Received: 27 February 2024 | Revised: 14 September 2024 | Accepted: 29 September 2024

DOI: 10.1002/vrc2.1014

VetRecord CaseReports

CASE REPORT

Food/farmed animals

Biochemical changes in the aetiology of floppy kid syndrome in goat kids

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Funding information
Research Project of the Ministry of Education and Culture of the Czech Republic, Grant/Award Number: MSM6215712403

Abstract
In the herd, 20 of 75 goat kids born became progressively ill. Twelve affected kids, aged 4–9 days, exhibited muscle weakness, pain without fever, diarrhoea and dehydration. Their health deteriorated rapidly, and within a few hours, one kid died. The results of the blood test revealed severe metabolic acidosis, hypokalaemia and changes in urea concentrations. Floppy kid syndrome was diagnosed. Treatment was initiated to correct the acid–base balance, and all affected kids were temporarily separated from their mothers. All the treated kids recovered. Before the onset of clinical signs, elevated urea concentrations and differences in maternal blood potassium levels were observed. This is the first report on changes in potassium concentrations in periparturient dams of goat kids with and without floppy kid syndrome, as well as the first report on changes in urea concentrations in kids before the onset of clinical signs.

BACKGROUND

Floppy kid syndrome, a metabolic disease of goat kids, was first reported in Canada as a muscular weakness without signs of diarrhoea and dehydration. If untreated, the animals died.[1] Later, floppy kid syndrome was defined as D-lactate acidosis in kids, characterised by a high anion gap without dehydration.[2] The syndrome is known by various names: neonatales lähmungssyndrom, syndrome chevreau mou, or Síndrome do Carbito mole.[3–5] Currently, the aetiology of floppy kid syndrome is primarily attributed to non-infectious causes (intensive milk feeding with subsequent clostridial overgrowth and intestinal dysbacteriosis), although the possible influence of bacterial agents from the mother or the environment has also been suggested.[6–9] The most common clinical sign is the sudden onset of profound muscle weakness in healthy newborn kids aged 3–10 days.[6] In the blood of affected kids, severe metabolic acidosis and occasionally hypokalaemia are evident.[1,2] Treatment involves correcting metabolic acidosis through intravenous or oral administration of sodium bicarbonate.[5,6] Although floppy kid syndrome has been known for a long time, there are only a few case reports on this disease, likely related to unconfirmed (undiagnosed) causes of neonatal mortality in goats under practical farming conditions.[10] In this report, we describe the clinical presentation and treatment of floppy kid syndrome in kids from one herd, living under the same diet and housing conditions. We described selected biochemical changes in goat kids and alterations in potassium concentrations in their periparturient mothers.

CASE PRESENTATION

The experimental goat herd, consisting of 34 white short-haired goats in their first and second lactation periods, was regularly monitored with daily health assessments. The goats were fed twice a day with a complementary diet made up of barley (30%), wheat (20%), alfalfa meal (18%), unpeeled sunflower seeds (10%), wheat bran (10%), maize (5%), malt sprouts (5%), dicalcium phosphate (1.1%), sodium chloride (0.7%) and calcium carbonate (0.2%). The daily dose of the complementary diet (0.6 kg per animal before delivery; 1.0 kg per animal after delivery) was provided in combination with hay, water and NaCl lick ad libitum.

FIGURE 1 Healthy newborn kids.

> **LEARNING POINTS/TAKE-HOME MESSAGES**
>
> Floppy kid syndrome is an acute disease in young kids, characterised by clinical signs such as depression, muscle weakness, lateral recumbency, a rolled-up belly and pain, along with typical biochemical changes in acid–base balance, urea and potassium levels.
> Urea concentrations and potassium can play a significant role in the aetiology of the disease.
> The prognosis depends on early diagnosis based on acid–base balance, and timely treatment includes the correction of metabolic acidosis.
> Preventive strategies can include monitoring maternal blood potassium concentrations before and after parturition, and frequent health monitoring of the kids.

