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ANTHRAX IN DOGS

Anthrax infection in dogs is described in this article. Historic facts of anthrax outbreaks in dogs, exposure routes, clinical and pathologic signs, serological evidence of exposure in wild animals, and regimens and doses of antibiotic therapy for domestic dogs are provided. The need to study anthrax prevalence in carnivores during anthrax outbreaks is supported.

Anthrax (German – Milzbrand; French – maladie du charbon, fièvre charbonneuse; Russian – сибирская язва; Ukrainian – сибірка) is an acute infectious zoonotic disease caused by the spore forming bacterium *Bacillus anthracis*. Anthrax is an especially dangerous infection, often with peracute or acute symptoms and a high fatality rate in susceptible animals. The name of the microorganism comes from the Greek word ἄνθραξ (anthrax) for coal, because of the formation of similarly coloured lesions on skin during infection [5].

In susceptible animal species, anthrax is a peracute disease; some animals may succumb to the disease within hours of the first clinical signs or in some cases without developing any clinical signs. There also are documented acute, subacute (animals die on 2nd–3rd day), chronic (lasts 2 months and longer) and atypical courses of this disease [6]. As animal carcasses decompose, *B. anthracis* sporulates, enabling it to survive in the environment and remain virulent for long periods of time, including many decades or longer, creating preconditions for new anthrax outbreaks in these foci [6].Carnivores are most likely infected by scavenging infected carcasses, although they may be exposed through insect bites (percutaneous route), or through spore inhalation, though evidence for inhalation in wildlife is speculative. Humans are usually infected after contact with infected animals, though other transmission routes are possible [5, 11].

Anthrax epizootics are reported nearly worldwide. Human cases of anthrax are usually registered in agrarian and underdeveloped countries [14]. According to data from the State Veterinary and Phytosanitary Service, reported anthrax outbreaks were rare in the last decade with few sporadic cases across Ukraine. As in the majority of agrarian countries, the national anthrax control strategy of Ukraine consists of passive monitoring

of anthrax in cattle. Cattle preventive vaccination and carcass and pasture decontamination during anthrax outbreaks are mandatory.

The idea that anthrax is prevalent predominantly in hoofed animals is widely accepted in post-soviet countries [2, 4, 6]. Ipatenko et al. (1996) also reported on possible anthrax cases in dogs, wolves, white polar bears, lions, tigers, panthers, cheetahs, pumas, foxes, minks, martens, arctic foxes, coypus, raccoons, and even birds [6]. It has also been reported that cats are susceptible only in young age. In his monograph, Kolesov (1976) points out that cat, dogs, wolves, foxes and arctic foxes are susceptible, but resistant to anthrax [2].

Despite evidence to the contrary, some Ukrainian scientists [1, 3] insist that dogs are resistant to anthrax. As a result, we believe it is necessary to review these opposing views of animal susceptibility, to ensure that future veterinarians and public health professionals are receiving accurate information on this consequential disease.

Reports on anthrax in dogs are limited in the scientific literature. In 1957, an outbreak of anthrax in a hound kennel in Kennett, Suffolk, England, was attributed to feeding raw meat from a Jersey cow that had died of anthrax [9]. Clinical findings included fever (39,4–40,5 °C), oedematous swelling of the face and throat, generalized lymphadenopathy, renal congestion, patches of haemorrhage of the enteric serosa, and slight but equivocal splenomegaly. Death was attributed to asphyxia. In 1971, three dogs died from anthrax in Louisiana, but neither clinical nor pathologic findings were described [12].

