COLOSTRUM FEEDING INDUCED ALKALINE INDIGESTION SYNDROME IN RIVERINE BUFFALOES- CLINICAL ASSESSMENT AND THERAPEUTIC MANAGEMENT

Ravindra Kaka Jadhav[1,*], Suresh Samadhan Ghoke[2], Anand Mohan[2], Neelam Kushwaha[1] and Anil Udhavrao Bhikane[3]

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ABSTRACT

The present study was undertaken to elucidate the alkaline indigestion syndrome in riverine buffaloes caused by inadvertent colostrum feeding, clinical pattern and its therapeutic management. Fifteen recently parturited buffaloes with history of feeding average 2.96 kg (1 to 5 kg) of colostrum immediately after parturition followed by sudden onset of anorexia, decrease in milk yield and nervous signs were investigated. Clinical examination showed normal body temperature (100.26±0.42 vs 100.62±0.18), respiration (23.73±3.53 vs 24.13±0.66) and heart rate (57.26±3.30 vs 53.20±1.79) compared to healthy buffaloes. Examination of rumen revealed impaction with highly significant ($P<0.01$) reduction in rumen motility (0.80±0.24 vs 3.80±0.17 per five minutes). Similarly, highly significant ($P<0.01$) increase in rumen pH (8.10±0.16 vs 6.25±0.07) and decrease in protozoal density (5.00±0.88 vs 32.93±1.72) with sluggish (+) to no (-) protozoal motility was observed in ailing buffaloes. Haematological analysis showed normal blood profile with significant ($P<0.05$) increase only in neutrophil count (51.60±5.1 vs 42.06±3.4). Prominent clinical signs of anorexia, congested conjunctival mucosae, decreased milk yield and varied nervous signs like restlessness, head pressing, staggering gait, incoordination, circling, convulsions, dummy syndrome, apparent blindness and coma were observed in affected buffaloes. Postural abnormalities like sternal / lateral recumbency and lateral deviation of neck were also observed in two buffaloes. All the ailing buffaloes were treated with Dextrose Normal Saline, single dose of preparation containing calcium, phosphorus and magnesium, vitamin B complex, antihistaminic, sedatives, laxatives, pre, probiotics, and oral antibiotics. All the treated buffaloes showed satisfactory improvement from 1st to 3rd day with complete clinical recovery by 4.93 (3 to 9) days of treatment. Buffalo owners should be made aware of unscientific practice of colostrum feeding in adult animals and accidentally intoxicated buffaloes could be successfully treated with the standardized treatment protocol.

[1]Department of Veterinary Clinical Medicine, Ethics and Jurisprudence, College of Veterinary and Animal Sciences, Maharashtra Animal and Fishery Sciences University, Udgir, India, *E-mail: jadhavrk11@gmail.com

[2]Department of Veterinary Epidemiology and Preventive Medicine, College of Veterinary and Animal Sciences, Maharashtra Animal and Fishery Sciences University, Udgir, India

[3]Maharashtra Animal and Fishery Sciences University, Nagpur, India

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INTRODUCTION

Numerous authors have reported syndrome of alkaline indigestion in adult bovines due to overfeeding of protein rich feed ingredients (Bartley *et al*., 1976; Raboisson *et al*., 2012; Haque and Dey, 1989; Misra and Tripathy, 1963), poorly digestible roughages like paddy straw (Mohan *et al*., 2015), decomposed or putrefied feed (Hoflund, 1967) and access to contaminated and sewage water (Nagrajan and Rajmani, 1973). Clinical syndromes due to accidental ingestion or experimental administration of urea in bovines leading to alkaline indigestion have been reported by several workers (Raboisson *et al*., 2012; Osebold, 1947; Kulkarni and Kulkarni, 2002; Sharma *et al*., 2017). Similarly, numerous experimental studies have been carried out to study the fate of protein rich diet in stomach of ruminants (Bartley *et al*., 1976; Raboisson *et al*., 2012; Davis *et al*., 1959).

