SMGroup

SM Journal of Public Health and Epidemiology

Review Article

Anthrax and Its Public Health Importance

Ahmed Mohammed*

School of Veterinary Medicine, Jimma University, Jimma, Ethiopia

Article Information

Received date: Feb 06, 2018
Accepted date: Feb 23, 2018
Published date: Feb 28, 2018

***Corresponding author**

Ahmed Mohammed, School of Veterinary Medicine, Jimma University, P.O.Box: 307, Jimma, Ethiopia, Tel: +251919106928; Email: ahmed.mohammedx@gmail.com

Keywords Animal; Anthrax; Bacillus Anthracis; Human; Outbreak

Abstract

Anthrax is an infectious disease caused by the bacteria *Bacillus anthracis*. The disease can affect both humans and animals, although it is more common among livestock and wild animals. The objective of this seminar is to review on anthrax and its public health importance. The disease occurs in herbivorous animals either through inhalation or the spores during grazing. Carnivorous animals are infected by consuming the affected herbivorous animals, whereas, infection in humans usually occurs through contact with the spores either through ingestion, inhalation or direct contact. The disease does not spread from infected persons directly and spores are the source of infection; since the spores of *Bacillusanthracis* are extremely resistant to natural condition and can survive for several decades in the environment. The spore enters in to the body and causes serious outbreak in tropical and sub-tropical countries with high rainfall. The clinical sign of the disease are characterized by sudden death, fever, staggering and in human it is pulmonary, cutaneous and gastrointestinal forms. The disease is diagnosed by giemsa or polychrome methylene blue and culture. It is evident that control of the infected animals, prevention of contact with the infected animals and contaminated animal products are quite important to disease control. It is recommended that if an animal anthrax case is confirmed, the affected property is quarantined, potentially exposed stock vaccinated; dead animals buried and contaminated sites disinfected. *Bacillus anthracis* are susceptible to different antibiotics like penicillin, chloramphenicol, streptomycin, tetracycline and erythromycin.

Introduction

Anthrax (charbon, splenic fever, Milzbra) occurs most commonly as a rapidly fatal septicemia in animals [1]. Anthrax is soil born diseases (not contagious) caused by gram-positive, rod-shaped bacteria. *Bacillus anthracis* can be found naturally in soil and commonly affects domestic and wild animals around the world. It occurs in animals (cattle, sheep, goat, horse), which are susceptible to the organism. Pigs, dog and cats relatively resistant and birds are highly resistant. Humans usually become infected when they come into contact with infected animals or their products [2].

Anthrax is a zoonotic infection, the causative agent of which is *Bacillus anthracis*. Ingestion or handling of the infected animal meat or other products can transmit the disease. *Bacillusanthracis* is non-motile, aerobic bacillus that produces central oval-shaped spores. It is characterized by rough, irregular and often comma-shaped colonies in blood agar medium. The mature spores can persist for years in dry soil but are destroyed by boiling in water for ten minutes. Grazing animals often swallow these spores which develop into the encapsulated vegetative mature bacilli in the circulation. *Bacillusanthracis* is an extra cellular pathogen that can avoid phagocytosis [3].

Anthrax is primarily an occupational hazard for handlers of processed hides and skin, bone products, wool and infected wildlife. It can also be contracted by contact with infected meat [4]. Anthrax has been an important cause of total human illness in most part of the world, which has higher case rates in Africa, Middle East and central and southern Asia. Whereas, in developed countries it is no longer a significant cause of human or livestock wastages because of appropriate control measures [5]. Anthrax is not a major issue of health in developed countries as only a few incidences are reported from such countries. However, for developing countries whose economy is mainly agriculture dependent, cutaneous anthrax is still a major concern of health [6]. An outbreak of inhalation anthrax occurred in Sverdlovsk near a Soviet military microbiology facility. This epidemic represented the largest documented outbreak of human inhalation anthrax in history. In October and November 2001, 22 cases of confirmed or suspected inhalation and cutaneous anthrax were reported associated with the intentional release of the organism in the United States. An additional case of cutaneous disease occurred in March of 2002 [7].

The anthrax status of a given country may be classified into one of the following categories: Epidemic, Endemic, Sporadic and Pandemic. The countries with epidemic status are more frequent in Africa. The number of cases of anthrax in animals has decreased significantly during the latter half of the twentieth century due to enhanced control and prevention programs which include efficacious vaccination.

Therefore, the objective of this paper is:

- To review on anthrax and its public health importance.

