MERIT RESEARCH JOURNALS
www.meritresearchjournals.org
Merit Research Journal of Medicine and Medical Sciences (ISSN: 2354-323X) Vol. 7(8) pp. 281-286, August, 2019
Available online http://www.meritresearchjournals.org/mms/index.htm

Case Report

Anthrax meningoencephalitis, a diagnostic and treatment challenge – a case report

Sapira Violeta[1], Telehuz Anca[2], Elkan Eva Maria[3] and Lungu Mihaela[1*]

[1]Emergency Clinical County Hospital "Sfantul Apostol Andrei" Galati, "Dunarea de Jos" University of Galati, Romania

[2]Slobozia Emergency County Hospital, Romania

[3]Pediatric Emergency Clinical Hospital „Sfântul Ioan" Galați, Romania

***Corresponding Author's E-mail: micalungu@gmail.com**

Abstract

***Bacillus anthracis* (anthrax) infection is rarely diagnosed in Romania, cases being sporadic, coming especially from the agrarian environment. Anthrax is a zoonotic infection, and humans are incidental hosts. Early nonspecific symptomatology makes detection of anthrax cases difficult, but compete anamnesis related to patient activity or exposure to animal products can raise suspicion of anthrax. A patient with a clinical suspicion of bacillus anthracis infection should receive an effective treatment very quickly to avoid neurological complications that have a high death rate.**

Keywords: Bacillus anthracis, Anthrax, Meningoencephalitis, Diagnosis, Treatment

Abreviations: GCS – Glasgow Coma Scale**; HIV** – human immunodeficiency virus**; CT** - computed tomography**; MRI –** magnetic resonance imaging; **CSF** – cerebrospinal fluid**; PCR** – polymerase chain reaction**; DNA** - deoxyribonucleic acid; **TPHA** – Treponema pallidum Haemagglutination Assay; **VDRL** - Venereal Disease Research Laboratory; **PMN** – polymorphonucleocytes; **HCV** – hepatitis C virus; **HBV** – hepatitis B virus.

INTRODUCTION

Currently, anthrax draws attention due to the possibility of using anthracis bacillus as a biological weapon (Khoddami, 2010). Anthrax is an acute infection produced by anthracis bacillus which is a gram-positive bacterium found in the environment as spores and can remain viable in soil for decades (Wenner, 2004; Sweeney, 2011). Anthracis bacillus is transmitted to humans by contact with infected animals or contaminated animal products that serve as a reservoir for human disease (Rajput et al., 2017). This infection can cause the following clinical syndromes, depending on the type of contamination: cutaneous anthrax (if there are previous skin lesions), gastrointestinal and inhalational anthrax (Swartz, 2001; Spencer, 2003; Maguiña, 2005). Recently an anthrax form has been described that occurs in subcutaneous drug users, "injectional anthrax" (Ringertz, 2000; Sweeney, 2011), with a higher mortality rate than cutaneous form. If the diagnosis of cutaneous forms is quite easy, due to the presence of black skin ulcers, the diagnosis of rare forms of anthrax is difficult (Khoddami, 2010; Kwong, 1997).

All clinical forms can be complicated by sepsis, meningoencephalitis and can have a fatal outcome. The inhalation form has the highest mortality (Maguiña, 2005; Bartlett, 2002; Lanska, 2002). Meningoencephalitis with anthracis bacillus is considered a rare manifestation of the disease, but also a complication associated with increased mortality (Sejvar, 2005). The incidence of meningoencephalitis is not yet well established. Of all patients with less than 5% anthrax developed meningoencephalitis (Doğanay, 2008; Özkaçmaz, 2017), regardless of the initial form of the infection: cutaneous,

gastrointestinal or inhalatory. Lanska (2002) and Haight (1952) evaluated the source of anthrax in 143 patients diagnosed with meningoencephalitis: 41% was cutaneous, 31% was inhalational, 13% it was gastrointestinal and 14% with unknown entrance gate.

Bacillus anthracis has three major virulence factors: an antiphagocytic capsule (protective antigen), the production of two exotoxins (lethal and edema) and the ability of the bacterium to reach high concentrations in the infected host (Friedlander, 2001). The protective antigen binds to target cell surface receptors, allowing the lethal and edema factors to bind and enter the cell. These contribute to the septic syndrome which can lead to multiorgan failure and death (Meyer, 2003; Khoddami, 2010). Anthrax meningoencephalitis is usually secondary to septicemia and develops by lymphatic or hematogenous dissemination of the primary lesion (Gürcan, 2005).

