**Hemorrhagic Septicemia**

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**Importance**

Hemorrhagic septicemia is a highly fatal bacterial disease seen mainly in cattle and water buffalo. In susceptible animals, the clinical signs often progress rapidly from dullness and fever to death within hours. Because the disease develops so quickly, few animals can be treated in time, and recovery is rare. Subclinical carriers can introduce hemorrhagic septicemia into a herd. Young animals are mainly affected in endemic regions, and outbreaks are particularly common during rainy weather, when the organism can spread readily. In areas where cattle have no immunity, severe disease is expected to occur in all ages.

**Etiology**

Hemorrhagic septicemia results from infection by *Pasteurella multocida* subsp. *multocida*, a Gram-negative coccobacillus in the family Pasteurellaceae. *P. multocida* can cause a variety of diseases in animals, but only two serotypes of this organism classically cause hemorrhagic septicemia. Because two systems, the Namioka-Carter and Carter-Heddleston systems, are used to designate *P. multocida* serotypes, each serotype has two names. One is known as B:2 in the Carter-Heddleston system and 6:B in the Namioka-Carter system. The other is E:2 in the Carter-Heddleston system and 6:E in the Namioka-Carter system. In both systems, the letter stands for the capsular antigen and the number designates the somatic antigen. In India, the serotypes A:1 and A:3 (Carter-Heddleston system) have also been linked to hemorrhagic septicemia.

Septicemic pasteurellosis, a disease that is clinically indistinguishable from hemorrhagic septicemia, has been reported sporadically from wild ruminants, especially cervids. This disease may also be called hemorrhagic septicemia in the literature. A variety of serotypes including B:3,4, A:3,4 and B:1 have been linked to septicemic pasteurellosis.

**Species Affected**

Epidemics of hemorrhagic septicemia occur mainly among cattle and water buffalo (*Bubalus bubalis*). These two species are also the major reservoir hosts. Outbreaks have been reported occasionally among pigs in some Asian countries, and cases are seen infrequently in sheep and goats. Goats have been infected experimentally. Hemorrhagic septicemia has also been reported in bison (*Bison bison*), African buffalo (*Syncerus caffer*), camels, elephants, horses, donkeys and yaks. Experimental infections are readily established in laboratory rabbits and mice.

Systemic pasteurellosis has been reported in various species of deer including fallow deer (*Dama dama*), sika deer (*Cervus nippon*) and chital deer (*Axis axis*), as well as elk (*Cervus elaphus canadensis*), pronghorn (*Antilocapra americana*) and other wild ruminants.

**Geographic Distribution**

Hemorrhagic septicemia is an important disease of cattle and water buffalo in Asia, Africa and the Middle East. The highest incidence is in Southeast Asia. Cases have also been reported in some countries of southern Europe. The B:2 serotype causes hemorrhagic septicemia in Asia. It has also been isolated in southern Europe, the Middle East, and parts of Africa. The E:2 serotype has been reported only in Africa. Type E strains appear to have decreased recently in southern Africa, while serotype B has become more prevalent. Neither the E:2 nor the B:2 serotype of *P. multocida* is known to occur among domesticated animals in the Americas, Australia or New Zealand.

Septicemic pasteurellosis can occur among wild ruminants in countries where hemorrhagic septicemia is absent. Some countries that have reported outbreaks include Denmark (fallow deer and sika deer), the United Kingdom (fallow deer), Australia (fallow deer and chital deer), and the United States (pronghorn and elk). A variety of serotypes have been reported from these outbreaks. Serotypes other than B:2 or E:2 were isolated from North American cervids.
In the U.S., the last reported outbreak of hemorrhagic septicemia occurred in a dairy cattle herd in 1968, but a reservoir might exist among the Yellowstone herd of bison. An outbreak among these animals in 1922 was caused by serotype B:2. Another outbreak among bison in Montana in 1965 was thought to be the same organism, but was later found to be serotype B:3,4. The World Organization for Animal Health (OIE) currently classifies the U.S. as “disease suspected” and Canada as “disease never occurred” for hemorrhagic septicemia. However, this disease would be considered exotic and immediately reportable if it occurred among domesticated animals in either country.

Transmission

*P. multocida* is transmitted by ingestion or inhalation, either during direct contact or via fomites such as contaminated feed and water. The serotypes that cause hemorrhagic septicemia are probably shed into the oropharynx. Some animals become carriers, maintaining the organism in the lymphatic tissues associated with the upper respiratory tract, and periodically shedding it in nasal secretions. Excretion may be triggered by stress.

