An outbreak of the peracute form of malignant catarrhal fever in Belgian cattle

Uitbraak van de peracute vorm van boosaardige catarraal koorts bij Belgische runderen

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ABSTRACT

A large outbreak of the peracute form of malignant catarrhal fever in cattle in Belgium is described. The main symptoms were nervous signs and high fever. Over a four-month period, 13 Belgian Blue yearlings and one cow died. Mortality was 16.3% of the herd. The diagnosis was confirmed by identifying ovine herpesvirus-2 DNA by PCR, both in the affected cattle and in the sheep on the farm. This case report illustrates the increasing importance of malignant catarrhal fever as a herd problem and the occurrence of the peracute form, as also reported in other European countries. It also illustrates the importance of malignant catarrhal fever as a differential diagnosis for nervous disorders in cattle.

SAMENVATTING

In deze casuïstiek wordt een grote uitbraak van de peracute vorm van boosaardige catarraal koorts bij runderen in België beschreven. De belangrijkste symptomen waren zenuwsymptomen en hoge koorts. Over een periode van 4 maanden stierven 13 Belgisch Witblauwe jaarlingen en één koe. De mortaliteit op het bedrijf bedroeg 16,3%. Het oviene herpesvirus 2 werd bij de zieke runderen en schapen op het bedrijf met PCR aangetoond. Deze casuïstiek illustreert het toenemende belang van boosaardige catarraal koorts als bedrijfsprobleem en het voorkomen van de peracute vorm, zoals ook waargenomen in andere Europese landen. Het belang van boosaardige catarraal koorts als een differentiaaldiagnose voor zenuwsymptomen bij rundvee komt eveneens aan bod.

INTRODUCTION

The prevalence of sheep-associated malignant catarrhal fever (SA-MCF) in cattle is low in Belgium. Few case reports exist, and all of them involve the head and eye form of the disease (Muylle *et al*., 1985; Desmecht *et al*., 1999; Deprez *et al*., 2004). Recent surveillance reports, however, show an increase in outbreaks of MCF in countries such as England (VLA, 2006) and Scotland (SAC, 2007). SA-MCF is traditionally known as a highly fatal disease in a small number of animals, but over the last decades large outbreaks of the disease have been reported, mainly in countries with a high density of sheep such as Great Britain (Foster, 1983; Sharpe *et al.,* 1987; Bonn, 1990; Holliman *et al*., 2007), Ireland (Hamilton, 1990; Collery and Foley, 1996), Spain (Yus *et al.,* 1999) and New Zealand (Thompson and Beckett, 1987). Recently, a large outbreak was also reported in the Netherlands (Van Wuijckhuise-Sjouke and Knibbe, 2007). In most outbreaks the clinical presentation of MCF is the typical head and eye form. Outbreaks of the peracute syndrome are rare. In 2005-2006, five outbreaks of the peracute form were reported in the United Kingdom (Holliman *et al*., 2007). In this article the first large outbreak of the peracute form of MCF in Belgium is described.

CASE REPORT

Case history

In April 2008 two Belgian Blue yearlings were presented at the Ghent University Clinic of Large Animal Internal Medicine. In the past three months the owner had lost 7 yearlings to an aggressive disease characterized by high fever, staggering gait, trembling, conjunctival congestion, lateral recumbency and death within 3-36 hours after onset of the symptoms. The herd consisted of 86 animals (41 cows, 3 bulls, 31 yearlings and 12 calves). In addition to the cattle, 72 sheep (27 adults and 45 adolescents in two separated herds) were present. During the outbreak most of the cattle were housed in one stable, with the yearlings distributed over 12 pens (6-8 animals in a pen). Together with the cattle, a group of adult sheep was housed in a corner pen of that stable. The adolescent sheep remained at pasture and could enter a second barn in which the remaining calves were housed. Direct contact between these sheep and the calves was possible. Only two bulls and one ram had been bought in the past three years; the last purchase had been made one year before. On December 23rd, an 8-month-old bull from the pen adjacent to the sheep pen showed a staggering gait and melaena. The animal died the next day. Three days later, a second bull from the same pen had identical symptoms and died. No other animals were sick at that time. On January 16th, a third animal became sick, this time in a pen 10 meters from the sheep pen. Again the animal died within one day. No new cases were seen until the beginning of April, when a bull and a heifer of the same age (8 months) died in the second pen. Four days passed, and on April 4th two animals (one bull and one heifer) became sick again in the first pen. The bull died on the farm and was presented for necropsy. The heifer was still alive upon admission to the clinic. The local practitioner had treated the animals with danofloxacine (Advocin 180®; Pfizer), flunixine meglumine (Finadyne®; Shering Plough), florfenicol (Nuflor®; Shering Plough) and dexamethasone (Rapidexon®; Eurovet), though without any improvement. The current status concerning Bovine Viral Diarrhea virus was unknown.