Two healthy bucks were used for breeding through natural mating, which took place in autumn (September). The dams of all kids were clinically healthy. Spontaneous parturitions were supervised by veterinarians in an accredited stable. The monitored kids were born in early March. None of the kids were born by caesarean section, all kids were apparently clinically healthy at birth and consumed colostrum and milk from their mothers (Figure 1). Milk intake was not monitored; all kids suckled from their dams. All goats and their kids were housed under the same conditions, staying indoors on straw in a building with natural ventilation through windows.

Metabolic monitoring of the goats was carried out during the periparturient period. Blood samples were taken from the jugular vein to assess biochemical parameters. Blood samples from adult goats were collected 1 week before parturition, on the day of parturition, and 7 and 14 days after parturition. Blood samples from goat kids were collected at 2, 7 and 14 days of age. An outbreak of the disease occurred among the kids during the monitoring period, and additional blood samples were collected simultaneously from both sick and healthy kids to evaluate acid–base balance, electrolytes, haematocrit (Ht) and haemoglobin (Hb). Blood samples were also collected to monitor changes in acid–base balance during infusion treatment. This repeated investigation was conducted only on a selected number of kids ($n = 4$); L- and D-lactate levels were determined to confirm the diagnosis in four sick kids as well.

The kids were divided into two groups based on the presence of clinical signs: sick kids (group F, $n = 12$, with one kid dying, leaving $n = 11$) and healthy kids (group C, $n = 7$). The control group was established simultaneously (by randomly selecting animals of the same age (4–9 days) kept under the same conditions. The mothers were divided based on the health status of their kids: mothers of kids with floppy kid syndrome (group MF, $n = 8$) and mothers of kids without floppy kid syndrome (group MC, $n = 8$).

The kids with floppy kid syndrome came from both bucks, and the 11 monitored kids that received infusion therapy came from eight mothers. Seven of these mothers were in their second lactation period, and one was in its first lactation period. Ten kids came from multiple litters, while only one kid was a singleton. The affected kids included six males, four females and one hermaphrodite.

Our report presents the results from monitoring the mothers during their periparturient period and the kids during their first 14 days of life.

INVESTIGATIONS

The first clinical signs of the disease were observed in kids aged 4–9 days (two at 4 days, three at 6 days, four at 7 days, two at 8 days and one at 9 days). The body temperature of the sick kids ranged between 38.2°C and 39.7°C (mean 39.2°C), which is lower than the reference values (39.5°C–40.5°C).[11] The initial clinical signs (apathy, somnolence) were observed in the afternoon, and their condition deteriorated dramatically overnight. The kids did not have diarrhoea. The clinical signs of the affected kids included apathy, somnolence, muscle weakness, lateral recumbency, reluctance to move, rolled-up belly and signs of pain (groaning, teeth grinding). Some of the kids exhibited hypothermia, abnormal posture (dog-sitting position), and two showed clinical signs of pneumonia (cough and lung sounds).

Clinical tests (skin elasticity and position of the eyeballs) did not confirm dehydration, and the results of Hb and Ht between Group F and Group C did not differ significantly. Before the diagnosis was established (based on the acid–base balance), one 6-day-old female kid out of 12 died within a few hours. A postmortem examination and histopathological analysis were performed. Pulmonary hyperaemia and oedema, and dilation of the right heart ventricle were found. Liver and intestinal congestion were confirmed

TABLE 1 Selected blood parameters (mean ± standard deviation) of kids with (Group F, $n = 11$) and without floppy kid syndrome (Group C, $n = 7$) during diagnostic examination.

Parameters	Group F	Group C
pH	7.098 ± 0.06**	7.330 ± 0.02
BE (mmol/L)	−19.6 ± 4.1**	−0.5 ± 2.7
Anion gap (mmol/L)	21.8 ± 4.4**	10.1 ± 2.6
HCO_3- (mmol/L)	10.0 ± 3.2**	25.0 ± 2.9
pCO_2 (kPa)	5.0 ± 0.9**	6.7 ± 0.7
K^+ (mmol/L)	3.3 ± 0.5**	4.3 ± 0.3
Cl^- (mmol/L)	109.0 ± 2.5	108.5 ± 2.7
Hb (g/L)	88.1 ± 8.1	83.3 ± 12.2
Ht (%)	26.0 ± 2.3	24.4 ± 3.4

Abbreviations: BE, base excess; Cl^-, chloride; Hb, haemoglobin; HCO_3-, bicarbonate; Ht, haematocrit; K^+, potassium; pCO_2, partial pressure of carbon dioxide.
**$p < 0.001$ significant difference between groups.

