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REVIEW OF ANTHRAX: A DISEASE OF ANIMALS AND HUMANS

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ABSTRACT

Anthrax is a bacterial infection that affects a number of animals but clinical form is mainly seen in herbivores and humans. It is caused by Bacillus anthracis, a spore forming gram positive bacteria that can persist in the environment for long period and natural climatic variations can spark an outbreak. A clinical disease presents with peracute manifestation and usually sudden death could be the only information from the farmer. Basic diagnosis of the disease is through Giemsa/ Grams stained blood smear. Opening of infected carcasses or leakages of the bacteria have been reported as the possible source of environmental contamination. Humans are infected by the vegetative forms from biological tissue and depending on the route of infection can manifest as respiratory, gastrointestinal or cutaneous anthrax. Despite the disease being in existence for long and several researches and control/preventive measures being put in place, it is still a challenge to livestock production in many countries. Therefore, a review of biological, clinical, epidemiological characteristics, control measures and public health significance of this disease are paramount.

Keywords: Anthrax. Review. Animals. Zoonosis.

1. INTRODUCTION

Anthrax is serious zoonoses with global distribution affecting livestock, wildlife and occasional outbreak sparks in humans with animal sources as the main origin [1]. It is one of the earliest zoonoses that has been described in ancient literature of the Hindus, Greeks and Romans, and is thought to be among the fifth and the sixth plagues described in the bible that struck the ancient Egypt [2]. It is enzootic in many African and Asian countries but it has also been reported in some countries in America and Europe [3, 4]. It is caused by a spore forming, Gram-positive bacterium known as *Bacillus anthracis*, which manifests in a bimodal lifestyle; the spore (in the environment) and vegetative (inside the host) forms [2]. The spores are dormant in the environment and can persist for several years and spark outbreaks when suitable conditions are provided for [5].The spores have been described as having unique association with plant roots, an adaptation that increases its ability of infecting ungulates host while grazing [6]. The susceptibility of domestic and wild animals vary, with domestic and wild herbivores most susceptible, equines less susceptible while carnivores and omnivores being relatively resistant [7]. Wildlife is also affected by anthrax and a number of studies have reported incidences in wildlife including Serengeti eco-system in Tanzania [8] and in Malilangwe Wildlife Reserve in Zimbabwe [9].

According to WHO, the global estimation of incidence of anthrax ranges from 20,000 to 100,000 human cases every year, with higher incidences in developing countries of Africa and Central and Southern Asia [10].There has been a surge in reported anthrax cases in both humans and animals in several countries over the past years and this is not clear whether the global burden has increased or there has been better reporting system [11].

In Kenya, anthrax outbreaks occur continuously from different parts of the country and therefore it has been ranked as the first zoonoses with high incidences [12].

The purpose of this review on anthrax disease is to compare the studies that have been done globally ranging from biology of the bacterium, pathogenesis, animal species affected, human forms, risk factors associated with outbreaks and spread, diagnosis and control measures.

2. BACTERIAL CHARACTERISTICS

2.1 The bacterium

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Bacillus anthracis is a spore forming, Gram-positive, aerobic and non-motile rod-like bacteria measuring 1-1.5mm by 3-10mm (figure 1) [13].Spores occur on exposure to air due to depletion of nutrients and are highly resistant, and can survive in the environment (soil, water, air and vegetation) for several years despite extreme temperatures, chemicals and even ultra-violet radiation [5]. This ability of the bacterium ensures persistence of the disease in a particular area. It grows readily in all conventional laboratory media to produce rough-edged, white non-hemolytic colonies of approximately 4-5mm with characteristic comma or tail shaped referred to as medusa heads or curly-hair colonies [13]. In cultures, it appears as long chains refer to as boxcar appearance while in tissues it occurs either singly or in pairs [14].

Figure 1. Transmission electron micrograph of negatively stained (2% uranyl acetate) *B. anthracis* endospores, including an intact spore (A), free exosporium (B), and an NS (C). Magnifications, _92,000 (A and C) and _13,500 (B). (courtesy of [15].