Literature Review

History

The disease "Anthrax" derives its name from the Greek word forcoal, due to the coal-like black lesions found on the skin in cutaneous anthrax [8]. Casimir Davaine and Pierre Rayer first observed rod-like organisms present in the blood of anthrax infected animals and humans. Davaine showed that those rods were most likely the cause of anthrax since unexposed sheep did not develop the disease [9]. *Bacillus anthracis*, the causative agent of anthrax was discovered by the famous Germany microbiologist and physician Robert Koch, known best for his postulates which are still used to prove microbial theory and disease etiology today. Koch's theories and early work with anthrax later led to discovery of germ theory. Koch isolated and identified *Bacillus anthracis* as the bacterial cause of anthrax. He developed a method for culturing pure *Bacillus anthracis*. This method allowed him to be the first to elucidate the complete life cycle of anthrax from spore to vegetative bacterium and back to spore again. Louis Pasteur created the first major vaccine against anthrax in livestock in 1881 [10].

Etiology

Anthrax is a disease of microbial origin caused by the bacterium *Bacillus anthracis*, a Gram-positive, endospores forming rod. It is an aerobic, non-motile that forms centrally located spore. *Bacillus anthracis* belong to the family Bacillaceae [11]. The first part of bacteria to intact with the host, when it is in its spore form, is the exosporium. It is made mostly of protein, with lipid and carbohydrate component. The vegetative *Bacillus anthracis* cells contain an extensive peptidoglycan, s-layer protein [12].

Biological characteristics

Bacillusanthracis is an aerobic, gram-positive, non-motile rod [13]. The bacterium measures 1-1.5 µm by 3-10 µm. Spore formation occurs centrally or paracentrally and causes no bacterial swelling [14]. Spore formation occurs when nutrients are depleted as happens after host death and exposure to air [15]. *Bacillus anthracis* spores are highly resistant to various environmental changes and can survive indefinitely in soil, air, water and vegetation despite extreme heat or cold, dessication, chemical treatment or ultraviolet exposure. The highly resistant nature of the spore aids in the persistence of the disease in an area [16].

The bacteria grow readily on all conventional microbiology media at 37°C including sheep blood agar and produce non-hemolytic, flat, dry and greyish colonies. Colony appearance on agar is typically white colonies with a characteristic comma shape or tail often referred to as curly-hair colonies [8]. It occurs alone or pairs in tissue and forms long manacles in culture give a typical boxcarform and Smears made from the culture growth showed Gram-positive rods in long chains. On tryptose soy broth, the colonies resembled interwoven threads (cotton) on the bottom with no surface growth [17].

Epidemiology

Geographical distribution: Anthrax is a zoonotic disease, which occurs worldwide distribution and severe disease, which is endemic in some countries and in defined regions of other countries (Sub Saharan Africa). Bacillus species are widely distributed in the environment mainly because they produce highly resistant endospores. In soil, endospores of *Bacillusanthracis* can survive for more than 50 years. Some Bacillus species can tolerate extremely adverse conditions such as desiccation and high temperatures [18].

Host range: Anthrax is a severe disease which affects virtually all mammalian species including human. Ruminants are extremely susceptible, often developing a rapid fatal septicaemic form of the disease. Horses are moderately susceptible to infection, while carnivores and pigs are comparatively resistant. Birds are almost totally resistant to infection, a characteristic attributed to their relatively high body temperatures [13].

Transmission: Anthrax cannot directly transmit from human-to-human contact. Rather, it is transmitted by the transfer of spores from infected humans or animals to potential hosts or by exposure to airborne anthrax spore [19]. That means it requires direct contact, inhalation, or ingestion of anthrax cells or spores to acquire an infection. Unfortunately contact with anthrax bacteria cells or endospores can happen in a wide variety of ways: touching or wearing infected clothing; contact with contaminated animal skins, hairs, hides, or bone products; being exposed to spore reserves in soil; eating or touching infected wool or meat and inhaling airborne particles. Contact with contaminated animal products or wool is the most common source of anthrax infections. In fact, anthrax was once so common among workers who sorted sheep wool [20]. Spread of the organism with in an area may be accomplished by streams insects, dogs, other carnivores, wild birds and by fecal contamination from infected animals. Introduction of infection into a new area is usually through contaminated animal products such as bone meal, fertilizers and hides or by contaminated concentrates or forage [21].

Risk factors: Host factor: The host factor of the disease occurs in all but is most common in cattle and sheep and less frequently in goats and horses. Humans occupy an intermediate position between this group and the relatively resistant pigs, dogs and cats. In farm animal the disease is almost invariable fatal, except in pig and even in this species the fatality rate is high [22].

