THE PERSPECTIVE OF NEW ERA SCIENCES
INTERNATIONAL SCIENTIFIC JOURNAL
www.global-science-review.com
editor@global-science-review.com

# ETIOLOGY, DIAGNOSIS AND TREATMENT OF PASTEURELLOSIS IN SMALL RUMINANTS

## (BASED ON A REVIEW OF THE LITERATURE)

Komilov H.
Assistant Lecturer
Tashkent Branch of Samarkand State University of Veterinary Medicine, Animal Husbandry and Biotechnology

**Abstract.** This article examines the origin, spread and causes of pasteurellosis in small ruminants; its economic impact; pathological and anatomical changes; diagnosis and differential diagnosis; treatment; prevention; and measures for disease control.

**Keywords.** Pasteurellosis, enzootic, epizootic, alimentary, necrotic, etiology, pathogenesis, clinical, pathological and anatomical, diagnosis, differential diagnosis, immunity, prevention, disinfection.

**Relevance of the topic.** At present, providing the population with high-quality livestock products and preventing the spread of infectious diseases are among the priority tasks of veterinary specialists. According to the literature and researchers' reports, many animals currently die from infectious diseases. These diseases also reduce productivity on livestock farms and may result in animal deaths or premature slaughter. More seriously, some diseases can be transmitted to humans, causing substantial economic losses or the risk of premature death. Such diseases include brucellosis, tuberculosis, foot-and-mouth disease and pasteurellosis. Since pasteurellosis occurs in almost all animal and bird species and may also be transmitted to humans, we decided to study this disease.

Pasteurellosis (Latin/English: pasteurellosis; Russian: haemorrhagic septicaemia) is a contagious infectious disease of mammals and birds. In its acute form, it is characterized by haemorrhagic septicaemia, lobar pneumonia, pleuritis

and oedematous swelling in various parts of the body. In subacute and chronic forms, it is characterized by suppurative-necrotic pneumonia, keratoconjunctivitis, arthritis, mastitis and haemorrhagic enteritis.

**Economic impact.** The economic losses caused by pasteurellosis are very high. Mortality may reach 70–75%. In addition, the productivity of sick animals decreases sharply: animals lose weight and milk production declines. Considerable expenditure is required for treatment and prevention.

Causative agent. Pasteurella multocida is a small, Gram-negative, non-motile, non-spore-forming bacterium. It is usually found singly or in pairs and occasionally arranged in chains. It stains with all aniline dyes. In the tissues of diseased animals, pasteurellae are small and ovoid (0.3–1.25 × 0.25–0.5 μm). They show bipolar staining with methylene blue or Giemsa–Romanowsky stain. They form a capsule in fresh culture.

Pasteurellae are facultative aerobes and grow at 37°C on conventional liquid and solid culture media. Bacterial growth causes the broth to become turbid. Epizootic strains of Pasteurella are highly virulent for white mice.

Resistance of the causative agent. Pasteurellae die rapidly under natural conditions. They can survive for up to 2–3 weeks in manure, blood and cold water; up to 4 months in carcasses; and up to 1 year in frozen chicken meat. Direct sunlight destroys them within a few minutes, while exposure to 70–90°C kills them within 5–10 minutes. Disinfectants at commonly used concentrations rapidly inactivate pasteurellae.

**Epizootiological information.** All species of domestic and wild mammals and birds are susceptible to pasteurellosis. The disease also occurs in humans. Among chickens and rabbits, it usually appears as an epizootic; epizootics are less common in other animal species. Horses and carnivores are relatively more resistant to pasteurellosis.

The sources of infection are sick animals and recovered animals that carry Pasteurella. The carrier state may last for 1 year. Long-term carriage of the pathogen by apparently healthy animals is characteristic of pasteurellosis. Cattle and sheep of all ages can become infected, although young animals are more susceptible. Characteristic features of the disease include its enzootic nature and the formation of persistent epizootic foci.

**Etiology and transmission.** Pasteurellae enter the body mainly through the respiratory and alimentary routes, via the mucous membranes of the mouth, nose, eyes and digestive tract.

In tropical countries, the disease spreads as an epizootic during the rainy season, with high mortality (70–100%). In temperate climates, the disease is observed in autumn and spring (1–53%). Sick animals shed the pathogen in nasal discharge, exhaled air, saliva and faeces.

