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Review

Peste des Petits Ruminants

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Abstract

Pest des petits ruminants (PPR) is a disease first described in 1941 in West Africa. It is caused by a virus belonging to the family *Paramyxoviridae*. The virus is highly virulent for goats and sheep. There is no virus carriage. The incubation period is 5-6 days. Mortality is high. Transmission of infection is through close contact between sick and healthy animals – infection occurs mainly through the respiratory tract. In pathogenesis, the lymphatic system plays a major role. The infection predominantly affects small ruminant flocks. Clinical symptoms are mainly lesions on the muzzle, nose and urogenital tract. Diagnosis is based on characteristic clinical and pathological anatomical evidence. Stringent measures are necessary to isolate and quarantine risky flocks and farms.

In connection with a PPR epizootic in Syria, the import of lambs from our country was terminated. The team of Bulgarian specialists sent to Syria proved that Bulgaria had nothing to do with the epizootic in that country. In 2018, the dangerous disease of sheep and goats was detected in Bulgaria. Data reported in the press and other media seriously differed from the well-known characteristics of the Pest des petits ruminants infection.

Keywords: Pest des petits ruminants (PPR), *Paramyxoviridae*, epizootology.

Резюме

Чумата по дребните преживни (Pest des petits ruminants) е заболяване установено за първи път през 1941 г. в Западна Африка. Принителят е вирус от семейство *Paramyxoviridae*. Вирусът е силно вирулентен за кози и овце. Няма вирусоносителство. Инкубационният период е 5-6 дни. Смъртността е висока. Предаването на инфекцията е чрез тесни контакти между болни и здрави животни – зразяването е главни по респираторен път. В патогенезата главна роля изпъ;нява лимфната система. Епизоотологията е предимно стадна. Клиничната симптоматика са главно лезии по муцуната, ноздрите и урогенуталния тракт. Диагнозата се поставя въз основа на характерните клинични и патологоанатомични данни. Предприемат се строги мерки за изолация и карантина на рисковите стада и ферми.

Във връзка с епизоотия от това заболяване в Сирия е прекратен вноса на агнета от нашата страна. Изпратенуият български екип от специалисти доказва, че нашата страна няма нищо общо с тази епизоотия. У мас през 2018 г. е обяено наличието на това опасно заболяване по овце и кози. От обявените в пресата и други медии данни се установяват седриозни различия от общоизвестните характеристики на чумата по дребните преживни.

Introduction

Notwithstanding existing difficulties in the scientific area, Bulgarian veterinary science has always sought to rise to the challenges posed by its profession and by the state regarding livestock wellbeing not only

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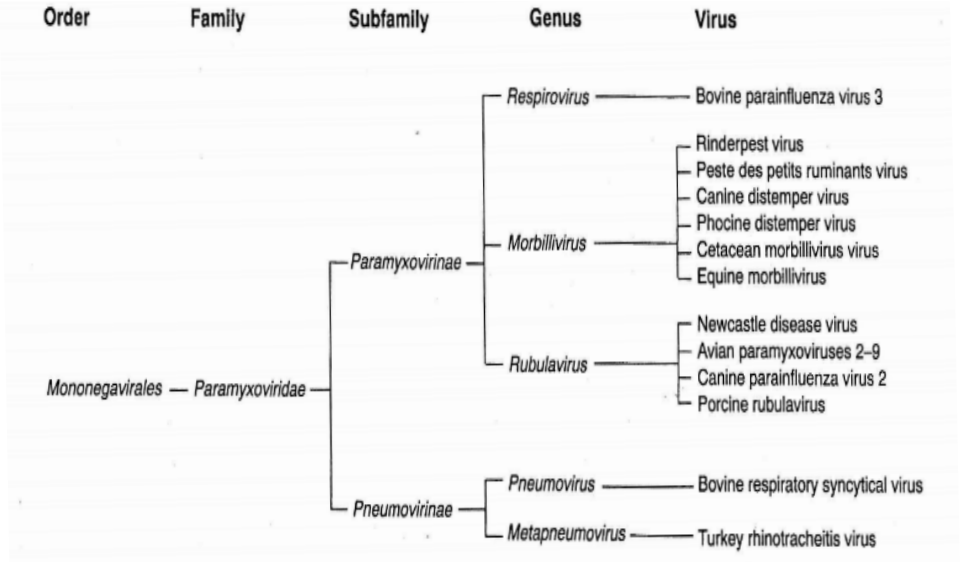

Fig. 1. Classification of parvomyxoviruses with emphasis on those of veterinary importance

in terms of trivial contagious diseases peculiar to a region but also against exotic diseases (Masalski, 1991; 2008).

