

DOI: 10.7251/VJRS1302101D

UDK 619:616.988:636.38/.39(495)

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SLOW VIRAL INFECTIONS OF SMALL RUMINANTS IN GREECE

Abstract

The slow viral infections are from the most important problems of sheep and goat production in Greece. Diseases as enzootic nasal tumor, Maedi-Visna, caprine arthritis-encephalitis, pulmonary adenomatosis and Scrapie can cause significant losses in sheep and goat flocks. They can be transmitted during grazing or with animal sales. Here are described the clinicopathological findings of the aforementioned diseases, as well as their diagnosis and control.

Key words: slow viral infections, small ruminants.

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ВИРУСНЕ ИНФЕКЦИЈЕ СПОРОГ ТОКА МАЛИХ ПРЕЖИВАРА У ГРЧКОЈ

Кратак садржај

Вирусне инфекције спорог тока су врло значајан проблем код оваца и коза у Грчкој. Обољења као што су *Maedi-Visna*, артритис и енцефалитис код коза (CAEV), аденоматоза и скрепи могу да узрокују значајне губитке у запатима оваца и коза. Могу да се пренесу у току испаше или продајом животиња. У овом раду је описан клиничко-патолошки налаз наведених болести, као и њихова дијагностика и контрола.

Кључне речи: вирусне инфекције спорог тока, мали преживари.

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INTRODUCTION

Chronic viral diseases of small ruminants are from the most important problems in Greek small ruminant industry, because they are not treatable, as well as no effective vaccines are available against them. The repercussions resulting from this are the loss of production and of whole flocks and consequently the loss of farmers' income. Of crucial importance is also that other related professions are affected (veterinarians, feedstuffs, drugs, farm supplementation), while this impact is reflected to the National Economy (Giadinis, 2012).

These diseases are sheep progressive pneumonia, goat arthritis-encephalitis, pulmonary adenomatosis, enzootic nasal tumor and Scrapie.

SHEEP PROGRESSIVE PNEUMONIA (MAEDI-VISNA)

Sheep progressive pneumonia (Maedi-Visna) is caused by a non-oncogenic, exogenous retrovirus, which belongs to the lentivirus family (Pritchard and McConnell, 2007). It is transmitted with suckling (colostrum, milk) and horizontally animal to animal (Eltahir et al., 2006), while some researchers believe that vertical transmission via placenta also exists (Cortez-Romero et al., 2011). The virus causes a chronic disease with clinical demonstration mainly from the respiratory and the central nervous system. During the last years the disease

has been diagnosed many times in sheep with non-suppurative arthritis and with mastitis. Usually animals older than 2 years old are infected, although it has been observed in younger ones (Belknap 2002; Pritchard and McConnell, 2007). It is useful to mention, that sheep progressive pneumonia is very commonly diagnosed in Greece, although a systematic epidemiological study has not been conducted to date.

Maedi-Visna is usually observed in animals older than 2 years old, although clinical symptoms of this disease in younger animals have been observed. The respiratory form is characterized by chronic weight loss, dyspnea and tachypnea worsening with time and exercise. At the final stages the animals can have orthopnoic stance and they die from dyspnea or secondary bacterial infections. Lung auscultation reveals reduction or absence of the alveolar sound. As far as the nervous form, the infected animals develop monoparesis, ataxia and proprioceptive deficits, while at the later stages they have paraparesis or tetraparesis, they become comatose and die. The arthritic form is characterized by non-purulent chronic arthritis, especially in the carpal joints and more rarely in the tarsal ones. The mastitic form (hard udder, hardbag) is evident with the reduction of milk production or of milk flow. In the case of mastitic and arthritic form the animals can have normal body condition. Maedi-Visna infection can predispose to pregnancy toxemia (Bel-

knap, 2002; Pritchard and McConnell, 2007; Giadinis, 2012a).

The clinical forms of Maedi-Visna can be diagnosed by necropsy, although many times histopathological confirmation or immunohistochemistry are necessary. Serology or PCR (blood, tissues) contribute to the detection of viral infection, but are not enough to confirm the clinical diagnosis (Pritchard and McConnell, 2007).

Maedi-Visna is a non-treatable condition. The disease can be prevented in non-infected flocks mainly by buying animals that are free of the virus and by avoiding the co-existence of healthy and infected animals, e.g. grazing. Also, it is useful in regular time intervals to examine serologically a number of animals. In infected flocks eradication is difficult. A number of control protocols have been suggested, but they have many disadvantages:

I) Slaughtering of the whole flock is difficult, as it has severe economic impact. However, sometimes (mastitis, high mortality) it seems to be the only solution (Pritchard and McConnell, 2007; Giadinis, 2012a).

