IDCases 40 (2025) e02216

Contents lists available at ScienceDirect

IDCases

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Case report

# Cutaneous anthrax rapidly progressed into septic anthrax resulting in death – A case report

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ARTICLE INFO

*Keywords:*
Cutaneous anthrax
Hypotension
Acidosis
Neck lesion

ABSTRACT

Anthrax is a fatal zoonotic disease and the acute risk associated with it cannot be underestimated. Cutaneous anthrax accounts for more than 95 % of anthrax and usually does not lead to death. We present a case of severe cutaneous anthrax treated with a three-day course of combination antibiotics therapy, which culminated into death due to multiple organ failure. The patient did not exhibit symptoms suspected of meningitis anthrax and pulmonary anthrax. Computed Tomography revealed subcutaneous exudates in the chest, back, left supraclavicular fossa, and bilateral axillary regions. Some newly factors, like hypotension, elevated creatinine, acidosis, and a urinary tract infection, provide valuable insights into the clinical intervention in early. In rural areas, educating residents about anthrax symptoms and risks is crucial.

## Background

Anthrax is a zoonotic disease caused by *Bacillus anthracis*, a Gram-positive, spore-forming bacterium that poses a significant threat to both livestock and humans. The bacterium harbors two plasmids, pXO1 and pXO2, which encode key virulence factors. Plasmid pXO1 is responsible for producing the anthrax toxins – such as protective antigen, lethal factor, and edema factor, while pXO2 encoded the capsule, escaping the host's immune system [1]. These gene products are key to the offensive (toxin) and defensive (capsule) mechanisms [2]; they are the primary virulence factors that determine the bacterium pathogenicity.

Globally, about 1.83 billion people (95 % confidence interval (CI): 0.59–4.16 billion) are at a risk of contacting anthrax due to their places of residence [3]. The primary mode of infection is through contact with an infected animal or the contaminated animal products. There are three forms of anthrax namely: cutaneous, intestinal, and pulmonary anthrax. China recorded a total of 1244 human anthrax cases between 2018 and 2022, with cutaneous anthrax constituting 98.4 % of these cases [4]. Research indicates that timely treatment of cutaneous anthrax results in positive clinical outcomes, which reduces its fatality rate to under one percent.

We present a case of severe cutaneous anthrax treated with a two-day course of combination antibiotics therapy, which culminated into death due to multiple organ failure. The clinical progression and unique traits observed provide vital insights for healthcare professionals in the prevention and treatment of anthrax infections.

## Case presentation

A 44-year-old male farmer from North China reported at the hospital at 7:54 PM on October 15, 2022. The patient presented with signs including localized redness and swelling on his neck and chest (Fig. 1A). His medical history indicated allergic reactions to penicillin drugs and over 20 years of smoking and daily alcohol consumption of 150 mL. Notably, the patient reported cessation of alcohol consumption.

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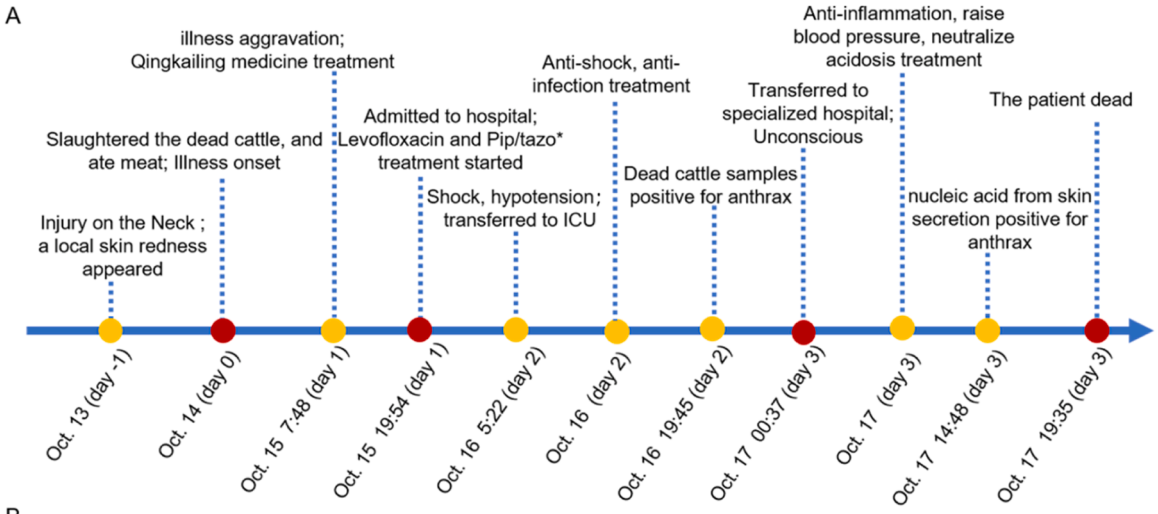
https://doi.org/10.1016/j.idcr.2025.e02216
Received 17 February 2025; Received in revised form 31 March 2025; Accepted 31 March 2025
Available online 3 April 2025