TABLE 2 Individual concentration of L-lactate and D-lactate in the blood of some kids with floppy kid syndrome ($n = 4$).

L-lactate (mmol/L)	D-lactate (mmol/L)
1.8	6.4
3.45	4.86
2.2	6.45
2.71	5.78
2.54 ± 0.71[a]	5.87 ± 0.74[a]

[a]Mean ± standard deviation.

and kidneys were pale. Cardiac and skeletal muscles were also pale. Histopathological examination of the heart and skeletal (gluteal) muscle showed granular cardiomyocyte dystrophy and muscle fibre dystrophy. The affected gluteal muscle fibres had an eosinophilic and hyalinophilic appearance in the section. Hyperaemia of the lung and liver was also found. The other samples did not have pathological findings.

The final diagnosis was made based on the history, clinical examination of the animals and laboratory blood tests, specifically, the results of pH and other parameters of the acid–base balance (Table 1) and D-lactate concentration (Table 2). After the initial 12 kids, eight more kids in the herd became ill. In total, 20 out of 75 kids born in the herd showed clinical signs of floppy kid syndrome. Morbidity was 28.6% and mortality was 1.3%.

DIFFERENTIAL DIAGNOSIS

Kids exhibiting clinical signs such as central nervous system depression, muscle weakness, lateral recumbency, rolled-up belly and pain were considered to possibly have muscle or metabolic diseases. Other toxic or neurological origins could not be fully excluded.

TREATMENT

Sodium bicarbonate (4.2% solution; 500 mmol/L $NaHCO_3$) was diluted to a concentration of 1.3% (155 mmol/L $NaHCO_3$) and used for intravenous treatment of kids (Figure 2). The

FIGURE 2 Four- to 9-day-old kids without diarrhoea and temperature with apathy and muscular weakness, treated with bicarbonate infusion.

amount of bicarbonate was calculated using the following formula: bodyweight in kg × 0.5 × (− base excess) = mmol of sodium bicarbonate required.[6] Floppy kids were administered half of the calculated amount (25–56 mmol of sodium bicarbonate; 160–360 mL of 1.3% $NaHCO_3$) over 1–2 hours, depending on the total amount of solution. The acid–base balance was then checked and the next dose of sodium bicarbonate was calculated and administered based on the results. In most cases, after intravenous administration of sodium bicarbonate, there was a significant reduction in clinical signs (most kids responded well to the treatment). Intravenous treatment lasted for 1 day. Furthermore, the kids were concurrently treated with lactated Ringer's solution, 5% glucose solution and a supportive solution containing vitamins and essential amino acids (Duphalyte inj., Zoetis) intravenously. After completing intravenous therapy, further treatment consisted of oral administration of sodium bicarbonate (4.5 g dissolved in 10–20 mL of water at 3-hour intervals) and supplementary oral rehydration therapy using a commercially available product (Aqua Viva plv., Bioveta). The total treatment duration was 3–5 days. In addition to the above therapy, kids with signs of pneumonia received amoxicillin (70 mg/kg) and clavulanic acid (17 mg/kg) subcutaneously once daily for 5 days. All affected kids were temporarily removed from their mothers while clinical signs persisted and during intravenous therapy. The other eight kids that became ill in the herd were treated only orally due to early diagnosis and recognition of the disease with mild clinical signs (apathy, muscle weakness, but with preserved movement).

OUTCOME AND FOLLOW-UP

The data obtained were analysed using the unpaired t-test in the statistical programme (Excel 6.5). First, the f-test was used to detect variances between two files. Subsequently, significance was determined using the t-test considered at $p < 0.05$. Results in the tables are presented as means with standard deviation.

The acid–base balance values (pH, BE, anion gap, HCO_3) confirmed severe metabolic acidosis in kids with floppy kid syndrome. Statistical analysis revealed significant differences

TABLE 3 Selected blood parameters (mean ± standard deviation) in the blood of some kids with floppy kid syndrome ($n = 4$) during infusion therapy.

