

A Review on Anthrax and its Public Health and Economic Importance

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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 with high economic significance. The disease occurs in herbivorous animals either through inhalation or by direct consumption of the spores during grazing in the wild. 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 *Bacillus anthracis* is extremely resistant to natural condition and can survive for several decades in the environment. The spore enters in to the body and causes a serious outbreaks in tropical and sub tropical countries with high rainfall. 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. The quarantine is not released until occurrences of anthrax cases have ceased and at least six weeks have elapsed since the last round of vaccinations on the property and people who are exposed might be given anthrax vaccine to prevent disease. *Bacillus anthracis* are susceptible to different antibiotics like penicillin, chloramphenicol, streptomycin, tetracycline and erythromycin

Key words: Animal • Anthrax • Bacillus Anthracis • Human • Outbreak

INTRODUCTION

Anthrax, a potentially fatal infection, is a virulent and highly contagious disease [1]. It is a serious infectious disease caused by gram-positive, rod-shaped bacteria known as *Bacillus anthracis*. Anthrax can be found naturally in soil and commonly affects domestic and wild animals around the world. It occurs in all food animals (cattle, sheep, goat, horse), which are susceptible to the organism. Pigs are more resistant than sheep and horse, where as 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. Anthrax is primarily an occupational hazard for handlers of processed hides, goat hair, bone products, wool and infected wildlife. It can also be contracted by contact with infected meat [2].

Descriptions of Anthrax begin in antiquity, with the best ancient account being by the Roman poet Virgil. During the 19th century, anthrax was the infection involved in several important medical developments. It served as the prototype for Koch's postulates regarding the causation of infectious disease [1] and in 1877, this organism was the first to be shown to cause disease by Robert Koch and verified by Louis pasture [3]. 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 [1].

In the 1900s, human inhalation anthrax occurred sporadically in the United States among textile and tanning workers, but the incidence of the illness had

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declined dramatically. An outbreak of inhalation anthrax occurred in Sverdlovsk near a Soviet military microbiology facility in 1979. 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 [3].

Characteristics of anthrax in Ethiopia include a known exposure to diseased animals, occurrence within families, frequent treatment by local healers and high morbidity and mortality. Twenty-seven patients with cutaneous anthrax were identified over a three-year period at Gondar College of Medical Sciences in North Central Ethiopia. Nine patients who delayed seeking medical care presented with severe symptoms and three patients died. Eighteen patients were clustered within four families in which an attack rate of 32% occurred. Ninety-three percent of patients could trace their disease to exposure to the products of a specific diseased animal [4].

Therefore, the objectives of this paper are to review anthrax and its public health importance and to highlight its economic significance.

Anthrax and its Public Health Importance

Etiology: Anthrax is a bacterial disease caused by the spore forming *Bacillus anthracis*, a gram positive, rod shaped bacterium. It is an aerobic, non motile that forms centrally located spore. *Bacillus anthracis* belongs to the family Bacillaceae [5]. The first part of bacteria to intact with the host, when it is in its spore forms, is the exosporium. It is made mostly of protein, with lipid and carbohydrate component [6]. While, the function of the exosporium is unknown it appears to have Pilli that seem to enhance spore attachment to surface area [7]. *B. anthracis* also has unique capsule which is considered to be majored contributor to its virulence. The capsule enhances the bacteria's ability to evade host defenses, as well as inducing septicemia. The s-layer is the layers of bacterium the covering of peptidoglycan [6]. The capsule has a negative charge which inhibits microphages from engulfing and destroying the *Bacillus anthracis* [8].

Anthraces belongs vegetative cells, impeding the host's immune response [9, 6]. The vegetative *Bacillus anthracis* cells are gram positive; therefore they contain an extensive peptidoglycan, s-layer protein. Cell wall polysaccharides functions in a choiring the protective s-layer to cell wall [10].



