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Effect of antimicrobials on haemato-biochemical profile in *Escherichia coli* (*E. coli*) associated diarrhoeic calves

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Abstract
The present investigation was conducted to study the effect of Antimicrobials on haematological parameter and biochemical parameter of *Escherichia coli* (*E. coli*) affected diarrhoeic calves. A total of 153 faecal swabs were screened from various cattle farms located in and around Guwahati of which, 78 samples showed positive for *E. coli*. Isolates were identified based on morphological and cultural characteristics from which total of 18 *E. coli* affected diarrhoeic calves selected and divided into 3 groups comprising 6 animals in each i.e., T1 – Consisted of 6 animals and were treated with ciprofloxacin at the dose rate of 10 mg/kg body weight orally twice daily for 5 days. T2 – Consisted of 6 animals and were treated with norfloxacin at the dose rate of 25 mg/kg body weight orally twice daily for 5 days. T3 – Consisted of 6 animals and were treated with ofloxacin at the dose rate of 20 mg/kg body weight orally twice daily for 5 days. C – Apparently healthy control (No treatment given). About 6 ml of whole blood was collected from diarrhoea affected calves by puncturing the jugular vein. Blood was collected on 0 day (pre-treatment), 5th day and 10th day (post treatment). Blood was analyzed for haematological parameter *viz*., Haemoglobin (Hb), Packed Cell Volume (PCV), Total Erythrocyte count (TEC) and Total leuckocyte count (TLC), Biochemical parameter like Total serum protein (TSP), Glucose, Sodium (Na^+), Potassium (K^+) and Chloride (Cl^-). Haemato-biochemical parameter in treatment trial revealed significant increase in Hb, PCV, TEC, TLC, TSP, Cl^- and decrease in glucose and Na^+ in diarrhoeic calves in comparison to apparently healthy control.

Keywords: Haematological parameter, biochemical parameter, *E. coli*, diarrhoea, calves

1. Introduction
Livestock is the integral part of the mixed-farming system that characterized agriculture in Assam. Besides contributing to food and crop production, livestock and poultry are important source of livelihood. Being a state with limited benefits of green revolution technologies and climatic uncertainties, livestock has the potential to contribute to farm diversification and intensification. Livestock products are integral parts of local diet as more than 95 percent of the population is non-vegetarian (Barbaruah, 2012) [1]. White revolution will be meaningless if the calf dies in great number as calf is the backbone of dairy industry (Malik *et al.*, 2013) [2]. New born animals suffer fairly higher mortality than their adult counterparts and it is one of major impact over economy in livestock industry (Radostitset *et al.*, 2000) [3]. Calf diarrhoea in farm animals, especially in neonatal calves is one of the most challenging clinical signs encountered by large animal veterinary practitioner (*Central Agricultural Census Commission*, 2003) [4]. Diarrhoea is a leading cause of economic losses to the cattle industry and major cause of calf mortality and morbidity during first few weeks of life (Radostits*et al*. 2000) [3]. Calf diarrhoea is a multi-factorial disease complex characterized by increased frequency, fluidity or volume of faecal excretion in calves due to excessive osmotic pressure in the intestine, intestinal damage caused by several organisms leading to malabsorption, toxins produced by organisms or excessive contractions of the intestine (Lorenz, 2006) [5]. The cardinal haemeto-biochemical features of calf diarrhoea are higher packed cell volume, leukopenia, lymphopenia, hyponatraemia, pre-renal failure and metabolic acidosis (Alsaad *et al.*, 2012) [6].

2. Materials and Methods
The present study was carried out at the Department of Veterinary Epidemiology & Preventive Medicine, College of Veterinary Science, Assam Agricultural University, Khanapara, Guwahati-781022 in collaboration with Department of Veterinary Microbiology, College of

Veterinary Science, Assam Agricultural University, Khanapara, Guwahati-781022 during the period from August-2019 to December-2020.

