When something very fishy is going on… An unexpected case of BVD

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FARM CONTEXT

A dairy farm with an average of 50 Prim' Holstein and a few Normandy cows, managed single-handedly by the farmer, and a milk quota of 336,000 litres. The farm is rather well kept; a barn with straw bedding and no apparent problems. Nevertheless, three abortions were noted in the previous months. Serological tests for Brucellosis, BVD and Neosporosis were all negative. Q-fever serological tests all came back positive. PCR analysis on tank milk confirmed the passage of *Coxiella burnetii* in the herd.

With regard to BVD, biennial analyses showed no circulation of the virus in the dairy herd.

CASE REPORT

On June 11, 2019, the farmer called for a 26-month-old Prim' Holstein heifer that had calved about 15 days previously with a drop in milk production and a decrease in appetite. Although calving had been uneventful, the heifer had a retained placenta. She was also hyperthermic and did not ruminate. The farmer had initiated treatment based on penicillin and dihydrostreptomycin (INTRAMICINE®), tolfenamic acid (TOLFINE®) and an oral food supplement to boost rumination (RUMI C3®). Given the lack of improvement, he decided to call us.

During the first visit, the cow was hyperthermic (40°C), had pink mucous membranes and no audible ruminal contraction. She presented with metritis and increased respiratory sounds with no added noise. Urinalysis revealed aciduria (pH 5).

Given the time and the clinical picture, Ehrlichiosis was suspected. The heifer was drenched and put on oxytetracycline (10 mg/kg) (OXYTETRACYCLINE® 10%). The farmer was asked to continue antibiotic and anti-inflammatory treatment as well as RUMI C3®.

A biochemistry panel was carried out to assess the inflammatory state of the animal. The globulins were found to be markedly increased (TP: 81 g/l; Alb: 27 g/l; Glob: 54 g/l and Alb/Glob: 0.5).

On June 14, the farmed called back as the state of the animal was degrading. A new examination was carried out, five days after the original onset of clinical signs. The cow was dehydrated and the mucous membranes slightly icteric. The animals was now normothermic (38.2°C) but still presented with a complete lack of rumination and profuse diarrhoea of two days. The animal showed digestive pain, tachycardia and tachypnoea. Urinalysis revealed aciduria (pH 5), proteinuria (+++), and traces of blood. Clinical signs also included cauda equina syndrome that had not been reported three days earlier.

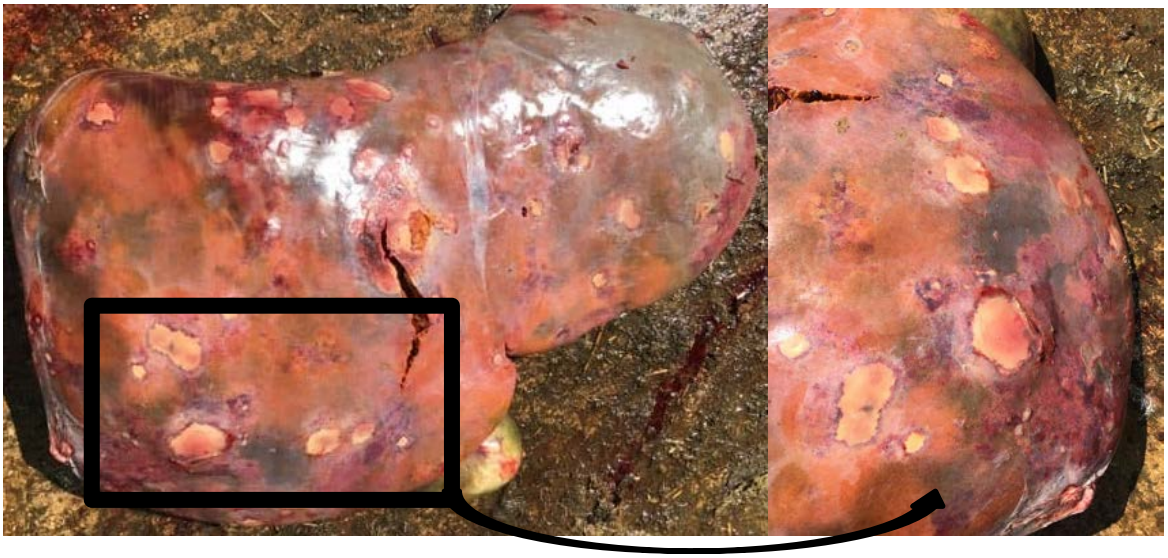
The clinical picture, the colour of the mucous membranes and the urinalysis were suggestive of renal and hepatic insufficiency, confirmed by biochemical analysis (urea 1.16 g/l, creatinine 16.7 mg/l; ASAT > 1083 U/l ; GGT 95 U/l). However, the profuse diarrhoea and the rest of the clinical picture confounded us. There can be many causes, including bacteraemia, salmonellosis, foreign body peritonitis, leptospirosis and toxaemia. Infusion therapy seemed vital but was refused by the farmer who did not want to waste further time and expenses on an animal with a very poor prognosis.