Fig. 1: Morphology of *Bacillus anthracis* [7]

Genome structure of *Bacillus anthracis* has a single chromosome [11]. The main virulent factor is encoded plasmid. The plasmid are circular, extra chromosomal, double stranded DNA molecule [12]. The toxin is a complex of three plasmid encoded protein. Two of the proteins are directly toxic, including LF (lethalfactor) and EF (edema factor). High LF levels destroy WBC and release the bacterium, while EF increases cyclic AMP (Adenosine mono phosphate) levels. Hosts are more susceptible to infection with EF. Energy and water balances impaired by increase in cyclic AMP, resulting in the accumulation of fluid in the cells [11].

Capsule formation is important because of it allows the organism to resist phagocytosis. The capsule is a polymer of amino acid (D-glutamate), unlike most other bacteria which have poly saccharide capsule. It serves as one of the principal virulence factor during anthrax infection. It is used to inhibit host defense through inhibition of phagocytosis of the vegetative cells by macrophage. In conjunction with lethal toxin and edema toxin, whose target cells include macrophages and neutrophills, respectively, the capsule allows virulent anthrax bacilli to grow in to the infected host [9]. The cells excrete the capsule for protection and virulence. The capsule and s-layer are compatible, but they can both be formed independently (without the presence of others). The characteristic mucoid or smooth colony variant is correlated with capsule production ability. Virulent strain all forms the capsule and rough colony capsule are a virulent. Growth in atmospheric carbon dioxide causes anti phagocyte capsule and anthrax toxin proteins to be synthesized [11].

Epidemiology

Occurrence: The disease probably originated in sub Saharan Africa and has speared to have a worldwide distribution, although the area prevalence varies with the

soil, climate and the efforts put in to spurring its occurrence *B.anthraces* live in soils worldwide. Therefore, when they are isolated from a certain environment, it does not necessarily imply that the specific environment in their habitat [11]. Accidentally ingestion of contaminated bone meal or pasture contaminated by tanner effluent is more common sources. In this circumstance out breaks are few and the number of animal affected is small. The development of an effective live stokes vaccine coupled with the use of penicillin and the implementation of quarantine regulation has caused a marked decline in the occurrence of anthrax in the most countries compared to its satirical incidence [12].

The ultimate reservoir of *B. anthraces* is contaminated soil, in which spores remain viable for long period. Herbivores the primary host become infected when foraging in a contaminated region. Because the organism does not depend on an animal reservoir, it can't readily be eradicated from a region and anthrax remains endemic in many countries [10].

Humans become infected almost exclusively through contact with infected animal or animal product. Human anthrax is traditionally classified as either nonindustrial or industrial anthrax depends on whether the disease in acquired directly from animal or indirectly during handling of a contaminated animal products. Nonindustrial anthrax usually affects a person who works with animal or animal carcasses, such as farmers, veterinarians and butchers and it's almost always coetaneous. Industrial anthrax, acquired from handling contaminated hair, hides, wool, bone meal or other animal products, has a higher chance of being pulmonary as a result of the inhalation of spore-laden dust [13].

Risk Factors

Host Factor: The host factor of the disease occurs in all vertebrates 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 cause fatality rate is high [12].

Agent Factor: The virulence factors of the virulent strains stems from the section of LF (lethal factor) and EF (edema factor). Toxins along with a spore forming unit known as the protective antigen. The toxin and capsule are the primary virulence factor of the bacillus anthraces. The bacillus anthraces is complex, consisting three protein

Table 1: Properties of *B.anthraxis* toxin components: [16]

| Toxin components | Edema production | Lethality | Immunogenicity |
|-----------------------|------------------|-----------|----------------|
| Edema factor(I) | - | - | - |
| Protective factor(II) | - | - | ++ |
| Lethal factor(III) | - | - | - |
| I and II | +++ | + | +++ |
| I and III | - | - | + |
| II and III | - | ++ | ++ |
| I,II and III | ++ | +++ | ++ |

Note: + represent degree of reaction, - represents no reaction

component; component one, component two, component three (I, II and III). Component one is the EF, component two is the PF (protective factor) 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 [14]. These three components act synergistically to produce the toxic effect seen in bacillus anthraces. Component one and two causes with low mortality, but when component three is included there is a maximum lethality. Only encapsulated, toxigenic strains are virulent [10]. Pathogen Factors of the disease when material containing anthrax bacilli is exposed to the air, spores are formed that protract that infectivity of the environment for very long periods, 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 anthraces* [15].