Bacillus anthracis has two plasmids pX01 (coding for tripartite protein exotoxin complex PA, LF and ED) and pX02 (coding the capsule genes). The bacterium is genetically homogeneous and this has been attributed to the prolonged dormancy of the bacteria in the soil that slows its evolution [15]. The three well-known strains are Ames, Sterne and Vollum. Ames is the highly virulent strain containing both plasmids and was first isolated in a dead cow in 1981 in Texas, USA [16]. The sterne strain (found in Canada) is avirulent as it has only pXO1 plasmid but toxigenic and is generally use in vaccine production [17]. The less virulent form is Vollum and is used mainly for research and is found in Spain, United Kingdom and Zimbabwe [18].

Bacillus anthracis is a member of the Bacillus cereus group of bacilli that include other members like *B. thuringiensis*, *B. mycoides* and *B. cereus*. *Bacillus anthracis* differ from other members in a the following ways; susceptible to penicillins, non-hemolytic, non-motile and having a D-glutamic acid capsule which is not the case with other species [18]. Biochemically *B. anthracis* ferments sugars (glucose, sucrose and maltose with acid production), liquefies gelatin and hydrolyze starch.

2.2 Anthrax toxic factors

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There are three toxic factors that have been extracted and purified from *B. anthracis*, the Protective Antigen (PA), Lethal Factor (LF) and Edema Factor (EF) [20]. Protective antigen is a toxin that must be present for any other toxin to be able to cause pathology. It acts as a binding domain (B) while LF and ED are the active domains (A), combining individually to form lethal toxin (LT) and edema toxin (ET), respectively [20].

Studies have shown that there is usually a synergy between the effects of the above toxins as the effects of each toxin in separate experiments showed less toxicity than when in combination [21].

These exotoxins form the two types of toxins A and B produce by *B. anthracis*. The subunit A comprises of separate polypeptides that are specialized for toxic (ED) or catalytic (LD) activities and subunit B (PA) for the delivery of toxins into the cell. Protective Antigen (PA) is a 83 KD precursor polypeptide that is cleaved by cell surface Furin type protease to generate an active 63 Kd product that binds and transport the enzymatic toxin moieties LF and ED subunits into the late endosomes in the cytoplasm [22]. Lethal Factor (LF) is released into the cytoplasm but EF remains attached to the endosomes around the nucleus [23].

Lethal Factor (LF) cleaves and inactivates all the protein kinases in the cytoplasm leading to the cell apoptosis, cell fate determination and response to different forms of cellular stress [24]. It also increases the expression of interleukin-1 (IL-1) and Tumor necrosis factor (TNF) inside the macrophages resulting in cell lysis and release of inflammatory mediators leading to apoptosis [25]. Edema Factor is calmodulin (CaM)-dependent adenylate cyclase whereby in low cytoplasmic PH it generates a gradient of cAMP that is destructive to the cell [26]. It causes loss of chloride ions and water from the cell resulting in extracellular edema. It also secondarily inhibits oxidative burst and phagocytic activity of neutrophils [25]. The high levels of intracellular cAMP alter several cellular processes resulting in hypermotility of infected macrophages. Edema Toxin (ET) also induces vascular endothelial growth factor and Syndecan-1 genes mimicking the normal chemotactic signaling by chemotactic G-protein causing rapid entry of macrophages into the lymph nodes [27].

2.3 Toxins disrupting cardiovascular system

During the terminal stage of the disease, death occurs because of toxic shock with severe pulmonary dysfunction and cardiac failure. It is believed that toxins produce cytokine storm like symptoms resulting in anaphylactic shock. Therefore, LT and ET breach the vascular barrier leading to several fatal systemic effects including cardiac failure, hemoconcentration, hypotension, tachycardia and meningitis [28].

2.4 The Bacillus endospores

Sporulation of bacteria occurs as a response to depletion of nutrients and is commonly seen in two genera of bacteria, Clostridium and Bacillus. Metabolism is reduced and a cascade of alternative factors are expressed that coordinate expression of mRNA responsible for sporulation [29]. Bacillus spores are metabolically inactive and have ordered structure that enable them survive harsh conditions for very many years. Germination of spores occurs when the spores' germinant sensors in an aqueous environment sense small molecules or ions. Bacillus spores recognize specific signals (specific amino acid and nucleoside) produced by the host cells and germinate rapidly [30]. This involves core hydration, ejection of cations and expulsions of dipicolinic acid, cortex breakup and initiation of vegetative metabolism with production of toxic factors [30]. A number of proteins including exosporium have been identified as the major surface membrane protein of *B. anthracis* that are exposed to the host cells and initiate intracellular changes [31].

