Brief communication

Experimental infection of reindeer with bovine viral diarrhea virus

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Abstract: Two 8-month reindeer (*Rangifer tarandus*) and a 1-month-old Hereford-Holstein calf (*Bos taurus*) were inoculated intranasally with the Singer (cytopathogenic) strain of bovine viral diarrhea (BVD) virus. Clinical signs in reindeer included loose stools containing blood and mucus, and transient laminitis or coronitis. Signs in the calf were limited to bloody mucus in the stool and lesions in the nasal mucosa. Antibody titers to BVD virus in the reindeer were intermittent, and titers in the calf persisted from days 14 to 63 post-inoculation (PI). Viremia was detected on PI day 4 in one reindeer, days 3–7 in the other, and days 2–7 in the calf. Bovine viral diarrhea virus was isolated from the lung of the calf at necropsy (PI day 63).

Key words: Reindeer, bovine viral diarrhea, BVD, respiratory, disease

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Serological surveys indicate reindeer (*Rangifer tarandus*) and caribou are naturally exposed to infectious bovine rhinotracheitis (IBR), parainfluenza type 3 (PI-3), and bovine viral diarrhea (BVD) viruses (Dieterich 1980; Elazhary et al. 1981). Clinical signs of BVD infection in domestic livestock include upper respiratory tract infections, reproductive disorders (Grahn et al. 1984), leukopenia, diarrhea, erosions of the oral mucosa, and immunosuppression (Kahrs 1981; Markham and Ramnaraine 1985; Radostits and Littlejohns 1988). Severity of disease ranges from mild to acutely fatal depending on the degree of viral-induced immunotolerance and cross-infecting strain of BVD virus (Baker 1987; Kahrs 1981). An investigation was conducted on a limited number of animals as a pilot study to investigate the effects of BVD virus on reindeer.

Two 8-months-old reindeer (*Rangifer tarandus*) and a 1-month-old Hereford-Holstein calf (*Bos tarandus*) were housed in a 10.5m^2 indoor disease containment room with shower-in-shower-out facilities. Each animal was inoculated

intranasally with 1 ml of media containing Singer (cytopathogenic) strain of bovine viral diarrhea (BVD) virus. The virus had been propagated in bovine embryonic testicular (BTES) cells (Evermann 1979; Coria and McClurkin 1978) grown in Minimal Essential Medium (MEM)[1] supplemented with 10% fetal bovine serum free of BVD virus and BVD virus antibody. Virus titer was calculated to be $10^{6.1}$ tissue culture infective doses - 50% per ml ($TCID_{50}$/ml).

Clinical signs were monitored. Heparanized blood samples were collected aseptically from the jugular vein daily for the first week for virus isolation (Evermann 1979). Jugular blood samples were collected twice weekly for hematologic studies and once weekly for serologic determinations (standard microtiter virus neutralization, Rossi and Kiesel, 1971). All animals were necropsied 63 days post-inoculation (PI). Samples of mesenteric lymph nodes, spleen, liver, kidney, lungs, and rumen were collected for histopathologic examination. Portions of buffy coat and liver, spleen, and lung were inoculated onto BTES cells for virus isolation (Evermann 1979; Coria and McClurkin 1978), and monitored for BVD viral antigens by direct immunofluorescence (Fernelius 1964) using bovine anti-BVD FITC conjugate received from the National Animal Disease Center, Ames, Iowa.

Clinical signs in both reindeer included loose stools containing blood and mucus, and transient laminitis or coronitis. A relative leukopenia was observed in each reindeer on PI day 17. A serous nasal discharge (PI day 8) progressed to a mucopurulent discharge (PI day 15), and became dry and crusty (PI day 29) in one reindeer. Signs in the calf included bloody mucus in the stool (PI day 7) and lesions in the nasal mucosa. Small pink macules 2 to 3 mm in diameter were observed in each nostril from PI days 16 to 19. A few macules and papules 1 to 2 mm in diameter were observed in the left nostril from PI day 23 to 28, and one 2 to 3 mm papule was noted in that same nostril from PI days 31 to 34. A leukopenia was observed on PI day 13.

Viremia was detected on PI day 4 in one reindeer, days 3–7 in the other, and days 2–7 in the calf. Antibody titers to BVD virus were detected on days 21, 28, 35, and 56 in one reindeer; and on days 28, 35, and 63 in the second. Antibody titers in the calf persisted from days 14 to 63 PI. Titers were lower in both reindeer than in the calf.

Gross lesions in the first reindeer at necropsy (PI day 63) were limited to excess hemorrhagic pericardial fluid. Microscopic lesions included peribronchular accumulations of polymorphonuclear and mononuclear cells. Rare focal accumulations of inflammatory cells were observed in the liver. No microscopic lesions were observed in the rumen.

No gross lesions were observed in the second reindeer. Microscopic lesions included periportal infiltration by mononuclear cells and areas of focal necrosis in the liver, and many areas of mononuclear infiltration primarily associated with the glomerular structures in the kidneys.

Bovine viral diarrhea virus was isolated from the lung of the calf at necropsy. Petechial hemorrhages were present on the rumen wall and tips of the papillae. Microscopic lesions appeared to be limited to small areas of necrosis and mononuclear infiltration in the rumen villi.

Clinical signs in all three animals were consistent with the spectrum of clinical disease associated with BVD virus infections (Evermann and Faris 1981; Blood et al. 1979). Although signs in animals in this study were considered mild, laboratory infections with BVD virus are often not as severe as field infections which may involve complicating factors such as secondary viral and bacterial infections, or superinfection of BVD-immunotolerant animals with a heterologous strain of BVD virus (Kahrs 1981; Radostits and Littlejohns 1988). Morbidity and mortality in cattle can be impressive when totally susceptible populations are infected (Kahrs 1981).

This study indicates that BVD virus is capable of replicating and causing clinical disease in reindeer. The potential exists for free-ranging reindeer in stressful situations to develop acute clinical signs of BVD infection. The possibility of manifestation of clinical signs of BVD or other respiratory infections should be considered in reindeer being translocated.

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