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 are commonly encountered. In some Africa countries the disease occurs every summer and reaches a devastating occurrence rate in years with heavy rain fall. Wild fauna it including hippos, Cape buffalo and elephants die in large numbers. It is probable that predators act as inert carriers of the infection [17]. In temperate, cool climate only sporadic out break drive from the soil borne infection. For example heavy rain after a prolonged drought, or dry summer months after prolonged rain and always in warm weather when the environmental temperature 15°C. The hypothesis that these climatic condition lead to sporulation and vegetative proliferation with the production of incubator areas for anthrax in the

soil appears improbable but spores have a high buoyant density and in wet soil could become concentrated and remain suspended in standing water with further concentration on the soil surface as the water evaporate. This relationship to climate has made it possible to predict anthrax life expectancy in the soil [18].

Transmission: Infections gain entrance to the body by ingestion, inhalation or through the skin. Spread of the organism within an area may be accomplished by streams, insects, dogs and other carnivores and 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, hides and wool or by contaminated concentrates or forage. Inhalation infection is thought to be of minor importance in animals, although the possibility of infection through contaminated dust must always be considered. Wool sorter's disease in humans is due to inhalation of anthrax spores by workers in the wool and hair industries but, even in these industries cutaneous anthrax is much more common [13].

Pathogenesis: When taken up by pulmonary route, *B.anthraxis* needs a lesion through which to enter the body, following entry the spores may have commenced germination and are carried to the lymphatic where they multiply and initially during the incubation period, 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 [7]. 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 [19].

Up on ingestion of the spores, infection may occur through the intact mucus membrane, through defects in the epithelium around erupting teeth, or through scratches from tough, fibrous food materials. The organism are resistant to phagocytosis, in part due to the presence of the body-D-glutamic acid capsule and proliferate 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. *Bacillus anthracis* produces a lethal toxin that causes edema and tissue damage, death resulting from shock and acute renal failure and terminal anoxia [11].

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 [13].

Clinical Finding

Clinical Finding in Animal: *Bacillus anthracis* is 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 [20].

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 [10]. Rigor mortis is absent or incomplete. Dark, tarry-like blood which does not clot is evident at mouth, anus, nostrils and vulva [21].

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 to 41.5°C, animals may abort and rumination ceases. Blood discharges from natural body orifice, usually lasts about 48 hours [20]. In pigs and horses there are fever, anorexia, listlessness with edema of throat, face, neck and abdomen with petechial hemorrhage on the skin. Dysentery may be present with bloody froth at the nostrils [21].

The chronic form is characterized by localized subcutaneous edematous swelling, most frequently at the area of the ventral neck, shoulders and thorax [1].

Clinical Finding in Human: The cutaneous form of anthrax accounts for over 95% of anthrax cases. Lesions usually occur on exposed skin and often commence with itchiness. They pass through several stage, papular stage, vesicular stage with a blister that often becomes haemorrhagic, eschar stage that appears two to six days after the haemorrhagic vesicle dries to become a depressed black scab (malignant pustule) which may have surrounding redness and extensive oedema (swelling). Anthrax lesions are usually painless but pain may result due to surrounding oedema. 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 [22].

The pulmonary (inhalational) form of anthrax is very rare and often presents with mild and non-specific symptoms including fever, malaise and mild cough or chest pain (upper respiratory tract symptoms are rare).

Table 2: Clinical signs of anthrax in different species [23]

| Hosts | Clinical sign |
|------------------|--|
| Cattle and sheep | Septicemia form of anthrax. |
| Pigs | Usually sudden death. Sub acute with edematous swelling in pharyngeal tissues and regional lymphadenitis or intestinal form with a higher mortality. |
| Horses | Oral route: septicemia with colic and enteritis. Wound infection: localized edema and lymphadenitis. |
| Carnivores | Comparatively resistant. Disease pattern similar to that in pigs. A massive dose from eating anthrax infected carcass can lead to septicemia. |
| Humans | Skin form: malignant pustule, pulmonary (wool sorter's disease) and intestinal forms are often fatal. |

Early symptoms may be confused with a flu-like illness. This is followed within three to six days by rapid onset of hypoxia, dyspnoea 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 prodrome significantly decreases the mortality rate [21].