CASE REPORT

We present the case of a 42-year-old male patient that came in the emergency department for speech disorders and right hemiplegia, symptoms starting during the morning of the admission day. We underline the fact that the patient was incarcerated about 15-20 years ago and is currently working as a shepherd – informations that were obtained after admission. At the time of admission the patient had no fever.

The general clinical examination reveals: lesions crusts, without edema or perilesional erythema, without spontaneous accretion or when the crust is detached and a bleeding lesion appears. The lesions highlighted on the patient are: two lesions in the right forearm, one in the right hand and another in the right leg (these are infracentimetric lesions) and also one lesion on the left police with dimensions of 20/10mm. It can be noted a left laterocervical adenopathy and a left axillary adenopathy with dimensions of 20/20mm, mobile on the superficial and deep plane, without signs of local inflammation.Other adenopathies are identified in the inguinal region (the left one larger than the right one) with dimensions of approx 20/20mm having the same characteristics. The neurological examination at the presentation in the emergency department shows the following: altered general state, stiffness of the neck, eyeballs with parallel axis, does not follow movement with the eyes, reactive intermediate pupils, right facial paresis, right hemiplegia, positive Babinski on the right leg, mixed aphasia, aware, uncooperative, agitated.

A cerebral tomography was urgently performed. A spontaneous hypodense lesion is detected at the level of the left cerebral hemisphere, raising the suspicion of ischemic stroke in the superficial territory of the left middle cerebral artery. The patient had normal temperature at the time of admission, and the onset of symptomatology could not be specified because of the lack of informations (from patient or next to him).

Twelve hours after admission the patient presented fever of 39 degrees C, the general condition progressively worsens up to 7 points Glasgow Coma Scale ($M_1V_2O_4$) and has acute respiratory failure (oxygen saturation 86%). A lumbar puncture is done and the cerebrospinal fluid (CSF) is clear, with increased proteins (0.69 g/l) and increased values of the elements (1750 elements/mm^3). Meningoencephalitis is suspected and the tomography aspect is interpreted as secondary cerebritis, the patient being transferred to the infectious diseases department. In the following days the lumbar puncture is repeated and a smear with the Ziehl Neelsen stain is made, also a Gram color smear, a Giemsa color smear, an Ink from China color smear and bacterial cultures classic system all of wich were negative, without identifying the pathogen of meningoencephalitis (Table 1). The dynamics of CSF changes are presented in table 1.

Also, there are cultures from the CSF in the BacTALERT system that are negative for *Staphylococcus* spp, *Streptococcus* spp, *Enterococcus* spp, *Enterobacteriaceae*, *Pseudomonas* spp, *Acinetobacter* spp, *Haemophilus* spp, *Neisseria meningitidis*, *Candida* spp, *Cryptococcus neoformans*, including *Bacillus anthracis*.

For a more rapid diagnosis, a Real Time PCR method for the detection of Mycobacterium tuberculosis DNA and Mycobacterium aviumis performedfrom CSF and crusts that appear to be negative, but DNA detection for anthracis bacillus in both CSF and skin lesions was positive.

At the admission the serologic tests for HIV, HBV, HCV, VDRL, TPHA were negative. Non-specific inflammation tests had high values (fibrinogen 819mg/dl, erythrocyte sedimentation rate 73mm/h, leukocytes 10600/mm^3) and the qualitative determination of procalcitonin being positive (the reference range between 2-10ng/ml) raise the suspicion of sepsis. Two sets of blood cultures were taken that were negative for aerobic and anaerobic flora.

Complementary imaging investigations of cerebral MRI with contrast highlights left parietal cortico-subcortical lesions with significant perilesional edema, with dimensions of 21/14mm and 10/8mm, having an appearance compatible with brain abcesses.

Pulmonary radiography performed at the admission shows the opacification of the right costodiaphragmatic sinus with a small amount of pleural fluid and the accentuation of the bilateral intercleidohilar interstitial drawing, with the tendency to form an alveolar focal intercleidohilar outbreak. Further, the serial x-rays highlight the complete resolution of the pneumonic outbreak and the resorption of the pleural effusion.