2.5Pathogenesis of anthrax

Soil is the primary reservoir for *Bacillus anthracis* spores [32]. Animals become infected by ingesting and or inhaling spores from infected soil. Herbivores are primarily susceptible to infection when they ingest sufficient amounts of spores on pasture, soil or drinking water or concentrated bone meal [7]. Carnivores are infected when they consume meat contaminated with the spores. Vultures and flies that feed on infected carcasses may also aid mechanical transmission of spores. Vultures are believed to contaminate water by washing their beaks and feathers after feeding [33]. However, they also aid in reducing the spread of the disease by feeding on carcasses before vegetative form sporulate in the environment [34]. Both non-biting (Chrysoma and blowfly) and biting flies (Tabanus or Stomoxys species) have been implicated in a number of anthrax outbreaks in both livestock and wildlife

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in USA, India and Africa [10]. Outbreaks are largely associated with changes in the climatic conditions including prolong drought, heavy rainfall and flooding and disturbance of soil layers [35].

It is believed that the spore are non-invasive and unless there is a break in the integrity of integumentary, intestinal mucosa or respiratory cilia, initiation of infection do not occur [36, 37]. Therefore, lesions are thought to be as a result of ingestion of thorny vegetation or grit while herbivores graze too close to the ground. Infection can occur either through the skin (cutaneous), gastrointestinal tract or respiratory system [35]. Cutaneous form occurs when bacteria gain access through wounded skin resulting in the edema of the site that progress to formation of painless black sores. Systemic infection consists of two stages, the prodromal and fulminant stages. Prodromal stage is an asymptomatic stage where the bacterial spores are engulfed by the phagocytic cells and transported to the regional lymph nodes [35, 38]. The cells burst and release spores that begin to germinate producing vegetative phase. The fulminant stage follows where bacteria proliferate and disseminate systemically via cardiovascular system to nearly all the body organs leading to death in 1-2 days [38].

Outbreaks of anthrax in humans is usually due to consumption of infected meat, contact with contaminated carcass, animal products including hides, hair, hooves plugs, infected animal body fluids or contaminated utensils.

4.CLINICAL MANIFESTATION OF ANTHRAX

4.1 Herbivores

Among the herbivores, cattle have been documented to be involved more than other species [39]. This phenomenon has been attributed to how different herbivores feed. Cattle tend to pull pasture out of the ground with roots unlike sheep or goats, which bite plants off at the ground level or browse on shrubs, respectively [39]. Therefore, cattle are more likely to ingest high doses of the bacterium from potentially contaminated soil compared to other herbivores. In Wales, bovine anthrax outbreaks occur four times that of sheep with thirteen times mortalities than in sheep [40]. The disease is usually peracute or acute in ruminants taking 3-5 days depending on the ingested dose and clinical sign are in apparent [36]. The death in peracute cases is preceded with a short phase of admixture of signs including; staggering, excitation/somnolence, convulsions, recumbency, spasms, dyspnea, colic and swellings around the neck, chest and abdomen [41]. Animals are usually found dead near water points or grazing fields where previous cases were reported to have been buried or disposed. Ulcers may be seen in the oral cavity and dying animal often bleed from the natural orifices (nose and anus), rapid bloating and incomplete rigor mortis [10, 36]. At death, herbivore blood contains titers of approximately 10^8 to 10^9 bacilli in a deciliter of blood that can sporulate on exposure to air. Acute forms may manifest with fever, congested and sometimes hemorrhagic mucous membranes, muscle spasms and labored breathing with terminal convulsions and death [41]. However, this could be preceded by a short phase of excitement followed by depression and listlessness. Abortions and blood stained or yellowish milk has been reported in dairy cattle [41].

Chronic or subacute cases are rare.

4.2 Horses

Horses suffer acute type of infection manifesting with fever, colic, dysentery, labored breathing, swellings with necrotic centers and bloody discharge from the nose and anus [10, 36].