Parameters	Before treatment	After 1st infusion	After 2nd infusion
pH	7.091 ± 0.07	7.255 ± 0.06	7.291 ± 0.05
BE (mmol/L)	−17.45 ± 5.3	−11.0 ± 4.2	−6.9 ± 5.2
Anion gap (mmol/L)	20.8 ± 5.7	20.6 ± 5.9	18.2 ± 6.0
HCO_{3-} (mmol/L)	10.8 ± 3.5	15.5 ± 3.2	18.6 ± 3.7
pCO_2 (kPa)	4.5 ± 1.0	5.1 ± 1.2	5.6 ± 0.5
K^+ (mmol/L)	3.3 ± 0.6	3.0 ± 0.1	2.7 ± 0.2
Cl^- (mmol/L)	108.6 ± 4.5	108.3 ± 3.3	105.5 ± 5.8

TABLE 4 Selected blood parameters (mean ± standard deviation) of kids with (Group F, $n = 11$) and without floppy kid syndrome (Group C, $n = 7$) during the first 2 weeks of life.

	Glucose (mmol/L)		Urea (mmol/L)		GSH-Px (µkat/L)	
Kids age (days)	Group F	Group C	Group F	Group C	Group F	Group C
2	6.8 ± 1.5	7.1 ± 1.2	7.2 ± 2.6**	4.6 ± 1.2	306.2 ± 82.5	299.2 ± 72.4
7	5.2 ± 1.8	6.8 ± 1.2	6.7 ± 2.8**	2.7 ± 1.2	331.9 ± 71.5	314.9 ± 91.6
14	5.7 ± 1.4	5.6 ± 2.0	3.9 ± 0.9	3.9 ± 1.1	409.0 ± 114.0	323.4 ± 67.0

Abbreviation: GSH-Px, glutathione peroxidase.
**$p < 0.001$ significant difference between groups.

TABLE 5 The potassium concentration (mean ± standard deviation) in the blood of mothers of floppy kids (Group MF, $n = 8$) and mothers of healthy kids (Group MC, $n = 8$).

	K^+ (mmol/L)	
Dams	Group MF	Group MC
1 Week before delivery	4.5 ± 0.2	4.3 ± 0.4
Day of delivery	4.8 ± 0.5*	3.9 ± 0.9
7 Days after delivery	4.7 ± 0.6	4.1 ± 0.4
14 Days after delivery	4.8 ± 0.4*	3.8 ± 1.2

*$p < 0.05$ significant difference between groups.

($p < 0.001$) in pH, BE, anion gap, HCO_3, pCO_2 and potassium values between sick and healthy kids. Chloride levels did not show significant differences (Table 1). Increased concentration of D-lactate, shown in Table 2, confirmed the diagnosis of floppy kid syndrome. The development of pH values and other parameters before treatment and during infusion therapy is presented in Table 3. GSH-PX and glucose concentration were determined as part of the differential diagnosis (Table 4), and Hb and Ht values were used to evaluate dehydration (Table 1). Furthermore, statistically significant differences ($p < 0.001$) in urea concentrations were revealed in 2- and 7-day-old kids (Table 4), and significant differences ($p < 0.05$) were observed in maternal blood potassium concentrations on the day of delivery and 2 weeks after delivery (Table 5).

DISCUSSION

Kids are typically born healthy, and signs of floppy kid syndrome usually occur within the first 14 days of life.[1,7] In our study, the kids were also born healthy, with the first clinical signs manifesting between 4 and 9 days of age, consistent with other studies.[1,5,6] Floppy kid syndrome is an acute disease, where healthy, vital kids become severely ill within a few hours. If intensive treatment is not initiated promptly, the kids may die.[2,12] Postmortem examination of a deceased kid demonstrated renal damage and myopathy. Moreover, pulmonary oedema was found due to cardiovascular failure. No signs of aspiration pneumonia were found (the respiratory airway and alveolar tree were free of milk/without congestion).

