleted. Therefore, on the morning of July 23, the rancher switched the water source from tap water to well water, and let the cattle drink it until they were satisfied. However, about 10 hours after giving well water, the rancher found two animals (Cases 1 and 2) lying down with neurological signs. On July 24, all animals were allowed to drink well water *ad libitum*, except during the night time, but on July 25, after the resumption of water supply, another two cows (Cases 3 and 4) were observed having similar clinical signs. The feed given to the cattle consisted of rice straw and commercial compound feeds which contained no salt. The daily ration was not changed even after water outage, but it was reduced to two-thirds after the occurrence of Cases 1 and 2.

All four affected animals were female and their ages (Cases 1 to 4) were 25, 22, 16, and 17 months, respectively. On the first day, Cases 1 and 4 were in recumbent positions with muscle twitches and coma and were unable to get up. Cases 2 and 3 were able to stand up but showed depression, staggering, and disturbed consciousness during which they kept moving against the bars of the stall and were unable to drink or eat by themselves. All of them showed symptoms of dehydration such as enophthalmus and skin hardening, and Case 1 exhibited frequent urination. Their rectal temperature were within normal ranges, and hemoglobinuria, which is one of the most common manifestations of water intoxication in calves<sup>3,8-11,14</sup>, was grossly unnoticeable in all cases.

The results of laboratory tests are presented in Table 1. Serum osmolality was estimated according to the following formula<sup>6</sup>:

$$\text{Serum osmolality (mOsm/kg)} = 2[\text{Na}] + [\text{Glu}]/18 + [\text{BUN}]/2.8$$

where Na = sodium (mM), Glu = glucose (mg/dl), and BUN = blood urea nitrogen (mg/dl).

Hyponatremia, hypochloremia, and low serum osmolality were revealed in three of the four animals, and all four cows had increased blood levels of glucose (Glu), creatine kinase (CK), and aspartate aminotransferase (AST). A urine test was performed only in Case 4. The specific gravity of the urine was 1.013, and the urine sodium concentration was 1 mM. Although the urine color was grossly normal, a slight occult blood reaction was observed.

The major diseases of adult cattle which cause neurological abnormalities include infectious thromboembolic meningoencephalitis, listeriosis, polioencephalomalacia, salt poisoning, and ruminal acidosis. In this case, the infections were ruled out based on the absence of fever, and polioencephalomalacia was ruled out because a number of animals developed neurological signs suddenly and for a short time. Salt poisoning was ruled out, based on the absence of salt in the feed. Therefore, we initially made a tentative diagnosis of acute ruminal acidosis in Cases 1 and 2. However, the results of the laboratory tests revealed hyponatremia with a low serum osmolality and suggested the possibility of water intoxication. In Case 4, we tried to classify the hyponatremia according to the diagnostic criteria applied in humans<sup>6</sup> to confirm the diagnosis of water intoxication. Firstly, the specific gravity of urine should be lower than 1.003 in euvolemic patients with hyponatremia and hypo-osmolality. However, that of Case 4 was inappropriately high (1.013), suggesting disturbance of free water excretion caused by volume overload, volume contraction, or syndrome of inappropriate antidiuretic hormone secretion. In this case, volume contraction was suspected on the basis of the history and the symptoms of dehydration. In

**Table 1. Results of the laboratory tests on the first day**

	Case 1	Case 2	Case 3	Case 4	Normal range
Peripheral blood cell count					
WBC ( $\times 10^2/\mu\text{L}$ )	178	94	220	103	40–120
RBC ( $\times 10^4/\mu\text{L}$ )	646	1024	1007	784	500–1000
Hematocrit (%)	33.7	46.5	45.0	35.9	24–46
Blood biochemistry					
Total protein (g/dl)	7.2	7.0	7.8	6.6	5.7–8.1
Albumin (g/dl)	3.2	3.5	3.9	3.5	2.1–3.6
CK (IU/l)	870	465	7389	1768	35–280
AST (IU/l)	198	358	154	144	41–109
Sodium (mM)	128	117	139	117	132–152
Potassium (mM)	2.5	3.7	3.6	4.5	3.9–5.8
Chloride (mM)	91	78	98	80	95–110
Calcium (mg/dl)	9.2	8.3	9.3	8.3	8.5–12.4
Total cholesterol (mg/dl)	98	101	78	207	65–220
Glucose (mg/dl)	176	239	99	91	45–75
BUN (mg/dl)	7.1	6.7	8.1	10.2	6.0–27
Serum osmolality (mOsm/kg)	268	250	286	243	270–306
Urine test					
Specific gravity				1.013	
Occult blood test				+	
Urine biochemistry					
Sodium (mM)				1	
Potassium (mM)				34.2	
Chloride (mM)				12	