Clinical examination

The Belgian Blue heifer (1 year, 257 kg) was depressed and demonstrated a stiff gait, high fever (40.6°C), tachycardia (120/min) and tachypnea (60/min). The conjunctivae were severely congested, the scleral vessels were clearly visible, and serous lacrimal fluid was present. No corneal opacity was seen. The skin turgor was normal and the lymph nodes were only moderately enlarged. A very small amount of bilateral mucopurulent nasal discharge was seen. Abnormal breathing sounds were prominent on auscultation. Oral inspection demonstrated congestion of the buccal papillae and erosions on the hard palate. There were no petechiae visible on the mucosae. Ruminal contractions were still present and intestinal sounds were heard. The animal had a foul smelling, yellow colored diarrhea and the urine was reddish. Neurological examination showed no cranial nerve deficits. The gait was insecure and slightly uncoordinated. Tremor and a high muscle tone were present in the limbs. Ultrasonographic examination of the abdomen and thorax did not reveal additional information. Leukopenia (2.0 x 10^9/L (6.0-9.0)) and a normal packed cell volume (PCV: 30% (25-35)) were seen on hematology. Biochemistry showed mild hyperbilirubinemia (10 µmol/l (2.5-6)) and a slight increase in creatinine phosphokinase (CPK: 213 U/l (<150)). Semi-quantitative urine analysis with a urine stick (Combur 10 Test; Roche) showed the discoloration of the urine to be caused by hematuria (> 250 red blood cells per µl). Mild glucosuria was also present (50 mg/dl). The next day the animal was found in lateral recumbency and was euthanized on ethical grounds. Two days later the owner reported a new case and a herd visit was performed. A 7-month-old bull, which had become sick in the second pen, was in lateral decubitus and showed tetany and mild opisthotonus (Figure 1). Fever (40.4°C) and tachycardia were present. The animal overreacted on the menace and palpebral reflex during the neurological examination. Oral inspection was impossible due to the tetanic state of the jaw muscles. The tail tone was normal and no diarrhea was seen. Blood for virological examination was taken from this animal, 5 herd mates and 5 sheep. The next morning the animal was found dead and it was transferred to the Department of Pathology. On April 14th, a 3-year-old Belgian Blue cow developed conjunctivitis and petechiae on the buccal mucosae. The animal died within a day.

Necropsy

Necropsy was performed on 4 animals: three youngsters and the 3-year-old cow. The carcasses were moderately dehydrated. In all cases, conjunctivitis was

Figure 1. A 7-month-old bull (case 8) in the terminal stadium. The animal is in lateral decubitus and demonstrates tetanic limbs and mild opisthotonus.

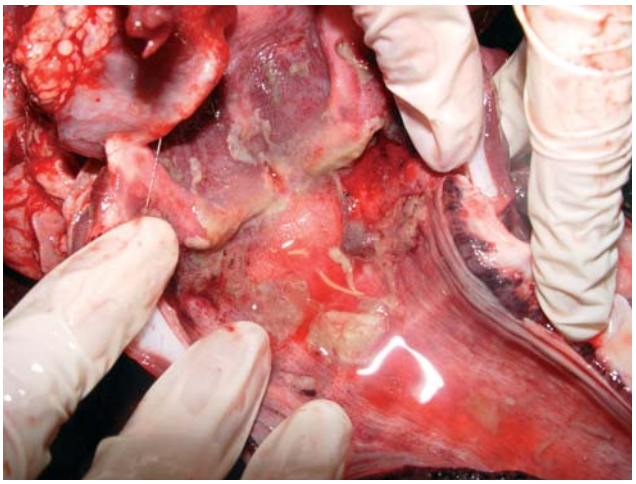

Figure 2. Mucopurulent exsudate on the arythenoids and proximal trachea in case 7.

