

Salmonella in Cows

Jeremy Cullwick

Veterinary Services (Dannevirke) Ltd, 9 Gordon Street, Dannevirke

Introduction

During the Spring of 2009 one of our local dairy farmer clients experienced a very severe outbreak of Salmonellosis in his Friesian cows.

Whilst Salmonellosis in individual dairy cows is not uncommon, the morbidity and mortality rate were high in this case. The duration of this outbreak was also long, lasting at least three months.

Background

The dairy farm milks a total of 350 Holstein Friesian dairy cows through a 35 aside Herringbone shed. There are 250 Spring calving cows and approximately 100 Autumn calving cows.

He has a feed pad beside the dairy shed. It is a concrete pad with 2 rows of concrete bins running down the center.

During the spring this year the cows are fed approximately 30% of their total DM as maize silage and/or palm kernel. The other 70% of the cows diet is made up of pasture.

40 replacement heifers are calved in the spring after returning from grazing four weeks prior to the planned start of calving. They had been introduced to maize silage on return to the farm and were being fed it through the transition stage and right on through lactation.

Calves are reared in a shed nearby.

Case History

I was initially called to examine two unwell 2 year old Friesian dairy heifers. One heifer had calved three days earlier and one was several days off calving.

They were noticed as being unwell because they had lost condition, were inappetent and were scouring. Their udders had been sprung right up with recent and/or imminent calving but had rapidly diminished to very little.

Clinical Examination

One heifer had an elevated temperature of 40 degrees, the other was normal.

Dehydrated

Fetid diarrhoea

Differential Diagnoses

1. Salmonellosis
2. Acidosis
3. BVD
4. Ostertagiosis

Took faecal sample and sent for culture.

Treatment

These heifers were treated initially with:

Oxytetracycline (Bivatop®) was given 1ml/10kg IM
Meloxicam (Metacam®) was given 2.5ml/100 kg S.C.
Abamectin (Genesis®) pour-on was applied.

The heifers were removed from the diet of maize silage and placed on hay and grass only.

Laboratory Results

Heavy growth of Salmonella, later confirmed by ESR Enteric Reference Laboratory as being Salmonella typhimurium.

At this stage the clients were advised of the results and advised that vaccination of the herd would be a good idea. The owners did not act on this advice.

Three weeks on

Three weeks later another vet in the practice was called to the farm again to examine a further three unwell heifers. These heifers had aborted their calves near full term and were showing the same clinical signs as above. Blood samples from two of the heifers and a faecal sample from one heifer were taken.

The next day I was called out to the property again to find approximately 10 cases of scouring cows. Already having had the initial positive culture of Salmonella typhimurium two weeks previously we initiated vaccination of the cows with Salvexin +B. Two more faecal samples from newly infected cows were taken.

The new cases were appearing in the springer and colostrums herds.

Results of further laboratory work

Neutrophilia in one heifer, increased fibrinogen in both heifers, electrolyte imbalance, non renal azotaemia, hyperbilirubinaemia. BVD Ag negative, Pepsinogen levels were normal.

The three faecal samples cultured two moderate growths of *Salmonella typhimurium* and a light growth of *Salmonella typhimurium*.

Treatment and Prevention

We were comfortable with our diagnosis. The clinical signs and positive cultures fitted with an outbreak of Salmonellosis due to *Salmonella typhimurium*.

The cows had their first inoculation of Salvexin +B. The sensitiser was given 4 weeks later.

Initially treatment of clinical cases was with injectable oxytetracycline (Bivatop®), oral sulphonamide and kaolin (Kaomide®) powder and oral electrolytes. The oral Kaomide® powder and electrolytes were administered using an Aggers cattle pump®. Some cases treated by vets were given IV hypertonic saline. Ketoprofen (Ketofen®) was used as well. After two months we changed our parenteral antibiotic treatment to use trimethoprim sulphur (Tribrissen®) instead of oxytetracycline.

The farmer was advised to isolate clinical cases. He was also advised to feed maize silage and palm kernel to these clinical cases in the paddock in order to keep them off the feed pad as much as possible.