It is only natural that along with the changes due to globalization exotic infectious diseases, once endemic, are now becoming a real global threat. Precisely for that reason, the knowledge about such diseases should be brought up to date so that adequate preventive and combat actions can be implemented. Undoubtedly, the requirements regarding the relatively novel disease worldwide, Peste des petits ruminants (PPR), are imperative. The disease was first reported in 1941 in Côte d'Ivoire, West Africa and is also known as Pseudorinderpest, Stomatitis-pneumoenteritis complex, Kata in Nigeria (Gargadannec and Lananne, 1942). It is endemic across the sub-Saharan countries, the Arabian Peninsula and some Middle East countries (Braide, 1981; Maztin, 1983; Taylor, 1984).

The cause of the disease is a virus belonging to the family *Paramyxoviridae* (Fig. 1) (Gibbs *et al*., 1979). Although it shares characteristics with the bovine plague virus, the two viruses are not completely identical and are differentiated by cross-seroneutralisation (Geering, 1986). The epizootiology of the disease is characterized by certain peculiarities depending on the virus specificity, the host animal species - sheep and goats only, and on the nature of the disease process. The virus is highly virulent for sheep and especially high for goats and the young animals of those species. After recovery, there is no viral persistence in the body, therefore there is no carriage of the virus or intermediate carriers (vectors), and the role of the wild animals is insignificant as contact with domestic animals is only short and infrequent. The virus found in the excretions and secretions from infected animals does not survive longer than 1-2 days in the external environment. The incubation period is short but the infected animals start shedding the virus several days after its onset in the absence of clinical signs. The clinical signs themselves are clearly expressed and the disease terminates within a short period of time with high mortality. All this determines the way the infection is transmitted and spread. Transmission occurs in extremely close contact between sick and healthy animals, and indirect transmission via feed, water or vehicles is almost impossible. A major source of contagion is infected animals introduced into a healthy flock. Epizootiological peculiarities make it predominantly a flock disease that does not tend to spread out of the affected flock or holding, although it spreads rapidly to almost all animals within the flock.

The cause of the mass disease in the flock is the way the virus is released, which initially occurs through nasal and conjunctival discharge and aerosols from the respiratory tract, and later, after the appearance of clinical signs, through faeces and urine. Although virus shedding and infection transmission is possible in all these routes, the primary mode of infection is through the respiratory system. Susceptible animals contract the disease by inhaling virus-containing droplets excreted by the sick animals in amounts sufficient to cause infection. This is the reason why it spreads at a great speed within the affected flock. In addition, contact between the sick and healthy animals is also facilitated by the high mobility of the sheep within the flock. In the countries where the disease is endemic, the spread within or across borders occurs through uncontrolled animal trade, and in particular through the formation of assembly flocks in preparation for their sale or by assembling animals in centres after their purchase from different sources and subsequent distribution to new owners. Such was the epizootic of PPR in Syria in 1987.

Pathogenesis. After inhalation of the virus, multiplication occurs in the pharyngeal and mandibular lymph nodes. Viraemia develops within 3 days and the virus spreads to other lymphoid organs, including the mucosa of the respiratory and digestive tracts. Necrotic processes occur in the lymphoid tissue and, as a result, animals develop leukopenia and immunosuppression. During the acute stage, the virus is shed in large quantities, but after the temperature returns to normal viral shedding also declines.

Clinical signs. The incubation period lasts from 5 to 6 days followed by sudden fever with a rise in temperature to 40-42°C and clinical signs are manifested 1 to 2 days thereafter. Fatigue, loss of appetite, serous nasal discharge, which becomes mucopurulent, are observed. The mucous membranes are red, breathing rate increases, constipation occurs. Three to four days after the onset of fever, lesions affecting the muzzle, nose, and urogenital tract begin to develop. Those in the oral cavity are accompanied by abundant salivation. Conjunctivitis and ocular discharge are one of the most common traits at this stage in the development of the disease. In the beginning, lesions are small necrotic areas. Within a short time, however, their number increases, they increase in size and merge. After removal of the necrotic tissue, uneven, well-defined erosions are found underneath. At this stage, the animals appear to be very sick. Their breath is putrid, the breathing is difficult with marked signs of tracheitis and pneumonia. The onset of diffuse diarrhea approximately coincides with the second day of the onset of mucosal changes. The faeces are dark, foul-smelling of rotting flesh, sometimes containing mucus, fragments of necrotic mucosa and blood. Severe leukopenia facilitates secondary pasteurella, streptococcus, salmonella and E. coli bacterial infection. Mortality is high, reaching 70-90% in young animals and goats and death occurs within 10 days of infection. The recovery of the few surviving animals is slow and takes a long time.

