INTERNATIONAL JOURNAL OF HEALTH & ANIMAL SCIENCE & FOOD SAFETY

UNIVERSITÀ DEGLI STUDI DI MILANO
DIPARTIMENTO DI SCIENZE VETERINARIE PER LA SALUTE, LA PRODUZIONE ANIMALE E LA SICUREZZA ALIMENTARE

Anthrax, fairly undetected in Papua New Guinea.

Johnson Makaen[1*] and Lydia Tasi[1]

[1] *Emerging and Environmental Disease, Papua New Guinea Institute of Medical Research, Goroka, EHP, Papua New Guinea.*

ABSTRACT.

Anthrax is caused by the organism Bacillus anthracis. The organism is globally occurring and epidemics are reported the world over. It is an important infectious disease of domestic animals. B. anthracis can survive harsh conditions that would otherwise be drastic for other microorganisms. The inherently robust nature of the organism enables its extended survival and facilitates re-emergence. To date, no outbreak has been reported in the Pacific Island region except Australia and New Zealand where outbreaks are reported in both wildlife and livestock. Papua New Guinea has had sporadic (reported) instances of anthrax outbreak, but has not been scientifically established. It is still unclear if the anthrax causing organism is present in the environment or wildlife. It remains to be so until scientific evidence becomes available. This article aims to review any form of documented evidence of anthrax in the countr.

KEYWORDS

Bacillus anthracis; anthrax; epidemic and Papua New Guinea.

PAGES

41 – 48

REFERENCES

Vol. 2 No. 1 (2015)

ARTICLE HISTORY

Submitted: April 12, 2015
Revised: June 11, 2015
Accepted: June 17, 2015
Published: July 16, 2015

CORRESPONDING AUTHOR

Johnson Makaen
Emerging and Environmental Disease, Papua New Guinea Institute of Medical Research, Goroka, EHP, Papua New Guinea

P O Box 60
Goroka, EHP 441
Papua New Guinea

e-mail: johnson.makaen@pngimr.org.pg

JOURNAL HOME PAGE

riviste.unimi.it/index.php/haf

1 Introduction

Anthrax is caused by the bacteria, B. anthracis. A gram-stained morphology of B. anthracis colony appears as gram positive rods, often linked at lengthwise to form a chain (Murray *et al.* 1990; Brooks *et al.* 1991; Spencer 2003; Schaechter *et al.* 2006; MA Hornitzky and Muller 2010; Todar 2012). It is ubiquitous; survives in both soil and vegetation, and can resist adverse conditions (Turnbull *et al*. 1998; Murray *et al.* 1990; Spencer 2003; Schaechter *et al.* 2006; Veterinary Services 2012). Anthrax is neither contagious nor invasive; however, it causes acute infection in susceptible animals (Fasanella and Garofolo 2008; Beyer and Turnbull 2009; Stoltenow *et al.* 2010). The organism is vegetative in host tissues but sporulates when exposed to the environment (Woodbury *et al.* 2002; Spencer 2003; Schaechter *et al.* 2006; Stoltenow *et al.* 2010). Sporulation shields the organism from unfavourable conditions and enables it to survive for years until it gets re-inoculated in another living host thereby continuing its vicious cycle of infection (Massachusetts Department of Public Health 2006; Schaechter *et al.* 2006; Fasanella and Garofolo 2008; Todar 2012) . In contrast to other zoonoses, anthrax affects a wide range of animals including human (Murray *et al.* 1990; Brooks *et al.* 1991; Spencer 2003; Beyer and Turnbull 2009; Stoltenow *et al.* 2010). It is believed that animals acquire the infection through ingestion of tainted feed or while foraging on contaminated field (Tweet 2010; Veterinary Services 2012). Nevertheless, human unintentionally contract it, usually through the skin while handling infected animals or their products (Turnbull *et al*. 1998; Ron Parker *et al.* 2002; Spencer 2003; Edwards *et al.* 2005; Beyer and Turnbull 2009; Todar 2012). Inhalation of the contaminated aerosol or ingestion of partly cooked meat has also been observed (Edwards *et al.* 2005; Fasanella and Garofolo 2008; Stoltenow *et al.* 2010; Todar 2012; Hajramurni 2013).

