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INFECTIOUS DISEASE

Transplacental Transmission of Ovine Herpesvirus 2 in Cattle with Sheep-associated Malignant Catarrhal Fever

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Summary

Sheep-associated malignant catarrhal fever (SA-MCF) is an important infectious disease of ruminants worldwide that is caused by ovine herpesvirus 2 (OvHV-2). OvHV-2 is transmitted predominantly by contact between infected and susceptible hosts, while the documentation of vertical transmission is rare. This report presents the pathological and molecular findings associated with transplacental transmission of OvHV-2 in cattle. Two Girolanda cows with corneal oedema, lethargy, mucopurulent nasal discharge and ulcerative stomatitis died spontaneously; one of these was pregnant with a 4-month-old fetus. Significant pathological findings included widespread lymphoplasmacytic necrotizing vasculitis and lymphoplasmacytic accumulations in several organs of both cows and the fetus. A polymerase chain reaction that targeted the tegument protein gene of OvHV-2 amplified viral DNA from the brain of the pregnant cow and her fetus, as well as from the kidney of the pregnant cow. The pathological findings observed in the cow and her fetus, together with the presence of OvHV-2 DNA in tissues of these animals, are suggestive of transplacental transmission of OvHV-2 in SA-MCF in cattle.

Keywords: cattle; malignant catarrhal fever; ovine herpesvirus 2; transplacental transmission

Introduction

Malignant catarrhal fever (MCF) is a fatal disease that affects domestic cattle, wild ruminants and occasionally pigs. MCF is caused by members of the genus *Macavirus* (Davison *et al.*, 2009), subfamily *Gammaherpesvirinae* (Russell *et al.*, 2009; O'Toole and Li, 2014). MCF has two principal epidemiological and clinical manifestations. The first is induced by alcelaphineherpesvirus 1 (AlHV-1), which uses wildebeest (*Connochaetes gnu* and *Connochaetes taurinus*) as a carrier and occurs predominantly within the African continent. The second is caused by ovine herpesvirus 2 (OvHV-2), which occurs outside the African continent and affects cattle, bison and deer, although sheep are the recognized carriers (Brown *et al.*, 2007; Russell *et al.*, 2009). Consequently, these manifestations are referred to as wildebeest associated (WA-MCF) and sheep-associated (SA-MCF) MCF, respectively (Brown *et al.*, 2007). However, other forms of MCF are described in several ruminant hosts (O'Toole and Li, 2014).

SA-MCF occurs worldwide (Russell *et al.*, 2009), affects a wide range of mammalian hosts (O'Toole and Li, 2014), and is endemic in Brazil, occurring in all geographical regions of the country, including the south (Rech *et al.*, 2005; Headley *et al.*, 2013a), midwest (Costa *et al.*, 2009) and northeast (Macêdo

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et al., 2007; Headley *et al.*, 2012). Transmission of OvHV-2 to susceptible animals is predominantly horizontal (Russell *et al.*, 2009), with nasal dissemination being demonstrated experimentally (Li *et al.*, 1998; Nishimori *et al.*, 2004). Vertical transmission has been suspected due to the detection of antibodies in virus-free and gnotobiotic lambs (Rossiter, 1981) and the identification of AlHV-1 from the fetus of wildebeest (Plowright, 1965). Although reports confirming the vertical transmission of OvHV-2 in cattle are sparse, viral DNA has been detected in an asymptomatic calf born to a cow with SA-MCF (O'Toole *et al.*, 1997). The present report describes the pathological and molecular findings associated with transplacental transmission of OvHV-2 in cattle.

Materials and Methods

Clinical History and Necropsy Examination

In late October 2013, a 3-year-old Girolanda cow (case 1) from the outskirts of the city of Cuiabá, Mato Grosso, central west Brazil, with clinical manifestations of profuse salivation, apparent blindness and anorexia was admitted to the Veterinary Teaching Hospital (VTH), Universidade de Cuiabá. Clinical evaluation revealed corneal opacity, lacrimation and severe, bilateral, mucopurulent nasal discharge (Fig. 1), fever, ruminal hypermotility, lethargy and ulcerative lesions in the oral cavity. In addition, the cow was carrying a 4-month-old fetus. The clinical condition of the cow deteriorated rapidly and the animal died spontaneously.

A 5-year-old cow (case 2) from the same farm was admitted to the VTH in early May 2013 with a history of bilateral ocular impairment that had reportedly started as keratoconjunctivitis 40 days earlier. This cow also died spontaneously. The interval between the onset of clinical manifestations and death of these cows was 7–40 days. An on-site visit to the farm revealed that there were 250 cows, a small flock of sheep reared for domestic consumption and intermingling between these species. The owner reported that all cows were maintained on pastures containing *Brachiaria brizantha* and were supplemented with corn silage and a soya bean-based ration, while water was supplied *ad libitum* at drinking troughs.

Both animals were submitted for routine necropsy examination soon after death. Tissue samples from the brain, lungs, kidneys, liver and myocardium of both cows and from the myocardium, lungs, kidneys and liver of the fetus were fixed in 10% neutral buffered formalin and processed routinely. Fresh tissue samples from the pregnant cow (brain and kidney) and fetus (brain) were kept at −80°C until used for molecular diagnostics.

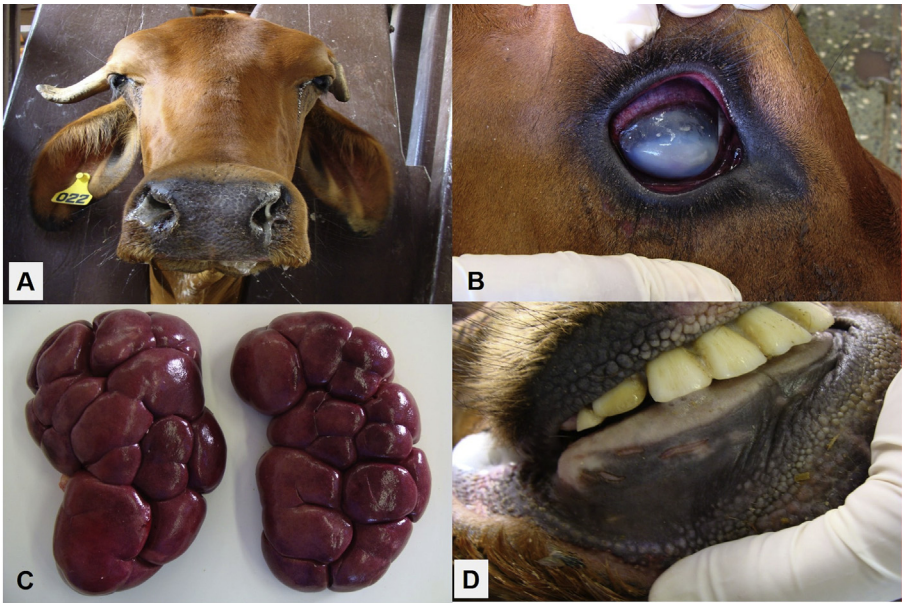

Fig. 1. Case 1 had mucopurulent nasal secretion (A) corneal oedema of the right ocular globe (B), fibrinopurulent bronchopneumonia, petechial haemorrhages within the mesenteric, thoracic and pleural surfaces, ulcerative stomatitis and rhinitis, pulmonary oedema, lymphadenomegaly and bilateral multifocal haemorrhagic nephritis (C). The fetus carried by this cow had normal external appearance and measured 30 cm in crown–rump length. Case 2 had bilateral corneal oedema, ulcerative stomatitis (D) and abomasitis.