Our initial diagnosis was not floppy kid syndrome because the acid–base balance of the kids was initially unknown. We ruled out white muscle disease and hypoglycaemia based on the enzyme activity of glutathione peroxidase in whole blood and blood glucose concentrations (Table 4). Dehydration was ruled out based on clinical examination and the results of Hb and Ht (Table 1), with no statistically significant differences ($p \geq 0.05$) between Group F and Group C. Infectious and non-infectious diseases, or metabolic diseases, can generally be distinguished based on the main indicators of health impairment (especially changes in body temperature and other signs related to organ damage, such as diarrhoea) in connection with individual infections. The body temperature of the sick kids ranged between 38.2°C and 39.7°C. We suggest that the moderate decrease in their body temperature was caused by reduced muscle activity, lying down and temporarily limited milk intake.

The results of the acid–base balance in this study agree with other findings.[2,12] The pH, BE, anion gap and HCO_3 values confirmed severe metabolic acidosis in kids. Chloride levels were within the normal reference range.[6] Lower pCO_2 values in kids with floppy kid syndrome indicated respiratory compensation for metabolic acidosis (Table 1), and during intravenous administration of sodium bicarbonate, pH values, BE, HCO_3 and pCO_2 gradually increased (Table 3). The main clinical manifestation of metabolic acidosis is central nervous system depression.[13] Factors other than simple deviation of acid–base balance can also contribute to clinical signs.[14] For example, azotaemia or hyperlactatemia should also be considered to be responsible for general health impairment and the appearance of clinical findings.[14] Although sensing acidosis can be an important somatosensory function in responses to ischaemia, inflammation and changes in metabolism, it is also a powerful factor in inducing pain.[15] In addition to neurological signs, other body systems can be affected.[13] Kidney and heart damage can occur due to over-acidification of the body. Increased blood urea concentration is an indicator of renal failure[2]; however, our results showed an increased concentration of urea in kids with floppy kid syndrome compared to healthy kids even before the onset of clinical signs (Table 4). Presented results suggest that the increase in urea concentration in the blood of kids before clinical signs is not caused by acidosis-induced renal damage, but may be related to excessive protein (N-substances) intake with increased colostrum/milk intake. Thus, this finding supports the theory that colostrum/milk overfeeding plays a role in floppy kid syndrome.[7]

Other clinical signs observed included abnormal postures, such as lateral recumbency and dog sitting position. While abnormal postures are not necessarily indicative of disease, when associated with other signs, they can indicate the location and severity of the disease process.[16] Two kids showed signs of pneumonia (cough and lung sounds). Pneumonia in some kids with floppy kid syndrome has also been reported in another study.[2] Besides floppy kid syndrome, we considered aspiration pneumonia in these two cases, which may be caused by the involvement of the lungs by a liquid diet due to impairment of the swallowing muscles, but clinical signs were not sufficient to confirm aspiration pneumonia.

D-lactate concentrations were determined to confirm the diagnosis in four sick kids. In humans, D-lactic acidosis with clinical signs occurs at concentrations greater than 3 mmol/L,[17] consistent with our results (mean 5.87 mmol/L) and with the results of another study.[2]

Moreover, all the affected kids were hypokalaemic. Hypokalaemia in kids with metabolic acidosis in this case should be due to potassium deficiency, as during metabolic acidosis, there is usually an exchange of K^+ from cells for H^+ from blood plasma, increasing the concentration of potassium in the blood plasma. Hypokalaemia refers to a lower blood potassium level (below 3.5 mmol/L).[18,19] Potassium is essential for normal acid–base balance and is an integral part of many enzymatic pathways, functioning as an intracellular cation.[20] This is fundamental for cell functions, the formation of resting membrane potential, nerve impulse transmission and muscle contraction.[21] Other cases have also shown lower potassium levels in floppy kids,[1,2] although hypokalaemia was not demonstrated (except in one case) in another study.[12] Maternal blood potassium concentrations were within the reference range, although significant differences were observed between mothers of kids with floppy kid syndrome and mothers of healthy kids. The lower potassium content in the blood of mothers of healthy kids can be considered physiological, possibly related to higher secretion of potassium into the colostrum. Therefore, we predict that healthy kids (of these mothers) could receive sufficient potassium through colostrum, whereas the kids who became ill probably did not receive sufficient potassium.