Colostrum is first food for mammalian newborns stored and secreted in the mammary gland in the first few days after parturition. It is essential for newborns during early days due to its physiochemical properties and immunoprotective role (Singh and Ahuja, 1993). Primarily it is a complete nutrition of young one if given as per the requirement. Unscientific or traditional feeding practices sometimes may lead to accidental poisoning in bovines. In certain parts of Maharashtra state, buffalo owners are practicing the feeding of surplus colostrum to buffaloes culminating in alkaline indigestion induced toxicity syndrome. Until date occurrence of colostrum feeding induced alkaline indigestion in adult bovines has not been reported. The present study highlights the unscientific practice of colostrum feeding as etiological agent of alkaline indigestion in buffaloes. Similarly, the manuscript elucidates the clinical syndrome of alkaline indigestion produced by administration of colostrum in lactating buffaloes and its therapeutic management.

MATERIALS AND METHODS

History and clinical signs

Fifteen recently parturited buffaloes with sure history of feeding of colostrum immediately after parturition followed by onset of anorexia, decrease in milk yield and nervous signs presented to Teaching Veterinary Clinical Complex, College of Veterinary and Animal Sciences, Udgir formed the material for the present study. Detailed history was recorded as per clinical protocol for all ailing buffaloes. Vital parameters like body temperature, respiration rate, heart rate and ruminal motility were noted, and detailed clinical examination of the ailing buffaloes (n=15) was conducted. Similarly detailed clinical examination of healthy buffaloes (n=15) was also carried out to serve as control healthy animals.

Rumen liquor examination

Rumen liquor was aspirated by left paralumbar fossa paracentesis by aseptic technique and analyzed for protozoal motility, protozoal density, and pH immediately after collection.

Haematology

Around 5 ml of venous blood was collected in EDTA vials from ailing buffaloes by

jugular venipuncture and complete blood count was analyzed on automated hemoanalyser (Abacus Junior Vet 3.11, Diatron, Hungary).

Treatment

All the ailing buffaloes were treated with Inj. Dextrose Normal Saline 3 to 4 liters every 4 to 6 h on first day followed by 4 liters every 12 h over a period of treatment. Adjunct therapy included Inj. Dexamethasone 0.2 mg/kg iv once in buffaloes with severe clinical signs and Inj. Diazepam 0.25 mg/kg iv in buffaloes showing excitatory nervous signs every 6 h till resolution of nervous signs. Inj. Calcium-phosphorus-magnesium-dextrose (Mifex[1]) 300 to 450 ml slow iv on the basis of size of ailing buffaloes was given as single treatment. Inj. Vitamin B complex 10 ml iv and Inj. Chlorpheniramine maleate 0.25 mg/kg im were administered once a day till recovery. Supportive therapy in the form of laxative (Protolax[2] 450 ml), rumenotorics (Rumentas[3] 2 boli), probiotics (Ecotas[4] 2 boli) and oral antibiotic (Tetracycline 2 gm) once a day was given in buffaloes with normal nervous demeanor on first day as well as in buffaloes with nervous signs after resolution of nervous signs on first or second day.

Inj. mifex

Ca- 8.37 gm, Mg- 2.08 gm, P- 5.31 gm and Dextrose (anhydrous)- 90 gm/ 450 ml bottle.

Rumentas bolus

Antimony potassium tartarate (USP)- 2 gm, Ferrous sulfate (IP)- 2 gm, Copper sulfate - 50 mg and Cobalt chloride - 100 mg/ bolus.

Ecotas bolus

Sacchromyses cerevisiae + *Lactobacillus sporogenes* + *Aspergillus oryzae* + Fructo oligosaccharide + Biotin + DL methionine + Zinc sulphate + Cobalt Sulphate + Copper sulphate

Protolax liquid

Cassia angustifolia, Terminlia chebula Retz., Terminalia bellirica, Embalica officinalis, Aspergellus racemosus, Terminalia chebula/ 450 ml bottle.