2 Epidemiology

Anthrax is caused by the bacteria, B. anthracis. A gram-stained morphology of B. anthracis colony appears as gram positive rods, often linked at lengthwise to form a chain (Murray *et al.* 1990; Brooks *et al.* 1991; Spencer 2003; Schaechter *et al.* 2006; Hornitzky and Muller 2010; Todar 2012). It is ubiquitous; survives in both soil and vegetation, and can resist adverse conditions (Turnbull *et al*. 1998; Murray *et al.* 1990; Spencer 2003; Schaechter *et al.* 2006; Veterinary Services 2012). Anthrax is neither contagious nor invasive; however, it causes acute infection in susceptible animals (Fasanella and Garofolo 2008; Beyer and Turnbull 2009; Stoltenow *et al.* 2010). The organism is vegetative in host tissues but sporulates when exposed to the environment (Parker *et al.* 2002; Spencer 2003; Schaechter *et al.* 2006; Stoltenow *et al.* 2010). Sporulation shields the organism from unfavourable conditions and enables it to survive for years until it gets re-inoculated in another living host thereby continuing its vicious cycle of infection (Massachusetts Department of Public Health 2006; Schaechter *et al.* 2006; Fasanella and Garofolo 2008; Todar 2012) . In contrast to other zoonoses, anthrax affects a wide range of animals including human (Murray *et al.* 1990; Brooks *et al.* 1991; Spencer 2003; Beyer and Turnbull. 2009; Stoltenow *et al.* 2010). It is believed that animals acquire the infection through ingestion of tainted feed or while foraging on contaminated field (Tweet 2010; Veterinary Services 2012). Nevertheless, human unintentionally contract it, usually through the skin while

  ISSN: 2283-3927

handling infected animals or their products (Turnbull *et al*. 1998 ; Parker *et al.* 2002; Spencer 2003; Edwards *et al.* 2005; Beyer and Turnbull. 2009; Todar 2012). Inhalation of the contaminated aerosol or ingestion of partly cooked meat has also been observed (Edwards *et al.* 2005; Fasanella and Garofolo 2008; Stoltenow *et al.* 2010; Todar 2012; Hajramurni 2013).

3 Acquisition

Anthrax is caused by the bacteria, B. anthracis. A gram-stained morphology of B. anthracis colony appears as gram positive rods, often linked at lengthwise to form a chain (Murray *et al.* 1990; Brooks *et al.* 1991; Spencer 2003; Schaechter *et al.* 2006; Hornitzky and Muller 2010; Todar 2012). It is ubiquitous; survives in both soil and vegetation, and can resist adverse conditions (Turnbull *et al*. 1998; Murray, Drew *et al.* 1990; Spencer 2003; Schaechter *et al.* 2006; Veterinary Services 2012). Anthrax is neither contagious nor invasive; however, it causes acute infection in susceptible animals (Fasanella and Garofolo 2008; Beyer and Turnbull 2009; Stoltenow *et al.* 2010). The organism is vegetative in host tissues but sporulates when exposed to the environment (Parker *et al.* 2002; Spencer 2003; Schaechter *et al.* 2006; Stoltenow *et al.* 2010). Sporulation shields the organism from unfavourable conditions and enables it to survive for years until it gets re-inoculated in another living host thereby continuing its vicious cycle of infection (Massachusetts Department of Public Health 2006; Schaechter *et al.* 2006; Fasanella and Garofolo 2008; Todar 2012) . In contrast to other zoonoses, anthrax affects a wide range of animals including human (Murray *et al.* 1990; Brooks *et al.* 1991; Spencer 2003; Beyer and Turnbull. 2009; Stoltenow *et al.* 2010). It is believed that animals acquire the infection through ingestion of tainted feed or while foraging on contaminated field (Tweet 2010; Veterinary Services 2012). Nevertheless, human unintentionally contract it, usually through the skin while handling infected animals or their products (Turnbull *et al*. 1998; Parker *et al.* 2002; Spencer 2003; Edwards *et al.* 2005; Beyer and Turnbull 2009; Todar 2012). Inhalation of the contaminated aerosol or ingestion of partly cooked meat has also been observed (Edwards *et al.* 2005; Fasanella and Garofolo 2008; Stoltenow *et al.* 2010; Todar 2012; Hajramurni 2013).

4 Pathogenesis

The anthrax pathogenesis initiates when the spore enters the skin through lesions, lungs via inhalation or intestines from ingestion of contaminated meat (Turnbull *et al*. 1998; Spencer 2003; Todar 2012). In the tissues, the spores are engulfed and ingested by resident or migratory macrophages. Secured spores are then presented to regional lymph nodes (Hanna 1993; Bush *et al.* 2001; Missiakas and MD 2005; Sherer *et al.* 2007). Due to the capsulated cell wall, the spores evade phagocytosis and eventually vegetate. At this stage, germinated cells exhibit its virulence and toxin factors (Spencer 2003; Edwards *et al.* 2005; Schaechter *et al.* 2006; Todar 2012). Productions of edema toxins induce blistering and necrotic lesions as in skins (Murray *et al.* 1990; Brooks *et al.* 1991; Missiakas and MD 2005). In the intestinal mucosa and alveoli, massive effusion and edema ensues leaving behind necrotising tissues the sum of

ISSN: 2283-3927

which triggers inflammatory responses. The combination of lethal toxins results in impaired immune response, host cell destruction and spread of bacterial infection (Hanna 1993; Spencer 2003; Quinn *et al.* 2004; Edwards *et al.* 2005; Missiakas and MD 2005; Ebrahimi *et al.* 2011). In time, the bacterium reaches the blood stream causing septicaemia, respiratory distress, meningitis and eventually death.

