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ANION GAP IN DIARRHOEIC CALVES WITH METABOLIC ACIDOSIS

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ABSTRACT: **This study was carried out on seventy calves (70), fifty five (55) were diarrhoeic calves and fifteen (15) clinically healthy calves. The affected calves were suffering from general malaise, poor appetite, dehydration and metabolic acidosis.**

Acidotic calves were divided according to the severity of clinical signs and biochemical data into two groups. The first group (32 calves) unable to stand up, severely acidotic with high anion gap, hyperchloraemia, hyperkalaemia, hyponatraemia. The second group (23 calves) with mild to moderate acidotic calves able to stand with normal anion gap, hyperchloraemia, hyponatraemia, normokalaemia. In all the acidotic calves the urea concentration was highly significantly increased.

All acidotic calves received electrolyte solution with bicarbonate intravenously to correct the blood acidosis and acid base disturbance as well as fluid loss during the undifferentiated diarrhoea and rehydration.

INTRODUCTION

Diarrhoea means a symptom of gastrointestinal diseases of different causes which lead to change in the consistency of faeces increasing its fluidity and its frequent passage **(Sadiek and Schlerka, 1995)**. Diarrhoea leads to mortality in young calves where it is associated with the major metabolic disturbances including dehydration, electrolyte loss and metabolic acidosis **(Groutides and Mitchell, 1990a, Grove-White and White, 1993)**.

Metabolic acidosis develops in the diarrhoeic calf as a result to the loss of bicarbonate via intestinal tract or excessive loss of saliva or a gain of acid by the body such as found in cases of grain overload, lactic acidosis and acetonaemia **(Howard, 1981)**. In addition, reduced tissue perfusion may result in lactic acid

production **(Naylor, 1987)**, reduced renal perfusion and reduced excretion of hydrogen ions exacerbate the acidosis **(Michell et al., 1992)**.

Diagnosis of different causes of metabolic acidosis had been made by calculating the anion gap **(George, 1986; DiBartola, 1992a, Halperin and Goldstein, 1994)**. The anion gap can be calculated as the difference between the sum of major cation (sodium + potassium) and the measured anions (chloride + bicarbonate). It can be calculated by using the formula: Anion gap = (sodium + potassium) – (chloride + bicarbonate) **(Carlson, 1989)**. The anion gap for most species of domestic animals appears to be similar to that defined for human with approximate value of 10 – 20 mEq/L = 10 – 20 mmol/L **(Carlson, 1989, Garry and Rings, 1987)**.

Anion gap gives the concentration of anions that are not normally measured such as sulphates, phosphates, plasma proteins, lactate and citrate **(Stocker et al., 1999b)**.

Two principal types of metabolic acidosis had been recognized, one with a normal or low anion gap, or hyperchloraemic metabolic acidosis and the other with a high anion gap, or normochloraemic metabolic acidosis **(Malley, 1990)**.

The causes of metabolic acidosis with a normal anion gap include the loss of bicarbonate ions through the intestine and dilutional acidosis. Metabolic acidosis with a high anion gap may result from an accumulation of lactic acid or ketone bodies or from uraemia **(Emmett and Narins, 1977, DiBartola, 1992a)**.

This study was designed to investigate the type and causes of metabolic acidosis by calculation of anion gap in diarrhoeic calves.

MATERIALS AND METHODS

I. Materials

a. Calves

This study was carried out on seventy calves of different breeds, three to six months of age. The calves were examined between August 1998 to February 1999. Fifty five (55) calves were suffering from mild, moderate to severe diarrhoea, general malaise, dehydration and depression. The rest (fifteen 15) were apparently healthy calves and used as a control group.

Inspected diarrhoeic calves were divided into two groups, the first group was recumbent (unable to stand). The second group was depressed (reluctant to stand).

b. Samples:

Three venous blood samples were collected from all investigated calves. One anaerobically obtained into heparinized plastic syringe by jugular venipuncture, the syringes were sealed, placed in an ice bag and were analyzed as soon as possible within 15 – 30 minutes for pH, blood gas values and bicarbonate

concentrations (acid – base determination). The second blood sample was collected with EDTA as anticoagulant for haematocrit % reading. The third one was collected for harvesting serum to biochemical analysis.

c. Treatment:

All diarrhoeic calves were given intravenous fluids. The fluids were prepared by adding 32.2 gm sodium chloride, 14.7 sodium bicarbonate and 1.5 gm potassium chloride to 5 liters of water to produce a solution with ionic concentrations of 144 mmol/liter sodium, 4 mmol/liter potassium, 113 mmol/liter liter chloride and 35 mmol/liter bicarbonate. Molar (8.4 percent) sodium bicarbonate solution was made by adding 33.6 gm to 400 ml of purified water to correct the acidosis in group two **(Grove and White, 1993)**. The rate of administration of fluid and the total quantity given was governed by the calves' clinical response. The calves received 5 – 10 liters over a period of 36 – 48 hours.

II. Methods:

An estimated blood gas analyser (ABL30 – Acid – Base analyzer, Radiometer, Copenhagen, Denmark) was used to measure blood pH, PCO_2 and HCO^-_3 values **(Ward et al., 1994)**. Haematocrit % was estimated according to **Schalm (1975)**, while serum concentrations of sodium, potassium, chloride and urea were measured by using spectrophotometric analyses with commercially available test kits supplied by HITECH laboratory, France according to **Tinder (1951), Terri and Sesin (1958), Schoenfeld (1964)** and **Fawcett and Scott (1960)**.