In the emergency department, the patient reported being in good physical condition before sustaining a neck injury from grass while conducting fieldwork two days earlier. Subsequently, a localized bulge with erythema and fluid exudation developed. Notably, the wound did not form a vesicle and the patient did not seek medical attention at that time. One day before reporting to the hospital, the patient, along with others, slaughtered and consumed meat from cattle that had been gored to death. The others did not suffer from any illness. Following this, he experienced pruritus and pain in the left neck and anterior chest, along with purulent discharge from the wound. At 7:48 AM on the day of presentation, a rural physician administered a 30 mL dose of Qingkailing. The condition of patient did not improve, with increased redness and swelling in the anterior chest.

The patient was alert and cooperative upon admission, exhibiting no fever, cough, hemoptysis, or dyspnea (Fig. 1B). Vital signs recorded were as follows: body temperature of 36 °C, heart rate of 122 beats per minute (bpm), respiratory rate of 22 breaths per minute, blood pressure of 66/44 mmHg, and blood oxygen saturation ($SpO_2$) of 90 % while breathing




B

Characteristics of Patient in Emergency Department

| SPO2 | Temperature (℃) | Pulse | Blood Pressure (mmHg) | Respiratory Rate (min $^{-1}$) | Cough | Dyspnea | Hemoptysis |
|---|---|---|---|---|---|---|---|
| 90% | 36 | 122 | 66/44 | 22 | N | N | N |
| White blood cell count (×10$^9$/L) | Neutrophil ratio (%) | Blood Urea Nitrogen (mmol/L) | Creatinine (μmol/L) | Blood glucose (mmol/L) | Sodion (mmo1/L) | Chloride | C-reactive protein (mg/L) |
| 12.27 | 82.2 | 7 | 168 | 10.76 | 129 | 95 | 114.11 |

