Anthrax, Screwworms, and Equine Piroplasmosis—Subdued but Not Eradicated

John R. Irby, DVM

With decreasing barriers to global livestock movement (brought on by international free trade agreements) and increasing concerns of threats of bioterrorism, the risk of introducing devastating infectious diseases continues to be a concern. The constant vigilance of the veterinary community is a major key to early detection. Author’s address: Texas Animal Health Commission, Area 4 Office, 313 West Alabama, Suite 1, Mt. Pleasant, TX 75455. © 2002 AAEP.

1. Introduction

Anthrax, screwworms, and equine piroplasmosis are related in that each disease can affect equids, has the potential to devastate domestic livestock, is present in many foreign countries, and may first be detected by veterinarians in private practice. In the post-September 11th atmosphere, there is the concern that livestock could be the target of a bioterrorist attack, using a disease agent available in another country. Each disease will be discussed based on its unique etiology, clinical presentation, diagnosis, and treatment and/or control. Anthrax outbreaks affecting significant numbers of livestock in the United States is an infrequent occurrence, but the causative organism, Bacillus anthracis, is known to inhabit the soil in many parts of the country.1 The most recent U.S. anthrax outbreak in livestock occurred in a five-county area of Texas from June 7 to September 20, 2001. Animal mortalities reported by the Centers for Disease Control included 69 bovids, 29 equids, and 222 exotics.2 Millions of dollars were spent eradicating screwworms from the United States by 1966 and from Mexico by 1991, but the pest still flourishes in Panama, some Caribbean islands, and most of South America. Private veterinarians found screwworm larvae on a military dog that returned to San Antonio, Texas, from Panama in 1977 and on a polo pony that returned to Florida from Argentina in 2000.3 Even though no clinical cases have been confirmed since the early 1970s, seropositive carrier horses are believed to have entered the U.S.4

2. Anthrax

Anthrax is an acute, usually fatal infectious disease caused by B. anthracis. Whereas primarily a disease of herbivores, such as cattle, sheep, horses, mules, and goats, it can affect virtually all warm-blooded animals, including humans. Although anthrax spores have been found naturally in soil samples from around the world, the organisms cannot be regularly cultivated from soils where there is an absence of endemic anthrax. In the United States there are recognized endemic areas of infection in South Dakota, Nebraska, Arkansas, Texas,
Etiology and Epidemiology

The virulence of *B. anthracis* is associated with the presence of two plasmids that carry the genetic coding for toxin and capsule production. After bacilli are discharged from an infected animal carcass through bloody discharges from natural body openings, they form spores that are resistant to extremes of temperature, chemical disinfectants, and desiccation. For this reason, necropy of an animal that died suddenly in an endemic area and exhibits bloody discharges from body openings is not recommended. Outbreaks are commonly associated with neutral or alkaline soils infested with the spores when the minimal daily temperature is >60°F. Soil-borne epidemics may be predicted during periods when excess rainfall, drought, or overgrazing occurs.1

Clinical Findings

Typically, the incubation period is 3–7 days. The clinical course ranges from acute to chronic in equids. In the acute form, signs may include fever up to 107°F followed by a period of excitement that progresses to depression, respiratory distress, stupor, staggering, convulsions, coma, and death within 48 h. The subacute stage can last for up to 8 days and usually presents as severe colic. The spleen is enlarged and can be palpated rectally, mucous membranes are hemorrhagic, and there may be muscle rigidity. Hot, painful, rapidly progressing swelling may develop on the neck and lower abdominal region. Dyspnea, cyanosis, and coma follow, and blood may exude from body openings.5 The chronic form is less frequent and involves the tongue and pharynx. Local edema and epistaxis may cause death as a result of suffocation. A few animals may recover, but the disease persists in regional lymph nodes.

Diagnosis

Diagnosis of anthrax may be difficult in areas where the disease is not endemic. It should be suspected when animals die suddenly in or near endemic areas. The differential diagnoses should include sudden death caused by lead poisoning, sunstroke or lightning strike, malignant edema, purpura hemorrhagica, acute equine infectious anemia,encephalitis, West Nile virus, and colic.5 The bacilli may be detected in Wright- or Giemsa-stained smears of peripheral blood or edema fluid. Blood can be submitted as a dried specimen on sterile cotton umbilical tape, on a sterile swab, or on slides. On microscopic examination of stained smears, *B. anthracis* will appear as single or short-chained bacilli with blunted ends, a capsule, and centrally located endospores that are highly refractive to light and resist staining. Tissue specimens, other than dried blood, should be shipped to the laboratory under refrigeration or frozen and in a shipping container approved by the carrier.