Pathological-anatomical changes. The carcasses of the dead animals are dehydrated, exhausted, soiled by loose stools and malodorous. Erosions of the conjunctiva, muzzle and nose can be observed (Fig. 2). In the oral cavity, erosions, ulcers and necrotic foci on the inside of the cheeks, tongue, gums, pharynx and the esophagus are found. The necrotic matter is gray, bran-coloured and easily removed, leaving an ulcerated reddish surface underneath (Fig. 3). Stomach mucosa is hyperemic, hemorrhagic and necrotic. Often, the

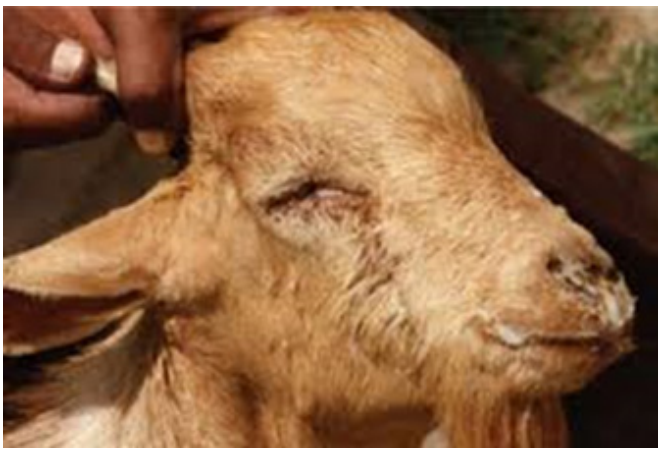

Fig. 2. Mucopurulent discharges from the eyes and nasal cavity

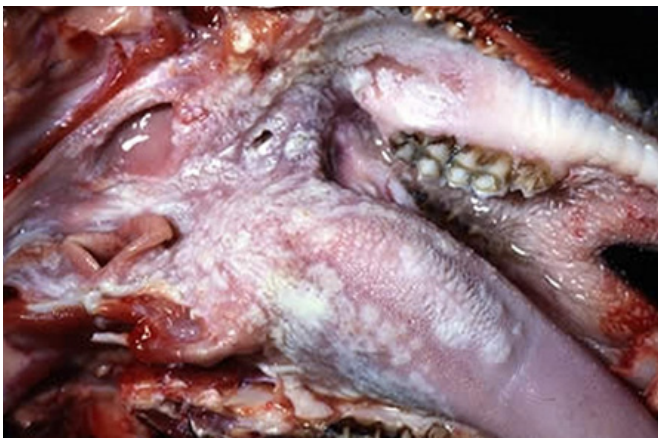

Fig. 3. Erosions and ulcerations on the tongue and soft palate

necrotic tissue has fallen off, and there are ulcerated areas underneath. Significant changes in the large intestine are manifested in severe hyperaemia, bleeding and necroses lending the folds a look of tiger stripes, likened by some to zebra stripes. Changes in the cecum at the sites of passage into the colon and into the rectum are also considerable. The lymph nodes are enlarged and hyperemic, the spleen is enlarged and with focal necrosis

Nasal mucosa is covered with a thick mucopurulent mass, and long strips of hyperemic mucosa can be seen in the trachea. The apical segments of the lungs reveal bronchopneumonic changes. The heart is covered with hemorrhages. Hemorrhages, necrosis and ulcers are also found in the mucous membranes of the urinary tract.

Diagnosis. Putative diagnosis is based on the characteristic clinical and pathological-anatomical features, and on the analysis of the epizootiological data. Specimens for life-time laboratory diagnosis confirmation are conjunctival and nasal discharge, rectal and buccal swabs. Anticoagulants are added to the blood samples. They are taken when the clinical

signs are most prominent. Suitable specimens for examination are those from the mesenteric and bronchial lymph nodes, spleen, colon and lung from dead animals. All samples must be fresh and stored at refrigeration temperature. The virus is isolated in tissue cultures of lamb kidney cells or VERO cells. The cytopathic effect occurs not earlier than the 5th day after inoculation. Virus confirmation is also possible by detecting it in specimens when encountered with standard positive sera through different reactions. Serological diagnosis reactions are based on the detection of antibodies in sick animals and in those surviving the viral infection. For that purpose, blood samples are taken during the illness and 2-3 weeks afterwards to determine the antibody dynamics. The presence of antibodies is confirmed in blood serum four years after the viral infection (Durojaiye and Taylor, 1984). Reversed arrangement of components is performed. The sera to be tested are associated with a known standard antigen derived from the PPR virus. Laboratory tests used for viral antigen detection are direct immuno-staining, agar-gel immunodiffusion, and ELISA among others. Virus-neutralization, indirect fluorescence assay, competitive ELISA and other tests are used for the detection of antibodies. RT-PCR is used to detect the presence of viral genome (OIE, 1992).

