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Short communication

Caprine herpesvirus type 1 infection in goat: Not just a problem for females

Michele Camero[a], Vittorio Larocca[a], Angela Lovero[a], Michele Losurdo[a], Francesco Cirone[a], Mariarosaria Marinaro[b], Canio Buonavoglia[a], Maria Tempesta[a,*]

[a] Department of Veterinary Medicine, University of Bari, Valenzano, Bari, Italy
[b] Department of Infectious, Parasitic and Immune-mediated Diseases, Istituto Superiore di Sanità, Rome, Italy

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ABSTRACT

Clinical, virological and serological analyses of a natural case of caprine herpesvirus 1 (CpHV-1) infection in a buck are reported. Three days after mating with a CpHV-1-infected female goat, the buck developed lesions referable to genital CpHV-1 infection. In particular, the animal suffered from typical painful ulcerative-crusted lesions associated with hyperemia, edema of the prepuce and healed completely within 15 days post-infection (p.i.). Infectious CpHV-1 was isolated from preputial swabs for 9 days p.i. while the virus was detected by real-time-PCR for 24 days p.i. Neutralizing antibodies were detected 7 days p.i. reaching maximal titers by day 14 p.i.

The prolonged shedding of the virus from the preputial route may impact the genital transmission of CpHV-1 in goat flocks during mating.

1. Introduction

Caprine herpesvirus infection 1 (CpHV-1) is a widespread infection of goats responsible for lethal systemic infection in kids and generally subclinical genital infections in adults.

Natural infection arises by the venereal route even though the respiratory and digestive routes cannot be ruled out. Indeed the virus is shed mainly and for a long period from the genital route.

Genital infections are characterized by clinical signs such as vesicular-ulcerative vulvo-vaginitis and abortion in females (Chenier et al., 2004; McCoy et al., 2007; Piper et al., 2008; Tempesta et al., 2000, 2004; Williams et al., 1997) and ulcerative balanoposthitis in males. Recovered animals become latently infected and the virus is detected in the sacral ganglia (Camero et al., 2010; Tempesta et al., 1999b). Spreading of the infection and seroconversion in a flock usually follow virus reactivation in infected animals during the mating season (Koptopoulos et al., 1988; Papanastasopoulou et al., 1990) and the use of natural mating is considered as a risk factor (Silva et al., 2013). Indeed, virus reactivation has been observed and reported only in coincidence with estrus in animals with low specific antibody titers (Tempesta et al., 1998).

Overall few papers have reported on the infection and disease in the male (Tarigan et al., 1987; Uzal et al., 2004). Lesions referable to previous CpHV-1 infection were observed in 1% of male goats regularly slaughtered in an abattoir in Australia (Tarigan et al., 1990).

* Corresponding author at: Department of Veterinary Medicine, University of Bari, Strada Provinciale per Casamassima km 3, 70010 Valenzano, Bari, Italy. Tel.: +39 0804679838; fax: +39 0804679843.
E-mail address: maria.tempesta@uniba.it (M. Tempesta).

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In this paper we describe a case of natural CpHV-1 infection and disease in a male goat.

2. Material and methods

2.1. Animals

The study was carried out in a goat flock in Southern Italy consisting of 20 adult females aged from 6 months to 4 years and a 3-year-old buck introduced into the flock 4 months earlier and separated from the females except during the mating season.

In September 2013, the farmer observed severe edematous and ulcerative vulvar lesions in a goat (goat no. 1). Lesions in the other goats and in the male were not observed during a specific clinical investigation. Importantly, the buck had mated with the goat no. 1 two days before the onset of lesions in the goat no. 1.

2.2. Sampling procedures

Blood samples with and without EDTA for virological and serological analysis, vaginal swabs for virological analysis from all of the goats apart from goat no. 1 and preputial swabs from the buck were collected. The buck and goat no. 1 were then housed separately in two different boxes.

Starting from the day of the visit to the goat farm (T0), all the animals were kept under observation; during this period buffy coats from peripheral blood, vaginal and preputial swabs for virological analysis were collected daily for 35 days (T35). Serum samples for serological analysis were collected weekly for 5 weeks.

