Overview of Caprine Arthritis and Encephalitis

Caprine arthritis and encephalitis (CAE) virus infection is manifested clinically as polysynovitis-arthritis in adult goats and less commonly as progressive paresis (leukoencephalomyelitis) in kids. Subclinical or clinical interstitial pneumonia, indurative mastitis ("hard udder"), and chronic wasting have also been attributed to infection with this virus. Most CAE virus infections, however, are subclinical. Infection with the CAE virus decreases the lifetime productivity of dairy goats and is a barrier to exportation of goats from North America.

CAE virus infection is widespread among dairy goats in most industrialized countries but rare among indigenous goat breeds of developing countries unless they have been in contact with imported goats. In countries such as Canada, Norway, Switzerland, France, and the USA, seroprevalence of CAE virus is >65%.

Etiology, Epidemiology, and Pathogenesis

The CAE virus is an enveloped, single-stranded RNA lentivirus in the family Retroviridae. There are several, genetically distinct isolates of the virus that differ in virulence.

The CAE virus of goats is closely related to the ovine lentiviruses causing ovine progressive pneumonia and maedi-visna in North America and Europe, respectively. Cross-species transmission is possible through feeding of infected milk and colostrum. Therefore, the ovine and caprine lentiviruses are now commonly referred to as small ruminant lentiviruses.

CAE virus infection is widespread in dairy goat breeds but uncommon in meat- and fiber-producing goats. This has been attributed to genetics, management practices such as feeding colostrum and milk from a single dam to multiple kids, and industrialized farming practices (eg, frequent introductions of new animals into a herd). Prevalence of infection increases with age but is not influenced by sex. Most goats are infected at an early age, remain virus positive for life, and develop disease months to years later.

The chief mode of spread of CAE is through ingestion of virus-infected goat colostrum or milk by kids. The feeding of pooled colostrum or milk to kids is a particularly risky practice, because a few infected does will spread the virus to a large number of kids. Horizontal transmission also contributes to disease spread within herds and may occur through direct contact, exposure to fomites at feed bunks and waterers, ingestion of contaminated milk in milking parlors, or serial use of needles or equipment contaminated with blood. Unlikely methods of transmission, as indicated by experimental studies, include in utero transmission to the fetus, infection of the kid during parturition, and infection through breeding or embryo transfer.

The pathogenesis of CAE is not fully understood. Virus-infected macrophages in colostrum and milk are absorbed intact through the gut mucosa. Infection is subsequently spread throughout the body via infected mononuclear cells. Periodic viral replication and macrophage maturation induces the characteristic lymphoproliferative lesions in target tissues such as the lungs, synovium, choroid plexus, and udder. Persistence of the CAE virus in the host is facilitated by its ability to become sequestered as provirus in host cells. Infection induces a strong humoral and cell-mediated immune response, but neither is protective.

Clinical Findings

Clinical signs are seen in ~20% of CAE virus–infected goats during their lifetime. The most common manifestation of infection is polysynovitis-arthritis, which is primarily seen in adult goats but can occur in kids as young as 6 mo old. Signs of polysynovitis-arthritis include joint capsule distention and varying degrees of lameness. The carpal joints are
most frequently involved. The onset of arthritis may be sudden or insidious, but the clinical course is always progressive. Affected goats lose condition and usually have poor hair coats. Encephalomyelitis is generally seen in kids 2–4 mo old but has been described in older kids and adult goats. Affected kids initially exhibit weakness, ataxia, and hindlimb placing deficits. Hypertonia and hyperreflexia are also common. Over time, signs progress to paraparesis or tetraparesis and paralysis. Depression, head tilt, circling, opisthotonos, torticollis, and paddling have also been described. The interstitial pneumonia component of CAE virus infection rarely produces clinical signs in kids. However, in adult goats with serologic evidence of CAE virus infection, chronic interstitial pneumonia that leads to progressive dyspnea has been documented. The “hard udder” syndrome attributed to CAE virus infection is characterized by a firm, swollen mammary gland and agalactia at the time of parturition. Milk quality is usually unaffected. Although the mammary gland may soften and produce close to normal amounts of milk, production remains low in many goats with indurative mastitis.