Treatment regime of diarrheic calves with antimicrobials was as follows: T1 – Consisted of 6 animals and were treated with ciprofloxacin at the dose rate of 10 mg/kg body weight orally twice daily for 5 days. T2 – Consisted of 6 animals and were treated with norfloxacin at the dose rate of 25 mg/kg body weight orally twice daily for 5 days. T3 – Consisted of 6 animals and were treated with ofloxacin at the dose rate of 20 mg/kg body weight orally twice daily for 5 days. C – Apparently healthy control (No treatment given).

About 6 ml of whole blood was collected from diarrhoea affected calves by puncturing the jugular vein. A total of 18 diarrhoeic calves were divided into 3 groups comprising 6 animals in each *i.e.* T1, T2 and T3 and 6 apparently healthy calves were kept as control group. Blood was collected on 0 day (pre-treatment), 5th day and 10th day (post treatment).

About 2.5 ml of blood was collected into K_3 EDTA vacutainer from the diarrhoeic calves. Blood samples were immediately brought to the laboratory for routine haematological examination with automated haematological cell counter, manufactured by Melet Schloesing Lab (France) MS4e model (MSAS). The following haematological parameters were determined: Haemoglobin (g/dL), Packed Cell Volume (PCV), Total Erythrocyte Count (TEC) and Total Leucocyte Count (TLC).

Remaining 3.5 ml of blood was collected in clot activator vacutainer and allowed to clot and then centrifuged at 3000 rpm for 15 minutes and serum was separated and transferred into an Eppendorf tube for further processing. Serum was used for analysis of Total serum protein, Glucose, Sodium, Potassium and chloride by spectrophotometric method using commercial kit as per standard protocol supplied by the manufacturer.

The statistical analysis of the data was carried out according to the standard statistical procedure using Critical difference test.

3. Results

3.1 Haematological profile

The results for haematological profile and biochemical profile parameters of T1, T2 and T3 and control are mentioned in table 1 and 2 respectively.

The mean±SE values of haemoglobin (g/dl) on day 0, 5th and 10th were recorded as 12.43±0.32, 12.22±0.29 and 11.60±0.42 in T1, 12.23±0.29, 12.03±0.30 and 11.90±0.34 in T2 and 12.37±0.33, 12.18±0.26 and 11.88±0.35 in T3, 11.20±0.29, 11.26±0.34 and 11.27±0.34 in control group, respectively.

The mean ± SE values of packed cell volume (%) on day 0, 5th and 10th were recorded as 42.60±0.44, 41.60±0.25 and 42.14±0.46 in T1, 40.97±0.49, 40.83±1.16 and 41.10±0.42 in T2 and 38.05±0.40, 39.23±0.40 and 39.89±0.32 in T3 and 37.04±0.24, 36.92±0.26, 37.01±0.44 in control group, respectively.

Table 1: Haematological profile of T1, T2 and T3 and control calves

	Periods	Groups			
		T1 (n=6)	T2 (n=6)	T3 (n=6)	C (n=6)
Hb (g/dl)	0 Day	12.43±0.32^{a}	12.23±0.29^{a}	12.37±0.33^{a}	11.20±0.29^{b}
	5th Day	12.22±0.29^{a}	12.03±0.30^{a}	12.18±0.26^{ab}	11.26±0.34^{b}
	10th Day	11.60±0.42^{a}	11.90±0.34^{a}	11.88±0.35^{a}	11.27±0.34^{a}
PCV (%)	0 Day	42.60±0.44^{Aa}	41.60±0.25^{Aa}	42.14±0.46^{Aa}	37.04±0.24^{Ab}
	5th Day	40.97±0.49^{Ba}	40.83±1.16^{ABa}	41.10±0.42^{ABa}	36.92±0.26^{Ab}
	10th Day	38.05±0.40^{Cac}	39.23±0.40^{Ba}	39.89±0.32^{Bb}	37.01±0.44^{Ac}
TEC (10^6 /cu mm)	0 Day	8.42±0.16^{Aa}	8.69±0.03^{Ab}	8.39±0.02^{Aa}	7.92±0.02^{Ac}
	5th Day	8.32±0.02^{Aa}	8.39±0.03^{Ba}	8.27±0.02^{Aa}	7.73±0.02^{Ab}
	10th Day	7.98±0.16^{Bac}	8.16±0.03^{Cab}	8.26±0.09^{Ab}	7.89±0.02^{Ac}
TLC (10^3 /cu mm)	0 Day	12.59±0.07^{Aa}	12.58±0.05^{Aa}	12.07±0.05^{Ab}	8.71±0.07^{ABc}
	5th Day	11.28±0.07^{Ba}	11.42±0.05^{Ba}	11.37±0.07^{Ba}	8.63±0.07^{Ab}
	10th Day	9.01±0.07^{Cac}	10.25±0.07^{Cb}	10.05±0.19^{Cb}	8.91±0.07^{Bc}