Buildings, air, hay, bedding and equipment contaminated with pasteurellae shed by sick animals serve as factors in disease transmission.

The incidence and mortality rates depend on the virulence of the pasteurellae, the immunological status of the herd, animal housing and feeding conditions, and the timely implementation of disease-control measures.

Moving animals without taking the farm's pasteurellosis status into account, overcrowding, failure to comply with veterinary and sanitary regulations, and the use of untreated waste products play an important role in the spread of the disease.

**Pathogenesis.** Under natural conditions, pasteurellae enter the body through the respiratory or alimentary routes and, less commonly, through the skin. They multiply at the site of entry, then enter the blood and lymph, causing septicaemia; death may occur within 12–36 hours. Septicaemia and toxic substances produced by pasteurellae sharply reduce phagocytic activity. The toxic substances damage capillaries, resulting in extensive subcutaneous oedema and haemorrhagic diathesis. The higher the virulence of the pathogen, the more rapidly septicaemia develops. In

resistant animals, or when the infecting pasteurellae have low virulence, septicaemia does not develop. The disease then follows a subacute or chronic course, with the pathogen localizing mainly in the lungs and causing lobar or serous-catarrhal inflammation. In peracute and acute cases, lobar pneumonia does not have time to develop; therefore, pulmonary oedema and hyperaemia are observed.

**Course, clinical signs and forms.** The incubation period ranges from several hours to 2–3 days. It depends on the animal's age and resistance, as well as the dose and virulence of the pathogen. The disease is characterized by a sudden rise in body temperature to 41–42°C, depression and, within a few hours, cardiac failure or pulmonary oedema. In some cases, animals die without showing clinical signs.

In the acute course, general depression, lethargy, anorexia and fever of up to 41°C are observed. The muzzle is cold and dry; rumination and milk production cease. At the onset of the disease, intestinal motility and defecation slow down, and the faeces become loose and may sometimes contain blood. Bloody nasal discharge, conjunctivitis and haematuria may also occur. Animals die within 1–2 days due to septicaemia and cardiac failure.

In the subacute course, in addition to fever, localized changes occur in three forms: oedematous, thoracic and intestinal.

In the oedematous form, painful, warm fluid accumulations develop rapidly in the subcutaneous tissues beneath the jaw, in the neck, abdomen and limbs. If the tongue and throat are affected, breathing becomes noisy and difficult; watery nasal discharge and thick, sticky saliva flow from the mouth. Numerous haemorrhages are observed in the mucous membranes.

In the thoracic form, lobar fibrinous pneumonia develops, accompanied by depression, anorexia, atony, difficult and rapid breathing, painful coughing and serous, frothy nasal discharge. Bloody diarrhoea occurs at the terminal stage, and the animal dies within 5–8 days.

In the intestinal form, the main signs are characterized by severe damage to the digestive system. Signs of pneumonia are not prominent, but progressive anaemia and general weakness are observed.

In the chronic course, respiratory and digestive signs are less pronounced than in the intestinal form; however, diarrhoea leads to cachexia.

In sheep, the disease rarely occurs as acute septicaemia. Fever, depression, subcutaneous oedema beneath the jaw, in the neck and abdomen, and pleuropneumonia are observed. The affected sheep becomes severely debilitated and dies within 2–5 days.



**Figure 1.** *Pathological and anatomical changes in the internal organs of a sheep that died from the disease.*

**Pathological and anatomical changes.** These changes depend on the duration and form of the disease. In animals that die from peracute and acute forms, haemorrhagic diathesis is observed (haemorrhages and inflammation in organs and on mucous and serous membranes). The liver and kidneys are damaged, the spleen is slightly enlarged, and the lymph nodes are swollen and dark red. Serofibrinous infiltrates are visible in the subcutaneous tissues in various parts of the body,

particularly in the oedematous form. Pulmonary oedema is characteristic of the initial stage of lobar pneumonia. In the intestinal form, fibrinous-haemorrhagic inflammation is observed in the gastrointestinal tract. In subacute and chronic cases, carcasses are emaciated and anaemic; the peribronchial lymph nodes are enlarged, reddened and contain numerous haemorrhages. Foci of necrosis are visible in the lungs. The spleen is slightly enlarged, and small necrotic foci are present in the liver and kidneys.