4.3 Pigs

Porcine are moderately resistant to infection but they may develop inapparent infection that is occasionally detected at slaughter or during post mortem. Pharingitis, local painful neck edemas and skin papule may be seen. In gastro intestinal tract involvement, sign like vomiting, icterus and diarrhea may be apparent. At post mortem, pathological lesions are confined to pharyngeal and intestinal sites [42].

4.4 Carnivores

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Carnivores may be infected by scavenging on infected carcasses tough they are relatively resistant [10]. Cheetahs and mink are more susceptible to anthrax compared to the other carnivores. A wide variety of carnivores has been reported to have been affected over the years [43]. However, domestic dogs have been reported to have fallen incidental victims of bioterrorism attacks with anthrax spores [44]. After an incubation period of about 3-5 days, they may present with fever (>40 ^{0}C), lethargy, anorexia, edematous lesions on the lips, jaws, tongue, neck, limbs and terminal death with bloody exudates from the orifices [44, 45]. Mink have a very short incubation period of less than 20 hours with 50-100% mortality rate with spleenomegally and pulmonary edema as the major pathological changes [46, 47]. In dogs, oropharynx and upper gastrointestinal tracts are the main portals of entry of the bacterium. This results in edema of the head, neck and mediastinal structures [48]. Hypersalivation, dysentery and swollen forelimbs have been reported in a dog that fed on contaminated carcass and succumb due to shock and toxemia [44, 49].

4.5 Birds

Apart from ostriches, anthrax cases have been reported only in birds in captivity, including poultry, pigeons and eagles [50]. However, these cases are rarely reported and when they occurs, birds manifest with septicemic signs and sudden death. Pathologically, dark skin coloration, edematous and hyperemic lungs and hemorrhagic enteritis are apparent [50].

4.6 Laboratory animals

Experimental studies on laboratory animals including guinea pigs, mice, or rabbits have shown that these species are susceptible. The spores germinate within 2-4 hours of intradermal injection resulting in extensive edematous and necrotic lesions. The bacilli spread through the lymphatic system to the spleen, where they multiply resulting in septiceamia and death [51]. Inhalational form has been described in rabbits with similar pathological manifestation to the human form [52] and in rhesus monkeys [53].

4.7 Anthrax in wildlife

Anthrax disease affect a wide range of wildlife including impala (*Aepyceros melampus*), kudu (*Tragelaphus strepsiceros*), buffalo (*Syncerus caffer*) and zebra (Equus spp.) [54, 55]. Within the same ecosystem, different animals are affected at different times and this is thought to be as a result of differences in the host specificity and susceptibility and pathogenic strains [8]. Sporadic outbreaks have been reported in Serengeti ecosystem (land coverage over 20,000KM^2 with varied environmental gradients) in localized foci affecting a few species of wildlife [56]. Giraffes, buffaloes, impalas, wildebeests, cheetah and livestock were found to have been affected in the Serengeti ecosystem. Seroprevalence was consistently high in the carnivores samples [8].
An anthrax outbreak investigation in Wamba area in Samburu showed 53 Grevy's zebra and 26 plains zebras, 5 donkeys and 2 camels succumbed [54]. In Zambia, there was a decline in the population of wild dogs in Luangwa valley and this was attributed to anthrax outbreak [57].

4.8. Humans

Anthrax infection in humans has been categorized into two; Agricultural and Industrial. Agricultural cases occur when people come in contact with tissues from infected or death animals accidentally or during slaughter [33]. These include veterinarians, butchers, slaughterhouse workers and even ranchers. On the other hand, Industrial cases are those that occur during cleaning and processing of infected animal products [58]. Inhalational and cutaneous forms have been reported due to exposure to contaminated hair, wool, hides and skins, therefore persons working in industries processing these materials are at risk of exposure to anthrax spores. Industrial cases consisted of 65% of all the cases encountered during the period 1955-1999 and reported to CDC [58].