Means with different superscripts within a column (capital letter) and within a row (small letter) differ significantly ($p<0.01$)

The mean ± SE values of total erythrocyte count (10^6/cubic mm) on day 0, 5th and 10th were recorded as 8.42±0.16, 8.32±0.02 and 7.98±0.16 in T1, 8.69±0.03, 8.39±0.03 and 8.16±0.03 in T2 and 8.39±0.02, 8.27±0.02 and 8.26±0.09 in T3, 7.92±0.02, 7.73±0.02 and 8.26±0.09 in control group, respectively.

The Mean ± SE values of total leucocyte count (10^3/cubic mm) on day 0 (pre-treatment), day 5th and 10th (post-treatment) were recorded as 12.59±0.07, 11.28±0.07 and 9.01±0.07 in T1, 12.58±0.05, 11.42±0.05 and 10.25±0.07 in T2, 12.07±0.05, 11.37±0.07 and 10.05±0.19 in T3, 8.71±0.07, 8.63±0.07 and 8.91±0.07 in control group, respectively.

3.2 Biochemical profile

The Mean ± SE values of total serum protein (g/dl) on day 0 (pre-treatment), day 5th and day 10th (post-treatment) were recorded as 7.52±0.09, 7.41±0.05 and 7.08±0.08 in T1, 7.43±0.09, 7.39±0.08 and 7.31±0.07 in T2, 12.07±0.05, 11.37±0.07 and 10.05±0.19 in T3 and 7.60±0.07, 7.40±0.04 and 6.83±0.07 in control group, respectively.

The Mean ± SE values of glucose (mg/dl) on day 0 (pre-treatment), day 5th and day 10th (post-treatment) were recorded as 62.60±0.19, 67.08±0.54 and 74.21±0.60 in T1, 63.87±0.76, 65.80±0.72 and 72.65±0.60 in T2, 63.00±0.47, 65.83±0.88 and 73.69±0.58 in T3 and 78.53±0.22, 79.11±0.54 and 78.62±0.24 in control group, respectively.

The mean ± SE values of Na^+ (mmol/l) on day 0, 5th and 10th were recorded as 124.93±0.88, 127.95±0.83 and 139.73±0.87 in T1, 125.01±0.82, 127.99±0.73 and 135.70±0.77 in T2, 124.90±0.93, 126.78±0.88 and 137.60±0.78 in T3 and 143.25±1.04, 142.02±1.04 and 143.88±1.05 in control group, respectively.

The mean ± SE values of K^+ (mmol/L) on day 0, 5th and 10th were recorded as 5.16±0.14, 5.08±0.11 and 4.93±0.05 in T1, 5.00±0.22, 4.91±0.16 and 4.89±0.16 in T2, 5.04±0.18, 4.82±0.20 and 4.80±0.19 in T3 and 4.81±0.07, 4.73±0.06 and 4.79±0.07 in control group, respectively.