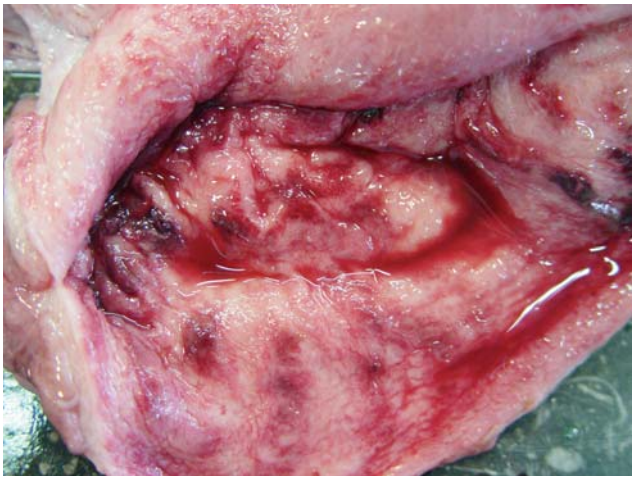

Figure 3. Edema, multiple hemorrhages and hemorrhagic urine in the bladder of case 6.

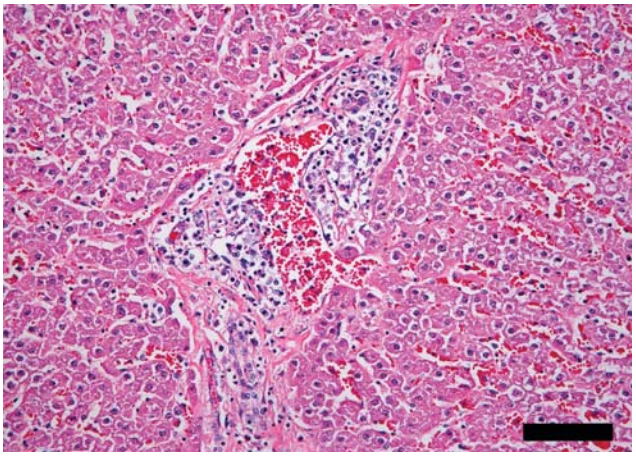

Figure 4. Fibrinoid necrotizing vasculitis with perivascular accumulation of mainly mononuclear cells in the portal triads of the liver. HE stain. Original magnification 100x (bar= 100µm).

evident, presenting as mucopurulent in the cow and catarrhal in the other animals. No corneal opacity was seen. Multiple petechiae and erosions were present on the muzzle and nares and in the buccal cavity. The nasal and laryngeal mucosa were heavily congested and covered with a mucopurulent exsudate (Figure 2), which was less pronounced in the trachea. Marked alveolar and interstitial emphysema was seen in the lungs. One animal showed small linear erosions in the cranial portion of the esophagus. There was a generalized hyperemia in the forestomachs. Multiple ulcerative lesions were found in the pyloric region of one animal. Hemorrhagic enteritis, often segmental, was prominent in all animals. Both liver and spleen were mildly swollen. Peripheral and intestinal lymph nodes were moderately enlarged. The urinary bladder mucosa had petechial and ecchymotic hemorrhages with multifocal erosion of the epithelium (Figure 3), and hematuria was present.

Histopathology of the oral lesions consistently showed a perivascular accumulation of mainly mononuclear cells around the walls of medium and small-sized vessels with apoptosis of epithelial cells secondary to ischemia. There was a marked lymphoid cell hyperplasia in the periarteriolar sheaths of the spleen. Perivascular accumulation of mononuclear cells with focal fibrinoid necrotizing vasculitis was present in the portal triads of the liver (Figure 4). In the brain, a nonsuppurative meningoencephalitis with necrotizing vasculitis was present. Cerebral vessels showed fibrinoid necrosis with severe infiltration of predominantly lymphocytes in the vessel wall and adventitia. There was a diffuse edema of the Virchow-Robin space and the meninges were infiltrated by mononuclear cells.

Virology

PCR for Ovine herpesvirus 2 (OvHV-2) on EDTA blood of two youngsters with clinical symptoms was positive (Center for Research in Veterinary Medicine and Agricultural Chemistry, Brussels). PCR for BTV-serotype 8 was negative in these animals. In order to confirm the sheep as the source of OvHV-2 infection 5 sheep (2 adults, 3 yearlings) were sampled. One adult and two yearlings were PCR positive. Of the five apparently healthy bovines, only one animal – from the second pen – was positive in the OvHV-2 PCR. This animal developed identical symptoms and died two weeks later. In addition, 6 youngsters, aged between 6 months and 1 year, were tested for Bovine Viral Diarrhea virus (BVD) antibodies and no antibodies were detected.

