RABIES IN SMALL RUMINANTS AND CAMELIDS

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THE RABIES VIRUS

Rabies is caused by a rhabdovirus of the genus Lyssavirus. The virus is excreted in the saliva of infected raccoons, skunks, foxes, and bats, as well as domestic dogs and cats. Elsewhere in the world, the mongoose and vampire bat are important vectors. Fowler reports that rabies has developed in camelids from exposure to all of the major strains of virus in the United States, although bats probably present less of a risk to sheep and goats than do rabid carnivores. The virus is relatively fragile and easily inactivated by exposure to ultraviolet light or common disinfectants.

INCIDENCE OF INFECTION

More than 55,000 people die of rabies each year, mostly in Asia and Africa. The Journal of the American Veterinary Medical Association publishes an annual report of rabies surveillance in the United States. These reports since 2002 are available at a CDC website, <http://www.cdc.gov/rabies/resources/publications/index.html?s_cid=cs_521> For the last 11 years, the average number of annual confirmed cases of rabies in sheep and goats has been 11, while 7 infected camelids in total have been reported during this period. Obviously, the true incidence of disease is likely to be higher, as the diagnosis will be potentially missed in many animals for lack of testing.

INCUBATION PERIOD AND CLINICAL SIGNS

The incubation period between exposure and development of clinical signs will depend in part on the location of the bite wound on the body. The virus ascends nerves to reach the brain and inoculation in the muscles of the head will be more rapidly fatal than a bite to a distal hind limb. According to Fowler, 20 alpacas from a herd of 160 were bitten by a rabid dog in Peru. Thirteen died or were euthanized in extremis, with incubation periods varying from 15 to 34 days. Affected animals died 6 to 8 days after development of clinical signs. Similar incubation periods have been reported in sheep and goats after experimental infection or observed attack by a rabid animal.

Early signs of rabies in llamas and alpacas may include lameness, ataxia, or posterior paresis. Similar prodromal signs along with inappetence and/or fine muscle tremors are reported in sheep and goats. Both furious and paralytic forms have been observed in small ruminants and camelids with rabies. Any neurologic signs are possible, including dysphagia, salivation, teeth grinding, bleating, voice change, and aggression, either head butting or trying to bite. Pruritus at the scar of the bite wound is reported inconsistently. Wool pulling is sometimes noted. Somewhat unique to small ruminants is an occasional marked increase in sexual activity. For instance, a female goat with

rabies might try to mount and breed herd mates or a tire in its enclosure. Camels are reported to yawn in the terminal stage of rabies, when they are recumbent.

DIAGNOSIS

Rabies is a fatal disease, usually but not always within 10 days of the first clinical signs. If the animal with suspect neurological signs dies or is euthanized, it is ideal to submit the entire carcass to the diagnostic laboratory for a complete necropsy examination, so that a diagnosis can be made even if rabies is not present. If that is not possible, either the head or appropriate portions of the brain should be submitted to the state laboratory for testing. Required samples in New York are a complete cross section of the brainstem plus parts of all three lobes of the cerebellum (fresh, not fixed). The New York State laboratory in Albany (Wadsworth Laboratory) will not call a sample negative if both sides of the brain are not represented.

To remove adequate samples through the foramen magnum, first disarticulate the skull from the spine at the atlanto-occipital joint and place the head dorsal side down on a flat surface. In addition to wearing gloves, use a face shield to avoid splash of CSF into the eyes. Insert a knife with a long thin blade inside the dura and using an apple-coring motion, free the brain stem from the meninges. Pull on the brainstem with forceps and sever it inside the foramen magnum with the knife. A long-handled spoon or spatula-like instrument can then be used to separate the cerebellum from the cerebellar peduncles and coax it out through the foramen magnum. Complete instructions are available at <http://www.wadsworth.org/rabies/prof/livestk.htm>.

If the fluorescent antibody test (FAT) is positive, the laboratory will confirm the diagnosis by virus isolation in cell culture, rather than the older mouse inoculation test. The viral strain involved can be further identified using a monoclonal antibody panel. Occasionally histology is done on a brain that was not previously submitted for rabies testing. A nonsuppurative encephalitis with mononuclear cell perivascular cuffs will be present if the animal died of rabies. Negri bodies may be seen, which are eosinophilic inclusions in the cytoplasm of neurons in the hippocampus and in Purkinje cells. They are a classic finding in animals with rabies and often described as being pathognomic, but in fact specificity is less than 100%. According to the CDC the presence of Negri bodies has a sensitivity of about 50% when compared with FAT for the diagnosis of known rabid animals. A newer Rapid Rabies Enzyme Immuno-Diagnostic test (RREID) uses biotinylated antibodies, streptavidin conjugate and monospecific polyclonal antibodies against lyssaviruses. This test is specific but not quite as sensitive as the FAT. It is very useful for epidemiologic studies and in situations such as third world countries where equipment for fluorescent antibody testing is lacking.