Initially, in the context of bacterial meningoence-phalitis, a broad spectrum of antibiotics is established:

Table 1. The dynamics of CSF changes during the admission time

	Day 1	**Day 2**	**Day 3**	**Day 14**	**Day 22**
Macroscopic aspect	clear	serocitrin	clear	clear	clear
Pandy reaction	++	++++	++++	negative	negative
Elements/mmc	1750	730	1140	57	24
Giemsa color smear	-	mononucler cells 75%, rare red blood cells	50% PMN, 50% mononucler cells	mononucler cells	very rare mononucler cells
Gram color smear	-	-	Absent: Gram positive, rounded and elongated in diplo coccus; absent Gram negative, round, in diplo and "coffee beans" like coccus; absent cocobacilli or Gram negative bacilli; absent Gram positive bacilli; absent oval formations (yeasts)	negative, without viewing coccus or bacilli	-
Ziehl-Neelsen color smear	-	-	negative	negative	-
Ink from China smear	-	-	Absent: Cryptococcus neoformans, Candida spp	negativ	-
Bacterial cultures	-	-	Absent: *Staphylococcus* spp, *Streptococcus* spp, *Enterococcus* spp, *Enterobacteriaceae,* *Pseudomonas* spp, *Acinetobacter* spp, *Haemophilus* spp, *Neisseria meningitis*, *Listeria monocytogenes*, *Candida* spp, *Cryptococcus neoformans*, **Bacillus anthracis**	-	-
Protein level (g/l)	0.69	4.2	3.298	0.382	0.407
Albumin level (g/l)	-	-	1.715	0.174	0.162
Glucose level (g/l)	0.58	0.37	0.75	0.50	0.60

meropenem (2 g at 8 hours, intravenous), vancomicyn (1.5 g at 12 hours, intravenous) and ciprofloxacin (400 mg at 8 hours, intravenous). After establishing the etiology of meningoencephalitis with bacillus anthracis, antibiotic treatment with meropenem (2 g at 8 hours) and ciprofloxacin (400 mg at 8 hours) was continued and total duration of the intravenous treatment was 27 days. High dose corticosteroid therapy is also performed (metilprednisolon 1000 mg per day, 5 days).

Evolution was favorable with remission of the coma 48 hours after admission with Glasgow Coma Scale 14 points ($M_6V_4O_4$), significant improvement of the right motor deficit and speech disorders. Bacillus anthracis DNA detection by PCR method is repeated in 20 days after admission and it was negative. The patient is discharged after 33 days from admission with recomandation to continue the treatment with ciprofloxacin (1000mg per day) for another 21 days.

DISCUSSIONS

In general, anthrax is considered not to be contagious and rare cases of human-to-human transmission have been described (Garg, 2018). Transmission to humans is due to contact with infected animals or the use of contaminated animal products. The normal evolution of systemic anthrax infection includes the spread of bacilli from the entrance gate (cutaneous, gastrointestinal or pulmonary) in the blood and from there to different organs, the central nervous system being probably one of the last organs affected, due to the need to overcome the hematoencephalic barrier (Ben-Shmuel, 2018). Neurological manifestations may be the initial symptoms that lead to the diagnosis of anthrax, as is our case.

Neurological complications of the skin anthrax are less common, occurring in about 5% of cases (Maguiña, 2005). Meningoencephalitis is characterized by a prodromal period of 1-6 days, it has a fast evolution and high mortality rates (Kanungo, 2002; Lanska, 2002; Meyer, 2003). Overall, 75% of patients die within the first 24 hours after the onset of neurological symptoms but the mortality of secondary meningoencephalitis in cases of cutaneous anthrax is estimated to be aproximately 95-100% (Lanska, 2002; Maguiña, 2005).

But meningitis may also appear as a complication in

the inhalation anthrax which is caused by inhalation and alveolar deposition of the spores with dimensions smaller than 5 µm (Wenner, 2004). It has a two-phase evolution, with an incubation period of about 4 days (Sweeney, 2011). The spores are phagocytosed by macrophages and transported to the mediastinal lymph nodes where it germinates in vegetative forms, it reproduces and causes hemorrhagic mediastinitis (Abramova, 1993; Sweeney, 2011). If the treatment is inefficient, bacteremia and toxemia will lead to meningoencephalitis. Pulmonary radiography shows pleural effusion (radiological aspect also identified in our patient) or an enlarged mediastinum, aspect present in 100% of these patients, regardless of their evolution (Sweeney, 2011).