II) Removal of neonatal lambs from their mothers after birth and use of artificial suckling or suckling of seronegative mothers. This protocol has been used in experimental flocks, but it is difficult to be approved in commercial ones. The factors which could lead to the failure of this protocol are the contact with their

mother and the suspected vertical transmission of the disease (Pritchard and McConnell, 2007).

III) All the adult animals of the flock are examined every 3–6 months and seropositives are slaughtered, as well as their progeny. Eradication has been completed, when 2 consecutive serological examinations are negative. For the already aforementioned reasons this protocol is ineffective, while it has high cost and is time-consuming (Pritchard and McConnell, 2007).

IV) An economical and relatively easy protocol is the one performed by our Clinic:

If the disease has been diagnosed in a flock, every animal with signs of the disease is slaughtered, as well as their progeny. The rest flock is regularly dewormed and the animals are fed with vitamins and trace elements for the whole year. Furthermore, good hygiene measures and husbandry are necessary. In this way, many flocks in Greece have become economically profitable (Giadinis, 2012).

CAPRINE ARTHRITIS-ENCEPHALITIS (CAEV)

Caprine arthritis-encephalitis (CAEV) is caused by a retrovirus, antigenically similar to the Maedi-Visna agent and is transmitted in a similar way. The disease has 2 clinical forms, the arthritic and the nervous. The arthritic form causes ancyrotic arthritis in animals older than 2

years old, while the nervous one can cause myelitis (less often encephalitis) in goat kids aged 2–6 months old. During the last years other forms of the disease have been diagnosed, as chronic indurative mastitis and interstitial pneumonia. Diagnosis, treatment and prevention are similar to sheep Maedi-Visna (Smith and Sherman, 2009).

PULMONARY ADENOMATOSIS

Pulmonary adenomatosis is caused by an exogenous retrovirus that is different from the Maedi-Visna virus and the disease is transmissible. It is a chronic cachexiogenic disease that concerns mainly sheep and rarely goats, whereas it does not affect cattle (Sharp and De lasHeras, 2007). In Greece it is not as widespread as Maedi-Visna is, although sometimes they do co-exist in the same animal (Giadinis, 2012). The annual losses are 2–10%, although in Iceland, where the disease was observed for the first time, the annual losses reached the level of 80 % (Sharp and De lasHeras, 2007). In Greece the disease is observed sporadically.

Pulmonary adenomatosis is transmitted with the respiratory secretions of the infected animals and usually animals infected early in their life (younger than 10 months) are getting sick. Also, recent studies have shown that the virus is excreted through colostrum and milk (Sharp and De lasHeras, 2007). Usually it infects animals at the age of 2–4 years

old, but the disease has been diagnosed also in lambs aged 1–2 months old. These are usually the progeny of infected dams (Sharp and De lasHeras, 2007; Scott, 2007). Sick animals lose weight, although their appetite is normal. Moreover, they have a progressively deteriorated dyspnea and tachypnea which are increased after exercise. The most important sign of the disease is the copious nasal discharge. If the infected animal is kept from its hind-legs (“wheelbarrow test”), the discharge is excreted. Its volume can be 30–50 ml, but it can sometimes reach 300 ml (equal to the quantity of a full glass). This finding is pathognomonic but it is not always a clinical symptom. It is common in Greece, while pulmonary adenomatosis usually co-exists with Maedi-Visna. Increased crackles are heard over a wide area of the chest during auscultation in the typical form of the disease. Infected animals die in 1–4 months due to bacterial infections (Sharp and De lasHeras, 2007; Scott, 2007; Sargison, 2008; Giadinis 2012).

Diagnosis is based, upon history, on clinical findings and necropsy, but it can be confirmed by histopathological examination or immunohistochemistry (Sharp and De lasHeras, 2007). Also, ultrasound can be helpful for clinical diagnosis (Scott, 2007). During the last years, a PCR technique has been developed. The last detects the virus in the blood and tissues not only of the host but also of the carrier animals (Sharp and De lasHeras, 2007).

The disease is non-treatable. The affected animals should be slaughtered immediately, while good nutrition and regular deworming increase the resistance to the disease. PCR and embryo transfer could help in the eradication of the disease in the future. However, in Iceland where the disease was endemic, it was eradicated after slaughtering the whole infected flocks. In the case that Maedi-Visna co-exists, then the best solution is slaughtering the whole flock (Sharp and De lasHeras, 2007; Sargison, 2008; Giadinis, 2012a).

ENZOOTIC NASAL ADENOCARCINOMA (TUMOR)

Enzootic nasal adenocarcinoma (tumor) is a contagious disease of the nasal cavities that focuses on mucosal glands and can be uni- or bilateral. The disease is sporadic (0,5–2 %), although morbidity in some flocks is up to 15 %. Nowadays, the disease is very common in Greece due to the uncontrolled animal sales. The infected animals have low production and the disease is always fatal (Giadinis, 2012a).

