Bovine Leukemia Virus Transmission by Dehorning in Dairy Heifers

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Introduction

A commercial dairy of about 400 Holstein cows with an average production of over 20,000 pounds of milk per lactation was experiencing an increase in the number of cows with lymphosarcoma. This herd was located thirty miles north of Seattle, Washington. Initial testing of a sample of different age groups in the herd for antibodies to bovine leukemia virus (BLV) was done to evaluate the age at which infection occurred (Table 1). Antibodies to BLV were detected in 3 of 21 calves bled at birth (precolostral) for 14% positive. Twenty-four calves aged six months to one year were bled with one positive for 4%. In cattle over one year of age, 44 or 54 (81%) had antibodies to BLV. The difference in prevalence between cattle less than and greater than one year of age was statistically ($p<0.05$) significant.

Several studies have shown that the incidence of *in utero* transmission of bovine leukemia virus occurs in less than 20% of calves from infected cows. [1 7] Hence, vertical transmission occurs but horizontal transmission is generally condidered the major mode of transmission of BLV.[1 3 8] Since the only cell known to be infected by BLV is the lymphocyte,[9 10] infection can only result from transfer of infected lymphocytes. At the dairy under study, our findings suggested considerable transmission of BLV in calves under one-year-old. Calves 6 to 12 months old were maintained in a covered freestall barn, 20 feet by 180 feet. They were fed hay, silage and 2-3 pounds of grain daily. Water was provided in metal troughs. Evaluation of management practices of the dairy revealed that the following procedures were performed in calves 6 to 12 months old: intramuscular and subcutaneous vaccination with a multidose syringe against several infections and dehorning by the gouge method. It was theorized that the practice of gouge dehorning might be involved in the transmission of BLV.

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TABLE 1. Prevalence of Antibodies to BLV in a Commercial Holstein Herd.

Age (years)	No. in herd	No.*	%
0†	100‡	3/21	14
½-1	200‡	1/24	4
1-2	190‡	8/10	80
2-3	170	6/10	60
3-4	123	8/12	67
>4	143	22/22	100

* Numerator = No. positive, denominator = No. tested
† Precolostral
‡ Estimate

Materials and Methods

Seventy-one calves, 6 to 12 months old were randomly divided into 3 groups: (1) 21 were not dehorned (control), (2) 30 were dehorned by the gouge method and (3) 20 were dehorned by the gouge method followed by cautery. Groups were formed concurrently; dehorning was done in the winter (February, 1983). Dehorning by the gouge method was performed by the dairyman with a Barnes dehorner. Bleeding vessels were removed with hemostats. Instruments were neither cleaned nor disinfected between calves. This practice had been in existence on the farm for several years. The group dehorned by the gouge method followed by cautery was done by a veterinarian. The Barnes dehorner was rinsed in a virucidal disinfectant[a] between calves. The dehorned areas were cauterized by the application of an electrical dehorner for 30 seconds. Further bleeding was arrested by application of ferrous sulfate powder.[b] Calves were bled at the time of dehorning, 3 and 6 months later. No other parenteral inoculations were administered. All three groups remained co-mingled.

Blood was collected from the jugular vein in a syringe and was transferred to evacuated tubes without anticoagulant.

[a] *Chlorhexidine dacetate, Fort Dodge Laboratories Inc., Fort Dodge, Iowa.*

[b] *Styptic Powder, Bio-Ceutic Laboratories Inc., St. Joseph, Missouri.*

Needles and syringes were used once and discarded. Serum was obtained after centrifugation of blood and assayed for BLV antibodies, using the agar-gel immunodiffusion test.[13] The antigen preparation contained glycoprotein and internal virion antigens. Immunodiffusion plates were evaluated after 48, 72 and 96 hours, and results were recorded as positive or negative. The prevalence of reactors between groups was compared by Fisher's exact test. The prevalence of reactors within groups was compared by the sign test.

Results

At the time of dehorning no significant difference in prevalence of BLV antibodies was detected between calves not dehorned (control), and calves dehorned by the gouge method or calves dehorned by the gouge method followed by cautery (Table 2). Three months after dehorning, 1 of 19 control calves had seroconverted, while 7 of 22 calves dehorned by the gouge method had seroconverted. The prevalence of BLV antibodies was significantly ($p<0.05$) greater in the gouge dehorned group than the control group. Calves dehorned by the gouge method followed by cautery had no seroconversions (Table 2). Comparison of the acquisition of antibodies to BLV within groups also revealed that calves dehorned by the gouge method had a significant ($p<0.05$) number of seroconversions (Table 3). Testing at 6 months after dehorning detected no further seroconversion in a calf dehorned by the gouge method followed by cautery.

TABLE 2. Prevalence of Antibodies to BLV in Holstein Calves by Dehorning Method.

