Student Clinical Reports

Editor's Note: The AABP Board of Directors meeting in Washington, D.C., on July 21, 1980 approved a recommendation from the Forward Planning Committee to encourage veterinary medicine students to write case reports for The Bovine Practitioner. Prizes were $200, $100 and $50 for the top three reports. The Editorial Board awarded the following for 1982 and their papers are published herewith:

First Prize ($200): Anthony L. Kiorpes, Kansas State (advisor — Dr. John Noordsy)

Second Prize ($100): Students in the class of 1983, Western College of Veterinary Medicine, University of Saskatchewan, Canada (advisor — Dr. E. D. Janzen)

Third Prize ($50) Mark R. Olson, Kansas State University (advisor — Dr. Thomas Avery)

Diarrhea and Hypoproteinemia in the Calf of a Mastitic Dam

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Diarrhea, dehydration, and failure to gain are serious problems in newborn ruminants. Perhaps the most common cause of diarrhea in calves is enteric infection of multiple etiology with dehydration and failure to gain as secondary complications. The susceptibility of ruminant neonates to enteric infection is believed to be especially high in those which fail to obtain adequate quantities of maternal antibodies (1, 2). It is well known that newborn ruminants are born immunodeficient, and must acquire protective immunoglobulins from the colostrum within the first 24 hours of life. There are numerous reasons for this failure of passive transfer, of which mastitis in the dam may be one. In this condition, the susceptibility to infection and diarrhea may be compounded by the overall lack of fluid and caloric intake, exacerbating the dehydration and failure to gain. It is relatively easy to spot acute mastitis; however, chronic mastitis in the dam represents one of the more cryptic aspects of this problem.

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CASE HISTORY

A one-week-old, female, Angus calf was presented to the veterinary hospital with the complaint of malaise and failure to gain. On physical examination, the patient was gaunt, enopthalmic, and weak. The owner reported an intermittant diarrhea, but upon admission there was no evidence of such. From the initial data base, the calf weighed 55 pounds, had a temperature of 103° F, a heart rate of 84, and a respiratory rate of 48. The dam was brought with the calf and was presented with active mastitis in both rear quarters.

Blood samples were taken from the calf by venopuncture and submitted to the clinical pathology laboratory for a total blood count, analysis of total protein, hematocrit, and hemoglobin determinations. The initial therapeutic regimen included administration of 3 liters of lactated Ringer's solution (LRS) intravenously, 1 gram of chloramphenicol subcutaneously, and 10 cc of vitamin B complex intramuscularly. The patient was additionally rehydrated with 1.5 liters of milk replacer *per os*. No further treatment was given on the day of admission (hospital day 0).

On hospital day 1, the patient had responded favorably to the therapy; temperature, pulse, and respiration (TPR) were all within normal ranges. Similarly, the leukogram was normal (see Table I) but the total protein was reduced to 5 g/dl. Antibiotic therapy was changed to oral chloramphenicol (Medichol) 20 mg/lb, and oral milk replacer was continued TID. Although the calf started to pass a dark, watery, fetid stool at this time, laboratory data suggested that the patient was otherwise normal. The oral milk replaced and Medichol

TABLE I

Analysis of blood parameters in the calf of a mastitic dam between hospital days 0 through 11

Hospital day	0	1	5	7	11
Parameter					
Total WBC($X10^3$)*	9.3	10.9	7.1	6.8	10.4
Polymorphonuclear leukocytes*	6045	8175	3550	3400	4680
Stabs*	372	0	0	0	52
Lymphocytes*	1953	2289	3053	2720	4004
Monocytes*	837	436	497	680	1664
Basophils*	93	0	0	0	0
Total RBC($X10^6$)*	11.6	9.9	7.6	11.2	9.2
Hemoglobin(gm/dl)	47.7	39.1	29.3	44	34.9
Hematocrit(%)	15.9	17.3	10.5	15.7	12.8
Total protein (gm/dl)	5	4.7	4.1	5.4	4.9
Fibrinogen (mg/dl)	500	500	500	700	500
Plasma protein: fibrinogen	10	9.4	8.2	7.7	8.8

* Cells/μl

were continued over the next three days and slight improvement was noted.

On the morning of hospital day 4, the rectal temperature began a slow increase to 103.6 F where it remained for several days. During that time, the treatment regimen continued unchanged. On hospital day 7, the calf developed a yellow, pasty diarrhea accompanied by an acute metabolic acidosis (base deficit 9 mEq/L). The total protein had fallen to 4.1 g/dl. Sodium bicorbonate, 40 mEq, was administered subcutaneously and 2 liters of a commercial oral electrolyte solution (Resorb) was given and continued BID. There was no improvement by hospital day 8, and the patient was started on 960 mg of trimethoprim and sulfadiazene (Tribrissen) with oral electrolyte plus bicarbonate given TID. The yellow diarrhea and elevated rectal temperature continued through hospital day 9. Blood gas analysis confirmed a metabolic acidosis with a base deficit of approximately 10 mEq/L (Table II). The total protein remained decreased. Antibiotic and oral