Table 2: Biochemical profile of T1, T2 and T3 and control calves

	Periods	Groups			
		T1 (n=6)	T2 (n=6)	T3 (n=6)	C (n=6)
TSP (g/dl)	0 Day	7.52 ± 0.09^{Aa}	7.43 ± 0.09^{Ab}	7.60 ± 0.07^{Ab}	6.92 ± 0.10^{Ac}
	5th Day	7.41 ± 0.05^{Aa}	7.39 ± 0.08^{Aa}	7.40 ± 0.04^{Aa}	6.88 ± 0.07^{Ab}
	10th Day	7.08 ± 0.08^{Ba}	7.31 ± 0.07^{Ab}	7.16 ± 0.08^{Ba}	6.83 ± 0.07^{Ac}
Glucose (mg/dl)	0 Day	62.60 ± 0.19^{Aa}	63.87 ± 0.76^{Aa}	63.00 ± 0.47^{Aa}	78.53 ± 0.22^{Ab}
	5th Day	67.08 ± 0.54^{Ba}	65.80 ± 0.72^{Bb}	65.83 ± 0.88^{Bb}	79.11 ± 0.54^{Ac}
	10th Day	74.21 ± 0.60^{Ca}	72.65 ± 0.60^{Cb}	73.69 ± 0.58^{Cb}	78.62 ± 0.24^{Ac}
Na^+ (mmol/L)	0 Day	124.93 ± 0.88^{Aa}	125.01 ± 0.82^{Aa}	124.90 ± 0.93^{Aa}	143.25 ± 1.04^{Ab}
	5th Day	127.95 ± 0.83^{Ba}	127.99 ± 0.73^{Ba}	126.78 ± 0.88^{Aa}	142.02 ± 1.04^{Ab}
	10th Day	139.73 ± 0.87^{Ca}	135.70 ± 0.77^{Cb}	137.60 ± 0.78^{Bab}	143.88 ± 1.05^{Ac}
K^+(mmol/L)	0 Day	5.16±0.14	5.00±0.22	5.04±0.18	4.81±0.07
	5th Day	5.08±0.11	4.91±0.16	4.82±0.20	4.73±0.06
	10thDay	4.93±0.05	4.89±0.16	4.80±0.19	4.79±0.07
Cl^-(mmol/L)	0 Day	104.20 ± 0.71^{Aa}	104.08 ± 0.76^{Aa}	103.93 ± 0.76^{Aa}	99.08 ± 0.47^{Ab}
	5th Day	103.68 ± 0.64^{Aa}	103.66 ± 0.67^{ABa}	103.79 ± 0.62^{Aa}	100.04 ± 0.59^{Ab}
	10thDay	101.07 ± 0.34^{Bab}	$102.89\pm0.29B^{a}$	102.23 ± 0.27^{Aab}	100.33 ± 1.30^{Ab}

Means with different superscripts within a column (capital letter) and within a row (small letter) differ significantly ($p<0.01$)

The mean± SE values of Cl^- (mmol/L) on day 0, 5th and 10th were recorded as 104.20±0.71, 103.68±0.64 and 101.07±0.34 in T1, 104.08±0.76, 103.66±0.67 and 102.23±0.27 in T2, 103.93±0.76, 103.79±0.62 and 102.23±0.27 in T3 and 99.08±0.47, 100.04±0.59 and 100.33±1.30 in control group, respectively.

4. Discussion

4.1 Haematological profile

The increased haemoglobin concentration in present study might be due to reduction in plasma volume and haemoconcentration due to loss of extracellular fluid in diarrhoeic faeces (Fisher., 1965 and Tennant *et al*., 1972) [7, 8] Resulting increase in the value of haemoglobin in diarrhoeic calves were in agreement with observations of Baruah *et al.* (1979) [9] who reported an increase in haemoglobin concentration in diarrhoeic calves. The higher level of Hb in *E. coli* affected groups could be due to haemoconcentration associated with dehydration and hypovolemia, as diarrhoea lead to excess loss of intestinal fluid resulting in severe dehydration.

The Increased PCV values in colibacillosis affected calves might be due to the haemo-concentration associated with dehydration and hypovolemia and also reduction in the plasma volume and haemoconcentration due to loss of extracellular fluid in diarrhoeic faeces (Roy *et. al.*, 2009 and Kumar *et. al.*, 2010) [10, 11].

The increased TEC values in diarrhoeic calves were in agreement with earlier observations of Baruah *et al.*, (1979) [9] who reported an increase in TEC value in diarrhoeic calves ranging from 4.80 to 8.00 million/mm^3. Kumar *et al.* (1981) [12] reported significant increase in TEC value in calves suffering from calf scour. Sridhar *et al.* (1988) [13] also observed increased in TEC value in scouring calves as compared to normal value. However, higher TEC values than the present study was noticed by Bali *et al*., (2000) [14] who reported significant increase in mean TEC values before treatment than normal value and that reduced after 96 hours of treatment which was also slightly more than that of healthy controls.