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, septicaemia and death. Case fatality rates of 25–75% have been reported. In oropharyngeal anthrax, fever, neck swelling due to lymphadenopathy, throat pain, oral ulcers and dysphagia may be followed by severe local ulcers and swelling, septicaemia and death. Case fatality rates are similar to the intestinal form [21].

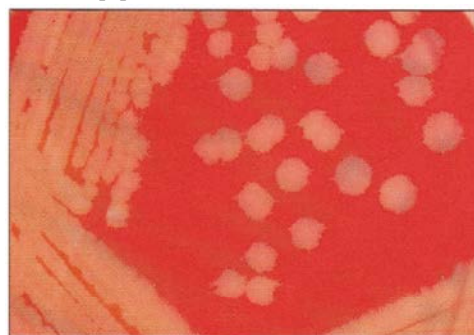
Diagnosis: To confirm the diagnosis on an unopened carcass, peripheral blood or local edema fluid should be collected by needle puncture. Since the blood clots poorly, jugular vein puncture may permit sample collection. Blood can also be carefully collected from an ear vein to avoid unnecessary contamination 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 specimens [13].

In Direct Microscopy, *Bacillus anthracis* produces a capsule *in vivo* and either Giemsa or polychrome methylene blue stains are used to demonstrate the capsule which is of diagnostic importance. The capsule material is more abundant if the blood smear has been taken from a recently died animals. Polychrome methylene blue stained smears reveals square ended, blue rods in short chains surrounded by pink capsular materials. In case of giemsa stained smears, the capsule is reddish [24].

Isolation and Cultivation of *Bacillus* species in the *Bacillus cereus* group growth well on sheep or ox blood agar, aerobically at 37°C in 24-48 hours. A MacConkey agar plate could also be inoculated with the specimen as a check on gram negative contaminants. Contaminated specimen such as hair, bone meal and other animal feeds should be ground finely, steeped in saline then heated at 65°C for 10 minutes. On cooling the suspension is stained through gauze and centrifuged. The deposit can be used for culture or animal inoculation [23].

When virulent strains are 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 [25].

Identification of *bacillus anthracis* in Colonial morphology to diagnose in a hospital microbiology laboratory is based on direct Gram's stained smear of the skin lesion, blood, or cerebrospinal fluid, 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 [8].



216 *B. anthracis* on sheep blood agar illustrating non-haemolytic, flat, 'ground-glass', dry colonies with irregular edges.

Fig. 2: Colonial morphology of *B. anthracis* [24]

Table 3: Pharmacological therapy for bacillus anthracis infection and its sequel [22]

| Drug | Dosage for adult | Dosage for children |
|--------------|--|--|
| Penicillin v | 200-500 mg only orally four times per day | 25-50 mg/kg body weight per day orally in divided doses two or four per days |
| Streptomycin | 30mg/kg intra muscularly or intravenously per day | - |
| Doxycycline | 200 mg orally or intravenously as a loading dose, then 50_100mg every 12 hours | Not provided for children |

Animal test is used for confirmation of the diagnosis. *Bacillus anthracis* is 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 [10].

Bacillus anthracis produces an inverted fir tree type of liquefaction with side shoots radiating from the stab line, while the other species rapidly liquefy nutrient gelatin. *Bacillus anthracis* and the majority of bacillus species do not normally produce capsules in or on 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% CO₂. The colonies are quite mucoid [24].

Differential Diagnosis: Depending up on clinical picture or signs the disease can be differentiated from: *Glander*; fever and inflammation of lymph node but not bloody diarrhea and after die there is no loss of tarry blood in the natural orifice [10]. *Plague*; includes fever, bleeding under the skin and swelling of lymph node but not pain less swelling of lymph node and not loss of clotted blood in the natural orifice [26]. *Typhoid fever*; shows highly fever, edematous on the chest and abdomen but not swelling of lymph node. *Q fever*; shows illness, influenza and pneumonia. *Black leg*; there is edema of skeletal and cardiac muscle. The ulcerative eschar of cutaneous anthrax must be differentiated from other popular 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 super infected with pyogenic bacteria [27].