Agent factor: The Virulence Factors (VF) of the virulent strains stem from the section of Lethal Factor (LF) and Edema Factor (EF) Toxins, along with a spore forming unit are known as the Protective Factor (PF). The toxin and capsule are the primary VF of the *Bacillus anthracis*. The bacterium is consisting of three protein component; component one, component two and component three (I, II and III). Component one is the EF, component two is the PF and component three is the LF. Each component is another movable protein. EF and LF gain entry in to the target cells by competitively binding with PF that has a membrane translocation function [23]. These three components act synergistically to produce the toxic effect seen in *Bacillus anthracis*. Component one and two cause with low mortality, but when component three is included there is a maximum lethality. Only encapsulated, toxigenic strains are virulent [24].

The spores are resistant to most external influences including the salting of hides, normal environmental temperature and standard disinfectant. Anthrax bacilli have remained viable in soil stored for 60 years in a rubber, stopper bottle and field observation indicate a similar duration of viability in exposed soil, particularly in the presence of organic matter, in an un drained alkaline soil and in a warm climate. Acid soils reduce the survival of *Bacillus anthracis* [25].

Environmental factor: In tropical and sub-tropical climate with high annual rain falls, the infection persists in the soil, so that frequent, serious outbreak of anthrax is commonly encountered. In some Africa countries the disease occurs every summer and reaches a devastating occurrence rate in years with heavy rain fall [26]. For example, heavy rain after a prolonged drought, or dry summer months after prolonged rain and always in warm weather [27].

Pathogenesis

When taken up the spore of *Bacillus anthracis* by pulmonary route, they need a lesion through which they enter into the body. Following entry, the spores may have commenced germination are multiply and initially during the incubation period, they carried to the lymphatic where they multiply. The bacteria are filtered out by the spleen and other part of the reticulo-endothelial system. However, the system finally breaks down due to toxin action and during the last few hours of life. Action of a toxin breaks the endothelial cell lining of the blood vessels, resulting in internal bleeding [28]. The Anthrax toxin believed to play roles in two stages of infection. Early during infection, they target the immune response to allow survival in the host and to facilitate dissemination. In systemic disease they target tissues and induce lethality. Up on ingestion of the spores, infection may occur through intact mucus membrane, through defects in the epithelium around erupting teeth, or through scratches from tough and fibrous food materials. The organism is resistant to phagocytosis, in part due to the presence of the Body-D-Glutamic Acid Capsule (DGAC) and proliferates in regional draining lymph nodes subsequently passing via the lymphatic vessels in to the blood stream; septicemia, with massive invasion of all body tissues, follows [29]. *Bacillus anthracis* produce a lethal toxin that causes edema and tissue damage, death resulting from shock and acute renal failure a4nd terminal anoxia [17]. In pigs, localization occurs in the lymph nodes of throat after invasion through the upper part of the digestive tract, Local lesions usually lead to a fatal septicemia [21].

Clinical signs

Clinicalsigns in animal: *Bacillus anthracis* are an obligate pathogen, having incubation period of 3-7 days or ranging from 1-14 days. In herbivores, the clinical course ranges from per acute to chronic [30]. The per acute form is characterized by sudden and rapid onset, staggering, dyspnea, trembling, collapse and a few convulsive movements may occur in cattle, sheep, or goat without any previous evidence of illness [31].

In the case of an acute form, there is an abrupt rise in body temperature and period of excitement followed by depression, respiratory or cardiac distress, staggering, convulsion and death. The body temperature may rise 41.5°C, animals may abort and rumination ceases. Blood discharges from natural body orifice, usually lasts about 48 hours. In pigs and horses there are fever, anorexia listlessness with edema of throat, face, neck and abdomen with Petechial Hemorrhage (PH) on the skin. Dysentery may be present with bloody froth at the nostrils. The chronic form is characterized by localized subcutaneous edematous swelling, most frequently at the area of the ventral neck, shoulders and thorax [2].

Clinical signs in human: Anthrax can manifest in a variety of different ways, depending on how it enters the body. There are three main varieties of anthrax: Cutaneous Anthrax (caused by skin exposure, usually through cuts); Pulmonary Anthrax (caused by inhaling anthrax; this form affects the lungs and then attacks lymph nodes in the chest); and Gastrointestinal Anthrax (caused by ingesting the bacteria through contaminated meat). Of the three types, pulmonary anthrax is the most frequently fatal if not treated. The fourth and least common form of anthrax, pharyngeal anthrax affects the throat and base of the tongue [6].

Cutaneous anthrax manifests with dark ulcers or lesions on the skin that typically develop within a week of exposure. Pulmonary anthrax symptoms set in faster, and can affect patients as soon as 1-7 days or up to a month after exposures. Symptoms of pulmonary anthrax include trouble breathing, nausea, fever, vomiting, etc. Without treatment the symptoms become worse and patient can go into shock, suffer toxemia, endure the collapse of lungs, or even die. Gastrointestinal anthrax usually manifests with stomach pain, fever, and loss of appetite, with symptoms increasing in severity if a patient is not treated with antibiotics. The cutaneous form of anthrax accounts for over 95% of anthrax cases [31].