In humans, three forms of the disease can be manifested depending on the route of infection

4.8.1 Cutaneous anthrax

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This is the most commonly encountered form comprising of upto 90% of all the human cases. It is characterized by popular skin lesion, which is black (coal-like) with necrotic centre surrounded by fluid-filled vesicle [59]. The centre ulcerates and dries up forming a black scab, a lesion that is usually painless. Regional lymphadenopathy, swelling of the face and neck may be apparent [60]. Secondary infection may result in pain, fever and pus production from the lesions. These cases resolve often spontaneously with early treatment and fatalities occur in approximately 5-20% of untreated cases and less than 1% of treated patients.

4.8.2 Gastrointestinal anthrax

This is the second commonly occurring form that develops after consuming contaminated meat. The spores penetrate the intestinal epithelia and germinate resulting in ulcerative lesions leading to obstruction, hemorrhage, or perforation. The symptoms may be mild including fever, malaise, vomiting, anorexia and diarrhea. Acute onset may follow with hematemesis, severe abdominal pain, massive ascites and bloody diarrhea that may progress to shock, coma and death [33]. Case fatalities ranges from 60-75% with treatment significantly lowering fatalities.

4.8.3 Inhalational anthrax

This is the most fatal and rare form of infection. It is due to inhalation of anthrax spores in the environment and commonly associated with industrial exposure (woolsorters' disease) or bioweapon [61]. Symptoms develop gradually and are usually non-specific [60]. Initial signs include malaise, fever, lethargy, weakness and dry cough with mild chest pain. The symptoms improve with time but prodromal phase ends with onset of acute dyspnea, tachycardia, cyanosis, stridor and fatal septicemia. Case fatality has been estimated at 90-100% of patient at the fulminate stage regardless of treatment.

4.8.4 Meningeal Anthrax

This form is rare and usually a complication of any of the above forms. It results from haematogenous or lymphatic spread of the bacteria or exotoxin to the brain, spinal cord and or the meninges [61].This clinically manifest with fever, headache, agitation, rigid neck and delirium. Necropsy shows cardinal caps or dark red meninges and bloodstained cerebrospinal fluid [25].

5. ANTHRAX SPORES AS BIOWEAPON

The bacterium has been documented as having been use as a biological weapon in various countries including United States of America at one time [62]. Bioterrorism has considered biological agents as attractive weapons because they are relatively inexpensive to produce, easy to access, preserve and it achieves a widespread panic and fear compared to the actual physical damage [20]. The spores of *B. anthracis* are the most commonly used biological agents as the spores are extremely resistant in the environment and can survive for many years [63] with a half life of about 100 years [64]. These spores can be aerosolized and disseminated as invisible and odorless aerosol and this has made it a potential for bioterrorism. In USA, 10 cases of inhalational anthrax were confirmed and they were linked to bioterrorism where mails deliberately contaminated with anthrax spores were delivered [65]. The LD_{50} of respiratory form has been estimated experimentally at 4130 spores with 95% confidence interval [66]. whereas natural infection ranges from 6200-22000 [67]. In the former Soviet Union, 64 deaths were reported after anthrax spores were accidentally released from microbiology laboratory in a military facility [68].

Outbreaks of anthrax

Outbreaks of anthrax cases both in animals and humans are usually sporadic, commonly seen in anthrax 'belts' where previous cases have been reported mainly in lowlands, floodplains and in areas with excessive disruption of soil [40]. Outbreaks have been reported in different parts of the world at different seasons and geographical features (Figure 2). Alkaline soil rich in calcium and organic matter and PH above six has been documented to incubate the bacilli and such area report sporadic outbreaks [69].

Animal anthrax outbreaks have been reported globally affecting different species. In Russia, 88.7% mortality rate was reported in Reindeer herds and similarly several outbreaks were reported from cattle and horses in Sweden [11].

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In Kenya, Anthrax is an endemic disease with outbreaks being reported almost every year. Central and Rift valley regions report outbreaks that have been associated with natural factors such as prolonged drought, heavy rains and desertification that has lead to bare soil ready for disruption by winds therefore exposing spores. Several media reports and personal communication by the practicing veterinarians indicate that the disease is in existence and human involvements are usually the primary indicator of the disease. Sudden deaths in livestock especially ruminants most of the times went unnoticed because of early disposal without reporting to the veterinary authority. Cultural beliefs in some communities that carcass should not be buried whole as this is seen as bad omen has led to contamination of the environment and therefore source of infection to other animals and even humans. Therefore community education on the signs, proper disposal procedures and the public health importance of this disease is necessary to reduce outbreaks and improve livestock production.