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 [27].

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 [28].

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 liters per square meter [27].

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 un capsulated strain of *Bacillus anthracis*. The vaccine gives protection for one year. The vaccines prepared from none living antigens do not give adequate immunity [26]. A new wave of research on vaccines against the capsule has also introduced new candidates for development [19].

The control of meat and milk producing animals in infected heard in such away 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. 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. 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. Bioendemic meals sterilized by dry heat (150°C per 3 hrs) or steam at 115°C for 15 minutes is very important [27].

Impact of Anthrax

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 for handlers of processed hides, goat hair, bone products, wool 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 knacker workers and those handling pet meat. 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 [22].

Anthrax is still a significant risk in some countries and outbreaks occasionally occur in humans. 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. In the U.S., approximately 130 human cases occurred annually during the early 1900's, but only one or two cases of cutaneous anthrax are now generally seen in a year. In many countries, cases of anthrax occur infrequently and sporadically, mainly as an occupational hazard among veterinarians, agricultural workers and workers who process hides, hair and 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. 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 regimens may decrease the mortality rate. In the 2001 mail-associated bioterrorist attack, six of eleven patients with inhalational anthrax recovered with treatment (case fatality rate of 45%). However, once a patient reaches the fulminant stage, one study suggests that the mortality rate is 97% regardless of treatment. Anthrax meningo-encephalitis is also deadly, with an estimated case fatality rate of 95-100%. Only limited information exists for gastrointestinal anthrax. The case fatality rate for the abdominal form is unknown, but it is estimated to be from 25% to 60-75%. Asymptomatic or mild infections have been described among adults in some outbreaks, with higher mortality rates in children [22].

Economic Significance: 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 [29]. 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 [22].

CONCLUSION

Anthrax is an infectious disease caused by the bacteria *Bacillus anthracis* and it affects both animals and humans. Generally, the disease 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. 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 is a special aspect of the control of anthrax. 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.

Generally, in order to reduce this disease the following explicit recommendations are forwarded:

- Eating of raw meat should be avoided
- Rapid diagnosis and treatment of sick animals with effective antibiotics are important.
- Control of the infected animals, prevention of contact with the infected animals and contaminated animal products are quite important.
- Environmental and personal hygiene, medical care, disinfection of wool, soil with formaldehyde should be practiced.
- Health education should be given on precaution of industrial hygiene for farmers, butchers, tanners and other industrial workers.
- It is better to draw vaccination schedule for individuals at risk.