Further development

Within the next two weeks, another four yearlings died. In total, 14 animals or 16.3% of the livestock died. Mortality among the youngsters was 45.0%. Before confirmation, the owner had sold his adolescent sheep. After the positive test results the remaining

sheep were slaughtered. The last animal died at the end of April. Since then no new cases have been observed.

DISCUSSION

Sheep-associated MCF is caused by the ovine herpesvirus-2 (OvHV-2). Sheep and probably also goats are asymptomatic carriers of the virus (Brenner *et al*., 2002). In recent years, the understanding of the pathogenesis and epidemiology of MCF has rapidly increased with the development of molecular technologies (Li *et al.,* 2004). Recent reviews concerning etiology, epidemiology, pathogenesis, clinical symptoms, pathology and diagnosis are readily available (Ackermann, 2006; Russell *et al*., 2008; Pardon *et al.,* 2009).

Five clinical presentations, namely the head and eye, the intestinal, the nervous, the cutaneous and the peracute forms, are distinguished, but none of them is pathognomonic by itself (Smith, 2002). Recent observations and the availability of diagnostic tests have made it clear that MCF has a clinical spectrum ranging from subclinical infection to clinical disease with the possibility of recovery or chronic infection (O'Toole *et al*., 1997; Penny, 1998). The peracute form is characterized by pyrexia, depression, diarrhea, dysentery and death within 24-72 hours (Smith, 2002). Few reports on this form of the disease exist, possibly because outbreaks of this rare form have not been recognized in the past (Gibson, 2007). Foster reported an outbreak of the peracute form in the United Kingdom (UK) in 1983. In 2005 and 2006, five outbreaks of the peracute form occurred in the UK (Holliman *et al*., 2007).

The Belgian outbreak distinguishes itself from the reported outbreaks in the UK in two ways. Firstly, the main clinical signs were neurological symptoms. Neurological symptoms usually occur in the terminal stage of the head and eye form (Smith, 2002). Recently, an outbreak of MCF involving predominantly nervous signs occurred in very young calves in the UK (Mitchell and Scholes, 2009). In all these outbreaks, as in this report, corneal opacity was not a consistent finding (Bonn, 1983; Holliman *et al.*, 2007; Mitchell and Scholes, 2009). The presence of hematuria or hemorrhagic cystitis on necropsy seems to be a hint for the diagnosis of peracute MCF. Secondly, it is remarkable that the clinical evolution was hyperacute in all cases (death within 2 days). In the literature, large outbreaks have usually been characterized by first cases that died within a few days, followed by cases with slower progression of the disease (usually the head and eye form) (Holliman *et al.,* 2007; Mitchell and Scholes, 2009). Although most MCF outbreaks occur in adult cattle, there is no clear age susceptibility. One description of an outbreak in Saudi Arabia even reports MCF in calves as young as 1 month (Elzein *et al.,* 2003). It is remarkable, however, that 5 of the 6 reported outbreaks of the peracute form occurred in young (<24 months) animals, as is the case in this report (Holliman *et al.,* 2007; Mitchell and Scholes, 2009).

In recent years, more reports on MCF are being published throughout Europe. In Belgium, 11 cases/outbreaks of MCF have been diagnosed at Ghent University during the period 1997-2008 and two have been reported in veterinary practices (J. Wullepit, personal communication, 2001; G. Lafaut, personal communication, 2008). Most of the cases have involved a single animal with the head and eye form (Muylle *et al.,* 1985; Desmecht *et al.,* 1999; Deprez *et al.,* 2004). An overview is given in Table 1. Recent reports from the UK mention an increase in the total number of individual cases and the appearance both of larger outbreaks (VLA, 2006; SAC, 2007) and of cases involving the peracute form (Holliman *et al.,* 2007). The reason for this evolution is not clear. The availability of better diagnostic techniques in recent years may have had an impact on the number of diagnoses. A further increase in the number of cattle being housed together with sheep seems unlikely in view of the continuing growth of modern farming practices. However, the co-stabling of large numbers of cattle and sheep might explain the apparent rise in larger outbreaks, due to the

Table 1. Overview of malignant catarrhal fever outbreaks reported at Ghent University between 1997 and 2008.