Statistical analysis

Data collected on vital parameters, haematological parameters and rumen liquor parameters from ailing (n=15) and normal healthy buffaloes (n=15) was analyzed by paired 't' test for equal number of observations as per Snedecor and Cochran (1994).

RESULTS

All ailing buffaloes showed inappetance to anorexia and decrease in milk yield. Clinical signs varied among ailing buffaloes based on quantity of colostrum ingested and duration lapsed from onset of clinical syndrome to admission of ailing buffaloes to clinics for treatment. All ailing buffaloes showed moderate to severely congested conjunctival mucosae (Figure 1). Vital parameters, rumen liquor pH, protozoal density and haematological parameters analyzed in ailing buffaloes as well as healthy buffaloes are depicted in Table 1. Body temperature (100.26±0.42 vs 100.62±0.18), heart rate (57.26±3.30 vs 53.20±1.79) and respiration rate (23.73±3.53 vs 24.13±0.66) revealed no significant difference between ailing and healthy buffaloes.

Clinical examination of left paralumbar fossa revealed slight to moderate distension. Examination of rumen revealed impaction with

highly significant (P<0.01) reduction in rumen motility (0.80±0.24 vs 3.80±0.17 per five minutes) in buffaloes ailing with colostrum feeding induced alkaline indigestion compared to healthy buffaloes. Highly significant (P<0.01) increase in rumen pH (8.10±0.16 vs 6.25±0.07) and decrease in protozoal density (5.00±0.88 vs 32.93±1.72) was observed in ailing buffaloes compared to control healthy counterparts. Protozoal motility was sluggish (+) to absent (-) in ailing buffaloes. Haematological analysis showed significant (P<0.05) increase in neutrophil count (51.60±5.1 vs 42.06±3.4) while rest of the parameters showed no significant difference between ailing and healthy buffaloes.

Moderately affected buffaloes showed anorexia, drop in milk yield, shivering, restlessness, arching of back, staggering gait and varying degree of dehydration. Prominent nervous signs like restlessness, head pressing (13.33%), staggering gait, incoordination and circling (46.66%), convulsions (13.33%), dummy syndrome (20%), apparent blindness/ absence of menace reflex (33.33%) and coma (13.33%) were reported. Postural abnormalities like sternal / lateral recumbency and lateral deviation of neck (40%) were also recorded (Figure 2 and 3).

Based on sure history of colostrum feeding, clinical signs and rumen liquor examination, the buffaloes were diagnosed for colostrum feeding induced alkaline indigestion and treated accordingly.

Administration of Dextrose Normal Saline (DNS) every 4 to 6 h revealed gradual resolution of depressed nervous system in buffaloes with dummy syndrome and coma while buffaloes with excitatory signs also revealed normalization of nervous demeanor with visible clinical improvement. After institution of aggressive fluid therapy along with single dose of polypharmacy preparation containing calcium borogluconate, magnesium, phosphorus and dextrose, buffaloes showed favorable response in the form of urination, regaining of nervous demeanor, gradual improvement in posture and gait from lateral to sternal recumbency or normal ability to stand and bear weight. Two buffaloes with blindness from the study animals also showed response to menace reflex after one-day treatment.

From second day onwards clinical examination of ailing buffaloes revealed normal vital parameters, normal posture, gait and behavior, initiation of feed and water intake as well as rumination. Administration of laxatives showed increase in fecal output with passage of loose, foul-smelling feces in treated buffaloes. Frequency of fluid therapy was reduced to twice a day from second day onwards with 3 to 4 liters DNS along with vitamin B complex 10 ml iv daily, chlorpheniramine maleate 0.25 mg/kg im daily, laxative, rumenotorics and probiotics till recovery. Oral antibiotic was administered for three days with objective of suppressing protein-degrading microflora in rumen. Complete clinical recovery (Figure 4) with subsidence of all nervous signs, restoration of normal feed and water intake, normal rumination and fecal output required average 4.93 days (3 to 9 days).