		Months after dehorning			
		0		3	
Dehorning method	Number tested	Number positive	Percent positive	Number positive	Percent positive
None	21	2	10	3	14
Gouge	30	8	27	15	50*
Gouge & cautery	20	6	30	6	30

* Prevalence significantly ($p< 0.05$) greater than calves not dehorned.

TABLE 3. Comparison of Antibodies to BLV in Holstein Calves Before and After Dehorning.

Dehorning method	Before dehorning	After dehorning		Total
		BLV−	BLV+	
None	BLV−	18	1	19
	BLV+	0	2	2
				21
Gouge	BLV−	15	7*	22
	BLV+	0	8	8
				30
Gouge & cautery	BLV−	14	0	14
	BLV+	0	6	6
				20

* Significant (< 0.05) number of seroconversions.

Discussion

The results of this study strongly suggest that BLV was transmitted by dehorning in Holstein calves at a commercial dairy. The mechanism of transmission was considered to be blood or tissue, remaining on the dehorning device from BLV infected calves, inoculated into susceptible calves when dehorned. It has previously been reported that transmission of BLV was suspected following routine blood sampling of herds and premunization against babesiosis using whole blood.[15] Cattle have been infected by intradermal inoculation of as few as 2,500 lymphocytes, the number of lymphocytes present in 0.0005 ml of blood.[11]

Assay for BLV antibodies three months after dehorning was based on experimental studies which detected antibodies to BLV in one to three months after inoculation.[11 12 16] Calves infected by dehorning also developing antibodies within three months after exposure. This study also demonstrated that rinsing the dehorning device in disinfectant between calves and cautery prevented transmission of BLV.

Since the beginning of this evaluation all vaccinations and injections have been by new needles for each animal. The heifers were dusted once a month for ectoparasite control. There has been no segregation as to BLV positive/negative. All heifers are segregated by age and cows by production groups.

Summary

In a commercial dairy, heifers dehorned by the Barnes gouge method without rinsing in disinfectant solution and not cauterizing had significantly ($p<0.05$) more heifers developing antibodies to BLV, as measured by agar-gel immunodiffusion, three months after dehorning than calves not dehorned. With the demonstration of this mode of transmission of BLV in the herd, the practice of gouge dehorning was discontinued and electrical dehorning at 6 to 12 weeks of age instituted. The entire herd is presently being bled at 6 month intervals to determine whether BLV infection will be delayed in time or eliminated.

References

1. Ferrer J.F., Piper C.E., Abt D.A., et al: Natural mode of transmission of the bovine C type leukemia virus (BLV). Bibl Haematol 43:235-237, 1976. 2. Ferrer J.F., Piper C.E., Baling V.: Diagnosis of BLV infection in cattle of various ages, *in* Burney A. (ed.): Bovine Leucosis: Various Methods of Molecular Virology. Luxembourg: Commission of European Communities, 323-336, 1977. 3. Piper C.E., Ferrer J.F., Abt D.A., et al: Postnatal and prenatal transmission of the bovine leukemia virus under natural conditions. J Natl Cancer Inst 62:165-168, 1979. 4. Van der Maaten M.J., Miller J.M., Schmerr M.J.: In utero transmission of bovine leukemia virus. Am J Vet Res 42:1052-1054, 1981. 5. Hofirek B.: The development of antibodies to BLV in a leukosis cattle herd, *in* Straub O. (ed.): Fourth International Symposium on Bovine Leukosis. Luxembourg: Commisison

of European Communities 475-488, 1982. 6. Thurmond M.C., Carter R.L., Puhr D.M., et al: An epidemiological study of natural in utero infection with bovine leukemia virus. Can J Comp Med 47:316-319, 1983. 7. Kono Y., Sentsui H., Arai K., et al: Serological methods to detect calves infected in utero with bovine leukemia virus. Jpn J Vet Sci 45:453-461, 1983. 8. Piper C.E., Abt D.A., Ferrer J.F., et al: Seroepidemiological evidence for horizontal transmission of bovine C-type virus. Cancer Res 35:2714-2716, 1975. 9. Baliga V., Ferrer J.F.: Expression of the bovine leukemia virus and its internal antigen in blood lymphocytes. Pro Soc Exp Biol Med 156:388-391, 1977. 10. Kettmann R., Burny A., Cleuter Y., et al: Distribution of bovine leukemia virus proviral DNA sequences in tissues of animals with enzootic bovine leukemia. Leuk Res 2:23-32, 1978. 11. Van der Maaten M.J., Miller J.M.: Susceptibility of cattle to bovine leukemia virus infection by various routes of exposure. Adv Comp Leuk Res, Elsevier/North-Holland Biomedical Press, 29-32, 1977. 12. Roberts D.H., Lucas M.H., Wibberley G., et al: An investigation into the susceptibility of cattle to bovine leukosis virus following inoculation by various routes. Vet Rec 110:222-224, 1982. 13. Committee on Bovine Leukemia of the American Association of Veterinary Diagnosticians: Protocol for the immunodiffusion test for bovine leukemia virus *in* Proceedings, 23rd Annu Meeting, Am Assoc Vet Lab Diag, 1-7, 1980. 14. Wilesmith J.W.: Needle transmission of bovine leukosis virus. Vet Rec 104:107, 1979. 15. Hugoson G., Vennstrom R., Hendricksson K.: The occurrence of bovine leukosis following the introduction of babesiosis vaccination. Bibl Haematol 31:157-161, 1968. 16. Roberts D.H., Lucas M.H., Wibberley G., et al: Investigation of the possible role of the tuberculin intradermal test in the spread of enzootic bovine leukosis. Vet Res Com 4:301-305, 1981.