Although clear prevention strategies are sparse in the literature, it is recommended to prevent floppy kid syndrome by separating neonate kids from their dams and rearing them with bovine colostrum/milk, although this treatment is laborious and time-intensive.[8] We recommend that breeders monitor the health of the kids regularly and frequently, at least three to four times a day for the first 10 days, so that even clinical signs (apathy, muscle weakness, but with preserved movement) can be detected and easily treated with early oral treatment and temporary weaning of the goats. Early detection of clinical signs (apathy, muscle weakness, but with preserved movement) and acid–base balance results are essential for successful treatment, reducing treatment costs and mortality. Based on our results, we also recommend monitoring maternal blood potassium concentration before and after parturition. If maternal blood potassium levels do not decrease after delivery, insufficient potassium concentration in the colostrum and subsequently in the kids' blood can be considered a risk factor (predisposing) for the possible development of the disease. Our findings on the dynamics of maternal blood potassium concentrations after birth are also noteworthy. Therefore, we can consider the lack of potassium in the blood of young kids as another hypothetical factor in the aetiology of floppy kid syndrome. However, the discussed results should be seen as hypotheses requiring further investigation, as the relationship between serum potassium concentration values and the occurrence of the disease in goats requires a larger sample size.

According to our retrospective study, floppy kid syndrome can be described as a non-communicable disease characterised by metabolic acidosis in kids under 10 days of age, accompanied by muscle weakness and pain. Several kids in the herd can be affected simultaneously, and they can be hypokalaemic. As potassium is an intracellular element required by striated and smooth muscles for function, its deficit may lead to intestinal smooth muscle motility disorders, resulting in stagnation of the milk volume and possible multiplication of *Clostridium perfringens*, with subsequent toxin production and excessive production of organic acids in the form of D-lactate. Thus, dietary errors, colostrum overfeeding or gastrointestinal tract motility disorders can cause clostridial overgrowth.

AUTHOR CONTRIBUTIONS

Lenka Kudělková: Veterinary care of the goat herd; diagnosed and treated the disease; wrote the article. **Ľubica Wittke**: Veterinary care of the goat herd; collected blood samples; diagnosed and treated the disease. **Leoš Pavlata**: Designed the project; diagnosed and interpreted results. **Alena Pechová**: Performed statistical analysis; proofread the text. **Kateřina Hauptmanová**: Proofread the text and references.

ACKNOWLEDGEMENTS

The authors thank the head of the Department of Animal Nutrition and Forage for making the publication possible.

CONFLICT OF INTEREST STATEMENT

The authors declare that they have no conflicts of interest.

FUNDING INFORMATION

This study was partially supported (collection of clinical data and laboratory results, including costs associated with pathological examination) by the Research Project of the Ministry of Education and Culture of the Czech Republic (No. MSM6215712403).

ETHICS STATEMENT

The authors confirm that they have adhered to the ethical policies of the journal, as noted on the journal's author guidelines page. Ethical approval is not applicable, as this report describes a clinical case.

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How to cite this article: Kudělková L, Wittke Ľ, Pechová A, Hauptmanová K, Pavlata L. Biochemical changes in the aetiology of floppy kid syndrome in goat kids. Vet Rec Case Rep. 2025;13:e1014. https://doi.org/10.1002/vrc2.1014

IMAGE QUIZ

Figure 2. Kids aged 4–9 days without diarrhoea and temperature with apathy and muscular weakness, treated with bicarbonate infusion.

MULTIPLE-CHOICE QUESTION

What is the likely cause/diagnosis of the condition of these kids with respect to treatment with bicarbonate infusion?

POSSIBLE ANSWERS TO MULTIPLE-CHOICE QUESTION

A. Metabolic alkalosis
B. Metabolic acidosis/floppy kid syndrome
C. Hypoglycaemia and hypothermia
D. Nutrition muscular dystrophy

CORRECT ANSWER

B. Metabolic acidosis/floppy kid syndrome.

Possible diagnosis based on anamnestic data: age of kids, clinical signs and bicarbonate treatment.