DISCUSSION

Alkaline indigestion, an important digestive disorder of ruminants affecting the health and milk yield of dairy animals. Numerous etiological agents like soybean meal, urea, excess feeding of paddy straw, decomposed and putrefied feed as well as drinking of contaminated and sewage water have been implicated as causative

factors for alkaline indigestion in bovines (Bartley *et al*., 1976; Raboisson *et al*., 2012; Haque and Dey, 1989; Misra and Tripathy, 1963; Hoflund 1967; Nagrajan and Rajmani, 1973). Administration of 5 gm urea per kg body weight along with cooked mixture of different grains via rumen fistula revealed positive correlation of rumen pH and blood ammonia concentration while rumen ammonia and blood urea were not correlated with toxicity signifying the role of readily fermentable feed in suppressing the toxicity. In addition, the toxicity was easily managed by rapid evacuation of rumen contents compared to oral administration of acetic acid (Bartley *et al*., 1976). Administration of 5 gm urea per kg body weight in non-adapted two fistulated cows revealed rise of ammonia concentration to 1.47 mg/dl (carotid blood) and 0.95 mg/dl (jugular blood) at the onset of toxicity which was characterized clinically by muscle tetany and rumen pH of 6.94 to 7.90. All the cases revealed complete recovery within one hour on emptying of rumen (Davidovich *et al*., 1977).

Experimental intoxication in cattle by administration of soybean meal (SBM) via cannula at 1 and 2% of body weight revealed marked accumulation of ammonia in blood and severe metabolic alkalosis (Raboisson *et al*., 2012). Further authors also revealed increased ammonia concentration in blood was due to continued ammonia production from protein degradation, decreased carbohydrate fermentation and overwhelming of hepatic detoxifying capacity.

Ruminant stomach is composed of four compartments *viz*., rumen, reticulum, omasum and abomasum. Reticulo-rumen being fermentation vat are non-functional during first few weeks of life (Reece *et al*., 2015; Radostits *et al*., 2010). Colostrum is a source of numerous important nutrients and immunoglobulin to newborn calves. Bovine colostrum contains around 14.9% proteins and 2.5% lactose (Kehoe *et al*., 2008). The nutrients in colostrum are absorbed in abomasum of young calves. On the contrary, excess colostrum accidently fed to adult buffalo could be degraded in reticulo-rumen leading to release of protein degradation products like ammonia. Increased ammonia levels in rumen, its increased absorption in blood with elevation of blood ammonia levels might be responsible for pathogenesis of colostrum feeding induced alkaline indigestion involving neurological signs in buffalo in the present study. Several clinical studies have signified the importance of oral administration of vinegar/ acetic acid to neutralize the ammonia accumulated in rumen (Haque and Dey, 1989; Kulkarni and Kulkarni, 2002; Davidovich *et al*., 1977). In the present study buffaloes with colostrum intoxication revealed lapse of 1-2 days between colostrum feeding and actual treatment initiation. Most of the buffaloes were already exhibiting neurological signs making oral drenching of vinegar practically impossible. Similarly, most of the ammonia produced in rumen from degradation of colostral proteins might have been absorbed in blood. Hence, major thrust was given on parenteral fluid therapy to dilute the ammonia concentration in blood and hasten excretion along with supportive therapy. Polypharmacy preparation might prove helpful in correcting existing subclinical hypocalcemia and to increase the ruminal motility. Buffaloes with excitatory nervous signs were given diazepam to reduce the intensity of excitatory nervous symptoms while dexamethasone was used as lifesaving drug to reduce nervous signs and to prevent shock in severe cases of alkaline indigestion. Vitamin B complex was used to stimulate hepatic metabolism while chlorpheniramine maleate was used to counteract histamine release in rumen and

Table 1. Values of vital, rumen liquor and haematological parameters of buffaloes with colostrum feeding induced alkaline indigestion and healthy buffaloes.