*P. multocida* does not remain viable for long periods in the environment, but it can survive for hours and possibly days in damp soil or water. Rainy conditions and high humidity facilitate transmission. Biting arthropods do not seem to be important in the epidemiology of this disease.

Incubation Period

The incubation period is usually 3 to 5 days but some animals can carry the organism for varying periods without symptoms. In experimental infections with lethal doses, cattle or buffalo develop clinical signs within a few hours and die within 18 to 30 hours.

Clinical Signs

Most cases in cattle and water buffalo are acute or peracute. Although the disease is very similar in both species, buffalo tend to have more severe clinical signs and a shorter course of disease. A fever, dullness and reluctance to move may be the first signs. Salivation and a profuse serous nasal discharge develop, and edematous swellings become apparent in the submandibular region. These swellings spread to the neck and brisket. In calves, hemorrhagic gastroenteritis has also been reported. Respiratory distress occurs, with frothing at the mouth, and the animal usually collapses and dies 6 to 48 hours after the initial clinical signs. Either sudden death or a protracted course up to a few days is also possible. Animals with clinical signs, particularly buffalo, rarely recover. Chronic cases have not been reported.

Similar clinical signs including severe depression, profuse salivation, edema of the head, neck and brisket, and severe respiratory distress with foamy nasal discharge have been reported in some wild ruminants with systemic pasteurellosis.

Post Mortem Lesions

At necropsy, cattle and buffalo often have widespread hematomas, edema and hyperemia. Subcutaneous edema is usually present in the submandibular region and neck, sometimes extending to the brisket. The edema consists of a gelatinous mass with straw-colored or bloodstained fluid. Similar swellings may also occur in the musculature. The lymph nodes may be enlarged, and the thoracic and abdominal cavities and pericardial sac often contain blood-tinged fluid. Petechiae are frequently found on many organs, especially on the serosal surface, throughout the body. They are common in the pharyngeal and cervical lymph nodes and on the heart, and may also occur in the subcutaneous connective tissues. Ecchymotic hemorrhages are sometimes noted, especially on the heart. The lungs are diffusely congested and edematous, but extensive pneumonia is atypical. The gastrointestinal tract may be hyperemic and congested to varying degrees, and the abomasum may contain petechial hemorrhages and ecchymoses. In some peracute cases, there may be few or no lesions other than a few scattered petechial hemorrhages. In goats inoculated with *P. multocida* B:2, the necropsy findings resembled cases among cattle, but subcutaneous edema of the submandibular region, neck and brisket was absent.

Lesions similar to those found in cattle and buffalo occur in cervids with septicemic pasteurellosis.

Morbidity and Mortality

The morbidity rate depends on environmental conditions, herd management, the animals’ immunity and other factors. Although hemorrhagic septicemia can be seen at any time of the year, close herding and wet conditions contribute to the spread of the disease; the worst epidemics occur during the rainy season. Stressors such as poor nutrition increase an animal’s susceptibility to clinical disease, and also stimulate shedding of the organism from carriers.

All ages are affected where hemorrhagic septicemia is not endemic, and the morbidity rate can be high. In endemic regions, outbreaks often occur when healthy carriers are introduced into a herd. In these areas, most adults have some immunity to the organism, and clinical cases tend to occur in young animals between the ages of 6 months and 2 years. However, massive epizootics are sometimes seen. The case fatality rate is nearly 100% unless the animal is treated very early; few animals survive once the clinical signs have become apparent. A few spontaneous recoveries may be seen, especially late in an outbreak. Up to 20% of the survivors can become carriers for a short period after an outbreak; by 6 months, the carrier rate is 5% or less. Buffaloes are thought to be more susceptible to illness than cattle, with higher morbidity and mortality rates.

Susceptibility to systemic pasteurellosis may vary among the Cervidae. In a series of outbreaks at a deer park.
in Denmark, more than 300 clinical cases occurred in fallow deer (*Dama dama*), but only a single death due to this disease observed in a sika deer (*Cervus nippon*). Red deer (*Cervus elaphus*) in the park were unaffected.

**Diagnosis**

**Clinical**

Hemorrhagic septicemia should be suspected in animals with a rapid course of infection, and fever and edematous swellings in the throat, neck and brisket. A high herd incidence and high case fatality rate are also suggestive of this disease. Sporadic cases of hemorrhagic septicemia may be difficult to diagnose.

**Differential diagnosis**

The differential diagnosis includes other causes of sudden death such as lightning strikes, blackleg (*Clostridium chauveoi* infection), rinderpest and anthrax. Acute salmonellosis and pneumatic pasteurellosis should also be considered.

