Bovine Virus Diarrhoea - A Clinical Perspective

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Introduction

This disease is a common problem in dairy and beef cattle in New Zealand. A search of the proceedings of this Society indicates that only 5 papers have been presented on this subject in its 34-year history. Three of these described complex case studies, one related to the link with Hairy Shaker disease in sheep and one dealt with vaccination protocols.

Serological evidence suggests that cattle are exposed to this virus frequently in New Zealand.

In the last 10-15 years the beef industry has become very reliant on the supply of 4-day-old bull calves from the dairy industry. These calves are generally reared to around 18 months and then killed for the United States burger market.

In the last 3 years I have dealt with a number of cases of Bovine Virus Diarrhoea (BVD) in weaner bulls where there have been significant numbers of deaths and inhibited growth rates. Added to this I have also dealt with 2 dairy herds and 1 beef herd where dry cow rates were 40%,30% and 25% respectively. Serological tests indicated that exposure to the BVD virus was widespread in these herds, but one could only speculate as to the significance of the virus on fertility in these herds.

Typical Clinical History

In August of 2002 I was contacted by a bull calf rearer who had a problem with ill-thrift and deaths in 5-month-old Friesian bulls.

The history of this case was that he had a similar problem in 2001. An investigation by another practice had been inconclusive but coccidiosis was considered the most likely problem.

He had purchased 120 x 4-day-old Friesian bull calves from several winter milking herds in March 2002. The calves were reared on milk powder and calf nuts until they were weaned at 80kg. In early August several of the calves started to loose weight, scour and not respond to anthelmintic treatment. I was called in mid-August. I examined 6 of the thin scouring calves. There were no outstanding oral mucosal ulcers found in any of the calves. Bloods and faeces were collected for serum copper, glutathione peroxidase, worm egg counts and BVD Ag.

Laboratory Results

Table 1: Results of laboratory tests from first farm visit

Calf No.	GSPxKU/L	Serum Copper (umol/l)	Strongyle epg	Nem egg	BVD Ag Elisa
45	24.2	9.6	0	0	
77	32.2	10	0	0	
44	33.3	10	0	0	-ve
53	35.6	6.3	0	0	-ve
39	31.2	10	0	0	
42	35	9.4	0	0	-ve

The parasitology report indicated that in calves 44,45 and 77 moderate to heavy numbers of coccidia were present. One of the calves died the next day. An autopsy was carried out and a range of samples were collected for histopathology. The two outstanding histopathological lesions present were enteritis and bronchopneumonia. The laboratory comment read "the most likely cause of diarrhoea is coccidiosis. In addition there is evidence of an eosinophilic reaction in the lungs, suggesting lungworm may also be playing a role in ill-thrift".

Treatment

The calves were re-introduced to a meal containing a coccidiostat and the scouring calves were treated orally with Scourban Plus for 3 days. As the serum coppers were marginal all the calves were treated with a 10gm Copacap.

Additional Results

Two weeks later I phoned the farmer to ask how the calves were progressing. Things had got worse! At this point I offered to re-examine the calves and carry out further testing at my expense. Rectal swabs were collected from 4 calves for bacteriology and faecal samples for parasitology. The results are recorded on Table 2.

Table 2: Results of laboratory tests from second farm visit

Calf No.	Yersinia	Salmonella	Strongyle epg	Nemt	Coccidia
73	-ve	-ve	0	0	0
79	-ve	-ve	0	0	0
71	-ve	-ve	0	0	0
72	-ve	-ve	0	0	0

I also carried out another autopsy a day later on a fresh dead calf. A range of samples were collected for histopathology. The findings of significance were ulceration of the oesophagus and rumen. The severity of the lesions lead the histopathologist to suggest the calf may have had access to an irritant caustic chemical. A thorough examination of the property could find no likely source of such a chemical.

A week later another calf lapsed into weight loss and diarrhoea. I arranged for the farmer to take the calf to Palmerston North for an autopsy at the Gribbles Laboratory. The results of this autopsy once again were inconclusive but stomatitis, glossitis, pharyngitis, oesophagitis, abomasitis and enteritis were how various tissues were described. The spleen was sampled for BVD Ag and was negative. Finally a BVD antigen immunostain was carried out on the spleen. This was weakly positive. It had taken 6 weeks to establish the presence of the BVD virus in this herd.

Traceback

When all the deaths (16/120) were checked back with records, all the dead and affected calves had come from one dairy herd. This was identified as the dairy herd previously mentioned that had an empty rate of 30%.

Subsequent to this, I visited the dairy farm supplying these calves to blood and milk sample a random group of cows. The results of the tests are in Table 3.

Table 3: Results of laboratory tests from visit to calf supplier farm.

Cow No.	BVD Antigen	BVD Antibody
158	-ve	
157	-ve	
375	-ve	+ve
3	-ve	+ve
195	-ve	
357	-ve	
102	-ve	+ve
194	-ve	-ve

The BVD Milk Antibody elisa test carried out on a bulk milk sample from the milk vat was ≥30%. 12 months after the first calf rearer case another client who purchased 4-day-old calves from the same dairy herd had a similar outbreak of ill-thrift and diarrhoea in a mob of 40 calves. In total 6 died.

In this case the initial parasitology and histopathology lead one to believe mild parasitism and coccidiosis were causing the problem. The 5 x BVD Ag tests and one BVD antigen immunostain were all negative. In 2 calves *Yersinia pseudotuberculosis* was isolated.

In this case establishing the presence of the BVD virus was not possible, as the owner was not prepared to carry out all the testing carried out in the first case.

In both calf-rearing cases the remaining calves recovered from mild diarrhoea but growth rates were depressed for several months.

Conclusion

It is my contention that acute, subacute and chronic BVD/mucosal disease is under diagnosed in cattle in New Zealand. In most cases it is possible to find significant numbers of cattle with positive antibody titres to BVD. The major difficulty with this disease is the cost of finding the animals that are BVD antigen positive. In most herds the prevalence of these is only 1-2%.

The immunosuppressive nature of the virus makes it is possible that many other causes of calf ill-thrift such as pneumonia, coccidiosis, yersiniosis and parasitism may have their initial genesis in the presence of the BVD virus in the herd.

What role does bulk milk testing, blood testing herds to identify P.I. animals, and vaccinations have in eliminating this disease?

I look forward to the following papers and hope they will further unravel the complex aetiology of this disease. I also hope they will offer some practical help in the approach to this problem in New Zealand.

References

1. Blood, Radostits, Henderson - Veterinary Medicine (8th Edition)
2. Hill, F :Pers.com.