Lesions usually occur on exposed skin and often commence with itchiness. They pass through several stage, popular stage, vesicular stage with a blister that often becomes hemorrhagic, eschar stage that appears two to six days after the hemorrhagic vesicle dries to become a depressed black scab (malignant pustule) which may have surrounding redness and extensive edema (swelling). Anthrax lesions are usually painless but pain may result due to surrounding edema. Untreated lesions can progress to involve regional lymph nodes. An overwhelming septicemia can occur in severe cases. Untreated cutaneous anthrax has a case fatality rate of 5-20 % but death is rare with early appropriate treatment [32].

Early symptoms may be confused with a flu-like illness. This is followed within three to six days by rapid onset of hypoxia, dyspnea and high temperature, with radiological evidence of mediastinal widening. Meningitis frequently occurs. The mortality rate approaches 100% with delayed or no treatment. Commencement of appropriate antibiotics during the prodromal significantly decreases the mortality rate [23].

The Intestinal/oro-pharyngeal form of anthrax are very rare forms of anthrax in developed countries but may occur in large outbreaks in developing countries following ingestion of meat from infected animals. In intestinal anthrax, gastro-intestinal symptoms may be followed by fever, septicemia and death. Case fatality rates of 25-75 % have been reported. In pharyngeal anthrax fever, neck swelling due to lymphadenopathy, throat pain, oral ulcers and dysphasia may be followed by severe local ulcers and swelling, septicemia and death. Case fatality rates are similar to the intestinal form [30].

Post mortem lesions

Rigor mortis is usually absent or incomplete, and the carcass is typically bloated and decomposes rapidly. Dark, tarry blood may ooze from the body orifices; some sources suggest this is not a common sign. Edema may be noted, particularly around the throat and neck, in horses. Necropsies should be avoided, to prevent contamination of the surrounding area with spores [33]. If a ruminant carcass is opened, signs of septicemia will be evident. The blood is dark, thick and does not clot readily. Edematous, blood-tinged effusions may be

seen in subcutaneous tissues, between skeletal muscles and under the serosa of organs. Petechiae and ecchymosed are often noted in the lymph nodes, serosae surfaces of the abdomen, thorax, epicardium and endocardium [34].

Hemorrhages and ulcers are also common in the intestinal mucosa; ulcers occur most often over Peyer's Patches (PP), but can also be found in other locations. Peritonitis and excessive peritoneal fluid may be noted. The spleen is usually enlarged and has a 'blackberry jam' consistency. The lymph nodes, liver and kidneys may be swollen and congested. Meningitis can also occur. Similar internal lesions can be seen in some horses; in others, the lesions may be limited to edema of the neck and throat [35].

Septicemia lesions may also be found in omnivores and carnivores, but are less common than edema and inflammation of the pharyngeal area, or gastrointestinal lesions. Pigs with chronic anthrax usually have lesions only in the pharyngeal area. The tonsils and cervical lymph nodes are typically enlarged and have a mottled salmon to brick-red color on cut surface. The tonsils may be covered by diphtheritic membranes or ulcers and pharynx is usually edematous [36]. A chronic intestinal form, with inflammation and lesions in the mesenteric lymph nodes, is also reported in pigs. Severe gastrointestinal inflammation, sometimes accompanied by hemorrhage, Peritonitis and necrosis, has been reported in some omnivores and carnivores [37].

Diagnosis

To confirm the diagnosis on an unopened carcass, peripheral blood or Local Edema Fluid (LEF) should collect by needle puncture. Since the blood clots poorly, jugular vein puncture may permit sample collection [31]. Blood can also be carefully collected from an ear vein to avoid unnecessary contamination and sporulation. The smears should be prepared and interpreted by an experienced and qualified microbiologist. Note that, zoonotic potential of this organism is high when handling carcass and submitting specimen [21].

In Direct Microscopy, *Bacillusanthracis* produce a capsule in vivo and either Giemsa or Polychrome Methylene Blue (PMB) stains are used to demonstrate the capsule which is diagnostic importance. The capsule material is more abundant if the blood smear has been taken from a recently died animals. PMB stained smears reveals square ended, blue rods in short chains surrounded by pink capsular materials. In case of Giemsa Stained Smears (GSS), the capsule is reddish [38].