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Abstracts

Factors Responsible for Post Parturient Metritis in Dairy Cattle, O. Markusfeld, Veterinary Record (1984) 114, 539-542.

Factors associated with retained placenta and post parturient metritis in Israeli-Friesian dairy cattle are examined. The overall incidence rate, in a total of 2017 calvings on seven farms, was 16.1 percent for retained placenta and 37.3 percent for primary metritis. Risk factors associated with retained placenta include rising parity, short gestations, induction of parturition, multiple births, summer calvings, left displacement of the abomasum and ketosis. Risk factors associated with metritis include declining parity, long gestations, induction of parturition, stillbirth, multiple births, low milk yield before drying off, left displacement of the abomasum, ketosis and winter calvings. A proposed aetiology of metritis is presented and the various possible factors involved are discussed.

Malignant Catarrhal Fever, H. W. Reid, D. Buxton, E. Berrie, I. Pow, J. Finlayson, Veterinary Record (1984) 114, 582-584.

Malignant catarrhal fever is briefly reviewed and recent findings are described. Initially the disease was observed as a disease of cattle in Europe where, although no cause could be identified, circumstantial evidence implicated sheep as a source of infection and it was thus designated 'sheep-associated' malignant catarrhal fever. Subsequently the disease was observed in Africa where it became evident that a herpesvirus which normally infects wildebeest was the cause. It is now apparent that deer are highly susceptible to both forms of the disease, the sheep-associated form being a serious problem in farmed deer. The wide spectrum of clinical and pathological changes that occur in affected deer are described. A major constraint to studies of sheep-associated malignant catarrhal fever has been the absence of an experimental laboratory system. However, from affected deer it has been possible to transmit the disease to rabbits and thus has allowed detailed pathogenesis studies to be made which are summarised in this paper. It is suggested that the agent of sheep-associated malignant catarrhal fever is a virus and that when a particular subpopulation of T-lymphocytes is infected a profound immunological perturbation results; the lesions of malignant catarrhal fever being explained by a benign T-lymphocyte hyperplasia accompanied by a deregulation of cytotoxic natural killer lymphocytes that gives rise to tissue necrosis.

Tail Painting Technique as an Aid to Oestrus Detection in Cattle, O. M. Kerr, W. J. McCaughey, *Veterinary Record* (1984) 114, 605-607.

The value of heat detection by tail painting was assessed by simultaneous visual observation and regular plasma progesterone assay in grazing animals; 88.1 percent of all heats were accompanied by a positive tail paint record and 30.1 percent of positive tail paint records occurred in dioestrous animals. Conception rates to the first insemination were 57.6 percent and 57.9 percent for heifers and cows, respectively. Of seven animals which showed behavioural oestrus but had intact tail paint, three conceived following insemination. These results suggest that tail paint may be a useful aid in heat detection.

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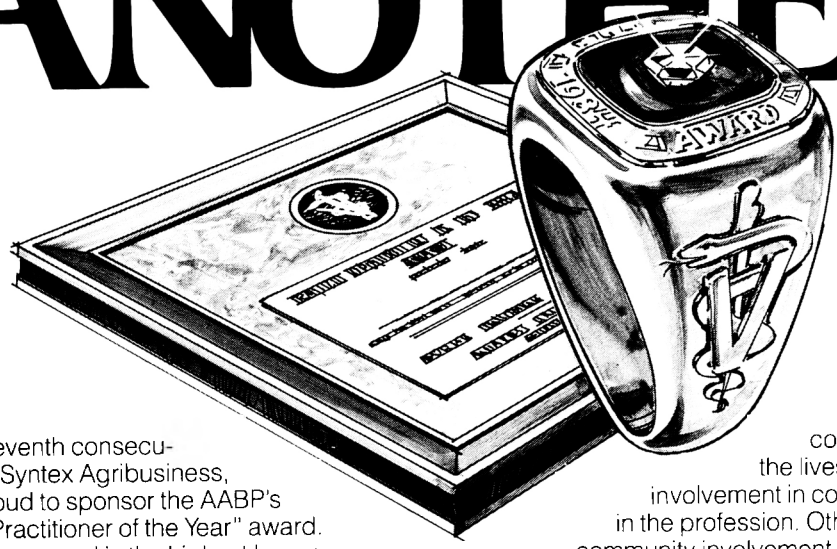

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