2.3. Evaluation of clinical signs

Both goat no. 1 and buck were scored for clinical signs consisting of hyperemia, edema, lesions, pain and body temperature as reported in previous studies (Tempesta et al., 2007). A cumulative clinical score was determined in each animal by grading the clinical signs as follows: absent → 0; mild → 1; moderate → 2; severe → 3. Body temperature rises above normal were graded as follows: >0.5–1 °C → 1; >1.1–1.5 °C → 2; >1.5 °C → 3.

2.4. Virus isolation and real-time PCR assay

Swab samples and buffy coats were processed as described elsewhere (Tempesta et al., 1999a). Virus isolated from the swabs was then titrated in MDBK cells.

Samples were analyzed by a real-time PCR assay. Viral DNA was extracted from genital samples and buffy coat using a commercial kit (QIAamp Blood Kit, Qiagen GmbH, Germany) according to the manufacturer's instructions. The DNA extracts were tested by a CpHV-1 real-time PCR assay, following the method as previously described (Elia et al., 2008).

2.5. Neutralization assay

Seroconversion was evaluated as described elsewhere (Tempesta et al., 1999a). Briefly, serial 2-fold dilutions of each serum starting from 1:2 in D-MEM were mixed with 100 tissue culture infectious doses ($TCID_{50}$) of BA-1 strain of CpHV-1 in 96-well microtiter plates. The plates were held for 45 min at room temperature and then 20,000 MDBK cells were added to each well. Readings were made after 3 days of incubation at 37 °C in a CO_2 incubator. The titer of each serum was expressed as the highest dilution able to neutralize the cytopathic effect of the virus.

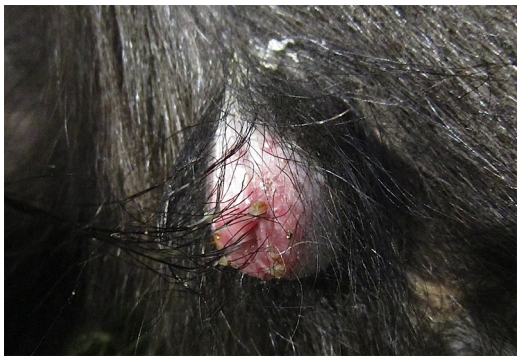

Fig. 1. Edematous, hyperemic ulcerative lesions on the prepuce of the buck.

3. Results

3.1. Clinical observation

In the buck, the infection was characterized by progressive onset of typical ulcerative-crusted lesions associated with hyperemia, edema and pain at the swabbing (palpation) (Fig. 1); the lesions appeared at T1, lasted for 10 days and were very severe at T5 and T6. A preputial discharge, initially serous with bloodstains and then purulent, appeared during the infection. The animal had a febrile reaction from T1 to T9 with the highest peak at T1 (40.8 °C). The clinical course of the infection was evaluated using the clinical score as reported in Fig. 2A.

In the goat no. 1, at T0, typical lesions referable to CpHV-1 infection, such as edema, hyperemia, vesicles, ulcers that were increasingly evident and evolving into crusts were observable on the vulva together with vaginal discharge. From T5, the lesions started to heal and by T8 the goat had clinically recovered (Fig. 2A).

3.2. Virological analysis

As reported in Table 1, using a CpHV-1-specific real-time PCR, at T0, the vaginal swab from goat no. 1 (clinically affected goat) was CpHV-1 positive (3×10^7 copies) whereas all the vaginal swabs from the other 19 goats and the preputial swab from the buck were CpHV-1 negative. Buffy coats samples were also CpHV-1 negative. During the observation period up to T17, the vaginal swabs from goat no. 1 were constantly CpHV-1 positive in real-time PCR. The buck resulted real-time PCR positive from T1 to T24, with the highest peak of CpHV-1 DNA copy number at T2 (9.99×10^9 copies). Vaginal swabs from the other 19 goats and buffy coats of all the goats, including the male, were