When virulent strains are cultured and grown in media containing serum or bicarbonate or both, they produce capsules and the colonies appear in 24 hours. They look flat, gray; are usually non-hemolytic and smooth to mucoid. In the absence of serum or bicarbonate, bacteria fail to produce capsules and the colonies are rough [39]. Identification of *Bacillus anthracis* in colonial morphology to diagnose in a hospital microbiology laboratory is based on direct GSS of the skin lesion, blood, or Cerebrospinal Fluid (CSF). Demonstrating encapsulated, large Gram positive bacilli in short chains after incubation for 18-24 hours. Growth occurs on blood agar and shows the characteristic morphology of grey/white, flat colonies, 2-5 mm in diameter, with irregular edges. Blood cultures are usually positive within six to 24 hours [40].

Bacillus anthracis are much more pathogenic for guinea pigs and mice than other bacillus species, causing death within 24 hours. Large encapsulated rods are demonstrated in smears of spleen and blood of infected animals [23]. *Bacillus anthracis* and the majority of bacillus species do not normally produce capsules in laboratory media and the colonies have a dry appearance. However, *Bacillus anthracis* can be induced to produce a capsule by growing it on nutrient agar containing 0.7% sodium bicarbonate under 10% CO_2. The colonies are quite mucoid [38].

Differential diagnosis

For differential diagnosis, other causes of sudden death should be considered. Among these are African horse sickness, botulism, blackleg (*Clostridium chauvoei*), per acutebabesiosis, chemical poisoning (heavy metals, other poisons), ingestion of toxic plants, snake bite, lightning strike or metabolic disorders such as lactic acidosis, magnesium deficiency and bloat [41]. In Black leg; there is edema of skeletal and cardiac muscle. The ulcerative eschar of cutaneous anthrax must be differentiated from lesions that present regional lymphadenopathy. If the lesion is purulent and the regional lymph nodes are palpable, staphylococcal lymphadenitis is the most likely cause, although cutaneous anthrax lesions can be infected [42].

Public health importance

Anthrax primarily affects herbivores animals. Humans usually become infected when they come into contact with infected animals or their products. Anthrax is primarily an occupational hazard among veterinarians, agricultural workers and workers who process hides and skin, wool and bone products and infected wildlife. It can also be contracted by contact with infected meat, for example in abattoir workers. New areas of infection in livestock may develop through introducing animal feed containing bone meal. Cutaneous outbreaks sometimes occur in tannery workers and those handling pet meat [43].

Anthrax can also be used as a bio-warfare or bio-terrorism agent, most likely spread as an aerosol therefore; any new case can be assessed with this possibility in mind, particularly but not exclusively in cases of pulmonary anthrax [44]. Anthrax is still a significant risk in some countries and outbreaks occasionally occur in humans [45]. In Africa, estimates suggest that each cow with anthrax can result in up to ten human cases. However, the incidence of anthrax has declined sharply in developed nations [31].

The cutaneous form accounts for at least 90-95 % of natural anthrax infections. The gastrointestinal form seems to be uncommon, but can occur in outbreaks associated with contaminated meat. Natural cases of inhalational anthrax are rare; however, aerosolized biological weapons would be expected to produce a high percentage of this form [43].

The effects of anthrax disease are a reduction of the efficiency which input or resources are converted into output or a product that means they reduced productivity. In most developing countries vaccination of susceptible animal in enzootic areas has reduced the prevalence of the disease to negligible proportions on national bases, but heavily losses may still occur in individual herds. Loss occurs due to mortality but also from withholding of milk in infected dairy herds and for a period following vaccination it causes a great problem by death of

animals, reducing animal products and complete condemnation of carcasses and by products as well as closure of abattoirs [43].

The mortality rate for anthrax varies with the species. Clinical infections in ruminants and horses are usually fatal; pigs often recover. In carnivores, mortality is also relatively low. However, this rate is not widely available for wild animals [31].

Sporadically, mainly as an occupational hazard among veterinarians, agricultural workers and workers who process hides, hair, wool and bone products. The cutaneous form accounts for at least 90-95% of natural anthrax infections. The gastrointestinal form seems to be uncommon, but can occur in outbreaks associated with contaminated meat. Natural cases of inhalational anthrax are rare; however, aerosolized biological weapons would be expected to produce a high percentage of this form [43].

In 2001, 11 cases of inhalational anthrax and 11 cases of cutaneous anthrax were associated with a bioterrorist attack via anthrax-contaminated mail. The mortality rate varies with the form of the disease. Cutaneous anthrax is thought to be fatal in 5-20% of untreated cases and less than 1% of patients treated with antibiotics. In contrast, the mortality rate is high for inhalational anthrax, even when treated appropriately. Earlier estimates suggested that the case-fatality rate for this form approached 90-100 % but newer, more intensive treatment regiments may decrease the mortality rate [46].