WBC: White blood cell count, RBC: Red blood cell count, CK: Creatine kinase, AST: aspartate aminotransferase, BUN: Blood urea nitrogen.

patients with hypovolemic hyponatremia and normal renal function, the urine sodium concentration should be lower than 20 mM. Therefore, the remarkably low urine sodium concentration of Case 4 (1 mM) suggested its normal renal function and the existence of extrarenal fluid losses, causing volume contraction. Extrarenal fluid losses may occur as a consequence of protracted vomiting, severe diarrhea, or sequestration of fluids in a third space. In this case, such symptoms were not present, so the volume contraction was assumed to be due to the failure of water supply. It is known that these fluid losses can typically cause hyponatremia when losses are replaced by ingesting plain water or liquids low in sodium<sup>6,9)</sup>. In this case,

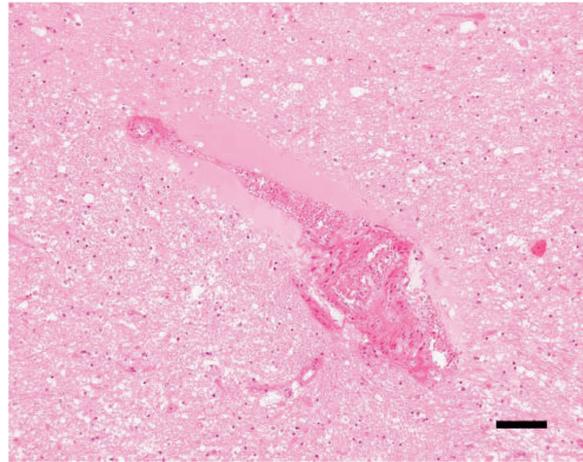
the volume contraction was subsequently resolved by drinking a large amount of well water. However, its sodium concentration was later revealed to be only 2.3 mM, and severe dilutional hyponatremia was suspected.

On the first day, we treated Cases 1 and 2 using a slightly hypotonic solution (sodium concentration, 106 mM and 76 mM, respectively) including sodium bicarbonate for suspected ruminal acidosis and Cases 3 and 4 using a hypertonic solution (sodium concentration, 420 mM and 355 mM respectively). Intravenous administration of hypertonic saline has been recommended for water intoxication in calves<sup>8,9)</sup>; however, in these cases, neither hypotonic nor

hypertonic solution was effective, and the final outcomes appeared to depend on the severity of the neurological signs observed on the first day.

Only Case 2 gradually recovered and was able to feed spontaneously four days after the onset. Cases 1, 3, and 4 finally died 3, 4, and 1 days after the onset, respectively, and were immediately subjected to autopsy. During the autopsies of Cases 1 and 4, a softening and a wetness of the brain and an increase in the cerebrospinal fluid were observed grossly. Moreover, histological examination revealed that all cases had accumulation of serous fluid in expanded Virchow-Robin spaces (Fig. 1). These results indicated the existence of cerebral edema<sup>7</sup>, which was considered to be the cause of the neurological abnormalities. Case 4, the most severe case, had a hemorrhagic lesion in the brain parenchyma and diffuse congestion and edema of the lung. Cases 1 and 3 had moderate fibrinous bronchopneumonia, which was suspected to contribute to the clinical exacerbation. Additional autopsy findings typically included mild partial myoedema around the chest wall, thinning and fragility of the mucosa of the rumina, and congested mucosa of the abomasum and a portion of the small intestine. Case 4 showed enlargement and fragility of the liver. In humans, rapid correction of hyponatremia is known to increase the risk of developing osmotic demyelination syndrome<sup>6</sup>. In the present report, although the affected animals were not to be treated with sufficient care regarding the rate of correction, examples of demyelination were not observed.