REFERENCES

1. Fulako, T., 2004. Immune system paralysis by anthrax lethal toxin. The role of innate and adaptive immunity. *Journal the Lacent Infectious Disease*, 4: 166-170.
2. Boron, E.J., R. Peterson and S.M. Finegold, 2002. *Diagnostic Microbiology*. 9thed, Bailey and Scot's, pp: 451-454.
3. Wattiau, P., S.R. Klee, D. Fretin, M. Van Hessche, M. Menart, T. Franz, C. Chasseur, P. Butaye and H. Imberechts, 2008. Occurrence and genetic diversity of *Bacillus anthracis* strain isolated in an active wool-cleaning factory, 78: 1207-1210.
4. National Center for Biotechnology Information, U.S. National Library of Medicine, 1989. <http://www.ncbi.nlm.nih.gov/About/disclaimer.html#disclaimer>[accessed on 15/05/015, 11:30pm].
5. Shafazand, S., R. Doyle, S. Ruoss, A. Weinacker and T.A. Raffin, 2001. Inhalational Anthrax: Epidemiology, Diagnosis and Management. *Chest*, 116: 1369-1376.
6. Mock, M. and A. Fouet, 2001. Anthrax. *Annual Review of Microbiology*, 55: 647-671.
7. Dixon, T.C., M. Meselson, Guillemin and P.C. Hanna, 2000. Anthrax. *New England Journal of Medicine*, 341: 815-826.
8. Spencer, R.C., 2003. "Bacillus anthracis". *Journal of clinical pathology*, 56(3): 182-187.
9. Ezzell, J.W. and S.L. Welkos, 2003. The capsule of *Bacillus anthracis*, areview on *Journal Application of Microbial*, 87(2): 250.
10. Carter, G.R. and D.J. Wise, 2004. *Essentials of veterinary bacteriology and mycology*. 6thed. IowaStatePress, Ames, IA., pp: 179-182.
11. Slonczewski, J.L. and J.W. Foster, 2010. *Microbiology*. 2thed. An Evolving science, pp: 1-22.
12. Read, T., 2003. "The genome sequence of *Bacillus anthracis* Ames and comparison to closely related bacteria". *Nature*, 423(6935): 81-86.
13. Radostits, O.M., C.C. Gay, K.W. Hinchchiff and P.D. Constable, 2007. *Veterinary Medicine. A textbook of the disease of cattle, horse, sheep, pigs and goats*. 10th ed. Saunders, pp: 815-819.
14. Paccani, S.R., O.F. Tonell, L. patrussi, N. Capitani, M. Simonton, C. Montecco and C.T. Baldari, 2007. Anthrax toxins inhibit immune cell chemotaxis by perturbing chemokine receptor signaling. *Journal of cellular Microbial*, 9: 924-926.
15. Lee, K., 2007. "Phenotypic and functional characterization of *Bacillus anthracis* biofilms". *Microbiology*, 153: 1693-1701.
16. Buxton, A. and G. Fraser, 1977. *Animal Microbiology*. Black well science, Edinburgh, pp: 195-203.
17. Radostits, O.M., D.C. Blood and C.C. Gracy, 1994. *A text book of Veterinary Medicine*, 8th ed. Bailler Tindall, pp: 673.
18. Van Ness, G.B., 2008. Ecology of Anthrax. *Science*, 172: 1303-1307.
19. MahtabMoayeri, Stephen H. Leppla, Catherine Vrentas, Andrei omerantsev and Shihui Liu, 2015. Anthrax Pathogenesis. *Annu. Rev. Microbiol.*, 69: 185-208.
20. Hungerford, T.G., 1990. *Disease of live stock*. 9th ed. McGraw-Hill, Sidney, pp: 329-332.
21. Gracey, J.F., D.S. Collins and R.J. Huey, 2000. *Meat hygiene*. 10th ed., pp: 507-509.
22. Nijm, H. and M. Hugh-Jones, 2001. 1996-97 Global anthrax report. *J. Appl. Microbial*, 87: 189-191.
23. Quinn, P.J., M.E. Carter, B. Markey and G.R. Carter, 1994. *Clinical veterinary microbiology*, pp: 178-183.
24. Quinn, P.J., B.K. Markey, M.E. Carter, W.J.C. Donney, C. Leonard and D. Maghire, 2003. *Veterinary Microbiology and Microbial Diseases*. Black well science, Dublin, pp: 80-83.
25. Pipkin, A.B., 2002. Anthrax. In B.P. Smith, *Large Animal Internal medicine*. 3rded. Mosby, St. Louis, MO., pp: 1074-107.
26. Sharma, S.N. and S.C. Adlakha, 2008. *Text book of Veterinary Microbiology*, pp: 138-141.

27. Hirsh, D.C. and Y.C. Zee, 2003. Veterinary microbiology. USA: Black well science, pp: 246-249.
28. Schlomovitz, J., S. Weiss, H. Levy, Z. Altboum, D. Kobiler, N. Rothschild and N. Rothschild, 2011. Lethal factor is not required for *Bacillus anthracis* virulence in guinea pigs. *Microb. Pathog.*, 51: 345-351.
29. Turn bull, P.C., M. Hugh-jones and O. Cosivis, 2005. World Health Organization activities on anthrax surveillance and control. *J. Appl. Microbial.*, 72: 318-320.