

The many faces of BVD virus

Bovine viral diarrhoea (BVD) has once again been receiving a lot of attention recently. The implementation of a national control programme has been announced in the Netherlands. The BVD type 2 outbreaks in 2013 and in the first half of this year have shown that BVD still represents a real risk.

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In America, BVD type 1 and type 2 are equally prevalent. In the EU, the prevalence of type 2 is less than 10 percent. Of type 2, subtype 2c is most common in Germany and was also the cause of the recent outbreaks in the Netherlands. Subtype 2a has also been detected in Germany. The most common form of the BVD virus in the EU is type 1b, followed by type 1d.

High temperature and diarrhoea

BVDV type 2 is associated with serious clinical symptoms such as fever, bloody diarrhoea and mucosal lesions. But severe, acute outbreaks on dairy farms in Belgium, in which BVDV type 1b played a role, have also been described by Laureyns. One outbreak affected one farm's fresh cows. The animals became seriously ill with symptoms of high fever, coughs, watery diarrhoea, udder inflammation and some animals died. Further research revealed that a PI (carrier) animal had been kept next to the calving pen at the time of the outbreak. It is assumed that cows have lower resistance during the transition period, which may have played a role in the severity of the symptoms. Last year, at least thirteen dairy farms in Germany had a type 2c outbreak. On these farms, there were clinical problems across multiple age groups simultaneously. The most distinct symptoms were bloody diarrhoea; respiratory problems accompanied by a fever; bleeds and mucosal lesions in the mouth and the gastrointestinal tract; and death. Initially, it was the calves that became ill, then later the heifers and the dairy cattle. A decrease in milk production and abortions were observed in the cows. Notably, the animals had a prolonged viraemic phase and shed large amounts of the virus over an extended period, sometimes in excess of eight weeks. The virus disseminated quickly within each farm, so a lot of animals were affected within a short time. The role of PI animals could not be established in these outbreaks. The prolonged shedding of the virus by animals with a transient infection and the speed with which it spread are likely to have been determining factors.

Extra factors

The severity of the clinical symptoms of BVD is determined by virus-related factors and cow-related factors. The risk of infection (the amount of virus), the length of exposure, the genotype (type 1 or 2), the subtype (1b, 2c, et cetera), the biotype (non-cytopathic (ncp) or cytopathic (cp)) and the virulence of the virus all have an influence. In addition, the animal's BVD immune status (naive or not), the general condition, the immune response and the gestational stage all play a role in the pathogenesis.

BVD virus is shed in almost all bodily fluids such as ocular discharge, nasal discharge, saliva, sperm, urine, uterine and vaginal discharge, amniotic membranes and the placenta. A 'Trojan' cow sheds large amounts of the virus for 24 to 48 hours after calving a PI calf, from the uterus as the amniotic fluid is released and the placenta comes away.

Transmission

Infection with the BVD virus generally occurs through horizontal transmission, whereby the virus usually enters through the nose or mouth. Transmission can also occur via materials such as gloves or instruments, through housing and through the air. Especially if large amounts of the virus have been shed in the environment by persistently infected animals. The BVD virus can survive for up to two weeks in the environment. BVD virus can be transmitted through covering, artificial insemination and embryo transplantation. Vertical transmission occurs through infection of a naive cow during gestation, in which the foetus becomes infected through the transplacental route, creating a PI animal. The literature describes one case of a twin birth, in which one animal was a carrier and the other calf was not. The gestational period and the stage of immune system development of the foetus at the time of infection, have an effect on the occurrence of persistently infected animals or birth defects. Around 10 percent of carriers are over two years old and can have a calf themselves. A PI animal always produces a PI calf. After entry, the virus first replicates in local lymph nodes, for example in the tonsils. Depending on the virulence of the virus it then disseminates further throughout the body. It spreads to the blood, other lymph nodes, spleen, thymus, skin, cartilage, placenta, fetus and with virulent types also to the gastrointestinal tract, bone marrow and the endocrine tissue. BVDV directly affects the immune system. The virus reduces the function of antigen-presenting cells, inhibits the proliferation of lymphocytes and affects white blood cell function, including the phagocytic activity of the macrophages. The effect on the immune system varies for the two biotypes (ncp and cp). With cp BVDV, infected cells die and they produce factors that also cause the cell death of non-infected blood cells, immune cells and epithelial cells. A field infection with a virulent ncp virus can cause severe lymphopaenia resulting in immunosuppression.

An acute BVD infection therefore causes a decrease in the amount of immune cells and their function, and leucopenia usually develops. The effect on the immune system then leads to secondary disorders.

Negative effect on fertility

The negative consequences of the BVD infection on fertility are also caused by the direct effects of the virus on the ovaries. Acutely infected cattle suffer from inflammation of the ovaries with necrosis of the granulosa cells, which normally play a role in the growth and development of follicles. This has a negative effect on fertility. In addition, BVD infection at the start of gestation leads to early embryonic death and after two months it can lead to abortion or mummification of the foetus, with financial consequences for the company. In addition to the losses resulting from the creation of PI animals and birth defects.

BVD comes in many forms and causes not only clinical symptoms and 'hidden' damage through, for instance, an increased cell count or reduced fertility. A bad reaction to a treatment provided or severe symptoms that do not fit any particular disorder can be caused by a BVD infection. In addition, the virus also influences the animals' well-being and has a negative effect on life expectancy. So there are plenty of reasons why we should tackle BVD: to keep the virus away from your door; to prevent the virus circulating on the farm; and to prevent the effects of a BVD infection.