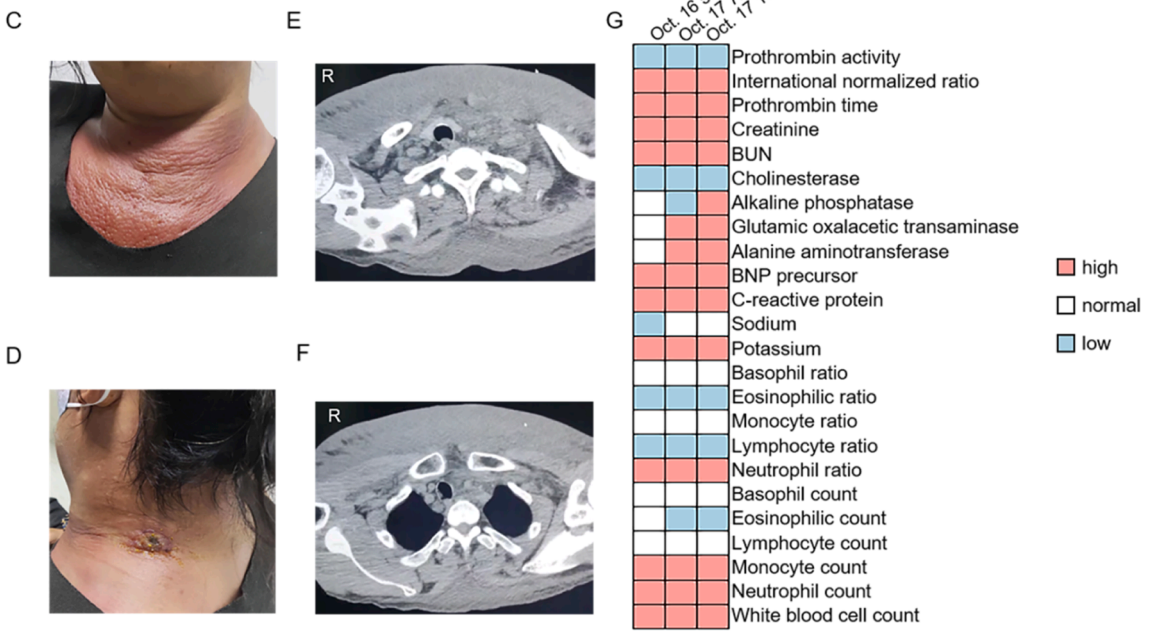



**Fig. 1.** A. Timeline of the clinical course of the patient and identification of the causative pathogen; B. Characteristics of patients in the emergency department; C and D. The photos of patient's lesion; E and F. Computed tomography of chest scan showed widened mediastinal shadows and multiple infections; G. The vital laboratory results for the patient at varying time points.

ambient air. Mildly coarse bilateral lung sounds were detected upon auscultation. Laboratory examination results revealed elevated C-reactive protein and lactic acid, mild leukocytosis, hyponatremia, an increased neutrophil ratio, elevated creatinine, along with normal blood urea nitrogen (BUN) and chloride levels. Physical examinations revealed redness and swelling of the anterior chest skin, along with a thorn-like wound on the left neck (Fig. 1C and D), with no evidence of papules or vesicles.

Nucleic acid testing produced negative COVID-19 results. Subsequently, computed tomography (CT) scanning of the chest was conducted with the results indicating widened mediastinal shadows and multiple infections. The trachea exhibited a mild rightward displacement due to compression (Fig. 1E and F). Observed lesions included subcutaneous exudates in the chest, back, left supraclavicular fossa, and bilateral axillary regions, along with enlarged mediastinal lymph nodes.

The patient was admitted with sepsis and skin infections and received empirical treatment with levofloxacin and piperacillin-tazobactam sodium in the emergency department. Due to a blood pressure of 80/40 mmHg, dopamine and norepinephrine were administered to elevate the blood pressure.

At 5:22 AM on October 16, the patient exhibited mild shock with a blood pressure of 70/40 mmHg. Subsequently, the patient was transferred to the negative pressure isolation unit within the Intensive Care Unit (ICU). Throughout this period, the patient remained conscious and demonstrated adequate limb movement, as well as absence of fever, significant chest pain, or respiratory distress. The patient experienced nausea and vomited three times, expelling stomach contents; Stomach contents could not be determined to be contaminated meat; Additionally, the patient experienced abdominal pain but without diarrhea. Laboratory examinations revealed elevated white blood cell and neutrophil counts, while lymphocyte levels were normal (Fig. 1G and Table S1 in supplementary file). Sodium levels were slightly decreased, while potassium levels were mild increased. And C-reactive protein levels were significantly increased, with C-reactive protein reaching 172.75 mg/mL. The B-type natriuretic peptide precursor (PRO-BNP) was recorded at 141 pg/mL, indicating a heightened risk of heart failure (Table S2). Cholinesterase levels were decreased, while BUN and creatinine were elevated (Table S2). The D-dimer levels were significantly increased and the antithrombin III ratio decreased, along with a slightly extended prothrombin time (Table S3). Arterial blood gas analysis revealed a slight decrease in pH and carbon dioxide ($CO_2$) partial pressure, along with a significant increase in lactate levels (Table S4). Interleukin 6 (IL-6) was recorded at 934.30 pg/mL. Routine blood urinalysis indicated a significant increase in white blood, red blood, and epithelial cells, along with elevated levels of urine crystals (X-TAL) (Table S5), suggesting a urinary tract infection.

The patient received a sequential administration of pantoprazole sodium, meta hydroxylamine bitartrate, methylprednisolone succinate sodium, and meropenem. The medications were aimed at protecting the gastric mucosa, inhibiting acid secretion, elevating blood pressure, replenishing body fluids, mitigating the degree of shock and infection, as well as maintaining proper organ function, acid-base balance, and electrolyte levels.