**Diagnosis.** A preliminary diagnosis of pasteurellosis is made on the basis of clinical signs, history, pathological and anatomical changes, and epizootiological data. The definitive diagnosis is established by laboratory examination of a smear prepared from the blood of a sick animal and stained with Giemsa–Romanowsky stain. If an animal dies from the disease, an official report is drawn up and a post-mortem examination is performed by the responsible animal-care worker, the animal owner or farm manager, together with a veterinarian. The intact heart and spleen, pieces of the liver and lungs, and a portion of the kidney are sent to the laboratory as pathological material. They must be preserved in a 40% aqueous glycerol solution, kept cool and dispatched with a covering letter within 3–5 hours.

**Differential diagnosis.** In sheep, pasteurellosis should be differentiated from anthrax, piroplasmosis, clostridial diseases and streptococcal infections.

**Treatment.** Treatment should begin by isolating sick animals from the herd and eliminating the factors that caused the disease. Sick animals should be provided with separate accommodation in a quiet, warm and dry building and given nutritious feed. Modern antibiotics (Penstrip 400, enrofloxacin and tylosin) are used after the pathogen's susceptibility to them has been determined. Sulfonamide and nitrofuran preparations are also used.




**Figure 2.** *Modern medicinal preparations used in treatment.*

Hyperimmune serum against pasteurellosis is effective when administered at the onset of the acute disease, as soon as the first clinical signs appear. The serum is administered subcutaneously or intravenously at twice the prophylactic dose (1.5–2 ml/kg). Treatment efficacy increases when the serum is administered together with antibiotics (penicillin, streptomycin) and sulfonamide preparations. The duration of treatment depends on the animal's condition.

**Immunity.** Animals that recover from pasteurellosis develop immunity lasting 6–12 months. For active immunization of cattle, sheep and pigs, a formol vaccine and a polyvalent vaccine against pasteurellosis, salmonellosis and colibacillosis (radio-vaccine) are used. Immunity lasts for one year.

For passive immunity, hyperimmune serum is used only for preventive purposes. It is administered to newly introduced livestock.

Vaccines are used for preventive purposes and are mandatory on affected farms and in high-risk areas. Dry live vaccines are used to prevent pasteurellosis in poultry. For passive immunization of cattle, buffaloes, sheep and pigs, hyperimmune serum against pasteurellosis is administered prophylactically to adult animals before transportation. Young animals (calves, piglets and lambs) receive it on the first day after arrival at livestock complexes.

**Prevention.** Animals, poultry and feed should be sourced only from farms free of pasteurellosis. Animals should be provided with opportunities for exercise in designated areas and fed balanced rations rich in nutritious, high-quality feed. The introduction of other animals onto the farm should be restricted. If the disease has previously been identified on the farm, all animals should be vaccinated against pasteurellosis annually, and only vaccinated animals should be admitted to the farm. Newly arrived animals must remain under preventive observation for 1 month.

**Control measures.** If pasteurellosis is confirmed by laboratory testing among cattle, sheep or goats on a farm, the area is declared affected by a decision of the local authority, based on a report by the chief veterinary officer of the district (city), and restrictions are imposed. All animals on the farm undergo clinical examination; sick animals are isolated and treated, while the remaining animals are vaccinated against pasteurellosis. Routine disinfection is carried out whenever a sick animal is detected and every 10 days thereafter until restrictions are lifted. Carcasses of animals that have died are burned. Restrictions are lifted 14 days after all animals have been vaccinated and final disinfection has been completed.

**Conclusion**. Based on our study of the disease, we reached the following conclusions: almost all animal and bird species are susceptible to pasteurellosis, and the disease may even be transmitted to humans. In addition, substantial financial resources are required for treatment and prevention. Therefore, veterinary and sanitary regulations must be observed on farms, and animals must be vaccinated against the disease in a timely manner.

**References:**

1. X. S. Salimov, A. A. Qambarov, I. X. Salimov. Practical Training Manual for the Course “Epizootology and Infectious Diseases”. Samarkand, 2024.
2. X. S. Salimov, A. A. Qambarov. Epizootology. Textbook. Samarkand, 2016.
3. M. N. Mamatova, S. U. Alamova. Special Epizootology. Tashkent, 2006.
4. Effective methods of clinical and laboratory diagnosis and treatment of hoof diseases in large animals. Komilov Kh.R. Normamatov R.Q. Kasimov S.J. World scientific research journal.