Treatment

Bacillus anthracis are susceptible to penicillin, chloramphenicol, streptomycin, tetracycline and erythromycin. Treatment should continue for at list five days. However, in acute anthrax, antimicrobial treatment is often useless [41]. Treatment initiated 24 hours after infection with any of for antibiotics protected the animals during treatment, but many of the animals died of anthrax after treatment was stopped. The antibiotics conferring degrees of protection ranging from 10-90 percent. Combining antibiotic treatment with a protective antigen vaccine left all animals fully protected even after the end of treatment. Animals whose treatment was delayed beyond 24 hours post-infection developed varying degrees of bacteremia and toxemia [47].

If a human is known to be exposed to anthrax, they should be promptly treated with prophylactic antibiotics. This is especially crucial in case of pulmonary (inhaled) anthrax, which is often fatal if not treated [48]. However, the disease must be treated in early stages before levels of the toxin grow in a host's body. Antibiotics only kill anthrax bacteria cells, and are not effective against toxins. Since early anthrax symptoms resemble a variety of less virulent infections, many victims may not even realize they need to be treated until the onset of severe symptoms, triggered by toxins. By the time anthrax is diagnosed, serotoxin levels may have already killed the host [49].

Ananthrax vaccine was developed in the mid 1970's, mainly for use on military service people and workers who come into frequent contact with livestock or animal products including hides and wool. However due to side effects and relatively inconvenient vaccine schedule (multiple doses are required for immunity) the vaccine has not been used or recommended to the general public. Microbiologists and medical professionals continue to work on refining the anthrax vaccine and therapies to treat anthrax infections including antitoxin therapies [50].

Control and prevention

When an outbreak or a case of anthrax occurs, animal health authorities must be notified to supervise control measures; for carcass disposal which involves incineration or deep burial; to treat and isolate sick animals; vaccinate susceptible stocks and quarantine the premises for 3 weeks subsequent to the last established case. Milk from infected animals must be discarded under appropriate precaution. Disinfecting burns and fences with 10% sodium hydroxide is mandatory. Boiling utensils for 30 minutes will kill spores and surface soils spores can be killed by treating with 3% per acetic acid solution at the rate of eight litters per square meter [41].

Vaccination has great value in the control of disease. The vaccine is protective but sometimes produces severe reactions. Recent vaccine which has proved to be useful is derived from a virulent encapsulated strain of *Bacillus anthracis*. The vaccine gives protection for one year. The vaccines prepared from none living antigens do not give adequate immunity. A new wave of research on vaccines against the capsule has also introduced new candidates for development [29].

The control of meat and milk producing animals in infected heard in such a way as to avoid any risk to the human population is a special aspect of the control of anthrax. It is necessarily to avoid unnecessary wastes when an outbreak occurs, the placing of the farm in quarantine, the distraction of discharge and cadavers and the vaccination of survivors are parts of the animal disease control program and indirectly reduce human exposure [51].

Prohibition of movement of milk and meat from the farm during quarantine period should prevent entry of the infection in to the human food chain. Discontinuation of infection source is an essential first step to breaking the cycle of infection. The most excellent method to stop anthrax is to keep away from or reduce contact with sources of contagion. This means exercising caution when handling animals or animal products and carcasses should be carefully destroyed from any animal suspected to suffer anthrax. Neat animal management practices and policies regarding vaccinating livestock have dropped the numbers of anthrax cases. Moving other animals away from the affected area is an important early action. If flies are suspected of being important vectors, therefore, fly control should be considered. Prevention of *Bacillus anthracis* exposure through animal products imported from areas requires disinfection of such material as hair and wool by formaldehyde. Bio endemic meals sterilized by dry heat (150°C per 3 hrs.) or steam at 115°C for 15 minutes is very important [52].

Conclusion and Recommendations

Anthrax is an infectious disease caused by *Bacillusanthracis* and it affects both animals and humans. Disease causes a great problem by death of animals, reducing animal products and complete condemnation of carcasses and byproducts as well as closure of abattoirs. It also results in pulmonary, intestinal or cutaneous forms of anthrax in humans. The control of meat and milk producing animals in infected heard to avoid any risk to the human population, When an outbreak occurs, the placing of the farm in quarantine, the distraction of discharge, cadavers and the vaccination of healthy animals are a special aspect of the control of anthrax [53]. Observations of the role of climatic factors such as season-of-year, ambient temperature, and drought in promoting anthrax epizootics have been made. There also

have been attempts to support these observations via quantitative methodology. The commonality of summer months, high ambient temperatures, drought and anthrax are so related. The roles of environmental factors such as soil types and soil disturbances via excavation are defined despite attempts to evaluate these potential factors.