The disease is caused by a retrovirus which is homologous but different to pulmonary adenomatosis virus. Also, the virus is different between sheep and goats and consequently there is not inter-species contamination. Reproduction of the disease is difficult. Usually, animals 2-4 years old are infected, although the disease has been reported in lambs 4 months old and goat kids 7 months old.

The animals have seromucous or mucopurulent nasal discharge, that is uni- or bilateral, stridor, that progressively becomes evident from a distance, exercise intolerance and inspiratory dyspnea which deteriorate gradually. At the final stages the animals have their mouth open when breathing. During the nasal auscultation a stridor is heard, which is intense at larynx but it is reduced and disappears lower. Furthermore, although it is rare, facial deformation and exophthalmus can be observed. The animals become cachectic and finally die from bacterial infections or asphyxia (Belknap, 2002; Sharp and De lasHeras, 2007; Giadinis, 2012).

Diagnosis is based upon clinical and necropsy findings, but is confirmed by histopathology or cytology. The disease should be differentiated from oestrosis, pararhinocolpitis, actinobacillosis and actinomycosis (Loukopoulos et al., 2010; Ioannou et al., 2011; Giadinis, 2012a).

Animals of high reproductive value could be treated with surgical excision that is usually ineffective. The best choice is to slaughter the infected animals before they lose weight. Prevention is also difficult. It is suggested to buy animals only from healthy flocks (Belknap, 2002; Giadinis., 2012).

SCRAPIE

Scrapie is a progressive degenerative encephalopathy of sheep and goats. The disease was known since 19th century in UK, while in Greece it was diagnosed for

the first time in the 80s (Argiroudís et al., 1987) and today it is widespread throughout the country. As it belongs to spongiform encephalopathies, it has zoonotic interest and it is intensively investigated. Scrapie, although is not zoonotic, it is a notifiable disease (Scott, 2007; Sargison, 2008).

Scrapie is caused by a modified glucoprotein (prion) that is self-reproduced in animal tissues, although it has not DNA or RNA. The disease has been reproduced. Transmission is horizontal and vertical. It can be transmitted with animal sales and during grazing. The last years it has been pointed out that prion is abundant in fetal fluids, is secreted through milk and it can be transmitted with endo- and ectoparasites. A genetic susceptibility to the prion is accepted (Jeffrey and González, 2007, Giadinis, 2012).

Scrapie is found in animals older than 2 years old, but there are also strains which infect younger animals (5–12 months old). Initially, the animals change their behavior and sometimes they deny to be milked. Then, they lose weight and have progressively deteriorated ataxia and other neurological signs, as muscular tremor, salivation, tympany, blindness and paresis. A characteristic symptom is pruritus at the back and hind legs. Sometimes the animals lick these areas and scrape them at fences. A slight pressure at the lumbar area causes reflexive motions of their lips (nibbling reflex). Pruritus in goats is absent or minimized.

The animals die after 1–6 months (Sofianidis et al., 2006, Jeffrey and González 2007, Giadinis, 2012).

Scrapie should be differentiated from other brain encephalopathies, like chronic coenurosis, Visna, CAEV. As far as the pruritus, it should be differentiated between chorioptic mange and pseudorabies (Giadinis, 2012). In goats, pruritus is minimized or missing. Diagnosis of Scrapie can be confirmed only in dead animals. The 3 most reliable diagnostic methods are brain histopathology, immunohistochemistry and Western blot (Jeffrey and González, 2007).

During the last years atypical forms of Scrapie have been diagnosed (Norvegic type or Nor-98). They are observed in animals older than 3 years old and they usually have similar clinical signs to the other two types, although pruritus can be absent or the animals can be asymptomatic (Benetsad et al., 2003; Jeffrey and González, 2007).

Prognosis for Scrapie is poor, as the disease is untreatable and the flocks often have high mortality. As far as prevention, it is suggested to buy animals which come from flocks that are not infected by Scrapie and also have animals resistant to the disease (ARR/ARR is the most resistant genotype for sheep). During the last years also resistant genotypes have been found in goats. In flocks where Scrapie has been diagnosed the existing legislation is followed (notifiable disease). In case of Scrapie outbreaks, it is

necessary all the infected animals to be killed. Moreover, other measures that increase the animal survival and decrease the infection rate (disinfections, dewormings, good nutrition, special places for parturition) would be useful. Last but not least, resistant genotypes are necessary for the flock reproduction (Jeffrey and González, 2007; Fragkiadaki et al., 2011; Giadinis, 2012).

CONCLUSION

In Greece slow viral infections seem to be common. These conditions are not treatable, as well as no vaccines are available and so preventive biosecurity measures seem to be necessary for avoidance of infections.

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