In diarrhoeic calves, significant leukocytosis might have occurred due to normal reaction of body defense mechanism against infections as well as haemoconcentration due to dehydration. Present findings are in accordance with Bali *et al*., (1999), Kumar and Mandial (2000), Boyd *et al*. (1974) and Sridhar *et al*. (1988) [15, 16, 17, 13]. They also observed significantly ($p<0.01$) higher mean value of TLC of colibacillosis affected diarrhoeic calves as compared to control. Similar finding was also reported by Bashir *et al.* (2015) and Shekhar *et al.* (2017) [18, 19].

4.2 Biochemical profile

The marked increases in total serum protein in diarrhoeic calves might be due to associated dehydration and general unthriftines which may affect hepatic parenchyma resulting in the failure of the liver for protein synthesis. (Tawfik, *et. al.*, 2004) [20]. The apparently higher values of total serum protein (TSP) in diarrhoeic calves as compared to the healthy control group was in conformity with earlier workers such as Phullan (2004), Ghanem (2012), Gupta (2020) and Eddy (2004) [21, 22, 23, 24]. The increase in total serum proteins may be due to release of intracellular protein from damaged tissues.

Marked decrease in serum glucose values were observed in *E. coli* associated diarrhoeic calves in the present investigation. Hypoglycemia in colibacillosis might be due to anorexia, decreased intestinal absorption of glucose and reduced rate of conversion of lactic acid to glucose (Morris *et al*., 1985) [25]. Similar findings were reported by several workers (Groutides and Michell, 1990 and Asati *et al*., 2010, Himanshu and Pal, 2015) [26, 27, 28], who recorded hypoglycemia in colibacillosis calves. The noticeable decrease in the level of serum glucose in diarrhoeic calves as compared to healthy control calves is in close agreement with Lewis *et al*., (1975) [29]. Hypoglycaemia may occur as a result of reduced rate of conversion of lactic acid to glucose.

A significant fall in the level of serum sodium in scouring calves was recorded in comparison to normal healthy calves. Hyponatraemia might be due to result of excessive secretion of the sodium ions by intestinal villus cells which are lost through the intestinal tract particularly in enterotoxigenic *E. coli* induced diarrhoea. It is in agreement with the finding of the few workers (Sridhar *et al*., 1988; Groutides and Michell, 1990; Bali *et al*., 2000; Himanshu and Pal, 2015) [13, 26, 14, 28] who also recorded hyponatreamia in diarrhoeic calves.

In the present study, non-significant increase in the serum potassium level in diarrhoeic calves in compared to healthy calves was observed. Hyperkalaemia might be due to increased retention of K^+ ion by kidney and increased tubular reabsorption of K^+ ion in response to metabolic acidosis, which might have induced translocation of potassium ion

from intracellular to extracellular compartment and also due to cellular damage. Movement of K^+ ion from intracellular to extracellular fluid also could have contributed to increased serum potassium concentration (Tasker., 1991) [30]. Hyperkalemia might be a major factor contributing to the high mortality rate in calf diarrhoea due to cardiac arrhythmia.
Hyperchloraemia in scouring calves might be due to liver dysfunction leading to an increase in capillary permeability with the loss of colloidal protein into tissue and resulting increased serum chloride (Sridhar *et al.*, 1988) [13]. Similar finding has also been reported by Himanshu and Pal (2015) [28]. Gross (1988) [31] reported that metabolic acidosis resulting due to the excess of H^+ and deficit of $NaHCO_3$ which could be due to overproduction of H^+ and Cl^- released from the RBCs to maintain the electro neutrality and to cope with the loss to anion (HCO_3^-) that results eventually in hyperchloraemia.

5. Conclusion
It was concluded that in diarrhoeic calves haemoconcentration occurs due to loss of extracellular fluid in faeces, GIT malabsorption as well as dehydration as manifested by increase in haemoglobin, PCV, TEC and TLC. Biochemical changes in diarrhoeic calves also manifest as higher serum protein and chloride and lower glucose and sodium.

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