Based on the above conclusion, the following recommendations are forwarded:

- Don't open the carcass died of anthrax.
- Eating of raw meat should be avoided.
- Rapid diagnosis and treatment of sick animals with effective antibiotics,
- Prevention of contact health animals with infected animals and contaminated animal products,
- Preventing animals from grazing on contaminated pasture or land.
- Environmental and personal hygiene,
- Annual vaccination in enzootic areas,
- Health education should be given on precaution for farmers, butchers, and tanners.
- Burning or burring of suspect and confirmed cases.

References

1. Conger TH. Anthrax epizootic. Texas, Animal Health Association: Hershey, Pennsylvania. Virginia: USA Animal Health Assoc. 2001; 207.
2. Fulako T. Immune system paralysis by anthrax lethal toxin. The role of innate and adaptive immunity. J, Infect. Dis. 2004; 4: 166-170.
3. Ezzel JW, Welkos SL. The capsule of Bacillus anthracis, J. App Microb. 2003; 87: 250.
4. Wattiau P, Klee SR, Fretin D, Van Hessche M, Ménart M, Franz T, et al. Occurrence and genetic diversity of Bacillus anthracis strains isolated in an active wool-cleaning factory. Appl Environ Microbiol. 2008; 74: 4005-4011.
5. Finegold SM, Boron EJ, Peterson RK. Diagnostic Microbiology. 9th ed, Bailey and Scot's. 2002; 451-454.
6. Rao TN, Venkatachalam K, Ahmed K, Padmaja IJ, Bharthi M, Rao PA. A mini-outbreak of cutaneous anthrax in Vizianagaram District, Andhra Pradesh, India. Indian J Dermatol Venereol Leprol. 2009; 75: 416-418.
7. Dai Z, Kabiret T, Yarbray K. Regulation of the Bacillus anthracis protective antigen and a trans-acting element activate transcription from one of two promoters. J. Bacteriol. 2003; 176: 586-595.
8. Inglesby TV, Henderson DA, Bartlett JG, Ascher MS, Eitzen E, Friedlander AM, et al. Anthrax as a biological weapon: Medical and public health management. Working Group on Civilian Biodefense. JAMA. 1999; 281: 1735-1745.
9. Compton J. In Military Chemical and Biological Agents: Chemical and toxicologic properties. 2005; 354-361.
10. Biddle W. In A field guide to germs. 1995; 20-22.
11. Shafazand S, Doyle R, Ruoss S, Weinacker A, Raffin TA. Inhalational anthrax: epidemiology, diagnosis, and management. Chest. 1999; 116: 1369-1376.
12. Mock M, Fouet A. Anthrax. Ann Rev Microbiol. 2001; 55: 647-671.
13. Watson A, Keir D. Information on which to base assessments of risk from environments contaminated with anthrax spores. Epidemiol Infect. 1994; 113: 479-490.
14. Dixon T, Meselson M, Guillemin, Hanna P. Anthrax Medical progress, Review art. New Eng J.Med. 1999; 341: 815-826.
15. Bartlett J, Ingles T, Borio L. Management of anthrax. Clin. Infect. Dis. 2002; 35: 851-857.
16. Dragon DC, Rennie RP. The ecology of anthrax spores; tough but not invincible, J, Canadian Veter. 1995; 36: 295-301.
17. Radostits OM, Arundel JH. Diseases caused by Bacillus species. In: Veterinary Medicine: A Textbook of the Diseases of Cattle, Sheep, Pigs, Goats and Horses. 9th Edn. Saunders WB Company Ltd, Philadelphia. 2000: 747-751.
18. Slonczewski JL, Foster JW. Microbiology. 2nd Edn. An Evolv Science. 2010; 1-22.
19. CDC: Center for Disease Control and Prevention. Fact Sheet: Anthrax Information for Health Care Providers. 2002.
20. Daniel R, Bailey G, Dunkin S. Diagnostic Microbiology. 9th ed, Asso. Content, the Hist Anth. 2008; 451-454.
21. Constable PD, Radostits OM, Gay CC, Hinchchiff KW. Veterinary Medic. A textbook of the disease of cattle, horse, sheep, pigs and goats.10th ed. Saunders. 2007; 815-819.
22. Read TD, Peterson SN, Tourasse N, Baillie LW, Paulsen IT, Nelson KE, et al. The genome sequence of Bacillus anthracis Ames and comparison to closely related bacteria. Nature. 2003; 423: 81-86.
23. Paccani SR, Tonell OF, Patrussi L, Capitani N, Simonato M, Montecco C, et al. Anthrax toxins inhibit immune cell chemotoxis by perturbing chemo receptor signaling. Cell Microbiol. 2007; 9: 924-926.
24. Carter GR, Wise DJ. Essentials of veterinary bacteriology and mycology, 6th ed, Iowa State Press, Ames, IA, USA. 2004; 179-182.
25. Lee K, Costerton JW, Ravel J, Auerbach RK, Wagner DM, Keim P, et al. Phenotypic and functional characterization of Bacillus anthracis biofilms. Microbiology. 2007; 153: 1693-1701.
26. Gracy CC, Radostits OM, Blood DC. A text book of Veterinary Medicine, 8th ed. Bailler Tindall. 2004; 673.
27. VasNess GB. Ecology of anthrax. Science. 1971; 172: 1303-1307.
28. Guillemin J, Dixon T, Meselson M, Hanna P. Anthrax. New England J. Med. 2000; 341: 815-826.
29. Moayeri M, Leppla SH, Vrentas C, Pomerantsev AP, Liu S. Anthrax Pathogenesis. Annu Rev Microbiol. 2015; 69: 185-208.
30. Hungerford TG. Disease of livestock. 9th .ed. McGraw-Hill, Sidney. 1990; 329-332.
31. Huey RJ, Gracey MJ, Collins DC. Meat hygiene. 10th ed. 2000; 507-509.
32. Nijm H, Hugh M. Global anthrax report. Microbiol. 2001; 87: 189-191.
33. CDC: Center for Disease Control and Prevention. Anthrax. 2006.
34. Bischof TS, Hahn BL, Sohnle PG. Characteristics of spore germination in a mouse model of cutaneous anthrax. J. Infect. 2007; 195: 888-894.
35. Binkley CE, Cinti SA, Samon DM, Colletti LM. Bacillus anthracisas an agent of bioterrorism: a review emphasizing surgical treatment. 2000; 236: 9-16.
36. Carter ME, Quinn PJ, Markey BL. Clinical veterinary microbiology, 4th ed, Mosby London. 1994; 178-183.
37. NAHIS. The National Animal Health Information System. Anthrax. 2001.
38. Quinn PJ, Markey BK, Leonard FC, FitzPatrick ES, Fanning S, Hartigan PJ. Veterinary Microbiology and Microbial Diseases. Black well science, Dublin. 2003; 80-83.
39. Pipkin J, Dublin AB, Smith BP. Black well science, Anthrax. 2002; 80-83.
40. Spencer RC. Bacillus anthracis. J Clin Pathol. 2003; 56: 182-187.