CSF in patients with anthrax meningoencephalitis is classically described as hemorrhagic (Lanska, 2002). In our case the CSF was clear, this case being rarely described in the specialized literature (nonhemorrhagic meningoencephalitis). Out of a total number of 43 cases in which the CSF examination was reported only in 3 cases the CSF aspect was clear and in only 4 cases the number of erythrocytes in CSF was below 100/mm^3 (Lanska, 2002). In another study (Katharios-Lanwermeyer, 2016), CSF was described as hemorrhagic in two thirds of the 65 cases evaluated with meningitis. Also, according to published studies, CSF may not be hemorrhagic at the onset of the disease (Kindler, 1952; Levy, 1981; Rangel, 1975), but in our case the lumbar serial punctures (5 lumbar punctures were performed) did not reveal the classic hemorrhagic aspect described in the literature, CSF being clear throughout the hospitalization. The repeated CSF examination allowed the monitoring of the effectiveness of the therapy administered (reduction of the number of leukocytes, normalization of protein and glucose level).

In terms of image evaluation (cerebral tomography or cerebral MRI) in this category of patients, studies revealed focal intracerebral hemorrhage, subarachnoid hemorrhage, intraventricular hemorrhage or diffuse cerebral edema (Garg, 2018; Gurcan, 2005; Lanska, 2002; Leblebicioglu, 2006; Domínguez, 1997; Dewar, 2015). Parenchymal lesions have not been reported in the literature (Kim, 2001), but the absence of these descriptions may only reflect the small number of neuroimaging evaluations in this category of patients. In our case, the evaluation of the cerebral MRI revealed abcesses with significant perilesional edema that showed a favorable evolution under the antibiotic treatment and the corticotherapy administered in high doses.

Pulmonary x-ray reveals pleural effusion and accentuation of bilateral intercleidohilar interstitial drawing, radiologic aspect remitted under the antibiotic treatment administered, which suggested that the patient also presented inhalatory anthrax. Because no pleural puncture was performed the diagnosis of certainty for inhalational anthrax could not be established.

The laboratory diagnosis of anthrax is based on the isolation of the anthracis bacillus from pathological products (skin lesions, blood or CSF) on a Gram colored smear. The bacilli are Gram positive, either isolated or in shortchains (Mari et al., 2017). On culture media blood agar, after incubation for 18-24 hours, virulent strains develop opaque round colonies, with rough surface and irregular edges, 2-5 mm in diameter (Spencer, 2003). Blood cultures are usually positive in the range of 6 to 24 hours. Anthrax should be taken into consideration immediately if the Gram coloration of the collected samples shows Gram-positive bacilli growing in chains (Sweeney, 2011). In our patient's case the Gram colored smear both from the CSF (Table 1) and from the skin lesions did not allow the identification of the anthracis bacillus. Gram colored smear from skin wounds revealed rare epithelial cells, Gram positive coccus, round diploid and stack cells and absence of yeasts. Aerobic bacterial cultures from superficial wounds were also negative for anthracis bacillus. The identification of the pathogen from both CSF and skin lesions was achieved only by the real-time polymerase chain reaction method (PCR), a secure and fast method for identifying the anthracis bacillus.

Given the severity of the infection and the high mortality in these cases, at the first suspicion of the disease antibiotic treatment should be instituted.

Penicillin was considered to be the first-line medication, and naturally resistant strains were rarely identified, but the fact that the Ames strain, that causes recent infections in the USA has beta-lactamase, systemic anthrax treatment is not only done with penicillin alone (Spencer, 2003).

Current Centers for Disease Control and Prevention (CDC) recommendations for the treatment of anthrax meningitis include the association of a fluoroquinolone (levofloxacin or ciprofloxacin) with a protein synthesis inhibitor (linezolid, as the first choice drug) and a β-lactam (meropenem as the first choice or imipenem) (Ben-Shmuel, 2018; Pillai, 2015). For systemic anthrax, without meningitis (including inhalational anthrax and other forms of anthrax with systemic involvement) recommendations also include vancomycin as a potential bactericidal agent (Hendricks, 2014). In our patient, inhalational anthrax also being suspected, the treatment with vamcomycin was initiated (in dose of 1,5 g at 12 hours).