McGee et al. (1994) reported that during the anthrax outbreak in Mississippi delta a 6-year-old male Labrador retriever was examined on 13 September 1991, two days after the initial clinical signs of ptyalism and swelling of the right forelimb. Findings included a rectal temperature of 41,1 °C and a total leukocyte count of 25,900 μL with 94 % granulocytes. Treatment with sulfadiazine/trimethoprim and clindamycin was begun on an outpatient basis. The dog was sent home but died later that day on the owner's premises. Necropsy findings within 1 hour postmortem included splenomegaly, a very friable liver, and blood in the stomach, small intestine, and colon. A haemorrhagic wound on the right foreleg was suspected to be a puncture wound or a spider bite. Only formalin-fixed kidneys, lungs, heart and spleen were collected for further examination. Histopathologic findings in hematoxylin and eosin-stained sections of spleen included generalized congestion, multifocal lymphocyte necrosis with fibrin exudation, and large numbers of degenerate neutrophils and intact bacterial rods throughout the red pulp. Periarteriolar lymphoid sheaths in the white pulp were found in low numbers. They contained scattered necrotic cellular debris, which were interpreted to represent lymphocytolysis. Discrete lymphoid follicles were not found. Pulmonary changes included congestion, multifocal atelectasis, bacterial rods and neutrophils in the alveolar septa1 capillaries, variable intraalveolar fibrin exudation, and edema. Bacteria were also within hepatic sinusoids, renal glomerular capillaries, and intramural myocardial blood vessels [17].

The case described above differs from those described in Suffolk by having conclusively confirmed splenomegaly and severe haemorrhagies in intestines, which is consistent with the intestinal form of anthrax. Also, this case did not have a significant throat oedema, and death was attributed to septicemia and toxemia, instead of asphyxia [17].

In summary, *B. anthracis* enters dogs through the pharynx and upper regions of the gastrointestinal tract. Therefore, lymph nodes in these regions are most frequently affected, and severe swelling of the head, neck and mediastinal regions is the most common clinical sign [9, 21]. Death is often caused by toxaemia and shock, although asphyxia can also play a role. In one dog case, haemorrhagic gastroenteritis, ptyalism, and swelling of forelimb were described [12].

More recently, serological monitoring of diverse animal species in the Serengeti ecosystem in Tanzania, Africa, yielded high seroprevalence among carnivores, which in the authors' opinion, suggests regular nonfatal exposure to *B. anthracis* [16]. Exposure patterns in dogs reflected known patterns of endemicity and provided new information about anthrax in the ecosystem, which indicated the potential of dogs as indicator species [16].

On 20 August 2012, a domestic dog died from anthrax on a private yard in Voznesenka village, Melitopol district, Zaporizhia oblast, Ukraine. The dog was fed meat and bones of a culled heifer, which was diagnosed with anthrax 5 days prior. On the evening before its death, the dog refused food and water. No other clinical signs were noted. Veterinary service specialists examined the carcass of the dog, collected biological samples and submitted them to State Regional Diagnostic Laboratory of Veterinary Medicine in Zaporizhia for laboratory examination. No pathological findings were recorded as necropsy was not performed. Anthrax was confirmed by laboratory methods.This was the first confirmed case of anthrax in a dog in Ukraine. It was characterised by limited clinical signs and a short period between the appearance of clinical signs and death.

The relative rarity of reports of anthrax outbreaks in domestic dogs and wild canines may be linked to experiments conducted by Gleiser et al. (1968) in the 60s of the last century [13]. These experiments indicated that dogs are resistant to systemic anthrax. However, these experiments inoculated dogs via a respiratory route, while it appears that dogs and other canines are usually infected through ingestion of infected carcasses. Since 1945, researchers have realized that carnivores become infected through consumption of infected carcasses [20], and recent field and case reports of the last two decades indicate that domestic and wild canines may be highly susceptible to anthrax after ingesting meat from infected carcasses [8, 17]. For example, anthrax was suspected in North American wolves during an anthrax outbreak in bison [19], was confirmed in African wild dogs [8], and reported in black-backed jackals in Namibia [7] and coyotes in North America [18]. These animals often scavenge animal carcasses during anthrax outbreaks.

The fatal case of anthrax in a dog in Voznesenka, as well as new data on seroprevalence in canines during anthrax outbreaks [16] suggest anthrax can affect a wider range of species and not only ruminants. The lack of specific clinical signs and subacute course of the infection in infected dogs indicates the need of a wider understanding of anthrax epidemiology and ecology. Broadly, the Ukrainian anthrax monitoring system should be more flexible. Anthrax outbreak investigations should encompass animal species which might be not examined otherwise, including canines and wild fauna. Dogs may be carriers of *B. anthracis* and contaminate the environment by excreting bacteria with faeces [10]. Veterinary doctors must be aware of the anthrax risk in non-ruminant species, monitor the associated wild fauna, and be able to diagnose anthrax and perform preventive therapy if necessary.