PCR analysis produced negative results for six pathogens, including *influenza A* and *Mycoplasma pneumoniae*. On October 16 at 12:32 PM, the report from the local Centers for Disease Control and Prevention (CDC) indicated negative results for nucleic acid from skin secretions and antibodies of the serum samples of the patient linked to anthrax infection. Despite these negative results on the patient samples, the CDC results confirmed that the samples from the deceased cattle tested positive for *B. anthracis* nucleic acid, supporting the clinical diagnosis of cutaneous anthrax.

Subsequently, at 13:44 on October 16, the patient received human blood albumin, heparin sodium, and methylamine bitartrate. After the treatment, arterial blood gas testing, at 3:48 PM, indicated normal pH, low total $CO_2$ levels, and significantly elevated lactic acid levels (see Table S4). These results indicated a significantly combined metabolic and respiratory acidosis. The condition of the patient did not improve, exacerbating into a shock, which led to the initiation of vasopressor therapy. Additionally, the patient underwent oxygen therapy and was intubated due to acute hypoxemic respiratory failure; the patient was administered continuous intravenous antihypertensive medications. The patient was continuously monitored with the subsequent results exhibiting a heart rate of 123 bpm, blood pressure of 123/79 mmHg, and blood oxygen saturation ($SpO_2$) of 100 %.

The patient's results indicated a potential of cutaneous anthrax and septic shock. The patient was transferred to a specialized hospital late at night. The vital signs recorded at the time of transfer included a body temperature of 36.2 °C, pulse of 150 bpm, respiratory rate of 33 breaths per minute, and blood pressure of 125/94 mmHg. The patient was unconscious, exhibiting flaky, red, and swollen skin, accompanied with exudates. The surrounding area had a texture reminiscent of grasping snow. Additionally, cyanosis was present in the oral cavity, lymph node swelling, or lower extremity edema. The strength of the patient's neck muscles is weakened, and the head cannot actively stabilize. He was received treatment comprising imipenem and cilastatin sodium, moxifloxacin to address inflammation, and assisted ventilation via an invasive ventilator.

At 7:18 AM on October 17, the patient's pulse was 139 bpm, and the respiration rate was 31 breaths per minute, with undetectable blood pressure and $SpO_2$. Laboratory examinations revealed a prothrombin time of 32.07 seconds, a partial thromboplastin time of 173.86 seconds, a pH of 7.113, and a potassium level of 7.17 mmol/L. Notably, since admission to the specialized hospital, the patient presented with anuria and elevated creatinine, alanine aminotransferase, and glutamic oxalacetic transaminase levels (Fig. 1G). The patient received treatment which included assisted ventilation, fluid infusion, glucose management with an insulin pump, vasoactive medications, parenteral nutrition, intravenous administration of omeprazole, imipenem and cilastatin sodium, and moxifloxacin.

At 1:44 PM on October 17, vital laboratory values included a prothrombin time of 68.75 seconds, partial prothrombin time of 180.44 seconds, and potassium level of 7.34 mmol/L. At this point, the patient exhibited infection, altered consciousness, hypotension, hypothermia, anuria, abnormal blood coagulation, elevated liver enzymes, hyperkalemia, and lactic acidosis. To eliminate the toxins and restore water, electrolytes, and acid-base homeostasis, continuous bedside renal replacement therapy was recommended; additionally, further recommendation involved complete plasma exchange. The local CDC reported that the sample from the patient tested positive for anthrax using PCR, with further validation of the PCR products conducted through sequencing, resulting in the ultimate diagnosis of cutaneous anthrax.

Despite maximal support and resuscitation after admission, the condition of the patient deteriorated, resulting in multiorgan failure and subsequently death. Over the few days, *B. anthracis* strains were noted from the skin exudate of the patient (Figure S1 in supplementary file).

## Discussion and conclusions

The case illustrates the progression of acute cutaneous anthrax. Notably, the patient presented without typical early-stage signs, such as papules, vesicles, or ulcer lesion and lacked associated symptoms such as fever (possibly influenced by Qingkailing medication) and headache [5]. The patient exhibited leukocytosis, elevated C-reactive protein, an increased neutrophil ratio [5,6], and unusual symptoms like hypotension, tachycardia, dyspnea, elevated creatinine, acidosis (characterized by hyperkalemia and elevated lactic acid), and a urinary tract infection. These unusual symptoms complicated the early diagnosis, thereby delaying intervention at the county and township-level hospitals.