Treatment and Control

Because anthrax is often fatal in horses, early treatment and implementation of a preventive program are essential. Potassium or sodium penicillin G at 25,000 to 55,000 units/kg, q 6 h, IV dosages may be effective early, followed in 3–4 days by procaine penicillin G at 32,500 units/kg, q 12 h, IM. Treatment should continue for 7 days. Anthrax in livestock can largely be controlled by annual vaccination of all grazing animals in the endemic area. The nonencapsulated Sterne-strain vaccine is used almost universally for livestock. Vaccination side effects, such as localized swelling, depression, and fever, are common. Analgesics such as phenylbutazone 2 mg/kg, q 12 h, IV may be used, but antibiotics are contraindicated because they nullify the vaccine. Rest for 7–10 days following the vaccination is advised.5 Vaccination should be done 2–4 wk before the season when outbreaks are suspected. Hygiene is the single most important factor in preventing the spread of disease. All contaminated materials and carcasses should be burned or covered with quicklime (calcium oxide) and buried at a depth of 2 m. The area should be quarantined, and regulatory officials should be notified. Contaminated clothing may be disinfected by soaking in 10% formaldehyde solution, and shoes may be sterilized using ethylene oxide.4

3. Screwworms (*Cochliomyia hominivorax*)

Many dipteran flies produce larvae that result in obligatory myiasis. However, the screwworm fly is the only primary invader of fresh, uncontaminated skin wounds of domestic animals.5

Etiology and Epidemiology

As a result of massive state, federal, and international eradication efforts using sterile male adults, screwworms are not found in the United States, Mexico, or any Central American country except for Panama. In addition to open wounds on imported livestock, it is important to examine the prepuce of stallions and geldings imported from prevalent areas of Central and South America and certain Caribbean Islands.6

Clinical Findings

Adult females are attracted to fresh skin wounds on warm-blooded animals where they lay batches of 200–400 eggs in overlapping rows on the wound edges. The larvae hatch in 12–21 h, migrate to the wound, and burrow into the flesh. After feeding for 5–7 days, the grown larvae exit the wound, fall to the ground, and burrow into the soil where they complete their life cycle. Wounds can become greatly enlarged because of multiple infestation, and unless treated, usually result in the death of the
animal. Equids from countries known to have screwworms are quarantined for 7 days at USDA import stations and examined for larvae beginning on day 6. A frequently noticed indication of infestation is a serosanguinous fluid dripping from the prepuce or reddish-brown staining of the hairs on the medial aspect of the rear legs of stallions and geldings. Closer inspection, involving fully extending the penis, has resulted in larvae being found inhabiting the prepuce.6

Diagnosis
Screwworm larvae can be distinguished from the larvae of other blow flies by their wood screw appearance and characteristic darkly pigmented tracheal tubes on the dorsal aspect of the caudal end of the third-stage larva. These tubes can easily be visualized through the larval cuticle. Suspect larvae should be collected, placed in formalin, and shipped to a state or federal diagnostic laboratory for positive identification.5

Treatment and Control
Screwworm infestation must be reported to both state and federal regulatory authorities so that appropriate epidemiological investigations are promptly initiated. Screwworms in wounds can be killed by direct application of a wound dressing containing coumaphos or permethrin. It is also recommended to thoroughly spray all animals on the premise with 1% coumaphos or 2% permethrin. It is also recommended to thoroughly spray all animals on the premise with 1% coumaphos or 2% permethrin to kill any undetected larvae that may be present. Extra label usage is not recommended.