5 Control measures in the event of an anthrax outbreak

As with other emerging infectious diseases, environmental and syndromic surveillance is crucial for prompt detection and control of anthrax outbreak in Papua New Guinea (Paul Horwood and Greenhill 2012). The potential for re-emergence and sporadic occurrence of anthrax is high due its innate ability to survive in nature (Turnbull *et al*. 1998; Spencer 2003; Durrheim *et al.* 2009). An outbreak in one kind of domestic animal can easily spread to other species since local animals in rural Papua New Guinea are free-ranging and often share the same surrounding (Hide 2003; Durrheim *et al.* 2009).

Environmental contamination of anthrax may arise from infected animal carcass being disposed off arbitrarily (Fasanella and Garofolo 2008; Stoltenow *et al.* 2010; Tweet 2010). This raises concern since the environment is a natural reservoir for B. anthracis or facilitates cross-infection of other animals that might feed on the carcass (Stoltenow *et al.* 2010). In view of this, burying of infected animals should be discouraged as the organism will endure long after the carcass disintegrates and disappears. Incineration of infected carcass in situ is important to minimise shedding of pathogenic agents in the environment that might serve as a reservoir for future infection (Caledonia *et al.* 2002; Fasanella and Garofolo 2008; Stoltenow *et al.* 2010; Australia 2012; Veterinary Services 2012). Isolation for vaccination or treatment and culling of infected animals, particularly the terminally ill or nearing dead are crucial to curb transmission and future outbreak of anthrax (Tweet 2010). However, culling may not be practised in rural settings. Even though the sudden death of one or more animal might stir inquisition, investigation of such occurrence by trained personnel is rarely expected. Remoteness of sites and funding limitation tends to be the usual impediment to such effort.

Vaccination is an effective control measure as it offers valuable protection for susceptible animals. It has proven to be successful during outbreak in animal herds (Gill 1993; Turner *et al.* 1999; Allan 2014; Tweet 2010). Human vaccination is purely recommended for individuals that deal with the organism as in laboratory facilities or livestock handlers where anthrax outbreak is endemic. Most cases of human anthrax are attributed to direct contact with infected animals or its products (WHO 2008; Stoltenow et al. 2010). Therefore, vaccination of susceptible animals not only protects them, but prevents the basic route whereby humans contract the infection. Nevertheless, animal vaccination is recommended just about the period when outbreak is detected (Turnbull *et al*. 1998; Gill 1993; Turner *et al.* 1999; Parker *et al.* 2002; Hide 2003; Spencer 2003; Fasanella and Garofolo 2008; Stoltenow *et al.* 2010; Hornitzky and Muller 2010; Tweet 2010; Veterinary Services 2012).

Proper Personal Protection Equipment (PPE) is mandatory for any form of anthrax-related work or epidemiological response. They include: face masks or visor (if available but gas masks

ISSN: 2283-3927

are highly recommended), goggles, boots gloves and overalls with hoods. These are single-use equipment and should be incinerated afterwards or thoroughly sterilized if re-usable.

6 Laboratory Diagnosis

It is a safety requirement that a laboratory facility dealing with anthrax should operate in a Level 2 Biosafety Cabinet and the laboratory personnel thoroughly attired in PPE. Level 3 BC is better still, if the amount of work is substantial or the potential for generating aerosols is high (Turnbull *et al*. 1998). Blood culture is routinely performed on virtually all suspected cases of B. anthracis infection (Veterinary Services 2012). Initial presumptive identification of B. anthracis can be performed through rapid test kits such as Immuno-chromatographic tests, using serum sample of infected animal or human (Hornitzky and Muller 2010). Smears for staining and microscopic examination can be obtained by swab from lesions, tissue fluids or blood sample. Heat-fixation is not applicable for B. anthracis as it not thoroughly bactericidal therefore; smears should be immersed in 40 percent potassium permanganate solution for at least 10 minutes (Cheeesborough 2000). Both gram stain and Loeffler's polychrome methylene blue staining are acceptable (Turnbull *et al*. 1998; Cheeesborough 2000; WHO 2008; Australia 2012). However, Loeffler's polychrome methylene blue staining depicts the encapsulated bacillus which is a clear, but presumptive identification of B. anthracis (Spencer 2003; WHO 2008). Further work up and confirmation would require pure culture and isolation, ELISA techniques, and if available PCR for definite diagnosis and genetic studies (Hornitzky and Muller 2010; Australia 2012).