TREATMENT AND PREVENTION OF HUMAN EXPOSURE

There is no effective treatment, so efforts should be directed at addressing other possible causes of the clinical signs such as polioencephalomalacia, listeriosis, meningeal worm, tick paralysis, and hepatic encephalopathy. Wear gloves when

examining or treating any small ruminant with neurologic signs. Keep out of the animal's mouth and warn the owner also of the potential for rabies.

Little data is available concerning the probability that a small ruminant or camelid clinically ill with rabies will excrete virus in saliva, however virus has been isolated from 4.6% of salivary glands and 1.6% of saliva samples from 87 cattle with naturally occurring paralytic rabies in Argentina. Transmission of rabies from alpaca to alpaca as a result of bites has been reported. Fowler reports that camelids with rabies are unable to regurgitate or spew stomach contents or saliva.

Rabies virus is widely distributed in tissues of rabid animals, and tissues and products from a rabid animal should not be used for human or animal consumption or transplantation. According to the National Association of State Public Health Veterinarians, pasteurization and cooking will inactivate rabies virus; therefore, inadvertently drinking pasteurized milk or eating thoroughly cooked animal products does not constitute a rabies exposure.

PREVENTION

There are several brands of killed rabies vaccine labeled for sheep over 3 months of age in the United States. Defensor-3® (Zoetis) and Prorab-1® (Intervet) require 2 ml IM, with annual boosters. Imrab® (Merial, 2 ml IM or SC) is labeled for sheep with boosters every 3 years, after the initial 1 year booster. No vaccines are approved for goats or camelids. Modified live vaccines should not be used in any of these species, and have been documented to cause post-vaccination paralysis in camelids.

In 1996, an unvaccinated goat was exhibited at a county fair in New York State. While at the fair, the goat developed clinical rabies due to a raccoon variant. Post exposure prophylaxis was administered to 438 people deemed to have been potentially exposed to this one animal. As a result of this event and the associated costs, many fairs in New York subsequently required that goats be separated from the public, as by a plexiglass barrier, because the animals could not be officially vaccinated with a labeled vaccine.

In 1999-2000, a goat vaccine trial was undertaken at Cornell University with substantial monetary support from Merial, New York State Department of Agriculture and Markets, and goat owners across the country. An Imrab® test vaccine was formulated to be of minimum potency and administered as a 2 ml subcutaneous dose in front of the shoulder to 30 goats approximately 1 year old. All goats developed high initial titers but the titers dropped off so that almost all were <1:16 a year later. Almost 14 months later the vaccinated goats and 10 unvaccinated controls were challenged by inoculation of virus into the masseter muscle. Peak mortality is usually at 2 weeks but the animals were observed for 3 months, as required by such trials. Although 28 of 30 vaccinates survived the challenge, only 6 of 10 controls died. Thus, even though a single dose of a minimally potent vaccine provided significant protection more than a year later, the response in control animals was insufficient (8 controls needed to develop rabies) to meet minimum requirements established by USDA to claim protection in this model.

Imrab® 2 ml subcutaneously with an annual booster is my choice for goats and camelids. A more conservative recommendation for these species when in contact with the public (petting zoos, fairs) is 2 initial doses 1 month apart followed by annual boosters. Camelids have been documented to get very high titers (up to 1:6000) after multiple doses. A booster should be given if the vaccinated animal is exposed to a known or suspect rabid animal. The exposed sheep is then to be observed for 45 days, but state officials are unlikely to recognize the goat or camelid as being vaccinated and euthanasia or a full 6 month quarantine may be required.

Official guidelines say that exposed unvaccinated livestock should be euthanized or slaughtered immediately. If the animal is not euthanized, its bite wounds should be cleaned and disinfected immediately and it should be observed and confined on a case-by-case basis for 6 months. Postexposure vaccination of exposed livestock has been recommended in Texas and appears to be generally effective. Vaccination should be repeated in the 3rd and 8th weeks of the isolation period. This approach is not considered in the National Association of State Public Health Veterinarians Compendium of Rabies Prevention and Control and may not be permitted by public health officials in all states.

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