Statistical analysis:

The obtained data were statistically analysed according to **Feldman et al. (1994).**

RESULTS

I. Clinical findings

Investigated calves were suffering from different degrees of diarrhoea, thirty two (32) were severely diarrhoeic and unable to stand, while, twenty three (23) were mildly to moderately diarrhoeic and were able to stand. These affected calves had varying degrees of appetite, 17 of them were suffering from anorexia, 38 with mild to moderate appetite (Table 1).

All these diarrhoeic calves were dehydrated, the dehydration was assessed clinically by the appearance of sunken eyes, elasticity test which varied from normal to decreased, and by the measurement of haematocrit % in Table (2).

The diarrhoeic selected calves were acidotic, the severity of acidosis was detected by clinical, biochemical and measuring the pH value.

II. Biochemical analysis

Obtained biochemical data are summarized in Table (2) that showed a comparison of relevant laboratory finding in acidotic calves that were unable to stand up (group 1) and those that could stand up with non-diarrhoeic control calves (group 2).

Table (1): Clinical findings in acidotic calves that were unable to stand up and those that could stand up.

Variable	Unable to stand up (n=32) severely acidotic	Able to stand up (n=23) mildly to moderately acidotic
Appetite		
- Absent	14	3
- Moderate to mild	18	20
General condition	Recumbent	Depressed and still standing
Skin fold test persists for (sec)	20 – 45	6 - 10
Eyeball	Moderate to severe sunken	Mild sunken
Diarrhoea	Severe to very severe	Mild to moderate

Sec = seconds

Table (2): Comparison of relevant laboratory finding in acidotic calves that were unable to stand up and those that could stand up with non-diarrhoeic control calves (mean ± standard errors).

Variable	Control (n=15)	Unable to stand up (n=32) severely acidotic	Able to stand up (n=23) mildly to moderately acidotic
Blood pH	7.36 ± 0.03	6.71 ± 1.03**	6.84 ± 1.51*
Haematocrit %	33.6 ± 1.01	48.3 ± 1.5**	43.1 ± 1.28**
PCO_2 (mm Hg)	39.6 ± 1.70	29.5 ± 1.01**	34.8 ± 1.32*
HCO_3^- (mEq/L)	22.1 ± 1.20	7.6 ± 1.32**	8.9 ± 1.01**
Sodium (mmol/L)	141.4 ± 1.30	135.6 ± 1.02**	137.2 ± 1.29**
Potassium (mmol/L)	4.9 ± 1.01	5.2 ± 1.05*	4.8 ± 1.82
Chloride (mmol/L)	96.0 ± 1.51	101.4 ± 1.22*	101.1 ± 1.52*
Anion gap (Ag mmol/l)	28.2 ± 1.10	32.1 ± 1.12**	28.6 ± 1.13
Urea (mmol/L)	3.3 ± 0.88	8.3 ± 1.04**	6.7 ± 1.06**

SE = Standard error.

* P<0.05 (Significant as compared with control calves).

** P<0.01 (Highly significant as compared with control calves).

PCO_2 = Partial pressure of carbon dioxide.

HCO_3^- = Bicarbonate concentration.

DISCUSSION

Despite the ease with which diarrhoeic and dehydrated calves could be identified, no single clinical sign, detail of history or combination of findings were a useful predictor of the degree of acidosis. From recorded results in Tables (1 and 2), the dehydration was cleared by the significant increase in haematocrit % and also by the signs appeared in diarrhoeic calves. All diseased calves had a metabolic acidosis whereas the blood pH was 6.71 and 6.84 in the acidotic range. Clinical and laboratory data showed that there were thirty two (32) calves were unable to stand and their appetite varied from anorexia to mild-moderate appetite with observed sunken eye, while in twenty three (23) calves that could able to stand these clinical signs were less in its severity than those were unable to stand. These clinical observations were common in acidotic calves **(Stocker et al., 1999a)**. These acidotic calves attempted to compensate the metabolic acidosis by the elimination of carbon dioxide CO_2 in the lungs, as indicated by reduction in partial pressure of carbon dioxide (PCO_2) and bicarbonate concentration **(Halperin and Goldstein, 1994)**. This reduction in bicarbonate concentration in diarrhoeic ones was due to loss of bicarbonate in diarrhoea through great loss of faeces from one side and through the consumption of bicarbonate in buffering of increased H^+ ion in the blood **(Sadiek and Schlerka, 1995)**.

The calculation of the anion gap helps to analyse the causes of metabolic acidosis which has cleared in Table (2) where in the thirty, unable to stand calves, its value was highly significant increased when compared with normal control value. Also, there were highly significant decrease in HCO_3^- concentration with significant hyponatraemia, significant hyperkalaemia and hyperchloraemia.

There was also highly significant increase in urea concentration in all diarrhoeic. The previous data showed that there were high anion gap metabolic acidosis with hyperchloraemia which may result from accumulation of lactic acid or ketone bodies or from uraemia. This uraemia was obvious in present results **(Emmett and Narins, 1977)**. On the other hand, in Table (2), the twenty three diarrhoeic calves, the anion gap was within normal value (28.6) and there was highly significant decrease in both HCO_3^- and sodium while potassium level was within the normal value. Also, there was significant hyperchloraemia with highly significant increase in the urea concentration. It was found from this data, that there was metabolic acidosis with normal anion gap because of the loss of bicarbonate ions through the intestine **(DiBartola, 1992a)**.

From recorded results it appears that the normal anion gap with hyperchloraemic acidosis was due to a loss of bicarbonate through the intestine while highly anion gap metabolic acidosis was caused by loss of bicarbonate and unidentified anions. These unidentified anions require further study.

In the light of these results about the anion gap, the calves with high anion gap metabolic acidosis, it is recommended that the bicarbonate should be administered in sufficient amount as mentioned in the treatment to increase the blood pH to the normal value in severely acidotic calves **(De Morais, 1997)** to avoid the development of late metabolic alkalosis.

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