Symptomatic treatment was based on clay, RUMI C3®, antispasmodics (scopolamine and metamizole: ESTOCELAN®), and, as a last resort, antibiotics were changed to trimethoprim sulfonamide (SEPTOTRYL®).

The animal died a few days later, and it was decided to carry out an autopsy on the farm to establish a diagnosis.

POST-MORTEM EXAMINATION

The carcase was slightly icteric and the digestive tract nearly empty. The organs were inspected one by one, as shown below.



Figures 1 and 2: Enlarged liver with dozens of whitish plaques of 1-5 cm in diameter, increased consistency and surrounded by haemorrhagic halos: Severe diffuse multifocal necrotising hepatitis associated with severe steatosis, suggestive of hepatitis due to *Fusobacterium necroforum*

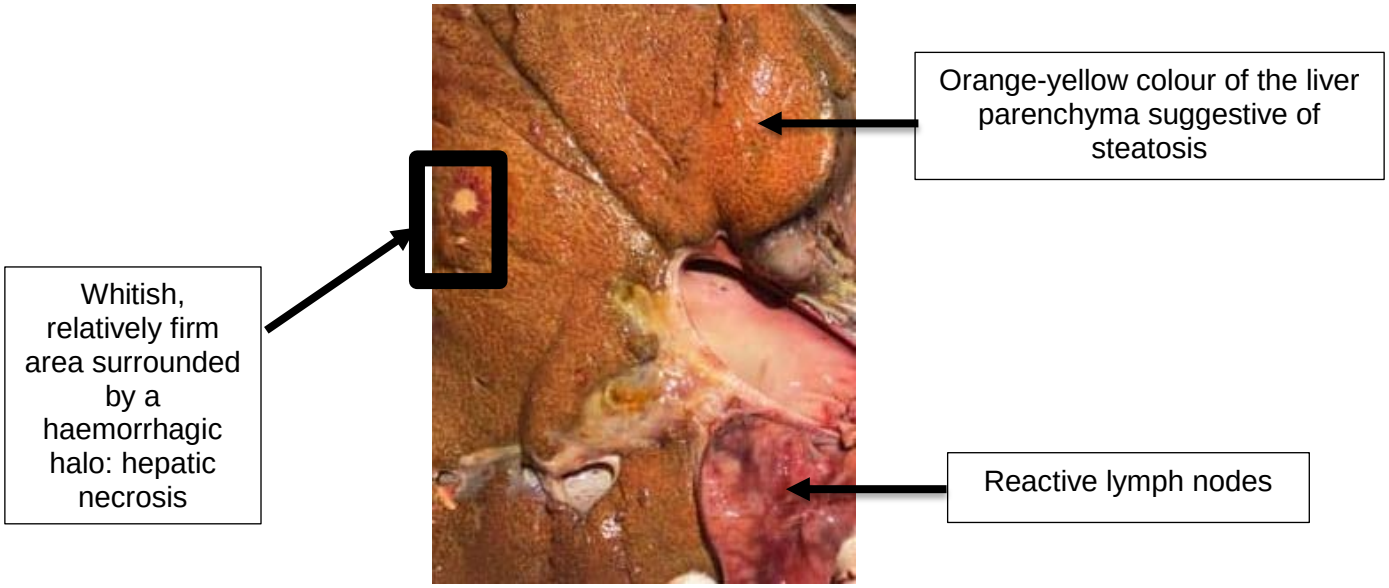
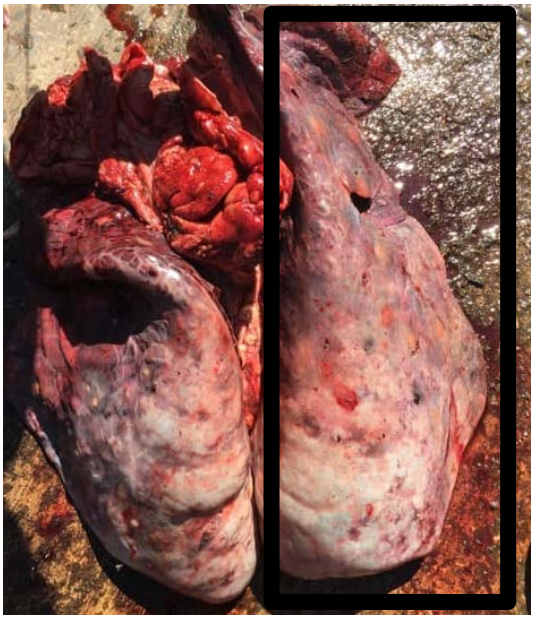
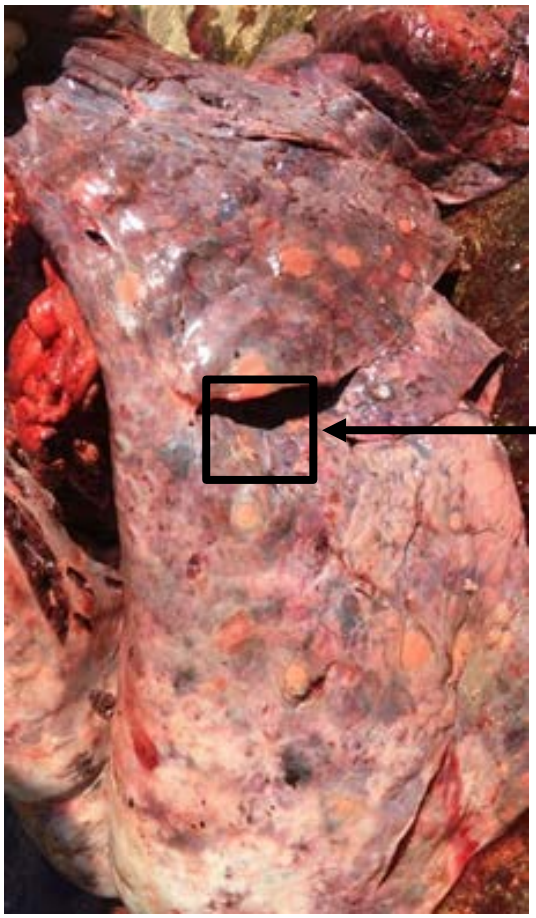