Sr. No.	Parameter	Buffaloes with colostrum induced alkaline indigestion (n=15)	Normal Healthy buffaloes (n=15)	't' value
1	Body temperature (°F)	100.26±0.42	100.62±0.18	-0.714
2	Heart rate (per minute)	57.26±3.30	53.20±1.79	1.05
3	Respiration rate (per minute)	23.73±3.53	24.13±0.66	-0.105
4	Rumen motility (per 5 minutes)	0.80±0.24	3.80±0.17	-9.27**
5	Rumen pH	8.10±0.16	6.25±0.07	10.12**
6	Protozoal count (per LPF)	5.00±0.88	32.93±2.72	-16.49**
7	WBC ($x10^9$/l)	9.10±0.94	9.84±0.38	-0.749
8	Lymphocytes %	45.55±4.44	54.41±3.63	-2.49
9	Neutrophils %	51.60±5.1	42.06±3.4	1.43*
10	RBC ($x10^{12}$/l)	5.47±0.29	6.20±0.22	-1.916
11	Hb (gm/dl)	11.26±0.34	11.42±0.23	-0.487
12	PCV (%)	33.65±1.42	32.13±1.21	1.075
13	Platelets ($x10^9$/l)	185.86±14.08	199.0±18.45	-0.632

* Level of significance P<0.05, ** Level of significance P<0.01, NS- Non-significant.

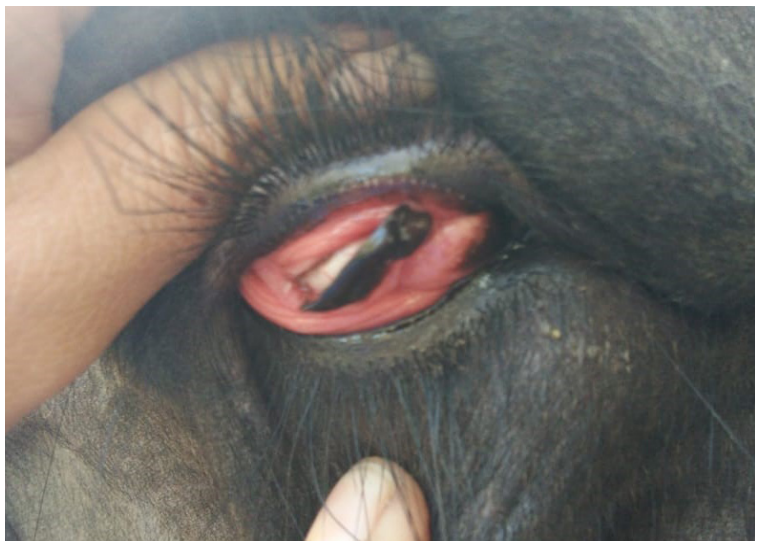

Figure 1. Congested mucosae in buffalo suffering from colostrum feeding induced alkaline indigestion.

Figure 2. Lateral deviation of neck and dummy syndrome in colostrum feeding induced alkaline indigestion affected buffalo before treatment.

Figure 3. Lateral deviation of neck and dummy syndrome in colostrum feeding induced alkaline indigestion affected buffalo before treatment.

Figure 4. Normal posture and demeanor of buffalo after response to treatment and complete clinical recovery.

thereby restore ruminal motility (Radostits *et al*., 2010). Supportive therapy in the form of laxative and rumenotorics was aimed to hasten ruminal emptying and regain rumen motility while oral antibiotic was used to destroy protein-degrading microflora in rumen. Probiotic was used to restore normal ruminal microflora, which was severely disturbed due to change in rumen pH towards alkaline range.

Colostrum being rich source of protein and its degradation in rumen might be responsible for intoxication similar to urea and soybean meal poisoning syndromes. Cases of colostrum intoxication induced alkaline indigestion in buffaloes could be successfully treated with adequate dextrose normal saline, calcium preparations, vitamin B complex along with laxatives, rumenotorics, probiotics and oral antibiotics. It is an unscientific practice to feed colostrum to adult ruminants and requires increasing awareness in the area towards ill effects of such traditional and unscientific feeding practice.

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