The patient experienced a rapid onset of the anthrax, which culminated into his death, potentially due to two key risk factors: excessive alcohol consumption and vulnerable wound location. Excessive alcohol

consumption impairs alveolar macrophages [7], the primary defense against *B. anthracis* [8]. The location of the wound is critical in the treatment of the disease, with most cases of cutaneous anthrax occurring on fingers, arms [9], and or the eyelids [9,10], with treatment often involving antibiotics. In this case, the location of the wound on the neck facilitated direct entry of the bacteria to the bloodstream, rapidly affecting the central nervous system. The infection increased D-dimer levels, risk of heart failure, organ insufficiencies, hypotension, lactic acidosis, oliguria, disseminated intravascular coagulation (DIC), and septic anthrax. The case eventually developed systemic anthrax with gastrointestinal introduction and skin lesions. These effects collectively resulted into multiple organ failure and ultimately death of the patient.

The patient exhibited intolerance penicillin, necessitating the modification of the treatment to include levofloxacin in combination with piperacillin and tazobactam sodium. Management of anthrax, caused by *B. anthracis*, should include bactericidal antimicrobials and an antitoxin, given that accumulation of toxins exacerbates the disease. Currently, three antitoxins have been approved by the U.S. Food and Drug Administration (FDA): anthrax immunoglobulin, raxibacumab, and obiloxaximab [11]. Unfortunately, rural hospitals may lack access to these critical medications. In addition, inadequate targeted treatment prior to diagnosis and a lack of knowledge among doctors in rural areas regarding the treatment of severe anthrax may contribute to the sudden deaths of patients. Furthermore, A regimen of antibiotics combined with protease inhibitors was not used.

Our investigation found that most anthrax outbreaks are associated with livestock contact, especially through slaughtering, skinning, or consumption of undercooked meat. This highlights a lack of awareness about anthrax prevention, which is further worsened by financial burdens on the affected individuals. Consequently, in rural areas, educating residents about anthrax symptoms and risks is crucial, and it should be conducted along with enhanced compensation programs for related losses to improve prevention and control efforts.

In conclusion, anthrax infection developed rapidly. The case eventually developed systemic anthrax and the death of patients. Some newly symptoms, like hypotension, elevated creatinine, acidosis, and a urinary tract infection, maybe provide evidence base into the clinical intervention in early.

## Author agreement

All authors have read and agreed to the published version of the manuscript.

## Ethics approval and consent to participate

This study was approved by the Ethics Committee of the Academy of Military Medical Sciences (ethics approval code AF/SC-08/02.392). Informed consent was obtained from the mother of the patient.

## CRediT authorship contribution statement

Yufei Lyu, Hua Shao and Dongshu Wang designed the study. Yufei Lyu, Shiyuan Li, Jia Zhou and Li Nie collected the data. Meijie Feng, Wenjun Li, Xian Liu and Yufei Lyu analysed the data. Chao Pan, Shujuan Yu, and Yan Guo interpreted the data. Yufei Lyu was the major contributors in writing the manuscript. Yufei Lyu, Shiyuan Li, Dongshu Wang and Meijie Feng played key roles in these efforts. We ensure that each first-author is qualified to be listed as a first-author.

## CRediT authorship contribution statement

**Li Wenjun:** Formal analysis. **Liu Xiankai:** Formal analysis. **Feng Meijie:** Formal analysis. **Guo Yan:** Data curation. **Shao Hua:** Supervision, Conceptualization. **Lyu Yufei:** Writing – original draft, Investigation, Data curation, Conceptualization. **Li Shiyuan:** Investigation. **Wang Dongshu:** Supervision, Conceptualization. **Zhou Jia:** Investigation. **Nie Li:** Investigation. **Pan Chao:** Data curation. **Yu Shujuan:** Data curation.

## Declaration of Competing Interest

The authors declare no conflict of interest.

## Acknowledgments

The authors are grateful to the staff from Tongliao Center of Disease Control and Prevention, Tongliao City People's Hospital, and The Sixth People's Hospital of Tongliao City for their valuable assistance with regard the clinical samples and information. We also would like to express our gratitude to the patients, their families, and suspected contacts for their cooperation in facilitating epidemiological investigations and taking measures to prevent the further spread of the disease.

This work was supported by the National Key R&D Program of China (grant number: 2022YFC2304003), and National Natural Science Foundation of China (grant numbers: 82102412 & 82172317).

## Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.idcr.2025.e02216.

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