Prevention and control. PPR control measures depend on whether the disease is endemic or has newly emerged in a particular country. In countries where it is detected for the first time, radical measures are taken to eliminate all affected animals in the flock or holding, destroy their carcasses and afterwards carry out appropriate disinfection. Stringent measures are established for spatial isolation and quarantine of risky flocks and farms, which are placed under constant surveillance. In those countries where the disease is endemic, measures are implemented to limit uncontrolled movement of sheep and goats as well as mass vaccinations. The close antigenic kinship between the PPR virus and the bovine plague virus allows the use of a vaccine prepared from bovine plague vaccine strains inducing long-term immunity (Geering, 1986). Recent studies have yielded vaccines consisting of attenuated PPR virus strains inducing immunity for at least 18 months (OIE, 1992). It should be borne in mind that despite the success of immune prophylaxis, many of the affected countries are unable to eradicate the disease, which proves the disease to be difficult to fight and requiring a lot of effort and resources. That is why the countries at risk must take effective measures through strict border control and mandatory meetings and consultations with the responsible veterinary services from neighboring countries with established virus outbreak in order to prevent cross-border transmission.

Our experience

It is well known that at the time of intensive sheep breeding in Bulgaria Syria was our main partner in exporting tens of thousands of lambs, suckling lambs and sheep. At the time of the first PPR outbreak in Syria (originally detected in assemblages of animals from different countries), it was alleged that the source of infection were the Bulgarian animals. Imports were suspended and due money transfers were blocked. The investigation conducted on Syrian territory proved the untenability of those accusations by highlighting several circumstances. The key argument was that the disease was circulating in the African countries in a west-to-east direction and had not reached the European continent, therefore Bulgaria, where it was not found either in individual farms or in assembly centres along the Black Sea coast in preparation of the animals for export. In addition, after inspection of documents, cattle centres and holdings, surveys and autopsies in Syria, it was established that the actual disease was peste des petits ruminants, confirmed by isolation of the virus at the reference laboratory in Brendbury, England, in samples taken and sent personally by FAO representative Dr. Singh, with whom we met and talked in person. Wrong actions and failings were also found in relation to the placement and distribution of imported animals. After unloading from the ships, animal were taken to assembly centres in the city of Hamas, East Syria, and then distributed to fattening farms, both in the city itself and in nearby and further distant areas of the country. In the holdings, imported animals were placed together with natively-bred animals as well as with animals imported from other countries, including Hungary. The same was true of small farms, where 20-30 young ovine animals were kept for further fattening. Young ovine animals arriving from Bulgaria and Hungary, where the disease did not exist, became infected on the territory of Syria, and, being more susceptible, suffered more severely than local sheep breeds, creating a deceptive impression that the disease had been imported with them. The farms where imported animals were fattened did not comply with the "full-empty" principle and animals from previous

lots were grouped together with animals from new lots. These and other arguments gave us reason to refute the allegations of the Syrian services against our country. Furthermore, we acknowledged that the strategy and tactics of the struggle for the eradication of the disease was within the competence of the national veterinary services and dependent on the epizootic situation, the structure of the sheep breeding, the economic and geographic features of the country and the rules generally accepted for fighting that dangerous disease. We competently proved that our country was unaffected by the peste des petits ruminants disease and facilitated the agreement for payment of the blocked $ 3 million funds, which the Damascus Central Bank instructed the UBAF in London to transfer to the Bulgarian side. The restricted import of sheep, suckling lambs and lambs from Bulgaria was also restored. At the request of the Syrian veterinary authorities, we presented them in writing and in detail with these findings and relevant recommendations for the protection of animals imported from Bulgaria against this disease. Later communication from the Ministers of Agriculture and Trade revealed that the infection had been imported through smuggled livestock from Lebanon.

This case confirms that the epizootic characteristics of the disease require a serious approach to diagnosis and outbreak announcement, especially when the suspected occurrence is in a country previously unaffected by the disease.

In our country, which was unaffected by PPR, this dangerous disease was detected in sheep and goats in several villages in 2018. However, there are no research reports from the scientific community, despite the importance of the disease and the economic losses incurred. This is also necessary as to identifying the source of the infection, the route of the infection into the flock, the characteristics of epizootiology and the causative agent, as well as the measures for its eradication. The reports in the daily press and media appear to differ seriously from the well-known characteristics of PPR discussed herein. Our experience is related to the disease that originated in Syria. This gives us reason to announce some important details, all the more so that our country was accused of being the source of the infection. Investigations and their findings may be in favor of serious scientific research by the veterinary scientific community. Their results should offer proper assessment of the situation that occurred in order to avoid similar complicated state of affairs in the future. The nation needs a precise and competent response from the veterinary science. Lack of such will always leave the public and professionals in doubt as to the origins of PPR occurring for the first time in a European country without elucidating the source of infection. All the more so, given the drastic and extreme measures taken to limit the disease administratively thus announced.

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