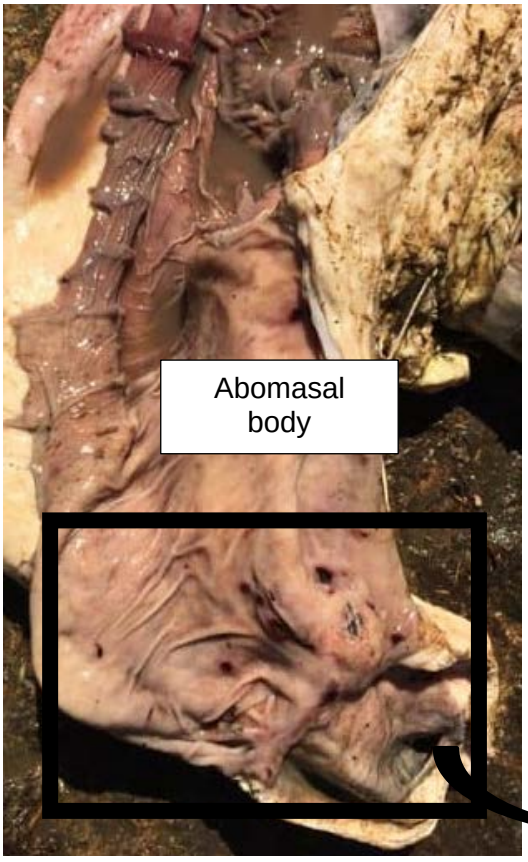
Figure no. 3: Cross section of the liver highlighting steatosis and areas of necrosis