Also based on reported cases, glucocorticoids may be used as adjuvant therapy in patients with meningo-encephalitis but this recommandation is based only onclinical experience in treating meningitis with another etiology (Inglesby, 2002; Landska, 2002).

In the case of inhalational anthrax, due to the 60 days latency period, for germination in the mediastinal lymphnodes of inhled spores (Swartz, 2001), prophylactic therapy with ciprofloxacin (500 mg bd) up to 60 days is recommended (Friedlander, 1993; Spencer, 2003). Given these recommendations, our patient was discharged with

recommentation to continue ciprofloxacillin therapy for another 21 days.

The evolution was favorable, the patient receiving 3 associated antibiotics which is consistent with the data in the specialty literature that reported a higher survival rate in patients with triple antibiotic therapy compared to patients receiving only two antibiotics (Pillai, 2015; Sejvar, 2005). Regarding the duration of the intravenous therapy, the studies recommend a therapy of at least 2 weeks (Pillai, 2015; Holty, 2006), in our patient the duration of intravenous therapy being 27 days.

CONCLUSIONS

Anthrax should be considered in the differential diagnosis of patients with fever and acute neurological symptoms that associate cutaneous necrotic ulcers. The identification of Gram positive bacilli in CSF, with or without hemorrhage, allows the diagnosis to be established. Suspicion is increased if the patients with meningitis have a history of contact with animals, such as our patient. For patients with anthrax early diagnosis and broad-spectrum antibiotic therapy can be life-saving.

Informed consent

The patient's informal approval has been obtained and recorded in the chart.

Author contributions

All the authors have equal contributions in this presentation.

Ethical approval

This article does not contain any studies with human participants performed by any of the authors.

Financial disclosure: None

Grant information

This article did not receive any specific grand from funding agencies in the public, commercial or not-for-profit sectors.

ACKNOWLEDGEMENTS

Thanks go to the proof readers for providing language help and for proof reading the article.

REFERENCES

Abramova FA, Grinberg LM, Yampolskaya OV, Walker DH (1993). Pathology of inhalational anthrax in 42 cases from the sverdlovsk outbreak of 1979. Proc Natl AcadSci USA;90:2291–2294.

Bartlett JG, Inglesby Jr TV, Borio L (2002). Management of anthrax. Clin. infect. Dis., 35: 851-858.

Ben-Shmuel A, Glinert I, Sittner A, Bar-David E, Schlomovitz J, Brosh T, Kobiler D, Weiss S, Levy H (2018). Treating Anthrax-Induced Meningitis in Rabbits. Antimicrobial Agents and Chemotherapy; 62 (7):e00298-18

Dewar S, Weir-McCall JR, Batchelor L, Cole S, Parcell BJ (2015). Bacillus anthracis meningitis during an outbreak of injectional anthrax, Scotland, UK. ClinMicrobiol Infect 2015; 21: e49–e50

Doğanay M (2008). Bacillus anthracis and other Bacillus species. In: Topcu AW, Soyletir G, Doğanay M, editors. Infectious diseases and their microbiology. Istanbul: Nobel Press. p.2102-14.

Domínguez E, Bustos C, Garcia M, Recio S (1997). Anthrax meningoencephalitis: radiologic findings. AJR Am J Roentgenol; 169:317.

Friedlander AM, Welkoss L, Pitt MLM et al (1993). Postexposure prophylaxis against experimental inhalation anthrax. J Infect Dis;167:1239– 43.

Friedlander AM. Tackling anthrax (2001). Nature;414:160–161.

Garg N, Panmei K (2018). First reported case of naturally acquired fatal anthrax from Northeast India. J Family Med Prim Care. 7(3): 632-634

Gürcan S, Akata F, Kuloğlu F, Erdoğan S, Tuğrul M (2005). Meningitis Due to Bacillus Anthracis. Yonsei Med. J. Vol. 46, No. 1, pp. 159-160

Haight TH (1952). Anthrax meningitis: review of literature and report of two cases with autopsies. Am J Med Sci;224:57–69.