4. Equine Piroplasmosis (Babesiosis)
Equine piroplasmosis is an infectious, tick-transmitted disease of equidae caused by protozoan parasites that invade and destroy red blood cells. Two species of protozoan known to affect equine, Babesia equi and Babesia caballi, are widespread in tropical and subtropical areas of the world. Only Canada, Australia, Japan, England, Ireland, and the United States are not considered endemic areas.6 Recent phylogenetic studies indicate that B. equi is a Theileria rather than a Babesia.1 Regulatory control of equine piroplasmosis relies on infected equine testing positive to the compliment fixation (CF) test at a USDA import station. It has been suspected that, before being presented for importation, infected horses are being treated with a babesicidal drug that could produce false-negative results on the CF test. There are increasing concerns that this practice could allow infected horses to enter the United States. Whereas the tick most capable of transmitting Babesia, Dermacentor nitens and Boophilus sp, are no longer widespread in this country, domestic ticks such as D. albipictus and D. variabilis could prove capable of transmission.4

Etiology and Epidemiology
Equine piroplasmosis is blood-borne and is generally transmitted by ticks.6 When outbreaks occur, ticks of the genera Hyalomma, Rhipicephalus, and Dermacentor are usually incriminated.1

Clinical Findings
Signs of equine piroplasmosis are often non-specific and can easily be confused with equine infectious anemia, equine ehrlichiosis, liver failure, autoimmune hemolytic anemia, or exposure to toxins such as phenothiazine, wild onions, or red maple leaves. B. caballi causes a less severe disease. B. equi infections are more severe, and recovered animals become lifelong carriers. The disease can occur in peracute, acute, and chronic forms. The peracute form, when horses are found dead, is rare. Acute cases are more common and are characterized by fever >40°C, anemia, icterus, anorexia, elevated respiratory and pulse rates, congestion of mucous membranes, and constipation. The animal will become emaciated and often dies within 10 days. Chronic cases usually present non-specific signs such as mild anorexia, poor performance, and weight loss. The spleen is usually found to be enlarged on rectal palpation.7

Diagnosis
Clinically, piroplasmosis is similar to other diseases causing severe anemia. It is extremely difficult to diagnose the organisms in carrier animals by means of microscopic examination of blood smears, and this method is not practical on a large scale. Serological testing with the CF test is the preferred method of diagnosis, especially before importing horses to countries where the disease does not occur, but the vector is present. Other types of tests, such as the competitive ELISA, the indirect fluorescent assay, and the polymerase chain reaction, are currently being considered by USDA as alternatives to the CF test.

Treatment and Control
There is no vaccine. Both B. caballi and B. equi respond to babesicial drugs, but B. equi is more resistant and carrier states are not uncommon. Imidocarb is the most widely used drug, but it has been proven toxic at therapeutic levels.5 Liver enzyme levels are not reliable indicators of imidocarb toxicity.5 Donkeys are subject to toxicity at the level of 2 mg/kg. Some horses are reported to have died from the treatment. For B. caballi, two treatments at a dosage of 4 mg/kg, q 24 h, IM seems effective. However, four treatments at a dosage of 4 mg/kg, q 72 h, IM are required for B. equi. Side effects such as restlessness, colic, and sweating are frequent after treatment. To offset these side effects, it has been recommended that the horse receive atropine sulfate (large animal) 0.1 mg/kg, IM and flunixin meglumine 1 mg/kg, IV 30 min before each treatment. It should be noted that atropine sulfate at this dose can produce serious ileus in the horse. The most reliable method to control equine piroplasmosis remains preventing entry of infected
equine and ensuring that animals entering from endemic countries are thoroughly checked and found to be free of ticks. Several years ago, a number of horses imported into Florida from Puerto Rico were negative before shipment but tested positive for equine piroplasmosis after arrival. To prevent another outbreak of equine piroplasmosis such as occurred in the 1960s, Florida now requires an import permit listing the destination, and the animal is quarantined to the premise for a retest 30–60 days after arrival, at owner’s expense. This applies to all equids that are negative on the CF test at a USDA import station and are imported from countries known to be infected with equine piroplasmosis. Horses that test negative, after being positive, are quarantined until undergoing three consecutive negative CF tests at 30-day intervals.

5. Summary

It is vital that veterinary practitioners recognize the importance of their role in early detection of exotic or unusual diseases affecting livestock. I was part of the first group of U.S. veterinarians detailed to the United Kingdom in 2001 to assist with the foot and mouth disease outbreak. I witnessed just how easily a contagious disease can be spread through marketing channels and how devastating it can be to the livestock industry of an entire nation. Even more impressive was the realization that a non-zoonotic disease involving food animals can also impact businesses seemingly unrelated to agriculture. Tourism, probably the number one industry in the United Kingdom, suffered terrible economic losses during the foot and mouth disease outbreak. Tourism is important to the U.S. economy. Our livestock are marketed on a much larger scale and subjected to less movement restrictions compared with those in the United Kingdom. We must never lose sight of our part in providing a strong national defense.

References