**Lesions**

Pathologic lesions of CAE virus infection are generally described as lymphoproliferative with degenerative mononuclear cell infiltration. Lesions in joints are characterized by thickening of the joint capsule and marked proliferation of synovial villi. In chronic cases, soft-tissue calcification involving joint capsules, tendon sheaths, and bursae is not uncommon. Severe cartilage destruction, rupture of ligaments and tendons, and periarticular osteophyte formation have also been described in advanced cases. Microscopic features of articular lesions include synovial cell hyperplasia, subsynovial mononuclear cell infiltration, villous hypertrophy, synovial edema, and synovial necrosis. Gross lesions associated with the neurologic form of CAE include asymmetric, brownish pink, swollen areas, most commonly in the cervical and lumbosacral spinal cord segments. Histopathologically, these lesions are characterized by multifocal, mononuclear cell inflammatory infiltrates and varying degrees of demyelination. On gross examination, lungs of affected goats are firm and gray-pink with multiple, small, white foci, and do not collapse. The bronchial lymph nodes are invariably enlarged. Histologic findings include chronic interstitial pneumonia with mononuclear cell infiltration in alveolar septae and in perivascular and peribronchial regions. In does with udder induration, mononuclear infiltration of periductular stroma obliterates normal mammary tissue.

**Diagnosis**

A presumptive diagnosis can be based on clinical signs and history. Infectious arthritis caused by *Mycoplasma* spp and traumatic arthritis are differential diagnoses for arthritis induced by CAE virus. Differential diagnoses for the progressive paresis and paralysis exhibited by young kids should include enzootic ataxia, spinal cord abscess, cerebrospinal nematodiasis, spinal cord trauma, and congenital anomalies of the spinal cord and vertebral column. If the neurologic examination indicates brain involvement, polioencephalomalacia, listeriosis, and rabies should be considered as possible causes. The pulmonary form of caseous lymphadenitis may have a similar clinical presentation to the pulmonary form of CAE in adult goats.

Both an agar gel immunodiffusion test and ELISA for CAE virus are considered sufficiently reliable for use in control programs. The agar gel immunodiffusion test is reported to be more specific but less sensitive than the ELISA. A positive test result in an adult goat implies infection but does not confirm that the clinical signs are caused by CAE virus. Kids infected at birth develop a measurable antibody response 4–10 wk after infection. However, positive test results in kids <90 days old usually reflect colostral antibody transfer. Negative test results do not reliably exclude CAE virus infection, because the time for postinfection seroconversion is variable and occasional goats have a very low titer that may not be detectable. Low antibody titers are common in late pregnancy. Because of the limitations of serologic testing, definitive diagnosis of clinical CAE requires demonstration of characteristic lesions in biopsy specimens or at necropsy. Virus isolation or PCR to demonstrate presence of viral antigen in tissues may be used to further substantiate the diagnosis.

**Treatment and Control**

There are no specific treatments for any of the clinical syndromes associated with CAE virus infection. However, supportive treatments may benefit individual goats. The condition of goats with the polysynovitis-arthritis may be improved with regular foot trimming, use of additional bedding, and administration of NSAIDs such as phenylbutazone.
or aspirin. Goats with encephalomyelitis can be maintained for weeks with good nursing care. Antimicrobial therapy is indicated to treat secondary bacterial infections that may complicate the interstitial pneumonia or indurative mastitis components of CAE virus infection. Providing high-quality, readily digestible feed to goats positive for CAE virus may delay the onset of the wasting syndrome.

In commercial herds, one or more of the following have been recommended for control of CAE: 1) permanent isolation of kids beginning at birth; 2) feeding of heat-treated colostrum (45°C [113°F] for 60 min) and pasteurized milk; 3) frequent serologic testing of the herd (semiannually), with identification and segregation of seronegative and seropositive goats; and 4) eventual culling of seropositive goats. If the control program includes segregation of herds into seropositive and seronegative groups, groups should be separated by a minimum of 6 ft (1.8 m), and shared equipment should be disinfected using phenolic or quaternary ammonium compounds.