Year	Region	Month	Number of cases	Clinical presentation
1997	Walloon Brabant	July	1	Head and eye form
2001	NA	NA	1	Head and eye form
2003	East Flanders	April	1	Head and eye form
2004	East Flanders	February	1	Head and eye form
2004	East Flanders	March	1	Head and eye form
2006	Antwerp	February	7	Head and eye form
2006	East Flanders	July	1	Head and eye form
2006	East Flanders	June	1	Head and eye form
2007	West Flanders	July	1	Head and eye form
2007	East Flanders	December-April	14	Peracute form
2008	West Flanders	May	2	Head and eye form
2008	East Flanders	June	1	Cutaneous form
2008	East Flanders	September	1	Peracute form

NA: No detailed information available

higher viral load in these stables. The apparent increase of larger outbreaks could be explained by a change in host or pathogen factors. A genetic evolution of the virus towards a better adaptation to cattle cannot be substantiated at the current time. Cattle seem to be only moderately susceptible to OvHV-2, compared to the highly susceptible bisons and sheep, which only show symptoms at very high infectious doses (Taus *et al.,* 2005, 2006; Li *et al.* 2005, 2006; Berezowski *et al.,* 2005). Immunosuppressive factors could increase the susceptibility of cattle. For example, copper deficiency (Otter *et al*., 2002), bracken fern toxicosis (Twomey *et al*., 2002), BVD virus (Sharpe *et al.,* 1987) and periparturient immunosuppression (Otter *et al.,* 2002) have all been linked to MCF outbreaks. Finally, the existence of different genetic predispositions between different lines could possibly be another contributing factor, as susceptibility for MCF has been linked to the polymorphism of the Major Compatibility Complex IIa in bison (Traul *et al.,* 2007) and a mother/calf relationship was found in nearly half of the affected animals in one outbreak (Otter *et al.,* 2002).

CONCLUSION

This case report documents an increase over the last decade of confirmed MCF cases in Belgium, similar to the situation in the UK. The financial implications of mingling cattle with sheep can be devastating, comparable to mingling cattle with poultry (botulism), with pigs (Aujeszky's disease) or with dogs (neosporosis). Avoiding direct contact between cattle and sheep is the best preventive measure. The report also illustrates the persistent importance of including MCF in the differential diagnosis of neurological problems in cattle.

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Uit het verleden

NIEUWE SLACHTMETHODE MET VOORHOOFDMASKER EN SCHIETPEN (1875)

Begin 1875 verzocht de Gentse burgemeester de Kerchove de Denterghem de directeur van het stedelijke slachthuis een nieuwe slachtmethode met voorhoofdmasker en schietpen uit te proberen. Hij had een verzoek daartoe gekregen van de Maatschappij voor Dierenbescherming en hij stemde volledig in met wat toen al de 'humanitaire' beweegredenen van die vereniging genoemd werd.

De hierna volgende beschrijving (in vertaling) is ontleend aan het *Bulletin Communal de la Ville de Gand* (1875, p. 361-362). Het procédé, op punt gesteld door M. Bruneau, president van het directiecomité van het grote Parijse slachthuis van La Vilette, maakt gebruik van een leren masker op het voorhoofd en de ogen van het slachtdier geplaatst en vastgemaakt met riemen. In het midden is er een ronde opening die doorgang geeft aan een pen, een soort uitgeholde bout die als doorslag (drevel) functioneert. Een hamerslag op de bout drijft deze in de schedelholte en laat de lucht geperst in de uitholling de hersenen samendrukken waardoor het dier ogenblikkelijk bewusteloos neervalt.

Slachtschiettoestel van iets modernere makelij aangedreven met patronen (Museumcollectie Diergeneeskundig Verleden, Merelbeke).

Een afvaardiging van het gemeentebestuur woonde een dergelijk experiment bij en kon met eigen ogen constateren dat er *maximaal* één minuut verliep tot wanneer het dier geen enkel teken van leven meer gaf. Het procédé is erg eenvoudig en geeft volgende voordelen: (1) de slachter slaagt steeds in zijn opzet, (2) het dier lijdt minimaal, (3) de doodstrijd is kort en (4) het vlees bewaart goed. Eén van de belangrijkste slachtersbazen van de stad had de nieuwe methode al aangenomen. Op unaniem voorstel van de leden van de gemeentelijke commissie voor handel en nijverheid samen met deze van volksgezondheid werd het procédé Bruneau via een gemeentebesluit verplicht gemaakt. De slachthuisdirecteur mocht er echter van afwijken in gevallen waarin hij dat nodig zou achten.

Noot van de vertaler: Later werden de pinnen aangedreven met cartouches (ontsteking). Stuiptrekken werd vermeden door een riet in het gat doorheen de schedelholte tot in het verlengde merg te steken. Hiervoor waren gespecialiseerde ervaren slachters van doen.

Luc Devriese