7 Treatment options

The acute nature of anthrax infection warrants prompt treatment. If delayed, it could lead to serious complications and even death. Hospitalisation is necessary in virtually all cases of anthrax infection. The 'CDC Guidelines for the Treatment of Anthrax' recommends the use of regular antibiotics including; ciprofloxacin, doxycycline, and penicillin for treatment (Prevention 2014). A large dose, administered both intravenously and orally over couple months is suggested for very serious cases of B. anthracis infections as it takes about such length of time for spores to germinate (Vyas 2013). The optimal antibiotic, dosage level, route of administration and the length of treatment may vary with patient and degree of infection. These basic antibiotic treatment options are available in Papua New Guinea. However, advance infections may necessitate the use of antitoxins (Vyas 2013; Administration 2012). The United States Food and Drug Administration have recommended the use of monoclonal antibodies to counteract bacterial toxins which are responsible for the irreparable tissue damage caused during the course of infection (Administration 2012).

ISSN: 2283-3927

8 Conclusion

No human case of anthrax has been reported in Papua New Guinea. If at all, there is no documentation or clinical evaluation of a patient that would otherwise suggest a probable infection. It is worth mentioning particularly, when Papua New Guinea is considered as anthrax endemic while other Pacific Island nations are deemed to be free (Caledonia, Community *et al.* 2002; Fasanella and Garofolo 2008; Stoltenow *et al.* 2010). Nevertheless, one case of an unknown disease outbreak in pigs in a remote area in Papua New Guinea has been reported. This report claimed that three people have died consuming pork from a carcass thought to have died from anthrax. However, it has never been established if *B. anthracis* was responsible. Animal infections were reportedly observed in porcine and village pigs which are commonest form of domesticated animals in the highlands region of Papua New Guinea (Hide 2003; Fasanella and Garofolo 2008; Stoltenow *et al.* 2010; Provet 2013). Still, no human infection has been reported despite the fact that local highlanders live in close proximity to native animals. Other than these, there is no documented evidence of infection in other animals including wildlife or livestock. Concrete scientific data is needed to establish the presence of *B. anthracis* in native animals, wildlife or the environment in Papua New Guinea.

References

Administration USFAD: FDA approves raxibacumab to treat inhalational anthrax. In: First monoclonal antibody approved using the Animal Efficacy Rule. New Hampshire. 2012.

Alla M.B.W., Mohamed T.E, Abdelgadir A.E., 2011. Detection of antibiotics residues in beef in Ghanawa Slaughterhouse, Khartoum State, Sudan. African Journal of Food Science. 5 (10): 574–580.

Animal Health Australia (2005). Disease strategy: Anthrax (Version 3.2).

Australian Veterinary Emergency Plan (AUSVETPLAN), Edition 3, Primary Industries Ministerial Council, Canberra, ACT.

Brooks, Butel, Ornston: Medical Microbiology, 19th edn: Appleton; 1991: 180-182

Bush L. M., Abrams B.H., Beall A., Johnson C.C. Index case of fatal inhalational anthrax due to bioterrorism in the United States. The New England Journal of Medicine 2001.

Caledonia IPN, Community SotP, Organization WH: Anthrax, Generic guidlines for the Pacific Islands. 2002.

Cheeesborough M. District laboratory Practise in Tropical Countries, Part 2. Cambridge: Cambridge University Press. 2000: 37-43

Durrheim D.N., Freeman P., Roth I., Hornitzky M. EpidemiologicQuestions fromAnthrax Outbreak,Hunter Valley, Australia. Emerging Infectious Diseases. 2009, 15(5): 3.

Ebrahimi C.M., Sheen T.R., Renken C.W., Gottlieb. RA: Contribution of Lethal Toxin and Edema Toxin to the Pathogenesis of Anthrax Meningitis. Infection and Immunity. 2011, 79(7): 2510–2518.

  ISSN: 2283-3927

Edwards K. A., Clancy H. A., Baeumner A.J. Bacillus anthracis: toxicology, epidemiology and current rapid-detection methods. Analytical and Bioanalytical Chemistry. 2005, 384(1): 73-84.