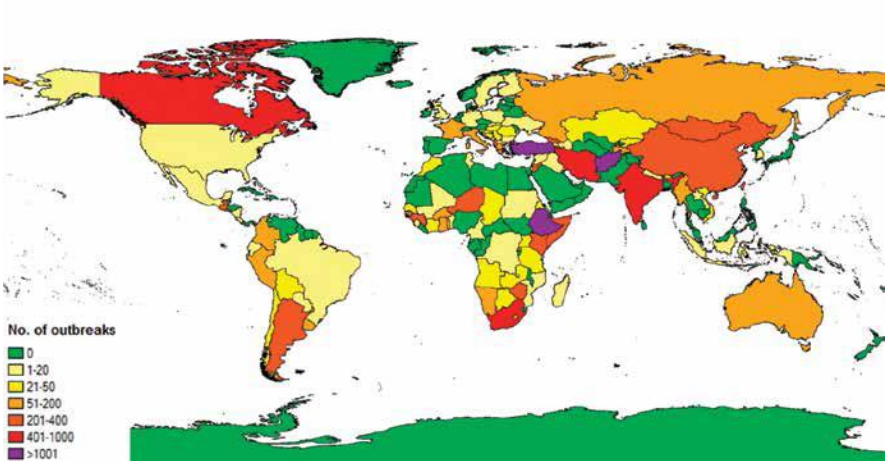

Figure 2. Anthrax outbreaks reported in livestock, wildlife, and humans, January 2005 to August 2016 (Source: FAO)

Risk factors

Some environmental conditions favor the survival of bacillus spores making certain regions referred to "anthrax areas" where they remain viable in soil for long. The endemicity has been associated with neutral or alkaline soil rich in Calcium and Nitrogen and temperature above 15.5ºC [70, 71]. However, different strains have preference to specific soil chemistry influencing the anthrax epidemic spread [72]. Seasonal climatic factors including rainfall and ambient temperature trigger outbreaks in anthrax areas. Outbreaks have been reported late in dry season after prolong drought [34], suggesting the possibility of lack of water and feed make livestock graze close to the ground, congregating at water sources and nutritional stress being the major animal factors [43]. On the other hand, heavy rains have been associated with outbreaks hypothesizing the rains unearth the spores as well as amplifying vector population [55, 73, 74]. Experimental research on spore germination and growth of vegetative forms in the rhizosphere has suggested a mechanism of rainfall sparking epidemics in dry summer after rains and when animals are in good body conditions [75]. Generally in enzootic areas, anthrax outbreaks occur following a prolonged dry hot period preceded or succeeded with heavy rains, where temperature are abnormally high. In temperate regions, thawing of permafrost has been associated with outbreaks.

The elevation of the area also plays an important role in the outbreak. In the low-lying areas, rain water collects from upper hills and concentrate the spores, contaminating soil, pasture and water in the plains [76]. These are the areas with high evaporation potential of flood water and green pasture throughout the year as they are waterbed and the last to dry off.

6.Community practices that increases risk of anthrax spread

6.1 Livestock rearing practices

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Many communities especially in pastoral areas keep their cattle in cowshed around the homestead (Figure 3) and small domestic ruminant in their houses or verandas in fear of theft [77]. Other people share the household utensils with their livestock; especially lactating animals are given water, kitchen waste and mineral with household sufurias. The close proximity increases the risk of transmission of diseases form one animal to the other and to humans.

Figure 3. A typical household with cattle in anthrax endemic Sirjganj area, Bangladesh (courtesy of [77].