**Laboratory tests**

Hemorrhagic septicemia is usually diagnosed by culturing *P. multocida* B:2 or E:2 from affected animals. Isolation is more likely to be successful if a fresh sample that is free from contaminating bacteria and post-mortem invaders can be collected. In Gram–stained blood or tissue smears, *P. multocida* is a Gram–negative, short bacillus or ovoid form with bipolar staining. Bipolar staining is more apparent with methylene blue or Leishman’s stain, and may be lost after serial passage. Some pleomorphism can be expected, especially in older cultures. *P. multocida* can be grown on blood agar, chocolate agar or casein/sucrose/yeast (CSY) agar with 5% blood. Dextrose starch agar or trypticase soy agar may also be used. Freshly isolated *P. multocida* colonies are smooth, nonhemolytic, grayish, translucent, glistening and approximately 1 mm in diameter after incubation on blood agar for 24 hours at 37°C. Larger colonies are seen on CSY agar, and smaller colonies may develop from old cultures. Biochemical and serological tests are used for identification. In addition to the other biochemical characteristics identifying the organism as *Pasteurella multocida*, the serotypes that cause hemorrhagic septicemia produce hyaluronidase. Tests used for serotyping include a rapid slide agglutination test or indirect hemagglutination test for capsular typing, an agglutination test for somatic typing, agar gel immunodiffusion for both capsular and somatic typing, and counter-immunoelectrophoresis for the rapid identification of the capsular types B and E. DNA fingerprinting and other genetic methods can characterize isolates further for epidemiological investigations, but are generally available only in research laboratories. Polymerase chain reaction (PCR) techniques can detect organisms in clinical samples and bacterial cultures.

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If *in vitro* culture is unsuccessful, samples may be inoculated into a mouse, and blood from the mouse used to identify the organism. This technique can be helpful when the carcass has been overgrown with invading bacteria during postmortem decomposition.

Clinically affected animals usually die rapidly, and serological assays are not normally useful in diagnosis; however, high titers (1:160 or higher by indirect hemagglutination) in surviving in–contact animals are suggestive of the disease.

**Samples to collect**

Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease. There are no confirmed reports of human infections with *P. multocida* serotypes B:2 and E:2; however, other serotypes do cause human infections and precautions should be taken to avoid exposure.

Although they are not consistently present, *P. multocida* B:2 and E:2 may be found in the nasal secretions of some live animals. Hemorrhagic septicemia strains can also be cultured from blood; however, animals become septicemic only in the terminal stages of the disease, and some blood samples collected before death may not contain the organism. At necropsy, the organism can be recovered from a blood sample or swab collected from the heart within a few hours of death. Other visceral organs may also be sampled. The spleen and brain are among the last organs to be colonized by bacteria post-mortem. If a necropsy is not feasible, blood samples can be taken from the jugular vein by aspiration or incision. Blood and organ samples should be kept cool and transported on ice.

A long bone freed of tissue should be taken from animals that have been dead for a long time; the marrow is cultured after sterilization of the bone’s surface. Serum samples are occasionally useful for retrospective diagnosis in survivors or other animals in the herd.

**Recommended actions if hemorrhagic septicemia is suspected**

**Notification of authorities**

Hemorrhagic septicemia must be reported to state or federal authorities immediately upon diagnosis or suspicion of the disease.

Federal Area Veterinarians in Charge (AVIC):

State Animal Health Officials (SAHOs):
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Control

Hemorrhagic septicemia can be eradicated with quarantines, movement controls, tracing of contacts, euthanasia of infected and exposed animals, and cleaning and disinfection of the premises. *P. multocida* is susceptible to most common disinfectants, as well as to mild heat (55°C/131°F).

In endemic areas, this disease is mainly prevented by vaccination. The removal of carriers from the herd is also helpful. Management to keep the animals in good condition can reduce the risk of clinical signs and/or transmission of the organism. Animals should not be crowded or stressed, especially during wet weather. Antibiotic treatment is effective only if it is started very soon after the onset of clinical signs.

Public Health

There are no reports of human infections with *P. multocida* serotypes B:2 and E:2; however, other serotypes of this organism do infect humans, and precautions should be taken to avoid exposure.

Internet Resources

http://www.spc.int/rahs/

The Merck Veterinary Manual
http://www.merckvetmanual.com

United States Animal Health Association. Foreign Animal Diseases

World Organization for Animal Health (OIE)
http://www.oie.int

OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals
http://www.oie.int/international-standard-setting/terrestrial-manual/access-online/

OIE Terrestrial Animal Health Code
http://www.oie.int/international-standard-setting/terrestrial-code/access-online/

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References


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