The Outcome

There were 40 clinical cases out of 350 cows over three months which was a morbidity of 11.4%. Ten of these 40 clinical cases died. Therefore 25% of those cows treated as described above ended up dying of Salmonellosis. 10 deaths out of a total 350 cows was a 3% mortality rate.

Surviving cows were in very poor condition with decreased milk yields for the rest of the season.

A frustrated farmer and veterinarian.

Discussion

Source of Infection

There has been speculation from different parties as to where the *Salmonella* came from to result in this outbreak. There are three possibilities below, but the original source cannot be identified:

1. Heifers returning back on to the farm from grazing carrying *Salmonella typhimurium* was a possible source of infection. The first five cases of Salmonellosis were in the heifers. The heifers could have been carrying the bacteria combined with a relaxation in immunity due to the stress of transport, the fact they were close to calving, and their change in diet. The heifer/s then develops a clinical infection shedding millions of bacteria into the environment.
2. It could have come in with the maize silage or palm kernel. The feed could have been contaminated prior to arrival or even after arrival through birds and/or vermin, or other cows.
3. The *Salmonella typhimurium* was already on the farm being carried by a carrier cow/s. These cow/s then started to shed the bacteria into the environment with a waning of immunity coinciding with calving. The heifers were naïve and the most stressed and therefore succumbed to clinical infection first.

What likely contributed to the spread within the herd and the high morbidity?

The initial two clinical cases of Salmonella in the heifers has resulted in the shedding of millions of bacteria from their faeces. This has been over different parts of the farm- springing mob/paddock, feed-pad, milking shed, and races. The rest of the herd has then come into oral contact in these locations on the farm.

Salmonella typhimurium has been reported to be very pathogenic.

The heifers and other cows in the herd could have had low natural levels of immunity to the Salmonella typhimurium and therefore were naïve and susceptible to infection. They then one by one started to succumb to clinical infection as they were exposed to such a high level of challenge due to the sheer numbers of bacteria in the environment.

Salmonella is spread in water. The topography of the farm – low lying wet Manawatu loam with drains running through the farm would have assisted water spread of bacteria once shedding began from clinical cases.

The feed pad in the spring is likely a factor assisting in the continuation of this outbreak. The cows spend possibly up to 3-4 hours on this pad. This is an ideal opportunity for spread and contamination with faecal to oral transmission. It is easy for feed to be contaminated in the big open feeding troughs. The change in diet in these heifers may also have been sufficient to contribute to the start of the outbreak.

Effluent spraying on paddocks. This is an excellent way to distribute bacteria over the paddocks close to the shed. Then there is further spread by paddock run-off and/or drains.

Vaccination

Vaccination in the face of disease is always problematic. The cow's immune system is under challenge already, and therefore does not develop as good immunity following vaccination as a healthy animal.

The salmonella vaccine currently available to us only provides strong systemic immunity, not good mucosal immunity in the lining of the gut.

The immunity provided by a killed vaccine is not as good as a live vaccine.

The vaccine reduces the amount of bacteria shed in a clinical case and also reduces the severity of the disease in a clinical case. Therefore it reduces the amount of environmental contamination and improves the likelihood of a cow surviving a clinical case when it is infected.

I think that whilst the vaccine has its limitations it has helped in this situation and I would continue to advise other farmers to vaccinate again in the same situation.

I advised the farmer to vaccinate the yearling heifers fully with a sensitiser and booster now and then a booster prior to their return to the farm. The calves were to be vaccinated with a sensitiser and booster at the same time as the lepto vaccination. All heifers and cows, having received the initial 2 vaccinations should receive **annual boosters** thereafter when they are dried off. This is to obtain maximum immunity possible prior to the subsequent calving. I believe this will be important in helping reduce any further outbreaks of clinical Salmonellosis on this farm.

Natural Immunity

There has been a high level of environmental contamination on this farm. This natural exposure itself will have provided the herd with a good level of natural immunity in addition to the vaccine. Herds seroconvert within 30-60 days when exposure occurs. The most invasive serogroups appear to result in some of the most consistent seroconversions. Once herd seroconversion occurs, clinical cases subside and are largely limited to new animals unless heavy exposure continues.

References

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