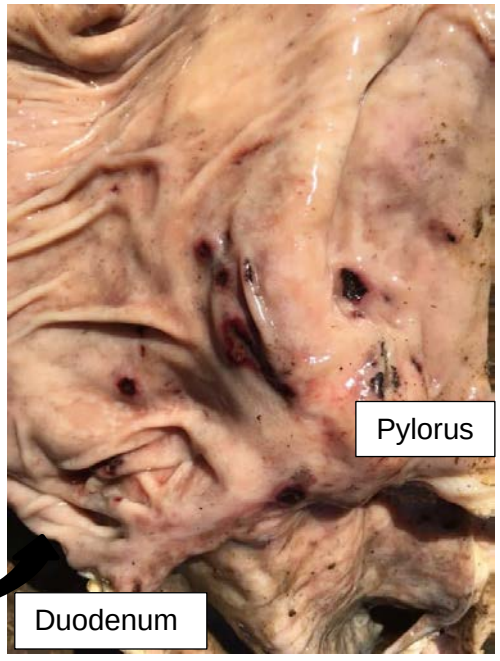


Figures 4 and 5: Lung with whitish areas similar to those seen in the hepatic parenchyma highlighting the phenomenon of bacteraemia

Figure 6: Dozens of linear, 2-5-mm-long chronic scratch-like ulcers in the oesophagus



Figures 7 and 8: Acute haemorrhagic and congestive pyloric ulcers



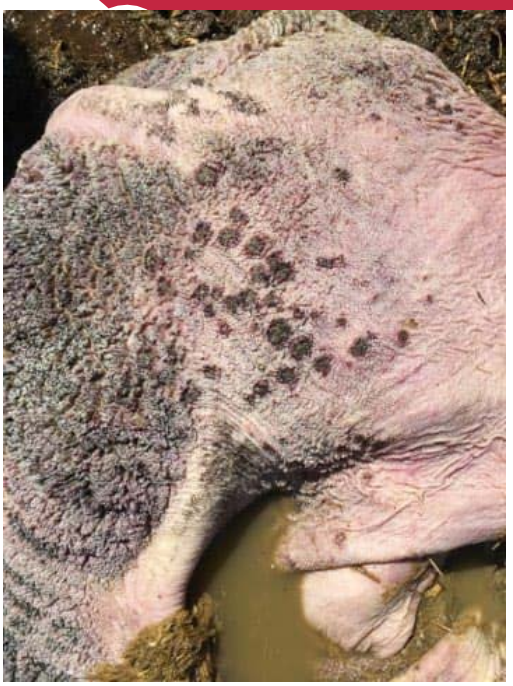


Figure 9: Dozens of round plaques, 3-5 cm in diameter, greyish brown with a necrotic centre, well delimited by a surrounding haemorrhagic halo: Mycotic ruminitis

DISCUSSION AND CONCLUSION

The lesions are surprising, but confirm our first hypothesis of bacteraemia. However, we still find it difficult to explain the oesophageal ulcers. They are strangely reminiscent of BVD but yet nothing on the farm refers to it. There is no health problem and the tank milk indicators have been at 0 for years. Nevertheless, the spleen was submitted for investigation for presence of the BVD virus by PCR. The results came back positive, the animal was therefore viraemic. Was this an immunotolerant persistently infected (PI) or a transiently infected (TI) animal? A Ct of 33 suggests that it is probably a TI. It would have been interesting to be able to test the calf of this cow but unfortunately it had already been sold. The Livestock health protection group (GDS) was contacted to put this farm under BVD surveillance as soon as possible. This consists in carrying out an analysis on the whole herd: PCR on tank milk and serology or PCR on all the other animals present.

Five calves under 6 months of age were found to be viraemic but no animal over 6 months of age had BVD antibodies. The five positive calves were re-tested 5 weeks after the first analysis. Three were still viraemic but only one of them had a Ct < 25. We suspect that the two others were transient viraemic (Ct = 31 and 42 during the first analysis and Ct = 36 and 39 during the second) although the viraemia seemed to last very long. When the animals were isolated and re-tested 9 weeks after the first analysis, only one viraemic animal remained (Ct = 24 for all three analyses). It was found to be a PI animal and was slaughtered.

BOVELA vaccination was implemented very quickly for the entire herd and youngstock to limit the creation of new PIs. Ear notch tissue was taken from all calves born during one year for E0 antigen immunoassay. Given the severity of the clinical signs that were probably secondary to immunosuppression caused by the BVD virus and the (exceptionally long) persistence of transient viraemia in certain calves, it was decided to have the BVD typed. We suspected BVD type 2 knowing that it is circulating at the borders of the Mayenne department, but it turned out to be type 1. This case kept us on the edge of our seats for a few weeks. It reminds us of the importance of post-mortem examination which turned a routine case into a true farm case. Thanks to the excellent collaboration with the farmer, all of the practice veterinarians, the GDS, the diagnostic laboratory and Dr. Laetitia Dorso (specialist in pathology), the economic repercussions were less than they could have been. Only one single new-born PI case has been detected since, 8 months after the clinical episode.