Hendricks KA et al (2014). Centers for disease control and prevention expert panel meetings on prevention and treatment of anthrax in adults. Emerg Infect Dis.; 20(2)

Holty JE et al (2006). Systematic review: a century of inhalational anthrax cases from 1900 to 2005. Ann Intern Med.; 144(4):270–80

Inglesby TV, O'Toole T, Henderson DA, Bartlett JG, Ascher MS, Eitzen E, Friedlander AM, Gerberding J, Hauer J, Hughes J et al (2002). Biodefense WGoC. Anthrax as a biological weapon, 2002: updated recommendations for management. JAMA; 287:2236–2252.

Kanungo R, Kumar A, Bhattacharya S et al. (2002). Problem of timely diagnosis in anthrax meningitis. J. Ass. Phycns India, 50: 913-915.

Katharios-Lanwermeyer S, Holty JE, Person M, Sejvar J et al (2016). Identifying Meningitis During an Anthrax Mass Casualty Incident: Systematic Review of Systemic Anthrax Since 1880. Clin Infect Dis. 15; 62(12): 1537–1545

Khoddami M, Shirvani F, Esmaeili J, Beladimogaddam N (2010). Two Rare Presentations of Fatal Anthrax: Meningeal and Intestinal. Archives of Iranian Medicine; 13(5): 432-435

Kim HJ, Jun WB, Lee SH, Rho MH (2001). CT and MR findings of anthrax meningoencephalitis: report of two cases and review of the literature. AJNR Am J Neuroradiol;22:1303–1305.

Kindler D (1952). Recovery from anthrax meningitis. Ind Med Surg;21:487–488.

Kwong KL, Que TL, Wong SN, So KT (1997). Fatal meningoencephalitis due to Bacillus anthracis. J Paediatr Child Health.; 33: 539 – 541.

Lanska DJ (2002). Anthrax meningoencephlitis. Neurology; 59: 327-334.

Leblebicioglu H, Turan D, Eroglu C et al (2006). A cluster of anthrax cases including meningitis. Trop Doc; 36: 51-53.

Levy L, Baker N, Meyer MP, Crosland P, Hampton J (1981). Anthrax meningitis in Zimbabwe. Cent Afr J Med;27:101–104.

Maguiña C, Del Pozo JF, Terashima A, Gotuzzo E, Guerra H, Vidal J, Legua P, Solari L (2005). Cutaneous anthrax in lima, peru: retrospective analysis of 71 cases, including four with a

meningoencephalic complication. Rev. Inst. Med. trop. S. Paulo 47(1):25-30.
Mari AS, Kamboh AA, Bhutto KR, Sahito IA, Abro SH (2017). Prevalence of Bacillus anthracis spores in soil of district Badin. J. Anim. Health Prod. 5(2): 79-84.
Meyer MA (2003). Neurologic complications of anthrax. A review of the literature. Arch Neurol; 60: 483 – 488.
Özkaçmaz S (2017). CT and MRI findings of anthrax meningoencephalitis; Case report. East J Med 22(3): 114-117
Pillai SK, Huang E, Guarnizo J, Hoyle J, Katharios-Lanwermeyer S, Turski T, Bower W, Hendricks K, MeaneyDelman D (2015). Antimicrobial Treatment for Systemic Anthrax: Analysis of Cases from 1945–2014. Identified through Systematic Literature Review. Health Secur.; 13(6): 355–364.
Rajput M, Kamboh AA, Dewani P, Umrani AP, Rind R (2017). Occurrence of Anthrax spores in small ruminants hair/wool in district Tharparkar, Sindh. J. Anim. Health Prod. 5(1): 5-9.
Rangel RA, González DA (1975). Bacillus anthracis meningitis. Neurology;25:525–530.
Ringertz SH, Høiby EA, Jensenius M, Maehlen J, Caugant DA, Myklebust A, Fossum K (2000). Injectional anthrax in a heroin skin-popper. Lancet;356:1574–1575.
Sejvar JJ, Tenover FC, Stephens DS (2005). Management of anthrax meningitis. Lancet Infect Dis.;5(5):287-95.
Spencer RC (2003). Bacillus anthracis. J ClinPathol;56:182–187
Swartz MN (2001). Recognition and management of anthrax: an update. New Engl. J. Med., 345: 1621-1626.
Sweeney DA, Hicks CW, Cui X, Li Y, Eichacker PQ (2011). Anthrax Infection. American Journal of Respiratory and Critical Care Medicine; 184:1333-1341
Wenner KA, Kenner JR (2004). Anthrax. DermatolClin; 22:247–256.