Ante-mortem diagnosis is based on clinical signs and demonstration of the microrganism in blood, lymph nodes, tissue aspirates, and pharyngeal swabs. Secondarily, determining the source of infection can provide epidemiological traceback to aid in diagnosis. Importantly, field and clinical personnel should be aware that anthrax spores survive nearly all microbial staining techniques, including heat fixing in Gram stain. Definitive diagnosis is based on culture of the microorganism. The animals that die of anthrax are usually septicemic, so that blood samples usually will reveal the microorganism during microscopic and bacteriological examination. Necropsy is not advised if anthrax is suspected, as exposure to air rapidly causes sporulation of the vegetative bacteria. If necropsy was already performed, however, a sample of spleen, lymph nodes, intestine, lungs, liver, bronchial lymph nodes, tonsil, and pharynx should be collected. The contaminated area, including the site of the carcass and the premises where the infected animals were kept, should be treated with sporicidal disinfectant; an excellent choice is sodium hypochlorite (i.e. bleach) [15]. The decontaminating solution should have a final concentration of 5 % of sodium hypochlorite.

If a domestic animal is thought to have been exposed to *B. anthracis,* prophylactic treatment with doxycycline at 5 mg/kg orally, every 24 hours, is recommended. In animal for which doxycycline is contraindicated (e.g., pregnant or young animals), amoxicillin at 20 mg/kg orally, every 12 hours, may be substituted. The required duration of prophylactic therapy is unknown. It should probably be the same as treatment duration in humans, which is 45 – 60 days. However, we do not have sufficient data on the duration of treatment in animals. If a pet is exposed to anthrax, thorough decontamination of the fur to avoid transmission to humans is recommended. Since no present sporicidal disinfectants are safe for use on living animals, repeated bathing is advised to mechanically remove the microorganism [15].

Langston (2005) suggests that treatment of clinical anthrax must be early and aggressive, usually with immediate use of parenteral antibiotic therapy:

- Benzylpenicillin is administered immediately intravenously according to the instructions of manufacturer (as a rule 12,000–22,000 U/kg body weight) with the following administration of long acting benzylpenicillin in 6–8 hours (usually 6,000–12,000 U/kg body weight) or other long acting drugs, such as amoxicillin or clamoxil P (15 mg/kg body weight), or oxytetracycline at 5 mg/kg IV every 24 hours;
- Penicillin/streptomycin intramuscular (streptomycin dose at 25–100 mg/kg body weight) and/or administration of serum against anthrax (50–100 cm^3), anti-anthrax gamma globulin (20–40 cm^3), or in combination penicillin-serum (50–100 cm^3 of the serum and penicillin at 6,000–12,000 U/kg body weight 2 times a day, 3 consecutive days);
- Enrofloxacin at 5 mg/kg every 24 hours.

There are no data to determine which of treatment regimens is more effective, if any. In addition to antibiotic therapy, general supportive therapy should be performed.

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Описано захворювання собак на сибірку. Наведено історичні факти спалахів сибірки серед собак, шляхи зараження, клінічні й патолого-анатомічні зміни, дані про сероконверсію серед диких тварин, схеми й дози проведення антибіотикотерапії. Обґрунтовується вивчення випадків захворюваності м'ясоїдних при спалахах сибірки.

Сибирская язва у собак. *В.Г. Скрыпник, Р.В. Козий, А.В. Скрыпник, И.А. Рубленко, Дж.К. Блэкберн, К.Н. Багамиан, Дж. Фарлоу, М.-Дж. Николич, А.А. Меженский, О.М. Неволько, Дж.К. Блэкберн*

Описано заболевание собак сибирской язвой. Приведены исторические факты вспышек сибирской язвы среди собак, пути заражения, клинические и патолого-анатомические изменения, данные о сероконверсии среди диких животных, схемы и дозы проведения антибиотикотерапии. Обосновывается изучение случаев заболевания плотоядных при вспышках сибирской язвы. ○

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