TABLE II

Analysis of venous blood gases in the calf of a mastitic dam between hospital days 5 through 11

Hospital day	5	7	8	9	10	11
Parameter*						
PO_2 (torr)	35.4	36.2	41.6	39.4	32.3	26.7
PCO_2 (torr)	46.2	43.6	48.3	47.7	49.7	52.9
pH	7.319	7.210	7.176	7.220	7.251	7.259
HCO_3^- (mEq/L)	22.2	16.3	16.7	18.3	20.4	22.2
Base Excess	- 1.7	- 9.0	- 9.5	- 7.2	- 4.5	- 2.5

*PO_2, PCO_2, and pH are corrected for differences between the calf's body temperature and that of the blood gas analyzer.

electrolyte therapy continued unchanged for another 24 hours. By hospital day 10, the acidosis was improving, but the TPR, diarrhea, and total protein were all outside of the normal ranges. The decision was made to switch antibiotics to gentamycin (Gentocin) and continued oral supplementation of electrolytes for another 24 hours.

By hospital day 11, the diarrhea was improving, the temperature was 102.6, and all acid-base parameters were normal. The calf was still severely hypoproteinemic and the assessment was made of malabsorption secondary to acute enteritis and a failure to obtain colostrum. A serum sample was submitted for electrophoresis. The results are summarized in table III. Based on these data, hypoproteinemia secondary to hypogammaglobulenemia was the assessment. The diagnosis of failure to obtain colostrum was made, and it was decided that homologous plasma be administered parenterally.

Table III

Comparison of plasma electrophoretic analysis of dam and calf blood before and after transfer of adult plasma to calf via intraperitoneal route

	Calf (Before)	Dam	Calf (After)
Parameter gm/dl			
Total Protein	4.0	7.8	4.9
Albumen	2.4	3.4	2.6
α	0.6	1.1	0.9
β	0.6	0.7	0.7
γ	0.4	2.6	0.7

Four liters of citrated whole blood were aseptically taken from the dam through a jugular cannula. The blood was spun down in a refrigerated centrifuge at 1000 g for 60 minutes. Aliquots of the plasma were removed with a sterile pipette and placed in empty caline infusion bags. A 6 x 6 cm area on the right flank of the calf was then clipped and prepared by multiple scrubbings with Betadine followed by an alcohol rinse. A sterile 14 ga. disposable needle was placed through the abdominal wall into the peritoneal space. Two liters of the fresh plasma were then administered over a one hour period to the calf using an IV infusion set. The calf was physically restrained in a quiet stall throughout the procedure. A blood sample drawn 12 hours after the infusion showed that the total protein had increased to 5 g/dl. Plasma electrophoresis of the 12 hour sample are summarized in Table III. The calf continued to improve over the next 48 hours. Both the calf and the dam were discharged on hospital day 14. The calf made an uneventful recovery.

DISCUSSION

The pathogenesis of diarrhea in calves is itself complicated by interacting organisms and environmental conditions. It is unclear if mastitis in the dam results in neonatal disease. It has been assumed as an article of faith that the underlying basis for neonatal diarrhea is lack of colostrum-derived surface and circulating immunoglobulins. However, Bradley *et al.* (3) have shown that there is no correlation between post-suckling immunoglobulin levels and the severity of undifferentiated neonatal diarrhea. It cannot be concluded from this study whether susceptibility to infection in otherwise healthy calves is related to colostral immunoglobulins. It has been shown in other studies that mastitis in the dam does result in lower serum immunoglobulin levels in the calf (4). It is not known whether mastitis affects the quality of the colostral secretions directly or whether the lack of passive transfer is due simply to indirect causes which delay suckling. While the possible complications arising from the ingestion by the calf of a highly contaminated product may play a role in the pathogenesis of disease, it must be noted that the feeding of mastitic colostrum to newborns is a common management technique which has not been reported to have untoward effects.

It is clear that in the present case report, almost no passive transfer of colostrum had occurred. Studies of 23 newborn calves (presuckling) showed serum gammaglobulin concentrations of 0.06-0.56 g/dl. Postsuckling mean gammaglobulin values should be approximately 2 g/dl or greater (3). Most authors agree that calves with gammaglobulin concentrations in the range exhibited by the calf in the present case have a poor prognosis for survival. It is difficult to say whether our procedure of giving IP plasma and raising the fraction from 0.4 to 0.7 gm/dl was a significant factor in the overall recovery, since the literature questions whether immunoglobulin levels can be used as predictors of survivability in individual animals (3).

There are other factors in the pathogenesis of calf diarrhea which may be only tangentially related to the finding of mastitis in the dam, such as nutrition, calving management, and environmental conditions (5). It must be concluded that defining the relationship between calf diarrhea and mastitis in the dam will require further investigation.

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