Evers M., Robinson S: Anthrax vaccination in NSW. In: Prime Facts. 3 edn. NSW. 2009

Fasanella A., Garofolo G. MHJ: Anthrax undervalued zoonosis. Veterinary Microbiology. 2008, 140(3-4): 318-31.

Gill J. Anthrax - Still history after all these years. Surveillance 1993, 20(1): 21-22.

Hide R. Pig Husbandry in New Guinea. In Canberra, 2003: pp 307.

Horwood P., Greenhill A. Cholera in Papua New Guinea and the importance of safe drinking water sources and sanitation. WPSAR 2012, 3(1): 1-3.

Jernigan J.A., Stephens D.S., Ashford D.A., Omenaca C. Bioterrorism-Related Inhalational Anthrax: The First 10 Cases Reported in the United States. Emerging Infectious Diseases. 2001, 7(6): 12.

Macfarlane D. Country Pasture/Forage Resource Profiles Papua New Guinea. FAO 2009.

Massachusetts Department of Public Health BoCDC: Guide to Surveillance, Reporting and Control of Anthrax. 2006: 11.

Missiakas D.M. Bacillus anthracis and the Pathogenesis of Anthrax. Biological Weapons Defense Infectious Disease. 2005: 79-97.

Muller H. J. Australia and New Zealand Standard Diagnostic Procedure. 2010: 15.

Murray, Drew, Kobayashi, Thompson. Medical Microbiology, vol. 1, International Student Edition edn: Mosby; 1990: 180-183

Prevention CfDCa. Guidelines for the Treatment of Anthrax. In: CDC 24/7: Saving Lives Protecting People™. Atlanta: Centers for Disease Control and Prevention 2014.

Price E.P., Seymour M.L., Sarovich D.S., Latham J., Wolken S.R., Mason J., Vincent G., Drees K.P., Stephen M., Beckstrom-Sternberg, Adam M. Phillippy et al. Molecular Epidemiologic Investigation of an Anthrax Outbreak among Heroin Users, Europe. Emerg Infectious Disease 2012.

Provet. Anthrax. In Provet Healthcare Infromation. Provet; 2013.

Quinn C.P., Dull P.M., Semenova V., Li H. Immune Responses to Bacillus anthracis Protective Antigen in Patients with Bioterrorism-Related Cutaneous or Inhalation Anthrax. The Journal of Infectious Diseases. 2004: 190-199.

Schaechter M., Ingraham J., Neidhardt, Microbe. F. Bacillus anthracis. In Microbe Wiki. Edited by Ucar G., Pogliano K., Holzhauer D. Washington DC: ASM Press; 2006.

Sherer K., Li Y., Cui X., Eichacker P.Q. Lethal and Edema Toxins in the Pathogenesis of Bacillus anthracis Septic Shock Implications for Therapy - Critical Care Perspective. Am J Respir Crit Care Med. 2007, 175: 211–221.

Spencer R.C. Bacillus anthracis. J Clin Pathol. 56:182-187.

Stoltenow C.L., Hugh-Jones M. Anthrax. Christian Veterinary Mission 2010, 33(3): 9.

  ISSN: 2283-3927

Todar K. Bacillus anthracis and Anthrax. In: Todar's Online Textbook of Microbiology. 2012.

Turnbull W.B.. PCB: Anthrax in animals. Molecular Aspects of Medicine 2009, 30: 9.

Turnbull, Böhm, Cosivi, Doganay, Hugh-Jones, Joshi, Lalitha, de Vos. Guidelines for the Surveillance and Control of Anthrax. In Humans and Animals. World Helth Organization. 1998, pp: 97.

Turner A.J., Galvin J.W., Rubira R.J., Condron R.J., Bradley. T: Experiences with vaccination and epidemiological investigations on an anthrax outbreak in Australia in 1997. Journal of Applied Microbiology. 1999, 87(2): 294-297.

Tweet. Preventing Anthrax in Livestock, Hobby Farms. 27 August 2010.

Veterinary Services AHA. Australian Veterinary Emergency Plan. 2012.

Vyas J.M.. Anthrax. In Medline Plus. Massachusetts: U.S. National Library of Medicine. 2013

Williams A.A., Parashar U. D., Stoica A., Ridzon. R: Bioterrorism-Related Anthrax Surveillance, Connecticut, September–December, 2001. Emerging Infectious Diseases. 2002, 8(10): 5.

World Health Organization. Anthrax in humans and animals. In 4 ed. Geneva: WHO Press, 2008.

World Health Organization. B051 - Anthrax. In World Health Organization, Guidelines for the Surveillance and Control of Anthrax in Humans and Animals.

  ISSN: 2283-3927