Evidence from laboratory tests and autopsies indicated that water intoxication might occur in adult cattle as well as in calves. In cases of water intoxication in calves, the most common manifestations is transient, uncomplicated hemoglobinuria<sup>3,11,14</sup>. However, in these adult cattle, obvious hemoglobinuria was not observed. It has been reported that the osmotic tolerance of bovine red blood cells changes according to age,



**Fig. 1. Accumulation of serous fluid in expanded Virchow-Robin spaces (Case 4).** Hematoxylin and eosin. Scale bar = 100  $\mu$ m.

being lowest around the age of 4 to 5 months<sup>10</sup>. This is considered to be the primary reason why the manifestations of water intoxication differ between calves and adult cattle.

A case of water intoxication in adult range cattle has been reported previously; however, detailed laboratory tests and autopsies were not performed<sup>4</sup>. Furthermore, in that report, the animals had suffered severe dehydration before water intake, and some of them had already died. Therefore, the animals might have developed serious hemoconcentration and hypernatremia and might have been in a condition of salt poisoning. In salt poisoning, sodium ions are accumulated in tissues, including the brain, and when water is made available, it migrates to the tissues according to the concentration gradient<sup>9</sup>. In the previous report, some of the cattle died soon after drinking a small amount of water. The cause of their death is supposed to have been exacerbation of salt poisoning. On the other hand, the affected cattle in the present case did not show serious dehydration before water intake, because the temperature at the time of the incident was lower (the daily maximum temperature was between 25 and 30°C), and the period of water restriction was shorter (about 1 day) than that of the previous report<sup>4</sup>. In addition,

there were no symptoms suggestive of salt poisoning, such as diarrhea or neurological signs, and no salt was contained in the feed. Therefore, it was unlikely that the animals in this case were in a condition of salt poisoning.

Hyponatremia was considered to be one of the most important findings in diagnosis of water intoxication in adult cattle. However, hyponatremia shown in Case 1 was rather mild, and Case 3 did not have hyponatremia. In humans, it is known that mild dilutional hyponatremia with normal renal function can be treated only by restriction of water intake because the kidneys produce large quantities of dilute urine and excrete excessive free water<sup>6</sup>. In the present report, Cases 1 and 3, which did not have serious hyponatremia, were unable to drink water spontaneously, and Case 1 showed frequent urination. Hence, it was possible that the two cows had developed water intoxication shortly before, and that hyponatremia had been already resolved by production of dilute urine when the rancher found their abnormalities. An increase in AST has been also encountered in the cases of water intoxication in calves<sup>3</sup>. In the present case, increased CK was also observed, and the damage of the muscle was suspected. In humans, rhabdomyolysis associated with water intoxication was reported, in which the peak of CK was about 48–96 hours after the onset of water intoxication<sup>1,12,13</sup>. Therefore, the level of CK may be useful for prediction of the length of elapsed time after the onset.

Chronic subclinical dehydration and salt deprivation are suggested to be the predisposing factors of water intoxication in calves. Dehydrated animals may develop chronic oliguria and fail to rapidly develop diuresis after water overloading<sup>3,4,8</sup>. Moreover, inadequate water intake causes a decrease both in total body water and in total body sodium content because the balance of water and sodium is maintained within very narrow limits<sup>6</sup>. Animals with decreased total body sodium content are more likely to develop

overhydration due to the relatively small amount of water ingested<sup>3,8,9</sup>. The mechanism by which salt deprivation increases the risk of water intoxication is still unclear, but the feed without salt might have contributed to decreased total body sodium content. In addition, the continued feeding of the cattle during the water outage might have increased their desire to drink and might have predisposed them to water intoxication.

In conclusion, this report has demonstrated that water intoxication may occur in adult cattle even in the absence of serious dehydration. Therefore, veterinarians should always bear this disorder in mind when failure or irregularities of water supply to cattle is suspected.

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