8.2 Slaughtering of sick animal

Salvage slaughter of moribund animals is a common practice in many communities. Once the animals are recumbent, they are slaughtered, as their culture does not allow consumption of dead animals [77, 78]. Therefore, livestock owners and their neighbors slaughter sick animals and sell their meat to recoup their financial investments [79]. Most of these animals are slaughtered in the backyard or cowshed around the homestead (Figure 4) or in the premises as they are not able to move to slaughter houses/abattoirs. The carcasses are rinsed with water to remove the blood and the waste products are disposed at the nearby ditches, garbage, water bodies or open fields. In other cases where livestock are taken ill in the grazing field, they are slaughtered immediately as they could die before they are moved to the homestead [79]. Carnivores and birds often scavenge on the discarded waste and possibly become infected or mechanically transmit to other susceptible animals [77].

Fig. 4. Butchering a cow on the back yard in front of the cowshed, Sirajgonj District, Bangladesh, 2010. (Courtesy of [77].

6.3 Handling and disposal of infected carcasses

When animals die, the local skinners are normally asked to skin and bury or remove the carcasses form the village to avoid bad smell 79]. In other communities, the skinners are given the skin to sell as a wage for burying or selling the carcass [77].

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6.4 Poor socio-economic status

Anthrax outbreaks have been associated to countries with low socio-economic status and capacity to address animal diseases effectively. These countries have weak veterinary and public health facilities to curb outbreaks. Recurrences have been reported where control programmes and vaccination campaigns are not well implemented because of civil conflict, political unrest and natural disasters. Human outbreaks are linked to poverty where livestock owners slaughter moribund animal to recoup losses, as livestock is their main source of income [77].

9. Diagnosis of Anthrax

Suspicion of anthrax outbreak is usually made from clinical manifestations in herbivores of sudden death with no other apparent signs and post mortem signs [36] However, profuse sweating is seen in camels before death [80]. Blood smear preparation and staining is the most effective and efficient diagnostic procedures especially in developing countries [81]. Bacterial culture and identification is superior to Gram staining but it is time consuming. Serologically, serum samples from wildlife, humans, and carnivores can be collected to analyze for presence of antibodies against the bacterial toxins. This is usually done in the areas where anthrax outbreaks have been reported as this ensures that the subjects have been exposed [80]. Immunoassay tests are carried out to detect antibodies against PA of the bacterium [56]. Molecular test including PCR have been carried out to identify the genetic material of the bacterium in animal tissues [41, 82].

7.Control of Anthrax outbreaks

Effective control of anthrax relies entirely in proper surveillance of the disease, including active investigation of outbreaks and use of historical data, effective and sustainable livestock vaccination programmes and proper disposal of infected carcasses. Epidemiological findings on the bacterial characteristics, its eco-epidemiological niche and risk determinants of natural occurrence play an important role in identifying 'Anthrax spots'. These will aid in predicting potential outbreaks and therefore imposition of preventive measures to curb the incidences. Prompt identification and reporting of anthrax outbreaks allow proper preparation and early response interventions to reduce further spread and the possible infection of humans [81]. To reduce occurrence in endemic regions, early and strategic deployment of vaccine and personnel is key before natural risk determinants occur. Similarly, proper and appropriate disposal of infected carcasses and disinfection of contaminated materials will significantly reduce the number of spores [80]. Appropriate disposal include either incineration, rendering, deep burial or leaving the carcass undisturbed to decompose [25]. Incineration is the most effective method as it ensures complete destruction of the bacteria and therefore the environment is free from spores [32]. Rendering or sterilization temperatures (100°C to 150°C for 10-60 minutes) sometimes do not destroy all the bacteria and rendered material could still act as a source of infection to animals feed. Deep burial is an alternative method but this should be combined with disinfection. It is recommended that the carcass should be buried six feet deep and covered with soil mixed with lime in the ratio 3:1. Disturbance of soil layers (ploughing, soil erosion or digging by scavenging animals) in such sites usually bring the spores to the surface that may result in new outbreaks. This method is unreliable because there have been reports of viable spores from burial sites of anthrax infected carcasses [32]. The least preferred option to dispose an anthrax-infected carcass is to leave it intact, unmoved, and away from access of scavenging animals and humans to undergo natural decomposition [83]. The carcass should be fenced off and covered to avoid access by bird and flies and post warning signs. Vegetative bacilli do not sporulate within a carcass and are inactivated with tome by putrefaction process. There is possible contamination of the environment by the bloody discharges from the natural orifices in dying animals; however, this could be reduced by scorching the site after complete putrefaction [83].

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