41. Fouet A, Mesnage S, Tosi-Couture E, Gounon P, Mock M. Bacillus anthracissurface: capsule and S-layer. J Appl.Microbiol. 1999; 87: 251-255.

42. Hirsh DC, Zee YC. Veterinary microbiology. Black well science USA. 2003; 246-249.

43. Turnbull PC, Hugh-Jones ME, Cosivi O. World Health Organization activities on anthrax surveillance and control. J Appl Microbiol. 1999; 87: 318-320.

44. Ibrahim K, Brown G, Wright D, Rotschafer J. Bacillus anthracis: Medical issues of biologic warfare. Pharmaco therapy. 1999; 19: 690-701.

45. Bell DM, Kozarsky PE, Stephens DS. Clinical issues in the prophylaxis, diagnosis, and treatment of anthrax. EmergInfecDise. 2002; 8: 222-225.

46. Takahashi H, Keim P, Kaufmann AF, Keys C, Smith KL, Taniguchi K, et al. Bacillus anthracis bioterrorism incident, Kameido, Tokyo, 1993. Emerg Infect Dis. 2004; 10: 117-120.

47. Levy H, Weiss S, Altboum Z, Schlomovitz J, Rothschild N, Blachinsky E, et al. Lethal factor is not required for Bacillus anthracis virulence in guinea pigs and rabbits. Microb Pathog. 2011; 51: 345-351.

48. Rosovitz MJ, Leppla SH. Virus deals anthrax a killer blow. Nature. 2002; 418: 825-826.

49. Chvyrkova M, Irina W, Xuejan S, Zhang F, Simon H, Terzyan J. Lethal Factor of Anthrax Toxin Binds Monomer Form of Protective Antigen National Instiute of Health. 2007.

50. Anon. Anthrax, Bacteriology Department of Medicine, Clinical Presentations, and Management, Georgia State. 2011.

51. Adlakha SC, Sharma S N. Text book of VeteMicrobio. 2008; 138-141.

52. Lacy DB, Collier RJ. Structure and function of anthrax toxin. Curr Top Microbiol Immunol. 2002; 271: 61-85.

53. Leendertz FH, Ellerbrok H, Boesch C, Couacy-Hymann E, Mätz-Rensing K, Hakenbeck R, et al. Anthrax kills wild chimpanzees in a tropical rainforest. Nature. 2004; 430: 451-452.